DISABILITY CAUSED BY BRAIN WOUNDS*

A REVIEW OF 1,166 CASES

BY

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This paper is based on a follow-up study which has continued throughout and since the late war. The study centres around the work done by many specialists at the Military Hospital for Head Injuries, Oxford, and with the mobile neurosurgical units in different theatres of war. The Medical Research Council has assisted throughout in providing for the preservation and copying of case records, and since the war, with aid from the Nuffield Provincial Hospitals Trust, it has been possible to organize a small Head Injury Advice Bureau which aims at providing a competent service to which the pensioner with a brain wound can turn for advice or help. At the same time, this bureau provides an admirable organization for scientific follow-up purposes. The staff consists of an almoner, secretary, psychologist, and any neurologists who may be available. The combination of social welfare and follow-up research studies seems to operate to the mutual advantage of both.

The main purposes of this research are concerned with the ever-stimulating problems regarding the effect of lesions of different areas of the brain, and also with the problems of rehabilitation which the cases present. I shall not, however, describe these special studies, but will rather attempt to give a broad picture of the effects of brain wounds.

Thanks to the helpful cooperation of Ministry of Pensions administrators we have been able to re-examine a good proportion of the cases many years after wounding. Others have been kept in touch with by questionnaire, letter, welfare officer visits, and other means. The features of each case have been transferred to a paramount card (Fig. 1, p. 36) designed to facilitate analysis.

The cases considered were all of men who reached this country alive. It was after the invasion of Normandy that most of the injuries occurred and the wounded often reached the hospital at

Oxford by air within one or two days of wounding. A proportion therefore of desperately ill cases arrived in this way, a fact which influenced the early death rate seen in Table I. The main feature

TABLE I
ANALYSIS OF 78 DEATHS OUT OF 1,166 CASES FOLLOWED UP FOR FIVE YEARS

Deaths due to head injury ..	71 (6%)
Interval after wounding:	
within 1 week ..	23
1 week to 1 month ..	27
1-2 months ..	9
over 2 months ..	12
Deaths from other causes ..	5
Suicide	2

of this table, however, is the amazingly low incidence of delayed deaths—a remarkable testimony to the work of many neurosurgeons, and to antibiotics. As to the causes of late death, meningitis and status epilepticus are the most important.

It is customary to separate wounds which penetrate the dura mater, "penetrators", from those which cause damage to scalp and skull without dural laceration. This separation is shown in Table II. As will be seen later, the brain is usually

TABLE II
PROPORTION OF 1,088 SURVIVING CASES WITH ADDITIONAL WOUNDS

	Total Surviving Cases	With Additional Wounds
Wounds of scalp and skull, dura intact	268 cases	55 cases (20%)
Wounds penetrating dura and brain	820 cases	254 cases (31%)
Total	1,088 cases	309 cases (28%)

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FIG. 1.—Photograph to illustrate the use of the "paramount" card* designed for analysis of these cases. A tracing of the patient's skull radiograph is superimposed.

* Printed by the Copeland-Chatterton Co. Ltd., Stroud, Glos.

injured focally in both types of injury, but the risk of infection is of course much reduced when the dura mater is intact. Table II also demonstrates the high incidence of multiple wounds even in a series of cases selected largely in the acute stage for transfer to a neurosurgical unit.

Early Focal Signs

In the late war most of the cases of head wound which reached hospital alive had been hit by small fragments of high explosive shells (scalp wound of brain, skull or scalp). These metal fragments on striking the head either lodge in the skull, glance away, or cut into the brain. In either case a small shower of bone fragments is often projected into the brain from the point of impact. Many of the resulting lesions are remarkably self-limited and provide unique material for the study of focal brain lesions. This is especially evident when, as often happens, the wounds cause little or no cerebral concussion, so that the duration of post-traumatic amnesia is very short, or indeed there is often no disturbance of consciousness (Table III). This is in striking contrast to the ordinary cases of accidental head injury (Symonds and Russell, 1943) where a period of amnesia is found in all except the slightest injuries. The gun-shot wound cases tend to have either a very short or a long amnesia, whereas in the "accidental" injuries all amnesic groups are well represented except the shortest.

In contrast, however, to the absence of amnesia

TABLE III
COMPARISON BETWEEN DURATION OF POST-TRAUMATIC AMNESIA IN SHELL WOUNDS OF THE HEAD AND OF ACCIDENTAL HEAD INJURY

	Duration of Post-traumatic Amnesia (Hours)					Total cases
	Nil	-1	1-24	>24	?	
Shell wound of brain	216	172	57	320	55	820
Shell wound of skull and scalp	123	68	23	36	18	268
"Accidental" head injury (Symonds and Russell, 1943) ..	28	75	65	69	—	237

in many cases, the incidence of gross focal signs immediately after wounding is surprisingly high in this series, even in cases without dural penetration (Table IV). It is of course likely that the occurrence of gross focal signs increased the liability for the case to be evacuated to a special centre. Never-

TABLE IV
INCIDENCE OF EARLY FOCAL SIGNS (SENSORY-MOTOR, VISUAL, OR APHASIC) IN 884 CASES

	Total	Cases with Early Focal Signs
Wounds of skull and scalp	191	129 (68%)
Wounds of brain ..	693	517 (75%)*

* Of remaining 176 cases, 152 were frontal wounds.

theless, it seems evident that wounds to all parts of the parietal, temporal, and occipital lobe are very likely to cause focal symptoms, temporary or permanent, such as paralysis, aphasia or partial blindness. As is seen in Table IV, 75% of penetrators had early focal signs. Of the remaining 176 cases, 152 were frontal wounds. These focal disorders are often transient and may be strictly localized. For example, a small mid-Rolandic wound causes paralysis of the opposite arm only, while the small parasagittal wounds of the same area lead to immediate paralysis of one or both legs.

Many students of brain function have considered the effects of trauma to be too uncertain for such cases to be used in the detailed study of focal lesions. My experience of this series, however, has led me to consider that among the cases can be distinguished a number of small wounds which provide excellent material for the study of brain function. These cases are distinguished by studying details of the wound, the operation, the early focal signs, and the permanent disability.

The very high incidence of *early* focal signs in brain wounds seems to depend partly on the spread of a shock effect from the site of wounding. Tables V and VI compare the incidence of early signs with

TABLE V

ANALYSIS OF EARLY AND PERMANENT SENSORI-MOTOR DISORDERS IN 370 OF 693 CASES WITH PENETRATING BRAIN WOUNDS

	Early	Permanent (excluding Slight)
Affecting one arm and/or face	107	43
" arm and leg	192	100
" one leg	9	15*
" both legs	12	4
" one arm and both legs	22	7
" 4 limbs	8	—
" uncertain	20	—
Total	370 (53%)	169 (24%)

* Increased figure is due to some early cases of paraplegia or triplegia recovering to crural monoplegia.

the permanent signs of more than trifling severity, and it is clear that a large number of the cases with early signs recover well. However, the incidence of permanent major physical disability is over 30%, and as will be seen later (Table XI) about a third of the men with penetrating wounds are drawing a 100% pension five years after wounding. It should be explained that the same case may appear in both Tables V and VI. On the other hand, though slight degrees of paresis are excluded from the permanent disabilities in Table V, the slightest

TABLE VI

INCIDENCE OF EARLY AND PERMANENT VISUAL AND APHASIC DISORDERS IN 693 CASES OF PENETRATING BRAIN WOUNDS

693 Penetrators with Focal Symptoms		
Symptom	Early	Permanent
Visual	302 (44%)	209 (30%)
Aphasic	217 (31%)	135 (20%)* (including "slight" cases)

* Entry on left in all but 7.

permanent disturbances of the visual fields or of the speech mechanism are included in Table VI. The overwhelming importance of the dominant (generally left) hemisphere in relation to aphasia is also evident.

Site of Wounding

In Fig. 2 a system of measurement is used (Russell, 1947) to record on one chart the site of wounding in a large number (631) of cases. The crosses represent wounds which were followed by post-traumatic epilepsy (306 cases), and the circles the site of wounding in 325 cases which did not develop epilepsy within five years of wounding. It will be seen that there are many examples of wounds to all parts of the brain except the brain-stem, while the limited number of cerebellar and low occipital wounds reflects the dangerous nature of wounds involving the posterior fossa of the skull. This figure does not indicate accurately the regional incidence of post-traumatic epilepsy, but it does show that the incidence is higher in some regions (e.g., the parietal lobe) than in others.

A more accurate assessment of the incidence of post-traumatic epilepsy is provided by analysing the paramount cards directly. Table VII gives the

TABLE VII

INCIDENCE OF EPILEPTIC FITS ON FOLLOW-UP AT OVER 5 YEARS IN 820 "PENETRATORS"

Epileptic fits on follow-up	356 cases (43% of total)
No fits in cases followed for—	
over 5 years	403 cases
2-5 years	21 "
1-2 years	21 "
under 1 year	19 "
Total	464 "

result of such an analysis and indicates a high incidence (43%) for the occurrence of one or more fits within the first five years after wounding. The

FIG. 2.—Chart to show the site of wounding in 631 cases. Charting depends on measurements taken from the skull radiographs (Russell, 1947). Wounds which lie within 5 cm. of the sagittal line are placed in the halo outside the skull outline. The cases marked X developed post-traumatic epilepsy while those marked O showed no epilepsy at a five-year follow-up.

interval after wounding at which the first of these fits appeared is given in Table VIII, and the type of fit in Table IX. This information cannot be considered very accurate as many of the cases had to be assessed from questionnaire or “fit form”. However, my impression is that the incidence of post-traumatic epilepsy in these tables is an underestimate, as the doubtful cases go in the “no-epilepsy” group. Although the incidence of post-

traumatic epilepsy is so high, the actual fits in most cases are so infrequent, and so easily controlled by anti-convulsant drugs, that post-traumatic epilepsy is seldom in our experience a very serious disability a point of great importance in assessing the need for surgical treatment.

These figures for post-traumatic epilepsy show no striking change from the published records following the first world war, and this in spite of the vastly reduced incidence of infection in the present series. Further, Table X shows that the incidence of post-

TABLE VIII

TIME OF ONSET OF POST-TRAUMATIC EPILEPSY IN 356 CASES

Interval after Wounding of First Fit	
Under 1 month	47* cases
1-6 months	94 „
6-12 months	62 „
1-2 years	49 „
Over 2 years	28 „
Unknown	76 „
Total ..	356 „

* Includes 14 cases with no fits after first month (5 year follow-up).

TABLE IX

TYPE OF EPILEPSY IN 356 CASES OF POST-TRAUMATIC EPILEPSY

Type of Fit	No. of Cases
Focal only	46
General only	192
Focal and general	118
Total	356

TABLE X

APPROXIMATE INCIDENCE OF POST-OPERATIVE INFECTION OF SCALP, SKULL, MENINGES OR BRAIN

	No. of Cases with Post-operative Wound Infection
356 cases of post-traumatic epilepsy	61 cases (17%)
466 cases. No epilepsy	61 cases (13%)

operative infection played no great part in the development of epilepsy in this series. These figures must cast doubt on the thesis put forward many years ago by Penfield (1927) in which he maintained that progressive gliosis in relation to a brain scar is the main factor which causes fits. Indeed this view has already failed to hold the field, as is evident from Earl Walker's recent approach (1949) to the subject of traumatic epilepsy, in which he leaves the scar alone and excises healthy-looking pieces of electrically unstable cortex in its neighbourhood.

Another popular view in recent years has suggested that a constitutional factor determines the occurrence of traumatic epilepsy. This, however, seems unimportant when the incidence is approaching 40%, nor does it offer any reason for the comparatively very low incidence in closed head injuries (less than 3%) with diffuse neuronal injury.

One cannot but look round for some other factor in the causation of traumatic epilepsy. In this connexion it should be noted that the ultimate incidence of post-traumatic epilepsy is likely to be little short of 50%, a figure which indicates even chances whether or not epilepsy occurs. It seems to me that this figure suggests that brain wounds may upset the balance between two antagonistic types of cerebral mechanisms and that the development of epilepsy may depend upon whether one or the other is the more damaged. There is certainly nothing very improbable in the suggestion that many cerebral thresholds are maintained by a balance between the two opposing mechanisms of stimulation and suppression, and that trauma may upset this balance one way or the other. Such a view adds uncertainty to the surgical treatment of traumatic epilepsy as we do not yet know how to redress the balance, but there are hopes that the future may bring new knowledge which will add to the surgeon's effectiveness in this field.

Finally, I should like to give some information regarding the social status of these pensioners. The Ministry of Pensions percentage disability five years after wounding is shown in Table XI, and the rough employment groups are given in Table XII.

TABLE XI

RATE OF PENSION AND FITNESS FOR EMPLOYMENT ON LAST FOLLOW-UP OF 606 CASES OF PENETRATING BRAIN WOUND

	Rate of Pension	No. Gainfully Employed
No pension ..	35	35
20-40% ..	136	136
50-70% ..	154	153
80-90% ..	72	71
100% ..	209	93
	606	488 (81%)

TABLE XII

TYPE OF EMPLOYMENT IN 488 CASES

Type of Employment	No. of Cases
Heavy manual ..	24
Light manual or unskilled ..	402
Intellectual or highly skilled ..	62
	488

It is clearly gratifying that such a high proportion (81%) are earning a living, but employment is generally unskilled and the mental adjustment to grave disability is often slow and painful. The marriage state of these cases is given in Table XIII.

TABLE XIII

SOCIAL STATUS OF 209 CASES DRAWING 100% PENSION

Social Status	No. of Cases
Single ..	84
*Married before wounding ..	75
Married since wounding ..	35
Not known ..	15

* Four separated and one divorced.

This is of importance, for the help of an understanding wife is quite invaluable to these men. Efforts made to settle the unemployed or unemployable are shown in Table XIV. Though many

TABLE XIV

OCCUPATIONAL STATE OF 117 "UNEMPLOYED" CASES

Occupational State	No.
In and out of work ..	23
Helping relations in shops, etc., or home ..	8
Rehabilitation centre or course ..	14
Poultry ..	3
Sheltered workshop ..	2
Mental hospital ..	4
Emigrated ..	1
No work or occupational therapy at home or Headington Hill Hall ..	62
	117

organizations assist in this type of work the contribution by our advice bureau has been, I think not unimportant. Further, the Red Cross and St. John organizations run an excellent rehabilitation centre at Headington Hill Hall in Oxford.

A little ingenuity will often find some profitable occupation for the severely disabled. Many of our cases at Headington Hill Hall suffer from severe hemiplegia and aphasia, and yet can be trained to make excellent furniture (of a value of £3 to £4 per week) in a special workshop; one such patient also can weave three yards of tweed in four hours (selling for over £2), and another working on a lathe makes wooden lamp stands at the rate of one a day which sell for 17s. 0d.

I hope that our experience may be of interest and help to those concerned with similar problems.

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