A UNITARY NEUROPHYSIOLOGICAL MODEL OF HYPNOSIS, DREAMS, HALLUCINATIONS, AND ESP*

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ULLMAN: Our next presentation has a very intriguing title: "A Unitary Model of Hypnosis, Dreams, Hallucinations, and ESP," by Dr. Hernández-Peón.

HERNÁNDEZ-PEÓN: If we consider paranormal perception or ESP as demonstrated as normal sensory perception, it should no longer be considered a magical phenomenon, but the result of certain neural events within the brain. The widespread reluctance among scientists to grant the existence of parapsychological phenomena resides in part in the difficulties one is confronted with when trying to obtain consistent experimental reproduction. This is the natural consequence of our ignorance of the physical and biological processes involved. Although we are still far from understanding the former, current advances in neurophysiological research enable us to formulate working hypotheses that may be useful for a more fruitful experimental study of these phenomena. The aim of this presentation will be concerned only with telepathic transmission to the exclusion of clairvoyance, psychokinesis, and precognition.

Since ESP has been observed alternately in ordinary wakefulness, sleep, and in the particular state known as hypnosis, in order to speculate about

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the possible mechanisms governing psi phenomena I will start by describing the modern concepts on the patterns of brain activity associated with the wakefulness-sleep continuum. New light on the understanding of the neurophysiological mechanisms underlying wakefulness and sleep was shed by the pioneer experiments of Moruzzi and Magoun.[1] They found that high-frequency electrical stimulation of the central area of the brain stem in lightly anesthetized cats elicited the electrocortical manifestations of arousal. Subsequent experiments demonstrated that the integrity of the rostral portion of this region is essential for the maintenance of conscious wakefulness. In fact, experimental lesions in cats[2] and monkeys[3] resulted in a state of unconsciousness entirely similar to the comatose state observed in patients with pathological lesions in the same region. Later, it was found that this brain-stem area designated by its discoverers Ascending Reticular Activating System (ARAS) is a locus of convergence for sensory impulses of all modalities and for corticifugal impulses arising from certain corical areas.[4] Whereas sensory activation of the ARAS accounts for arousal by sensory stimuli and produces what may be termed "obligatory wakefulness," activation of the corticoreticular projections would explain voluntary arousal and "facultative wakefulness."

More recent experiments carried out in our laboratories utilizing the method of localized chemical stimulation of the central nervous system (CNS) have disclosed an arousing noradrenoceptive pathway extending throughout all the levels of the neuroaxis which we have termed "vigilance system."

It has also been found that electrical stimulation of the midbrain reticular formation exerts important inhibitory influences upon sensory input. Until a few years ago, it was traditionally believed that the sensory signals originating at the receptor organs were simply relayed along the specific classic afferent pathways, and that upon their arrival at the specific cortical receiving area they would produce sensory perception. This view cannot be supported at the present time because, as mentioned before, a lesion in the rostral part of the brain stem leaving intact the specific afferent pathways eliminates conscious sensory perception. On the other hand, ablations of the specific cortical receiving areas do not abolish simple sensory discriminations. For instance, after ablation of the visual cortex, monkeys are still able to discriminate light intensity, but not shapes or figures.

The question arises as to what is the functional role of the centrifugal influences that modify the entrance of sensory signals to the CNS at the first synapse. In an attempt to answer this question, we have recorded

sensory evoked potentials in cats with permanently implanted electrodes.

In brief, it was found that sensory evoked potentials recorded at the cochlear nucleus, at the retina, at the olfactory bulb, at the trigeminal sensory nucleus, and at the lateral column of the spinal cord became significantly reduced when the animal's attention was focused upon a motivation-arousing stimulus (the presence of a rat, a fish odor, a scratching noise, etc.).[5] The partial blockade of afferent signals at the first sensory synapse during distraction parallels the introspective reduction of awareness experienced in everyday life. On the other hand, it was observed that the sensory potentials evoked by a stimulus on which attention was focused were facilitated.

This mechanism of "sensory filtering" has also been found at subcortical levels in the human brain. In some patients with electrodes implanted in the optic radiations, which represent the subcortical terminal part of the visual pathway, Hernández-Peón and Donoso[6] recorded potentials evoked by flashes of light. The amplitude of these potentials was consistently reduced during a mental task, such as trying to solve an arithmetic problem, and returned to its original values when the solution was reached.

The inhibitory influences which partially block the entrance of sensory signals to the brain appear to be tonically acting during wakefulness, with phasic fluctuations correlated with the degree of attention. If the mechanisms underlying extrasensory perception have a functional organization similar to those of sensory perception, an inhibition of this kind is likely to be acting on whatever pathways the brain utilizes for detecting the corresponding information.

Besides sensory inhibition, all the available neurophysiological evidence supports the view that the pattern of brain activity during wakefulness requires an extensive background of inhibition throughout all the levels of the CNS, thereby permitting selective facilitation at the particular central pathways involved in a given physiological situation. This view is opposed to the traditional one, which assumes that the brain is more active as a whole during wakefulness. In our view, the brain is quantitatively more inhibited during wakefulness than during sleep, reaching a maximal degree of inhibition during extreme alertness or emotional excitement.

An important question concerning the neurophysiological mechanisms of sleep is: How does sensory filtering act during the two main phases of sleep? By recording tactile evoked potentials at the spinal trigeminal sensory nucleus, Hernández-Peón, O'Flaherty, and Mazzuchelli-O'Flaherty[7]

found that those potentials were enhanced during "slow" (or "light") sleep and reduced again during the periods of "rapid" (or "deep") sleep. As the latter periods in humans are usually accompanied by vivid dreams, it is evident that sensory inflow to the CNS is maximally reduced during those states of the wakefulness-sleep continuum associated with the most intense conscious experiences, such as sensory perception and dreaming. The latter cannot be responsible for sensory inhibition during these periods, because during rapid sleep the arousal threshold is considerably raised, owing to intense inhibition of the vigilance neurons. Since a lesion in the midbrain tegmentum abolishes sensory inhibition in both states, it is evident that the inhibitory source corresponds to reticular neurons functionally different from those involved in wakefulness, although anatomically overlapping with them. Therefore, we have recently postulated the existence of a conscious experience system independent from the vigilance system and regulating sensory filtering. In this way, whereas the conscious experience system controls the inflow of sensory signals, the vigilance system regulates cortical excitability (Fig. 1).

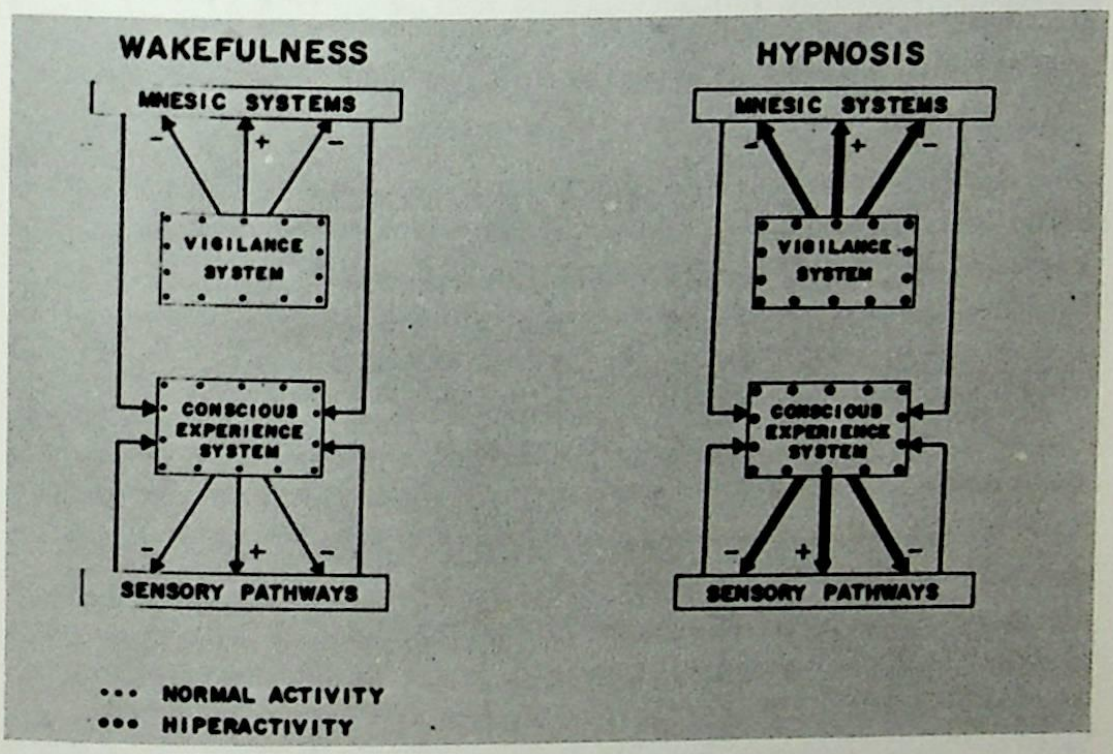

Figure 1

The views just presented allow us to elaborate a working hypothesis on the mechanisms underlying hypnosis. All the available evidence indicates that the physiological activities during hypnosis cannot be differentiated

from those of wakefulness. Therefore, we postulate that during hypnosis there is hyperactivity of both the vigilance system and the conscious experience system (Fig. 2). But in addition, other neural systems play a role during hypnosis. In ordinary wakefulness, sensory signals arriving at the

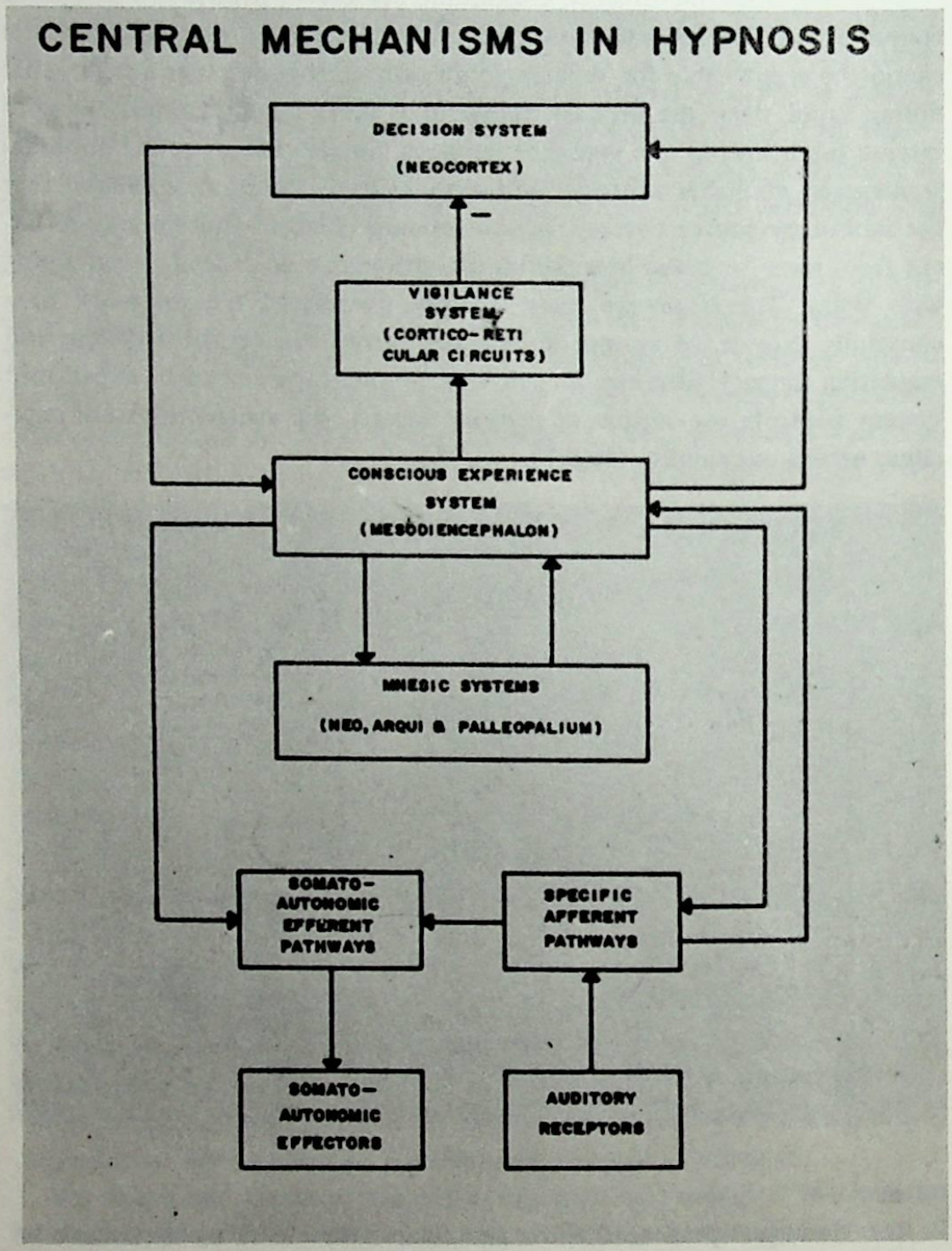

Figure 2

conscious experience system must be compared with information stored in the mnesic systems. Only after comparing what arrives with what is stored, can a decision be made and orders sent to either sensory, somato-autonomic, or particular mnesic circuits. An inhibitory bypass of the matching-decision or executive mechanisms involving corticosubcortical circuits during hypnosis allows verbal suggestions to directly activate the corresponding neural circuits, thereby producing a wide variety of sensory, motor, visceral, or memory changes.

In order to obtain some information about cyclic changes of excitability or reactivity of the recent memory system, we have recorded afterdischarges and locally evoked potentials from the entorhinal cortex of cats, which is the homologue of the hippocampal cortex in primates. Both electrical indexes were smaller during alertness and largest during "rapid" sleep, with a practically linear function through the wakefulness-sleep continuum.

Correlating cortical reactivity with the capacity of mnesic association underlying mental activity, it is obvious that emotional excitement and active wakefulness are accompanied by lowest degrees of cortical reactivity and mnesic association: during relaxed wakefulness and "slow" sleep, logical mnesic association prevails, with a moderate degree of cortical reactivity; during "rapid" sleep, maximal cortical reactivity accounts for the illogical mnesic associations underlying the manifest content of dreams. For completing a neurophysiological model of dreams, the author has postulated that similar changes of reactivity occur in the emotional and motivational limbic systems, during the wakefulness-sleep continuum. Maximal disinhibition of those limbic systems during the periods of "rapid" sleep would account for the latent content of dreams.[8]

In summary, the main patterns of brain activity during wakefulness and sleep can be viewed in the following way.

During attentive wakefulness, activity of the conscious experience system and of the vigilance system coexists, resulting in a simultaneous extensive degree of inhibition at first sensory synapses and at the cortex. As the activity of the sleep system increases, and as that of the vigilance system becomes inhibited, "slow" sleep ensues, resulting in partial cortical disinhibition. Since the activity of the conscious experience system is also reduced, sensory filtering is released with a resultant increase in the amount of sensory signals admitted to the CNS. As the sleep system becomes more active during the periods of "rapid" sleep, the vigilance system is correspondingly more inhibited, resulting in a higher degree of

cortical disinhibition. When the abundant neural discharges with coded information from the cortical mnesic neurons reach the conscious experience system, oneiric activity is triggered. At this stage, sensory inhibition acts again, thus preventing a chaotic sensory bombardment of the underlying neural activity.

The described neurophysiological model of dreams can be analogically applied for understanding the mechanism of hallucinations produced in a variety of circumstances. In sensory deprivation, the changes of spontaneous activity in sensory receptors may be linked to a decreased activation of the inhibitory neurons restraining the excitability of cortical mnesic neurons. In sleep deprivation, postexcitatory refractoriness or fatigue of the vigilance neurons and their associated inhibitory interneurons would be responsible for cortical disinhibition. It is possible that hallucinogenic drugs achieve the same cortical inhibitory process, by chemical interference with either the synthesis, release, or action of the specific inhibitory synaptic transmitter.

Unpublished experiments in our laboratories have shown a cortical disinhibitory action of picrotoxin and alcohol by local application of these substances in the entorhinal cortex. Similar findings have been obtained in preliminary experiments with LSD-25.

CAVANNA: I think it would be very interesting to try topical application not only of drugs, but also of naturally occurring active substances like the catecholamines, serotonin, etc.

HERNÁNDEZ-PEÓN: In 1960 we started to use the method of local application of microcrystals of acetylcholine and, after an extensive systematic exploration of the CNS, we were able to trace a cholinoceptive hypnogenic pathway, or sleep system, anatomically highly circumscribed. We have made a less extensive exploration with epinephrine and norepinephrine, and we plan to do the same with serotonin.

CAVANNA: How do you correlate your vigilance and sleep systems with Hess's classic systems?

HERNÁNDEZ-PEÓN: This is a very interesting question. What Hess called ergotropic system is part of our vigilance system, and what Hess called trophotropic system is part of our sleep system. Our sleep system includes several intralaminary thalamic nuclei, such as the reticular nucleus and the region of the *massa intermedia* where Hess located what he called the "sleep center."

In speculating about the possible brain circuits involved in psi operation, it may be postulated that extrasensory stimuli are capable of activating both the mnesic system and the emotional system, which in turn would discharge into the conscious experience system, thus giving rise to sensory or emotional experiences (Fig. 3). In this way, psi could manifest

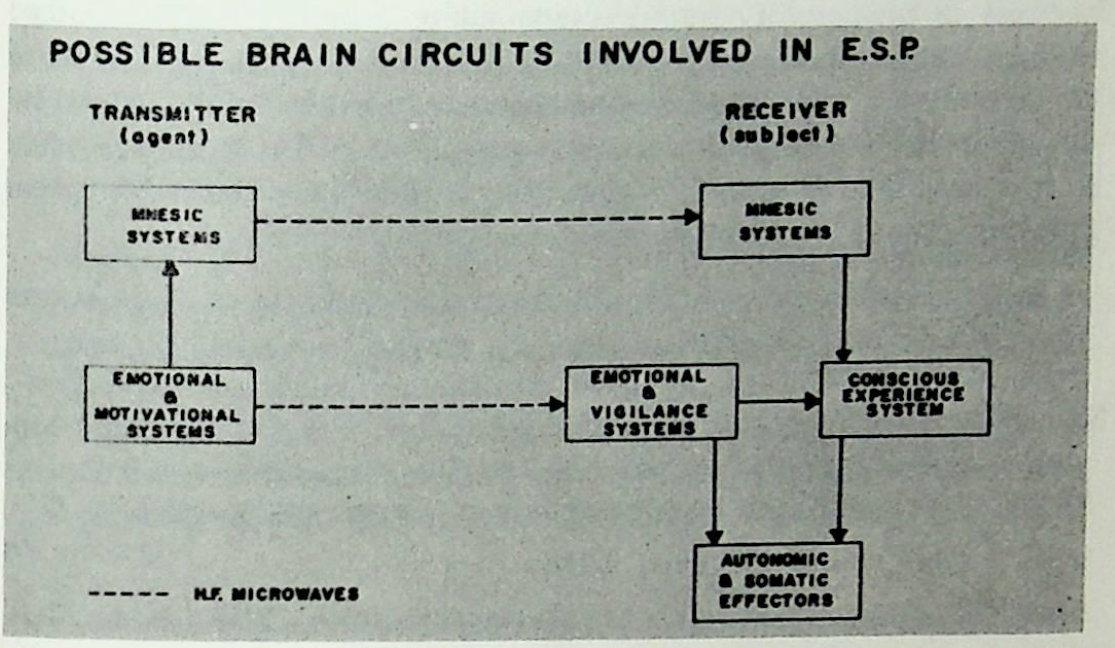

Figure 3

itself by highly complex integrated imageries as well as by unconscious autonomic responses. Implicit in this hypothesis are complex patterns of cortical activation involving neurons storing highly coded information. It is also implicit that cortical and limbic neurons are capable of producing and detecting minute perturbations of an unknown field of energy.

TART: Dr. Hernández-Peón, could you make it clear at this point why you feel that extrasensory stimuli might go through the memory system, whereas other stimuli wouldn't? I didn't follow that point.

HERNÁNDEZ-PEÓN: Yes. I think that extrasensory stimuli should activate the mnesic systems, I mean the cortical neurons, in order to have an integrated visual imagery.

KRIPPNER: Dr. Hernández-Peón, are you familiar with William Roll's theory on mechanisms involved in ESP?[9] He feels that anything that an agent transmits to a subject activates certain engrams or memory traces within the cortex of the subject, and that the subject will respond only in terms of his personal repertoire of experiences.

HERNÁNDEZ-PEÓN: I am not familiar with that theory, but my model is not limited to already existing memory traces.

I have listed some possible factors which may favor psi operation. These factors concern both the transmitter or agent, and the receiver or subject. As far as the transmitter is concerned, it is likely that a threshold intensity of extrasensory stimuli is required, in forms of intense emotional feelings, highly specific motivations, or recent memory tracings. As far as the receiver is concerned, an optimal state of cortical disinhibition is required, somewhat different from that postulated by Dr. Rýzl. According to this view, relaxed wakefulness or sleep would be more favorable for psi operation than any state of alertness or emotional excitement.

Facilitating factors could be the pharmacological actions of drugs that produce cortical disinhibition, such as alcohol[10] and hallucinogens.[11] Other factors could be selective cortical facilitation and generalized sensory inhibition as produced during hypnosis. Finally, we should consider maximal excitability of the conscious experience system as it appears to occur physiologically during REM sleep, and as it may be pharmacologically induced by hallucinogenic drugs.

It is important also to keep in mind the factors that may interfere with psi operation. In the transmitter, we should consider subthreshold intensity of extrasensory stimuli due to non-significant or lacking specific motivation. On the side of the receiver, several conditions might interfere: (1) cortical inhibition, as present in disturbing emotions or preoccupations, which could block the mnesic systems; (2) deficient sensory inhibition, as probably present in neurotic subjects, which would lead to an excessive sensory bombardment; and (3) decreased excitability of the conscious experience system, as observed in drowsiness, fatigue, slow sleep, sleep deprivation, or as produced by pharmacological actions of drugs such as alcohol in high doses and barbiturates.

RAO: There are a few things I would like you to consider. Theorization in parapsychology is generally based on one or another of the following three assumptions: One assumption, the one you also seem to make, is that there are such things as extrasensory stimuli. Somehow an object transmits signals, which we interpret and experience as extrasensory perceptions. Another assumption is that the mind goes out to the object—the object having no role, except that of passive target, as in the case of sensory perception. The third assumption is that our minds are constantly in touch with all that is real, with the result that all we have to do for ob-

taining extrasensory information is to locate that particular thing and bring it into consciousness.

Your theory assumes that some stimuli come through unknown channels and excite some parts of the brain, giving extrasensory perceptions. Do you have any room in your theory for postulating that the active agents are not the stimuli—no matter how intense—but the individual himself? There is a great deal of evidence in parapsychology to suggest that it is not the target per se, as much as the attitude of the subject toward the target which is important.

HERNÁNDEZ-PEÓN: As I said in my introduction, I am dealing only with telepathy; i.e., transmission of information from brain to brain by means of an energy completely unknown to us.

RAO: Perhaps, even in telepathy, it is not a question of transmitting from one mind to another, but just a matter of one mind making contact with a target, which—in this case—happens to be another mind. It still makes sense.

BELOFF: Well, it makes sense to you maybe, but it doesn't make sense to Dr. Hernández-Peón. He wants us to begin with the basic assumption that there is an interaction between two brains with some unknown channel between. We've got to grant him this assumption if we're going to follow through his theory. The weakness of his theory, as he frankly admits, is that he is limiting himself to telepathy, as most physicalists do.

TART: Dr. Hernández-Peón, I'd like to ask you a question about the processing of the psi information within the nervous system. For most sense organs there is a specific receptor, which goes through a subcortical thalamic relay system and eventually ends up as conscious experience. Is your basic postulate here that the cortex itself is somehow the receptor system for psi impulses, that the information is then transferred to wherever memory is stored, and then into consciousness? Do I understand this correctly?

HERNÁNDEZ-PEÓN: Yes. In other words, the cortical neurons have to be activated by this unknown energy, and then the impulses from the cortical neurons have to be projected down to the conscious experience system in the brain stem in order to have extrasensory perception.

TART: Via the memory system?

HERNÁNDEZ-PEÓN: Yes, of course. In the cortex.

TART: So the specific receptor would be a certain specialized area in the cortex?

HERNÁNDEZ-PEÓN: Yes, probably within the cortex itself there are particular neurons that play a major role in those phenomena.

BELOFF: Could you explain in more detail what is meant in your model by "consciousness system"? I gather from your diagrams that you locate it in the midbrain. Are you saying that when certain processes go on in the midbrain, they may be accompanied by subjectively conscious experience, without necessarily referring to what's going on in the cortex?

HERNÁNDEZ-PEÓN: Yes. Conscious experience is located in the brain stem. This is the only part of the brain where a lesion produces a state of unconsciousness. For instance, if we remove the cortex from a cat or dog, the animal's behavior can still be interpreted as conscious.

BELOFF: Well, we can't be sure.

HERNÁNDEZ-PEÓN: Since a decorticated animal can attain simple sensory discriminations, and if we admit that sensory discrimination requires conscious perception, the conclusion is warranted that the cortex is not essential for basic conscious experiences. Nevertheless, the cortical processing of information is necessary for more complex and integrated conscious experiences.

BELOFF: During dreaming, would the cortex be involved?

HERNÁNDEZ-PEÓN: Yes. Actually cortical hyperactivity is mandatory for production of oneiric material. Although normally there is an interplay between cortex and brain stem, the final event leading to conscious experience appears to take place at the brain stem.

BELOFF: Is this a generally accepted postulate, or is it a theory of your own?

HERNÁNDEZ-PEÓN: It is a theory of my own which I proposed two years ago in a symposium on the brain stem at the International Congress of Neurosurgery held in Copenhagen, but it is based on experimental evidence.

VAN OVER: I have three questions: 1. Have you conducted any research specifically involving cortical activity in relation to ESP? 2. Are you familiar with Grey Walter's work on the "expectancy wave," as he calls

it?[12,13] 3. Are you familiar with Grey Walter's work with Mrs. Garrett? By the way, I'd like to ask Mrs. Garrett to tell us about her work with Dr. Grey Walter.

HERNÁNDEZ-PEÓN: I'm familiar with Grey Walter's work on the ex-wave, but not with his study with Mrs. Garrett.

GARRETT: I felt I should know something about the neurophysiological correlates of trance, clairvoyance states, and telepathy. Therefore, I went to Bristol and worked with Grey Walter for ten days, during which I underwent several experiments: my EEG was recorded during a trance state, under hypnosis, and under the effect of 250 micrograms of LSD. He found the "expectancy wave" in most of my graphs. I think you would be interested in discussing these findings with him.

OSMOND: Yes, as his techniques are rather sophisticated, and it would be very difficult to understand their impact for parapsychology without discussing his results directly with him.

ULLMAN: Would you like to repeat your first question, Mr. Van Over?

VAN OVER: Yes. Have you conducted any research specifically involving cortical activity in relation to ESP?

HERNÁNDEZ-PEÓN: Yes. We have conducted only a few experiments by simultaneously recording the EEG of two persons—an agent and a subject. In one case we instructed the agent to mentally call the name of the subject, and there was blocking of the alpha rhythm of the subject. This was repeated three times, with progressively shorter durations of alpha blocking. The results of the next experiment with the same subjects were negative.

HOFMANN: Do you know the experiments of Dr. Baldwin, from the National Institute of Mental Health?[14] He worked with chimpanzees, who have a very pronounced social life, as you know. When a normal animal received LSD, his altered behavior under drug disrupted the established social patterns of the clan. When both his temporal lobes were removed, he behaved normally after recovery from the operation. If he then was given LSD, he didn't show any reaction to it. Could you explain these findings?

HERNÁNDEZ-PEÓN: I didn't know about those experiments, but they would lend support to my interpretation of the action of hallucino-

genic drugs. In other words, our hypothesis is that these drugs act first by disinhibiting that part of the cortex which lies in the temporal lobes and corresponds to the recent memory system. Therefore, if that part of the cortex is removed, the drug cannot act, and thus there would be no hallucinations.

RAO: I'm not sure I understood one point about your ESP experiments with the EEG. Was the alpha rhythm blocked simultaneously in both agent and subject when you said the name to the lady?

HERNÁNDEZ-PEÓN: The agent mentally called the name of the subject, and there was simultaneous blocking.

ULLMAN: This is an intended replication of an experiment; this was the first attempt of simultaneous alpha induction in identical twins.[15]

BELOFF: But in that first experiment merely opening of eyes was used to interrupt the alpha rhythm. Here you have them doing a mental task.

FINER: May I ask you to clarify two points, please. In the work with visual stimuli and mental arithmetic, what was the time lapse between the stimulus and the response?

HERNÁNDEZ-PEÓN: We didn't measure the latency of the evoked potentials, because we were working with the EEG machine and it was impossible to make an accurate measurement.

FINER: My second question concerns the centrifugal blocking of signaling in hypnosis where you mention the first sensory synapse. There has been recent work on this presynaptic effect.[16] Can one say that hypnosis works on the presynaptic system, or is it still unknown?

HERNÁNDEZ-PEÓN: That is still unknown. Actually, when I say the first synapse, I mean both presynaptic terminals and postsynaptic inhibition during attention in cats. In humans nobody has made recordings from the first synapse during hypnosis. The only electrophysiological recordings we have made in humans during hypnosis were concerned with spinal evoked reflex potentials.[17] and average evoked potentials from the scalp in collaboration with Stross, Shevrin, and Rennick. In both groups of experiments, the evoked responses were significantly diminished during hypnotic anesthesia of the corresponding arm.

ULLMAN: I would like to see if we can extract from Dr. Hernández-

Peón's contribution anything more relevant to parapsychology. What can we do with the fact that we can now physiologically define an optimal condition for psi operation as a state of massive excitation of the brain, far greater than anyone experiences while awake? This kind of cortical activation may be the physiological correlate of an external scanning operation occurring during sleep which may range further than the vigilance scanning that occurs during the normal waking state. This broader scanning of the sensory spectrum may be underlying the paranormal experience.

TART: Dr. Hernández-Peón, I would like to ask you two questions about your theory. We usually think of sensory pathways as starting with a specific receptor, the stimulus being relayed through several somatic stages until it reaches the cortex. You're proposing the opposite route, beginning at the cortex through the memory system into awareness. Therefore, my first question is, how would you experimentally descriminate this different type of routing of information from that of the usual classic sensory pathways? Secondly, how would you go about enhancing the operation of ESP?

HERNÁNDEZ-PEÓN: It would be very difficult to experimentally discriminate the participation of classic afferent pathways in psi phenomena. A possibility would be to perform those experiments in complete deprivation of sensory stimuli.

TART: That still does not exclude the existence of an ESP receptor located, for instance, in the big toe, which would have synaptic relays just as any other classic pathway. This is the discrimination I'm trying to get at.

HERNÁNDEZ-PEÓN: Only animal experimentation would allow us to control the participation of special sensory pathways and other central structures in psi operation. For this purpose it would be highly desirable to experiment with monkeys, because if such a mechanism exists in the human brain, it is probably also present in other mammals. Animal experimentation would reduce the number of possible complicating factors, opening a new avenue of research for these elusive phenomena.

TART: If this is the pathway, how would you go about enhancing the operation of ESP?

HERNÁNDEZ-PEÓN: By enhancing the excitability of the cortical mnesic system and of the conscious experience system. This could be done pharmacologically.

TART: Does this imply a prediction that any pharmacological agent that enhances cortical excitability should also enhance ESP?

HERNÁNDEZ-PEÓN: According to my neurophysiological model, any drug that enhances the excitability of the cortex and of the subcortical conscious experience system, such as the "psychedelic" drugs, should favor psi. However, the ideal drug would be one that would at the same time increase sensory inhibition in the specific afferent pathways.

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