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Arousal, Activation and Effort: Separate Neural Systems

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In summary, the remainder of this volume attests to the rapid strides in our knowledge that are being made in the quantitative determination of local cerebral blood flow and in detailing the connection between such determinations and cerebral metabolism. In this chapter, we want to present data that originate from a different orientation: a neurophysiological and behavioral analysis which can provide a broader context within which the metabolic data become psychologically meaningful. An extensive review of such data relevant to this issue, data obtained in our own laboratories and elsewhere, was prepared but, due to limitations of space, has been abridged for presentation here. The full account appears in the journal, *Psychological Review* (Pribram & McGuinness 1975).

Ask the man in the street to describe the occasions that demand brain work and he will most likely have recourse to such phrases as: "when you *pay* attention"; and "the *effort* it takes to solve problems". Brain physiologists and psychologists often address the same issue by suggesting that the brain is working when it is "aroused" or "activated". For the most part the terms attention, effort, arousal and activation are used interchangeably by layman and scientist alike (see for example Kahneman 1973). However, we have reason to believe that the data currently available clearly distinguish a variety of brain work processes and that *different brain systems* are involved in each.

Specifically, we distinguish between a neural system involved in *arousal*, a phasic reaction to input and another involved in *activation*, a tonic

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readiness to respond. And we also discern a brain circuit that coordinates arousal and activation, a process that demands resistance to or the initiation of rapid shifts in cerebral metabolism and is experienced as *effort*. Here is some of the evidence upon which these distinctions are based.

AROUSAL.

The Neuronal Representation of Input

Arousal is said to occur when an input change produces a measurable incrementing of a physiological (e.g., single unit recording of neural potentials; galvanic skin response) or behavioral (e.g., response amplitude of a spinal reflex; frequency of a locomotor response) indicator over a baseline. The types of input change that produce arousal have been studied extensively and have been labeled by Berlyne (1969) as collative (def., to collect and compare carefully in order to arrange into informative order) variables. These include sudden changes in intensity to which the organism is unaccustomed; changes in the timing of inputs; and changes in the ground in which a stimulus figure appears. In short, arousal results when, in the history of the organism's experience, an input is scarce, surprising, complex and novel. Such collative characteristics also define the concept "information" as it is used in the study of communication systems (e.g., Brillouin 1962); thus it has become customary to treat organisms subject to arousal as "information processing systems". Inherent in such treatment is the assumption that the input is matched against some residual in the organism of his past experience, or some competence (Miller *et al.* 1960, Pribram 1971). Without such matching, there could be no novelty or information, nor even a measure of change in intensity.

During the 1960's a great deal of experimental evidence accumulated to substantiate the assumption that an organism's experience results in a residual in the nervous system. Behavioral research was addressed to issues such as adaption level (Helson 1964), expectancy (Bruner 1957) and the development of "neuronal models" (Sokolov 1960, 1963) while neuroscientists were content to demonstrate the occurrence of any permanent or semipermanent modifiability of neural tissue (see Pribram & Broadbent 1970, Horn & Hinde 1970 for reviews).

The Corebrain Arousal System

An extensive series of experiments (reviewed by Groves & Thompson 1970) has distinguished a system of "arousal" neurons in the medial portions of the spinal cord. This system of neurons converges with another more laterally placed set of decrementing neurons onto a final common path that habituates and dishabituates much as does the behavior in which these neural systems are involved. There is every reason to believe that the rostral extension into the mesencephalic brain stem of this 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 rostrally in the diencephalon in a continuation of this neuron system into the hypothalamus where episodes of fighting and fleeing are produced by electrical or chemical stimulation to the so-called "defense" region of the hypothalamus. These can be related to the orienting reaction. Abrahams & 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 pilo erection occur. In the later study, alerting behaviors were measured in greater detail, and during mild stimulation the authors observed changes in pupil dilation, head movement, pricking the ears, respiration and blood flow. 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.

The Amygdala Circuits and the Control of Arousal

Converging on these hypothalamic structures are two reciprocally acting circuits regulating arousal. These circuits center on the amygdala (see the more extensive review by Pribram & McGuinness (1975); Figs. 1, 2, 3, 4 and Table I). One of these circuits involves the dorsolateral frontal cortex and is excitatory since resections of this structure *invariably* eliminate visceral-autonomic orienting responses. The other, opposite in function, is

more likely related to the orbitofrontal cortex which has been shown to be the rostral pole of an extensive inhibitory pathway (Kaada *et al.* 1949, Wall & Davis 1951, Pribram 1961, Skinner & Lindsley 1973, Sauerland & Clemente 1973). These data relate to the early descriptions of the behavior of amygdalectomized animals (Pribram & Bagshaw 1953) which focused on the fact that they were tame, unresponsive to threat and non-aggressive. However, the opposite finding was also occasionally observed (e.g. Rosvold *et al.* 1954) and more recent behavioral studies by Ursin & Kaada (1960) using restricted lesions and electrical stimulations have confirmed the suggestion that at least two more or less reciprocal system can be identified in the amygdala.

Such reciprocal innervation allows sensitive modulation (tuning) of the arousal mechanism. This is in accord with evidence on other control functions of the amygdala and related structures. Thus, injections of carbachol into the appropriate hypothalamic site will initiate drinking; such injections 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 in an exquisitely accurate relationship (Russell *et al.* 1968). Extrapolating to the issue before us, the fronto-amygdala influence can be conceived as a finely tuned determinant controlling viscero-autonomic arousal initiated by the hypothalamic mechanism during orienting. 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 – and not only their cessation, but also their initiation (Fuller *et al.* 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). So far, when we have described psychophysiological data including changes in EEG and evoked potentials, we have referred to phasic, i.e., trial by trial changes in the initial period of observation. These changes to which we have related arousal often reach asymmptote within 3–5 stimulus presentations. But, we also recorded longer term sustained tonic changes in the response measures, which, as we shall see in the next sections, are indicative of activation and effort. That baseline changes occur after amygdala

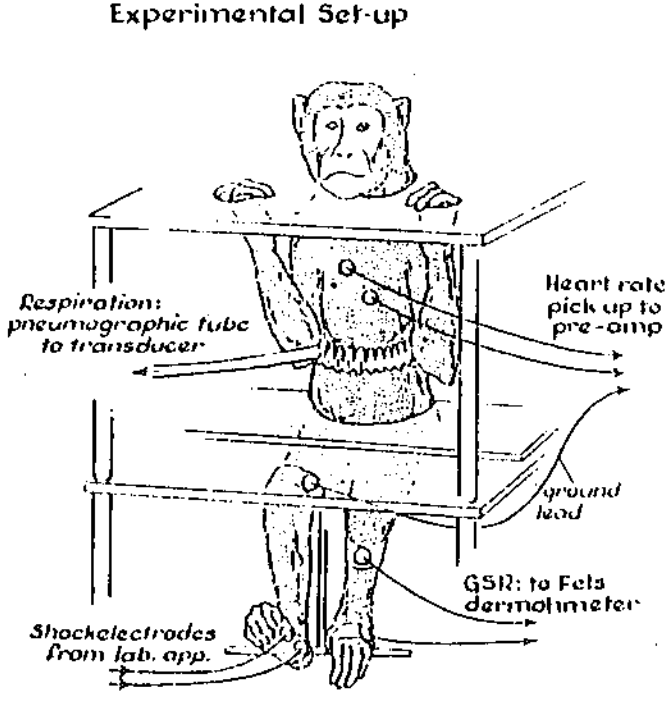

Fig. 1. Picture showing the set-up in which GSR, heart rate, respiratory rate, and EEG were recorded from monkeys during habituation studies.

lesions is demonstrated by various studies in which we showed that although behavioral and some electrocortical responses appeared to be normal during orienting (Schwartzbaum *et al.* 1961, Bagshaw & Benzies 1968) the background level of these responses is lower than for controls. Ear flicking is practically absent during interstimulus intervals, 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.* 1974). These results indicate that at the forebrain level, just as at the spinal level in Thompson's experiments (Groves & Thompson 1970) arousal and decrementing systems converge to produce orienting, habituation, and dishabituation.

Perhaps the most striking tonic psychophysiological change to follow amygdalectomy was the finding of a paradoxically elevated basal heart rate

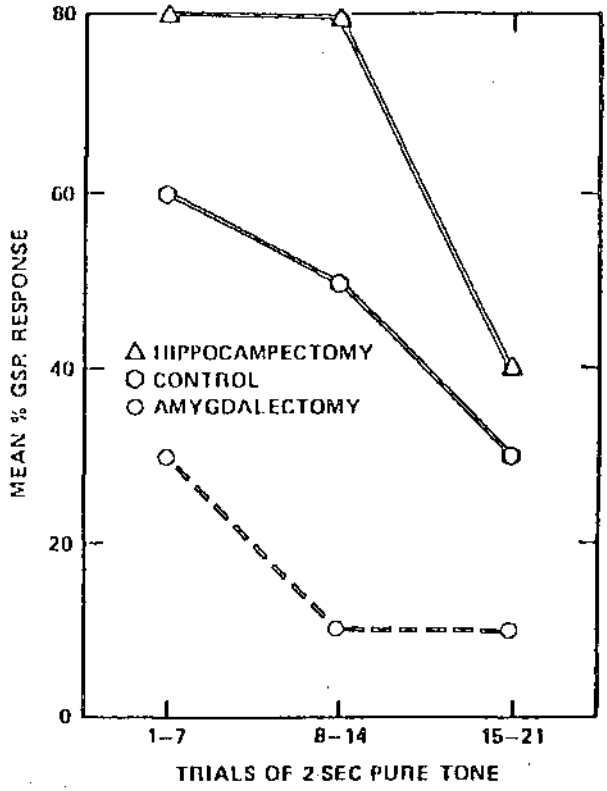

Fig. 2. Curves for an analysis of the first 21 trials (split into 7 trial blocks) on % of GSR response to a 2 sec. tone for the amygdalectomized, hippocampectomized and unoperated monkeys. See also Table I.

(Bagshaw & Benzies 1968, Pribram *et al.* 1974). This puzzled us considerably and made data 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 *et al.* 1970) and his analysis clarify the issues. He expected an elevated tonic heart rate to accompany arousal (defined as a response to collative variables such as surprise, uncertainty, novelty and complexity of input much as we have defined them here) but as he was recording tonic rather than phasic changes he found the opposite: "these collative variables either had no effect on

Table I

Summary table of the GSR response measures taken on the 1st 21 trials of stimulus presentation (shown in 7 trial blocks) of (1) % responses (as compared with a baseline period of the same duration) when no stimuli (2 sec. tones) are presented; and (2) the amplitudes of these responses for normal, amygdalectomized and hippocampectomized monkeys. The numbers in parentheses indicate the size of each group studied. See also Figs. 2 and 3.

Group	% Response			Amplitude Kohms		
Trials	1–7	8–14	15–21	1–7	8–14	15–21
Normal	63	54	43	3.0	2.9	3.2
Hippocampectomy (7)	71*	70*	62	5.9*	5.8*	3.5
Amygdalectomy (6)						
Hyperresponsive (2)	90*	50	40	11.4*	4.3*	1.9
Hyporesponsive (4)	14*	00*	00*	2.0	--	--

* = p less than .05.

tonic heart rate or they had an effect (decelerator) opposite to expectations; but response factors and incentive factors (reinforcing consequences) had strong accelerating effects." Thus, arousal, though it may show a brief initial phasic acceleration (Obrist *et al.* 1965), is in many circumstances accompanied primarily by tonic heart rate *deceleration*, which is indicative of *activation*. In the next section we will detail the neural mechanisms involved in activation which, as Lacey (Lacey & Lacey 1975) has repeatedly shown, produces a readiness to respond to environmentally produced input or to that produced by the consequences of actions. In short, our monkeys with absent arousal reactions are consistent in showing an elevated tonic heart rate. As we shall see in the final sections of this review, such elevated tonic heart rate is manifest when the situation demands effort on the part of the organism.

We therefore interpret the effects of amygdalectomy as follows: because the specific controls on arousal are removed, arousal results not in the registration of the situation by altering the neuronal model, but in immediate nonspecific defensive *effort* to cope with the situation. This 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 tonic variables indicative of a lack of readiness to respond specifically to the

input. Thus "effort" is manifest in the absence of readiness. This interpretation is borne out by the results of an experiment in which we raised infant kittens in isolation, and showed that when examined at the age of six months their viscero-autonomic and endocrine reactivity in orienting experiments was essentially similar to that of amygdalectomized subjects: they had not learned to cope with situations and thus showed the "defensive" syndrome suggestive of considerable *effort* (Konrad & Bagshaw 1970).

In summary, studies relating brain function and the orienting reaction to sensory input have pointed to the presence of a system of neurons responding to the amount of input to them by maintaining or incrementing their activity. 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 viscero-autonomic responses. By way of its diffuse connections, this system is responsible for the more ubiquitous "arousal" responses recorded throughout the brain concomitant with orienting. Forebrain control over this core-brain arousal system is exerted by reciprocal facilitatory and inhibitory circuits centered on the amygdala. These circuits control the onset and duration of neural arousal much as they control the onset and duration of viscero-autonomic and appetitive responses.

Our interpretation of the relationship between the lack of viscero-autonomic responses to orienting and the failure to habituate behaviorally has been to suggest that a deficiency is produced in the more ubiquitous central mechanism by which organisms "register" input. When such failure in registration occurs, the organism's nervous system is temporarily swamped by the arousing input and reacts defensively to shut out all further input and thus leads to automatisms. This interpretation fits the clinical picture of the amnestic states ("deja" and "jamais vue") and the automatisms occurring during psychomotor seizures produced by epileptic lesions in the region of the amygdala. There is also considerable congruity between this interpretation and those of Mednick & Schulsinger (1968) and of Venables (Gruzelier & Venables 1972) in their report of two classes (GSR responders and non-responders) of patients diagnosed as schizophrenics. However, the interpretation also suffers from the difficulties that plague understanding of these clinical syndromes: how do disturbances of registration in immediate awareness influence subsequent retrieval? More of this in the following sections.

ACTIVATION

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 1960, 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 central representation or "neuronal model" must include such "demand" characteristics. Thus he proposes that Pavlov's "What is it?" reaction (which we have called "arousal", the registration of input in awareness) may not occur unless there is also 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: registering input, indicating "What is it?" and vigilant readiness signaling "What's to be done?" Strictly behavioral analyses have led to a similar dissociation. The early studies of Lawrence (1949, 1950) and the more recent work of Broadbent & Gregory (reviewed by Broadbent 1971) in terms of independent manipulations of stimulus set and of response set are perhaps the best known.

The CNV and TNVs

Probably the simplest situation which demands that responses become serially organized 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. A large body of data has been gathered in this situation.

These data concern the *contingent negative variation* (CNV) of brain electrical activity discovered by Grey Walter and his colleagues (Walter *et al.* 1964). Lacey & Lacey (1970) have established a correlation between the amplitude of CNV and a particular form of tonic change in heart rate

observed in intact subjects: *a deceleration was invariably observed* to follow the initial phasic acceleration which, as noted above, only sometimes accompanies arousal during orienting. Lacey & Lacey, along with Malmo & Belanger (1967), distinguish between their one-minute-long changes in heart rate and "enduring" changes in background level. We, as do Elliott (1969) and Elliott *et al.* (1970) shall call both of these "tonic" in the present review, but it may well turn out that subsequent experiments will show the Lacey types changes are related to activation while more enduring levels of heart rate reflect effort.

The CNV was originally proposed to reflect an expectancy developed when a specific response was contingent on awaiting the second of two stimuli. This would suggest that the CNV can be considered to be another central event indicating that an input is being matched against the organism's neuronal model. However, other workers suggested that the negative shift in potential reflects intended motor activity (e.g., Vaughan *et al.* 1968, Kornhuber & Deecke 1965). Still others, Weinberg (1972) and Donchin *et al.* (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, if no other than the suspension of random and irrelevant activity. Weinberg (1972) for instance, has shown in man that the CNV continues until feedback from the consequences of reinforcement of the response occurs, and we have obtained similar evidence in monkeys (Pribram *et al.* 1967). In a review of the CNV literature, Teece (1972) suggest that three types of negative potentials interact depending upon the demands of the experiment. These are: 1) CNV due to expectant attentional processes; 2) the motor readiness potential signaling intention to act; and 3) more or less "spontaneous" shifts whose occurrence cannot yet be attributed to specific task situations. This classification though consonant with results obtained by us in a series of studies (Donchin et al. 1971, 1973) does not indicate the full diversity of the CNV. We made recordings from several cortical locations under a variety of vigilance conditions. These studies showed that *transcortical negative variations* (TNVs) could be recorded from various sites in the brain and that the location in which the TNV occurs is dependent on the type of vigilance task. Thus, frontal TNVs are recorded only early in a task and when the task is changed; motor negative potentials are recorded only in anticipation of the necessity to make an overt response;

post-central negative potentials are largest when the organism must hold a response (continuously depress a lever) until a signal to release it occurs; and special sensory systems respond to their specific inputs. Many years ago, stimulated by the Gestalt formulations of Wolfgang Köhler, we had investigated with him, D.C. shifts in potential in the primary sensory projection areas resulting from stimulating the appropriate sensory channel. We found negative shifts to occur and these were invariably associated with the desynchronization of the EEG associated with activation (see Pribram 1971, p. 111). We conclude, therefore, that the TNV is an indication of either arousal or activation of the brain tissue from which it is recorded.

The Basal Ganglia and the Control of Activation

Sustained, tonic, change in potential is, however, not only of cortical origin. Subcortical negative potential shifts have been recorded in animals by Rebert (1972, 1973a, 1973b) and in man by Grey Walter (1967) and by Haider (1970). It is as yet too early to characterize the meaning of such shifts for every location, but in general, it appears that negativity develops whenever a portion of brain tissue is maintaining a readiness for processing. This conclusion is also reached by Hillyard (1973) in an analysis of the CNV and human behavior. Hillyard, however, notes, as do we (see Pribram & McGuinness 1975) that brain stem controls on overall cerebral negativity exist. We therefore will continue to use the term TNV for the multiple local readiness of cerebral tissues, and reserve CNV for the controls on these local potential shifts.

At the end of the negativity, if and when the organism actually begins to *do* something, a sharp positive deflection is usually observed and this positivity has been related to consummatory behavior (Clemente *et al.* 1964). In some brain locations (e.g., the visual cortex), the sharp positivity has also been associated with a sharp increase in power not only in the alpha frequencies, but also in the theta (4–8 Hertz) range (Grandstaff 1969). It remains to distinguish between the TNV that occurs as a function of arousal and the desynchronization, with its concomitant decrease of power in the alpha and theta frequency range, accompanying the negativity of activation.

EFFORT

Thus the brain systems involved in arousal and activation can be distinguished; arousal defined as a phasic reaction to input, activation, as a tonic readiness to respond. Yet, under many circumstances, the two reactions appear to be yoked: at the cortex by the TNV and also in the hypothalamic region (see Pribram 1971, Chaps. 9 and 10). In such situations they share the function of reflexly coupling input to output, stimulus to response. In the absence of controlled arousal and activation, behaving organisms would be constantly aroused by their movements and moved by arousing inputs. There must be some process that involves both arousal and activation which allows the uncoupling to take place. This process is habituation, a process critical in the development of the neuronal model. Action generated inputs (the outcomes of actions, their reinforcing consequences) appear to generate more complexly structured neuronal models than repetitions of simple inputs per se. This complexity is largely due to the participation of the central motor systems in generating input. Thus it takes longer to form a habit in, than to habituate to, the same situation. Complex inputs such as repeated exposure to the same musical performances do not readily induce habituation but in such situations a good deal of motor readiness – listening – is also involved.

The Hippocampal Circuit

Animals with bilateral hippocampectomy tend to show a % reactivity and amplitude of the GSR opposite to that observed in the non-responding amygdalectomized monkeys in the ordinary orienting paradigm. In addition, two somewhat more subtle changes have been shown to occur. The most important of these is that the phasic skin response terminates considerably more rapidly in hippocampectomized subjects than in controls. It appears from this that hippocampectomized monkeys (and amygdalectomized hyperresponders) re-equilibrate more rapidly than normal subjects whose slower GSR recovery may indicate a more prolonged processing time. As we shall see below, this is consistent with other data that show impaired processing of the disequilibration produced by a mismatch of input to the neuronal modal as a result of hippocampectomy.

The second change is that such animals show delayed or absent orienting

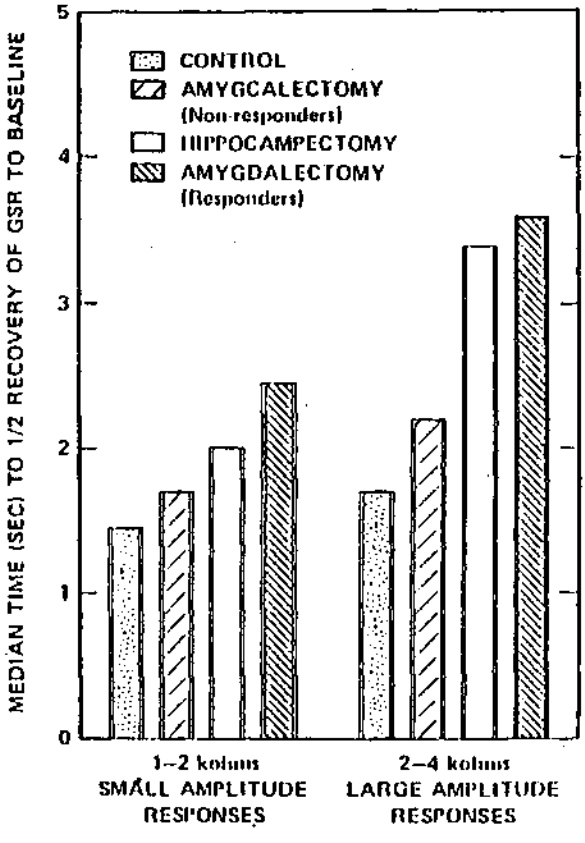

Fig. 3. Bar graph showing the *time* taken to attain half recovery to baseline of the visceroautonomic perturbation measured as an electrodermal response (GSR). Small (1–2 Kohms) and large (2–4 Kohms perturbations are treated separately since amplitude of response has an obvious effect on recovery time. See also Table II.

reactions when thoroughly occupied in performing some other task (Raphelson *et al.* 1965, Riddell *et al.* 1969, Wicklegren & Isaacson 1963, Crowne & Riddell 1969, Kimble *et al.* 1965). In short, these animals appear to be abnormally undistractible.

But in some situations this appearance of undistractibility is restricted to the overt responses of the organism and not to distraction *per se*. Douglas & Pribram (1969) used distractors in a task in which responses were required to each of two successive signals. Hippocampectomized monkeys initially responded much as did their controls by overtly manipulating the distractors

Table II

The *rate* of recovery of visceroautonomic perturbation (as measured by the GSR) to half the value above baseline for the first 21 trials in normal, amygdalectomized and hippocampectomized monkeys. Numbers in parentheses indicate size of groups. See also Fig. 3 where *time* taken rather than *rate* of response recovery is portrayed.

Group	Half recovery rate for controlled amplitudes Kohms/Sec.	
Amplitude	Small *1–2k*	Large *2–4k*
Normal (14)	.320	.460
Amygdalectomy (6)		
Hyperresponsive (2)	.730*	.730*
Hyporesponsive (4)	.250	.460
Hippocampectomy (7)	.580*	.620*

* = p less than .05.

and thus increasing the time between the two required responses. However, in contrast to the behavior of their controls who simultaneously habituated overt manipulation of the distractors and the interresponse time, the hippocampectomized group showed decrementing only of the overt manipulations – their interresponse time failed to habituate at all. Thus, in this situation, hippocampectomized monkeys continue to be perceptually distractible while becoming behaviorally habituated and undistractible. This result is identical with 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).

This dissociation between habituation of perceptual responses and habituation involving somatomotor performance appears to be part of a more general effect of hippocampal lesions. Dissociation between observing and instrumental response becomes manifest in other situations in which hippocampectomized monkeys are tested. In a discrimination reversal situation, extinction of previously learned behavior and acquisition of newly correct responses was practically indistinguishable from that of control subjects. However, in contrast to their controls, the monkeys with the hippocampal

lesions remained at a chance level of performance for an inordinately long time (Pribram *et al.* 1969). This effect of the lesion was shown to be due to the "capture" of the behavior by a position bias to respond to the 50 % schedule of reinforcement – a bias present in both control and lesioned subjects but hardly manifest in unoperated monkeys whose behavior came under the control of discriminative stimuli much more readily (Spevack & Pribram 1973). This result suggested that hierarchy of response sets was operative in the situation such that "observing" responses (indicative of "attention") were relinquished when the probabilities of reinforcement of discriminative stimuli 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 highly programmed relationships between perception and action, between observing and instrumental responses, and between stimulus and response are relinquished for more primitive relationships in which either input or output capture an aspect of the behavior of the organism without the ordinary highly structured coordinating intervention of central control operations. The mechanism by which the hippocampal circuit accomplishes this structured relationship has to some extent been elucidated by recordings of electrical activity from the hippocampus both with micro and with macroelectrodes, and by precise electrical stimulations of selected parts of the hippocampal circuit.

The Hippocampus, Arousal and Activation

Let us therefore look at some of the evidence that delineates the hippocampal mechanism and helps account for the general observation that the *greatest* change in gross electrical activity observed during habituation are recorded from the corebrain (especially mesencephalic reticular and limbic control systems, John 1967, John & Killam 1960).

Lindsley has recently elaborated the mechanism by which the hippocampally controlled reticular formation can effect these changes in registration. Lindsley and Macadar (1975) in keeping with many other recent publications (e.g., Fibiger *et al.* 1973, Ungerstedt 1974) have dissociated two systems of neurons that influence the hippocampal circuit. One system originates in the median raphé and associated structures of the mesencephalic reticular formation, the other originates more laterally in the locus ceruleus and

periaqueductal grey. We have come to know these two systems (see Pribram 1971, and Pribram & McGuinness 1975) as a serotonergic (indole amine) "stop" mechanism associated with arousal and a dopaminergic and norepinephrinergic catechol amine "go" mechanism associated with readiness and activation. Lindsley's findings were obtained by electrical stimulations of the appropriate structures in the mesencephalic reticular formation. Such stimulations of the "stop" mechanisms produced hippocampal desynchronization and at the same time a synchronization of the amygdala circuits. This suggests a reciprocal process by which the controls on arousal are maintained as long as hippocampal inhibition of the reticular formation is in progress – much as Vinogradova (1970) suggests. Only when mismatch from the neuronal model is signalled to the reticular formation does this inhibitory control become loosened producing hippocampal desynchronization – and concomitant relaxation, synchronization, of the arousal functions of the amygdala circuits. Lindsley has found that often, though not always, such hippocampal desynchronization is accompanied by desynchronization of the sensory motor porjection systems, suggesting that "registration", an alteration of the neuronal model, of the cortical representation, is occurring.

The second mechanism discerned by the Lindsley studies is the catechol amine activation system which was the focus of the previous section. This mechanism makes possible the "What is to be done" reaction, the processing of response produced inputs. When electrically stimulated, the mesencephalic portions of the "go" mechanism initiates hippocampal rhythmic activity in the theta range of frequencies. Early studies (Green & Arduini 1954) had uncovered the paradox that the desynchronization of the EEG recorded from the brain's convexity during "activation" was accompanied by synchronization in the recordings obtained from the hippocampus. (Though such synchronization is not as obvious in records obtained in monkey and man, computer analysis has shown it to occur and that it can be studied as well in the primate as in nonprimate mammals: Crowne *et al.* 1972). This synchronous rhythm is in the theta range (4–8 Hz) and has become the focus of a long series of studies, the results of which are pertinent to our analysis.

That theta frequencies are especially prominent in records made from the hippocampus was noted by Jung & Kornmuller (1938). Later a series of studies (Green & Arduini 1954, Grastyan 1959, Grastyan *et al.* 1959) described the occurrence of hippocampal theta to the orienting stage in a

conditioning situation. Still later, however, the occurrence of hippocampal theta was related instead to intended movement (Dalton & Black 1968, Black *et al.* 1970, Black & Young 1972): intended rather than overt movement because the theta rhythm occurs in completely curarized preparations who have been trained in the uncurarized state to lever press. These conclusions have been supported by another series of experiments conducted by Vanderwolf and his associates (Bland & Vanderwolf 1972a, b, Vanderwolf 1969, 1971, Whishaw *et al.* 1972). Rats were observed while moving freely in the test situation and hippocampal electrical activity was recorded continuously. Theta activity occurred almost exclusively when the rats were making "voluntary" movements, here defined as acts or "response sequences characterized by flexibility, modifiability and associability with a variety of physiological drives".

Adey and his group (Adey 1970, Adey *et al.* 1960, Elazar & Adey 1967, Radilovacki & Adey 1965) took the investigation of behavioral effects on hippocampal theta rhythms a step further by showing that a shift (from 4 to 6 Hertz) in power within the theta frequency range occurs in cats from the prestimulus period, through stimulus presentation, to the correct response. Bremner (1970) has further analyzed the changes in theta activity along three

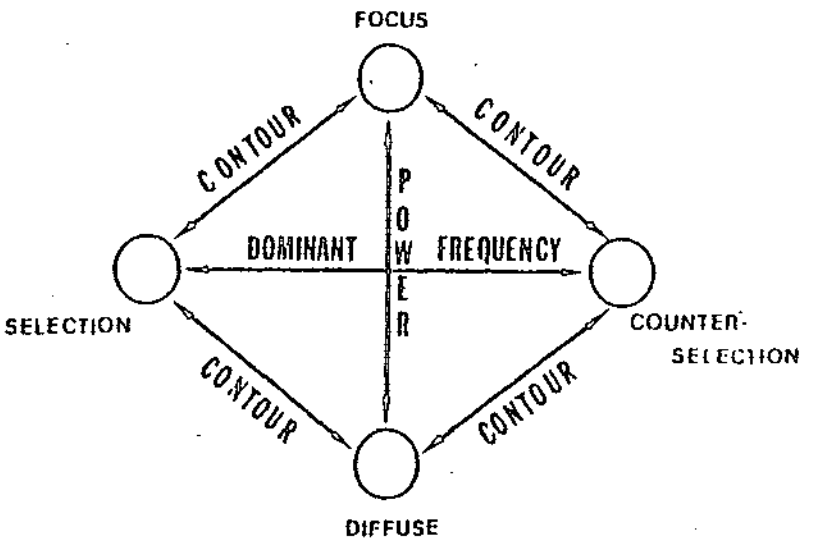

Fig. 4. Diagram of the relationship among dimensions of hippocampal theta activity and behavior.

dimensions: (1) an increase or decrease in the total amount of power in the theta range; (2) a narrowing or broadening of the range of energy distribution around the peak frequency; (3) the location of that peak frequency in the EEG spectrum. Change in total power of theta depends on the viscero-autonomic arousal (increase) *vs* somatomotor readiness (decrease) distinction delineated here, and the changes in contour and their distribution correspond to the categorizing-reasoning distinction pursued in the paper by Pribram & McGuinness (1975).

Summary

By way of review, we note that there exists evidence for the organization of a central representation of input, the construction of a (cortical) neuronal model. Changes in this neuronal model are controlled by two subcortical systems; one is located in core portions of the neuraxis and contains neurons that increment to or monitor input, while another more laterally placed contains neurons which rapidly decrement when they are repeatedly stimulated. In addition, we have delineated three mechanisms in the rostral portions of these control systems. One centers on the amygdala. This circuit

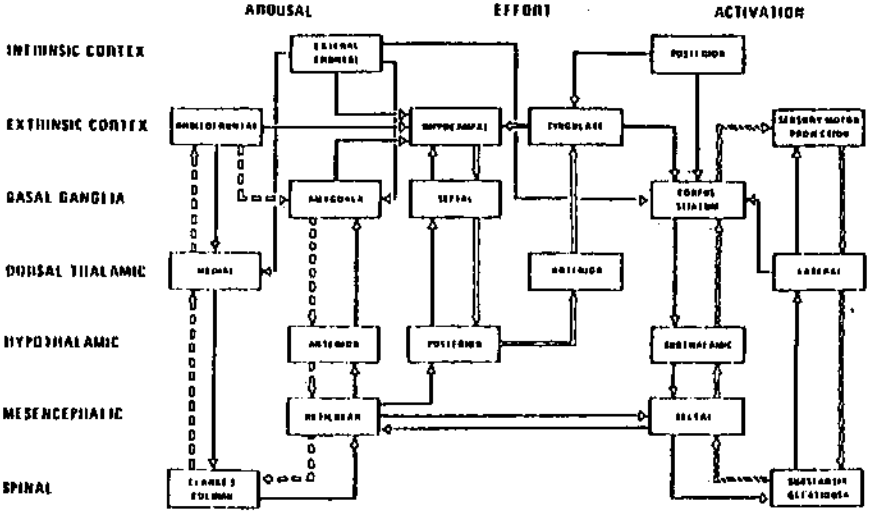

Fig. 5. A highly oversimplified diagram of the connections involved in the arousal (amygdala), activation (basal ganglia) and effort (hippocampal) circuits.

regulates the monitoring or "*arousal*" neurons and becomes organized into a "stop" or reequilibrating mechanism. A second is centered on the basal ganglia. This circuit involves the *activation* of "go" mechanisms – expectancies (perceptual) are readiness (motor). Finally, a third mechanism comprising the hippocampal circuit has been identified. This circuit is involved in uncoupling stimulus from response by coordinating the amygdala and basal ganglia mechanisms so that appropriate changes in the central representation can occur, a process that entails *effort*.

In the older comparative anatomical literature, the amygdala is usually considered to be one of the basal ganglia, albeit a special one related to the olfactory and visceral systems. Thus brain structures similar in their morphology, though different in their connections, control arousal and activation. Their coordination in effecting change in central representation, on the other hand, devolves on a circuit very different in morphology, more akin to that characterizing the cerebellum, another mechanism notable for coordinating complex sequential processes.

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