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PSYCHOPHYSIOLOGY AND THE STUDY OF PROTOCRITIC PROCESSES

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INTRODUCTION

The brain is a complex organ which defies simple description because of the overlap and interdigitation of its various systems. Nonetheless, considerable progress was made during the 1970's in reaching preliminary understandings of some parts of this complexity. I want here to address one area of interest in which psychophysiology, when properly joined with neuropsychology, neurophysiology and neurochemistry, has had a major impact.

The problem concerns the conceptualization of the functions of the core formations — especially the limbic systems — of the forebrain. A variety of conceptualizations has been forwarded to acount for the special relationship that exists among limbic structures and the effects of limbic lesions and excitations on behaviour. These conceptualizations run from limbic involvement in emotion and motivation (Papez, 1937) through limbic control over visceroautonomic activity (MacLean, 1952) to the loosening of innately determined constraints on behaviour by virtue of limbic activity (Isaacson, 1974).

The problem, of course, is a part of the classical one of localization of function in the brain. As I have repeatedly pointed out (Pribram, 1954, 1960, 1971, 1977a) the problem is not unique to the brain-behaviour relationship. All physiology shares this problem: it is easy to specify the functions of the lungs; it is difficult to 'localize' the mechanisms of respiration in any one organ.

Taking heed of this way of approaching the 'localization' problem, it becomes evident that we can perhaps specify the functions of identifiable tissues in brain but that such identification ought not to attempt to localize psychological functions. The identification of functions of part of the brain is better, therefore, defined in 'neutral' terms which relate to neural or engineering concepts.

In this spirit I wish to propose a theory of limbic system functioning that avoids the localization of emotion and motivation or even the localization of visceroautonomic control. I have elsewhere reviewed the evidence that makes such conceptualizations erroneous (Pribram, 1960, 1967, 1969, 1971, 1979). As an alternative, my proposal, based initially on psychophysiological data, is that limbic system function can best be described in terms of its 'protocritic' properties (Pribram,

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1977a, 1977b).

The term 'protocritic' is derived from Henry Head (1920) who, on the basis of experiments in which he severed his own peripheral nerves and studied his sensations as regeneration occurred, discerned two modes of feeling: epicritic and protopathic. Epicritic sensations carried local signs, i.e. they allowed the stimulus to be referred to a point in space-time. By contrast, protopathic feelings, which occurred while the nerves were regenerating and before they had re-established their normal spectrum of fibre-sizes, globally reflected the intensive dimension of the stimulus.

In Head's experiments these protopathic sensations were clearly linked to pathology: the nerves were cut. Subsequent research, however, showed that two classes of nerve fibres, distinguishable by their fibre size, were responsible for the two types of sensation: a large diameter system with a fast conduction rate mediates epicritic processes while a set of small fibres is responsible for protopathic sensations (Gasser and Erlanger, 1922). In view of the fact that such sensations are part of the normal functioning of the organism, the term protocritic rather than protopathic is more appropriate.

Much of this research dealt with pain and discomfort. Recently, the relationship between epicritic and protocritic processes has been viewed as rather more complex than just two parallel systems. Melzak and Wall (1965) have proposed that epicritic processes act as a 'gate' on the protocritic, i.e. when there is sufficient organization of input in space-time, protocritic sensations are eliminated.

Once the gate theory had been forwarded a further complication became apparent. In tracing the central spino-thalamic tract, the severing of which abolished pain (Sjoquist, 1938), towards the head, only about one-third of the fibres reached the ventrobasal thalamas and parietal cortex. The other two-thirds of the fibres disappeared along the way, many of them ending in core brain stem structures such as the periaqueductal grey and medial thalamus (Kunc, 1966).

These anatomical data were supplemented by the results of psychosurgical procedures undertaken to reduce pain and distress. Parietal cortex resections helped little while frontal leucotomies did (White and Sweet, 1969) and frontal cortex received its input from the medial thalamus (Pribram, Chow and Semmes, 1953; Pribram and Fulton, 1954). What, then, might be the relationship at the thalamic level between epicritic and protocritic processes? Could a ventrobasal-parietal or a closely related posterior thalamic-posterior parietal system, as suggested by Mountcastle, Poggio and Werner (1963), act as a gate on the more medially placed corebrain-frontal system? And perhaps vice-versa?.

PAIN AND TEMPERATURE

The above questions could not be answered while we knew little about the corebrain-frontal mechanism involved in the epicritic process. However, to study pain in animals in the experimental laboratory is difficult. Threshold studies can be done readily, but the organism's behavioural response to aversive stimulation is primarily one of escape and avoidance, and these measures involve other factors such as level of activity, memory, conflict, etc., which pose problems of analysis for the experimenter.

In order to circumvent some of these problems I searched for another sensory modality closely related to pain that would not bring with it these disadvantages.

In the spinal cord, the pathways for pain and temperature appear to be inseparable. The temperature sense thus suggested itself as an obvious candidate for exploration.

Further, just as in the case of pain, parietal lobe excisions had failed to influence temperature discrimination in a host of studies (e.g. Blum, Chow and Pribram, 1950; Downer and Zubeck, 1954). Perhaps the cortical involvement in temperature, as in pain, is frontal rather than parietal.

An experiment was performed in which temperature discrimination was disrupted by electrical stimulation of the posterior orbital surface of the frontal lobe, the amygdala and the stria terminalis (Chin, Pribram, Drake and Greene, 1976). Parietal lobe stimulations had no such effect. These results suggest that a neural system based on the pain and temperature modalities may remain separate not only in the spinal cord but through the brain stem and into the forebrain. The orbital locus of the rostral terminus of the system is not far removed from the site of the temperature regulating mechanism in the anterior hypothalamus; it should not be altogether surprising to find the regulatory and discriminative functions adjacent to one another.

In the brain stem and diencephalon the sites from which pain (aversive response) can be obtained are adjacent and often intermingled with those from which positive reinforcement due to electrical self-stimulation is elicited. Further, as is now well known, electrical stimulation of many of these sites with low frequency (10-20 Hz) currents produced analgesia (Liebeskind, Guilband, Besson and Oliveras, 1973; Liebeskind, Mayer and Akil, 1974) and when such stimulations are performed in man sensations of cooling accompany the analgesia (Richardson and Akil, 1974).

These data suggest the hypothesis of a neural system, or set of systems, including temperature as well as the pain sense and dealing with the hedonic dimension (distress-comfort). Thus, the term protocritic (rather than protopathic since discrimination, not pathology, is critical) distinguishes a hedonic dimension devoid of local signs which is separate from the epicritic which deals with organism-environment relationships in space-time (Pribram, 1976).

THE AMYGDALA

The central locus of the effect of electrical stimulation on temperature discrimination is the amygdala. (The other two effective sites, the orbital cortex and stria terminalis are respectively the source of a heavy input to the amygdala and serve its output.) The amygdala, classically classified as a basal ganglion and more recently as a part of the limbic forebrain, has over the past 30 years received considerable attention from the neuroscientific community (Eleftheriou, 1972). In addition to influencing temperature regulation (Satinoff, 1975) and discrimination (Chin, Pribram, Drake and Greene, 1976), the amygdala has been implicated in a complex of behaviours initially brought together under a rubric 'the four F's — Feeding, Fighting, Fleeing and Sex' (Pribram and Bagshaw, 1953; Pribram, 1954; Pribram and Weiskrantz, 1957; Pribram, 1960). The involvement of amygdala function was then further extended to encompass a variety of problem-solving behaviours related to reinforcement (Schwartzbaum, 1960), stimulus equivalence (Schwartzbaum and Pribram, 1960; Bagshaw and Pribram, 1965; Hearst and Pribram, 1964), delayed alternation (Pribram, Lim, Poppen and Bagshaw, 1966), the orienting reaction (Bagshaw, Kimble and Pribram, 1965; Bagshaw and Benzies, 1968) and classical conditioning (Bagshaw and Coppock, 1968).

These apparently disparate behaviours appear to be part of a common mechanism (Pribram, 1969, 1971; Goddard, 1972; Pribram and McGuinness, 1975). It is worth summarizing the highlight of this analysis because identifying a common mechanism operating on apparently disparate behaviours is a recurring problem in psychophysiology as it is in behavioural genetics (where it involves identifying genotype from phenotypical behaviours).

With regard to feeding, the amygdala has been shown to be a modulator of the satiety mechanisms centred in the ventromedial region of the hypothalamus. First, it was noted that the increasing feeding of amygdalectomized subjects was due to their failure to *stop* eating as readily as their controls (Fuller, Rosvold and Pribram, 1957). Then, a very precise relationship was established between the amounts of carbachol injected and amount of feeding (or drinking) once they had been initiated (Grossman, 1967; Russel, Singer, Flanagan, Stone and Russell, 1968).

This modulation of a *stop* mechanism was also shown to be responsible for changes in fighting behaviour. Fall in a dominance hierarchy after amygdalectomy was, when it occurred, related to the amount of aggressive interaction between the dominant and submissive animals of the groups. After amygdalectomy such interactions were overly prolonged leading to a reorganization of the dominance hierarchy (Rosvold, Mirsky and Pribram, 1954). It was as if the amygdalectomized monkeys approached each interaction as novel. Prior experience which modulated the behaviour of the control subjects seemed to have little influence after amygdalectomy. We shall have occasion to return to this finding repeatedly.

Analyses of the effects of amygdalectomy and electrical stimulations of the amygdala on avoidance (fleeing) behaviour have come to a similar conclusion. Escape behaviour is unaffected (Pribram, 1954; Pribram and Weiskrantz, 1957) and sensitivity to shock is not diminished (Bagshaw and Pribram, 1968). Nor is there a change in the generalization gradient to aversive stimulation (Hearst and Pribram, 1964). What appears to be affected primarily is the mnemonic aspect of avoidance: the expectation that aversive stimulation will occur unless the behaviour is *stopped*. Such expectations are ordinarily referred to as 'fear' but it must be clearly kept in mind that what distinguishes fear from pain (i.e. avoidance from escape) is an expectancy that stops the behaviour from occurring.

The theme recurs when the effects of amygdalectomy on sexual behaviour are analyzed. Hypersexuality is found to be not so much a quantitative increase in sexual behaviour but an increased territory and range of situations over which the behaviour is manifest (Greene, Clemente and deGroot, 1957; Pribram, 1960). Ordinarily cats *stop* such behaviour in unfamiliar territory.

The gap between the involvement of amygdala function on the Four F's and on the problem solving behaviour is therefore not as great as it initially seemed. Another fact is so-called passive avoidance, which sets up a conflict between approach and avoidance behaviour. After amygdalectomy animals fail to *stop* their approach on the basis of an aversive experience. Such conflict is, however, not limited to situations that involve aversive reinforcement. Approach-approach conflicts such as occur in delayed alternation partake of the same sorts of processes. Therefore we tested amygdalectomized subjects on various forms of alternation tasks and found the monkeys with lesions to be impaired (Pribram, Lim, Poppen and Bagshaw, 1966). Once again, the function of the amygdala is not limited to the aversive domain but rather extends wherever immediately current behaviour involves *stopping* prior ongoing behaviour.

The finding that the amygdala is involved in *stopping* ongoing behaviour led to a series of studies on its role in the orienting reaction. This series of studies clearly showed that the visceroautonomic components of orienting were markedly affected by amygdalectomy and that the habituation of orienting was dependent on the occurrence of these visceroautonomic responses. Behavioural habituation, the indicator of familiarity, occurs in part, therefore, as a result of visceroautonomic activity. What is oriented to, the novel, is a function of the familiar, the expected, on the basis of prior experience. However, the prior episode must have included a visceroautonomic reaction to be effectively experienced.

It is, of course, clear from a host of other studies relating brain and behaviour,

that all memory processes do not critically depend on the occurrence of viscero-autonomic responses. The learning of motor skills, perceptual differentiation, rote memorization, etc., are examples where the memory mechanism operates more on the basis of simple repetition (Vinogradova, 1975; Pribram, 1977a). Still, it is equally clear that there are occasions when memory is dependent on a 'booster' that *stops* ongoing behaviour and derives from the importance (novel, intense, distressing or hedonic) of the situation to the organism. It is this booster type of memory process in which the amygdala is involved.

AROUSAL, ACTIVATION, THE HYPOTHALAMUS AND BASAL GANGLIA

A precise operational definition of this involvement can be given (Pribram and McGuinness, 1975). This definition is based on the studies of visceroautonomic indicators. Such studies show that amygdalectomy influences the phasic components of the indicators rather than their tonic components. The term 'arousal' is commonly used to describe the organism's phasic, i.e. brief, response to input as in the orienting reaction, in alerting when expectations are disconfirmed, etc.

The advantage of defining arousal precisely comes when it is distinguished from other similar processes with which it is ordinarily confounded. Confusion occurs most often when the phasic and tonic reactions of organisms are lumped together. Elsewhere (Pribram and McGuinness, 1975) we have reviewed in detail the evidence that tonic visceroautonomic reactions are regulated by the brain mechanisms that control the organism's readiness to respond, mechanisms which centre on the basal ganglia (caudate nucleus and putamen) of the forebrain. We can therefore clearly separate, both on the basis of peripheral indicators and the brain mechanisms involved, the process of phasic arousal from that of tonic activation. Arousal is a function of a set of neural systems whose forebrain extension is the amygdala; activation is a function of a set of neural systems whose forebrain extensions are the basal ganglia.

The basal ganglia of the forebrain have, until recently, been thought of primarily as regulators of muscle tone. There is now a body of evidence which shows that the basal ganglia also control sensory input (Pribram, 1971, 1977). This finding is not altogether disparate to the motor control functions of the basal ganglia since these are to a large extent affected by changes in the bias of muscle spindles, receptors that reflexly regulate muscle contraction by way of feedback.

We are now in a position to take up another experimental result which has posed explanatory difficulties for decades. When lesions are made in the region of the ventromedial nucleus of the hypothalamus, rats over-eat and become obese. As noted earlier, this finding led to a series of experimental results that indicated that the ventromedial hypothalamus is a critical part of a 'satiety' mechanism. Before these results were available, however, it was also shown that these same rats would eat less than their controls and might even starve if an easily surmountable barrier were placed between them and the food. The initiation of behaviour and its maintenance (stop mechanism) were dissociated. Other experiments showed that the initiation of feeding was controlled by a mechanism centred on the far-lateral region of the hypothalamus, a region devoid of neurons but rich in fibre tracts (Teitlebaum, 1955; Pribram, 1971). Recently, the far-lateral hypothalamic syndrome has been replicated by administering drugs that inhibit the formation of dopamine, the putative transmitter that characterizes the nigro-striatal basal ganglia system. More of this shortly.

Further, it was found that excitation of the ventromedial region of the hypothalamus not only stopped eating behaviour but led to the stopping of other behaviour. Alerting, escape and attack could be elicited depending on the strength of stimulation. These findings led Grossman (1967) to suggest that the ventromedial hypo-

thalamus is involved in regulating 'affect', not 'appetite'. Affect in this instance is defined on the basis of phasic reactions to input and thus fits the definition of arousal already presented (Pribram, 1971).

In summary, the experimental evidence falls into place when it is grouped on the one hand according to a phasic, stop, satiety mechanism which regulates arousal and, on the other, a tonic, start, appetitive readiness mechanism that regulates sensory and motor activation. Arousal is controlled by a neural system that includes the ventromedial hypothalamus and amygdala. Activation is controlled by the basal ganglia, in particular the nigrostriatal system whose pathways course through the far-lateral hypothalamic region.

EFFORT AND THE HIPPOCAMPUS

In addition to phasic arousal and tonic activation, a third process has been distinguished by psychophysiological analysis. This third process is also tonic but differs from the activation of readiness in that the visceroautonomic indicators are influenced in an opposite direction. Thus, during readiness heart-rate decelerates while acceleration accompanies this third process which we have called 'effort'. Other terms that are used are chronic arousal, anxiety, and reaction to stress. A detailed review of the relevant neurobehavioural and psychophysiological evidence (Pribram and McGuinness, 1975) shows that the hippocampus is central to the neural systems involved in regulating 'effort'. In this review, effort was shown to be necessary to coordinate phasic arousal and tonic readiness in situations that invoke both processes, such as discrimination reversal (Pribram, Douglas and Pribram, 1969), alternation (Pribram, Wilson and Connors, 1962), problem-solving under distraction (Douglas and Pribram, 1969) and when reasoning depends on computable variations in the situation (Douglas and Pribram, 1966; Douglas, Barret, Pribram and Cerney, 1969; Spevak and Pribram, 1973). We have begun to understand how the hippocampus performs this coordinating function (Pribram and Isaacson, 1976; Isaacson and Pribram, 1976).

SOME NEUROCHEMICAL PRELIMINARIES

This has been a brief overview of the methods and results of some thirty years of neurobehavioural and psychophysiological research. Currently data have accumulated relating a variety of brain amines and peptides (many of the peptides being derivatives of ACTH) to a variety of behaviours. Interestingly, the behaviours that have become involved in neurochemical research are largely the same as those involved initially in amygdala research and then shown to be dependent on hypothalamic, basal ganglia and hippocampal function as well. Thus the neural organization of the mechanisms of arousal, activation and effort delineated by neurobehavioural and psychophysiological techniques may well be relevant to the analysis of the relationship between neurochemical and behavioural processes.

Perhaps the easiest place to start is the well established and dramatic finding of a dopaminergic nigro-striatal system (Ungerstedt, 1974; Fibiger, Phillips and Clouston, 1973) which has already been discussed. The evidence has repeatedly been reviewed to the effect that dopamine is involved in the maintenance of postural readiness and motivational activation (Snyder, 1974; Matthysse, 1974). It is also known (e.g. King and Hoebel, 1968) that assertive behaviour such as predatory aggression depends on the activation of a cholinergic mechanism. Thus it is likely that the dopamine fibres interdigitate a cholinergic matrix (Fuxe, 1977) to determine the activation level of the nervous system and the readiness of the organism.

Two other well known neurochemical systems are those involving serotonin and nor-

epinephrine. A large amount of research (Jouvet, 1974; Barchas, Ciaranello, Stolk, Brodie and Hamburg, 1972) has related these substances to the phases of sleep: serotonin to ordinary (slow wave) sleep and norepinephrine to paradoxical (rapid-eye-movement) sleep during which much dreaming occurs. The relationship between serotonergic and norepinephrinergic mechanisms and the amygdala seems to be similar to that between acetylcholine and dopamine and the striatum of the basal ganglia. Serotonergic and norepinephrinergic systems of fibres densely innervate the amygdala, the norepinephrinergic interdigitating a serotonergic matrix (Pribram and Isaacson, 1976).

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

However, norepinephrine has been related to a behavioural function in which the amygdala is thoroughly implicated — the effects of reinforcing events (Stein, 1968). Norepinephrine has also been related to orienting and affective agonistic reactions. Once again a phasic 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 extent to be dependent on a serotonergic mechanism (Reis, 1974; Goldstein, 1974).

These data suggest that norephinephrine acts by modulating a serotonergic substrate (which is determining one or another basic condition of the organism) to produce paradoxical sleep, reinforcement, orienting to novelty and perhaps other behaviourally relevant neural events that interrupt an ongoing state. The data are not as clearly supportive of this suggestion as those that relate acetylcholine to an assertive state that becomes modulated by the activity of dopamine to produce specific readinesses. Nonetheless, as a first approximation to the data at hand, let us hold these possible neurochemical relationships in mind as a tentative model with which to analyze the mass of evidence on the behavioural neurochemistry of the polypeptides.

NEUROPEPTIDES AND THE EFFORT MECHANISM

The neurochemical evidence on ACTH related peptides leads directly to the hypothesis that they are involved in the hippocampal mechanism. To begin with, Bohus (1976) and McEwen *et al.* (1976) have shown that the hippocampal circuit (hippocampus and septum) is the brain site most involved in 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). Much to our surprise, the brain region which binds the most corticosterone is not the hypothalamus but the hippocampus.'
>
> (McEwen *et al.*, 1976)

Thus the receptors of adrenal cortical hormones can set the neural state which becomes modulated by ACTH related peptides. Evidence that such modulation of a corticosterone determined state involves the hippocampus is presented in van Wimersma Greidanus and de Wied (1976).

Second, as noted in the review by Pribram and McGuinness (1975), the hippocampal circuit functions to coordinate arousal (phasic response to input) and activation (tonic readiness to respond). Thus, in a complex behavioural situation, coordination would be influenced by manipulations of this circuit, and a host of apparently conflicting results might be obtained with very slight changes in the conditions of the experiment. The best known of such slight changes is the one-way *vs* two-way conditioned avoidance task (Pribram *et al.*, 1966; van Wimersma Greidanus and de Wied, 1976).

Further, effects on phasic and tonic processes (arousal and activation) as well as on their coordination (effort) would be expected. This expectation is borne out in the catalogue of effects of manipulations of ACTH related peptides: extinction of two-way but not one-way avoidance (de Wied, 1974), intereference with passive avoidance (Levine and Jones, 1970), interference with learned taste avoidance (the Garcia effect – Levine *et al.*, 1977), interference with discrimination reversal (Sandman *et al.*, 1977), facilitation of memory consolidation (van Wimersma Greidanus and de Wied, 1976), facilitation of exploratory behaviour and conditioning (Endroczi, 1972, 1977).

Just as in the case of manipulation of hippocampal activity, *ongoing* behavioural activity (memory consolidation, exploratory behaviour) is faciliated while any *change* in behaviour (two-way shuttle, passive avoidance, learned taste aversion, discrimination reversal) is interfered with. This appears initially as tilting the bias towards readiness. But as Pribram and Isaacson (1976) show for hippocampal function and Sandman's group conclude (1977), such an interpretation does not hold up. In the case of hippocampal research, the initial formulation stated that after hippocampal resections animals could not inhibit their responses (McCleary, 1961). This interpretation foundered when such animals were found to perform well in go/no-go alternation tasks (Pribram and Isaacson, 1976; Mahut, 1972) and that they could withhold behavioural responses despite an increase in reaction time when distractions were presented (Douglas and Pribram, 1969).

The most cogent analysis has been performed on discrimination reversals. Isaacson *et al.* (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 position response patterns. In short, the monkeys' behaviour 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 tate of reward. Champney, Sahley and Sandman (1976) have shown ACTH related peptides to operate on just this aspect of the reversal experience and have shown interactions with sex differences.

Evidence such as this makes highly plausible the hypothesis that ACTH related peptides operate on the hippocampal circuit and therefore the 'effort' process. But there is more: Strand *et al.* (1976) present direct evidence that muscle fatigue is reduced by ACTH related neuropeptides and that this effect must be central. Pribram and McGuinness (1975) in their analysis review the evidence for peripheral metabolic events that contribute to effort but could at the time show only indirect evidence for a central process devoid of peripheral concomitants (Pribram and McGuinness, 1975b). Strand *et al.*'s current contribution is thus a most welcome addition.

THE PROTOCRITIC DIMENSION

The foregoing analysis and review of evidence indicates that systems of core brain stem, basal ganglia, and limbic forebrain structures can be discerned in which neurochemical events underlie to a large extent the behavioural functions that are regulated by these structures. Three classes of systems were discerned. One class determines specific neuromuscular and neurosensory readinesses. A second deals with the momentary cessations of ongoing behaviour, cessations due to interrupting distractors, the intervention of satiety or the recurrence of reinforcing events. The third class of systems coordinates the readinesses of the organism with the processes that lead to their momentary suspension.

The proposal was made that states of specific readiness were due to a cholinergic mechanism operated upon, i.e. modulated by, dopaminergic systems. The basal ganglia are the major gross forebrain embodiments of the readiness mechanism.

The gross forebrain locus upon which the systems that deal with momentary cessation of behaviour converge is the amygdala. Neurochemically, these systems are positioned to be basically serotonergic with norepinephrinergic operators modulating the basic serotonergic state.

Finally, a coordinating mechanism was discerned whose forebrain extension lies within the hippocampal circuit. The neurochemical constitution of this class of systems is hormonal with neuropeptides operating on the hormonally induced neural state to regulate behaviour. Corticosteroids and ACTH related neuropeptides are examples of the functions of this third class of systems.

CONCLUSION

In closing, I would like to venture that the protocritic process — the brain organization centred on the pain-temperature dimension of experience — is central to these three classes of systems. As first proposed by Brobeck (1948) and reviewed in detail by Grossman (1967), temperature regulation anchors muscular tonicity, water metabolism and food intake. As reported by Feldberg and Myers (1963) and elaborated more recently by Myers (1969), *two* reciprocal hypothalamic neurochemical mechanisms can be discerned as controlling these functions. One is a serotonin-norepinephrine mechanism (serotonin elevates and norepinephrine lowers temperature) and the other is an acetylcholine-dopamine mechanism (acetylcholine elevates and dopamine lowers temperature). Acetylcholine also induces drinking and the catechols induce feeding. Thus, once again, the 'arousal' and 'activation' systems can be separately identified. However, according to the proposal made here norepinephrine should operate on the satiety mechanism in the ventromedial hypothalamus. So far, the evidence is not clear whether the increased food intake resulting from hypothalamically injected norepinephrine does in fact result from such action. Amphetamines, usually found to stimulate norepinephrine receptor sites in the brain stem (Bradley, 1968), decrease appetite but it is not known whether this is due to an enhancement of satiety.

At a higher level of control are the coordinating (effort) mechanisms that utilize hormones and neuropeptides to organize behaviour dependent on the smooth interaction of tonically activated sensory and motor readinesses and episodic (phasic) arousals to internal and external inputs.

The role of pain in these sets of hierarchies of controls is just beginning to be established. The discovery of a morphine-like neurosecretion (enkephalin) by Hughes (1975) and Hughes *et al.* (1975), makes it plausible to treat the regulation of pain (and itch) in homeostatic terms (Pribram, 1976). Further, the evidence presented by Gispen *et al.* (1977) that ACTH and some of the related neuropeptides

could serve as endogenous ligands on opiate receptors provides an initial suggestic that the pain-analgesia (effort-comfort) process may function at the coordinating (hippocampal) level of the hierarchy of controls.

On earlier occasions I have identified emotional processes as rooted in the phasic arousal mechanisms discussed here (Pribram, 1967, 1971, 1979; Pribram and Melges, 1969; Young, 1973) and distinguished them from motivational processes rooted in the readiness mechanisms. The classification of arousal, activation and effort mechanisms was developed in order to understand the effects of brain operations and recordings on attentional and intentional behaviour (Pribram and McGuinness, 1975). The relationship of attention and intention to learning and remembering has been reviewed as well (Pribram, 1976). Thus the analysis of the neurochemical mechanism undertaken here is relevant. The analysis would predict that neuropeptides would be only indirectly involved in the regulation of emotion (affect) and motivation. Only when emotional and motivational processes need be coordinated (as in stressful, effort-producing situations) would neuropeptide manipulations show an effect. Emotion and affect are found minimally influenced by ACTH related compounds in man (Ehrensing and Kastin, 1976). Conflict producing tasks such as passive avoidance (Levine and Jones, 1970) learned taste aversion (Levine *et al.*, 1977), two-way shuttles (de Wied, 1974; Gispen *et al.*, 1977), and frustrative non-reward (Gray and Garrud, 1977) are the instruments of choice for demonstrating the effects of neuropeptides. One-way shuttles and simple punishments show either no effect or a mild facilitation of the reinforcing process.

As in the case of emotion and motivation, the effects of neuropeptides on learning and memory consolidation appear to be secondary. This is brought out most clearly in the myriad of neurochemical effects of neuropeptide manipulation described in papers dealing with these topics which pinpoint their relationship to stress, i.e. to an effort-comfort dimension rather than to learning and memory *per se* (Gold and McGaugh, 1977; Meyer and Beattie, 1977).

These neurobehavioural and neurochemical contributions illustrate the power of an analysis that was originally initiated by psychophysiological data. Essential to such power are: (1) careful and rigorous definitions of terms, concepts and their relationship to each other, and (2) adducing data from neuropsychology, neurophysiology and neurochemistry to test and amplify the conceptualizations derived from psychophysiological data. As can be seen from the example provided, the field is ripe to such contributions. As psychology proceeds into its second century of experimental analysis, topics such as image and information processing as well as protocritic processing should provide a rich yield of systematization of available psychophysiological data and the concepts derived from them.

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