T-67

22 How Is It That Sensing So Much We Can Do So Little?

KARL H. PRIBRAM

ABSTRACT I summarize in this chapter the reports made by the others and interpret their results in terms of my own experimental findings. These are (a) information is distributed in the striate cortex; (b) nonvisual information becomes encoded in the visual cortex; (c) resections of the inferior temporal cortex produce devastating deficits in visual discriminations involving choices but do not interfere with ordinary visual processing; (d) radical resections of the circumstriate cortex do *not* interfere with the performance of behavior involving such visual choices. On the basis of these data, a proposal is entertained that the functions of the inferior temporal cortex are carried out by addressing in parallel (attending) the information relevant to the decision, information that is encoded in a distributed (holographic) fashion in the primary visual system. Experimental evidence to support this proposal is adduced.

Introduction

MY STARTING point in delineating the neural mechanisms that relate the separate visual functions of the anatomically separable visual systems is a set of experimental data that do not fit what I was taught (see, e.g., Pribram, 1960). Kornhuber elsewhere in this volume reviews with you the classical view of the functions of the cerebrum as composed of transcortical reflex arcs: Beginning in the sensory projection areas, converging on association cortex and leaving the brain via motor cortex, these processes were initially couched in terms of the association of ideas but today are still put forward in the language of information processing. Nor are they completely false: Other contributors to this 1972 Third Study Program have reviewed the evidence that in fact the discrete organization that characterizes the primary sensory and motor projection systems gives way to a broader organization in the perisensory areas and that, often beyond this perimeter, cellular electrical response depends on input from more than a single sensory modality. Yet, as Kornhuber details for us, the transcortical reflex arc has been found wanting as a model for explaining not only the results of experiments on the motor systems but also of clinical observations on the agnosias and apraxias.

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It is just these clinical entities we set out to study a quarter of a century ago by making animal models, producing brain damage hopefully comparable to that found in man (Pribram, 1954). The results of these experiments are the ones that have raised the need for a more useful model.

The model that has gradually emerged from the data, some of which I will present now, might be called a *recognition program-tape model*. At the brain level of organization, the model resembles in many respects that presented for the cellular level by Edelman elsewhere in this volume. As he pointed out, the antigen-antibody problem is that of Maxwell's recognition demon: Is specificity due to selection or associative instruction, or perhaps both? As I understood his presentation, there is overwhelming evidence for an alphabet of preformed antibodies within the cell that becomes assembled into a specific "receptor" at the cell surface by a series of steps involving feedback at each step between antigen and the cell's response to that antigen.

I want now to present evidence that a similar stepwise process characterizes the construction of Maxwell's psychological recognition demon by the brain. The apparatus consists of an alphabet of image elements distributed in the primary sensory systems. This alphabet becomes assembled by steps involving feedback at each step between a particular input and certain elements of the alphabet into a specific program tape, the analogue of Edelman's cell membrane receptor. The program tape then preprocesses, that is, is specifically sensitive to, subsequent occurrences of that particular input.

There are five separate classes of empirical questions that are generated by the model: (1) those that characterize the alphabet; (2) those that testify to its distributed nature; (3) those that inquire into the mechanism of distribution; (4) those that specifically concern the assembly of the program tape; and (5) those that delineate the functions of such a program. There is considerable independence between the data sets that constitute the domains of each of these questions—the degree to which one data set is supportive or destructive to hypotheses within a domain should therefore, at this stage of model

building, not influence credibility too greatly in another domain. Thus, I am able to spell out fairly precisely both the data that gave rise to the overall model and the limitations exposed in each domain when specific hypotheses were tested.

Characterizing the alphabet

The alphabet of image elements is characterized by the receptive field properties of units in the primary visual system. I want to discuss especially that part of the alphabet that shows the most striking properties, the orientation sensitive neurons discovered by Hubel and Wiesel. Pollen showed us in his preceding chapter that the output from any one of these neurons is actually ambiguous with regard to orientation: Changes in number, width, and contrast of lines influence output as much as does orientation. Only a population of neurons can code orientation. Pollen distinguishes, as do Hubel and Wiesel, between the properties of *simple* and *complex* cells; but overall, both his work and that of Fergus Campbell, in the preceding part, show that the job of this population of orientation sensitive elements is to respond selectively to spatial frequency that specifies not only orientation but also number, width, and luminance contrast of the input lines (gratings) used as stimuli.

Specification of spatial frequency can produce reconstructions of images in detail far beyond any that can be obstructed using orientation-sensitive mechanisms only.

In terms of the analogy to a verbal alphabet, we might think of the spatial frequency elements as the *vowel* part of our receptive field alphabet. Vowels, of course, are the carriers of speech onto which the consonants are grafted. Let me now show you what this vowel part of the alphabet of image elements looks like by visualizing for you the receptive fields of cortical neurons in cat and monkey (Figure 1). These receptive field maps were made by a technique devised by Spinelli (1966).

A small white spot is held against a black background by a magnet, which is attached to an *X-Y* plotter. The *X-Y* plotter is controlled by a small computer that therefore *knows* where the spot is. The spot is moved about the background, and a record is made with tungsten microelectrodes of the number of impulses evoked in a neuron in the visual system for every location of the spot. The computer then displays this record either in a three-dimensional contour diagram, a two-dimensional cross-section of that contour diagram usually taken two standard deviations above background activity, or a series of histograms. The most useful display for us has been the cross-sectional display. Here are some orientation sensitive receptive fields portrayed by this method. Note, as Colin Blakemore remarked in one of the discussions, that each

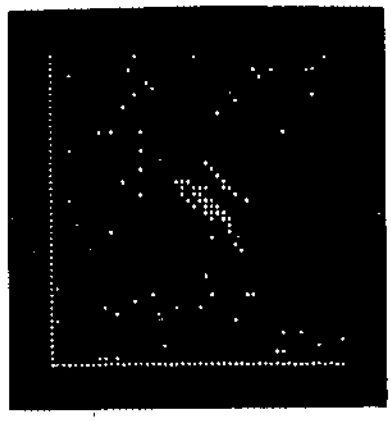


FIGURE 1 Bar shaped receptive field. Direction of scan: Vertical. Firing levels: 1 or greater. Note adjacent inhibitory bar and secondary excitatory field.

is characterized not only by an excitatory bar but by an inhibitory region to one side of that bar and often by another somewhat less distinct excitatory bar. It is this configuration that suggests spatial frequency sensitivity, and Pollen has just recently demonstrated with a different technique that complex cells are maximally sensitive to four such bars.

But let us remind ourselves at this point (Figure 2) that these *vowel* parts of the *alphabet* are not the only receptive fields demonstrable. The orientation sensitive units—simple and complex cells—are only part of population, about 10% at the foveal representation of the rhesus monkey cortex (Jung, 1961; Creutzfeldt, 1961; Spinelli, Pribram, and Bridgeman, 1970). Ernst Pöppel made this point in one of the discussions by registering his surprise on finding so few orientation sensitive units when he first mapped visual cortex. We tend to neglect the many other sensitivities of cells (for example, those to color, De Valois, 1960; or those to auditory stimuli, Spinelli, Starr, and Barrett, 1968) in the primary visual cortex. It remains an open and important question, however, whether the pattern recognition mechanism is dependent solely on the spatial frequency analyzing neurons in the visual system.

Evidence for the distributed nature of the alphabet

Just as an alphabet of antibodies appears to be distributed relatively randomly within a cell, the alphabet of image elements appears to be randomly distributed within the striate cortex. This is not to deny the columnar organization of related orientation sensitive elements (Powell and Mountcastle, 1959; Hubel and Wiesel, 1968; Werner, 1970) but only to point out that the information encoded within any block of columns is repeatedly replicated in other blocks considerably removed anatomically from one

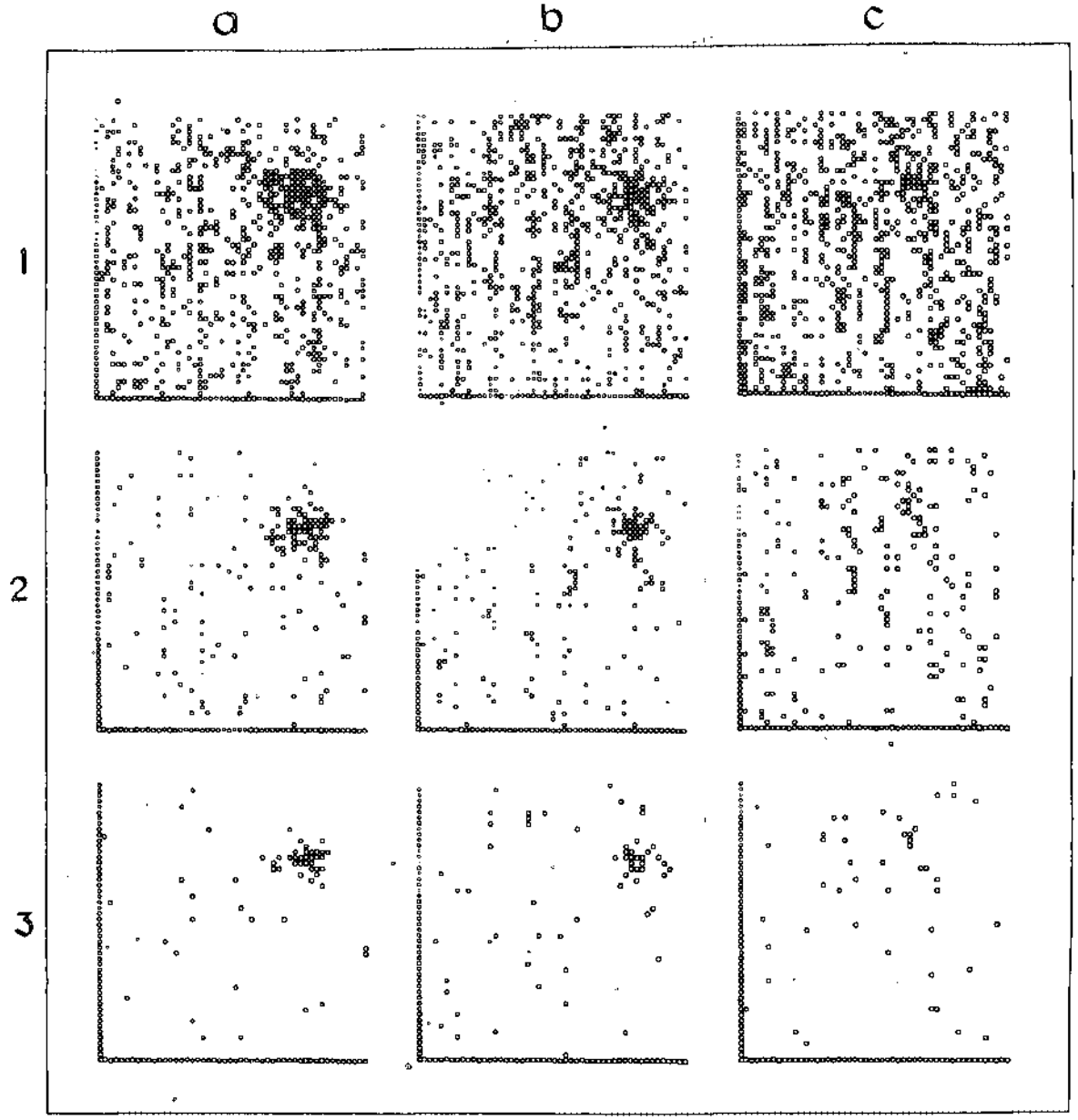


FIGURE 2 This figure shows a disk-shaped receptive field. In column a the unit was mapped with both eyes open; in columns b and c with the left and the right eye respectively. Rows 1, 2 and 3 represent regions where the unit fired 1, 2, 3 times or more, respectively.

another. Indirect evidence for such wide distribution of encoded information comes from observations and experiments on patients and animals who have suffered damage to their visual brain. When a patient suffers a stroke that wipes out half or more of his visual system, he does not go home to recognize only half of his family. With whatever visual field he has left he is able to recognize all that he ever recognized. Weiskrantz earlier in this part delineated the refinement of the laboratory model of this clinical fact; he reviewed the earlier work of Lashley (1929) and of Klüver (1941) on this topic. Perhaps less well known are the recent experiments of Galambos et al. (1967) and of Chow (1970). Galambos cut as much as 98% of the optic tracts of cats bilaterally—the 98% was verified anatomically—and the cats showed remarkable retention of the ability to discriminate figures such as the letter *F* from an upside down *F*, even when changes in size or a reversal of the figure-ground relationship (black-on-white to white-on-black) were made. In order to control for the possibility that scanning with a small tunnel of

remaining visual system would account for these results, Chow took these experiments one step further by combining such lesions of the optic tract with extensive removals of the cat's striate cortex and again demonstrated remarkable retention of the animal's ability to make visual discriminations.

But there is also direct evidence for anatomical distribution of information in the primary visual system (Figure 3). In a series of experiments Spinelli and I showed that

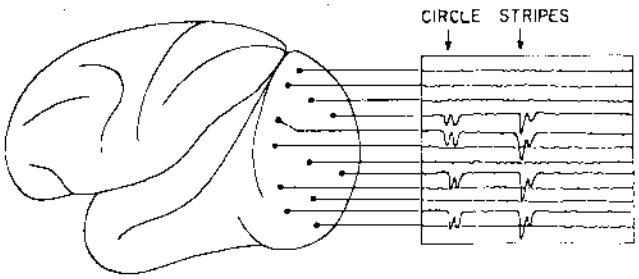


FIGURE 3 A diagrammatic representation of the finding that the differences in the potentials evoked by circles and stripes are distributed over the striate cortex. Note that not every lead shows the differences.

we could record, with small macroelectrodes implanted in the striate cortex of awake monkeys, different configurations of electrical responses evoked when the animal was exposed to brief (10 μsec) flashes of circles and of stripes. Of interest, here is the fact that these differential configurations were not recorded from every electrode, rather they were recorded from apparently random locations over the extent of striate cortex. However, each electrode location that showed the differential evoked response did so reliably for weeks and months.

The mechanism of distribution

The question arises as to how information becomes distributed in input systems. One simple way would be if the brain were like a randomly connected net, but the exquisite anatomical sensory-topic organization of the visual, auditory, and somatosensory systems rules this out. The random-net explanation has been utilized extensively and almost to the exclusion of any other by the computer simulation community, and even there has not fulfilled the earlier promise (see Minsky and Papert, 1969). For years, therefore, neuroscientists were baffled by this problem, if they faced the problem at all. For me the issue of the mechanism of distribution became an experimental one with the advent of a realizable alternative to the random-net proposal. This realizable alternative is provided by the holographic process.

What is the holographic hypothesis of brain function in perception? The weak form of the hypothesis simply states that percepts (images of objects) are reconstructed by activation from input of a distributed information store. A writeout from the core memory of a computer would satisfy this definition. The strong form of the hypothesis is much more interesting, however, because it specifies process and therefore experiments to test its validity. The strong form of the holographic hypothesis states that images are reconstructed from a distributed information store by a transform of the input, a transform that on a prior occasion was responsible for the distribution of the information. Fourier holograms and their equivalents in the spatial frequency domain are the models for this strong form of the hypothesis (Pribram, 1966, 1972).

A hologram arises in any system, whether optical, computer, or neural, when neighborhood interactions among elements (e.g., spatial frequency) become encoded in the process of transformation. This chapter is not the one that permits my detailing for you the mechanism of neighborhood interaction, but this is extensively covered in the first half or so of my book *Languages of the Brain* (Pribram, 1971). Essentially, I make the case that lateral inhibitory networks are involved in organizing a microstructure of slow potentials occurring at synapses and in dendrites and show that the resulting interactions can be described in spatial frequency terms. Fundamental to the proposal are the findings (1) that prior to the ganglion cell layer in the retina, practically no nerve impulses are generated; thus, receptive field organization at the optic nerve level is structured by interactions among slow potentials. (2) At the cortex, both intracellular (Benevento, Creutzfeldt, and Kuhnt, 1973) and extracellular (Phelps, 1972) recordings have demonstrated inputs to be excitatory (depolarizing) while horizontal interactions appear to be exclusively inhibitory (hyperpolarizing).

What, then, would be the advantages to holographic encoding? They are (1) Equivalence of functional parts (the distribution of information) and therefore resistance to damage. From very small parts of the hologram, the entire image can be reconstructed. (2) Large memory storage capacity: In physical holograms 100 million bits of information have been stored in 1 mm^3. (3) Associative recall: When only part of the input that originally constituted the hologram recurs, the remainder of the scene is reconstructed as a *ghost* image. (4) Translational invariance: Recognition and recall can take place irrespective of the position or size of the input. This provides a mechanism for a zoom effect which when pathological becomes macropsia or micropsia. (5) Instantaneous cross-correlation between stored and input patterns and among input patterns. (6) Reversibility (invertibility): The transform restores the original in all its textural detail.

Thus holographic processes can serve as catalysts to other brain mechanisms. A corollary to this is that there are other brain mechanisms; even the strongest form of the holographic hypothesis does not suggest that these are the only transformations that occur in the input systems or elsewhere in the brain. May I again resort to my analogy of vowels in our verbal alphabet. They are the essential binding elements that make speech possible; they do not, however, completely specify the entire range of the alphabet or its combinatorial powers.

And where do we stand with regards to neurophysiological and neurobehavioral data that relate this model to brain function? What are the virtues and the limitations encountered when tests of the model are made? You have already been exposed to the evidence presented in support of the existence of a series of spatial frequency sensitive mechanisms operative in the visual (and auditory) systems (Campbell, earlier in this volume; Pollen, in the preceding chapter). But these mechanisms appear to be relatively broadly tuned. Any invertible process such as the Fourier transformation demands independent, narrowly tuned channels to be effective. Pollen has proposed that simple cells function as strip integrators that provide some independence. Whitman and Spitzberg (1972) have suggested, on the basis of their evidence, that the spatial frequency domain functions much as does the color domain: that three fairly broadly tuned retinal processes become neurally analyzed into a spectrum of narrowly tuned spatial frequencies at the cortical level. To subject these suggestions to neurological test is feasible; e.g., can opponent processes be demonstrated to operate for visual cells in the spatial frequency domain as DeValois (1960) has shown them to operate in the color domain? Henry and Bishop (1971) have devised an interesting technique using binocular stimulation to demonstrate opponent properties of simple cells.

Pollen, in his contribution to this section, has made several additional suggestions. In our laboratory as well as his, experiments are completed or under way to investigate the sensitivity of cortical units to bars spaced at different distances, the effect of presenting several spots or lines in various orientations simultaneously, etc. The issue is: How closely do the quantitative descriptions of these interactions come to expressions of invertible transforms? During our discussions, MacKay suggested that a modification of a Fourier process called a *logon* may be expected to fit better the interactive receptive field characteristics of visual neurons than any simple invertible transform. Gabor (1969) has already published alternative mathematics that could accomplish a holographic process as does the Fourier transform.

But perhaps the most critical limitation on the strong form of the holographic hypothesis to date is the evidence presented to us by Pollen in the preceding chapter. This limitation comes from the small size of the visual receptive fields of striate cortical neurons—especially in the foveal representation. What is necessary to make the holographic hypothesis swing is a mechanism that simultaneously covers a large number of receptive field elements.

Assembly of recognition program

The anatomical and lesion evidence presented by Weiskrantz and by Jones earlier in this part suggests that the circumstriate belt (peristriate or prestriate cortex, Brodmann's areas 18 and 19, Zeki's areas V_2 and V_3) is the locus of neural elements that could provide this mechanism by assembling the input from a number of striate cortex neurons. Recall that striate plus circumstriate cortical resections lead to a monkey sensitive primarily to luminous flux and that the organization of the circumstriate belt is such that as one moves forward within the belt, larger and larger visual receptive fields become organized. It remains to be shown what are the spatial frequency sensitivities of neurons in this belt.

Sounds simple, doesn't it? All we should now have to do is relate the functions of the temporal lobe cortex to this mechanism and then have a holiday. This is the truth table we have been presented so far, which is presented by Jones earlier in this part. Note the unidirectional arrows. Professor Jones assures me that truth table gremlins are responsible for some omitted arrows: In fact, cortico-cortical connections are for the most part reciprocal over short distances. Alas, the truth table conflicts with, to use Weiskrantz's challenging homily, *a table of ignorance* that has been generated by lesion experiments performed on the circumstriate belt. Removal of the belt without damage to the striate cortex does not irretrievably destroy visual pattern recognition: In fact, in this monkey (Figure 4) formal testing showed a complete sparing of the ability to recognize.

But I must pause here a moment to analyze a discrepancy in results that has plagued those of us working on this problem. Mishkin (1966), you will recall from Weiskrantz's presentation, has shown that partial lesions restricted to the upper or lower half of the circumstriate belt produce no effect on discrimination performance, while total lesions of the entire belt do. This evidence apparently conflicts with that of Gross, Cowey, and Manning (1971) who report that lesions of the *foveal* (i.e., ventral) portion of the circumstriate belt *do* result in a deficit. The results of both of these investigations are seemingly at odds with my observations that I have just shown you. A closer look at the data and techniques goes a long way toward resolving these discrepancies. First,

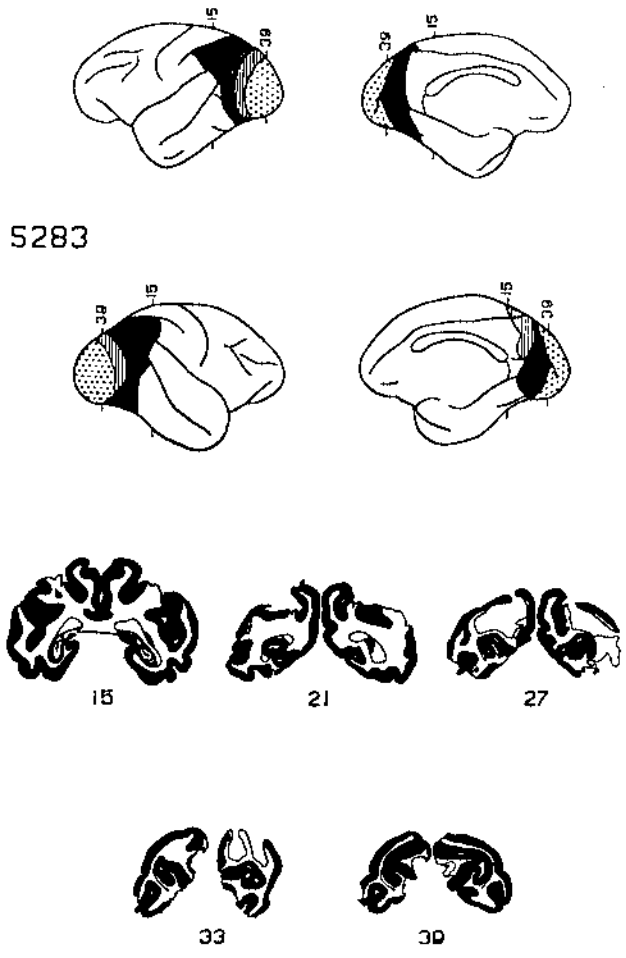


FIGURE 4 Reconstruction of bilateral prestriate lesions after which monkey could still perform a visual discrimination (the numerals 3 versus 8) at 90% criterion.

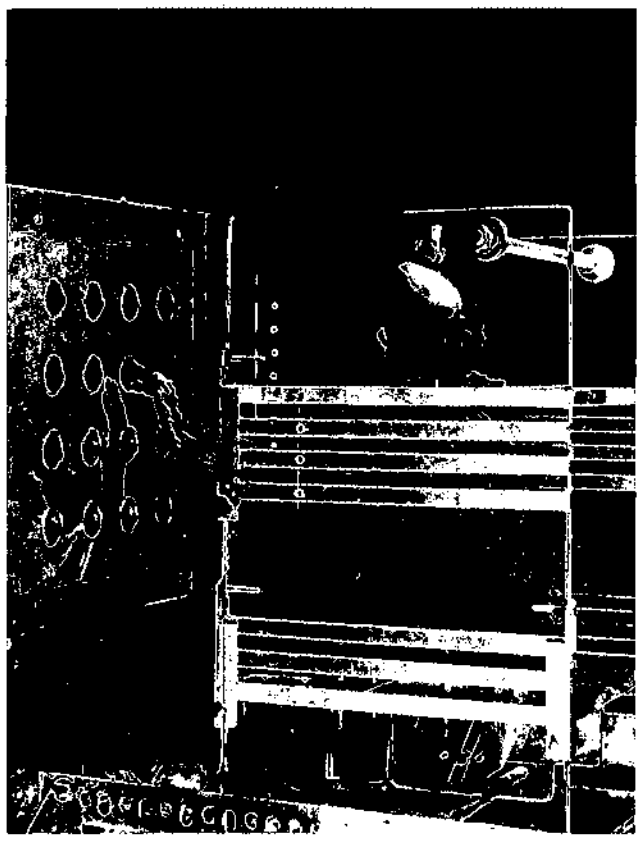


FIGURE 5 Monkey performing in Discrimination Apparatus for Discrete Trial Analysis (DADTA). A general purpose computer (PDP-8) programs stimulus presentation, records behavioral and electrophysiological results on magnetic tape, and provides typed or oscilloscope display readouts. Simple collations of data are performed on-line. More complex analyses are performed on taped data store.

in a current series of monkeys I have replicated Gross and Cowey's results exactly; these results are that the monkeys do have difficulty in relearning the discrimination after surgery (recall that even the best monkey whose lesion I demonstrated was essentially blind for weeks and had to be slowly retrained to respond visually). However, all monkeys do relearn and therefore the foveal circumstriate cortex cannot be, by itself, essential to the recognition process. With respect to the total lesions, Weiskrantz has suggested during our sessions here, that my technique (Figure 5) of randomizing the position of cues over 16 vertically presented locations aids recovery; Mishkin used a horizontal two-choice situation (Figure 6) for testing. This technique maximizes the disturbance because a ventral hemianopia is almost invariably produced in resecting the circumstriate belt by interruption of the dorsal part of the geniculostriate radiations that lie close to the surface just under the circumstriate cortex. Such interruption of geniculostriate radiation does not by itself produce any visual discrimination deficit (Wilson, 1957), but the combination of striate and peristriate removals might. Weiskrantz is now testing this interpretation in his laboratory at Oxford.

Another possibility remains: That the functions of the circumstriate belt are more widely distributed and that the functions of the cortex on the inferior convolutions of the temporal lobe overlap those of the circumstriate system. Our discovery of the visual functions of this temporal lobe cortex, in fact, included the entire extent of circumstriate preoccipital-temporal cortex (Blum, Chow, and Pribram, 1950). Only later did we find that the circumstriate portion of the lesion was dispensable in producing the effect on visual discrimination.

With regard to the temporal lobe cortex, another major discrepancy needs to be resolved. Charles Gross (1969) has elegantly demonstrated that the visual receptive field characteristics of cells in the inferior temporal gyrus are dependent both on the presence of the ipsilateral striate cortex and on an input from the thalamus (pulvinar). This contrasts sharply with the facts

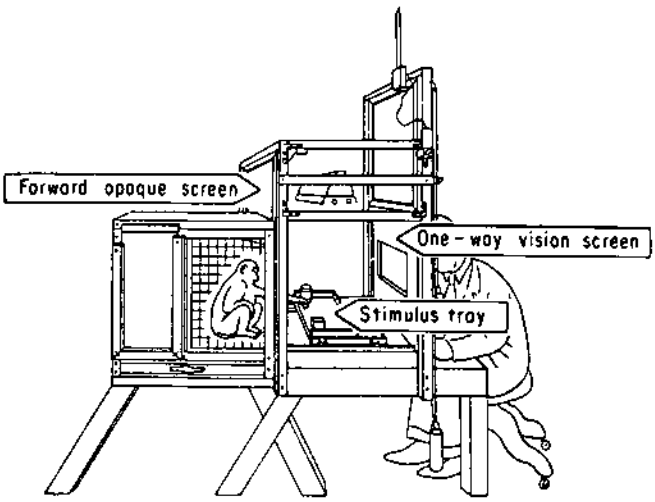


FIGURE 6 Wisconsin General Testing Apparatus (WGTA). Used to test monkeys in a variety of discrimination and learning problems.

I have just presented that radical resections of the cortico-cortical connections between striate and temporal cortex only temporarily impair visual recognition and Mishkin's recent experiments (35 monkeys) that visual recognition remains intact after massive posterior thalamic lesions that destroy the entire pulvinar (unpublished, Mishkin).

I would like to suggest that this discrepancy is also amenable to possible resolution by experiment. Some years ago Rosenblith, Rosner, and I (Pribram et al., 1954) showed that the responses evoked in Auditory Area III were dependent on the presence of the medial geniculate nucleus and not on connections from Auditory Areas I and II. In acute experiments, removal of Auditory Areas I and II did not alter the responses evoked in Auditory III, but when, after surgical removals in chronic preparations, time for degeneration of the medial geniculate nucleus was allowed, the responses disappeared. We inferred that a collateral projection from the medial geniculate nucleus to Auditory III could account for the results. A parallel experiment could be tried to see whether the receptive field of Gross's cells in the temporal cortex would be affected in acute experiments (his data so far are based on chronic preparations in which the lateral geniculate nucleus has degenerated). Perhaps, as for Auditory III, the receptive field characteristics of cells in the inferior temporal cortex are dependent on a booster from direct or indirect collaterals from the geniculate nucleus.

Where then does all this evidence leave us with regard to the problem of the mechanism of assembly of a recognition mechanism? My view is that these data unequivocally tell us what cannot be, but they leave us in open ignorance as to what actually does constitute the assembly mechanism. The mechanism, and I must emphasize again that this is my opinion based on my data, cannot be solely a transcortical hierarchical process. My response to the experimental results has been to emphasize an alternative to a simple transcortical reflex model: Over the years, I have suggested that a corticofugal efferent control system emanates from the temporal cortex downward to subcortical structures, there to influence by a parallel processing mechanism the visual input (Pribram, 1958, 1960, 1969, 1971). (Arbib has facetiously made the point that just because the brain looks like a bowl of porridge does not mean that it is a serial [cereal] computer.)

Functions of the program

Let us therefore look briefly at some of the electrophysiological data that provide evidence for the existence of such a parallel processing efferent system and how it functions before attempting a synthesis. In one of these experiments, we stimulated the inferior temporal cortex of cats to determine the effect on visual receptive field organization. We found changes occurring as far peripheral as the optic nerve, but the most cleancut and systematic effects were shown at the lateral geniculate level (Figure 7). Note the marked shrinking of the excitatory center and expansion of the inhibitory surround produced in this unit. (Note also that frontal lobe stimulation has an opposite effect

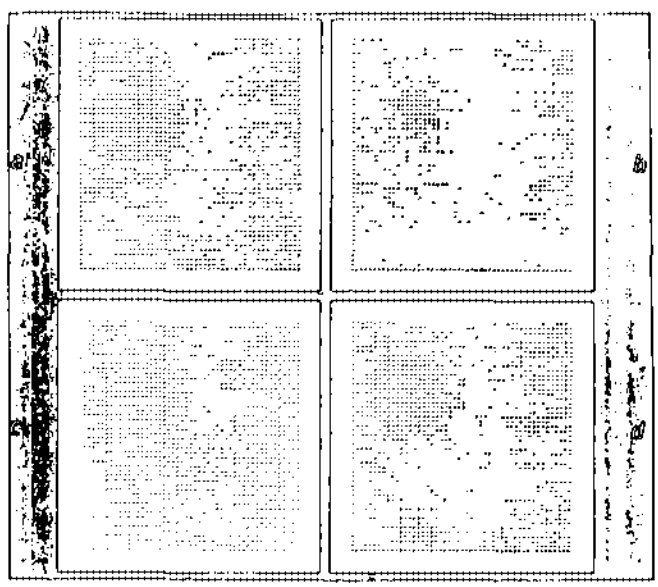


FIGURE 7 Visual receptive field maps show how information flowing through the primary visual pathway is altered by stimulation elsewhere in the brain. Map a is the normal response of a cell in the geniculate nucleus when a light source is moved through a raster-like pattern. Map b shows how the field is contracted by stimulation of the inferior temporal cortex. Map c shows the expansion produced by stimulation of the frontal cortex. Map d is a final control taken 55 min after recording a.

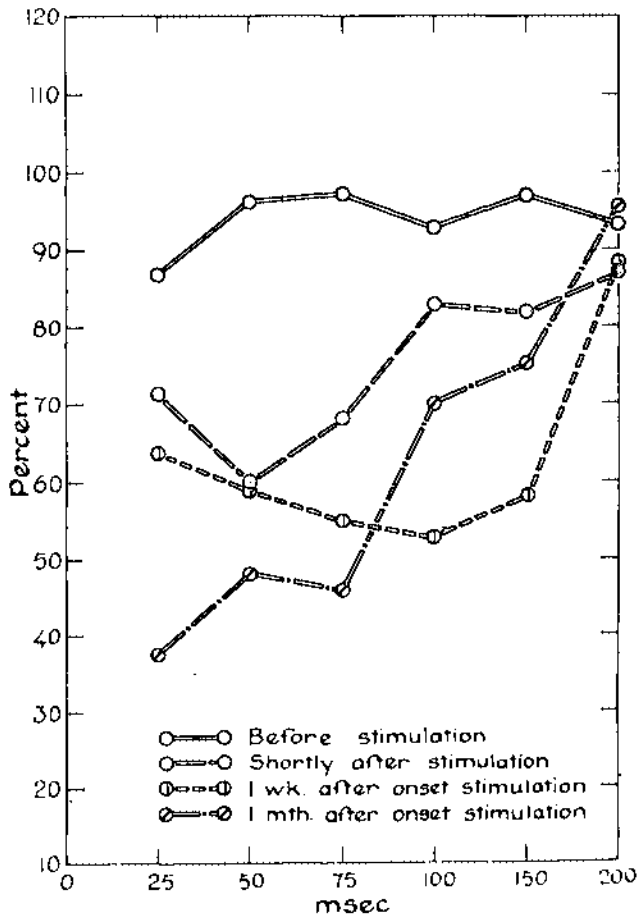


FIGURE 8 A plot of the recovery functions obtained in one monkey before and during chronic stimulation of the inferotemporal (IT) cortex.

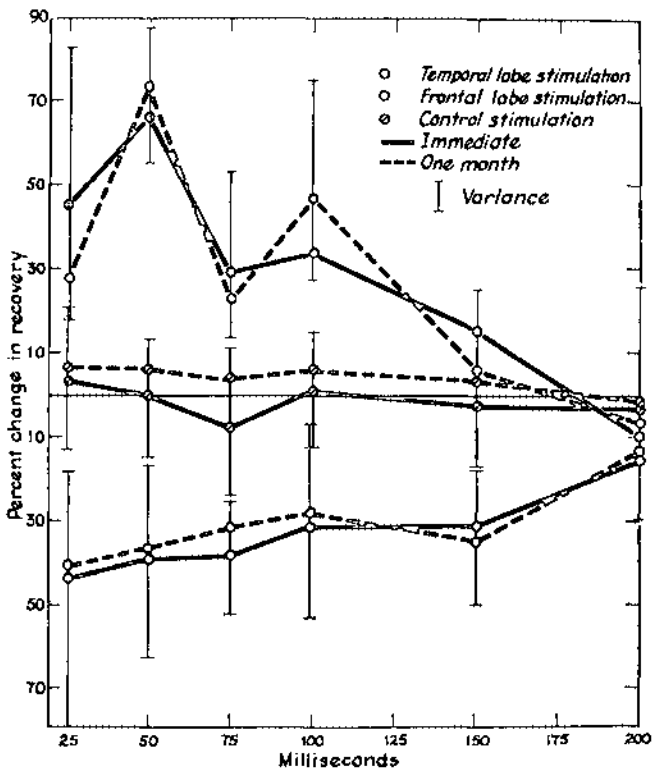


FIGURE 9 This figure plots the percent change in recovery for all subjects in the various experiments. It is thus a summary statement of the findings.

and that parietal and precentral stimulation has no effect on the receptive field organization of these cells.)

In another experiment we demonstrated changes in recovery cycles recorded from the striate cortex of fully awake monkeys sitting in restraining chairs (Figures 8 and 9). Electrical stimulation of inferior temporal lobe cortex shortens the recovery cycle of the response evoked in striate cortex by brief flashes of light (whereas frontal cortex stimulation lengthens it and no effect is produced by parietal and precentral stimulation). We interpreted this effect on recovery as indicating reduction (or, in the case of frontal stimulation, on enhancement) in redundancy of the visual channel. It is interesting to note that Waterman, collaborating with Wiersma (1966), found a similar redundancy control mechanism in invertebrates.

We quickly found that this effect could be demonstrated only when the monkeys were not attending to some other aspect of their environment. We therefore designed experiments to test specifically the relationship between the functions of the inferotemporal cortex and attention

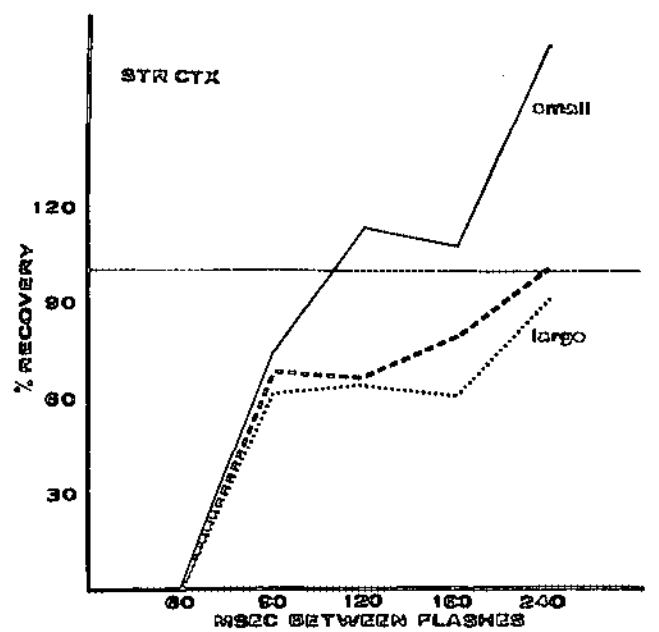


FIGURE 10 Comparison of flash recovery functions obtained when the probe stimulation of the lateral geniculate nucleus results in small (solid line) or large (dotted line) striate cortex response. Control without probe stimulation is indicated by dashed line. Note that when the probe stimulation produces a small response (solid line), i.e., when the monkey is attending, recovery is speeded. This same effect is obtained with temporal lobe (IT cortex) stimulation.

and showed in fact that attentional factors were critical (Figure 10; Gerbrandt, Spinelli, and Pribram, 1970). Gross (1972) also has shown that monkeys must be attending his experiment if he is to obtain unit responses in this cortex. But probably the most clear-cut demonstration of the process involved comes from one such experiment in which monkeys were trained to discriminate between flashed (10 μsec) cues that varied in two dimensions: color and form (Rothblat and Pribram, 1972). The monkeys were first trained to respond to red by differentially reinforcing a response to the color dimension. They were then subjected to a discrimination reversal procedure, green was now the rewarded cue. Next, responses to the stripes were differentially reinforced and finally circles became the rewarded cue. In each stage the electrical activity from inferior temporal, circumstriate, and striate cortex was recorded for three days of criterion performance, 90% and 100 consecutive trials (Figures 11 and 12). Note the pattern of electrical responses evoked in the inferior temporal cortex when the monkey selectively responds to color and the different pattern when he responds to form. These evoked potentials are discernible only when the records are correlated to the time of response; in this respect (and several others)

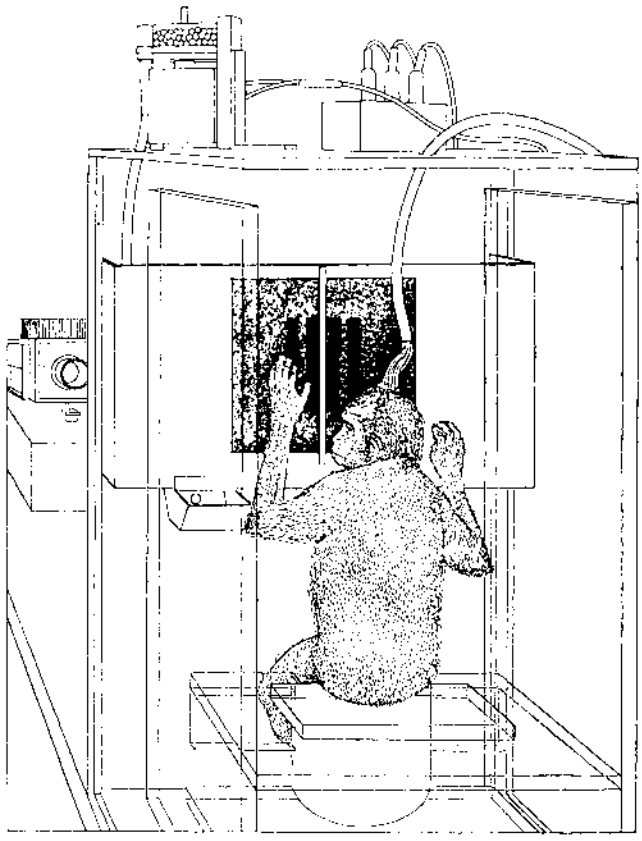


FIGURE 11 Set-up of an experiment demonstrating the functions of the visual areas. A monkey initiates a flashed stimulus display and responds by pressing either the right or left half of the display panel to receive a reward while electrical brain recordings are made on line with a small general purpose computer (PDP-8). On the translucent panel in front of him the monkey sees either a circle or a series of vertical stripes, which have been projected for 0.1 msec from the rear. He is rewarded with a peanut, which drops into the receptacle at his left elbow, if he presses the right-half of the panel when he sees the circle or the left-half when he sees the stripes. Electrodes record the wave forms that appear in the monkey's visual cortex as he develops skill at this task. Early in the experiments, the stimulus-locked wave forms show whether the monkey sees the circle or stripes. Eventually they reveal in advance which half of the panel the monkey will press. Each trace sums 300 trials of 500 msec of electrical activity following the stimulus flash.

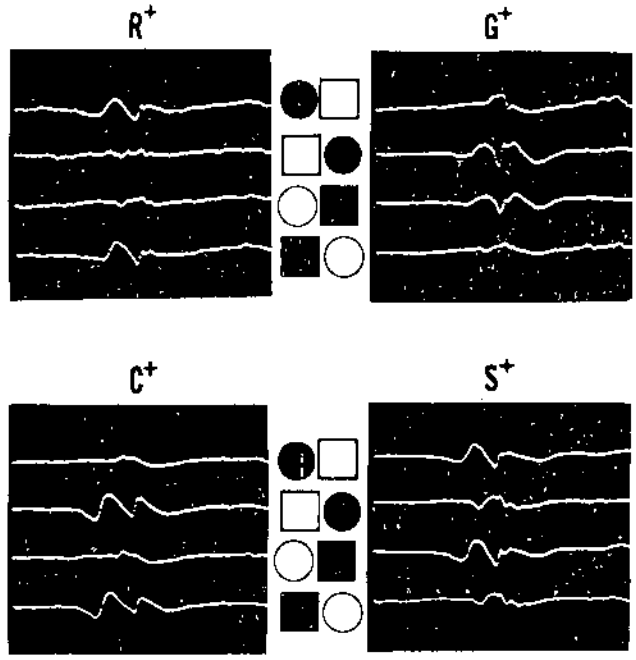


FIGURE 12 Results of an experiment demonstrating the functions of the inferotemporal cortex using a set-up similar to that shown in Figure 11. Comparison of *response-locked* activity evoked in temporal cortex (IT) when monkeys are performing (90% correct) color (top panels) and pattern (bottom panels) discrimination. Each tracing sums, over 300 consecutive trials, the activity recorded when the stimulus configuration presented to the monkey appeared as in the diagrams between the panels. Each tracing includes 500 msec of electrical activity −250 prior to and 250 just after each response. Note that during the color discriminations the 1st and 4th (and the 2nd and 3rd) traces are similar, while during the pattern discriminations the 1st and 3rd (and 2nd and 4th) traces are alike. These similarities reflect the position of the color cues in the color task and the position of the patterns in the pattern task. Position per se, however, is not encoded in these traces. Note that this difference occurs despite the fact that the retinal image formed by the flashed stimulus is identical in the pattern and color problems.

they are different from the stimulus-locked evoked potentials in the striate cortex I showed earlier.

Perhaps the most interesting finding, however, came when we traced the emergence of the evoked response differences as a function of changing the reward from one cue dimension to the other (Figure 13). Note in the lower panel of tracings that the left-most and right-most patterns are almost identical to those I showed on the last slide. These are from a different electrode in a different monkey, however. Note now what happens while the animal shifts from selectively responding to (attending) the color dimension to responding to (attending) the form dimension. While the monkey is performing at chance, the evoked electrical activity shows no regularity. When he performs at about 75% his temporal lobe activity begins to take on the pattern related to the form discrimination. This pattern becomes enhanced at criterion performance and now, for the first time, appears also in the record made from the striate cortex. With overtraining, the striate cortex record becomes almost as striking as that obtained from the temporal cortex.

Conclusion

This experimental result, more than any other single finding, has led to my conviction that, for pattern per-

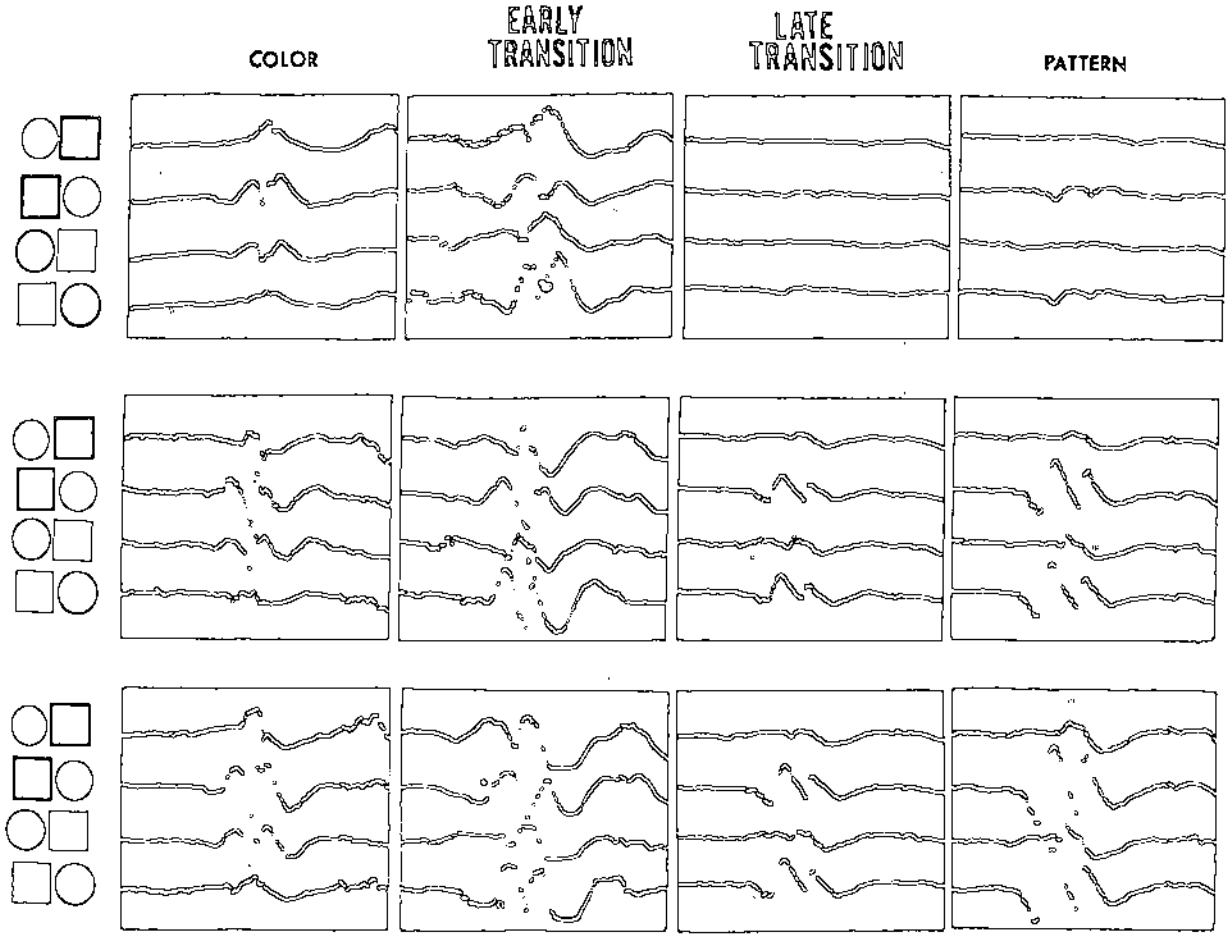


FIGURE 13 Experiment shows the development of the response-locked activity. In this experiment the flashed stimulus again consisted of colored (red and green) stripes and circles, exactly as in Figure 12. Reinforcing contingencies determined whether the monkeys were to attend and respond to the color (red versus green) or pattern (circle versus stripes) dimension of the stimulus. As in the earlier experiment, shown in Figure 11, stimulus, response, and reinforcement variables were found to be encoded in the primary visual cortex. In addition, this experiment showed that the association between stimulus dimension (pattern or color) and response shown in Figure 12 occurs first in the inferotemporal cortex. This is shown in the lower panels where the electrophysiological data averaged (summed) from the time of response (forward for 250 msec and backward 250 msec from center of record) again show clear differences in wave form depending on whether pattern or color is being reinforced. Note that in these tracings the response-locked difference in recorded activity can already be seen in the temporal lobe recording when the monkey is performing at 75% correct but does not appear in the striate cortex recording until criterion performance is attained. Overtraining enhances this difference in the striate cortex recording.

ception to occur, a program tape must become assembled to address the input when called for. It really looks as if the activity of the inferior temporal cortex, having become organized by the reinforcing contingencies of the situation, throws a programmed filter or program tape into the visual system that thereupon addresses and organizes (categorizes) the electrical responses, and presumably the image elements of the striate cortex. The assembling of such a program tape involves a great amount of processing; no wonder that sensing so much, we can do so little.

It is, of course, imperative to know the pathways by which such an assembling of a program tape can occur, and I remind you of Graybiel's Figure 6 showing the three visual systems and their corticocortical interactions. Recall that the three systems are distinguished on the basis of their afferent connections. However, the question remains whether any corticocortical organization supports the trichotomy: The effects of lesions of the various visual systems are completely dissociable from those of the premotor, frontal, and limbic systems—as are the effects of each of these systems from those of any of the others.

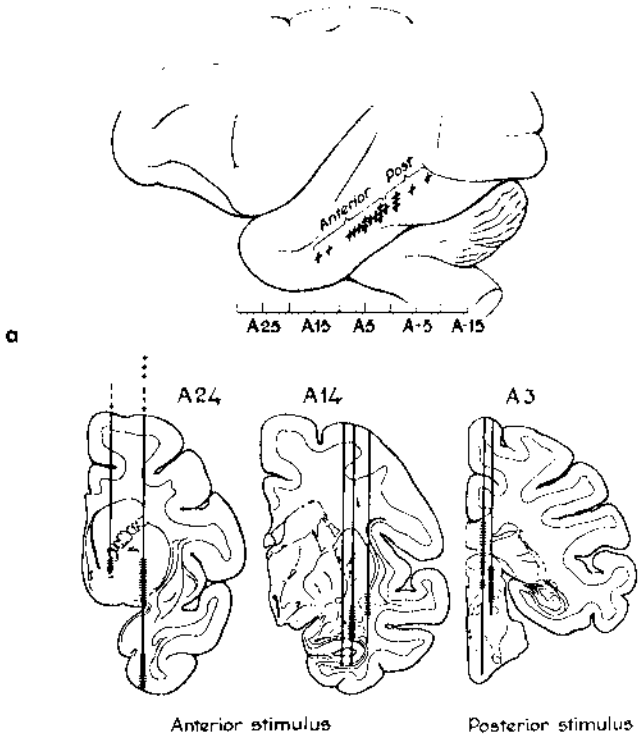


FIGURE 14 (a) Side view of the brain showing stimulation sites in experiment that traced the subcortical connections of the inferotemporal cortex. (b) Selected cross-section showing sites (‡) where response was evoked by inferotemporal cortex stimulation. Note especially the responses in putamen and superior colliculus.

No such dissociation occurs when the effects of lesions of these areas are compared with those produced in subcortical structures to which efferents project. For instance, the behavioral deficit that follows frontal cortex resection can be produced as well from lesions of the head of the caudate nucleus. And lesions deep to the inferotemporal cortex in the region of the tail of the caudate nucleus and ventral putamen (Rosvold and Szwarcbart, 1964) produce deficits in visual discrimination. Thus, we should have been alerted (but were still surprised by the size and extent) when we obtained strong evidence of efferent connections to the entire ventral putamen by mapping the electrical responses evoked by stimulation of the inferotemporal cortex. (See Figures 14 and 15.) Here were powerful connections from association cortex to a nucleus usually identified with the motor system (Reitz and Pribram, 1969).

However, as stated by Kornhuber and Ito and others elsewhere in this volume, the motor functions of the nonpyramidal motor systems function in large part as organizers of programs controlling more reflex levels of function. Some of this control is exerted via the γ system and is thus receptor, i.e., input, control. A fascinating task ahead is to determine experimentally whether the basal ganglia influence the input from the special senses as well as those of the motor systems—and if so, just where and how.

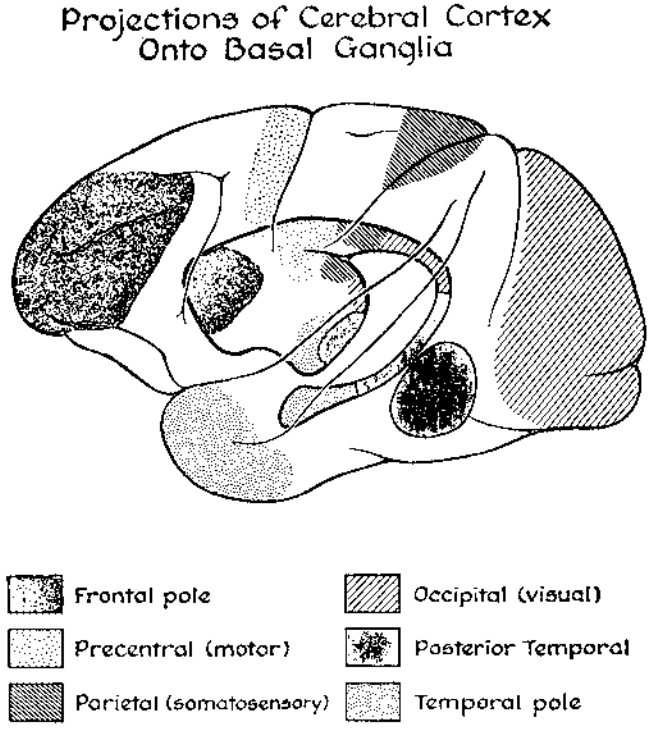


FIGURE 15 Corticofugal connections to the basal ganglia. (Drawn from study by Kemp and Powell, 1970.)

The model developed in these pages reads, therefore: Input becomes distributed in sensory systems through the action of lateral inhibitory networks into an *alphabet* of spatial frequency sensitive elements at the striate cortex by a more or less invertable transformation. This alphabet becomes temporarily assembled for the purposes of any specific recognition, not by some hierarchical process leading to a pontifical "grandfather" neuron in the circumstriate cortex. Rather, a parallel processing mechanism initiated in the inferotemporal cortex addresses (categorizes) the elements of the alphabet via motor structures (e.g., the putamen) much as a program tape organizes a program by addressing elements in the memory of a computer. The model thus constitutes a progressively differentiating self-organizing system. We have seen that, as a heuristic, the model has had considerable merit. A great amount of otherwise conflicting data are subsumed and perhaps more important, five *areas of ignorance* can now be detailed sufficiently to generate specific experiments. What more can we ask when just a few years ago there was only enigma?

REFERENCES

BENEVENTO, L. A., O. D. CREUTZFELDT, and U. KUHNT, 1973. Significance of intracortical inhibition in the visual cortex. *Nature* (*London*), (in press).

BLUM, J. S., K. L. CHOW, and K. H. PRIBRAM, 1950. A behavioral analysis of the organization of the parieto-temporo-preoccipital cortex. *J. Comp. Neurol.* 93:53–100.

CHOW, K. L., 1970. Integrative functions of the thalamocortical visual system of cat. In *Biology of Memory*, K. H. Pribram and D. Broadbent, eds. New York: Academic Press, pp. 273–292.

CREUTZFELDT, O. D., 1961. General physiology of cortical neurons and neuronal information in the visual system. In *Brain and Behavior*, M. A. Brazier, ed. Washington, D.C.: American Institute of Biological Sciences, pp. 299–358.

DEVALOIS, R. L., 1960. Color vision mechanisms in monkey. *J. Gen. Physiol.* 43:115–128.

GABOR, D., 1969. Information with coherent light. *Opt. Acta* (*London*) 16:519–533.

GALAMBOS, R., T. T. NORTON, and C. P. FROMMER, 1967. Optic tract lesions sparing pattern vision in cats. *Exp. Neurol.* 18: 8–25.

GERBRANDT, L. K., D. N. SPINELLI, and K. H. PRIBRAM, 1970. The interaction of visual attention and temporal cortex stimulation on electrical activity evoked in the striate cortex. *Electroenceph. Clin. Neurophysiol.* 29:146–155.

GROSS, C. G., D. B. BENDER, and C. E. ROCHA-MIRANDA, 1969. Visual receptive fields of neurons in inferotemporal cortex of the monkey. *Science* 166:1303–1305.

GROSS, C. G., A. COWEY, and F. J. MANNING, 1971. Further analysis of visual discrimination deficits following foveal prestriate and inferotemporal lesions in rhesus monkeys. *J. Comp. Physio. Psychol.* 76:1–7.

GROSS, C. G., C. E. ROCHA-MIRANDA, and D. B. BENDER, 1972. Visual properties of neurons in inferotemporal cortex of the macaque. *J. Neurophysiol.* 35:96–111.

HENRY, G. H., and P. O. BISHOP, 1971. Simple cells of the striate cortex. In *Contributions to Sensory Physiology*, W. D. Neff, ed. New York: Academic Press.

HUBEL, D. H., and T. N. WIESEL, 1968. Receptive fields and functional architecture of monkey striate cortex. *J. Physiol.* 195:215–243.

JUNG, R., 1961. Neuronal integration in the visual cortex and its significance for visual information. In *Sensory Communication*, W. A. Rosenblith, ed. New York: John Wiley & Sons, pp. 627–674.

KLÜVER, H., 1941. Visual functions after removal of the occipital lobes. *J. Psychol.* 11:23–45.

LASHLEY, K. S., 1929. *Brain Mechanisms and Intelligence*. Chicago: University of Chicago Press.

MINSKY, M. L., and S. PAPERT, 1969. *Perceptrons*. Cambridge: MIT Press.

MISHKIN, M., 1966. Visual mechanisms beyond the striate cortex. In *Frontiers of Physiological Psychology*, R. Russell, ed. New York: Academic Press, pp. 93–119.

PHELPS, R. W., 1972. Inhibitory interactions in the visual cortex of the cat. Ph.D. Thesis, Stanford University, California.

POWELL, T. P. S., and V. B. MOUNTCASTLE, 1959. Some aspects of the functional organization of the cortex of the postcentral gyrus of the monkey: A correlation of findings obtained in a single unit analysis with cytoarchitecture. *Bull. Johns Hopkins Hosp.* 105:133.

PRIBRAM, K. H., 1954. Toward a science of neuropsychology. In *Current Trends in Psychology and the Behavioral Sciences*, R. A. Patton, ed. Pittsburgh: University of Pittsburgh Press, pp. 115–142.

PRIBRAM, K. H., 1958. Neocortical function in behavior. In *Biological and Biochemical Bases of Behavior*, H. F. Harlow and C. N. Woolsey, eds. Madison: University of Wisconsin Press, pp. 151–172.

PRIBRAM, K. H., 1960. The intrinsic systems of the forebrain. In *Handbook of Physiology, Neurophysiology II*, J. Field, H. W. Magoun, and V. E. Hall, eds. Washington: American Physiological Society, pp. 1323–1344.

PRIBRAM, K. H., 1966. Some dimensions of remembering: steps toward a neuropsychological model of memory. In *Macromolecules and Behavior*, J. Gaito, ed. New York: Academic Press, pp. 165–187.

PRIBRAM, K. H., 1969. The amnestic syndromes: Disturbances in coding? In *Pathology of Memory*, G. A. Talland and N. C. Waugh, eds. New York: Academic Press, pp. 127–157.

PRIBRAM, K. H., 1971. *Languages of the Brain*. Englewood Cliffs: Prentice-Hall.

PRIBRAM, K. H., 1972. Some dimensions of remembering: Steps toward a neuropsychological model of memory. In *Macromolecules and Behavior*, 2nd Ed., J. Gaito, ed. New York: Appleton-Century-Crofts, pp. 367–393.

PRIBRAM, K. H., M. NUWER, and R. BARRON, 1973. The holographic hypothesis of memory structure in brain function and perception. In *Contemporary Developments in Mathematical Psychology* (in press).

PRIBRAM, K. H., B. S. ROSNER, and W. A. ROSENBLITH, 1954. Electrical responses to acoustic clicks in monkey: Extent of neocortex activated. *J. Neurophysiol.* 17:336–344.

PRIBRAM, K. H., D. N. SPINELLI, and M. C. KAMBACK, 1967. Electrocortical correlates of stimulus response and reinforcement. *Science* 157:3784.

REITZ, S. L., and K. H. PRIBRAM, 1969. Some subcortical con-

nections of the inferotemporal gyrus of monkey. *Exp. Neurol.* 25:632–645.

ROSVOLD, H., and M. K. SZWARCBART, 1964. Neural structures involved in delayed-response performance. In *The Frontal Granular Cortex and Behavior*, J. M. Warren and K. Akert, eds. New York: McGraw-Hill, pp. 1–15.

ROTHBLAT, L., and K. H. PRIBRAM, 1972. Selective attention: Input filter or response selection? *Brain Res.* 39:427–436.

SPINELLI, D. N., 1966. Visual receptive fields in the cat's retina: Complications. *Science* 152:1768–1769.

SPINELLI, D. N., K. H. PRIBRAM, and B. BRIDGEMAN, 1970. Visual receptive field organization of single units in the visual cortex of monkey. *Int. J. Neurosci.* 1:67–74.

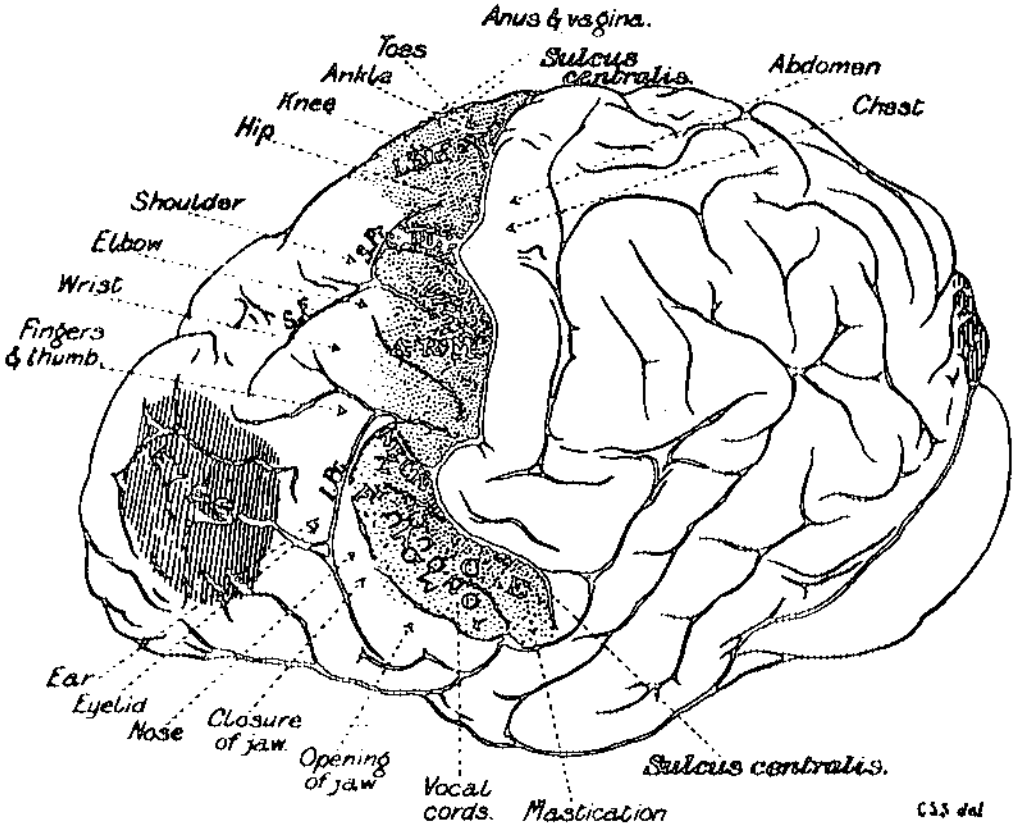
SPINELLI, D. N., A. STARR, and T. W. BARRETT, 1968. Auditory specificity in unit recordings from cat's visual cortex. *Exp. Neurol.* 22:75–84.

WATERMAN, T. H., 1966. Systems analysis and the visual orientation of animals. *Amer. Sci.* 54:15–45.

WERNER, G., 1970. The topology of the body representation in the somatic afferent pathway. In *The Neurosciences, Second Study Program*, F. O. Schmitt, Editor-in-Chief. New York: Rockefeller University Press, Vol. 2, pp. 605–616.

WHITMAN, R., and R. SPITZBERG, 1972. Spatial-frequency channels: Many or few? Paper presented at the Annual Meeting of the Optical Society of America, 1972.

WILSON, M., 1957. Effects of circumscribed cortical lesions upon somesthetic discrimination in the monkey. *J. Comp. Physiol. Psychol.* 50:630–635.


Anus & vagina.
Toes
Ankle
Knee
Hip
Sulcus centralis.
Abdomen
Chest
Shoulder
Elbow
Wrist
Fingers & thumb.
Ear
Eyelid
Nose
Closure of jaw.
Opening of jaw
Vocal cords.
Mastication
Sulcus centralis.
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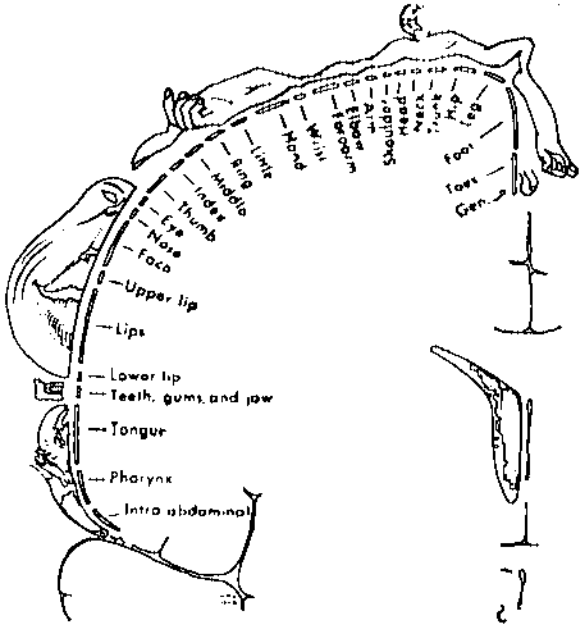
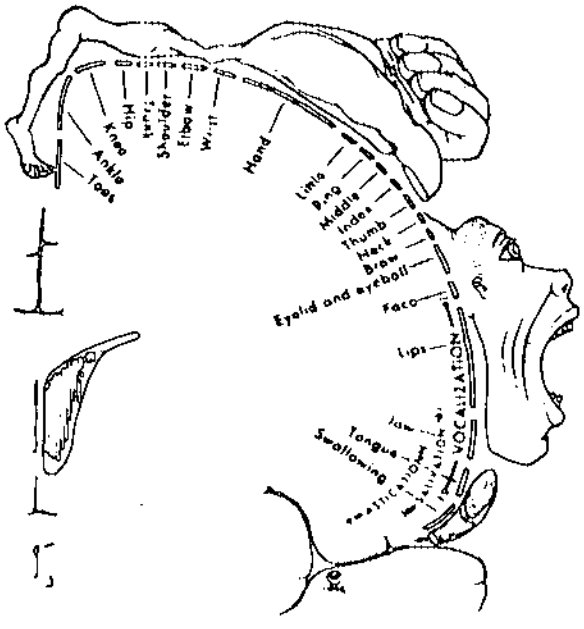

Upper lip
Lips
Lower lip
Teeth, gums and jaw
Tongue
Pharynx
Intra abdominal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Swallowing
VOCALIZATION