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Activity of Spindle Afferents From Cat Anterior Thigh Muscles. II. Effects of Fusimotor Blockade

G. E. LOEB AND J. A. HOFFER

Laboratory of Neural Control, IRP, National Institute of Neurological and Communicative Disorders and Stroke, National Institutes of Health, Bethesda, Maryland 20205

SUMMARY AND CONCLUSIONS

1. Chronically implanted electrodes and nerve cuff catheters were used to record the activity of individual muscle spindle afferents during treadmill walking as low doses of lidocaine were infused around the femoral nerve to progressively block gamma motoneuron activity.

2. Both primary and secondary endings from both the monarticular knee extensors and the biarticular hip/knee muscles of the anterior thigh showed large decreases in afferent activity, usually well before changes in the electromyographic activity, force output, or length and velocity were seen in the parent muscles.

3. This decline in the proprioceptive signal feeding back onto the spinal cord, which we presume to have involved most of the spindles supplied by the femoral nerve, did not cause noticeable irregularities or instability of the walking gait.

4. At the peak of the fusimotor blockade, spindle afferents from knee extensor muscles lost about half of their usually brisk activity during the near-isometric contraction of the stance phase. Significant decreases in the response to passive stretch during the flexion phase also occurred.

5. At the peak of the fusimotor blockade, spindle afferents from the biarticular muscles lost all of their activity during the rapidly shortening swing phase and about half of their activity during the rapidly lengthening stance phase.

6. For both monarticular and biarticular muscle spindles, the activity decreases in stance and swing phase often occurred at distinctly different stages of the progressive fusimotor blockade, indicating several different sources of fusimotor control.

7. From these data, we infer that the sensitivity of most spindle afferents is substantially influenced by fusimotor activity during phases of both extrafusal activity and extrafusal silence. At least some of this influence appears to come from fusimotor neurons whose recruitment is independent of the extrafusal recruitment. The extent and type of fusimotor effects on spindle afferent sensitivity (dynamic or static) appear to be specialized for the mechanical events that tend to occur during those phases.

INTRODUCTION

The role of the intrafusal muscle fibers in mammalian motor control has been a controversial and technically difficult research problem. On one hand, their efferent innervation is derived from gamma and beta motoneurons that are at least anatomically closely related to the pools of alpha motoneurons responsible for the mechanical action of the extrafusal muscle. On the other hand, the intrafusal muscle tension is insignificant to the work of the muscle but can completely dominate the activity of the proprioceptive sense organs known as muscle spindles. It remains unclear whether the fusimotor apparatus functions in close synergy with the extrafusal muscle or whether it may be recruited independently to provide unrestricted control of sensory transduction, largely because records of the normal activity of the fusimotor neurons have been almost impossible to obtain.

Interpretation of records from muscle spindle afferents of the cat hindlimb during normal locomotion has led to inferences about fusimotor activity by qualitative (16, 20) and, recently, by quantitative (14) comparisons of the afferent activity with that generated by similar movements applied to muscles that are either deefferented or subjected to electrically controlled patterns of fusimotor stimulation. However, there are at least three distinctly different intrafusal muscle fibers under more or less independent control by different types of fusimotor neurons (2, 4). Their individual effects on afferent activity are complex and nonlinear, and their combined effects interact nonlinearly with each other and with the particular movements being applied (13). The conclusions of these simulation experiments (14) are somewhat in conflict with the activity recorded directly from gamma motoneurons in decerebrate preparations making walking movements (17b), but it is not known how normal the fusimotor recruitment is in decerebrate preparations.

Another way to assess fusimotor contribution to spindle activity is to block fusimotor neurons during performance of the behavior under study. Critchlow and von Euler (5), in their study of spindle afferent activity during respiration in anesthetized cats, made use of the differential sensitivity of small diameter gamma motoneuron axons to be blocked from conducting action potentials by low concentrations of lidocaine infused around the intercostal nerves. We have adapted this technique to studies in awake, walking cats by incorporating a percutaneous catheter into a nerve cuff implanted around the femoral nerve. Progressive infusions of low concentration lidocaine solution caused significant changes in the activity of most spindle afferent units, recorded by floating electrodes in the fifth lumbar dorsal root ganglion (L_5 DRG). Changes in afferent firing usually occurred well before the unit became completely silent (which would indicate block of the afferent axon) and before the onset of significant changes in the limb motion, force output, or EMG of the parent muscles (which would indicate α-motoneuron block), permitting strong inferences about the extent and nature of fusimotor activity under unblocked conditions. A preliminary report of the techniques has been published previously (10).

MATERIALS AND METHODS

The detailed fabrication, implantation, and characteristics of the implanted recording devices are described and/or cited in a companion paper (17). Briefly, each of eight adult male cats was surgically implanted under general anesthesia and aseptic conditions with an external connector assembly that permitted access to the various implanted devices listed in Table 1. After recovery from surgery, the animals were exercised daily by walking on a treadmill while they were connected to electronic recording equipment and videotaped. Unit activity was recorded from one or more of the implanted floating microelectrodes in L_5 DRG. The origin and conduction velocity (CV) of afferents was determined by spike-triggered averaging of two neurograms recorded by a multipolar nerve cuff chronically implanted on the femoral nerve, by using the latency and physical distance between them to determine conduction velocity (11). The modality and location of the receptor was determined by various manipulations under anesthesia described in the companion paper (17), including passive motion of the joints and application of a vibrating probe.

During the walking period, small incremental doses of various dilutions of lidocaine or normal saline (NS) were infused into the lumen of the femoral nerve cuff via its percutaneous catheter, while

TABLE 1. *Implanted devices and identifying abbreviations for figure traces*

DRG	Floating microelectrode in the dorsal root ganglion
FP	Femoral nerve cuff, proximal tripolar electrode site
FD	Femoral nerve cuff, distal tripolar electrode site
VI	Vastus intermedius EMG (intramuscular multipolar)
VM	Vastus medialis EMG (intramuscular multipolar)
VL	Vastus lateralis EMG (intramuscular multipolar)
RF	Rectus femoris EMG (intramuscular multipolar)
SA-a	Sartorius pars anterior EMG (bipolar patch)
SA-m	Sartorius pars medialis EMG (bipolar patch)
L_V	Length of vasti muscles (stretch upwards)
$\dot{L}_V$	First derivative (velocity) of L_V
L_R	Length of RF and SA-a muscles (stretch upward)
$\dot{L}_R$	First derivative (velocity) of L_R
F_P	Force at patellar ligament strain gauge
T	Treadmill tachometer (5 cm/tick)
Stance	Ipsilateral foot contact with treadmill (from videotape)

the spindle afferent activity was monitored (see figures for dosage schedules). In most cases, the spindle afferent activity underwent one or more abrupt changes in its pattern until either it ceased completely (indicating blockage of conduction in the afferent fiber itself) or the walking became markedly abnormal as a result of muscle weakness (indicating blockage of a significant number of alpha motoneurons). Abnormal gait could represent a manifestation of reduced spindle afferent drive to motor or interneurons, and some subtle effects discussed below may be consistent with this hypothesis. However, major gait changes were observed only after much higher doses of lidocaine than were necessary to cause most of the change in the afferent activity being monitored. In most cases, the spindle afferent was monitored until its activity returned to control levels, which varied from minutes to hours. In all cases, the animal appeared to suffer no discomfort and no cumulative or residual abnormalities in motor behavior were noted as a result of performing such nerve blocks on successive days. A postmortem examination of one of the nerve cuff assemblies using blue dye to trace the flow of a typical volume of infusate revealed that it tended to flow in narrow, elongated, and tortuous paths around the nerve within and outside of both ends of the cuff electrode. No significant amount of the infusate appeared likely to have traveled far enough to influence the conduction in any other major nerve trunk beside the femoral nerve. The initial flushing volume of normal saline often led to a temporary increase in the level of block because it tended to push the column of lidocaine solution in the catheter and cuff (~0.3 ml) into fresh regions of the nerve.

In two animals, the activity of muscle spindle afferents during lidocaine block was compared with that during similar limb motions applied to the limb by a servo-controlled torque motor during deep pentobarbital anesthesia, which blocks all spontaneous activity in all intra- and extrafusal motoneurons. The earlier-recorded length gauge record was used as the drive signal to the torque motor controller.

The EMG records from the various muscles shown in these figures have been processed in two different ways. When identified with only the abbreviation of the muscle, these records have been full-wave rectified and integrated into 2 ms wide bins by using a pulsed sample and hold device described previously (1). When the abbreviation is preceded by an integral sign, these records have been full-wave rectified and integrated by a Paynter filter (three-pole Butterworth) with a 50-ms time constant (6). All unit activity is shown as instantaneous frequencygrams (inverse of the time interval between two successive spikes).

RESULTS

Lidocaine-induced changes in spindle activity occurred in all of the 14 units for which the procedure was attempted. In two of these units, the activity of the afferent ceased entirely before less drastic changes were noted, presumably as a result of afferent conduction block. The results reported here summarize the progressive effects seen in 12 afferents from 7 different cats, including 4 afferents from the vasti muscle group (all primary endings) and 8 from the sartorius muscle complex (6 primary and 2 secondary endings). Typical records from eight of these afferents at various stages of fusimotor blockade are shown in the following figures.

Vasti muscles

Figure 1 shows a complete sequence consisting of several stages of change in the activity of muscle spindle primary T9A22 (CV = 111 m/s) from prelidocaine control (*A*) to nearly complete return to control (*F*). Some changes in the extrafusal pattern of locomotion can be seen at various stages including progressively greater yielding at the knee joint (upturn of trace L_V and slower rate of rise of trace F_P during stance in stages *B* through *E*) as well as complete blockade of the medial sartorious EMG (trace SA-m, stages *D* and *E*). The decline in SA-m EMG started very early in the infusion, along with the first change in spindle activity (stage *B*). This was not a consistent finding and probably relates to the vagaries of flow in the nerve cuff with respect to a clustered fascicle of SA-m alpha motoneurons.

The spindle primary showed a large reduction in the swing phase bursts (stages *B* and *C*) accompanied by a paradoxical increase in the already high stance phase activity (stage *B*). The normal swing phase activity was highly velocity dependent (see also Fig. 1 of previous paper), and this reduction suggests removal of gamma dynamic activity. The increased stance phase activity occurred mostly in mid to late stance and may reflect the absence of the active muscle shortening that occurs normally in this phase. In stage *C*, the stance phase activity also decreased and became much less modulated, with an envelope of activity looking very much like the extrafusal VM EMG pattern. The reduction in stance phase spindle activity from

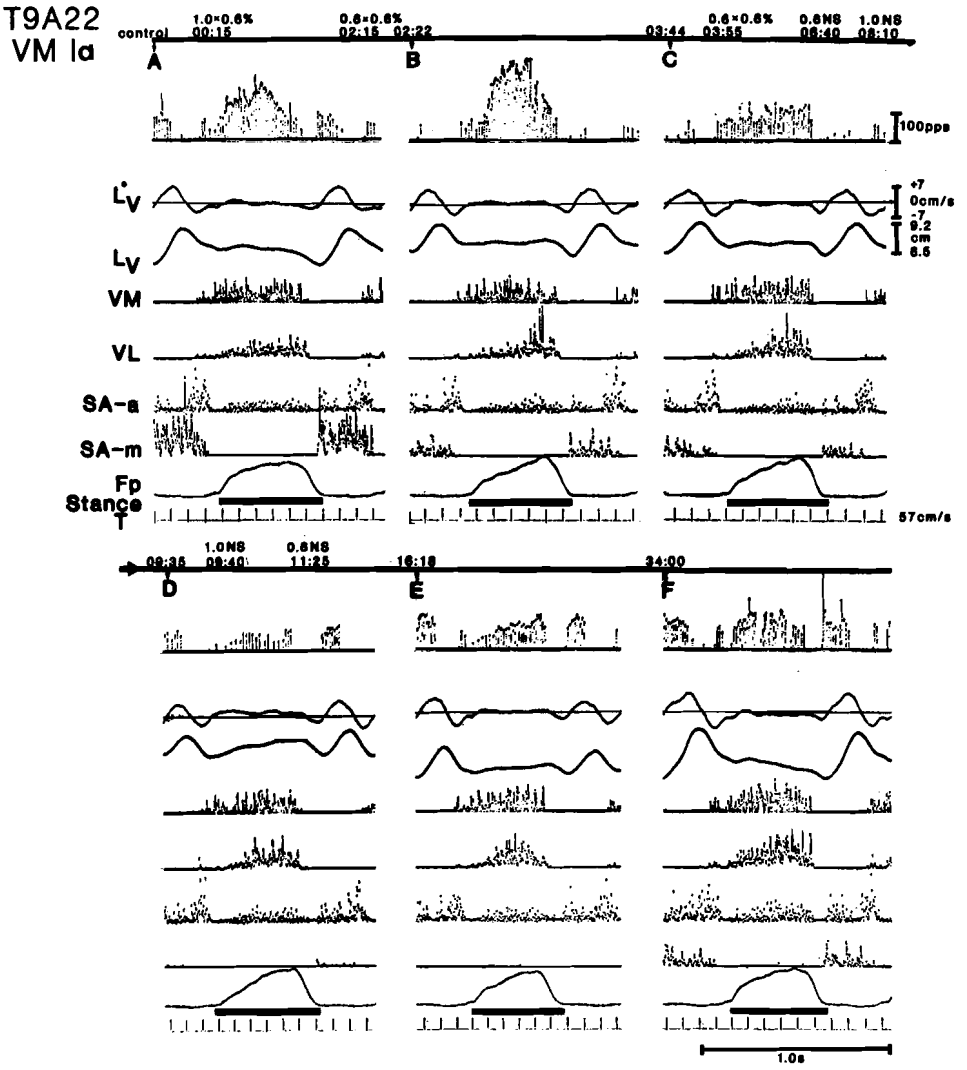

FIG. 1. Spindle primary afferent, CV = 111 m/s, from vastus medialis muscle (see identification data in Fig. 3 of the previous paper). See Table 1 for identification of traces. Time line at top indicates time in minutes and seconds at which records *A* through *F* were obtained as well as the timing of doses of lidocaine (number of milliliters times percentage concentration) and flushing with normal saline (milliliters NS). The peak of the blockade occurred at stage *D*, where significant yielding at the knee and loss of the medial sartorious EMG (SA-m) can be seen. Note that swing phase spindle activity changed in phase *B* and was returning by phase *D*. Recovery was nearly complete by stage *F* at 34 min.

B to *C* despite no apparent further changes in the length trace suggests removal of some fusimotor drive, possibly of the static type. This appears to have occurred again in stage *D*, where the stance phase activity was further reduced despite rather marked yielding, further stretching the parent muscle. Interestingly, at this stage most of the swing phase activity had returned to control values, with a strong, velocity-dependent response. Through stages *E* and *F* there were progressive small changes in both the swing and stance phases of spindle activity, whose interpretation is complicated by ongoing recovery of the extrafusal system as well.

There are two, not necessarily exclusive, interpretations of the yielding seen in the vasti muscles during the stance phase. First, it is

possible that some of the extrafusal motoneurons were blocked, resulting in an unexpected weakness that was somehow detected and compensated after some delay with further recruitment of the knee extensor motor pool. Second, it is more likely that the normal recruitment of the knee extensor pool was dependent on a contribution from the homonymous spindle afferent feedback, which had been reduced by the blockade of fusimotor fibers. The amount of feedback would fall short of normal levels until an abnormally large yielding had further stretched the partially deefferented spindles. In both cases, we might expect to see primarily timing rather than amplitude changes in the knee extensor EMGs, such as the subtle delay in onset and mid to late stance peaking of VL EMG seen in traces *B–E*. The paradoxical increase in spindle activity in *B* is actually consistent with either hypothesis because it may represent one of the few spindles not yet directly affected by fusimotor blockade, at a time when the muscle was failing to contract normally because of either alpha motoneuron blockade or loss of afferent feedback from other deefferented muscle spindles.

Figure 2 shows the activity of another spindle primary from vastus medialis (CV = 109 m/s). The animal was only six days postop-

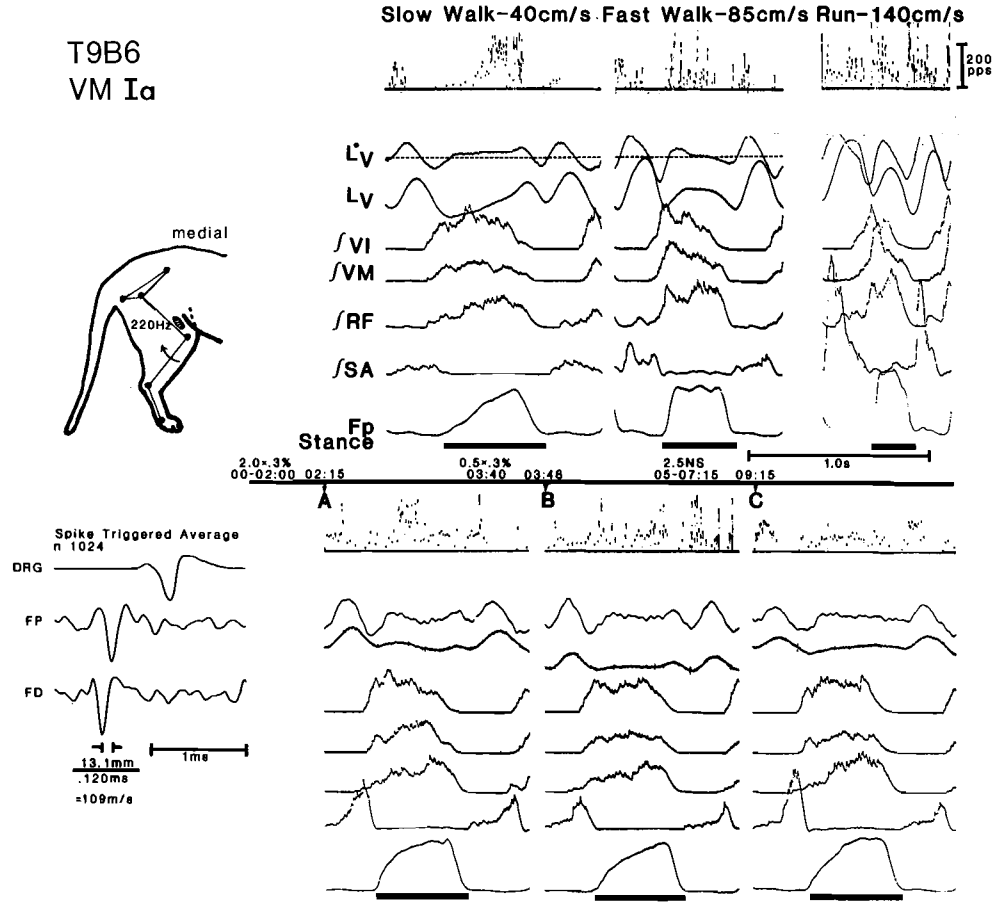

FIG. 2. Spindle primary afferent, located in vastus medialis based on sensitivity to manipulation and vibration (upper left), CV = 109 m/s based on spike triggered averaging from femoral nerve tripolar electrodes (*lower left*). Top set of traces shows activity for various control gaits prior to lidocaine administration. Time line over bottom set of traces shows stages of fusimotor blockade *A*, *B*, and *C*, with timing of lidocaine and saline (NS) flushes.

erative when these records were obtained, and it had some variability in its gait, particularly when walking more slowly than normal (40 cm/s; usual preferred walking speed is ~60 cm/s). However, the control traces at top have generally similar features to those of Fig. 1, including a period of high activity during the stance phase even when isometric (fast walk) and velocity-dependent activity during the swing phase (note unit activity increase as gait speeds and velocity of flexion phase muscle stretch increased). Lower doses of lidocaine were used, resulting in relatively little change in the extrafusal activity (see force, EMG, and length traces in stages *A–C*, all ~40 cm/s slow walk). The main change in spindle activity was ~50% reduction in the mid to late stance phase activity, which also became more regular in its pattern. Note also the disappearance of the initial stance phase burst normally related to muscle yielding at footfall (trace *C* compared with *A;* not seen in slow walk control unit records because of more tentative foot placement, as seen in L_V and F_p).

Figure 3 shows the activity of an unusual spindle primary afferent located in the vastus lateralis muscle. This unit was remarkable for having very little activity during the stance phase in the control state (*A*), despite the usual velocity-dependent response during the swing phase. Lidocaine dosage sufficient to cause significant yielding during the stance phase (traces L_v and F_p in stage *B*) caused no change in spindle activity during swing. The increased muscle stretch during stance appeared to increase the activity of the spindle afferent.

The pattern of motion of the knee joint given by trace L_V in stage *A* was used to drive

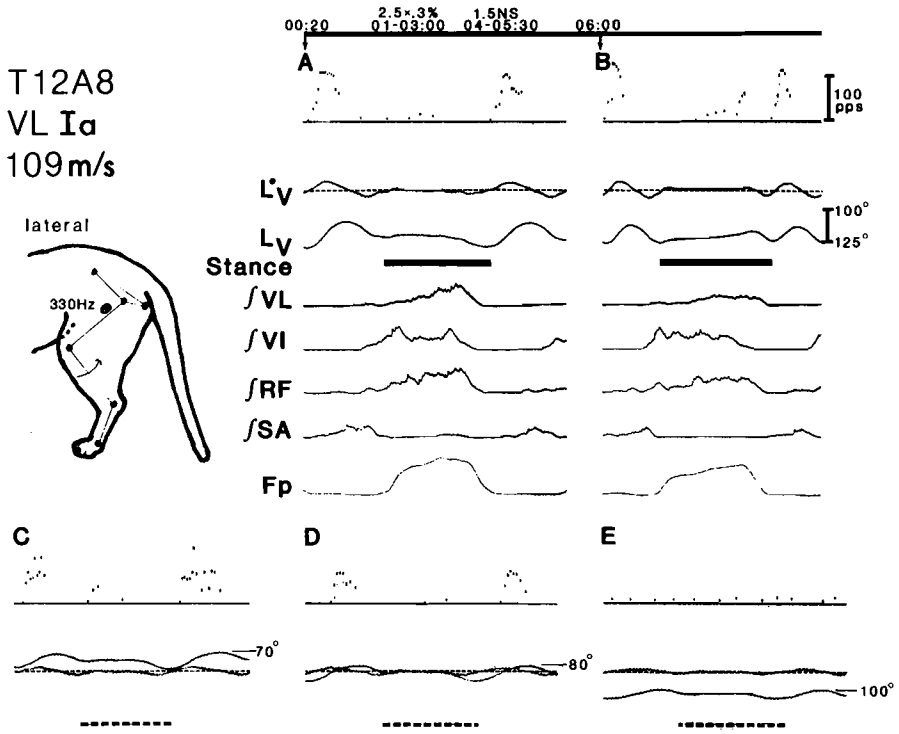

FIG. 3. Spindle primary afferent from vastus lateralis. Control walking in *A;* little change in *B* at height of lidocaine blockade. Length gauge across vastus muscles (L_V) calibrated in degrees of knee extension; note yielding during stance phase of *B*. Records *C, D,* and *E* obtained during deep pentobarbital anesthesia, with motion of knee joint applied by torque motor (calibration shows point of maximal knee flexion). *Fine dashed line* indicates zero reference for velocity trace L_V in all records; *coarse dotted line* in *C–E* indicates periods corresponding to stance in the motion applied to the limb. *C–E* represent similar temporal patterns of such motion superimposed on progressively more extended knee angles, with *E* closest to the normal walking trajectory seen in *A*.

a torque motor (panels *C–E*) that moved the limb during deep barbiturate anesthesia. The spindle afferent activity in trace *C* looks rather similar to that in control *A*, but the motion was superimposed on a markedly flexed limb position (see calibrations expressed in knee joint angle). When the limb was shifted to a more physiological walking posture, the spindle reverted to a low frequency discharge that was virtually unmodulated by the applied length changes (*E*) (although these were unfortunately attenuated by the poor following characteristics of the torque motor employed). It seems possible that the spindle sensitivity noted in *A* and *B*, which by itself offers no suggestion of fusimotor drive, was dependent on some tonic bias contributed either by intrafusal activity that we were unable to block or by some tonus of the extrafusal muscle that was eliminated by the deep anesthesia (the vasti are pinnate muscles with a long, elastic insertion tendon distributed within the muscle mass).

Sartorius muscle complex

The normal activity of different spindle afferents from the bifunctional sartorius muscle was noted to be quite heterogeneous (see Ref. 17) with significant differences in the relative amounts of activity occurring during stance phase lengthening and swing phase shortening, respectively. However, these spindles all behaved much more similarly at the peak of the fusimotor blockade, suggesting that much of this heterogeneity arises from fusimotor influences rather than intrinsic mechanical properties.

Figure 4 shows a spindle primary (CV = 119 m/s) in the anterior part of the sartorius muscle that had several complex features in its typical activity pattern (as seen in the control stage *A*). The general level of activity was about equally high during both swing and stance for moderate walking at 50 cm/s. There were two brief but consistent bursts of a few high-frequency spikes apart from the main envelopes. One occurred just at the beginning of the swing phase (solid arrows) and appeared to be at least temporally correlated with a slight pause at the onset of the muscle shortening (which can be seen as a small positive deflection in the generally negative velocity at this point). The other was a somewhat more diffuse burst at the beginning of the stance phase (open arrow), just as the yielding at the knee began to stretch the muscle while the hip had passed to the point of maximal flexion.

The first change following lidocaine was a marked reduction in the stance phase activity with relative preservation of the swing phase activity (stages *B* and *C*). At the height of the block in stage *D*, most of the swing phase activity also disappeared. The stance phase activity became quite low and regular, except for the previously noted tendency to burst at the beginning of stance (open arrows). Ten minutes after the normal saline flush (stage *E*), both the stance and swing phase activity had returned to ~50% of normal. Throughout the various stages, the stance phase activity showed variations that seemed at least loosely correlated with small inflections in the velocity of the stretch. The swing phase activity seemed to be correlated with the time course of the swing phase EMG in the anterior sartorius muscle, which was somewhat reduced during the lidocaine blockade and showed irregular bursts during the recovery (stage *E*). The hip and knee movements remained almost unchanged throughout, including the particular bumps in the velocity trace associated with the two brief spindle bursts at the arrows. The slow rate of force development in stage *C* was probably related to the long stance phase rather than weakness (note lack of excess yield in the L_V trace); the record was selected because it shows the early stance phase burst (open arrow) clearly separate from the bulk of the much attenuated stance phase activity.

Figure 5 shows another spindle primary in anterior sartorius, which had a similar pattern of velocity-dependent stance phase activity (note increase as walking speed increased) and maintained activity during swing phase regardless of velocity of shortening. This unit also had a burst at the beginning of stance, which tended to fuse with the end of the swing phase activity (solid arrows). As the lidocaine blockade progressed, the swing phase activity decreased and finally ceased entirely (stages *A–C*). This revealed the independence of the early stance phase burst (solid arrows, *A* and *B*), which itself disappeared at the height of the block (open arrows, stages *C* and *D*). The main stance phase activity was at most slightly affected at the height of the block, with the

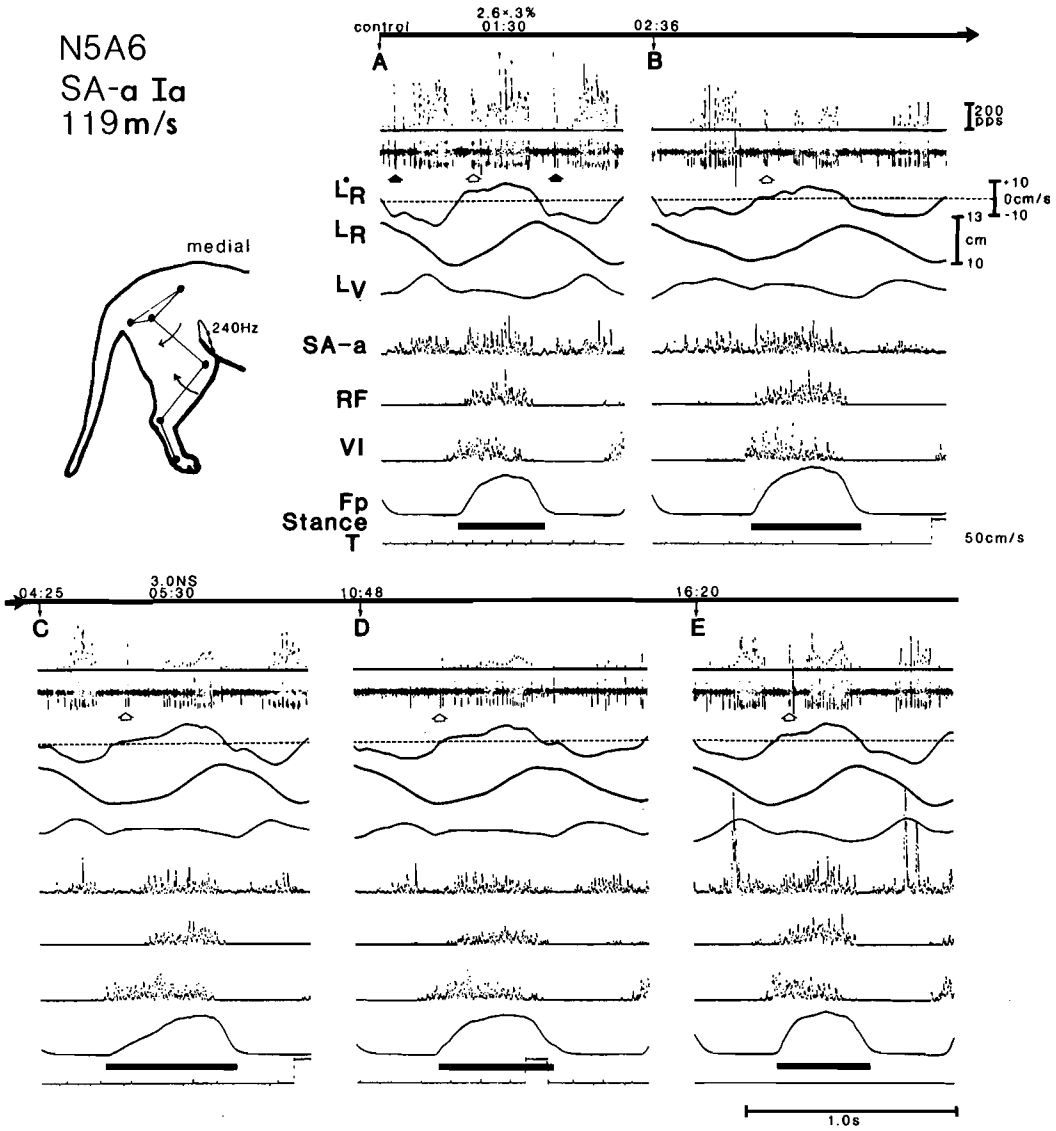

FIG. 4. Spindle primary afferent from anterior sartorius. Control walking in *A*, various stages of fusimotor blockade in *B* through *E*. Raw microelectrode trace shown under unit frequencygram; see text for explanation of *arrows*.

same peak firing rate confined to the later part of the stance when the muscle was at its longest (stage *C*).

Figure 6 shows two simultaneously recorded afferents from a single microelectrode. The larger spike, I9A17, proved to be from a secondary ending in medial sartorius, with a 67 m/s conduction velocity. The smaller spike, I9B17, was from a primary ending (CV = 109 m/s) in anterior sartorius. The spindle primary was qualitatively similar in its behavior to those discussed previously, although it generated much more of its normal activity during the stance phase lengthening than during the swing phase shortening. The stance phase activity occurred at the peak of the velocity of lengthening, although it was not increased beyond its already quite high levels when the speed of gait was increased from 50 to 95 cm/s, going from a walk to a trotting gait. The secondary ending had lower levels of activity during stance and late swing (solid arrow). Be-

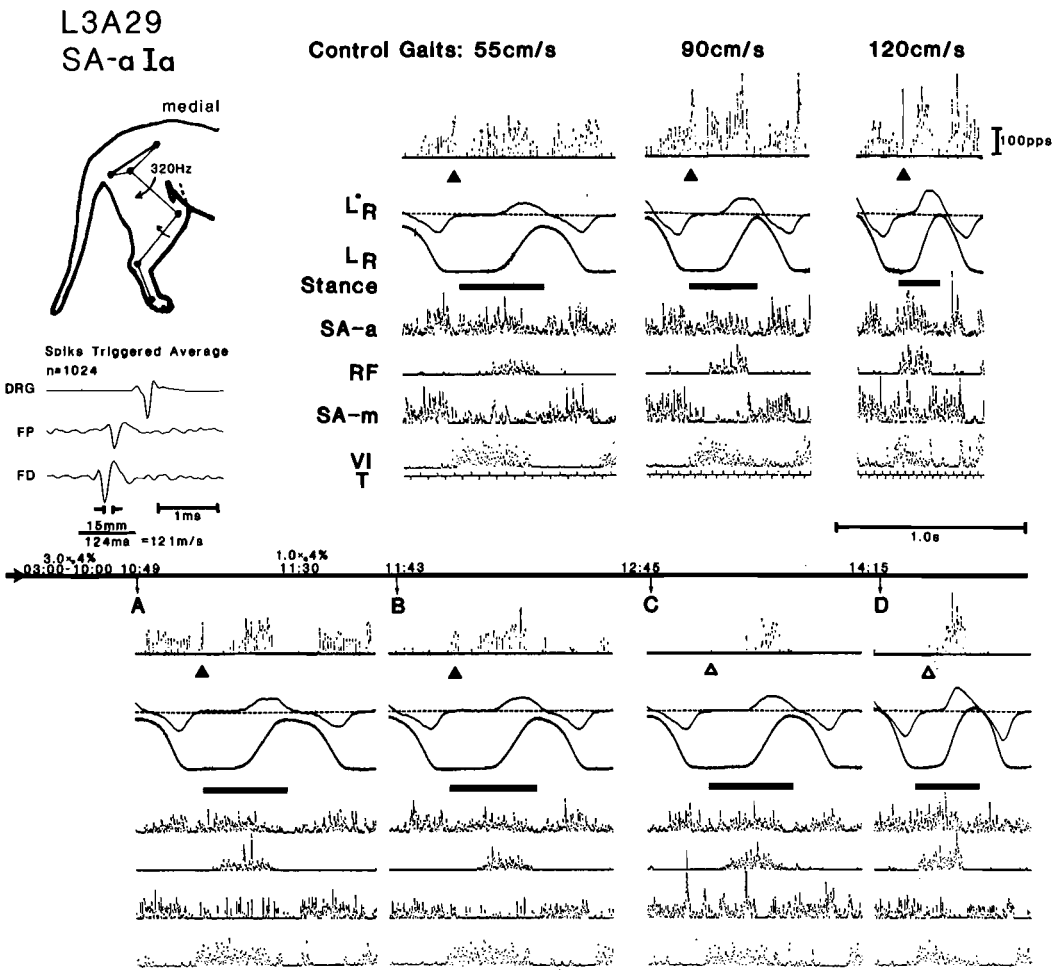

FIG. 5. Spindle primary afferent from anterior sartorius muscle, with spike triggered averaging from femoral nerve cuff records indicating CV = 121 m/s. Three control gaits at top show slow walk (55 cm/s), fast walk (90 cm/s), and trot (120 cm/s). Traces *A* through *D* at bottom show activity during slow walk at various stages of lidocaine blockade. Unusual amount of stance phase EMG in SA-m probably indicates cross talk. Flattened bottoms of length trace L_R and associated zero velocity in $\dot{L}_R$ are the result of the gauge becoming slack at the shortest extremes of its excursion.

cause this ending was located in the medial sartorius, it is likely that both periods of activity were correlated with muscle lengthening, as the E_1 phase knee extension would have stretched this part of the muscle significantly. This may account for the large increase in this burst during trotting (solid arrows). This important difference between the mechanical input to these two proprioceptors is best seen at the open arrow in the record marked "pounce," where the animal made a series of quick, exaggerated motions while chasing a toy. The burst of the anterior sartorius spindle afferent occurred during the knee flexion, whereas the burst of the medial sartorius spindle followed during the knee extension (solid arrow).

The effect of the progressive fusimotor blockade on the primary ending in anterior sartorius (top trace in stages *A* and *B*) was first to reduce the stance phase activity to about one-third and then to eliminate the swing phase activity. The secondary ending in medial sartorius lost the late swing (E_1) burst and had somewhat reduced activity in the remainder of stance (stage *A*). The E_1 activity was beginning to return (solid arrows, stage *B*) when the unit stopped all activity (stage *C*), probably as

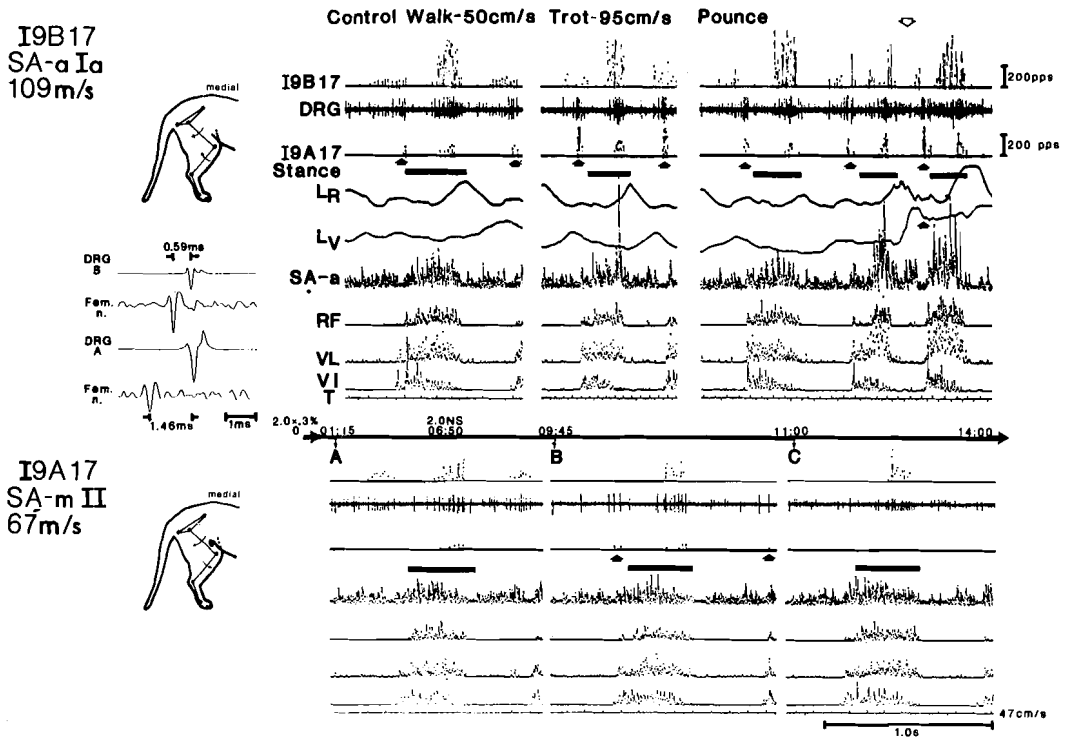

FIG. 6. Two simultaneously recorded spindle afferents from the same DRG microelectrode. Unit I9B17 (smaller of 2 unit potentials) was a spindle primary from anterior sartorius, CV = 109 m/s. Unit I9A17 (larger of 2 unit potentials) was a spindle secondary from medial sartorius, CV = 67 m/s. Spike-triggered average records at left show only averaged potentials from single femoral nerve cuff locations; actual conduction velocities were obtained from incremental latency between proximal and distal femoral nerve cuff recording sites. Three control records at top show walk (50 cm/s), trot (95 cm/s), and a sequence of accelerating steps ending with an exaggerated knee flexion (*open arrow*) just before the forepaws landed on a toy that the animal was chasing. Traces *A–C* below show activity of both units (during 47 cm/s walking) at various stages of lidocaine blockade. Activity of both units returned to near control levels at 14 min (not shown). Length gauge was functioning poorly in the control period and failed completely during lidocaine infusion; videotaped gait appeared grossly normal throughout.

a result of direct blockade of the afferent axon itself. Both units returned to near-control activity at 14 min (not shown). The EMG patterns during the blockade were essentially unchanged. Unfortunately the various transducers became progressively more noisy from their already poor performance in the control traces, and did not provide good records of the details of the movement, which appeared grossly normal on the videotape.

DISCUSSION

The method of fusimotor blockade used in these experiments depends on the differential sensitivity of the smaller diameter fibers to critical concentrations of lidocaine to permit the afferent itself to continue conducting and the alpha motoneurons to continue activating the muscles to walk. Several factors must be kept in mind in interpreting these results: *1*) The exact distribution of the lidocaine solution and its uptake by the femoral nerve was probably patchy and irregular, so that blockade would not proceed in a precisely size-ranked order. Deep-lying fibers would be relatively less affected than superficial fibers of the same caliber. *2*) In addition to blocking gamma motoneurons directly, the lidocaine probably first blocked most ongoing activity in smaller diameter group III and group IV afferents, at least some of which are known to have influences on gamma motoneurons (18). *3*) Some fusimotor activity was probably the result of

beta motoneurons (3), whose fiber sizes overlap those of both alpha motoneurons and the spindle afferents themselves (particularly the secondary endings), and some of which probably were not blocked. *4*) The animal could easily compensate for at least some blockade of the smaller alpha motoneurons by increasing the recruitment of larger motoneurons which produce larger forces. This might not be detectable on EMG, force, or length records, but might change the distribution of small internal movements due to local elasticity.

It is likely that even at the height of gamma blockade reached in these experiments, many spindles might well have had residual fusimotor activity. This may account for a further decrease of sensitivity noted during barbiturate anesthesia in Fig. 3. However, there was a clear general tendency for spindle activity to be significantly reduced and to take on patterns that were more homogeneous and consistent with the known properties of deefferented spindle afferents (see Refs. 7, 8).

It is noteworthy that even at the height of the fusimotor blockade, the animals had no significant instability or unsteadiness of gait. The only changes noted were consistent with a partial block of and/or lowered feedback to alpha motoneurons, with yielding of the weakened antigravity knee extensors. It is likely that the blockades affected most of the saphenous nerve cutaneous afferents, the majority of the median articular nerve joint receptors, and a significant percentage of spindle secondary endings, as well as causing a quantitative reduction and qualitative change in spindle primary activity from the muscles that provide virtually all of the active knee extension and most of the hip flexion. Of course, the locomotor pattern generator can function independently of all proprioceptive input in fictive preparations (19). However, our animals were walking unsupported on a belt moving at a fixed speed, and apparently were able to make use of the available proprioceptive information from other sources to correct their motor output to compensate at least adequately for changes such as the weakness induced by the partial motoneuron block. As has been noted before in deafferentation experiments (for review see Ref. 22), more demanding tasks such as walking on a narrow beam may be required to reveal functional deficits.

These experiments point out the large qualitative and quantitative influence of the fusimotor system on all of the spindles studied, including both those with clearly "active" responses during muscle shortening as in hip flexors and those which might have been called "passive" as in the knee extensors. The fractionation of the blockades into several different stages with isolated effects on a particular phase of spindle activity is consistent with the known multiplicity of independent gamma and beta motoneurons innervating each spindle (3). Furthermore, it suggests that the different phases of each muscle's motion may be subject to separate and independent adjustment of the instantaneous spindle sensitivity to each motion. In the previous paper (17), we noted that different types of fusimotor influence—dynamic versus static—seemed to be required to account for the similarity of spindle afferent firing rates observed at kinematically different points in the step cycle. This is consistent with the sequential appearance of discrete changes in particular phases of afferent activity as the blocks progressed (see Figs. 1, 4, 5, and 6).

The vasti muscle spindles provide similar evidence for separate fusimotor programs in stance and swing even though the muscles are extrafusally silent during most of the swing phase (see Fig. 1). The stance phase gamma activity may well have been temporally correlated with the ongoing extrafusal activity, although it does not seem particularly useful to invoke the term *alpha-gamma coactivation* in view of the swing phase fusimotor influences. It would be difficult to ascribe the stance phase fusimotor influence to any particular mix of static and dynamic effects because of the uncertain direction and magnitude of motion transmitted to the spindles in this pinnate muscle under near-isometric conditions. The larger swing phase excursions seemed to be accompanied by enhanced dynamic sensitivity.

In the sartorius muscle complex, the fractionation of fusimotor activity into separable swing phase and stance phase effects on afferents is more readily understood in light of the fractionation of the alpha motoneurons into task groups. The two bursts of EMG activity

in the anterior sartorius occurring during stance and late swing, respectively, appear to be caused by separate alpha motoneurons (12). This might provide some basis for invoking two separate "coactivated" groups of gamma motoneurons: the dynamic enhancement of spindle afferent sensitivity seen during stance phase lengthening could be the result of gamma dynamic motoneurons recruited with the stance phase alpha motoneuron group, whereas the sustained spindle activity during swing phase shortening could represent gamma static motoneurons recruited with the swing phase alpha motoneuron group.

Previous attempts to preserve the coactivation hypothesis as a general rule have postulated tonically activated (unmodulated) fusimotor activity and residual fusimotor effects from slow relaxation (20) to account for the apparent facilitation of afferent activity during periods of extrafusal silence. Although these experiments do not rule out either effect, they also suggest that most of the fusimotor effects at any time may be specifically controlled by gamma and beta motoneurons (e.g., see Fig. 1, stage *B*).

These experiments provide only a lower bound to the number of separately controlled aspects of muscle spindle sensitivity. However, they clearly exceed the degrees of freedom evident in the much more simple and orderly recruitment of the alpha motoneurons. The size principle (9), which so greatly simplifies the central command signals needed to operate the mechanical output of the muscles, cannot be extended usefully to explain the operation of the sense organs located within them. The mammalian muscle spindle has evolved a complex and mechanically independent set of elements for controlling sensitivity and dynamic range (see Ref. 15). It would appear that the central nervous system takes advantage of this feature.

In the previous paper, we advanced the hypothesis that the fusimotor system is used to try to maintain the spindle afferent firing rates within a relatively narrow range (50–200 pps for primaries, somewhat lower for secondaries) that was "optimal" in terms of resolution and sensitivity. This optimization involves two major considerations: *1*) the avoidance of saturated states (where firing rates are too low for rapid assessment of changes or too high for reliable conversion of generator potentials to smoothly modulated spike rates; see Ref. 17a) and *2*) the avoidance of insensitive states (where the range of perturbations to be detected produces only small modulations in spike frequency). These boundaries are not rigid but are somewhat analogous to the corner frequencies of a first-order filter, past which frequencies the output begins to decline gradually. The second consideration indicates why it would be useful for the system to operate at least occasionally outside the preferred range. There may also be limitations in the speed with which fusimotor influences can be reprogrammed (e.g., slow relaxation time of some intrafusal muscle fibers), which may force the sensory apparatus to go through some "down time." This may account for occasional silences in afferent activity at the transitions between stance and swing phases in some of these records.

The results of the experiments described in this paper indicate that the generally well-modulated behavior of the muscle spindle afferents during normal use is not a quirk of their passive properties, their mechanical linkages in the muscle, or any "simple" fixed recruitment pattern for alpha, beta, and gamma motoneurons. It is, indeed, the result of multiple independent sources of static and dynamic fusimotor modulation.

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