Effects of stimulation of hindlimb flexor group II afferents during fictive locomotion in the cat

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1. This study examines the effects of electrical stimulation of hindlimb flexor nerves on the fictive locomotion pattern. Locomotion was initiated by stimulation of the mesencephalic locomotor region in the decerebrate paralysed cat and monitored by recording the electroneurogram from selected hindlimb flexor and extensor muscle nerves. Flexor nerves were stimulated using short trains (20–50 stimuli at 100 Hz) during either the flexor or the extensor phase of the fictive locomotor cycle.

2. Stimulation of tibialis anterior (TA), posterior biceps and semitendinosus (PBSt) or sartorius (Sart) nerves at 5 times threshold (T) during the flexor phase of the fictive locomotor cycle terminated on-going activity in flexor nerves and initiated activity in extensors. Thus, flexor nerve stimulation during flexion shortened the locomotor cycle by resetting to extension. The failure of lower intensity ($2T$) stimulation of PBSt or Sart nerves to reset the step cycle to extension suggests that group II afferents are responsible for these actions. Resetting evoked by $2T$ stimulation of the TA nerve may be due to a high proportion of group II afferents with low electrical threshold.

3. During extension, stimulation of TA and PBSt nerves at $5T$ did not perturb the locomotor rhythm whereas Sart stimulation prolonged the locomotor cycle.

4. Stimulation of cutaneous or knee joint afferents failed to produce effects similar to those evoked by stimulation of flexor muscle nerves at group II strength. These findings are at odds with those obtained elsewhere in the acute spinal, DOPA fictive locomotion preparation. The possibility that group II resetting during fictive locomotion is not mediated by flexion reflex pathways but by previously unknown pathways released in the present preparation is discussed.

5. Since many of the flexor afferents recruited by $5T$ electrical stimulation are the length-sensitive group II fibres, spindle secondaries may act to regulate the duration and onset of flexor and extensor activity during real locomotion. The resetting from flexion to extension also suggests that unexpected or enhanced activity of flexor secondaries during swing would promote a switch of the step cycle to stance.

It is well established that the mammalian spinal cord contains neuronal circuitry capable of generating the basic locomotor pattern, i.e. the central pattern generator (CPG; for review see Delcomyn, 1980). Information from sensory afferents is, however, essential in order to adapt and compensate for changing conditions during locomotion (for review see Rossignol, Lund & Drew, 1988). One way that sensory input may influence locomotor movement is by actions on the spinal neuronal networks comprising the CPG. Accordingly, sensory input can modify the operation of the CPG with a resulting change in the locomotor pattern within a limb and between limbs. The ability of group I muscle afferents to modify locomotion has been investigated in several studies (Conway, Hultborn & Kiehn, 1987; Pearson, Ramirez & Jiang, 1992; Pearson & Collins, 1993; Gossard, Brownstone, Barajon & Hultborn, 1994; Guertin, Angel, Perreault & McCrea, 1995). During both mesencephalic locomotor region (MLR)-evoked and DOPA-induced fictive locomotion, stimulation of extensor group I muscle afferents promotes hindlimb extensor activity either by resetting the locomotor step cycle to extension or by increasing the amplitude and duration of extensor activity. While the influence of flexor group I afferents appears weak (Conway *et al.* 1987), the possibility that group II afferents

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in flexor nerves can affect the fictive locomotor step cycle has not been investigated.

At rest, activation of group II muscle spindle fibres produces complex reflex actions (for review see Matthews, 1972; Lundberg, Malmgren & Schomburg, 1987*b*). One of the routes by which group II afferents can exert reflex actions is through flexion reflex circuits. Thus, stimulation of any of the flexion reflex afferents (FRA) including the group II afferents produces ipsilateral flexion and contralateral extension in the low-spinal animal (Eccles & Lundberg, 1959*a*). However, depending on the preparation, stimulation of the FRA may produce excitation in ipsilateral extensor motoneurones (Eccles & Lundberg, 1959*a*; Hongo & Pettersson, 1988). There is also extensive evidence for descending control of spinal FRA pathways (see Holmqvist & Lundberg, 1961).

It has been proposed that during locomotion there is a reorganization of spinal FRA pathways such that short latency FRA reflexes evoked at rest are replaced by longer latency reflexes. Because these longer latency actions are seen in preparations displaying alternating excitation of flexor and extensor motoneurones, it was proposed that FRA stimulation could activate interneurones in the spinal CPG for locomotion (Jankowska, Jukes, Lund & Lundberg, 1967). However, group II muscle spindle afferents also have access to spinal reflex pathways other than FRA pathways (Edgley & Jankowska, 1987; Lundberg *et al.* 1987*b*). In this regard, Edgley & Jankowska (1987) have suggested that group II afferents could contribute to the shaping of the locomotor pattern by activation of midlumbar interneurones receiving strong input from group II afferents but less affected by higher threshold skin and joint afferents (i.e. other FRA).

The present study examines the effects of activation of group II afferents in hindlimb flexor nerves during fictive locomotion evoked by stimulation of the MLR in the decerebrate cat. This preparation is identical to that used in the accompanying study in which extensor group I afferent activation was found to be effective in resetting fictive locomotion to extension (Guertin *et al.* 1995). As will be shown, stimulation of flexor nerves at group II but not group I strength can also reset the step cycle to extension or enhance on-going extension.

Some of the present results have been reported previously in abstract form (Angel, Guertin, Perreault, Carr & McCrea, 1993; Perreault, Angel, Guertin, Carr & McCrea, 1993).

METHODS

Data were obtained from thirteen cats of which seven were also used in the accompanying study in which the methods used are described in detail (Guertin *et al.* 1995). Briefly, dissection was performed under halothane–nitrous oxide anaesthesia (~1·5% halothane; 70% N_2O–30% O_2), which was discontinued after a precollicular–postmammillary decerebration with removal of all tissue anterior to the transection. The nerves innervating the following muscles were cut and dissected for either recording or stimulation: sartorius (Sart, medial and lateral branches combined); quadriceps (Q); semimembranosus (Sm) and anterior biceps (AB, sometimes taken together as SmAB); posterior biceps and semitendinosus (PBSt); lateral and medial gastrocnemius (LG and MG); soleus (Sol); plantaris (Pl); tibialis anterior (TA) and extensor digitorum longus (EDL; deep peroneal when taken along with TA); and flexor digitorum longus (FDL). In three cats the posterior articular (Joint) and the combined lateral and caudal sural (Sur) nerves were also dissected. In one cat, the ipsilateral L4 and L5 ventral roots were cut.

The animal was paralysed with intravenous gallamine triethiodide (2–3 mg kg^{-1} h^{-1}) and periods of fictive locomotion (60–120 s) were induced by stimulation of the MLR. Peripheral nerve stimulation (0·1 ms pulse duration) was triggered from the onset of activity recorded in one of the flexor or extensor nerves. The duration of the stimulus train was between 200 and 500 ms (trains of 20 or 50 stimuli at 100 Hz). Such durations are of the order of, or less than, the duration of flexor activity during locomotion and are similar to the duration of afferent feedback that might occur during locomotion. The strength of nerve stimulation was expressed in multiples of threshold (T) for the most excitable afferent fibres.

The onset of activity in the Sart or TA nerves was used to define the beginning of the flexion phase of the locomotor cycle. The onset of extension was determined from the onset of activity in hip or ankle extensor nerves. The locomotor cycle period was defined as the time between the onset of two consecutive bursts of activity in the ipsilateral AB or SmAB nerves. Control cycle periods were measured during fictive locomotion in the absence of peripheral nerve stimulation.

RESULTS

Stimulation of flexor nerves during flexion

The effects of the activation of flexor nerve afferents on the locomotor step cycle were investigated during MLR-evoked fictive locomotion in thirteen decerebrate, paralysed cats using electrical stimulation of Sart, PBSt and TA nerves. Figure 1 shows fictive locomotor activity in eight ipsilateral and one contralateral muscle nerve and the effect of trains of stimuli to the TA nerve. The pairs of vertical dashed lines indicate the onset and termination of TA stimulation which was delivered 160 ms after the onset of activity in the Sart nerve. In this example, TA stimulation was delivered every fifth step. Fifty milliseconds after the onset of TA stimulation the activity in the Sart nerve ceased and locomotor bursts appeared in hip (SmAB) and ankle extensor (MG, LG, Sol and Pl) nerves. Thus 5T stimulation of the TA nerve during flexion reset the fictive step cycle to extension. During unperturbed fictive locomotion, the onset of activity in all extensor nerves in the limb was almost synchronous (Fig. 1, arrow and continuous vertical line). During resetting, however, the burst initiated in SmAB and Q (not shown) nerves preceded that in MG, LG, Sol or Pl nerves. The onset of activity in the Sol nerve was

similar to that of the other ankle extensors; the small deflection during the time of stimulus delivery is a stimulus artifact.

In most experiments, the PBSt nerve exhibited a short burst of activity at the onset of flexion. In some cases, however, activity in PBSt consisted of an additional burst of longer duration during extension (compare IpPBSt and CoPBSt activity in Fig. 1; see also Perret & Cabelguen, 1980). When TA (Fig. 1) or Sart (not illustrated) stimulation was delivered after the short duration ipsilateral PBSt burst but still during flexion, an additional burst of activity in the ipsilateral PBSt nerve which preceded extensor nerve activity could be evoked. This type of response can be seen during the second but not the first train of TA stimulation illustrated in Fig. 1. When the stimulation was given earlier in the flexor phase, the amplitude of the on-going burst in ipsilateral PBSt was increased (not shown). The effects of ipsilateral flexor nerve stimulation on the activity of contralateral nerves were inconsistent and it was not possible to generalize about changes in the contralateral limb.

Figure 2 shows that 5*T* stimulation of TA (*A*), PBSt (*B*) or Sart (*C*) nerves during flexion produced similar changes in the locomotor pattern. In all cases, the stimulation shortened the period of activity of flexor nerves and evoked activity in ipsilateral extensor nerves. Following PBSt, Sart or TA stimulation, newly initiated bursts in SmAB and Q preceded those recorded in the ankle extensors. The ability of TA, PBSt or Sart stimulation to reset the fictive locomotor step cycle was assessed in thirteen experiments. Resetting was observed following TA stimulation in 8/10, Sart stimulation in 5/8 and PBSt stimulation in 4/7 experiments. In one experiment, both TA and Sart stimulation reset the step cycle but PBSt did not.

The effects on the cycle period of stimulation of TA, PBSt or Sart nerves during flexion are illustrated in the graphs in the right-hand panels of Fig. 2. The cycle period (ordinate) was calculated by measuring the time between the onset of two consecutive bursts of activity in either the AB or SmAB nerves. The mean and standard deviation of unstimulated (control) cycle periods are plotted as continuous and dotted lines, respectively. In all graphs of

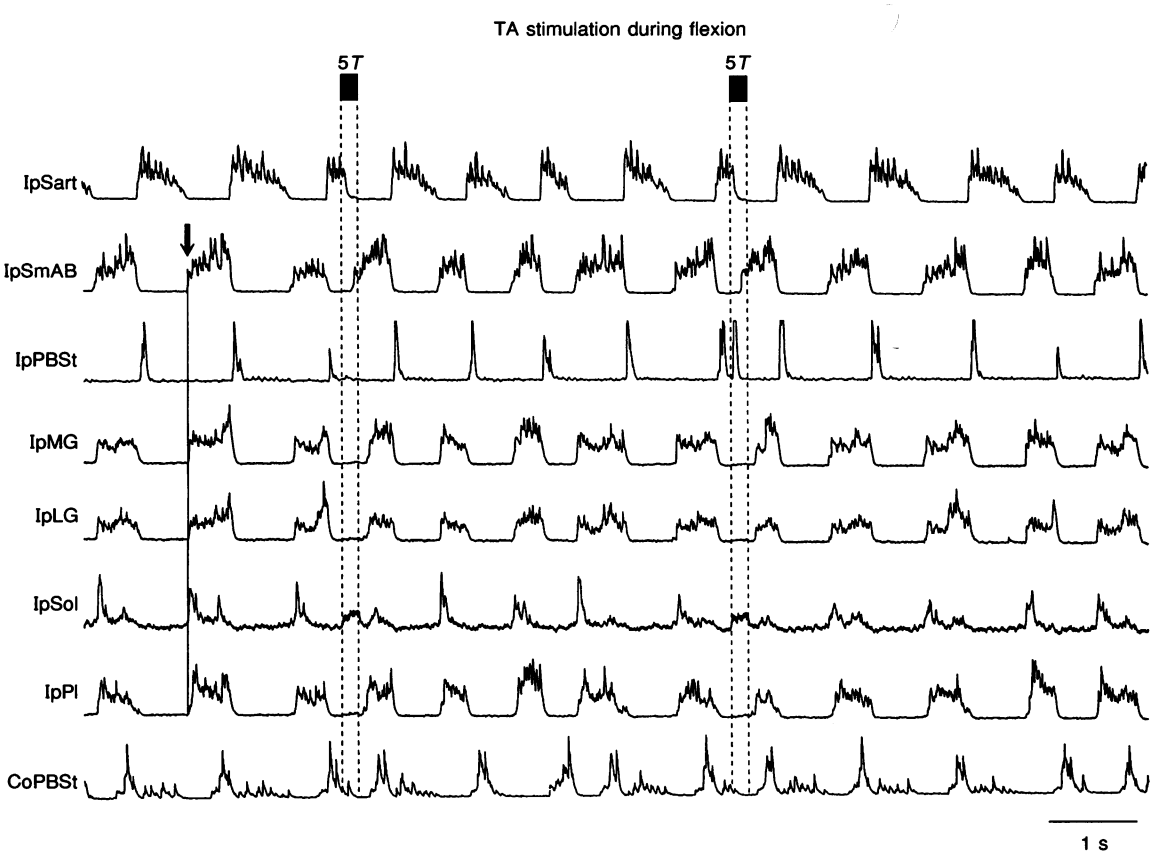

Figure 1. Flexor nerve stimulation at group II strength during flexion resets to extension
MLR stimulation (15 Hz, 80 μA) was delivered continuously to evoke fictive locomotion. The traces shown are integrated and rectified electroneurogram records of ipsilateral (Ip) and contralateral (Co) extensor and flexor nerves. The stimulus train (20 shocks, 100 Hz) to the TA nerve at 5*T* (filled rectangles) was triggered from Sart activity. The vertical dashed lines indicate the onset and offset of TA stimulation. TA nerve stimulation terminated on-going flexion and initiated extension during fictive locomotion.

Figs 2 and 4, the mean control cycle periods were obtained by averaging locomotor cycles during intervals of fictive locomotion without peripheral nerve stimulation. In Fig. 2, mean control periods were obtained during the 0–25 s interval in *A*, the 47–53 and 67–71 s intervals in *B*, and the 0–5 and 78–85 s intervals in *C*. Locomotor step cycle periods during which peripheral nerves were stimulated are indicated by the symbols. Stimulation (5*T*; filled rectangles) of either Sart, PBSt or TA nerves clearly reduced the period of the locomotor cycle. In most cases, the stimulated cycle period was shorter than the mean minus one standard deviation.

The graphs in Fig. 2 also allow a comparison between 5*T* (filled symbols) and lower strength stimulation (open symbols). Lower strength stimulation of TA did not affect the mean cycle period (Fig. 2*A*, right panel). Similarly, PBSt 2*T* stimulation in most cases did not change the cycle period (Fig. 2*B*, right panel). The occasional reduction or increase in the duration of individual step cycles evoked by 2*T* stimulation may result from competition between

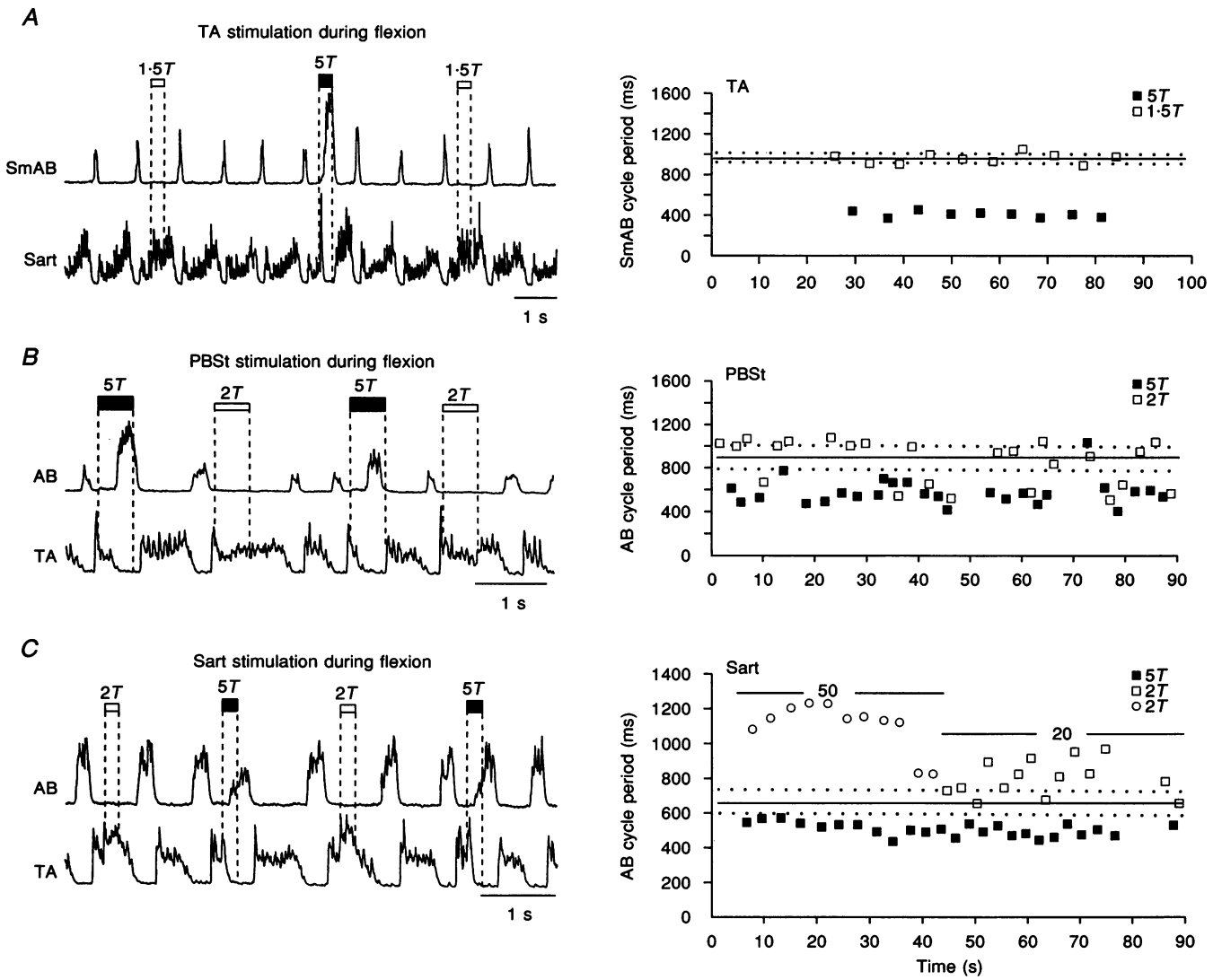

Figure 2. Effects of flexor nerve stimulation during flexion on the locomotor cycle period

The effects of stimulation of the TA (*A*; 100 Hz trains of 30 pulses), PBSt (*B*; trains of 50 pulses) and Sart (*C*; trains of 20 pulses) nerves during flexion on the step cycle period were examined. Filled and open rectangles represent stimulation at group I and II intensities, respectively. Left panels show representative responses evoked in ipsilateral extensor and flexor nerves. Right panels, corresponding analysis of changes in the locomotor cycle period evoked by flexor nerve stimulation. The horizontal continuous lines indicate the mean control locomotor cycle and the dotted lines the standard deviation. The number of unstimulated cycles that were averaged to calculate the mean control cycle period was 29, 14 and 11 for the graphs in *A*, *B* and *C*, respectively. In the graph shown in *C*, ○ indicates trains of 50 stimuli (50) and □ indicates trains of 20 stimuli (20).

opposite actions evoked by near maximal group I and near minimal group II afferent activation. Near maximal group II afferent activation ($5T$) produced a clear and consistent initiation of extensor activity as evidenced by the reduction in AB cycle period.

Stimulation of Sart at $2T$ prolonged the fictive step cycle (Fig. 2*C*, right panel). This prolongation was a result of increased activity in flexor nerves (Fig. 2*C*, left panel). Increasing the number of stimulus shocks from 20 (□) to 50 (○) further prolonged the step cycle emphasizing the fact that resetting was only produced by $5T$ stimulation. The finding that $5T$ but not $2T$ stimulation of Sart and PBSt during flexion reset the step cycle to extension, indicates that resetting requires the recruitment of fibres conducting in the group II range (see Discussion).

Figure 3 shows the effects produced by stimulation of the TA nerve at 5, 2, 1·8 and 1·5T on the activity in SmAB and Sart nerves. Each trace is the averaged activity of SmAB or Sart nerves during stimulation of TA at the strengths indicated. The traces were superimposed and aligned at the onset of the stimulus train which consisted of 50 pulses at 100 Hz. As shown, stimulation at $\geqslant 1{\cdot}8T$ shortened the period of activity in Sart and advanced the onset of activity in SmAB. Following $5T$ stimulation, flexion was terminated and extension initiated by the fifth pulse in the stimulus train. This figure also illustrates the lack of overlap between flexor and extensor activity during resetting. Only TA stimulation consistently reset the step cycle to extension at stimulation intensities below those considered optimal for recruitment of group II afferents. Arguments that activation of flexor group I afferents is insufficient to produce resetting to extension are presented in the Discussion.

Stimulation of flexor nerves during extension

The effects of $5T$ stimulation of TA, PBSt and Sart nerves were examined during the extensor phase of the locomotor cycle in two, three and six experiments, respectively, with examples illustrated in Fig. 4. Unlike the effects of $5T$ stimulation delivered during flexion (Figs 1 and 2) TA, PBSt or Sart stimulation at $5T$ (200–500 ms trains) during extension did not decrease the locomotor cycle period (Fig. 4, right panels). TA and PBSt stimulation had little effect on the cycle period while Sart stimulation prolonged the extensor phase and the cycle period (Fig. 4*C*, right panel). The effects on the locomotor pattern are also illustrated by the integrated and rectified electroneurogram records in the left panels of Fig. 4. As shown, TA, PBSt and Sart stimulation produced some increase in the activity of the hip extensor. In some experiments, ankle extensor activity was clearly reduced by $5T$ flexor nerve stimulation during extension (not illustrated). Activity in the FDL nerve was often increased, especially following PBSt and TA stimulation (Fig. 4*A* and *B*).

Although not shown, 1·5T stimulation of TA and $2T$ stimulation of PBSt during extension produced effects similar to those resulting from $5T$ stimulation. It is thus likely that activation of group I afferents in these nerves is sufficient to produce the effects illustrated in Fig. 4*A* and *B*.

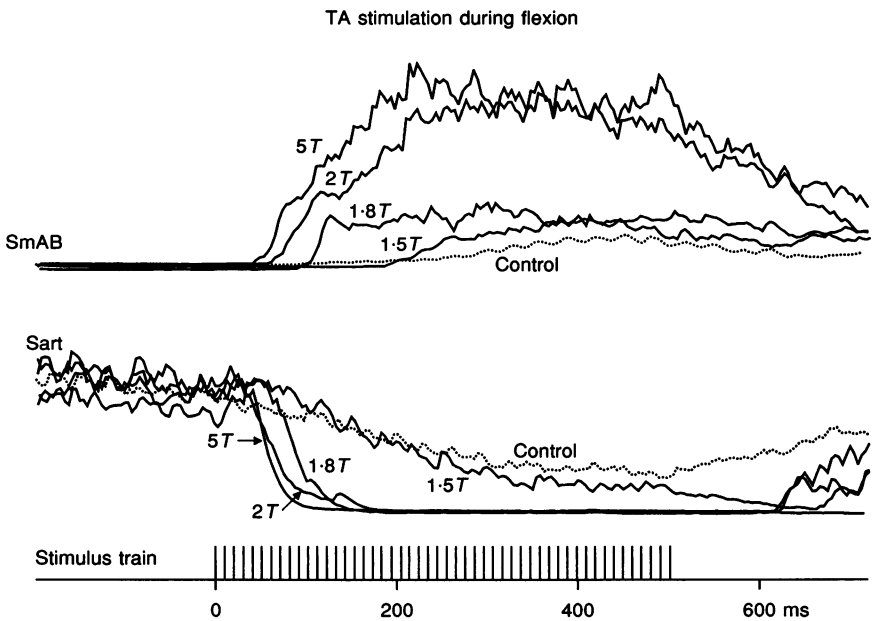

Figure 3. Stimulation of the TA nerve at different intensities during flexion

The averaged activity of a flexor (Sart) and extensor (SmAB) nerve following stimulation of the TA nerve (50 pulses, 100 Hz) at the indicated intensities is shown. Averages were calculated from 7–9 trials and aligned at the onset of the stimulus train. TA stimulation greater than 1·5T terminated Sart activity.

Stimulation of Sart at 1·5T, however, shortened the duration of activity in extensor nerves and initiated a new burst of activity in flexor nerves thus resetting the locomotor cycle to flexion. This strongly suggests that the activation of group II afferent fibres in the Sart nerve is responsible for the prolongation of the locomotor cycle seen during 5T stimulation (Fig. 4C, right panel).

Stimulation of cutaneous and joint afferents

Recently, Schomburg, Petersen, Barajon & Hultborn (1993) reported that during DOPA-induced fictive locomotion in high spinal cat, stimulation of cutaneous, joint and group II muscle afferents produced similar and consistent resetting of the fictive locomotor step cycle to flexion. In three of the present experiments, the effects of stimulation of the cutaneous Sur and Joint nerves were compared with flexor nerve stimulation. Figure 5 shows an example of the effects produced by stimulation of these nerves (open rectangles) compared with those evoked by 5T stimulation of TA (filled rectangles). As shown in Fig. 5A, stimulation of Joint prolonged the duration of Sart bursts while activity in the extensor Pl was delayed until the end of flexion. TA stimulation shortened the on-going activity in Sart and initiated extension. The effect of Sur stimulation (Fig. 5B) was complex producing a short lasting excitation followed by an inhibition and then an excitation in the Sart nerve. An excitation of SmAB was also seen during the period of Sart inhibition. Thus neither Sur nor Joint stimulation produced the clear resetting of the fictive locomotor step cycle seen following TA, PBSt or Sart stimulation.

Section of the ventral roots

Since stimulation of muscle nerves will activate motor axons antidromically, the L4 and L5 ventral roots were

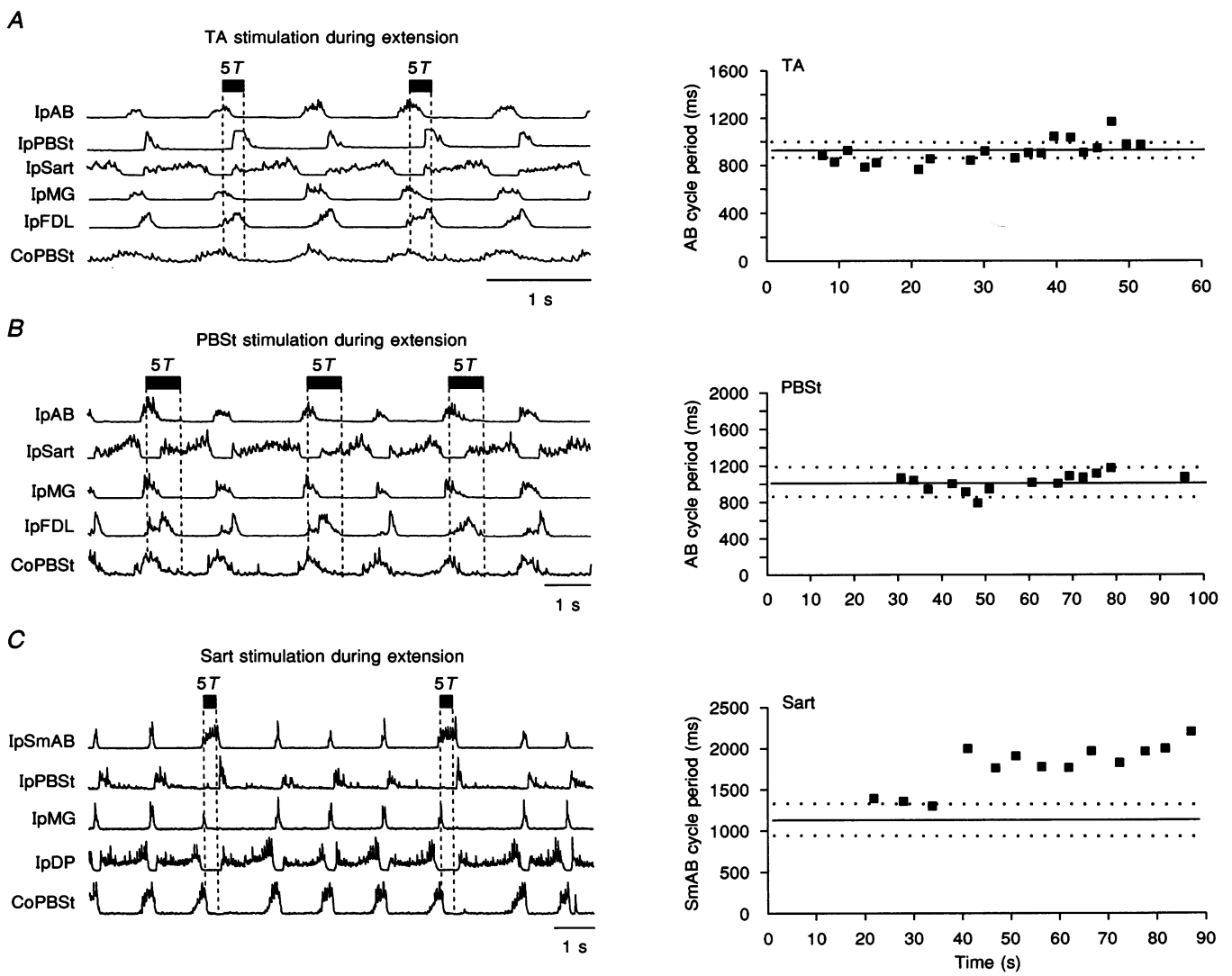

Figure 4. Effects of flexor nerve stimulation during extension on the locomotor cycle period

The effects of TA, PBSt and Sart nerve stimulation at 5T during extension (trains of 20, 50 and 30 pulses in A, B and C, respectively) are shown in a format similar to that in Fig. 2. Control cycle periods in A, B and C were calculated from 14, 17 and 28 unstimulated cycles, respectively. Only Sart stimulation during extension increased the period of the fictive locomotor step cycle.

sectioned in one experiment to avoid activating axon collaterals of Sart motoneurones (Romanes, 1964). Sectioning Sart motor axons did not impair the ability of 5*T* Sart stimulation to reset the locomotor cycle (not illustrated). Thus, activation of the spinal circuitry from motor axon collaterals is not required for resetting the step cycle with 5*T* stimulation of Sart. Similar experiments were not attempted for PBSt and TA stimulation since the ability to record fictive locomotor activity in many nerves following section of the L6, L7 and S1 ventral roots would have been severely compromised.

DISCUSSION

Type of receptors innervated by fibres in the group II range

The present study demonstrates that electrical stimulation of hindlimb flexor nerves at group II strength during flexion can reset the fictive locomotor rhythm by terminating on-going flexion and initiating extension. These results suggest that flexor group II afferents can have powerful actions on the CPG. One issue arising from this is the type of receptors innervated by these afferents. While the majority of group II afferents in muscle nerves arise from secondary muscle spindles (Boyd & Davey, 1968; MacLennan, 1972; Jack, 1978), afferents from paciniform corpuscules, joint receptors and free endings might also have contributed to these effects. Since muscle nerves contain only few axons innervating paciniform corpuscules (Coppin, Jack & McIntyre, 1969; Jack, 1978), the contribution of paciniform corpuscule afferents to resetting seems unlikely unless one assumes a very powerful effect from single afferent fibres. Although the Sart nerve contains some knee joint afferents (Freeman & Wyke, 1967), their contribution to resetting also appears unlikely since direct stimulation of the posterior knee joint nerve produces effects different from those evoked by Sart stimulation. The involvement of afferents innervating muscular free nerve endings in resetting is more difficult to exclude. The TA nerve in particular contains a substantial number of axons arising from muscular free endings (Stacey, 1969; MacLennan, 1972) preventing a firm conclusion about the relative involvement of muscular free endings and spindle secondaries. Nevertheless, the similar actions of stimulation of TA, Sart and PBSt nerves at group II strength suggest that activation of spindle secondaries can reset the step cycle to extension.

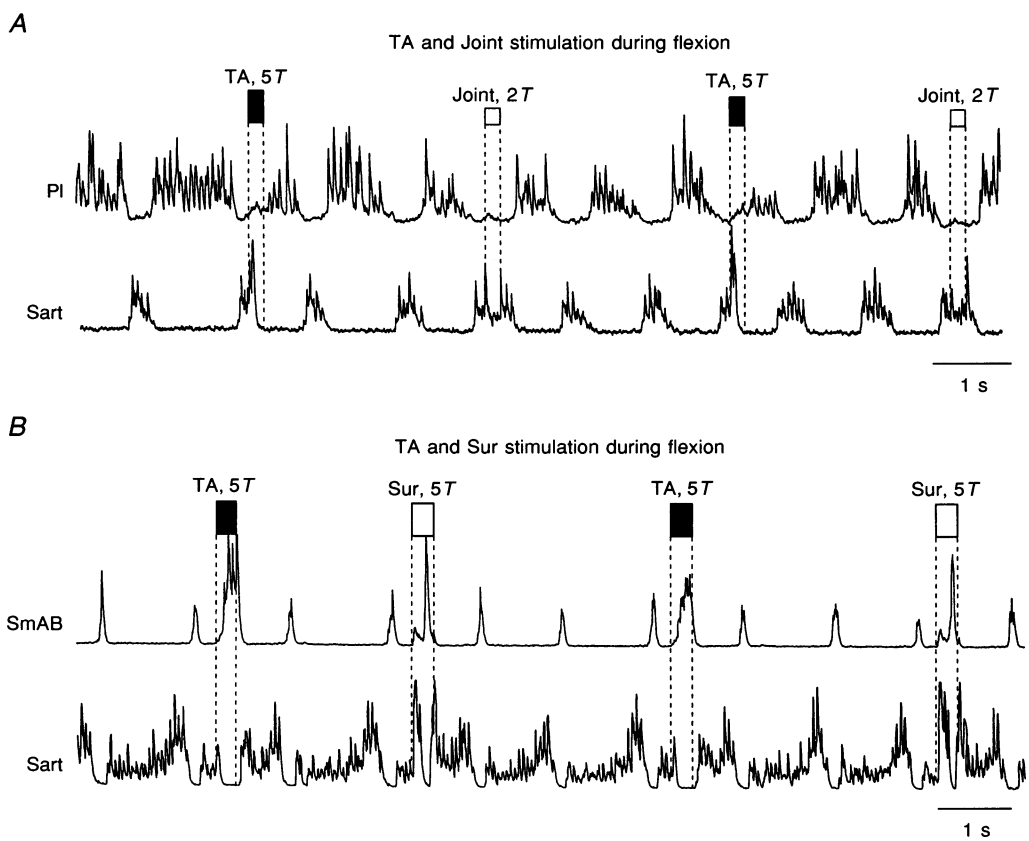

Figure 5. Stimulation of cutaneous, Joint and group II afferents evokes dissimilar actions during fictive locomotion

A comparison of the effects of 5*T* TA stimulation with Joint nerve stimulation (*A*; 2*T*, 20 pulses) and cutaneous (Sur) stimulation (*B*; 5*T*, 30 pulses) delivered during flexion. Neither Joint nor Sur stimulation produced a clear resetting of the step cycle.

Possible contribution from group I fibres and motoneurone axon collaterals

The majority of group II afferents are recruited by raising the stimulation intensity from 2 to 5T (Eccles & Lundberg, 1959*a*; Edgley & Jankowska, 1987). However, because the most excitable group II afferents have thresholds below 2T and some group I afferents have thresholds slightly above 2T, an absolute distinction between the effects evoked by group I and II afferents may not be possible on the basis of electrical threshold (Jack, 1978; Ellaway, Murphy & Tripathi, 1982). In the cases where 5T but not 2T stimulation reset the step cycle there is little doubt that recruitment of group II afferents is responsible, while the ability of 2T TA stimulation (but not 1·5T) to reset the locomotor cycle may reflect a contribution from the least excitable group I TA afferents. On the other hand, resetting evoked by 2T TA stimulation may also be the result of the relatively high proportion of low threshold group II afferents in the TA nerve (Jack, 1978). This in turn suggests that even a few group II TA afferents may have powerful actions on the spinal locomotor generator. A potent action of a few group II afferents may also explain the occasional resetting observed with 2T PBSt stimulation (Fig. 2*B*, graph). We are unaware of data concerning the relative proportion of low threshold group II afferents in the Sart nerve but we expect that the great majority are activated by stimulation greater than 2T. This suggestion arises from the observation that activation of the majority of group I Sart fibres during flexion prolonged flexion while group II activation reset the step cycle to extension (compare 2T and 5T data in Fig. 2*C*). Thus, the contribution of flexor group I afferents to resetting to extension appears weak. Activation of group I flexor afferents is also ineffective in resetting the fictive locomotor cycle in the acute spinal, DOPA cat (Conway *et al.* 1987).

The possible contribution of antidromically activated motoneurone axon collaterals to resetting has also been considered. In the case of Sart stimulation, this possibility has been ruled out by the persistence of resetting after sectioning of the appropriate ventral roots. A preliminary report (Hammond, Miller & Scott, 1981) showed that antidromic activation of an entire ventral root can influence the locomotor rhythm. Other investigations, however, suggest that the contribution of systems activated from motoneurone axon collaterals is minor. Blocking the activation of Renshaw cells alters motoneuronal firing during a locomotor burst but does not affect the timing of the bursts or duration of locomotor drive potentials recorded in motoneurones (Noga, Shefchyk, Jamal & Jordan, 1987). In summary, available evidence suggests that resetting evoked by stimulation at group II strength is primarily the result of activation of afferents arising from secondary muscle spindle receptors.

Neuronal pathways mediating resetting from flexor group II afferents

At present, little is known about the organization of neurons through which group II flexor afferent activity can alter the locomotor step cycle. The only interneurones with strong group II input that have been studied during MLR-evoked fictive locomotion are those located in the midlumbar segments. They are active during flexion (Shefchyk, McCrea, Kriellaars, Fortier & Jordan, 1990) and remain candidates for mediation of at least the TA and Sart group II resetting reported here. Neither the lower lumbar (Lundberg, Malmgren & Schomburg, 1987*a*) nor sacral (Jankowska & Riddell, 1993) group II interneurones have been studied during fictive locomotion. Further studies on the activity of lumbar interneurones with group II input during fictive locomotion will be needed to address the question of which interneurones mediate group II effects during fictive locomotion. It is also possible that some of the actions reported here are mediated in part through supralumbar pathways.

Because some of the data in Guertin *et al.* (1995) were obtained in the experiments reported here, a direct comparison between the effects of extensor and flexor nerve stimulation is possible. The present data and the accompanying paper (Guertin *et al.* 1995) show that, during MLR-evoked fictive locomotion, stimulation of either flexor group II afferents or ankle extensor group I afferents during flexion resets the locomotor cycle to extension. Whether stimulation of flexor group II and ankle extensor group I afferents produces resetting by common or parallel neuronal pathways remains to be determined. The interneurones responsible for flexor group II afferent-evoked resetting to extension are unlikely to have a strong input from flexor group I afferents since flexor group I afferent stimulation has little or no effect on the fictive step cycle.

During extension, Sart stimulation at group II strength prolongs step cycle duration. Thus during extension, activity in Sart group II afferents can affect the CPG in much the same way as activity in group I extensor muscle afferents (Guertin *et al.* 1995). Stimulation of PBSt or TA nerves during extension has little effect but a subthreshold influence of PBSt and TA group II afferents on the locomotor pattern generator cannot be excluded. Another observation that may be useful for future studies to determine the neuronal targets of the group II (and group I) afferent system is that activation of group II flexor afferents resets to either flexion or extension depending on the preparation used (see below). In contrast, activation of group I extensor afferents resets to extension in the acute spinal DOPA (Conway *et al.* 1987), acute spinal clonidine (Pearson *et al.* 1992) and intact spinal MLR (Guertin *et al.* 1995) preparations.

Flexor group II resetting is not mediated by FRA pathways during MLR-evoked locomotion

One tenet of the FRA concept is that wide convergence from different sensory afferents produces similar actions through common spinal interneurones (Lundberg, 1979). Recently, Schomburg *et al.* (1993) have shown that during DOPA-induced fictive locomotion in high spinal cats, stimulation of flexor muscle nerves at group II strength resets to flexion. Since stimulation of group II, cutaneous and joint afferents in the high spinal cat produces similar responses during fictive locomotion, Schomburg *et al.* (1993) suggested that the common actions of these afferents are mediated through FRA pathways. One characteristic of FRA reflexes is that under appropriate conditions a switch can occur from excitation of flexors to excitation of extensors (see introduction). Thus, it is conceivable that the resetting to extension seen in the present spinal cord intact preparation may result from the activation of alternate (Lundberg, 1979; see McCrea, 1992) FRA reflex pathways. Arguing against this possibility is our observation that stimulation of cutaneous and joint nerves does not reset to extension. This separation of effects from group II and other FRA afferents suggests that resetting to extension from group II afferents is not evoked through the FRA reflex pathways.

It is well documented that at rest in the midcollicular decerebrate animal (Eccles & Lundberg, 1959*b*; Holmqvist & Lundberg, 1961) transmission in FRA pathways is under inhibitory descending control. In the present precollicular decerebrate preparation there is the strong possibility that FRA reflex pathways are also inhibited at rest. During MLR-evoked fictive locomotion, we speculate that short latency FRA reflex pathways remain inhibited and that flexor group II afferents reset the step cycle through other, non-FRA pathways. The identity of these pathways released during fictive locomotion is unknown. The idea that FRA reflexes should be depressed during locomotion is in accord with the first reports concerning FRA reflex pathways; Eccles & Lundberg (1959*b*) suggested that it would be inappropriate for group II afferents to continually elicit flexion reflexes during locomotion and argued for a suppression of short latency, group II-evoked flexion reflexes.

Functional significance and concluding remarks

The limited data on the activity of group II afferents during walking show that while group II afferents in different muscles are maximally active at different times in the step cycle (see Prochazka, Trend, Hulliger & Vincent, 1989) both PBSt (Loeb & Duysens, 1979) and Sart (Loeb, Hoffer & Pratt, 1985) group II afferents show some activity during swing. Although the activity of TA group II afferents during locomotion is unknown, Prochazka *et al.* (1989) suggested that spindle secondaries have maximal firing rates when the muscle is longest. According to this suggestion, TA spindle secondaries should also be active during swing. The present results suggest that increased (excessive) activity of spindle secondaries from TA, Sart or PBSt during swing would terminate flexion and reset the step to extension. During extension, activation of Sart group II afferents can enhance extension (Fig. 4). This suggests that their increased activity at the time of transition from stance to swing would prolong stance and hence assist forward propulsion.

The actions of flexor group II afferents cannot be considered in isolation and we suggest that they function along with group I, cutaneous and joint afferents to help shape both the amplitude and timing of each step. The long duration of flexion often seen during fictive locomotion contrasts with the dominance of extensor activity observed during real locomotion (Goslow, Reinking & Stuart, 1973). It is possible that the extensive denervation and lack of rhythmic afferent activity contributes to the prolonged flexion in some preparations during fictive locomotion. Activity in segmental afferents including flexor secondary muscle spindles could thus be a key determinant of the relative durations of swing and stance. This shaping of the locomotor pattern complements the role that segmental afferents have in initiating the response to limb perturbations.

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