Adaptive Changes in Motor Activity Associated With Functional Recovery Following Muscle Denervation in Walking Cats

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Pearson, K. G., K. Fouad, and J. E. Misiaszek. Adaptive changes in motor activity associated with functional recovery following muscle denervation in walking cats. J. Neurophysiol. 82: 370-381, 1999. In this investigation we examined the changes in the pattern of activity in the medial gastrocnemius (MG) muscle in walking cats following transection of the nerves innervating synergist muscles (lateral gastrocnemius, soleus, and plantaris). Immediately following the nerve transections, there was a large increase in ankle flexion during early stance (from ~10° to ~30°) and a marked increase in the magnitude of the MG bursts during stance. We attribute this increase in the magnitude of the MG bursts to an increase in afferent feedback from the abnormally stretched MG muscle. During the week after the nerve transections, there was a progressive decrease in ankle yield. This improvement in ankle function was correlated with an increase in magnitude of two components of the MG bursts; the initial component starting during late swing and ending ~40 ms after ground contact, and a late component associated with stance. The time courses of the increases in the initial and late components of the MG bursts were different. Large and significant increases in the late component occurred the day after the nerve transections, whereas increases in the initial component occurred more gradually. This difference in time course was reflected in the kinematics of ankle movement. Over the first few days after the nerve transections, improvement in ankle movement occurred primarily late in the stance phase, and there was little change in ankle yield during early stance. At 1 wk, however, there was a significant reduction in ankle yield during early stance. This decreased yield was most likely due to an increase in stiffness of the MG muscle at the time of ground contact resulting from the increase in magnitude of the initial component of the MG bursts. The increases in the magnitude of the initial and late components of the MG bursts, as well as the improvement in ankle function, depended on use of the leg. All these changes were delayed by immobilizing the leg for 6 days in an extended position. We discuss possible mechanisms underlying the increase in the magnitude of the MG bursts and propose that proprioceptive signals from the stretched MG muscles provide an error signal for rescaling the magnitude of the centrally generated initial component. Our data support the concept that proprioceptive feedback functions to scale the magnitude of feed-forward motor commands to ensure they are appropriate for the biomechanical properties of the musculoskeletal system.

INTRODUCTION

A necessary condition for maintaining precision of movement is the ability of motor systems to adapt to changes in the biomechanical properties of the moving elements. These changes occur naturally during development and aging (Forsberg et al. 1991), or they can result from injury to the muscu-
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predicted that the early stages of functional recovery (over the 1st wk) would be associated with a progressive increase in MG activity and that this increase would be correlated with a decrease in yield at the ankle joint. Although this simple prediction was confirmed, the characteristics of the changes in MG activity and ankle kinematics were found to be more complex than anticipated. Preliminary results of this study have been published in abstracts (Misiaszek and Pearson 1998; Pearson et al. 1997).

METHODS

Experiments were performed on 8 adult male and female cats weighing between 2 and 3.5 kg. The experimental procedures were approved by the Animal Welfare Committee at the University of Alberta.

Experimental procedure

All animals were first trained to walk on a motor-driven treadmill at speeds ranging from 0.3 to 0.8 m/s. Training sessions were given daily and lasted for ~20 min. The number of training sessions required for obtaining satisfactory walking varied from animal to animal but was never longer than 14 days. After training was complete, EMG recording electrodes were implanted into the following hind leg muscles in seven of the eight animals: MG and vastus lateralis (VL) in the right hind leg, and MG in the left hind leg. The eighth animal was used only in a study of kinematics following hind leg immobilization (see the end of this section). The procedure for implanting EMG electrodes for recording in intact, walking animals has been described elsewhere (Whelan and Pearson 1997). Two pairs of recording electrodes were implanted in the right MG muscle in four animals (cats 3, 4, 6, and 8). One pair was located proximally in the muscle ~3 cm from the knee insertion, and the other pair was located ~3 cm more distally. This procedure provided a safeguard in the event of failure of a recording pair. In addition, the similarity of data recorded with each pair indicated that the changes in EMG profiles were not due to movement of the electrodes. The EMG electrodes consisted of two multistranded stainless steel wires (Cooner Wire Company, AS632) insulated except for ~3–4 mm length positioned in the muscle. The end of each electrode wire was threaded through the muscle using a 21-gauge needle crimped to the end of the wire. The emerging ends of the two electrode wires were knotted and then fixed to the muscle with a silk suture. Two to three days after the implantation of the EMG electrodes, the patterns of activity in the implanted muscles were recorded as the animals walked on the treadmill. This was repeated daily for 3–5 days to ensure that signals were stable from one recording session to the next.

The main purpose of the experiments was to examine changes in the EMG patterns and leg kinematics following denervation of the LG, S, and PL muscles of the right hind leg (Fig. 1A). Under halothane anesthesia (halothane mixed with 95% oxygen and 5% carbon dioxide) and aseptic conditions, the common nerve to LG and S (the LGS nerve) and the nerve to PL were exposed and transected. Sutures were placed on the proximal ends to allow later identification of the transected nerves. About 5 h after recovery from the anesthetic, EMG and video recordings were made while the animals walked on the treadmill over the range of speeds used during training. In four animals these recording sessions were repeated on an almost daily basis for at least 1 wk. These sessions were extended to 10 days and 13 days in two animals. Before all the recording sessions, reflective markers were placed on the skin over the iliac crest, the hip, knee and ankle joints, and on the paw to allow measurement of joint angles from video recordings.

In four other animals the right hind leg was immobilized on the day the LGS and PL nerves were transected. In each animal, the leg was immobilized with a Quicksplint (Jorvet) plastic splint immediately after a recording session done 5 h after the nerve transections. The splint fixed the knee and ankle joints in an extended position. In three animals the splint was removed after 6 days. EMG and video recordings were made in two of these animals within 30 min after splint removal, on a daily basis for 1 wk thereafter, and less frequently over the next 2–3 wk. Only video recordings were made in the third animal because no EMG electrodes were implanted in this animal. In the fourth animal we were unable to maintain the splint fixed on the leg for more than 1 or 2 days. This animal provided no useful data.

Data analysis

The raw EMG signals were recorded onto magnetic tape using a Vetter 4000A PCM recording adapter. Later these signals were full-wave rectified, filtered (low-pass 20 Hz), and stored on computer disk using the Axotape (Axon Instruments) data acquisition system. The sampling rate on each channel was 700/s. The magnitude and timing of the yield on ankle angle during stance before cutting the nerves (middle), and 7 days later (right). Thin traces: averages of 10–20 steps for individual animals. Thick traces: mean of the averages. Each trace begins 2 frames (66.7 ms) before ground contact. Walking speed was 0.6 m/s. Note the large increase in yield (flexion) during early stance on the day the nerves were cut, and the reduction in the magnitude of the yield on day 7. C: superposition of plots of change in ankle angle during stance (relative to the angle at the onset of stance) showing recovery toward control from day 0 to day 7. These traces correspond to the thick traces in B.

FIG. 1. Kinematics of ankle movement following transection of the nerves supplying the lateral gastrocnemius (LG), soleus (S), and plantaris (PL) muscles of a hind leg in the cat. A: experimental preparation. B: plots of ankle angle during stance before cutting the LGS and PL nerves (left), 5 h after cutting the nerves (middle), and 7 days later (right). Thin traces: averages of 10–20 steps for individual animals. Thick traces: mean of the averages. Each trace begins 2 frames (66.7 ms) before ground contact. Walking speed was 0.6 m/s. Note the large increase in yield (flexion) during early stance on the day the nerves were cut, and the reduction in the magnitude of the yield on day 7. C: superposition of plots of change in ankle angle during stance (relative to the angle at the onset of stance) showing recovery toward control from day 0 to day 7. These traces correspond to the thick traces in B.
of the EMGs were measured using custom software capable of retrieving and displaying Axotape files (see RESULTS for details of measurements).

To quantify the changes in the profile of the MG EMG during recovery following the transection of the LGS and PL nerves, the average level of the EMG was measured over two periods (Fig. 4B). The first period was the initial 120 ms of the burst, and the second was a 100-ms period centered on the peak of EMG activity that followed ground contact. We refer to these two periods of activity as the initial and late components of the MG EMG, respectively. The rationale for measuring these two components was that the predominant increase in

The first period was the initial 120 ms of the burst, and the second was a 100-ms period centered on the peak of EMG activity that followed ground contact. We refer to these two periods of activity as the initial and late components of the MG EMG, respectively. The rationale for measuring these two components was that the predominant increase in the magnitude of the MG bursts over the first day after nerve transection commenced $\sim$120 ms after burst onset (Fig. 4A). This suggests that different mechanisms regulate the generation of the initial and late components and is consistent with data from previous studies. Gorassini et al. (1994) reported that the initial 120 ms of ankle extensor activity is uninfluenced by sensory signals generated at the time of ground contact, and that the later component of the burst is strongly dependent on afferent feedback (Gorassini et al. 1994). Hiebert and Pearson (1999) also found a strong dependence of the late component on afferent feedback in walking decerebrate cats. The two components of activity in ankle extensor muscles have different functions. The initial component establishes the stiffness of the muscle at the beginning of stance thus functionally to support the animal’s weight during early stance, whereas the late component contributes to forward propulsion (Grillner 1972). Because we wished to determine changes in the profile of the MG EMG in the days after the transection of the LGS and PL nerves, all averages were normalized relative to values measured during the recording session on the day the nerves were transected (day 0). Because this initial recording session was done 5 h after surgery, it was important to establish that MG activity was uninfluenced by the anesthetic and surgical trauma. Thus in two animals being used in an unrelated series of experiments, we performed a sham operation (exposing the LGS and PL nerves in the usual manner) and compared MG burst activity recorded 5 h later with that recorded before the sham operation.

The kinematics of stepping in the right hind leg were determined from digitized images from the video recordings. A Miro DC20 video capture card was used for digitizing the images at 30 frames/s, and custom-written software was used to calculate joint angles from these images. Movement of the reflective marker at the knee relative to the joint was not considered in the analysis because this had only a minor influence on the measurement of the kinematics of movements at the ankle joint, which was our primary objective. Calculations of ankle angles using knee positions derived from triangulation from the hip and ankle markers revealed that the error of measurement using the knee marker was within $\pm 2^\circ$. The reason for the small error is that the knee marker moved primarily along a path parallel with the shank. The kinematics at the ankle were determined by averaging the joint angles of 10–20 steps while the animal was walking regularly at one position on the treadmill. The EMG and video recordings were synchronized by matching a pulse-code on one channel of the Vetter recorder with a corresponding number displayed in the field of the video recorder.

The preferred walking speed for most animals was in the range of 0.5–0.7 m/s. Thus we chose the walking speed of 0.6 m/s to quantify the MG EMGs and the ankle kinematics. At this speed, long sequences of stepping occurred in all animals.

RESULTS

Changes in ankle kinematics following LGS and PL nerve transection

During normal walking in cats, there is a flexion at the ankle during the initial part of the stance (the E2 phase of the step cycle). As the animal’s weight is transferred to the supporting leg, the active ankle extensor muscles are loaded and stretched eccentrically (Goslow et al. 1973). Transsection of the LGS and PL nerves, which left only the MG muscle controlling movements about the ankle, led to an increase in the magnitude and duration of yielding at the ankle during early stance (Fig. 1, B and C). In animals walking at 0.6 m/s, the average yield before nerve transection was 9.7 $\pm$ 3.5° (mean $\pm$ SD; $n$ = 7), and this increased to 29.3 $\pm$ 7.8° after nerve transection (the recording session was delayed 5 h to allow recovery from the anesthetic). The duration of the yielding approximately doubled from $\sim$100 ms to 200 ms. In addition to this increase in ankle yield, there were other less obvious effects of LGS and PL nerve transection. One was a decrease in the maximum extension of the ankle at the end of stance from $\sim$139° to 111°. Another was a decrease in ankle angle just before stance onset from 116° to 104°. And a third was an increase extension at the hip. We did not examine the latter change in detail, because the primary focus of this investigation was the change in activity of the isolated MG muscle (see next section) and the associated changes in ankle movements.

During the first week following transection of the LGS and PL nerves, there was a progressive improvement in stepping that was obvious by casual visual observation, most noticeably indicated by a decrease in the drop of the hindquarters during the stance phase of the experimental leg. Two factors contributed to this improvement. The first was a progressive decrease in the yielding at the ankle (Fig. 2A). At 1 wk the average yield was 16.7 $\pm$ 6.3° ($n$ = 4), which constitutes a 64% reduction of the acute deficit (Fig. 2B). In no animal, however, did the yielding at the ankle return to normal by 1 wk. The second factor was a return of the ankle angles at the beginning and end of stance to values close to normal (Fig. 1B). For the four animals studied, the average values of these angles before the nerve transections were 116° and 139°, respectively. One week after the nerve transections, they were 113° and 135°, respectively. Thus the first conclusion is that substantial functional recovery occurs after transection of the LGS and PL nerves, and a large part of this recovery occurs during the first week.

In this investigation we did not establish whether full functional recovery could be achieved by keeping the animals for long periods after the nerve transections. However, in one of the initial group of four animals, it was clear that a deficit in ankle movement (increased yield during early stance) persisted for 2 wk (Fig. 2A, the remaining 3 animals were not examined to this length of time). Furthermore, in the 3 animals used to investigate the effect of leg immobilization following the nerve transections (see Effects of leg immobilization; Figs. 9 and 10), the ankle yield during early stance also did not return to normal within 2 wk of removing the immobilizing cast even though these animals showed substantial functional recovery during the first week of leg use.

Changes in the profile of EMG activity in MG during recovery

Immediately after transection of the LGS and PL nerves, there was a large increase in the magnitude of burst activity in MG (Figs. 3 and 4B). We attribute this increase mainly to reflex activation of MG motoneurons in response to the large stretch and additional loading of the MG muscle (see next section). Subsequently, the peak magnitude of the EMG bursts...
continued to increase noticeably over the next 2 days (Figs. 3 and 4A).

The changes in the profile of the MG EMG during recovery following the transection of the LGS and PL nerves were quantified by measuring the magnitude of EMG activity over two periods (Fig. 4B; see METHODS for rationale). The first period was the initial 120 ms of the burst (initial component), and the second was a 100-ms period centered on the peak of EMG activity that followed stance onset (late component). To quantify the changes in the MG EMG, the magnitude of the bursts was measured over 2 periods: the 1st 120 ms (initial component) and a period of 100 ms at the peak of activity (late component). See text for more details.

FIG. 2. Functional recovery of ankle movements following transection of the LGS and PL nerves. A: plot for one animal showing the progressive reduction in yield over a period of 2 wk. Note that most of the recovery occurred within 1 wk. Each data point is the average of the yield measured for 10 consecutive steps. Error bars are standard deviations. The control value was determined on the day before the nerve transections (day -1), B: histograms showing the average ankle yield the day before the nerve cuts (day -1), the day of the cuts (day 0), and 1 wk later (day 7). The data for days -1 and 0 came from 7 animals, whereas the data for day 7 came from 4 of these 7 animals (the remaining 3 had their leg immobilized on day 0). Error bars are standard deviations. All data obtained when animals were walking at 0.6 m/s.

FIG. 3. Examples of electromyograms (EMGs) recorded from the medial gastrocnemius (MG; top) and vastus lateralis (VL; bottom) muscles in one animal before (day -1), 5 h after (day 0), and 2 days after (day 2) transection of the LGS and PL nerves. Top traces: raw EMGs. Bottom traces: rectified and filtered EMGs. Note the increase in the magnitude of the MG EMG on day 0 and a further increase in magnitude on day 2. These increases in the magnitude in MG EMG were not associated with changes in the magnitude of VL EMG, the latter remaining relatively constant throughout.

FIG. 4. Changes in the profile of the MG EMG following transection of the LGS and PL nerves. Each trace is the average of 20 rectified and filtered bursts. A: averaged records from 4 cats showing the increase in MG EMG that occurred during the 1st day following the nerve transections. Thin traces, 5 h after transections (day 0); thick traces, 24 h after nerve transections (day 1). Note that in all animals the major increase in magnitude commenced -120 ms (dotted line) after the beginning of the bursts. B: example from one animal (cat 2) of the changes in the profile of the averaged MG EMG soon after the nerve transections (days 0 and 1) and 6 days after the nerve transaction (day 6). For reference, the thin traces show the profile of the MG EMG on the day before the nerve transections. Note that the main difference in the EMG profiles on days 1 and 6 was an increase in the magnitude of the initial 120 ms of activity. To quantify the changes in the MG EMG, the magnitude of the bursts was measured over 2 periods: the 1st 120 ms (initial component) and a period of 100 ms at the peak of activity (late component). See text for more details.
initial component increased more gradually than the increase in the late component. By day 7 the percentage increase in the magnitude of the initial and late components were comparable. In the two animals in which measurements were extended beyond 1 wk (cats 1 and 3), the initial component increased slightly after 1 wk, and the late component decreased.

The difference in time course of the changes in the initial and late components of the MG EMG was associated with different aspects of functional improvement in ankle movement (Fig. 6). The kinematic and EMG data in each set of records in Fig. 6 are from the same walking sequences in the same animal (cat 1). The large increase in the magnitude of the late component of the MG EMG in the first few days after the nerve transections was associated with an increase in ankle extension during the latter half of the stance phase. In the example shown in Fig. 6A, the increase in magnitude of the late component on day 3 relative to day 1 was associated with a delayed increase (latency, 110 ms) in ankle extension. The yield of the ankle joint during early stance (the initial 165 ms of the stance phase) was similar on both days. By day 7 the initial component of the MG EMG had increased relative to that on day 1, and this increase was associated with a reduction in the initial rate of yielding as well as a decrease in maximum yield (Fig. 6B). Qualitatively similar observations were made in all other animals.

An important issue is whether or not the quantitative assessment of the changes in the MG EMG was influenced by the anesthetic and surgical trauma before the measurements on day 0. To quantitatively assess the adaptive changes in the MG EMG, the amplitudes of the first and second components were normalized relative to the values recorded 5 h after transecting the LGS and PL nerves. If these control values were influenced by anesthetic and surgical trauma, then our quantification of the changes would be incorrect. Our main concern was if the control values were lowered by surgical trauma and the anesthetic, then our quantification procedure would overestimate the adaptive increases in MG activity. A number of observations indicate that anesthetic and surgical trauma do not have a noticeable influence on the magnitude of MG activity recorded on day 0. The first was the absence of any difference in the profile of the MG bursts before and 5 h after a sham operation (data not shown). A sham operation was performed in two animals. The animals were anesthetized in the usual manner, and the LGS and PL were exposed as usual but not cut. Second, the observation in cats 1 and 3 that there were only small changes in the initial component over the first 2 days suggests that residual effects of the operative procedure on day 0 were

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**FIG. 5.** Increases in the magnitude of the initial and late components of the MG EMG occur with different time courses. Each set of graphs shows plots of the magnitude of the initial (– – –) and late (—) components vs. days following cutting the LGS and PL nerves for each of the 4 cats studied. Each data point is the average of at least 50 individual bursts. Note the relatively slow increase in the magnitude of the initial component.
minimal. A third observation was that the magnitude of the VL activity was not altered by transection of the LGS and PL nerves (Fig. 3). This demonstrates that the operative procedure did not globally alter leg extensor activity. Furthermore, it indicates that the animal was not favoring the leg by reducing the load carried by the leg because activity in the VL muscle is sensitive to loading (Hieber and Pearson 1999). Finally, in unpublished experiments examining the effect of injecting botulinum toxin into ankle extensor muscles other than MG (a procedure requiring anesthetic and surgery), we have never observed an immediate decrease in the magnitude of MG bursts. Any increases, if they occurred, were small compared with those produced by transection of the LGS and PL nerves.

**Afferent contribution to the generation of the late component**

An understanding of the mechanisms underlying the changes in the profile of the MG EMG requires knowledge of how the MG bursts are generated. There is some evidence that the processes contributing to the generation of the initial and late components are not identical (Gorassini et al. 1994; Hiebert et al. 1994). In particular, afferent feedback during the stance phase may make a significant contribution to the generation of the late component. Two observations in this study support this concept. The first was that unloading the hind legs by gently lifting the hindquarters via the tail reduced the magnitude of the late component without noticeably influencing the initial component (Fig. 7A). This phenomenon was observed in two animals that continued to walk in a coordinated and regular manner while lifted. Squeezing the tail alone without lifting did not alter the magnitude of the late component. The reduction of the late component was associated with decreased flexion of the ankle joint (Fig. 7B) and hence decreased stretch of the MG muscle. The second observation was that the magnitude of the late component was correlated with the magnitude of ankle flexion (Fig. 8). In all animals there were times when walking was continuous but irregular. The irregularities were associated with changes in head position and alterations in position on the treadmill. During these times the magnitude of the MG EMG was quite variable, and the larger bursts were associated with larger flexion movements at the ankle (Fig. 8A). A positive correlation between the magnitude of the late component of the MG EMG and the amplitude of ankle flexion (Fig. 8B) was observed in all animals (n = 4). This correlation is consistent with the notion that afferent signals generated by lengthening of the MG muscle during early stance contributes to the generation of the late component of the MG bursts. During periods of irregular walking, no consistent correlation was found between the magnitude of the initial component of the MG EMG and the magnitude of ankle flexion. This absence of correlation was most likely due to variations in the loading of the leg when the animal was walking irregularly. In this investigation we did not attempt to measure loading with either force plates or implanted force transducers.

The timing of the increase in the amplitude of the late component of the MG EMG in the first few days following transection of the LGS and PL nerves is also consistent with the idea that afferent signals contribute to the late component. At these early times there was a noticeable increase in MG activity beginning ~120 ms after burst onset (Fig. 4). This increase occurred ~40–50 ms after ground contact, a value that is appropriate if afferent signals generated soon after the time of stance onset contribute the late component of the MG EMG.

**Effects of leg immobilization**

An interesting issue is whether the functional recovery in ankle movement we observed, and the associated increases in the magnitude of both components of the MG EMG, depends on use of the leg during the recovery period. To examine this issue we immobilized the leg in three animals for 6 days, commencing on the day of the nerve transections (day 0). EMG (2 animals) and video recordings (3 animals) were made ~5 h after the nerve transections and immediately before leg immobilization. After removal of the immobilizing cast, these recordings were made daily. In contrast to previous animals, none of the animals in which the legs were immobilized showed significant functional recovery 7 days after nerve transection (1 day after cast removal; Fig. 9). However, 8 days after cast removal (day 14) there was a decrease in ankle yield from average values of ~32° to ~18°. This improvement was
similar to that observed over the first week in animals in which
the leg was not immobilized (compare with Fig. 2). Thus our
kinematic data indicate that use of the leg was required for
functional recovery.

Support for this conclusion came from our EMG recordings
in two of the animals in which the experimental leg had been
immobilized (Fig. 10). On the day of cast removal (6 days after
the nerve transections), the magnitudes of the initial and late
components of the MG EMG were similar to, and sometimes
less than, the values recorded on the day of the nerve transec-
tions. Normally, in the absence of immobilization, both com-
ponents increased by $\geq 40\%$ within 6 days (Fig. 5). In the week
following cast removal, the magnitudes of the initial and late
components increased. At 1 wk after cast removal (day 13), the
initial and late components had increased by $\sim 50$ and 100%,
respectively, compared with values on the day of cast removal.
These values are similar to the increases measured on day 7 in
some animals in which the leg was not immobilized (compare
with Fig. 5).

A few days after immobilizing the hind leg, all animals
learned to walk using the other three legs. This enabled us to
record EMGs in the immobilized leg during walking. In both
animals there was no rhythmic activity in the MG and VL
muscles associated with tripedal stepping. Occasionally bursts
of activity would occur, but these were not obviously corre-
lated with stepping in the other legs. Often both muscles were
silent, but when they were tonically active the level was low.
We conclude, therefore, that leg immobilization significantly
reduced activation of the MG muscle and completely sup-
pressed rhythmic locomotor activity in this muscle.

**DISCUSSION**

In this investigation we have examined the adaptive changes in
kinematics and motor activity following partial denervation of
the ankle extensor muscles in walking cats. The MG muscle was
forced to function as the sole ankle extensor by transecting the
nerves supplying all other ankle extensors (S, LG, and PL). The
main finding of this investigation was that two processes contrib-
ute to changes in the burst activity in the MG muscle that are
associated with functional recovery of movements around the
ankle. The first was an increase in activation of the MG muscle
during the mid to late stages of the stance phase. The second,
which is slower than the first, was a progressive increase in the
initial burst of activity in MG that begins before stance onset.
These increases in the initial and later components of the MG
EMG were correlated with distinctly different features of the
kinematics of ankle movement. The increase in the late compo-
ment was associated with an increase in the magnitude of ankle
extension in the second half of stance, whereas the increase in the
initial component was associated with a reduction in the initial
flexion at the ankle during early stance.

**Technical considerations**

Because our conclusions are based to a large extent on
measurements of EMG activity over periods from 1 to 3 wk, an
important technical issue is the stability of the EMG record-
ings. It is conceivable that progressive changes in the electrode positions or in the physical properties of the electrodes could contribute to the changes we found in the MG EMG during the period of functional recovery. Although we cannot exclude these effects, a number of observations indicate that they cannot be responsible for producing the increase in MG EMG that occurred after transection of the LGS and PL nerves. The most compelling observation is the difference in time course of the increase in the initial and late components of the MG EMG (Fig. 5). Movement or physical changes in the electrodes would be expected to alter both components in parallel, i.e., the time course of changes would be the same for both components of the EMG. A second observation was that similar changes in the magnitude of MG EMG activity were measured by the two sets of electrodes implanted in the MG muscle of the experimental leg (data not shown). This indicates that there were no major shifts in the positions of the electrodes in the muscle due to mechanical factors. Finally, progressive changes in the electrical properties of the interface between the recording wires and the muscle can be excluded by the fact that these changes were not observed in other muscles. Moreover, there was not a progressive increase in the magnitude of MG EMG activity during the period before transection of the LGS and PL nerves (up to 6 days). In a few animals, we did observe a change in magnitude early in this period, but this was always a small decrease, not an increase.

Another technical issue is the extent to which our data may have been influenced by the anesthetic and surgical trauma on the day of the nerve transections (day 0). Our data were normalized to values measured on day 0. In all animals we delayed recordings on day 0 for at least 5 h after the termination of the anesthetic. After short surgical procedures (<1 h) using low levels of anesthetic (2–2.5% halothane in 95% oxygen and 5% carbon dioxide), it has been our experience that animals recover the capacity for walking within ~30 min. Thus we consider that locomotor ability would have fully recovered by 5 h. Indeed, all our animals readily walked (albeit with a deficit produced by the nerve transections), and there were no indications of sluggishness. Moreover, stepping in the contralateral leg appeared normal by visual inspection (we did not measure the kinematics of movement of this leg), and the bursts of EMG activity in ipsilateral VL muscle (Fig. 3) and the contralateral MG muscle were similar in magnitude to the bursts recorded before the anesthetic. Further evidence that surgical trauma and/or anesthetic did not influence the levels of activity in the MG muscle on day 0 was that no changes were observed.

**FIG. 8.** Variation in the magnitude of the late component of the MG EMG was correlated with the magnitude of flexion of the ankle during early stance. Data for the plot in B were obtained from cat 3 when it was walking irregularly but continuously 5 h after transection of the LGS and PL nerves. The change in ankle angle was determined by comparing ankle position at the time of stance onset with ankle position 180 ms later. A: section of rectified and filtered MG EMG showing variability in magnitude, and stick figures of leg positions for a small (left) and a large (right) burst. Thin and thick traces in the stick figure show the leg position at stance onset and 180 ms later, respectively. Shaded triangles indicate the changes in ankle angle. Data points for these 2 examples are indicated by arrows in B.
Both deficits decreased over the week after the nerve transection, and a decrease in ankle extension at the end of stance was associated with a large increase in yield (flexion) at the ankle during early stance. The immediate effect of transecting the LGS and PL nerves was a large increase in yield (flexion) at the ankle during early stance and a decrease in ankle extension at the end of stance (Fig. 1B). Both deficits decreased over the week after the nerve transections and was obvious by day 14. The changes in the activity in the isolated MG muscle during functional recovery following transection of peripheral nerves (Carrier et al. 1997; Wetzel et al. 1973; Whelan et al. 1995) and removal of muscles (Walsh et al. 1978). In addition, chronic decerebrate cats have the ability to slowly adapt stepping patterns in response to changes in the nature of ground support (Yanagihara et al. 1993). The present study is the first to quantify the time course of changes in the locomotor pattern in the cat during functional recovery following damage to the neuromuscular system.

Functional recovery of ankle movement

The immediate effect of transecting the LGS and PL nerves was a large increase in yield (flexion) at the ankle during early stance and a decrease in ankle extension at the end of stance (Fig. 1B). Both deficits decreased over the week after the nerve transections. The magnitude of ankle extension at the end of stance returned to almost normal values, whereas the deficit in yield was reduced by ~64%. The recovery of these two aspects of ankle movements appear to be independent processes because the enhancement of ankle extension commenced on the day after the nerve transection and was obvious by day 3, whereas only a minor reduction in yield occurred during this period (Fig. 6A). Reductions in the magnitude of yield were prominent by 1 wk (Fig. 6B).

The changes in the activity in the isolated MG muscle during the first week also indicated that two processes contributed to functional recovery. During the first few days there was a large increase in the magnitude of MG EMG activity beginning ~120 ms after the onset of burst activity and persisting throughout the burst (Fig. 4). By contrast, the initial component of the MG EMG (the 1st 120 ms of burst activity) increased more gradually during the week after the nerve transections, with only relatively small increases occurring in the first few days. At 1 wk the increases in the initial and late components of the MG EMG were similar.

From a functional viewpoint it appears that the increase in the late component of the MG EMG contributes to enhancing the ankle extension movement during the stance phase, whereas the increase in the initial component reduces the yield during early stance. The main function of the initial component of EMG activity in the ankle extensors is to establish the stiffness of the ankle at the time of stance onset (Grillner 1972). It is well-known that the intrinsic stiffness of the ankle extensor muscles increases with the level of activity (Grillner 1972; Hoffer and Andreassen 1981). Thus the increase in the magnitude of the initial component of the MG bursts would increase the stiffness of the muscle at the time of stance onset, and this increase in stiffness would function to decrease the magnitude and rate of yield. The function of the late component of the MG EMG is to support the animal’s weight during mid to late stance and provide a forward propulsive force. In this study we did not measure the force in the isolated MG muscle or the ground reaction forces during functional recovery, but we assume that these increased as a result of the increase in the magnitude of the late component.

This investigation provides additional evidence that motor patterns for walking can adapt in a functionally relevant manner to alterations of the biomechanical properties of the hind legs. Earlier studies in intact animals have reported significant functional recovery following transection of peripheral nerves ( Carrier et al. 1997; Wetzel et al. 1973; Whelan et al. 1995) and removal of muscles (Walsh et al. 1978). In addition, chronic decerebrate cats have the ability to slowly adapt stepping patterns in response to changes in the nature of ground support ( Yanagihara et al. 1993). The present study is the first to quantify the time course of changes in the locomotor pattern in the cat during functional recovery following damage to the neuromuscular system.

Mechanisms for adaptive changes in MG EMG

Our data revealed that the profile of the increase in MG activity did not occur uniformly throughout the burst. In the first few days of recovery, there was a rapid increase in the component associated with the stance phase of the step cycle and only a relatively small increase in the component associated with extension during late swing (Fig. 6). The initial component eventually increased in magnitude to values that approached the increases in the late component (Fig. 5). A previous investigation of these two components in normal walking animals concluded that the initial component is generated by central mechanisms largely independent of sensory feedback, whereas the generation of the late component depends to some extent on afferent feedback from leg receptors ( Gorassini et al. 1994). In this investigation we obtained additional data that support the view that afferent feedback contributes to the generation of the late component of the MG EMG. First, the late component was markedly decreased when the hind legs were unloaded by slightly lifting the hindquarters (Fig. 7). This procedure had only a minor effect on the initial component. Moreover, the onset of the difference between the EMGs in the normal and unloaded conditions occurred ~40 ms after stance onset. This latency is consistent with the idea that afferent signals, generated at the time of stance onset, contribute to the late component of the MG EMG. Second, during periods of irregular walking, there was a positive correlation between ankle flexion and the magnitude of the late component (Fig. 8). Because larger ankle flexion is associated with larger stretch of the MG muscle, this observation indicates that feedback from stretch-sensitive afferents in the MG muscle contributes to the generation of the late component.

If afferent feedback does contribute to the generation of the late component of the MG EMG, the increase in the magnitude of this component during recovery may be due to facilitation of transmission in reflex pathways reinforcing the central drive to the MG motoneurons. Previous studies have concluded that at
At least three excitatory reflex pathways could contribute to extensor burst generation in walking animals (Guertin et al. 1995; Pearson 1995; Pearson and Collins 1993). One is the monosynaptic pathway from group Ia afferents, another is a disynaptic pathway from group Ia and Ib afferents opened during extensor activity, and the third is a polysynaptic pathway from group Ia and Ib afferents acting via the extensor half-center. Currently we have no information which, if any, of these pathways might be facilitated during the initial stage of functional recovery, although previous findings appear to exclude changes in the monosynaptic group Ia pathway. Intracellular recordings from MG motoneurons have revealed no significant changes in the magnitude of homonymous or heteronymous group Ia excitatory postsynaptic potentials (EPSPs) within 1 wk of transecting the LGS nerve (Fouad and Pearson 1997; Gallego et al. 1979; Whelan et al. 1995).

The process of facilitation, if it occurs, could be driven by additional feedback from proprioceptors in the MG muscles. During the first few days of recovery, feedback from both muscle spindles and Golgi tendon organs would be enhanced because of increases in stretch and force in the MG muscle during stance. A process akin to long-term potentiation could then enhance transmission in the pathways from these proprioceptors to MG motoneurons. For this mechanism to work, it requires use of the leg. Hence our finding that the increase does not occur when the leg is immobilized (Fig. 10) is consistent with this hypothesis. Furthermore, this hypothesis predicts alterations in synaptic transmission in the spinal cord. An analysis of field potentials evoked from MG group I afferents has revealed increases in the strength of synaptic transmission to interneurons in the intermediate nucleus following transection of the LGS nerve (Fouad and Pearson 1997), but whether this increase is in afferent pathways contributing to MG burst generation is unknown.

Another possible explanation for the progressive increase in the late component of the MG EMG is that it is due to an increase in dynamic fusimotor drive to the MG muscle spindles. Any increase in dynamic fusimotor activity would be expected to increase the stretch sensitivity of primary spindle afferents (Matthews 1972), thus increasing the contribution of feedback from muscle spindles to the generation of the MG bursts. Again, if this occurs, it must depend on use because the increase in the late component does not occur when the leg is immobilized.

Finally, it is possible that neither increased dynamic fusimotor activity nor facilitation of reinforcing reflex pathways contribute to the increase in the late component. Instead the increase may be due to a greater central drive to the MG

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**FIG. 10.** Leg immobilization prevents the increase in the initial and late components of the MG EMG. Plots of the initial (top) and late (bottom) components of the MG EMG in 2 animals in which the hind leg was immobilized for 6 days (shaded area) after transecting the LGS and PL nerves. Magnitudes of both components were normalized relative to values recorded on day 0. Note the progressive increase in magnitude following freeing the leg. These increases are qualitatively similar to those occurring in animals allowed the use of the leg in the days immediately after the nerve cuts (see Fig. 5).
motoneurons. Previous studies in walking decerebrate cats have estimated that central drive and afferent feedback contribute about equally to the activation of ankle extensor motoneurons during the stance phase (Hiebert and Pearson 1999; Severin 1970). If this is the mechanism, then there must be a process for independently regulating the central inputs for generating the late and initial components of the MG EMG, because the increases in these two components do not occur in parallel (Fig. 5). Furthermore, these regulatory mechanisms would have to be controlled by some sort of afferent signal because the increase in both components of the MG EMG did not occur in the absence of movement.

An intriguing issue is the mechanism responsible for increasing the magnitude of the initial component of the MG EMG. Currently, nothing is known about the cellular mechanisms responsible for generating the initial burst of activity in ankle extensors associated with the E1 phase of the step cycle. This burst occurs during stepping in spinal animals (Forssberg et al. 1980), and it is uninfluenced by the unexpected removal of ground support (Gorassini et al. 1994). Thus the initial component appears to be generated centrally by networks within the lumbar cord, although a contribution of afferent feedback during the swing phase has not been excluded. Because this component is essential for establishing an appropriate amount of yield at the ankle, it follows that its magnitude must be matched to the task, e.g., walking versus running, and to biomechanical parameters influencing loading of the ankle extensor muscles, such as the animal’s weight and the dimensions of musculoskeletal elements. Indeed we have noted that the magnitude of the early component increases with walking speed (unpublished observations). Therefore a more general question is how is the magnitude of the early component scaled to be appropriate for the locomotor task and body biomechanics?

One possibility is that the animal develops an internal model for controlling activation of leg muscles that includes the appropriate scaling of the initial component. This is analogous to suggestions for a role of internal models in the generation of motor commands for voluntary arm movements and eye movements (Jordan 1996; Kawato and Gomi 1992; Wolpert 1997). There is now considerable evidence for the existence of internal models guiding limb movements, that these models are established during the course of normal development, and that they can be rescaled in response to changes in internal and external conditions (Wolpert 1997; Wolpert et al. 1995). With this view it is conceivable that the increase in the early component of the MG EMG that occurs after cutting the LGS and PL nerves is due to a rescaling of an internal representation of stiffness at the ankle joint. We can only speculate on the location of this internal representation and the mechanisms for rescaling. One possibility is that the representation is in the spinal cord and that this representation is modified either directly by persistent alteration in afferent signals or indirectly by phasic and/or tonic supraspinal signals. Another possibility is that stiffness is represented in supraspinal regions, and signals from these regions modulate the spinal system generating the initial component of MG activity.

Finally it is of interest to consider which signals could be responsible for regulating the rescaling of the initial component of the MG EMG. An obvious possibility is an enhanced activation of proprioceptors in the MG muscle during the period of abnormally large yield soon after ground contact. The additional stretch of the isolated MG muscle would probably strongly activate muscle spindles, and presumably there would be an increase in force in the muscle thus increasing activity in Golgi tendon organs. Of these two groups of receptors, the muscle spindles are the most likely candidates for driving the rescaling of the initial component because their activity would reflect the error in magnitude of ankle yield. Soon after the nerve transections, feedback from spindles would be high due to the large increase in stretch (from ~2 to 7 mm), but as the yield decreases with time, this feedback would be reduced. On the other hand, activity in the Golgi tendon organs would not necessarily decrease, and may even increase, during the functional recovery because the force in the MG muscle probably increases over this period. Thus our current working hypothesis is that phasic sensory feedback from the MG muscle spindles during stance acts as an error signal to rescale the magnitude of the initial, centrally generated component of the MG EMG. This hypothesis is similar to the scheme proposed by Kawato (1996) for the scaling of motor commands for voluntary arm movements.

The obvious way to test this hypothesis is to eliminate feedback from muscle spindles and determine whether the increase in the initial component still occurs. Unfortunately, there is not a simple procedure by which this can be achieved. Nevertheless, our finding that immobilizing the leg prevented the increase in the initial component of the MG EMG (Fig. 10) is consistent with the hypothesis. However, it is also consistent with other hypotheses, one being that the magnitude of the initial component is regulated by more global sensory signals related to the abnormal posture and movement of the hindquarters during stance. In this case the increases in the magnitudes of the initial and late components of the MG EMG may be entirely independent phenomena, yet both depend on the use of the leg. It is imperative, therefore, to examine more closely the relationship between the changes in feedback from muscle proprioceptors and the changes in the initial component.

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ADAPTIVE CHANGES IN WALKING MOTOR PATTERN


