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RESEARCH ARTICLE

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Comparison of the effects of stimulating extensor group I afferents on cycle period during walking in conscious and decerebrate cats

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Abstract Previous studies have reported that stimulation of group I afferents from extensor muscles prolongs stance duration during walking in decerebrate cats. The main objective of this investigation was to determine whether this phenomenon occurs during walking in conscious cats. In conscious cats without lesions of the central nervous system (CNS), stimulation of group I afferents in the lateral gastrocnemius/soleus (LGS) nerve during stance prolonged extensor burst duration and increased the cycle period in five of seven animals. The mean increases in cycle period were modest, ranging from 6 to 22%. In five of six animals that walked both quadrupedally and bipedally at the same rate, the effects on cycle period were stronger during bipedal stepping (18% mean increase in cycle period compared with 9%). The stimulated nerves were transected and the experimental procedure was usually delayed in the conscious animals for 2–3 days following implantation of the stimulating electrodes. To assess whether chronic axotomy of the LGS nerve was a factor in the decreased effectiveness, four of the cats with chronic nerve section were decerebrated and their LGS nerves were stimulated after the animals began to spontaneously walk on a motorized treadmill. In all four of these animals, the effects of stimulating the chronically cut LGS nerve on the step cycle period became stronger following decerebration. However, these effects were not as strong as those produced when an acutely sectioned LGS nerve was stimulated. During both quadrupedal and bipedal walking, stimulation of the LGS nerve increased the amplitude of the medial gastrocnemius (MG) electromyogram. The augmented activity of the MG muscle contributed to an increased extension of the ankle during stimulated steps. The conclusion from these experiments is that stimulation of the group I afferents in extensor nerves can prolong stance in the conscious cat, but this effect is weaker than in decerebrate animals. It is likely that transmission in the polysynaptic group I pathways controlling stance duration is regulated in a complex fashion by descending signals from the brain in the conscious animal.

Key words Walking · Group I afferent · Reflex · Cat

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Introduction

Sensory feedback is an essential component of mammalian locomotor systems, stabilizing the centrally generated locomotor rhythm (Grillner and Zangger 1984) and adjusting the motor pattern in response to variations in the terrain (Grillner 1985). Much of this interaction between segmental afferents and the central pattern generator (CPG) can occur without conscious control. For example, chronic spinal cats that have been trained to walk on a treadmill rely on afferent feedback from the hind legs to regulate stepping. These animals can support their weight during stepping, adjust their cadence to the speed of the treadmill, and even coordinate stepping of the two hindlimbs when each leg is driven at a different speed by a split treadmill (Barbeau and Rossignol 1987; Forssberg et al. 1980a, b). Similar observations have been made in decerebrate animals (Kulagin and Shik 1970; Yanagihara et al. 1993).

Considerable progress has been made in identifying afferent systems involved in regulating locomotor activity in spinal and decerebrate preparations (Gossard et al. 1994; Guertin et al. 1995; Hiebert et al. 1996; Pearson and Collins 1993; Perreault et al. 1995; Whelan et al. 1995a; for reviews, see Pearson 1995; Whelan 1996a, b). For example, feedback from primary muscle spindle (group Ia afferents; Guertin et al. 1995) and Golgi tendon organs (group Ib afferents) in extensor muscles can prolong the extensor burst and delay the onset of the flexor burst in decerebrate locomoting cats (Whelan et al.

1995a). From investigations on spinal and decerebrate cats, it has been proposed that extensor group I afferents can affect the timing of the step cycle via a locomotor-dependent oligosynaptic pathway that accesses the rhythm-generating circuitry of the spinal cord (Conway et al. 1987; Pearson et al. 1992; see Pearson 1995 for review). In reduced preparations, this pathway is not opened in an all-or-none fashion, since it can be gradually expressed following the administration of l-Dopa or stimulation of the MLR (Gossard et al. 1994). These findings suggest that supraspinal pathways may modulate reflex pathways in conscious animals. An important question arising from these recent studies is whether regulatory reflex systems identified in reduced preparations (spinal and decerebrate) function in a similar manner during walking in conscious animals. This may not necessarily be the case (for example, see Duysens and Stein 1978; Forssberg 1979; Hoffer et al. 1990; and Wolpaw and Carp 1993). Different reflex pathways could be selected in the conscious cat to provide timing cues during locomotion, and there may be greater emphasis on descending commands to allow the integration of visual and somatosensory information (Drew 1991).

The main question addressed in the current investigation was whether stimulation of group I extensor afferents increases the cycle period during walking in conscious cats. To investigate this issue, group I afferents in extensor nerves were stimulated in cats without lesions of the CNS that had been trained to walk quadrupedally and bipedally on a motorized treadmill.

Materials and methods

All animals used in this study were cared for in accordance with the guidelines published by the American Physiological Society, and the University of Alberta Animal Welfare Committee approved the experimental procedures. Experiments were conducted on 12 adult cats of both sexes. Of these 12 animals, 3 were rejected due to failure of the stimulating cuff.

Under halothane gas anesthesia, stimulating cuffs were implanted on the lateral gastrocnemius and soleus (LGS, seven animals), vastus lateralis and vastus intermedius (VL/VI, two animals), and/or plantaris (Pl, two animals) nerves. The study focussed on the effects of stimulating group I afferents from the lateral gastrocnemius and soleus (LGS) muscles, because these afferents have a particularly powerful effect on extensor bursts in walking decerebrate animals compared with stimulation of other ankle extensor group I afferents (Whelan et al. 1995a, b). Each nerve was exposed and transected close to the muscle. A 1-cm length was freed and tied into a small bipolar stimulus cuff (see Whelan et al. 1995a for full details). To record the stimulus volley from the LGS or Pl nerve, a recording cuff was placed around the sciatic nerve just distal to the hamstrings' nerve junction. In the two animals with VL/VI stimulating cuffs, the stimulus volley was monitored by recording a cord dorsum potential. A laminectomy was performed at the level of L4–5, and recording wires (AS632, Cooner Wire Company) were placed on the dorsal surface of the spinal cord just beneath the laminae. The wires were held in place by inserting two small pins into each of the L4 and L5 spinous processes and fixing the wires in place by applying a small amount of dental acrylic around the pins and wires. The threshold of the electrical stimulus (pulse duration 0.2 ms) to the extensor nerve was taken as the minimum voltage necessary to produce a just-detectable potential from either the sciatic nerve or the cord dorsum. The strength of the electrical stimulus was expressed in multiples of this threshold level. Bipolar stainless steel electrodes were sewn into combinations of the following muscles of the experimental leg to record the electromyographic (EMG) activity during walking: medial gastrocnemius (MG), vastus lateralis (VL), vastus medialis (VM), semitendinosus (St), tibialis anterior (TA), and lateral gastrocnemius (LG). In some animals EMG electrodes were implanted in one or more of these muscles in the opposite leg.

The wires from both the recording and stimulating electrodes were led subcutaneously and connected to a socket imbedded into a headpiece constructed from dental acrylic. After this procedure, an antibiotic (Amoxicillin, 0.5 g) and an analgesic (Buprenorphine, 0.005–0.01 mg/kg) were administered. Usually each animal was allowed to recover for 2–3 days before beginning the experimental procedure. In one animal the implantation of the EMG electrodes, sciatic recording cuff, and the headpiece was completed 1 week prior to the implantation of the LGS stimulating cuff. During a second surgical procedure, the cuff was implanted onto the LGS nerve as described above. This allowed recordings to be made as soon as 8 h after the implantation of the cuff because of the fast recovery time following the relatively minor second surgical procedure.

All animals were trained to walk quadrupedally (nine out of nine cats) and in some cases bipedally (six out of nine cats) on a motorized treadmill before the surgical procedure(s). Both bipedal and quadrupedal stepping were performed on the same treadmill. During periods of bipedal walking a small platform was placed into the treadmill chamber on which the animal placed ist front paws. The animals were induced to walk by a mixture of food rewards and affection. During the recording of experimental data, markers were placed on the hip, knee, and ankle to enable the kinematics of leg movements to be determined from a video recording. Many animals stepped in bouts following their surgery so that the frequency of the stimulus trains was usually not administered at a fixed rate. Rather, during periods of reliable quadrupedal or bipedal walking, a train of stimuli was applied to an extensor nerve during midstance manually by the experimenter. In all cases the timing of the stimulus train relative to the onset of MG activity was determined automatically using computer software to monitor the amplitude of the rectified and filtered MG EMG. A minimum of three normal step cycles separated each stimulus train. Typically the stimulus trains were delivered every fourth or fifth cycle during bouts of regular walking. The number of stimulus trains delivered during each bout ranged from two to ten depending on the length of the bout. The most commonly used parameters of the stimulus trains were 1000 ms duration, 1.8×*T*, 200 Hz delayed 200 ms from the onset of the MG EMG. These stimulus parameters were similar to those used previously in walking decerebrate cats (Whelan et al. 1995a). In two animals, stimulus trains (300 ms duration, 1.8×*T*, 200 Hz, 0–30 ms delay from the onset of the TA or St EMG) were delivered during the flexion phase of the step cycle during bipedal (one animal) or quadrupedal walking (one animal).

To compare the effects of stimulation of the LGS nerve on increasing the cycle period in conscious and decerebrate animals, five of the conscious cats in which data had been recorded were decerebrated. During this procedure the animal was anesthetized using halothane gas. A minimum amount of dissection was performed and the chronically implanted cuff was used to stimulate the LGS nerve. The decerebration was performed by transecting the brainstem just rostral to the superior colliculi and continuing the cut just rostral to the mammillary bodies, thus producing a premammillary preparation (Shik et al. 1966). Approximately 1 h after decerebration, walking commenced in four of the five animals. This walking lasted for 1–3 h (depending on the animal) and occurred in bouts lasting for approximately 5–10 min each. The animals walked at a speed that ranged from 0.25 to 0.4 m/s. During walking, a stimulus train was applied to the LGS nerve using stimulus parameters identical to those used when the animal was conscious. Stimulus trains were delivered at a rate of one every 3 cycles during decerebrate walking. An additional stimulus cuff was implanted onto the LGS nerve of the contralateral leg in three out of four decerebrate cats. During decerebrate walking the effects of stimulating the chronically sec-

tioned LGS nerve were compared with those produced by equivalent stimulation of the acutely sectioned LGS nerve to test whether long-term axotomy caused a reduction in the effects.

All data were recorded on magnetic tape using a Vetter 4000A PCM recorder. Later, sequences were selected by matching the video footage to the EMG traces to determine whether walking was regular. These selected sequences were digitized and stored on computer disc using the Axotape (Axon Instruments) data acquisition system installed on a Microexpress 486 computer. The EMGs were rectified and low-pass filtered (100-Hz cutoff). Data analyses were carried out using custom programs that could retrieve data from the Axotape files. The cycle periods before, during, and after the stimulus were calculated only from sequences of regular walking. Each cycle period was calculated as the time between the occurrence of successive St or Ip bursts. The mean and standard deviation for these cycle periods were calculated and Student's *t*-tests were done to detect significant differences between the conditions. Comparisons between bipedal and quadrupedal trials were only made when the mean cycle periods of both control trials were similar ($P > 0.1$). Data that did not meet these criteria were excluded from this section of the analysis. Two methods were used to normalize the data: (1) percentage increase in cycle period, $[(b–a)/a] \times 100$, where b indicates the stimulated cycle period and a the control cycle period; and (2) the percentage of stimulated trials in which the cycle period exceeded the sum of the control cycle period and its associated standard deviation.

Kinematic data, including stick figures, were obtained by analyzing selected video sequences (30 frames/s) acquired using Video Blaster software (Creative Labs) and custom-designed software.

Results

Influence of extensor group I afferents on the cycle period during walking in conscious animals

Stimulation of the group I afferents in the extensor nerves (trains, 1000 ms duration, 200 ms delay from the onset of MG EMG activity, 200 Hz, 1.8×*T*) usually increased the duration of the extensor burst and delayed the onset of the following St burst, thus affecting the cycle period (Fig. 1B). However, the effectiveness of extensor group I stimulation in prolonging the cycle period was quite variable from one trial to the next (Figs. 1, 2A). Figure 1A shows that occasionally (13% of the trials in this animal) stimulation of the LGS nerve strongly increased the cycle period. In the trial immediately following the one shown in Fig. 1A (Fig. 1B), LGS stimulation only modestly increased the cycle period. The influence on cycle period during quadrupedal walking is summarized in the bar graphs for individual animals in Fig. 2B. Stimulation of the LGS nerve significantly ($P<0.05$) increased the cycle period in five of seven animals. The mean increase was modest, ranging from 6 to 22%. Stimulation of the Pl (cat 7) or VL/VI nerves (cat 8) also significantly increased the cycle period by similar amounts. Stimulation of the extensor group I afferents usually affected both the ipsilateral and the contralateral step cycle. When stimulation of group I extensor afferents delayed the onset of the ipsilateral swing phase, the duration of the contralateral stance phase was always increased to support the animal's weight. The pattern of stepping would usually return to normal one to two step cycles following the perturbed step (data not shown).

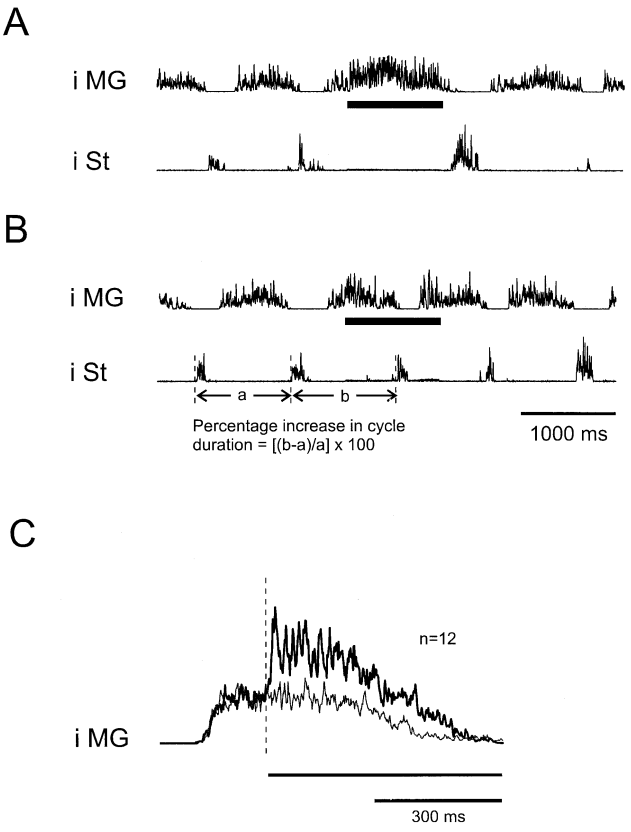

Fig. 1A–C Stimulation of the extensor group I afferents prolongs the extensor burst and delays the onset of the flexor burst in the conscious, quadrupedally walking cat (cat 2, 3 days post-surgery). **A** and **B** show rectified and filtered EMG traces from the ipsilateral medial gastrocnemius (*iMG*) and semitendinosus (*iSt*) muscles for a trial where stimulation of the lateral gastrocnemius/soleus (LGS) nerve (*horizontal bar*, 1000 ms duration; 1.8×*T*, 200 Hz) prolonged the MG EMG burst and delayed the onset of the St burst for the duration of the stimulus train (**A**), and the very next stimulus trial in which the same stimulus produced only a modest effect on MG burst duration (**B**). **C** Rectified and filtered mean MG EMG (n = 12) showing the increase in amplitude that occurred following stimulation of the LGS nerve at 1.8×*T*. The *heavy lines* and *light lines* refer to the stimulated and unstimulated trials, respectively. The *dotted line* indicates the onset of the stimulus

One animal compensated for large increases in ipsilateral stance duration by making a quick double step of the contralateral leg. This type of compensation was only observed during bipedal walking. A more common observation was for the animal s cadence to be slowed as a result of an increase in the ipsilateral and contralateral stance phases. Another behavior employed by conscious animals was to simply stop walking following a presentation of a stimulus that strongly increased the cycle period. Presumably, the animals stopped walking so that they could maintain their balance following the perturbation.

During walking, LGS stimulus trains increased the amplitude as well as the duration of the MG bursts (Figs. 1C, 3A). Although this increase in the amplitude of the MG EMG occurred in all animals, it was only significantly increased in three animals. The latency for the increase in

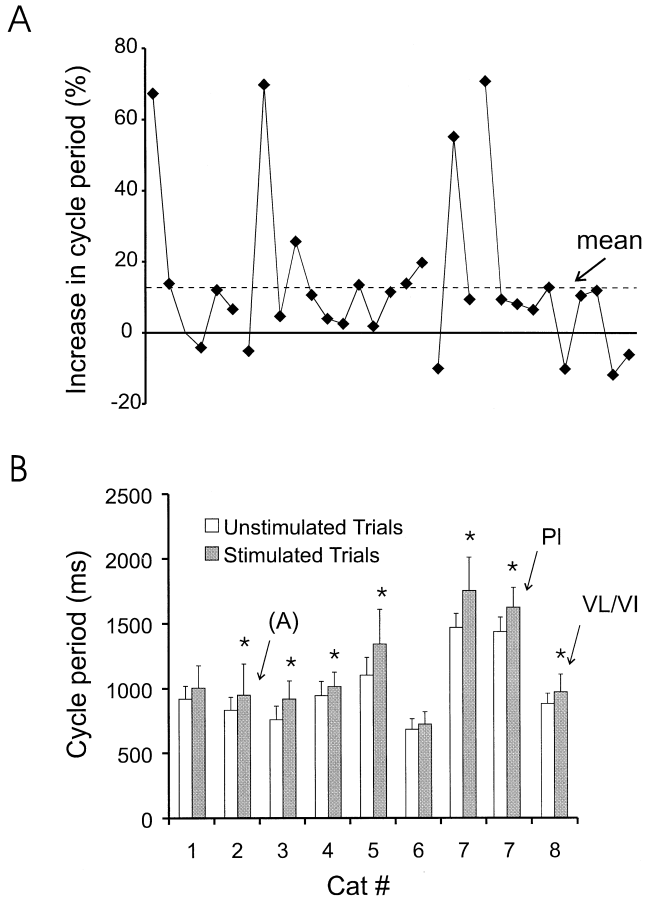

Fig. 2A, B Stimulation of extensor group I afferents has a modest effect on cycle period during quadrupedal walking. **A** The effects of stimulating the LGS nerve [1000 ms duration, 2×*T*] on the cycle period in one animal (cat 2). The *dotted line* indicates the mean percentage increase in the cycle period. The *breaks in the curve* indicate separate sets of trials. **B** The results from eight quadrupedally walking animals. (**A**) indicates same animal as part A. All animals were walking at 0.35–0.4 m/s, except for animal 7, which was walking at 0.25–0.3 m/s. The nerves stimulated were lateral gastrocnemius/soleus (LGS) only (cats 2–6), LGS and vastus lateralis/vastus intermedius (*VL/VI*; cat 1), VL/VI only (cat 8). In cat 7 we stimulated either the plantaris (*Pl*) or the LGS nerve. An *asterisk* indicates that the data are significantly different ($P<0.05$). Data were obtained 1 day following surgery in cats 5 and 6, 2 days after surgery in cats 3 and 7, and 3 days after surgery in cats 1, 2, 4, and 8

amplitude was between 7 and 10 ms. These increases in amplitude were reflected in the kinematics of the hindlimb (Fig. 3B, C; data from a bipedally walking animal). Stimulation of the group I afferents in the LGS nerve could eliminate the yield of the ankle and knee that occurred during midstance when the contralateral hindlimb entered its swing phase (Fig. 3C). This yield resulted in a decrease in the vertical positions of the joints, which was visually apparent in a marked sag of the hindquarters during stance (Fig. 3B, left stick figure). Increases in the amplitude of the VL burst did not occur in any of the animals tested. In two animals, stimulation of the PL nerve did not result in an increase in the amplitude of the MG EMG (data not shown).

In two animals, stimulation of extensor group I afferentst during early flexion (LGS and Pl nerves in one cat; LGS nerve in the one other animal; trains, 300 ms duration; 200 Hz, 1.8×*T*, triggered 0–50 ms from the beginning of either the TA or St bursts during bipedal walking) produced an earlier onset of the next extensor burst (Fig. 4). Overall, there was a mean reduction in the duration of the ipsilateral step cycle as a result of extensor group I stimulation during flexion ($n = 2$; 9 ± 10% SD and 8 ± 10% SD; $P<0.05$]. In both animals the amplitude of the flexor burst during extensor nerve stimulation was not noticeably decreased compared with control trials.

Comparison between bipedal and quadrupedal walking

Visual observation of stepping animals indicated that the effect of stimulating LGS group I afferents on the cycle period was dependent on the behavior of the animal. For example, when an animal was approaching its food dish, looking around, or when it was walking irregularly, the effect appeared to be stronger. These effects were impossible to quantify, but they did suggest that the effectiveness of the stimulus was related to the behavioral state of the animal. To examine this idea, a detailed comparison of stimulating the LGS nerve in the same animal during bipedal and quadrupedal walking was made. The comparisons were made during the same recording session in six animals stepping at the same rate in the two situations. During periods of bipedal walking, the effects of stimulating the LGS nerve on the cycle period were more pronounced than those observed when the animal was walking quadrupedally. In Fig. 5A the trial by trial effect of the LGS stimulus on increasing the cycle period in one animal during quadrupedal or bipedal walking is plotted, while Fig. 5B shows a summary of data from all six animals comparing the effects of stimulating LGS (cats 2–6) and LGS plus VL/VI (cat 1) (trains, 1000 ms duration, 1.8–2×*T*) during bipedal or quadrupedal stepping. During bipedal walking, the percentage of trials in which the stimulus train increased the cycle period by a value greater than the sum of the control cycle period and its associated standard deviation was increased in five out of six animals. When the mean data for each animal were pooled, a significant difference was found to exist between the bipedal and the quadrupedal stimulus trials (bipedal walking trials, mean increase in cycle period 18 ± 10% SD; quadrupedal walking trials, mean increase in cycle period 9 ± 6% SD; $P<0.05$).

A decline in LGS effectiveness occurred over time

The effects of stimulating the group I afferents in the LGS nerve on cycle period progressively decreased from one day to the next (Fig. 6). In one animal the ability of LGS stimulation to prolong the duration of the cycle period gradually decreased over a period of 5 days, with the initial recording being made 8 h after the implantation of

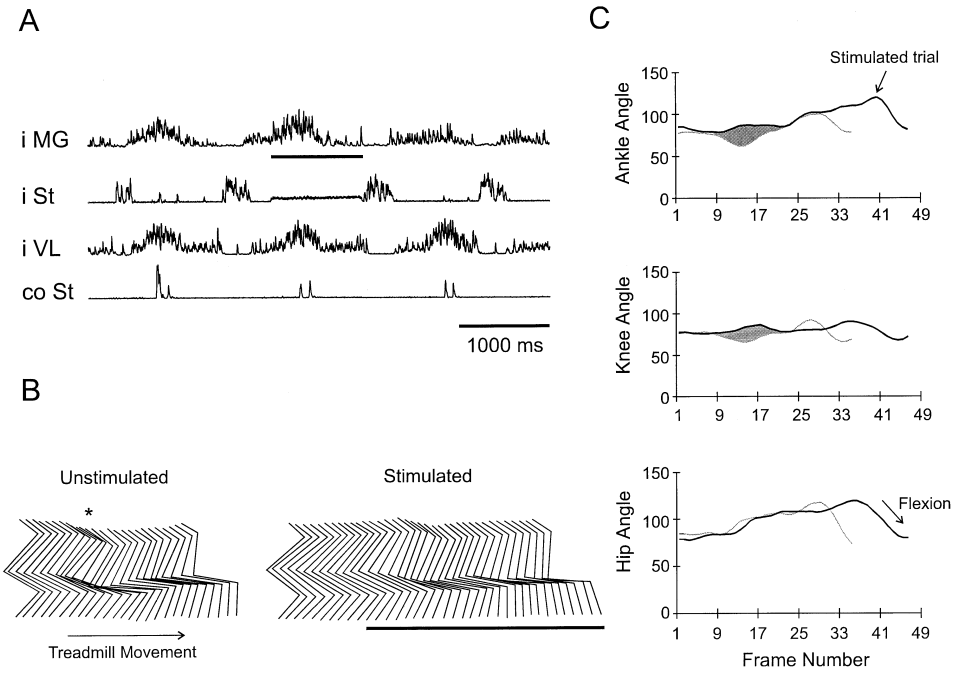

Fig. 3A–C Stimulation of the LGS group I afferents during extension increases the magnitude of activity in MG and abolishes the yield at the ankle and knee. Cat 5, 1 day after surgery. **A** Rectified and filtered EMGs from hind leg muscles during bipedal walking. The stimulus trains (*horizontal bar*, 1000 ms duration, 200 Hz) were triggered approximately 200 ms after the onset of iMG activity. Note the increase in the iMG amplitude on this trial and that the iSt burst was delayed for the duration of the stimulus train. **B** Stick figures showing leg movements during the first two extensor bursts in **A**. The *heavy line* on the *right* indicates when the stimulus train was on. Note that in the unstimulated trial (*left*) there was a pronounced yield of the knee and ankle and a downward movement of the hindquarters during midstance (*asterisk*). The yielding at the ankle and knee were greater than normal due to axotomy of the LGS nerve. The yield of the ankle and the knee were eliminated during LGS stimulation. **C** Plots of the ankle, knee, and hip angles taken from the stick figures shown in **B**, illustrating the abolition of yield in the ankle and the knee with LGS nerve stimulation (*thick line*, stimulation during stance; *thin line*, no stimulation during stance). The *frame number on the abscissa* indicates the video frame for each line in the stick figures in **B** (*co St* contralateral semitendinosus)

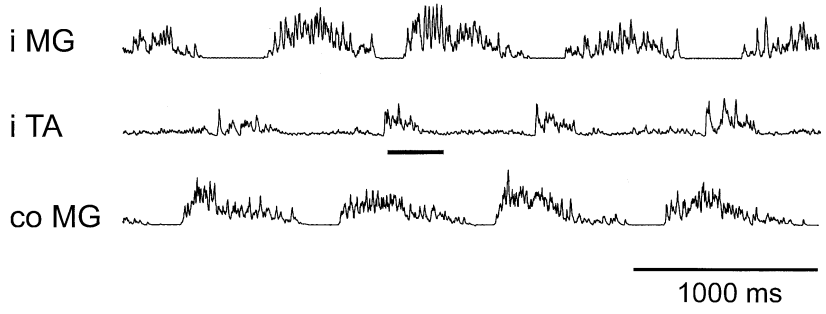

Fig. 4 Stimulation of the LGS nerve during flexion causes an earlier onset of the ipsilateral extensor burst. Rectified and filtered EMG traces in an conscious cat walking bipedally showing that the stimulus train (*horizontal bar*, 300-ms trains, 200 Hz, 2×*T*, triggered 20 ms after the onset of TA bursts) shifted forward the onset of the ipsilateral MG burst (indicated by the shorter period of silence between bursts) and decreased the cycle period. Note that the stimulus train did not affect the Tibialis anterior burst (*i TA* ipsilateral tibialis anterior)

the nerve cuff (Fig. 6). One possible explanation for this progressive reduction in the effects of stimulating the LGS nerve was that the implanted stimulus cuff was damaging the group I afferents. One observation that suggests that the stimulus cuff did not damage the group I afferents in the LGS nerve was that heteronymous H-reflexes from the MG muscle could be recorded in all of the animals tested over a period of days (stimulus parameters, 1-ms pulse duration, 2 Hz). If group I afferents were selectively damaged, the threshold for eliciting an H-reflex should have risen. This did not occur (data not shown).

Decerebration increases the effectiveness of LGS group I afferents

Since the effect of stimulating the LGS group I afferents on the cycle period declined over time in the conscious cat, this could account for the differences between the

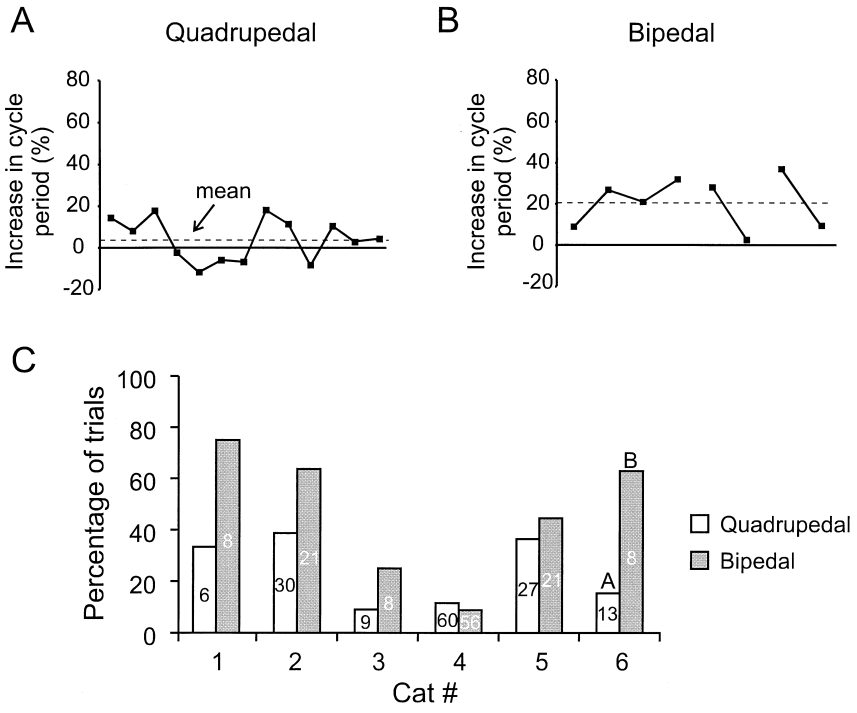

Fig. 5A–C Stimulation of group I afferents in the LGS nerve has a greater effect on the cycle period when the animals were walking bipedally compared with quadrupedally. **A, B** The effects of the individual LGS stimulus trials (trains, 1000 ms duration, 2×*T*) on the cycle period during quadrupedal (**A**) and bipedal (**B**) walking in one animal (cat 6). The *dotted lines* indicate the mean percentage increase in the cycle period. The *breaks in the curve* in **B** indicate separate sets of trials. **C** The percentage of the trials that exceeded a value that was equal to the sum of the mean control trials plus the associated SD for all six animals that walked both quadrupedally (*white bars*) and bipedally (*gray bars*) at the same rate. The stimulus trains (1000 ms duration) commenced approximately 200 ms after the onset of extensor activity in all trials (number of trials indicated by *numbers in bars*). In cat 1 the VL/VI nerve was stimulated along with the LGS nerve. Data were obtained 3 days after surgery in cats 1–4, 2 days after surgery in cat 5, and 1 day after surgery in cat 6. *A* and *B* for cat 6 indicate data corresponding to parts **A** and **B**. The mean cycle periods prior to the stimulated trial for bipedal and quadrupedal walking were: cat 1, 911 and 918 ms; cat 2, 901 ms and 831 ms; cat 3, 912 ms and 877 ms; cat 4, 937 ms and 945 ms; cat 5, 1149 ms and 1020 ms; cat 6, 735 ms and 738 ms

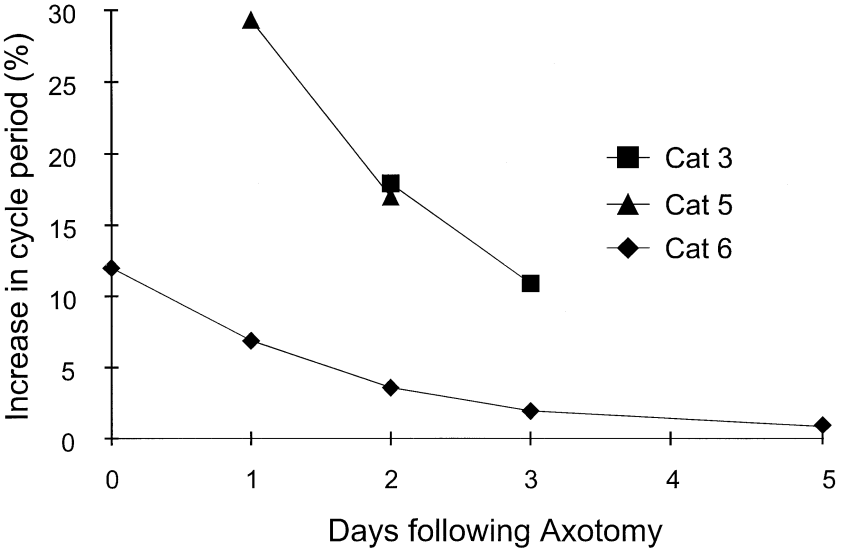

Fig. 6 The influence of group I afferents in the LGS nerve on the cycle period decreases with time after axotomy of the LGS nerve. The mean effects of stimulating the LGS nerve (trains, 1000 ms, 1.8×*T*, 200 Hz, triggered 200 ms after onset of MG bursts) in three conscious animals that were tested during bipedal walking for more than 1 day after cutting the LGS nerve

conscious and decerebrate animal. To test for this possibility, a direct comparison was made in four animals by first stimulating the LGS nerve at group I strengths while the animals were conscious and 3–4 h later after they had been decerebrated. In all animals the percentage of stimulus trials greater than the sum of the control cycle period and its associated standard deviation increased during decerebrate stepping (range of increase 25–55%; Fig. 7). To estimate the extent that axotomy reduced the influence of LGS group I afferents on prolonging the cycle period, we

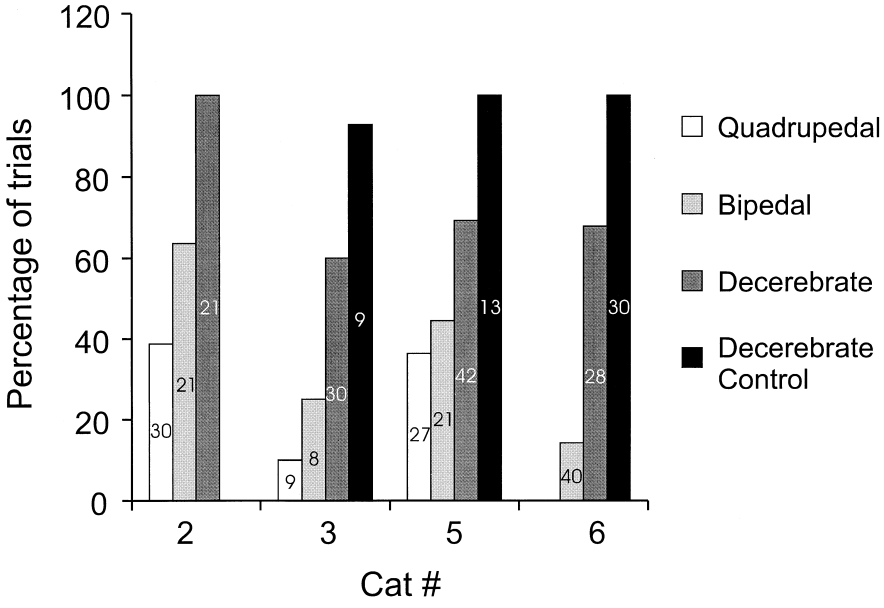

Fig. 7 Stimulation of group I afferents in the LGS nerve was more effective in increasing the cycle period during decerebrate walking. Comparison of the effects of LGS stimulation during quadrupedal, bipedal, and decerebrate walking. In three of these animals, the effects of stimulation of the acutely sectioned LGS nerve in the contralateral leg after decerebration were also recorded (*black bars*). In all four animals, stimulation of the LGS nerve while the animal was conscious resulted in a lower percentage of trials exceeding the sum of the mean control trials plus its associated SD. During decerebrate walking, stimulation of the LGS nerve in the contralateral leg (*black bars*) resulted in a greater percentage of trials exceeding the sum of the control trials plus the SD than for the ipsilateral leg. Data for cats 2, 3, 5, and 6 were obtained 3, 3, 2, and 7 days following LGS nerve section, respectively. *Numbers in bars* indicate number of trials

compared the effects of stimulating the chronically sectioned nerve with the effects of stimulating the acutely sectioned LGS nerve in the opposite hindlimb in three of the four decerebrate cats. In all of these animals, stimulation of the chronically sectioned LGS nerve was less effective in prolonging the cycle period (Fig. 7). Stimulation of the acutely sectioned nerve generally caused close to 100% (range 92–100%) of the stimulus trials to exceed the sum of the control step cycle period plus its associated standard deviation, whereas equivalent stimulation of the chronically sectioned LGS nerve caused 66% of the stimulus trials to increase the cycle period by an amount exceeding the standard deviation of the control cycle period.

In one of the four animals (cat 6), 7 days elapsed before decerebration took place. In the conscious state only a small percentage of LGS stimulus trials had an effect on the cycle period. However, following decerebration, stimulation of the same LGS nerve caused over 60% of the stimulus trials to exceed the control period plus its associated standard deviation (Fig. 7). This percentage was 100% when the acutely cut contralateral LGS nerve was stimulated in the decerebrate state (in this case extensor bursts were prolonged for the duration of the stimulus train). Thus, input from the chronically cut LGS nerve became more effective following decerebration, but not as effective as input from an acutely sectioned LGS nerve. This animal was of particular interest, since data were collected in the conscious state on the same day as the stimulating cuff was implanted. Stimulation of the LGS nerve during bipedal walking 8 h after implanting the cuff increased the cycle period by 11–23%, which was considerably less that the effect of stimulating the contralateral LGS in the decerebrate state (77% increase in the cycle period). The latter observation was made about 3 h after cutting the nerve.

Discussion

The main result of this study was that stimulation of group I extensor afferents can prolong extensor activity and delay the onset of flexion in the conscious walking cat. In addition, stimulation of the LGS group I afferents often caused a large increase in the amplitude of the MG EMG. Although these results were qualitatively similar to previous investigations that have used decerebrate stepping (Whelan et al. 1995a, b) or fictive locomoting (Conway et al. 1987; Guertin et al. 1995) preparations, there were a number of important differences. The effects on the duration of the extensor bursts were not as large as in decerebrate walking cats. In these preparations, stimulation of the LGS group I afferents could powerfully extend the leg for the duration of the stimulus train (Fig. 7; Whelan et al. 1995a), a phenomenon that was rarely observed in the conscious animal. Another difference was observed when extensor group I afferents were stimulated during early flexion. Rather than inhibiting the flexor burst and resetting the rhythm to extension, as has been reported in reduced preparations (Conway et al. 1987; Guertin et al. 1995; Whelan et al. 1995a), the initiation of the extensor burst was advanced with little effect on the amplitude of the flexor burst (Fig. 4). Finally, stimulation of the group I extensor afferents produced variable

effects on the duration of extension. For example, stimulus trains could prolong extension for the duration of the train in some trials, yet the next trial could have a modest effect (Figs. 1, 2).

Variability in the effectiveness of group I extensor afferents on increasing the cycle period

Stimulation of extensor group I afferents produced variable effects on the duration of step cycle in the conscious cat (Figs. 1, 2, 5). In some animals the effectiveness of LGS stimulation on increasing the cycle period appeared to decrease following repeated presentations of the stimulus (Fig. 2A). One possible explanation for this variability, and the generally weak effects of extensor nerve stimulation in conscious animals, is the artificial nature of the electrical stimulus to the extensor nerves. Synchronous activation of all the group I afferents in a single nerve that occurs during electrical stimulation is unlikely to occur naturally. In this regard, Johansson and Westling (1987) have demonstrated that humans adapt rapidly to the electrical stimulation of cutaneous afferents that normally regulate precision grip but never adapt to normal tactile stimuli.

A wide range of sensory receptors presumably signals natural perturbations and it is the overall pattern of activity in multiple receptors that is important in controlling stance duration and other aspects of the step cycle. Evidence from the decerebrate locomoting cat shows that group Ia and II input from flexor muscles (Hiebert et al. 1996; Perreault et al. 1995) as well as cutaneous input (Duysens and Stein 1978; Guertin et al. 1995) can help shape the timing of the step cycle. Thus it is conceivable that more robust effects could be produced if multiple groups of afferents were stimulated with a pattern that more accurately mimics normal sensory feedback during stepping movements.

The weaker effects of stimulating the LGS group I afferents in conscious animals are only partly due to axotomy

In the animals in which recordings were made on successive days there was a progressive reduction in the ability of LGS group I stimulation to prolong the cycle period (Fig. 6). The reduced effectiveness of LGS nerve stimulation in conscious animals compared with decerebrate animals may have been due to axotomy of the nerve (Whelan et al. 1995b). In most conscious animals data were collected 2–3 days after cutting the LGS nerve. To examine this possibility, the effects of LGS nerve stimulation in conscious cats and in the same animals a few hours later following decerebration were compared (Fig. 7). If axotomy of the nerve was the sole reason for the reduced effects in the conscious cat, then stimulation of the extensor group I afferents should have similar effects in both preparations. This was not the case. The effects of LGS stimulation were greater in the decerebrate state, suggesting that part of the difference must be dependent on the preparation.

Although the effects of stimulating the LGS group I afferents increased following decerebration, the effects were still less than stimulation of an acutely sectioned LGS nerve in the contralateral leg (Fig. 7). Hence chronic axotomy is partially responsible for the weaker effects in the conscious animal.

The effects of stimulating extensor group I afferents depended on the locomotor behavior

Stimulation of the LGS group I afferents tended to prolong the step cycle for longer periods during bipedal than during quadrupedal walking (Fig. 5). There are two possible explanations for this difference. First, locomotor activity in the forelimbs may decrease the effectiveness of extensor group I feedback. Support for this possibility comes from observations (Whelan 1996b) of decerebrate animals. In these animals stepping of the hind legs can often occur without stepping of the forelegs. When this occurs, the effectiveness of stimulating extensor nerves on extensor burst duration can increase. Cruse and Warnecke (1992) have shown that in conscious animals movement of the forelimbs can affect the timing of locomotor activity in the hindlimbs, suggesting that propriospinal input from the forelimbs influences the hind leg pattern generating network. If this occurs it may reduce the ability of inputs from hind leg proprioceptors to influence the timing of the step cycle.

A second possibility is that the effectiveness of group I stimulation during walking in conscious animals is task-dependent. Task-dependent modulation of afferent pathways has been described in humans, other vertebrates, and invertebrates (Marsden et al. 1981; Matthews 1991; Nashner 1976; Pearson 1993; Prochazka 1989). This modulation ensures that afferent regulation of motor programs is appropriate for different behaviors. In conscious locomoting cats, few studies have concentrated on task-dependent modulation of specific reflex pathways. However, since descending pathways are well known to be able to adjust the strength of spinal reflex pathways in nonlocomoting preparations (Baldissera et al. 1981), it is likely that descending signals from the motor cortex and the brainstem can also alter reflex pathways in a task-dependent manner during locomotion.

Because bipedal walking is a simpler task than quadrupedal walking, and presumably relies far less on descending supraspinal signals, the relative importance of segmental proprioceptive signals in regulating stepping is likely to be greater during bipedal stepping. It would be some interest to determine whether the influence of the extensor group I afferents on the timing of locomotor activity is altered when the animal is walking quadrupedally in different situations. When an animal is walking normally, multiple afferent systems are probably involved in controlling stance duration, and the relative importance

of each system may change depending on the behavioral state of the animal.

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