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SOME FEATURES OF THE CENTRAL CO-ORDINATION OF A FAST MOVEMENT IN THE CRAYFISH*

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INTRODUCTION

It has been shown (Wiersma, 1947; Roberts, 1968) that a single impulse in any one of the central giant fibres of the crayfish is sufficient, in free-moving animals, to evoke a complete escape response in which the crayfish gives a vigorous flexion of its tail and, as a consequence, darts backwards through the water. This single impulse is fairly certainly the normal central nervous 'command' which initiates the escape response. The aim of this report is to describe the pattern of nervous and muscular activity resulting from such a single impulse under conditions when peripheral reflexes cannot occur, to see how neurones and muscles in the tail are affected, or used, by a normal central nervous 'command'. It was hoped that such a description would throw some light on (*a*) the capabilities of central nervous mechanisms to form a complex pattern of nerve impulses and muscular contractions in the absence of any peripheral reflexes, (*b*) the co-ordination of different muscles, the flexors and extensors and the separate slow and fast muscle groups, and (*c*) the function of the multiple excitatory and inhibitory innervation of the muscles (a problem already partially clarified for some abdominal muscles by Kennedy & Takeda, 1965*a*, *b*). The crayfish is especially suitable for this kind of study since it is possible to record the individual activity of all the efferent neurones running to the muscles responsible for movements of the abdominal segments (see Kennedy & Takeda, 1965*a*, *b*; Kennedy, Envoy & Fields, 1966; Parnas & Atwood, 1966; Otsuka, Kravitz & Potter, 1967; and below).

METHODS

The animals (*Procambarus clarkii*) and procedures for keeping, operating, intracellular recording and photography are described in a previous paper (Roberts, 1968). All recordings were from isolated abdomens which were pinned down in a chamber containing Dudel's crayfish saline (Dudel, 1961) cooled to 15 ± 1 °C by a thermoelectric cooler. For stimulation and recording, nerves or connectives were raised on fine bipolar platinum hook electrodes into little drops of viscous mineral oil floating on the saline. (The oil was thickened by adding petroleum jelly.) This method gives

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good signal-to-noise ratios but avoids the complications and mess of extensive oil layers. The lateral giant fibres were stimulated by a threshold shock to the connectives between the fifth and sixth abdominal ganglia. Tension was recorded with an RCA transducer fitted with small forceps.

Two basic preparations were used. The first was for study of flexor efferent neurones and their cell bodies in abdominal ganglia (see also Otsuka *et al.* 1967). The whole abdomen, except for the nerve cord, was cut in half longitudinally along the mid line. The two halves were then pulled slightly apart and pinned rigidly ventral side up. In this position nerve roots and cell bodies in the ganglia could be seen beautifully with trans-illumination. In the second preparation used to record from the extensor muscles and their efferent neurones, the abdomen was also cut in half, this time transversely at the fourth abdominal segment. The nerve cord was left intact and the two halves were rotated 180° relative to each other. The posterior half was then pinned ventral side up so that the nerve cord could be stimulated and recording electrodes placed to check the results of stimulation. The anterior three segments were pinned dorsal side up and a small window was cut from the exoskeleton to expose the extensor muscles and the motor branches of the second root supplying these muscles (cf. Eckert, 1961*a*).

Serial sections of crayfish third abdominal ganglia, used in the identification of flexor efferent neurones, were kindly lent by Mrs J. Hanawalt, Dr J. Kendig and Dr D. Kennedy. They were cut at 10 μ in transverse, sagittal and longitudinal planes and stained by Rowell's (1963) silver method. Cell processes were followed in photographs and tracings of the sections.

RESULTS

Anatomy

(*a*) *The musculature* has been described recently by Pilgrim & Wiersma (1963). Something of the complexity of its function is indicated by Rayner & Wiersma (1967) but, for the present purpose, these complexities are ignored and the muscles are lumped into four groups: fast flexors, slow flexors, fast extensors, and slow extensors. It is these muscles which are responsible for postural maintenance and movement of the abdominal segments. As in fish (Bone, 1966), the fast muscles are bulky and deep while the slow muscles form thin superficial sheets.

(*b*) *The efferent neurones.* Only those efferent neurones in the second and third roots of the third abdominal ganglion which supply the four muscle groups listed above are considered. Most of them can be identified by their characteristic spike height in records from the motor nerves together with the type of junction potential that they evoke in the muscles (see Kennedy & Takeda, 1965*b*; Parnas & Atwood, 1966). Some can only be recorded from separately by micro-electrodes placed in their cell bodies. These can be located visually in abdominal ganglia and identified by physiological techniques of stimulating and recording. Using this method, Otsuka *et al.* (1967) have identified the cell bodies, or somas, of a large number of efferent neurones in lobster abdominal gnaglia. Among these neurones are all those supplying the fast flexors. These are shown in Fig. 1 A, which is based on one of the maps of the second abdominal ganglion of the lobster drawn by Otsuka *et al.* Figure 1 B is a map of some of the same flexor efferent neurones in the third abdominal ganglion of the crayfish. This

was made by reconstruction from silver-stained serial sections and is less complete than the lobster map. Comparison of the two maps shows that, although there are differences in relative sizes and spacing, a common complement of neurones runs caudally to supply the fast flexor muscles of both animals. I have numbered the crayfish cells like those of the lobster to show this correspondence. However, only in one case (I_2) is the homology certain. The other cells seem to be arranged in pairs (e.g. (4, 5), (6, 7) and (9, 10)) which are clearly homologous with those in the lobster, but numbering of the members of these pairs is uncertain in the crayfish until the peripheral innervation fields of the neurones are known. Rostrally travelling fast flexor

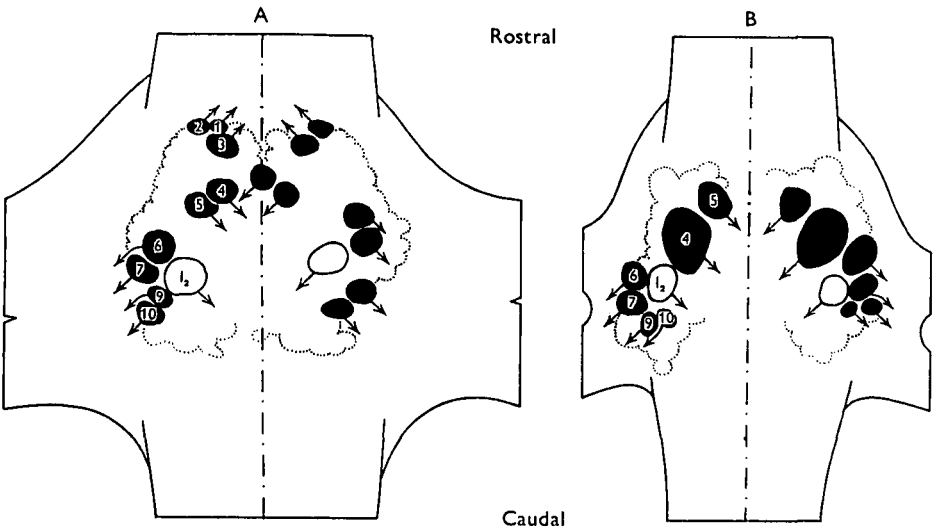

Fig. 1. Efferent neurones supplying fast abdominal flexor muscles. A, Second abdominal ganglion of the lobster (based on fig. 14A in Otsuka, Kravitz & Potter, 1967). B, Third abdominal ganglion of the crayfish. Black cells are the excitatory motoneurones. White cells (e.g. I_2) are the peripheral inhibitors. The arrows indicate whether the axon exits rostral or caudal to the ganglion and on which side (e.g. I_2 exits caudally and contralaterally). The dotted lines indicate approximate extents of cell somas in the ganglia. A and B are shown at different magnifications.

motoneurones are present in the crayfish (Takeda & Kennedy, 1964) but these were not successfully traced in the sections. Though the cell somas of the caudally running efferent neurones are widely scattered, the sections showed that all but the motor giant (no. 4) pass through a confined caudal, dorsal region of coarse neuropile just ventro-lateral to the medial giants in each ganglion (cf. Otsuka *et al.* 1967). This neuropile would seem to be the centre for control of the fast flexor muscles.

In their study Otsuka *et al.* were able to stimulate cell bodies while recording intracellularly in muscles. In this way they could find whether the cell penetrated excited or inhibited any muscle. Their identification of the cell marked I_2 in Fig. 1A as the inhibitor of the fast flexors was based on this technique but was also confirmed by chemical analyses of individual cell somas. These analyses showed that the inhibitory cells contained more GABA (gamma-amino-butyric acid) than excitatory cells. By comparison with the lobster the inhibitor for the fast flexors in the crayfish can be

identified (I_2 in Fig. 1 B). It is the most caudal cell to send its axon to contralateral muscles. Consequently it can be identified easily, both physiologically and in sections.

Physiology

Using a combination of recording techniques (extracellularly from nerves, intracellularly from muscles and cell somas) activity in all the efferent neurones to the principle tail muscles can be observed. Responses in the different groups of efferent neurones to impulses in the lateral giant fibres will be described. Only slight latency differences have been observed between these responses and those to impulses in the medial giants (see also Wiersma, 1947).

(*a*) *Fast flexor efferent neurones.* When a giant fibre fires an impulse there is a burst of impulses in the anterior third roots supplying the fast flexor muscles. This burst is followed by a rapid tension increase in these muscles which are responsible for the flexing flip of the tail (see Fig. 2A; Wiersma, 1947; Kennedy & Takeda, 1965*a*). It is hard, in the burst recorded from the fast flexor motor nerve (anterior third root

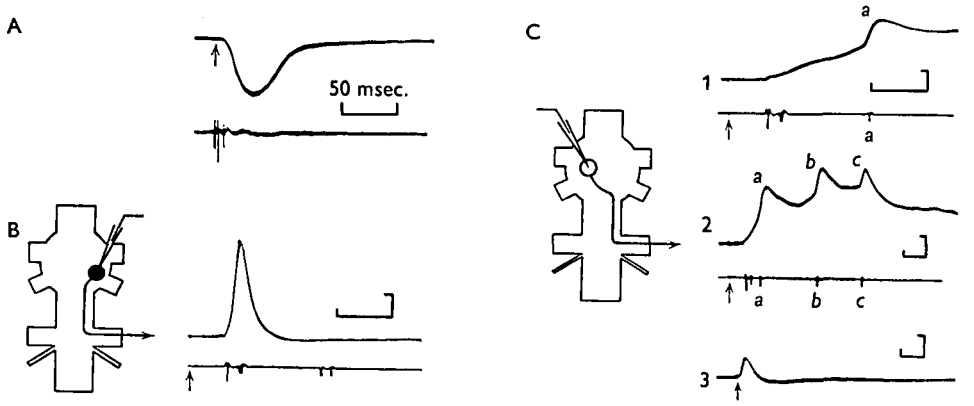

Fig. 2. Fast flexor responses to stimulation of the lateral giants. Arrows indicate the timing of the stimulus. The diagrams in this and Fig. 3 show the 3rd abdominal ganglion and nerve cord in ventral views. The position of the cell penetrated is indicated. A, Tension in a group of fast flexor muscles (anterior oblique, tension increase moves trace down) and, lower beam, efferent activity in the anterior third root (R 3 ant) supplying these muscles. B, Intracellular recording from a motoneurone (number 6 or 7) in relation to extracellularly recorded activity, lower beam, in R 3 ant in which motoneurone's axon leaves the C.N.S. C, Intracellular recordings from a peripheral inhibitor (number I_2) in the same ganglion and preparation and with the extracellular electrodes in the same position as in B. Small letters show correspondence of root and intracellular records. (N.B. C 2 and 3, are at a slower sweep speed than B and C 1.) C 3 shows the response of I_2 to antidromic stimulation of R 3 ant delivered via the electrodes used in B, C 1 and C 2 for recording the extracellular responses. Calibration for B and C are 5 mV and 5 msec.

R 3 ant), to separate the responses of the individual neurones (Takeda & Kennedy, 1964). This can be achieved with certainty by intracellular recordings made from the cell somas of the efferent neurones on the ventral sides of the abdominal ganglia.

When all sensory roots in the isolated abdomen are cut one observes that, with the possible exception of cell no. 5, which I was not able to identify physiologically with certainty (see Fig. 1 B), all the efferent neurones to the fast flexors respond to a giant-fibre impulse. In a fresh preparation the excitatory cells each give a single impulse

after a short (0·5 msec. or more) delay. This conclusion is based on the correlation of spikes in the flexor motor nerves (R3ant) with fast-rising, depolarizing components recorded in cell bodies (see Fig. 2B). It is endorsed by recordings of large, early spikes from unidentified fast flexor motoneurones made in the neuropile and by the very similar responses reported by Takeda & Kennedy (1964). Only one cell behaves differently and this is the inhibitor whose response is much more prolonged. An example is shown in Fig. 2C. The cell was identified by its posterior lateral position and by the fact that it gave an antidromic response to stimulation of the contralateral R3ant (Fig. 2C, 3). It can be seen that the later root spikes correspond to faster-rising cell soma depolarizations which ride on a longer-lasting depolarization. From the similarity of the soma responses to antidromic root stimuli and giant-fibre impulses one can be fairly certain that the faster depolarizations occur when there are impulses in the main axon of the inhibitory neurones. These impulses do not actively invade the

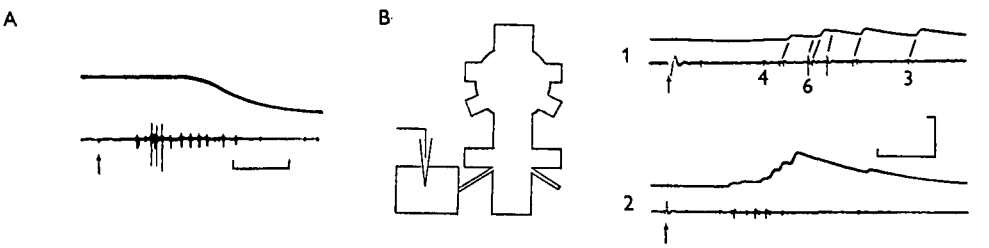

Fig. 3. A, Tension in a group of slow flexor muscle fibres (tension increase moves trace down) and, lower beam, efferent activity recorded extracellularly from the posterior third root supplying this muscle. B, Intracellular records from slow flexor muscle fibres for identification of active fibres in the root discharge: (1) from a lateral fibre allows clear identification of the active axons (3, 4 and 6); (2) shows a more typical response. Calibrations are 50 msec. and, for intracellular records, 10 mV. The arrows indicate the timing of the stimulus to the lateral giants.

cell body. In the cell of Fig. 2C there were generally three impulses and this was the maximum number ever seen. Other preparations gave two late spikes but in some there was just one. The latency of the first of these 'late' spikes ranged widely in different animals, from 5 msec to as much as 20 msec.

(*b*) *Slow flexor efferent neurones.* When all other nerves were cut, activity was monitored in a posterior third root (R3post) supplying the slow flexor muscles of one segment. At the same time, tension in a group of these muscle fibres was recorded. For about 40 msec after a giant-fibre impulse spontaneous discharge appeared unaffected. Inhibition of various levels of spontaneous discharge was looked for but not observed. A burst of impulses in the motor nerve follows this period and results in a small tension increase in the slow flexors which lasts for some seconds (see Fig. 3A).

The composition of the burst of motor impulses can be uncovered by a comparison of nerve impulses of different amplitudes and intracellularly recorded slow flexor muscle potentials. In this was the activity of separate axons can be studied as was shown by Kennedy & Takeda (1965*b*), who numbered the axons from 1 to 6 in order of increasing diameter and spike amplitude. Typically (see Fig. 3B) axons 3, 4 and 6 are each excited and give a burst of spikes. As in the case of the inhibitor to the fast flexors there is considerable variation in the form of the bursts. Axon 6 invariably

fires at least one impulse, but 3 and 4 are less reliable and sometimes neither responds.

(*c*) *Extensor efferent neurones.* In contrast to the flexor muscles the extensors appear to receive no efferent impulses when the giant fibres fire. A search was made using nerve and intracellular muscle recording but giant-fibre impulses had no observable effect on either the slow or fast extensors or their efferent neurones. Trials with other methods of preparation gave the same result. Any responses seen could always be shown to depend on the integrity of some peripheral reflex pathway (cf. Wiersma, 1952; Eckert, 1961*b*).

(*d*) *Other efferent neurones.* A barrage of impulses travels towards the swimmerets via the first roots (see Wiersma, 1952) but description of this activity is outside the scope of this paper. A further efferent response is seen in the dorsal branch of the second root where the accessory neurone, which inhibits the slow abdominal stretch receptor, gives a burst of impulses. Resting discharge of the stretch receptor, if present, can be blocked by this burst (cf. Eckert, 1961*a*). A tracing of one of these accessory neurone bursts is shown in Fig. 4. (For bursts which were photographed, latencies to the first spike were 20–60 msec., the number of spikes in the burst ranged from 1 to 4, and the frequencies were around 100/sec.)

(*e*) *Central responses.* Within the ventral nerve cord the motor and lateral giant neurones are inhibited in a very similar manner. In both, inhibition lasts for about 70 ± 20 msec. and is accompanied by a post-synaptic conductance increase and depolarizing IPSPs (for details about motor-giant inhibition see Hagiwara, 1958, and Furshpan & Potter, 1959). Intracellular stimulation of central giant axons has shown that in Procambarus the motor-giant inhibition is definitely a consequence of central giant-axon impulses. However, in fresh preparations it is not strong enough to prevent the motor giant giving an overshooting impulse in response to such central giant impulses. The synaptic pathways producing inhibition of the motor giants have many features in common with those producing inhibition of the lateral giants. In both, inhibition is evoked by an impulse in any central giant fibre, repeated stimulation rapidly leads to reduced inhibition, and for both there is similar evidence for a common pathway excited by central giant fibres and producing the inhibition (see Roberts, 1968).

Other neurones respond to giant-fibre impulses and a few of these cells have been recorded intracellularly. Most respond with a single early impulse, some give two closely spaced spikes and some give low-amplitude, long-lasting depolarizations.

DISCUSSION

(*a*) *General reservations*

This paper is concerned with a sequence of events in the central nervous system and its relation to a sequence of movements in the free animal. The events in the nervous system were studied under highly artificial conditions, with no blood circulation or afferent sensory inflow and with most of the central nervous system removed. This was done for convenience, especially in ensuring that peripheral reflexes were eliminated.

As a consequence of the removal of many normal, tonic influences (afferent and from other parts of the central nervous system), the general level of excitability of the

abdominal nerve cord must be changed. This change is probably towards lowered excitability. Without being certain of this, one can predict that latencies, amplitudes of PSPs, frequencies—in fact most features of nervous activity—will undergo shifts. For this reason the exact parameters of the responses described in this paper are not as important as the general observation that a response is present. Further, the total absence of a response may not be of special significance in every case since it may be a reflexion of lowered general excitability. The impressive feature is the extent of patterned and apparently adaptive activity which survives the removal of afferent input and most of the nervous system.

(*b*) *Efferent activity underlying the tail flip*

In Fig. 4 the results are summarized in a diagram of nervous and muscular responses during the tail-flip escape movement. Only muscles and neurones which are clearly affected by central giant impulses are included and the responses will now be discussed chronologically.

(i) The first response to central giant impulses is from the motor giant fibres, which are excited electrically (Furshpan & Potter, 1959) and appear to be specialized for rapid excitation of the flexor muscles. Branches of the motor giants innervate the majority of muscle fibres in the different groupings of the fast flexors (Kennedy & Takeda, 1965*a*; Otsuka *et al.* 1967). Consequently, nearly all the fast flexors are excited a very short time after central giant impulses. This makes it rather unlikely that any complex pattern of nervous impulses is required for the execution of a fast tail flexion. Rather, the muscles are excited nearly synchronously and the pattern of movement results from the arrangement and response characteristics of the muscles themselves (cf. Rayner & Wiersma, 1967).

(ii) This conclusion receives support from the behaviour of the other, non-giant motoneurones whose innervation fields within the flexor musculature are more confined. With the possible exception of number 5 (see Fig. 1) the motoneurones all respond with a spike at a short latency (0·5–2 msec.). Such pattern as there is must be confined to this interval. Soma responses are similar to those described by Takeda & Kennedy (1964) for some of these neurones. The anatomical connexions between these motoneurones and the central giant fibres have not been described in detail but the short latency of their responses suggests a close relationship, perhaps even direct synaptic contact as Takeda & Kennedy (1964) have suggested. These workers (Kennedy & Takeda, 1965*a*), have shown that, in contrast to the motor giants, the muscle EJPs (excitatory junction potentials) produced by non-giant motoneurones facilitate on repetition. They suggest that, during the repeated tail flips in swimming, though the response to motor giant impulses rapidly declines, that to the slightly later non-giant impulses increases. Consequently the muscle fibres with a dual excitatory innervation can contract repeatedly when response to the motor giant impulse has virtually vanished. This gives a rationale for the presence of a dual excitatory innervation.

(iii) Having been excited by the motoneurones, the fast flexor muscles then receive an inhibitory burst of one or more impulses (see Fig. 4). This behaviour is very interesting in the light of comments by Kennedy & Takeda (1965*a*) on possible functions for peripheral inhibition in fast musculatures. They found the inhibitor to the

fast flexors to be rather ineffective in reducing peak tension in these muscles, and argued that in these twitch-type muscles the idea of inhibition and excitation subtly interacting to produce gradations of contractions was out of place (cf. Runion & Usherwood, 1968, unpublished results). Rather, peripheral inhibition functioned '...to cut short excitatory depolarizations and hence to terminate lingering tension

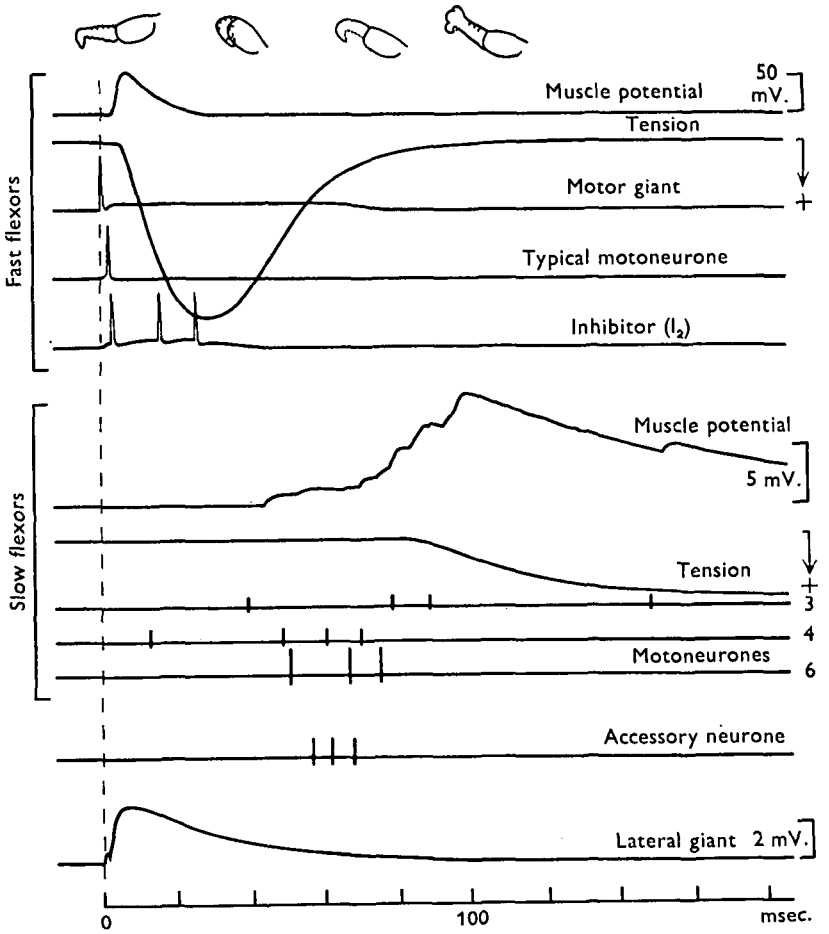

Fig. 4. Efferent and central effects of a medial giant-fibre impulse. This diagram is based on typical records drawn from a number of preparations. The dashed line shows time of medial giant impulse. Only cell types affected by giant-fibre impulses are included. For the fast flexor efferent neurones diagrammatic intracellular responses are shown. Note the long depolarizing IPSP in the motor giant. The slow flexor diagram is based on the record in Fig. 3 B2. The pictures at the top showing animal position are tracings from high-speed ciné films. Voltage calibration for fast flexor muscle potential applies also to fast flexor efferent neurones.

that might oppose subsequent reflex actions'. That is, inhibition may lead to faster relaxation of the fast flexors. Involvement of peripheral inhibition in relaxation of slow crustacean muscles had been proposed by Pantin (1936), Katz (1949) and Bush (1962). Perhaps Bush's suggestions are closest to those of Kennedy & Takeda (1965*a*). His idea was that background peripheral inhibition could lead to faster relaxation and thus aid the execution of more rapid walking movements in crab legs. The crayfish

tail makes fast oscillations in swimming, and intracellular recordings from fast crayfish tail muscles have shown that inhibitory activity speeds up the repolarizing phase of EJPs (Kennedy & Takeda, 1965*a*; Atwood, Parnas & Wiersma, 1967). However, where tension has been recorded in fast and slow abdominal muscles, inhibitory activity apparently has no effect on the rate of relaxation (Atwood, Parnas & Wiersma, 1967; Evoy, Kennedy & Wilson, 1967).

The situation at present is somewhat confused. In the tail-flip escape movement, triggered by central giant-fibre impulses, the fast flexors are first excited and then receive a burst of inhibitory impulses. This kind of inhibitory activity would fit well with the hypothesis that muscular inhibition was concerned with hastening the relaxation phase so as to facilitate oscillatory movements. The next step is to test this hypothesis by looking more carefully at the effects of inhibitory bursts, like those described in this paper, on the electrical and mechanical responses of fast flexor muscle units. Having found a way in which the central nervous system uses the peripheral inhibitor we must now unravel the effect on the muscles. The usefulness of this type of approach in providing a real understanding of motor mechanisms has recently been shown by Runion & Usherwood (1968, unpublished results) studying the role of peripheral inhibition in locusts.

(iv) While the fast flexors are inhibited peripherally there is further inhibition in the ventral nerve cord. The lateral and motor giant neurones are inhibited for from 50 to 90 msec. following giant-fibre impulses (see Fig. 4 and Roberts, 1968). Inhibition of the lateral giants has been considered in another paper (Roberts, 1968) where it was suggested that the inhibition would supress further responses while the escape flip was in progress and in this way avoid conflicting commands to the muscles. The role of inhibition in the motor giant is not so clear since it is not sufficiently strong, in a fresh preparation, to prevent the motor giants from giving an impulse in response to excitation from the central giants. It could be that their inhibition only prevents such responses under particular circumstances when the inhibition is supplemented by reflex or central inputs. Alternatively, the inhibition could act against some other pathways exciting the motor giants. Wiersma (1952) reported that stimulation of the first roots could excite the motor giant to fire without central giant impulses. This is the only reported alternative excitatory input to the motor giant. Its description was based on extracellular recordings and I have not been able to confirm it using extracellular or intracellular recording. Further, Kennedy and his co-workers (personal communication) have not found any central command units apart from the central giant fibres, which, when stimulated, evoke rapid abdominal flexion. This also suggests an absence of alternative excitatory pathways to the motor giants. Inhibition as a consequence of central giant-fibre impulses has not been observed in the other, non-giant motoneurones (see also Roberts, 1968).

(v) The complete absence of any efferent traffic to the extensor muscles as a result of giant-fibre impulses was quite a surprise. Eckert (1961*a*, *b*) had recorded vigorous bursts of extensor activity during swimming movements in intact crayfish and Wiersma (1952) recorded one large spike and a burst of smaller ones in the second root (supplying the extensors) following stimulation of a central giant fibre. In an earlier paper (Wiersma, 1947) this response had not been seen and its appearance was attributed to fresher preparations. This lability of second root activity is endorsed by

my own observations. In intact animals held by a clamp on the carapace, there is invariably a maintained flexion response to repeated stimuli to central giant fibres. On the other hand, extension is never full and soon wanes. Under these circumstances activity of abdominal proprioceptors, such as the dorsal muscle receptors (see further, Eckert, 1961 *a*, *b*), should initially be fairly close to that in a free animal. It is therefore unlikely that the failure of the extension command is due to the absence of afferent input, unless some critical feedback, perhaps from water currents or backwards acceleration, is lacking. Neither Fields (1966) nor I have found any way of centrally exciting the fast extensor motoneurones. It is probable that these motoneurones simply require more 'normal' central nervous and reflex input for their activity. This suggests that their central connexions may be more extensive and complex than those of the fast flexor motoneurones.

The slow extensor muscles, though spontaneously active, appeared to be neither inhibited or excited as a consequence of giant-fibre impulses.

(vi) The rate of tension development in the slow flexors is altogether too slow for them to be directly involved in the flexion phase of the tail-flip (see Figs. 2, 4). Consequently it was possible that tension in them might interfere with fast tail movements. Denny-Brown (1928) had suggested that inhibition of postural motor activity would reduce stiffness and thus facilitate fast movements. However, motor outflow to the slow abdominal flexors of the crayfish does not appear to be centrally suppressed during any phase of the escape tail-flip. The motoneurone activity and consequent tension rise in the slow flexors seem appropriately timed to damp the final phases of tail extension (see Fig. 4). They may thus help to stabilize the tail preparatory for another flip or for the re-establishment of postural tone.

The motoneurones excited by the central giants are those which lead to faster tension rises in these slow muscles. Numbers 3 and 4 produce the largest EJPs and number 6 facilitates rapidly (Kennedy & Takeda, 1965*b*; Evoy *et al.* 1967). These authors have reported that these three neurones are the ones most usually involved in more phasic reflex responses. The tonic units, numbers 1 and 2, are apparently not affected during the tail-flip.

(vii) The behaviour of the accessory nerve during repeated swimming movements was described by Eckert (1961*b*). He reported that it discharged vigorously during the flexion phase of the tail-flip even when the muscle receptor organs were destroyed and flexion was prevented by denervation of the flexor muscles. I can confirm that the accessory neurones are excited by the central giants. However, the timing of the accessory neurone burst was very variable. If the burst functions to reduce extensor resistance reflexes during the flexion phase of the tail-flip, as seems likely (see Fields, 1965; Fields, Evoy & Kennedy, 1967), then it would sometimes seem a little late in the cycle. This could be a result of the lowered central excitability in my preparations, which has already been mentioned.

(*c*) *The formation of central impulse patterns controlling movement*

It is disappointing how little can be said about the mechanisms of formation of the pattern of nerve impulses described here. The basis for long PSPs and long delays has been studied (see Roberts, 1968) but with relatively little success. As is so often the

case, especially in invertebrates, the lack of detailed anatomical information about the neurones involved leaves too many plausible physiological explanations for every feature of the response. This makes experimental analysis difficult.

To investigate other aspects of the central impulse pattern should, on the other hand, be straightforward. Two main questions stand out. First, how does the pattern develop? A genetically specified central pattern must, for example, be modified to suit animals differing in size by as much as 10 times. Secondly, how is the pattern modified, changed or added to by proprioceptive reflexes once it is established? It must be adapted to suit a variety of immediate physical conditions. The modification could be achieved by the same proprioceptive reflexes in the two cases. However, it is likely that mechanisms other than simple reflexes will be involved in changing the central impulse pattern during ontogeny. Among many possibilities, long-term conditioning of the pattern by proprioceptive input is an attractive suggestion.

SUMMARY

(i) The nervous and muscular activity pattern produced in an abdominal segment by a single giant-fibre impulse when all peripheral reflexes are abolished is described.

(ii) This description depends on criteria for the identification of efferent neurones to the tail muscles. These are discussed and the identification of the inhibitor to the fast flexor muscles is established by anatomical homology.

(iii) The pattern of response to a giant-fibre impulse is summarized in Fig. 4.

(iv) Inhibition of the fast flexors, the lack of an extension command, the slow flexor contraction, the accessory neurone discharge and central nervous inhibition during the escape movement are among the features discussed.

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