J. exp. Biol. **162**, 167–183 (1992)
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ABDOMINAL POSTURAL MOTOR RESPONSES INITIATED BY THE MUSCLE RECEPTOR ORGAN IN LOBSTER DEPEND UPON CENTRALLY GENERATED MOTOR ACTIVITY

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Accepted 7 August 1991

Summary

1. Stretch stimulation of the abdominal muscle receptor organ of the lobster *Homarus americanus* initiated spike discharge of its tonic sensory neuron (SR1). This sensory response evoked a series of tonic postural reflex responses in the motor neurons that innervate the superficial extensor and flexor muscles of the abdominal postural system. The type of motor response depended on whether a flexion or extension pattern of spontaneous activity was being generated by the postural efferents. Spontaneous shifts between these centrally generated motor activities completely changed the SR1-evoked reflex responses.

2. During spontaneous centrally initiated flexion activity, tonic SR1 neuron discharge elicited an assistance response that included excitation of a medium-sized flexor excitor (f3) and the peripheral extensor inhibitor (e5), and inhibition of at least one extensor excitor. Neither the other flexor excitors nor the peripheral flexor inhibitor (f5) were affected by SR1 excitation.

3. During spontaneous centrally initiated extension activity, SR1 activity elicited a response that included excitation of the extensor excitors and the flexor peripheral inhibitor (f5) only, f3 and e5 spontaneous activities were unchanged. This response was a resistance reflex, since SR1 discharge normally resulted from an imposed abdominal flexion.

4. The SR1-initiated control of postural motor activity in lobster differs from previously published results in the crayfish *Procambarus clarkii*.

Introduction

The abdominal muscle receptor organs (MROs) of decapod crustaceans are among the most extensively studied of all proprioceptor systems in Crustacea (see

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Key words: stretch receptors, postural reflexes, reflex reversal, *Homarus americanus*.

Bush and Laverack, 1982; Fields, 1976, for reviews). Each MRO consists of a sensory neuron (SR) innervating a specialized receptor muscle (RM) that runs parallel to the superficial extensor musculature in each abdominal hemisegment. The stretch receptor neurons are excited by changes in tension produced by stretch or contraction of their RMs.

Each abdominal hemisegment contains a pair of MROs whose SRs differ in their sensitivity and speed of adaptation to stretch and in the motor reflexes that they initiate. The sensory neuron (SR1) of the lateral tonic MRO is characterized by high sensitivity to stretch, a low adaptation rate and responsiveness to contraction of the tonic (superficial extensor and flexor) abdominal musculature (Wiersma *et al.* 1953; Fields, 1966). In contrast, the sensory neuron (SR2) of the medial phasic MRO adapts rapidly to stretch stimulation and responds primarily to phasic movements produced by contractions of the phasic deep abdominal extensors and flexors responsible for swimming and tail-flip escape behavior (Wiersma *et al.* 1953; Kennedy *et al.* 1966).

Although the MROs were originally described by Alexandrowicz (1951) in the lobster *Homarus vulgaris*, most subsequent studies of MRO function, including reflex responses of the abdominal postural neurons, were conducted on crayfish, primarily *Procambarus clarkii*. The tonic MRO in crayfish is innervated by at least one superficial extensor motor neuron (SEMN) and several inhibitory accessory neurons (Alexandrowicz, 1967; Fields *et al.* 1967; Jansen *et al.* 1971; Wine and Hagiwara, 1977). Extensive investigations of motor activity produced by SR1 activity in the crayfish have demonstrated three separate reflex responses. These include: (1) an intrasegmental reflex that excites only a single SEMN (SEMN2) and contributes to the maintenance of extended abdominal posture (Fields, 1966; Fields *et al.* 1967); (2) excitation of several phasic efferents that generate the extension component of the tail-flip response (Wine, 1977); and (3) intersegmental reflex excitation of the inhibitory accessory neurons that inhibit the MRO. This has been observed in several species of crayfish and also in the lobster *H. americanus* (Kuffler and Eyzaguirre, 1955; Jansen *et al.* 1970, 1971; Page and Sokolove, 1972).

This report describes the MRO reflex responses of the superficial extensor and flexor motor neurons that control abdominal posture in the lobster *H. americanus*. While it was known that the anatomical position of the MROs relative to the extensor musculature differed between lobster and crayfish (Alexandrowicz, 1951), it was assumed that the basic physiological nature of the MRO was the same in both animals. However, the extensive reflex responses observed in our study, including the excitation and inhibition of reciprocal sets of extension and flexion efferents, provide an unexpected contrast to the very restricted response reported for crayfish. In addition, whereas the crayfish responses were relatively independent of the level and type of centrally generated, postural motor activity (Kennedy *et al.* 1966; Fields, 1976), spontaneous shifts between flexion and extension motor activity in lobsters were associated with changes in the SR1-evoked responses.

Materials and methods

The preparation

Lobsters, *Homarus americanus* (Milne-Edwards), were maintained in circulating artificial sea water at 15 °C. Lobsters weighing 0.5 kg were anesthetized in ice for 30 min prior to severing the abdomen from the thorax. The abdomen was pinned ventral side up in a dissecting dish containing cold, oxygenated lobster saline (Cole, 1941) plus 1 % glucose and the entire abdominal nerve cord, from the first ganglion, A1, to the last, A6, was exposed. All ganglionic nerve roots were severed except for the left second (extensor) root of the second abdominal ganglion (A2), which innervates the muscle receptor organs (MROs) of the third abdominal segment. The ventral sclerite overlying this second root was left intact to minimize stretch damage to the nerve. All branches of the second root were cut except for those that terminate on the MROs. The branch that continues medially past the MROs to innervate the medial head of the superficial extensor muscle was also severed.

The isolated nerve cord with the left second root of A2 attached to the MROs was transferred to the experimental chamber and pinned ventral side up on a Sylgard-coated surface in cold (12–13 °C), continuously oxygenated lobster saline containing 1 % glucose. The dissections were carried out in the late afternoon. The preparation was allowed to recover from the trauma of dissection and manipulation overnight before experimentation. This produced greater consistency in spontaneous and evoked motor activities.

Extracellular and intracellular recordings

At any one time, extracellular spike activity was recorded from the cut ends of up to nine different roots, using polyethylene suction electrodes. These included the superficial third (flexor) roots of A1–A3, the second (extensor) roots of A1 and A2 and the left hemiconnectives (ipsilateral to the stimulated MROs) between the fifth thoracic ganglion and A1, and between A2 and A3. Spike activity in the left second nerve of A2 (which innervates the stimulated MROs) was monitored *en passant*. Two electrodes were attached to this nerve, one close to the MRO and the other near the ganglion, to differentiate between afferent and efferent spikes.

Intrasomatic recordings were made with glass microelectrodes filled with 3 mol l^{-1} KCl (50–100 MΩ) and amplified conventionally. The somata of the superficial tonic extensor and flexor motor neurons, whose axons run in the superficial second and third roots, respectively, were identified either by passing depolarizing current intrasomatically and observing a 1:1 correlation between somatic action potentials and extracellular root spikes or by stimulating the root through an attached suction electrode and recording antidromic somatic action potentials. The locations of the flexor somata recorded in this study have been determined previously. Spikes recorded in the superficial third roots were identified by their relative sizes and activity patterns (Thompson and Page, 1982). While the extensor inhibitor (e5) soma is located near the flexor inhibitor (f5)

soma in the contralateral hemiganglion, the somata of three small tonic extensor excitors are clustered in the ipsilateral hemiganglion. Each of the cells in this cluster was recorded from at least once; no attempt was made to identify them individually. Since all responded similarly, the data for these cells were combined and they will be referred to as small extensor excitors (Se).

MRO stimulation

One end of both RMs (tonic RM1 and phasic RM2) was pinned to the Sylgard-coated bottom of the dish. The approximate length of each RM, in a relaxed state, was 4–5 mm. A piece of dental floss tied onto the other, free, end of the RM was attached to a recorder–galvanometer pen motor. A Grass S44 stimulator was used to drive the pen motor, whose angle of deflection was aligned with the longitudinal axis of the RMs. The typical stimulus was a constant-velocity ramp-and-hold stretch, duration 1.8 s, with an average stretch of the RMs of 10–25 % of their initial length.

Data analysis

For each identified motor neuron, data were collected from a minimum of seven different preparations. Motor neuron responses to MRO stimulation were measured as the change in spike or EPSP frequency. The percentage change was calculated as $[(y-x)/x]100$, where y is the number of spikes either during or after MRO stimulation and x is the number of spikes before MRO stimulation. Time intervals were identical for each set of measurements (typically 1.8 s). The data were analyzed using the Student's paired t-test with significance set at the 5 % level.

Results

Stretch stimulation of the RMs of the MROs always excited one or both of the MRO sensory neurons, the tonic SR1 and the phasic SR2. The resulting afferent spike discharge excited the thick accessory nerve, which inhibits the SRs, and elicited characteristic patterns of activity in the superficial flexor and extensor motor neurons. Before considering the effects of MRO stimulation on the postural motor neurons, spikes produced by stretching the RMs must be identified.

SR spike identifications

Stretching the pair of receptor muscles initiated an afferent response picked up in the second root (Fig. 1Ai, B trace E), which included a brief burst of SR spikes followed by a 'quiet' period when SR spiking was suppressed.

The quiet period was terminated by the resumption of tonic SR1 spike discharge (with occasional phasic SR2 spikes) lasting the duration of the MRO stretch stimulus. SR1 and SR2 spikes were identified as second root afferents because their spikes were always recorded by the *en passant* electrode nearer to the MRO (E) before they were detected by the electrode closer to the A2 ganglion (EG)

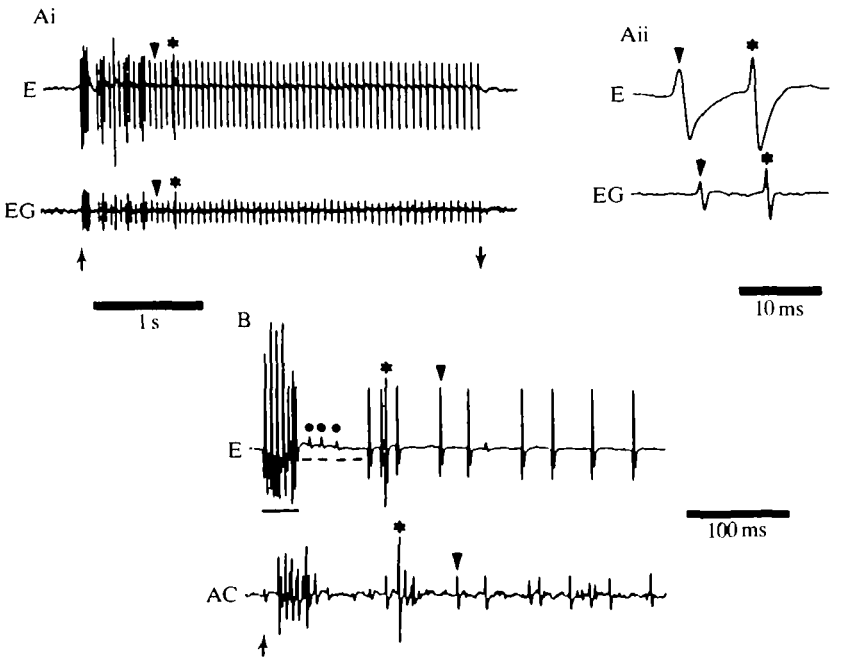

Fig. 1. Responses of the tonic and phasic sensory neurons and accessory nerve efferent to muscle receptor organ (MRO) stretch. (Ai) Traces from two separate *en passant* electrodes attached proximally (E) and distally (EG) to the stretch receptors (SRs) on the second root to monitor MRO afferent and efferent spike activities. The response includes a high level of tonic SR1 spiking (arrowhead) accompanied by occasional large phasic SR2 (asterisk) spikes. The single extra-large spike in the E trace reflects summation resulting from the simultaneous occurrence of SR1 and SR2 spikes at the E electrode. In this and subsequent figures, the little arrows at the bottom of the traces indicate the start and end of MRO stretch stimulation. (Aii) Note the 1:1 correlation between both tonic (arrowhead) and phasic (asterisk) SR spikes recorded from the E electrode and, following a slight delay, with the EG electrode in an expanded portion of the Ai recording. (B) The typical response to MRO stretch included a brief initial burst of tonic and phasic (solid line in E trace) SR spikes followed by a 'quiet' period (dashed line) in which three small spikes (dots) were recorded by the E electrode. Comparison of the E trace with a simultaneous recording from the ipsilateral anterior hemiconnective (AC) between the fifth thoracic and first abdominal ganglia demonstrates a 1:1 correlation between SR spikes in the AC and the E traces.

(Fig. 1Aii). To confirm the identity of the SR spikes, recordings were obtained from the ipsilateral hemiconnective (Fig. 1B), because both SR axons project through the second nerve into the ganglion, where they bifurcate into ipsilateral cephalic and caudal branches that run the length of the ventral nerve cord (Bastiani and Mulloney, 1988). Both SR1 and SR2 spikes, picked up by the *en passant* electrodes on the second root (E), were always correlated 1:1 with SR spikes recorded from the hemiconnectives.

Several very small spikes were always observed during the quiet period when SR spiking was suppressed (Fig. 1B trace E). Evidence that these spikes were produced by an accessory nerve was provided by several observations. First, the small spikes were efferents because they were always detected in the EG electrode (electrode closer to the A2 ganglion) before being recorded in the E electrode. Second, accessory nerve spiking was correlated with increased interspike intervals in the SR1 spike trains (trace E in Fig. 1B), as expected for an inhibitory efferent to the SRs (Kuffler and Eyzaguirre, 1955). Finally, these small spikes were eliminated by cutting the ipsilateral posterior hemiconnective between A2 and A3. The disappearance of these spikes probably results from the interruption of the accessory nerve innervation of the A2 MROs, since their somata are located in A3 and project their axons anteriorly in the ipsilateral hemiconnective to enter the second root of A2 (Alexandrowicz, 1967).

The small 'accessory' neuron spikes should not be confused with other small spikes recorded from the second nerve during central extension activity (see Figs 6A–9A). The latter represents extensor motor neuron spikes which do not disappear when the posterior hemiconnective is cut.

The response of SR1 to MRO stretch had a critical threshold of about 1.5 mm, which reflected an approximately 10% increase in the length of the RMs (Fig. 2A). Additional small increases in RM lengths produced large increases in SR1 spiking (Fig. 2B *versus* 2C). While firing of the tonic SR1 could exceed 50 Hz, strong postural motor effects were consistently obtained whenever the stretch stimulus resulted in SR1 discharge of at least 20 Hz.

Postural motor responses

The effects of SR1 discharge on the flexor and extensor motor neurons varied according to the patterns of 'spontaneous' activity generated by the motor neurons. Two patterns of activity, observed from extracellular root recordings, were observed: flexion and extension. Flexion activity occurred when all six of the flexor motor neurons were spontaneously active and no activity could be measured extracellularly from any of the excitatory extensor motor neurons (however, spontaneous EPSPs were observed in intracellular recordings from the e5 inhibitor). Extension activity included spiking of 3–4 of the small extensor motor neurons and the f5 inhibitor, accompanied by an absence of spiking in the excitatory flexor motor neurons and e5.

In 80% of the preparations, there was continuous 'spontaneous' flexion activity. However, in 20% of the preparations, the spontaneous motor activity shifted several times per hour between flexion and extension (see Figs 6–9), with flexion being the dominant activity. Each period of extension activity lasted for several minutes. These shifts were not associated with any detectable changes in motor neuron membrane potential or the responses of SR1 and SR2 to stretch stimulation. The cause of these switches is unknown.

While the normal preparation consisted of all six abdominal ganglia, preparations containing only a single ganglion (A2) showed identical responses of the

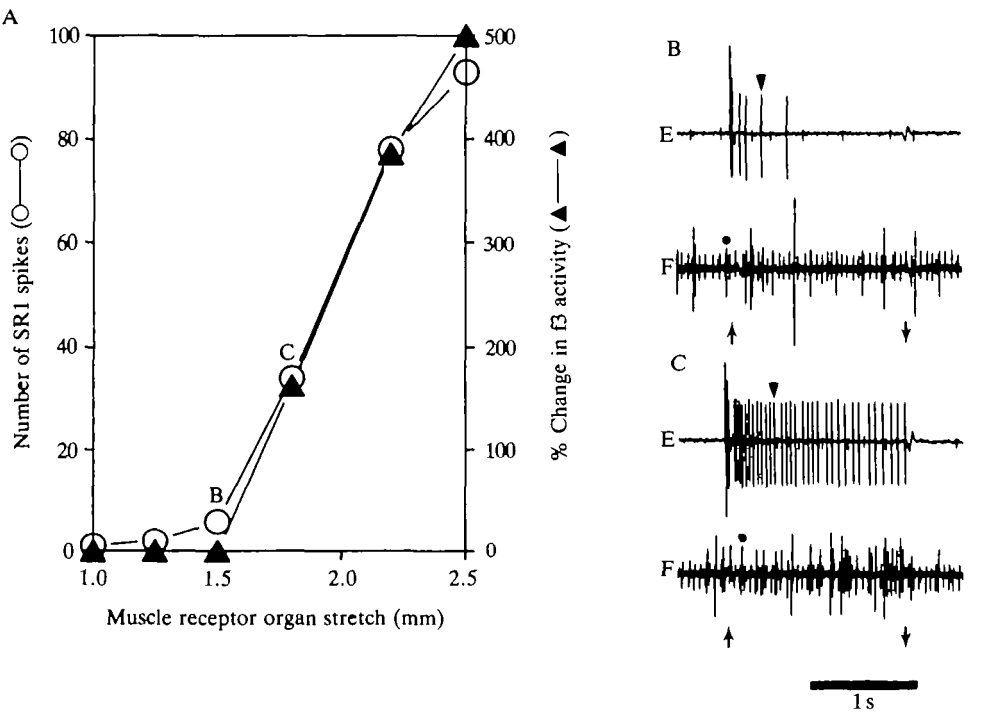

Fig. 2. The effect of increasing the amplitude of MRO stretch stimulation on spike discharge responses of SR1 (○) and the medium-sized flexor motor neuron f3 (▲). (A) Each point on the graph indicates the number of SR1 spikes and the percentage increase in f3 spikes generated during a single RM stretch. (B,C) The traces used to obtain the values for the points that mark 1.5 mm and 1.8 mm stretches, respectively, in A. Comparison of the second root traces (E) shows that a small increase in stretch (0.3 mm) produces a large increase in SR1 spiking (arrowheads). Dots mark examples of f3 spikes in the flexor root traces (F).

postural neurons to MRO stimulation. Therefore, neither the extensive synaptic contacts formed by SR1 and SR2 axons in the terminal (A6) abdominal ganglion (Bastiani and Mulloney, 1988) nor the intersegmental inhibitory interactions known to suppress postural motor responses evoked by mechanostimulation of the swimmeret appendages (Kotak *et al.* 1988) contribute significantly to the postural motor responses initiated by MRO stretch stimulation.

Responses during central flexion activity

Flexors

SR spike discharge excited the medium-sized flexor excitor f3 (Figs 2–4). Intrasomatic recordings from f3 show that this motor neuron responded to SR activation with a slow depolarization that resulted in an increase in its firing frequency (Fig. 3A). For those trials in which spontaneous f3 spiking was absent, MRO stimulation initiated a depolarization of f3 that was accompanied by spike

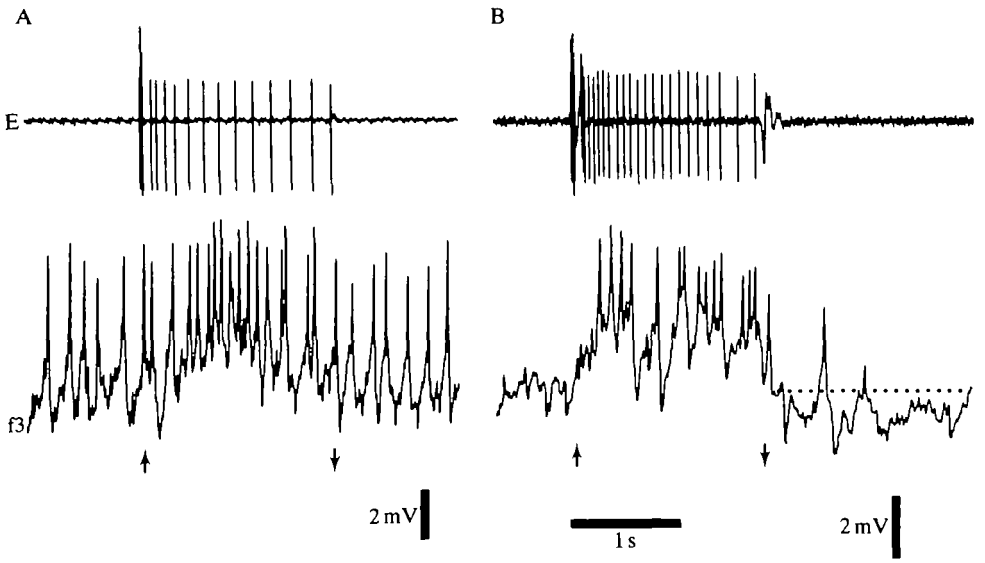

Fig. 3. Responses initiated in a medium-sized flexor excitor motor neuron, f3, by MRO stretch stimulation. (A) When the MRO was stimulated during spontaneous f3 activity, a slow depolarization was generated in f3 and was accompanied by an increase in the frequency of f3 spiking. f3 activity returned to its pre-stimulation level after termination of MRO stretch. (B) In those instances when f3 was silent, it responded with a slow depolarization that led to the discharge of a burst of action potentials. After termination of MRO stimulation, a slow hyperpolarizing 'off' response was observed (dotted line). The dotted line indicates the approximate level of the resting membrane potential in this and later figures.

discharge (Fig. 3B). The delay between the onset of the first tonic SR spike and the beginning of f3 depolarization was 30–50 ms. This long delay and the absence of a 1:1 correlation between SR spikes and motor neuron depolarization suggest a polysynaptic coupling. After termination of the stimulus, f3 activity returned to its pre-stretch level (Fig. 3A). In some instances, a small hyperpolarizing 'off' response was detected (Fig. 3B).

Although the tonic and phasic SR neurons with their associated receptor muscles were not separated and individually stimulated, it is likely that tonic SR1 spiking was the primary source of f3 excitation, since f3 activity was proportional to the frequency of SR1 spike discharge (Fig. 2A). Even a very strong stretch stimulus rarely evoked more than three phasic SR2 spikes.

In contrast, the other flexor excitors (f1, f2, f4 and f6) and the flexor inhibitor (f5) were unaffected even by stretches that drove SR1 spiking at a rate of more than 50 Hz. The absence of any SR-initiated response in these flexors was deduced from the observations that neither their rates of spike discharge (Fig. 4A) nor their intrasomatically recorded membrane potentials (determined for f2, f4 and f5) changed as a result of MRO stretch stimulation.

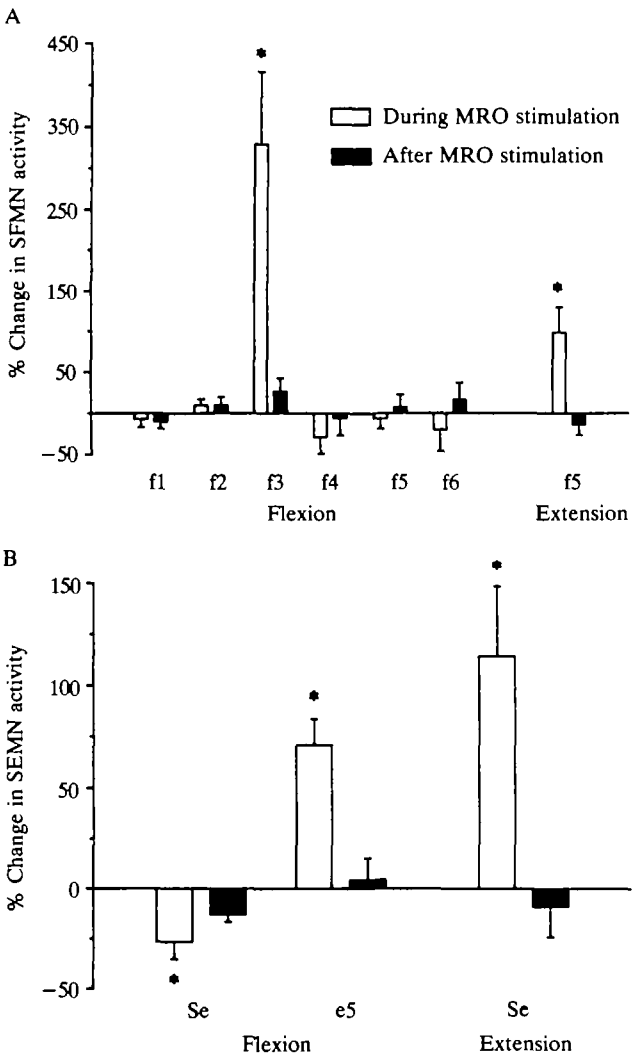

Fig. 4. Histograms summarizing the changes in spike activity of the abdominal postural motor neurons resulting from MRO stretch stimulation during centrally generated flexion and extension motor activity. (A) Superficial flexor motor neuron (SFMN) responses. Only flexor motor neuron f3 activity was affected by MRO stimulation during flexion. In contrast, during extension, only flexor inhibitor f5 activity was changed in response to MRO stimulation (N=10–15). (B) Superficial extensor motor neuron (SEMN) responses. During centrally generated flexion, Se spiking was inhibited and e5 inhibitor spiking increased in response to MRO stimulation. Only Se spiking increased when the MRO was stimulated during extension (N=7–11). Open bars indicate the change in motor neuron spike frequency during stretch stimulation relative to the spontaneous level of spiking before stimulation. Filled bars show changes in the level of spiking after MRO stimulation when compared with spiking before stimulation. An asterisk signifies a significant change in spiking activity when comparing the period before with the period during MRO stimulation (P<0.05). Values are mean+S.E.

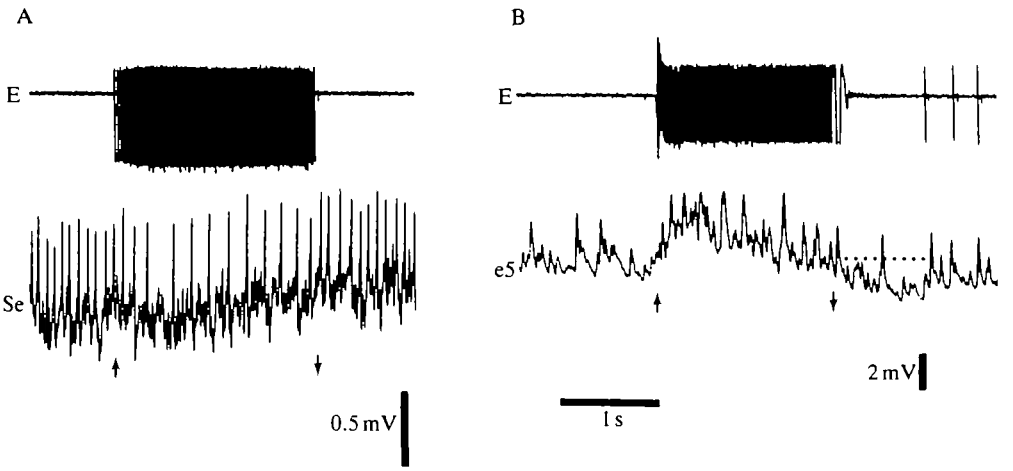

Fig. 5. Responses of small extensor excitors (Se) and the extensor inhibitor (e5) to MRO stimulation during flexion. (A) SR discharge (E) suppressed Se spike activity. After termination of MRO stimulation, spike activity returned to pre-stretch levels. This Se innervated the extensor musculature contralateral to the stimulated MRO. (B) The effect of MRO stimulation on e5, the extensor inhibitor, was excitatory. Slow depolarization of e5 was accompanied by an increased number of EPSPs (e5). An 'off' response (dotted line), included both slight hyperpolarization and suppression of EPSP production.

MRO stretch stimulation affected both the ipsilateral and contralateral f3s. The strength of the response of the contralateral f3 was similar to that described above for the ipsilateral f3. Spread of SR-initiated flexor responses to neighboring segments was never observed.

Extensors

MRO stimulation resulted in the inhibition of the small extensor excitors (Se) and the excitation of the peripheral extensor inhibitor (e5) (Figs 4B and 5). Intrasomatic recordings revealed that SR discharge produced a slow hyperpolarization in the Se, while e5 underwent a slow excitatory depolarization accompanied by an increase in the frequency of EPSPs. SR-evoked excitation of e5 was usually insufficient to cause the discharge of e5 action potentials in our recordings. A small hyperpolarizing 'off' response was frequently observed in e5 when MRO stretch stimulation was terminated (Fig. 5B).

Responses during central extension activity

Flexors

In contrast to the f3 excitation observed during centrally generated flexion motor activity, SR spike discharge had little apparent effect on f3 during centrally generated extension motor activity (Fig. 6). Instead, MRO stimulation excited the flexor inhibitor f5 (Figs 4A and 7). MRO excitation of f5 during extension was less

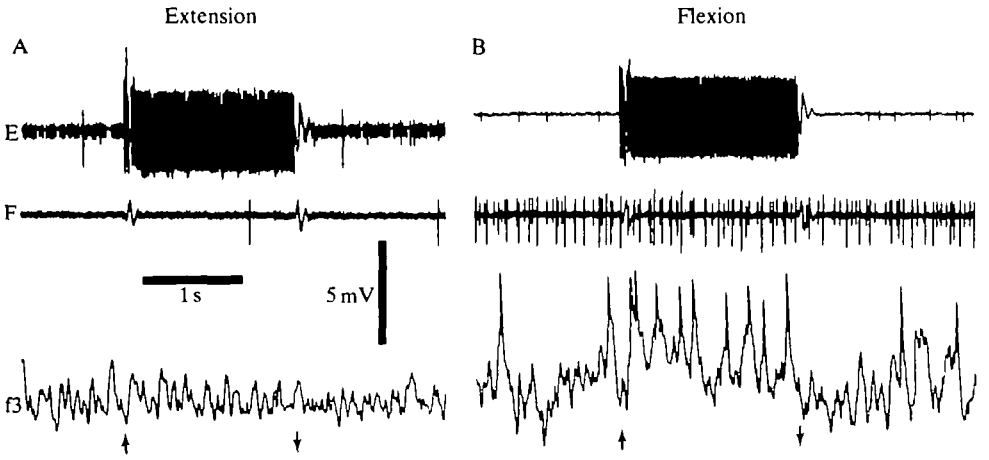

Fig. 6. The effects of switches between centrally generated extension and flexion activities on SR1-initiated excitation of flexor excitor f3. During extension (A), a high level of excitatory extensor spike activity (small units in E) and the suppression of flexor excitor spiking (F) is seen, in contrast with the spike discharge of the flexor excitors and inhibition of the extensor excitors recorded during flexion (B). Spikes seen in trace F in A are due to f5. Intrasomatic recordings from the f3 flexor excitor (f3) show that, in contrast to the f3 excitatory response observed during spontaneous flexion (B), there was no effect on f3 activity during extension (A). Recordings in this and subsequent figures (Fig. 7–9) illustrate responses during and immediately after spontaneous switching between extension and flexion activities; preparation, resting membrane potentials, stimulus strength and duration were unchanged between the A and B recordings.

strong than f3 excitation during flexion (Fig. 4A). There was a statistically significant increase in f5 activity in response to MRO stimulation during extension (N=15); however, there were some preparations where f5 activity was not affected, or even decreased slightly, in response to SR spike discharge (see Figs 8A and 9A). None of the other flexor excitors (f1, f2, f4 and f6) was affected when the MRO was stretched during centrally generated extension.

Extensors

MRO stimulation produced excitation of Se during extension activity, which differed from the inhibitory responses that characterized Se responses obtained during flexion activity (Figs 4B and 8). In contrast to the weak excitation of the e5 inhibitor observed during flexion, intracellular recordings suggest that MRO stimulation does not affect e5 activity during extension (Fig. 9).

Discussion

Stretch stimulation of the muscle receptor organs, similar to that occurring during imposed abdominal flexion, had a strong effect on the activities of the

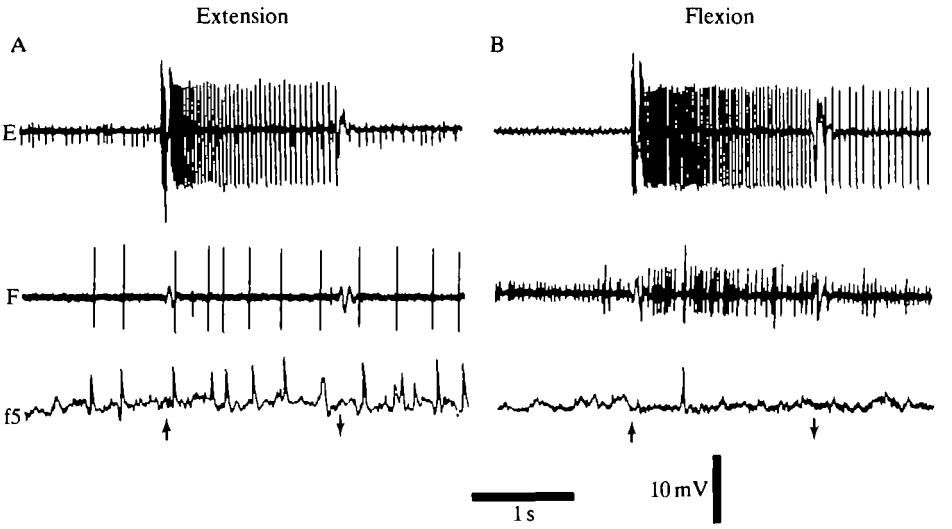

Fig. 7. The effects of MRO stimulation on flexor inhibitor f5 activity during extension and flexion activities. (A) Extension was characterized by spontaneous spiking of the extensor excitors (E) and the f5 inhibitor (f5). Intracellular recording from the soma of f5 and extracellular recordings from the flexor root (F) show that the MRO stretch stimulation excited the flexor inhibitor. (B) During flexion, characterized by discharge of small flexor excitors (F) and suppression of the extensors (E), MRO stretch stimulation had no effect on f5 inhibitor activity (f5) but initiated an intense discharge of f3 excitor spikes (F). The source of the single f5 spike is unknown.

abdominal postural motor neurons in the lobster. Centrally initiated motor activity had a critical role in determining the response of these motor neurons. Spontaneous switches between flexion and extension activity were accompanied by changes in the MRO-initiated intrasegmental motor responses, from a resistance reflex during extension to an assistance reflex during flexion, thus reinforcing the central motor activity. These reflex responses were produced by the reciprocal activation and suppression of sets of motor antagonists.

The evidence presented in this study indicates that there are fundamental differences in the MRO–postural neuron connectivity in the lobster and crayfish. These differences include both the number of motor neurons affected by MRO stimulation and the types of reflex elicited. During spontaneous centrally initiated extension in lobsters, tonic SR1 neuron discharge causes increased spike activity in the Se and the f5 inhibitor, while suppressing responses in f3 and the e5 inhibitor. This is a resistance reflex (since SR1 discharge normally results from an imposed flexion of the abdomen) that involves the appropriate (extensor) excitors and (flexor) peripheral inhibitor. In contrast, SR1 discharge in crayfish evokes a much more limited resistance reflex response, with excitation of extensor excitor SEMN2 only (Fields, 1966; Fields *et al.* 1967). Surprisingly, neither other extensor

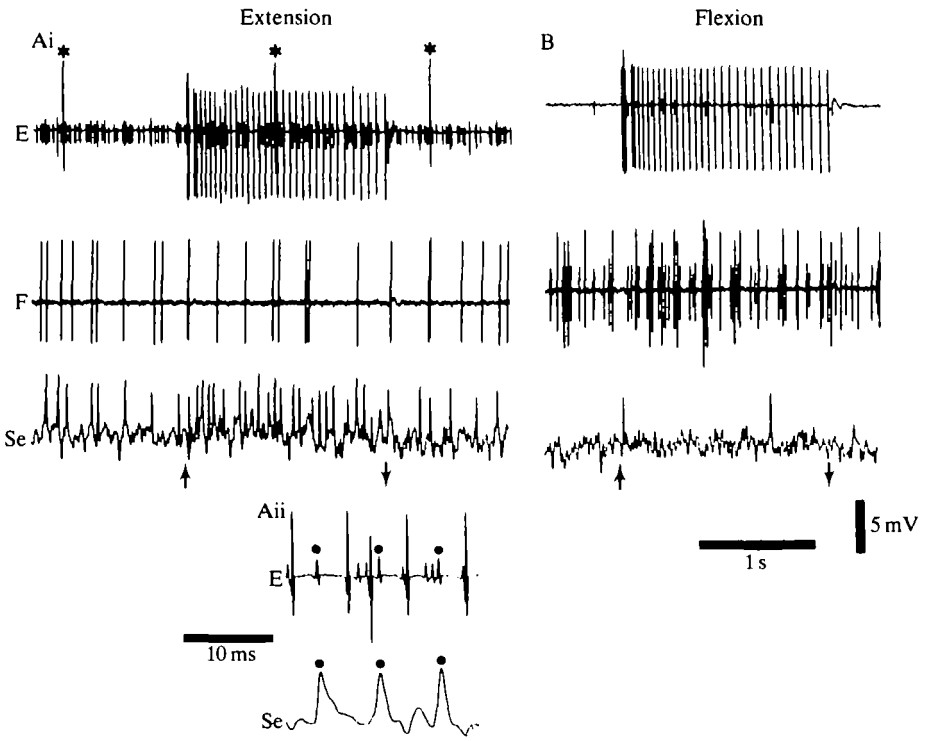

Fig. 8. Responses of extensor excitors to MRO stimulation differ during extension and flexion activites. (Ai) During extension, there is an increase in the level of activity of Se. In this particular preparation, f5 activity did not change (F). (Aii) Expanded section of E and Se traces in Ai to show the correlation between intracellular Se spikes and extracellular spikes in the E trace. (B) In contrast, intrasomatic recording indicated that MRO stimulation had no measurable effect on Se activity during flexion. The two spikes in the Se record reflect random activity and are not stimulus-related. Three phasic spikes that appear to be slightly truncated are indicated by asterisks in A.

motor neurons nor any of the flexors (including f5) were affected by MRO stimulation in crayfish (Fields, 1966; Kennedy *et al.* 1966).

In lobsters, discharge of SR1 during central flexion initiates a completely different reflex response from that observed during 'spontaneous' extension activity. This assistance response includes strong excitation of f3 and moderate excitation of e5, accompanied by inhibition of the Se, with f5 remaining unaffected. The effect of the reflex is to reinforce both the flexion that was imposed on the abdomen to initiate SR1 discharge and the 'spontaneous' central flexion activity, so that it constitutes an 'assistance reflex'. Any similar assistance response is apparently absent in crayfish, since SEMN2 is the only postural efferent affected by SR1 discharge (Kennedy *et al.* 1966; Fields, 1976).

Although the concept of reflex reversal and switches between central motor activity is relatively new, no evidence was found for such reversals in the earlier

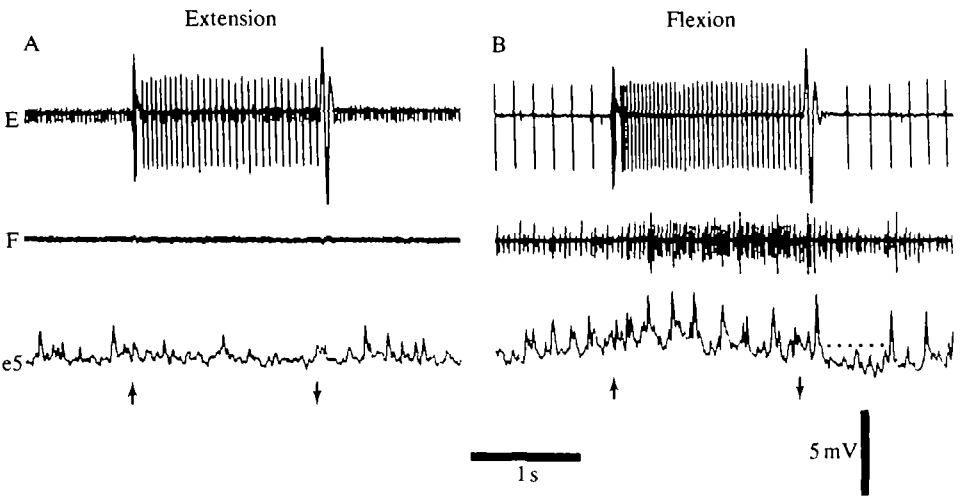

Fig. 9. Change in extensor inhibitor responsiveness to MRO stimulation during extension and flexion activites. (A) Intrasomatic recording shows that e5 excitation was suppressed (e5) during extension. Both the amplitude and the number of EPSPs were reduced during MRO stimulation. (B) During flexion, e5 responded to MRO stimulation with a slow depolarization accompanied by a burst of spikes. A hyperpolarizing 'off' response (dotted line) was observed when the stretch stimulus was terminated. The 'off' artifact was unusually large in this example.

crayfish studies of MRO-initiated postural reflex responses (Kennedy *et al.* 1966; Fields, 1976). Resistance reflexes are well-documented, with examples ranging from vertebrate muscle spindles to many invertebrate sensory feedback systems (Barnes and Gladden, 1985). The term 'assistance reflex' is more recent and was initially employed to describe a reversal of a proprioceptive reflex with a change in the central neural state in a stick insect (Bässler, 1976). Other examples of reflex reversals, generally from resistance to assistance, have been demonstrated in the crab thoracic–coxal leg joint (DiCaprio and Clarac, 1981), the antennal motor system of rock lobsters (Vedel, 1980), the crayfish *Pacifastacus leniusculus* thoracocoxal muscle receptor organ (Sillar and Skorupski, 1986; Skorupski and Sillar, 1986, 1988), the lobster anterior gastric receptor (Simmers and Moulin, 1988), the chordotonal organ in stick insects (Weiland and Koch, 1987) and in spinal cats during walking (Forssberg *et al.* 1975). In all cases, reflex reversal was strongly linked with the type of central program expressed or the level of activity in the central nervous system. The present report shows that this is also the case for the MRO–postural motor system in lobster, in that reflex reversal was always correlated with a change in the central motor activity.

While the source of the changes in central motor activity observed in our study is not known, it is important to note that intact freely behaving lobsters demonstrate not only switches in motor activity, i.e. flexion *vs* extension of the abdomen, but that either position of the abdomen can be held for a very long period. At a

physiological level, several recent studies have suggested possible causes or controlling mechanisms for motor activity switches. In the case of lobsters, shifts between extension and flexion can be produced by direct stimulation of command fibers (Thompson and Page, 1982; Jones and Page, 1986), tactile stimulation of the swimmeret appendages (Kotak and Page, 1986), shifts in body tilt of the lobster *Nephrops norvegicus* (Knox and Neil, 1991) and changes in levels of biogenic amine neuromodulators (Harris-Warrick and Kravitz, 1984). Heitler (1986) has shown that, depending on how a neural program was initiated, i.e. spontaneously or as a result of the stimulation of command fibers, different central pathways may be activated. Heitler (1985) has also reported that changes in motor programs in the crayfish swimmeret system can be initiated at the level of individual motor neurons. Differences in reflexes and types of motor programs may also be related to the 'intactness' of the preparation, i.e. the number of isolated ganglia, the whole nerve cord or the intact animal (Kotak *et al.* 1988). Resistance reflexes appear predominantly in highly dissected preparations while more complex responses are obtained from intact animals. This suggests that resistance reflexes are often suppressed or supplanted by spontaneous central activity (Barnes *et al.* 1972; Bush *et al.* 1978; DiCaprio and Clarac, 1981). Resistance reflexes, evoked during 'unintended' movements as compensatory responses to externally applied forces (i.e. loads), function to correct posture and equilibrium. Intended movements, however, do not activate resistance reflexes for locomotion or changing body posture (Barnes *et al.* 1972).

In this study, the stretch stimulus applied to the RMs reflects an 'imposed' movement and cannot be equated with the voluntary movements of an intact lobster. However, we were still able to demonstrate both resistance and assistance reflexes. Thus, the differences between crayfish and lobster postural responses to MRO stimulation suggest basic differences in the connectivity of the MRO and postural neural systems, which may relate to the different physiology of these two decapod crustaceans. Although crayfish and lobsters appear similar in many ways, they differ in their habitats; crayfish can leave the water and walk on land while lobsters are always submerged in the water. Consequently, for a crayfish on land, the removal of the abdomen from the buoyant water medium acts to increase abdominal weight and thereby force the abdomen into a more flexed posture (Sokolove, 1973). Therefore, it is important in crayfish that the SR1-initiated reflex responses always resist the flexing effects of gravity regardless of the existing central motor program. Since lobsters are rarely, if ever, subject to gravity-imposed abdominal flexion, they have greater flexibility in tailoring their SR1-initiated responses to reinforce the existing motor activity.

We are grateful for the help of Dr V. C. Kotak in the initial stages of the study and Dr M. V. K. Sukhdeo for critically reading the manuscript. This work was supported by a postdoctoral fellowship from the National Sciences and Engineering Research Council of Canada to S.C.S. and Busch Research Grant and NIH grant NS-19983 to C.H.P.

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