T-179

Attention and Para-Attentional Processing

Event-Related Brain Potentials as Tests of a Model

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INTRODUCTION

Event-related electrical brain potentials are unique in providing a "window" or "lens" with a resolving power of milliseconds through which input processing can be assessed. They are therefore ideal in tracking the rapid sequence of brain responses that immediately occur when a sensory input is processed. These responses make up the orienting reaction. Those components of orienting which are accessible to awareness are "attended;" the remaining components comprise para-attentional processes.

The aim of the review is to relate, to the extent possible, the operations of various brain systems to the attentional and para-attentional components of orienting. With repetition of an input pattern there is a shift from attentional to para-attentional processing: processing which is automatic but directly influences attention. The shift is called habituation, the pattern becomes familiar. With any change of input pattern or the context in which it occurs, the orienting reaction recurs, it is dishabituated. Dishabituation reflects the response to the novel configuration which has been produced by the change.

The processes of habituation and dishabituation are disturbed when certain parts of the brain are resected. The disturbances are selective; some of the components of orienting are vulnerable to one site of brain resection, other components to other sites. It is therefore necessary to review first the evidence which furnishes the basis for a component analysis of orienting. This evidence comes mainly from psychophysiology, recordings of visceroautonomic indicators of orienting. Next, the relationships between these indicators and brain systems are reviewed. The relationships are established by neurobehavioral and neurochemical studies.

These psychophysiological, neurobehavioral and neurochemical analyses yield a model of orienting which is "tested" below. The tests consist of relating brain electrophysiological evidence to the model. This evidence allows specification of the neural processes involved in the various phases of orienting. In addition, the model is updated with respect to the delineation of an extralemniscal processing system involved in targeted awareness. Thus, a lemniscal automatic para-attentional process becomes defined upon which a set of generalized and targeted attentional control processes operate in a top-down fashion.

Historical Overview

Initially, behavioral and physiological manifestations of central processing of input—attention—led neuroscientists to the view that attention could be ordered along a quantitative continuum; an organism is more or less "aroused" or "activated" and that this occurs because of the way certain brain systems are functioning.

In early research one source of activation or arousal (terms at that time used synonymously) was found in the mesencephalic reticular activating system of the brain stems. Lesions of this network of fibers led to somnolence, coma or even death (Moruzzi & Magoun, 1949) while stimulation of the same system produced alertness and behavioral activity (Lindsley, 1961). Further research produced similar results following electrical stimulation of the hypothalamus of the diencephalon (Abrahams, Hilton & Zbrozna, 1964) and also from the amygdala of the forebrain (Gastaut, 1954). A continuum of behavior from orienting to rage and attack occurred as the amount of stimulation increased. Certain peripheral physiological indices such as the Galvanic skin response (GSR) and heart rate were found to correspond to these levels of arousal. The question fundamental to this line of inquiry was the *degree* or amount to which an organism is aroused. Research of this sort asks questions such as: How *much* attention can be paid?; How *long* can attention be paid?; and can attention be *maintained* in the face of distraction?

In experimental psychology the focus has been different. It has been accepted that, in general, animals and humans attend to something. If they did not, no experiments could be carried out. Interest was generated in the study of attention only when it appeared to break down. Animals were found to notice some features of a compound stimulus but not others, such as color but not shape. Humans and animals alike behaved as if their processing capabilities were limited. They produced responses that indicated they had not processed all the available sensory input.

Thus the initial question arose, spearheaded by Broadbent's classic text in 1958: Are the limits on attention due to filters on the input side, or because of limitation on the organization of behavior? In effect this asked: *Where* is the bottleneck?

Bottleneck models carry the implicit assumption that the brain is indeed like a bottle and that input from the environment is the substance that flows into it, always in one direction. The question "where is the bottleneck?" has never been answered satisfactorily for the simple reason that the brain is not built like a bottle. In a sophisticated review of the problem, Erdelyi (1974) concludes on the basis of 20 years of data, that the limits on processing (selection) are "ubiquitous throughout the nervous system" and need not occur with conscious awareness. In other words, there are a multitude of overlapping systems, some parallel in operations (a wide bottleneck—or several bottles), some sequential (narrow bottleneck), and those intermediate between them.

A beginning was made toward a neuropsychological analysis of this problem when we realized that the issues raised by the hypothesis of a bottleneck of limited capacity might be more productively phrased in terms of a central *competency* (Pribram, 1971, 1974, 1976).

Competencies can be multiple, both on the input and output sides, and the ultimate capability, rather than being conceived of as the capacity of a box of finite limits, can be better construed as a flexible matrix of interlocking competencies. Evidence has confirmed that with extensive practice a formerly limited "capacity"

becomes less and less restricted (Logan, 1979; Hirst, Spelke, Reaves, Caharack & Neisser, 1980).

Earlier studies reviewed by Garner (1962) demonstrate that with appropriate experimental designs almost unlimited processing is revealed. For instance, Anderson and Fitts (1958) showed that subjects could handle 17 alternative bits of information at one time. This represented stimulus parameters of color, shape and location, each with nine alternatives, $9 \times 9 \times 9$ or 729 differentiable signals! One of the mechanisms by which such large amounts of information can be processed is by grouping or chunking the bits into larger categories (Miller, 1956; Simon, 1974). In contrast to the rigid external structure implied by bottleneck of limited capacity models, the evidence on chunking shows that an endoskeleton, an internal structure, can be formed which determines the competence of a processing channel.

Focus on the competence to *organize* information has led to the second question asked by experimental psychologists, which is, in effect: "What is the nature of selectivity in processing?" This is the question that lies at the heart of research on attention *span* which deals with such issues as how much can we attend to at any one time, and what kind of stimuli can be attended to more readily than others? The two questions are intimately interwoven because it has become clear that "what kind" determines "how much."

Problems arise with questions of this type because they move us away from the study of attention as a simple function toward a study of attention as a process based on structure. Because of this, research on attention has come to resemble research on cognitive efficiency. We can attend more readily to stimuli that are comprehensible, or have become so through learning or practice.

The model of attention described in this essay is based on data that go a long way toward integrating the neurophysiological and psychological traditions. The current model extends our earlier model (Pribram and McGuinness 1975) by incorporating neurochemical and neurophysiological data that have accumulated since its initial publication. Furthermore, data are here presented according to technique: this should allow for an easier disciplinary evaluation of the model.

Outline of the Model

A considerable body of evidence is accruing to the effect that the central processing of sensory input proceeds automatically under certain circumstances and more deliberately under others. Posner (1973) has data which indicate that automatic processing proceeds by virtue of activity of the "extrinsic" sensory-motor projection systems. Controlled processing entails activity of the "intrinsic" sensory associated system not only of the frontal but also of the posterior cortical convexity (Bolster & Pribram, in preparation). While automatic processing is para-attentional and in large part parallel in nature, controlled processing involves steps, the serial engagement of several attentional systems which range from orienting to effortful search.

Originally, three classes of neural systems were discerned to involve the control of orienting and the major portion of this review will be devoted to these systems. However, in the section on event-related electrical brain potentials, an additional system will be described, which details the basic automatic process upon which the three control systems are shown to operate.

Initially, the three classes of attentional control systems were identified as dealing with (1) "arousal," (2) "activation," and (3) "effort," but these terms of

themselves are not of prime importance. As evidence accrued each of these forms was shown to signify one pole of a dimension. Thus, arousal became paired with familiarization and activation with targeted readiness, and effort was better represented by a comfort-effortful innovation dimension. What is important to retain is that operational definitions of the three dimensions are available and thus the concepts underlying the classification can be subjected to further test.

The defining operations upon which the classification was based center on the orienting reaction. Orienting *per se* was shown (*e.g.*, Sharpless & Jasper, 1956) to consist of two components: a brief reflexive response signalled by psychophysiological indicators and a somewhat more prolonged reaction signalled by behavioral orienting. Furthermore, with repetition of the stimulus, the orienting reaction ordinarily decrements—this is called habituation. The brief reflexive portion of the orienting reaction habituates rapidly while the more selective, target behavioral portion habituates slowly or not at all.

Habituation of the reflex component of the orienting reaction is impaired when the orbitofrontal cortex or the temporal pole including the amygdala are damaged (Kimble, Bagshaw & Pribram, 1965; Bagshaw, Kimble & Pribram, 1956; Bagshaw & Benzies, 1968; Luria, Pribram, & Homskaya, 1964, Grueninger & Grueninger, 1973; Pribram, Reitz, McNeil & Spevack, 1979). Such lesions result in a total absence of the ordinarily present viscerautonomic components of the orienting reaction, and we have suggested that there may be a causal relationship between the occurrence of visceroautonomic responses and the production of habituation. In the absence of visceroautonomic activity the orienting stimulus fails to become familiar with the result that behavioral orienting to the same stimulus continues unabated.

Whereas the behavioral component of the orienting reaction is resistant to orbitofrontal and amygdala lesions, this component is impaired when the nigrostriatal (basal ganglia) system, the cingulate and the cortical convexity become damaged (Heilman & Valenstein, 1972; Wright, 1979, 1980a, 1980b). Such damage leads to "neglect" of the stimulus, a failure to orient within the sensory field affected by the damage, especially when the system is put out of balance by unilateral lesions. In such instances the neglect is limited to the sensory field contralateral to the lesion.

The visceroautonomically reinforced aspects of orienting thus appear to result in generalized "arousal" while more selective "activation" characterizes behavioral readiness to orient.

In the course of our experimental analysis, a third distinction became necessary. Under many circumstances generalized arousal and selective activation appear to reflexively couple input to output and output to input. On other occasions, however, the components of orienting become uncoupled—such uncoupling appears to entail more prolonged chronic "arousal" involving internally controlled dishabituation often experienced as anxiety, "discomfort" or "effort." Prolongation provides an opportunity for innovation. Damage to the hippocampal system of the brain interferes with uncoupling: animals with such lesions are hyperdistractable (*i.e.*, they dishabituate more readily than controls) provided they are not engaged in a task, in which they become highly resistant to distraction (Douglas & Pribram, 1966; Crowne & Riddell, 1969).

Arousal—familiarization, activation—targeted readiness, and comfort-effortful innovation are therefore three separate dimensions of controls on attention initiated by the orienting reaction. These dimensions can be "dissected" by making the appropriate brain lesions. The next section is devoted to portraying more fully the relationships between these three aspects of orienting and to attention more generally.

Arousal—Familiarization: Habituation of the Orienting Reaction

The arousal component of the orienting reaction is said to occur when an input change produces a measurable brief (several seconds) change in a physiological (*e.g.*, GSR) indicator over a baseline. In psychophysiology such brief changes are referred to as phasic. The types of input change that produce arousal have been studied extensively: they are changes in stimulations that are in one way or another relevant to the well being of the organism. They include sudden changes in intensity to which the organism is unaccustomed, changes in timing of inputs, and changes in the context in which a figure appears. In short, arousal results when, in the history of the organism's experience, a relevant input is novel. Inherent in these operations is the inference that the input is matched against some residual in the organism of its past experience, some familiar representation, a neuronal model of iterated inputs, a competence (Bruner, 1957; Miller, Galanter & Pribram, 1960; Pribram, 1971). Without matching there could be no novelty nor even a measure of change.

Any small change in a parameter of the signal will reconstitute the arousal reaction (Sokolov, 1960, 1963). The waning or habituation of the arousal response must therefore be due to the establishment of a residual neuronal model of that event. Further, certain stimuli which have special relevance, such as one's name, produce dishabituation in an appropriate context, suggesting that familiarization is a process that makes the neuronal model readily accessible. Thus, there are two related consequences of arousal, 1) a visceroautonomic reaction and 2) with stimulus repetition, familiarization.

Activation—Readiness: the Maintenance of Targeted Orienting

The interaction between behaving organisms and their environment is not one-sided. The organism is not just a switchboard for incoming stimulation. Rather, the essence of behaving organisms is that they are spontaneously active, generating changes in the environment often by way of highly programmed, *i.e.*, serially ordered responses (Miller, *et al.*, 1960; Pribram, 1960a, 1962, 1963, 1971). These organizations of behavior must involve the construction of neuronal models in at least two ways: 1) control of the somatomotor system which effects the responses, and 2) feedback from the outcomes (reinforcing consequences) of the behavior. Sherrington (1955), in discussing central representations, framed the question: "Is the organism intending to *do* something about the stimulus variables in the situation?" Germana (1968, 1969) in a review of the evidence suggested that any "neuronal model" must include such "demand" characteristics. Thus he proposed that Pavlov's "What is it?" reaction (which we have called "arousal" and the process of familiarizing the input) does not occur in isolation from a "What's to be done?" reaction. As we shall see, our analysis would suggest that both reactions occur and that they can be distinguished: arousal and familiarization indicating "What is it?" and activation of targeted readiness signalling "What's to be done?"

Readiness differs from familiarization, therefore, in selectively targeting possible outcomes of behavior. Maintaining readiness is reflected in an increase in cortical negativity (CNV) (*e.g.*, Walter, Cooper, Aldridge, McCallum & Winter, 1964; Donchin, Otto, Gerbrandt & Pribram, 1971) and heart rate deceleration (Lacey & Lacey, 1970) which is measured over minutes (and therefore referred to in psychophysiology as tonic).

Comfort—Effort: Innovative Attention

Thus the systems involved in familiarization and targeted readiness can be distinguished: arousal defined as a visceroautonomic reaction which is critical to familiarizing the input, activation as a maintenance of targeted readiness to respond. Under many circumstances, the two reactions appear to be yoked. In such situations they share the function of reflexively coupling input to output, stimulus to response. In the absence of control, behaving organisms would be constantly aroused by their movements and moved by arousing inputs. There must be some long range, or sustained, control process that involves both generalized arousal and active selection which allows *uncoupling* and recoupling to take place. As a rule, initiated inputs (the reinforcing consequences of actions) appear to produce more complexly structured neuronal models than repetitions of simple inputs *per se*. This is largely due to the participation of the central motor systems in *generating* input: *i.e.*, in producing the environmental outcomes that reinforce behavior. Thus, it takes longer to form a habit in, than to habituate to, the same situation. The coordinating process, requiring innovative change from primitive input-output (stimulus-response) states, can be experienced as discomfort or *effort*.

The effort accompanying innovative change (during problem solving) is reflected, both centrally and peripherally where isometric muscular contraction (Berdina, Kolenko, Kotz, Kuzetzov, Rodinow, Savtchencko & Thorevsky, 1972) and increased blood flow are accompanied by chronic accelerations of heart rate (Lacey & Lacey, 1970).

Effort is here defined as a measure of the *efficiency* with which energy (metabolic output) is expended in producing a "change of state" in control systems. Our definition of energy is in keeping with the definition of energy in physics (see also McFarland, 1971) as the capacity for doing work, *i.e.*, for innovation, for changing the state of a system (or maintaining a state in the face of changes in external parameters). Effort additionally measures the *cost* of such change and is thus an index of the efficiency, the negentropy, with which the work is accomplished.

Basis for the Model

The Amygdala Circuits and Familiarization

Studies on the behavior of neural systems during arousal in animals have revealed that brief psychophysiological responses to sudden changes in stimulus events are a ubiquitous property of certain portions of the central nervous system. In an extensive series of experiments, reviewed by Groves and Thompson (1970), these authors distinguished a system of "arousal" neurons in the medial portions of the spinal cord. This system of neurons in turn converges with another more laterally placed set of decrementing neurons onto a final common path that habituates and dishabituates much as does the motor behavior in which these neural systems are involved. There is every reason to believe that the rostral extension into the mesencephalic brainstem of the column of medially placed cells accounts for the well documented arousal effects of stimulations of the reticular formation (see Lindsley, 1961; Magoun, 1958 for review). Such effects are obtained even more rostally in the diencephalon in a continuation of this neuron system into the hypothalamus where episodes of general alerting, fighting and fleeing are produced

by electrical or chemical stimulation to the so-called "defense" or "stop" region of the hypothalamus.

General alerting is produced as well by electrical stimulation of the orbitofrontal cortex, midline and medial thalamus and amygdala. The reaction closely resembles that produced by stimulation of the hypothalamus and mesencephalic reticular core (Wilcott & Hoel, 1973). Such stimulation also results in visceroautonomic activity and in the activation of the cells of the reticular nucleus of the thalamus, momentarily closing sensory input gates (Skinner, 1989). More on this below.

These effects have been shown to be related to the psychophysiological components of the orienting reaction. Abrahams and Hilton (1958) and Abrahams *et al.* (1964) found that in attempting to produce a defense response by stimulation of the hypothalamus, at first a much lower degree of arousal occurred, indicated by pupil dilation and postural alerting. Only when the level of stimulation was increased and maintained for a few seconds, did hissing, snarling, running and piloerection occur. In the later study, alerting psychophysiological components were measured in greater detail, and during mild stimulations the authors observed changes in pupil dilation, respiration and blood flow to accompany head movements and pricking the ears. These same changes were also recorded during responses to simple auditory, visual or cutaneous stimuli, in the absence of hypothalamic stimulation. Since these physiological changes are the same as those observed in all orienting responses, the defense reaction could therefore be considered in part as due to an increase of arousal.

Converging on these hypothalamic structures are two reciprocally acting circuits regulating arousal. These circuits center on the amygdala. This structure is classified as a basal ganglion and part of the limbic forebrain (for an extensive review see Pribram & Kruger, 1954; and Pribram & McGuinness, 1975). One of these circuits involves the ventrolateral frontal cortex and is excitatory since resections of this structure *invariably* eliminate visceral-autonomic orienting responses. The other, opposite in function, is related to the orbitofrontal cortex which has been shown to be the rostral pole of an extensive inhibitory pathway (Kaada, Pribram & Epstein, 1949; Pribram, 1961, 1987; Sauerland & Clemente, 1973; Skinner & Lindsley, 1973; Wall & Davis, 1951).

Observations of the behavior of amygdalectomized animals (Pribram & Bagshaw, 1953), confirm the opponent nature (Solomon, 1980) of these two systems. Ordinarily amygdalectomy produces monkeys that are tame, unresponsive to threat and nonaggressive. However, the opposite finding has also been occasionally observed (*e.g.*, Rosvold, Mirsky & Pribram, 1954). Studies by Ursin and Kaada (1960) using more restricted lesions and electrical stimulations have identified two reciprocal amygdala systems that account for opponent reciprocity.

Reciprocal innervation allows sensitive modulation (tuning) of the arousal mechanism. This is in accord with evidence from other control functions of the amygdala and related structures. For instance, injections of carbachol into the amygdala have no effect unless the animal is already drinking, in which case the amount of drinking becomes proportional to the amount of carbachol injected (Russell, Singer, Flanagan, Stone & Russell, 1968). The fronto-amygdala influence finely tunes viscero-autonomic arousal initiated by the hypothalamic mechanism. It is as if, in the absence of the fronto-amygdala systems, the animal would fail to control his drinking behavior: once started he would drink under circumstances in which others would stop. This is exactly what happens—and more. Both eating and drinking are controlled in this fashion (Fuller, Rosvold & Pribram, 1957).

A clue to what these controls on arousal accomplish, comes from the finding that despite an essentially normal reactivity to shock, the amygdalectomized

subjects have fewer spontaneous GSRs during the shock sessions, suggesting a change in base level (Bagshaw & Pribram, 1968). That baseline changes do occur after amygdala lesions was demonstrated directly in sustained chronic response measures (see below) and indirectly by various studies which showed that although behavioral and some electrocortical responses appeared to be normal during orienting (Schwartzbaum, Wilson & Morrisette, 1961; Bagshaw & Benzies, 1968) the background level of these responses is lower than in controls. Ear flicking is practically absent during interstimulus intervals (Bateson, 1972), and it takes less time for the lesioned animals to attain a criterion of slow wave activity in the EEG (Bagshaw & Benzies, 1968) in the preparatory phase of the experiment. While electromyographic (EMG) responses occur with normal latency, the amplitude of these responses is considerably reduced (Pribram et al., 1979). These results indicate that at the forebrain level, just as at the spinal level in Groves' and Thompson's experiments (1970), arousal and decrementing systems converge to produce orienting, habituation and dishabituation.

Perhaps the most striking chronic psychophysiological change to follow amygdalectomy was the finding of a paradoxically elevated basal heart rate (Bagshaw & Benzies, 1968; Pribram *et al.*, 1979). This puzzled us considerably and made data collection analysis difficult (operated and control monkeys had to be matched for basal rate; it had to be shown that no ceiling effect was operating). We wondered whether "arousal" as a concept was in fact untenable in the face of lack of evidence for orienting coupled with an elevated heart rate. Experimental results obtained by Elliott (Elliott, Bankart & Light, 1970) and their analysis clarified the issues. They expected an elevated heart rate to accompany arousal (defined as a response to collative variables such as surprise, and novelty of input much as we have defined them here) but as they were recording longer lasting rather than brief changes he found the opposite: "These collative variables either have no effect on tonic heart rate or they had an effect (deceleratory) opposite to expectations; but response factors and incentive factors (reinforcing consequences) had strong accelerating effects."

Arousal is ordinarily followed by heart rate *deceleration*, which is indicative of activation. By contrast, the monkeys with absent arousal reactions show an *elevated* heart rate. They thus appear to be working with considerable *effort*. In accord with the psychophysiological data on humans, such elevated heart rate is manifest when the situation demands the concentration of attention. Our observations suggest that without such expenditure of effort the amygdalectomized monkeys tend to fall asleep.

We therefore interpret the effects of amygdalectomy as follows: because the specific controls on arousal are removed, arousal results not in familiarization of the situation by altering the access to the neuronal model, but in immediate reflexive distraction. This increased distractibility evokes a defensive effort to cope with the situation. The defense reaction is characterized by an attempt to shut off further input (see Pribram, 1969), an effect inferred from neurophysiological evidence of control over input. The effort is reflected in an elevated heart rate and other changes in chronic autonomic variables indicative of a continuing defense against impending breakdown in the coordination involved in maintaining a set in the face of distraction.

This interpretation is borne out by the results of an experiment in which infant kittens were raised in isolation. When their orienting behavior was examined after six months of isolation, the kittens' visceroautonomic and endocrine reactivity was essentially that of amygdalectomized subjects: they had not learned to cope

with situations (had not built up neuronal models) and thus showed the "defensive" syndrome suggestive of considerable effort (Konrad & Bagshaw, 1970).

In summary, studies relating brain function to the visceroautonomic components of the orienting reaction have identified a system of neurons which familiarize a novel input. This core system of neurons extends from the spinal cord through the brain stem reticular formation, including hypothalamic sites and lies in close proximity to those responsible for the engenderment of visceroautonomic responses to novelty. Forebrain control over this corebrain arousal system is exerted by reciprocal facilitatory and inhibitory circuits centered on the amygdala. These circuits control the onset and duration of arousal by controlling the onset and duration of visceroautonomic responses.

It is the relationship between the lack of visceroautonomic responses to orienting and the failure to habituate behaviorally that indicates that a deficiency is produced in a central process by which organisms become familiar with an input: that is, they have ready access to their neuronal model for updating or orienting (dishabituating). Mild disturbances of this process produce the clinical picture of "déjà" and "jamais vu." More severe disturbances produce the automatisms occurring during psychomotor seizures in the presence of epileptic lesions in the region of the amygdala.

Based on the results of the experiments reviewed here, Mednick and Schulsinger (1968) and Venables (Gruzelier & Venables, 1972) have reported two classes (GSR responders and nonresponders) of patients diagnosed as schizophrenics. Responders have a much better prognosis than nonresponders. In fact, the classification has been successfully used as a screening device to identify children in families with a history of schizophrenia who are at risk. Identification can be made before the children show overt symptoms and can, therefore, be sheltered from being exposed to overly traumatic situations.

The Basal Ganglia and the Maintenance of Targeted Readiness

In structures such as the mesencephalic reticular formation and the hypothalamic region a system can be identified with the familiarization process detailed above: when excited as by a novel input, this system operates to stop behavioral reactions to that input by virtue of habituation and/or satiety. Closely coupled to this "stop" or "interrupt" process is its reciprocal, a process that operates to continue targeted behaviors. This readiness process was discovered in relationship to food appetitive processes: in collaboration with one of us (KHP), Anand and Brobeck (1952) discovered that stereotaxic lesions of the "far-lateral" hypothalamic region produced aphagia (animals who failed to eat and starved to death if left alone). Anand (1963) went on to show electrophysiologically (with unit recordings) the activity in this region was reciprocal to that in the ventromedial nucleus of the hypothalamus; when an animal began eating or drinking, unit recordings in the far lateral hypothalamic region were active and those obtained from the ventromedial nucleus were inactive; when satiety set in due to an increase in blood sugar level (as reflected in the arteriovenous ratios), the cells of the ventromedial nucleus became active, while recordings from the far-lateral region showed diminished activity.

The aphagia produced by far-lateral hypothalamic lesions turned out to be peculiar. Teitelbaum in a long series of studies (Teitelbaum, 1955; Teitelbaum & Epstein, 1962; Teitelbaum & Milner, 1963) showed that animals with such lesions would eat if given food which had proven to be highly attractive to nonlesioned

animals—sweets, for instance. It was as if the lesioned animals were "finicky" and simply ignored food because their appetite threshold had been markedly raised.

A similar decrease in responsivity to other forms of stimulation has been classically observed to follow certain lesions in the frontal and parietal regions of the cerebral hemispheres of humans (*e.g.*, Semmes, Weinstein, Ghent & Tueber, 1963) and animals (see below). Ignoring becomes especially manifest after unilateral lesions when both the ipsilateral and the contralateral hemifields are simultaneously stimulated. In such instances the stimulus contralateral to the lesion is routinely ignored. This is the syndrome of "neglect."

Heilman and his group (*e.g.*, Heilman & Valenstein, 1972; Heilman & Watson, 1977) have systematically produced "neglect." These investigators find that certain lesions of the mesencephalic reticular formation and of the far-lateral hypothalamic system interfere with the targeted aspects of orienting. Behavioral orienting to food and water has been shown to follow electrical stimulation of this system. Such orienting is prolonged and maintains readiness. Behaviorally, targeted orienting is markedly different from the generalized alerting produced by stimulation of ventromedial hypothalamic system which interrupts ongoing adaptive behavior even to the point of producing sham rage (Hoebel, 1974, 1976; Hernandez & Hoebel, 1978; Abrahams & Hilton, 1958).

There are no cells in the far-lateral hypothalamic region. Rather, this region consists mainly of the median forebrain bundle connecting the mid- and forebrain. The bundle is crossed with fibers connecting the amygdala with the ventromedial hypothalamic system. Ungerstedt (1974) showed that the dopaminergic fibers originating in the substantia nigra and terminating in the basal ganglia (caudate, putamen and globus pallidus) make up a great portion of the median forebrain bundle as it traverses the far-lateral hypothalamic region. Teitelbaum (1955) and Fibiger, Phillips and Clouston (1973) have established that the food "neglect" syndrome is due to lesions of this tract by using antidopaminergic agents to produce "finickiness' and neglect.

Recall that lesions of the amygdala (and those of the ventromedial nucleus of the hypothalamus which results in excessive eating) produced a failure to habituate and thus a continuation of generalized orienting over repetitions of a sensory input. Contrast this to the effects of lesions of the basal ganglia system coursing through the far-lateral hypothalamic region which produce a failure in targeted orienting, neglect and finickiness. It is these reciprocal effects that provide a strong support for the distinction between a "familiarize" and a "readiness" system.

Studies on animal and human patients with lesions in the basal ganglia (Bowen, 1976) also show this inability to maintain targeted attention. In a series of studies employing multiple small stereotactic lesions in the globus pallidus, putamen and caudate nucleus Denny-Brown and Yanagisawa (1976) report their findings with the following summary: "What then is absent? It would appear to be the activating 'set' or 'pump primer' for a certain act, the preparation of the mechanism preparatory to a motor performance oriented to the environment." They also note a particular type of ramp discharge in electrical activity in putamen neurones (see also DeLong & Strick, 1974) which precedes the motor performance at every stage. They suggest this operates as a facilitatory discharge which establishes a "climate" for performance.

They further suggest ". . . the basal ganglia have all the aspects of a 'clearing house' that accumulates samples of ongoing cortical projected activity and, on a competitive basis, can facilitate any one and suppress all others." This indicates that the part of this system relates to an ability to transfer attention from one type of stimulus to another and maintain that attentional set.

The Hippocampal System and Innovative Effort

Data on animal behavior following hippocampectomy indicate that this structure and its connections are critical in coordinating the familiarization and readiness systems. While orienting, subjects with bilateral hippocampectomy show a greater number of, and a greater amplitude of galvanic skin response than controls—a visceroautonomic reactivity opposite to that observed in nonresponding amygdalectomized monkeys. In addition, brief galvanic skin responses terminate considerably more rapidly in hippocampectomized subjects than in controls. It appears from this that hippocampectomized monkeys restabilize more rapidly than normal subjects whose slower galvanic skin response recovery may indicate a more prolonged processing time.

A further change is that such subjects show delayed or absent orienting reactions when thoroughly occupied in performing some other task (Crowne & Riddell, 1969; Kimble, Bagshaw & Pribram, 1965; Raphelson, Isaacson & Douglas, 1965; Riddell, Rothblat & Wilson, 1969; Wicklegren & Isaacson, 1963). In short, the animals appear to be abnormally undistractible while occupied. But in some situations this apperance of undistractibility is restricted to the overt *responses* of the organism, not to orienting *per se*. Douglas and Pribram (1969) used distractors in a task in which responses had been required to each of two successive signals. Hippocampectomized monkeys initially responded much as did controls by manipulating the distractors which appeared between the two signals, increasing the time between the two responses.

However, the controls began to ignore the distractors and speeded their inter-response time. In the hippocampectomized group the number of manipulations declined but their inter-response time remained slow. In this situation, hippocampectomized monkeys continued to be *perceptually* distractible while becoming behavirorally habituated and undistractible. This result is reminiscent of that obtained in man with medial temporal lesions: instrumental behavior can to some considerable extent be shaped by task experience, but verbal reports of the subjective aspects of experience fail to indicate prior acquaintance with the situation (Milner, 1958).

The dissociation between habituation (familiarization) of perceptual responses and habituation involving somatomotor performance appears to be part of a more general effect of hippocampal lesions. In a discrimination reversal situation, extinction of previously learned behavior and acquisition of new responses was observed. In contrast to their controls, the monkeys with the hippocampal lesion remained at a chance level of performance for an inordinately long time (Pribram, Douglas & Pribram, 1969) despite the fact that their recovery from extinction and the slope of their reversal learning curves was completely normal. This was due to the "capture" of the behavior by the 50% intermittent schedule of reinforcement (Spevak & Pribram, 1973). This result suggested that self-directed "observing" responses (indicative of "attention") were relinquished when the probabilities of reinforcement ranged around the chance level.

Taken together, these experimental results suggest that interference with the hippocampal circuit reduces the organism to a state in which the more effort demanding relationships between perception and action, between observing and instrumental responses, and between stimulus and response are replaced by more primitive relationships in which either input or output captures an aspect of the behavior of the organism without the coordinating intervention of central control. The mechanism by which the hippocampal circuit accomplishes the more complex relationship has been studied by making recordings of electrical activity from the

hippocampus, with both micro- and macroelectrodes. Before we come to these studies, however, we need to review the neurochemistry, not only of the hippocampal but also the amygdala and basal ganglia systems.

Extension of the Model: Neurochemical Analysis

The evidence reviewed so far has indicated that the neural systems involved in orienting are composed of sets of reciprocally acting mechanisms. Reciprocity has been analyzed by Fair (1965) as an "answering" process and has been the subject of an extensive series of studies by Solomon and his group (see Solomon, 1980 for review) under the label of "opponent process theory." Pribram (1977) has suggested that reciprocity is based on the action of neurochemical systems that to a considerable extent coincide with the three sets of systems (familiarization, readiness and effort) delineated by psychophysiological and neurobehavioral techniques.

A caveat: Each of the "systems" described are of course sets of systems. As already noted, the amygdala is made up of three groups of nuclei: basolateral, central and corticomedial (see Pribram & Kruger, 1954 for review). The basal ganglia are composed of the caudate nucleus, putmen, nucleus accumbens and pallidum. The hippocampus has, in subprimate mammals, a dorsal and a ventral portion—the dorsal portion becomes a vestigial rudiment in primates, the induseum griseum. Furthermore, different layers of the hippocampal formation have different functions in behavior (see Lindsley & Wilson, 1976; in Isaacson & Pibram, *The Hippocampus*, Vol. II). Thus, when matching neurochemical systems to the sets of systems described so far, this can be done at present only with broad strokes.

Generally speaking, the following scheme can be made out: a serotonergic-adrenergic interaction involving the amygdala systems; a cholinergic-dopaminergic interaction involving mesolimbic (n. accumbens), pallidal and caudate basal ganglia systems; and a cholinergic-aminergic interaction involving the hippocampal systems. These reciprocal interactions are superimposed on or activated within a set of steroid, adrenocortical-adrenocorticotrophic and peptide mechanisms that further modulate processing.

Serotonergic-Adrenergic Interactions

A large amount of research (*e.g.*, reviews by Jouvet, 1974; Barchas, Ciaranello, Stolk & Hamburg, 1972) has related the serotonergic and adrenergic systems to the phases of sleep: serotonin to ordinary (slow wave) sleep and norepinephrine to paradoxical (rapid eye movement) sleep during which much dreaming occurs.

For the most part serotonergic and adrenergic pathways overlap and converge rostrally on the amygdala. Thus Cooper, Bloom and Roth (1978, p. 206) note that "most raphé neurons (the origin of the serotonergic systems) are more norepinephrine-like than dopamine-like in their topography. (One) group appears to furnish a very large component of the 5-HT innervation of the limbic system." This innervation reaches the amygdala via stria medularis and stria terminalis.

The regulation of sleep by the amygdala has not been quantitatively documented although sleep disturbances are commonplace immediately following amygdalectomy, the animals often falling into a torpor from which they are difficult to rouse for from several days to several weeks.

However, norepinephrine has been related to a behavioral function in which the amygdala systems are consistently implicated—the effects of reinforcing events (Stein, 1968). Norepinephrine has also been related to orienting and affective agonistic reactions. Once again a response to novelty—sensed against a background of familiarity—is norepinephrinergic, whereas "familiarity" in the guise of "territoriality" and "isolation" has been shown to some considerable extent to be dependent on a serotonergic mechanism (see reviews by Reis, 1974; Goldstein, 1974).

These data suggest that norepinephrine acts by regulating serotonergic substrate (which is determining one or another basic condition of the organism) to produce paradoxical sleep, reinforcement, orienting and perhaps other behaviorally relevant neural events that interrupt an ongoing state. In all likelihood there is a third level of modulation—the neuropeptides which also show some reciprocity in their activity. Thus substance P and the endorphines act reciprocally and both are found in abundance in the amygdala. More on this shortly.

Cholinergic-Dopaminergic Interactions

The most clear-cut evidence regarding neurochemical control systems is the now well established and dramatic findings of a dopaminergic nigrostriatal and mesolimbic (n. accumbens) mechanism that reaches the lateral frontal cortex (Fibiger, Phillips & Clouston, 1973; Ungerstedt, 1974; Goldman-Rakic & Schwartz, 1982). The evidence has been repeatedly reviewed to the effect that dopamine is involved in the maintenance of postural and targeted readiness (Matthysse, 1974; Snyder, Simantov & Pasternak, 1976).

In addition to the nigrostriatal and mesolimbic dopaminergic system, there is another that intimately involves the basal ganglia. This is the cholinergic system (reviewed extensively by Fuxe, 1977) which reaches the globus pallidus from which it innervates the cortex. It is also known that assertive agonistic behavior such as predatory aggression depends on the activation of cholinergic mechanism (see, *e.g.*, King & Hoebel, 1968). Thus it is likely that the dopaminergic process regulates a cholinergic substrate (see Fuxe, 1977) to determine the maintenance of targeted readiness of the organism.

Cholinergic-Aminergic Interactions

Cholinergic and aminergic (both serotonergic and norepinephrinergic) pathways converge on the septo-hippocampal system (Cooper *et al.*, 1978, p. 165, 206); a convergence which could account for the part this system plays in integrating the activity of the amygdala and basal ganglia systems. The regulation of septo-hippocampal cholinergic neurons by catecholamines has been delineated by Robinson, Cheney & Costa, (1981) and Butcher, Woolf, Albanese & Butcher, (1981). Oderfeld-Nowak and Aprison (1981) have presented evidence that those same cholinergic mechanisms are modulated by serotonergic indolamines. The interaction between cholinergic hippocampal neurons and adrenergic mechanisms on the one hand, and cholinergic hippocampal neurons and serotonergic mechanisms on the other, are, however, independent of one another (Ladinsky, Consolo, Tirelli, Forloni & Segal, 1981). We must therefore look at another "higher" level of neurochemical interaction for integration of these independently operating (perhaps opponent) processes. This higher level is reviewed in the next section.

Adrenocortical-Adrenocorticotrophic and Peptide Interactions

There is a matrix of steroid and peptide processes upon which and within which the cholinergic and aminergic mechanisms operate. For instance the amygdala systems are intimately interconnected with hypothalamic nuclei (supraoptic) which are rich in sex steroids and the nucleus of the amygdala is itself a site of concentration of such steroids. The hippocampal system is intimately involved in the pituitary-adrenocortical axis in the regulation of stress. Thus the receptors of adrenal cortical hormones can set the neural state which becomes regulated by ACTH. Bohus (1976) and McEwen (McEwen, Gerlach & Micco, 1976) showed that it is, in fact, the hippocampal formation that is the brain site most involved the selective uptake of adrenal cortical steroids. As McEwen states:

> It is only quite recently that we have come to appreciate the role of the entire limbic brain, and not just the hypothalamus, in these endocrine-brain interactions. Our own involvement in this revelation arose from studies of the fate of injected radioactive adrenal steroids, particularly corticosterone, when they entered the brain from the blood. These studies were begun, under the impetus of recent advances in molecular biology of steroid hormone action, to look for intracellular hormone receptors in brain tissue. We expected to find such putative receptors in the hypothalamus, where effects of adrenal steroids on ACTH secretion have been demonstrated (Davidson *et al.*, 1968; Grimm & Kendall, 1968). Much to our surprise, the brain region which binds the most corticosterone is not the hypothalamus but the hippocampus (McEwen *et al.*, 1976).

As the hippocampal circuit functions to coordinate familiarization with targeted readiness to make innovation possible, manipulations of any of the neurochemical mechanisms thus far described can be expected to produce a host of apparently conflicting results with very slight charges. An example is changing a one-way versus two-way conditioned avoidance task (see Pribram, Lim, Poppen & Bagshaw, 1966; van Wimersma, Greidanus & de Wied, 1976) which dramatically changes the results obtained under different drug conditions.

Effects on familiarization and readiness as well as on their coordination (effort) would be predicted. This expectation is borne out in the catalogue of results obtained with manipulations not only of ACTH but also of ACTH-related peptides: extinction of two-way but not one-way avoidance (de Wied, 1974); interference with passive avoidance (Levine & Jones, 1965); interference with learned taste avoidance (the Garcia-effect—Levine, Smotherman & Hennessay, 1977); interference with discrimination reversal (Sandman, George, Nolan & Kastin, 1976); facilitation of memory consolidation (van Wimersma *et al.*, 1976); and facilitation of exploratory behavior and conditioning (Endroczi, 1972).

Just as in the case of manipulation of hippocampal activity, *ongoing* behavioral activity (memory consolidation, exploratory behavior) is facilitated, while any change in behavior (two-way shuttle, passive avoidance, learned taste aversion, discrimination reversal) is interfered with. This appears initially as tilting the bias toward readiness. But as Pribram and Isaacson (1976) show for hippocampal function, and Sandman's group conclude (see Miller, Sandman & Kastin, 1977) such an interpretation is not valid. In the case of hippocampal research, the initial formulation states that after hippocampal resections, animals could not inhibit their responses (McCleary, 1961). This interpretation foundered when it was shown that such animals performed well in go/no-go alteration tasks (Pribram & Isaacson, 1976; Mahut, 1971) and that they could withhold behavioral responses despite an increase in reaction time when distractors were presented (Douglas & Pribram, 1969).

The most cogent analysis has been performed on discrimination reversals. Isaacson, Nonneman and Schualtz (1968) and Nonneman and Isaacson (1973) have shown that reversal learning encompasses three stages: extinction of the previously correct response, reversion to a position habit, and acquisition of the currently correct response. Pribram, Douglas and Pribram (1969) and Spevak and Pribram (1973) have shown that hippocampally lesioned monkeys are intact with regard to both the extinction and the new acquisition phases of the reversal training experience. However, such monkeys seem to become "stuck" in the 50% reinforcement phase or in the position response patterns. In short, the monkeys' behavior seems to be taken over by a relatively low variable interval schedule of reinforcement and they fail to "make the effort" to "pay attention" to the cues which would gain them a higher rate of reward. Champney, Sahley and Sandman (1976) have shown ACTH-related peptides to operate on just this aspect of the reversal experience—and, in fact, have shown interactions with sex differences.

Finally, ACTH and related peptides, the enkephalins, are endorphins—endogenous hormones that have morphine-like effects and, in fact, act as ligands on morphine receptors. These neuropeptides and the hippocampal circuit in which they are operative function therefore to modulate an effort-comfort dimension of experience and behavior.

Evidence such as this makes highly plausible the hypothesis that ACTH and ACTH-related peptides operate on the hippocampal circuit and therefore the "effort" process. Moreover, Strand, Cayer, Gonzalez and Stoboy (1976) present direct evidence that muscle fatigue is reduced by ACTH-related peptides and that this effect must be central. Before this study, the only evidence of metabolic shifts due to the effort of paying attention came from Berdina *et al.* (1972) (noted in the initial section of this review). It now appears that these peripheral anaerobic shifts affecting muscle tonicity may be a reflection of central processing modulated by ACTH and ACTH-related neuropeptides.

Test of the Model: Analysis of Event-Related Brain Electrical Potentials

The recording of brain electrical potential changes has added an all important dimension to the analysis of controls on attention. They have the advantage over other measures in that they are more immediate indicators of the brain activities that operate the relevant controls. They provide, therefore, an excellent opportunity to test, amend and add to the model of attention and para-attentional processes proposed in the previous sections.

To briefly summarize the nomenclature used in this section, event-related brain electrical potentials have been analyzed into the following process-related components: 1) The early components of event-related potentials which occur within approximately 50 milliseconds (depending on modality) reflect activity in the extrinsic systems. 2) The beginning of selection processing is heralded by a positivity occurring roughly at 60 msec to be followed by a processing negativity, occurring about 80–100 msec after the stimulus. This negativity is an indicator of sensory channel selection on the basis of sensory features. 3) Once again a new processing phase is reflected in a positive deflection followed by a negativity, which begins approximately 200 msec after the stimulus and may extend beyond the 400-msec range. This negativity has been shown to reflect within-channel selection. 4) Within-channel processing must be updated and the onset of this process is signalled by a positive component. 5) However, this positive component has two rather different sources; only one component, the P3b, reflects the initia-

tion of the updating procedure. 6) The other, the P3a, which is usually found in a frontal location, reflects generalized orienting. 7) The P3b often, though not always, reflects a rebound from a prolonged negativity, the contingent negative variation (CNV). 8) But the CNV itself is not of unitary origin. This negativity also has a frontal component related to generalized orienting and a set of other components which are modality specific and include a motor readiness potential. Only a brief review of the evidence supporting this nomenclature is presented; more comprehensive reviews make up the remainder of this volume. Our aim here is to relate relevant findings to test and sharpen our model.

Positive Brain Electrical Potentials, Generalized and Targeted Orienting

There is an old observation made in the 1930s by Morison, Dempsey and Morison (1941) in which they reported that resections of the medial portion of the temporal lobe especially the amygdala, interfere with the production of secondary (*i.e.*, late components) responses evoked by sensory stimulation. In addition, Halgren *et al.* (1980) have recorded late (300-msec) components of event-related brain electrical activity (correlated with scalp recording) in the amygdala and hippocampus of human subjects during brain surgery.

An extensive set of studies has been performed in an attempt to determine the psychological process(es) coordinate with the occurrence of such positive deflections, especially those involving stimuli relevant to the organism. Hillyard and Squires thoroughly review this evidence (Hillyard, Squires, Baver & Lindsay, 1971) and conclude that these positive deflections reflect more than one process: a generalized orienting response and a more complex and active attentional process. Generalized orienting is reflected in a deflection which is early and maximal at frontal leads, while active attending produces later positive deflections that are maximal at posterior leads.

The positive deflection occurring around 300 msec after the stimulus, is made up of two subcomponents: a P3a and a P3b. The P3a component is related to generalized orienting and is largely frontal in distribution while the somewhat later P3b is influenced by a set of within-channel selection variables as is the prior processing negativity (Nd). What is of special interest is that this P3b component can be shown to occur—in reaction time experiments—*after* an overt response has already taken place. Thus the P3b cannot be a direct correlate of targeting but must reflect the initiation of a new phase of processing in which the sequelae, the consequences, of targeting are processed.

When the P3a component is prolonged, it is accompanied by desynchronization of the EEG (Grandstaff & Pribram, personal observation) and reflects the continuation of the response, usually in consummatory behavior (Clemente, Sterman & Wyrwicke, 1964). In such instances, the positivity is accompanied by a sharp increase in power both in the alpha (8–12H3) and in the theta (4–8H3) ranges (Grandstaff, 1969) recorded from the cortex of the cerebral convexity. (Conversely, negativity is accompanied by desynchronization; Pribram, 1971, p. 111.)

The P3b as recorded in the "odd-ball" task, signals the onset of an updating process in response to the unpredictable sequential structure of the task. Although updating has been ascribed to the P3 positivity [as attributed by Donchin (Donchin & Coles, 1988) to Pribram and McGuiness (1975)] a more likely interpretation is that updating is reflected in a late (400–600-msec) negativity. (See also the critique by Verlerger, 1988.)

The effects of generalized and targeted orienting are also reflected in the

electrical activity recorded from the hippocampus. As a rule, however, synchronization (in the theta range) is recorded when desynchronization occurs in the cortical convexity and hippocampal desynchronization accompanies convexal synchronization. Lindsley (Macadar, Chalupa & Lindsley, 1974) in keeping with many other recent publications (*e.g.*, Fibiger *et al.*, 1973; Ungerstedt, 1974) has dissociated two systems of neurons that influence hippocampal synchronization and desynchronization. One system originates in the anterior portion of the median raphé and associated midline structures of the mesencephalon and courses through the medial portion of the hypothalamus. The other originates more laterally in the median forebrain bundle through the lateral hypothalamus. Electrical stimulations of the lateral mechanism produce hippocampal desynchronization and a momentary "locking on" to a specific aspect of the environment. Stimulations of the medial mechanisms result in a synchronized hippocampal theta rhythm (4–8 Hz), which is accompanied by isocortical desynchronization and in targeted orienting and exploration.

Theta frequencies were first recorded from the hippocampus by Jung and Kornmuller in 1938. Since this discovery theta has been implicated in generalized orienting (Green & Arduini, 1954; Grastyan, 1959; Grastyan, Lissak, Madarasz & Donoffer, 1959) and to intended movement, even when tested under curare (Dalton & Black, 1968; Black & Young, 1972; Black, Young & Batenchuck, 1970). Vanderwolf and his associates (Bland & Vanderwolf, 1972a; 1972b; Vanderwolf, 1969, 1971; Whishaw, Bland & Vanderwolf, 1972) noted that theta activity occurred almost exclusively when animals (rats) were making "voluntary" movements. Though synchronization in the form of a theta rhythm is not as obvious in records obtained in monkey and man, computer analysis has shown it to occur under similar circumstances in primates (Crowne, Konow, Drake & Pribram, 1972).

The results of the Lindsley studies (Lindsley & Wilson, 1976; in Isaacson & Pribram, *The Hippocampus*, Vol. II) as well as those of many others thus indicate that the hippocampal process can operate in at least two modes which regulate orienting: 1) Tonic inhibitory discharge of hippocampal neurones signified by theta rhythms leads to targeted exploration of more or less familiar territory during which the organism is presumably comfortable and updates his processing competence. 2) When generalized orienting occurs because something relevant (such as food) has been encountered, the inhibitory neurones are shut off, and hippocampal rhythms become desynchronized (while, as noted, those of the cortical convexity become synchronized), attention becomes focussed and, to a considerable extent, the organism is insulated from distracting explorations.

Negative Brain Electrical Potentials, the Selection of Sensory Input and the Targeting of Readiness

CNVs, TNVs, Generalized Arousal and Targeted Readiness. In the introduction we defined activation in terms of a readiness to respond, a readiness which allowed behavior to become or remain targeted by virtue of being resistant to generally destabilizing interruptions.

The simplest situation which demands that responses become or remain on target is one in which two successive input signals are separated by an interval. The first input signals the organism to become ready to make a response to the second, which determines the outcome. In this situation, a large body of data has been gathered regarding slow changes in brain electrical activity, *i.e.*, *contingent negative variations* (*CNVs*) (Walter *et al.*, 1964). In turn, these negativities have

been related to the tonic slowing of heart rate (Lacey & Lacey, 1970) which was the psychophysiological basis of our definition of tonic activation.

The CNV was originally proposed to reflect an expectancy developed when a response was contingent on awaiting the second of two stimuli. This would suggest that the CNV reflects a central process activating the organism's neuronal model of this contingency. Other research indicated that the negtive shift in potential reflects intended motor activity (*e.g.*, Kornhuber & Deecke, 1965; Vaughan, Costa & Ritter, 1968). However, still another group of investigators (Weinberg, 1972; Donchin, Gerbrandt, Leifer & Tucher, 1972) demonstrated that a CNV occurs whether or not an overt motor or even a discriminative response is required, provided some set or expectancy is built into the situation. Such sets do, of course, demand postural motor readiness. Weinberg (1972), for instance, has shown that in man the CNV continues until feedback from the consequences of reinforcement of the response occurs. Similar evidence has been obtained in monkeys (Donchin, Otto, Gerbrandt & Pribram, 1971, 1973).

Teece, reviewing the literature on the CNV (1972) noted that, in humans, three types of negative potentials could interact depending upon demands of the experiment: (a) a CNV due to expectant attentional processes; (b) the motor readiness potential signaling intention to act; and (c) more or less "spontaneous" shifts. This classification was considerably sharpened by results obtained in a series of nonhuman primate studies (Donchin, Otto, Gerbrandt & Pribram, 1971, 1973) which specify more completely Tecce's last category. Bipolar (surface to depth) recordings were made from several cortical locations under a variety of conditions. These studies showed that sites which produced transcortical negative variations (TNVs) depended upon the type of task. Thus, far frontal TNVs were recorded sporadically early in the task and whenever the task was changed; precentral motor negative potentials were recorded only in anticipation of the necessity to make an overt response (release a depressed lever); while special sensory systems responded to their specific inputs—*e.g.*, parietal negativity occurred while the monkey was holding down the lever.

These data were paralleled by a study on humans (Gaillard, 1977) in which preparation was compared to expectancy in three tasks, one involving speed, another accuracy and the third, detection, but no response. The far frontal leads mirrored generalized expectancy in the no response condition in which no parietal CNV occurred. The other leads were affected by the task demands. The speed condition produced maximal CNV shifts in the parietal leads, the accuracy condition in the motor leads.

The evidence thus indicates that the CNV has a multiple composition: a frontal O (generalized orienting or arousal) wave which can peak as late as 500–800 msec and frequently occurs prior to a late parietal positivity; and a set of E (expectancy) waves which are modality specific and include as one of their manifestations the motor readiness potential.

Hillyard and the Squires (1971) identify the E waves with readiness on the basis of correlations with psychophysiological measures such as Lacy and Lacy's slowing of the heart rate, as do Pribram and McGuinness (1975). However, Hillyard and the Squires (1971) also identify the E waves of the CNV with effort. They show that the amplitude of these waves is a function of task difficulty. This constitutes a major disparity between the systematization attempted here and that which they provide in theirs.Their inference is based on the fact that when multiple tasks which are compatible are processed, the amplitude is additive. However, as they also note, when the tasks are incompatible (which to our view would increase the demand for effort) the amplitude is *reduced*: amplitude appeared in some

instances at least to be inversely correlated with effort. It is thus more likely that readiness and effort reflect the operations of two separable neural systems, and that the E waves of the CNV reflect only the operation of the readiness system.

Event-Related Negative Potentials, Sensory Selectivity and the Targeting of Readiness. Analysis of event-related negative potentials has allowed a further processing distinction to be made. One process depends on "the rapid efficient selection of inputs by virtue of their physical attributes or features" (Hillyard *et al.*, 1971). This process corresponds to Broadbent's (1977) stimulus filtering process. A second slower, serial process (Naatanen, 1982, 1990) occurs whereby comparisons of input are made against "dictionary" units in memory prior to classification—Broadbent's pigeon-holing. This distinction has also been termed a between-channel vs a within-channel selection. Hillyard and collegues (1973) present data which relate the early component of the event-related potential to between-channel selection and the mid components to within-channel selection. It is the timing of these two processes—and some, dependent on matching the semantics of linguistic inputs may take as long or longer than 400 msec—that distinguishes the two. It appears that both stimulus filtering and pigeon-holing can proceed simultaneously but that the pigeon-holing process takes longer to complete.

Keys and Goldberg (unpublished manuscript) in an interesting study using microelectrodes have presented evidence regarding the nature of a variety of such parallel processes. Units in the primary sensory projection systems were found responsive to stimulus relevance (*i.e.*, reinforcing history) and "task difficulty independent of spatial location or task strategy." These results with unit recordings fit more general findings obtained in our laboratory from ensembles of units (Pribram, Spinelli & Kamback, 1967). In these studies stimulus features, response selection and reinforcing contingencies were all found to influence recordings from groups of neurons in the striate cortex of monkeys. Only the stimulus features (stimulus filtering), not response strategies, become encoded in primary visual cortex. Task difficulty determined by response strategy (pigeon-holing) is reflected in the electrical activity of the inferotemporal (posterior intrinsic) association cortex (Rothblatt & Pribram, 1972; Nuwer & Pribram, 1979; Pribram, Day & Johnston, 1976; Bolster & Pribram, in preparation).

In a set of beautifully executed studies on clinical patients, the Velascos (Velasco & Velasco, 1979; Velasco, Velasco, Machado & Olvera, 1973) confirm the distinction between the events initiated in sensory (lemniscal) systems and those which subsequently develop in (extralemniscal) systems whose connections are intrinsic, *i.e.*, restricted to brain stem and brain. Their evidence is in agreement with that obtained from scalp recordings that the early (under 60 msec) components of event-related potentials are related to the extrinsically connected sensory systems. In addition their results go one step further in confirming that indeed these potentials occur in, and only in, the extrinsically connected sensory (lemniscal) systems.

Late components of event-related potentials are shown by the Velascos to be due to processing in intrinsic systems. Lateness could be due to slower conduction times in collaterals from the lemniscal to extralemniscal pathways which is the classical view. Alternatively, generation of activity secondary to that evoked in the entire gamut of sensory connected structures could be responsible for the delayed processing. Timing of event-related activity as recorded from their implanted electrodes indicates that the classical view is in error, that in fact the late components originate in thalamocortical circuitry and only then involve the brain stem. Processing control is top-down.

Processing in the sensory systems is gated by a system of extralemniscal (brain

stem tecto-tegmental) inputs to the reticular nucleus of the thalamus. Rose (1950) and Chow (1952, 1970) demonstrated a front-to-back arrangement of the projections from the reticular nucleus onto cortex. These projections have since been shown to be dependent on connections within the sensory projection thalamus. Furthermore, this nucleus receives an input from an equally exquisite arrangement of fibers from the mesencephalic tectum (n. cuneiformis) and possibly from the supradjacent deep tectum. These tectal inputs are multimodal and show marked spatial congruence: thus each tectal locus can be interpreted as coding for a point in the three-dimensional envelope surrounding the organism. Complimentary data on effector responses show the existence of a tegmental motor map closely matched to the sensory map.

Tecto-tegmental stimulation produces positive going slow waves and temporary *inhibition* of neuronal discharges in the thalamic reticular nucleus. An external stimulus or any prethalamic electrical stimulation of sensory pathways produces a similar inhibitory effect. By contrast, as shown by Skinner (1989), these thalamic reticular nucleus units are *driven* by stimulations of orbitofrontal cortex, inhibiting those of the sensory thalamus. Thus a reciprocal mechanism exists by virtue of the cells of the reticular nucleus of the thalamus: inhibition by tecto-tegmental inputs opens the "gates" for sensory processing: excitation by orbitofrontal activity closes those gates.

The orbitofrontal system is, of course, centered on the amygdala through the uncinate fasciculus (Pribram & MacLean, 1953). Skinner (1989) describes generalized arousal as characterized by "slow onset sustained potentials" elicited in frontal cortex by novel and other meaningful stimuli. Skinner also notes that generalized arousal involves visceroautonomic responses (Kimble, Bagshaw & Pribram, 1965; Grueninger & Grueninger, 1973) sustaining the process which would, when necessary close the thalamic gates to further sensory processing. Habituation occurs.

In the original model, targeted readiness was shown to be a function of the basal ganglia systems. In the analysis of the data obtained from studies using event-related electrical potentials, however, targeted readiness appears to depend on tecto-tegmental input to the reticular nucleus of the thalamus. Is there any evidence of a critical connection between these two sets of systems?

Recall the Velascos' finding that the latency of the responses evoked in the tecto-tegmental system (responses that correspond to the late components of simultaneously recorded scalp potentials) precluded an origin in the adjacent sensory systems. Rather, their data pointed to a top-down thalamic origin of the process.

Recall also the early experiments of Morison and Dempsey in which they showed the effects of amygdalectomy on the late components of the responses which could be evoked by stimulation of the midline and in intralaminar nuclei of the thalamus. The basal ganglia are intimately connected with these midline and intralaminar nuclei (*e.g.*, globus pallidus with the centromedian nucleus).

There is at present no direct evidence that the late components of scalp or tecto-tegmentally recorded event related potentials are the result of midline-intralaminar activity, nor is there any direct evidence of basal ganglia (and cortical) control of such activity. The indirect evidence just noted can only point to the locus of the initiation of inquiry that needs to be undertaken.

SUMMARY AND CONCLUSION

In 1972 when we began to analyze the vast amount of material from the laboratories of physiological psychologists, we had only a vague conceptualization

of what a model of attention might look like. We began where everyone else had, with the view that everything had something to do with "arousal" but with Lacey's (1967) warning in mind that all of the dependent variables might not actually be measuring aspects of the same process.

With this warning in mind, we were forced by the data to organize them into a three-systems mode. Since the first publication of this model in 1975, we have found increasing amounts of evidence to support and extend it. This evidence is briefly reviewed in the present paper in terms of the techniques employed in various types of investigation.

Further, the current review of data has made it possible to specify the para-attentional substrate (the extrinsic lemniscal primary projection systems) upon which the three systems described in the earlier model operate. The earlier model was based on psychophysiological, neurobehavioral and neurochemical analyses while the current specification results from the results of recordings of event-related brain electrical responses. The conclusions derived from these results can be summarized as follows:

First. It has become possible to distinguish controlled attention from the para-attentional pre- and post-attentive automatic processes upon which controls operate.

Second. The pre- and post-attentive processes appear to be coordinate with activity in the extrinsic lemniscal primary sensory projection systems. Processing in these systems is reflected in the early components of event-related brain electrical potentials. These extrinsic systems are, however, not just throughputs for further processing. Rather, they are sensitive to the history of reinforcement which the subject has experienced. The concept of a limited channel capacity must, therefore, be modified to encompass this ability of organisms to improve, through practice, their competence to process a great deal of information in parallel. Competence, not capacity, limits central processing span.

Third. A set of intrinsic extralemniscal processing systems has been identified to operate via a tecto-tegmental pathway to the reticular nucleus of the thalamus. The later components (N_2,P_3, etc.) of event-related potentials have been shown to reflect processing in these systems and those that control them. Activity in these systems has been related to targeted conscious awareness.

Fourth. The late components of the event-related potentials recorded from the intrinsic extralemniscal systems are not due to activation of collaterals from the sensory systems but to top-down influences converging on them in the thalamus.

Fifth. According to our model these top-down influences are, on the one hand, the orbitofrontal-amygdala system responsible for familiarization and, on the other, the basal ganglia system responsible for targeted readiness. As yet, evidence for the latter relationship is only indirect.

Sixth. A third set of systems operates to enhance processing efficiency by modulating the functions of the orbitofronto-amygdala and nigrostriatal systems. This third set converges on the hippocampal system which exerts its influence on familiarization rostrally by way of frontocorticothalamic connections and on readiness posteriorly by way of brain stem connectivities.

Seventh. The components of the event-related electrical brain potentials, when carefully analyzed, differentially reflect the difference between automatic para-attentional and controlled attentional processes. However, little direct evidence regarding interconnections and operations of the systems involved in generating the late event-related components which reflect attentional processes is as yet available. Obtaining such evidence with depth recordings made in animals and in patients should be a high priority objective of future research.

REFERENCES

ABRAHAMS, V. C. & S. M. HILTON. 1958. Active muscle vasodilation and its relation to the "fight and flight reactions" in the conscious animal. J. Physiol. 140: 16–17.

ABRAHAMS, V. C., S. M. HILTON & A. W. ZBROZYNA. 1964. The role of active muscle vasodilation in the alerting stage of the defense reaction. J. Physiol. 171: 189–202.

ANAND, B. K. 1963. Influence of the internal environment on the nervous regulation of alimentary behavior. *In* Brain and Behavior. M. A. B. Brazier, Ed. Vol. II: 43–116. American Institute of Biological Sciences. Washington.

ANAND, B. K. & J. R. BROBECK. 1952. Food intake and spontaneous activity of rats with lesions in the amygdaloid nuclei. J. Neurophysiol. 15: 421–430.

ANDERSON, N. S. & P. M. FITTS. 1958. Amount of information gained during brief exposures of numerals and colors. J. Exp. Psychol. 56: 362–369.

BAGSHAW, M. H. & S. BENZIES. 1968. Multiple measures of the orienting reaction and their dissociation after amygdalectomy in monkeys. Exp. Neurol. 20: 175–187.

BAGSHAW, M. H., D. P. KIMBLE & K. H. PRIBRAM. 1965. The GSR of monkeys during orienting and habituation and after ablation of the amygdala, hippocampus, and inferotemporal cortex. Neuropsychologia 3: 111–119.

BAGSHAW, M. H. & J. D. PRIBRAM. 1968. Effect of amygdalectomy on stimulus threshold of the monkey. Exp. Neurol. 20: 197–202.

BARCHAS, J. D., R. D. CIARANELLO, J. M. STOLK & D. A. HAMBURG. 1972. Biogenic amines and behavior. *In* Hormones and Behavior. S. Levine, Ed. 235–329. Academic Press. New York.

BATESON, P. G. R. 1972. Retardation of discrimination learning in monkeys and chicks previous exposed to both stimuli. Nature 237(5351), 173–174.

BERDINA, N. A., O. L. KOLENKO, I. M. KOTZ, A. P. KUZETZOV, I. M. RODINOV, A. P. SAVTCHENCKO & V. I. THOREVSKY. 1972. Increase in skeletal muscle performance during emotional stress in man. Circ. Res. 6: 642–650.

BLACK, A. H. & G. A. YOUNG. 1972. Electrical activity of the hippocampus and cortex in dogs operantly trained to move and to hold still. J. Comp. Physiol. Psychol. 79: 128–141.

BLACK, A. H., G. A. YOUNG & C. BATENCHUCK. 1970. The avoidance training of hippocampal theta waves in flaxedized dogs and its relation to skeletal movement. J. Comp. Physiol. Psychol. 70: 15–24.

BLAND, B. H. & C. H. VANDERWOLF. 1972a. Diencephalic and hippocampal mechanisms of motor activity in the rat: effect of posterior hypothalamic stimulation on behavior and hippocampal slow wave activity. Brain Res. 43: 67–88.

BLAND, B. H. & C. H. VANDERWOLF. 1972b. Electrical stimulation of the hippocampal formation: behavioral and bioelectrical effects. Brain Res. 43: 89–106.

BOHUS, B. 1976. The hippocampus and the pituitary adrenal system hormones. *In* The Hippocampus. R. L. Isaacson & K. H. Pribram, Eds. Vol. II: 323–353. Plenum Press. New York.

BOLSTER, R. B. & K. H. PRIBRAM. Cortical involvement in visual scan in the monkey. In preparation.

BOWEN, F. P. 1976. Behavioral alterations in patients with basal ganglia lesions. *In* The Basal Ganglia. M. D. Yahr, Ed. Raven Press. New York.

BROADBENT, D. E. 1977. The hidden preattentive process. Am. Psychol. 32(2), 109–118.

BRUNER, J. S. 1957. On perceptual readiness. Psychol. Rev. 64: 123–152.

BUTCHER, L. L., N. J. WOOLF, A. ALBANESE & S. H. BUTCHER. 1981. Cholinergic-monoaminergic interactions in selected regions of the brain: histochemical and pharmacologic analyses. *In* Cholinergic Mechanisms. G. Pepeu & H. Ladinsky, Eds. 723–738. Plenum Press. New York.

CHAMPNEY, T. F., T. L. SAHLEY & C. A. SANDMAN. 1976. Effects of neonatal cerebral ventricular injection of ACTH 4-9 and subsequent adult injections on learning in male and female albino rats. Pharmacol. Biochem. Behav. 5: 3–10.

CHOW, K. L. 1952. Regional degeneration of the thalamic reticular nucleus following cortical ablations in monkeys. J. Comp. Neurol. 97(1), 37–59.

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

CLEMENTE, C. C., M. B. STERMAN & W. WYRWICKE. 1964. Post-reinforcement EEG synchronization during alimentary behavior. Electroencephalogr. Clin. Neurophysiol. **16:** 355–365.

COOPER, J. R., F. E. BLOOM & R. H. ROTH. 1978. The Biochemical Basis of Neuropharmacology. Oxford University Press. New York.

CROWNE, D. P., A. KONOW, K. J. DRAKE & K. H. PRIBRAM. 1972. Hippocampal electrical activity in the monkey during delayed alternation problems. Electroencephalogr. Clin. Neurophysiol. **33:** 567–577.

CROWNE, D. P. & W. I. RIDDELL. 1969. Hippocampal lesions and the cardiac component of the orienting response in the rat. J. Comp. Physiol. Psychol. **69:** 748–755.

DALTON, A. & A. H. BLACK. 1968. Hippocampal electrical activity during the operant conditioning of movement and refraining from movement. Commun. Behav. Biol. **2:** 267–273.

DAVIDSON, J. M., L. E. JONES & S. LEVINE. 1968. Feedback regulation of adrenocorticotropin secretion in "basal" and "stress" conditions: acute and chronic effects of intrahypothalamic corticoid implantation. Endocrinology **82:** 655–663.

DELONG, M. R. & P. L. STRICK. 1974. Relation of basal ganglia, cerebellum, and motor cortex to romp and ballistic limb movements. Brain Res. **71:** 327–335.

DENNY-BROWN, D. & N. YANAGISAWA. 1976. The role of the basal ganglia in the initiation of movement. *In* The Basal Ganglia. M. D. Yahr, Ed. Raven Press. New York.

DE WIED, D. 1974. Pituitary-adrenal system hormones and behavior. *In* The Neurosciences, Third Study Program. F. O. Schmitt & F. G. Worden, Eds. 653–666. MIT Press. Cambridge, MA.

DONCHIN, E. & G. H. COLES. 1988. Is the P300 component a manifestation of context updating? Behav. Brain Sci. **11:** 357–374.

DONCHIN, E., L. A. GERBRANDT, L. LEIFER & L. TUCKER. 1972. Is the contingent negative variation contingent on a motor response? Psychophysiology **9:** 178–188.

DONCHIN, E., D. OTTO, L. K. GERBRANDT & K. H. PRIBRAM. 1971. While a monkey waits: electrocortical events recorded during the foreperiod of a reaction time study. Electroencephalogr. Clin. Neurophysiol. **31:** 115–127.

DONCHIN, E., D. OTTO, L. K. GERBRANDT & K. H. PRIBRAM. 1973. While a monkey waits. *In* Psychophysiology of the Frontal Lobes. K. H. Pribram & A. R. Luria, Eds. 125–138. Academic Press. New York.

DOUGLAS, R. J. & K. H. PRIBRAM. 1966. Learning and limbic lesions. Neuropsychologia **4:** 197–220.

DOUGLAS, R. J. & K. H. PRIBRAM. 1969. Distraction and habituation in monkeys with limbic lesions. J. Comp. Physiol. Psychol. **69:** 473–480.

ELLIOTT, R., B. BANKART & T. LIGHT. 1970. Differences in the motivational significance of heart rate and palmar conductance: two tests of a hypothesis. J. Pers. Soc. Psychol. **14:** 166–172.

ENDROCZI, E. 1972. Pavlovian conditioning and adaptive hormones. *In* Hormones and Behavior. S. Levine, Ed. 173–207. Academic Press. New York.

ERDELYI, M. H. 1974. A new look at the new look: perceptual defense and vigilance. Psychol. Rev. **81(1),** 1–25.

FAIR, C. M. 1965. The Physical Foundations of the Psyche: a Neurophysiological Study. Wesleyan University Press. Middletown, CT.

FIBIGER, H. C., A. G. PHILLIPS & R. A. CLOUSTON. 1973. Regulatory deficits after unilateral electrolytic or 6-OHDA lesions of the substantia nigra. Am. J. Physiol. **225:** 1282–1287.

FULLER, J. L., H. E. ROSVOLD & K. H. PRIBRAM. 1957. The effect on affective and cognitive behavior in the dog of lesions of the pyriform-amygdala-hippocampal complex. J. Comp. Physiol. Psychol. **50:** 89–96.

FUXE, E. 1977. The dopaminergiz pathways. Proc. Am. Neuropathol. Assoc.

GAILLARD, A. W. K. 1977. The late CNV wave: preparation versus expectancy. Psychophysiology **14:** 563–568.

GARNER, W. R. 1962. Uncertainty and Structure as Psychological Concepts. Wiley New York.

GASTAUT, H. 1954. Interpretation of the symptoms of "psychomotor" epilepsy in relation to physiologic data on Rhinencephalic function. Epilepsia **3:** 84–88.

GERMANA, J. 1968. Response characteristics and the orienting reflex. J. Exp. Psychol. **78:** 610–616.

GERMANA, J. 1969. Autonomic-behavioral integration. Psycholphysiology **6:** 78–90.

GOLDSTEIN, M. 1974. Brain research and violent behavior. Arch. Neurol. **30:** 1–35.

GOLDMAN-RAKIC, P. S. & M. L. SCHWARTZ. 1982. Interdigitation of contralateral and ipsilateral columnar projections to frontal association cortex in primates. Science **216:** 755–757.

GRASTYAN, E. 1959. The hippocampus and higher nervous activity. *In* The Central Nervous System and Behavior. M. A. B. Brazier, Ed. Josiah Macy, Jr. Foundation. New York.

GRASTYAN, E., K. LISSAK, I. MADARASZ & H. DONOFFER. 1959. Hippocampal electrical activity during the development of conditioned reflexes. Electroencephalogr. Clin. Neurophysiol. **11:** 409–430.

GREEN, J. F. & A. ARDUINI. 1954. Hippocampal electrical activity in arousal. J. Neurophysiol. **17:** 533–557.

GRIMM, Y. & J. W. KENDALL. 1968. A study of feedback suppression of ACTH secretion utilizing glucocorticoid implants in the hypothalamus: the comparative effects of cortisol, cortisterone and their 20 acetates. Neuroendocrinology **3:** 55–63.

GROVES, P. M. & R. F. THOMPSON. 1970. Habituation: a dual-purpose theory. Psychol. Rev. **77:** 419–450.

GRUENINGER, W. E. & J. GRUENINGER. 1973. The primate frontal cortex and allassostasis. *In* Psychophysiology of the Frontal Lobes. K. H. Pribram & A. R. Luria, Eds. Academic Press. New York.

GRUZELIER, J. H. & P. H. VENABLES. 1972. Skin conductance orienting activity in a heterogeneous sample of schizophrenics. J. Nerv. Ment. Dis. **155:** 277–287.

HALGREN, E., N. K. SQUIRES, C. L. WILSON, J. W. ROHRBAUGH, T. L. BABB & P. H. CRANDALL. 1980. Endogenous potentials generated in the human hippocampal formation and amygdala by infrequent events. Science **210:** 803–806.

HEILMAN, K. M. & E. VALENSTEIN. 1972. Frontal lobe neglect. Neurology **28:** 229–232.

HEILMAN, K. M. & R. T. WATSON. 1977. Mechanisms underlying the unilateral neglect syndrome. *In* Advances in Neurology. Vol. 18. Hemi-Inattention and Hemisphere Specialization. E. A. Weinstein and R. P. Friedland, Eds. Raven Press. New York.

HERNANDEZ, L. & B. G. HOEBEL. 1978. Hypothalamic reward Vol. and aversion: a link between metabolism and behavior. *In* Current Studies of Hypothalamic Function. **2:** 72–92. Karger. Basel.

HILLYARD, S. A., R. F. HINK, U. L. SCHWENT & T. W. PICTOR. 1973. Electrical signs of selective attention in the human brain. Science. **182:** 177–180.

HILLYARD, S. A., K. C. SQUIRES, J. W. BAVER & P. H. LINDSAY. 1971. Evoked potential correlates of auditory signal detection. Science **172:** 1357–1360.

HIRST, W., E. S. SPELKE, C. C. REAVES, G. CAHARACK & U. NEISSER. 1980. Dividing attention without alternation or automaticity. J. Exp. Psychol. **109:** 98–117.

HOEBEL, B. G. 1974. Brain reward and aversion systems in the control of feeding and sexual behavior. J. K. Cole & T. B. Sanderegger, Eds. Nebr. Symp. Motiv. **22:** 49–112.

HOEBEL, B. G. 1976. Brain-stimulation reward and aversion in relation to behavior. *In* Brain-Stimulation Reward. A. Wauquier & E. T. Rolls, Eds. 335–372. North-Holland. Amsterdam.

ISAACSON, R. L., A. J. NONNEMAN & L. W. SCHUALTZ. 1968. Behavioral and aurtomical sequalae of the infant limbic system. *In* The Neuropsychology of Development: a Symposium. R. L. Isaacson, Ed. Wiley. New York.

JOUVET, M. 1974. Monoanesnergic regulation of the sleep-waking cycle in the cat. Neurosciences **3:** 499–508.

JUNG, R. & A. E. KORNMUELLER. 1938. Eine methodik der Abkitung lokalisierter Potentialschwankungen aus sobcorticalen Hirngebieten. Arch. Psychiatr. Nervenkr. **109**: 1–30.

KAADA, B. R., K. H. PRIBRAM & J. A. EPSTEIN. 1949. Respiratory and vascular responses in monkeys from temporal pole, insula, orbital surface and cingulate gyrus: a preliminary report. J. Neurophysiol. **12**: 347–356.

KEYS, W. & M. E. GOLDBERG. Unit potentials. Unpublished manuscript.

KIMBLE, D. P., M. H. BAGSHAW & K. H. PRIBRAM. 1965. The GSR of monkeys during orienting and habituation after selective partial ablations of the cingulate and frontal cortex. Neuropsychologia **3**: 121–128.

KING, M. B. & B. G. HOEBEL. 1968. Killing elicited by brain stimulation in rats. Commun. Behav. Biol. **2**: 173–177.

KONRAD, K. W. & M. H. BAGSHAW. 1970. Effect of novel stimuli on cats reared in a restricted environment. J. Comp. Physiol. Psychol. **70**: 157–164.

KORNHUBER, H. H. & L. DEECKE. 1965. Hirnpotentialaenderungen bei Wilkurbewegungen und passiven Bewegungen des Menschen: Bereitschaftspotential und reafferent Potentiale. Pflügers Arch. gesamte Physiol. Menschen Tiere **284**: 1–17.

LACEY, J. I. 1967. Somatic response patterning and stress: some revisios of activation theory. *In* Psychological Stress: Issues in Research. M. H. Appley & R. Trumball, Eds. Appleton-Century-Crofts. New York.

LACEY, J. I. & B. C. LACEY. 1970. Some autonomic central nervous system interrelationships. *In* Physiological Correlates of Emotion. P. Black, Ed. 205–227. Academic Press. New York.

LADINSKY, H., S. CONSOLO, A. S. TIRELLI, G. L. FORLONI & M. SEGAL. 1981. Regulation of cholinergic activity in the rat hippocampus: *in vivo* effects of oxotremorine and fenfluramine. *In* Cholinergic Mechanisms. G. Pepeu & H. Ladinsky, Eds. 781–793. Plenum Press. New York.

LEVINE, S. & L. E. JONES. 1965. Adrenocorticotropic hormone (ACTH) and passive avoidance learning. J. Comp. Physiol. Psychol. **59**: 357–360.

LEVINE, S., W. P. SMOTHERMAN & J. W. HENNESSY. 1977. Pituitary-adrenal hormones and learned taste aversion. *In* Neuropeptide Influences on the Brain and Behavior. L. H. Miller, C. A. Sandman & A. J. Kastin, Eds. Raven Press. New York.

LINDSLEY, D. B. 1961. The reticular activating system and perceptual integration. *In* Electrical stimulation of the brain. D. E. Sheer, Ed. Austin: University of Texas Press.

LINDSLEY, D. B. & C. L. WILSON. 1976. Brainstem-hypothalamic systems influencing hippocampal activity and behavior. *In* The Hippocampus. R. L. Isaacson & K. H. Pribram, Eds. Vol. **2**: 247–274. Plenum Press. New York.

LOGAN, G. D. 1979. On the use of a concurrent memory load to measure attention and automaticity. J. Exp. Psychol. **5**: 189–207.

LURIA, A. R., K. H. PRIBRAM & E. D. HOMSKAYA. 1964. An experimental analysis of the behavioral disturbance produced by a left frontal arachnoidal endothelioma (meningioma). Neuropsychologia **2**: 257–280.

MACADAR, A. W., L. M. CHALUPA & D. B. LINDSLEY. 1974. Differentiation of brain stem loci which affect hippocampal and neocortical electrical activity. Exp. Neurol. **43**: 499–514.

MAGOUN, H. W. 1958. The Waking Brain. Charles C. Thomas. Springfield, IL.

MAHUT, H. 1971. Spatial and object reversal learning in monkeys with partial temporal lobe ablations. Neuropsychologia **9**: 409–424.

MATTHYSSE, S. 1974. Schizophrenia: Relationship to Dopamine transmission, motor, control, and feature extraction. Neurosciences **3**: 733–737.

MCCLEARY, R. A. 1961. Response specificity in the behavioral effects of limbic system lesions in the cat. J. Comp. Physiol. Psychol. **54**: 605–613.

MCEWEN, B. S., J. L. GERLACH & D. J. MICCO. 1976. Putative glucocorticoid receptors in hippocampus and other regions of the rat brain. *In* The Hippocampus. R. L. Isaacson & K. H. Pribram, Eds. Vol. **2**: 285–322. Plenum Press. New York.

MCFARLAND, D. J. 1971. Feedback Mechanisms in Animal Behaviour. Academic Press. New York.

MEDNICK, S. A. & F.SCHULSINGER. 1968. Some premorbid characteristics related to breakdown in children with schizophrenic mothers. *In* The Transmission of Schizophrenia. D. Rosenthal & S. S. Kety, Eds. 267–291. Pergamon Press. New York.

MILLER, G. A. 1956. The magical number seven, plus or minus two, or, some limits on our capacity for processing information. Psychol. Rev. 63: 81–97.

MILLER, G. A., E. H. GALANTER & K. H. PRIBRAM. 1960. Plans and the Structure of Behavior. Holt, Rinehart & Winston. New York.

MILLER, L. H., C. A. SANDMAN & A. J. KASTIN. 1977. Neuropeptide Influences on the Brain and Behavior. Raven Press. New York.

MILNER, B. 1958. Psychological defects produced by temporal lobe excision. *In* The Brain and Human Behavior. H. C. Solomon, S. Cobb & W. Penfield, Eds. Williams & Wilkins. Baltimore, MD.

MORISON, R. S., E. W. DEMPSEY & B. R. MORISON. 1941. Cortical responses from electrical stimulation of the brain stem. Am. J. Physiol. 131: 732–743.

MORUZZI, G. & H. W. MAGOUN. 1949. Brain stem reticular formation and activation of the EEG. Electroencephalogra. Clin. Neurophysiol. 1: 455–473.

NAATENEN, R. 1982. Processing negativity. Psychol. Bull. 92(3), 605–640.

NAATENEN, R. 1990. The role of attention in auditory information processing as revealed by event-related potentials and other brain measures of cognitive function. Behav. Brain Sci. 13: 201.288.

NONNEMAN, A. J. & R. L. ISAACSON. 1973. Task dependent recovery after early brain change. Behav. Biol. 8: 143–172.

NUWER, M. R. & K. H. PRIBRAM. 1979. Role of the inferotemporal cortex in visual selective attention. J. Electroencephalogr. Clin. Neurophysiol. 46: 389–400.

ODERFELD-NOWAK, B. & M. H. APRISON. 1981. On modulation of cerebral cholinergic mechanisms by endogenous indoleamines and their derivatives. *In* Cholinergic Mechanisms. G. Pepeu & H. Ladinsky, Eds. 739–761. Plenum Press. New York.

POSNER, M. I. 1973. Coordination of internal codes. *In* Visual Information Processing. W. G. Chase, Ed. 35–73. Academic Press. New York.

PRIBRAM, K. H. 1960a. The intrinsic systems of the forebrain. *In* Handbook of Physiology, Neurophysiology II. J. Field, H. W. Magoun & V. E. Hall, Eds. American Physiological Society. Washington, DC.

PRIBRAM, K. H. 1961. Limbic system. *In* Electrical Stimulation of the Brain. D. E. Sheer, Ed. 311–320. University of Texas Press. Austin.

PRIBRAM, K. H. 1962. Interrelations of psychology and neurological disciplines. *In* Psychology: a Study of a Science. Vol. 4. Biologically Oriented Fields: Their Place in Psychology and in Biological Sciences. S. Koch, Ed. 119–157. McGraw-Hill. New York.

PRIBRAM, K. H. 1963. Reinforcement revisited: a structural view. *In* Nebraska Symposium on Motivation. Vol. 11. M. Jones, Ed. 113–159. University of Nebraska Press. Lincoln.

PRIBRAM, K. H. 1969. Neural servosystems and the structure of personality. J. Nerv. Ment. Dis. 140: 30–39.

PRIBRAM, K. H. 1971. Languages of the Brain: Experimental Paradoxes and Principles in Neuropsychology. Prentice-Hall. Englewood Cliffs, NJ.

PRIBRAM, K. H. 1974. How is it that sensing so much we can do so little? *In* Central Processing of Sensory Input. K. H. Pribram, Contrib. Ed. The Neurosciences Third Study Program. F. O. Schmitt & F. G. Worden, Eds. 249–261. MIT Press. Cambridge, MA.

PRIBRAM, K. H. 1976. Mind—it does matter. *In* Philosophical Dimensions of the Neuromedical Sciences. S. F. Spicker & H. T. Englehardt, Jr., 97–111. D. Reidel. Dordrecht, Holland.

PRIBRAM, K. H. 1977. Peptides and protocritic processes. *In* Psychopathology and Brain Dysfunction. L. H. Miller, C. L. Sandman & A. J. Kastin, Eds. 77–95. Raven Press. New York.

PRIBRAM, K. H. 1987. Subdivisions of the frontal cortex revisited. *In* The Frontal Lobes Revisited. E. Brown and E. Perecman, Eds. 11–39. IRBN Press. New York.

PRIBRAM, K. H. & M. H. BAGSHAW. 1953. Further analysis of the temporal lobe syndrome utilizing frontotemporal ablations in monkeys. J. Comp. Neurol. 99: 347–375.

PRIBRAM, K. H., R. U. DAY & V. S. JOHNSTON. 1976. Selective attention: distinctive brain electrical patterns produced by differential reinforcement in monkey and man. *In* Behavior Control and Modification of Physiological Activity. D. I. Mostofsky, Ed. 89–114. Prentice-Hall. Englewood Cliffs, NJ.

PRIBRAM, K. H., R. J. DOUGLAS & B. J. PRIBRAM. 1969. The nature of nonlimbic learning. J. Comp. Physiol. Psychol. 69: 765–772.

PRIBRAM, K. H. & R. L. ISAACSON. 1976. The Hippocampus, Vol. 2: Neurophysiology and Behavior. Plenum. New York.

PRIBRAM, K. H. & L. KRUGER. 1954. Functions of the "olfactory brain". Ann. N. Y. Acad. Sci. 58: 109–138.

PRIBRAM, K. H., H. LIM, R. POPPEN & M. H. BAGSHAW. 1966. Limbic lesions and the temporal structure of redundancy. J. Comp. Physiol. Psychol. 61: 368–373.

PRIBRAM, K. H. & P. D. MACLEAN. 1953. Neuronographic analysis of medial and basal cerebral cortex. II. J. Neurophysiol. 16: 324–340.

PRIBRAM, K. H. & D. McGUINNESS. 1975. Arousal, activation, and effort: separate neural systems. *In* Brain Work: the Coupling of Function, Metabolism and Blood Flow in the Brain. D. H. Ingvar & N. A. Lassen, Eds. 428–451. Alfred Benzon Foundation. Copenhagen.

PRIBRAM, K. H., S. REITZ, M. McNEIL & A. A. SPEVACK. 1979. The effect of amygdalectomy on orienting and classical conditioning in monkeys. Pavlovian. J. 14(4), 203–217.

PRIBRAM, K. H., D. N. SPINELLI & M. C. KAMBACK. 1967. Electrocortical correlates of stimulus response and reinforcement. Science 157: 94–96.

RAPHELSON, A. C., R. L. ISAACSON & R. J. DOUGLAS. 1965. The effect of distracting stimuli on the runway performance of limbic damaged rats. Psychon. Sci. 3: 483–484.

REIS, D. J. 1974. The chemical coding of aggression in brain. *In* Neurohumeral Coding of Brain Function. R. D. Myers & R. R. Drucker-Colin, Eds. 125–150.

RIDDELL, W. L., L. A. ROTHBLAT & W. A. WILSON, JR. 1969. Auditory and visual distraction in hippocampectomized rats. J. Comp. Physiol. Psychol. 67: 216–219.

ROBINSON, S. E., D. L. CHENEY & E. COSTA. 1981. Regulation of septal-hippocampal cholinergic neurons by catecholamines. *In* Cholinergic Mechanisms. G. Pepeu & H. Ladinsky, Eds. 705–713. Plenum Press. New York.

ROSE, J. 1950. The cortical connections of the reticular complex of the thalamus. Patterns of Organization in the Central Nervous System 30: 454–479.

ROSVOLD, H. E., A. F. MIRSKY & K. H. PRIBRAM. 1954. Influence of amygdalectomy on social interaction in a monkey group. J. Comp. Physiol. Psychol. 47: 173–178.

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

RUSSELL, R. W., G. SINGER, F. FLANAGAN, M. STONE & J. W. RUSSELL. 1968. Quantitative relations in amygdala modulation of drinking. Physiol. Behav. 3: 871–875.

SANDMAN, C. A., J. GEORGE, J. D. NOLAN & A. J. KASTIN. 1975. Enhancement of attention in man with ACTH/MSH 4-10. Physiol. Behav. 15: 427–431.

SAUERLAND, E. K. & C. D. CLEMENTE. 1973. The role of the brain stem in orbital cortex induced inhibition of somatic reflexes. *In* Psychophysiology of the Frontal Lobes. K. H. Pribram & A. R. Luria, Eds. 167–184. Academic Press. New York.

SCHWARTZBAUM, J. S., W. A. WILSON, JR. & J. R. MORRISSETTE. 1961. The effects of amygdalectomy on locomotor activity in monkeys. J. Comp. Physiol. Psychol. 54: 334–336.

SEMMES, J., S. WEINSTEIN, L. GHENT & H. L. TEUBER. 1963. Correlates of impaired orientation in personal and extrapersonal space. Brain 86: 747–772.

SHARPLESS, S. & H. JASPER. 1956. Habituation of the arousal reaction. Brain 79: 655–680.

SHERRINGTON, C. 1955. Man on His Nature. Doubleday. Garden City, NY.

SIMON, H. A. 1974. How big is a chunk? Science 183: 482–488.

SKINNER, B. F. 1989. The origins of cognitive thought. Am. Psychol. 44(1), 13–18.

SKINNER, B. F. & D. B. LINDSLEY. 1973. The nonspecific medio-thalamic-fronto-cortical system: its influence on electrocortical activity and behavior. *In* Psycholphysiology of the Frontal Lobes. K. H. Pribram & A. R. Luria, Eds. 185–234. Academic Press. New York.

SNYDER, S. H., R. SIMANTOV & G. W. PASTERNAK. 1976. The brain's own morphine, "enkelphalin:" a peptide neurotransmitter? Soc. Neurosci. 1:

SOKOLOV, E. N. 1960. Neuronal models and the orienting reflex. *In* The Central Nervous System and Behavior. M. A. B. Brazier, Ed. Josiah Macy, Jr. Foundation. New York.

SOKOLOV, E. N. 1963. Perception and the Conditioned Reflex. MacMillan. New York.

SOLOMON, R. L. 1980. The opponent-process theory of acquired motivation: the cost of pleasure and the benefits of pain. Am. Psychol. 35: 691–712.
SPEVACK, A. & K. H. PRIBRAM. 1973. A decisional analysis of the effects of limbic lesions in monkeys. J. Comp. Physiol. Psychol. 82: 211–226.
STEIN, L. 1968. Chemistry of reward and punishment. *In* Psychopharmacology. a Review of Progress, 1957–1967. D. H. Effron, Ed. 105–135. U.S. Government Printing Office. Washington, DC.
STRAND, F. L., A. CAYER, E. GONZALES & H. STOBOY. 1976. Peptide enhancement of neuromuscular function: animal and clinical studies. Pharmacol. Biochem. Behav. 5: 179–188.
TEECE, J. J. 1972. Contingent negative variation (CNV) and psychological processes in man. Psychol. Rev. 69: 74–90.
TEITELBAUM, P. 1955. Sensory control of hypothalamic hyperphagia. J. Comp. Physiol. Psychol. 48: 156–163.
TEITELBAUM, P. & P. MILNER. 1963. Activity changes following partial hippocampal lesions in rats. J. Comp. Physiol. Psychol. 56: 284–289.
TEITELBAUM, P. & A. N. EPSTEIN. 1962. The lateral hypothalamus syndrome: recovery of feeding and drinking after lateral hypothalamic lesions. Psychol. Rev. 69: 74–90.
UNGERSTEDT, U. 1974. Brain dopamine neurons and behavior. *In* The Neurosciences Third Study Program. F. O. Schmitt & F. G. Worden, Eds. 695–704. MIT Press. Cambridge, MA.
URSIN, H. & B. R. KAADA. 1960. Functional localization within the amygdala complex within the cat. Electroencephalogr. Clin. Neurophysiol. 12: 120.
VAN WIMERSMA GREIDANUS, TJ. B. & D. DE WIED. 1976. The dorsal hippocampus: a site of action of neuropeptides on avoidance behavior? Pharmacol. Biochem. Behav. 5: 29–34.
VANDERWOLF, C. H. 1969. Hippocampal electrical activity and voluntary movement in the rat. Electroencephalogr. Clin. Neurophysiol. 26: 407–418.
VANDERWOLF, C. H. 1971. Limbic-diencephalic mechanisms of voluntary movement. Psychol. Rev. 78: 83–113.
VAUGHAN, H. G., L. D. COSTA & W. RITTER. 1968. Topography of the human motor potential. Electroencephalogr. Clin. Neurophysiol. 25: 1–10.
VELASCO, F. & N. VELASCO. 1979. A reticulo-thalamic system mediating propriceptive attention and tremor in man. Neurosurgery 4: 30–36.
VELASCO, N., F. VELASCO, J. MACHADO & A. OLVERA. 1973. Effects of novelty, habituation, attention, and distraction on the amplitudes of the various components of the somatic evoked responses. Int. J. Neurosci. 5: 30–36.
VERLEGER, R. 1988. Event-related potentials and cognition: a critique of the context updating hypothesis and an alternative interpretation of P3. Behav. Brain Sci. 11: 343–427.
WALL, P. D. & G. D. DAVIS. 1951. Three cerebral cortical systems affecting autonomic function. J. Neurophysiol. 14: 507–517.
WALTER, W. G., R. COOPER, V. J. ALDRIDGE, W. C. MCCALLUM & A. L. WINTER. 1964. Contingent negative variation: an electric sign of sensorimotor association and expectancy in the human brain. Nature 23: 380–384.
WEINBERG, H. 1972. The contingent negative variation: its relation to feedback and expectant attention. Neuropsychologia 10: 299–306.
WHISHAW, I. Q., B. H. BLAND & C. H. VANDERWOLF. 1972. Hippocampal activity, behavior, self-stimulation, and heart rate during electrical stimulation of the lateral hypothalamus. J. Comp. Physiol. Psychol. 79: 115–127.
WICKLEGREN, W. O. & R. L. ISAACSON. 1963. Effect of the introduction of an irrelevant stimulus on runway performance of the hippocampectomized rat. Nature 200: 48–50.
WILCOTT, R. C. & C. E. HOEL. 1973. Arousal response to electrical stimulation of the cerebral cortex in cats. J. Comp. Physiol. Psychol. 85(2), 413–420.
WRIGHT, J. J. 1979. Changed cortical activation and the lateral hypothalamic syndrome: a study in the split-brain cat. Brain Res. 151: 632–636.
WRIGHT, J. J. 1980a. Intracranial self-stimulation, cortical arousal, and the sensorimotor neglect syndrome. Exp. Neurol. 64.
WRIGHT, J. J. 1980b. Visual evoked response in lateral hypothalamic neglect. Exp. Neurol. 65.