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THE NEURAL BASIS OF ESCAPE SWIMMING BEHAVIOUR IN THE SQUAT LOBSTER *GALATHEA STRIGOSA*

III. MECHANISMS FOR BURST PRODUCTION

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SUMMARY

1. Intracellular recordings have been made from a variety of phasic motor neurones during expression of the motor programme for backward swimming in *Galathea strigosa*.

2. Fast flexor motor neurones (FFs) are driven by a large depolarization mediated by chemical synapses and are inhibited in the interburst interval.

3. Fast extensor motor neurones (FEs) are driven by a barrage of unitary EPSPs during the extension phase and may receive unitary synaptic inhibition while the flexors are active.

4. FFs all have similar spike thresholds and fire bursts of spikes superimposed upon the depolarized peak of the input. FEs show a gradation in spike threshold which is correlated with soma size. The largest FEs (type 2) have higher thresholds than smaller FEs (type 1), and fire fewer spikes.

5. The phasic inhibitor motor neurone to flexor muscles (FI) is driven by complex central pathways and fires a single spike shortly following flexion.

6. The extensor inhibitor (EI) appears to receive the same depolarization as do the FFs, but has a low spike threshold and thus fires on the rising phase of the depolarization. Spiking in EI is terminated by unitary IPSPs which occur in phase with FF activity and which may have the same origin as the interburst inhibition seen in the FEs.

INTRODUCTION

The squat lobster, *Galathea strigosa*, escapes from threats by means of repeated extension-flexion cycles of the abdomen which result in rapid backward propulsion. This behaviour is probably homologous with non-giant swimming in the crayfish (Schrameck, 1970; Wine & Krasne, 1982). Escape is controlled by a central pattern generator (CPG) located either in the suboesophageal ganglion or the thoracic ganglionic mass (Sillar & Heitler, 1985*b*). The motor programme for swimming consists of alternating bursts of impulses in antagonistic extensor and flexor motor neurones, and can be recorded from the roots of deafferented abdominal ganglia. Escape usually

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begins with extension and is followed at short latency by flexion. There are four or five fast extensor motor neurones (FEs) and nine fast flexor motor neurones (FFs) in each abdominal hemi-ganglion (Sillar & Heitler, 1985*a*). A large number of these are activated during swimming. However the cellular mechanisms for burst production in swim motor neurones are not known. In addition, the extensor and flexor muscles are innervated by two phasic inhibitory motor neurones, EI and FI, respectively. Their activation, which must play an important role in swimming, has not previously been studied in detail.

In this paper we have analysed the central mechanisms for burst production and the activation sequence of swim motor neurones. Our approach has been to record intracellularly from the somata of these cells during the expression of the swimming rhythm in deafferented preparations. The results suggest that the pools of extensor and flexor motor neurones receive different modes of activation during swimming. The central drive to the phasic inhibitors is also described. This drive ensures that the inhibitors fire at a precise point in the swim cycle.

METHODS

All experiments were performed on the first, second or third abdominal ganglion of the deafferented nerve cord of male or female squat lobsters, *Galathea strigosa*. The preparation was the same as that described in the preceding paper (Sillar & Heitler, 1985*b*). Animals were secured ventral side up in a Sylgard-based dish in cooled (10–12 °C), oxygenated lobster saline. After removing the ventral abdominal cuticle, the abdominal nerve cord was deafferented but left continuous with the intact rostral nervous system and with the uropods and telson. A wax platform was placed beneath the nerve cord and the ganglion under study stabilized with insect pins. In most preparations extracellular recordings were made from the left and right second roots (r2L, r2R) and from the left or right third root (r3L, r3R) with fine polyethylene-tipped bipolar suction electrodes. The second and third roots of abdominal ganglia contain the axons of fast extensor and flexor motor neurones, respectively. It was found difficult to obtain pure fast flexor recordings from the main branch of the third root and occasionally the activity of slow flexor motor neurones in the superficial third root was also recorded. These recordings provided a monitor of the occurrence and timing of the motor programme for swimming. Swimming activity was induced by tactile or electrical stimulation as described in the preceding paper.

Intracellular recordings were made from the somata of neurones on the ventral surface of abdominal ganglia using glass microelectrodes backfilled with 5% Lucifer Yellow (Stewart, 1978) in 1 mol l^{-1} LiCl. Penetrations were made through the ganglionic sheath with electrodes of 20–60 MΩ resistance. After penetrating the sheath, electrode resistance frequently fell to 10–30 MΩ, but this was still suitable for recording. Neurones were routinely stained with Lucifer Yellow by injecting negative current *via* the electrode (10-nA, 500-ms pulses repeated every second). The following results are based on over 50 recordings from flexor and extensor motor neurones.

RESULTS

Motor neurones involved in swimming

Criteria for identification

Since most swim motor neurones form a cluster around the base of the first root (Sillar & Heitler, 1985*a*) it was often difficult to identify a particular motor neurone on the basis of its soma position. Thus penetrated motor neurone somata were identified using a combination of the following electrophysiological and anatomical criteria.

(1) Presence of an antidromic spike with short and constant latency on electrical stimulation of the appropriate nerve root.

(2) A 1 : 1 correspondence of spikes recorded intracellularly with spikes recorded extracellularly in the appropriate nerve root.

(3) Correlation of the firing pattern of the neurone during swimming with the extracellularly recorded rhythm.

(4) Anatomical characteristics of the neurone following injection with Lucifer Yellow. This enabled distinctions to be made between motor neurones belonging to a functionally homologous group, and in some experiments clarified cases where a neurone's firing pattern was not an accurate indication of its type.

Properties of swim motor neurones

Somata penetrated by microelectrodes usually had resting potentials of -45 to -70 mV as measured on withdrawal at the end of a recording. Phasic motor neurones did not spike spontaneously or show any sign of injury discharge upon initial penetration. Normally spikes could not be elicited in response to 10 nA depolarizing current pulses injected through the recording electrode. Injection of up to 100 nA depolarizing current could usually induce spiking, but this was avoided since such large amounts of injected current frequently reduced the subsequent probability of firing of a motor neurone during swimming. Some phasic motor neurones showed a low level of spontaneous synaptic activity in quiescent ganglia, particularly FI. Episodes of swimming activity normally resulted in spiking in these motor neurones. Spikes recorded in the somata reached an amplitude of 2–12 mV and never became positive at peak potential. As with most other arthropod neurones, therefore, the somata of swim motor neurones appear incapable of generating overshooting action potentials (see Kennedy & Davis, 1977, for references).

Fast flexor motor neurones (FFs)

During periods of induced swimming activity, FFs displayed large (5–15 mV) oscillations in membrane potential phase-locked to the peripherally recorded rhythm, with bursts of attenuated soma spikes superimposed upon depolarized peaks (Fig. 1). A few brief, unitary potentials were visible in the depolarizing waveform of the bursting neurones, but these were invariably small in amplitude. The most significant event in the generation of the rhythmic FF bursts appears to be a large, sustained depolarization in which synaptic potentials are not clearly visible. There is no direct evidence, however, to suggest that this depolarization does not result from the

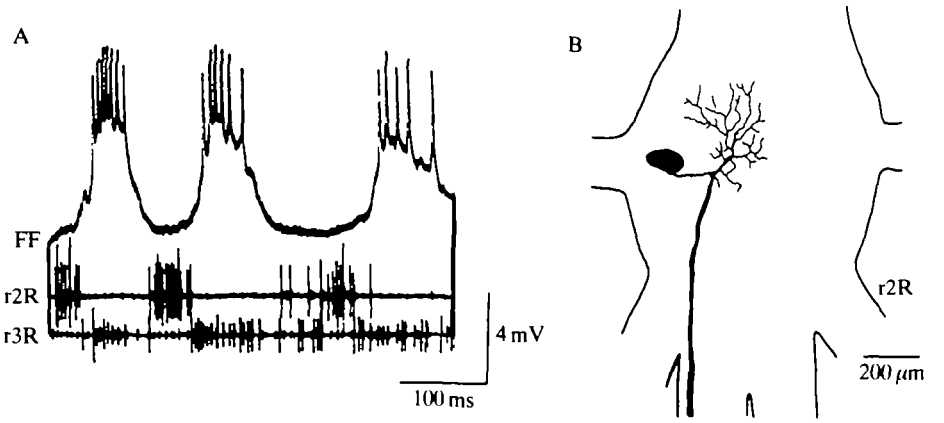

Fig. 1. Physiology and anatomy of an FPI FF active during the swimming rhythm. (A) During a bout of swimming induced by tactile stimulation of the thorax the FF (top trace) displays rhythmical oscillations in membrane potential with bursts of spikes phase-locked to the rhythm recorded extracellularly in the right second root (r2R, middle trace) and right third root (r3R, bottom trace) contralateral to the FF axon. Note the gradual decline in FF spike frequency and amplitude of membrane potential oscillation as cycle period increases. (B) *Camera lucida* drawing of the neurone recorded in A and stained with Lucifer Yellow.

summation of many small unitary synaptic inputs. The rhythmic response of every excitatory FF recorded during swimming was qualitatively the same regardless of whether the motor neurone belonged to the FMC or FPI cluster (see Sillar & Heitler, 1985*a*; Mittenthal & Wine, 1978, for classification; FAC FFs not studied). Most recordings were made from FPI FFs since these were found easiest to penetrate. MoGH, the neurone homologous with the crayfish MoG, is anatomically similar to other FFs, and its firing pattern during swimming closely resembled that of the others in the flexor pool (Fig. 2). Thus each member of the FMC and FPI clusters, with the exception of FI (see below), has a similar firing pattern, and in this respect the FFs are a functionally homogeneous population.

The amplitude of the depolarization underlying bursting varied between preparations. For a given recording there was a clear relationship between the amplitude and the cycle period. In bouts of swimming consisting of two or more cycles, an increase in cycle period was accompanied by a decrease in amplitude and a decrease in instantaneous FF spike frequency (Fig. 1A). The duration of the depolarizing wave also increased as cycle period increased, so that FFs fired for longer and at lower frequency.

There is evidence that the depolarizations underlying bursting result largely from synaptic input to the motor neurones, rather than from intrinsic membrane properties. Injection of constant current *via* the microelectrode for the duration of a bout of swimming changed the amplitude of depolarization recorded in FF somata by up to a factor of 10 (Fig. 3). Thus 10 nA depolarizing current injected into an FF soma greatly decreased the amplitude of membrane potential oscillation (Fig. 3A), while injection of 10 nA hyperpolarizing current greatly increased its amplitude (Fig. 3C). No consistent effects on cycle period were observed. These current-induced effects

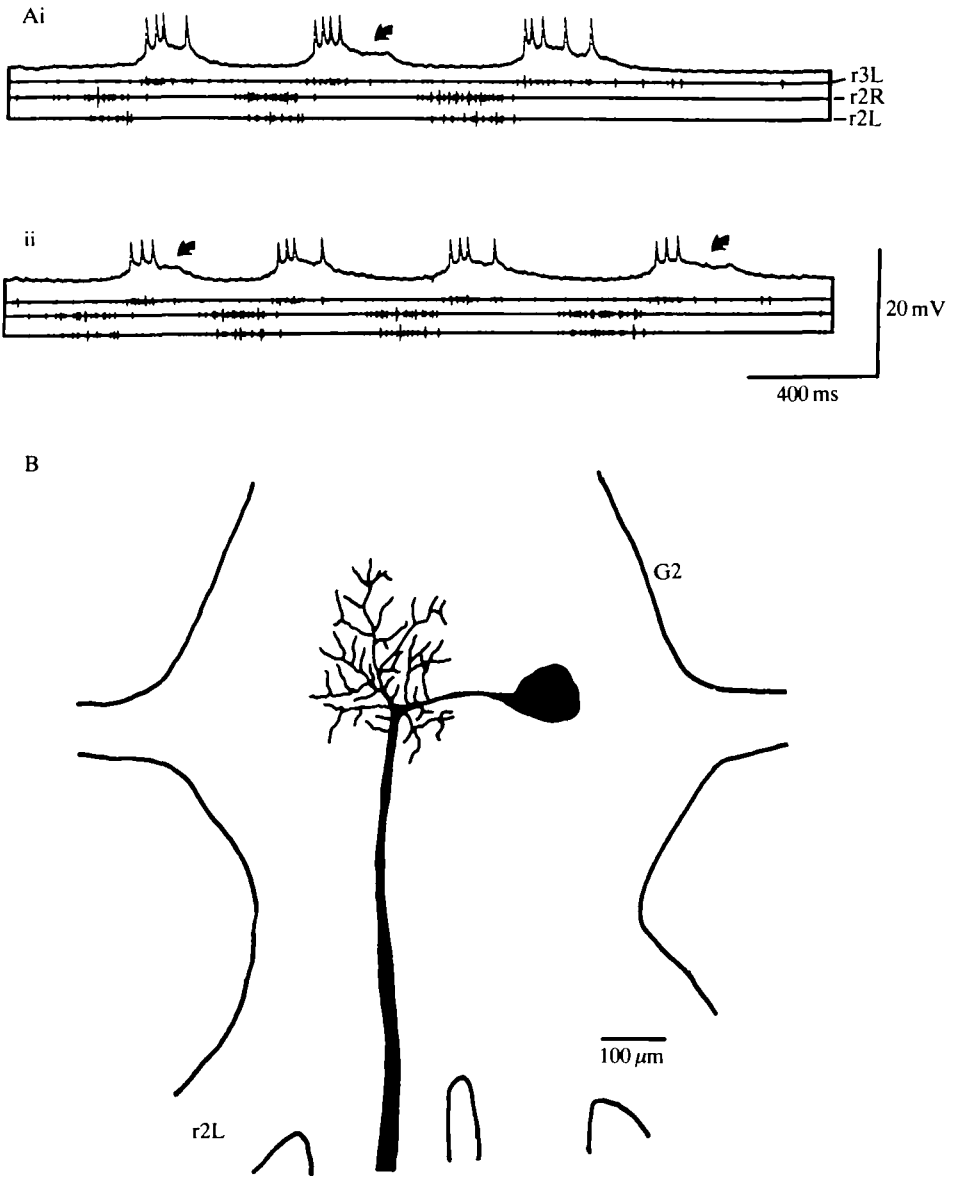

Fig. 2. Anatomy and physiology of the motor giant homologue (MoGH) during swimming activity. (Ai, ii) Two examples from the same preparation of activity recorded in the soma of G2 MoGH (top trace). Extracellular recordings from r3L ipsilateral to MoGH axon (second trace), r2R (third trace) and r2L (fourth trace) monitor the rhythm peripherally. Arrows indicate the depolarization underlying the spike train (see text). (B) The neurone recorded in A was stained with Lucifer Yellow.

on the amplitude of the oscillatory waveform suggest that FFs receive periodic excitation which is mediated by chemical synaptic input.

As well as excitation during burst production, the FFs receive inhibition in the interburst period. This inhibition is particularly apparent if current is injected into

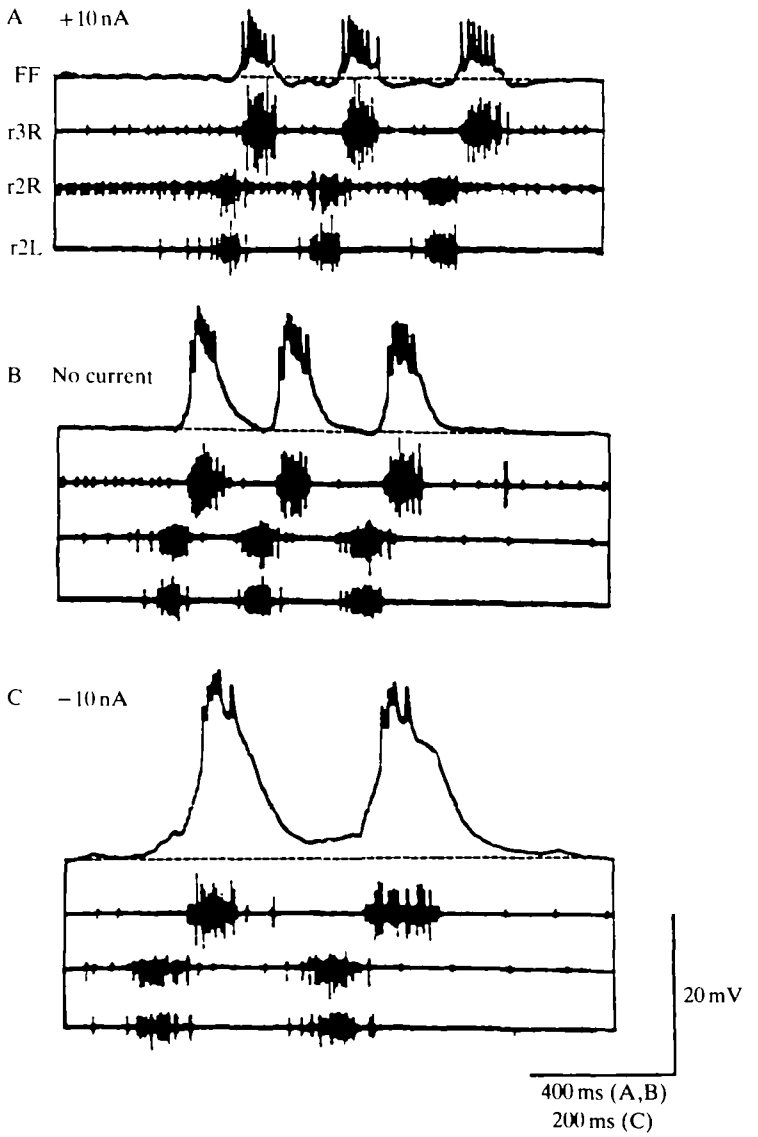

Fig. 3. Effects of 10 nA current (not monitored) injected into the soma of a FPI FF (top trace) during swimming induced by tactile stimulation of the ventral thorax. Extracellular recordings of activity in r3R ipsilateral to FF axon (second trace), r2R (third trace) and r2L (fourth trace) monitor the occurrence of swimming activity. (A) + 10 nA injected into the FF. (B) No current. (C) − 10 nA injected into the FF. Dashed lines represent approximate new resting potential of the FF in each case.

the neurone. Thus with 10 nA hyperpolarizing current (Fig. 3C) the membrane potential did not return to its resting level following the FF burst, but remained relatively depolarized. When 10 nA depolarizing current was injected into the cell the membrane potential became slightly hyperpolarized in the interburst period. This inhibition had two phases – one immediately following the FF burst, the second immediately preceding the next FF burst. The latter phase was coincident with the

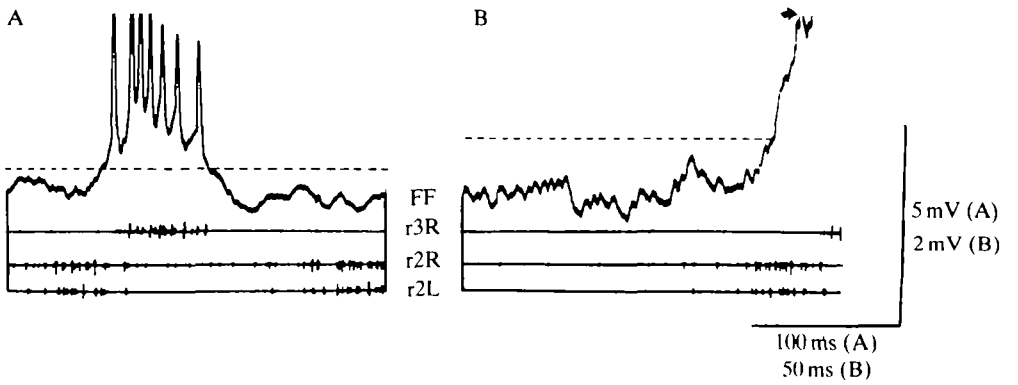

Fig. 4. Inhibitory synaptic input to a FPI FF (top trace) during swimming activity monitored extracellularly in r3R contralateral to FF axon (second trace), r2R (third trace) and r2L (fourth trace). (A) The first cycle in a bout of swimming during which the FF was depolarized by injecting 10 nA current into its soma. Dashed lines represent approximate new resting potential of FF. Note pre- and post-burst inhibition. (B) Expanded portion of pre-burst activity in the same preparation in a different bout of swimming (arrow indicates first FF spike in burst).

extensor burst. Depolarizing current revealed similar inhibition prior to the first FF burst in a bout of swimming (Fig. 4). Thus the FFs were inhibited both preceding and following the excitatory depolarizing input, which presumably ensures that they are effectively clamped near resting potential while the extensors are active. The inhibitory component is not very evident at resting potential, but can be phase-inverted by current injection. In contrast, the excitatory depolarization cannot be phase-inverted, probably because its reversal potential lies close to threshold.

Fast extensor motor neurones (FEs)

Type 1 fast extensors

The majority of FEs fired high frequency bursts of spikes during the extension phase of the swim cycle. These are referred to as type 1 FEs. Spikes recorded in the somata of these neurones were similar in amplitude and time course to those recorded in FFs. However, EPSPs were clearly visible underlying the depolarizations driving the rhythmic firing (Fig. 5). Spike frequency reached a maximum at approximately mid-extension of greater than 50 Hz, and decreased slightly towards the end of each bout. Bursts terminated with a repolarization phase driving the membrane potential towards and occasionally below resting level. In the interburst interval (during flexion phase) unitary synaptic potentials were often evident. At least some of these were IPSPs, suggesting that interburst inhibition occurs. Towards the end of flexion phase the membrane potential was again driven towards threshold. The rate of depolarization driving the FEs in the first cycle of a bout of swimming was usually more gradual than in subsequent cycles (compare the second cycle of Fig. 5A with the first cycle, and with the single cycle of Fig. 5B). This may account for the longer burst duration of the first extension phase of each bout in this and other records of the swim motor programme (see Sillar & Heitler, 1985*b*).

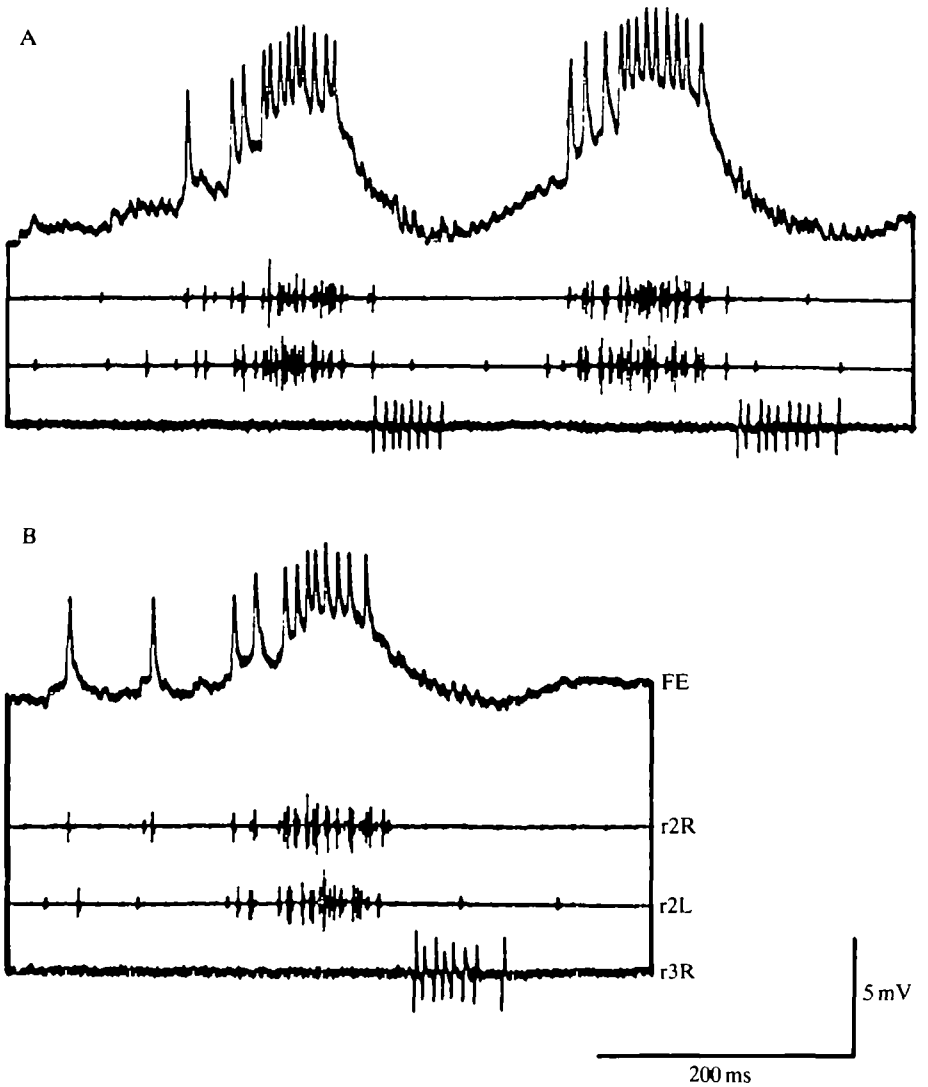

Fig. 5. Type 1 FE activity during the swimming rhythm. (A) Two cycles of activity in response to tactile stimulation of the ventral thorax. The FE (top trace) is depolarized and fires bursts of spikes which increase in frequency towards mid-extension. The rhythm is monitored extracellularly in r2R (second trace), r2L (third trace) and r3R (fourth trace). Note the apparent IPSPs occurring in phase with flexion. (B) A single cycle of swimming activity recorded in the same preparation. Note the long burst duration caused by initial low frequency of firing of the FE. The neurone is depolarized by 10 nA injected current for the duration of this record (B).

Attempts to alter the amplitude of synaptic events in FEs were largely unsuccessful. Any effects caused by the injection of depolarizing or hyperpolarizing current were always small. For example in one preparation peak membrane potential oscillation was 3·5 mV with 10 nA depolarizing current, 4 mV in the absence of injected current, and 4·5 mV with 10 nA hyperpolarizing current. This result implies that type 1 FEs

are driven by periodic chemical synaptic excitation similar to the FFs, but the magnitude of current-induced effects is considerably smaller than in the latter neurones. In each case when this experiment was repeated in good penetrations of FEs with healthy resting potentials, spike amplitude was comparable with FFs. Thus these effects are probably not the result of poor penetrations but may result from properties intrinsic to the membranes of FEs.

Type 2 fast extensors

On several occasions, penetrations were made of an FE which received a barrage of EPSPs driving it towards threshold during extension phase, but which rarely spiked (Fig. 6). On occasions when it did spike (e.g. the third cycle of Fig. 6B), the spike occurred approximately at the mid-extension phase when type 1 FEs were firing near maximum frequency. The occurrence of a high frequency of EPSPs of varying amplitude phase-locked to the peripherally recorded extensor burst implies that during swimming type 2 FEs receive input from a number of premotor elements which discharge almost synchronously. As cycle period increased the initial rate of EPSPs declined (e.g. Fig. 6A, fourth cycle). On three occasions that type 2 FEs were successfully stained they were found to have very large (up to 110 μm diameter) somata

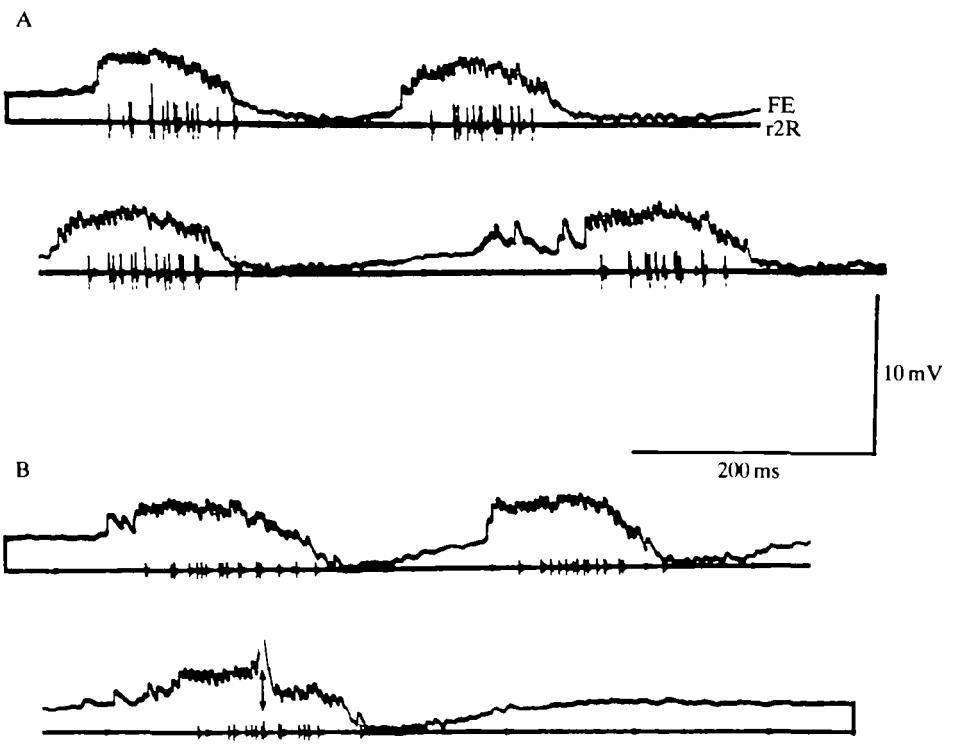

Fig. 6. Type 2 FE activity during the swimming rhythm. (A) and (B) are two examples from the same preparation. The FE (top trace) is driven by a barrage of EPSPs during extension phase, but has a high threshold, and rarely spikes (e.g. third cycle in B, arrows indicate extracellular and intracellular spikes). As cycle period increases the frequency of impinging EPSPs decreases (e.g. fourth cycle of A).

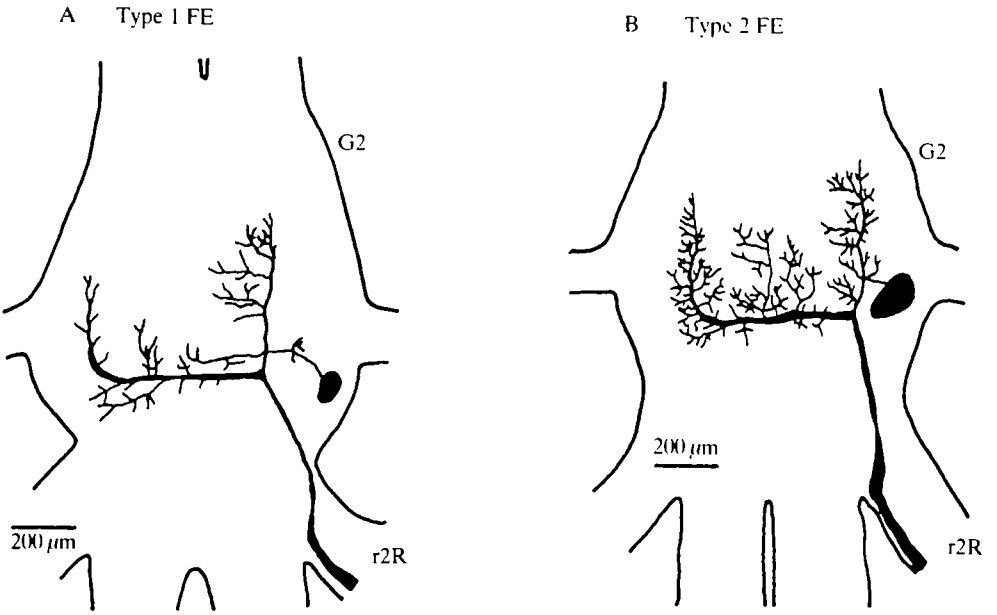

Fig. 7. The anatomy of type 1 (A) and type 2 (B) FEs revealed by staining with Lucifer Yellow after physiological identification during swimming activity. Both types of FE have extensive bilateral neuropilar branches and an axon which leaves the second root (r2) ipsilateral to the soma. The type 2 FE has a larger soma than the type 1 FE. Anterior is at the top.

(Fig. 7). By comparison, the diameters of type 1 FE somata were consistently smaller (50–70 μm). Thus it appears that within the available pool of FEs, those with larger somata (type 2) have higher spike thresholds than those with smaller somata (type 1). The difficulties encountered in recording successfully from FEs have prevented a more quantitative analysis of FE spike thresholds.

Phasic inhibitor motor neurones

Flexor inhibitor (FI)

The phasic inhibitor of the flexor muscles (FI) is one of the largest of the phasic swim motor neurones. The dorsal position of its soma at the extreme lateral edge of the ganglion near the base of the first root greatly facilitates microelectrode penetration. A characteristic feature of FI activity during the swimming rhythm, in contrast to the FFs, was that it gave very few spikes per cycle (usually not more than one), and frequently failed to spike at all in the later cycles of a bout of swimming (Fig. 8). The sequence of events underlying spiking in FI is complex and difficult to interpret. However, FI started to depolarize towards the end of the flexor burst, but never reached threshold until after the flexor burst terminated. FI remained depolarized during the initial part of the extensor burst, but did not spike (at least in our restrained preparations) and then repolarized towards the end of extension, before the next flexor burst started. Thus the central drive to FI ensures that if it spikes at all, it does so shortly after flexion.

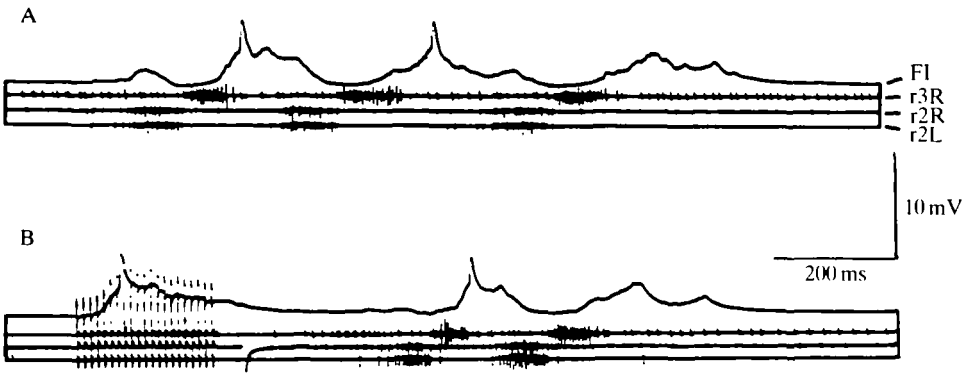

Fig. 8. Activity recorded in the flexor inhibitor FI (top trace) during the swimming rhythm. Extracellular recordings monitor the rhythm in r3R contralateral to FI axon (second trace), r2R (third trace) and r2L (fourth trace). (A) Three cycles of swimming evoked by tactile stimulation. (B) Two cycles of swimming evoked by high frequency stimulation of r2R. Artefacts caused by the stimulus train and switching are visible at the beginning of the record. Note the similarity between FI activity during stimulation and during the first cycle of swimming activity.

Simultaneous penetrations of ipsi- and contralateral FIs showed that many, although not all, of the synaptic potentials were common to both neurones, implying that during swimming at least some input to the two FIs comes from common presynaptic elements. Consistent with this is the observation that, like the other flexors, the dendritic domain of each FI is strongly bilateral. The contralateral

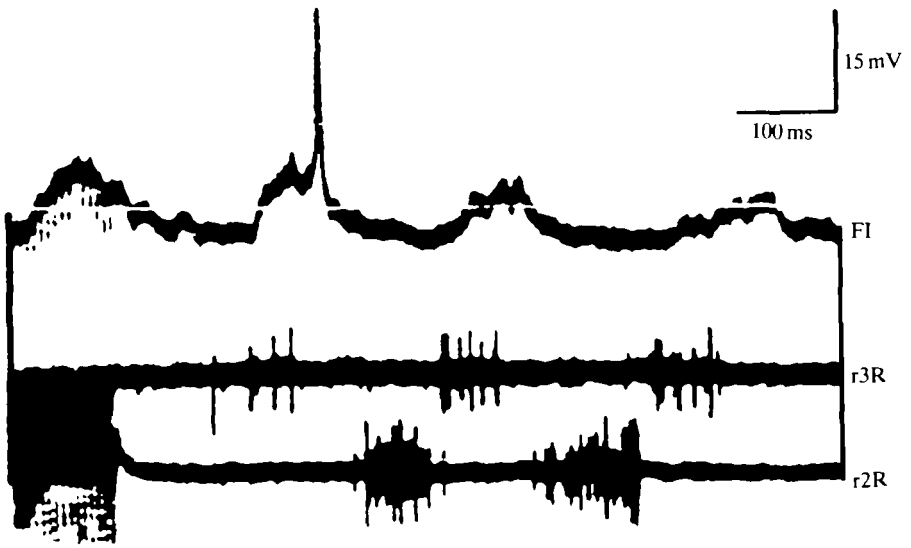

Fig. 9. Activity recorded in an FI (top trace) during a bout of swimming which began with flexion rather than extension. Extracellular recordings from r3R contralateral to the FI axon (second trace) and r2R (third trace) monitor the rhythm peripherally. Swimming was induced by electrical stimulation of r2R.

processes of one FI occupy similar regions of neuropile to the ipsilateral processes of its contralateral homologue.

Sometimes the first flexion of a bout of swimming occurs without prior extension (see Sillar & Heitler, 1985*b*). On one such occasion the initial FF burst was weak, but FI received similar input to that observed during normal swimming activity (Fig. 9). This indicates that neither FF nor FI burst generation is dependent on prior FE activity. Furthermore, in this recording, the most powerful excitation of FI occurred in the first cycle when FF discharge was weak. This suggests that FI activity is not dependent on prior FF discharge either.

Extensor inhibitor (EI)

The phasic inhibitor of the extensor muscles (EI), like FI, has a large, laterally located soma, but stable recordings were difficult to obtain. EI fired a maximum of two spikes in each cycle of swimming and displayed oscillations in membrane potential phase-locked with flexion which resembled the depolarizations seen in FFs (Fig. 10). Injection of current into the soma of EI induced similar changes in oscillation amplitude to those seen in FFs. Dual intracellular recordings from EI and an ipsilateral FF show that many inputs are common to the two neurones (Fig. 11). However the pattern of spiking of the two cell types was different. FFs usually produced a burst of spikes over the broad peak of their depolarization, whereas if EI spiked at all, it did so only on the rising phase of its depolarizing input, and was rarely co-active with the flexors. Synaptic potentials, which may be inhibitory, were often visible later in the depolarization of EI, and these may contribute to prevent its continued spiking. EI may also simply adapt to the continued depolarization. Thus EI was activated prior to flexor activity (although it received similar synaptic drive to the FFs), and after extensor activity.

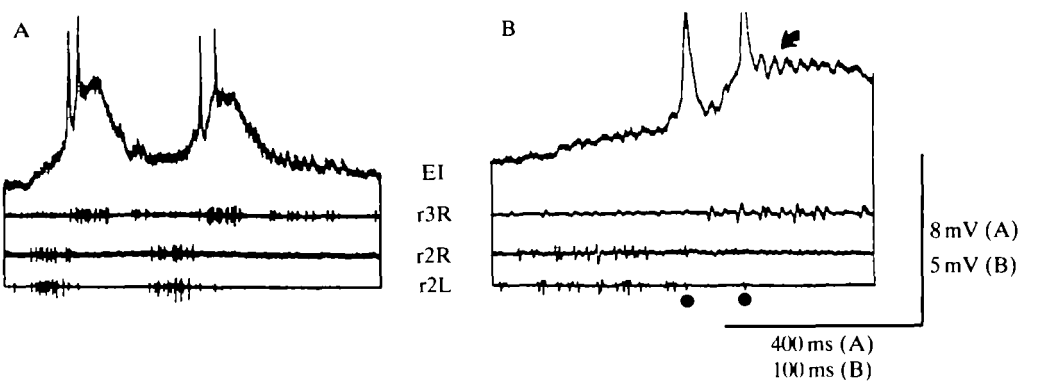

Fig. 10. Activity of the extensor inhibitor EI (top trace) during the swimming rhythm, monitored extracellularly in r3R (second trace), r2R (third trace) and r2L ipsilateral to EI axon (fourth trace). (A) EI is driven by a large depolarization which occurs in phase with flexion and which resembles the depolarizations seen in FFs. EI spikes on the rising phase of this input, rather than at its peak. (B) The EI spikes are followed by a barrage of PSPs (arrow) which occur in phase with the flexor burst, and which may be inhibitory. Dots under fourth trace indicate extracellular spikes of EI. B is an expanded version of the second cycle of activity in A.

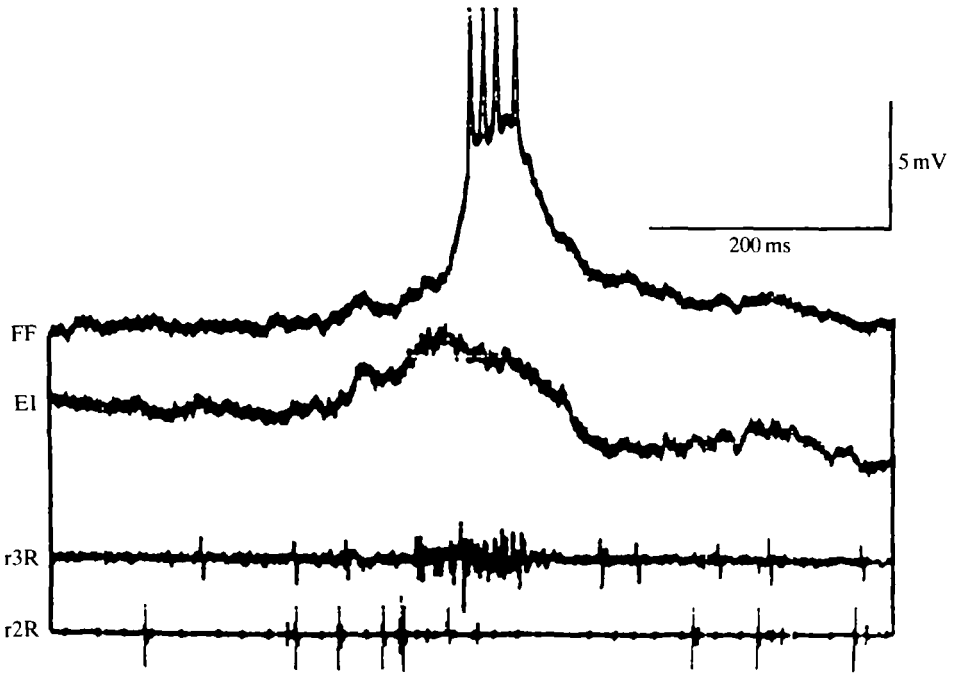

Fig. 11. Dual intracellular recordings from an FPI FF (top trace) and EI (second trace) during swimming activity monitored extracellularly in r3R (third trace) and r2R (fourth trace). The overall depolarizations exciting the neurones are similar, and several inputs appear to be common to the two cells.

DISCUSSION

Mechanisms for burst production

We have recorded from four classes of motor neurone in this study: fast flexor excitors (FFs), fast extensor excitors (FEs), the flexor inhibitor (FI) and the extensor inhibitor (EI). Our identification of neurones is partly direct, and partly a result of clear anatomical homology of neurones in *Galathea* (Sillar & Heitler, 1985*a*) with those in abdominal ganglia of crayfish (Treistman & Remler, 1975; Wine & Hagiwara, 1977; Wine & Mistick, 1977) and lobster (Otsuka, Kravitz & Potter, 1967). There are at least three separate modes of activation underlying burst production in these motor neurones during swimming activity.

The FFs and EI receive a similar, in-phase, large depolarizing input in which individual EPSPs are not clearly visible, but whose amplitude can be substantially altered by injected current. This latter observation suggests that the input is largely chemical in origin, and that it is electrotonically not too distant from the recording site. The relative scarcity of distinguishable EPSPs suggests that the input either derives from non-spiking interneurones, or from high frequency activity of spiking neurones, each of which produces only a small EPSP. The FFs spike throughout their excitatory input, but EI only spikes once or twice in the early part of the cycle. This is probably due to a combination of intrinsic factors (low threshold, adaptation) and

superimposed inhibition which impinges on EI later in the cycle. This inhibition may be synchronous with interburst inhibition in FEs. The result of this activation pattern is that EI spiking is coupled to FF burst production, but always precedes it. This ensures that the extensor muscles are relaxed as the flexor muscles build up tension in the power stroke. The FFs, and possibly EI, receive a brief period of inhibition both preceding and following FF depolarization.

In contrast to the FFs, the FEs receive excitatory input in which individual EPSPs can be distinguished, but which cannot be easily altered in amplitude by injected current. FE somata sizes range from 50 to 110 μm, and those with the smaller somata fire many spikes per cycle (type 1), whereas those with the larger somata fire only a few spikes (type 2). In a number of crustacean motor systems soma diameter has been correlated with the extent of peripheral distribution (e.g. Wine & Hagiwara, 1977). If this also applies to the extensors of *Galathea* then the high threshold of the type 2 FEs may ensure that the neurones with the most powerful and widespread peripheral effects are only recruited during intense bouts of swimming when CPG amplitude is highest. The apparent absence of similar gradations in burst production within the FF motor pool may be a result of the functional difference between power and return stroke. FF bursts usually follow FE bursts at fairly short latency. However, FE bursts do not trigger FF bursts, since the latter can occur without preceding FE activity.

The third mode of activation is found in the flexor inhibitor (FI). Like EI, this neurone has a large soma and only fires a few spikes per cycle. FI receives a complex synaptic input which is unlike that of any other motor neurone. The major excitatory input begins towards the end of the FF burst and usually overlaps with the start of the succeeding FE burst. FI burst termination is not dependent on the following FE burst, since a normally-terminated FI depolarization is often the last event in a bout of swimming cycles. The origins of the pattern of FI activation are not known. However, in crayfish, FFs are electrically coupled to FI (Wine & Mistick, 1977). If these connections are present in *Galathea* they could contribute significantly towards firing FI. The firing pattern of FI may be further modified in the intact animal by input from the abdominal muscle receptor organs, which excite FI in *Galathea via* monosynaptic synapses (Sillar, 1983) just as they do in crayfish (Wine, 1977).

Comparisons with other systems

The demonstration of differing types of central drive to antagonistic pools of motor neurones in the swimming rhythm of *Galathea* contrasts with a number of other rhythmic motor systems in crustaceans. In the swimmeret and scaphognathite CPGs, for example, one set of motor neurones receives similar drive to its antagonists but in approximate antiphase (Heitler, 1981; Simmers & Bush, 1980) and the motor programme is essentially symmetrical. In each of these systems the rhythm can spontaneously switch so that the power stroke becomes the return stroke and *vice versa* (Heitler, 1981; Simmers & Bush, 1983*b*). In *Galathea* flexion is invariably the power stroke of the swimming behaviour.

The excitation of FFs in *Galathea* closely resembles the oscillatory potentials seen in scaphognathite motor neurones (Simmers & Bush, 1983*a*). However the synaptic mechanisms underlying burst production in the two systems are completely different.

Antagonistic levator and depressor motor neurones of the scaphognathite derive their rhythmicity from periodic inhibitory chemical synaptic drive, since injecting increasing levels of hyperpolarizing current causes the rhythmic membrane potential oscillations to diminish and eventually reverse in sign (Simmers & Bush, 1980, 1983*a*). A similar situation occurs in some motor neurones of the pyloric CPG of the stomatogastric ganglion (Selverston & Miller, 1980). In *Galathea*, however, bursting is driven by a combination of excitatory and inhibitory inputs. In neither system does current injection reveal an endogenous bursting capability as in some pyloric motor neurones (Selverston, Russell, Miller & King, 1977).

In the scaphognathite system two classes of non-spiking premotor interneurone have been reported which depolarize in phase with either power stroke or return stroke motor neurone activity. These may be responsible for the smooth membrane potential oscillations seen in the motor neurones (Simmers & Bush, 1980). Although it is possible that FFs in *Galathea* are driven by local non-spiking interneurones, the CPG is located up to several centimetres away from the abdominal motor neurone pools. It seems more likely that the depolarizations seen in FFs result from summated spiking input from a large number of descending premotor elements. The sporadic occurrence of unitary PSPs in FFs may reflect interganglionic interactions of a smaller pool of spiking interneurones.

The swimming CPG in *Galathea* resembles that controlling uropod beating in the anomuran sand crab, *Emerita* (Paul, 1979), in that the two phases of the rhythm are driven by different mechanisms. This is perhaps not surprising since it is possible that these two patterns of behaviour share a common evolutionary origin. Although the uropods are segmental homologues of the swimmerets, their behaviour during beating has evolved from abdominal tailflipping in the related sand crab, *Blepharipoda* (Paul, 1981*a*, *b*). It has been suggested that both types of behaviour derive from macruran tailflipping, and therefore it is likely that they are also related to swimming in *Galathea*. However, there are significant differences between the uropod CPG and that described here. Although direct observations of central drive are not yet available for the two sand crabs, it has been suggested that the uropod CPG drives only power stroke directly, with return stroke being activated on release from inhibition (Paul, 1979). A second difference is that in the deafferented uropod preparation, return stroke follows power stroke with fixed latency and the duration of power stroke is virtually independent of frequency.

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