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RESPONSE HETEROGENEITY IN ADDUCTOR MUSCLE EFFERENTS OF THE SURF CLAM*

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Neuromuscular control in the Mollusca presents numerous interesting problems to the comparative physiologist. Among these are the extent of polyneural and multiterminal innervation, the possibility of peripheral inhibition, and the anatomical basis for wholly peripheral reflex activities. Perhaps in no area are the problems more intriguing—and the experimental results more equivocal—than with regard to the physiology of lamellibranch tonic smooth muscle. These muscles are unique, not only for the fact that they are able to exert the largest known force for weight of muscle tissue, but also for their well-known ability to remain in the contracted state for prolonged periods of time with very little expenditure of metabolic energy (for a review, see Hoyle, 1964). While these muscles can also contract in a phasic manner, relaxing quickly after a brief twitch-like response, in some cases there is no histological differentiation to complement the functional dichotomy. It is therefore logical to inquire whether the different states of activity may have their bases in different types of excitatory synapses made with the muscle fibres by specific 'tonic' and 'phasic' motoneurons. Another suggestion (Takahashi, 1960) is that phasic responses occur following simultaneous activity in excitatory and inhibitory nerve fibres, the latter drastically limiting the duration of the contracted state of the muscle.

It seems apparent that decisive experiments with lamellibranch nerve-muscle preparations must eventually include single-unit electrical recordings, both from cellular elements within the central nervous system and from the muscle fibres themselves. The latter especially will be needed to determine the functional nature of various efferent fibres which supply the muscles, while recordings from ganglion cells within the central nervous system must be taken to establish the reflex control of the different efferent fibres by various input pathways, and, especially, to permit controlled electrical access to individual neurons, so that events at the neuromuscular junctions can be precisely analysed.

In the present paper a description is given of electrical responses from the two known populations of efferent neurons supplying a tonic smooth muscle, the posterior adductor of the surf clam. The functional connexions made by these ganglion cells at the periphery have not yet been established; however, their input organization is interesting in its complexity and may itself provide answers to some fundamental questions regarding the relationship between structure and function in the molluscan nervous system.

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MATERIALS AND METHODS

Medium to large specimens of the surf clam, *Spisula solidissima*, were used. All recordings were confined to the visceroparietal ganglion, which was removed from the animal and placed in artificial sea water at a controlled temperature. Electrical stimulation of most nerve trunks was accomplished by pairs of fine platinum wires. However, when working with the small short nerves supplying the posterior adductor muscle, suction electrodes were a necessity. These devices were of simple construction, and amounted to little more than a piece of flexible plastic tubing which connected a 5 c.c. syringe to a short glass pipette. The tip of the latter was drawn out and fire-polished to fit the nerve, 2–3 mm. of which was pulled up into the pipette, and electrical contact was completed through a short column of sea water between the nerve and a silver lead inserted into the plastic tube. Electrical pulses 1·0 msec. in duration were applied between this lead and another placed in the bathing solution.

Intracellular recording techniques were used as described previously (Mellon, 1967). These included the use of glass micropipettes filled with 3 M-KCl and having electrical resistances of 20–50 MΩ. On occasion, two preamplifiers were used to record simultaneously from neighbouring cells; under these conditions, some capacitative coupling between the input circuits was evident in the electrical records. However, no physiological electrical coupling was observed between the cells of either type described below. Additional information concerning recording and stimulating methods employed in these studies are inserted at appropriate places in the Results section below.

RESULTS

(1) *Anatomical*

The anatomy of the visceroparietal ganglion has been previously described (Mellon, 1965). The nerves to the posterior adductor muscle emerge on each side of the dorsal surface of the ganglion, and after giving off a branch to the dorsal region of the animal each enters the muscle through a cleft in its ventral aspect. By cutting the nerve at this point a length of 3 or 4 mm. was available peripheral to the major branch point for purposes of stimulation. Exclusion of the dorsal branch was absolutely necessary to obtain pure input characteristics. Recordings were confined to the dorsal surface of the large paired anterior lobes of the ganglion, and only cell bodies in the peripheral layer were penetrated. In the majority of more than 200 units examined cells of the kind designated type I (described below) were identified only at the surface of the ganglion, and type II neurons were usually located 50–100 mμ deeper.

(2) *Electrical*

(A) *Type I cells*

Responses to volleys. A description and analysis of some of the response characteristics of these cells has been published previously (Mellon, 1967). Volleys set up in most nerve roots generate very characteristic sequential responses (Fig. 1); depolarizing–hyperpolarizing sequences are evoked by even liminal electrical stimuli, and both phases grow in similar proportion as stimulus strength is increased. Moreover, wave-form polarity is a reliable indicator of function; following maximal volleys an impulse

is generated by the early depolarizing phase, and postsynaptic hyperpolarization is accompanied by a decrease in transmembrane resistance which can inhibit membrane excitation.

If electrical stimuli are confined to the small nerve branch which supplies the posterior adductor muscle, the responses of type I cells to single volleys are entirely different. Only excitatory postsynaptic potentials are generated by the afferent input, and single maximal volleys can evoke one or more orthodromic impulses, as in Fig. 1. In contrast to all other nerve roots those to the posterior adductor muscle usually contain the axons of type I neurons, for antidromically propagating impulses are often observed following electrical stimulation. These impulses have a brief and

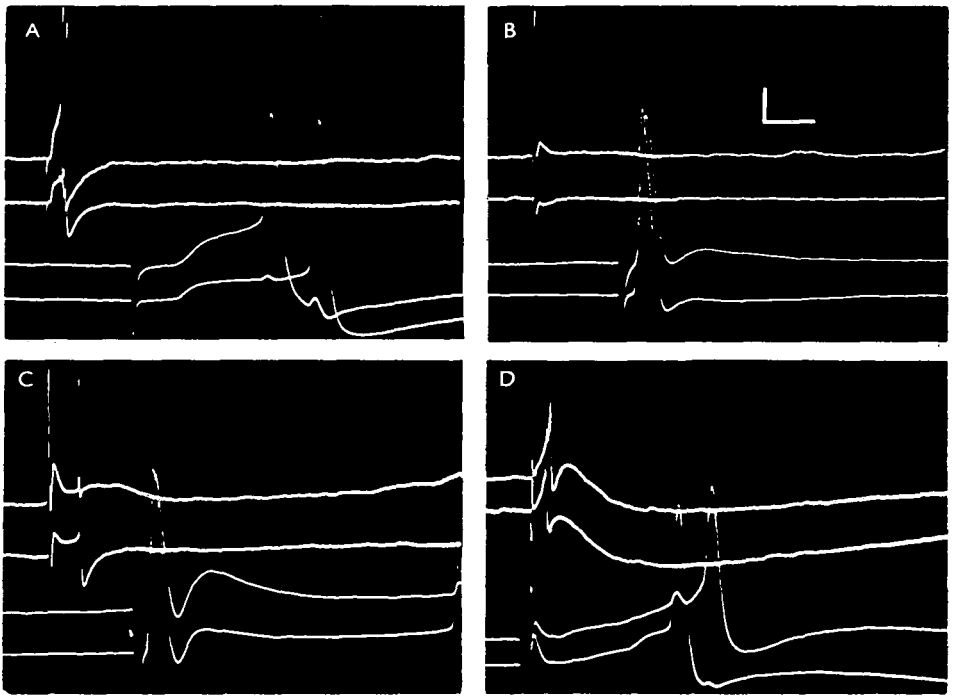

Fig. 1. Intracellular records obtained simultaneously from two type I neurons located in opposite sides of a visceroparietal ganglion. In each frame the lower traces represent an expanded version of part of each upper trace. A–C, Excitatory responses to increasing volleys evoked in an adductor muscle nerve. Low-threshold presynaptic fibres generate excitatory postsynaptic potentials and spikes in A. The axons of the impaled cells had higher thresholds and antidromic impulses first appear in B. Because antidromic spikes presumably pass through the point of origin of orthodromic impulses, the latter are blocked for several milliseconds in B and C by the refractory state of the membrane. D, Compound responses evoked in the same cells by maximal volleys in a posterior pallial nerve. Calibration: upper traces, 10 mV. and 100 msec.; lower traces, 10 mV. and 10 msec. (17° C.)

constant latency and a distinct threshold to cathodal stimuli, and they follow stimulus trains recurring at frequencies as high as 50/sec. It is worth while to note that there is little functional distinction between ipsilateral and contralateral neurons. Antidromic impulses occur in cells on both sides of the ganglion when stimuli are confined to the nerve on one side only (Fig. 1). Moreover, orthodromic response latencies in the respective neurons are almost identical. Neither of these findings is surprising; the

adductor muscles are oriented transversely, and since the animal is not fixed within its environment the behavioural response to excitation of the nerve supplying one end of the muscle would be qualitatively indistinguishable from that following excitation of both roots.

Some evidence was previously given that the sequential compound postsynaptic responses to electrical stimulation of the major ganglionic nerve trunks are due, not to recurrent collateral pathways, but to the afferent fibres themselves, acting directly and through inhibitory interneurons on the type I cells. Confirming data favouring this view are now at hand; for antidromic activation of type I cell axons is never effective in generating the prolonged hyperpolarization which invariably results from presynaptic activation.

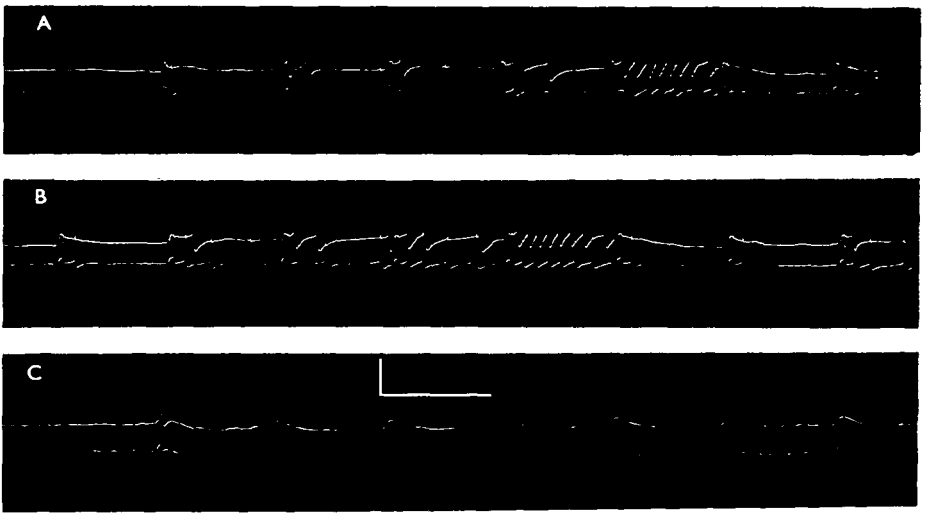

Fig. 2. Response of the same type I neurons of Fig. 1. to repetitive presynaptic volleys. Record A is continued in B and shows two cycles of facilitation and defacilitation to volleys evoked once per second in an adductor muscle nerve. Note the very similar behaviour of the two cells. C, Responses of the same two cells to repetitive volleys set up in a pallial nerve. Further details in text. Calibration: 40 mV. and 1 sec. (17 °C.).

Facilitation. Another variation in the responses of type I neurons is revealed by repetitive stimulation. Input trains in the pallial and cerebrovisceral pathways result in maintained hyperpolarization and suppression of excitatory postsynaptic responses; by contrast, identical volleys evoked in an adductor muscle nerve at a frequency of just one per second facilitate the excitatory response. As may be seen in Fig. 2, the response of cells to successive volleys increases progressively for several seconds, resulting in larger EPSP amplitude and an increased number of impulses. However, the facilitated output is itself temporally unstable and may suddenly collapse (Fig. 2 *a b*), only to grow in size once again with additional stimuli. It is difficult to localize the mechanisms for these fluctuations in response magnitude, and an analysis is beyond the scope of the present paper. The defacilitation which occurs so abruptly appears to be the result of decreased duration of the EPSP, but whether this is brought about through presynaptic or postsynaptic events is not clear.

Organization of input. Consideration of the input organization of type I cells is

necessary with regard to the argument presented in the Discussion section below. Criteria were established in a previous paper (Mellon, 1967) which suggest an afferent collateral arrangement as the basis for sequential activation of the two phases of the compound response. These are (1) identical sensory modalities, (2) similar electrical thresholds and (3) identical extraganglionic conduction velocities of the input pathways. None the less, this conclusion must still be regarded as tentative, and it will remain so until the response of type I cells can be observed following selective stimulation of single afferent nerve fibres.

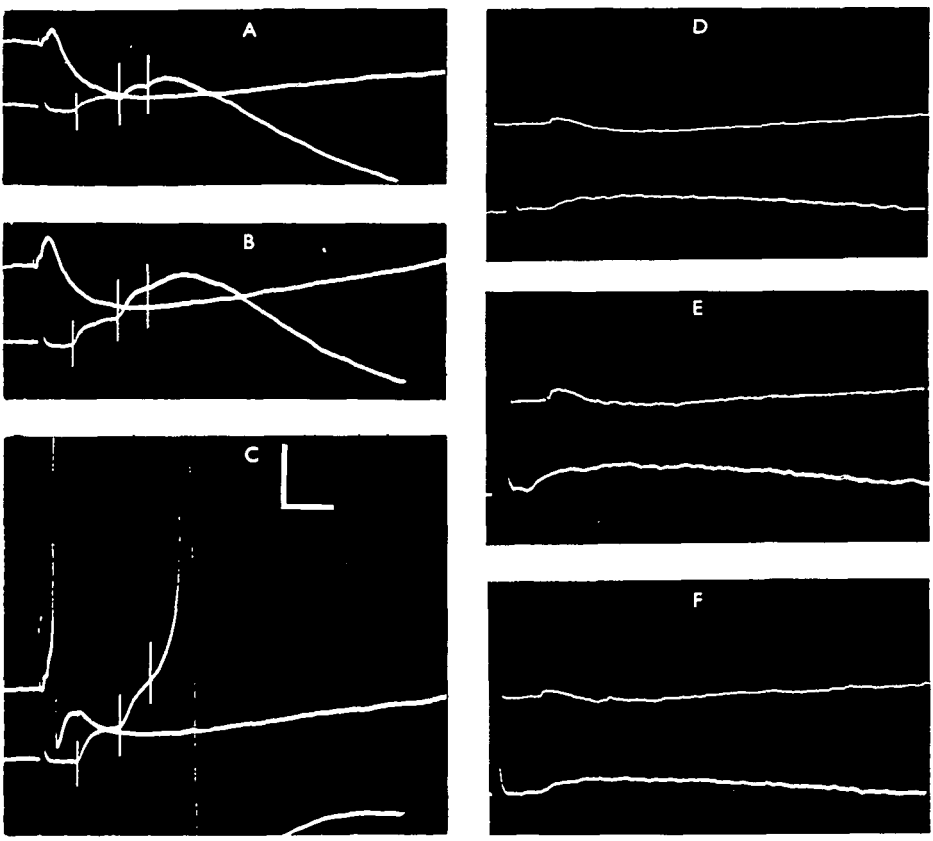

Fig. 3. A–C, Subthreshold and suprathreshold responses of a type I cell to increasing volleys in a pallial nerve. In each frame, an expanded version of the upper trace appears directly beneath it at increased gain. Individual component latencies of the postsynaptic potentials remain constant, as measured by vertical bars occurring at 14, 31, and 44 msec. after each stimulus. D–F, Records obtained from three different type I cells in response to weak shocks applied to the pallial nerve at its point of entry to the ganglion. All records obtained at a temperature of 15° C. Calibration: upper traces, 20 mV. and 200 msec.; lower traces, 10 mV. and 20 msec.

Excitatory input to type I cells from the pallial nerves and cerebro-visceral connectives is probably monosynaptic, but this is difficult to demonstrate by any direct experimental procedure. However, observations of EPSP waveform in favourable preparations suggest such an organization. The records of Fig. 3*A–C* are especially clear in this regard. Responses of a type I neuron to increasing volley strengths are shown. Note that as the stimulus strength was increased the separate component

latencies of the resulting EPSP remained constant, although the waveform components themselves increased in amplitude two- or threefold. If this compound waveform were driven by interneurons (i.e. polysynaptically) one would expect that the individual components would decrease in latency as stimulus strength (and, thus, spiking frequency in the interneurons: cf. Mellon, 1965, fig. 6) was increased. Since this clearly does not occur, the observations are best explained by a monosynaptic arrangement; the basis for different component latencies would then necessarily reside in variations in presynaptic conduction time and central delay of several afferent pathways.

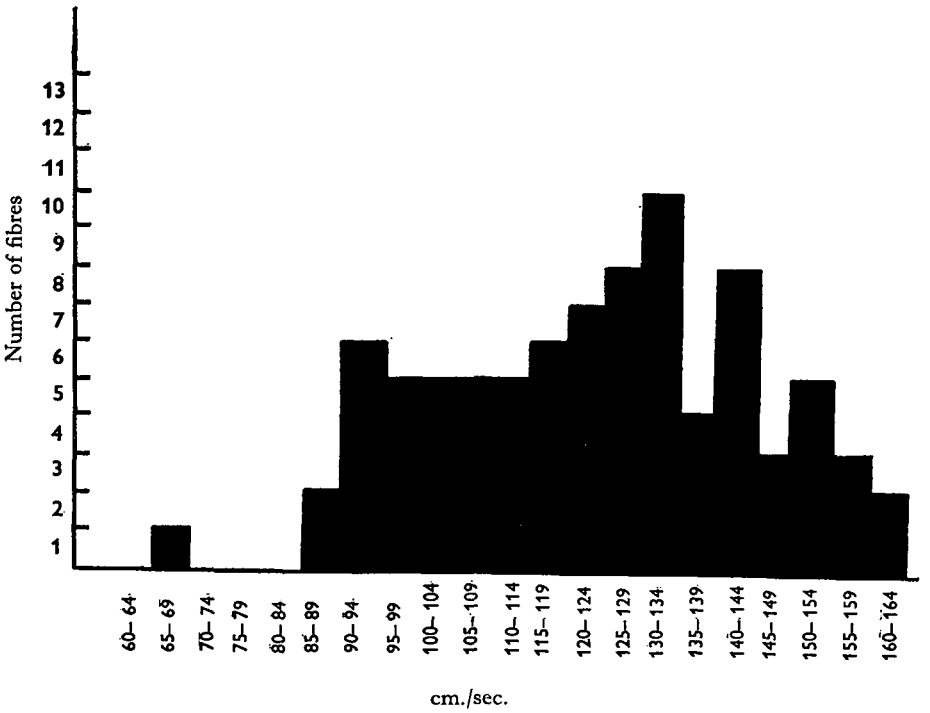

Fig. 4. Histogram showing the distribution of extraganglionic conduction velocities in eighty-five tactile sensory fibres afferent to the visceroparietal ganglion. The results are expressed as the average conduction velocities, as measured by the time taken for the action potential to pass between two different recording sites on the nerve. All measurements were made at 15° C.

In Fig. 3A–C the briefest component latency is 14 msec., and the stimulating cathode was 5–6 mm. from the ganglion. We were anxious to examine the possibility that the excitatory phase of the compound response is monosynaptic, and therefore it was necessary to obtain measurements for the range of conduction velocities encountered in the afferent fibres driving the response. To do this, several preparations were dissected in a manner such that all branches of the posterior pallial nerve were left connected to the end-organs at the periphery, and two pairs of recording electrodes were placed along each branch farther centrally. A crystal-driven stylus was then used to excite mechanically the tactile receptors on the siphons and the posterior mantle, and the conduction velocity of individual units could then be calculated from the time taken for a particular impulse to traverse the known distance between the

leads of the two recording amplifiers. The results, arranged as a histogram, are shown in Fig. 4. At 15° C very few of the sensory axons had conduction velocities slower than 0·9 m./sec. and the majority of them ranged between 1·0 and 1·4 m./sec. There is no way of knowing from our data whether these measurements reflect a representative sample of the afferent fibres which specifically innervate the type I cells. For example, if the unit of Fig. 3A–C were innervated only by the slowest fibres, this would still leave at least 4 msec. of the total latency unaccounted for. If faster afferent pathways were available to type I cells, this central delay factor could be much larger—as much as 10·5 msec. in the example given. These values seem quite large—especially for a monosynaptic pathway—and so more direct methods of measuring central delay were sought for purposes of comparison. To accomplish this the small sea-water-filled capillary previously used as a suction electrode was positioned against a pallial nerve at its point of entry to the visceroparietal ganglion. Shocks delivered to the nerve by this method were fully effective in evoking typical compound postsynaptic potentials from type I neurons; for the measurements the stimulus strength was kept at a minimum to avoid the possibility of current spread to input pathways other than those in the nerve directly beneath the electrode. Typical results are shown in Fig. 3D–F. The latency for barely threshold responses in three different cells was found to be 18, 14, and 20 msec. respectively, values which are even longer than that calculated for the unit in Fig. 3A–C. It is at present impossible to say exactly what percentage of the overall centrall delay time is specifically utilized by synaptic transfer. Most type I neuron somata are more than 1 mm. away from the point of entry of the pallial nerves into the ganglion; if the conduction velocity of all nerve fibres was reduced by a factor of ten within the ganglion, an additional 7–10 msec. of the total central delay time could be accounted for, and this would bring the synaptic delay of the proposed monosynaptic pathways reasonably close to previous estimates made on molluscan nervous systems (Bullock & Hagiwara, 1957; Mellon, 1965). Some records of Tauc (1966) indicate that monosynaptic pathways entirely within a single ganglion of *Aplysia* can show synaptic latencies of up to 15 msec., indicating that it is perhaps unwise to compare the time-course of events in these nervous systems with those values and associated anatomical pathways known from the central nervous systems of vertebrates and arthropods. None the less, the possibility of monosynaptic excitation for the type I cells of *Spisula* must remain tentative until more is known of the conductile and synaptic properties of intraganglionic pathways.

There is still no experimental evidence which bears on the nature of the receptors associated with the afferent fibres found in the adductor muscle nerves. The first suggestion which comes to mind is that they are proprioceptors sensitive to muscle tension and/or passive stretch. In partially de-afferented clams a manual forcible abduction of the valves invariably excites the adductor muscles, suggesting the presence of a stretch reflex mediated by muscle receptors. However, final confirmation of this must come from electrical recordings of activity in the adductor nerves in response to adequate stimulus. The function of such a proprioceptive loop might be twofold, serving not only as a purely defensive resistance to passive opening of the valves, but possibly, following marginal or just suprathreshold tactile input to the type I cells, serving to set the sensitivity of adductor motoneurons at a greater level for a period of time. Isolated random events may be considered of less importance to a

vulnerable animal than successive repeated stimuli, which are suggestive of purposeful i.e. predaceous, behaviour.

(*B*) *Type II cells*

Neurons designated as type II are usually located several microns below the dorsal surface of the ganglion. They are driven by all input pathways which excite type I cells, and convergence is widespread. Their postsynaptic potentials are purely excitatory. Records in Fig. 5 are typical. Liminal volleys evoked in the designated

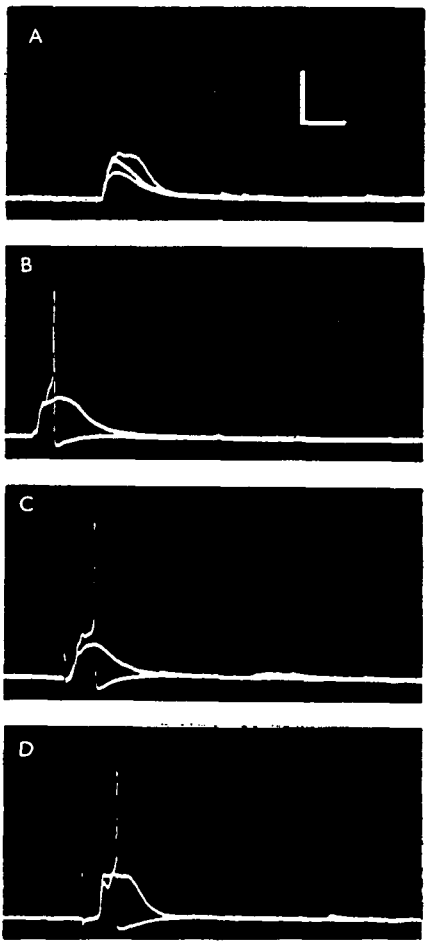

Fig. 5. Superimposed responses of a type II cell to subthreshold (A–D) and suprathreshold (B–D) volleys evoked in, respectively, (A) the ipsilateral posterior pallial nerve, (B) the contralateral posterior pallial nerve, (C) the ipsilateral cerebrovisceral connective, and (D) the contralateral cerebrovisceral connective. Increasing volley strengths were used in A to demonstrate the graded nature of the postsynaptic potential. In B–D, note that the subthreshold membrane response is abolished by the impulse. Calibration: 20 mV. and 100 msec. (20° C.).

presynaptic pathways generate excitatory postsynaptic potentials of constant latency which increase in amplitude and duration as stimulus strength rises. At threshold amplitudes of synaptic potential a single impulse is generated which, from its size

and overshoot, probably invades the soma membrane. In addition, although the falling phase of subthreshold EPSP's can be markedly prolonged by the appearance of a superimposed local membrane response, both of these phenomena are abolished by regenerative activity, indicating that the capacitance of the membrane beneath the electrode has been effectively discharged. (Fig. 5*a–d*; Fig. 6*a*). This situation changes with increasing stimulus strength. The falling phase of the spike is progressively interfered with by an increasingly prolonged EPSP, the effect being emphasized as

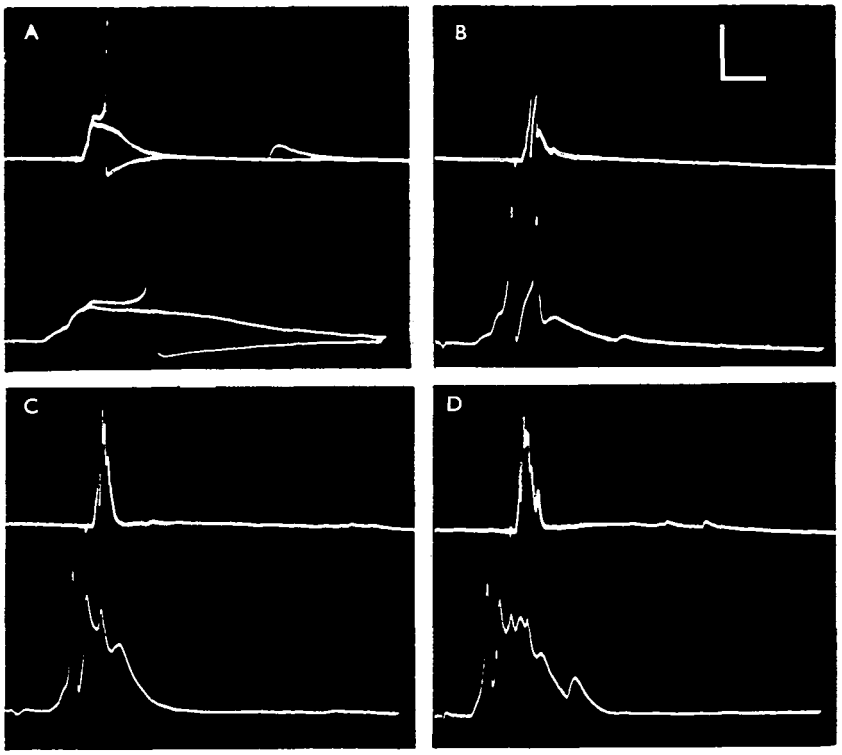

Fig. 6. Effects of stimulus magnitude upon impulse waveform in a type II cell. Expanded sweep on lower trace in each frame. A, Superimposed responses to subthreshold and suprathreshold volleys evoked in the ipsilateral pallial nerve. B–D, Increases in stimulus strength evoke multiple firing in the postsynaptic cell, but the later spikes are distorted and reduced in amplitude. See text. Calibration: upper traces, 20 mV. and 100 msec.; lower traces, 20 mV. and 20 msec. (20° C.).

more impulses are triggered (Fig. 6A–D). In some cells, maximal volleys evoke large depolarizations with only one or two small superimposed transients. These events would be understandable if it were shown that impulses are initiated at some point on the axon, while the synapses are located on the soma itself. Molluscan ganglion cells can have large associated capacitances; at threshold, impulses initiated at a region of high excitability on the axon might be expected to propagate in retrograde fashion and invade the soma membrane after some delay. However, with large depolarizations, impulses could be prevented from invading the soma by cathodal block, a situation which can occur in the crustacean stretch-receptor neurons during inordinately large depolarizations (Eyzaguirre & Kuffler, 1955; Tauc, 1958). Adequate

histological data which would be of help in locating regions of synaptic contact are virtually non-existent for bivalve nervous systems. None the less, silver impregnation techniques have revealed axo-somatic contacts in at least two other molluscan classes (Veratti, 1900; Graziadei, 1965) so that this concept is not unique among invertebrates. The large size of the synaptic potentials in the type II cells also suggests a high degree of proximity between the synaptic current generator and the microelectrode's location in the cell body.

Antidromically propagating impulses are the most prominent form of response observed from type II cells following electrical stimulation of the posterior adductor muscle nerves; and this establishes the existence of axons from these cells in the

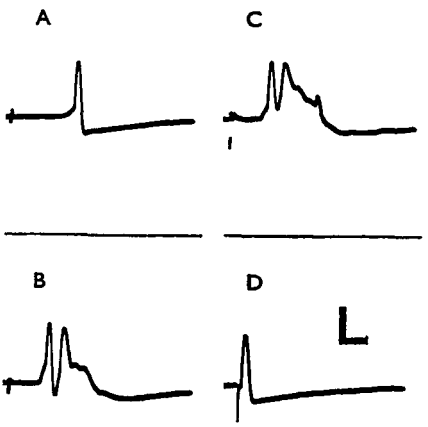

Fig. 7. Comparison of the responses in a type II neuron to volleys evoked in the pallial and adductor nerves. A–C, Stimulus intensity series delivered to the ipsilateral pallial nerve. D, An antidromic response following a shock to the adductor nerve. Calibration: 20 mV. and 20 msec. (15° C.).

efferent complement to the adductors (Fig. 7). There may also be a very weak afferent excitation to the type II cells via these roots but this usually consists of brief subthreshold excitatory synaptic activity and is not always easy to demonstrate, due to a high afferent threshold and interaction with the afterpotential of the antidromic impulse. The excitation is not nearly as strong as that observed with type I neurons.

DISCUSSION

The suggestion was made previously that a system of delayed inhibition driven by afferent collaterals would possess improved flexibility over one employing recurrent collateral inhibitory feedback. In the latter arrangement feedback is an inevitable consequence to excitation of the target neuron, and the duration of impulse activity may be limited to the conduction time of the feedback loop. The alternative system is more flexible; no inherent feedback exists, and the function, polarity and duration of the target-cell response depend specifically upon the identity of the input pathway and the duration of the applied stimulus. The response of type I cells to most inputs is a biphasic postsynaptic potential during which several impulses may be generated; excitation is always brief, however, for inhibition predominates, even with strong and repetitive input. A very different response pattern is obtained by stimulating the

nerve afferents of the adductor muscle. Postsynaptic potentials in response to such input are strictly excitatory, and successive stimuli facilitate the impulse activity of the target neuron. On the basis of these and previous findings a diagram (Fig. 8A) has been constructed to represent the input organization of type I neurons. Arguments have been presented above in favour of the illustrated arrangement of input from the pallial nerves; a monosynaptic input by adductor nerve afferents is also assumed to occur, but quantitative data are yet not available to support this assumption.

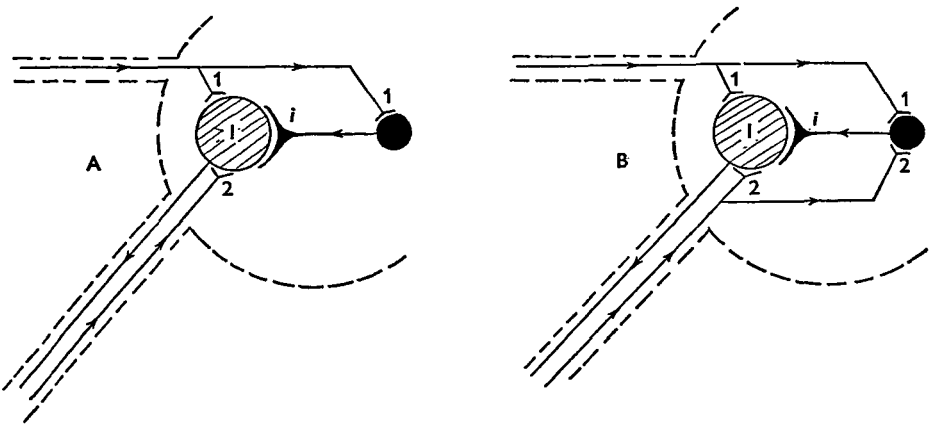

Fig. 8. Possible arrangements of excitatory and inhibitory synaptic inputs to type I cells. No implication is intended in the diagram that synaptic contacts occur on the somata of type I neurons. A, Suspected input organization based on the experimental findings discussed in the text. Excitatory synaptic transmitters of unknown chemical identity are released at the synapses labelled '1' and '2'. An inhibitory synapse is labelled *i*. B, Diagram based on Horridge's proposal that each input makes synaptic contact with all ganglion cells. The electrical sequences from type I cells would then occur if both inhibitory and type I cells were sensitive to the excitatory substance released by '1' synapses; however, only type I cells would be sensitive to '2' synaptic substance, and the resulting electrical activity would be purely excitatory. Dale's principle presumably applies.

The accepted view regarding the basis for co-ordinated output in the vertebrate nervous system acknowledges dependence upon cells exhibiting a degree of anatomical, as well as chemical, specificity. For example, the functionally differentiated afferent fibres concerned with spinal reflexes do not make indiscriminate synaptic contact with every cell in the segment of entry into the cord but are distributed in a highly selected fashion to specific motoneurons involved in postural control. However, there are also nervous systems where an extremely limited input to central neurons can trigger complex patterned output, such as the sequential firing of impulses among a group of motoneurons. This problem has been examined by Horridge (1961*a*, *b*); in particular he described an instance of this kind in the bivalve *Mya*. In that preparation a single impulse evoked in only one axon of the cerebrovisceral connectives triggers an identifiable impulse sequence among motoneurons in the anterior pallial nerves. In discussing the central physiological basis for this sequential output Horridge departed from the orthodox view concerning the relationship of structure and functional uniqueness in nervous systems; instead, he suggested that specificity at the synaptic level in bivalves may be entirely chemical in nature. Anatomical specificity is hardly an obvious feature of the unstratified molluscan neuropile. Horridge's view would suggest its absence entirely, with the possibility of all nervous elements making synaptic contact

with all others. The functional integrity of a nervous system operating by these principles would of course demand extensive chemical differentiation, and different transmitters would necessarily be involved in the execution of the various behavioural patterns triggered by the different afferent pathways. In the example of *Mya* quoted above the observed motoneuron sequence would be the consequence of a sequential *general* release of several very specific synaptic agents into the neuropile, each one secreted as the product of a selectively activated neurons (or neuronal group) which themselves had been triggered by a unique chemical agent.

While Horridge's proposal is unorthodox and seemingly without relevance to the vertebrate nervous system, the objections which can be levelled against it are not so forceful when derived from invertebrates, and at the very least it is difficult to disprove. The apparent absence of precise anatomical arrangement in the molluscan neuropile may merely reflect the inadequate powers of resolution of present-day techniques used for the examination of nervous fine structure, but we are unable to know this. Present physiological techniques are equally gross, and at best they can only detect the release of a transmitter if it has been effective in altering the electrical properties of the postsynaptic membrane. On the other hand, there is some indication that a pharmacological analysis of synaptic function in type I neurons of *Spisula* might provide a critical evaluation of Horridge's proposal. The situation has been diagrammatically illustrated in Fig. 8B. It is necessary for the argument to stipulate that an afferent collateral input is the basis for the compound postsynaptic potential observed in type I cells, and that the excitation is driven monosynaptically. Evidence favouring these assumptions has been outlined above in the 'Results' section.

In the Horridge system each separate input element makes synaptic contact with all ganglion cells. Only the respective specificities of the released transmitters and the various postsynaptic target cells can determine the output pattern which will result from a stimulus in any particular input pathway or combination of inputs. It is therefore clear that the transmitters mediating both genera of EPSP's in type I cells could not be identical; for if these two modes of response are not brought about by precise anatomical connexions, specific chemical addressing of the neurons must occur, logically requiring at least two transmitter substances to be released into the neuropile. Any observations which indicated the presence of different transmitters at the excitatory synaptic loci would thus lend tentative support to Horridge's proposal; however, the latter would be negatived by the discovery of identical transmitters at these loci. In view of these conclusions, however, it should be stated that the pharmacology of the bivalve nervous system is poorly understood. Horridge found that acetylcholine inhibits synaptic transfer, while 5-hydroxytryptamine generated spontaneous electrical activity in the moneurons supplying the mantle. No published investigations of a similar nature are available for the *Spisula* nervous system. Some recent unpublished observations in our laboratory have established, however, that the cholinergic blocking agent D-tubocurarine is without any effect on synaptic transmission of any kind involving type I neurons, even when applied in massive concentrations. Thus, the role of acetylcholine in the ganglion remains a matter for speculation.

The functional connexions made at the periphery by either of the two populations of efferent fibres have not yet been examined. In another tonic smooth muscle preparation (Takahashi, 1957) there is evidence that both excitation and relaxation

are under direct nervous control; however, there is little evidence from the above studies to indicate which, if either, of these functions in the *Spisula* adductor the two efferent populations subserve. As mentioned above, the adductor muscles appear to contract reflexly following passive stretch, and of the two groups of efferent cells examined only type I receive an appreciable afferent input from the adductor muscle nerve; this would appear to identify these cells as the motoneurons responsible for the reflex. However, the precise functional identities of the efferent neuron populations must await the results of simultaneous recordings from cellular elements both within the central nervous system and at the periphery.

SUMMARY

1. Two populations of neurons in the visceroparietal ganglion of the surf clam have been identified as efferent to the posterior adductor muscle. Both populations are driven by input from the cerebrovisceral connectives and the posterior pallial nerves. The latter pathways originate in tactile receptors on the siphons and posterior mantle.

2. Cells designated as type I respond to input over the four major nerve roots by compound postsynaptic potentials, and to input over the adductor muscle nerves by facilitating strictly excitatory potentials. Type II neurons respond in a purely excitatory manner to input over all pathways tested. It is suggested that pharmacological analysis of the input excitatory synaptic contacts on type I cells would have implications for the structural organization of molluscan neuropile.

3. Neither efferent pathway has yet been identified in terms of functional connexions made at the periphery. It is assumed from behavioural observations that type I cells are motoneurons controlling phasic contraction of the adductor, but electrophysiological confirmation of this is still wanting.

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