combination of spinal analgesia with a light general anaesthesia must not be lost sight of. Such a plan, like the adoption of nerve blocking and nitrous oxide anaesthesia, would seem to combine the advantages of both methods.

The dangers from sepsis in cases of diabetes lie in the special liability of such patients to septic conditions, due to the poor nourishment of the tissues owing to endarteritis, the interference with the proper nerve supply owing to neuritis, and the presence of free sugar in the blood and tissues forming a good breeding ground for micro-organisms. These dangers require special precautions apart from the ordinary measures taken in every surgical operation. For example, the employment of a tourniquet is best avoided, and every effort should be made to prevent bruising of the tissues from rough handling. The question of drainage is a serious one. In the case reported the drainage opening would not heal for some time and was exceedingly sluggish and indolent in appearance. Sinclair White⁵ has recommended that "drainage should be unnecessary if haemostasis has been properly attended to." While considering such a practice to be somewhat risky, since a collection of fluid in the flaps would be a source of more danger than the insertion of a drainage tube, I cannot but think that the drainage opening should be small, and that a suture should be inserted ready for tying when the tube is removed.

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The Morison Lectures

ON

EPILEPSY.

DELIVERED BEFORE THE ROYAL COLLEGE OF PHYSICIANS
OF EDINBURGH.

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LECTURE II.—THE PROBLEM OF EPILEPSY (*continued*).

TOXAEMIC EPILEPSY.

It has for a long time been contended that an excitant of epileptic seizures might be found in toxic or autotoxic causes arising in connexion with the body metabolism or gastro-intestinal disorders. The discrepancies, however, in the results of observations by different workers upon urinary and blood toxicity in epilepsy have not afforded satisfactory evidence upon which to found such a theory. It is now generally accepted that the altered condition of the urine and blood which has been found in association with epileptic attacks by many observers is the temporary effect and not the cause of the seizures. Some explanation of these variations may be found in differences in the nature of the cases upon which the researches were carried out. Changes have not been found in the minor, or "petit mal," type of the disease. Those cases which have shown the most pronounced reactions both of blood and urine have been instances of serial epilepsy, the status epilepticus, and fits accompanied by acute mental symptoms—types of the disease in which, from the general constitutional disturbances, there is more reason to suspect an infective or toxic causation.

Binswanger¹ has called attention to a small group of epilepsies which may have a toxæmic basis. This group is characterized by—

(a) Well-marked premonitory signs, of which the most common are of a psychological type; irritability, fitfulness, quarrelsomeness, lethargy, somnolence and delusions.

(b) Fits of frequent occurrence developing either into serial epilepsy, or the status epilepticus, accompanied by constitutional disturbances, such as furring of the tongue, constipation, acceleration of the pulse and elevation of temperature.

(c) More or less prolonged intervals free from attacks between the seizures.

I pass over with a mere reference the observations of Voison and Peron,² who found a hypotoxic condition of the urine before and a hypertoxic condition after the fits; of Haig,³ who laid stress upon the relation of epileptic fits to the normal diurnal variations in the excretion of uric acid; and of Krainsky,⁴ who attributed the convulsions of epilepsy to the formation of carbamic acid, one of the derivatives of urea.

The presence of cholin in the blood and cerebro-spinal fluid of epileptics was originally described by Donath.⁵ This substance, which was found by Mott and Halliburton⁶ in the cerebro-spinal fluid in organic nervous disease, was attributed by them to disintegration of the myeline sheaths of the nerve fibres. Some observers hold that this is the cause of convulsions, owing to its toxic action, as it has also been found in cases of general paralysis with epileptiform seizures. On the other hand, it has been looked upon as the result of the fits. It has been shown experimentally (Dide, Donath) that moderate doses of cholin injected into the circulation do not produce convulsions, but that in large doses both convulsions and paralysis may result. Buzzard and Allen⁷ showed that the large doses of cholin necessary to produce convulsions in animals were much in excess of what could be produced under ordinary conditions in man. In a recent paper Donath⁸ records the results of his experiments into the convulsion-producing properties of the substances found in the blood and urine of epileptics. He showed that when injected into guinea-pigs and dogs, uric acid, neutral urate of sodium, carbonate of ammonium, kreatin, and lactic acid were entirely innocuous. The only convulsion-producing substances were ammonia and the organic ammonium bases—trimethylamin, cholin, kreatinin, and guanidin; the last body is not found in human urine, but is one of the uric acid products.

A generally recognized feature of infection is increased coagulability of the blood. Dr. John Turner⁹ has shown (1) that the average rate of coagulation is quicker in severe cases of epilepsy; (2) that the rate is quickened during the period that the patient is having fits (serial epilepsy); (3) that there is a further quickening of the coagulation rate up to twenty-four hours before a seizure; and (4) that, from twenty-four to forty-eight hours after a fit, there is a rebound and retardation in the rate of blood coagulation.

Intravascular clotting occurs clinically in cases of infection, most probably as a result of the liberation of nucleo-proteid. Dr. John Turner¹⁰ has suggested that just before an attack the leucocytes and blood plates shed their nucleo-proteid. In consequence a hyaline material is found in the smaller arterioles and capillaries, obstructing the free course of the circulation. These appearances are most conspicuous in the brains of persons dying in status epilepticus, as well as in general paralytics in whom epileptiform attacks have occurred. It is, therefore, not improbable that some types of epileptic convulsions, notably serial epilepsy and the status epilepticus, may be associated with nucleo-proteid thrombosis arising from toxæmic influences within the body. A further sign of toxæmia, described by Lewis Bruce as occurring in some forms of acute insanity of toxic origin, is a hyperleucocytosis associated with a high percentage of polymorpho-nuclear cells. It would appear from the observations of Pugh and Bruce that a hyperleucocytosis was found in cases of serial epilepsy, and those complicated with acute mental symptoms.

The general conclusion, therefore, which might be drawn from these observations, is that the type of epilepsy due to toxic causes may be found in those acute forms of the disease known as serial epilepsy and the status epilepticus. These may, therefore, be regarded as accidental occurrences in the course of the disease.

THE IMMEDIATE CAUSES OF EPILEPTIC SEIZURES.

It has been observed that the onset of an epileptic fit may be accompanied by a temporary cessation of the heart's action and the pulse beat. This transient stoppage is attributed to cardiac inhibition; the loss of consciousness and the fit to the resulting sudden cerebral anaemia. The most recent advocate of the cardiac inhibition theory of the causation of epileptic seizures is Dr. A. E. Russell,¹¹ who

has collected a quantity of evidence in its favour in his Goulstonian Lectures for 1909. He does not, however, contend that complete cardiac arrest occurs in every epileptic convulsion, for the pulse may persist throughout a fit, although in his opinion an extreme feebleness, short of absolute cessation, might be sufficient to initiate a fit.

It is well known that convulsions may be due to the sudden cutting off or to the prolonged deprivation of the cerebral blood supply. The convulsions of Stokes-Adams disease, the Kausmall-Tenner fits provoked by ligature of the internal carotid arteries, and the spasms consequent upon electrical stimulation of the peripheral end of the cut vagus nerve are all due to this cause. Russell contends that the fundamental factor underlying the epileptic fit is cerebral anaemia, probably associated with vaso-constriction of the cerebral blood vessels. He holds that the sudden and profound loss of consciousness, so characteristic of the major epileptic seizure, is not due to an intense "discharge" from the cortical grey matter, as is commonly stated, but to a sudden cessation of the whole cerebral circulation. This may vary in degree, so as to cause either transient unconsciousness or a severe convulsive fit.

The direct evidence in support of this view is meagre, apart from the temporary cessation of the heart's action sometimes observed at the commencement of an epileptic fit. It would be, therefore, well to look for collateral evidence which might support the vasomotor spasm theory of the origin of epileptic fits. A comparison has been drawn between what occurs in epilepsy and some of the phenomena of Raynaud's disease. The retinal arteries have been found on ophthalmoscopic examination to be narrowed during the paroxysmal attacks of impairment of vision which occur in some cases of this disease. The fundus of the eye has also been examined ophthalmoscopically at the commencement of an epileptic seizure. Sir William Gowers did not observe any vaso-constriction of the retinal arteries under these circumstances. Recurring attacks of transient hemiplegic paralysis have been described by W. Russell, Langwill, and others, as due to 'intermittent closure of the cerebral arteries—a condition which might, if sufficiently sudden and complete, conceivably give rise to convulsions in predisposed persons. The well-known phenomena of migraine, also are in many ways favourable to the hypothesis of vasomotor spasm as the underlying cause. Leonard Hill¹² produced a typical fit in himself by digital compression of one carotid artery. He says :

The first effect on applying the compression was a sensation in the eye on the same side: then there followed a sensory march of formication down the opposite side of the body. This began in the fingers, spread up the arm, and then down the leg. Finally, clonic spasms of the head occurred, accompanied by an intense feeling of vertigo and alarm.

The evidence in favour of cerebral anaemia being the cause of convulsions is therefore clear, but as yet there is no direct demonstration of either vaso-constriction or cerebral anaemia being the exciting cause of the recurring seizures of epilepsy.

Paralysis of the vaso-constrictor nerves rendered permanent by removal of the superior cervical ganglion has not led to that favourable outcome which was expected of it when introduced as a means of treating epilepsy, on the theory that the fits were due to cerebral anaemia from vaso-constriction.

Admitting the instability of the vasomotor system as a feature of most epileptics and vaso-constriction and cerebral anaemia as a cause of some fits, we have still to arrive at an explanation of the cause of a periodic tendency to arterial spasm.

Whether the primary excitant of the epileptic attack be a spontaneous "discharge" of nervous energy, or cerebral anaemia induced by vaso-constriction and cardiac inhibition, the fit would seem to originate in some portion of the cerebral cortex. I accept the old view of Hughlings Jackson that epilepsy is a disorder of the highest level, and I contend that the observations of many subsequent workers—anatomical, experimental, and clinical—have placed the highest level between the intermediate precentral and prefrontal zones.

Campbell¹³ has shown that in association with each recognized cortical centre there exists a corresponding

higher or psychical centre. He has described the psychomotor, psycho-sensory, psycho-visual, and psycho-auditory centre in close relation to the motor, sensory, visual, and auditory centres, situated respectively in the precentral, occipital, and superior temporal gyri. If a higher olfactory centre exists, it is probably to be found in the lobus pyramiformis, but its delimitation is less clear than in the case of the other centres.

The nature of the aura determines the portion of the cerebral cortex in which the fit starts. Warnings of special sense are either crude sensations or sensations of a more elaborate and psychical character. The latter are especially characteristic of idiopathic epilepsy. It is, therefore, conceivable that the "discharge" commences either in the lower or higher portion of a special sense region, according to the nature of the warning sensation.

A noteworthy point is the constancy of the warning in particular cases. As Herpin pointed out, the warning sensations, however diversified they may be, are always, or nearly always, similar in the same subject. There are only few exceptions to this. During the course of the disease the aura of the major seizure may disappear, but the minor attacks retain the character of the original warning.

In the case of the large number of epileptics in whom no warning of the seizure is present, but in whom the fit is ushered in by a sudden and profound loss of consciousness, the discharge conceivably originates in the frontal area. Here the onset of the fit is essentially psychical. The patient is dazed, or stuporous, or drops down suddenly. In many of these cases transient conjugate deviation of the eyes occurs, or a cry is uttered.

The completeness or incompleteness of an epileptic fit—that is to say, whether the attack is an aura sensation, a minor, or a major seizure—depends upon the intensity, duration, and extent of the cortical discharge. Hughlings Jackson's idea of epilepsy was the occasional sudden, excessive, violent discharge of a nerve centre, sensory, motor, or psychical. The conditions which favoured such discharge were congestion and excess of blood in the cortical capillaries, with retarded motion and consequent alteration of the nutrition of the cortical cells. This view has lately been revived by the observations of Dr. John Turner, who has found in the brains of persons dying from epilepsy minute thromboses of the cortical capillaries. He suggests that just prior to the onset of a fit the blood plates shed their nucleo-proteid contents. The result is the formation of granular or hyaline thrombi, which in persons hereditarily or structurally predisposed to cortical instability is sufficient to provoke a cortical discharge and epileptic fit.

Whether in any particular case the morbid condition, which gives rise to the "explosion" is inherent in a cortical centre, common to the whole cerebral cortex, or is attributable to a vasomotor instability or blood condition, are points which still remain mysterious.

It may be asked: What is the cause of the enormous variability in the frequency of epileptic seizures—a frequency which varies from two or three fits in a year to several score in twenty-four hours? Is the same cause at work in both these types of the disease?

In whatever part of the cerebral cortex the discharge arises it rapidly spreads along the association and commissural tracts so as to involve both sides of the brain, although one side is usually more affected than the other. Hence it comes about that the examination of the reflex, motor, and sensory systems after a major epileptic fit reveals evidence of temporary paralysis, and of alterations in the reflexes, indicative of a transient disturbance of the pyramidal system. The knee-jerks are at first abolished and then exaggerated, the abdominal reflexes are lost, and the plantars show a temporary extensor response. These symptoms vary in accordance with the character of the seizure. They are not present after minor attacks; they may be observed on one side only, in cases where the convulsions have been preponderantly unilateral. They are most obvious, and persist longest after serial outbursts or the status epilepticus. Temporary impairment of motor power (exhaustion paralysis) is observed on one or both sides, but more especially on the side which has shown the greater convulsion. Diminution or loss of cutaneous sensibility may also be found after a fit. It is most marked in the limbs which show the greater motor weak-

ness. Amblyopia, deafness, and impairment of smell and taste may also be of temporary occurrence.

THE BORDERLAND OF EPILEPSY.

I have adopted this term from Sir William Gowers,¹⁴ who has recently described a number of conditions which are near to epilepsy but not of it.

In the sense used by Gowers, the term includes some phenomena which will not be discussed in this lecture, such as the minor seizures of labyrinthine disease, attacks often difficult to differentiate from epilepsy; migraine, which will not receive further consideration here, although the paroxysms of this disease are well worthy of study as they reveal much that is interesting in connexion with epilepsy; and vertigo, a general symptom of intracranial disease, which would lead us too far away from the prime object of the lectures.

Epilepsy and Hysteria.

In any discussion, therefore, of the phenomena on the borderline of epilepsy, brief consideration should, in the first place, be given to hysterical seizures and their differentiation from epileptic attacks.

Both epilepsy and hysteria are diseases founded upon a hereditary degenerative endowment. Both disorders have a common clinical symptom, namely, the periodic occurrence of paroxysmal attacks, characterized by an alteration of consciousness. This is hardly the place to enter into an analytical consideration of the distinction between these two great psycho-neuroses apart from their paroxysmal phenomena.

I have in the preceding lecture attempted to point out what may be regarded as the outstanding symptoms of epilepsy, and have endeavoured to show that although in recent and slight cases it may remain throughout a so-called "functional" disease, in the severe and the confirmed cases with mental impairment the symptoms become grafted upon an organic alteration of the cortical tissues. Epilepsy, in consequence, is a dementing disorder. Hysteria, on the other hand, remains throughout a purely functional and mainly psychological disease. According to Janet,¹⁵ its fundamental characteristic is a dissociation or severance of some mental processes from the main personal consciousness. It has no pathological anatomy, and no tendency towards dementia.

Clinical study of the attacks which occur in these two diseases has led to difficulty and confusion rather than to simplification in their differential diagnosis. The most recent observations tend to show that there is no such generally recognized condition as "hystero-epilepsy" in the usually accepted sense. Binswanger states that interparoxysmal hysterical symptoms may be added to genuine epilepsy and vice versa. It would also appear probable that a prolonged hysterical attack may terminate with symptoms in no way differing from those of genuine epilepsy. Bratz and Falkenberg found that all their cases were either hysteria or epilepsy, or a combination of the two diseases.

Major hysterical attacks are, as a rule, fairly distinct in character from major epileptic seizures. But the minor attacks of both disorders are sometimes not easy to differentiate. If an attack is being described by an untrained onlooker it may be a difficult matter for a physician to decide whether the paroxysms are those of epilepsy or hysteria, as the duration of the minor seizures in both diseases is almost the same and they may both occur in series. Moreover, the perversion or dissociation of consciousness in the hysterical attack may readily be mistaken for the abolition or impairment of consciousness which is the characteristic feature of the epileptic seizure.

On the other hand, if the attack should be observed by the physician, there is, as a rule, less difficulty. The rolling or squinting movements of the eyeballs, and the oscillations of the head in the hysterical attack, are quite distinct from the tonic conjugate deviation of the head and eyes in the epileptic; the tonic spasm of the limbs inducing flexion of the arms and extension of the legs in the latter are in contrast to the tonic spasm mainly of the muscles of the neck and back in the former; the steady development and eventual yielding of the clonic spasms in epilepsy stand out in striking contrast to the vibratory tremors of

the hysterical attack; and the sudden relaxation of the spasm, and the almost instantaneous return to consciousness in the hysterical seizure is a feature of noteworthy importance. The hysterical seizure also differs essentially from the epileptic in the three following additional features:

1. The hysterical seizure does not terminate in psychical disturbance, confusion, or automatism. Hystericals come out of their attacks suddenly, and often express a feeling of relief after the attack is over.
2. As pointed out by Janet, there is a possibility of awakening the phenomena of the hysterical seizure by hypnotism. Epileptics are not at all hypnotizable, and in consequence it is not possible to produce in them a real somnambulistic state with consecutive amnesia.
3. Should it be possible to examine the reflexes of the patient recovering from a seizure, certain changes will be found in most cases of major epilepsy.

The most important transient changes indicating disturbances of the pyramidal system are alteration of the plantar reflex from the normal flexor to the abnormal extensor response, abolition or impairment of the abdominal reflexes, and exaggeration of the knee-jerks.

If, therefore, it is not always possible to distinguish the epileptic from the hysterical seizure during the attack, an examination of the patient at or immediately subsequent to its termination will usually be sufficient.

EPILEPTOID PHENOMENA.

I now enter upon a subject of great complexity. There is no doubt that many people are prone to attacks which cannot be classified either under hysteria or epilepsy. The attacks are in the main purely subjective, but in some cases objective phenomena, chiefly of a circulatory and vasomotor character, may be observed.

Numerous writers, whose special work lies chiefly in neurological practice, have described attacks having features which will be depicted presently. Owing to the absence of any systematic classification, there has been a tendency to group the symptoms under different headings, each author devising his own terminology for the sake of convenience or according to his conception of the malady.

Psychasthenic Attacks.

Oppenheim was the first to describe, under the term "psychasthenic attacks," certain periodic seizures, mainly because they occurred in persons subject to common psychasthenic symptoms, such as fears, dreads, apprehensions, obsessions, and tics. The leading features of these attacks were:

- (a) That they did not occur during childhood.
- (b) That there was a special cause for the attacks.
- (c) That they were of infrequent occurrence.
- (d) That they were characterized by prolonged unconsciousness without convulsion, and only slight twitching of a few muscles.
- (e) That there was an absence of mental changes.

Such attacks would seem to be extremely rare, and in my opinion difficult to differentiate from epilepsy.

The Dreamy State.

Janet¹⁶ has described under the term "psycholepsy" conditions characterized by an alteration of consciousness, which favours the development of a "dreamy state," in which a feeling of unreality or of non-existence is the outstanding symptom.

Mention has already been made of the "dreamy states" described by Hughlings Jackson as the warning of some fits with epigastric and olfactory auras. It is important to distinguish between this aura sensation and the condition which is now being described. Patients describe the mental state as a feeling of non-existence, with doubts as to the reality of things, even as to the material existence of their own bodies; they also have doubts as to whether what they see and hear are real. It may more appropriately be termed a "reverie" state, and is frequently associated with feelings of an obsessional or psychasthenic character. Symptoms of this kind are not uncommon in epileptics. Crichton-Browne and Pick (of Prague) have described them as becoming permanent in some cases of confirmed epilepsy, and I have seen and described them as psychical epileptic equivalents.

Other Periodic Seizures.

Sir William Gowers,¹⁴ in his most instructive lectures, has described seizures whose symptoms were referred chiefly to the distribution of the pneumogastric nerve. To these he has given the term "vagal or vaso-vagal attacks."

Finally, Dana¹⁷ has designated under the term "para-epilepsy" seizures of a psychical, vertiginous, or vasomotor type, occurring in neurotic or psychasthenic individuals, more especially about adolescence. As these seizures, according to this observer, resemble "aura fits," or abortive epileptic attacks, he has classed them in a group of para-epileptic phenomena.

It is therefore clear that observers now recognize a type of periodic seizure, which is neither hysterical nor epileptic, occurring in persons of a neurasthenic or psychasthenic temperament. Attacks of a not dissimilar kind, however, may be observed in subjects of genuine epilepsy, and there are other attacks in which the diagnosis from hysteria is most difficult and uncertain.

These attacks may therefore be provisionally placed in a group by themselves, but it seems to me as if a name is still lacking which will fitly embrace the whole series.

I have suggested the term "epileptoid" as one which seems to include all the symptoms of a paroxysmal and recurring character which lie upon the borderline of epilepsy.

It would seem possible from our present knowledge to subdivide these symptoms into two groups:

(a) Attacks having features especially of a vasomotor character.

(b) Attacks having symptoms especially of a psychical character.

Vasomotor Attacks.

These seizures are characterized by throbbings, thumpings, and beatings of the heart, flushings of the face and a fullness of the head, dizziness, and frequently sensations of a paraesthetic character in the extremities, such as numbness, tingling, and pricking.

They may be of daily occurrence, and last from a few minutes to several hours. They occur in persons of a nervous temperament. Dana notes that there is usually an associated element of thyroidism. In one case of my own there was a decided tendency towards dermographism.

Gowers also describes seizures characterized by definitely vasomotor symptoms, such as coldness of the limbs, pallor of the face, shivering almost amounting to rigor, tingling and numbness of the extremities, and sometimes a slight tetanoid spasm.

A fuller development of these seizures, bringing them into a relationship more akin to epilepsy, is well illustrated by the vaso-vagal attacks described by Sir William Gowers. The symptoms as given by him are:

1. Sensations referred to the stomach, heart, and respiratory systems.
2. The ascent of the sensation from the stomach to the chest, throat, and head.
3. It is accompanied by a feeling of respiratory distress and cardiac oppression, fear, and a sense of impending death.
4. There is no true loss of consciousness, but the mental operations are slow, and sometimes characterized by a feeling of unreality.
5. The attack ends with a great acceleration of the heart's action.
6. The whole attack lasts for about fifteen or twenty minutes.

Women suffer more frequently from these attacks than men. This, and the fact that the pneumogastric and vasomotor systems are readily influenced by emotion, have probably led to the frequent submergence of these attacks beneath the vague conception of hysteria!

Psychical Attacks.

These are seizures in which the outstanding features are of a psychical character—usually a sense of apprehension, dread, or fear. They are commonly associated with sensations of nervousness, numbness, coldness, nausea, cephalic sensations, and frequently accompanied by sensations of a vague, indescribable character. Sometimes the feeling of fear is intense, and may be replaced by a sense of impending death.

In the true attacks of this kind consciousness is never abolished, but the mental state may assume a dreamy attitude, with a feeling of unreality. The attacks occur in persons who present other features of a psychasthenic

character, such as dreads, fears, and apprehensions. There is usually also a well-marked neuropathic heredity. Although in many cases the attacks are of the kind just described, there are other cases in which they merge into epileptic seizures.

The attacks described here as "psychical" are clearly closely related to those of a more purely vasomotor type, the vaso-vagal attacks of Gowers being seemingly the connecting link between the two types of seizure, as well as between them and attacks of an epileptic nature.

Relation to Epilepsy.

This is a suitable place to draw attention to some of the outstanding features of these attacks, and to compare them with epilepsy.

One of the commonest forms of warning in epileptic seizures is the epigastric sensation, consisting of a subjective sensation referred to the stomach or upper abdominal area. Epigastric sensations are of various kinds. Some are vague and indefinite, others are of an indescribable character, and others are painful, spasmodic, or cramp-like. These sensations may or may not be associated with gastric disturbances, such as flatulence or any abnormal condition of the stomach contents. In the majority there are no signs of local gastric disorder.

Closely allied to the epigastric sensation is the cardiac warning, which consists mainly of palpitation of the heart, and sometimes of pain, and is generally accompanied by a sensation of suffocation, strangulation, or oppression, sometimes with a sensation of impending death. So closely does the condition resemble angina pectoris that a variety of minor epileptic attacks has been designated "epileptic pseudo-angina." A notable accompaniment of the epigastric warning and its allied sensation of cardiac oppression is sensation of intense fear, or apprehension, and sometimes a desire to be alone.

The symptom of fear, in association with fits accompanied by epigastric warning, is well known, and was pointed out by Hughlings Jackson many years ago. But it is not confined to fits with epigastric warning. It is also found as a very characteristic feature in that remarkable group of fits known as "uncinate seizures," or epileptic attacks with a warning of smell and taste, due to organic disease of the uncinate gyrus. In addition to the sense of fear, which has been already mentioned, a peculiar "dreamy state" has been described in which consciousness is lost or perverted.

Whatever may be the explanation, it would appear to be a well-recognized fact that a definite relation exists between epigastric sensations and a sensation of fear or apprehension. This relation is not confined to epileptic attacks. It is also present in other conditions. In many states of fear or alarm a vague sensation is referred to the abdomen. Persons who suffer from melancholia constantly refer to the feeling of weight or oppression in the region of the stomach; and many individuals, obsessed by some fear or panic, refer to a vague and indefinite sensation in the stomach as the source of their discomfort.

If we now apply the principles which have been already laid down when considering the march of an epileptic attack, it is obvious that minor epileptic seizures may occur, characterized by an epigastric or cardiac sensation, accompanied by a sensation of fear, or of oppression or even of impending death, in which consciousness may be retained, or at the most merely blurred or perverted.

I have ventured at some length into this discussion, because, as Dana has shown, the attacks already described as "psychical seizures" have many of the qualities of epileptic auras and abortive epileptic fits; hence the term "para-epilepsy" which he has applied to them.

The close relation between epilepsy and the vaso-vagal attacks has also been emphasized by Sir William Gowers, who regards them as a long-drawn-out or extended epileptic seizure. He says:

Some of the facts we have glanced at suggest that it is possible that the elements of an epileptic attack may sometimes be extended, and thereby made less intense, though not less distressing. . . .

If we could conceive a minor epileptic attack that is extended, its elements protracted with no tendency to be terminated by loss of consciousness, its features would be so different, that its nature would not be suspected. Swiftness is an essential element of ordinary epilepsy, but this does not preclude the possibility of deliberation.

Dr. A. E. Russell, who has already been quoted as the most recent supporter of the cardio-inhibitory theory of epileptic seizures, interprets these attacks in the following way:

A sudden depression of the normal vasomotor tone in the splanchnic area leads to a rapid accumulation of blood in the capacious vessels of that region. If only momentary in duration the result is merely the sudden sinking at the pit of the stomach. If of longer duration this preliminary sensation or aura is succeeded by a faint. . . . The dreamy mental state and the fear of impending death may be due to the vascular depression, which must of necessity affect the brain. If consciousness is lost it shows that the vascular depression has reached a point incompatible with consciousness, and that this is the missing link connecting it with epilepsy.

The position of these seizures in the periodic phenomena allied to epilepsy therefore depends upon the view taken of the nature of epileptic attacks. Russell regards the whole series, from simple syncopal attacks or faints up to epilepsy, as primarily of vasomotor origin. He favours the doctrine that such symptoms are signs of "a vasomotor ataxia," a condition in which the vascular mechanism is unable to adjust itself to strain. Whether this is the true explanation of a remarkable series of phenomena is a point upon which it is difficult to offer an opinion. The theory just expressed certainly brings a train of not dissimilar periodic seizures into line.

Epilepsy, however, seems to me to be primarily so much a cerebral or psychical disorder that I hesitate to classify it along with faints and syncope as a disease of the vasomotor mechanism. Many of the features of the borderland conditions just described are not psychical, but seem to be the consequences of functional disturbances of the lower centres in the medulla oblongata. I would also emphasize the fact, already referred to in the first lecture, that the convulsive element is only one feature of epilepsy. True epilepsy is always accompanied by some psychical alteration, apart from symptoms of a psychasthenic kind. I am not aware that the epileptoid conditions just described ever present any tendency to mental impairment or dementia.

Sleep Phenomena.

The physiological condition underlying sleep is especially favourable for the development of epileptic attacks. It was shown in the first lecture that the few hours after falling off to sleep are the hours most favourable for the occurrence of epileptic fits. In consequence of this there is in many cases of epilepsy a striking regularity in the periodicity of the attacks, a fit occurring at or about the same hour on each occasion. This period of deepest sleep is also a favourable time for certain sleep symptoms which appear to be upon the borderline of epilepsy—namely, night terrors in the adult. Night terrors are common in neurotic children, but their significance is merely that of an unstable nervous system. Their occurrence in adults, or their continuance from childhood into adult life, is of greater significance, and may raise the question of their epileptic character in some cases. They usually reveal themselves in the utterance of loud piercing screams, sometimes a single scream, at other times more frequently repeated. Their chief characteristic is that, having occurred at some time during the first few hours after falling off to sleep, they do not occur again during the same night. They have been known to recur every night for many months in succession; on the other hand, they may recur only at long intervals. In most cases the sufferer is entirely unaware of their occurrence, except in those cases in which sleep-walking also occurs, where the patient awakes and finds that something unusual has taken place.

I have known cases in which the attack has been associated with a dream, but these are more likely to be cases of epilepsy.

Sleep-talking and sleep-walking either replace or may be associated with nocturnal screaming. Adult patients subject to these and similar sleep symptoms sometimes present the stigmata, both structural and psychical, which are characteristic of epilepsy. In all there is a well-marked neuropathic heredity. It is well known, however, that attacks in which a patient may wake suddenly from sleep in a state of nervous fear, apprehension, or panic are of temporary occurrence, and indicate a state of nervous stress or strain, induced by overwork or anxiety, and

not infrequently excited by some gastric or intestinal disorder.

I wish, however, to point out that sleep symptoms are found from time to time in persons of highly nervous temperament, which should be regarded as symptoms of importance and should be classed as phenomena upon the borderline of epilepsy.

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Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

RUPTURE OF BOTH QUADRICEPS EXTENSOR CRURIS TENDONS.

THE two cases of rupture of both quadriceps extensors reported by Drs. Chichester and J. Wallace Milne in the *BRITISH MEDICAL JOURNAL* of November 6th, 1909, p. 1343, and January 22nd, 1910, p. 199, have interested me very much and brought to my mind a similar case which I had in August, 1904.

J. H., aged 60, a coal salesman, slipped while carrying a bag of coals from his lorry to the cellar. He fell forwards, losing all power in his right leg. He tried to get up by putting his left beneath him; this also gave way, so that both legs were completely powerless beneath the knee. He was carried home, and I found both quadriceps tendons ruptured, leaving large gaps above the patellae.

Horseshoe-shaped strips of plaster were applied to bring down the ruptured ends of the tendons, and long splints applied to both legs. The legs were kept rigid for six weeks, when splints and plaster were removed and passive movement and massage applied for four weeks more.

After removing the splints large pads were placed behind the knees for several hours daily, leaving the legs and feet without support, so that their weight assisted considerably in producing flexion of the knee-joints, and in a short time he was able to take walking exercise.

He made an excellent recovery and resumed work twenty-four weeks from the time of the accident. He does not follow his former occupation, but is employed at the present time as coal shedman, and his legs never give him any trouble whatever, although he experiences many heavy lifts in the performance of his duties.

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PARACENTESIS OF THE PLEURA.

I HAVE read with interest the abstract of the paper by H. Hochhaus on "Puncture of the Pleura," which appeared in the *EPITOME OF THE BRITISH MEDICAL JOURNAL* for January 29th, but so far as the details are given, I am inclined to think that they are less convenient than the method which I first used while resident medical officer at Brompton Hospital, and have been in the habit of employing for the last twenty-eight years.

I described and demonstrated this method before the Medical Society of London (*Proceedings*, vi, 304), but the account published at the time was incomplete, and from the paper now under notice I am led to think that a short description may be useful. Given a sterile trocar and surgically clean skins of surgeon and patient, the apparatus prevents any possible infection from without, or any unintentional introduction of air or liquid. It is also in several respects easier to handle than that referred to by H. Hochhaus or any of those I have seen used or described.

For the indispensable purpose of removing any obstruction in the cannula with the minimum of interference to the operation, I selected a trocar designed and made by Matthews which certainly seems to me much more convenient than that described by Hochhaus. This trocar or