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CHAPTER 36

Control of forelimb muscle activity by populations of corticomotoneuronal and rubromotoneuronal cells

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We review and synthesize evidence on the activity of corticomotoneuronal (CM) and rubromotoneuronal (RM) cells and single motor units in forearm muscles in monkeys performing alternating wrist movements. The CM and RM cells were identified by post-spike facilitation of rectified forelimb EMG activity. RM cells facilitated more muscles per cell (mean: 3.0 of 6 synergist muscles) than CM cells (2.4/6). Both groups had "reciprocal" cells which also suppressed antagonists of their facilitated target muscles. Unlike CM cells, some RM cells co-facilitated flexor and extensor muscles (5.8 of 12 muscles). During performance of a standard ramp-and-hold force tracking task the firing patterns of CM and RM cells, as well as single motor units, fell into distinct response types. Each population had phasic-tonic and tonic cells. Unique to the CM population were cells whose discharge increased during the static hold period; unique to RM cells were bidirectionally responsive and unmodulated neurons. Many motor units showed decrementing discharge. To estimate the ensemble activities of these populations the response histograms of different cells were summed (with force ramps aligned) in proportion to the relative frequency of each cell type. The population response histogram of CM cells was phasic-tonic, consistent with the predominant response type. The population response of RM cells was also phasic-tonic, but showed a shallower phasic modulation relative to discharge that was sustained during both directions of movement. The population histogram of motor units of a muscle was proportional to the average of rectified multiunit EMG, and typically exhibited decrementing activity during the static hold. The effects of excitatory postsynaptic potentials (EPSPs) on firing probability of motoneurons previously documented in intracellular studies are combined with the mean firing rates in the population histograms and the known amplitudes of CM-EPSPs and RM-EPSPs to infer the relative contributions of the supraspinal cells to tonic discharge of active motoneurons. This analysis suggests that for intermediate levels of force, the CM cells would increment motoneuron discharge by about 9 impulses/second (i.p.s.) and RM cells by about 2.4 i.p.s. The analysis also reveals differences in the population activity of CM and RM cells compared to their target motoneurons, which may be due to other input cells and to recruitment properties of motoneurons.

Key words: Muscle; Motor cortex; Red nucleus; Spike triggered average; Neuropopulation; Primate

Introduction

The relations between firing patterns of neurons in the motor system and parameters of voluntary movement are commonly used to infer which movement parameters are "coded" by central neurons. A causal explanation of the neural mechanisms that generate voluntary movement should ultimately provide a quantitative account of how populations of cells control activity of spinal motoneurons. It is not enough to contend that certain response patterns observed in particular cells *could* underlie the programming and execution of movements. A causal explanation also requires demonstration that the responses of these cells *do* contribute to the movement. Proving

causality is usually difficult, but is possible for premotoneuronal cells that have demonstrable post-spike effects on motoneurone firing.

Investigators have recently found that the activities of populations of cells can provide functions that match movement parameters more closely than the firing patterns of any single neurones. Humphrey et al. (1970) first showed that the smoothed activity of a population of motor cortex neurones could be used to match several different movement parameters. Weighted averages of the cells' firing rates could match the force trajectories and wrist displacements, as well as their temporal derivatives, if the weighting factors for each cell could be optimally chosen for each trajectory. Moreover, the match between the cells' weighted activities and the mechanical parameters improved with the number of cells included. The ability to freely optimize the weighting coefficients, of course, helped to assure convergence on the movement trajectories; closer matches are obtained with larger populations because each additional nonredundant cell could only serve to further reduce the remaining difference.

More recently, Georgopoulos et al. (1984) have shown that populations of motor cortex cells could be used to match the direction of limb movement by invoking the "vector hypothesis" to sum the activity of directionally tuned cells. For a given movement direction each cell is assumed to make a vector contribution pointing in the direction of its maximal activity, and by an amount proportional to its mean rate during the given movement. The vector sum of the population then approximates the direction of arm displacement. Again, the match improved as more cells with diverse directional preferences were included. This match with movement direction could be taken to suggest that arm *displacement* is coded in motor cortex populations rather than muscle force, as previously proposed (e.g., Evarts, 1968; Cheney and Fetz, 1980). The direct match between the population function and arm displacement is appealing because it conveniently avoids the intervening complexities of synaptic connections and limb mechanics, which present formidable obstacles to a causal explanation. Indeed, the vector hypothesis will produce a match with movement direction whether the directionally "tuned" cells have any output effects on muscles or not.

Although one can find good *descriptive* matches between functions of the activity of neuronal populations and particular movement parameters, this correspondence is no proof of neuronal coding in the *causal* sense. To demonstrate that the candidate cells actually make a causal contribution requires additional evidence that they have appropriate output effects. Evidence for synaptic linkages of cells recorded in behaving animals can be obtained by cross-correlation techniques, and evidence for direct effects on muscle activity can be demonstrated by spike-triggered averaging of EMG. Here we synthesize information on cells in the primate motor system that have demonstrable effects on muscles, namely, corticomotoneuronal (CM) and rubromotoneuronal (RM) cells. The activity of these supraspinal premotoneuronal cells and forelimb motor units was documented under behavioural conditions which were sufficiently similar across different experiments to compare their activity. The cells were recorded in monkeys performing a step-tracking task, and generating ramp-and-hold wrist torques, consisting of a change of force between flexion and extension zones followed by a static hold in a force zone. The monkeys typically performed the responses with similar timing across experiments, allowing comparisons of the cells' response patterns.

The synaptic connections of the cortical and rubral cells to motoneurones was inferred from post-spike effects observed in spike-triggered averages of forelimb EMG activity. This correlational linkage identifies these output cells and their target muscles, and also implies that the premotoneuronal cells affect their target muscles in proportion to their firing rates. Other studies have provided quantitative evidence on the magnitude of correlational effects of single cells synapsing on motoneurones (Cope et al., 1987). These observations can be combined into a quantitative picture

of the time-varying interactions between populations of connected cells in the primate motor system during voluntary wrist movements.

Correlational linkages obtained by spike-triggered averaging

The spike-triggered averaging (STA) technique can best be understood by examining the expected effects of a single input cell to a motoneurone. Fig. 1 illustrates a hypothetical CM cell synapsing on a motoneurone. The illustrated EPSP is taken from a STA of intracellularly recorded membrane potential in a cat motoneurone at rest, triggered from a single Ia afferent fibre (Cope et al., 1987). When the motoneurone was induced to fire rhythmically, this unitary EPSP transiently increased the motoneurone firing probability, as measured by the peak in the cross-correlation histogram between the spikes of the Ia fibre and the motoneurone. In these experiments, the measure of the cross-correlogram peak that was most strongly related to the EPSP was the peak area, i.e., the number of motoneurone action potentials above baseline triggered by each EPSP. This peak area (N_p) was proportional to the EPSP amplitude (h), and is given by the following relation:

$$N_p = 1.1 \times 10^{-4} \, h \, \mu V \qquad (1)$$

Thus, the effect of unitary EPSPs of 100 μV is to trigger about one firing per 100 EPSPs. This suggests that if the unitary EPSPs of height h arrive at a rate of f per second, the mean increment in motoneurone firing would be approximately:

$$df_m \cong 10^{-4} * h * f \qquad (2)$$

Relation 2 assumes that the increment in firing given by Relation 1 is independent of the firing rates of the presynaptic cell and the motoneurone; this assumption seems reasonable in light of preliminary evidence, but requires further investigation. The relation also assumes that the correlogram peak is followed by a negligible trough below baseline. Under these conditions, Relation 2 provides the proportionality between the firing rate of the premotoneuronal cell (f) and its effect on the motoneurone firing rate.

In chronic recording studies, one can compile spike-triggered averages of multi-unit EMG (Fetz and Cheney, 1980; Buys et al., 1986) more readily than correlograms with single motor units – although the latter have also been documented (Mantel and Lemon, 1987; Smith and Fetz, 1989). The typical effect observed in STA of coactivated muscles was a post-spike facilitation (PSF), which rose above baseline after some onset latency, reached a peak and declined again. This post-spike facilitation represents the summed effects of all the motor units in the EMG record that were facilitated. The contribution of a single facilitated motor unit would be the convolution of its correlogram peak with its rectified motor unit poten-

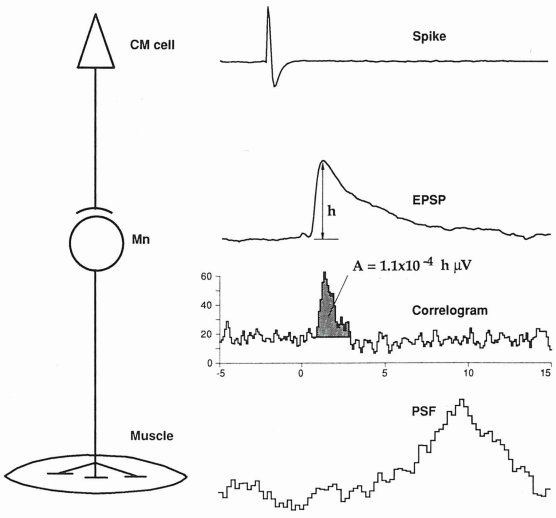


Fig. 1. Events mediating the post-spike effects of a premotoneuronal cell. The monosynaptic connection produces an EPSP and increased motoneurone firing probability – represented here by STA of a Ia EPSP and the associated correlogram (data from Cope et al., 1987). The area (A) of the correlogram peak was found to be proportional to the height (h) of the EPSP. STA of multiunit EMG produces post-spike facilitation (PSF), representing the contribution of all facilitated motor units.

tial. Since the EMG includes the activity of multiple units with unknown and variable relative timing, the net PSF is some nonlinear function of the contribution of the underlying motor units. Although the shape and size of the PSF have limited quantitative significance, they serve to identify those cortical and rubral cells that have output effects on their target muscles, and also identify their muscle fields.

The magnitude of PSF was measured as a mean percent increase (MPI) above baseline. CM cells produced a somewhat greater facilitation of their target muscles (MPI = 7.0%) than RM cells (5.1%). The mean onset latency of PSF was longer for CM cells (6.3 ms) than RM cells (5.2 ms); this difference probably reflects the difference in conduction distance, although conduction velocities of descending cells and motoneurones could add considerable variance.

In addition to the excitatory effects in the PSF, spike-triggered averaging has also demonstrated inhibitory linkages from cells to muscles, usually to antagonists of the coactivated target muscles. Such inhibitory linkages were first deduced from stimulus-triggered averages obtained by single-pulse microstimulation applied during both flexion and extension (Cheney et al., 1985). At about one-third of the sites of CM cells that facilitated target muscles, microstimulation produced a post-stimulus suppression in their antagonists. Post-spike inhibitory effects from single CM cells were directly demonstrated from action potentials evoked by glutamate during the antagonist phase of movement, when the CM cell would normally be inactive (Kasser and Cheney, 1985). The mean percent decrease below baseline for post-spike suppression (PSS) was −4.1% for CM cells and −4.5% for RM cells. The latencies of PSS tended to be about 3 ms greater than the latencies of PSF for the same cells (i.e., 9.3 ms for CM cells and 8.4 ms for RM cells).

Muscle fields

In our experiments spike-triggered averages were typically compiled simultaneously for six coactivated synergistic muscles. The muscles that were coactivated with the cell are referred to as agonists, and those facilitated by the cell are its target muscles. The number of facilitated target muscles ranged from one to all six of the coactivated agonist muscles. The mean number of facilitated muscles per cell was somewhat larger for RM cells (3.0 of 6 muscles) than for CM cells (2.4/6). In both groups the excitatory muscle fields tended to be slightly larger for extension cells than for flexion cells. Buys et al. (1986) found that the proportion of facilitated muscles was larger for intrinsic hand muscles than forearm muscles.

Some of the supraspinal cells that facilitated their coactivated agonist muscles also exhibited post-spike effects on antagonists of their target muscles. The patterns of post-spike effects produced in agonist and antagonist muscles are summarized in Fig. 2. About 59% of CM cells and 39% of RM cells produced a *pure facilitation* of agonists, without any detectable effect on the recorded antagonist muscles. About 30% of both

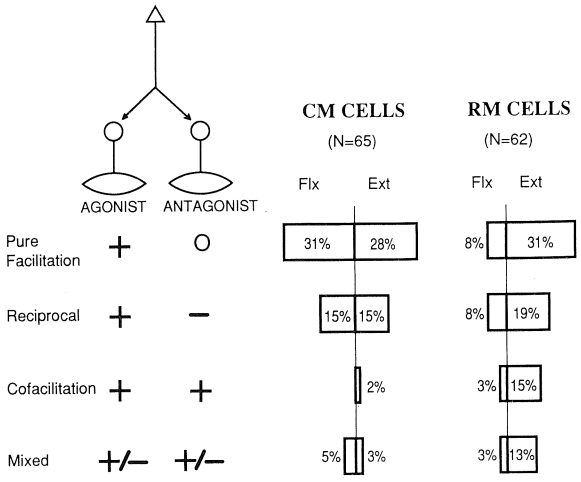
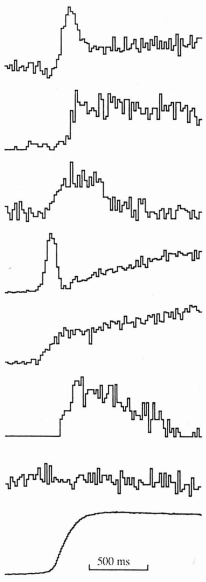


Fig. 2. Projection patterns of CM and RM cells. The post-spike effects on coactivated agonist muscles and their antagonists include facilitation (+), suppression (−), or no effect (0). The mixed category includes cells which facilitated and suppressed agonist muscles, and some "pure suppression" cells. The proportions of each type of projection pattern are given separately for cells which facilitated flexor and extensor muscles.

CM and RM cells produced post-spike suppression in antagonists of their target muscles. The activity of these *reciprocal* cells simultaneously facilitated agonists and inhibited antagonists. The magnitude of inhibition tended to be stronger in flexor than extensor muscles.

A third type of cell produced post-spike facilitation in both flexor and extensor muscles. Such *co-facilitation* cells were common among RM cells, but were relatively rare in the CM cell population. Most of these co-facilitation cells produced greater post-spike facilitation of extensor than flexor muscles. RM co-facilitation cells were often active during both flexion and extension movements (like other RM cells), suggesting that their output might function to raise the excitability of motoneurons, independent of the direction of movement.

Finally, a miscellaneous category of cells produced *mixed* effects on their target muscles. Most of these facilitated particular agonist muscles and suppressed other agonists. A few exerted no measured effect on their coactivated muscles, but only suppressed the antagonist muscles (so-called "pure suppression" cells). Still other cells exhibited stronger modulation with antagonists of their facilitated muscles. These miscellaneous types are combined in the "mixed" group.



RESPONSE TYPE	POPULATION CM	RM	MU
PHASIC-TONIC	48%	46%	23%
TONIC	28	8	33
PHASIC	2	20	5
PHASIC-RAMP	10	0	0
RAMP	6	0	0
DECREMENTING	5	3	39
UNMODULATED	0	23	0
TORQUE N =	211	61	86

Fig. 3. Response patterns of CM and RM cells and motor units during ramp-and-hold wrist responses. Examples of each type of discharge pattern are shown at left, and the proportions of cells in each population are tabulated on the right.

Discharge properties during active movements

The response patterns of CM and RM cells, as well as motor units, fell into several distinct categories on the basis of their discharge during the dynamic and static phase of the torque trajectory. Fig. 3 illustrates these patterns and tabulates the relative proportion of cells within each population exhibiting these patterns.

A common response pattern in all three groups was *phasic-tonic,* consisting of a phasic discharge at onset of movement, followed by a tonic firing during the static hold period. This represented the predominant type for both CM and RM cells. Another category observed in all three groups were *tonic* cells, which discharged with a steady firing rate during the hold period, with a time course resembling the torque trajectory. The tonic firing rate of cells in all groups (CM, RM and motor units) was an increasing function of the static force exerted by the monkey.

The remaining categories of firing patterns appeared predominantly in one or another of the three populations. Many RM cells and some motor units exhibited a purely *phasic* discharge pattern at onset of movement, with no sustained increase in activity during the hold period. Unique to the cortical population were CM cells that exhibited a gradually increasing *"ramp"* discharge during the static hold period; some of these also had a phasic discharge at onset of movements. A *decrementing* discharge during the hold period was characteristic of many motor units, but was seen more rarely in the supraspinal premotor populations. This decrementing discharge dropped steadily at a rate exceeding any change in static torque.

Remarkably, many RM cells were *unmodulated* during alternating movements. These cells exhibited strong PSF and were located within the region of the magnocellular red nucleus. All of these cells showed a higher continuous discharge during movement of 6 – 20 Hz; this allowed spike-triggered averages to be computed for both flexor and extensor muscles.

These response categories encompass the patterns seen at intermediate force levels. A few motor units showed a transition from one type of pattern to another. For example, some phasic motor units exhibited phasic-tonic or decrementing discharge at the highest force levels. The proportions of these discharge patterns given in Fig. 3 are representative for intermediate load levels.

Whether particular types of supraspinal cells preferentially affect specific types of motor units remains to be determined. In microstimulation experiments all types of motor units were facilitated by S-ICMS applied at a single cortical site (Palmer and Fetz, 1985b). Cross-correlations between CM cells and motor units indicate that most of the motor units in a facilitated muscle were affected by a CM cell (Mantel and Lemon, 1987). This would suggest that CM cells, like Ia afferent fibers, probably send divergent terminals to most, if not all motoneurons of their target muscles.

The variety of response patterns seen for individual CM and RM cells, and the differences between these responses and those of their target muscles, indicates that these response patterns alone provide no reliable guide to inferring causal linkages. A clear example is the phasic-ramp CM cells, whose sharply modulated discharge pattern is radically different from that of its target muscles. At the other extreme, the steady discharge of the unmodulated RM cells would make them unlikely candidates for causal involvement in the task, although such contribution is revealed by their post-spike effects. Given the PSF as evidence that these different types of cells *do* have synaptic linkages to motoneurons, one could infer a rationale for their observed discharge patterns. For example, the prominent phasic discharge at movement onset exhibited by many premotoneuronal cells appears necessary to activate the target muscles; in the case of reciprocal cells, this phasic activity would simultaneously inhibit the antagonist muscles. In contrast to the unidirectional activation of CM cells with either flexion or extension, many RM cells fired during both movements; those bidirectional cells which also cofacilitate both sets of muscles may be more involved in sustaining motoneuron excitability than controlling the movement direction.

The ramp discharge in CM cells may be viewed as a mechanism for overcoming the tendency of motoneurons to adapt during the hold period; a steady increase in synaptic input would help to sustain motoneuron firing during the static hold period. It may be significant that a phasic-ramp pattern can be obtained by subtracting the decrementing discharge of motor units from the common phasic-tonic pattern of CM cells. This would suggest that phasic-ramp cells may represent a difference between a phasic-tonic command and the decrementing response of agonist motor units; such an "error signal" in CM cells would provide a proportional excitatory effect on their target muscles, reducing the difference between command and response.

Relation to active force

The activity of CM and RM cells clearly "encodes" active force, in the sense that this activity causally contributes to force through the correlational linkage with motor units. Yet the relation between neural activity and force is significantly different for these supraspinal cells compared to their target motor units. These differences are illustrated by graphs of the tonic firing rate observed during the static hold period plotted as a function of the active force for representative units from each group (Fig. 4). Both CM and RM cells showed some discharge during the hold period at even the lowest force levels. In contrast to motor units, the supraspinal premotoneuronal cells were not sequentially recruited into activity at higher

forces. However, it should be noted that although the external load was off at the origin of the abscissa, a small amount of force was required to overcome internal loads, such as stretch of the antagonist muscles. Above these minimal force levels, CM and RM cells increased their firing rates rapidly. In contrast, motor units contribute to increasing force by two mechanisms: recruitment of new motor units at successively higher force levels, and increases in firing rate as a function of force. In addition, the discharge of motor units typically saturated at rates well below those which supraspinal cells attained.

The rate – torque slope, namely, the increase in firing rate per increase in static force over the linear range, was higher for extensor cells than flexor cells, for both CM and RM populations. The average rate – torque slope for all extensor CM cells (480 Hz/Nm) was about twice that of flexor CM cells (250 Hz/Nm). Similarly, the average rate – torque slope for all extensor RM cells (128 Hz/Nm) was larger than that of flexor RM cells (22 Hz/Nm). In contrast, motor units showed no statistically significant difference in the mean rate – torque slopes of extensor and flexor motor units (260 and 410 Hz/Nm, respectively). This suggests that the difference exhibited by supraspinal populations is not simply related to the mechanical advantage of flexion over extension, but represents an intrinsic asymmetry in their relation to flexor and extensor muscles.

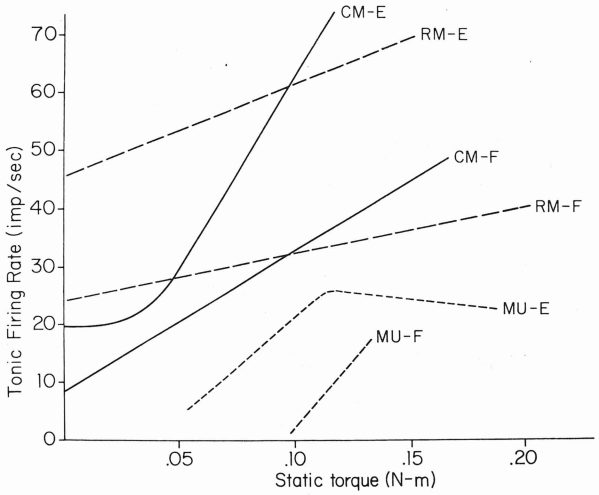


Fig. 4. Firing rates of CM and RM cells and motor units as function of static torque. The tonic rates during the static hold period were measured from response averages compiled at different force levels. Representative curves are shown for flexor and extensor cells.

Population responses

Since the monkeys performed the ramp-and-hold responses similarly across different recording sessions, it is possible not only to compare the response patterns of these cells, but also to combine the individual contributions to obtain a more complete picture of the ensemble activity. Summing the individual response histograms can provide a more comprehensive population average.

Such a summation has been performed previously for motor units recorded in forearm muscles (Palmer and Fetz, 1985a). As shown in Fig. 5, adding the response histograms of seven motor units (left) yielded a population histogram which matched the rectified EMG activity of their parent muscle quite well (right). This suggests that the response averages of target muscle EMG obtained with CM and RM cells can be taken as representative of the activity of their target motoneurone pool.

The response histograms of the supraspinal cells were added in a similar manner, as illustrated in Fig. 6. Selecting cells associated with comparable force ramps, we calculated population histograms by aligning the force ramps and summing the response histograms of the units. We also summed the average EMG of their target muscles and the torque trajectories, and normalized by the number of cells. Subpopulation averages were first compiled for cells of each response type (Fig. 6, middle). Then these averages were combined in proportion to the relative frequency of each response type to get the overall population average (Fig. 6, right).

The overall population histograms of CM and RM cells both exhibited a phasic-tonic discharge pattern, as shown in Fig. 7. However, the CM

population showed greater depth of modulation with the task: the difference between tonic rates during agonist and antagonist hold period was larger, and so was the relative height of the phasic burst.

The activity of these supraspinal populations began well before their target muscle activity, and clearly differed from their target muscles. Since these cells facilitated their target motor units, one can interpret their activity as representing the direct synaptic influence of these cells on motoneurons. Any motoneuron receiving converging input from the supraspinal populations would receive a time-varying current proportional to the population histogram, and displaced by a conduction delay of several milliseconds (considerably less than the histogram bin width). The difference between the ensemble discharge of these pre-

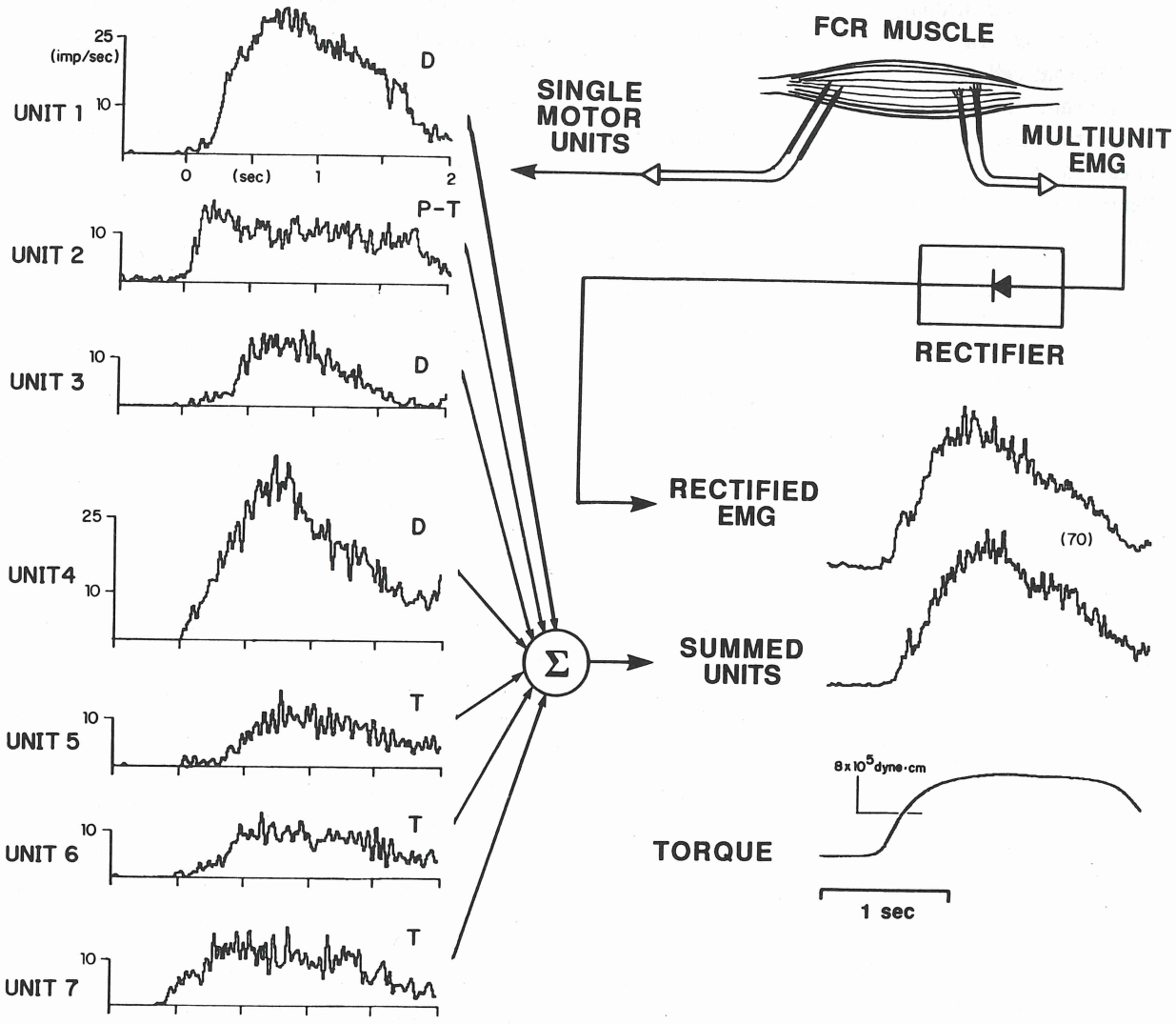


Fig. 5. Response histograms of 7 single motor units (left) recorded in the same flexor carpi radialis muscle. The sum of the response histograms gives a good approximation to the average of multi-unit EMG recorded with wire electrodes (modified from Palmer and Fetz, 1985a).

motoneuronal populations and their target motoneuron pools is illustrated in the bottom traces of Fig. 7. The initial phasic peak in this difference trace indicates that synaptic input from CM and RM cells precedes the overt activity of motor unit by several hundred milliseconds. This initial input would bring the motoneurons to threshold; it could also counter any inhibitory effects from antagonist input cells producing reciprocal inhibition on these motoneurons.

The profile of EMG activity in these response averages can also be interpreted as a measure of the net excitatory drive that the motoneurons are receiving from *all* their inputs. This can be concluded from the experiments of Hoffer et al. (1987), who reported that intracellular injection of current proportional to EMG activity that had been recorded previously in moving animals could generate motoneuron discharge mimicking activity of motor units recorded in the active muscles. Thus, the difference between the synaptic drive from premotoneuronal cells (reflected in their population histograms) and the net synaptic excitation in their target muscles (reflected by the EMG averages) suggests the presence of other inputs during the dynamic phase of movement. Analysis of

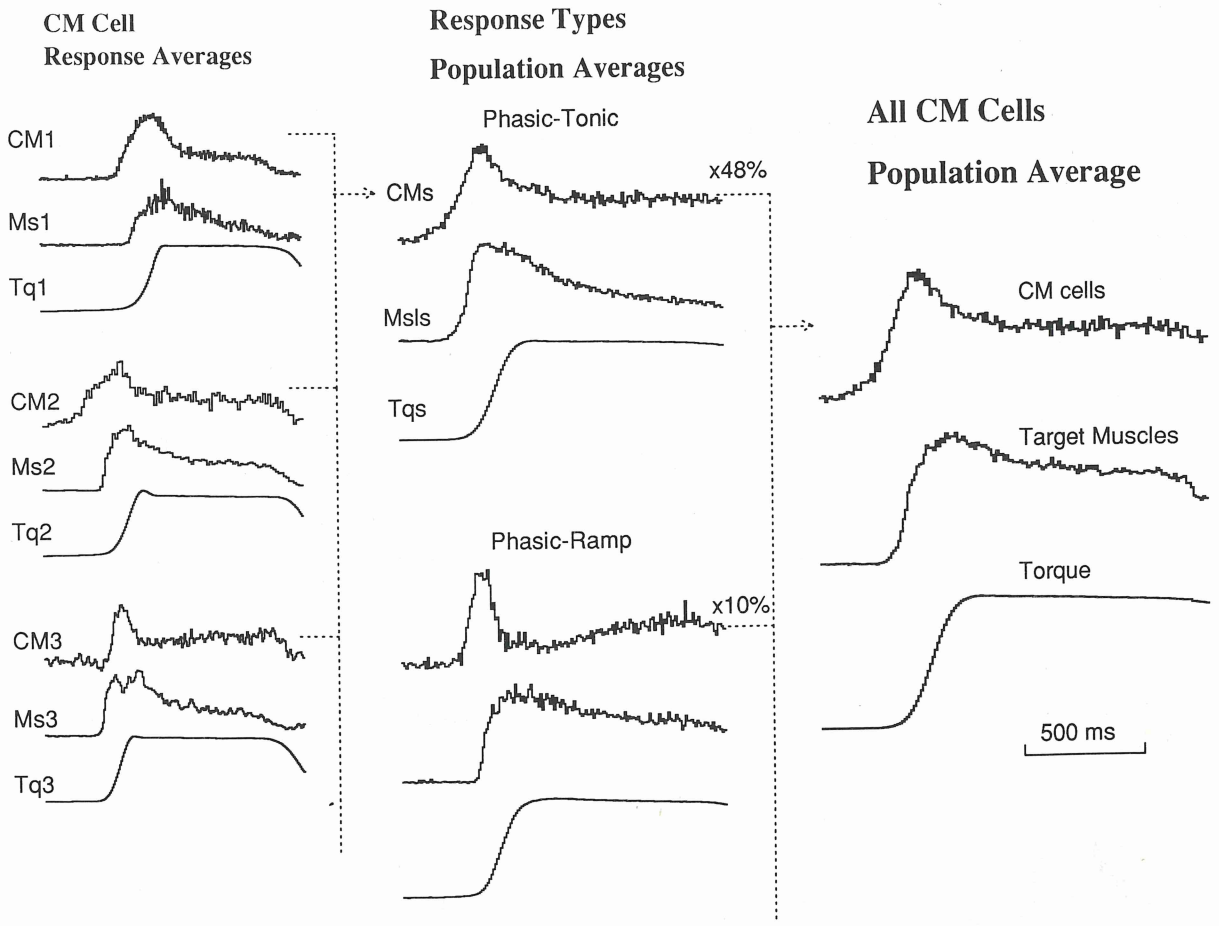


Fig. 6. Derivation of the population histograms of CM cells. The response averages of individual cells in each category were summed, along with their target muscle and the corresponding torque trajectories. The population averages for each response type were then weighted in proportion to the relative number of cells in each type. This provided the net population average of the CM cells.

the neural mechanisms involved in the transition between flexion and extension zones is complicated by many unknown variables, including the changing activity of inhibitory interneurons, the currents required to bring motoneurons to threshold, and potential inputs from many other premotoneuronal cells whose discharge patterns are not known.

In contrast to the complex factors during the dynamic phase of movement, the activity during the static hold period presents a steady-state situation in which activities of the input populations and motoneurons may be quantitatively compared. In particular, the firing rates of CM and RM cells at specific force levels can be used to infer their effect on the activity of motoneurons. Given the correlational effect of unitary EPSPs in motoneurons (Relation 1), we can estimate the contribution of the colony of CM cells to the tonic firing of a motoneuron as the sum of Relation 2 over the population:

$$df_{m;\,c} \cong \Sigma\, 10^{-4}\, h_i f_i$$

where $df_{m;c}$ is the increment in firing rate of a

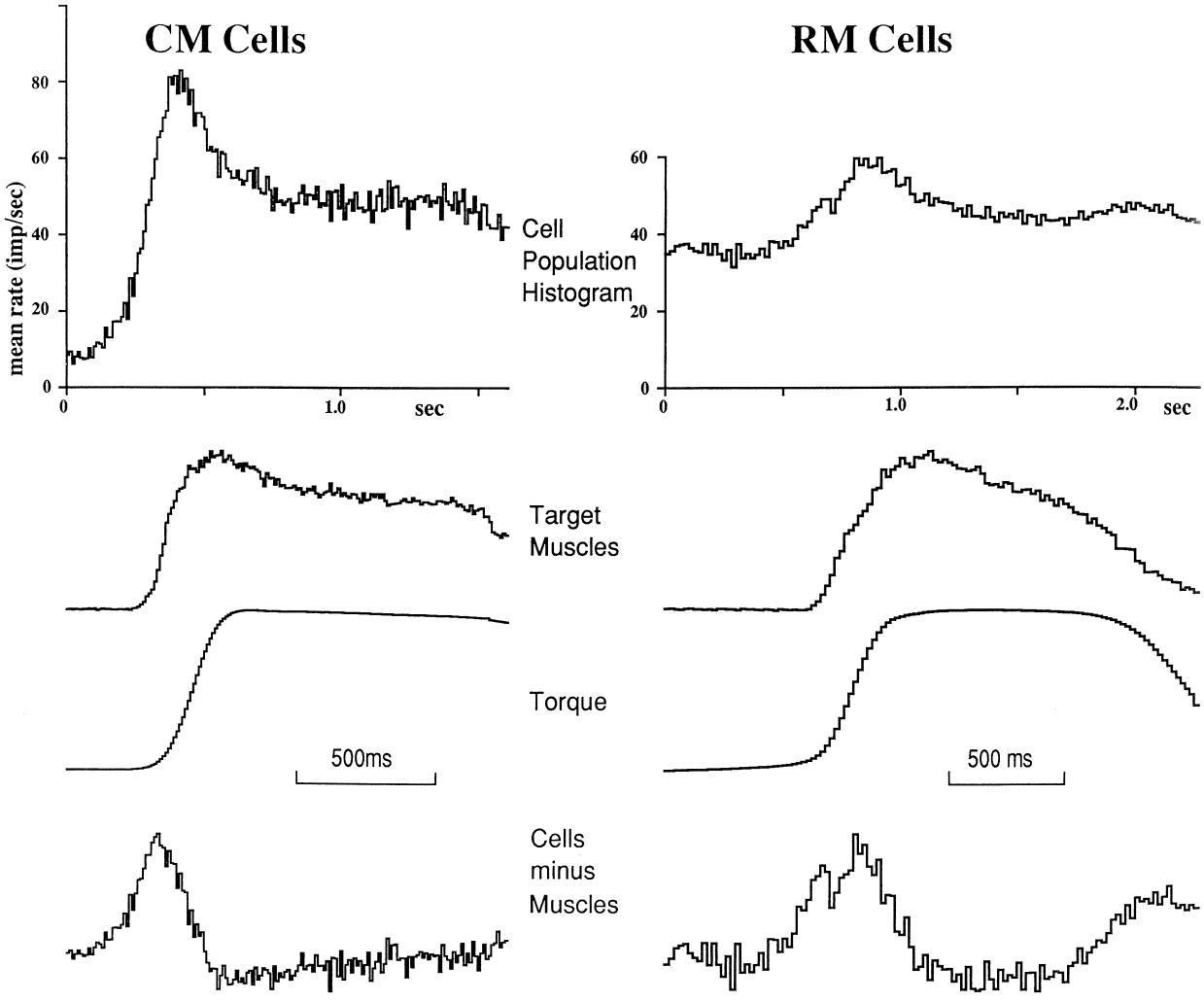


Fig. 7. Population averages for CM and RM cells, with corresponding target muscles and torque trajectories. The difference between the population activity and their target muscle activity is plotted at the bottom. This shows the net surplus of descending activity at movement onset and the incrementing difference during the static hold.

motoneurone caused by its colony of CM cells; h_i is the height of the CM-EPSP of the ith CM cell and f_i is its firing rate. If h_i and f_i are not correlated, we can estimate the summation over the population by using their mean values. In this case the sum is equal to the number of CM cells converging onto the motoneurone (N_c), times the mean amplitude of the unitary CM-EPSP (h_c), times the mean firing rate of a CM cell (f_c):

$$\sum_{i=1}^{N_c} 10^{-4}\, h_i f_i \cong 10^{-4}\, N_c h_c f_c$$

The first two terms on the right – the number of CM cells in the colony and the mean height of their unitary CM-EPSPs – are generally unknown; however, their product is the maximal CM-EPSP H_c, which has been experimentally measured. Thus,

$$df_{m;c} = 10^{-4}\, H_c f_c$$

Note that the use of H_c in this expression does *not* imply that the CM cells are firing synchronously. The derivation is valid for asynchronous firing, but leads to the product of two terms, N_c and h_c, whose product is equal to the measured quantity H_c. When measured in cervical motoneurones of barbiturate-anaesthetized baboons, the value of H_c ranged from 1 to 3 mV (Clough et al., 1968; Phillips and Porter, 1977). In hindlimb motoneurones of barbiturate-anaesthetized macaques, Shapovalov et al. (1971) found the mean H_c evoked from cortex to be 1.0 mV. In forearm motoneurones of chloralose-anaesthetized macaques, Fritz et al. (1985) found the mean H_c evoked from the pyramidal tract to be 2.0 mV. Taking the latter estimate for the behaving monkey, the contribution of the CM population with a mean firing rate of f_c would be

$$df_{m;c} = 10^{-4} * 2000\ \mu V * f_c = 0.2\, f_c$$

Given the mean static firing rate of 45 Hz for the CM population (Fig. 7), the CM cells would increment motor unit activity by about 9 impulses/second (i.p.s.).

A comparable analysis applies for the RM population. The maximal RM-EPSPs obtained in macaques by Shapovalov averaged to 0.6 mV. This suggests that the population of RM cells would increment the motoneurone firing by:

$$df_{m;r} = 0.06\, f_r$$

where f_r is the mean firing rate of RM cells. For an f_r = 40 i.p.s., the contribution of RM cells to motor unit firing is about 2.4 i.p.s.

Finally, the sum of these terms would represent the estimated contribution of both CM and RM populations to motoneurone activity. For example, at an intermediate extension load level of 0.1 Nm, we have, from Fig. 4, f_c = 45 i.p.s. and f_r = 40 i.p.s., giving a net df_m = 9 + 2.4 = 11.4 i.p.s. This represents over half of the maximal firing rate of the extensor motor unit illustrated in Fig. 4.

Of course this estimate is qualified by several major assumptions. One significant assumption is that the population average reflects the mean input to the motoneurone; it may well turn out that each motoneurone receives inputs preferentially from particular types of premotoneuronal cells. Another assumption is that the ratio between EPSP amplitude and the increase in motoneuron firing probability (Relation 2) applies across experimental conditions and is independent of motoneurone firing rate. Until more information is available these assumptions appear reasonable, and provide the best quantitative inference from the existing evidence.

The curves in Fig. 4 indicate that additional mechanisms must come into play at higher force levels, which limit the increase in motoneurone firing rates. While the supraspinal populations continue to increase their firing, the motor unit rates saturate, and sometimes begin to decline. Since these levels are well below the maximal rates obtained by intracellular stimulation, these results would suggest the presence of some inhibitory control to restrict their rates, perhaps mediated by Renshaw or other inhibitory interneurones.

Concluding comments

Our analysis of population effects on motoneurons from identified premotoneuronal cells can be compared to previous studies of population coding in motor cortex cells. The vector hypothesis of Georgopoulos et al. (1984) is based on unit vectors derived from the cells' net neural activity over the entire movement, whereas the present approach provides a quantitative picture of the time-varying drive on motoneurons. The study of Georgopoulos et al. (1984) also illustrates a time-varying population histogram of cortical unit activity, analogous to ours; this net population histogram is presumably quite similar for each direction of movement. A time-varying population vector can also be generated from the sum of unit vectors whose lengths are proportional to the cells' instantaneous activity. This function can be calculated whether the cells have any output effects or not. In contrast, we are utilizing cells whose contribution to muscle activity is confirmed by spike-triggered averaging, and so are dealing with a population of premotor cells whose activity causally codes muscle force, and whose population histogram is proportional to their synaptic drive on their target motoneurons. The population study of Humphrey et al. (1970) computed matches between weighted averages of neural activity and movement parameters by calculating the coefficients required to optimize the match. In contrast, our model incorporates a weighting factor derived from physiological experiments on motoneurons, and calculates the estimated contributions to motoneuron activity.

This analysis may be extended eventually to include other populations of cells affecting motoneurones. It may also be applied to other types of repetitive responses; the study of Prochazka et al. (1989, this volume) calculates population histograms of afferent fibers during the hindlimb step cycle. Afferent fibers can also be recorded in the behaving primate in cervical dorsal root ganglia (Schieber and Thach, 1985). Preliminary evidence from monkeys performing our ramp-and-hold task indicates that afferent fibres in the C8 root can produce strong post-spike effects in multiple muscles and that these have response patterns similar to supraspinal premotoneuronal cells (Flament and Fetz, in press). Other sources of premotoneuronal inputs include various spinal interneurons; the activity of inhibitory interneurons would be of particular interest in resolving the difference between the excitatory input from supraspinal cells and the net excitation reflected in muscle activity. The effects of these different populations may eventually be synthesized into a quantitative picture of the neural mechanisms underlying execution of voluntary movement.

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