

A CRITICISM ON THE PATHOLOGY OF EPILEPSY.

In the whole realm of practical and theoretical medicine there is no subject more interesting than a study of the so-called functional diseases of the nervous system. These conditions Hysteria, melancholia, epilepsy etc. are probably related to one another in one essential - they arise through a morbid activity of the nerve cells due to a congenital or acquired defect in the cell protoplasm. There is no sharp line of demarcation between health and disease and this is peculiarly exemplified in nervous derangements. The onset of disease in all non-traumatic cases is gradual and in its first stages is merely functional, that is, there is aberration of function due to no discoverable structural alteration. The progress of disease is so insidious and the deviation from perfect health so slight at first that the condition is not recognised. But it is at this time, when the patient is on the borderland between health and disease, when he has indefinite and erratic symptoms of ill health which are too often attributed to fancy, alternating with periods of normal health that treatment is most likely to be successful. If these

initial phases and early undeveloped manifestations are unrecognised there comes a time though it may be years in arriving at a state of maturation when the condition is revealed and we are able to diagnose the disease but it is then often outside the domain of practical therapeutics. The investigation of neural pathology is beset with many difficulties. The physiological and the pathological are so intimately associated we cannot form any conception of the latter unless we have a precise knowledge of those principles involved in the building up and maintenance of the organism. It is this precision which is wanted. The microscope has opened up a hitherto undiscovered region, it has rendered possible the actual demonstration and depiction of the minute structure of the tissues. We know through its revelation that there are various kinds of nerve cells, with their two essential constituents - the nucleus and the protoplasm. The nucleus has two activities - a nutritive and formative - the protoplasm one which is functional. The nucleus is the fundamental unit of the organic world and it is engaged in the processes of self preservation, the nutrition of the cell and other parts more distant and the formation of new tissue. This has been ascertained by the actual observance of the leading part

? taken by the nucleus in cellular division - when it goes through the striking metamorphosis of Karyomytosis. And further Prof. Weigert demonstrated the importance of the nucleus in relation to nutrition by showing that in necrosis of tissue the nuclei disappear first, and they are no longer rendered visible and prominent by staining. The nuclei of the various kinds of cells are remarkably similar, whereas the protoplasm which subserves a particular function, shows great variety in structure and form. The protoplasm is thus a distinguishing feature and differentiates not only tissue from tissue but the instrument of one function from the instrument of another. We are able to see these comparatively grosser characteristics in a cell engaged in the generation of a motor impulse, in the transformation of sugar into Glycogen, in the creation of a digestive fluid, or as in the case of leucocytes, in the absorption and digestion of matter which is deleterious to the life of the individual. But beyond this we cannot go, and we have no means to ascertain those minute and hidden chemical combinations whose activity determines the performance of such diverse functions. We may regard the protoplasm as a complex arrangement of atoms, submolecules, + molecules, (groups of atoms and groups of submolecules) - and that energy is

evolved by the liberation of atoms from molecules. We know that nerve cells in a healthy condition can retain impressions, and reproduce them at some subsequent period that they elaborate and construct new forms by a process of analysis and comparison of previous receptions, that by their automatic activity cardiac and respiratory centres they maintain those organic conditions which are essential to life. These marvellous and multiple functions of nerve cells are known to us but we cannot understand them nor does it seem credible that the mind will ever be able to appreciate the processes of its own activity, if these were revealed a solution would be found to the deepest problem of life. And yet this knowledge of the composition of the protoplasm and nucleus, their exact relationship to each other - the manner of their sustenance and the causes which operate in the evolution of their energy is of paramount importance in the pathology of epilepsy. All pathological processes depend upon some defect or excess in the nutrition, formation or function in the cellular elements. A slight alteration in either of these activities will lead to functional disturbance although the alteration is unrecognisable in the present state of our knowledge. Besides the nerve cells we have to consider - the nerve fibres - the conducting elements of the cells. These

are almost as complex as the cells which give them origin. It has been observed that the axis cylinder of a nerve consists of a number of fibrillae - each separated from the other by interstitial tissue but beyond this we can only surmise. It can hardly be doubted though that each fibril is a separate conducting path - that it can act in conjunction or independently of the others and that it has a distinct termination, both at the periphery and the centre. There still remains the unsolved chemical problems:

1. What is nerve force?
2. " " nerve conduction?

And yet it is these chemical processes which subserve function and it is in them that we have the beginnings of disease. The pathology of epilepsy therefore resolves itself into two divisions:

- (1) The grosser pathology which includes -
 - (a) The seat of the initial disturbance.
 - (b) " path of secondary involvement.
- (2) The minute pathology which includes a consideration of -
 - (a) The nature of the lesion in nerve cells.
 1. Is it due to defective formation?
 2. " " " " " nutrition?
 3. " " " " an excess in function?

- (b) The nature of the protoplasmic explosion.
- (c) The nature of the conduction of the
nerve storm.

All pathological processes are due to massive or molecular disease of the tissue. Epilepsy and other paroxysmal neuroses are molecular diseases, and if we would resort to rational treatment we must remember that chemical energy is the source of nerve force and thus that perverted chemical energy is the foundation of functional derangement. There is a time when we can promote and facilitate a re-establishment of the normal chemical processes, but on the other hand their insistence leads to a degradation of the processes of nutrition and finally to molecular decay and permanent degeneration. These considerations seem to indicate the rational basis for the exhibition of drugs, and more recently of organic compounds derived from particular tissues or obtained by the chemical activity of germs. The more modern methods are pregnant with brilliant possibilities and although the results have not as yet realised the anticipations of the originators, they show that the investigations are in the right direction and that it is recognised that the processes which lead to organic disintegration cannot be disassociated from those which are required for its reparation. They have for their ultimate goal the prevention and restoration of intractable disease.

P A T H O L O G Y.

The various definitions of epilepsy are sufficient to show us that we must expect considerable antagonism in the theories regarding the Pathology. We have to consider three main things.

1. Where is the seat of the disease?
2. What is the lesion?
3. What are the causes influencing and determining the occurrence of a fit.

I. Dr. Wilkes declares that epilepsy is a disease of the whole brain.

Taylor says it is probable that the seat of the disturbance which brings about a fit, is in the Cortex of the brain.

Van der Kalk advanced arguments showing that the initial disturbance was in the Medulla.

Fagge believed the seat of epilepsy to be in the Basal ganglia and that the arguments favouring a cortical origin are more easily explained on the view that cortical irritation spreads to lower ganglia and there starts the epileptic phenomena.

Robert says: One view as to the nature of functional epilepsy is that it depends upon some nutritive change

in the medulla - upper part of cord and vaso-motor changes which leads to excessive and perverted action in these parts including sudden contraction of the vessels of brain and cord as well as of those supplying the muscles of face, pharynx, larynx, respiratory apparatus and limbs to which all subsequent phenomena of fit may be traced.

Another view is that a sudden discharge of nerve force takes place from an immense number of nerve cells at the beginning of a fit which leads to shock and the convulsions like other forms of this disorder are the result of a discharging lesion.

Gowers think it to be the result of diminished resistance on part of gray matter rather than of increased production of energy.

Ferrier's theory is that in the cortex of the brain there are individual centres for each separate muscular action involved in the epileptic convulsion, that they are related to each other in a constant and definite order and that the attack is due to the discharge of these centres in a tolerably uniform order. Much, he says, depends on the primary source of the irritation but adds that from whatever part of the hemisphere this proceeds whether from motor centres or from the sensory areas towards the back of the brain the order most com-

monly observed is that the centres discharge from before backwards beginning with those of the head and eyes and ending with those of the leg. He says that epileptic convulsion can be produced with as great readiness by application of the irritation to the sensory areas as to the motor centres themselves: also that it is doubtful whether consciousness becomes lost when the motor centres of the brain are alone complicated.

It seems probable that a typical epileptic fit is due to a diseased condition of the Cortex, with secondary involvement of other parts of the brain - chiefly the Basal Ganglia.

In arriving at this conclusion we have to guide us -

- (1) The Causes which operate in the production of the neurosis.
- (2) The sensations and symptoms experienced by the patient.
- (3) An analysis and comparison of the pathology of other forms of convulsions such as Focal epilepsy, reflex epilepsy with that of the true or idiopathic disease.
- (4) Experiments made on the brain of living animals.

(a) That the seat of origin of epilepsy is mainly a cortical one is strengthened by some known facts concerning the causation of the disease.

(1) The Hereditary transmission of nervous disease is a powerful factor in the production of idiopathic

epilepsy and if there is a congenital weakness of the nervous system it would naturally partake of a general instability of nervous structures especially of the more highly specialised. It is not necessary that the parents of an epileptic should themselves suffer from the disease in order to transmit it. Dr. Reynolds says that 12% of his cases were so transmitted while 30% had a history of some nervous disorder such as insanity, hypochondriasis, hysteria, marked neurotic weakness - nervousness etc. having occurred in at least one near relative. There is thus a close relationship between the various so-called functional diseases of the brain, insanity in the parent may be productive of epilepsy in the offspring or epilepsy in the parent of insanity in the children and further epilepsy may and often does lead to a more serious derangement in the same individual. The lower parts of the brain - the medulla, pons, and Basal ganglia are chiefly concerned in the movements and the maintenance of the organic functions they are more automatic in their innervation and are not so highly specialised as the Cerebrum. At birth the Cerebrum has only a low degree of evolution but it rapidly acquires further elaboration. Puberty is recognised on all hands as a period most fruitful in neurosis of every kind; then it is that epilepsy and other functional disturbances are most likely to be

made manifest. These facts seem to direct us to regard the Cortex as a more powerful factor in originating the disease than the less elaborated structures on which it is superposed.

2. Mental disturbance. When the disease is acquired and I am now referring to idiopathic or essential epilepsy the mental state of the patient preceding its onset will be found to be the predisposing cause. All mental disturbance such as worry, anxiety, prolonged grief is not only injurious but will cause in some people epilepsy, even though the family history of the patient is free from neurotic tendencies. How these psychical states influence the onset of a more serious derangement is not known. The process is insidious - we do not know in what direction it will lead. At first a patient has fits of depression, alternating with periods of comparative health. Under favourable circumstances the depression may gradually disappear - or it may lead to a settled hypochondriasis and this in turn to attacks of petit mal and finally Insanity. Each is a distinctly diseased condition but so slight is the deviation from health at the beginning that we cannot discriminate between the normal and the abnormal. The part of the brain which is concerned in the evolution of ideas, and performs work of a purely intellectual nature - or which is the seat of the emotions has not been ascertained although the part in front of and behind the motors

areas and by some the Cerebellum is credited with these functions. It is impossible to dissociate one part of the cortex from another for the intercommunication between the various cortical regions and between the Cerebrum and the Cerebellum is so extensive and complex that an abnormal functional activity in one part even if it were originally confined to a particular area would soon implicate large areas. The disease therefore which manifests itself in depression of spirits - melancholia, which may eventually conduct to epilepsy or insanity must be located in the cerebral cortex. Besides these disturbances which insidiously progress and continue over a long period before producing an epileptic seizure, we have another condition - an emotional one - to consider viz: Fright. Fright has undoubtedly been the means of inducing most serious nervous derangements. It acts with great rapidity and the patient who has been subjected to some terrifying experience may immediately or within a short time have a characteristic fit which is the precursor of life-long attacks. Why it is that in one case epilepsy results, in another chorea, or another insanity admits of no explanation but they are rare consequences and if they do occur children only as a rule are affected. In them as before mentioned the highest centres are in a

transitional state. The inhibitory power has not acquired its constraining and controlling influence over the lower centres and probably the severe shock paralyses its further development so that it never acquires that ascendancy which is essential to harmonious and rational nervous activity.

Sensations and Symptoms, as evidences of cortical origin.

(1) Dreamy mental states. Some patients who are subject to attacks of minor epilepsy complain of experiencing peculiar mental conditions. These conditions of mind have been felt and commented upon by many who have never had any other neurotic affection. They are not essential to epilepsy and not only does epilepsy occur without them - but they also occur without other disturbance. Still there is a connection, and these dreamy mental states may have a similar relationship to an attack of petit mal (an attack which involves complete unconsciousness without convulsions) that a simple spasm does to a fully developed convulsive seizure. Patients have great difficulty in expressing exactly the nature of these states. They say that suddenly an idea comes to them which seems to paralyse further volition - they lose themselves, and are for the time being completely dominated by the one idea. It may be a reminiscence, a repro-

duction of a former state, or it may consist of a loss of identity. They say I have felt or seen something exactly like this before but cannot remember when, or they suddenly cease to recognise their relationship to surrounding objects. Sir James Crichton Browne regards them "as revivals of hereditarily transmitted or acquired states in new and special combinations. . . . They involve disorder of the mind dependent on a defect of consciousness in one direction, indicated by vagueness as to present surroundings and an increase of consciousness in another direction indicated by the too vivid revival of former surroundings. - There is a negative element in the loss of control of the highest centres - a positive element in the raised activity of other nervous arrangements." He further says "They consist in an exaltation of subject consciousness and a degradation of the power of attention for the time being."

We notice of dreamy mental states that:

- (1) They are Hereditary and are transmitted to the children by patients who had such mental taints.
- (2) They occur before the Cerebrum is fully evolved especially when cerebral and mental development is rapidly progressing. They may vanish with the attainment of maturity.

- (3) They seize the patient when his attention is not actively engaged. When he is alone or during the invasion of sleep, that is to say at those times when the brain is freed from external solicitations, and volitional control or is in a transitional state.
- (4) As a rule they are harmless and disappear but those which are associated with fear, emotion, visceral sensations, or especially spasm often develop into epilepsy.

These peculiar psychical phenomena seem then indicative of a transitory and almost momentary functional aberration an aberration which appears to consist in a loss of inhibitory power of the highest centres and a consequent excessive, involuntary functional activity of others.

The Epileptic Aura affects about half the patients and as it is the ~~the~~ first notification they have of an impending fit it is of great importance as indicating the seat of origin of the disease. If we were able to positively localise the part where this initial disturbance arises the pathology of the disease would be materially simplified. The auras, however have such diverse characteristics, and appear in so many different ways it is impossible that they originate in the same particular

area. There is no uniformity in their appearance neither can there be uniformity in the seat of their formation. They have been classified by some as sensory, motor, vasomotor and psychical.

- Sensory (Tingling or numbness in the arm, leg, face or
(tongue.
(
(
- Motor. (Twitching or spasm in the arm, leg, face or
(tongue, co-ordinated movements, running,
(jumping. Patient may turn round.
- Vasomotor. (Flushes of heat - coldness, Palpitation of
(Heart.
(Sensation of choking, nausea, vertigo, epigastric
(pain.
(" " weight in a limb.
(Secretion of tears, saliva, or of sweat.
- Psychical. (Visual hallucinations, loss of vision, flashes
(of light, or colour.
(Aural hallucinations. Unpleasant smells or
(odours.
(An indefinite sense of fear or anxiety.

We will take the Psychical aura first. A Psychical Aura appears to be due to the involuntary, and automatic action of some portion of the brain which subserves consciousness. The influence of the Will upon these centres is abrogated for the time being. The difference between a dreamy mental state and a psychical aura is one of degree rather than character. The former is a fleeting, indefinable and intangible mental state - the latter is more pronounced, it is less under the control of the Will, and the patient has a distinct recollection at least in the

early stages of the disease of the idea or imaginative conception which was produced. Psychical auras are more often associated with minor epilepsy but in all cases the constant recurrence of attacks tends to quicken the rapidity of their onset and thus this premonitory symptom may afterwards disappear - it becomes unrecognisable through an almost instantaneous involvement of many areas.

Dr. Gregory mentions the case of a man who always had an aura which took the form of a visual hallucination he fancied he saw a little old woman, dressed in a red cloak, come towards him with a stick raised and then she struck him with it on his head and at this moment consciousness was lost.

In another case a patient came for advice as he felt convinced he was subject to epileptic attacks. He gave the following account. His family history was good and free from all neurotic tendencies. He was naturally bright, intelligent and took an interest in his work. In his 17th. year he commenced to abuse himself and continued the habit for 2 years and then he stopped entirely knowing full well an injurious effect had already been wrought, and his health undermined. He now suffered from emissions as often as twice or three times a week and occasionally was depressed, and experienced greater difficulty with his work. His memory was less

retentive. The depression gradually increased and sometimes amounted to melancholia. He was easily fatigued and slept less soundly. After suffering from emissions for 2 years he had his first fit. About this time the slightest exertion caused considerable fatigue. If he sat down after a short walk in the afternoon he felt very sleepy or dozed. The sleep would last a few minutes and the patient awoke and thought he had been asleep an hour. One afternoon he went to sleep in this way. On sleep supervening he began to dream. He describes being in a familiar field when suddenly he saw a light and what appeared to be a thunder-bolt coming towards him and with an explosive noise he was struck. For a momentary period there was oblivion and then consciousness partially returned. Patient said he felt as though his whole body was in commotion and so peculiar were his sensations he thought the roof of the house had fallen on him and he was struggling to extricate himself from the mass. He realised that something had occurred and yet felt powerless to do anything or to regain full consciousness. When consciousness returned completely he was convinced his muscles had been convulsed. From this time for the next 2 years no further attack occurred. During this 2 years he occasionally took bromide at night and nux vomica and Ac. Phos. Dil during the day. The depression of

spirits was much less marked and he passed the time pleasantly with occasional periods of melancholy. The 2nd. fit happened about two years after the first and under almost precisely similar circumstances. There was the fatigue and the sleep. He dreamt he was driving with some ladies when for no obvious reason the trap was suddenly over-turned. He appeared to be struggling violently and when fully awake or conscious he felt somewhat dazed and had a peculiar flushed sensation in the head. A week after this as he was talking he felt a peculiar sensation come over him. His face seemed to violently flush and burn. He seemed as if a veil were coming over his consciousness but by an effort and by directing his attention from dwelling even for a short time on one thing he warded off an attack. A few days later a similar condition occurred as he was in bed but before the invasion of sleep - in this case though he had distinct spasms and convulsive movements and became partially unconscious. - These last attacks have led him to seek advice. I have given this case in detail to show the nature of a psychical aura and its relationship to a fit.

(1) The writer once saw a medical man have a fit which was peculiar in some respects. The patient was writing down the morning's work when he got up from his desk, entered another room, and after a few minutes he

fell like a log in one corner. He was completely unconscious, but there were no convulsions, or spasms. He laid insensible for a minute or so - then got up and returned to his desk and writing. While in the inner room patient had dropped a large bottle of some preparation. He sat writing for a short time, then got up and again entered the inner room to discover the broken bottle. He asked how it got broken and possibly thinking he had had an attack, refrained from further enquiry.

We notice in Dr. Gregory's case -

(1) That the same spectral illusion initiated each attack and so we may conclude that the same particular area of the brain was the primary seat of the disturbance. Spectral illusions have their origin in Cerebral changes, since they represent creations of the mind and are not reproductions of past sensorial states but there is a construction of a new form which if it had occurred consciously we should call imagination.

Where then is the seat which originates the spectral illusions. Is it in the gray matter of the pulvinar, exterior geniculate body and anterior corpus quadrigeminum or it is on the occipital cortex.

We know that external visual impressions only reach the cerebrum through the medium of the pulvinar, and ~~corp~~ quadrigeminum and exterior geniculate body. Is it possi-

ble therefore that these bodies can through their own activity and without any appropriate retinal stimulus lead to impulses which when they reach the occipital cortex result in a fully developed visual sensation and in this way indirectly create a spectral illusion. It does not seem feasible that this should be the case but rather that the illusion is brought about chiefly through the activity of those cells which are concerned in the reception of the impulses from the lower ganglia and which link visual sensations on to psychical events. Foster says "Between the visual impulse as it travels along the optic nerve or tract and its ultimate psychical effect, a whole series of events intervene and the chain may be broken or split at any of its links at the later as well as at the earlier ones. It is possible that the break or damage may occur at the links by which the fully developed visual sensation join on to psychical operations - that the object is seen and yet does not affect the mind at all or affects it in an abnormal way." In the case of the patient seeing a woman approaching him, the mind was active, he was conscious of this one impression only and yet the will was in abeyance - there was as in Dreamy mental conditions an increased and exaggerated activity of some portion of the cortical areas resulting in a visual hallucination - with an abrogation or diminished

activity of others resulting in loss of volitional control.

2. We notice in both Dr. Gregory's case and in the patient whose fit occurred during sleep that the psychical events lead up to a condition associated with Fright. The one is struck on the head with a stick - the other is hit with a thunderbolt or else thrown out of a carriage, and it is at this moment that unconsciousness supervenes even if there is no fright there is at least the idea of an explosion. The centres which are associated with fright must be in the lower ganglia. Fright is an instinctive endowment and affects the nervous organisation of the lower forms of life. What happens then is that the unconsciousness is due either -

(1) to an extended discharge from many cortical areas or (2) to a discharge from the lower centres causing a sensation of fright which secondarily affects the higher centres.

If this be so we have in one case a trivial disorder, or exaggeration of function of a small area of the brain; it increases and suddenly implicates a large area giving rise to an explosive sensation and simultaneously unconsciousness follows. In the other a similar derangement leads to a sudden discharge from the Basal ganglia giving rise to an instinctive emotional condition of fear which

secondarily affects the higher centres.

Supposing that the derangement originates in the sensorium it must necessarily follow that a violent, incoherent and convulsive discharge of nerve energy in these gangliomic masses will produce unconsciousness even if the discharge does not secondarily produce a similar commotion in the cerebral cells because the sensorium would cease to act in its normal capacity as mediator between the mind and the external world. The cells engaged in mental processes, in the analysis and reception of impulses received from the basal ganglia make us conscious and consciousness depends for its sustenance upon these external stimuli, if they cease to be transmitted there is also abrogation of the function of these cells which subserve consciousness. Thus whether the cortical cells, subserving consciousness, are implicated in the explosive discharge or not, provided the lower gangliomic masses are affected - there will still be unconsciousness.

Aurae Motor and Sensory auras. These auras are so closely connected they may be considered together - for it often happens that the seizure is at times ushered in by peculiar sensations in the part movement of which inaugurate the march of convulsive movements. A sensory aura consists mostly in a sensation of tingling, creeping

or numbness in some part of the body, and if it affects a limb the sensation may pass upwards but it does not follow the course of any particular nerve. The peculiar character of the sensation, its non-association with the course of a nerve and the initial motor disturbance commencing in the same part of the limb shows that the disturbance must be central, and probably in the motor cortical areas. Fagge says "there is every reason to believe the aura is itself part of the attack and due to a change in some region of the brain which has close connections with the roots of certain cutaneous nerves: the disturbance is accordingly referred to the peripheral distribution of these nerves. Foster says "The cortical area has close if not direct connection of a sensory nature with the part in whose movements it is concerned. The development of the processes in the cortex leading to the issue of what we have agreed to call efferent impulses along the pyramidal fibres is markedly affected by sensory impulse, especially by sensory impulses started in the skin over-lying and corresponding to the muscles put into movement." Some remarkable experiments have been made by Dr. Mott and Prof. Sherrington which show that section of all the sensory roots of the nerves distributed to a limb is followed by permanent motor paralysis in addition to the anaesthesia which might

naturally be expected to occur from which they conclude that the whole sensory path from periphery to cortex cerebri, is in action during voluntary movement. The assumption that such motor centres exist in the cortex cerebri has been combated by Dr. Bastian, who maintains that there are no motor but only sensory centres in this region: sensory impressions and the activity of sensory centres being the real guides for volitional action and the true motor centres only existing in the spinal cord. Further removal of a cortical area not only paralyses the part over which it presides but also in many cases the sensation is interfered with. There is certainly an intimate connection between sensation and motion and we may probably regard a sensory aura as indicative of cortical disturbance in the motor area itself or at least in an area which has most intimate association with it. This disturbance is the nucleus and from it arises a general implication of the other parts.

Besides motor auras of a simple character such as the spasm and involuntary twitching of one or more muscles, the movements may be very complex, the patient running about or turning round. To all appearances these movements would seem to be voluntary. Foster says - "We have to seek for the distinction between voluntary and involuntary movements not in the co-ordination of the

muscular and nervous components of the movement but in the nature of the process which starts the whole act."... "The more closely the movements are studied the more probable it appears that the localisation which obtains in the cortex is essentially a localisation corresponding not to parts of the body or to nerves, or to muscles but to movements. That it is the movement ^{and} ~~is~~ not ~~the~~ the part moved which is represented in the cortex is further shewn by the relative magnitudes of the several cortical areas when they are mapped out according to parts of the body" e.g. that for the thumb being very large in recognition of its many and delicate manipulations. Considering that the cortical motor areas, are then the areas for movements rather than the parts moved, can we not regard them as the areas for the memory of movement as Broca's convolution is that for the memory of word movements, speech, phonation. We notice that all complex movements have to be learnt and that afterwards when the act has been initiated by the Will - it can go on for a short time at least without volition. A piece of music which requires extremely delicate and varied movements of the fingers is played only after a laborious training. The pupil is able after a time to play it by heart, without volitional aid - it comes to his fingers automatically. The patient who has an aura which consists

in a complex movement must perform that movement through the automatic activity of the motor areas and this activity arises thus either -

- (1) The centres which are the birthplace of the Will, start the motor impulse, they then become deranged from an abnormal functional activity or there is entire suspension of their activity but the motor impulse which has been started continues automatically until the involvement of the Will centres is suddenly communicated to other parts and a typical epileptic attack occurs.

or else

- (2) The disturbance arises in the motor areas - and the cells are in such an unstable condition the Will centres are no longer able to control their activity, this disturbance spreads as in the former case and the fit ensues.

Vaso-motor aurae are associated with minor rather than with major epilepsy, they are rare concomitants.

We can derive some confirmation as to the cortical origin of epilepsy by studying the different varieties of symptoms which characterise the actual fit. There are two chief varieties of fit, the petit mal and the haut mal. The petit mal is a distinctly milder attack than

characteristics
relates
to the actual
attacks
evidences
cortical
origin

the petit mal and we may perhaps be justified in regarding it as an incomplete form of the latter as attacks of minor epilepsy may alternate with the severer manifestation. There is not a sharp line of demarcation between the two varieties so that the pathology of one is the pathology of the other in an extended form.

An attack of minor epilepsy

- (1) may consist in a mere momentary suspension of consciousness.
- (2) may consist in a mere momentary suspension of consciousness of longer duration so that patient falls but still has no motor disturbance.
- (3) in a momentary suspension of consciousness with muscular rigidity simulating catalepsy, or slight convulsive movements.

In the major attack there is

- (1) Sudden and complete unconsciousness.

Then a period of tonic contraction.

Succeeded by clonic convulsions and then gradual subsidence.

In the attack in which there is only suspension of consciousness it is probable the disturbance is confined to the cerebral cells which subserve that function. If the cerebellum were affected we should expect some interference with equilibrium or at least some movements of the eyeballs rather than the fixity of expression which is associated with the unconsciousness. The motor areas must

also be unaffected for the patient is able to maintain the same position he was in when the attack began and there are no twitchings of the muscles. If however, the unconsciousness is profound,-the patient falls - in this case the motor areas are not involved they merely cease to innervate from the cessation of the necessary stimuli. There remains the basal ganglia but if these ganglionic masses were involved in the primary disorder we should have some motor evidence, such as rigidity or spasm.

If there is some twitching of the muscles or rigidity we must regard the process as implicating the motor areas as well as those which subserve consciousness.

In the major attack there appears to be an almost simultaneous involvement of many structures so that it is difficult to localise the primary seat of its origination. If it were in the motor areas, or in the psychical centres the discharge is so violent the other parts appear to participate in it at one and the same time. And when the first attacks are preceded by a characteristic aura their constant repetition seems to lead to a more sudden and easier development so that they occur without the aura. The cerebral mechanism like all other parts of the organism grows to the manner in which it is habitually exercised and so it is with epileptic attacks. Such

attack accentuates the pathological pathway and it is this tendency to recurrence which is the essential feature of the disease.

Post-epileptic conditions. After an attack of petit mal - and sometimes following the major attack - the patient may be subject to peculiar mental conditions which show that the cerebrum must be considerably influenced by the disease. The patient may execute various automatic actions of which he is then and afterwards entirely unconscious. The actions are accompanied with mental disturbance perhaps delirium often great terror or passion.

(3) Analysis and comparison of the pathology of other forms of convulsions e.g. Focal epilepsy, reflex epilepsy etc.

Reflex Epilepsy.

(1) Epilepsy due to errors of refraction. Cases are recorded in which epilepsy has been cured after disorders of accommodation have been remedied so that although it is unlikely that such optical defects would in themselves be productive of epilepsy it is certain they may influence the frequency and severity of the fits.

B.M.J.
p.30.93. Dr. John Hern says "Although it would be obviously incorrect to state that because an epileptic had an error

of refraction it must be the cause of his epilepsy, yet it appeared probable that in some cases at any rate the eye strain necessary in hypermetropia and others to focus a clear image on the retina might be the starting point of the epileptic attack." He had shown (at a meeting of the Ophthalmological Society) the first case he had met with in which true epileptic seizures ceased on a correction of hypermetropia by glasses. The patient had no fit if he used his glasses for reading but if he neglected this an attack of true epilepsy occurred.

In this connection, Liveing writes: "The accommodation of the eyes for distinct and harmonious vision is a very nice piece of consensual mechanism.... it has been often observed that the attempt to accomplish a nicely adjusted act. . . has been followed in persons of nervous constitution by a serious disturbance of the sensorium."

Nasal epilepsy.

Med.)
Times)
Nov.)
1895.)

Sicthoff has lately reported two cases of reflex nasal epilepsy. Both cases were men 38 and 33 years of age. The first suffered for twenty years with epileptic attacks, the fits growing longer and harder each year. Rhinoscopy showed hypertrophy of inferior and middle turbinated bodies and of crest of cartilaginous septum. Application of 10 p.c. sol. of cocaine averted a threatened fit and the treatment of the nasal mucous membrane

with the galvano-cautery finally caused complete cessation of fits. The second case presented many similar points. Slight fits occurred for a long time getting worse year by year. Attacks were always accompanied by an olfactory aura. Right inferior and middle turbinate were hypertrophied and pressed against the septum causing entire occlusion of nostril. Treatment was entirely satisfactory.

Dr. Greville Macdonald says "There are on record indubitable cases of epilepsy which were cured by intra nasal treatment. I have had but 2 cases of epileptiform convulsions under treatment - one was certainly not true epilepsy and as certainly had his convulsions completely removed by curing nasal obstruction. The other a pure and simple epileptic case was not benefitted thereby. There are cases of real epilepsy beyond dispute: but there probably are cases in which no doubt intra nasal irritation may be the immediate cause of the seizures although the morbid condition of nervous centres is the real disease: given the latter any other peripheral irritation may be responsible for the immediate attack. Here phenomena may be reflex but depending on epileptic conditions.

Aural epilepsy. Mendoza (revue mens de Laryng No.8) tells how he was able to induce an epileptic fit by

inical
cture
throat
spital.
r 27.
1893.

pressing upon an aural polypus.

Other forms of irritation, such as teething, intestinal worms etc. may be productive of reflex convulsions, but the convulsions cease with the removal of the exciting cause. It is in infants and young children that such irritation leads to convulsions even though the nervous structures may be perfectly healthy but if they occur in adults although we must regard the irritation as the determining factor, the convulsions are distinctly traceable to diseased states of the highest nerve centres. The pathology of Reflex epilepsy is that of true epilepsy the only difference is that the discharge in the former is prompted by a distinct stimulus - while in the latter the discharge occurs without any apparent ^{excit} ~~excit~~ation.

*mental
physiology*

Carpenter however says that it appears probable that the sudden but temporary suspension of the functions of the brain may be due to spasmodic contraction of the vessels of the sensorium induced by the extension of reflex motor impulse to the Vaso motor nerves. The relationship between reflex convulsions in children and true epilepsy is important and it was the subject of an address by Dr. Gowers (Medical Times June 1894) entitled "Rickets and Epilepsy." He says that at birth the nerve structures which are lowest in function and in part in position - the motor elements and reflex centres - are

developed before the higher. They are consequently not under the control of the higher centres and their activity is manifested in restless, and aimless movements. The motor centre in the cortex is a centre relatively low and it is not under the influence of the higher until comparatively late. This explains the frequency of convulsions in children and any retardation of development such as is induced by rickets or unwholesome food, and non hygienic surroundings acts chiefly on the imperfectly developed higher centres. Hence excessive activity of lower centres in infancy is reproduced by the influence of rickets etc. This uncontrolled activity of lower centres leads to a morbid action even in these centres themselves in which development is complete and function established. When the normal developmental processes are restored the higher centres acquire control over the lower and the tendency to convulsions ceases. But the spontaneous over-action has left its residual effects. Hindrance to development gives rise to convulsions.

Inherited tendency disposes to a residual disposition to recurrence.

Residual disposition - - - recurrence of fits and especially in consequence of this residual effect to recurrence at some period in later childhood or youth.

He describes three forms of epilepsy traced to infantile convulsions - 1st. That arising from general convulsions.

"In defective development all parts are implicated so the convulsions are general. The persistent convulsions which may carry on the malady from infancy to adult life are of the same general character. The establishment of control of the higher centres when it does not arrest the process of discharge modifies its form in a way we cannot yet understand and attacks of minor character replace the convulsions to be succeeded at the time when epilepsy is most prone to develop by convulsive attacks of greater severity. The convulsions of rickets in slowness and tonic character of spasm are nearer minor than to major epilepsy."

2nd. This form differs from the above in three features:

- (a) Convulsion is one sided - at any rate when moderate in degree. If there is an aura it is in one limb or in face on one side. Spasm commences locally in hand or face. When both sides are convulsed one is affected before the other except in most violent attacks. It will be found on enquiry that the infantile convulsions which preceded the epilepsy were also confined to one side.

(b) First attack of infantile convulsions was of great severity. Often there was a series of convulsions one after the other for several hours. Sometimes this series was the only infantile attack.

Initial attacks were followed by others for a month or two which ceased to recur when a few years had passed.

Complete continuity of occurrence from infancy to adult life is less common in these than in cases first considered.

(c) First convulsion often occurs during some acute illness or soon after a fall or in a state of general physical prostration.

3rd form - cases of epilepsy originating in infancy.

The first symptom can be traced back not only to infancy but through infancy to the first two or three days of life. There are convulsions during the first two or three days of life or at least convulsive twitchings and other indications - such as difficulty of swallowing - of grave impairment of the brain. These cases occur in first born children whose birth was long and tedious. The symptoms are the result of damage to the cortex of brain commonly the effect of meningeal haemorrhage.

Focal Epilepsy. Attacks of convulsions, identical with those of essential epilepsy are caused by tumours, spiculae of bone, haemorrhagic spots, indentations of the skull and degenerations. These convulsions may be preceded by a sensory aura and in some cases the epileptic fits which are indistinguishable from true epilepsy are arrested for a time by good feeding and small doses of bromide and become much worse under starvation, prison fare and other adverse circumstances. The convulsions resulting from these diseased spots may be initiated by a first attack simulating idiopathic epilepsy but their onset is generally more gradual. They may commence with spasm of a few muscles, then involve the whole limb and finally end in general convulsions. The pathology of Focal like reflex epilepsy differs from true epilepsy in its excitation and origination rather than in the morbid processes involved in the production of a fit.

Dr. Alexander says in this connection - "Probably there are three conditions necessary to an epileptic fit.

- (1) A sufficiently mobile brain.
- (2) A weakness of inhibitory power.
- (3) A cause capable of liberating the energy stored up in the cells of the cortex.

Med.
Journal
v.25.
393.

That the exciting cause must be of a peculiar kind as proved not only by the fact that identical tumours, indentations of the skull and degenerations may and may not be accompanied by epilepsy - and in surgical operations and vivisection experiments, irritation of the cortex by the knife or needle produce no muscular movement but a very mild electrical current immediately causes the proper muscle to respond."..... "We have at the present time no idea of the actual cause of liberation of nerve energy in essential epilepsy. When we remove a cerebral tumour we feel we may not have removed the cause of the disease that the tumour was probably only a *caus-a caus-arum* and that causes brought into being by the tumour may exist when the primary cause is removed and the tumours or other lesion may only have a co-incidental relation to the fits and the operation may either have no effects upon them or may cure them in an unknown way that has no connection with the removal of the tumour."

Dr. Gowers says: "The unilateral character of the convulsions means unilateral instability of the motor structures of the brain. Commencement in one limb means local instability in a certain part of these motor structures. Such local change excludes a general cause which would act on the whole brain. The suddenness of

Med.
mes
cture
ne 1894.)

onset indicates a sudden development of the instability i.e. a sudden change in one spot and a sudden change means organic disease. The effects of the focus of disease on the adjacent tissue which is slightly changed is such as to induce a permanent alteration in nutrition and function.

If disease is in motor region and is more than minute there is loss of power-hemiplegia - the amount depending on extent of lesion. It may persist through life or it may be transient and pass away in a few months. When disease is only near motor region any initial loss of power may be too slight to be recognised. At present the opinion which seems to myself to deserve most weight is that there is a sudden occlusion of a small surface vein by a clot with the consequent intense congestion and haemorrhagic softening of this region of the cortex. In softened region nerve elements are destroyed: an indurated contracted area ultimately represents the disease... On the margin of the chief destruction in every form there is a region of slighter damage and it is no doubt from this that discharges proceed. In the structures in which nutrition is deranged by the previous process the energy is not retained in its latent form until the proper stimulus relieves it: it accumulates and escapes without any influence that

we can recognise as acting upon the structures to cause the release of the force. **W**herever it occurs, **W**herever it spreads, in every place the discharge leaves behind it a residual state disposing to its repetition and the spread of the discharge.

We notice in connection with Focal Epilepsy -

- (1) That it occurs after falls with injury to head and from organic disease of the membranes and cortex of brain when demonstratable lesions are found to exist.
- (2) That it occurs after injury to the head when no such lesions exist and nothing abnormal either in the bone, membranes and cortex can be discovered.
- (3) It may develop suddenly but more often appears after a longer or shorter time, during which the disease is passing through a period of incubation.
- (4) There is a gradual increase in the intensity of the fits and from at first consisting of a few muscular spasms they may involve the whole limb - then the whole of one side or become general.
- (5) Headache, depression of spirits, and attacks of petit mal are usually present.

(6) The fits which were at first dependent upon the injury and the irritation resulting from it, tend to persist after the removal of the cause and the longer it operates the more probable it is that the accidentally epileptic will become essentially epileptic.

(7) After Trephining the removal of some gross lesion is generally followed by a diminution or cessation of the fits but if nothing abnormal is found the operation may be beneficial but more generally the radical interference has no appreciable influence.

Dr. Alexander. Med. Journ. Nov. 25. 1893 in reference to the results of operation, says:

	fatal.	cured.	unchanged.	improved
Dr. Billings reports 72 cases of which - -	16	41	4	11
Dr. Briggs mentions 92 American operations -	14	63	2	13
- - quotes 130 cases) (Walsham & St. Barth.)) Hosp. reports.)	30	75	7	18
- - own statistics.	1	25	1	3

In the above cases the fatalities are far above what they would be at the present time but I am afraid the cures are also as far above the results of our operations now as are the deaths. I have no hesitation in saying that

twelve months after enquiry would have told a different tale.

The above characteristics of Focal Epilepsy are interesting because they show the intimate relationship between it and essential epilepsy and if we can arrive at any reasonable conclusions concerning its pathology - the same will hold good for the idiopathic disease.

We have to consider:-

- (1) Whether the irritation spreads from the focus of disease to other parts of the Cerebrum giving rise to an instability of these structures and the consequent involuntary discharge which produces the actual fit.
- (2) Whether the irritation spreads from the injured centre to the basal ganglia and lower centres and causes an explosive discharge from these parts which produces the fit.

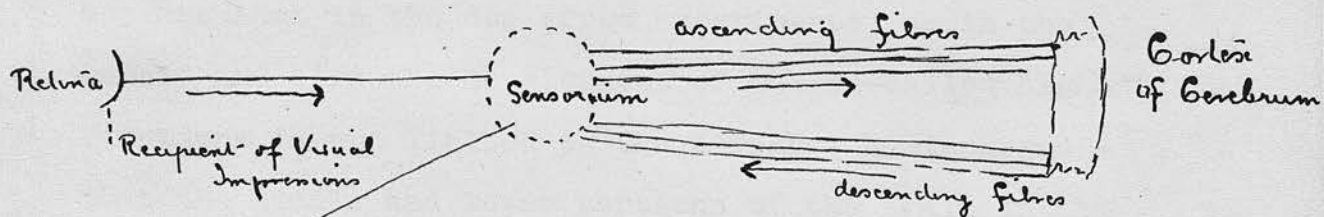
Before considering the location and nature of the processes which produce the epileptic fit it will be of advantage to review some of the physiological deductions arrived at concerning the functions of various parts of the brain.

Dr. Carpenter in his Mental Physiology tries to prove that the Basal Ganglia which he designates as the Sensorium

have a two-fold function: that they are the instruments whereby we become conscious of external sense impressions and thus feel sensations and also that they translate the cerebral modifications reflected down to them, and render us conscious of those modifications as states of ideation emotion etc. The substance of the Cerebrum is itself insensible and this seems to show that its presence is not essential to consciousness - and that the cerebrum is not cognisant of its own activity. The activity of the cerebrum is as automatic as that of other nerve centres and it derives all its stimulus from the impulses transmitted to it from the Sensorium. He argues that although the cerebrum is superposed upon the Sensory ganglia in the vertebrated series it still leaves the existing centres in the discharge of their original duties - so that the basal ganglia are the instruments of consciousness in man; "each pair of centres ministering to that peculiar kind of sensation for which its nerves and the organ they supply are set apart. We may also consider them as the instruments whereby sensations of whatever kind either original or direct instinctive movements" e.g. the start occasioned by sudden noises - closure of eyes on the approach of bodies - Sneezing caused by irritation of nostril.

The part of the brain below the cerebrum constitutes the fundamental ^{and} essential part of the nervous system - it

supplies the conditions requisite for the maintenance of his organic functions and it ministers to the operations of the cerebrum itself. He illustrates this relationship by analysing the conditions following stimulation of retina by light. "We only become conscious of the luminous impression by which nerve force has been excited in the retina when its transmission has excited a change in the Sensorium - So it would seem probable that we only become conscious of further change excited in our Cerebrum by the sensorial stimulus transmitted along its ascending fibres when the reflexion of the Cerebral modification along its descending fibres has brought it to react on the Sensorium. - According to this we no more think and feel with our Cerebrum than we see with our eyes: the **Ego** becomes conscious through the same instrumentality of the retinal changes which are translated by the Sensorium into visual sensation and of the cerebral changes which it translates into Ideas and Emotion.



The one centre of Consciousness
for

1. Visual impressions on the Eye
2. Ideational and emotional modifications in the cerebrum.

He further says "there seems strong probability that there is not a direct continuity between even all or any of the nerve fibres distributed to the body and those of medulary substance of cerebrum. For whilst nerves of special sense have their own ganglionic centres it cannot be shown that the nerve of common sensation have any higher destination than Thalami Optici - So Motor fibres which pass forth from Brain, though commonly designated as cerebral, cannot be certainly said to have a higher origin than Corpora Striata. And the movements which are usually designated voluntary (or more correctly volitional) are only so as regards their original source - the stimulus which immediately calls the muscles into contraction being still supplied from automatic centres. In connection with the relationship of the cerebrum and the other parts of the nervous system Foster summarises the results of the experiments made by removing the hemispheres thus: "It appears that in the dog (from experiments) as in the rabbit and bird the development of the so-called higher functions is not limited to the cerebral hemispheres - that the middle and lower portions of the brain in the higher animals as compared with the lower do not increase in bulk merely as the instruments of the hemispheres but like the hemispheres acquire more and more complex func-

tions. In the higher animals including at least some mammals after the removal of Cerebral Hemispheres even though conscious volition and intelligence appear to be largely (if not entirely) lost the body is still capable of executing all the ordinary movements therefore the nervous machinery for the execution of these movements lies in some part of the brain other than the cerebral hemispheres that is in the structures forming the middle and hind brain.

Dr. Ferrier found that stimulation of either Corpora Striata produced an immediate and rigid pleurosthotonos, or bending of the body to one side was excited in opposite half of body, head being made to approximate the tail and the fore and hind limbs fixed and rigidly flexed.

Stimulation of Optic thalamus gave no motor results it may thus be concluded they have no direct connection with movement. The irritation did not call forth cries or other signs of pain this might be supposed equally conclusive against a sensory function - it must be remembered though that the animal was under influence of chloroform and also that the connection of irritated Thalamus with Cerebral centres of movement which express pain had been destroyed in order to expose ganglia.

Foster says: Seeing that the thalamus appears on the one hand to be connected with all or nearly all parts of

the cortex and on the other hand to serve as the front of the tegmental system we may suppose it plays an important part in sensation pertaining to the body generally as part of it the pulvinar certainly does with reference to the special sense of sight. The part it plays is not an exclusively sensory one since both experimental and morbid lesions of the thalamus are apt to produce disorders of movement as well as other efferent effects. It is a complex body having many ties and probably performing many duties.

Dr. Ferrier Stimulation of Corpora Quadrigemina (of anterior tubercles) immediately calls forth a violent opisthotonos or backward flexure of body. The jaws are always violently clenched and pupils dilated - these results do not militate against the idea of the connection of these centres with sense of vision - they show that they are also motor centres especially for extens^{ors}~~ions~~. Stim: of post tubercle occasioned noises of various kinds.

The functions of the basal ganglia and their precise relationship to the cerebral cortex is thus still enveloped in considerable obscurity. And it is because of this imperfect appreciation that the pathology of epilepsy is equally obscure.

~~Before it is possible to understand the abnormal we~~

Because the Cerebrum is itself insensible it does not follow that it is not the seat of consciousness and there is no evidence to make us assume as Dr. Carpenter does that the Basal Ganglia are concerned not only in the reception of our external impressions but that they also interpretate the downward reflexion of cerebral modifications of those impressions which are the mental pabulum to cerebral activity. If this were so the Basal ganglia instead of being the instruments of the Cerebrum - must be considered the primary and the Cerebrum secondary factors in the nervous economy. The Sensorial gangliomic masses are exceedingly small compared to the whole cortical grey matter and it seems incredible that they should be able to interpretate the results of activity in so large an area. Besides the grey matter of the cerebrum is not structurally connected with the Basal Ganglia to admit of such a functional relationship: for only a small part of the Cerebrum is directly connected with the ganglia the greater part of the gray matter having connections only by the commissural fibres both transverse and longitudinal with the opposite hemisphere, with other gray matter on the same side, and with the cortex of the cerebellum. While the Basal Ganglia can more safely be regarded as the seat of consciousness for sensations we must admit that the

Cerebrum alone renders us cognisant of the ideas which it formulates, that the elaboration of sensations and their analysis and in fact all mental processes whether initiated from impressions received from the Sensorium or the result of the automatic working of cerebrum as in dreaming, reverie etc. are not only due to the innervation of the cerebral cells but we become conscious of them through the same instrumentality.

Removal of cortical areas. Foster says: "The dog and monkey agree perfectly in so far that removal of a particular area leads as an immediate result to loss of corresponding movement but though in some instances recovery takes place in others paralysis is permanent - probably because movements are more skilled.... The evidence of clinical study tends to show that in man the loss of movement due to destruction by disease of an area is a permanent one though actual demonstration of this is wanting." Then again he says, Lesions of the pyramidal system, - of the internal capsule for instance - lead to the loss not only of skilled but of all voluntary movements: according to the character and position of the lesion this or that part of the body is wholly withdrawn from the influence of the Will. And it is possible to maintain the thesis that man has become so developed as to his nervous system and the motor cortex so accustomed to make use exclusively of the pyramidal system that the Will has lost the power still possessed by lower animals to gain access by some path other than the pyramidal one to the immediate nervous mechanism of movement.

Faradization of Cortical areas.

Foster says: After removal of cortex stim. of white matter underlying the area - produces the appropriate contraction but stronger stimulus is necessary and latent

period is appreciably shortened. While we may speak of a particular co-ordinate movement as being the normal outcome of an ordinary careful stimulus^{ation} of a particular area in a normal condition it is no less true that diffuse - unco-ordinated movements culminating in general epileptiform convulsions are the natural outcome of the stimulus^{ation} of any area in an abnormal condition."

Starling says: The direct excitability of gray matter is proved by the following: (1) There is greater lost time in the gray matter than in the underlying white matter - that is if we first stimulate gray matter and then shave this off and stimulate white matter below it is found that latent period is far greater in former than in latter case.

(2) Excessive stimulation produces an epileptic convulsion starting from the original excited muscles.

Convulsions consist of 2 stages - (a) Tonic in which all the muscles of body are in state of continued contraction. (b) Clonic which lasts longer than the tonic stage and consists of rapid jerking movements followed by - (c) Stage of exhaustion in which cortex is relatively inexcitable.

(3) If gray matter on both sides be removed stim: of white fibres of corona radiata does not produce a

typical epileptic fit.

Ferrier's experiment. Stim. excited partial or general convulsion. The severest fits following when electrodes were applied at greatest distance. In all cases says Dr. Ferrier (Op. Cit. p. 39) whether fits were partial or general the immediate antecedent was an excited hyperaemic condition of cortical matter.

He found (a) Hyperaemia of cortex is ^{used} ~~indirect~~.

(b) There was a distinct interval of time between application and 1st. convulsion.

(c) There was a distinct interval of time after withdrawal of stimulus before the convulsion.

(d) The frequent persistence after discontinuance of stimulation.

Dr. Bidwell and Prof. Sherrington report a case of Focal epilepsy in which the cortical areas involved were removed.

The case was that of a boy aet 15. with no reliable history of injury but mother thought he had a fall on his head some 2 years previous to the fits. The fit started with a tickling sensation on sole of right foot, then followed contraction of great toe - extension of ankle and flexion of knee - occasionally slight flexion

Med.
Journ.
Nov. 4.
1893.

of fingers of right hand. He fell down but there was no loss of consciousness.

Just before operation he had six to twelve fits daily and one of these developed into a true epileptic fit lasting 10 minutes with unconsciousness and involuntary escape of urine and faeces. In the first operation a small haemorrhagic focus was removed to the depth of $\frac{3}{4}$ inch - afterwards no fit for 3 days but then they recommenced. There was no loss of power on foot or leg.

A second operation was undertaken and Dr. Sherrington stimulated the cortex electrically. The area by stim: of which these movements of foot and knee were produced was freely excised after operation there was,

Absolutely no loss of power in right leg or foot.

No impairment of any movement.

- loss - sensation or fall of temperature.

- ankle clonus was developed, knee jerks were natural.

Slight attacks of clonic spasms reappeared 2 days after

They began without any apparent contraction of foot or leg.

Right arm was flexed and underwent series of clonic spasms

Chin was depressed upon right shoulder.

Right side of face was contorted - jaw open and angle of mouth drawn up.

Some slight lateral flexion of body.

No spasm of leg but during fit voluntary movements were possible.

No loss of consciousness.

At Present time 1893 - There is no wasting of right limb or foot.

No hyperaesthesia or loss of sensation.

Patient walks without a trace of a limp.

They emphasize the following:-

(1) That no true epileptic fits returned after the operation.

- After the removal of a considerable piece of motor area.
- (2) There was an absence of any alteration in voluntary power in the leg.
- (3) There was an absence of any alteration in sensation in the leg.
- (4) The shifting of the site of spasms and the consequent freedom of ankle and foot.
- (5) The absence of any sign of sensitiveness in the dura mater.
- (6) Primary movement in convulsions was dorso-flexion at ankle and in the slighter fits this was the only movement, and in severer fits this was the earliest movement.

(7) Patient stated that fit commenced with a tickling in the right sole and on one occasion tickling right sole induced a fit.

Dr. Hale White (Med. Jour. July 1893) reports a case of focal epilepsy in which he removed an oval area of cortex infiltrated with a sarcomatous growth. Piece removed was on the left side and extended from fissure of Rolando in front to just across lower extremity of interparietal sulcus behind.

After the operation:

Motor. (There was aphasia, and paralysis of face, tongue and
(leg after operation. These symptoms did not com-
(pletely pass off probably because other growths
(rapidly developed and patient died.

Sensory. (Sensibility to touch, pain, heat, cold and muscular
(sense were absolutely unimpaired before and after
(operation.

Munk showed that if a large area of the motor part of cortex is excised there is loss of sensation on the opposite side of body. Dr. Mott confirmed these results on monkeys. Prof. Schafer shows that when a small area of the lower part of the ascending frontal convolution was excised there was no discernible loss of sensation in the

paralysed part - but if area was large there was loss of sensation. It is often noticed in Jacksonian epilepsy that the fits are preceded by numbness in part in which they occur. The fits in this case were preceded by numbness but after excision of cortex she never had any numbness. This seems to suggest that motor area has sensory attributes.

The results of removal of cortical areas, their electrical stimulation^{ation} and the records of cases in which diseased areas have been extirpated help us considerably in the pathology of Focal epilepsy.

They seem to emphasize the fact that the seat of the disease is mainly in the Cortex Cerebri with secondary involvement of the lower centres and thus enable us to advance arguments in support of this theory.

- (1) Focal epilepsy - as previously stated - in most cases shows a gradual increase in the intensity of fits and it is often a long time before the irritation of the organic lesion gives rise to a typical epileptic fit. Dr. Fagge says when the irritation of the surface of the brain causes a fit the actual motor impulses start from the basal ganglia or from centres still lower down in the pons, the bulb or the cord. We know that in reflex action a slight irritation which is unable to produce movement does so after a more or less prolonged application - there occurs a summation of the stimulus until it is sufficient-

ly strong to liberate the nerve energy. Now if the epileptic fit were due to a discharge from the basal ganglia and lower parts should we not expect a similar summation of the irritation from the cortex. The impulses from the irritated centres - whether motor or not would culminate in the sensorium and lead to a violent, incoherent and convulsive discharge from these gangliomic masses. But such is not the case the irritation is there, and the spasms occur but it is a long time perhaps months before essential epilepsy develops. Then again a patient may receive a blow on the head and within a short time have a characteristic fit. He is operated upon but nothing abnormal is found and yet the fits do not recur. Can this have been due to a discharge from the Sensorium initiated by the cortical irritation? The rapid onset of convulsive attacks does not support Dr. Fagge's contention nor does the absence of a lesion but rather that the attacks are due to a cortical nerve storm or explosion which is propagated downwards the basal ganglia being merely the instruments of the cortex increasing or diminishing the initial storm of the cortical cells.

Then, again, if the injury is over the occipital lobes - or at the back of the head it is more probable that the disturbance would implicate other cerebral areas and the cortical cells of the cerebellum than the grey

matter surrounding the ventricles. . . . The anatomical relations support this, for the occipital lobe has these connections.

- (1) The fibres pass down by way of the corona radiata.
- (2) They divide at the corpus callosum into 2 main groups -
 - (a) One group traverses this commissure to reach opposite side.
 - (b) One group continues into the internal capsule of same side.
- (3) Of those which remain on the same side (the side we are considering)
 - (a) The majority end in grey matter of pulvinar, external geniculate body and anterior corpus quadrigeminum.
 - (b) Others descend into the crusta of crus cerebri.
- (4) Of the crossed fibres.
 - (a) The majority pass into white matter of opposite occipital lobe and no doubt end in the cells of its grey matter.
 - (b) Others turn forwards and descend in the internal capsule and ultimately reach the crusta of crus cerebri.
- (5) Some of fibres in both crustae probably end in grey matter of the substantia nigra - while others reach the pons.

(6) Some fibres also pass towards posterior commissure some of these gain opposite side - others remain uncrossed. Both sets apparently end in grey matter round aqueduct of Sylvius - thus supplying a connection between cortex and the nuclei of nerves to the eye muscles on both sides.

*ostry's
Physiology*

(7) Through the superior peduncle the grey matter of the corpus dentatum, red nucleus and legmentum are connected and also the cortex cerebri. It is a crossed tract. It used to be called the processus a cerebello ad cerebrum. It connects one nucleus dentatus (and thus the fibres of inferior peduncle ending in that body) and fibres proceeding from the superficial grey matter of one side of cerebellum with the red nucleus and other parts of legmentum of crossed side and thus with cortex of cerebrum.

(8) Lastly there is a crossed connection through the middle peduncle between the lateral hemisphere of cerebellum by way of the pons and pes with the cerebral cortex of extreme frontal, temporo-occipital and possibly scattered elements of the parietal regions. It is noteworthy that congenital deficiency or atrophy of the cerebrum on

one side is often associated with a similar deficiency in the crossed cerebellar lobes.

We thus see that the grey matter of occipital lobes is related -

- (1) With cerebral gray matter of same side and in all directions.
- (2) With cerebral gray matter of opposite side.
- (3) A crossed connection with lateral cerebellar lobes.
- (4) A crossed connection with gray matter of cerebellum through the red nucleus and corpus dentatum - as the fibres going to corpus dentatum from cord are efferent it is conceivable that it has an efferent function in its relation to cerebrum, and therefore not implicated in a convulsive discharge arising in Cerebrum.
- (5) With the gray matter round fissure of Sylvius - an efferent tract.
- (6) With the gray matter of posterior part of optic thalamus external geniculate body and anterior corpora quadrigemina an efferent tract and therefore not implicated in a convulsive discharge.

A discharge from an irritative lesion in the occipital or neighbouring lobes of the cerebrum must be diffused chiefly through the implication of many other cortical areas of the cerebrum and cerebellum before the lower ganglia are affected: for they are intimately connected with them. But the tracts to the basal ganglia and gray matter in pons are few and these are chiefly engaged in conveying visual impressions to the cortex - or sensory impressions possibly from the posterior tracts of cord through the intermediary of the corpus dentatum.

2. Electrical stimulation of motor areas if confined to a small area gives rise to the appropriate contraction only but if the electrodes are separated and applied some distance apart epileptiform convulsions occur and yet if the white matter only is stimulated we get contractions but no epileptic manifestation. If the lower ganglia were such essentials, in the production of the fit we should naturally expect stimulation of the fibres of white matter would result in the same disordered action as is produced when the cortex is stimulated.

3. Removal of the irritation in a case of Focal epilepsy is often followed by a cessation of the fits but in some cases it is not when the accidentally has become essentially epileptic. Now if the fits were due to the irritation

spreading to the basal ganglia they would cease after the cause was removed. Their continuation shows that the cortical cells have become so influenced by the morbid process - that a residual disposition remains and continues the derangement.

4. In both essential and focal epilepsy we notice a uniform spreading of the convulsions, corresponding to the positions of the motor areas - this would not occur if the propagation occurred in the lower ganglia.

Abnormal constituents in the blood and their relation to Epilepsy.

The toxic origin of epilepsy is supported by a consideration of the resemblance it bears to other paroxysmal neuroses (such as megrim, headache, hysteria etc.) and the association of attacks with alterations in the excretions.

Journ
of Nerv
and
Mental
Diseases
Aug. 1892.

Dr. Hester has investigated the excretion of uric acid and of the organic sulphur compounds of the urine in epileptics in relation to the fits. He found that the amount of uric acid excreted shortly before a seizure rarely deviated from the normal limits but generally the urine passed after a fit had a higher uric acid ratio than at other times. In grand mal the increase was to be regarded as a consequence of conditions that determined the fits or possibly as due to the fits.

In petit mal a constantly high uric acid eliminated was observed. In 21 out of 29 cases of grand mal there was excess of sulphur compounds in the intestinal excretions and this excess was more pronounced about the time of the seizures than in the intervals.

Reduction of putrefactive processes, and of uric acid excretion was associated with a favourable influence.

Dr. Haig holds that excess of uric acid in the urine indicates excess of urate in the blood, that this excess in the blood produces contraction of the arterioles and capillaries and thus causes headache - mental depression in) Uric Acid) or epilepsy. Urine excreted during an attack of epilepsy, Causation) of) Disease) megraini or hysteria is scanty and contains excess of page 132.) to 169.) uric acid. After the attack the excess passes off and copious excretion of urine takes place. The degree of contraction of the peripheral arterioles throughout the body is directly proportional to the amount of urate in the blood. He assumes the following sequence -

- (1) Uric Acidaemia.
- (2) General contraction of arterioles and capillaries, producing high arterial tension and consequent
- (3) Intracranial hyperaemia and stasis.

He cites a case of epilepsy permanently cured by vegetarian diet and periodical catharsis.

Dr. Campbell says: "Excess of uric acid in the blood produces a characteristic group of symptoms which depend

Headaches largely upon its influence on the Vaso-motor system. There and other is tendency to cold hands and feet or to general chilli- Morbid Cephalic ness. Dead hands and Raynaud's disease are assumed to sensations result from Vaso-motor influence of the uric acid in the page 145. blood as are epilepsy and megrim from its action on certain vaso-motor areas of the head and glycosuria from

constriction of all the systemic arteries save those of the liver which hence become hyperaemic." The excess of uric acid in the blood cannot be the cause of epilepsy independent of its effect on the arterioles which will be considered under modifications in the cerebral circulation.

- (1) Because if so we should expect epileptic attacks in gouty subjects.
- (2) The convulsions which are produced in uraemic poisoning and uric acid is then possibly one of the toxic agents - are very different to a typical epileptic fit.
- (3) We should further anticipate that the epileptic seizures would alternate with asthma, migraini or other spasmodic conditions which are influenced by uric acid but this is not so for migraine is only occasionally associated with attacks of petit mal.

(Dr. Wilks contended against the relationship between Headaches migraine and epilepsy. Dr. Campbell favours the connection & other but believes there is a certain antagonism between Morbid sensa- tion them so that the presence of one to a large extent confers tions. immunity from the other.)

Claus supports the opinion that migraine is due to auto-intoxication and that chlorosis is a strongly predisposing cause. Dr. Haig attributes it to uric acid.)

- (4) Uric Acid is due to defective metabolism and is probably itself a manifestation of disturbance in the nervous processes which subserve nutrition - it is therefore an effect rather than a cause of the disease.

The excess of uric acid in the blood during a fit may be accounted for when we consider the large amount of metabolism which then takes place combined with the imperfect oxygenation of the blood. But even if it is already in the blood it is more probable that though it may precipitate or determine an attack it has no connection with its creation.

In connection with the toxic origin of epilepsy Dr. Boyce seeing the effects of absinthe poisoning closely resemble an epileptic attack experimented with animals to arrive at the part played by the various divisions of the central nervous system in the production of a fit.

He arrived at these conclusions.

- (1) That fits may occur in the absence of the cerebellum
- (2) Removal of one lobe of cerebellum, coupled with that of the opposite cerebral hemisphere tends to produce unilaterality.
- (3) One hemisphere, with an intact cerebellum is capable of discharging impulses to both halves of the body.

The seat
of
origin
and
path
of con-
duction
of the
fits in
Absinthe
Epilepsy
B.M.J.
Nov.18.
93.

- (4) Removal of both hemispheres with intact cerebellum is followed by an arrest of the typical tonic and clonic fits.
- (5) That impulses may descend through other channels than the pyramidal tracts.
- (6) That absinthe has an action upon the whole of the central nervous system.

Modifications in the Cerebral circulation in relation to epilepsy.

Variations in the cephalic blood supply must have a considerable influence on the activity of the nervous centres. The nerve cells are surrounded by plasma the tension of which varies with that of the blood.

Dr. Marshall Hall attributes migraine and other nervous seizures to congestion of the head, accounting for the congestion by supposing a contraction of the muscles of either (1) the neck - whereby the large veins were compressed or (2) the glottis, producing dyspnoea and so cerebral congestion. In this way he thought to explain the production of the nerve storm by peripheral irritation in accordance with his doctrine of reflex action. Dr. Broadbent believes that vaso-motor changes may lead to very definite and complex results by affecting various cephalic areas.

Reference)
Headaches)
by)
Dr. Camp-)
bell.)

Dr. Carpenter says it appears probable from recent researches that the sudden but temporary suspension of the functions of the brain may be due to spasmodic contraction of the vessels of the sensorium induced by the extension of reflex motor impulses to the vaso-motor nerves.

Ref.
Mental
Physiology
113.

It seems tolerably certain that the vaso-motor centres are implicated in an epileptic fit for we notice that -

- (1) The patient may suddenly become pale when he has an attack of petit mal or he complains of feeling as though the blood were rushing to his head.
- (2) Pressure upon the ^{carotids} ~~cerebral~~ arteries may stop an impending attack or shorten the duration of the fit - this fact probably leads to ligature of one of the ^{vertebral} ~~vertebral~~ arteries in the treatment of the disease.
- (3) The inhalation of nitrite of amyl has an undoubted beneficial effect when the patient is in the status epilepticus.
- (4) S. Bogdanik (Vratch No. 32 1893 p. 897) exposed and excised the left middle ganglion of the sympathetic nerve in a case of idiopathic epilepsy. The operation was followed by decided improvement and after three weeks the fits ceased

altogether. He regards this procedure as a substitute for ligature of the *Vertebral* artery.

- (5) Stimulation of the cortex is followed by hyperaemia which persists after the stimulation. Some observers think this hyperaemic condition influences the convulsions.

Although sympathetic changes do occur in the circulation it is far more likely that they are effects rather than the cause of the nerve storm. The emotional centres are intimately associated with the vaso-motor centres and in Hysteria in which we have an almost uncontrolled activity of the former centres, there are various sympathetic manifestations but even in this disease it is the cerebral disturbance which determines the erratic action of the vaso-motor system. The improvement which occurs after the inhalation of nitrite of Amyl - or the prevention of an epileptic attack by pressure on the carotid arteries is no evidence that vaso-motor changes cause the nerve storm - it only shows that as in the case of the initiation - the prevention of the nerve storm may depend upon some slight change in tension or other seemingly inadequate cause.

Then again in other cases we find exactly opposite results - compression of the carotids causing a fit

B. Naunyn, Strassburg (Zeitschrift für Klin. Med., Bd XXVIII Hefte 3 & 4) reports three cases of senile epilepsy in each of which attacks similar to those observed in patients spontaneously could be induced by compression of the carotids. Each of these patients had in addition to the fits either hypertrophy or valvular disease of the heart and arterial degeneration. Naunyn carried out some trials on compression of the carotids in persons under thirty but the experiment led to no result; in two men over 50, unconsciousness, with slowing of pulse and slight general convulsions, set in. The origin of the albuminuria which often arises with an epileptic fit is very doubtful and many writers deny its connection with nervous changes in the medulla. It has been experimentally proved that puncture in the floor of the 4th. ventricle will produce albuminuria and many cases of functional albuminuria probably arise from some temporary derangement of this centre in the medulla. The association of this condition with epilepsy even if its nervous origin be admitted, is no proof that the neighbouring vaso-motor centre is implicated in the production - but rather that all the automatic centres in the medulla are more or less secondarily influenced by the sudden and violent discharge from large portions of the great brain. That vaso-motor changes are not

responsible for epilepsy we can advance these arguments.

- (1) Many patients exhibit no evidences of these changes - nor is the sympathetic affected with consequent dilatation of the pupil.
- (2) When a person faints the fainting fit is due to an anaemic condition of the brain produced by sudden constriction of the vessels. If an epileptic fit were due to a sudden spasm of the vessels of the sensorium should we not get a sudden passive insensibility instead of the violent discharge which does occur?
- (3) In all congested conditions of the brain - due to heart or lung disease we do not get epileptic fits but only asphyxial spasms.
- (4) Dr. Broadbent believes that definite and complex results occur if various cephalic areas are implicated. Now if this were so in epilepsy - and in essential epilepsy one fit is similar to another - we should have either a dilatation or spasms of the vessels supplying the same particular areas in each fit. But this is not feasible for if the vaso-motor centre were deranged we should have first one area and then another either dilated or constricted and the resultant fits would be equally dissimilar.

It seems improbable that a simple modification in the plasmic surroundings of the nerve cells whether it

be a sudden change of increased or diminished tension would be responsible for so specialised a result as an epileptic fit although it might produce an irregular, and disordered activity and so induce a set of symptoms which we should designate as convulsive.

The minute Pathology.

We have now to consider the lesion which is the cause of the epileptic fit. We know there must be some alteration in the constituents of nerve tissue to produce such violent derangement but our knowledge of these minute processes is enveloped in obscurity: reason alone can lead us, and it must be our substitute for the more precise methods of actual observation.

In cases of long-standing the bones of the skull and the cerebral meninges have been thickened. The Dura mater is often opaque and may contain bony neoplasms. The capillary blood vessels in the Medulla may be congested, especially the nuclei of the hypoglossal and vagus nerves and in some cases there is an induration of the substance of the medulla, causing fatty degeneration and softening. The cortex of cerebrum is occasionally sclerosed. Prof. Obersteiner says: "A very peculiar form of sclerosis is limited to the Cornu Ammonis in which this structure becomes as hard as cartilage and much shrivelled. This form is almost restricted to epileptics - it is present in more than half the number of cases (Pfleger) and may be uni- or bilateral." The medullated fibres may be

decreased in number. He further mentions that "Amyloid bodies are frequently to be seen in the ependyma of the ventricle especially on the mesial wall of the optic thalamus and in the inferior *horn* when the cornu ammonis is sclerosed. Their origin is doubtful - their substance is not related to starch but to albumen. They are always present where nerve substance is slowly disintegrating and may possibly represent the last phase in the series of chemical changes associated with atrophy of the nerve fibres." In essential epilepsy as a rule nothing abnormal is found and if any minute structural alterations are discovered they are the effects and not the cause of the disease. It is a paroxysmal disease and the patient has, especially in the early stages, long intervals of immunity from attacks. The lesion - therefore cannot be a permanent structural alteration but rather a chemical defect in the protoplasmic elements.

(a) The nature of the lesion in the nerve cells.

We do not know what this is but having regard to the causes which operate in the production of the neurosis it is probable that the three activities, formative, nutritive and functional of the cell are implicated. Heredity would produce defective formation and if the disease is acquired we should expect its acquisition to depend on

imperfect nutrition or function. All nervous activity is the result of stimuli and even the automatic centres are not independent of them. Every stimulus should have a two-fold function - it should be followed by the appropriate reaction and also should act as a stimulant to fresh nutrition. In the acquired disease it is probable the power of repair is defective - the imperfection resulting from shock - undue commotion in the nervous system or from conditions of the blood e.g. in alcoholism.

The
Dynamics
of life
in
relation
to
Epilepsy
Lancet
Nov.10.
94.

Dr. Gowers says: "the source of nerve force is latent chemical energy conceived as minute motion, liberated and released by added motion; nerve energy is therefore a form of molecular or atomic motion . . . In the structures concerned the minute motion that is the latent energy is more nearly equal to the restraining force than it is in health so that an amount of added motion slighter than is needed under normal conditions permits the liberation of atoms and energy. The motion may equal the restraint and then exceed it by mere accumulation - balance is disturbed and energy is liberated without any more added motion than the slow nutritional accretion - that is without a stimulus. This might result from a very minute general change in the composition of the nerve tissue concerned, perhaps even from an abnormal relation of the molecules in their constituent submolecules. This undue readiness of action -

instability is the result of abnormal chemical constitution. Every change in chemical composition entails a change in function and all persistent changes in function depend on a change in chemical constitution."

(b) The nature of the protoplasmic explosion.

We have no knowledge of what occurs when the patient has a nerve storm nor are we able in many cases to assign any adequate cause for the occurrence of a first attack and their subsequent continuance at longer or shorter intervals. Attacks are more frequent if the patient is worried, anxious or depressed. Insufficient and unwholesome food and bodily exhaustion tend also to increase the number whereas anything which improves the mental condition, inspires hope and promotes the patient's well-being is followed by their diminution.

The majority of first attacks occur when the patient is between 10 and 20 years of age and a considerable percentage before the age of 10 is reached. Dr. E.M.Sympson reports two cases of senile epilepsy which occurred at the age of 73. It is also noticed that few attacks take place when the patient is in a dangerous position e.g. riding or is engaged in something which requires close attention. In these circumstances the mind is actively employed in controlling and watching the lower centres.

In three cases which I attended at the time of the initia-

tion of the disease - the first attacks happened at night: One of these was a man - aged 46, and he did not recover consciousness for two days. After this condition - status epilepticus - he was delirious for some days and had to be removed but returned home two weeks later apparently in the best of health. Sometimes the first attack is excited by a fall which in a healthy person would have no effect. This happened in the case of a boy, aet 15. His family was neurotic and one of his parents suffered from epilepsy. He was playing one afternoon with some companions when he fell on a slide. On trying to rise he fell back unconscious and had a convulsive seizure. He was taken indoors where he slowly recovered consciousness. The scalp was bruised and slightly swollen and he appeared dazed and failed to remember what had happened. The injury itself was too trivial for this to have been a case of Focal epilepsy. The disease was no doubt inherited and the accidental fall was the determining cause of its manifestation. In another case a severe fright seems to have not only been the cause of a first attack but of the disease itself. The patient was nearly run over by a van when four years old and since that time (six years ago) he has suffered from epileptic attacks. The parents and other children are quite healthy.

In reference to the nerve discharge - Dr. Gowers says: The disordered action of nerve centres in epilepsy presents, more than any normal action - the opposite effect which the same influence may exert when in different degree. The process of discharge may be manifested by an arrest of action or by over-action according to its degree. The commencing process in visual centre for instance may cause sudden darkness and then as it increases bright stars may be seen. The same process may thus stop all influence on the higher centres of those impulses which should reach it from retina; as it increases it may cause liberation of energy to the higher centre with which conscious sensation is associated. Here we have the two apparently opposite effects as the result of the same process over the same centre. We may have a like effect in related centres. In "local epilepsy" a sensory aura may commence in the fingers, pass up the arm and down the side to the leg. As it proceeds down the arm this becomes almost powerless. The discharge in the structures which affect consciousness inhibits the related motor centres and arrests their activity. A similar energetic discharge may be accompanied by spasm. . . . We have reason to believe there must be inter-atomic motion, and that it is the liberation of the atoms that is the chief means

A Dynamical conception of inhibition and nerve discharge.
 Lancet Nov.10. 94.

of releasing the energy of motion they hold. Also there must be inter-molecular motion since molecules may escape. Complex molecules are conceived to be formed by the union of groups of atoms - submolecules (or radicals) and the compounds which result from muscular action give countenance to the belief that the submolecules may be separated and released. This involves the conception that the constituent atoms of molecules and of submolecules must be united by the attraction stronger than that which unites them to the atoms of other molecules otherwise the existence of molecules would be impossible. The groups of atoms must exist as such - they must be held together by attraction which keeps the atoms from joining those of other submolecules.

The groups are held together as such in the molecule therefore there must be energy keeping the groups separate. as well as an attraction " " atoms of each group together. and likewise " " " " submolecules together in the molecule. This energy we cannot separate from our conception of motion.

Inter-molecular motion is involved in the escape of molecules.
 Inter-atomic " " " " " " of atoms
 Intra-molecular (that between) " " " " sub-
 (sub-molecules.) (sub-molecules.) molecules.

If there is motion between the molecules and also between the submolecules an increase in either seat will tend to resist the escape of the atoms - will tend to restrain and prevent the liberation of the energy they hold. It is the release of atomic motion that is the chief source of manifested energy, an increase of that between the groups of atoms will hinder the release of energy and will prevent a stimulus having a normal effect. The added motion may conceivably pass chiefly between the groups of atoms, if it is excessive or under certain conditions - It would then have purely a restraining effect.

(c) The nature of the conduction of the nerve storm.

In many cases the epileptic attack appears to originate in a small and particular area of the brain and then to rapidly spread and involve many parts. After repeated attacks this pathological pathway is accentuated and the nervous tissue has grown to the manner of previous excitations so that the accession of the morbid activity is simplified. Some patients at first only have nocturnal attacks - when the inhibitory influence of the Will is to a great extent withdrawn from the lower centres later on however the attacks occur indiscriminately. Then again there may be a premonitory aura - or as in the case of focal epilepsy localised spasms - or a minor attack of

petit mal with partial unconsciousness, but in advanced cases they occur less frequently and we have instead the almost instantaneous implication of many areas and centres. Dr. Gowers says: "The function of the generating and conducting structures differs only in degree, the process of conduction is of the same nature as that of production and chemical action underlies both. . . those structures in which nerve energy arises have a slightly greater amount of material and that is all the conspicuous difference.... Instability spreads in the brain and such extension even with extreme rapidity is a normal phenomenon... Released energy excites the release of atoms and of nerve energy ~~in~~ on all connected and related structures, in those adjacent and remote. When action is thus multiplied the effect of even a minute difference in constitution of nerve tissue may obviously be very great.

Dynamics
of
Disease.
Lancet
Nov.10.
94.

C O N C L U S I O N .

Although the nature of the pathological processes involved in epilepsy admits of no definite exposition, yet we have sufficient analytical evidence to justify deductions which have more than an approximate value. In summarising these we have to remember that we cannot disassociate one part of the brain from another for the intercommunication between the various centres are so extensive and elaborate that distant parts, which are probably only secondarily affected, appear likewise to participate in the actual production of the initial nerve storm. We may then define epilepsy as a disease due to a morbid activity which primarily originates in the nerve cells of one or both hemispheres and rapidly spreads, implicating other and lower ganglionic centres. And in support of this the foregoing deductions may be briefly recapitulated.

- (1) The disease is transmitted to the child by parents who have suffered from a recognised disease of the hemispheres such as insanity, hypochondriasis.
- (2) It arises sometimes after prolonged mental disturbance e.g. worry, anxiety, or hypochondriasis; and it

occasionally is induced by a severe emotional disturbance such as Fright.

- (3) Excessive alcoholic indulgence may produce the disease, and although in these cases the epileptic attacks are not exactly a reproduction of idiopathic epilepsy the analogy warrants the conclusion that as in these cases the cerebrum is chiefly implicated, it also plays the principal part in the hereditary disease.
- (4) Dreamy mental states may be followed by attacks of petit mal and grand mal and as the former undoubtedly originates in the cerebrum it is more than probable that the more pronounced disease is but an exaggerated manifestation of the same derangement.
- (5) Many attacks are preceded by a characteristic aura which localises the initial disorder to some particular area of the hemisphere.
- (6) A comparison of the variations in the actual fits - from those which involve a temporary suspension of consciousness to the most pronounced attacks, seems to confirm their cortical origin.
- (7) In cases of focal epilepsy, removal of the damaged cortex, is sometimes followed by a cessation of the fits and further the fact that epileptic attacks do occur after injuries to the cerebrum, shows that the

disease is closely associated with altered cortical conditions.

- (8) After an attack the patient may have post epileptic delirium which shows the cerebrum is considerably influenced.
- (9) Stimulation of the motor cortex with the electrodes some distance apart may lead to epileptic convulsions and yet stimulation of the underlying white matter has no such effect.
- (10) The invasion of the convulsions follows the arrangement of the motor cortical areas.
- (11) Destructive lesions of the nucleus lenticulus or of the cauda may produce no special symptoms and in a case of tumour of the right Optic thalamus there was slight motor paralysis of opposite side chiefly of arm, and no marked anaesthesia; if back part of thalamus is implicated hemiopia may result (Fagge)
The clinical history of cases of disease of the basal ganglia seems therefore to support the fact that they are only indirectly concerned in an epileptic attack for if they were essentials in the initiation of the disease we should anticipate that convulsions similar to those of epilepsy would result from their involvement in organic changes.

(12) Cases have been recorded in which epileptic fits have ceased after the development of disease in the internal capsule (Taylor.)

(13) Dr. Boyle found that, in his experiments with Absinthe poisoning, the typical tonic and clonic spasms did not occur after removal of both hemispheres and that one hemisphere was able to transmit the convulsive activity to both sides of the body.