Motor and Sensory Properties of Primate Corticomotoneuronal Cells

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Introduction

The functional role of motor cortex in volitional movement has received much attention since Evarts' first recordings from pyramidal tract neurons in trained monkeys (1966, 1967). However, the interpretation of single-unit recordings in relation to movement has been hindered by lack of evidence for causal relations between the recorded cells and muscle activity. Recently, Fetz and Cheney (1980) used spike-triggered averaging of rectified EMG activity to reveal postspike facilitation (PSF) of muscle activity from single corticomotoneuronal (CM) cells. The purpose of this paper is to review the technique of spike-triggered averaging of EMG activity and some of the functional properties of primate motor cortex cells with functional linkages to forelimb muscles. We will discuss three major issues concerning the motor and sensory properties of CM cells: (a) the output organization of single CM cells, (b) the encoding of movement parameters by CM cells, and (c) the role of CM cells in long-latency stretch-evoked responses of muscle.

Methodology of Spike-Triggered Averaging of EMG Activity

The rationale for spike-triggered averaging of EMG activity as a means of identifying CM cells is that the individual EPSPs produced by these cells in target motoneurons should increase the firing probability of motor units, albeit weakly, at a fixed latency following the occur-

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rence of the CM cell spike. This increase in firing probability, time-locked to the occurrence of the CM cell spike, may be detected by averaging the segments of EMG activity associated with many spikes. To illustrate this procedure, Fig. 1 shows the spike discharge of an extension-related CM cell and the EMG activity of a representative extensor muscle. EMG activity is full-wave rectified to avoid possible cancellation of opposite phases of facilitated motor unit potentials occurring at varying latencies. Rectification also distinguishes postspike facilitation from postspike suppression. The middle column in Fig. 1 shows the perispike EMG activity, on an expanded time scale, associated with each of the first five spikes in the record at the left. These segments of the analog EMG waveform, extending from 5 ms before to 25 ms after the cortical cell spike, are selected by the computer, digitized at 4 kHz, and averaged. Cumulative averages of these five EMG records are shown in the right-hand column of Fig. 1. Record 1 is simply the digitized form of the analog EMG waveform and demonstrates that the sampling rate is adequate to resolve even the smallest EMG peaks. The fortuitous postspike peaks in this first EMG record quickly become submerged in noise as additional EMG segments are averaged. However, the average rectified EMG activity associated with 2000 cell spikes (bottom record) shows a well-defined postspike facilitation at a latency of 9 ms following the cortical spike. Overall, PSF had a mean onset latency of 6.7 ms (n = 346), a mean peak latency of 10.2 ms (n = 343), and a mean amplitude of 9.0% (n = 164) above baseline. Several factors combine to determine the actual shape and latency of a particular PSF, including the shape of facilitated motor unit potentials, the conduction velocity of the facilitated motoneurons, and the shape of the underlying CM-EPSP waveforms. Nevertheless, a correlation peak, such as that in Fig. 1, is evidence that the trigger cell is synaptically linked, probably monosynaptically, to motoneurons innervating the muscle whose activity was averaged. We refer to cells generating clear PSF as CM cells. However, it must be remembered that PSF is a direct measure of a cell's correlational linklage to motoneurons, not proof of its anatomical linkage.

The method of spike-triggered averaging is capable of identifying CM cells and their facilitated target muscles, but its application to testing a cell's effect on antagonists of the target muscles is limited by the fact that CM cells are normally inactive during the antagonist phase of alternating movement. To overcome this limitation we de-

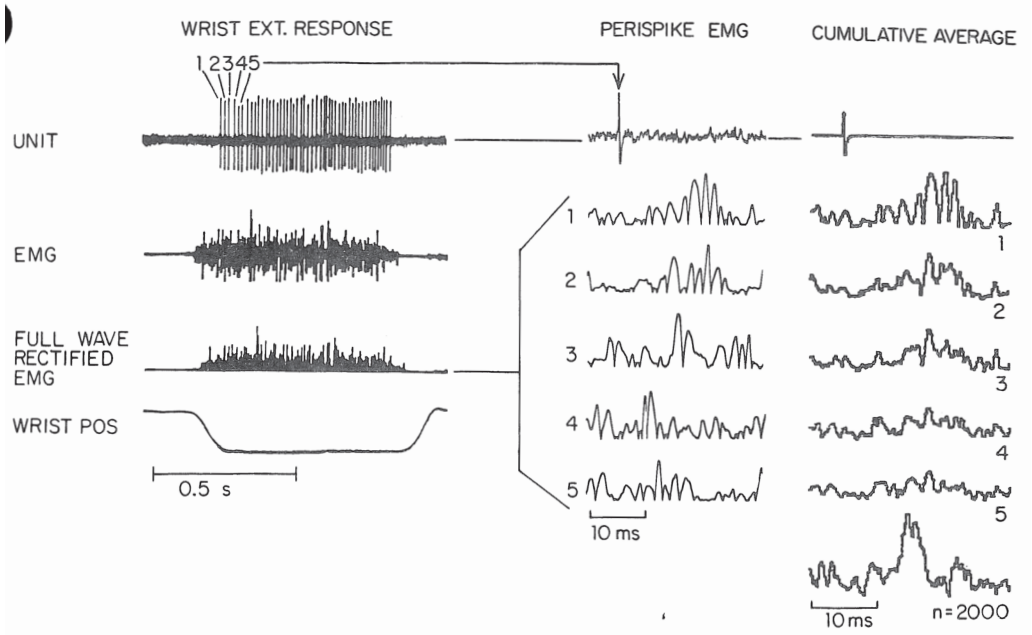

Fig. 1. Spike-triggered averaging procedure used to detect postspike effects. Single extension response of a corticomotoneuronal (CM) cell is shown at *left* with normal and rectified EMG activity. *Middle column* shows the rectified EMG associated with each of the first five spikes in the response at left. *Right column* shows the cumulative averages for the first n spikes where n = 1,2...5, and 2000. (Fetz and Cheney 1980)

veloped a double-barreled electrode for use in chronic preparations, which enables simultaneous unit recording and glutamate iontophoresis to maintain the cell's activity during the antagonist phase of movement (Kasser and Cheney 1982). By combining spike-triggered averaging with glutamate iontophoresis, it is possible to test the output effects of motor cortex cells on both agonist and antagonist muscles.

Motor Properties of CM Cells

The motor properties of CM cells can be divided into two categories: (a) organizational features of the cell's output, including the sign, distribution, and efficacy of its synaptic coupling with motoneurons, and (b) functional relations between the cell's discharge and parameters of active movement. The contributions of spike-triggered averaging to the understanding of these properties are discussed below.

Output Organization of Corticospinal Neurons

The excitatory or inhibitory nature of a cell's effect on motoneurons of agonist and antagonist muscles can be revealed using spike-triggered averaging together with glutamate iontophoresis to maintain cell activity during the antagonist phase of alternating movement. We have identified three basic patterns of synaptic influence on agonist and antagonist muscles, as illustrated in Fig. 2. Agonist muscles are defined as those with which the cell coactivates during motor tasks. The agonists of each of the cells illustrated in

Fig. 2a–i. Types of CM cell output organization. Examples of response averages and spike-triggered averages of both agonist and antagonist muscles for each cell type are shown. All cells were extension related. Firing rates of the pure facilitation and reciprocal cell examples are abnormally high during the antagonist phase of movement because of glutamate excitation. Number of events averaged in this and all subsequent figures is given in parentheses in the lower right corner of each panel. *Asterisks* in **a**, **b**, and **c** indicate muscles showing either postspike facilitation or suppression. Abbreviations for muscles in this and following figures are: *ECR-L*, extensor carpi radialis longus; *ECU*, extensor carpi ulnaris; *ECR-B*, extensor carpi radialis brevis; *ED*, extensor digitorum; *EDC*, extensor digitorum communis; *FCR*, flexor carpi radialis; *FCU*, flexor carpi ulnaris; *FDP*, flexor digitorum profundus; *FDS*, flexor digitorum superficialis; *PL*, palmaris longus; *PT*, pronator teres

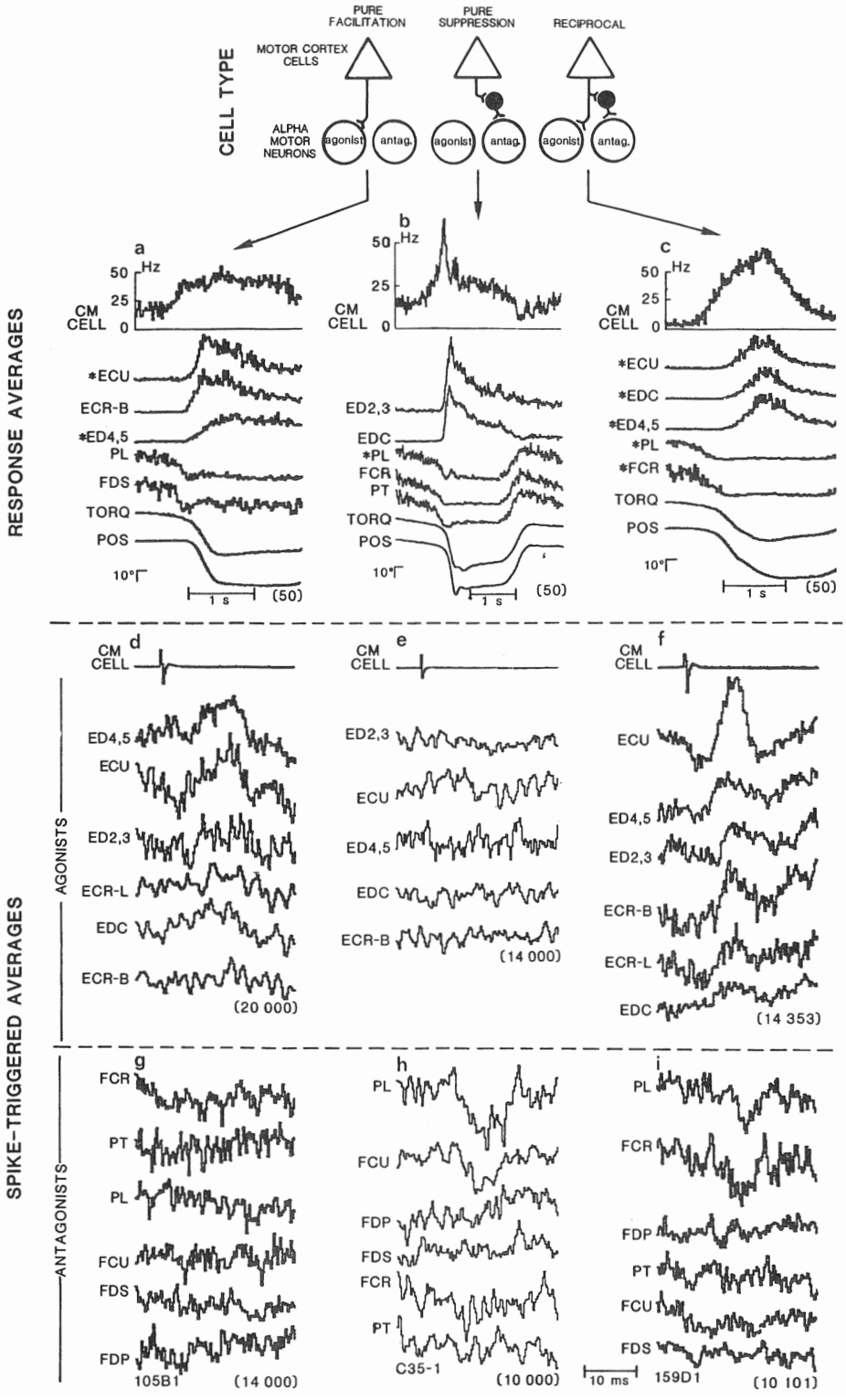
PURE FACILITATION
PURE SUPPRESSION
RECIPROCAL
CELL TYPE
MOTOR CORTEX CELLS
ALPHA MOTOR NEURONS
agonist
antag.
RESPONSE AVERAGES
a
b
c
50 Hz
25
0
CM CELL
*ECU
ECR-B
*ED4,5
PL
FDS
TORQ
POS
10°
1 s
(50)
ED2,3
EDC
*PL
FCR
PT
*EDC
*FCR
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Fig. 2 are extensor muscles, as shown by the response averages. *Pure facilitation* cells produce PSF in agonist muscles, but have no effect on antagonists. Figure 2a is an example of the spike-triggered averages of agonist and antagonist muscles for one such cell. In this and other examples, spike-triggered averages were computed from only the spikes occurring during activity of the muscles being averaged. Clear facilitation appears in extensor digitorum (ED 4,5) and extensor carpi ulnaris (ECU), but the flexors show no significant postspike effect. We conclude that these cells have an excitatory coupling with agonist motoneurons, but exert no measurable effect on motoneurons of antagonist muscles. A second cell type, termed *"pure suppression,"* is illustrated in the middle column of Fig. 2. These cells have no effect on the agonist muscles with which they coactivate, but only suppress the antagonists. The cell illustrated in Fig. 2b suppressed palmaris longus (PL) and flexor carpi ulnaris (FCU), but had no effect on any of five recorded extensor muscles in either spike-triggered averages or stimulus-triggered averages (not shown, but see below). The third cell type, termed *"reciprocal,"* facilitates agonist muscles and also suppresses antagonists. The output of such cells is ideally suited for mediating alternating movements which require a reciprocal pattern of flexor and extensor muscle activity. Spike-triggered averages for one reciprocal cell are shown in Fig. 2c. Note the clear postspike facilitation of all the extensor muscles and reciprocal postspike suppression of PL and flexor carpi radialis (FCR). The mean onset latency of reciprocal postspike suppression in these two muscles (9.7 ms) is 3.7 ms longer than the onset latency of PSF in the six extensors (6.0 ms). Overall, reciprocal postspike suppression from 12 CM cells had an onset latency of 8.9 ± 3.1 ms (n = 20), compared with 5.3 ± 1.6 ms (n = 37) for PSF from the same cells. Postspike suppression was typically weaker and appeared in fewer muscles than PSF. The mean decrease below baseline of peak suppression was 4.1 ± 1.6% (n = 20), compared with a mean increase of 8.0 ± 7.0% (n = 37) for peak facilitation from the same cells. Of 11 reciprocal CM cells whose output effects were determined on five or six agonists and five or six antagonists, the mean number of agonist muscles facilitated per cell was 3.1, compared with 1.7 antagonists suppressed. All these factors suggest that reciprocal suppression is not direct, but is most likely mediated by CM axon collaterals to inhibitory interneurons, probably Ia inhibitory interneurons, which are known to receive convergent input from corticospinal neurons (Jankowska and Tanaka 1974). These basic types of corticospinal output organization represent fundamental

organizational units available to motor cortex for the control of muscle activity. A particular movement would require selection by the central motor program of cells whose output is appropriate in terms of their basic output organization and in terms of their specific target muscles. As yet, no examples of cells which clearly facilitate both wrist flexor and extensor muscles have been encountered.

A further property of the output organization of CM cells is the extent of divergence of their effects on motoneurons of multiple agonist muscles. By computing simultaneous spike-triggered averages of multiple synergist muscles, we determined that 50% of wrist-related CM cells produced clear PSF in only a single muscle, consistent with a high degree of specificity in the CM control of muscles; however, the remaining cells clearly facilitated two or more synergistic muscles (Fetz and Cheney 1980). These findings do not support the notion that motor cortex output is organized solely in terms of specific control of single muscles, at least for cells related to wrist movements which involve coactivation of many synergists. However, using a more discrete task, precision finger grip, Lemon and Muir (1983) found that all of the seven CM cells they tested facilitated only one of five recorded muscles of the hand and digits.

Another question concerns the distribution of PSF to different motor units within a muscle. This question could in principle be answered by cross-correlating the CM cell spike train with the spike discharges of many single motor units sampled from a single muscle. In practice this experiment has proved to be technically difficult, although the effects of microstimuli on single motor units have been studied (Sawyer and Fetz 1981). Cross-correlating trains of microstimuli applied to cortical output sites (see Fig. 3) with the spike trains of motor units from a single facilitated muscle has shown that minimal single microstimuli typically facilitated all the motor units of a muscle (95%). These findings on the effects of cortical output zones, coupled with others suggesting that neighboring CM cells have similar patterns of terminations with motoneurons of synergist muscles (see below), support the hypothesis than individual CM cells, like Ia afferents, may influence a large fraction of the motor units within a muscle.

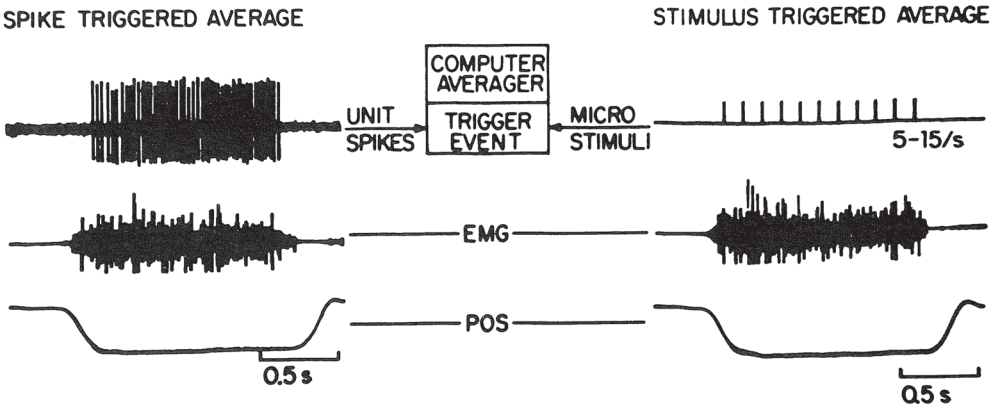

Fig. 3. Comparison of spike- and stimulus-triggered averaging procedures. For stimulus-triggered averaging, intracortical microstimuli (5 - 10 μA) were applied at low frequency (5 - 15 Hz) to the recording microelectrode during the phase of movement which engaged the activity of the cell recorded at that site

Output Effects Revealed by Stimulus-Triggered Averaging

Further insights into the cortical organization of CM cells were obtained by comparing the output effects of a single CM cell with those of single intracortical microstimuli. Stimulus-triggered averages (Fig. 3) were computed by applying microstimuli at low intensity (5 - 15μA) and low frequency (5 - 15 Hz, to avoid temporal summation) to the site of a recorded CM cell. In tests at 22 CM cell sites, poststimulus facilitation was observed in the same muscles which showed PSF in the spike-triggered average; moreover, the relative amplitude of poststimulus facilitation across muscles usually matched that of postspike facilitation. The absolute amplitude of poststimulus facilitation, however, was much greater than postspike facilitation; single 5-μA stimuli evoked facilitation that was six times stronger than PSF. Figure 4 shows an example of postspike and poststimulus facilitation for a single cortical site. The greater amplitude of poststimulus facilitation suggests that it is mediated by several cells located near the electrode and activated by the stimulus (Rank 1975), whereas postspike facilitation is mediated by only one cell. However, the fact that the profile of poststimulus facilitation (across muscles) remains similar to the profile of postspike facilitation, despite a contribution from additional CM cells, suggests that the output effects of each cell activated by the stimulus are similar to that of the single cell used to compile the spike-triggered aver-

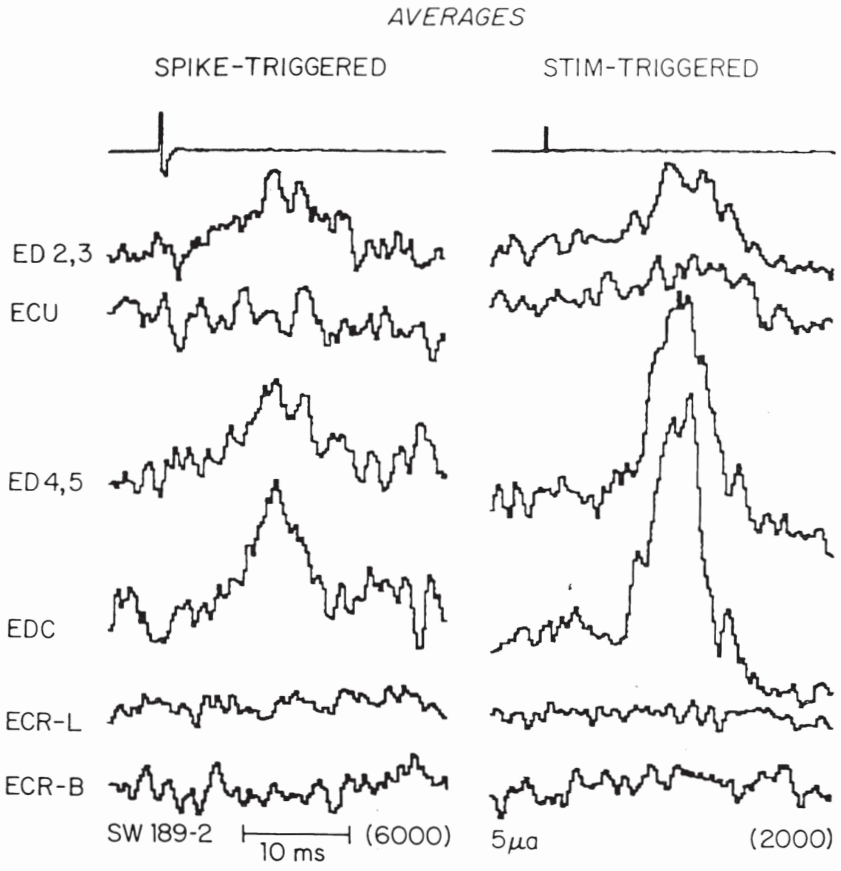

Fig. 4. Example of spike- and stimulus-triggered averages obtained from a single cortical site. Note that the relative amplitude of facilitation is the same in both sets of averages, but poststimulus facilitation is stronger than postspike facilitation. Spike-triggered averages in this case are based on 6000 trigger events, stimulus-triggered averages on 2000 events

age. This conclusion is supported by the observation that neighboring CM cells typically facilitated the same target muscles.

The average size of cortical output zones producing a uniform pattern of poststimulus facilitation in synergist muscles was estimated to be about 800 μm, from measurements of the amplitude of poststimulus facilitation evoked from sites along the bank of the precentral gyrus (Sawyer et al. 1979). These optimal output sites often coincided with the location of clusters of horseradish peroxidase (HRP)-labeled corticospinal neurons identified after cervical injection of HRP.

Jones and Wise (1977) also reported such clustering of HRP-labeled corticospinal neurons. We propose that one of the properties common to cells belonging to a cluster is that they share a similar distribution of synaptic terminations with motoneurons of synergist muscles.

Functional Relations Between CM Cell Activity and Active Movement

CM Cell Types

CM cells identified by their facilitation of agonist muscles have been categorized into four types, based on their discharge pattern during active movement (Cheney and Fetz 1980). These types and their relative frequency of occurrence are: phasic-tonic (59%), tonic (28%), phasic-ramp (8%), and ramp (5%). Both phasic-tonic and tonic cells show steady, sustained, repetitive discharge during the hold phase of agonist movement, but differ in that phasic-tonic cells discharge at a higher frequency during the dynamic (ramp) phase of movement (Fig. 5). Ramp cells show an incrementing discharge during the hold phase of movement. Pure phasic cells, without sustained discharge during the hold period, are common in motor cortex, but never produced PSF, and apparently are not CM cells for agonist muscles involved in wrist movements.

Single motor units in agonist forearm muscles commonly exhibited similar discharge patterns in relation to ramp-and-hold wrist movement (Sawyer and Fetz 1981). Fifty-nine percent of motor units were either phasic-tonic or tonic; 39% showed decrementing discharge during the hold period, a pattern that was the reverse of the ramp CM cells. Five percent were only phasic and showed showed no sustained discharge during the hold period. All four types of motor units were facilitated by microstimuli at a given cortical output site.

Encoding of Movement Parameters by CM Cell Output

The encoding of movement parameters by motor cortex output has attracted much attention since Evarts (1967) reported that pyramidal tract neuron discharge is related to active force. However, in experiments in which the aim is to characterize the output signal transmitted from motor cortex to motoneurons, it is essential to know that a

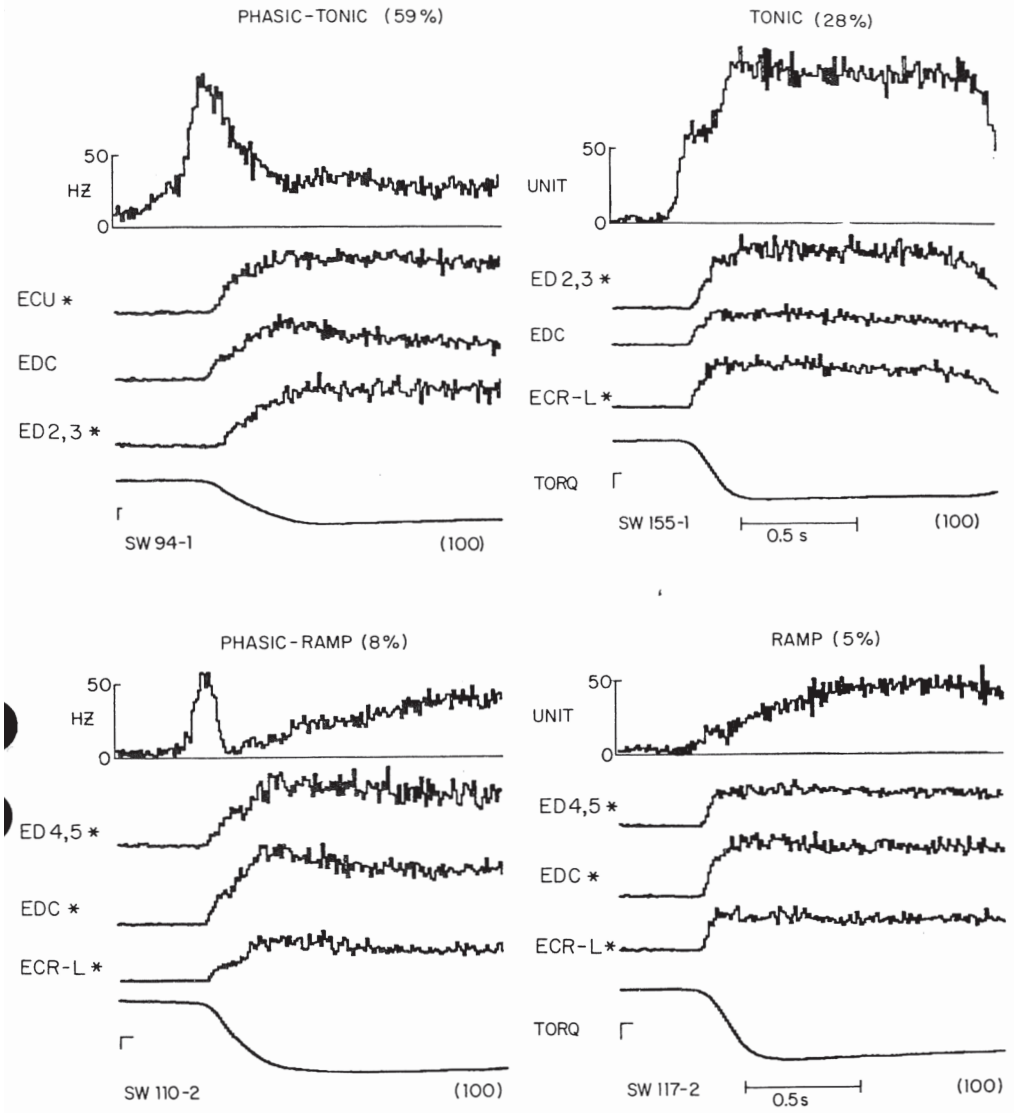

Fig. 5. Four response patterns charcteristic of CM cells during isometric ramp-and-hold wrist responses. Responses during ramp-and-hold wrist displacements were qualitatively the same. Average rectified muscle activity is also shown; *asterisks* indicate the cell's target muscles. (Cheney and Fetz 1980)

particular signal recorded from a cortical cell is actually reaching motoneurons. Therefore, we used spike-triggered averaging to identify CM cells with a documented effect on motoneurons and examined the relation of their discharge frequency to active force. The tonic firing rate of all CM cells investigated was linearly related to static wrist torque over a large part of the torque range examined (Cheney and Fetz 1980). The mean rate-torque slope for extension cells was 4.8 Hz/10^5 dyne-cm, about double that for flexion cells (2.5 Hz/10^5 dyne-cm). That this difference in rate-torque slope was not due to any obvious mechanical advantage of the flexor muscles over the extensors is supported by the fact that flexor motor units do not have greater rate-torque slopes than extensor motor units (Sawyer and Fetz, unpublished observations). In addition to the differences in rate-torque slope of flexion- and extension-related CM cells, some further observations serve to contrast the CM control of flexor and extensor motoneurons: (a) PSF was stronger and occurred more frequently in extensor muscles than flexors (Fetz and Cheney 1980) and (b) Clough et al. (1968) found the largest EPSPs in digit extensor muscles, particularly extensor digitorum communis (EDC). These differences taken together suggest a greater role of motor cortex in generating extensor muscle activity than flexor activity. They also correlate with clinical experience and experimental findings that cortical damage results in tonic wrist flexion and a greater weakness of extensor muscles than flexors (Denney-Brown 1966).

Although the static firing rate of CM cells encodes the active muscle force required to hold a steady position, it also shows smaller variations consistent with compensation for the length-tension properties of muscle (Cheney and Fetz 1980). For example, a particular active force is associated with a higher discharge frequency if the length of the target muscles is decreased by appropriate joint displacement. Therefore, the discharge frequency of CM cells encodes the force of movement through a largely linear rate-torque relation which is shifted appropriately to compensate for the length-tension properties of muscle. In view of this, it may be most accurate to regard CM cell output as encoding a particular level of motor unit discharge or muscle activity. The force associated with this activity will then depend upon the muscle's length-tension property.

Dissociation of CM Cell and Target Muscle Activity

The activity of alpha motoneurons is rigidly linked to the activity of the muscles they innervate and is predictable, based on the principle of orderly recruitment. Since CM cells are premotor neurons, we were interested in whether a similar rigid linkage would apply to their activity or whether the linkage might show greater flexibility. To answer this question we trained monkeys to perform two different motor tasks - alternating wrist movements and power grip - which differed in the temporal pattern of agonist and antagonist muscle activity (Kasser and Cheney 1983). Alternating wrist movements involved a reciprocal pattern of wrist flexor and extensor muscle contraction, whereas power grip required the monkey to squeeze a pair of nylon bars and involved co-contraction of flexor and extensor muscles to stabilize the wrist. Some CM cells (4 of 12) increased their firing rate during both power grip and either the flexion or extension phase of alternating movements. However, the remaining cells (8 of 12) increased their activity only during alternating movements and were unrelated to power grip despite the fact that power grip involved activation of the cell's target muscles. Figure 6 illustrates one such cell whose target muscles were extensors. Its activity co-varied reliably with the extension phase of alternating wrist movements, but during power grip, which involved coactivation of the cell's target muscles and their antagonists, its firing rate decreased sharply. This functional uncoupling of activity in a CM cell from that of its target muscles may be related to the fact that it reciprocally suppressed the antagonist muscles (Fig. 6c). Since such suppression would interfere with co-contraction, the neural mechanisms producing co-contraction may exclude reciprocal CM cells.

Another situation in which we observed dissociation of the activity of a CM cell and its target muscles was during ballistic wrist movements (Cheney and Fetz 1980). Ballistic movements were rapid, uncontrolled oscillations between flexion and extension position zones which one of our monkeys periodically produced after becoming frustrated with the task requirements. The two cells in Fig. 7 increased their discharge consistently during the extension phase of ramp-and-hold movements, but failed to show a consistent relation to ballistic movements involving much greater target muscle activity. Both CM cells began discharging during ramp-and-hold movements well in advance of target muscle EMG activity; hence, the results are not explained by inadequate

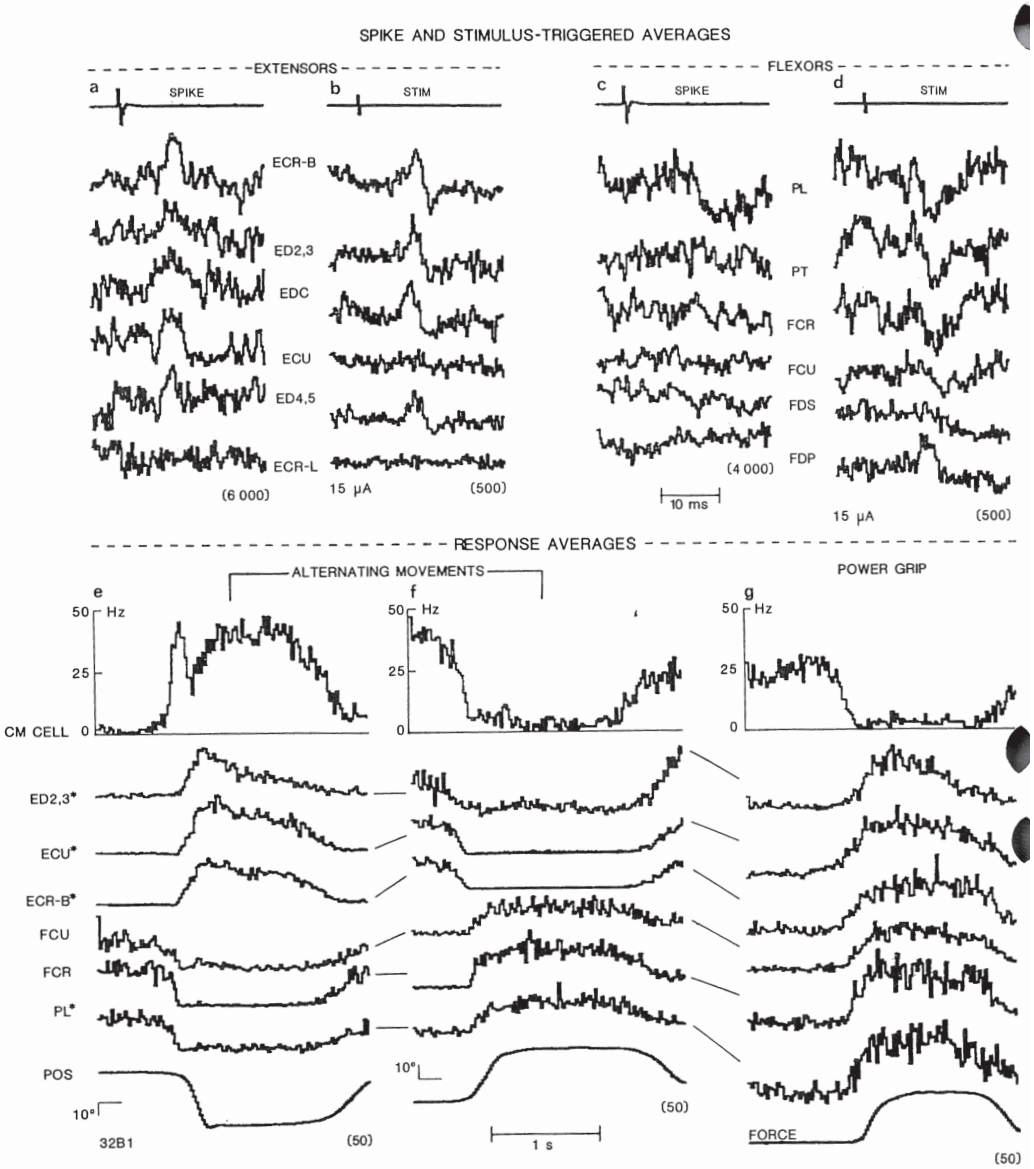

Fig.6a-g. Average activity of a reciprocal CM cell and its target muscles during alternating movements and power grip. Note the opposite relation between cell and target muscle activity during power grip **(g)**, compared with the extension phase of alternating movements **(e)**, even though both involve activation of cell's target muscles. Spike-triggered averages **(a, c)** computed during ramp-and-hold movements identify this cell's reciprocal output effects, i.e., postspike facilitation of agonists and postspike suppression of antagonists. These output effects were confirmed in stimulus-triggered averages **(b, d)** computed from stimuli applied to the site of CM cell recording during ramp-and-hold movements. *Asterisks* indicate muscles facilitated or suppressed by the cell

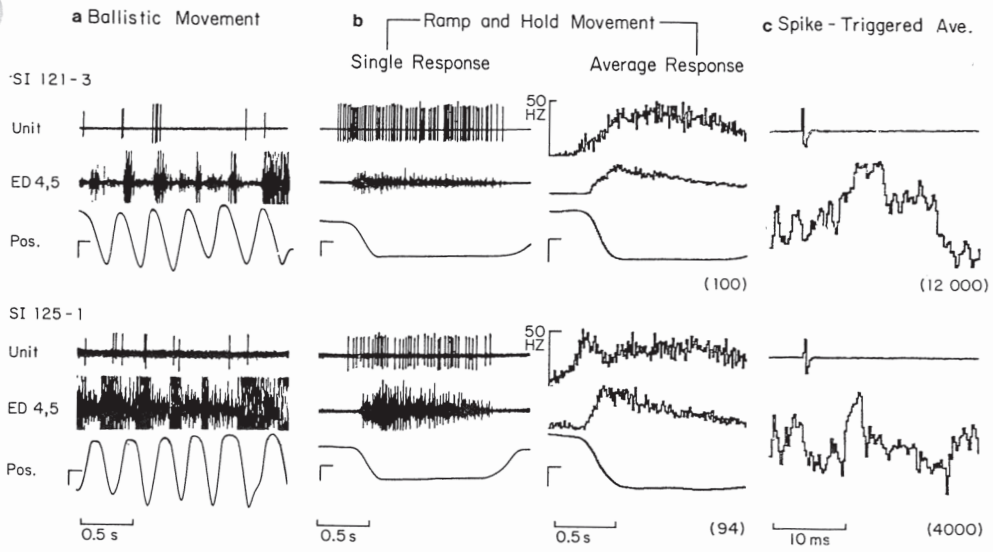

Fig. 7a-c. Activity of two CM cells during controlled ramp-and-hold wrist movements and ballistic movements. Note the intense activity during ramp-and-hold movements **(b)** and inactivity during ballistic movements **(a)** despite greater activation of the cell's target muscles. Spike-triggered averages **(c)** identify each as a CM cell. Position calibration bar is 10°. (Cheney and Fetz 1980)

time for cell activation. Some CM cells, therefore, appear to have a preferential role in accurate movements and these cells are excluded by motor programs for ballistic movements. This view is consistent with the finding of Fromm and Evarts (1977) that motor cortex neurons discharge more intensely during small, accurate movements than during ballistic movements. Similar findings have also been reported by Muir and Lemon (1983) who found greater CM cell activity related to precision grip than to a power grip.

Sensory Properties of CM Cells

Role of CM Cells in Transcortical Stretch-Evoked Muscle Responses

Motor cortex cells, including corticospinal neurons, are known from work in anesthetized animals to respond to afferent signals from both cutaneous and muscle receptors (Wiesendanger 1973; Hore et al. 1976; for additional references see Phillips and Porter 1977). Although the existence of these inputs is now generally accepted, their functional

role is not. Phillips (1969) postulated that muscle afferent input to motor cortex may form the afferent limb of a long-latency transcortical stretch reflex. Indeed, rapid stretch of an active muscle does elicit two or sometimes three peaks of EMG activity (Tatton et al. 1975). The first peak (M1) has a latency appropriate for a segmental stretch reflex (Fig. 8), the second peak (M2) has a longer latency, appropriate for mediation by a transcortical loop. Indeed, pyramidal tract neurons have been shown to respond at appropriate latencies to mediate M2 (Evarts and Tanji 1976; Conrad et al. 1975). However, recent evidence has demonstrated that muscle stretch can elicit multiple EMG peaks in proximal muscles of spinal cats (Ghez and Shinoda 1978), spinal monkeys (Tracey et al. 1980), and decerebrate monkeys (Miller and Brooks 1981). Furthermore, torque perturbations elicit small oscillations of muscle length (Eklund et al. 1982) and multiple spindle afferent responses (Hagbarth et al. 1981), suggesting that the multiple EMG peaks may simply represent sequential spinal stretch reflexes.

Since CM cells have a documented effect on EMG activity, their response to torque perturbations is an important test of the role of motor cortex in long-latency stretch-evoked muscle responses. Therefore, we examined the responses of the CM cells to perturbations which stretched the cell's target muscles. Of 21 cells, 20 responded to target muscle lengthening torque perturbations; only one cell was unresponsive to the torque perturbations applied. Figure 8 illustrates the average torque pulse response of one CM cell and a target muscle. Torque perturpations which stretched the target muscles elicited both M1 and M2 EMG peaks and a brisk CM cell discharge, whose onset preceded M2 onset. In these experiments, transcortical loop time was measured as the sum of its afferent component (the onset latency of the stretch evoked CM cell discharge) and its efferent component (the onset latency of postspike facilitation). The mean PSF onset latency of 7.0 ms sums with the mean CM cell onset latency of 23.4 ms to yield a total mean transcortical loop time of 30.4 ms, which is comparable to the mean M2 onset latency of 27.9 ms. The duration of a CM cell's response to torque perturbations provides an additional measure of the extent of its potential contribution to the M2 muscle response. In all cases but two, the CM cell response, delayed by PSF onset time, overlapped with some part of the M2 EMG response.

Spike-triggered averages revealing PSF from our cells have been computed from spikes occurring during the static hold period of active

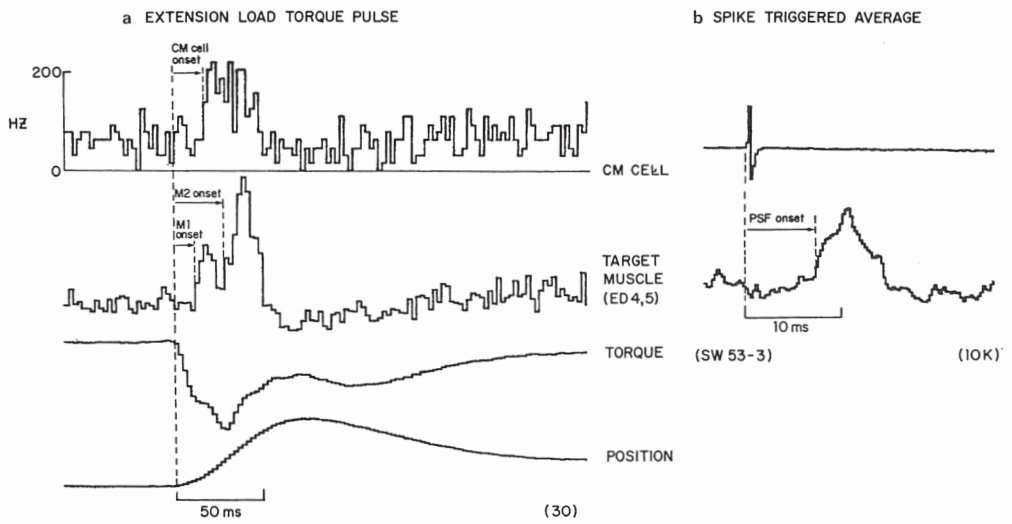

Fig. 8a,b. Average response of a CM cell **(a)** and its target muscle evoked by transient muscle lengthening torque perturbations applied during active wrist extensions. The spike-triggered average **(b)** showing postspike facilitation identifies this as one of the cell's target muscles. (Cheney and Fetz 1983)

movements. But do the spikes evoked by the torque pulse also facilitate muscle activity during the M2 response? Thorough testing of this issue with existing data has been limited by the relatively small number of torque-pulse-evoked spikes available for spike-triggered averaging for a given cell. Nevertheless, torque-pulse-evoked spikes from one CM cell produced potent facilitation with only 685 triggers. Based on these findings, we conclude that CM cells do contribute to the long-latency M2 response evoked by target muscle lengthening perturbations.

In addition to responding to perturbations which stretched their target muscles, 8 of 18 CM cells tested also responded at short latency (22.0 ± 7.4 ms) to perturbations which shortened their target muscles. Although these responses seem paradoxical, they are consistent with the fact that the cell's target muscles also exhibited a shortening response which had an onset latency similar to M2 in lengthened muscles (33.9 ms compared with 27.9 ms for M2). These results suggest that the transcortical CM cell loop, and the muscle responses to which it contributes, may constitute a mechanism for stiffening the joint through co-contraction of flexor and extensor muscles.

Response of CM Cells to Passive Joint Movement

CM cells recorded in awake inactive monkeys also exhibit sensory responses to passive wrist movements at the joint about which the cell's target muscles act. This test, like torque pulse perturbations, evokes a complex afferent input consisting of cutaneous, joint, and muscle receptor components, and in awake monkeys it has not been possible to assess the relative contributions of these. Nevertheless, a cell's response to passive movement is an important functional property and, therefore, we have characterized it for 19 CM cells. Of 19 cells, 17 responded to passive wrist movements. Of these 17, ten responded to wrist rotation in only one direction; seven responded bidirectionally. Seven of the ten unidirectionally responsive cells were activated by passive movements which stretched their target muscles (opposite direction to active movements); three were activated by passive movements and active movements in the same direction. All evoked responses were phasic.

None of four wrist-related CM cell's we tested were activated by natural stimulation of the glabrous or hairy skin of the hand. However, Lemon and Muir (1983) reported that four of seven CM cells they tested could be activated by brushing the glabrous skin of the hand. These cells all produced PSF in small hand muscles and were highly active during exploration movements of the fingers.

Summary and Conclusions

Since functional properties may vary widely with a cell's axonal projection, identification of these projections is particularly important in establishing the cell's functional role in movement. Spike-triggered averaging of rectified EMG activity has emerged as a useful means of identifying CM cells in awake monkeys, where functional relations between cell activity and movement can also be investigated. This method is capable of revealing both postspike facilitation and postspike suppression as well as their distribution across different muscles. Glutamate iontophoresis can be combined with spike-triggered averaging to increase cell activity and enable adequate testing for suppression of antagonist muscles.

Using spike-triggered averaging in awake monkeys we have established the following properties of the primate corticomotoneuronal system.

1. CM cell output organization is of three basic types: *pure facilitation* - these cells facilitate agonist muscles, but have no effect on antagonists; *pure suppression* - these cells suppress antagonist muscles, but have no effect on agonists; *reciprocal* - these cells simultaneously facilitate the agonists and suppress the antagonists. Postspike suppression is weaker and about 3 ms longer in latency than in facilitation. We conclude that it is probably mediated by a spinal inhibitory interneuron. These three cell types constitute fundamemtal organizational units involved in direct motor cortex control of forearm muscles.
2. Half of the wrist-movement-related CM cells produced PSF in two or more synergist muscles. We conclude that many CM cells make excitatory synaptic connections with motoneurons of multiple agonist muscles.
3. Neighboring CM cells produce the same profile of PSF across muscles and therefore appear to have similar synaptic connections with motoneurons of agonist muscles. Such neighboring cells with similarly organized output effects may form clusters in layer V of motor cortex.
4. CM cells can be divided into four types, based on their discharge during ramp-and-hold wrist movements: phasic-tonic, tonic, phasic-ramp, and ramp. The net output effect of a CM cell on target muscle activity during active movement is a function of both its PSF and its firing pattern during movement.
5. CM cell discharge encodes relatively simple parameters of active movement. Under static conditions the discharge rate of CM cells encodes the force of active movement.
6. CM cells are the efferent limb of a transcortical reflex loop activated by external perturbations. The consequence of this long-latency reflex is to increase joint stiffness by coactivating flexor and extensor muscles.
7. CM cell discharge, unlike that of alpha motoneurons, is not invariably linked to the activity of its facilitated target muscles. Rather, CM cells exhibit more complex movement relations in which activation depends not only on muscles the cell facilitates but also on those which it suppresses.

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