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Intracortical mechanisms for the recruitment of motor cortex neurons

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Abstract. Neurons project out of motor cortex to the spinal cord and to other targets. Not all projection neurons recruit in the same way during behavior, but instead recruitment patterns depend on the projection target of the neurons. The problem is to understand how neurons projecting to different targets are recruited selectively. We have investigated possible mechanisms for the recruitment of motor cortex neurons with electrophysiological approaches in anesthetized cats. To determine if neurons projecting out of motor cortex to different targets have selective input connectivity from extrinsic sources we electrically stimulated corticocortical, callosal and thalamocortical pathways. Subthreshold effects of input pathways were detected by monitoring latency variations of antidromic responses. Intracortical connections to identified output neurons were evaluated by cross-correlation and a new variation of the antidromic latency method. Output neurons in different layers along single electrode tracks usually had different inputs from extrinsic sources. Neurons in close proximity were most likely to share the same inputs, especially when they projected axons to the same target. These results support the conclusion that combinations of inputs from extrinsic sources could selectively recruit efferent neurons from separate cortical layers or from within groups of nearby neurons, according to the target of their axonal projections. In contrast, the data on intracortical connectivity suggest that common drive causes a more synchronous activation of nearby cortical neurons. Combining the conclusions on effects of inputs from extrinsic and intracortical sources leads to the speculation that motor cortex neurons that might at one time be recruited selectively by action of extrinsic afferent pathways to cortex could at another time be bound into synchrony by a common drive shared with their neighbours.

Key words: motor cortex, intracortical connectivity

INTRODUCTION

The motor cortex contains many classes of projection neurons that are likely to be involved in the cortical control of motor behaviours, even if only a few classes of these neurons have been studied in behaving animals. From studies of pyramidal tract neurons, corticospinal neurons, corticorubral neurons and corticostriate neurons it is clear that recruitment patterns during behavior vary with axonal target of motor cortex neurons (Evarts 1968, Armstrong and Drew 1984, Cheney et al. 1988, Bauswein et al. 1989), so that motor cortex sends different signals to its different output targets. Because of the importance of the activity of these neurons in the regulation of motor behavior there has been a long-standing interest in how these activity patterns are determined. Of course, we know that, in general, activity of neurons results from their intrinsic properties and from their synaptic inputs. Synaptic inputs to cortical neurons originate from sources extrinsic to cortex as well as intracortical circuits. We have been investigating effects of inputs from extrinsic and intracortical sources upon motor cortex neurons that in turn project their axons to different subcortical targets and to cortical areas outside of motor cortex. The input connectivities that we have found from extrinsic sources help explain how unique recruitment properties could be created for subpopulations of motor cortex neurons. In contrast, the most prominent feature of intracortical connectivity is shared excitatory drive that should tend to cause synchronous discharge of groups of neurons.

METHODS

The experimental procedures that have been published (Zarzecki and Wiggin 1982, Zarzecki 1989, 1991) will be described only briefly. Extracellular recordings were made in the forelimb region of cat motor cortex. Neurons were identified by antidromic activation. Animals were anesthetized with sodium pentobarbital (35 mg/kg, ip, supplemented as necessary) for the duration of the experiment. They were not paralysed or ventilated artificially. The frontal cortices of both cerebral hemispheres were exposed by craniotomy. A recording chamber was attached to the skull, filled with warmed mineral oil and supported manipulators for cortical recording and stimulating electrodes.

Effects of inputs from extrinsic sources were tested on motor cortex neurons identified antidromically. We identified six output classes of motor cortex neurons. These were : (1) ipsilateral corticocortical neurons projecting to somatosensory cortex, (2) callosal neurons projecting to opposite motor cortex, (3) corticorubral neurons, (4) corticoreticular neurons projecting to the lateral reticular nucleus, (5) corticospinal neurons and (6) corticothalamic neurons. Three inputs to motor cortex were tested for their influences on these identified cortical efferent neurons. The tested inputs originated from ipsilateral somatosensory cortex, opposite motor cortex and ventral thalamus.

Subthreshold effects of input pathways were detected by monitoring latency variations of antidromic responses ("antidromic latency method", Merrill 1972, Swadlow et al. 1978, Lipski 1981, Zarzecki and Wiggin 1982). In the antidromic latency method a reduction of antidromic latency is taken as evidence of excitatory (depolarizing) synaptic input and a lengthening of antidromic latency suggests an inhibitory (hyperpolarizing) synaptic input. Figure 1 is an example of the change in shape of an antidromic wave form caused by subthreshold excitatory input.

Synapses of local origin account for as many as 70% of the synapses in cerebral cortex (reviewed by White 1989). To test the hypothesis that there are specific patterns of intrinsic connectivity to the different groups of output neurons, extracellular recordings were made in motor cortex with two electrodes. In the studies of intracortical connectivity four classes of output neurons were identified antidromically. These were (1) corticospinal, (2) corticothalamic, (3) corticorubral and (4) corticoreticular neurons. Intracortical connectivity was assessed by standard cross-correlation methods and a

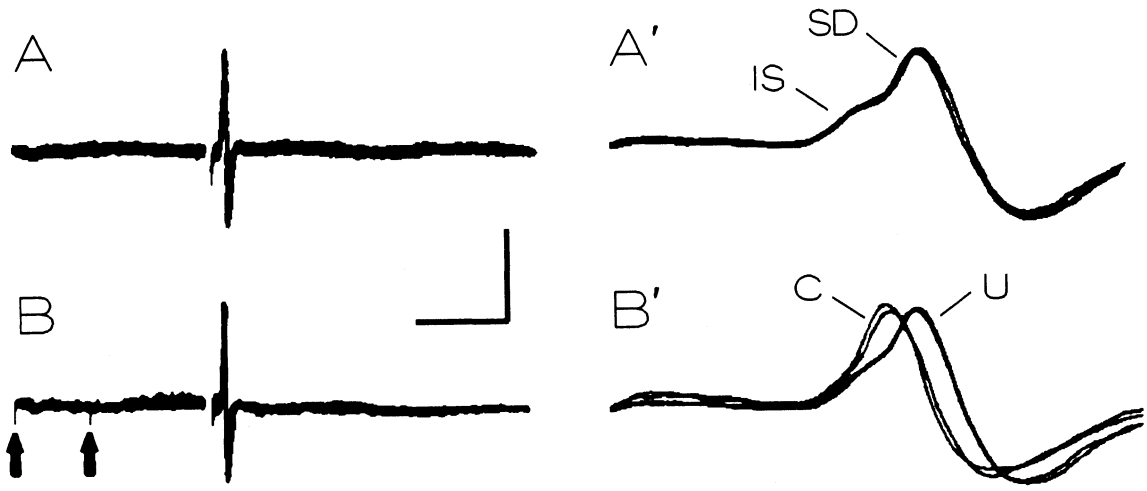

Fig. 1. Antidromic invasion is modified by conditioning stimuli. The wave form of the antidromic invasion of a cortical output neuron is shown in slow (*A*-*B*) and fast sweeps (*A'*-*B'*). Four sweeps are superimposed in each panel. Initial segment (IS) and soma-dendritic (SD) components are labelled. *A*, *A'*. The unconditioned antidromic response. *B*. *B'*. Stimulation of an afferent pathway (arrows) is used for conditioning in two of the four sweeps. The SD component of the conditioned response ("C") occurred earlier than in the unconditioned sweep ("U"). Voltage calibration 200 μV. Time calibration 10 ms *A*, *B*; and 1.0 ms *A'*-*B'* (modified from Zarzecki and Wiggin 1982).

new variation of the antidromic latency method. Furthermore, separating the spikes of several individual neurons from multiunit recordings allowed us to study the intracortical connectivity of multiple neurons simultaneously. Spike separation, latency measurements and compiling of cross-correlation histograms were performed with a software package (Personal Scientific Workstation, BrainWave Systems Corporation). The procedure for separating and identifying spikes of individual neurons needs to be described in some detail but this will be deferred to the Results section so that it can be illustrated with our own data.

RESULTS

Extrinsic connectivity to motor cortex output neurons

The three sources of extrinsic inputs were not equally effective on motor cortex output neurons. Ipsilateral corticocortical and thalamocortical excitation were found for the majority of neurons; the influenced proportions ranged from 55 to 100% according to the target of the output neurons. Effects from the opposite hemisphere were found for only 5 to 30% of the neurons in the same projection classes. Many neurons (36/81, 44%) were excited from more than one source, but few (5/37, 14%) were influenced by all three possible sources of input, even in small regions of cortex innervated by all three of the inputs. Among electrode tracks where all three inputs were present, there were only two tracks where all the neurons shared the same combination of inputs.

Because the recording tracks were not strictly perpendicular to the cortical surface, neurons separated by as much as 1.6 mm along electrode trajectories could have been located in different "patches" of afferent fibers. Therefore, it was important to study groups of neurons separated by smaller distances. We analyzed groups of neurons recorded simultaneously at one electrode position or that could be recorded (at maximum spike amplitudes) by moving the electrode no more than 200 μm along the track. Each neuron was identified individually by its antidromic invasion. Input patterns were determined for 68 neurons studied in 25 groups. It was unusual for all the neurons in groups to have the same input pattern. In 18 of the 25 groups these neurons in close proximity had different combinations of effective inputs. It is noteworthy that for five of

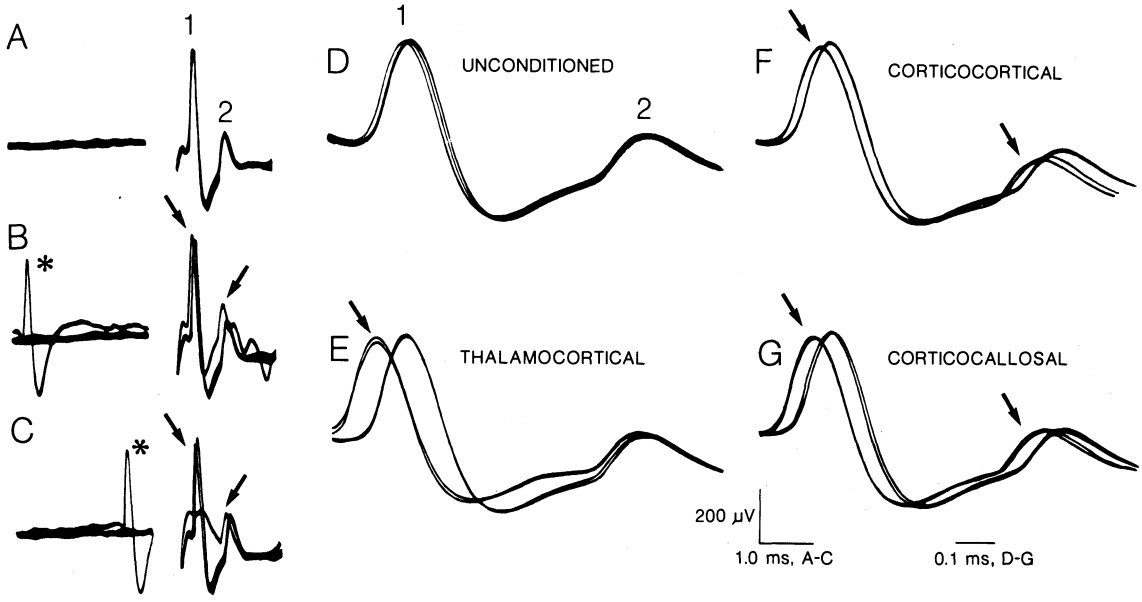

Fig. 2. Neurons located close together and projecting axons to the same target were most likely to have the same pattern of input. *A*, Two corticospinal neurons (1 and 2) were recorded simultaneously. *B,C*, When conditioning stimulation activated thalamocortical pathways, invasion of both neurons was facilitated (arrows). Thalamic stimulation at 200 μA evoked discharge of neuron 1 (asterisks) which, when it occurred within the "critical interval" (*C*), was followed by collision-extinction of the antidromic response. *D*, wave forms of the antidromic spikes of the two neurons without conditioning stimulation. *E*, Conditioning stimulation of thalamus at a lower intensity (120 μA) than that used in *B-C* evoked no discharge, instead subthreshold input altered invasion of neuron 1 (arrow). This intensity of thalamic stimulation is near the threshold for an effect on neuron 2. Corticocortical excitation *F* and callosal excitation *G* also reached both neurons (from Zarzecki 1991).

the seven groups where all the neurons shared the same input pattern, the neurons in the group projected to the same target (for example, the two corticospinal tract neurons of Fig. 2).

The strongest evidence for selective distribution of influences of input pathways came from study of neurons in closest anatomic proximity. Several factors were assessed for how well they predicted similarities in input patterns of neurons in groups, including the number of neurons in the groups, the diversity of the axonal projections and the anatomic proximity of neurons. There was no association between the numbers of input patterns and the numbers of neurons in tracks (Fig. 3*A*, Spearman Rank Correlation, $P>0.05$), the numbers of targets of the tracks (Fig. 3*B*, $P>0.05$), or the number of neurons in the clusters (Fig. 3*C*, $P>0.05$). Projections of corticocortical, callosal and thalamocortical pathways seem to be contacting some neurons while bypassing others, even for neurons in close anatomic proximity in electrode tracks where all three inputs were found. Only the number of targets of clusters was associated with the number of their input patterns (Fig. 3*D*, Spearman Rank Correlation, $P=0.3$), which could result if there was a unique pattern of input for neurons projecting to each target. Therefore, even though they did not inevitably share the same inputs, neurons in closest anatomic proximity and projecting axons to the same targets were most likely to have the same pattern of inputs. These variable combinations of inputs to motor cortex neurons suggest that efferent neurons could be recruited selectively from separate cortical layers or from within groups of nearby neurons according to the target of their axonal projection.

Intracortical connectivity of motor cortex output neurons

Intracortical connectivity was evaluated with two techniques. These were standard cross-correlation analysis and a modification of the antidromic

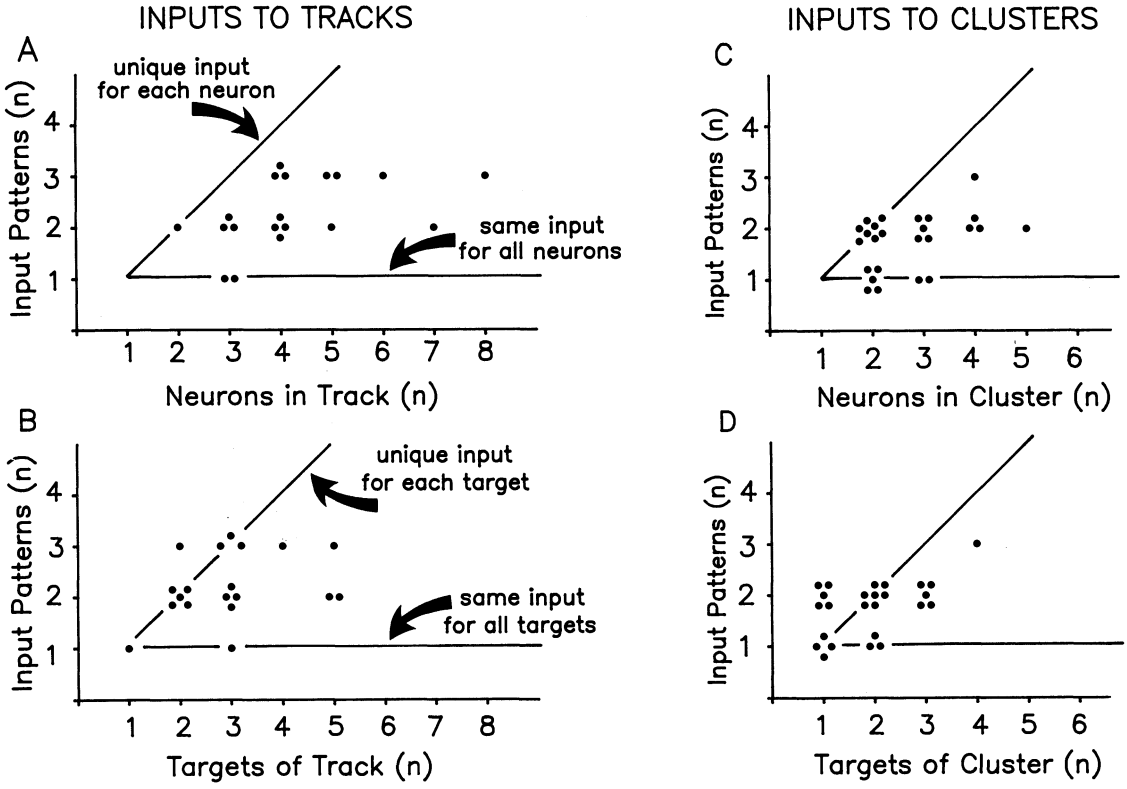

Fig. 3. Inputs to neurons in tracks and in clusters. An input pattern was defined for each neuron by its combination of inputs from corticocortical, callosal, and thalamocortical pathways. *A*, number of input patterns vs. number of neurons studied in 19 tracks. Each data point (•) represents one track. Most data points fell between the two extremes of "same input for all neurons" in the track (horizontal line) and "unique input for each neuron" in the track (diagonal line). *B*, When the number of input patterns was plotted vs. number of targets of each track, more data points fell on the diagonal line (i.e., number of input patterns = the number of targets), as if there could be a unique pattern of input for each target reached by neurons of these tracks. *C*, relationship between number of input patterns and number of neurons studied in each of 25 clusters. Each data point represents one cluster. *D*, Clusters projecting axons to more than one target usually had more than one input pattern (from Zarzecki 1991).

latency method (Fig. 4). For both of these approaches it was necessary to be able to identify, and separate, the spikes of several different neurons recorded simultaneously at each electrode position.

In order to identify the extracellular action potential wave forms originating from individual neurons among several neurons monitored simultaneously, eight parameters of the action potential wave forms were measured (Fig. 5*A*). The measured parameters were peak voltage of the wave form and time of the peak voltage, the voltages and times of the "valleys" before and after the peak, the spike height (difference between peak voltage and second valley voltage), and the spike width (time between peak and second valley). The values of each of these parameters from the action potentials recorded at a given electrode position formed clusters of data points when parameters were plotted against one another (the data points in plots of Fig. 5*B* and *C*). The clustering of the spike parameters quantifies the differences in the spike wave forms of the several neurons recorded simultaneously. Using all eight parameters of spike shape allowed for examination of wave form differences in a total of 28 parameter plots. The following procedure, called "cluster cutting", was used for determining whether or not a cluster of dots was a unique wave form that could have been generated by a single neuron. A "rubber band box" was placed by the operator around a cluster of measures in a parameter plot (for example, cluster r1 of Fig. 5*C*). The program assigned an identification number to all the spikes from which

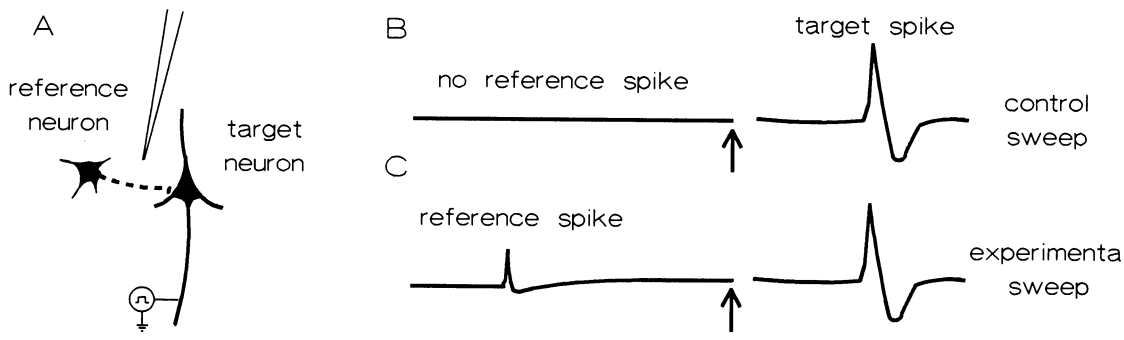

Fig. 4. *A*, Intracortical connectivity was investigated by recording simultaneously from neurons invaded antidromically and from nearby neurons. The invaded neuron is considered the "target neuron" and was tested for effects from simultaneously recorded "reference neurons". *B*, Measurements of antidromic latency were obtained from *control sweeps* where there were no reference spikes. *C*, Control latencies were compared to antidromic latencies in *experimental sweeps* where reference spikes were detected. Arrows in *B* and *C* indicate time of axonal stimulation to evoke antidromic invasion.

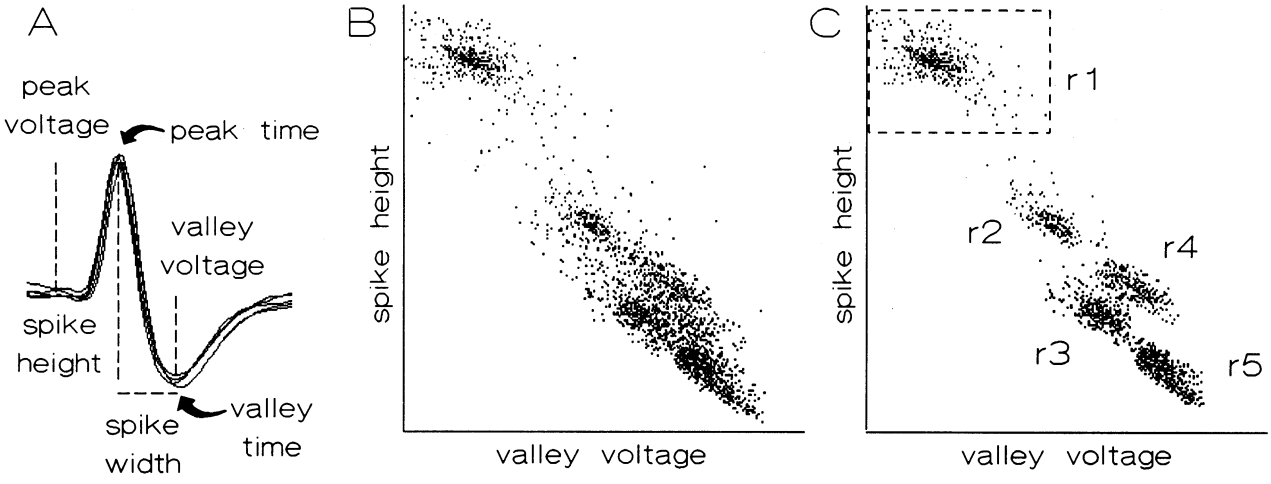

Fig. 5. Spikes of individual neurons were "sorted" from multiunit recordings. *A*, Several parameters of spike wave forms were measured. The duration of the sweep is 1 ms. *B*, The scatter plot of spike height versus valley voltage for spikes recorded simultaneously at one electrode position. Each dot represents the spike height and valley voltage from a single action potential. There are several concentrations of data points ("clusters"). All of the parameters defined in *A* were plotted against one another in pair-wise comparisons ("projections") to identify spikes of several neurons. Each cluster of dots was isolated from the others in at least one projection, similar to the cluster of data points in the upper left hand corner of the plot. *C*, The same data replotted but limited to the points that could be attributed to the spikes identified by the cluster cutting procedure. The dashed box is one of the "rubber band boxes" used in the cluster separation process. The clusters of data points labelled r1 through r5 were measured on the five action potential wave forms illustrated in Fig. 6.

the measured parameters in this rubber band box were obtained. Measures of spike shape in the 27 other plots were colour-coded according to their particular identification numbers and a box was automatically placed around all the measures belonging to the same identification numbers on each plot. There were a total of eight parameters of spike wave form measured, thus the rectangular two dimensional box seen when displaying the plot of any two parameters was a two dimensional view of a 28 dimensional matrix. The "box" enclosing the measures in each plot was reduced in size and number of included spikes by the program automatically eliminating measures that were outside two standard

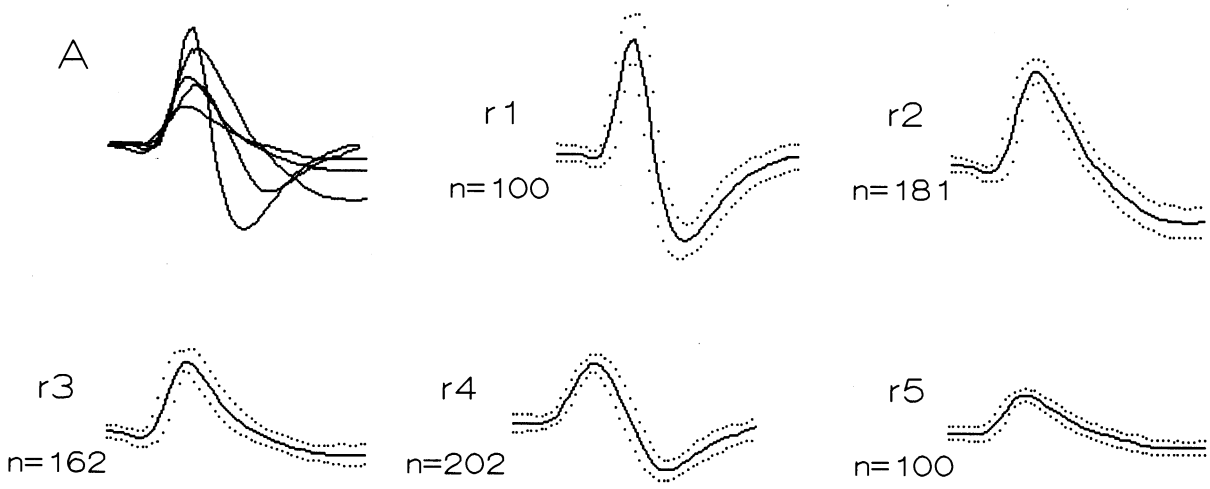

Fig. 6. Results of discrimination of action potential wave forms. Five distinct action potential wave forms were separated from the clusters shown in Figs. 5*B* and *C*. All wave forms were recorded from a single electrode position. The duration of each record is 1 ms. *A*, Superimposition of the five separate wave forms, r1 through r5. Averages of separate wave forms (solid lines) and their standard deviations (dotted lines). Spike r1 is also illustrated in Fig. 5*A*. *n* = number of occurrences of each spike contributing to the averages.

deviations of the mean of the values in the box for each of the 28 plots.

The result of cluster cutting was verified by viewing the action potential wave forms that the program had identified, called the "cut" clusters, and those which were not identified, "uncut". Several outcomes were observed. One outcome was that a labelled group of action potentials was convincingly of the same wave form and it was decided that no further modifications of the inclusion criteria were needed. In other cases, a labelled group of action potentials seemed to contain primarily a single wave form, but also a variable number of other wave forms which were not the same, i.e., "illegal" wave forms. Further tightening of the "box" was performed in an interactive fashion to eliminate these "illegal" wave forms until the operator was finally satisfied that the spikes with different identification numbers were from different neurons. Nevertheless, there was at least one group of labelled action potentials at each recording site that may have been a variety of wave forms that could not be separated and these action potentials were excluded from further analysis. These were usually spikes with amplitudes close to the noise level.

The outcome of one "cluster cutting" operation is shown by the wave forms discriminated from one recording electrode position in Figure 6. In this case, the wave forms fell into five separate groups (the "clusters" of data points in Fig. 5), each attributed to a separate neuron (r1 through r5). The goal of the spike separation procedure was not to include all possible action potentials but rather to be confident that those spikes grouped together were of a distinct wave form and could have been recorded from a single neuron. The average number of wave forms that could be discriminated per electrode position was 3 (range = 2 to 6).

A variation of the antidromic latency method was used to investigate intracortical connectivity of motor cortex output neurons. The neuron invaded antidromically was considered the putative "target neuron". For each invaded neuron, sweeps with antidromic responses were sorted according to whether or not spontaneous spike discharges occurred in a detection period preceding the stimulus. These spontaneous spikes were designated "reference spikes". Each reference spike triggered an extraction from the sweep starting at the reference action potential and continuing up to and including

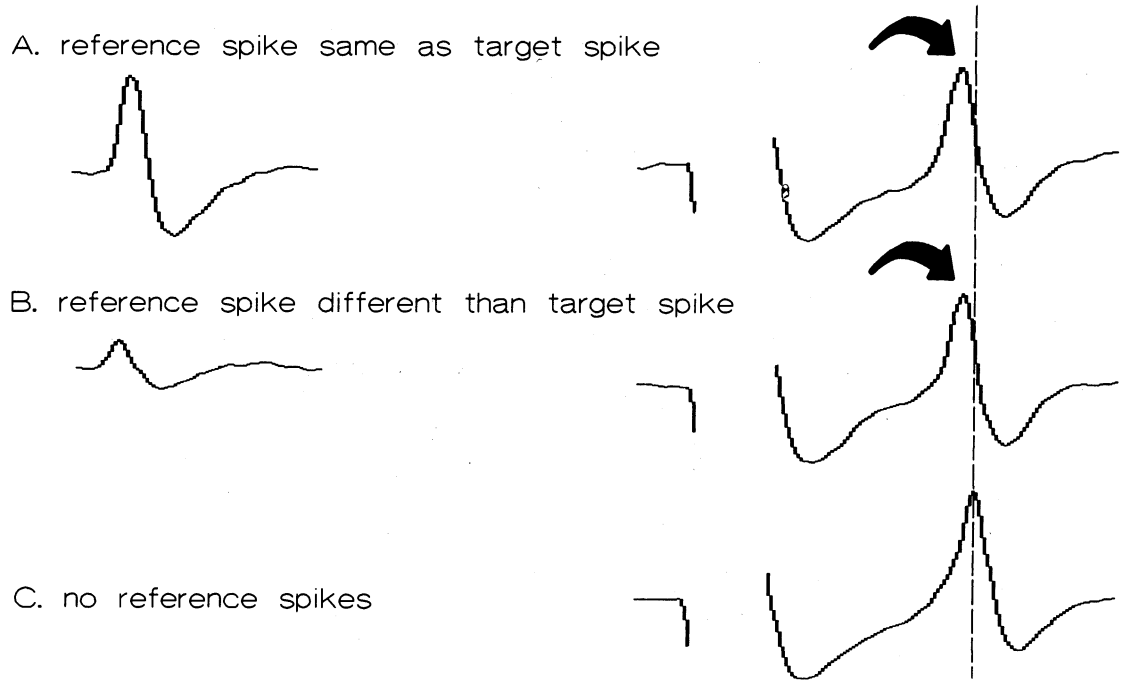

Fig. 7. The antidromic latency was shorter than control values in two circumstances. Data from one corticospinal tract neuron. Sweeps with antidromic responses of the "target neuron" were sorted according to whether or not discharges of "reference neurons" were detected in the 10 ms time period before the antidromic response. *A*, The antidromic response in a sweep where the reference spike was from the neuron invaded antidromically. Note similarity of wave forms of reference and antidromic spikes. The antidromic latency was shorter than for antidromic responses in control sweeps (vertical dashed line). *B*, In other sweeps the detected reference spike was from a different neuron than the one invaded. Note difference in wave forms of reference and antidromic responses. The antidromic latency was shorter than in control sweeps. *C*, Control antidromic latencies were measured in sweeps where no reference spikes were detected before the antidromic response. Responses in *A*, *B* and *C* are single sweeps selected for illustration because the antidromic latencies were equal to the mean latency of their respective groups. Conclusions on differences were based on statistical comparisons among the groups (Wilcoxon Signed-Ranked Test).

the antidromic response of the target neuron. The latency of antidromic invasion was measured in these extracted segments as the time from stimulus delivery to the peak of the negative component of the spike wave form (stimulus-to-peak time). Latency measures were sorted according to spike identification of the reference neurons (from the "cluster cutting" procedure described above). Comparisons were made between latencies of antidromic responses with and without preceding reference spikes (Wilcoxon Signed-Ranked Test).

A reduction in antidromic latencies occurred in two circumstances. Latency reduction occurred when firing of the target neuron occurred in the detection period, i.e., spikes of the neuron invaded antidromically were among the identified reference spikes (Fig. 7*A*). Latency changes also occurred in cases where reference action potentials were not attributed to the invaded neuron but were rather discharges of another neuron recorded simultaneously (Fig. 7*B*).

The two situations where latency changes occurred were classified separately because they were thought to have different causes. In cases where the latency change was associated with the invaded neuron generating the reference spikes (Fig. 7*A*), an increase in axonal conduction velocity associated with the "supranormal period" of the axon would be expected to reduce the latency. This is most readily explained as an axonal effect (Merrill 1972, Swadlow et al. 1978). Latency shifts likely due to axonal effects were classified separately from the latency

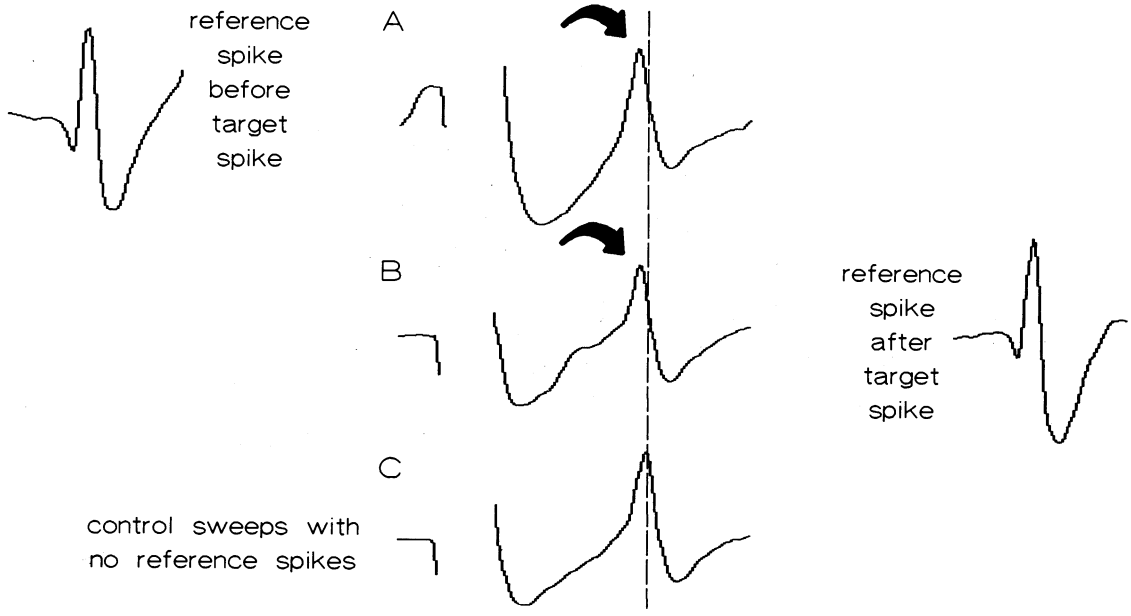

Fig. 8. The shortening of antidromic latency cannot be attributed exclusively to serial synaptic connections from reference neurons to target neurons. Even though the latency was shorter when reference spikes occurred before the antidromic response (*A*) a similar shortening was present also when reference spikes were detected after the antidromic response (*B*). In both cases, the antidromic latency was shorter than in control sweeps (*C*) where no reference spikes were detected.

shifts that occurred after reference action potentials that were not of the target neuron. The reductions in latency associated with the discharges of reference neurons that were not the invaded neuron (Fig. 7*B*) are attributed to a change in the speed of invasion of the action potential from the axon to the soma-dendritic region (Lipski 1981, Zarzecki and Wiggin 1982). This facilitation of invasion of the soma-dendritic region is caused by depolarization of the invaded neuron. The possible sources of the depolarization are considered below.

For the cases where the antidromic latency decreased after reference spikes that were not the spike of the invaded neuron (e.g., Fig. 7*B*), the neuronal depolarization responsible for the latency decrease could be attributed to at least two sources. The reference neuron might be serially connected to the target neuron and produce a EPSP large enough to cause a latency decrease. Alternatively, excitatory input common to both reference and target neurons from a third source could depolarize both these neurons simultaneously (so called "common drive"). If so, spikes of reference neurons would be more likely to occur during times when the invaded neuron was depolarized. Thus, the latency change does not necessarily imply a serial connection from the reference neuron to the target neuron unless common drive can be excluded. Therefore, a series of controls was carried out on a subset of the sample to determine if common drive could be responsible for the observed latency decreases. For these controls we selected 14 neuronal pairs where reference action potentials were detected in the period from 2 ms to 10 ms after the evoked antidromic firing of the target neuron. The length of the detection period after the antidromic action potential was the same as the length of the detection period before the antidromic action potential. If a latency change was due solely to a serial connection from the reference neuron to the target neuron then the latency should return to the control value when the reference neuron fired after invasion of the target neuron because the target neuron could not have been depolarized by the reference neuron during the antidromic response. If, on the other hand, the change in the latency remained, then the depolar-

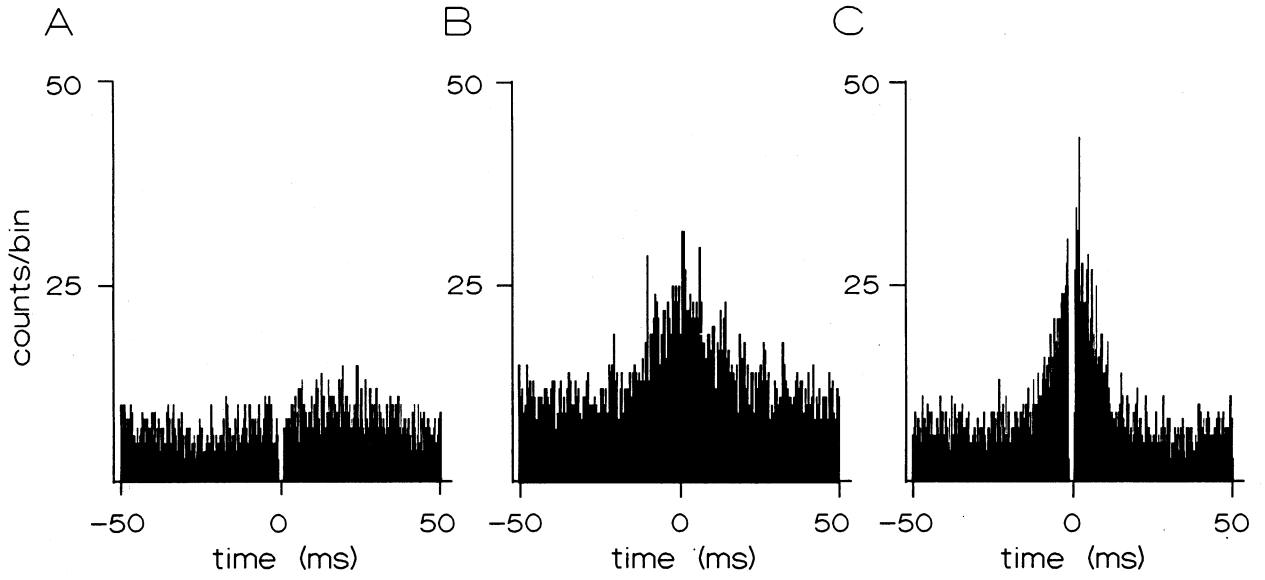

Fig. 9. Cross-correlation histograms compiled between reference neurons and target neurons had several shapes. *A*, Some correlograms were flat. *B*, *C*, A broad, centred peak was the dominant feature of correlograms that did display a change in probability of discharge around the time of the reference spike. *C*, A peak offset from zero time suggests shared input for the two neurons combined with serial synaptic coupling from one neuron to the other. The incidence of each shape of correlogram is given by the percentages in the panels.

ization of the target neuron must have resulted from the action of other inputs coupled loosely in time to discharge of the reference neuron. For 13 of 14 pairs of reference and target neurons the latency decrease found in sweeps with discharges of reference spikes before the antidromic response persisted even when the reference spike discharged after the antidromic invasion e.g., Fig. 8. Therefore, in most cases the latency decrease does not require a serial synaptic connection from the reference neuron to the target neuron.

Intracortical functional connectivity was also evaluated by cross-correlation analyses. Forty-two reference neurons were analyzed by cross-correlation to test for their functional interrelationships with the eleven antidromically identified target neurons that generated sufficient numbers of spontaneous discharges for cross-correlation histograms to be compiled. From one to four reference neurons were examined per target neuron. Thus, forty-seven cross-correlation histograms were calculated. The average number of reference spikes was 5907 and of target spikes was 7602. Cross-correlation histograms contained several prominent features that allowed them to be divided into groups.

In some correlograms there was no obvious change in the probability of discharge of the target neuron around the time of discharge of the reference neuron, i.e., the histogram was flat (Fig. 9*A*). A broad, centred peak, usually interpreted as synaptic input shared by the reference and target neurons (Moore et al. 1970, Gochin et al. 1991), was the most common feature of correlograms that did show a change in probability of discharge around the time of the reference spike (Fig. 9*B*). The width of central peaks ranged from 30 to 60 ms. In some cases, the peak was offset from zero time, suggesting shared input for the two neurons combined with serial synaptic coupling from one neuron to the other (Fig. 9*C*).

The dominant characteristic of functional intracortical connectivity that was revealed by the antidromic latency method and by the cross-correlation analysis was common excitatory input shared by nearby neurons. Neurons in all of the classes of output neuron identified by antidromic invasion shared this excitatory input with their near neighbours that were not invaded antidromically. The result of such ubiquitous common input would be that neurons would be recruited synchronously. In the Discus-

sion we speculate how mechanisms for synchrony could operate together with mechanisms for selective recruitment.

DISCUSSION

Previous anatomical and electrophysiological studies using a variety of techniques and described in detail in the Introduction have lead to the conclusion that afferent systems are selective in the distribution of their actions upon cortical neurons. There is anatomical and physiological evidence for this conclusion. Afferent inputs to cortex, in particular corticocortical and thalamocortical fibers, synapse preferentially upon certain classes of efferent neurons (Strick and Sterling 1974, Deschenes et al. 1982, Ichikawa et al. 1985, Liu et al. 1986, Porter and White 1986). When afferent systems are activated with electrical stimulation, neurons within small groups are recruited selectively (Zarzecki 1989, 1991). Thus, afferent systems may recruit selectively particular efferent neurons that project to different targets. Consequently, these neurons would have different time-varying patterns of activity during behavior, i.e., they would not discharge in temporal synchrony, so that even a small region of motor cortex would send different signals to its various targets.

In some ways the results from our study of intracortical connectivity seem to be in conflict with the above conclusions of selective actions upon individual neurons located within circumscribed groups in cortex. For example, both the cross-correlation analysis and the antidromic latency method indicated that a high proportion of reference and target neurons have common input that would cause the neurons to discharge in synchrony. Synchronous activation may have several functions. For example, groups of simultaneously active neurons could be considered "functional assemblies" of neurons because they share common sensory properties, i.e., they are the building blocks of cortical representations of the external sensory world (Engel et al. 1992). Furthermore, the simultaneous recruitment of many neurons projecting to the same distant target will have a greater effect than individually discharging single neurons (Engel et al. 1992).

Up to this point we have discussed two mechanisms of cortical activation, i.e., "synchrony" and "selectivity", as if they were two distinct operations each with their own roles and without relation to one another. Nevertheless, it is possible they are actually the two extremes of what really is a continuum. Imagine for the purposes of speculation that the intracortical circuitry of the motor cortex has capacities for the fast "modulations of effective connectivity" that have been modelled by Aersten et al. (1989) and detected in visual cortex (Engel et al. 1992). In those theoretical and actual circuits the association of neurons into "functional assemblies" are not fixed rigidly by preferential anatomical wiring. Instead, individual neurons become temporally associated or disassociated with their neighbours by fast modifications of synaptic efficacy in intracortical circuits. Consequently, a given neuron that might at one time be recruited selectively by action of afferent pathways to cortex could at another time be bound into synchrony with nearby neurons by an enhancement of the potency of common drive. It is hypothesized that the appearance and disappearance of such synchrony is stimulus feature-dependent or motor action-specific (Engel et al. 1992). This hypothesis could be tested by simultaneous recording from spatially separated groups of neurons and monitoring their functional connectivity with cross-correlation and antidromic latency methods while features of sensory stimuli are changed or animals are engaged in motor behaviours.

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