THE ELECTRO-ENCEPHALOGRAM IN CHRONIC POST-TRAUMATIC STATES

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THE electro-encephalographic changes following a recent head injury have been described (Williams, 1941). The observations were made upon 74 cases of head injury selected from a total of 600 because the injury had occurred less than 20 days previously. From these observations it was concluded that the electro-encephalogram (e.e.g.) accurately reflects the abnormal cerebral state caused by a recent head injury, that the progressive changes are a direct indication of improvement or deterioration in this state, and that in the milder degrees of cerebral dysfunction it may demonstrate residual damage after all clinical signs have subsided. As symptoms may persist long after full recovery from a head injury might reasonably be expected the whole series of cases was used to observe the subsequent changes in the e.e.g. after the acute effects of the injury had subsided. It is the purpose of this paper to record the results and to relate them to some of the ætiological causes of the chronic post-traumatic syndrome.¹

Material

Six hundred admissions to a special head injury centre were used. They were unselected except that they were all men serving in the Army or R.A.F. who had symptoms referable to a head injury incurred before or during service. Their ages ranged from 18 to 60 years, but only 35 (5.8 per cent.) were over 35 years old. The investigations were made from 20 days to 12 years after the head injury, the time intervals being grouped below :

TIME AFTER HEAD INJURY	NUMBER OF CASES	TIME AFTER HEAD INJURY	NUMBER OF CASES
Up to 1 month	70	$\frac{1}{2}$ to 1 year	41
1 to 2 months	48	1 to 2 years	12
2 to 3 months	43	2 to 5 years	8
3 to 6 months	81	over 5 years	22

¹ It is difficult to give an exact name to the well-known group of symptoms, outstanding among which are headaches, giddiness, defects of memory, lack of concentration, fatigue, irritability, and changes in mood, which arise after head injuries. The old term "post concussional syndrome" has been discarded as inexact and the term "chronic post-traumatic state" is used because its limits are wide. The qualification of "chronic post-traumatic cerebral state" seems undesirable because the syndrome rarely follows injury elsewhere and it is not always due to demonstrable cerebral damage.

Method

The experimental procedure, including the types of electrodes used, their positions on the scalp, and connections to the recording system, as well as the form of clinical examination employed, has already been described (Williams, 1941). Electroencephalographic recording was made with three independent condenser coupled amplifiers activating three ink writing oscillographs (Grass) which recorded simultaneously, time being marked in seconds (Fig. 1).

Clinical data, which were subsequently correlated with the electro-encephalographic findings, were obtained upon the first 325 patients, and were tabulated under the following headings: age; time after the injury; duration of retrograde and post traumatic amnesia; presence of a cranial fracture, and of an open wound with dural penetration; residual symptoms; fits or faints; personality change; results of psychological examination; abnormal physical and psychological signs; previous head injuries; abnormal family or personal history, with special reference to epilepsy, migraine, constitutional instability, psychopathy or psychosis; the result of treatment, with return to duty or discharge from the service; assessment of the probable causation of the symptoms under three headings—predominantly organic, constitutional, or exogenous psychological factors; and finally the character of the e.e.g.

The assessment of the e.e.g. was based upon records obtained with the patients lying or sitting at rest with the eyes closed, standard amplification, recording speed, and amplifier characteristics being used. The clinical factors to be related to the e.e.g. were so numerous and variable that no attempt was made to quantitate the abnormality in the e.e.g. (Williams, 1941) in each case of this large series. Instead the proportion of abnormals in each group was determined, the norm being based upon the e.e.g. findings in two large control groups of subjects. The criteria of abnormality were similar to those described by Lennox, Gibbs, and Gibbs (1940).

Results

Controls

The first group of normals were 100 flying personnel of the R.A.F. who had undergone rigorous medical selection a few months previously and who had all satisfactorily completed their initial training and were undergoing operational instruction at the time of examination, when their physical health was excellent. Four of these hundred were considered to have abnormal e.e.gs. These four subjects, specimens of whose e.e.gs. are shown in Fig. 1, denied any abnormal personal or family history and may be considered normal by all the usual standards.

The second group consisted of 60 male and female members of the hospital staff. They had passed an army medical examination within a year previously, and their medical records were scrutinized to exclude evidence of abnormality in themselves or their families. In five of these (8 per cent.) the e.e.g. was considered abnormal. The lower percentage of abnormal records in the group of flying personnel than in the nursing staff of the hospital is consistent with their more critical selection. The percentage of abnormality in the hospital staff is of the same order as that obtained by Lennox, Gibbs, and Gibbs (1940) upon a series of non-epileptic controls. An attempt was made to maintain this a constant normal standard throughout the present investigation.

Patients with Head Injuries

Of the 325 patients with head injuries 162 (50 per cent.) had an abnormal

Fig. 1.—Representative records from three control subjects with abnormal electroencephalograms.

e.e.g. As this group of patients was unselected it included patients with injuries, both trivial and severe, as remote as 12 years before the accident. The age group of the patients was the same as that of the normal controls, so that this could not have modified the proportion of abnormal records.

Nature of the Abnormality.—A detailed description of the changes seen in the e.e.g. shortly after a head injury has already been given (Williams, 1941). This description also applies to many of the e.e.gs. seen in the chronic post-traumatic state. The abnormalities seen in this group were, however, less dramatic than those seen immediately after an injury, resembling the later grades of recovery, which have already been amply illustrated. The disturbance was almost invariably generalized, and consisted of low voltage 2 to 7 a second waves underlying the normal dominant frequencies of 8 to 12 and 15 to 25 a second (Fig. 2). After the initial severe abnormalities had subsided during the first few weeks, improvement slowed down and appeared to cease in many of the persistently abnormal cases. The outbursts resembling sub-clinical epileptic attacks encountered in the immediate post-traumatic period were not seen in non-epileptic patients, nor were very slow waves of high voltage encountered. Thus a uniform type of record, showing mild but persistent generalized abnormality underlying the usual normal frequencies was characteristic of the chronic post-traumatic state (Fig. 2).

Interval after the Injury.—The effect of time upon the incidence of abnormal e.e.gs. after head injuries is shown in Fig. 3. This graph shows that after a

Fig. 2.—Representative records from three patients with abnormal electro-encephalograms 2 months, 5 months, and 5 years respectively after a head injury.

Fig. 3.

60 to 70 per cent. abnormality rate in the first four weeks a decline occurs until ten weeks after the injury. The curve of abnormality then climbs until a further decline takes place at six months. Subsequently there is a steady climb to 12 months, when 55 per cent. of the patients had an abnormal e.e.g. In the main, however, the curve is in the neighbourhood of 50 per cent. and abnormality was still present in 47 per cent. of the cases investigated more than two years after the injury.

Unavoidable selection of cases by their admission to the special centre is responsible for some of the changes seen in the curve. Patients admitted immediately after an injury include many with slight cerebral damage without any prolonged sequelæ, in whom the e.e.g. returns rapidly to normal, concomitantly with the clinical recovery (Williams, 1941). These cases are not comparable with those admitted with symptoms a year after the injury. The climb in the curve between three and six months after the accident is partly due to the admission of many patients who have not recovered after treatment of the acute post-traumatic state in another hospital, many of whom had a severe injury, with a persistently abnormal e.e.g. Again, the level of abnormality throughout the curve is in part determined by the severity and persistence of the symptoms which led medical officers to refer the patients to the centre. Even so it is clear that years after a head injury between 40 and 50 per cent. of patients with symptoms severe enough to warrant admission to a special neurological hospital had an abnormal e.e.g.

Post-traumatic amnesia.—The relationship between the percentage of abnormal e.e.gs. and the length of post-traumatic amnesia is shown in Fig. 4. It is evident that there is a direct correlation between this duration and the percentage of abnormality. As the length of post-traumatic amnesia has a closer relationship to the severity of cerebral damage than any other clinical sign, it can be concluded that there is a positive correlation between the degree of cerebral damage and the persistence of abnormality in the e.e.g. This has special significance when it is considered that 67 per cent. of cases of severe head injury have an abnormal e.e.g., irrespective of the time which has elapsed since the injury. Although this general correlation is close, individual variations occur, and in one instance an abnormal e.e.g. was found without any amnesia.

Retrograde amnesia, whose duration has little relationship to the severity or type of injury was not found to correlate with the changes in the e.e.g.

Open wounds with dural penetration and cerebral laceration were found in 32 instances. The e.e.g. was abnormal in 30 (94 per cent.), the two patients with normal records having been injured 12 and 18 months previously. In these cases a focal abnormality corresponding with the position of cerebral laceration was invariable, although it was usually accompanied by a general disturbance.

Fracture of the skull without opening of the dura was seen in 63 cases, in 30 of whom (47 per cent.) the e.e.g. was abnormal. This figure was not significantly different from that for the whole series, so that the electro-encephalo-

Fig. 4.—The patients with head injuries have been divided into four groups in which the post traumatic amnesia was less than an hour, less than a day, and less or more than a week.

graphic evidence supports clinical experience that a fractured skull does not alone increase the hazards of a head injury.

Persistence of Symptoms.—All excepting 38 of the 325 patients had post-traumatic symptoms when the e.e.g. was recorded. These 38 were either symptom free or had a slight and intermittent headache which soon subsided, and only three (8 per cent.) of these had an abnormal e.e.g. This contrasts with 55.4 per cent. of those with persistent symptoms. It has been shown that in the immediate post-traumatic period the clinical recovery and improvement in the e.e.g. follow each other closely (Williams, 1941). While investigating the later stages of recovery a similar correlation was found, and it seemed that interruption of this parallel recovery had bad prognostic significance, for in a symptom-free patient with a normal e.e.g., recovery has already taken place while in a symptom-free patient with an abnormal e.e.g. either cerebral recovery is incomplete or the e.e.g. was already abnormal before the injury. In the first event return of symptoms may occur, and in the second the abnormality in the e.e.g. may be related to an abnormal personality (Lindsley and

Cutts, 1940). An instance of relapse in the first group occurred in Case No. 137.

Case No. 137.—A Gunner, aged 35, was admitted to hospital in coma a few minutes after a motor cycle collision which was subsequently found to have caused between 3 to 4 weeks' post-traumatic amnesia and 2 hours' retrograde amnesia. The next day he was semiconscious and was vomiting repeatedly. There were no signs of local cerebral damage and no wounds. He became restless as unconsciousness became lighter. A week after the accident he was still confused and disorientated, but was improving steadily. Ten days after he felt perfectly well and was anxious to return to duty. His mood was normal, but he was garrulous, euphoric, and uninhibited. On examination he was completely disorientated in time and place, his memory was very bad, and he could only retain 4 or 5 digits. His comprehension, judgment, and calculation were all poor, and he made 6 mistakes in the 100-7 test in 90 seconds. He had no insight and confabulated occasionally. There was slight left lower facial weakness, the tongue was protruded to the left, the tendon jerks were all brisk but symmetrical, all abdominal reflexes were absent, and both plantars were extensor. At this time the C.S.F. was normal, the pressure being 175 mm. of water. X-ray showed a short supratentorial fissure fracture of the occipital bone. At this time the e.e.g. showed widespread and very marked abnormality. There was a high voltage abnormal rhythm, with a frequency of 1 to 2 a second throughout both hemispheres. Underlying this were 3 to 7 a second waves of lower voltage. The disturbance was uniform and symmetrical, and was most evident in the posterior parts of the hemispheres (Fig. 5).

A month after the accident he was still confused and not fully orientated, but he had no abnormal physical signs. Six weeks after he was alert and correctly orientated, and his mental state was improving steadily. The e.e.g. then contained abnormal slow waves of low voltage in both hemispheres, showing a striking improvement upon the initial record. No focal area of abnormality could be found. After two months his mental state was normal except for instability of mood, as he easily became depressed and was unduly worried about the accident. The family and personal histories were good. He had been a steady sober man working as a brick maker before the war. He had a low average intelligence without any abnormal personality traits. His N.C.O. reported that the man was "an excellent and most enthusiastic worker, very popular and well thought of by everyone."

When the patient left the hospital three months after the accident he was considered to have recovered his normal personality and intellectual levels. His memory was thought to be as good as before, although he only retained 6 digits. His general information and calculation were average, his judgment was fair, and his insight and comprehension good. There were no abnormal physical signs. The neurological specialist reported at that time—"I think he has probably made an almost complete recovery, although he may be a little more emotional than previously. His memory is probably up to its former standard. He is fit for duty and I would be very surprised if he cannot carry it out as well as before."

After a month's convalescence he went home to 14 days' sick leave, during which he felt well except for occasional attacks of a postural vertigo which had not been present previously. He returned to his unit anxious to resume his work, but was exhausted by the journey and found that he was "not half the man" he had been. He was given very light work in the billet. The attacks of vertigo began to increase in number and severity, and he had a pressing feeling over the vertex. He rapidly found that he could not cope with his duties, he had no confidence in anything he did, and he felt ashamed of himself. He became anxious, depressed, and emotional. He lost weight and eventually broke down completely and was taken to the medical

officer, who referred him back to hospital a fortnight after resuming work. Psychological examination did not reveal any deterioration since previous testing, and physical examination was negative. He recovered and returned to duty, but after a further two weeks' trial on light work he again broke down, was readmitted, and as his capacity for any but the simplest life seemed limited, he was invalided from the service. Each time the e.e.g. was identical with that recorded before his first discharge from hospital, and it showed a persistence of the abnormal slow waves which had been present previously (Fig. 5).

Fig. 5.—Case No. 137. The high-voltage waves with a period of nearly a second recorded 20 days after the injury, have disappeared before discharge from the hospital. The normal dominant frequency has returned, but medium-voltage waves with a frequency of three a second occur frequently, especially in the parieto-occipital region. These abnormal waves were still present on readmission after relapse had occurred.

Comment.—This case report illustrates full clinical recovery after a prolonged period of confusion resulting from a severe head injury, which was followed by a relapse on two occasions when the patient returned to duty. The patient's past record was excellent. The only persistent abnormality was in the e.e.g.

When post-traumatic symptoms had persisted with an abnormal e.e.g., recovery very frequently took place, for cerebral damage giving rise to abnormality in the e.e.g. is usually reversible. As is common experience, the rate of clinical recovery from a head injury is variable, but while the improvement in the e.e.g. is following this recovery resolution appears to be the rule.

When, on the contrary, post-traumatic symptoms persisted with a normal e.e.g. full recovery was not so common. In this series 30 patients with normal

e.e.g.s. were invalided because the prognosis seemed so unsatisfactory. The reason for this is apparent if it is accepted that a normal e.e.g. invariably indicates the absence of damaged cerebral substance (Williams, 1941). Post-traumatic symptoms occurring with a normal e.e.g. are either primarily unrelated to cerebral damage or are due to permanent irreversible destruction of cerebral tissue, for absolute destruction of cerebral substance cannot be detected in the e.e.g. unless the involvement is sufficiently extensive to reduce the normal electrical activity. An example of such cerebral destruction with residual symptoms, a normal e.e.g. and incomplete recovery is illustrated by Case No. 331.

Case No. 331.—A Private, aged 25, fell from a lorry on Boxing Day 1940. When admitted to hospital he was in coma and had a spastic right hemiplegia. When he regained consciousness five days later the hemiplegia was unchanged and he was found to have a global aphasia and to be blind in the left eye. X-ray showed a horizontal fracture of the left frontal bone, without any superficial wound. He was admitted to the head injury centre five weeks after the accident, when the following physical signs were found: anosmia, absolute blindness with optic atrophy on the left, ptosis on the left and facial weakness of the right. There was a fairly severe right hemiplegia with spasticity, wasting, and limitation of movement and power, increased tendon jerks, diminished abdominals, ankle clonus, and a flexor plantar response. Sensation was normal. He had a predominantly motor aphasia which was severe enough to prevent psychological testing. The e.e.g. recorded from the right hemisphere at this time was perfectly normal, but there were abnormal slow waves over a very large area of the left hemisphere. Their period was 1 to 2 seconds, and their voltage was moderately high. They were most evident in the parietal and parieto-occipital region (Fig. 6). The C.S.F. was normal in all respects. He improved

Fig. 6.—Case No. 331. *Electro-encephalogram.* Persistent one-a-second waves were recorded from the left parietal and occipital region 5 weeks after the injury, although records from the right hemisphere were normal. Twelve weeks after the injury, records from identical positions in the left hemisphere were quite normal.

steadily until three months after the accident, when the power and movement on the right side enabled him to walk without support and to feed and dress himself, while the aphasia slowly resolved. At this time intellectual testing showed that he was childish and facile, powers of retention and recall were defective, and his ability to calculate was poor. He was well orientated, had fair insight, but his judgment was poor. An encephalogram showed the anterior horn, body, and temporal horn of the left lateral ventricle to be dilated in all directions, the right side of the ventricular system being normal in size and position (Fig. 7). It was concluded from this and from an increase in the air over the left side of the convexity that there was severe left frontal and temporal cortical atrophy. Specimens of the encephalograms are shown in Fig. 7. Repeated records had been made and had by this time become perfectly normal, all evidence of local disturbance on the left having disappeared (Fig. 6). The patient was observed for over four months after the accident, but at that time there was residual left-sided spastic weakness, dysphasia, and considerable intellectual impairment. Because of the incomplete resolution of symptoms and evidence of gross destruction of cerebral substance the patient was invalided from the Service.

Comment.—This case showed persistence of signs of local cerebral dysfunction with corresponding ventricular dilatation, indicating gross destruction of cerebral tissue. The e.e.g. at first showed local abnormality corresponding to the expected site of cerebral damage. This abnormality disappeared, partly perhaps owing to resolution, but largely to absolute destruction of the abnormal tissue. The dissociation between the full recovery in the e.e.g. and persistent symptoms and signs was thought to indicate irreversible destruction of cerebral substance, and an encephalogram subsequently confirmed this opinion.

An example of failure to recover in a patient with persistent post-traumatic symptoms without abnormal physical signs and with a normal e.e.g. is as follows :

Case No. 174.—A Sapper, aged 25, was knocked down by a car and remained semiconscious for two days. Post-traumatic amnesia was later found to be four weeks, and retrograde amnesia a few seconds. He had bilateral extensor plantar responses on admission to hospital. Two weeks after the accident he was restless, grossly confused, and disorientated. There was confabulation and perseveration, he occasionally had organized visual hallucinations, and was incontinent of urine and faeces. There were mild signs of pyramidal damage on the left, with an extensor plantar response, and he had a nominal dysphasia. Lumbar puncture revealed no abnormality except for 80 mgm. of protein. X-ray of the skull was normal. The patient improved very slowly, and a month after the accident was still confused and disorientated. The pyramidal signs had disappeared at this time, but he was still dysphasic. Seven weeks after the accident there was very slight dysphasia. He could retain seven digits but confabulated when attempting to recall simple stories. He had no judgment or insight, could only attempt the most elementary calculations, and made absurd mistakes on being questioned in general knowledge. He slowly improved and ultimately became able to live a normal life, to look after himself, and to carry on spontaneous simple conversations. He remained without insight, and three months after the accident, when he went to a convalescent home, he still had a grossly defective memory, little judgment, only limited knowledge of current affairs, and no insight. He maintained that he was perfectly well. After a month's convalescence he was so anxious to return to duty that he was graded down and returned to simple duties after a month's sick leave. Two weeks after this he was readmitted to hospital, as he was unfit to carry out his light duties in the cookhouse. He complained that the noise of the kitchen had given him intense, continuous headache with tinnitus, and

Fig. 7.—Encephalography.—Twelve weeks after the injury shows uniform dilatation of the left ventricular system, without displacement of the third ventricle, with a normal lateral ventricle on the right. There is cortical air in the left side, but not on the right. The pictures are taken in the straight antero-posterior and half-axial postero-anterior positions.

he said he had become nervous and anxious. He had persistent feelings of unreality which were often so intense that he thought himself dead. He was irritable, was liable to violent outbursts of temper, and still had occasional visual hallucinations. When readmitted, six months after the injury, he still had no insight. His judgment was defective, and he remained euphoric and wanted to return to duty. He could retain six digits, but could not recount short paragraphs from newspapers, and was unable to obey a series of simple commands. There was obviously mild dementia and considerable personality change. He was invalided from the Service as his condition had been absolutely stationary for three months. Repeated e.e.gs. were normal both before his return to duty and on re-admission to hospital six months after the injury.

Comment.—A man who improved slowly after a severe closed head injury retained considerable intellectual defect with poor memory, little judgment, no insight, and a deterioration in his personality with irritability and outbursts of temper. His condition appeared to be quite stationary six months after the injury. The e.e.g. was originally abnormal, but became normal three months after the injury and did not change subsequently. The disparity between the clinical state and the electroencephalographic findings suggested considerable permanent destruction of cerebral substance. His ultimate degree of disablement supported this view.

Previous Head Injury.—Forty-two patients had had a previous head injury without residual symptoms. Twenty-two of these (52 per cent.) had an abnormal e.e.g., the changes in six being pathognomonic of epilepsy. This series is small, but it suggests that unless epilepsy supervenes, a previous head injury without sequelæ does not appreciably modify the e.e.g. This observation is supported by the presence of normal records in 92 per cent. of the symptom-free patients in this series.

Abnormal Family History.—Evidence of emotional instability, psycho-neurosis, psychosis, mental defect or epilepsy was present in near relatives of 73 of the patients. The disorder was considered significant in parents, siblings, parents' siblings, or first cousins. Only 29 (40 per cent.) of these 73 patients had abnormal e.e.gs. and eight of these showed changes characteristic of epilepsy. In view of the low incidence of epileptic changes in the e.e.gs. of the present series of cases (*vide infra*) it is probable that constitutional predisposition was largely responsible for the changes in the eight patients with epileptic records. Excluding the specifically epileptic changes, therefore, only 28 per cent. of this group had abnormal e.e.gs. compared with 50 per cent. of the total. This difference is statistically significant and may have a simple explanation. These 73 patients who came from constitutionally inferior families were probably more liable to have persistent symptoms after a head injury which would not cause sequelæ in more stable patients. Consequently many of them had comparatively trivial injuries, which did not give rise to abnormalities in the e.e.g. Lennox, Gibbs, and Gibbs (1940) have shown that abnormal families have a higher proportion of members with abnormal e.e.gs. than have normal families, so that some of the 29 patients undoubtedly had an abnormal e.e.g. before the head injury.

Abnormal Past History.—Evidence of nervousness, mood swings, psycho-neurosis, psychopathy or psychosis was present in 57 patients before the injury; 29 of these (51 per cent.) had an abnormal e.e.g. after a head injury.

As this proportion is the same as in the total series it might seem that the abnormal past history has no significance, but Lindsley and Cutts (1940) have shown that the e.e.g. is abnormal in a higher proportion of such subjects than in normals so that the pre-traumatic e.e.g. was probably abnormal in some of these subjects, the abnormality resulting from the injury being in fact less frequent than in the total group. In attempting to decide if an abnormal e.e.g. resulted from the injury, records were repeated at long intervals. If the character of the record had not changed it seemed more likely that the abnormality was permanent and so might have been present before the injury.

Disposal of Patients.—Two hundred and seventy-nine of the patients had left hospital when this survey was made. Of 87 who were invalided from the services 67·8 per cent. had an abnormal e.e.g., while only 25·5 per cent. of 192 who returned to duty showed abnormality. It cannot be concluded, however, that a quarter of those returning to duty did so with an abnormal e.e.g., for in most cases the patient went to a convalescent home and then to sick leave after the e.e.g. had been recorded.

Causation.—In attempting to assess the predominant ætiological factor in each example of the chronic post-traumatic state it was realized that in many cases several factors were present simultaneously. It is probable that in some the assessment may have been too strongly biased in one direction, and in others it was even found impossible, but the series was thought to be large enough to minimize individual mistakes. The ætiological factors responsible for the syndrome were divided into three broad groups of organic damage, constitutional predisposition, and exogenous psychological causes, on the basis of the patients' past record, family history, the relationship of the severity of the injury to the resulting symptoms, and the part played by environmental factors in perpetuating the symptoms. An opinion was given upon 316 of the cases; in 207 the symptoms were thought to be due primarily to organic damage; 50 patients appeared to be constitutionally predisposed, and in 58 the symptoms were thought to be mainly due to an exogenous neurosis. In the primarily organic group 114 (55 per cent.), in the constitutionally inferior group 19 (37 per cent.), and in the neurotic group 12 (20 per cent.) showed abnormality in the e.e.g.

Epilepsy.—Forty-eight of the 325 patients had one or more epileptic attacks after the head injury; 32 had grand mal, and in all but two the attacks fell into the group of "immediate post-traumatic epilepsy." Seven patients had a history of fits or faints prior to the injury. Thirty-one of this total (65 per cent.) had an abnormal e.e.g. and the changes seen suggested epileptic outbursts in 20 (42 per cent.); eight of the epileptic patients had a family history of epilepsy, and all but one of these had an epileptic type of abnormality in the e.e.g.

Excluding the epileptic type of change seen in the e.e.g. in the immediate post-traumatic period (Williams, 1941) epileptic disturbances occurred without clinical fits in only seven out of 277 patients (2·5 per cent.), thus the percentage of abnormal e.e.gs. (65 per cent.) in the patients with fits, the incidence of characteristically epileptic changes (42 per cent.) in those patients and the

incidence of similar changes in the non-epileptic patients (2.5 per cent.) are all similar to the data relating to idiopathic epilepsy.

Less than a year has elapsed since this series of cases was commenced, so that prognosis cannot be hazarded. It has been shown that the incidence of epileptic changes in the e.e.g. in the epileptic and non-epileptic patients was the same as in similar groups of subjects without head injuries. The character of the changes seen in the e.e.g. record in traumatic epilepsy appear to be the same as in idiopathic epilepsy.

Discussion

This present series does not represent all cases of head injury, for most subjects of concussion or even moderately serious head injury recover without any sequelæ. All except 11 per cent. of this series had sequels of the injury at the time of examination, although the majority (78 per cent.) were investigated after the acute stage had passed. The patients were mostly admitted to a special centre because a severe injury had been sustained, or because recovery was not satisfactory ; but although there was a preponderance of the more severe degrees of cerebral damage the series does represent the group of patients with persistent post-traumatic symptoms. This group presents problems in ætiology, management and prognosis which are not encountered in the milder post-concussional states, so that although a 50 per cent. morbidity in the e.e.g. in no way represents the figure for all head injuries, it is obviously important in relation to these aspects of the chronic post-traumatic syndrome. The importance is enhanced by the persistence of the abnormality for years after the injury.

There are two possible explanations of this persistent abnormality. It is either a direct result of organic cerebral damage, or it represents an abnormal pre-traumatic e.e.g. in a subject in whom latent abnormality caused symptoms to persist after a head injury. If the second hypothesis were correct the cases would represent the 8 per cent. of clinically normal subjects with abnormal e.e.gs. The results of this investigation show that the proportion of abnormal to normal e.e.gs. was directly related to the severity of injury ; it was higher when the symptoms clinically seemed to be due to cerebral damage than in those patients in whom endogenous or exogenous psychological factors were present ; it was very high when actual cerebral laceration had occurred, while it was low in the absence of symptoms or signs of cerebral damage. Again, the percentage abnormality was not influenced by fracture of the skull, known to be unrelated to the degree of cerebral trauma, nor was it increased by a previous head injury without residua ; lastly, it was no higher in those with a morbid personality or in members of constitutionally inferior families. All these results indicate that actual organic cerebral damage is mainly responsible for the abnormal e.e.g. seen in the chronic post-traumatic states.

It is likely that some of the patients whose symptoms seemed to have been constitutionally predetermined had abnormal e.e.gs. before the injury (Lindsley and Cutts, 1941), but this small number cannot have materially affected the total. It therefore follows from the results obtained in the acute post-traumatic

period (Williams, 1941) that abnormality persisting in the e.e.g. after a head injury is usually due to continued abnormality of cerebral tissue which has resulted directly from the injury. This fact has importance in the management of these cases, for it places a syndrome which superficially has the characters of a "functional" state on to an organic basis. It explains the recurrence of symptoms in some patients after an apparently satisfactory clinical recovery, for the persistent cerebral abnormality reflected in the abnormal e.e.g. cannot be detected by the psychological tests used in routine examination. The residual cerebral damage does not become evident until the patient has to respond to the relatively severe demands of daily employment. Thus undue fatigue, failure of concentration, memory defect and mood changes are related to a loss of "functional reserve" in an organically abnormal brain. This conception is of course an old one, but the changes in the e.e.g. give objective evidence of the organic basis of the chronic post-traumatic syndrome. The evaluation of a normal record in the chronic post-traumatic state is more difficult, for here the symptoms may have arisen as a result of irreversible destruction of cerebral parenchyma or through the perpetuation of symptoms of cerebral damage in an abnormal personality.

It may be impossible on clinical grounds to distinguish the ætiological factor responsible, but whatever the cause of the changes described, the application of the e.e.g. to the clinical data may be summarized as follows :

1. Clinical recovery is followed closely by improvement in the e.e.g. and so long as the improvement in the clinical state and the e.e.g. run a parallel course the outlook is good.
2. Abnormality usually persists in the e.e.g. after clinical recovery appears complete in subjects whose progress is satisfactory.
3. Persistence of abnormality in the e.e.g. usually indicates residual cerebral dysfunction which may not be demonstrable clinically.
4. A normal e.e.g. indicates the absence of abnormal cerebral tissue, so that when associated with full clinical recovery, relapse is unlikely. When symptoms persist in the presence of a normal e.e.g. the ultimate prognosis may be bad, for complete resolution or complete destruction of abnormal cerebral tissue has occurred.
5. No difference is apparent in the e.e.g. of traumatic and idiopathic epileptics.
6. Prediction of traumatic epilepsy does not appear possible by electro-encephalographic examination during the period of recovery from a head injury.

Summary and Conclusions

Electro-encephalograms (e.e.gs.) were recorded on 600 service personnel with head injuries. The interval after the injury varied from a few hours to 12 years, all types of injury being included. The records were divided into normal and abnormal groups on the basis of 160 controls, and the results correlated with detailed clinical data in the first 325 cases.

Fifty per cent. of the patients with head injury had abnormal e.e.gs. This high percentage abnormality persisted for many years after the injury.

There was a positive correlation between it and the severity of the injury, the persistence of symptoms, and the presence of dural penetration.

Simple fracture of the skull, an abnormal family history, an abnormal personal history, and a previous head injury without residua did not increase the percentage abnormality.

Of 87 patients who were invalided from the services, 67 per cent. had an abnormal e.e.g., but of 192 who returned to duty only 25.5 per cent. were abnormal while in hospital.

The cause of the symptoms was assessed. In 207 patients it was thought to be primarily due to organic damage, and in 55 per cent. of these the e.e.g. was abnormal. In 50 who were constitutionally inferior it was abnormal in 37 per cent., and in 58 whose symptoms were due to an exogenous neurosis 20 per cent. were abnormal.

It was concluded that:

(1) After a head injury an abnormal e.e.g. usually indicates organic cerebral abnormality resulting directly from the injury. This abnormality is present in half the cases of the chronic post-traumatic syndrome.

(2) Clinical recovery and improvement in the e.e.g. are usually closely related, and so long as the relationship holds, the prognosis is good.

(3) Abnormality may persist in the e.e.g. in any one patient after clinical recovery appears complete. This indicates residual cerebral dysfunction which is not demonstrable by the usual methods of psychological investigation. It is usually followed by complete recovery, but the abnormality may explain the relapse of some patients after they have returned to their employment.

(4) A normal e.e.g. after a head injury almost invariably indicates absence of abnormal cerebral tissue which may be due either to full resolution or to complete destruction of damaged cerebral substance. When a normal e.e.g. is associated with full clinical recovery prognosis is consequently good, but when accompanied by persistent symptoms the ultimate prognosis may be bad.

(5) There does not appear to be any difference between the e.e.g. of traumatic and idiopathic epileptics, and prediction of traumatic epilepsy does not seem possible by electro-encephalography during the period of recovery from a head injury.

(6) The e.e.g. appears to reflect the degree of cerebral dysfunction resulting from trauma with considerable accuracy, and it has a useful place in the management of cases of head injury when used in conjunction with other methods of clinical examination.

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