Reports from the Research Laboratories
of the
Department of Psychiatry
University of Minnesota

NEUROPSYCHOLOGY AND PSYCHOPHYSIOLOGY IN PERSONALITY RESEARCH

Part I. Physiological Psychology and Personality Theory

by

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(List of references to Part I may be found at the end of Part II, PR-65-4)

Report Number PR-65-3

June 1, 1965

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PART I. Physiological Psychology and Personality Theory

[To be published as a chapter in the Handbook of Personality Theory and Research, edited by E. F. Borgatta and W. W. Lambert, Rand McNally & Co.]

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Not many years ago it was still possible for theoretical psychologists to defend an ignorance of neurophysiology on the grounds that this molecular discipline had as yet failed to develop many facts, concepts or principles which could be usefully translated into the language of psychology. Most psychologists assumed, then as now, that it is the nervous system which mediates the relationship between stimulus and response, between input and output, and therefore that any adequate theory which claims to account for these relationships must, in some ultimate sense, be a theory about the nervous system. Although the language of such a theory might be exclusively psychological, making no direct reference to neurons, synapses or nuclei, any adequate theory of, say, the emotions must be structurally homeomorphic with the description that an omniscient neurophysiologist would give of the aspect of the nervous system which in fact mediates emotional phenomena, i.e., the psychological theory must in principle be reduceable to the corresponding physiological account (although such a reduction may be difficult and inexpedient). But the concepts of psychology were so molar, and the building-blocks of physiology so molecular, that there seemed to be little common ground on which a mutually rewarding interchange might be established.

However, developments within and communication between these two disciplines have accelerated greatly in the past ten or twenty years.

It is permissible to infer that the basic unit of current statistical learning theories or of the structural theories proposed by Blum (1961), Deutsch (1960), or Hebb (1949) may be identified with the single neuron or synapse. Physiologists, in their turn, are identifying complex systems which clearly seem to constitute the substrate for such psychological states or processes as sleep, dreaming, arousal, pleasure, reward, pain, fear, rage, memory, and the like. The present extensive use of physiological techniques on the part of psychologists and the equal popularity of psychological or behavioral techniques among many physiological researchers (including pharmacologists) is probably both symptomatic of and facilitating to this greatly increased interchange at the level of theory.

The study of sensation and perception provides just one of many possible examples of this kind of development. Psychologists of the "softer" variety have known since before the time of Freud that what a man perceives and even senses can be considerably influenced by the nature of his attitudes, expectations and beliefs ("Such tricks hath strong imagination that, if it would but apprehend some joy, it comprehends some bringer of the joy; or in the night, imagining some fear, how easy is a bush suppos'd a bear!"--Midsummer Night's Dream). Psychologists of the "harder" type have understood much the same thing at least since the work of Bruner and Goodman (1947). It is possible that these admittedly imprecise notions of the psychologists helped to motivate or guide the physiological research of Golambos, Hernández-Peón and many others on the centrifugal control of afferent processes (vide infra), research which has written a new and exciting chapter of molar neurophysiology in just the past ten or fifteen years. These developments, in turn, should be of

heuristic value to psychologists working with such phenomena as perceptual defense and the like and should give impetus as well as guidance to attempts to elaborate a psychological theory of perception.

But the relevance of modern physiology to psychology in general, and to personality research in particular, is technological as well as theoretical. An increasing proportion of studies in the personality field make use of experimental techniques--independent or dependent variables--derived from the physiological laboratory. Some examples: In the study of temperament, autonomic responses such as heart rate changes or the GSR are used as indicants of the intensity of emotional responses shown by different types of individuals or in reaction to different types of stimulus conditions. So-called psychotropic drugs, like adrenaline, amphetamine, or the various tranquilizers, may be used to manipulate states of arousal or emotional reactivity as an aid in the study of the defense mechanisms or other affective-cognitive or affective-sensory interactions. There are areas of personality research which already demand some familiarity with these technological developments and it is difficult to imagine an area in which some such competence would not prove to be an asset to the investigator.

A convention has recently been proposed (Stern, 1964) aimed at clarifying and stabilizing a distinction already implicit in our use of the designations physiological psychology and psychophysiology. It is suggested that we characterize as "physiological psychology" research in which the independent variables are physiological manipulations--surgical procedures, brain stimulation, drugs--while the dependent variables are psychological--test performance, Skinner box behavior, subjective report, behavioral observations or ratings, and so on. Obversely, "psychophysiological

research" will imply the use of psychological manipulations and the measurement of physiological effects, e.g., a study of fear conditioning using the GSR as an indicant, research on stage fright as monitored by heart rate changes, the use of pupillary dilation to measure the interest value of stimuli. By this definition, a few personality researches qualify as "physiological psychology," studies using drugs, principally, like Schachter and Singer's (1962) experiment (a mixed case, really, since psychological manipulations were also used) in which Ss pretreated with adrenaline became angry in the presence of an angry-acting stooge or, with equal readiness, gay and light-hearted in the presence of a frivolous stooge. But personality researchers seldom employ surgery or stimulate the brain (no pun intended) so that the relevance here of physiological psychology is mainly theoretical or heuristic, whereas, as suggested above, the use of physiological dependent variables is already widespread in the personality area. The present chapter is organized in accord with this distinction, the first part being devoted to a discussion of some recent research in physiology and physiological psychology which would seem to be of interest to the personality theorist while the second part is primarily technological, concerned with problems of measuring and interpreting certain psychophysiological dependent variables which have been and will continue to be profitably employed in personality research.

PART 1. PHYSIOLOGICAL PSYCHOLOGY AND PERSONALITY THEORY

Rather than attempt a comprehensive survey, which must needs would be both superficial and incompetent, I shall give only passing mention to several important areas which the interested reader can follow up elsewhere on his own, and then essay a more detailed examination of recent work on

reticular activiting system and the physiological psychology of arousal, perception and attention.

BIOCHEMICAL INDIVIDUALITY

A collection of stomachs or livers or hearts, obtained from a random sample of normal, healthy human beings may appear so extraordinarily diverse in size, shape and internal structure as to lead the uninitiated to conclude that they must have been obtained from several different species of animals. Some normal hearts can pump 11 liters of blood per minute and others only three; the size, capacity and branching pattern of the blood vessels to the brain show a similar variability. In a summary of the 19 enzymes for which data are available, the "normal" range of inter-individual variation in enzyme efficiency was found to be never less than two-fold, typically three or four fold and, in some instances, differences of 1000 to 5000 per cent were recorded. Healthy human thyroids range in weight from about eight to 50 gms. and stable measures of thyroid activity vary between individuals over at least a five-fold range. Normal variation in pituitary weight is from about 350 to 1100 mg. while individual levels of secretion of the eight or more hormone products of this gland appear to be at least equally variable.

In a fascinating monograph, Roger Williams (1963) has assembled a multitude of such illustrations of anatomical and biochemical variability, mostly constitutional and some actually genetic in origin. That these are all "normal" subjects, each coping reasonably successfully with rather similar environmental demands, emphasizes the extraordinary capacity of the organism to compensate for idiosyncratic patterns of biological endowment by suitable adjustments in its pattern of activity and of

nutrition. Williams' thesis, in part, is that individual differences in neurophysiological--micro-structural, biochemical--endowment, while less well documented thus far, may be expected to be of similar magnitude, that no single pattern of nutrition--defined broadly enough to include vitamins and drugs at one extreme and special patterns of training and experience at the other--can be expected to produce optimum development and function in all cases, and that special patterns of nutrition, rationally derived from an improved understanding of neurophysiological individuality, may enable different individuals to approach the same goals in different ways or, at least, different goals of equal value.

In spite of its polemical overtones, this book is the best compensation I can think of for those psychologists not fortunate enough to have been able to hear the late D. G. Paterson discourse on the nature, extent and importance of individual differences. All students of personality would benefit from reading it. Psychologists who tend to assume that, if a characteristic does not clearly run in families, it must be learned; clinicians who refuse to consider possible constitutional etiology of a condition on the grounds that this implies "therapeutic nihilism"--in the face of the manifest difficulties of modifying a lifetime of mislearning and the many successes of organic medicine in compensating for constitutional defects by surgical, pharmaceutical, or nutritional means--should take Williams' data and his message under serious advisement.

MOTIVATION AND REWARD

Just over ten years ago, W. R. Hess (1954) reported the elicitation of voracious eating behavior in cats by electrical stimulation in the hypothalamus and, in the same year, Olds and Milner (1954) showed that

brain shocks in similar locations also had the properties of behavioral reward, that rats would learn new responses and work almost untiringly at old ones if such stimulation was provided as a reinforcer. In that same productive year, Delgado, Roberts and Miller (1954) found that stimulation of certain regions in the thalamus and hippocampus could produce the behavioral effects of normal pain or fear, viz., punishment, emotional arousal, and the learning of escape and avoidance behavior. These exciting discoveries have precipitated a flood of research, much of which can be found ably summarized by Olds (1962).

It is now clear that stimulation in many brain regions will produce the reinforcing effect in varying degrees. In some areas the effect is subject to satiation, but it appears that rats, cats and monkeys will work for stimulation in the lateral hypothalamus without any sign of satiation until exhaustion supervenes. At least some of these areas seem to be related to specific primary drives, e.g., shocks in some regions are rewarding when the animal is hungry but less so or not at all when he is satiated for food. There is considerable overlap between the motivating and reinforcing effects; in many areas where the animal will work for stimulation that same stimulation will also lead him to consume food if it is available or to emit previously acquired food-finding responses. In at least some loci, rewarding brain stimulation also provides its own motivation. Animals will not begin again to self-stimulate if interrupted for a brief interval but must be "primed" by one or more brainshocks which seem to re-institute the "drive" for additional stimulation (Deutsch, 1963). In addition to other brain regions where stimulation is unequivocally nociceptive, producing emotional and escape or avoidance behavior, there

are loci at which both the onset and the offset of stimulation have been shown to be reinforcing, i.e., the animal will learn one response to turn the brain-shock on and a different response with which he then promptly turns it off. There is also evidence for reciprocal inhibition between these "plus" and "minus" centers with some dispiriting indications that the latter tend to dominate the former (although cf. Brady and Conrad, 1960).

EMOTION

The pioneering work of Hess (1954) on the technique of electrical stimulation in the depths of the brain substance led also to an acceleration of research on the related problems of the neurophysiology of emotional behavior. The anatomical substrate of emotion appears to reside in a kind of shell of tissue which surrounds much of the innermost core of the hemispheres, made up of such structures as the hippocampus, the amygdala, the cingulate gyrus, the hypothalamus, fornix, mammillary bodies and anterior thalamus, and which has come to be known as the limbic system or "visceral brain" (MacLean, 1955). The intimate relation between the emotions and the systems of drive and reward is illustrated anatomically by the fact that the medial forebrain bundle, the region in which stimulation seems to produce the most potent reinforcing effects, is also the major line of communication between the limbic lobe on the one hand and the hypothalamus, midbrain and other brainstem structures on the other.

The lesion studies of Kluver and Bucy in the late 1930s demonstrated that bilateral ablation of the amygdala converted wild monkeys into tame and docile, unemotional creatures, showing compulsive orality and a bizarre hyper-sexuality--the "Kluver-Bucy syndrome." MacLean (1963) has

emphasized the anatomical proximity in the amygdala of centers concerned with oral or eating behavior--salivation, chewing movements--and those concerned with sexual responses including penile erection. Noting that stimulation in the nearby region of the anterior commissure may produce angry or fearful vocalization and the showing of fangs, MacLean suggests that these anatomical linkages may help explain the behavioral interrelation of sexuality, orality, fear and aggression.

Rage-like or aggressive behavior can be elicited by stimulation in several brain areas. Roberts and Kiess (1964) showed that stimulation in the anterior hypothalamus produces in cats not only hissing and other affective displays, but also leads the animal to attack a rat that it had hitherto ignored, even turing away from the food dish to do so, and, most importantly, under the influence of such stimulation these animals would learn to run a Y-maze to obtain a rat that they could attack; without stimulation, performance deteriorated and there were no attacks. "It was concluded that the performance of the attack was rewarding, and the central readiness for attack elicited by the stimulation possessed motivational and cue properties salient in the evocation of the learned responses leading to prey."

Stimulation of the Human Brain

The sparse literature so far available on the effects of stimulation of these regions in unanesthetized human subjects (e.g., Delgado and Hamlin, 1960; Heath and Mickle, 1960; Sem-Jacobsen and Torkildsen, 1960; King, 1961) indicates that electrical excitation of roughly the same areas which are concerned with positive reinforcement in animals produces in humans feelings of well-being, euphoria or sometimes even a kind of erotic

ecstacy; the temptation to refer to the Olds "plus" centers as "pleasure" or "joy" regions thus finds some additional justification. In contrast, Penfield (1958) was able to elicit feelings of fear, sorrow, loneliness or disgust by stimulating the anterior and inferior temporal cortex. Penfield and Roberts' (1959) reports of the hallucinatory effect of temporal lobe stimulation are particularly arresting. As described by the patients afterwards, these experiences are vivid evocations apparently of the veridical past which begin and end with the switching of the current and can sometimes be re-run or restarted where they ended by stimulating again in the same area. This truly seems to be "the stuff that dreams were made of" and, taken together with a considerable quantity of less dramatic but equally essential work on the role of the hippocampus in learning and recall, focuses attention on the hippocampal zone as the probable storage site for long-term memory, either "the actual respository of ganglionic patterns or the mechanism of reactivation of the record (of the stream of consciousness)" (Roberts, 1961).

One way of dramatizing the significance of these developments is to contemplate this fact: it is now quite within the purview of established technology to chronically implant a small cluster of electrodes deep within a human brain, attached to a small power cell and radio receiver screwed to the skull--small enough to hide under one's hat--and then by telemetry to produce in that subject an intense, protopathic feeling of terror, or an equally intense experience of euphoria, ecstacy or exhaltation, or (probably although localization here may be less certain) a feeling of implacable hatred or a frenzy of rage--at the whim of the experimenter and the press of a button. It is a sobering thought.

Schachter's Contributions

I shall devote this section to a review of some recent work by a social psychologist using physiological techniques (among others) to produce findings which are of great interest to the personality theorist and to the student of emotion in particular. It is well known that the peripheral sympathetic arousal produced by the injection of adrenalin most commonly yields a state of "as if" emotion--"I feel as if I ought to be scared, but I'm not"--although a real emotional reaction (fear) sometimes does "break through" (Marañon, 1924; Hawkins et al, 1960). This ability to experience the usual visceral accompaniments of fear without subjective fear was a principle basis for Cannon's (1927, 1929) refutation of the James-Lange theory of emotion. However, a recent study of human paraplegics, deprived by high spinal lesions of most peripheral sympathetic activity, suggests that the subjective emotional experience of these individuals is also distorted--weaker, more superficial, rather passionless; "It's a mental kind of anger;" "I say I am afraid....but I don't really feel afraid" (Hohmann, quoted in Schachter, 1964).

Schachter (1964) sees these two sets of introspections as "opposite sides of the same ccin. Marañon's subjects report the visceral correlates of emotion, but in the absence of veridical cognitions do not describe themselves as feeling emotion. Hohmann's subjects describe the appropriate reaction to an emotion-inducing situation but in the absence of visceral arousal do not seem to describe themselves as emotional. It is as if they were labeling a situation, not describing a feeling." Schacter and Singer (1962) found that they could produce apparently genuine subjective emotions (euphoria, anger) in normal subjects after epinephrine injection, providing

(1) the subjects were misled as to the effects to be expected from the drug and thus not allowed to attribute their visceral sensations to the injection and (2) an identifiable emotional context was provided by having the subject, after the injection, sequestered with another "subject" (a stooge) who behaved either in a gay or in an angry fashion. Schachter (1964) concludes, "given a state of physiological arousal for which an individual has no immediate explanation, he will 'label' this state and describe his feelings in terms of the cognitions available to him. To the extent that cognitive factors are potent determiners of emotional states, it could be anticipated that precisely the same state of physiological arousal could be labeled 'joy' or any of a great diversity of emotional labels depending on the cognitive aspects of the situation. Given the same cognitive circumstances, the individual will react emotionally or describe his feelings as emotions only to the extent he experiences a state of physiological arousal."

These experiments provide the most striking empirical support yet available for Lindsley's (1951) activation theory of emotion--the notion that there is a single dimension of activation or arousal common to all at least of the 'excited' emotions--although Schachter emphasizes rather more than Lindsley does the role of visceral as well as the central components of arousal. Arousal, however, is a necessary but not sufficient condition for emotional behavior or experience. The "other side of the coin" is the cognitive activity of the subject who will feel emotion only if he labels his aroused condition as emotional. The quality of the emotion he then feels depends also on the particular emotional label he chooses. But, surely, neither cognitive activity nor labeling is a

sine qua non for emotional experience. One supposes that the presence of a particular emotion implies the activity of some particular center or assembly in the diencephelon which is reciprocally interconnected with the "higher" centers subserving perception, symbolic activities and the like; thus do we know how we feel and thus feel what we know. The activation theory implies further (or so it would seem to me) that each such primary emotion center is also reciprocally connected with the brainstem activating system; nonspecific arousal however induced, whether naturalistically or by epinephrine injection, would seem to lower the thresholds of the primary excited emotions, such as joy, fear and rage. (Is grief a primary emotion and does it share the activation component? Depression, at least, seems to involve reduced CNS arousal). Thresholds lowered, the primary centers wait upon centrifugal activation from above. Clinical evidence strongly suggests that there must be some sort of mutually inhibitory process interlinking the primary centers or at least a positive feedback mechanism so that, e.g., fearful cognitive content activates the (potentiated) fear center which in turn provides stimulus support for continued fearful mentation, and so on; once fear holds sway, it is surely difficult to elicit euphoria or rage. By the same token, anger is an inhibitor of fear and one suspects that self-induced rage is one of the more popular tranquilizers.

If fear is a chief inhibitor of antisocial or psychopathic behavior, and if undifferentiated arousal is a sine qua non for fear, then normal subjects in the presence of ethical conflict should be more inclined to yield to temptation if their autonomic arousal is somehow artifically damped. Schachter and Latené (1964) found that some 20 per cent of a control group of college girls cheated while correcting their own answer

sheets in a psychology course quiz, but that nearly 40 per cent of another group cheated in the same situation after premedication with the tranquilizer, chlorpromazine. The present writer had found some years ago that the true primary psychopath of the Cleckley type shows low clinical anxiety, deficient anxiety conditioning and negligible avoidance learning in a task motivated in normal subjects by fear of a painful shock (Lykken, 1957). After replicating the latter finding on two sets of prison inmates selected to meet criteria of 'primary psychopathy', Schachter and Latené then found that the psychopaths showed a sharp increase in avoidance learning after premedication with epinephrine. The implication here, of course, is that the poor avoidance learning of the psychopath, manifested both in the laboratory and in the antisocial activities leading to his incarceration, is the consequence of a relative lack of fear resulting from a relative deficiency in nonspecific arousal; artifically augmenting arousal by means of epinephrine injection produces an evanescent display of normal avoidant behavior.

Schachter and Latené, however, noted that their psychopathic subjects had actually higher heart rates than did their nonpsychopathic prisoners at the start of the experiemnt and also a greater elevation in HR during the avoidance task under the epinephrine condition. Similarly, in their college girl sample referred to above, subjects scoring below the median on the Lykken (op. cit.) scale of anxiety reactivity showed significantly higher heart rates after taking the examination than did those whose anxiety scores were above the median. Finally, Valins (1963), also using college students, found that subjects with low Lykken anxiety scores gave a significantly greater increase in HR in response to a stress

situation than did high-anxious subjects as defined by the same test. These findings lead Schachter and Latené to speculate along the following lines. Perhaps some psychopaths tend to be hyper-reactive to virtually any titillating event, to be autonomically aroused by circumstances which would be frightening to others and also by events which most people would consider harmless. "Such generalized, relatively indiscriminant reactivity is, we would suggest, almost the equivalent of no reactivity at all. If almost every event provokes strong autonomic discharge, then, in terms of internal autonomic cues, the subject feels no differently during times of anger than during relatively tranquil times. Bodily conditions which for others are associated with emotionality are, for the sociopath, his 'normal' state. It would appear from the data on the effects of adrenaline on avoidance learning that only intense states of autonomic reaction, presumably stronger than and differentiable from his normal reactions, acquire emotional attributes for the sociopathic subject." Schachter and Latené do not reject the notion that some psychopaths are relatively hypo-reactive but argue rather that a similar behavioral phenotype may result, under certain circumstances of e.g. childhood experience, from the obverse condition of autonomic hyper-reactivity and, indeed, were able to report that their psychopathic prisoners gave a cleanly bimodal distribution of heart rates, one mode well above and the other symmetrically below the single mode of the HR distribution given by the nonpsychopathic prisoners.

This idea that there may be important individual differences in the ability to discriminate different emotional or other subjective states is provocative and has implications for other phenomena beyond psychopathy.

Thus, Schachter is currently following up the suggestion of the psychoanalyst, Hilde Bruch (1961) that the root problem in chronic obesity is a failure to discriminate between the feeling of hunger and the feelings of anxiety or emotional stress, a confusion due perhaps to a parental habit of feeding the infant in response to every indication of distress whether hunger-related or not. Alternatively, however, Schachter's finding that the psychopathic subject displays accelerated heart rate in stress situations may possibly be related to the interesting report of Lacey, et al (1963)--treated more fully later in this chapter--that nociceptive stimulation tends generally to accelerate the heart. This, Lacey believes, decreases cortical arousal and attenuates exteroceptive input via an inhibitory feedback circuit from the corotid sinus. Perhaps Schachter's "hyper-reactive" psychopaths were really individuals in whom this cardioacceleratory-CNS-inhibitory mechanism is unusually well developed, allowing them to damp stressful input or the distress response which it produces in them. In any event, it seems clear now that heart rate does not vary monotonically with arousal or emotional excitement and HR alone cannot be safely used as an indicant of emotional reactivity. Similarly, Schachter's analysis of the inter-relation of anxiety and oral stimulation in obesity should be contrasted with the compelling proposal of Kessen and Mandler(1961) that oral stimulation is an innate inhibitor of undifferentiated distress; it seems very possible that the obese individual eats not because he misinterprets anxiety for hunger but because eating actually quelles his emotional discomfort, at least temporarily.

THE EXECUTIVE FUNCTIONS OF THE BRAIN STEM RETICULAR FORMATION

One of the most active and exciting areas of neuropsychological research

during recent years has been the study of the reticular activating system (RAS) in the brain stem, which, together with related higher structures, appears to govern such basic functions as sleep, wakefulness and cortical arousal, the modulation or filtering of sensory input and the orientation of awareness or attention. As early as 1937, Bremer had shown that cutting the brain stem just above the level of the pons (this is the cerveau isolé preparation) produced, in the cat, a state of coma together with a pattern of EEG activity characteristic of normal sleep. The real surge of research into these matters, however, waited upon the demonstration by Moruzzi and Magoun in 1949 that direct electrical stimulation of the midbrain reticular formation in the sleeping animal produces immediate behavioral awakening together with the EEG arousal reaction that would normally accompany arousing the animal by means of sensory stimulation. There followed a crescendo of research which still continues in laboratories all over the world. Discussion and resumés of subsequent developments which are of particular interest to psychologists may be found in the excellent review by Samuels (1959), in Lindsley's authoritative chapter in the Handbook of Physiology (1960), in Magoun's elegant little book, The Waking Brain (1963) and in a wide-ranging and important monograph by Berlyne (1960).

Anatomical Relationships

The reticular formation is a strip of nervous tissue, including nerve cells of many types and sizes with diffusely branching or reticulated nerve fibers, which extends upward through the central core of both sides of the brain stem from the level of the medulla to the thalami (see Fig. 1). All

sensory pathways which run from the peripheral receptor organs or from the

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Figure 1

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interoceptors via the thalamus to the primary projection areas in the cortex, give off in addition collateral fibers into this reticular substance. Significantly, in view of the role played by the reticular mechanism in arousing the cortex, tracts subserving pain sensation are especially widely interconnected in these regions; indeed, about two-thirds of the fibers in the classical lateral "spinothalamic" tract appear to terminate within the upper brain stem (Magoun, 1963). Higher brain centers including many regions of the cortex, the cerebellum, and the structures of the limbic system thought to be concerned with emotional experience, send fibers into the reticular substance through which they can influence its activity. The most important sources of cortico-reticular influence appear to be regions of the frontal poles, the sensorimotor cortex and the cingulate gyrus. Thus, the reticular formation (RF) is equipped to monitor the entire flux of sensory input, to receive excitation, information or commands from other regions, and presumably, to subject this incoming information to some degree of analysis and integration, as indicated by the rich interconnections of the myriad reticular neurons and the fact that single units have been shown to be responsive to stimulation from different parts of the body and even through different modalities.

The outgoing or efferent connections of the RF are equally complex and ubiquitous. In the upward direction, there is an extensive reticulo-cortical pathway involving fibers which radiate diffusely to nearly all

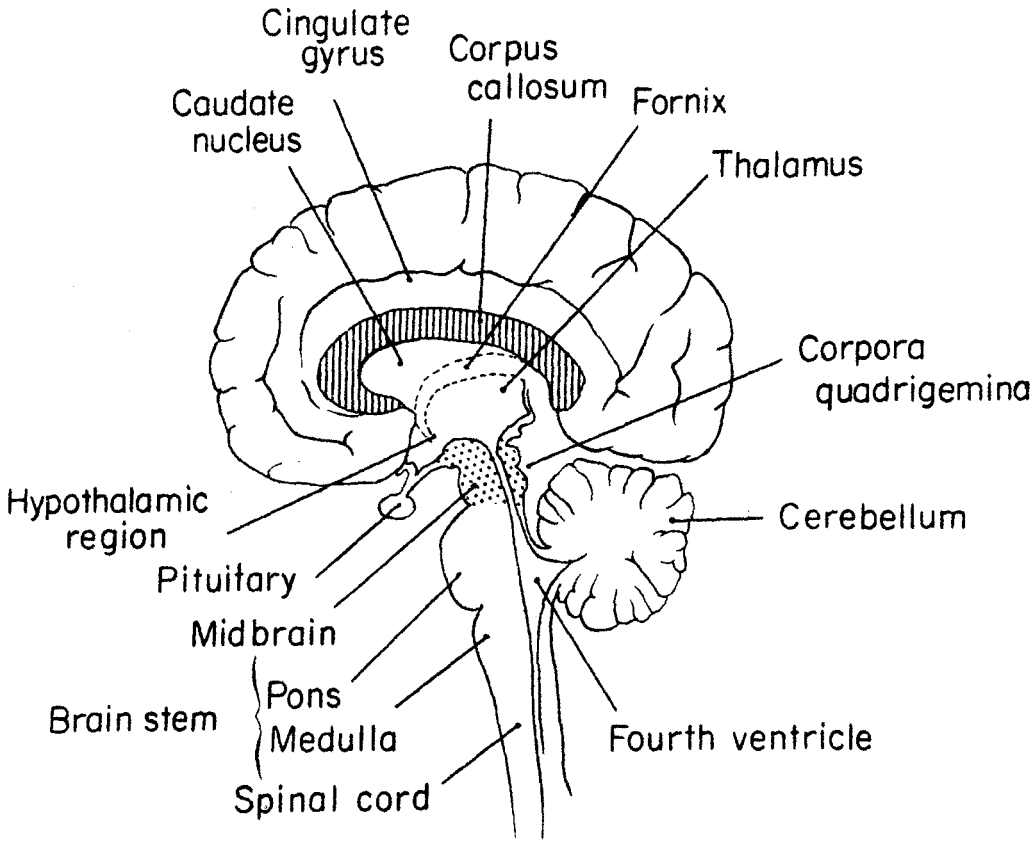

Figure 1. Median view of right cerebral hemisphere.

areas of the cerebral cortex from origins in the midbrain reticular substance; this is the ascending reticular activating system (ARAS) of Moruzzi and Magoun. Another and relatively independent system appears to originate somewhat higher in the intralaminar nuclei of the thalamus, considered to be the rostral extremity of the reticular formation. This is the diffuse thalamo-cortical projection system (DTPS) which is now known to be anatomically distinct from the specific thalamo-cortical system (STPS) over which sensory information is transmitted in a specific, point-to-point fashion from the relay nuclei of the thalamus to the sensory projection regions of the cortex. There appears to be at least one synaptic relay in the DTPS since cortical destruction does not lead to degeneration within the intralaminar nuclei but the site of this relay and origin of the final common path of the DTPS has not yet been definitely located.

Stimulation of the midbrain ARAS produces a long-lasting and very general activation of the cortex coupled with behavioral arousal--waking of the sleeping animal or an increase in alertness or excitement in a subject already awake. High-frequency electrical stimulation of the DTPS can produce similar results although the activating effects of this system appear to be more phasic and delicate in characeer, with shorter latencies and briefer duration, and, unlike the ARAS, the DTPS appears capable of activating localized regions of the cortex with some selectivity. The DTPS is known to be capable also of an opposite, inhibitory influence on the cortex. Low frequency stimulation of the nonspecific thalamic nuclei can produce synchronization rather than activation of cortical areas which, if sufficiently wide spread, may be accompanied by behavioral relaxation, lassitude or sleep. Centers capable of driving this inhibitory mechanism of the DTPS have been

localized in limbic system structures and in the hypothalamus (Magoun, 1963). The initial and lighter stages of sleep may result from influences arising in an inhibitory center in the lower, bulbar portion of the RF which tends to deactivate the ARAS. Still another inhibitory center, situated in the tegmental region of the pons, has recently been shown to be responsible for the deepest level of normal sleep in which midbrain and diencephalic structures are inhibited and the cortex, while cut-off from the ARAS excitation, is also freed from the inhibitory influences of the DTPS.

In the down-stream direction, there are first the classical reticulo-spinal efferent fibers capable of both facilitating and inhibiting spinal motoneurons and which are thought to be concerned with the integration and coordination of striped-muscle activity. Secondly, there is the complex gamma-efferent system of fine, myelinated gamma fibers which lead off to the receptor organs of the proprioceptive system, the muscle spindles. By means of this mechanism, the RF is capable of either augmenting or attenuating proprioceptive feedback, thus providing an additional and versatile means of controlling motor activity. Even more generally, it has now been established that the reticular formation sends efferent fibers outward to the relay nuclei of all incoming sensory pathways as well as to the peripheral receptor organs themselves.

Thus, deep within the very stem and core of the brain, we find a kind of inner brain or communications center equipped to monitor all incoming sensory information and to deliver some sort of commands to the peripheral pathways carrying that information and which is richly interconnected with the higher brain structures and with the cortex itself. Included here are

systems for controlling sleep and wakefulness and the level of tonic activation of the cortex generally as well as the phasic fluctuations of excitability of more localized cortical areas. The mechanism of attention appears to reside here as well as an apparatus for screening sensory input which, as we shall see, seems to be capable of crude perceptual analysis in its own right. In collaboration with the (not necessarily activated) cortex, this mechanism may be capable of complex pattern recognition as well, and may primarily determine which information is to be passed on to the higher executive system.

Cortical Activation and Behavioral Arousal

The EEG and the Arousal Continuum

Much of our understanding of the reticular system depends heavily upon the use of the EEG as an indicant of the level of cortical arousal. Table 1 (modified from Lindsley, 1961) summarizes some of the principal relationships. EEG tracings recorded from the occipital and parietal areas of an awake but relaxed subject tend to be dominated by the alpha

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Table 1

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rhythm which consists of waves with a frequency in the range from 8 to 13 per second and an amplitude of some 20 to 50 microvolts. When the subject is aroused, as by emotional stimulation or by some task demanding concentration, these alpha rhythms give way to a pattern of low voltage fast activity (often called beta waves) which represents the activated or desynchronized EEG. Unexpected or novel stimuli, presented to a relaxed waking subject, produce a transitory interval of fast activity sometimes

Table 1. The Arousal Continuum: EEG and Behavioral Correlates (Modified from Lindsey, 1960)

Arousal Continuum	EEG	State of Awareness	Electrocortical Response	Behavioral Characteristics
Strong, excited emotion; fear, rage, anxiety	Desynchronized: low to moderate amplitude; fast mixed frequencies	Restricted awareness, narrowed attention span, confusion, strong sensory suppression	Probably widespread inhibition of evoked potentials	Disorganized, poor discrimination & coordination, may be increased strength & indurance for primative "flight-flight" functions
Alert attentiveness	Partially synchronized (periods of alpha activity) mainly low voltage, fast activity (beta waves)	Selective attention, concentration, anticipation	Selective inhibition and facilitation of evoked potentials	Efficient, fast reaction time, optimum discrimination and coordination. May be too well focused for optimum creativity
Relaxed wakefulness	Synchronized: Mainly alpha rhythm at moderate voltage (8-12/sec, 30 μ volts)	Attention wanders not forced, favors free association	Either alpha blockage or evoked potentials in alpha background	Broadened attention span, quickly activated, capable of routine activities, perhaps optimal for reflection and creative thought.
Drowsiness	Stage 1 (D&K), stages A & B (Loomis) Alpha fades out, low voltage, irregular fast frequency. Transition phase only	"Dreamy", hypnogogoic state, partial awareness, imagery & reverie		Reduced awareness, slower to arouse, poor sensorimotor function. GSR suppression.
Light sleep	Stage 2 (D&K), Stage C (Loomis) Low voltage background with 14/sec sleep spindles and K-complexes	Loss of consciousness but rather easily awakened	Evoked potentials changed from waking wave shape	Capable of simple conditioned motor responses, Sidman-avoidance. Responds selectively to own name. Graded EEG and vascular response to graded intensity of auditory stimuli. GSR reappears.

Table 1. (cont.) The Arousal Continuum: EEG and Behavioral Correlates

Arousal Continuum	EEG	State of Awareness	Electrocortical Response	Behavioral Characteristics
Moderate sleep	Stage 3 (D&K), Stage D (Loomis) Intrusion of random high voltage, slow waves (1-4/sec, 50-100 m volts) some spindling	Sound sleep	Evoked potentials further modified in wave shape, especially K-complex	
Deep sleep	Stage 4 (D&K), Stage E (Loomis) Mainly large, random slower delta waves (1-2/sec, 50-150 m volts) No K-complexes	Sound sleep, high arousal thresholds	Evoked potentials similar to Stage 3	Intermittent simple motor response to stronger stimuli, Sidman avoidance, reduced EEG responsiveness but vascular response to noise stimulus unaffected.
	Stage 5, Stage 1_{REM} (Williams et al, 1963). Identical appearance to stage 1 (desynchronized, low voltage) but accompanied by rapid eye movements (REM), occasional jerky movements, reduced muscle tone. (Jouvet's "rhombencephalic sleep")	Deep sleep, highest arousal thresholds, frequently or always accompanied by dreaming	Evoked potentials markedly attenuated	Motor and vascular responsivity as in stage 4, heart rate slows and blood pressure drops, reduced EMG activity, penile erection, possibly random variation of palmar conductance.
Barbiturate Narcosis, Coma	Large, slow waves (delta) with periods of no activity in deeper stages	Complete loss of consciousness, little or no response to stimulation, amnesia	Evoked potentials present and strong	As in deep sleep?

called alpha blockade. Conversely, as the subject drifts off into sleep, the alpha fades out and is replaced by irregular 14-per-second bursts known as sleep spindles, often punctuated by large, slow oscillations called K-complexes. Deeper sleep is signalled by an increasing proportion of slow, large waves, called theta waves in the range from 4 to 7 per second or delta waves when slower than 4 per second. Finally, in deep sleep, the record is dominated almost entirely by still larger, slower delta activity. Several methods of classifying the EEG stages of sleep have been proposed, the most commonly used being a letter system (Loomis, Harvey and Hobart, 1937) and the number system more recently employed by Dement and Kleitman (1957). The two schemes can be roughly equated as indicated in Table 1.

As arousal increases above the level of drowsiness, marked by the low voltage irregular activity of Stage 1, there is an increase both in the amount of alpha present in the record ("percent-time alpha") and also an increase in the amplitude of the alpha waves. Further increase in arousal decreases alpha once again as the record approaches the completely "desynchronized" pattern characteristic of excitement. This inverted U-shaped relationship of alpha amplitude to arousal has been specifically supported by Stennett (1957) using palmar conductance as an indicant of arousal.

Figure 2

One can see here a rough trend for the brain waves to decrease in voltage but increase in frequency as the subject moves from deep to light sleep to relaxed wakefulness and finally to more alert activity. This picture has been complicated, however, by recent evidence that normal

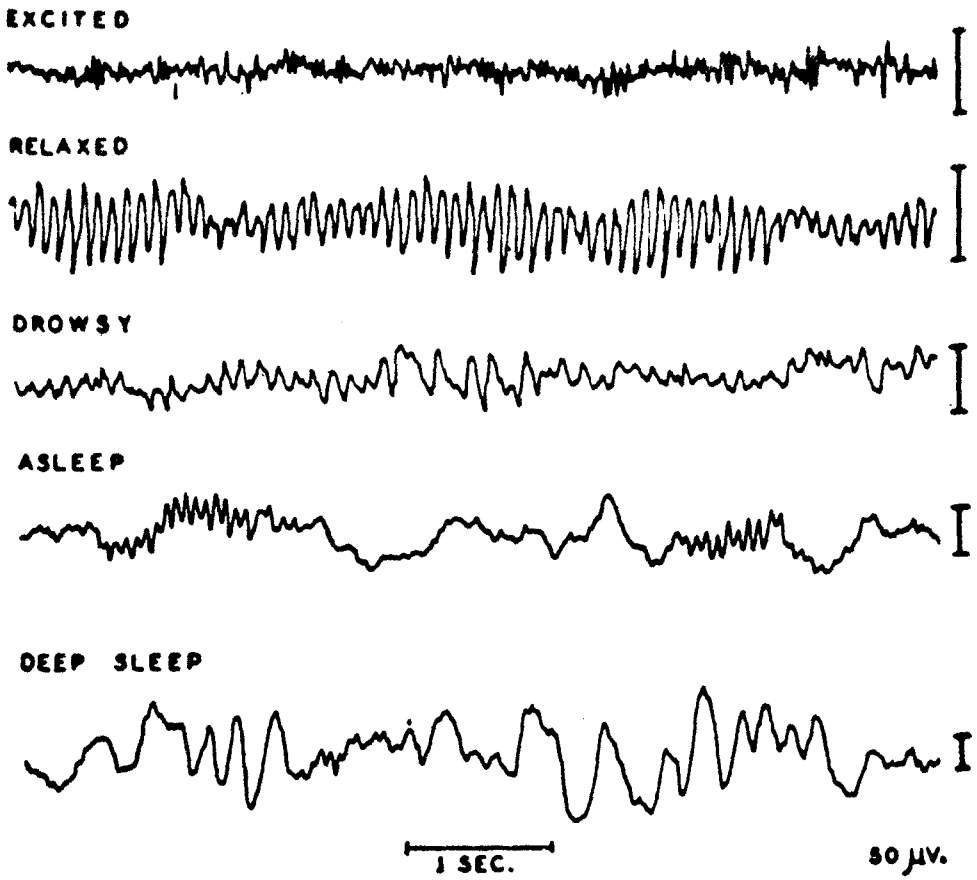

Figure 2. Characteristic EEG tracings from a normal human subject during various excitatory states from sleep to wakefulness (From Jasper, 1941.)

sleep is punctuated by intervals in which the EEG reverts to the low voltage irregular activity characteristic of activation but during which the subject is substantially more difficult to awaken than even in the high voltage slow wave sleep of Stage 4 (Jouvet, 1961; Williams et al, 1964). During these intervals of "paradoxical sleep," rapid eye movements (REM) can be detected and there is a general lowering of muscle tone and EMG activity although occasional jerky limb movements may be seen. Systolic and diastolic blood pressure is sharply reduced during these periods (Guazzi and Zanchetti, 1965). The work of Kleitman and his students (e.g., Kleitman, 1961) indicate that it is during these intervals when most or all of normal dreaming occurs. This deepest sleep level has been referred to as Stage 1_{REM} (Williams, et al, 1964) because its EEG pattern seems to be identical to the low voltage, irregular activity of the transitional Stage 1 which follows the disappearance of alpha in drowsiness and preceeds the onset of sleep spindling and delta activity. Since this stage is now well established to be deeper, in the sense of heightened arousal thresholds, than the hitherto deepest Stage 4, it seems appropriate and simpler to refer to this level of "activated" sleep as Stage 5, keeping in mind that this "stage" is not in fact discriminable from Stage 1 on EEG criteria alone.

Jouvet (1961) has shown in the cat that Stage 5, which occurs only after periods of lighter sleep (often Stage 2), is controlled by a center in the pontile tegmentum which appears to exert inhibitory control over the nonspecific systems of both the thalamus and midbrain. Pontile inhibition of the synchronizing mechanisms of the DTPS releases the cortex into desynchrony (it may be this effect which allows the presumably

cortical activity of dreaming during Stage 5, as Magoun [1963] suggests) and pontile inhibition of the ARAS increases by several times the intensity of sensory stimulation required to arouse the sleeper. Cortical evoked potentials produced by sensory stimuli, which are modified as to shape in the deeper stages of synchronized sleep but not reduced in amplitude, are very much smaller during Stage 5 (Williams, Tepas and Morlock, 1962) which may reflect the absence of the normal supplementary feedback from the now inhibited reticular mechanisms. Huttenlocker (1960) reports that

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Figure 3

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evoked potentials recorded from the midbrain RF and elicited by auditory click stimuli are large and undiminished even after 1000 repetitions in the waking animal but rapidly diminish to about half-amplitude during slow-wave sleep. During Stage 5 sleep there is almost complete suppression of midbrain response to these repeated clicks.

An important investigation by Williams, Hammack, Daly, Dement and Lubin (1964) provides additional evidence that cortical and behavioral arousal can vary to some extent independently of the state or stage of the EEG. Subjects were stimulated by repeated accoustical noise bursts of varying intensities during all EEG sleep stages both before and after 64 hours of sleep deprivation. Three types of response to these sounds were recorded: EEG changes, including K-complexes and alterations in frequency, finger vasoconstriction, and an operant response (pressing a microswitch) which the subject, while in the waking state, has been instructed to emit after each sound. It was found that the amplitude

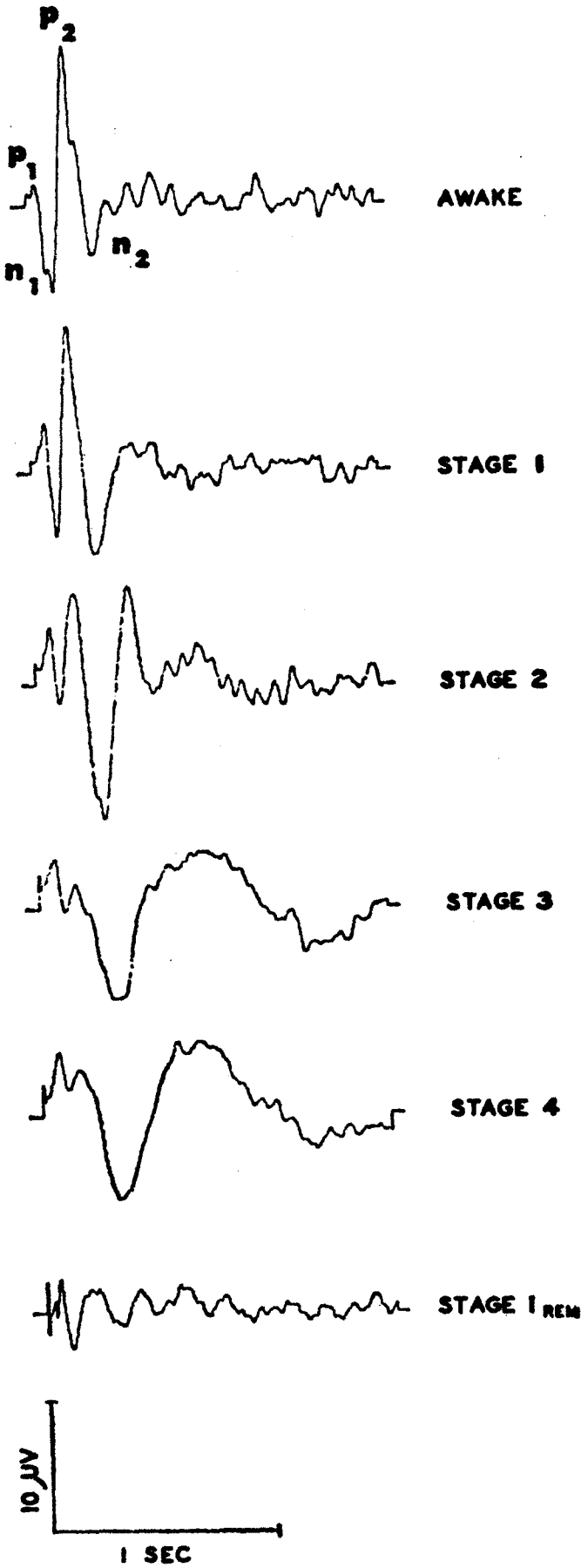

Figure 3. Averaged evoked cortical responses to auditory clicks as a function of the stage of sleep. EEG recorded between an electrode on the vertex and another in the left occipital region. Each response represents an average of 100 EEG segments, each 1536 mSec long following the presentation of the click stimulus. (From Williams, Tepas & Morlock, 1962.)

or frequency of response increased systematically with increasing sound intensity at all sleep stages. Even noise levels near the subjects' waking threshold of audibility produced fairly consistent EEG and vasoconstrictor responses during both light and deep sleep. Response amplitudes were considerably diminished at all sleep levels after extended sleep deprivation, however, indicating that depth of sleep as defined in terms of the reactivity of the brain is not perfectly correlated with electrocortical activity. As additional evidence, responsiveness to stimulation during moderate Stage 2 sleep (which occurred frequently enough throughout the night to allow such tabulation) was found to decrease systematically as the night wore on in the case of both the behavioral and the vascular responses, although the EEG reactivity did not systematically change. While these subjects showed less EEG and behavioral reactivity to the stimuli during Stages 4 and 5 (Stage 1_{REM}), consonant with other evidence that these stages represent deeper sleep levels, there was no differential responsiveness in vasoconstriction. One wonders whether this latter finding may be related to the activated condition of the diencephalon during Stage 5 sleep (Jouvet, 1961) and to the labile emotionality which it thought to be characteristic of dreaming. The recent demonstration by Fisher, Gross and Zuch (1965) that in human male subjects penile erection is an almost invariable accompaniment of Stage 5 sleep intervals seems relevant in this connection.

Finally, some indication must be given of the limits of this concept of an arousal continuum. It is possible, but certainly not proven, that the level of nonspecific excitation of the ARAS increases monotonically as one moves from the bottom to the top of Table 1, but clearly

"behavioral efficiency" does not do so, falling off at the high end, nor does "cortical activation" which is apparently higher in Stage 5 than in presumably lighter stages of sleep. Moreover, the inflection in psychological functioning at the junction of sleep and waking is so abrupt that this schema is potentially misleading. Indeed, the existence of so many inter-related excitatory and inhibitory mechanisms, both neural and hormonal, counterpoised in a still poorly understood manner, makes the notion of an unidimensional arousal continuum seem somewhat precarious, except perhaps as a temporary working hypothesis subject to prompt reformulation as the steady flow of new evidence may require.

Sleep.

Although some of the most basic questions still remain unanswered, many facts about the properties and mechanisms of sleep have been uncovered in the course of the last two decades. First of all, it seems fairly well established that sleep is the stage which supervenes when the cortex is deprived of the energizing influence of the ARAS. There is a reciprocity in the connections between cortex and midbrain so that the cortex can control its own activation to a certain extent by corticifugal excitation or inhibition of the ARAS. The onset and lighter stages of sleep begin with a synchronization of the cortex via the inhibitory mechanism of the DTPS followed by a cortically induced inhibition of the ARAS. The synchronized cortex apparently drives the ARAS into parallel slow-wave activity, thereby lowering its reactivity (Jouvet, 1961). Perhaps equally important, the inhibited cortex transmits less tonic excitation over the cortico-reticular circuits. An inhibitory center in the bulbar RF appears also to inhibit the ARAS during sleep either directly (Moruzzi,

1964) or indirectly by driving the DTPS as Magoun (1963) suggests. Once sleep has been attained, the inhibitory center in the pons is somehow triggered into action periodically (perhaps at about 90-minute intervals in man) during which time the ARAS is directly and powerfully inhibited together with the thalamic mechanisms (Jouvet, 1961).

A segment of the midbrain tegmentum is known to be strongly responsive to adrenalin (Rothballer, 1956) so that built-up epinephrine concentrations resulting from excitement and high arousal tend to retard the deactivation and inhibition of the ARAS. It has long been supposed that there must be a symmetrical hormonal mechanism having an inhibitory effect which, once in action, would act to damp the more labile neural mechanisms and help maintain uninterrupted sleep. A recent article (Monnier and Hosli, 1964) reports the discovery of such a substance, although its chemistry and site of action are not yet known,which when concentrated from the blood of a sleeping animal can be injected into the bloodstream of a waking animal thereby causing it to fall asleep.

Magoun (1963) tends to regard the cortical inhibitory mechanism of the DTPS as the agent involved in those phenomena which Pavlov grouped under the heading of <u>internal inhibition</u>: Any stimulus associated with the withholding of reinforcement may come to produce spindle bursting and synchronization in cortical regions: a CS during extinction trials; a discrimination stimulus which is never reinforeed; a conditioned inhibitory stimulus whose presentation together with the CS means that reinforcement will be withheld on the trial; or the early portions of a protracted CS-US interval in the "inhibition of delay" experiment. Similar effects have been observed on the EEG of experimental animals for intervals

following copulation or feeding, and it seems probable that stimuli associated with satiety and with sleep or relaxation can become able through conditioning to produce similar inhibitory responses of the DTPS. Bonvallet, Dell and Hiebel (1954) demonstrated that distention of the carotid sinus, as might result from an increase in blood pressure, acts through the bulbar inhibitory center to drive the DTPS mechanism, producing cortical deactivation and slow waves. This finding is the basis of Lacey's conjecture, discussed further in a later section, that blood pressure increase following noxious stimulation may serve to attenuate sensory input while blood pressure decrease in situations requiring attention to the environment may have the opposite effect.

These phasic and relatively localized inhibitory effects upon the cortex by the DTPS must serve specialized adjustive functions during the waking state, but whenever they are unusually strong or occur against a background of reduced ARAS excitation, their influence can spread. Heads nod during the Sunday sermon not because of the excesses of the night before (at least, not in every case) but because sensory input is minimized and proprioceptive feedback as well, while inhibitory activity is maximized; stop coughing, quit squirming, don't look around. Add to this, post-prandial inhibition following a large breakfast, a regular, hypnotic rhythm in the speaker's voice, insufficient content in the sermon to support cortical activity, and spreading inhibition of the cortex must result. Trying not to go to sleep under these conditions is notoriously ineffectual, possibly because trying not to do anything involves inhibitory processes. A better solution, although not without risk, is to yield to drowsiness in the hope that one will catch one's

self just as the head falls limply forward; the orienting reflex and adrenaline secretion which this narrow escape produces can be counted upon to countereact somnolence for as long as half an hour.

The loss of adaptive function during sleep is probably relatively much greater for man than for other animals because the capabilities most affected are precisely those which are distinguishingly humanoid. The sleeper cannot perform any sort of logical analysis of a complex stimulus nor enrich his interpretation by means of recollections or association of ideas. Much of our waking commerce with the environment involves a continual process of learning--one learns something about the identity of the speaker and interprets what he says accordingly; one notes and remembers that no one else is in the room so that what is said must be addressed to one's self, etc.--and it appears that at least this kind of learning ceases during sleep (Lindsley, 1960).

However, the contrast between sleep and coma is considerable also. First, of course, the sleeper can awaken reasonably quickly in reaction to almost any strong stimulus, the exceptions being those recurrent disturbances to which the subject has become habituated after long experience: the traffic noises, the passing train, the moderate and chronic pains. The sleeper can still make use of his biological clock to awaken at the accustomed time just before his alarm goes off or to be disturbed in his sleep if the Midnight Limited fails to rumble past on schedule. After some practice while awake, he can continue to perform on a Sidman-avoidance schedule even during deep sleep, regularly pressing a key which delays an electric shock another three seconds (Granda and Hammack, 1961). The same subjects also pressed a second key with their other hand, being

rewarded after a fixed number of responses by a five to eight minute "time-out" period, during which shock was stopped so they could sleep undisturbed and this behavior showed much the same regularity found with waking subjects. A conditioned stimulus previously associated with electric shock can produce partial or complete arousal (Rowland, quoted in Magoun, 1963), although that same tone at that same loudness would not affect a naive animal. The human sleeper can be selectively aroused by stimuli having special significance for him. Oswald, Taylor and Triesman (1961) instructed subjects to press a key during sleep in response to hearing their own names. When a long list of spoken names was played over and over during the night, most subjects showed selective partial arousal to their own names and did press the key appropriately on a high proportion of trials. Considerable research will be required before the true limits of behavior under sleep can be established and one cannot help but think that such information must make an important contribution to our understanding of the waking organism also.

Dreaming. The work of Kleitman and his students (reviewed in Kleitman, 1961) inaugurated a new era in the scientific study of dreaming. At intervals of about 70 to 90 minutes during the night, a normal subject displays a minutes-long period of desynchronized (Stage 5) EEG activity coupled with frequent rapid eye movements (REM). Wakened during such periods, most subjects report that they were dreaming whereas dreaming is seldom reported when the sleeper is awakened during other EEG stages (Dement and Kleitman, 1957a, 1957b). It appears that some 20 per cent of one's sleeping hours are occupied with dream activity, and this value holds with surprisingly little variation across individuals. When

subjects were deprived of the opportunity to dream by awakening them within a minute or two after the start of each REM period, they showed a striking increase in the number of such dream periods initiated and a considerable increase in "dream time" measured during undisturbed nights following the four to seven night deprivation interval (Dement, 1960). However, both Berger and Oswald (1962) and Williams, et al (1964) find that when subjects are deprived of all sleep for a lengthy period (e.g., 64 hours), the first recovery night shows a sharp increase in time devoted to Stage 4 sleep with perhaps even a small decrease in time devoted to (Stage 5) dreaming. Stage 4 time is reduced by the second recovery night while Stage 5 time increases, leading Williams, et al, to infer that, with total sleep deprivation, the need for Stage 4 sleep is dominant and must be partly compensated before the lesser need for dreaming can assert itself.

Getting coincident results with several methods of recording, Fisher, Gross and Zuch (1965) have recently shown that nearly all Stage 5 periods are accompanied by partial or complete penile erection in the human male. Combined with other reports of decreased heart rate and blood pressure with labile "spontaneous" GSR activity, this suggests that dreaming may be commonly accompanied by a conditioned of generalized parasympathetic activation, probably related to the disinhibition of limbic system mechanisms during "rhombencephalic" sleep (Jouvet, 1961). Fisher, et al, speculate that the need for these Stage 5 intervals and the distress and irritability shown by Dement's subjects after deprivation may be related to some sort of emotional tension discharge which normally occurs at these times.

Before leaving the topic of sleep, one must ask why it is that we must spend a third of our lives in this curious condition? Muscle fatigue

can be relieved by rest without sleep. The brain itself may be less active and therefore "resting" during sleep but it is strange that its metabolism does not, in that case, seem to slow; Kety (1961) reports that cerebral blood flow and oxygen consumption is not reduced during sleep, indicating that the brain continues to consume its customary 20 watts of energy. Other contributors to the same symposium provide electrographic evidence that during sleep the activity of cerebral neurons changes in pattern but does not dimish in total amount. On the other hand, the majority of cortical neurons are too small to be studied individually by present microelectrode techniques and it is possible that these smaller units do, for some reason, "rest" during sleep.

Not only is it uncertain as to why we sleep but also the mechanism that initiates normal sleeping is still largely a mystery. In a recent authoritative review, Moruzzi (1964) points out that there must be some process which, during the waking hours, gradually increases the need for and the probability of sleep, but this process has not yet been identified. One would think that electronarcosis, the induction of sleep by application of mild, rhythmic electrical stimulation of the brain, would be a valuable experimental technique in this connection, e.g., the amount or duration of electronarcotic stimulation necessary to produce sleep might serve as a dependent variable in the study of factors thought to be related to sleep susceptibility. Although much used in the USSR in sleep therapy with Psychiatric patients (Obrosow, 1959), the electrosleep method seems to have not yet found its way into Western research laboratories (although see Forster, Post and Benton, 1963).

Cortical Excitability Cycles

The low-voltage high-frequency EEG characteristic of the alert, waking subject is referred to as "desynchronized" because of the belief, for which there is some support from microelectrode studies, that the excitability of individual cortical cells fluctuates at a fairly constant frequency and that the activity of these individual units is brought into phase or synchronized through the influence of the DTPS to yield the regular and relatively large ten per-second voltage waves of the alpha rhythm. ARAS stimulation disrupts this synchrony so that local regions cycle independently of their neighbors. Figure 4 (from Lindsley, 1961) illustrates how a number of such cortical regions operating out of phase but at similar

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Figure 4

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frequencies would produce an EEG picture of low voltage, irregular activity with the usual sort of bipolar scalp recording. Lansing, Schwartz, and Lindsley (1959) have shown that warning signals shorten reaction times when they occur early enough to produce desynconization of the EEG before the advent of the critical stimulus. EEG activation typically requires about 250 mSec and warning intervals shorter than this seem to have little or no facilitating effect. Lansing (1957) demonstrated that visual signals which reach the occipital cortex during a critical portion of the alpha cycle (the interval when the wave changes from positive to negative) produce shortened reaction times and similarly it appears that response commands are reacted to more quickly when they reach the motor cortex at the corresponding point in its "alpha excitability cycle."

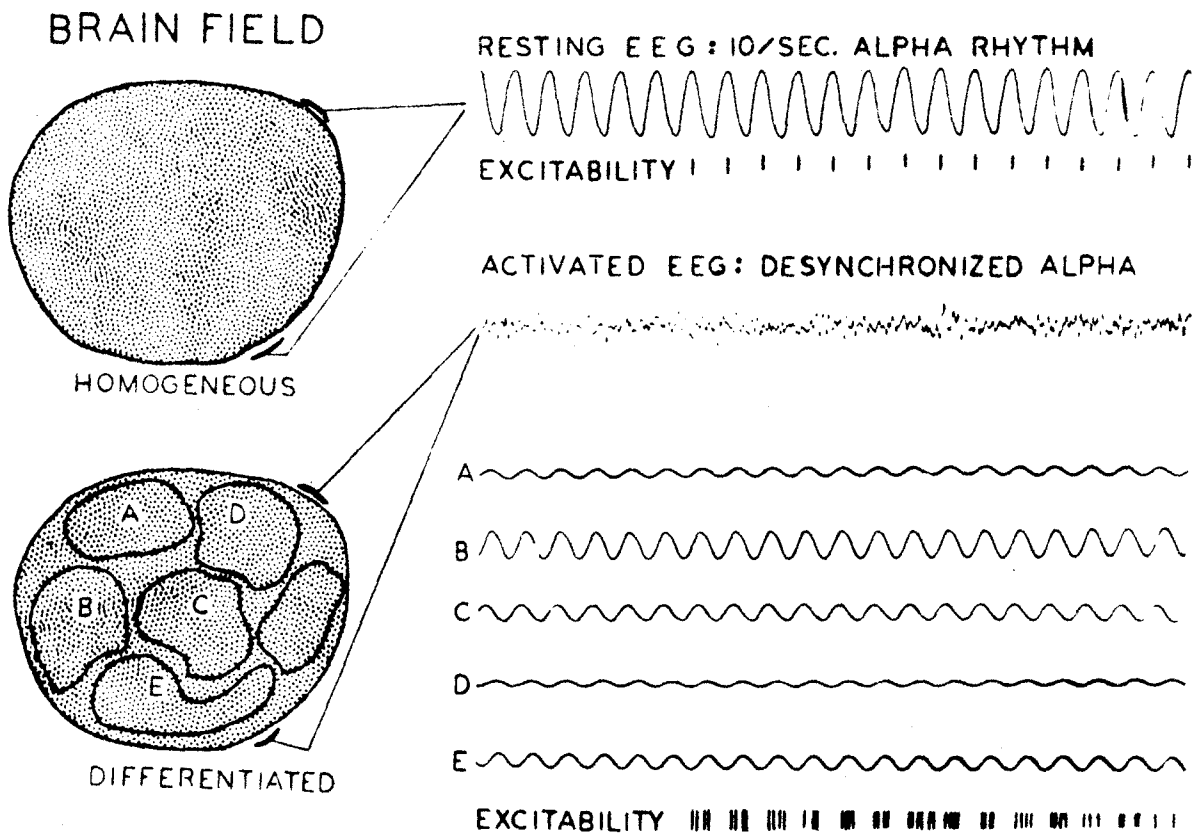

Figure 4. Hypothetical brain fields, illustrating Lindsley's theory of EEG desynchronization. Resting alpha at 10/sec characterizes the "homogeneous", or relaxed, condition. A desynchronized, activated EEG is associated with a "differentiated" state of brain function. According to the concept that an excitability cycle is associated with the waxing and waning phases of the waves, it is evident that alternating periods of excitability and inexcitability could occur only 10 times per second in the case of the resting EEG, whereas almost continuous excitability is represented in the case of the desynchronized EEG resulting from a differentiated brain field. (From Lindsley, 1961.)

Lindsley (1961) argues that these hypothetical excitability cycles in cortical units or in synchronized aggregates of units provide a kind of scanning or gating mechanism for sensory input and that desynchronization of a receiving area increases the probability that a given signal will find some unit(s) in a state of maximum receptivity at the moment of its arrival. (Perhaps the very slow delta waves of deep synchronized sleep work in the opposite direction, reducing sensory excitability. It would be interesting to compare arousal thresholds to brief sounds or shocks presented at different points in the delta wave cycle during Stage 4 sleep.)

Quite recently, Surwillo (1963, 1964) has reported some striking relationships between brain-wave frequency and reaction time in human subjects. Using careful measurements of the number of brain-wave cycles intervening between the stimulus (a tone) and the motor response (a switch closure), he divided this number into the S-R time interval, getting average brain-wave period--the reciprocal of frequency. This concurrent EEG period was found to correlate + .72 with simple reaction time across a sample of 100 normal males ranging in age from 29 to 99 years. Partialing out the correlation between reaction time and age raised this value to + .76. Moreoever, P-type or within-subject correlations between average EEG period and reaction time were almost all positive and averaged + .30. In a second study (Surwillo, 1964) involving 54 normal males, EEG period correlated + .76 with simple reaction time and also + .76 with "disjunctive" or choice reaction time. In both studies, EEG frequency was found to decrease with age.

Surwillo proposes, "that the EEG cycle is the basic unit of time in terms of which events are programeed by the central nervous system, [which] means simply that time in this domain is reckoned by the brain-wave cycle.

Hence, the speed with which a response may be elicited will be determined by the number of cycles required for organizing the particular events leading to the response" (Surwillo, 1963, p. 105). One possible difficulty in these experiemnts lies in the fact that EEG period is computed from a ratio in which reaction time figures as the numerator; the EEG is sampled for a shorter interval when reaction times are shorter, a procedure which conceivably might lead to a spurious correlation. These data have such important implications that one would like to see them confirmed, possibly by measuring EEG frequency during a constant time interval just preceeding the stimulus.

Cortical Activation and Speed of Perception

Lindsley (1958, 1961) reports that pairs of brief light flashes produce discrete evoked responses in the visual cortex of the cat or monkey when the flashes are separated by 100 mSec, but only a single evoked response when the separation is shortened to 50 mSec; with similar light intensities, human observers can perceive both flashes at the 100 mSec interval but only one at the 50 mSec interval. During about 10 seconds following electrical stimulation of the animal's midbrain reticular system, the 50 mSec flash pair evoked two distinct responses in the visual projection area, suggesting that reticular activation had increased the temporal resolving power of the cortex and, presumably, that a similar effect in a human subject might allow him to preceive the 50 mSec pair as a double flash.

At least two investigators have since employed the two-flash treshold (TFT) as an indicant of individual differences in cortical activation in experiments with human subjects. Venables (1963a) studied the relationship

between TFT and performance on a card-sorting task with a sample of schizophrenics. Although there was no correlation with amount of learning during the first four trials (and, apparently, no relationship with average performance, although the report is unclear), the disruption in performance caused by changing the basis for scoring on the fifth trial correlated about +.60 with the TFT, which the author regards as evidence that selectivity of attention--freedom from distraction by the now irrelevant sort criteria--increases with greater cortical arousal. Rose (1964) found correlations between two-flash thresholds and a measure of palmar skin conductance of -.53, -.70, -.72 and -.76 in four different samples and a correlation of -.47 in a single patient tested on 16 occasions over a period of three months. When 38 normal subjects were retested after several days, but at the same time of day, the test-retest correlation for the TFT was +.81, indicating favorable psychometric properties for this variable. Rose found that the TFT was significantly higher in a sample of psychiatric patients receiving some form of tranquilizer than in samples of normals or drug-free patients. Interestingly, the correlation between the TFT and palmar conductance for the tranquilized patient group was only -.26 (not significant). Finally, in a sample of drug-free psychiatric patients, the TFT was found to correlate -.63 with the scale of anxiety proneness described by Lykken (1957). Clinicians judged the MMPIs of patients showing low thresholds to indicate high anxiety, agitation or depression while patients with high thresholds were judged to be less anxious, more impulsive, more hysteroid or psychopathic.

It may be noted in passing that this finding of lower two-flash thresholds in drug-free (than in tranquilized patients) and in anxious patients, while

consistent with the idea that lower thresholds result from increased reticulocortical activation, appears to conflict with earlier reports that critical flicker fusion frequencies (CFF) are lower in anxious subjects (Krugman, 1947; Goldstone, 1955). As Lindsley (1961) has pointed out, visual flicker provides in its rhythmic character a kind of information to the brain not available from a single pair of flashes and which may be responsible for the fact that continuous trains of flashes can be perceived as flicker at inter-flash intervals far below those necessary for the resolution of a single pair. The matter is still unclear, but there does appear to be sufficient basis for assuming that the CFF implicates perceptual mechanisms not involved in the TFT. King's (1962) finding of zero-order correlations between TFT and CFF measured in samples of normal and schizophrenic subjects further indicates that these two variables should properly be treated as reflecting different, though related, parameters of brain function. (One would like to think that measuring CFF with irregular inter-flash intervals, by eliminating rhythmic information, would lower fusion frequency to a value nearer that found with two flashes. Lindsley (1961) found that, e.g., alternating 35 mSec and 65 mSec intervals gives flicker at light intensities for which a steady 50 mSec interval is seen as fused, i.e., the more complex pattern raises rather than lowere CFF. However, the 35-65 alteration does still constitute a pattern and the prediction that random inter-flash intervals--varying, say, from 35 to 65 mSec--should lower the CFF remains to be tested.)

The oft quoted experiment by Fuster (1958), more recently extended by Fuster and Uyeda (1962), was concerned with the effect of electrically stimulating the midbrain reticular formation on performance in a previously

learned tachistoscopic discrimination problem. Monkeys were trained to find food in a compartment under one of two stimulus figures. Then a series of trials were run in which the stimuli were flashed for durations of from 10 mSec, where performance fell to chance levels, up to 40 mSec, where performance averaged about 85 percent correct. On half of these trials, mild reticular stimulation was given, beginning two seconds before the flash and continuing until the animal had made its choice. Both numbers of errors and mean reaction time were reduced under reticular stimulation at all exposure times. Stimulation higher in the reticular system, in the midline nuclei of the thalamus, may also facilitate learning of a similar two-choice problem. Mahot (1964) found such facilitation when her monkeys were stimulated while looking at the test stimuli. Stimulation just before presentation of the stimuli had no effect while stimulation during the ten seconds right after a response disrupted learning.

Activation and Sedation Tolerance

It is well established that the reticular system is particularly sensitive to barbituarte anesthesia (French, Verzeano and Magoun, 1953; Gellhorn, 1953; Killam and Killam, 1957). Therefore, the amount of barbiturate, per unit of body weight, necessary to produce sedation should provide an index of the level of pre-existing reticular activation. In an extensive series of investigations (summarized in Shagass, 1962), Shagass and his co-workers have developed a measure of sedative susceptibility using paced, cumulative intravenous doses of barbiturate and a rather complex EEG criterion of the point at which "sedation" has occurred. Using this measure, Shagass has reported remarkable discriminations between various clinical groups; thus, patients with neurotic depression have much higher sedation

tolerance than patients with psychotic depression (Shagass, Naiman and Mihalik, 1956) and sedation tolerance is strongly associated with degree of manifest anxiety (Shagass and Naiman, 1956). Other investigators have reported considerable difficulty in using Shagass' EEG criterion for sedation, however, and the procedure developed by Rose (1964) is both simpler to use and more reliable.

In Rose's method, a brief electric shock is presented every 30 seconds concurrently with the administration of one cc. of thiopental, diluted to 0.25 mg/kgm body weight and injected via an indwelling I-V apparatus without disturbance to the subject. The sedation criterion is based on the decreasing amplitude of the palmar GSR elicited by the shock; viz., sedation tolerance is defined as the amount of thiopental required to reduce the shock-GSR to 10 percent of its average value during the three shocks given before starting the drug. Using this method, Rose tested a small group of ten male interns on two occasions, finding a retest rank correlation of +.96. Since the sedation tolerance test presumably measures the increment in arousal produced by the subject's reaction to the test conditions added to the basal level characteristic of him, i.e., both state and trait in Cattell's and Scheier's (1961) sense, such remarkably high stability suggests that these interns adapted more readily to the injection situation than would be expected of most subjects. In another study, using the less reliable "sleep threshold" criterion, Rose tested ten male chronic schizophrenics on three occasions, finding a retest stability of +.57 for tests I vs. II and +.86 for tests II vs. III; corresponding stability coeficients for palmer conductance measured before each test were +.38 and +.83. If we can assume from the stability found between test II and test III that these patients had

adapted to the circumstances of the testing situation by the end of the first session, so that tests II and III represent real trait values which are therefore more stable, then it appears that there was considerable variability in their reaction to the unfamiliar and somewhat threatening circumstances of the first testing which was not correlated with their basal or trait values, e.g., some normally low-activation subjects showed high state arousal on the first test while other high-activation patients reacted relatively less. This finding, although it clearly should be extended under other conditions, suggests an important methodological caution. Wherever possible, when one is interested in assessing some trait which one expects to be reasonably stable over time, one should carefully consider the possibility that the testing procedure itself may induce considerable change in the variable being used to estimate that trait. When, as in this instance, it appears reasonable to expect that even subjects all having the same trait value might produce considerable score variability due to variations in their reaction to the unfamiliar test conditions, then one should attempt to repeat the testing on at least a second occasion, regarding the first testing merely as an adaptation trial.

The Arousal Reaction and the Orienting Reflex

In their now classical paper, Sharpless and Jasper (1956) demonstrated that "repetition of a specific tone, which initially produces long-lasting arousal of a sleeping cat, fails to do so after 20 or 30 trials." This habituation of the arousal reaction was found to be specific to the quality, modality or pattern of the stimulus used. Thus, after habituation to a 500 cycle tone, a tone of 100 cycles still produced EEG activation and behavioral arousal. Arousal caused by a particular sequence or pattern of

tones could be seletively habituated in some animals but this was no longer possible after destruction of the auditory cortex; cortical damage did not, however, affect the efficiency or specificity of habituation to a particular tonal pitch. Specific evoked potentials recorded from the intact cortex are, if anything, larger after habituation of the arousal reaction, indicating that the effect cannot involve interference in the specific afferent pathways.

It was possible by means of surgical procedures to show that the arousal reaction consists of two components. One, mediated by the non-specific nuclei of the thalamus and the DTPS, responds rapidly to relatively weak stimulation and is exceedingly resistant to habituation, recovering rapidly with rest. The other, mediated by the ARAS, responds relatively slowly, requires stronger or repeated stimulation, and habituates quite rapidly. DTPS activation can be described as phasic, producing an EEG desynchronization which begins rapidly but does not outlast the eliciting stimulus for more than a few seconds. ARAS activation, more sluggish, endures longer so that the animal may remain aroused for many minutes after the stimulus has been removed. In the intact animal, EEG activation is observed nearly simultaneously in the cortex, thalamus and midbrain and is initiated by signals transmitted directly to the RF over the collateral afferents without the participation of the specific sensory systems.

This <u>arousal</u> of Sharpless and Jasper seems to parallel in many respects the <u>orienting reflex</u> (OR) described by Pavlov and elaborated in the work of the contemporary Russion neuropsychologist, E. N. Sokolov (1960, 1963). The OR is thought of as a central-nervous-system event, signalled by such observable phenomena as behavioral arrest, "propriomuscular" changes (Berlyne, 1960, p. 84) producing orientation and increased sensitivity of the receptors,

increased muscle tonus, EEG activation, and by a characteristic pattern of autonomic changes which includes the GSR and a combination of vasoconstriction in the fingers with vasodilation in the blood vessels of the head. The OR may be initiated by an increase, decrease or qualitiative change in a stimulus and is subject to habituation on repeated presentation of the stimulus. The functional significance of the OR is thought to be that it produces an increase in the "discriminatory power of the analyzers," lowering sensory thresholds, increasing resolving power, and the like.

Figure 5

Just as Sharpless and Jasper found with sleeping cats, Sokolov finds in his work with waking human subjects that the habituation of the OR to a particular tone is frequency specific (see Figure 5). Should a stimulus occur at regular intervals until the OR habituates, then the withholding of that stimulus will elicit an OR. (This recalls the story of the man, living near the railroad tracks, who had so adapted to the roar of the Midnight Express, that, on a night when the train was unaccountably late, he awoke with a start in the midnight stillness and asked, "What was that?") Such findings lead Sokolov to conclude that repeated experience with a stimulus leads to the formation within the nervous system of a neuronal model, embodying the salient features of that stimulus against which the signal resulting from each new stimulation is compared. The orienting reflex then is a function of the discrepancy obtaining between the current signal and this model (see Figure 6). Making use of the fact that neural transmission is faster in the specific afferent pathways to the cortex than

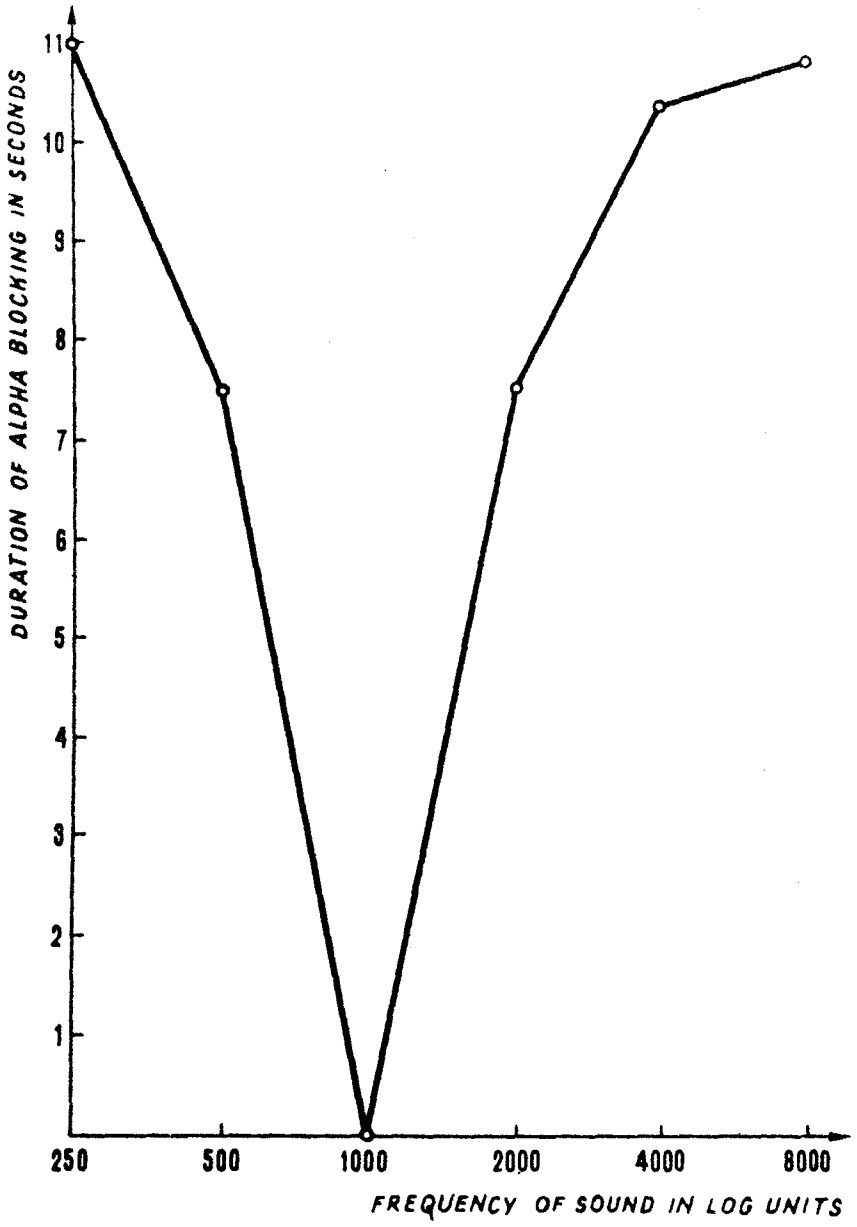

Figure 5. Amplitude of orienting reflex (duration of alpha blocking) following presentation of tone stimuli at various pitches above and below the pitch (1000 cps) to which the subject had become accustomed. The OR seems to vary as a function of the degree of mis-match between the characteristics of the stimulus and the characteristics of the neuronal model (i.e. "expectation"?) built up in the subject by prior habituation to a particular stimulus. (From Sokolov, 1960.)

it is in the nonspecific reticular afferent system, Sokolov proposes that

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Figure 6

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signals found to be concordant with the neuronal model in the cortex intiate inhibitory cortico-reticular impulses which tend to block transmission of that signal through the collateral fibers to the RF.

That the cortex is capable of powerful and selective inhibition of RF activities has been shown by the work of Hugelin and Bonvallet (quoted in Berlyne, 1960). In the decorticate animal, RF stimulation tonically facilitates spinal motor reflex activity; with the cortex intact, this initial increase in reflex strength dissipates within a few seconds, even though the RF is continuously stimulated. This indicates that, at least in its motor function, the RF receives selective inhibitory regulation--negative feedback--from the cortex. Significantly, Hugelin and Bonvallet found no reflex facilitation at all when the stimulation to the RF was increased gradually from zero which seems to show that cortical inhibition can keep pace with slow changes in RF activation, neutralizing them before they can have any effect. More speculatively, Sokolov assumes that <u>any</u> RF activation, such as might be initiated by a novel stimulus as part of an OR, immediately elicits inhibitory feedback from the waking cortex so that the recovery phase of an OR is not merely the result of the withdrawal of excitation but an active corticifugal inhibitory process. Moreover, he assumes that this inhibitory response can be conditioned,so that, e.g., it begins earlier with each repetition of the stimulus.

As evidence that habituation of the OR is a cortical process, that the "neuronal model" resides in the cortex, Sokolov describes two experiments

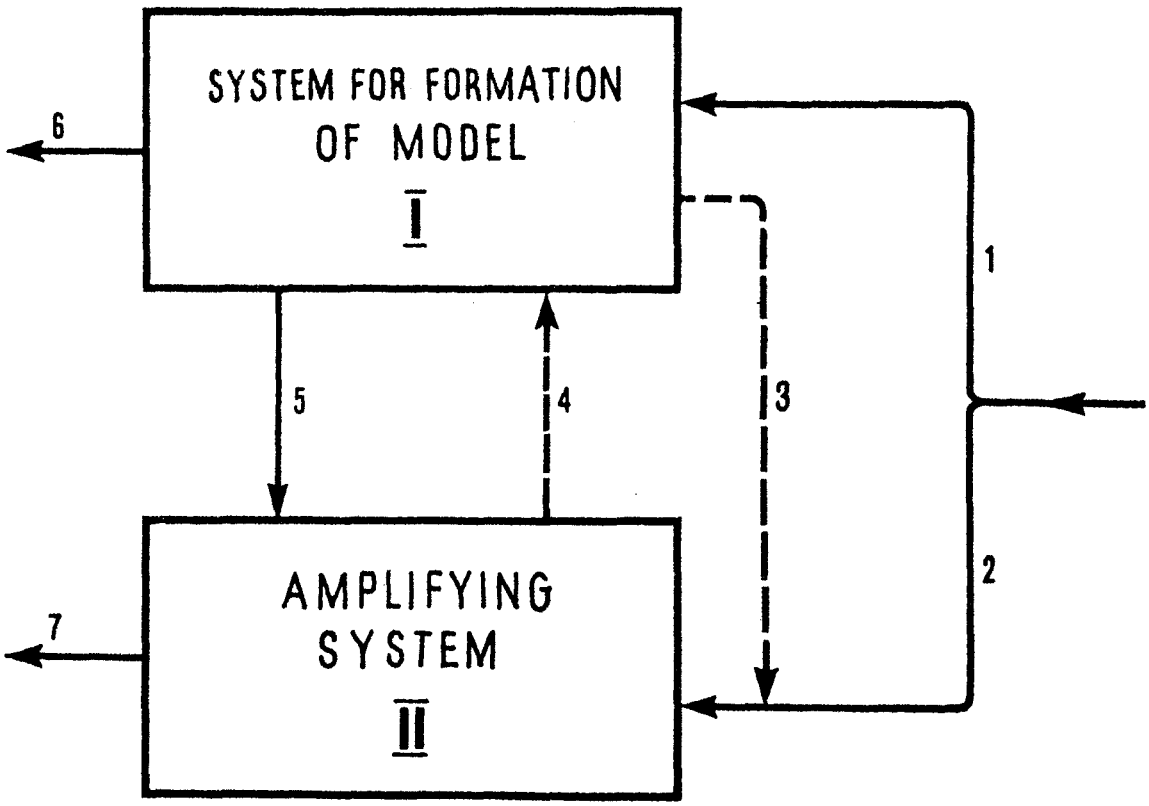

Figure 6. Sokolov's "neuronal model" for the orienting reflex. I. Modeling system. II. Amplifying system. 1 = specific pathway from sense organs to cortical level of modeling system; 2 = collateral to reticular formation (represented here as an amplifying device); 3 = negative feedback from modeling system to synaptic connection between collaterals from specific pathway and R.F.; 4 = ascending activating influences from the amplifier (R.F.) upon modeling system (cortex); 5 = pathway from modeling system to amplifying system (this is the pathway through which the impulses signifying concordance are transmitted from the modeling system to the amplifying system); 6 = to the specific responses caused by coincidence between the external stimulation and the neuronal model elaborated in the cortex; and 7 = to the vegetative and somatic components arising from the stimulation of the amplifying system (R.F.). (From Sokolov, 1960.)

in which an OR was readily habituated in a waking subject only to return as the subject became drowsy; moreover, it was found to be impossible to rehabituate the OR while in this drowsy state, because, Sokolov argues, cortical function is lost in this state and with it the specific cortical inhibition upon which OR habituation depends.

But this finding is paradoxical in the light of the Sharpless and Jasper results which clearly show that arousal--reticular activation--produced by a particular stimulus can be selectively habituated during actual sleep. The key to this difficulty may be contained in the results reported by Huttenlocker (1960) concerning specific evoked responses recorded from the midbrain RF during repeated auditory click stimulation in both the waking and sleeping conditions. It was found that these responses continue undimished in the RF long after the OR has habituated when the animal is awake but that they are rather quickly supressed during sleep. This would seem to indicate that OR habituation does not result from disruption of transmission through the afferent collaterals as Sokolov has supposed but rather from a change in the responsiveness of the RF to the signal. Huttenlocker's data suggest that habituation of the arousal reaction during sleep involves an entirely different mechanism than does habituation of the waking OR. With specific cortico-reticular inhibition lost, the sleeping animal apparently resorts to filtering or blocking afferent input to the RF, probably in the afferent collaterals rather than more peripherally since the cortical evoked potentials are undiminished. During drowsiness, as the subject fluctuates between sleep and waking, perhaps neither mechanism can establish full control so that habituation is difficult in this transition phase. Dumont and Dell (1960) provide further evidence that

this phase is "special," showing that the effect of reticular stimulation upon cortical evoked potentials is very markedly enhanced during the crossing-over from sleep into wakefulness.

It is possible that OR habituation will reflect itself also in habituation of the arousal reflex provided that sufficient presentations of the stimulus are experienced while the subject is awake. There seems to be reason for supposing that considerable overlearning is required for selective performance during sleep. We may distinguish between stimuli which can be responded to automatically while awake and those which require that one focus one's attention upon them; practice continued long after we first "learn" to identify a new stimulus pattern or to organize some new response can often render these reactions automatic and capable of being run off without conscious awareness. Perhaps it is at this point that a function becomes capable of operating under sleep conditions. Could subjects who responded to their own names during sleep (in the study by Oswald, et al, 1960, cited above) have performed as well had their instructions been to react only to "dit-dit-dah," from a list of letters sounded in Morse Code? One suspects not and yet it is likely that experienced telegraphers could do so.

Functions of the Orienting Reflex. The orienting reflex, as instanced by finger vasoconstriction coupled with vasodilation in the hand, is to be distinguished from a specific adaptive reflex, exemplified by a vasodilation in both loci upon presentation of a warm stimulus, and also from a defensive reflex, which might be a vasoconstriction in both loci in response to an electric shock. Typically, a new stimulus first elicits the OR. Thus, before starting a tone-shock conditioning

series, the CS and US were first presented singly until the OR to the tone habituated (after 17 presentations) and the OR to the shock gave way to a defensive reflex (after 47 presentations). When now the CS and US were combined, the first effect was a return of the OR and it required 35 CS-US pairings before a conditional defensive reflex was elaborated, as indicated by vasoconstriction in both head and hand to the presentation of the CS alone. Thus, "stabilization of a conditional reflex is connected with the habituation of the orienting reflex and all changes in the conditional reflex are connected with an increase in activation of the orienting reflex." (Sokolov, 1960, p. 223).

In another experiment, the CS was a 500 cycle tone and the vasomotor reactions to other tone frequencies were sampled after increasing numbers of reinforements with shock. During the earlier stages, frequencies from 300 to 900 cycles all evoked the defensive reflex (vasoconstriction, both hand and head) while frequencies outside this range produced ORs (dilation in the head). After many more reinforcements, the defensive reflex was obtained only in the range from 490 to 510 cycles and the OR from about 450 to 490 cycles and from about 510 to 550 cycles with negligible response of any kind outside these limits. Thus, as discrimination improves, the OR serves to demarcate the zones of stimulus uncertainly, a result which, incidentally, Sokolov's neuronal model theory seems unable to encompass.

However mediated, the OR seems to correspond in amplitude to some joint function of the intensity and the predictability of the eliciting stimulus. This is very nicely illustrated in an experiment by Kimmel (1960) in which five groups of subjects were exposed to 20 presentations of a 1000 cycle tone at a constant intensity of 35, 55, 75, 95 or 115 db.

(Twenty shocks were also given, either paired or unpaired with the tones, for reasons unrelated to the present point.) Following these 20 experiences of the tone at some given intensity, each subject then received a single test trial in which the tone was sounded at either the same or a different intensity and his GSR to that test tone was measured. The results were analyzed in terms of the disparity between the loudness of the test tone and the loudness to which that subject had been exposed on the previous trials and presumably had come to expect. The findings are

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Figure 7

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illustrated in Figure 7. The GSR, which we can interpret as an indicant of the orienting reflex, was minimum for small changes in the stimulus and larger when the test tone was either louder or softer than expected; however, a given increase in loudness produced a greater GSR increment than a decrease of the same size. Discussing this together with other similar studies from his laboratory, Grings (1960) shows that the size of such "perceptual disparity responses" increases with the number of prior exposures to the standard stimulus (e.g., with the strength and specificity of the subject's expectation concerning the nature of the stimulus on the test trial).

The ability of the orienting reflex to "increase the discriminatory power of the analyzers" is illustrated by Sokolov's (1960) report that the presentation of a light intensity 0.8 db below threshold failed to produce a depression of the alpha rhythm until the light was preceeded by a sound stimulus which evoked an OR. Similarly, Lansing, Schwartz and Lindsley (1959), studying reaction times in human subjects, found that where the average

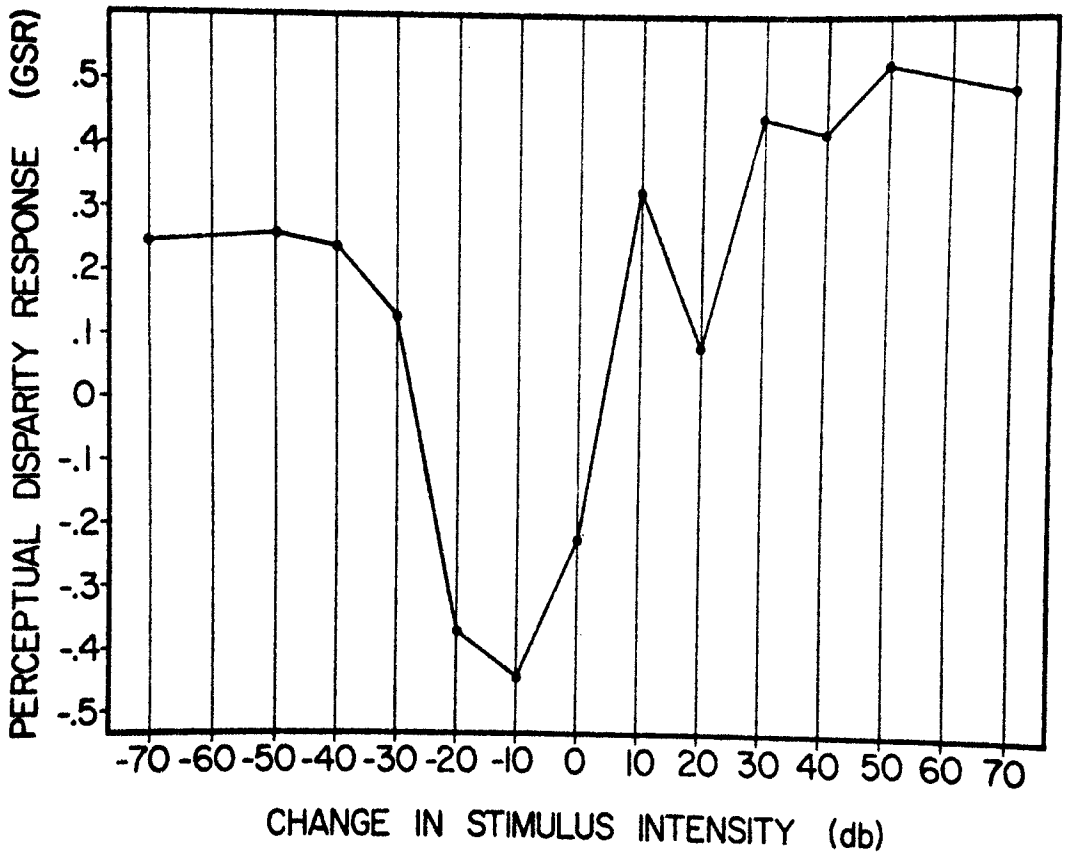

Figure 7. Amount of "perceptual disparity response" (GSR) as a function of the amount of change in the intensity (loudness) of the stimulus from the value to which the subject had become accustomed. (From Kimmel, 1960.)

reaction time without a forewarning signal averaged 280 mSec, warning intervals long enough to permit blockade of the alpha rhythms before the reaction stimulus occurred (e.g., from 300 to 100 mSec) yielded a decrease to 206 mSec, apparently by reducing central processing time. It is not clear, however, that the OR produces these effects merely by eliciting a phasic, transitory activation of the cortex. Research on the two-flash threshold, discussed earlier, indicated that the temporal resolving power of the visual cortex can be phasically enhanced by electrical stimulation of the midbrain RF and that it is tonically elevated in subjects who are in a state of high arousal. A recent study by Horn and Venables (1964) suggests that a "warning" stimulus presented 300 to 600 mSec before the paired flashes will also lower the TFT, presumably by increasing cortical arousal, i.e., by evoking an orienting reflex. Stimuli occurring less than 200 mSec before the first of the paired flashes markedly elevate the TFT; the interval here is too short for activation to develop. The fact that such short intervals raise the TFT rather than simply not affecting it may have to do with the distraction of attention (see the discussion of the single-channel theory of attention below).

If the effect of the OR upon perception were due only to its nonspecific activiting of the cortex, then a subject with moderate arousal should perform as well without warning signals as a low arousal subject does with them and the performance of a subject already aroused should actually be better when no warnings are provided. As its name implies, the orienting reflex undoubtedly provides something more than nonspecific arousal, information which allows the organism to orient

or tune its perceptual apparatus so as to expedite the handling of a particular signal at a particular time.

Arousal, Energy Mobilization and Drive

The close relationship between reticular activation and the psychological concept of drive has been marked by a number of writers (e.g., Hebb, 1955; Lindsley, 1957; Duffy, 1957; Malmo, 1962; Berlyne, 1960). As Berlyne (p. 166) points out, "excess of carbon dioxide, hunger, and sexual deprivation modify the chemistry of the blood in ways that sensitize the reticular formation [and] the responsiveness of the reticular formation to adrenaline and noradrenaline, hormones whose secretion has much to do with fear and anger, has been clearly demonstrated". Reticular activation, like increased drive, may reduce response latencies, facilitate reflex activity and produce restlessness and agitation. The inverted U-shaped relationship known as the Yerkes-Dodson Law, between efficiency of performance and drive level, also appears to hold true when activation is plotted on the abscissa in place of drive.

The most detailed analysis thus far of this coordination of concepts is to be found in the highly recommended monograph by Berlyne (1960). Berlyne first analyzes the drive notion into three components: $drive_1$, representing the energizing aspect as in Hull's D; $drive_2$, representing the directional or cue aspect as in Hull's SD; and $drive_3$, representing the "condition whose termination or alleviation is rewarding." He then defines arousal tonus as the minimum level of RF activation of which the individual is capable at a particular time. "The location of the tonus level will depend on the pattern of cortico-reticular interaction, with, no doubt, other subcortical structures playing their part also.

This intereaction will, in its turn, depend on internal factors, such as how often the environment has been issuing calls for urgent action". (p. 193). Having identified drive$_1$ with arousal level, drive$_3$ is related to any increase in arousal above the prevailing level of arousal tonus; in these circumstances, a decrease in arousal back toward the tonus level is assumed to be rewarding.

We know that most of the classical methods for increasing drive also increase reticular activity (at least under certain conditions of measurement; see below) and we have seen that satiation of such needs as hunger and sex is followed by a decrease in cortical and RF arousal. The problem which Berlyne's or any drive-reduction theory of reinforcement has to face is that of dealing with behavior which appears to arise out of a state of low arousal, and behavior which is learned and maintained even though it seems to result in an actual increase in drive or arousal. In the case of boredom, the consequence of minimal or unvarying stimulation which ought to lead to decreased arousal and even sleep, Berlyne suggests that monotonous stimulation disorganizes the cortex thus presumably disrupting both its excitatory and its inhibitory cortico-reticular influences. The initial result in a fatigued individual may indeed be sleep but this must eventually be followed by awakening and increasing restlessness and agitation. Deprived of its usual cortical restraints, the RF becomes more and more aroused, stimulus-seeking behavior is initiated and the attainment of novel or excitatory stimulation is experienced as rewarding because, by activiting the cortex and re-establishing cortico-reticular inhibitory control, such stimulation results in an actual decrease in arousal. Outside the context of monotony and boredom,

the "aperitif phenomenon," the seeking out of stimuli whose immediate effect is an undeniable increase in drive or arousal--eating salted nuts, precoital sex play, riding the roller-coaster, i.e., what Hebb (1955) refers to as "the positive attraction of risk-taking and of problem solving"--is handled by assuming that the eventual result of such behavior is an even larger decrease in arousal.

Laying out so starkly the bare bones of Berlyne's position does a serious injustice to the ingenuity of his argument and the wealth of evidence which he has brought together in provocative array. Still, this over-simplification does highlight certain genuine difficulties. For example, one suspects that Berlyne sought out a secluded (i.e., unstimulating, monotonous) environment in which to do his writing but was not bored therein (nor did he, obviously, go to sleep). The extent to which the cortex can maintain its functional integrity independent of exteroceptive stimulation varies widely from person to person and from time to time and one would like to know why. Berlyne's hypothesis, that boredom is a state of agitation resulting from a failure of cortical control, is plausible and suggestive but needs further elaboration. More seriously, the maneuver of assuming that drive increase is not punishing although drive decrease is always rewarding, which Berlyne employs in the attempt to save the drive reduction theory from drowning in the flood of his own collection of evidence, seems like supererogation. Hebb's (1955) notion that some intermediate level of arousal is hedonically optimum and that increases below that level are rewarded and those above that level punishing appears to be both simpler and more reasonable on current evidence. Indeed, retaining the identification

of activation with drive$_1$ or D, it really seems most probable that reinforement is only contingently and incidentally related to drive reduction. The reinforcement associated with the presentation of an appropriate stimulus seems to increase monotonically with the level of arousal. With aversive drives, such as pain or fear, the "appropriate" stimulus will probably produce a decrease in arousal but this clearly need not follow when the arousal has a nonaversive basis. In the case of sexual excitement, although objective measurements seem to be lacking, appropriate stimuli do appear to be more rewarding as arousal increases and levels of activation can be attained which seem as high or higher than those involved in common aversive experiences.

Berlyne and others have pointed out that autonomic and EEG indicators of arousal tend to increase during sleep deprivation (e.g., Malmo and Surwillo, 1960) and have found in this support for the identification of arousal and drive$_1$ or D. Although the need to sleep can be real enough, one might feel uncomfortable about thinking of the sleep drive in the same conceptual category as, say, the hunger or the sex drive. More importantly, these findings of increased activation during sleep deprivation are typically obtained only when the subject is tested during the periodic performance of some task. Since many aspects of task performance do not deteriorate even with fairly extended deprivation (e.g., Williams, Lubin and Goodnow, 1959; Wilkinson, 1961), it is possible that the subject expends more energy, producing higher concurrent levels of arousal, in order to maintain his performance as the vigil progresses and that continuous monitoring would show that his intercurrent levels of arousal actually do decrease with deprivation. Alternatively, Berlyne

might expect that arousal would consistently increase, due to decreased cortical inhibition, given only sufficiently strong and varied stimulation to keep the subject awake but without requiring any organized response from him. The reaearch necessary to resolve this question does not appear to have been done.

The only other direct test of the arousal-drive$_1$ linkage that seems to be available is the study by Belanger and Feldman (1962) in which it was found that heart rate in the rat increases monotonically with from 24 to 72 hours of water deprivation. Again, however, the relationship held true only for heart rate measured during the time when the animals were actually working for and receiving water reinforcement. The more thirsty animals did not show elevated heart rates while resting in their cages nor even when placed in sight of the lever and water magazine but separated from it by a transparent screen. Granted that heart rate is one of the more dubious indicants of activation, it seems unreasonable to argue from these results that arousal is identifiable with drive level which <u>is</u> normally expected to increase monotonically with deprivation.

Lykken and Meisch (1965) found that when brief electric shocks are presented at random intervals to rats busy pressing a lever for food reinforcement, the rate of pressing increases for some 30 to 60 seconds following each shock. Both pre- and post-shock rates decreased as the shocks were made stronger until pressing ceased entirely and the behavior was no longer under appetitive control. These results could mean that

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Figure 8

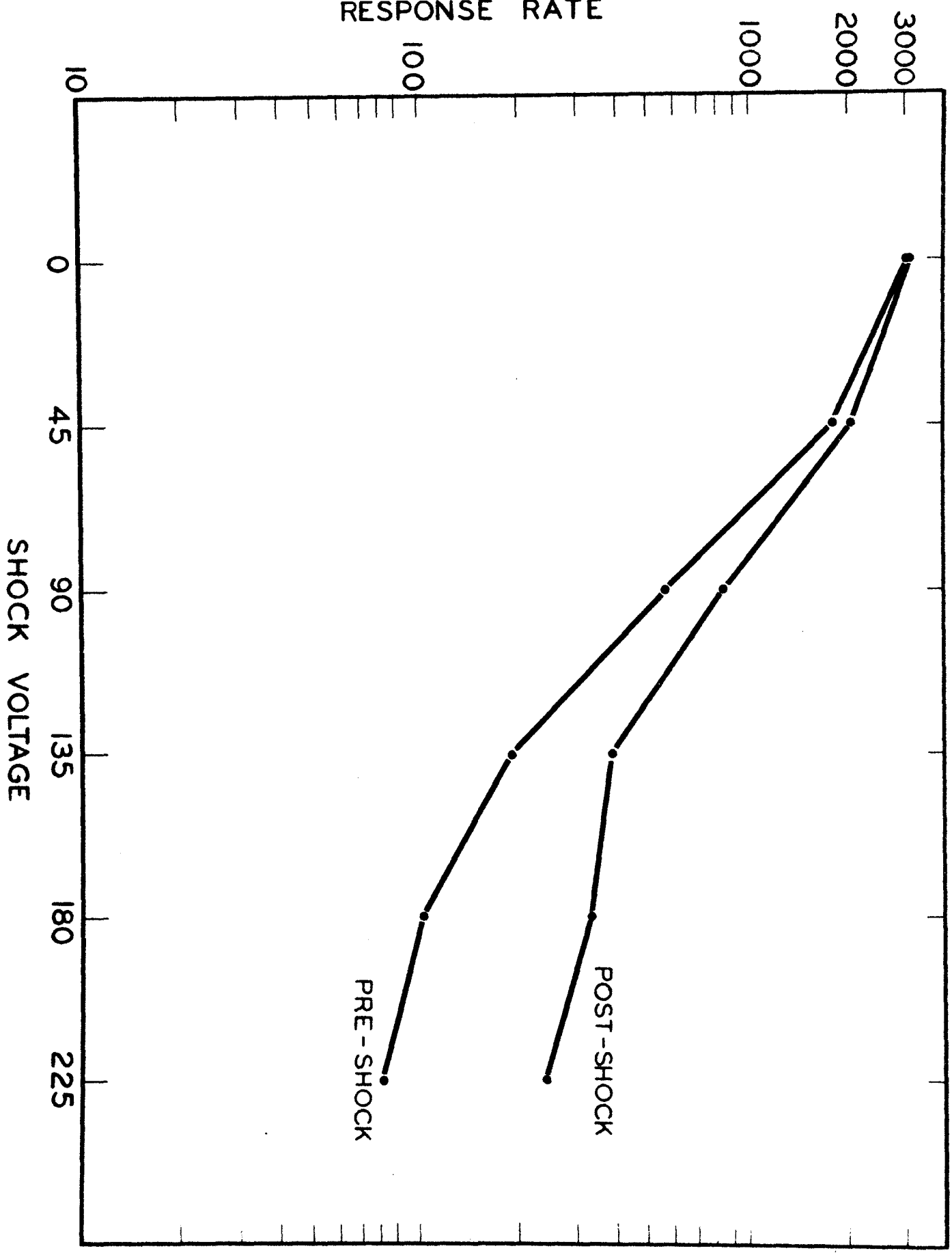

Figure 8. Logarithm of response rate (lever pressing) as a function of the intensity of aperiodic, non-contingent shocks. Each point is the mean of three one-hour sessions for four rats. Lower curve represents rate during the 30 second periods preceding shocks. Upper curve represents rate during 30 seconds following shocks and illustrates apparent activation of response rate produced by the presentation of the shock. (From Lykken & Meisch, 1965.)

the shock produced a transitory increase in nonspecific activation which, acting like D in Hull's formula for reaction potential, augmented whatever behavior was then dominant, e.g., lever pressing. But why does the overall rate decrease as the random shocks get stronger? A conventional explanation would be that the shocks produce a state having $drive_2$ or cue properties different from those of the hunger state and which tend to channel the combined $drive_1$ components into different activities which are incompatible with lever pressing.

CENTRAL MODULATION OF AFFERENT INPUT: PERCEPTION AND ATTENTION

The major sense organs, the specialized receptors of the skin, the interoceptors and proprioceptors, combine to inundate the central nervous system with a never-ending flood of raw sense data. How the brain can cope with this embarrassment of riches, selecting what it needs and ignoring what it doesn't, has been a perennial problem for students of perception. The discovery that the reticular system sends efferent fibers to all peripheral afferent relay stations as well as to the receptors themselves suggested that part of the answer might lie in a system of afferent filtering under central control. One example has already been described; the sleeping subject apparently can selectively inhibit transmission of specific sensory impulses across the collaterals linking the reticular formation to the afferent pathways, thus preventing arousal. The perceptual analysis required for this is accomplished subcortially, perhaps in the RF itself, in the case of simple stimuli; habituation to more complex stimuli requires some participation of the (sleeping) cortex although it may be that complex patterns can also be handled by lower structures when these patterns have been sufficiently overlearned.

When the subject is awake, afferent modulation might serve to (a) help determine which signals are admitted to or retained in the focus of attention and (b) modify the affective quale of the signal by, e.g., attenuating noxious sensations or amplifying pleasurable ones. It appears that such "perceptual tuning" may be either anticipatory, evoked by some sort of warning signal, or reactive, being elicited by the onset of the stimulus itself. As to specificity, evidence of varying quality suggests that the afferent system can selectively amplify (or attenuate) a specific signal, all signals in a given modality, or the entire flux of sensory input.

Neurophysiological Background

Evoked Potentials.

The afferent signal can be followed on its way to the brain as a series of evoked potentials recorded from electrodes placed near the nerve tract or one of its relay nuclei. The evoked potential represents a composite of the many individual neuron action potentials. At each relay, the postsynaptic potential may be smaller, if transmission across the synapse has been inhibited in some way, or larger, if for example the postsynaptic neurons have been subjected to prior subthreshold excitation. In the sensory receiving areas of the cortex, the evoked potential is a much more complicated waveform; it includes a presynaptic component, representing the arrival of the impulse through the ascending axons; a postsynaptic component, representing the reaction of the intracortical neurons to this impulse; and normally, an after-discharge, which consists of a complex and variable series of waves related to persisting local activities and to reverberating impulses traveling back and forth between

the cortex and distant structures, particularly in the thalamus.

Intracranial recording is not always feasible with human subjects (!) so it is fortunate that the cortical evoked potential is also present in the EEG activity recorded from properly positioned electrodes on the scalp. In the raw EEG recording, these evoked potentials are typically hidden amidst the fluctuations of the spontaneous brain rhythms; during recent years, however, techniques have been developed for eliminating this background "noise" by an averaging process. To illustrate the method, suppose one were to take 100 one-second segments at random from an EEG record and superimpose them so as to get a kind of average. At any point within this one-second interval, about half the segments making up the average will have been in the positive half of a cycle and the remainder in the negative half (since the segments were chosen randomly, there is no reason for them to be synchronized in any way). Therefore, the average or composite record will tend toward a straight line of zero voltage because these positive and negative components will tend to cancel out. Now suppose that these 100 segments had all been chosen to begin at the instant that a flash of light was presented to the subject. Then each EEG segment would include within it a weak trace of the cortical potential evoked by the light; this trace would have very nearly the same shape and the same latency in each segment. When the 100 segments now are averaged, while the noisy EEG rhythms cancel out, the evoked potentials will summate, standing out more and more clearly the more segments are included in the average.

This averaging process is now done automatically by means of a specialized computer; the EEG is recorded on magnetic tape which is later played back into the computer which in turn draws out the averaged

wave on an X-Y recorder. Averaged cortical evoked responses have been shown to correspond quite well with what would have been obtained recording directly from the cortex (Domino, Matsuoka, Waltz and Cooper, 1964). Examples of evoked responses to auditory click stimuli in a waking subject and at various levels of sleep are illustrated in Figure 3.

Significance of the Cortical Evoked Response. Geisler, Frish, Kopf and Rosenblith (1958) reported that averaged cortical responses to auditory clicks first appear as the stimulus rises above the auditory threshold. Shagass and Schwartz (1961) obtained similar results with somatic and visual stimuli. A careful parametric study by Wicke, Donchin and Lindsley (1964) shows that it is the first of the two diphasic waveforms in the visual evoked response which decreases with decreasing luminance and disappears at or near the threshold. Moreover, these authors find that this component varies with the perceived brightness of the stimulus rather than with its physical intensity (luminance). Haider, Spong and Lindsley (1964), studying averaged cortical responses to dim signal light flashes in a protracted vigilence experiment, found that the average response to signals detected by their subjects was substantially smaller than that evoked by signals which were not detected. However, it is well known that the cortical response is actually enhanced during sleep and under anesthesia; hence, the presence of the response cannot guarantee that the subject will be aware of the stimulus or have it influence his behavior. Davis (1964) reports that when a subject is required to make a difficult discrimination in, say, pitch between the second and third sequence of four tone bursts presented to him, the cortical response evoked by the third tone is sharply augmented although that tone neither

is nor does it sound any louder to the subject; hence, perceival intensity does not always vary with the amplitude of the cortical evoked response. Similarly, Chapman and Bragdon (1964) found that a meaningful stimulus evokes a larger response than does an interpolated stimulus which is not relevant to the task set for the subject. Electroretinographic responses (ERGs) produced by the two stimuli did not differ. Most recently, Spong, Haider and Lindsley (1965) report consistent changes in the relative amplitude of evoked responses from temporal and occipital areas, produced by alternating clicks and flashes, as the subjects were required to attend to the clicks while ignoring the flashes, and vice versa.

These findings are the first gleanings of a rich harvest of research in which traditional psychophysical measurement is being supplimented by electrocortical recording. Much remains to be learned about the significance of the several components of the complex cortical response; it is already plain, for example, that to speak of the "amplitude of the response" is an over-simplification since its components can vary in size independently of one another. It would appear that the initial disphasic wave does vary with perceived intensity of the stimulus in a situation where the subject is attending about equally to all stimuli in the series. Since giving special attention to a stimulus amplifies its cortical evoked response without increasing its perceived intensity, it would appear that the intensity property may be separately decoded, perhaps subcortically. Another reasonable speculation is that the amplitude of an evoked reponse, figured in relation to the activity evoked by concurrent competing stimuli, determines the probability that the evoking stimulus will be attended to at all.

Evoked potentials in the midbrain reticular formaiion can only be recorded from implanted electrodesand are correspondingly more difficult to study. We know that these responses are reduced or absent during pontile sleep and also when the waking subject's attention is distracted (e.g., Winter, 1964). Jouvet, Benoit and Courjon (1956) have found cortical regions at which stimulation inhibits evoked potentials in the ARAS without affecting activity in the specific sensory system. We have already seen that evoked responses in the ARAS diminish rapidly with repeated stimulation during sleep but not when the animal is awake (Huttenlocker, 1960) and therefore attributed habituation of the orienting reflex to a corticifugal mechanism which inhibits reticular reactivity without blocking sensory transmission to the RF (Dell, Bonvallet and Hugelin, 1961).

It is tempting to suppose that one of these inhibitory mechanisms, that which reduces reticular reactivity to stimulation or that which attenuates sensory impluses transmitted to the RF, is employed by the cortex under special circumstances to deprive stimuli of the disturbing or excitatory effects they might normally have upon the ARAS. It is quite clear that the intensity of a stimulus and its arousing or excitatory quality, while noramlly correlated, are not the same thing and can sometimes vary independently. Even in the case of pain, for example, we know that individuals under hypnotic suggestion, morphine analgesia or after frontal lobotomy ofter show intact capacities for discriminating pain intensities and unaltered pain thresholds while at the same time experiencing little of what Kessen and Mandler (1961) have called the "distress" component of the usual pain response. This distress reaction

includes reticular activation which, in the absence of cortical influence, is probably proportional to the strength of the signal transmitted to the RF over the collateral afferents.

Tonic Reticular Afferent Inhibition.

It is now generally established that cortical potentials evoked by natural receptor stimulation tend to increase in amplitude as the gross activity of the midbrain and pontile RF decreases. Thus, cortical potentials are larger in deep than in light anesthesia (Derbyshire, et al, 1936), larger in sleep than during waking (e.g., Williams, et al, 1962) and diminished in the waking animal by electrical stimulation of the RF or by exciting the animal by naturalistic methods (e.g., Bremer, 1961).

Normally, reticular activation also depresses evoked potentials recorded earlier in the afferent pathway and it was at first believed that reticulofugal inhibition of the afferent relays as early as the first sensory synapse was responsible for these effects and, through them, for the depression of the cortical responses (e.g., Hernández-Peón, 1961, 1964). Later it was found that cutting the tympanic muscles of the middle ear or paralyzing them with curare prevented RF stimulation from reducing auditory evoked potentials in the dorsal cochlear nucleus, although the diminution of the cortical response persisted (Hugelin, Dumont and Paillas, 1960). In the visual system, RF stimulation actually increases peripheral evoked responses although at the same time decreasing the cortical response; the peripheral effect was shown to be due to the pupillary dilation which regularly accompanied cortical activation (Naquet, et al, (1960). Thus, it appears that the peripheral effects of nonspecific arousal may be limited to essentially mechanical changes in the receptor

and that these effects vary with modality and cannot be responsible for the consistent diminution observed in the cortical evoked response. The latter effect, however, seems enough to justify the claim (Hernández-Peón, 1961) that the RF tonically inhibits sensory transmission--or at least the registration of sensory input at the cortex--and that this generalized inhibition is roughly proportional to the level of arousal.

Attention and Distraction.

In a series of studies summarized in Hernández-Peón (1964), using freely moving cats with electrodes implanted in various afferent pathways, it was observed that evoked potentials elicited by stimuli in one modality are immediately reduced or blocked when the animal's attention is distracted by a stimulus presented to some other sense modality. Thus, cochlear potentials evoked by auditory clicks disappear when a container of mice is placed before the animal or when a delightfully fishy odor is wafted into his cage. The same sort of inhibition produced by the presentation of a "more significant" stimulus in some other modality has been demonstrated in the optic tract and for tactile and pain stimuli. Conversely, associating the click or flash with a "significant" stimulus like food or pain yields an enhancement of the respective evoked potentials. In human subjects, stimulated by flashes of light, evoked potentials coursing up the optic radiations toward the visual cortex were reduced or abolished when the subject's attention was diverted by, e.g., pricking him with a pin, and intensified when he was asked to count the flashes (Hernández-Peón and Donoso, 1959; Jouvet, 1957). Jouvet and Lapras (1959) reported that potentials evoked in the thalamus by tactile stimulation of

the face were diminished by distraction of attention.

Using scalp recording and averaging techniques, Garcia-Austt, Bogacz and Vanzulli (1964) as well as Spong, et al (1965) confirm that the amplitude of the cortical evoked response varies with the extent to which the subject's attention is focused upon the stimulus. The studies by Davis (1964) and by Chapman and Bragdon (1964), mentioned earlier, show that more meaningful or task-relevant stimuli, both auditory and visual, produce stronger cortical responses than do less meaningful stimuli of the same intensity. Using microelectrodes to record the activity of individual cells in the auditory cortex of unrestrained cats, Hubel, Henson, Rupert and Golambos (1959) reported finding some units which responded to sounds only when the animal appeared to be paying particular attention to the stimuli. Some of these "attention" units responded only during the first few presentations of a stimulus although other more typical auditory units responded faithfully to each sound even during sleep. One "attention" unit responded to the noise of paper rustled in the experimenter's hand if--but only if-- the cat also turned its head to look toward the source of the sound. As any cat-lover knows, the ears of these creatures--the external meatus--have an extraordinary capacity to orient, independently of one another, toward novel noises to the side or the rear. This easily monitored "orienting reflex" should be a delicate indicant of the momentary fluntuations of the animal's attention and an interesting correlate of electrocortical events.

Habituation of Evoked Responses.

We have already seen that habituation of the arousal reaction and the

orienting reflex can be accomplished after some 20 to 40 repetitions of the stimulus and that such habituation is not accompanied by any significant reduction in the amplitude of evoked potentials recorded from the specific afferent system. However, a series of studies by Hernández-Peón and others (reviewed in Hernández-Peón, 1964) seemed to indicate that these evoked potentials also can be habituated by continuing to present the stimulus repeatedly for perhaps hundreds of trials. This phenomenon, which has been called afferent neuronal inhibition (Hernández-Peón, 1961), was thought to depend upon the development of stimulus-specific negative feedback in the reticulofugal circuits to the sensory relays of the afferent pathway concerned. However, in a recent and careful attempt to replicate some of these observations, Worden and Marsh (1963) presented click stimuli (1/sec or 10/sec) continuously for six hours to cats both waking and asleep without finding any consistent changes in the responses recorded at the dorsal cochlear nucleus. Fernandez-Guardiola, et al (1961) did obtain consistent habituation of visual evoked responses; after about two hours of flash stimulation, all of their cats showed a diminution of evoked responses in the optic chiasm, the lateral geniculate body and in the visual cortex. However, cortical habituation preceeded changes in the periphery and their animals also displayed progressive relaxation, sleepiness and general deactivation accompanied by increasing constriction of the pupils; when the pupils were dilated and fixed by topical application of atropine, habituation was no longer observed in the peripheral pathway (although the cortical responses did still habituate to some extent). These authors believe that the rhythmic flashes have a kind of hypnotic effect, reducing reticular activation accompanied by pupillary constriction;

this in turn diminishes the peripheral afferent signal.

Thus, habituation of peripheral evoked responses is secondary to more central changes in the visual system and may not occur in the auditory system at all. Habituation of cortical evoked responses, however, seems to be a reliable finding (Bogacz, et al, 1962; Garcia-Austt, et al, 1964).

Conculsions.

There is a tonic attenuation of afferent input, effected somewhere above the first sensory relay, which is greater during reticular arousal and less when RF activation is decreased during relaxation, sleep or anesthesia. Both cortical and peripheral evoked responses are at once reduced or disappear when attention is diverted; at least the cortical component is phasically augmented when the evoking stimulus is at the focus of attention. Although these effects of attention and distraction may involve phasic changes in peripheral afferent transmission, it is clear that the role of peripheral modulation has been exaggerated. The alternative picture which seems to be emerging is that much of the "peripheral" afferent modulation may be accomplished in the cortex itself by the interplay of specialized units influenced from below by the structures of the nonspecific sensory system.

Related Psychological Research

Arousal, Fatigue and Tonic Afferent Inhibition.

That the reticular system maintains a tonic inhibition of afferent input implies that sensory thresholds as ordinarily measured should be, paradoxically, lower in a relaxed or even drowsy subject than in one who is more aroused. This surprising result has in fact been obtained in pilot

work by Rosenblith (quoted by Bremer, 1961, p. 46). In a series of studies reported nearly forty years ago, Spencer and Cohen (1928a, 1928b; Spencer, 1928) found very high correlations (.8 to .9 and higher) between the simple sensory threshold and the increase in that threshold measured in the presence of an additional, "inhibitory" stimulus. These findings, obtained both across a sample of 50 subjects (R-correlation) and also across 50 daily measurements on the same subject (P-correlation), were interpreted to support the theory advanced earlier by Heymans (1899) that the sensory threshold is normally greatly elevated above its physiological minimum by an active process of inhibition and that the inhibition is a function of the aggregate intensity of all ambient stimulation. The higher a subject's simple threshold, the higher his immediate capacity for inhibition and therefore the more will his threshold be further elevated by increasing ambient stimulus levels. In their study of the single subject over 50 days, Spencer and Cohen also correlated his simple (brightness) threshold against the amount of sleep the subject had on the preceeding night (all measurements were made first thing in the morning). This correlation was about +.60 over all 50 days and about +.80 over just the final 25 days when the threshold data were presumably most reliable. Correlations between thresholds and the subject's self-ratings of "freshness" were of about the same magnitude.

These findings are impressive and deserve to be extended. It is at first surprising that fatigue lowers sensory thresholds rather than raising them; on the other hand, most people would agree that they are more irritable, more distractable, more sensitive to pain and the like, at

the end of a tiring day than when they are well rested. Assuming that one's normal protection from the continuing noise, discomfort and distraction in the environment is provided by an active inhibitory process which necessarily would be effortful and energy consuming, it is reasonable that this protection should be diminished by fatigue. Although arousal probably tends to decline with fatigue, sleep deprivation research has shown that arousal can be at least phasically elevated to meet the demands of some task even under conditions of extreme fatigue. It may be that Spencer and Cohen's subject showed lower thresholds when more fatigued because he was also then less aroused, or it may be rather that thresholds vary with fatigue independently of arousal.

Another related phenomenon is that of audio analgesia in which high levels of auditory noise stimulation have been found capable of inhibiting the pain of dentistry and other surgical procedures. The relevant data, as reviewed by Licklider (1961), are quite interesting. Among other things, it appears that pain suppression is most effective if the noise is turned up before the pain begins, which may be related to another observation, that the method generally works less well with patients who are already tense or anxious. Some patients report that they do feel pain but that "it doesn't hurt." suggesting that the noise has suppressed the secondary elaboration or distress component of the primary pain. The extreme individual differences in response to this procedure are of special interest; one feels that there must be important temperamental differences between persons who experience full analgesia with only moderate noise levels and those who feel pain even with intense noise, or between those for whom white noise is most

effective and others who respond better to music. Reasoning from the Spencer and Cohen findings, one would expect that the effectiveness of audio analgesia should vary directly with the simple sensory threshold and inversely with fatigue.

A different but related phenomenon was observed in the course of a study of individual differences in fear conditioning in neurotic and "primary" psychopaths (Lykken, 1957). A number of subjects failed to show any appreciable conditioned GSR to a buzzer CS in spite of repeated pairings with a painful electric shock; most of these were "primary" psychopaths who showed abnormally little anxiety reactivity either clinically, in their prison and earlier records or in other psychometric findings. Four subjects with low conditioning scores, however, were clearly anxious individuals, with neurotic-looking histories and test scores, who seemed genuinely frightened of the shock and responded strongly to it but not to the associated CS. Absolute or tonic palmar skin resistance had been recorded at intervals during the 40 minute session and these data, reflecting changes in general level of arousal before, during and after the conditioning trials, proved to differentiate these four anxious, but strangely unreactive, subjects from all the other individuals tested. As shown in Figure 9, the four "primary" psychopaths with the lowest conditioning scores showed a gradually decreasing palmar resistance during the session; this was typical also of most of the other subjects tested. Three of the four anxious but unreactive "neurotic" psychopaths showed very elevated resistances with a unique tendency for the resistance to increase still further during part of the session. The fourth subject's resistance was not high overall but he was the only subject

tested whose resistance increased sharply after he experienced the first shock (given alone prior to the conditioning trials) and then remained higher throughout the stressful conditioning series. Apparently, then, these four individuals reacted to the stress of the conditioning trials with decreasing arousal and under these conditions, although they showed about normal GSRs to the shock, they gave remarkably little GSR to the CS which warned that the shock was to follow.

Figure 9

Selective Afferent Inhibition.

Lacey, et al, (1963) have reported that stimulus conditions which encourage or require the subject to attend to or "take in" the external environment will tend to elicit cardiac deceleration while noxious stimuli, or tasks which are facilitated by a focusing of attention inward, "shutting out" the environment, elicit cardiac accelerations. Thus, a mental arithmetic task, a reverse spelling task and another involving sentence production were expected to be intratensive stimuli for which a lowered reactivity of the sensorium would be adaptive; as predicted, all three tasks produced cardiac acceleration as did a painful cold pressor stimulus. Conversely, a task requiring detection of colors and patterns in visual flashes and another in which the subject was asked to "empathize" with the dramatic recorded soliloquy of a dying man, both extratensive in their intended effect, produced cardiac deceleration. Finally, two tasks which required careful attention both to external input and to internal cognitive processing gave intermediate results with relatively little

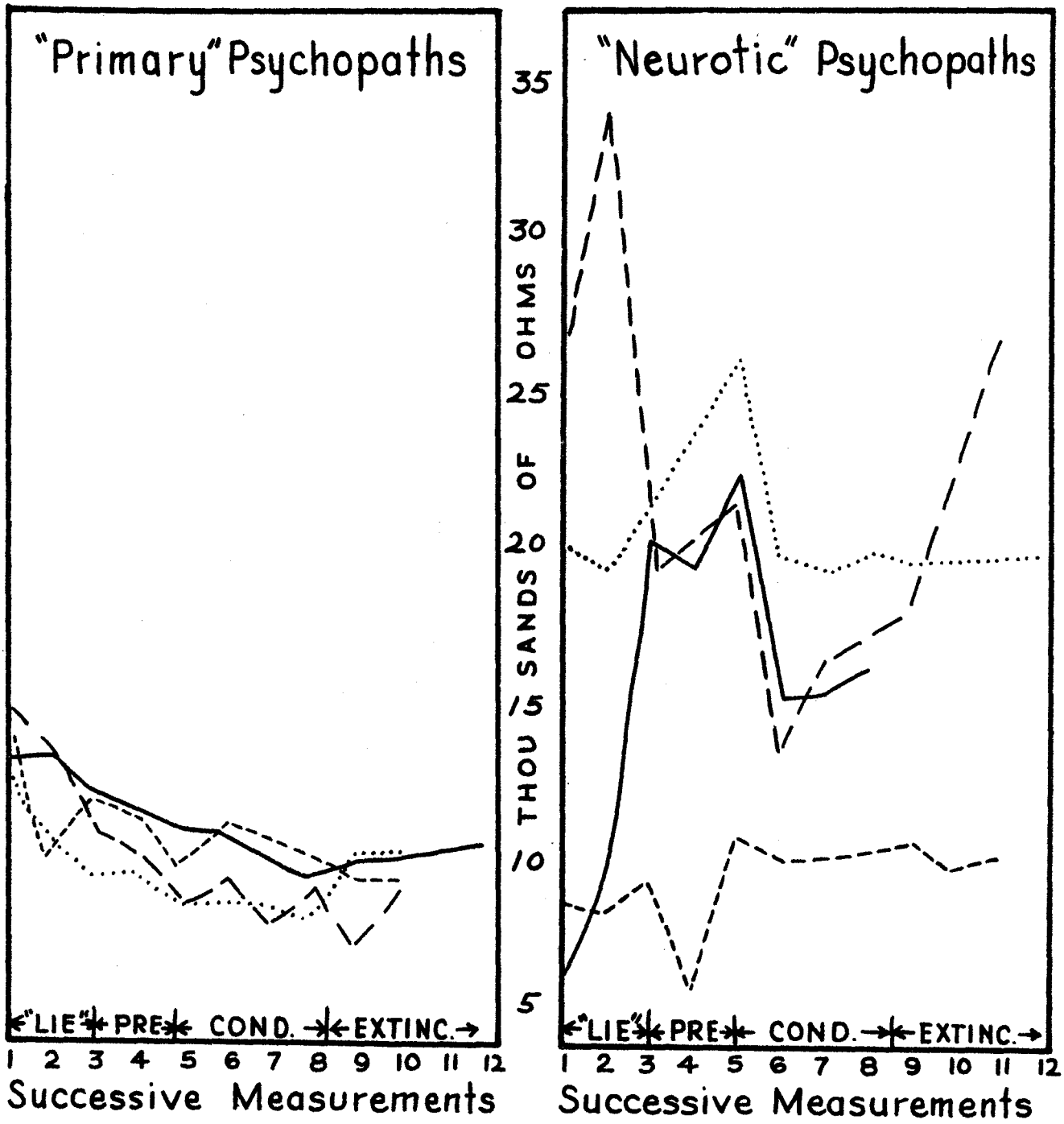

Figure 9. Individual curves of tonic skin resistance (SR) before and during conditioning trials involving painful electric shock. Subjects on the left are "primary" psychopaths showing very poor GSR conditioning and little clinical anxiety. Subjects on the right also showed reduced conditioned GSR, anticipating shock, but were clinically anxious or neurotic. These subjects, alone of those tested, showed anomalous tendency for tonic SR to increase (in the direction of decreased alertness or sleep).

change in heart rate, although both produced elevations in skin conductance as did all tasks in the series, thus demonstrated their generally excitatory or arousing character.

This experiement was planned as a test of Lacey's earlier speculation Lacey, 1959) that pressure receptors in the carotid sinus, known to exert tonic inhibitory control on electrocortical activity and on the spinal motoneurons, may also act (via the reticular system?) to inhibit sensory input as well. That is, Lacey is supposing that a set to exclude external input may, by some unspecified mechanism, generate excitation of the cardiovascular system, increasing heart rate and blood pressure which, in turn, will cause the baroreceptors of the carotid sinus to feed back impulses which result in inhibition of afferent transmission. In a recent replication, Obrist (1963) reports that each of his three extratensive tasks (e.g., a hidden pictures test) produced cardiac deceleration in all 28 of his subjects. However, blood pressure did not change significantly, suggesting that baroreceptor reflex inhibition was not operative. Conversely, the intratensive cold pressor and mental arithmetic situations elevated both HR and systolic BP.

The inductive leap to this neurophysiological hypothesis, from the observation that the two classes of stimuli produce opposite heart-rate reactions, is a long one as Lacey would no doubt be the first to admit. But it has the compensating virtue of directly suggesting a whole series of related and potentially important studies. At the physiological level, one might investigate whether afferent avoked potentials wax and wane with experimental variations in baroreceptor activity. Do sensory thresholds increase with increasing heart rate?

Is the subjective intensity of a noxious stimulus, as assessed by magnitutde estimation or by GSR, decreased when heart rate and blood pressure rise? Do persons scoring toward the hysteroid end of the hysteria-psychasthenia continum and who show repressive behavior in the perceptual defense situation show higher heart rates and blood pressures in that situation than their opposite numbers, the "sensitizers"? This research of the Lacey group offers a good example of a way in which the methods, problems and concepts of personality research, experimental psychology and physiology can be conmingled with mutual facilitation.

The provocative findings reported by Hess and Polt (1960), that pupil size varies with the interest value of visual stimuli, would seem to provide another instance of selective modulation of afferent input, in this case by means of a change in receptor sentitivity. A later study, however, rather complicates the picture (Hess and Polt, 1964; Hess, 1965). Here it was found that the pupils dilate while the subject is solving a mental arithmetic problem, with more difficult problems producing greater dilation. Since it would seem more adaptive to attenuate sensory input while solving problems in one's head, one might have expected pupillary constriction in this case. As Naquet, et al, (1960) showed in the cat, pupil size varies directly with cortical activation; one suspects that Hess and Polt's findings are not teleologically concerned with afferent modulation at all. "'What big eyes you have, Grandmother!' said Red Riding Hood. 'A mere epiphenomenon of my general state of arousal,' replied the Wolf." In any case, these experiments do underscore the significance of pupil size as a dependent variable of interest to the personality researcher.

Preception.

When a noxious stimulus, such as a brief electric shock, is made predictable in time by the use of a warning signal, the GSR elicited by that stimulus is reduced in amplitude, suggesting that there has been a reduction in the subjective intensity of the sensation. Similarly, if several shocking electrodes are attached to different parts of the body, it will be found that shocks always presented through the same electrode will yield smaller GSRs than when the site of the shock is unpredictably varied from trial to trial. I have suggested(Lykken, 1959, 1962) that such findings illustrate a kind of anticipatory afferent tuning or preception which, in the case of a noxious or interferring stimulus, attenuates the afferent signal in proportion to the subject's ability to predict the source, quality and time-of-occurrance of that stimulus. It is assumed that such tuning is effortful and cannot be maintained at peak efficiency for more than a brief time. Thus vigilance, which exemplifies "positive preception", is more efficient over brief intervals and fatiguing if continued too long. Negative preception, as indicated by decreased shock-GSR amplitudes, was greater in human subjects for 5-second than for 30-second warning intervals (Lykken, 1959) and, in the rat, showed a sharp optimum with constant warning intervals of one-second (Lykken, 1962).

One must also assume that the direction of the effect--whether the afferent system is tuned to amplify or to attenuate the expected stimulus--must depend on the subject's expectations as to whether the stimulus will be stronger or weaker than he wishes it to be. If one is sipping a fine wine, one's gustatory system should show positive

preception; while taking a dose of castor oil, negative preception is indicated. When sampling your first fried grasshopper, a little afferent attenuation will protect you while still permitting you to discover that, well salted, grasshoppers can be tasty. Positive preception would be expected in a subject whose auditory threshold is being determined but one would expect negative preception to supervene, and the threshold to rise, if one began giving a 100 db tone on a few of the trials at the moment when the subject was expecting a tone at near-threshold intensity.

An interesting aspect of this research has to do with the remarkable range of individual differences in "preception ability" which are observed even in the relatively homogeneous population of male college students. Some subjects cease responding altogether to the predictable shocks (those preceeded by the warning tone) within 10 to 15 trials, although continuing to respond consistently to the interpolated shocks given without warning. Other subjects, although their average GSRs to the predictable shocks may be smaller than those produced on the shock-alone trials, still continue to emit sizeable GSRs under both conditions even after 100 or more repetitions of the stimulus. This ability to ignore or attenuate unavoidable noxious stimuli has such adaptive significance that is seems important to inquire into the possible causes of such marked individual variability, both situational (e.g., fatigue) and also constitutional. One is led to wonder what effect extreme over- or under-endowment in "preception ability" might have upon the course of personality development.

The Mechanism of Attention.

Any harried housewife will tell you that she "can't do two things at once!" She can, of course; she can tell Johnny to tie his shoes while at the same time hurrying across the kitchen to the stove, without neglecting to breathe, blink her eyes or dodge the cat. What she can't do, it appears, is to perform two functions absolutely simultaneously, both of which require her conscious attention. That highest level of cerebral processing, wherein seems to reside the seat of awareness, is apparently a single-channel mechanism which can admit, process and respond to only one input, one "chunk" of information, at a time. Experimental evidence for this is illustrated by the work of R. Davis (1956, 1957, 1959). The general paradigm is to provide a subject with two response keys with instructions to press one key with the left hand upon seeing the first signal, S_1, and the other key with the right hand upon seeing S_2. When the time interval between S_1 and S_2 is more than 250 mSecs, the reaction time is about the same to both signals (e.g., 150 mSec for visual signals). As the interval between S_1 and S_2 is shortened below 250 mSec, the reaction time to S_2 starts to lengthen; for intervals of less than 150 mSec, shortening the interval by 50 mSec lengthens the reaction time to S_2 by about 50 mSec. Even if the subject is not required to respond to S_1 at all the effect is very nearly as great; most of the delay arises "as a result of paying attention to the first signal, rather than the performance of any overt response to it". (R. Davis, 1959, p. 220).

Many details remain to be filled in. This "psychological refractory period," (Welford, 1952) seems to be longer than the estimated time required for central processing of a single reaction. In one of

R. Davis' experiments (1957), the single central channel appeared to be blocked, unable to begin processing S_2, for some 180 mSec after the signal of S_1 reached the cortex, even though the motor impulse for R_1 was initiated within about 100 mSec and the response actually completed 50 mSec later. It is known that S_1 and S_2 may be in different modalities without changing the effect, i.e., a subject cannot begin processing a visual signal or an auditory signal until the end of a refractory period of, say, 180 mSec after a preceeding visual signal first captures his attention (R. Davis, 1959). Although individual differences appear not to have been studied systematically as yet, subjects do seem to differ in the length of their refractory periods and one suspects that this parameter should vary inversely with arousal. It is natural to think of some sort of scanning operation here; the attention channel may open briefly at regular intervals so that S_1 must wait until the next "opening" and S_2 must wait in its turn until the first "opening" after S_1 has been processed and the attention channel is released to accept a new input. Is the cortical alpha rhythm related to this scanning process? At an alpha frequency of ten per second, S_1 would have to wait 50 mSec on the average to capture the attention channel (assuming one "opening" per cycle, perhaps during the downward crossing of the equipotential axis as implied by Lansing's study [1957], mentioned earlier); allowing then one full cycle for processing, this would yield a "psychological refractory period" of about 150 mSec, which is perhaps close enough to the values estimated in Davis' experiments to be worth investigating.

Of special interest is the question of whether even greatly over-learned automatized reactions to moderately strong signals would also

display this refractory period effect. Perhaps any signal, no matter how mundane, familiar and highly practised, will capture the attention of a quiescent subject who has nothing better to do; such a signal figuring as S_1 in the paradigm might be followed by the usual refractory interval. But used as S_2, a signal for an automatized reaction might not show a lengthened latency even when closely following S_1, indicating that S_2—R_2 was being processed at a lower level.

What constitutes a single signal for this mechanism? How much perceptual information can be admitted to the single attention channel at one time? Undoubtedly, as Miller (1956) has suggested, the information-handling limitations of the channel can only be defined in relation to the past learning of the individual. Thus, one can identify a familiar printed word at tachistoscopic presentation speeds too fast to allow one to spell out all the letters of a nonsense word of equal length. Just a little information about a familiar gestalt or "chunk" is adequate to trigger the appropriate schemata already present in one's mind. The activated schemata adds information to that provided by the senses in the same manner that a scientific hypothesis adds to the information contained in the experimental facts which suggested it; in either case, of course, one may be wrong.

As the Deutschs (1963) have argued, it appears to be the case that a great deal of complex perceptual processing takes place outside of awareness, before the perceptual signal even becomes a candidate for admission to the attention channel. We have seen that one can respond selectively to one's own spoken name during sleep (Oswald, Taylor and Triesman, 1961). Moray (1959) found when a subject is fed different

messages to the two ears simultaneously (this is called "dichotic" listening) and has "tuned out" on one channel in favor of the other, speaking his name on the rejected channel may cause him to switch his attention back to this channel. Thus, the complex pattern analysis required to identify one's name can clearly be accomplished outside of attention. Other data on dichotic listening, summarized by Deutsch and Deutsch (1963) support the belief that what might be called the "preconscious analyzers" (corresponding to Broadbent's [1958] "filter"), must be capable of very high-level analysis (discrimination, identification) indeed. Thus, we may reasonably suppose that the raw flux of sensory input is analyzed preconsciously, not just for such primative attributes as intensity, figure-ground properties, and the like, but also into patterns and, to a certain extent, for "meaning." Patterns are identified as familiar or not, as salient or not, and we may assume that patterns having some immediate emotional significance can initiate the appropriate emotional reaction, or at least that such patterns receive a special increment in their attention-getting power.

At each interval at which the attention channel opens, we can thus imagine an array of partially predigested signals, all competing for admission. It seems reasonable to assume that the signal gaining admission will be the one which is at that moment in some sense the strongest; but, as a result of the preconscious analysis, the "strength" of a signal at this level will depend less on the intensity of the original physical stimulus involved and more on the modulation resulting from the "significance" which has already been attributed to that signal by the analyzers. As in the theory suggested by Blum (1961),

we might also expect to find that a continuing signal which has already achieved admission to the attention channel will be given an advantage in this competition at each successive "opening," by means of some kind of positive feedback or "reverberation" as Blum calls it, in order to maintain a better continuity of action of this highest level analyzer.

Since we are assuming a very large number of competing inputs, the problem arises of how the attention mechanism can determine the "strongest" without running through an impossibly large number of paired comparisons. The Deutschs (1963) suggest that some intensity mechanism, perhaps the reticular activation system, may have its level determined, pre-consciously, by the "strength" of the strongest oompeting input; the attention channel is then able to ignore all inputs whose "strengths" are less than this level. This device, however, seems merely to push the problem back one step; how then does the intensity mechanism determine which input is "strongest?" Perhaps one should not be too quick to reject the paired-comparison idea (brains and computers make such comparisons easily; it is selecting one from many directly that is difficult to engineer); with its notoriously large number of inter-connected units, the brain may do just that, perhaps in some hierarchical sequence in which all "winners" advance to the next higher level. Although the present writer is most sympathetic to Deutsch's (1960) structural approach to theory building--"how might I build a machine which would behave this way?"--it is perhaps pushing the limits of respectable speculation too far to carry this problem any further at the moment.