THE JOURNAL OF NERVOUS AND MENTAL DISEASE
Vol. 141, No. 6

Printed in U.S.A.

A NEUROPHYSIOLOGIC MODEL OF DREAMS AND HALLUCINATIONS[1]

RAÚL HERNÁNDEZ-PEÓN, M.D.[2]

While dreaming represents a particular state of consciousness occurring normally every night during certain periods of sleep, hallucinations are considered as a pathologic sign of abnormal mental function. However, a similarity between both types of subjective phenomena has been recognized since very ancient times, and even a similar substrate was assumed by Plato (20). It seems logical that any insight into one of those processes might help in clarifying the other. The difficulty in understanding the origin and meaning of the often vivid and odd experiences of dreaming has led all primitive human races to attribute magical properties to the unexplained phenomenon. It was not until Freud (29) achieved a sound and systematic investigation of dreams by correlating them with the individual past experiences and personality, that a comprehensive interpretation became available to psychologists and psychiatrists. The recent progress of neurophysiology in attempting to bridge the realms of brain and mind allows us now to formulate working hypotheses which may guide and stimulate future research on this fascinating subject.

Based on experimental findings, I will present in this paper some speculative ideas integrated into a neurophysiologic model which can account for the main features of dreaming and hallucinations. Without pretending to give the final answer to these important problems, the aim of this effort is to build up a theoretical framework modifiable according to forthcoming experimental evidence.

Two well substantiated concepts have provided a basis for a hopeful insight into the neural mechanisms of dreaming, namely 1) sleep is the result of an active inhibitory process originated from specific hypnogenic brain structures; 2) two main patterns of physiologic changes accompanied by low and high arousal thresholds respectively, occur during sleep, the latter being more often associated with dreaming. A brief introductory account of the experimental evidence supporting these current views is germane to the neurophysiologic discussion of dreaming.

SLEEP INDUCED BY CENTRAL ELECTRICAL STIMULATION

To Hess (56, 57) we owe the first experimental demonstration that behavioral sleep can be elicited by low frequency electrical stimulation of certain brain regions, and in particular of a midline thalamic area in the vicinity of the massa intermedia. In confirming this finding (4, 5, 77, 78, 95) it has been additionally shown that high-frequency electrical stimulation of the same midline thalamic area produces EEG and behavioral arousal. More recently, differential arousing and hypnogenic effects have been obtained by high-frequency or low-frequency electrical stimulation of all the brain stem reticular formation (25) and of the medullary region of the solitary tract (80). It has also been reported that low-frequency electrical stimulation of the head of the caudate nucleus produces behavioral quieting or sleep and cortical spindle bursts (14, 15, 33, 89). The fact that sleep can be induced from widespread

[1] This work was supported by the National Institutes of Mental Health USPHS under grant No. MH-10003-01, by the Foundation's Fund for Research in Psychiatry under grant No. 64300 and by the Army Research Office under grant No. DA-ARO-49-092-G62.

[2] Instituto de Investigaciones Cerebrales, A.C., Moras 445, México 12, D.F.

regions in the brain seemingly suggested a nonspecific effect of diffuse origin. Of particular importance in this regard was the finding independently obtained by Sterman and Clemente (102) and by Hernández-Peón (38) that both low- and high-frequency electrical stimulation of the medial forebrain bundle at the lateral preoptic region elicited the behavioral and electrocortical manifestations of sleep. Therefore, although there is an extensive overlapping of arousing and hypnogenic neurons, the hope of finding localized specific hypnogenic structures regained experimental support.

SLEEP INDUCED BY CENTRAL CHEMICAL STIMULATION

Accepting current views about the chemical nature of central synaptic transmission in the mammalian nervous system, we have experimentally approached the problem of mapping out specific hypnogenic regions by local application in the brain of naturally occurring substances normally synthesized as possible synaptic transmitters as well as of pharmacologic agents which are known to interfere with the synthesis, membrane action or enzymatic destruction of those substances.

In brief, it has been found that the application of minute crystals of acetylcholine, carbamylcholine or eserine elicited all the behavioral and electrographic manifestations of sleep only when applied along well defined anatomic central pathways (38, 39, 41, 43–45, 49, 50).

The chemical specificity of the effect is demonstrated by the fact that no other substance so far tested (epinephrine, gamma-aminobutyric acid, nialamide, strychnine sulfate) ever produced sleep. The highly discrete anatomic localization of the hypnogenic pathway and the diffusionless ability of the method were dramatically shown when application of the same cholinergic agent in the same animal 1 mm above or below the hypnogenic site evoked entirely different behavioral responses. So far, sleep has been induced by acetylcholinic stimulation of a number of localized regions anatomically connected and extending from the spinal cord up to the cortex. This pathway has been termed Sleep System (44). As depicted in Figure 1 the Sleep System is made up of two components: an ascending spino-bulbo-pontine component which arises from the gray substance of the spinal cord at the thoracic level (44, 54), and a descending cortico-limbic-midbrain-bulbar component represented by structures belonging to Nauta's limbic midbrain circuit (83, 84) and its connections to the bulbar n. reticularis gigantocellularis (49, 50, 52), as well as by corticofugal projections to this circuit arising from the prepyriform, pyriform and periamygdaloid cortex of the temporal lobe (52), from the orbital surface of the frontal lobe, from the perisylvian cortex, and from the anterior cingulate gyrus (75). In addition, cholinoceptive hypnogenic areas have been detected in midline and intralaminary thalamic nuclei (n. rhomboidalis, n. centralis medialis, the limit of n. centralis medialis, the limit of n. centralis medialis and n. reuniens, n. centralis lateralis and n. reticularis) (44, 85, 108) as well as in striate structures (head and tail of the caudate nucleus, globus pallidus, claustrum superior and entopeduncular nucleus) (52).

The hypothesis postulating that acetylcholine may be an excitatory synaptic transmitter along the multisynaptic Sleep System is supported by two findings: 1) local application of an anticholinestearasic substance (eserine) in the hypnogenic preoptic region elicited sleep; 2) local application of a cholinergic blocking agent (atropine) at caudal segments of the limbic midbrain hypnogenic circuit prevented sleep otherwise produced by acetylcholinic stimulation of the hypnogenic preoptic region (105).

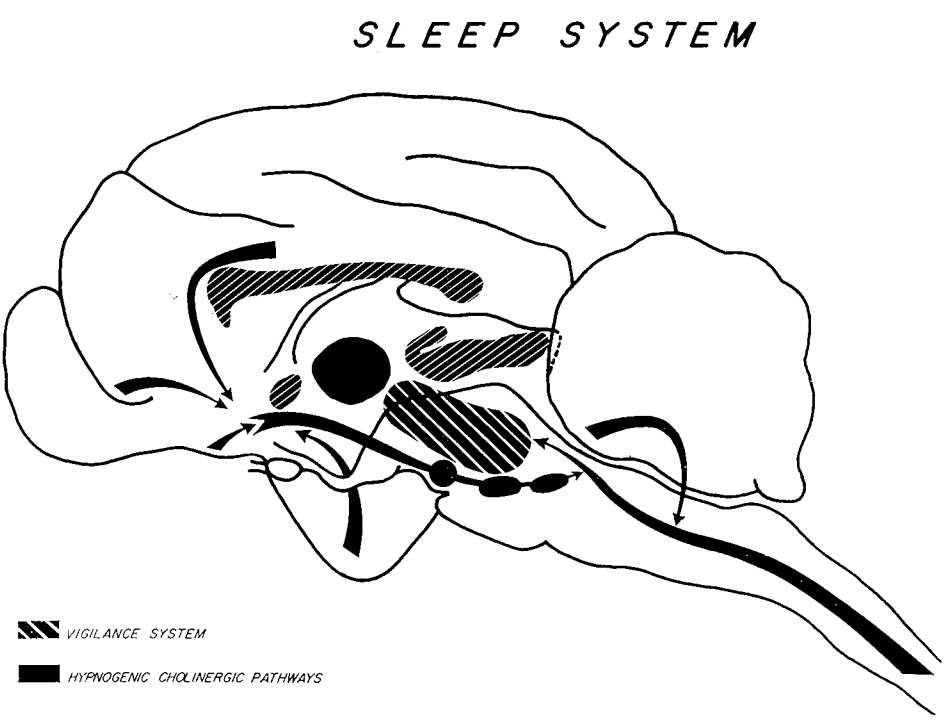

FIG. 1. Diagrammatic representation of the sleep system traced by acetylcholinic stimulation of the central nervous system. This hypnogenic pathway is made up of 2 components: 1) a spino-bulbo-pontine component starting from the spinal cord and represented by the ascending arrow impinging upon the vigilance system (hatched); 2) a descending cortico-limbic-midbrain-bulbar component represented by the arrows originated at the cingulate gyrus, the orbito-frontal cortex, and the pyriform cortex converging upon the descending limbic-midbrain circuit.

TONIC ACTIVITY OF CENTRAL HYPNOGENIC STRUCTURES

Just as the Vigilance System is tonically active, all the available evidence indicates that the Sleep System is also endowed with tonic activity all along the neuroaxis. This has been shown either by localized lesions, or by reversible depressions produced by drugs or cooling.

There is evidence that ascending hypnogenic influences arise at the spinal cord, at the medulla oblongata and at the posterior part of the pons. Hodes (58) has demonstrated that unilateral injections of small amounts of novocaine in the spinal cord at C-1 causes bilateral electrocortical desynchronization which may last 15 minutes or less. Similar but shorter effects were obtained by unilateral anesthetization of the caudal medulla. The view of ascending spinal hypnogenic influences is supported by experiments in which sleep elicited by acetylcholinic stimulation of the spinal gray substance was prevented by local novocainization of higher spinal segments (44, 54).

Direct evidence of tonic activity in medullary structures antagonistic to the Vigilence System has been provided by the following experiments. The EEG desynchronization produced by direct electrical stimulation of the midbrain reticular formation or by arousing sensory stimuli in cats with spinal transections at C-1 was considerably prolonged after a prebulbar transection (11). Since this effect was ob-

tained when the lesion was made 2 mm laterally to the midline but not when the transection was made on the midline (12), it has been concluded that the main synchronizing structures are laterally located in this part of the medulla. The presence of prevailing synchronizing structures in the medulla oblongata has been shown by localized cooling of the floor of the fourth ventricle which produces EEG desynchronization in contrast with the synchronizing effects elicited by cooling the pons (10, 81).

The existence of pontine hypnogenic structures with tonic activity is supported by two lines of evidence. While hemisection of the midbrain and rostral pons produces a predominance of unilateral EEG synchronization, hemisection of the middle part of the pons is followed by a reduction of EEG synchronization with inconsistent presence of the desynchronized pattern of deep sleep. Hemisection of the caudal pons does not produce EEG asymmetry (92). These results suggest that the middle pons contains a high concentration of hypnogenic neurons. On the other hand, by placing bilateral localized lesions in the pontine tegmentum, Jouvet (63) found that destruction of the n. reticularis pontis caudalis (NRPC) resulted in a state of insomnia with constant desynchronized EEG and waking behavior during the first two days.

A similar interference of sleep has also been produced by forebrain lesions. Nauta (82) observed that bilateral transections of the preoptic region involving the medial forebrain bundle resulted in a state of insomnia terminating in letal exhaustion after a few days. Manceau and Jorda (74) reported a state of sleeplessness in the guinea pig after similar lesions. Modern electrophysiologic recordings made before and after localized basal forebrain lesions (Sterman and Clemente, personal communication) have revealed significant reductions of the amount of sleep measured throughout the daily 24 hours.

By the same token, local application of minute crystals of atropine at the preoptic region, interpeduncular neucleus, or Bechterew's and Gudden's nuclei resulted in a transient state of alertness and motor hyperactivity (105).

In summary, it may be concluded that reversible or irreversible local inactivation of any part of the Sleep System, either at the spinal cord, brain stem or forebrain, is followed by a state of hypervigilance of variable duration. The most plausible interpretation is that tonic activity exists at each level which may depend from an intrinsic neuronal automatism. If we accept the existence of fixed cyrcadian rhythms in the overall activity of the Sleep and Vigilance Systems as well as reciprocal inhibitory connections between them, as postulated by Hernández-Peón and Chávez-Ibarra (45), the alternation of wakefulness and sleep through the phylogenetic scale becomes more understandable. Furthermore, the prevailing excitatory influences impinging upon each system and arising from sensory receptors and from the cortical mantle as illustrated in Figure 2 would explain the phylogenetic and ontogenetic development of the sleep-wakefulness rhythms.

A DUALISTIC THEORY OF SLEEP

It is now well established that sleep is associated with two main patterns of physiologic indices. At the onset of sleep the cortical EEG is characterized by slow waves and spindle bursts, and there is a slight diminution of the EMG of the nuccal muscles. After a variable period usually lasting more than 30 minutes, the slow cortical activity is replaced by low voltage fast waves, the EMG of the nuccal muscles becomes practically isoelectric, the blood pressure drops significantly, the spinal reflexes are markedly inhibited,

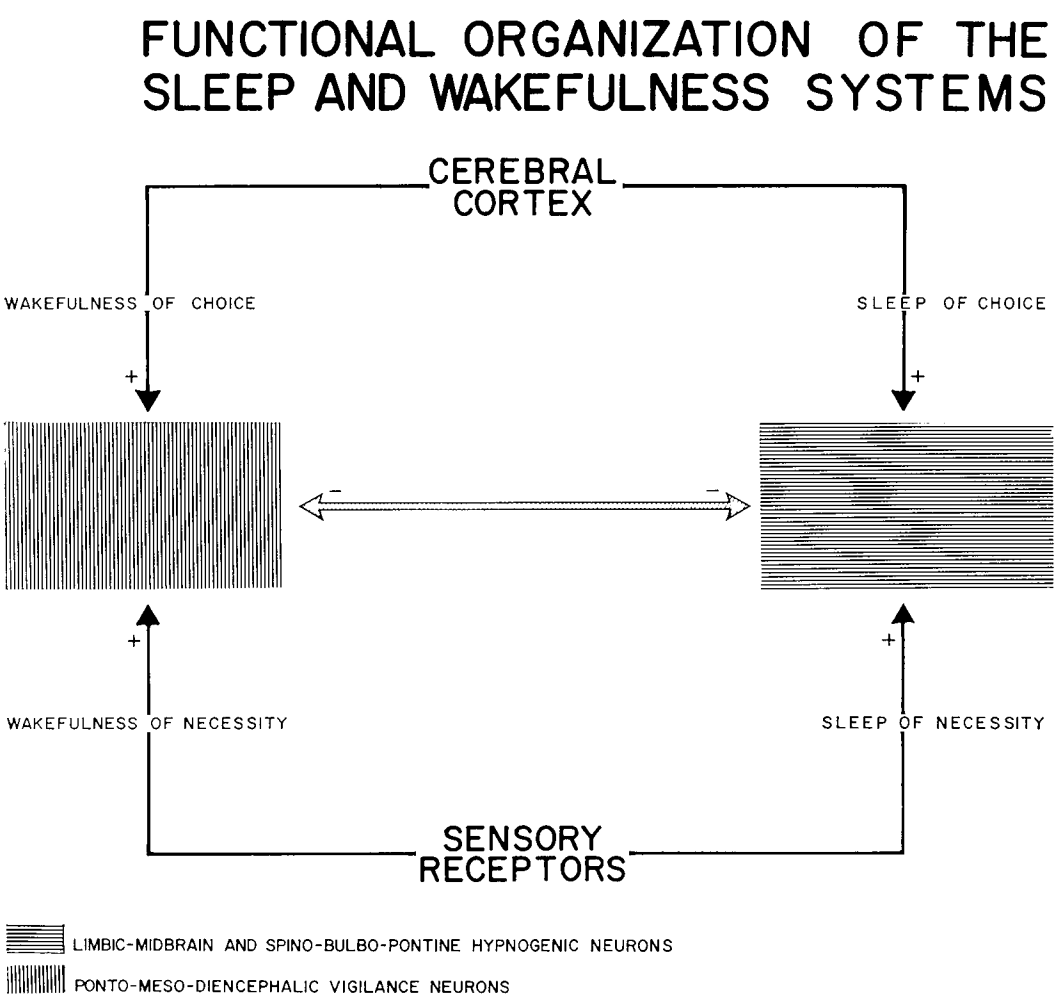

FIG. 2. This figure illustrates the dynamic equilibrium between the vigilance and the sleep system maintained by reciprocal inhibitory connections. The intrinsic automatic activity within each system can be increased by excitatory impulses coming either from sensory receptors or from the cortical mantle.

high voltage sinusoidal theta waves are recorded in the entorhinal cortex, and bursts of rapid eye movements and twitches of body muscles appear intermittently. The latter pattern is associated with a marked elevation of the arousal threshold in the cat, and very often with dreaming in the human. The remarkable dissimilarity between the two patterns of sleep has led some neurophysiologists to believe that they represent two separate entities, and even to search for different mechanisms. From a series of experiments designed to explore the neural mechanisms underlying the two basic patterns of sleep, Jouvet (64) has reached the conclusion that the light stage or slow sleep (SS) depends from descending influences originating in the neocortex whereas the deep stage or rapid sleep (RS) results from an independent neural mechanism located in the pons at the level of NRPC (Figure 3). In support of this hypothesis he claims the following evidence: 1) Total removal of the neo-

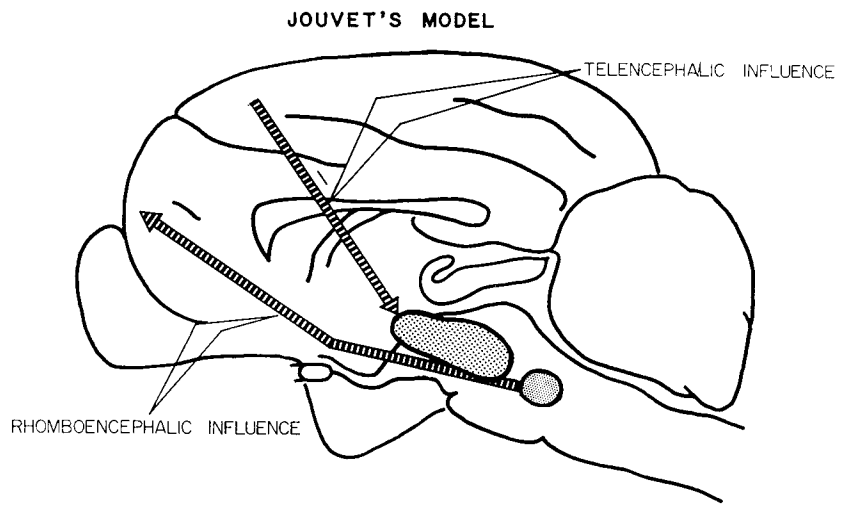

FIG. 3. Diagrammatic representation of Jouvet's hypothesis for explaining the two main patterns of sleep. According to this hypothesis slow sleep is produced by descending inhibitory corticofugal influences whereas rapid sleep would result from ascending influences originated at the nucleus reticularis pontis caudalis and acting upon the cortex.

cortex eliminates slow electrical activity in the midbrain during sleep leaving intact all the electrophysiologic signs of RS. 2) A localized lesion involving the NRPC prevents the desynchronized EEG, the flat EMG of the neck muscles and the bursts of REM normally associated with the phase of RS. An ascending pathway by-passing the arousing reticular formation and following the limbic midbrain circuit from Gudden's and Bechterew's nuclei to the medial forebrain bundle has been pointed out as responsible for electrocortical desynchronization during RS. 3) Chronic pontine preparations with total removal of the brain in front of the pons present periods of hypotonia of the neck muscles regularly alternating with periods in which the muscle tone recovers (65, 66).

A more critical appraisal of these arguments weakens the convincing strength of their fallacious logic. Evidently, in the mutilated decorticate and pontine preparations the author uses the absence or presence of different electrophysiologic signs pertaining to the sleep constellation as a decisive criterion for identifying each stage of sleep. Since the EEG slow waves are cortically generated and the spindle bursts are triggered by thalamocortical activity initiated in the thalamic recruiting nuclei, it is logical to expect a disappearance of the slow activity propagated to the brain stem when an important part of the generator is ablated. Undoubtedly, it is difficult to show by ablation that a certain phenomenon depends upon a structure where the cause and effect are supposed to take place. In order to have a more reliable criterion it would be more convenient to perform in the same animals, before and after decortication, quantitative measurements of other physiologic indices such as the arousal threshold measured by direct electrical stimulation of the mesencephalic reticular formation, recordings of the blood pressure and of spinal reflexes. On the other hand, it seems unwarranted to describe sleep in a pontine preparation which has been deprived of the structures essential for wakefulness. The concept of a sleeping brain in front of the rostropontine transection seems more justified than the idea of a sleeping spinal cord below. Actually, it can only be stated that such a vegetative preparation shows rhythmic ac-

tivity of inhibitory neurons acting upon spinal motoneurons. The statement that RS and its associated oneiric experiences can only be triggered from a restricted area in the pontine reticular formation needs to be re-evaluated in the light of more recent experimental evidence. It has been shown that RS can be elicited after a brief phase of SS by acetylcholinic stimulation at specific regions located in the forebrain, midbrain, hindbrain or spinal cord (43, 44). Likewise, lesions involving the NRPC are not the only ones which impair RS. Utilizing multiple electrophysiologic recordings Sterman and Clemente (personal communication) have found that basomedial forebrain lesions specifically decreased the occurrence of 4 to 8/second slow waves associated with drowsiness, whereas basolateral lesions at the same level significantly decreased the percentage occurrence of RS over a 24-hour period. Both groups of results are incompatible with the hypothesis of a "pontine RS or dream center." Furthermore, the opinion that EEG desynchronization during deep sleep episodes is mediated by ascending limbic midbrain pathways is not supported by more recent experiments (16).

A MONISTIC THEORY OF SLEEP

The results obtained by localized chemical stimulation of the central nervous system have clearly demonstrated that the sleep continuum can be induced by acetylcholinic stimulation of a highly circumscribed anatomic pathway extending throughout all the levels of the neuroaxis. When minute crystals of acetylcholine or carbamylcholine were applied at any point of this pathway whether at the spinal cord or at the forebrain, SS always preceded RS. Since the same sequence of events was elicited from such remote central regions, the conclusion seems warranted that the neural mechanisms responsible for the physiologic manifestations of the two patterns of sleep must be triggered by a final common region of convergence. The most plausible location of this juncture is the posterior pontine or rostral bulbar tegmentum from where the final hypnogenic neurons conceivably send ascending inhibitory connections to the arousing neurons located in the rostral part of the pons, midbrain and posterior hypothalamus. Indeed, electrolytic lesions involving the nucleus reticularis gigantocellularis in the medulla oblongata prevented sleep otherwise produced by acetylcholinic stimulation of the pyriform cortex (52, 53). The anatomic organization of a homogenous anatomically circumscribed Sleep System made up of an ascending spino-bulbo-pontine component and a descending cortico-limbic-midbrain-bulbar component explains why a midpontine transection interrupting the final end of both limbs results in a persistently awake brain (8). The progressive recurrence of longer periods of EEG synchronization in the chronic midpontine preparation suggests the existence of higher collateral inhibitory connections from the descending hypnogenic pathway at diencephalo-mesencephalic levels which then might act upon sensitized chronically denervated arousing neurons.

According to this view, the continuum of sleep from SS to RS would result from different degrees of activity within a unique hypnogenic system independently of the segment initially more activated. For instance, it is possible that somatic sensory stimuli activate primarily the ascending spino-bulbo-pontine hypnogenic pathway, that post-coital somnolence might arise from limbic-diencephalic hypnogenic structures, and that conditioned sleep associated with habits may be initiated by activity originated in cortical hypnogenic areas. A progressively increasing inhibition ascending from the rostropontine to the thalamic limits of the Vigilance System would explain the EEG manifestations of the two patterns of sleep without having to postulate two different and independent anatomic

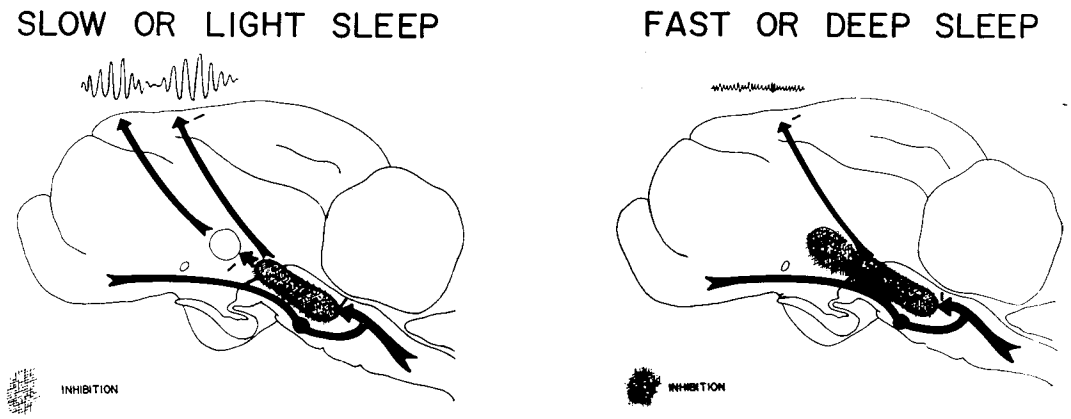

FIG. 4. Diagrammatic representation of the monistic hypothesis for explaining the cortical electrical activity recorded during the two patterns of sleep. According to this hypothesis an ascending inhibition starting from the bulbar juncture of the sleep system invades progressively the midbrain first and later the midline recruiting thalamic nuclei. Therefore, while the latter are released at the first stage giving rise to spindle bursts, they are no longer capable of triggering the high voltage slow activity during deep sleep.

neural mechanisms (Figure 4). The spindle bursts and cortical slow waves of the first stage of sleep would result from disinhibition of the cortex and of the recruiting thalamic nuclei when the ascending tonic inhibition from the mesencephalic arousing neurons is released as they are inhibited by the hypnogenic neurons. When inhibition invades the midline recruiting thalamic nuclei during the desynchronized stage of sleep, thalamic spindle bursts are no longer sent to the cortex which then shows a predominantly low voltage pattern of activity. At the same time that ascending hypnogenic inhibition progresses, recruitment of bulbopontine inhibitory neurons is produced with a faster rising slope at the end of SS. This would explain the almost abrupt decrease of excitability of spinal motor, cardio-pressor and respiratory neurons observed at the onset of deep sleep episodes. Short lasting diminutions and enhancements of activity of different groups of inhibitory neurons would explain the phasic physiologic changes accompanying the bursts of REM.

This monistic physiologic model of sleep has been supplemented by evolutionary phylogenetic considerations (43, 44). According to this theory, both the Vigilance and the Sleep Systems were early represented in the primitive neural tube. Through encephalization, their main part remained localized at the brain stem level which later received limbicofugal and neocorticofugal projections as the pallium emerged and developed.

A NEURONAL DREAM SYSTEM

With these available data, it is possible now to discuss a possible neurophysiologic explanation of dreaming. The correlation found between RS and dreaming in the human has led some neurophysiologists to assume that certain changes of brain electrical activity detected during REM sleep episodes might be identified with the neural process underlying dreaming. Because this pattern of sleep is seemingly eliminated by a lesion or NRPC, Jouvet (64) has suggested that dreams are triggered by activity in this region. Criticisms to this view have already been mentioned. Based on the appearance of monophasic waves at the lateral geniculate body and visual cortex simultaneously with bursts of REM during sleep, Moruzzi (80) has advanced the hypothesis that postsynaptic

discharges of geniculocortical neurons might be held responsible for the triggering of the neural process underlying the visual imagery of dreams. Admitting that the geniculocortical discharges may have a pontine origin, it is doubtful that this random activity may give rise to organized visual imagery since it is known that electrical stimulation of the human visual cortex has never produced this type of experiences. On the other hand, complex organized dream-like experiences or vivid memories have been elicited by electrical stimulation of the temporal cortex (90) and hippocampus (88). It seems more reasonable to conceive that dream experiences result from activation of mnemic neurons with stored memory tracings according to the following neurophysiologic model.

One of the basic premises requires the existence of a specific neuronal system essential for dreaming, which henceforth will be termed "Dream System." The Dream System is probably represented by the same anatomic structures involved in recent memory, since it is known that the main part of the manifest content of dreams is elaborated with stored material from recent experiences, and particularly the so-called day's residues (29). It is now known that some limbic structures, and in particular the hippocampal formation made up of the hippocampus and the hippocampal gyrus with their subcortical connections represent an important part of the brain for recording recent experiences. Indeed, it has been shown in humans that bilateral lesions of the hippocampal formation impair recent memory (30, 76, 97). Therefore, this part of the limbic system with other related structures may well represent the anatomic substrate of the Dream System.

How can this hypothesis be experimentally tested? If the Dream System is at least partially located in the hippocampal formation, the activity within it should be increased during the periods of RS associated with dreaming in the human (21). Some initial recordings with bipolar electrodes made in our laboratory suggest that the activity in the hippocampal formation is increased during RS. Figure 5 illustrates simultaneous recordings of the eye movements, the electrical activity of the neocortex, that of the entorhinal cortex (which would correspond to the hippocampal gyrus in the primate's brain) and the EMG of the neck muscles. During SS when spindle bursts and slow waves are recorded in the neocortex, there is an activity in the entorhinal cortex which is similar to that recorded during the waking states, although there are some waves of higher voltage at this moment. But what is particularly remarkable is that, during RS, the electrical activity in the entorhinal cortex is represented by a sinusoidal theta rhythm which increases both in frequency and amplitude during the bursts of REM. This has been described also for the hippocampus. However, when simultaneous recordings are made from the hippocampus and from the entorhinal cortex, the phenomenon is more outstanding in the latter region. Although this finding alone does now warrant a definite conclusion, since the waves are increased both in frequency and amplitude, it is possible that an increase of activity occurs precisely during the periods of RS. Certainly, microelectrode studies will be necessary to show whether this pattern of electrical activity recorded with macroelectrodes corresponds to an increase of unitary neuronal discharges.

WAKING INHIBITION OF THE NEURONAL SYSTEMS UNDERLYING MEMORY, EMOTIONS AND MOTIVATIONS

In this model the Dream System is tonically inhibited by the Vigilance System, which in turn is tonically inhibited by the Sleep System. These three systems would have a similar organization: the activity in each of them can be increased by impulses coming either from sensory

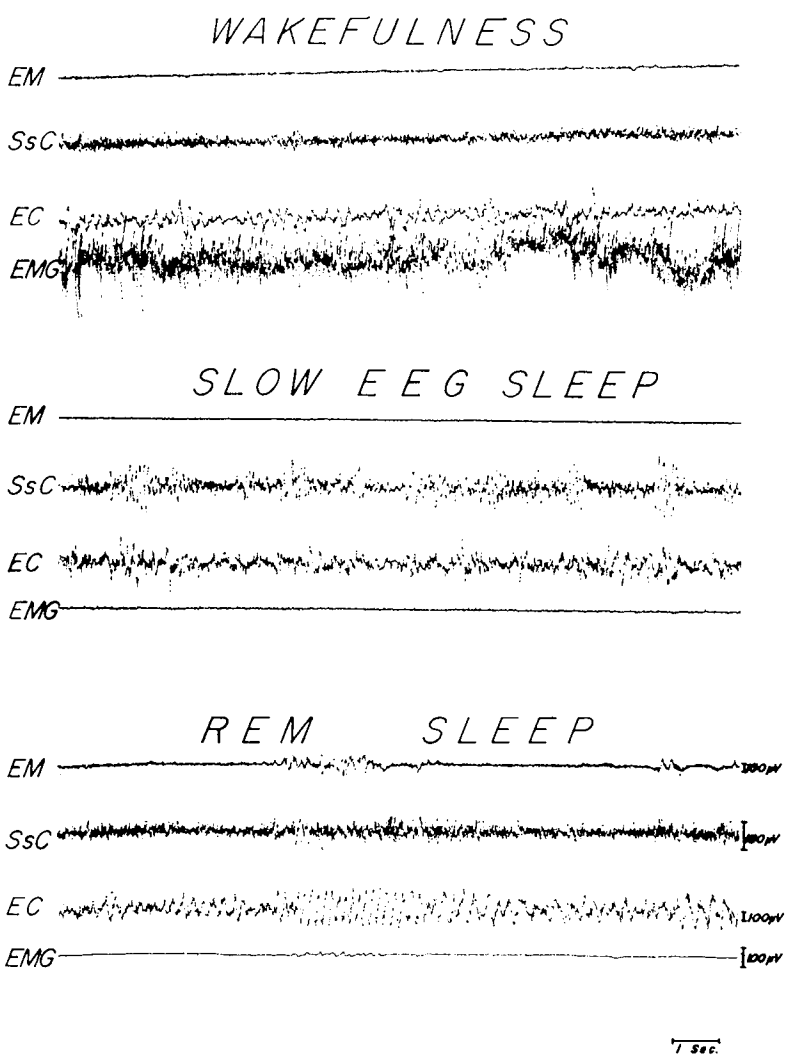

FIG. 5. Simultaneous recordings of eye movements (EM) electrical activity from the supra-Sylvian cortex (SSC) from the entorhinal cortex (EC) and from the neck muscles (EMG) in a cat during wakefulness and during the phases of SS and RS. Notice the appearance of high voltage theta rhythmic waves in the entorhinal cortex during the bursts of rapid eye movements in RS.

receptors, or from the neocortex (Figure 6). Whereas "wakefulness of necessity," "sleep of necessity" and "dreaming of necessity" are produced by sensory stimulation, corticofugal impulses would be responsible for "wakefulness of choice," "sleep of choice" and "dreaming of choice." Just as it is possible to awaken or fall asleep voluntarily, common experiences have taught us that it is possible, although not easy, to continue at will an interrupted dream. Simultaneously with the tonic inhibition arising from the Vigilance System during wakefulness and acting upon the Recent Memory System, this model also includes inhibitory influences acting upon the Remote Memory System as well as upon the Emotional and Motivational Systems. While the anatomic substrate of the Remote Memory System certainly involves

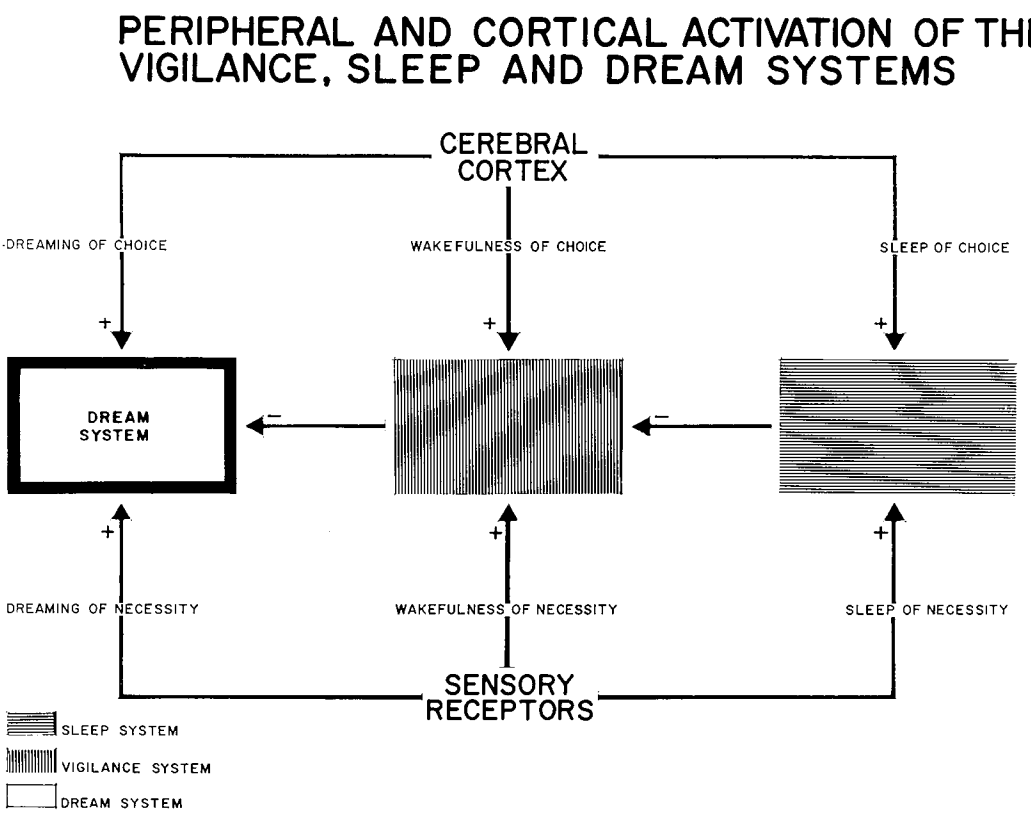

FIG. 6. Functional organization of the neural systems responsible for sleep, vigilance and dreaming with their corresponding inhibitory influences and their excitatory influences coming from sensory receptors and from the cerebral cortex.

the neocortex, the Emotional and Motivational Systems have been located in the limbic system by many neurophysiologic studies.

The important participation of the limbic system in the basic motivations of hunger, thirst and sexual appetite has been experimentally demonstrated in the last few years. For example, it has been established that electrical or chemical (noradrenalinic) stimulation of the lateral hypothalamus elicits food intake in an animal previously satiated, and that the destruction of this hypothalamic region produces aphagia. Contrarywise, a lesion in the ventromedial hypothalamus elicits hyperphagia which may lead to obesity, and electrical stimulation of this area prevents food intake (13). The lateral region of the hypothalamus mentioned above has been designated "hunger center" while the ventromedial region has been termed "satiety center." It would be better to call them "phagogenic area" and "anti-phagogenic area," respectively. In turn, these two areas are influenced by higher amygdaloid structures which may also modify food intake (79). Thirst may be elicited by electrical, acetylcholinic or osmotic stimulation of a region medially located in the hypothalamus. Correspondingly, a lesion in this hypothalamic "dipsogenic area" produces adipsia (13). As far as sexual appetite is concerned, the recent experiments of Mac Lean (70–72) in the squirrel monkey have demonstrated that stimulation of a diencephalic region medially located and extending between the septum and the mid-

brain produces penile erection. On the other hand, Harris *et al.* (32) have found in the female cat that implantation of small amounts of diethylstilbestrol in a localized area of the hypothalamus produces estrous behavior. Some other experimental data indicate that there are in the brain inhibitory neural pathways tonically inhibiting those structures underlying sexual behavior. Indeed, bilateral ablation of the pyriform cortex located in the base of the temporal lobe produces hypersexuality in the male cat which leads it to copulate not only with other animals of the same species and opposite sex, but also with other male cats and with animals of different species such as dogs, monkeys, chickens, etc. (96, 100). In agreement with the above-mentioned experimental data, states of hypersexuality have been observed in humans after bilateral ablation of the tip of the temporal lobe. As reported originally by Klüver and Bucy (67) similar disturbances of sexual behavior were observed in the macaque after bilateral ablation of the tip of the temporal lobe. The corresponding excitatory and inhibitory neural systems participating in sexual behavior may be termed "erogenic" and "anti-erogenic" systems respectively. The erogenic system can be activated not only by sensory impulses of various modalities, but also by the corresponding gonadal hormones.

A brilliant series of experimental studies carried out by Olds (86) utilizing the technique of self brain stimulation has established the localization along the limbic midbrain circuit of two neural systems functionally opposed, the stimulation of which produces a tendency to repetition or avoidance of the behavior immediately preceding such stimulation. These systems have been designated respectively Positive and Negative Reinforcing Systems. The antropomorphic interpretation that stimulation of the Positive Reinforcing System produces pleasure feelings whereas stimulation of the Negative Reinforcing System elicits unpleasant sensations has received support from experimental studies in humans. Indeed, Heath (34) has demonstrated that electrical or acetylcholinic stimulation of the septum in unanesthetized patients yields pleasant sensations. It is conceivable that human motivations of higher order reached through complex learning processes usually involving second or higher order types of reward (such as money, prestige, social status, etc.) may encompass both the primitive limbic structures plus phylogenetically newer neocortical circuits.

The anatomic substrate of emotional behavior has also been located in the limbic system. The pioneer studies of Hess (57) in cats with electrodes permanently implanted in their brains have demonstrated that it is possible to produce all the objective manifestations of rage or fear by electrical stimulation of a localized region in the posterior hypothalamus. These results have been repeatedly confirmed and extended by other authors (26, 69). Behavioral manifestations of rage or fear have also been elicited in the cat and monkey by electrical stimulation of suprahypothalamic limbic structures such as the amygdala and the septum. Recent experiments carried out in our laboratories with the method of localized chemical stimulation of the brain introducing microcrystals of various substances through cannulae permanently implanted have demonstrated the existence of a septo-periaqueductal pathway where acetylcholinic stimulation elicited rage behavior (50). On the other hand, local application of noradrenaline in the preoptic region elicited an "escape response" which is known to be normally associated with fear (Hernández-Peón and Chávez-Ibarra (45)). Similarly to what has been observed on motivational behavior, localized lesions in the brain are capable of diminishing the threshold of emotional behavior. For instance, a localized lesion in the fornical

area might produce an enhancement of rage behavior. Likewise, a lesion in the ventromedial area of the hypothalamus transforms a tame animal into an aggressive one.

Which is the evidence for waking inhibition of neocortical and limbic neurons? In his original description of the human EEG during relaxation and alertness, Berger (9) interpreted the now classical "EEG arousal reaction" as the result of cortical inhibition. Although in their earlier work Adrian and Matthews (3) proposed that the EEG patterns characterized by high voltage slow waves and by low voltage fast activity translated synchronized and desynchronized neuronal discharges respectively, on the basis of later studies on the olfactory bulb Adrian (2) stated "the break up of the rhythm seems to involve a decrease in the activity of the cells, and it is not merely that their activity is no longer synchronized." Recent microelectrode studies on the cerebral cortex of the cat have supported Berger's early insight. In acute preparations, Whitlock *et al.* (106) observed that the EEG arousal reaction is associated with an arrest of unit activity in the motor cortex. Later, when microelectrode recordings were made in chronic intact animals, it was confirmed that the discharge of many cortical neurons diminished during alertness (91) and increased during physiologic sleep (59). Similar findings have been reported by Evarts (22–24) who has additionally observed that the activity of some cortical units becomes maximally enhanced with a different pattern of discharge during RS. The results of experiments utilizing the technique of evoked potentials also substantiate the view of greater cortical excitability during sleep than during wakefulness. Fleming *et al.* (28) showed that the cortical visual evoked potentials and their recovery rate increase during sleep. In a study of thalamocortical sensory transmission in the somatic pathway of unrestrained cats, Allison (6) found that the excitability of the cortical receiving area is higher during RS than during SS, and lowest during alert wakefulness. These findings have been confirmed by other investigators particularly by the group of Rossi in Genoa (93, 94), and by Cordeau *et al.* in Canada (18, 19).

All the cited experimental evidence is incompatible with the Pavlovian hypothesis which considers sleep as resulting from cortical inhibition, and warrants a revision of the classical "activating view" postulating that wakefulness and arousal are associated with an excitatory cortical process (73).

There is also evidence suggesting the existence of inhibitory influences arising from the mesodiencephalic Vigilance System and acting upon limbic structures. In cats with electrodes permanently implanted, amygdaloid and hippocampal after-discharges elicited by localized electrical stimulation were significantly reduced when the animal was alerted either by sensory stimuli, or by direct electrical stimulation of the mesencephalic reticular formation. Accordingly, amygdaloid potentials evoked by single shocks applied 2 mm away from the recording site were also reduced during alertness elicited either by environmental stimuli, or by electrical stimulation of the posterior hypothalamus (40).

All the above mentioned findings have led me to the following unorthodox view concerning the general pattern of activity in the waking brain. As illustrated in Figure 7, widespread inhibitory and facilitatory influences arising from the mesodiencephalic Vigilance System regulate the excitability of most of the central nervous system. Although continuously shifting in space and degree, quantitatively, reticulofugal inhibition appears to be more extensive than facilitation (41, 42). The latter process would be restricted only to those neurons of the various circuits necessary at any given moment for a particular

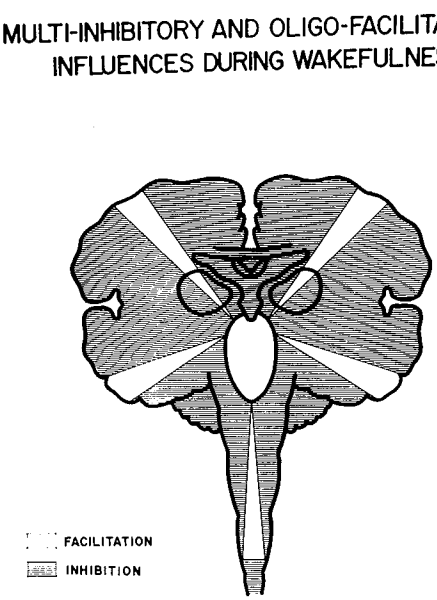

FIG. 7. Diagrammatic representation of the extensive inhibitory influences and the selective facilitatory influences arising from the rostral part of the brain stem and acting at all the levels of the central nervous system during wakefulness.

physiologic situation, while inhibition would prevent possible chaotic interference by impulses converging on the selected pathways.

NEURAL EVENTS UNDERLYING DREAMING

In addition to the increase of activity within the Sleep System, dreaming certainly involves a sequence of events in many other parts of the brain. The main neural mechanisms underlying dreaming may have the following sequence: an increased activity of the Sleep System results in progressive inhibition of the Vigilance System. When the level of deep sleep is attained, the Dream System represented by the structures involved in recent memory are released, increasing the discharge of the plastic neurons with memory tracings. By the same token, the neurons belonging to the Remote Memory System and to the Emotional and Motivational Systems are also disinhibited. The released memory tracings of the Recent and Remote Memory Systems would give rise to what Freud (29) designated as *manifest content* of dreams, whereas the flow of impulses from the Emotional and Motivational Systems would provide certain direction to the apparently disorganized memory tracings, thus underlying the so-called *latent content* of dreams (Figure 8). It should not be surprising that a large proportion of dreams have a sexual latent content since it is known that, at least among the basic motivational systems (for hunger, thirst and sexual appetite), the Motivational System most inhibited during ordinary wakefulness because of the human social organization is that concerned with sexual behavior.

SENSORY FILTERING DURING DREAMING

How does the brain handle the arrival of sensory signals during sleep, and partic-

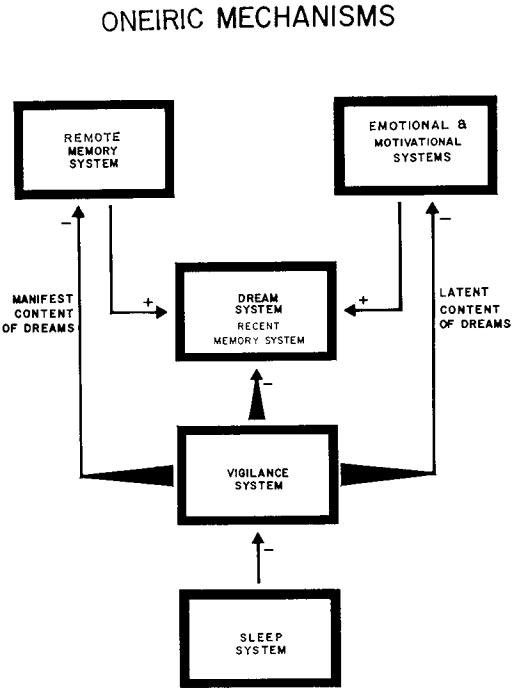

FIG. 8. This diagram summarizes the neurophysiologic hypothesis proposed for accounting some psychoanalytic observations on dreams. Release of inhibition acting upon the memory systems accounts for the manifest content of dreams, and release of inhibition acting upon the emotional and motivational systems accounts for the latent content of dreams.

ularly during the periods of REM which correspond to dreaming?

In previous papers with several collaborators, we have shown that there are important reticulofugal influences arising from the central core of the brain stem, particularly at the midbrain level, which regulate by inhibition and facilitation the entrance of sensory signals to the central nervous system (35, 36, 37).

The starting point of this investigation was the finding that electrical stimulation of the midbrain reticular formation produces a synaptic blockade of afferent impulses recorded at the first synapse of the somatic pathways (31, 48). Figure 9 illustrates an experiment in which single shocks applied to the dorsal columns of the spinal cord elicited afferent volleys recorded at the gracilis nucleus. Electrical stimulation of the midbrain reticular formation depressed the postsynaptic wave of the evoked potential without altering the presynaptic spike (55). In recent microelectrode studies of the cuneate nucleus, presynaptic inhibition has also been demonstrated (7).

Other experiments showed that this inhibitory descending influence arising from the midbrain reticular formation is tonically acting during wakefulness because the potentials recorded at the spinal V sensory nucleus (which represents the first synapse of the trigeminal pathway) were significantly increased after a lesion was made in the midbrain tegmentum (35, 98). Moreover, it has been demonstrated that this descending influence does not arise in supramesencephalic structures because in decerebrate preparations with a transection at the intercollicular level removing all the brain in front of the mesencephalon, the potentials evoked by tactile stimuli applied to the skin, and recorded from the spinothalamic tract in the lateral column of the spinal cord, were remarkably enhanced after a high spinal transection (46).

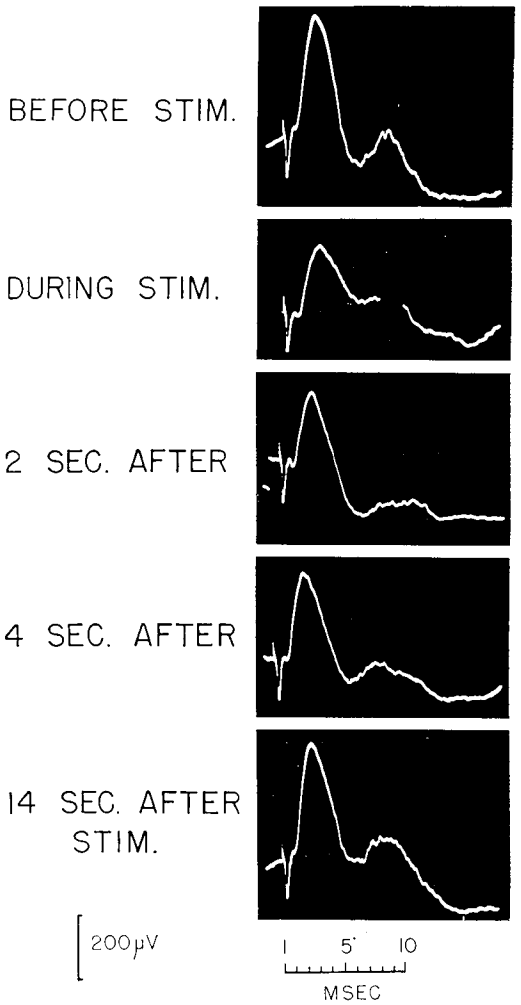

FIG. 9. Potentials recorded from the gracilis nucleus in the cat and evoked by single shocks applied to the dorsal columns. The first downward spike represents presynaptic impulses and the following wave represents postsynaptic impulses arising the nuclear neurons. Notice that during electrical stimulation of the mesencephalic reticular formation (three seconds) the postsynaptic wave was significantly reduced without modification of the presynaptic spike. Several seconds elapsed before the nuclear wave recovered its original magnitude (from Hernández-Peón, Scherrer and Velasco (55)).

There is experimental evidence indicating the physiologic participation of the above mentioned sensory inhibition during attention. Figure 10 illustrates an experiment in a cat with indwelling elec-

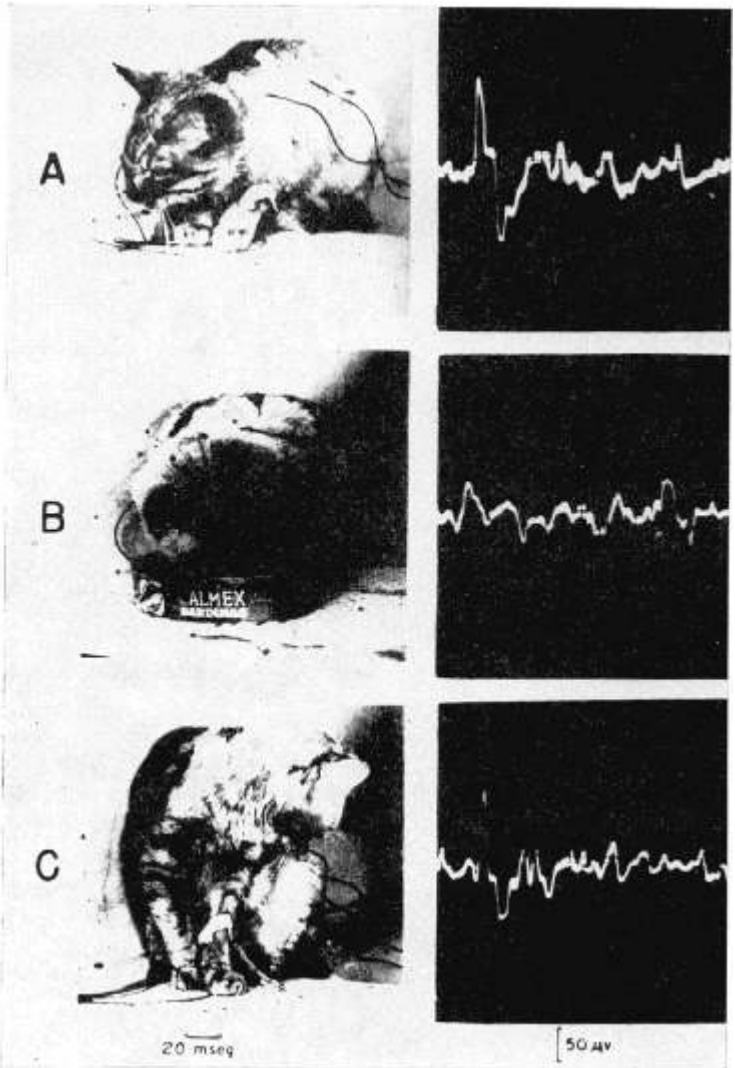

FIG. 10. Blocking of tactile potentials recorded from the spino-thalamic tract in the spinal cord of a cat while the animal was eagerly eating sardines (from Hernández-Peón, R., and Brust-Carmona, H. (47)).

trodes implanted in the spinothalamic tract at the spinal cord for recording tactile evoked potentials. When the cat was relaxed, evoked potentials of a stable magnitude were recorded. However, when the cat was very attentive eagerly eating sardines, the tactile spinal potentials were practically absent. A few seconds later, during postprandial relaxation, the potentials reappeared again (47).

From all those studies, we have reached the conclusion that there is a filtering mechanism which selects the entrance of sensory signals to the Central Nervous System at least at the level of the first synapse (37, 39, 42), and possibly also at the level of some receptors. The essential part of this filtering mechanism appears to be located in the mesencephalic tegmentum. Since this is a region of convergence not only for plurimodal sensory impulses but also for impulses arising in

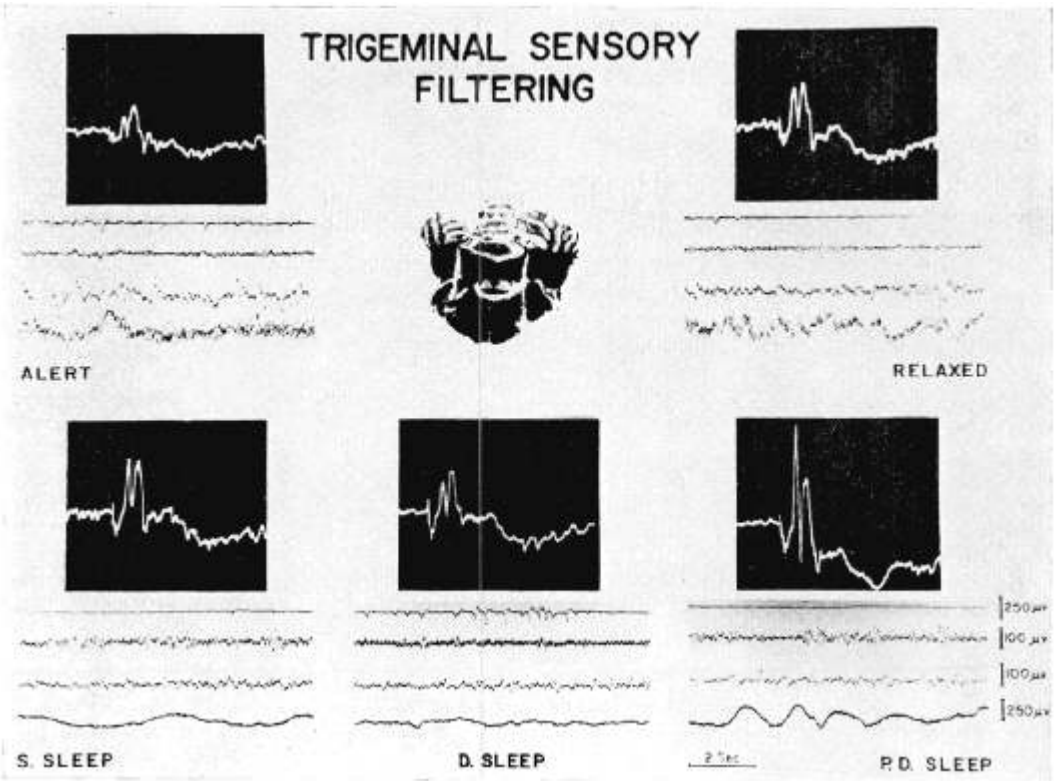

FIG. 11. Tactile evoked potentials recorded in the cat from the spinal trigeminal sensory nucleus during wakefulness and sleep. Simultaneous recordings were made of the eye movements, the electrical activity of the neocortex, of the entorhinal cortex, and the EMG of the neck muscles. Notice that the trigeminal potentials became significantly reduced during alertness and during RS, whereas the potentials became enhanced during SS. This experiment shows a rebound effect of the trigeminal evoked potentials immediately after the termination of RS (from Hernández-Peón, O'Flaherty and Mazzuchelli-O'Flaherty (51)).

the neocortex and in limbic structures, it follows that the regulation of sensory inflow is importantly linked with the central mechanisms involved in instinctive and intellectual functions (36).

After these introductory remarks we can return to the problem about the functional role of the sensory filtering mechanism during sleep. With this aim, we have studied sensory transmission in cats at the first trigeminal sensory relay during sleep (51). Electrodes were implanted in the spinal Vth sensory nucleus, and potentials evoked by tactile stimuli applied to the skin of the face were recorded. After confirming the significant reduction of these potentials during alertness, it was found that they were enhanced during SS. But what is particularly interesting is that the potentials became reduced again during the stage of desynchronized EEG. Sometimes, at the end of these periods, there was an increase of the potentials which reminds a post-inhibitory rebound phenomenon (Figure 11). Figure 12 shows the average variations of the tactile evoked potentials recorded at the trigeminal sensory nucleus in 67 experiments. A significant increase of the potentials during SS and a significant decrease of the potentials during RS is evident. Presynaptic inhibition appears to play an important

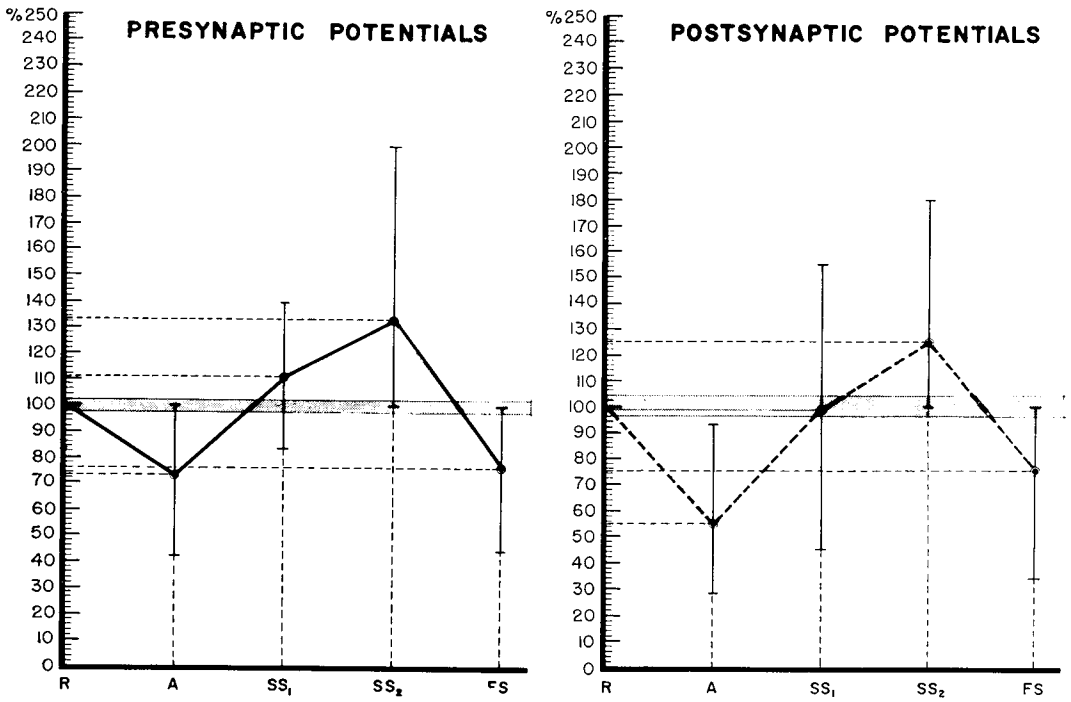

FIG. 12. These curves represent the average of the amplitude of the presynaptic and postsynaptic components of nuclear tactile trigeminal potentials recorded from 67 experiments. The vertical lines represent the ranges of variation. R, relaxed wakefulness; A, alertness; SS_1, first stage of SS; SS_2, second stage of SS; FS, fast or rapid sleep.

role in this phenomenon. The above mentioned experimental results indicate that during RS which is commonly associated with dreaming in the human, the entrance of sensory signals to the central nervous system is partially blocked to a similar degree as it occurs during attentive behavior.

Further experiments in which lesions were made in the midbrain tegmentum have demonstrated that this region is essential for the sensory blockade detected at the first synapse of the trigeminal pathway both during alertness and RS (Figure 13). On the other hand, electrical stimulation of the limbic midbrain hypnogenic pathway did not affect sensory transmission at this level. Since the reticular neurons belonging to the Vigilance System are seemingly inhibited during RS as indicated by a marked elevation of the arousal threshold at this moment (measured by direct electrical stimulation of the mesencephalic reticular formation), it is evident that the inhibitory neurons responsible for "sensory filtering" are not directly linked neither with the arousing nor with the hypnogenic neurons. It seems more logical to postulate that those inhibitory neurons are associated with a different neural system. Because the elements of this system appear to be maximally active during alertness as well as during "dream sleep," they would be independent of the arousing neurons, although anatomically interspersed with them in the rostral brain stem. The paradoxic finding that during RS the activity of some reticular units in the midbrain decreases while the discharge of some other elements is enhanced (60), falls in line with this interpretation. What is the functional role of this newly postulated brain stem system? A common outstanding function oc-

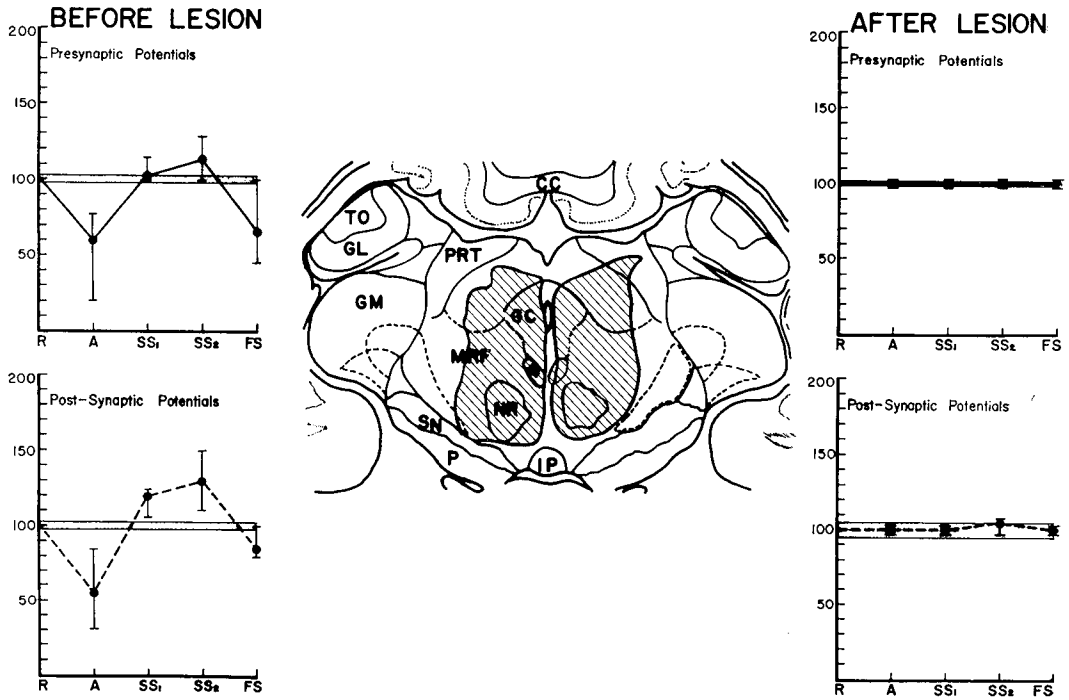

FIG. 13. This figure shows the absence of the changes of the trigeminal evoked potentials obtained after a tegmental lesion in the midbrain. Each graph represents the average of five consecutive experiments in the same cat before and after the lesion. R, relaxed wakefulness; A, alertness; SS_1, first part of slow sleep; SS_2, second part of slow sleep; FS, fast or rapid sleep with desynchronized EEG.

curring during attentive wakefulness and dreaming is that which Fessard (27) termed "experienced integration." Therefore, it is not ilogical to assume that the neural system linked with the sensory filtering mechanism might be involved in the formation of conscious experiences.

The operation of "sensory filtering" associated with the "Conscious Experience System" is illustrated in Figure 14. The hatched area represents the "Vigilance system" is the mesodiencephalic region; the Dream System is located in the hippocampal formation; the Sleep System is represented by the ascending and descending arrows; and finally, the "Conscious Experience System" corresponds to the stippled area overlapping the Vigilance System. During wakefulness, the described descending tonic inhibition filters the entrance of certain sensory signals at the first synapse. In addition, there is an ascending tonic inhibition acting upon the neocortex, and upon the hippocampal formation. During SS, the first synapse is disinhibited, permitting the admittance of more sensory signals into the central nervous system. The excitability of the neocortex is also increased, facilitating the activation of cortical neurons by the arriving afferent impulses. At the same time, the Recent Memory System is partially released becoming more active. During the periods of RS resulting from a higher degree of activity within the Sleep System, the excitability of the neocortex and of the hippocampal formation is maximally heightened because of an almost complete release of the tonic inhibition acting upon those structures. An increased neuronal discharge may then flow from the specific receiving cortical areas to the

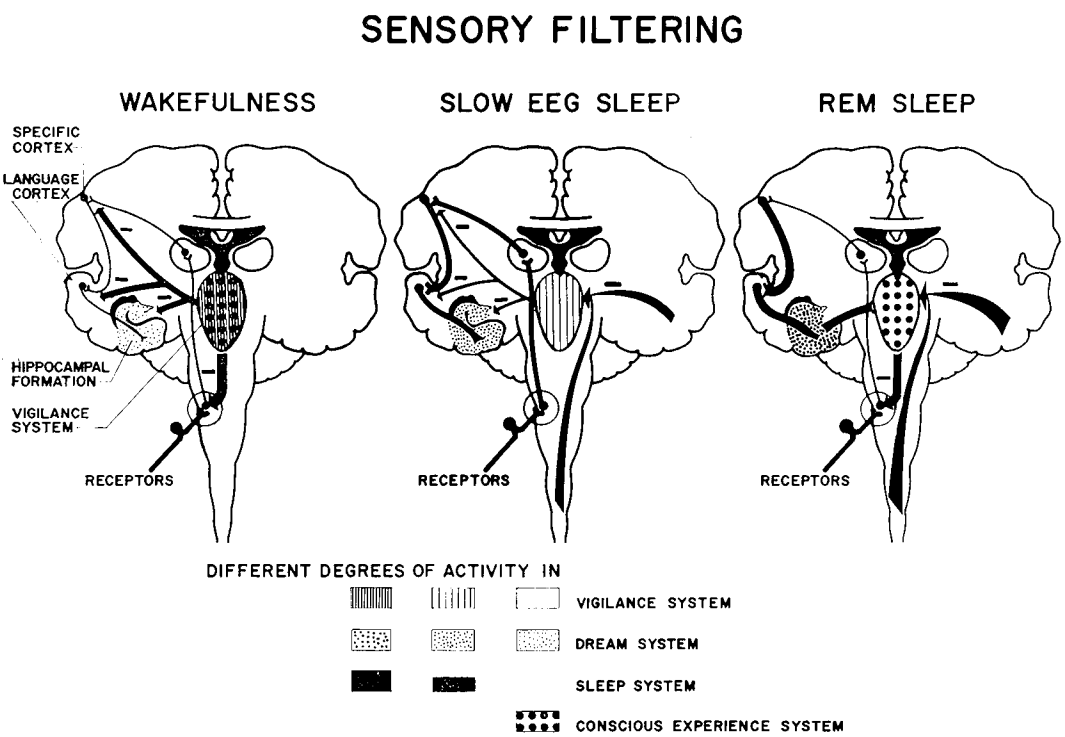

FIG. 14. This diagram summarizes the proposed sequence of events occurring during the transition from wakefulness to SS and to RS. The hatched area represents the vigilance system with different degrees of activity. The sleep system is represented by the arrows terminating upon the vigilance system. Activation of the conscious experience system during wakefulness of REM sleep is represented by the dots overlapping with the vigilance system. The ascending reticulocortical and the descending inhibitory influences acting at the first synapse of the specific afferent pathways are represented by the arrows with the minus sign.

areas involved in verbal communication, and also to the hippocampal formation. From there, an avalanche of apparently disordered impulses would flow to the "Conscious Experience System" in the brain stem resulting in the conscious experiences known as dreams. This sequence of events can account for dreams triggered by words which activate neurons storing related past experiences. Oswald (87) reported experiments by Berger in which single spoken names were repeatedly presented during each REM period of sleep without causing awakening. It was found that the sleeper incorporated the corresponding acoustic stimuli into the dream life in modified form, usually by assonant connection, e.g., a stimulus Jenny provoked a dream of opening a safe with a jemmy. Finally, at the onset of the oneiric activity, sensory inhibition acts again to restrict, at least partially, sensory inflow to the brain. In this way, there are less chances for the neural activity underlying dreaming to be interrupted by irrelevant sensory bombardments.

COMMON NEURAL MECHANISMS OF SENSORY PERCEPTION, DREAMING AND HALLUCINATIONS

The assumption that sensory perception, dreaming and hallucinations involve some common brain mechanisms has been repeatedly expressed in the past (61, 62, 99). According to the present model, the signals arising in sensory receptors ascend to the neocortex, and only after being amplified and analyzed there, the relayed

activity descends to the Conscious Experience System located in the brain stem in order to give rise to conscious sensory perception. Simultaneously with this process, the sensory impulses activate the Emotional and Motivational Systems from where a flow of impulses are projected down to the Conscious Experience System, thus providing the emotional and motivational tones of our sensory perception (Figure 15).

Dreaming also involves the flow of coded information into the Conscious Experience System from the same storage and driving neurons participating in waking perceptions. The main difference between the neural processes underlying perception and dreaming would be a heightened cortical and limbic excitability in the latter state which increases the probability of association between a great number of memory tracings.

A basic assumption in this model is that there is a tonic inhibition arising from the Vigilance System and acting upon the Memory Systems. This inhibition is maximal during wakefulness, and appears necessary for the selection of a limited amount of information to be integrated into sensory perceptions. As represented in Figure 16, failure of the inhibitory influence during wakefulness may account for the abnormal conscious experiences known as hallucinations. This influence might be eliminated or interrupted:

a) By lesions in the brain stem, as it happens in cases of peduncular hallucinosis described by Lhermitte (68) and in the Syndrome of basilar insufficiency (107).

b) By lesions in some part of the circuit connected with the hippocampal formation, as it has been shown to occur in some cases of Korsakoff's psychosis.

c) By certain pharmacologic agents, such as hallucinogenic drugs (LSD, psylocybin). Recent experimental results obtained by Adey and his collaborators (1, 17) support this hypothesis. Indeed, they have found in cats and humans an increase of hippocampal activity after administration of LSD.

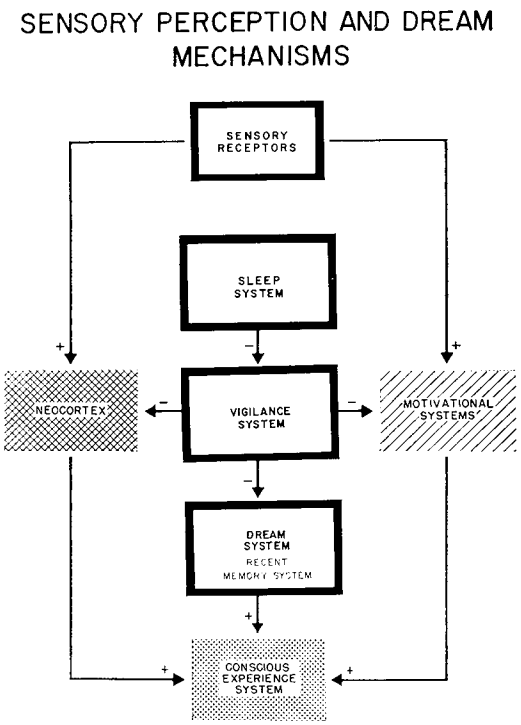

FIG. 15. Diagramatic representation of the main neural mechanisms involved in sensory perception and dreaming. The impulses arising in sensory receptors activate the neocortical, emotional and motivational systems before reaching the conscious experience system in order to give rise to sensory perception. The impulses released from the dream system give rise to the experience of dreams when they reach the conscious experience system.

d) By decreased sensory bombardment of the central nervous system, as it occurs during prolonged sensory deprivation (101). It is likely that the cumulative development of this and other types of inhibition formerly discussed is conditioned by constant sensory stimulation of the brain. According to this view, afferent impulses arising from the sense organs would activate important inhibitory neurons linked at one end with the vigilance neurons essential for wakefulness, and at the other, with a great functional variety of elements including those with stored memory tracings.

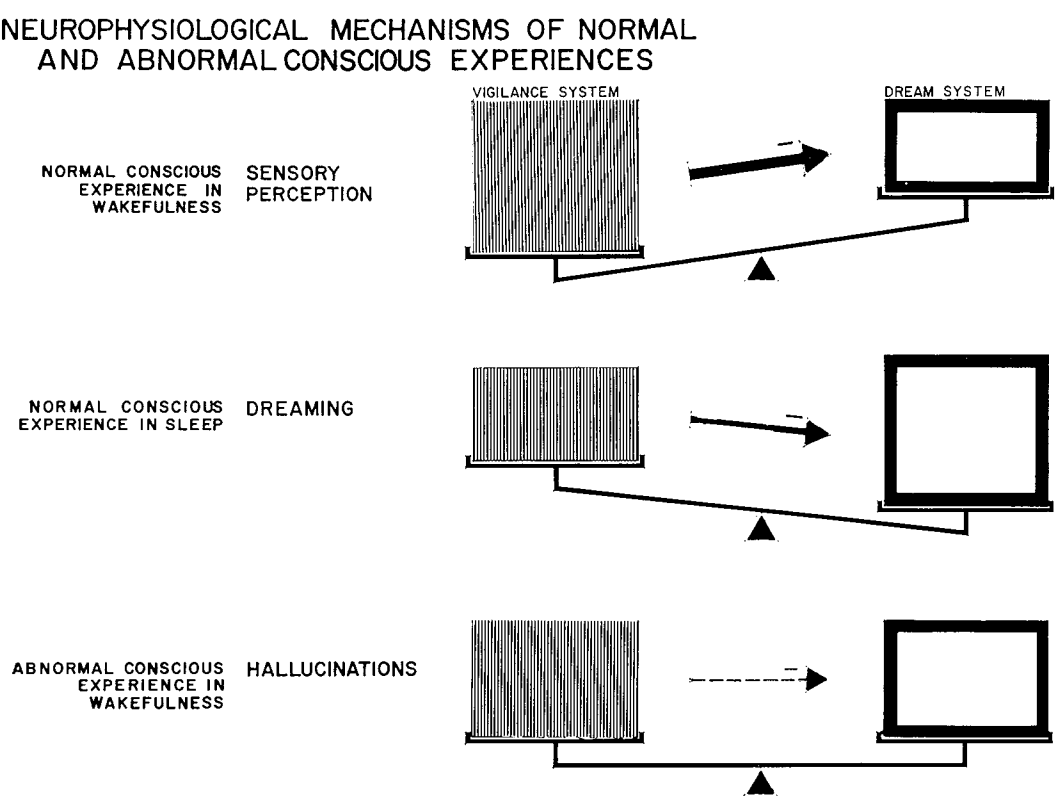

FIG. 16. Oversimplified representation of the degree of activity of the inhibitory neurons linked at one end with the vigilance system, and at the other end with the dream or recent memory system. Hallucinations are accounted for by a deficiency of that inhibitory influence.

e) By fatigue or post-excitatory refractoriness of the inhibitory neurons consecutive to prolonged activation of the vigilance neurons as it occurs during sleep deprivation.

BIOLOGICAL SIGNIFICANCE OF DREAMING

Wondering about the biologic significance of dreaming inevitably leads to philosophical and teleologic reasonings. It is natural that different views might be proposed according to the central interest, background information and intellectual perspectives of the thinkers.

Abundant clinical psychoanalytical observations indicate that the content of dreams is constituted by apparently disorganized fragments of individual past experiences often associated to disguised motivations. The reorganization of those memory tracings occurring during dreaming has been thought to serve as a protective device which prevents the access to consciousness of those motivations submerged into the unconscious by censorship. According to Freud (29) dreams "serve the purpose of prolonging sleep instead of waking up. Dreams are the guardians of sleep and not its disturbers." Although this point of view is debatable and has not gained general acceptance (104), the participation of motivational factors in dreams has a better clinical foundation. Most psychotherapists would agree with Ullman's statement that dreams have a "remarkable capacity to integrate past experiential data with current life situations in a manner that discloses more significant information pertaining to

current conflicts than is available to the individual at any given moment in the waking state" (103).

Besides the motivational factors involved in dreams, the release of stored information during dreaming might have a cognitive function. Recent data provided by electrophysiologic recordings during sleep in humans and animals lend support to the long held layman's idea that lower mammals such as dogs and cats may also have dream-like experiences at certain periods of sleep. Admitting that dreams are elaborated with memory tracings of past individual experiences, logically there must be a strict parallelism between sensory modality dominances and degrees of sensory organization occurring in the individual worlds of dreams and reality in different animal species. Vision has reached greatest development in the human, and, therefore, it is not surprising that visual imageries prevail in our waking and dreaming worlds. In blindborn persons, in whom telereceptor dominance has been transferred from the visual to the auditory system, their dreams and pharmacologically induced hallucinations (personal observations) mainly involve auditory experiences. It is conceivable that macrosomatic animals might have dominant olfactory experiences in waking and dreaming states. If dreaming is a privilege not limited to the highly evolved *homo sapiens,* but is a process common to other species of the animal scale, the difficulty in establishing its phylogenetic origin is comparable to that of defining the phylogenetic origin of conscious experience.

At any rate, it is obvious that current psychoanalytical interpretations cannot be extended to subhuman animal species. One then is confronted with an evolutionary physiologic model as the most plausible at the present time. According to the neurophysiologic mechanisms here proposed, a great deal of memory tracings normally inhibited during wakefulness become accessible to the Conscious Experience System during dreaming. This seems to be the result of transient inactivity of the corresponding inhibitory neurons probably related to their metabolic recovery. Following this view, dreams would be an effect or epiphenomenon associated with the recovery process of mnemic inhibitory neurons essential for normal waking behavior and logical thinking. But does this mean that the act of dreaming is of no value for the organism? In this, as in many other examples, there is no logical incompatibility between physiologic and teleologic viewpoints. Part of the material forming the manifest content of dreams is undoubtedly represented by the so-called day's residues or recent residues usually originated from trivial experiences which the individual is not aware of. It is tempting to speculate further that during dreaming the Conscious Experience System processes some of that information which has been stored without awareness, so that the most relevant items can be selected by a variety of types of associations with memory tracings consciously available, and thus be utilized for future adaptive behavior.

If this hypothesis is correct, it may explain many examples of ideational problem solving occurring during dreaming. It is not unreasonable to assume that a great deal of unconscious associations occurring during wakefulness and sleep may lie at the basis of creative thinking processes.

SUMMARY

Most of the authors who have dealt with this subject have accepted the similarity between oneiric and hallucinatory phenomena. Moreover, similar anatomic substrates and neural mechanisms have been assumed. In this paper, a common neurophysiologic model is proposed which accounts for all the commonly recognized features of dreams and hallucinations. This model assumes the existence of a Dream

System identifiable with the brain structures involved in recent memory, and subject to tonic inhibition by the Vigilance System during wakefulness. Similar inhibitory influences operate upon the neurons storing remote memories and upon the limbic structures involved in emotions and motivations. When the Vigilance System is inhibited by the Sleep System responsible for the onset and maintenance of sleep, the activity of the neurons with memory tracings and of the Emotional and Motivational Systems is released. While the neural discharges carrying stored information underly the manifest content of dreams, the activity from the latter limbic structures underly the latent content of dreams. Dreaming is initiated when the flow of all those impulses arrive to a highly specialized neural system located in the rostral brain stem which has been termed "Conscious Experience System." This system, where "experienced integration" takes place, would be independent of the overlapping Vigilance System. It becomes active during wakefulness and during "desynchronized sleep" commonly associated with dreaming, whereas it would be inactive during "synchronized sleep." During released activation of the neural circuits underlying dreaming, the "sensory filtering" mechanism partially blocking the entrance of afferent signals to the CNS becomes as active as during attentive wakefulness.

Hallucinations are accounted, at least in part, by a failure of the inhibitory influences acting upon the Memory Systems during wakefulness. It is suggested that the cumulative development of that and other types of central inhibition is related to continuous sensory bombardment of the brain.

An evolutionary theory of dreams is proposed in which sensory dominance in waking and dreaming states runs in parallel with the development of teleceptive sensory systems. Dreams are considered as an epiphenomenon related to metabolic recovery of mnemic inhibitory neurons, the activity of which is essential for normal waking behavior and logical thinking. A cognitive function of dreams is suggested. According to this hypothesis, information stored in the brain without awareness may be processed during dreaming by the integrating Conscious Experience System allowing associations with memory tracings consciously available, and thus be utilized in adaptive waking behavior.

REFERENCES

1. Adey, W. R., Bell, F. R. and Dennis, B. J. Effects of LSD-25, psilocybin on temporal lobe EEG patterns and learned behavior in the cat. Neurology, *12:* 591–602, 1962.
2. Adrian, E. D. The control of nerve-cell activity. In *Symposia of the Society for Experimental Biology. IV. Physiological Mechanisms in Animal Behavior.* Academic Press, New York, 1950.
3. Adrian, E. D. and Matthews, B. H. C. The interpretation of potential waves in the cortex. J. Physiol., *81:* 440–471, 1934.
4. Akert, K., Koella, W. P. and Hess, R., Jr. Sleep produced by electrical stimulation of the thalamus. Amer. J. Physiol., *168:* 260–267, 1952.
5. Akimoto, H., Yamaguchi, N., Okabe, K., Nakagawa, T., Nakamura, I., Abe, K., Torii, H. and Masahashi, K. On the sleep induced through electrical stimulation on dog thalamus. Folia Psychiat. Neurol. Jap., *10:* 117–146, 1956.
6. Allison, T. Cortical and subcortical evoked responses to central stimuli during wakefulness and sleep. Electroenceph. Clin. Neurophysiol., *18:* 131–139, 1965.
7. Andersen, P., Eccles, J. C., Schmidt, R. F. and Yokota, T. Depolarization of presynaptic fibers in the cuneate nucleus. J. Neurophysiol., *27:* 92–106, 1964.
8. Batini, C., Moruzzi, G., Palestini, M., Rossi, G. F. and Zanchetti A. Persistent patterns of wakefulness in the pretrigeminal midpontine preparation. Science, *128:* 30–32, 1958.
9. Berger, H. Über das Electrokephalogram des Menschen. Arch. Psychiat., *87:* 527–570, 1929.
10. Berlucchi, G., Maffei, L., Moruzzi, G. and Strata, P. Mecanismes hypnogenes de tronc de l'éncéphale antagonistes de systeme réticulaire activateur. In M. Jouvet, ed., *Neurophysiologie des États de Sommeil,* pp. 89–105. Centre National de la Recherche Scientifique, Paris, 1965.

11. Bonvallet, M. and Bloch, V. Bulbar control of cortical arousal. Science, *133:* 1133–1134, 1961.
12. Bonvallet, M. and Allen, M. B. Prolonged spontaneous and evoked reticular activation following discrete bulbar lesions. Electroenceph. Clin. Neurophysiol., *15:* 969–988, 1963.
13. Brobeck, J. R. Regulation of feeding and drinking. In J. Field, H. W. Magoun and V. E. Hall, eds., *Handbook of Physiology: Neurophysiology, Vol. II,* pp. 1197–1206. American Physiological Society, Washington, 1960.
14. Buchwald, N. A., Wyers, E. J., Okuma, T. and Heuser, G. The "caudate-spindle." I. Electrophysiological properties. Electroenceph. Clin. Neurophysiol., *13:* 509–518, 1961.
15. Buchwald, N. A., Wyers, E. J., Lauprecht, C. W. and Heuser, G. The "caudate-spindle." IV. A behavioral index of caudate-induced inhibition. Electroenceph. Clin. Neurophysiol., *13:* 531–537, 1961.
16. Carli, C., Armengol, V. and Zanchetti, A. Electroencephalographic desynchronization during deep sleep after destruction of midbrain-limbic pathways in the cat. Science, *140:* 677–679, 1963.
17. Chapman, L. F., Walter, R., Adey, W. R., Crandal, O. H., Rand, W. R., Brazier, M. A. B. and Markhan, C. H. Altered electrical activity of human hippocampus and amygdala induced by LSD-25. Cited in *Current Research on Sleep and Dreams.* Public Health Service Pub. No. 1389, pp. 46, 1965.
18. Cordeau, J. P., Walsh, J. and Mahut, H. Sensory transmission during various stages of sleep and wakefulness. Electroenceph. Clin. Neurophysiol., *17:* 442, 1964.
19. Cordeau, J. P., Walsh, J. et Mahut, H. Variations dans la transmission des messages sensoriels en fonction des différents états d'éveil et de sommeil. In M. Jouvet, ed., *Neurophysiologie des États de Sommeil,* pp. 477–507. Centre National de Recherche Scientifique, Paris, 1965.
20. Cornford, F. M. *Plato's Cosmology.* Humanities Press, New York, 1952.
21. Dement, W. and Kleitman, N. The relation of eye movements during sleep to dream activity: an objective method for the study of dreaming. J. Exp. Psychol., *53:* 339–346, 1957.
22. Evarts, W. V. Neuronal activity in sensorimotor cortex during sleep and waking. Fed Proc., *22:* 637, 1963.
23. Evarts, E. V. Temporal patterns of pyramidal tract neurons during sleep and waking in the monkey. Electroenceph. Clin. Neurophysiol., *17:* 443, 1964.
24. Evarts, E. V. Neuronal activity in visual and motor cortex during sleep and waking. In M. Jouvet, ed., *Neurophysiologie des États de Sommeil,* pp. 189–212. Centre National de Recherche Scientifique, Paris, 1965.
25. Favale, E., Loeb, C., Rossi, G. F. and Sacco, G. EEG synchronization and behavioral signs of sleep following low frequency stimulation of the brain stem reticular formation. Arch. Ital. Biol., *99:* 1–22, 1961.
26. Fernandez-de Molina, A. and Hunsperger, R. Organization of the subcortical system governing defence and flight reactions in the cat. J. Physiol., *160:* 200–213, 1962.
27. Fessard, A. E. Mechanisms of nervous integration and conscious experience. In J. P. Delafresnaye, ed., *Brain Mechanisms and Consciousness,* pp. 200–236. Blackwell, Oxford, 1954.
28. Fleming, T. C., Huttenlocher, P. R. and Evarts, E. V. Effects of sleep and arousal on the cortical response to lateral geniculate stimulation. Fed. Proc., *18:* 46, 1959.
29. Freud, S. *Obras Completas.* Editorial Biblioteca Nueva, Madrid, 1948.
30. Glees, P. and Griffith, H. B. Bilateral destruction of the hippocampus (cornu Ammonis) in a case of dementia. Monatsschr. Psychiat. Neurol., *123:* 193–204, 1952.
31. Hagbarth, K-E. and Kerr, D. I. B. Central influences on spinal afferent conduction. J. Neurophysiol., *18:* 388–411, 1954.
32. Harris, G. W., Michael, R. P. and Scott, P. P. Neurological site of action of stilboestrol in eliciting sexual behavior. In G. E. W. Wolstenholme and C. M. O'Connor, eds., *Neurological Basis of Behavior,* pp. 236–254. Churchill, London, 1958.
33. Heath, R. G. and Hodes, R. Effects of stimulation with electrodes chronically implanted in rhesus monkeys. In R. Heath, ed., *Studies in Schizophrenia.* Harvard Univ. Press, Cambridge, 1954.
34. Heath, R. G. Pleasure response of human subjects to direct stimulation of the brain: physiologic and psychodynamic considerations. In R. G. Heath, Ed., *The Role of Pleasure in Behavior,* pp. 219–243. Hoeber, New York, 1964.
35. Hernández-Peón, R. Central mechanisms controlling conduction along central sensory pathways. Acta Neurol. Latinoamer., *1:* 255–264, 1955.
36. Hernández-Peón, R. Centrifugal control of sensory inflow to the brain and sensory perception. Acta Neurol. Latinoamer., *5:* 279–298, 1959.
37. Hernández-Peón, R. Reticular mechanisms of sensory control. In W. Rosenblith, ed., *Sensory Communication,* pp. 497–520. Wiley, New York, 1961.
38. Hernández-Peón, R. Sleep induced by localized electrical or chemical stimulation of the forebrain. Electroenceph. Clin. Neurophysiol., *14:* 423–424, 1962.
39. Hernández-Peón, R. Attention, sleep, motivation and behavior. In R. G. Heath, ed., *The*

Role of Pleasure in Behavior, pp. 195–217. Hoeber, New York, 1963.
40. Hernández-Peón, R. Psychiatric implications of neurophysiological research. Bull. Menninger Clinic, *28:* 165–185, 1964.
41. Hernández-Peón, R. Neurophysiological mechanisms of wakefulness and sleep. Acta Neurol. Latinoamer., *10:* 18–24, 1964.
42. Hernández-Peón, R. Neurophysiology of attention. *Year Book of Sciences and Technology,* pp. 283–285. Academic Press, New York, 1965.
43. Hernández-Peón, R. A cholinergic hypnogenic limbic forebrain-hindbrain circuit. In M. Jouvet, Ed., *Neurophysiologie des États de Sommeil,* pp. 63–88. C.N.R.S., Paris, 1965.
44. Hernández-Peón, R. Central neurohumoral transmission in sleep and wakefulness. In K. Akert and J. P. Schade, eds., *Progress in Brain Research: Sleep Mechanisms, Vol. 18,* pp. 96–117. Elsevier, Amsterdam, 1965.
45. Hernández-Peón, R. and G. Chavez-Ibarra. Sleep induced by localized electrical or chemical stimulation of the forebrain. In R. Hernández-Peón, ed., *The Physiological Basis of Mental Activity.* Electroenceph. Clin. Neurophysiol. Supp., *24:* 188–198, 1963.
46. Hernández-Peón, R. and Brust-Carmona, H. Functional role of subcortical structures in habituation and conditioning. In J. P. Delafresnaye, ed., *Brain Mechanisms and Learning,* pp. 393–408. Blackwell, Oxford, 1961.
47. Hernández-Peón, R. and Brust-Carmona, H. Inhibition of tactile and nociceptive spinal evoked potentials in the cat during distraction. Acta Neurol. Latinoamer., *7:* 289–294, 1961.
48. Hernández-Peón, R. and Hagbarth, K-E. Interaction between afferent and cortically induced reticular responses. J. Neurophysiol., *18:* 44–45, 1955.
49. Hernández-Peón, R., Chavez-Ibarra, G., Morgane, J. P. and Timo-Laria, C. Cholinergic pathways for sleep, alertness and rage in the limbic midbrain circuit. Acta Neurol. Latinoamer., *8:* 93–96, 1962.
50. Hernández-Peón, R., Chavez-Ibarra, G., Morgane, J. P. and Timo-Laria, C. Limbic cholinergic pathways involved in sleep and emotional behavior. Exper. Neurol., *8:* 93–111, 1963.
51. Hernández-Peón, R., O'Flaherty, J. J. and Mazzuchelli-O'Flaherty, A. L. Modifications of tactile evoked potentials at the spinal trigeminal sensory nucleus during wakefulness and sleep. Exper. Neurol., *13:* 40–57, 1965.
52. Hernández-Peón, R., O'Flaherty, J. J. and Mazzuchelli-O'Flaherty, A. L. Sleep and other behavioral effects induced by acetylcholinic stimulation of basal temporal cortex and striate structures. Brain Research, in press.
53. Hernández-Peón, R. Neural systems in the brain stem involved in wakefulness, sleep and conscious experience. Proc. Int. Cong. Neurol. Surg. (Copenhagen), in press.
54. Hernández-Peón, R. and Rojas-Ramirez, J. A. Cholinoceptive spinal hypnogenic neurons. In preparation.
55. Hernández-Peón, R., Scherrer, H. and Velasco, M. Central influences on afferent conduction in the somatic and visual pathways. Acta Neurol. Latinoamer., *2:* 8–22, 1956.
56. Hess, W. R. Le sommeil. Compt. Rend. Soc. Biol., *107:* 1333–1360, 1931.
57. Hess, W. R. *Das Zwishchenhirn: Snydrom, Lokalizationen, Funktionen.* Schwabe, Basel, 1949.
58. Hodes, R. Electrocortical desynchronization resulting from spinal block: evidence for synchronizing influences in the cervical cord. Arch. Ital. Biol., *102:* 183–196, 1964.
59. Hubel, D. H. Single unit activity in striate cortex of unrestrained cats. J. Physiol., *147:* 226–240, 1959.
60. Huttenlocher, P. R. Evoked and spontaneous activity in single units of medial brain stem during natural sleep and waking. J. Neurophysiol., *24:* 451–468, 1961.
61. Jackson, J. H. Selected writings of Hughlings Jackson. In J. Taylor, G. Holmes and F. M. R. Walshe, eds., *Vol. 2. Basic Books,* New York, 1958.
62. James, W. *Principles of Psychology, Vol. 2.* Dover, New York, 1950.
63. Jouvet, M. Recherches sur le structures nerveuses et les mecanismes responsables de differentes phases du sommeil physiologique. Arch. Ital. Biol., *100:* 125–206, 1962.
64. Jouvet, M. A study of neurophysiological mechanisms of dreaming. In R. Hernández-Peón, Ed., *The Physiological Basis of Mental Activity.* Electroenceph. Clin. Neurophysiol. *Suppl., 24:* 133–156, 1963.
65. Jouvet, M. Sleep in pontine cats: studies on the mechanisms of the rhombencephalic phase of sleep. Electroenceph. Clin. Neurophysiol., *17:* 446, 1964.
66. Jouvet, M. Etude de la dualité des états de sommeil et des mécanismes de la phase paradoxale. In M. Jouvet, ed., *Neurophysiologie des États de Sommeil,* pp. 397–449. C.N.R.S., Paris, 1965.
67. Kluver, H. and Bucy, P. C. Preliminary analysis of functions of the temporal lobes in monkeys. Arch. Neurol. Psychiat., *42:* 979–1000, 1939.
68. Lhermitte, J. *Les Hallucinations: Clinique et Physiopathologie.* Doin, Paris, 1951.
69. Mac Lean, P. D. and Delgado, J. M. R. Electrical and chemical stimulation of frontotemporal portion of limbic system in

the waking animal. Electroenceph. Clin. Neurophysiol., *5:* 91–100, 1953.
70. Mac Lean, P. D., Ploog, D. W. and Robinson, B. W. Circulatory effects of limbic stimulation, with special reference to the male genital organ. Physiol. Rev., *40:* 105–112, 1960.
71. Mac Lean, P. D., Robinson, B. W. and Ploog, D. W. Experiments on localization of genital function in the brain. Amer. Neurol. Ass., 105–109, 1959.
72. Mac Lean, P. D., Robinson, D. B. and Ploog, D. W. Experiments on localization of genital function in the brain. Electroenceph. Clin. Neurophysiol., *11,* 3: 617, 1959.
73. Magoun, H. W. *The Waking Brain.* Thomas, Springfield, Ill., 1963.
74. Manceau, A. and Jorda, M. Les centres mésodiencephaliques du sommeil. Sem. Hôp., *24:* 3193–3202, 1948.
75. Mazzuchelli-O'Flaherty, A. L., O'Flaherty, J. J. and Hernández-Peón, R. Sleep and other behavioral responses induced by acetylcholinic stimulation of frontal and mesial cortex. Brain Research, in press.
76. Milner, B. Psychological defect produced by temporal lobe excision. A.R.N.M.D., *36:* 244–257, 1958.
77. Monnier, M., Hosli, L. and Krupp, P. Moderating and activating systems in mediocentral thalamus and reticular formation. In R. Hernández-Peón, ed., *The Physiological Basis of Mental Activity.* Electroenceph. Clin. Neurophysiol. *Suppl., 24:* 97–112, 1963.
78. Monnier, M. Action de la stimulation électrique du centre somnogene sur l'électrocorticogramme chez le Chat. (Réactions hypniques et réactions d'éveil). Rev. Neurol. (Paris), *83:* 561–563, 1950.
79. Morgane, P. J. and Kosman, A. J. A rhinencephalic feeding center in the cat. Amer. J. Physiol., *197:* 158–162, 1959.
80. Moruzzi, G. Active processes in the brain stem during sleeping. *The Harvey Lectures, Series 58.* Academic, New York, 1963.
81. Moruzzi, G., Berlucchi, G., Maffei, L. and Strata, P. Hypnogenic brainstem mechanisms antagonistic to the reticular activating system. Electroenceph. Clin. Neurophysiol., *17:* 448, 1964.
82. Nauta, W. J. H. Hypothalamic regulation of sleep in rats. An experimental study. J. Neurophysiol., *1:* 285–316, 1946.
83. Nauta, W. J. H. Hippocampal projections and related neural pathways to the mid-brain in the cat. Brain, *81:* 319–340, 1958.
84. Nauta, W. J. H. Some neural pathways related to the limbic system. In E. R. Ramey and D. S. O'Doherty, eds., *Electrical Studies on the Unanesthetized Brain,* pp. 1–16. Hoeber, New York, 1960.
85. O'Flaherty, J. J., Mazzuchelli-O'Flaherty, A. L. and Hernández-Peón, R. Thalamic cholinergic areas involved in sleep and emotional behavior. In preparation.
86. Olds, J. Hypothalamic substrates of reward. Physiol. Rev., *42:* 553–604, 1962.
87. Oswald, I. Sleep and perception. Acta Psychol., *23:* 277–278, 1964.
88. Pampiglione, G. and Falconer, M. A. Electrical stimulation of the hippocampus in man. In Field, J., Magoun, H. W. and Hall, V. E., eds., *Handbook of Physiology: Neurophysiology, Vol. II,* pp. 1391–1394. American Physiological Society, Washington, 1960.
89. Parmeggiani, P. L. Sleep behavior elicited by electrical stimulation of cortical and subcortical structures in the cat. Helv. Physiol. Acta, *20:* 347–367, 1962.
90. Penfield, W. The excitable cortex in conscious man. In *The Sherrington Lectures,* pp. 1–43. Thomas, Springfield, Ill., 1958.
91. Ricci, G., Doane, B. and Jasper, H. Microelectrode studies of conditioning; technique and preliminary results. Acta Med. Belg., pp. 401–415, 1957.
92. Rossi, G. F., Minobe, K. and Candia, O. An experimental study of the hypnogenic mechanisms of the brain stem. Arch. Ital. Biol., *101:* 470–492, 1963.
93. Rossi, G. F., Palestini, M., Pisano, M. and Rosadini, G. An experimental study of the cortical reactivity during sleep and wakefulness. Electroenceph. Clin. Neurophysiol., *17:* 449, 1964.
94. Rossi, G. F., Palestini, M., Pisano, M. and Rosadini, G. An experimental study of the cortical reactivity during sleep and wakefulness. In M. Jouvet, ed., *Neurophysiologie des États de Sommeil,* pp. 509–532. C.N.R.S., Paris, 1965.
95. Russek, M. and Hernández-Peón, R. Olfactory bulb activity during sleep induced by stimulation of limbic structures. Acta Neurol. Latinoamer., *7:* 299–302, 1961.
96. Sawyer, C. H. Reproductive behavior. In J. Penfield, H. W. Magoun and V. E. Hall, eds., *Handbook of Physiology: Neurophysiology, Vol. II,* pp. 1225–1240. American Physiological Society, Washington, 1960.
97. Scoville, W. B. The limbic lobe in man. J. Neurosurg., *11:* 64–66, 1954.
98. Scherrer, H. and Hernández-Peón, R. Inhibitory influence of brain stem reticular formation upon synaptic transmission in trigeminal nucleus. Fed. Proc., *14:* 71, 1955.
99. Schilder, P. Experiments on imagination, after-images and hallucinations. Amer. J. Psychiat., *90:* 597–611, 1933.
100. Schreiner, L. and Kling, A. Behavioral changes following rhinencephalic injury in cat. J. Neurophysiol., *16:* 643–659, 1953.
101. Solomon, P., Kubransky, P., Leiderman, P. H., Mendelson, J. H., Trumbul, R. and Wexler,

D. *Sensory Deprivation.* Harvard Univ. Press, Cambridge, 1961.
102. Sterman, M. B. and Clemente, C. D. Forebrain inhibitory mechanisms: sleep patterns induced by basal forebrain stimulation in the behaving cat. Exp. Neurol., *6:* 103–117, 1962.
103. Ullman, M. The adaptative significance of dream. J. Nerv. Ment. Dis., *129:* 144–149, 1959.
104. Ullman, M. Dreaming, life style, and physiology: a comment of Adler's view of the dream. J. Ind. Psychol., *18:* 18–25, 1962.
105. Velluti, R. and Hernández-Peón, R. Atropine blockade within a cholinergic hypnogenic circuit. Exp. Neurol., *8:* 20–29, 1963.
106. Whitlock, D. C., Arduini, A. and Moruzzi, G. Microelectrode analysis of pyramidal system during transition from sleep to wakefulness. J. Neurophysiol., *16:* 414–429, 1953.
107. Williams, D. The syndromes of basilar insufficiency. In Garland, H., ed., *Scientific Aspects of Neurology,* pp. 202–215. Livingstone, Edinburg, 1961.
108. Yamaguchi, N., Ling, G. M., and Marczynski, T. J. The effects of chemical stimulation of the preoptic region, nucleus centralis medialis, or brain stem reticular formation with regard to sleep and wakefulness. In Vol. VI, pp. 9–20. *Recent Advances in Biological Psychiatry,* Plenum Press, New York, 1963.