EXECUTIVE FUNCTIONS OF THE FRONTAL LOBES

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INTRODUCTION

The subject of this symposium concerns the mechanisms of transmission of signals necessary for conscious behavior to occur. Implicit in framing the question in this fashion is a view of the nervous system as a processor of input signals, signals which constitute information for the organism. This 'information processing' view of neuronal mechanisms finds considerable support in the current experimental and theoretical literature on brain function.

My purpose here is to emphasize a complementary view of the brain mechanisms coordinate with consciousness. My view stresses the fact that, while signal transmission does, of course, occur, the essential mechanism involved in the production of awareness is the pattern of local graded potential changes, the depolarizations and hyperpolarizations which occur at synaptic junctions and in dendritic networks.

Further, I want to present evidence that what an organism becomes aware of is related as much to the internal activities of its brain as it is due to the external situation that ordinarily provides the contents of awareness. This process by which an organism becomes conscious of selected aspects of a situation is usually called attention. Thus my presentation falls into two parts: one, a brief description of the brain state presumably coordinate with awareness; and two, a more detailed description of the attentional control processes that organize this state.

THE HOLOGRAPHIC HYPOTHESIS

At a recent meeting of the European Brian and Behavior Society Weiskrantz and Warrington (1974) presented the remarkable case history of a patient who had sustained an occipital lobe operation with its consequent hemianopia. However, Weiskrantz and Warrington were able to train this patient to respond accurately to the location of objects in the hemianopic

field and to discriminate among fairly complex patterns presented in the 'blind' portion of the field. The object placement was identified by the patient's pointing to it and the discriminations were performed by depressing an appropriate button. What is so remarkable about this case history is that the patient insisted that he was *un*aware of the stimuli to which he was responding, stating that he was only guessing on the basis of some vague feeling of what an appropriate response might be. Yet his performance was in the range of 86—90% correct.

These observations suggest that, in man at least, structured conscious awareness may be dependent on the integrity of his cerebral cortex, a view, by the way, which was practically universally held in neurological circles toward the end of the 19th century (see, for example, Pribram and Gill, 1976). This view does not, of course, deny that the more global determinants of conscious states are regulated by core brain structures, thalamus, mesencephalic reticular formation and the like. What the Weiskrantz and Warrington observations point to is that the experiencing of detail in awareness is a function of the integrity of the cortex.

The involvement of the cortex in the structuring of awareness is also suggested by the experiments of Libet (1966) who showed that electrical excitation of the postcentral cortex of man leads, after several seconds, to a state of awareness of the part of the body represented in the cortex being excited. I have elsewhere (Pribram, 1971) taken especial note of the fact that several seconds of excitation are necessary and that this suggests that some sort of brain state must become established before structured awareness can occur. The Weiskrantz and Warrington observations make it plausible that this state is in fact cortical.

What is the nature of this cortical state? In this presentation I want only to mention my hypothesis which has been detailed elsewhere (Pribram, 1966 and 1971; Pribram et al., 1974). This hypothesis suggests that at any moment, a state composed of the microstructure of local junctional and dendritic (pre- and postsynaptic) potentials is the neural mechanism coordinate with structured awareness. Bennett presents in this volume (Chapter 16) a detailed and excellent review of the composition of such slow potential states in receptor organs and Purpura (Chapter 10) has once again presented evidence (see also Purpura, 1958 and 1969; Purpura and Yahr, 1966) that similar processes occur at the cortex. Because a wave-mechanism description of the microstructure of such states is plausible (slow potentials are wave forms) and has proved fruitful (Pribram, 1975), I have suggested that the mathematics of optical information processing (i.e., of holography) be used to describe these states. The strong form of this hypothesis suggests that the input channel is, at its cortical termination, composed of narrowly tuned channels (i.e., is akin to a Fourier hologram) and this strong form of the hypothesis is being tested at both the neural and psychophysical levels for the visual mechanism in several laboratories (Pollen et al., 1971; Glezer et al.,

1973; Pollen and Taylor, 1974; Stromeyer and Klein, 1974, 1975a and b; Pollen and Ronner, 1975).

Given that the terminations of the input systems in the cerebral cortex are important to structured awareness, the question remains as to how the state of the cortex becomes structured. Obviously, the input per se is largely responsible. However, there are a series of experimental results which indicate that other processes, more central in origin, also play a crucial role. I will here review the evidence that relates the functions of the frontal cortex to these more centrally organized processes, although I could use the functions of the inferotemporal cortex (Pribram, 1974) or hippocampus (Pribram and McGuinness, 1975) just as readily. As noted earlier, the neural processes that organize the structure of awareness are usually subsumed under the rubric 'attention' and Horn, in his paper in this symposium (Chapter 13 and also Horn, 1970), has introduced the issues involved in his admirable presentation. His suggestions are compatible with those presented in a somewhat more comprehensive review that distinguishes three separate neural attentional systems (Pribram and McGuinness, 1975). Since these overall views of the issues are available, I prefer here to summarize in somewhat greater detail recent experiments on the primate frontal cortex as they relate to the problems of attention and awareness.

THE INPUT-OUTPUT RELATIONSHIPS OF THE FRONTAL CORTEX

The major themes of the research of the past decade have been (a) to discover the critical input-output relationships between frontal (eugranular) cortex and the rest of the brain; (b) to subdivide the frontal (eugranular) cortex into functional subunits; and (c) to reach some better understanding of the functions affected by frontal resections and stimulations.

The input-output relationships between frontal eugranular cortex and other brain structures have been assessed by making resections or stimulations in most other brain locations to see whether such manipulations influence the performance of delay tasks. Manipulations of most brain structures do not affect such performances (Pribram, 1954).

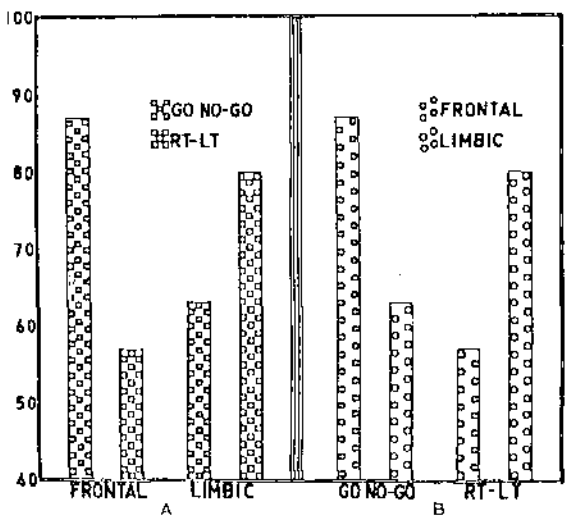
A major puzzle to investigators derives from the fact that the input to frontal cortex from subcortical structures derives almost exclusively from the nucleus medialis dorsalis of the thalamus, an intrinsic nucleus (i.e., one which derives its subcortical connections largely from other thalamic structures). Yet resections or stimulations of this thalamic nucleus do not, as a rule, disturb delay task performance (Chow, 1954; Peters et al., 1956). By contrast, when the limbic formations are invaded, e.g. the amygdala, hippocampus and cingulate cortex, the performance of some, though not all, delay tasks becomes markedly deficient (Pribram et al., 1952 and 1962; Pribram and Fulton, 1954). The only other brain structures consistently involved in influencing delay task performance are the head of the caudate nucleus and

elated parts of the globus pallidus and, in the thalamus, the centrum medinum. (See for instance early experiments by Rosvold: Rosvold and Delgado, 953; and by Pribram: Migler, 1958; reviewed and extended by Rosvold and zwarcbart, 1964; and Rosvold, 1972.)

These results suggest that the frontal eugranular cortex has special functional affinities with the limbic forebrain and with parts of the basal ganglia. his suggestion is supported by the finding that the head of the caudate nueus and the amygdala respond with extremely large electrical potential anges when the frontal eugranular cortex is stimulated (Pribram, unpubshed results) and anatomical techniques have shown major connections to ese structures (Kemp and Powell, 1970; Whitlock and Nauta, 1956).

Thus, the involvement of the frontal cortex in delay tasks is not a function f input to that cortex but of the complex relationships among the strucres of the frontolimbic forebrain and especially between these and the tput functions of the amygdala and the caudate nucleus of the basal nglia (Fig. 1).

Recent research has also emphasized the diversity of the functions of the ontal cortex anterior to the motor regions. Though generally related to lay tasks, the type of task influenced by limited resections differs deending on whether dorsal, ventral or orbital cortex is resected or stimulated ee, for example, early experiments by Blum, 1949 and 1952; by Mishkin, 957; and by Pribram et al., 1966a; and more recent studies by Passingham, 974; and by Oscar-Berman, 1975). In general, these studies suggest that atial delay tasks are affected by dorsal cortex manipulation; that visual



g. 1. Comparison of the effect of frontal and limbic lesions on A: go no-go and right-ft alterations; comparison on the basis of lesion locus, and B: comparison on the basis task (darkened circles represent the frontal group; open ones the limbic group).

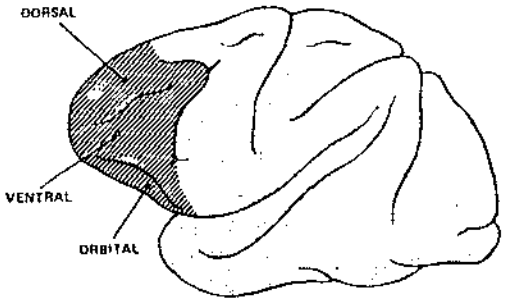


Fig. 2. Outline of monkey brain indicating dorsal, ventral and orbital frontal regions of the cortex.

delayed matching is related to the ventral frontal regions, and that successive, go/no-go tasks suffer most when orbitofrontal cortex is manipulated. What more general functions each of these subcategories of delay tasks represents is at present unclear and considerably more work is necessary to untangle the various variables that now confound interpretation of these nonetheless reliable results (Fig. 2).

Somewhat more headway has been made in understanding the functions represented by the general category of delay tasks. Such understanding may, or course, have to be revised when a clearer view is obtained of the meaning of the subcategories. But, at the same time, elucidating the meaning of the subcategories may well depend on first understanding the overall problem.

Delay tasks, by definition, represent short-term memory processes: the subject is asked to perform on the basis of cues not present at the time performance is sought but present some short interval (seconds to minutes) previously. But the locus of the disturbance produced in the short-term memory process by frontal lesion can be due to: (1) improper encoding of the cue, an attentional and/or intentional deficiency; (2) a rapid decay of an encoded trace, a consolidation impairment; or (3) confusion at the time of response, a retrieval deficit. Behavioral analysis has ruled out the trace-decay and retrieval deficit hypotheses (Pribram, 1961), and this conclusion has been amply substantiated by the results of electrical stimulation of the frontal eugranular cortex during the performance of delay tasks: the monkeys fail a trial when the stimulation to the frontal cortex occurs during the time of cue presentation and immediately (a few milliseconds) thereafter (see, for example, Stamm and Rosen, 1973), but not when such stimulations are made during the delay period per se or at the time when response is demanded. Thus, the role of the frontal cortex in short-term memory has so far been shown to involve attention and encoding appropriate to the intended behavior, not trace decay or retrieval per se.

THE FRONTAL CORTEX, ATTENTION AND INTENTION

There is a good deal of additional evidence that *attention* to input (arousal) and *intention*, readiness or set to respond (activation), are both regulated by the frontolimbic formations of the forebrain. This evidence is the subject of the recent review mentioned above (Pribram and McGuinness, 1975) which identifies three separate but interacting frontolimbic systems. One system centers on the amygdala and deals with phasic *arousal* of the organism to a novel, surprising input. A second system centers on the head of the caudate nucleus and related basal ganglia and tonically *activates* the brain, readying the organism for intended behavior. The third system centers on the hippocampus and coordinates arousal and activation, making it possible to maintain behavior in the face of distraction or to shift from one state of readiness to another without undue disruption (Fig. 3).

It is tempting to relate the three frontal subsystems to the three frontolimbic mechanisms. The hypotheses might, therefore, be fruitfully entertained that the orbital cortex is primarily related to the amygdala arousal system; the dorsal frontal cortex to the caudate readiness system; and the central frontal cortex to the hippocampal coordinating mechanism. The anatomical connections and physiological results obtained from stimulating these frontal subdivisions make the orbital and dorsolateral parts of the proposal plausible (Pribram et al., 1950; Kaada et al., 1949; Pribram and McLean,

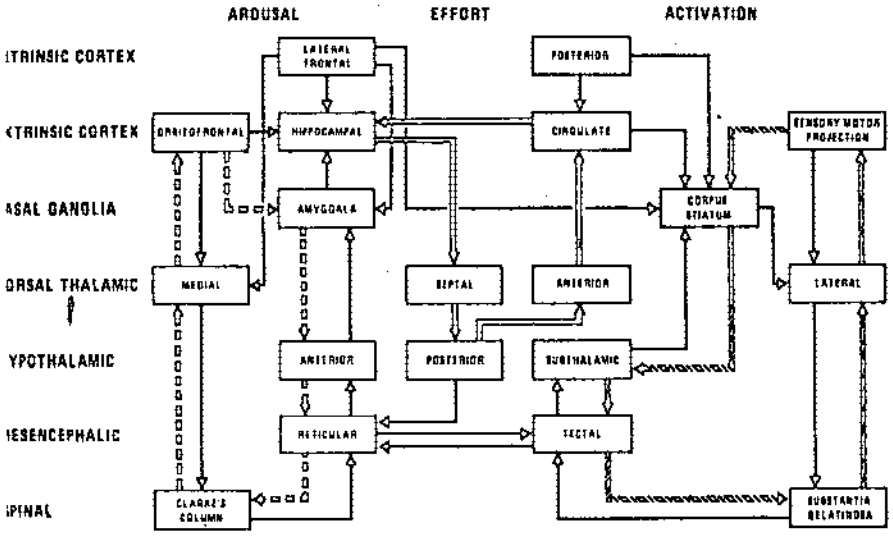


Fig. 3. Highly oversimplified diagram of the connections involved in the arousal (amygdala), activation (basal ganglia), and effort (hippocampal) circuits.

1953; Nauta, 1964). Behavioral results obtained from resections of the dorsal and the orbital areas also support the hypotheses (Rosvold, 1972; Pribram et al., 1966a). With regard to the ventral frontal cortex, however, the effects on delayed matching from sample need to be tested with hippocampal resections. Other evidence (i.e., the fact that spatial delayed response remains unaffected by hippocampal lesions: Mishkin and Pribram, 1954) suggests that this correlation may not, in fact, occur. It is more likely that the known anatomical connections between the hippocampal system and the medial frontal and cingulate cortex (Pribram and Fulton, 1954) will be the substrate of the arousal-activation coordinating system and that the ventral frontal cortex has yet another function related to the temporal isocortex with which this part of the frontal lobe is heavily connected (Mettler, 1935; von Bonin and Bailey, 1947; Jones, 1974). The temporal isocortex deals with selective attention (Rothblat and Pribram, 1972; Pribram et al., 1975) via connections to the putamen, the remaining basal ganglion of the corpus striatum (Reitz and Pribram, 1969; Buerger et al., 1974). In short, the functions of the ventral frontal cortex remain in doubt: they may relate to the hippocampal circuit, but are more likely to tie into a temporal lobe isocortex-putamen system which raises the unanswered question of the possible circuitry involved.

In recent years a few new facts have confirmed earlier findings and extended them. The new data concern two related domains: (1) the problem of orienting reactions to novel stimuli and therefore the organism's distractibility; and (2) the importance of the frontal eugranular cortex in organizing sets or contexts that regulate the organism's behavior. Again, these domains can be conceptualized in terms of attention and intention, respectively.

As noted earlier, frontal lobe resections interfere dramatically with the autonomic nervous system components of the orienting reaction. This effect of the lesion is coupled to an increased behavioral response to novelty: a failure to habituate to repetitions of a novel stimulus in both man and monkey (Luria et al., 1964; Pribram, 1973; Grueninger and Grueninger, 1973). The failure to habituate to an orienting stimulus is reflected in increased distractibility, which in monkeys is especially evident when spatial distractors, i.e. changes in the placement of cues, are involved (Grueninger and Pribram, 1969). This finding suggests that, contrary to the more common interpretation, frontal resections influence the response to spatial cues by *dis*inhibition: the common view is that dorsolateral frontal lesioned monkeys can no longer respond to spatial input; the more recent data suggest that the spatial input is responded to, but a failure in processing (ordinarily evidenced by habituation) is responsible for the observed deficit in behavior. For example, in a recently completed experiment (Brody, 1975) monkeys were taught to press a panel *next to* another that was marked by being lighted green. Both normal and frontally lesioned monkeys learned to do this readily until the marked panel was shifted among 16 placements from trial to trial. Now

only the normal monkeys were able to perform the task, the frontal lobe-lesioned animals failing completely.

Taken together with the finding that interruption of the efferent connections of the frontal cortex are responsible for the lesion effects, the question is raised as to how the efferents work. Electrophysiological experiments by Lindsley and his students (especially Skinner) and Clemente and his group have traced inhibitory pathways in cat and monkey from frontal cortex, through midline diencephalic pathways to the mesencephalic reticular formation (Skinner and Lindsley, 1973; Sauerland and Clemente, 1973). The relationship needs now to be investigated between these pathways and the efferent connections from frontal cortex to the basal ganglia, spelled out via anatomical and behavioral techniques reviewed above. As noted, we were able to distinguish a separate neural system that deals with orienting (an arousal system centering on the amygdala) which includes these frontodiencephalic-reticular inhibitory pathways (Pribram and McGuinness, 1975). Cutting the pathways or resecting the cortex of their origin ought to be *dis*-inhibiting and the behavioral result using spatial distractors is therefore in consonance with the electrophysiological data.

According to this view, then, the distractibility due to frontal lesions is due to disinhibition of the ordinary control exercised by the frontal cortex.

In another set of experiments we tried to place the effects of frontal lesions in a somewhat more general framework. The delayed response test is similar in many respects to a task used to trace the development of intelligence in the infant by Piaget. In fact, delayed response was invented by Hunter at the University of Chicago shortly after World War I in order to determine whether children and animals could hold ideas in mind. In Piaget's work the task is called an 'object constancy' problem (Piaget, 1954; Table I).

TABLE I

Stages in the development of the object concept

Stage	Time	Description
Stages 1 and 2	0—4 months	sucking reflexes; transient images primary circular reactions
Stage 3	4—10 months	interrupted prehension; secondary circular reactions
Stage 4	10—12 months	coordination of secondary schemas; retrieval of hidden object
Stage 5	12—18 months	sequentional displacements
Stage 6	18—24 months	invisible displacements

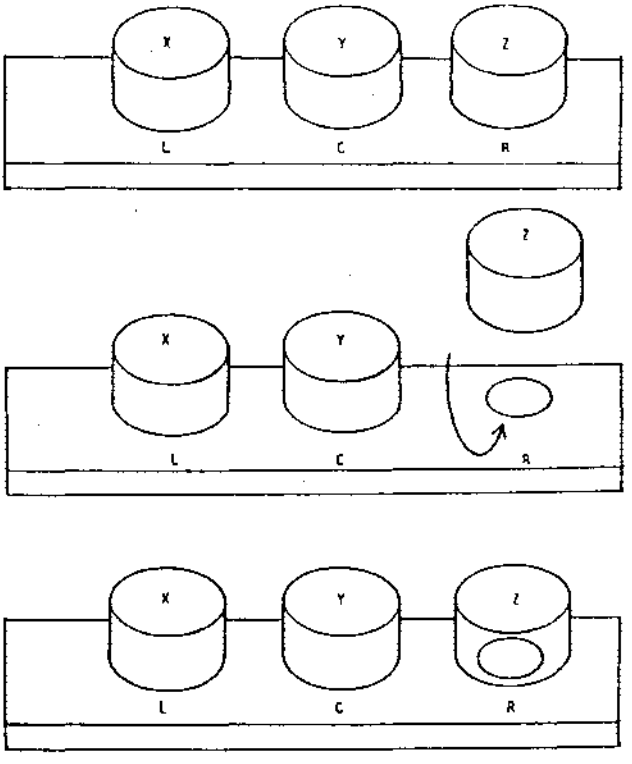


Fig. 4. Diagram showing delayed response type problem which illustrates Stage 4 of Piaget's object constancy paradigm.

In a just completed study (Anderson et al., 1976) we were able to show that frontally lesioned monkeys do in fact have difficulty when first faced with the object constancy task. In full view of the monkey a grape was hidden under one of three inverted baskets on a tray which was then pushed forward to allow the animal to lift the appropriate basket. This is a very rudimentary form of the delay task and I found many years ago that patients with ongoing pathology in frontal tissue (but not lobotomized patients) fail even this simple task (Figs. 4 and 6).

But we were not content with this result. On the basis of some of the findings reviewed above, the hypothesis had been constructed that much of the difficulty experienced by monkey and man after frontal resections was due to a failure to develop appropriate sets or contexts within which behavior could become arranged. The object constancy-delayed response task (really the old-fashioned shell game) was therefore complicated so that the baskets were moved about (without lifting them) after the placement of the grape, all within view of the monkey. The baskets were conceived as the

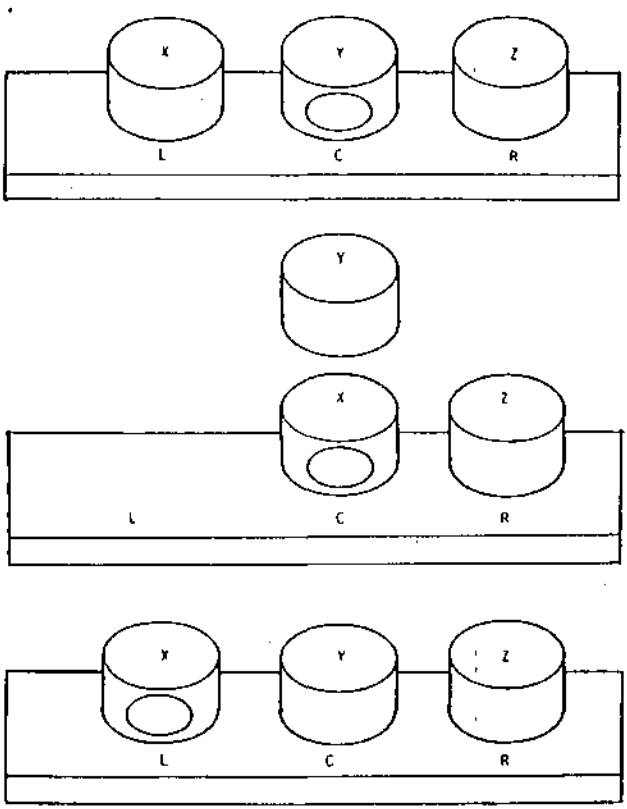


Fig. 5. Diagram showing delayed response type problem which illustrates context-dependency paradigm described in text.

context within which the grape was hidden. Whereas the object constancy problems (there was a series of them) were finally mastered by the frontally lesioned monkeys, albeit with a deficit, the context problems were *never* performed correctly, despite the fact that for normal monkeys these problems proved to be as easy as the object constancy versions (Figs. 5 and 7).

We initially interpreted these results as showing that two separate frontal lobe functions had been tapped by the experiment: one dealing with object constancy and the other with context processing. However, we learned that Bower had shown that the reason infants were defective in the object constancy situation was that they were distracted by the contextual cues within which the object became hidden (Bower and Wishart, 1972).

These results, therefore, again point to a disinhibiting role of frontal lesions which leave the organism more distractible. The results suggest additionally that distractibility interferes primarily with the establishment (perhaps by habituation) of a stable set or context within which novel stimuli

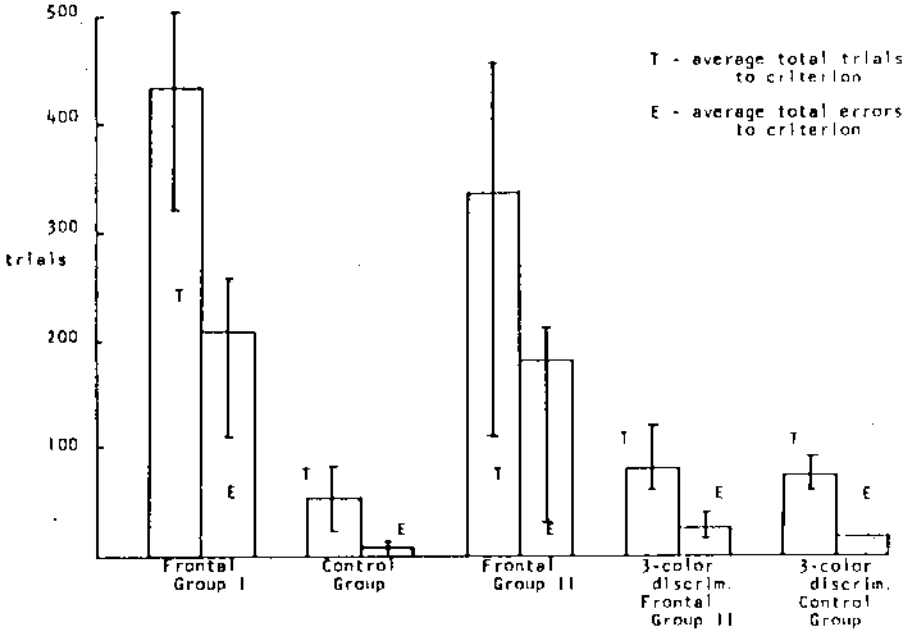
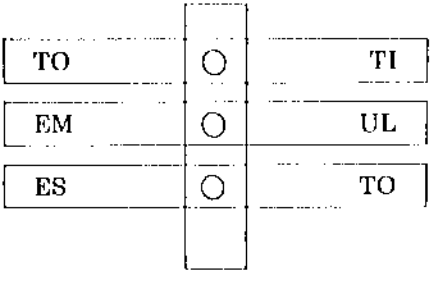


Fig. 6. Bar graph showing results obtained in the active search for vanished object problem (Piaget's Stage 4).

can be processed, so that behavior can become appropriate to the situation at hand.

A final experimental result bears on this interpretation regarding the relationship between frontal eugranular cortex and the importance of context in determining appropriate perceptions and behavior. Warren McCulloch used to enjoy startling his audiences with readings (accompanied by sonorous intonations) and picturizations of the Marzy Doates (Mares eat oats) type:

INMUDEELSARE
INCLAYNONEARE
INPINETARIS
INOAKONEIS



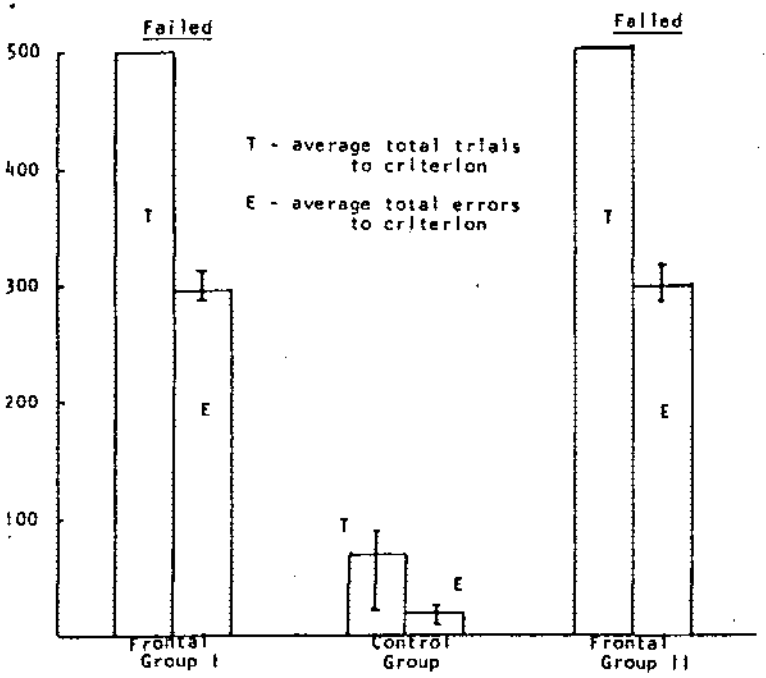


Fig. 7. Bar graph showing results obtained in the transient context modification problem.

were two of his favorites. I wondered whether, in the absence of an established context, the world of the frontal lobe-lesioned monkey looked somewhat like the McCulloch presentations. In fact, I had devised a match task in 1946 to test just this possibility on lobotomized patients. Instructions were given primarily non-verbally by showing the subject how to pick up the alternate match in regularly spaced series of three rows of twelve matches.

1 1 1 1 1 1 1 1 1 1 1 1
1 1 1 1 1 1 1 1 1 1 1 1
1 1 1 1 1 1 1 1 1 1 1 1

Then the following array was presented and the subject asked to do the same thing he had just done with the regularly spaced series:

11 111 1 1111 11
111 11 11111 1 1
11111 1 1 1 111 1

Unfortunately I found that many control subjects, as well as the lobotomized patients had difficulties in performing this task.

With monkeys the following test was devised as a modification of the de-

layed alternation procedure: ordinarily the delay interval between responses is kept constant. A peanut or grape is alternately placed in one of two inverted baskets but not in view of the monkey. Thus the task goes: R (right basket) 5 sec, L (left basket) 5 sec, R 5 sec, L 5 sec, R 5 sec, etc. The modification entertained on the basis of McCulloch's readings was to alter the equal spacing of the delay period into an unequal spacing: R 5 sec, L 15 sec, R 5 sec, L 15 sec, R 5 sec, etc. Behaving according to prediction, the frontal lobe-lesioned monkeys failed the equal spaced task but were practically indistinguishable from unoperated controls in their performance of the unequally spaced task (Fig. 8).

Milner (1971 and 1974) has shown a somewhat similar effect for frontal patients using a test where 'temporal tagging' (rather than the spatial 'tagging' I had tried earlier) is used. A ready interpretation of these results would be that frontal lesions interfere with the temporal organization, the 'parsing', of input so that it makes sense: becomes meaningful. A time tag, such as unequal spacing, would provide the necessary cues to parsing by the frontally lesioned subject which the normal organism ordinarily can supply to some extent himself.

In a not yet completed automated replication of the monkey experiment, a further control procedure was inserted. Each day the 5-15 spacing was

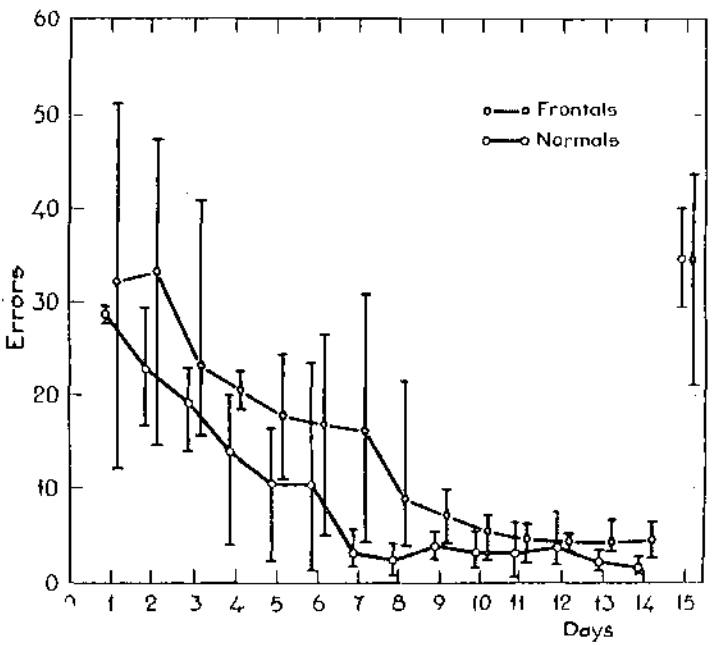


Fig. 8. Results obtained in the modified (6—15) alternation task referred to in text.

reversed so that on Monday the monkey was tested on R 5 sec, L 15 sec, R 5 sec, L 15 sec, R 5 sec, etc., while on Tuesday the order would be R 15 sec, L 5 sec, R 15 sec, L 5 sec, R 15 sec, etc. Again, the frontally resected monkeys are performing essentially as did their unoperated controls (although they cannot maintain a criterion performance as readily as do the controls; see also Pribram et al., 1966b) while doing more poorly on the equally-spaced alternation task (Anderson, Leong and Pribram, in preparation).

For both the operated and unoperated monkeys, this version of the unequally spaced alternation was extremely difficult. This, together with the results relating frontal cortex to spatial context already reviewed, raises the question as to whether temporal tagging is in fact the critical variable or whether temporal tagging is only one of several potent determiners of context. Another way of stating this question is to ask whether perhaps the frontal cortex is involved in the categorizing of relationships, much as the posterior intrinsic cortex is involved in the categorizing of properties. Of course, the possibility remains that different classes of categorization (e.g., temporal and spatial) are dependent on different portions of the frontal eugranular cortex and this possibility needs now to be tested.

In short, the current experimental results confirm and extend earlier ones in suggesting that the frontal eugranular cortex ordinarily serves to inhibit the distracting effects of novel inputs by processing the input (via habituation) in terms of an established context with controls what is attended and intended.

CONCLUSION: THE RELATIONSHIP OF FRONTAL LOBE FUNCTION TO THE TRANSMISSION OF SIGNALS IN THE NERVOUS SYSTEM

With respect to the concern of this symposium, I draw the following conclusion from the results reviewed. The fashion today is to consider brain function in terms of information processing. Usually implicit in these formulations — though occasionally made explicit (for example as by Gibson, 1966) — is the assumption that the information being processed 'resides in' the *input* to the brain, even in the *world* from which the senses derive their input.

The data on frontal lobe function reviewed here, while not denying the importance of input, do focus our attention on a currently neglected aspect of brain function: its spontaneous activity, its generative capacities. True enough, the spontaneous neural rhythms become initially programmed by input (unless the programs are pre-established, i.e. innately given) but they are then maintained as central states by memory mechanisms that serve as the context within which subsequent input becomes processed. Our search for the routes taken by information processing need not, therefore, necessarily come up with an input → central processor → output paradigm. Rather, as demonstrated here for frontal lobe function, and elsewhere (Pribram,

1971 and 1974) for other parts of the brain, a more practical and realistic paradigm is central processor $\begin{cases} \nearrow \text{input.} \\ \searrow \text{output.} \end{cases}$

The emphasis in this paradigm becomes the organization of central brain states which control input and output rather than the transmission of signals from receptors to effectors. The change in view is comparable to that in chemistry where analysis of simple one-way reactions gave way to the analysis of reciprocally interacting thermodynamic systems. We therefore must, for instance, begin to look in the nervous system for variables (e.g., time constants) similar to rate-limiting reactions studied by biochemists. Thus we may come to understand that neural inhibition is an organizing process, not one which necessarily leads to the inhibition of perception and behavior: the neural disinhibition resulting from frontal lesions described here results in perceptual and behavioral disorganization (disruption of context) which may in the same animal be manifested as increased distraction (behavioral disinhibition) or perseveration (behavioral inhibition), depending on the situation in which the monkey is studied (Pribram et al., 1964). The task ahead is to formulate additional currently feasible neurophysiological experiments which can detail the mechanism by which the frontal cortex organizes the context — categorizes the relationships — within which behavior occurs.

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DISCUSSION

HIGHSTEIN: Is not the hemianopia different in occipital and frontal lesions?

PRIBRAM: I think the valid point is irrespective of the frontal lobe lesion which gives rise to a very different kind of hemianopia than, let us say, occipital lobe lesion. But the point here is that in one patient reported by Weiskrantz and Warrington there is a dissociation very much as there is in the split brain patient I think of right hemisphere blocking. The verbal report of awareness, I must say, is very operational, it is dissociated from the discriminatory instrumental response, and you see that dissociation may begin to have at least one loop around the problem of what we report to each other on the cerebral context of awareness.

HIGHSTEIN: What cortical lesions are you dealing with?

PRIBRAM: Most of us are dealing with lesions restricted to the occipital cortex and, of course, in the monkey studies it is the same thing.

GILMAN: Along the same lines is Sprague's experiment in which a cat lost discriminatory power after occipital lesions in the hemianopic field, but regained it again after lesions in the contralateral geniculate?

PRIBRAM: No, it is colliculus.

GILMAN: It indicates that it is present whether or not there is an occipital lesion. I think the Highstein point a good one. One has to be careful in dealing with patients who are hemianopic, because many of them have either cortical blindness and retain vision which they recognise, or they can have an agnosia in which they are able to see things but not perceive things. But the data you have mentioned is solid, neurologically.

RAMAMURTHI: I would like to ask a question and to make a remark. You said the input connections of the transcortical connections do not affect the function of the frontal cortex.

PRIBRAM: I did not say that. I said for the tests we have performed.

RAMAMURTHI: This is what we have also learnt in psychosurgery, that the ancient days of prefrontal lobotomies which knock off all the input connections do not give the results, whereas a precise orbital frontal cortex lesion we now make delivers the goods. Secondly, you were talking about the attention that does not concentrate or stay in one point. I do not exactly understand what you said. Did you say that it was dependent on the amygdala connections, or what?

PRIBRAM: No, what I said was that three neural systems are involved. One of them is the amygdaloid nucleus and another is the posterior part of the putamen and the hippocampus.

RAMAMURTHI: That scheme is meaningful in psychosurgical procedures in the human and lesions in the amygdala for certain. Your scheme was facinating, also, from our concept of psychosurgical procedures of arousal, effort and activation. For instance, we make cingulum lesions in obsessions with excellent results. So I think perhaps we may provide neurosurgically or psychosurgically some support for the table that you have made.

PRIBRAM: Well, I hope so. You remember that lesions were tried many years ago in the head of the caudate nucleus and you get the full blown syndrome as you do with frontal lobotomy. The only afferent connections known at that time to the frontal cortex were from the dorsomedial thalamic nucleus, we made lesions in the afferent paths and the effects were not produced. That is very good I think, the efferent paths are important. All I am saying is that here is a little tip of a handle to hang on to the problem of awareness.

PURPURA: I am going to give a silly sentence. When I leave a blank you answer it. I, Karl Pribram, believe that the role of the frontal lobe is to ...

PRIBRAM: ... act as an executor to the rest of the brain. It sets up a programme or a context in which all the other activity takes place, a programme that has all the executive functions of the brain.