STUDIES IN CERTAIN PATHOPHYSIOLOGICAL AND PSYCHOLOGICAL PHENOMENA IN CONVULSION THERAPY.

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(Received May 23, 1940.)

DURING recent years so much has been written about convulsive therapy that it would seem rash to burden still further the very extensive literature on this subject. Nevertheless, in spite of the admirable work that has been done, it cannot be said that the implications of convulsive treatment are fully understood, nor that the fullest possible use has been made of this treatment for research into the pathophysiology of epilepsy. Furthermore, the recent introduction by Cerletti and Bini (1) of a method of treatment with electrically produced epileptic fits has increased still further the scope of research. I have had at my disposal case material provided by the treatment of various forms of mental disorder with cardiazol for more than two years, and with the electrical method in collaboration with Mr. Grey Walter for about six months. This has enabled me to conduct certain investigations of a sort that have received little or no attention hitherto, and to make observations covering various aspects of the physiology and psychopathology of induced epileptic states.

Observations on Motor Phenomena.

The attention of the observer is first drawn towards the exhibition of powerful motor activity which succeeds the injection of an adequate dose of the convulsant. Much of this activity conforms with that of the typical fit of spontaneous epilepsy, but there are, in addition, other types of movement which occurs only in certain individuals and have therefore not attracted general attention. These motor phenomena are seen either immediately before the clonic stage of the convulsion or during the period of unconsciousness which follows the clonic stage or, less frequently, during an incomplete fit. The phenomena to be described made their appearance fairly consistently in certain individuals early in treatment, but tended to disappear after a number of fits. Some of these movements have been observed and commented on by

https://doi.org/10.1192/bjp.86.364.799 Published online by Cambridge University Press

others in both insulin and cardiazol treatment. I have observed types of movements which can be classified as follows :

1. *Purposeful movements.*—These occur most frequently. Usually they are grasping, groping or reaching movements of the hands and arms. The purposeful character of these movements is well demonstrated by actions such as rubbing the eyes or repeatedly grasping a part of the body. Occasionally these movements have an athetotic character.

2. *Rolling movements.*—Some patients habitually roll over, sometimes completing the whole circle several times. The rolling movements frequently terminate by the assumption of a foetal attitude in which the patient sleeps. The direction in which the patient rolls is always the same, and it is notable that it is usually towards the side to which the patient turns when sleeping after the fit and in normal sleep.

3. *Hemiballistic movements.*—Extensive and violent rotation of one or the other arm, similar to hemiballism, is occasionally seen.

4. *Opisthotonic movements.*—These are of a powerful character. They are relatively common, and not infrequently occur in association with other types of movements.

5. *Automatic movements of the lower limbs.*—(*a*) Thrashing : Beating movements of the extended lower limb, usually accelerating and increasing rapidly in amplitude. (*b*) Bicycling : Rhythmical alternating movements of the lower limbs, similar to the action of the cyclist in pedalling. This action starts slowly, and works up to a maximum speed of about six cycles per second. In certain subjects bicycling was succeeded by blind running with great violence, strongly reminiscent of epilepsia procursiva.

6. *Cataleptic postures.*—These are chiefly observed in the incomplete fit. They are often of several minutes' duration and are of the nature of flexibilitas cerea. They are more marked in the upper than in the lower limbs.

Other types of movements may be observed, but it would seem that the above classification covers in a general sense most individual categories. Tonic movements of the eyes are probably of a different nature, and as they have been described by others they will not be referred to here.

Most of the above movements represent types of abnormal motor activity seen in lesions of lower motor centres. Involuntary movements of a purposeful character have been seen in disorders of the striate body and the frontal lobes. Hemiballism has been found to follow lesions of the body of Luys. Slow bicycling movements have been observed in cases where there was softening of the anterior part of the globus pallidus. Thrashing and bicycling, as well as opisthotonos, resemble movements seen in lesions of the mesencephalic motor nuclei, observed clinically in tumours of the midbrain and produced experimentally by stimulation of these regions (Graham Brown, 2). Fraser and Reitmann (3) have shown recently that bicycling accompanies the return of motor activity during the recovery from experimentally produced anoxaemia. A similar complex of all the movements described before has been observed by various workers (Wagner-Jauregg, 4; Gerstmann, 5; Strauss, 6; Gamper and Kral, 7) in individuals during resuscitation from severe transitory anoxia of the brain produced by attempts at hanging, or avalanche disasters. Localized lesions in the basal ganglia were demonstrated in cases which had succumbed. This may suggest that these movements are due to the return of activity of

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deeper centres as the effects of anoxia are subsiding. The fact, however, that such movements occur both in the incomplete reaction and before the development of the clonic stage, when anoxia could not have been severe, suggests that in certain individuals cardiazol can affect certain deeper parts of the brain to an unusual degree at a time when its effect upon the cortex is not fully developed.

These movements, which are not the usual accompaniments of the spontaneous epileptic fit, appear to occur less frequently in the electrically induced fit, and obviously only in the post-convulsive stage. This would suggest that in the electrically induced fit certain parts of the brain which are involved in the cardiazol fit are not usually affected, and when atypical movements occur they are probably due to anoxia. This would conform with the experience that the electro-fit is considerably shorter than the cardiazol fit, being very constantly about 45 seconds (Fleming, Golla and Grey Walter, 8).

Relationship between the Motor Reactions and Individual Features of the Patient.

Observation of a long series of induced convulsions creates the impression that individual patients seem to favour a particular type of reaction to convulsant measures, in both the complete and incomplete fits. Naturally, individualities are most evident where the reaction is particularly mild or contains uncommon features.

Even with the so-called normal reaction one notices small peculiarities, such as the sequence in which the march of the fit occurs, the type of the cough, or the way in which the eyes are screwed up or the mouth opened. Furthermore, it would seem as if the amount of discharge during the convulsive stage of the fit varies in different individuals.

The motor reactions appear to be either of or between two extremes, namely, fits of a mild nature unaccompanied by great motor activity, and fits associated with a storm of movements. The " mild " fit is nevertheless a typical fit, but is mild by comparsion with those fits where the motor discharge is extreme.

With regard to the mild type of reaction, one gets the impression that those individuals whose reactions are mild with a comparative paucity of motor activity are, in their behaviour during their illness, simple and devoid of aggressive action. I have not observed any case whose reaction to cardiazol was mild in whom, during the period of detention in hospital, strong spontaneous psychotic motor activity or a high degree of negativism could be seen. The conduct of these cases, in spite of a variety of delusions, hallucinations, states of fear or apprehension or apathy, did not take the form of active and violent opposition to the environment. It was not uncommon to learn that such patients had been particularly good tempered or docile before the development of the illness.

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With this type of personality, muscular activity before and after the fit is slight, and no marked muscular energy nor extraordinary movements accompany the incomplete fits. Such patients usually tend to sleep on quietly after the convulsive stage without exhibiting restless behaviour. At the other extreme are those patients who react with a great expenditure of muscular energy, showing not only an extreme motor discharge during the fit, but also various movements before and after the convulsive stage, and whose incomplete reactions are rich in motor phenomena. In the psychosis of such patients negativism and motor activity are prominent, expressed in violent action against property and staff or against themselves, and in resistance to treatment. Their histories do not show that they had been noticeably submissive and gentle prior to the mental illness, and sometimes the reverse seems to be the case. Between these extremes are patients whose reactions to injections and whose psychotic behaviour cannot be assigned to one type or the other.

These observations show that there is a relationship between certain forms of involuntary motility and features of the personality. If it is true that the atypical movements described are related to activity of deeper parts of the brain, and that such movements appear chiefly associated with certain personality features, then it could be inferred that there is an intimate relationship between personality features and the activity of deeper parts of the brain.

A Postconvulsive Reflex Phenomenon.

In the majority of individuals treated there is a short period of reflex excitability during the postconvulsive stage. This stage of excitability appears shortly after breathing has terminated the postconvulsive apnoea, and lasts for upwards of a minute. During this period, a brisk stimulus applied to the skin of the trunk produces a brisk flexion contraction of all four limbs with anteroflexion of the neck, accompanied by some protrusion of the eyeballs and some dilatation of the pupils. This phenomenon seems to be related to respiration, as it can be elicited only immediately after the end of inspiration, no reaction being obtained to stimuli applied at any other moment in the respiratory cycle. The susceptible phase in some patients seemed to be not more than a few seconds ; it is therefore likely that in the few patients who did not show this reaction the stimulus had been applied when the susceptible phase was over. This reaction, which can be demonstrated in many cases during cardiazol therapy, was seen only once in 12 cases treated with electrically induced convulsions.

The phenomenon may be regarded as resulting from the state of hyperactivity of the motor cortex, which persists for some time after the convulsive stage and which gives rise to motor discharges in response to external stimuli. This reflex hyperactivity has some resemblance to what is seen in strychnine poisoning, where external stimuli provoke motor discharges. The relationship

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of this reaction to the passing off of anoxia, and to the restoration of more normal circulatory conditions, indicates that it marks the return of activity in parts of the brain which have been exhausted or suppressed in the course of the epileptic discharge.

Reflex Movements of the Eyes.

The occurrence and features of spontaneous ocular movements during the cardiazol fit suggested that it was desirable to investigate in some way the reflex behaviour of the eyes. The eyes did not respond reflexly to alteration of the position of the head during the fit and reflex compensatory eye movements could not be elicited. It was of interest to investigate the response of the eyes to the stimulus, which under normal conditions invariably produces a certain sequence of eye movements, that is, irritation of the labyrinth, leading to physiological nystagmus. As far as I am aware, investigations of this sort have not been conducted during epileptic fits in human subjects.

The method employed was that of syringing the ear with cold and hot water as in the usual tests for labyrinthine function. Only patients free from any disorder of the auditory apparatus or labyrinth were investigated. Syringing was performed with the patient lying on the back—

(*a*) Under normal conditions on the day before cardiazol injection.
(*b*) In the post-convulsive stage immediately apnoea had ceased.
(*c*) During the convulsive period.
(*d*) During an incomplete fit.

The results were striking, both because of their form and of the absolute consistency with which they appeared. They were as follows:

(*a*) *Under normal conditions.*—All patients examined showed normal physiological responses.

(*b*) *In the post-convulsive stage.*—One or two seconds after syringing with cold water, the eyeballs turned towards the side of the irritation, reaching an extreme deviation. This deviation could not be altered by turning the head. Between 10 and 30 seconds later the eyes left this position and moved slowly towards the middle line. Sometimes the movement of the two eyes was not synchronous, and the eye of the irritated side lagged behind the other. The eyeballs did not remain in the middle position, but showed a slow rhythmical movement to right and left of 2–4 mm. amplitude. These movements were of the type of pendular nystagmus. This pendular nystagmus was followed by some jerky movements of the type of the normal labyrinthine nystagmus. On several occasions deviation of the eyes was accompanied by active turning of the head towards the side of the deviation, but the head could be moved in the opposite direction without much resistance. Just before the end of the period of deviation, and also when nystagmus had commenced, it was sometimes possible to produce compensatory deviation of the eyeballs by turning the head forcibly. For example, turning of the head from the middle position to the left, when the eyes were already deviated to the left, caused the eyes to leave their position of deviation relative to the head and to remain in their position in space, thus preserving their original angle of deviation from the body axis.

(*c*) *During the period of convulsion.*—Syringing with cold water was performed in the tonic and clonic stages. The results were largely the same. The eyes turned, as described before, to the side of the irritated labyrinth. Compared with the results of syringing in the post-convulsive state, there were the following differences:

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(i) There was a latent period, sometimes 5–10 seconds, before the results of syringing appeared. This period was especially prolonged when a tendency to spontaneous deviation in the opposite direction existed already, so that although deviation towards the side of irritation always occurred, it obviously took some time to overcome the impulses drawing the eyes in the opposite direction.

(ii) The appearance of muscular tension of the eyes and the very extreme degree of deviation gave the impression that the eyes were being turned with far greater muscular force than in the post-convulsive state.

(iii) Occasionally the eyes moved from a position of lateral deviation to that of extreme deviation obliquely upwards. Tonic deviation remained until the end of the clonic stage; return to the middle position occurred as described above, with occasional squinting, or a phase of pendular movements, to be succeeded by a few jerks of physiological nystagmus. No patient complained of dizziness or nausea when questioned immediately after the return of consciousness.

It was felt that syringing of the ears might not be without effect on the general condition of the patient, and observation seemed to suggest that that was the case. Reflex activity in all patients seemed to be somewhat increased and to persist for a longer period than usual when the ear was syringed in the post-convulsive stage. Stimulation of the skin and front of the abdomen provoked a more brisk and more frequent reaction than it had done hitherto without syringing. This effect was not evident when syringing was done during the fit. When syringing was done during the fit the return to normal respiration seemed to be much smoother, and cyanosis and pallor seemed to disappear more quickly than usual, and it seemed as if the motor discharge of the epileptic fit was more complete.

(*d*) *During the incomplete fit.*—Syringing was performed during incomplete fits, when the patient was in a state of semi-consciousness and was not accessible nor co-operative. The threshold obviously had to be nearly reached. The result was essentially the same as in the post-convulsive stage.

Syringing with hot water produced reactions similar to syringing with cold, except that deviation of the eyes occurred away from the side of the irritation.

Summary of the Findings in the Labyrinthine Investigations.

The notable results of these investigations are as follows:

Syringing of the ear, both during and immediately after convulsion, fails to cause the normal physiological nystagmus.

Syringing with cold water consistently causes an extreme deviation towards —with hot water away from—the side of labyrinthine irritation, in each case followed by a period of pendular nystagmus, followed in its turn by a short period of nystagmus of expected physiological type.

Reflex irritability in the post-convulsive period seems to be increased by syringing in this stage.

Labyrinthine stimulation during the fit seems to facilitate the return of normal respiration and circulation.

It is not intended to enter into a full discussion of these findings. It is only possible to consider a few of the factors involved. As the direction of deviation corresponds to the side of the slow component in the normal physiological nystagmus, it is possible that this deviation is the result of the suppression of the quick component during the epileptic state. The phenomenon would therefore be an example of an inhibition of a certain action, namely, of the

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quick component of the normal nystagmus, which component is generally considered to be related to the cortex. It has been shown by Wilson (9) that inhibition as well as discharge of activity takes place during the epileptic state. However, it is not likely that such an extreme and powerful deviation of the eyes takes place without some positive influence on the part of the cortex irrespective of the inhibition of the cortically controlled quick component. The experiments of Spiegel (10) have shown how labyrinthine stimuli under certain conditions can influence the cortical activity of animals, thereby demonstrating the existence of connections between the cortex and the labyrinth. Further investigations on problems arising out of these findings are not yet completed.

It is of interest that labyrinthine stimulation was found to have a marked effect on the re-establishment of circulation and respiration. It can therefore be assumed that stimulation of the labyrinth affects in some way the vasomotor and respiratory centres. This has already been suggested to explain certain vasomotor and respiratory alterations which occur in various conditions, such as Menière's disease, where irritation of the labyrinth exists.

Conditions Influencing the Response to the Convulsant Drug.

Various observers have reported on factors which influence the threshold, i.e. the minimum dose of the drug necessary to produce a fit. Such factors are—the speed of injection, the concentration of the drug, the administration of sedative drugs or substances such as histamine with a vasodilating action. (Dennyson and Watterson, 11; Watterson and Macdonald, 12). My observations show that there are other factors which have an effect upon the threshold. Such factors are :

(*a*) *Follicular hormone* :—A number of female patients suffering from amenorrhoea were treated with 50,000 units of oestrone, injected five times in 21 days, while undergoing treatment with cardiazol. In almost every patient the threshold was considerably raised while oestrone was being absorbed, and in one patient it was impossible to induce a fit with doses of less than 50 per cent. more than what had been effective previously. At the conclusion of hormone treatment the threshold returned to its original lower level. Furthermore, it was observed in certain other patients that on the days immediately before the menstrual period there was a reduced susceptibility to cardiazol. Although this appears to be somewhat at variance with the common experience that there is an increased incidence of fits in epileptics at the menstrual period, it may be that in cardiazol epilepsy follicular hormone influences the susceptibility to the blood-borne drug by causing alterations in the cerebral circulation. Recent experimental work by M. Reiss and Y. M. L. Golla (13) has shown that the administration of sex hormone markedly increases the cerebral blood content in animals. My observations indicate that the same conditions may

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be true in the human subject and may be responsible for raising the threshold to cardiazol.

(*b*) *Behaviour of the patient at the time of the injection.*—Patients who were obviously terrified and struggled violently, showing physical signs of extreme fear, with a rapid pulse and pale face, failed to respond to the injection more often than those who were co-operative or apathetic, and if the degree of anxiety was great, anything but a very large dose was ineffective. At first sight one would expect to find that in states where cortical excitation was already present, and where the rate of circulation of the blood was increased by the rapid and pounding heart-beat and the extreme muscular activity, the threshold to a given dose of cardiazol would be lower rather than higher; there must, therefore, be other conditions related to states of anxiety and emotional tension, unfavourable to the production of a fit. In the literature it appears that Ribbeling alone refers to this problem. He mentions that it is necessary to give an increased dose of cardiazol in agitated states.

It is possible that the raised threshold to the cardiazol fit in states of anxiety is related to changes in the cerebral blood-flow. Furthermore, it may be imagined that physiological conditions similar to those described by Cannon (14) exist in the anxious and agitated human being, and if so may contribute to the inhibition of the fit. Perhaps the liberation of a substance with an action similar to histamine produces circulatory changes affecting the distribution and local concentration of cardiazol.

Changes in the Optic Fundi During the Induced Fit.

In genuine epilepsy there is seldom an opportunity for making a systematic examination of the fundus throughout the epileptic fit. Hughlings Jackson (15) records vasoconstriction, and Rossi (16) a paraxysmal angiospasm of the retinal vessels in the course of the epileptic fit.

I have investigated the optic fundi of a number of patients during cardiazol treatment. The patients were also examined from time to time on days when cardiazol was not administered. Homatropine was used as a mydriatic. At first no mydriatic was used, but it was found that, although not strictly necessary, its use greatly aided a full inspection of the fundi, especially at times when deviation or movement of the eyes occurred. As similar results were obtained in the same patient whether homatropine was used or not, it can be assumed that this drug has not influenced the findings.

The first essential was familiarity with the appearance of the fundi of each patient under normal conditions, for individual fundi and discs differ, some showing marked pallor. Therefore, what one can call the "complexion" of the fundi had to be taken into account before comparisons could be made.

It is hardly possible unless the patient co-operates fully to observe the fundus until unconsciousness has supervened, and at the onset of the tonic

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stage it may be impossible if there are extensive movements of the head or trunk or eyes. With the aid of an assistant the observations can usually be made during the tonic stage. These difficulties do not exist later, when the eyes remain open or even staring and, except for the inconvenience of deviation of the eyes, or the more rarely seen violent postconvulsive movements, the fundi can usually be kept under observation continuously until consciousness returns.

In all patients examined some changes were observed in the condition of the blood vessels and the appearance of the discs, which were as follows: During the tonic stage there is marked contraction of the arteries accompanied by a growing pallor of the whole fundus and disc. In some cases this blanching seems to cause the disappearance of small vessels. The veins also become less distinct. This picture remains until the end of the first half of the clonic stage. During the second half of this stage the vessels dilate, veins and arteries become more prominent, and the pallor subsides. In some cases, vessels which were not obvious before become visible. This state of hyperaemia deepens, and the disc loses something of the sharpness of its outline; in some cases one even gets the impression that swelling of the disc has occurred. By the end of the clonic stage the hyperaemia reaches its climax; the whole fundus and disc have then a distinctly reddish appearance, considerably more red than is seen in the fundi of the patient under normal conditions. The hyperaemia gradually subsides during the postmyoclonic stage, but even when breathing has become normal there is still a trace left. The fundi are usually normal when consciousness has returned, but occasionally a certain amount of hyperaemia and even a slight degree of swelling of the disc remain for a considerable time afterwards. In one patient the discs never regained the original sharpness of outline during the whole period of treatment and several subsequent weeks.

The literature on cardiazol therapy contains the following observations: Meduna (17), quoting Fesus, who investigated Meduna's patients, states that there is no constant change in the fundi during the cardiazol fit, except "a certain degree of increased vascularity of the connective tissue with slight dilatations of the vessels of the episclera." He (Meduna) considers that these observations are in contradiction to the behaviour of the cerebral vessels of animals during experimental convulsions. Kennedy (18) reports that there is no change in the retinal vessels except the widening of the veins during cyanosis. Watterson and Macdonald (12) observed retinal vasodilatation only during the cardiazol fit.

The behaviour of the retinal vessels that I have reported is of interest, as vasomotor changes in the brain have received so much attention in the discussion of the causation of epileptic convulsions. This behaviour is in keeping with the processes of vasoconstriction and vasodilatation on the brain surface that have been observed in experimental epilepsy. Fischer and Lowenbach (19) photographed the surface of the brain during a cardiazol fit. Their photographs

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showed that the visible arteries and veins became dilated during the fit, and tiny vessels were seen which had previously been invisible. Foerster (20), Dandy (21), Leriche (22) *et alia* have observed vasoconstriction when epileptic fits took place during an operation on the brain surface.

It has been suggested that the epileptic fit is caused by vasoconstriction of cerebral vessels. This theory has found and still finds support. Certain changes in the brain tissues in epileptics—for instance, the well-known lesions of the cornu ammonis—have been ascribed to vasomotor disturbances. Alf. Meyer (23) has recently demonstrated that similar changes in other parts of the cortex, the " dropping out " of nerve cells, are related to the distribution of blood vessels. These findings agree with Jackson's (15) idea that anæmia of the brain might be responsible for the epileptic fit. It is true that this theory has met with opposition from the beginning. It was denied by Gowers (24), and more recently Golla (25) has argued that, although the presence of vasomotor disturbances in the brain cannot be denied, there is no direct evidence that they are responsible for the epileptic fit. My own observations show that vasomotor changes take place in the fundi during the whole cardiazol fit, and that these changes occur in the same sequence and are of the same order as those observed in the cerebral circulation during epileptic fits. It is very likely that the respective changes take place at the same time in the fundi and the brain. But, as Wilson (26) has pointed out, whether the undeniable association of vascular changes with the paroxysm is one of cause or effect has still to be discovered.

Some Observations on the Psychopathology of Convulsive Therapy.

It is clear that an understanding of the mechanisms responsible for a change of symptoms in shock treatment cannot be gained from study of the physical events alone. It is unnecessary to refer in detail to the rich literature on this subject. I propose to record certain observations which are of special interest because they have attracted little attention up to the present, and to draw attention to certain psychological events with regard to which my experience differs somewhat from that of others.

Attitudes of Patients Towards Treatment.

The various attitudes which patients may show towards treatment may be classified as *co-operation, indifference, hostility, fear, pathological terror.* Of these co-operation is probably the most frequently observed and is certainly the most favourable from the point of view of prognosis. Indifference and hostility seem to be related closely to the fundamental features of schizophrenia, and where they are present treatment has little effect. Fear should not be considered as identical with what I propose to call pathological terror. Fear can be observed in many patients irrespective of the nature of their psychoses,

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and does not differ from the usual apprehension shown by so many individuals for physical interference or painful forms of treatment. Pathological terror is a most impressive and extreme degree of fear with widespread manifestations of terror. It is remarkable in that it does not necessarily appear after the first injection, and that in certain cases it disappears later in treatment. It is generally assumed that failure to produce the complete reaction on one occasion leads to pathological fear of subsequent injections; in my experience any such connection is fortuitous. The occurrence of states of terror is not derived from a fear of undergoing pain, but is obviously determined by something entirely different. Nightingale (27) found that states of anxiety occurred in four patients who never missed a fit, and that 10 of his 44 cases who frequently showed the incomplete reaction exhibited no fear.

Without discussing this phenomenon at length it may be stated that a careful examination of patients showed that the underlying factor was a conscious fear of death expressing itself by the fullest possible opposition to being rendered unconscious. It is remarkable that the organism is capable of some successful opposition, for, as has been already pointed out, pathological terror can raise the cardiazol threshold to a very considerable extent. Perhaps this can be compared to the experience of anaesthetists that some patients can by an effort of will resist the hypnotic effect of the anaesthetic to an unexpected extent. Some patients cannot be hypnotized, even with the aid of hypnotic drugs, if the psychic resistance is too great. Vasomotor changes have been shown to raise the threshold. It is reasonable to assume that such changes accompany the state of pathological terror and inhibit the fit, but do not operate to any significant extent in the milder reaction of fear. Pathological terror was confined to schizophrenics and did not occur in any of the melancholics treated, irrespective of the fact that they may have been by nature timid or that anxiety was a prominent feature of the illness. This is an illustration of the so-called psychic anaesthesia of the melancholic.

The Stages in the Returning of Consciousness.

From the psychological point of view, the period following the end of the clonic stage can be divided into various stages:

(1) *The stage of complete inactivity* while the vascularization and respiration are being restored to normal. In this stage no response can be obtained to the application of external stimuli.

(2) *The stage of involuntary movements*, the pathophysiological character of which has been described. This stage is not always clearly defined and sometimes cannot be observed. The patient sometimes gives the impression of indulging in these movements, sometimes of trying to suppress them. He is not quite inaccessible to external stimuli, as is shown by the return of the blink reflex. If one tries to restrain him in this stage he resists or avoids

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restraint in a purposeful way. For example, a patient who tried to leave the bed would brush aside obstacles placed in his way. He would even turn in the direction of spoken words and withdraw from painful stimuli, but he obviously could not understand speech nor would he speak himself.

(3) *The stage of semi-consciousness*, where contact between the patient and his environment is gradually being re-established. At first the patient looks round in apparent bewilderment. He does not speak spontaneously, and shows an obvious resistance on being pressed to speak. Mental activities take place slowly at this stage. Some patients try hard to overcome the difficulties. Others give up the struggle and turn away and try to sleep. Such patients give the impression not only of being disinterested, but of objecting to being disturbed. They curl themselves up in a foetal attitude on one side and draw the bedclothes over them. One sees from the expression and gestures of those who try to rouse themselves that the world around them appears to be unfamiliar, and they give the impression that they are trying hard to remember something that they once knew and have forgotten. While this bewilderment is prominent the patient's response to questions is poor and there is little attempt to fulfil commands. Questions, if answered, are met by "I don't know."

(4) The next phase can be described as *the period of aphasic reactions*. This phase is very short and cannot be observed in all patients. During this phase the patient tries to answer questions and obey commands; in his responses he shows aphasic phenomena. These phenomena occur in the following order:

At first the patient is unable to name any object or thing perceived. If asked to name an object he seems to search for words he has forgotten, sometimes employing circumlocutions. He is, however, able to volunteer remarks the extent of which seems to depend upon his power of attention. He will sometimes say "I don't know," or "I can't say."

Next the patient can name parts of his body and obey the examiner correctly and without obvious difficulty.

Following this, the ability to name familiar objects returns. The knowledge of names is still very limited, and at first only objects with which the patient has some personal connection are remembered. For example, a gold ring is easily named by married women at a time when a bunch of keys or a pen can be named only with difficulty.

Paraphasias do not seem to occur. There is, however, in some patients a form of expression in the naming of objects which, although not aphasic in itself, is observed only in this stage, while the difficulty of naming objects persists. This is the use of a particular qualifying adjective with each noun. The adjectives used are usually one of these, namely, "real," "little," "lovely," or a combination of two. For example, a wallet is named as "a real little wallet"; a ring as "a real gold ring"; a pen as "a lovely little pen," etc. That these expressions are peculiar to this stage is shown by the fact that they were not employed by the patients either before the fit or after complete recovery from it.

There is a wide variation in the purity and the development of this stage in different individuals or in the same individual on different occasions. Sometimes the entire aphasic stage seems to be too brief to permit of examination. On the other hand, the stage of aphasic reaction in certain patients may be of several minutes' duration. Perseveration is commonly observed.

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Typical results of the examination of patients in this stage are the following :

CASE 20.—Strong epileptic fit, with dilatation of pupils and turning upwards of eyes. Fit lasted 65 seconds, followed by strong rolling movements with outward flinging of the arms. Cyanosis of face lasted two minutes. Four minutes after the end of the myoclonic stage various parts of the body were named correctly, quickly and without difficulty. When asked to name other objects, the patient was very slow and seemed to have an obvious difficulty in doing so, expressing itself in perseveration, e.g. :

Objects presented :	Fountain pen.	Answers :	Pen.
	Nail file.		Pen.
	Pencil.		Pen.
	Purse.		No answer.
	Money.		No answer.

The patient would make no further attempt until consciousness was complete. In this case the greater facility in naming parts of the body than external objects was very obvious.

CASE 21.—Typical fit. Immediately after the fit patient was restless, with rolling movements, combined with slow movements of the eyes to both sides, more to the left. Attempts to name objects two minutes after the fit gave the following results :

Objects presented :	Watch.	Answers :	White and black.
			White, green, blue, black, white, gold.
	Nose.		Nose.
	Eye.		2 eyes.
	Coin.		3 eyes.
	5*d*. bus ticket.		5 D.
	Nose.		A little nose.
	Bunch of keys.		A real little key, not all alike.
	Watch.		A real little watch.
	Stethoscope.		A real —— ?
	Pen.		A real little pen.
	Mouth.		My mouth. Tooth and nose.
	Right arm.		Left arm.
	Golden ring.		A real gold ring.

Three minutes later :

Red tie.		The colour of real red.
Blue pencil.		A real blue pencil.

This examination gives examples of the use of qualifying adjectives and of perseveration. At the time when it was conducted any difference in the relative ease of naming parts of the body and external objects which may have existed had disappeared.

The aphasic phenomena described suggest that there is a short stage of what has been called amnesic aphasia, or in Head's (28) system, nominal aphasia. But in distinction to the typical amnesic aphasia, in these cases there is a short period in which naming of parts of the body alone is intact. That the aphasic reactions cannot be attributed to confusion or gnostic failure is shown by the general behaviour of the patients and the use of circumlocution.

Aphasic reactions of a similar type have been observed in chronic epileptics

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following fits, and in epileptic twilight states, where the reaction sometimes persisted for several days. A similar reaction, too, has been seen during the re-awakening from insulin coma (Stengel, 29).

The peculiarity of applying qualifying adjectives to objects, noted before, occurs also in chronic epileptics. In descriptions of Stengel (29), a chronic epileptic after a series of fits applied the adjective " real " to objects presented to him. It is well known that chronic epileptics habitually use qualifying terms such as " lovely " or " beautiful," which adds to their conversation a verbosity and unctuousness. This form of speaking is recognized as the result of a change of character in epileptics. It is surprising that this peculiarity, which in epileptics takes years to develop, can appear for a brief period after a cardiazol convulsion.

When the last traces of speech defect have disappeared, the patient seems to show a certain slowness of mental activity, with the desire to sleep and to avoid contacts.

The reactions described are not specific to cardiazol. I have been able to observe them in a smaller series of patients undergoing treatment by electrically induced convulsions.

Amnesia.

It has been suggested that the effects of amnesia contribute considerably to favourable results in cardiazol therapy, and that the amnesia is retrograde. The theory was put forward that abnormal mental contents, hallucinations and delusions, were also subjected to the amnesic process and were eventually forgotten altogether. The real facts of the amnesia were investigated in a group of ten cases undergoing treatment, chosen irrespective of prognosis or history. Eight pictures were presented to each patient 30 minutes before the injection. These pictures were chosen from subjects likely to be of general interest. In order to make quite sure that the material of the pictures had been understood, the patients were asked to describe the pictures at the time of showing. About an hour after the injection each patient was asked whether anything had been shown to him that morning. No patient, at any time, failed to recollect having been shown a series of pictures; three remembered all the pictures, and the remaining patients remembered a fair part of them; no patient remembered less than three of the series. Incidentally the three patients with the best scores were responding very favourably to treatment and afterwards recovered, while those with the lowest scores were those who seemed to have the poorest power of concentration.

The absence of a retrograde amnesia after the electrically induced fit was easily demonstrated. Many patients remembered the electrodes being strapped to their foreheads, and even the passage of the small calibrating current, just before the initiation of the fit.

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Examination therefore showed quite clearly that a retrograde amnesia is not one of the common after-effects of convulsive therapy, and cannot be regarded as a mechanism essential to cure.

However, there is always an amnesia for the period of the convulsion and for the time after it when consciousness is still clouded. No patient could recall features of behaviour during the period of semiconsciousness, irrespective of how long this period lasted. Many patients complain that there is some disturbance of memory during treatment. But the existence of such a disturbance can only be demonstrated within a period of an hour or so after the convulsion.

These amnesic features agree with what Gillespie (30) has described in an account of a personal experience of a cardiazol convulsion.

Induction of Symptoms.

In the course of recovery of patients during cardiazol treatment, I have observed a remarkable tendency which as far as I am aware has not yet been described. At the time when interest in personal delusions and hallucinations is fading, patients show a great interest in the experiences of others and sometimes a tendency to adopt their psychotic ideas.

For example, when one patient of a group of six was questioned about her menstrual condition, the other five within a day or two reported spontaneously irregularities in their own, although not one of them had sought advice on menstrual troubles before. It was obvious that their concern was not very deep-seated and had been inspired by the remarks of the first patient.

The increase of interest in others can be extended to cover hallucinatory experiences. One patient who had apparently lost her own characteristic delusional ideas reported that she was dead and had no body; she had copied another patient, who was then under treatment and expressed these beliefs. She admitted as much in examination, and no great difficulty was experienced eventually in persuading her that this idea was untrue. Another case, who had the idea that her "bowels were stopped up" and that she had been "wicked," transmitted these ideas to two other patients under treatment at the same time, who asserted quite emphatically that they "must be wicked too." These ideas were expressed each morning before treatment and persisted for a few days.

These are instances of the "induction" of pathological mental products, which occurs in *folie à deux*, and which is most frequently seen in paranoic subjects ("induced paranoia") and sometimes in cases of hysteria (Coleman and Last, 31). They are evidence of an increased susceptibility to influences from without the patient, in opposition to the autism of the schizophrenic, as if the wall between the schizophrenic and the outer world had become less impervious and less definite. At the same time, they are further evidence of the interest

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that the patient takes in the outer world, and of the tendency to enter into groups with common interests. Therefore this symptom, although in itself pathological, is a sign of growing sociability. It is of great practical importance for, as the examples suggest, the increased susceptibility of the patient may lead to injurious results if he is subjected to strong pathological elements in his environment. In addition, this heightened susceptibility may provide a good reason for refraining from questioning the patient about pathological ideas which are becoming less real to him. There is a certain danger of confirming delusions which the patient hopes to forget, by a process of what may be called re-induction.

I have been unable to ascertain from the literature or from personal experience if the induction of symptoms described above occurs in the course of spontaneous remissions. It seems probable that it does not, and if this is the case it provides one definite point of difference between the mechanisms of spontaneous and of therapeutic recovery.

Some Considerations on the Mechanisms of the Change of Symptoms.

It must be conceded that, irrespective of the ultimate therapeutic result, a certain change in the clinical picture occurs in almost every patient in the course of convulsive therapy. In what way the observations recorded in this paper can assist in throwing light on the mechanisms of the change of symptoms can now be considered. The importance that may be attached to a particular set of observations depends upon what personal conception is held as to the nature of the psychoses under treatment. For those who are inclined towards an organic aetiology, the pathophysiological observations will be of greater interest than the psychological. But in the present state of limited knowledge neither aspect can be neglected.

There is no doubt that the cardiazol fit brings about a repeated change of functional relations between cortical and subcortical parts of the brain. This is shown by the transitory appearance of types of movements related to the deeper centres, which, as has been described before, occurs before and after the fit proper, in addition to the typical motor phenomena of epilepsy. This indicates that the relationship between different systems of the nervous organization is deeply disturbed by the action of the drug, and that a great readjustment and re-establishment of this relationship must take place before full consciousness returns. It is not unlikely that the process of readjustment has some lasting effect upon the nervous organism as a whole, and that in the course of repeated disturbances and reorganization of neural relations some fresh orientation takes place, bringing the parts of the nervous system into a state of more normal balance.

If one feels justified in regarding schizophrenic symptoms from a neuropathological angle, as Kleist (32) *et al.* have done, one finds in schizophrenics

https://doi.org/10.1192/bjp.86.364.799 Published online by Cambridge University Press

various signs of physical change that can be related to neurological syndromes. The states of hypo- and hyperkinesis in schizophrenia have their counterparts in forms of postencephalitic Parkinsonism. The disturbance of affectivity has been related to functional disorders of deep centres, especially the hypothalamus. Similar views have been expressed with regard to the affective psychoses. Cushing (33), Foerster and Gagel (34) and Alpers (35) record manic states in hypothalamic disorders. These views received practical importance in the works of Moniz (36), who has reported successful results in schizophrenia and melancholia, after severing the connection between the deeper parts of the brain and the frontal lobe.

Therefore, it may be suggested that a physical agent which brings about repeated alterations in the functional relation between deeper and higher levels of the brain, such as has been observed in induced epilepsy, may lead to a re-establishment of normal functional relations between these centres. Although such a theory is far from proven, it has the advantage of being related to clinical observations, and it may have some value as a working hypothesis.

The possible objections to the "organic" theories have led some workers to seek a psychological approach to this problem. The psychological theories have the advantage of embracing a wider field and being non-exclusive to other forms of shock treatment. Further, they are based on what is known of schizophrenia, and the conception of schizophrenia itself is after all psychological.

In dealing with the psychological mechanisms, use will be made of observations recorded earlier in this paper. There are three psychological events in the course of a single therapeutic convulsion, each of which provides an interruption in the psychotic life of the patient.

The first event is the realization of being treated. This realization is evidenced by various attitudes on the part of the patients, and it is striking that, irrespective of their behaviour, nearly all patients seem to realize that the therapeutic interference is intended to be beneficial. The treatment seems to stir up an innate tendency to be restored to normal health.

The second event is represented by the return from the death-like state of the epileptic fit to reality. It has been shown that this return is gradual and that mental functions return in a definite order. The state of aphasia is of special interest for an interpretation of the psychic readjustment towards reality. It has been described how the patient in this stage is able to identify and name parts of his own body before any object of the external world. This phenomenon can hardly be ascribed to a disturbance of organic function alone, but must be determined to some extent by psychological conditions. Hughlings Jackson (15) has shown to what extent psychological and personal conditions can influence the capabilities of the aphasic. If one regards the order in which the faculty of naming is restored as reflecting the re-establish-

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ment of the patient's relation with the world, one finds self-interest, or rather an awareness of the ego, returns first, to be followed by an awareness of other objects and the relationship they bear to him.

In chronic epilepsy similar phenomena can be observed, and seem to suggest that personality changes may influence or even determine certain features of speech disorders. Chronic epileptics, as is well known, are highly self-centred, and their personality undergoes a change in the direction of restricted egotism. Another point of similarity between the reactions of chronic epileptics and those of patients awaking from induced convulsions is their habit of using certain adjectives which are evidence of an exaggerated emotional attitude towards objects of the environment. It may be that this similarity is not accidental, but has a bearing on the psychological readjustment of the psychotic patient towards his own personality and the outer world. Repeated induced epileptic fits in schizophrenics might lead to what has been described as an epileptic feature of the personality. It may well be that the appearance of such features of the epileptic personality might prove antagonistic to the schizophrenic dissociation and result in benefit to the patient.

There is one fundamental difference between the epileptic and the schizophrenic; the epileptic experiences his personality as a whole, even to an exaggerated extent, while the personality of the schizophrenic is "split up." From the psychological point of view these features are really antagonistic. It is possible that the epileptic seizure, which overwhelms the whole organism, compels the schizophrenic to re-establish the unity of his personality. It may be for a similar reason that autistic schizophrenics have been known to react normally in situations of real danger. The assumption that the temporary imposition of epileptic features produced by convulsion therapy contributes to the change of the schizophrenic personality, is in no way related to Meduna's unjustified assumption of a biological antagonism between schizophrenia and epilepsy.

The third event is the encounter with the environment after re-establishment of consciousness. This takes place when the patient is fully conscious and in full possession of his mental capacities, but still experiencing the physical aftermath of the shock. In this stage the patient feels dependent and shows a tendency to attach himself to persons in his environment. Aggressiveness is usually absent. This stage might last for hours, or even endure. The convulsive episode has forced the patient to assume an infantile relationship towards his environment, absolutely opposed to the psychotic attitude. The nature of the new situation is sometimes indicated by an outpouring of eroticism, which obviously has the same source as the tendency to express a pleasant emotional feeling towards objects during the stage of aphasic reactions, as described before. Repeated occurrences of the state of dependence on others, which replaces for a while the psychotic attitude, may very well lead to a definite breaking of the psychotic withdrawal from human society.

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Such a conception as this of the return to an infantile state may have been in the minds of those workers who have attributed the therapeutic action of shock treatment to psychic death and rebirth. The interpretation given above seems to be in closer relationship to objective clinical observations.

Such psychological considerations are applicable to all forms of shock treatment and other forms of treatment that operate through the production of a change of consciousness, e.g. continuous narcosis and general anaesthesia. It is, however, necessary to realize that the disturbance of consciousness in continuous narcosis is not so profound as in epileptic fits, and therefore the adjustments which the patient must make in the process of recovery from these conditions are necessarily less extreme.

This theory is also applicable to improvements observed in melancholic depression and mania treated by forms of "shock." In these states, too, the relationship of the individual to the outer world and also to himself is disturbed, and perhaps in the course of shock treatment readjustment takes place. Observations are not sufficient to allow the analysis of the course of this readjustment. It is not uncommon to encounter cases of melancholia where marked improvement has followed a serious and unsuccessful attempt at suicide, especially where, as in gas poisoning, hanging, and drowning, the attempts were followed by unconsciousness. Such cases may be examples of the beneficial influence of forms of shock in affective psychoses.

It might at first sight be argued that a psychological theory must be an alternative to an organic. But if it be held that the manifestations of affectivity are the expression of processes taking place in the deeper parts of the brain (and this can be regarded as proved by what is seen in certain brain disorders, such as postencephalitic Parkinsonism), it becomes clear that the psychological theory for an alteration of affectivity is not necessarily opposed to a theory derived from observations on organic activity. Both theories really postulate the same mechanism, but reach their conclusions by the investigation of two entirely different forms of human behaviour, motility and affectivity.

In the interests of economy of space it is not possible to publish case-histories nor to discuss the therapeutic results, which in my series were not markedly different from those obtained by the majority of other workers. The presentation of the views of others and of bibliographical references have for the same reason been reduced to the minimum.*

I should like to express my gratitude to Dr. J. J. B. Martin, Medical Superintendent of the Bristol Mental Hospital, for permission to publish the result of these investigations and for his constant interest and encouragement, and also to Mr. Grey Walter, of the Burden Neurological Institute, who was responsible for the application of the electrical treatment.

* The last number of this Journal, published in May, 1940, contains several interesting papers dealing with various aspects of convulsive therapy. This paper was completed before it was possible to consider these contributions.

https://doi.org/10.1192/bjp.86.364.799 Published online by Cambridge University Press

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