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[REVIEW]

The Role of Proprioceptive Signals in the Crayfish Escape Circuit

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ABSTRACT—A single proprioceptor in the tailfan of the crayfish, *Procambarus clarkii* (Girard), innervated by only twelve sensory neurones encodes the position and the direction and velocity of movement of the exopodite relative to the endopodite. Most of the sensory neurones project to, and terminate in, the terminal abdominal ganglion where they form a map in which projection position is based on the velocity threshold of the sensory neurone. The sensory signals from this small proprioceptor have significant effects on the neuronal circuits mediating escape swimming and activate the lateral giant interneurone directly through monosynaptic connections and indirectly via a disynaptic pathway involving a number of interposed intersegmental interneurones. The lateral giant interneurones are activated through electrical synapses whereas the ascending interneurones in the disynaptic pathway are excited through both electrical and chemical synapses. The proprioceptive signals are also responsible for evoking widespread presynaptic inhibition of exteroceptive afferents that reduces the efficacy of their outputs. This pathway therefore reduces afference caused by water movement as a result of an animals own escape movements. Movements of the chordotonal organ also lead to a delayed input to giant motor neurone that is timed to occur during flexion movements of the abdomen. Thus not only do the proprioceptive signals activate the escape pathway leading to a tail-flip, but they also protect it from unwanted sensory input, and may also prevent depression of its neuromuscular synapses.

INTRODUCTION

Crayfish escape from predators and threatening stimuli by producing a series of escape movements, or tail-flips, that consist of short latency rapid flexions of the abdominal segments that propel an animal away from the threat. The first tail-flips in a series are mediated by two pairs of giant interneurones, the lateral (LG) and medial (MG) giant interneurones. These interneurones generate tail-flips with very short latencies of 15–20 ms from the initial stimulus to the start of abdominal flexion. The MGs are activated by mechanical and visual stimuli to the rostral part of an animal and produce a swimming stroke that moves the crayfish directly backwards away from the point of stimulation. On the other hand, the LGs are activated by stimuli to the tailfan and posterior abdomen, and their activation leads to a swimming stroke that pitches the crayfish up and forwards (Wine and Krasne, 1972). A third class of tail-flip, the non-giant tail flip, occurs with a much longer latency (80–500 ms) and is produced during longer swimming sequences (Reichert and Wine, 1983). As the name implies this form of tail flip is produced without the participation of the giant interneurones.

Few neuronal networks have received the same detailed analyses as that generating the escape response of crayfish (see recent review by Edwards *et al.*, 1999), however, all studies that have analysed the contribution of sensory neurones to the excitation of the giant interneurones have focused on exteroceptors, and have therefore overlooked the potential role of tailfan proprioceptors in the escape circuitry.

The tailfan consists of the terminal appendages, the uropods, which are composed of a basal region, the protopodite, and two blade-like structures, the exopodite and endopodite, and the telson. The uropods are involved in a variety of behaviours, from equilibrium reflexes such as righting and steering to defensive displays and escape. They have a relatively large surface area and are able to generate substantial forces that lead to the high velocity escape movements necessary for swimming (Bowerman and Larimer, 1974; Wine

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and Krasne, 1972). The surface of the tailfan is covered with different types exteroceptive hairs, some of which respond to touch while others respond to water movements, and these have been the subject of intensive analysis in previous studies on activation of escape swimming (see review by Wine, 1984). Spanning the joints of the tailfan, however, are a number of internal proprioceptors that monitor the relative movements of different parts of the uropod, and also the movements of the uropods relative to the abdomen and telson (Field *et al.*, 1990).

In the last few years there has been considerable progress in our understanding of how proprioceptors regulate or control locomotion. That understanding has come primarily from analyses on walking in cats, stick insects and crayfish (see Pearson, 1995 for a recent review; Burrows, 1987; El Manira *et al.*, 1991). The role played by the tailfan proprioceptors in escape movements, however, was until recently poorly understood. We have focused our analyses on just one of those proprioceptors and here review what we currently know of its role in the escape circuits of crayfish.

Proprioceptors in the tailfan

Several proprioceptors have now been described in the tailfans of decapod crustaceans (Barth, 1964; Field *et al.*, 1990; Laverack, 1989; Maitland *et al.*, 1982). A single chordotonal organ spans the exopodite-endopodite joint (the so-called exopodite-endopodite chordotonal organ or exo-endo CO) that consists of a strand of connective tissue that is innervated by a small branch of nerve 3 (Fig. 1) (Field *et al.*, 1990). The receptor is innervated by approximately 12 sensory neurones that encode the position, velocity, direction and probably acceleration of movement. Most of the twelve sensory

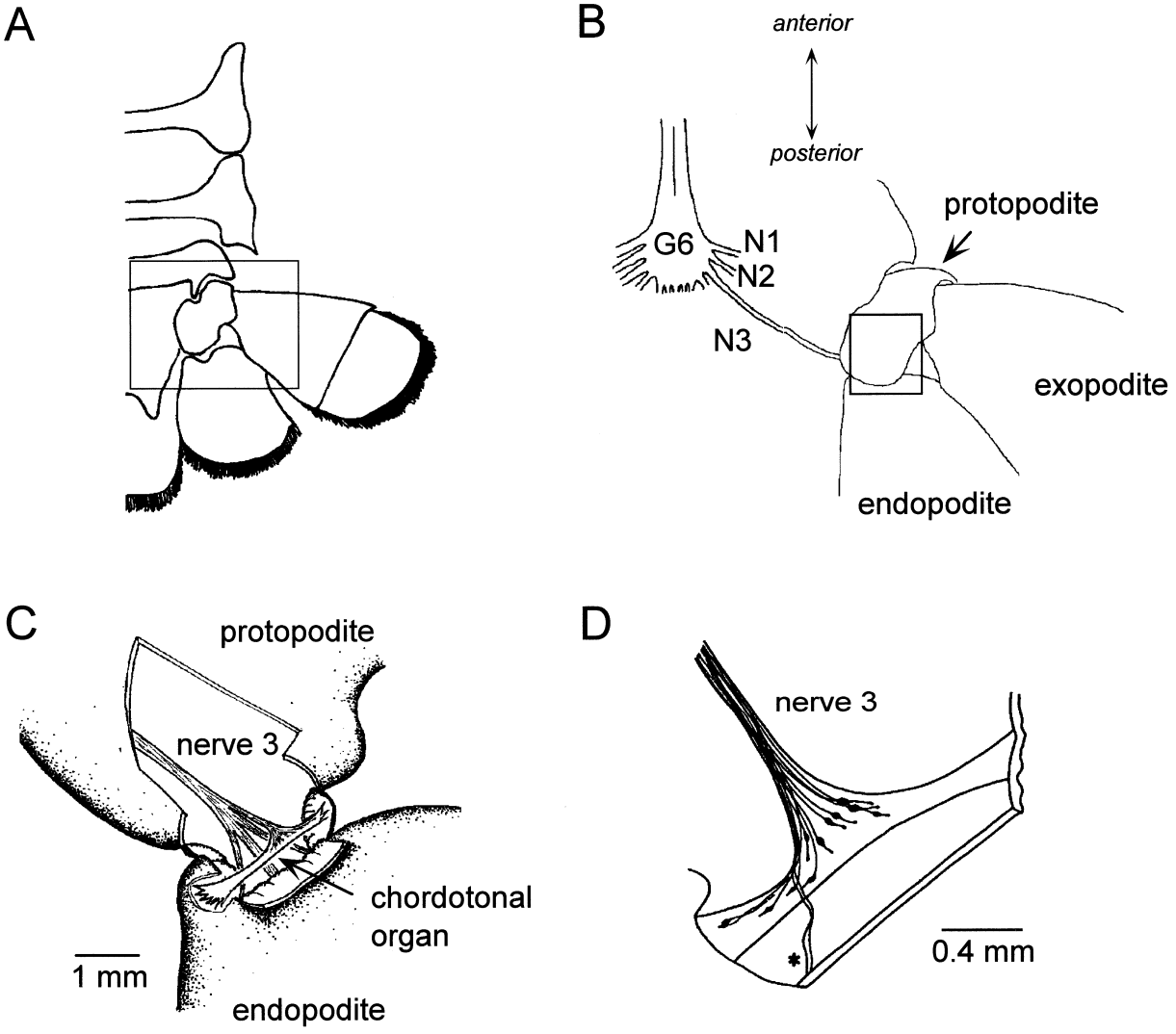

Fig. 1. The exopodite-endopodite chordotonal organ. **A**. Ventral view of the left half of the tailfan and last three abdominal segments. **B**. Enlargements showing the terminal (sixth) abdominal ganglion and the location of the chordotonal organ. **C**. The organ spanning the joint between the exopodite and endopodite, and its nerve entering nerve 3. **D**. Enlargement of a Methylene-Blue stained organ showing the 12 sensory cells and a pair of axons (asterisks) which project past the organ and into the endopodite hypodermis. Sixth abdominal ganglion, G6; nerves 1–3, N1–N3. Drawings are modified from Field *et al.* (1990) and Newland *et al.* (1996).

neurones terminate in the sixth abdominal ganglion, but a few have intersegmental projections (Nagayama and Newland, 1993). In common with many other sensory systems in invertebrates (for example, locust: Newland, 1991; crickets: Murphey, 1981), the projections of the chordotonal afferents form a map in the terminal ganglion. Projection position varies systematically across the map depending upon the velocity threshold of each afferent, so that those with low velocity thresholds project most anteriorly in the ganglion while those with higher velocity thresholds project more posteriorly. The closest analogy to this crayfish map comes from the tonotopic arrangement of auditory afferents of the bushcricket. Each of the auditory afferents is tuned to a particular best frequency and their somata are tonotopically organised within the auditory organ itself (Römer, 1983). The afferents project to different regions of the prothoracic ganglion in positions dependent upon the frequency to which they are tuned.

In addition, in the crayfish the CO afferents project to more dorsal areas of neuropil than do afferents that innervate water motion sensitive hairs, which project to the most ventral neuropil (Nagayama and Sato, 1993). A similar modality specific spatial segregation of the projection areas of exteroceptive and proprioceptive afferents has also been described in the locust (tactile afferents, Newland, 1991; chordotonal afferents, Pflüger *et al.*, 1988).

The sensory neurones make monosynaptic, chemical synaptic connections with specific spiking and nonspiking local interneurones and with intersegmental ascending interneurones in the terminal ganglion (Newland and Nagayama, 1993). Several lines of evidence suggest that these proprioceptive sensory neurones may be important in the escape pathway.

- First, they can encode the high velocity movements of the uropods that occur during swimming (Nagayama and Newland, 1993; Webb,1979).
- Second, they have conduction velocities several times faster than most tactile sensory neurones (Nagayama and Newland,1993; Nagayama and Sato, 1993).
- Third, they make monosynaptic connections with interneurone A (Newland and Nagayama, 1993) which is known to excite LG (Zucker,1972), and that input is sufficient to drive the interneurone to produce spikes (Newland and Nagayama, 1993).

Proprioceptive afferents make synaptic connections with the lateral giant interneurones

The LGs are activated by parallel mono- and disynaptic pathways from sensory neurones innervating exteroceptive hairs on the telson, uropods and posterior abdomen (Fig. 2A). Some of these sensory neurones make monosynaptic electrical synapses with LG that evoke a short latency EPSP in LG called the α-component (Fig. 2B), while others make monosynaptic, chemically mediated synapses with a specific set of intersegmental interneurones. These interneurones in turn make rectifying electrical synapses back onto LG (Zucker, 1972) that evoke a longer latency input in LG called the β-component (Fig. 2B). The inputs from the mono- and disynaptic inputs sum in LG and may give rise to spikes and initiate the tail-flip. The LGs activate the giant motor neurones (MoGs) in anterior abdominal segments (1st - 3rd) that innervate fast flexor muscles, whose activation leads to the rapid flexion of the abdomen (Wine and Krasne, 1972).

The LGs also receive synaptic input from the exo-endo CO. Displacements of the strand of the chordotonal organ evokes a sustained spiking in many of its sensory neurones in nerve 3, and depolarisations in LG (Fig. 3A). Superimpos-

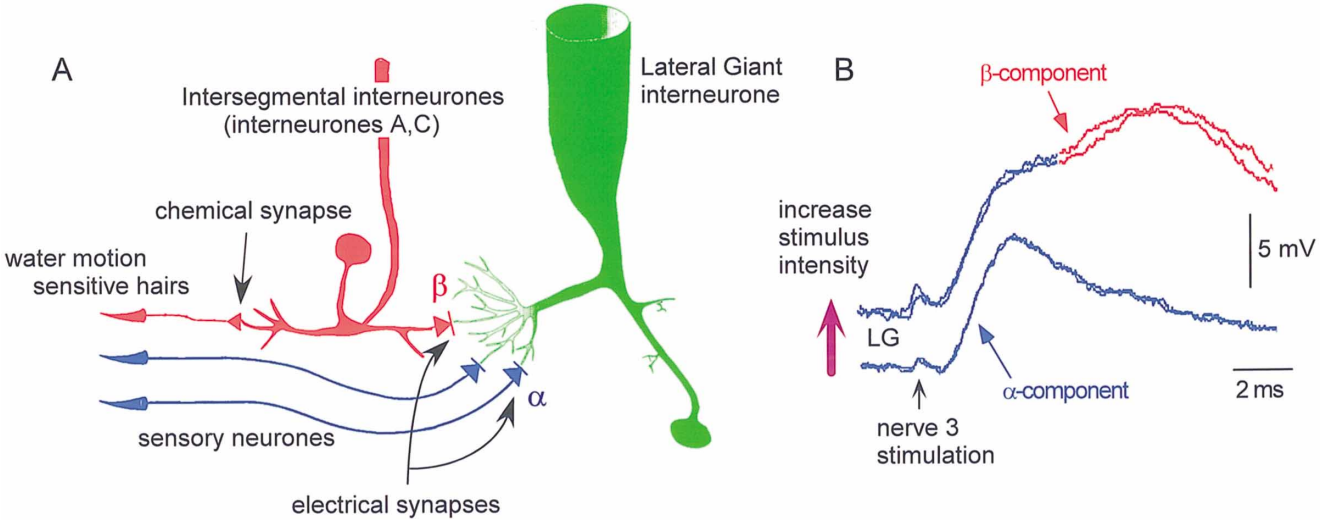

Fig. 2. LG is activated through both mono- and disynaptic pathways from exteroceptive hair afferents. **A**. The monosynaptic input from water motion sensory neurones gives rise to short latency EPSPs in LG, the α-component. **B**. In the disynaptic pathway particular intersegmental interneurones receive chemical inputs from the sensory neurones and, in turn, excite LG through rectifying electrical synapses. At higher stimulus intensities of nerve 3 this disynaptic pathway to LG is recruited and leads to a longer latency input , the β-component, that sums with the α-component. **A** is modified from Yeh. *et al.* (1997) and B is modified from Newland *et al.* (1997).

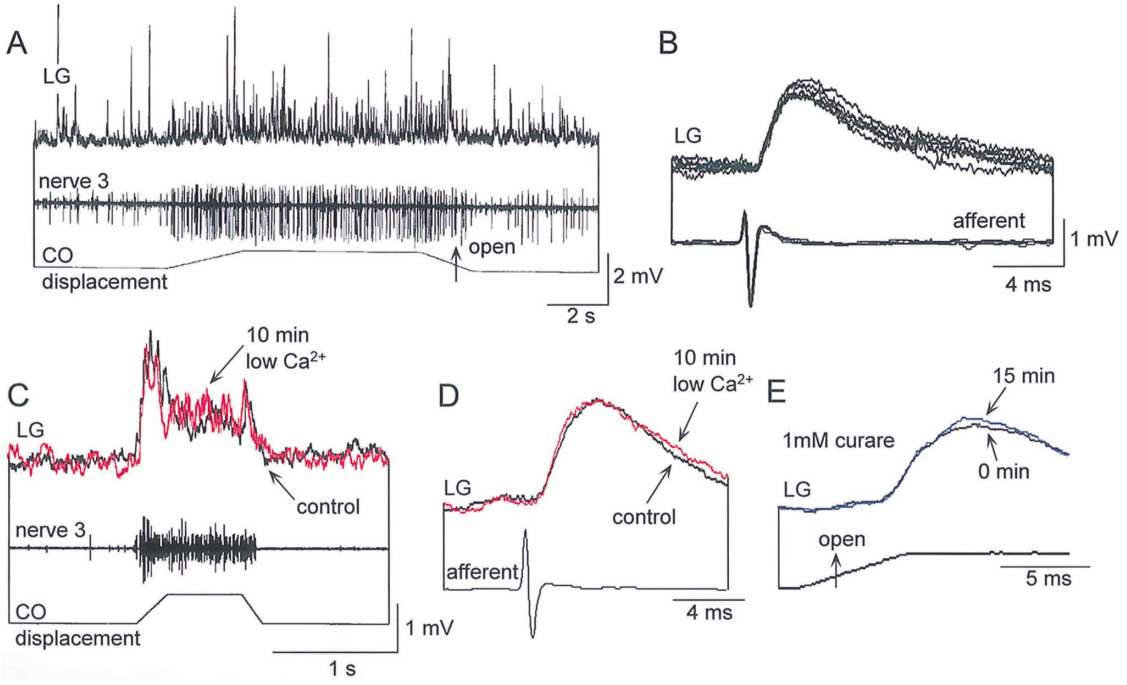

Fig. 3. Connections of chordotonal afferents with LG. **A**. Displacing the exo-endo CO strand evokes spikes in nerve 3 and excitatory potentials in LG. **B**. Superimposed sweeps triggered from sensory spikes show potentials occurring in LG with short and constant latency, indicative of a monosynaptic connection. **C**. and **D**. Changing the bathing saline to one containing a low calcium/high magnesium concentration has no significant effect on the responses to ramp stimuli (**C**), nor on individual EPSPs triggered from specific chordotonal afferent spikes (**D**), in comparison to control saline. **D**. Bath application of 1mM d-tubocurarine for 15 min has no effect on the compound potential in LG evoked with a ramp stimulus with a velocity of 1600°s^{-1}. Data are modified from Newland *et al.* (1997).

ing sweeps of the oscilloscope, triggered from a chordotonal organ sensory spike, shows that the potentials in LG follow with a short and constant latency (Fig. 3B). Not all 12 sensory neurones within the chordotonal organ, however, make synaptic connections with LG.

LG receives both tonic and phasic inputs from chordotonal afferents. The inputs are evoked in LG during maintained displacement of the chordotonal organ strand, although they are greatest in amplitude during the movement phase of the stimulus. Increasing the velocity of imposed movement from 4°s^{-1} to 1600°s^{-1} results in a summation of the inputs, giving a fourfold increase in the amplitude of the depolarisation. We never found these inputs to evoke spikes in LG.

Properties of the exo-endo CO-evoked potentials in LG

A number of observations suggest that the inputs to LG from proprioceptive afferents are mediated through electrical synapses.

- First, chordotonal organ stimulation evokes EPSPs with little central synaptic delay. In the example shown in Figure 3B a sensory neurone evokes postsynaptic potentials in LG with short latencies of only 1.1–1.2 ms. Since the chordotonal organ sensory neurones are known have rapid conduction velocities of 3.1±1.2 $m \cdot s^{-1}$ (Nagayama and Newland, 1993) a sensory spike would take approximately 1.1 ms to conduct the 3.4 mm distance between the extracellular and intracellular recording sites. This would, however, leave insufficient time for the synaptic delay of conventional chemical synaptic transmission.
- Secondly, bathing the nervous system with a saline containing a low calcium concentration of 2.7 mM (compared to 13.5 mM in normal saline) reduces the amplitude of the evoked potentials where chemical transmission is known to occur (Nagayama *et al.*, 1997). However, neither the amplitude nor dynamics of the response in LG are changed after bath application of low Ca^{2+}/high Mg^{2+} saline (Fig. 3C), nor is the amplitude of individual synaptic potentials triggered from chordotonal afferents affected (Fig. 3D).
- Thirdly, the depolarisations in LG evoked by ramp displacements of the chordotonal organ are unaffected by altering the membrane potential of LG.
- Finally, bath application of 1mM d-tubocurarine, a nicotinic antagonist, for 5 min also fails to change the amplitude of the chordotonal evoked potential in LG (Fig. 3E).

Taken together these results suggest that the LG receives both tonic and phasic proprioceptive inputs that are mediated through monosynaptic electrical synapses.

Proprioceptive inputs to interneurones in the disynaptic pathway exciting LG

In parallel with the monosynaptic electrical inputs from proprioceptive afferents onto LG is an excitation of LG through a disynaptic pathway involving interneurones A and C (Zucker, 1972), also known as interneurones NE-1 and RC-8, respectively (Nagayama *et al.*, 1994). These intersegmental

interneurones in turn excite LG via electrical synapses (Zucker, 1972) and the combined mono-and -disynaptic electrical inputs from exteroceptive afferents onto LG sum to evoke spikes and hence initiate the tail-flip escape response.

These same intersegmental interneurones also receive proprioceptive inputs from the exo-endo CO. Displacing the chordotonal organ strand evokes synaptic inputs in both interneurone A (Fig. 4A) and interneurone C (Fig. 4E) that sum to produce spikes in the interneurones at higher velocities of stimulation (interneurone A, Fig. 4A–C and interneurone C, Fig. 4E–G). The inputs to both interneurone A (Fig. 4D) and interneurone C (Fig. 4H) follow sensory spikes with short and constant latencies suggesting that they are mediated through monosynaptic pathways.

In interneurone A the EPSPs follow sensory spikes with two distinctly different, but constant, latencies (Fig. 5). The first, triggered from large amplitude extracellular sensory spikes, have short latencies of approximately 1.5 ms and are of large amplitude (1–2 mV) (Fig. 5A). The second, triggered from small amplitude extracellular spikes, have longer latencies of approximately 2.5 ms and are smaller in amplitude (approx. 0.5 mV) (Fig 5B). Taking into account the distances between recording and stimulating sites and the conduction velocities of the proprioceptive afferents, for the short latency potentials there would be little time for the synaptic delay of conventional chemical synaptic transmission. This suggests that either rapidly conducting afferents mediate transmission of these short latency EPSPs in interneurone A, or that transmission is via electrical synapses. On the other hand, central delays of 1–1.5 ms would account for the longer latency inputs onto interneurone A, implying that they could be mediated by chemical synaptic transmission from slowly conducting afferents.

To resolve this issue, we used three tests to obtain further evidence for electrical and chemical synaptic inputs onto interneurone A. First, continuous hyperpolarising current was injected into interneurone A while the chordotonal organ was stimulated mechanically. The amplitudes of the short latency EPSPs were unaltered by the accompanying changes in membrane potential (Fig. 5C). Such a lack of change is characteristic of many electrical synapses. By contrast, the amplitudes of the longer latency EPSPs were increased dramatically with hyperpolarising current, so that they are up to 50% greater with 2nA hyperpolarizing current (Fig. 5D). A change of this

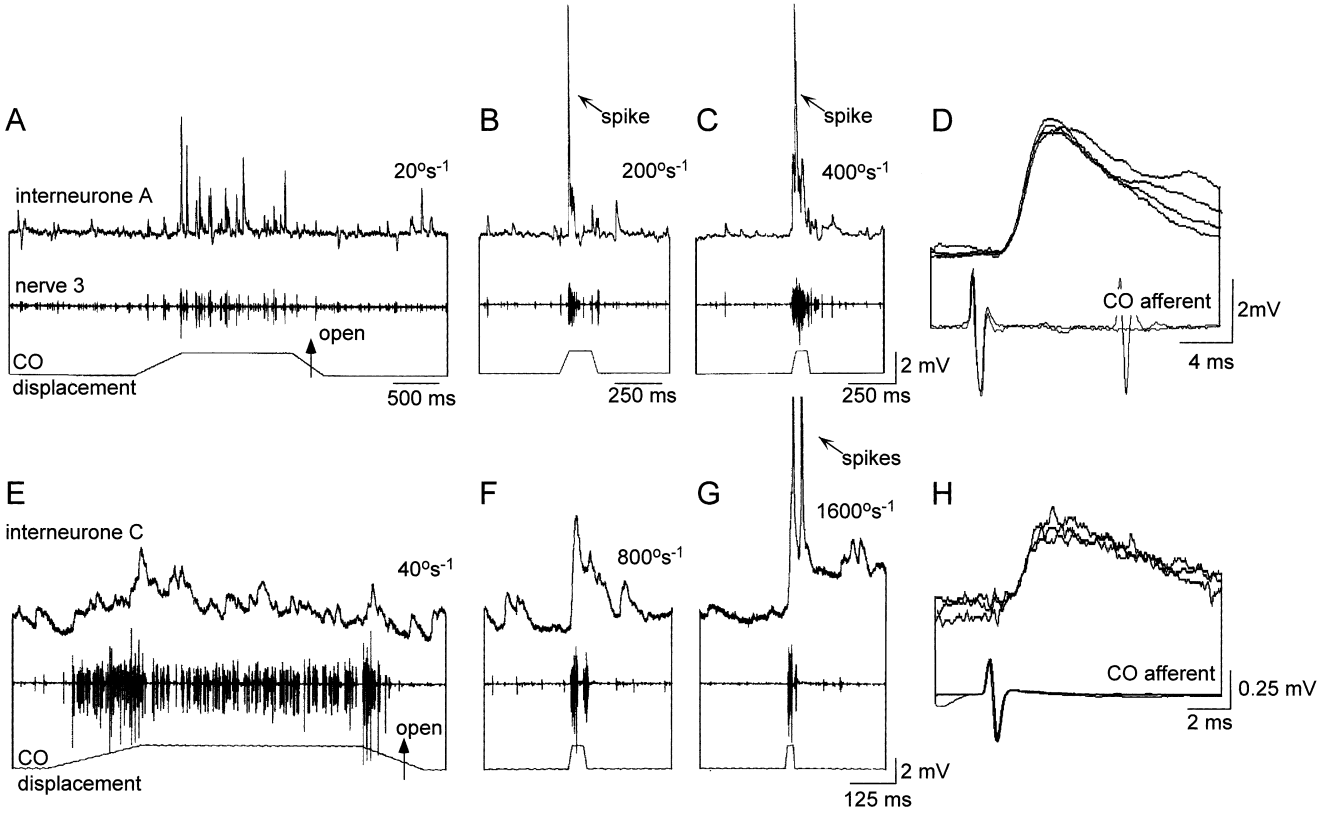

Fig. 4. Interneurones in the disynaptic pathway exciting LG. **A**. A ramp stimulus applied to the exo-endo CO evokes a burst of its sensory spikes in nerve 3 and EPSPs in interneurone A. Increasing the ramp velocity to 200°s^{-1} (**B**) and 400°s^{-1} (**C**) causes a summation of inputs to interneurone A that give rise to spikes. Superimposing the sweeps of an oscilloscope triggered from a chordotonal afferent spike in nerve 3 show potential in interneurone A with short and constant latency, indicative of a monosynaptic connection. **D**. A ramp stimulus to the exo-endo CO also leads to EPSPs and a depolarisation in interneurone C. These potentials sum at ramp velocities of 800°s^{-1} (**F**) and 1600°s^{-1} (**G**) to give rise to spikes in the interneurone. **H**. Superimposing the sweeps of an oscilloscope triggered from a chordotonal afferent spike in nerve 3 show potentials in interneurone C with short and constant latency, again indicative of a monosynaptic connection from chordotonal afferents. Data for A-D is modified from Aonuma *et al.* (1999) while that for E-H is unpublished.

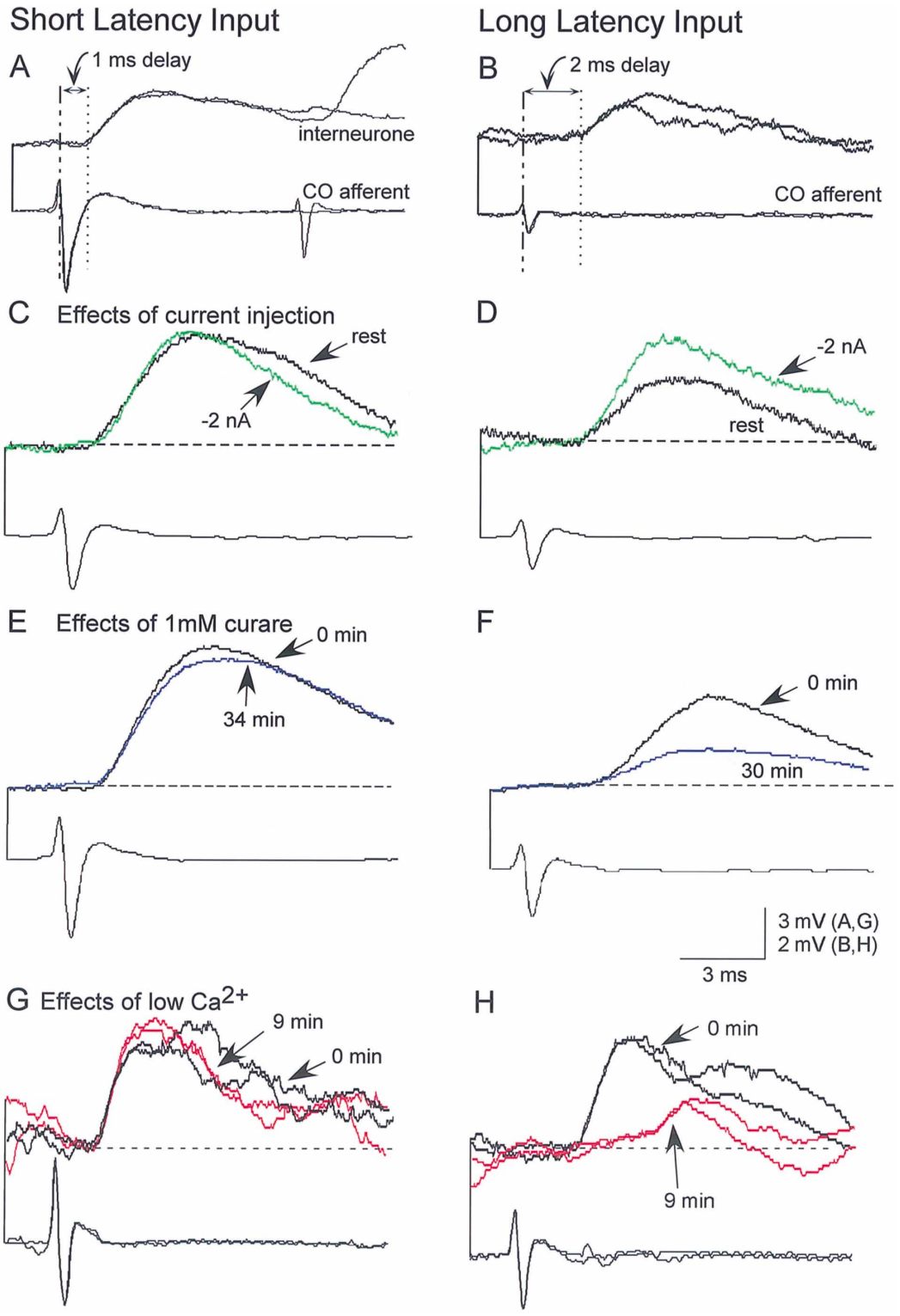

Fig. 5. Properties of the synaptic inputs to interneurone A. Particular chordotonal afferent spikes evoked by ramp displacement lead to both short (**A**) or long latency (**B**) EPSPs in interneurone A. The short latency inputs are unaffected by current injection of -2nA (**C**) while the longer latency EPSPs triggered by different afferents are increased in amplitude by almost 50% (**D**). **E**. Effects of 1mM d-tubocurarine, a nicotinic antagonist, on the EPSPs evoked in interneurone A. Curare has no effect on the short latency EPSPs in interneurone A, but the long latency inputs are reduced by over 50% (**F**). **G**. The short latency inputs are unchanged during bathing in a saline containing a low Ca^{2+} concentration, whereas the longer latency inputs are substantially decreased in amplitude after 9 min (**H**). Data modified from Nagayama *et al.* (1997).

nature is characteristic of chemical transmission.

Secondly, bath application of 1mM curare, a nicotinic antagonist, had little effect on the short latency EPSPs in interneurone A even after 34 min bath application (Fig. 5E), but significantly reduced the amplitude of the longer latency EPSPs, reversibly (Fig. 5F).

Thirdly, bathing the nervous system in a low Ca^{2+}/high Mg^{2+} saline again had no effect on the short latency EPSPs (Fig. 5G), characteristic of many electrical synapses (Zucker *et al.*, 1971), but almost abolishes entirely the longer latency EPSP after 9 min (Fig. 5H). A reduction in EPSP amplitude in a low concentration of Ca^{2+} is again a feature of chemical transmission.

The evidence suggests that the intersegmental interneurones in the disynaptic pathway exciting LG receive both monosynaptic electrical and chemical inputs in the form of short and long latency inputs from sensory neurones innervating the exo-endo CO. EPSPs mediated by electrical transmission at other crayfish synapses are known to have little or no central synaptic delay, are in many cases unchanged by depolarising or hyperpolarising current injection, bathing in low Ca^{2+}/high Mg^{2+} saline (Zucker *et al.*, 1971; Zucker, 1972). All of these features are characteristic of the short latency potentials from chordotonal afferents to interneurone A. Thus the EPSPs from chordotonal afferents onto interneurone A are likely to be mediated by electrical transmission. The longer latency potentials, however, are typical of chemical synaptic inputs, being significantly altered by current injection (Burrows and Pflüger, 1988; Nagayama and Sato, 1993; Zucker,1972) reduced in low Ca^{2+} saline (Parker and Newland, 1995) and reduced by bath application of curare, typical of cholinergic transmission (Miller *et al.*, 1992; Ushizawa *et al.*, 1996). All insect and crustacean mechanosensory neurones so far investigated use acetylcholine as their transmitter (Barker *et al.*, 1972; Casagrand and Ritzman,1992; Leitch and Pitman, 1995; Miller *et al.*, 1992; Parker and Newland, 1995; Trimmer and Weeks, 1989; Ushizawa *et al.*, 1996) and while it is likely that the chordotonal afferents also use acetylcholine we have not yet carried out a detailed study of the pharmacology of transmission.

A cautionary note should be introduced here since none of the characteristic features of either type of synapse, on there own, provide conclusive evidence for one form of synaptic transmission or the other. For example, Zucker *et al.* (1971) showed that for some chemical synapses in the crayfish bath application of low Ca^{2+} had little effect on the postsynaptic potential. Moreover, electrical synapses between primary afferents and LG are voltage sensitive (Edwards *et al.*, 1991), while those described here were unaffected by current injection, although this may be due to the fact that we were able to inject only small hyperpolarising currents into interneurone A. Taken together, however, all the features we describe for these synapses provide strong evidence for the convergence of chemical and electrical synaptic inputs from different proprioceptive afferents onto interneurone A.

Only recently has it been suggested that LG interneurones receive chemical as well as electrical synaptic inputs from water motion sensitive afferents (Miller *et al.* 1992; Yeh *et al.*, 1993). Likewise we now show that interneurone A, which itself excites LG through rectifying electrical synapses (Edwards *et al.*, 1991; Zucker, 1972), also receives both electrical and chemical inputs from a proprioceptor in the tailfan.

The fact that interneurone A receives electrical synaptic input from proprioceptive afferents challenges our view of the role of exteroceptive input onto this interneurone (Zucker, 1972; Zucker *et al.*, 1971). The synapses from the hair afferents have been regarded as being crucial in the exteroceptive input pathway to LG since they probably act as sites of habituation to repetitive stimulation (Krasne, 1969), and thus provide the neuronal pathways necessary for behavioural plasticity. Our finding that interneurone A also receives electrical inputs from chordotonal afferents raises a further question as to why both types of synaptic transmission are necessary at this synapse. Presumably this dual mode of transmission allows more plasticity than electrical transmission alone can provide but also with the additional benefit of speed of transmission in a neuronal circuit where rapid transmission is paramount to produce the short latency escape behaviour.

Presynaptic inhibition of exteroceptive afferents

Descending inhibition of exteroceptive afferents has been well documented (Kirk, 1985; Kirk and Wine, 1984) and is thought to prevent reafference causing further giant activation. In addition to a direct role in activating the escape pathway, we have found that the exo-endo CO also has a major role in regulating exteroceptive sensory input to the local circuits within the terminal ganglion. Exteroceptive hairs that are sensitive to water displacement and touch are distributed over the surface of the tailfan. The sensory neurones innervating these hairs receive a primary afferent depolarisation (PAD) from sensory neurones innervating the exo-endo CO (Fig. 6). This PAD only occurs during high velocity movements of the exopodite (Fig. 6A), similar to those that occur during escape swimming (Newland *et al.*, 1996). The effects that the proprioceptor mediates are widespread, so that afferents in four sensory nerve roots of the terminal abdominal ganglion, innervating hairs on the protopodite, exopodite, endopodite and telson, all receive PAD (Fig. 6B). The PAD is unlikely to be mediated through monosynaptic pathways since there is no anatomical overlap between the central projections of chordotonal afferents and many of the exteroceptive afferents. Like other sensory afferents that receive PAD in crayfish, the depolarisation is associated with a conductance increase and can be increased by the injection of hyperpolarising current or reversed by injection of depolarising current (Cattaert *et al.*, 1992; Fricke and Kennedy, 1983). Moreover, it is mimicked by γ-aminobutyric acid (GABA) and reduced by bath application of the chloride channel blocker picrotoxin (Fig. 6C), suggesting that it is a depolarising IPSP and, therefore likely to be mediated through chemical synapses. Further support for this comes from observations in the electron microscope that the exteroceptive afferents receive chemical input synapses

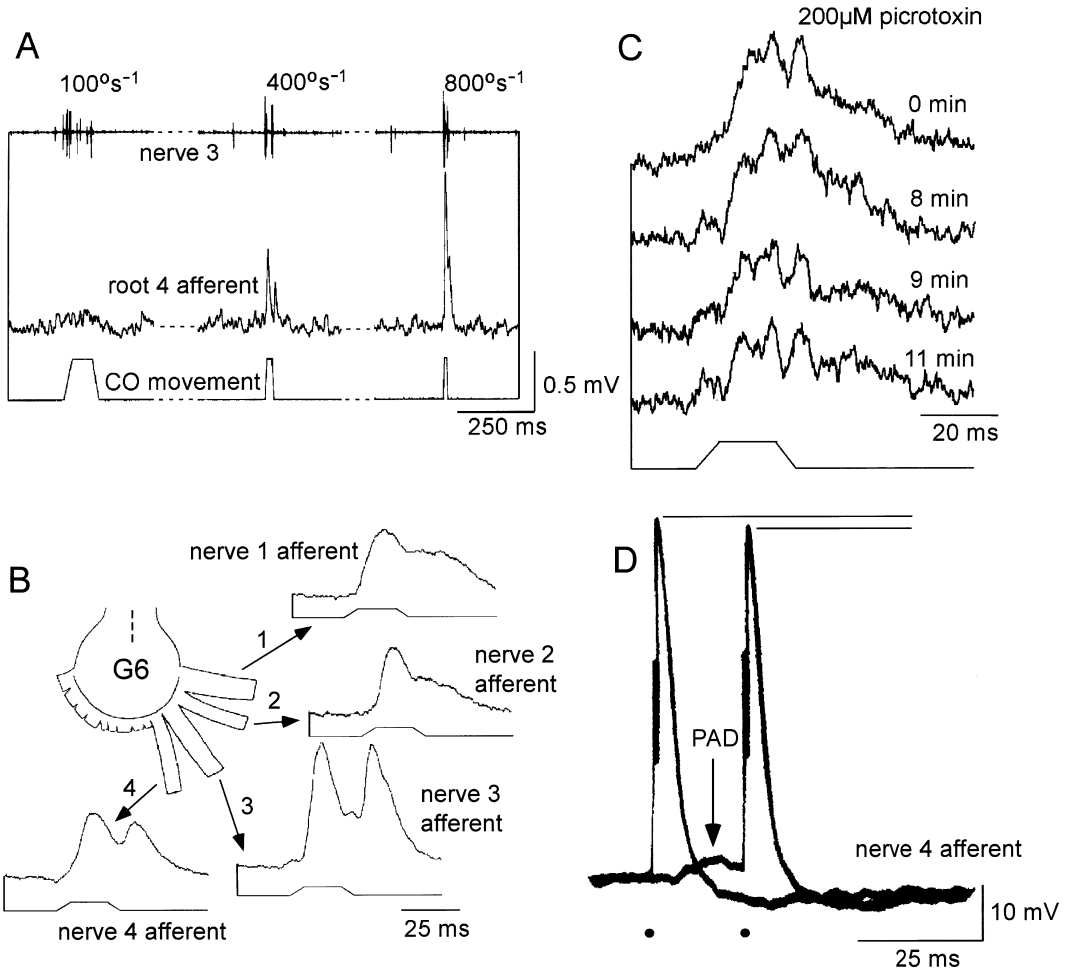

Fig. 6. Presynaptic inhibition of water-motion sensitive afferents. **A**. Intracellular recording from a hair afferent in root 4 during stimulation of the exo-endo CO strand at three different ramp velocities. The inputs to the sensory afferent are largest at higher displacement velocities. **B**. The effects that the exo-endo CO mediate are widespread. Signal averages triggered from the ramp displacement of the exo-endo CO strand show compound potentials in afferents recorded in nerve roots 1–4. **C**. The PAD in a root 3 afferent is reduced by bath application of the chloride channel blocker picrotoxin. **D**. PAD leads to a reduction in spike amplitude of water motion sensitive afferents. Electrical stimulation of a nerve 4 exteroceptive afferent evokes a spike (indicated by dots). When the spike is timed to coincide with PAD caused by CO stimulation, the spike is reduced in amplitude. Data modified from Newland *et al.* (1996).

(Newland *et al.*, 1996). The important feature of this presynaptic inhibition is that it reduces the amplitude of exteroceptive afferent spikes which is thus likely to reduce transmitter release and the efficacy of synaptic transmission (Fig. 6D).

In adult crayfish, the afferent synapses onto the giant interneurones depress very rapidly, leading to a behavioural habituation of swimming. Krasne and Teshiba (1995) suggested that descending inhibition from higher centres plays a role in preventing this depression. Thus to prevent it, the afferent-to-interneurone synapse is protected presynaptically during giant fibre activation (Krasne and Bryan, 1973). The inhibition provided by the exo-endo CO might explain how the same depression prone synapses might be protected during non-giant swimming, when the velocity of movement is similar to that during giant tail flips and, as such, will generate similar drag forces on the sensory hairs. These movements will produce a similar volley of afferent spikes as during a giant fibre tail flip, and this in turn will excite the same interneurones which have depression prone synapses with the afferents. Our observations suggest that the inhibition of the hair afferents during displacements of the exo-endo CO could modify that input during rapid movements, and also protect the afferent-to-interneurone synapses from depression.

Proprioceptive inputs to giant motor neurones

Our most recent work has been directed towards the output motor targets of the LGs, the giant motor neurones (MoGs). Whereas the MGs are known to form electrical outputs with the MoGs in each abdominal segment, the LGs only make output connections with these motor neurones in rostral segments (Mittenhall and Wine, 1973). This ensures that during MG tail-flips all segments flex, while during LG tail-flips only the rostral segments flex to generate the correct direction of thrust to pitch an animal forward. The MoGs receive recurrent feedforward inhibition from the giant fibres that can shunt subsequent input to the MoGs to prevent their multiple activation

and overuse of their depression prone neuromuscular synapses.

We have recently found that displacement of the exo-endo CO evokes depolarising potentials in the MoG in each abdominal segment at velocities over 200°s^{-1} (Fig. 7A), which sum sum at even higher velocities still, but never give rise to action potentials. The inputs only occur during opening movements of the CO (Fig. 7B), are not direct, occur only 20–40 ms after the proprioceptive stimulus is applied, i.e. at a time coinciding with flexion of the abdomen, and with latencies twice that of the LGs and interneurones A and C which receive monosynaptic input from chordotonal afferents. We know from Wine (1977) that the MoGs are inhibited following giant fibre activation and Fraser and Heitler (1993) identified the motor giant inhibitors (MoG-Is) anatomically and physiologically and showed that their principle mode of activation was via electrical synaptic transmission through the segmental giant interneurones (SG) from the giant interneurones. In addition, the same authors suggested that electrical stimulation of nerve 3 also evokes dIPSPs in MoG-I1 that are the result of the interneurones themselves having branches in nerve 3 and being activated by the extracellular nerve stimulus. In the terminal ganglion MoG-I1 has no branches near any nerve (Kirk, 1985) and cannot directly be activated by nerve stimulation to produce inhibitory inputs onto MoG. It is tempting to suggest that the inhibition in MoG in the terminal ganglion may result from inputs from the exo-endo CO afferents in nerve 3 which could also protect the MoG neuromuscular synapses during longer, non-giant swimming sequences. Clearly further work is needed by us to analyse the properties of these CO evoked potentials in MoG to determine whether they are excitatory or inhibitory and how they contribute to the giant or non-giant mediated tail-flips.

Conclusion and Perspectives

We have found that proprioceptive signals from the exo-endo CO appear to have considerable effects on the neuronal circuits mediating escape in the crayfish. They excite

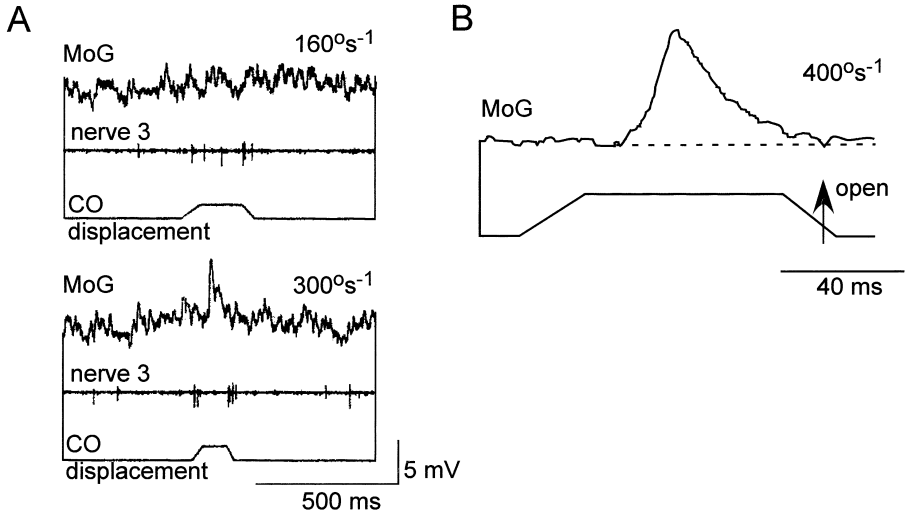

Fig. 7. Proprioceptive inputs to giant motor neurones. **A**. Exo-endo CO evoked inputs in MoG occur only at velocities of CO movement greater that 200°s^{-1}. **B**. The inputs to MoG occur with a delay of 20–40 ms after the onset of the stimulus and only occur during stretches of the CO strand equivalent to opening movements of the exopodite relative to the endopodite. Unpublished data.

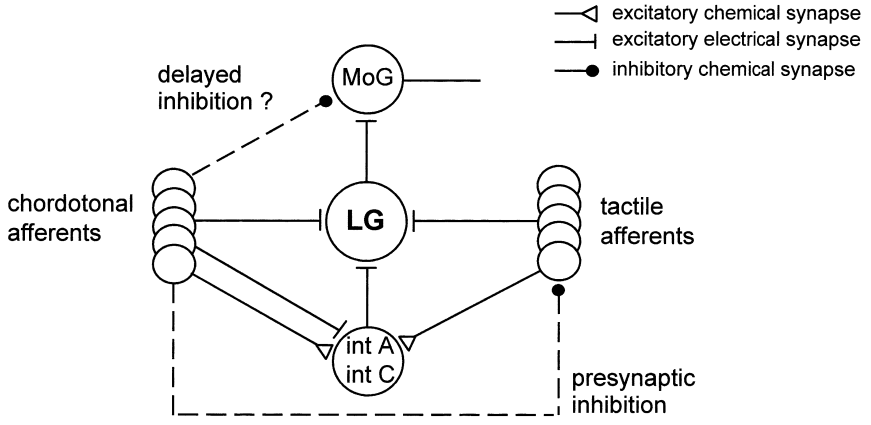

Fig. 8. Summary diagram indicating the known effects of exo-endo CO on the LG escape circuit in the terminal abdominal ganglion. Effects made through indirect pathways are indicated by dashed lines.

the giant fibres directly and indirectly through other identified intersegmental interneurones. Moreover they inhibit unwanted sensory inputs that could lead to conflicting behaviour during the tail-flip, and finally they may inhibit the giant motor neurones to protect their output synapses (Fig. 8). Although we are only now beginning to understand a little about proprioception in the tailfan we still have much to learn. We still do not know the identity of the neurones mediating PAD or the role of all the proprioceptors in the tailfan. For example there are at least 2 other known proprioceptors (Field *et al*., 1990) whose coding properties, patterns of connectivity and role in behaviour have yet to be described. Moreover, while we have reviewed here only the role of proprioception during escape we also know that the same proprioceptor, the exo-endo CO, is involved in postural reflexes of the tailfan (Newland and Nagayama, 1993) and its signals are processed in local circuits in the terminal abdominal ganglion (Nagayama *et al*., 1994). How these apparently conflicting or differing roles are resolved must remain one of our overriding priorities for future research. It is clear that we need to address how different behaviours are chosen and how they interact to prevent conflicting central motor patterns.

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