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Contribution of Force Feedback to Ankle Extensor Activity in Decerebrate Walking Cats

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Donelan, J. M. and K. G. Pearson. Contribution of force feedback to ankle extensor activity in decerebrate walking cats. *J Neurophysiol* 92: 2093–2104, 2004; 10.1152/jn.00325.2004. Previous investigations have demonstrated that feedback from ankle extensor group Ib afferents, arising from force-sensitive Golgi tendon organs, contributes to ankle extensor activity during the stance phase of walking in the cat. The objective of this investigation was to gain insight into the magnitude of this contribution by determining the loop gain of the positive force feedback pathway. Loop gain is the relative contribution of force feedback to total muscle activity and force. In decerebrate cats, the isolated medial gastrocnemius muscle (MG) was held at different lengths during sequences of rhythmic contractions associated with walking in the other three legs. We found that MG muscle activity and force increased at longer muscle lengths. A number of observations indicated that this length dependence was not due to feedback from muscle spindles. In particular, activity in group Ia afferents was insensitive to changes in muscle length during the MG bursts, and electrical stimulation of group II afferents had no influence on the magnitude of burst activity in other ankle extensors. We concluded that the homonymous positive force feedback pathway was isolated from other afferent pathways, allowing the use of a simple model of the neuromuscular system to estimate the pathway loop gain. This gain ranged from 0.2 at short muscle lengths to 0.5 at longer muscle lengths, demonstrating that force feedback was of modest importance at short muscle lengths, accounting for 20% of total activity and force, and of substantial importance at long muscle lengths, accounting for 50%. This length dependence was due to the intrinsic force-length property of muscle. The gain of the pathway that converts muscle force to motoneuron depolarization was independent of length. We discuss the relevance of this conclusion to the generation of ankle extensor activity in intact walking cats. These findings emphasize the general importance of feedback in generating ankle extensor activity during walking in the cat.

INTRODUCTION

Muscle proprioceptors, receptors that sense the biomechanical state of muscles, are an essential source of feedback during walking. One important function of these receptors is to control the *timing* of muscle activity. In cats, for example, sensory feedback from hip flexor and ankle extensor muscles controls the timing of a limb's transition from stance to swing, and stepping of a hind leg can be stopped altogether by preventing leg extension or electrically stimulating group I afferents from the ankle extensor muscles (Grillner and Rossignol 1978; Hiebert et al. 1996; Whelan et al. 1995). Sensory feedback is also involved in regulating the *magnitude* of muscle activity during the stance phase of walking. A clear example of this influence in intact, decerebrate, and spinal cats is the large reduction in the magnitude of ankle extensor activity when a hind leg unexpectedly steps into a hole (Gorassini et al. 1994; Hiebert and Pearson 1999; Hiebert et al. 1994). This reduction in activity is most likely due to the loss of feedback from proprioceptors in the ankle extensor muscles because loading the muscles as the foot enters the hole can restore muscle activity to normal values (Hiebert and Pearson 1999). In addition, applying length changes that mimic those occurring in walking to the isolated ankle extensor muscles in decerebrate walking cats increases the average muscle force by ~30% (Stein et al. 2000). Similarly, in spinal cats, feedback related to length change accounts for about one-quarter of the ongoing force in ankle extensor muscles (Bennett et al. 1996). In humans, there is also strong evidence that sensory feedback from the ankle extensor muscles contributes substantially to the activation of the soleus muscle during stance. Sinkjaer et al. (2000) have reported that an imposed rapid shortening of the ankle extensors during the stance phase produced a 50% reduction in soleus muscle activity, thus indicating that sensory feedback contributes at least half of the suprathreshold input to soleus motoneurons. This measure is consistent with a previous estimate derived from the analysis of reflex responses evoked in soleus by an imposed rapid lengthening of the ankle extensors early in the stance phase (Yang et al. 1991). Collectively, these studies clearly demonstrate that proprioceptive feedback has an important contribution to generating ongoing ankle extensor activity.

Given the substantial role of afferent feedback to the generation of ankle extensor activity, it is important to identify the individual contributions from each proprioceptive pathway. There is a growing body of data suggesting that group Ib afferents, arising from force-sensitive Golgi tendon organs (GTOs), contribute substantially to ongoing muscle activity. The most direct demonstration of an excitatory action of group Ib afferents is that electrical stimulation at an intensity that recruits both group Ia (from primary muscle spindle endings) and group Ib afferents has a larger excitatory action on ankle extensor activity than activation by vibration of the group Ia afferents alone (Pearson and Collins 1993). Guertin et al. (1995) have also reported that stimulation of ankle extensor nerves at group I strength produces a widespread increase in ipsilateral extensor activity that cannot be attributed solely to activation of group Ia afferents. Intracellular recordings indicate that this excitatory action is mediated in part by a disynaptic excitatory pathway that is open only during the extensor phase of the locomotor cycle (Angel et al. 1996; McCrea et al. 1995). Another pathway may involve interneurons in the cen-

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tral-pattern-generating network (Gossard et al. 1994). Ensemble averaging of activity in group Ib afferents from ankle extensors in normal walking cats shows that these afferents have a high level of activity during the stance phase and a strong covariation with ongoing muscle activity (Prochazka and Gorassini 1998). In humans, there is also a suggestion that feedback from Golgi tendon organs contributes to the activation of the soleus muscle. Ischemic block on the thigh to reduce feedback from group Ia afferents, injection of lidocaine into the common peroneal nerve to reduce reciprocal inhibition and ingestion of tizanidine to selectively depress transmission in group II pathways, all had little effect on the reduction in soleus activity produced by forcibly extending the ankle (Sinkjaer et al. 2000). In support of these empirical findings, modeling studies indicate that positive force feedback is an effective method for controlling limb muscles to provide load compensation during walking (Prochazka et al. 1997) and for sustaining rhythmic movements (Geyer et al. 2003). While the collective insight gained from these studies suggests that positive force feedback via group Ib pathways may play an important role in the modulation of ongoing ankle extensor activity, the contribution of this pathway, relative to that of other contributors to motoneuron activity, is not yet known.

Our objective in the present study was to gain insight into the relative importance of force feedback from GTOs to ongoing ankle extensor activity during walking in the cat. To meet this objective, we used a preparation that allowed us to isolate the influences of feedback from homonymous GTOs on medial gastrocnemius (MG) muscle activity in decerebrate walking cats. We then determined the loop gain of this force feedback pathway using a simple model of the neuromuscular system and the measured relationship between MG activity and force. From the loop gain estimates, we calculated the fractional contribution of force feedback to MG muscle activity in our preparation. Combining our measures of loop gain with knowledge of ankle extensor length in intact walking cats (Carlson-Kuhta et al. 1998; Goslow et al. 1973) provided insight in to the normal contribution of positive force feedback to ankle extensor activity.

METHODS

Procedures common to all experiments

All procedures were approved by the University of Alberta Health Sciences Animal Policy and Welfare Committee. A total of 22 adult cats weighing between 2.5 and 5 kg were anesthetized with halothane. A tracheal cannula was then inserted to deliver anesthetic throughout the surgical procedures. Both carotid arteries were ligated, and a cannula was inserted into one artery to monitor blood pressure. To administer fluids and drugs, a cannula was placed in one jugular vein. The iliac crests were exposed, and a sturdy steel wire was inserted through each crest to support the hindquarters during walking. After further dissection (as described in subsequent sections), the animal was transferred to a stereotaxic frame mounted over a treadmill and decerebrated. The level of the decerebration cut was anterior to the edge of the superior colliculus at a 50° angle to the horizontal. If the blood pressure dropped <60 mmHg, we administered a bolus (2–5 ml) of 5% dextrose solution volume expander (Dextran). Animals typically began walking spontaneously within 1 h. Otherwise, we induced walking by stimulating the mesencephalic locomotion region [MLR; 15 Hz, 0.5-ms duration, 100–200 μA; coordinates P2, L4, H6; (Shik et al. 1966)]. No consistent differences were observed in the responses of animals walking spontaneously and animals walking in response to MLR stimulation.

Influence of feedback from the MG muscle

This procedure determined the effect of MG muscle-tendon length on its own activity and the activity of other hind leg muscles during rhythmic contractions of the isometric MG muscle-tendon unit. In all 10 animals, the following nerves were transected in the right hind leg during the initial surgical procedure: obturator, sartorius, hamstrings, sural, common peroneal and distal tibial. In 6 of the 10 animals, the femoral, lateral gastrocnemius/soleus and plantaris nerves were also transected leaving only the MG and proximal hip muscles innervated. Bipolar electrodes (Cooner wire AS632) were implanted into the iliopsoas (IP) muscle to record electromyographic (EMG) activity during the swing phase of stepping. After transferring the animal to the stereotaxic frame, EMG electrodes were inserted into the MG muscle in all preparations, and if left innervated, into the vastus lateralis (VL), lateral gastrocnemius (LG), and soleus (SOL) muscles.

Before transferring to the stereotaxic frame, we exposed the ankle extensors. The length of the MG muscle-tendon unit was noted when the ankle and knee were held at 90° by suturing a small thread through the MG distal tendon level with a pin inserted into the tibia. We referenced the length of the muscle-tendon unit to this length, referred to as 0 mm, throughout the experiment. Next, the LG, SOL, and plantaris tendons were cut close to their insertion on the calcaneus, and these muscles were separated from MG and each other. A hole was drilled in the calcaneus and a thick steel wire was inserted into the hole. After transferring to the stereotaxic device, the leg was fixed rigidly to the supporting frame with the hip, knee, and ankle joint angles set to 90° (Fig. 1*A*). A fragment of the calcaneus containing the wire and the insertion of the MG tendon was removed from the remaining bone and connected to a servo-controlled motor (Model JR12M4CH, PMI Motion Technologies) via a custom-built force transducer. The motor was used to hold the MG muscle-tendon unit at constant lengths within the range of –2 to +4 mm. When the three free legs began to step after decerebration, the muscles of the isolated leg generated rhythmic bursts of activity. EMG and force measurements were made during periods of stable stepping while the length of the isolated MG muscle-tendon unit was varied in steps of 2 mm. We

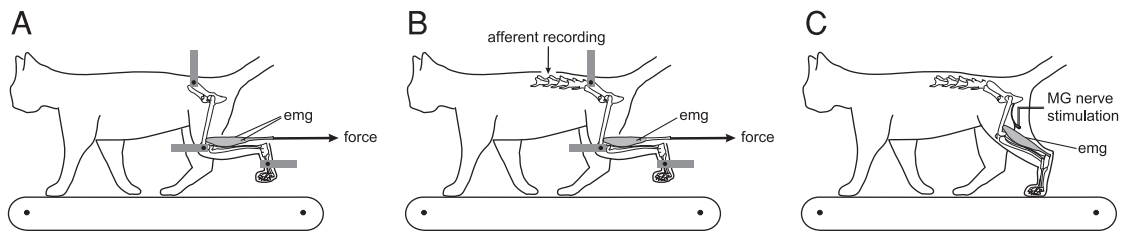

FIG. 1. Illustrations of the experiment procedures used in this investigation. All procedures were performed on decerebrate cats walking on a treadmill. The figures show the arrangement to examine, the influence of proprioceptive feedback from the medial gastrocnemius (MG) muscle (*A*), the activity of afferents from the MG muscle (*B*), and the influence of electrically stimulating afferents in the MG nerve (*C*) (see METHODS for more details).

allowed the muscle to remain at each length for 4–20 stride cycles. The EMGs were amplified and stored with force and length signals on DAT tape (Model VDAT8, Vetron Technology).

After the experiment, we digitized the data at 1.5 kHz with 12-bit resolution (Axotape, Axon Instruments) and stored it on computer disk. The digitized EMG data were rectified and filtered using a low-pass, first-order, one-way, digital Butterworth filter with a cut-off frequency of 30 Hz. The force signals were filtered using a two-way digital filter of the same design. Sequences of consistent stepping lasting ~3 min that included a range of MG lengths were selected for analysis. Restricting each set of analyzed data to a relatively short period of time reduced the possibility of a change in the excitability of the animal, independent of muscle length, confounding our results.

We computed the average EMG and force magnitudes within two regions of MG bursts (Fig. 2). The *early region* was defined as a 30-ms interval beginning with the onset of MG activity. We evaluated this region to study the effect of length on muscle activity in the absence of a significant change in force (Fig. 3). The *middle region* was defined as a 100-ms interval centered on the middle of the MG burst chosen to correspond with high levels of muscle activity and force. To make comparisons of feedback effects across muscles and between animals, we normalized the value of the EMG in each region to each muscle's average middle region value at a length of 0 mm.

Afferent recordings from the MG muscle

This procedure determined the effect of MG muscle-tendon length on the activity of single afferents from the MG muscle during rhythmic contractions of the isometric MG muscle-tendon unit (Fig. 1*B*). Unless otherwise described, this preparation was identical to the previous preparation. The right hind leg and tail were extensively denervated by transecting the following nerves: femoral, obturator, common peroneal, distal tibial proximal to the plantaris nerve, lateral gastrocnemius/soleus, hamstrings, gluteus, and all nerves to the right side of the tail. A stimulating bipolar cuff electrode was placed around the intact MG nerve to identify the afferents. A laminectomy exposed the L_7 and S_1 dorsal roots. After transferring the animal to the stereotaxic frame, small filaments containing single afferents from the MG muscle were dissected from the L_7 and S_1 roots on the right side. Within a paraffin bath, the filaments were lifted onto monopolar silver wire recording electrodes. Afferent type was identified on the basis of conduction velocity, and responses to muscle vibration, ramp-and-hold stretches, and muscle contractions (Matthews 1972). We minimized the number of dissected filaments to maximize the afferent input to the spinal cord from the MG muscle. In the absence of afferent input, rhythmic activity in the MG muscle usually does not occur during walking (unpublished observations). Two to four afferents were isolated in each animal, but some did not remain viable after decerebration and the initiation of walking. Nine animals yielded measurements from 10 Ia afferents, 5 Ib afferents, and 4 group II afferents. When the intact legs began to step and the isolated MG generated rhythmic bursts of activity, the activity in the surviving afferents was recorded at different lengths of the isolated MG muscle-tendon unit. As described in the previous section, afferent activity, EMG and force signals were amplified and stored on tape.

After the experiment, data were digitized at 7 kHz to capture accurately unit activity. Spikes were identified by determining when each unit signal exceeded a set threshold. Data were accumulated into an event histogram (50-ms bin width) relative to the onset of MG burst activity. The average discharge rate (in Hz) for each bin was computed from the number of events in each bin divided by the number of strides and the bin width. We evaluated unit activity over two regions of the stride cycle (Fig. 6). The *prior region* was defined as a 100-ms interval (2 bins) immediately prior to the onset of MG

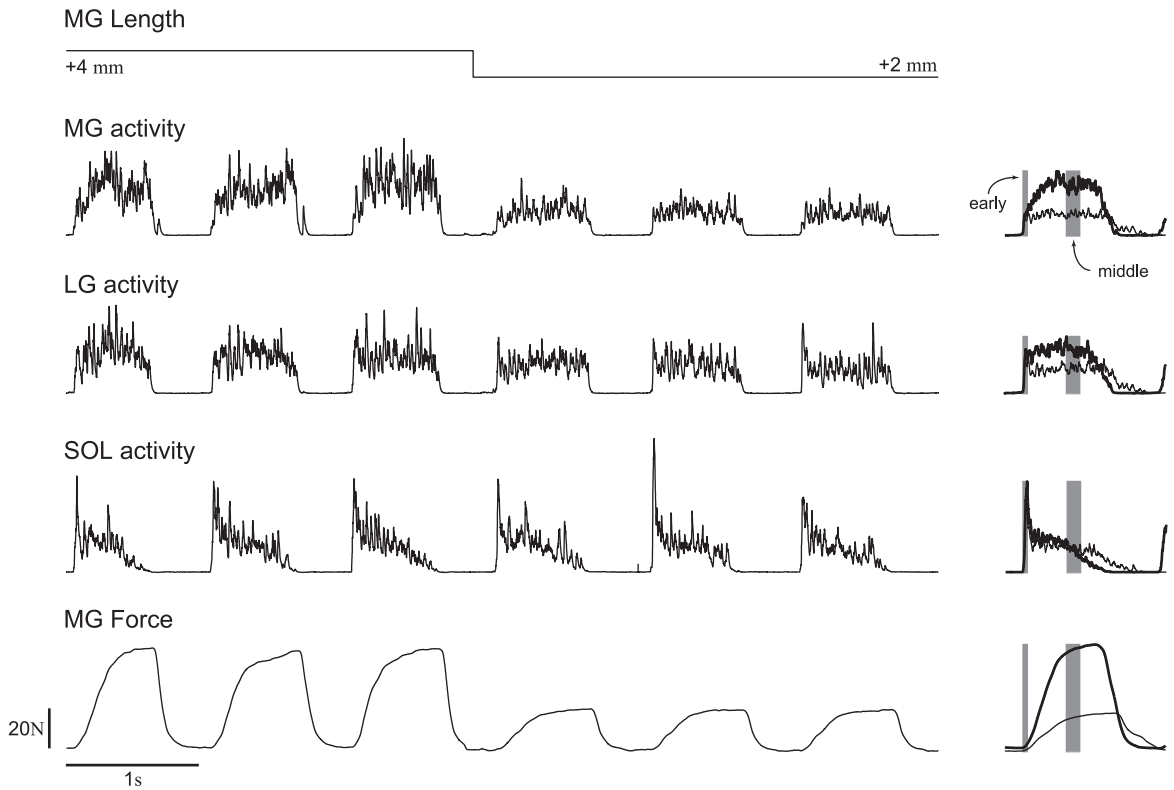

FIG. 2. Longer MG muscle-tendon lengths during a sequence of rhythmic contractions resulted in greater MG and lateral gastrocnemius (LG) muscle activity, and greater MG force. In this example, length did not influence the magnitude of burst activity in the soleus (SOL) muscle. These effects are further illustrated by the averages of 7 stride cycles at each length (*right*). Thick and thin lines, lengths of 4 and 2 mm, respectively; shaded vertical bars, the early and middle regions used in subsequent analyses.

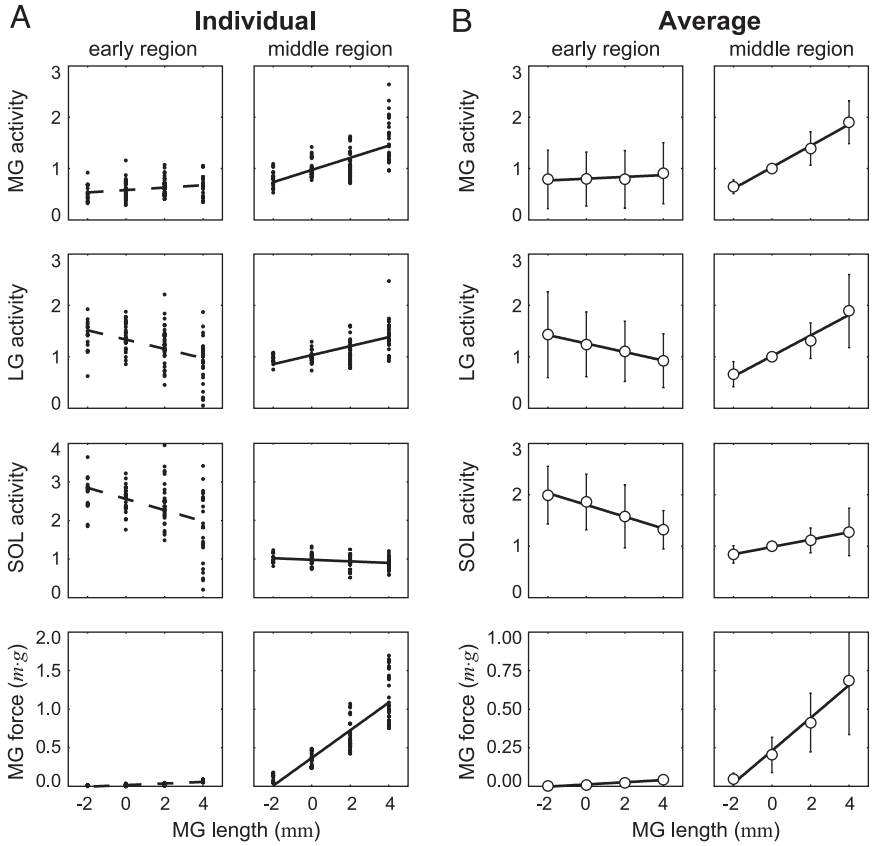

FIG. 3. *A*: representative data illustrating the relationship between the length of the MG muscle-tendon unit and muscle activity and force for a single animal during the early region (*left*) and middle region (*right*) of the MG bursts (see Fig. 2). Each data point is for a single burst, and the lines are best-fit linear regression lines. *B*: the average relationship between MG length and muscle activity and force for 4 animals during the early region (*left*) and middle region (*right*). Symbols represent the average response of 4 animals, error bars indicate the SD, and lines are best-fit linear regression lines.

muscle activity. We evaluated this region to study the effect of muscle length on afferent activity in the absence of a significant change in muscle activity or force. The *middle region* was defined as a 100-ms interval (2 bins) centered on the middle of the burst in MG muscle activity chosen to correspond with the middle region from the previous preparation.

Electrical stimulation of afferents from the MG muscle

This procedure determined the influence of electrically stimulating the group I and II afferents from the MG muscle on the magnitude of burst activity in the LG and SOL muscles. This procedure was performed on three cats. During the initial surgical procedure, the MG nerve of the right hind leg was cut close to the muscle, and the proximal nerve end was tied into a bipolar cuff electrode to stimulate afferents in the MG nerve. A bipolar cuff electrode was also placed on the sciatic nerve to monitor the afferent volleys evoked by stimulation of the MG nerve. Stimulus strength was measure relative to the lowest stimulation intensity (1T) that produced an afferent response (0.2-ms pulse duration, single pulse; Grass model S88 stimulator). This threshold intensity was checked throughout the experiments and small adjustments were made when required. EMG electrodes were implanted in the IP, LG, and SOL muscles of the right hind leg.

During stepping, the MG nerve was stimulated either with trains of stimuli or with single pulses. The trains (typically 300-ms train length, 0.2-ms pulse duration, 100- to 200-Hz pulse frequency) were triggered every third or fourth stride and began 50 ms after the onset of LG activity. The intensity of stimulation was varied between 2T and 5T. The single pulses (typically, 2-3T, 0.2-ms duration) were delivered at a rate of 2 Hz to evoke heteronymous H-reflexes (Misiaszek 2003) distributed over all phases of the stride cycle. Data were recorded to tape for subsequent digitization and analysis.

To examine the evoked nerve potentials, a higher acquisition rate (5 kHz) was used for digitizing the data. During the train stimulation protocol, EMG and force signals were averaged over the final 100 ms of stimulation corresponding approximately to the middle of the extensor bursts. A corresponding section was averaged during the immediately preceding burst. The effect of stimulation at each intensity was measured as the difference between the stimulated burst average and the preceding burst average, normalized to the preceding burst average. For example, a value of 0 would indicate that this stimulation intensity had no effect on EMG, whereas a value of 1 would indicate that the stimulation doubled EMG magnitude. For the H-reflex protocol, we assigned each response to 1 of 10 equally sized bins corresponding to when it occurred during the step cycle and then found the average reflex response within each bin. We defined a stride cycle as beginning with onset of LG activity and ending with the subsequent onset of LG activity.

Estimation of force feedback gain

We used a simple linear model of the neuromuscular system to estimate the contribution of homonymous force feedback to total MG muscle activity and force (Fig. 11*A*). Supported by our empirical results, we assumed that the only afferent pathways responsible for our measured feedback effects during the middle region of the MG bursts were the group Ib pathways arising from force-sensitive GTOs. We also assumed that there was linear summation of the central drive, $e_{c,}$ to the MG motoneurons and the force feedback signal from group Ib afferents, e_f. The sum of the feedforward and feedback contributions yields total motoneuronal/muscle activity, e_t. Total muscle force, f_t, is the product of muscle activity and a parameter, M, related to the intrinsic properties of muscle (termed "muscle gain"). We used the relationship between EMG (a measure of motoneuronal activity) and force during the middle region of the MG contractions to estimate the model parameters for each animal. Because e_t is equal to e_c when the muscle is too short to generate force, we estimated e_c as the y intercept of the best-fit linear regression line for the relationship between total

muscle activity and force (Fig. 11*B*). We then normalized total activity by e_c and total force by body weight, $m \cdot g$. This normalization makes our subsequent calculations independent of size and nonphysiological factors like electrode placement allowing for more meaningful comparisons between animals. This estimate of e_c, accompanied by our measurements of e_t and f_t, allow for the algebraic solution of pathway gains. For each stride, we estimated force feedback gain, *K*, as

$$K = \frac{e_f}{f_t} = \frac{e_t - e_c}{f_t} \quad (1)$$

The muscle gain, *M*, was defined as

$$M = \frac{f_t}{e_t} \quad (2)$$

The dimensionless product of the muscle and force feedback gains, termed "loop gain" (Prochazka et al. 1997), is of particular importance. First, it gives the relative contribution of force feedback to total muscle activity and force

$$\frac{e_f}{e_t} = \frac{f_f}{f_t} = K \cdot M \quad (3)$$

where f_f is the contribution of force feedback to f_t. Second, for steady-state muscle activity and force, the average loop gain in this positive feedback system must be less than unity (Prochazka et al. 1997).

These calculations were performed using the middle region data from our experiments studying the influence of feedback from the MG muscle. At each muscle length, we estimated gains by first calculating *K*, *M*, and $K \cdot M$ for the middle region of each stride and then averaging these values at each length within each animal. Their dependence on MG length was determined using best-fit least-squares linear regression and testing for a slope statistically different from zero.

Statistical analysis

The significance of measured relationships was generally tested using linear regression. In addition to determining the slope and *y* intercept of the line that best fit the data, this procedure yielded a measure of the probability that these coefficients were zero (i.e., *P* value). If the probability was $<5\%$ ($P = 0.05$), we accepted the relationship as significant. To compare the effect of stimulation intensity, rather than use linear regression, we used one-tailed paired *t*-test with the same level of significance.

RESULTS

Influence of feedback from the MG muscle

The purpose of the first set of experiments was to establish the effect of proprioceptive feedback originating from MG on its own muscle activity and on the activity of other muscles during walking in decerebrate cats (Fig. 1*A*). The primary observation was that activity and force in the MG muscle during isometric contractions of the MG muscle-tendon unit was greater at longer lengths. This is illustrated in Fig. 2 by typical muscle activity and force patterns when the length of the MG muscle-tendon unit was reduced from 4 to 2 mm. LG activity was also elevated at longer muscle lengths, whereas the magnitude of bursts in SOL was relatively unaffected. To quantify these changes in EMG magnitude and force at different lengths and in different animals, we averaged these signals over two regions (gray vertical bars; Fig. 2). We choose an early region to study the effect of length on muscle activity before MG developed substantial force. The middle region corresponded with high levels of both muscle activity and force.

Figure 3*A* illustrates the effect of length on the magnitude of the early and middle regions in a representative animal. To make comparisons of feedback effects across muscles and between animals, we normalized the value of the EMG in each region to each muscle's average middle region value at a length of 0 mm. Figure 3*A* demonstrates that although there is variability in burst magnitude and force, consistent effects of length are present. During the early region, LG and SOL activity decreased at longer lengths while MG activity increased slightly ($P = 1.8e\text{-}6$, 6.4e-6 and 0.01, respectively). During the middle region, MG and LG activity increased at longer lengths while SOL activity decreased slightly ($P = 2.1e\text{-}12$, 1.5e-8 and 0.01, respectively). We observed these general patterns in the four animals in which we studied heteronymous effects, and the magnitude did not differ substantially between animals. The notable exception was SOL middle region activity. It increased slightly in two animals, decreased slightly in one animal, and remained relatively unchanged in one animal.

Figure 3*B* plots the average results from the four animals in which we recorded heteronymous influences on LG and SOL. During the early region, before the MG muscle could develop substantial force, MG length did not have a significant effect on MG activity ($P = 0.77$). While LG and SOL activity appeared to decrease at longer muscle lengths, these trends were not statistically significant ($P = 0.24$ and $P = 0.06$, respectively). Middle region results were quite different from early region responses. MG activity and force were significantly elevated at longer muscle lengths ($P = 4.4e\text{-}6$ and $P = 2.5e\text{-}4$, respectively). Comparing changes between -2 and 4 mm, the average increase in MG activity was 203%. We did not study lengths shorter than -2 mm because MG did not generate substantial force at short lengths in this preparation. While LG activity was similarly elevated (194% increase; $P = 4.4e\text{-}4$), SOL activity was less dependent on MG muscle length (50% increase; $P = 0.03$). The difference in response between LG and SOL indicates that proprioceptive feedback originating from MG was directed predominantly to MG and LG.

We also observed that increasing MG length did not have a generalized excitatory action on locomotor activity. Holding the MG muscle at increasingly longer lengths had no effect on the ipsilateral IP muscle activity ($P = 0.12$; Fig. 4), and no obvious changes were noted in the stepping of the contralateral leg and the forelegs. In addition, a doubling of MG burst activity was related to only a 4% increase in VL burst activity ($P = 2.6e\text{-}4$; Fig. 4).

Afferent recordings from the MG muscle

The purpose of this set of experiments was to measure activity in identified muscle afferents to gain insight into which afferent pathways were responsible for the increase in MG muscle activity at longer lengths of the isometric muscle-tendon unit (Fig. 1*B*). Figure 5 illustrates typical activity of a group Ia afferent from a primary spindle ending, a group II afferent from a secondary spindle ending, and a group Ib afferent from a GTO during rhythmic isometric contractions of the MG muscle. The characteristic feature of activity in all

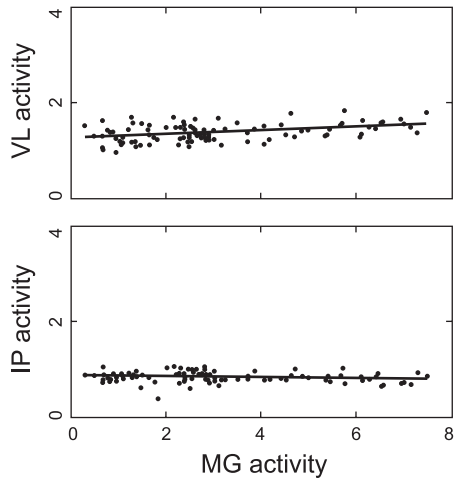

FIG. 4. The magnitude of burst activity in ipsilateral vastus lateralis (VL), a knee extensor, and iliopsoas (IP), a hip flexor, was not substantially influenced by the magnitude of burst activity in MG. IP activity was averaged over a 100-ms interval centered on the middle of its own period of activity while VL was averaged over the previously defined middle region. Each data point is from a single burst. Lines are best-fit linear regression lines.

group Ia and II afferents we recorded was that their activity was reduced during the contractions with a minimum near the middle of the contraction. This effect is likely a result of the extrafusal fibers unloading the intrafusal fibers. In contrast, all group Ib afferents increased their activity during the contractions and were silent between the contractions. Figure 6 illustrates the effect of changing MG length on activity in these representative afferents. While the group II afferents and the Ib afferents showed substantial increases in middle region activity at longer lengths, increasing muscle length had little effect on the Ia afferent activity. Figure 7, *left*, further illustrates this pattern by plotting the effect of MG length on the magnitude of prior and middle region activity in these representative afferents. Although group Ia activity increased with length during the prior region ($P = 6.1e\text{-}3$; Fig. 7*A*), the effect of length on activity in the middle region was not significant ($P = 0.44$; Fig. 7*A*). In contrast, group II activity increased during both the prior and middle regions ($P = 0.03$ and 0.01, respectively; Fig. 7*B*). Group Ib afferent middle region activity also increased with length ($P = 7.6e\text{-}5$; Fig. 7*C*). There was no activity during the prior region because the muscle was not yet generating force. The slopes of the linear regression lines shown in these plots estimate the sensitivity of each afferent to length change within each region. Averaging sensitivities across the measured afferents demonstrates that group Ia afferents were relatively insensitive to length change during the middle region [1.9 ± 1.9 (SD) ΔHz/Δmm; $n = 10$; Fig. 7*A*, *right*]. In contrast, group II afferents (6.4 ± 4.6 ΔHz/Δmm; $n = 4$; Fig. 7*B*, *right*) and Ib afferents (8.8 ± 5.2 ΔHz/Δmm; $n = 5$; Fig. 7*C*, *right*) were, on average, quite sensitive to length change during the middle region.

Electrical stimulation of afferents from the MG muscle

The purpose here was to examine the influence of stimulating afferents in the MG nerve to establish whether group II afferents have the appropriate action to contribute to the elevation of ankle extensor activity occurring at longer MG lengths. The effect of electrical stimulation on the magnitude of bursts in the close synergists LG and SOL was examined using two stimulation intensities; one intensity (2T) was at a level that just begins to recruit group II afferents and the other (5T) was high enough to recruit most group II afferents (Jack

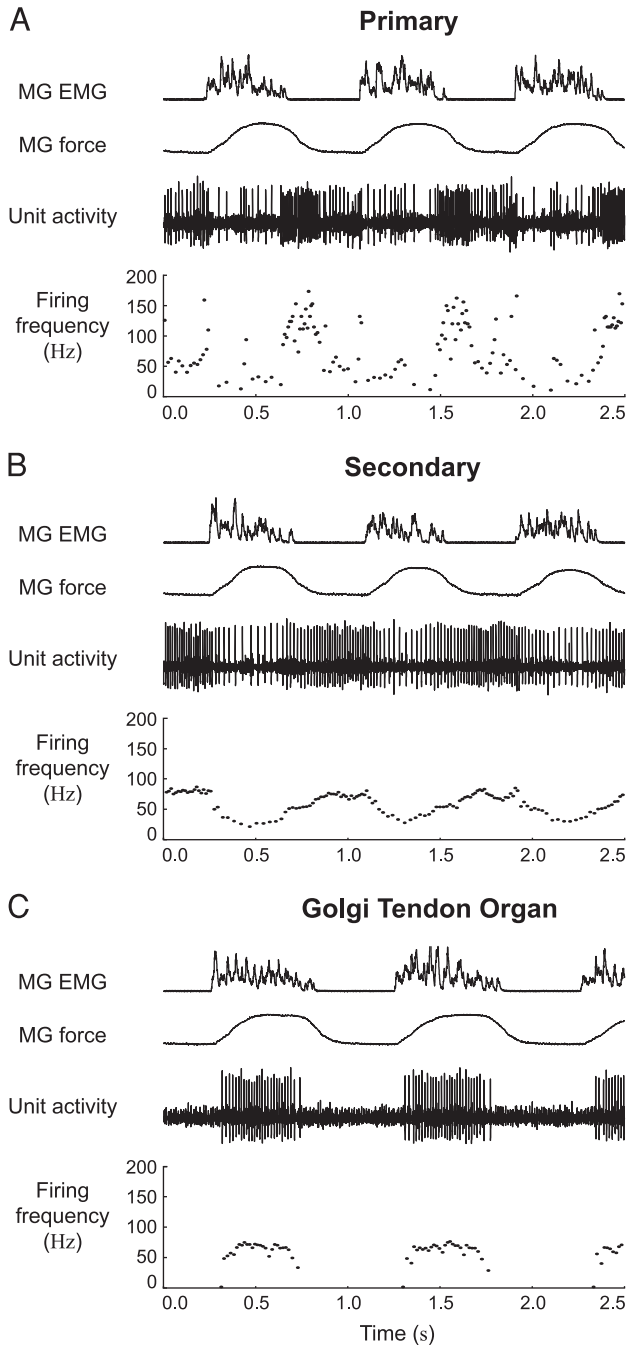

FIG. 5. Representative data illustrating the activity in afferents from a primary spindle ending (*A*), a secondary spindle ending (*B*), and a Golgi tendon organ (GTO; *C*), from the MG muscle and tendon during isometric contractions of the muscle-tendon unit. *Top*: the rectified and filtered electromyograms (EMGs) from the MG muscle. *Middle top*: the forces in the MG muscle. *Middle, bottom*: the spikes recorded from isolated afferents. *Bottom*: instantaneous firing frequency of the afferents. Note the activity in both spindle endings were reduced during the contractions with a minimum near the middle of the contractions while the GTO activity increased during the contraction.

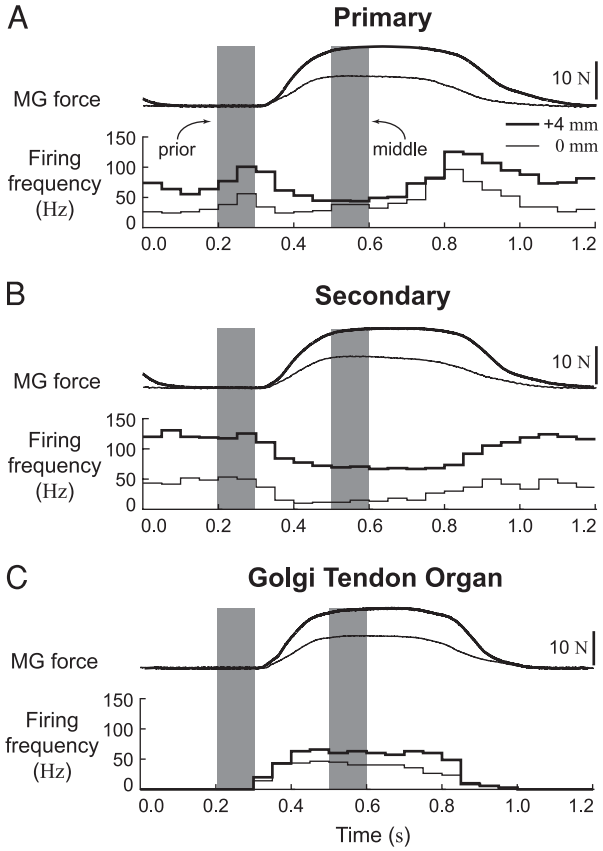

FIG. 6. Histograms illustrating the effect of length on activity in a primary spindle ending (*A*), a secondary spindle ending (*B*), and a GTO (*C*) from the MG muscle during isometric contractions of the muscle-tendon unit at 4 mm (thick traces) and 0 mm (thin traces). Force traces are shown above each set of histograms. Note that during the middle region of the contractions (shaded vertical bar) the activity in the primary spindle ending was only slightly increased at the longer length.

1978). It was necessary in these experiments to cut the MG nerve distal to the stimulating electrodes to prevent contractions of the MG muscle, thus we were not able to examine the homonymous actions of the MG group II afferents.

Figure 8 illustrates the typical effect of MG nerve stimulation on LG and SOL muscle activity, and Fig. 9 presents the average effect for three animals. Comparing the stimulated cycle to the stride immediately prior to stimulation demonstrates that stimulation had a clear excitatory effect on both LG activity ($P = 0.02$) and SOL activity ($P = 3.4\text{e-}4$) at 2T (Fig. 8*B* and 9), thus demonstrating a strong heteronymous action of MG group I afferents. However, stimulating at 5T (Figs. 8*C* and 9) did not further increase activity over the increase observed at 2T for either LG ($P = 0.22$) or SOL ($P = 0.88$). Because this intensity range represents the range over which most of the group II afferents are normally recruited, it appears that transmission in pathways from MG group II afferents to motoneurons innervating other ankle extensor muscles was either very weak or functionally absent.

We also determined whether monosynaptic pathways from group Ia afferents were functional in our preparation. Representative heteronymous H-reflex responses from a single animal illustrates that heteronymous connections from MG group Ia afferents to LG and SOL motoneurons were open (Fig. 10). Measurements in the two other animals yielded qualitatively similar results.

Estimation of force feedback gain

The purpose of this procedure was to estimate the contribution of homonymous force feedback to MG activity and force. The basic assumption of this analysis is that the increase in magnitude of the MG bursts at longer muscle lengths (Figs. 2 and 3) is entirely due to feedback from force-sensitive afferents in the MG muscle. In the DISCUSSION, we justify this assumption based on the empirical results of this investigation. Using a linear model for positive force feedback (Fig. 11*A*) and empirical data for the relationship between the middle region of MG EMG activity and the MG force (Fig. 11*B*), we could estimate the contribution of force feedback to MG activity (see METHODS). The relationship between muscle force and EMG activity was similar in all 10 animals in that the level of EMG activity always increased substantially at longer muscle lengths. We used the equations outlined in METHODS to estimate pathway gains and their dependence on muscle length.

Figure 11*C* plots the estimated force feedback gain, *K* (*Eq. 1*), as a function of muscle length. The magnitude of *K* varied from animal to animal. At 0 mm, for example, *K* ranged from 0.69 to 3.91 and averaged 1.90 ± 0.97 [in units of $e_c \cdot$

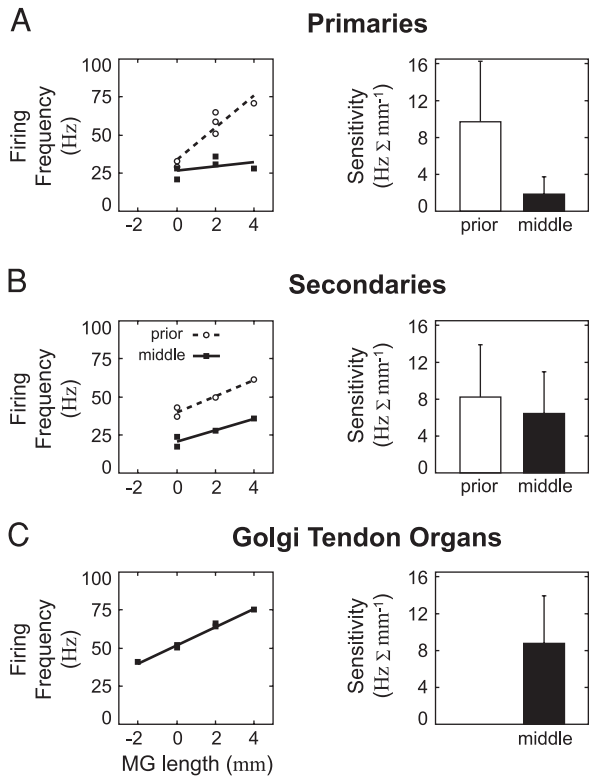

FIG. 7. *Left*: plots illustrating the influence of MG length on the activity of a single primary spindle ending (*A*), secondary spindle endings (*B*), and GTO (*C*) during the prior region (- - -) and middle region (—) of the contractions in the MG muscle. Lines are best-fit linear regression lines. Their slopes estimate the sensitivity of each afferent to length change during each region. *Right*: bar plots illustrating the average sensitivities for all afferents in each group. Error bars denote the SD. Note that primary spindle endings were insensitive to length during the middle region.

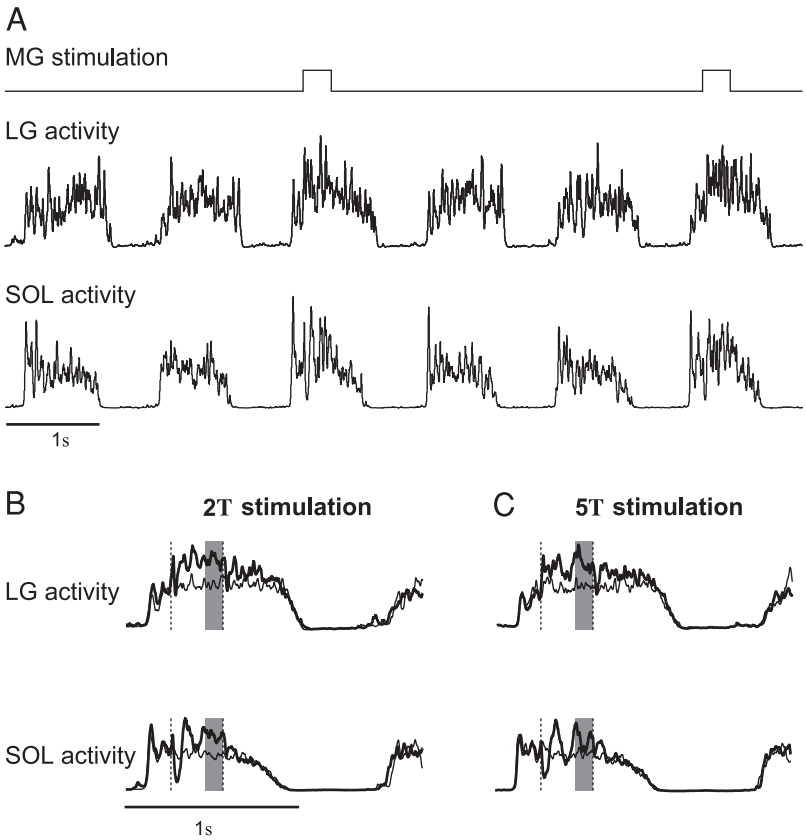

FIG. 8. *A*: representative data illustrates that MG nerve electrical stimulation (2T) during a sequence of stepping resulted in greater LG and SOL muscle activity. We stimulated the nerve every 3rd stride with trains of stimuli, 300 ms in duration (*top*). *B* and *C*: the magnitude of the effect of 2T and 5T stimulation on LG and SOL activity was similar. The thick line and thin lines denote the stimulated stride and the stride immediately prior to stimulation, respectively, averaged for 10 consecutive strides. The dotted vertical lines represent the period of stimulation. The shaded vertical bars denote the regions used in subsequent analyses.

$(m \cdot g)^{-1}$]. Interestingly, K was independent of muscle length in all animals. Linear regression on each animal's data yielded P values that ranged from 0.06 to 0.89 with a median value of 0.47. To determine the average force feedback gain and its dependence on length, we combined our measurements from all animals and found the best-fit linear regression line (Fig. 11*C*). On average, K was also independent of muscle length ($P = 0.39$; slope $= 0.05 \pm 0.12$; y intercept $= 1.72 \pm 0.26$).

Figure 11*D* plots the estimated muscle gain, M (*Eq. 2*), as a function of muscle length. Similar to force feedback gain, M varied from animal to animal. At 0 mm, for example, M ranged from 0.06 to 0.33 and averaged 0.19 ± 0.09 [in units of $m \cdot g \cdot (e_c)^{-1}$]. In contrast to force feedback gain, M generally increased with muscle length. Linear regression on each animal's data yielded P values that ranged from 2.1e-5 to 0.08 with a median value of 5.7e-3. Two animals did not demonstrate significant effects of length on M ($P = 0.08$ and 0.05). While muscle gain in these animals showed a similar dependence on length as the other animals, the relationship was not significant because we were only able to make measurements at three muscle lengths before the animals stopped walking. Combining our measurements from all animals and determining the best-fit linear regression line demonstrated that, on average, M increased with muscle length ($P = 1.3e\text{-}7$; slope $= 0.04 \pm 0.01$; y intercept $= 0.21 \pm 0.03$; Fig. 11*D*). We expected this increase due to the well known force-length properties of muscle (Gordon et al. 1966).

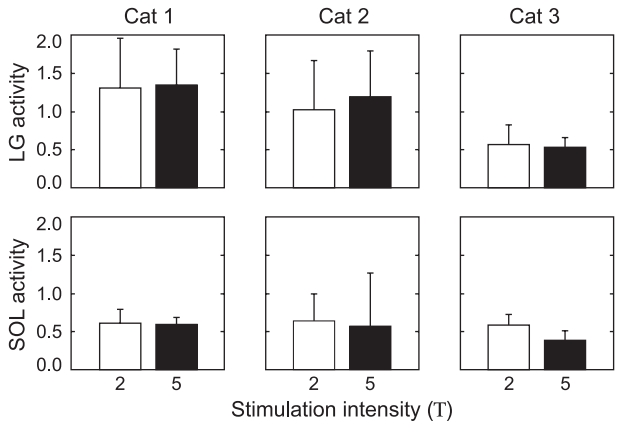

FIG. 9. Electrical stimulation of group II afferents in the MG nerve does not influence the magnitude of burst activity in the LG and SOL muscles. The bar graphs compare the average relative effect of stimulation at 2T (□) and 5T (■) during a 100-ms period (shaded bars in Fig. 8) for 3 cats. This range of intensities represents the range over which most of the group II afferents are recruited (Jack 1978). Error bars denote the SD.

Figure 11*E* plots the estimated loop gain, $K \cdot M$ (*Eq. 3*), as a function of muscle length. This dimensionless number also varied from animal to animal. At 0 mm, for example, $K \cdot M$ ranged from 0.11 to 0.44 and averaged 0.30 ± 0.10. While $K \cdot M$ generally increased with muscle length, there was considerable variability between animals. Linear regression on each animal's data yielded P values that ranged from 0.01 to 0.20 with a median value of 0.04. Although four animals had P values greater than our level of significance (0.20, 0.18, 0.06, and 0.06), all animals demonstrated an increase in loop gain with length. Our statistical power was limited for some animals because we were only able to make measurements at a subset of muscle lengths before they stopped walking. Combining our measurements from all animals and determining the best-fit linear regression line demonstrated that, on average, $K \cdot M$

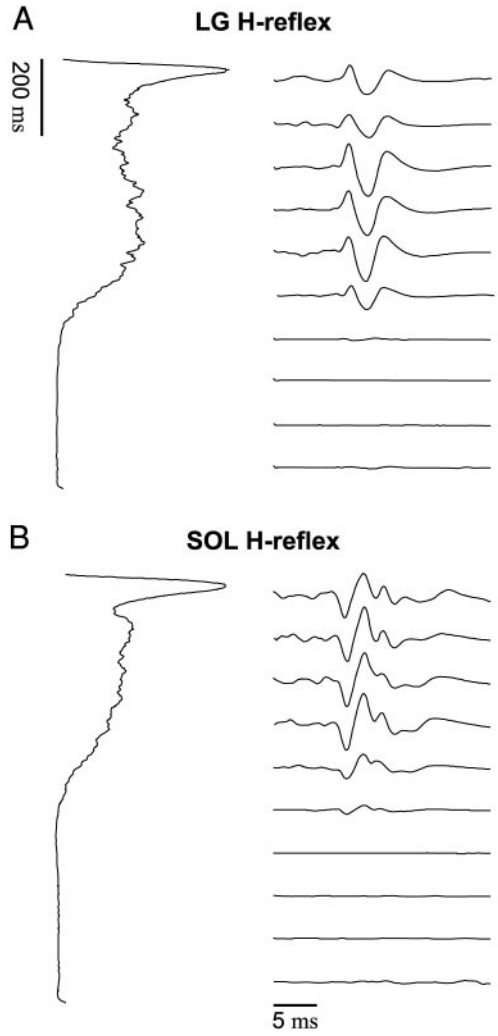

FIG. 10. Representative data illustrates the H-reflexes evoked in the LG (*top*) and SOL (*bottom*) muscles by electrical stimulation of the MG nerve. The step cycle was divided into 10 equal bins. *Right*: traces show average EMG responses to single stimuli (3 T, 0.2-ms duration) for each bin (on average 43 stimuli per bin). The traces begin when the MG nerve was stimulated. *Left*: the average EMG in the LG and SOL muscles is oriented vertically to indicate the corresponding time within the step cycle of each average.

increased with muscle length (P = 3.8e-7; slope = 0.05 ± 0.02; y intercept = 0.31 ± 0.04; Fig. 11*E*). This increase was a result of the loop gain being the product of force feedback gain that, on average, was constant and muscle gain that, on average, monotonically increased with muscle length. Loop gain increased from 0.20 at −2 mm to 0.52 at 4 mm. Because loop gain is the relative contribution of force feedback to total muscle activity and force (*Eq. 3*), these results indicate that force feedback was of modest importance at short muscle lengths, accounting for ~20% of total activity and force, and of substantial importance at long muscle lengths, accounting for 50%. Loop gain was always less than unity, indicating that this positive feedback system was stable (Prochazka et al. 1997).

DISCUSSION

Numerous investigations over the past decade have demonstrated that sensory feedback contributes substantially to ankle extensor activity during the stance phase of walking in humans and cats (Dietz and Duysens 2000; Donelan and Pearson 2004). Currently the origin of the reinforcing sensory signals is uncertain, but it seems likely that they arise from multiple groups of sensory receptors including ankle extensor Golgi tendon organs, ankle extensor muscle spindles, and cutaneous receptors in the feet. An important issue, therefore is to establish the contribution of each class of receptors to the activity in the ankle extensor muscles and the conditions under which each class exerts its influence. In this study, we determined the contribution of feedback originating from MG GTOs to MG muscle activity by estimating the loop gain of the positive force feedback pathway during walking in the cat.

A fundamental observation in our investigation was that the magnitude of MG muscle activity was elevated at longer muscle lengths (Figs. 2 and 3). It was quite clear that this was caused by changes in sensory signals from receptors in the MG muscle or tendon. Under the experimental conditions, the hind leg was extensively denervated, and MG was isolated from the other ankle extensor muscles (Fig. 1). This denervation included nerves innervating the knee joint thus removing feedback from joint receptors. The enhancement of MG activity at longer lengths was not due to a generalized increase in excitability because there was limited influence of MG length changes on activity in some of the other muscles crossing the hip, knee, and ankle (Figs. 2–4) and on muscles of the contralateral leg (unpublished observation). However, the activity in LG, a close synergist of MG, was enhanced when MG was lengthened (Figs. 2 and 3). These observations demonstrate that the important sources of feedback in our investigation are afferent signals arising from the MG muscle-tendon unit that have an excitatory action on homonymous motoneurons as well as motoneurons innervating specific ankle extensors.

Which receptors in the MG muscle are responsible for enhancing the MG burst activity at longer lengths? It is impossible to distinguish the relative contribution of different proprioceptors knowing only the relationship between muscle length and EMG activity. This is because force also increased at longer lengths (Fig. 3). This increase in force was due to both the increase in muscle activity and the intrinsic force-length properties of the MG muscle (Gordon et al. 1966). In addition, although the muscle-tendon length was isometric, there was internal shortening of muscle fibers (as indicated by the decrease in activity of group Ia and II afferents) that may have been dependent on length. Thus sensory signals related to length, velocity, and force all have the potential for modifying the MG activity in a length-dependent manner.

A number of observations allow us to exclude a major contribution of muscle spindles to the enhancement of MG middle region activity. First, the activity in group Ia MG afferents (arising from primary spindle endings) was relatively insensitive to changes in muscle length (Figs. 7). Second, burst activity in SOL was sometimes completely uninfluenced by changes in MG length, yet heteronymous connections from MG group Ia afferents to SOL motoneurons were open as demonstrated by the capacity to elicit an H-reflex in SOL (Fig. 10). This supports our observation that group Ia afferent activity was not significantly elevated during the middle of the muscle contraction. This insensitivity may have been compounded by inhibition of group Ia pathways at the level of the spinal cord; Gosgnach et al. (2000) have reported that group Ia

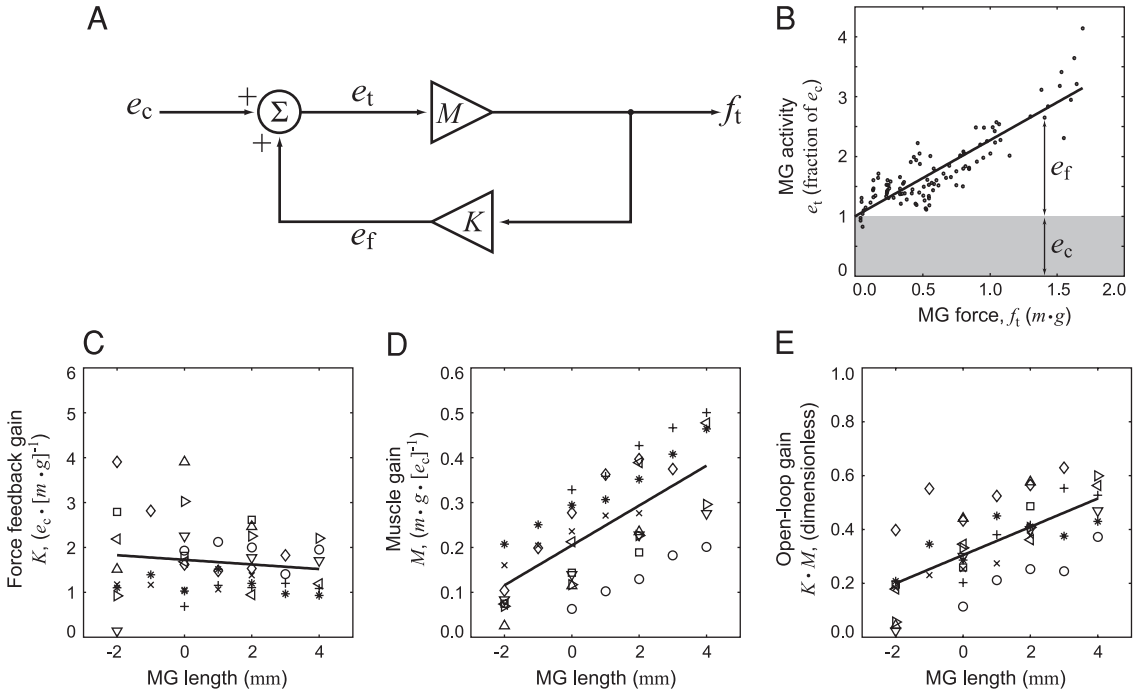

FIG. 11. Plots showing values of force feedback gain, muscle gain, and loop gain (*C–E*, respectively) that were derived using a simple linear model of the neuromuscular system (*A*) and the relationship between MG activity and MG force (*B*). *A*: for more model details, refer to METHODS. *B*: representative data from a single animal. Each symbol is a single burst's middle region magnitude. The line is a best-fit linear regression line. Its *y*-axis intercept estimates the feedforward contribution to muscle activity, e_c (denoted by the horizontal shaded area). *C*: force feedback gain, *K*, was independent of muscle length. *D*: muscle gain, *M*, increased with muscle length due to the intrinsic properties of muscle. *E*: the product of force feedback gain and muscle gain yielded loop gain, $K \cdot M$. It also increased with length. Each symbol in *C–E* represents measurements from a different animal ($n = 10$).

pathways from the ankle extensor muscles are presynaptically inhibited during locomotion in decerebrate cats. However, the fact that we could evoke H-reflexes demonstrates that these pathways were not completely inhibited. Third, although group II MG afferents (arising from secondary spindle endings) were sensitive to length (Fig. 7), electrical stimulation of these afferents had negligible influence on the magnitude of LG activity (Fig. 9). The degree to which this observation reflects on the homonymous action of these afferents depends on our assumption that the same afferents were responsible for the elevation of both MG and LG muscle activity with length. This assumption is supported by our observation that middle region LG and MG activity increased by similar amounts with increased length (Fig. 3). Although an alternative explanation for these results is that stimulation at group I strength saturated interneurons common to both group I and II afferent pathways, previous investigations from other laboratories have also failed to find any excitatory action of extensor group II afferents on cat ankle extensor motoneurons (Gossard et al. 1994; Perreault et al. 1995). Finally, the early region of the MG muscle activity was uninfluenced by changes in muscle length (Fig. 3) despite a strong influence of muscle length on activity in both group Ia and II afferents during the same region (Fig. 7). All these observations allow us to conclude that, under the conditions of our experiment, MG muscle spindles were not the major source of feedback responsible for elevating MG muscle activity at longer muscle lengths. If muscle spindles are excluded, then we are left with the Golgi tendon organs and free nerve endings responsive to length and force as potential sources of the sensory signals enhancing MG activity. It is unlikely that free nerve endings are responsible for the measured feedback effect because currently there is no evidence that group III and IV muscle afferents have an excitatory action on triceps surae motoneurons during walking (Schomburg et al. 2001).

By exclusion, the results indicate that MG GTOs were the main source of sensory signals acting to elevate MG muscle activity in our preparation. This is a reasonable conclusion because previous studies have demonstrated that group Ib pathways from ankle extensors have an excitatory action on extensor activity during walking in spinal and decerebrate cats (Pearson and Collins 1993; Whelan et al. 1995) as well as fictive locomotion in spinal and decerebrate cats (Angel et al. 1996; Conway et al. 1987; Gossard et al. 1994). In addition, this conclusion is consistent with our observation that GTO activity is elevated during the region of interest (Figs. 6 and 7).

This isolation of the contribution of GTOs allowed us to use a simple neuromuscular model to estimate the loop gain of this positive force feedback pathway (Fig. 11; outlined in METHODS). Average loop gain, a quantitative measure of the relative contribution of MG GTO force feedback to MG muscle activity, ranged from 0.2 at short muscle lengths to 0.5 at long muscle lengths. While loop gain was greater at long lengths than short lengths in all animals, the magnitude of loop gain at a given length varied considerably from animal to animal (Fig. 11*E*). While we endeavored to control differences between experiments, some were unavoidable. To understand the source of this variability, it is useful to consider potential contributors to differences in the constituents of loop gain: force feedback gain and muscle gain. Force feedback gain is a property of the nervous system and can be extensively modified. Indeed, the

nervous system modifies the gain of the force feedback pathway so that it is negative during standing and positive during locomotion (Pearson and Collins 1993). Thus variability in our estimates of force feedback gain may reflect differences in the state of each animal's nervous system due to differences in factors like core temperature, time under anesthetic, and duration of experiment. Muscle gain, an intrinsic property of muscle, is the amount of force generated per unit of muscle activity (*Eq. 2*). This relationship depends on the condition of the muscle, which may have been slightly compromised in some preparations, and muscle fatigue, which tends to increase with the duration of the experiment. In addition, it is important recognize that some of the variability is a consequence of the intrinsic differences between animals. While we endeavored to isolate the force feedback pathway, it is possible that other afferents made small, length-dependent, contributions to muscle activity. Thus our values are likely slight overestimates. It is important to note that this loop gain does not estimate the total contribution of force feedback because force feedback from heteronymous sources may also be present in the intact walking cat (Nichols 1999).

Our estimates of gain were made in a very artificial situation, so it is necessary to consider the relevance to conditions in an intact walking animal. During normal walking, MG muscle-tendon length during stance ranges from –1 to –9 mm with length measured in the same manner as in our experiment (Goslow et al. 1973). This upper end of this range just overlaps the low end of the range of lengths we examined in our current investigation because MG did not generate substantial force at lengths shorter than −2 mm (Fig. 3*B*). The range of lengths used in normal walking corresponds to lengths at which the estimated gain of the force-feedback pathway is low, suggesting that the contribution of force feedback to MG activation during walking on a level surface is modest. When walking up slopes, however, the MG muscle can be 7 mm longer during the early part of the stance phase (our calculations using Carlson-Kuhta et al. 1998). This is in the range where the gain of the force feedback pathway is high. Thus force feedback may make a major contribution to the elevation in the magnitude of MG burst activity that is known to occur during uphill walking (Carlson-Kuhta et al. 1998). The observation that MG force increased substantially during uphill walking (Kaya et al. 2003) is consistent with this prediction.

Another important fact to take into account when considering the functional relevance of our data is that the length of the MG muscle is continuously changing during the stance phase: lengthening in early stance and shortening during mid- to late stance. The intrinsic force-velocity properties of the MG muscle (Hill 1938) will increase muscle gain, and thus loop gain, during early stance, yielding a force-feedback contribution higher than that predicted based only on muscle length. On the other hand, the force-velocity properties will tend to reduce loop gain during late stance, decreasing the contribution of force feedback below that estimated from length alone. These velocity-dependent modifications in muscle force are probably enhanced by increases and decreases in the velocity signals from group Ia afferents during early and late stance, respectively.

The measured loop gain does not estimate the total contribution of all homonymous and heteronymous feedback to MG activity during normal walking. It is likely that other afferent pathways contribute to MG motoneuron depolarization under normal walking conditions. Assuming an excitatory action of additional afferent feedback, then our loop gain values are best viewed as estimates of the lower bound of the total contribution of feedback to ongoing ankle extensor activity. For example, because homonymous force feedback is responsible for 50% of MG activity at long muscle lengths, then the remaining 50% is due to feedforward sources as well as other sources of afferent feedback. Under these circumstances, the maximum contribution of feedforward control is 50% but possibly much less due to the contribution of other afferent signals.

In summary, our results indicate that homonymous force feedback from GTOs has a substantial contribution to ongoing ankle extensor activity during walking in the cat. During isometric contractions of the MG muscle-tendon unit, this contribution of feedback from GTOs is modest when the muscle is short but doubles MG muscle activity and force when the muscle is long. The length dependence is due to the intrinsic force-length property of muscle. The gain of the pathway that converts muscle force to motoneuron depolarization, *K*, is independent of length. This positive feedback system is quite stable as the loop gain is less than unity at all measured lengths. In the intact cat, it is likely that homonymous force feedback is not the only important source of feedback as other afferent pathways may make significant contributions when muscle length is allowed to change. This emphasizes the general importance of afferent feedback for generating ongoing ankle extensor activity during walking.

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