THE PRIMATE PREMOTOR CORTEX:
Past, Present, and Preparatory

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THE CONCEPT AND HISTORY OF THE PREMOTOR COMPLEX

Campbell and the Invocation of J. Hughlings Jackson

The concept of a premotor cortex was first proposed by Campbell (1905), who called it the “intermediate precentral cortex.” Campbell linked the precentral motor cortex to the middle of J. Hughlings Jackson’s three levels of motor organization and postulated that the rostrally adjacent region, the intermediate precentral cortex, might be the site of Jackson’s highest motor level. Brodmann (1905, 1909) agreed with Campbell’s cytoarchitectonic definitions and emphasized the idea that together the precentral cortex (area 4) and the rostrally adjacent field (area 6) constituted a group of functionally related, presumably motor, areas.

Other investigators also thought that they could identify architectonic subdivisions of the primate frontal cortex (Vogt & Vogt 1919, von Economo 1929, Bucy 1933, 1935, von Bonin & Bailey 1947, von Bonin 1949, Bailey & von Bonin 1951, Jones et al 1978), although their schemes differed markedly from one another. In the years since the beginning of the century, several of these

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subdivisions have been identified as the “premotor cortex,” but that term has been employed with an inconsistency and vagueness that has hindered the study of this cortical region. This review is submitted in an attempt to clarify some of the issues involved in the definition of the premotor cortex and the study of its function. Transcending the narrow questions about the definition of premotor cortex, however, are problems representative of those widely encountered in cortical areas not linked in a straightforward fashion with either a receptor system or effector organ. In this sense, then, study of the premotor cortex is relevant to the more general problem of nonprimary, or “association,” cortical areas.

**Terminology of the Premotor Cortex**

A large part of the frontal lobe of primates is thought to play an important role in the cerebral control of movement. Those regions commonly designated as motor fields lack a clearly defined internal granular layer, layer IV, and thus often are referred to as the *agranular frontal cortex*. When used in this way, the term “agranular frontal cortex” excludes not only the granular prefrontal cortex, but also medial and orbital frontal regions of cortex. A three-part division of the agranular frontal cortex (Figure 1) is possible on the basis of a constellation of electrophysiological, connectional, architectonic, and behavioral findings that are outlined below. Two subdivisions, the *primary* (or *precentral*) motor cortex (MI) and the *supplementary motor cortex* (MII), are located approximately as described by Woolsey and his colleagues (1952).

![Figure 1](image_url)  
*Figure 1*  
Surface view of the left cerebral hemisphere of a macaque monkey. The medial surface of the hemisphere is shown, inverted, at top. The approximate locations of the primary motor cortex (MI), the supplementary motor cortex (MII), and the premotor cortex (PM) are indicated. Each *dotted line* represents the fundus of a sulcus. The *open squares* show a rough estimation of the rostral boundary of PM.
The third subdivision, the focus of this review, is the part of the agranular frontal cortex lateral to MII and rostral to MI: the premotor cortex (PM).

Hines (1929) appears to have been the first to use the term "premotor cortex," which was adopted later by Fulton and his collaborators (Fulton et al 1932, Bucy & Fulton 1933, Fulton 1934, 1935, Jacobsen 1934, Kennard et al 1934). However, their "premotor area" contained the supplementary motor cortex as it was later defined by physiological methods (Woolsey et al 1952, Penfield & Welch 1951). It seems advisable, therefore, to restrict the name "premotor cortex" to the more laterally situated part of the agranular frontal cortex, the region outside of the boundaries of both the primary motor cortex (MI) and the supplementary motor cortex (MII) as defined physiologically. Although the terminology used here is not identical to all of the historical uses of the terms "premotor cortex" or "premotor area," this system of nomenclature is consistent with that of several contemporary investigators (Brown et al 1979, Brinkman & Porter 1983, Roland et al 1980a, b, Wiesendanger 1981, Weinrich & Wise 1982, Freund & Hummelsheim 1984). Together, MII and PM are called the "nonprimary motor cortex" in this review in order to contrast them with the primary motor cortex (Wise 1984), and the term "motor cortex" is used in its general sense to refer to all motor cortical fields.

The relation of MI, MII, and PM to cortical cytoarchitectonics remains uncertain. In one view, MI is taken to be identical to Brodmann's area 4, and PM and MII together occupy area 6 (see e.g. Hartmann-von Monakow 1979, Asanuma et al 1983). There are many alternative views, however, most of which depend upon the architectonic descriptions of Vogt & Vogt (1919). Based on a comparison of MI as defined by physiological methods and the cortical map of Vogt & Vogt, several investigators (e.g. Woolsey et al 1952, Humphrey 1979, Wiesendanger 1981) have concluded that MI corresponds to area 4 plus a part of area 6. It is often assumed, invariably without architectonic documentation, that the part of area 6 to be included within MI corresponds to a cortical field termed 6α by Vogt & Vogt (1919), but no one since the Vogts has been able to distinguish area 6α from the rostrally adjacent field, 6αβ. Accordingly, and in view of the uncertainty about the exact location of the area 4/6 boundary within the precentral gyrus, it seems most useful to abandon these architectonic terms and instead use those derived from neurophysiological studies.

**History of Premotor Cortex Study**

**FULTON'S FORMULATION** Fulton's studies of the nonprimary motor cortex marked the starting point of the experimental investigation of this region. One should not be too surprised to find that these early studies now appear primitive
and inconclusive, or that they engendered controversy when other investigators, with different views of the nebulous cortical boundaries involved, approached similar questions. However, many of the concepts entertained by Fulton and his colleagues in early studies of the premotor cortex appear more accurate, in the main, than many of the more popular views that dominated the intervening years.

The primary contribution of Fulton and his school was to solidify the concept of a nonprimary motor cortex with distinct motor functions. The lengthy and confusing arguments about the site necessary for and types of spasticity and abnormal grasping reflexes are not reviewed here. Several treatments of this subject are available (e.g. Humphrey 1979, Wiesendanger 1981), but such observations have failed to elucidate the problem of motor cortex function and specialization. In brief, Fulton and his colleagues asserted that ablations of the nonprimary motor cortex in monkeys, apes, and man led to spasticity, forced grasping, and vasomotor disturbances (Fulton et al 1932, Kennard et al 1934, Jacobson 1934, Kennard 1935, Fulton 1934, 1935), but the most interesting effect they claimed to observe was a specific deficit in the execution of skilled movements.

Of course, there are serious problems with Fulton’s experiments. In none of the studies purporting to describe a premotor syndrome was the extent and location of the cortical damage accurately assessed. Indeed, in view of the lack of agreement concerning the architectonic correlates of frontal cortical fields, it is easy to understand the problems these investigators had in the localization of ablations and tumors. Even accurate localization of the damaged sites would not have entirely solved the problem, since the cortical fields had not been well defined. In the context of these difficulties and imprecise formulations, controversy and disagreement would seem inevitable, and they developed rather early in the study of the premotor cortex. Foerster (1936), for example, stated that in his experience with 40 patients with surgical damage to the premotor cortex, he observed none of the symptoms that Fulton described. Walshe (1935, p. 61) went further to deny almost all of Fulton’s fundamental observations as “so paradoxical that their interpretation has so far proved impossible.” Reviewing these controversies in detail would not be fruitful, but it should be noted that it was in the context of such uncertainty that subsequent investigations, most notably Woolsey’s, took place.

WOOLSEY’S WORK Woolsey, along with Penfield & Welch (1951), definitively established the concept of a nonprimary motor cortex by discovering the supplementary motor cortex, a motor representation on the medial aspect of the agranular frontal cortex. However, the studies of Woolsey and his colleagues (Woolsey et al 1952, Travis 1955) led them to the conclusion that the premotor cortex was not a part of the motor cortex and played no role in the cerebral
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control of movement. Two sets of observations led Woolsey and Travis to these conclusions: (a) electrical shocks applied to the premotor cortex of monkeys deeply anesthetized with barbiturates did not evoke movements, and (b) ablations of premotor cortex that were added to previous lesions of either the supplementary motor cortex or the precentral motor cortex did not cause additional gross abnormalities in locomotion or muscle tone. Certain problems with these studies are described below, but even if one were to accept these negative results, it is difficult to understand the strength and confidence with which these investigators eliminated the premotor cortex from a role in the cerebral control of movement. Woolsey et al (1952, p. 259) concluded that it “. . . should be evident that our findings are incompatible with the idea of a premotor cortex as that concept has been understood for the past two decades [1931–1950].” And Travis (1955, p. 186) concluded, in the same vein, that “. . . lesions of the dorsal and lateral ‘area 6’ not included in the precentral motor area do not contribute observable motor deficits in the limbs. The only functional portions of ‘area 6’ are those included in either precentral or supplementary motor areas.”

However, aside from their negative nature, the observations that formed the basis of these conclusions were further weakened by methodological problems. It had already been reported that the level of anesthesia was a critical variable in experiments involving premotor cortical stimulation in monkeys (Bucy 1933, Bucy & Fulton 1933). It is therefore not surprising that stimulation of the premotor cortex failed to evoke movements in the deeply anesthetized preparations used by Woolsey. Moreover, the aphysiological nature of electrical stimulation should lead to rather cautious interpretation of such results; certainly the absence of “electrical excitability” of a cortical field should not be construed as demonstrating its lack of participation in the cerebral control of movements. As for the study of Travis, an assessment of the effects of premotor cortex ablations that includes mainly gross observation of locomotion, muscle tone, etc. without quantitative measures, should be viewed with skepticism and interpreted with caution. In any event, the lack of chronic gross motor deficits after ablation of the premotor cortex should have been expected if, as had already been suggested at the time of Travis’s study, it is involved in “higher-order” aspects of motor control (for further discussion see Wise 1984).

In conclusion, neither the work of Woolsey nor that of his collaborators yielded any convincing evidence against the concept of a premotor cortex as a distinct cortical field with a specialized role in the cerebral control of movement. However, although a few neuroanatomists and clinical neurologists continued to have an interest in the premotor cortex, neurophysiologists and neuropsychologists rarely studied this region for the next quarter of a century.
Definition of the Premotor Cortex

GENERAL CONSIDERATIONS Cortical field definition typically requires synthesis of data obtained by many methods from a large number of different laboratories. The criteria that can be used to define a cortical field include architectonics, connectivity, effects of electrical stimulation, activity of neurons or groups of neurons in the field, and the behavioral effects of damage to the cortical field. Rarely does any one of these features clearly and unambiguously serve to define a cortical field, and even more rarely can such a criterion be applied uniformly to a large number of species or to more than one boundary of a field. Usually, and this is clearly the case for the premotor cortex, many bits of information must be collated and compared to arrive at a constellation of properties that serve to define a cortical field. Complicating this process further is the unfortunate but unavoidable fact that not all the published reports are equally thorough and reliable in their description and documentation of results. Additional problems have been caused by the practice of using several terms and concepts in vague, ambiguous, and often confusing ways. In the discussion below, the premotor cortex of macaque monkeys is treated as a single cortical field located roughly as indicated in Figure 1. The term “premotor cortex” is used regardless of the terminology of the cited reports, and it is taken to include the arcuate premotor area (Schell & Strick 1984), peri-arcuate cortex (Godschalk et al 1981), post-arcuate cortex (Rizzolatti et al 1983), and area 6 of others.

DEFINITION BY EFFERENT CONNECTIVITY In this review, I discuss only those aspects of cortical connectivity that may help to distinguish premotor cortex from adjacent cortical fields. Two aspects of corticofugal organization are most relevant. First, it has been reported that PM (along with MII) projects only to the parvocellular part of the red nucleus, whereas MI projects to both its parvocellular and magnocellular parts (Kuypers & Lawrence 1967, Catsman-Berrevoets et al 1979, Hartmann-von Monakow et al 1979). Second, according to Sessle & WiesendANGER (1982), whereas MI and MII project directly to the spinal cord, PM does not. This view is consistent with older reports that PM sends its most prominent corticofugal projection to the medullary reticular formation (Kuypers & Brinkman 1970, Catsman-Berrevoets & Kuypers 1976). It should be noted, however, that other reports indicate that some corticospinal neurons may be located within PM (Murray & Coulter 1981, see also Toyoshima & Sakai 1982). Nevertheless, it seems likely that the lack of a prominent, direct corticospinal projection may serve to distinguish PM from both MI and MII. Other corticofugal projections (see Künzle 1978) are not very useful for the definition of PM because, like the corticopontine and corticostriatal projections, they arise from most of cerebral cortex (Kemp
& Powell 1970, Brodal 1978). Perhaps future studies will reveal specific corticofugal connections, e.g. corticopretectal or corticosubthalamic projections, that will serve to improve the definition of PM. Descriptions of corticocortical projections from the premotor cortex to the primary motor cortex (Pandya & Vignolo 1971, Muakassa & Strick 1979) have also helped to define the field, and more detailed analysis promises a further contribution in this regard.

DEFINITION BY AFFERENT CONNECTIVITY Schell & Strick (1984) have recently concluded that the main input to at least a part of premotor cortex arises from the caudal parts of the deep cerebellar nuclei and relays via nucleus X of the ventrolateral complex of thalamic nuclei (see also Rizzolatti et al 1983). The thalamic input from this part of the ventrolateral thalamus distinguishes it from MI and MII, which receive their main thalamic input from other parts of the thalamus. Further, Avenaño et al (1983) reported recently that the amygdaloid complex projects to PM, but not to MI.

Corticocortical inputs also help to distinguish PM from other motor cortical fields. Lateral parts of area 5, but more particularly areas 1 and 2, have been reported to project to MII and MI, but not to PM (Jones et al 1978). In addition, a number of frontal, visual, and auditory areas (Pandya & Kuypers 1969, Chavis & Pandya 1976, Godschalk et al 1983), including area 7, are thought to project to PM and not to MI.

DEFINITION BY NEUROPHYSIOLOGICAL METHODS Weinrich & Wise (1982) and Sessle & Wiesendanger (1982), supporting an earlier observation of Sakai (1978), have reported that intracortical electrical stimulation in the cortex rostral to MI rarely evokes movements, whereas within MI such movements are quite commonly elicited (Asanuma & Rosén 1972, Kwan et al 1978). This is not to say that movements cannot be evoked from premotor cortex, especially if stimulation points are narrowly spaced and higher currents are used, but rather that the frequency with which such movements can be evoked by relatively low current levels serves as a useful and convenient artifice for distinguishing PM from MI.

In addition to the features described above that may serve to distinguish PM from other cortical fields, the patterns of neuronal activity observed during the performance of a motor task are quantitatively different in MI and PM. The proportion of neurons apparently responding to visual stimuli that instruct a monkey about forthcoming movements (signal-related cells), or that show sustained changes in discharge rate during periods of motor preparation (set-related cells), is much larger in PM than in MI (Weinrich & Wise 1982, Weinrich et al 1984, Godschalk et al 1981, see also Lamarre et al 1983, but cf Kwan et al 1981). Rizzolatti et al (1981b) also report visual inputs to a region just lateral to the main recording site used by Weinrich & Wise. It
seems most likely that the region studied by Rizzolatti and his colleagues is also within the premotor cortex. Brinkman & Porter (1979, 1983, Porter 1983) have reported that PM neurons rarely respond to somatic sensory input, in contrast to neurons in MI, and, if true, this feature may help to distinguish these cortical fields. However, Rizzolatti et al (1981a) found substantial somatic sensory inputs to PM, so further study is needed to determine whether the relative sensitivity to somatic sensory inputs can be used to distinguish PM and MI.

DEFINITION BY CYTOARCHITECTONICS  Since the differences between the structure of PM and MI are much less pronounced than their similarities, it is at best difficult to describe and document reliably the cytoarchitectonic differences between these fields. This difference is often stated to be the presence of giant layer V pyramidal cells (of Betz) in MI and their absence in PM, a criterion that dates from Brodmann (1905, 1909). While there is an obvious difference between the rostral and caudal parts of the agranular frontal cortex in the density of giant pyramidal cells, the “Betz-cell criterion” suffers from two major difficulties. First, the distribution of cell-body diameters in the agranular frontal cortex shows no obvious bimodality, and therefore the distinction between Betz and non-Betz cells on the basis of size is, by necessity, arbitrary. Second, there are some very large cells, Betz cells by any published definition, located so far rostral in the agranular frontal cortex that it is inconceivable that they are all confined to the primary motor cortex (or area 4 by anyone’s definition). Thus, the attempt at a simple distinction between MI and PM on the basis of the existence of Betz cells alone (e.g. Brodmann 1909, Bucy 1933, von Bonin & Bailey 1947, Denny-Brown & Botterell 1948) is bound to be unreliable.

In spite of these difficulties, it has been possible to show that the change in microstimulation threshold described above for the PM/MI boundary corresponds, at least within 2 mm, with a marked change in the density of giant layer V pyramidal cells. As electrode penetrations move from the lower-threshold (MI) to higher-threshold (PM) microstimulation zones, the density of large layer V pyramidal cells abruptly drops (Weinrich & Wise 1982, Wise 1984, see also Sessle & Wiesendanger 1982). Thus, the classical distinction drawn between PM and MI is generally correct, but the differences are not absolute. Other cytoarchitectonic differences are apparently subtle or inconsistent, although the possibility remains that future quantitative architectonic investigations, in conjunction with axonal fiber tracing techniques, neurophysiology, or other methods, may yield a clearer structural definition of PM. Regarding the boundary between PM and MII, no architectonic difference has ever been reported.

Most of this discussion focuses on an attempt to distinguish PM from MI. The distinction between premotor cortex and the “prefrontal” cortex, i.e. the frontal granular cortex, is presumably possible on purely cytoarchitectonic
grounds, but very little work has been directed toward this question. It is known, however, that the distribution of contralaterally directed corticotectal cells respects this boundary. These cells are found almost exclusively within the rostral part of the agranular frontal cortex (Distel & Fries 1982). Whether this part of the frontal cortex is a separate cortical field or part of the premotor cortex is not yet known.

CONCLUSIONS  On the basis of a constellation of physiological, connectional, and architectonic properties it is possible to draw the conclusion that the premotor cortex constitutes a distinct cortical field within the frontal agranular cortex. MI can be distinguished from PM on the basis of the following characteristics: its lower threshold of microstimulation; its complete representation of the body as revealed by electrical stimulation; its lower proportion of certain neuronal activity patterns, e.g. those indicating visual inputs; its higher density of giant layer V pyramidal cells; its projection to the magnocellular red nucleus; its lack of input from the amygdala; its input from a different main relay nucleus in the thalamus; and its major projection to the spinal cord.

MII, in turn, can be distinguished from PM by virtue of its complete body representation, its pronounced corticospinal projection, and its input from a distinct thalamic main-relay nucleus. The prefrontal cortex and PM can, presumably, be distinguished on cytoarchitectonic grounds. The former has a clearly observable internal granular layer, whereas the latter is agranular. The behavioral effects of damage to the premotor cortex (Halsband & Passingham 1982), discussed below, provide further evidence that it is a distinct cortical field.

That the premotor cortex should be considered part of the motor cortex is supported by the pattern of its connections with structures involved in motor control: its input from the cerebellum via the thalamus (Schell & Strick 1984); its output to the red nucleus, striatum, basilar pontine nuclei, subthalamic nucleus, and medullary reticular formation; and the high proportion of neurons closely related to voluntary limb movements (Kubota & Hamada 1978, Weinrich & Wise 1982, Brinkman & Porter 1983).

PREMOTOR CORTEX AND ITS ROLE IN THE CEREBRAL CONTROL OF MOVEMENT: PREPARATION, PROGRAMMING, AND OTHER PROPOSALS

Complex Movements
As mentioned above, Fulton (1935, 1938) and, later, Luria (1980) speculated that the primate premotor cortex is especially important in the synthesis of skilled motor sequences. The fundamental observation in this area was that of Jacobsen (1934), who reported that a chimpanzee, after ablation of the
premotor cortex, was deficient on a task involving the movement of a series of latches in order to open a box. However, because Jacobsen included the supplementary motor cortex within his “premotor cortex,” it is possible to attribute any observed deficit to MII damage. Recently, Deuel (1977) showed some deficits in latch-box opening in macaque monkeys after frontal cortex damage, but she also ablated a large amount of cortex outside the premotor cortex. In any event, correct performance of the latch-box and related tasks requires complicated combinations of sensorially guided movements, independent movements of muscle groups that usually work in concert, and memory of motor sequences. In addition, possible motivational and attentional variables make these experiments difficult to interpret. Still, the idea that the premotor cortex is necessary for complex sequencing of movement remains an intriguing possibility and one that might be important in understanding human speech mechanisms (Passingham 1981). However, Halsband & Passingham (1982) reported no deficit in the performance of a fixed sequence of forelimb movements after ablation of the macaque premotor cortex. Thus, if one accepts that their monkeys’ lesions were as intended, the premotor cortex is not necessary for the execution of a fixed motor sequence. Of course, this finding does not imply that the premotor cortex is uninvolved in the performance of such tasks or less fixed sequences.

Roland and his collaborators (1980a,b) have shown, in human subjects, that significant increases in regional cerebral blood flow occur in the premotor (and the supplementary motor) cortex when subjects perform a complicated sequence of digit movements, but not when they perform a single repetitive digit movement or a sustained isometric contraction. This finding supports the idea that the premotor cortex may play an important role, along with other parts of the nonprimary motor cortex, in the execution of relatively complex movements. However, this view requires further direct experimental analysis and should be considered speculative at this time (see also Freund & Hummelsheim 1984).

Sensory Guidance of Movement

A number of studies support the idea that the premotor cortex may play an important role in the sensory guidance of movement. Neuroanatomical studies (e.g. Pandya & Kuypers 1969, Chavis & Pandya 1976) have been taken to support this view insofar as they show putatively visual (and other sensory) cortical areas projecting to the general region of the premotor cortex. In addition, some specific experimental evidence supports this hypothesis. Attempts to disconnect visual areas from premotor regions in macaque monkeys lead to marked deficits in visually guided movements, especially those requiring independent digit movements (Keating 1973, Haaxma & Kuypers 1975).

Several other behavioral reports could be interpreted in terms of deficits in sensorial guidance of movements. For example, Halsband & Passingham (1982)
have reported that monkeys with ablations of the premotor cortex are deficient in an abstractly guided visuomotor task (see also Petrides 1982). Specifically, the monkeys were impaired in performing a task that required them to turn a handle by pronation in response to a signal of one color, and to pull the same handle in response to a signal of another color. Rizzolatti et al (1983) have also found deficits in sensorially guided movements after more restricted premotor cortex lesions. Many effects of cortical ablations directed toward the arcuate sulcus or prefrontal cortex, but probably involving the premotor cortex, can also be interpreted in terms of deficits in certain types of sensorially guided movements (see Wise 1984).

In man, Roland et al (1980b) have shown that the premotor cortex (along with MII) increases its blood flow during the execution of a sensorially guided movement. In one of these tasks, the “maze task” in their terminology, a subject is verbally instructed to make a limb movement of a certain magnitude and direction. There are, of course, many areas that show blood flow increases during such a task, but increases in the premotor cortex are consistent with the idea that this region is important for sensorially guided movements, especially those of an abstract nature. In addition, Roland & Larsen (1976) report blood flow increases in the premotor cortex during stereognostic testing, which may also be considered to involve the sensory guidance of movement.

Single-unit recording methods, as applied to unanesthetized monkeys, have also been used to support the argument that the premotor cortex plays a role in sensorially guided movements. For example, Rizzolatti et al (1981b) have reported visual inputs to premotor cortex. The receptive fields of premotor cortex cells may be independent of gaze direction (Gentilucci et al 1983), an important property for visual control of movement. Other investigators have reported similar neuronal activity without explicitly studying visual receptive fields (Godschalk et al 1981, Weinrich & Wise 1982, Godschalk & Lemon 1983, Wise et al 1983, Brinkman & Porter 1983). The role of this visual input in visuomotor mechanisms is unknown, however. Regarding the motor aspects of sensorially guided movements, the vast majority of premotor cortex neurons have discharge modulations temporally correlated with and preceding the onset of movement (e.g. the “movement-related cells” of Weinrich & Wise 1982, Brinkman & Porter 1983). The magnitude of modulation in some cells is correlated with movement parameters such as acceleration or velocity (Kubota & Hamada 1978, Weinrich et al 1984), but the number of such cells appears to be small. At present, the role of these patterns of activity in the control of sensorially guided or any other movements is unknown.

Preparation for Motor Action

Most commonly, the concept of motor preparation is thought of in terms of motor programming, i.e. the assembly of motor subroutines for later execution
(Sternberg et al 1978) or the assembly of force and time specifications for all of the muscles involved in an individual movement (Brooks 1979, Paillard 1983), including prime mover and postural support muscles. But there are several aspects of the preparation for movement—motor set (which includes the concepts of programming, postural stabilization, and reflex suppression), selective attention, arousal, goal selection, and strategy formulation—and all of these concepts may be important in understanding premotor cortex function.

**MOTOR SET** A general aspect of motor preparation is *motor set*, the state of readiness to make a particular movement (Evarts et al 1984). It is possible to test the idea that the premotor cortex plays a role in motor set by examining the activity of premotor cortex cells during an *instructed delay period*, i.e. after an animal has received an instruction for a limb movement, but before it has received a signal allowing it to execute the movement. Neuronal activity during periods of time when the monkey can reasonably be inferred to be set to move his limb can then be compared with conditions in which such preparation is unlikely.

One class of cells in premotor cortex exhibits directional specificity; that is, these cells show sustained excitation after an instruction for movement in one direction and show inhibition or unchanged activity after an instruction for movement in the opposite direction (Figure 2). However, for most of this class of premotor cortex neuron, when the visuospatial stimulus signals the monkey to withhold movement during a given behavioral trial, there is little or no change in neuronal activity (Wise et al 1983, Weinrich et al 1984). Thus, the activity of these cells seems to be specific for situations in which the monkey is preparing to move the limb. This class of premotor cortex neuron also typically shows directional specificity regardless of whether the instruction is given in the auditory or visual modality (Weindch & Wise 1982)—a feature to be expected of a cell involved in the preparation for movement. Further, if activity during an instructed delay period reflects motor set, then (a) removing the instruction stimulus should have no long-lasting effect on that activity and (b) changing the instruction should overtly change the pattern of neuronal activity. Both of these predictions have been confirmed (Mauritz & Wise 1983). For example, as the instruction is changed from one requiring leftward limb movement to one requiring rightward movement, the pattern of activity rapidly changes to reflect the new motor set (Figure 2). These data are consistent with the view that the premotor cortex plays a role in the preparation for specific voluntary movements, i.e. motor set.

**MOTOR PROGRAMMING** Substantial interest in premotor cortex has focused on motor programming, one aspect of motor set. Roland et al (1980b, pp. 137, 146) have speculated on the basis of their regional cerebral blood flow
Figure 2. Three peri-event rasters and histograms for a premotor cortex neuron. Each line of each raster represents the neuronal discharge during one behavioral trial, centered on the presentation of a visuospatial instruction stimulus (IS). The IS informed the animal about the next movement to be made with the forelimb. An IS for a limb movement to the right (RIS) or left (LIS) on any given trial was followed, at a variable time, by another stimulus that signaled the monkey to begin the movement. The time of this later signal, the trigger stimulus (TS, open arrow), is indicated by the heavy square mark on each raster line. In the right panel, the instruction is changed at the time indicated by the second vertical arrow (LIS → RIS), 1 s after the first instruction for those trials. The three conditions illustrated separately here were randomly intermixed during recording of the cell’s activity. The unit activity appears to represent the animal’s motor set for future movements to the right, and the unit also shows modulation in association with the movement of the limb to the right. Histogram scale, impulses/s; binwidth, 40 ms. The time from the IS to the vertical bar bordering each raster on the right is 4 s.

Studies in man that the “premotor areas are activated when a new motor program is established or a previously learned motor program is modulated,” i.e. during nonrepetitive voluntary movements, or when a program is “changed on the basis of sensory information, as will be the case during exploratory manipulation of objects.” Rizzolatti et al (1981a–c, 1983), based on their studies of macaque monkeys, also have suggested a role for premotor cortex in programming (“praxic”) functions for complex movements, with subregions of the premotor cortex involved in the organization of specific motor acts, such as bringing food to the mouth by hand.

Although these suggestions are interesting, the hypothesis that the premotor cortex is necessary for motor programming or for the storage of learned motor programs still lacks clear experimental support. These speculations need to be elaborated and pursued with experiments employing controlled motor tasks. It seems likely, though, on the basis of presently available information, that the premotor cortex is not necessary for all types of programming. For example, monkeys with premotor cortex ablations remain capable for performing a variety of motor tasks, including motor sequences and movements involving finely controlled digit movements (Halsband & Passingham 1982). Thus, the
aspect of motor programming that involves the specification of time and force parameters for skeletal movements does not require the premotor cortex. If the premotor cortex is necessary for motor programming, it might be specific for those programs guided by external environmental stimuli.

**AXIAL AND PROXIMAL LIMB STABILIZATION**  One common proposal for a function of the premotor cortex involves a role for it in stabilizing the limb, mainly or exclusively via proximal and axial musculature. This aspect of motor programming is thought to be important for the execution of distal movements such as visually guided grasping of an object (Humphrey 1979).

Pribram et al. (1955/1956) proposed a proximal and axial specialization for the rostral part of the agranular frontal cortex on the basis of cortical ablations in macaque monkeys. However, their lesions included the supplementary motor cortex, and those cases with the clearest proximal motor dysfunction also involved the rostral part of the primary motor cortex. Accordingly, it is difficult to attribute the deficits they observed to the premotor cortex. The historical development of the idea that the premotor cortex controls exclusively proximal muscles also might have depended, in part, on confusion over the term "area 6." The most rostral part of the primary motor cortex, sometimes held to overlap into area 6, is usually thought to contain the proximal and axial parts of the motor representation (Woolsey et al. 1952, Kwan et al. 1978, Fetz et al. 1980). Accordingly, the image of axial and proximal motor control has sometimes been unjustifiably extended to area 6 as a whole, including the premotor cortex (and the supplementary motor cortex). A recent clinical study has indicated that humans with damage to the premotor cortex have weakness of the proximal musculature and difficulty in motor tasks that require proximal support of the arm (Freund & Hummelsheim 1984). This finding implies a significant role for the premotor cortex in the control of proximal limb muscle under certain circumstances, but should not be construed to rule out an additional role in the control of distal limb movements (see also Brinkman & Porter 1983).

The hypothesis that the premotor cortex participates in a limb-stabilization function may find support in the older observations of cortical electrical stimulation effects, which are suggestive of an axial or proximal specialization (e.g. Bucy 1933, Bucy & Fulton 1933). However, these findings and similar ones in humans are difficult to interpret for a variety of reasons, mainly the apophysiological nature of the technique itself and the effects of anesthesia.

As for neuroanatomical data, Künzle (1978) states that the projections from area 6 to precentral motor cortex are directed to the proximal parts of its representation. This finding might also suggest a proximal specialization, but Künzle presents no documentation for his contention. Muakassa & Strick (1979), on the other hand, report a strong input from the premotor cortex to
the “wrist representation” of the primary motor cortex. Of course, an input to a distal part of the primary motor representation does not, in itself, provide evidence that premotor cortex is important in distal motor control, but the converse proposition is unsupported by current anatomical data.

Behavioral experiments are also relevant to this problem. Halsband & Passingham (1982) compared the effects of premotor ablations on a number of behaviors. The requirement for postural upper arm support was not a determining factor in the pattern of deficits they observed; their monkeys could perform a fixed sequence of movements that required comparable postural support to their “conditional motor task,” described above, on which the animals were deficient. At the present time, a cautious estimate would be that whereas the premotor cortex may be very important in the control of proximal and axial muscles, it is unlikely to be exclusively concerned with such muscles. In the present context, it should be noted that proximal fixation of the limb for distal action can be considered an aspect of the preparation or programming for such movements.

SUPPRESSION OF “LOWER” MOTOR ACTS One of the roles of “higher motor centers” might be to suppress or facilitate “lower” motor control circuits. This concept, although not always explicitly stated in these terms, has been influential in thinking about the function of the premotor cortex. Denny-Brown (1966, Denny-Brown & Botterell 1948) thought that the premotor cortex functioned to suppress certain reflexes and that its removal caused “release” of these reflexes. Although this view cannot be ruled out, it reflects a curious pattern of thinking about the effects of brain lesions. In this view, ablations reveal a negative image of the functional specialization of the region in question. In the analysis of complex central nervous system circuits, such interpretations are unlikely to be of any enduring conceptual value.

Nevertheless, it may be realistic to view one important role of premotor cortex as suppression of relatively automatic responses to certain sensory stimuli. Moll & Kuypers (1977) interpreted the effects of large nonprimary motor cortex ablations (including the premotor cortex and several additional frontal fields) in this context. In their experiment, monkeys with such brain damage continued to reach directly toward a desired object, even when the pathway was obstructed by a transparent barrier and an alternative trajectory would have been successful. It was suggested that the monkeys were deficient in this task because they could not suppress direct and immediate reaching for the object. Such suppression might be another aspect of the preparation for movements, especially for delayed movements or those of a complex nature.

VASOMOTOR EFFECTS Very little is known about possible autonomic functions of the cerebral cortex in primates, but vasomotor regulation might be
important in the preparation for movement. Vasomotor changes were reported after premotor cortex damage in both humans (Kennard et al. 1934) and monkeys (Kennard 1935), and Woolsey et al. (1952) speculated about an autonomic role for this part of the cortex. Of course, these effects may well have been due to involvement of cortical fields other than the premotor cortex.

**ATTENTION** Rizzolatti and his colleagues (1983) have recently proposed that the premotor cortex of monkeys is necessary for selective attention, especially to stimuli near the animal. It is difficult, however, to distinguish attentional deficits from those involving the sensory guidance of movements. Since the animals' motor orientation toward a stimulus is the method used to determine an attention deficit, it remains possible that such deficits reflect difficulty in sensorially guided movements. If this is the case, then the results of Rizzolatti et al. suggest that somewhat different mechanisms guide movements in relation to near and distant stimuli. Nevertheless, mechanisms of selective attention are undoubtedly important in the preparation for motor action and have many features in common with motor set (see Evarts et al. 1984).

**SUMMARY**

The concept of a separate premotor cortical field involved in the cerebral control of movement went out of favor among neurophysiologists during the quarter century from 1952 to 1977, but recent studies have led to its rehabilitation. The premotor cortex appears to be one of at least three fields within the motor cortex, two others being the primary motor cortex and the supplementary motor cortex. Several proposals have been presented concerning the functional specializations of the premotor cortex. Although no specific hypotheses have very strong support at present, the best evidence favors a role for premotor cortex in the preparation for and the sensory guidance of movement.

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