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ELECTROTONIC COUPLING BETWEEN IDENTIFIED LARGE CELLS IN THE BUCCAL GANGLIA OF *PLANORBIS CORNEUS*

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INTRODUCTION

Numerous cases of electrotonic coupling between molluscan neurones have recently been discovered (Dorsett, Willows & Hoyle, 1969; Frazier *et al.* 1967; Gardner, 1971; Gorman & Mirolli, 1969; Kandel, 1969; Levitan, Tauc & Segundo, 1970; Tauc, 1969; Waziri, 1969; Willows & Hoyle, 1969). Such coupling sometimes occurs between a number of neurones and may give them the property of selection of inputs that affect many individuals of the group simultaneously and rejection of those that excite only a few (Levitan *et al.* 1970; Willows & Hoyle, 1969). A previous paper (Berry, 1972) described a similar group of small cells in the buccal ganglia of *Planorbis*. The present report is concerned with electrotonic coupling between pairs of cells rather than groups. Three pairs are considered and the nature of their coupling is compared and contrasted.

METHODS

The techniques used were similar to those described earlier (Berry, 1972). The isolated buccal ganglia with their nerves and connectives were pinned to the base of a wax container where they were covered with saline. The cells were clearly visible through the ganglion sheath and were penetrated without its removal. They were recorded from, two at a time, with microelectrodes filled with 2 M potassium acetate having a resistance of 10–50 megohm. Current could be passed through the recording electrode using a bridge circuit. Nerves and connectives were recorded from and stimulated via three suction electrodes. Amplifying, recording and electronic stimulating equipment was of the conventional type.

RESULTS

Structure of the buccal ganglia

There are two buccal ganglia, joined by a commissure, and each communicates with the rest of the central nervous system via a cerebro-buccal (c.b.) connective. They innervate the buccal mass symmetrically and control its feeding movements. They also supply the oesophagus and salivary glands. In each ganglion six large somata have been identified, each with a symmetric partner in the other ganglion. The properties

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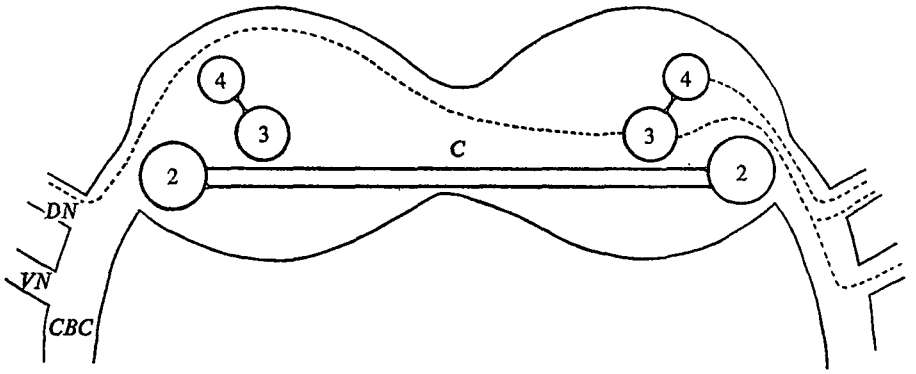

Fig. 1. Schematic diagram of the electrical connexions between large cells in the buccal ganglia. Cell 2 is cross-coupled to its symmetric partner. Cell 3 is coupled to a smaller cell, cell 4, in the same ganglion but neither is directly connected to its partner. Dotted lines show distribution of peripheral axons for the right side only. Two buccal nerve trunks on each side are shown, the dorsal nerve (*DN*) and ventral nerve (*VN*), both of which are large and emerge from the c.b. connective (*CBC*). The cells are on the dorsal surface of the ganglia.

of partner cells are similar and include common synaptic input and symmetric axon distribution.

The following neurones are considered here:

(1) At the periphery of each ganglion where the connectives emerge there is a large, visually identifiable soma; it is usually the second largest in the ganglion and has been numbered cell 2. It is cross-connected electrically to its symmetric partner.

(2) Near the centre of each ganglion is a large soma which is sometimes, but not always, visually distinguishable. Once penetrated it is readily identifiable by properties that will become apparent later. It is not cross-connected to its partner but to a smaller neurone in its own ganglion. It has been numbered cell 3, and the smaller cell, cell 4. Fig. 1 illustrates connections and axon distribution of these cells. The nature of the coupling and its effects on activity are described separately for the two cases.

Coupling between cells 3 and 4

Records were made from cell 3 in intact preparations (buccal ganglia attached to buccal mass), and when stimulated intracellularly its spikes produced muscle twitches in the buccal mass with a 1:1 correspondence; it is therefore regarded as a motoneurone. Cell 4 produces bursts of spikes during rhythmic feeding movements and is probably also a motoneurone, but this has not yet been confirmed (its soma is not very large and is difficult to locate, and it was not found in the few intact preparations that were made).

In the isolated buccal ganglia activity which is similar to that in intact preparations during feeding movements can be elicited by stimulation of a c.b. connective. There is a variable delay and then cell 4 produces a burst of spikes. The spikes can be identified by recording from the ipsilateral dorsal buccal nerve in which the cell has an axon. Most recordings of activity in cell 4 were extracellular because of the difficulty of locating the soma.

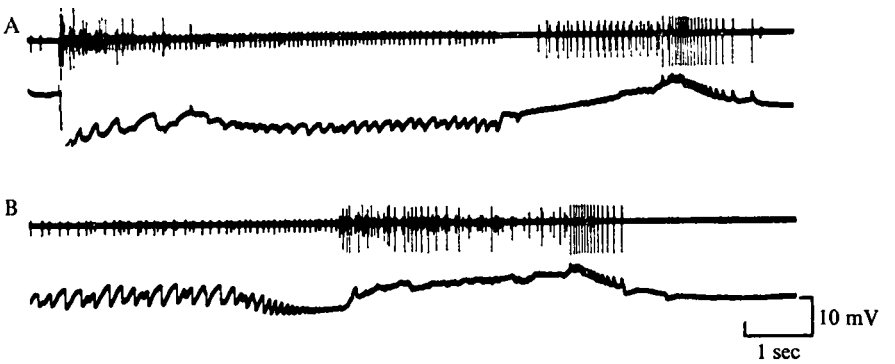

Fig. 2. A. Production of a burst of spikes in cell 4 and concurrent electrotonic EPSPs in cell 3 following stimulation of a c.b. connective. Cell 3, recorded intracellularly on the lower trace, is hyperpolarised on stimulation and is then gradually depolarized until it receives a burst of short-duration EPSPs which have a 1:1 correspondence with the large unit (cell 4) at the end of the dorsal nerve record (upper trace). B. Shows a spontaneous burst; no stimulus was given.

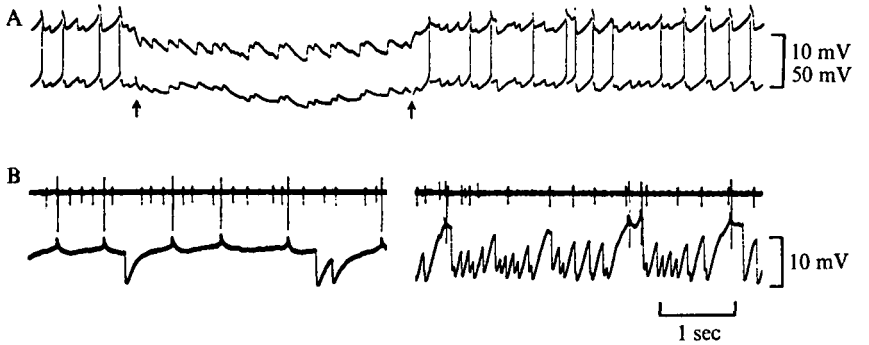

Fig. 3. A. Demonstration of electrotonic coupling between cells 3 and 4. A hyperpolarising pulse in cell 4 (lower trace; bridge unbalanced) is transmitted to cell 3 (upper trace) and causes inversion of IPSPs in both cells. Note that the inhibitory input is similar in both cells but is of greater amplitude in cell 4. B. Although the soma of cell 4 is difficult to locate its extracellularly recorded spikes in the dorsal nerve, even when not in a burst, are readily identifiable by the characteristic, synchronous EPSPs in cell 3.

A. *Demonstration of electrotonic coupling*

The electrotonic coupling was deduced from observations that in cell 3 short-duration EPSPs occurred on a 1:1 basis with an ipsilateral dorsal nerve unit (cell 4) which fired a burst of spikes following stimulation of a c.b. connective (Fig. 2A), and also occasionally in the absence of stimulation (Fig. 2B). In addition to producing bursts the unit sometimes fires spontaneously at a fairly regular low frequency (Fig. 3B). The amplitude of the synchronous EPSPs in cell 3 could not be influenced by altering the membrane potential whereas the rest of the excitatory input increased in size with increased membrane potential. Further, inward current tended to decrease the frequency of the spontaneous short-duration EPSPs, presumably by spreading to cell 4 and hyperpolarizing it. These deductions were confirmed (similar deductions for certain unidentified neurones remain unconfirmed) when the soma

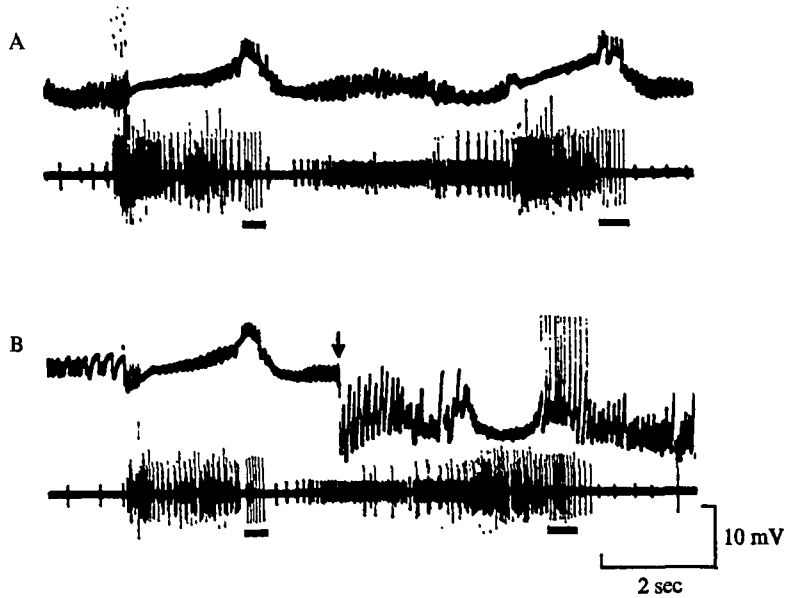

Fig. 4. Subthreshold depolarisation of cell 3 enables it to fire a simultaneous burst of spikes with cell 4. The upper trace is from cell 3 and the lower from the ipsilateral dorsal buccal nerve. Cell 4 spikes in the dorsal nerve record are underlined. Bursts were elicited by 30/sec. stimuli to a c.b. connective. A. Cell 3 receives two bursts of electrotonic EPSPs from cell 4 but as usual does not fire. B. Activity is again elicited but after the first EPSP burst cell 3 is depolarized (arrow; bridge not balanced). Now, during the firing of cell 4, cell 3 fires also, but individual spikes are not synchronous.

of cell 4 was located in the ganglion and recorded simultaneously with cell 3. Fig. 3A shows the transmission of an inward current pulse, causing inversion of IPSPs in both cells.

B. *The effect of coupling on input and output*

The two cells receive similar synaptic input (Fig. 3A) some of which is of greater amplitude in cell 4, possibly indicating that the input is to this neurone alone and is conducted electrotonically to the other. In spite of the coupling and similar PSPs their behaviour differs because cell 4 has a much lower threshold than cell 3. When a c.b. connective is stimulated both neurones become hyperpolarized and receive inhibitory input lasting for several seconds (Fig. 2). They are then depolarized, and cell 4 produces a burst of spikes, but cell 3 fails to reach threshold even though receiving electrotonic EPSPs. In only a few preparations did cell 3 fire, and then only a few spikes.

By directly polarizing cell 3 the effective strength of the coupling can be increased. For example, by depolarizing it close to threshold it can be made to fire simultaneously with cell 4 at very low frequency. It is possible that cell 3 might, in certain circumstances, receive additional excitatory input to take it nearer to threshold. To simulate such additional input cell 3 was depolarized close to threshold and the effect of connective stimulation was again analysed (Fig. 4). This time when cell 4 produced a burst of spikes cell 3 did so too. Perhaps cell 4 usually fires alone to produce a particular movement, whereas if they both fire together a different movement may result.

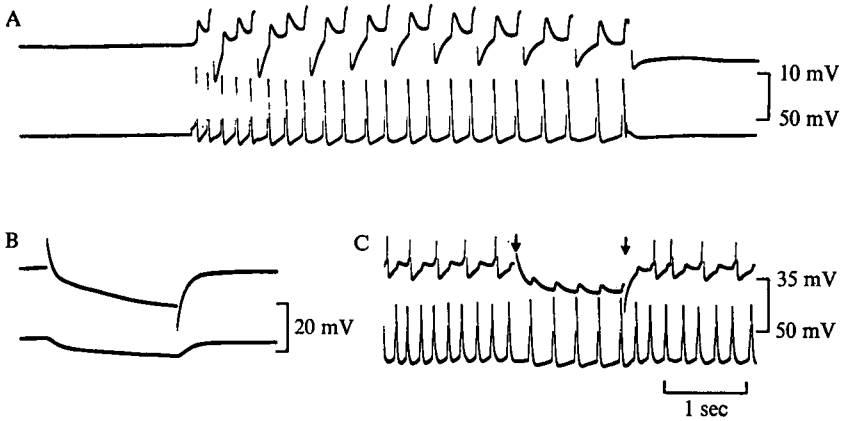

Fig. 5. Demonstration of electrotonic coupling between cell 2 and its symmetric partner. A. A spike in one of the two cells (lower trace) produces an EPSP at constant latency in the other. Spikes were elicited by intracellular depolarisation. B. A direct hyperpolarizing pulse in one cell (upper trace) is transmitted to the other. C. Evidence for the absence of an additional, chemical synapse is obtained by observing that cellular hyperpolarization (arrows) does not markedly increase the size of the EPSP (cf. Fig. 9). Cell 2 recorded on the lower trace was depolarized to make it fire.

Coupling between cell 2 and its symmetric partner

Recordings were made from cell 2 in intact preparations. Intracellular stimulation produced no observable muscle twitches, and no peripheral axons were found. Activity was not co-ordinated with buccal mass movements, or with activity in other recorded neurones apart from the trigger system (Berry, 1972). The cells therefore appear not to be motoneurones or to be involved in feeding or oesophageal movements. They are closely associated with the trigger system, a group of electrically coupled small cells whose activity elicits patterned activity in many large cells. They are driven synaptically by the trigger system but are not coupled electrotonically to it, and were not found to influence other neurones. There is no positive evidence for their function.

A. *Demonstration of electrotonic coupling*

A spike in one of the two cells is always associated with an EPSP at constant latency in the other, indicating direct cross-connexions. The EPSPs are often large enough to reach threshold alone but summation may be necessary (Fig. 5A). The electrotonic nature of the coupling is demonstrated by the transmission to the partner cell of intracellularly applied pulses of either polarity (Fig. 5B). Since hyperpolarization of the cell has little effect on EPSP size (Fig. 5C) this indicates that there is not, in addition, a chemical synapse (cf. Tauc, 1959).

B. *The effect of coupling on spontaneous activity*

In the absence of stimulation the cells are usually either silent or firing regularly at low frequency, the spikes being produced by a pacemaker depolarization (Fig. 6A). After each spike, which has a large undershoot, there is a gradual smooth recovery to

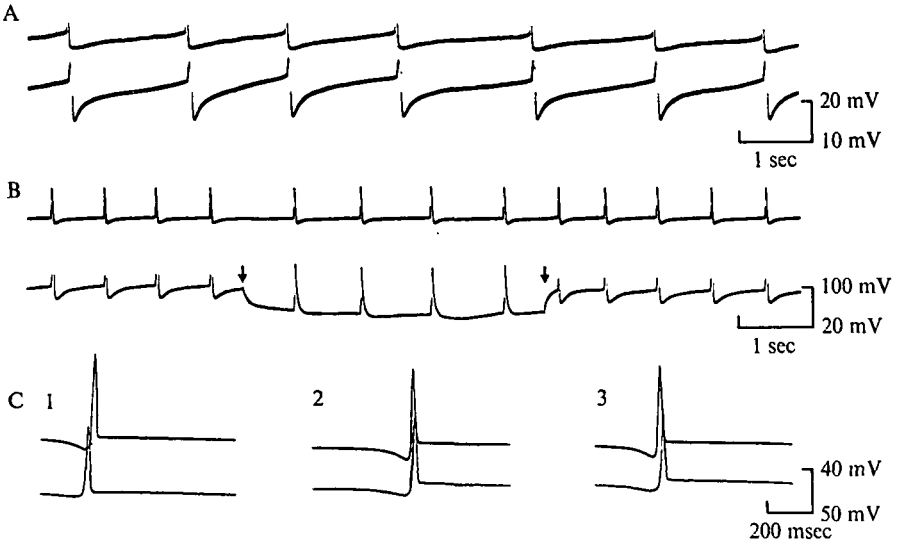

Fig. 6. Spontaneous firing and the near synchrony of spikes in cell 2 and its partner. Both are recorded simultaneously in each case. A. Following each spike there is a pacemaker depolarization which usually reaches threshold in one cell (driver, upper trace) and the discharge directly elicits a spike in the partner cell (follower). B. Hyperpolarization (lower trace, arrows) blocks the soma spike of the follower and is conducted to the driver where it decreases the frequency of spontaneous firing. C. Records read from right to left. Gradual depolarization of the follower (lower trace, depolarization increases from 1 to 3) produces exact synchrony with the driver (C2) and then reverses their roles.

threshold; no EPSPs which might generate the depolarization and spike can be demonstrated by cellular hyperpolarization, and such hyperpolarization reduces the frequency of firing in a regular manner (Fig. 6B).

The cells often fire exactly in phase but usually one reaches threshold first (driver) and the EPSP it produces in its partner (follower) triggers a spike (this spike in turn returns to the driver and produces an EPSP in it, but the EPSP occurs during the refractory period and is ineffective). Neither cell was found to lead habitually, in the preparations studied, and their roles occasionally reversed during one experiment.

The EPSP in the follower summates with its pacemaker depolarization and may not reach threshold if the pacemaker has a slow rise time (the pacemaker depolarizations are not always similar in both cells). Follower cells without a pacemaker often failed to reach threshold at all, even though the partner was firing regularly, and followers with a variable pacemaker tended to fire intermittently. The degree of synchrony of firing in the two cells appears to depend on relative values of threshold and pacemaker slope. It can be altered by polarizing the follower; for example, by gradually depolarizing the follower it could be made to fire exactly in phase with its partner and eventually act as driver (Fig. 6C). It is possible that synaptic input could influence the degree of synchrony in a similar manner (compare the effect of depolarizing cell 3). The cells show a wider variation in the effective strength of their coupling than cells 3 and 4, i.e. in their capabilities for both synchronous and to some extent independent activity.

The EPSPs produced by the firing of the partner cell are large and of long duration compared with those produced mutually by cells 3 and 4 (this partly results from a

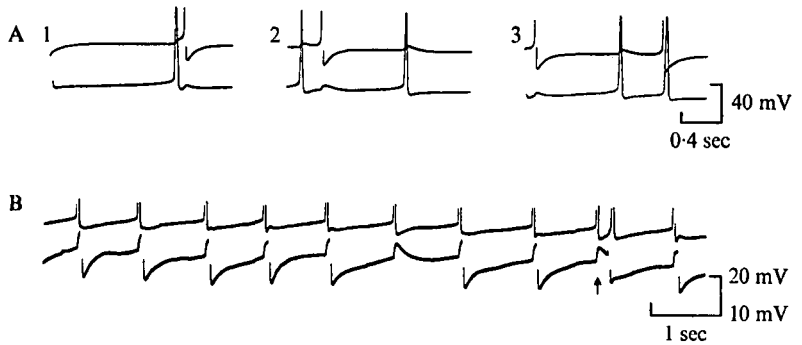

Fig. 7. Reverberation between members of the cell 2 pair. A1. Normally a spike in the driver (lower trace) elicits a spike in the follower (upper trace) which in turn returns to the driver and produces an EPSP. This EPSP occurs during the recovery from the undershoot and cannot reach threshold, so that no reverberation occurs. Occasionally the follower reaches threshold slowly, A2, and if the spike is delayed enough, A3, the EPSP it produces in the driver occurs after the refractory period and can elicit a spike. Unless this spike is similarly delayed the process stops. B. Shows an example of reverberation (arrow) during spontaneous firing. Upper trace is from the driver cell.

longer pre-synaptic spike but is also presumably due to different membrane properties). The EPSPs often generate a spike, and are capable of summation even at low frequency. There is a finite delay between the spike in one cell and the appearance of an EPSP in the other; presumably the EPSP does not have an appreciable size until the spike producing it has traversed the commissure. There are the conditions, therefore, for reverberating activity. This contrasts with cells 3 and 4 where even if the EPSPs were large there could be no reverberation because the cells are so close that the spike and resultant EPSP are almost simultaneous. It may be significant in this respect that the soma of cell 2 is as widely separated from that of its partner as possible. However, in spite of these conditions reverberation rarely occurs partly because of the long spike undershoot; a follower cell spike returning to the driver arrives during the undershoot and cannot reach threshold. Reverberation can occur if the EPSP in the follower reaches threshold slowly so that the follower cell spike returns after the refractory period. Fig. 7 illustrates such a case, but the periods of reverberation were usually small and infrequent.

Synaptic input to cell 2

Neither cell 2 shows much sign of spontaneous synaptic input; no IPSPs were seen but there were occasional small EPSPs (Fig. 8A). These were often synchronous in both cells but also appeared in one alone, indicating some independence of input. They were smaller than EPSPs from the partner but occasionally reached threshold.

Stimulation of a nerve or connective produces a burst of very large EPSPs which depolarize the cells by up to 30 mV but usually produce only a small, low-frequency burst of spikes (Fig. 8B, C). The burst is often followed by several smaller bursts of EPSPs. The spikes produced by the input are considerably reduced in size. The input is synchronous and of similar amplitude in both cells, suggesting that they each receive EPSPs from common interneurones. However, there is the possibility of some

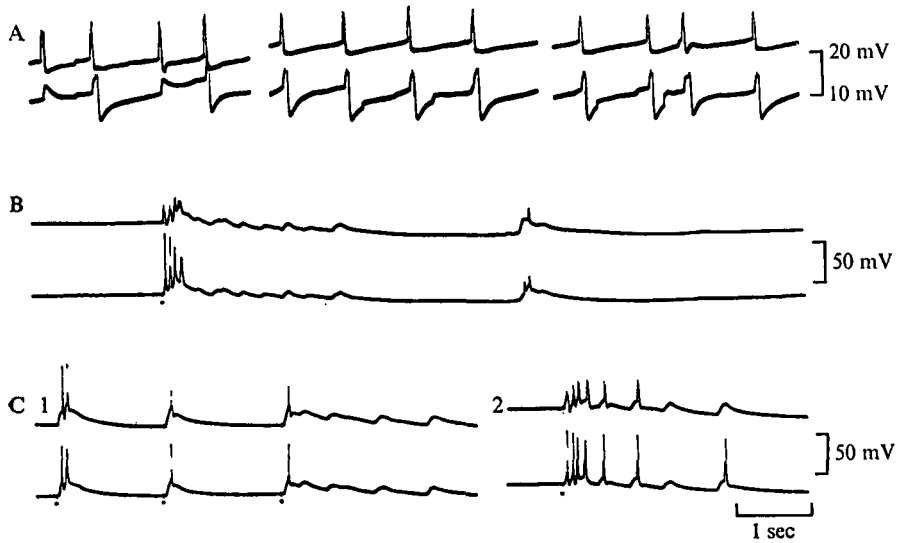

Fig. 8. Synaptic input tó cell 2. The two neurones are recorded simultaneously in each case and stimuli are indicated by dots. A. Spontaneous input is relatively infrequent and consists of small EPSPs which may or may not be synchronous in both cells. B. Stimulation of a connective produces a burst of synchronous EPSPs followed by a smooth hyperpolarization. Stimulation of the buccal nerves produces similar responses; C1 shows stimulation of a ventral nerve, and C2 stimulation of a dorsal nerve.

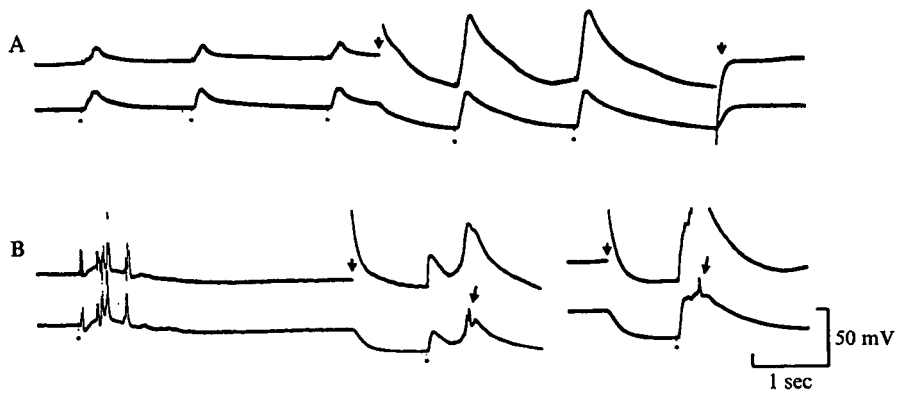

Fig. 9. Large increase in the amplitude of EPSPs in cell 2 during direct hyperpolarization. In each case the two neurones are recorded simultaneously, and compound EPSPs are produced by stimulation of a c.b. connective (stimuli dotted). One cell is directly hyperpolarized (arrows, upper trace; bridge unbalanced) and the partner cell is hyperpolarized to a smaller degree by electrotonic conduction. Note the small spike-like responses (arrowed) in the two records in B.

input to one cell alone being conducted electrotonically to the partner. Following the burst is a long smooth hyperpolarization. In silent cells this lasts for up to several minutes but in spontaneously active cells only a few seconds. It often hyperpolarizes them enough to cause the bursts which follow the initial burst to fail to reach threshold. If the stimulus is repeated there is eventual failure of spikes but small EPSPs remain.

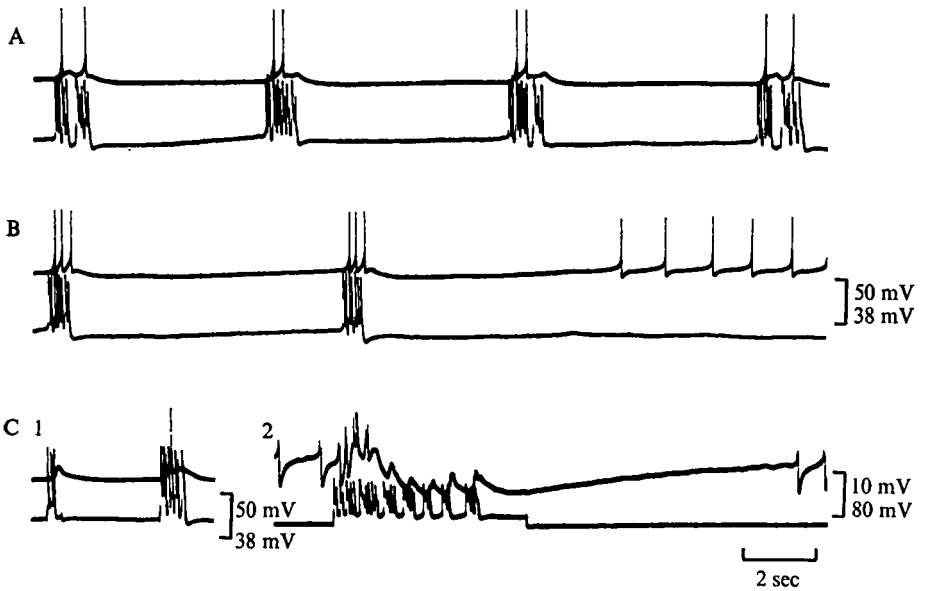

Fig. 10. Excitatory input to cell 2 from the trigger system. In A and B one member of the cell 2 pair is recorded simultaneously with a contralateral trigger cell which was firing spontaneous bursts. Each burst was associated with depolarizations in cell 2, and when they stopped continuous firing was resumed, B. In C the trigger cell is stimulated intracellularly to illustrate that the trigger system is directly responsible for the depolarizations rather than both cells being driven by common input. Two short depolarizing pulses were given in C 1 and a long pulse in C 2.

The nature of the input was investigated by hyperpolarizing the cells (Fig. 9). Large hyperpolarizations produced an enormous increase in the amplitude of the EPSPs which usually depolarized the membrane to the level prior to the hyperpolarization. This is evidence for chemical mediation and possibly also for synchrony of input to both cells, the input to one could summate with that in the other to produce the observed large EPSPs.

The production of a synaptic depolarization by stimulation of any buccal nerve or connective suggested the coupled neurones of the trigger system as origin of the input because of their widespread axon distribution and effects on other cells. Simultaneous records from cell 2 and from a trigger neurone confirmed this, and it was found that spontaneous bursting in the trigger network produced bursts of spikes in cell 2 (Fig. 10). Since the network consists of electrically coupled neurones, and cell 2 and its partner were two of the very few cells which were directly stimulated by trigger neurone activity, it was suspected that they might form part of the trigger system. However, intracellular stimulation of cell 2 could not elicit activity in the trigger system or, apparently, in any other neurone, and applied pulses were not conducted between cell 2 and trigger cells. Further, the input is chemically rather than electrically mediated. Nevertheless, the way in which the two cells are driven by the trigger group is indicative of a close relationship and may suggest some similarity of function.

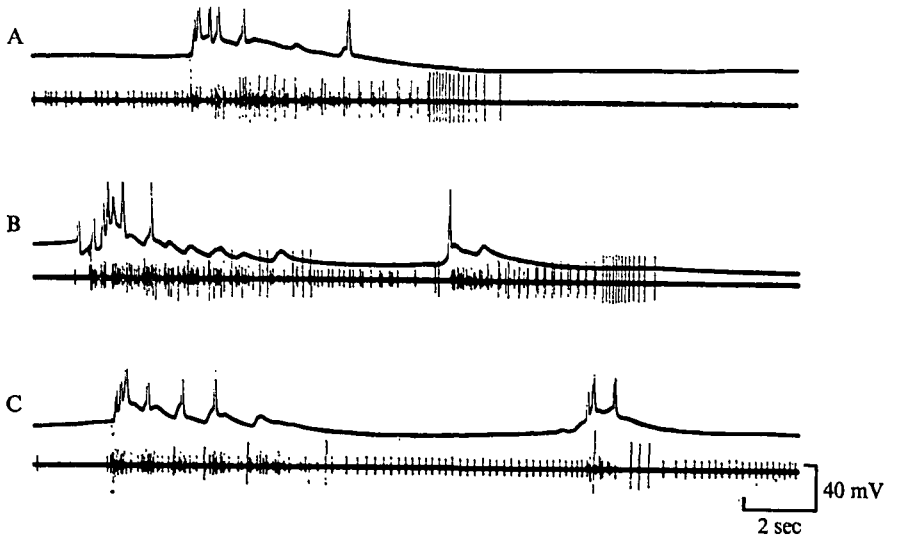

Fig. 11. Relationship of input to cell 2 with the burst of spikes in cell 4 following stimulation of a c.b. connective. Cell 2 is recorded from the ipsilateral dorsal nerve, and activity is elicited by stimulation of the contralateral connective (stimuli dotted). Depolarizations in cell 2 appear related to bursts of small spikes in the dorsal nerve which may be from trigger-cell axons. There is generally no relationship to the firing of cell 4 but C shows the excitation of cell 2 (final burst) and concurrent inhibition of cell 4. Both effects are produced by trigger cell activity. All input to cell 2 immediately following stimulation of a c.b. connective appears to be attributable to the trigger system.

Relationship of input to activity in cells 3 and 4

Cell 2 was recorded simultaneously with the ipsilateral dorsal nerve to determine whether its input is in any way co-ordinated with the elicited sequence in the dorsal nerves, especially the burst in cell 4. As expected from the reponse to trigger system stimulation, there appeared no overall pattern (Fig. 11 A, B) and no relationship to the burst in cell 4. Input is associated with some small units in the dorsal nerve (more apparent in Fig. 11 C) but these may be axons of trigger neurones. Fig. 11 C shows the firing of cell 2 during a fractionated cell 4 burst, both effects being a result of spikes in the trigger system. All input to cell 2 during the elicited dorsal nerve sequence appears merely to reflect activity in this network. Apart from synaptic input directly attributed to the trigger group no relationship was found with input to any other large neurone.

DISCUSSION

Function of the electrotonic coupling

Two questions are asked: (i) Why are the cells coupled – how would they behave if they were not coupled? (2) Why are they coupled electrotonically?

Cell 2 is possibly simpler to understand. The coupling is strong and allows synchronization of spikes; one cell can directly drive the other, and their spikes are synchronous when initiated by input following nerve stimulation. The electrotonic nature of the coupling allows synchronization of pacemakers, which further aids synchrony of spikes and which could not occur with chemical coupling (the fact that

the pacemakers may differ in the two cells indicates that here is synchronization of two separate pacemakers rather than one pacemaker driving both cells).

With regard to synaptic input, the coupling may allow summation between the two cells. The input depolarizes them by up to 30 mV. A comparison with other large cells suggests that the coupling is unlikely to be necessary for synchronization of synaptic input; many unconnected cells produce almost identical output as a result of similar synaptic input.

By contrast, the coupling between cells 3 and 4 has, under the experimental conditions, no obvious function. It is evidently not for the synchronization of spikes because even when cell 3 is depolarized close to threshold and a burst is elicited by connective stimulation, its spikes are not synchronous with those of cell 4. Each neurone can therefore produce its own output, depending on its membrane properties, with little interaction of spikes from the other. In the intact animal the excitatory input is presumably stronger, and able to elicit bursts in cell 3, whereas this does not usually occur in isolated ganglia.

The nature of the coupling between cells 3 and 4 is similar to that between ten large neurones in the buccal ganglion of *Navanax* (Levitan *et al.* 1970), where a spike in one cell produces a fast depolarizing deflexion in the coupled cells. The group has properties which are thought to enable the cells all to fire together when there is sufficient simultaneous input. In *Planorbis* this does not occur; the synaptic input is sufficient to produce a burst of spikes but in only one of the cells. However, in a few preparations unidentified neurones other than cell 3 were found which had EPSPs with a 1:1 correspondence with cell 4 spikes recorded from the dorsal nerve. Cells 3 and 4 might therefore form part of a larger network from which it is necessary to record to explain their observed properties.

Anatomical arrangement of the connexions

The anatomical nature of the connexions described needs to be studied by histological and dye-injection methods, but certain deductions can be made from the intracellular records. For example, for cell 2, among the possible types of connexion are: (1) two independent axons synapsing in the neuropil of each ganglion, (2) a single axon in the commissure with a two-way junction in one ganglion, and (3) two axons making synaptic contact in the commissure. It appears likely that there are two independent axons synapsing in the neuropil because a spike which crosses in the commissure elicits a spike in the partner cell which crosses back. Such two-way conduction might not be expected with a single junction, in which case the spike would reach the partner cell and stop. There is the possibility of two-stage invasion of the soma (see Tauc, 1960) whereby when the spike reaches the cell body the somatic spike triggers another spike in the axon. The recording from the soma, however, generally looks like a conventional EPSP and spike.

In cells 3 and 4 there is no appreciable delay between spike and resultant EPSP because the somata are close together. The observed coupling can be explained either by assuming two independent axons or a single two-way connexion. The latter is depicted for cells 3 and 4 in Fig. 1 to distinguish the observed nature of the coupling from that of the cell 2 pair; it is not meant to imply the anatomical arrangement, merely to emphasize the different functional arrangements of the two cases.

Interpretation of synaptic input

The excitatory input to cell 2 and its partner is presumed to be chemical in nature because of the large increase in amplitude during cellular hyperpolarization. By contrast, the connexion with the partner cell was said to be purely electrotonic because the input from it did not increase in amplitude by any significant amount. However, a small increase could sometimes be observed, but this could be attributed to the spread of hyperpolarization to the pre-synaptic cell increasing the size of the action potentials. The increase in spike height can be seen in Fig. 5, and the spike was no doubt even larger at the synaptic site (see Bennett, 1968, for details of this type of problem).

Some features of electrotonic coupling in the buccal ganglia

There was evidence for electrotonic coupling and cross-coupling between a number of unidentified neurones other than the cells described here and the small cells of the trigger system. In all cases transmission of spikes was poor, resembling that between cells 3 and 4, though it was usually weaker than this and had no obvious function. The electrotonic coupling between cell 2 and its partner is unusual in that there is strong transmission of spikes. Also, it is an example of a direct connexion between symmetric cells. In molluscs the general rule would seem to be that co-ordination between symmetric neurones is effected solely by means of common input from interneurones rather than direct connexions (Gardner, 1971). The other identified large cells in the buccal ganglia of *Planorbis* are not directly connected to their partners but share common inputs. Presumably, large interactions between symmetric neurones could result in an unstable rather than a co-ordinated output.

SUMMARY

The electrotonic coupling between three pairs of large neurones in the buccal ganglia is compared.

1. In both ganglia a large motoneurone (cell 3) is coupled to a smaller, ipsilateral neurone (cell 4) which is probably a motoneurone also. Neither cell is coupled to its symmetric partner. The coupling is quite strong when measured by the transmission of long, intracellularly applied pulses but spikes are considerably attenuated. Resultant EPSPs are of short duration and usually ineffective. Cell 4 produces bursts of spikes but cell 3 is silent unless artificially depolarized close to threshold when it produces a burst of spikes simultaneous with the cell 4 burst; individual spikes are not synchronous.

2. Cell 2, whose function is unknown but which is not involved in feeding, is cross-coupled to its symmetric partner. The coupling is strong, and a spike in one cell produces a large electrotonic EPSP, which often reaches threshold, in the other. One cell may drive the other or they may fire simultaneously owing to synchronous pacemaker depolarizations. Short periods of reverberation occur. All observed input was excitatory and chemically mediated. Following nerve stimulation the input is of similar large amplitude (up to 30 mV) in each cell and the resulting spikes are synchronous. The cells are therefore co-ordinated both by common input and direct connexions.

The former method of co-ordination between symmetric cells is common in the buccal and other ganglia of molluscs, but direct connexions seem to be rare.

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