Critical Review.

THE CEREBRAL CORTEX DURING UNCONSCIOUSNESS:

A critical review of the theory of conditioned reflexes, with reference to the symptoms of epilepsy and narcolepsy.

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IN view of the recent experimental and clinical work on brain function, it is perhaps permissible to raise the question of consciousness and unconsciousness in a form devoid of speculative ideas based on introspection. The brain researches of Pavlov and the clinical studies of Kinnier Wilson have important points of contact, already indicated by the latter in his *Modern Problems in Neurology*, from which conclusions may be drawn regarding the neurological basis of unconsciousness.

Pavlov and his collaborators, working on dogs and on human beings, have shown clearly that reflex action is not confined to the lower nervous centres. They have demonstrated in thousands of experiments that the cerebral cortex is the seat of a particular kind of reflex action, which is interlaced with that of the lower nervous centres in the manifold, complex, and ever changing activities of an animal. Without going into great detail of this work, the main conclusions may be stated.

The reflexes elaborated through the cortex depend for their existence on the association of their stimuli with the stimuli for reflexes of the lower nervous centres. For this reason the reflexes of the cortex are termed *conditioned reflexes*, and those of the lower nervous centres, *unconditioned reflexes*. Conditioned reflexes are unstable, temporary, and not inborn, in contrast with unconditioned reflexes, which are stable, constant, and present from birth.

Reflexes are 'regulated responses to the external world.' Every reaction of a higher animal is a reflex, of the conditioned or unconditioned type. Any agent of the external world can act as the stimulus, or signal, for a conditioned reflex, by which a 'temporary union' is established between this phenomenon of the external world and the responding animal organism. The finer differentiation made by an animal between different components of the external world is made possible by the activity of the cerebral cortex, which thus has an analysing function.

At first, when a conditioned reflex is set up, there is a 'generalization of stimuli,' with a comparative lack of differentiation between allied (similar) stimuli. Later, with repetition of the stimulus for the conditioned reflex, always associated ('reinforced') with the unconditioned stimulus, and with a contrasting of this reinforced conditioned stimulus with the allied stimuli not so reinforced, a differentiation is established between the one specific stimulus and other agents of the external world. This process of differentiation is described by Pavlov, who says¹: "It is a well-known fact, which may be observed with all agents (of the external world), that when they have just become conditioned stimuli their action, during the first days, is very general, i.e., there now act as conditioned stimuli (calling out the same effect as the original stimulus) all phenomena similar or akin to the elaborated conditioned stimulus. Only by and by, under the influence of definite circumstances, does the conditioned stimulus become specialised (specific), i.e., the action of all those accessory stimuli, which do not coincide with the conditioned reflex [read stimulus] is obliterated by the process of inhibition, and only the conditioned stimulus remains active."

What is this process of cortical inhibition by which an animal is enabled to differentiate between allied (similar) stimuli? It is, according to Pavlov, complementary and opposite to the process of cortical excitation. He expresses the view², "which occupies an ever larger place in physiology, that inhibition always follows excitation, that it is in a certain sense the reverse side of excitation."

How are the antagonistic processes of excitation and inhibition distributed in the cortex? The only answer from experiments on conditioned reflexes is that irradiation and concentration of excitation and of inhibition around various points of the cortex are constantly occurring. As excitation irradiates from a point of the cortex in the initiation of a conditioned reflex by a specific stimulus, other allied stimuli also become capable of evoking the reflex. As excitation concentrates, being followed up by inhibition, the allied stimuli fail to evoke the reflex, only the one specific stimulus producing it; that is, differentiation between stimuli occurs. Pavlov here describes³ this supremely important conception of cortical activity: "The process of analysis and the process of differentiation must be presented thus: If our chosen special agent of the external world is brought for the first time into connection with a definite physiological function, then the stimulation called out by this agent, coming to a certain point of the cortex, irradiates or spreads over the corresponding receptor centres; and thus not only the single point in the brain end of the given analyser* enters into the definite connection, but the whole analyser

* "Under the name 'analyser' Pavlov includes as a functional unity the surfaces of the body receiving the stimulation from the outer world (sense organs), the nerve or nerves conveying the impulse to the central nervous system and the cells in the central nervous system to which this process flows it also is endowed with the ability to unite several elementary phenomena into one complex stimulus, i.e., it performs synthesis as well as analysis." (Translator's note, *Lectures on Conditioned Reflexes*, p. 117).

or a greater or smaller part of it. And only later, owing to the opposition of the inhibitory process, does the field of influence of the stimulation become smaller until at last an isolated action is obtained."

Many experiments illustrating the process of irradiation and concentration of excitation in the cortex are recorded by Pavlov in his two published works in English, *Conditioned Reflexes* (1927), and *Lectures on Conditioned Reflexes* (1928). Here is an example.⁴

"If you form a conditioned reflex, for example, to the ticking of a metronome, and then try other sounds, you will find that these other sounds at first also produce the salivary flow. Consequently the stimulation from a certain group of cells irradiates over a large part of the cerebrum, and therefore every other auditory stimulus provokes the secretion of saliva. If you make a conditioned stimulus from a tone of 1,000 vibrations, and afterwards try other tones of various vibrations, all of them have an effect. . . . Besides the law of irradiation, there is another law, that of concentration of excitation, i.e., the irradiated excitation gathers along certain lines and towards certain foci. This is a fact which is seen in the laboratory every day. If you have formed a conditioned reflex to the metronome and then repeat this reflex many times, other sounds gradually lose their effect, and at last only the metronome calls out the excitation. And this concentration of the excitation proceeds still further; if you repeat the stimulation with the metronome long enough, it finally happens that only the metronome with the number of strokes you have constantly used will be effective; the dog may react to your stimulation of 100 ticks per minute, but not to one of 96. . . . In these cases of high concentration of the excitation, besides repetition of the given stimulus it is also important to repeat the other neighbouring and related stimuli, but without feeding (i.e., without the corresponding unconditioned reflex)."

Irradiation and concentration of inhibition, as well as of excitation, are also constantly taking place. The cortex, then, consists of regions of excitation and inhibition, now irradiating, now concentrating, according to the conditions and changes of the environment.

It has already been mentioned that excitation of a particular region of the cortex is followed by inhibition of the same region. Not only does this happen, but excitation in one region causes a more or less rapid development of inhibition in surrounding regions. This type of development of inhibition from excitation (and vice versa) Pavlov has termed *induction* (following the terminology of Sherrington⁵). In the case of a point of excitation causing adjacent surrounding inhibition there is 'negative' reciprocal induction, a process which has been demonstrated in a series of recorded experiments.⁶

Pavlov thus describes⁷ reciprocal induction:—

"An excitation arising in a certain place causes an inhibitory process around this region and owing to this the spread of the original excitation becomes limited. On the other hand, the inhibitory process induces an

excitatory process, and this in turn checks the spread of the inhibition. Thus the whole cortical area is partitioned off into excited and inhibited points."

Inhibition is found to be more labile than excitation; its irradiation proceeds more freely than that of excitation. Hence in 'negative' induction the spread of inhibition from around a focal point of excitation is relatively wider, and may be complete. "Negative induction," writes Pavlov,⁸ "consists in this, that a stimulation of the cortex at one point leads to inhibition to the rest of the cortex." As we shall see, this process of 'negative induction' may be highly significant for the interpretation of certain clinical symptoms.

SLEEP.

According to Pavlov,⁹ it is the spread of inhibition over the whole cortex which is the basis of sleep; "The fundamental condition of the appearance and development of internal inhibition and sleep is exactly the same. It consists in the more or less prolonged or many times repeated isolated action of a conditioned stimulus producing stimulation of the cellular structures in the cortex."

A great many experiments have been performed demonstrating both localised and general cortical inhibition (sleep); even the transition from local inhibition to general inhibition (and vice versa) has been produced experimentally. Pavlov's conclusions as to the brain processes involved in these experiments are these ¹⁰: "Internal (localised) inhibition during the alert state is nothing but a scattered sleep, sleep of separate groups of cellular structures; and sleep itself is nothing but internal inhibition which is widely irradiated, extending over the whole mass of the hemispheres and involving the lower centres of the brain as well."

Pavlov states ¹¹: "The complete and continuous proof of the contention is spread over the whole of our twenty-five years work upon the hemispheres, and at the present time no part of the physiology of the hemispheres studied by the method of conditioned reflex is better substantiated."

The spread of this general inhibition of the cortex commences from a point of initial stimulation. "A prolonged stimulation of one and the same point in the cortex leads to a great and profound inhibition, and this irradiates widely so as to involve the whole of the cortex and the lower parts of the brain," causing sleep.

What is the chief characteristic of sleep? It is surely a more or less complete loss of consciousness, designated as *unconsciousness*. The question naturally arises: If a general irradiation of cortical inhibition is the neurological basis of sleep, the chief feature of which is unconsciousness, is it also the neurological basis of unconsciousness in other conditions? Let us take first the symptoms of epilepsy, a condition in which unconsciousness is a common feature.

EPILEPSY.

The muscular movements of epileptic fits vary in character, but it is usually agreed that the neural site of any epileptic fit is in the cerebral cortex*. In the localised movements of Jacksonian epilepsy the involvement of parts of the motor area of the cortex is often unmistakable. As regards major fits, Kinnier Wilson writes¹²: —“It is probably thought generally, or is at least a tacit assumption, that ordinary major fits are the outcome of discharges of cortical nerve-cells throughout the middle physiological level of both hemispheres (the rolandic motor cortex)—as far, that is to say, as visible movements of trunk and limbs are concerned.”

The development of localised muscular movement into the powerful convulsive movement of a general fit is due to the irradiation of the ‘discharge’ of motor nerve-cells over previously unaffected areas of the cortex. The neurological process which apparently takes place is described by Kinnier Wilson¹⁵ as follows:—

“The important conclusion is reached that very little of a Jacksonian fit is directly due to the discharge of abnormally nourished neural units, and that by far the greater part of it is produced by consecutive discharges of normal stable cells. If this be granted, the radiation of an epileptiform fit is entirely a physiological phenomenon, and offers proof of the fact (for which there is abundant evidence otherwise) that healthy neural mechanisms may become epileptogenous, without being in the remotest degree diseased.

“In the case of the general or major epileptic fit the circumstances cannot be materially different. While on occasion a local commencement (turning of head and eyes to one side) is indicative of the cell-groups first implicated in the liberation of energy, in many instances universalization of the convulsion is almost immediate, and the fit is said to be severe. The supposition here is that the initial discharge is particularly sudden and powerful, and that many series of bilaterally situated cell-groups have become unstable and, as it were, explosive. With a great quantity of this explosive material radiation through various neural levels evidently occurs with extreme rapidity . . .”

The chief significance of this statement is the view that radiation of the motor discharge spreads over normal nerve-cells of the cortex. The intimate nature of this discharge is unknown, but we are justified in classifying it as excitation—an abnormal, exaggerated excitation, but nevertheless essentially akin to cortical excitation causing the co-ordinated movements of normal muscular contraction. We might well term the abnormal cortical excitation of epilepsy, *explosive excitation*.

This viewpoint is closely related to that of Kinnier Wilson, who says¹⁴: “Following the profound discussion of this matter by Hughlings Jackson in his Lumsian lectures of 1890, I believe that the essence of a fit with convulsive movement consists in the exaggeration of a normal physiological process—

* The arguments of those who refer to subcortical centres for the production of the movements of epileptic fits are replied to by Kinnier Wilson in *Modern Problems in Neurology*.

that is to say, in sudden, excessive, and yet purely temporary liberation of kinetic energy in a series of motor nerve-cells, the visible consequence of which is a sudden and excessive development of many movements at once."

We can now compare this process of irradiation of cortical excitation in epilepsy with the irradiation of excitation demonstrated in Pavlov's experiments. The two types of excitation appear to be essentially similar, the former differing in being 'explosive' in character, with (in a general fit) a more rapid and widespread irradiation.

According to Pavlov's results, we should expect to find also in epilepsy the reverse process of inhibition in the cerebral cortex, either local or general, or both. Other symptoms of epilepsy do strongly support the view that they are manifestations of cortical inhibition. Remembering Pavlov's discovery that excitation of a region of the cortex induces inhibition of the same region (mutual negative induction) the interpretation of further symptoms of epileptic fits is as follows.

Inhibition always follows excitation, in physiological processes of the cortex. After an intense, exaggerated excitation of the motor cortex such as occurs in generalized epileptic convulsions, we should expect a correspondingly intense inhibition of the same region. The symptoms point to this, for after such a general fit, the patient lies in a state of coma, with complete muscular relaxation. The facts point to this muscular relaxation being the result of an inhibition, i.e., an inhibitory action—exaggerated and intense, of the motor cortex, following inevitably on the previous exaggerated explosive excitation causing the convulsions. This inhibitory action of the motor cortex will be mentioned again later in connection with the symptoms of cataplexy.

Coming now to the symptom of *unconsciousness* in epilepsy, its development is described by Kinnier Wilson¹⁵ thus: "Some epileptic attacks are attended by loss of consciousness; others are not. Unconsciousness may supervene at the outset, or develop subsequently; it may be extremely brief in duration, or may continue for some time after all convulsions have ceased. Further, of itself it forms no gauge of the severity of the fit; a severe Jacksonian attack may from first to last involve no loss of the senses, while a petit mal seizure may consist of little else than a transient conscious 'blank,' an 'absence épileptique.'"

In the sleep of the subjects of Pavlov's experiments, the underlying neural process was found to be a generalized irradiation of cortical inhibition. It appears likely that the unconsciousness of epileptic fits is due also to a generalized irradiation of inhibition over the cortex. The general cortical inhibition of epilepsy may differ *quantitatively* from the general inhibition of sleep, for example in intensity and rapidity of irradiation, but there is no reason to suppose that the irradiating process (like the irradiation of the motor discharge) is not a physiological one. *Qualitatively*, the processes of cortical inhibition that appear to underlie unconsciousness in sleep and in epilepsy must surely be the same.

Unconsciousness in epilepsy, with its variability as a symptom, is explicable on the basis of irradiation and concentration of cortical excitation and inhibition, with their mutual and reciprocal induction.

If unconsciousness supervenes during the period of motor excitation—of convulsive movements, then there is 'induction' of a general irradiation of cortical inhibition by a local area of more or less intense excitation (in the motor cortex). We may recall Pavlov's words: "Negative induction . . . consists in this, that a stimulation of the cortex at one point leads to inhibition of the rest of the cortex."

If unconsciousness commences at the beginning of a fit, then the general irradiation of cortical inhibition from the initial motor area of stimulation is immediate and rapid. If, on the other hand, unconsciousness develops later during the convulsions, the general irradiation of inhibition (by 'negative induction') is, for some reason, delayed.

When the fit consists of little more than a transient loss of consciousness, the process of inhibition is particularly labile, and irradiates over the cortex from a small area of stimulation, which is no more intense and no wider than in a normal conditioned reflex. The actual cause of the exaggeration and distortion of the normal cortical processes of excitation and inhibition that apparently occurs cannot at present be surmised.

Suggestions have been made that the symptoms of epilepsy are due primarily to cerebral vasomotor changes, but while such changes have been shown to accompany epileptic symptoms, the improbability that they are the cause of the symptoms is pointed out by Kinnier Wilson in referring to objections to this explanation.

The return of consciousness after a seizure may be regarded as the result of a concentration of inhibition, subsequent to irradiation. The briefer the period of unconsciousness in a fit, the more rapid is the concentration of inhibition. In this connection, we may note that, in Pavlov's view, consciousness is the result of a nervous activity of a certain part of the cerebral hemispheres, that is, of a more or less concentrated activity. He says¹⁸: "I shall . . . endeavour to answer provisionally what kind of physiological phenomena, what sort of nervous processes, proceed in the hemispheres of the brain when we say we are 'conscious' and speak of our 'conscious' activity. From this point of view, consciousness appears as a nervous activity of a certain part of the cerebral hemispheres, possessing at the given moment under the present conditions a certain optimal (probably moderate) excitability. At the same time all the remaining parts of the hemispheres are in a state of more or less diminished excitability."

We seem to be confronted with the likelihood that consciousness is the subjective effect of a (constantly shifting) area of cortical excitation, which is wiped out during unconsciousness by a general irradiation of inhibition over the whole cortex.

CATAPLEXY.

In order to clear up certain difficulties in the application of Pavlov's conclusions to the interpretation of epilepsy and narcolepsy, reference must be made to cataplexy, a condition associated with narcolepsy. In cataplexy, an individual may sink to the ground with complete muscular relaxation, unable to move, but retaining consciousness. Here, if we are not mistaken, there must be an inhibitory action (inhibition) of the motor cortex, abolishing the postural tone of the muscles (as in the state of muscular relaxation following a general epileptic fit) by obliterating the tonic contractile action of the lower motor centres.

Quite a different effect is produced by destructive lesions of the motor cortex, in which release of the lower motor centres causes a tonic muscular contraction. Evidently this inhibition of the motor cortex in cataplexy is not a complete loss of cortical control. It is rather an exaggeration and distortion of a certain phase of normal cortical control. This normal action is the relaxation of muscles which takes place in the ordinary movements of an animal. We know that we can 'voluntarily' relax a set of muscles. We know also that through reciprocal innervation one set of muscles is relaxed while the opposing set contracts. Apart from the fact that what we know as 'voluntary' movement is connected with cortical control, there is experimental evidence to show that reciprocal innervation is obtainable from stimulation¹⁷ of "any particular point on the motor cortex proper," as well as of lower nervous centres. Hence, whatever the exact mechanism in reciprocal innervation (concerning the relation between cortex and lower nervous centres), it is probable, to say the least of it, that cortical inhibitory action plays a part in obliterating (partially and very temporarily) the tonic contractile action of lower motor centres on antagonistic muscles in the movements of a normal animal.

Cortical inhibitory action causing muscular relaxation is also seen in the experiments of Graham Brown, who showed that cortical activity could wipe out the tonic contractile action of the lower motor centres, and concluded¹⁸ that "non-postural cortical activity seems to abolish postural midbrain activity."

The term 'inhibition' applied to this non-postural activity of the motor cortex by no means signifies that the control of the motor cortex over the muscles is in abeyance. On the contrary, the control is manifestly present, although the result (muscular relaxation) is of a negative character. Inhibition of the motor cortex (in whole or in part) means that there is a certain process, the reverse of excitation, in the motor cortex, causing muscular relaxation. In cataplexy, this process of inhibition must descend into the lower motor centres, obliterating their tonic contractile action. This inhibition of the motor cortex might therefore be called 'descending inhibition'; or perhaps a complete muscular relaxation might be referred to as 'full inhibition' of the motor centres of the (intact) central nervous system.

In cataplexy, then, physiological inhibition in the cortex produces a definite pathological result because :—

- (a) In the cortex it is confined to and includes the whole of the motor area ;
- (b) It descends to the lower motor centres, obliterating their tonic contractile action ;
- (c) It persists for a considerable period of time.

Instances of the production of a pathological result (e.g. cataplexy) through the agency of a physiological process (e.g. inhibition) are by no means confined to the nervous system. In the alimentary system, for example, a pathological result—intussusception, is produced through a physiological process—peristalsis. Certainly the primary cause of the pathological result is uncertain, in intussusception as well as in cataplexy. However, a definite advance is made if the process immediately underlying a symptom is revealed. In cataplexy this process appears to be a physiological inhibition of the cortex, selecting specifically the whole motor area, and involving the lower motor centres.

NARCOLEPSY.

There are several clinical varieties of narcolepsy, the chief feature of which is sleep, usually in recurring diurnal attacks. In these attacks of 'natural' sleep the individual becomes unconscious. However, in some cases the attacks of 'sleep' are in reality trance-like states, in which the subject is conscious, but unable to move or speak. These cases with recurring trance-like states link the 'natural' sleep of narcolepsy to other cases where the attack is one of cataplexy, in which there is more or less complete loss of muscle tone ; the individual may sink to the ground, immobile and speechless, but remaining fully conscious all the time. The great majority of these cataplectic attacks are associated with attacks of sleep. Their initiation is a matter of great interest, for it is usually an external stimulus evoking an expression of emotion which produces the cataplectic state.

The attack of cataplexy (loss of muscle tone and power, with full conservation of consciousness), is described by Kinnier Wilson, who writes¹⁹ : "The exciting cause of the attacks is declared almost universally to reside in a stimulus of the emotional series, among which anger, annoyance, compassion, anxiety or agitation, amusement or joy may be specially mentioned. The *act* of laughing perhaps provokes the attack more readily than any other cause, but in this case the emotional stimulus by itself is insufficient ; anger, and excitement generally, will precipitate the attack, but a stimulus arousing laughter will not produce it should the patient be able to inhibit the *motor act* of laughing."

Not only cataplectic attacks, but also certain cases of narcoleptic (unconscious) sleep may be evoked by emotional stimuli²⁰ : "Emotional excitement may lead not to the cataplectic but to the narcoleptic state."

Also²¹: "Sleep may be associated with or follow the cataplectic state," i.e., during or immediately after an attack of powerlessness, the individual concerned may feel an irresistible desire to sleep. Furthermore, in some cases, if the person forcibly keeps himself awake, an attack of powerlessness will supervene instead of the narcoleptic sleep."²²

It is evident that in many cases of narcolepsy (with or without cataplectic states) a stimulus evoking an expression of emotion initiates the attack. We are concerned chiefly with those cases where unconsciousness is also a feature of the attack. *It is suggested here that the neurological basis of unconsciousness in narcolepsy is essentially the same as that of unconsciousness in epilepsy, which, as we have seen, appears to correspond with the general cortical inhibition of Pavlov's experiments.* If this is actually the case, we should expect to find that the general irradiation of cortical inhibition producing unconsciousness in narcolepsy proceeds from a central cortical point of initial stimulation, as it does in the sleep of Pavlov's experiments. Can we locate such a point?

In those attacks of narcoleptic sleep apparently unaccompanied by any emotional excitement, no focal point of cortical stimulation can be determined. But in those cases where the narcoleptic sleep is initiated by a stimulus evoking an expression of emotion (e.g. laughter), the location of such a point is not altogether impossible, judging by the facts available. The question then arises: Can we find a point in the cerebral cortex which is concerned specifically with the transmission of efferent impulses causing expression of emotion?

It is suggested by Kinnier Wilson that certain anatomically defined corticifugal tracts supplying the facial and respiratory muscles, distinct from the 'voluntary' innervation of these muscles conveyed by the pyramidal tracts, are responsible for the 'involuntary' expression of emotion. He describes²³ these tracts as follows:—

"One is an arresting and the other an accelerating path. The former arises from the under surface of the frontal lobe, the latter from the sensory cortex . . . they come together towards the middle line at the mesial aspect of the lower optic thalamus, bordering on the third ventricle, and run down, near the midline of the tegmentum, to the medulla. Both are far removed from the 'voluntary tract' for respiratory innervation in the capsule and crus." Kinnier Wilson states²⁴: "I believe it is a feasible speculation that these are the paths for emotional activation of the faciorespiratory mechanism"; and he concludes²⁵: "There are corticifugal paths to faciorespiratory centres in pons and medulla that are independent of voluntary cortico-ponto-bulbar tracts to the same nuclei; on excitation they will either arrest or accelerate, i.e. interfere with, normal rhythmic activity of the respiratory centre; the available evidence warrants the speculation that they are the routes taken by emotional impulses to modify the faciorespiratory synkinesis in the direction either of laughter or the reverse." The dominant role of the facial and respiratory muscles in the expression of emotion is obvious. The additional innervation of these muscles through the corticifugal tracts

described above has been demonstrated. If these tracts subserve expression of emotion, we are left with two cortical areas concerned specifically with expression of emotion, one in the sensory cortex, and the other in the frontal lobe. It is inconceivable that these two areas should not be functionally connected as a basis for co-ordination of the faciorespiratory muscles in the expression of emotion. Where then is the meeting point of these two areas, of which Kinnier Wilson writes²⁶: "We presume the existence of a cortical nodal point co-ordinating them. Its situation is at present indeterminate, yet it is likely to have some definite position"?

Leaving aside for the moment the question of the location in the cortex of this nodal point, an interpretation of narcolepsy based on the theory of conditioned reflexes will be somewhat as follows: An agent of the external world—a situation, the behaviour of another person, a spoken or written word—affects the sense organs of an individual in such a way as to cause a stimulation of the cerebral cortex, particularly of the nodal point subserving expression of emotion. The individual therefore bursts into laughter, or shows signs of other emotional excitement. For some unknown reason the stimulation of the cortical nodal point results, through reciprocal induction, in a general process of inhibition irradiating from the nodal point of stimulation over the whole cortex. Consequently the individual becomes unconscious and is said to 'fall asleep.' In cases of narcoleptic sleep where there is no emotional excitement, the initial point of stimulation may be located elsewhere in the cortex. With the subsequent concentration of cortical inhibition, consciousness returns to the individual, who 'awakes.'

This interpretation, however, while not inconsistent with certain types of narcolepsy, does not fit the facts of other cases, in which cataplectic attacks occur. How are we to explain loss of muscle tone and power following expression of emotion? A consistent explanation on the above lines is impossible unless the nodal point for expression of emotion is in the motor area of the cortex. Events in a cataplectic attack would then be something like this: An emotional stimulus causes an excitation of the nodal point in the motor cortex. Again, a spread of cortical inhibition from the nodal point occurs through induction, but does not extend beyond the motor area. Moreover, as previously indicated, the process of inhibition descends to the lower motor centres, obliterating their tonic contractile action. Therefore the individual concerned, all his motor centres now in a state of inhibition (inhibitory action), sinks motionless and speechless to the ground. Because the process of inhibition has not spread over the whole cortex, he does not lose consciousness. With the subsequent concentration of inhibition, the individual recovers the use of his muscles. The attack of cataplexy is then over.

This interpretation is consistent with the transitional cases between narcoleptic and cataplectic attacks. When sleep supervenes on a cataplectic attack, the process of cortical inhibition is first confined to the motor area, and then irradiates further over the whole cortex. When a cataplectic attack

ensues through a person preventing himself succumbing to a sleep attack, a rapid irradiation of inhibition over the whole cortex is checked half way, as it were, and reduced to the motor area only, where it descends to the lower motor centres, with a resultant cataplectic attack, instead of sleep.

In a normal person, not subject to these attacks, an emotional stimulus exciting the cortical nodal point produces an expression of emotion, but the process of widespread inhibition through induction does not occur. Instead, stimulation of the nodal point soon ceases, and the expression of emotion disappears.

Naturally, a great deal remains unexplained, particularly the intra-cellular or intercellular fault responsible for the widespread irradiation of inhibition in a case of narcolepsy. Again, the connection between cortex and lower nervous centres in the expression of emotion is not clear. The work of Cannon and Bard²⁷ shows that in dogs and cats, at any rate, expression of emotion is possible ('sham rage') in the absence of the cerebral hemispheres. Perhaps the cortical mechanism for expression of emotion outlined by Kinnier Wilson is the basis for that expression as a conditioned reflex, while the subcortical (thalamic) mechanism is for expression of emotion as an unconditioned reflex, on which the cortical reflex is built up, as it were. Another question still unanswered is: What determines the variation in the kind of emotion bringing on attacks of narcolepsy? And why do the subjective feelings in some cases correspond not at all to the expression of emotion?

A brief mention must be made of a difficulty raised by Kinnier Wilson to the explanation of narcolepsy on the basis of Pavlov's work. Taking the case of a narcoleptic keeping on his feet, and even walking, while unconscious ('asleep'), he says²⁸: "To hold that in such cases internal inhibition is confined to the cortex, subcortical centres remaining in activity, is illegitimate, for this physiological condition results, according to Pavlov, in catalepsy, which is signalised by absence of movement."

This difficulty may perhaps be removed by taking into account the peculiar conditions of Pavlov's experiments. The environment of his animals is the artificially static one of a specially built laboratory, in which all extraneous stimuli are excluded. Furthermore, the animal is kept more or less motionless by being strapped in an apparatus. Under these conditions, it is not surprising that the sleep produced by a monotonous stimulus is unaccompanied by movement, thus giving a condition of catalepsy. The cerebral cortex is in a state of inhibition, and the stimuli for subcortical movement are not present. The stimuli for movement and walking through subcortical reflexes (manifold environmental changes, recurrent pressure on the paws, with corresponding proprioceptive stimuli) are excluded by the conditions of experimentation. Consequently, instead of the subcortical centres serving the reflexes of walking, only the reflexes of motionless posture are maintained through them, while the general cortical inhibition results in sleep. This experimental catalepsy, then, is the result of special and unusual circumstances.

In the movements of a certain type of narcoleptic attack, there is also a general cortical inhibition, as in the catalepsy of Pavlov's experiments, but there are also the appropriate stimuli for the subcortical reflexes of walking. Hence the features, not of catalepsy, but of narcolepsy of a certain type appear (unconsciousness, with movements, e.g. of walking). Interpreting another type of narcoleptic attack resembling the motionless condition of catalepsy, it would be supposed that there is a general cortical inhibition, again without descent to the lower motor centres, and without the adequate stimuli for reflexes of movement subserved by those lower motor centres.

In all attacks of narcolepsy with stimulation of the cortical nodal point for expression of emotion, that point itself enters almost immediately into a state of inhibition, as well as the surrounding cortex. Therefore expression of emotion does not continue into the period of unconsciousness or powerlessness. But in epileptic attacks the motor area may remain in a state of excitation while, through reciprocal induction, the rest of the cortex is in a state of inhibition. Therefore in epilepsy convulsive movements may continue into the period of unconsciousness.

The variation in the physical signs of different conditions in which unconsciousness is a feature is due to particular changes in various bodily functions. In any condition where there is unconsciousness, as judged by a person's failure to respond to certain stimuli, and perhaps by his subsequent verbal account of his own subjective experiences, it is feasible to assume that in one way or another the cerebral cortex has been caused to enter into a state of general inhibition, whatever the other physical signs present. This would apply to normal conditions, e.g. the sleep of normal individuals, as well as to abnormal conditions such as epilepsy and narcolepsy, in which unconsciousness occurs. This point of view obviates the necessity for debate on whether the 'sleep' of narcolepsy is a true sleep or not. We need only know that certain physical signs in an individual constitute an attack of narcolepsy, and that in such an attack the individual may be unconscious.

SUMMARY.

Summarising, we may say that experimental and clinical evidence supports the view that unconsciousness is the result of a process of physiological inhibition extending over the whole of the cerebral cortex. This irradiation of cortical inhibition spreads from a focal point of initial stimulation. In epilepsy the focal point of stimulation is in the motor area of the cortex—the area of stimulation may include the whole motor cortex, with, in cases of unconsciousness, an irradiation of inhibition over the rest of the cortex through reciprocal induction. In many cases of narcolepsy the point of initial stimulation is also in the motor cortex, but is distinguished by being the co-ordinating point for efferent impulses to the faciorespiratory muscles, via a separate path, causing 'involuntary' expression of emotion,

We may conclude this tentative outline of an aspect of cortical function by quoting once again²⁹ from the authority on whose work it is largely based: "Inhibition of tone, inhibition of innervation—local or general, cortical or infracortical, or both—here surely is the clue to the diverse phenomena of narcolepsy, cataplexy, and probably also of catalepsy, so intimately interrelated in the cases that have occupied our attention."

REFERENCES.

- ¹ PAVLOV, *Lectures on Conditioned Reflexes*, 1928, p. 208.
- ² Idem, p. 211.
- ³ Idem, p. 172.
- ⁴ Idem, pp. 157, 158.
- ⁵ Idem, p. 322.
- ⁶ Idem, pp. 323, 347 (experiments of K. N. Krzhishkovsky, D. S. Fursikov, V. V. Stroganov, E. M. Kreps, M. P. Kalmikov).
- ⁷ Idem, p. 333.
- ⁸ Idem, p. 382.
- ⁹ PAVLOV, *Conditioned Reflexes*, 1927, p. 251.
- ¹⁰ Idem, p. 253.
- ¹¹ Idem, p. 250.
- ¹² WILSON, S. A. KINNIER, *Modern Problems in Neurology*, 1928, p. 6.
- ¹³ Idem, pp. 5, 6.
- ¹⁴ Idem, p. 4.
- ¹⁵ Idem, pp. 14, 15.
- ¹⁶ *Lectures on Conditioned Reflexes*, p. 221.
- ¹⁷ *Modern Problems in Neurology*, p. 233.
- ¹⁸ BROWN, GRAHAM, *Proc. Roy. Soc.*, 1913, B. lxxxvii, 145—quoted by KINNIER WILSON, *Modern Problems in Neurology*, p. 129.
- ¹⁹ *Modern Problems in Neurology*, pp. 97, 98.
- ²⁰ Idem, p. 98.
- ²¹ Idem, p. 99.
- ²² Idem, pp. 81, 99.
- ²³ Idem, p. 287.
- ²⁴ Idem, p. 288.
- ²⁵ Idem, p. 290.
- ²⁶ Idem, p. 294.
- ²⁷ CANNON, W.B., *Amer. Jour. of Psychol.*, 1927, xxxix, 106; *New England Jour. of Med.* 1928, cxviii, 877.
- ²⁸ BARD, P., *Amer. Jour. Physiol.*, 1928, lxxxiv, p. 490.
- ²⁹ *Modern Problems in Neurology*, p. 112.
- ³⁰ Idem, p. 115.