Critical Points in the Forelimb Fictive Locomotor Cycle and Motor Coordination: Evidence From the Effects of Tonic Proprioceptive Perturbations in the Cat

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Saltiel, Philippe and Serge Rossignol. Critical points in the forelimb fictive locomotor cycle and motor coordination: evidence from the effects of tonic proprioceptive perturbations in the cat. J Neurophysiol 92: 1329–1341, 2004; 10.1152/jn.00563.2003. During locomotion, the limbs of one girdle must remain coordinated in different conditions. To understand the neural mechanisms underlying such coordination, tonic protraction/retraction of one shoulder or tonic flexion/extension of one elbow was applied during fictive locomotion in high decerebrate and paralyzed cats. We studied bilateral changes in the timing and amplitude characteristics of electroneurographic (ENG) muscle nerve bursts of cleidobrachialis (CIB, elbow flexor and shoulder protractor) and the two heads of triceps (long, TrlLo, elbow extensor and shoulder retractor and lateral, TrlLa, elbow extensor). Perturbations induced bilateral changes in amplitude and timing of ENG bursts that were anchored on certain critical points in the cycle. These critical points could correspond to morphological characteristics within the bursts or to bilateral onsets or offsets of ENG bursts. For instance, in response to shoulder and elbow perturbations, burst changes occur in relation to a fixed point, labeled point C, occurring at about mid-extensor burst and corresponding to a simultaneous abrupt increase in TrlLa amplitude and a decrease in amplitude of contralateral CIB. At a point labeled B, corresponding to about mid-flexor burst, CIB amplitude increases above control with elbow extension or starts decreasing with shoulder protraction. Although cycle reorganization is specific for each type of tonic perturbation, a common feature is that the changes in burst duration are achieved through discrete shifts between consecutive critical points. It is postulated that coordination may be based on a discrete temporal cycle structure along which critical points delimiting burst components are shifted.

INTRODUCTION

The basic hindlimb locomotor rhythm is generated centrally because “fictive” locomotion can be evoked by 3-4 hydroxy-l-phenylalanine (DOPA) in spinalized and paralyzed cats (Grillner and Zanger 1979) or spontaneously after deafferentation or paralysis in decorticate cats (Perret and Cabelguen 1980). In the forelimbs, a centrally generated rhythm with typical complex activity of certain muscles can also be obtained in paralyzed decorticate or decerebrate cats (Arshavsky et al. 1986; Cabelguen et al. 1981; Göddertz et al. 1990; Shimamura et al. 1984).

In real life, the locomotor pattern must be modulated by peripheral inputs to adapt to the environment. To change trajectory, turn, and walk on slopes or uneven grounds, a locomoting cat may have to modify its posture or range of joint excursion. How are these bilateral postural changes, signaled by proprioceptive inputs, integrated into the locomotor pattern?

In nonwalking acute spinal preparations, the contralateral hindlimb tonic position influences its responses to a cutaneous stimulus causing ipsilateral flexion withdrawal, in a way seemingly optimal for locomotion (Rossignol and Gauthier 1980). If the contralateral hindlimb is fully retracted, an inadequate position for weight support, it flexes rather than extends, which brings it forward to a new position where it could sustain weight. Thus proprioceptive inputs can coordinate bilateral responses in a behaviorally meaningful way.

Proprioceptive inputs influence treadmill locomotion. In premammillary cats, stretching the hindlimb triceps surae such that its contraction force exceeded 4 kg inhibited ipsilateral flexor burst generation; the limb extensors remained tonically active to support the animal (Duysens and Pearson 1980). In low spinal cats, the hip angle also determines initiation of the swing phase. If one hindlimb is held and slowly brought backwards, while the other continues to walk, its extensors remain tonically active until a critical hip angle is reached, at which point it suddenly flexes. This initiation of swing tends to occur during the contralateral mid-phase of -swing, suggesting maximal interactive signals responsible for interlimb coordination during these mid-phases (Grillner and Rossignol 1978). A different study using split belts driven at different speeds concluded that probably several interlimb coordination mechanisms exist (Forssberg et al. 1980). In favor of a mid-phase mechanism were first, that an alternating relationship was maintained between the mid-points of the quadriceps extensor bursts (more so than between onsets or ends of these bursts) and second that most timing adjustments occurred around mid or late swing. However, the neural basis for such coordination remained unknown.

The fictive locomotor preparation offers the unique opportunity to study how afferent feedback interacts with the centrally generated pattern. Few studies have addressed the effect of tonic proprioceptive inputs. Andersson and Grillner (1983) showed that passive movements of the hip of an otherwise denervated limb entrain the fictive locomotor rhythm bilateral...
ally, confirming the importance of hip angle (see also Krielaars et al. 1994). In rabbit hindlimb locomotion, Vidal et al. (1979) reported an increased relative duration of the flexor phase by tonic limb extension, repetitive electrical stimulation of ipsilateral cutaneous nerves, or with a serotonin precursor; conversely, the extensor phase was prolonged by tonic limb flexion, contralateral cutaneous stimulation, or by Dopa. In chronic spinal cats, Pearson and Rossignol (1991) also reported effects of the hip angle on the fictive pattern. Overall, however, these studies described the general effects of peripheral inputs on the locomotor pattern rather than attempted at understanding how these changes are organized within one limb or between the two limbs of the same girdle.

Is it possible, by studying the modifications in the fictive locomotor rhythm resulting from changing limb position and thus proprioceptive inputs, to learn about how the spatial features of posture could be integrated into the temporal structure of the rhythm? Can this provide clues to the structure of the locomotor central pattern generator (CPG)? The present paper describes the bilateral effects of a tonic shoulder protraction/retraction or an elbow flexion/extension. Emphasis was placed on examining closely how the locomotor cycle is reconstructed by the changes in tonic position of the forelimb to obtain clues about mechanisms of interlimb coordination and about critical points in the cycle around which postural and locomotor changes are integrated. The results have been partially published previously (Rossignol et al. 1993; Saltiel and Rossignol 1991). The companion paper examines the effect of phasic perturbations.

Methods

The results in this paper are based on eight cats (2.75–5 kg), although the effects of limb manipulations were initially explored in six other cats without potentiometers to monitor joint angles. These exploratory experiments have not been analyzed except for some electromyographic (ENG) recordings that corroborate the results from the eight cats.

Preparation

Cats were anesthetized with intravenous Saffan (a mixture of alphaxalone 9 mg/ml and alphadalone 3 mg/ml) with repeat doses of 0.25 mg/kg as necessary. After venous, arterial, and tracheal cannulations, the head was fixed in a stereotactic apparatus. Cats were decorticated by suction (Perret and Cabelguen 1980) or decerebrated with a spatula at the precollicular postmammillary level after bilateral carotid ligation. The tentorial bone was partly removed for access to the mesencephalic locomotor region (MLR).

Spinalization was performed at T13 to favor the emergence of forelimb locomotion (Amemiya and Yamaguchi 1984; Arshavsky et al. 1986). Anesthesia was stopped and paralysis induced with gallamine triethiodide [gallamine triethiodide (Flaxedil) iv, 20 mg hourly]. We maintained expired CO2 at 4–6%.

The joint angles and amplitudes of descent (see RESULTS) and calculated the means and SDs of their ENG display was sufficient (Zomlefer et al. 1984). Alternatively we selected for each cycle the onset and end of each burst as well as other events of interest such as the TriLa inflection point and the CIB onset of descent (see RESULTS) and calculated the means and SDs of their phase values with respect to the reference event. Control and perturbed cycles were tagged differently for statistical comparison. Nor-

Data analysis

ENGs during the sustained perturbation and 10–15 preceding control cycles were rectified, low-pass filtered (time constant 0.01 s) and digitized at 1 kHz.

We focused on comparing the average perturbed and control cycles, normalized to the same duration and synchronized on the same reference point. To describe the perturbed cycle alone requires a value for each burst onset, offset, or intra-burst event of interest. Comparing the perturbed cycle to a reference cycle is more parsimonious because several events may remain unchanged in timing, especially with the control cycle as reference. Comparing directly two perturbed cycles (e.g., elbow flexion vs. elbow extension) may be less simple, e.g., both burst onset and offset times may change, while one perturbation compared with control may just change burst onset and the other just burst offset. Further, synchronizing on a burst onset or offset cannot distinguish whether it becomes longer because it starts earlier, finishes later, or both. Therefore we used reference points located within bursts and recognizable by morphological correlates in the burst shapes (see RESULTS). The compared perturbed and control cycles were normalized in duration.

SELECT PROGRAM. For subsequent averaging, selecting with a cursor the reference point for each consecutive cycle in the appropriate ENG display was sufficient. Alternatively we selected for each cycle the onset and end of each burst as well as other events of interest such as the TriLa inflection point and the CIB onset of descent (see RESULTS) and calculated the means and SDs of their phase values with respect to the reference event. Control and perturbed cycles were tagged differently for statistical comparison. Normalized burst amplitudes were the integrated value of the whole burst divided by its duration.

AVERAGING PROGRAM. This program displayed the normalized control or perturbed cycle, drawn twice consecutively, with the
reference point of synchronization in the center, set to zero. After subdividing each cycle in 256 bins, these were added up separately for each ENG. The bin containing the greatest sum determines the scale maximum on each ENG display.

When averaging ENGs on the TriLa inflection point that was not necessarily clear in each cycle, the selected events included the TriLa burst onset as the initial reference point. Using a “wrap-around” technique, the TriLa inflection point was reassigned a phase value of zero; the other events were also reassigned new phase values by subtracting the same quantity from their old phase values.

Finally the control and perturbed cycle averages were displayed together, on the same reference point, for comparison.

COMPARISON BETWEEN TWO MEAN PHASE VALUES. Paired t-tests were used to compare the mean phase values of two events of interest, across a set of tonic perturbations of one type, and/or their preceding controls within one cat or across cats, depending on the analysis being conducted. The source of this mean phase value was either the Select program (after selecting the event for each individual cycle) or a manual measure on the printed display of the average cycle obtained with the Averaging program.

In each of two cats (cats 112 and 90), eight mean phase values of the events being compared were available from either program. In each cat, the paired t-test led to the same conclusion independently of the source of the mean phase values.

COMPARISON OF THE CRITICAL POINTS FOR TONIC ELBOW EXTENSIONS AND SHOULDER PROTRACIONS. For each cat, the time at which R ClB amplitude diverges above control with elbow extensions was determined from visual inspection of the superimposed averages of the combined elbow extensions and their preceding controls, synchronized on R ClB onset. The time of R ClB premature onset of descent with shoulder protractions was determined similarly. The times of these critical points, expressed as a percentage of the control L TriLo burst, were compared using an unpaired t-test.

RESULTS

General effects of a tonic shoulder retraction or protraction on the locomotor pattern

The effects of a 20° sustained R shoulder retraction and protraction are shown in Fig. 1. Shoulder retraction decreases R TriLo burst duration and amplitude and increases the period of silence between R TriLo bursts. Contralaterally, it reciprocally increases L TriLa burst duration and amplitude and decreases the period of L TriLa silence. Shoulder protraction has opposite effects to shoulder retraction, although with less marked contralateral effects. No cycle duration change occurs. It was 1,013 ± 121 ms (n = 43) during three shoulder retractions compared with 1,009 ± 90 ms (n = 45) during control (forelimb pendent) and 1,199 ± 210 ms (n = 33) during three shoulder protractions compared with 1,182 ± 168 ms (n = 33) during control.

Bilaterally coupled events in the structure of the locomotor ENG bursts define a critical point

The values of burst duration can increase or decrease in absolute terms, but when looking at bilateral reciprocal changes, as is the case here, it is more meaningful to describe these changes in relation to reference points common to both sides. These points do not generally correspond to obvious points such as offsets or onsets of bursts. Rather, one or more reference points located within bursts, and recognizable by their morphological correlates in the burst shapes on both sides, were identified and will now be described. These points allow us to describe the electromyographic (EMG) changes in terms of increase or decrease of bursts in relation to these coupling points; this facilitates the understanding of how the
reciprocal changes are organized to maintain bilateral rhythmicity.

Figure 2 shows the bilateral activity in TriLo, TriLa, and CIB during unperturbed fictive locomotion. TriLo and TriLa are synchronous in time, but their waveforms are different because the maximum amplitudes occur in different phases. The TriLo maximum occurs in the first half of the burst, whereas the TriLa maximum is in the second half. In fact, nearly every TriLa burst has an abrupt amplitude increase marked by an inflection point at about mid-burst. This is less evident in the L TriLa due to a poorer signal to noise ratio. Vertical lines drawn through the R and L TriLa inflection points show two other events occurring simultaneously: ipsilaterally, TriLo begins to decrease in amplitude (usually gradually); contralaterally, CIB begins its rather abrupt descent (onset of shutting off) toward silence. In nine cats, 12/13 recorded TriLa nerves showed this inflection point, whereas it was absent in three cats (5 TriLa nerves). It sometimes did not persist throughout the experiment (4/12 nerves, e.g., Fig. 1). There was no such upward inflection in 20/20 TriLo nerves recorded. This typical contrast between TriLa and TriLo waveforms was seen in 7/10 simultaneously recorded TriLa/TriLo pairs on the same side (6 cats); in 2/10 pairs, there was no TriLa inflection, but TriLa and TriLo still had their amplitudes greatest in their second and first halves respectively; in 1/10 pairs, both had a TriLo-like waveform.

These relationships are even more obvious when averaging as was done in Fig. 3 for 20 consecutive cycles synchronized on the R TriLa inflection point. This inflection point occurred at 51 ± 3% of TriLa (n = 11) or at 55 ± 5% of the simultaneously recorded TriLo (n = 7). When expressed as a percentage of the cycle, this point occurred at 33 ± 6% of the cycle starting with TriLa onset and 33 ± 9% of the cycle starting with TriLo onset. The larger SDs obtained when measuring on the whole cycle suggest that the timing of this inflection point is better reported as a percentage of the extensor burst. We will use the percentage of the TriLo burst as this was the most commonly recorded extensor. The onset of descent of CIB occurred at 67 ± 3% of CIB (n = 6). The mean phase difference between the onset of descent of contralateral CIB and the TriLa inflection point was −0.002 ± 0.028 (n = 5), which was not significantly different from zero (P = 0.86, paired t-test).

We will label the point where increase in R TriLa, decrease in R TriLo and L CIB are coupled events in the control locomotor program as critical point C (Fig. 3). This label is chosen to leave room for other critical points occurring earlier in the cycle and that will be described later.

FIG. 3. Average ENG burst structure in the normalized control fictive locomotor cycle, synchronized on the inflection point of right (R) TriLa (n = 20). Same cat as Fig. 2. The inflection point in R TriLa coincides with the onset of descent in left (L) CIB and onset of decreased amplitude in R TriLo. These coupled events define critical point C, which is labeled in the figure (- - -, phase value of zero). The abscissa is phase value (0–1) in the normalized control cycle. Time base of 100 ms is derived from the real-time value of the mean control cycle. Each ENG is scaled to the maximum of its ordinate, which is in arbitrary units. With respect to the fictive locomotor cycle, stance essentially corresponds to the period of TriLo and TriLa activity, and swing to the period of CIB activity.
Effects of tonic change in forelimb position on locomotor bursts analyzed in relation to critical point C

Data will now be presented on four cats with a clearly identifiable point C. In general, cycle duration was little changed by shoulder protractions, shoulder retractions, and elbow extensions, whereas it was generally prolonged by elbow flexions (Table 1).

Figure 4 illustrates for the cat of Figs. 2 and 3 (L TriLa no longer recorded) the effects of R-sided shoulder protractions and retractions, and elbow flexions and extensions. This is shown by comparing the normalized averaged perturbed (heavy lines) and control cycles (thin lines ± 1 SD) both synchronized on point C, i.e., the R TriLa inflection point (see METHODS).

Shoulder protraction increases the duration and amplitude of the ipsilateral extensor R TriLo and the contralateral flexor L ClB and reciprocally decreases R ClB and L TriLo durations. Note that R TriLo being a shoulder retractor and elbow extensor would be stretched by the shoulder protraction whereas R TriLa (elbow extensor) would not. Consequently, R TriLa duration remains unchanged, but its amplitude is decreased. When looking at changes in relation to the R TriLa inflection point, it can be seen that R TriLo and L ClB are prolonged because they start earlier with respect to the trigger point C, while they finish at the same time. Furthermore R ClB and L TriLo are shortened because they end earlier, while they start at the same time.

Shoulder retraction has the opposite effects of shoulder protraction. It decreases R TriLo and L ClB durations and amplitudes and increases R ClB duration and amplitude and L TriLo duration. R TriLa duration remains unchanged so that R TriLa starts later and then becomes markedly attenuated after completion of an initial component at a time earlier than point C. On the other side, L ClB is shortened because it starts later, but its amplitude is preserved, whereas L TriLo is increased.

Thus many effects of elbow extension resemble those of shoulder retraction. However, elbow extension decreases R TriLo amplitude, whereas shoulder retraction increases it. R TriLo amplitude is decreased by both perturbations. Further, unlike shoulder protraction versus shoulder retraction, elbow flexion and elbow extension do not simply have opposite effects. In Fig. 4, R TriLo/R TriLa prolongation with elbow flexion and shortening with elbow extension do not occur in the same portions of these bursts, with respect to the R TriLa inflection point. Also, elbow extension has more marked contralateral effects than elbow flexion.

To evaluate the consistency of the R TriLa inflection point and the onset of descent of L ClB, we computed their mean phase differences separately for each tonic R forelimb position. These differences were 0.006 ± 0.016 for shoulder protractions (n = 5), −0.001 ± 0.012 for shoulder retractions (n = 6), 0.004 ± 0.010 for elbow flexions (n = 3), −0.010 ± 0.004 for elbow extensions (n = 4), and 0.002 ± 0.022 for the pendent position preceding these perturbations (n = 18). These phase differences between R TriLa inflection and L ClB onset of descent were not significantly different from zero when the R forelimb was pendent, protracted, retracted, or flexed (paired t-test, P = 0.76, 0.43, 0.97, and 0.82, respectively). Although the phase difference was perhaps significant for elbow extensions (P = .012), it corresponded to the onset of descent of L ClB preceding the R TriLa inflection by only 1% of the cycle duration. Thus this statistical analysis largely confirms the impression from Fig. 4 that these events remain simultaneous across the different R forelimb positions.

To ensure that the changes described in the preceding text in one cat applied to other cats, the results from four cats were averaged and illustrated in Fig. 5. In this figure, average unperturbed ENG bursts are represented as white rectangles

| TABLE 1. Cycle durations for tonic perturbations and their controls |
|----------------------|----------------------|----------------------|----------------------|
|                      | Cat 112              | Cat 90               | Cat 108              | Cat 77               |
| Control              | 916 ± 46 (±3.6)      | 792 ± 42 (±4.0)      | 779 ± 49 (±10.0)     | 719 ± 6 (±1.3)       |
| Protraction          | 883 ± 71 (±5)        | 824 ± 71 (±6)        | 857 ± 52 (±4)        | 710 ± 168 (±2)       |
| Control              | 883 ± 25 (±0.2)      | 787 ± 21 (±1.4)      | 756 ± 37 (±1.2)      | 719 ± 6 (±14.2)      |
| Retraction           | 881 ± 18 (±6)        | 776 ± 21 (±3)        | 765 ± 41 (±4)        | 617 ± 79 (±2)        |
| Control              | 911 ± 21 (±3.4)      | 906 ± 134 (±15.7)    | 797 ± 46 (±8.3)      | 685 ± 42 (±15.2)     |
| Flexion              | 880 ± 37 (±3)        | 1048 ± 194 (±2)      | 863 ± 60 (±3)        | 789 ± 2 (±2)         |
| Control              | 925 ± 11 (±6.7)      | 1168 ± 28 (±3.9)     | 758 ± 49 (±1.7)      |                     |
| Extension            | 863 ± 7 (±4)         | 1213 ± 117 (±2)      | 745 ± 42 (±3)        |                     |

Cycle durations (means ± SD, ms) for the different types of tonic perturbations (shoulder protraction, shoulder retraction, elbow flexion, and elbow extension) and their respective controls in the pendent position in 4 different cats. The percentage change in cycle duration is indicated (↓ decrease, ↑ increase). The numbers indicated in parentheses apply both to the number of averaged perturbations and their controls. In cat 77, the numbers in parentheses refer to 2 groups each of 4 and 2 shoulder protractions, 5 and 2 shoulder retractions, and 2 and 1 elbow flexion and their groups of controls, as there was no individual control available for each single perturbation.

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whose extremities indicate the mean phase of onset and offset of the four cats. Adjacent shaded rectangles represent the changes in timing and duration for each type of perturbation. In addition, the change in the height of the rectangles relative to control indicates the effect on the average normalized burst amplitude.

The reference event in Fig. 5 (phase 0) is point C. Although the SDs of the phases of burst onset and offset may be large because of differences in the structure of the fictive locomotor cycle between cats, it is clear that the features illustrated in Fig. 4 for one cat are generally confirmed for four cats in Fig. 5. For example, we again see that shoulder protraction shortens R CIB because it finishes earlier and prolongs R TriLo because it starts earlier; whereas elbow flexion produces these changes in duration through a later R CIB onset and a later R TriLo offset. R TriLo and R TriLa amplitudes again change in opposite directions with shoulder protraction or retraction but in the same direction with elbow flexion or extension.

**Critical point for reorganization of the R flexor phase: point B**

Although synchronizing on point C as in Figs. 4 and 5 reveals burst components affected differently by different perturbations, a close examination of burst shape changes in integrated ENG averages such as Fig. 4 gives further insight into individual components.

We now focus on other important features of Fig. 4. With shoulder protraction or elbow extension, the initial part of the control and perturbed R CIB bursts are superimposable and
then diverge as the perturbed R CIB begins its early descent (protraction) or as the perturbed R CIB rises in amplitude (extension). The time of divergence is similar for shoulder protraction and elbow extension (41 and 40% of the control L TriLo burst, respectively), and this critical point is labeled B in Fig. 4.

These results were confirmed in the other cats. The time at which elbow extension increased R CIB amplitude above control showed the smallest SD across cats when expressed as a percentage of L TriLo burst, occurring at 44.3 ± 2.6% of the control L TriLo burst (n = 4 cats, based on 3, 2, 1, 4 extensions from cats 112, 90, 79, and 100; in a 5th cat, elbow extension had no effect). The time to which shoulder protraction advanced the onset of descent of R CIB was 41 ± 6.7% of the control L TriLo burst (n = 5 cats, based on 3, 4, 2, 2, 2 protractions from cats 112, 90, 77, 79 and 55). The timing of these two events was not significantly different (unpaired t-test, P = 0.40), located overall at 42% of L TriLo, which corresponded to 42% of R CIB.

Thus at this critical point B in the locomotor cycle, the R flexor phase may be reorganized in two essentially opposite ways. With elbow extension, R CIB increases in amplitude above control and is ultimately prolonged (Fig. 4, extension). With shoulder protraction, the time of burst decline, which corresponds to point C, is now phase-advanced to this critical point B (Fig. 4, protraction, see also Fig. 6A).

Further evidence for this critical point B occurring at 42% of L TriLo is obviously its symmetrical counterpart defined from R TriLo. A characteristic effect of elbow extension was an initial R TriLo component the amplitude of which was less or even not decreased compared with the rest of the burst (as delimited by the unlabeled vertical line drawn on the left-hand side of Fig. 4, extension, see also Fig. 6B). In superimposed averages of control and perturbed cycles synchronized on R CIB onset (as in Fig. 6B), this initial component during elbow extension ended at 44.8 ± 3.9% of the control R TriLo burst (n = 4 cats). This point is the symmetrical counterpart of the critical point B described in the preceding text at 42% of L TriLo.

In summary, the identified critical points are not at “obvious” locomotor transitions, but within the flexor/contralateral extensor phase. One is point B at 42% of L TriLo/42% of R CIB, the time when R CIB either increases or decreases in amplitude in response to the perturbation. Another is point C at 55% of L TriLo/67% of R CIB, symmetrical to the reference point in Figs. 3 and 4 and corresponding to the normal onset of descent of R CIB.

However, yet another point (labeled point D) that would approximately correspond to the onset of weight transfer to the ipsilateral forelimb in the nonparalyzed animal is the end of R CIB and the onset of R TriLo. This point will be detailed in the companion paper, but this point is also critical for tonic perturbations as will be shown in the following text.

Reorganization of locomotor bursts by tonic change in forelimb position seen as shifts between consecutive critical points

That the onset of descent of R CIB (point C) is advanced to the earlier critical point B by shoulder protraction represents a shift in time of a critical point to the next earlier one in the locomotor cycle. We now show similar shifts between another pair of consecutive critical points: the onset of descent of R CIB (point C again), and the R TriLo burst onset (point D). This is illustrated for shoulder protraction and elbow extension in Fig. 6 from the same cat as Fig. 4. The perturbed and control
R ClB bursts remain superimposable until critical point B is reached. With elbow extension (Fig. 6B), R ClB increases in amplitude above control at this time; further, R ClB is prolonged in such a way that its delayed descent precisely begins at the phase when the control R TriLo burst normally begins. Thus elbow extension shifts (delays) critical point C to a new phase $C'$ that coincides with that of critical point D in the control cycle (Fig. 6B). This latter observation also holds for the prolonged R ClB burst seen with shoulder retraction (Fig. 4). With shoulder protraction (Fig. 6A), there is premature onset of descent of R ClB at point B; further, the R TriLo burst starts earlier precisely at the phase when the control R ClB normally begins its descent. Thus shoulder protraction shifts (advances) critical point C to a new phase $C'$ that coincides with that of critical point B in the control cycle and shifts (advances) critical point D to a new phase $D'$ that coincides with that of critical point C in the control cycle (Fig. 6A).

These shifts between critical points C and D, produced by changes in tonic forelimb position, were further studied statistically in this cat, with R ClB onset as the reference point (as in Fig. 6, A and B). A paired $t$-test between the elbow extensions ($n = 4$) and their controls showed no significant phase difference between the delayed onset of descent of R ClB during elbow extension and the onset of R TriLo burst in the pendent position ($P = 0.61$, Table 2). By contrast, during the same controls, a highly significant phase difference was easily detected between the phases of onset of descent of R ClB and onset of R TriLo ($P = 0.0006$). Shoulder retractions ($n = 6$) had an effect similar to elbow extensions with no significant phase difference between the delayed onset of descent of R ClB during shoulder retraction and the onset of R TriLo burst in the pendent position, although the $P$ value was lower ($P = 0.17$). Finally there was no significant phase difference between the earlier onset of R TriLo burst during shoulder protractions ($n = 5$) and the onset of descent of R ClB in the pendent position ($P = 0.94$).

Similar results were obtained in five other cats besides cat 112 as assessed by paired $t$-test (Table 2). Specifically, in two cats elbow extensions delayed the R ClB onset of descent to a similar phase than the R TriLo burst onset in the pendent position ($P = 0.60$ and 0.70, Table 2), while it was not quite the case for a single elbow extension in a third cat (Fig. 7). The same result held for shoulder retractions in two cats ($P = 0.93$ and 0.57) but not in a third cat ($P = 0.008$) where they had
little effect. Pooling across the cats including cat 112, there was no significant phase difference between the delayed R CIB onset of descent during elbow extension or shoulder retraction and the R TriLo burst onset in the pendent position. This is indicated in Table 2 ($P = 0.21$, $n = 11$ shoulder retractions, omitting the cat with ineffective retractions). Shoulder projections done in four other cats besides cat 112 advanced in all the R TriLo burst onset to a similar phase than the R CIB onset of descent in the pendent position ($P = 0.86, 0.81, 0.83$, and $0.13$ and across cats, $P = 0.43$, $n = 15$ shoulder projections, Table 2).

To determine whether tonic perturbation amplitude had an effect on the shift of critical points, we divided the perturbations from one cat (cat 112) in two groups: small amplitude (mean: $18 \pm 4^\circ$, range: $11–23^\circ$, $n = 7$) and large amplitude (mean: $29 \pm 3^\circ$, range: $24–33^\circ$, $n = 8$). For each perturbation, we computed the phase difference between the relevant critical points: (D’ minus C) for shoulder projections and their controls, and (C’ minus D) for shoulder retractions or elbow extensions and their controls (Fig. 6). Thus for any perturbation a zero phase difference meant a “perfect” critical point shift, a positive phase difference a critical point “under-shift” (e.g., D’ not quite completely advanced to C by a shoulder projection) and a negative one an “over-shift.” The average phase differences for the critical point shifts were $0.005 \pm 0.022$ ($n = 7$) and $0.010 \pm 0.033$ ($n = 8$) for the small- and large-amplitude tonic perturbations, respectively. These means were neither statistically different from each other (unpaired t-test, $P = 0.75$) nor significantly different from zero ($P = 0.58$ and 0.44 respectively). We conclude that the same discrete critical point shifts occur for a range of perturbation amplitude.

We have not recorded from distal nerves, but one instance where we did supports the existence of the same critical points. Figure 7 illustrates a single elbow extension during fictive locomotion with the R deep radial nerve (DR) innervating distal muscles recorded. Again R CIB amplitude rises above control at point B. While the control R DR signal is very weak, the perturbed R DR begins at point C, finishes its ascending

![Figure 7](http://jn.physiology.org/)

**Figure 7.** Average of a single elbow extension and its control, synchronized on L CIB onset of descent (17 control and 22 perturbed cycles, cat 79). The right deep radial (R DR) nerve was also recorded in this cat. The 1st vertical line shows where the perturbed R CIB burst increases in amplitude above control (point B). The 2nd vertical line shows that during elbow extension, the onset of R DR coincides with the onset of descent of R CIB in the control cycle (point C). The 3rd vertical line shows that during elbow extension, the end of the ascending phase of R DR coincides with the onset of R TriLo in the control cycle (point D). The 4th vertical line shows that during elbow extension, the offset of R DR coincides with the end of an initial component in the control R TriLo burst. DR has a poor signal in the control cycle. In this cat, the delayed onset of descent of R CIB during elbow extension (C’) does not quite reach the time of onset of R TriLo during the control cycle (D).

**TABLE 2. Results of paired t-tests examining shifts between critical points C and D**

<table>
<thead>
<tr>
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<th>Elbow Ext.</th>
<th>Shoulder Retr.</th>
<th>Shoulder Protr.</th>
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<tbody>
<tr>
<td>Cat 55</td>
<td>—</td>
<td>0.93 (3)</td>
<td>0.86 (2)</td>
</tr>
<tr>
<td>Cat 77</td>
<td>—</td>
<td>0.57 (2)</td>
<td>0.81 (2)</td>
</tr>
<tr>
<td>Cat 79</td>
<td>NA (1)</td>
<td>—</td>
<td>0.83 (2)</td>
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<tr>
<td>Cat 90</td>
<td>0.60 (2)</td>
<td>no effect</td>
<td>0.13 (4)</td>
</tr>
<tr>
<td>Cat 100</td>
<td>0.70 (2)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Cat 112</td>
<td>0.61 (4)</td>
<td>0.17 (6)</td>
<td>0.94 (5)</td>
</tr>
<tr>
<td>Pooled perturbations</td>
<td>0.87 (9)</td>
<td>0.21 (11)</td>
<td>0.43 (15)</td>
</tr>
</tbody>
</table>

$P$-value results of paired t-tests examining shifts between critical points C [onset of descent of clidobrachialis (CIB)] and D [onset of long head of triceps (TriLo)] in response to tonic proprioceptive perturbations. The 2nd column tests whether there is a significant phase difference between the delayed onset of descent of right (R) CIB during elbow extensions (C) and the onset of R TriLo burst in the pendent position (D). The 3rd column tests the same for shoulder retractions. The 4th column tests whether there is a significant phase difference between the earlier onset of R TriLo during shoulder protraction (D’) and the onset of descent of R CIB in the pendent position (C). The number of perturbations and their controls is indicated in parentheses for each individual cat and in the last row for the pooled perturbations. —, the absence of data for that perturbation type; NA, a paired t-test could not be conducted for a single elbow extension. In cat 55 where R TriLo was not recorded, its onset in the pendent and protracted positions was respectively estimated as the onset of R TriLa and the end of R CIB in those positions. In cat 77, n refers to 2 groups each of shoulder protraction and retractions as explained in Table 1. In cat 90, shoulder retractions had little effect (see text), whereas 2 shoulder protractions done later in the experiment at a time when they frequently slowed down the rhythm markedly and had an atypical elbow flexion-like effect were not included.
phase at point D, and stops at the end of the initial component in the control R TriLo burst.

**DISCUSSION**

This paper reports the changes in the characteristics of the forelimb fictive locomotor cycle when the shoulder of one limb is protracted or retracted or the elbow flexed or extended. The central finding is an integration of locomotor rhythm and posture, based on the existence of critical points in the cycle. Besides a critical point at the end of the ipsilateral flexor phase/onset of ipsilateral extensor phase, point D, two other critical points are located within these phases. In particular, we find a critical point in the ipsilateral extensor phase and the contralateral flexor phase that remain bilaterally coupled, point C, and a critical point in the ipsilateral flexor phase at which flexor burst modification occurs, point B. It is also suggested that these critical points are disposed on a temporal "grid" and that changes in locomotor burst duration produced by tonic proprioceptive inputs occur through discrete shifts between consecutive critical points disposed on that fixed grid.

**Morphological correlate of bilaterally coupled critical points in the locomotor burst structure**

The control locomotor cycle shows simultaneously an ipsilateral TriLa upward inflection, and an ipsilateral TriLo and contralateral ClB onset of descent at 55% of ipsilateral TriLo burst (Figs. 2 and 3). To our knowledge such bilateral ENG coupling has not been reported.

Previous work has not focused on burst shape. During fictive locomotion, some extensor motoneurons (including triceps, head not specified), show an upward inflection at about mid-burst (Göddez et al. 1990). In turtle fictive scratching, some knee extensors have a TriLa-like inflection at about mid-burst, while others are TriLo-like (Currie and Lee 1996). The intact cat TriLa shows no inflection, but TriLo amplitude drops after midstance, unlike TriLa (Drew and Rossignol 1984, 1987), reminiscent of our findings. Shape differences are expected between fictive and intact locomotion given the added cyclical proprioceptive feedback in the latter. In the intact cockroach, unloading of the limb signaled by force receptors at swing onset adds an element to the centrally generated flexor burst to obtain its final shape (Zill et al. 2001). Burst shape may also change during different forms of intact locomotion. In particular, the shape of the vastus lateralis (VL) burst appears very different in forward and backward cat locomotion (Buford and Smith 1990; their Fig. 2), although the burst timing remains the same. In forward locomotion, VL has a TriLa-like shape, whereas in backward locomotion, it has a TriLo-like shape. Given our evidence for different components within the extensor bursts, one may ask whether these burst shape changes could arise through a reorganized recruitment of sets of interneurons underlying these components.

**Effects of tonic change in forelimb position on locomotor bursts**

Preserved coupling between ipsilateral TRILA inflection and contralateral ClB onset of descent during tonic perturbations and its postulated role in interlimb coordination (point C). Critical point C corresponds to a morphologically recognizable inflection in R TriLa and to the onset of descent in L ClB, thus linking the extensor and flexor on both sides. Our results suggest that this bilateral coupling is centrally programmed and contributes to maintaining interlimb coordination during locomotion occurring under various postures.

Such intraburst coupling might help the spinal cat to preserve 1:1 coordination on split-belts of different speeds. The midpoints of the quadriceps (Q) bursts maintained a tighter alternation than their onsets or offsets (Forssberg et al. 1980). The Q bursts had a TriLa-like waveform, but this or a possible coupling of the Q midpoint with contralateral flexors was not discussed.

The burst duration changes with tonic perturbations are those expected for a negative feedback scheme (e.g., shoulder protraction increases ipsilateral protractor and extensor TriLo and shortens ipsilateral retractor and flexor ClB). The coordinated contralateral changes (e.g., both ipsilateral TriLo and contralateral ClB increase with shoulder protraction) may be a consequence of the preserved coupling.

Von Holst (1973) suggested that a preferred phase relationship between two rhythms, or position of coaction, underlies their coordination: when drifting apart from that position, the rhythm ahead decelerates and the rhythm behind accelerates. With shoulder protraction, during the R extensor phase, the R central program is ahead of the presumed peripheral movement signaled by proprioceptive inputs. Thus during shoulder protraction, the prolonged R TriLo first component would represent deceleration of the "ahead" central rhythm, the increased R TriLo duration would help accelerate the "behind" peripheral rhythm. L ClB onset occurs earlier than normal during shoulder protraction (Fig. 4), which with the preserved coupling described in the preceding text, prolongs L ClB prior to its descent. This would represent deceleration of the "ahead" L central rhythm with respect to the R central one. Therefore the position of coaction between R- and L-sided forelimb locomotor rhythms could be the coupling between R TriLa inflection and L ClB onset of descent. The coordination role we had postulated for this preserved coupling agrees with Von Holst’s view of the position of coaction being crucial for coordination.

**Extensor phase prolonged in a different way by shoulder protraction and elbow flexion.** Synchronizing on point C indicates that shoulder protraction prolongs R TriLo because it starts earlier and elbow flexion because it finishes later. In treadmill locomotion, hindlimb stance may be prolonged by preventing the hip from reaching a critical angle or by maintaining a load on the ankle extensors (see INTRODUCTION). In fictive locomotion, shoulder protraction and elbow flexion might simulate these two methods of prolonging stance, with the additional result that the extensor phase is prolonged in a different way by the two perturbations.

**Critical point for reorganization of the R flexor phase (point B)**

We found a critical point B at which R ClB amplitude rose above control with elbow extensions and to which the R ClB onset of descent was advanced by tonic shoulder protractions. This point B is clearly before critical point C. This suggests three subdivisions to the flexor phase: onset of R ClB to point B; point B to point C; point C to end of R ClB.
The notion of flexion neural subphases is recent unlike the established kinematic subphases (F and E1) (Philippson 1905). From locomotor variations and cutaneous reflex modulation, Burke et al. (2001) suggested two or three subdivisions of fictive hindlimb flexion. Thus flexor digitorum longus is typically active during the first 1/3 of flexion, without overlap with extensor digitorum longus, a mid- to late flexor, and their durations can vary independently.

In intact forelimb locomotion, Drew (1993) also indicated that "flexor muscles are not all activated simultaneously, but rather in a strict temporal order." We only recorded CIB as a flexor. Nevertheless, its two critical points, B at 42% where a small notch or upward inflection in amplitude is sometimes seen in the burst (Figs. 4 and 6), and C, its onset of descent, appear to coincide in Drew (1993) with the onset of descent and the end of teres major (shoulder retractor), respectively. Further, extensor digitorum communis (EDC, wrist and digit extensor) ascending phase begins and ends at points C and D, whereas extensor carpi radialis (ECR, wrist extensor) ascending phase finishes at C. During obstacle locomotion, EDC bursts twice rather than once (Drew 1993; Drew et al. 1996) with the first burst ascending phase advanced to the B-C interval and the second burst onset delayed to D. Thus our critical points do seem to relate to important subdivisions of flexor phase activity throughout the forelimb musculature, including distal activity (Fig. 7).

During obstacle locomotion, many pyramidal tract neurons enhanced discharges seemed restricted to periods from points B to C of the modified ipsilateral CIB burst or from point C to burst offset (Drew 1993; his Figs. 7 and 8). Although related to modified wrist and digit dorsiflexors, these discharge periods could more generally indicate a key role of critical points in mediating proximal-distal forelimb musculature coordination by the motor cortex. Van Kan and McCurdy (2001a,b) recently demonstrated precisely timed interpositus and red nucleus discharges to coordinate distal (hand preshaping) with proximal musculature during reaching to grasp.

On split-belts, coordination adjustments occur primarily in the mid- and late parts of the swing phases of both limbs (Forssberg et al. 1980). This early swing "sparing" resembles our R CIB remaining initially identical to control, during elbow extension or shoulder protraction (Figs. 4 and 6).

**Discrete shifts between critical points**

How do the tonic perturbations reconstruct the locomotor cycle? A key result is the shifts between critical points. Point C is phase-advanced by shoulder protraction to coincide with point B in the control cycle (Figs. 4 and 6A), the time at which elbow extension increases R CIB amplitude above control (Figs. 4, 6B, and 7). Similarly, shoulder protraction advances point D (R TriLo onset) to coincide with point C in the control cycle (Fig. 6A); conversely, elbow extension or shoulder retraction delay point C to coincide with point D in the control cycle (Figs. 6B and 7). Because perturbations amplitudes were not varied systematically, the possibility of gradual rather than discrete critical point shifts remains. The bilateral coupling at point C, preserved despite perturbations, the same point B for shoulder protractions and elbow extensions, the same critical points for phasic perturbations effects in the companion paper, all argue for discrete points. Further we documented in one cat the same critical point shifts for two different ranges of perturbation amplitudes.

The C-to-D delay by elbow extension, with no change until point B, represents prolongation of the B-C interval to the duration of B-D. A preserved temporal structure is necessary to account for that exact degree of prolongation. Although the C to B advance by shoulder protraction represents a B-C interval deletion, the D to C advance by shoulder protraction does not simply follow from this deletion because the control B-C and C-D durations are not necessarily equal. A preserved temporal structure again needs to be postulated.

In summary, in each cycle, events (e.g., burst onsets and offsets, inflection points, and onset of descent) occur in a certain sequence. Because tonic perturbations shift the times of some events to coincide with the position which earlier/ later events of the sequence occupied in the control cycle, the same temporal structure must be operating in the control and perturbed situations. To reconstruct the cycle, tonic perturbations do not alter the sequence or the temporal structure but shift the sequence with respect to the temporal structure in specific portions of the cycle, resulting in burst duration changes.

Tonic perturbations may also affect burst amplitude selectively. In particular, elbow extensions increase R CIB, or depress markedly R TriLo amplitudes only past a first component at point B of the control cycle (Figs. 4, 6B, and 7). Tonic proprioceptive inputs can first, therefore shift locomotor sequence components with respect to an unchanged temporal structure and second, modulate burst component amplitude.

Proprioceptive projections include motoneurons, premotor interneurons, and CPG interneurons (Lam and Pearson 2001). According to Burke et al. (2001), burst-amplitude changes would reflect a premotoneuronal effect, downstream of both a timing network, and a pattern shaper. Lennard and Hermanson’s (1985) postulated a model comprising a rhythm-producing central timing network (CTN) and an intracycle pattern generator (ICPG), determining burst phase relationships. In their model, brief electrical stimuli of cutaneous nerves during real turtle monopodal swimming modified the CPG coupling. Our postulated temporal structure has features of both the CTN and ICPG. Although not dictating fixed phase relationships between bursts as the ICPG, it constrains where critical points delimiting locomotor subphases may be located in the cycle.

In a theory (although not at its core), Strehler (1990) suggested periodic updating within a movement of its vector, with opportunity for corrections limited to those times. Even in unperturbed locomotion, some movement trajectory modification is necessary from one subphase to the next, which could be considered an update, even though not a "correction." Thus already for the control cycle, we can envision the critical points as times where the movement needs to be updated for the next subphase with the temporal grid dictating these update times as in Strehler’s theory. During tonic proprioceptive perturbations, the same update times are still the ones constraining where a subphase may end or may be modified in amplitude because these times are where corrections are allowed by definition. This would explain the discrete shifts between critical points.
Biomechanical correlates of critical points and of the effects of tonic perturbations

In this paralyzed preparation, the tonic perturbations effect on the limbs spatiotemporal relationships is unknown. Perhaps the periods with a different control and perturbed burst time course, achieved through shifting the locomotor sequence with respect to the temporal structure, are aimed at restoring normal spatial relationships between the limbs (Hasan 1992; Soechting 1988; Verschueren et al. 1999). The preserved temporal structure would indicate that the cause of the altered spatiotemporal relationship is spatial not temporal. Here we argue that the critical points undergoing these shifts correspond to biomechanically important transitions in locomotion and subserve a biomechanically meaningful integration of the CPG with tonic proprioceptive inputs.

Biomechanical correlates of the coupled TrilA inflection and contralateral ClB onset of descent (Critical point C). These coupled events occur at 55% of the extensor (TriLo) phase.

Manter’s work (1938) on intact cat locomotion shows several events at 57% of forelimb stance (his Figs. 4 and 7). These events are: an upward inflection in vertical force amplitude, horizontal ground reaction force reversal from backwards to forwards, ipsilateral elbow passage above the foot (forearm vertical), and contralateral elbow extension onset during swing in preparation for landing (E1 phase onset). Thus we suggest that the simultaneous events at 55% of the fictive extensor phase (TriLa inflection and contralateral ClB onset of descent) are electromyographic equivalents of this transition in the intact cat.

The preserved bilateral coupling suggests that under different postural circumstances vertical passing by the forearm keeps the biomechanical significance of contralateral landing preparation. The unchanged ipsilateral TriLa timing relative to its inflection during the asymmetrical shoulder protraction or retraction cycle (Fig. 4), raises the question of whether in real asymmetrical locomotion (e.g., turning), limb excursion about the vertical remains constant. In quiet stance, cats prefer to keep their limbs vertical (Lacquaniti et al. 1984, 1990).

More generally, matching critical point C events with biomechanical events at 57% of stance could represent a phase of coaction between the CPG and the periphery, useful for locomotor adaptation to the external world.

Different effect of shoulder perturbations on ipsilateral TrilO and ipsilateral TriLa amplitudes. TriLa, unlike TriLo, is mono-articular with no shoulder action. That shoulder perturbations modify TriLa amplitude supports recent findings that mono-articular muscle recruitment does not depend only on torques at the joint on which it is acting (Gribble and Ostry 1999; Hasan 1992; Nozaki and Nakazawa 2001).

Shoulder protractions increase TriLo and decrease TriLa amplitudes and vice versa for shoulder retractions. Opposite TriLo/TriLa effects have been seen in other experiments. Headward translation of a standing platform in alert cats inhibited TriLa and excited TriLo (Macpherson 1988; Rushmer et al. 1983). Although this perturbation caused limb protraction, it was through scapular rotation with unchanged shoulder and elbow angles; the TriLa and TriLo responses were attributed to unloading of the forelimbs. In cerebrerate, labyrinthine-
tomized cats, neck ventrifthexion excited TriLo, while neck dorsiflexion excited TriLa (Wilson et al. 1986). Such TriLo excitation would again occur in a situation of vertical unloading of the forelimb, e.g., during upward tilt of a standing platform, where the animal may ventrifthexion its neck to keep the head level; TriLa would be activated in the opposite situation.

Shoulder protraction might thus simulate a real locomotion situation of forelimb vertical unloading. The forelimb musculature likely detects events related to interaction between the locomoting limbs rather than just local limb manipulation. The effects of shoulder protraction during fictive locomotion might represent the adaptation to the common center of pressure of the forelimb and hindlimbs being shifted posteriorly. Increasing retractor activity (TriLo) would help return that common center of pressure forward. Decreased TriLa amplitude would reflect decreased vertical loading of the forelimb.

Biomechanical correlate of critical point B. We suggested earlier that at point B (42% L TriLo), R teres major (retractor) starts shutting off while R ClB (protractor) continues. Further, ~40% of L forelimb stance is when the swinging R humerus starts to protract after an initial retraction (Manter’s Fig. 4). This might explain why shoulder protraction advances R ClB (protractor) onset of descent to this point (Figs. 4 and 6A). Contralateral TriLo and ipsilateral elbow angle recordings (Drew and Rossignol 1984, 1987; Udo et al. 1980) also suggest that elbow flexion stops at point B. With cerebellar cooling, the elbow angle trajectory shows hyperflexion only after this point (Udo et al. 1980; their Fig. 6) reminiscent of elbow extension increasing R ClB (elbow flexor) amplitude above control only after point B (Figs. 4 and 6B).

Perturbations do not simply evoke locally resisting responses because ClB remains unchanged by elbow extension during a period of elbow flexion. As the shoulder perturbations effects on TriLa amplitude also suggested, more globally meaningful variables are controlled in locomotion (Gurfinkel et al. 1988). During early swing, humerus retraction clears the foot from the ground. Responding to an elbow extension during this period with increased ClB amplitude (protractor) would be inappropriate. Once humerus protraction starts, such response to elbow extension appropriately ensures sufficient elbow flexion (limb sufficiently short) for the foot not to hit the ground during swing. Thus critical point B appears useful for intralimb coordination where the appropriate response to an elbow perturbation (extension) is timed to coincide with what is happening at the shoulder.

In summary, we suggest that bilateral linkage of critical points represents a means of synchronization across the cord to maintain a 1:1 bilateral coupling during normal and perturbed locomotion. Consecutive critical points are also linked "seri-ally" so that shifts between them allow adaptation of the cycle structure to proprioceptive inputs. These critical points correspond to key biomechanical events that have been incorporated in the central program.

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Tonic forelimbs perturbations in fictive locomotion


