Section of Neurology

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DISCUSSION ON TRAUMATIC EPILEPSY

Major Hugh G. Garland: The term "head injury" covers a multitude of different pathological conditions which have trauma as a common denominator but which have little else in common, especially as regards their epileptogenic properties. It is time that some of these problems were settled, for besides their academic importance there is the return of war injuries, the increase in the number of road accidents (which has been followed by a multitude of medico-legal problems), and the new therapeutic approach to this type of epilepsy offered by modern neuro-surgery. A good deal of what is written about traumatic epilepsy is only the result of clinical impression and of speculation. Review of the literature on any aspect of epilepsy is no light task as it seems there have been at least 2,000 papers written in the last twenty years alone.

One fact is clear, namely, certain types of injury are much more likely to be followed by epilepsy than others. Outstanding amongst these is the gun-shot wound (G.S.W.). One of the best recent reviews of epilepsy following a G.S.W. is that of Ascroft (1941), a paper based on a follow-up of 317 cases from the records of the Ministry of Pensions; all these were casualties from the last war. In this group no less than 34 per cent. have developed epilepsy, a finding which is surprising in view of the previously quoted incidence of 4·5 per cent. in 18,000 cases of G.S.W. This latter figure is frequently found in the literature from 1921 onwards (Sargent, 1921; Stevenson, 1931), and is also obtained from Ministry of Pensions records. Ascroft's group was selected by him only to the extent that the notes were complete, and all his cases were presumably included in the larger series. The discrepancy is not due to the late onset of fits, as Ascroft found that most of the cases developed epilepsy within the first few months. Dr. Prideaux (1942) thinks it is partly due to the fact that the less severe cases had their claims settled early (7,600 by 1923) and presumably these cases were at that time suffering neither from epilepsy nor any other serious symptom. If this is the case Ascroft's group is selected to the extent that it consists largely of the more severe injuries; it is, however, almost certainly a maximal figure. Ascroft's series includes a variety of different pathological conditions and he has analysed his results accordingly. The effects of a G.S.W. are frequently localized and it is often the case that a severe local lesion of this type is not associated with loss of consciousness, though there is sometimes such loss after an interval (Eden and Turner, 1941); there may be a scalp wound only, or underlying this there may be severe cerebral contusion with or without fracture or penetration of the dura, or there may be a compound depressed fracture with laceration, in-driven bone, metallic or other foreign bodies, as well as sepsis; the end-result of such injuries also includes such differing conditions as scars, cysts, aerocele, abscess, &c. Ascroft's figures show that epilepsy occurred in 24 per cent. of those with scalp wounds only, but clearly there must have been brain damage in these cases which was unsuspected at the time. The incidence of fits was twice as high when the dura was penetrated. The presence of foreign bodies did not appear to increase the development of fits (unlike Wagstaffe's findings, 1928), but epilepsy was twice as common after there had been sepsis, whether the dura was opened or not. This high incidence of epilepsy after gross localized lesions is perhaps not unexpected when one thinks of the incidence of epilepsy in other gross and localized brain lesions of non-traumatic pathology. According to Penfield (1939*a*) fits occur in 44 per cent. of all supratentorial tumours. I think the incidence of epilepsy after recovery from a brain abscess is not less than 50 per cent., just as epilepsy is a common symptom in the acute stages of brain abscess, in fact a frequent presenting symptom in metastatic abscess. We can say at this stage that epilepsy following a G.S.W. is now well known. It is associated with a gross pathology, has opened up a promising surgical therapeutic field, and forms that subdivision of traumatic epilepsy about which there is a maximum of fact and a minimum of speculation.

Epilepsy following other varieties of head injury is much less clearly understood. These include injuries known as "closed" or "blunt" injuries and are those commonly seen

in civilians; they usually result from road accidents, from falling on the head and from blows from falling objects, usually of low velocity. This is again a mixed pathological group but certainly in cases without penetration of the dura there tend to be more diffuse and smaller brain lesions, gross focal lesions being very much less common. At the same time it is probable that a number of focal lesions are overlooked in this group owing to inadequate clinical examination in the acute stages. It is largely on this type of civil injury that most of the figures relating to traumatic epilepsy are based, and, especially in the older literature, there is little attempt to subdivide the cases according to severity or type of injury. That such injuries may be followed by epilepsy is accepted but to what extent the epilepsy is the result of the injury is less certain. The majority of these cases die a long time after the injury and after epilepsy has been present for years; post-mortem examination of the brain is rarely made and is usually inconclusive and, unlike the case of G.S.W., observation of the brain at operation is rare. The problems, therefore, have to be approached in other ways, the most important of which appears to be statistical analysis. Many series of cases have been followed up over long periods, but most of these are unsatisfactory, and it will not be until well-documented records of a large series of cases, such as that of Russell (1932), have been followed over years that accurate figures will be available. One of the most reliable of recent figures is that of Rowbotham (1942), who found epilepsy in 2·5 per cent. of 450 cases of injury of the "blunt" type. He regards this as probably the upper limit, the lower being Feinberg's (1934), who found epilepsy in 0·1 per cent. of a remarkable collection of 47,130 unselected cases of head injury. This latter figure is all the more striking in view of the fact that epilepsy is said to exist in 0·5 per cent. of the population of any country; if this be true Feinberg's figure can only show that a head injury confers a considerable degree of immunity to epilepsy. Russell (1934) found epilepsy in 3·5 per cent. of 200 cases within the first eighteen months of the injury and as Russell's and Rowbotham's cases were obtained from very similar sources this figure of about 3 per cent. is probably somewhere near the truth; but Kinnier Wilson (1940) quotes figures varying from 3·6 per cent. to 21 per cent., while Symonds (1935, 1941) regards epilepsy as being rare after this type of injury. These varying figures can only mean that the cases are in some way selected and that different authors are not discussing the same problem. I think it will be generally accepted that a single cerebral concussion is at least unlikely to be the sole cause of epilepsy, or even to be an important factor in its production, and such cases have no doubt diluted the figures of some observers. In his small series of epileptics Rowbotham found a significant preponderance of severe injuries. Here again, however, there is little agreement as to what constitutes a mild or a severe head injury and for this reason I do not think that further analysis of older published records will be of any value. Suffice it to say that the highest incidence claimed is about 20 per cent. and the lowest a good deal less than that of epilepsy in the general population; and that it is at least very probable that the first figure relates to a selected group of severe injuries and the second is diluted with many trivial cases.

Another method of statistical approach to the traumatic factor in epilepsy is to record the incidence of previous head injury in an unselected group of epileptics. Here again there are difficulties as it is usually impossible to confirm the history and, still more, the details of the accident. Textbooks usually dismiss this subject by saying that a history of injury occurs in less than 5 per cent. of all epileptics. I have analysed a group of 2,600 Service patients; these are selected in that they are all males (in whom a history of injury will be higher than in a mixed group) between the ages of 18 and 50, the majority between 20 and 35; they were all referred to me as falling in the "neuro-psychiatric" group. There were 244 cases of epilepsy, of which 77 per cent. appeared to be idiopathic (Table I). Of the remainder there was a history of head injury prior

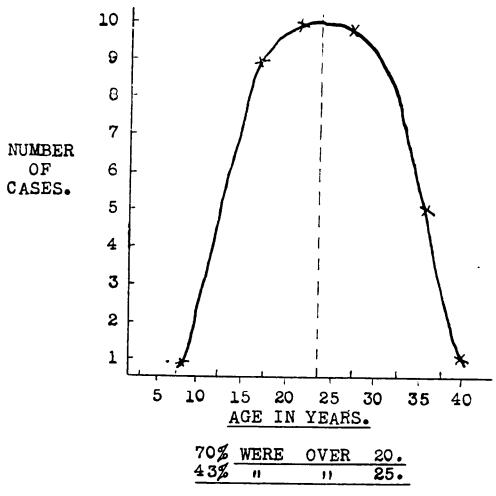
TABLE I.—CAUSATION OF EPILEPSY.

	Number of cases	Percentage
Idiopathic epilepsy	190	77
Mixed symptomatic group ...	15	7
History of trauma	39	16
Total	244	100

to the onset of fits in 16 per cent., the remaining 7 per cent. being cases of cerebral tumour, syphilis, or cysticercosis. This is a surprisingly high figure, which would have been lower had not leading questions been asked about previous trauma; unfortunately I have no suitable control group. It does not, of course, follow that the injury is in any way related to the epilepsy, but certain facts suggest that in many cases it was of importance. It is generally accepted that idiopathic epilepsy starts in childhood or adolescence and, while there may be some disagreement as to the upper age limit for

the onset of idiopathic fits, many put it at 20, and probably most at 25. In my group of cases 70 per cent. had their first fit after the age of 20 and 43 per cent. after the age of 25 (Table II). These figures suggest very strongly that most of the cases were not

TABLE II.



of the idiopathic type, and there was no evidence of any pathological condition other than the previous trauma. In idiopathic epilepsy there is a family history of fits in about one case in five; in 34 of my cases where the information appeared to be reliable there was a family history in only 4, but these figures are too small to be significant. Assuming that the injury has been a factor in these cases it is interesting to classify the injuries according to severity. Of the 39 cases one man had been a boxer and had had multiple slight injuries; I have subdivided the remaining 38 into 3 groups, choosing arbitrary limits; a post-traumatic amnesia (P.T.A.) of less than half an hour is called a "slight" injury, a P.T.A. of half to three hours "moderate" and more than three hours "severe" (Table III). It will be seen that there is a strikingly high incidence of

TABLE III.—SEVERITY OF HEAD INJURY.

Severity	Number of cases
Slight	
P.T.A. under ½ hour	2
Moderate	
P.T.A. under 3 hours	8
Severe	
P.T.A. more than 3 hours	28

severe injuries, which is again at least suggestive that the injury is an ætiological factor. It is generally accepted, and in the case of G.S.W. is certainly proved, that the interval between the injury and the onset of fits may be anything up to a number of years, but here again there is considerable disagreement. Foerster and Penfield (1930) in a short series of cases found an average latent period of five and a half years, with limits of five months and fourteen years. On the other hand Ascroft found the latent interval to be much shorter; he found the commonest onset was within the first month, though there were extremes of a few hours to twenty years, and Russell (1942) thinks the late development of epilepsy in closed injuries is rare, i.e., after two or three years. Table IV

TABLE IV.—LATENT PERIOD.

Years	Cases
Under 1	24
1– 3	4
4–10	4
Over 10	6

shows the interval in my group of cases; this varied from a few weeks to eighteen years, but the large majority started within the first year. With regard to the longer latent

intervals, it is interesting that Penfield (1939*b*) has found evidence that cerebral scars may sometimes increase in size over a number of years. I might add that an "average" figure representing the latent period, when taken from a group varying from a few weeks to many years, is of no significance whatever. In reviewing these cases from the point of view of whether the injury was a causal factor or not one can, therefore, say that in the majority of cases the age of onset of epilepsy was later than is usually seen in the idiopathic form, that the severity of the injury was in most cases considerable and that the latent period was usually short; a family history was uncommon and there was in no case evidence of any other further causal factor. It is interesting that trauma in this group of cases was much commoner than all other causes of symptomatic epilepsy, though this, no doubt, was largely the result of the age group under consideration.

Taking traumatic epilepsy as a whole, irrespective of the type of injury, it is interesting to study the clinical varieties of fits. The common idea that head trauma tends to be followed by Jacksonian fits is very wide of the mark and nearly all authors are agreed that much the commonest attack is a generalized convulsion. At the same time there is frequently an aura indicating the focus of onset, as in any other type of epilepsy. It has often been observed that other variants are uncommon, for example, Kinnier Wilson (1940) says: "It is curious how seldom *petit mal* or any epileptic variant is either ascribed to injury or evoked by it." Major fits occurred in all my cases and there was no example of Jacksonian attacks; psychomotor attacks co-existed with major fits in 2 cases and minor with major attacks in 3. The electro-encephalogram (E.E.G.) may ultimately solve this problem. For example I am not sure whether the characteristic E.E.G. of either *petit mal* or psychomotor epilepsy is ever seen after a head injury. If not it may be the case that trauma does not produce fits of these types and that in cases where they occur the trauma may not have been a causal factor, or may perhaps have precipitated fits in a potential idiopathic epileptic; but here again there is disagreement as to the frequency of variants and Symonds (1935) believes the minor attack to be a frequent early manifestation of traumatic epilepsy. This aspect of the problem needs a good deal of further investigation in the light of recent advances, as it may be the case that attacks previously called "minor," which presumably means *petit mal*, may be syncopal, or short attacks of vertigo. Stevenson (1931) actually states that "vertigo" is common in the latent period and Rawling (1922) found that "fainting" occurred in 16 per cent. of cases following G.S.W. The great importance of these diagnostic problems lies in the prognosis in cases where only attacks of a minor nature are occurring, but whatever the final verdict I think there can be no question that by far the commonest attack is a generalized convulsion. This again should not be unexpected as both *petit mal* and psychomotor attacks must be very uncommon in any symptomatic epilepsy. In this respect it is of some significance that such variants have never, I think, been produced by any form of electrical cortical stimulation nor in my experience do they occur after a water-pitressin test.

I have no personal records relating to multiple head injuries. There is no doubt that headache and other common post-traumatic symptoms tend to be more marked and to carry a worse prognosis if there have been previous injuries, and in this respect my one example of epilepsy in a professional pugilist is of interest. The "punch drunk" syndrome seems quite clearly to result from multiple minor injuries and in my case it seemed probable that the epilepsy was so caused.

To assess the influence of trauma in cases of epilepsy following head injury in civilians is to solve the problem whether such are not in fact cases of idiopathic epilepsy, or whether the trauma may have acted only as a precipitating factor. Because of the undoubted lower incidence of epilepsy in these cases, as opposed to G.S.W., it seems clear that pathological processes left behind by such an injury are not so epileptogenic as are the grosser lesions. The development of epilepsy may be entirely a question of the quantity of cerebral tissue damaged, the number of individual lesions, their size, or their position; or it may depend on some other unknown property of the lesion itself. The lesions produced by cerebral cysticercosis are so epileptogenic that there is no recorded case of them existing for very long in the absence of epilepsy (MacArthur, 1942); on the other hand such a gross lesion as disseminated sclerosis has little tendency to produce fits. It has been said that for a head injury to be followed by epilepsy there must be a certain type of brain and that unless the brain is of this type no amount of trauma will result in fits. This may or may not be true but a similar statement would apparently not be true in the case of cysticercosis. Similarly, such differing stimuli as electrical discharge across the frontal lobes, intravenous cardiazol and hypoglycæmia can produce convulsions in any type of brain. On the other hand Lennox, Gibbs and Gibbs (1939) state that there are three times more epileptics in the near relatives of all cases of symptomatic epilepsy than there are in those of non-epileptics (though I doubt whether

their cases include cysticercosis); this suggests an inherited tendency to epilepsy in all the symptomatic epilepsies. To sum up, therefore, it appears that in the civilian type of head injury epilepsy follows in about 3 per cent. of all cases, but this figure probably excludes simple concussion, and the highest incidence follows the most severe injuries. The fits are almost invariably major in type and tend to come on early, the majority starting within the first year. Whether these cases are due solely to the injury or whether there was a previous tendency to epilepsy, of the type of inherited background that occurs in idiopathic epilepsy, is not yet clear. It must always be remembered that both head injuries and epilepsy are common and that their co-existence will sometimes occur by chance. Further, recent work with the E.E.G. suggests an epileptic tendency in 10 per cent. of the population, which might well account for all the cases of epilepsy following civilian head injuries. At the same time in medico-legal work trauma is as important as a precipitant as if it were the only causal factor.

As traumatic epilepsy so commonly appears within a year of a severe injury it is only to be expected that in the majority of cases there will be such bridging symptoms as headache, dizziness and some form of mental incapacity. I have not sufficient information relating to such symptoms in cases where the epilepsy only supervened after years but I doubt whether their presence or absence is of much diagnostic or prognostic importance; Symonds (1941) finds epilepsy to be more common in those with persistent intellectual impairment or personality disorder, a state of affairs by no means limited to the most severe injuries.

Other important aspects of traumatic epilepsy include the significance of birth injury, which certainly accounts for some epilepsies of early life; the significance *qua* epilepsy of the site of the lesion; the prognosis, which is not always gloomy but which becomes worse the longer the latent period; and the co-existence of other symptoms, such as dementia, both in relation to diagnosis and prognosis.

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Mr. W. Grey Walter: The clinical statistics of traumatic epilepsy are contradictory and confusing, perhaps because the statistical method is not entirely suitable for studying this problem. It may be more profitable to consider in greater detail the physiological phenomena observed in each case. From the study of electro-physiological data, several facts are available. The first is that the electroencephalogram has shown that the convulsive seizure is only a part of the epileptic picture; persistent electrical abnormalities exist in many epileptics both "essential" and traumatic. The second fact is that electrical abnormalities seem to be much commoner in young patients, being almost invariable in children who have suffered birth injury. Thirdly, the electroencephalogram changes during the convulsion are always the same, irrespective of the cause of the fit. Records of the *petit mal* type are very rare in the traumatic cases, and it seems most probable that the true *petit mal* attack is never directly or entirely attributable to injury.

Apart from the short term, and temporary effects of head injury, there is no evidence of any specific effect of trauma in combination with convulsions on the electro-encephalogram, and there seems little hope, therefore, of distinguishing between the latent epileptic and the person in whom the injury was the direct cause of the condition—if such exists.

Further evidence is available from the electrical convulsion therapy, in the course of which it is possible to measure quite accurately with alternating current the convulsion threshold of thousands of patients. This threshold varies very widely from patient to patient, but is fairly constant in each individual. The variation is of the order of 1,000 per cent. Moreover, the threshold can be greatly raised by administering the con-

ventional anti-convulsant drugs. These facts suggest that convulsion thresholds vary enormously in each individual, and there is some reason for supposing that this is another aspect of E.E.G. data, leading to the idea of specific epileptic liability.

Two cases may illustrate this view. One is a woman who complained of attacks of twitching in the left arm and face which had developed after a comparatively trivial blow on the right side of the head. This persisted despite surgical removal of a part of the cortical area identified through the course of the abnormal electrical discharge which could be observed, during the attack, by the E.E.G. (fig. 1). The histopathological report

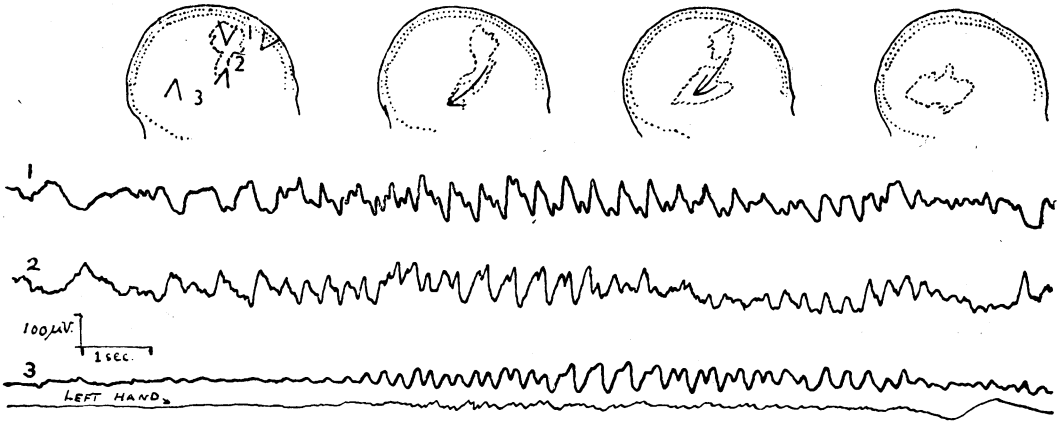

FIG. 1.

on the specimen removed suggested that the phenomena here might be due to minor interference with the blood supply to the area by perivascular gliosis, resulting in interference with oxygenation and removal of metabolites. The inference here is that, in spite of the local pathology, the genetic background was such as to discourage spreading of the electrical abnormalities beyond the neighbourhood of the mechanical interference, and, therefore, no generalized convulsion could develop. This would be the tentative explanation of so-called continuous partial epilepsy.

The second case had had a series of major fits in 1938; an E.E.G. at that time had shown bilateral synchronous slow waves of the epileptic type. In 1940 the patient was readmitted in status epilepticus, and shortly died. The last series of attacks were Jacksonian in type, starting in the right hand and face. At autopsy a small glioma was found in the left frontal lobe. Here the explanation would be that a strongly epileptic inheritance favoured the development of generalized convulsions at the earliest stage of the new growth.