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GAIN CHANGES IN SENSORIMOTOR PATHWAYS OF THE LOCUST LEG

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Summary

Feedback systems that control the leg joints of animals must be highly flexible in adapting to different behavioural tasks. One manifestation of such flexibility is changes in the gain of joint control networks. The femur–tibia (FT) control network of the locust leg is one of the feedback systems most thoroughly studied with regard to its neural circuitry. Despite excellent information concerning network topology, however, actual gain changes and their underlying mechanisms have not yet been examined because of the marked spontaneous variations in the action of the control network for this joint. We describe a behavioural situation and a preparation in which the locust (*Locusta migratoria* L.) FT control network exhibits reproducible changes in gain, allowing investigation of the neuronal basis of gain control. After ('fictive') flight motor activity, the gain of resistance reflexes in the FT joint of the locust middle leg is significantly decreased, with the flexor tibiae muscles being affected more strongly than the extensor muscles. Immediately after flight motor activity, the gain may be as low as 30 % of pre-flight levels. It returns to pre-flight values in under 150 s.

The decrease in gain following flight motor activity is due to a decrease in motoneurone recruitment in the resistance reflex elicited by stimulation of the appropriate mechanoreceptor, the femoral chordotonal organ. Motoneurone recruitment is changed as a result of a drastic decline in the stimulus-related synaptic input to the motoneurones, which appears to be produced exclusively at the level of the pre-motor network. Two factors led to this conclusion: first, we found no indication of changes in membrane potential or membrane conductance of the tibia flexor and extensor motoneurones; second, recording from identified pre-motor nonspiking interneurones demonstrated that these may be involved in the observed gain changes. The putative behavioural relevance is discussed.

Key words: locust, *Locusta migratoria*, gain control, joint control, insect, locomotion.

Introduction

Vertebrate and invertebrate animals using legs for locomotion require subtle and versatile control over the movements of their jointed appendages. In adapting to different tasks, such as standing or walking, the neuronal networks controlling the individual leg joints exhibit marked flexibility; for instance, in the (reflex) motor output generated in response to a given proprioceptive signal, such as that monitoring joint movement (for reviews, see Bässler, 1983*a*; Pearson, 1993; Prochazka, 1989). These adaptive changes span a broad range, from subtle fluctuations in reflex gain when the joint control networks of the standing animal work in the 'postural maintenance mode' to complete reversal of the motor output (i.e. inversion of gain) when these networks are in the mode of 'control and reinforcement of ongoing movement' during locomotion (for a review, see Pearson, 1995). Within the past decade, neuronal mechanisms which might account for the changes in gain observed under different behavioural conditions have been investigated in a variety of systems. In the case of the reflex reversals generated by the joint control networks of many vertebrates (e.g. Forssberg *et al.* 1975; Pearson and Collins, 1993) and invertebrates (e.g. Bässler, 1976; DiCaprio and Clarac, 1981; Wolf, 1992) during locomotion, the analysis has reached a level of considerable detail. In the stick insect and the cat, for example, it is known that such reflex reversal is mediated by the strengthening of particular interneuronal pathways from proprioceptors to leg motoneurones (cat, McCrea *et al.* 1995; Gossard *et al.* 1994; stick insect, Bässler and Büschges, 1990; Driesang and Büschges, 1996). Presynaptic inhibition of sensory afferent terminals by central commands is known to contribute to this strengthening of selected neuronal pathways (e.g. El Manira *et al*. 1991; Gossard *et al.* 1990).

The situation is different, and apparently more complicated, for the subtle adjustment of motor responses, such as the changes in the gain of sensorimotor pathways which contribute to the adjustment of joint control network action in the

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'postural maintenance mode' of the standing animal (Bässler, 1993; Prochazka, 1989). The network topology of these control systems is well known in many invertebrates (for reviews, see Bässler, 1993; Burrows, 1994; Büschges, 1995; Wolf, 1995), but it is still unclear how changes in gain are brought about. This is true despite the fact that a number of candidate neuronal mechanisms have been described which could affect the gain in joint control networks (Burrows and Matheson, 1994; Laurent and Burrows, 1989; Büschges and Schmitz, 1991). In most cases, the possible contributions of these mechanisms to gain control were inferred from the fact that they were investigated in the appropriate sets of neurones, rather than from observation and manipulation of gain changes in functional networks. In summary, then, little is yet known about the neuronal mechanisms that are actually responsible for the control of reflex gain (Bässler, 1993; Bässler and Nothof, 1994; Kittmann, 1991), and investigation of these mechanisms has just begun (Bässler and Stein, 1996; Stein and Sauer, 1995). This also holds true for the question of how gain changes are mediated at the motoneuronal level. With regard to the motoneurones, gain changes might be mediated by shifting the membrane potential towards or away from spike threshold, a phenomenon reported to occur during habituation in insect sensorimotor pathways (Bässler, 1983*b*). Shifting towards spike threshold is, however, likely to increase the excitability of a motoneurone, thereby increasing the reflex motor output and, consequently, the reflex gain. Shifting away from the threshold could decrease both motoneuronal excitability and reflex gain. Adjustment of gain might also be accomplished by changes in the amplitude of stimulus-related synaptic input from joint proprioceptors to motoneurones. A given synaptic input might change in amplitude because of changes in the input (membrane) resistance of the motoneurones (e.g. Laurent and Burrows, 1989) or because of changes in the relative weighting of parallel excitatory and inhibitory interneuronal pathways converging onto the motoneurones (e.g. Büschges, 1990).

In the present paper, we examine the neuronal mechanism of gain control for a sensorimotor feedback system of the locust middle leg, the neuronal network controlling the femur–tibia (FT) joint. The layout and response characteristics of this network have been studied extensively in locusts and other orthopteran insects (e.g. Burrows *et al.* 1988; Büschges and Wolf, 1995). Despite the considerable overall variability of the motor output of the locust joint control system (Bässler, 1992), we were able to identify and describe a behavioural situation that is associated with reproducible changes in gain, thus enabling a study of neuronal mechanisms that contribute to gain changes. The gain of the FT joint control network transiently decreases after ('fictive') flight motor activity, and the neuronal mechanisms underlying these gain changes affect the amplitude of stimulus-related synaptic input to the motoneurones. That is, gain changes are brought about primarily, if not exclusively, at neuronal levels presynaptic to the motoneurones. Sample recordings from identified nonspiking interneurones substantiate this finding.

Materials and methods

Experiments were carried out under daylight conditions at room temperature (20–22 °C) on adult locusts (*Locusta migratoria* L.) of either gender. Locusts were raised in the animal facilities of the Universities of Kaiserslautern and Konstanz.

Preparation and recordings

We used a dorsal locust preparation adapted from Robertson and Pearson (1982) for our experiments, modified with respect to the presence and the means of immobilization of one middle leg. The wings and legs, except the right middle leg, of a locust were severed and the animal was pinned dorsal side up to a foam platform using minuten pins. The thorax was opened by a dorsal midline incision and the gut, fatty tissue, tracheal sacs and muscles dorsal to the thoracic ganglia were removed to expose the ventral nerve cord. Meso- and metathoracic ganglia were supported on a steel platform for intracellular recording from neurones in the central nervous system. The dorsal halves of the thoracic pleurae, including the dorso-ventral flight muscles, were sometimes removed to allow better simultaneous access to thoracic ganglia and middle leg. Flight motor activity was recorded by electromyogram (EMG) electrodes in the metathoracic depressor muscle 127 (Snodgrass, 1929). Flight motor activity was initiated by blowing air onto the head of the animal; sequences of 'fictive' flight elicited in this way lasted approximately 1–20 s.

The right middle leg was glued to the foam platform with dental cement (ESPE), with the anterior-dorsal surface of the femur uppermost. The femur–tibia (FT) joint was adjusted to an angle of about 120 ° before the cement was cured. A flap of cuticle was removed from the anterior surface of the femur to expose the receptor apodeme of the femoral chordotonal organ. The apodeme was inserted into a stimulation clamp close to the FT joint and cut distally. Ramp-and-hold stimuli with amplitudes of 600 μm were applied, corresponding to changes in joint angle of 40 ° (Field and Pflüger, 1989) or joint movement through a range from 120 to 80 °. Activity of the flexor and extensor tibiae muscles was monitored by means of EMG electrodes in muscles 107 and 106 (Snodgrass, 1929). Force was measured by attaching a force-displacement transducer (Grass FTO3C) to the tibia (approximately 3 mm distal to the FT joint) which, in the range used, measured forces linearly (see also Wolf, 1990). With this arrangement, the force measured by the transducer is the net force acting at the FT joint produced by the antagonistic muscles of the joint, i.e. the flexor and the extensor tibiae. As a consequence of this arrangement, an increase in force measured in one direction does not necessarily derive from an increase in the activity of the joint muscle pulling in this direction, but can also be due to a decrease of force in its antagonist (for a detailed discussion on this aspect, see Bässler and Stein, 1996).

Intracellular recordings from pre-motor interneurones and motoneurones were performed in the ipsilateral mesothoracic neuropile (details described in Büschges and Wolf, 1995). Glass microelectrodes were filled with solutions of 0.05 mol l^{-1}

KCl/2 mol l^{-1} potassium acetate (most motoneurone recordings) or 5% Lucifer Yellow in distilled water (interneurone recordings) and had tip resistances of 30–60 MΩ. Injection of the dye by hyperpolarizing current (2–10 nA for up to 10 min) upon completion of electrophysiological tests allowed identification of the penetrated interneurones by combined morphological and physiological criteria (Büschges and Wolf, 1995). Interneurones were named according to Büschges (1990). Tibial motoneurones were identified according to their well-known responses to femoral chordotonal organ (fCO) stimulation (e.g. Field and Burrows, 1982; Burrows *et al.* 1988) and by the one-to-one correlation of their intracellularly recorded action potentials with extracellularly recorded muscle potentials (EJPs) in the EMG recordings. Relative changes in the input conductance of motoneurones were investigated under bridge mode conditions, injecting hyperpolarizing constant-current pulses of 1–5 nA amplitude and 10–100 ms duration into the penetrated neurone (see also Wolf and Burrows, 1995).

The statistical significance of differences between means was assessed by a modified *t*-test, according to Dixon and Massey (1969). Samples were regarded as significantly different for $P<0.02$.

Results

Gain changes in the femur–tibia control network of the locust middle leg

In the inactive, quiescent locust, stimulation of the femoral chordotonal organ (fCO), the mechanoreceptor monitoring movement of the femur–tibia joint, resulted in resistance reflexes of the flexor and extensor tibiae muscles (Ebner and Bässler, 1978; Field and Burrows, 1982), as in other orthopteran insect species (Bässler, 1974, 1993). Owing to its position and attachment (Fig. 1A), elongation of the fCO signals flexion of the FT joint and elicits both reflex activation of tibia extensor motoneurones and inactivation of tibia flexor motoneurones. Relaxation of the fCO signals extension of the FT joint and induces the opposite motor effects. Fig. 1B illustrates the forces generated by this resistance reflex in our

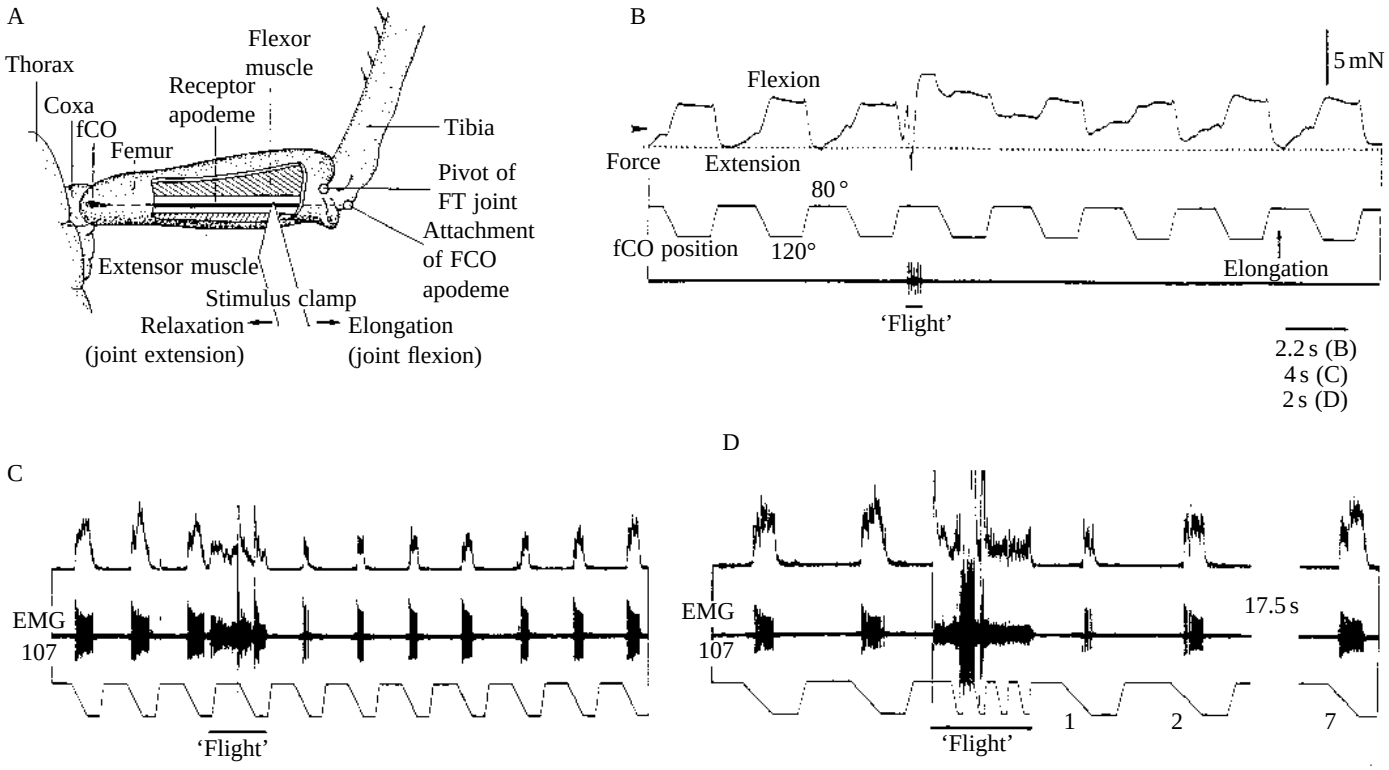

Fig. 1. Gain of the femur–tibia (FT) control network of the locust middle leg is decreased after flight motor activity. (A) Schematic drawing of the experimental arrangement for stimulating the femoral chordotonal organ (fCO) of the locust middle leg. (B) Recording of the net force (zero level is indicated by an arrowhead) generated at the femur–tibia joint of the right middle leg (top trace) in response to stimulation of the fCO with ramp-and-hold stimuli of 40° amplitude (fCO elongation, indicated by upward deflection of the position trace, mimics leg flexion and elicits reflex activation of the extensor muscle). Flight motor activity of the locust preparation was monitored with an electromyogram (EMG 127, bottom trace) from the metathoracic first basalar muscle (127). For details of the force measurement, see Materials and methods. (C) Electromyographic recording of tibia flexor activity (EMG 107) before, during and after 'fictive' flight. The top trace shows the rectified and integrated (τ=100 ms) EMG signal, which is shown as an original recording in the middle trace. Stimulus trace (bottom) as in B. (D) Sample recording from a different animal illustrating the decreased gain of the resistance reflex after an unusually long sequence of 'fictive' flight. The time scale is compressed fivefold during the flight motor episode; ramp stimuli delivered after the termination of 'fictive' flight are numbered consecutively below the stimulus trace.

experimental situation (‘open-loop’ conditions because of the immobilized leg and transected receptor apodeme) in response to ramp-and-hold stimulation of the fCO.

Touching the experimental animal at various points (e.g. head, antennae, abdomen) with a small paintbrush either increased or decreased the gain of the resistance reflex in an unpredictable manner. This observation is in agreement with previous results (Bässler, 1992). The same was true when active leg movements were elicited by a tactile stimulus. These could be detected by vigorous movement of the coxal stumps of the severed legs and in the EMG recordings from the immobilized middle leg. The gain of the FT feedback system changed reproducibly in only one behavioural situation. After flight motor activity had been elicited by a wind puff directed onto the animal’s head or by tactile stimulation, the gain of the resistance reflex was always decreased compared with pre-flight levels. This was evident in force measurements, such as the sample recording shown in Fig. 1B, where the force changes observed in response to both elongation and relaxation of the fCO were markedly decreased immediately after ‘fictive’ flight (see Materials and methods). For force measured at the tibia, the decrease in gain was often as large as 70 % (Fig. 1B). The gain of the resistance reflex returned to pre-flight levels within 8–150 s after flight motor activity (Fig. 1B,D). The time required for recovery was apparently not related to the duration of the preceding flight motor episode. During flight motor activity, the force output fluctuated between high values of flexion or extension and appeared not to be correlated with either fCO movement or fCO position. These latter findings also held true when muscle (sample recordings in Fig. 1C,D) or motoneurone activity (sample recordings in Figs 3B, 4B) was examined.

The observed decrease in gain of resistance reflex activation in the FT joint occurring with flight motor activity was associated with a decrease in muscle activation as revealed by EMG recordings from the tibia flexor (Figs 1C,D, 2A) and tibia extensor muscles. Monitoring activity of the tibia flexor muscle (N=5 animals, N=40 trials), for instance, revealed that the strength – that is, the gain – of reflex-induced motor activity was reduced after ‘fictive’ flight. This decrease was always more pronounced in the flexor branch of the joint control network than in the extensor branch (sample recording from

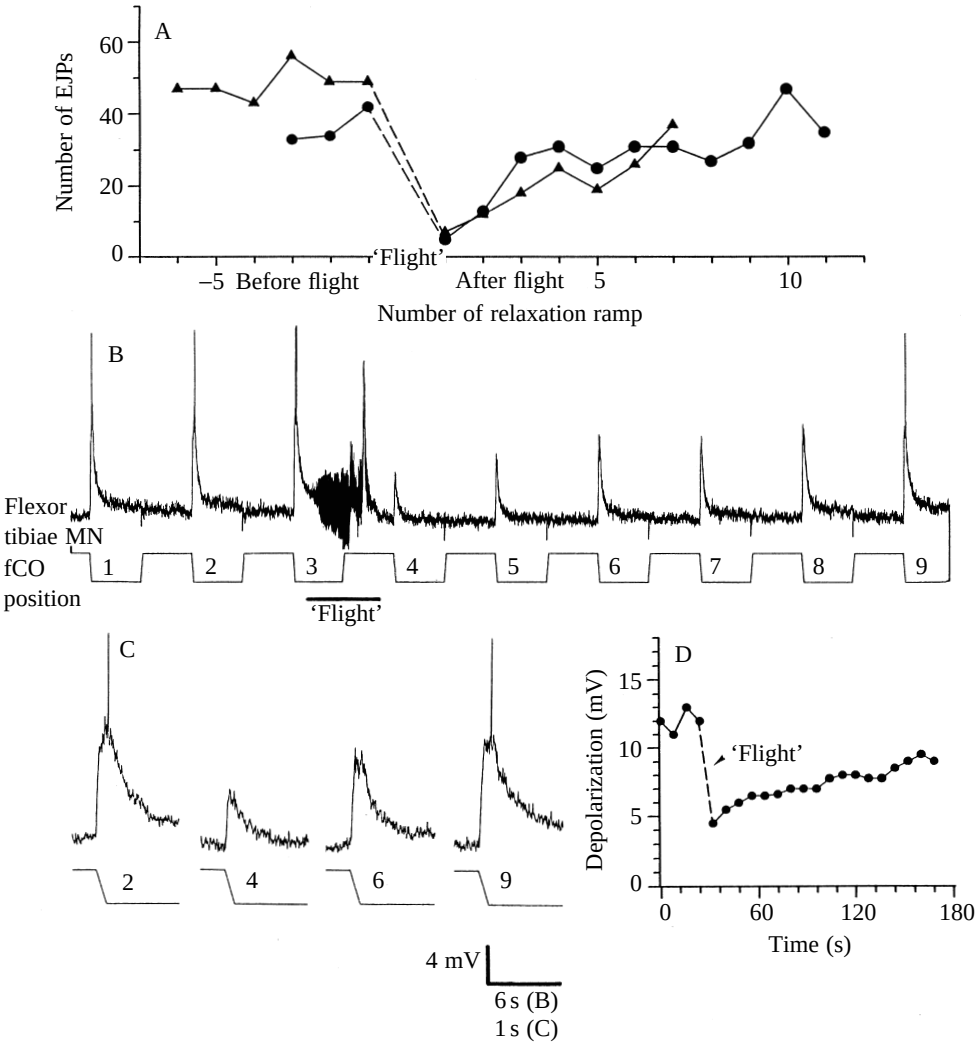

Fig. 2. The response of flexor tibiae motoneurones to fCO stimulation is altered after flight motor activity. (A) The change in the number of extracellular flexor muscle potentials (EJPs) generated per relaxation stimulus (stimulus regimen as in Fig. 1) as a function of the stimulus number after the end of flight motor activity (negative numbers are before the onset of ‘fictive’ flight); circles and triangles are for data from two different animals. (B) Intracellular recording from a fast flexor tibiae motoneurone (MN), with continuous stimulation of the fCO. A brief flight motor episode was elicited, after which the depolarization evoked by fCO relaxation was markedly decreased. Depolarization amplitude recovered over the following 60 s. fCO stimuli (relaxation) are numbered consecutively in this recording; motoneurone responses to stimuli 2, 4, 6 and 8 are shown on an expanded time scale in C for more detailed comparison. (D) Change in depolarization amplitude of the flexor motoneurone (taken from the experiment in B) as a function of time; flight motor activity was elicited between 20 and 30 s. Data are from a different flight episode.

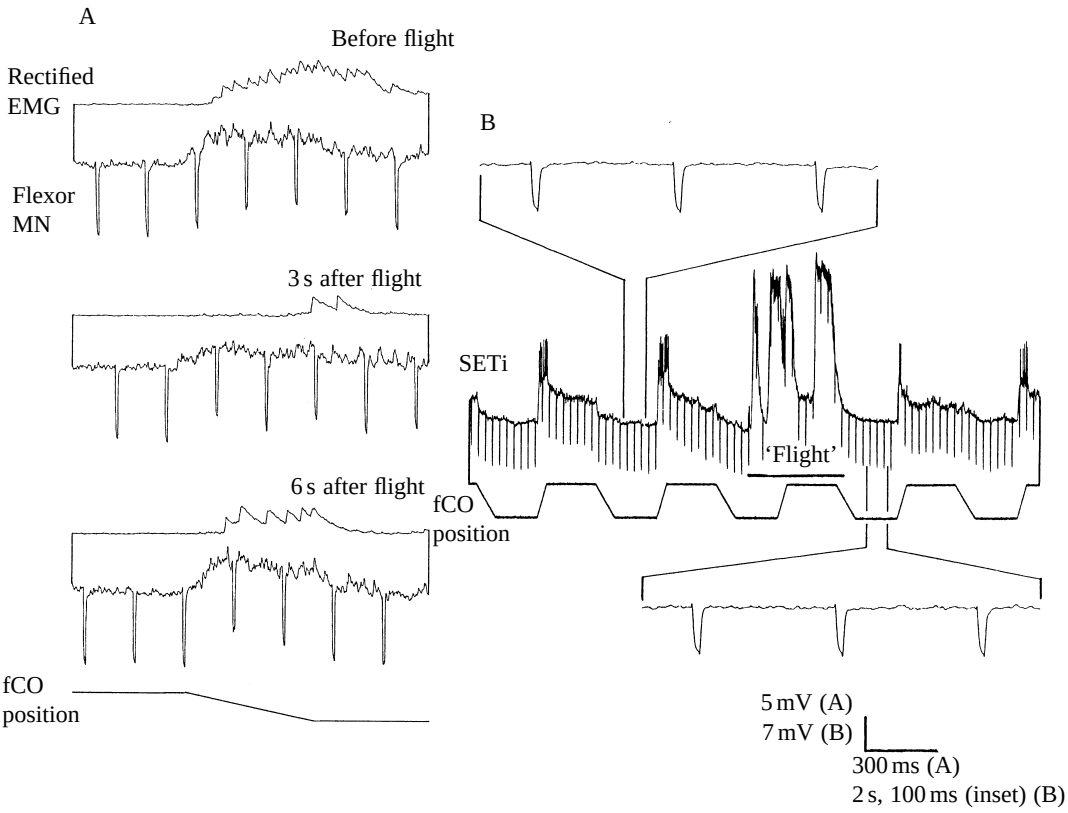

Fig. 3. Tibia motoneurones do not change their membrane conductance in the context of flight motor activity. (A) Intracellular recording of a fast flexor tibiae motoneurone (MN); the responses to three fCO relaxation stimuli (bottom trace, valid for all three sample recordings) are shown. Changes in input resistance of the motoneurone were determined by the injection of hyperpolarizing current pulses (approximately –3 nA, 10 ms duration). The first ramp stimulus was given immediately before the start of flight motor activity, the other two stimuli were delivered 3 and 6 s after the end of the flight motor episode. The upper traces show the respective rectified and integrated electromyogram (EMG) signals recorded from the flexor tibiae muscle 107 (as described for Fig. 1C,D). The amplitude of the voltage deflections produced by the current pulses before fCO stimulation does not change during the experiment, indicating that there is no change in the input resistance of the motoneurone. (B) The same type of experiment was carried out for the slow extensor tibiae motoneurone (SETi). Two recording segments, one sampled before and the other just after flight motor activity, are shown on an expanded time scale to illustrate the constant amplitude of the voltage deflections.

flexor motoneurone, Fig. 3A, from extensor motoneurone, Fig. 3B). The reflex response was never observed to remain constant, or to increase, after flight motor activity.

Gain changes in the FT control network are based on changes in the stimulus-related synaptic inputs to motoneurones

Two factors were responsible for the decrease in gain of the FT resistance reflex observed after flight motor activity. (i) The number of motoneurones recruited (i.e. excited above spike threshold) in response to a given stimulus decreased, and (ii) if a particular motoneurone was recruited, its response amplitude (i.e. the spike discharge per stimulus) was decreased. Both effects were caused by a decline in the stimulus-related synaptic input elicited in the motoneurones by fCO stimulation (Fig. 2). This is illustrated for a 'fast' flexor tibiae motoneurone (Fig. 2B–D). Immediately after 'fictive' flight, the depolarizations evoked in this motoneurone by fCO relaxation were reduced in amplitude by more than 60 % (see Fig. 3A for an example of a recording from another flexor tibiae motoneurone; amplitude reduction was approximately 45 % in this neurone). Over time, the amplitude of the depolarizing synaptic inputs returned to pre-flight levels, as did the spike discharge of the motoneurones. No significant tonic shift in the resting membrane potential of the motoneurones was observed, and particularly not in correlation with flight motor activity (Figs 2B, 3B). In five flexor motoneurone recordings, yielding a total of 20 flight motor sequences, an average change in motoneurone membrane potential of –0.13±1.01 mV (mean ± S.D.) was measured, the more hyperpolarized values occurring after flight motor activity. These changes in membrane potential were not statistically significant (P>0.2).

It is evident from these results that the amplitude of stimulus-related synaptic inputs to the motoneurones determines the strength of the motor output of the FT joint control network and, thus, its gain. Two mechanisms might account for changes in the amplitude of the synaptic input to motoneurones: (i) the drive the motoneurones receive from presynaptic interneurones and sensory neurones might change, owing to altered activation of the interneurone network or presynaptic inhibition (e.g. Burrows and Matheson, 1994), or (ii) the amplitude of synaptic potentials might change as a result of changes in the input resistance of the motoneurone; for instance, because of a tonic inhibitory drive that is adjusted according to the required gain of the feedback system. To decide which of these two hypotheses is correct, we injected short pulses of hyperpolarizing current into the flexor and

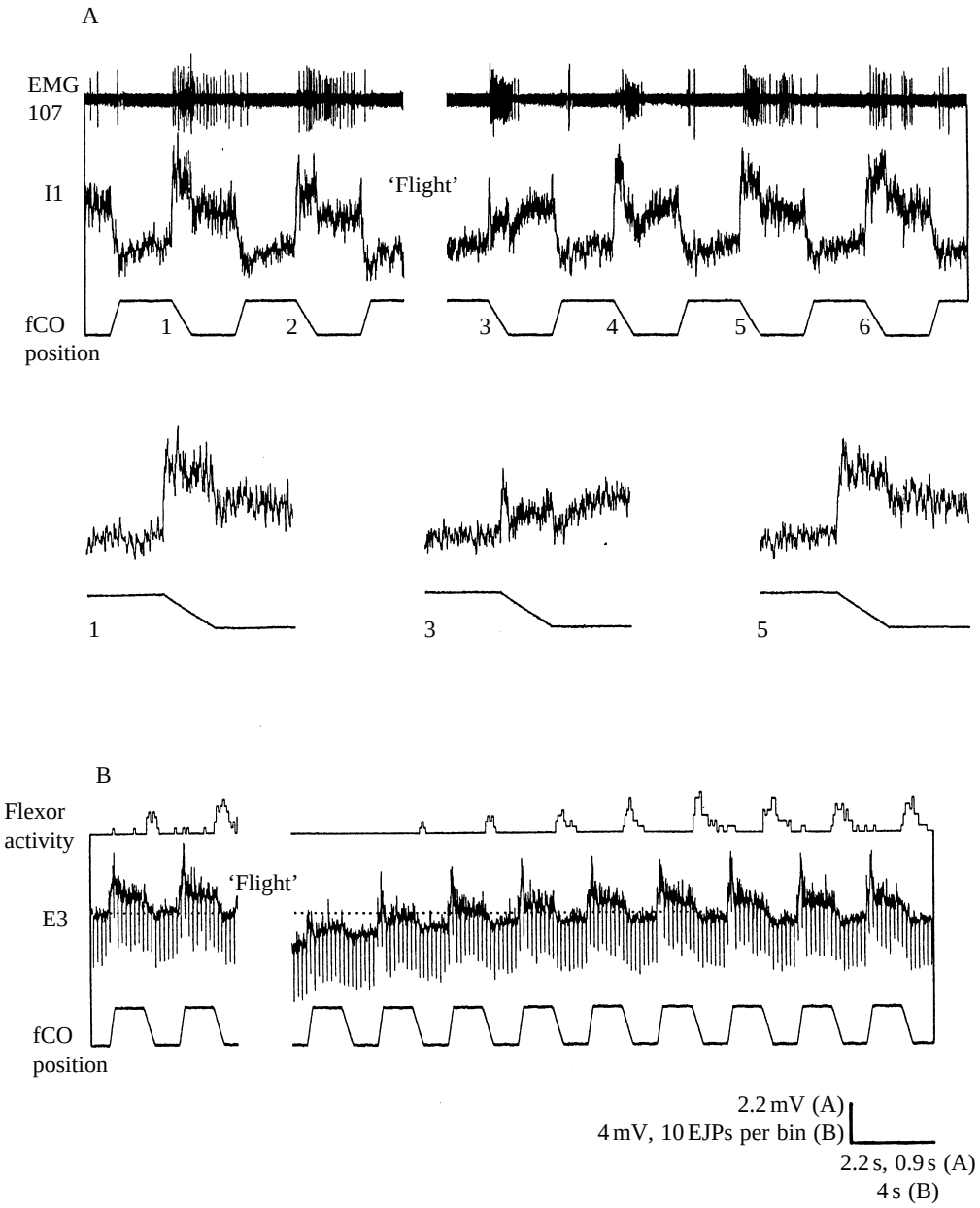

Fig. 4. The response of local nonspiking interneurones to fCO stimulation may change in conjunction with flight motor activity. Both types of interneurone are established elements of the middle leg femur–tibia control network (see Büschges and Wolf, 1995). (A) The depolarizing response of interneurone I1 to fCO relaxation was strongly reduced immediately after flight motor activity. Relaxation stimuli are numbered, and the responses to three selected stimuli, 3 s before and 500 ms and 7700 ms after the flight episode, are shown on an expanded time scale below the original recording. Tibia flexor muscle activity is monitored by an EMG recording (EMG 107). (B) The response of interneurone E3 to fCO elongation changed after flight motor activity. First, the depolarization generated in response to an elongation stimulus was reduced in amplitude (as, apparently, was the position-dependent change in membrane potential) and, second, the membrane potential of the neurone was shifted to more negative values, recovering within approximately 10 s. For monitoring changes in gain of reflex activation, tibia flexor muscle activity is given by a peristimulus time histogram with a bin width of 200 ms. The dotted line gives the resting membrane potential in interneurone E3 before flight motor activity. The brief negative deflections in the voltage trace are caused by hyperpolarizing current injection (compare Fig. 3).

extensor tibiae motoneurones, while recording from neuropilar processes of the motoneurones. Alterations in their input resistance can be detected by changes in the amplitude of the resulting voltage deflections, because the input resistance is proportional to the change in membrane potential caused by injection of constant-current hyperpolarizing pulses (Fig. 3). In none of our recordings (N=5 flexor and N=2 extensor motoneurones) did we observe any consistent changes in the input resistance of the neurone, as determined by the amplitude of the voltage deflections produced by current injection before and after flight motor activity. The same was true for the period during which the amplitude of the synaptic potentials recovered to pre-flight levels. Since there is no obvious change in the membrane conductance of the leg motoneurones in the context of flight motor activity, the change in the amplitude of the synaptic inputs must be brought about at the level of the pre-motor network connecting the fCO to the motoneurones.

The role of the pre-motor network in gain changes

It is known that, in orthopteran insects, sensory signals from the fCO are transmitted to the leg motoneurones by direct (i.e. monosynaptic) connections from the afferents to the motoneurones (Burrows, 1987) and by parallel polysynaptic pathways including local spiking and nonspiking interneurones (Burrows *et al.* 1988; Büschges, 1989, 1990; for summaries, see Burrows, 1994; Büschges, 1995). In both the locust and the stick insect, 11 types of pre-motor interneurones have so far been identified that provide parallel synaptic drive to the tibia motoneurones (stick insect, Büschges, 1990; Sauer *et al.* 1996; locust, Büschges and Wolf, 1995). Which of these pathways

connecting the fCO to the motoneurones contributes to the gain changes observed in the FT feedback system? In a first attempt to address this question, we recorded from identified pre-motor interneurones in the FT control network while eliciting flight motor activity and the associated changes in reflex gain. In contrast to the situation in the motoneurones, we observed both shifts in the resting membrane potential of the interneurones and changes in the amplitude of stimulus-related synaptic inputs when gain changes were induced by 'fictive' flight. This is exemplified in Fig. 4 by recordings from local nonspiking interneurones of type I1 and type E3 that are both known to transmit sensory information from the fCO onto tibia motoneurones (Büschges and Wolf, 1995). The amplitude of stimulus-related synaptic input to interneurone I1 was markedly decreased following flight motor activity, while the 'resting' membrane potential remained constant (Fig. 4A). This is most clearly seen in the depolarizing response of interneurone I1 to fCO relaxation. In the case of interneurone E3, the depolarizations recorded in response to fCO elongation were decreased after flight motor activity and, in addition, there was a transient hyperpolarization of the neurone's membrane potential lasting approximately 10–12 s (Fig. 4B).

Discussion

The main conclusion of this study is that the femur–tibia (FT) control network of the locust middle leg shows reliable changes in gain after flight motor activity. The resistance reflex generated in quiescent, inactive animals is decreased after fictive flight by up to 70 % of the force output and recovers to pre-flight values within 8–150 s (Figs 1A, 2). The FT control system of the locust thus provides an opportunity to study neuronal mechanisms of gain control under behaviourally relevant and reproducible conditions. This is particularly interesting in view of the variability of joint control properties that has been reported previously for the locust (Ebner and Bässler, 1978; Bässler, 1992).

The decrease in the gain of resistance reflexes associated with 'fictive' flight might function in the context of landing, for instance, if the stiffening of the leg joints by powerful resistance reflexes should conflict with the need for softening the impact of touchdown or grasping the substratum. Preliminary observations indicate, however, that the decrease in gain we observed after 'fictive' flight may be cut short or even abolished as soon as the locust has tarsal contact with the ground. The situation in our immobilized and dissected preparation, with five legs severed and the remaining one cast into epoxy resin, may therefore merely produce an artificially extended flight situation after rhythmic motor activity has ceased, corresponding perhaps to gliding. Although these behavioural aspects certainly need further attention, they do not impair the merit of the preparation for the analysis of neuronal mechanisms of gain control.

We began our analysis of the neuronal mechanisms contributing to gain changes in the FT feedback loop by examining the role of the motoneurones. It is evident from the experiments shown in Fig. 2B,C that changes in amplitude of stimulus-related synaptic input to the motoneurones of the tibia muscles contribute to the observed changes in motor output and reflex gain. We were unable to detect any contribution of motoneurone properties, such as changes in resting membrane potential or in input conductance. In concurrence with this result, it was possible to demonstrate that interneuronal pathways that mediate resistance reflexes in the FT control network are involved in eliciting the gain changes observed after 'fictive' flight.

The results outlined above raise the question of the neuronal mechanisms that effect the gain changes at the level of the pre-motor network. Previous investigations on joint control networks suggest four different mechanisms, which are not mutually exclusive. (i) Presynaptic inhibition of sensory afferents has been reported in a variety of proprioceptive feedback systems, including those in vertebrates (e.g. Rudomin *et al.* 1983) and crustaceans (e.g. Cattaert *et al.* 1992). For insects, in particular, a role for the inhibitory potentials recorded in afferent terminals of proprioceptive neurones has been discussed in connection with gain control (e.g. Burrows and Laurent, 1993; Burrows and Matheson, 1994), both in the quiescent, standing insect and during leg movement (Wolf and Burrows, 1995; Hedwig and Burrows, 1996). Tonic presynaptic inhibition of fCO afferent terminals – regulated according to behavioural requirements – would indeed be able to influence the synaptic drive provided by the afferents to the motoneurones and interneurones of the FT control network. Such state-dependent regulation of the presynaptic inhibitory input to mechanosensory terminals has been described for the locust middle leg during walking (Wolf and Burrows, 1995) and for the hindleg during kicking (Hedwig and Burrows, 1996). Definite proof of a role for presynaptic inhibition in gain control of a joint control network, however, has not yet been obtained, and there are no data on the role of presynaptic inhibition during actual gain changes in a joint control network as described above. (ii) A change in the weighting of parallel excitatory and inhibitory pathways, which eventually converge onto the motoneurones, would provide another mechanism for gain control (Büschges and Wolf, 1995; Sauer *et al.* 1996; Kraft *et al.* 1996). For example, nonspiking pre-motor interneurones were shown to receive parallel excitatory and inhibitory synaptic potentials from velocity-sensitive neurones responding to elongation of the fCO (Sauer *et al.* 1995). Altered weighting of such antagonistic pathways would strongly affect the stimulus-related net synaptic input to tibia motoneurones. (iii) Changes in the relative efficacy of the synaptic contacts which motoneurones receive directly from fCO sensory neurones and from parallel interneuronal pathways may provide a third mechanism of gain control. Studies on the topology of the FT control network (Burrows *et al.* 1988; Büschges, 1990; Büschges and Wolf, 1995; summary in Burrows, 1994; Büschges, 1995) have demonstrated the existence of these parallel pathways, and it appears possible that a differential change in synaptic efficacy is brought about by the state-dependent release of neuromodulators from

neurosecretory cells in the segmental ganglion. Evidence suggesting a similar neuromodulatory mechanism was obtained for the transition from resistance reflex to assistance reflex in the FT control network of the stick insect (Büschges *et al.* 1993). Recent studies on the behavioural specificity of the spike discharge in a group of neurosecretory cells, i.e. the dorsal unpaired median (DUM) neurones (Ramirez and Orchard, 1990; Burrows and Pflüger, 1995), provide further support of this hypothesis. (iv) The role of alterations in sensory sensitivity of fCO afferents through the action of neuromodulators must also be considered. As has been shown for a variety of sensory systems, including the chordotonal organs of insects (Ramirez and Orchard, 1990; Ramirez *et al.* 1993), the responsiveness and sensitivity of sensory cells in proprioceptive sense organs can be strongly modulated by the action of neuromodulators, such as biogenic amines (e.g. octopamine). As mentioned above, these mechanisms are not mutually exclusive but may well act in concert to achieve the gain changes observed in sensorimotor pathways.

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