Control of Locomotor Cycle Durations

S. Yakovenko,1 D. A. McCrea,2 K. Stecina,2 and A. Prochazka3

1Department of Physiology, Université de Montréal, Montreal, Quebec; 2Department of Physiology, University of Manitoba, Winnipeg, Manitoba; and 3Centre for Neuroscience, University of Alberta, Edmonton Alberta, Canada

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Yakovenko, S., D. A. McCrea, K. Stecina, and A. Prochazka. Control of locomotor cycle durations. J Neurophysiol 94: 1057–1065, 2005. First published April 7, 2005; doi:10.1152/jn.00991.2004. In intact animals and humans, increases in locomotor speed are usually associated with decreases in step cycle duration. Most data indicate that the locomotor central pattern generator (CPG) shortens cycle duration mainly by shortening the durations of extensor rather than flexor phases of the step cycle. Here we report that in fictive locomotion elicited by electrical stimulation of the midbrain locomotor region (MLR) in the cat, spontaneous variations in cycle duration were due more to changes in flexor rather than extensor phase durations in 22 of 31 experiments. The locomotor CPG is therefore not inherently extensor- or flexor-biased. We coined the term “dominant” to designate the phase (flexion or extension) showing the larger variation. In a simple half-center oscillator model, experimental phase duration plots were fitted well by adjusting two parameters that corresponded to background drive (“bias”) and sensitivity (“gain”) of the oscillator’s timing elements. By analogy we argue that variations in background drive to the neural timing elements of the CPG could produce larger variations in phase duration in the half-center receiving the lower background drive, i.e., background drive may determine which half-center is dominant. The fact that data from normal cats were also fitted well by the model indicates that sensory input and central drive combine to determine locomotor phase durations. We conclude that there is a considerable flexibility in the control of phase durations in MLR-induced fictive locomotion. We posit that this may be explained by changes in background excitation of neural timing elements in the locomotor CPG.

INTRODUCTION

Many movements are rhythmic in nature, for example breathing, chewing, swimming, walking, and flying. In these examples, numerous muscles are cyclically activated in a coordinated sequence by neural commands. In mammals, rhythmic locomotor movements can be generated by the spinal cord even in the absence of supraspinal control (Brown 1911). To explain the mechanism of rhythogenesis, Brown posited a spinal intrinsic factor comprising mutually inhibitory groups of neurons he named half-centers. The intrinsic factor was later renamed the central pattern generator (CPG) (Grillner and Zanger 1975) and developed into a schematic of the mammalian CPG in which interneurons involved in reorganized flexion reflexes functioned as half-centers coupled by mutual inhibition (Lundberg 1981). Although the precise location and structure of the locomotor network controlling the hind legs in vertebrates is poorly understood, a variety of studies indicate that it is widely distributed throughout the spinal cord (Cowley and Schmidt 1997; Kiehn and Kjaerulf 1996) and can be activated pharmacologically using a variety of neurotransmitters (Rossignol et al. 1998) or through reticulospinal pathways that in turn can be activated by stimulation of other brain stem areas such as the midbrain locomotor region (MLR) (see (Jordan 1998; Noga et al. 2003).

Attempts have been made to deduce the functional organization of the CPG on the basis of the mutability of the patterns of muscle activation during different locomotor behaviors, e.g., comparing forward, backward, uphill, and downhill walking (Grillner 1981; Stein and Smith 1997). The results of these investigations have been used to suggest that the CPG does not consist of a simple flexion-extension oscillator controlling the whole limb. Instead, a mechanism involving several oscillators was suggested (Grillner and Wallen 1985) in which each joint is controlled by its own “unit” oscillator that is flexibly coupled to the other unit oscillators of the limb. However, there is no compelling evidence of localized regions of activation corresponding to separate oscillators controlling hip, knee, and ankle (Prochazka et al. 2002b). Furthermore, in fictive locomotion, preliminary evidence suggests there is a tight coupling between bursts of activity of extensor nerves of the entire limb and a similar coupling of flexor bursts, hip flexors being slightly phase-advanced on ankle flexors (Quevedo et al. 2000b). Thus as a first approximation we consider the spinal CPG to be a bipartite organization that produces alternating and out-of-phase activation of flexor and extensor muscles throughout the hindlimb. Additional circuitries also exist that shape the activity of individual motoneuron pools, including those innervating bifunctional muscles, during various locomotor tasks (Burke 2001).

It is well known that locomotor cycle periods, phase transitions, and phase durations can be affected by descending inputs (Armstrong 1988; Drew 1991) as well as sensory input to the spinal cord (McCrea 2001; Pearson and Rossignol 1991; Rossignol 1996). Afferent feedback from muscle proprioceptors and low threshold cutaneous receptors is used continuously to shape and regulate motoneuron activity during stepping (Bouyer and Rossignol 1998; Prochazka et al. 2002a). Results from fictive locomotor preparations show that depending on which afferents are stimulated, segmental proprioceptive feedback can be directed either to flexor or extensor components of the CPG (McCrea 2001). These observations suggest a flexible organization of the locomotor CPG that can be accessed and manipulated by sensory feedback (as well as descending systems) to control the activity and timing of both flexor and extensor phase motoneuron activity. Numerous conceptual and mathematical models of locomotor pattern generators and their sensory control have been presented, e.g., Marder and Bucher 2001; Pearson 2004; Yakovenko et al. 2004.
The timing of transitions between locomotor phases is crucial for matching the CPG output to actual limb forces and motions. During normal overground gait in intact animals and humans, an increase in the speed of locomotion is usually accompanied by a decrease in the step cycle duration, mostly due to the shortening of the stance phase. The duration of the swing phase changes much less (Arshavsky et al. 1965; Goslow et al. 1973; Halbertsma 1983; Murray 1967). The same has been reported in spinalized cats during treadmill locomotion (Forssberg and Grillner 1973; Prochazka et al. 2002b) and in fictive locomotion (Dubuc et al. 1988; Leblond and Gossard 1997; Pearson and Rossignol 1991). These results suggest that the CPG is hard-wired to produce adjustments of step cycle duration by mainly modulating the durations of extensor bursts rather than flexor bursts (Grillner and Dubuc 1988). This is in contrast to the flexibility of control of both the flexion and extension phases by afferent feedback.

In the present study, we sought to clarify whether the locomotor CPG is extensor biased with a preferential control of extensor phase duration. To this end, we used the fictive locomotion preparation in adult decerebrate cats. The advantage of this reduced preparation is that without rhythmic afferent input and without cortical control, the locomotor pattern should reflect more of the operation of the basic spinal CPG than it would in more intact preparations. In this study, we report that in MLR-induced fictive locomotion in the cat, the locomotor cadence varied more as a result of changes in flexor rather than extensor phase durations in many preparations. In a simple half-center model of the locomotor CPG, coupled changes in background excitation and sensitivity of the timing elements reproduced the observed range of phase-duration properties. The results are consistent with the idea that the balance of drive to the flexor and extensor half-centers of the locomotor oscillator results either in flexor- or extensor-dominated control.

 Portions of this work have appeared in Abstract form (Yakovenko et al. 2001).

METHODS

Surgical procedures

All data reported in this paper originated from experiments done for other reasons in David McCrea’s laboratory, University of Manitoba (Gosgnach et al. 2000; Quevedo et al. 2000a). All surgical and experimental protocols were in accordance with the guidelines set out by the Canadian Council for Animal Care. The experiments were performed on 23 cats (weight: 2.2–3.2 kg). The animals were anesthetized with halothane delivered in a mixture of 30% oxygen and 70% nitrous oxide for the duration of the surgery. Surgical procedures consisted of a tracheotomy, cannulation of arteries and veins, dissections of various hindlimb nerves and a craniotomy. The position of both the ipsilateral and contralateral limbs was essentially the same in all preparations used in the present study. Furthermore, both limbs were extensively denervated by bilateral sectioning of the sciatic, femoral, and obturator nerves, and tendons surrounding the hips were cut. Mechanical, precollicular-postmammillary decerebration was performed by removing all the tissue rostral to the transection. This cut. Mechanical, precollicular-postmammillary decerebration was per-

femoral, and obturator nerves, and tendons surrounding the hips were extensively denervated by bilateral sectioning of the sciatic, and biceps femoris anterior (SMAB), flexor hallucis longus (FHL), flexor digitorum longus (FDL), or sometimes FDL and FHL together (FDHL), gastrocnemius and soleus (GSol), plantaris (Plan), and quadriceps (Quad).

Up to 12 nerves were recorded simultaneously in each cat. A laminectomy was performed usually from the L4 to the S1 regions in some animals, but in others, it was more extensive, including thoracic and cervical segments. A lethal injection of pentobarbital anesthetic was administered at the end of the experiment.

Data recording and analysis

Automated analysis was applied to 31 episodes of MLR-evoked fictive locomotion from 23 different animals in which spontaneous changes in the cycle timing occurred. Episodes in the same experiment were treated independently because they were recorded hours apart, often with modified parameters of MLR stimulation. We analyzed as many segments of ENG recording as were present in the data from a given episode. This amounted to an average of 117 individual cycles per episode. All individual cycles from a given run were then analyzed to produce phase-duration scatter plots of the type shown in Fig. 2.

Electroneurogram (ENG) activity in the hindlimb muscle nerves listed in the preceding text was filtered (30 Hz to 3 kHz), rectified, low-pass filtered (100 Hz), and digitized at 500 Hz. The waveforms were then analyzed to determine onsets and terminations of bursts using an adaptive threshold crossing method described in the following text. Recordings with a signal-to-noise ratio <5 were rejected. Cycle period was determined as the time between two onsets of the ENG envelope. We intentionally restricted the analysis to cycles with periods between 0.3 and 1.5 s corresponding to speeds of overground locomotion between 0.2 and 3.1 m/s (Halbertsma 1983). Regression lines were fitted to scatter plots of burst duration and cycle period for individual nerves. One-way ANOVA and multiple comparison of regression line slopes with Tukey’s honestly significant difference criterion was performed to determine significant differences (α = 0.05).

ADAPTIVE THRESHOLD CROSSING METHOD. A novel means of determining the onsets and terminations of ENG bursts was developed for this study. To understand the method, consider a portion of a noisy signal containing bursts of activity (Fig. 1A). Now consider a hori-

zontal line representing a test level, moving upward through the signal (dashed lines Fig. 1A). Within the noise band, there are many intersections of the signal and the test level. The number of intersections reaches a peak somewhere within the noise band, as plotted in Fig. 1B. As the test level moves above the noise band and into the region of burst activity (dashed line b in Fig. 1, A and B), the number of intersections drops and then remains constant. As the test level moves up into the region of peaks (line c) the number of intersections again rises and then declines to zero (line d).

We developed a software algorithm to detect onsets and terminations of the ENG signal based on an examination of threshold crossings in the rectified-integrated ENG signal (Fig. 1B). For a given segment of recording, the algorithm first constructs the intersections plot for test levels ≤20% of maximal signal amplitude (the 1st 8th of the plot in Fig. 1B). Next, the differential of this plot is computed (dotted line in Fig. 1B). A biphasic profile in this differential plot is now sought. If it is absent, the noise is assumed to be >20% of maximal signal amplitude and the recording is discarded. If it is
However, in a separate analysis we confirmed that the mean ENG activity does not span all or most of the corresponding phase. and cycle duration may thereby emerge if the duration of the nerve Significance in the estimation of the relationship between phase duration of excitatory drive to the corresponding motoneuron pool. Significant errors in the estimation of the relationship between phase duration and the error of underestimating the duration of activity common to the summed mean and SD of "noise." This last manipulation reduces the amount of variation in burst duration depended on which of the two behaviors applied to that particular cat. We therefore decided to divide the data into two groups, corresponding to extensor- and flexor-dominated behavior.

Figure 2A shows a recording of nerve activity during fictive locomotion evoked by the stimulation of MLR. Note that the bursts recorded in the flexor nerve (Sart) occupied >50% of the cycle durations, whereas those of the extensor nerve (SMAB) occupied <50% of the cycles. Using automatic threshold detection as described in METHODS, the flexor and extensor phase durations were computed and plotted against cycle duration in Fig. 2B. The - - - reference line has a slope of 0.5 and passes through the origin. Data points on this line correspond to cycles in which phase duration is 50% of cycle duration. Data points above and below the - - - correspond to cycles in which phase duration is greater or <50% of cycle duration, respectively.

In Fig. 2B, flexor phase durations are mostly above the line and extensor phase durations are mostly below. When the average of phase durations of all flexor nerves in an episode is >50% of cycle duration, we will refer to such patterns as flexor-dominated. Figure 2C shows recordings from another fictive locomotor cat. This time extensor bursts (GSol nerve) were longer than flexor bursts as in intact cats. Figure 2D confirms that in this case extensor phase durations are mostly above the line and flexor phase durations are mostly below. Because the average of phase durations of all extensor nerves in this episode is >50% of cycle duration, we call this an extensor-dominated pattern.

Figure 3 shows pooled data from 31 episodes in 23 fictive MLR cats, 22 episodes showing flexor-dominated patterns (Fig. 3A) and 9 showing extensor-dominated patterns (Fig. 3B). The nerve recordings were divided into two groups, those from the flexors Sart, TA, EDL, PerL, and RF and those from the extensors SMAB, FHL, GSol, Plan, Quad, and FDL/FDHL. This grouping is based on the biomechanical action of the corresponding muscles in flexor and crossed-extensor reflexes and the spatiotemporal activation of their motoneurons in the spinal cord during locomotion (Eccles and Lundberg 1958; Prochazka et al. 2002b; Sherrington 1910).

Each gray regression line represents a set of phase-duration data for a given muscle nerve in a given cat. Solid lines are the means of the individual regression lines. Most, though not all, lines corresponding to flexors in Fig. 3A are above the (dashed)
reference line and have slopes >0.5, whereas most, but again not all, extensor lines are below the reference line and have shallower slopes. The reverse is true in Fig. 3B.

Many of the individual regression lines (gray) did not extrapolate to the origin, meaning that phase duration was not a constant proportion of cycle duration. Several had negative slopes (e.g., Sart in Fig. 3B) meaning that phase durations declined as cycle durations increased. These findings have important implications for the mechanism of central pattern generation, as discussed later.

Figure 4, A and B, shows the means of all the individual regression lines from Fig. 3, A and B, combined into flexor and extensor groups. The regression slopes in Fig. 4A (flexor-dominated pattern) were 0.75 (flexor) and 0.14 (extensor) and in Fig. 4B (extensor-dominated pattern), 0.27 (flexor) and 0.82 (extensor). The flexor and extensor slopes in Fig. 4B (extensor-dominated pattern) are similar to those previously reported for normal cats during overground gait (Halbertsma 1983) (see Fig. 8C), whereas the slopes in Fig. 4A suggest a fundamental shift in the balance of rhythm generation in these experiments.

Figures 5 and 6 deal with the statistical analysis. Figure 5 shows the results of a one-way ANOVA of the slopes of the phase adjustments during flexor and extensor-dominated fictive locomotion. The mean slope of the bifunctional (knee extensor, hip flexor) RF was not significantly different from those of flexors or extensors. Figure 6 shows a comparison of the means in the groups of Fig. 3, RF omitted. In flexor-dominated (Fig. 6A) and extensor-dominated (Fig. 6B) fictive locomotion, the slopes of the flexor and extensor groups of nerves were significantly different.

**DISCUSSION**

In intact animals and humans, changes in locomotor cadence are usually due more to variations in stance (extensor phase) durations rather than swing (flexor phase) durations (Arshavsky et al. 1965; Goslow et al. 1973; Halbertsma 1983; Prochazka et al. 2002b). The present results demonstrate that during fictive locomotion and without rhythmic afferent feedback, the locomotor step cycle period can vary spontaneously and display unequal flexion and extension phase durations. In 22 of the 31 experiments involving MLR-induced fictive locomotion, we analyzed, the variations in flexor burst durations were larger than those of extensors. In the remaining 9 experiments, extensor bursts varied more. The term “dominant” corresponds to the phases showing the larger variation. Figure 4 shows that for the largest cycle durations, the dominant phase occupies ~80% of the cycle. For cycle durations of 0.35–0.45 s, the phase durations converge to 50% of the cycle and for still lower cycle durations the relative share reverse. In normal cats, flexor and extensor phase durations converge at gait velocities of 1.3–1.8 m/s (Goslow et al. 1973). In reviewing the literature, we found a few anecdotal reports of occasional flexor-dominated patterns in experiments involving fictive locomotion in the cat (Berkinblit et al. 1978; Douglas et al. 1993; Gossard et al. 1994; Grillner and Zangger 1979; Noga et al. 2003).

What is it about MLR-induced fictive locomotion that often results in flexor-dominated patterns? Or to put it the other way, what is it about locomotion in the intact animal that results in extensor-dominated patterns? During normal locomotion, biomechanical events elicit varying sensory inputs that modulate the centrally generated rhythm, predominantly in the stance phase of the cycle. Consider the effect of a small increase in swing duration. This results in a more forward placement of the...
foot relative to the body. The initial ground reaction force is angled back further toward the hip, decelerating the body and causing kinetic energy to be lost. The time taken for the body to vault over the point of ground support increases, as does the period of extensor thrust needed to accelerate the body again and re-extend the hip. Given that the transition to the next swing phase is held off by sensorimotor mechanisms until the leg is far enough back and unloaded and the contralateral leg is on the ground (Prochazka 1996), it is easy to see how the stance phase might be more deeply modulated by biomechanical factors and corresponding sensory feedback than the swing phase. This argument is further illustrated by the relative increase in modulation of flexor phase durations during air-stepping (Smith et al. 1986). Sensory-mediated prolongation of the extensor phase could therefore explain why extensor-dominated patterns predominate in normal intact locomotion.

In MLR-induced fictive locomotion, spontaneous cycle period changes can occur with a constant level of midbrain subcortical activity.
stimulation. Presumably these variations result from fluctuating synaptic input from local neuronal networks, fluctuations in intrinsic neuronal properties, or alterations in the excitability of the MLR-reticulospinal pathway system. During such variations, phase durations co-vary in an orderly way, as shown by the linearity of the phase duration plots. We wondered whether these properties might provide clues regarding basic oscillator mechanisms.

To explore this, we designed an oscillator model with the use of Matlab Simulink software. Figure 7 shows the Simulink model, with parameters chosen to model the phase duration plots of Figs. 2B and 8A (flexor-dominated data). The circuit is typical of electronic oscillators and hypothesized biological oscillators (Cohen et al. 1982; Marder and Bucher 2001) in that it has two mutually inhibitory “half-centers” the duty cycles of which are determined by timing elements, in this case a pair of resettable integrators the outputs of which trigger bistable “flip-flops” to change state from low to high when they receive an input equal to 1.

Phase durations can be set to the longest and shortest phases in a given set of experimental data by setting the offset and gain parameters to appropriate values. Consider offset A (0.864) and offset B (2.877) in Fig. 7. Integrator B outputs a ramp that reaches the trigger value 1 after $1/0.864$ (i.e., 1.2 s, the longest flexor phase duration in Fig. 8A). This triggers flip-flop B to go from 0 to 1, resetting integrator A, which in turn triggers flip-flop A after $1/2.877 = 0.35$s, the longest extensor phase duration in Fig. 8A. Integrator B is reset and the next cycle commences. In addition to the offsets, a very slow ramp input is supplied to each of the integrators through gains A and B. This gradually speeds up the integrators, decreasing phase durations. Thus at 100 s, the input to integrator A is $2.459/0.864 = 3.32$, resulting in a phase duration of $1/3.32 = 0.30$s, the shortest flexor phase in Fig. 8A. Similarly the input to integrator B is $1.476/2.877 = 0.43$, resulting in a phase duration of $1/0.43 = 0.23$s, the shortest extensor phase in Fig. 8A.

FIG. 5. The results of 1-way ANOVAs of the slopes of phase duration plots in flexor-dominated (A) and extensor-dominated (B) fictive locomotion. The whiskers show the 95% confidence intervals about the medians.

FIG. 6. Results of the 1-way ANOVA and multiple comparisons of slopes of phase duration plots using Tukey’s honestly significant difference criterion in flexor(A) and extensor (B) dominated fictive locomotion. Whiskers show confidence intervals about the median.
Note that these oscillators produce a constant amplitude output that represents the basic alternating flexion-extension pattern. The model could be made more realistic by adding elements that distribute oscillator output to motoneurons and adjust their activity levels (Rybak et al. 2004).

Three sets of data were fitted: Fig. 8, A (flexor-dominated MLR fictive), B (extensor dominated MLR fictive), and C [data from normal cats (Halbertsma 1983)]. We were surprised at how closely the modeled phase duration plots matched those of the raw data (in the case of the Halbertsma et al. data, right down to the slightly curved relationships which fitted the raw data better than the linear regressions used by the authors). This suggests that the basic CPG oscillator behaves like reciprocally coupled integrators. What other inferences can be drawn? First, it is clear that to fit the various slopes and offsets of the phase duration curves, separately adjustable timing elements for flexion and extension are required. This agrees with the conclusion of a previous study on fictive scratching and locomotion (Berkinblit et al. 1978). Second, because changes in flexor and extensor phase duration nearly always occur in parallel, it seems likely that there is common or parallel input to the timing elements. Third, because phase durations co-vary by different amounts, either the common input is unequal, the sensitivity to the common input is unequal, or both. Fourth, additional nonvarying input ("offset") is required to match the mean phase durations in the data. Fifth, the goodness of fit of the modeled data from normal cats (Fig. 8C) indicates that the inclusion of rhythmic sensory input does not change the basic way in which phase durations within the step cycle period are accomplished. For example, the phase prolongation produced by extensor group I afferent input could be accomplished by adding an additional offset to integrator A in Fig. 8.

Suppose that "offset" in the model corresponds to background excitation of the neuronal networks of the CPG that make up the timing elements. This excitation would include synaptic input such as that elicited by MLR stimulation as well as other sources and could be preparation dependent. The neuronal analogy of the "gain" term in the simple oscillator is the range of variation of recruitment and spiking of timing-generating neuronal populations. Because both recruitment and firing increases are saturable phenomena, the half-center with the lower background excitation would have the higher gain. Thus variations in background drive would result in larger variations in phase duration in this half-center (i.e., this would be the dominant one). Variability in the balance of background excitation between preparations could help explain why some of our cats were flexor-dominant and others were extensor-dominant. Note that anything that changed the excitability of some of the members of an oscillator could change the phase dominance. This could include random or preparation-specific changes in either excitation or inhibition of any of the elements.

FIG. 7. Oscillator model with 2 mutually inhibitory “half-centers” the duty cycles of which are determined by the resettable integrators. The modulating input on the left is supplied to both integrators through separate gain stages with individual offsets. The inputs to the integrators determine how quickly their outputs reach an arbitrary trigger level and thereby reset the output of the other integrator (details in text). The model was developed in Matlab Simulink 6.5. The modified Runge-Kutta (odetb23tb) variable-step integration method was used for simulations.

FIG. 8. Burst duration plots from Fig. 2 (A and B) and phase duration plots from normal cats [C: Halbertsma (1983)]. Filled circles: flexor burst or swing-phase durations, open circles: extensor burst or stance-phase durations. Triangles show the trajectories of phase durations obtained from the model in Fig. 7, having adjusted the gain and offset parameters to fit the start and endpoints of the regression lines (solid) fitted to the data points. Note the remarkable correspondence of the actual and modeled phase duration plots.
affected by a variety of factors such as head, trunk or leg posture (Pearson et al. 1992), neurotransmitter balance (Jacobs and Fornal 1999), the balance of descending drives (Noga et al. 2003) and overall state.

In this regard, it is interesting to note that Pearson and Rossignol showed that limb position can influence the intensity and relative durations of the flexor and extensor bursts of activity in fictive locomotion in chronic spinal cats. Moving the legs from flexion to extension stretched flexor muscles and progressively increased the duration of flexor bursts and decreased the cycle period, though not to the extent of switching from extensor- to flexor-dominated patterns, at least in the data shown (Pearson and Rossignol 1991).

Could the full range of phase duration plots presented above be obtained with a model that only had gain elements and dispensed with offsets? The answer is no. If gains were the only contributing parameters, the curves would all pass through the origin because each half-center’s share of the cycle duration would be constant. Many of the regression lines we presented in this paper did not pass through the origin.

Several individual regression lines had negative slopes, meaning that phase durations declined as cycle durations increased. In the model, such lines can readily be matched simply by setting the gain of the relevant integrator to an appropriate negative value (the offset remaining positive). The neural correlate of this would be a declining excitation or an increasing inhibition of the drive to the relevant timing neurons.

One of the implications of the present findings is that while most preparations showed a particular bias for the flexion or extension phase during fictive locomotion, the structure of the locomotor CPG does not appear to be inherently extensor or flexor biased. In other words, the flexor or extensor sides of the locomotor oscillator can operate in similar fashion (compare Fig. 8, A and B). This in turn supports models of CPG structure in which either the flexor or extensor phases can dominate (see Rybak et al. 2004).

To conclude, in this paper, we report a considerable flexibility in the control of phase durations in MLR-induced fictive locomotion, which can be explained by changes in background excitation and sensitivity of timing elements in a simple half-center model of the locomotor CPG.

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Present address of S. Yakovenko: Dept. of Physiology, University of Montreal, CP 6128 Succ. A, Montreal, PQ H3C 3J7, Canada.

GRANTS

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