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RESPONSES AND CENTRAL INTERACTIONS OF TENSION RECEPTORS IN THE LEG FLEXOR MUSCLE OF *CARCINUS**

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Abstract—1. The physiology of the detection of tension in the limb flexor muscle of *Carcinus* is described. The tension receptors respond primarily to the rate of tension increase in the muscle, and their activity is closely related to movement of the apodeme, whether induced by external forces or by flexor muscle contraction.

2. The immediate effect of tension afference in whole animal preparations in all cases is short-term excitation of flexor motoneurones.

3. If there is spontaneous activity in the motoneurones, or if activity is induced by stimulation of non-limb areas, tension afference also evokes long-term excitation.

4. Resistance reflexes are always inhibited by tension afference.

INTRODUCTION

Receptors that monitor muscle tension have been found in a number of arthropods (Macmillan & Dando, 1972; Eagles & Hartman, 1975; Eagles, 1978; Theophilidis & Burns, 1979). The morphology and ultrastructure of the tension receptors of the flexor muscles in the legs of Brachyuran crustaceans have been recently described (Parsons, 1980) as has the excitatory innervation, some histochemistry, and the neuromuscular physiology of the muscle (Parsons, 1982; Parsons & Mosse, 1982). Interactions between tension receptor activity and motor output in the crab have been reviewed (Macmillan, 1976) with the conclusion that the receptors can function to damp the passive resistance reflex movements by inhibition of the motor activity to the homonymous muscle and excitation of motor activity in the antagonistic muscle. On the other hand, excitation of the homonymous muscle motoneurones can occur when the animal is active. Clarac & Dando (1973) reported that high (unspecified) levels of tension nerve afference could excite both the homonymous and antagonistic motoneurones, but it is not clear in which situations this result was found. Just how the tension afference modulates motor activity at the cellular level is yet to be studied.

In the flexor muscle of *Carcinus*, the tension receptors are distributed along one face of the apodeme, and consist of two anatomically distinct populations of receptor cells: a small number of Proximal Sensory Cells (PSC's) possessing approximately 70 μm cell bodies, and numerous smaller (<35 μm) Distal Sensory Cells (DSC's) that are found mainly in the distal third of the apodeme. The sensory processes (dendrites) of these cells are embedded in the apodeme cuticle (Parsons, 1980). The close association of the receptor sensory processes with the cuticle suggests that tension receptor output is closely related to the forces that act on the apodeme during muscular contractions.

In this paper the responses of the apodeme tension receptors to muscular contractions are investigated in detail to provide a basis for subsequent examination of the tension receptor system. The effects of tension nerve afference on the flexor motoneurones in whole animal preparations are also examined.

MATERIALS AND METHODS

Walking legs of the crab *Carcinus maenas* were obtained and prepared for whole animal and isolated leg preparations as previously described (Parsons, 1982). Tension was measured with a micro manipulator—mounted DMC Dual Mount Deflection Sensor (Kistler-Morse Corporation, Bellevue, WA.). Passive movements of the carpus were applied through the transducer by an ADAK PMP Leg Moving System (ADAK Electronics, Eugene, ORE) driven by a Tektronix signal generator. Transducer compliance was approximately 10 μm/g. Transducer output was amplified when necessary, and in these cases the signal was passed through a low-pass active filter which introduced negligible distortion to the tension-level signal.

Activity in the Flexor Apodeme Sensory Nerve (FASN) was recorded *en passant* with monopolar hook electrodes, or from the cut end of the nerve using polyethylene-tipped suction electrodes. The FASN or the Flexor Motor Nerves (FMN) were stimulated electrically with the same types of electrodes. In most cases all information was stored on analogue FM magnetic tape (Tandberg Series 115D), for subsequent filming and analysis.

Movements of the anterior edge of the flexor apodeme during muscle contractions were filmed with a Bolex H16 reflex movie camera at 48 frames/sec through a Wild M8 dissecting microscope. The edge of the apodeme was marked in several places by single particles of carborundum, to allow quantification of the distances moved by the apodeme during contractions. Small light-emitting-diodes (LED's) with adjustable flash rates were placed in the edge of the film field to allow film frames to be matched with

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physiological data stored on tape. The film was projected by an LW Photo-Optical analyser (Model 223A, Mk IV) onto the measuring tablet of a MOP-3 Image Analyser (Carl Zeiss Inc.) which was used to analyse the apodeme movements.

All preparations were post-stained with methylene blue to check that the electrode placement was correct.

RESULTS

Tension receptor responses

The response of apodeme tension receptors to an isometric contraction of the flexor muscle typically involves the firing of a number of tension units of different amplitudes in phasic, phaso-tonic, and in a few cases tonic patterns. The activity of these phasic and phaso-tonic units is concentrated about the time of tension rise. Increasing the duration of the train of stimuli to the FMN increases both the tension level and the tension receptor activity recorded in the FASN (Fig. 1). With an increase in stimulus pulse frequency, the rate of tension development is usually more rapid, and this elicits greater FASN activity: increased numbers of units responding, and those already responding generally discharge at higher frequencies. Similarly if the stimulus-pulse voltage or duration is increased sufficiently to recruit further motor axons, the tension and FASN activity also increase (Fig. 2). The level of resting tension in the flexor muscle also affects the response of the tension receptors. A given contraction of the flexor muscle elicits a higher level of tension receptor activity when an increased level of resting tension is present in the muscle (Fig. 3). Very slow or small contractions, in general, do not elicit observable tension receptor activity. In most cases isotonic contractions do not produce observable tension receptor activity unless the carpus encounters resistance to its movement.

The anatomical and ultrastructural arrangements of the receptors (Parsons, 1980) suggested that apodeme flexion or distortion is the necessary receptor stimulus. To test this hypothesis the movement of the anterior apodeme edge was followed during isometric contractions of the flexor by filming the movements of

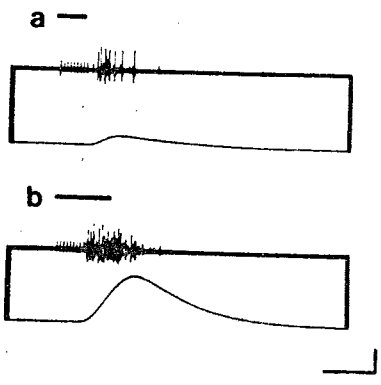

Fig. 1. Response of flexor apodeme tension receptors to isometric contraction of the flexor muscle. The receptor activity occurs mainly during the rise in tension, and longer contractions elicit larger tension and increased tension receptor activity. Top trace: FASN; lower trace: tension. (a) 100 ms train stimulation of the FMN at 80 Hz. (b) 200 ms train stimulation of the FMN at 80 Hz. Upper bars show duration of stimulus. Scale bars: 200 msec; 3 g.

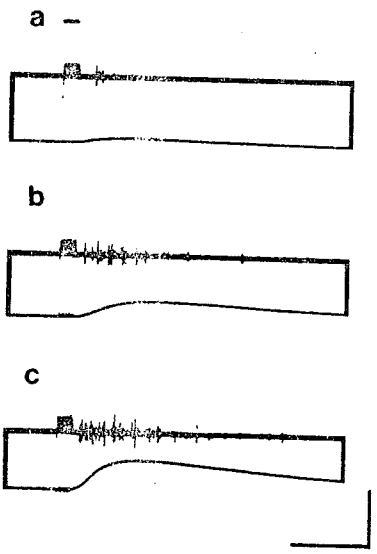

Fig. 2. Effect of increased isometric contraction strengths on activity in the FASN in one preparation. The number of motor axons stimulated was checked with a distally placed electrode. Stimulation (marked by upper bar) at 95 Hz, 0.1 msec pulse length, for 100 msec. Voltage of stimulus pulses increased to recruit motor axons. Top trace: FASN; lower trace: tension. (a) 2 motor units; slight FASN activity. (b) 3 motor units; with the faster contraction and higher tension produced here, there is an increase in the number of tension receptor units responding and in the duration of the activity. (c) 4 motor units; a further increase in activity in the FASN is evident. Scale bars: 0.5 sec; 5 g.

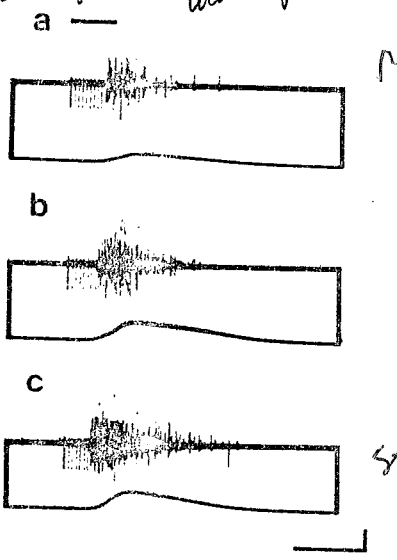

Fig. 3. Effect of increased muscle length (by increasing M-C joint angle) on tension receptor response. 300 msec train stimulation of the FMN at 80 Hz, 0.5 msec, 0.5 V. Note increase in tension receptor activity with increase in joint angle. (a) 70°, most muscle fibers flacid. (b) 110°, all muscle fibers taut. (c) 140°. Note that the resting tension level here is approximately 2.9 g heavier than in (b). Upper bars show duration of stimulus. Scale bars: 0.5 sec; 5 g.

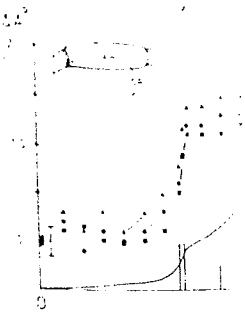

Fig. 4. Graph showing to tension developmen sion receptor units. Iso following FMN stimul 0.6 V. Note the associ unit with the initial ra change in apodeme po unit shows tonic phas increase in tension. A Inset shows how AA level. ▲ = most distal at M-C joint. ● ■ = 17.7 mm from ret (from the fixed poin tion = 35.5 mm. Mea trace

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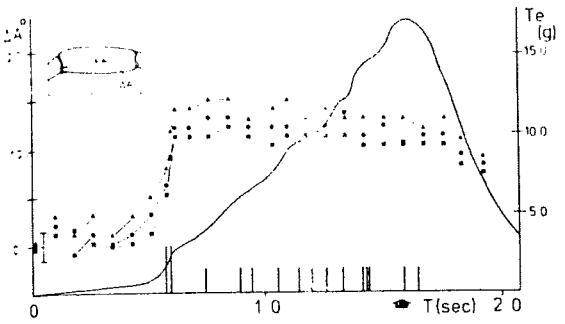


Fig. 4. Graph showing relationship of apodeme movement to tension development and to activity in two of the tension receptor units. Isometric contraction of flexor muscle following FMN stimulation for 1.6 sec at 50 Hz, 0.05 msec, 0.6 V. Note the association of the larger tension receptor unit with the initial rapid tension increase and the rapid change in apodeme position. The second tension receptor unit shows tonic-phasic activity for the duration of the increase in tension. Arrow (↑) denotes end of stimulation. Inset shows how ΔA is measured. Te = isometric tension level. ▲ = most distal point, 11.7 mm from reference point at M-C joint. ● = 14.6 mm from reference point. ■ = 17.7 mm from reference point. Length of flexor muscle (from the fixed point on M-C joint) to I-M articulation = 35.5 mm. Measurement error bar at start of ΔA trace applies to all points.

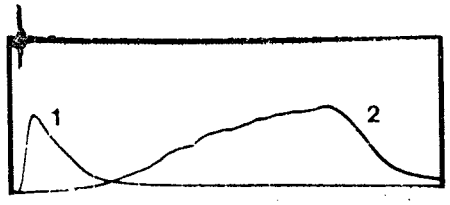


Fig. 5. Effect of the rate of isometric tension increase on tension receptor activity. Top trace: FASN activity, trial 1 and trial 2 (no FASN activity) superimposed. Lower trace: isometric tension, trace 1 = fast tension rise, FMN stimulation 100 msec, 85 Hz; trace 2 = slow tension rise, same stimulation but at 25 Hz for 3.6 sec (stimulus artefact visible on top trace; end of stimulation 2 at arrow). With fast tension development (1), a short burst of activity in a number of FASN units was seen. Slow tension development (2), although attaining the same final tension, elicited no FASN activity. Scale bars: 0.5 sec; 20 g.

single particles of carborundum placed on accessible parts of the anterior edge of the apodeme. This edge is the only portion of the apodeme visible or accessible in the intact flexor muscle system. The angles moved by the particles with respect to a fixed point at the merus-carpus articulation were measured (see inset, Fig. 4). The changes in angle were plotted on the same time scale as both the tension changes and the FASN activity data. In the example shown here (Fig. 4), the initial rapid tension rise was associated with rapid apodeme movement and with the phasic activity of one unit, whilst another unit responded phasotonically during the remainder of the contraction when there was little apodeme movement.

To examine how externally imposed forces applied to the apodeme affect receptor activity, the carpus was removed and the tension transducer attached directly to the distal end of the flexor apodeme with forceps. This arrangement was used to study tension receptor responses when forces were applied either along the axis of the apodeme or at right angles (laterally) to it. Axially applied stretching forces elicited FASN activity only at levels considerably greater than those required by laterally applied forces. For example, in one preparation it was found that an axially applied force had to be forty times the level of a laterally applied force to achieve a comparable response in the FASN. Furthermore, when all muscle origins were severed the FASN responses to axial or lateral forces were similar to those found when the muscles were intact. In this case, the proximal end of the apodeme was held in its "at-rest" position with mounted forceps.

As might be expected from examination of the tension receptor responses in other arthropods, the rate of tension increase is a major factor in the production of FASN output. With a faster rate of tension increase, FASN activity also increases. Although the same absolute tension may be reached, slow rates of tension increase produce little or no activity in the FASN (Fig. 5).

The responses of some of the PSC's to flexor muscle contraction was studied by recording from the FASN after cutting it just distal to the entry point of the PSC axon and then severing any other axons entering the FASN more proximally (Fig. 6). PSC units were active usually only during the tension rise. Fast contractions elicited phasic or phaso-tonic PSC activity, but during slower tension development, in which DSC units would normally be responding, the

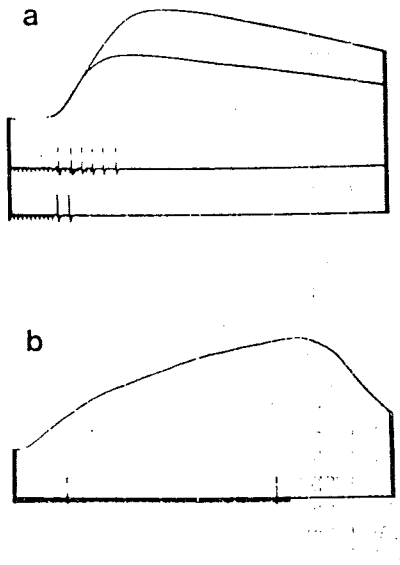


Fig. 6. Responses of the most proximal PSC to isometric flexor muscle contraction. Top traces: tension; lower traces: PSC activity. (a) Effect of stimulation for 100 msec (lower tension trace and nerve activity trace) and 200 ms respectively (90 Hz, 0.05 msec, 6 V). (b) Longer stimulation (3.7 sec) at reduced stimulus strength (40 Hz, 0.05 msec, 6 V). Note poor PSC activity here. Although large tensions are associated with PSC activity in this preparation, this was not always the case. Scale bars: (a) 0.1 sec; 30 g; (b) 0.5 sec; 30 g.

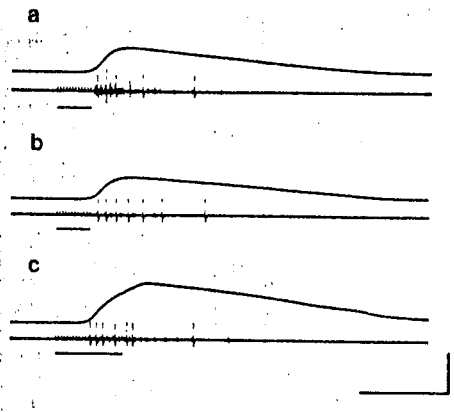


Fig. 7. Responses of DSC's to isometric flexor contraction. Top trace: tension; lower trace: DSC activity recorded in the FASN. (a) Response in intact FASN to FMN stimulation (150 msec, 70 Hz, 0.07 msec, 5.5 V). (b) Same stimulation after crushing the FASN until only two units remained. (c) Same stimulation but for 300 msec. Average discharge frequency and duration of the large unit activity is similar to (b) but the frequency and duration of the activity of the smaller unit has increased. Lower bars show duration of stimulus. Scale bars: 400 msec; 5 g.

PSC's were usually silent. In most cases, PSC responses fatigued more rapidly after repeated stimulations than did the DSC responses.

Since a large number of DSC units responded simultaneously, it was not possible to identify the response characteristics of single units when recording from the entire FASN because of spike superposition. Single unit responses were therefore studied by progressively crushing the FASN distal to the recording electrode until the response to tension involved only a few units that could be unambiguously identified. The responses of some of these units are shown in Fig. 7. Here, the activity of the two units falls off slowly, and, so extends their activity past the peak of tension. With a longer duration motor nerve stimulation, the tension elicited is greater and the smaller unit is active for a longer period. The phasic nature of the larger unit is revealed when a number of trials are superimposed (Fig. 8). This unit responds predominantly to the rise in tension, and the smaller unit, in addition to responding to tension rise, shows slowly adapting activity that extends into the decreasing tension phase of the response. Other units were found that responded tonically during the entire period of elevated tension. Most units ceased firing close to the end of the tension rise. Some units responded with a tonic rate of discharge that showed little change in frequency once above a threshold, and were also independent of the rate of tension increase.

Because not all motor units are involved in all contractions (Parsons, 1982), the direction of apodeme movement or bending depends on which specific motor units, and therefore muscle fibers, are used in the contractions. In most cases the apodeme bends dorsally ("axially"; Parsons, 1980) probably due to the greater number and consequently the larger volume of muscle fibers present on the dorsal side of the apodeme.

The relationship between the contraction of different motor units and tension receptor output was investigated by stimulating single flexor motor axons in isolated leg preparations (see Parsons, 1982 for methods). The possibility of a relationship between the activity of specific tension receptor units and the contraction of individual motor units was thoroughly investigated but no relationship was found. It is clear that contractions involving the faster motor units result in greater FASN activity than do contractions caused by the slow motor units. Such a relationship is predicted by the previous results. However, activation or modulation of particular FASN units did not appear to be related to stimulation of specific excitatory axons.

To investigate the relationship of muscle fiber contractions to tension and to tension-receptor activity, individual muscle fibers throughout the flexor were intracellularly depolarised, and the tension and FASN activity that ensued recorded. The greatest tensions produced by single flexor muscle fibers are produced by those fibers that have their insertions in approximately the central third of the apodeme. In most cases the tensions produced were very small, and only rarely did a contraction in one of these fibers elicit any visible FASN activity.

Effects of tension receptor afference on flexor motor activity

Whole animal preparations were used to assess the effects of FASN activity on the ongoing motor output to the flexor muscle. To gain access to the flexor muscle and nerves, the extensor muscle was removed. Single pulse stimulations of the FASN elicited reflex motor activity in the FMN. The extent of the reflex FMN activity depended on the frequency, strength and duration of the stimulus pulses. An indication of the number of FASN units activated by a given stimulus level was generally obtained from the record of FMN activity because the latter was recorded at the proximal end of the nerus where the FASN and FMN run together in one bundle. At high stimulus levels however, much of the FASN activity was obscured by the stimulus artefact. Some FASN activity was present at stimulus levels below that necessary to elicit reflex FMN activity.

Increasing the amplitude of single pulse stimuli to the FASN increased the number and frequency of discharge of the motoneurones responding (Fig. 9). As the stimulus intensity increased, the latency of the reflex FMN activity decreased. When trains of repeti-

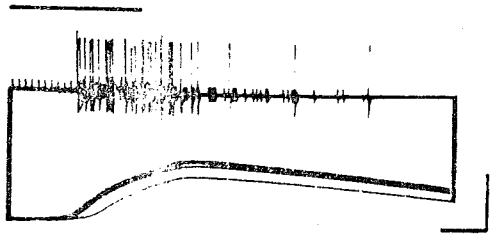
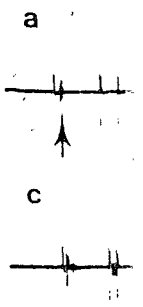


Fig. 8. Superimposed traces of 5 stimulations at 300 msec (upper bar) of preparation in Fig. 7(c). Top trace: activity in the two DSC's; lower trace: tensions. Note the clustering of large unit DSC activity during the tension development phase of the response. The small unit responds more tonically and extends its activity into the relaxation phase of the response. Scale bars: 100 msec; 5 g.



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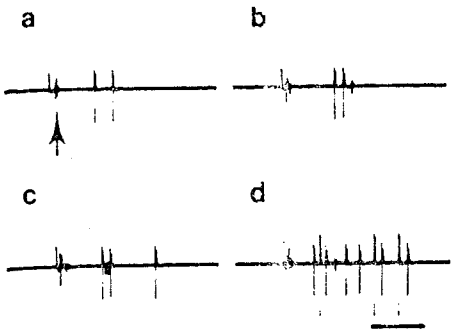

Fig. 9. Effect of single pulse extracellular stimulations of the FASN with increasing stimulus strength (voltage) in a whole animal preparation, recorded in a more proximal electrode that records both FASN and FMN activity. Background activity was a single tonic unit (same unit as in (a) below, discharging at ~5 Hz). Pulse duration = 0.5 msec. (a) 4 V. Arrow shows the number of FASN units stimulated, and these are recorded soon after the stimulus artefact in the recording electrode. Approximately 36 ms later the response of the flexor motoneurone is seen. In this case, two spikes from the tonic motor unit result (this is the tonic unit that is spontaneously active in the FMN in whole animal preparations). (b) 8 V. Further FASN units are stimulated, and the response in the FMN includes a single spike from a small unit (unidentified), and the frequency of the tonic unit is slightly increased. (c) 10 V. The increased FASN afference elicits a slight increase in the activity of the motor units. (d) 16 V, another motor unit now responds to the increased FASN stimulation, and this is the 1st phasic axon recruited in most flexor muscle contractions. The activity of the tonic axon has also increased. In all cases these responses were repeatable at a particular stimulus level. Scale bar: 50 msec.

tive, low frequency stimuli are applied to the FASN in a resting aninal, only the reflex FMN activity associated with each stimulus pulse is observed. At higher frequencies, FASN stimulation can induce massive FMN discharge, which is usually followed by a period of silence in the FMN. In addition, the tonic unit is usually inhibited during the discharge (Fig. 10). Similarly, in some animals, very high amplitude, low frequency FASN stimulations (e.g. 1 Hz) can induce the same type of extended FMN discharge and delayed inhibition.

Resistance reflexes

Resistance reflexes to passive carpus extension, that is, FMN activity that tends to oppose exogenous extension movements (Bush, 1965), can be altered by FASN stimulation. Single pulse stimulations of the FASN elicit brief reflex FMN activity, but do not inhibit the resistance reflexes. Repetitive stimulation at the same amplitude increased the FASN afference. Above a threshold this increased input caused inhibition of the resistance reflexes. If a given train of FASN stimuli did not produce inhibition of the resistance reflexes, then an increase in either the pulse frequency, or the stimulus pulse voltage or the duration eventually led to inhibition (Fig. 11). Similarly, if a given stimulus train to the FASN did not inhibit resistance reflex activity, an increase in train duration eventually caused inhibition. In general, once the threshold for inhibition was reached, further increases in pulse-amplitude, frequency, or duration induced longer periods of inhibition than lower level stimulations (e.g. Fig. 11c,d).

Centrally generated flexor motor activity

The effect of FASN activity on centrally driven (i.e. non limb-reflex) motor activity was, in general, excitatory to the flexor motoneurones. By itself, FASN stimulation elicited FMN activity as shown in Fig. 10. Abdominal flap stimulation alone usually induced a strong FMN discharge and flexor muscle contraction (Fig. 12) along with general struggling movements in other limbs. When these two methods of FMN stimulation were applied simultaneously, the resultant flexor motor activity was greater than would be present by simple summation of the individual responses. This increased FMN activity was sometimes followed by a period of inhibition of FMN activity.

DISCUSSION

The results show that the response of tension receptors in the flexor apodeme is related to the amount of apodeme movement or distortion produced by forces acting on the apodeme. The applied forces may be internal due to muscular contraction or external due to passive carpus movement. Through the coupling of muscle, apodeme cuticle, and receptor dendrite, the

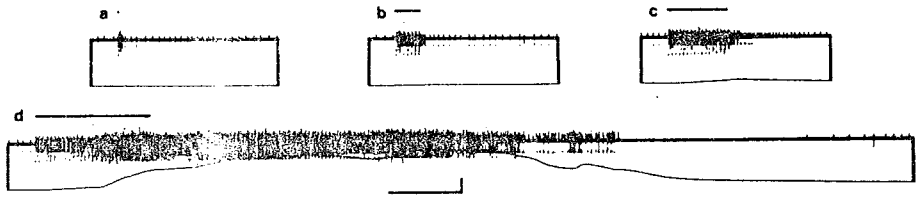

Fig. 10. Responses of flexor motoneurones to FASN stimulation. Different preparation to that in Fig. 9. Top trace records activity in the FMN and FASN, but FASN activity cannot be seen at this gain. Stimulation at 30 V, 0.5 msec. (a) Single pulse stimulation causes slight change in subsequent tonic unit activity. Level of reflex FMN activity is similar to Fig. 9(d). (b) 300 msec train stimulation at 20 Hz. Slight tension induced by the reflex activity and again the post-stimulus tonic motor unit frequency increases slightly. (c) 800 msec train, 20 Hz. Stronger tension is developed and a small discharge of a phasic unit occurs just after the end of stimulation. Tonic unit frequency increased substantially, but like all previous stimulations, returned eventually to the background level. (d) 1600 msec train, 20 Hz. This stimulation of the FASN induces massive FMN discharge over many seconds. Activity in the tonic unit is apparently inhibited during and after the phasic unit burst activity. After a few seconds the tonic unit background activity begins again. Bars indicate duration of stimulus. Scale bar: 1 sec; 10 g.

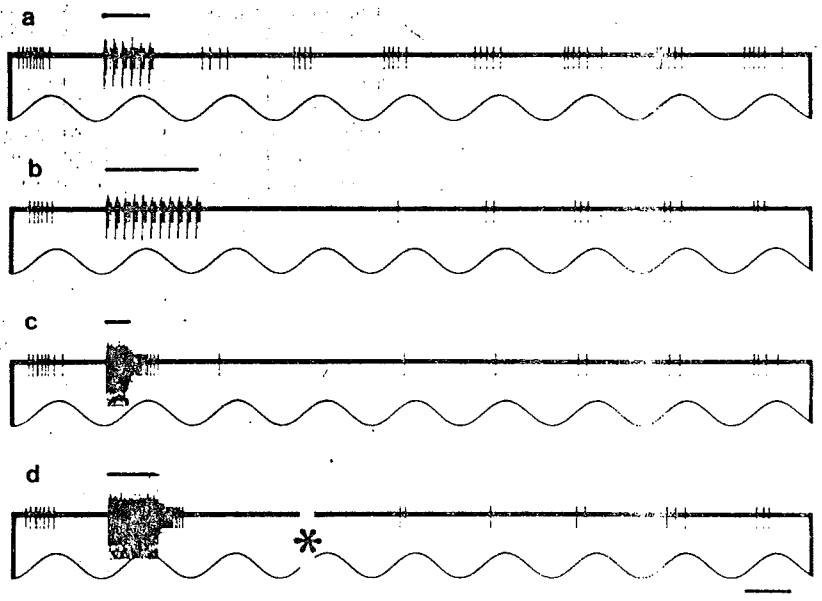

Fig. 11. Effect of FASN activity on resistance reflex activity. Whole animal preparation, stimulating the FASN (upper bar) distal to its junction with the FMN bundle. Activity recorded from the FMN/FASN bundle proximally. Top trace: FMN activity; lower trace: leg-mover motor amplifier readout, showing approximately 10° movements at the M–C joint. Joint extension is upwards. Joint angle initially set to approximately 100°. The normal resistance reflex activity, which at this level involves only the spontaneously active tonic unit, can be seen in the cycle just prior to stimulation in each case. (a) Stimulation of the FASN for 1 sec at 5 Hz (0.5 msec, 10 V) produces mild FMN inhibition, although each stimulus pulse elicits its own reflex FMN activity (similar to that shown in Fig. 9(d)). (b) Longer stimulation of the FASN at the same stimulus level produces complete FMN inhibition for two cycles. Resistance reflex activity then returns slowly. (c) With high frequency stimulation (50 Hz, other stimulus parameters as before) a one-half second train stimulation is sufficient to induce FMN inhibition. Note that a short burst of tonic unit activity occurs at the end of the stimulation. (d) One second of high frequency stimulation causes extended FMN inhibition. At *, 4 cycles of complete inhibition have been removed for ease of presentation. Again, this high frequency stimulation has elicited a post-stimulus burst of tonic unit activity. Once restarted, resistance reflex activity returns to pre-stimulus levels over a similar time course in each case. Scale bar: 1.0 sec.

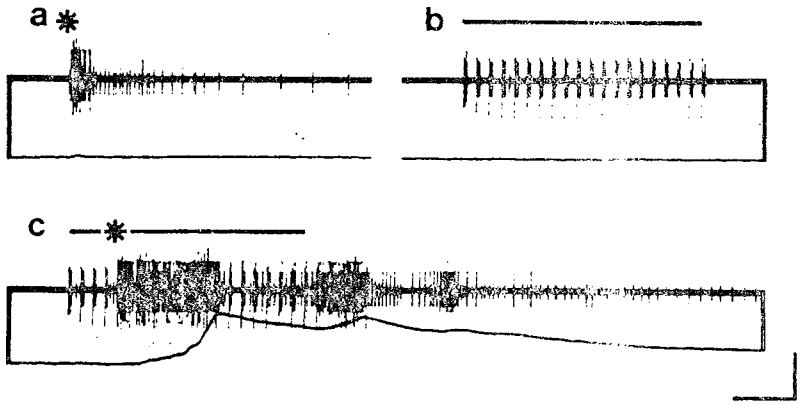

Fig. 12. Effect of FASN activity on centrally produced (non limb-reflex) FMN activity. Same preparation and same stimulus voltage, pulse duration and frequency used as in Fig. 11. Top trace: FMN activity; lower trace: tension. Upper bars show duration of electrical stimulation. (a) Responses of FMN to brief abdominal-flap stimulation at *. In this animal, the response was briefer than in other animals (e.g. see Fig. 5, Parsons, 1982). (b) Responses of FMN to FASN stimulation for 4 sec whilst animal is at rest. Only the immediate reflex FMN activity associated with each stimulus pulse is present. (c) Effect of concurrent FASN stimulation on the response to abdominal flap stimulation (at *). The activity in the FMN is greatly increased and extended. In some animals, a period of inhibition follows this strong FMN activity. Scale bars: 1 sec; 20 g.

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duration of the applied force and the speed of the tension rise induced by the force can modify the intensity of tension receptor discharge. When the length of stimulation is increased, the duration of receptor output also increases (Fig. 1). Although the overall relationship is clear, much of the detail of the transformation processes between motor impulse and tension receptor discharge is masked by unknown anatomical and physiological factors. Some of these factors are discussed below.

In the resting animal there is some variation in the level of spontaneous discharge. FASN activity recorded in these situations included zero activity; steady, very low frequency discharges of one, or rarely two units; and patterned discharges of one unit. The amplitude of some of the activity could be below the noise level of the recording system. In other arthropods, the tension receptors also show a range of spontaneous activity (Macmillan & Dando, 1972; Eagles, 1978). Macmillan and Dando found that spontaneous activity was present in the tendon nerve (recorded in a more proximal segment) even when the segment containing the tension receptors was not dissected or damaged. The discharge properties of the receptors might also be influenced by the bathing medium. Peetz & Winter (1980) have shown that active substance in the haemolymph can alter the generator potentials and discharge frequencies of the receptor cells of the crayfish abdominal muscle receptor organ (MRO). Artificially prepared crustacean salines must lack these substances and so receptor discharges may be affected. This does not appear to be a problem in these preparations because FASN activity recorded in whole animal preparations by means of a small "window" in the merus cuticle was not noticeably different from that recorded in the more dissected preparations that were bathed with the crab saline. Individual variability undoubtedly also contributes to the variation in spontaneous discharge of the tension receptors.

In vertebrates, muscular tension is monitored by the Golgi Tendon Organs (GTO's). In the higher vertebrates, the GTO's are intimately associated with the muscle fibers themselves. They lie in series with some fibers and in parallel with others (Stuart *et al.*, 1972). In the lower vertebrates such as the lizard, the receptors lie in the tendon, at a distance from the muscle–tendon junction. Because of this the receptors probably respond to overall tensions as well as localised tensions produced by individual muscle units (Proske, 1975). The arrangement of the lizard receptors resembles that of the crustacean leg apodeme (=tendon) receptors described here. The ultrastructural and physiological evidence shows that in the crustacean receptors tension transduction occurs within the apodeme: the transduction locus is separate from the contractile apparatus. These similarities suggest that the apodeme tension receptors may be sensitive to whole muscle tension as well as to the localised tensions produced by the contractions of individual muscle fibers or motor units. These differences have not been investigated here.

Significant tension receptor activity does not occur unless the apodeme bends. Filming the apodeme movements allows one to conclude that apodeme movement is closely associated with tension and FASN activity (Fig. 4). It does not give information on localised tensions that may affect apodeme bending, on the relationships of these localised tensions to the whole tension experienced, or of the tension to apodeme-flexion process. Whilst the apodeme possesses an underlying symmetry due to its formation by cuticular invagination (Parsons, 1980), the external morphology of the apodeme is quite complex (e.g. see Macmillan & Dando, 1972). Because of this, the movements of the anterior edge of the apodeme (Fig. 4) are unlikely to be related in a simple way to local tensions produced by the muscle fibers. The activity of the receptors does not depend on the presence of the muscle fibers, and so the relationship between receptor activity and apodeme flexion should be amenable to *in vitro* analysis. The analysis of the relationship between apodeme flexion and motor unit contraction in this muscle is likely to prove more difficult.

A number of factors influence the degree and direction of apodeme movement and bending during contraction. Several fast or slow motor units could be involved in flexor contractions, and the direction and extent of apodeme movement may depend on which unit or units are being used. The muscle fibers are not symmetrically arranged about the apodeme, and so the distribution of forces about the apodeme will be also influenced by this asymmetry. In most cases, however, the apodeme moves towards the axis of the merus (i.e. dorsally) during contraction. It is not yet known how the inhibitory motor axon (or axons, see Parsons, 1982) affects the flexor contractions.

In the vertebrate GTO's there appears to be no simple relationship between the contractions of an individual motor unit and the intensity of the response. As might be expected, some motor units can exert a more powerful excitatory effect on the receptors than others (Proske, 1979). Similarly, in the crab tension detection system, no clear correlation is found between the activity of apodeme tension receptor units, or of the whole FASN activity, and the contractions of the individual motor units. Faster units naturally elicit stronger and shorter-latency receptor activity than do slower units.

The poor response of the receptors to a slow rate of muscle-induced tension increase may be a consequence of the distribution of the muscle fibers. Slow contractions of the flexor can be performed by the slow fibers situated at the proximal, and to a lesser extent, the distal end of the flexor. The proximal fibers are distant from the tension receptors which are distributed along the distal half of the apodeme. The lateral (dorso-ventral) vector of the contractile force of these fibers is minimal there. Slow contractions are thus transferred to the carpus with little concurrent apodeme bending and therefore little activation of the tension receptors.

Slow passive movements also elicit very little tension receptor activity. During passive movements of the carpus, all muscle fibers are quiescent, and they would tend to resist any apodeme movement that caused muscle lengthening because of their inherent visco-elastic properties. In slow passive movements, lengthening of the muscle fibers would be slow enough to allow the fibers to stretch at the same rate that the imposed apodeme movement demands, and

so the apodeme would not become appreciably distorted. In contrast, in fast passive movements, the speed of the imposed lengthening probably exceeds the capability of the muscle fibers to stretch at the speed the induced apodeme movement demands. The asymmetry of the fibers about the apodeme would then result in an imbalance in the forces resisting apodeme movement, and so distort the apodeme and elicit tension receptor activity.

The ultrastructural basis of the responses of *Limulus* tailspine and limb tension receptors is not yet known, but it is of interest to note that many of the dendrites of the cell bodies of the tailspine receptors, and some of the limb tension receptors, apparently terminate in the area of the muscle insertions onto the apodemes. The tailspine apodemes are pliable (Eagles & Hartman, 1975) and so could also be distorted during tailspine movements. These features suggest that the tension transduction mechanism in the tailspine receptors might be similar to that found here in the *Carcinus* flexor apodeme. Ultrastructural examination of the *Limulus* tension receptors would be required to test this hypothesis.

In intact animals, tension receptor activity is dependent on the rate of development and the duration of the tension as it is in isolated leg preparations. In isotonic contractions against zero load, carpus movement is unimpeded and little or no FASN activity is recorded. Zero-load contractions may not normally occur, but the result does show that none of the receptors present is sensitive to muscle-length changes, as are some of the receptors in the *Limulus* tailspine and limb segments (Eagles & Hartman, 1975; Eagles, 1978; Eagles & Gregg, 1979). On the other hand, increased muscle length produces increased resting tension and so isometric contractions from a higher base tension show greater activity to a given stimulus (Fig. 3).

Some effects of tension receptor activity on motoneurone output have been reported previously (Clarac & Dando, 1973; Macmillan, 1976). Clarac & Dando (1973) found that stimulation of the tension receptor nerve inhibits resistance reflex motoneurone activity in the homonymous muscle and causes some degree of excitation in the motorneurones of the opposing muscle. In the flexor muscle of *Carcinus*, stimulation of the tension receptor nerve also causes inhibition of resistance reflexes (Fig. 11), however, some FMN excitation precedes the inhibition, as can be seen in the response to single-pulse FASN stimulation (Fig. 9). Only train stimulation causes resistance reflex inhibition. Single pulse stimulations cause only brief excitation of the FMN's, and the level of reflex FMN excitation depends on stimulus strength. Usually only two FMN units are involved – the tonic unit that is spontaneously active in the resting animal, and a large phasic unit. The phasic unit is active only at the higher stimulus strengths. Sometimes a third, small amplitude FMN unit is also recruited (Fig. 9).

The effect of tension receptor activity on non-resistance reflex FMN activity is quite different. Low strength single-pulse stimulations again result in the FMN excitatory activity described above. After a certain threshold is reached, repetitive stimulation of the FASN in the resting animal usually causes a massive discharge in most of the FMN units, followed by a period of inhibition of FMN activity (Fig. 10). In a few animals, a very high strength single pulse stimulation often had the same effect. When FASN stimulation is performed in the presence of increased centrally driven motor activity, the effect is to greatly increment, and then in some cases, to inhibit the discharge of the flexor motoneurones (Fig. 12). It is clear that either positive or negative feedback onto the motoneurones by the tension receptors can occur in *Carcinus*, even within one response.

It is not possible to directly compare previous findings on the effects of tension nerve afference (Clarac & Dando, 1973; Macmillan & Laverack, pers. comm.) since different animals were used, and in some cases the details of the stimulus regimes were not reported. Some general comparisons can be made however.

Spontaneous motor activity is inhibited in *Cancer* by apodeme nerve stimulation (Clarac & Dando, 1973; Macmillan and Laverack, pers. comm.) as is increased motor activity produced by a variety of stimulus methods (Macmillan and Laverack, pers. comm.). In *Carcinus* however, the opposite effect occurs. FASN stimulation has an excitatory effect on spontaneous motor activity (Figs. 9, 10) and this tension nerve afference is also excitatory if higher levels of centrally produced FMN activity are present (Fig. 12).

As noted above, resistance reflexes in both *Cancer* and *Carcinus* are inhibited by apodeme nerve stimulation, and the degree of inhibition is dependent on stimulus strength in both cases (Clarac & Dando, 1973; Fig. 11, this paper).

Clarac & Dando (1973) found that "high strength" sensory nerve stimulation produces strong motoneurone discharge in *Cancer*. In *Carcinus*, high strength sensory nerve stimulation produces extensive motoneurone discharge when the animal is at rest (Fig. 10), but if resistance reflexes are operating, inhibition occurs (Fig. 11). Unfortunately, Clarac & Dando (1973) did not note in which situations the motoneurone excitation they observed occurred.

Clearly some of the motor responses to tension nerve stimulation in *Cancer* are very different to those in *Carcinus*. In particular, Figs. 9, 10 and 11 show that whether the overall effect of FASN stimulation is excitatory or inhibitory, the immediate response of the flexor motoneurones to each stimulus pulse is always one of reflex excitation once the FASN stimulus threshold is reached. There is no evidence of this immediate excitatory effect in the responses of *Cancer* to tension afference. The reasons for this difference in response are not known.

DiCaprio & Clarac (1981) have found that the resistance reflexes associated with passive movement of the thoracic coxal joint in *Carcinus* could in some cases become assistance reflexes. These reflexes were mediated by afference from the muscle receptor organ situated in the joint (the TCMRO). The change in reflex occurred when the level of central activity was increased. They concluded that the effect of sensory information from the TCMRO was being modified centrally. Similarly the effect of apodeme tension nerve afference on resistance reflexes in *Cancer* can become excitatory when the animal is active (Macmillan, 1976). In *Carcinus*, tension nerve afference elicits a different overall motor response depending on

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whether the limb's M C resistance reflex is operating (Fig. 11), or whether other stimuli such as abdominal flap extension (Fig. 12) are affecting the output of the motoneurones.

The understanding of the effects of flexor tension receptor afference is complicated by the dual capability for positive and negative feedback in any response. The effect of a single, just supra-threshold, stimulus pulse to the FASN, is a brief, short latency excitation of a small number of flexor motoneurones (Fig. 9). On the other hand, repetitive stimulation at the same stimulus level can evoke strong excitation (Fig. 12), excitation followed by inhibition (Fig. 10), or inhibition (Fig. 11).

An increase in the number of FASN units interacting with the central motoneurones can also result in strong motoneurone excitation in some animals, and again it is followed by inhibition in some cases. An understanding of the multiplicity of effects of tension receptor afference on motor output found here awaits anatomical study of the connections.

In the experimental manipulations performed here, the responses of the PSC's are essentially similar to those of some of the DSC's. Why this is so, and why these two receptor groups are anatomically so distinct is still unknown.

because they may respond to different muscle fibers (Fig. 5)

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