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EPILEPSY AND GUNSHOT WOUNDS OF THE HEAD.

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SUFFICIENT time has elapsed since the termination of hostilities to reveal the later effects of gunshot wounds of the head. Of these sequelæ epilepsy is one of the more important. The figures, so far available from the records of the Ministry of Pensions, show that only a small percentage of those who have received such injuries have developed epileptic attacks—out of 18,000 cases of gunshot wound of the head, 800, or rather less than 5 per cent, have become epileptic (Sargent¹).

In the absence of particulars of the nature of the head injury it is difficult to say whether these figures refer to all varieties of gunshot wound of the head, or only to those of a serious nature in which the skull has been penetrated. They may, however, be compared with some given by Allen,² who mentions that of 167 cases of injury of the skull received in the American Civil War, 23, or 13·7 per cent, were on the Pensions list as suffering from epilepsy.

In the Franco-Prussian War of 1870 there were 571 cases of recovery from gunshot wounds of the skull; of these, 25, or 4·3 per cent, developed epilepsy—a figure remarkably similar to that from our own Pensions list.

Holmes and Sargent³ investigated the effects of gunshot wound of the head in 610 cases between two and eighteen months after the infliction of the wound. They eliminated all cases of scalp wound, and others in whom the nature of the wound was uncertain. On this

basis they found that 37 cases, or 6 per cent, were suffering from fits, but whether Jacksonian or generalized epilepsy there was no means of knowing. Their conclusion was that epilepsy following gunshot wound of the head was much less common than had been foretold.

On the other hand, Rawling,⁴ who collected 452 cases of all varieties of gunshot wound of the head—scalp, penetrating, non-penetrating, and perforating wounds and fractured base—reports that 25 per cent of these developed 'fits' as a remote effect of the head injury. In his series, the generalized fit was more common than the Jacksonian type of seizure, and the cases were observed at a considerably longer period after the wound than those of Holmes and Sargent.

From these figures it would appear as if the frequency of traumatic epilepsy following gunshot wound of the head varied to a large extent with the interval which had elapsed between the infliction of the wound and the date of examination, as well as upon the nature and severity of the wound itself.

From my own observations upon officers who had received penetrating wounds of the head, and who were seen at an Appeal Board for the assessment of their disability, a third condition of variability might be added, viz., the recognition of minor or 'petit mal' fits and epileptic equivalent attacks of a vertiginous character. These are readily overlooked, and may not be referred to by the patient unless inquiry is made into their occurrence. I therefore incline to the view that epilepsy (using this term in the sense used in the description of the idiopathic disease) is more frequent after gunshot wound of the head than the earlier figures would lead us to expect.

In civil practice an injury to the head is a commonly assigned cause of epileptic fits. In some of these cases there is little, if any, relation between the assigned cause and the malady; in others the onset of the epilepsy is correctly attributed to the trauma.

In the war cases the relation between the trauma and the epilepsy is more definite, and the cases may be divided into two main groups:—

1. *Those with a definite organic lesion of the skull, membranes, and brain* (penetrating wounds). Epilepsy following a penetrating wound of the head may be either partial, focal, or Jacksonian; or the seizures may conform to those of generalized idiopathic epilepsy with loss of consciousness. It will be shown here that focal or cortical epilepsy following gunshot or shrapnel wound of the skull and brain is relatively rare, occurs early, and tends to disappear, while the generalized type of epilepsy is common by comparison, of later development, and runs a chronic course.

2. *Those without obvious evidence of injury to the skull (scalp and*

non-penetrating wounds). There are numerous cases in which a minor head trauma has been followed by fits of a generalized epileptic character within a short period of the injury and without a history of previous epilepsy. *X-ray* examination of the skull may not reveal fracture of the bone, and yet a short period of loss of consciousness may have followed the infliction of the wound. In the observed cases the first fit followed within a short period—a month or two—after the trauma, and the subsequent course of the disability has been that of generalized epilepsy.

Of somewhat similar character, and probably classifiable in this group, are those cases of epilepsy which follow upon aeroplane crashes and falls on the head from horseback, motor cars, etc. Several such cases have come under observation, and have been attributed to fracture of the base of the skull, of which, however, no direct evidence has been obtained by *x-ray* examination. It is impossible in these cases to eliminate a cerebral concussion, local contusion, or laceration, which may have been the starting-point of the disability. Jefferson,⁵ in an investigation into the early effects of gunshot wounds of the scalp, without fracture of the calvarium, reports a series of 54 such cases, of whom 17, or 31.5 per cent, showed neurological evidence of local cerebral contusion, and all but five a cerebral disturbance of some kind. Trotter says that it is quite unimportant whether the bone shows evidence of injury on *x-ray* examination. The bone indeed may have recovered its shape completely and have sustained no fracture, and yet have caused a distinct local lesion of the brain.

In cases which have been submitted to operation, a fracture of the inner plate of the skull, an extradural clot, or a contusion of the subjacent cortex has been observed; and in old-standing cases an ill-defined cystic degeneration, a patch of sclerosis, or of adhesion between the membranes and the brain has been detected.

TRAUMATIC EPILEPSY.

This is defined as a disability characterized by seizures having the features of ordinary generalized epilepsy, occurring as a late phenomenon in consequence of an injury to the brain and its membranes, following a gunshot, shrapnel, or other wound of the skull. Under the term are included all fits, seizures, or attacks recognized as of an epileptic character, whether of the major, minor, or psychic variety. The observations were made solely upon officer patients, who came before a medical board for the assessment of their disability. They had been certified by competent medical authorities as suffering from traumatic epilepsy. The majority were observed upon one occasion only, but a full record of the medical history was before the board at the time of the examination.

Generalized traumatic epilepsy may follow a wound of any region of the brain except the cerebellum. Of the 38 cases recorded in *Table A*, 10 were wounds of the frontal lobe, 16 of the parietal, 9 of the occipital, and 3 of the temporal lobe. The parietal region provides the greatest number of cases, and there is reason to believe that wounds of this region have a greater tendency towards epilepsy, quite apart from the greater frequency of the wounds in this locality. Behague⁶ indeed states that twice as many parietal wounds are followed by epilepsy as occipital wounds.

Table A shows also the great frequency of associated paralytic and other symptoms. In the case of the frontal injuries, impairment of memory was a prominent feature, while certain local paralyses, more especially from injury of the optic and oculomotor nerves, were observed. In the parietal region, with only two exceptions, all the cases showed some degree of motor or sensory paralysis, or a combination of motor and sensory paralytic symptoms. The sensory defect consisted mainly of some degree of 'sensory hand' characteristic of cortical sensory paralysis. Nine out of ten occipital-lobe cases presented some degree of hemiopic visual defect. In two of the three cases of wound of the temporal region, deafness of the corresponding ear was present.

A gunshot, shrapnel, or other wound of the head of a penetrating character may be followed at once by generalized epileptic fits, or by unilateral fits in the limbs on the side opposite the injury. The local lesions vary considerably, and consist of fracture of the bones of the skull usually associated with contusions of the cerebral tissue and œdema, and sometimes followed by sepsis and abscess formation. In other cases a hæmatoma may be present. In less severe cases a localized contusion or laceration of the brain may be the immediate consequence of the wound. X-ray examination of the head in some cases of epilepsy reveals the presence of fragments of metal deeply implanted in the cerebral hemispheres. In one case (No. 25) a fracture of the inner table causing contusion of the cortex was followed by epilepsy, which persisted until a trephining operation was performed three years later.

Neither the extent of the bony lesion nor the severity of the original cerebral trauma appears to influence the development of the seizures. In the majority of cases the original trephining or subsequent operation had left a large gap in the skull, with or without visible pulsation or impulse on coughing. The exceptions to this were seen only in cases of 'gutter wound', or in 'through-and-through' wound.

In most of the cases of wound, especially of the parietal and occipital regions, the neurological examination showed evidence of

destruction of cerebral tissue, leading to hemiplegia or hemianopia, with extensive scarring or cicatricial adhesion of the brain to the membranes and the scalp.

It was especially striking in how few of the observed cases was there a history of convulsions at the time of the infliction of the wound. Although cases with a history of convulsions immediately following the trauma in some instances develop traumatic epilepsy later on, such a sequence is rare.

Adie and Wagstaffe, in a report to the Medical Research Committee (1918, No. 1) upon the immediate effect of gunshot wounds of the head, refer among other matters to the development of fits. They state that fits occur immediately in only 5 per cent of cases which recover, including all varieties of gunshot wound. These fits are observed during the first week, they never spread out over a period longer than three days, and there is an exact correlation between the site of the wound and the point of origin of the fit. The authors conclude that these cases are not more likely to develop traumatic epilepsy later on than other cases of head wounds.

One of the outstanding features therefore of traumatic epilepsy is the development of the disease some months after the receipt of the trauma, and the absence of any definite or constant relation between the injured region of the brain and the method of onset of the major fit.

The *time of onset* of the epilepsy after the receipt of the wound is a point of interest. Of the 36 cases in which the date of the first seizure was definitely known, fits commenced in 21 at some period between the second and the twelfth month; and in 25 during the first year; in the remainder the onset was at any time from the end of the first year up to two and a half years after the wound. In the majority of cases, therefore, epilepsy develops before the lapse of twelve months; but the 'latent period' may persist for much longer.

As regards the *frequency* of the seizures, there is no essential difference between them and those of ordinary epilepsy, although in the traumatic form the appearance of a fit at long intervals is probably more common than those of frequent recurrence.

In *character* the fit resembles that of ordinary generalized epilepsy, and all varieties of seizure may be seen, from a momentary 'absence', 'lapse', or epileptic vertigo up to the fully-developed seizure of major epilepsy with fall, tongue biting, and unconscious urination.

Sometimes there is a focal or Jacksonian aura. In these cases the aura alone may occur in the intervals between the major fits and form the minor type of seizure. Thus, in wounds of the parietal region a focal commencement has been noted in the face or hand; but the generalized seizure is the common expression of the malady.

In occipital wounds, 'flashes of light' or 'flickerings' have been described as warning sensations; but the major attacks are generalized. In the frontal-lobe cases the attacks were of a general character, without warning. On the other hand, according to Netter,⁷ generalized traumatic epileptic seizures differed from those of idiopathic epilepsy in the greater frequency of an aura, the nature of which depends upon the seat of the lesion and the predominance of convulsions on the opposite side of the body to that of the trauma.

It has been noted by several writers that psychical attacks are rare, but that epileptic equivalent symptoms occur not uncommonly in the form of vertiginous attacks and paroxysmal headaches.

Attacks of vertigo in cases of gunshot wound of the head are very common, and frequently offer difficulty in diagnosis. In some cases they are undoubtedly epileptic in character, and in one instance a trephining operation arrested the major fits, but a petit mal form of vertigo persisted. In other cases vertiginous seizures would appear to be due to slight changes in the labyrinth, unaccompanied by gross evidence of labyrinthine disorder. Chartier⁸ refers to a 'reflex vertigo' which has a close relation to the 'formes frustes' of epilepsy. This occurs in small attacks of sudden origin, and may be accompanied by a transitory disturbance of equilibrium and temporary congestion or pallor of the face.

In nearly all the cases some change was noted in the *mental condition*. Memory defect, impairment of concentration, an anxious outlook, and 'nervousness' were commonly associated symptoms, irrespective of the site of the wound. In some the mental reaction was definitely retarded. In others a fear, amounting almost to an obsession, that a blow on the head over the bony gap would have fatal consequences, was admitted by the patient. In none of these respects, however, does the epileptic differ from what is found in non-epileptic cases of serious gunshot wound of the head with large bony deficiency.

FOCAL OR JACKSONIAN EPILEPSY.

In *Table B*, 10 cases of focal epilepsy are recorded. In 6 the attacks were of a temporary duration, and disappeared after trephining or spontaneously ceased. Of the remaining 4 cases, focal fits or occasional involuntary twitchings persisted, and were continuing when the patient was last observed. In one case (No. 8) there was a history of a generalized convulsion nearly four years after the infliction of the wound, but the continuing epilepsy was in the form of minor Jacksonian attacks without loss of consciousness. With only one exception (No. 10) these patients had been trephined, and presented a large gap in the cranium, with or without pulsation, and several of them showed associated paralytic symptoms, such as hemianopic

visual defect or sensory paresis. There were no paralytic symptoms in the two cases in which the frontal region was affected.

The outstanding feature of these cases is the tendency which exists towards spontaneous cure. In this respect they differ materially from the cases of generalized traumatic epilepsy. This condition is not epilepsy in the ordinarily accepted meaning of the term. It is a condition of localized cortical irritation. It arises at an earlier period than traumatic epilepsy, and is more amenable to surgical interference. Once the attacks have ceased, there does not appear to be any special tendency towards relapse.

PROGNOSIS AND TREATMENT.

Out of 38 cases of generalized traumatic epilepsy, the fits ceased for a period of twelve months or more in only 5 instances, and in one of these a relapse occurred after freedom for two and a half years. Some of the patients took bromides, but many resorted to no medicinal remedy. Of the series, 3 patients had been subjected to bone-grafting operations upon the skull, and 1 to the insertion of a celluloid plate. Of these, 2 derived some benefit, and the other 2 were definitely worse after the operation.

It is difficult to say what features of traumatic epilepsy favour cessation of the fits or promote their recurrence. The severity of the original wound does not appear to be material, and the extent of the gap in the bone would seem to play no part in the continuance of the disability. Infrequent attacks, as one would surmise, are more favourable than frequently recurring seizures. There were a few instances in which the major fits ceased, but the disorder was continued by the recurrence of attacks of an 'aura' or petit mal type. Lesion of the frontal lobe was the most favourable locality, as 3 of the 4 arrested cases had wounds in this situation.

Operations such as bone-grafting or the insertion of a celluloid plate between the brain and the scalp (Sargent's method) have been well spoken of, and the publication of the results upon the cases treated in this way is awaited with interest. Of the 4 cases in this series in which plastic operations had been performed, the results leave considerable room for improvement.

The unsatisfactory outlook in the majority of cases of generalized traumatic epilepsy is due probably to the fact that the fits are not attributable solely to an irritation or the local effect of scarring or cicatricial adhesions about the cortex cerebri, but are evidence of a generalized epilepsy in which local destructive lesions play only a minor part, once the epilepsy has been established.

A reference to *Table B* will show that true focal, cortical, or Jacksonian epilepsy develops more early than the generalized form,

and rarely passes into it. The 'latent interval' in the generalized variety is an essential and characteristic feature of the disease.

Cranioplasty in conjunction with permanent separation of the brain from the scalp in traumatic epilepsy has been advocated and carried out in a number of patients.

Sargent⁹ has had large experience of operation in this class of case, and states that the results are promising, even when the fits have recurred over a long period of time. He states also that fits can in many cases be either abolished or very considerably reduced in numbers by an operation which succeeds in removing, or at least modifying, the local existing cause. We have yet, however, to see the permanent effects of operation upon the incidence of the seizures in traumatic epilepsy, as sufficient time has not elapsed to establish the permanency of a cure. Moreover, a relapse even after a number of years' freedom from fits is no uncommon feature of epilepsy.

On the other hand, operation is regarded by several writers with disfavour. Rawling¹⁰ states that his experience is such that he does not consider operative measures are of much avail, though in some instances the plating of the defect has brought about some benefit. Redlich¹¹ writes unfavourably of the results of surgical treatment, as well as of the effects of the bromides and of luminal.

Behague¹² states that surgical intervention is dangerous, and that cranioplasty may favour the onset of epilepsy. In pre-war days also it was recognized that the results of surgical interference in cases of traumatic epilepsy were not encouraging.

CAUSATION.

When attention is diverted from the clinical history and features of the generalized seizures of traumatic epilepsy and is directed towards finding an explanation of these symptoms, a difficulty is at once encountered; and it is necessary to inquire how far the local conditions at the seat of wound may explain the phenomena, or to what extent a constitutional predisposition to nervous instability or epilepsy may play a part.

The immediate cause of epileptic fits has been for a long time the subject of speculation and conjecture. The theories of cardiac inhibition, sudden cerebral anæmia, intravascular clotting, and vasoconstriction of the cerebral arterioles have been urged from time to time as physical factors in their production. In cases of Jacksonian epilepsy following gunshot wounds of the head, Leriche¹³ noticed that the onset of a seizure was preceded by an arrest of cerebral pulsation and a blanching of the pial blood-vessels—a concrete observation bearing upon the theory of vasoconstriction as the cause of epileptic seizures.

He noticed also that the tension of the cerebrospinal fluid was below the normal in these cases.

Sargent also observed the rapidity with which circulatory changes occur in the exposed cerebral cortex, and has contended that the vasomotor changes in the brain at the seat of the wound may explain the phenomena of the tonic stage of the epileptic fit, and has suggested that the cause of these changes may be found in the 'anchoring' of the brain to the overlying membranes and scalp; and that the cicatricial changes which have succeeded to the original lesion favour the onset of convulsions under conditions of sudden change of posture in those who have "a low degree of stability of the nervous tissue".*

In this connection also it should be borne in mind that the seizures are those of generalized epilepsy, and may or may not be preceded by a local warning. It has been shown in this paper, and the observation has received corroboration by others, that Jacksonian or focal epilepsy is rare except in the early stages immediately succeeding the trauma. The recurring epilepsy associated with gunshot wounds of the head does not arise as a rule until some weeks have elapsed. In the cases personally studied, the 'latent period' extended up to over two years in several instances; the most common time for the onset of the general epileptic fits being from the second to the sixth month after the injury to the head.

From these observations it would appear that traumatic epilepsy is not a focal but a generalized type of fit with loss of consciousness, and that a latent period extending up to a couple of years or more may intervene between the trauma and the first seizure, and it is a characteristic feature of the disorder. Moreover, the major or convulsive fit is one element only of the disease, all varieties of known epileptic attack being observed in cases of traumatic epilepsy.

The contention that the local conditions at the seat of wound may be the exciting agent in the production of the fit receives some support from the observation that freeing of the cicatrices from the scalp, and the introduction of a bone-graft or celluloid plate, may arrest or relieve the attacks in some cases.

It may be argued, on the other hand, that only a relatively small percentage of cases of gunshot wound of the head develop epilepsy in consequence of their wound. A large number of ex-service men, amounting to several thousands, have received severe gunshot wounds of the head in war with extensive destruction of the skull, laceration and 'anchoring' of the brain, and associated paralytic symptoms,

* H. W. Collier (*Proc. Assurance Med. Soc.*, 1922, May) states that, when taking the blood-pressure of a patient, the onset of an epileptic fit was accompanied by a drop from 120 to 80 m.m.

who have not developed epilepsy. It is therefore difficult to avoid the conclusion that something more than local tissue alterations are requisite for the production of the seizures of traumatic epilepsy, and the determining agent, in my opinion, is an inherited or inborn constitutional predisposition to nervous instability and epilepsy.

I am unable to bring forward corroborative statistical evidence of this statement from the war cases, inquiry not having been made into the family history, as it has been customary at the Medical Board at which these cases were seen to assess direct attributability irrespective of hereditary predisposition. R. G. Gordon¹⁴ has stated that a direct family history of epilepsy is seldom obtained in these cases, but that 75 per cent have a neuropathic predisposition. In a small series of 9 cases of traumatic lesion of the head, observed in civil practice, 4, or 44 per cent, gave a family history of epilepsy or mental defect—a percentage comparable with what is found amongst epileptics in general.

Whatever the nature, position, or extent of the lesion, the fit is characterized by those features which are common to and typical of an ordinary epileptic seizure.

We have yet to await observations as to the possibility of psychogenic influences in the causation of the attacks in traumatic cases.

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- ⁴ RAWLING, *Brit. Jour. Surg.*, 1922, x, 93.
- ⁵ JEFFERSON, *Brain*, 1919, xlii, 93.
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- ⁷ NETTER, quoted by MOTT, *War Neuroses*, 1919, 210.
- ⁸ CHARTIER, *Neurologie de Guerre*, Paris, 130.
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- ¹² BEHAGUE, *op. cit.*
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- ¹⁴ GORDON, R. G., *Brit. Med. Jour.*, 1922, ii, 264.

Table A.—GUNSHOT WOUNDS OF THE HEAD.

NO.	WOUND	SUBSEQUENT CONDITION	PARALYTIC OR OTHER SYMPTOMS	EPILEPSY
a. Frontal Region.				
1	Oct. 1915. Trephined several times	Large pulsating gap in left frontal	Headaches	Fits shortly after wound and up to Oct. 1919. <i>No further recurrence</i> to Dec. 1921. Taking bromides
2	May 1915. Right fronto-malar	Scar fronto-malar region, right side	Loss of right eye. Palsy of left 5th nerve	Fits began January 1916. Continued up to Sept. 1921
3	March 1915. Fractured bone removed	Gap in left frontal bone with visible pulsation	Memory poor	Fits began June 1915 and continued every few weeks until Feb. 1921. Taking bromides
4	Jan. 1915. Mid-frontal	Transverse gap across upper frontal region	Memory poor	Fits began March 1918 and recurred till Oct. 1919. <i>No recurrence</i> up to Dec. 1921
5	April 1915. Entry below right eye. Communion right frontal	Large irregular gap in left frontal bone	Blindness left eye with ptosis. Concussion changes in retina	Four fits between Nov. 1917 and Dec. 1918. <i>No recurrence</i> up to Sept. 1920.
6	April 1917. Trephined: escape of brain matter	Pulsating gap over left frontal	Impairment of concentration	Fits began March 1918, and continued at intervals to July 1919
7	Aug. 1917. Communited fracture. Hernia cerebri	Pulsating gap over bregma and root of nose	Optic atrophy left eye	Fits began April 1918 and continued irregularly until Dec. 1920. Occasional status epilepticus. Taking bromides
8	April 1918. Trephined	Trephine wounds over left upper frontal and fronto-parietal regions	Headaches, disturbed sleep	April 1919, numerous fits; trephined. Recurrence of fits in Feb. 1920 continuing
9	Feb. 1917. Abscess. Hernia cerebri	Fracture of both frontal bones	Mental impairment	Fits began Oct. 1917 and were continuing at intervals up till Dec. 1920
10	Sept. 1915. Trephined	Gap in right upper frontal bone	None	Fits began Oct. 1916, recurring at intervals up to Feb. 1922
b. Parietal Region.				
11	Oct. 1916. Trephined	Large pulsating bony gap in right parietal region	Left hemiparesis and 'sensory' hand	Fits began March 1917, continuing up to Oct. 1921. 'Aura' in left hand
12	July 1916. Trephined over left parietal	June 1919. <i>Bone-graft</i> of gap in left parietal bone	Temporary right hemiplegia	Fits began shortly after wound and continued to Sept. 1920. <i>No recurrence</i> up to July 1921
13	Jan. 1918. Trephined over left parietal	Feb. 1919. <i>Osteoplastic</i> closure of bony gap	Right hemiparesis	Fits began Oct. 1918 and continued to March 1921; of a severe character
14	Sept. 1916. Trephined. Hernia cerebri	Large bony gap with pulsation in left upper parietal	Right hemiparesis. Memory poor. Slow mental reaction	Fits began Nov. 1917 and were continuing frequently up till Feb. 1921
15	Jan. 1916. Right parietal vertex	Depressed bony scar in right upper parietal region	Weakness of left hand grip. Headaches. Memory poor	May have had a few faints at school. Fits began in Dec. 1916 and he has had 5 up to Feb. 1921
16	July 1916. Right parietal	No evidence of any lesion of bone	Left hemiparesis. Headaches	Fits began late in 1916 and were continuing up to Sept. 1920
17	May 1915. Trephined	Gutter depression with slight pulsation in left parietal bone	Right hemiplegia with 'sensory' hand	Date of onset of fits uncertain, but continuing in June 1920 at irregular intervals

Table A.—GUNSHOT WOUNDS OF THE HEAD—Continued.

No.	WOUND	SUBSEQUENT CONDITION	PARALYTIC OR OTHER SYMPTOMS	EPILEPSY
18	Nov. 1917. Trephined	Large bony gap with visible pulsation in right upper parietal	Left hemiplegia. Phobia	Fits began in Jan. 1918 and were continuing at frequent intervals up to Jan. 1921: local warning in left face and arm
19	Aug. 1916. Trephined	Gap in bone over right parietal eminence. No pulsation	Amputation of right arm. Defective memory	Fits began May 1917 and were continuing every two or three months in Dec. 1920
20	April 1915. Trephined. Abscess formed	Gap in left parietal bone below and behind parietal eminence	Partial sensory aphasia and slight visual defect in lower quadrants	Fits began in March 1916. Has had—last in January 1919. <i>No recurrence</i> up to April 1920
21	Nov. 1917. Right parietal wound superficial	Superficial scar over upper right parietal near sagittal suture	Neurasthenia (scalp-wound case)	Fits began in Dec. 1917 and continued to July 1919. <i>No recurrence</i> to March 1920
22	Oct. 1917. Trephined	Gap in right fronto-parietal region. No pulsation	Left hemiplegia. Memory defective	Fits began early in 1918 and were continuing in Nov. 1920. No relief from bromides
23	May 1918. Trephined	Gap in bone in right parietal region	Temporary left-sided hemiparesis	Fits began several months after wound and were continuing in June 1919
24	Aug. 1919. Trephined for fractured skull	Large gap in right upper parietal with pulsation	Left hemiplegia. Dysarthria	Fits began Nov. 1919 and were continuing at intervals Oct. 1922
25	Nov. 1917. In Oct. 1920 trephined for fracture of inner table of skull and contusion of cortex cerebri	Gap in bone in left upper parietal region	Headaches. Memory poor	Fits began in Jan. 1918 and continued until operation in Oct. 1920. Since then occasional 'vertiges'
26	May 1916. Trephined	Gap in bone in right parietal. Pulsation	Left hemiplegia	Fits began in July 1916 and were continuing in April 1922
c.	Occipital Region.			
27	July 1916. Trephined on left upper occipital region	In Oct. 1920 cicatrix excised and <i>celluloid plate inserted</i> into gap in occipital bone	Sector loss of visual fields downwards to right side	Had a fit after original operation. About 15 fits up to Oct. 1920 (second operation). <i>Since this only 'visual aura' sensations</i> up to Feb. 1921
28	Feb. 1917. Trephined	A gap in left occipital bone with pulsation	Sector loss of visual fields downwards to right side	Fits began in June 1917 and continued to June 1919, but numerous 'flickerings' persisted up to March 1920
29	February 1915	Gap in right occipital bone with pulsation	Right-sided hemianopia and small sector to left side	Fits began in Nov. 1915 and have continued at intervals (with 2½ years freedom) to April 1920
30	Sept. 1917. Trephined. Hernia cerebri	Gap in right occipital bone with pulsation	Right-sided hemianopia	Fits began Nov. 1918 and were continuing in Sept. 1920
31	May 1915. Trephined. Hernia cerebri	Large gap in bone in occipital region mainly on right side	Left-sided hemianopia	Fits began in Oct. 1917 and were continuing in Sept. 1920
32	April 1918. Trephined. Abscess in left occipital lobe	Feb. 1920. <i>Bone grafting operation</i>	Right-sided hemianopia	Seems to have had minor attacks up to June 1921, when major fits began, which were continuing in April 1922
33	June 1916 in left occipito-temporal region. Abscess said to have formed in frontal lobe	Large bony gap with pulsation in left fronto-parietal region	Right hemianopia. Memory bad	Date of onset of fits uncertain, but fits of a 'psychical' type were continuing frequently in Jan. 1922

Table A.—GUNSHOT WOUNDS OF THE HEAD—Continued.

No.	WOUND	SUBSEQUENT CONDITION	PARALYTIC OR OTHER SYMPTOMS	EPILEPSY
34	July 1916. Lambdoidal region	Irregular depressed bony scar over lambdoidal region	A temporary weakness of right arm	Vertiginous attacks began in Aug. 1916 and were continuing in May 1922
35	Sept. 1917. Trephined in Jan. 1918	Bony gap in left parieto-occipital region	Right-sided hemianopia. Headache	Fits began in Nov. 1919 and were recurring at intervals in Oct. 1922
d.	Temporal Region.			
36	Sept. 1916. Posterior temporal region. Trephined	Depressed scar right posterior temporal region: slight pulsation	Deafness right ear. Mental reaction slow	Fits began June 1918 and were continuing at intervals in Sept. 1921
37	March 1918. Zygomatic region right side: bone removed	Depressed bony scar behind right angular process	Detachment of right retina	Fits began in Oct. 1919 and were continuing in May 1920 with considerable frequency
38	Sept. 1916. Left mastoid region	Depressed scar and bony deficiency over left mastoid	Atresia left external meatus: deafness left ear, and left facial palsy	Fits began in Jan. 1920 and were continuing in Oct. 1922. There is an aura of smell

Table B.—JACKSONIAN EPILEPSY.

No.	WOUND	PERMANENT STATE	ASSOCIATIONS	FOCAL EPILEPSY
1	March 1918. Right parietal region	Gap in right parietal bone	Headaches	Fits on left side after wound. phined—removal of clot. No further fits at Jan. 22, 1920
2	March 1916. Left parietal region: depressed fracture. Trephined	Gap in left parietal bone	Partial sensory aphasia	Two focal fits in Sept. 1916. No further fits at July 7, 1919
3	Feb. 1915. Left parietal region: bone depressed. Trephined	Gap in left parietal bone	Right 'sensory' hand	Focal fits shortly after wound. No further fits up to April 8, 1920
4	Sept. 1916. Right frontal abscess of brain followed	Gap in bone in right fronto-temporal region	None	Convulsions following wound. No further fits up to Oct. 27, 1920
5	Oct. 1914. Right parietal region. Trephined	Gap in lower part of right parietal bone	Slight weakness of left hand	Focal fits in June and Oct. 1915 occasional involuntary twitchings of fingers of left hand persisting up to Oct. 1920
6	Dec. 1914. Gutter wound of left parietal region: bone splintered	Antero-posterior gutter wound of left parietal bone	Giddiness: Tinnitus. Memory poor	Occasional twitchings of right arm without loss of consciousness persisting from time of wound up to July 1920
7	Dec. 1916. Left parietal region	Gap in left parietal bone	Giddiness. Mentally dull	Two focal fits after wound for which he was trephined. No further fits up to July 1919
8	Jan. 1917. Left fronto-parietal region. Trephined	Large gap in bone, left fronto-parietal region. Pulsation	Impaired power of concentration. No paralysis	Occasional twitchings and spasms in right arm and hand, with temporary loss of power continuing to March 1922. History of a convulsion in Oct. 1921
9	Aug. 1918. Left parietal region. Trephined. Hernia cerebri	Large gap with pulsation in left post-parietal bone	Some hemianopic visual defect to right side, and right deep reflexes in excess of left	Attacks of numbness of right hand with temporary aphasia recurring every 3 or 4 months and persisting up to Sept. 1921
10	Aug. 1916. Right parietal region	Scar without loss of bone over right parietal eminence	Vertigo. Headache.	Focal fits followed the wound, but no further fits up to Nov. 1922