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THE ORGANIZATION OF INPUTS TO MOTONEURONS OF THE LOCUST METATHORACIC LEG

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CONTENTS

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1. Resistance reflexes at each joint of the leg arise from sense organs directionally sensitive to motion. Sensory impulses, acting indirectly through interneuron combinations, excite one set of motoneurons and inhibit their antagonists.

2. These resistance reflexes are not switched off during 'voluntary' movements elicited by touch elsewhere on the body, but instead they sum with other responses.

3. Although readily recorded in motoneurons, the resistance reflexes are mechanically so weak compared with voluntary movements that they are not a major item in the control of the leg. This applies also to posture.

4. When a voluntary movement or direct stimulation of a motoneuron causes a muscle to act against a *load* the reflexes upon motoneurons are quite different from those to motion caused by the same muscle. Therefore the reflex modification of walking patterns, in which legs are loaded by each other's movements, and by the insect's weight, cannot be analysed by a study of leg *motions*, although this has always been the method to date.

5. The positive feedback upon the motoneuron from receptors to loading of the muscle is exemplified in the jump. Between motoneurons to the femur muscles there are other neuronal interactions which accommodate the mechanics of the jump.

6. The angle of the tibial–tarsal joint depends upon sense organs which monitor the angle of the femoral–tibial joint. This inter-joint reflex sums with the local resistance reflex, centrally determined posture and voluntary movements.

7. The study of postsynaptic potentials (p.s.ps) makes possible a number of inferences about the nature of the interneuron pathways antecedent to the motoneurons.

8. Motoneurons to the same muscle usually have a high proportion of p.s.ps in common. Fast motoneurons have phasic properties because they have higher thresholds than the corresponding slow ones, as shown in situations where the p.s.ps are similar.

9. Motoneurons to antagonistic muscles can have synchronous p.s.ps of opposite sign.

10. The intraleg reflex in (6) is controlled by separate interneurons to the motoneurons that are excited together.

11. Study of the inputs upon groups of named motoneurons allows identification of the interneurons which act on them, and reveals that posture, elicited responses and spontaneous movements are determined by selection of different combinations of interneurons.

12. The same method, of recording simultaneously from several known central neurons can progressively reveal the unknown mechanisms by which interneurons integrate sensory input and generate coordinated responses.

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Introduction

Much has been published on the control of walking in arthropods and the part played by 'resistance' reflexes that oppose passive motion of the joints of a leg. The reason for adding a further account is that an impasse was reached when recordings could be made only from sensory and motor axons, whereas the present work is concerned mainly with the synaptic inputs to the motoneurons recorded in the central nervous system. The main problem in dealing with the electrical events in central neurons is to digest the data in such a way that mechanisms are revealed. Volumes of oscilloscope recordings, catalogued neuron by neuron, have to be fitted together in the context of normal coordinated activities. As a side issue, we soon find that the appropriate details of behaviour, and the action of sense organs and muscles, are not available to be matched with the progressively discovered properties of the central neurons that actually coordinate them. We have tackled these problems by interpreting our recordings in terms of flow diagrams for the physiological interactions at every stage, and then indicating, and sometimes filling, the gaps in the models.

The effects of ground contact and resistance to motion of the leg have been considered as important in the generation of walking patterns because large changes in gait follow amputation of a leg (von Buddenbrock 1921; Delcomyn 1971) and because recording of myograms shows that resistance reflexes persist under experimental conditions at the highest frequencies of stepping that occur in walking (Wilson 1965). Sense organs known or suspected to be involved in different ways are the companiform sensilla of the trochanter in the cockroach (Pringle 1939), the chordotonal organ of the femoral–tibial joint in the locust (Usherwood, Runion & Campbell 1968), the hair plates of the coxa in the stick insect (Wendler 1964) and the numerous sense organs of the tarsal joints and footpads (Kendall 1970).

Central programmes for the control of leg movements also have been inferred (*a*) from the similarity between the motor impulse pattern in a normal movement of the leg and that in a subliminal response which excites no proprioceptors (Hoyle 1964), (*b*) from the weakness of inter-leg reflexes and inflexibility of the phase relations in the resistance reflexes (Wilson 1965), and (*c*) from the recordings of fairly typical efferent impulse patterns after sensory excitation has been partially eliminated (Pearson & Iles 1970, 1973; Pearson 1972).

Now that it is possible to record postsynaptic potentials in identified motoneurons in an insect with legs free to move (Hoyle & Burrows 1973*a*, *b*), some of the convergence of excitation upon the motoneurons as they participate in a variety of responses can be directly observed. Furthermore, by recording from two or three motoneurons together we observe now that patterns of inputs to them differ for various reflexes, elicited and 'voluntary' movements, and that proprioceptive effects usually sum with inputs from other interneurons. Such recordings allow examination of the output of particular interneurons that are identified by their p.s.ps upon the motoneurons.

We try to maintain a distinction between the different types of response. *Resistance reflexes* are properties of single joints, in which proprioceptors indirectly excite the joint muscles to oppose an imposed motion. In this paper other so-called reflexes, as produced by bending joints of other legs, touch to other parts of the body or even to other sense organs of the leg itself, are called '*elicited responses*' because they are usually not stereotyped, and analysis shows that they are better considered as triggered central commands. The distinction is arbitrary, but the main purpose of this paper is to examine the summation between the stereotyped proprioceptive

reflexes and other responses in which it is impossible to draw sharp dividing lines between arousal by sensory inputs, triggered central commands, 'voluntary' movements and 'spontaneous' background activity.

Methods

For recording from motoneurons the *Schistocerca gregaria* was embedded on its back in Plasticine and the intact metathoracic ganglion supported on a platform in a flow of locust Ringer solution as described by Hoyle & Burrows (1973*a*). In different parts of the work impulses were recorded from nerves *en passant* by a pair of 100 μm platinum-wire hooks while protected from desiccation by petroleum jelly, and from muscles by insulated 50 μm silver wires embedded in them. Leg movements were recorded by a black flag which passed in front of a photocell, and torque at the femoral–tibial joint was recorded by an RCA 5734 transducer. Electrodes were filled with 2 M potassium acetate solution and had resistances of about 40 MΩ. The leg could be moved passively by a solenoid driven by a waveform generator with the RCA 5734 mounted between the leg and the driving arm to record the torque reaction of the tibia. The abbreviations for the motoneurons, e.g. FETi, fast extensor tibiae motoneuron, are taken from Hoyle & Burrows (1973*a*).

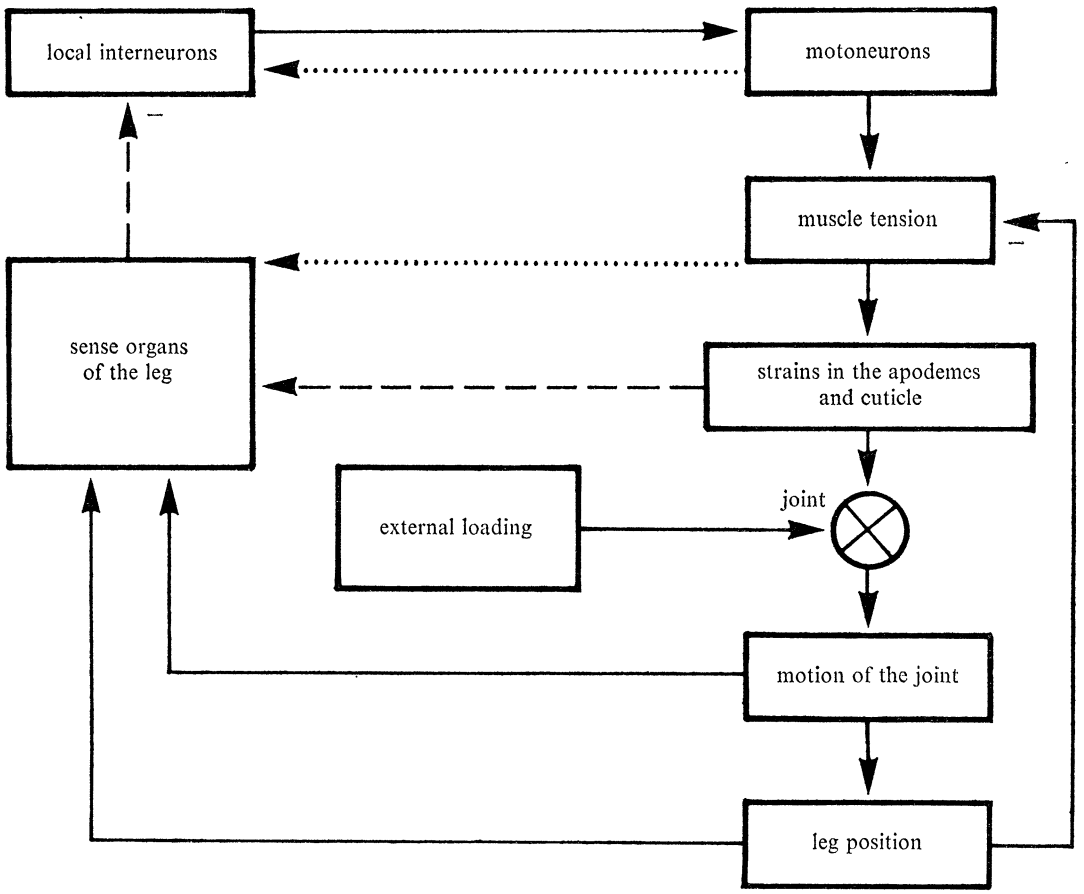

Figure 1. Feedback loops in the effects of motoneurons upon sense organs of a joint. Solid lines show subsequently *observed* effects, dotted lines show further *possible* effects not subsequently eliminated by experiment and dashed lines show *inferred* effects, a convention used in all block diagrams. The diagram serves for either extension or flexion about any joint.

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Results

The control system for one joint

To present the details of synaptic events in motoneurons it is convenient to have a scheme of relationships in mind so that any response can be placed among many other interrelated events that proceed simultaneously. All known relations, and especially the feedback loops which can confuse the interpretation of recorded events, are brought together in flow charts which act as a guide to the system and as a caution in the interpretation of results. Complex interactions emerge from the experimental results, but the basic interactions between muscles, joints, sense organs and central commands can be drawn out from established work, and some anticipation of findings.

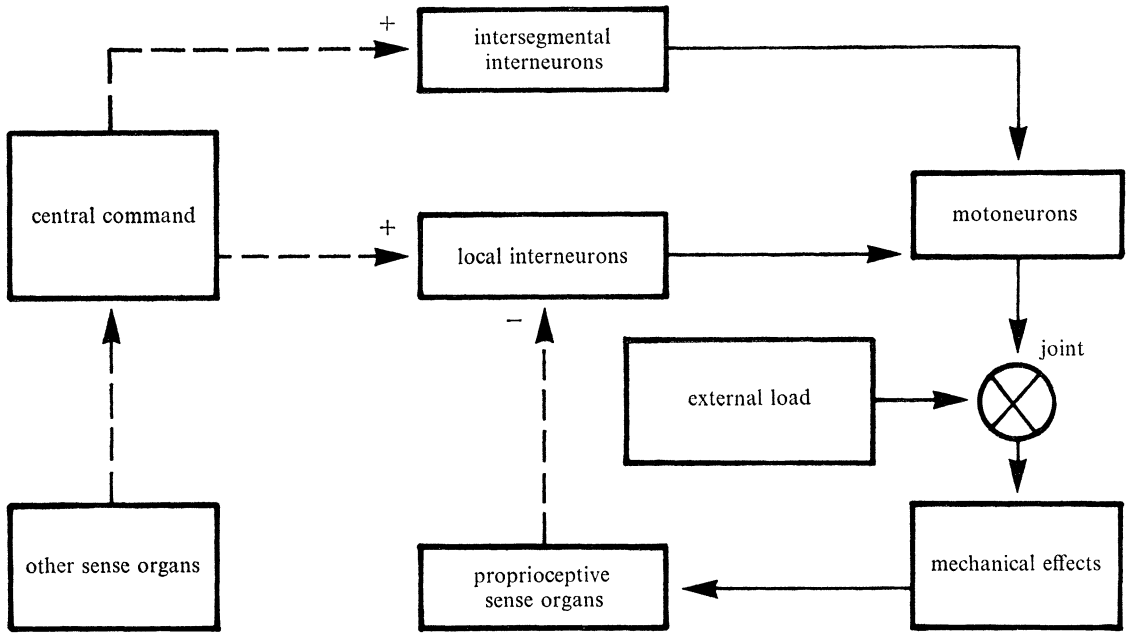

Figure 2. Abbreviated form of figure 1 showing the point in the sensory feedback loop at which central commands interact upon the local interneurons. A problem is caused by the lack of clear distinction between a central command and excitation of a local interneuron, and so to be inclusive of all phenomena 'central command' is the name given to whatever it is which sets off an organized response apart from proprioceptive reflexes. Solid lines show subsequently observed effects; dashed lines, inferred effects.

The first step is to clarify the relation between active and passive movement of a joint in relation to its sense organs, motoneurons, load and movement (figure 1). To draw the relationships in this way implies attention to experiments that bear upon the following points:

(*a*) In insects no efferent control of sense organs is known except that caused by contractions of typical somatic muscles.

(*b*) Sensory fibres are in no example known to connect directly with motoneurons.

(*c*) No central effect of motoneurons on sensory pathways has been found.

(*d*) Intraganglionic effects of motoneurons on interneurons are as yet unknown.

(*e*) Prevention of movement necessarily leads to new sensory effects.

One use of our electrophysiological data has been the examination of the above statements, as described in later sections. The central commands can now be dovetailed into this system in

only one way which meets all requirements, as in figure 2, which is based on subsequent findings as follows:

(*a*) Resistance reflexes represented by the feedback loop are effective during movements that are initiated centrally.

(*b*) Recording from motoneurons shows only summation of p.s.ps from interneurons.

(*c*) Central commands act via interneurons, not via sensory neurons or directly upon motoneurons, as shown by the lack of identical inputs to most pairs of motoneurons in simultaneous recordings.

(*d*) Responses initiated locally by touch to the same segment are best considered as locally triggered central commands because a particular selection of motoneurons is excited by a pattern of interneurons.

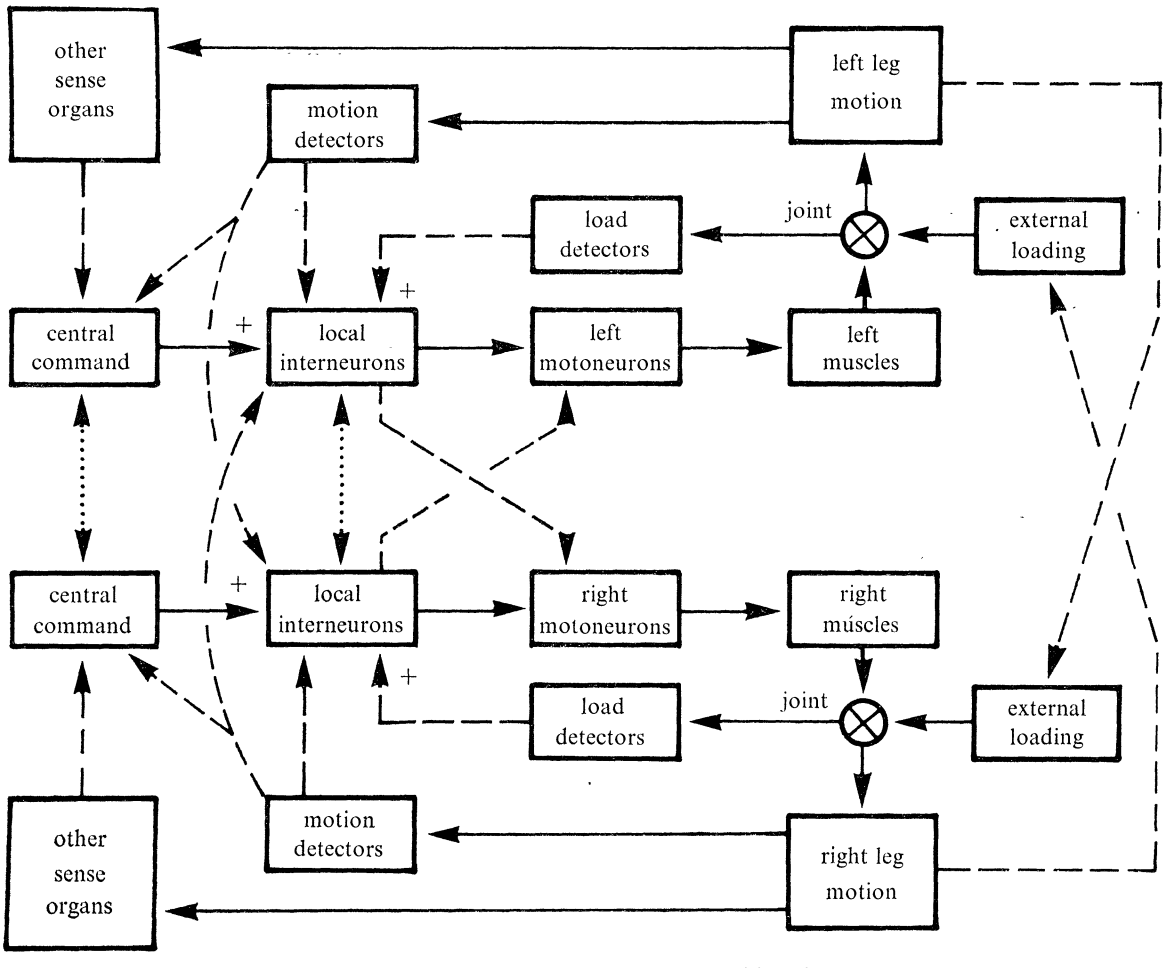

FIGURE 3. Extension of figure 2 to include the two sides of the segment.

Experiments will be directed to each of the above points, but figures 2 and 3 suggest that the system will have the following properties which govern the design of experiments.

(*a*) It is impossible to see the central commands in isolation unless the feedback loop is made ineffective.

(*b*) By particular central commands, antagonistic muscles can be excited together, and muscles can be excited at apparently inappropriate times.

(*c*) On account of the feedback loop, central commands do not necessarily generate constant patterns of motor impulses or movements.

(*d*) Effects of tonic sensory impulses signalling leg *position* must have some effect on control of movement to prevent drift.

(*e*) Whether the gain in the resistance reflex is itself under central control is now open to experiment.

(*f*) The relative strengths of central commands and reflexes becomes significant.

The system in figure 2 is incomplete in that it fails to distinguish influences from other joints of the leg, other segments, and the contralateral leg of the same segment; also it portrays inadequately the antagonistic muscles, the effect of load, the directionality of the sense organs and the numerous neuron pathways in parallel. The relations between the two sides of the animal are symmetrical (figure 3) and based on the following findings.

(*a*) Interactions from the contralateral leg are weak as recorded electrophysiologically but stronger via movements that would be caused in the leg itself by traction on the ground.

(*b*) Coordination between the two sides is done by interneurons, as inferred from the anatomical limitation of sensory and motoneuron arborizations to the side where their soma lies. This has been shown in unpublished results of cobalt ion electrophoresis on the locust ganglion.

The relations between the flexion and extension systems are more complex (figure 4) and the hypotheses embodied in the flow diagram approach the limits to be demonstrated in the recordings from motoneurons. This arrangement is an enlargement of figure 2 based upon the following observations of reflexes.

(*a*) Flexion and extension motions exert nett effects centrally in opposite directions to each other and usually with opposite but indirect effects on flexor and extensor motoneurons.

(*b*) Other interneurons can excite flexors and extensors simultaneously and usually do so in elicited responses.

(*c*) Leg motions are more effective stimuli than leg positions.

(*d*) Responses of peripheral inhibitory neurons are complex.

The most serious remaining inadequacies in figure 4 arise from a variety of subsequent findings and limitations of the methods, as follows.

(*a*) The mechanical effects of the muscles depend on joint position.

(*b*) Indicators of load are significant, and it cannot be assumed that the leg is free to move.

(*c*) Special situations such as contact with the ground certainly involve a switch from one system of control to another not studied here because the animal is pinned on its back.

(*d*) Flexion and extension receptors may overlap in their central effects, because only nett effects are observed.

(*e*) Cessation of background activity when a proprioceptor is moved in its null direction could be a significant stimulus. However, no findings support this idea and the lack of a background inhibitory input upon most motoneurons is against it.

(*f*) The components in the locust are duplicated, so that many sensory neurons, many interneurons, many motoneurons, at least seven to the flexor muscles, and at least three inhibitors, run in parallel. Theoretically, in a small system of identifiable neurons recordings can eventually be made from all.

(*g*) Electrophysiological recording may not reveal all the inputs to a motoneuron because fine dendritic branches may be too far from any electrode wherever inserted. This is an insurmountable technical limitation.

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(*h*) Inputs upon interneurons are only inferred, so that the local interneuron pool becomes the convenient repository for interactions that have not been directly observed.

Experiments will reveal further inadequacies but we have now reached a point where the components of the control mechanism of the leg and their responses can be examined with this system of interactions as a framework for design of experiments.

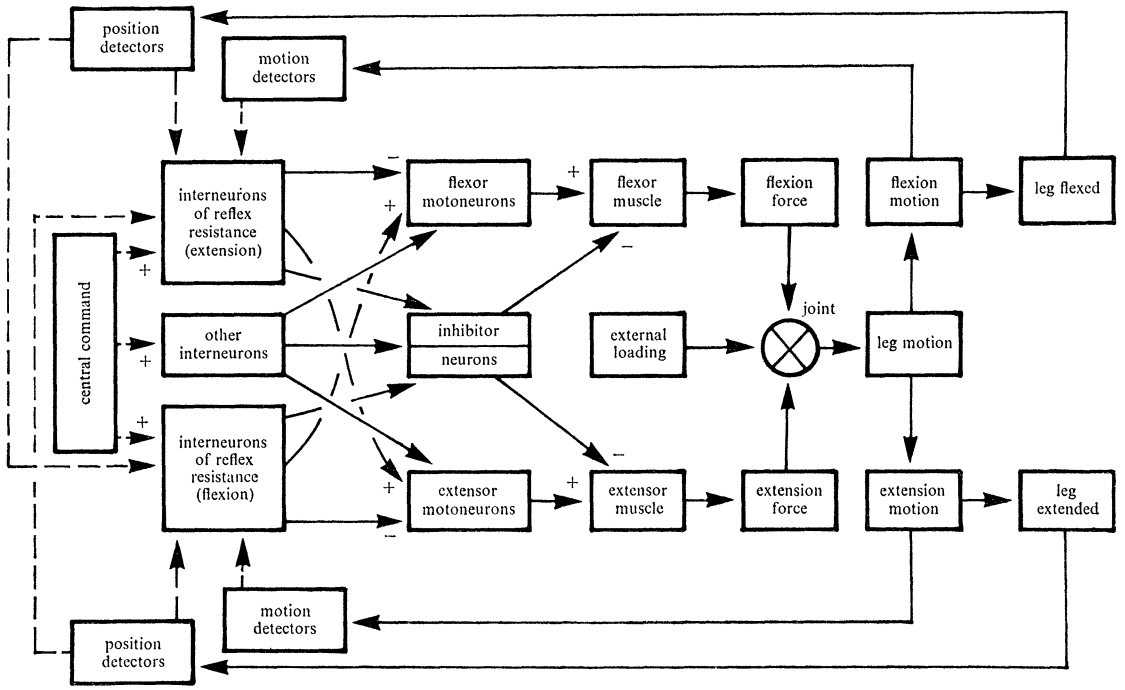

FIGURE 4. Extension of figure 2 to take account of flexors and extensors and the summation of their effect with external load at the femoral-tibial joint to produce leg motion. Receptors for loading of the joint, and their effects, are omitted (see figure 8). The place of the inhibitors is not yet clear. This diagram serves equally well for the tibial–tarsal joint. Most of the components shown here are not single, but numerous elements in parallel.

The tibial–tarsal joint

Components

A rich variety of sense organs, including campaniform sensilla, hair sensilla of various kinds, and chordotonal organs, is clearly one of the features of the tarsus (Kendall 1970). The responses of one type, the large hair sensilla, are phasic spikes to delicate touch (Runion & Usherwood 1968). Pressure to the foot pad and bending of the tarsal joints readily elicit sensory impulses in the leg nerve and responses from central neurons, but the sense organs have not been identified.

The tibial–tarsal joint is operated from the tibia by depressor and levator muscles which are each supplied by several motoneurons including fast and slow, and possibly by axons of inhibitor neurons. In addition to those formerly described (Burrows & Hoyle 1973) we have found a second slow depressor motoneuron. The tendons of these muscles extend distally and work the tarsus as a whole, so that it acts like a foot. The terminal claw is separately depressed by the retractor unguis muscle which has parts on the dorsal side of the femur and in the tibia, and is supplied by at least two motoneurons (Usherwood & Machili 1968).

Maintenance of tarsal posture

The tarsal joint is held at a particular 'set point' by continual background activity in the slow levator and depressor motoneurons. As described below, these frequencies are controlled centrally and also influenced by the angle of the femoral–tibial joint. The continual impulse initiation by the slow depressor tarsus and slow levator tarsus motoneurons is augmented and inhibited by appropriate opposite directions of motion of the joint; levation of the tarsus excites the depressor motoneurons and inhibits the levators (figure 5*a*, *b*). This applies to the fast and slow motoneurons (figure 5*c*, *e*) but we have not recorded from both types simultaneously to

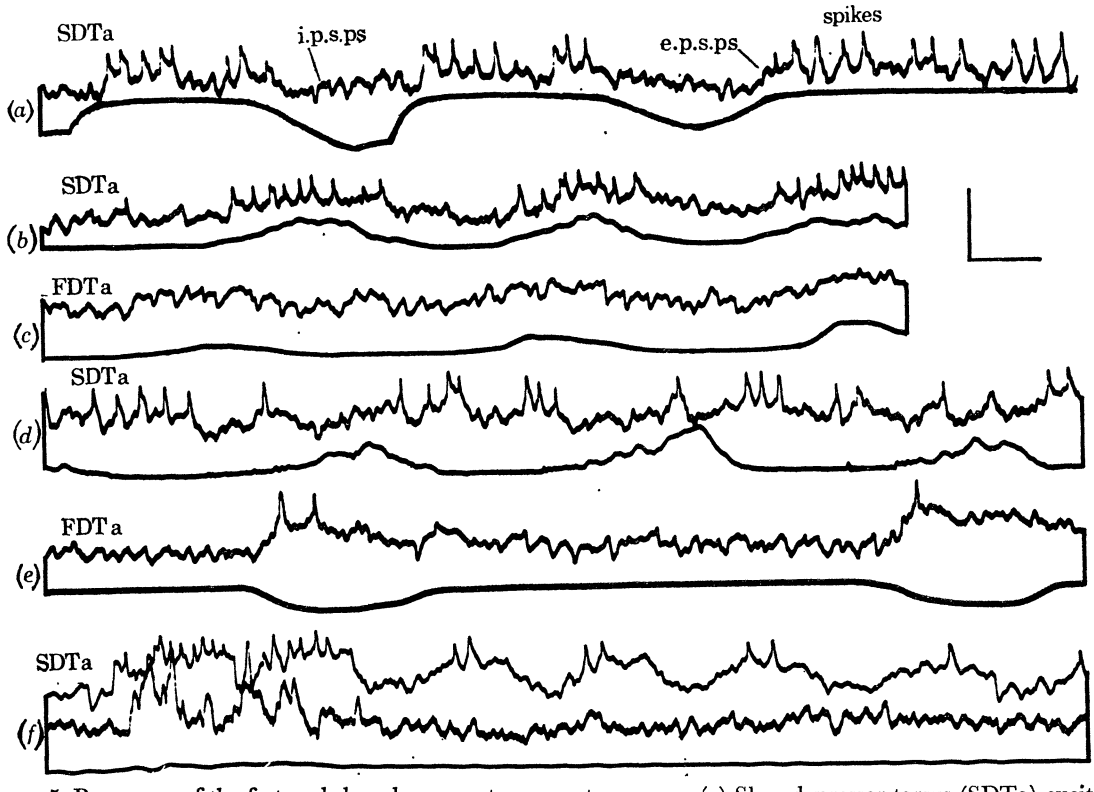

FIGURE 5. Responses of the fast and slow depressor tarsus motoneurons. (*a*) Slow depressor tarsus (SDTa) excited by levation of the *tarsus* on lower trace and inhibited by depression, with the femoral–tibial joint fixed at 130°. (*b*) As in (*a*) but with the tibia at 90°, so that there is a lower background spike frequency before the imposed movement. (*c*) The fast depressor tarsus (FDTa) is depolarized by a levation of the *tarsus* but does not spike. (*d*) A 10° flexion (up) of the *tibia* about 90° inhibits the SDTa. (*e*) The FDTa is excited and may occasionally spike to a 20° extension (down) of the *tibia* about 90°. (*f*) SDTa (upper trace) and a flexor (PFFlTi). The tarsus is passively levated by a touch which causes excitation followed by oscillations of the membrane potential back to the set point. The flexor gives an independent elicited response but no oscillations. Calibration: vertical (*a*–*e*) 4 mV: (*f*) trace 1, 12 mV: trace 2, 5 mV: horizontal 200 ms.

know whether they have common inputs. In some locusts displacement of the tarsus from its set point results in an oscillatory motion, presumably because the reflex control system overshoots when the tarsus is not in contact with anything (figure 5*f*). Levation of the tarsus causes excitation followed by a damped oscillation in the slow depressor tarsus (SDTa), with no effect upon a flexor recorded at the same time. The oscillation slowly declines as the centrally determined set point is regained. The set point about which these reflexes operate depends on the angle of the tibia about the femur (figure 5*a*, *b*).

Although sensory impulses from the opposite side of the segment normally have a weak effect on motoneurons, a strong influence often can be found upon the tarsal joint. Levation of the contralateral tarsus causes inhibition of the depressor tarsus and conversely depression causes excitation, following the general rule that sensory inflow from the contralateral leg indirectly evokes p.s.ps of a sign opposite to those to the corresponding motoneuron of the ipsilateral leg.

Responses to stimulation of the tarsus

Previous work shows that stimulation of the hairs at the sides of the tarsal pads is followed by additional impulses in the slow extensor tibiae motoneuron (SETi) and one of the inhibitor neurons, the common inhibitor (CI) (Runion & Usherwood 1968). When the tarsi are lifted off the ground phasic impulses no longer originate from the large hairs at each small movement, and at the same time the background frequency of spikes in the SETi and the CI drops to a low level. When one tarsus is removed the angle between the tibia and femur increases and when both tarsi are removed the stumps are held off the ground (Runion & Usherwood 1968). Therefore tarsal receptors are important in posture of other joints of the leg, and they must be active during walking, but other sensilla of the tarsus may be involved, other muscles may be acted upon, and the effects of ground contact and loss of contact are undescribed. Runion & Usherwood (1968) observe that the inhibitor is active just prior to tibial flexion in slow walking, and suggest that it serves to accelerate relaxation of the extensor muscle, but they have no means of showing whether the mechanism of activation is central or peripheral, whether tarsal sense organs are involved, or what other effects the inhibitor may have.

Although the tarsus has a variety of receptors we were unable to find responses of motoneurons which suggest a range of specific pathways to particular motoneurons. Possibly there exists a diversity of specific inputs upon interneurons that generate the walking movements and other movements such as grooming, but we do not see these because we record only from a limited repertoire of motoneurons in the resting inverted animal. For example, touch to the tarsus generally excites all the motoneurons and inhibitor neurons to the leg, and never inhibits them. Similarly, touching the underside of the tarsus stops flight under experimental conditions, but does not cause direct inhibition of the flight motoneurons (Burrows 1973*b*) showing that the mechanism of interest precedes the motoneuron response. Loss of the foot, or loss of contact with the ground affects many motoneurons but the nett effect is observable only as a final movement of the leg. Our results do not predict a particular modulation of the walking pattern by the tarsal sense organs, instead they illustrate how the electrophysiological analysis reveals interactions which may underly the observed responses, but does not allow predictions of the effects of numerous neurons in parallel.

The femoral–tibial joint

Components

Sense-organs related to this joint. The chordotonal organ (CO), lying within the distal end of the femur, is attached by a tendon to the tibia (Usherwood *et al.* 1968). There is a resting spike discharge, and the *summed* tonic units have a minimum impulse rate at a joint angle of about 60°, increasing with change in either direction. Phasic responses are extremely sensitive to a movement of 3′. Extension is a stronger stimulus than flexion but both directions are effective. Removal of the CO has the postural tonic effect of increasing the femoral–tibial angle, and in

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walking the joint now moves in a larger arc, the frequency of stepping is reduced, the extensor muscle fails to act properly and alternation between the legs of the two sides is less regular.

No doubt many motoneuron responses to joint motion are due to the chordotonal organ, but inferences about its central action are limited because we lack the following details: (*a*) whether some or all units are directional in their response, (*b*) whether the two directions of motion excite the same or different units in different parts of the range, (*c*) whether vibration at different parts of the range excites different units which could thus signal position although not tonically.

Another type of sensory neuron occurs in three groups in the cuticle of the femoral–tibial joint (Coillot & Boistel 1968, 1969). These neurons are tonic and extension excites two groups, but although their response properties are documented in detail, the part they play in control of posture is unknown.

Motor components. The extensor muscle has a fast axon (FETi) which causes a twitch at each spike, a slow axon (SETi) with known summation properties at the muscle and at least one inhibitor axon, the common inhibitor (Hoyle 1955).

The flexor muscle has at least seven motoneurons – three fast, two intermediates and two slow, of which one fast is not previously mentioned (Hoyle & Burrows 1973*a*) – and at least

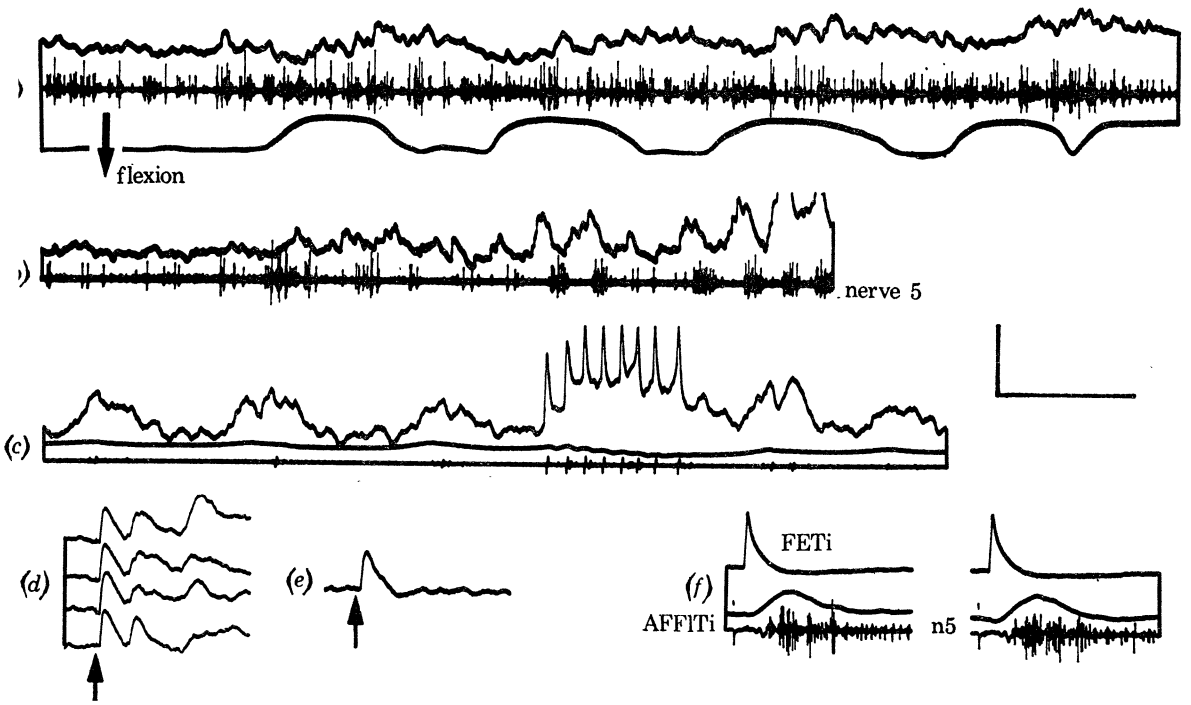

FIGURE 6. Properties of a fast flexor tibiae (AFFITi). (*a*) Passive movements of 10° of the tibial joint from the 90° position (flexion down) showing e.p.s.ps in response to extension but no clear correspondence with sensory impulses in nerve 5. (*b*) As in (*a*) during a spontaneous movement caused by other motoneurons. (*c*) Passive oscillation of the tibia causes excitation of AFFITi at extension (up on middle trace) and when an active flexion is aroused, the threshold for spike initiation is apparently lowered. The flexor impulses each cause a flexion twitch (middle trace) and a potential in the myogram (lower trace). (*d*) An antidromic impulse causes a follow-up excitation which is abolished when the leg is cut off (*e*). Four sweeps are shown in (*d*). (*f*) An FETi antidromic impulse (upper) causes a centrally mediated depolarization of the fast flexor (middle). The reflex sensory response to the extension twitch recorded still 1 cm from the ganglion occurs too late to cause the response. Calibration: vertical (*a* and *b*) 5 mV, (*d–f*) 10 mV; horizontal (*a–c*) 400 ms, (*d–f*) 160 ms.

two inhibitory neurons, not previously described (Burrows, unpublished). The flexor motoneurons differ in soma position, peripheral distribution and action, for reasons that are not understood.

Previous work on flexor and extensor tibiae motoneurons

When the locust is standing the simultaneous contraction of the flexor and extensor tibiae muscles is common; during slow walking they usually alternate, while in fast walking one or the other may have a background tone while bursts of impulses in the SETi or a fast flexor neuron controls the movement (Hoyle 1964; Usherwood & Runion 1970). Recordings from flexor motoneurons have shown that there is no direct coupling or central interaction between them, 'nor was coupling evident during a variety of spontaneously and reflexly evoked movements' (Hoyle & Burrows 1973*a*).

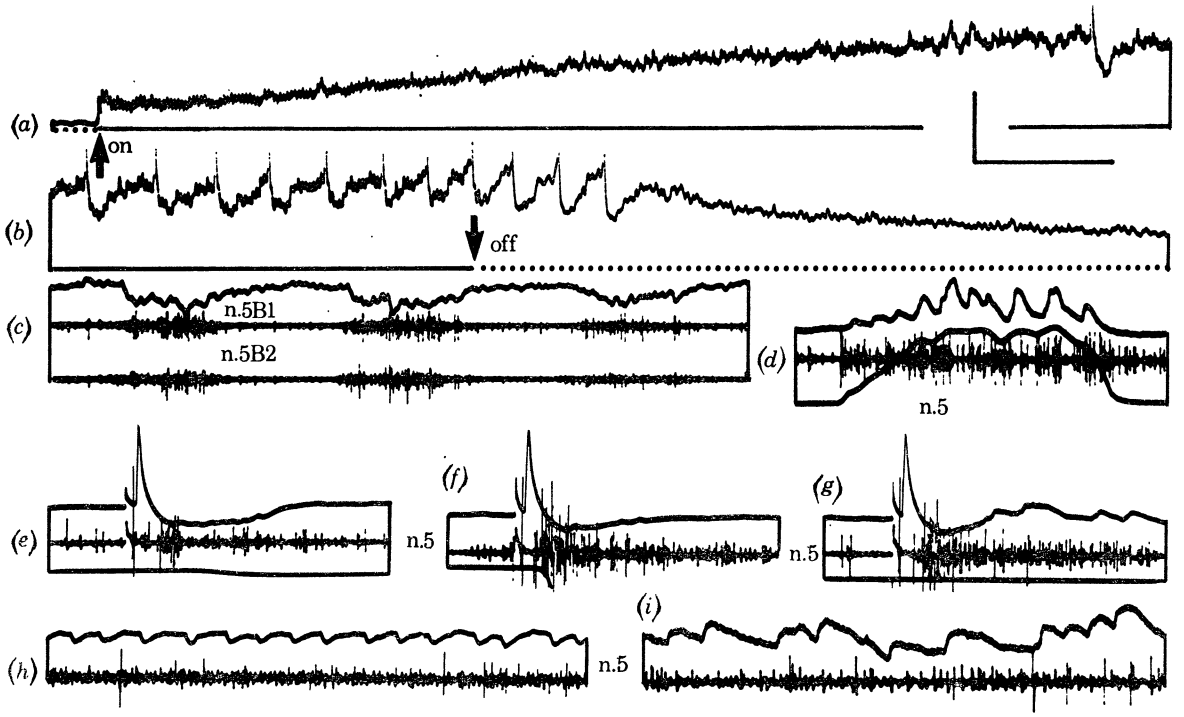

FIGURE 7. The fast extensor tibiae, FETi. (*a*) Slow depolarization to spike threshold upon stimulation of the ipsilateral thoracic cord at 70 Hz and the slow decline (*b*) after the termination of the stimulus (at arrow). (*c*) Inhibition on brushing tibial spines, with simultaneous records in n5B1 (trace 2) and n5B2 (trace 3) showing sensory impulses. (*d*) Forced flexion of the tibia (lower trace) causes e.p.s.ps (upper) that are not related to sensory impulses in nerve 5 (middle trace). (*e*) One stimulus to the FETi axon in the femur with the leg fully extended causes few sensory impulses in nerve 5, but i.p.s.ps follow the antidromic spike. (*f*) As in (*e*) but with the tibia at 90° and free to move; few p.s.ps follow the antidromic spike. (*g*) As in (*f*) with tibia fixed at 90°; e.p.s.ps now follow the antidromic spike. (*h*) i.p.s.ps in the FETi caused by full tibial extension are not related to tonic sensory impulses in nerve 5. (*i*) e.p.s.ps coming late upon extension of the tibia are unrelated to sensory impulses in nerve 5. Calibration: vertical (*a–d*, *h*, *i*) 5 mV, (*e–g*) 12 mV; horizontal (*a–d*) 400 ms, (*e–g*) 85 ms, (*h*, *i*) 100 ms.

During reflexly evoked responses (by bending the femoral–tibial joint) the slow extensor and the posterior slow flexor neurons show marked reciprocity, and some e.p.s.ps in one coincide with i.p.s.ps in the other, suggesting a common input with opposite effects. The reciprocity is not maintained by direct inhibitory feedback from one motoneuron upon the other, but by

common inputs. On the other hand, the slow extensor and an intermediate flexor of the anterior group of motoneurons were not reciprocally activated but simultaneous excitation was common (Hoyle & Burrows 1973*a*).

Although the muscles are antagonistic, a spike in the FETi motoneuron causes a depolarization in all flexor motoneurons. The latency ranges from 25 ms for the slow flexors (with a labile response) to 10–12 ms for intermediate flexors and only 3–4 ms (not labile) for the posterior group fast flexor (figures 6*f*, 20*c*, 31). A spike in the FETi or in the SETi briefly inhibits the other (figures 16*f*, 30*e*). To any strong stimulus flexor motoneurons generate impulses at the same time as the extensors, but the fast extensor, with its greater mechanical effect, overcomes the flexors. The present intention is to extend these findings of Hoyle & Burrows (1973*a*, *b*).

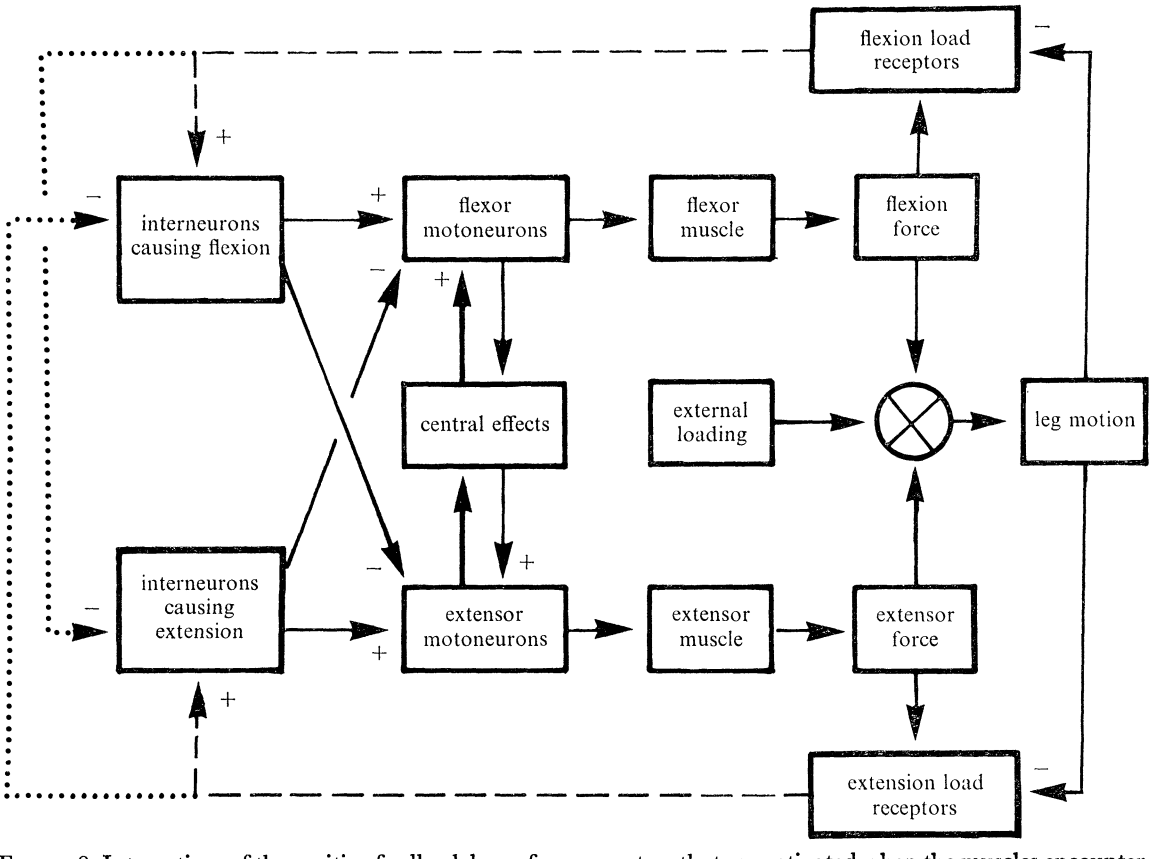

FIGURE 8. Interactions of the positive feedback loops from receptors that are activated when the muscles encounter an external load. These pathways should be added to those in figure 4.

Restraint of the joint

There are several ways of producing movement of a joint experimentally via its own muscles. (*a*) The axon terminals entering the muscles may be stimulated directly and the antidromic spikes recorded in the motoneurons to show which are excited (figure 7*e–g*). (*b*) The motoneurons can be excited by depolarizing current and the effects monitored by the myogram. (*c*) Movements that can be ambiguously called reflex, but which we term elicited responses, are brought about by touching the locust on the leg, head or abdomen.

When the joint is restrained so that the motor activity, however generated, fails to cause movement, there is a late excitation of the motoneuron, visible as a long-lasting depolarization

which usually causes additional spikes. This occurs in flexors (figures 6*d*, *e*) and particularly strongly in the fast extensor (figures 7*g*, 30*e*, *f*). Two mechanisms can be distinguished: (*a*) when the movement no longer occurs the motoneuron is not inhibited as it would be by the motion which it itself causes in the unrestrained leg; (*b*) a much stronger effect is caused by sense organs which are excited by loading of the skeletal elements, either in cuticle or apodemes, so that different sensory neurons are excited when the muscle meets an external resistance. The two effects together, recorded at the motoneuron, show that special sense organs with positive feedback to the motoneurons respond to the load (figure 8) as illustrated in records (figures 6*d*, 7*g*, 30*e*). All voluntary movement is made in continual interaction with the external mechanical reaction to the movements themselves. The system behaves as if stretch receptors lie upon the apodemes, because the muscles are excited by loading only when there is an active movement. Although few motoneurons are involved, a quantitative account of how antagonists interact during continuous activity becomes prohibitively complex when the leg is loaded.

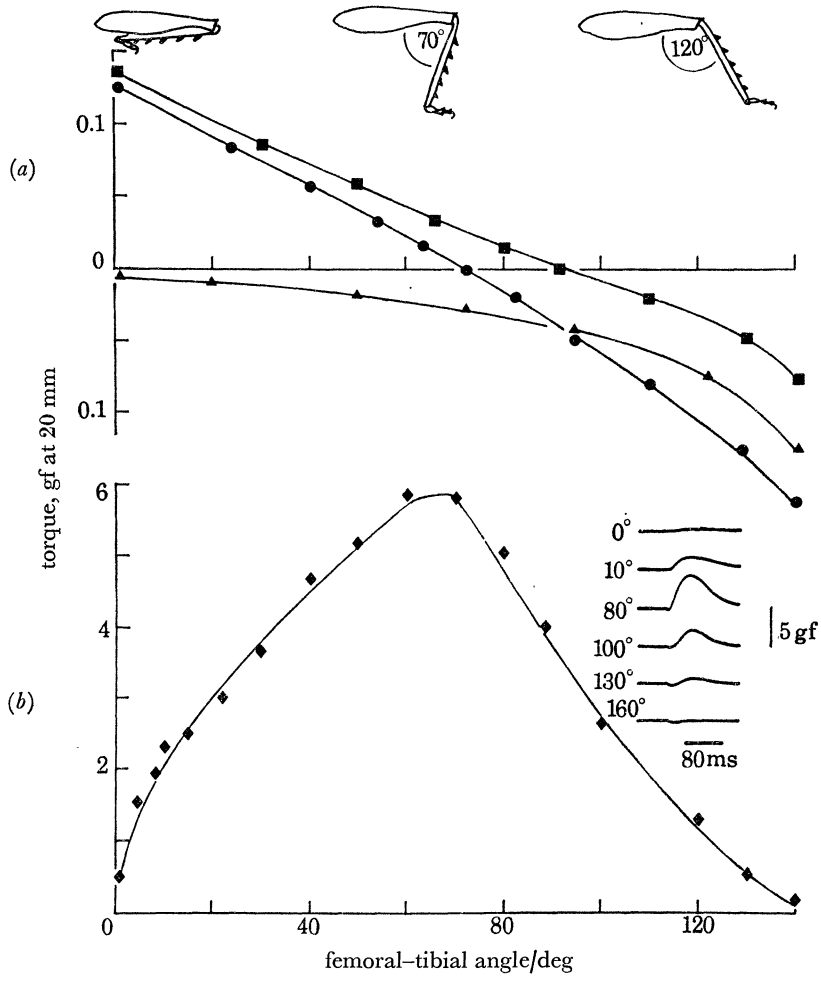

FIGURE 9. Torque measured at the distal end of the tibia about the femoral–tibial joints. (*a*) passive positions of the isolated fresh leg; ●, intact leg; ■, flexor tendon cut; ▲, extensor tendon cut. (*b*) torque generated by an active twitch of the extensor tibiae muscle caused by a single impulse in FETi motoneuron. The inset shows responses at representative parts of the curve when the flexor (downward response) is also excited by the same stimulus. Note the weak torque of the extensor at the extremes.

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Effects of tibial position on extension torque

Grasshoppers and locusts always bring the tarsus of the hind leg beneath the body and fully flex the tibia about the femur before they jump (Brown 1967). The point of thrust then lies beneath the centre of gravity and the extensor muscle is stretched. A third factor, however, which is mentioned by Brown (1967) but omitted from more detailed studies (for example, Usherwood & Runion 1970), is that when the tibia is fully flexed the torque exerted by the extensor muscle about the joint is very small and increases only as the joint begins to extend (figure 9*b*). The torque reaches a maximum at a femoral tibial angle of 60–80° and then declines so that near full extension (150°) the weaker flexor muscle can again exert more torque than the extensor. The mechanism lies partly in the structure of the joint, where the extensor apodeme is attached to a projection which moves over the hinge, so that the torque about the closed joint is very small, and although the muscle is then maximally stretched it operates with poor mechanical advantage. The length–tension properties of the muscles are also unknown and may be relevant; the flexor has long parallel fibres, the extensor fibres arranged in chevrons. Whatever the combination of mechanisms, in the fully flexed position a single FETi spike is ineffective in producing extension, so that during a burst of impulses in the FETi motoneuron, power is built up by the isometric tension of the extensor muscle before it is able to move the tibia. A fourth factor is a locking device on the flexor tendon (Heitler 1974). A rapid and powerful extension then occurs as a result of the progressive opening of the joint. If there is an external load, there is an additional neurally mediated feedback.

Measurements of the passive elasticity of the muscles show that the resulting torques at the joint are small but that the passive resistances to stretching the muscles are quantitatively greater than the reflex resistances caused by motion of the joint. With the extensor tendon cut, the torque measured at the distal end of the tibia is negligible when the joint is fully flexed, and increases smoothly to 0.1–0.15 gf at 20 mm, as the leg is extended to maximum (figure 9*a*). With the flexor tendon cut the joint assumes a resting angle of about 90°. A torque of 0.120 gf at 20 mm is then required at the distal end of the tibia to flex the joint fully, and, in the opposite direction, a torque of 0.04–0.07 gf at 20 mm to open it wide. With both muscles intact a torque of about 0.020 gf at 20 mm is required to deflect the joint each 10° away from its resting angle of near 70°. Torques of this magnitude were common during the central recordings of resistance reflexes and can all be attributed to passive resistance of the muscles and joint. The resistance forces are little changed by section of the nerve to the leg.

Inhibitory neurons to the femur

Two previously undescribed inhibitory neurons to muscles of the leg are involved in leg reflexes. The posterior inhibitor (PI), with its soma between the midline and the root of nerve 5, and the anterior inhibitor (AI) with its soma anterior to CI (Burrows, unpublished) each have an axon in nerve 5 to the flexor tibiae muscle and possibly other unknown destinations. Passive extension or flexion of the tibia excites the PI and the AI but in general they behave like inhibitors of flexion (figures 10, 11). There are no centrally mediated effects of motor impulses upon them, but both PI and AI are usually excited during any elicited response or voluntary movement. The posterior inhibitor shows one of the few contralateral effects of passive movement, being excited by motion of the contralateral tibia either way (figure 26). The active extension motion of the ipsilateral tibia, caused by stimulation of the FETi in the femur, causes

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an inhibition of the PI with a 50 ms latency in some animals. At the same time the flexor motoneurons are also excited reflexly by the motion of extension. In other locusts active extension causes excitation of AI, and also of PI. There is, however, in both AI and PI (figure 10*b*) a maintained discharge of e.p.s.ps as a measure of leg extension, one of the few tonic effects. A full description of these inhibitory neurons is contemplated. They behave like slow motoneurons in excitability and the ease with which they generate impulses, although possibly there exist phasically acting inhibitors which have been overlooked.

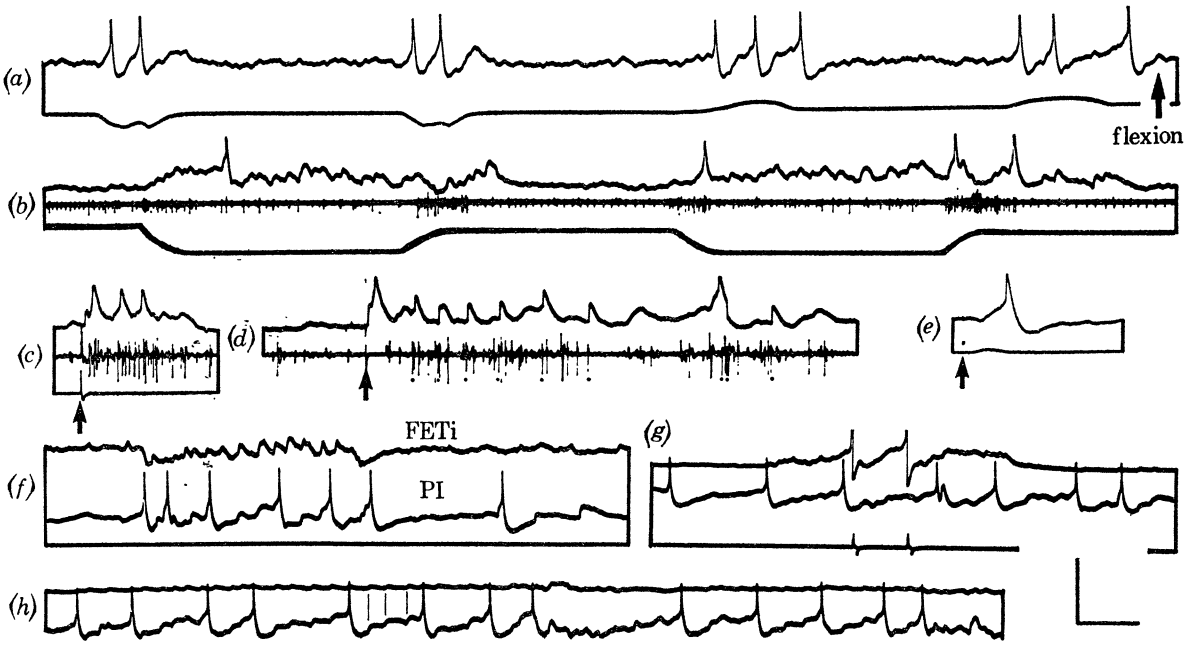

FIGURE 10. Properties of the posterior inhibitor, PI. (*a*) Flexion of the tibia indicated by upward arrow (on 2nd trace) or extension both excite the PI. (*b*) As in (*a*) but the PI is now hyperpolarized to reduce the number of spikes and reveal e.p.s.ps. Sensory impulses in nerve 5 (middle trace) do not correspond to PI inputs. (*c*) An antidromic impulse in FETi (recorded in the muscle on the lower trace) causes the tibia to extend and excites the PI. (*d*) Sometimes the reflex response to an FETi antidromic spike generates antidromic spikes in the PI; the extracellular PI spikes from nerve 5 are identified by dots (middle trace). (*e*) One stimulus to the flexor in the femur causes an active tibial flexion (lower trace) and a reflex excitation of the PI which disappears when tibial movement is prevented. (*f*) Passive extension of the tibia causes i.p.s.ps followed by e.p.s.ps in the FETi (upper trace) with some common inputs with the PI (lower). (*g*) Passive flexion of the tibia excites both neurons. (*h*) The e.p.s.ps evoked upon extension are common in the two neurons (vertical lines give an indication of some of the coincidences). Calibration: vertical (*a–e*) 10 mV, (*f, g*) 4 mV and 12 mV, (*h*) 25 mV and 15 mV; horizontal (*a, b*) 200 ms, (*c, e*) 80 ms, (*d, f–h*) 100 ms.

A common inhibitor (CI) neuron, with its somata near the centre of the ventral surface of the ganglion, has an axon that branches to nerves 3, 4 and 5 and thence to the extensor tibiae, anterior coxal adductor, and other unknown muscles of one side of the segment. The following are conclusions from a study on the CI neuron (Burrows 1973*a*). There is no direct electrotonic or synaptically mediated central pathway between it and motoneurons innervating the same muscles. The CI neuron is strongly excited to produce a burst of spikes by forced depression of the tarsus of its own leg, and more weakly by depression of the mesothoracic ipsilateral or the metathoracic contralateral leg. Inputs which excite or inhibit the anterior adductor coxa motoneuron, which raises the leg, have a reciprocal effect on the CI. One of these inputs is known now to be from visual descending interneurons (Burrows & Rowell 1973). Although each

joint has a resistance reflex involving antagonistic muscles, the CI does not act reciprocally with one motoneuron and synergistically with another, and no clear function has been found.

The following additional observations have been made. In active animals the CI neurons are subjected to a barrage of i.p.s.ps, abolishing any tonic background spike discharge there might normally be. There are no unitary p.s.ps in the CI neuron that can be identified with any known sensory neuron, interneuron (except descending movement detectors) or motoneuron (figure 11*d*, *e*) when their spikes are recorded in the thoracic cords or from the leg nerves. This includes ascending auditory interneurons and chordotonal sensory neurons. No common input, indicating a common controlling interneuron, has yet been found between the CI neuron and any of the motoneurons to the leg studied here.

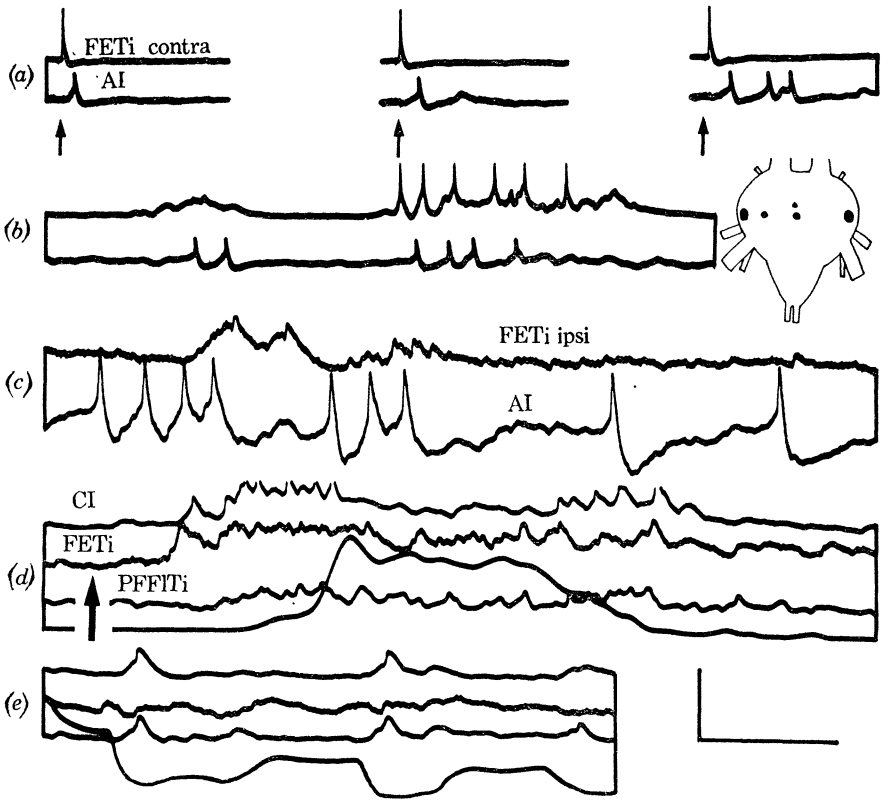

FIGURE 11. Fast extensor tibiae, FETi, with the inhibitor neurons AI and CI. (*a*) An antidromic spike in the contralateral FETi (at arrow in upper trace) causes an excitation after a delay of 40 ms of the ipsilateral AI. (*b*) A forced flexion of the contralateral leg, to which the contralateral FETi (upper) responds with similar but not common inputs to the ipsilateral AI. (*c*) FETi (upper) with common inputs to the AI of the same side during passive extension of the tibia. (*d*) CI (upper) FETi (middle) and PFFlTi (third trace) during a spontaneous active flexion (indicated by upward arrow on the fourth trace). (*e*) As in (*d*); during passive motion of the tibia the CI and the flexor often spike at the same time. Calibration: vertical (*a*, *b*) 12 mV, (*c*) trace 1, 2.5 mV; trace 2, 5 mV; (*d*, *e*) trace 1, 10 mV; trace 2, 5 mV; trace 3, 15 mV; horizontal (*a*, *b*) 400 ms, (*c*–*e*) 200 ms.

We consider that the CI neuron, cutting as it does across the boundaries of muscle groups and participating in a definite but undefined way in several movements but not in others (Usherwood & Runion 1970) is an important indicator of the way in which interneurons may function centrally in the organization of behaviour at the segmental level. The CI neuron is active during flight, presumably then contributing to the flight posture of the legs, but does not participate notably in the respiratory rhythm, which affects only body muscles. The CI

neuron is perhaps a central inhibitory interneuron which has spread peripherally as a way of extending its regionalized influence. This view of the CI neuron not only explains all that is known of it, but assists in designing experiments on it and in preparing an appropriate outlook for work on central interneurons.

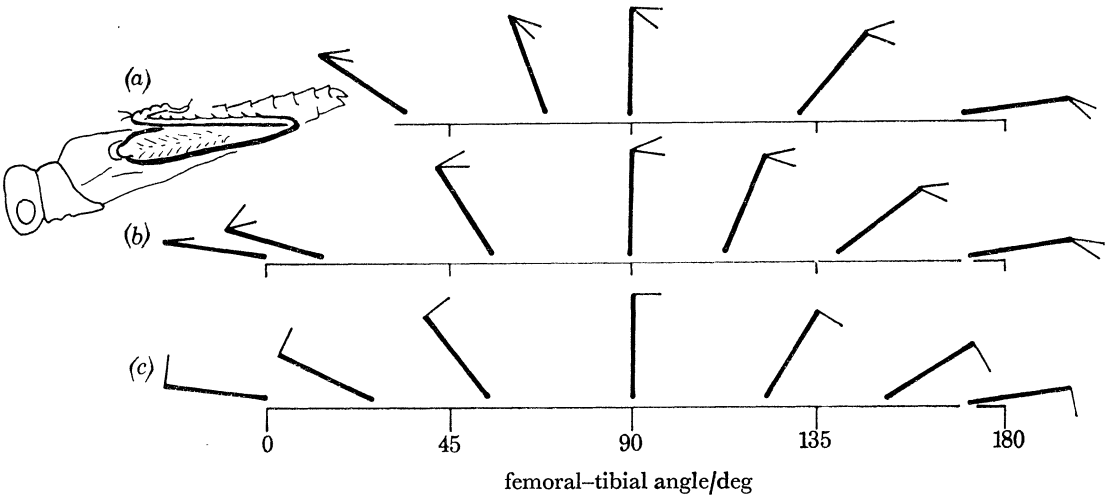

FIGURE 12. The range of angles taken up by the tarsus as a function of the angle at which the tibia is passively set. The femur is horizontal throughout, as in (*a*). (*a*) Locust intact. (*b*) With the connectives on either side of the metathoracic ganglion cut. (*c*) After section of the leg nerves.

Interaction between two joints of the leg

We ourselves find it natural to point the foot when the knee is straight; the same is true for the metathoracic leg of the locust. When the locust is resting on a flat surface, there is a relation between the angle at the femoral–tibial joint and that at the tibial–tarsal joint. That this relation is not entirely due to the passive resting of the tarsus on the ground is demonstrated by forcibly bending the tibia when the locust is held in the air. As the femoral–tibial joint is bent passively, in either direction within the range 0–170°, the tibial–tarsal angle changes appropriately so that the tarsus maintains a constant attitude relative to the body. The experimentally measured angles, taken from any one locust, show that there is a tonic postural reflex (figure 12*a*), although other factors cause large differences between animals. The interaction between the two joints is unchanged when the connectives are cut anterior to the mesothoracic and posterior to the metathoracic ganglia (figure 12*b*). To show that the effect is not a mechanical consequence of tendons, such as that of the retractor unguis muscle pulling between the femur and the tarsus, the same measurements are made finally with the leg nerve cut, which removes the effect on the tarsus (figure 12*c*).

Recordings from tarsal motoneurons show that extension of the tibia causes indirectly a phasic and tonic excitation of the fast and slow depressor tarsus motoneurons (FDTa, and SDTa), with flexion causing weaker effects in the opposite direction (figures 5, 13, 27*c*). Oscillation of the femoral–tibial joint causes oscillations of the potentials of the tarsal motoneurons in the appropriate directions. When flexors or extensors of the tibia are recorded as well, the tarsal motoneurons fail to follow oscillations faster than about 5 Hz which fast flexor tibiae motoneurons follow easily (figure 14*g*). When the extensor or flexor muscles of the tibia are excited directly in the femur the active movement of the tibia causes a reflex effect in the appropriate direction upon the tarsal motoneurons with a latency of about 30 ms (figures 13*f*,

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14*c–e*). This can be distinguished from a centrally mediated response such as that seen upon the flexor tibiae motoneurons following a spike in the FETi motoneuron, by its failure to appear when movement of the tibia is prevented (figure 13*f*). The reflex effects of imposed tibial movement are smooth polarizations caused by many small p.s.ps in tarsal motoneurons. There are also a few large p.s.ps, especially large i.p.s.ps, which have a tonic effect on the slow levator tarsus when the tibia is extended (figures 28, 29). Of the various reflex effects indicated in figure 15, the one which shows it most strongly is the depolarization of the tarsal depressor motoneurons upon sudden extension of the tibia, such as that caused by a single impulse in the FETi motoneuron. The inter-joint relation is almost one way only. Tarsal position or tarsal motion has no effect upon tibial motoneurons except that the slowest flexor tibiae is sometimes inhibited by tarsal levation.

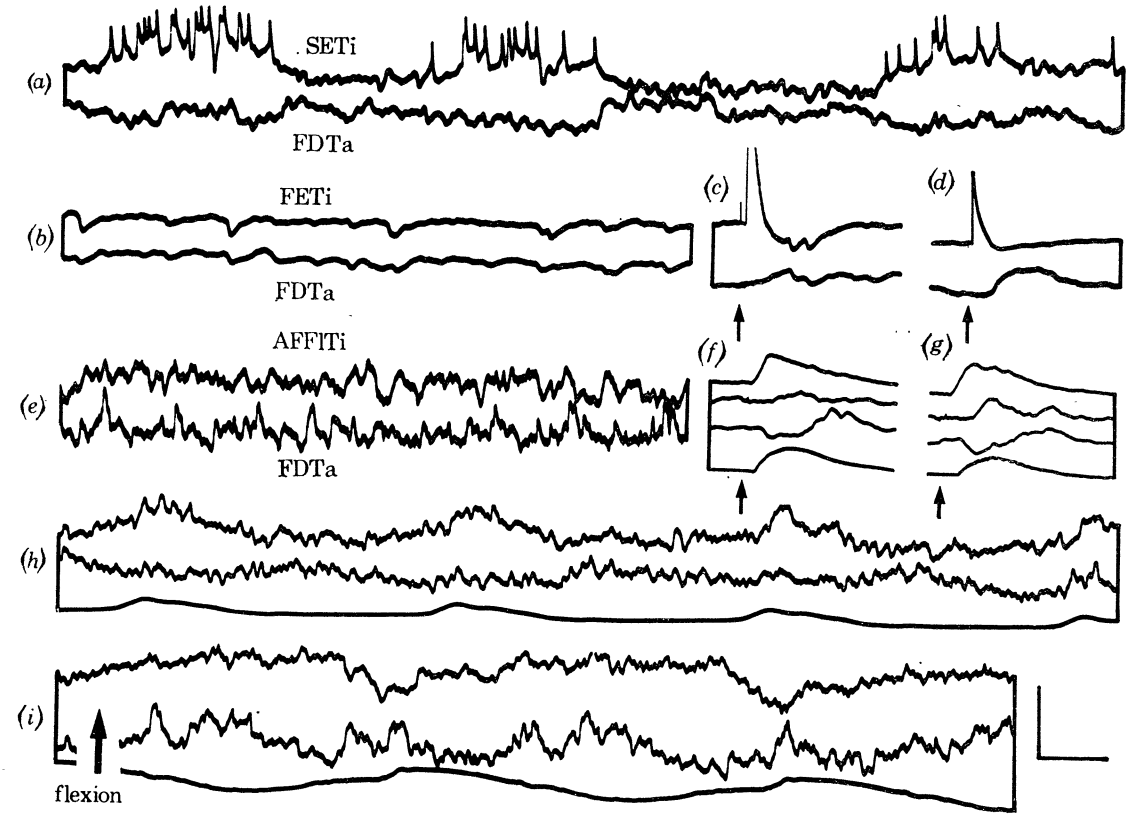

FIGURE 13. Relations between the fast depressor tarsus (FDTa) and motoneurons to the femur. (*a*) Imposed movements of the tibia cause responses of SETi (upper) and FDTa in opposite directions but without common inputs. SETi is excited upon flexion. (*b*) Common i.p.s.ps between FETi (upper) and FDTa upon maintained full extension of the tibia. (*c*) Extension motion caused by an antidromic FETi impulse at full extension of the tibia evokes common i.p.s.ps in the two motoneurons. (*d*) As in (*c*) but with the femoral–tibial angle at 90°; there is now a reflex excitation of the FDTa. (*e*) AFFlTi (lower) and FDTa showing no visible common inputs in background activity. (*f*) With tibial motion restrained (recorded as a flexion force on trace 4), an antidromic FETi spike (at arrow) causes a central excitation of AFFlTi (trace 1), no response in FDTa (trace 2) and an initial inhibition of SETi (trace 3). (*g*) As in (*f*) but with tibia allowed to extend before hitting the stiff transducer, showing an additional reflex effect upon the flexor and now a depolarization of the depressor. (*h*) As in (*e*) with imposed movements of the *tarsus* (levation upwards). (*i*) As in (*e*) with imposed movements of the *tibia* (flexion is upwards arrow).

Calibration: vertical (*a*) 6 mV, (*b*, *c*) trace 1, 5 mV; trace 2, 10 mV; (*d*) 6 mV and 12 mV, (*e*) 2.5 mV, (*f*, *g*) trace 1, 25 mV; trace 2, 15 mV, trace 3, 12 mV; (*h*, *i*) 5 mV; horizontal (*a*, *e*, *h*, *i*) 200 ms, (*b*, *c*, *d*, *f*, *g*) 80 ms. Torque in (*h*, *i*) 50 mgf at 20 mm.

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Tests for common inputs from one interneuron to motoneurons which operate muscles at different joints, by simultaneous recording and examination of p.s.ps, reveals only one example (figures 13, 14). Upon a maximal active extension of the tibia, there are common i.p.s.ps between FETi and FDTa motoneurons (figure 13*b*, *c*). The failure to find other examples over the more widely used range of femoral–tibial angles strengthens the inference that the interaction between the two joints is a purely reflex one from femoral–tibial joint receptors indirectly upon tarsal motoneurons (figure 15).

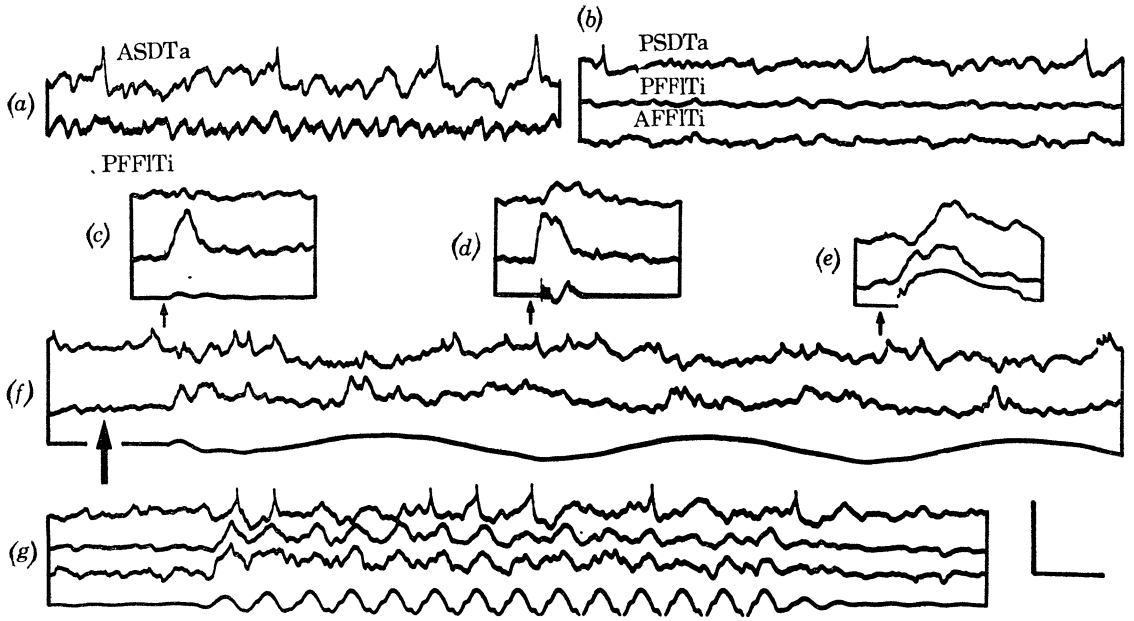

FIGURE 14. Lack of common inputs between tarsal depressors and tibial flexors. (*a*) Anterior slow depressor tarsus, ASDTa (upper trace) and the posterior fast flexor (PFFlTi, lower trace) with no common inputs in background activity. (*b*) Posterior slow depressor, PSDTa (upper) has no inputs in common with two flexors, posterior fast (middle) and anterior fast (AFFlTi, lower) in background activity. (*c*) With movement almost prevented, an antidromic FETi impulse (arrow) has no effect on ASDTa (upper) and only the central effect on PFFlTi (middle). (*d*) With the tibia free to move and strike the transducer (lower), an antidromic FETi spike (arrow) causes a reflex response in ASDTa (upper). (*e*) A faster sweep to show the delay between the central effect on PFFlTi (middle) and the reflex effect on ASDTa (upper). (*f*) The posterior slow depressor tarsus (PSDTa) (upper trace) and PFFlTi; flexion of the tibia is upwards (arrow) on the lower trace. (*g*) As in (*b*). The PSDTa fails to follow tibial oscillation at 8 Hz but the flexors do.

Calibration: vertical (*a*) trace 1, 12 mV; trace 2, 4 mV; (*b*, *g*) 15 mV, 12 mV and 5 mV, (*c–e*) 15 mV and 12 mV, (*f*) 20 mV and 10 mV; horizontal (*a–d*, *f*, *g*) 200 ms, (*e*) 80 ms. Torque in (*f*) is 50 mgf at 20 mm.

In an active retraction of the leg in a free locust the tarsus is pressed against the tibia before the tibia is pressed against the femur. This sequence in the response still occurs when the four connectives of the ganglion are cut. Again, when the base of the abdomen is touched the leg is drawn in to kick away the offending object, using the spines on the end of the tibia, and the tarsus is not pressed against the tibia at all. These details show that the tarsus is not necessarily linked behaviourally with the femoral–tibial joint. The reflexes are persistent during voluntary movements but mechanically too weak to resist them.

Common inputs and individual control of motoneurons

By recording from two or more motoneurons simultaneously at an amplification that reveals potentials of 0.25 mV, the incidence of e.p.s.ps and i.p.s.ps in the neurons can be compared.

The intention is to reveal as much as possible of the organization of inputs upon the motoneurons at a time when we lack techniques to record from and identify the premotor interneurons. A limitation is that only the most obvious p.s.ps can be compared by eye in the neurons, and they must coincide many times. An assumption must also be made that depolarizing potentials are e.p.s.ps and hyperpolarizing potentials i.p.s.ps. Sometimes the reversal potential can be determined but usually current injected into the soma is unable to influence the conductance

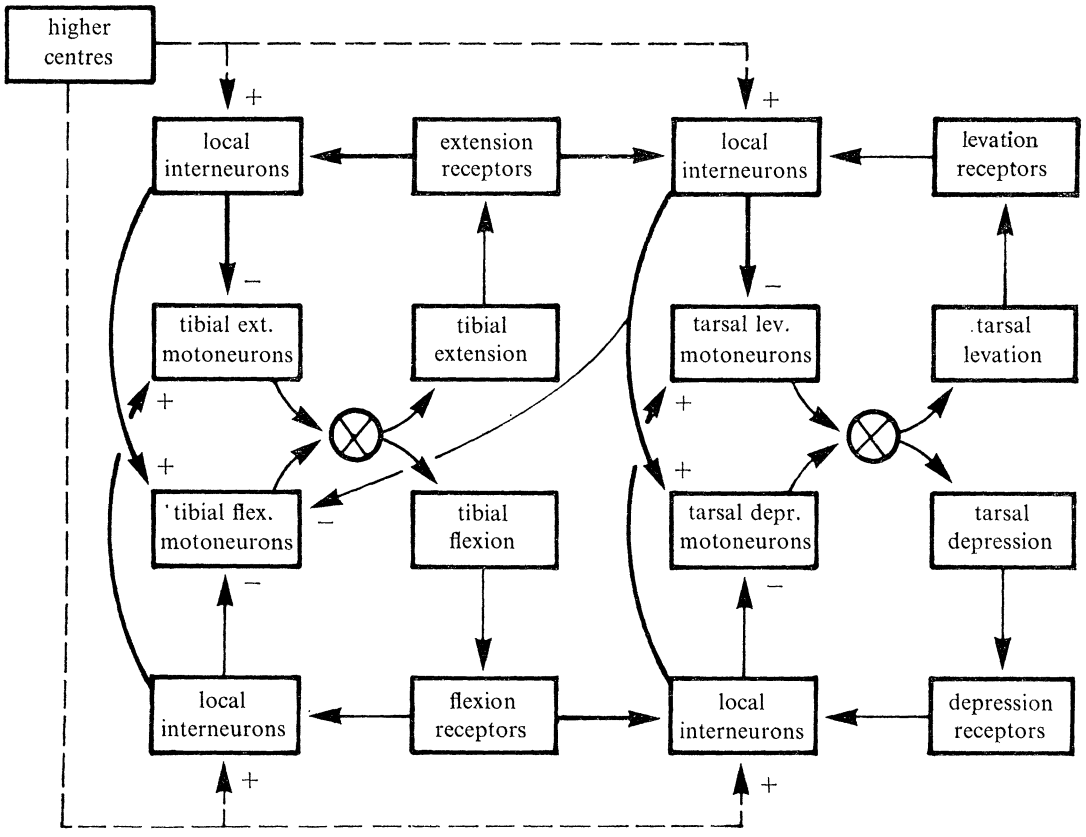

FIGURE 15. The interactions between femoral–tibial reflexes (left) and tibial–tarsal reflexes (right). Stronger interactions are shown as thicker lines. Each box shown indicates several or many components in parallel.

changes at the more distant synapses. Numerous combinations of motoneurons are needed to test for common inputs, and only some have so far been obtained. To show that an input common to two neurons is also common to a third one, it is necessary to record from all three together, and the labour soon becomes prohibitive. Comparable results have been obtained by simultaneous recordings from several neurons at once in *Aplysia*, which has the advantage that the presynaptic neurons can be found, but the disadvantage that the functional significance is unknown (Kandel, Frazier, Waziri & Coggeshall 1967; Strumwasser 1967).

Common inputs to synergistic muscles

The two motoneurons to the extensor tibiae muscle, the FETi and the SETi, have many e.p.s.ps in common in spontaneous background activity, in resistance reflexes, and in elicited responses of all kinds (figures 16, 17, 30*f*). One of the e.p.s.ps common to the FETi and SETi is synchronous with an i.p.s.p in the posterior fast flexor tibiae (PFFlTi) (figure 17*d*) and another

is synchronous with an i.p.s.p. in the posterior intermediate flexor (PIFlTi) and possibly, therefore, with i.p.s.ps in other flexors. These two interneurons therefore have common inputs to at least three motoneurons, with similar effects on synergists and the opposite effect on antagonists. There are also common inputs to FETi and SETi which do not act visibly on the flexors. The most obvious is the common e.p.s.p. elicited in the FETi and SETi in the second part of the effect of extension of the tibia after the initial inhibition (figure 16*b*, *c*). This e.p.s.p. is also common upon the anterior and the posterior inhibitors (AI and PI) (figures 10*h*, 11*c*).

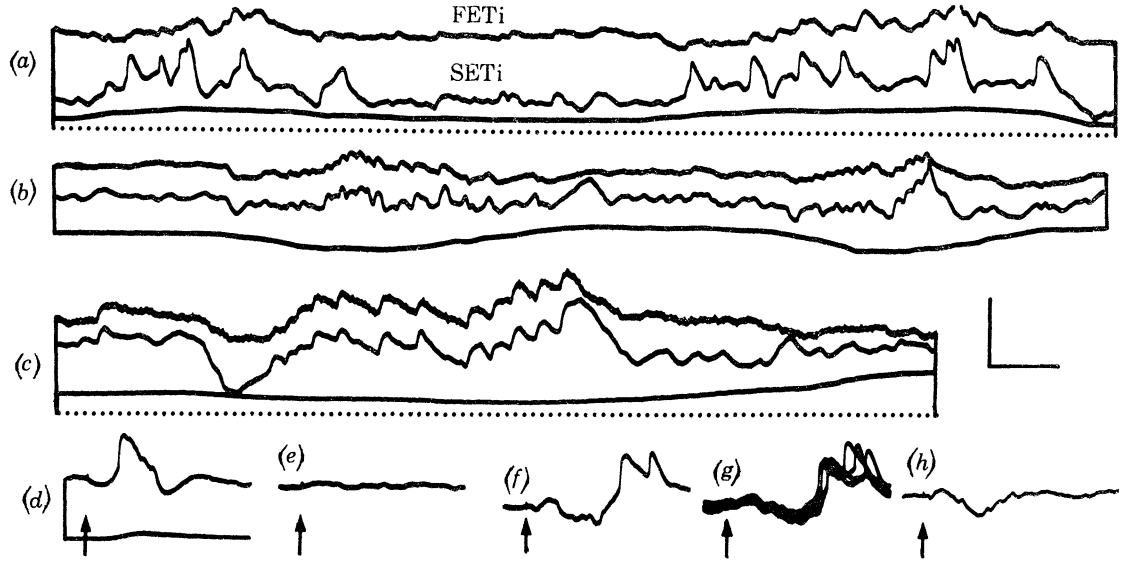

FIGURE 16. Fast and slow extensor tibiae, FETi and SETi, showing common inputs in some responses. (*a*) E.p.s.ps in response to passive flexion of the tibia (up on trace 3). (*b*) I.p.s.ps followed by e.p.s.ps which are common to both neurons in response to passive extension (down on trace 3). (*c*) As in (*b*) but at different relative amplification. (*d*) Depolarization of the SETi to one direct stimulus (arrow) of the flexor tibiae muscle when the tibia is allowed to move. (*e*) The depolarization is abolished when the tibia is held still. (*f*, *g*) SETi response to an antidromic FETi spike (at arrow) showing centrally mediated inhibition and delayed excitation caused reflexly. Multiple sweeps show the consistency of the response in (*g*). (*h*) Only the brief central inhibition caused by the antidromic FETi spike remains when the leg is amputated. Calibration: vertical (*a*, *b*) trace 1, 5 mV; trace 2, 10 mV; (*c*) trace 1, 2 mV; trace 2, 5 mV; (*d–h*) 5 mV; horizontal (*a*, *b*) 100 ms, (*c*) 50 ms, (*d–g*) 80 ms, (*h*) 90 ms. Torque in (*a–c*) is 50 mgf at 20 mm.

This pattern of inputs makes functional sense because the PI and AI supply the flexor tibiae muscle and therefore they ensure that the antagonistic muscle is inhibited peripherally when the extensor is excited. There is no functional explanation of why these e.p.s.ps are elicited by extension. Another example is an i.p.s.p. in the FETi in common with an e.p.s.p. in the PI, which is also unexplained in terms of normal movements (figure 10*f*).

Tibial flexor motoneurons, recorded in pairs, show different proportions of inputs in common. The posterior (PFFlTi), the anterior (AFFlTi) and the lateral (LFFlTi) flexor tibiae motoneurons have many of their larger synaptic inputs in common (figures 18, 19). Records from these three neurons during resistance reflexes and elicited responses are, except for their spikes within a burst, almost identical (figures 18–20). One of the e.p.s.ps common to these three neurons is also common to the posterior slow flexor motoneuron (PSFlTi) (figure 21*a*, *b*). This input is caused by intraganglionic connexions because total isolation of the meso- and metathoracic ganglia from other parts of the nervous system and from the periphery has no effect (figure 19*b*).

Instances can be found where inputs are common in one response but independent in another, and occasional gating of a p.s.p. is found (figure 20*a*, *b*). The anterior intermediate flexor (AIFlTi) and the posterior fast flexor (PFFlTi) have no p.s.ps visibly in common, although the general form of their responses are similar (figure 22). With seven motoneurons innervating the one flexor muscle the combinations which must be examined for common inputs are numerous so that our instances are only an indication of the total. The examples described reveal that the inputs to fast and slow motoneurons can be not only similar, but have much in common, p.s.p. by p.s.p.

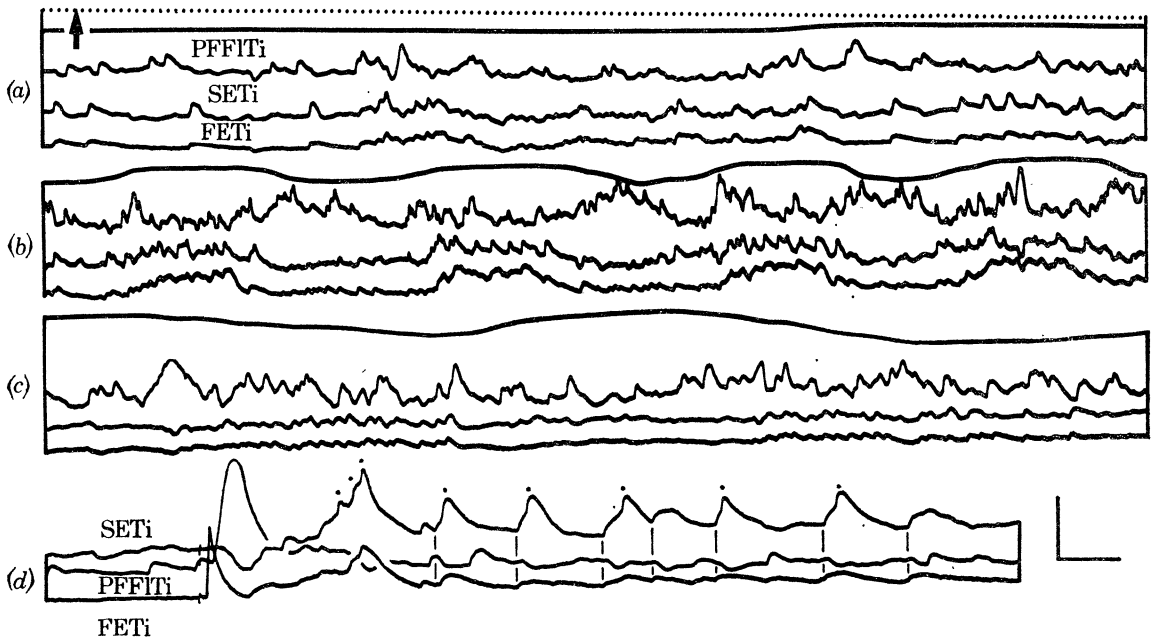

FIGURE 17. Reciprocal inputs to a flexor, PFFlTi (upper trace except in (*d*), where it is the middle one) and the two extensors, SETi (middle trace except in (*d*), where it is the upper one) and FETi (lower trace). (*a*) Background activity. (*b*, *c*) Response to passive flexion (up) and extension of the tibia. (*d*) Response to one direct stimulus to the extensor muscle which evokes an antidromic spike in the FETi. This causes a centrally mediated inhibition of the SETi and an excitation of the PFFlTi. The tibia then attempts to extend against an applied load causing a train of e.p.s.ps leading to spikes in the SETi, e.p.s.ps in the FETi and i.p.s.ps in the PFFlTi. These p.s.ps occur at the same time (indicated by vertical lines) in each motoneuron indicating a common source. Calibration: vertical (*a*, *b*) traces 1 and 3, 10 mV; trace 2, 15 mV; (*c*) trace 1, 5 mV; traces 2 and 3, 10 mV; (*d*) all traces 12 mV; horizontal (*a*, *c*) 100 ms, (*b*) 200 ms, (*d*) 50 ms. Torque in (*a*, *b*) is 50 mgf at 20 mm.

The difference between the impulse patterns of fast and slow motoneurons is inferred to arise from the lower excitability of the fast motoneuron as well as from possible additional inputs from phasic interneurons. Fast motoneurons are excited mainly by sudden strong stimuli whereas slow motoneurons are excited or inhibited best by maintained stimuli. We found exactly this phenomenon in the crab eyecup, where slow optomotor stimuli excite slow motoneurons tonically and fast stimuli fast motoneurons phasically (Burrows & Horridge 1968*a*). This difference, found in many arthropod responses, has been attributed to the effect of the larger size of the faster neurons upon their time constant and excitability (Davis 1971). The finding that one interneuron causes a common p.s.p. in the PSFlTi and LFFlTi which reaches the threshold for impulse initiation in the former but not in the latter (figure 21*a*) shows that the differences in impulse patterns arise from differences between the motoneuron

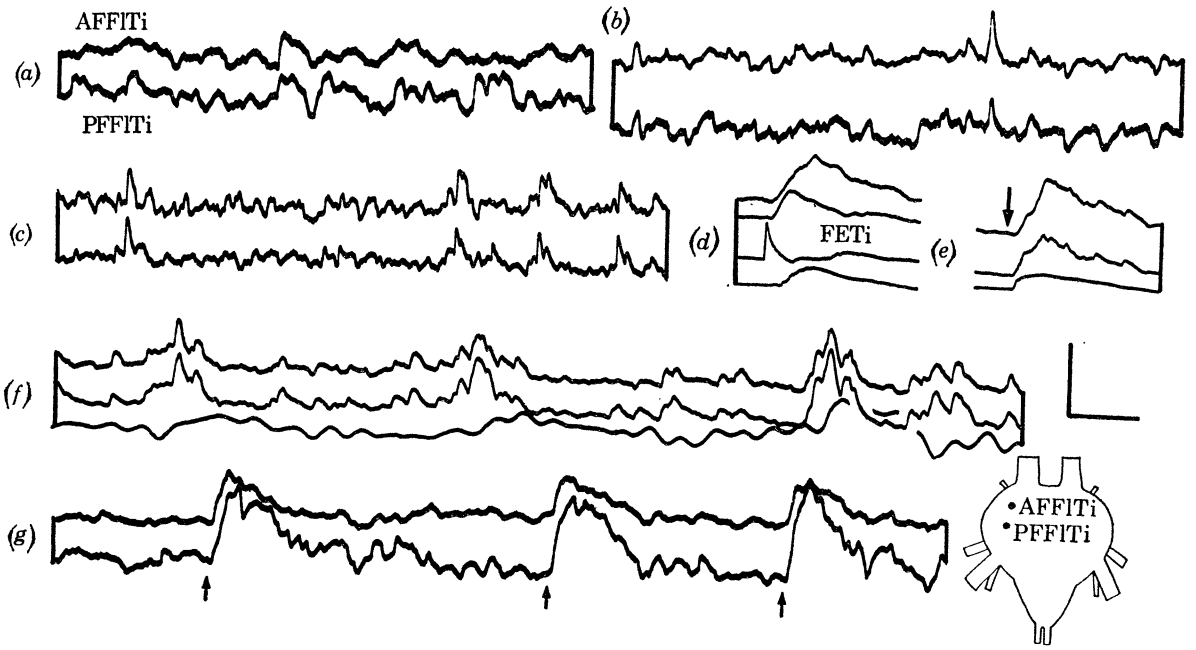

FIGURE 18. Anterior fast flexor tibiae AFFITi and posterior fast flexor tibiae PFFITi, showing common inputs. (*a–c*) Spontaneous background activity in these pairs of neurons in three different animals. (*d*, *e*) Central plus reflex effects of an FETi impulse, which is also recorded in (*d*) on the third trace showing common inputs in the centrally mediated effect and in the late reflex effects, especially in (*e*). (*f*) Identical inputs in elicited responses; flexion of the tibia is upwards on the third trace. (*g*) Reflex effects of imposed flexion and extension (arrows indicate the start of the extension). Calibration: vertical (*a*) 2.5 mV, (*b*) 2.5 mV and 5 mV, (*c*) 3 mV and 5 mV, (*d*) traces 1 and 2, 12.5 mV; trace 3, 25 mV; (*e*, *f*) 16 mV, (*g*) 5 mV; horizontal (*a*, *b*, *g*) 100 ms, (*c*, *f*) 200 ms, (*d*, *e*) 80 ms.

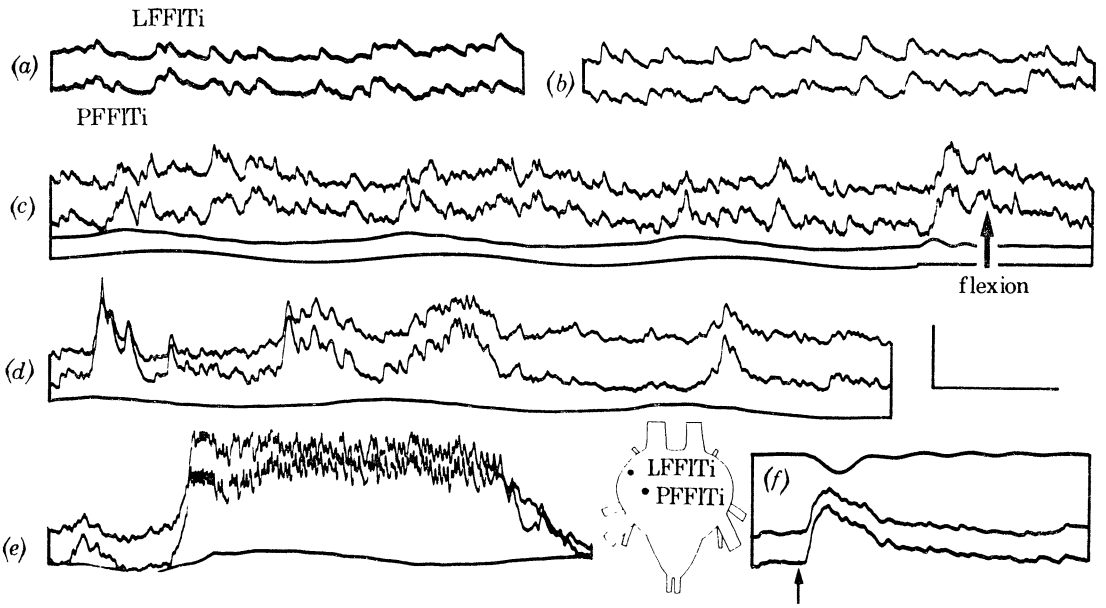

FIGURE 19. Two flexors LFFITi (upper) and PFFITi (lower) with few inputs not in common. (*a*) Background activity. (*b*) Background activity in a different locust after total isolation of the ganglion. (*c*) During passive oscillation of the tibia (applied movement signal on trace 4, tibial reaction in trace 3 where up (arrow) is flexion) the inputs to the two motoneurons are the same. (*d*) Elicited responses during oscillation of the tibia in which the inputs are again common to both neurons. (*e*) A flexion to touch of the head with spikes that are not synchronous in the two neurons. (*f*) Mainly central effect of an antidromic FETi impulse (at arrow), with the extension movement on the top trace and common p.s.ps. Calibration: vertical (*a*, *b*) 8 mV, (*c*) 5 mV, (*d–f*) 12 mV; horizontal (*a*, *b*, *f*) 200 ms, (*c–e*) 400 ms. Torque is 50 mgf at 20 mm.

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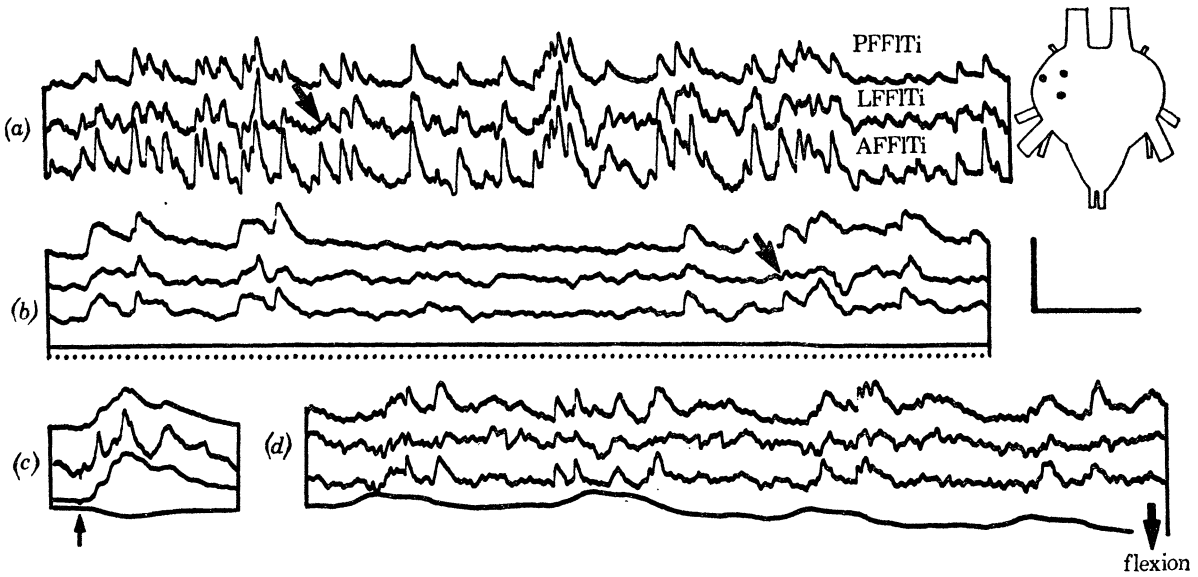

FIGURE 20. Three flexors with almost identical synaptic inputs; PFFITi (upper), LFFITi (middle) and AFFITi (lower) simultaneously recorded. (*a*, *b*) Background activity at high amplification. The arrows show where an e.p.s.p. is gated or abnormally small in one flexor. This e.p.s.p. is one which can be repeatedly recognized in all three motoneurons with long recordings, so that the gating is probably not an artefact caused by another interneuron with no input to one of the three motoneurons. (*c*) Central, followed by reflex effects, of an FETi spike induced at the arrow, when the tibia is allowed to move freely. (*d*) Imposed movements of the tibia cause many additional but mostly common inputs (flexion downwards on trace 4).

Calibration: (*a*) trace 1, 10 mV, trace 2, 5 mV, trace 3, 1.5 mV; (*b*, *d*) traces 1 and 3, 20 mV, trace 2, 8 mV; (*c*) trace 1, 25 mV, trace 2, 5 mV, trace 3, 10 mV; horizontal (*a*, *d*) 300 ms, (*b*) 150 ms, (*c*) 120 ms. Torque in (*d*) is 50 mgf at 20 mm.

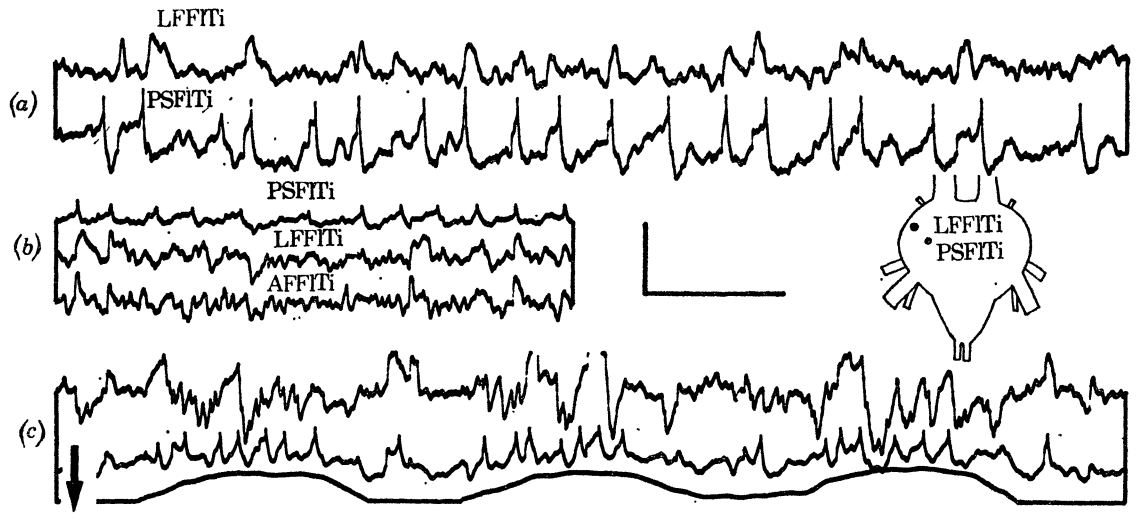

FIGURE 21. Slow (PSFITi) and fast (LFFITi) flexor tibiae motoneuron with common inputs. (*a*) Background activity in LFFITi (upper) and PSFITi in which the large e.p.s.p. is common to both; in the slow it evokes a spike. (*b*) Background activity showing much in common between the LFFITi (middle) and AFFITi (lower) but only one e.p.s.p. in common with PSFITi (upper). (*c*) As in (*a*). Passive extension of the tibia (flexion downwards, arrow) evokes spikes in the slow but not the fast flexor. Calibration: vertical (*a*) 5 mV; (*b*) trace 1, 10 mV, traces 2 and 3, 6 mV; (*c*) 7 and 15 mV; horizontal 400 ms. Torque in (*c*) is 50 mgf at 20 mm.

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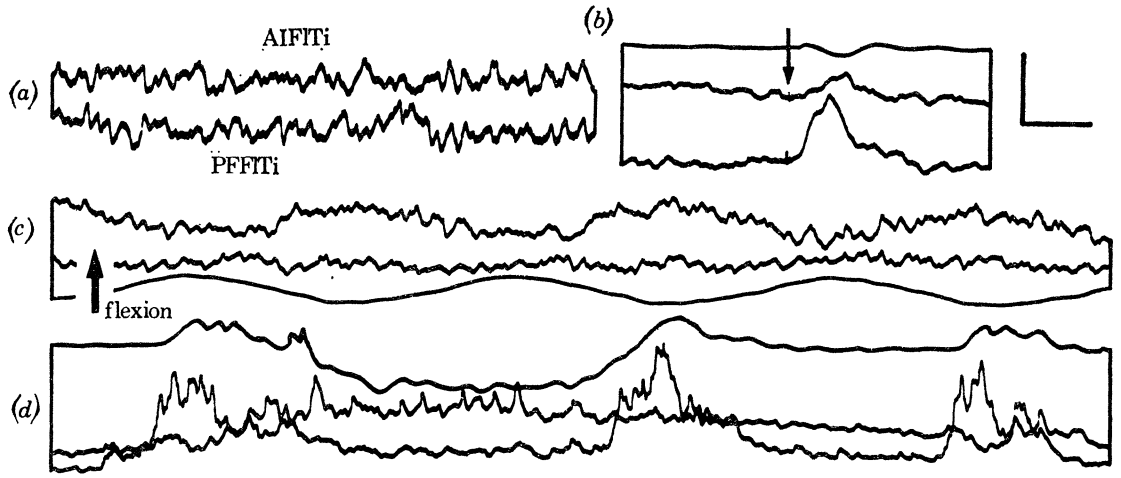

FIGURE 22. Two flexors, AIFlTi and PFFlTi, with no inputs in common and showing different input patterns in four situations. (*a*) Background activity. (*b*) An antidromic FETi spike (at arrow) evokes only a weak central effect on AIFlTi and which occurs later. (*c*) Passive oscillation of the tibia (flexion upwards) excites both to different extents. (*d*) Active flexion and extension elicited by a touch to the head calls forth different responses. Calibration: vertical (*a*) 3 mV, (*b*, *d*) 15 mV, (*c*) 6 mv; horizontal (*a*, *c*, *d*) 200 ms, (*b*) 100 ms. Torque in (*c*, *d*) is 50 mgf at 20 mm.

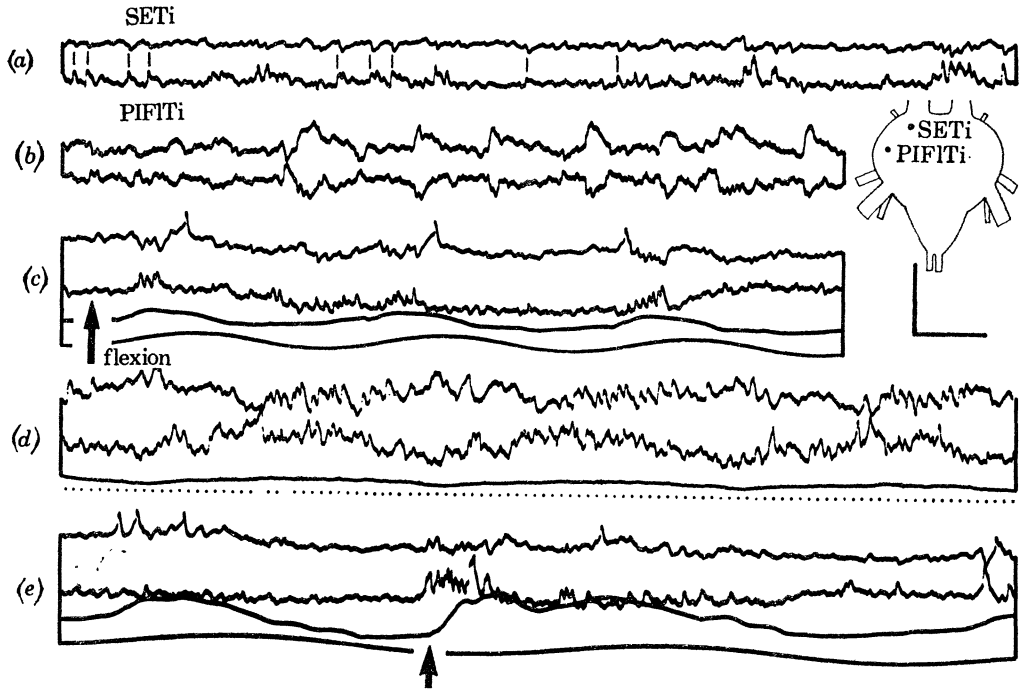

FIGURE 23. Opposite synchronous inputs to the slow extensor SETi (upper) and flexor PIFlTi. (*a*) Background activity with some common inputs indicated by vertical lines. (*b*) The same pair of neurons in a different locust. (*c*) As in (*a*). Passive oscillation of the tibia (flexion is upwards in 4th trace and tibial reaction in 3rd line) causes p.s.ps of opposite sign in the two neurons. (*d*) A repeat of (*b*) but with the two neurons from the locust in (*b*). (*e*) As in (*c*). A touch to the head (at arrow) causes an elicited response in which inputs are neither synchronous nor opposite as they are in the background or in imposed oscillation. Calibration: vertical (*a*, *c*, *e*) 10 mV and 5 mV, (*b*) 5 mV, (*d*) 7 mV; horizontal 200 ms. Torque in (*c*, *d*, *e*) is 50 mgf at 20 mm.

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responses. Exactly the same contrast in the effectiveness of inputs in common is found in FETi and SETi (figure 17*d*). Therefore it is not necessary to speculate that fast and slow motoneurons must always be separately excited by fast and slow interneurons, which derive their excitation from phasic and tonic receptors. Parallel pathways of this kind may indeed be separated among the interneurons, as among sensory and motoneurons, but as yet phasic and tonic interneurons are unknown, and the alternative mechanism has been demonstrated.

It has not escaped us that the absolutely consistent appearance of interneurons which form common inputs upon a particular group of motoneurons in every locust (figure 18*a–c*) implies that these motoneurons obey the same set of rules of growth in forming these synapses.

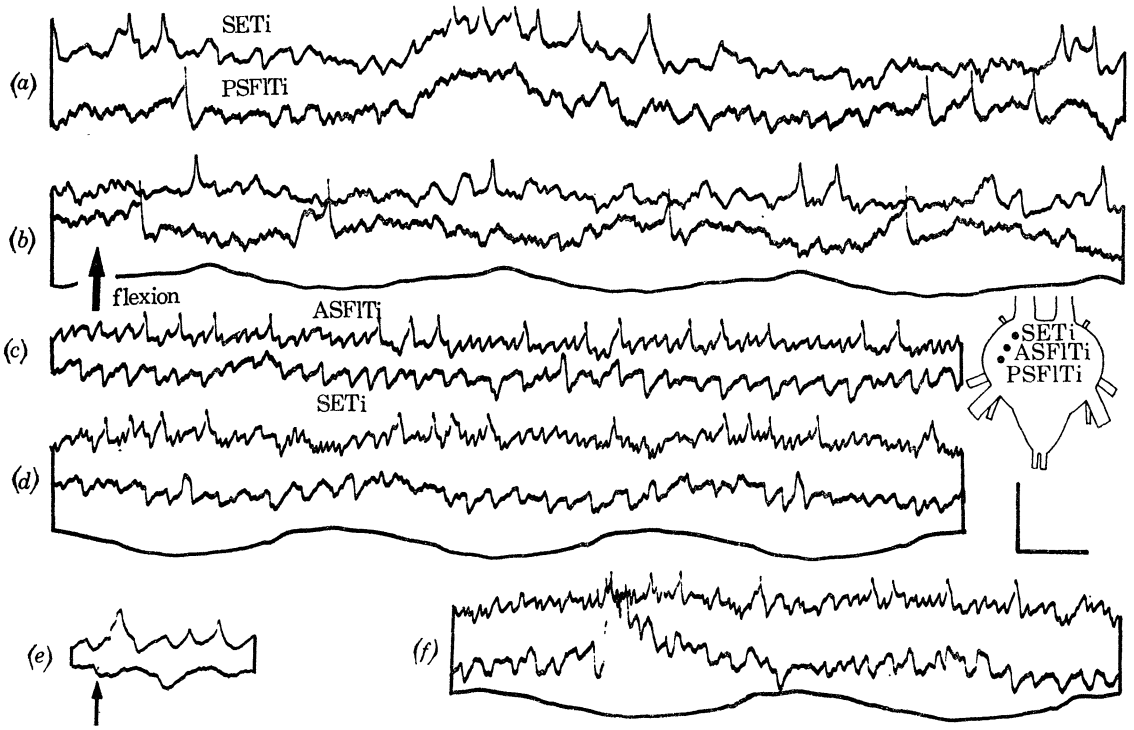

FIGURE 24. Lack of common inputs between slow extensor tibiae, SETi and the slow flexors, PSFITi and ASFITi. (*a*) An elicited response superimposed on background activity in SETi (upper) and PSFITi (lower) causing depolarization of both neurons. No p.s.ps are common. (*b*) As in (*a*), opposite responses to imposed tibial movements (flexion upwards, arrow). (*c*) Background activity in ASFITi (upper) and SETi (lower) with no i.p.s.ps in common. (*d*) As in (*c*) with imposed tibial movement (flexion upwards). (*e*) An antidromic spike in FETi (at arrow) causes reflex excitation of ASFITi, but inhibition of SETi. (*f*) As in (*d*) with an elicited response superimposed on the imposed tibial movement. Calibration: vertical: trace 1, 12 mV, trace 2, 1 mV; horizontal: (*a–d, f*) 200 ms, (*e*) 80 ms. Torque is 50 mgf at 20 mm.

Opposite synchronous inputs to antagonist motoneurons

A most striking lesson in the organization of this nervous system is to see simultaneous records from the slow extensor (SETi) and especially the PIFITi or the PFFITi and LFFITi from among the flexors. These three motoneurons, but certainly not all flexors, have i.p.s.ps when there is an e.p.s.p. to the SETi, and vice versa, so that one record resembles a mirror image of the other (figure 23). This happens in the background discharge upon the resting motoneurons, in their reflex responses to joint motion, but not necessarily in elicited responses. A mechanical resistance to the action of the extensor muscle causes synchronous e.p.s.ps in FETi and SETi, with an

i.p.s.p. in the PFFlTi (figure 17*d*). This is part of the mechanism of exerting increased extension force when there is an external resistance (figure 30). In contrast, other flexors (PSFlTi and ASFlTi) have no observed inputs in common with SETi or FETi (figure 24).

Opposite synchronous inputs during spontaneous background excitation are most simply interpreted as due to interneurons which have opposite effects on the two motoneurons; an inhibitory response to its transmitter at one interneuron output, an excitatory response to it at the other.

Other common inputs

We found a common i.p.s.p. between the fast extensor tibiae (FETi) and the fast depressor tarsus (FDTa) when the tibia is fully extended (figure 13*b*, *c*). This makes sense functionally, for when the leg is near the limit of tibial extension and tarsal depression, a sensory input of unknown origin indirectly inhibits these actions together via interneurons. This input is in addition to the e.p.s.p. in common between FETi, SETi, AI and PI in response to extension motion. There is also a common i.p.s.p. between at least two of the flexors PFFlTi and AFFlTi when the tibial spines are touched but we do not known whether this i.p.s.p. is synchronous with the i.p.s.p. in the FETi from the same stimulus.

On occasion we found common e.p.s.ps between a flexor tibiae motoneuron and one of the coxal adductor motoneurons and inferred that there may exist other sets of as yet unknown common inputs between coxal and other motoneurons of the leg. We found no other common inputs between motoneurons acting at different joints, even between synergistic muscles, and therefore the control which ensures coordination between different joints occurs in interactions preceding the responses that we record in motoneurons.

Common inputs on opposite sides of the ganglion

Simultaneous recordings from pairs of motoneurons has so far revealed only the following examples.

The motoneurons to the tergosternal wing elevator muscles on the two sides have a large proportion of e.p.s.ps in common when the flight tendency is strong but not in the background activity of a quiescent locust or from inputs from the wind receptors of the head (Burrows 1973*b*). The common inputs in the flight control of this neuron on the two sides are in keeping with, but not a prerequisite for, the simultaneous action of the two tergosternal muscles in flight.

The large visual interneurons which signal a small movement in the visual field, namely the descending contralateral movement detector (DCMD) and the descending ipsilateral movement detector (DIMD) from each connective, making four fibres in all, form synapses with observable simultaneous inputs upon the FETi motoneurons and inhibitory neurons (Burrows & Rowell 1973). The function could be related to the final release of a jump when the FETi motoneurons are almost on the point of generating impulses. Apart from the above, the FETi motoneurons on the two sides do not have common inputs. Other motoneurons on the two sides have not yet been examined in pairs, but reciprocal common inputs are unlikely because contralateral leg reflexes are weak or absent.

Features of common inputs

The following general conclusions emerge from numerous recordings of this kind, in which the arrivals of individual p.s.ps are noted in two or more motoneurons (figure 25).

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(*a*) Each response occurs independently in a particular selection of motoneurons which are acted upon by a set of interneurons with recognizable p.s.ps.

(*b*) Motoneurons to the same muscle can have a large proportion of their inputs in common for a particular movement, and other inputs, not in common, for other movements (figures 18–21).

(*c*) There are examples of motoneurons of antagonistic muscles where some of the i.p.s.ps in one neuron correspond to an e.p.s.p. in the other (figure 23). Other inputs, not in common, can depolarize both neurons together.

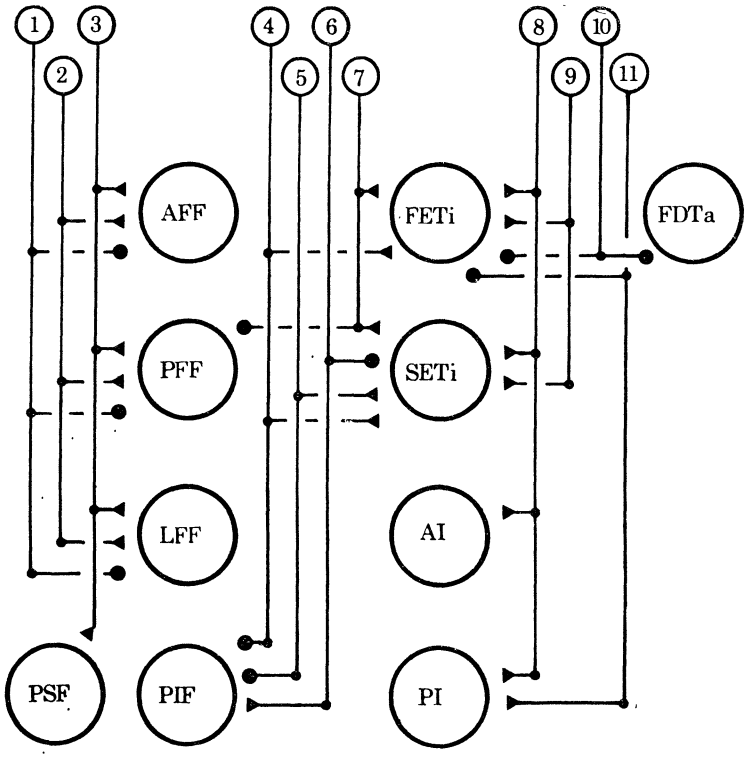

FIGURE 25. Identified interneurons with their inputs to ten motoneurons. These interneurons may have other connexions, and certainly the motoneurons have numerous additional inputs. These interneurons are identified under the following conditions.

Interneurons 1–3: background activity to flexor motoneurons (figures 20, 21); there are many interneurons of this type, and with the same connexions. *Interneurons 4 and 7*, seen during active extension against a load (figure 17*d*). *Interneurons 5 and 6*, mirror-image inputs to antagonistic femoral motoneurons (figure 23). *Interneuron 8*, in the second phase of a large passive extension of the tibia (figures 10*h*, 11*c*, 16*c*). *Interneuron 9* to slow and fast motoneurons to the extensor tibiae muscle (figures 16*a*, 30*f*). There are many interneurons of this type. *Interneuron 10* on extreme extension of the tibia, common to muscles of different joints (figures 13*b*, *c*). *Interneuron 11* on passive extension of the tibia (figure 10*f*). ●, inhibitory synapses, ▲, excitatory synapses.

(*d*) Most pairs of motoneurons, of muscles not having the above special relations, show no inputs in common. Even one pair of tibial flexors have none in common (figure 22).

(*e*) The inputs referred to above include those excited by proprioceptive and other sensory excitation from the leg itself or from other parts of the body, and from background central activity that is also clearly driven by interneuron terminals within the metathoracic ganglion.

(*f*) The pattern of p.s.ps can change with time in two or more motoneurons simultaneously; an input which is common to two motoneurons can disappear although no overt behavioural difference is observable.

(*g*) The frequency and pattern of the background discharge of p.s.ps and the occurrence

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of common inputs is not influenced by the section of all nerves and connectives to the ganglion (figure 19*b*). This implies a central control of posture.

(*h*) Occasionally, when motoneurons have inputs in common, indicating driving from a particular interneuron, the p.s.p. to one but not to the others can be dropped out, as if gated presynaptically (figure 20*a*, *b*).

(*i*) There are common inputs to corresponding motoneurons of the two sides from the descending visual interneurons (DCMD and DIMD) to the FETi motoneurons (Burrows & Rowell 1973), and from a variety of sources to paired flight motoneurons of the two sides (Burrows 1973*b*); other examples have not been found.

(*j*) The characteristic input patterns are found in every individual locust, except that some are found only in particular activity states, and they identify the inferred interneurons. The synaptic sites and dendritic patterns must be sufficiently accurate for p.s.ps to be constant from animal to animal.

General features of motoneuron responses

Lack of two-neuron arcs

Direct connexions between sensory neurons and motoneurons occur from the mammalian Ia afferents, and in the cord of the leech (Nicholls & Purves 1970), but are not known in crustacea or insects (for the most likely case, see Sandeman 1969).

Although we searched we found no evidence against the general conclusions that sensory impulses are not directly presynaptic to motoneurons (figures 6*a*, *b*, 7*h*, *i*). The main evidence is the lack of correspondence between sensory impulses and the p.s.ps in motoneurons, which respond apparently only indirectly. P.s.ps in motoneurons have latencies and distributions which suggest that they arise from interneuron impulses. The only positive identification of p.s.ps in motoneurons with known presynaptic impulses are from the DCMD and DIMD interneurons, and possibly in the brief excitation of fast flexor motoneurons by a FETi impulse.

In experiments where all nerves and connectives to the ganglion were cut while recording simultaneously from two flexor motoneurons with numerous inputs in common (figure 19*b*), there was no change in background activity of p.s.ps on the two motoneurons showing that sensory impulses are not involved.

A lack of two-neuron arcs makes sense in an animal with so few motoneurons that none can be reserved for particular fixed sensory inputs. A probable arrangement, with which observations agree, is that sensory impulses excite interneurons, and that motoneurons are controlled by interneurons. Once this rule is enunciated, exceptions to it can be sought.

Tonic sensory impulses and 'set' of the leg

One of the problems of insect posture is the origin and control of the background discharge in the slow motoneurons which is directly responsible for muscle tone. Known sense organs play a part; in the stick insect removal of ventral subcoxal hair plates has an effect on the joint position at rest and on the excursion of the leg in walking but removal of hair plates on all six legs apparently has little effect on coordination of the legs (Wendler 1964). In the locust, removal of the femoral–tibial chorotonal organ has an effect on resting position and excursion of the tibia (Usherwood *et al.* 1968). These or other unknown sense organs cannot account, however, for the choice of different 'set points' or postures to which the joint returns after displacement at different times. Although the locust lying on its back is not an ideal preparation it illustrates the following aspects.

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Proprioceptors exist with a background rate that is constant for a given position of the stationary leg, but tonic impulses recorded in the leg nerves are not directly presynaptic to the principal motoneurons involved in posture, i.e. no p.s.ps in motoneurons correspond with the tonic sensory impulses (figures 6*a*, 7*h*, *i*, 10*b*). Secondly, at the femoral–tibial joint tonic sensory impulses have no profound effect on the reflex effect of motion that can be recorded in motoneurons. In particular, the short-lived responses of the motoneurons controlling this joint are not modified when the joint is moved through a similar angle at the same velocity but at a different position in its traverse. The most extreme experimental evidence is that oscillating the fully bent femoral–tibial joint has a similar effect to oscillating it when fully extended although in the first case extension raises the body and in the second lowers it relative to the ground.

Thirdly, the tonic effect of the angle of the femoral–tibial joint upon the background rate of i.p.s.ps and e.p.s.ps to the tarsal motoneurons has already been noted (figure 5). Here we see that modulation by tarsal oscillation or by tibial oscillation is summed upon a 'set point' of the tarsus that depends on the tibial angle, and in different locusts the set point can be different for unknown reasons.

The posture of our dissected locust is controlled by a combination of the central mood combined to a small extent with the effects of tonic sensory impulses, as found by electrophysiological recording, so that 'set points' must be controlled in another way. Even if there were an insect equivalent of the muscle spindle, some central determination of set points is still essential. Besides other as yet unimagined mechanisms, two clearly important aspects have not been investigated; the part played by phasic receptors as they are excited by vibrations, and whether receptors for a particular body position control background impulse frequency of slow motoneurons by central mechanisms which integrate over a long time.

At any moment, therefore, posture appears to be the result of a centrally controlled pattern of tonic impulses in a selection of the slow motoneurons. The evidence that the locust has a variety of centrally determined patterns of motoneurons that cause different common postures is that the background rates of e.p.s.ps and i.p.s.ps in motoneurons differ in different preparations that are placed in the same posture. As a result, the frequencies of tonic discharge of slow motoneurons assume different patterns in different locusts. For example, adoption of a given femoral–tibial angle in two locusts can involve tonic background impulses in the SETi motoneuron in one but a replacement of impulses by i.p.s.ps in the other. Active preparations, but which are not moving their joints, have inhibitory neurons that are strongly suppressed by i.p.s.ps and slow motoneurons that are continually depolarized beyond threshold by e.p.s.ps. These differences must lie in the activity of particular interneurons. The point is that the central state is little influenced by immediate sensory inputs that cause resistance reflexes. A voluntary movement, however, always involves a shift in the settings of premotor inputs, and a consequent change in background discharge of slow motoneurons, although the sensory background can return to its former level and have as little effect as before.

Contralateral effects

Direct stimulation of nerve 5 causes a small depolarization in the contralateral FETi motoneuron that is independent of the occurrence of an impulse in the FETi motoneuron on the stimulated side. The initial response has a latency of 20 ms and is not attributable to direct action of the sensory volley on the contralateral FETi motoneuron because neither sensory arborizations nor motoneuron dendrites cross the midline. The exact sensory fibres involved

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are not known, and the effect is weak and transient, but bursts of impulses in the same sensory fibres probably account for the other effects below.

Active extension of the contralateral femoral–tibial joint has an excitatory effect on the posterior inhibitor neuron (PI), causing spikes if this neuron is already near threshold, and p.s.ps in the FETi motoneuron (figure 26*a*). There are also weak contralateral tarsal effects upon CI.

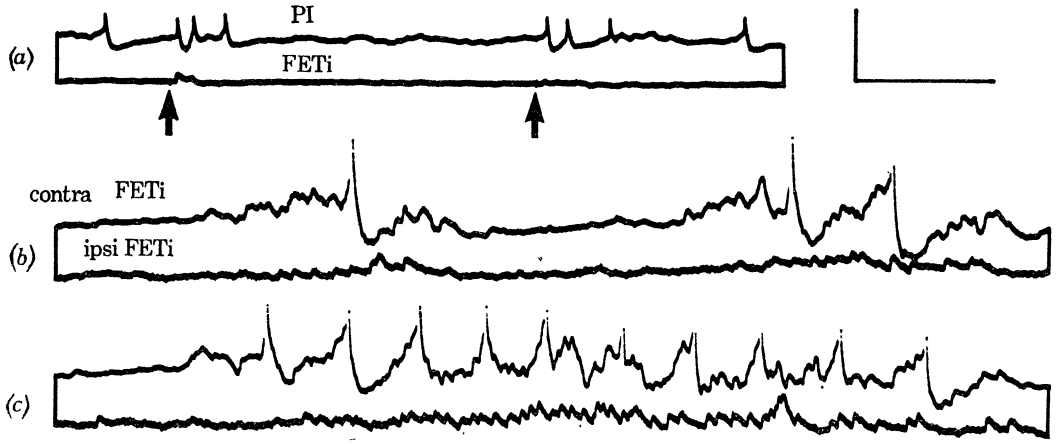

FIGURE 26. Responses to motion of the contralateral femoral–tibial joint. (*a*) The posterior inhibitor (PI, upper trace) and the FETi are depolarized by an active extension (at arrow) of the contralateral tibia caused by an antidromic FETi spike. (*b*, *c*) Contralateral FETi (upper trace) and ipsilateral FETi (lower trace). Forced flexion of the contralateral tibia causes a crossed excitation of the ipsilateral FETi but with no common p.s.ps. The e.p.s.ps are similar to those evoked by passive extension of the ipsilateral tibia. Calibration: vertical (*a*) 12 mV, (*b*, *c*) trace 1, 5 mV; trace 2, 2.5 mV; horizontal (*a*) 400 ms, (*b*, *c*) 200 ms.

Forced flexion or active extension against a load causes e.p.s.ps in the contralateral FETi motoneuron but not the burst of spikes that occurs in the ipsilateral FETi (figure 26*b*, *c*). The e.p.s.ps in the contralateral FETi appear to be the same as those seen in the ipsilateral FETi in the secondary response that follows the initial inhibition upon forced extension (figure 16*c*).

The only other contralateral effect that we have found in motoneuron recording is a crossed resistance reflex to motion of the tibial–tarsal joint. Levation of the contralateral tarsus has effects on the tarsal motoneurons similar to depression of the ipsilateral tarsus, and depression of it has the opposite effects. These interactions, not shown in the flow charts, are usually too weak to be seen as changes in impulse frequency or overt movement. The contralateral effects that we have found certainly could not account for the regular alternation of leg pairs during walking.

Signs of driving by different interneuron combinations

One of the basic hypotheses of the mechanism of movement control in most animals, vertebrates and invertebrates alike, is that various sets of interneurons produce different movements by exciting different combinations of premotor interneurons. For example, the eye muscles of the crab produce three types of movements, optokinetic, compensatory, and protective withdrawal (Burrows & Horridge 1968*b*, *c*). The first two involve the same motoneurons which are excited in different proportions for the different directions of movement, the third involves additional motoneurons although the same muscles participate in all the movements. Examples of central mechanisms, in which branching interneurons in combinations are presynaptic to

particular patterns of other neurons, are illustrated in *Aplysia* (Kandel 1969) but without relation to the normal function or significance of the output. In the locust we record from motoneurons of known function and can illustrate the mechanisms by which these are controlled in groups by patterns of interneurons.

With many locust motoneurons it is obvious that the p.s.ps which arise from one set of inputs are quite different in size, shape, and pattern of arrival from those initiated by other stimuli (figures 6*c*, 24*f*). Some of the tests for the summation between the resistance reflex and the response to a stimulus elsewhere show well the differences between p.s.ps from different interneurons (figure 6*c*). These summation patterns do not necessarily make functional sense, for example CI, FETi and PFFlTi are all depolarized together in the elicited response (figure 11*d*). On account of the subsequent summation on motoneurons, simultaneous records from interneurons would be less likely to make functional sense, because the function lies in the final effect on the leg.

In all recordings from motoneurons p.s.ps sum until they reach the threshold of orthodromic impulse initiation. This summation operates smoothly over periods of a second or more. One surprising feature of the FETi is the slowness with which it can be depolarized to threshold by stimulation of the ipsilateral thoracic connective, and the slow recovery on cessation (figure 7*a*, *b*) although we think of the FETi as one of the largest and most phasic motoneurons.

That spatially separated inputs act upon motoneurons independently can sometimes be inferred from the potential at which impulses arise. For example, an imposed extension of the tibia causes depolarization of a flexor tibiae (AFFlTi) motoneuron which does not evoke impulses. An elicited response, however, calls forth an active flexion and the membrane potential of the soma is now *lower* than that previously exceeded without spike initiation (figure 6*c*). Therefore the inputs causing the spontaneous movement are farther from the electrode and less effective in depolarizing the soma than those recorded before the spontaneous movement. Similar differences in the recorded threshold for impulses are seen in figure 13*a*.

Despite this problem caused by inputs at different distances from the electrode, it is usually clear that motoneuron impulses arise when threshold is reached and not by electrical transmission of impulses from other neurons. In the FETi motoneuron (figure 30*c*) we prove this by showing that an antidromic spike resets the regular sequence of impulses (confirming Godden 1969), and therefore the impulse initiation point, but not the source of dendritic depolarization, is accessible to the antidromic impulse.

Often one sense organ arouses dissimilar responses in a group of motoneurons by activating different interneurons. Examples are the tibial spines which excite common i.p.s.ps in at least two flexors but independent e.p.s.ps in other motoneurons. Independent e.p.s.ps in the slow extensor tibiae and the common inhibitor are caused by touch to tarsal hairs, but still account for the impulses aroused from both these neurons as seen in peripheral records (Runion & Usherwood 1968).

The SETi and the FETi have many inputs in common, but the SETi is sometimes held in a suppressed state by i.p.s.ps that are not visible in the FETi (figure 17*a*). These i.p.s.ps are clearly inhibited by tibial flexion, which at the same time indirectly causes e.p.s.ps in both SETi and FETi (figure 17*b*, *c*). A stimulus to another part of the body may produce identical patterns of p.s.ps upon two synergist motoneurons, but in the spontaneous background discharge of p.s.ps upon them, the two may have less in common. Even more striking, the SETi and a flexor (PIFlTi), with inputs in opposite directions, frequently are depolarized together by a

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stimulus elsewhere on the body (figure 23*e*). The interneuron combinations can also change with time. In one example, the SETi and a flexor motoneuron (PIFlTi) had almost mirror-image inputs when first recorded, but subsequently had much less in common during spontaneous background activity after the locust had been touched on the antennae or on the legs.

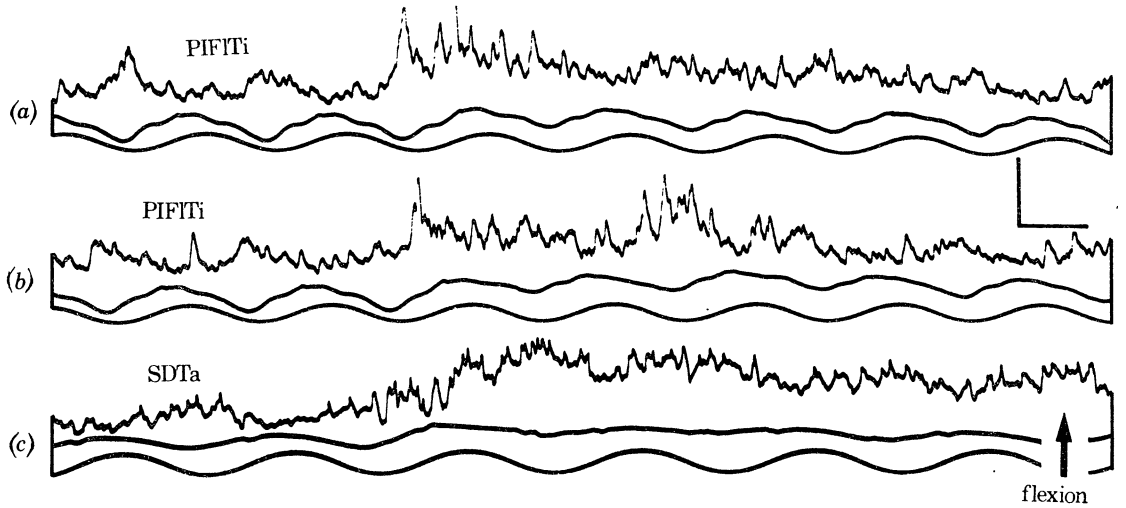

FIGURE 27. An elicited response superimposed on a reflex response to an applied movement of the tibia showing that the two effects sum upon the motoneuron. The lower trace is the movement of the tibia (flexion upwards) the middle trace shows the reaction of the tibia. (*a*, *b*) The PIFlTi is excited by imposed extension and by an elicited response causing an incipient flexion. (*c*) Modulation of the SDTa by an imposed flexion of the tibia. When an active flexion is evoked the SDTa shows i.p.s.ps preceding the large depolarization and the adoption of a new set point. Calibration: vertical 12 mV; horizontal 200 ms. Torque is 50 mgf at 20 mm.

Summation and gating at motoneuron level

As outlined in figure 2, the control of leg movement involves both central and peripheral effects which sum locally upon interneurons. The questions arise as to whether the reflex loop is changed by central effects such as arousal, and whether the resistance reflex can be temporarily cut off when other movements are elicited, or by excitation elsewhere in the nervous system. In the stick insect passive extension of the tibia causes an opposing contraction of the flexor muscle which varies on different occasions as if the central gain in the feedback loop is variable (Bässler 1972*a*). Further analysis, however, shows that the central gain is a product of many components in series, and the apparent adaptation in the control loop may be caused by a change in the set point of the tibia. To study summation and search for changes in gain we imposed a background oscillation on the femoral–tibial joint and observed the effects on this of eliciting other leg movements while recording from various motoneurons and the torque at the femoral–tibial joint.

The general result is that resistance reflexes at the femoral–tibial joint, observed as potentials in the motoneurons, are not interrupted or changed in gain by inputs of any other kind. They merely sum (figures 27–29), but there are exceptions which show that a constant reflex response, modified only by habituation (figure 28*a*) is not obligatory. Summation that occurs on premotor interneurons is sometimes observed as a gating effect (figure 20*a*, *b*) or change of gain at the motoneuron level.

The resistance reflex at the femoral–tibial joint is singularly resistant to central modification

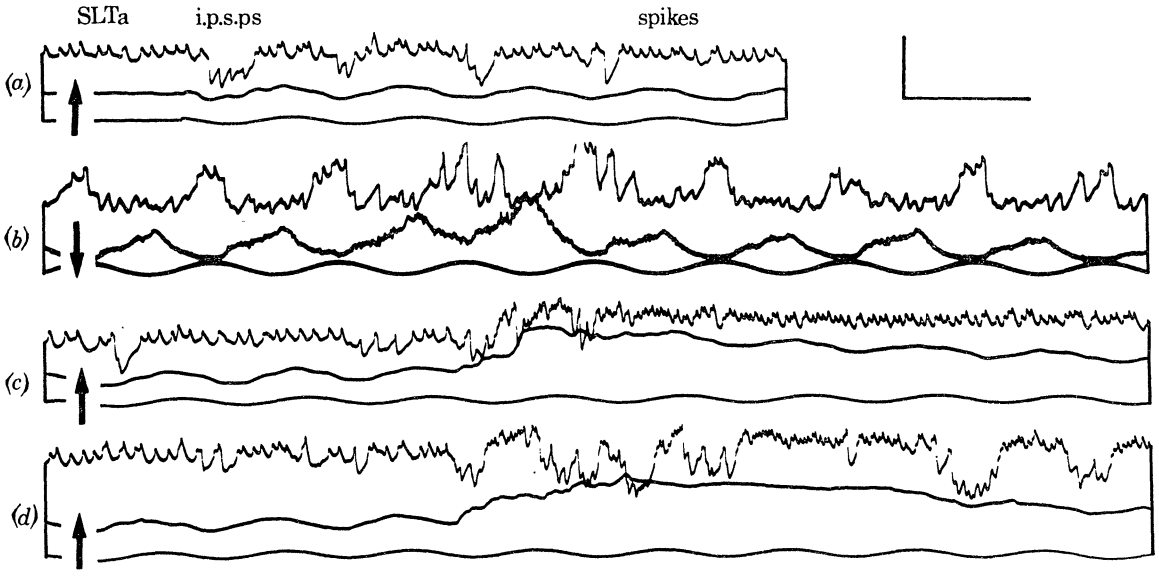

FIGURE 28. Summation of reflex and elicited responses upon the slow levator tarsus (SLTa). The lower trace is the oscillation of the *tibia* with flexion in the direction of the arrow. The middle trace shows the reaction of the tibia and the elicited response that is imposed on the oscillation. The tarsal motoneuron shows its own modulation, controlled reflexly by the tibial motion. (*a*) Start of tibial oscillation and habituation of i.p.s.ps. (*b*) Oscillation such that tibial flexion causes spikes, with a superimposed voluntary tibial extension also summing on SLTa. (*c*) The temporary loss of the i.p.s.ps on tibial extension during an attempted flexion of the tibia. (*d*) Summation of the periodic and elicited flexion to give an irregular mixture of effects on the SLTa. The i.p.s.ps upon extension are enhanced when they return. Calibration: vertical (*a*, *c* and *d*) 10 mV, (*b*) 15 mV; horizontal 400 ms. Torque is 50 mgf at 20 mm.

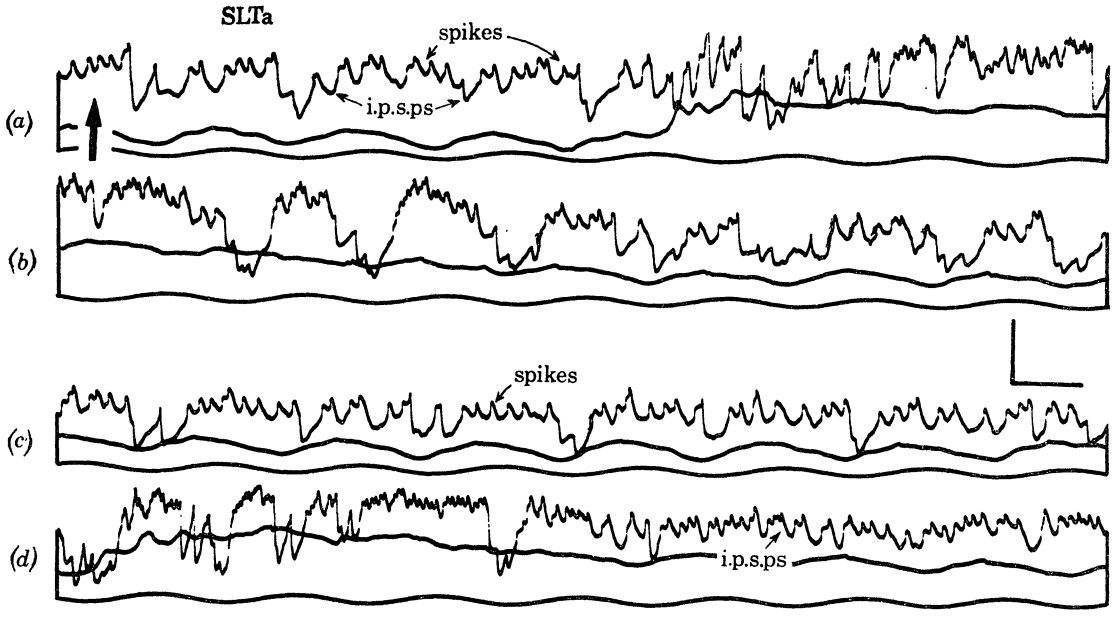

FIGURE 29. Central effects during modulation of the slow levator tarsus (SLTa) by oscillation of the femoral–tibial joint. The lower trace shows the tibial movement, the middle trace the reaction of the tibia. (*a*) continued in (*b*). At each extension of the tibia the SLTa is inhibited by a few large i.p.s.ps, which after the elicited response shown in (*a*) become more pronounced in (*b*). (*c*) continued in (*d*). The large i.p.s.ps upon tibial extension now disappear during the elicited response and take time to recover. Calibration: vertical 10 mV; horizontal 200 ms. Torque is 50 mgf at 20 mm.

as observed at the motoneuron. Despite numerous efforts to modify the oscillatory responses of extensor and flexor motoneurons we found only one example, in which e.p.s.ps to the PIFlTi caused by extension of the tibia were blocked temporarily during a response to touch on the mouthparts (figures 27*a*, *b*) but the effect is neither strong nor consistent.

The intra-leg interaction controlling tarsal position indirectly from femoral–tibial joint receptors showed more lability (figures 27–29). The SDTa motoneuron responds regularly to oscillation of the *tibia*, but at a touch to the mouthparts the tension in the tibial reflex response increases and the oscillation of the SDTa electrical response temporarily fails, then progressively recovers (figure 27*c*).

Similarly a regular, repeated pattern of i.p.s.ps grow larger during an elicited response which is imposed on a background oscillation in the slow levator tarsus (SLTa) (figures 28, 29). This effect is clearly a change in central gain in the reflex and could be interpreted as a gating of the proprioceptive response, but that is merely to describe it in other words. The records suggest that during the elicited response the premotor interneurons causing the reflex response are temporarily shifted by another input to a different part of the range of their dynamic response curve.

All of the above are temporary effects of touch elsewhere on the body, and could be called 'arousal' or its opposite. Because most of the modulation observed in the motoneurons is sub-threshold, it is not directly related to the mechanical effect, which may be caused by only one slow motoneuron. We find that resistance reflexes are mechanically insignificant in comparison with the strong effects of central commands, so that changes in gain become unimportant.

Central interactions between motoneurons

Changes in membrane potential or impulses in one motoneuron in general have no direct effect upon other motoneurons via pathways within the ganglion even when such might appear desirable. We found, for example, no central effects from impulses in motoneurons which control the femoral–tibial joint upon motoneurons which control the tarsal joint although the two joints are coupled via persistent reflexes (figures 13*f*, *g*, 14). The exception to this rule is that curious interactions, described by Hoyle & Burrows (1973*a*), occur between the motoneurons that control the tibia. A single FETi spike causes a brief inhibition of SETi (figure 16*f*, *g*) with a latency of 15–20 ms, and also a depolarization of flexor neurons (figures 20*c*, 22*b*). A SETi spike causes a labile brief inhibition of the FETi with a latency of 20–25 ms. These effects are central as shown by their occurrence before sensory impulses signalling extension 'motion' can arrive at the ganglion (figure 6*f*) and by their persistence when movement is prevented (figure 13*f*) or when the leg is removed (figure 16*h*).

Control of particular functions

The organization of the jump mechanism

In the jump of the locust, motor impulses to the corresponding muscles of the two sides are not synchronized (Godden 1969), in agreement with the finding that FETi motoneurons of the two sides are not electrically coupled (Hoyle & Burrows 1973*b*) and that they have no common inputs except from DMD visual fibres. Another fact already known is that the flexor muscle is excited at the same time as the extensor before the jump occurs (Godden 1969). Other relevant information is that the locust will not jump unless the tibia is fully flexed (Brown 1967) or if the femoral chordotonal organ is removed (Bässler 1968).

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An alarmed locust flexes its metathoracic legs so that the tarsi are under the body, with the tibia fitting into a groove on the femur. In agreement with this preparatory movement, any stimulus to a locust causes depolarization of the tibial flexor motoneurons (figures 19*e*, 22*d*, 27*a*, *b*) and halts the walk and other movements. This is in marked contrast to many other insects; the cockroach, for example, never stops to jump but instead runs faster.

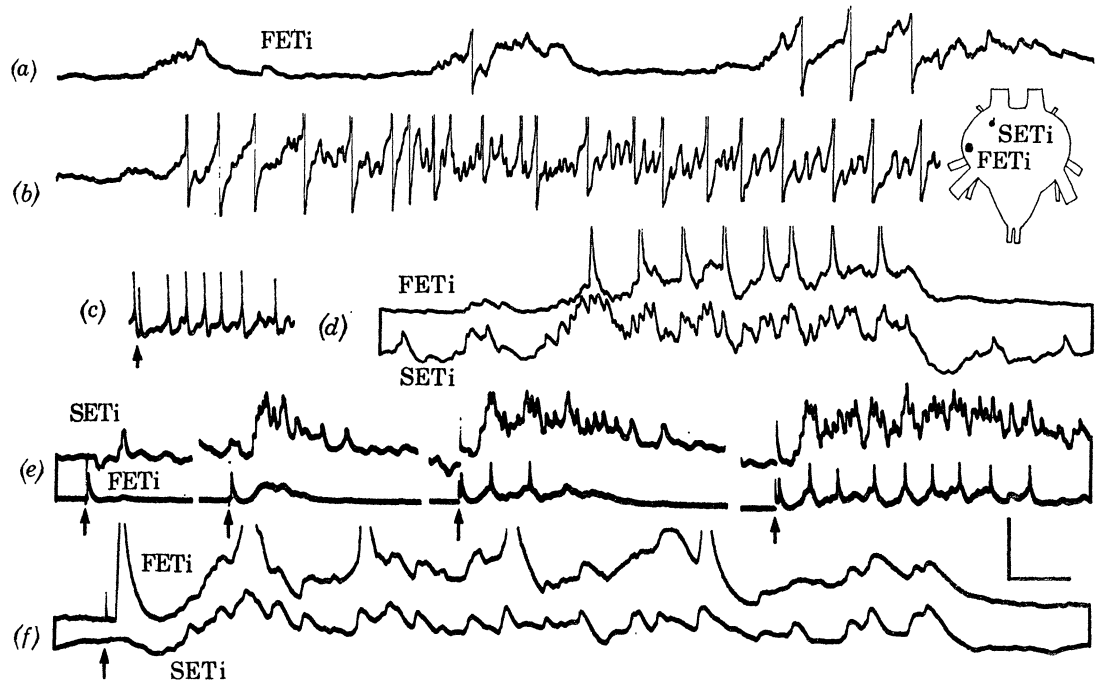

FIGURE 30. Responses of SETi and FETi related to the mechanism of extension against a resistance. (*a*) The tibia is forcibly flexed to an increasing degree, until the positive feedback caused by the resistance maintains a repetitive discharge of impulses in (*b*). (*c*) An antidromic impulse during the FETi discharge causes a brief pause and resets the spike sequence. (*d*) Parallel responses in the FETi and SETi (lower trace) during forced flexion of the tibia. (*e*) An antidromic FETi spike (lower trace) causes a brief extension of the tibia. On the four sweeps in (*e*) the extension is progressively resisted. (*f*) As in (*e*) showing the common e.p.s.ps to SETi (lower trace) and FETi. Calibration: vertical (*a–c*) 15 mV, (*d*, *e*) 10 mV, (*f*) 5 mV; horizontal (*a–c*, *e*) 200 ms, (*d*) 100 ms, (*f*) 50 ms.

The jump is preceded and can be accompanied by impulses to the flexor muscle which have no central effects on other motoneurons, except that impulses in one flexor (AIFlTi) indirectly cause small e.p.s.ps in the FETi motoneuron (Hoyle & Burrows 1973*a*). The central command to jump often comes suddenly in an otherwise stationary locust. The FETi motoneuron generates an impulse which causes a small increase in tension in the extensor muscle, but no movement if there is a slight tension in the flexor because the latter is aided by a catch mechanism on the flexor tendon (Heitler 1974) and has much greater mechanical advantage when the joint is fully flexed. The extensor is thus acting against a large external resistance and detectors of the extension force rapidly add to the excitation in the FETi, causing a burst of impulses (figure 30). The tension in the extensor muscle rapidly builds up, but is resisted by additional impulses of the flexor motoneurons which receive a component of depolarization from the centrally mediated effect of the FETi spikes. When the tibia is held flexed under experimental conditions the FETi motoneuron generates a long series of impulses (figures 26*c*, 30*b*) which is maintained by the positive feedback loop (figure 31). As long as the tibia remains

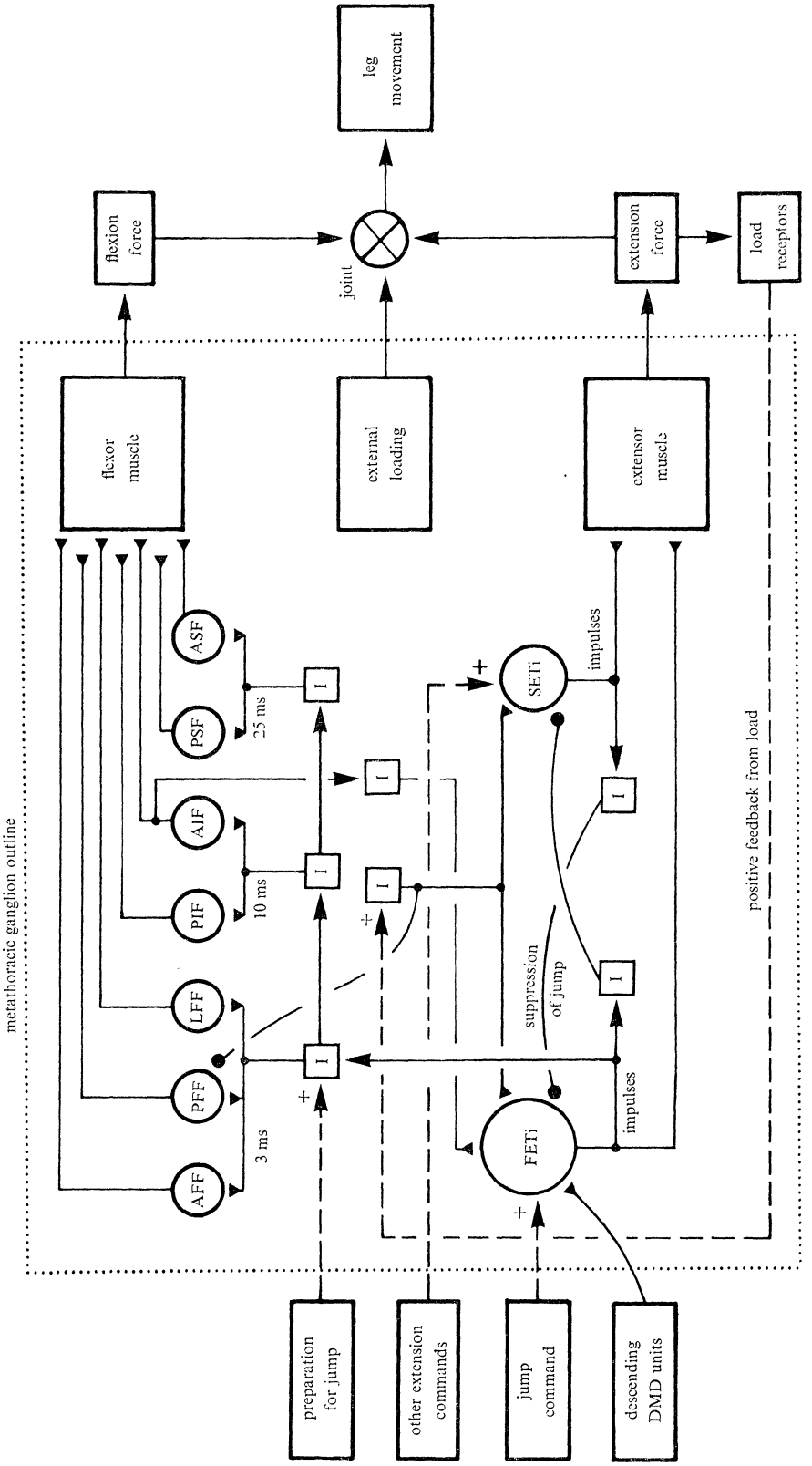

FIGURE 31. The central interactions between femoral motoneurons which are related to the jumping behaviour. Data is taken from Hoyle & Burrows (1973*a*, *b*) as well as from this paper. ●, inhibitory synapses; ▲, excitatory synapses.

flexed and the extensor is working against a load this positive feedback generates a rapidly growing torque at the femoral–tibial joint, so that the command to jump need initiate only a single FETi impulse.

When the flexor is no longer able to prevent the extension of the tibia, the mechanical advantage of the extensor muscle is increased by the extension of the joint so that the energy stored in the extensor muscle is released and the locust jumps. The tibia extends over a period of 20 ms (Brown 1967).

The release of the load on the extensor brings to an end the positive feedback upon it and full extension causes i.p.s.ps in the FETi motoneuron (figure 13*c*). Towards the end of extension the extensor muscle has lost its effectiveness in generating force (figure 9*b*) because its fibres are short and attached to the extensor tendon at an angle which increases as extension proceeds. The flexor muscle, on the other hand, becomes more effective at greater extensions because its long straight fibres are progressively more stretched. The flexor muscle therefore acts as a smooth brake to prevent over-extension and is able to flex the leg rapidly in the recovery.

There is no evidence that the resistance reflexes are suppressed during a jump; presumably the rapid extension assists the recovery of the flexed position but reflexes are usually swamped by much larger central interactions and sudden changes in the centrally controlled set point are more important. When the locust takes to flight, instead of landing again as in the evasive jump, the legs assume a new posture but how this is controlled is unknown.

Extensions of the leg other than the jump are brought about by the slow extensor motoneuron, impulses in which indirectly cause a labile inhibition of the FETi motoneuron, so that the jump mechanism is not excited by stimuli that elicit normal movements, although the SETi and FETi have many synaptic inputs in common.

All the known central interactions relate to the muscles that control the femoral–tibial joint, and all except the brief inhibition of SETi by FETi impulses can be accounted for in functional terms related to the jump (figure 31). Therefore it is a reasonable hypothesis that these central interactions are specializations to accommodate the mechanics of the jump, and it is a testable hypothesis that these central interactions will be found only in the metathoracic segment of jumping Orthoptera.

The defensive kick

When a locust is touched, particularly on the abdomen, it brings the terminal tibial spines up to the offending object and accurately pushes it away. When held roughly it kicks so that the strong spines of the metathoracic tibia become an effective weapon.

Touch to the abdomen causes depolarization of flexor and extensor motoneurons via indirect pathways that presumably generate a central pattern that turns the leg remarkably accurately in the appropriate direction, then flexes it in preparation for a kick. Evidence for the central control is that animals with one leg recently removed show patterns of depolarization similar to normal ones. A sufficiently strong stimulus depolarizes the FETi motoneuron, which generates a spike. One impulse in this motoneuron causes an extension by 10–20° at the femoral–tibial joint when the leg is free to move. The leg therefore quickly meets the resistance of the offending object, and the resultant loading indirectly causes coincident e.p.s.ps in FETi and SETi and e.p.s.ps in three of the flexors (AFFlTi, PFFlTi and LFFlTi) (figure 25). The FETi motoneuron is thereby so depolarized that it generates a rapid series of impulses that cause a vigorous kick and push. Contact of the tibial spines, however, inhibits the FETi motoneuron (figure 7*c*) so

the extension is not uncontrollable as it would be if spine stimulation also caused a positive feedback to the FETi neuron.

As soon as the external resistance gives way the motion of the joint during extension inhibits the FETi neuron and excites the flexors reflexly (figure 6*a*), bringing the movement to an end most effectively when the leg is most extended.

Discussion

A marked shift of analytical procedure has been made, away from previous methods of inferring mechanisms from leg movements and extracellular records, mainly of peripheral impulses, in the direction of systematic collection of data about central components. The actions of other neurons upon motoneurons are made as explicit as possible. The results illustrate one way of analysing behaviour by direct observation of mechanisms, and reveal the large part played by central pattern in contrast to reflexes from joint movements. The method also shows the importance of the motoneuron responses for further progress, which now depends on the identification of interneurons.

This method of analysis of behaviour

As outlined in the section on 'analysis', the first essential step in the unravelling of a nervous control system is the listing of components, but this is almost never completed. In the movements analysed here the list of the simplest components is not complete, despite the fact that the locust is one of the animals most intensely studied by physiological analysis. The total distribution of motoneurons has not been worked out, the action and distribution of the inhibitors is almost entirely undescribed, and the responses of the sense organs are known only partially. Receptors for load, new sense organs, and possibly novel forms of interaction remain to be described. Even new types of components are likely to appear; for example, some of the dorsal cells described by Crossman, Kerkut & Walker (1972) have axons to both legs but obscure functions (Hoyle, Dagan, Moberly & Colquhoun 1974).

The second step is to set out the interactions between components in summaries of observations as in figures 1–4. While the locust is making spontaneous movements, however, the motoneuron potentials become so complex that interactions cannot be distinguished. One way to progress is then to refine the measurements and increase the number of recording channels so that the simple interactions become better known and hopefully the complex ones are a logical consequence of them. By this method we showed directly that the motoneurons are necessarily excited and inhibited in various permutations by interneurons that are active in different sets in different voluntary activities.

The third step is to make more explicit the boxes in the flow chart which represent the interneurons and thereby uncover the mechanism of generation of patterned movement. As yet none of the interneurons are identified, but a way of distinguishing interneurons lies in the observations of the common inputs of some of them upon particular sets of motoneurons. Several can be characterized (figure 25), and are constant in every locust. Interneuron identification will also depend upon recording from them together with the motoneurons which they control. Our efforts so far to find pairs of appropriate neurons only impress us with the magnitude of the task.

The alternative approach, and until recently the only one available, is to record the patterns

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of motor impulses, or even the leg movements, and infer interactions of components. One cannot then distinguish between a large number of models because excitation is divided between many channels in parallel, and there are many ways of generating the data in the outputs from a ganglion of several hundred neurons. The method of model building only from observations of outputs will always give way before the method, advocated here, of observing how the components actually interact.

Control of movement in the locust leg

From myograms of motoneuron activity in freely moving locusts Hoyle (1964) inferred the following points that have been substantiated. (*a*) Motoneurons are driven by interneurons and interact little among themselves. (*b*) Reflex pathways and patterned central commands act on these interneurons, not on the motoneurons. (*c*) Reciprocal inhibitory cross-connexions lie between interneurons only. (*d*) The hypothesis of a 'general driver interneuron', which can simultaneously excite many motoneurons, some of which are functionally antagonistic. (*e*) The hypothesis of 'specific driver interneurons' which excite one or few synergic motoneurons. (*f*) The motor impulse pattern to the legs in a subliminal response is similar to that in a stronger response that causes a movement. Therefore elicited responses are basically central commands, at least when just initiated, and before proprioception complicates the pattern.

The shift in analytical procedure from periphery to motoneuron has revealed numerous new interactions. A finding is that as excitation flows through the nervous system in the generation of a movement the pattern of which neurons are excited becomes progressively more important, from sensory, through interneurons to motoneurons. As we also inferred for the control of the crab eyecup (Burrows & Horridge 1968*a*, *b*, *c*), each stimulus situation generates its own appropriate patterned central command, and posture at any time is the result of the history of stimulus situations. An advantage of the crab eyecup preparation was that proprioceptive reflexes are absent and all movement and posture is a centrally determined pattern of motoneuron impulses. In the insect leg the presence of proprioceptive reflexes has delayed the conclusion that here also control of movement is by central patterns that are preformed in the connections of interneurons. At present we do not know how patterns are generated or how the choice of an appropriate pattern is made. The term 'central command' is a convenient way of referring to this central activity but analysis of actual interneuron activity must progressively show that central commands are better expressed in terms of flow charts of interneuron actions. If some aspect of a central command is demonstrably not an interneuron relationship then it becomes a mechanism definable in some other terms. Although now a convenient concept, 'central commands' will disappear as they are resolved into demonstrable interactions.

Inputs to motoneurons

A conclusion from the study of inputs to motoneurons is that each movement is caused by excitation of a particular set of motoneurons by a selection of interneurons that generate an appropriate pattern. The study of *common* inputs shows that one interneuron can control several motoneurons simultaneously, but most inputs cannot be shown to be of this type by recording from only a few motoneurons. Therefore the study of inputs reveals what lies behind the motoneurons. To characterize interneurons, which are not self-labelling, one can record from them together with the motoneurons upon which they have inputs, or pick them out as common inputs from simultaneous recordings of numerous motoneurons. This can be done only when

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motoneuron p.s.ps are individually attributable to interneuron impulses because only this strict causal relationship isolates one particular pathway. The detailed study of p.s.ps is one way to distinguish one out of a number of alternative interneuron circuits.

All recordings from the motoneuron somata show smooth changes in membrane potential with insufficient unitary p.s.ps to account for them. Some inputs lie beyond the range of the electrode or are possibly nowhere present as unitary p.s.ps in the dendrites (Hoyle & Burrows 1973*a*). Even so, many positive inferences have been made from the p.s.ps. To suggest that p.s.ps recorded in the soma as entities above noise are only a small proportion of p.s.ps falling upon the motoneuron and thereby claim that inferences cannot be based upon partial evidence is to ignore that evidence, by its nature, is a partial story. The immense ramifications of the motoneuron dendrites (Burrows 1973*c*) suggest that no point would yield a total picture of the p.s.ps, although subsequent analysis will no doubt extract more data from the waveforms than has been possible in our preliminary visual inspection.

The part played by resistance reflexes

The analysis of reflexes caused by motion of the leg joints showed that they are the result of interneuron activity which excites or inhibits the motoneurons appropriately. The reflexes are stable, not easily blocked or changed in gain, and persist through other movements. Therefore the reflex effect of joint motion necessarily influences any motoneuron which is near its threshold and causes it to participate earlier or later than otherwise in other movements. Perhaps this is their whole purpose because alone they are mechanically inadequate to match the loads of body weight or of spontaneous leg activities.

The reflex response to joint motion involves a few millivolts change in slow motoneuron potentials and mechanical effects of a few milligrams; elicited responses involve depolarization of fast motoneurons by 10–20 mV and loads measured in grams. The analysis of the reflexes eliminates them as important in movement control. Furthermore, the sense organs that are described are those sensitive to motion not to load, and it is leg movement rather than leg loading which has been studied previously.

Although there is general agreement in the literature that resistance reflexes at leg joints are present during posture and passive movements of insects (Pringle 1940; Wilson 1965; Delcomyn 1971), only Bässler (1967, 1972*a*, *b*) measured the forces quantitatively. Bässler gives values of up to about 1 gf for the reaction to stretching of the femoral–tibial joint of the stick insect at 2.4° s^{-1} and measurements showing that about 0.1 gf can be exerted for many minutes by this joint after a maintained passive bending. To account for the eventual decay, Bässler inferred that a phasic filter stands in the pathway of the tonic proprioceptive impulses (figure 32*a*) but omitted possible changes in the central control of posture by integration of sensory impulses upon interneurons.

In measurements of the mechanical reaction of the femoral–tibial joint of *Schistocerca* there were three main findings. (*a*) The elasticity and tension of the muscles *in situ* in the isolated leg generate a torque of about 30 mgf at 20 mm measured at the distal end of the tibia for a movement of each 20° around the resting position. (*b*) The torque reactions to an oscillation of about 20° at a frequency of 0.1 Hz to 5 Hz are rather greater values up to 50 mgf at 20 mm at the same point. (*c*) There is no significant fall in the torque reaction when the leg nerves are cut in the intact animal. Therefore the resting tensions in muscles are larger than the mechanical effects of the resistance reflexes, but still small compared with normal loadings. This agrees

with our finding that the posture of the leg is determined mainly by a centrally determined background discharge and to a minor extent by peripheral tonic receptors. The same conclusion follows from an entirely different study of posture in the learning of leg position in the locust (Horridge 1965; Hoyle 1965).

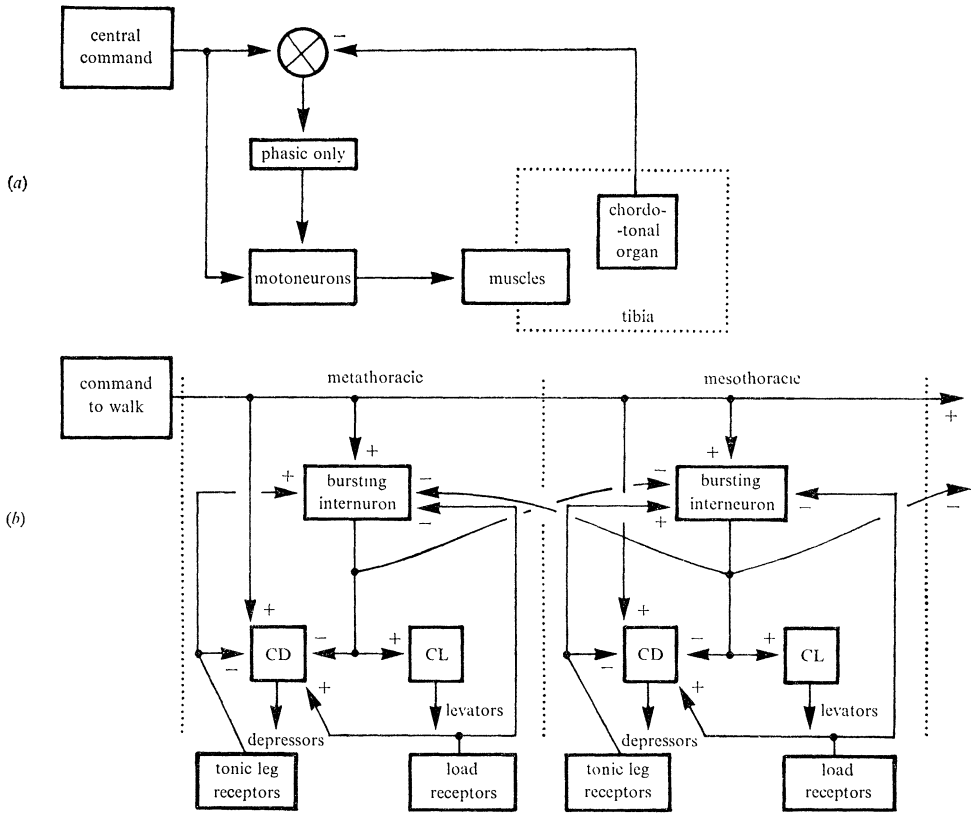

FIGURE 32. Hypothetical relations inferred previously for two interactions discussed. (*a*) In the stick insect a maintained stretch of the chordotonal organ causes the tibia to move or exert a force but slowly it returns to its former state, i.e. the feedback loop has a phasic filter (Bässler 1972*b*). (*b*) In the cockroach, tonic leg receptors excite the central burst generator for levation and inhibit depressor motoneurons. Load detectors have positive feedback upon depressors and inhibit the central oscillator, which excites levator and depressor motoneurons in alternating bursts in the absence of sensory feedback. The bursting interneuron inhibits its partners in neighbouring segments (Pearson 1972; Pearson & Iles 1973).

Relevance to the analysis of walking

As Pearson & Iles (1973) point out, the nervous mechanisms responsible for coordinating the legs in insect walking are poorly understood. They derive a model (figure 32*b*) for the co-ordination of bursts of impulses by coxal levator and depressor motoneurons of the cockroach from three main findings: (*a*) In the absence of sensory input the coxal levator and depressor motoneurons can generate alternating bursts similar to those seen in walking. (*b*) Receptors to loading of the leg, presumably companiform sensilla of the trochanter, as inferred by Pringle (1961), have a positive feedback upon the coxal depressor motoneuron and an inhibitory effect on the central mechanism controlling bursting in the coxal levator motoneuron. (*c*) Bursts of impulses in the meso-metathoracic connectives are timed in such a way that they could be the interganglionic pathway whereby coxal levator bursts do not occur simultaneously in two

adjacent legs. The model proposed is really no more than a restatement of these observations, and bears little relation to the diversity and numbers of components revealed by recording from the central neurons.

The relative effects of types of afference from the leg itself upon its own motoneurons are relevant to the analysis of walking. Touch to the footpad strongly excites most of the motor and inhibitory neurons. If these responses persist during walking the control of motoneurons must be strongly influenced by the movements themselves and the irregularities of the terrain. Touch to tibial spines inhibits several of the motoneurons but the function is not clear except during a defensive kick. All the efferent neurons to the leg are weakly influenced by movement of tarsal or tibial joints. Therefore passive motion caused by traction from the ground as other legs move is not likely to be of major importance in the control of walking (at least for these joints in the locust). On the other hand, during an active movement, receptors to load generate a strong and specific positive feedback that must be significant when there is a mechanical resistance to movement.

At first sight the observation that movement or touch of the leg of the opposite side has only a relatively weak effect on the motoneurons suggests that reflexes from the contralateral leg are always weak, and therefore that the regular alternation of the two sides in walking is centrally coordinated. This may turn out to be so, but cannot as yet be accepted for two reasons that apply to all inferences from the resting to the walking animal. First, we have not studied responses during alternation, and new responses of motoneurons that are relevant during walking may appear only when the insect is in the act, and others described here may be blocked. Secondly, we have not actually examined the effects of loading, of stimulating hair plates, and of traction effects via the ground in each leg, and so cannot make a general inference that the coordination is always central. As stressed elsewhere, there is a wide and uncharted gap between our observations of motoneuron responses and the inference of mechanisms of normal movements under load.

The way forward is to record from the central neurons during walking and other movements, but the same obstacles which we have found will be met. Recording from motoneurons does not reveal the origin of pattern in the sets of interneurons which lie behind them. In the interneuron networks none of the components or their interactions are known except for the signs given by inputs to motoneurons. Further advance depends on the identification of interneurons for the elaboration of flow charts which summarize interactions between actual components.

This work was completed while one of the authors (G.A.H.) was Visiting Fellow to Balliol College, Oxford, and enjoyed hospitality at the Department of Zoology, Oxford University.

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