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The Subdivisions of the Frontal Cortex Revisited

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INTRODUCTION

Gallia est omnia divisa in partes tres. With these words Caesar set out to conquer Gaul and to unify it. My purpose in this chapter is no less ambitious: to describe the various subdivisions of the frontal lobe in order to find some unitary principle that unites them. Fortunately, there are available the endeavors of massive troops, endeavors that make the enterprise possible. The hoped-for conquest will proceed as follows: The initial section describes anatomical data, and serves as an orientation; the second section is concerned with reviewing and interpreting the mass of neurobehavioral data that deal with the functional parcellation of the anterior frontal systems; and finally a summary and synthesis section will attempt to portray an understanding of primate frontal lobe function in terms of the currently available data.

SOME ANATOMICAL CONSIDERATIONS

Thalamocortical Definition of Subdivisions

As with Caesar's Gaul, the frontal cortex of primates can be divided into three major parts, each of which is made up of subprincipalities. The three major divisions are the precentral (including the pre- and supplementary motor), the anterior (also called prefrontal, orbitofrontal, and far frontal), and the cingulate (also called limbic). These major divisions can be defined on the basis of their thalamic projections: The precentral derives its thalamic input from the ventrolateral group of nuclei, the anterior frontal from the nucleus (n.) medialis dorsalis, and the cingulate from the anterior group (for reviews, see Pribram 1958a, 1958b).

[9]The subdivisions of these major divisions can also be defined in terms of their thalamic input: The immediate precentral cortex receives an input from the n. ventralis lateralis, pars caudalis, and the n. ventralis posterior, pars oralis, which in turn are the major terminals of cerebellar projections. The premotor portions of this division receive an input from the n. ventralis lateralis, pars oralis, which in turn is the major termination of input through the globus pallidus of the lateral nigrostriatal system. A further subdivision can be made between the lateral premotor and the supplementary motor systems in that the more laterally placed systems deal more with orofacial, and the supplementary motor systems with other axial muscular projections (Goldberg, 1985).

The subdivisions of the cingulate cortex follow the subdivisions of the anterior thalamic nuclei: N. anterior medialis projects to the anterior cingulate cortex, n. anterior lateralis to the posterior cingulate cortex (Pribram & Fulton, 1954). The n. lateralis dorsalis (which ought to be classified as part of the anterior group) projects to the retrosplenial portion of the cingulate gyrus.

Finally, the primate anterior, far frontal cortex can be subdivided according to the subdivisions of the n. medialis dorsalis: The microcellular portion projects to the dorsolateral frontal cortex, the perilamnilar magnocellular portion to the periarcuate cortex, and the midline magnocellular portion to the orbitofrontal cortex (Pribram, Chow, & Semmes, 1953).

A Frontolimbic versus Cortical Convexity Distinction

There are additional, hitherto ignored, interesting and important (for understanding the functional relationship to psychological processing) findings regarding the thalamocortical projections. The thalamus is a three-dimensional structure, whereas the cortex is (from the standpoint of thalamic projections) essentially a two-dimensional sheet of cells. Thus, the projections from thalamus to cortex must "lose" one dimension. When one plots the precisely arranged "fan" of projections from each thalamic nucleus one can readily determine which dimension is eliminated.

With regard to the projections from the anterior nuclear group and the n. medialis dorsalis, the anterior-posterior dimension is eliminated. An anterior-posterior file of cells in the thalamus projects to a single locus of cortex. Thus, for example, one finds degeneration of such an extended row of thalamic cells ranging from the most anterior to the most posterior portion of the n. medialis dorsalis after a resection limited to the frontal pole (Pribram et al., 1953).

With regard to the ventrolateral group of nuclei, the situation is entirely different. Here the anterior-posterior dimension is clearly maintained: The front part of the nucleus projects to the forward parts of the cerebral convexity; as one proceeds back in the thalamus the projections reach the more posterior portions of the cortex, curving around into the temporal lobe when the projections of the pulvinar are reached. On the other hand, a file of cells extending, more or less,

dorsoventrally (but angled somewhat laterally from its medial edge) projects to a single locus on the cortex (Chow & Pribram, 1956).

This distinction between the anterior and medial nuclei, on the one hand, and the ventrolateral group of nuclei, on the other, is supported by the fact that the internal medullary lamina separates the two classes of nuclei. Clearly, therefore, we should seek commonality among the functions of the anterior, far frontal parts of the cortex and the limbic formations, as well as among the functions of the precentral and postcentral portions of the cerebral mantle (Pribram, 1958a, 1958b).

The close anatomical relationship of the far frontal cortex and the limbic medial forebrain is also emphasized when comparative anatomical data are reviewed. In cats and other nonprimates, gyrus proreus is the homologue of the far frontal cortex of primates. This gyrus receives its projection from the midline magnocellular portion of the n. medialis dorsalis. This projection covers a good share of the anterior portion of the medial frontal cortex; gyrus proreus on the lateral surface is limited to a narrow sliver. It is as if there has been a rotation of the medial frontal cortex laterally (just as there seems to have occurred a rotation medially of the occipital cortex—especially between monkey and man) during the evolution of primates.

Apraxia: A Rolandic versus Extra-Rolandic Distinction

A further lesson can be learned from an analysis of the precise arrangement of thalamocortical projections and from comparing nonprimate with primate cortical anatomy. In tracing the thalamic projections to the precentral cortex, a surprising finding came to light. The dorsoventral arrangement of terminations, both pre- and postcentrally, is diametrically opposite to the arrangement of the projections farther forward and farther back. The dorsoventral terminations of the Rolandic projections reflect a lateral-medial origin from the thalamus; the dorsoventral terminations both forward and back of the peri-Rolandic cortex reflect a medial to lateral origin (Chow & Pribram, 1956).

Again, comparison of nonprimate with primate cortical anatomy clarifies this surprising finding. In nonprimate species such as the carnivores, the suprasylvian and ectosylvian gyri extend the full length of the lateral surface of the cerebral convexity. The cruciate sulcus, the homologue of the Rolandic fissure, is mainly found on the medial surface of the hemisphere, with only a minimal extension onto the lateral surface. It is as if in the evolution of primates this sulcus has migrated laterally to become the prominent central fissure, which is so intimately related to the cerebellar system.

Such a migration seems to have split the supra- and ectosylvian gyri into anterior and posterior segments. That such a split has occurred is supported by the fact that terminations of thalamocortical projections to the anterior and posterior segments originate in adjacent parts of the ventrolateral nuclei. Should

this conjecture regarding a split be correct, it would go a long way in accounting for the difficulty in making a differential diagnosis between apraxias that are due to frontal damage and those that are due to parietal damage.

Brown (1985), in a review of frontal lobe syndromes, defines apraxia as "a substitution or defective selection of partial movements with lesions of the left premotor cortex [which] is due to an alteration of motor timing or a change in the kinetic pattern for a particular motor sequence" (p. 37).

To test whether, in fact, damage to both parietal and frontal (premotor) systems can produce apraxia and to pin down in a quantitative fashion just what changes in timing, in the kinetic pattern of movement, occur in apraxia, the following (K. H. Pribram, unpublished) experiment was performed: Monkeys were trained (using peanuts as reinforcements) to move a lever in a T-shaped slot beginning at the juncture of the arms of the T with its stem. The movements were then to be directed to the right, to the left, and finally down and up, in that order. Records were kept of the monkeys' abilities to perform the movements in the correct order and the number and duration of contacts with the sides of the slots that formed the T. (This was done by having the sides and the lever lined with copper and wiring them so that contact could be recorded.)

Resections were made of precentral cortex, of the cortex of the inferior parietal lobule, of the premotor cortex, and of the latter two lesions combined. Precentral resections led to many more and briefer contacts along the path of the lever within the T slot, a loss of fine motor skill. No change in overall sequencing occurred. Both the parietal and the premotor resections produced a breakdown in the sequencing of the movements but only insofar as the same movement was carried out repetitiously, which was interpreted as evidence of apraxia. There was no observed difference between the effects of the anterior resection and those of the posterior resection, and the overall order of the act was not disturbed. When the parietal and premotor resections were combined, this deficit was enhanced; still, there was no change in overall ordering of the action. I will say more about this distinction between the systems that deal with skill and with apraxia in the summary and synthesis section.

SUBDIVISIONS OF THE ANTERIOR FRONTAL CORTEX

The main body of this review concerns the parcellation of the anterior frontal cortex, in part because so much work has been done on the topic and this work has not been adequately reviewed elsewhere, and in part because such a review leads directly to the current aim—which is to find some unifying principle for frontal lobe functioning.

When lesions occur in the Rolandic and premotor portions of the frontal lobe, neurological signs and symptoms occur that are relatively easy to spot. By contrast, the lesions of the anterior frontal cortex are essentially "silent" unless

specific and sophisticated inquiries are addressed to the organism. Such inquiry has been greatly aided by the use of nonhuman primate models of deficits in behavior produced by anterior frontal lesions.

Description of Tasks

The tasks that have been found most useful in delineating the deficit following anterior frontal damage are all characterized by a delay between stimulus presentation and the opportunity for a response to occur. During this delay distractors are introduced and the cue to the correct response disappears. The tasks fall into two main categories: delayed response and delayed alternation. Further, variations in the tasks have produced several subcategories of each category, variations that have been found to be extremely useful both as tools for subdividing the anterior frontal cortex and for understanding the nature of the deficit.

The delayed-response task, in its direct form, involves hiding, within sight of the subject, a reward in one of two identical-looking boxes set side by side, bringing down a distracting opaque screen for at least 5 seconds, and then raising the screen to provide the subject with just one opportunity to locate the reward. The boxes are immediately withdrawn beyond the subject's reach and the next trial begun. Should the subject fail to find the reward, the trial is repeated (correction technique); that is, the reward is again hidden within sight of the subject in the same box as in the previous trial. Should the subject succeed in finding the reward, another location (i.e., the box) for the hiding of the reward is chosen according to a (pseudo)random order number table.

The indirect form of the delayed-response task is more often called a delayed matching from sample. In this task a cue is presented instead of the reward during stimulus presentation; at the time of choice this cue and some other are available and the subject must choose the same cue as that initially presented in order to obtain the reward. A further variant of this task is the delayed nonmatch in which the subject must choose the cue that was not present at the time of stimulus presentation. This version combines the attributes of the delayed-response task with those of the delayed-alternation procedure.

In the delayed-alternation task the subject is not shown where the reward is located; he is simply given the opportunity to choose between two boxes. On the first trial both contain a reward. After the choice has been made, a distracting opaque screen is interposed between the boxes and the subject for at least 5 seconds and the next opportunity for choice is given. On this second trial the subject will find the reward in the box other than the one he chose initially and, if he continues to choose successfully, he will do so by adopting a win-shift strategy. Should the subject choose the empty box, the trial is repeated (correction technique). Unless this correction procedure is used, monkeys, when they are the subjects, fail to learn the alternation task (at least in 5,000 trials: K. H. Pribram, unpublished data).

Three variants of delayed alternation that have proved especially useful are a go/no-go version, the object-alternation procedure, and discrimination reversals. In the go/no-go task the subject must alternately go to fetch the reward on one trial and withhold his response on the subsequent trial. Failure to go or failure to withhold results in the repetition of the trial (correction procedure). In the object-alternation procedure the reward is alternated between two different objects rather than between two different locations. In this variant the spatial aspect of the task is reduced, a reduction that is enhanced when the objects are placed among 6, 8, or 12 locations according to a random number table (Pribram, Gardner, Pressman, & Bagshaw, 1963, 1969a). Discrimination reversals are, in fact, alternations that vary the numbers of trials that occur between the shift of reinforcement that signals the alternation. There is a gradual transition between alternation, double alternation, triple alternation, etc., and the ordinary nonreversal discrimination task. The inflection point occurs at three nonalternation trials in normal subjects, but is raised to four or five such trials after frontal lobe damage (Pribram, 1961a).

Description of Lesion Sites

Earlier, an anatomical rationale for subdividing the anterior frontal cortex was given in terms of the thalamic projections that terminate in different portions of this cortex. Unfortunately, all of the investigators involved in pursuing the parcellation experiments did not adhere to this particular mode of subdividing: Many experimenters simply divided the anterior portion of the frontal lobe into a dorsal portion centered on the sulcus principalis and a ventral portion that included both the lip of the lobe and the entire orbital surface. Furthermore, surgical result does not always match surgical intent. The fibers in the depth of the sulci (medial, orbital, and principal) in the anterior portion of the frontal lobe are separated by only millimeters and can be differentially spared only by exercising the greatest care and skill.

Despite this, meaningful conclusions can be teased out of the results of such experiments provided the various lesions are kept clearly differentiated by appropriate labels. It is therefore necessary to adopt a uniform terminology for the resections that often differs from that used in the original reports because different investigators used the same term to describe different lesions or different terms to describe the same lesion.

The greatest problem arises from the use of the term *orbital*. Here the convention will be followed that the term orbital refers to the general expanse of the ventral portion of the lobe and that, when specific portions of this cortex are referred to, orbital will be conjoined to a modifier. Thus, posterior orbital refers to the agranular cortex located in the most posterior part of the orbital cortex (Area 13 of Walker, the projection of the midline magnocellular portion of n.

medialis dorsalis of the thalamus). This cortex is intimately related through the uncinate fasciculus to the anterior insula, temporal pole, and amygdala.

The term *medial orbital* will be used to refer to the dysgranular cortex of the medial orbital gyrus that is continuous with the cortex on the medial surface of the lobe and receives a projection from the anterior thalamic nucleus (Pribram & Fulton, 1954). In keeping with the agranular and dysgranular cytoarchitecture of the posterior and medial orbital cortex, it was found to be electrically excitable; that is, head and eye movements and a host of visceral responses (respiratory, heart rate, blood pressure) are obtained when this cortex (as well as that of the anterior cingulate gyrus with which it is continuous) is electrically stimulated (Kaada, Pribram, & Epstein, 1949). This finding gave rise to the concept of a mediobasal motor cortex, and to the existence of a limbic system motor cortex, in addition to the more classical Rolandic and precentral systems (Pribram, 1961b).

The eugranular cortex on the lateral orbital gyrus is continuous with that forming the ventral lip and adjacent ventral gyrus of the frontal lobe. This cortex is part of the projection of the microcellular portion of the n. medialis dorsalis. When a lesion of this cortex is reported in conjunction with a lesion of posterior and medial orbital cortex, the lesion is here labeled as *orbitoventral*. When a lesion of this cortex is made in isolation, the lesion is referred to as *ventral*. When the resection extends laterally up to the gyrus adjacent to the sulcus principalis, the lesion is called *ventrolateral*.

Finally, a *dorsolateral* resection is identified as including the eugranular cortex surrounding the sulcus principalis. Such lesions usually extend to and include the marginal gyrus. The dorsolateral cortex is the termination of the remaining projection of the microcellular portion of the n. medialis dorsalis.

When smaller lesions are reported (e.g., periarcuate, around the arcuate sulcus; periprincipalis, around the sulcus principalis), the nomenclature is reasonably clear. When larger lesions are made they are simply referred to as *lateral frontal* when they exclude the posterior and medial orbital gyri. The resections are referred to as *medial frontal* when they are restricted to these gyri and to the medial surface of the lobe. When the entire anterior frontal cortex is removed, the lesion is referred to as *anterior frontal*.

The Orbital Contribution: The Feeling of Familiarity

A good place to begin is the orbital contribution to psychological processing because it is so closely linked to that of the limbic forebrain. Damage limited to either the medial orbital (Pribram, Mishkin, Rosvold, & Kaplan, 1952) or the posterior orbital (Pribram & Bagshaw, 1953) does not produce any impairments in performance of the direct form on the delayed-response task. Damage to both the medial and posterior orbital cortex does, however, produce a deficit in delayed-alternation performance (Pribram, Lim, Poppen, & Bagshaw, 1966; Pribram et al., 1952; Pribram, Wilson, & Connors, 1962). This deficit is due to

the accumulation of many repetitive errors of both commission and omission, which become especially apparent in the go/no-go version of the task. In fact, these lesions produce a greater deficit in this variant of the task than on the right/left version (Pribram, 1973), a result which is opposite that obtained when lateral frontal resections are made (Mishkin & Pribram, 1955).

Other effects observed after resections of the medial and/or posterior orbital damage are a decrease in aggression (Butter, Mishkin, & Mirsky, 1968; Butter, Snyder, & McDonald, 1970) and an increased tendency to put food items in mouths (Butter, McDonald, & Snyder, 1969). Both of these effects had previously been observed when posterior orbital lesions are combined with those of the anterior insula, temporal pole, and amygdala (Pribram & Bagshaw, 1953). It is such results that link the effects of orbital lesions on behavior to those of the limbic forebrain.

The following question arises: To what are such changes in behavior due? Brutkowsi has argued that the orbital lesions in monkeys and dogs produce disinhibition of ordinarily present drive inhibition rather than the more obvious perseverative interference (see the extensive reviews of the conditioning literature by Brutkowsi, 1964, 1965, and Konorski, 1972). The finding that monkeys with orbital resections continue to work harder than normals for nonfood items despite a normal preference for food items (Butter et al., 1969), a result similar to that obtained with amygdalectomized monkeys (Weiskrantz & Wilson, 1958), would seem to support Brutkowski's hypothesis, which was based mainly on work with dogs.

However, data showing that the response rates following orbital or lateral frontal resections are the same as those of normal monkeys during conditioning of an intermittently reinforced bar-press response (Butter, Mishkin, & Rosvold, 1963) plus the additional data that monkeys with orbitoventral lesions stop responding for longer periods than do monkeys with dorsolateral frontal resections when novel stimuli are introduced during a similar bar-pressing task (Butter, 1964) cast considerable doubt on a disinhibition hypothesis based solely on an increased drive for food.

The fact that failure in delayed alternation is characterized by proportionately as many errors of omission as of commission also mitigates against the drive disinhibition hypothesis (Pribram et al., 1966). Similarly damaging to a drive disinhibition hypothesis were the results of an experiment testing the object reversals using the go/no-go technique with monkeys who had sustained resections of orbital cortex (McEnaney & Butter, 1969). Once again the animals not only made more errors of commission than normals, but also more errors of omission. They perseverated their refusal to respond to the previously negative stimulus.

Further evidence along these lines comes from the fact that monkeys with large orbitoventral lesions show a greater resistance to extinction of a bar-press response even in the absence of food reinforcement (Butter et al., 1963). These results confirmed and extended those obtained earlier with total anterior frontal

and limbic (posterior orbital, insula, temporal pole, and amygdala) resections (Pribram, 1961b; Pribram & Weiskrantz, 1957) and are consistent with the finding that frontal and limbic lesions enhance the extinction of a conditioned avoidance response (Pribram & Weiskrantz, 1957).

These last results would readily fit a response disinhibition hypothesis (one that plagued limbic system research for many years) were it not for the finding of errors of omission in the delayed-alternation task. Also, monkeys with large orbitoventral resections take longer to habituate to novel stimuli (Butter, 1964) than do monkeys with total anterior frontal resections (Pribram, 1961b; and those with amygdalectomy, Schwartzbaum & Pribram, 1960). These results and those from a long series of conditioning experiments led Mishkin to propose that anterior frontal resections produce perseveration of "central sets" of whatever origin. Subsequent experimental results (Butter, 1969) showed, however, that monkeys with orbital resections do not perseverate in place- or object-reversal tasks. Furthermore, the meaning of central set, when it is extended to include a failure to habituate to novelty, tends to lose whatever precision it might previously have had.

The enhanced distractibility and sensitivity to proactive and retroactive interference, which accounts for the failure to habituate (see Malmo, 1942; Pribram, 1961a), may well be dependent on the organization of drive states, provided we understand by this that such states are composed of endocrine and other neurochemical systems (Estes, 1959). The limbic forebrain has been found to be a selective host to a variety of neuroendocrine and neurochemical secretions that can form the basis of a neural representation of the internal state of the organism by way of which neural control over peripheral endocrine and exocrine secretions is exerted (Martinez, 1983; McGaugh et al., 1979; Pribram, 1969b, 1977).

The import of this research for this review is that such neuroendocrine and neurochemical factors influence the organization of attention and intention: Habituation to novelty (registration and consolidation in the face of distraction), and therefore the organization of what is responded to as familiar, is disturbed by the lesions. Experimental psychologists test for familiarity with "recognition" tasks, and recently Mishkin (1982) has used the delayed nonmatching from sample as an instance of such a recognition procedure. Not surprisingly, he has found deficits with limbic (amygdala and hippocampus) resections and drawn the conclusion that these structures are involved with recognition memory. For those working in the neurological tradition where, since the time of Freud and Henry Head, agnosias, have been related to lesions of the parietal convexity, this conclusion is confusing. The confusion is resolved when it is realized that the delay tasks test for the dimension novelty/familiarity and not the identification of objects, which is the neurologist's definition of recognition. In short, the orbital contribution to psychological processing is to provide a critical facility to the feeling of familiarity based on processing both interoceptive and exteroceptive inputs.

Parcellation of the Lateral Frontal Cortex: Alternation Tasks

The performances of animals with partial resections of the lateral surface of the anterior frontal cortex show that a small midlateral periprincipalis lesion is sufficient to produce severe deficits in both delayed response (Blum, 1952; Goldman, Rosvold, Vest, & Galkin, 1971; Gross & Weiskrantz, 1962; Pinsker & French, 1967) and delayed alternation (Goldman & Rosvold, 1970; Mishkin, 1957). These results have been confirmed and refined by experiments in which electrical stimulation across the sulcus principalis has impaired performance (Stamm, 1967). In fact, these experiments as well as lesion data (Butters & Pandya, 1969) have demonstrated that the middle third of the sulcus serves as the focus for the deficit.

Although these forms of the tasks failed to help with further parcellation, their variants have proved most useful. In an early experiment attempting to analyze the variables important in producing the frontal deficit in solving delay problems, Mishkin and Pribram (1955) found that changing the delayed-alternation procedure from a left/right spatial alternation to an up/down spatial alternation did not improve the performance of the frontal animals. On the other hand, changing the task to a nonspatial go/no-go alternation did improve their performance. The logical conclusion from these results was that it was the spatial nature of the delayed-alternation and delayed-response cues that was significant. To test that conclusion, they followed that experiment with experiments using object-alternation tasks. These were difficult experiments as no one up to that time had been able to train monkeys successfully on object alternation. Pribram and Mishkin (1956) succeeded in training highly sophisticated monkeys on the task using the traditional two-box tray described earlier. Later Pribram (1961a) designed a six-box tray to eliminate the interference of position preferences. Both experiments found that monkeys with lateral frontal lesions were significantly impaired. In fact, the monkeys with lateral frontal lesions did almost as poorly on object alternation as on spatial alternation.

However, when monkeys were given dorsolateral lesions that did not include the ventrolateral cortex, their performances on object alternation were significantly better than those of monkeys given orbitoventral lesions (Mishkin, Vest, Waxler, & Rosvold, 1969). Moreover, examination of the data from the previous study testing monkeys with total lateral lesions (Pribram, 1961a) reveals that the orbitoventral lesion produced as much of a deficit as had the total lateral lesion. Taken together, these results suggest that the focus for producing the deficit lies in the ventrolateral cortex.

Thus, dorsolateral lesions interfere more with classical delay tasks, and ventrolateral lesions interfere more with object alternation. Further, as noted in the previous section, classical right/left alternation is more impaired by lateral frontal lesions than is go/no-go alternation, whereas limbic lesions produce the reverse. These data suggest that the variations of the delayed-response and

delayed-alternation tasks tap somewhat different functions. Pribram and his collaborators, therefore, performed a series of experiments (Anderson, Hunt, Vander Stoep, & Pribram, 1976; Pribram, Plotkin, Anderson, & Leong, 1977) using monkeys with total lateral frontal lesions that aimed at discerning the difference between the two tasks. The deficit in delayed-response performance was shown to depend on the elimination or production of distraction. Further, spatial distractors had been found to be especially potent (Grueninger & Pribram, 1969) and proved to be critical even in a test of "object constancy," where a piece of food was simply hidden as in the ordinary delayed-response task. Finally, when the location of the hiding place was shifted within sight of the monkeys, a profound deficit was produced in the operated group despite the fact that the task proved to be a rather easy one for the control subjects.

A replication of the experiment in which "parsing" with temporal tags overcame the deficit on delayed alternation was used to analyze the factors critical to the performance of that task (Pribram et al., 1977; Pribram & Tubbs, 1967; Tubbs, 1969). As expected, in this situation, spatial variables were found to be subservient to temporal. However, location of prior response proved more potent than whether that response had been rewarded in both the operated and control groups, a result that had previously also been obtained by Wilson (1962).

Thus, a mix of spatial and temporal factors is critical to the performance of both delayed response and delayed alternation. But the mix is not equal. The spatial factor is more important in delayed response than in delayed alternation where it can be dispensed with entirely in variants such as the go/no-go and object alternation. As was noted earlier, the delayed-response deficit is maximal when lesions are made in the dorsolateral cortex, whereas lesions of ventrolateral cortex produce the greater deficit on object alternation. These results suggest that there is a focus for spatial tasks in the dorsolateral and one for the visual (and perhaps other exteroceptive tasks) in the ventrolateral frontal cortex.

These modalities are, however, superimposed on a temporal factor that is common to all delay tasks and is disrupted most severely not by lesions of the lateral frontal cortex but by limbic lesions (the go/no-go alternation evidence). If sensory modality is a factor in parcellation of the lateral frontal cortex, variations of the indirect variant of delayed response using matching from sample should prove even more useful than variants of the alternation tasks. In addition, discrimination reversal problems can be used to the same end. The next section examines such evidence.

Parcellation and Discrimination Problems

Mishkin (1964) initiated the attempts to parcel the total lateral frontal lesions, using the matching problems. He began by contrasting the effects of dorsolateral lesions with those produced by resecting a combination of ventrolateral, medial, and posterior orbital cortex. He reported that these orbitoventral

lesions produced an even greater deficit than dorsolateral lesions on all of the nonspatial tasks he and his colleagues (Brush, Mishkin, & Rosvold, 1961; Mishkin, Prockop, & Rosvold, 1962) had previously found difficult for monkeys with total lateral lesions. He suggested at the time that the hypothesis of perseverative interference to account for the animals' difficulties might be most appropriate to those with orbitoventral damage.

Since that report, the impairment in object discrimination reversal learning after orbitoventral lesions has been replicated (Butter, 1969; Butters, Butter, Rosen, & Stein, 1973; Goldman, Rosvold, & Mishkin, 1970). The impairment in nonreversal shifts between different types of discrimination problems (object and position) reported by Settlage and his colleagues (Settlage, Butler, & Odoi, 1956; Settlage, Zable, & Harlow, 1948) was replicated in animals with orbitoventral lesions, and also with lesions limited to the ventrolateral surface (Passingham, 1972a). Animals with dorsolateral lesions excluding the ventrolateral surface did not show a deficit either on object discrimination reversal learning (Butter, 1969; Goldman et al., 1970) or on reversal shifts between color, position, and size in visual discrimination problems (Passingham, 1972b). Further, although monkeys with dorsolateral lesions were at a higher performance level than monkeys with ventrolateral lesions on a successive visual-spatial discrimination problem, when a delay was added to change the problem into an indirect delayed-response task the performance of the dorsolateral group became much worse than that of the ventrolateral group (Oscar-Berman, 1975).

Place reversal is another spatial task in which significant impairments were found after total lateral surface anterior frontal lesions (Butter, 1969; Mishkin, 1964) or dorsolateral lesions (Pohl, 1973). Again, monkeys with total anterior frontal lesions performed more poorly than those with principalis lesions, whereas monkeys with damage to small nonprincipalis areas either dorsal to the principalis region or in the arcuate sulcus showed normal performance on this task (Goldman et al., 1971). By contrast, the ventrolateral lesions produced a severe deficit equal to that produced by the total lateral lesion (Butter, 1969). This finding is in accord with that on object-reversal learning as noted above. Neither monkeys with dorsolateral lesions (Pohl, 1973) nor monkeys with total lateral lesions (Butter, 1969; Pribram, 1961a) show any performance impairments, but monkeys with the ventrolateral lesion show severe initial impairments (Butter, 1969; Iversen & Mishkin, 1970).

There has also been some attempt to use discrimination problems to pin down a specifically auditory role for a subdivision of lateral frontal cortex. Gross and Weiskrantz (1962) found that animals with nonprincipalis lesions were significantly worse on an auditory go/no-go differentiation than principalis animals, in contrast to their superior performance on delayed response. However, stimulation of the arcuate and the principalis regions failed to confirm the speculation (Weiskrantz & Mishkin, 1958) that the arcuate sulcus was a focus for the anomalous auditory deficits; stimulation of either region did not produce a

drop in auditory differentiation performance (Weiskrantz, Mihailovic, & Gross, 1962). Furthermore, Gross (1963a) was unable to repeat his earlier finding of significant differences in auditory differentiation performance after nonprincipalis lesions when he redid the study using younger animals.

By contrast, more recently Petrides (this volume, Chapter 5) was able to demonstrate an auditory deficit on resection of the tissue dorsal to the arcuate sulcus. This result places the focus for auditory function proximal to the superior limb of the arcuate sulcus beyond the part of the frontal cortex ordinarily resected when lateral frontal lesions are made. Furthermore, arcuate lesions have been shown to affect performance differentially on the auditory conditional position task (Goldman & Rosvold, 1970; Stamm, 1973). Such lesions have no detrimental effects on performance of simultaneous visual discrimination tasks (Chow, 1952; Pribram et al., 1952; Pribram, 1954), successive visual discrimination (Pribram & Mishkin, 1955; Stamm, 1973), or place reversal (Goldman et al., 1971).

The auditory conditional position task was intended to test the animal's ability to make spatial associations when immediate memory is not taxed. As might be expected from what Petrides has shown, animals with dorsolateral lesions excluding principalis and the ventrolateral surface performed better than the arcuate animals (Goldman & Rosvold, 1970), as did animals with ventrolateral lesions excluding arcuate (Stamm, 1973), suggesting a possible arcuate focus.

It is not clear from these results alone whether the auditory or the spatial aspect of the task is most relevant, a consideration that also plagues the finding that monkeys with ventrolateral lesions are as poor as dorsolateral monkeys on an auditory delayed-response task, though they perform better than they do on a visual delayed-response task (Oscar-Berman, 1975). However, Stepien and Stamm (1970) tested a nonauditory spatial opposition task in which the monkey was required to go to a side of the maze opposite from the visual signal. They found that animals with total lateral lesions or lateral lesions excluding principalis were impaired, but animals with damage limited to any of the smaller segments of the dorsolateral surface, including the anterior banks of the limbs of the arcuate sulcus, were unimpaired. This finding suggests that it is the spatial rather than the auditory aspect or the confound between them that is responsible for the deficits following lesions of the anterior frontal cortex forward of the arcuate sulcus.

The evidence from discrimination testing of monkeys with partial lateral frontal lesions is thus not as clear-cut as one would like. Nonetheless, a focus for visual functions may tentatively be discerned in the anterior ventrolateral cortex and one for auditory functions in the dorsal periarcuate cortex (see also Milner, & Petrides, 1984, and Petrides, this volume, Chapter 5). Place reversal that taxes both a temporal and a spatial factor fails to distinguish between portions of the lateral frontal cortex—a finding consonant with that obtained with the classical right/left alternation which, as noted in the previous section, also tests both factors. What remains to be reviewed are the experiments that aim to determine

the biological nature of the spatial and temporal factors and their relationship to sensory input.

The Spatial Deficit Is Not Kinesthetic

On the basis of his work with cats and dogs, Konorski (1967) elaborated the hypothesis that the lateral frontal deficit was specifically important for kinesthetic memory and suggested that the cue in the delayed-response task is encoded into memory as the differential movements that are to be made at the end of the delay. Pribram (1961a) had earlier considered this hypothesis on the basis of results in which two types of operant schedules (fixed ratio and fixed intervals) were alternated without benefit of the visual signals used in training. When the signals were removed, the monkeys with lateral frontal lesions, in contrast to their controls, responded indiscriminately. But Pribram was forced to abandon the hypothesis after demonstrating that monkeys with total lateral lesions also had deficits on nonspatial alternation tasks that did not involve kinesthetic feedback.

However, as the data previously discussed accumulated, indicating that the nonspatial alternation deficit had a more central focus, Stamm began to reexamine the kinesthetic hypothesis by adding kinesthetic cues to the delayed alternation. He first trained monkeys on the delayed-alternation task in apparatuses that required different degrees of movement and hence allowed different amounts of kinesthetic feedback (Stamm, 1970). In support of Konorski, Stamm did find that the animals with dorsolateral frontal lesions performed best in the primate chair and worst in the maze, although preoperatively the opposite trend had been significant. Konorski's hypothesis could also account for the data indicating that stimulation across the principalis region at the beginning of the delay has no effect if the task is a delayed match-to-sample task (Kovner & Stamm, 1972) but produces a striking drop in performance if the task is a delayed successive visual discrimination task (Cohen, 1972). Two different visual cues must be differentiated on the basis of pattern, not position, for both of these tasks, but only in the second task does the visual cue signal a spatially distinct response. In an earlier study Mishkin and Pribram (1956) also found that monkeys with total lateral lesions failed the task when it was presented in a manner similar to Cohen's technique.

In another ablation study Stamm and Weber-Levine (1971) found that adding either conditional color or kinesthetic cues to the alley leading to the response doors greatly improved the performances of monkeys with total lateral or principalis lesions on delayed alternation tested in a maze. Stamm and Weber-Levine further found that the improvement produced by the addition of these conditional cues lasted through the subsequent testing of delayed alternation without additional cues for the monkeys with principalis, but not total lateral lesions. Similarly, Gentile and Stamm (1972) found that adding different weights to the manipulanda to provide additional cues improved the performance of

monkeys with principalis but not total lateral lesions during delayed alternation tested in the Wisconsin General Testing Apparatus (WGTA). The introduction of supplementary articular-somesthetic cues, on the other hand, improved delayed-alternation performance of both the monkeys with principalis lesions and those with total lateral lesions. Such improvement is in contrast to the lack of improvement for monkeys with total lateral lesions after the addition of supplementary visual (Gentile & Stamm, 1972; Tubbs, 1969) or auditory cues (Tubbs, 1969), or for monkeys with principalis lesions after the addition of supplementary visual cues (Gentile & Stamm, 1972; Stamm & Weber-Levine, 1971).

On the basis of these data, Stamm and Gentile concluded that they had evidence for the hypothesis that kinesthetic memory is relevant to the role of the frontal deficit in ordering spatial responses and that their data indicated that the use of kinesthetic memory was specific to the principalis area. Gentile (1972) then elaborated the kinesthetic concept, bringing it more in line with a suggestion by Pribram (1960) that the frontal cortex acts to partition sets of afferent activity. She suggested that, in the standard delayed-alternation tasks, movement-produced feedback from left and right responses cannot be distinguished and therefore cannot be coded with respect to recency. Unlike additional visual or auditory cues, the additional force or articular-somesthetic differences helped to partition the feedback cues. By this reasoning, temporal parsing (Pribram & Tubbs, 1967) would aid in differentiating the movement-produced cues because the trace of the kinesthetic cues would be different after responses to the one side followed by the longer delay.

However, there is an objection to localizing this kinesthetic deficit to the cortex surrounding the sulcus principalis. Pribram and Tubbs (1967) and Pribram et al. (1977) have argued that temporal parsing provides monkeys with anterior frontal lesions with a stimulus organization they could not provide for themselves since they lack their frontal cortex. Gentile and Stamm would similarly like to argue that, since providing kinesthetic cues improves the performances of monkeys with principalis lesions, the principalis must normally utilize kinesthetic cues. However, monkeys with total lateral lesions were unable to utilize the additional kinesthetic cues, so it is likely that it was the nonprincipalis frontal cortex that enabled the monkeys to learn to use these cues. Even the fact that additional articulosomesthetic cues allow monkeys with total lateral lesions to improve their performance means only that other cortical areas are able to utilize these particular cues, much as in humans verbal cues are used to overcome the deficit (Luria, Pribram, & Homskaya, 1964). In fact, Gentile's (1972) data showing that preoperative training that emphasizes kinesthetic cues makes it difficult for the animals to utilize additional articulosomesthetic cues after surgery suggest that some kind of adaptive cortical reorganization is going on.

More devastating to localizing the kinesthetic deficit in the region of the sulcus principalis, however, is a deficit on object-reversal performances when the reversals happened after only 30 trials. Gross (1963b) used animals with

lesions confined to the principalis to obtain these effects. Monkeys with nonprincipalis lesions showed normal performance. Thus, even when the cues are not spatial or kinesthetic, the principalis region itself is essential for encoding them into short-term memory (see also Stamm, Chapter 4, and Petrides, Chapter 5, this volume).

In summary, the kinesthetic hypothesis as the basis for the spatial aspect of the frontal deficit, though it has found substantial support, fails to account for all of the data. Certain discrepancies plague the evidence, which makes the kinesthetic-spatial connection difficult to accept uncritically. The spatial deficit is conceived to be primary in causing difficulty in delayed-response performance. Principalis lesions are the focus for this difficulty, but, as I have argued above, not for the kinesthetic deficit. Further, kinesthesis is essentially a response-produced stimulus and the early experiments of Mishkin and Pribram (1955, 1956) reviewed above had shown that cue differentiation rather than response distinction was the critical variable in determining the delayed-response deficit. Interestingly, Stamm, who has provided the most persuasive evidence in favor of the kinesthetic (which is essentially a response distinction) hypothesis, argues most strongly against another response-based hypothesis, namely, the suggestion that failures in response inhibition are responsible for the frontal deficit. It is likely, therefore, that the kinesthetic hypothesis relates more to the temporal than to the spatial deficit that follows anterior frontal lesions. We thus turn next to a review of the evidence that has been gathered with this hypothesis in mind.

The Temporal Deficit and Kinesthetic Stimulus Differentiation

The initial argument that failure in response inhibition accounts for the frontal deficit came from the observation of an impairment on visual and auditory go/no-go discrimination tasks that follow orbitoventral lesions (Brutkowsy, Mishkin, & Rosvold, 1963; Lawicka, Mishkin, & Rosvold, 1966). In these tasks the errors of the operated monkeys, unlike those of the normals, seemed to occur mainly on no-go trials (Iversen & Mishkin, 1970). Then, in order to further characterize the nature of the deficit due to ventrolateral damage, Iversen and Mishkin (1970) tested monkeys with specific medial and posterior orbital lesions and with ventrolateral lesions on auditory and visual go/no-go differentiation as well as on an object-reversal series. The medial and posterior orbital group made more go/no-go errors than the normals on the successive visual discrimination task and demonstrated a significant nonspecific difficulty on the object reversals that lasted throughout the series. The ventrolateral group, on the other hand, showed perseverative no-go errors on both the auditory and visual tasks, an extremely poor original performance on the auditory task, and a large number of perseverative errors confined to the first reversal of the object-reversal series. Thus, the ventrolateral animals showed perseverative interference that was transient in nature.

This transience of the perseverative effect had also been noted after total lateral lesions in nonreversal shifts (Settlage et al., 1956), go/no-go differentiation (Battig, Rosvold, & Mishkin, 1962), and delayed alternation and successive visual discriminations (Stamm, 1970; Stamm & Weber-Levine, 1971). The transience of the effect might also explain why sophisticated monkeys with lateral lesions did not perseverate their initial preferences (Oscar & Wilson, 1966) when trained in the same learning set paradigm in which more naive monkeys with lateral lesions did perseverate their initial preferences (Brush et al., 1961).

Iversen and Mishkin (1970) concluded that there was a separable transient perseveratory factor that could be attributed to the ventrolateral region. However, Stamm (1973) later found that animals with lesions confined to the ventrolateral surface excluding the arcuate sulcus were impaired on successive pattern discrimination tasks but not on the auditory conditional position task or on the spatial opposition task (Stepien & Stamm, 1970). Stamm argued that since both tasks involve spatial differentiation between instrumental responses, a perseverative interference theory ought to have predicted a deficit on both tasks, which did not occur. In agreement with Stamm, the previously discussed data make it appear more likely that any perseverative interference factor has more to do with spatial differentiation between responses. But it is not the spatial factor that is involved by the ventrolateral lesion. More likely, as noted above, the ventral lip of the frontal lobe is the focus of the temporal aspect of the frontal deficit, but relatively extensive damage surrounding the lip is required to produce the full-blown behavioral effect.

Passingham and Ettlinger (1972) presented evidence that the tactile deficit previously seen in monkeys with lateral lesions (Ettlinger, Morton, & Moffet, 1966; Ettlinger & Wegner, 1958) was specific to the orbitoventral region and that the impairment could be alleviated by adding weights to the manipulanda to make the responses more effortful. Under these conditions the orbitoventral monkeys made as many stimulus comparisons as did the controls and considerably more than they had made during the no-effort condition, suggesting that their difficulty had indeed been due to a lack of response inhibition.

However, Passingham (1972a) was not able to demonstrate that animals with orbitoventral lesions responded incorrectly more than the control animals to a panel that had been deliberately given a higher probability of containing the correct visual stimulus. Passingham had reasoned that the animals would have developed a response set to the higher probability panel and would have to inhibit that set to respond to the other panel correctly. But, despite the fact that orbitoventral animals had previously demonstrated a significant impairment on a simultaneous visual object discrimination, they distributed their errors in the differential probability problem in the same manner as did the normals. The results of this study confirm those of two experiments (Gruenninger & Pribram, 1969; Wilson, 1962) performed in Pribram's laboratory on monkeys with total lateral frontal lesions.

But Stamm is correct in questioning the response inhibition hypothesis as adequate in accounting for the temporal aspect of the anterior frontal deficit. Recall that, in discussing the drive inhibition hypothesis forwarded by Brutkowski (1964) to explain the posterior and medial orbital deficit, we noted that errors of omission were proportionately as frequent as errors of commission in the go/no-go alternation task (Butter, 1964; Pribram et al., 1966). Errors of omission—especially failures to respond on the go trials—provide a strong argument against a response inhibition hypothesis. In addition, it should be pointed out that occasional monkeys with either posterior and medial orbital lesions or lateral frontal lesions are reluctant to be tested after surgery despite prior experience. Thus, their overall experience, which has trained them primarily to "go," appears to be negated by a tendency to "no-go." Sometimes these monkeys appear so confused and so reluctant that extensive gradual reshaping must be undertaken before they can be tested. Of course, such shaping trials do not appear in the quantitative descriptions of test performance, which can therefore be misleading when hypotheses as to basic process are being derived.

Stamm's evidence suggests that, rather than response inhibition, the temporal factor in the lateral frontal deficit is related to kinesthetic stimulus differentiation, perhaps on the basis of a central representation of kinesthetic events. Such kinesthetic stimuli are produced by a convergence of muscle afferents with others, such as those from the skin, to produce a motor representation (Malis, Pribram, & Kruger, 1953; Pribram, 1971; Pribram, Sherafat, & Beekman, 1984). The anatomical adjacency of the entire anterior frontal cortex to the classic precentral and to the limbic mediobasal motor cortices makes the hypothesis a reasonable one (see also Petrides, this volume, Chapter 5).

As noted above, the hypothesis received attention in earlier experiments. For instance, Pribram et al. (1952), in an unpublished portion of their study, attempted to show that the delayed-response performance of normal monkeys is dependent on self-generated kinesthetic cues. It was found that movements specific to the correct solution of the delay problem did occur initially but that, as the monkeys became proficient, these peripheral indicators became less and less frequent. The assumption was made that the proficient monkeys used a central representation to solve the problem—that brain events replaced the peripheral kinesthetic stimuli. It was anticipated that, perhaps after anterior frontal surgery, the monkeys might revert to a peripheral response mode (as was shown for the chimpanzee by R. A. Blum, J. Semmes, and K. H. Pribram, presented at the annual meeting of the APA in 1947), but this did not occur—thus, the data were left unpublished. Further, as noted above, other experimental results (e.g., the deficit in object alternation) mitigated against the kinesthetic hypothesis until Stamm noted that these results could be ascribed to a ventral focus in the lateral frontal cortex. This left the possibility that some other focus could be found responsible for the kinesthetic deficit. Stamm has suggested that this focus is the cortex surrounding the sulcus principalis but, as reviewed above, this localiza-

tion does not hold up. Instead, Petrides's (this volume, Chapter 5) evidence leads to a more ventral and posterior periarcuate focus, which, of necessity, must be separable from the visual focus (more anteriorly situated around the anterior lip of the arcuate sulcus extending to the lip of the lobe) that produces the object alternation deficit if the kinesthetic hypothesis is to be supported.

To summarize this section, the results of attempts to subdivide the lateral frontal cortex lead to the following conclusions:

1. There is a focus centering on the sulcus principalis that influences performance on both the spatial delayed-response and the spatial delayed-alternation tasks but *not* on the go/no-go or object versions of alternation, suggesting that a spatial factor important to task performance has been interfered with by the lesion of this cortex. Further, the presumed kinesthetic basis for the spatial deficit common to the impaired performances proves to be related to the temporal and not the spatial aspects of these and other tasks. This leaves the spatial deficit unexplained. An explanation of the spatial deficit in terms of the effects of spatial distractors was suggested and will be enlarged on in the next section.

2. The remainder of the lateral frontal cortex influences all types of alternation performance and can be further subdivided according to modality by tests involving variants of alternation (e.g., object alternation, discrimination reversal). Dorsal periarcuate auditory, perhaps anterior periarcuate visual, and posterior periarcuate kinesthetic subdivisions can be identified. The deficit produced by lesions in these subdivisions is sensitive to the *sensory load* imposed as a requirement for performing adequately. This suggests that some sort of sensory servocontrol (negative feedback) mechanism is involved. Goldman-Rakic (1978; Goldman-Rakic & Schwartz, 1982) has elegantly worked out the connections between frontal and parietal cortex and these with the corpus striatum, connections that can serve such a sensory servosystem.

SUMMARY AND SYNTHESIS

When I began research on the functions of the anterior frontal cortex I found that neurobehavioral considerations related this part of the brain to the functions of the limbic portions of the forebrain, not to the motor functions of the precentral cortex. The peri-Rolandic cortex, on the basis of neurobehavioral analysis, belonged with the remainder of the cerebral convexity. Thus, a major distinction was made between the functions in behavior of the frontolimbic formations and those of the posterior cerebral convexity (see reviews by Pribram, 1954, 1958a, 1958b, and the initial part of this chapter).

Nonetheless, the proximity of the anterior frontal cortex to those portions of the cortex that were electrically excitable in terms of motor functions (including

those on the medial and basal surfaces of the hemisphere) continued to be of considerable concern. Only recently have I hit upon an idea around which this concern can be precisely formulated. It is this formulation that forms the core of the final portion of this review.

The idea is simple. There is an important attribute by which the systems in the central portion of the cerebral mantle differ from others: They are concerned with somatosensorimotor processes. Somatic processing differs from all other processing in that whatever is experienced, whether through the epicritic systems of the posterior convexity or the protocritic (interoceptive plus pain and temperature—see Chin, Pribram, Drake, & Green, 1976, Pribram, 1977, for data and definition) systems of the frontolimbic formations, no precise communication with other organisms or the physical or cultural environment is possible without the participation of somatosensorimotor mechanisms.

For the anterior frontal cortex, this means that we should be able to discern in its functions a mechanism that relates protocritic processing to somatosensorimotor functions. As with any such endeavor based on an arbitrary dichotomous classification, problems immediately arise: The exteroceptors are part of the body, and those processes that are concerned not so much with regulating their specific function but with controlling their overall "somatic" expressive relationship to the world need, on the basis of the evidence, also to be included in the somatosensorimotor mechanism.

A good place to begin the attempt to tie all this together is Brown's (1985) review of frontal lobe syndromes, which is organized within the frame of three major groups of disorders: "Damage to frontal limbic formations leads to impaired activation (response bias, motor neglect, and lack of initiation); damage to 'integration' cortex on the convexity leads to derailment of the action after adequate initiation (distractibility, confabulation); and damage to premotor and precentral cortices leads to a defect of final implementation (misarticulation, dyspraxia)" (p. 37).

Pribram and McGuinness (1975) have further delineated the evidence for "stop" and "go" systems within the frontolimbic forebrain. The "stop" mechanism deals with emotion (to be "hung up," out of motion) and involves those portions of the frontal and temporal lobes connected to the amygdala by way of the uncinate fasciculus. The other mechanism deals with motivation and is constituted of the "go" dopaminergic nigrostriatal-frontal system (see also Goldman-Rakic & Schwartz, 1982).

With regard to Brown's second category, the data reviewed in the section on the subdivision of the anterior frontal ("integration") cortex show that distractibility is responsible for the "spatial" deficit obtained when the dorsolateral frontal cortex is damaged. Confabulation, on the other hand, may well be the human counterpart of the "temporal" deficit that follows periarcuate and ventrolateral frontal damage, a deficit sensitive to sensory input.

Brown's third category, centering on the premotor and precentral cortex,

holds the key to bringing together the various aspects of frontal lobe function. The key is provided by the proposals made by Goldberg (1985; also this volume, Chapter 15) regarding the functions of the premotor systems, which, in turn, are based on the concepts of Sanides (1966, which are also reviewed and extended by Pandya and Barnes, this volume, Chapter 3). These proposals divide the premotor cortex into a medial, supplementary premotor region and a lateral, periarcuate premotor region. On the basis of evidence from comparative anatomical studies, the medial region is shown to be derived from archicortical origins, and the lateral region from paleocortical primordia. The two regions are suggested to function differently: The medial is concerned in developing models that program behavior in a feedforward fashion; by contrast, the lateral region programs behavior via a variety of sensory feedback mechanisms.

This analysis can be readily extended to the remainder of the motor cortex: The evidence regarding the difference in orientation of the projection fan of thalamocortical connections, presented in the first part of this chapter, indicates that the primary somatosensorimotor cortex also derives from the medial surface of the hemisphere, perhaps from the cortex of the cingulate gyrus. Accordingly, it would seem that the supplementary motor cortex participates in sketching the outlines of the model while the precentral cortex implements its finer aspects. Such a scheme is supported by the fact that the supplementary motor cortex receives an input from basal ganglia (known to determine postural and sensory sets) while the precentral motor cortex, in its involvement with the cerebellum, provides the details necessary to carry out a feedforward regulated action. I have elsewhere (Pribram et al., 1984) provided a review of the evidence and a mathematical description based on a model developed by Houk & Rymer (1981), by which such a feedforward process operates.

The lateral premotor region is intimately interconnected with the inferior-posterior parietal cortex as indicated by Schwartz and Goldman-Rakic (1984), by Goldberg (1985), and by the thalamocortical and comparative anatomical data reviewed at the beginning of this chapter. As indicated there, it is damage to this system that produces apraxias, which, according to Goldberg's thesis, should devolve on faulty feedback processing. It is not too farfetched to wonder whether the repetitions which the lesioned monkeys made in the task reported in the first part of this chapter might not have been due to the necessity for gaining additional sensory feedback before proceeding.

There is one further speculation regarding apraxia that is worth considering. Elsewhere (Pribram & Carlton, 1987) I have described the neural mechanism involved in the construction of objects from images. Essentially this mechanism operates to extract invariances (constancies) from sets of images by a process of convolution and correlation. An object is experienced when the resultant correlation remains constant across further transformation of the set of images.

When objects are constructed in the somatosensorimotor domain they are of two kinds. One sort of object is the familiar external "objective" object. Damage

to the peri-Rolandic cortex (including the superior parietal gyrus) results in object agnosia. When, however, the lateral premotor and inferior parietal cortex is damaged, apraxias and neglect syndromes develop. Could the apraxias be thought of as a mild form of neglect in the sense that the "object" that is constructed by this premotor-parietal system is the "self"? If this hypothesis is correct, apraxias result from a failure in the appreciation (based on feedback?) of self: an awkwardness more pervasive than the impairment of skills. Thus, one can envision a gradual increased impairment ranging from apraxia through Parkinsonian tremors at rest, etc., to neglect. This syndrome can be clearly distinguished from the one produced by cerebellar-Rolandic damage, which is characterized by loss of skill, intention tremor, and paresis.

A word of caution: The statements made above could be interpreted as a denial of distinctions between such syndromes as Parkinson's, neglect, and apraxia. This is definitely *not* what is meant. Even apraxias of frontal origin can be expected to differ subtly from those of parietal origin and it may well be as Brown (1975) suggests—that the lesions which produce apraxia must invade the limbic forebrain. As evident in the work of Terrence W. Deacon (personal communication), parietal and frontal cortex, though reciprocally connected, show an upstream/downstream relationship to one another. According to Deacon, a downstream corticocortical connection terminates most heavily in Layers iiic–iv; an upstream connection terminates in Layer i and sometimes in bands in vb. Thus, there is a clear hierarchical connectivity from anterior cingulate to anterior frontal to periarcuate to premotor and motor cortices. At the same time, parietal cortex is upstream from posterior cingulate, as well as from all of frontal cortex.

What I *am* trying to convey is that a *class* of disorders due to damage to systems of paleocerebral origin can be discerned. Within that class a variety of syndromes traceable to differences in neuroanatomical and neurochemical substrates can be made out.

How does this approach to the problem help connect the functions of the anterior frontal cortex to those of the somatosensorimotor regions? As noted in this chapter, delay problem performance is related to sensory mode: a periarcuate locus for auditory and visual, a more anterior location for kinesthetic. These relationships fit with the general hypothesis that the function of the anterior frontal cortex is to relate the processes served by the limbic forebrain to those of the sensorimotor systems, broadly defined as above. The results also support the suggestion that these relationships are of a feedback nature, namely, Stamm's experiments in which kinesthetic feedback was manipulated.

Furthermore, there are the strong connections through the uncinate fasciculus to the structures of the temporal lobe derived from paleocerebral systems (amygdala, pyriform cortex, and adjacent temporal polar juxtallocortex), which indicate that these portions of the anterior frontal cortex are to be considered relatives of the lateral premotor system rather than relatives of the precentral motor system.

On the other hand, there are heavy connections between the cortex surrounding the sulcus principalis and the hippocampus (Nauta, 1964). It is this part of the anterior frontal cortex that has resisted fractionation with respect to sensory mode, but which is especially sensitive to the "spatial" aspects of the delay task. This is exactly the situation with regard to hippocampal function. In fact, the deficits produced by resections of the primate hippocampus and those produced by resections of the cortex surrounding the sulcus principalis mimic (with the critical exception that spatial delayed response remains intact after hippocampectomy) each other to such an extent that it is hard to distinguish between them.

I have extensively reviewed above and elsewhere (Pribram, 1986) the evidence for considering the difficulty with "spatial" problems as being due to an increase in sensitivity to distraction under certain specifiable conditions. Briefly, the essential evidence is that, when such interference is minimized, as when the delay interval is darkened, monkeys with frontal resections can perform the delay task (Anderson et al., 1976; Malmo, 1942). Further, spatial cues have been found to be more distracting than visual and auditory cues for normal monkeys, and especially so for monkeys with resections of the anterior frontal cortex and, to a somewhat lesser extent (thus the sparing of delayed response?) of the hippocampal cortex (Douglas & Pribram, 1969; Grueninger & Pribram, 1969). Whatever the interpretation of the "spatial" deficit, the data are consonant with the conclusion that the cortex surrounding the sulcus principalis is derived from an archicerebral primordium.

The profusion of data collected by hard labor over the past 50 years can thus be fitted into a tentative scheme. No longer are we stuck with vague concepts of frontal lobe function. The role of the anterior frontal cortex in emotion and motivation is seen as relating protocritic (interoceptive plus pain and temperature) to epicritic processes in the feedback mode. Evaluation (what Arnold [1970] calls appraisal) of one's feelings with regard to what one wishes to do is the function of the periarcuate and ventrolateral portions of this cortex (Konow & Pribram, 1970). Evaluation is a sort of internal rehearsal, a feedback by way of which the feeling becomes refined, that is, more in keeping with current sensory input and with the consequences of actions.

The role of the anterior frontal cortex in attention and intention (planning) relates protocritic to epicritic processing in the feedforward mode. This is the function of the dorsolateral frontal cortex. In the feedforward mode, current and consequent inputs form the context within which "models" are constructed in "fast time," models which in turn are used to modify subsequent behavior. Thus the role of the frontal cortex in one form of "short-term memory" is clarified. The close connection between the dorsolateral frontal cortex and the hippocampus; the similarity of the cytoarchitecture of the hippocampus and that of the cerebellum; the close connection of the peri-Rolandic cortex (which is most likely derived, as noted, from the archicerebrum, as is the hippocampus) and the cerebellum; and the known function of the cerebellum as a feedforward mechanism, etc.

e.g., Pribram, 1971, 1981; Ruch, 1951) all attest to the likelihood that the dorsolateral frontal cortex is indeed involved in such "projective" processes.

One final word: Brown (this volume, Chapter 14) has suggested that the mechanism for feedback and feedforward depends on the operation of sets of tuned relaxation oscillators that constitute the brainstem and spinal cord systems, which are influenced by the various frontal lobe processes under consideration. The evidence for the existence of such tuned oscillators has been repeatedly presented from the time of Graham-Brown (1914) through von Holst (1937, 1948) and Bernstein (1967) and his group (Gelfand, Gurfinkel, Tsetlin, & Shik, 1971). This evidence has been thoroughly reviewed by Gallistel (1980). The mechanism whereby a cortical influence can be imposed on such systems of oscillators has also been worked out within the concept of an "image of achievement." Such a motor image must operate within the spectral frequency domain (Pribram, 1971), and Pribram et al. (1984) have presented evidence that neurons in the motor cortex are tuned to different frequencies of movement (independent of velocity and acceleration). These authors also detail the mechanism whereby such tuned cortical cells can program the subcortical motor systems.

Is the task then completed? Heavens, no! We have as yet only begun to explore *how* the various portions of the frontal cortex do their work. This is especially true of the anterior frontal cortex, the part of the lobe that was so cavalierly severed from the rest of the brain during the heyday of the leukotomy (lobotomy) procedure. Nonetheless, as this review and the contents of the other chapters of this volume indicate, a half-century of investigation has not been in vain, and the promise of the future is that we will, in due time, also get to know the *how*.

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Indeed, our understanding of the frontal lobes increased considerably between 1973 and 1986, and the present volume attests to that. As Karl Pribram concludes from his "revisitation" of the frontal lobes in Chapter 2 of this volume: "The profusion of data collected by hard labor over the past 50 years can . . . [now] be fitted into a tentative scheme. No longer are we stuck with vague concepts of frontal lobe function." Yet, in the tradition of scientific progress, Pribram interprets this achievement as a new challenge reminds us that "we have as yet only begun to explore *how* the various portions of the frontal cortex do their work" and proposes that it is the *how* to which we now turn.

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