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Karl H. Pribram was born in Vienna in 1919. He took his M.D. at the University of Chicago, following which he entered neurological surgery research at the Chicago Memorial Hospital. He was for two years neurosurgeon and neurophysiologist in the Yerkes Research Laboratory, subsequently a member of the faculty of Yale Medical School, then Director of Research and Laboratories, Institute of Living, and at present U.S.P.H.S. Research Professor of Psychology and Psychiatry and Director of the Neuropsychological Laboratory at Stanford University. Dr. Pribram was a Fellow at the Center for Advanced Study in the Behavioral Sciences in 1958–59, where he collaborated with George A. Miller and Eugene Galanter to prepare the well-known *Plans and the Structure of Behavior*. A Diplomate of the American Board of Neurological Surgery, Dr. Pribram's interests are the brain cortex and behavior, and neurological functions in intellectual processes.

HOW THE BRAIN CONTROLS ITS INPUT

KARL H. PRIBRAM

ROSS ASHBY in his now classical *Design for a Brain*[1] models a system which, though stable, adapts to the ever-changing circumstances with which it is faced. The first half of his task concerns the description of one set of conditions that evoke stability: "The stability belongs only to the combination; it cannot be related to the parts considered separately . . . the presence of stability always implies some coordination of the action between the parts." His design for this set achieves "ultrastability" through the multilinking of homeostats, an arrangement of servos that keeps certain "essential variables" within bounds while allowing a freer range of values to the remainder.

Unfortunately for this ingenious design, the thing is so constituted that it can't learn—or rather that progressive adaptation is so sluggish that any resemblance to the function of the wet brain is lost. Ashby tackles this problem in the second half of his design by invoking the concept of temporary independence between parts of the system. Such independence is assumed to occur whenever constancies are achieved between the part and some aspect of its environment—through the process of habituation to the repetitiveness of the stimulus situation. Thus

"constancies cut the system to pieces" and adaptation of parts of the system is immeasurably speeded.

There is thus a jarring opposition in design between the first and latter half of Ashby's model: multilinking and temporary independence of parts vie to capture the system, each process giving assurance of stability to be accomplished in its own fashion. What kind of brain is this that can't make up its mind about its own organization?

My concern here is with a model derived not from engineering considerations, as is Ashby's Design, but from neurobehavioral and neurophysiological data obtained in my laboratory. My argument will be that these data lead to a conception so similar to Ashby's as to be encouraging to both efforts. The mammalian brain as well as Ashby's Design appears to have at its disposal two opposing methods of organization to assure stability—and each has its virtues and limitations.

The immediate data which lead to the model are electrophysiological. These data were obtained, however, against a background of neurobehavioral evidence which posed the problems to which the physiological experiments were addressed. Briefly, the neurobehavioral data concerned the functions of the then silent regions of the forebrain: the so-called association areas and the limbic systems. In the primate brain two large divisions of these regions were identified: the posterior "association" cortex and the frontolimbic formations. Experimental evidence accrued to show that the posterior cortex dealt with the organism's ability to make differential discriminations—to select among alternatives —and that subunits could be identified each of which served one or another sensory model.[2] The frontolimbic formations, on the other hand, were shown to be important whenever the organism had to make a sequence of responses in order to adapt to circumstances, i.e., whenever behavior had to be guided by some internal program which determined serial order.[3]

The puzzle remained as to how these parts of the forebrain effected their function. The puzzle is not yet solved but one part of the picture is becoming clear. The concept which had dominated thinking about these regions of the brain for over a half century was that these areas were the "highest integrating centers," the locus where inputs were amalgamated, "associated" into more complex functions. A considerable literature had accumulated of reports to test this conception—much of it stimulated by the late Karl Lashley. And this evidence overwhelmingly failed to confirm the guiding hypothesis.[4-10] The question thus arose as to an alternative view and I proposed the possibility that these forebrain systems effected their influence through cor-

ticofugal, efferent pathways that operate by making transformations on the input through direct control over the functions of the input channels.[11–13] The tests of this possibility led to the following results:

Experiments were performed on fully awake monkeys implanted with small bipolar electrodes and a device which allows chronic repetitive stimulation of one of the electrode sites.

The monkeys were presented with pairs of flashes and the interflash interval was varied from 25 to 200 msec. Electrical responses were recorded from the striate cortex and the amplitude of the responses was measured. A comparison of the amplitude of the second to the first response of each pair was expressed and plotted as a function. The assumption underlying the interpretation of this function is that when the amplitude of the second of the pair of responses approximates that of the first, the responding cells have fully recovered their excitability. In populations of cells such as those from which these records are made, the percent diminution of amplitude of the second response is used as an index of recovery of the total population of cells

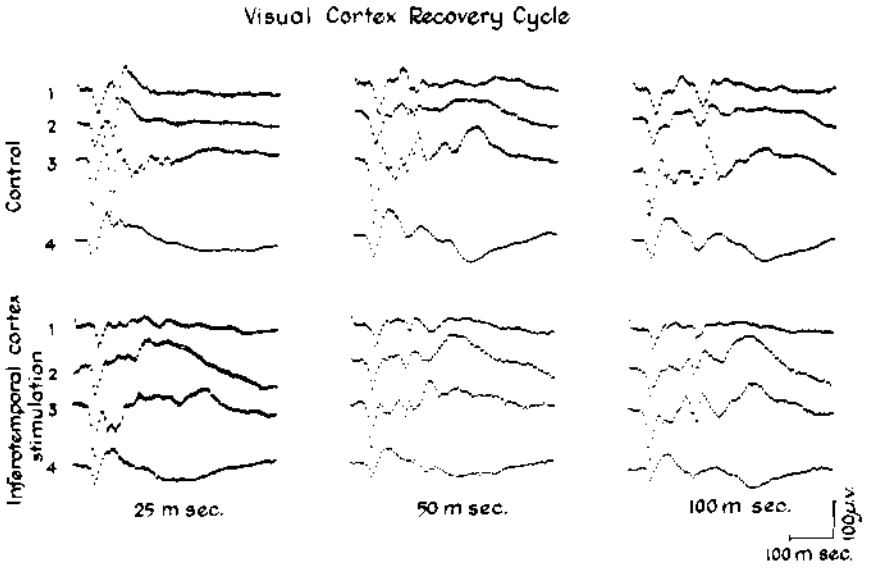

Figure 1. A representative record of the change produced in visual evoked responses by chronic stimulation of the inferotemporal cortex. Upper set of records was taken before stimulation, the lower set, during stimulation. All traces were recorded from the visual cortex; in the first column are responses produced by a pair of flashes separated by 25 msec; flash separation is 50 msec in the second column, and 100 msec in the third.

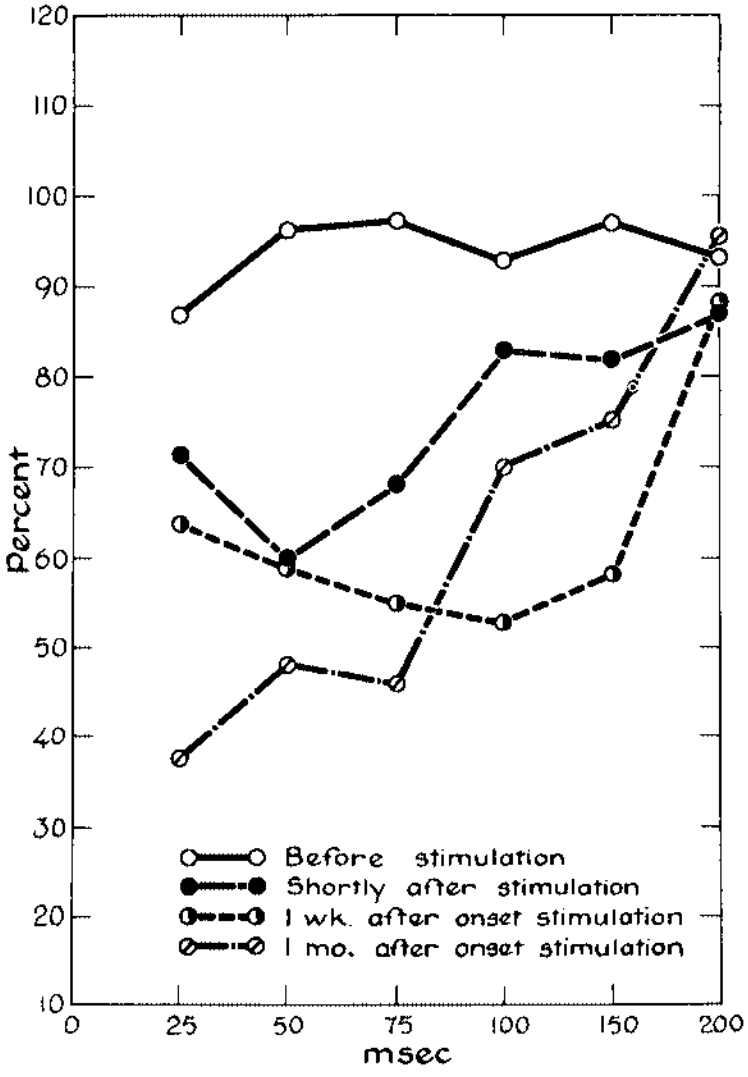

Figure 2. A plot of the recovery functions obtained in one monkey before and during chronic stimulation of the inferotemporal (IT) cortex.

—thus the smaller the percent, the fewer the number of recovered cells in the system.

Chronic stimulation (8–10/sec) of several cerebral sites alters this recovery function (Figs. 1 and 2). When the inferotemporal cortex

of monkeys is stimulated, recovery is delayed. Stimulation from control sites (precentral and parietal) has no such effect. Nor does the stimulation of inferotemporal cortex alter auditory recovery functions. These, however, can be changed by manipulations of the insular-temporal cortex as was shown in a parallel experiment performed on

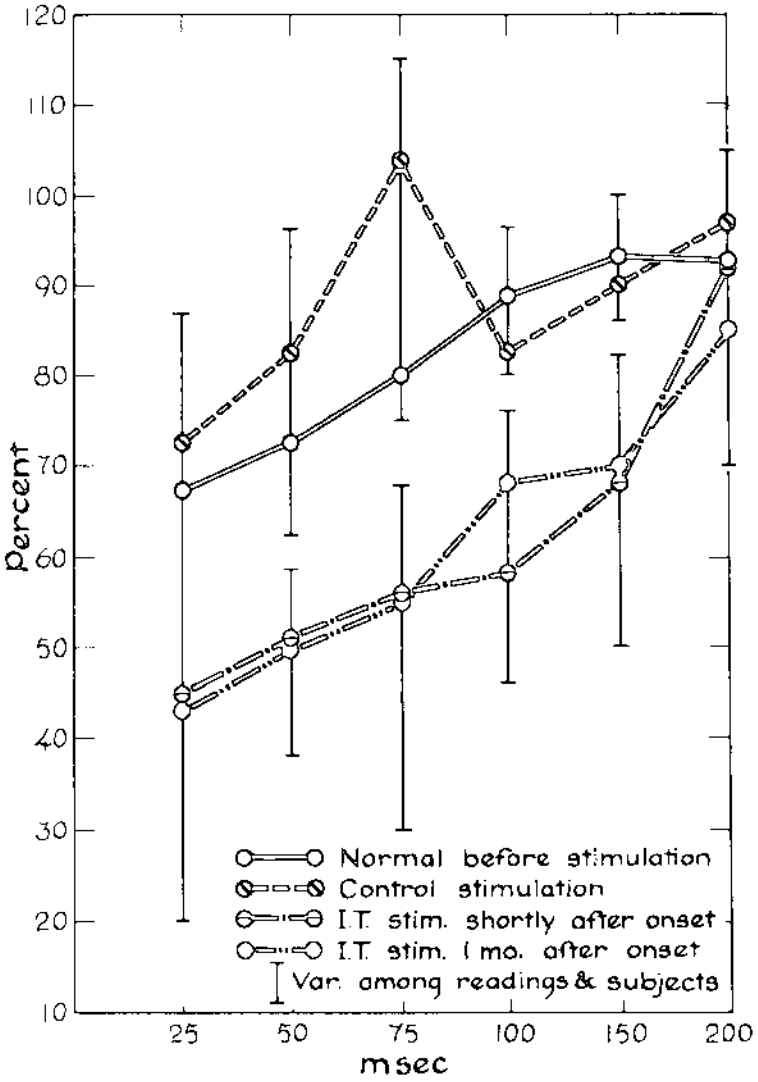

Figure 3. A plot of the recovery functions obtained in five monkeys before and during chronic cortical stimulation.

cats. Here the crucial cortex was removed and recovery functions obtained on responses recorded from the cochlear nucleus.[14] Removal of insular temporal cortex shortens recovery in the auditory system.

A great many neurobehavioral experiments have shown the importance of these isocortical temporal lobe areas (and not others) to visual and auditory discrimination. These studies are reviewed elsewhere.[2, 13] What concerns us here is that a corticofugal, efferent mechanism is demonstrated and that this mechanism alters the rapidity with which cells in the visual and auditory afferent systems recover their excitability. Further, since stimulation delays and ablation speeds

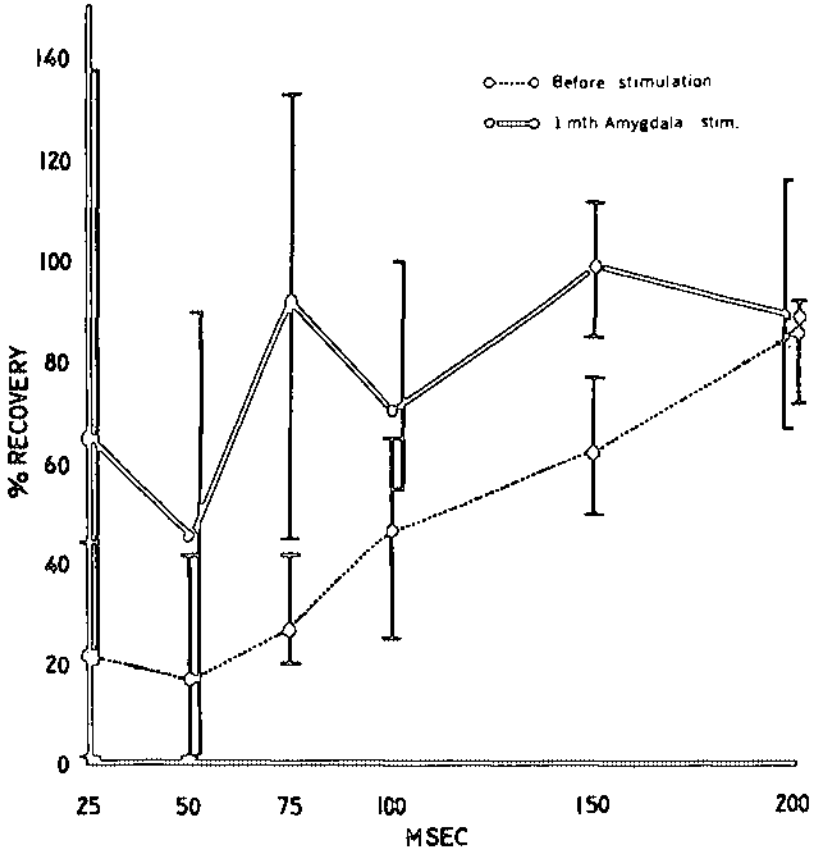

Figure 4. The effect of chronic stimulation of the amygdaloid complex on recovery function. The dotted line indicates the function before, the solid line after, one month of stimulation. Bars perpendicular to the curves show variability among subjects. Each curve is based on the average response of four subjects.

up recovery, the inference is that the normally afferent inhibitory processes which delay recovery are enhanced by the ordinary operation of these temporal lobe isocortical areas.

But the opposite effect—namely inhibition of afferent inhibition—can also be obtained when cerebral tissue is chronically stimulated (Figs. 3 and 4). In these experiments the cortex of the frontal lobe and the cortical nucleus of the amygdala were chronically stimulated and recovery of cells in the visual system was shown to be speeded. This

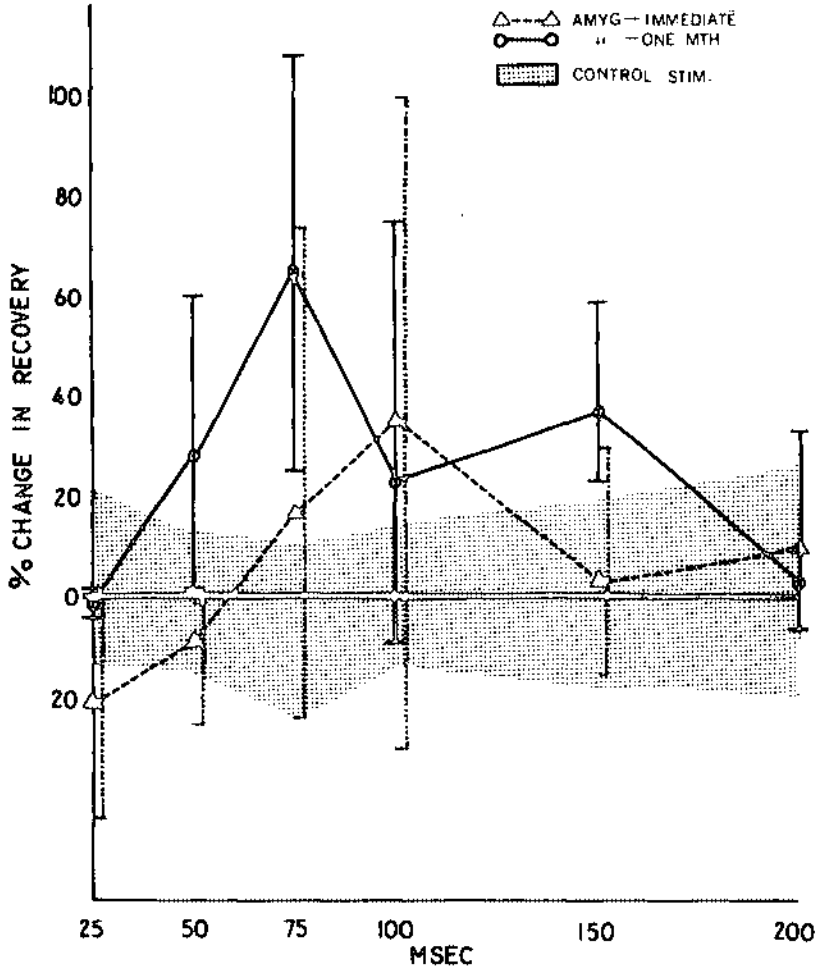

Figure 5. This figure represents the same data as those represented in Fig. 4. However, here percent change in recovery is plotted. Shaded area indicates range of recovery for *un*stimulated subjects.

result has suggested that the frontal and medio-basal portions of the forebrain—the frontal cortex and limbic regions—function as efferent systems which inhibit afferent inhibitory processes.

This antagonistic effect of these two efferent control systems is perhaps best illustrated by data obtained at the unit level (Figs. 5 and 6).

These unit recordings were made from the striate cortex of Flaxedilized cats to whom flashes of light were presented. Note that the silent period of a cell can be lengthened by concurrent inferotemporal stimulation. Note also that concurrent frontal stimulation can shorten this

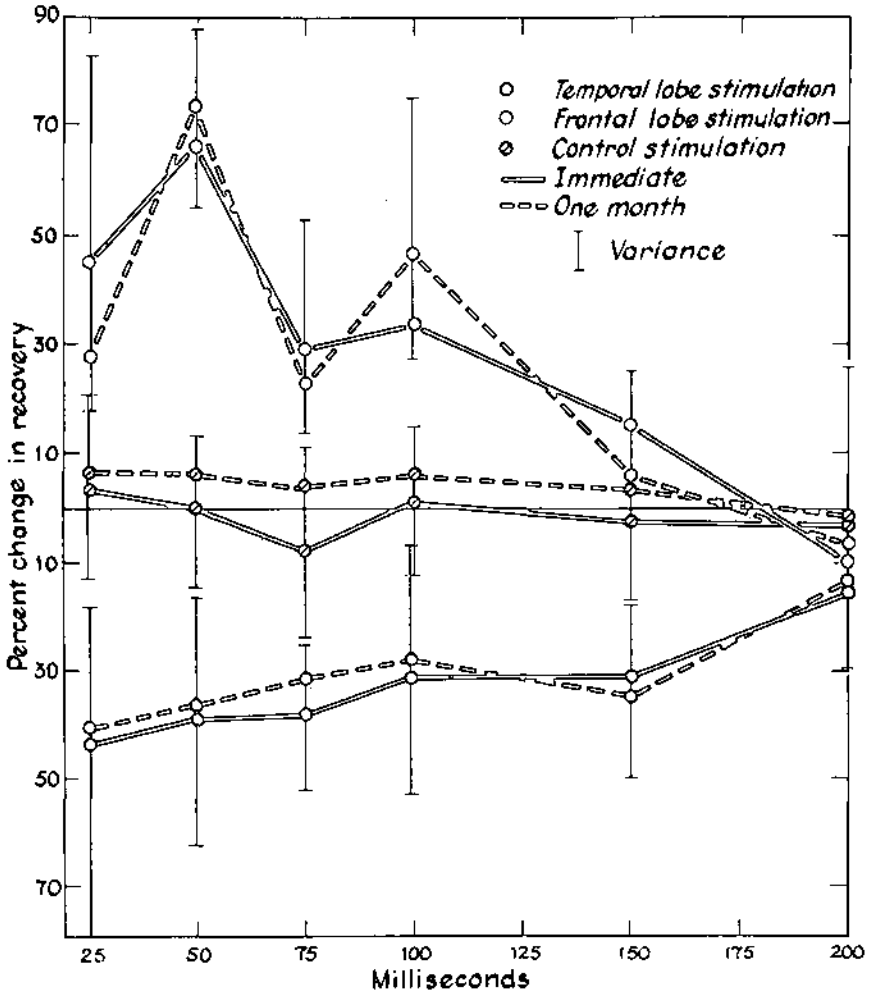

Figure 6. This figure plots the percent change in recovery for all subjects in the various experiments. It is thus a summary statement of the findings.

silent period. Finally, note the unit whose silent period is lengthened by inferotemporal and shortened by frontal stimulation.

These data demonstrate the existence of mechanisms in the brain which can exert afferent, corticofugal control over the input channels. Such effects have been shown by similar experiments to extend as far peripheral as the cochlear nucleus in the auditory system[16] and the optic nerve in the visual system.[17] There can therefore be little question that the brain controls its input.

The results of these experiments also give a clue as to at least one dimension over which this control is exercised. The classical interpretation of recovery functions is what their name implies: When the second response to a pair of stimuli is smaller than the initial response of the pair, the assumption is that some cells in the system are still occupied with processing the initial stimulus. This interpretation suggests that any parameter which delays recovery effectively decouples the system with regard to successive inputs while any parameter which enhances recovery effectively links the units of the system to each other. This independence-interdependence dimension is exactly the one which emerges so clearly in Ashby's Design. The further suggestion is therefore that the mammalian brain, just as its hardware model, has at its disposal two antagonistic reciprocal mechanisms by which it can assure stability to the system in which it acts: a mechanism of "external" control, through which constancies are achieved between the parts of the system and their environment; and a mechanism of "internal" control, which relies on achieving a join between parts.

I have detailed elsewhere[15, 17–19] the explanatory power which this interpretation of the electrophysiological findings brings to bear on the neurobehavioral data which generated these experiments. I should like to take this occasion to develop a few of the questions raised by the model.

The first of these deals with the problem of the permanency of adaptation achieved through external control. How is it possible for a differential discrimination, the recognition and identification of a situation, to become part of the permanent repertoire of the organism if the discriminatory process depends on the achievement of temporary independencies among the parts of the system? Obviously, these temporary independencies and the part constancies through which they are attained are idiosyncratic to the environmental situation, the input configurations in which they occur. Altered input will alter the organization of the constancy structure. How will "recognition," i.e., recurrence of the appropriate organization, be realized when the initial situation

or a similar one is again in effect? In other words, what is stored of the configuration of temporary constancies and how is this storage accomplished?

It is unlikely that storage by way of a system of sequentially organized programs will account for the almost instantaneous recurrence of the configurations of constancies demanded by the "immediacy" of recognition. Much of the "memory" for the event is, of course, a function of the input itself; "storage" is found in the characteristics of the situation itself as well as in the brain. But some sort of neural mechanism must be available, ready to respond to this idiosyncratic input configuration. A content-addressable, parallel processing neural process is the most likely candidate to achieve what is required. And the anatomical structure of the input processing mechanism is especially suited to just such a process.

More difficult to visualize is a mechanism which allows the fully joined system to adapt to recurrent regularities in the situation. Is such adaptation achieved only by a partial giving up of the interdependence among parts as suggested by Ashby? This is unlikely since ordering of behavior sequences is apparently dependent on the mechanism of internal control. More consonant with the data available is the suggestion that multilinking is not statistically haphazard as in Ashby's Design but is itself exquisitely structured.[20, 21] This structure is felt to take the form of the branching programs that constitute the software of present-day computers. However, we have not yet been able to devise a straightforward neurophysiological test of this view. There is some hope, however, that the current interest of neurophysiologists in the mechanisms of neuronal inhibition may prove the key to the problem. A precise rendering of the model here presented in terms of afferent neural inhibition has therefore been constructed.[22] Our hope is that tests of this aspect of the model will turn up the missing clue and that steps toward a neurological explanation of the serial order enigma are thus within reach.

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DISCUSSION

Burke: I would like to ask whether one could analogize, from the two kinds of control you talked about, to two situations in people. In one case, a situation might be modified by being acted upon through external control; in the other, the individual himself might adopt an attitude of resignation or philosophic calm. Could one analogize in that way?

Pribram: Not quite. The word control has nonscientific pejorative connotations, but in engineering language something precise is meant. The control mechanisms to which I referred are internal. In a more popular sense, they are mechanisms of self-control. So when I suggest that an organism seeks stability through external control, he does not control others. That is not what is meant. What is meant is that he allows influences from other organisms to alter his own image, his memory traces, and in this fashion allows a constancy to develop between himself and other organisms.

Burke: Neurologically is there anything that would correspond to the difference between, on the one hand, my striving to modify a situation that is bothering me, and on the other, my passive acceptance of it or yielding to it?

Pribram: I have been talking about emotion rather than motivation. By this I mean that one way organisms have of coping is to control input rather than *doing* something to the environment. In other words, a program or plan can be run in either direction. One can have a program that is carried through or the program can "allow" itself to be altered or to return to some earlier phase. I have not been talking about different directions behavior can take, i.e., motivation. Motivation determines whether one writes a poem or becomes an executive. I believe this subject takes us away from my topic—which is aimed at the properties of the system which allow that system to control its own input. One such adjustment allows constancy to be attained by relating to the environment through making internal changes. The other achieves stability by conserving the internal structure through altering input.

Foa: Given that some internal steady state had been achieved relative to status, which kind of control, in your terms, would be brought into play in case of changing status stimuli?

Pribram: That's internal control. I can usually tell whether someone is largely under internal or external control. If there is a glassy look and he says, "Yes, of course," what I say goes in one ear and comes

out the other; it doesn't register. We have done some research on this topic. Using the orienting reaction, we have found two components that can be distinguished. One indicates registration and the other the sampling function I have already described. Sampling you have experienced, I'm sure. You will be talking to your wife and a little while later she says, "You aren't listening to me." To show her that you have been, you can repeat everything that she has said. Yet if she should ask you five minutes later what topic she was discussing you really wouldn't know. The content never registered. And we know some of the physiological bases for this registration function.

Foa: But these are not happening at the same time.

Pribram: They do exist contemporaneously and quite often they project out onto the same cells. We have examined several cells so far that can be moved in this direction or that direction depending on whether the stimulation is of the frontal or the posterior cortex.

Vickers: Once more about external control: This presumably provides a selective acceptance of what we are to experience. Is that right?

Pribram: That's right. Without getting into a model of the perceptual process, I can't really talk about it easily. But the external control mechanism arrives at constancies with respect to certain environmental events; if enough of those occur, the system becomes stabilized.

Foa: Is this not a change of field?

Pribram: They're not exactly fields. From my perspective, they are changes in the pattern of function in the input channels.

Foa: This is not the same sort of thing then as the controls that are exercised within a group to stabilize it?

Pribram: No, it is not. These are two different levels of systems.

Foa: Let me give an example. Assume that at the beginning of this meeting, you had to take care of the internal control by yourself. After a time you become part of the group and then these stabilization functions are taken up by the group.

Pribram: Fine, that's the notion of external control. That's Ashby's notion of external control.

Shands: I am interested in the nonspecific effects of hospitalization. For example, you might admit a wildly excited patient and then three days later, whether it's a good hospital or a poor hospital, the patient is recovered and leaves. I should think that this is a function of the reduction of the potential variabilities of the situation. Is this, then, similar to what you are calling external control?

Pribram: No. The manic person probably doesn't have much in the way of either internal or external control: His stability is almost

completely disrupted or you wouldn't call it manic. Among other things, there is a constant struggle to build up constancies. By cutting down on the input this process may be aided. With people who are unstable, a higher level of control—control from outside the "organism's" system—may be useful. Put them into a stable situation, and provided they haven't abandoned the "external control" mechanism, they are amenable to this kind of stabilization.

Shands: Where would you put the effects of drugs into your scheme?

Pribram: You remind me of something I meant to cover. Purpura showed that, under LSD, recovery functions are slowed—suggesting that a mechanism similar to that operating in "external control" is involved.

Orr: Another question in the same vein: Is the contrast you have established between internal control and external control analogous to that between inner-direction and outer-direction at the personality level?

Pribram: I have been tempted to think so. But how would one go about testing this hypothesis at the social level?

Foa: Is the control function inferred from the state of the system, or would notions like need and goal have to be considered?

Pribram: We do have a physical science description of what a steady state is. In anything that gets organized, the very fact that structure can be identified suggests that a steady state of some sort has been achieved. So there is more to it than just its function.

McCulloch: I would like to suggest that it is not a matter of being "inner-directed" or "outer-directed." It is a matter of which way one seeks his stability. Does he seek it within himself or does he seek it through his relations with the world? One can do it either way and he has to switch from one to the other according to how familiar he is with the situation. But I don't think that is the same thing as inner-direction or outer-direction.

Lillywhite: Did you relate interference to input in those situations in which there had been actual brain damage?

Pribram: Yes. These experiments began as a study in subhuman primates of the problem of agnosia. I believe that there is a connection between what in humans we identify as agnosia and what in our monkeys shows up as a failure to identify visual or auditory (or somatic or gustatory) cues. The neurobehavioral evidence is pretty well in—and I have tried here to spell out a possible neural mechanism to account for the gnostic function and its disability.

Upton: Have all your remarks operated at a level too high to give consideration to the nature of the iris under the influence of light?

Pribram: No, not of light. But pertinent here, of course, is the observation of Hess that when people are interested in something, their pupils dilate slightly, and when they are essentially "gating out" their surround, their pupils constrict. It would be very fruitful to have such data. So far we've been talking about changes occurring inside the nervous system. It is natural to wonder whether or not these changes are accompanied by changes in the motor apparatus of some cerebral palsy patients, where over-input from the inside is countered by increased input from the outside?

Meerloo: Would this be comparable to what has been going on in perception?

Pribram: It may be. It's a very complicated question, particularly in view of recent new evidence produced by Berman and his group about the function of dorsal roots in spasticity. These sorts of questions concern the control of output, and as yet we have very little evidence. My emphasis here has been on the control of input mechanisms, and these are the things for which we do have some evidence.

Meerloo: It's a local control of input.

Pribram: You may be right. What you are saying is that the input coming from upstairs is being swamped at that particular location. The result you describe sounds reasonable. But what we don't know is how input control affects output mechanisms.