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

II. THE MOTOR PROGRAMME AND SENSORY FEEDBACK INTERACTIONS

BY KEITH T. SILLAR* AND WILLIAM J. HEITLER

Gatty Marine Laboratory and Department of Zoology, University of St Andrews, St Andrews, Fife, KY16 8LB, Scotland.

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SUMMARY

1. A motor programme underlying backward swimming in the squat lobster *Galathea strigosa* is described. Swimming is accomplished by repeated flexions and extensions of the abdomen. This investigation indicates that the behaviour is generated centrally, possibly in the suboesophageal or thoracic nervous system, and is probably homologous with non-giant escape behaviour in crayfish.

2. The effects of sensory feedback on the swimming rhythm have been investigated in free-swimming and restrained preparations. Proprioceptive feedback, probably originating in the abdominal muscle receptor organs, is involved in the maintenance of high frequency swimming.

3. During swimming, the walking legs and unmodified male swimmerets are rhythmically active in phase with abdominal flexion. Swimmeret 'flicking' in the male is effected by high frequency spiking in a single phasic swimmeret motor neurone. The results suggest that, when active, the central pattern generator for swimming dominates other neural oscillators for rhythmic limb movements.

INTRODUCTION

It is now widely accepted that most rhythmic behaviour results from the interplay between central motor programmes and feedback from peripheral sense organs (Delcomyn, 1980). The essential neural activity underlying a particular rhythmic behaviour is produced by a central pattern generator (CPG), that is located entirely within the central nervous system (CNS) (e.g. Moffett, 1977; Wyman, 1977; Delcomyn, 1980; Grillner, 1981). The resulting motor programme is moulded by peripheral feedback into a behaviourally appropriate form depending upon the prevailing internal and external environment.

Evidence from both invertebrates and vertebrates has shown that sensory feedback is important in the initiation, termination and modulation of central motor

* Present address and address for reprints: Department of Physiology, School of Veterinary Science, Park Row, Bristol BS1 5LS, England.

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programmes (Delcomyn, 1980). Our knowledge of how the diverse array of proprioceptors available to an animal affects rhythmic motor output, is, however, limited.

In crayfish, tailflip escape behaviour involves rhythmic flexion-extension cycles of the long muscular abdomen which result in rapid backward propulsion (Wine & Krasne, 1972, 1982). The behaviour is complex, and two neural systems may be involved in its production. An initial very rapid flexion may be induced by a system of giant axons, followed by a chain reflex causing re-extension (Reichert, Wine & Hagiwara, 1981). A second system, called the non-giant system, mediates all other types of tailflip including the repeated extension-flexion cycles of the abdomen during swimming (Schrameck, 1970; Wine & Krasne, 1972). The non-giant system is thought to be controlled by a CPG located in the suboesophageal ganglion (Wine & Krasne, 1972). Peripheral feedback is an important feature in each escape circuit. A strong sensory stimulus is capable of switching on both giant and non-giant circuits. The transition between giant and non-giant escape results from the different latencies of each system (Reichert & Wine, 1982, 1983). In restrained animals both escape systems are inhibited (Krasne & Wine, 1975). Sensory feedback resulting from flexion is crucial to post-giant extension (Reichert *et al.* 1981). In contrast, sensory feedback onto the extensor system is inhibited during swimming, although at least some proprioceptive feedback is retained in the form of monosynaptic excitatory connections between the muscle receptor organs (MROs) and the extensor motor neurones (Wine, 1977). This may serve functions such as adjusting output to compensate for variations in load (Reichert *et al.* 1981). However the modulation of non-giant escape behaviour by sensory input is poorly understood.

The squat lobster, *Galathea*, escapes from threats by a series of rapid abdominal tailflips which resemble macruran tailflipping (Sillar & Heitler, 1982). In contrast to macrurans, however, this species lacks a giant fibre system. In the preceding paper (Sillar & Heitler, 1985) we have suggested from anatomical evidence that escape in *Galathea* is homologous with non-giant swimming in crayfish and that the specialized giant fibre system found in the Macrura may have evolved from a non-giant circuit like that found in *Galathea*.

In this report we analyse the motor programme underlying backward swimming. The motor programme which is elicited in the absence of sensory feedback is described first. The results suggest that escape is controlled by a CPG located in the suboesophageal or thoracic nervous system which drives extension first, followed by flexion. Next, the roles of proprioceptive and exteroceptive feedback on the motor programme are described in intact animals under free-swimming and restrained conditions. The results indicate contrasting roles for proprioceptive and exteroceptive feedback. Proprioceptive input, perhaps from the MROs, has a predominantly excitatory effect and appears to maintain high frequency swimming, whereas exteroceptive input created by obstacles in the environment inhibits swimming. Finally the behaviour of the walking legs and swimmerets during escape is described. The four pairs of legs and the swimmerets in the male are rhythmically active during escape. This activity, which is significantly different from other rhythmic behaviour involving these appendages, indicates that the CPG for escape swimming has distributed effects throughout the animal and, when active, dominates other local neural oscillators for behaviour such as walking and swimmeret beating.

MATERIALS AND METHODS

All experiments were performed on male or female *Galathea strigosa* (Anomura) measuring 8–10 cm from tip of rostrum to caudal edge of telson.

Deafferented preparation

Intact animals were secured ventral side up to the Sylgard bottom of a Petri dish in cooled (10–12 °C), oxygenated lobster saline, with the abdomen in an extended position. The ventral abdominal cuticle was removed and the underlying layer of chromatophores dissected away to reveal the chain of five abdominal ganglia. All ganglionic roots of the abdomen were cut except those to the uropods and telson. A wax platform was placed beneath the abdominal nerve cord, and the chain of ganglia was secured with insect pins through cut roots. Extracellular recordings of motor output from up to four roots of abdominal ganglia were made simultaneously, either with silver-wire hook electrodes or with fine polythene-tipped suction electrodes. These electrodes could also be used for stimulating.

Electromyograms

Recordings of abdominal flexor and extensor muscle activity and of leg promotor muscle activity were made using pairs of 100-μm diameter copper wires insulated except at their tips. These were implanted either directly into exposed muscles or through small holes made in the carapace. In the latter case EMG electrodes were secured in place with superglue. Following surgery animals were allowed at least 15 min to recover before recordings were made. Further details of different preparations are given at the relevant points in the text.

RESULTS

The motor programme

The squat lobster swims by a series of alternating extension-flexion movements of its abdomen. These movements are driven by segmental muscles exclusively, which are solely innervated through the 2nd and 3rd roots of the appropriate abdominal ganglia. Alternating rhythmic activity could be recorded from these roots after cutting all the abdominal roots. This activity was very similar in its phase relationships within a cycle to that occurring during swimming in the intact animal. We thus conclude that this activity constitutes the motor programme for swimming (fictive swimming), and that it is produced by a CPG. Transection of the nerve cord rostral to the first abdominal ganglion (G1) abolished swimming, while removal of the brain did not. Thus the rostral CNS is essential for swimming but the exact location of the CPG for the behaviour is not known. The integrity of the rostral nervous system means we cannot rule out the possibility that sensory input from this region is involved in the generation of swimming. However swimming could be recorded in preparations in which all major leg nerves had been cut and therefore this possibility seems unlikely. In the majority of experiments nerve roots arising from the last abdominal ganglion

(G5) to innervate the uropods and telson were left intact since this increased the overall excitability of preparations. When these roots were cut, recordings of the motor programme were identical in all essential respects to those recorded in the normal preparation.

Initiating fictive swimming

Fictive swimming could be induced in an abdominally deafferented preparation either by tactile or electrical stimulation. Gentle stroking of the head, ventral thoracic carapace or leg stumps was often sufficient (Fig. 1A). Up to seven cycles have been recorded in this way. An electrical stimulus train (50–100 Hz for 100–200 ms) delivered to sensorimotor 2nd roots of abdominal ganglia *via* extracellular electrodes could evoke fictive swimming with delays ranging between 100 ms and 1 s (Fig. 1B). The motor programmes resulting from tactile and electrical stimulation were very similar, but the latter technique rarely evoked more than three cycles.

Cycle period

Cycle period, measured as the interval between the onset of phasic extension in successive cycles, varied between 100–600 ms. In each bout of swimming, cycle

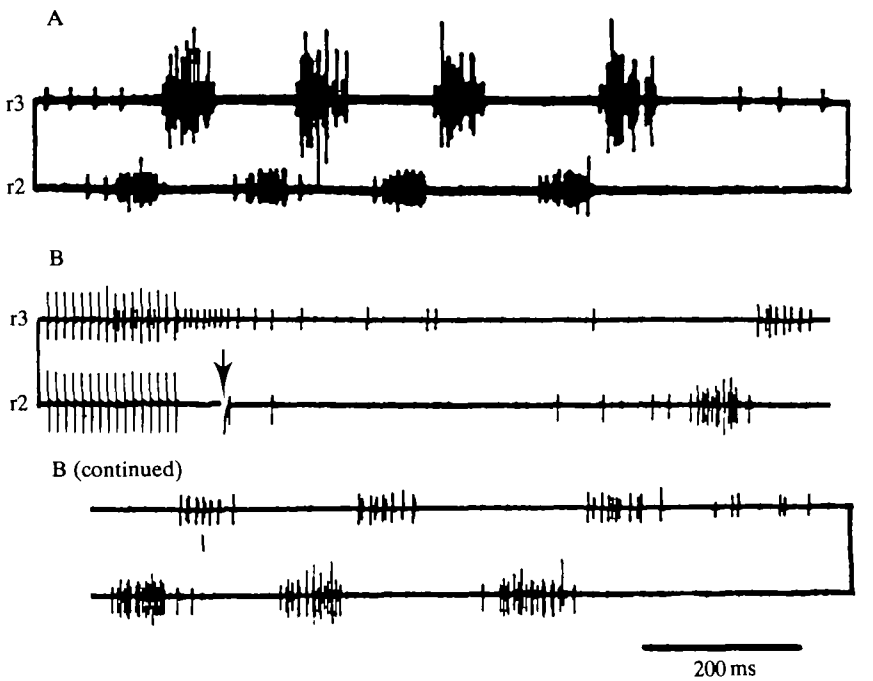

Fig. 1. The motor programme for swimming as recorded from the deafferented roots of G2. (A) and (B) are examples from two different preparations. Swimming activity was evoked by tactile stimulation of the ventral thorax (A) and electrical stimulation of the second root (B, note stimulus and switching, arrow, artefacts). In each record the top trace shows flexor motor neurone (power stroke) activity in the third root (r3) and the bottom trace shows extensor motor neurone (return stroke) activity in the ipsilateral second root (r2).

period initially was short and increased in later cycles, almost doubling over six cycles in most cases (Fig. 2). However this increase did not normally occur between the first and second cycles and frequently the duration of the first cycle was slightly longer than the second.

Extension phase

The duration of extension in the first cycle of a bout of swimming (Fig. 3Ai) was significantly longer than in subsequent cycles (Fig. 3Aii). Initial extension occupied a mean 0·58 of cycle period while in subsequent cycles this was reduced to 0·35 of cycle period. Thus the first cycle of a bout of swimming was often longer than the second. Over a wide range of swim frequencies (about 2–10 Hz) the durations of both initial extensions and subsequent extensions increased linearly with cycle period. The two regression lines cross at a cycle period of 90 ms (Fig. 3A), which is close to the maximum swim frequency recorded. The significantly longer duration of the first extension phase of each bout was due to the activity of a single FE motor neurone which fired earlier in the first cycle than in subsequent cycles (Fig. 3B).

Both fast and slow extensor efferents have axons which exit from the CNS *via* r2. In most recordings of extensor discharge during swimming there was a background level of activity in tonically active motor neurones which was small in amplitude compared with phasic motor neurone activity. The high intensity and short duration of the extension phase made it difficult to measure the precise number of motor neurones active. The approximate number varied between experiments, between

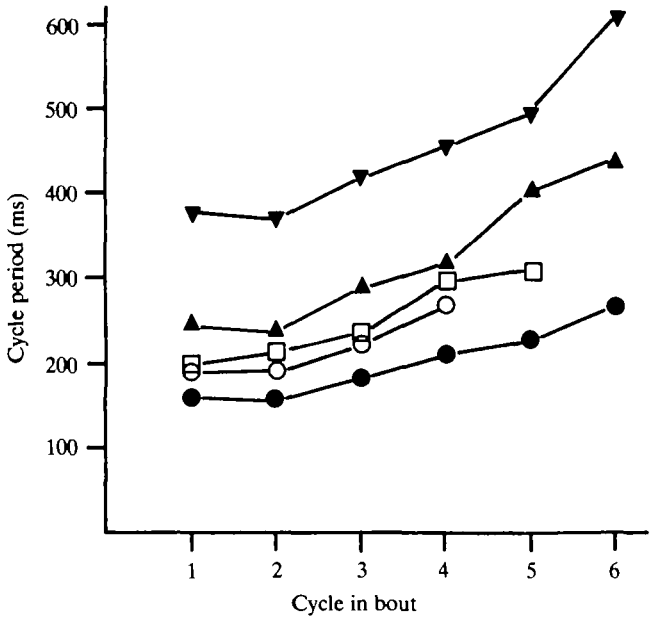

Fig. 2. Graphs of cycle period against cycle number for five bouts of swimming recorded in five different deafferented preparations. In each bout, cycle period was measured as the onset of extension in one cycle to the onset of extension in the following cycle. Note the gradual decline in swim frequency in each case.

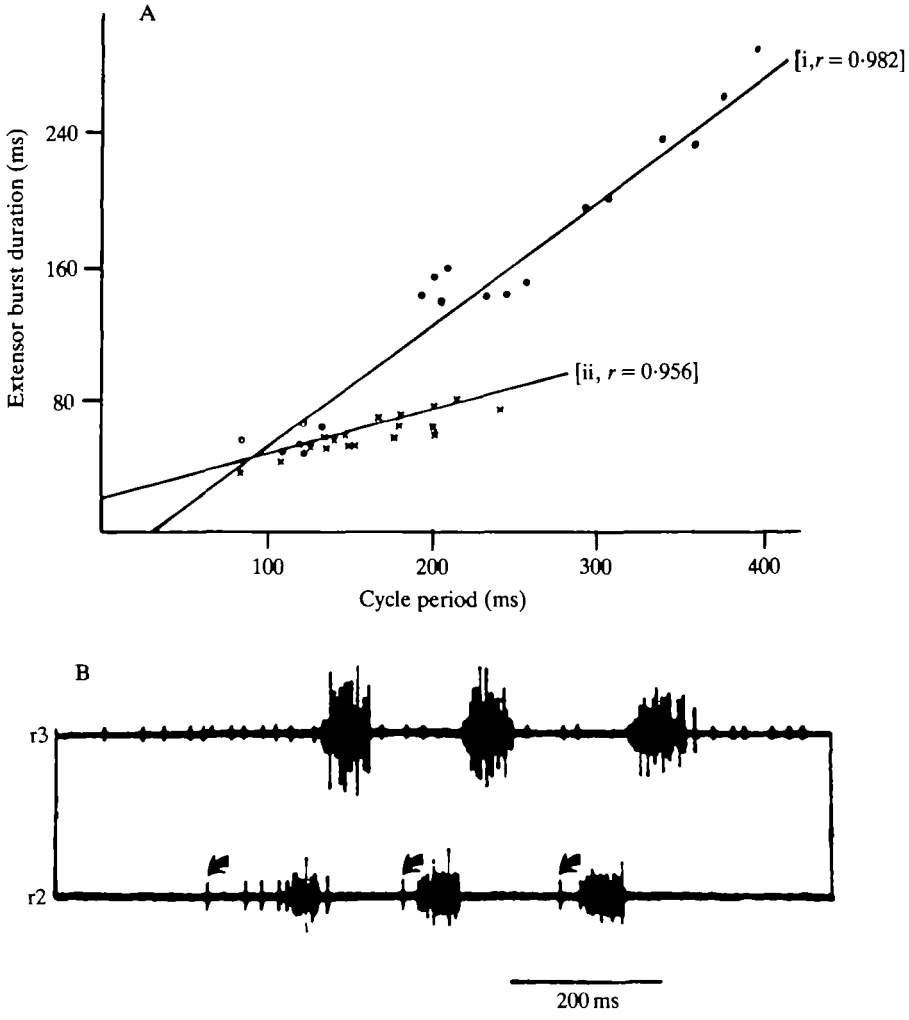

Fig. 3. Extensor burst duration varies linearly with cycle period. (A) Graphs of extensor burst duration *versus* cycle period for the first cycle in a bout of swimming (i) and for all subsequent cycles (ii). The correlation coefficients (r) are highly significant. The extensor burst in the first cycle is usually longer than in subsequent cycles (see text and B for explanation). (B) Sample recording of activity from the second (r2) and third root (r3) during fictive swimming. Arrows indicate a fast extensor motor neurone which fires earlier in the first cycle than in subsequent cycles.

different bouts of swimming in the same preparation and between successive cycles in the same bout. In most recordings larger units were recruited in the latter half of each extensor burst.

Flexion phase

The duration of the flexion phase of the swim cycle also increased linearly with cycle period (Fig. 4A) but was much shorter than extension, occupying on average 0·22 of the cycle period. Unlike extension, the first flexion in a bout of swimming was shorter

than subsequent flexions and each flexor burst occupied a constant proportion of cycle period. During flexion, several phasic motor neurones discharged synchronously and at high frequency. In a number of recordings of r3 activity, one or more slow flexor motor neurones (sF) fired tonically (Fig. 3B). In the examples shown in Fig. 1 a single sF appeared to be inhibited for the duration of each bout of swimming.

The latency between the onset of extension and the onset of flexion increased linearly with cycle period (Fig. 4B). Thus the two components of the swim cycle had constant internal phase relationships over a wide range of swim frequencies.

Bilateral coupling

The motor programme for swimming is bilaterally symmetrical. Paired recordings of extensor or flexor activity from any one abdominal ganglion (G1–G4) revealed that ipsilateral and contralateral pools of homologous motor neurones discharge with a high degree of synchrony. In particular, the extensor motor neurones fire at almost exactly the same time and in some cases it is possible to identify individual units with very similar firing patterns (Fig. 5). On no occasion have asynchronous contralateral motor neurone discharges been observed in recordings from G1 to G4. Thus the facility for directional change which can be seen in the intact, freely-swimming animal does not occur at the level of abdominal motor neurone activity in fictive swimming.

Intersegmental metachrony

A motor output pattern, identical in its intraganglionic phase relationships, could be recorded from both the 2nd and 3rd roots of each abdominal ganglion (G1–G4) during swimming. Simultaneous recordings of homologous 2nd roots from successive ganglia showed a metachronal wave of excitation which passed posteriorly (Fig. 6A). However, the interganglionic latency was not constant; the delay between G1 and G2 extensor activity was less than between G2 and G3 which, in turn, was less than between G3 and G4 (Fig. 6B). The functional value of this latency distribution is not known. However, it probably results in a gradual unfolding of the abdomen during

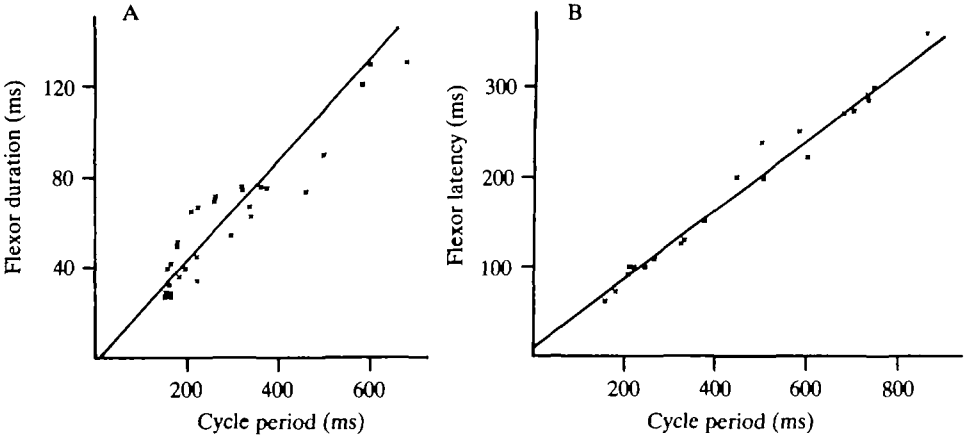

Fig. 4. The onset and duration of the flexor burst varies linearly with cycle period. (A) Graph of flexor burst duration *versus* cycle period ($r = 0{\cdot}955$). (B) Graph of flexor latency from the onset of extension *versus* cycle period ($r = 0{\cdot}99$).

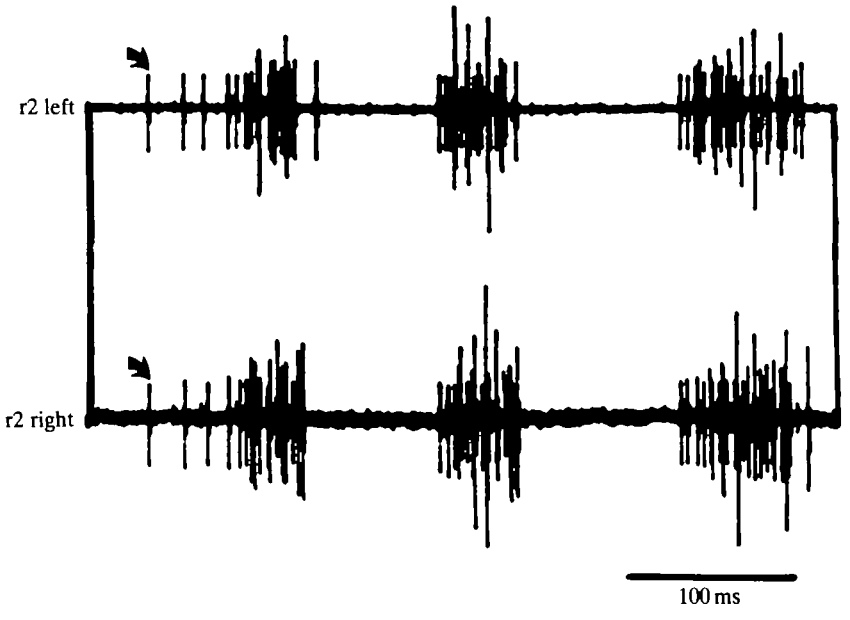

Fig. 5. The motor programme for swimming is bilaterally symmetrical. Paired recordings of ipsilateral and contralateral second roots of G2 show the synchronous discharge of extensor motor neurones during swimming (flexion not shown). Note the gradual increase in burst intensity, with larger units recruited later in each burst. The neurone which fires earlier in the first cycle of swimming (arrowed in Fig. 3B) is recorded on both traces (arrowed) and has a similar spike height and discharge pattern in each case.

extension and might be a mechanism for the generation of minimal counter thrust during the return stroke of the behaviour.

Sensory feedback effects on swimming

In the intact animal proprioceptive and exteroceptive information concerning abdominal position and environmental conditions may modulate the basic, centrally-generated rhythm. Therefore, backward swimming has also been studied under three conditions of restraint to compare the effects of proprioceptive feedback and exteroceptive information on the swimming rhythm.

Free-swimming preparation

In this preparation proprioceptive and exteroceptive feedback were both intact. EMG electrodes were inserted in the appropriate muscles. The animals were then placed in a large Perspex chamber (30 cm × 15 cm × 15 cm) filled with sea water and induced to swim by tactile stimulation of the head or thorax. Swimming was always highly intermittent and a single tactile stimulus rarely resulted in more than three consecutive cycles of activity (Fig. 7B). Extensor activity always preceded flexion, by 20 to 40 ms. Swims were usually terminated through mechanical interference when, for example, the side of the experimental chamber was encountered. When faced with

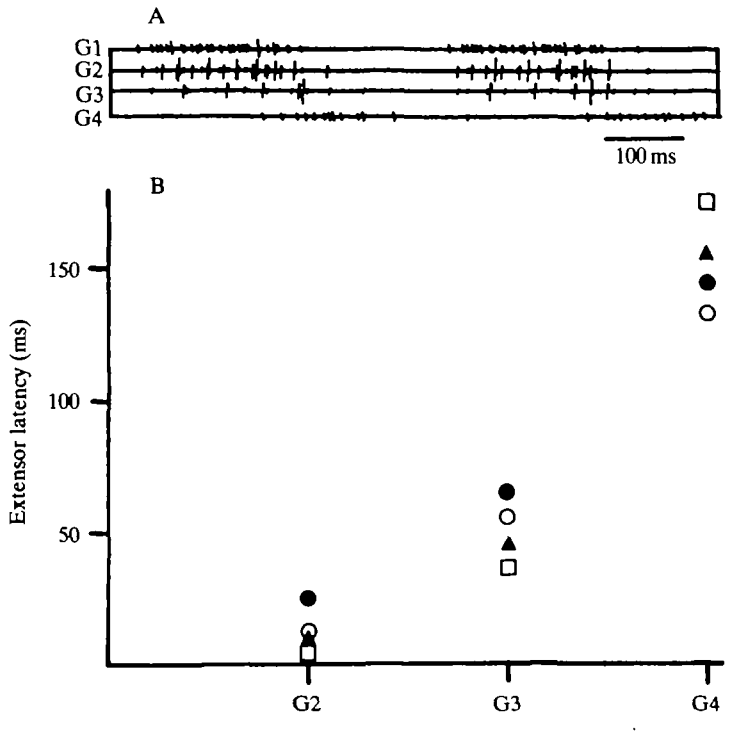

Fig. 6. The motor programme is expressed in G1–G4 as an anterior to posterior metachronal wave. (A) Sample recording of r2 activity during swimming activity in G1 (top trace), G2 (second trace) G3 (third trace) and G4 (bottom trace). Note the increase in interganglionic delay in the caudal ganglia. (B) Graph of extensor latency in G2, G3 and G4 relative to onset of extension in G1 for four cycles of swimming (a different symbol for each cycle). Measurements were made from records similar to those shown in A.

a continuous tactile stimulus animals responded with prolonged periods of swimming despite encountering the side of the chamber repeatedly. Bouts of swimming of this sort usually showed a highly variable cycle period (Fig. 7A). However, when this occurred the relative durations of extension and flexion were always the same and large cycle periods involved a long interburst interval between the end of flexion in one cycle and the onset of extension in the next. Our interpretation of this result is that swimming is highly susceptible to exteroceptive feedback. Once swimming has been initiated, external perturbations are capable of switching the behaviour off. This is substantiated by the experiment shown in Fig. 7C, in which the animal was manually held in one position and induced to swim. After five consecutive cycles a pencil was positioned so as to prevent abdominal extension. Swimming ceased but was resumed as soon as the pencil was withdrawn. Thus, obstructions in the animal's path appear to have a predominantly inhibitory effect on the CPG for swimming.

Semi-restrained preparation

To investigate the role of proprioceptive feedback on swimming, animals were secured ventral side up with their abdomens free to move and with EMG electrodes

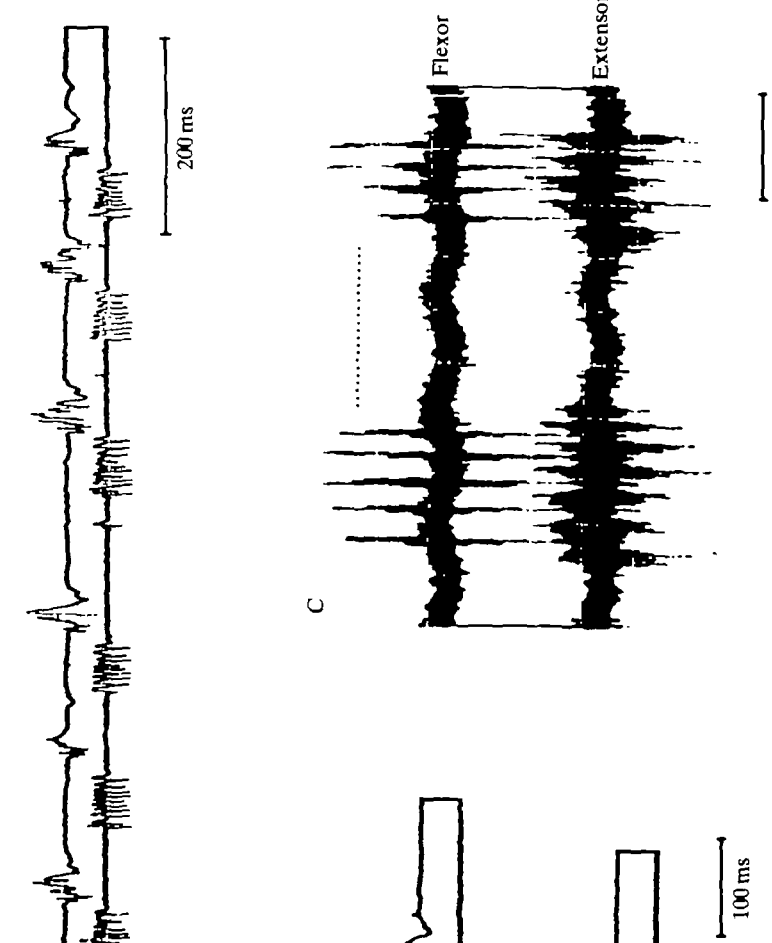

Fig. 7. EMG activity recorded in phasic flexor (top traces) and extensor (bottom traces) muscles in the 2nd abdominal segment of freely-swimming animals. (A) A continuous threat resulted in a series of extension-flexion cycles which began with extension. Note the variable frequency. (Bi, ii) Responses to a single tactile stimulus were usually short bouts of swimming which terminated when the animal contacted the side of the experimental chamber. (C) Semi-restrained preparation. The animal was held by the thorax and induced to swim. A pencil placed against the flexed abdomen so as to prevent extension for the duration of the dotted line inhibited swimming. Note the relatively constant frequency of swimming before and after inhibition (see also Fig. 11).

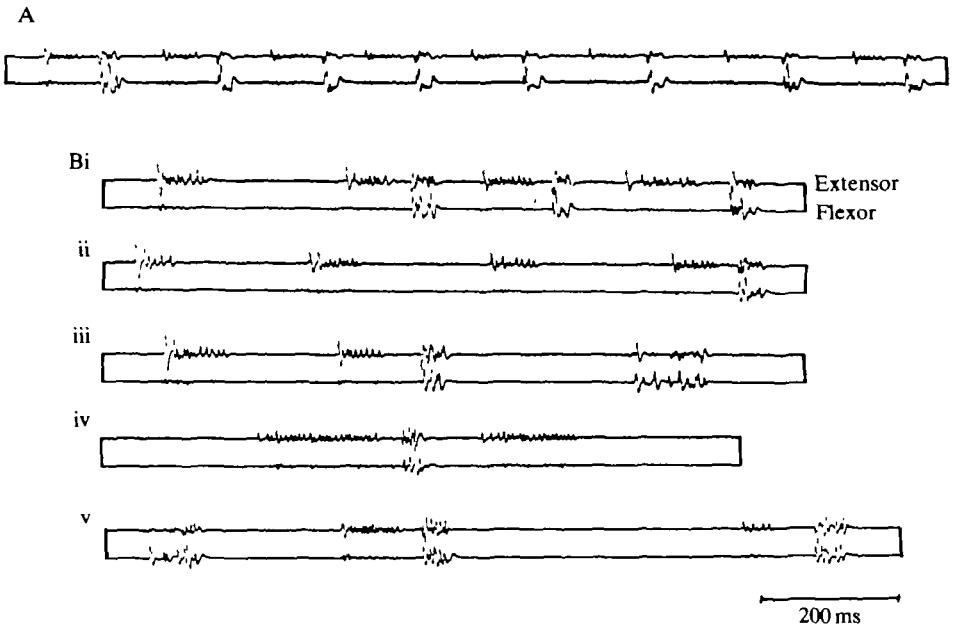

Fig. 8. EMG activity in extensor (top traces) and flexor muscles (lower traces) of the 2nd abdominal segment in a semi-restrained preparation (see text). (A) Typically, a single tactile stimulus to the ventral thorax resulted in constant frequency swimming in a well rested animal. (Bi–v). A variety of responses were recorded in the same preparation after frequency stimulation. Some crosstalk occurs particularly from flexor activity in extensor EMGs.

inserted into phasic extensor and flexor muscles to monitor swimming activity. Hence, in this preparation swimming activity in response to a tactile stimulus generated nearly normal proprioceptive feedback, but exteroceptive input was considerably reduced. The most consistent feature of bouts of swimming recorded from this preparation was that the number of consecutive cycles was much greater than in freely-swimming animals (Fig. 8). This occurred when the animal was either fully submerged under sea water or when all sea water was removed from the experimental chamber. For convenience, recordings were made in the latter conditions. Up to ten consecutive cycles have been recorded in response to a single tactile stimulus to the thorax, although many more cycles have been observed in unoperated animals. Characteristically, frequency remained very constant for the duration of each bout (Fig. 8A). In healthy preparations swimming usually began with extension closely followed by flexion and then repeated high frequency extension-flexion cycling. However on several occasions, particularly in the later stages of an experiment after frequent stimulation, several variations were observed (Fig. 8B). Swimming could begin with up to four bouts of phasic extension, without accompanying flexion (Fig. 8Bi, ii); end in flexion without prior extension (Fig. 8Biii); end in extension without subsequent flexion (Fig. 8Biv); or begin with flexion (Fig. 8Bv). While this may be the result of a deteriorating preparation it nevertheless shows that in certain

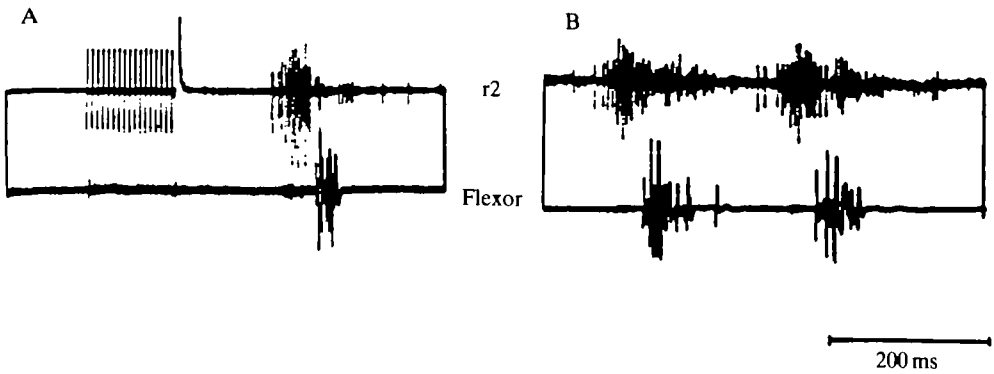

Fig. 9. In fully restrained preparations swimming is partially inhibited, a maximum of two cycles of swimming being recorded. The top trace is r2 G2 activity and the bottom trace is a flexor EMG in the same segment. (A) Swimming activity evoked by electrical stimulation of r2 G2 (note artefacts). (B) Swimming evoked by tactile stimulation of the ventral thorax.

conditions the mechanisms responsible for phasic extension and flexion can be functionally uncoupled at the level of motor output.

Fully restrained preparation

Animals were secured with their abdomens restrained in an extended position. Under these conditions normal exteroceptive and proprioceptive feedback were both disrupted. All nerve roots were left intact and therefore during swimming activity induced by tactile stimulation it is likely that phasic contractions of abdominal muscles generated some reafference. In this preparation swimming consisted of short bouts of phasic extensor and flexor activity similar in phase to those recorded in the preceding preparations. However responses consisted of only one or two cycles of activity (Fig. 9). In each case extension preceded flexion with flexion following at short latency.

Comparison of four preparations

Swimming activity has been analysed in four different preparations: deafferented, free swimming, semi-restrained and fully restrained. In each, the phasing of the rhythm within a cycle was essentially identical, with extension normally preceding flexion. However each preparation displayed swimming activity which differed considerably in frequency and duration (Fig. 10). Deprived of all sensory feedback from the abdomen, swim frequency declined linearly in each bout (Fig. 10B) and bouts normally lasted for 3–6 cycles. In the presence of appropriate proprioceptive feedback, bouts of swimming were longer and swim frequency was relatively constant (Fig. 10C). The addition of exteroceptive feedback (free swimming) resulted in highly intermittent swimming with variable cycle periods (Fig. 10A). When normal proprioceptive and exteroceptive feedback were disrupted (fully restrained) swimming was inhibited (Fig. 10D).

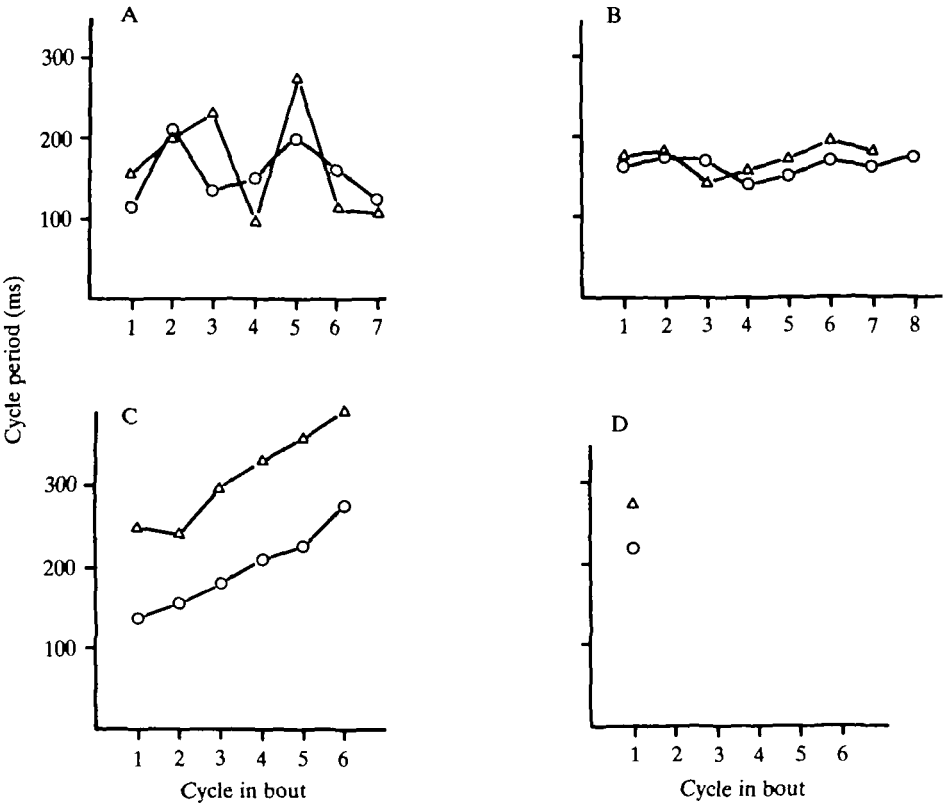

Fig. 10. Comparison of the frequency and duration of swimming activity in each of the types of preparation described in the text. Each graph of cycle period *versus* cycle in bout includes data of two bouts of swimming from different animals. (A) Freely-swimming animals in response to a continuous threat stimulus. Note that frequency is highly variable. (B) Deafferented preparation. Note the gradual decline in frequency. (C) Semi-restrained preparation. Frequency is quite constant. (D) Fully restrained preparation. Swimming activity never lasted more than two cycles.

Coordination of segmental limbs

Activity of the swimmerets

In *Galathea* there are five pairs of swimmerets in the male and four in the female. The swimmerets of the female are all fragile feather-like structures utilized primarily for egg bearing and egg ventilation. In the male the anterior two pairs of swimmerets are also structurally and functionally modified for reproductive purposes (Heitler, Myers & Maitland, 1983) while the posterior three pairs are paddled shaped. During swimming the swimmerets of the female, and the sexually modified swimmerets of the male, are tonically protracted. The paddle-shaped male swimmerets are 'flicked' posteriorly and laterally in phase with each flexion. This rapid retraction is often preceded by a slower and less powerful protraction which occurs in phase with extension.

During episodes of fictive swimming in deafferented preparations recordings from the 1st roots to the modified male and female swimmerets showed a gradual increase in activity which was not rhythmic and which did not follow the swimming rhythm

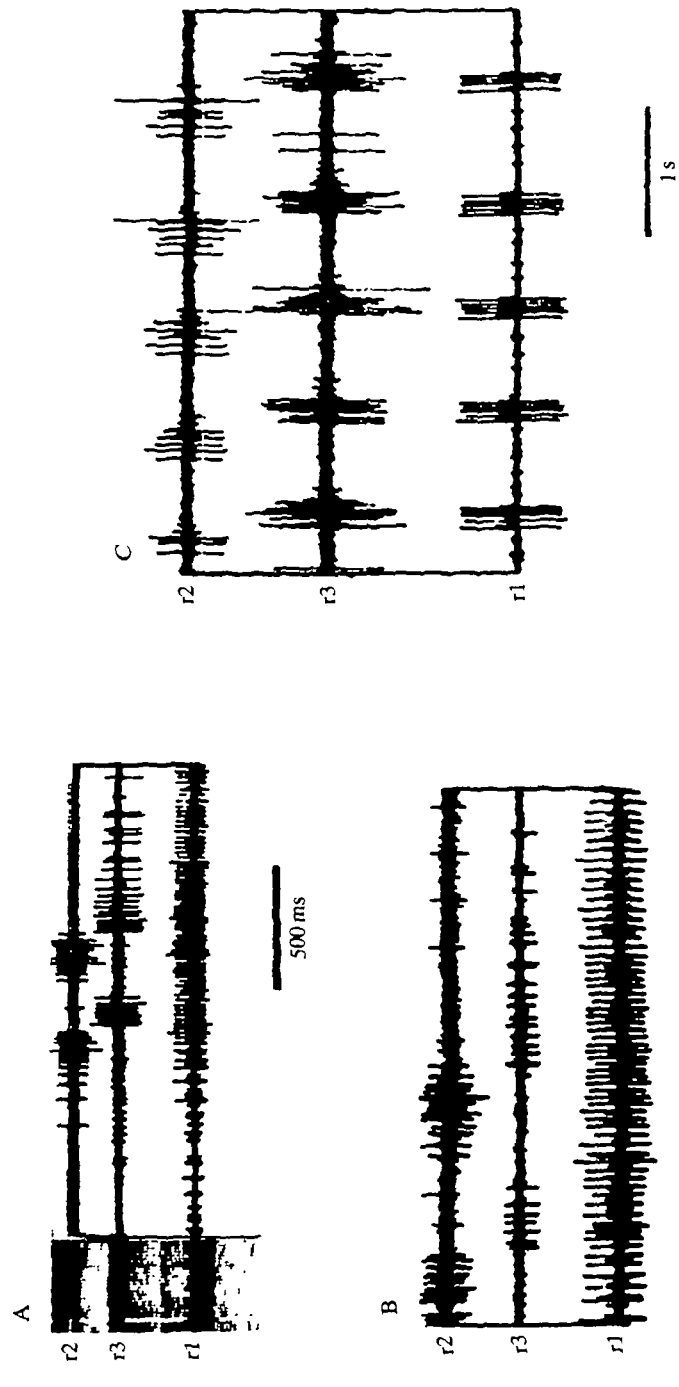

Fig. 11. Neural activity underlying swimmeret movements during swimming. In each of A,B and C the top traces are 2nd root, middle traces are 3rd root and bottom traces are 1st root recordings in deafferented preparations. (A) r1 G1 in females shows a gradual non-rhythmic increase in motor activity. (B) In males the same occurs in r1 G1, which innervates the sexually-modified swimmeret. (C) The unmodified male swimmerets are rhythmically flicked during the flexion phase of the swim cycle. In r1 G2 there is a phasic burst of spikes in a single unit which occurs in phase with flexion. Similar recordings can be made from r1 G3 and r1 G4 in males.

(Fig. 11A,B). In contrast the 1st roots of G2, G3 and G4 which innervate the unmodified male swimmerets displayed oscillatory motor output that was phase-locked to the motor programme for swimming (Fig. 11C). During flexion a single 1st root neurone (called S1) discharged at high frequency. S1 was characterized by a large extracellularly recorded potential, at least double that of other 1st root units in most cases. Dual 1st root recordings during swimming allowed S1 to be identified as a motor neurone with a conduction velocity of 5–7 m s^{-1}. Its characteristic discharge pattern was recorded in many preparations and it appeared to be the only swimmeret motor neurone to be activated during the flexion phase of the swim cycle. Therefore S1 alone must be responsible for the rapid flicking behaviour of the swimmerets during swimming. Since the behaviour involves both retraction and lateral movement S1 may innervate a twister muscle. The number of spikes recorded in S1 during swimming varied in different preparations from none (on rare occasions) to 11 at up to 100 Hz (Fig. 12). Its frequency and duration of spiking also varied with the intensity and duration of the flexor burst, suggesting common or similar drive during swimming.

During the most intense bouts of swimming a number of different swimmeret motor neurones were occasionally active during the extension phase of the swim cycle. This type of activity may be responsible for the less powerful protraction of the swimmerets which can be observed in intact animals during restrained swimming.

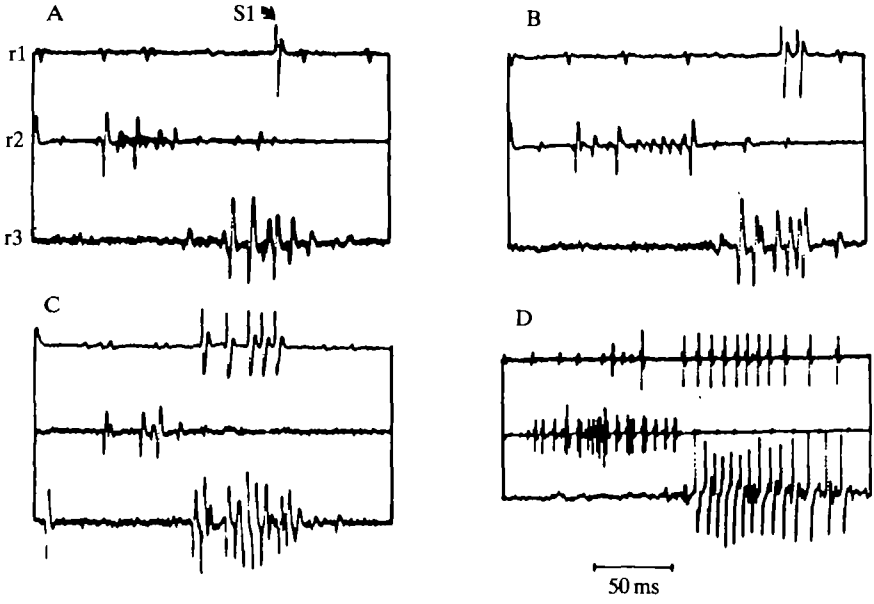

Fig. 12. The activity of the 1st root unit (S1, arrow in A) evoking swimmeret flicking in the male is correlated with flexor motor output. (A) to (D) are four cycles of swimming with increasing flexor burst intensity. Traces are from the 1st root (r1), 2nd root (r2) and 3rd root (r3) of G2. A,B and C are from the same preparation. As flexor burst intensity increases, the frequency and duration of spiking in S1 also increases. Note that spiking in S1 can either follow or lead the onset of flexion.

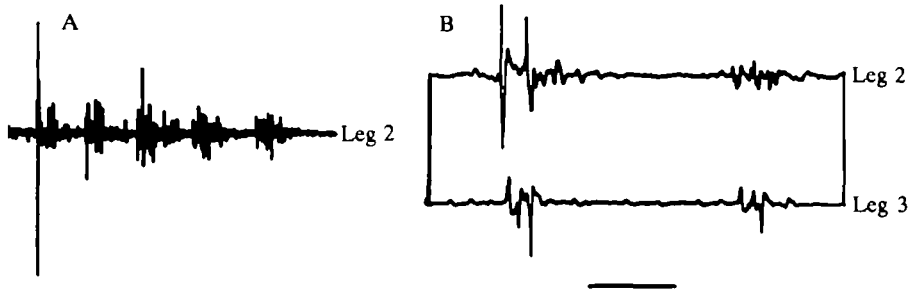

Fig. 13. EMG activity recorded in leg promotor muscles during swimming activity. (A) Promotor activity in leg 2 during a bout of swimming lasting five cycles. Each burst of promotor activity occurred approximately in phase with abdominal flexor muscle activity (not shown). Note the larger amplitude burst on the first cycle. (B) Simultaneous recordings from the promotor muscles of leg 2 (top trace) and leg 3 (bottom trace) during swimming. Scale bar, 200 ms (A), 20 ms (B).

Leg movements

Observations of unrestrained animals reveal that at the onset of swimming the walking legs and the chelae are extended and promoted, presumably in order to reduce drag. EMG recordings from the promotor muscles of a single leg show that this was induced and maintained by bursts of activity occurring in each swim cycle, in phase with abdominal flexion (Fig. 13). The phasic promotor activity lasted for 20–40 ms, and declined somewhat in amplitude with successive cycles. The overall effect was to throw the legs vigorously forward in the first swim cycle, and to maintain them promoted in subsequent cycles. Adjacent legs were sequentially promoted in an anterior-to-posterior metachronal wave, with a latency that was sufficiently short (5–10 ms) to be accounted for in terms of intersegmental conduction velocity.

DISCUSSION

The rhythmical alternating bursts of activity in phasic extensor and flexor motor neurones which underlie backward swimming in *Galathea* appear to be produced by a central pattern generator (CPG) located either in the thoracic nervous system or in the suboesophageal ganglion. In the complete absence of sensory feedback from the abdomen it is possible to evoke activity similar in all essential respects to that recorded in the intact, freely-swimming animal. However, a direct demonstration of central pattern generation in the isolated nervous system has not been obtained in this study. In deafferented preparations extension always precedes flexion, with flexion following at short, variable latency. Each phase of the motor programme occupies a constant proportion of the cycle period over a wide range of swim frequencies, and the latency between phases varies linearly with cycle period. Thus the CPG can be described as a skewed oscillator which drives power stroke and return stroke asymmetrically and oppositely within a cycle. The asymmetry occurs because the extension phase of the cycle has evolved to produce minimal counter thrust and is therefore more complicated than flexion (Wine & Krasne, 1982).

In the preceding paper in this series (Sillar & Heitler, 1985) we have suggested, on the basis of anatomical homologies among abdominal motor neurones, that *Galathea* escape is behaviourally homologous with non-giant swimming in crayfish. The present data provide further support for this notion, since in crayfish the non-giant system is also thought to be controlled by a rostral CPG which drives extension first followed by flexion (Schrameck, 1970; Reichert & Wine, 1982; Wine & Krasne, 1972, 1982). However, it has been shown that flexion follows extension at short and *near constant* latency in crayfish and this contrasts with the present findings. It is possible that the wide range of frequencies in the deafferented preparation permits a more accurate measurement of the latency between flexion and extension. Two species of sand crab, *Emerita* and *Blepharipoda* (Anomura), provide further examples of contrasting decapod escape behaviour from which possible homologies can be drawn (Paul 1981*a*,*b*). *Blepharipoda* performs a rapid abdominal cycling behaviour which resembles macruran tailflipping (Paul, 1981*a*) while the abdomen of *Emerita* remains folded upon itself and swimming is accomplished by rapid sculling movements of the uropods (Paul, 1971). Strong homologies exist between the tailfan neuromuscular systems of *Emerita* and *Blepharipoda* (Paul, 1981*b*) and *Galathea* (Maitland, Laverack & Heitler, 1982), suggesting that the three species and their escape behaviour evolved from a common ancestor. It is intriguing that in *Emerita* the uropod CPG appears to function differently, since in deafferented preparations the return stroke follows the power stroke with fixed latency and the power stroke duration is relatively constant regardless of frequency. It has been suggested that only the power stroke of uropod sculling is driven directly by the CPG, with the return stroke rebounding from inhibition (Paul, 1979).

Segmental limb activity in swimming

The motor programme for swimming is not restricted to activation of the phasic flexor and extensor muscles of the abdomen. There is a simultaneous activation of portions of the segmental limb motor circuitry which is sufficiently powerful to override any rhythmic activity in the walking legs and swimmerets.

During swimming the walking legs are both extended and promoted in phase with each abdominal flexion. It is likely that the major function of this forward thrusting movement is to reduce drag as the animal is propelled backwards. However the fact that leg promotion occurs in phase with flexion (the power stroke) suggests that the legs may also generate some backwards thrust. It is feasible, furthermore, that small changes in the degree of leg promotion and extension could result in lateral steering during escape. The precise role of the thoracic limbs during escape may therefore be more complex than a simple streamlining function.

Concurrent with leg promotion, the paddle-shaped swimmerets of the male are phasically retracted and twisted laterally during the flexion phase of each cycle of swimming. This behaviour is effected by spiking in a single swimmeret motor neurone, S1 (Figs 11, 12). The function of the behaviour is not clear. It is unlikely that such movements of the relatively small swimmerets could contribute to power production during swimming, and one would expect the rapid flexion of the abdomen to force the swimmeret into a retracted position anyway. Therefore we suspect that swimmeret flicking may also have a streamlining function. It is of interest that in the

highly specialized giant fibre system of crayfish a primary central driver neurone, the segmental giant (SG), may have evolved from a swimmeret motor neurone, which subsequently lost its peripheral function (Roberts *et al.* 1982; Kramer, Krasne & Wine, 1981). This neurone, which amplifies and distributes the activity of the giant fibres onto the fast flexor motor neurones, has an axon which runs into the 1st root but then decreases in diameter and fails to reach the swimmeret. Since the giant fibre system of crayfish may have evolved from a non-giant circuit like that in *Galathea*, it is possible that S1 is a primitive homologue of the SG.

Sensory feedback interactions

Sensory feedback has both excitatory and inhibitory effects on the motor programme (Fig. 10). The experimental protocol used in this study was aimed at separating the effects of proprioceptive and exteroceptive information. When proprioceptive feedback is intact but exteroceptive feedback reduced, as in the semi-restrained preparation, swim frequency is normally high and constant and the duration of each bout of swimming is longer than in the deafferented preparation. Thus normal proprioceptive information generated by abdominal cycling has excitatory effects on the CPG for swimming. The particular proprioceptors responsible for this phenomenon have not been identified nor their mode of action investigated. However, for the following reasons the abdominal muscle receptor organs (MROs, Alexandrowicz, 1951) are likely candidates. The MROs are the only major identified proprioceptors in the abdomen which are known to respond to abdominal flexion. In crayfish the MROs monosynaptically excite phasic extensor motor neurones and the phasic flexor inhibitor (Wine, 1977), and hence may increase the firing frequency of these cells during swimming. In *Galathea*, as in crayfish (Hughes & Wiersma, 1960), the ascending branches of the MROs have been traced anatomically to the suboesophageal ganglion and thus project into the region where the CPG is thought to be located. Thus, ascending MRO activity induced by tail flexion may interact with the CPG to set and maintain the overall frequency of swimming. The intraganglionic effects may ensure that motor neurones fire at a frequency appropriate to the output frequency of the CPG.

In the fully restrained preparation, swimming is inhibited but we have not identified either the origin or target of this inhibition. Although the only known effects of the MROs are excitatory (Wine, 1977) it cannot be ruled out that these or other proprioceptors have inhibitory effects when their normal firing pattern is disrupted. It is more likely, however, that inhibition is mediated by exteroceptive feedback. In freely-swimming animals for example, sensory feedback, generated by contact with obstacles in the environment, is sufficient to terminate swimming. Under constant threat, animals swim despite repeated contact with the experimental chamber, but with highly variable frequency. This can be attributed to exteroceptive inhibition–the behaviour is repeatedly switched on by the threat and off by mechanical interference. Restraint-induced inhibition is probably focused on the rostral CNS and not on the motor neurones of the abdomen since it can be overridden by a strong sensory stimulus to produce a restricted number of otherwise normal swim cycles.

Thus there is a dual function for sensory input in the swimming system of *Galathea*. The inhibitory effects of exteroceptive feedback may compensate for

perturbations in the external environment, while proprioceptive feedback is largely excitatory and apparently involved in the normal patterning of activity.

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