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Oct 98

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"EPILEPSY"; A theory as to its probable cause with some remarks regarding its relation to Insanity and Hysteria.

In the following pages I wish to consider the aetiology of Idiopathic Epilepsy with the probable morbid conditions which characterize it, and the possible relations existing between it and the allied diseases, Insanity and Hysteria. At the present day, our knowledge of the true causation and pathology of Epilepsy is very meagre and this for the definite reason that the disease cannot be referred to any lesion recognisable by the naked eye, or the microscope, but rather to an obscure departure from the normal condition of the interlacing fibrillae and association centres in the grey matter of the cerebral cortex. At the outset it may be stated, without fear of contradiction, that the region of primary disturbance in Idiopathic Epilepsy is the grey matter on the surface of the cerebral hemispheres. Facts in proof of this statement will be given later, and it is only mentioned here as a reason for the description[1] of the histological structure of the grey matter which follows, as thorough familiarity with the up-to-date knowledge of the structure and probable function of the cerebral grey matter is thus rendered indispensable to an accurate consideration of the aetiology of Epilepsy. The surface of the brain is divided into four great areas, named according to their position, the frontal, parietal, temporal, and occipital. Spread over the surface of these areas, and varying somewhat in thickness, is the grey matter composed of numerous cells with interlacing fibrillae, and supported by the ground tissue, neuroglia. According to distinguishing structural features, the grey matter has been divided into three layers parallel to the surface of the brain, namely, the deep layer, or layer of polymorphous cells, the mid layer, or layer of pyramidal cells, and lastly, the superficial, or molecular layer. Taking these layers in detail

from within, outwards, we consider first, the layer of polymorphous cells which lies in apposition to the white matter of the cerebrum. The distinctive feature of this layer is the presence of polymorphous cells, which are characterized by their variety of form. Upwards, these cells send processes or dentrites, which, however, do not penetrate sufficiently far to reach the molecular layer, but generally end in the substance of the mid layer. Downwards, descending processes or neuraxons pass from the polymorphous cells into the subjacent white matter, but the subsequent distribution of these fibres is at present unknown. In the upper portion of this layer are to be found one or two special cells known as Golgie's cells which are characterized by the possession of short non-medullated branching processes. Through the substance of the layer, and breaking up the grey matter, pass bands of vertical medullated fibres known as medullary rays. Superficial to the layer of polymorphous cells lies the mid layer, or layer of pyramidal cells, the characteristic feature of which is the presence of pear-shaped cells, which vary much in size. The large pyramidal cells are situated in the deeper portion of the layer, while the smaller ones are more superficial. In appearance, the large pyramidal cells are very characteristic, being pear-shaped, with their apices pointing towards the surface, and giving off many and varied processes. From the substance of the cell at its apex issues the apical process or dentrite, which, passing through the upper portion of the mid layer reaches the superficial or molecular layer. Here the dentrite divides and sub-divides, and interlacing with other dentrites, as well as with the terminal neuraxons of distant cells, forms a dense network of fibrillae, which, by Golgie's method can be easily demonstrated as forming the greater portion of the superficial layer of grey matter. Several processes issue from the base of the cell, one of which

termed the axon or neuraxon, starts from the centre of the base and courses into the white matter beneath. The final destination of the neuraxon varies. It may pass through the descending tract to the crossed pyramidal tract of the cord, and supply some muscular system, it may pass as a commissural fibre to the opposite hemisphere, or, finally, it may constitute one of the so-called association fibres by means of which, functional, as well as histological connection, is establish -ed between various regions of the same hemisphere. From the neuraxon collaterals generally arise, some of which wind back to the same cortical area, while others pass to distant regions of the same, or opposite side. From the sides and lower angles of the pyramidal cell emanate the processes known as the basal dentrites, which branch and quickly disappear in the neuroglia. Besides the cellular element in the mid-layer, there are present certain groups of fibres arranged in horizontal and vertical bands. Thus, in the deeper portion of the layer is placed Baillarger's Band, formed by numerous delicate medullated fibres, which pass in a horizontal direction, while another band of similar horizontal fibres, known as Gennari's Band, is located about the middle of the layer. The medullary rays issuing from the subjacent layer of polymorphous cells are in the deeper portion very distinct, but more superficially, they become much less conspicuous as they taper into the molecular stratum above. The characteristic feature of this molecular or superficial layer is the preponderance of the fibrillae constituting the ground substance over the cellular element; it is in reality a dense network of interlacing fibrillae, principally formed by the arborizations of apical dentrites and the neuraxons of Martinottie's and distanct cells.

The above description applies to the structure of the grey matter generally, but the number of the cells and the thickness of the individual layers differ in the various regions of the cortex. Thus, in the Motor or Rolandic area, the mid-layer is characterized by its great thickness, by the size and number of its pyramidal cells, and by the presence in its deep portion of ganglionic or giant cells. In the occipital region, on the other hand, the small pyramidal cells predominate, althoughin the deeper part of the layer a few cells resembling the large pyramidal cells in the motor area are to be recognised. In the frontal region the structure of the grey matter more closely follows the general description given above, its outstanding feature being the thickness of the layer of polymorphous cells.

From the pyramidal cells in the mid-layer of the grey matter, the neuraxons descend in the medullary rays and converge in the corona radiata to the internal capsule. The fibres from the frontal lobe occupy the anterior portion of the fore-limb of the internal capsule, after which they course through the pes to the pons, in the grey matter of which they seem to terminate. The motor area is located along the ascending frontal and parietal convolutions on each side of the fissure of Rolando, and, dipping over the margin of the hemisphere, at the longitudinal median fissure, extends as far as the gyrus fornicatus. The fibres from this area occupy in the internal capsule the posterior portion of fore limb, genu, and anterior portion of hind limb, some fibres also pass to the caudate and lenticular nuclei. From the internal capsule, the motor fibres can be traced through the middle of pes of crus, anterior portion of pons, to the decussation of the pyramids in the medulla, after which they constitute in the cord the crossed pyramidal tracts and the columns of Turck. But besides being the seat of origin of motor fibres, the grey matter

is also the recipient of the terminals of sensory fibres. The posterior root fibres, conveying sensory impulses to the cord, enter at Burdach's column, in which they run up for a short distance and then cross to the column of Goll. In the medulla, the columns of Goll and Burdach terminate in the nucleus gracilis and nucleus cuneatus respectively, while the direct cerebellar tract passes to the restiform body. Some fibres from the columns of Goll and Burdach also pass to the restiform bodies, which latter are continuous with the inferior peduncles of the cerebellum. From the nucleus gracilis fibres pass to nuclei in the vermis of the same and opposite side, while the nucleus cuneatus gives fibres to nuclei in the vermis of the same side only. From the cortex of the cerebellum and the nucleus dentatus, fibres cross the mesial line to the red nucleus of the opposite side; these fibres are said to degenerate in cerebellar lesions. The main sensory paths, however, are the fibres which pass upwards from the nucleus gracilis and nucleus cuneatus, and decussate behind and above the anterior pyramids, but ventrally to the central canal, after which they form the fillet. In the pons the fillet forms deep longitudinal fibres in its white matter, and passes upwards in the tegmentum of the crura external to the red nucleus. Here the fillet divides into two strands, the lateral, and the median fillet. The lateral fillet, which receives fibres from the cochlear nucleus, ends in the grey matter of the posterior Corpus Quadrigeminum and the white matter underlying the anterior Corpus Quadrigeminum. The median fillet, which receives fibres from the accessory Vago-Glosso-Pharyngeal nucleus and from cells of the ascending root of the fifth nerve, ends chiefly in the optic thalamus. "From the internal capsule, external capsule, and optic thalamus, the sensory fibres pass up as three great systems. Some enter the central convolution of the Rolandic

area, others reach the paracentral lobule, the inferior frontal convolution, the insula and small parts of the middle and superior frontal convolutions, whilst considerable numbers reach the gyrus fornicatus, and the hypocampal gyrus, which Ferrier[II] had previously localised as a centre of common or tactile sensibility." The final termination of these sensory fibres in the cortical grey matter is of interest in connection with the subject under consideration, and can be demonstrated by Golgie's method. Passing up in the medullary rays through the layer of polymorphous cells as the neuraxons of distant trophic cells, these fibres give off collaterals which interlace with the collaterals from the apical dentrites of pyramidal cells, while their terminating fibrillae assist in the formation of the dense arborizations so characteristic of the molecular layer. But besides the two great systems of motor and sensory fibres which connect the brain with all parts of the body, there are other systems of fibres of great physiological importance, which establish connection between various portions of the brain itself. Thus, there are certain areas in the cerebral hemispheres located in the three tiers of convolutions in the frontal lobe, and in the parieto-occipital, and occipito-temporal regions,[III] which are called association areas or centres. The fibres from these association centres are short and medullated, and are the means of establishing histological connection between the association centres and various areas of the cortex. Lastly, we have commissural fibres, which connect the hemispheres with each other, across the median line, and thus establish a co-relation in function, by means of which the hemispheres act in perfect unison.

Before leaving this subject, let us consider for a moment the manner in which the production of nervous impulses in the grey matter takes place. It is now generally agreed that efferent impulses result from a condition of molecular energy starting primarily in the branching dentrites of motor nerve cells, and further, that this condition of molecular energy is induced by impulses communicated to the dentrites from the terminal neuraxons of near or distant cells. Professor Foster[IV], in beautiful and elegant language thus describes the action of the nerve cell and its processes.

"Thanks to the black tract which silver when handled in a certain way leaves behind it in the animal body - as, indeed, it does everywhere - we can now trace out within the central nervous system the pathway afforded by the nerve cell and the nerve cell alone. We see its dendrites branching out in various directions, each alert to dance the molecular dance assigned to it at once by the more lasting conditions which we call structural, and the more passing ones which we call functional, so soon as some partner touch its hand. We see the body of the cell with its dominant nucleus ready to obey and yet to marshal and command the figure so started. We see the neuraxon prepared to carry that figure along itself, it may be to far-distant parts, it may be to near ones, or to divert it along collaterals, it may be many or it may be few, or to spread out at once among numerous seemingly equipollent branches. And whether it prove ultimately true or no that the figure of the dancing molecules sweeps always onwards along the dendrites towards the nucleus, and always onwards away from the nucleus along the neuraxon, or whatever way in the end be shown to be the exact differences in nature and action between the dendrites and the neuraxon, this at least seems sure, that cell plays upon cell only by such a kind of contact as seems to afford an opportunity for change in the figure of the dance, that is to say, in the nature of the impulse, and that in at least the ordinary play it is the terminal of the neuraxon (either of the main core or a collateral) of one cell which touches with a vibrating touch the dendrite or the body of some other cell".

In conclusion we may state that besides the sensory fibres, others ascend in the medullary rays to terminate in the arborizations of the molecular layer. These are association and commissural fibres, the neuraxons of cells in the association areas, and of the opposite hemisphere, and the vibrating terminals of which have the power of calling forth the latent molecular energy in the branching dentrites of motor cells. With this description of the histological structure of the grey matter before us, we shall now, with a greater degree of confidence, pass to the consideration of the causation of epilepsy.

At the outset, we may state that we regard Epilepsy as resulting from two causes, viz: predisposing and exciting, the former being a condition of atypical cerebral development, whilst the latter is either normal sensory stimulation, or abnormal irritation. Taking the predisposing cause first, we have now to consider the site and nature of the morbid conditions which constitute it. Regarding the site of these morbid changes, that, as mentioned previously, is now generally agreed to be the grey matter in the cerebral cortex. Fagge, however, believed that the seat of Epilepsy was in the basal ganglia, and he explained the existence of Jacksonian Epilepsy by the extension of cortical irritation to the lower ganglia. Van-der-Kolk, on the other hand, located the morbid changes in the medulla. In speaking of this subject Gowers[V] says:- "Epilepsy and Insanity are alike disorders of the cortex of the brain, the results of changes in the finer nutrition which have yet to be discovered, if the discovery is possible. I speak thus hesitatingly, because I am satisfied that, as regards Epilepsy, no alleged pathological observations are true, and least of all, those which at one time led the name Epilepsy in the reports of the Vienna Hospital to be changed to "Disease of the Cornua Ammonis". If the recent extension of our knowledge and of our conceptions of the central nervous system be trustworthy, and trustworthy it seems to be, we have never yet looked in the right place for the morbid changes which may underlie either the one disease or the other, and we have not yet reached even the position whence we can attempt to look for them. According to these conceptions

it is in the spongy grey matter in the interlacing network of fibrillae which intervenes between the cells, in which these branching processes end, it is in this that all nerve energy is elaborated, that all ordered action arises, and that all disordered action is produced."

Frederick Taylor[VI] mentions the following facts for consideration in connection with this subject.

(1) The association of coma with the convulsions.

(2) The existence of cases of masked Epilepsy in which the symptoms are mainly psychical, such as epileptic mania and delirium.

(3) The fact that definite lesions of the cortex, such as tumours or gumma produce convulsions identical in character with those of Epilepsy.

(4) The general uniform spread of the convulsions from face to arm, and arm to leg, corresponding to the relative positions of the motor areas for those parts on the surface of the brain, a result which may be observed, not only in the Idiopathic Epilepsy of man, but in the epileptic convulsions produced by experimental irritation of the brain in animals.

(5) That some cases of Epilepsy (Traumatic) begin with definite lesions of the cerebral cortex, e.g. as results of blows.

(6) The cases occasionally recorded in which epileptic fits have ceased after the development of disease in the internal capsule.

From the above considerations, and with the minute structure of the cerebral grey matter before us, I think we may with confidence confine the morbid changes in Epilepsy to the interlacing fibrillae in the cortical grey matter. The clinical aspect of Epilepsy alone suggests a morbid activity in the molecular layer, at first sustained, later erratic, and producing respectively, tonic and chlonic

convulsions. Having thus arrived at a definite conclusion as to the seat of the morbid conditions in Epilepsy, we shall now endeavor to explain the nature of these morbid conditions. Fagge considered Epilepsy to be a nerve storm and classed it with migraine and the allied neuroses. Van-der-Kolk deemed vaso-motor changes in the medulla the principal cause, while Gowers thinks it results from a condition of diminished resistance on the part of the cortical grey matter which is certainly most probable. At this point we go a step further and hazard the opinion that the morbid condition in Idiopathic Epilepsy is a delay, derangement, or arrestment in the development and complete myelination of association centres and fibres in the grey matter of the cortex. That the brain of the Epileptic is atypical may be concluded from a study of the so-called stigmata of degeneration. These stigmata which are indicative of arrested or deranged cerebral development may be anatomical, physiological or psychical. Under anatomical stigmata, Peterson VII mentions certain anomalies of the head, palate, teeth, nose, eye, limbs, spine, skin, and sexual organs. Under physiological stigmata are mentioned perverted motor and sensory function along with anomalies of the appetite and function of the genito urinary organs, while under psychical stigmata are classes Insanity, Idiocy, Imbecility, Moral Delinquency, feeble-mindedness, and sexual perversion. Now certain of these stigmata of degeneration are almost invariably present in Epilepsy. Thus Charon has found 76 per cent of abnormal palates in Epileptics, while Peterson pictures cases of phocomelus of right arm, facial asymmetry, and Darwinian ear as rarer stigmata occurring in Epileptics.

Psychical stigmata are also by no means uncommon in Epilepsy. In three cases of this disease which came under my notice while an assistant in England, sexual perversion (between the attacks) was a characteristic feature. In two of them a definite history of masturbation was obtained, and here it is well to emphasize the fact that masturbation is not a cause of Epilepsy, but is to be regarded as a symptom of the condition of cerebral insufficiency. The almost universal occurrence then of these stigmata in Epileptics strongly supports the hypothesis that Idiopathic Epilepsy results from a mal-developed condition of the cerebrum. Now, in the two epochs of life, namely, youth and advanced years, the human brain is the most liable to be the seat of functional disturbances, and morbid conditions. While in the first decades of life, disturbances arise from perversion of, or delay and arrestment in the developmental process, the brain, after the fifth decade of human life, becomes the seat of those degenerative changes in its minute structure, which predispose to the nervous diseases and mental conditons which are so frequently the concomitants of advanced years. One of the most frequent diseases in the earlier part of life is Epilepsy. Gowers mentions seventy-five per cent of Epileptics commencing under the age of twenty. Further infantile Eclampsia is so characteristic of childhood that it impresses upon one the fact that the condition of the brain during the developmental process must predispose

to convulsive seizures. In studying the process of cerebral development, we find that the sensory fibres first become myelinated, and consequent upon this, and the receipt in the brain of sensory ~~impressions~~ impulses, the motor fibres next display myelination. On the other hand, the myelination of the association fibres which pass between the sensory-motor areas and the association centres takes place at a later period, adult life at times being reached before they are functionally complete. This fact, according to Foster, indicates the complex function of these fibres. Foster suggests that the myelination of these association fibres in normal brains is dependent for its completeness upon education and environment. Inheritance, however, we must remember, may also by its obscure, yet subtle influence, induce a condition of arrested or perverted myelination of these fibres, apart altogether from the effect of environment. On the completion of the myelination of the sensory and motor fibres, the human brain has reached crucial point in thr process of its development. It is then that the myelination of the association fibres takes place, and the development of the higher centres is consummated. That the myelination of these fibres takes place from the sensory motor areas towards the association centres, can with confidence be inferred. As the child grows, and the brain becomes the recipient of more numerous and varied sensory impulses, the association fibres, which in the fully developed brain would convey the resultant form of nerve energy to the association centres, first become myelinated.
At this point the association centres in the superior and frontal aspects of the brain, which, according to Flechsig, constitute the material antecedents of mental activities, gradually become perfected.

It is by means of these centres that excessive reflex stimulation of the motor cells is inhibited, and that sensory impulses are transmuted and subsequently induce, by the contact of terminal neuraxons with the dentrites of the motor nerve cells, varied and complex muscular movements. This group of association neuraxons are the last to become myelinated by which time the brain has become functionally complete. We thus see that in the normal brain the Rolandic area is subservient to, and the activity in the molecular layer inhibited by the association centres. In Epilepsy however, we have a departure from the normal condition of the cortical grey matter as is evidenced in the severer types of Epilepsy by microgyria in the superior and frontal aspects of the brain.[VIII] The following are the varieties of atypical development which the brain may assume as indicated by Meickle[IX] in an elaborate paper on the subject:

(1) Deranged development.

(2) Development arrested with persistence of foetal characters.

(3) Reversion towards the type of lower races of men.

(4) Reversion towards the type of lower animals.

Now, if we compare the normal brain of an adult man with these types, we shall see that in the latter the convolutions in which are located the association centres are poorly and inadequately developed. Thus, in the brain of the newly born infant, we find that while the convolutions in the Rolandic area are well marked, the association convolutions are very poorly developed, less even than those in the brain of a chimpanzee three or four years old. It is in the first two varieties of atypical cerebral development mentioned by Meickle that we have the conditions which predispose to Epilepsy. In the first variety we have a condition of deranged development, while the second is characterized by arrested development with the persistence of foetal characters. Further, there is a third variety of atypical development occasionally met with, which predisposes to Epileptic seizures and which is characterized by a delay in the process of myelination of the association fibres. That this conception of the condition of the brain in Epilepsy is true, is shown by the following facts.

(1) The fact that the brain in infancy which has the association system of centres and fibres as yet undeveloped, predisposes to epileptiform seizures.

(2) The condition of arrested development characterized by microgyria in the association areas seen in Epileptic Idiocy and Imbecility.

(3) The fact that loss of inhibitory power and rapid response to sensory impulses so characteristic of the infantile brain is also a marked feature in cases of Chronic Epilepsy.

(4) The fact that injury to the frontal region where is located the special group of controlling association centres (McKendrick) is followed in 50 per cent of cases by convulsions, a greater percentage than follows injury to the Rolandic area.

(5) The occasional occurrence of Epileptic fits during the first stage of chloroform narcosis, when the inhibitory power exercised by the association centres over the Rolandic area has been withdrawn. Two such cases are recorded by Baumgartner in the "London Medical Record" for 1882.

In considering the subject in detail, we have first, the most serious condition of complete arrestment with persistence of the foetal type. The process of brain development subsequent to birth depends upon two factors:

(1) An inherited capacity to develop.

(2) Suitable educational influences for the induction of myelination of the association fibres, and development of the association centres.

Now, in the type of brain under consideration, the association centres and fibres remain embryonic due to an inherited lack of capacity on the part of the brain to benefit by the receipt of educational impulses. Such a type of brain is characteristic of Epileptic Idiocy and Imbecility as is indicated by the condition of microgyria in the areas in which are located the association centres. The clinical aspect of such cases also point to this conclusion.

A less severe type, however, is that in which the process of brain development has been deranged. One hemisphere may be histologically complete, while the other, from inherited incapacity to develop, retains its foetal characters. Such cases display clinically unilateral convulsions.

In writing upon this subject under the head of "Dual Brain Action" Dr. Bruce[21] draws the following conclusions:-

"That in the normal state of certain epileptics under consideration, the left cerebral hemisphere controlled the functions of the right, the development of which must have been low, either from atavism or from congenital deficiency not having the capacity to develop. The loss of inhibitory power and the tendency to irritability common to all patients suffering from Epilepsy was mentioned. In the brain of those, who through heredity were predisposed to Epilepsy and the allied neuroses, there was possibly a considerable difference in the mental development and education of the two hemispheres".

Dr. Bruce states that these conclusions were drawn from observations made upon patients suffering from unilateral right-sided Epilepsy. On the other hand, the development of both hemispheres may be incomplete, and this alone can account for the occurrence of bilateral Epilepsy. Heredity associated with a dearth of normal educational influences accounts for such a condition. In such cases however, the brain has a certain capacity to develop as the association centres which are the material antecedents of mental activities are fairly well developed, as is indicated by the standard of mental development, but the association fibres which link these centres to the molecular layer in the motor and other areas are presumably defective. Their myelination is crude or incomplete, and although under normal circumstances they are sufficiently developed to perform their important function of controlling the production of motor nerve energy, i.e. automatic movements, and transmuting sensory impulses through the medium of the higher centres into complex and varied motor impulses, yet in the presence of exciting causes, e.g. sensory irritation, their

insufficiency is made manifest by their inability to inhibit the over stimulated dentrites in the molecular layer, and dissipate along collaterals the morbid excess of nerve energy. Clinically, cases of Epilepsy arising from a condition of deranged development may be recognised by the persistence of the epileptic attacks, either as grand mal or petit mal throughout life. There is marked evidence of cerebral instability in the rapid response to sensory impulses and loss of inhibitory power characteristic of these cases. The existence, in most cases, of a sensory aura indicates that the wave of morbid activity starts primarily in some portion of the sensory system and lashes into a convulsive state the vibrating dentrites of the motor nerve cells. The association system being imperfect, inhibition is erratic, and thus the unrestrained nervous activity explodes in an epileptic attack. The presence of tonic and chlonic convulsions in the same attack points to the manner in which the motor cells respond to the impulses transmitted to them along their apical dentrites. Previous to the onset of an epileptic convulsion, the motor nerve cell is in the normal state of latent activity, but ready to respond to any impulse which may reach it. On the sudden outburst of the morbid nerve activity which ushers in an epileptic attack, the molecular changes in the cell which anticipate the flash of efferent impulses along the neuraxons, occur with such rapidity as to keep up a continuous stream of nerve energy which induces a state of tonic or continuous contraction. Under such circumstances, the nerve cell soon becomes exhausted, and consequently, it responds in an erratic manner to the impulses reaching it, with the result that chlonic convulsions are induced. Brain exhaustion follows, and the patient passes usually into a deep sleep. The unconsciousness characteristic of Epilepsy can only be regarded as analagous to that produced by concussion and is probably induced by the violent condition of unrestrained activity in the vibrating terminals of the molecular layer. The nature of attack, whether grand mal or petit mal, depends on the severity of the exciting cause and

the extent of the derangement in the association system, both separately and combined. In Petit Mal, although the morbid activity is sufficient to produce unconsciousness, yet it falls short of inducing convulsions.

Lastly, we have constituting a small minority, cases of Epilepsy in which the predisposing cause can only be regarded as a delay in the normal process of brain development. In such cases we have epileptic attacks occurring in childhood and youth but which disappear when adult life is reached. To what extent this delay depends upon inheritance or the absence of suitable environment, it is impossible definitely to say. Personally, I have met with one such case in which attacks of grand mal gradually became less severe and less frequent as adult life was approached. The patient is now 21, and while at the age of 14 she suffered from frequent attacks, the exact number I could not ascertain, she at the present time has only had two fits within the past twelve months. The father states that, as a child, she had for a considerable time been under medical treatment, but with no effect, and as he considered her incurable, it was ultimately abandoned. He also states that during the last few years they have noticed gradual signs of improvement.

Having thus considered the atypical brain condition which predisposes to Epilepsy, we shall now pass to the study of the exciting causes which, as mentioned before, we consider to be the receipt in the grey matter of sensory impulses, either normal or morbid. The consideration of the following facts throws light upon the subject.

(1) The physiological fact that the motor nerve cells in the Rolandic area are stimulated through their apical dentrites by afferent impulses terminating in the arborizations of the molecular layer.
(2) The clinical fact that sensory irritation induces epileptiform seizures, e.g. Infantile Convulsions, convulsions brought on by the passage of a renal calculus, &c.
(3) The fact that in many cases of Idiopathic Epilepsy, sources of reflex irritation are present, the removal of which may considerably ameliorate the condition, if not bring about complete recovery.
In considering these facts in detail, we wish first to emphasize the physiological statement first mentioned. Foster and Sherrington draw attention to its importance in their combined volume upon the nervous system, while Prof. McKendrick in his lectures, lays considerable stress upon its significance. In the earlier portion of this paper we spent some time in studying the various paths followed by sensory impulses on their way to the brain. It is at this point that the importance of some degree of minuteness in histological study in connection with this subject is made manifest. The structure of the cortical grey matter and the fact that the motor fibres myelinate subsequent to the sensory, are sufficient to prove beyond all doubt that motor impulses depend for their birth upon the receipt of sensory impulses in the molecular layer of the grey matter. Further, the clinical fact that sensory irritation induces epileptiform seizures, shows that the receipt of abnormal sensory impulses produces disordered action.
Infancy is characterized by the absence of inhibitory power which is subsequently developed when the brain becomes complete, hence the convulsions induced by the cutting of a tooth, the presence of worms, or the circulation of toxins in the blood.

yet, on the other hand, the same cause, but many times intensified, may induce convulsions in the adult, although the brain, as far as we can learn, seems perfectly normal. This, however, is nowise difficult to explain. The passing of a renal calculus may produce waves of impulses so intense that the inhibitory power of the normal brain is quite inadequate to prevent them lashing into morbid activity the apical dentrites of the motor cells, and thus inducing disordered action. Lastly, the fact that in many cases of Idiopathic Epilepsy, sources of reflex irritation are present, goes far to prove that this conception of the exciting cause is correct.
Lamphear[XII] classifies the various forms of Epilepsy into three groups, The toxic, reflex, and irritative (Jacksonian). In the first are placed all Epileptics in which no post mortem changes are to be detected, these being regarded as due to toxic agencies. The reflex varieties he ascribes to phimosis, haemorrhoids, nasal obstruction, and the involvement of nerves in cicatrices. Voisin and Petit[XIII] "consider that for the production of epileptic onsets a hereditary predisposition exists in the central nervous system; that the epilepsy may result from some reflex nervous disturbance or from an auto-intoxication and that this may be caused by an auto- or hetero-infection".
During the past few years, considerable stress has been laid upon auto-infection as a causative factor in epilepsy, and there is every reason to believe that when present, it constitutes the exciting cause. In the light of the predisposing cause considered above, it is self evident that if the blood circulating through the

brain possesses irritating qualities, it will, with the loss of inhibitory power, be the means of morbidly stimulating the motor cells or their dentrites with the consequent outburst of disordered action. In the majority of cases however, there is sufficient evidence to prove that sensory irritation mainly constitutes the exciting cause. "In his Lettsonian lectures, De Havilland Hall, in speaking of the relation of Epilepsy to throat and nose, mentions cases reported by Dr. Siethoff of reflex Epilepsy of nasal origin, cured by intra-nasal treatment, one case being accompanied by an olfactory aura. Similar cases have been reported by Dr. Crossfield and Mr. Barclay. Lichtivitz has seen epileptiform convulsions attended with loss of consciousness resulting from irrigation of the sphenoidal sinus, and in a boy aged ten, crises resembling petit mal were cured after removing pus from the sphenoidal sinus. Kjellman (Stockholm) has collected fifteen cases of epileptiform convulsions which ceased after the cure of the following pathological conditions of the nasal cavities: polypi, foreign bodies, and swelling of the mucous membrane of the turbinated bones. The tonsils may also be the exciting cause of the epileptiform crisis, as shown by a case recorded by Boulay of Paris. In Siethoff's second case, it is stated that the attacks were always preceded by an olfactory aura."

The following cases have come under my own observation:

Case (1). J. M. a female, has suffered since puberty from transient attacks resembling petit mal. Family history good; sister of nineteen had convulsion on reaching puberty, but none since.

Other members of family are in perfect health. There is no history of epilepsy on either the father's or mother's side. Prior to the first attack, the patient had enjoyed good health. In appearance she is well formed, though somewhat anaemic. Her head is somewhat small and her forehead distinctly narrow. The attacks from which she suffered, not more than twice or thrice annually, were characterized by complete unconsciousness, conjested face, and a certain amount of muscular rigidity. The pupil was generally dilated and fixed. Probable exciting causes present were neuralgia arising from decayed teeth, conjested condition of the pharynx, and swelling of the mucous membrane of the Eustachian Tubes. During the process of treating these conditions, she suffered from two attacks - once when a decayed molar, which had caused her considerable pain, was being removed, and again when the cautery was being applied to the pharynx. Since undergoing this treatment, however, over a year ago, she has had no recurrence of the attacks. Finally, it is interesting to note in connection with this case, that the patient was distinctly conscious of a definite aura referable, first to her stomach, and then to her head, when she became unconscious.

Case (2). The facts in connection with this case were given me by the surgeon who had seen her in consultation. The patient was a young adult female who had suffered for some time past from frequent epileptiform seizures. The family history was unimportant. The attacks were characterized by unconsciousness with tonic and clonic convulsions. The patient was distinctly neurotic. Examination revealed the fact that many of her teeth were in an advanced state of decay.

These being regarded as the exciting cause of the attacks, were removed, with the result that she showed considerable improvement, the seizures being fewer and less severe. As, however, a further exciting cause was suspected, she was again carefully examined, with the result that pus was found ousing from the orifice of the right eustachian tube. The ear condition was consequently treated, with the result that after some lapse of time the convulsions ceased altogether. During twelve months she has had no recurrence.

Case (3). D. H. an adult male aged thirty has suffered since childhood from frequent attacks of petit mal, the exciting cause of which is, in every instance, the receipt in the brain of sensory impulses. On the mother's side there are definite traces of neurosis, but otherwise, the familt history is unimportant. The patient's head is distinctly microcephalic, pointing to interference in the process of brain development. The attacks from which he suffers are a somewhat severe form of petit mal. On one occasion while sitting at dinner, the sudden closure of a drawer was sufficient to induce the condition. Again, the unexpected presence in his way of any obstacle, the accidental slipping of his foot, or any sudden noise, have, on more than one occasion, been observed by the writer to act as exciting causes. The attacks are mainly characterized by complete unconsciousness and muscular rigidity. His pupils are dilated and fixed, his mouth partially open, while there is slight conjugate deviation of head and eyes to the right.

The attacks only last for a few seconds, but their frequency depends altogether upon the presence or absence of exciting causes. The interesting phase of this case is the fact that the exciting causes are normal sensory impulses and not any local source of irritation which can be combated with. Treatment, as might be expected, is quite unavailing.

Case (4). Mrs. M. Widow, aged 65, has for many years suffered from frequent attacks of grand mal. Examination revealed the probable exciting cause to be dyspepsia, to which she is frequently a martyr. She states that when suffering from dyspepsia the fits are more frequent and severe. Suitable treatment for the stomach condition was certainly followed by a diminution in the frequency and severity of the attacks, but it must be added that no hope could be held out for complete recovery.

A study of these cases therefore, cannot but impress upon one's mind the probability that Epilepsy either as grand mal or petit mal depends for its birth upon the receipt in the molecular layer of afferent impulses arising from the normal or morbid stimulation of sensory nerve filaments.

Having thus considered the probable causation of Epilepsy, we shall now conclude with some remarks regarding its possible relation to Insanity and Hysteria. That a relation does exist between Epilepsy and Insanity is proved by the fact that many Epileptics end their days in the asylum. Mercier[XIV] says that thirteen hundred Epileptics are annually admitted into the asylums of England and Wales. The clinical study however of this subject is beset with many difficulties, for, while the general practitioner may have ample opportunity of studying the clinical aspects of Epilepsy itself, he has little or no opportunity of participating in the study of any morbid mental condition which may subsequently develop, as the patient is immediately removed to some asylum when signs of mental derangement or failure are observed. This is certainly to be regretted, as the lack of co-operation between the practitioner who is conversant with the clinical features of the epileptic crisis, and the psychologist who subsequently studies the nature of the mental derangement retards the elucidation

of many important points regarding the relation of the one disease to the other. The first point to be considered in connection with this subject is the light which the study of inheritance may throw upon the relationship. Among the cases which have come under my own observation, I have in one instance only been able to get a history of Insanity, and that is the case of J. M. an Epileptic suffering from dementia, whose brother was also insane. Clouston, XY in writing on this subject, says:

"Hereditarily ordinary insanity and epilepsy are more closely allied than any other two of the neuroses. The son or daughter of an Epileptic is just as likely to be weak-minded, drunken, or insane, as to be epileptic, and certainly, Epilepsy occurs frequently in the children of families with a strongly insane heredity. To Gowers, however, we must turn for statistical information. In opening a discussion on this subject in the section of psychology at the meeting of the British Medical Association held in London in 1895, Gowers says:

"Inheritance, epilepsy or insanity, was traceable in the first series in 35 per cent. In the second series the proportion was 44.5 per cent., but in the case of this series seen in private the proportion was 48.4 per cent., or nearly half. But this, we may be confident, is below the truth, and that at least in one-half of the cases there is inheritance. The lesson is that statistics regarding inheritance taken from cases in lower classes are at least 30 per cent below the truth, that one-third ought to be one-half, and that it is essential that the cases collected should be separately considered according to the position in life of the patients.

I mention this general fact on account of its importance, but we are now concerned with insanity only as an antecedent of epilepsy. It is an interesting fact that, in my second series, although inheritance was traceable in so much larger a proportion of the whole, the proportion of the cases with heredity taken separately in which insanity could be ascertained is almost the same as in the first series. In each insanity had occurred in the relations of one-third of the cases in which there was evidence of heredity; while epilepsy existed alone in two-thirds. In the first series inheritance was traced in 439, in the second series in 457. These two sets of cases

with inheritance 439 and 457, are nearly equal. In the 439 cases, insanity was heard of in 157; in the 457 cases, insanity was heard of in 170. The proportion in the first series was 33 per cent., in the second 37 per cent. I think that the fact that the great increase in the proportion of heredity, due to better information, is only attended with a slight increase in the proportion of insanity, is a striking indication of the general trustworthiness of these facts. This is the more satisfactory, because we are absolutely dependent upon the statistical method for such knowledge."

We thus see that in the family history of Epileptics, Insanity is frequently met with, a fact which shows that the morbid condition of the brain which predisposes to Epilepsy may be inherited from an ancestor suffering from some form of Insanity, and further, that an Epileptic may transmit to his offspring some form of mental derangement just as readily as the Epileptic crisis itself.

Here is the family history of four individuals suffering from mental derangement, which is interesting and instructive.

(1) B.D. a married female, age forty, suffering from melancholia; father was epileptic and a sister insane.

(2) E.P. a girl aged eighteen suffering from stupor; one grandfather was melancholic with delusions, while her father suffered from severe epileptic attacks after which he was very excitable.

(3) E.T. married female aged forty seven suffering from general impulsiveness; brother insane and epileptic, and a sister insane.

(4) J.N. a female aged twenty one, suffering from hysterical Insanity has an aunt epileptic.

These cases are of interest as showing the occurrence of Epilepsy in the family history of the insane. More satisfactory information, however, regarding the relation to Epilepsy may be obtained by the combined clinical and pathological study of the subject The primarily morbid brain condition in all cases of mental derangement can only be inferred as in Epilepsy alone, from the clinical features of the disease. Yet, notwithstanding this, the brain should in every case of Insanity and Epilepsy, if opportunity affords, be carefully examined post mortem, the size and weight of the hemispheres contrasted, the depth of the sulci and the development of the

convolutions compared, and the minute structure of the nerve cells and their processes carefully studied. Bevan Lewis has done much important work in connection with this subject. In his work on mental diseases he states that his observations point to a degenerative condition affecting the small irregularly shaped cells in the second layer of the grey matter of the cortex. The Nucleus, according to this observer, is first affected, its centre being replaced by a highly refractive globular body, fatty in nature, which enlarges until there is marked vacuolation of the nucleus; subsequently, the surrounding protoplasm also shows degenerative changes. Lewis considers that these changes in the cell nucleus are constant accompaniments of cerebral disturbances characterized by loss of inhibitory control. He also mentions that they occur in other diseases, such as alcoholic disease of the brain. Now, while it is perfectly obvious that certain degenerative changes in the nerve cells must follow the condition of prolonged morbid activity in the complex network of the molecular layer Lewis has by no means clearly demonstrated that these changes constitute the initial morbid condition which induces Epilepsy or Insanity. He has omitted to state whether the brains examined by him belonged to recently developed or long standing cases. Middlemass and Robertson of Edinburgh have found certain changes in the cortical cells differing somewhat from those observed by Lewis. The nuclei in certain cases were perfectly normal, while the protoplasm was the seat of degenerative changes. Further, it was observed that these changes were not confined to the cells of any special layer, but were scattered indiscriminately throughout the cortical grey matter. In many chronic varieties of Insanity, however, nuclear vacuolation was noted by these observers. The presumption then is that these morbid changes are the sequence of a condition of initial morbid activity in the molecular layer rather than the cause of morbid mental manifestations or epileptic seizures; that as atheromatons changes in the great vessels of an otherwise healthy navvy follow the prolonged condition of high arterial tension, so do the fatty changes in the nerve cells follow in the footsteps of prolonged abnormal stimulation. With these preliminary remarks upon the visible pathological changes which are present in many, but not all cases of Epilepsy and Insanity, we shall now consider in detail the various manifestations of mental

diseases related to Epilepsy.

(1) Post Epileptic Mania is, as Clouston mentions, by far the most common manifestation of mental derangement associated with Epilepsy. Out of sixteen cases of Epileptic Insanity seen by me in Woodilee Asylum, no less than twelve suffered, or had suffered from acute mania following the epileptic crisis. Such patients are extremely violent and may have strong homicidal impulses.

The following are typical cases of Post Epileptic Mania:

(a) C.C. a little stout pale-faced woman who has suffered since the age of ten from frequent Epileptic Convulsions. When questioned regarding the existence of an aura, she stated that she was always aware of the approach of a fit by the fact that she felt she was at home, seated in a favored chair, with her mother alongside engaging her in conversation. Associated with the delusion and hallucinations of sight and hearing, is a certain amount of irritability and excitement. Her mental condition following the convulsion is one of acute mania, when she is very violent, necessitating the presence of several attendants, throwing herself about and endeavoring to tear her clothes off. Her disposition is very sullen and she has marked homicidal tendencies, attacking with ferocity those nearest her. When well she has no recollection of what occurs during these attacks.

(b) M.K. a young married woman aged thirty, of pleasing appearance but somewhat excitable manner, was admitted to the asylum after a violent attack of "Status Epilepticus" during which, her friends say, she had no fewer than twenty-nine convulsions. The patient has had convulsions since a child, but subsequent to the attack of "Status Epilepticus" each convulsion has been followed by violent maniacal outbursts. On interrogation, she states that before the onset of a convulsion she is conscious of a peculiar feeling in the region of her heart, and intense headache, co-existing with which is a considerable amount of irritability and excitement. The attacks of mania are very violent, the patient throwing herself about, tearing her clothes, and attacking with homicidal intent, whoever is near her.

The other cases of Post Epileptic Mania seen in Woodilee Asylum, displayed similar features to the above. Although they were all

carefully questioned as to the presence of a psychical aura, in one only - C.C. above mentioned - was there any real evidence of such existing. It is impossible however to place much reliance upon the state-ments of these patients, as the majority of them are more or less demented. Many of them however, emphasized the presence of a severe headache and feeling of confusion as indicating the near approach of an epileptic attack. Of the twelve patients suffering from this form of mania, four were males, and the rest females; the ages of the men ranging from 30 to 50, and those of the women from 26 to 40. The disease in all these cases ran a somewhat similar course; the epileptic convulsions starting under the age of ten, in the majority diurnal, but in a few nocturnal, and recurring for years at frequent intervals with the subsequent development of acute mania. The maniacal attacks gradually become subacute, coincident with which is the slow but progressive development of dementia.

When we come to consider the explanation of attacks of post epileptic mania, we approach a difficult subject. The clinical features of such cases point to a transference of the condition of morbid activity from the motor to the mental centres, setting up thereby a mental Epilepsy. What then is the relation of the motor centres to the mental, and in what manner does the morbid activity reach the latter? In the light of present day physiology, we cannot conceive a nerve impulse starting de novo in a nerve cell; the molecular changes in the cell which anticipate the impulse are initiated by the contact of the apical dentrite with some terminal neuron. We thus see that the mental centres in post epileptic mania must be morbidly stimulated by impulses reaching them from some other region of the brain and that region is presumably the sensory motor area of the cerebral cortex. In studying the histological structure of the brain, we saw that many sensory fibres terminated in the motor area by inosculating with the dentrites of motor cells, thus forming a reflex arc which accounts for automatic movements, but we must remember that many of the sensory fibres end in various areas of the cortex in special sensory cells, the neurons of which pass to association centres located in the parieto-occipital, and occipito-temporal regions; these association centres are closely identified with the mental processes. The motor area is also connected with these higher centres by short medullated fibres.

Lastly, it has been observed that special groups of fibres stream from the posterior association areas in the cortex to the frontal region, where it is presumed that the special centre associated with volition is located, this fact is of interest when we remember that metaphysicians define volition as an attribute of the brain to which all others are subservient. The presumption then is that in mania following attacks of grand mal we have primarily morbid nerve activity in the molecular layer of the grey matter, an activity the excess of which when the motor cells become exhausted, is diverted along the neurons of other cells to the mental centres which thus become the seat of epileptic discharges owing to the loss of inhibitory control. In these cases however, in which mental derangement follows attacks of petit mal the morbid activity seems to affect principally the dentrites of those cells, the neurons of which pass to the mental centres.

(2) The second form of Epileptic Insanity is épilepsie larvée or masked epilepsy, in which we have the mental disturbance taking the place of Epileptic Convulsions. Clouston records a very interesting case of a patient, who, while he suffered at intervals from attacks of grand mal and petit mal associated with mania, was also the victim of violent outbursts of épilepsie larvée. During these attacks, he would suddenly strike out at those nearest him, or his expression of face would change and become furious while he would stare at anyone beside him and shout fiercely: "What the Devil do you mean sir?" On one occasion while playing at a quiet game of whist he suddenly and without any warning let fall his cards with a profane exclamation, and sprang across the table at his partner's throat. He was gently restrained, and in a few minutes resumed the game quite unconscious of what had happened.

The following case of épilepsie larvée I had the opportunity of seeing in Woodilee Asylum:

Mrs. M. a young married woman aged thirty possessed of an excitable and emotional disposition. The attacks of disordered mental action occur at frequent intervals and are characterized by extreme excitement and violence, the patient rushing at and attacking with great fierceness the first person upon whom she can lay hands. She is unconscious of her actions and quite unable to control them, and when the attack is over she is quite ignorant of having conducted herself in any

extraordinary manner. When questioned regarding her illness, she declined to give any information, in fact, refused to speak, indulging all the time in meaningless laughter: she is evidently become demented. In these cases disordered mental action takes the place of epileptic convulsions which have ceased. The clinical features of such cases point to epileptic discharges in the mental centres; the morbid activ-ity having been diverted from the dentrites of motor cells to those of cells associated with the mental process.

(3) The slow but progressive development of dementia invariably accompanies repeated attacks of Epileptic Mania, and is also frequently the termination of those who have suffered from convulsions only. The twelve cases of Epileptic Mania seen in Woodilee Asylum showed unmistakably by their conduct between the attacks that they were becoming demented. The following cases are illustrative of a severe and somewhat rapid type of dementia associated with Epileptic attacks.

(a) J.M. had up to the age of puberty been a bright, intelligent, and apparently healthy girl, but with the development of the menstrual function she commenced to take Epileptic Convulsions while there gradually ensued symptoms of dementia. She is now at the age of 26 hopelessly demented: her face is large, heavy, and expressionless, her stare vacant, while her mouth remains partially opened. Her memory as to past events is a blank, and she has lost the power of articulate speech. In listening to her meaningless chatter for some minutes, I was, during that time, only able to make out one word which was Kilmarnock.

(b) F.T. like the former patient had been an intelligent healthy child until the onset of puberty, when she was seized with Epileptic Convulsions associated with the gradual development of dementia. She is now at the age of 30 a little old-looking woman, with a heavy repulsive hairy face. Her memory is gone, and she also has lost the power of articulate speech, indulging when spoken to, in a meaningless idiotic chatter. She is hopelessly demented.

These two cases illustrate the somewhat rapid onset of dementia in young persons who suffered from very violent and frequent attacks of grand mal. The explanation of dementia as a sequence of acute maniacal and epileptic attacks is apparent. The condition of brain decadence characteristic of advanced years, depends for its extent upon

the amount of strain and over exertion to which the nervous system as a whole has been submitted. Clouston, in the section of Psychology at the annual meeting of the British Medical Association held in Edinburgh this year, delivered a very interesting address on the neuroses and psychoses of decadence, in which he called attention to three types of nervous and mental lesion connected with decadence: "The first consists chiefly of a vaso trophic degeneration, the second is best represented by those degenerations of the motor and sometimes sensory systems which constitute the ataxies, scleroses, &c., while the third is constituted by the mental disturbances grouped under the climacteric and senile insanities." It is the last type in which we are specially interested, in which the mental centres are the first seat of degenerative changes, but what these changes are, pathologists cannot as yet with confidence, say. In advanced years the nerve cell has been observed to be granular, and its processes diminished in number, while by Golgie's method, its dentrites have displayed atrophy or degenerative swelling. The primary site of degenerative changes is undoubtedly the dense network of interosculating neurons and dentrites in the molecular layer, and any condition of atrophy or bulbous swelling will interfere with the normal contiguity of these innumerable fibrillae, and thus check the sweep of molecular activity to the nerve cells, which, when associated with the mental processes, will be evidenced by the failure of mental activities. Such being the case, as observed by pathologists in senile dementia, it is presumed that in dimentia associated with epilepsy, the prolonged condition of strain and brain irritability arising from repeated epileptic seizures associated also with loss of inhibitory power has induced degenerative changes in the terminal neurons and branching dentrites in the mental areas, that we have in fact, a normal brain decadence induced at an early age by the prolonged and morbid brain irritation.

Finally, let us consider briefly the clinical relations of Epilepsy to Hysteria. Epilepsy and Hysteria may co-exist together as the Hystero Epilepsy or Hysteria Major of the French or Hysterical symptoms may follow or precede the Epileptic crisis. D'Oliers discusses eighteen cases which illustrate the four groups into which Charcot has divided Hystero Epilepsy:

(1) Hysteria supervening in a subject already epileptic.

(2) Epilepsy occurring in a patient previously hysterical.

(3) Convulsive Hysteria co-existing with petit mal.

(4) Epilepsy superadded to non-convulsive manifestations (contracture anaesthesia, &c.)

D'Oliers' cases showed that these forms of disease are met with in the male as well as in the female. His studies led him to add a fifth group of cases to the above four. It consists of those cases in which Epilepsy and Hysteria not only co-exist, but as it were, coincide, that is to say, each Epileptic fit is accompanied by hysteriform symptoms.

The following case which came under my own observation, illustrates the severe condition of Hysteria occurring in a patient already Epileptic. The patient was a young married woman belonging to a highly neurotic family, and having, prior to her present illness, a definite history of Epileptic seizures. Her family history was significant, one brother being Epileptic, another dying when a youth in the "Status Epilepticus", while her mother was also said to have suffered from occasional fits. The exciting cause of the present attack was anxiety and sleeplessness, occasioned by the prolonged nursing of a child suffering from broncho-pneumonia. For a few days prior to the onset of convulsions many of the prodromal symptoms characteristic of this severe form of hysteria were present. The most striking of these were persistent globus hystericus, palpitation of the heart, gastralgia, marked ovarian hyperaesthesia, and severe headache with exquisite tenderness to cranial percussion. Her pulse was rapid and of very low tension, her breathing hurried and sighing, and frequently she gave way to violent paroxysms of weeping. The pupils showed continued dilation. The tongue on protrusion was very tremulous and the hands betrayed muscular twitching. Menstruation was suppressed owing to lactation, and the bowels were obstinately confined; her urine however, was normal in character and quantity. The convulsions which followed, though at first slight and occurring at considerable intervals, increased so rapidly in frequency and intensity as to bear a strong similarity to the severe form of epilepsy the "status epilepticus". Each attack was ushered in with full dilatation and fixation of the pupil, succeeded by conjugate deviation of the head and eyes to the right. No cry, however, was emitted, and on

subsequent questioning the patient stated that she was not conscious of any definite aura.

Three stages now followed (1) the tonic stage, (2) the clonic stage, and (3) the stage of emotional excitement. The tonic stage was short but intense, and the convulsions varied in character with each attack. At one time the patient became rigid as she lay; at another, suddenly and with great violence, she would shoot out her closed hands and rigidly assume the crucifixion attitude; or, again, the tonic stage would start with trismus, rigidity of the muscles of the neck, and marked flexion of the forearms. The pulse during this stage increased in tension.

The clonic convulsions which followed were characteristic of an hysterical element; they were most violent and varied in character, yet without exception they showed to a greater or less degree a supervision of volition. At times the head would roll from side to side, followed by rapid flexion and extension of both extremities, or violent lateral and anteroposterior movements of the trunk. Again the patient would suddenly sit up in bed and endeavor to seize with her teeth, or otherwise injure attendants present, failing which she would bend herself forwards in a posture suggestive of emprosthotonos, or throw herself back with no inconsiderable violence. During these attacks there was little or no frothing at the mouth, and her face, though pale, was never livid.

Invariably the clonic stage ended with the patient resting on her heels and vertex in the posture of opisthotonos, during which there was great muscular rigidity, with fixation of the chest and labored breathing. The patient, now thoroughly exhausted, would lie quiet for a brief period, but gradually she passed into the stage of high emotion, in which she would weep bitterly, or point with a look of rapture and expectancy to imaginary forms, and give utterance to the names of friends long since dead. Thus her emotions would sway until she gradually quieted down or passed into another convulsion.

In the above case there are several points worthy of note, as indicating an epileptic tendency associated with hysterical manifestations. The family history, the story of previous epileptic seizures, the presence of severe headache and persistent dilatation of the pupils, all point to the fact that the patient was undoubtedly epileptic: on the other hand, the prodromal symptoms, the character

of the convulsions, especially the crucifixion and opisthotonos attitudes, the partial consciousness, and the final display of emotional excitement point to hysteria as playing a considerable part in the attack.

One other case of hystero epilepsy I have seen, the patient being a male aged 26 at present confined in Woodilee Asylum. The attacks, which it is interesting to note always occur just prior to a well defined epileptic seizure, display all the features of typical hysteria major, crucifixion attitude, purposive movements instead of typical convulsions and excessive emotional excitement. The patient, subsequent to the attacks of true epilepsy is very excitable.

Further, we may have hysterical symptoms appearing after an attack of true epilepsy, as the following case illustrates:
M.C. aged 26 a stout well developed female has suffered since puberty from attacks of grand mal occurring always at the time of menstruation. Immediately following the attacks are evidences of hysteria in the violent paroxysms of weeping with ringing of hands, and sudden outbursts of laughter in which the patient indulges. The conclusion from the study of these cases is that while epilepsy and hysteria are generally combined in the same attack, the latter may in some cases precede or follow the former.

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In conclusion we may state that cases of epileptic insanity and hystero-epilepsy show that there is a great tendency for the morbid excitability to spread from the brain area in which it primarily originates, and involve other regions associated with psychical processes and mental equilibrium. The reason of this is obvious when we bear in mind the contiguity of neuron and dentrite, and recall the loss of inhibitory control and rapid response to sensory stimulation which is so characteristic of the epileptic brain. Develop the inhibitory power and endeavor, during the period of growth, to surround the nervous system with such influences as will insure the most perfect and least erratic sensory impulses, and the human brain, provided it has inherited the capacity to develop, will attain a state of perfection which is quite incompatible with the presence of epileptic discharges either in the motor area or in the higher centres associated with mentalization.

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