E
tarily in the course of his extensive clinical studies of cortical function and of epilepsy, Dr. Wilder Penfield made a chance observation that has led to a major contribution to the understanding of cerebral function.

In the nineteenth century, neurosurgeons first began to employ the experimental techniques of Fritsch and Hitzig, Ferrier, Sherrington, and others, to study the cortical localization of motor functions in man. Frequently these techniques were used to map out the area of the brain to be removed in patients with focal epilepsy.

"More exciting were the two cases published in 1909 by Harvey Cushing on sensations resulting from stimulations of the post-Rolandic area in man. Operating under local anaesthesia he established the sensory function of the human post-central strip. Penfield, since that day . . . , has greatly extended these observations on both the motor and sensory and other responsive areas of the brain. . . ."

"Ferrier many years ago said that all parts of the cortex would prove to be excitable, but he could not demonstrate excitability himself except from restricted areas of the brain: the greater part was unresponsive. Ferrier's prophecy was long unfulfilled, and only recently has come closer to confirmation. Thus we now know that responses must be looked for not only in movements of the limbs or in sensations or in flashes of light or sounds or odours but in two other directions. The first is in the autonomic or visceral changes that may be the replies to stimulation of some areas. . . . This work has mainly been done on animals. The second and much the more exciting, even dramatic, discovery was made by Penfield—that stimulations of the temporal lobe in human beings may evoke experiences, pictures in the mind. . . ."

The following is Dr. Penfield's account of his first experiences with this phenomenon:

". . . I was operating upon a woman under local anaesthesia in the Royal Victoria Hospital and was applying to different points on the temporal lobe of her brain a stimulating electrode. She (E.W.) told me suddenly that she seemed to be living over again a previous experience: she seemed to see herself giving birth to her baby girl. That had happened years before, and meanwhile the girl had grown up. The mother was now lying on the operating table in my operating room, hoping that I could cure her attacks of focal epilepsy.

"This, I thought, was a strange moment for her to talk of that previous experience, but then, I reflected, women were unpredictable and it was never intended that men should understand them completely. Nevertheless, I noted the fact that it was while my stimulating electrode was applied to the left temporal lobe that this woman had had this unrelated and vivid recollection. That was in 1931.

"It was more than five years later when a somewhat similar psychical state made its appearance during electrical stimulation. This time, however, it seemed certain that the stimulus had somehow summoned a past experience.

"The Montreal Neurological Institute was opened in 1934, and a patient, J.V., a girl of 14 years, was admitted in June 1936. . . . She was complaining of seizures during which she sometimes fell unconscious to the ground in an epileptic convulsion. But, immediately preceding such an episode, she was aware of what seemed to be a hallucination. It was always the same: an experience came to her from childhood. . . ."

"At operation, under local anaesthesia, I mapped out the somatic sensory and motor areas for purposes of orientation, and I applied the stimulator to the temporal cortex. 'Wait a minute,' she said, 'and I will tell you.' I removed the electrode from the cortex. After a pause, she said: 'I saw someone coming toward me, as though he was going to hit me.' It was obvious also that she was suddenly frightened.

"Stimulation at a point farther forward caused her to say, 'I imagine I hear a lot of people shouting at me.' Three times, at intervals and without her knowledge, this second point was stimulated again. Each time she broke off our conversation, hearing the voices of her brothers and her mother. And on each occasion she was frightened. She did not remember hearing these voices in any of her epileptic attacks.

"Thus the stimulating electrode had recalled the familiar experience that ushered in each of her habitual attacks. But stimulation at other points had recalled to her other experiences of the past, and it had also produced the emotion of fear.
Our astonishment was great, for we had produced phenomena that were neither motor nor sensory, and yet the responses seemed to be physiological, not epileptic. . . .”

Dr. Penfield’s subsequent experiences with similar patients has led to two important neurophysiological concepts: the definition of the centrencephalic system and its importance to consciousness, and the role of the temporal lobe in memory and in psychomotor epilepsy. 1-27, 29, 30, 33, 34, 37-40 Reproduced below is Penfield’s initial account of these ideas, as given in the Harvey Lecture delivered October 15, 1936.

References

THE CEREBRAL CORTEX
AND CONSCIOUSNESS*1

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A NEUROSURGEON has a unique opportunity for psychological study when he exposes the brain of a conscious patient, and no doubt it is his duty to give account of such observations upon the brain to those more familiar with the mind. He may find it difficult to speak the language of psychology, but it is hoped that material of value to psychologists may be presented, the application being left to them. It seems to me quite proper that neurologists should

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1 Lecture delivered October 15, 1936

push their investigations into the neurological mechanism associated with consciousness and inquire closely into the localization of that mechanism without apology and without undertaking responsibility for the theory of consciousness.

To make such an inquiry is to ask a very old question, as is shown by the following quotation from Zophar, the Naamathite, in the Book of Job:

"Surely there is a vein for the silver
And a place for gold where they fine it;
But where shall wisdom be found?
And where is the place of understanding?"

REVIEW OF RELEVANT LITERATURE

The cerebral cortex has apparently acquired an increased functional specialization as the mammalian scale is ascended, ending in a very remarkable increase in man. Lashley (1920) concluded from his work on rats that the capacity of these animals to learn (maze habits) was reduced, depending upon whether more or less cerebral tissue was destroyed. This decrease was not influenced by the nature of the cytarchitectural fields removed. This function therefore depended, in his opinion, only upon the amount of cortical tissue present and not upon its anatomical specialization.

Pavlov (1927), working upon dogs, concluded that the special function of the cerebral cortex is to establish new nervous connections and so to ensure a perfect functional correlation between the organism and its environment. It is, he said, the essential organ for the maintenance and establishment of conditioned reflexes. In contrast to Lashley's findings in the rat, Pavlov admitted that removal of the posterior portions of the cerebral cortex destroyed the activity of the special analyzers for acoustic and visual reflexes, while tactile reflexes were disturbed very little indeed. On the other hand, bilateral removal of the anterior one-half of the cerebral cortex destroyed the tactile analyzer and interfered little with learning and retention of visual and auditory reflexes. He found that bilateral removal of the temporal lobes in dogs damaged the auditory analyzer most. Babkin, working in Pavlov's laboratory, showed that after such an operation the dog learned to respond to single tone auditory stimuli but he never reacted to the calling of his name, never appreciated successive compound auditory stimuli.

Fulton (1934) and his associates, working upon monkeys and chimpanzees, have accurately demonstrated a considerable amount of specialization of function in the pyramidal and "extra-pyramidal" portions of the cerebral cortex, a specialization which to a large extent is exclusive.

In man the increase of specialization of certain areas of the cerebral cortex is as striking as the
enormous increase in the total quantity of the cerebral cortex. Capacity for replacement, described by Lashley as almost universal in the rat, is still present in man to a very considerable extent, especially in infancy. But in the adult man, one occipital lobe is essential to useful vision of any sort in the opposite field; lesions of the motor cortex on one side result in irrecoverable crossed hemiplegia, and the cortical sensory areas are irreplaceable for certain forms of sensation in the opposite limbs. Furthermore, the appearance of speech has resulted in the development of a unilateral highly specialized localization within the cortex. The aphasia which becomes permanent following destruction of certain areas of the human brain, still so poorly defined, indicates an enormous increase in specialization as compared with the disturbance of the understanding of a dog for compound auditory stimuli after removal of both temporal lobes.

The cerebral cortex of man has been divided into separate cytoarchitectural areas by the histological studies of Vogt (1926), von Economo (1929), Campbell (1905), Brodmann (1925) and others, as shown by figure 1. It would seem reasonable, a priori, that the differences that exist in the cell arrangement of these regions should correspond with a difference in function. This certainly is the case in the motor and visual areas and must be so to some extent in the others. The fact that marked substitution is possible in infancy and that some substitution is possible later does not alter the fact that under normal conditions there is specialization of use of the cortex for special purposes in focal areas.

Foerster (1936) has made an effort to delimit function in the human cerebral cortex according to the cytoarchitectural fields of Vogt and Brodmann. This attempt has been successful to some extent at least. After a study of well over one hundred cases of electrical exploration of the human cortex in conjunction with Dr. Edwin Boldrey, I have been unable as yet to outline functional representation within the same sharp limits, with the exception of the precentral and postcentral gyri and perhaps the calcarine cortex.

Hughlings Jackson (1931) pointed out years ago that recoverability from clinical lesions of the nervous system is chiefly a matter of quantity of nervous tissue involved, a conclusion startlingly like that of Lashley and Pavlov. Jackson, however, described, or rather predicted, three levels of functional differentiation in the central nervous system. The lowest level was in the spinal cord, medulla and pons, where the individual units of the body, such as muscles, had individual representation. At the middle level, which he suggested would be found in the sensori-motor portion of the cerebral cortex, there was re-representation, not of the individual parts, but of peripheral function, such as co-ordinated movements and elaborations of sensations.

Pavlov, after exhaustive physiological analysis, arrived at a similar conclusion in regard to the cerebral cortex which he did not limit to sensori-motor cortex as Jackson had done. He stated that probably the entire cortex represented a complex system of analyzers of internal as well as external environment of the organism. He suggested that all tissues of the body would eventually be found to be included in this representation.

Jackson went one step further, inferring that there must also be a still higher level of integration, a final sensory and motor arrangement which might form the neural substratum of consciousness, and he suggested that this might be found in the frontal and prefrontal regions.

STIMULATION OF THE CEREBRAL CORTEX IN CONSCIOUS PATIENTS

The patients referred to below were operated upon without any anesthetic and without receiving a pre-operative sedative. Local anesthesia was used to avoid pain and the brain exposed widely by means of osteoplastic craniotomy. The operative wound was carefully ringed about and a sheet arranged perpendicularly so that the patient's face and body were fully exposed below the operative field. Thus he could be observed by those who sat beyond the sterile barrier and could converse with them.

Response is obtained from the human cortex most easily from the vicinity of the central fissure of Rolando, that is, from Brodmann's areas 4 and 6 anteriorly, and 1, 2 and 3 posteriorly (Fig. 1). These responses are not obtained from exactly fixed areas like the keys of a piano, but they vary considerably from case to case. During a specific operation they remain constant in position but can be influenced by facilitation and inhibition. Outside of the motor cortex the same area in different brains may give marked differences of response depending apparently upon the frequency of some previous experience, such as the aura of an epileptic seizure. These unusual responses may well be explained by the process which Pavlov has termed conditioning.

VOCALIZATION

The discussion may be best opened by a description of vocalization which has been produced by us only recently since we have begun to use a thyratron stimulator. It has not previously been described as an isolated phenomenon in man, and Leyton and Sherrington (1917) observed that they could not produce it in anthropoids with faradie stimulation. Gibbs and Gibbs (1936) pro-

To be published presently.
FIG. 1. Cytoarchitectural fields of cerebral cortex (Brodmann).

duced purring in cats by stimulation in the vicinity of the infundibulum.

(1) The patient, H. My., was an intelligent man of 32 who worked as a railroad fireman. He had complained of epileptic seizures for three years before admission. The cause of these seizures was found to be a small glioma of benign type deep in the frontal lobe near the midline and anterior to the motor gyrus (Fig. 2, T). In April 1935 the right hemisphere was exposed by osteoplastic craniotomy and careful stimulation experiments were carried out. Reference to figure 2 shows a photograph of the cerebral cortex of this patient during operation. The patient was quiet and co-operative and talked freely with the operator and with the specially trained observer, Miss Mary Roach. The numbers on the small paper squares which may be seen on the surface of the brain indicate the order in which stimulation with positive result was carried out; number one representing the first stimulation and number two the second, and so forth. The rest of the exposed brain was explored completely with the electrode without result. The results of the positive stimulations were as follows:

When point 13 was touched with the electrode the patient reported sensation in the left little finger and extension of the little finger was ob-
served. This was repeated twice without warning to the patient and with the same result. At 1 a feeling of "electricity" was produced in the left middle finger. There was no movement associated with the sensation. At 2 a feeling of "electricity" was produced in the left index finger; at 11 flexion of the left arm and forearm and extension of the fingers. This last was repeated once with the same result.

Stimulation at 10 produced closure of the left hand. The patient reported feeling a strong sensation "like hold on to electricity," meaning that his hand tingled while it closed. At 3 there was sensation over the lower lip on the left side. At 4 a sensation was produced like electricity in the left side of the tongue. Stimulation at 6 produced violent swallowing and after a short interval of silence the patient stated he had felt "electricity" in the mouth. Stimulation at 7 produced a feeling in the mouth.

These numbers are therefore seen to lie on either side of the central fissure of Rolando. There is no way of determining the central fissure in a living patient except by stimulation because of the great variability in cortical pattern, especially in pathological cases. Number 7 lies just below the fissure of Sylvius; number 6 just above it. On the precentral gyrus at the point 5 which lies between areas from which movement of the upper extremity and of the face were produced, stimulation resulted in vocalization. Because of the fact that this was the first example of such vocalization, stimulation of this point was repeated thirty-one times, but without undue fatigue.

At the first response the patient emitted a somewhat groaning "Oh." After stimulation stopped he said, "I do not know why I made that noise." This was repeated four times with the same result. The intensity of thyratron stimulation was 28, the frequency of the stimulus being between 60 and 70 per second. When asked why he continued to make this noise the patient said "I don't know. Something made me speak and I felt something touch up there." This last may have been due to some pressure upon the unanesthetized scalp. The next time he was stimulated he said, "You must have made me do that." The same strength of stimulus produced numerous sensory results in the other areas of the cortex but no other motor response.

At the seventh stimulation Dr. Colin Russell observed the patient carefully during his vocalization. He remarked that the mouth opened widely without any expression of fear or emotion while crying. At the eighth stimulation he vocalized loudly and when asked afterwards whether he felt anything he said, "Felt anything—sure it felt as though you were pulling the voice out of me." The longer the stimulation was continued the louder the tone seemed to become and the higher the pitch. The fourteenth stimulation was therefore prolonged to see the effect. In this instance vocalization continued for a period of six seconds and ended in a tremolo (probably when his

![Figure 2](image_url)

**Fig. 2.** Cerebral cortex of patient H. My. photographed during operation. The numbers placed upon the brain indicate points from which motor or sensory responses were obtained by stimulation. T indicates position of oligodendroghoma. Inset shows position of craniotomy exposure.
breath gave out). On one occasion he vocalized, then drew a deep breath and continued to cry.

At the eighteenth trial area 12, about 6 mm. below 5, was stimulated, using a somewhat stronger stimulus. This was repeated at both areas on the twentieth trial and it was noted that the tone of the voice was higher at 5 than it was at 12, but it was true that the effect seemed greater when 5 was stimulated, which may perhaps account for the higher tone.

On the twenty-second stimulation the patient was informed that he was to try not to call out when stimulated. He said he would try. I warned him when I was going to stimulate but the vocalization began almost immediately after stimulation and continued until the electrode was withdrawn. I then said to the patient, "I win," and he replied, "You did," and laughed. But he added, "I guess I would have won if I had been on that side of my head."

In general, the patient was unable to stop the cry or to influence it in any way. He was as surprised at the first sound of his own voice as we were and he dissociated himself at once from this artificial employment of his own cortex. He knew he had not willed it.

At a distance of 1 mm. from a circumscribed area, using a monopolar electrode, no result was obtained but on moving the electrode 1 mm. nearer, the vocalization would regularly occur in typical fashion. With the same intensity of stimulus used to produce this vocalization no motor movements could be obtained anywhere. A stronger stimulation had to be used to produce flexion of the hand on the same convolution above this point, and below the same, stronger stimulation produced violent swallowing. On returning to the vocalization area even the strong stimulation did not produce any additional associated motor movements but only the sound of his voice in the vowel "O" or "A." There was nothing at any time to suggest words.

At ward rounds nine days later the following note was made on the patient's history: "On discussing with the patient his sensation at the time when vocalization was produced on the operating table, he states that it did not sound as though he were saying anything he wanted to say, but as though his voice came with a rush, as something beyond his control. There was no sensation of the mouth, tongue or face at the time of vocalization.

"He says he felt no sensation anywhere but 'just as though something drew it out of my mouth.'"

"He dreamed next day after operation that someone was making him speak but he did not seem to be on the operating table. There is at present no speech disturbance nor has there been since operation."

In five subsequent cases vocalization has been produced by stimulation of the cortex in a roughly corresponding area upon the precentral gyrus of the left side in two and of the right in three cases. The location of the vocalization point in all six
cases is transposed to the left hemisphere in figure 3.

(2) H. Mi. was an intelligent young man of 27 years. Stimulation in the precentral gyrus produced vocalization (II, Fig. 3). On the same gyrus above the area, elevation of both brows symmetrically was produced; below the area, swallowing.

(3) Patient F. R. gave vocalization after stimulation of a similar point (III, Fig. 3) which lay just anterior to points in which mouth and tongue sensation could be produced. It was below the locus for movements of the right eye but it was not so discrete an area as in the first case mentioned above, and it was associated with drawing of the face to the right and a feeling of nausea. This spread was doubtless due to epileptic habit, as stimulation 2 cm. anterior and below produced an epileptic seizure which resembled those from which he habitually suffered and in which there was an epileptic cry at the beginning.

(4) E. M. was a young woman of 22. Vocalization was produced from the left precentral gyrus (I, Fig. 3) in an area no greater in diameter than 3-4 mm. The cry was somewhat quavering and was produced three times. Above it flexion of the wrist was obtained. Below it twitching of the lower lip and jaw resulted, more on the right side.

(5) F. W. was a boy of 17 years. Vocalization was produced from the left hemisphere at IV in figure 3, just below an area from which closure of both eyes was produced. When he had closed them both, the patient remarked that he could not help closing the right eye. The vocalization point was just anterior to the source of numbness in the right side of the tongue.

(6) E. L. was a housewife of 39 years who had suffered from epileptic seizures for six years following a vascular accident. This vascular lesion had produced the cyst seen in figure 4, situated in the left parietal lobe. There was no aphasia but the patient was a little childish.

The central fissure was mapped out by thyatron stimulation as may be seen by reference to figure 4. The inset in that figure indicates the cerebral localization.

A—pricking sensation right fingers; B—shaking of whole right hand; C—sensation in right fingers; D—sensation in nose; E—sensation in right thumb and closure of jaw; F—sensation in right side of chin; G—sensation in tongue and chin; I—flexion of elbow; K—flexion of fingers; L—slight tremor in right side of face with sensation in that cheek and chin.

M—vocalization “Umgh.” Patient then said “What is that?” Without replying I repeated the stimulation. Vocalization was repeated and her mouth pulled to the right. I then asked her why

Fig. 4. E. L. Woman of 39 years who had had occlusion of a cerebral artery six years previously leaving deep cyst roofed by translucent membrane and floored by the ependyma of the underlying ventricle. Photographed during operation. Inset indicates localization of cyst.
she made that noise. She replied that it hurt her in the chin. N—vocalization in same tone but clonic as though regularly interrupted. O—vocalization in definite tremolo. Patient was saying “I can’t do—” and carried the “do” over into the sound of vocalization as the area was stimulated. When asked again why she made that noise she replied “I don’t know.”

The stimulation of O was repeated for longer duration. Vocalization resulted until the patient’s breath seemed to be exhausted, whereupon a small epileptiform attack was reported, consisting of pulling of nose, mouth and face to the right, and inward rotation of the right arm.

The size of the area (M, N, O in figure 4, or V in figure 3) from which vocalization was obtained was not greater than from 6–7 mm. in diameter. It was observed that stimulation at O produced a sound different from that at M. It was discontinuous so that it sounded like speech but one could make out no words.

When the patient was told to count to twenty stimulation at a little distance did not influence the counting. When the electrode was brought nearer she seemed to continue the “eleven” sound. Stimulation below M during the counting caused her to stop on two trials. When asked why she stopped she replied, “I tremble so.”

The electrical exploration was continued and it is interesting that sensation in the chin and right face which was found to accompany vocalization when M and O had been stimulated, was produced by stimulation at a distance over the surface of the cyst at T, 5 and 7 with no vocalization. Stimulation at 2 and 3 produced sensation in both sides of the face, in chin and tongue, and slight closure of mouth in clonic manner. These stimulations just mentioned were upon the translucent covering of a fluid-filled cyst. After removal of the cyst this translucent material was found still to contain nerve fibres. This is an example of the spread of excitability that so often is found in cases of epilepsy. It may be called epileptic facilitation perhaps. In this case it seems evident that it is produced by activation of superficial nerve fibres which cross the surface of the cyst to the postcentral convolution at F and G, as there was no grey matter present and no other fibre layers.

**GENERAL OBSERVATIONS ON CORTICAL STIMULATION**

Simple movements, when they are produced, appear to the patient to be quite involuntary. In our whole series of patients we have found none who was under the impression that he was carrying out these movements of his own volition. A public school teacher remarked about the movement that had been produced in her face, “It seems involuntary.” And another intelligent young woman observed, “My leg moved itself.”

Likewise, sensation when produced electrically is referred at once to the periphery as an unexplained sensation.

I have often asked a patient to make an effort not to move the hand, face or foot and followed this by stimulation of the motor area for this part. The result is usually a movement over which he has no control and he is never at any time in doubt about this. With or without warning restimulation of that area will ordinarily reproduce the same movement.

On the other hand, a patient is sometimes able to prevent a movement by an act of will power. In the case of F. W., stimulation of area 6a beta of Brodmann (Fig. 3), which Vogt has named the frontal adversive field, produced closure of the right hand and a hot feeling down the right side of the body. The patient was instructed to make an effort to keep his right hand still if he could do so. The stimulation was repeated without warning the patient. He reported the same feeling of a flush in the right side of his trunk and he held his hand quite still. A somewhat more complicated example of a patient’s effort to oppose the effect of cortical stimulation is the following.

The cortex of an intelligent young girl, H. T., was stimulated anterior to the lower end of the right precentral gyrus while she was counting from one to forty. At the time of stimulation the patient hesitated slightly and the observer reported that there was pulling of the mouth to the left. She continued to count, however. When her task was finished the patient stated to me that, “It was hard to continue.” She was able, therefore, to continue to count although the left side of her mouth had been forced to go into involuntary movement by stimulation of the right cortex.

Patients sometimes state that they have a strong desire to move which they are able to control, or at all events do control. F. S., an Italian who found some difficulty in expressing himself in English, after stimulation at nearby points in area 6a beta (Fig. 3), made the following observations: (1) “My nerves shook all over; wanted to pull me to the left.” (He showed agitation probably because he thought he was about to have one of his habitual epileptic fits.) (2) “Wanted to fall to the left side.” (3) “Head feels like it wants to move down to left leg and left leg up to head.”

After the patient, R. M., had been subjected to stimulation in the same area, Vogt’s frontal adversive field, he said that he had felt as though his “eyes were going to turn to the left.” There was, however, no turning of his eyes at that time. Another patient, H. T., when stimulated in the same field on the right side, said she felt as though she wanted to turn her head to the left. She made no obvious movement.

The patient J. H. tried to resist movements produced by stimulation of areas 4 and 6 of Brodmann without success. However, stimulation
at a distance produced downward plantar flexion of the opposite foot together with an aura of an habitual attack. This was repeated without warning. The patient explained that he had a desire to move the foot down at that time. He was asked to resist it and stimulation of the same point was repeated. The patient's foot moved upward in the opposite direction and the whole leg was drawn up in an exaggeration of opposition.

On the other hand, at times a patient may have the feeling of movement without there being any observable change of position of the part. F. W. stated that at the time of stimulation of the left postcentral lobule he had sensation of movement in the right thumb. No movement in the thumb could be seen, however. Stimulation at this point was repeated at a later time without warning to the patient and he reported the same sensation.

Stimulation may sometimes produce a feeling of paralysis in a part without any objective change in the appearance of that part. For example, H. M., when stimulated in the left postcentral lobule reported that he felt a tremor in the upper lip on the right side, and that he lost control of the lip. There was no obvious movement. When the right precentral lobule was stimulated, H. T. reported that she was unable to speak and unable to open her eyes. This stimulation was at a point just below an area where marked closure of both eyes had been produced. This sort of response may possibly indicate the production of an inhibitory discharge.

When a sensation is produced from stimulation of the cerebral cortex it is usually described as numbness or electricity. Quite exceptionally the patient uses an adjective which suggests a feeling of cold or warmth. For example, when the cortex of the patient D. R., an intelligent young boy, was stimulated in the left postcentral region, he reported at 2 "electricity" in the thumb and first two fingers of his right hand. At 4 he said he felt something cold at the right side of his mouth, both inside and out. At 5 he reported a feeling of electricity in the thumb and first finger. At 7 he stated there was a feeling inside of his mouth in the lower jaw, which he described as a "sticking" sensation. The feeling of heat down the side has been reported above under the patient F. W.

Pain has been produced by stimulation of the cerebral cortex, particularly in the frontal adverse field, but this has been in relation to the aura of an epileptic seizure which perhaps it would be better not to discuss at this time. Stimulation of the olfactory lobe causes the patient to exclaim with some surprise that he smells something. Patient E. M. said the odor resembled "oxygen." Patient F. R. said that it smelt like "something burning."

More complicated sensations may also be produced. For example, the patient J. C. said that she felt afraid and that she had a sensation in her abdomen, when the anterior end of her second temporal convolution was stimulated. The next stimulation at an adjacent point produced a train of phenomena which must be considered epileptiform but which illustrates a number of points made above. The patient had first a feeling in the abdomen, next the feeling of fear and then a desire to move the left hand. There was a plucking movement with her left hand. When she was asked, "Why do you move your left hand and not your right?" she replied, "That one wants to." She added that the movement was "involuntary." This stimulation was repeated several times without warning but with the same result. On one occasion she was warned that the stimulation was coming and was urged to keep her left hand still, if she could. On this occasion she reported the same sensation but kept her hand quite still. During this stimulation she cried a little; afterwards she said she wanted to move her hand but prevented herself from doing so. It should be added here that the feeling in the abdomen and the feeling of fear were phenomena which sometimes appeared in the epileptic seizures to which this patient was subject.

What do these observations indicate as far as the relationship of consciousness to the cortex is concerned? Crude motor movements are made by stimulation of the pyramidal and parapyramidal cortical motor areas but the individual is fully conscious that he has not done it—he has not willed it. He says rather, that it was involuntary, or the hand moved itself. If a bit of sensory cortex is activated the patient feels a crude sensation in the hand but it never occurs to him that he has imagined it. He hears his own voice crying as the result of stimulation and when he has finished he exclaims in surprise that his voice was drawn out of him, and next day he may live over the experience in a dream that someone is making him speak.

As far as introspection is able to go the patient concludes that the activity of his own stimulated cerebral cortex is on a plane quite distinct from his own conscious thinking. The electrode of the surgeon may provide him with a new sensory experience or it may initiate a motor movement against which he is able to struggle by means of such other cortical mechanisms as are still at his disposal.

But before generalizing any further a brief consideration of certain aspects of epilepsy may be interposed.

**Epilepsy**

"Epilepsy," Hughlings Jackson said, "is the name for occasional sudden, excessive, rapid and local discharges of grey matter." As this discharge
may take place in different areas of the nervous system, so the character of the discharge may vary greatly from movement to sensation and from dream to mental lapse. But in general the movements are violent and purposeless, the sensations crude and the dreams simple.

Jackson conceived that during a seizure there was a discharge in the grey matter of the brain which began at some local point and spread from that point, producing a march of outward phenomena. This conception had two results. Firstly it provided an hypothesis for the understanding of epilepsy. Secondly, it provided him with a key to the localization of function in the brain. By the study of epileptics he concluded that cerebral convolutions represented the function of certain peripheral parts, ten years before Hitzig and Ferrier demonstrated this to be a fact by electrical stimulation of the convolutions of animals.

Consider as an example the "Jacksonian" seizure which begins by localized movement of the great toe and spreads in succession to the leg, arm, hand and face of the same side. The discharge of the pyramidal cells in the toe area of the precentral gyrus progressively spreads downward through the length of the gyrus. In so doing it maps out the contiguity of cortical representation of these parts. The spread is from one area of grey matter to another, rather than along internal association pathways to more distant centres.

Another example may be cited. An epileptogenic focus in the posterior part of one temporal lobe (Penfield 1935) produced in a patient the following habitual chain of phenomena: (1) the hearing of a roaring noise, (2) a sense of dizziness, (3) salivation and (4) micropsia or the hallucination that everything looked small. Of these phenomena two are sensory, one motor and one hallucinatory. Discharge of certain areas of the brain has produced each. But the spread has taken place in this manner not because special association pathways exist between these areas. The spread has followed the pattern described because of contiguity of representation.

Epileptogenic discharge of ganglion cells is sudden, overwhelming and undiscriminating, as though they were for the moment affected by a miniature hurricane. The result of this focal hurricane indicates that there exists focal representation in the brain and indicates which areas are neighbors. The site of the initial discharge must be sought by the clinician. The cerebral cortex apparently is not the only region where epileptic discharge may take place. It may involve the grey matter at other levels in the central nervous system including the diencephalon and midbrain, and even on rare occasions the spinal cord.

An epileptic seizure is in some ways the direct opposite of paralysis. A unilateral seizure is on some occasions followed by unilateral paralysis which usually affects the part that was earliest and most severely involved in the seizure. Such a post-seizure paralysis may consist in a monoplegia, a hemiplegia, an aphasia, a mental stupor or even complete coma and diplegia.

The cause of these post-seizure paralyses was thought by Jackson to be fatigue of the discharging cells. It was suggested by Kinnier Wilson to be a phenomenon of after-discharge inhibition. The present evidence suggests that these paralyses are due to post-convulsive spasm of the cerebral arteries or at least to the focal cerebral anemia which frequently follows epileptic seizures of whatever variety (Penfield 1933).

The important point to be born in mind is that, except during the duration of complete coma and diplegia, these paralyses are due to focal or local inactivity of nerve cells just as the epileptic discharge is due to regional activation of the same. Thus the post-epileptic negative state may have for our present purpose a localizing value similar to that of the positive local discharge of a focal epileptic seizure.

It should be remembered, further, that there is paralysis of voluntary function in any part of the brain during epileptic discharge in that part. If it be an area which is too complex to express itself during the discharge there will be no evidence of the fit other than absence of function. A discharge occurring within the speech area signalizes its occurrence only by silence, by the inability to speak. This has the same outward effect as post-epileptic paralysis although the cause is quite different.

Epileptic disturbances of consciousness will be taken up under the headings of dream state and automatism. Further discussion of loss of consciousness in epilepsy will appear in the final section on generalities.

**DREAM STATES**

Hughlings Jackson applied this term to alterations of consciousness which appear in epileptic states, alterations without loss of consciousness. In a dream state a patient may have a sudden feeling of strangeness, of unexplained familiarity, but some capacity is retained for conscious insight, and he may know that this is another fit. He may suddenly see a complicated scene which in fact comes from some past experience, but he can still reason and is aware of the unreality of the condition. Jackson called this double awareness mental diplopia.

An example of such a dream state may be given somewhat in detail: J. V., a young girl of 14 years, had suffered from epileptiform seizures from the age of 11, characterized by sudden fright and screaming when she would hold on to people for protection. This was followed by falling and occasionally a major convolution. In infancy, at the close of an anesthetic, she had had a single convul-
Following a typical but severe seizure which was induced after her admission to the hospital she had transient weakness of the left side and a positive Babinski sign on the left side. On careful questioning it was learned that during the preliminary period of fright she invariably saw herself in a scene that she remembered at the age of 7 years.

The scene was as follows: A little girl was walking through a field where the grass was high. It was a lovely day and her brothers were walking ahead of her. A man came up behind and said, "How would you like to get into this bag with the snakes?" She was very frightened, screamed to her brothers and they all ran home, where she told her mother about the event. Her mother remembers the fright and the story and the brothers still remember the occasion and remember seeing the man.

Following that she occasionally had nightmares in which the scene was re-enacted. Three or four years later, at the age of 11, she was recognized as having attacks by day and in the attacks she habitually saw the scene of her fright. She saw a little girl whom she identified with herself in the now-familiar surroundings. She was conscious of her environment at the time of the attack and would call those present by name and yet she saw herself as a little girl with such distinctness that she was filled with terror lest she should be struck or smothered from behind.

At operation under local anesthesia adhesions between dura and arachnoida were found indicating an old subdural hemorrhage and cortical atrophy most marked in area 19 of the occipital lobe, the area from which her curious complicated aura could be reproduced. This change was probably due to a hemorrhage in infancy at the time of the anesthesia.

The central fissure was mapped out as seen in figure 5, from Y to M. At point 14 and also 17 stimulation caused her to state that she saw stars on the opposite side. At 16 and 14 the aura of an attack was produced. After stimulation at 16 she said, "Wait a minute and I will tell you." Then a little later she said, "I held on to the bar" (as she had been asked to do) "and the bar seemed to be walking away from me. I saw someone coming toward me as though he were going to hit me." In a moment she called, "Don't leave me." The point 14 was stimulated without the patient's knowledge. She suddenly said, "Say something."—then a little later, "I had the funny feeling. It was like an attack."

At another point nearby, but from which the mark was accidentally displaced before the photograph was taken, stimulation had previously produced her first aura. She stared suddenly and then cried, "Oh, I can see something coming at me. Don't let them come at me." She remained staring and fearful for 30 seconds, although the stimulation was of much shorter duration. A little later she said, "It didn't feel like an attack at first but right after it felt like an attack coming on; it sort of started and then passed off. First time it has

Fig. 5. J. V. Girl of 14 years suffering from epileptiform seizures ushered in by a complicated aura which could be initiated by electrical stimulation at points 14 and 16. Brain photographed during operation.
felt like an attack since last Wednesday." Wednesday had been the day when several seizures were induced.

Stimulation was carried out at a number of points from the first temporal convolution backward through cortical area 22 to the site of the origin of her aura in area 19 (Fig. 1). These stimulations caused the patient to cry out that she heard a large number of people shouting. Once she said, "They are yelling at me for doing something wrong; everybody is yelling." On enquiry she said she could hear her mother and brothers. Stimulation at 2 (Fig. 5) caused her to say, "I imagine I hear a lot of people shouting at me." The stimulation was repeated twice without warning and for not over 2 seconds. Each time the voices were heard again, the duration of the voices being 8 and 7 seconds respectively. A third time the stimulation was repeated without warning and she said, "I hear them again." At 10, "Oh, there it goes, everybody is yelling," and after an interval, "Something dreadful is going to happen." At 11, "There they go, yelling at me, stop them!"

If her epileptogenic discharge began at 16 or 14 and passed forward over points 11, 10 and 2, she would have had her typical hallucination, felt the dread and eventually heard accusing voices before any convulsive phenomena were observed. As she did not remember the voices in an ordinary seizure it seems likely that she had an amnesia for this part of the march, not an infrequent occurrence in this region.

This is an example of the reproduction of a complicated memory of an event which actually occurred between the time of receiving an injury to the brain and the onset of the seizures. This is not without precedent. I have studied a patient whose attacks were originating somewhat farther forward in one temporal lobe as proven by stimulation and who saw herself giving birth to a child during the aura. The picture she saw reproduced the surroundings of that event as it had actually happened.

A somewhat different type of complicated aura may be mentioned briefly. M. B., an intelligent young woman of 30 years, suffered from epileptiform attacks characterized by (1) an hallucination of being far away or of seeing things small, (2) dizziness or sensation of turning, (3) tinnitus, (4) numbness in right hand, all followed at times by loss of consciousness and a generalized convulsive seizure. At operation, in area 5b, a somewhat yellowish soft convolution was found and from this convolution and its vicinity her aura could be reproduced. In figure 6 the numbers 3 and 4 are laid upon it.

Stimulation of the post-central convolution at 1 produced numbness in the thumb and at 2 numbness in the right side from axilla to umbilicus; 4—"Felt like I was going far away; sometimes like that before an attack. Like when things look far away." 3—Dizziness "like before an attack." 15—"Felt as though I was falling out of bed." The stimulation was repeated without warning. She
said suddenly, "Yes, the same thing—felt as though I was falling."

If the epileptogenic disturbance began at 4 and moved to 3, then 15 and finally 2 and 1, she might well have complained first of an hallucination of being far away, next of dizziness, then of falling, and finally numbness in the right hand. If the disturbance had spread still further forward there would have been convulsive movements.

In such cases it may be said that there is an alteration in consciousness but actually there is a new phenomenon being presented to the conscious individual. In the case of J. V., this phenomenon arises from a discharge that is occurring within the cortex of cytoarchitectural area 19. At the same time that patient, because she retained consciousness, differentiated between the spurious phenomenon which might be called synthetic and the actual existence of the outside world as presented to her by the unoccupied regions of the cerebral cortex. In the case of M. B., a disturbance originating in area 5b gave her the hallucination of being far away and yet she retained an understanding of her surroundings.

AUTOMATISM

Quite different from the above dream state is the post-epileptic automatic state. The subject is in full control of his body but does not know what he is doing. He may respond if spoken to. He may obey a command or may resist violently and dangerously any interference.

You may prefer to call this an alteration in consciousness but it seems to me a loss of consciousness on an exceedingly high level, if the expression is permissible. He has no present responsibility nor will he have future memory of what he does.

A brief example may be cited. An intelligent and sensitive young patient gave a history of major seizures sometimes followed by violent behavior which distressed him as well as his parents. While in bed on the public ward of the Royal Victoria Hospital he had a seizure during his sleep, characterized by convulsive movements of both sides and frothing at the mouth. He then got out of bed and without putting on his slippers or bathrobe he began to look for something under his bed—possibly his slippers. He walked about and then got into bed with another patient. When nurse, doctor and prospective bed-fellow opposed him he fought them violently. On coming to himself he had no recollection of the incident and was chagrined to learn of it.

Jackson considered such a state to be invariably due to post-epileptic paralysis, paralysis of the highest level of neural activity, the substratum of consciousness. He therefore considered the state to be a phenomenon of release from higher control. If it is in truth post-convulsive paralysis I would suggest, on the basis of what we now know, that there remains an effective anemia in the region wherein consciousness is represented. Sometimes the preceding seizure is very slight indeed. At other times there is no evidence of any such seizure. But the same is true in other spheres. A hand may become suddenly paralyzed without sign of convulsion, doubtless due to a spontaneous anemia without preceding epileptogenic reflex.

It is quite possible, however, that it is not always post-epileptic. If an epileptic fit may take place in the same level or area of the brain as the post-epileptic paralysis, then automatism must be possible during a fit caused by discharge in this highest level as well as in the post-discharge period. Some petit mal seizures must be of this nature.

GENERALITIES

For the purposes of this discussion it seems to be unnecessary to inquire into the mechanism by which a conscious decision receives its initial activation in neuronal conduction or even to inquire as to whether such decisions are reflex. It is enough to be able to recognize when an individual is conscious and when he loses consciousness, to be able to describe what portions of the central nervous system may be paralyzed without abolishing consciousness and what portions may be still active when consciousness is gone. Finally one may hope to indicate those areas which are necessarily inactive when consciousness is abolished by a paralyzing lesion.

In the epileptic's dream state as described above, an hallucination is presented to him by discharge within one portion of the cerebral cortex but the patient retains insight into his real environment due, no doubt, to normal function in other parts of his cerebral cortex. He may even be able to say to an observer (as J. V. did) "Wait a minute," and after the dream is over to recount it in detail. This may be in a sense a doubling of consciousness but it is not a loss. In the post-epileptic automatic state the patient still has cortical mechanism intact, and furthermore he still has co-ordinating control of those mechanisms. He is a perfect machine but he has either lost consciousness exclusively or there has been a temporary removal of one element of consciousness. By analogy, this must either be produced by paralysis of a small area of the brain or by paralysis of scattered mechanisms with a unit blood supply.

Most often consciousness is lost by such patients in association with certain epileptic ple-
neural

neurological phenomena and as one part in the habitual pattern. In general, an epileptic disturbance may spread a considerable distance over the cortex, especially the post-central cortex before consciousness is lost. On the other hand, the loss of consciousness during a true seizure may be primary without any manifestation other than a blank expression and arrest of speech (petit mal). The return of consciousness in such a patient may be without sign and he may continue his train of thought without knowledge of the gap. The neurological mechanism, temporarily inactive, must be the same or similar to that which is paralyzed in automatism.

In a more severe petit mal seizure there are usually, as pointed out by Jackson, certain associated phenomena such as "deep pallor and a slight wave of universal movement." Such a lapse is sometimes regularly associated with loss of the mechanism for maintaining erect posture and the patient suddenly falls to the ground. If there is an aura preceding simple loss of consciousness it is most often an epigastric or a visceral one.

Let us consider for the moment that consciousness has a localizable representation in the brain: like movement, vision, hearing and speech. From the nature of the associated phenomena just described, one might suggest that that representation finds its topographical localization near to the representation of autonomic function in the hypothalamus and close to the third ventricle from which region facial blanching might be produced and where visceral sense may be represented and adjacent to the upper end of the nerve circuits in the midbrain which maintain standing. This topographical localization does not signify a belief in a punctate centre but rather a general region. The exact position may be wrong but the reasons to search for such localization are valid.

We may make another observation about consciousness and epilepsy. It is invariably lost at the beginning of those attacks in which the convulsion is generalized from the start. Now if there is an as yet uncharted area of the brain where all sensory and motor processes are re-represented epileptogenic discharge here would result in involvement of all somatic and visceral functions simultaneously and would obliterate consciousness from the beginning.

Hughlings Jackson found this problem a favourite one. He often quoted from Herbert Spencer these words: "The seat of consciousness is that nervous centre to which mediately or immediately the most heterogeneous impressions are brought."

On a lower functional level vision has its nervous mechanism in the occipital lobes and hearing in the temporal lobes. That is, a sound produces a neural activity in the temporal lobes which forms the basis of hearing. In the motor region the neural activity forms the basis for voluntary movement. If we had no other evidence, knew no anatomy of tracts, had never used an experimental animal, the study of human epilepsy would have shown us these facts concerning voluntary movement, hearing and seeing.

In an analogous manner the evidence from the study of epilepsy suggests that there is such a region where those neural activities converge, which are the indispensable substratum of consciousness. The elements are there both for sensation and for the initiation of movement.

But perhaps it would be well, before yielding to the temptation of anatomical localization, to reconsider the physiological and psychological results of cortical stimulation independent of epilepsy.

The phenomena produced by cortical stimulation are crude. The movements involve many different muscles that depend on a very wide innervation in the brain stem and spinal cord, and yet the result is simple flexion or extension of a part or a turning movement which accomplishes nothing purposive. If stimulation of a cortical motor area is carried out during the execution of some voluntary act by the subject, the motor mechanism in question is snatched away from his control, but he may continue to carry out the act if there are other mechanisms available not so stimulated. Thus the girl (H. T.) continued to count aloud even though one side of her mouth was caused to contract in the useless manner of a motor discharge. She said she found it hard work but she succeeded, no doubt due to the fact that she was using the other side of her mouth appropriately by means of the mouth motor mechanism from the opposite hemisphere.

If, however, vocalization is being produced by stimulation of one hemisphere, the larynx, diaphragm and all the accessory muscles of phonation are pre-empted by this discharge. The individual cannot speak or alter the vocalization however hard he may try. A homologous vocalization mechanism exists in the opposite hemisphere but the final common path of this discharge has already been pre-empted by an electrode.

The demand of an electrode is not always irresistible. Although stimulation of Brodmann's area 4 or 6 produces a movement which the patient cannot inhibit, nevertheless in the epileptic brain stimulation at a distance from the motor cortex sometimes produces what the patient describes as a desire to move a certain part. By making a conscious effort he can prevent such movement and in his effort to do so he may, at the time of stimulation, make the opposite movement (J. H.). On the other hand, if the fingers of one hand are caused to close by an irresistible electrical stimulus the patient can quite easily use the other hand in an efficient manner to try to force the fingers open.

An electrode can produce no more evidence of a
directed, purposeful, skilful movement than is to be found on the mockery of action produced by an epileptic cortical discharge.

Sensory and hallucinatory phenomena of a more complicated nature may be produced in the posterior two-thirds of the hemispheres, but only when those phenomena have previously formed a part of a recurring epileptic fit which seems to prepare that portion of the cortex for local conditioned reflexes. The stimulation seems to reproduce a familiar chord the harmony of which has become the characteristic expression of that area of cortex when stimulated.

The above discussion applies to the sensorimotor cortex. No mention has yet been made of the frontal lobes anterior to areas 4, 6a alpha and 6a beta of Brodmann. This is what Campbell (1905) called the frontal and prefrontal cortex (Fig. 7). It is dangerous to give this area a name because of the confusion of terminology that already exists, as pointed out by Walshe (1955), but for convenience of discussion it may be called the extra-motor frontal cortex.

In regard to this portion of the frontal lobe Pavlov wrote that it should (probably) be included with the rest of the cerebral cortex. But a clearer impression of his eventual opinion may be gathered from the letter of his pupil, Professor Boris P. Babkin, who wrote to me as follows: "Many of the experimental data obtained by you on man point in the same direction as the facts obtained by us in Pavlov's laboratory, namely that the cortex is primarily a representation of the receptors scattered over the external and internal surfaces of the body. However, in the last years of his life Pavlov came to the conclusion that the frontal region of man serves for higher neural activities than the formation of conditioned reflexes. Thus, if a conditioned stimulus is regarded as a signal, there also may be formed 'signals of signals,' e.g. words. The 'centre' for the formation of these signals of signals would be the
frontal region. Pavlov never touched the problem of consciousness. At any rate in the dog it is not located in the frontal lobes, as I know from my own experiments involving extirpation of this part of the hemispheres."

Hughlings Jackson suggested that in the "pre-frontal" area was to be found the highest level of re-representation. But he suggested this in the same tentative manner that he once suggested, in the days before Hitzig, that motor and sensory representation would be found in the corpus striatum.

In my own experience no convulsive phenomena have been produced by electrical stimulation of the extra-motor frontal cortex as yet, and no responses of any sort have been produced other than what may be called autonomic alterations, but these so rarely as to make conclusion premature. Possibly other forms of electrical current may give a different result. But, so far, no alteration and no arrest in consciousness have been produced here even by strong stimulation, and neither has been reported by others.

Fits which originate from focal lesions in the frontal pole are characterized, it is true, by initial loss of consciousness followed by aversive movements before the appearance of generalized convulsions. Radical extirpation of the whole of one frontal lobe, including all of the extra-motor cortex and back into area 6a beta, may be carried out in a conscious patient without his losing conscious insight during the procedure or memory of the details of the event afterward. After immediate recovery the most important detectable sequel, in my opinion, is "impairment of those mental processes which are prerequisite to planned initiative" (Penfield and Evans, 1935).

If this large area of cortex in both frontal lobes were necessary for the existence of consciousness it seems likely that there would be at least a temporary loss of consciousness during the removal of one lobe and until the remaining lobe could take over the function of both. And yet one occipital lobe may be removed without the patient being aware of any interference with vision or of the reduction of his visual field. Furthermore, the fact that epileptiform discharge in the extra-motor frontal cortex obliterates consciousness may be likened to the fact that such discharge, if it be a strong one, in one occipital lobe may produce complete temporary blindness, a blindness that applies to the whole visual field and the patient says everything is dark.

All of this would suggest that the frontal lobes anterior to recognized motor areas are utilized in conscious processes but that they are not indispensable to the existence of consciousness. It is conceivable that these lobes represent an elaborative field for a more essential concentration of nerve tracts which lie posteriorly and more centrally in the brain. But it should be pointed out that stimulation of this area of the brain in man has cast no light upon its function as yet and I should like to exclude the extra-motor frontal cortex from any conclusions reached here in regard to the remainder of the cerebral cortex.

Long-continued unconsciousness appears clinically in patients who have a lesion of an area somewhere above but not far removed from the midbrain and in the vicinity of it. Details will be omitted here, but twice, after posterior fossa operations, patients of mine have gone into a condition eventually resembling sleep, in which they lived several months with stomach-tube feeding. In another case a similar state followed operative removal of a tumour which extended into the pulvinar and which was followed by unconsciousness from which the patient began to rouse after six weeks of artificial feedings. Such localization as has been achieved for sleep, normal and pathological, also implicates this general region (Hess, 1932).

It is, of course, impossible to state that in such unconscious states as those just described all sense perception is gone. One may conclude no more than that volitional aspects of consciousness are non-existent and that no later memory of the state persists. In a discussion of this general field with Professor Charles Hendel he has pointed out to me that in one sense I cannot escape the formulation of some definition of consciousness, at least by implication, and that my argument applies particularly to volitional consciousness or the motor element of consciousness. It is true that if there is such a thing as sense perception or pure sensory consciousness in the absence of all volitional capacity the evidence produced in this study seldom applies to it, and patients have no memory of such a state. Nevertheless, after an epileptic seizure a patient may say he heard and saw what was happening during the seizure but could give no outward sign. The physiological explanation that seems evident for this state is that the necessary cortical motor mechanisms were taken away from him by the epileptic disturbance while certain sensory cortical mechanisms were not so pre-empted. At such a time the patient could hardly be called unconscious.

The common published conception of the cerebral cortex seems to be that it represents the highest level in the scale of nervous activity. The objective evidence derived from a study of epilepsy and from the study of conscious patients during brain operations supports the view of Hughlings Jackson that the sensori-motor cerebral cortex represents only a middle level of integration.

Man is one of a million species of animals, some of which at least would seem to be conscious. Parker (1934) pointed out the conservatism of
nature in regard to changes of the central nervous system as compared with the radical variety of outward form. The human cerebral cortex has developed coincidentally with man's acquisition of new skills and new adjustments to his environment. *A priori,* there seems to be no reason why the neural mechanism essential to consciousness should migrate outward into the newly exfoliated hemisphere.

In a conscious individual it must be from somewhere that neuronal impulses pass to the motor areas of the hemispheres producing complicated behaviour which cannot be simulated by electrical stimulation anywhere over the cortex. Hallucinations involving elaborate memory of visual and auditory pictures can be produced by such stimulation but the patient retains conscious insight into the unreality of the experience. The same epileptic and post-epileptic processes which paralyze discrete areas of the cerebral cortex also may abolish consciousness. Therefore it seems reasonable to assume that there is a discrete area of the brain the integrity of which is essential to the existence of conscious activity.

Finally, there is much evidence of a higher level of integration within the central nervous system that is to be found in the cerebral cortex, evidence of a regional localization of the neuronal mechanism involved in this integration. I would suggest that this region lies, not in the new brain, but in the old, and that it lies below the cerebral cortex and above the midbrain.

Such localization does not signify that other parts of the brain play no rôle in this mechanism. All parts of the brain may well be involved in normal conscious processes but the indispensable substratum of consciousness lies outside of the cerebral cortex, probably in the diencephalon.

This discussion has avoided the subject of the nature of consciousness. That is a psychological problem. It has been concerned with the localization of the "place of understanding" and by place is meant the localization of those neuronal circuits most intimately associated with the initiation of voluntary activity and with the sensory summation prerequisite to it.

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