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INTERACTIONS BETWEEN NEURONES MEDIATING TUFT WITHDRAWAL IN *TRITONIA HOMBERGI*

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SUMMARY

The seven neurones that command the three stages of branchial tuft withdrawal interact by electrotonic and chemically mediated polysynaptic pathways.

The pleural tuft retractors, *L* and *R Pl* 6, make electrotonic synapses with the ipsilateral neurones *Pd* 2, which cause retraction of the tips of the tufts.

The chemically transmitting pathways, between these and other retractor neurones, are mostly reciprocal and can be classified as weak or strong. The former are small in amplitude, with long latencies (1–3 sec) and are labile to repeated activation; the latter are of large amplitude and shorter latency (0·5–0·8 sec), but may still show decrement with repeated use. Frequently the p.s.p. shows indications of 1:1 correlation with the spike pattern in the driven neurone, but the long latencies require the presence of at least one interneurone in the pathway.

The progressive spread of the behavioural response (withdrawal of the tips, complete unilateral withdrawal, complete bilateral withdrawal of all tufts), which occurs with increasing stimulus intensity, is not dependent on a central hierarchy in the activation of the tuft retractor neurones. Reciprocal feedback leads to a general increase in central excitability, the threshold for more extensive responses being probably determined largely by the sensory input to individual neurones.

The unique pleural cell *R Pl* 5 is exceptional, both in the variety of motor activity it commands and in the absence of reciprocal connexions from other retractor neurones.

INTRODUCTION

Numerous examples are now available of excitatory and inhibitory interactions between identified neurones in molluscan ganglia (Hughes & Tauc, 1968; Kandel & Wachtel, 1968; Wachtel & Kandel, 1971; Cottrell, 1971; Gardner & Kandel, 1972; Berry, 1972). In most cases the functional attributes of the neurones involved are unknown or implied, but in others a sequence of unitary interactions has been directly correlated with specific reflex activities recognized as part of the normal behaviour of the animal (Willows & Hoyle, 1969; Kupfermann *et al.* 1970; Levitan, Tauc & Segundo, 1970; Spira & Bennett, 1972).

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In the study of the neural activity correlated with the swimming escape behaviour of *T. diomedia* (Willows, Dorsett & Hoyle, 1973*a*) the close synchrony and alternation observed in the activity of synergistic and antagonistic groups of flexion neurones suggested the existence of strong excitatory and inhibitory interactions within and between them. Despite prolonged searching, evidence for direct connexions in the form of unitary p.s.p.'s was not found, but excitatory and inhibitory influences of a more subtle kind were observed which suggested that weak interactions occurred between individual pairs of neurones at sites remote from the neurone somata, or possibly through the intermediary of other cells (Willows, Dorsett & Hoyle, 1973*b*).

In a previous study of the role of several identifiable neurones in controlling the withdrawal of the branchial tufts of *T. hombergi* stimulating single cells gave responses which frequently could not be fitted to the general rules governing the latency and extent of the muscular field normally controlled by that cell (Dorsett, 1974). Previously (Hoyle & Willows, 1973) it had been suggested that such results may be due to the level of excitability prevailing in the peripheral plexus, which also innervates the tufts and is thought to be interposed between the central neurones and the muscle fibres of the tufts. We have now obtained new evidence of widespread excitatory interactions between all the neurones innervating the tufts of *T. hombergi*, which may to some degree explain variability encountered in these responses.

METHODS

Tritonia hombergi were collected by trawling in 40 m off the Great Orme in Liverpool Bay, and maintained under circulation in the laboratory aquarium. Experimental animals were prepared according to the method described for *T. diomedia* (Willows *et al.* 1973*a*). The animals remained responsive and in good condition for up to 24 h. Experiments on isolated brains were performed by dissecting the brain together with lengths of the nerve trunks free from the animal and pinning it to a wax platform just below the surface of the aquarium. The sea water entering the chamber was at a temperature of 10–12 °C during the experiments. Stimulation and recording from various pairs of cells were made through glass micro-electrodes filled with 3 M-KCl, using conventional recording and stimulating techniques. The coupling factors between pairs of electrically coupled cells were measured using a technique similar to the one described by Brenneke & Lindemann (1971), its application being more fully described elsewhere (Willows & Dorsett, 1974). The abbreviations and grid co-ordinates given in the text refer to the system of reference adopted for *T. diomedia* and *T. hombergi*, and described in previous publications (Willows *et al.* 1973*a*, *b*; Dorsett, 1974).

RESULTS

The branchial tuft retractor system

The co-ordinated withdrawal of the branchial tufts of *T. hombergi* may be accomplished by stimulating any one of seven neurones visible from the dorsal aspect of the central ganglia (Dorsett, 1974). Two pairs of neurones in the pedal ganglia effect ipsilateral withdrawals, *L–R Pd* 1 (Fig. 1) bringing about complete retraction, while *L–R Pd* 2 cause contractions which are restricted to the tips of the pinnae.

The three pleural neurones, *L–R Pl* 6, *R Pl* 5, mediate bilateral responses, as-

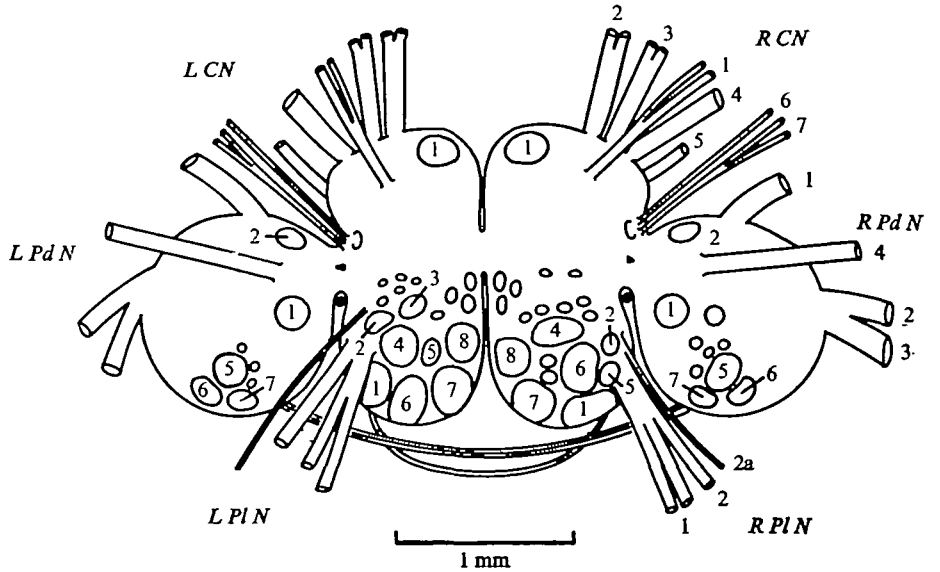

Fig. 1. Diagram of the brain and principal identifiable neurones of *T. hombergi*.

might be expected from their wider axon distribution to nerve trunks on both sides of the body. *L Pl* 6 and *R Pl* 5 both cause complete bilateral tuft withdrawal, whereas the response to stimulating *R Pl* 6 is more variable, but normally produces contractions of the tips of the tufts affecting both sides of the body. Simultaneous intracellular recordings from pairs of tuft retractor neurones indicate a high proportion of common excitatory synaptic inputs, particularly from mechanoreceptors and chemoreceptors widely distributed over the mantle surface. The several categories of integrated response shown by the tufts, in addition to purely local reflexes, suggest a hierarchy in the intensity thresholds required to activate the central retractor neurones. In such circumstances, in addition to common inputs, it would be reasonable to expect some kind of information feedback between the retractor neurones themselves.

Electrotonic junctions between tuft retractor neurones

If *L Pd* 2 and *L Pl* 6 are penetrated by independently mounted micro-electrodes and their activity is recorded simultaneously, it is noted that an action potential in either neurone is accompanied by a post-synaptic potential in the other cell. A depolarizing current, applied to *L Pl* 6 through the micro-electrode and of sufficient intensity to generate a series of action potentials, is accompanied by slight depolarization and a parallel series of p.s.p.s in *L Pd* 2 (Fig. 2A). A hyperpolarizing current applied to *L Pl* 6 produces simultaneous hyperpolarization of *L Pd* 2. Similar results are obtained from the reverse experiment, by passing current through the electrode inserted in *L Pd* 2 and recording the effect in *L Pl* 6 (Fig. 2B).

As the post-synaptic potentials (*a*) are 1:1, (*b*) show no appreciable delay, and (*c*) do not fatigue with repetition, and as hyper- and depolarizing currents are equally transmitted, it is presumed that the neurones are electrotonically coupled.

An exactly similar relationship is found between *R Pl* 6 and *R Pd* 2 in the opposite ganglia (Fig. 2C, D). Measurements of the coupling factor between these pairs

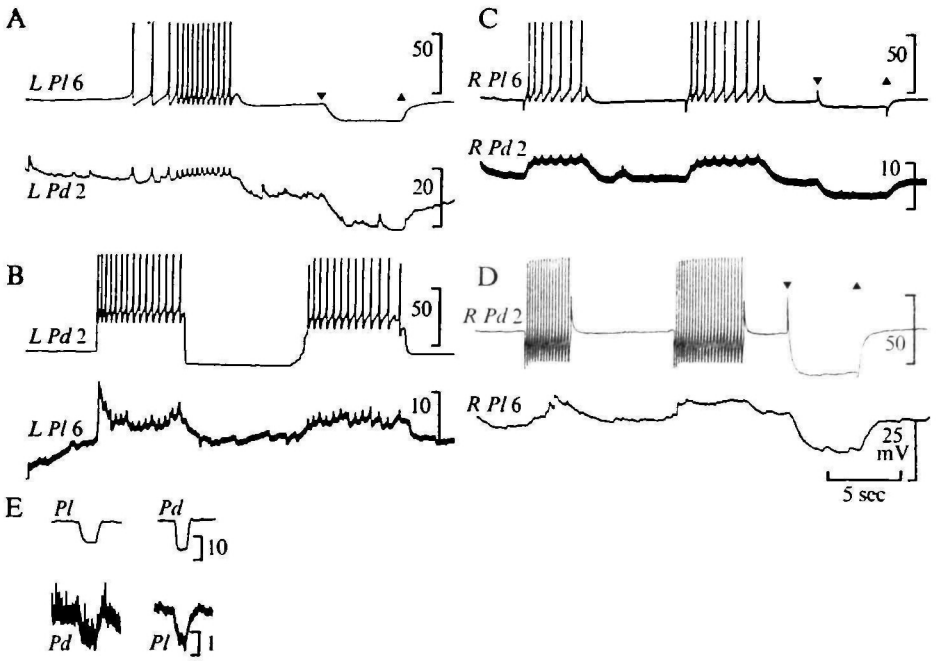

Fig. 2. Electrotonic junctions between tuft retractor neurones. (A) *L Pl* 6 and *L Pd* 2. A depolarizing current pulse causes a burst of impulses in *L Pl* 6, each accompanied by a simultaneous p.s.p. in *L Pd* 2. A hyperpolarizing pulse (arrowed) is also transmitted across the junction. (B) The experimental situation reversed by stimulating *L Pd* 2 and recording from *L Pl* 6. (C) Coupling between *R Pl* 6 and *R Pd* 2. (D) Coupling in the reverse direction. The switching artefacts and displacement of membrane potentials in the stimulated cells are due to imbalance in the bridge circuit used for passing current. (E) Extract from records from *L Pl* 6 and *L Pd* 2 used for measuring coupling factors via a single electrode in each cell.

(Fig. 2E) made following the method described by Willows & Dorsett (1974), gave values of 0·13 (*L Pd* 2–*L Pl* 6), 0·12 (*L Pl* 6–*L Pd* 2), 0·1 (*R Pd* 2–*R Pl* 6), 0·11 (*R Pl* 6–*R Pd* 2). These differences are sufficiently small to be accounted for by experimental error, and it appears that no rectification takes place in the junctional membrane. Some estimate of the influence of coupling on the interaction of the two neurones was obtained from two preparations in which *R Pd* 2 was showing a regular iteration. *R Pl* 6 was made to fire a burst of impulses by intracellular depolarization and an analysis of variance was made on the inter-pulse interval in *R Pd* 2, before and during the activity in *R Pl* 6. A 10 sec burst at 1·6 Hz produced no significant alteration in the firing rate of *R Pd* 2, but a burst of similar duration at 2·4 Hz produced a highly significant increase (*F* test, *p* better than 1 %). No electrotonic junctions have been found between any other tuft retractor neurones.

Synaptic interactions of pedal tuft retractor neurones

The use of similar stimulating and recording techniques has provided evidence of reciprocal excitatory pathways between *L Pd* 1 and *R Pd* 1 (Fig. 3A, B). Intracellular current stimulation of one member of the pair, sufficient to produce a 2–4 sec burst of impulses at about 6 Hz, reliably evokes a compound e.p.s.p. of 5–15 mV amplitude in the post-synaptic partner. The long latency of the response, which varies between

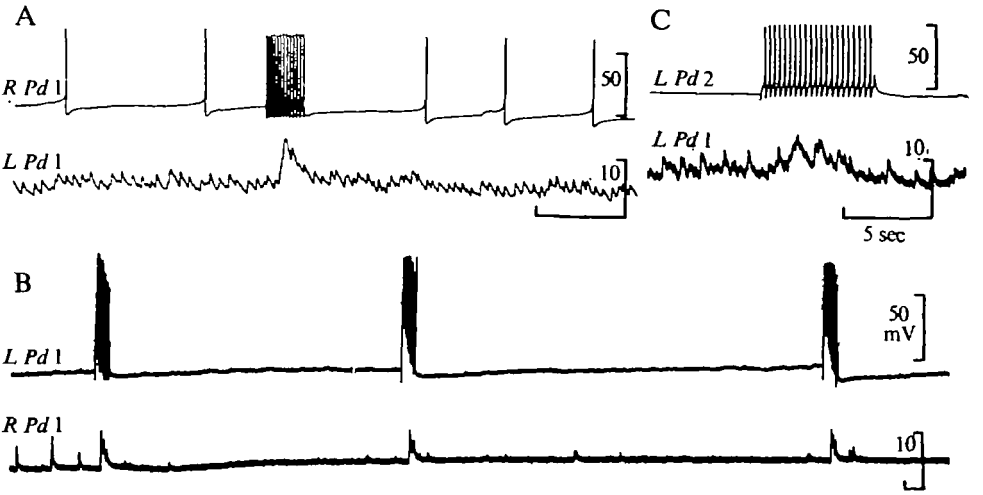

Fig. 3. Interactions between tuft retractor neurones in the pedal ganglia of an isolated brain. (A) A depolarizing pulse in *R Pd* 1 causes a burst of spikes followed by a compound p.s.p. in *L Pd* 1. Latency 7–800 msec. 1 *Pd* is hyperpolarized to prevent spiking. (B) The reverse experimental showing three responses in *R Pd* 1 to stimulation of *L Pd* 1. At this interval there is a slight increase in the delay in the later responses, but no decrease in amplitude. (C) Interaction between *L Pl* 2 and *L Pd* 1. *L Pd* 1 is hyperpolarized to stop spiking, which enhances the e.p.s.p.

0·6 and 2 sec, and the inability to resolve individual e.p.s.p.s suggest that the pathway is not monosynaptic and may involve at least one intermediate neurone.

A characteristic feature of many interactions between the central neurones is their relative lability with repeated use. Activation of this pathway at intervals of 1–2 min produces an increase in response latency from 2 to 4 sec (Fig. 3 B), while activation at intervals of 10–15 sec leads to a rapid failure in transmission, which then returns after a period of rest.

The post-synaptic response is easily masked by high levels of ongoing synaptic activity in the neurone which come from unidentified sources in the ganglion and often lead to irregular spike activity. It is more easily observed, and its amplitude often enhanced, during periods when the synaptic drive has subsided or spiking is prevented by moderate hyperpolarization of the post-synaptic cell membrane.

A much weaker and more labile interconnexion has been noted between *L Pd* 2 and *L Pd* 1, but in this instance evidence of transmission in the reverse direction has not been obtained (Fig. 3 C). The latency, judged as the interval between the onset of the stimulus and the point where the membrane potential of the post-synaptic neurone first rises above that due to previous ongoing activity, is about 1–2 sec. This is exceptionally long for two such closely apposed neurones and the pathway is probably polysynaptic.

The possibility of the delayed response being due to afferent proprioceptive input from the peripheral movements generated by the stimulated neurone has been precluded by recording identical potentials from completely isolated brains.

It has not yet proved possible to confirm a similar relationship between the homologous neurones of the right ganglion as *R Pl* 1 often occurs in the *R Pl* ganglion in a position where access for recording is difficult.

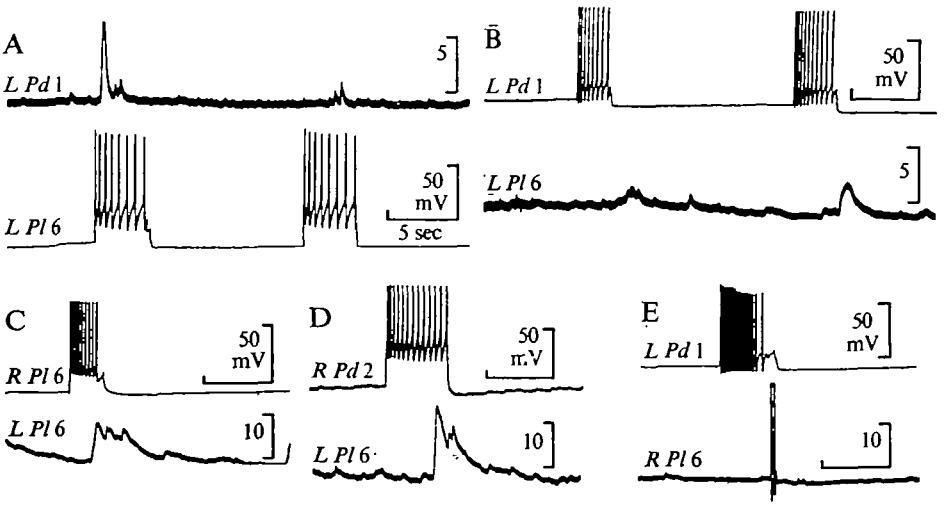

Fig. 4. Interactions between pleural and pedal tuft retractor neurones. (A) Successive responses of *L Pd* 1 showing adaptation to bursts in *L Pl* 6. The initial response is a large depolarization followed by two smaller p.s.p.s. The response to the second burst 11 sec later is smaller, but three component p.s.p.s can still be recognized. The latency increases from 0·5 to 2 sec. Note each compound potential is of a shorter duration than the burst in the driven neurone. (B) The reverse pathway is much weaker, the p.s.p. having a latency of 2–3 sec. (C) The weak excitatory pathway from *R Pl* 6 to *L Pl* 6. The p.s.p. amplitude is enhanced by hyperpolarization, but continues after the termination of the burst in *R Pl* 6. (D) Excitation in *L Pl* 6 by impulses in *R Pd* 2. There is no electrical coupling as with the ipsilateral pairs of these neurones. (E) Response of *R Pl* 6 to stimulation of *L Pd* 1 at normal resting potential. Two action potentials are generated in the pleural neurone at the termination of the burst.

Interaction of pleural and pedal retractor neurones

Stimulation of the bilateral tuft retractor *L Pl* 6 evokes a strong excitatory response in the ipsilateral retractor neurone *L Pd* 1, the amplitude of which depends on the previous spike activity in the driven neurone. A burst of spikes in *L Pl* 6 following several minutes of inactivity produces a large compound p.s.p. in *L Pd* 1 followed by one or two smaller and possibly unitary potentials (Fig. 4A). A second burst of similar duration some 10 sec after the first evokes a group of smaller unitary-type responses after a longer latency. The post-synaptic events in *L Pd* 1 terminate before the completion of the driven burst in *L Pl* 6, suggesting both a rapid and long-lasting adaptation at some point in the intervening pathway.

A weaker, reciprocal pathway is found between *L Pd* 1 and *L Pl* 6 (Fig. 4B), but the latency of 2–3 sec is much longer than in the reverse direction. *L Pd* 1 also makes excitatory connexions with *R Pl* 6, and in preparations where the ongoing synaptic drive to this neuron is sufficiently low to allow the response to be recorded at normal values of the membrane potential, it is potent enough to generate one or two impulses in the pleural cell (Fig. 4E).

Excitatory polysynaptic pathways of comparable efficacy and latency exist from *R Pd* 2 to *L Pl* 6 (Fig. 4D) and from *R Pl* 6 to *L Pl* 6 (Fig. 4C), in the latter case the post-synaptic response lasting for several seconds after termination of the burst in the former neurone.

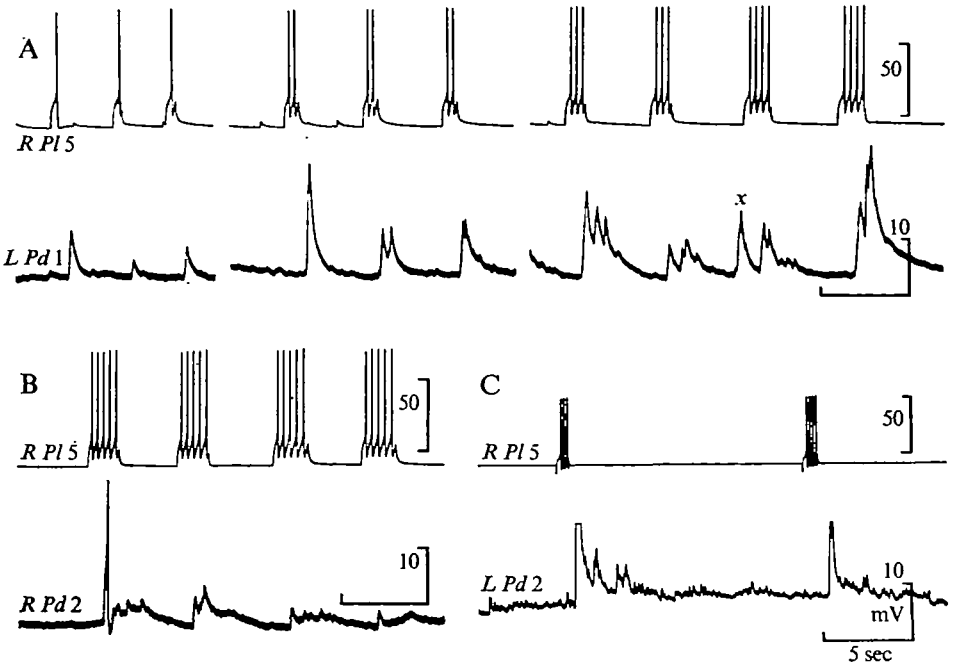

Fig. 5. Interactions of *R Pl* 5. (A) Responses of *L Pd* 1 to different numbers of impulses. Note the first response to each group of spikes is generally larger than succeeding ones. The p.s.p.s can be resolved into component units which correspond in number to impulses in *R Pl* 5. The amplitude of the p.s.p. increases with the number of impulses in the burst. *x* marks a spontaneous potential. (B) Response of *R Pd* 2 to a sequence of bursts in *R Pl* 5. The amplitude of the p.s.p. diminishes with each following stimulus, but the latency remains constant at 600 msec. Despite hyperpolarization of *R Pd* 2 the intensity of the input from the first burst is sufficient to generate a spike. The unitary p.s.p.s resolved within each response have a general correlation with spikes in *R Pl* 5. (C) Interaction of *R Pl* 5 and *L Pd* 2. Note the prolonged synaptic input outlasting the short stimulus.

Interactions of R Pl 5

This neurone commands activity in a number of muscle groups controlling extension and 'squaring off' of the body, branchial tuft and rhinophore withdrawal, raising and lateral spreading of the oral veil and dorsal curling of the foot margins. The neurone is not implicated in the swimming behaviour and is relatively insensitive to salt crystals dropped on the mantle.

Considering the diversity of its motor field, it is not surprising to find that *R Pl* 5 establishes excitatory, and in one case inhibitory, connections with a variety of pleural and pedal neurones. It evokes an excitatory response in a number of the smaller neurones in areas *G* 1–2 of the right pleural ganglion, and an inhibitory potential in a group of pleural neurones adjacent to the commissure which are particularly sensitive to drops of water on the surface of the experimental chamber.

R Pl 5 provokes very strong excitatory responses in all the neurones forming the branchial tuft retractor system. The consistency of these interactions allows some quantitative assessment to be made of its post-synaptic effect on the other cells. The response of *L Pd* 1 to a series of 1–4 impulses in *R Pl* 5 illustrate some of the general properties of this pathway (Fig. 5A). Single impulses each generate a p.s.p. of variable

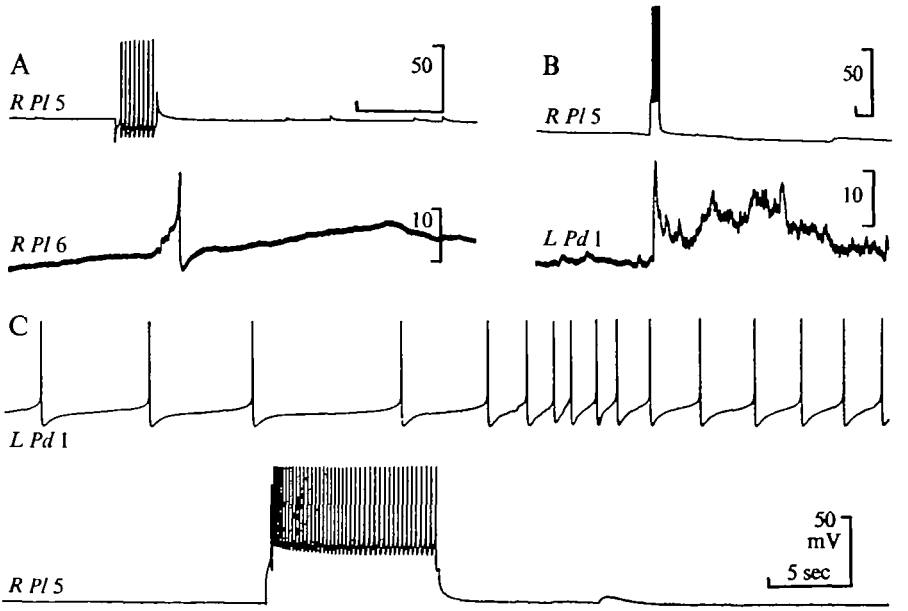

Fig. 6. Interactions of *R Pl* 5 continued. (A) The excitatory pathway to *R Pl* 6 is sufficiently strong to generate spike in the lightly hyperpolarized soma. (B) A 2 sec burst in *R Pl* 5 results in 60 sec of intense synaptic activity in *L Pd* 1. (C) Interaction with *L Pd* 1 at normal resting potential. The burst interrupts the slow iteration, but is followed by a delayed, synaptically driven excitation which increases the firing rate.

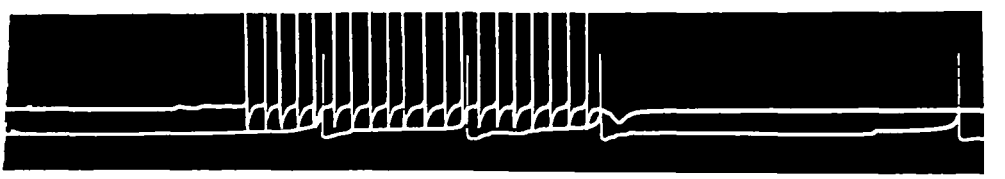

Fig. 7. Excitation of *L Pl* 6 by *R Pl* 5 at normal resting potential. Some evidence of synaptic input can be discerned in *L Pl* 6.

amplitude, but of constant latency (800 msec). The first of a series of paired spikes evokes a large summated p.s.p., but succeeding pairs give smaller unitary responses to each spike of the pair.

Increasing the number of spikes in each burst to 3 or 4 increases the amplitude of the post-synaptic response, and peaks corresponding in number to the individual action potentials of the driven neurone can be clearly distinguished. The latency of the p.s.p. remains constant at about 800 msec. Although the long latency and variability in the amplitude of the response suggest the pathway is not monosynaptic, the input–output relationship clearly approximates to unity.

The interaction with *R Pd* 2 appears to be one-way from the pleural to the pedal neurone, and provides clear evidence of the decrement shown by the post-synaptic unit to successive bursts of impulses in the driven cell (Fig. 5 B). Despite the decline in the amplitude of the response, the latency remains constant (600 msec) and there is again some indication that the number of unitary potentials corresponds to spike

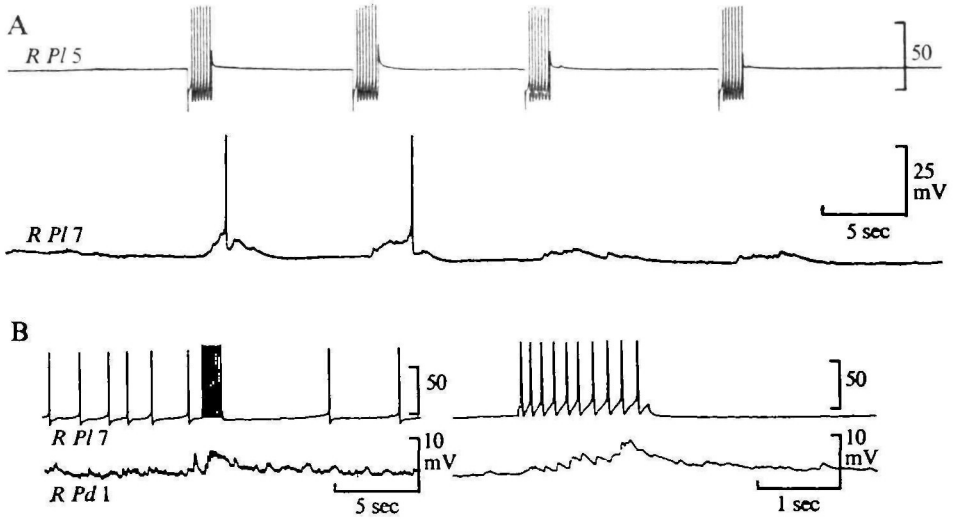

Fig. 8. The indirect excitatory pathway from *R Pl* 5 to *R Pd* 1. (A) *R Pl* 5 makes strong excitatory connexions with *R Pl* 7. Note the decrement in the response with repeated activation. (B) Excitation of *R Pd* 1 by *R Pl* 7. The very short latency and the close correspondence of spikes to p.s.p.s suggests that this pathway may be monosynaptic, but the expanded time base in the latter half of the record reveals small discrepancies which make this uncertain.

in *R Pl* 5. A similar relationship is found with *L Pl* 2, the homologous cell on the left side.

A further example of the interaction of *R Pl* 5 with *L Pd* 1 is shown in Fig. 6B, C. A short burst of six spikes in the former induces an abrupt depolarization of the pedal cell followed by a period of intense synaptic bombardment lasting nearly 60 sec, as the membrane potential returns slowly to its original level. In this record, the membrane of *L Pd* 1 has been slightly hyperpolarized to prevent it firing. At normal values of the membrane potential, a 10 sec burst in *R Pl* 5 results in a pause in the iterative activity of *L Pd* 1, which is followed by an acceleration of the firing rate due to increased synaptic input as the burst terminates (Fig. 6C).

The excitatory input resulting from activity in *R Pl* 5 is sufficiently strong to evoke action potentials in the large pleural tuft retractor neurones *L–R Pl* 6 (Figs. 6A, 7).

Evidence has yet to be obtained of direct interconnexions between *R Pl* 5 and *R Pd* 1, corresponding to that of its homologue on the left side. Records have been obtained which suggest a more indirect pathway. *R Pl* 5 has a strong excitatory influence on *R Pl* 7 (Fig. 8A) which again shows the characteristic decline in amplitude with repeated use previously noted with *L–R Pd* 2. Although no specific function is known for *R Pl* 7, it in turn produces a strong excitatory response in *R Pd* 1 (Fig. 8B). The record suggests that the response in *R Pd* 1 might be both monosynaptic and unitary but close examination shows that the 1:1 correspondence is not sufficiently precise for this to be certain. However *R Pl* 5 is capable of influencing *R Pd* 1 through its action on *R Pl* 7.

DISCUSSION

This first report of extensive synaptic interactions between neurones in the central ganglia of *Tritonia* describes events of an increased order of complexity when compared to the monosynaptic and unitary potentials typically recorded from the neurones

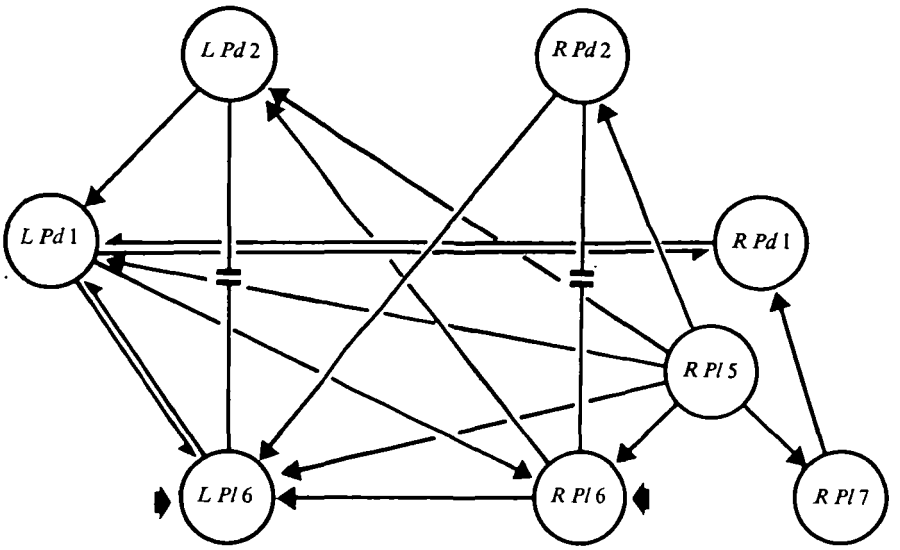

Fig. 9. Synoptic chart of the interactions of the tuft withdrawal neurones. Where pathways are uni-directional the reverse connexion has proved negative when tested. Not all possible combinations have yet been studied. With the exception of *R Pl* 5, which receives no inputs from other tuft retractors, the interactions between the partial, unilateral and bilateral withdrawal units are mostly reciprocal, and not polarized in the direction of the lesser to the greater response.

of the abdominal ganglion of *Aplysia* (Tauc, 1959; Kandel, Frazier & Wachtel, 1969; Kupfermann *et al.* 1970). With the exception of the electrically coupled neurones, none of the post-synaptic responses recorded between the neurones of the tuft withdrawal system had latencies of less than 500 msec, the majority showing delays of between 1–3 sec. An average figure for the time taken for an action potential in one of the large pleural cells to cross the brain and emerge in a pleural nerve trunk on the opposite side might be 15–20 msec (Dorsett 1967), which must preclude any possibility of the connections described here being monosynaptic.

Many features of the interactions become explicable if interneurones occur between the driven and the recorded cells. It is unusual for single e.p.s.p.s to generate action potentials in molluscan neurones, so any of the depressive or facilitatory properties of their synapses (see Tauc, 1966; Dorsett 1974, for reviews) may account for the long latencies and various input–output relationships observed in different pairs. The point is best made by reference to specific examples. The interactions between the pedal neurones and between *L Pl* 6 and *L Pd* 1 (Figs. 3, 4A) can be classified as weak in two ways. First, the duration of the post-synaptic response represents a fraction of the length of the burst in the driven neurone, presumably by a rapid adaptation in the intervening pathway. Secondly, repeated activation leads to a decline in the amplitude and an increase in the latency of succeeding responses, which may indicate the operation of more long-term anti-facilitatory effects.

In other instances, particularly in the strong interactions of *R Pl* 5 with the pedal neurones, relatively short bursts provoke prolonged synaptic input to the recorded cell due to the concurrent activation of convergent pathways to the same neurone. The initial latency is short and it remains constant with a variety of stimulus pro

grammes. Characteristically, it is the strong interactions which seem more direct and show evidence of 1:1 matching of p.s.p.s to spike patterns in the driven cell.

It is difficult to find a satisfactory explanation for the ipsilateral electrical coupling between neurones *Pd* 2–*Pl* 6 on the right and left sides, when the cross-coupling and pathways to the neurone *Pd* 1 from the pleural neurones are effected by chemical transmission. Electrical coupling can lead to synchronous output, reciprocal excitation and, in some circumstances, spike suppression (Bennett, 1968; Getting & Willows, 1973), but none of these effects seems relevant to this specific pairing.

A synoptic diagram of the synaptic and resistive pathways between the tuft retractor neurones (Fig. 9) indicates that the interaction between neurones responsible for partial, unilateral and bilateral responses is almost entirely reciprocal. The naturally observed progression from the simpler to the more extensive behavioural responses must depend upon individual thresholds to stimulus intensity accompanied by a general build-up in central excitation, rather than a central hierarchy in the order of recruitment, obtained by polarization of the pathways between retractor neurones.

The inputs from the flexion neurones in the pedal ganglia to the bilateral retractors *R–L Pd* 6 (Willows & Dorsett, 1974) provided a link between this system and the swimming behaviour, when the tufts are normally held retracted to reduce the drag on the body as it moves through the water. Alone among the retractor neurones, *R Pl* 5 apparently receives no inputs from the neurones with which it interacts so strongly, emphasizing the special but so far undiscovered role of this neurone in the biology of the animal.

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