AN ELECTROPHYSIOLOGICAL STUDY OF THE
ANATOMIC RELATIONS OF TWO GIANT
NERVE CELLS IN APLYSIA DEPILANS

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Histological studies of the central nervous system of invertebrates using methylene-
blue and silver-staining techniques, alone or in conjunction with degeneration
experiments, have provided a considerable amount of information about neuronal
pathways. The use of electrophysiological methods, however, makes it easier to trace
the path of individual neurones over long distances and can be especially helpful in
invertebrates where the path of an axon is complicated by its passage through the
neuropiles. In addition, they may give some insight into the types of functional
connexion which exist between neurones.

The technique of dissecting connectives between pairs of ganglia and recording
activity of single units in small bundles has proved successful in some arthropods
(Wiersma, 1958; Hughes & Wiersma, 1960; Fielden & Hughes, 1962), but many
molluscan nerves are not so amenable to such an approach because they are not easy
to split. However, the use of microelectrodes inserted into single ganglion cells makes
it possible to investigate single units in such animals. From this point of view the
material used in the present studies was very suitable, partly because the ganglia are
widely separated, and also because some of the nerve cell bodies are so large. Further-
more, a considerable amount of information is available about the different sorts of
electrical activity which may be recorded from individual ganglion cells in isolated
preparations (Tauc, 1955, 1962a), and in addition some account has already been given
of the general nature of responses recorded from this ganglion in the intact animal
(Hughes & Tauc, 1962).

The present paper is concerned particularly with the anatomy and function of the
so-called 'giant cell' of the abdominal ganglion, which has been used more than any
other in studies of isolated ganglion preparations. An interesting finding in the present
work was the great ramification of the axon of this giant cell, which extends throughout
nerves innervating both the foot and parapodia on the right side. A homologous giant
neurone on the left side of the animal has also been found and shown to have similar
anatomical relations. Because of their size and distribution and by analogy with other
large neurones (e.g. Mauthner) it seemed justifiable to ascribe some motor function
to the cells and to suggest that they might operate, for instance, during escape responses.
However, from the results obtained, no indications were found for such a function.

A preliminary report of this work has already been published (Hughes & Tauc, 1961).

MATERIAL AND METHODS

All experiments were carried out at room temperatures (20–22° C.), using large specimens of *Aplysia depilans*, recently collected and kept in sea water. In many cases, whole-animal preparations were used for recording intracellularly from the giant cell of the abdominal ganglion in addition to recording by external silver/silver chloride electrodes from different connectives and peripheral nerves. Other preparations were made in which the principal central ganglia and the main pedal and parapodial nerves were isolated from the animal and pinned down in a waxed Petri dish. In these preparations the giant cell bodies were clearly visible under a binocular microscope and the sheath overlying them was carefully cut with a micro-scalpel. Details of the dissections, apparatus and recording methods have been described previously (Hughes & Tauc, 1962).

RESULTS

I. PHYSIOLOGICAL ANATOMY OF THE GIANT CELLS

(1) *The right giant cell (RGC)*

This cell is located dorsally on the right side of the abdominal ganglion complex, in that part which has been presumed to represent the right parietal ganglion (Fig. 1).

Path of the RGC axon

Within the central nervous system of *Aplysia* there are a relatively large number of direct pathways passing through individual ganglia (Hughes & Tauc, 1962). A notable example is the RGC axon, which has a surprisingly extensive distribution. Previous workers have established that an axon of this cell only leaves the abdominal ganglion in the right connective. Its path thereafter has remained unknown because most preparations were of the abdominal ganglion isolated from the rest of the central nervous system. Electrical stimulation experiments using preparations of the whole central nervous system, *in situ* or in isolation, have given convincing evidence, developed in the following paragraphs, that this axon divides in the right pleural and/or pedal ganglion and sends a branch into each parapodial nerve and at least the main pedal nerve. In addition, an axon continues along the right cerebro-pleural connective (C-Pl) to the cerebral ganglia and from here to the left cerebro-pleural connective (Fig. 1).

(i) Intracellular recordings during antidromic stimulation of the RGC axons. The arrival of an impulse set up antidromically in the axon of the RGC produces a clearly defined antidromic potential recorded intracellularly in the cell soma. Fig. 2 shows such recordings accompanied by external electrode recordings on the right connective which demonstrate the antidromic nature of the spike. Slight differences in the precise shape of the potential recorded are due to synaptic effects resulting from stimulation of other pathways in the nerves concerned. Stimulation of the right C-Pl, for example, produces a large synaptic effect in the cell. In these experiments, however, it was found in several instances that conduction between different branches of the cell and main axon in the right connective was blocked at the ramification within the pleural–pedal ganglia. When this happened, a stimulus to the nerves failed to evoke an antidromic potential in the RGC. Failure to conduct at relatively low frequencies and the ease with which transmission became fatigued were characteristic of this condition.
Several stimuli were often necessary to allow the spike to cross the ramification. In some cases of repetitive stimulation of a nerve, e.g. the right posterior parapodial nerve ($S_9$, Fig. 3e), the first shock produced only a synaptic potential in the RGC, whereas the second produced two impulses in the right connective followed by two antidromic potentials in the cell. Subsequent stimuli evoked both a single impulse in the right connective and an antidromic potential in the cell.

(ii) Stimulation of the RGC by depolarization of the soma. With a micro-electrode in the cell body the polarization of the cell membrane was varied, using a bridge circuit similar to that of Araki & Otani (1955). When polarization of the cell membrane was suddenly decreased, either relative to its normal resting potential or by restoring the normal potential following hyperpolarization, the cell could be excited and give a burst of quite high frequency which gradually adapted (Fig. 4). This technique
proved very valuable for tracing the orthodromic propagation of a potential along the branches of the RGC. External electrodes placed on any of the nerves containing an RGC axon recorded a potential which followed that in the right connective after a

![Fig. 2](image1)

**Fig. 2.** Potentials recorded intracellularly (R') in the RGC and extracellularly in the right connective (R) following stimulation to the right C-Pl (S), right posterior parapodial nerve (S), and right pedal nerve (S). In each case an impulse is propagated posteriorly in the right connective and precedes the antidromic spike recorded intracellularly. Calibration: 25 msec., 50 mV.

![Fig. 3](image2)

**Fig. 3.** Stimulation of the RGC axon in (a) right pedal nerve (S), (b) right posterior parapodial nerve (S), and (c) the right C-Pl(S). In each case the RGC also receives synaptic input, which is particularly marked following stimulation of the C-Pl. At lower frequencies in this record (c) only a single antidromic spike is produced but at higher frequencies and intensities (d) or when the preparation is less fatigued a spike is produced to almost every stimulus. Notice in these two records the small spike in the right connective which precedes the giant spike as it is more rapidly conducting. Stimulation of the posterior parapodial nerve (b and e) also gives synaptic input and in both records shown the first shock to produce any antidromic spike in the right connective gives rise to two impulses.
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short delay. The large potential in the right connective is easily recognizable but RGC impulses in the other nerves are often difficult to distinguish from the background activity. However, by using the spike in the right connective to trigger the time base, the recognition of a potential in another nerve associated with the RGC was made more easy. Mechanical as well as electrical stimuli could then be tested, as the RGC gives a discharge of impulses following slight pressure on the somatic membrane. Fig. 5 shows the value of this technique in tracing the branch of the RGC into the right C-Pl and thence into the left C-Pl. In this way the branches of the RGC axon were traced into all the nerves shown in Fig. 1. Failure of transmission at the branching within the pleural–pedal ganglion complex was never observed when propagation took place in this orthodromic direction.

(iii) Transmission between the parapodial nerves and connectives containing RGC axons. During the course of studies on central pathways it was found that the RGC was excited by electrical stimulation of many different nerves. Excitation of this cell was recognized again by the presence of either an antidromic spike in the soma or the large action potential in recordings from the right connective. The RGC could also be excited synaptically by similar stimulation but conduction was then in an orthodromic direction and was easily recognized from the change of polarity of the components of the conducted spike or from recordings at two points on the right connective.

From observations based on many preparations it is clear that stimulation of any nerve containing a branch of the RGC typically leads to the propagation of an action potential.
potential in its axon in the right connective. It is also apparent from the upper beams of Fig. 6b-d that a spike appears in the right C-Pl whenever this large spike is propagated antidromically along the right connective. It is notable that stimulation of the posterior parapodial nerves excites a fibre which conducts more rapidly into the right connective although it has a smaller spike amplitude (Fig. 6a). The recognition of impulses in a RGC branch within a parapodial nerve was not easy, because of their relatively small amplitude and the presence of many other axons. Once again, however, by superimposing many traces it was possible to distinguish a spike associated with the giant spike in the right connective because of its identical threshold and constant delay with respect to that spike. The threshold relationships are illustrated in Fig. 7. The presence of a small potential on the upper beam in the sweeps in which the RGC axon discharges is indicated by an arrow. This part of the record becomes quite distinct in the suprathreshold recordings, when it is present in every sweep.

The transmission of impulses between the different branches having their origin in the right pleuro-pedal ganglia is not equally easy in all directions. It is readily accomplished in the orthodromic direction, i.e. following electrical stimulation of the right connective. The pathway from the right cerebro-pleural connective to the right connective was clearly a direct one, because, if transmission failed at higher frequencies (10/sec.), the interval between the pre- and post-ganglionic responses remained constant. But increases in conduction times along the RGC axon in the right con-
nective were noted during repetitive stimulation. The pathway from the parapodial nerves to the C-Pl in some preparations showed fatigability and failure to transmit at high frequencies, and in some instances repetitive stimulation was necessary before transmission occurred. Constancy in the time for transmission in either direction suggested the presence of a single pathway without a synapse (unless the synapse was able to conduct equally well in both directions, which is highly improbable). It may be assumed, therefore, that the failure of transmission in an antidromic direction is due to the low safety factor in the branching, especially as the more distant axonal branches are almost certainly smaller than the main axon in the right connective. Evidence for a similar failure to transmit between different regions of *Aplysia* neurones has been obtained by a study of the branches in the neighbourhood of the soma of pleural ganglion cells (Tauc & Hughes, 1961, 1963).

![Fig. 6. Extracellular recordings of the RGC spikes in the right connective (R₁) and right anterior parapodial nerve (R₄) and right C-Pl (R₃). (a), (b), Stimulation of the posterior parapodial nerve (S₁) leads to the excitation of a rapidly conducted small spike at a lower threshold (a) than the RGC spike (b). In (c) the same RGC spikes are excited by stimulation of the anterior parapodial nerve (S₂). (d), Stimulation just at the threshold of the RGC axon in the right C-Pl (S₃). The single RGC spike in the right connective is associated with an extra deflexion in the anterior parapodial nerve (arrow).]
Further studies on the distribution of RGC axons within individual parapodial nerves were made in a few preparations. In these experiments, evidence was obtained for differences between the parapodial nerves. For example, it is almost certain that an RGC axon passes into all the main branches of the posterior and middle parapodial nerves. But, in the case of the anterior parapodial nerve, evidence was obtained for the presence of an RGC axon only in the posterior branches. Fig. 8 is from an experiment in which a branch of the posterior parapodial nerve was stimulated and recording electrodes were placed on the right connective ($R_1$) and on another branch of the posterior parapodial nerve ($R_2$). Superimposed sweeps at threshold and above show the presence of impulses in an RGC axon within all these nerves. Similarly, propagation was also possible between branches of the posterior parapodial nerve and the posterior branch of the anterior parapodial nerve. However, stimulation of the anterior branches of the latter nerve failed to produce an impulse in the giant cell.
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axon at any of the recording sites (Fig. 8, $R_1, R_2$ or $R_3$). But it was possible to show that stimulation of one branch of a nerve led to the blockage of impulse conduction from another branch, partly because of the collision of impulses and also of the refractory period following the propagation of a spike past the branching or the second stimulating site.

(2) The left giant cell (LGC)

During work on an isolated preparation of the whole C.N.S., a particularly large cell was noticed in the left pleural ganglion. Subsequent preparations confirmed that it is a constant feature of this ganglion. The size of the cell is of the same order as the

![Fig. 8. Transmission between the RGC axons in the parapodial nerve branches. A spike also appears in the axon in the right connective in all except (a). (a–f), Stimulation of the main branch of the posterior parapodial nerve ($S_4$) at three different intensities. Recording electrodes on the right connective ($R_1$) and (a–c) fine branch of the posterior parapodial nerve ($R_3$) or (d–f), the posterior branch of the anterior parapodial nerve ($R_3$). (c) and (f) are supra-threshold for the RGC axon, whereas (b) and (e) are just at threshold. The record in (g) is of stimulation to the posterior branch of the anterior parapodial nerve ($S_4$) with recordings in the right connective and fine branch of the right posterior parapodial nerve. Transmission is therefore possible in both directions between these two branches of the two parapodial nerves. And in each case a spike is propagated in the main axon of the RGC.

RGC, i.e. 400–800 μ, and it is usually found close to the point of entry of the left pleuro-visceral connective. In preparations from which intracellular recordings were made it was found that the sheath round the ganglion was difficult to remove without causing the cell to burst. Nevertheless, when preparations were obtained, it was relatively easy to carry out experiments similar to those already described for the RGC.
Path of the LGC axon

This axon appears to branch in a very similar if not identical way to the RGC. However, no evidence has been found for the presence of a branch of the LGC in the left pleuro-visceral connective. Experiments similar to those described for the RGC showed that stimulation of any left parapodial nerve results in the propagation of an impulse to the ipsilateral parapodial nerves and the left C-Pl. That an axon passes through the cerebral ganglia was shown, for instance, by stimulating the pleural ganglion mechanically while recording intracellularly from the LGC soma and in one

Fig. 9. Intracellular recordings (R') of antidromic potentials in the LGC soma following stimulation of the right and left C-Pl (S1, S2), the left anterior parapodial nerve (S3) and the left posterior parapodial nerve (S4).

of the cerebro-pleural connectives; the delay between the somatic spike and the impulse recorded in the right C-Pl was greater than that for the left C-Pl. Again, however, the most convincing demonstration of continuity between different branches was obtained by recording antidromic spikes in the LGC soma following stimulation of the nerves and connectives containing a branch of its axon. Fig. 9 shows such potentials following stimulation of the right and left C-Pl’s, and the anterior and posterior parapodial nerves. Similar potentials were also recorded by stimulating the middle parapodial nerve and the left pedal nerve. No antidromic potentials were recorded when any of the parapodial nerves on the right side of the animal were stimulated. A comparison of the potentials recorded in the soma following stimulation of their branches showed
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a difference between the two giant cells. In the RGC, stimulation of any branch produced identical axonic potentials or A-spikes (Tauc, 1962a) when the cell was hyperpolarized. Here, however, two distinct types of A-spike were produced, depending on the branch of the axon which was stimulated. A-spikes resulting from stimulation of the parapodial nerves were larger than those obtained by stimulating one of the C-PI's. This suggested that impulses from two directions were being blocked at different sites of the neurone in a region close to the soma. Several series of experiments were carried out on this and other cells in order to investigate this possibility in more detail (Tauc & Hughes, 1961, 1963). In the LGC preparation conduction between the two main branches was studied in relation to the potential recorded in the soma. Fig. 10 is

![Fig. 10: Intra-somatic recordings from the LGC (R') following stimulation of the left anterior parapodial nerve and extracellular recording from the right C-Pl (a–c), and the left C-Pl (d–f).](image)

In the records from the right C-Pl notice the presence of a second spike which is not the LGC axon. In both sets of records transmission from the parapodial nerve to the cerebro-pleural connective is possible in the absence of a full invasion of the soma (b, c, e). In other cases where the cell membrane is more fatigued the potential recorded in the soma antidromically is smaller and is not associated with transmission of the spike across the branching (a, d).

from such an experiment following stimulation of the anterior parapodial nerve. External electrodes placed on the right and left C-Pl's indicated when impulses were transmitted between the two branches at the same time as the type of potential within the cell was recorded intracellularly. With the full somatic or S-spike (Tauc, 1962a), the soma was invaded and an impulse was propagated into the cerebro-pleural branch. At other times, either because of fatigue or increased hyperpolarization, no S-spike was present but transmission persisted across the branching. Further fatigue resulted in the antidromic impulse being blocked more distally and only an A-spike of smaller amplitude was recorded, and there was no transmission between the two branches. Of course, whenever a spike was propagated into the left C-Pl it continued into the right C-Pl but with a greater delay.

During studies of this type another phenomenon was observed. A single shock to a parapodial nerve sometimes produces two impulses in the cerebro-pleural connective
(Fig. 11). Apparently it occurs when the delay between the axonic and somatic potentials of the cell is sufficient to allow the intermediate axonal region to recover from its refractory state and to be re-excited by the somatic discharge as found in the RGC (Tauc, 1962a). Under these conditions, a second spike can then propagate along the C-Pl axon shortly after the directly conducted impulse (Fig. 11b).

II. ACTIVITY OF THE GIANT CELLS IN WHOLE-ANIMAL PREPARATIONS

It was difficult to record intracellularly from ganglia of the circumoesophageal complex in these preparations mainly because movements of the buccal mass made it impossible to keep a micro-electrode in position for any length of time. External electrodes on the left C-Pl had previously shown the presence of a very large spike, especially when the head was touched. These spikes may be in the LGC axon but in the absence of intracellular recordings this supposition remains unconfirmed.

The following account is therefore restricted to the RGC.

(i) Spontaneous activity

In many preparations, especially when fresh, the animal makes periodic dorsal movements of the parapodia which are often accompanied by retraction of the head, gill, and reduced mantle cavity (Fig. 12). When an intracellular recording is made from the giant cell during these contractions, repeated discharge may be observed. This response of the RGC is more easily deduced from the presence of a large spike in extra-
Cellular recordings from the right connective. Other units are active during these rhythmic movements, for example the one that propagates more rapidly but has a smaller spike than the RGC axon (Tauc, 1957). The rhythmic movements which normally accompany these bursts occur at frequencies between 0.5 and 1 per minute, but sometimes the rhythm is not regular. This activity is referred to as 'spontaneous' because there is no apparent external stimulus applied at the time when the bursts of

Fig. 12. Pen recording to show the periodic movements of the right parapodium and the associated electrical activity in the right connective. These movements are frequently accompanied by the large spike of the RGC but when this is excited by touching the animal there is no interference with the normal rhythmic movements of the preparation.

Fig. 13. Aplysia, whole-animal preparation. Superimposed recordings from nerves which contain a branch of the RGC axon following mechanical stimulation to the body surface. The time base is triggered by the spike in the right connective, left C-Pl or right C-Pl. These recordings show that impulses are propagated along all branches of the RGC axon following mechanical stimulation applied anywhere on the body surface. In the last two records (h, i) the sweep was triggered by a large spike in the left C-Pl. The conduction of a spike into the left middle and posterior parapodial nerves is shown on the lower beam. Sweep speed is lower in (a), (b), (h) and (f).
impulses and movements take place. In nearly all cases, the RGC is not active unless such movements occur or in response to some peripheral stimulus. Stimulation which causes discharge of the RGC does not necessarily affect the rhythmic spontaneous bursts in other units.

(ii) Mechanical stimulation

In freshly dissected whole-animal preparations mechanical stimuli, wherever applied on the outside of the animal, tend to produce discharges of impulses in the right connective which usually include the RGC spike. These impulses are conducted along all the branches of this cell (Fig. 13). In cases where an action potential is not initiated, either under normal conditions or because of imposed hyperpolarization of the cell, synaptic potentials are almost always recorded by an intracellular electrode in the RGC. Thus, in Fig. 14, touching different parts of the animal produces synaptic and somatic potentials in the cell, but in other cases only synaptic potentials are recorded. The size of the latter can be graded by varying the strength of the mechanical stimulus applied with a brush, either as strokes or jabs. Some adaptation or habituation of this response was observed if the animal was stimulated too frequently.

These observations make it fairly clear that the RGC is influenced by stimulation applied anywhere on the body surface and that a relatively small stimulus is sufficient to evoke firing of the cell, especially when the animal is at rest and has not become habituated. Attempts were made many times to distinguish the movements and mechanical responses of the animal accompanying discharge of the RGC from those when it did not fire, but they did not give any conclusive results. An important feature of the discharge of this cell which was found consistently, however, was that a somatic discharge always preceded the propagation of an action potential forwards along the right connective. This happened regardless of the body region that had been stimulated (Figs. 13, 14) and indicates that the normal functioning of this cell is such that action
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potentials are only set up in the vicinity of the cell body. There is no evidence that there are synapses elsewhere along its path at which impulses can be initiated.

(iii) Electrical stimulation of peripheral nerves

Stimulation of any of the main nerves through external electrodes produced a discharge of the giant cell provided the intensity of the shocks was above a certain threshold. In this case the threshold is determined by the number of axons it is necessary to stimulate in order to have sufficient synaptic input to the RGC. Once again it was apparent that propagation of the spike always occurred from the soma anteriorly in the right connective. The results obtained by mechanical stimulation could be simulated by using electrical shocks suitably graded in strength and frequency. This was especially clear when the giant cell was hyperpolarized, for it was then possible to observe summation effects of the EPSP's. Fig. 14f shows the effect of mechanically stimulating the gill at different intensities, and quite similar recordings were obtained when the strength of an electrical stimulus applied to the branchial nerve was graded. However, the EPSP's produced by higher intensities of electrical stimulation were much larger than it was possible to evoke by mechanical stimuli. Comparable effects were obtained by increasing the frequency of stimulation (Fig. 14g).

(iv) Possible functions of the RGC

The existence of such a large cell, with axons branching into so many nerves, naturally raises the question of its function. One possibility is that it has a function similar to that of giant axon systems in other invertebrates and might therefore be involved in an escape reaction. However, there is no rapid and well-defined motor response of the animal following direct stimulation of the RGC by means of an intracellular electrode within the RGC soma. It seems highly improbable, therefore, that the RGC has such a motor function. Nevertheless, recordings, both intracellular and from the RGC axon in the right connective, have shown many times that the cell does discharge during motor activities of the animal. These activities may be rhythmic movements of the parapodia, or they may result from mechanical stimulation anywhere on the body. The convergence of pathways on to the synapses within the abdominal ganglion is a very characteristic feature of the organization of the RGC even though its function does not appear to be that of a final escape pathway.

The possibility that the cell plays a role by affecting the properties of other ganglion cells along its pathway was investigated in many preparations using the right pleural ganglion. With intracellular electrodes in these cells, the RGC soma was excited either through an intracellular electrode or mechanically by pressing upon it with a small needle. No evidence was obtained for the presence of direct synaptic effects of the RGC on pleural ganglion cells.

DISCUSSION

It has been known for at least 80 years (Vignal, 1883) that Aplysia depilans has nerve cells about 500 µ in diameter. The largest of them, the right giant cell, is found in the abdominal ganglion and the present investigations have indicated how extensive are its ramifications. This cell is activated when the animal is stimulated anywhere on the periphery, but the discharge of the cell does not produce any motor reaction of the
animal and it is therefore not responsible for the protective folding of the parapodia above the dorsal surface. The function of the RGC remains unknown, then, but the evidence is against it having properties analogous to the giant fibre systems of other animals.

The extent of the ramification of the RGC suggests a possible reason for the large size of its soma. The soma of all nerve cells is primarily trophic in function and, in general, the larger the amount of protoplasm which depends upon the soma, the greater is its size. It has been shown (Tauc, 1962 a) that there do not appear to be any synaptic endings on the soma, although the latter plays an important part in synaptic transmission because its electrical properties serve to extend the time course of the synaptic potentials. If this trophic interpretation of the large soma size is correct, then it raises the question of whether any of the other large cells of gastropod ganglia have greatly branched axons. Extensive branching of axons in nerves innervating the foot of Ariolimax was demonstrated electrophysiologically by Turner & Nevius (1951).

The branching of a large cell in the left pleural ganglion has been shown to be almost a mirror image of the RGC. It is remarkable that this cell should have such a similar branching and it again poses problems concerning its functions. For this cell, experiments with whole-animal preparations have not so far been possible and little is known of its synaptic connexions. However, the fact that the two largest cells, one on each side of the c.n.s. of Aplysia, have similar ramifications suggests that they may be homologous. The main difference between them is the ganglion in which they lie. The RGC is found in the complex abdominal ganglion at the point where the connective from the right pleural ganglion enters. The LGC is found in the left pleural ganglion, again very close to the origin of the pleuro-visceral connective. The major difference in the branching of these two cells appears to be the absence of any axon of the LGC in the pleuro-visceral connective, whereas such an axon is readily identifiable on the right side. It is possible that this difference may be a result of the morphogenetic changes which take place during torsion and detorsion. As these processes have not been investigated in great detail in opisthobranchs, and as in this group variations occur in the resulting fusion between originally discrete ganglia (Hoffmann, 1939), some speculations about the nature of the ganglia in Aplysia may be excused.

Eales (1921) has indicated that the typical gastropod nervous system has a visceral nerve loop which connects the two pleural ganglia and along its path are usually two parietal ganglia and a single visceral or abdominal ganglion. 'In Aplysia all these ganglia, with the exception of the left parietal (the infra-intestinal ganglion of Prosobranchs), are present as distinct ganglia. There is evidence that the left parietal ganglion has fused with the unpaired visceral ganglion. The right parietal (supra-intestinal ganglion of Prosobranchs) and the visceral ganglion form an apparent pair owing to the shortenings of the pleuro-visceral connective along which they lie' (Eales, 1921). On this view it is the fate of the left parietal ganglion which is least understood and one possibility not apparently considered so far is that it has become fused with the left pleural ganglion. Such a fusion takes place in other opisthobranchs though usually each parietal ganglion fuses with its ipsilateral pleural ganglion. We may suppose that a giant cell originates in each parietal ganglion and that the left ganglion (or at least the part containing the LGC) becomes fused with the left pleural...
ganglion, whereas the right parietal ganglion (or that part containing the RGC) becomes fused with the single visceral ganglion. One piece of evidence in favour of the fusion of the parietal ganglion with the pleural ganglion, on the left side and not on the right, is the presence of nerves leaving the left pleural ganglion, which are normally absent from pleural ganglia. Otherwise, however, this interpretation remains speculative.

**SUMMARY**

1. On either side of the nervous system in *Aplysia* there is a giant cell (RGC and LGC) whose axon branches within the nervous system. The distribution of these branches has been traced in experiments involving stimulation and recording and the use of intracellular electrodes (in the soma) and extracellular electrodes (on nerves containing the axons).

2. On the right side the RGC sends an axon along the pleuro-visceral connective to the pleural–pedal ganglia where it divides and gives branches to the cerebro-pleural connective and each of the main nerves supplying the foot and parapodium.

3. Stimulation of any nerve containing a branch of this axon produces a large spike in the right connective and an antidromic potential in the soma. Transmission between the different branches is not always easy following antidromic stimulation but is always present in the orthodromic direction whether produced synaptically, by direct stimulation of the soma, or by mechanical pressure applied to the somatic membrane.

4. The LGC soma is in the left pleural ganglion near the origin of the left pleuro-visceral connective. Similar techniques have shown that this cell sends branches to the corresponding nerves on the left side.

5. An hypothesis is suggested to account for the presence of the cell bodies of the RGC and LGC in two different ganglia, despite the similarities in branching of their axons. Possibly differences during torsion and detorsion in the fate of the ganglia in which these cells originate may account for their different locations in the adult.

6. The function of the RGC was investigated in whole-animal preparations. Although it tends to discharge when the animal makes spontaneous protective movements or is touched anywhere on its surface, stimulation of the cell directly through an intracellular electrode gives no overt movements of the animal.

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