Organization of Nonprimary Motor Cortical Inputs on Pyramidal and Nonpyramidal Tract Neurons of Primary Motor Cortex: An Electrophysiological Study in the Macaque Monkey

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To elucidate the functions of nonprimary motor cortical (nPMC) areas whose afferents synapse onto output neurons of the primary motor cortex (PMC), we examined the responses of pyramidal tract neurons (PTNs) and non-PTNs (nPTNs) to electrical stimulation in the three nPMCs, the supplementary motor area (SMA) and the dorsal and ventral divisions of the premotor cortex (PMd and PMv), with extracellular unit recording in alert monkeys. Typical responses of PTNs to nPMC stimulation were early orthodromic excitatory responses followed by inhibitory responses. Among 27 PTNs tested by constructing peri-stimulus time histograms, 19 (70.4%) showed inhibitory responses to stimulation in all of the nPMC areas. In contrast, 5/33 PTNs (15.2%) and 10/72 nPTNs (13.9%) showed excitatory responses to stimulation in all of the nPMCs. The inhibitory responses of PTNs were mediated by inhibitory interneurons, some of which may correspond to nPTNs in the superficial layers of the PMC. These interneurons probably possess widely extended axons and nonspecifically inhibit multiple PTNs in layer V. The excitatory and inhibitory influences, and the patterns of convergence of inputs from the nPMCs onto the PTNs, are important to understand motor control by the nPMC–PMC–spinal cord pathway.

Introduction

It is generally accepted that multiple motor areas of the primate frontal cortex participate in controlling forelimb movements (Wise et al., 1991; Tanji, 1994; Tanji et al., 1996). According to morphological and functional criteria, these areas can be divided into two categories: the primary motor cortex (PMC) and other motor areas [i.e. nonprimary motor cortices (nPMCs) or premotor areas] (Dum and Strick, 1991; Wise et al. 1991). The PMC was defined as Brodmann’s area 4, located in the precentral gyrus, where orofacial, forelimb and hindlimb body parts are represented from lateral to medial, in this order. Most of the nPMCs are situated in the medial and lateral parts of Brodmann’s area 6, which include the supplementary motor area (SMA), and the dorsal and ventral divisions of the premotor cortex (PMd and PMv). A forelimb representation exists in each of these areas.

Intracortical microstimulation (ICMS) (Asanuma and Sakata, 1967) in the PMC evokes simple or single-joint movements with low threshold (Asanuma and Sakata, 1967; Kwan et al. 1978; Sesse and Wiesendanger, 1982; Sato and Tanji, 1989). Neurons in the PMC show activities related directly to parameters of movements or muscles, such as static force, muscle length, direction, velocity and acceleration (Hepp-Reymond, 1988). In the nPMCs, higher-current or longer-train stimulation has usually been applied to evoke movements with ICMS (Weinrich and Wise, 1982; Mitz and Wise, 1987; Luppino et al., 1991; Godschalk et al., 1995; Nambu et al., 1997; Takada et al., 1998). The nPMCs have been implicated in higher motor control, such as preparation, sequencing and bilateral control of movements (Tanji et al., 1987, 1988, 1996; Wiesendanger, 1993; Tanji, 1994; Tanji and Shima, 1994). Since the forelimb regions of the SMA, PMd and PMv project directly to the forelimb region of the PMC (Muakkassa and Strick, 1979; Leichnetz, 1986; Dum and Strick, 1991), it is conceivable that information of nPMC neurons is received and processed by PMC neurons, and is finally conveyed to the spinal cord by the pyramidal tract neurons (PTNs) in the PMC. Although the nPMCs also send projection fibers to the spinal cord directly (He et al., 1993, 1995), one can postulate that the nPMC–PMC–spinal cord pathway plays particular roles in transforming information from the nPMCs to the spinal cord. In this context, it is interesting to investigate the flow of information through the nPMC–PMC–spinal cord pathway by analyzing the influence of nPMC–PMC inputs on output neurons of the PMC.

In the present study we examined the influence of projections from the nPMCs onto PMC neurons using extracellular recording in awake monkeys. PTNs and non-PTNs (nPTNs) in the forelimb region of the PMC were differentiated using antidromic activation evoked by electrical stimulation in the medullary pyramid (Py). We then examined the response patterns of PTNs and nPTNs to electrical stimulation in the forelimb regions of the SMA, PMd and PMv, which were identified with ICMS mapping.

Materials and Methods

Preparation of Animals

Experiments were performed on two female Japanese monkeys (Macaca fuscata) weighing 5.3 and 6.2 kg. The use of the animals in the present study followed guidelines approved by the animal experiment committee at the Tokyo Metropolitan Institute for Neuroscience. Each monkey was anesthetized with ketamine hydrochloride (10 mg/kg body wt, i.m.) and sodium pentobarbital (30 mg/kg body wt, i.v.), and surgically prepared for the recording experiments. Under anesthetic conditions, the skull was widely exposed, and small screws were attached to the skull as anchors. The exposed skull and screws were covered completely with transparent dental acrylic. Two pipes were mounted in parallel over the frontal and occipital lobes for head fixation.

A few days later, the monkeys were tranquilized with ketamine hydrochloride (10 mg/kg body wt, i.m.) and xylazine hydrochloride (1–2 mg/kg body wt, i.m.) and sat quietly in the monkey chair with their heads restrained. After making a small hole at the midline of the skull for SMA mapping and a larger hole over the lateral frontal lobe for access to the PMd, PMv and PMC, extracellular recording and ICMS were performed. A glass-insulated Elgiloy-alloy microelectrode (impedance 0.7–1.5 MΩ at 1000 Hz, exposed tips 7–15 µm) was inserted perpendicular to the cortical surface into the SMA, PMd and PMv. After extracellular unit recording and examination of neuronal responses to passive somatic movements, currents of <50 µA were delivered with a train of 22 cathodal pulses (200 µs duration at 333 Hz) for ICMS, and evoked movements were observed. Pairs of bipolar stimulating electrodes made of enamel-coated stainless steel wire (200 µm in diameter; intertip distance, 2 mm) were implanted into forelimb areas of the SMA, PMd and PMv. After extracellular unit recording and examination of neuronal responses to passive somatic movements, currents of <50 µA were delivered with a train of 22 cathodal pulses (200 µs duration at 333 Hz) for ICMS, and evoked movements were observed. Pairs of bipolar stimulating electrodes made of enamel-coated stainless steel wire (200 µm in diameter; intertip distance, 2 mm) were implanted into forelimb areas of the SMA, PMd and PMv at a depth of 4, 3 and 4 mm respectively from the dural surface. Electrodes were then fixed with dental acrylic. A square chamber was fixed onto the hole on the PMC for the following experimental session.

A pair of stimulating electrodes was implanted into the ipsilateral Py. A pair of Elgiloy-alloy microelectrodes coated with cashew resin (exposed...
tips, 0.3–0.5 mm; intertip distance, 2 mm) was inserted vertically through the parietal cortex at $A = 3$, $L = 2$. Currents of 10–30 $\mu$A were delivered through one of the electrodes with a train of 22 cathodal pulses (200 $\mu$s duration at 333 Hz) while the electrodes were moved. When jerky contralateral body movements, especially forelimb movements, were elicited at the lowest threshold, the electrodes were fixed with dental acrylic.

**Data Acquisition and Analysis**

After implanting the stimulating electrodes, the same type of Elgiloy-alloy microelectrode as used in ICMs mapping of the nPMCs was inserted perpendicular to the cortical surface into the PMC to record neural activities. We used one negative monophasic square pulse for stimulation (duration, 300 $\mu$s), with stimulus strength ranging from 100 to 500 $\mu$A for stimulation in the nPMC sites, and from 100 to 200 $\mu$A for stimulation in...
the Py through the implanted electrodes. The effect of changing the stimulus strength on the patterns of the neuronal responses was not examined systematically. Stimulus strength used in the following analysis was actually determined as that evoking neuronal responses more than four times in five trials of stimulation at the minimal current intensity. Responses to stimulation in the Py were judged to be antidromic if they appeared at a fixed latency and collided with spontaneous spikes. Stimulus-evoked responses were judged to be orthodromic when spikes occurred at variable latencies during the delivery of 5–10 stimuli at 0.5–0.8 Hz. Neuronal responses to the stimulation were continuously sampled from 5 ms before to 20–100 ms after onset of stimulation at a rate of 10 kHz and stored by a computer program. The response latency was defined as the mean interval between the onset of stimulation and the appearance of spikes calculated over five trials.

Responses of PTNs to stimulation in the three nPMCs were further analyzed by constructing peri-stimulus time histograms (PSTHs, sum-

Figure 2. A pyramidal tract neuron (PTN) showing both early excitatory and late inhibitory responses to stimulation in all of the nPMC areas: the SMA, PMd and PMv (b–d). The antidromic responses (asterisk) to stimulation in the Py collided with spontaneously occurring spikes (double asterisk) (a, left, 3rd trace). (a–d) Left: five traces were superimposed. Right: peri-stimulus time histograms (PSTHs). Summation of 100 trials. Bin width, 0.5 ms. Open arrows, excitatory responses; Black arrows, onset of stimulation.
of 100 trials, bin width, 0.5 ms) using a window discriminator and a computer. The onset and duration of inhibitory responses of units after stimulation were judged by comparison with the frequency of spikes at 100 ms before stimulation (Mann–Whitney U-test, P < 0.05).

The effect of ICMS with a train of 12 cathodal pulses (200 µs duration at 333 Hz) was observed to determine the location of the electrode in the PMC. However, ICMS was carefully performed at only one or two sites with one microelectrode penetration to avoid tissue damage and the alteration of the responses of PMC neurons. When ICMS evoked movements other than forelimb, we stopped the penetration of the microelectrodes and the sampling of neural activities.

**Histology**

At the end of the electrophysiological experiments, electrolytic microlesions (anodal currents of 20 µA, 30 s) were placed at several sites in the PMC. The monkeys were anesthetized deeply with an overdose of sodium pentobarbital (60 mg/kg body wt, i.v.) and perfused transcardially with 2 l of phosphate-buffered saline (pH 7.3), followed by 5 l of 8% formalin in 0.1 M phosphate buffer (pH 7.3). The monkeys were then perfused with 3 l of 0.1 M phosphate buffer (pH 7.3) containing 10% sucrose and, finally, with 2 l of the same buffer containing 30% sucrose. The brains were removed immediately and saturated in the same buffer containing 30% sucrose at 4°C. They were then cut into frontal sections (60 µm)

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**Figure 3.** A PTN showing the early excitatory responses (b, left) followed by late inhibitory responses (b, right) to stimulation in the SMA, and the inhibitory responses to stimulation in the PMd (c) and PMv (d). The antidromic responses (asterisk) to stimulation in the Py collided with spontaneously occurring spikes (double asterisk) (a, left, 3rd traces). (a, b) Left: five traces were superimposed. Right: PSTHs. Summation of 100 trials. Bin width, 0.5 ms. Arrows, see legend to Figure 2.
thick) on a freezing microtome. All sections were serially mounted onto gelatin-coated glass slides, counterstained with 1% Neutral Red, and observed with a light microscope and a profile projector under a bright-field illumination.

**Results**

**Locations of Stimulating Electrodes**

The forelimb areas of the SMA, PMd and PMv were identified using ICMS before implanting the stimulating electrodes. Results from ICMS mapping of these areas were in good agreement with the previously reported pattern of body part representation in nPMCs (Mitz and Wise, 1987; Luppino et al., 1991; Godschalk et al., 1995; Nambu et al., 1997; Takada et al., 1998). Forelimb movements were elicited in the medial wall of the hemisphere, the lateral bank of the superior precentral sulcus and the posterior bank of the genu of the arcuate sulcus, which correspond to the forelimb region of the SMA, PMd and PMv respectively (Fig. 1). The central region of the forelimb representation of each nPMC were selected for implanting pairs of stimulating electrodes. During experimental sessions, cortical stimulation up to 500 µA through the electrodes evoked only forelimb movements. Current spread to neighboring areas representing other body parts was negligible. The locations of stimulating electrodes were confirmed by histological examination of serial sections cut through the nPMC areas (Fig. 1c). The stimulating electrodes used in the present study were thick enough (200 µm in diameter) for histological identification of their tracks (Fig. 1c, arrows). The tips of all stimulating electrodes were located in the gray matter of the nPMCs. Although the tip of the anterior electrode for bipolar stimulation in the PMd was located near the border between the gray and white matter (Fig. 1c, section 81), stimulation through this electrode might cause minimum current spread to the underlying white matter. The tips of electrodes for stimulation of PTN axons were located in the Py (Fig. 1d).

**Responses of PTNs**

Responses from a total of 105 neurons in the PMC to electrical stimulation in the SMA, PMd, PMv and Py were analyzed. Typical examples of neuronal responses to electrical stimulation in the nPMCs and Py are shown in Figures 2–5, and patterns of convergence from nPMC inputs are shown in Figure 7. Of 105 neurons tested, 33 (31.4%) were identified as PTNs, which showed antidromic responses with a fixed latency and were considered to be PTNs. None of these slow PTNs showed orthodromic excitatory responses to nPMC stimulation, and were considered to be slow PTNs. No responses without subsequent inhibitory responses were observed in any of the PTNs examined. In addition, we did not observe antidromic responses to nPMC stimulation in any PTN.

As shown in Figure 8, three PTNs showing antidromic responses to stimulation in the Py had latencies of 3.3–4.9 ms, and were considered to be slow PTNs. None of these slow PTNs showed orthodromic excitatory responses to nPMC stimulation,
while 23 of the other 30 PTNs (i.e. fast PTNs) showed excitatory responses to stimulation in at least one of the nPMCs. The difference in the proportion of neurons receiving excitatory nPMC inputs between slow PTNs and fast PTNs was statistically significant (Fisher's exact probability test, \( P < 0.05 \)).

Stimulation in the Py evoked effects other than antidromic responses. Of 33 PTNs, nine (27.3%) showed excitatory responses to stimulation in the Py. In addition, we constructed PSTHs to stimulation in the Py for 12 PTNs. Among them, four PTNs (33.3%) showed excitatory–inhibitory responses (Figs 2a, 3a), seven PTNs (58.3%) showed only inhibitory responses, and one PTN (8.3%) showed no response (Steffanis and Jasper, 1964; Armstrong 1965; Takahashi et al., 1967). The duration of the inhibition was shorter than that evoked by stimulation in the nPMCs, while the latency of the excitation was longer than that evoked by stimulation in the nPMCs (Fig. 6).

Responses of nPTNs
Seventy-two neurons that did not show antidromic responses to stimulation in the Py were considered to be nPTNs. Of these, 32 neurons (44.4%) showed orthodromic excitatory responses to nPMC stimulation (Fig. 5b–d). The latency of the excitatory responses was slightly shorter than the latency of the excitatory responses of PTNs (Fig. 6). Although PSTHs were not constructed to analyze the responses of nPTNs, the duration of the excitatory responses were 3–5 ms. Of the 32 neurons showing orthodromic responses to stimulation in at least one of the nPMCs, 15 (46.9%) also showed orthodromic responses to the stimulation in the Py (Fig. 5a). We found two nPTNs that were antidromically activated by stimulation in the PMv. However the number of these neurons were small, as observed in the previous study (Ghosh and Porter, 1988); we made no systemic analysis of these neurons. During the present experiments, nPTNs and PTNs were not distinguishable by observing their spontaneous activities.

Patterns of Convergence of nPMC Inputs
Figure 7 shows the convergence patterns of excitatory and inhibitory nPMCs inputs to PTNs and nPTNs. Among 27 PTNs tested by constructing PSTHs (Table 1), 19 (70.4%) showed inhibitory responses to stimulation in the SMA, PMd, and PMv. In contrast, 5/33 PTNs (15.2%) and 10/72 nPTNs (13.9%) showed early excitatory responses to stimulation in all of the nPMCs. The convergence of inhibitory inputs from all three nPMCs was significantly more frequent than that of excitatory inputs (Fisher’s exact probability test, \( P < 0.001 \)). No PTNs and nPTNs showed responses to stimulation in the PMd alone in the two monkeys presented here.

Distribution of the Examined Neurons
In the present experiments, the recording microelectrodes were advanced from the superficial to the deep layers of the PMC. In such penetrations, we usually found nPTNs showing excitatory responses to stimulation in the nPMCs and PTNs in this order, suggesting that nPTNs showing excitatory responses are located in more superficial layers than are the PTNs. This finding was confirmed by histological examination of the frontal sections (Fig. 9). PTNs were located mainly in layer V, whereas nPTNs showing excitatory responses were largely found in layer II/III. PTNs showing excitatory responses to nPMCs stimulation were intermingled with those showing no excitatory responses to nPMCs stimulation. We did not find an obvious difference in the distribution of responding neurons between the surface and bank regions of the PMC or between the distal and proximal representations of the PMC.

Discussion
The present investigation revealed the following findings on the responses of PMC neurons to electrical stimulation in the nPMCs. (i) The majority of the PTNs showed inhibitory responses to stimulation in the nPMCs. Some showed early excitatory responses followed by inhibitory responses. (ii) Frequent convergence of all three nPMC inhibitory inputs to PTNs was observed. This convergence was found much less frequently in excitatory inputs to PTNs and nPTNs. (iii) Slow PTNs that we examined did not show excitatory responses to stimulation in the nPMCs. (iv) PTNs were mainly located in layer...
V of the PMC, while nPTNs were distributed in more superficial layers (layer II/II) of the PMC.

**Excitatory Responses**

Orthodromic excitatory responses to nPMC stimulation were seen in both nPTNs and PTNs. The mean latency of the excitatory responses of PMC neurons to stimulation in the nPMCs (Fig. 6) is comparable to data reported in an extracellular recording study on orthodromic responses to SMA stimulation [3.7 ms (Aizawa and Tanji, 1994)]. The mean latency obtained in the present experiments is also congruent with the data from an intracellular recording study on orthodromically evoked excitatory postsynaptic potentials (EPSPs) of PTNs to PMv stimulation [2.36 ms (Ghosh and Porter, 1988)] and from an extracellular recording study on antidromic responses of PMv neurons to PMC stimulation [1.2 ms (Godschalk et al., 1984)], since with the techniques used in these studies, shorter latency is commonly obtained than in studies on orthodromic responses in extracellular recording.

In the present results, there were no important differences between the latencies of the excitatory responses of nPTNs and PTNs, although those of nPTNs were slightly shorter than those of PTNs. Ghosh and Porter showed that there was no significant difference between the latencies of EPSPs evoked monosynaptically in layer III PMC neurons and those evoked in layer V neurons (Ghosh and Porter, 1988). No clear selectivity or difference in the terminations of nPMC axons onto PTNs and nPTNs may exist.

None of three slow PTNs showed orthodromic excitatory responses to nPMC stimulation. It has already been shown that none of five slow PTNs showed EPSPs in response to stimulation in the PMv, while all of them showed responses to the stimulation in the primary somatosensory cortex (Ghosh and Porter, 1988). The slow PTNs do not seem to participate in motor control by the nPMC–PMC–spinal cord pathway. These neurons are known to show slow tonic and regular discharges without movement (Evarts, 1965) and to be related to slow or tonic arm movements (Tanji et al., 1978).
Inhibitory Responses

The most striking results obtained in the present study were that a considerable proportion (70.4%) of identified PTNs showed inhibitory responses to stimulation in all of the nPMC areas. Inhibitory postsynaptic potentials (IPSPs) have already been described in an intracellular recording study of PMv inputs onto PTNs, and these may be mediated by inhibitory interneurons in the PMC (Ghosh and Porter, 1988). However, the patterns of convergence of inputs from multiple nPMCs onto PMC neurons was not examined in that study.

The proportion of PTNs showing inhibitory responses to the SMA, PMd and PMv was significantly higher than that of nPTNs showing orthodromic responses to stimulation in all of the nPMCs. The highly convergent pattern of inhibitory inputs to PTNs may be ascribable to the divergent intracortical distribution of inhibitory interneuron axons that receive excitatory inputs from the nPMCs. In fact, inhibitory interneurons responsible for evoking fast IPSPs possess horizontally spreading parallel axons that make synaptic contacts along the apical dendrites of pyramidal neurons (Kang et al., 1994). Interneurons that extend their axons horizontally are known to be basket cells, which exhibit parvalbumin and glutamate decarboxylase immunoreactivity (DeFelipe et al., 1986; Hendry et al., 1989). These parvalbumin-immunoreactive neurons were recently shown to be fast-spiking cells in layer II/III that exhibit an abrupt episode of nonadapting repetitive discharges (Kawaguchi, 1995; Kawaguchi and Kubota, 1997).

Long-lasting inhibitory responses of PTNs to nPMC stimulation were observed in the present experiments. If nPTNs evoke only fast IPSPs in PTNs, nPTNs must be active for a sufficient period of time to account for the inhibitory responses observed in PTNs. Nevertheless, the duration (3–5 ms) of excitatory responses of nPTNs was much shorter than the duration (~250 ms) of the long-lasting inhibitory responses of PTNs. Thus the temporal pattern of the inhibitory responses of PTNs is derived from long-lasting IPSPs mediated by axon terminals of nPTNs making synaptic contacts on PTNs. The cortical pyramidal neurons are known to show slow IPSPs lasting hundreds of milliseconds which are evoked by activation of interneurons in vitro (Connors et al., 1988; McCormick, 1989; Kang et al., 1994). Furthermore, monkey PTNs show long-lasting IPSPs after stimulation in the PMv in vivo (Ghosh and Porter, 1988). Thus, the long-lasting inhibitory responses of PTNs observed in the present study could be accounted for by slow IPSPs mediated by nPTNs. However, slow IPSPs have been suggested to be mediated by double bouquet cells with vertically descending axons (Kang et al., 1994). The spatiotemporal patterns of inhibitory responses of PTNs revealed in the present study may be ascribable to mixed inhibitory influences mediated by basket cells and double bouquet cells. Indeed, the duration of the inhibitory responses of PTNs to stimulation in the nPMCs ranged from 4 to 250 ms in the present experiments (Fig. 6).

Another possible explanation for the long-lasting inhibition of PTNs is the loss of excitatory drive from neighboring PTNs sending their axon collaterals widely into layer V. About one-third of the PTNs showed excitatory responses to stimulation in the Py, as shown in earlier studies (Armstrong 1965; Takahashi et al., 1967). The activities of PTNs receiving axon collaterals from neighboring PTNs may be decreased both by activation of the superficial inhibitory interneurons evoked by stimulation in the pyriform cortex.
Figure 9. Spatial distribution of examined neurons in the PMC. PTNs identified in the present study (filled circles) were distributed in layer V, while nPTNs (open circles) were distributed in more superficial layers (layer II/III). Arrows indicate sites where the effect of ICMS was examined. Letters indicate the body parts where evoked movements were observed. Abbreviations of movements elicited by microstimulation as in Figure 1. A series of 60 µm thick frontal sections are arranged rostrocaudally. Each number indicates the section number shown in Figure 1b.
nPMCs and subsequent loss of activities of neighboring PTNs, which are also inhibited by interneurons after nPMC stimulation. Although functional properties of axon collaterals of PTNs have not been analyzed in detail, particularly in monkeys performing motor tasks, the function of these axon collaterals of output neurons of the PMC should be analyzed in future studies.

Excitatory Responses Followed by Inhibitory Responses
We often observed early excitatory responses followed by late inhibitory responses to nPMC stimulation. A previous intracellular recording study (Ghosh and Porter, 1988) showed that EPSPs were followed by IPSPs in all neurons that responded to stimulation in the PMv. A more recent extracellular recording study (Baker et al., 1998) demonstrated that PTNs showed excitatory responses followed by inhibition after ICMS in the PMv, where stimuli were delivered with stimulating electrodes that were positioned at a distance of 1.5–2 mm from the recording electrodes. Some such PTNs were additionally identified as corticomotoneuronal neurons using the spike-triggered averaging method. It is reasonable to consider that the responses of PTNs are evoked after ICMS by the excitation of axons that have excitatory inputs into the recorded PTNs and of inhibitory interneurons that synapse onto the recorded PTNs.

Function of the nPMC–PMC–Spinal Cord Pathway
Here we describe spatiotemporal patterns of excitation and inhibition after nPMC stimulation. The difference in the convergence patterns between excitatory inputs and inhibitory inputs to PTNs from the nPMCs might be required for suppressing unnecessary activities of the PMC neurons and focusing on the flow of information of intended movements mediated by the more specific excitatory link in the nPMC–PMC–spinal cord pathway.

One of the motor control functions of the nPMC is the preparation of movements. Preparatory or set-related activities of nPMC neurons have been reported repeatedly (Tanji et al., 1980; Weinrich and Wise, 1982; Tanji and Kurata, 1985; Kurata and Wise, 1988a,b). However, experiments designed to identify the input or output of these neurons showing preparatory activities is still limited. Tanji and Kurata showed that identified SMA–PMC projection neurons show preparatory activities (Tanji and Kurata, 1985). These neurons also show selective activities to different (tactile or auditory) motor instructions. Furthermore, a recent study (Aizawa and Tanji, 1994) showed that PMC neurons that have preparatory activity preferentially receive inputs from other areas, e.g. the parietal cortex and thalamus, and outputs to subcortical motor areas, e.g. the basal ganglia, thalamus and precrrebellar nuclei, should be examined to fully elucidate the information processed by the PMC.

References


