TRAUMATIC EPILEPSY IN ITS MEDICO-LEGAL RELATIONS.*

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Although the clinical phenomena of the epileptic condition have been recognized since very ancient times, yet when we come to the consideration of the medico-legal questions involved in the study of this subject, we again encounter the often repeated situation in medico-legal investigations; that the nature and limitations of the condition under consideration are as yet unsettled questions, for it clearly appears in evidence from the works of the recent writers on medicine that we are even at the present time unable to give an undisputed definition to the condition under consideration. Yet the aim of law is exactness: and as stated by Spratling "its medico-legal importance requires that the physician should be able to define what the malady is."

Strümpell defines it as "a frequent disease, characterized by paroxysmal losses of consciousness, and often associated with violent general convulsions."

Starr that "it is a disorder of the control of brain energy, due to weak or defective organization, and due to any lesion which weakens this control."

Gowers that epilepsy "is a disease in which there are convulsions of a certain type, or sudden losses or impairment of consciousness: but in which the convulsions are not due to active organic brain disease, reflex irritation or abnormal blood states."

Dana that "idiopathic epilepsy is a chronic functional disorder characterized by losses of consciousness and usually by convulsions: that mental disturbances may accompany or take the place of the convulsions: that symptomatic epilepsy is a form in which periodic convulsive attacks are due to gross organic brain changes: that Jacksonian epilepsy is a form of symptomatic epilepsy usually, is characterized by periodic convulsions affecting only certain groups of muscles, and often unattended by loss of consciousness: and that hystero-epilepsy is not epilepsy, but a form of hysteria."

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Oppenheim that "epilepsy is a disease which in its developed form is characterized by attacks of unconsciousness combined with convulsions."

Spratling that "epilepsy is a disease or disorder affecting the brain, characterized by recurrent paroxysms which are abrupt in appearance, variable in duration but generally short, and in which there is impairment or loss of consciousness, together with impairment or loss of motor coordination, with or without convulsions."

It appears then that the definitions of Starr, Dana, and Spratling are the only ones of these which can be made to include all the types of convulsions now grouped under the title of epilepsy: and as will appear later some of the types of traumatic forms would be excluded by such definitions as those of Strümpell and Oppenheim.

The same conflict of opinion also appears in the views of these writers as to the nature of the disease.

Strümpell states that of the actual causes we know nothing: that Brown-Séquard produced epilepsy in rabbits by injury to the cord, medulla, and peripheral nerves: that the progeny of these animals suffered from spontaneous epilepsy: that Westphal produced the disease in guinea-pigs by blows on the head and considered the disease due to minute hemorrhages in the medulla and cord: that the fact that between the paroxysms the patient shows no signs of disease, shows that there is no permanent macroscopical lesion: and that the cause is an intermittent functional irritation of the cortical cells.

Hamilton, that the fit is due to altered cell metabolism from toxic substances accumulating in the blood.

Gowers, that in most cases changes in the brain are not visible to the naked eye and are therefore termed idiopathic.

Oppenheim, that Chaslin found a gliosis of the cortex, which is confirmed by Bleuer.

Starr that epilepsy is usually if not always an organic disease, that there is no essential difference between the Jacksonian and the idiopathic forms: that the difference between them is in the extent of the spasm, and if in the Jacksonian form the convulsions become general consciousness is lost: that the organic nature of the disease is shown in its occur-

rence in mal-development of the brain: that it is the exception that epileptics are normal mentally and physically: that idiopathic epilepsy is due to gross and microscopical brain defects: that the influence of a bad heredity shows an inherited organically imperfect brain: and that many of the cases of so-called idiopathic epilepsy, especially those characterized by uniform motor or sensory aura, originate in acute encephalitis in infancy or childhood.

Spratling states that no satisfactory classification can be based on the etiology but that based on the symptomatology is: that Esheverra held that epilepsy is not a morbid entity and there is scarcely a disease of the nervous system in which epileptic convulsions might not happen: that Féré says that the convulsions experimentally produced in animals are not epileptic: that the fundamental factor in epilepsy is a loss of control over the inhibitory power of the cortical nerve cell's function to store up energy: that the lesions include almost every possible change of the cerebro-spinal axis; that for the most part these lesions occupy a secondary place in the causation: that as epilepsy is now regarded as a cortical disease only those lesions which involve the cortex are of pathological primary importance: that the toxic, dynamic, and gliosis theories are each inadequate: that the convulsions are due to a diseased state of the cortical sensory elements: that the most marked changes are found in the cells of the second cortical layer and especially in the nucleus: that the poison in epilepsy is a nuclear poison: that the neuroglia hyperplasia is due to the cell destruction and irritation of the toxins: that the epileptic impulse is transmitted by other than the ordinary motor paths: that heredity and trauma alone are insufficient causes: that there must be a toxin present but its nature is unknown: and that the disease is engrafted upon a cortical organic cellular anomaly induced largely by faulty heredity.

It appears then that the weight of evidence is in favor of epilepsy being considered as an organic disease of the cerebral cortex, which weakens the inhibitory power of the cortical cells: and it is further alleged that this degeneration and epileptic discharge only occur from the presence of certain unknown toxins. It would appear then

if this latter theory is accepted that injury and disease of the cortex can produce epilepsy only in persons predisposed from the presence of this toxin. This theory can not as yet be accepted as proven until it can be shown that this toxin really exists, and that injury and disease are unable to produce the epileptiform state without its presence. Again, as it will afterwards appear, the assigned causes of epilepsy are very numerous and the number of epileptics very large, so it would appear as if this toxin must be present in a large percentage of human beings. The confusion which surrounds this subject has been increased by the use of the words "true epilepsy" by authors without their defining just what cases they would designate as such. At the present time it appears then that all our real knowledge consists in the fact that a certain type of convulsions can be originated from a great variety of causes, producing disease of the cerebral cortex.

The next question of importance is, Are the convulsions of which the plaintiff is alleged to suffer, of the epileptic type; and second, is there anything objective which would indicate whether they existed before the alleged injury? It is unfortunate that in these cases, we often have no guide beyond the history of the plaintiff, and difficult as it is to get a reliable history in private practice it is much more so in medico-legal work. This situation is often made worse by the confusion which exists in some medical minds between true and hystero-epilepsy. The accepted indications of a true epileptic seizure would seem to be its sudden onset, its occurrence during sleep, complete loss of consciousness in cases of general convulsions, the pallor followed by flushing or cyanosis of the face, the dilated and irresponsive pupils, loss of the corneal reflex, the turning of the eyes towards the side of a discharging lesion, the short sharp electric like nature of the spasms, the biting of the tongue in about half of the cases, the loss of the reflexes during several hours, the voiding of the urine and feces, the occurrence of petechiæ in the skin which do not fade for several days, a rise in temperature, a duration of only a few minutes, and a sequela of coma or headache. In answer to the second question, it is stated by Spratling "that in traumatic cases mental deterioration rarely occurs until after some years, and not then unless predisposition exists." It

can not be disputed then that if only a year or two have elapsed since the injury and the patient shows well-marked dementia, and especially if this is combined with the well-known stigmata of degeneration, that the disease existed before the alleged trauma. The epileptic condition, as has been shown, has many assigned causes and the medical witness must therefore be able to exclude these before he can say with reasonable certainty that the alleged injury was the producing cause. From the study of these alleged causes, it again appears that there is a conflict of medical opinion as to the power of some of these assigned causes to produce epilepsy. Epilepsy is one of the most common diseases, occurring in one of every five hundred persons, and the larger number being idiopathic. The influence of a neurotic heredity seems to be unquestioned. It is stated to be present in one-third of all the cases, and a direct inheritance in one-third of these. The effect of chronic alcoholism in the parents is also undisputed. Chronic lead poisoning in the parents is assigned by Oppenheim and Spratling, and syphilis by Gowers, Dana, and Spratling. Spratling also assigns tuberculosis in the parents as a predisposing cause, and also rheumatism, scrofula, rickets, morphine, diabetes, chorea, and degenerations of the ovaries and testes; but Gowers denies that phthisis or gout in the parents has any relation. That idiopathic epilepsy is a disease of youth and young adult life seems to be undisputed. In the vast majority of the cases it develops before thirty, and in three-quarters of the cases before the age of twenty. Defective brain development as a cause also seems to be undisputed. Persons so predisposed frequently show some of the stigmata of degeneration. It appears then that predisposed persons can either develop epilepsy spontaneously, or from causes which would not be sufficient in persons not so predisposed; but it also appears that a congenitally healthy brain may become so diseased from such causes as alcohol, that any added source of irritation may result in an outbreak and thus the apparent cause may not be the real cause. This first view is supported by Starr, who states that "the influence of a bad heredity shows an inherited organically imperfect brain, and the supposed causes can only be accepted when they produce organic brain disease: by Gowers, who states that "the causes are usually inadequate

except in cases with a powerful predisposition, and occurs as the result of a defective congenital brain development," and by Spratling "that in the majority of the cases both exciting and predisposing causes play a part."

The post-natal factors, outside of trauma, which are claimed as exciting or producing causes, can be divided into four classes: toxic, organic brain disease, mental, and reflex. Among the toxic causes the power of alcoholism to produce epilepsy appears to be unquestioned. Chronic lead poisoning is assigned by Oppenheim, but he is uncertain about cocaine, antipyrin, physostigmine, and chloroform. Spratling considers lead as a rare cause. The acute infectious diseases, especially scarlet fever, measles, and typhoid, are assigned as causes by all writers; and Starr believes that they do so by the formation of sclerotic plaques about the arteries. The power of gout is denied by Oppenheim and Gowers. Syphilis is considered as a common cause late in life by Oppenheim, Dana, and Spratling: but Strümpell considers that it has no direct connection with epilepsy. Starr and Gowers hold that syphilis produces epilepsy by causing disease of the vessels and membranes; but the latter considers it doubtful whether it can cause true epilepsy. Chronic nephritis is assigned as a cause by Oppenheim and Spratling, conditions of anemia and plethora by Strümpell, rickets by Dana, and Spratling holds that "derangements of the gastro-intestinal canal are of greater etiological importance than is generally supposed, that the gastro-intestinal cases are due to toxins either absorbed into the blood or act directly on the gastro-intestinal nerves: and that the influence of bad teeth and dentition is through the gastro-intestinal disorders so caused."

That organic diseases of the brain such as tumors, abscess, foci of sclerosis, hemorrhage, embolism, thrombosis, and arterial sclerosis are causes for convulsions of the epileptic type is undisputed: and after a time, it is stated by all writers, that these convulsions may resemble those of true epilepsy, but as already shown they do not define what the meaning of true epilepsy may be. Infantile encephalitis, meningitis, and post-natal palsies are as a rule undisputed causes, but Spratling holds that the polioencephalitis theory of Strümpell has received little corroboration.

Strong mental emotions, especially anger and terror, are undisputed exciting causes. Mental and physical over-work are also assigned by Strümpell and Spratling: but Starr considers the former as vague and uncertain.

The irritations which arise from scars or foreign bodies in any part are assigned as causes by Oppenheim, Golebiewski, Hamilton, and Dana: but Strümpell considers this etiological relationship as doubtful, Gowers as rare except in cases with a strong predisposition, Spratling considers it as possible but has never seen such a case, and Starr does not believe that epilepsy is ever so produced. Spratling adds that "the convulsions are epileptiform, like epilepsy, in the beginning": but as already shown he does not define what he means by true epilepsy.

Various diseases of the nose, larynx, uterus, intestinal worms, foreign bodies in the ears, ear diseases, errors of refraction, and carious teeth are assigned by Hamilton, Dana, Strümpell, and Spratling: but Gowers considers such causes as rare, and Oppenheim as not definitely shown. There seems to be no conflict of medical opinion that trauma is a competent cause for epilepsy, and according to Starr it is so in 11% of all the cases; but when the injury is so slight as not to produce fractures of the skull, severe cerebral contusion, or gross organic brain disease, it is a matter of grave doubt whether such trivial trauma can cause epilepsy in non-predisposed persons. Kirchhoff, Gowers, and Spratling state that cerebral concussion may produce a condition resembling true epilepsy: and this opinion is supported by Starr and Westphal who believe that in these cases, capillary hemorrhages occur, leaving a scar. Golebiewsky, Oppenheim, Bailey, and Hamilton concur in this view, but point out the fact that when epilepsy follows slight injuries there is usually a strong neurotic hereditary predisposition present, alcoholism, syphilis, or some nervous degeneration. Bailey holds that the general character of the convulsions would indicate that the disease was latent. Strümpell is somewhat conflicting in his statements, for he asserts that these cases may begin like genuine epilepsy, but are not genuine cases from the fact that there is an anatomical lesion and the symptoms may begin with a local spasm.

It appears then that in these cases assigned to slight cerebral contusions, in persons predisposed, where the symptoms were slight, where a considerable interval of time intervened between the injury and the onset of the epilepsy, and where the two were not connected by any mental or physical symptoms, that it can not be said that the disease would not have developed without the occurrence of the injury.

In cases with more serious injury to the head, as in fracture, especially depressed, or in injury to the brain, such as laceration, meningitis or hemorrhage, the evidence of the causal relationship of the trauma to the epilepsy seems to be undisputed: and the disease to be due to an irritation of the cortex from localized thickening of the cranium, splinters of bone invading the cortex, meningeal cicatrices or localized inflammations. Van Gieson has shown that from these lesions the membranes become adherent, wedged-shaped areas of connective tissue extend into the brain, the cortical cells degenerate, and areas of gliosis form about the cortical arteries. Bailey, while admitting that these lesions form a pathological basis for the disease, denies that they explain the paroxysmal nature of the condition. Bailey also considers that the severity is the most important element of the injury, and that severe injuries are frequently followed by epilepsy.

Traumatic epilepsy may develop at once after the receipt of an injury, or not until after a period of months or years: but the largest number of cases develop within a year. Hamilton considers cases which develop at the end of two or three years as a rule suspicious, but admits that genuine cases have occurred after a period of five years. During this interval it must be admitted that the patient may appear normal, but more often gives a history of cerebral symptoms, such as mental weakness, irritability, headaches, or hemiplegia.

In the cases which are assigned to cerebral contusion the convulsions are from the first, as a rule, of the grand mal type: and in those cases due to coarse organic lesions, the convulsions may also from the first be of the grand mal type, but more often they begin as localized spasms without loss of consciousness. After a time the convulsions grow more and more extensive, and finally become general, and then consciousness is lost. Such paroxysms may be followed by a

temporary loss of power in the muscles first convulsed, and if the lesion is progressive these muscles may become permanently paralyzed. From this study of these cases, it appears then: that for the medical witness to give an opinion with the reasonable certainty that the law requires, that the injury claimed was the cause of an epilepsy: that the plaintiff must be able to show that there were no evidences of the disease before the injury: the absence of other producing causes; that the disease occurred within a reasonable time after the injury: and that it was connected with the injury by some train of cerebral symptoms. For although it may be recognized in medicine that this connecting link may sometimes be absent, for the medical witness to claim that the disease is due to a thickening of the inner table or irritation of a splinter of bone, without any real knowledge that these conditions exist, is the purest speculation and has no place among the facts required in action at law.

Finally as to the future of these cases. This question, naturally from the conflict of opinion shown to exist concerning the previous questions, is also unsettled. So long then as we can not definitely say just which cases are to be included under the title of true epilepsy, opinions will differ as to the prognosis: and it is also evident that the general prognosis of the disease can not be in any individual case altogether admitted as satisfactory. It is evident then that our opinion as to the future must be influenced by the etiological class to which the plaintiff belongs and the individual surroundings which exist in his case.

As to the general prognosis of this condition: Oppenheim states that "epilepsy of itself does not shorten life," while Strümpell and Dana hold that it does to some extent: and Spratling points out that the condition of status which is often fatal occurs in one-third of the cases. Oppenheim states that recovery is rare, but more common than generally supposed, Dana states that from 5% to 10% recover, Spratling that 5% recover, and Strümpell points out that the natural history of this condition may cause it to disappear spontaneously and then return after many years. It is admitted by all writers that the prognosis is better in the grand mal type, in males, when there is a hereditary history, in the nocturnal

form, when the convulsions are infrequent (three or more weeks apart), when mental changes have not occurred, when a considerable period separates the aura and the fit, and most authors hold, when it begins before the age of twenty, and Spratling considers that a beginning between 15 and 20 is of good prognosis. Long duration and mental weakness are as a rule regarded as of bad prognosis: but Spratling holds "epilepsy is not infrequently a curable disease, irrespective of its duration, but recent cases respond twice as quickly to treatment." Hamilton states that in cases due to organic cerebral lesions, surgical procedures have shown good results. This is not in accord with the observations of the writer, for in the five cases of his operated upon, though the convulsions disappeared for several months they afterwards returned. The removal of the cause in reflex cases according to Oppenheim and Hamilton has resulted in a cure: but Starr denies that their removal ever cured the disease.

To illustrate the points of this paper I have collected from my hospital records the histories of thirty-eight cases in which an injury was assigned as the producing cause. These can be divided into three classes.

The first class contains those cases in which, although put down on the hospital records as traumatic, careful analyses reveals the fact that the assigned cause can not be said with any reasonable certainty to be the real producing cause.

There were ten cases of this class. In eight the injury was trivial and the immediate symptoms produced were also in seven. The ages at which the epilepsy developed in eight of the cases was from five to twenty years, the usual period for the appearance of the idiopathic form. The interval which elapsed between the injury and the epilepsy varied from two to twelve years. During this interval there was no evidence of any cerebral trouble. Three of the patients gave a family history of epilepsy in the parents, two of insanity, one of a discharge from the ear, and in this the spasms were at first confined to the opposite side of the body, and of the two cases which were over twenty years of age one suffered from alcoholism and the other from syphilis. In all the convulsions were of the grand mal type. In the second class are grouped those cases in which the injury to the head produced a well-marked state

of cerebral contusion, which seemed to be the only recognizable cause for the epilepsy. In all the injury to the head was severe, producing marked cerebral symptoms lasting from four hours to three days.

In six cases the first convulsion occurred within twenty-four hours after the symptoms of cerebral contusion subsided: and in the remaining four the interval varied from three days to four months. During this period the patients suffered from headache, vertigo, and mental confusion. All but three of the patients were over twenty-five years of age. The convulsions were all of the grand mal type. In none was there any evidence of other causes.

The third class contains those cases in which there were evidences of organic cerebral injury. Of these there were sixteen cases. Their ages varied from twelve to forty-four years. In all the injury to the head was very severe. In all the coma lasted from nine to twenty-four hours. In eight there were depressed fractures of the vault, and in three, of the base. Two had paralysis of an arm, and eight hemiplegia. In twelve the convulsions were at first of the Jacksonian type, gradually passing into grand mal, and in four of the latter type from the first. These four developed immediately, and in the others the length of time before the convulsions appeared varied from three days to four years.

It will be seen then that in this last group of cases there can be no question that there was a brain injury, and that it acted as the cause: and there was nothing in the histories of these patients to suggest the existence of any other etiological factor. If such injuries to the brain can produce epilepsy the trauma must be accepted as the sole cause: but if the theory that injury can only do so in persons predisposed by the presence of an unknown toxin in their blood, then injury can only be accepted as the exciting cause. It appears then that from a medico-legal standpoint our knowledge as yet will only enable us to state that certain injuries to the brain can excite epilepsy: and that the toxic theories can not be advanced as facts until there is some evidence upon which to base such statements: and if this latter statement be true it must be shown that this toxemia is present before the physician can say that the disease was the immediate and necessary result of the injury.