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Leg kinematics and muscle activity during treadmill running in the cockroach, *Blaberus discoidalis*: II. Fast running

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Abstract We have combined kinematic and electromyogram (EMG) analysis of running *Blaberus discoidalis* to examine how middle and hind leg kinematics vary with running speed and how the fast depressor coxa (Df) and fast extensor tibia (FETi) motor neurons affect kinematic parameters. In the range 2.5–10 Hz, *B. discoidalis* increases step frequency by altering the joint velocity and by reducing the time required for the transition from flexion to extension. For both Df and FETi the timing of recruitment coincides with the maximal frequency seen for the respective slow motor neurons. Df is first recruited at the beginning of coxa-femur (CF) extension. FETi is recruited in the latter half of femur-tibia (FT) extension during stance. Single muscle potentials produced by these fast motor neurons do not have pronounced effects on joint angular velocity during running. The transition from CF flexion to extension was abbreviated in those cycles with a Df potential occurring during the transition. One effect of Df activity during running may be to phase shift the beginning of joint extension so that the transition is sharpened. FETi is associated with greater FT extension at higher running speeds and may be necessary to overcome high joint torques at extended FT joint angles.

Key words Locomotion · Electromyograms · Kinematics · Fast motor neurons · Velocity

Abbreviations *EMG* electromyogram · *Df* fast depressor coxa motor neuron · *CF* coxa-femur · *FT* femur-tibia · *FETi* fast extensor tibia neuron · *T2* mesothoracic · *T3* metathoracic · *Ds* slow depressor coxa neuron · *SETi* slow extensor tibia neuron

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Introduction

Arthropod neuromuscular systems provide useful models in which to study problems involving control of locomotion. The legs of various arthropods are characterized by a pattern of few motor neurons supplying each muscle, with each motor neuron having markedly diverse properties. In all insects studied so far, femoral depressor and tibial extensor muscles that are used during stance are innervated by at most three axons (Pearson and Iles 1971; Burrows and Hoyle 1972; Wilson 1979). The triply innervated muscles receive a single slow excitatory axon, a single fast excitatory axon, and a common inhibitory axon shared with an undetermined number of other muscles. Nevertheless, the problems that must be overcome by the systems that control neuromuscular properties in arthropods are common to all legged animals: they must be able to adjust to load and smoothly change direction and speed of joint movement.

The characterization of the excitors as "fast" and "slow" denotes a wide difference in the muscle potential and muscle forces generated by each axon. Recordings of both muscle force (Usherwood 1962) and joint movements (Watson and Ritzmann 1995) in isolated or unloaded legs have consistently shown non-overlapping responses to slow versus fast motor neuron stimulation. Fast motor neurons produce brief, powerful muscle forces in response to each action potential causing an unloaded leg to undergo rapid joint movements in excess of 10°. Single action potentials in slow motor neurons are ineffective. Only high-frequency stimulation produces discernible muscle force and joint movement.

The effect of fast motor neurons on movement of unloaded legs, coupled with the fact that they are recruited during faster running movements, could imply that insects simply switch power plants to run faster. This notion presumes that selecting fast motor neurons and their associated fast muscle results in much more rapid joint movements than would be possible with slow

muscle alone. Such a notion must be tested against two considerations. First, fast motor neuron potentials occur against a background of high-frequency slow motor neuron potentials during running, and the actual contribution of each type of activity to movement may be more complex. To understand their contributions, one must examine the effects of differential recruitment of fast and slow motor neurons during actual running behavior when the animal's legs are being subjected to normal loading. Second, there are several distinct mechanisms which could produce variation in running speed. Therefore, the role of motor neuron activity should be considered in light of kinematics.

Given that motor activity directly affects joint angle movement, there are a finite number of parameters that could account for an animal's increase in running speed. First, the excursion of the joint can increase over the same period of time as the animal lengthens its stride. The second possibility is that the animal could increase stride frequency by either of two mechanisms while maintaining excursion constant. The velocity of joint movement during either flexion or extension could be increased so that the same excursion is covered in less time. In addition, the transition periods from flexion to extension or from extension to flexion could be shortened. A decrease in transition time would shift the next joint movement to an earlier start point resulting in a decrease in cycle time, even if the joint velocity during extension and flexion remained unchanged. Finally, it is possible that an increase in force at any or all joints could cause the animal to cover more ground without any change in such kinematic parameters as joint excursion or cycle time. This would only occur if the movement included an airborne phase. Given the variety of mechanisms by which faster running could be achieved, a complete understanding of neuromuscular control during locomotion requires an understanding of the exact role played by fast motor neuron recruitment.

The combined kinematic and electromyogram (EMG) analysis we have employed provides a detailed record of the joint kinematics associated with muscle activity patterns. High-speed (200 Hz) motion records allow precise characterization of the timing of motor neuron activation relative to footfall and joint extension. In the companion paper (Watson and Ritzmann 1997), we presented the relationships between slow motor neuron activity and joint kinematics. In this paper, we examine the changes in joint movements that account for increases in running speed over the range 2–10 Hz. This range of running speeds was chosen to include cycles with and without fast motor neuron activity. We could, therefore, examine the roles of fast depressor coxa (Df) and fast extensor tibia (FETi) motor neurons in executing the changes in joint kinematics that are pertinent to control of running speed. The results are not entirely consistent with the immediate impressions derived from observing preparations with unloaded legs.

Materials and methods

The animals and procedures used in this experiment are described in detail in the companion paper (Watson and Ritzmann 1997). The joint cycle period was the interval from the onset of joint flexion to the start of the next flexion. Joint excursion was calculated by determining the maximum joint angle (the extension/flexion transition joint angle) and subtracting the minimum joint angle (the flexion/extension transition joint angle). The duration of the flexion to extension transition was defined as the time it took the joint to go from 10° greater than minimum joint angle during flexion to 10° greater than minimum joint angle during extension.

For both the Df and FETi EMG records, slow and fast motor neuron potentials were different enough in amplitude that they could be distinguished in all records. In all steps examined, both Df and FETi were active in association with the extension phase of each joint cycle [which occurred mostly during stance phase (see Watson and Ritzmann 1997)]. For this paper, our data focused primarily on stepping speeds below 10 Hz, which included steps with up to two fast motor neuron potentials per extension phase. Additional fast potentials in each cycle tended to obscure the background slow motor neuron activity in our electrical records, making assessment of the total motor activity impossible.

Results

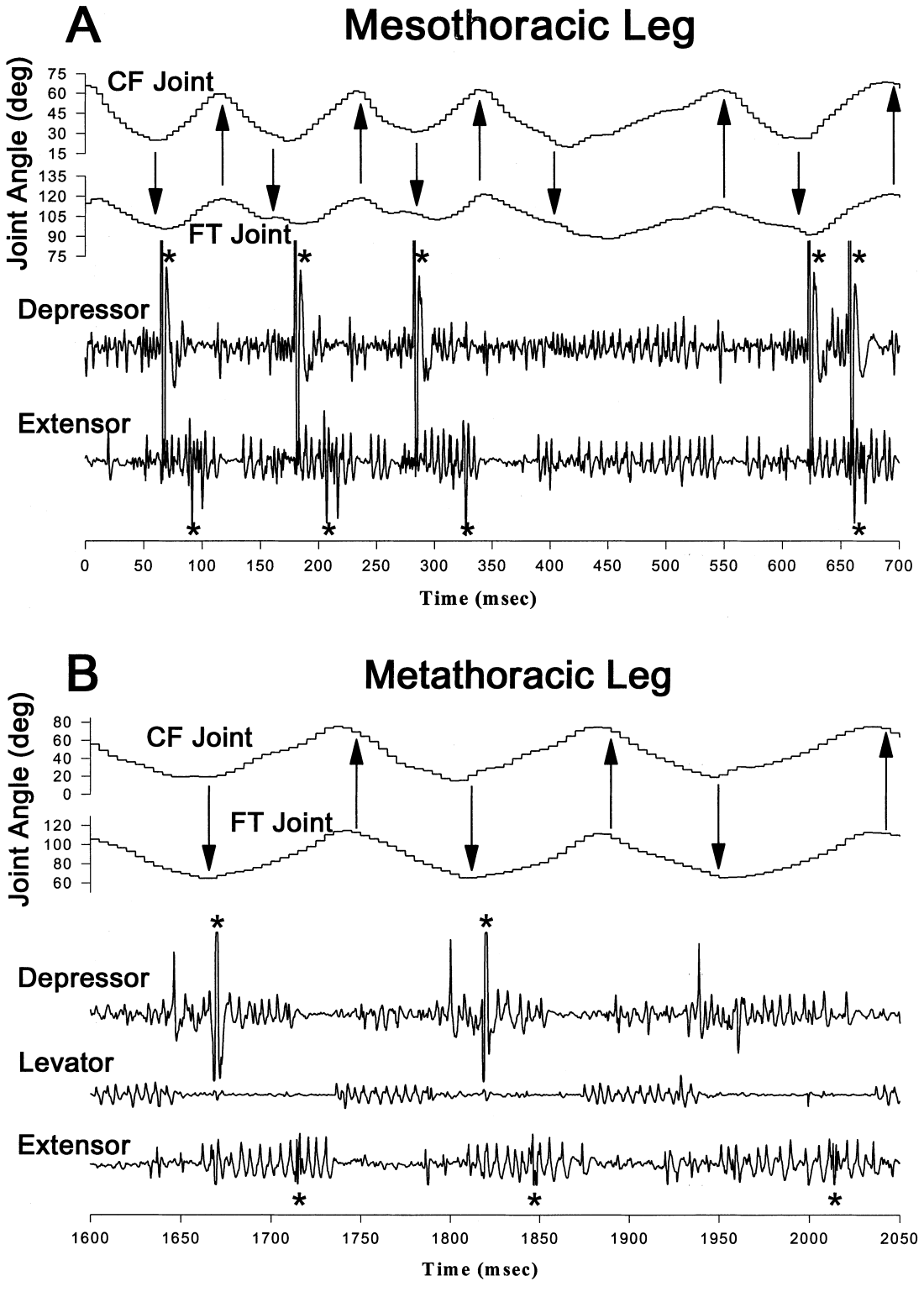
The data reported herein are based upon 112 steps from four roaches from which EMGs were recorded in the mesothoracic (T2) leg and 104 steps from five roaches from which EMGs were recorded in the metathoracic (T3) leg. For this paper, in addition to the steps analyzed for the companion paper (Watson and Ritzmann 1997), we analyzed data from joint cycles associated with one to three fast muscle potentials per cycle (Df: one potential/cycle, $n = 39$; two potentials/cycle, $n = 11$; three potentials/cycle, $n = 1$; FETi: one potential/cycle, $n = 73$; two potentials/cycle, $n = 28$: three potentials/cycle, $n = 0$). Although we attempted to match the running speeds of the two sets of data, it should be noted that the median step period of the runs from which T3 steps were taken was significantly greater than the median of those runs from which the T2 steps were taken (193.6 ms versus 167.8 ms, $P < 0.01$). The majority of our data were runs similar to those depicted in Fig. 1 in that the recorded leg showed recruitment of Df and/or FETi in some cycles interspersed with cycles with no fast motor neuron activity.

►

Fig. 1A, B Raw data from rapidly running cockroaches shows the occurrence of fast and slow motor activity relative to joint movements. CF and FT joint angle records synchronized with depressor coxa and extensor tibia records from the mesothoracic leg **A** of a cockroach running at 4–8 Hz and **B** CF and FT joint angle records synchronized with depressor coxa, levator coxa and extensor tibia EMG records from the metathoracic leg of a cockroach running at 6–7 Hz. *Asterisks* mark fast depressor coxa and fast extensor tibia EMGs. Superimposed between the motion records, the *downward pointing arrows* indicate when the tarsus contacted the substrate and the *upward pointing arrows* indicate when the tarsus lifted from the substrate. The intermediate-sized spike seen at the start of each depressor burst in **B** was an artifact which occurred consistently in this animal but was easily distinguished from the much larger Df spike. We believe this artifact was due to wire noise

Variation of kinematics with running speed

Over the range of running speeds we investigated, increased speed could not be accounted for by an increase in joint excursion. There was no change in coxa-femur (CF) joint excursion in either leg, or in femur-tibia (FT) excursion in T3 (Fig. 2). In the T2 leg, the FT joint excursion actually decreased slightly as walking speed increased (Fig. 2C1). This latter effect was due primarily to a decrease in flexion of the T2 FT joint during each step (Fig. 2C2), i.e. maximal joint angle remained relatively constant as the animals ran faster, while the minimum joint angle increased.



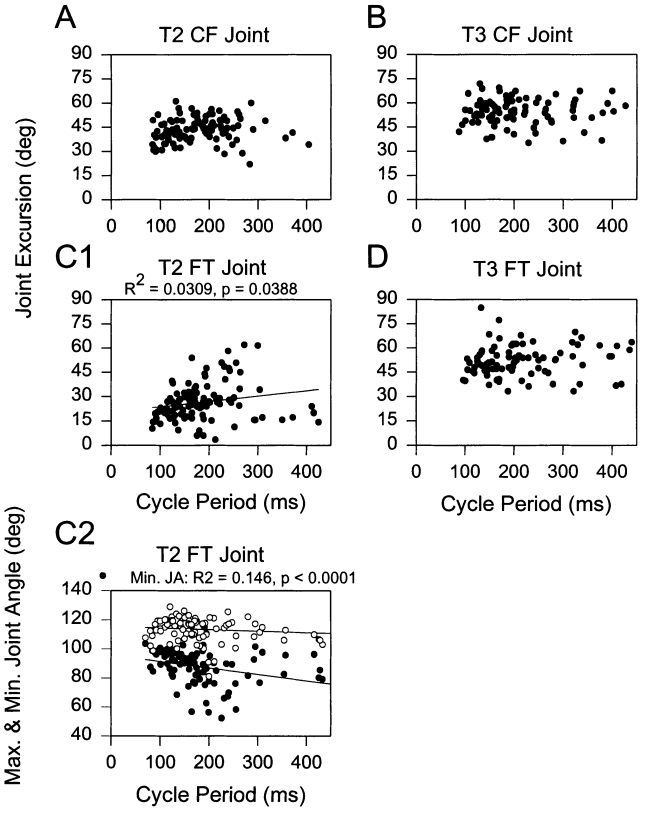
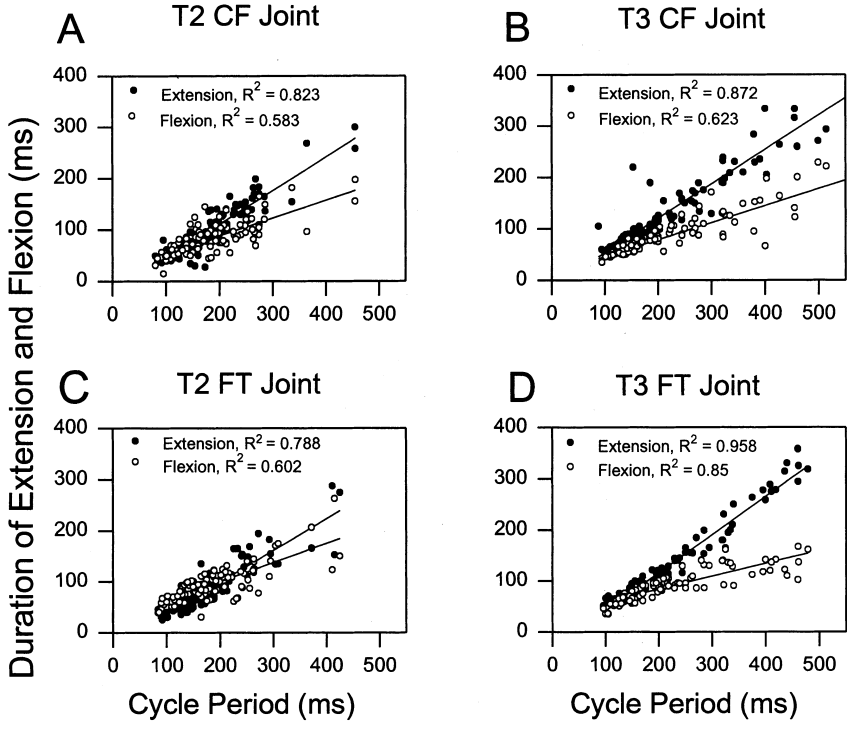


Fig. 2A–D Over the step-frequency range 2.5–10 Hz there was no increase in CF and FT joint excursion with increased running speed. Joint excursion as a function of joint cycle period for the **A** T2 CF joint, **B** T3 CF joint, **C1** T2 FT joint, and **D** T3 FT joint. (**C2**) The maximum (*white circles*) and minimum (*black circles*) joint angle attained by the T2 FT joint as a function of cycle period

In the absence of greater joint excursion, the animal could increase stepping frequency, and therefore speed, by altering the velocity of either extension or flexion phases, or by shortening the transition phase between flexion and extension. In all of the pertinent joints, the duration of both flexion and extension phases were positively correlated with cycle period (Fig. 3). Because the excursion is unchanged with cycle period, positive correlations between cycle period and duration of both flexion and extension implie increases in joint velocity at both phases of the joint cycle with increasing stepping speed. However, consistent with previous data (Delcomyn 1971; Pearson 1972, 1976), cycle duration correlated more strongly with duration of joint extension than with duration of joint flexion. A direct measure of mean angular joint velocity for extension around both joints in both T2 and T3 legs shows that the velocity of joint extension does in fact increase as cycle period decreases, i.e. increasing walking speed (Fig. 4). In addition to joint velocity, the transition period from flexion to extension decreased with faster running speeds in all joints examined (Fig. 5). Thus, greater step frequency was achieved by increasing the velocity of joint movement and decreasing the associated transition period.

The fourth possible way of increasing running speed is to alter force in the absence of a change in joint kinematics, thereby causing greater body movement for each step. This possibility would only be a factor at running speeds that include an airborne phase, which does in fact occur at very fast speeds in *Periplaneta americana* (Full and Tu 1991). Neither we nor Full and Tu (1990) detected an airborne phase during rapid running in *Blaberus discoidalis*.

Fig. 3A–D CF and FT extension duration varied more than flexion as a function of running speed. Relationships between joint cycle period and the durations of extension (*black circles*) and flexion (*white circles*). Segments and joints designated as in Fig. 2



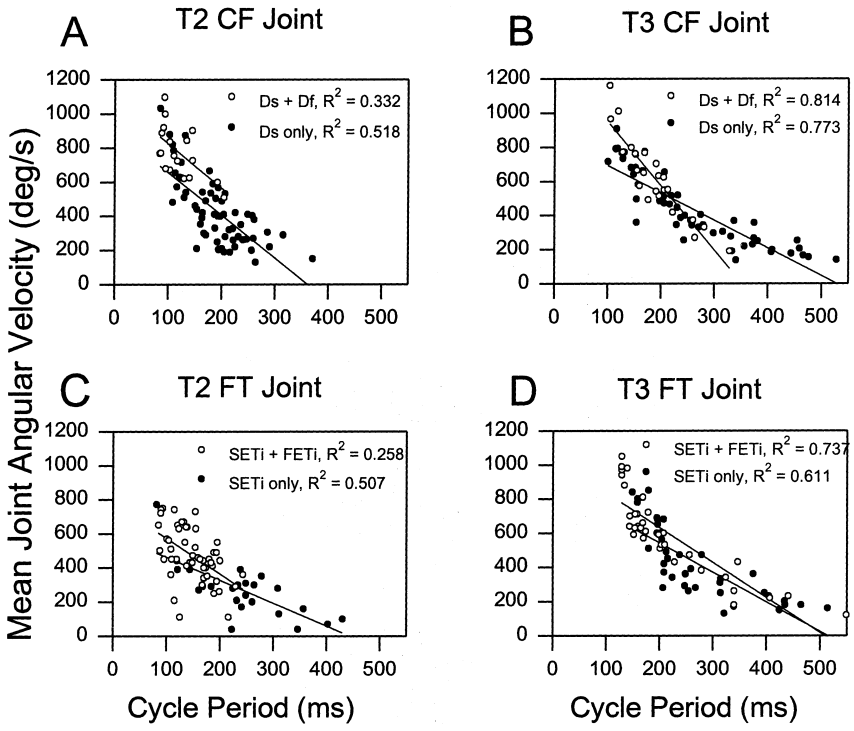


Fig. 4A–D Mean joint extension velocity increased as running speed increased. However, slow motor neuron activity could produce joint extension velocities as high as those associated with fast motor neuron activity. Mean joint angular velocity during extension as a function of joint cycle period, for those steps in which only the slow depressor coxa or extensor motor neurons were active (*black circles*) and for those steps in which fast depressor coxa or fast extensor tibia motor neurons were active (*white circles*). Segment and joints designated as in Fig. 2

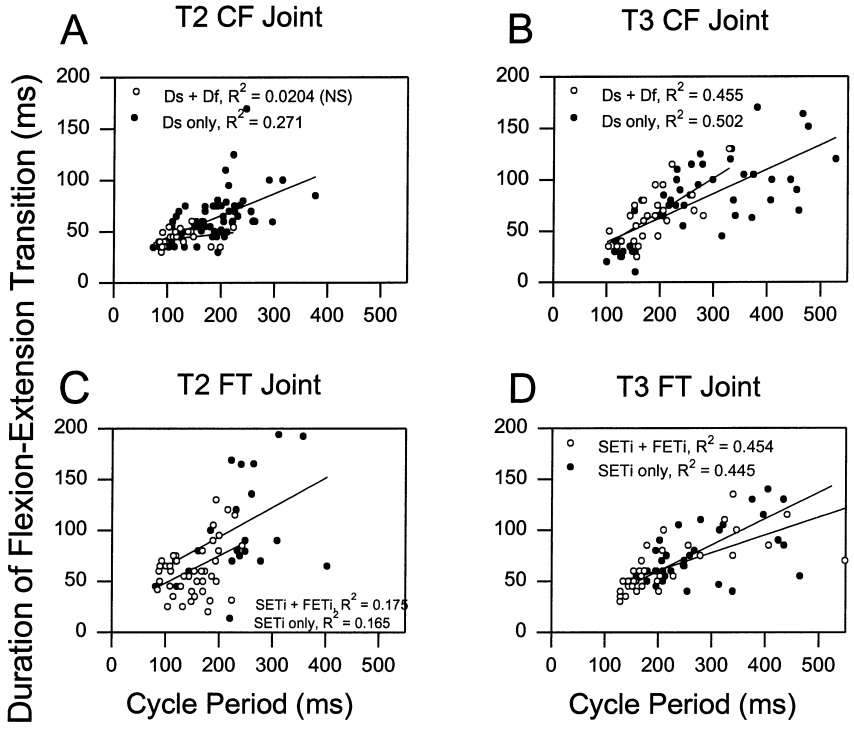


Fig. 5A–D The duration of the transition from flexion to extension decreased as running speed increased. Duration of transition from flexion to extension as a function of joint cycle period, for those steps in which only the slow depressor coxa or extensor motor neurons were active (*black circles*) and for those steps in which fast depressor coxa or fast extensor tibia motor neurons were active (*white circles*). Segment and joints designated as in Fig. 2

Effects of fast motor neurons Df and FETi on joint angular velocity

As shown in the companion paper, the increasing frequency of slow motor neuron potentials is correlated with increased joint angle velocity. This raises the question of whether the recruitment of the fast motor neurons (i.e. Df and FETi) causes an increment of velocity that is greater than would be expected from simply increasing slow motor neuron frequency. The data which compare the effect of fast and slow motor neurons on joint movement in unloaded legs (Usherwood 1962; Watson and Ritzmann 1995) suggest that recruitment of fast motor neurons would be expected to completely override slow motor neuron effects and take over control of the leg joint, causing a great increase in joint velocity.

If the influence of Df is so strong that it essentially renders the slow depressor coxa neuron (Ds) contribution inconsequential, the cycles including single Df potentials (the majority of cases in our data set) would be expected to have a constant joint velocity relative to those controlled by varying frequencies of Ds potentials.

To determine whether such an incremental increase occurs in freely moving animals, we looked at transitional running speeds that included cycles containing fast and slow motor activity as well as those that contained only slow motor activity, often in adjacent cycles. Surprisingly, the data did not support the notion that recruitment of the fast motor neuron resulted in an incremental increase in joint angular velocity (Fig. 4). Although the cycles that included fast motor neurons were clustered in regions of highest joint velocity and shortest cycle period, those joint velocities were also seen in cycles in which slow motor neurons were recruited alone. Indeed, some of the cycles with fastest joint angle velocity and shortest cycle period did not include activation of fast motor neurons. Thus, it is certainly possible to generate the same joint velocities with or without recruitment of fast motor neurons.

Our data further suggest that recruitment of a fast motor neuron does not eliminate the influence of slow motor neuron frequency upon joint velocity as described in the companion paper (Watson and Ritzmann 1997). In both FT joints and in the T3 CF joint, angular velocity still varied as a function of slow motor neuron frequency even when fast motor neurons were active (Fig. 6). For the FT joints in both pairs of legs, the relationship for velocity of extension versus slow extensor tibia neuron (SETi) frequency was highly significant even when FETi was recruited ($P < 0.0001$). For the CF joints, the relationship in T3 legs was also highly significant ($P = 0.0018$), while in T2 legs the probability for the same relationship was slightly above the level of significance ($P = 0.066$). The correlation for both CF joints are probably underestimates of the relationship that exists between slow motor neuron activity and joint angular velocity when Df is activated. The Df spike occurs in the depressor burst at about the same time as the high-frequency burst in Ds (Figs. 1, 7). Thus, many Ds muscle potentials are obscured by the much larger Df spike, making it difficult to obtain an accurate value for Ds mean spike frequency. Given this problem, it is remarkable that a significant correlation of any magnitude is seen for the T3 CF joint. In all likelihood the correlation is even greater than indicated in Fig. 6B and a significant correlation may in fact exist for the T2 CF joint.

The analysis described above focuses upon the effect of a single fast motor neuron potential acting on mean velocity over an entire joint cycle. It is possible that the influence of fast motor neurons occurs only over a small period of time during that cycle. If the fast motor neurons were dramatically increasing joint velocity, even for a short time, the peak joint velocity should predictably follow the occurrence of the fast motor potentials. However, there was no correlation between the phase of a fast motor neuron potential and the phase of the maximum joint angular velocity for that joint cycle. Moreover, within our data set joint velocity was not further increased when multiple fast potentials were recorded. Mean extension velocity of cycles with two fast potentials was not significantly faster than that for cycles with only one fast potential.

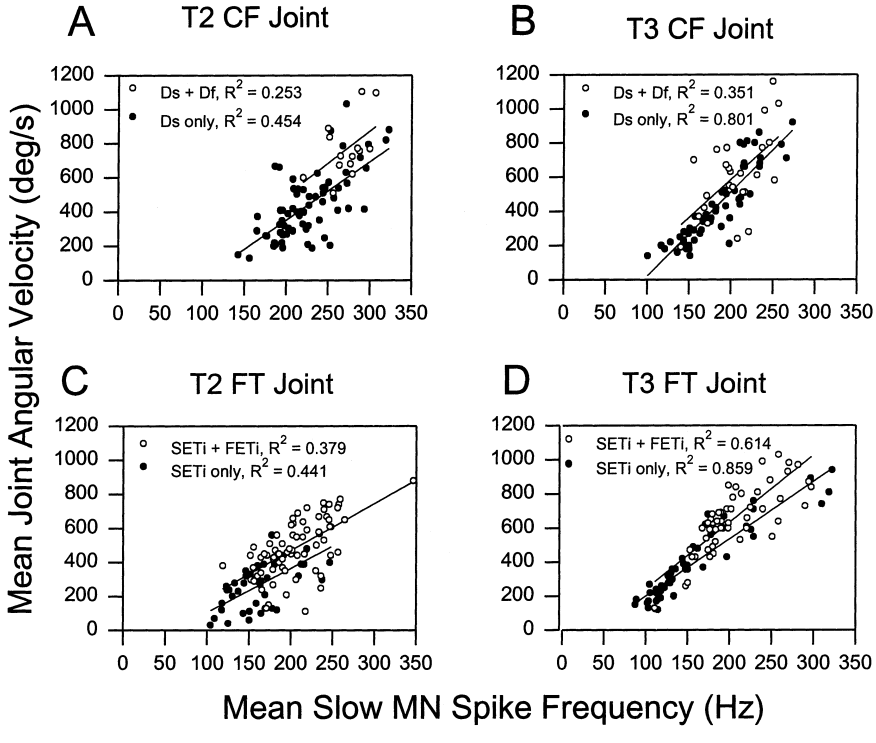


Fig. 6A–D Joint extension velocity varied as a function of slow motor neuron frequency even when a fast motor neuron potential occurred during the extension. Average slow motor neuron EMG frequency versus average joint angular velocity for the joint cycles with only slow motor neuron activity (*black circles*) and for joint cycles with slow and fast motor neuron activity (*white circles*). Segment and joints designated as in Fig. 2

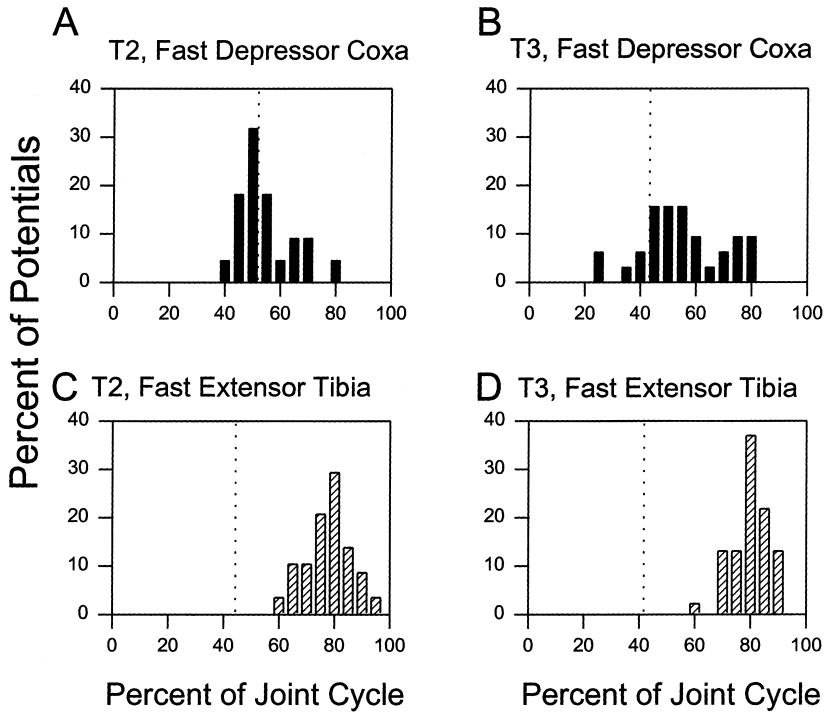


Fig. 7A–D The fast coxal depressor motor neuron was recruited at the transition from CF flexion to extension during running, while the fast extensor tibia motor neuron was recruited during latter half of FT extension. Normalized fast depressor coxa activity from all joint cycles containing one or more fast motor neuron potentials, recorded from **A** T2 legs (21 joint cycles) and **B** T3 legs (27 joint cycles), and normalized fast extensor tibia activity from all joint cycles containing one or more fast potentials, recorded from **C** T2 legs (21 joint cycles) and **D** T3 legs (27 joint cycles) legs from running *B. discoidalis*. The mean phase of the transition from flexion to extension is indicated by the *dashed vertical line*. For these plots, the joint cycle starts at the extension to flexion transition

Recruitment timing of depressor coxa and extensor tibia motor neurons

At slow stepping speeds, the point at which Ds or SETi activity commenced and ceased relative to joint extension remained reasonably constant. Typically, the start of Ds or SETi activity was near 40% of the joint cycle (i.e. at the beginning of extension of the joint) and the end was 95% of the joint cycle. However, as speed increased and the duration of extension dropped below 80 ms, the burst was compressed so that activity for each of these joints tended to end earlier.

As in earlier studies (Ewing and Manning 1966; Pearson 1972; Delcomyn 1973; Krauthamer and Fourtner 1978), our data indicated that Df and FETi are recruited at faster running speeds. Recruitment of Df occurred at 54.6 ± 10.8% (mean ± SD) of the joint cycle (Fig. 7). Df potentials usually coincided with the end of the high-frequency Ds activity that occurs at the beginning of each Ds burst (Watson and Ritzmann 1997). This meant that Df potentials occurred just after the tarsus contacted the treadmill, i.e. foot touchdown (16 ± 5.3 ms in T3, 10 ± 3.8 ms in T2). In contrast, FETi typically fired at 81 ± 8.44 % of the joint cycle and coincided with the end of the SETi burst at the time of highest frequency SETi activity. Because the end of SETi bursts was shifted to an earlier phase of the joint cycle at these running speeds, this recruitment timing of FETi coincided with the middle phase of FT extension. We found no difference between the T2 and T3 legs in the joint cycle phase at which Df and FETi were recruited (Fig. 7). FETi was recruited more often than Df. Of joint cycles with fast motor activity, both Df and FETi were active in 38 joint cycles, FETi was active alone in 42 joint cycles, and in only 5 joint cycles was Df active without concomitant FETi activity.

Effects of Ds and Df on transition from flexion to extension

The last effect on running speed that we detected in our kinematic analysis was a shortening of transition from flexion to extension at each joint. The highest frequency Ds activity as well as single Df potentials typically occur near 50% of the joint cycle (Fig. 7), which is at the flexion to extension transition. Therefore, we examined the relationship between motor neuron activity and the kinematic changes of this transition. There was a significant negative correlation between the frequency of Ds during the initial high-frequency burst and the duration of the transition from flexion to extension (T2: $R = 0.463$, $P = 0.0005$; T3: $R = 0.495$, $P = 0.0003$). Unlike the correlations with joint angle velocity reported by Watson and Ritzmann (1997), where the highest correlations were with the middle of Ds bursts, the correlation between Ds frequency and transition duration was seen only for the first part of Ds bursts in the T3 leg. In T2 legs, correlations were seen for both the first and middle parts of the burst, but the relationship was much greater for the first part (middle segment in T2: $R = 0.404$, $P = 0.0244$). Thus, higher frequency in the initial portion of Ds bursts contributed to shortening the duration of the transition from flexion to extension.

To assess the additional contribution made by Df in shortening transition time, we compared the transition kinematics of steps with Ds alone versus steps that included Df activity where mean CF joint angular veloc-

ities during extension were similar (T2: 600–1000° s^{-1}–Ds: $n = 13$, Df: $n = 19$; T3: 400–800° s^{-1}–Ds: $n = 10$, Df: $n = 15$). The duration of the flexion to extension transition was significantly shorter for the steps in which Df was active (T2: $P = 0.0401$; T3: $P = 0.0274$). Because we only compared cycles with similar extension velocity, the decrease in duration associated with Df activity reflects shorter time in the delay between flexion and extension (Fig. 8). Thus, the more rapid progression from flexion to extension associated with Df activity was due to CF extension starting sooner relative to flexion, not to a greater joint angular velocity during extension.

Effect of Df potential location on transition from flexion to extension

Although most Df potentials occurred near the flexion-extension transition, there were some potentials that occurred at other times in the joint cycle. There was some indication that Df potentials were most effective in accelerating the flexion-to-extension transition if they occurred in a narrow time window within the joint cycle. For both T2 and T3 legs, there was a significant positive correlation between the phase at which the Df potential occurred and the duration of the flexion to extension transition (T2: $R = 0.581$, $P = 0.0017$; T3: $R = 0.654$, $P = 0.0032$, see Fig. 9). This correlation was seen over 40–90% of the joint cycle phase. In T3 legs, Df potentials occurring before 40% of the joint cycle were associated with cycles whose transition durations were widely scattered (Fig. 9A). Thus, if Df potentials occurred significantly before or after 40–50% of the joint cycle, they seemed unable to shorten the transition to extension.

Effects of FETi on transitions

There was no pronounced effect of FETi on the flexion-to-extension transition or the extension-to-flexion transition in the FT joint. In both T2 and T3 legs, there was no discernible difference in duration of transition between cycles with FETi and cycles with only SETi. We did note a significant increase in the maximum joint angle that was attained when FETi was active (for T2, $P < 0.0001$; for T3, $P < 0.01$). This change in maximum joint angle did not cause an increase in excursion because of a concomitant increase in minimum joint angle.

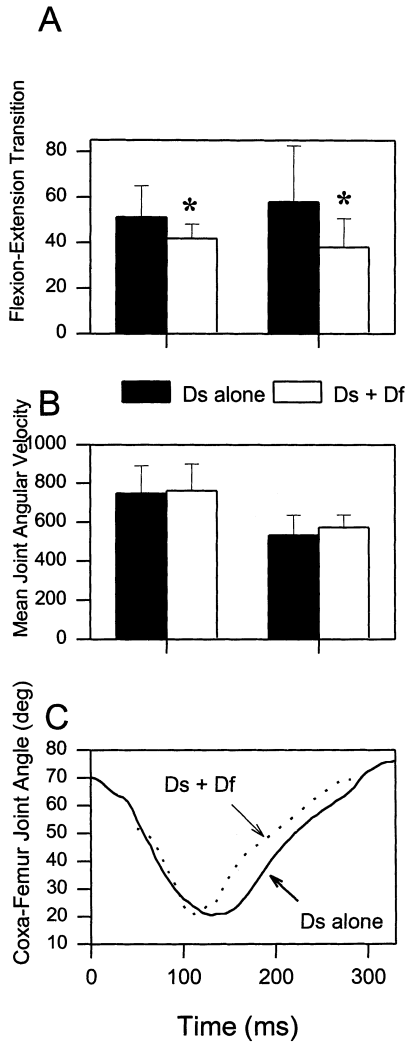


Fig. 8A–C For steps with the same CF joint extension velocity, there was a significant shortening of the transition from flexion to extension associated with recruitment of the fast depressor: **A** duration of transition from flexion to extension; **B** joint angular velocity during extension for T2 and T3 CF joint movements associated with exclusively slow depressor coxa neuron activity (Ds alone) and joint movements associated with concomitant slow and fast depressor coxa neuron activity (Ds + Df), $^{*}P < 0.05$. **C** Example of timing of a T2 CF joint movement associated with exclusively slow depressor (Ds alone) activity (*solid line*) and a joint movement associated with concomitant slow and fast depressor (Ds + Df) activity (*dashed line*). Ds and Df joint angle records were aligned by matching flexion movements

Discussion

The data presented show that, in the range 2.5–10 Hz, *B. discoidalis* increases step frequency by increasing the joint velocity during extension and by decreasing the CF flexion to extension transition period. The increase in stride frequency, as well as the lack of any increase in joint excursion or any airborne phase at these speeds, are consistent with other observations of cockroach running (Delcomyn 1971; Full and Tu 1990, 1991), and suggest that the observed effects on stride frequency account for increased running speed over the range examined. Therefore, in our analysis of EMGs, we focused on those EMG parameters which varied with stepping frequency, namely frequency of slow motor neuron potentials and recruitment of fast motor neurons, and attempted to associate such EMG variations with changes in joint velocity and transition duration.

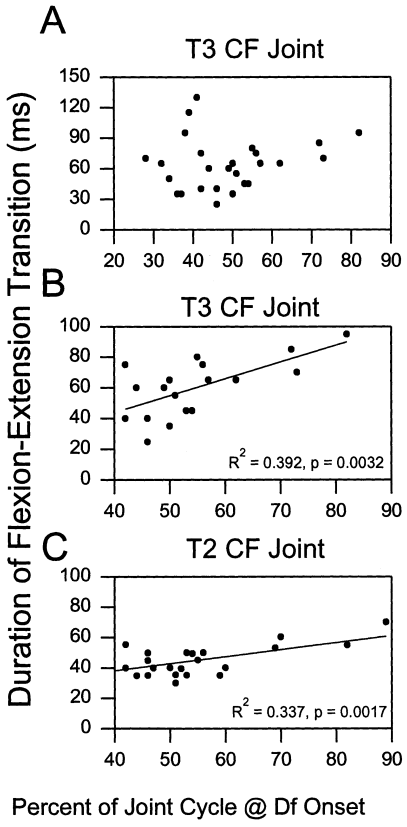


Fig. 9A–C The shortening effect of Df on the flexion-to-extension transition was correlated with the phase of the joint cycle at which Df was recruited. Duration of the flexion-to-extension transition as a function of the phase of the joint cycle at which a Df potential occurred. **A** T3 leg and **C** T2 leg plots include all data available. T3 plot **B** includes only those points from the T3 data set in which Df occurred between 40% and 90% of the joint cycle

The data presented in this paper tested the role of fast motor neurons in animals that were freely running on a treadmill and, therefore, experiencing normal forces of load and inertia. Contrary to our expectations, we did not observe dramatic changes in joint velocity when fast motor neurons were recruited. First, there is a considerable range of overlap in joint velocities between cycles generated by fast and slow motor neurons and those generated solely by slow motor neurons. Thus, although the fastest movements typically include fast motor activity, slow motor activity is capable of generating joint movements whose velocity is equal to that of cycles that are associated with single fast motor neuron potentials. Second, even with fast motor neuron recruitment, T3 CF and both T2 and T3 FT joint velocities continue to increase as a function of slow motor neuron frequency. Third, within each joint cycle that included fast motor neuron activity, the occurrence of fast motor neuron potentials did not suddenly accelerate the movement of the joint as would be indicated by an inflection in the joint kinematics. We conclude that recruitment of fast motor neuron activity at transitional running speeds does not completely override the effects of slow motor neuron activity.

Nevertheless, fast motor neuron activity is certainly associated with faster running speeds. Thus, we looked at other aspects of the joint cycle that might be affected by fast motor neuron activity and, therefore, contribute to faster running speeds. Df potentials are localized to the point in the joint cycle when the transition from flexion to extension occurs. The timing of single Df potentials coincided with the phase of the joint cycle at which the highest frequency of Ds potentials occurred. This suggests that the additional, brief tension generated by single Df potentials added to the background maximum tension produced by high-frequency Ds activity seems to modulate one part of the joint cycle, namely, the transition from flexion to extension. This precisely timed high-frequency Ds and Df activity could serve to overcome a particularly high muscle loading at this point due to either whole body inertia or a poor mechanical advantage of the depressor coxa muscle relative to the antagonistic levator coxa. The result would be a shorter joint cycle in spite of the fact that the actual joint velocity during extension was unaffected. While our data do not distinguish between the possible sources of loading at the flexion to extension transition, the effect on the transition is apparent. Recruitment of Df correlated with shorter duration transitions, and the phase of the joint cycle at which Df is effective is limited. Confirmation of the importance of the timing of tension peaks in the depressor coxa muscle can be found also in the negative correlation between Ds firing rate during the initial high-frequency burst and the duration of the transition from flexion to extension.

While our data do not show a significant effect of one or two Df potentials on joint angular velocity at the transition running speeds of 5–10 Hz, it is possible that at faster running speeds, the tension produced by multiple Df potentials produces higher joint velocities in the same manner as high-frequency Ds activity. In *B. discoidalis*, the rate of increase of stride frequency with running speed begins to level off above 10 Hz (0.25 m s^{-1}). At the maximum sustainable stride frequency of 14 Hz, the running speed is 0.35 m s^{-1}, and further increases in speed are attained by increasing stride length (Full and Tu 1990). In our data, a 10-Hz stepping rate was associated with one to two Df potentials per cycle. The data of Full and Tu (1990) suggest that any additional Df potentials associated with higher running speeds do not result in much of an increase in stepping frequency. Additional Df potentials may instead be necessary to increase joint excursion to produce greater stride length. Thus, increased joint velocity would be achieved by traversing a greater excursion in the same time.

The only other data correlating Df activity with CF joint movement during a locomotory behavior in cockroaches come from the escape response of *P. americana*. In the T2 leg, the number of Df potentials is correlated with the total CF joint excursion (Levi and Camhi 1996). The mean T2 CF joint excursion seen in our data associated with one to two Df potentials (50°) is similar to

the excursion associated with two Df potentials in the escape movement. We did not analyze data from cycles including more than two Df potentials because any more than two Df potentials masked Ds activity in EMG records, making assessment of the total muscle activity impossible.

There is evidence that several of the muscles in the depressor coxa complex act as active springs during rapid running, and some do more negative than positive work during the joint cycle (R. J. Full, personal communication). The active spring effects of these muscles may resolve the apparent paradox between fast motor neuron effects in unloaded and loaded legs. The function of Df may be to rapidly modulate tension to alter the spring characteristics of these muscles during running. Such spring characteristics and associated joint stiffness may be affected by co-contraction of depressor and levator muscles. The ineffectiveness of Df recruitment before about 40% of the step cycle to alter the flexion to extension transition may be due to co-contraction or residual tension in the levator coxa muscles until this phase. Our levator coxa recordings were limited to one of many muscles which may be active during running, so we cannot address this hypothesis fully.

Although the effect of SETi frequency on joint angular velocity is similar to that of Ds as shown by Watson and Ritzmann (1997), the role of FETi is not clear. As with Ds and Df, the timing of FETi coincides with the highest-frequency SETi activity. However, in the extensor tibia muscles this component occurs at the end of the burst, where there are no transitions or inflections evident in the extension movement. The high-frequency SETi potentials and the coincident activation of FETi at the end of SETi bursts may reflect a low mechanical advantage of the extensor tibia muscle relative to the flexor tibia muscle. The mean FT joint angle at maximum extension is greater when FETi is active than when it is not. Alternatively, the high-frequency phases of FETi may serve to overcome high joint torque produced by the biomechanical configuration of the leg at maximal extension. Indirect evidence for such a phenomenon comes from other species. In *P. americana*, a considerably lighter cockroach than *B. discoidalis*, an inflection at extended joint angles is often discernible in the FT joint movement during running (Krauthamer and Fourtner 1978; Watson and Ritzmann 1995; Larsen et al. 1995). Finally, in the T2 leg of the locust, the maximal longitudinal force on the tibia is registered at mid-stance (Newland and Emptage 1996), and declines thereafter, even though SETi continues to fire and FT angle continues to increase.

In conclusion, the current paper and the companion study (Watson and Ritzmann 1997) provide the basis for further experimental investigations of particular aspects of the running movements. They present relationships between leg kinematic parameters and the timing and frequency of depressor and extensor motor neurons. As is appropriate for a basic analysis, these data provide more questions than answers regarding the control of leg movement during locomotion. Any model for motor control of leg movement in running must now account for the joint kinematics and EMG relationships that we have documented. Moreover, the data provide several testable hypotheses regarding the transfer functions from motor neuron activity to movements that can be pursued in future studies. For example, the timing of fast motor neurons (Df and FETi) coincides with the timing of high-frequency activity of the respective slow motor neurons within each burst. Clearly, this is a point in the joint cycle when the muscles must exert extra force to accomplish an important feature. One hypothesis is that the joint muscles may be performing like active springs to stiffen the joint and, thereby, shorten the transition period from flexion to extension. Further experiments will be necessary to examine joint stiffening and thereby test this hypothesis. Ultimate resolution of the function of frequency variation and fast motor neuron recruitment in the insect leg will require detailed knowledge of how whole-body kinetics are expressed biomechanically as joint torques during running, as well as accurate data on muscle mechanics.

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References

Burrows M, Hoyle G (1972) Neural mechanisms underlying behavior in the locust *Schistocerca gregaria*. III. Topography of limb motorneurons in the metathoracic ganglion. J Neurobiol 4: 167–186

Delcomyn F (1971) The locomotion of the cockroach *Periplaneta americana*. J Exp Biol 54: 443–452

Delcomyn F (1973) Motor activity during walking in the cockroach *Periplaneta americana*. I. Free walking. J Exp Biol 59: 629–642

Ewing AW, Manning A (1966) Some aspects of the efferent control of walking in three cockroach species. J Insect Physiol 12: 1115–1118

Full RJ, Tu MS (1990) Mechanics of six-legged runners. J Exp Biol 148: 129–146

Full RJ, Tu MS (1991) Mechanics of a rapid running insect: two-, four- and six-legged locomotion. J Exp Biol 156: 215–231

Krauthamer V, Fourtner CR (1978) Locomotory activity in the extensor and flexor tibiae of the cockroach, *Periplaneta americana*. J Insect Physiol 24: 813–819

Larsen GS, Frazier SF, Fish SE, Zill SN (1995) Effects of load inversion in cockroach walking. J Comp Physiol A 176: 229–238

Levi R, Camhi JM (1996) Producing directed behaviour: muscle activity patterns of the cockroach escape response. J Exp Biol 199: 563–568

Newland PL, Emptage NJ (1996) The central connections and actions during walking of tibial campaniform sensilla in the locust. J Comp Physiol 178: 749–762

Pearson KG (1972) Central programming and reflex control of walking in the cockroach. J Exp Biol 56: 173–193

Pearson K (1976) The control of walking. Sci Am 235: 72–86

Pearson KG, Iles JF (1971) Innervation of coxal depressor muscle in the cockroach, *Periplaneta americana*. J Exp Biol 54: 215–232

Usherwood PNR (1962) The nature of 'slow' and 'fast' contractions in the coxal muscles of the cockroach. J Insect Physiol 8: 31–52

Watson JT, Ritzmann RE (1995) Combined intracellular stimulation and high speed video motion analysis of motor control neurons in the cockroach. J Neurosci Methods 61: 151–157

Watson JT, Ritzmann RE (1997) Leg kinematics and muscle activity during treadmill running in the cockroach, *Blaberus discoidalis*, I. Slow running. J Comp Physiol A 182: 11–22

Wilson JA (1979) The structure and function of serially homologous leg motor neurons in the locust. I. Anatomy. J Neurobiol 10: 41–65