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Response Patterns and Postspike Effects of Peripheral Afferents in Dorsal Root Ganglia of Behaving Monkeys

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SUMMARY AND CONCLUSIONS

1. The activity of single afferent units was recorded in cervical dorsal root ganglia (DRG) in two macaque monkeys as they generated alternating flexion and extension torques about the wrist during a step-tracking task. During these isometric and auxotonic muscle contractions, electromyographic (EMG) activity was recorded with electrode pairs in up to 12 independent forearm muscles. Spike-triggered averages (STAs) of rectified EMG activity were used to identify afferents that were associated with correlated facilitation of active muscles.

2. Our aim was to find peripheral afferents producing postspike effects in muscles and to compare their properties with those of corticomotoneuronal (CM) and rubromotoneuronal (RM) cells previously obtained under identical behavioral conditions. We documented the timing, magnitude and distribution of their postspike facilitation (PSF) of forearm muscles and investigated the response properties of task-related units.

3. Of 125 afferent units tested with STAs, 68 showed PSF of EMG activity in at least one muscle. Fifty-nine DRG units provided sufficiently long recordings to generate averages with ≥2,000 triggers, the minimum number considered to demonstrate reliable effects. Of these 59 units, 29 (49%) were associated with facilitation of forearm muscle activity.

4. Many STAs showed a gradual increase in EMG activity starting before or near the afferent trigger spike; often superimposed on this broad facilitation was a sharply rising PSF starting at a longer latency. The earliest poststimulus facilitation evoked by single microstimuli delivered in DRG occurred in stimulus-triggered averages at a latency of 3.5 ms. In STAs the broad facilitation beginning at latencies shorter than the responses to electrical stimulation was attributed to synchronous discharges in other afferent units. The sharper postspike EMG increases occurring with latencies of ≥3.5 ms were identified as PSF produced by the afferent. The PSF parameters documented in this study were measured after subtracting the effects of synchrony facilitation.

5. PSF of EMG activity began at a mean latency of 5.8 ± 0.3 (SE) ms and peaked at a mean latency of 7.5 ± 0.3 (SE) ms. In previous studies, the PSFs from CM and RM cells had mean onset latencies of 6.3 and 5.6 ms, respectively, and mean peak latencies of 10.2 and 9.1 ms.

6. A measure of the PSF amplitude is the mean percent increase (MPI), defined as the increase of the PSF above its base measured as a percentage of the prespike baseline mean. Muscles facilitated by the 29 afferent units that produced PSF had an average MPI of 4.6 ± 0.3% (SE). This is smaller than the MPIs reported for RM (5.1%) and CM (7.0%) cells (which were not similarly corrected for possible synchrony facilitations).

7. Units that facilitated EMG activity usually facilitated a subset of the coactivated muscles. The 29 adequately tested afferents facilitated an average of 46% of the synergistically coactivated muscles. This is comparable to the percentage of muscles facilitated by CM (40%) and RM (50%) cells. The average number of muscles facilitated per afferent was 2.6.

8. During the step-tracking task the monkeys generated ramp-and-hold torques about the wrist. The afferent units that showed PSF either increased their discharge during wrist flexion alone (10 units) or during extension alone (13 units), or were bidirectionally active (6 units). The most common response pattern, seen in 52% of units, was a tonic discharge. Twenty-one percent of the units had a phasic-tonic discharge, and 27% had a phasic discharge. The latter were often bidirectionally active.

9. The time between the first change in afferent unit activity and the onset of activity of its facilitated target muscles was calculated for 79 unit-muscle pairs from averages of isometric responses. Many afferent units began to discharge before the onset of their target muscle activity. On the average, afferent unit discharge began 52 ± 13 (SE) ms before activation of the target muscle. The onset latencies of the phasic cells tended to be earliest (−150 ± 59 ms), followed by phasic-tonic (−72 ± 14 ms) and tonic cells (−24 ± 17 ms). In previous studies phasic CM and RM cells also tended to discharge earliest within their populations.

10. The effects of torque pulse perturbations, introduced during auxotonic contractions, were tested in seven units. In general, stretching the muscles facilitated by the afferent unit produced a burst in unit activity followed by an EMG burst, whereas shortening the target muscle produced a pause in afferent unit and EMG activity.

11. The results reveal some salient differences between the discharge properties of DRG afferents and supraspinal premotoneuronal (PreM) cells in motor cortex and red nucleus. All three PreM populations include tonic, phasic-tonic, and phasic cells but in significantly different proportions. The PreM afferents were typically active only with their target muscles, like CM cells and unlike RM cells. The hypothesis that these PreM afferents arose from muscle spindles is supported by their short-latency PSF, their responses to perturbations, and their onset before activity of their target muscles; the early activation would further suggest that γ-motoneurons can activate spindles before α-motoneurons become active.

INTRODUCTION

A quantitative picture of the neural inputs to motoneurons of primate forearm muscles has emerged from recent studies of supraspinal premotoneuronal cells. Spike-triggered averages (STAs) of muscle activity have shown that some cells in motor cortex directly facilitate α-motoneuron activity during movement (Fetz and Cheney 1980; Lemon et al. 1986). Similar analysis of neurons in red nucleus has shown that many rubral cells also facilitate muscle activity

during active movement (Cheney et al. 1988; Mewes 1988; Mewes and Cheney 1991). Histograms of the discharge of corticomotoneuronal (CM) and rubromotoneuronal (RM) cells aligned with the torque trajectory during the performance of a ramp-and-hold motor task reveal several characteristic discharge patterns of these cells (Buys et al. 1986; Cheney and Fetz 1980; Cheney et al. 1988). The combination of discharge pattern and postspike effects provides insight into the control exerted by supraspinal premotoneuronal (PreM) cells on their target muscles. Simple ramp-and-hold movements reveal the relation of neural discharge to dynamic and static components of force. Moreover, the similarity of the ramp-and-hold responses across experiments allows the activity of single units to be synthesized into quantitative estimates of the effects of populations of CM and RM cells on muscles (Fetz et al. 1989). At intermediate loads, these supraspinal PreM populations could contribute appreciably to the activity of motoneurons, but other inputs are clearly needed to account for the total activity of motor units. The aim of the present study was to investigate the potential contribution of peripheral afferent fibers to forelimb motoneuron activity.

The activity of peripheral afferents during voluntary limb movement has been elucidated in previous studies (Hulliger 1984; Prochazka 1981; Vallbo et al. 1979), although the correlational links of those afferents with motor units were usually not tested. Schieber and Thach (1985) found that muscle spindle afferents were very active in monkeys performing slow ramp movements of the wrist. STAs of their data have revealed postspike facilitation (PSF) for one of their units (unpublished observations). Experiments designed to investigate the excitation of α-motoneurons by single spindle afferents in human leg muscles with the use of STAs of electromyographic (EMG) activity, failed to reveal postspike effects (Gandevia et al. 1986). In the cat, however, Prochazka (1981) found PSF of gastrocnemius muscle activity in averages triggered from spikes of spindle primary afferent fibers.

The present experiments were undertaken to investigate the effects of primate peripheral afferents on α-motoneurons in motor tasks identical to those used previously to study CM cells (Cheney and Fetz 1980), RM cells (Mewes, 1988), and single motor units (Palmer and Fetz 1985). A major aim was to contrast the relative roles of supraspinal and afferent PreM cells in voluntary movement. Preliminary reports of these findings were published previously (Flament and Fetz 1989; Flament et al. 1990).

METHODS

In two male rhesus macaques (*monkey S*, weighing 5.2 kg, and *monkey T*, weighing 3.0 kg), we recorded the activity of single units in dorsal root ganglia (DRG) C_8 and T_1 extracellularly with glass-coated tungsten microelectrodes. With the monkeys under halothane anesthesia, we performed a unilateral laminectomy of vertebrae C_5–T_1 to expose three to four dorsal roots and their ganglia. We implanted a recording chamber over the cervical vertebrae by centering it laterally to the midline and cementing it with dental acrylic to vitallium screws in the vertebrae (Fig. 1). The elongated shape of this chamber provided access to all exposed ganglia and to the lateral portion of the spinal cord. A microdrive holder with a ball-and-socket joint enabled the electrode to reach any point within the chamber under visual guidance. The implants remained securely fixed to the vertebrae for 7 wk in *monkey S* and 5 wk in *monkey T*.

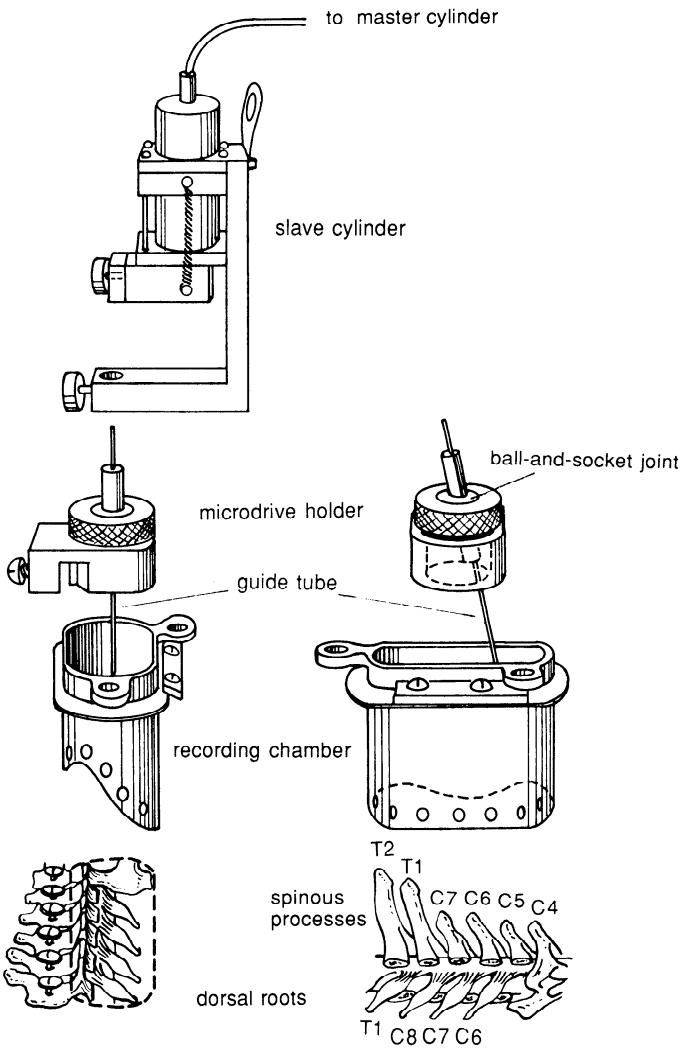

FIG. 1. Exploded view of afferent unit recording system. An elongated recording chamber was cemented to vitalium screws in the lower cervical and upper thoracic vertebrae with dental acrylic; spinous processes embedded inside the chamber provided added support. Ball-and-socket joint of the microdrive holder permitted access to all points within the chamber. Long guide-tube was visually aimed toward a ganglion, and the electrode was advanced by a hydraulic microdrive to penetrate target ganglion.

During recording of afferent unit activity, each monkey was seated in a primate chair with its head and upper back restrained via flexible threaded nylon rods. Two nylon rods (diameter: 4 mm, length: 38 mm) were threaded into nuts cemented to the occiput, and two attached the recording chamber to a bar fixed to the chair. This flexible restraint permitted the monkey to make small postural adjustments but maintained enough stability to allow us to record unit activity. In addition, the arms were restrained by nylon straps. Because forceful movements generally resulted in loss of unit isolation, releasing the limbs for sensory stimulation was kept to a minimum. The recording sessions typically lasted ~3 h.

EMG activity from wrist and digit flexor and extensor muscles was recorded with stainless steel multistranded wires implanted transcutaneously. Once implanted, these wires were taped to the arm and could be left in place for 2–3 wk without loss of recording quality. The monkeys wore a jacket to protect the implants when they returned to their cages. Forearm muscles were identified under anesthesia by the carpal, metacarpal, and phalangeal move-

ments elicited by intramuscular electrical stimulation (400 Hz, 20-ms train, 0.2-ms pulses, 0.01–1.0 mA).

The following muscles were implanted: flexor digitorum superficialis (FDS), flexor carpi ulnaris (FCU), brachioradialis (BR), flexor carpi radialis (FCR), palmaris longus (PL), flexor digitorum profundus (FDP), pronator teres (PT), supinator (SUP), extensor digitorum communis (EDC), extensor carpi radialis (ECR), abductor policis longus (APL), extensor digitorum 4-5 (ED45), extensor digitorum 2-3 (ED23), and extensor carpi ulnaris (ECU).

Behavioral paradigm

Monkeys were trained to make ramp-and-hold torque responses of the wrist in an isometric step-tracking task. Flexion and extension targets were displayed successively on a video monitor placed in front of the monkey. A cursor whose position followed wrist torque was displayed continuously on the screen and had to be positioned in the target zones for a minimum hold time. Accurate behavioral performance was rewarded with fruit juice or applesauce. Afferent unit recordings remained stable for 10–40 min under isometric conditions (Fig. 2).

Monkeys were also trained in a torque pulse paradigm, where they performed auxotonic step-tracking (wrist movement against an elastic load). They were rewarded for returning the torque cursor into the flexion or extension targets after a small perturbation produced by a torque motor coupled to the manipulandum.

In a few cases, when the unit was still isolated after postspike effects were sufficiently documented, we released and manipulated the monkey's arm to identify the modality and locus of adequate natural stimuli (touch, pressure, or joint rotation) activating the afferent unit. This procedure typically led to loss of unit isolation due either to the passive limb movements or to the monkey's reaction, so our information on the units' natural responses was limited.

Measurements of PSF

STAs of rectified EMG activity were computed on-line to identify units for study and off-line from tape recordings. PSF was defined as an increase in full-wave rectified EMG activity after the trigger spike, which rose significantly above baseline fluctuations after a minimal latency of 3.5 ms (see Fig. 3*B*). In some cases, the PSF appeared on top of a broader increase that started before or near the trigger point (Fig. 3*C*). This early increase could be due only to synchronous activity in other units that fired before the trigger and that also facilitated EMG activity (cf. Smith and Fetz 1989).

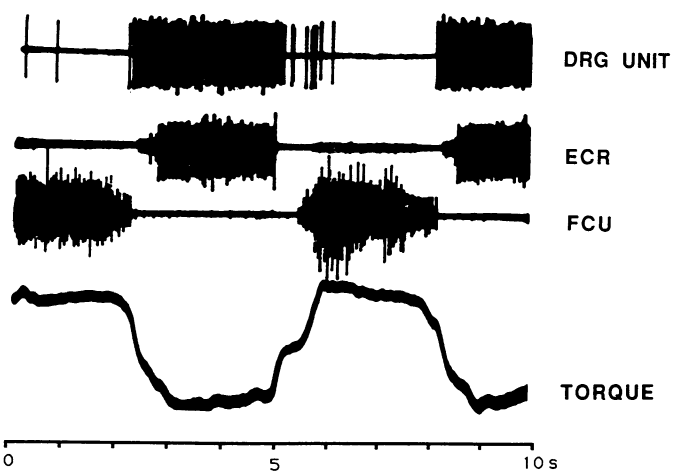

FIG. 2. Recordings of afferent unit and EMG activity from *monkey T*. Single oscilloscope sweep shows action potentials of a C_8 afferent unit, flexor and extensor EMGs, and torque record (flexion upward) for two cycles of step-tracking.

The latencies of the onset and peak of the PSF, as well as the magnitude of the PSF, were calculated after the effects of synchrony were excluded, as shown in Fig. 3. First, the mean baseline level of the STA was calculated for the time period from 20 to 5 ms before the afferent spike. Spike-related increases in EMG were identified by sustained increases above baseline and characterized as either PSF from the afferent spike or EMG facilitation due to synchrony. The synchrony facilitation was identified by steadily increasing EMG above the baseline level beginning before or within 3.5 ms of the afferent trigger. Synchrony facilitation usually appeared as a broad increase in EMG activity (Fig. 3, *C* and *D*). Because 3.5 ms was the minimal latency of effect in the muscles evoked by electrical stimulation of the DRG (Fig. 15), this synchrony facilitation began too early to be a postsynaptic effect of the afferent unit. The PSF that we attributed to the afferent unit was identified by a sharp increase beginning at a latency consistent with afferent facilitation of muscle activity. The PSF could appear alone (Fig. 3*B*) or on top of the broad synchrony facilitation (Fig. 3*C*). In the absence of synchrony facilitation (Fig. 3*B*), the PSF base was equal to the baseline mean; in the presence of synchrony facilitation (Fig. 3*C*), the PSF base was visually identified as the mean level between the onset and end of the PSF. The PSF onset latency was calculated as the time from the trigger spike to the first bin that rose above the base of the PSF. The PSF peak latency was the time from the trigger to the highest bin in the PSF. The magnitude of the PSF was quantified with the mean percent increase (MPI), defined as: MPI = (mean bin amplitude of PSF above base/mean baseline level) × 100.

Previous experience with STA from CM and RM cells had shown that postspike fluctuations may appear in averages compiled with substantially fewer than 2,000 triggers, but that the PSF is reliably and repeatedly produced in averages of 2,000 events. An apparent postspike increase obtained with <1,000 trigger events may change and disappear as more triggers are included. We therefore chose 2,000 triggers as a minimal reliability criterion for PSF identification and measurement.

Elimination of redundant EMG recordings

Redundant EMG recordings were identified by cross-correlating EMG activity off-line (Fetz and Cheney 1980; Lemon et al. 1986). EMG-triggered averages of all muscles were compiled with triggers generated from each muscle in turn. Completely independent EMG records should produce flat, featureless averages when triggered from each muscle. Peaks of activity in EMG averages, occurring at or near the time of the trigger, can reflect synchronous firing produced by common synaptic input as well as redundant recording of the same motor units. To eliminate the possible redundant records from our analysis, we excluded EMG records that showed peaks in EMG averages triggered from other muscles that were >15% of the amplitude of the self-triggered average peaks. This criterion may have excluded some records whose cross-correlation peaks were produced by strong synchronous synaptic drive.

Response patterns of afferents

To identify the response patterns of the afferents during ramp-and-hold torque responses, we compiled averages aligned at onset of the torque ramps. These included time histograms of the afferent unit activity, averages of rectified EMG activity, and the isometric torque. The onset latencies of the afferent units relative to the onset of activity in their facilitated target muscles were calculated from the response averages. The onset latency measured the

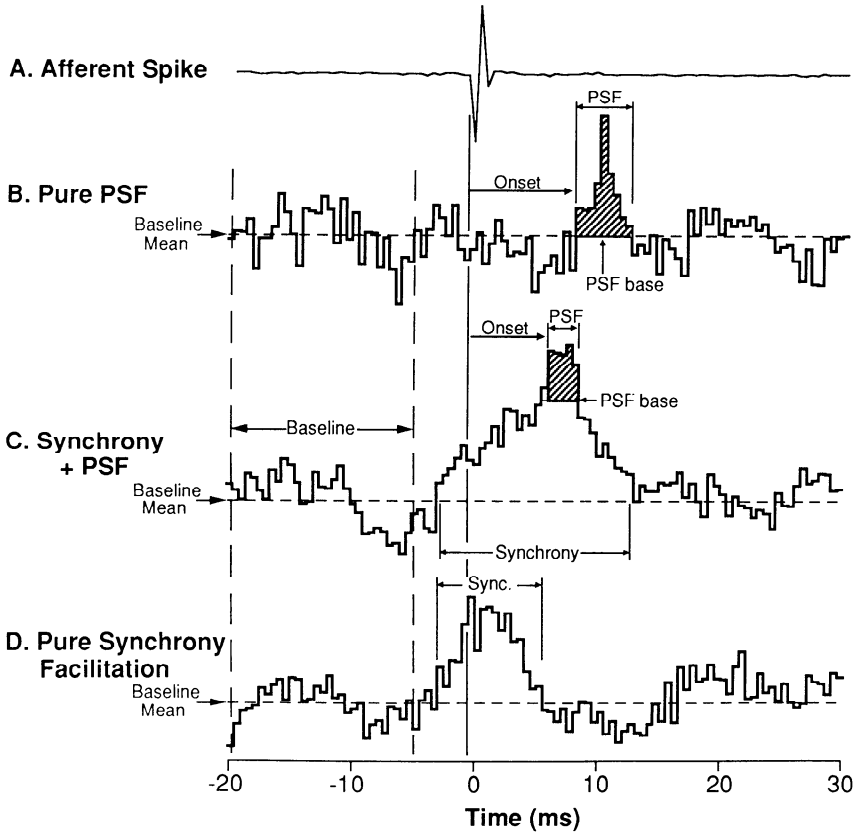

FIG. 3. Spike-related features in STAs of EMG. The 4 traces illustrate (*A*) trigger spike, (*B*) pure PSF, (*C*) PSF superimposed on synchrony facilitation, and (*D*) facilitation produced by synchrony alone. PSF base is identical to baseline mean in *B* and is estimated as the average of pre- and post-PSF bin levels in *C*. Onset latencies were measured relative to onset of spike (vertical line), which preceded the acceptance pulse (origin of time axis).

relative time between the first change in the afferent unit activity and the first change in target muscle activity.

Electrical stimulation of afferents

In some experiments, we stimulated the dorsal roots with 0.2-ms pulses and currents ranging from 10 μA to 8 mA during the same ramp-and-hold torque responses. The latency of poststimulus facilitation was measured from stimulus-triggered averages of rectified EMG activity. This latency was taken as a measure of the time required for transmission of impulse activity from the afferent to the α-motoneuron and thence to the target muscle fibers.

RESULTS

Percentage of DRG afferents producing PSF

Figure 2 shows raw records of the discharge of a DRG afferent and the EMG activity from a flexor and extensor muscle associated with the step-tracking task. Unit recording was quite stable over successive response cycles; this record shows movements made ~15 min after the unit was isolated. Averages of rectified EMG activity triggered from spikes recorded in DRG C_8 and T_1 revealed PSF for many task-related units. From a total of 225 cells recorded in the two monkeys, we tested 125 using STAs of four to six coactivated muscles with >500 triggers. Sixty-eight units showed some degree of spike-related facilitation, which was often much clearer than PSFs from CM cells with comparable triggers. Fifty-nine units were sufficiently active and stable to provide STAs of >2,000 triggers; 29 of these (49%) showed PSF in at least one muscle. The following analysis is restricted to these 29 afferent units. Seventeen were recorded in *monkey S* from DRG C_8, and 12 were recorded in *monkey T* (7 from DRG T_1 and 5 from C_8).

STAs and response averages for four representative afferent units are illustrated in Figs. 4–7. The formats of the figures are the same. The *top left trace* is the afferent trigger spike used to average the EMG activity of coactive muscles illustrated below. To represent their relative sizes accurately, the STAs are shown in proportion to the mean percent increase in activity relative to baseline (this is equivalent to equalizing their baseline levels). The response averages, shown on the *right*, include a histogram of the afferent unit discharge at top, averages of rectified EMG activity of the coactive muscles, and isometric torque trajectories (*bottom trace*). Figures 4 and 5 show tonic and phasic-tonic units recorded in *monkey S*; Figs. 6 and 7 show phasic and tonic afferent units recorded in *monkey T*. These three response patterns (tonic, phasic, and phasic-tonic) were the only patterns observed for afferent units during isometric ramp-and-hold torques. Both the STAs and response averages of muscle activities showed larger modulations in *monkey S* than in *monkey T* (compare Figs. 4 and 5 with Figs. 6 and 7). The lower levels of muscle activity in *monkey T* reflect the fact that this monkey worked at a lower force level. The STAs illustrated for this monkey (Figs. 6 and 7) show that each of these afferent units facilitated only one muscle. On the other hand, *monkey S* showed greater muscle activity, and the STAs showed increases in EMG activity in all of the coactive muscles. However, some of these increases (e.g., STA of PL in Fig. 4) began before the afferent spike. These early increases were attributed to synchronous activity in other inputs to the motoneuron that discharged before the recorded unit.

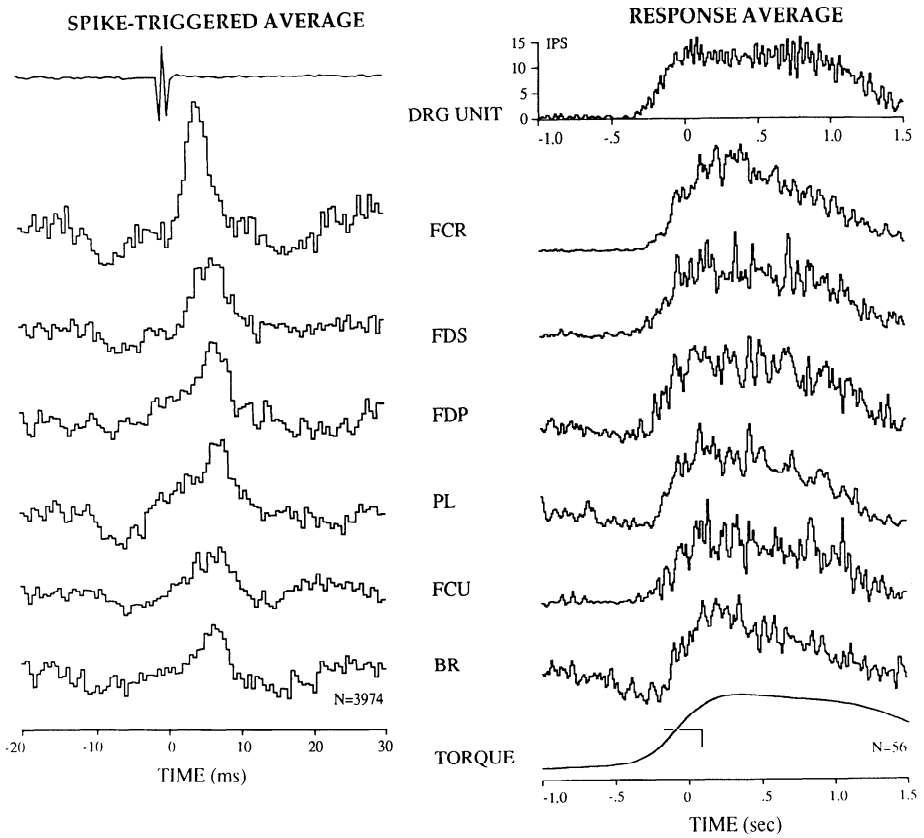

FIG. 4. STAs and response averages of flexor-related tonic afferent unit recorded from C_8 DRG in *monkey S*. This unit produced PSF in 6 independent flexor muscles (*left*) and was coactivated with the same muscles during torque tracking in the flexion direction (*right*). In this and other figures, number of trigger events in each average is given at lower right. Vertical torque calibration bar represents 0.1 nm in all figures; horizontal bar indicates zero torque.

Latency and magnitude of PSF

The latencies of PSF onsets (after synchrony effects are discounted) are plotted separately for the two monkeys in Fig. 8*A*. The overall mean latency of PSF onset for both monkeys was 5.8 ± 0.3 ms (mean ± SE). The mean PSF onset latency was slightly less in *monkey S* (5.6 ± 0.3 ms; range: 3.5–14.0 ms) than in *monkey T* (6.6 ± 0.5 ms; range:

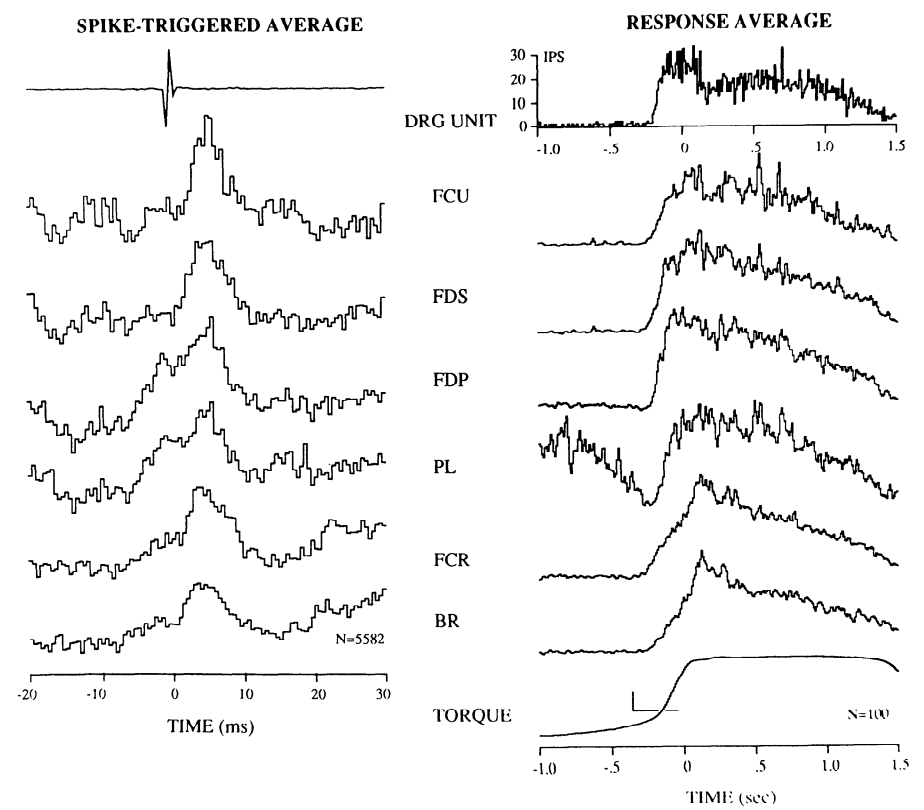

FIG. 5. STA and response averages of flexor-related phasic-tonic afferent unit recorded from C_8 root in *monkey S*. Same format as in Fig. 4.

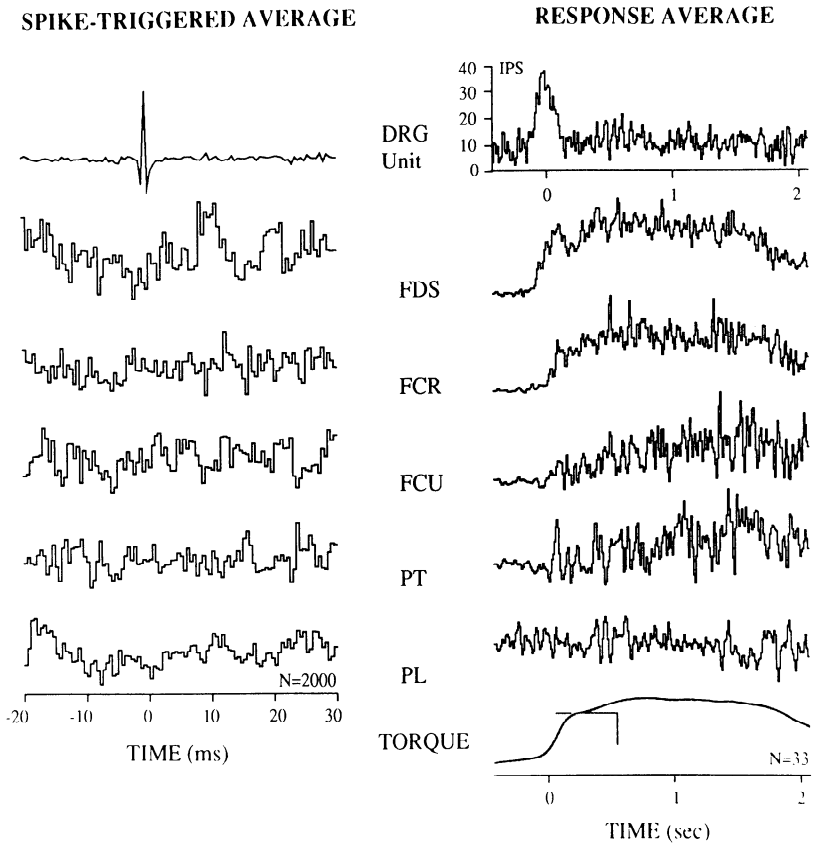

FIG. 6. STA and response averages of flexor-related phasic afferent unit recorded from C_8 DRG in *monkey T*. Only one muscle (FDS) was facilitated. Same format as in Fig. 4.

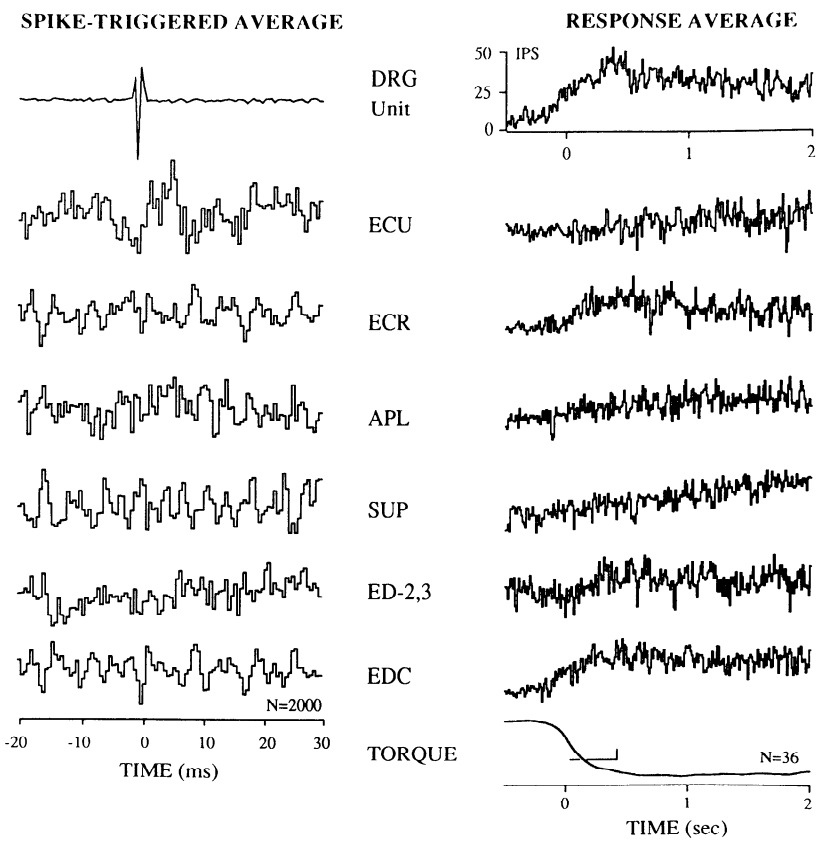

FIG. 7. STA and response averages of extensor-related tonic afferent unit recorded from T_1 DRG in *monkey T*. Same format as in Fig. 4.

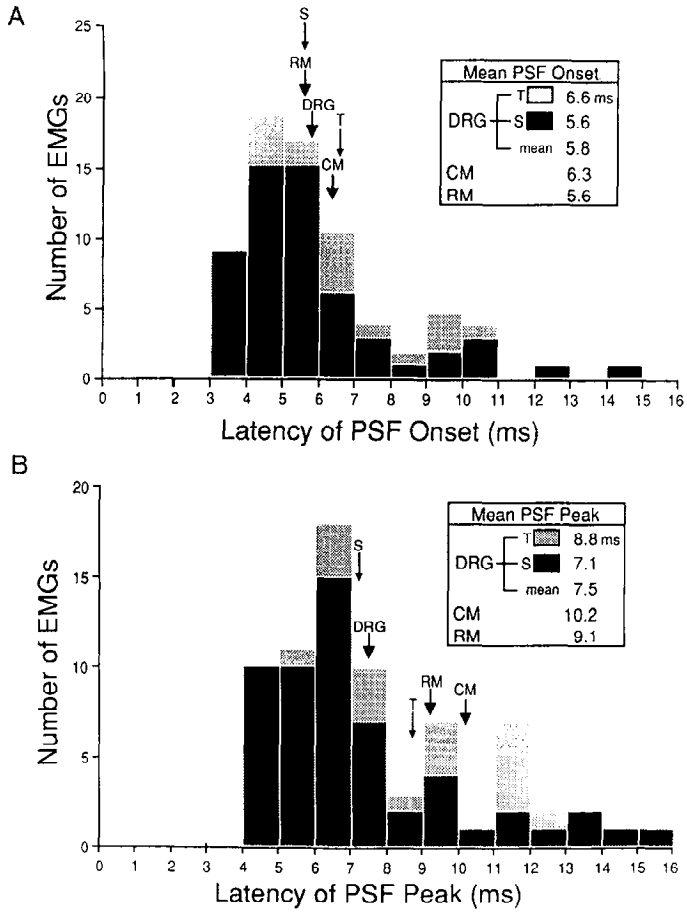

FIG. 8. *A*: Distribution of PSF onset latencies for *monkeys S* and *T*. Abscissa gives latency of PSF onset after afferent trigger spike, and ordinate shows number of EMGs with PSF onset latency within bar interval. Arrows identify the mean onset latencies for afferent units from each *monkey* (*S* and *T*) and overall mean onset for all PreM afferents. Also indicated are the mean PSF onsets for the CM and RM cells taken from previous studies (Cheney et al. 1988). *B*: distribution of PSF peak latencies for *monkeys S* and *T*. Abscissa: latency of PSF peak after afferent trigger spike; ordinate: number of EMGs with a specific PSF peak latency. Arrows identify mean peak latency for afferent units from each *monkey* (*S* and *T*), overall mean peak for all PreM afferents, and mean peak latencies for CM and RM cells (from Fetz and Cheney 1980 and Mewes 1988).

4–10 ms); this difference was not significant ($P < 0.05$). The latencies of the peaks of the PSF (i.e., the highest bin in the PSF) are shown in Fig. 8*B*. The net mean peak latency for both monkeys was 7.5 ± 0.3 ms. Again, the peak latency was less in *monkey S* (7.1 ± 0.4 ms; range: 4.5–15 ms) than in *monkey T* (8.8 ± 0.6 ms; range: 5.5–12.5 ms); this difference was significant at $P < 0.05$.

The onset of the synchrony facilitation (not plotted) had mean latency of 0.2 ± 0.3 ms for the two monkeys. The synchrony facilitation had significantly earlier onset in *monkey S* (0.1 ± 0.2 ms; range: −7.5–3 ms) than in *monkey T* (1.9 ± 0.7 ms; range: 0.5–3 ms). The latency of the peak of the synchrony facilitation usually equaled that of the PSF because the latter appeared on top of the synchrony facilitation.

The magnitude of the postspike effect of afferents on target muscles was quantified by the MPI, measured as the mean amplitude of the PSF above its base, as a percentage of the pretrigger baseline. After synchrony was discounted, the overall average MPI for both monkeys was 4.6 ± 0.3%. As shown in Fig. 9, the PSF was significantly larger ($P < 0.05$) in *monkey S* (mean MPI of 4.9 ± 0.3%; range: 1.1–11.4%) than in *monkey T* (mean MPI of 3.6 ± 0.5%; range: 1.2–7.7%). Inclusion of the synchrony facilitation with the PSF (not shown) yielded mean MPIs of the combined facilitation of 9.7 ± 0.5% in *monkey S* and 4.2 ± 0.8% in *monkey T*. Synchrony enhancements were found in more STAs of *monkey S* (56/74; 75%) than in *monkey T* (5/18; 28%). Thus *monkey S* showed more synchrony in afferent fiber discharge than did *monkey T*.

Distribution and prevalence of PSF in different muscles

Most of the PreM afferent units facilitated more than one muscle. After we had eliminated potentially redundant EMG records, an average of 5.6 independent muscle records were available for the afferent units that showed PSF. The number of muscles facilitated by individual DRG units is plotted in Fig. 10. These 29 units facilitated one to six muscles, with a decreasing probability distribution. The afferent units from *monkey T* had smaller muscle fields than those from *monkey S*. The dependence of the STA technique on EMG activity and the lower muscle forces generated by *monkey T* could have contributed to this difference.

The proportion of independent EMG records showing PSF was similar for flexor muscles (42/92; 45.6% of EMG records) and extensor muscles (31/67; 46.3%). Overall, an average of 46% of all independent EMGs was facilitated, representing an average of 2.6 out of 5.6 muscles facilitated by a given afferent unit.

Target muscles of DRG units

To determine whether any muscles were preferentially affected, we plotted the percentage of tested afferent units that facilitated a particular muscle (Fig. 11*A*). The muscles are displayed in order from the flexor that was most often

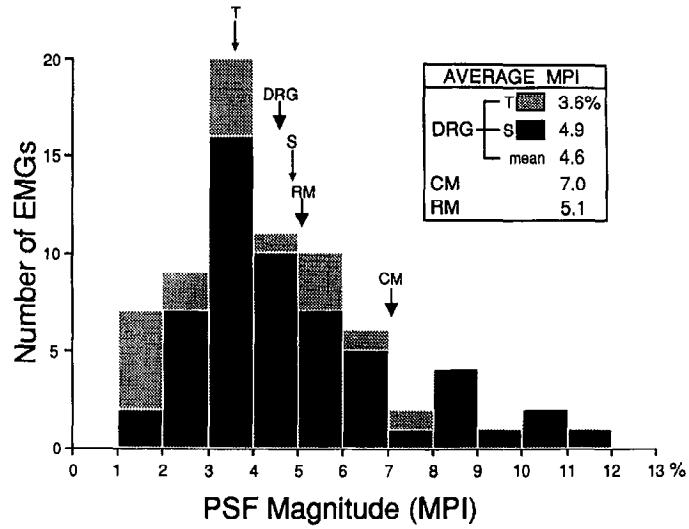

FIG. 9. Distribution of PSF magnitude for *monkeys S* and *T*. Abscissa represents mean percent increase (MPI) of PSF above base relative to baseline activity and ordinate gives number of EMG records with a particular MPI. Arrows identify mean MPI for DRG units from each *monkey* (*S* and *T*) and overall mean MPI for all PreM afferents. Also shown are the mean MPIs for the CM and RM cells (from Cheney et al. 1988).

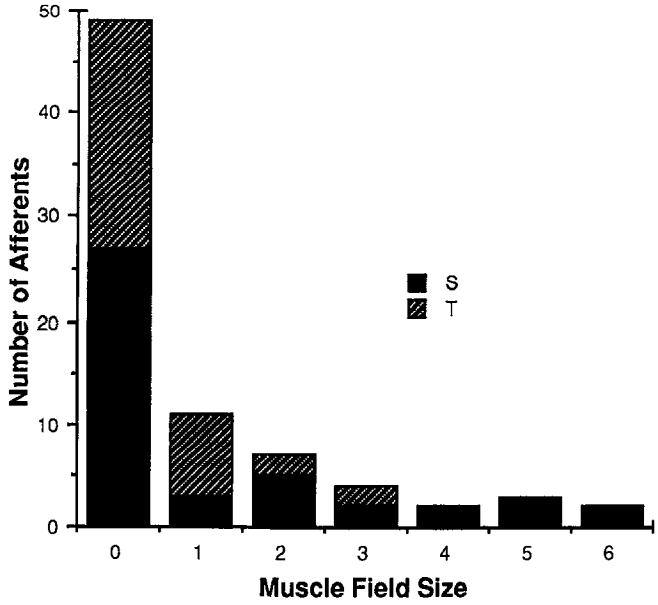

FIG. 10. Size of muscle fields of DRG afferents from *monkeys S* and *T*. The histogram bars indicate the number of DRG units that facilitated the number of muscles on the abscissa. The first bar indicates the number of units that did not facilitate any of the recorded muscles.

facilitated to the extensor that was most often facilitated. Postspike effects were observed most frequently in FDS and in the extensors ECU, ED23 and ED45. The thumb abductor (APL), located on the extensor side of the forearm, also showed a high proportion of postspike effects. The average PSF magnitudes for the corresponding muscles are shown below in Fig. 11*B*. The muscles with the largest PSFs correspond relatively well to the muscles that were most often the targets of the afferent units (cf. Fig. 11, *A* and *B*). Plotting the percentage of PreM afferents for a muscle against the average MPI showed a significant correlation (R = 0.72); the linear regression yielded a percentage of PreM afferents equal to 16.2 ± 7 times MPI.

To indicate how particular muscles were affected from afferents in C_8 and T_1 dorsal roots, Fig. 11 displays the effects of afferents in each root. The C_8 afferents had comparable effects for *monkeys S* and *T*, so these were combined. A small number of DRG afferents were recorded in T1, all from *monkey T*. Effects on muscles from C_8 and T_1 afferents were similar, consistent with the intraspinal branching of afferent fibers in rostral and caudal directions from the spinal cord entry zone (Burke et al. 1979).

Discharge patterns of afferent units during active behavior

Afferent units that showed PSF were classified according to their discharge pattern during the ramp-and-hold torque trajectory generated in the step-tracking task. Most units were active with only one direction of torque, increasing their discharge during either extension (13 units) or flexion (10 units); 6 units were active during both flexion and extension.

Response patterns of afferent units producing PSF fell into three general categories. The most common was a tonic pattern, characterized by a steady increase in activity to a constant level, which was maintained throughout the static hold period (*top right* of Figs. 4 and 7). The second was a phasic-tonic pattern characterized by an abrupt increase in activity during the dynamic phase of the trajectory, followed by a lower but constant level of activity during the hold period (Fig. 5). In both cases the tonic dis-

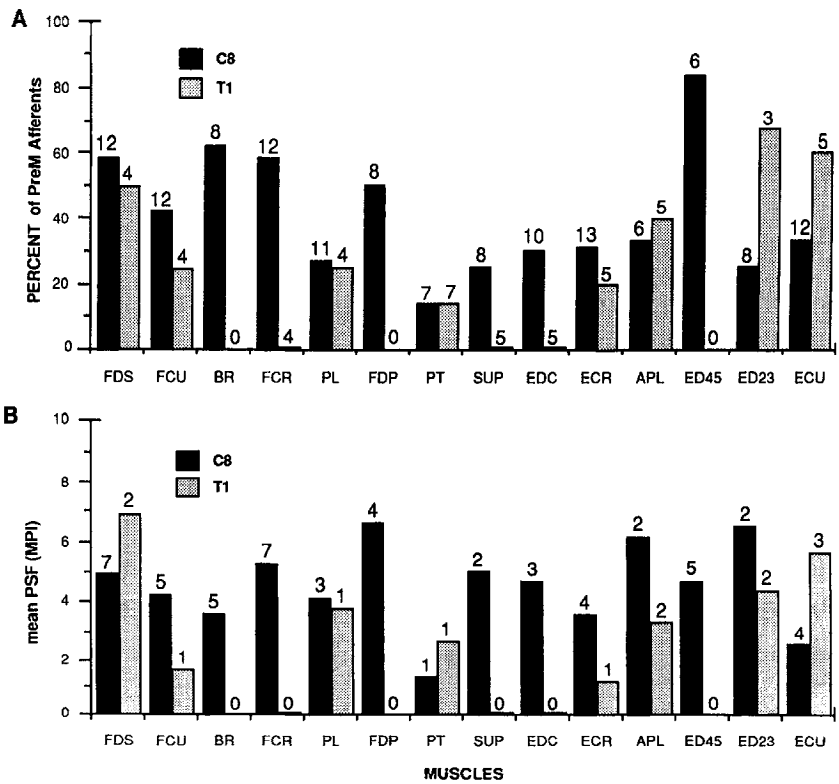

FIG. 11. Distribution and strength of effects on identified forearm muscles from PreM afferents. *A*: percentage of afferents facilitating particular muscles (muscle abbreviations are identified in METHODS). *B*: mean PSF magnitude observed for each forearm muscle. The data for C_8 and T_1 roots are displayed in adjacent bars. Numbers above the histogram bars represent the sample size. For example, 12 units were recorded in C_8 with FDS (*panel A*), and 7 of these units produced a PSF (*B*). *A* shows that the percentage of C_8 PreM afferents facilitating FDS was 7/12 or 58% (dark bar) and the percentage of T_1 PreM afferents facilitating FDS was 2/4 or 50% (light bar).

charge was maintained despite a tendency of the target muscles to show decrementing levels of activity. The third pattern was a phasic discharge: a brief increase in activity with change of torque (Fig. 6). The tonic response pattern was most common, appearing in 15/29 afferent units (52%). The phasic-tonic pattern was seen in 6/29 (21%) units and the phasic pattern appeared in 8/29 (27%) units.

Five of the eight phasic units, and one tonic unit, fired bidirectionally. Bidirectional cells were recorded in both C8 and T1. These cells facilitated either flexors or extensors, but not both; they generally showed a stronger discharge with the direction in which their target muscles were active.

Onset latencies of afferent unit activity

The onset latencies of afferent units were calculated relative to the onset of activity of their facilitated target muscles. Figure 12 illustrates a phasic unit that began to discharge well before its target muscles and before all other recorded synergists. The relative onset latencies are plotted in Fig. 13 for the tonic, phasic, and phasic-tonic afferent units. Most afferent units increased their activity before the onset of EMG activity in the target muscles. The onsets ranged from −380 to 180 ms, with a mean onset latency for

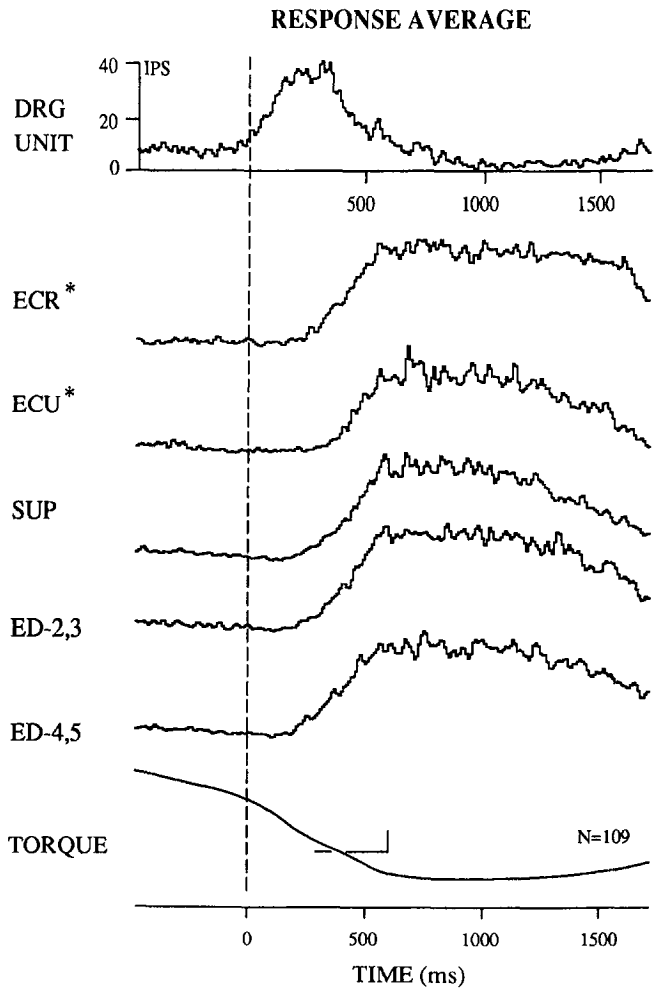

FIG. 12. Response average of a PreM afferent fiber that became active well before any of the recorded agonist muscles, including its two target muscles (*). The vertical dashed line indicates the onset of unit activity.

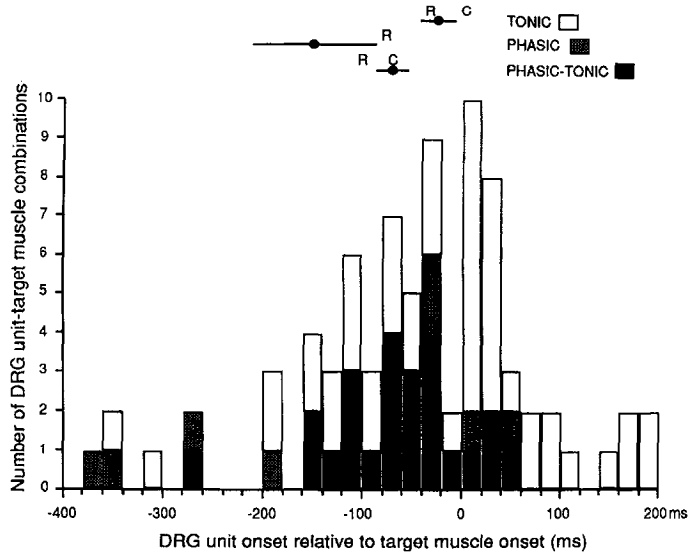

FIG. 13. Onset latencies of afferent units relative to onset of activity in the facilitated target muscles. Zero of abscissa is onset of target muscle activity. Results are presented individually for cells with tonic, phasic, and phasic-tonic response patterns. Histogram displays latencies of all afferent-muscle pairs; dots and horizontal bars at the top designate the means ± SE of these distributions. Mean onset latencies of the CM and RM cells, obtained from Cheney and Fetz (1980) and Mewes (1988), are also presented for each response type by locations of C and R.

all cells of −51 ± 13 (±SE) ms. The cells with a phasic component in their discharge patterns tended to discharge earlier than tonic cells. This can be seen at the *top* of Fig. 13, which shows the mean (●) and SE (——) for the three discharge patterns. The earliest afferent units recruited were the phasic cells with a latency of −150 ± 59 ms, followed by the phasic-tonic cells (−72 ± 13 ms) and even later by the tonic cells (−24 ± 17 ms). Twelve of 29 afferent units showed increases in activity before the first detectable change in EMG activity of each one of their target muscles.

Responses to passive manipulation of limb

TORQUE PULSE RESPONSES. We tested seven units for responsiveness to torque perturbations that briefly flexed or extended the wrist during the active hold period of auxotonic movements; all seven showed a response. A phasic-tonic unit that facilitated flexor muscles (illustrated in Fig. 5) responded to a torque pulse that stretched the flexors with a burst of discharge at the onset of the perturbation, as shown in Fig. 14 (*left*). The EMGs show recordings of independent facilitated muscles. Torque pulses that shortened the flexors (Fig. 14, *right*) were followed immediately by a period of reduced cell activity. The short latency and polarity of these responses are compatible with the response expected from primary muscle spindle afferents. The tonic unit in Fig. 4, which fired during active flexion, also responded with a small burst of activity to a torque pulse that stretched the flexors (not shown). In all seven cases, the afferent units were activated by torque pulses that stretched their target muscles.

RECEPTIVE FIELD IDENTIFICATION. Some tests with natural stimulation were possible for 12 of the 29 PreM afferents. Nine of these responded to manipulation of the hand or wrist; they increased their discharge to passive movement

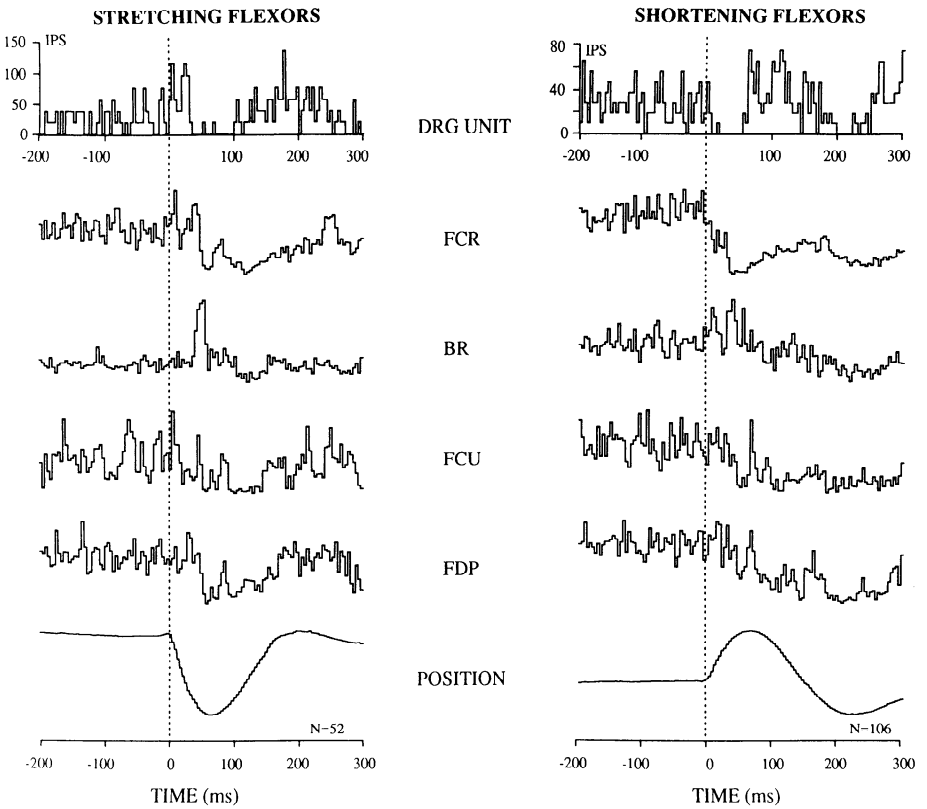

FIG. 14. Response averages of C_8 afferent and rectified EMG activity during torque perturbations. Torque pulses were applied during flexion hold phase of auxotonic movements (*monkey S*).

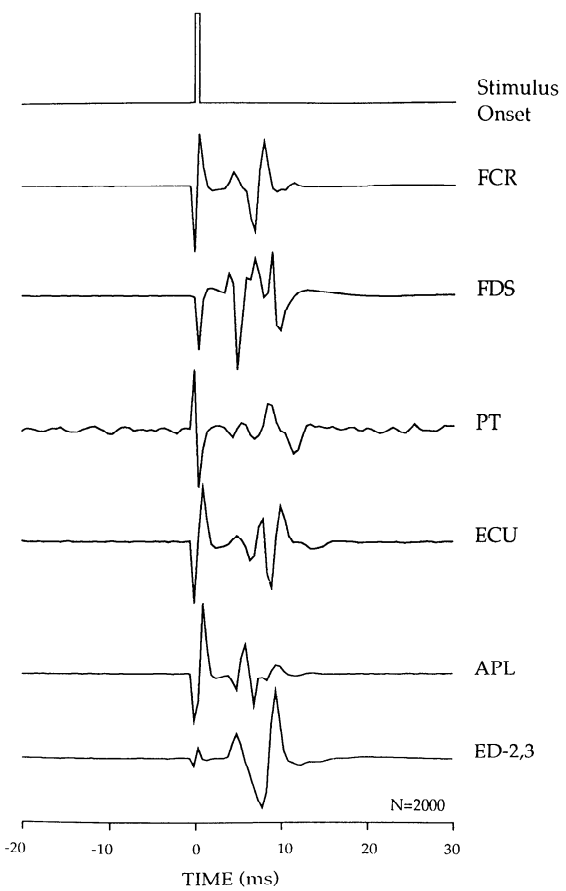

FIG. 15. Stimulus-triggered averages of unrectified EMG activity. DRG T1 in *monkey T* was stimulated with 0.3-mA, 0.2-ms pulses at 10 Hz. Latency of earliest poststimulus effect was 3.5 ms (in FCR and FDS).

of the wrist and/or fingers and to deep pressure on the forearm tendons at the wrist. Cutaneous stimuli were not effective in driving these cells (3 other afferent units were activated by cutaneous stimulation but did not show PSFs). Receptive fields could not be identified for two units, and one unit responded to pressure on the upper arm and shoulder.

Electrical stimulation of dorsal roots

To document conduction times, we looked for responses evoked by electrical stimulation of the dorsal roots in both monkeys. In *monkey S*, large stimulus artifacts precluded accurate measurements of the onset of post-stimulus effects in EMG activity. In *monkey T*, artifacts also appeared in some records, but evoked responses could be identified in many records. Figure 15 shows the responses evoked by stimulation of T_1 with 0.3-mA, 0.2-ms biphasic pulses. The onset latencies of poststimulus facilitation ranged from 3.5 ms to 7.5 ms, with a mean of 4.4 ± 1.7 (SD) ms for all records.

DISCUSSION

Identification of dorsal root afferents

Our afferent units were recorded from C_8 and T_1 DRG, which contain fibers from cutaneous, muscle, and joint receptors in the forearm. We found it very difficult to accurately identify the sensory receptors of the afferents in the awake monkeys. The detailed probing possible in anesthetized animals was precluded by the monkeys' tendency to resist extensive manipulations and by the precarious recording conditions. Consequently, many units were lost when

we tried to determine their response properties. Nonetheless, the majority (75%) of units that were tested responded to joint rotation and deep pressure, both of which are compatible with a spindle origin for these afferents.

Although it was not feasible to prove that these afferents arise from spindles, several observations indirectly support this possibility. The fact that they produced PSF is evidence that they had excitatory correlational links to motoneurons of their target muscles. Clear PSFs were often obtained with <2,000 trigger events, suggesting relatively strong synaptic linkages. Although some of these PSFs may have been mediated by disynaptic links, we believe the afferents producing strongest PSF probably contacted α-motoneurons monosynaptically. These afferents would represent group Ia and group II spindle afferents (Kirkwood and Sears 1974). Group Ib muscle afferents (Watt et al. 1976) and cutaneous afferents (Jenner and Stephens 1982) can also excite motoneurons, but they are at least disynaptically linked and would therefore produce weaker and later PSF, if any. Indeed, confirmed cutaneous afferents did not produce PSF.

The reciprocal responses of the afferent units to torque pulse perturbations is also consistent with the possibility of a spindle origin. All units that produced PSF exhibited short-latency excitation to stretch of their target muscles and inhibition or no response to target muscle shortening. In contrast, afferent units that did not facilitate muscle activity often exhibited bidirectional excitatory torque pulse responses, which are characteristic of cutaneous afferents.

Finally, the 12/29 afferent units that were active before any detectable EMG activity were probably spindle afferents. As discussed below, fusimotor drive to the muscle spindles is the most likely source that could account for increases in afferent activity before EMG onset.

Synchrony facilitation in STA

STAs revealed that many of the muscles had a spike-related increase in average EMG. These increases often began before or near the afferent trigger spike, preceding any effects that could have been produced by the trigger cell. We defined PSF as an increase beginning with a latency of ≥3.5 ms, the minimal time for electrical stimulation of the DRG to produce a muscle response (Fig. 15). The earlier facilitation is probably produced by other afferents that discharged synchronously before the recorded afferent unit. Consequently, we distinguished the "synchrony facilitation" of EMG activity due to other afferent units from the later PSF attributed to the effect of the triggering unit.

Synchrony facilitation was significantly larger and more common in *monkey S* than in *monkey T*. *Monkey S* also generated more EMG activity in association with larger wrist flexion and extension torques. The possibility that greater muscle activity might be associated with greater synchronous activity in afferents could be tested in principle by examining the effects of higher muscle activities on synchrony facilitation in the same monkey; however, our records revealed that each monkey consistently worked within too restricted a range of force levels to resolve this issue.

The synchrony facilitation seen in STAs of *monkey S* was also considerably greater than in previously documented STAs from supraspinal cells. In previous studies, the effects of synchrony in STAs of CM and RM cells were less obvious and were considered negligible. However, recording pairs of CM cells simultaneously, Smith and Fetz (1989) measured synchrony directly and found it could contribute early components of the PSF calculated from a CM cell.

Latency of PSF

All our PSF parameters were measured after the presumed synchrony component of facilitation was excluded. The resulting mean onset latency of the PSF was 5.8 ms after the afferent spike. This was comparable to the mean PSF onset latencies of RM cells (5.2 ms) and CM cells (6.3 ms), although the latter would be expected to be longer for two reasons. The distance from the DRG to the motoneuron (~12–18 mm) is less than the distance from supraspinal neurons to the same motoneuron (several centimeters). Second, the conduction velocity of the large afferent fibers (70–105 m/s; Cheney and Preston 1976) is greater than that of cortical pyramidal fibers (average ~50 m/s; Humphrey and Corrie 1978) or the rubrospinal fibers (mean of 83 m/s; Shapovalov et al. 1971). Indeed, the relative latencies of stimulus-evoked effects are more consistent with these distances: the onset latencies of poststimulus facilitation were earliest for afferent units (3.5 ms) and longer for RM cells (6.5 ms; Mewes 1988) and CM cells (8.0 ms; Cheney and Fetz 1985). Because the PSFs of RM and CM cells were not corrected for synchrony effects, their latency measures could have included an early component of synchrony facilitation.

If one assumes a conduction velocity of 50–110 m/s for α-motoneurons, a conduction distance of 0.2 m from motoneuron to forelimb muscles, and a synaptic delay of 0.5 ms, one would expect the PSF to begin ~2.5–4.5 ms after the afferent spike. This is consistent with our finding that electrical stimulation of the dorsal root evoked poststimulus facilitation at latencies as short as 3.5 ms (Fig. 15); assuming a utilization time of ~0.2 ms (Bagshaw and Evans 1976), one would expect the earliest postspike effects at 3.3 ms. The PSF onset latencies ranged from 3.5 to 14 ms, with most beginning 4–6 ms after the afferent spike (Fig. 8). The earliest PSF would be expected to result from monosynaptic connections of DRG afferents to motoneurons with high conduction velocities, whereas later effects would be due to motoneurons with low conduction velocities and possibly also to transmission through polysynaptic connections. Spinal interneurons could transmit effects from single afferents to many motor units in different muscles (cf. Baldissera et al. 1981). Although monosynaptic connections would mediate the largest postspike effects, disynaptic linkages can also produce second-order postspike effects (Fetz and Cheney 1980). Disynaptic links are necessary for postspike suppression (Kasser and Cheney 1985) as well as facilitation associated with thalamic cells (Mewes and Cheney 1990). Thus excitatory spinal interneurons could also mediate later components of our PSF.

Magnitude of PSF

The magnitude of the PSF was quantified in terms of the MPI, which measures the size of the PSF above its base as a

percentage of the pretrigger baseline EMG activity. The average MPI calculated for all PreM afferent units from both monkeys was 4.6% after synchrony effects were subtracted. This is smaller than the average MPI of RM (5.1%; Mewes 1988) and CM cells (7.1%; Fetz et al. 1990). However, the latter measures may include an undetermined amount of synchrony facilitation.

The magnitude of the PSF is determined by several mediating factors. One factor is the size of the excitatory postsynaptic potentials (EPSPs) produced by the trigger cell in the facilitated motoneurons, because motoneuron firing probability is raised in proportion to the amplitude of the EPSP. In motoneurons of anesthetized cats, single-fiber Ia EPSPs produced peaks in cross-correlation histograms whose counts above baseline were proportional to EPSP amplitudes (Cope et al. 1987). In cats, the sizes of unitary Ia EPSPs range from 10 to 200 μV (Cope et al. 1987; Mendell and Henneman 1971; Munsen 1990; Watt et al. 1976), which would produce cross-correlogram peaks with MPI of 3–60%.

A second factor affecting the size of PSF is the proportion of motor units facilitated by the trigger cell, because the nonfacilitated motor units would contribute only to baseline and reduce the MPI. Thus afferent fibers that distribute terminals to more motoneurons of the pool would produce a larger PSF. Mendell and Henneman (1971) found that Ia afferents produce EPSPs in virtually all motoneurons of their homonymous muscle. Because smaller EPSPs may produce no statistically significant correlogram peaks (Cope et al. 1987), the correlational data may underestimate the extent of divergent connections.

Finally, the contribution of a facilitated motor unit is proportional to the size of its action potential. In cats, the Ia-afferent fibers produce larger EPSPs in small motor units (Munsen 1990), which probably comprise the bulk of our EMG records.

Distribution of DRG effects on target muscles

The number of muscles facilitated by our PreM afferent units (an average of 2.6) is compatible with previous studies of afferent inputs to motoneurons. Recordings of motoneuron EPSPs evoked by stimulation of forearm Ia afferents in the baboon revealed convergence from two to five muscle nerves (Clough et al. 1968). A similar experiment in the cat showed convergence from four to six forearm muscles (Fritz et al. 1989). The distribution of the muscle field sizes of our DRG units (Fig. 10) is similar to that reported for CM and RM cells (Fetz and Cheney 1980; Mewes 1988). Multiple target muscles were observed more frequently than single target muscles. The facilitated muscles were always synergistic agonists activated with either the flexion or extension torques. Postspike effects on antagonist muscles could be revealed with the STA technique only when DRG units were active during both flexion and extension; six cells were bidirectionally active and showed no postspike effects on antagonist muscles. RM cells, in contrast, show both coactivation and cofacilitation of antagonistic muscles (Mewes 1988; Mewes and Cheney 1991).

The muscles most often facilitated by our C_8 and T_1 DRG units tended to be those whose motoneurons had the highest density in segments C_8 and T_1 (including FDS, FCU, ECU, ED23 and APL, Jenny and Inukai 1983). This suggests that the DRG units preferentially connect to motoneurons within their spinal cord entry level, consistent with the input organization previously observed in the cat. Lüscher and Vardar (1989) reported that "the probability that a motoneuron would receive functional connections from a given population of afferent fibers was related to its size and its proximity to the spinal entry level of the afferent fibers." Our results further showed that the muscles facilitated most frequently by the DRG afferents also exhibited the largest postspike effects (Fig. 11). These observations suggest that spindle afferents branch to several motoneuron pools but preferentially affect motoneurons within their spinal entry level.

The frequency with which CM cells facilitated particular target muscles (Fig. 13 of Fetz and Cheney 1980) was different from that observed in our DRG units (Fig. 11). Similarly, the largest postspike effects were observed in different muscles: the largest CM effects were found in wrist extensors (ED45, EDC and ED23), whereas the afferent effects were equally strong in flexor (FDS) and extensor (ED23 and ECU) muscles. These differences are not entirely attributable to the location of the motoneuron pools. For example, EDC, whose motoneurons reside predominantly in the C_8 and T_1 segments, is facilitated relatively weakly by the C_8 and T_1 afferents, yet is strongly and frequently facilitated by both CM and RM cells (Fetz and Cheney 1980; Mewes 1988). This suggests that certain muscles are preferentially controlled from supraspinal inputs, a result consistent with the intracellular data of Clough et al. (1968).

Discharge characteristics of peripheral afferents

Although PSF identifies the target muscles affected by the peripheral afferents, the net effect on target motoneurons during active movement is proportional to the afferents' discharge rate. The discharge patterns of peripheral units during isometric ramp-and-hold torque trajectories fell into three categories (Table 1). The most common pattern, observed in over half of the afferent units, was a tonic

TABLE 1. *Properties of PSF and response patterns of DRG afferents, compared with those of CM, RM, and MU cell populations*

	DRG	CM	RM	MU
PSF				
Magnitude, MPI in %	4.6	7.0	5.1	NA
Onset latency, ms	5.8	6.3	5.6	NA
Peak latency, ms	7.5	10.2	9.1	NA
% of muscles facilitated	46	40	50	NA
Response pattern, %				
Phasic-tonic	21	49	46	23
Tonic	52	28	8	33
Phasic	27	2	20	5

PSF magnitudes, onset latencies, and number of facilitated muscles for CM and RM cells taken from Cheney et al. (1988). PSF peak latency for CM cells taken from Fetz and Cheney (1980) and that for RM cells taken from Mewes (1988). Response types for CM, RM, and MU columns taken from Fetz et al. (1989). PSF, postspike facilitation; DRG, dorsal root ganglia; CM, corticomotoneuronal; RM, rubromotoneuronal; MU, motor unit; MPI, mean percent increase.

discharge during the direction of torque in which their target muscles were active. Such tonic discharge patterns were found in a third of motor units (Palmer and Fetz 1985). The tonic pattern is also relatively common for CM cells (28%) but rare among RM cells (8%); however, the unmodulated RM cells (23%) have a constant tonic discharge during both directions of movement. The tonic discharge pattern most accurately reflects the torque trajectory itself and, when tested for the other populations, the level of tonic discharge has been proportional to torque.

The phasic-tonic discharge pattern was observed in 21% of peripheral afferent units and in all other populations (Table 1). The phasic pattern of activity was seen in 27% of afferent units. These often fired bidirectionally, although the discharge was typically stronger with the direction in which their target muscles were active. Phasic RM units, which make up 20% of the rubral population, are also predominantly bidirectional (Mewes 1988). Relatively few CM cells (2%; Fetz et al. 1989) show pure phasic discharge during the ramp-and-hold wrist task, although a larger proportion show phasic responses in a precision grip task (Lemon et al. 1986). Phasic discharges before the onset of movement in PreM cells would contribute to rapid activation of motoneurons.

In addition to these three response patterns, the suprapinal CM and RM cells exhibit other patterns of activity during isometric step-tracking (Fetz et al. 1989), which could provide additional types of control over the motoneurons. The absence of these patterns in afferent units suggests that the corresponding supraspinal cells do not exert compelling effects on gamma motoneurons. Documenting the activity of single motor units, Palmer and Fetz (1985) found that the largest proportion of motor units (39%) showed decrementing activities. None of our PreM afferents had a decrementing response pattern, even though the associated multiunit EMG often showed a decrementing profile. Thus the steady level of afferent activity did not reflect the decreasing activity of extrafusal muscle fibers, consistent with α-γ dissociation (Prochazka et al. 1985; Schieber and Thach 1985; Vallbo 1971).

In contrast to the predominantly reciprocal response patterns observed in the present study, Schieber and Thach (1985) observed bidirectional spindle afferent activities during controlled wrist movements. One relevant feature of their task was the requirement that the monkey track a slowly moving target. They postulated that the low velocity of the movements required finer control of the wrist; in this task, the central nervous system might adjust the fusimotor drive to permit continuous Ia afferent feedback of muscle length during both flexion and extension of the wrist.

Onset times of afferent units

The early onset times of afferent units relative to their target muscles are relevant to the peripheral sources that could contribute to the afferent units' activities. The alternating isometric wrist flexion and extension torques would induce changing pressures between the manipulandum and the surfaces of the hand, leading to modulations in cutaneous and deep receptors. Although the hand and forearm were restrained, slight changes in wrist angle (2 or 3°) can occur, owing to compression of the padding in the restraints. These small displacements could stimulate sensory receptors as the direction of torque is reversed. Another potent sensory input could arise from fusimotor activity, because descending motor commands can activate both α- and γ-motoneurons (Hulliger 1984; Hunt and Kuffler 1951; Prochazka 1981). Fusimotor drive to intrafusal fibers during isometric contraction would stretch the equatorial region, leading to increased spindle afferent activity.

Activity of spindle afferents has been studied during normal alternating movements, such as walking (Prochazka et al. 1977) and chewing (Goodwin and Luschei 1975; Taylor and Cody 1974), and during wrist flexion and extension movements (Schieber and Thach 1985). Particularly pertinent to the issue of onset time is Vallbo's work on spindle afferent discharge during isometric contractions of human forearm muscles (Edin and Vallbo 1990; Vallbo 1971). Most of their spindle afferents began to discharge 0–150 ms after onset of the EMG activity; unit onsets preceded onset of EMG activity by 100 ms in only ~1% of the cases. In contrast, we commonly observed earlier afferent onset times. This apparent discrepancy may be related to the difference in response conditions. Our monkeys made more forceful and rapid contractions than the controlled isometric forces generated by Vallbo's (1971) subjects; moreover, the monkeys made alternating flexion and extension responses rather than increasing force unidirectionally. Under our conditions, most afferent units discharge −150–100 ms relative to the onset of target muscle activity (mean −51 ms), and 14% of the unit-PSF muscle combinations showed afferent onsets preceding muscle activity by more than 150 ms (Fig. 13). The afferent discharge preceding muscle activity is probably Ia afferent activity produced by γ-motoneuron contraction of muscle spindles. It is significant that 12/29 afferent units increased their activity before each one of their target muscles.

Spindle afferent activity occurring before muscle activation has been suggested to subserve a follow-up length servo mechanism (Eldred et al. 1953). This hypothesis suggests that the central motor commands control desired muscle length by activating γ-motoneurons, thus generating appropriate afferent activity to produce the desired change in extrafusal muscle length. However, the excitatory inputs to motoneurons from spindle afferents cannot be acting alone to produce muscle contraction, because other PreM cells also fire well before the onset of EMG activity and movement. Cell activity begins before target muscle activity in 78% of the cases for CM cells (mean −82 ms; Cheney and Fetz 1980) and in 88% of the cases for RM cells (mean −85 ms; Mewes 1988). Thus the early inputs from supraspinal and segmental PreM cells would combine to activate motoneurons. This is consistent with α-γ-motoneuron coactivation in the generation of movement. The fusimotor reflex loop is not essential for deafferented monkeys to produce goal-directed movements (see Wylie and Tyner 1989 for a review), but it is important for accuracy and dexterity of movements.

Interestingly, the afferents with a phasic component in their discharge patterns tended to be recruited earlier than tonic afferents, similar to RM and CM cell populations (Fig. 13). This is consistent with the greater functional effi-

cacy of phasic activity in bringing the motoneurons to threshold and initiating discharge.

Functional role of dorsal root afferents

A major goal of the present study was to compare the correlational linkages and response properties of afferent PreM fibers with those of descending PreM cells in motor cortex and red nucleus. Comparison of postspike effects is complicated by the exceptional degree of synchrony facilitation observed in *monkey S*. After this was subtracted, the distribution of PSF of afferent PreM fibers was comparable to that of descending cells; all three groups facilitated similar percentages of coactivated muscles (Table 1). The relative conduction times to motoneurons from dorsal roots, red nucleus, and cortex were commensurate with the relative latencies of the PSF peaks and the post-stimulus facilitation onsets; however, the onset latencies of the uncorrected PSFs from CM and RM cells were relatively shorter, suggesting that they may have included some early synchrony components. The magnitudes of the PSF from the supraspinal neurons were larger than the amplitudes of the corrected PSF from peripheral afferents; again, the possible contribution of synchrony effects to the former remains to be determined.

The discharge patterns of PreM cells during flexion and extension torques represent their excitatory inputs to motoneurons. The phasic, phasic-tonic, and tonic response patterns of the afferent units correspond well to similar patterns in the CM and RM cells, although the supraspinal PreM units also exhibit other characteristic discharge patterns during wrist step-tracking. In general, the deeply modulated alternating patterns of the DRG afferents resemble more the activity of CM cells than RM cells, which frequently discharge during both directions of movement. Further differences between supraspinal and segmental PreM cells may become evident under other behavioral conditions.

Perhaps the most surprising similarity was the early onset of the afferent discharges. We expected early onsets to occur preferentially in supraspinal neurons initiating movement, and later discharges in peripheral afferents responding to stimulated receptors. Instead, most of the PreM afferents were also part of the population of PreM cells contributing early inputs to motoneurons before their activation. This suggests that central commands exert effective control via gamma motoneurons and spindle afferents, as well as through direct descending inputs to motoneurons.

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REFERENCES

BAGSHAW, E. V. AND EVANS, M. H. Measurement of current spread from microelectrodes when stimulating within the nervous system. *Exp. Brain Res.* 25: 391–400, 1976.

BALDISSERA, F., HULTBORN, H., AND ILLERT, M. Integration in spinal neuronal systems. In: *Handbook of Physiology. The Nervous System. Motor Control.* Washington, DC: Am. Physiol. Soc., 1981, sect. 1, vol. II, p. 509–595.

BURKE, R. E., WALMSLEY, B., AND HODGSON, J. A. HRP anatomy of group Ia afferent contacts on alpha motoneurons. *Brain Res.* 160: 347–352, 1979.

BUYS, E. J., LEMON, R. N., MANTEL, G. W. H., AND MUIR, R. B. Selective facilitation of different hand muscles by single corticospinal neurones in the conscious monkey. *J. Physiol. Lond.* 381: 529–549, 1986.

CHENEY, P. D. AND FETZ, E. E. Functional classes of primate corticomotoneuronal cells and their relation to active force. *J. Neurophysiol.* 44: 773–791, 1980.

CHENEY, P. D. AND FETZ, E. E. Comparable patterns of muscle facilitation evoked by individual corticomotoneuronal (CM) cells and by single intracortical microstimuli in primates: evidence for functional groups of CM cells. *J. Neurophysiol.* 53: 786–804, 1985.

CHENEY, P. D., MEWES, K., AND FETZ, E. E. Encoding of motor parameters by corticomotoneuronal (CM) and rubromotoneuronal (RM) cells producing postspike facilitation of forelimb muscles in the behaving monkey. *Behav. Brain Res.* 28: 181–191, 1988.

CHENEY, P. D. AND PRESTON, J. B. Classification and response characteristics of muscle spindle afferents in the primate. *J. Neurophysiol.* 39: 1–8, 1976.

CLOUGH, J. F. M., KERNELL, D., AND PHILLIPS, C. G. The distribution of monosynaptic excitation from the pyramidal tract and from primary spindle afferents to motoneurons of the baboon's hand and forearm. *J. Physiol. Lond.* 198: 145–166, 1968.

COPE, T. C., FETZ, E. E., AND MATSUMURA, M. Cross-correlation assessment of synaptic strength of single Ia fibre connections with triceps surae motoneurons in cats. *J. Physiol. Lond.* 390: 161–188, 1987.

EDIN, B. B. AND VALLBO, A. B. Muscle afferent responses to isometric contractions and relaxations in humans. *J. Neurophysiol.* 63: 1307–1313, 1990.

ELDRED, E., GRANIT, R., AND MERTON, P. A. Supraspinal control of the muscle spindles and its significance. *J. Physiol. Lond.* 122: 498–523, 1953.

FETZ, E. E. AND CHENEY, P. D. Postspike facilitation of forelimb muscle activity by primate corticomotoneuronal cells. *J. Neurophysiol.* 44: 751–772, 1980.

FETZ, E. E., CHENEY, P. D., MEWES, K., AND PALMER, S. Control of forelimb muscle activity by populations of corticomotoneuronal and rubromotoneuronal cells. *Prog. Brain Res.* 80: 437–449, 1989.

FLAMENT, D. AND FETZ, E. E. Response patterns of peripheral afferent fibers producing post-spike facilitation of forelimb muscles in the behaving monkey. *Can. J. Physiol. Pharmacol.* 67: Axi–Axii, 1989.

FLAMENT, D., FORTIER, P. A., AND FETZ, E. E. Response patterns and post-spike effects of dorsal root ganglion units recorded during wrist torque tracking in monkeys. *Soc. Neurosci. Abstr.* 16: 112, 1990.

FRITZ, N., ILLERT, M., MOTTE, S., REEH, P., AND SAGGAU, P. Pattern of monosynaptic Ia connections in the cat forelimb. *J. Physiol. Lond.* 419: 321–352, 1989.

GANDEVIA, S. C., BURKE, D., AND MCKEON, B. Coupling between human muscle spindle endings and motor units assessed using spike-triggered averaging. *Neurosci. Lett.* 71: 181–186, 1986.

GOODWIN, G. M. AND LUSCHEI, E. S. Discharge of spindle afferents from jaw-closing muscles during chewing in alert monkeys. *J. Neurophysiol.* 38: 560–571, 1975.

HULLIGER, M. The mammalian muscle spindle and its central control. *Rev. Physiol. Biochem. Pharmacol.* 101: 1–110, 1984.

HUMPHREY, D. R. AND CORRIE, W. S. Properties of pyramidal tract neuron system within a functionally defined subregion of primate motor cortex. *J. Neurophysiol.* 41: 216–243, 1978.

HUNT, C. C. AND KUFFLER, S. W. Further study of efferent small-nerve fibers to mammalian muscle spindles. Multiple spindle innervation and activity during contraction. *J. Physiol. Lond.* 113: 283–297, 1951.

JENNER, J. R. AND STEPHENS, J. A. Cutaneous reflex responses and their central nervous pathways studied in man. *J. Physiol. Lond.* 333: 405–419, 1982.

JENNY, A. B. AND INUKAI, J. Principles of motor organization of the monkey cervical spinal cord. *J. Neurosci.* 3: 567–575, 1983.

KASSER, R. J. AND CHENEY, P. D. Characteristics of corticomotoneuronal postspike facilitation and reciprocal suppression of EMG activity in the monkey. *J. Neurophysiol.* 53: 959–978, 1985.

KIRKWOOD, P. A. AND SEARS, T. A. Monosynaptic excitation of motoneurones from secondary endings of muscle spindles. *Nature Lond.* 252: 243–244, 1974.

LEMON, R. N., MANTEL, G. W., AND MUIR, R. B. Corticospinal facilitation of hand muscles during voluntary movement in the conscious monkey. *J. Physiol. Lond.* 381: 497–527, 1986.

LÜSCHER, H.-R. AND VARDAR, U. A comparison of homonymous and heteronymous connectivity in the spinal monosynaptic reflex arc of the cat. *Exp. Brain Res.* 74: 480–492, 1989.

MENDELL, L. M. AND HENNEMAN, E. Terminals of single Ia fibers: location, density and distribution within a pool of 300 homonymous motoneurons. *J. Neurophysiol.* 34: 171–187, 1971.

MEWES, K. *Characteristics of rubromotoneuronal cells and their role in the control of the hand in primates* (PhD thesis). Kansas City, KS: University of Kansas, 1988.

MEWES, K. AND CHENEY, P. D. Postspike facilitation (PSpF) of EMG activity from neurons in primate motor thalamus. *Neurosci. Abstr.* 16: 425, 1990.

MEWES, K. AND CHENEY, P. D. Facilitation and suppression of wrist and digit muscles from single rubromotoneuronal cells in the awake monkey. *J. Neurophysiol.* 66: 1965–1977, 1991.

MUNSEN, J. B. Synaptic inputs to type-defined motor units. In: *The Segmental Motor System,* edited by M. D. Binder and L. M. Mendell. New York: Oxford Univ. Press, 1990, p. 291–307.

PALMER, S. S. AND FETZ, E. E. Discharge properties of primate forearm motor units during isometric muscle activity. *J. Neurophysiol.* 54: 1178–1193, 1985.

PROCHAZKA, A. Muscle spindle function during normal movement. *Int. Rev. Physiol.* 25: 47–90, 1981.

PROCHAZKA, A., HULLIGER, M., ZANGGER, P., AND APPENTENG, K. "Fusimotor set": new evidence for alpha-independent control of gamma-motoneurons during movement in the awake cat. *Brain Res.* 339: 136–140, 1985.

PROCHAZKA, A., WESTERMAN, R. A., AND ZICCONE, S. P. Ia afferent activity during a variety of voluntary movements in the cat. *J. Physiol. Lond.* 268: 423–448, 1977.

SCHIEBER, M. H. AND THACH, W. T. Trained slow tracking. II. Bidirectional discharge patterns of cerebellar nuclear, motor cortex, and spindle afferent neurons. *J. Neurophysiol.* 54: 1228–1270, 1985.

SHAPOVALOV, A. I., KARAMJAN, O. A., KURCHAVYI, G. G., AND REPINA, Z. A. Synaptic actions evoked from the red nucleus on the spinal alpha-motoneurons in the rhesus monkey. *Brain Res.* 32: 325–348, 1971.

SMITH, W. S. AND FETZ, E. E. Effects of synchrony between primate corticomotoneuronal cells on post-spike facilitation of muscles and motor units. *Neurosci. Lett.* 96: 76–81, 1989.

TAYLOR, A. AND CODY, F. W. J. Jaw muscle spindle activity in the cat during normal movements of eating and drinking. *Brain Res.* 71: 523–530, 1974.

VALLBO, A. B. Muscle spindle response at the onset of isometric voluntary contractions in man. Time difference between fusimotor and skeletomotor effects. *J. Physiol. Lond.* 218: 405–431, 1971.

VALLBO, A. B., HAGBARTH, K.-E., TOREBJORK, H. E., AND WALLIN, B. G. Somatosensory, proprioceptive and sympathetic activity in human peripheral nerves. *Physiol. Rev.* 59: 919–957, 1979.

WATT, D. G. D., STAUFFER, E. K., TAYLOR, A., REINKING, R. M., AND STUART, D. G. Analysis of muscle receptor connections by spike-triggered averaging. I. Spindle primary and tendon organ afferents. *J. Neurophysiol.* 39: 1375–1392, 1976.

WYLIE, R. M. AND TYNER, C. F. Performance of a weight-lifting task by normal and deafferented monkeys. *Behav. Neurosci.* 103: 273–282, 1989.