THE LOCUST JUMP

I. THE MOTOR PROGRAMME

BY W. J. HEITLER* AND M. BURROWS†

Department of Zoology, University of Oxford, and Department of Zoology,
University of Cambridge

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SUMMARY

A motor programme is described for defensive kicking in the locust which is also probably the programme for jumping. The method of analysis has been to make intracellular recordings from the somata of identified motorneurones which control the metathoracic tibiae while defensive kicks are made in response to tactile stimuli. Three stages are recognized in the programme.

1. Initial flexion of the tibiae results from the low spike thresholds of tibial flexor motorneurones to tactile stimulation of the body.

2. Co-contraction of flexor and extensor muscles follows in which flexor and extensor excitor motorneurones spike at high frequency for 300–600 ms. Flexor inhibitory motorneurones are inhibited. The flexor muscle thus holds the tibia flexed while the extensor muscle develops tension isometrically to the level required for a kick or jump.

3. Trigger activity terminates the co-contraction by inhibiting the flexor excitor motorneurones and simultaneously exciting the flexor inhibitors. This causes relaxation of the flexor muscle and allows the tibiae to extend. If the trigger activity does not occur, the jump or kick is aborted, and the tibiae remain flexed.

INTRODUCTION

The metathoracic legs of the locust are specialized for the production of rapid and forceful tibial extensions, used mainly in jumping and defensive kicking. This paper is the first of a series in which we have examined the neural and mechanical mechanisms involved in the control of these legs in defensive kicking. We have concentrated in particular upon the motorneurones innervating the flexor and extensor muscles of the tibia. It is these muscles which are the power producers and power controllers for a jump and kick.

Previous accounts of the action of the various motorneurones during a jump are confusing. Hoyle (1975) recorded intracellularly from motorneurone somata and describes a motor sequence which he calls 'jump-like' in which there is first a burst of fast flexor motorneurone spikes, followed by a burst of fast extensor spikes, but with no co-activation. Usherwood & Runion (1970) recorded extracellularly from the

* Present address: Department of Zoology, University of California, Berkeley, California 94720, U.S.A.
† Balfour Student, University of Cambridge.
axons of motorneurones in intact locusts and describe a jump in which the fast extensor motorneurone spiked only just before take-off, and a defensive kick in which this motorneurone did not spike at all. Before the jump the slow extensor and the flexor motorneurones spiked together so that there must have been a co-contraction of the 20–30% of fibres in the extensor muscle innervated by the slow motorneurone (Usherwood & Grundfest, 1965) and the flexor muscle. Godden (1969, 1975) recorded extracellularly from the tibial muscles and found co-contraction between extensors and flexors in some jumps and kicks, but in others the activity of fast flexor motor-neurones was absent.

Mechanical and energetic considerations indicate that the extensor muscle must contract considerably before the jump and that there must be a co-activation of flexor and extensor muscles. Measurements of femoral cuticular strain preceding a strong jump show that each extensor tibiae muscle develops a stress of 1.2–1.6 kg just before the moment of take-off (Bennet-Clark, 1975). This level of tension can only be developed by a tetanic contraction of the extensor muscle. In an experimentally induced tetanic contraction the force of the extensor muscle rises slowly to reach the maximum only after some 350 ms of stimulation at 100 Hz (Ewer, 1952; Bennet-Clark, 1975). This force would extend the leg prematurely were it not for the mechanical arrangement of the femoral–tibial joint. When the tibia is flexed, the flexor muscle enjoys a considerable mechanical advantage over the extensor muscle and can develop greater torque (Brown, 1967; Heitler, 1974). Therefore, during the period of presumed extensor muscle contraction preceding a jump or kick, the tibia could be locked in the flexed position if there were a simultaneous contraction of the flexor muscle.

In this paper we describe the motor programme for a kick, addressing two questions in particular. First, is the prediction made from mechanical and energetic considerations, of a co-contraction of flexor and extensor muscles before the kick, confirmed by the electrical activity recorded in the motorneurones innervating those muscles? Secondly, what is the mechanism that allows the rapid extension of the tibia? We will show by intracellular recording from the somata of identified motorneurones while the locust performs a defensive kick, that there is co-contraction, and that the final trigger for a kick is excitation of the flexor inhibitory motorneurones and inhibition of the flexor excitors. The advantage of the intracellular approach is that the sub-threshold synaptic inputs which underly the pattern of the motor output are revealed and unambiguous identification of the neurones is made possible. The latter is important because during periods of intense sensory and motor activity (such as a jump or kick) it can be impossible to interpret extracellular recordings of nerve or muscle activity in terms of single neurones. The obvious disadvantage is the restrained and dissected nature of the preparation required, and the restriction this places on the behavioural repertoire of the experimental animal. It is an assumption to extend the results obtained from restrained locusts performing a defensive kick to unrestrained locusts performing a jump, but we consider the assumption to be justified in as far as the sequence of actions performed by the individual tibiae appears to be the same in both behaviours.
MATERIALS AND METHODS

The Preparation

Experiments were performed at room temperature (21°C) using adult Schistocerca americana gregaria (formerly S. gregaria; Dirsh, 1974) of either sex or occasionally, as indicated in the text, S. nitens nitens (formerly S. vaga). Both species were obtained from crowded laboratory cultures. A similar preparation to that described by Hoyle & Burrows (1973) was used, in which the locust was restrained on its back. The metathoracic tibiae could move freely but only restricted movements of the other legs were possible. A diffuse light source was placed above the abdomen, and a photocell arranged anterior to the femur of each metathoracic leg registered tibial movement.

The locusts were persuaded to kick by tickling various body parts (e.g. the mouthparts, antennae, abdomen, etc.) with a brush. Visual and auditory stimuli, which are powerful releasers of jumping in the intact locust, were not used in these experiments.

Intracellular recordings were made from the somata of motorneurones using glass microelectrodes which were filled with 2 M potassium acetate and had resistances of 20–50 MΩ. Current could be injected into the motorneurones through the electrodes via a bridge circuit. Motorneurones were identified using criteria established by Hoyle & Burrows (1973). Extracellular recordings of muscle potentials were made by recording differentially across a pair of 50 μm diameter copper wires which were insulated up to the tips and implanted in the muscles. Most of the recordings were stored on magnetic tape and filmed after the experiment.

The motorneurones

The abbreviations used for the motorneurones are shown below. The positions of the cell bodies in the metathoracic ganglion are described in the references given.

<table>
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<tr>
<th>Motorneurone</th>
<th>Abbreviation</th>
<th>Reference</th>
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<tbody>
<tr>
<td>Fast extensor of the tibia</td>
<td>FETi</td>
<td>Burrows &amp; Hoyle, 1973</td>
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<tr>
<td>Slow extensor of the tibia</td>
<td>SETi</td>
<td></td>
</tr>
<tr>
<td>Anterior fast flexor of the tibia</td>
<td>AFFIti</td>
<td></td>
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<tr>
<td>Anterior intermediary flexor of the tibia</td>
<td>AIFIti</td>
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<td>Anterior slow flexor of the tibia</td>
<td>ASFIti</td>
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<tr>
<td>Posterior fast flexor of the tibia</td>
<td>PFFIti</td>
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<tr>
<td>Posterior intermediary flexor of the tibia</td>
<td>PIFIti</td>
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<td>Posterior slow flexor of the tibia</td>
<td>PSFIti</td>
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<tr>
<td>Lateral fast flexor of the tibia</td>
<td>LFFIti</td>
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<tr>
<td>Anterior inhibitor of the flexor of the tibia</td>
<td>AlnFIti*</td>
<td>Burrows &amp; Horridge, 1974</td>
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<tr>
<td>Posterior inhibitor of the flexor of the tibia</td>
<td>PInFIti*</td>
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* These neurones were called simply AI and PI by Burrows & Horridge (1974), because of the possibility that they innervate other muscles as well as the flexor tibiae. However, in this paper we are concerned solely with the flexor and extensor muscles. These inhibitors do not innervate the extensor muscle, and so they have been called anterior and posterior inhibitors of the flexor (AlnFIti and PInFIti).

RESULTS

The behaviour

Movements of the hind tibiae in freely moving locusts range in speed from slow extensions as in walking, through intermediate speeds as in hopping, to the highest velocities which produce a strong jump with large displacement of the locust.
Fig. 1. Activity of the fast extensor tibiae (FETi, upper traces) and a flexor tibiae (PFFITi, lower traces) motorneurones in slow movements of the tibia. (a) Slow flexions and extensions of the tibia. The flexor spikes, but EPSPs in the FETi are subthreshold, and the extensions are due only to slow extensor spikes (not monitored). (b) In more rapid movements the FETi spikes, but the activity of flexor and extensor motorneurones is still strictly reciprocal. (c) Touching the abdomen causes depolarization of the PFFITi, which spikes, and of the FETi in which the EPSPs are sub-threshold. Calibration: vertical (a) upper 16 mV, lower 8 mV, (b) upper 8 mV, lower 4 mV, (c) 6 mV; horizontal (a, b) 400 ms, (c) 200 ms.

A similar range of speeds was found in kicks performed by restrained locusts. The greatest velocities were always produced after complete tibial flexion and distortion of the femoral cuticle. We use the following definitions of the various movements: a strong kick is one preceded by a large distortion of the femoral cuticle; a weak kick is preceded by a small distortion; a slow extension is preceded by no perceptible cuticle distortion.

Excitatory motor activity

Slow flexions and extensions which resemble those that occur when the locust walks are produced by alternating groups of spikes in fast flexor motorneurones and the single slow extensor motorneurone. The only fast extensor motorneurone (FETi) is depolarized by excitatory post-synaptic potentials (EPSPs) when the antagonistic flexor is inhibited but does not itself spike (Fig. 1a). More rapid extensions occur when the FETi is depolarized beyond spike threshold, but the spiking of fast flexor and extensor motorneurones is still reciprocal (Fig. 1b). These movements are not powerful enough to be classed as kicks, but might produce a series of hops in the intact locust. Opposite synaptic inputs to flexors and extensors also occur in resistance reflexes if the tibia is forcibly flexed and extended (Burrows & Horridge, 1974). In contrast, stimuli which tend to make the locust jump or kick, such as a light mechanical stimulus to the mouthparts or abdomen, often produce a simultaneous excitation of the antagonist motorneurones (Fig. 1c). The individual EPSPs of the excitation are not precisely matched in the antagonist motorneurones, and therefore do not stem from
Motor programme of the locust jump

Fig. 2. The motor programme for defensive kicks. Intracellular activity in the fast extensor (upper traces) and anterior fast flexor (lower traces (a, b), middle trace (c)) and anterior slow flexor (lower traces (c, d)) during five defensive kicks. The co-contraction before the kick, and the re-flexion after the kick is seen in all records. (d) The initial flexion occurred before the beginning of the record shown and there is an unusually long pause before co-contraction. (e) There is no pause between the initial flexion and co-contraction. The increase in the frequency of flexor spikes following the first FETi spike can be clearly seen (open triangle). The trigger inhibition of the flexors preceding the extension is clear in (a—d) but absent in (e).

In all the figures of this paper an arrow has been used to indicate the deflection in the photocell trace at the moment of the kick (lower trace (e)). In some records the photocell trace has been removed, leaving the arrow alone to indicate the movement (a—d). Calibration: vertical (a, b and e) 30 mV (c, d) upper 40 mV, lower 16 mV.

the same pre-motor neurone. The flexor motorneurones spike more readily to these general excitatory stimuli than does the FETi (Fig. 1c; Burrows & Horridge, 1974), so that the first response of a walking locust to an arousal stimulus is to stop and flex its metathoracic tibiae (Hoyle, 1954). If the stimulus is strong enough, and the locust is in an excited state, this flexion becomes the first part of the motor programme for a jump or kick. The features that are consistently observed and constitute the excitatory motor programme for a kick are:

(1) Initial flexion of the tibia. This is caused by a burst of spikes in flexor motorneurones usually lasting 100–300 ms (Fig. 2a–c). The FETi is also depolarized (Fig. 2c), and occasionally spikes late in the flexor burst (Fig. 2a), but owing to the greater mechanical advantage of the flexor muscle when the tibia is nearly flexed (Heitler, 1974), these spikes do not produce an extension. After the initial flexion
there is often a pause of 100–200 ms before the next stage of the programme (Fig. 2a–c).

(2) **Co-contraction of flexor and extensor muscles.** Flexor and extensor motorneurones now spike concurrently for 300–600 ms. The FETi spikes with a maximum frequency of 60–100 Hz (Fig. 2a–e) and the slow extensor (SETi) at frequencies up to 130 Hz (Fig. 3a). The excitation causing the spikes is similar in both extensor motorneurones (Fig. 3b). The spikes in the flexor motorneurones may start before those in the FETi (Fig. 2b, c, e), or follow upon the first FETi spike (Fig. 2a, d), but there is always an increase in the frequency of the flexor spikes when the FETi itself spikes (Fig. 2e). The fast flexors spike at about 60 Hz (Fig. 2a, b), while the slow flexors can reach frequencies of 200 Hz (Fig. 2c, d). This stage has been termed the 'quiescent period' by Godden (1975), because there is no movement of the tibia from the flexed position. In view of the intense motor activity during this period, however, the term is inappropriate, and we will refer instead to the 'co-contraction' stage of the motor programme.

(3) **Rapid extension of the tibiae.** Co-contraction is terminated by inhibition of the flexor excitatory motorneurones. Common synaptic potentials are seen during the inhibition of at least the anterior (AFFITi) and posterior (PFFITi) fast flexors. The inhibition allows relaxation of the flexor muscle, and is followed 30–60 ms later by the rapid tibial extension of a kick (Fig. 2a–d). On only one occasion (of 100 observed kicks) has a kick been observed without a preceding hyperpolarization of the flexor that was penetrated (Fig. 2e). In this flexor the frequency of spikes was below normal and the last spike occurred at about the time before the movement when inhibition would have been expected. It must be emphasized that this kick was exceptional and we do not know whether inhibition was present in other flexors.
Abortive kicks, (a) An abortive kick is followed by a normal kick. (b, c) Abortive kicks. Note the absence of trigger inhibition of the AFFTfi (lower traces) in abortive kicks. The FETi activity (upper traces) is sometimes divided into two phases: an initial burst at high frequency very similar to a normal kick, followed by a slowly decelerating burst of spikes which is typical of abortive kicks. This is particularly clear in (b).

(4) Re-flexion of the tibia. Immediately after the kick the FETi is inhibited. There is often a further inhibition of the FETi about 200 ms later which is accompanied by excitation of flexor motorneurones. The spikes in the flexors cause the tibia to return to the flexed position (Fig. 2b, c, e).

The motor programme can be terminated at any of the stages outlined above. It is common for an arousing stimulus to cause the initial flexion without the completion of the rest of the programme. It can also be terminated after the co-contraction phase and the result has been called an abortive kick (Godden, 1975). Abortive kicks lack the inhibition of the flexor excitor motorneurones which occurs in normal kicks (Fig. 4). Without this inhibition the flexors continue to spike and the tibia remains firmly flexed. Therefore, the inhibition of the flexor excitor motorneurones and consequent relaxation of the flexor muscle is the final trigger which actually releases the kick. If this trigger does not occur then the co-activation of the antagonists continues with gradually declining spike frequencies (Fig. 4b, c).

Inhibitory motor activity

The anterior inhibitor of the flexor tibiae muscle (AIMFTi) is excited during the initial flexion before a kick and may spike a few times, but once co-contraction has started the inhibitor is itself inhibited (Fig. 5). This inhibition is particularly effective in the first part of the co-contraction when there are no AIMFTi spikes, but in the later part the inhibitor may start to spike again. Finally there is a rapid crescendo of spikes starting some 60 ms before the kick and reaching a peak frequency just before the movement. After the kick the AIMFTi may spike once or twice, but its membrane potential is quickly repolarized so that it undergoes a short period of inhibition (Fig. 5).

The posterior inhibitor (PIMFTi) is not so effectively inhibited during the co-contraction phase, but does receive a similar trigger activation to that of the anterior inhibitor. The only difference between the two inhibitors in a jump seems to be one of threshold; common EPSPs which are subthreshold in the anterior inhibitor often evoke spikes in the posterior inhibitor.
The burst of inhibitory spikes preceding the extension of the tibia should speed the relaxation of the flexor muscle. Therefore, the excitation of the flexor inhibitors acts in concert with the inhibition of the flexor excitor motoneurones and constitutes part of the trigger activity which allows extension of the tibia. The trigger activity occurs synchronously in a fast flexor excitor (LFFITi) and the anterior inhibitor (Fig. 6). There are several mechanisms which could ensure this reciprocal activity. For example, there could be an inhibitory connexion from the inhibitor to the flexor excitor motoneurones. There is no evidence for this because there are no IPSPs in the flexor excitors when the AInFlTi is caused to spike by the injection of depolarizing current. Other possibilities are reciprocal activation by one pre-motor neurone, or by several pre-motor neurones co-ordinated by higher centres. It is not possible to decide between these alternatives by precisely matching EPSPs with IPSPs in the motoneurones.

Whatever its origin, there is always close matching of the trigger activity in excitor and inhibitor motoneurones. This was illustrated clearly in one preparation in which a series of tibial extensions started as strong kicks but became progressively weaker (Fig. 7). In the first kick, despite the low frequency of FETi spikes the activity of the flexor motoneurone was normal and the AInFITi was inhibited during the co-contraction; typical trigger activity occurred in both flexor inhibitor and excitor motoneurones (Fig. 7a). In the next kick there were fewer FETi spikes, the burst of flexor motoneurone spikes was broken up by IPSPs, and the AInFITi spiked a few times during the period in which it is normally inhibited. The trigger activity, however, was still apparent in both motoneurones (Fig. 7b). In the final tibial extension the AInFITi spiked continuously and there was only a slight indication of trigger activity following the two or three FETi spikes. The LFFITi showed no sign of any
trigger inhibition, but there was a steady polarization of its membrane potential which began 150 ms before the first FETi spike (Fig. 7c).

**Co-ordination of the two hind tibiae**

The movements of the hind tibiae are usually synchronized to within a few milliseconds of each other when a locust jumps (Bennet-Clark, 1975). The synchrony between the two legs may gradually break down if the locust jumps repeatedly, so that sometimes only one leg is used, or one may merely be extended in mid-air. That the locust can produce jumps in which movements of the tibiae are well synchronized indicates that accurate mechanisms must exist for the co-ordination of the two legs.

In a kick the situation is somewhat different. Irritation of a particular region of a free-walking locust usually results in a well directed kick by one leg. If, however, the locust is immobilized on its back with only its hind tibiae free to move, its response to such a stimulus will often be a simultaneous kick with both legs. Repeated stimulation leads to a decrease in the synchrony between the two legs, but the co-ordination of the first few kicks is often as good as that which occurs in jumping.
Fig. 7. The trigger activity is a result of reciprocal inputs to flexor excitor and inhibitor motorneurones. (a–c) Three successive kicks of diminishing strength. The number of FETi spikes in the myogram (dotted spikes in lower traces) gives an estimate of the strength of each kick. The trigger activity in both the AlnFITi (upper traces) and the LFFITi (middle traces) is well defined in (a) (a fairly strong kick), less well defined in (b) (a weaker kick) and non-existent in (c) (a slow extension). There was no monitor of tibial movement in this experiment, but arrows have been used to show the estimated time of tibial extension. Calibration: vertical, upper 8 mV, middle 16 mV.

The moment at which extension of the tibiae starts in a kick depends upon the balance between the tension in the extensor and flexor muscles. The tension in the extensor muscle is largely determined by the spikes of the FETi, and so recordings have been made from the left and right FETi’s during a variety of kicks. There is no direct coupling between these motorneurones (Hoyle & Burrows, 1973), and there is no obvious phase relationship between their spikes, although the general pattern tends to be similar in the two (Godden, 1969). Sometimes the FETi spikes occur in groups which are similar in the two neurones (Fig. 8a). This is caused by the similarity of the inputs, but precise matching of individual PSPs is rare. Only the descending
Fig. 8. The activity of left and right FETi's in four double-leg kicks. (a) The spike frequency is similar in both neurones, and the tibiae move synchronously (arrow). (b) The two legs move synchronously in weak kicks, although the right FETi spikes more often than the left. Common EPSPs mediated by a descending movement detector interneurone can be seen before the burst of spikes (dots). (c) A double-leg kick in which the tibiae move synchronously despite the difference in spike patterns of the two neurones. (d) A double-leg kick in which the right tibia moves before the left, although the left FETi spikes more than the right. Calibration: vertical \((a, c, d) 16 \text{ mV}, (b) 30 \text{ mV}\).

movement detector interneurones have so far been shown to synapse on to both FETi's (Burrows & Rowell, 1973), and the amplitude of the EPSPs caused by those inputs is small in the somata, relative to the depolarization that occurs during a kick (Fig. 8b). The similarity in the waveform of the membrane potential is not always reflected in the spikes of the two motorneurones; a depolarizing wave occurring in both can evoke spikes in one and not the other (Fig. 8b). The timing of the movements
Fig. 9. The activity of the right anterior fast flexor (upper traces) and the left posterior fast flexor (and traces) during three double-leg kicks. (a) The left and right kicks are separated by about 50 ms (arrows). There is no inhibition of the flexor on one side when the flexor on the other side receives its trigger inhibition. (b) The tibiae extend simultaneously, but the trigger inhibition is very rapid, and no IPSPs can be identified on the falling waveform. A small section of the movement monitor has been included in this record (traces 3 and 4). (c) The tibiae extend simultaneously, but this time the trigger inhibition is not so rapid. (d) An expanded view of the dotted section in (c). No common IPSPs can be identified on the falling waveform. Calibration: vertical (a, b, c) 16 mV, (d) 8 mV; horizontal (a-c) 200 ms, (d) 50 ms.

of the tibiae does not bear any simple relation to the number or frequency of spikes in the FETI motorneurones. The FETI on one side may spike more often than its contralateral partner, and yet the tibiae move at the same time (Fig. 8c). Conversely, when the tibiae do not move synchronously it is not always the side on which the FETI has spiked most whose tibia moves first (Fig. 8d). There is no obvious change in the pattern of spikes, such as a sudden increase in frequency just before the movement, which could represent a decision in the FETI about the precise timing of the tibial movement.

These observations suggest that control of the spike patterns of FETI (and hence extensor muscle tension) does not play a major part in controlling the timing of the tibial movements. Therefore this timing is controlled by the relaxation of tension in the flexor muscle, which in turn is controlled by the trigger inputs to the flexor motorneurones. The simplest way of achieving synchronized relaxation of the flexor muscles on the two sides would be for the trigger activity to be common to the two sets of motorneurones. Simultaneous recordings from flexor motorneurones on the two sides of the locust during poorly-synchronized double-leg kicks usually show no sign of shared trigger inputs (Fig. 9a). The trigger inhibition of the flexor excitors in well-synchronized kicks is often so rapid that it is not possible to identify any synaptic potentials underlying it (Fig. 9b), but even when the inhibition is slower there is no
**Motor programme of the locust jump**

![Diagram showing motor programme](image)

**DISCUSSION**

**The motor programme**

The activity in motorneurones which controls the metathoracic tibiae while the locust performs a defensive kick is sufficiently stereotyped to justify the use of the term 'motor programme' (Fig. 10). The first stage of the programme is excitation of the flexor excitor motorneurones, which folds the tibia tightly against the femur thus ensuring that the flexor muscle has the maximum possible mechanical advantage over the extensor muscle. Fast and slow flexor and extensor motorneurones then spike at high frequencies for a period of 300–600 ms. During this period the anterior flexor inhibitor is itself inhibited and does not spike. The tension in the flexor muscle ensures that the tibia remains flexed while the extensor muscle develops tension. The only movement is a slight proximal and ventral shift of the tibia caused by distortion of the distal femoral cuticle. The co-contraction is terminated by the inhibition of the flexor excitor motorneurones and the simultaneous excitation of the flexor inhibitors. This is termed the trigger activity, because it releases the tibia from the flexed position. Immediately after the tibial extension of a kick, the two extensor motorneurones are...
inhibited. This usually is followed about 200 ms later by a further extensor inhibition coupled with flexor motorneurone excitation which causes re-flexion of the tibia.

The trigger activity

The trigger activity is the final determinant of whether or not the tibia will be extended rapidly in a kick. What is the source of this activity? One source could be a measure of tension in the femoral muscles. This does not seem to be the signal which evokes the trigger activity, firstly because abortive kicks, in which there is no trigger activity, can occur even when flexor and extensor motor activity, and hence presumably muscle tension, is as great as in some real kicks. Secondly, synchronized kicks by both legs can occur in which the number and frequency of FETi spikes, and hence presumably the tension, are very different in the two legs. Thirdly, in poorly synchronized kicks it is not always the leg with the greatest extensor motor activity that kicks first. If the trigger activity were to be a result of a peripheral reflex monitoring unilateral muscle tension, synchronized kicks should occur only when tension in the muscles on the two sides is identical; in poorly synchronized kicks the side with the greatest tension should kick first. This suggests that the timing of trigger activity is controlled by some, as yet undefined, central command. What form could this take? It could be a single trigger neurone with bilateral outputs. No common IPSPs can be identified in flexor motorneurones of the left and right sides during trigger inhibitions that occur simultaneously on the two sides. Furthermore, there is usually no indication of inhibition of the flexors on one side at the time of inhibition of flexors of the other leg that performs a single-leg kick. Therefore, co-ordination of the tibiae in synchronised double-leg kicks or in a jump is achieved either by two independent trigger networks each with a unilateral output, but coupled at higher levels, or by a non-spiking interneurone (cf. Pearson & Fourtner, 1975) with a bilateral output and which is separate from the trigger system used in poorly co-ordinated kicks. Clearly, the next stage is to identify these putative interneurones because little more can be inferred about the interneurones from recordings made in motorneurones.

Design principles for locust kicks and jumps

The chief requirements in the escape jump and defensive kick of the locust are first, great force delivered quickly (i.e. a high power output), and secondly, accurate timing. The first is achieved by the development of tension under almost isometric conditions in the power producing extensor muscle, and the second by the separation of power producing and power controlling functions. It is the massive extensor muscle that provides the energy for the movement, but the release of this energy is controlled by the relatively weak flexor muscle.

High power output

Muscles cannot contract with both high force and high velocity; a muscle operating at maximum power only produces about one-third of its potential isometric force (Hill, 1938). In the locust jump or kick the metathoracic tibiae are locked in a flexed position while the extensor muscles contract virtually isometrically, thus enabling the development of maximum force. The work done is then stored in the distortion of the mechanical elements of the legs (Bennet-Clark, 1975), and is retrieved when relaxation
Motor programme of the locust jump

of the flexor muscles releases the tibiae from their flexed position. Since the elastic energy stored in the cuticle strain does not suffer from the same velocity limitations as muscle, the work is retrieved quickly, and the tibiae move at high velocity. In a jump the energy is put into the system over about 500 ms, the duration of the extensor contraction, and is released over 25 ms, the duration of the extensor movement (Brown, 1967). Thus in a jump there is an average power amplification of about 20, and the power limitations of the muscles are overcome.

Separation of power production and control

There are two advantages to this. First, it allows maximum utilization of the power-producing muscle. The threshold of the mechanical lock is controlled by a separate muscle (the flexor tibiae) and can initially be set considerably above the maximum force capability of the power-producing muscle (the extensor tibiae). Once maximum force is achieved by the extensor muscle, the lock threshold is reduced by relaxation of the flexor until the tibia extends. Were there no separate control of the lock, the threshold would have to be set below the maximum force of the extensor, and a considerable safety margin would be necessary to ensure that under extreme conditions (such as fatigue, or a drop in temperature reducing the force capability of the muscle) the extensor could still overcome the fixed threshold of the lock. It is obviously advantageous, in an escape jump or defensive kick, always to be able to use the maximum force available. The separation of power production and control ensures that this is possible. The second function of this separation is to increase the precision of the timing of the movement. If the threshold of the lock could not be separately controlled, then the timing of the movement would be determined by the rate of rise of tension in the power-producing muscle. As tension approaches the maximum, this rate becomes relatively slow, and so there would be considerable variation in the time at which the tension crosses the threshold. By having the threshold controlled by a separate muscle, it can be arranged so that the threshold is crossed when the tension of the controlling muscle is changing very rapidly. The flexor tension at which the tibia extends with maximum extensor tension has been estimated to be about 15 g (Heitler, 1974). If the flexor muscle is stimulated electrically to produce maximum tension, and then allowed to relax, the 15 g threshold occurs during the rapid phase of declining tension (Bennet-Clark, 1975). When flexor inhibitor motorneurones are active, as in a defensive kick, the rate of decline of tension should be even more rapid. In this way the time at which the threshold is crossed can be very precisely controlled.

Principles in the production of rapid movements

The principles outlined above do not seem to be restricted only to locust jumping but occur, for example, in the prey capture strike of the mantis shrimp (Stomatopoda, Crustacea) (Burrows, 1969), the snap of the snapping shrimp (Alpheus heterochelis (Decapoda, Crustacea) (Ritzmann, 1974), the jump of the rabbit flea (Siphonaptera, Insecta) (Bennet-Clark & Lucey, 1967) and the jump of the click beetle (Coleoptera, Insecta) (Evans, 1972). Nevertheless, the only arthropod escape behaviour, besides that of the locust reported here, of which the neural control has been studied in detail is the crayfish tail flip. The principles underlying this behaviour are totally different. The locust produces an extremely rapid movement by using the mechanical system as
a power amplifier. The penalty it pays for this is the relatively long latency of the response: it takes about 500 ms to store the energy for the jump. The crayfish tail flip is less rapid but occurs with a very short latency. The lateral giant-fibres initiate the behaviour in response to a tactile stimulus with a latency of less than 10 ms, and the massive fast flexor muscles of the abdomen complete the flip within 70–80 ms (Wine & Krasne, 1972). The penalty in the crayfish is that the muscles work directly on the moving mechanical elements and contract rapidly, and therefore not with maximum force. Both animals produce effective escape behaviours. The locust is immobile for \( \frac{1}{2} \) s, and then produces a bodily displacement which is huge relative to body size. The crayfish responds quickly, but produces a displacement which is much smaller relative to its body size.

The significance of this difference can be understood by considering the different environments of the two animals. The crayfish is limited by the viscous drag of the water, which increases rapidly with increasing velocity. Therefore it is of little advantage to the crayfish to increase the velocity of the abdominal movements above a certain optimum, and so specialization has taken the form of reducing the latency. The locust escape jump is largely ballistic, and only marginally limited by the viscous drag of the air (Bennet-Clark, 1975). Therefore the greater the take-off velocity, the greater the displacement that can be achieved. Specialization in the locust has been to produce maximum take-off velocity.

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REFERENCES


Motor programme of the locust jump


