Critical Points in the Forelimb Fictive Locomotor Cycle and Motor Coordination: Effects of Phasic Retractions and Protractions of the Shoulder in the Cat

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Saltiel, Philippe and Serge Rossignol. Critical points in the forelimb fictive locomotor cycle of the cat and motor coordination: the effects of phasic retractions and protraction of the shoulder. J Neurophysiol 92: 1342–1356, 2004; 10.1152/jn.00564.2003. This study investigates the responses to phasic shoulder retractions or protraction given at different times in the fictive locomotor cycle of the forelimbs of decerebrate cats. Generally, the responses in flexor and extensor muscles acting at the shoulder or elbow were bilaterally coordinated according to a negative feedback scheme. Perturbations in the direction of the movements that would have taken place if the animal had been paralyzed tended to shorten the duration of the burst of activity of the muscles active during that phase and vice versa in the opposite phase. Changes in response patterns took place around critical points corresponding to the critical points B–D described in the companion paper using tonic perturbations of the limb. Past point C, at 58% of the ipsilateral extensor burst, protraction no longer prolonged the burst and no longer delayed onset of the contralateral extensor. At point B, occurring at 41% of the contralateral extensor burst, ipsilateral protraction maximally shortened the ipsilateral flexor phase, advancing ipsilateral extensor onset (point D) to point C of the contralateral extensor burst. During a critical period from the end of the ipsilateral flexor (point D) until the contralateral flexor onset, retractions elicited two alternative responses. Either the contralateral extensor activity was abolished and the contralateral flexor turned on, or it persisted for another cycle. We argue that the critical points found here correspond to critical biomechanical events in real locomotion and may underlie a phase-dependent motor coordination.

INTRODUCTION

For real-world adaptation, the central locomotor program interacts not only with exteroceptive inputs (Rossignol et al. 1988) but also with phasic proprioceptive inputs from the limbs (Grillner 1981; Rossignol 1996). The latter may arise from encountering obstacles or from changes in limb velocity during movement. Furthermore, the locomotor program must be capable of adjusting both limbs of the same girdle when one limb is perturbed so that progression and equilibrium are preserved.

Proprioceptive afferents generally have similar effects in decerebrate walking cats and fictive locomotion. Group I afferents from hindlimb extensor muscles facilitate the extensor phase (Conway et al. 1987; Gossard et al. 1994; Guertin et al. 1995; Hiebert et al. 1995; McCrea et al. 1995; Whelan et al. 1995) with some parcelling in how afferents from different extensors distribute their effects to different extensor muscle groups (Hiebert and Pearson 1999). Group Ia afferents from hindlimb flexor muscles facilitate the flexor phase (Degt-yarenko et al. 1998; Hiebert et al. 1996; Lam and Pearson 2001; Perreault et al. 1995; Quevedo et al. 2000). For example, in the fictive preparation, stimulating electrically hindlimb group I extensor muscle afferents during the ipsilateral extensor burst prolonged it as well as the contralateral flexor burst; the same stimulation during the ipsilateral flexor burst shortened it. The responsible afferents were believed to be group Ib from extensor muscles (Conway et al. 1987).

These studies identifying relevant proprioceptive inputs did not “dissect” the locomotor cycle beyond flexion and extension. One notable exception is the pioneering work of Andersson and Grillner (1981). In cat spinal fictive locomotion, recording primarily from distal and ipsilateral hindlimb nerves, they found ipsilateral extensor burst shortening by phasic hip extension (negative feedback) and flexor burst prolongation by hip flexion (positive feedback). Interestingly, effects often differed in the early versus late part of a given (flexor or extensor) phase. However, except for one obviously abrupt transition in effect seen for hip extensions, one could generally not say whether the changes in sensitivity to proprioceptive inputs were discrete or gradual.

In the companion paper, we found that tonic shoulder and elbow perturbations of one forelimb during fictive locomotion changed the cycle structure (relative duration of flexor and extensor bursts) and burst amplitude in relation to certain critical points in the cycle (labeled B–D). Furthermore these points generally had a correlate in the burst morphology. For example, point C consisted of the simultaneous occurrence of an ipsilateral lateral head of triceps burst (TriLa, elbow extensor) inflection point and the contralateral cleidobrachialis burst (CIB, shoulder protractor and elbow flexor) onset of descent. The coupling of these events remained preserved with tonic perturbations, suggesting its importance in bilateral coordination.

Furthermore these critical points corresponded to important biomechanical events in real locomotion. They should therefore influence the locomotor program responses to phasic proprioceptive inputs. Here we investigate this with phasic shoulder protraction and retractions during forelimb fictive locomotion. We used cuff electrodes to record from nerves in...
any position of the limb. All other nerves were left intact to approximate the effects of such perturbations in intact animals.

**METHODS**

Ten cats were used in this study and prepared as described in the companion paper. Briefly, cats were anesthetized and decerebrated (1 cat was decorticated). Bilateral cuff electrodes around forelimb flexor and extensor nerves recorded fictive locomotion after paralysis.

**Stimulation**

A computer-controlled motor and gear arrangement was designed to ensure constant perturbation amplitudes within a given series. A metal bar attached a mid-humerus screw to a motor-controlled wheel. Either the point of this attachment or wheel-rotation amplitude determined perturbation amplitude. Controlled parameters included wheel rotation direction and amplitude, the length of time at the new position before returning to the initial position, and the speed for reaching the new position. Delivery time was controlled by triggering the motor on the onset of activity in selected nerves.

From its pendent position, the right shoulder was rapidly protracted or retracted (15–20° angle change over 160 ms, i.e., 94–125/s; the other joints remaining fixed) and generally held there for 1–2 s before returning to the original position. Thus the studied cycle, i.e., the one perturbed by bringing the shoulder to its new position, was already completed before returning to the original position.

**Data analysis**

Right shoulder and elbow angles and amplified electromyograms (ENGs) (filters 300 Hz to 10 kHz) were recorded on magnetic tape (Honeywell 101, frequency response: 0–2.5 kHz). The data were replayed on a Gould recorder and also digitized at 1 kHz and stored on disk.

To analyze the effect of the perturbations, we selected time intervals of interest (e.g., ipsilateral extensor cycle duration, contralateral extensor cycle duration, interval from ipsilateral extensor onset to contralateral extensor onset) and plotted their duration against the time of perturbation onset within the interval. These measurements were done either manually on Gould paper (5- to 10-ms accuracy) or with the computer by displaying the digitized bursts on a screen and selecting the bursts onsets and offsets within the perturbed cycle as well as the perturbation onset times with a cursor (Zomlefer et al. 1984). For each perturbed cycle, a program produced the time of the selected events with respect to one of them chosen as the reference. It also computed the integrated amplitudes of the selected bursts.

For each series of perturbations, the selected time intervals were also measured for 10–15 consecutive cycles during a control period with the forelimb pendent and their means ± SD computed.

**RESULTS**

**General effects of phasic shoulder retractions and protractions**

Figure 1 illustrates the effects of phasic shoulder perturbations on the fictive locomotor cycle. In this example with only bilateral extensors recorded, the silent phase is referred to as the flexion.

Retraction of the right (R) shoulder during the R extensor burst (a phase of forelimb retraction in the nonparalyzed animal) shortens it as well as the left (L) flexor phase. Retraction during the R flexor phase prolongs it as well as the L extensor burst.

Shoulder protraction has opposite effects. Protraction during the R extensor burst prolongs it as well as the L flexor phase. Protraction during the R flexor phase shortens it as well as the L extensor burst.

Thus each type of perturbation evokes bilaterally coordinated effects, which depend on the phase of application. The points in the cycle where changes in response pattern occurred did not always correspond to simple events such as bursts onset or offset. To identify these points, a detailed description of the effects of perturbations applied at all phases of the fictive locomotor cycle is needed.

**Phasic shoulder retractions**

The data are from eight cats. Two cats in which the retracted limb was almost immediately returned to its original position (Figs. 1 and 5), i.e., somewhat differently than in METHODS, are not included in plots of cycle duration.

Figure 2 illustrates the effects on the ENG activity of shoulder retractions applied at different points in the fictive locomotor cycle. These effects are also summarized schematically in Fig. 3.

Shoulder retraction during activity of R ClB, protractor and flexor muscle, prolongs it as well as the concomitantly active contralateral L TriLo (long head of triceps, retractor and extensor; Figs. 2, A and B, and 3, A and B). Retractions near the end of R ClB cause its greatest prolongation (Figs. 2B and 3B). Retraction immediately after R ClB, just before or at R TriLo onset (retractor and extensor) can elicit two different “strategies” (Figs. 2, C and E, and 3, C and E). In strategy 1, R TriLo is moderately shortened, whereas L TriLo, although somewhat prolonged (Figs. 2C, 3C, and 5A), is replaced by a L ClB burst (Fig. 2C), thus maintaining the normal forelimb alternation. In strategy 2, R TriLo is aborted and rapidly gives way to a second R ClB burst, whereas throughout this period, L TriLo maintains its activity (Figs. 2E and 3E), establishing temporarily a 2:1 rhythm. Retractions very early in the extensor phase, before about halfway between R TriLo onset and L TriLo offset (Figs. 2, D and F, and 3, D and F), elicit the same two strategies. Retractions later in the extensor phase, although still before L TriLo offset (Figs. 2G and 3G) can no longer prevent the switch from L TriLo to L ClB. They shorten both R TriLo and L ClB bursts as do later retractions (Figs. 2H and 3, H and I).

Thus a critical period exists from R ClB offset till L ClB onset, during which shoulder retractions can elicit two quite different response patterns (strategies). A more precise description requires plotting of cycle and burst durations according to retraction time.

**EFFECTS OF SHOULDER RETRACTIONS ON CYCLE DURATION: UNCHANGED CYCLE DURATION AT ~58% OF THE EXTENSOR PHASE, AND A CRITICAL PERIOD AROUND EXTENSOR BURST ONSET.** Figure 4A shows a roughly linear relationship between cycle duration and shoulder retraction time measured from R TriLo onset, although the graph is more complex at its extremes (i.e., around R TriLo burst onset). Early retractions shorten the cycle, whereas later retractions prolong it. It is at 51% of the R TriLo burst, that the control (dotted horizontal line) and perturbed cycles are equal. In the four other retraction series with a
symmetrical control rhythm, cycle duration was unchanged by retractions at 52, 60 and 64% of the R TriLo burst (in the fourth series, cycle period was hardly changed by retractions at any time). Therefore R shoulder retractions had no effect on R TriLo cycle duration when applied at 57/6% of the R TriLo burst (n = 4).

Shoulder retractions near the end of R CIB may abruptly no longer prolong the cycle, with return to control value (right extreme of graph). This is because these retractions may no longer prolong the R CIB burst (see also later Fig. 6A).

Shoulder retractions very early in R TriLo, before L CIB onset, can cause variable changes in cycle duration ranging from very shortened to nearly control values (left extreme of graph). This corresponds to the critical period during which different strategies can occur. In strategy 2, R TriLo burst is short, and followed by a second unprolonged R CIB burst (Fig. 3, E and F) resulting in a much shortened R TriLo cycle duration. In strategy 1, R TriLo burst shortening is less pronounced (Fig. 3, C and D), while the subsequent R CIB is prolonged by the tonic component of the retraction, resulting in a R TriLo cycle duration close to control.

Figure 4B examines cycle duration for the same series of shoulder retractions but measured now from the contralateral (L) TriLo onset. In this instance, the critical period now lies in the center of the graph, showing a bifurcation, whereas outside of it, L TriLo cycle duration is linearly related to retraction time. Before the critical period, the L TriLo and R CIB bursts prolongation becomes greater, the later the retraction is applied during R CIB. The critical period begins at the end of R CIB, when retraction no longer prolongs it, at which point L TriLo burst (and cycle) duration may change in two possible ways. In strategy 1, L TriLo burst duration decreases to a value only slightly prolonged above control, resulting from the increased overlapping R TriLo/L TriLo activities (Fig. 3D), and this...
gives the downward branch of the bifurcation in Fig. 4B. In strategy 2, L TriLo burst becomes markedly prolonged as its activity is maintained during the temporary 2:1 rhythm (Fig. 3F), and this gives the upward branch of the bifurcation. After the critical period, retractions shorten L TriLo silence and R TriLo burst, with the degree of shortening diminishing as retraction is applied later during R TriLo.

In Fig. 4A, both the highest points on the left and the lowest points on the right have perturbed R TriLo cycle durations close to control, while the shortest R TriLo cycle values on the left and the longest ones on the right differ by a duration close to one control cycle. Similarly in Fig. 4B, the perturbed L TriLo cycle values of the highest and lowest points of the bifurcation differ by one control cycle duration. Thus duplicates of Fig. 4A translated by one control cycle duration will link to each other and reproduce the result shown in Fig. 4B. The similar adjustment of R and L TriLo cycle durations to phasic shoulder retractions reflects preserved coordination between the two forelimbs.

In different series, shoulder retractions during the critical period may elicit both strategies 1 or 2 (both branches of the bifurcation are seen in the L TriLo cycle duration plot) or only strategy 1 (only the downward branch is seen) or only strategy 2 (only the upward branch is seen). Tonic contralateral (L) forelimb position influences the choice of strategy. In Fig. 5, A and B, the L forelimb was kept retracted and in C and D, protracted. In Fig. 5, A and C, phasic shoulder retraction is applied just before R TriLo and in B and D, just after R TriLo onset. In Fig. 5A, R TriLo is not aborted and L TriLa is silenced, though with some delay (strategy 1). In Fig. 5C, R TriLo is aborted and L TriLa is prolonged for an additional cycle till the next R TriLo burst onset (strategy 2). In Fig. 5, B and D, the R TriLo bursts are similar, but again L TriLa becomes silent in B, while it is prolonged for an additional cycle in D. Thus contralateral forelimb retraction and protraction respectively favored strategy 1 and 2 responses to retractions during the critical period. In other cats, one of the two strategies was similarly favored although not to the exclusion of the other one as in Fig. 5.

**EFFECTS OF SHOULDER RETRACTIONS ON BURST DURATION: LOSS OF EFFECT ON EXTENSOR BURST DURATION AT ~58% OF THE EXTENSOR PHASE.** The later shoulder retraction is applied during R CIB, the greater this burst is prolonged (Fig. 6A). Only with retractions given at its very end does R CIB duration return to control.

Overall, shoulder retractions applied in the early part of R TriLo shortens it more than later retractions (Fig. 6B). However retractions before L TriLo offset may shorten R TriLo burst less than later retractions because of prolongation of overlapping R TriLo/L TriLo activity (strategy 1, Figs. 3D and 5A). Alternatively, retractions during the same period may shorten R TriLo burst considerably (strategy 2), but this is missing from Fig. 6B because marked R TriLo burst attenuation made its duration uncertain (see, however, Fig. 4A). Past L TriLo offset, there may be a brief plateau followed by a linear
relationship where R TriLo burst is gradually less shortened until it reaches another plateau at 58 ± 4% of the burst (n = 7 cats), during which R TriLo is only slightly shortened.

In general at the linear relationship onset, the degree of R TriLo burst shortening below control approximately equals the duration of the interval with the linear relationship. This suggests that shoulder retractions given at the linear relationship onset (or any immediately preceding plateau) may be deleting that interval. Thus R TriLo burst duration graphs may point to the same "within extensor phase" interval in two ways: a linear change with retraction time during that interval and deletion of that interval by earlier retractions. This was observed in five cats.

In summary, the later shoulder retractions are given during R ClB, the more they prolong it. Once R ClB has ended, such prolongation is not possible and a critical period begins, which will end with L ClB onset. During this critical period, two alternative strategies exist to abolish or not L ClB that normally follows this period. At ~58% of the R TriLo burst, cycle duration crosses the control value, and R TriLo burst becomes only slightly shortened at the end of a linear relationship of its duration with retraction time. Retractions prior to the interval characterized by this linear relationship may be deleting this interval.

**Phasic shoulder protractions**

Series of protractions were done in seven cats (in 1 cat, data were sufficient for the flexor phase, whereas the extensor phase analysis was limited to the contralateral flexor). Although shoulder protractions generally produced effects opposite to shoulder retractions (Fig. 1), abrupt transitions within the flexor and extensor phases strongly suggested subphases.

PHASIC SHOULDER PROTRACIONS DURING THE EXTENSOR PHASE CONFIRM A CRITICAL POINT AT 58% OF THE EXTENSOR PHASE, AND REVEAL AN EARLIER CRITICAL POINT WITHIN THAT PHASE. The point at 58% of the extensor phase is revealed to be critical by examining the effect of shoulder protractions during this phase which would correspond to a period of forelimb retraction in the nonparalyzed animal.

In Fig. 7, A and B, R TriLo burst duration as well as the interval from R TriLo onset to L TriLo onset are plotted as a function of protraction time measured from R TriLo onset.
Past an abrupt transition at ~58% (54–62%) of the R TriLo burst, shoulder protractions no longer delay L TriLo onset and prolong much less, if at all, the R TriLo burst. The similarity of effects in A and B reflects coordination between the two sides. Note that this cat is the same as in Fig. 6B where a critical point also occurred at 58% of the R TriLo burst, past which the shortening effect of shoulder retractions became minimal.

The abrupt transition at ~58% (54–62%) of the R TriLo burst can also be identified plotting the perturbed R TriLo cycle duration (Fig. 7C). Shoulder protractions during the R TriLo burst given before and after this critical point prolong and shorten the cycle respectively to approximately constant levels. Protractions given after the R TriLo burst shorten the cycle in a way better appreciated in Fig. 9A from the same cat.

Figure 7C is representative of the three cats with the best fictive locomotion as judged by a stable frequency typical of walking, and symmetry documented by the bilateral recording of three to six ENGs. The critical point for the effects of shoulder protractions on R TriLo cycle duration occurred at a mean of 59% of the R TriLo burst in these three cats (58, 57, and 63%).

FIG. 4. Effects of R shoulder retractions on cycle duration. A: effects on R TriLo cycle duration, with retraction time measured from R TriLo burst onset. B: effects on L TriLo cycle duration with retraction time measured from L TriLo burst onset. These retractions were done starting from a protracted position. In B, the shaded region indicates the critical period during which the bifurcation occurs. Boxes in this and the following figures represent the mean control bursts with the small horizontal bars representing 1 SD. The box disposition under the figure corresponds to time on the abscissa and helps to visualize where in the cycle a perturbation is applied. For example, a shoulder retraction at time 0 on the abscissa in A is applied at R TriLo onset and at time 0 in B is applied at L TriLo onset. 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. Dotted horizontal lines indicate the control cycle duration. Linear regression lines computed for the intervals shown help appreciate the linear portions of the data.

FIG. 5. The effect of phasic R shoulder retractions given during the critical period for retractions, according to whether the contralateral (L) forelimb tonic position is a retracted (A and B) or protracted one (C and D). A and C are given just prior to onset of R TriLo; B and D are given early in R TriLo before the end of L TriLo. All data are from the same cat as Fig. 1. Only extensor bursts are recorded, and the silences between the extensor bursts correspond to the flexor phases. When the contralateral forelimb position was a retracted one, strategy 1 was evoked during the critical period with switch from L extensor to L flexor activity (A and B). When the contralateral forelimb position was a protracted one, strategy 2 was evoked during the critical period with L extensor activity persisting for an additional cycle (C and D).
An earlier critical point at 35–42% of the R TriLo burst also influences the responses during the extensor phase as determined by two additional results.

First, besides the critical point identified at 58% of R TriLo burst, the cats represented by Fig. 7C showed evidence for an earlier critical point when plotting the L (contralateral) CIB integrated amplitude against the time of shoulder protraction. In Fig. 8A, L CIB burst amplitude increased most for protractions prior to a critical point at 37% of R TriLo burst, increased less for protractions between 37 and 61% of the R TriLo burst (the latter already identified as our ~58% critical point), and did not change for later protractions. Furthermore, the increased L CIB amplitude by protractions given prior to this earlier critical point consisted of the L CIB burst initially remaining of similar amplitude to control, and then abruptly increasing in amplitude at 37 ± 5% (n = 98) of the control R TriLo burst.
Fig. 8B. Thus this same earlier critical point is manifested both on the “input” side because it is protractions prior to this point that maximally increase L CIB amplitude (Fig. 8A, abscissa), and on the “output” side because it is the portion of L CIB burst after this point that displays the increase in amplitude in response to these same protractions (Fig. 8B, ordinate). The other cat of Fig. 7C with L (contralateral) CIB recorded showed very similar manifestations at a critical point situated at 42% of the control R TriLo burst. In total, these two manifestations of an early critical point for the effects of protractions on contralateral CIB were seen in three of four cats where it was recorded; in the last cat, contralateral CIB was mostly affected in duration with little amplitude change.

Second, evidence for this earlier critical point comes from some cats that did show in their plots of R extensor cycle duration a transition earlier than at 58% of R TriLo burst. In contrast to Fig. 7C, the transition from prolonged to shortened cycle duration started earlier in two cats at 35% of the R TriLo burst, whereas it ended at a comparable time at 53% of the R TriLo burst (the latter already identified as our ~58% critical point). Finally one cat with only R TriLa recorded showed a bifurcation beginning at the earlier critical point (32% of the R TriLa burst) and finishing at the later critical point (53% of the R TriLa burst). This bifurcation began as cycle duration became longer or shorter than during the initial plateau and ended as the perturbed cycle durations of the two branches differed by a value equal to control cycle duration. The shoulder protrac- tion amplitude delivered by the motor varied in this cat (10–40°) and was on the average higher for the upper than the lower branch (21 vs. 12°). In one other cat with a similar bifurcation beginning at 35% of the R TriLo burst, contralateral recording showed the upper branch to correspond to abolition of the R TriLo silence normally accompanying the L TriLo burst.

Together these results indicate that besides the critical point at 58% of R TriLo burst, an earlier critical point at ~35–42% of the R TriLo burst also importantly influences the response to phasic shoulder protractions during the extensor phase.

PHASIC SHOULDER PROTRACIONS DURING THE FLEXOR PHASE IDENTIFY THE SAME TWO CRITICAL POINTS WITHIN THE CONTRALATERAL EXTENSOR PHASE. Two critical points, symmetrical to those identified with shoulder protractions during the extensor phase, also influence the responses to shoulder protractions during the flexor phase. The earlier critical point determines when an abrupt change in the response pattern occurs with a maximal effect, whereas the second critical point determines a boundary for the magnitude of that effect.

Shoulder protraction during the flexor phase (when the nonparalyzed animal mainly protracts the forelimb), shortened this phase as assessed by the R ClB burst, R TriLo/TriLa silence, and contralateral L TriLo/TriLa bursts durations. But this shortening effect did not occur homogeneously as only
FIG. 8. Evidence for an earlier critical point during the extensor phase revealed by 2 analyses of the effects of R shoulder protraction on contralateral (L) CIB amplitude. A: integrated L CIB amplitude against the time of R shoulder protraction, measured from R TriLo onset (time 0 on the abscissa). Past the 1st vertical line at 37% R TriLo, protraction becomes less effective at increasing L CIB amplitude and past the 2nd vertical line, no longer increases it (control amplitude indicated by the horizontal dotted line). B: the time at which individual L CIB bursts abruptly increase in amplitude above control in response to R shoulder protraction. Time zero on both axes is R TriLo onset. Almost up to the first vertical line in A, 71% of the L CIB bursts showed an initially normal amplitude that then rose abruptly above control. The rather constant time of this rise in amplitude, fitted by a nearly horizontal least squares line, was at 37 ± 5% of the control R TriLo burst, therefore identifying the same critical point as in A. Data from 1 of the cats represented by Fig. 7C.
protractions during the late flexor phase had the characteristic effect of almost immediately terminating R CIB and starting R TriLo. Thus R CIB was actually more shortened by a later than by an earlier protraction. Another difference between protractions early and late in flexion was a comparable shortening of R TriLo silence and R TriLa silence by early protractions, whereas only R TriLo silence was shortened by later protractions.

Figure 9 details these effects in two cats by plotting the L TriLo onset to R TriLo onset interval according to protraction time. This interval allowed comparison between the greatest number of cats. Both graphs show a bifurcation as some data points “ascend” along an upper branch, whereas others “fall” to a lower level. As the vertical line indicates, we focus on the time when the upper branch ceases, and the lower group of data points, which corresponds to a maximally advanced R TriLo
onset, continues into a linear relationship. This critical point for shoulder protractions during the flexor phase was seen in 5/7 cats. One cat with a fast rhythm showed a linear relationship throughout, and another cat had only R TriLo recorded.

Selecting L TriLo burst as the reference, the time of this critical point was comparable across 4/5 cats. For the two cats of Fig. 9 with a symmetrical rhythm and R ClB recorded, it occurred at 41 and 40% of L TriLo or 42 and 36% of R ClB. In two other cats with asymmetrical rhythms (R TriLo ended before L TriLo onset), it occurred at 37 and 32% of L TriLo. In a fifth cat with a symmetrical rhythm, it occurred later at 57%. Thus overall, this critical point occurred at 41.4 ± 9.4% of the control L TriLo burst (n = 5). This point is symmetrical to the early critical point identified above at 35–42% of the R TriLo burst by shoulder protractions during the extensor phase.

At this point the response pattern consisting of rapid termination of R ClB burst and R TriLo silence by shoulder protractions during the flexor phase becomes established. Protractions at this critical point maximally advanced R TriLo onset to a point situated, respectively, at 58, 61, 60, 60, and 70% of the control R TriLo silence in these five cats. What does this “boundary” represent in the ipsilateral flexor and contralateral extensor bursts? Two of these cats (those of Fig. 9) had R ClB recorded and 60 and 61% of the R TriLo silence corresponded to the time of R ClB onset of descent in their control cycles. In the fifth cat, 70% of the L TriLo silence was also when L ClB began its descent, and by symmetry we can assume the same held on the R side. Therefore in these three individual cats protractions applied at the critical point identified in this section maximally advanced R TriLo onset to a point coincided with the R ClB onset of descent in their control cycle. In these same three cats with symmetrical rhythms, this R TriLo onset advance was to a mean of 58% (52, 57, and 64%) of the control L TriLo burst. This point is symmetrical to the critical point identified previously in the same three cats at a mean of 59% of the R (ipsilateral) TriLo burst (58, 57, and 63%, respectively) where the effect of shoulder protractions during the extensor phase abruptly changed (Fig. 7). It also corresponds to the symmetrical critical point identified at 58% of the R TriLo burst with shoulder retractions.

In summary, past a critical point at 58% of the R TriLo burst, phasic shoulder protractions no longer delay L TriLo onset, prolong much less the R TriLo burst, and considerably shorten the R TriLo cycle (Fig. 7). In addition, past an earlier critical point at 35–42% of the R TriLo burst, protractions already increase less L ClB integrated amplitude (Fig. 8A).

Furthermore the symmetrical critical point at 58% of the L (contralateral) TriLo burst is the point to which R TriLo burst onset is maximally advanced by shoulder protractions during the R flexor phase, while the symmetrical critical point at 41% of the L (contralateral) TriLo burst is the time at which protractions have this effect (Fig. 9).

EFFECT OF THE RETURNING PHASE OF PHASIC SHOULDER PERTURBATIONS. To determine whether the responses to phasic shoulder perturbations are to limb position or to limb movement, we have studied in two cats the effects of the returning phase of these perturbations where the limb is “retracted”/“protracted” back to the pendent position.

The results are generally very similar to the perturbations done from the pendent position, strongly suggesting that limb movement rather than position primarily determines the responses.

Returning a protracted limb during its shortened ClB pro- longs it beyond pendent ClB, even though the limb is not retracted beyond the pendent position.

Returning a protracted limb during its prolonged TriLo shortens it to even less than pendent TriLo when done before a time coinciding with the protracted cycle TriLa inflection in the cat with TriLa recorded.

Returning a retracted limb during its prolonged flexion generally shortens it to even less than pendent flexion. The L TriLo onset to R TriLo onset interval shows at a time corresponding to 55% of the L TriLo burst of the pendent cycle a qualitatively similar transition to that found at 41% of the L TriLo burst for shoulder retractions from the pendent position.

Returning a retracted limb during its shortened extension restores its duration to pendent, when applied before a time corresponding to 42% of the R TriLo burst of the pendent cycle. The R TriLo onset to L TriLo onset interval shows at this point a qualitatively similar transition to that found at 58% of the R TriLo burst for shoulder protractions from the pendent position.

DISCUSSION

Phasic shoulder retractions during ipsilateral fictive extensor activity shorten bilaterally the bursts active in that phase and during ipsilateral flexor activity prolong them. Shoulder protractions have the opposite effect. Because the unparalyzed limb generally retracts during stance and protracts during swing, burst durations appear altered according to a negative feedback scheme. The central pattern generator (CPG) seems to correctly evaluate the consequences of the perturbation and produce bilateral adjustments to match the central and peripheral status. The similar effects of retractions on R TriLo and L TriLo cycle durations, as explained in RESULTS (Fig. 4), reflect the preserved bilateral coordination.

The details are more complex. For example, the greater R ClB prolongation by late than by early shoulder retractions, whereas R TriLo is more prolonged by early than by late shoulder protractions indicates a negative feedback operating differently in the two cases. Furthermore, although cycle structure is usually described as bursts onsets and offsets, our results indicate that critical points exist within bursts where the responses to phasic perturbations may abruptly change from one pattern to another. Furthermore these critical points correspond to those identified with tonic perturbations in the companion paper.

Definition of a critical point for the effects of phasic perturbations

When a bifurcation occurred in plots of cycle duration or portions of it, its beginning and end defined critical points. At its onset, two branches diverge from a common region while at its end, subtracting a value equal to control cycle duration from the upper branch reunites it with the lower branch. Besides bifurcations, abrupt transitions in how the perturbed interval
duration varied with the time of the perturbation also defined critical points because they could be identified both by shoulder protraction and retraction and they could be the sites of bifurcation in another cat or in another series of the same cat.

A critical point could be identified in one phase, as well as the symmetrical phase. For example, ~37–42% of R TriLo burst was the point prior to which shoulder protractions were most effective at enhancing L (contralateral) CIB (Fig. 8A), whereas at ~41% of L TriLo burst protractions maximally advanced R TriLo onset (Fig. 9).

A critical point identification was also solidified when seen on one plot’s abscissa and ordinate of another. Thus at ~58% of R TriLo burst, the shoulder protraction effect during the extensor phase abruptly changed (abscissa Fig. 7), but also protractions during the flexor phase maximally advanced R TriLo onset to ~58% of the L (contralateral) TriLo burst (leftward arrows toward ordinate Fig. 9).

Identified critical points

The location of critical points revealed by phasic R shoulder perturbations may be compared with Fig. 10 showing critical points B–D identified in the companion paper using burst morphology and tonic perturbations. The critical bifurcation for shoulder retractions extends from R CIB offset/R TriLo onset to L CIB onset. R TriLo onset corresponds to point D. A second critical point at ~58% of the R TriLo burst past which phasic shoulder protractions/retractions only slightly prolong/shorten R TriLo burst (Figs. 7A and 6B) corresponds to point C identified in the companion paper at ~55% of R TriLo and coincident with L CIB onset of descent. The greater effect of phasic protractions and retractions given prior to this point in the extensor phase is reminiscent of R TriLo burst lengthening/shortening by tonic protraction/retraction being limited to its component preceding point C (Figs. 4–5 of companion paper).

The symmetrical point C at ~58% of the L TriLo burst, which coincides with the R CIB onset of descent, is the point to which R TriLo onset (point D) is maximally advanced by phasic shoulder protractions (Fig. 9). This is similar to tonic shoulder protraction shifting point D to C (Fig. 6A of companion paper).

A third critical point is at ~41% of L TriLo burst, when shoulder protractions maximally advance R TriLo onset, corresponds to point B. This is confirmed by an ANOVA showing no significant difference for the times of this point for tonic shoulder protractions, tonic elbow extensions (Fig. 6, A and B, of companion paper), and phasic shoulder protractions during the flexor phase (Fig. 9). These times are 41 ± 6.7% (n = 5), 44.3 ± 2.6% (n = 4), and 41.4 ± 9.4% (n = 5) of the control L TriLo burst, respectively (F = 0.267, critical F value at α = 0.05 is 3.98 for df 2,11). Thus maximal R TriLo onset advance might correspond to immediately initiating R CIB descent at this critical point similarly to tonic shoulder protractions.

Shoulder protractions prior to the symmetrical point B at ~37–42% of R TriLo burst enhance most L CIB (Fig. 8A) but with the burst amplitude increase delayed until this point (Fig. 8B). This delayed L CIB amplitude increase by phasic shoulder protractions until point B is reminiscent of the R CIB amplitude increase by tonic extensions beginning at point B (Figs. 4, 6B, and 7 of companion paper).

In summary, the critical points identified with phasic shoulder perturbations closely correspond to points B–D reported in the companion paper using tonic shoulder and elbow perturbations (Saltiel and Rossignol 2004).

Andersson and Grillner (1981) also found a critical period for hindlimb retractions. The bifurcation again began when retractions no longer prolonged the flexor activity, but this occurred within the ipsilateral flexor (tibialis anterior, TA) phase rather than at its end as in our case. In addition, the perturbed TA cycle duration crossed the control value at ~50–57% of the extensor (medial gastrocnemius, MG) burst, a similar result to ours. Critical points are otherwise not evident in that study. Early protractions within MG increased its amplitude, but not duration, and produced no obvious transition within the MG phase in the (incompletely sampled) cycle duration plot. We did not observe flexor enhancement by protraction, or flexor burst inhibition by late retractions, which in our case prolonged flexion most until close to its end (Fig. 6A). Differences in our study include intact forelimb innervation (except for the recorded nerves), and investigation of proximal forelimb ENGs.

Possible role of critical points in interaction of the CPG with feedback (sensorimotor integration) and motor physiology

Hasan (1992) concluded that proprioceptors assist spatial steering and temporal coordination in multi-joint movements. How this is accomplished remains unclear. In our two papers, proprioceptive perturbations yielded evidence for critical points. What role do these have in the CPG interaction with afferent feedback? Several findings are relevant to this question.

A first aspect of critical points is “sensory” whereby the output modification elicited by a perturbation depends on its timing with respect to the critical points in the fictive program. A second aspect is bilateral coupling of critical points, i.e., R TriLa inflection and L CIB onset of descent remain simultaneous at point C during tonic perturbations, indicating a role for interlimb coordination. Here two observations further substantiate this. Phasic shoulder protractions prolonged R TriLo burst and R TriLo onset to L TriLo onset interval mostly when applied before point C (Fig. 7, A and B). This suggests that they prolong only the R TriLo portion before ~58% of the burst and the L CIB portion before its descent onset, i.e., the two burst portions before point C. This would again imply a preserved coupling at point C. Thus when protraction prolongs R TriLo, the first L CIB component prolongation might reflect L CIB “waiting” for a coupling signal from the R side to begin its descent. With respect to identifying interlimb coupling signals, Stein (1976) suggested a method. A stable point for absolute coordination between oscillators is the phase in the modulated oscillator cycle where a modulator oscillator pulse causes a phase shift equal to the difference in their natural frequencies (zero for identical natural frequencies). Critical point C with unchanged R TriLo cycle duration by phasic shoulder retraction is such a point. Thus an interlimb coupling signal at point C, somehow mimicking a R phasic shoulder retraction, could support absolute coordination. In a recent study, >50% of rhythmically active commissural interneurons located in L₄–L₅ segments and descending to contralateral L₃ segments
preferentially fired at the mid-phase of the ipsilateral $L_2$ root burst (Butt et al. 2002) and were postulated to subserve bilateral coordination.

A third aspect is consecutiveness of critical points in the locomotor cycle (B–D). In the companion paper, tonic shoulder protraction shifted C to B, and D to C, whereas tonic elbow extension and tonic shoulder retraction shifted C to D. Here phasic shoulder protractions at point B advance point D to point C. These discrete shifts mean that critical points do not only determine whether a burst duration will be changed by a perturbation but also by how much. For example, R ClB burst is prolonged through a C (R ClB onset of descent) to D (end of R ClB/R TriLo onset) shift, whereas it is shortened through a D-to-C shift. We suggested in the companion paper that the locomotor cycle possesses a temporal structure or grid with discrete times along which the critical points are disposed and

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FIG. 10. Structure of the fictive locomotor cycle and location of critical points identified in the companion paper on the basis of burst morphology together with the effects of tonic proprioceptive perturbations. This average is of 19 control cycles (both forelimbs pendent) from cat 112, synchronized on the R TriLa inflection point. Critical points B–D in each half-cycle are indicated by vertical lines. The location of points B and C as percentages of the TriLo bursts is indicated. Point D corresponds to TriLo onset/end ClB.
may be shifted. By aligning critical points together on both sides of the cycle, this grid could also subserve different modes of bilateral coupling, which might perhaps underlie asymmetrical rhythms. Such altered coupling might automatically redistribute the sensitive zones to proprioceptive inputs such that various adaptive reflexes may remain optimal despite different locomotor kinematics.

In particular, during tonic changes in limb position, we would expect a redistribution of the sensitive zones through a discrete redistribution of the critical points. The observed shifts in the timing of the transitions for the effects of the returning phase of shoulder protraction or retractions, compared with when the limb is retracted or protracted from the pendent position, argue for such a discrete redistribution.

A fourth aspect is that critical points determine, besides the response type evoked by a phasic perturbation, its onset time. Perturbation effects may be immediate (a shoulder retraction near the very end of R CIB may still prolong it markedly) or delayed. Shoulder protraction prior to point B maximally increased L CIB amplitude (Fig. 8A), but this effect was delayed to the burst portion after point B (Fig. 8B). Thus the role of critical points is not uniquely “sensory,” dictating how the CPG will adapt according to perturbation time, but also “motor,” dictating when to instantiate this modification.

The critical points probably delimit different motor program components rather than just periods of different sensorimotor processing. Clues already exist in the fictive intra-burst structure (e.g., TriLa inflection at point C, Fig. 10). This subcomponents idea relates to the concept of a spinal cord modular organization (Bizz et al. 1991; Saltiel et al. 1998).

A fifth aspect is critical points subserving both interlimb and intralimb coordination. Point B determines L (contralateral) CIB amplitude adjustment to phasic shoulder protraction (in- terlimb coordination), whereas R CIB amplitude adjustment at point B to tonic elbow extension reflected intralimb coordination of the elbow adjustment with the shoulder motion (see companion paper DISCUSSION).

These different aspects all relate to phase. Critical points determine a phase where perturbation elicit a particular response pattern, where this response will be instantiated, a phase of preferred bilateral coupling, and discrete phase shifts. Thus critical points may underlie a sophisticated phase-dependent motor coordination.

Critical points might also coordinate multi-joint voluntary movements. In a recent study, human subjects reached for a handle at three different heights or orientations with movements constrained in time (Cordo et al. 1993). In superimposed averages, the electromyographic modifications to the target variations occurred only at discrete periodic points within the movement either as amplitude adjustments or shifts in burst onsets time. This is reminiscent of the discrete time of CIB amplitude adjustments and of the discrete shift in R TriLo onset reported in this and the companion paper.

**Possible biomechanical correlates of critical points**

Where should critical points be located in the fictive motor program for interaction with afferent feedback? Their timing appears to correspond to biomechanically significant events in real locomotion.

The critical period for shoulder retractions extends from R CIB offset to L CIB onset, halfway between R TriLo onset and L TriLo offset (Fig. 10), i.e., from just before R forelimb landing till about midway during double forelimb support. Shoulder retraction makes this landing unfavorable. One strategy consists of still accepting weight transfer on the R forelimb and initiating L swing. In the other strategy, the L forelimb continues to support weight, while the R forelimb aborts landing and takes off for a short swing prior to a second landing. L forelimb tonic protraction is a good position to facilitate continued weight support (Fig. 5). The critical period ends when this is no longer feasible because significant weight transfer has already happened between the forelimbs. This influence of L forelimb position on strategy choice resembles the result where tonic L hindlimb protraction/retraction favored its crossed extension/flexion respectively to R hindlimb cutaneous stimulation in the clonidine-treated nonwalking acute spinal cat (Rossignol and Gauthier 1980). The interpretation was similar in terms of adequacy for weight support during locomotion.

With respect to critical point C, the companion paper indicated from Manter’s work that vertical passage of the ipsilateral forearm, horizontal ground reaction force reversal to forwards, and contralateral elbow extension onset during swing occur at this time. Past point C, protraction long much less the R TriLo burst. Biomechanically, the rationale could be that once the R forearm has passed the vertical, a) shoulder protractions might interfere less with R stance progression, or b) enough forward progression has already occurred to allow weight transfer to the L forelimb, thus “bypassing” the protraction. Before this, the delayed L extensor onset in parallel to the R extensor burst prolongation (Fig. 7, A and B) would delay landing on the L forelimb until R stance has sufficiently progressed for weight transfer to become appropriate.

With respect to critical point B, we indicated in the companion paper that it appears to correspond to the time when the swinging upper arm movement changes from retraction to protraction (Drew and Rossignol 1987; English 1978; Manter 1938). Protractions would thus be opposite to the upper arm movement direction prior to this critical point but in the same direction afterward. Indeed R TriLo onset may be less advanced by shoulder protraction applied earlier than point B rather than later (Fig. 9).

That protraction past point B cannot advance R TriLo onset to a limit earlier than ~58% of the L extensor phase suggests an interlimb-coordinated response to protractions during the flexor phase based on the same principle than for protractions during the extensor phase. This would be that past 58% of the ongoing stance, weight transfer can happen without a problem. Thus shoulder protraction applied later than 58% of the R stance no longer delay L stance onset, and shoulder protraction applied in R swing do not advance R stance onset earlier than 58% of the control L stance.

The increased L CIB amplitude to shoulder protractions indicates interlimb coordination at point B. This could serve to bring the contralateral limb in a similar anteroposterior range as the protracted limb as seen when isometrically protracting one forelimb in a nonparalyzed decerebrate cat during treadmill locomotion and observing the free contralateral forelimb (unpublished observations). Another role could be to provide energy to the body and R forelimb whose stance is subjected to...
the protraction. Why would the L CIB amplitude increase only after point B? Increasing L CIB amplitude (protractor as well as flexor) before point B would be counterproductive to the normal retraction-protraction sequence in swing. Another reason may relate to energy transfers. Computing those for Manter’s data in cat locomotion (Manter 1938) in a similar way to Elftman (1939) for human gait, shows that at ~45% of forelimb stance, i.e., approximately at point B, the energy flow reverses to become directed to the body and forelimb in stance from the swinging forelimb.

In summary, the critical points identified in the fictive cycle in the companion paper delimit different effects of phasic proprioceptive perturbations. We suggest that these points in the structure of the fictive cycle are features of the pattern generator corresponding to the incorporation of specific biomechanical events in real locomotion. We further suggest that these points are important for intralimb and interlimb coordination, both to adapt to perturbations and to construct rhythms of different cycle structure.

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