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VA Medical Monograph

A FOLLOW-UP STUDY OF HEAD WOUNDS IN WORLD WAR II

by

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Preface

The volume before you is the fifth in a series of monographs prepared under the sponsorship of the Veterans Administration by the Follow-up Agency of the National Academy of Sciences–National Research Council. In cooperation with the Veterans Administration and the Department of Defense, the studies reported here have been directed toward a longitudinal view of the head injury problem. The work represents several years of careful examinations, statistical evaluations, and correlations of clinical data from four centers in the United States. The authors have focused their attention chiefly on the presentation and validation of findings. They have not undertaken an exhaustive discussion of the subject or review of its voluminous literature. In a highly condensed (largely tabular) form, the monograph brings to the profession one of the most extensive and complete studies of its kind.

We hasten to note that these records and conclusions have importance well beyond the circumstances of war. Indeed, the diabolical talents of warmaking are hard pressed to exceed the risks of modern civil life as a potential source of trauma.

Quite aside from its important topic, the monograph is a veritable gold mine of information, direction, example, and commentary on the collection and evaluation of medical data. Working under the severe handicap of any retrospective survey, the authors have with circumspection and precision handled an elusive and capricious body of information. By examining their sources of error and exposing every artifact of observation, they have created a valuable sourcebook for those who wish to conduct researches of a similar nature.

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The Medical Statistics Division, Office of The Surgeon General, U.S. Army, kindly provided duplicate punchcards on World War II head injuries to serve as the sampling base. Similarly, the Veterans Administration provided lists of veterans receiving disability compensation in 1950 for residuals of head injuries and resident in the areas of study. Records officials of the Department of Defense and the Veterans Administration have generously provided access to the records needed for the research. The American Red Cross extended its helping hand in each study area, taking on the difficult task of interpreting to the subjects the nature and purpose of the study, and obtaining their cooperation when the personnel of the follow-up center had exhausted their resources.

We also owe particular thanks to Mr. A. Hiram Simon, who supervised the statistical processing in the Follow-up Agency; to Miss Vivian Heidenblut of his staff, under whose direction the demanding task of abstracting and coding the medical records was performed; and to Mrs. Myrtis E. Hillman, without whose devoted editorial efforts this report could not have been completed.

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Foreword

Great wars between nations are a part of the history of man, deciding nothing permanently. The prospects for their outlaw are as dim as they have ever been, and military preparations for the next mount with increasing seriousness, demanding more and more sacrifices from citizens of all nations.

If one looks for a modicum of contribution to the welfare of man from war, it is to be found only in the benefits he may receive from the medical and surgical experiences gathered during war at an expensive price. The facts gained from the treatment of chest wounds, injuries to the peripheral nerves, the control and prevention of contagious diseases, and infections are examples of the application of advancements made in medicine and surgery during wartime.

This volume, a *post hoc* study, made under servere obstacles, of a group of patients who received craniocerebral injuries, is the result of several years of careful examinations, statistical evaluations, and attempted correlations of head trauma, symptoms, and objective findings at four centers in the United States. This monograph is a part of the program of studies of the Follow-up Agency of the National Academy of Sciences-National Research Council developed by the Committee on Veterans Medical Problems in cooperation with the Veterans Administration and the Department of Defense. It is the companion of a similar study, under the same auspices, of the results of peripheral nerve injuries received during World War II.

It is regrettable that this study and that of peripheral nerve injuries were handicapped by the absence of a prepared plan of observations which should have been outlined between World Wars I and II. The lessons learned during World War I were not utilized to the fullest extent during World War II, an outstanding example being the treatment of peripheral nerve injuries. Missing are data which might easily have been obtained even under the stress of battle and evacuation problems if, during the brief intervals between wars, thought had been given and work expended upon how the greatest future good could be obtained from the terrible sacrifices asked of men.

Aside from the data which have been so carefully collected and evaluated, it is to be hoped that this book will stimulate thought about plans which can be put into effect during any future conflict, so that future evaluations of medical and surgical experiences can be improved beyond the costly record of the past.

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Table of Contents

Chapter I

INTRODUCTION

A. HISTORICAL BACKGROUND

War, which seems to date far into antiquity, and man's proclivity accidentally to hit his head, have been prolific sources of brain injuries since the beginning of time. That man has been very much interested in the exciting events which follow blows upon the head would seem evident from his early art, the relics of primitive trephining, and the importance which this subject is given in the earliest medical records (133). In the prehistoric skulls discovered in the caves of France, there have been found manmade holes, presumably bored for the relief of symptoms caused by head injuries, of which the still visible fracture lines bear mute testimony. The tombs of Paracas in the New World also contained cracked skulls, with bony defects presumably trephined for the effects of cranial violence. And in the earliest extant manuscripts of Egyptian medicine, explicit directions are given for the care of head wounds (22). It therefore seems likely that for thousands of years man has been aware, not only of the primary effects of head injuries but of the longer lasting and sometimes permanent damage which may be inflicted by a blow upon the head. Yet, the fact that the phenomena were recognized by early physicians is no indication that there was a real understanding of their nature. Indeed, it has only been in the past century that the physiological principles involved have been suggested. Just 200 years ago Beckett (13), in a surgical textbook, wrote that "to explain the cause of a person's falling to the ground immediately on the reception of a blow, we ought to observe that the blow caused a violent commotion of the whole brain, and so consequently put the spirits in a great confusion and disorder; which making irregular incursions into the several parts of the body, without the direction of the will, could not be confined to the nerves, whose office it was to distribute them into those muscles that keep the body in an erect posture; for which reason the machine must unavoidably fall to the ground."

Irrespective of the bizarreness of the explanation, the fact that blows upon the head would render an individual unconscious has been known throughout history, and is unquestionably the reason for the early adoption by soldiers of various types of protective headgear. The chronic ill effects produced by such blows upon the head have also been noted, both in legend and in historical records. Although little emphasis is placed upon headache and dizziness as the result of head injuries, the fact that convulsive attacks might follow a head injury was observed. In the 14th century Valescus de Tharanta (129) described a patient who had seizures seven or eight times a day following a penetrating head wound. Berengarius (16) gives an interesting description of a patient who, about 2 months after an injury, developed epileptic paroxysms. He was strung feet up, the head wound

opened, and a large quantity of milky material evacuated, whereupon the convulsions stopped. Duretus (67) was aware that fits might appear many years after a head injury, for he had observed the convulsions of an 18-year-old youth whose head some 6 years previously had been cracked, depressing a bony fragment. Duretus related how he perforated the bone, elevated the depression, and cured the condition.

Even in the 19th century, although physicians considered head injuries a possible cause of epilepsy, their etiological role was not regarded as very important. Echeverria (39) concluded that almost 10 percent of his cases of epilepsy had a traumatic origin, but other authors did not find such a high correlation. Bouchet and Cazauvieilh (19) found only 1 in 69 cases, and Leuret (79) 1 in 67 cases in which a blow to the head was thought related to the convulsions. Hammond (61), in his textbook, does not refer to head injury as a cause of seizures. To some extent this may be due to the lethal nature of compound wounds of the head in those days. If the initial shock and blood loss did not kill, the victim usually succumbed to infection. This was so serious that many surgeons gave up trephining for head injuries. In St. Bartholomew's Hospital between 1860 and 1867, the trephine was not used once, and in all France in this period only 4 trephinations were performed. Obviously, under such circumstances, patients with serious brain injuries rarely survived, so that posttraumatic epilepsy was not often observed.

With the introduction of antiseptic and later aseptic surgical techniques, however, many brain-injured patients survived. Accordingly, our statistics for the chronic effect of head injuries date from the time of Lord Lister. Since that time there have been many reports of the incidence of posttraumatic epilepsy following wounds incurred in war and peace.

From the Franco-Prussian, Russo-Japanese, and Boer Wars, and particularly the war of 1914, have come many series of head-injured patients, emphasizing not only the special problems in diagnosis of brain injury but also the difficulties in treatment of the neurological deficit, and in vocational and social rehabilitation. As the surgical care of these wounds improved, the number of survivors increased and the severity of the neurological deficit was greater, so that the subsequent care of these patients became a greater problem. Since it was early recognized that the State was obligated in case of war injuries, disability compensations or pensions were more thoroughly studied.

B. LEGISLATIVE PROVISION FOR DISABLED SOLDIERS

The principle that the State was morally obligated to care for its disabled soldiers seems to have been accepted from colonial times (40). In 1636 the Pilgrims enacted a disability pension law to the effect that any man engaged as a soldier, who was maimed, should be cared for by the colony during his life. Regular meetings were held in Boston to hear the applications of wounded soldiers. Shortly after, disability pension laws were passed in Virginia, Maryland, New York, and, somewhat later, in Rhode Island. The first national pension law of August 26, 1776, provided half pay during disability to any member of the forces losing a limb or being so disabled in the services of the United States as to render him incapable of earning a living. However, at the conclusion of the Revolutionary War the Con-

federation had no funds, and the commutation certificates given soon dwindled to a mere 12.5 cents on the dollar. In June 1785 a uniform method of handling disability pensions was adopted by the States, which were reimbursed from the Confederation, but because of certain limitations of time invoked subsequently, veterans were generally dissatisfied. The general pension law of 1792 was not much better, for it imposed upon the Circuit Courts of the United States the duty of examining applicants for pensions and determining the nature and degree of disability. The judges questioned the constitutionality of this law to impose upon them duties which were not properly judicial. Although some judges, as commissioners, did hear claims under this law, the issue was so controversial that Congress in February 1793 repealed the objectionable sections and issued new regulations. These provided that claims be presented to the judges, but Congress reserved the final action on their allowance. However, disability or invalid pensions of the Revolutionary War were not numerous; in 1792 only 1,472 men were on the rolls.

In the next 100 years, service pensions and pensions for widows and dependents of soldiers and sailors were frequently modified by Congress, but the disability pensions were little changed until the Civil War. An act of 1862 provided pensions for disability incurred in the line of duty since March 4, 1861, while in the service of the United States. The rates for total disability ranged according to rank from $30 (colonel) to $8 (non-commissioned officers and privates). Partial disability was pensioned proportionately. Subsequently, the pension rates were gradually increased and the specific disabilities better defined.

In February 1811 there was established by legislation the Naval Home in Philadelphia, which, however, was not occupied by disabled members of the Navy and Marine Corps until 1833. Realizing the need for further homes for invalided soldiers, Congress passed an act in March 1851 creating two military asylums, one at Harrodsburg, Ky., which was disposed of by public sale in 1860, and the other in Washington, D.C., which is now known as the Soldiers' Home. A few years later a further enactment on March 3, 1865, authorized the establishment of the National Home for Disabled Volunteer Soldiers (53). This was erected in Togus, Maine, in 1866, and additional branches were built in various parts of the country, so that by 1930, when the National Home for Disabled Volunteer Soldiers was consolidated with the U.S. Veterans' Bureau, there were 11 such homes.

The next major change in disability pensions came shortly after the declaration of war against Germany in 1917. At that time, legislation was introduced to amend the War Risk Insurance Act of 1914 to allow a program of allotments and allowances for dependents of the fighting men, monetary insurance against death or permanent total disability, pensions or compensation for disability of a veteran, awards to his dependents for his death due to injury or illness as a result of service, medical treatment and supply of required orthopedic appliances for service-connected disability, and vocational rehabilitation.

On August 9, 1921, the U.S. Veterans' Bureau was created by uniting the Rehabilitation Division of the Federal Board of Vocational Education, the Bureau of War Risk Insurance, and certain hospitals, caring for veterans, which were previously controlled by the U.S. Public Health Service.

Some 9 years later a further consolidation of the activities of the Bureau of Pensions, the National Home for Disabled Volunteer Soldiers, and the U.S. Veterans' Bureau established the Veterans Administration (65).

C. SPECIAL PROBLEMS OF BRAIN-INJURED MEN

This brief survey of the development of modern systems for the care and rehabilitation of military casualties, especially the head injured, would not be complete without some mention of the special problems of the brain-damaged individual. Such patients were not simply hemiplegics, epileptics, or aphasics, alone or in combination, but presented a more general problem requiring a holistic view of the individual. This situation was early recognized in certain European countries where special institutes with well-organized staffs highly specialized in various neurological, psychological, therapeutic, and rehabilitation techniques were set up to study and treat head-injured patients. In Germany the "Institut zur Erforschung der Folgeerscheinungen von Hirnverletzungen" carried out such a program (55). As a result of these enterprises and the activities of the various veterans' pension boards, in stimulating studies of the biological course of head injuries, islands of data are available on certain aspects. In order to obtain a longitudinal view of the head injury problem, a project was set up after World War II, under the auspices of the Veterans Administration, to follow up a representative group of head-injured patients in order to determine the natural history, the stresses, social and biological which may modify it, and the fate of such a group. It was hoped that in a period of 5 to 10 years the general trends might be apparent. This report is the result of such a study of a series of head-injured veterans of World War II.

No attempt has been made to review or cover the voluminous literature on the subject of head injuries and their sequelae. The occasional reference in the text to other studies we hoped might place the results of this follow-up in a proper setting and give a perspective to the whole. This monograph is a presentation of data rather than an exhaustive discussion of the subject of head injuries.

Chapter II

METHODS AND MATERIALS

A. SAMPLING PLAN AND ORGANIZATION OF STUDY

The Medical Statistics Division, Office of The Surgeon General, Department of the Army, made available duplicate punchcards representing the 31,178 battle casualty admissions to Army hospitals in 1944 with the diagnosis of a traumatism of the head. It had been hoped to use this file as the source of all cases for the study, but elimination of the thousands of death cases, scalp lacerations, ear wounds, etc., reduced the roster to less than a third of its original size. The records of the Veterans Administration were then consulted to obtain current addresses. Since it was feared that a poor response would be encountered by an attempt to examine men living at great distances from the clinical centers, certain areas were defined for each of the 4 centers, and only men residing in the defined areas were allocated to the centers for examination. The choice of particular areas for each center was influenced by ease of transporation and the number of patients available. The areas chosen and the numbers of men allocated (hereafter called collectively the "Army roster") are shown below.

	Men
Johns Hopkins Hospital (Baltimore, Washington, Philadelphia, and vicinity of each)	159
Montefiore Hospital (New York City)	193
Cushing VA Hospital (Boston and vicinity)	73
Long Beach VA Hospital (Los Angeles and vicinity)	38
Total	463

The method of selecting the Army roster was not such as to produce a sample representative of all head wounds. This was, however, no part of the purpose, which was to elucidate the course of the patient with a head wound in terms of the characteristics of the injury and, if possible, to assess the influence of certain factors associated with treatment.

Since the Army files did not supply adequate numbers of cases for the clinical centers, the roster was supplemented by men drawn from lists of veterans receiving disability compensation from the Veterans Administration. Each of the more than 60 regional offices of the Veterans Administration maintains lists of the veterans of the area who receive a disability compensation or pension. Men are classified by the nature of the disability; if two or more disabilities coexist, for the purpose of these listings, assignment is made to the most important single disability. Of course, in the individual veteran's record all causes of disability are shown.

The lists of veterans receiving disability compensation seemed quite attractive as a source of case material since, as they are kept on a regional office basis, it was possible to obtain lists which were very largely made up of men whose place of residence made them suitable for study at one or another of the 4 centers.

A review of the records of 60 head-injured men drawn from the Army roster revealed that the majority was being compensated for 1 of 3 particular causes:

a. Posttraumatic personality disorder.
b. Posttraumatic encephalopathy.
c. Loss of part of inner or outer tables of skull.

Lists were obtained from the Veterans Administration which included all men who resided in the study cities and who were, in 1950, receiving disability compensation for one of these disorders.

The number of men allocated to the 4 centers from the "VA roster" were:

Baltimore	157
New York	107
Boston	93
Los Angeles	112
Total	469

After review of the records of men drawn from the VA roster had begun, it became plain that this roster included many men who had suffered noncombat injuries—falls and the like. Thereafter, men were included only if they had suffered a bullet or shell fragment wound. However, 98 men for whom records had already been abstracted, and for whom the wounding agent was not a missile, were continued in the study. The other 371 men had missile wounds of the type specified. All except 4 of the 98 men referred to above were Baltimore cases, since the Baltimore center operated as a pilot study and Baltimore cases were the first for which records were abstracted.

The New York area was the only one in which it was possible to find more cases than could be examined in the time available. It was thought that a total of 300 represented a maximum practicable load. Therefore, the men drawn from the VA roster for New York represented the first 107 eligible men on the listing. Since the listing was arranged in claim number order, this was equivalent to choosing those eligible men who earliest filed claims for compensation.

A sample which is selected because of the manifestation of a residual of injury cannot ordinarily be expected to contribute usefully to a study of the probability of residuals, although it may be employed in a study of the course in time of specified residuals. In fact, however, in this instance the VA roster, or at least part of it, is capable of providing useful information with respect to the probability of specified residuals for severely injured men. The reason for this is that men who were badly wounded almost invariably received VA disability compensation for one reason or another; for lightly injured men, the VA roster is heavily biased in the direction of residuals, but for heavily injured men it is not. This subject is discussed more fully below.

B. CHARACTERISTICS OF THE SAMPLE AS DETERMINED FROM ARMY RECORDS

Age. Only 14, or 1.5 percent, of the 932 men were nonwhite. Since, in the main, these men were combat troops they were predominantly young

men, 81.0 percent being under 30 years of age at the time of the head injury (table 1).

Agent. Three-quarters of the men (693, or 74.4 percent) had been injured by shell or grenade fragments, while 132, or 14.2 percent, had bullet wounds of the head. These percentages do not reflect the proportions of all injuries to the head caused by shell fragments and bullets, respectively, because men who were killed in action or who died of wounds are not included in the study, and there is evidence (14) that bullet wounds are more often lethal than shell fragment wounds. In 33 instances, or 3.5 percent, the head injury was caused by a blunt object or resulted from a fall; and in 68 cases, or 7.3 percent, the wound was due to such miscellaneous causes as blast injury, sharp-cutting objects, etc. For six men the agent could not be determined from the records.

Table 1.—*Age at Time of Injury*

Age	Number	Percent	Age	Number	Percent
18–20	170	18.2	33–35	60	6.4
21–23	234	25.1	36–38	25	2.7
24–26	208	22.3	39–41	3	.3
27–29	143	15.3			
30–32	89	9.5	Total	932	100.0

Type of Wound. The type of wound was most commonly a penetrating wound of the brain, i.e., a compound, comminuted fracture; such wounds accounted for almost 60 percent of the cases. The other large group, in contrast, consisted of men who had scalp lacerations, usually with concussion [1] (about 22 percent). A complete breakdown is in tables 2 and 3.

Depth of Wound. In almost 30 percent of the cases, only the scalp was overtly involved in the trauma; in another 19.4 percent, there was cranial fracture without deeper penetration; and in 2.6 percent, although the dura mater was torn, there was no obvious evidence of damage to the underlying brain tissue. Thus, in a total of 482 cases, or not quite 52 percent, there was no apparent destruction of gray matter.

In 410 cases (44.0 percent) the brain itself was penetrated either by metallic foreign bodies or by bony fragments of the cranium; and in 27 additional cases, 2.9 percent, fragments penetrated into the ventricles.

Cranial Defect. There was no cranial defect in 347 instances (37.2 percent); in 22 cases (2.4 percent) the cranial defect was of the nature of a gutter wound, the size of which could not be easily defined. In 72 cases the oversea records were so vague that it was not possible to determine the size of the cranial defect which apparently was present, but in 491 cases the average diameter of the cranial defect could be determined. The most frequent size of defect (diameter) among men who survived was in the range 2 to 4 cm. (table 4).

Additional Wounds of the Head. In 111 instances (11.9 percent) a second head wound, distinct from the primary injury, was present. Of these,

[1] Concussion is rarely defined in the records; presumably it implies loss of consciousness for an appreciable period of time.

Table 2.—*Type of Injury*

Type of head wound	Number	Percent	Type of head wound	Number	Percent
Closed, without fracture	65	7.0	Compound comminuted fracture of outer table only	43	4.6
Closed, with fracture of convexity or base	1	.1	Scalp laceration	209	22.4
Closed, with depressed fracture	3	.3	Other and unspecified	6	.6
Perforating	28	3.0	Total	932	99.9
Penetrating	551	59.1			
Linear fracture, compound	26	2.8			

Table 3.—*Deepest Structure Penetrated*

Depth of penetration	Number	Percent	Depth of penetration	Number	Percent
Scalp	277	29.7	Ventricles	27	2.9
Cranium	181	19.4	Unspecified	13	1.4
Dura mater	24	2.6			
Brain	410	44.0	Total	932	100.0

Table 4.—*Size of Cranial Defect*

Average diameter of cranial defect	Number	Percent	Average diameter of cranial defect	Number	Percent
No defect	347	37.2	>4 cm	100	10.7
Gutter wound	22	2.4	Unspecified	72	7.7
≤2 cm	157	16.8			
2–4 cm	234	25.1	Total	932	99.9

78 (70.3 percent) were wounds of the scalp only; 13 (11.7 percent) involved the cranium; and in 1 case the dura mater was penetrated but apparently not the brain itself. In only four cases was brain tissue damaged by the second wound as well as by the first.

Other Wounds. Accompanying wounds of other parts were present in 497 cases, or 53.3 percent. Many of the men who had additional wounds were hit in several places. Some 497 men were coded for a total of 936 locations other than the head, so that these men suffered, on the average, at least 1.9 additional hits each. Most of these, of course, were caused by shell fragments rather than bullets. Most frequently involved in additional wounds were the upper extremities, closely followed by the lower extremities (table 5).

It should be emphasized that the group of men being discussed did not die of their injuries; presumably one of the reasons that thoracic and abdominal wounds are not more frequent in this sample is the higher fatality rate associated with wounds of these regions.

Table 5.—*Site of Other Wounds*

Location	Number	Percent of men	Location	Number	Percent of men
Ear	53	10.7	Spinal cord and vertebrae.	9	1.8
Face	144	29.0	Lower extremity	209	42.1
Neck	51	10.3			
Upper extremity	286	57.5			
Thorax	122	24.5	Total number of men with additional wounds.	497	.....
Abdomen	27	5.4			
Pelvis	35	7.0			

Traumatic Unconsciousness. Although the oversea records were often not explicit, apparently unconsciousness was usual after the head wound. For 165 men (17.7 percent) the records definitely indicated that there was no unconsciousness, and for 213 men (22.9 percent) it was impossible to decide whether or not there had been coma. However, the remaining 554 individuals (59.4 percent) were known to have been unconscious after the injury. Of these 554, almost half (202) were unconscious for 2 hours or less and 105 (19.0 percent) for more than 24 hours. However, in 144 cases (26.0 percent), although it was clear from the records that the soldier had been unconscious, it was not possible to determine how long.

Confusion. Because amnesia related to the wounding is considered an index of the severity of brain injury, an attempt to examine this factor was made by noting the state of confusion or disorientation of the patient after recovery from coma (if present); it was even less well recorded than the facts regarding unconsciousness itself. In 444 cases, or 47.6 percent, it could not be determined whether there had been confusion. In 91 instances it seemed clear that there had been no disorientation, while in 397 cases there clearly had been. However, even in these cases it was impossible to determine the duration of confusion in 142, or 35.8 percent. Of the remaining 255 men, i.e., those known to have been disoriented for a specific time period, the duration of disorientation was under 2 hours in 60 cases (23.5 percent); between 2 and 24 hours in 36 cases (14.1 percent); between 1 day and 1 week in 77 cases (30.2 percent); between 1 and 4 weeks in 60 cases (23.5 percent); and over 4 weeks in 22 cases (8.6 percent).

Complications. In 42 cases the presence of complications of the head wound could not be determined, but in the remaining 890, for which this factor was known, in 707 cases, or 79.4 percent, there were no complications. The complications most frequently noted were intracranial hemorrhage and herniation of brain tissue, each present in 88 cases, or 48.1 percent of the 183 men known to have had complications. Other complications were relatively infrequent: seven instances of cerebrospinal fistula; five of cerebrospinal rhinorrhea; three each of cerebrospinal otorrhea, frank infection, and abscess; and two instances of meningitis. There were also seven cases with other unclassified complications.

Neurological defects following the wounding were usually documented if present; in only seven cases was the neurological deficit unknown. Of the

925 for whom the records were clear, 538, or 58.2 percent, had no deficit. The most frequent defect was hemiplegia or hemiparesis (sensory or motor), which was present in 180 cases, or 19.5 percent. Hemianopsia was noted in 73 instances (7.9 percent) and aphasia in 118 (12.8 percent). The combination of hemiplegia and aphasia was quite common, being found in 63 cases, or about a third of the cases of hemiplegia and over half of the cases of aphasia. In addition to the categories mentioned above, there were 144 cases of a miscellaneous nature involving such conditions as nerve deafness, monoplegia, triplegia, quadriplegia, quadrantanopsia, facial paralysis, etc. Exactly 100 of the 144 men with miscellaneous deficits had none of those specifically categorized (hemiplegia, hemianopsia, aphasia).

Debridement. As might be expected from the fact that many of the wounds seemed rather minor, 128 men (13.7 percent) had no debridement, while for 15 others the oversea hospital records were incomplete, and it could not be determined whether or not a debridement had been done. Of the 789 men who were debrided, in 590 (74.8 percent) the debridement was done within 24 hours of the wounding and in 126 (16.0 percent) between 1 and 3 days posttrauma. However, 47 (6.0 percent) were delayed beyond 3 days, and in 26 instances the time could not be determined from the records.

Second debridements were performed on 211 men. The most frequent causes were retained fragments (91 men); infection (20 men); intracranial bleeding (16 men); abscess (12 men); and draining sinus (9 men). In 63 instances, the reason for the second debridement was not specifically coded; a host of miscellaneous reasons, such as exploration for suspected (but not discovered) fragments, secondary closures to aid healing following the first debridement, and the like, were reported.

Third debridements were done in 52 men and, indeed, 14 had 4 or more. Again, the most frequent reason for a third debridement was an attempt at removal of retained fragments (16 cases), while the second most common reason was infection (7 cases). Three men had draining sinuses, three had abscesses, and one had intracranial bleeding. The remaining 22 tertiary debridements were done for miscellaneous reasons which were not explicitly coded.

Cranioplasty. Cranioplasties were performed on 371, or 39.8 percent, of the 932 men studied. These were distributed by time with respect to the original injury as shown in table 6. In 21 instances the cranioplasty was done at a debridement, 4 times at a first debridement, 13 times at a second debridement, and 4 times at a third debridement.

Table 6.—*Time From Injury to Cranioplasty*

Time from injury to cranioplasty	Number	Percent	Time from injury to cranioplasty	Number	Percent
≤3 months	88	23.7	2–3 years	1	.3
3–6 months	180	48.5	3–4 years	2	.5
6–9 months	63	17.0	>4 years	1	.3
9–12 months	25	6.7			
1–2 years	11	3.0	Total	371	100.0

The overwhelming majority of the plates (341, or 91.9 percent) were of tantalum. However, there were 20 methacrylate plates used, as well as 5 of bone and 1 of celluloid. In two instances cartilage grafts were employed.

Intracranial Foreign Bodies. According to the records, no intracranial foreign bodies were present in 380 men; in 476 men the status of intracranial foreign bodies both before and after definitive debridement was explicitly given, but in 45 men the records are unclear in this regard. Bone fragments only were present in 237 cases; in 168 of these (70.9 percent) the fragments were entirely removed at the debridement, while in the remainder at least some of the fragments remained *in situ*. Only 31 men had metal fragments alone, and in only 8 of these (25.8 percent) were all fragments removed. Finally, 208 men had both bone and metal intracranial foreign bodies; all fragments were removed at debridement in 49 cases (23.6 percent); all bone fragments (but not all metal) were removed in 67 cases (32.2 percent); all metal foreign bodies (but not all bone fragments) were removed in 21 cases (10.1 percent); and foreign bodies of both kinds remained after the debridement in the remaining 71 cases (34.1 percent).

Postdebridement Complications. Few men had complications following debridement: 655, or 83.0 percent, of the 789 men with debridements definitely had none. Most frequent of the complications reported was frank infection (30 cases), followed by abscess (19 cases); fungus cerebri (16 cases); hematoma (14 cases); cerebrospinal fistula (12 cases); meningitis (11 cases); and cerebrospinal rhinorrhea and cerebrospinal otorrhea (3 cases each).

Cranial Roentgenograms. A report of the predebridement X-ray of the cranium was available for 448 of the 789 men who were debrided. Of these, 117 (26.1 percent) showed a normal skull. The most frequent findings were of retained bone fragments with or without accompanying metallic pieces: 227, or 50.7 percent, of the reports mentioned bone fragments. In 151 cases (33.7 percent) intracranial metallic foreign bodies were present, usually (104 cases) accompanied by bone fragments (table 7).

For 744 men it was possible to obtain the report of the last X-ray taken in any Army hospital (table 8). Only 142 (19.1 percent) were thought to show a normal skull. Bone fragments had been entirely removed in many instances. Only 77, or 10.3 percent, still had such fragments, whether with

Table 7.—*Radiographic Findings Before Debridement*

X-ray report	Number of men	X-ray report	Number of men
Normal skull	117	Retained foreign body	47
Fracture of outer table only	8	Retained bone fragments and foreign body	104
Fracture only	21	Other abnormality	1
Skull defect only	13		
Fracture and defect	14		
Retained bone fragments	123	Total	448

Table 8.—*Radiographic Findings at Last Army Examination*

X-ray report	Number of men	X-ray report	Number of men
Normal skull	142	Retained foreign body	103
Fracture of outer table only	17	Retained bone fragments and foreign body	31
Fracture only	14	Other abnormality	17
Skull defect only	131		
Fracture and defect	33	Total	744
Defect plated	210		
Retained bone fragments	46		

or without foreign bodies. However, 134 men (18.0 percent) had foreign bodies still present, accompanied only in 31 cases by bone fragments.

Neurological Complaints at Discharge From Service. More than a third of the men (376, or 40.3 percent) reported no neurological symptoms at the time of discharge from service. Records were inadequate for 15 men, but the remaining 541 men reported a host of symptoms (table 9). The term "posttraumatic syndrome" as used here implies the presence of at least 3 of the specific complaints listed in the table; the specific complaints were coded for men who claimed not more than 2 symptoms. Hence, the total number of men having headache at the time of discharge probably approximated 464.

Neurological Abnormalities. More than two-fifths (405, or 43.5 percent) of the men were free from objective neurological abnormalities when discharged from service. Records were incomplete for 14 men. The 513 men for whom abnormalities were present were classified as shown in table 10. The very large number of men coded "Other" had a wide variety of abnormalities, ranging from nerve deafness and loss of sense of smell to psychoneurosis.

Pneumoencephalograms. Pneumoencephalograms had been performed in Army hospitals on 129 of the men. These were usually made within 6 months of the head injury, although in 23 instances they were made after this time. Only 41 of the 129 men so examined were found to have normal air studies. The most frequent abnormal findings (table 11) were generalized ventricular dilatation and unilateral ventricular dilatation on the side of the lesion, each of which was observed in 28 men (21.7 percent).

Table 9.—*Neurological Symptoms at Discharge From Service*

Symptom	Number of men	Symptom	Number of men
Headache	292	Insomnia	7
Dizziness	117	Easy fatiguability	4
Impaired memory	32	Other	26
Tinnitus	29	Posttraumatic syndrome	172
Impaired mentation or lack of concentration	25	Total	541
Irritability	14		

Table 10.—*Neurological Abnormalities at Discharge From Service*

Finding	Number of men	Finding	Number of men
Hemiplegia or hemiparesis	131	Personality changes	27
Aphasia	82	Visual disturbances	20
Hemianopsia or other field defect	74	Paraparesis	6
Cortical sensory disturbances	49	Other	314
Spasticity	29	Total	513

Table 11.—*Pneumoencephalographic Findings in Military Hospitals*

Finding	Number of men	Finding	Number of men
Normal	41	Abnormal air over cortex	3
Generalized ventricular dilatation	28	Local absence of air in subarachnoid space	10
Unilateral ventricular dilatation on side of lesion	28	Inadequate filling of ventricle	13
Local ventricular dilatation on side of lesion or over cortex	34	Other abnormality	13

Electroencephalograms. Electroencephalograms (EEG's) were made more frequently than pneumoencephalograms. Tracings made during the first 6 months after injury were coded for 295 men, and during the second 6 months for 162 men. Beyond 1 year after injury the material became much thinner, but 103 examinations were made in the second year, 23 in the third year, 25 in the fourth year, and 43 were made more than 4 years posttrauma.

About half of the 295 EEG's which were done within 6 months of injury were considered to show no definite abnormality: 103 (34.9 percent) were normal, while 45 (15.3 percent) were borderline. Thus, only 147 (49.8 percent) were interpreted as definitely abnormal. Focal abnormalities were most frequently found and were present in 102 tracings; i.e., in 34.6 percent of all tracings and in 69.4 percent of those considered abnormal. Generalized abnormalities were noted in 85 instances, or 28.8 percent of the whole number, and 57.8 percent of those called abnormal. In 41 instances generalized and focal abnormalities were found in the same men, so that 40.2 percent of the 102 men with focal abnormalities had generalized abnormalities also, while 48.2 percent of the 85 men with generalized abnormalities had accompanying focal abnormalities. In 49 cases the focal abnormalities were right-sided, in 47 cases left-sided, and in 5 instances bilateral. In one case it was not possible to determine from the records on which side the focal abnormality was found.

The nature of the generalized abnormalities was usually either dysrhythmia or paroxysmal slow waves (table 12). It will be noted that 122 different abnormalities were coded for the 85 men having generalized abnormalities.

Table 12.—*Generalized EEG Abnormalities*

Finding	Number of men	Finding	Number of men
Dysrhythmia	42	Paroxysmal slow waves	37
Slow waves	13	Paroxysmal fast or spiky waves	20
Fast waves	10		

Focal abnormalities were frequently paroxysmal slow waves. These were noted for 57 men, or 55.9 percent, of the 102 men with focal abnormalities. The only other abnormality which occurred frequently was asymmetry, which was noted 28 times, accounting for 27.5 percent of focal abnormalities (table 13).

Focal abnormalities showed no remarkable tendency to cluster in particular locations (table 14).

Hyperventilation activation was employed in 219 of the 295 EEG's. Changes were not noteworthy (table 15).

Disposition. Finally, of the 932 men studied, 691, or 74.1 percent, were discharged from the Army for disability (CDD). Routine discharges were given to 201 men (21.6 percent), while 26 (2.8 percent) remained in service. In 14 instances, the circumstances of separation from service were not explicitly shown in the records.

C. THE FOLLOW-UP EXAMINATION

After the identification of those veterans who resided in one of the 4 selected areas, their names and current addresses were supplied to the clinical centers, whose responsibility it was to persuade the men to report for examination.

While practice at the four centers varied somewhat, ordinarily appointments were made for two patients each morning. The examination was divided into two parts: One consisted of the history, neurological examination, electroencephalogram, and roentgenogram of the head, while the other part consisted of the personality evaluation (Minnesota Multiphasic Personality Inventory), intelligence test (Wechsler-Bellevue), and performance test (Goddard Tactual). The two parts were administered in alternate order to the two patients seen each day—one part in the morning and the other part in the afternoon. Thus the neurologist and clinical psychologist each examined one man in the morning and one in the afternoon.

Table 13.—*Focal EEG Abnormalities*

Finding	Number of men	Finding	Number of men
Irregularity	14	Paroxysmal fast waves	13
Asymmetry	28	Spikes	7
Spindles	5	Spike and wave	1
Paroxysmal slow waves	57	Other	2

Table 14.—*Site of Focal EEG Abnormalities*

Region	Number of men	Region	Number of men
Entire side	6	Frontoparietal	5
Frontal	16	Frontotemporal	6
Parietal	12	Parietotemporal	9
Temporal	19	Parieto-occipital	5
Occipital	16	Temporo-occipital	7
Vertex	1		

The examiner had available an abstract of the Army clinical history in the form of "code sheets" (app. 2). These sheets were prepared at the Follow-up Agency and represented information culled from the clinical records of the Army hospitalization, which were available usually from the VA claims folders but sometimes from the Army's Demobilized Personnel Records Branch, St. Louis, Mo. Results of examination were entered into code booklets (app. 3), which were returned to the Follow-up Agency for cardpunching and subsequent analysis.

The practice of having the clinical centers enter examination results in coded form has both advantages and drawbacks. Reports in narrative style often lack the specificity which is needed for later coding. On the other hand, although space was provided for remarks and the examiners were encouraged to enter notes of conditions which were not adequately represented by the code positions, it did happen that certain cases were given unusual combinations of code positions which were not always immediately explicable. Weighing the advantages and disadvantages, it is considered that the coding of cases by the clinician worked well.

The initial approach to the patients was made by the examiner, who wrote a letter to the veteran in an effort to gain his cooperation (app. 4). Repeated correspondence and telephone calls sometimes failed to induce cooperation. American Red Cross social workers rendered invaluable assistance in reducing the number of nonparticipants by visiting the most reluctant men, explaining to them the purpose of the study, and arranging transportation. Not infrequently veterans who had made appointments under the pressure of repeated urging failed to appear, and further efforts

Table 15.—*Results of EEG Activation*

Finding	Number of men	Finding	Number of men
No change	130	Spike and wave complex	5
Generalized slowing, within normal limits	36	Focal spiking	5
Generalized fast, within normal limits	5	Focal slow wave	16
Generalized subnormal slowing	18	Other alterations	2

Table 16.—*Results of Solicitations for Examinations*

Center	Number allocated	Number examined	Percent
Baltimore	316	257	81.3
New York	300	221	73.7
Boston	166	152	91.6
Los Angeles	150	109	72.7
Total	932	739	79.3

were necessary. Despite all efforts, however, a certain proportion of men could not be examined (table 16).

There is clear cause for some concern over the fact that just over 20 percent of the veterans who had been selected for study could not be induced to cooperate. It seemed plain that a variety of motivations were at work in different men, but the possibility could not be excluded that the men who did not cooperate were quite different from those who were examined, and that the results of the study might be seriously affected. In order to obtain some measure of the magnitude of error so introduced, the VA claims folders of the men not examined were reviewed. Claims folders contain not only information concerning payments for disability compensation but also copies of the reports of physical examinations on which rates of payment are based. It was thought that comparison of rates of disability compensation of the nonexamined men with those for a sample of men who were examined, coupled with comparisons based on the original Army clinical records, would provide the basis for an estimate of the importance of bias attributable to nonresponse in the survey.

D. DIFFERENCES BETWEEN MEN WHO REPORTED FOR FOLLOW-UP EXAMINATIONS AND THOSE WHO DID NOT

1. Characteristics of the Injury and Status at the Time of Separation From Service

Analysis of differences between men who could be persuaded to report for the follow-up examination and those who declined revealed some interesting contrasts, which were not always immediately explicable. Table 17 presents an extract of these data.

The men who were examined did not differ in age from those who were not examined, but they more often had penetrating or perforating wounds, as opposed to scalp lacerations; they more often had large cranial defects, definite penetration of brain tissue, and epileptic seizures before discharge from the Army. However, they also were more often free of neurological symptoms and objective abnormalities at discharge.

2. Information From VA Claims Folders

Of the 616 men allocated to the Baltimore and New York centers, 473 had VA disability claims. Of these men, 359 were examined, while 114 were not. Since the VA claims folders contain the report of examination on which the disability rating is based, it was decided to compare the re-

spondents and nonrespondents with respect to these examination reports to learn how much importance might attach to neurological status at follow-up in influencing men to report for examination or to decline. A summary of the comparisons is presented in table 18. It was surprising to find that the 2 groups of men seemed to differ very little with respect to follow-up status. Only a single factor, of those examined, differed significantly between the 2 groups of men: While 30 percent of examined men were paralyzed or had impaired motor power, the corresponding figure for non-examined men was only 20 percent. For many other observations, differences, even if not statistically significant, were in the direction suggesting that the more severely impaired men were more likely to report for examination than those with little disability. Thus, less than 16 percent of nonrespondents had ever had a fit, while 22 percent of respondents had had at least 1 seizure. Average percentage disability was slightly higher for the examined men, who also had somewhat more aphasia and muscle atrophy. On the other hand, nonexamined men more often complained of impaired hearing, were more often reported to show apathy or anxiety on psychiatric examination, and were more often unemployed.

Table 17.—*Comparison of Respondents With Nonrespondents; Date From Army Clinical Records*

Characteristic	Respondents		Nonrespondents	
	Number	Percent	Number	Percent
Total	739	100.0	193	100.0
≤20 years old at injury	133	18.0	37	19.2
30+ years old at injury	142	19.2	35	18.1
With perforating wound	24	3.2	4	2.1
With penetrating wound	448	60.6	103	53.4
With scalp laceration	158	21.4	51	26.4
No cranial defect	264	35.7	83	43.0
Cranial defect ≥4 cm. diameter	85	11.5	15	7.8
No penetration of brain tissue	366	[1] 49.5	116	[1] 60.1
Definite penetration of brain	363	[1] 49.1	74	[1] 38.3
No fits prior to separation	578	78.2	162	83.9
At least 1 fit in service	161	21.8	31	16.1
Posttraumatic syndrome at separation	127	17.2	45	23.3
Other or unknown symptoms at separation	303	41.0	81	42.0
No neurological symptoms at separation	309	41.8	67	34.7
No neurological abnormalities at separation	325	44.0	80	41.5
Hemiplegia or hemiparesis	110	14.9	21	10.9
Aphasia	71	9.6	11	5.7
Field defect	54	7.3	20	10.4

[1] Percentage difference between respondents and nonrespondents significant at $P = .05$ level.

Table 18.—*Comparison of Respondents With Nonrespondents; Data From VA Claims Ratings Examinations*

Baltimore and New York Cases With Claims

Information from claims folders	Respondents		Nonrespondents	
	Number	Percent	Number	Percent
Total	359	100.0	114	100.0
Clinical symptoms:				
Abnormal speech	42	11.7	10	8.8
Visual complaints	95	26.5	28	24.6
Parasthesias	64	17.8	26	22.8
Tinnitus	75	20.9	22	19.3
Impaired hearing or deafness	44	12.3	21	18.4
Motor weakness	79	22.0	25	21.9
Psychiatric status:				
Normal	143	39.8	39	34.2
Apathy	14	3.9	8	7.0
Anxiety	120	33.4	42	36.8
Neurological examination:				
Aphasia	38	10.6	9	7.9
Impaired facial sensation	19	5.3	7	6.1
Impaired facial movement	34	9.5	12	10.5
Paralyzed or impaired motor power	109	[1] 30.4	23	[1] 20.2
Muscle atrophy	44	12.3	9	7.9
Impaired superficial sensation	48	13.4	17	14.9
Impaired deep sensation	12	3.3	2	1.8
Romberg positive	14	3.9	5	4.4
Gait ataxic or hemiplegic	30	8.4	6	5.3
Work status: Unemployed	74	20.6	30	26.3
VA disability rating:				
≤60 percent	172	47.9	60	52.6
70–100 percent	185	51.5	52	45.6
Epileptic seizures	79	22.0	18	15.8

[1] Percentage difference between respondents and nonrespondents significant at $P=.05$ level.

3. Data From Telephone Interview

For those patients who would not report for examination, attempt was made by telephone interview to obtain certain information in regard to status at the time of follow-up. The most significant findings are shown in table 19.

The differences were small with respect to employment, but with respect to clinical symptomatology were quite large. Respondents were much more likely to present complaints characterized as a posttraumatic syndrome; the nonrespondents usually had but a single complaint, usually headache, and more nonrespondents were free of symptoms. Similarly, fits were less common in the nonrespondents than in respondents, although for about 5 percent of nonrespondents it was not clear from the telephone interview whether the man had epilepsy or not. In particular, a significantly greater proportion of the respondents than the nonrespondents had had more than a single fit.

4. Conclusions

It seems plain that the men who failed to report cannot be simply characterized; they appear to have, as a group, somewhat less objective evidence of neurological disability than those who were examined, but also to have somewhat more in the way of personality deviation. However, it seems unlikely that differences of the magnitude found here would seriously affect the conclusions of the study.

E. FOLLOW-UP RESULTS AND VARIABILITY BETWEEN CENTERS

As a preface to discussion of the results of the clinical examinations, it is useful to consider the difficulties which may arise from the fact that observations were made by four different clinical groups.

Table 19.—*Comparison of Respondents With Nonrespondents; Data From Telephone Interview for Nonrespondents*

Information from telephone interview	Respondents		Nonrespondents for examination but with telephone interview	
	Number	Percent	Number	Percent
Total	739	100.0	116	100.0
Employment status:				
In hospital	8	1.1	.....	.....
Unemployed or occasional jobs	110	14.9	20	17.2
Employed regularly	577	78.1	96	82.8
Clinical symptomatology:				
Posttraumatic syndrome	419	[2]56.7	22	[2]19.0
Other symptoms or unknown	256	[2]34.6	79	[2]68.1
No symptoms	64	8.7	15	12.9
Epilepsy:				
No fits at any time	532	72.0	88	75.9
Borderline attacks only	10	1.4	3	2.6
One definite attack	26	3.5	1	.9
More than 1 definite attack	169	[1]22.9	18	[1]15.5

[1] Percentage difference between respondents and nonrespondents significant at P=.05 level.
[2] Percentage difference between respondents and nonrespondents significant at P=.01 level.

The subject of what has been called "observer error" was brought to the fore in striking fashion, insofar as clinical observations are concerned, by the work of Birkelo et al. (18), with respect to the reproducibility of interpretations of chest X-rays. By now most medical scientists have come to realize that observations called by the same names by different observers may represent quite different measurements. In clinical medicine it is seldom that the details of the process of observation are specified to that degree of exactitude which would be needed for the reproducibility of the observation by different observers or even by the same observer at different

times. This is sometimes summed up by saying that there is a large "subjective element" in many observations.

In a very real sense "observer error" is often not an apt designation for the phenomenon in question. Error, according to one definition at least, is: "The difference between an observed or calculated value and the true value." While this may represent a valid point of view for considering, say, the values for hemoglobin reported by different laboratories on aliquots from a single specimen, it seems not to be germane to the situation when presumably equally competent observers differ as to, say, whether a given patient's liver is or is not palpable. There are a host of clinical variables which share this characteristic, namely, that there is no "true" value, but each observer measures according to his own private scale, which may or may not be susceptible of calibration against the scales of others. This is especially true of those observations which depend wholly or in part upon an interaction between the observer and the patient; the personalities of different observers react differently with that of the patient.

Despite all this, clinicians continue to make observations and to find them useful. This is natural enough. Each individual, in the course of time, creates a body of experience appropriate to his own measuring rods, and judges each new observation, whether explicitly or implicitly, by this yardstick. Difficulties become of importance when he attempts to communicate to others, and the situation becomes critical when the two parties to the communication erroneously believe that because they employ the same name they are talking about the same thing. This difficulty was anticipated in this study and, in an attempt to minimize it, the authors visited each center at the beginning of the follow-up, reviewing the desired clinical interrogation and examination and the criteria for coding. In spite of this briefing, the centers seemed to have maintained their individuality.

From this point of view, it was essential to examine the evidence regarding the degree to which the four participating clinical centers employed comparable instrumentation (in the broadest sense) in making the follow-up observations. The ideal, of course, would have been to present a sample of patients to the four centers in turn, and to compare the results. That being impossible, the next best is to subject comparable samples of men to examination at the four centers and then to compare the results. That has been done below in the course of the description of the results of the examination. However, data from centers which apparently did not differ have been combined.

The Baltimore center began pilot work about 2 years prior to the entrance into the study of the other three centers. Thus, most men examined in Baltimore were 5 to 8 years after injury, while at the other centers the intervals were 7 to 9 years (table 20).

More than one-fourth of the Los Angeles patients gave a history of having been knocked unconscious prior to the head injury which brought them into this study; only 6.8 percent of the men seen at the other centers related a history of having been knocked out. This gross difference is unexplained, but seems unlikely to be caused by a real difference of this magnitude in the previous histories of the men examined. We must conclude that the examiner at Los Angeles inquired into this question in a more searching way than did the others (table 21).

Table 20.—*Time From Injury to Follow-up Examination*

Time after injury	Number of men		Time after injury	Number of men	
	Baltimore	Other centers		Baltimore	Other centers
3–4 years	1	.....	8–9 years	5	148
4–5 years	6	.....	9–10 years	.....	12
5–6 years	129	1			
6–7 years	66	11	Total	257	482
7–8 years	50	310			

The information with respect to previous illnesses (table 22) is rather discouraging from the point of view of center variability. The proportion (percent) reported as "none" was, respectively, 87.2, 94.6, 75.5, and 79.8. This variation far exceeds what the blind workings of chance alone might be expected to produce. Again, 13.2 percent of the men examined at Boston gave a history of enuresis, far higher than at any other center. Allergic conditions were noted for only a single man at the New York center, but were not uncommon at the other centers. It seems clear that the ordinary history, although taken with care, produces information of dubious validity.

In Boston 68.4 percent denied nervous disorders in the family, while at the other centers the combined proportion was 85.7 percent—strikingly different. Some of the particular differences which were quite marked were: "fainting," 6.6 percent and 1.7 percent, "neurosis," 13.2 percent and 2.9 percent, "psychosis," 6.6 percent and 3.4 percent, and "psychopathic traits," 2.6 percent and 0.2 percent (table 23).

Some evidence of difference between the centers with respect to reporting of work status may be seen in the relatively large numbers coded at Los Angeles for school or rehabilitation training and at Baltimore for "occasional jobs" (table 24).

Certain differences between the centers in "general work progress" are readily apparent (table 25); the Boston center classified many more men as "none" or "regression" (71.7 percent) than did any other center, while

Table 21.—*History of Previous Head Injuries*

Type of injury	Number of men		Type of injury	Number of men	
	Los Angeles	Other centers		Los Angeles	Other centers
None significant	77	580	Severe head injury without sequelae	2	4
Knocked out once or twice	19	42	Severe head injury with sequelae	.....	1
Knocked out more than twice	10	1	Unknown	2	2
			Total men	109	630

Table 22.—*History of Previous Illness*

Condition reported	Number of men			
	Baltimore	New York	Boston	Los Angeles
None, other than uncomplicated childhood diseases	224	209	115	87
Childhood diseases with neurological complications	2	1	3	2
Meningitis	4	1	.....	1
Febrile convulsions	1	.....	3	.....
Allergic conditions	16	1	12	15
Enuresis	2	6	20	.....
Temper tantrums	.....	2	1	.....
Breath holding	.....	1	.....	.....
Syphilis	6	1	.....	3
Other	.....	1	.....	.....
Unknown	2	1	2	2
Total men	257	221	152	109

New York so classified relatively few (30.3 percent). At New York many men were coded for "advancement" (61.1 percent), while only a few (15.9 percent) were so classified at Boston. It seems clear, retrospectively, that such words as "advancement" are much too imprecise for comparable use by different observers.

The Boston center tended not to classify men at the high end of the home adjustment scale as frequently as the other centers: Only 55.3 percent were considered to be happy and content as against 74.4 percent at the other centers. On the other hand, the distinction between home adjustments which are "happy and content" and those which are merely "reasonably satisfactory" seems arbitrary (table 26).

The characterization of social adjustment did not appear to be more consistent between centers than were the other judgment items (table 27). Almost 90 percent of men at Baltimore were said to "mix well," while at Boston this was said of less than two-thirds. As an isolated observation, this might be ascribed to the proverbial Boston reserve, but it more probably represents yet another instance of lack of agreement between examiners

Table 23.—*Familial History of Nervous Disorders Other Than Epilepsy*

Condition reported	Number of men		Condition reported	Number of men	
	Boston	Other centers		Boston	Other centers
None	104	503	Other nervous diseases	10	15
Fainting	10	10	Unknown	3	8
Migraine	4	18			
Neurosis	20	17			
Psychosis	10	20	Total men	152	587
Psychopathic	4	1			

as to the meaning of the words used in defining the classifications. Few men were considered at any center to be antisocial, but the proportions characterized as "feels out of place" or "asocial" vary markedly. Thus, at Baltimore twice as many men were labeled asocial as were given the former designation, while at New York the ratio was more than reversed.

Table 24.—*Work Status at Time of Examination*

Work status	Number of men		
	Baltimore	Los Angeles	Boston and New York
None, unemployed	27	9	53
Army or VA hospital	1	4	3
School or rehabilitation training	15	13	13
Return to former job, or advanced in former job	56	20	72
Former job modified	20	9	21
New work	121	51	207
Occasional jobs	17	2	2
Unknown	.....	1	2
Total	257	109	373

Table 25.—*Changes in Work Status*

Trend	Number of men			
	Baltimore	New York	Boston	Los Angeles
None or regression	107	67	109	56
School and advancement	52	17	18	9
Advancement	97	132	24	41
Unknown	1	5	1	3
Total	257	221	152	109

Table 26.—*Assessment of Home Adjustment*

Adjustment	Number of men		Adjustment	Number of men	
	Boston	Other centers		Boston	Other centers
Happy and content	84	437	Unhappy	9	26
Reasonably satisfactory	45	75	Unknown	3	12
Depressed	2	9	Total	152	587
Restless	9	28			

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Table 27.—*Assessment of Social Adjustment*

Adjustment	Number of men			
	Baltimore	New York	Boston	Los Angeles
Mixes well and quite satisfactorily	230	171	97	75
Feels out of place	8	29	28	12
Asocial	16	13	20	17
Antisocial	1	2	2	1
Other and unspecified	2	6	5	4
Total	257	221	152	109

Assessment of economic adjustment was reasonably uniform at the low end of the scale, but apparently the distinction between "satisfactory" and "fair" adjustments was variously interpreted by the raters (table 28). The proportions called "fair" at the four centers were, respectively, 22.2 percent, 64.3 percent, 29.6 percent, and 41.3 percent.

The clinical symptomatology was reported more uniformly by the four centers than were many of the clinical observations (table 29). The Los Angeles center much more frequently characterized men as having "impaired mentation" or "impaired memory" than did the other centers; Boston and Los Angeles characterized few men as without symptoms. One feature complicating analysis of the clinical symptomatology was the practice of coding as "posttraumatic syndrome" any combination of three or more specific complaints, so that the fact that the Boston center reported a smaller proportion of men complaining of headache must be coupled with the fact that this center also reported the highest proportion with posttraumatic syndrome.

Complaints with respect to speech, vision, and hearing were not frequent, and specific tables are therefore omitted. It was apparent that the centers did not define comparably anomia, global aphasia, tinnitus, impaired hearing, blurred vision, and field defects.

Complaints of paresthesias and motor weakness did not appear to vary remarkably between centers. The number of men who complained of sensory and motor disturbances was about the same (table 30).

Table 28.—*Assessment of Economic Adjustment*

Adjustment	Number of men			
	Baltimore	New York	Boston	Los Angeles
Satisfactory	151	55	88	49
Fair	57	142	45	45
Dissatisfied	40	23	17	12
Unknown	9	1	2	3
Total	257	221	152	109

Table 29.—*Complaints at Follow-up Examination*

Symptom	Number of men			
	Baltimore	New York	Boston	Los Angeles
No symptoms	30	20	8	6
Headache	71	50	25	37
Dizziness	17	13	8	11
Irritability or nervousness	21	17	23	9
Impaired mentation or lack of concentration	1	7	2	12
Impaired memory	4	7	1	8
Easy fatiguability	9	15	4	.....
Insomnia	7	2	2	.....
Posttraumatic syndrome	140	131	98	50
Unknown	1	1	2	1
Total men	257	221	152	109

Table 30.—*Sensory and Motor Complaints*

Location to which complaints refer	Number of men		Location to which complaints refer	Number of men	
	Somato-sensory paresthesias	Motor weakness		Somato-sensory paresthesias	Motor weakness
None	538	513	Other, unknown, and complaints not due to head wound	52	39
Face	12	17			
Arm	46	45			
Leg	22	27			
Arm and leg	40	72			
Entire side	30	32	Total men	739	739

The Los Angeles center was clearly less sensitive to complaints referable to other systems with the single exception of the peripheral nervous system. The Boston center, on the other hand, reported many complaints with respect to the alimentary and respiratory systems. Other discrepancies include the emphasis at Baltimore on the autonomic system and at New York with respect to the endocrine system (table 31).

The four centers differed markedly in their reports of the mental status of the men (table 32). The Boston center concluded that two-thirds of their patients had abnormalities of mental state not related to the cerebral injury, whereas other centers had few such cases. The New York center reported 22 percent of their men as having impaired judgment or mentation, more than three times the number of the other centers except Los Angeles, which reported 15 percent.

As might be expected, the tests of memory and the serial 7 subtraction test (table 33), being relatively objective, varied little by center in contrast with some of the more subjective items which have been considered.

Table 31.—*Symptoms Referable to Other Systems*

System	Number of men			
	Baltimore	New York	Boston	Los Angeles
None	197	191	91	101
Alimentary	25	13	37	1
Respiratory	6	3	11	.....
Cardiovascular	2	1	8	.....
Peripheral vascular	1	1	3	.....
Genitourinary	12	1	6	1
Endocrine	.....	4	.....	.....
Autonomic	12	.....	2	.....
Peripheral nervous	7	3	6	4
Other and unknown	6	8	8	3
Total men	257	221	152	109

The Baltimore center noted an aphasic abnormality in about 6 percent of examinations, New York and Boston in about 10 percent, and Los Angeles in about 18 percent (table 34).

The New York center most frequently found visual abnormalities (only 76 percent of men had normal vision); Los Angeles occupied middle ground (about 83 percent normal), while Baltimore and Boston found relatively little abnormality (about 88 percent normal) (table 35). At Los Angeles almost all defects found consisted of homonymous hemianopsia, while at the other centers the defects noted were more varied. The difference among centers in the proportions reported to have homonymous hemianopsia is statistically significant at the 5 percent level.

The examination of variability between centers, demonstrating differences between examiners, not only for subjective judgments but also for

Table 32.—*Examiner's Assessment of Mental Status*

Status	Number of men			
	Baltimore	New York	Boston	Los Angeles
Normal	228	147	35	78
Uncooperative	.....	1	1	1
Confusion	2	.....	.....	8
Depression	14	4	.....	4
Euphoria	2	9	.....	1
Wisecracking	2	1	.....	2
Impaired judgment or mentation, or lack of concentration	3	48	10	16
Irritability	2	12	2	6
Abnormal finding not due to head wound	6	10	105	.....
Other and unknown	.....	4	3	1
Total men	257	221	152	109

Table 33.—*Results of Memory and Serial 7 Test*

Number of errors	Number of men		Number of errors	Number of men	
	Memory	Serial 7 test		Memory	Serial 7 test
None	204	333	8	9	8
1	244	129	9	3	7
2	127	75	≥10	17	14
3	68	53	Not done or unsatisfactory	14	39
4	22	39			
5	14	16			
6	8	15	Total	739	739
7	9	11			

Table 34.—*Assessment of Aphasia at Follow-up*

Type	Number of men		
	Baltimore	New York and Boston	Los Angeles
None	242	337	89
Expressive	2	14	6
Nominal	.....	5	1
Global	7	12	9
Combinations	3	2	.....
Other and unknown	3	3	4
Total	257	373	109

Table 35.—*Assessment of Vision at Follow-up*

Vision	Number of men		
	Baltimore and Boston	New York	Los Angeles
Normal	359	168	90
Blind	4	3	.....
Vision impaired	9	13	1
Homonymous hemianopsia	28	24	14
Other, unknown, and defects not due to head wound	14	17	5
Total men	409	221	109

presumably objective evaluations, is not reassuring. To be sure, the inference of differences between examiners rests on the assumption that the men examined at the various centers were essentially alike, and this assumption is probably not wholly valid. This is illustrated by the consideration of the hemianoptic disturbances, the demonstration of which is essentially objective. It seems more reasonable to conclude that the differences demonstrated for this observation are attributable more to variability in the samples of men studied at the centers than to differences in interpretation of examiners. On the other hand, the very great variation in the proportions of psychiatric diagnoses assigned by the centers hardly allows any conclusion but that standards varied between examiners. This is not unexpected, since the examiners in the various centers had different specialty backgrounds, representing psychiatry, neurology, and neurological surgery.

The existence of this variability must be borne in mind in connection with the analyses to be reported below and, where especially pertinent, will be referred to explicitly.

F. THE INJURY SCORE

It seems clear, in an intuitive sense, that head wounds vary with respect to a parameter that may be called "severity," but just how to assess this is not immediately apparent. Evidently, from the standpoint of severity, head wounds are multidimensional; such aspects as *which* organs or kinds of tissue have been damaged, and *how much* tissue of a particular kind has been destroyed, are not simply additive.

The necessity in this study for finding some relatively simple way to characterize the severity of each head wound arises from the fact that the extent of damage is correlated with every aspect of treatment and prognosis. Thus, if it be inquired whether a cranioplasty increases the probability of subsequent epilepsy, there has to be taken into consideration that (*1*) the more severe the wound, the greater the chance that a cranioplasty was done, (*2*) the more severe the wound, the greater the chance that the patient has had fits; and, therefore, (*3*) the incidence of epilepsy is higher in men with cranioplasty than in men without.

Since what is desired is a measurement of the direct effect of cranioplasty on epilepsy, *independent* of correlations with wound severity, it seemed necessary to create a simple characterization of the latter. Tabulations might be controlled by such a measure, and groups of men whose wounds were of equivalent average severity might be defined. For these purposes an "Injury Score" has been devised.

The general method used to create the scoring system was to select an end-result criterion, and by this to evaluate the apparent relative importance of the different aspects of severity, and so to assign a score to every particular category in each aspect. The resulting scores were added together for each patient to form the "Injury Score."

The specific end result employed was the presence or absence of epileptic seizures during the first 2 years after injury. This information was generally available, even for men who were not examined. This criterion was assessed against each of 15 items descriptive of the injury. The different categories in each item were then combined into a smaller number of

groups rather arbitrarily, the choice depending not only on what seemed to be a natural way of grouping, but also on the number of cases available for each category and the variation in the proportion with epilepsy. The logarithm of the percent with epilepsy was then used as a raw score for the category. The final score for the category was obtained from the raw score by first multiplying by the ratio of the mean difference between men with and without epilepsy to the variance of the raw scores in all 932 men, and then subtracting a suitable constant so that the category corresponding to the least severe injury would have a score of zero. The procedure may be expressed algebraically more understandably than in prose:

Let r_i be the percent with epilepsy in the i^{th} category.
Then z_i is the raw score, where $z_i = \log r_i$.
Let n_i and m_i be the numbers of men with and without epilepsy, respectively, in category i. If

$$\bar{z}_1 = \frac{\Sigma n_i z_i}{\Sigma n_i} \qquad \bar{z}_2 = \frac{\Sigma m_i z_i}{\Sigma m_i}$$

then

$$\bar{z} = \frac{\Sigma[(n_i + m_i) z_i]}{\Sigma(n_i + m_i)};$$

and

$$\sigma^2 = \frac{\Sigma[(n_i + m_i) z_i^2]}{\Sigma(n_i + m_i)} - \bar{z}^2$$

The final score, χ_i, is given by

$$\chi_i = \frac{(z_i - z_0)(\bar{z}_1 - \bar{z}_2)}{\sigma^2}$$

where z_0 is an arbitrary constant.

The categories that were established, and the final scores for them are shown in table 36, while the final result of all these arithmetic procedures is displayed in table 37. The Injury Score, which is the sum of the 15 individual item scores, varied between 0 and 750. Men whose scores fell into the low range (below 250) had an incidence of epilepsy of only 7.4 percent, while in the intermediate range (250–499) the incidence was 24.2 percent, and in the high range (500 and over) it was 50.9 percent. Moreover, among men with low scores who did have fits, only about one-tenth had focal attacks; among men with high scores the majority (58 percent) had focal attacks.

It is worth remarking that the particular scoring system used here is dependent on the choice of epilepsy as an end-result criterion. It is quite possible that if another criterion had been used the scores given in table 36 might have been quite different. Since the main interest of the present study is in posttraumatic epilepsy, the scoring system derived here seems appropriate to our purposes. It may or may not be suitable to other purposes.

It should be noted that the data used in constructing the scores were those available in military records. The significance assigned to each factor by the scoring system depends not only on the intrinsic importance of the factor but also on the accuracy with which the records reflect the true condition of the subject.

Table 36.—*Scoring System for Injury Scores*

Characteristic of injury	Total number of men	With epilepsy		Score
		Number	Percent	
Type of wound:				
Closed head injury without fracture, fracture of outer table only, scalp laceration, other or unknown	323	27	8.4	
Closed head injury with fracture of convexity or base, or compound noncomminuted (linear)	27	7	25.9	44
Closed head injury with depressed skull fracture or compound comminuted fracture (penetrating wound)	554	150	27.1	46
Perforating wound	28	8	28.6	49
Agent:				
Other (falls) and unknown	74	8	10.8	0
Bullet, shell fragment, blunt object	858	184	21.4	26
Regions wounded:				
Vertex, basal, sinuses, mastoid, orbit, sphenoid, other or combinations of these	94	10	10.6	0
Temporal or occipital only	155	20	12.9	1
Frontal alone, or temporal or occipital together or with preceding	143	22	15.4	21
Frontal and other, but not parietal	74	14	18.9	25
Parietal alone	213	54	25.4	37
Parietal and other	253	72	28.5	46
Mean diameter of cranial defect:				
No defect	347	35	10.1	0
≤2 cm. or unknown	229	55	24.0	38
2+through 4 cm	234	61	26.1	42
>4 cm	100	32	32.0	52
Other (gutter wounds)	22	9	40.9	66
Depth:				
Scalp, cranium or dura mater (after debridement)	471	47	10.0	0
Cranium, before debridement	20	4	20.0	31
Dura mater before debridement or brain	414	130	31.4	54
Ventricle	27	11	40.7	69
Second head wound:				
None, or not involving frontal or temporal regions	882	174	19.7	0
Of frontal or temporal region	50	18	36.0	41
Immediate complications:				
None	707	116	16.4	0
Hematoma, frank infection, abscess, other and unknown	126	39	31.0	32
Meningitis or cerebrospinal fluid leak	11	4	36.4	46
Fungus cerebri	88	33	37.5	50
Period of unconsciousness:				
None, or <2 hours, or unknown	578	88	15.2	0
2–24 hours, or unconscious for unstated time	204	55	27.0	35
1–7 days, or initially conscious, with later coma	131	42	32.1	42
>1 week	19	7	36.8	50
Period of confusion:				
None	91	10	11.0	0
≤24 hours or unknown	540	91	16.9	21
1–3 days or confused for unstated period	184	46	25.0	43
>3 days	117	45	38.5	70

Table 36.—*Scoring System for Injury Scores*—Continued

Characteristic of injury	Total number of men	With epilepsy		Score
		Number	Percent	
Neurological deficit at time of wound:				
None	538	68	12.6	0
Hemianopsia or unspecified deficit alone	152	30	19.7	35
Aphasia, with or without hemianopsia	55	17	30.9	51
Hemiplegia, without aphasia or hemianopsia	107	40	37.4	61
Hemiplegia with aphasia or hemianopsia or both	80	37	46.3	76
Extent of first debridement:				
None, or involved scalp or skull only	441	44	10.0	0
Involved dura mater	59	13	22.0	35
Unstated	53	14	26.4	44
Involved brain	361	113	31.3	53
Involved ventricle	18	8	44.4	74
Intracranial foreign bodies:				
No foreign bodies, or no debridement	387	48	12.4	0
Unstated	46	9	19.6	21
Bone or metal or both, completely removed at debridement	236	56	23.7	30
Bone or metal or both, incompletely removed at debridement	263	79	30.0	43
Complications of debridement:				
No debridement	143	17	11.9	0
None, or cerebrospinal fluid leak only	669	131	19.6	25
Hematoma, frank infection, abscess, meningitis, fungus cerebri or other	120	44	36.7	66
Second debridement.				
Not done	716	128	17.9	0
Done for retained foreign bodies or unspecified reason	146	34	23.3	16
Done for infection, draining sinus, intracranial bleeding or abscess	70	30	42.9	61
Additional debridements beyond second:				
Not done	880	174	19.8	
Three debridements done	38	10	26.3	0
>3 debridements	14	8	57.1	39

G. ROSTER-INJURY GROUPS

As stated earlier, there seemed reason, initially, to fear that rosters drawn from VA compensation lists might be biased in the direction of residual impairment. The Injury Score, previously described, was used in an attempt to determine to what extent cases drawn from the two rosters might differ as to the probability of residual impairment, given wounds of equal severity.

With respect to the incidence of epilepsy, it was quite apparent that among men with low scores (relatively mild injury), those drawn from the VA rosters were much more likely to have fits than those who came from the Army admissions lists (table 38). For men whose scores were 250 or more, however, there seemed to be essentially no difference. Between 250

Table 37.—*Relation Between Injury Score and Incidence of Epilepsy*

Injury score	Without epilepsy		With epilepsy		With focal attacks		With grand mal		Percent of men with this score who have epilepsy		Percent of men with epilepsy who have focal attacks
	Number	Percent	Number	Percent	Number	Percent	Number	Percent			
Total	740	100.0	192	100.0	65	99.9	106	100.0	20.6		33.9
0–49	54	7.3	3	1.6	.....	.....	2	1.9	5.3		
50–99	124	16.8	5	2.6	.....	.....	3	2.8	3.9		
100–149	83	11.2	11	5.7	1	1.5	4	3.8	11.7	7.4	10.7
150–199	41	5.5	7	3.6	2	3.1	4	3.8	14.6		
200–249	48	6.5	2	1.0	.....	.....	2	1.9	4.0		
250–299	45	6.1	8	4.2	2	3.1	5	4.7	15.1		
300–349	76	10.3	18	9.4	6	9.2	7	6.6	19.1		
350–399	72	9.7	27	14.1	2	3.1	14	13.2	27.3	24.2	27.1
400–449	76	10.3	30	15.6	9	13.8	15	14.2	28.3		
450–499	66	8.9	24	12.5	10	15.4	18	17.0	26.7		
500–549	29	3.9	26	13.5	13	20.0	17	16.0	47.3		
550–599	13	1.8	20	10.4	14	21.5	9	8.5	60.6		
600–649	9	1.2	3	1.6	1	1.5	1	.9	25.0	50.9	57.9
650–699	3	.4	7	3.6	4	6.2	5	4.7	70.0		
700–750	1	.1	1	.5	1	1.5	.....	.....	50.0		

Table 38.—*Comparison of VA and Army Rosters as to Incidence of Epilepsy Within 2 Years of Injury in Relation to Injury Score*

Injury score	Total	From VA rosters	From Army rosters	Total	From VA rosters	From Army rosters
	Number of men			Percentage fits in 2 years		
Total	932	469	463	20.6	19.2	22.0
0–99	186	107	79	4.3	7.5	.....
100–199	142	105	37	12.7	15.2	5.4
200–299	103	58	45	9.7	10.3	8.9
300–399	193	73	120	23.3	20.5	25.0
400–499	196	68	128	27.6	20.6	31.3
500–599	88	43	45	52.3	58.1	46.7
≥600	24	15	9	45.8	40.0	55.5

and 500, the incidence of epilepsy seemed to vary but little, but rose rather sharply for scores of 500 or more.

On the basis of these findings it seemed appropriate to divide the men into four groups (table 39), henceforth referred to as the Roster-Injury groups (or R–I groups), defined as follows:

R–I group 1: From Army roster, injury score 0–249.
R–I group 2: From VA roster, injury score 0–249.
R–I group 3: Both rosters, injury score 250–499.
R–I group 4: Both rosters, injury score 500–750.

These four groups were subsequently used throughout the analysis in an attempt to free correlations from at least the grossest artifacts. Thus, as will be seen, the frequency of abnormal electroencephalographic tracings increases as one passes from R–I group 1 to group 4, as does the frequency of epilepsy. Hence, if the material be taken *as a whole,* it would be impossible to know to what extent correlations between these two factors were fortuitous and to what extent the brain wave tracings were actually revealing inherent characteristics related to epilepsy. No *post hoc* survey, such as the present study, can ever hope really to free itself of such difficulties, but it is believed that here it has been possible to minimize these disturbances by means of controlling tabulations by the R–I groups.

Table 39.—*Composition of R–I Groups*

R–I group	Number of men	Incidence of epilepsy in 2 years	R–I group	Number of men	Incidence of epilepsy in 2 years
		Percent			*Percent*
1	135	1.5	4	112	50.9
2	243	10.7			
3	442	24.2	Total	932	20.6

Chapter III

THE CHRONIC NEUROLOGICAL DEFICIT

A. INTRODUCTION

In general, reparative and recuperative processes tend to reduce a neurological defect for about 2 years. Rehabilitation and special training may be effective over a somewhat longer period of time, but usually in an adult the neurological dysfunction reaches a plateau after 3 to 5 years and in the ensuing next few years regresses very little. It seems probable that an examination made 7 to 10 years after injury will represent the relatively stable state of the nervous system—in other words, the end result of the natural healing processes and the various special therapies. To evaluate this state, all aspects of nervous function must be investigated in order to assess the overall condition of the individual. In this study special emphasis has been placed on certain aspects, such as the presence of headaches or convulsions, but it is planned to present in this chapter the neurological status at the time of the follow-up examination, and, by correlating the abnormalities with the varying conditions operating at the time of injury and shortly thereafter, to determine the factors which may be important in their production.

B. GENERAL SURVEY

A glance at the summary table 40 of the chronic neurological deficits indicates that even years after injury few men who suffered a head wound considered themselves perfectly normal, although almost half had no abnormal findings on neurological examination. Granted that the wounding in this series is much more serious than in the ordinary civilian series of head injuries, it is still remarkable that 81 percent of patients 7 to 8 years after injury were suffering headaches. This is not, however, a higher frequency than that reported by Rawling (100), who found 88 percent of his English patients suffering headaches 2 years after injury, by Alajouanine et al. (4), who reported 87 percent of the French head injuries in 1914–18 having cephalalgia, nor by Gliddon (54), who stated that 87 percent of his Canadian veterans of World War I had headaches as long as 19 years after wounding. Similarly, German military casualties suffering head injuries had a high incidence of headache, Brun (24) reporting 77 percent and Usbeck (128) 85 percent.

The more favorable outcome of civilian injuries, probably less severe and to a younger population, is apparent from Pennington and Mearin's (97) review of 4,822 male naval inductees. Thirteen percent of these men had suffered craniocerebral injuries, 473 being so mild that consciousness was impaired less than 10 minutes, 69 having a period of unconsciousness between 10 and 60 minutes, and 110, classified as severe, being out a longer period. Some years after the injury only 4 percent of the mildly injured, 19

Table 40.—*General Summary of Neurological Status at Time of Follow-up*

Symptomatology	
Normal	63
Posttraumatic syndrome	419
Headache	183
Other and unknown	74
Total	739
Objective finding	
No neurological deficit	346
Hemiplegia or hemiparesis	159
Hemianopsia	68
Cortical sensory impairment	148
Aphasia	59
Mental impairment	102
Cranial nerve palsy	175
Cerebellar syndrome	7
Other	13
Unknown	2
Total	739

percent of the moderately injured, and 29 percent of the severely injured group had neurological symptoms referable to the effects of the accident.

That 57 percent of the wounded in this series should have a full-blown posttraumatic syndrome is also quite remarkable.

In men with some neurological disability, hemiparesis, usually associated with hemihypesthesia, was common. As might be expected, a large proportion of these men (15 percent) had an aphasia, which may be a greater disability than paralysis of an extremity. That almost 15 percent of the patients had some mental impairment emphasizes a fact often overlooked, namely, that organic brain disease does give rise to mental aberration varying from simple memory loss and confusion to profound dementia. It is further enlightening to note that almost one-fourth of the wounded in this series had some evidence of cranial nerve impairment. This is probably an indication of the destructiveness of present-day high velocity missiles. On the other hand, the paucity of cerebellar syndromes is likely due to the same factor since violence in the posterior fossa is apt to be incompatible with life.

C. NEUROLOGICAL SYMPTOMATOLOGY

For a short time almost all persons sustaining a head injury sufficient to cause an impairment of consciousness experience such symptoms as headache, dizziness, amnesia, mental confusion, nervousness, etc. These have been recognized as temporary sequelae, but their persistence is considered, in general, as abnormal.

The duration of these immediate posttraumatic symptoms varies in different individuals and according to severity of the wounding. Cedrmark (27) stated that 25 percent of patients suffering various civilian head wounds considered themselves well in 10 days, and 50 percent were free of symptoms in 3.5 weeks and 75 percent in 10 weeks. Those patients having no skull fractures had a median at 3 weeks and those having fractures had

a median at 9 weeks, but, strangely, almost 50 percent of the latter had some complaints as long as 3 to 10 years. Russell (107), in a series of 200 cases, noted that symptoms lasted less than 2 months in 80 cases (40 percent) and more than 18 months in 79 cases (40 percent), but 61 percent of working men and women returned to their usual tasks within 2 months after injury. Denny-Brown (36) states that 55 percent of a series of 200 cases of head injury had symptoms in convalescence, of which only 8 percent were due to demonstrable organic disease. The remainder seemed to be related to psychologic features, especially anxiety concerning occupation, compensation, family, or, more generally, the future. Akerlund (3), in a series of civilian head injuries, found that 37 percent were back at work in 2 weeks, 69 percent in 4 weeks, and 89 percent in 8 weeks.

Certain sequelae of a head injury have been recognized to fall into a fairly consistent clinical picture, which has been termed the posttraumatic syndrome or concussion state. Typically this includes headaches, dizziness, tinnitus, lack of ability to concentrate, nervousness, anxiety, and, less commonly, other mental symptoms. For the purpose of this discussion, any patient complaining of any three symptoms (usually including headache and dizziness) has been considered as having the posttraumatic syndrome; a person with less than three such complaints has had his symptoms coded individually. Accordingly, patients considered to have the posttraumatic syndrome often have many more than three of the above complaints. This arbitrary definition of the syndrome vitiates analysis of the individual complaints.[2]

Examination of the present series of head-injured patients for these general symptoms at the times of discharge and of follow-up (table 41) reveals an interesting contrast.

At the time of discharge, averaging approximately 9 months after injury, 42 percent of the patients did not complain of any neurological symptoms. But at the time of examination 6 to 7 years after injury, only 9 percent were free of such complaints. Did 33 percent of the men conceal such symptoms or did they develop the symptoms later? Since the major-

Table 41.—*Comparison of General Symptomatology at Discharge From Service and at Follow-up*

Complaint	At discharge from service	At follow-up	Complaint	At discharge from service	At follow-up
None	309	63	Impaired mentation	18	22
Posttraumatic syndrome	127	419	Impaired memory	26	20
Headache	241	183	Insomnia	4	11
Irritability or nervousness	6	70	Other	48	5
Dizziness	94	49	Unknown	5	.....
Easy fatiguability	4	28	Total	739	739

[2] Drs. Friedman and Mikropolous have made a separate study of posttraumatic headache at the New York center, and the data are presented in appendix 1.

Table 42.—*Correlation of Neurological Symptomatology at Follow-up and Severity of Wounding*

Neurological symptomatology at follow-up	Total		R–I group							
			1		2		3		4	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Total	739	100.0	99	100.0	182	99.9	362	100.0	96	100.0
None	63	8.5	20	20.2	7	3.8	27	7.5	9	9.4
Posttraumatic syndrome	419	56.7	37	37.4	119	65 4	205	56.6	58	60.4
Headache	183	24.8	31	31.3	47	25.8	95	26.2	10	10.4
Other symptoms	74	10.0	11	11.1	9	4.9	35	9.7	19	19.8

ity were released by CDD (Certificate of Discharge for Disability) and expected disability compensation, it would seem improbable that such symptoms, which might increase their awards, would not be mentioned. The hospital clinical records were scrutinized with care, so that it can be assumed that few men with symptoms were missed. In other series of head-injured in World War II (82, 136), the percentage of such patients having headache or the posttraumatic syndrome at or near the time of discharge is 22 to 54 percent, which would suggest that the figure given here is correct. If this is accepted, then, the stresses of civilian life would seem to have been sufficient to produce further symptoms. This delay in the onset of posttraumatic symptoms has been noted by many writers. Roussy and Lhermitte (104) called this interval the phase of incubation or contemplation. Adler (1) believes this interval varies in length according to the severity of the physical injury, being delayed until the patients recover physically and are faced again with their obligations. This type of reaction, originating in a conflict between a sense of duty and self-respect and the patient's idea of self-preservation, is designated a conflict neurosis by Symonds (123). Denny-Brown (35) believes that this accounts for the surgical opinion that severely injured patients often fail to develop this syndrome since the surgeon discharges the patient before the symptoms arise. Ruesch and Bowman (106) believe that patients with brain damage tend to improve, whereas those with an unstable pretraumatic personality, even if not so injured, worsen. Bay (12) also emphasized the necessity of assessing the premorbid mental structure in patients with posttraumatic complaints.

On analysis of the 309 men who were asymptomatic at discharge, it is found that only 40 are so at follow-up, 136 have the posttraumatic syndrome, 80 suffer headaches, and lesser numbers suffer other posttraumatic symptoms. On the other hand, only 24 (6 percent) of those men having any symptoms at discharge were without complaints 7 to 8 years later. In the group of 127 men having posttraumatic syndrome at discharge, only 9 were entirely free of symptoms at follow-up, and in the group of 241 men suffering headache at discharge, only 6 were free of neurological complaints at the time of follow-up examination. This, then, suggests that only about 6 percent of men having neurological complaints 9 months after a head injury will become completely free of them in time. On the contrary, there is an excellent chance that posttraumatic symptoms may be reprecipitated after a free interval of some months. It is interesting to speculate as to the cause of this recrudescence of symptoms. Were these more neurotic individuals who could not stand the stresses or were they more severely wounded persons?

If the table showing symptomatology at time of follow-up is analyzed in terms of the factors at the time of wounding, it becomes evident that a higher percentage of patients with mild head injuries than with severe brain damage are without symptoms (table 42). There is little difference in the frequency of posttraumatic syndrone as between men in R-I groups 3 and 4, but the mildly wounded patients in group 1 have less posttraumatic syndrome than the more severely wounded men in groups 3 and 4. The men in R-I group 2 were, it will be remembered, selected from a roster of

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Table 43.—*Correlation of Neurological Symptomatology at Follow-up With Various Characteristics of the Wound and Early Complications*

Characteristics of wound and early complications	Neurological symptomatology at follow-up					
	Total		None	Posttraumatic syndrome	Headache	Other symptoms
	Number of men	Percent	Percentage distribution			
All men	739	100.0	8.5	56.7	24.8	10.0
Brain laceration:						
None	238	100.0	10.9	55.5	25.6	8.0
Present	497	100.0	7.4	57.0	24.5	11.1
Unknown	4	100.0	.....	100.0	.....	.....
Location of wound:						
Frontal	251	100.1	7.6	57.4	24.7	10.4
Parietal	372	100.0	9.4	56.5	22.0	12.1
Temporal	187	100.0	9.6	53.5	26.7	10.2
Occipital	176	100.0	4.0	61.4	24.4	10.2
Immediate neurological deficit:						
None	417	100.0	8.6	54.7	29.7	7.0
Hemiplegia, hemianopsia, aphasia	233	100.0	6.4	60.5	17.6	15.5
Other and unknown	89	100.0	13.5	56.2	20.2	10.1
Complications:						
None	552	100.0	9.1	55.4	27.2	8.3
Infection or fungus cerebri	76	100.0	7.9	61.8	14.5	15.8
Hematoma	65	99.9	1.5	60.0	24.6	13.8
Other	46	99.9	13.0	58.7	13.0	15.2
Foreign bodies:						
None	297	100.0	9.1	56.9	26.3	7.7
Bone only	195	100.0	9.2	51.8	26.7	12.3
Metal, with or without bone	210	100.0	7.1	59.5	22.4	11.0
Unknown	37	100.0	18.1	64.9	16.2	10.8
Healing:						
Primary	485	99.9	8.0	54.2	27.4	10.3
Secondary	103	100.0	9.7	56.3	19.4	14.6
Unknown	151	100.0	9.2	64.9	19.9	6.0
Period of unconsciousness:						
None	146	100.0	11.0	41.1	41.1	6.8
0–2 hours	165	100.1	8.5	57.6	26.1	7.9
2–24 hours	52	100.0	11.5	75.0	7.7	5.8
1–7 days	74	100.0	5.4	68.9	17.6	8.1
Other and unknown	302	100.0	7.6	57.6	20.9	13.9

men receiving compensation from the Veterans Administration for residuals of a head wound, so it is not surprising that few of them are without symptoms. The apparent decrease in proportions of headaches in category 4 is due to the increase in the frequency of concomitant symptoms. Few, however, of the specific factors of wounding—the type, location, depths, or

size of the wound—seem to influence significantly the frequency of the posttraumatic syndrome, headache, etc., seen at follow-up (table 43). Even the immediate neurological deficit does not seem to be related to symptomatology or the posttraumatic syndrome at follow-up. One factor with some relation to symptomatic status at follow-up is the presence of initial complications; only 1 of 65 men with an initial hematoma was asymptomatic at follow-up. Finally, the initial period of unconsciousness appears to be associated with symptomatic status at follow-up; in general, longer periods of unconsciousness seem prognostic of multiple complaints at follow-up. However, men not rendered unconscious by the head wound were no more frequently asymptomatic than men having a period of traumatic unconsciousness.

On the other hand, posttraumatic symptoms correlate well with personality and intelligence. In table 44, it is apparent that the patients with the posttraumatic syndrome have high hysteria, depression and hypochondriasis scores, and relatively high psychasthenia and schizophrenia scores in the Minnesota Multiphasic Personality Test. Although the group with one or two posttraumatic symptoms have somewhat higher scores than the asymptomatic group, statistical significance is attained only in the hysteria score. This is of clinical importance as it suggests that the population having posttraumatic symptoms have, on the whole, a different personality type from the general population. That this is a psychoneurotic type cannot be said since the number of cases bearing that diagnosis, except in the Boston group, is very small. That the basic types are different is further borne out upon an analysis of the IQ's of the various groups (table 45). It is apparent that the group with posttraumatic symptoms has a high percentage of men with low intelligence and a low percentage of men with high intelligence. This is further demonstrated by the observation that the percentage of mentally deteriorated men is significantly higher in the group with posttraumatic syndrome than in the asymptomatic group (table 46).

An analysis of the work and social adjustment (tables 47 and 48, respectively) brings out the fact that patients with the posttraumatic syndrome are likely to have difficulties in both of these spheres. Which is cause and which is effect cannot be stated; even that there is any causal relationship is not certain since both phenomena may be under the influence of another factor. It is of interest that in the group having the posttraumatic syndrome only 8.8 percent attended school after discharge, whereas 16.5 percent of the mono- or disymptomatic group and 25.4 percent of the asymptomatic group did so. The home adjustment of the group with posttraumatic syndrome was much better than their social or work rehabilitation; in fact, in this regard there is little difference in the three groups, which may simply be a reflection of the fact that the patients were receiving disability compensation sufficient to enable them to live at a satisfactory level with their woes.

In this series the family adjustment seemed to be about the same in the men having a posttraumatic syndrome and those not so afflicted; the only possible exception might be the divorce or separation rate of 11.4 percent (8 of 70) in the "nervous" men, as opposed to a 5.3 percent incidence in men not so classified.

Table 44.—*Correlation of Minnesota Multiphasic Personality Factors With Neurological Symptomatology at Follow-up*

Symptom	Number tested	Percentage of cases with abnormally elevated score								
		Hs (percent)	D (percent)	Hy (percent)	Pd (percent)	Mf (percent)	Pa (percent)	Pt (percent)	Sc (percent)	Ma (percent)
No symptoms	48	6.2	8.3	10.4	4.2	6.2	0	2.1	4.2	2.1
One or 2 symptoms	193	[1] 26.0	15.1	[1] 21.9	5.7	2.6	1.0	8.9	10.9	2.1
Posttraumatic syndrome	244	[1] 51.6	[1] 43.4	[1] 43.0	7.8	1.6	2.9	[1] 23.4	[1] 23.4	7.8

[1] Statistically significant. The percentage for the subgroup with symptoms exceeds the percentage for the symptom-free group with $P<.05$.

Table 45.—*Correlation of Wechsler-Bellevue Intelligence Scale With Neurological Symptomatology at Follow-up*

Symptom	Number tested	Percentage of cases with IQ			
		≤89	90–119	≥120	Total
No symptoms	57	1.8	63.2	35.1	100.0
One or 2 symptoms	237	8.9	65.8	25.3	100.0
Posttraumatic syndrome	389	[1] 17.0	71.5	[1] 11.6	100.0
Total	683	12.9	68.8	18.3	100.0

[1] Statistically significant. The percentage for the subgroup with symptoms differs from that in the symptom-free group with $P < .05$.

Table 46.—*Correlation of Mental Deterioration, Determined by Wechsler-Bellevue Intelligence Scale, With Neurological Symptomatology at Follow-up*

Symptom	Number tested	Abnormally deteriorated for age	
		Number	Percent
No symptoms	57	4	7.0
One or 2 symptoms	237	40	16.9
Posttraumatic syndrome	389	102	[1] 26.2
Total	683	146	21.4

[1] Statistically significant. The percentage for the subgroup with symptoms differs from that in the symptom-free group with $P < .05$.

Table 47.—*Correlation of General Work Progress With Neurological Symptomatology at Follow-up*

Symptom	Number tested	Work progress				
		Regressed	Unchanged	Progressed	Unclassified	Total
		Percentage distribution				
No symptoms	63	7.9	22.2	68.3	1.6	100.0
One or 2 symptoms	257	15.2	20.2	63.0	1.6	100.0
Posttraumatic syndrome	419	25.3	29.4	44.1	1.2	100.0
Total	739	20.3	25.6	52.8	1.4	100.0

Table 48.—*Correlation of Social Adjustment With Neurological Symptomatology at Follow-up*

Symptom	Number	Social adjustment			
		Good	Poor	Other and unknown	Total
		Percentage distribution			
No symptoms	63	90.5	7.9	1.6	100.0
One or 2 symptoms	257	86.4	10.9	2.7	100.0
Posttraumatic syndrome	419	70.2	27.7	2.1	100.0
Total	739	77.5	20.2	2.3	100.0

The posttraumatic state was recognized as a frequent sequel to head injuries almost a century ago. Many years ago Friedmann (49) and others described as a syndrome a constellation of symptoms consisting of headache, dizziness, mental disturbances as noted above, and an intolerance to alcohol. This train of disorders was attributed to actual physical damage to the brain or disordered intracranial vasomotor control.

Since that time much has been written on the subject without solving the basic problem. Two schools of thought have developed—the one considering the syndrome essentially organic and the other believing it to be a neurosis. Arguing for the first view in the early part of this century was Aschaffenburg (7), more recently Penfield (94), who emphasized the role of dural adhesions, McConnell (84, 85, 86), who found excess subdural fluid in the intracranial cavity, and Watts et al. (137), who considered the contusion of the scalp to be related to the syndrome. On the other side, Charcot (28) assumed the symptoms to be purely functional. This viewpoint is still prevalent but placed in a somewhat different setting. It is agreed generally that a patient who develops a neurosis after a head injury (and it may be of any type—hysterical, anxiety, terror, neurasthenia, or hypochondriacal) had the basic neurotic personality before the head injury. As Minkowski (90) stated, the "pretraumatic factors" are tripped by the head injury into the neurotic state. Courville (30) maintains that this is typically quite distinct from the posttraumatic syndrome, being characterized by mental alertness, emotional depression, exaggeration of preexisting personality defects, elaboration of symptoms both in statement and behavior, the presence of hysterical components, and the multiplicity, changeability, and indefiniteness of the symptoms. The two conditions—posttraumatic syndrome and posttraumatic neurosis, Strauss and Savitsky (120) emphasize, may not be independent clinical entities. Schilder (114)

believes that the organic concussion syndrome may facilitate attitudes of a neurotic character and precipitate a type of neurosis different from the usual traumatic neurosis following injuries of other parts of the body. That this neurosis builds upon the warp and woof of the acute concussion syndrome is understandable.

The findings in this series of head-injured patients would seem to indicate that the two conditions are very closely associated and that stresses and strains which might produce a pure neurosis in a noninjured person induce a recrudescence of posttraumatic symptoms in the head-injured. Often the recurrent headache resembles the vascular type of cephalalgia which is associated with tension states. In fact, Friedman et al. (47) found that the responses to drugs in posttraumatic and psychogenic headaches were very similar and believed that the pathogenesis was closely related.

The mental symptoms—nervousness, anxiety, mental confusion, lack of concentration, impaired mentation, etc.—are almost as common and sometimes more disabling than the nervous symptoms of headache, dizziness, etc. Adler (1) emphasizes that the posttraumatic mental symptoms are present in approximately 31.5 percent of cases after civilian injuries. The most common type is the posttraumatic anxiety state which was present in her series in 48 of 200 patients and frequently accompanied headache and dizziness, nightmares, or other physical sequelae of the injury.

It has been suggested that cranioplasty might prevent the syndrome of the trephine, which is practically identical to the posttraumatic syndrome. Gardner (51), in particular, emphasized the movements of the brain with each arterial or venous pulsation. Grantham and Landis (58) noted that 54 of 100 patients with calvarial defects complained of headache, but that cranioplasty relieved only 16 of these and precipitated headaches in 4 other patients. Eleven of 24 men having vertigo were relieved after cranioplasty. Grant and Norcross (57) also suggest that cranioplasty relieves the headache and dizziness in the syndrome of the trephined. They report complete relief of headache in 5 of 20 cases. The incidence of the posttraumatic syndrome in this series was not influenced by the presence or absence of a skull plate.

Because most of these posttraumatic complaints are subjective, little can be noted on clinical examination. However, a few of the alterations were

Table 49.—*Examiner's Evaluation of Mental Status at Follow-up*

Mental status	Number of men	Mental status	Number of men
Normal	609	Impaired judgment or mentation	77
Uncooperative	3	Irritability	22
Confusion	10	Others	19
Depression	22		
Euphoria	12		
Wisecracking	5	Total	739

subject to verification. Irritability was noted by the examiner in some of the 70 patients who complained. Other aspects of the mental status are subject to clinical and psychological testing, and these results will be considered at this point.

D. MENTAL STATUS

A rather large number of mental aberrations were noted, although the majority of the patients had no complaints referable to their mental status. In table 49 the detailed breakdown of the findings referable to the mental state is given. Only the category of impaired judgment or mentation is sufficiently large to warrant correlations; this abnormality seems to be related to the severity of injury (table 50) and the various factors influencing it, but not to the location of the wound (table 51).

That deficiency in mentation is related both to a low intelligence (table 52) and mental deterioration (table 53) is understandable. However, alterations in personality fail to correlate with impaired mentation, as indicated in table 54. Moreover, patients in whom examiners noted other alterations, particularly depression, euphoria, and irritability, had elevated scores for all elements of the personality scale, most of them, indeed, significantly elevated.

Mentation was examined by two series of tests: One, the ability to repeat forward and backward certain numbers and to give the names of four presidents of the United States of America, and the other, the ability to carry out the serial subtraction test. The first test, mainly dependent upon memory, is roughly correlated with the severity of injury (table 55) in that patients with very minor injuries had few errors, whereas more than 25 percent of the more severely injured men made three or more errors. Whether or not brain laceration had occurred does not seem to be a prominent factor in this test (table 56), nor is the location of the lesion significant (table 57). Similarly, the various individual factors influencing the severity of wounding, namely, the diameter, depth of debridement, and complications, do not correlate with the ability to carry out these memory tests. Patients without a neurological deficit made fewer errors than individuals having a gross disability (table 58).

Most patients were able to carry out the serial 7 test with no more than two errors (table 59). The severity of wounding does not seem to be a strong determinant of impairment in this test, but the proportion of men in group 4 who made three or more errors was significantly higher at the 5-percent level than the corresponding proportion in group 3. It would seem that the memory and serial 7 tests are dependent upon more general factors than localized damage of the brain, and hence reflect only weakly the varying grades of brain wounding. The findings in these tests do correlate strongly with the intelligence and deterioration scores (table 60). In fact, the serial 7 and memory tests might be considered the clinician's intelligence test.

Table 50.—*Relationship of Mental Status to Severity of Wounding*

Mental status	Total		R–I group							
			1		2		3		4	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Total	739	100. 0	99	99. 9	182	100. 0	362	100. 0	96	100. 0
Normal	609	82. 4	92	92. 9	158	86. 8	306	84. 5	53	55. 2
Impaired judgment or mentation	77	10. 4	4	4. 0	8	4. 4	34	9. 4	31	32. 3
Other abnormalities in mental status	53	7. 2	3	3. 0	16	8. 8	22	6. 1	12	12. 5

Table 51.—*Relationship of Mental Status to Location of Wounding*

Mental status	Location of wounding							
	Frontal		Parietal		Temporal		Occipital	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Total	251	100.1	372	100.0	187	100.1	176	100.0
Normal	204	81.3	296	79.6	157	84.0	146	83.0
Impaired judgment or mentation	27	10.8	48	12.9	17	9.1	22	12.5
Other abnormalities of mental status	20	8.0	28	7.5	13	7.0	8	4.5

Table 52.—*Relationship of Mental Status to Wechsler-Bellevue Intelligence Scale*

Mental status	Number tested	Total	Percentage with IQ		
			≤89	90–119	≥120
Total	683	100.0	12.9	68.8	18.3
Normal	562	100.0	10.1	68.7	21.2
Impaired judgment or mentation	71	100.0	25.4	73.2	1.4
Other abnormalities in mental status	50	100.0	26.0	64.0	10.0

Table 53.—*Relationship of Mental Status to Mental Deterioration as Determined by Wechsler-Bellevue*

Mental status	Number tested	Abnormally deteriorated for age	
		Number	Percent
Total	683	145	21.2
Normal	562	90	16.0
Impaired judgment or mentation	71	38	[1] 53.5
Other and unknown	50	17	[1] 34.0

[1] Statistically significant. Percentage differs from that for men of normal mental status, $P < .01$.

Table 54.—*Relation Between Mental Status and Minnesota Multiphasic Personality Factors*

Mental status	Number tested	Scale								
		Hs	D	Hy	Pd	Mf	Pa	Pt	Sc	Ma
		Percent with abnormally elevated score								
Total	485	36.9	28.7	31.3	6.6	2.5	1.9	15.5	16.5	4.9
Normal	422	34.8	26.5	29.1	5.2	2.4	1.4	14.0	14.2	4.5
Impaired judgment or mentation	33	48.5	36.4	36.4	12.1	3.0	.....	18.2	24.2	6.1
Other and unknown	30	53.3	[1]50.0	[2]56.7	[2]20.0	6.7	[1]10.0	[2]33.3	[2]40.0	10.0

[1] Statistically significant. Percentage differs from that for men of normal status, $P < .05$.
[2] Statistically significant. Percentage differs from that for men of normal status, $P < .01$.

Table 55.—*Correlation of Memory With Severity of Wounding*

Memory	Total		R–I group							
			1		2		3		4	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Total	739	100. 0	99	100. 0	182	99. 9	362	100. 0	96	100. 0
0–2 errors	575	77. 8	94	94. 9	132	72. 5	286	79. 0	63	65. 6
≥3 errors	150	20. 3	5	5. 1	49	26. 9	70	19. 3	26	27. 1
Test not done or unsatisfactory	14	1. 9	.....	.....	1	0. 5	6	1. 7	7	7. 3

Table 56.—*Correlation of Memory With Cerebral Wounding*

Memory	Total		Brain laceration					
			None		Present		Other and unknown	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Total	739	100.0	238	100.0	497	100.0	4	100.0
0–2 errors	575	77.8	187	78.6	385	77.5	3	75.0
≥3 errors	150	20.3	50	21.0	99	19.9	1	25.0
Test not done or unsatisfactory	14	1.9	1	.4	13	2.6	.....	.....

Table 57.—*Correlation of Memory With Location of Wounding*

Memory	Location of wounding							
	Frontal		Parietal		Temporal		Occipital	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Total	251	100.0	372	100.1	187	100.0	176	100.0
0–2 errors	192	76.5	287	77.2	148	79.1	133	75.6
≥3 errors	51	20.3	75	20.2	31	16.6	38	21.6
Test not done or unsatisfactory	8	3.2	10	2.7	8	4.3	5	2.8

Table 58.—*Correlation of Memory With Neurological Deficit*

Memory	Immediate neurological deficit							
	Total		None		Hemiparesis		Other and unknown	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Total	739	100.0	417	100.0	148	100.0	174	99.9
0–2 errors	575	77.8	344	82.5	102	68.9	129	74.1
≥3 errors	150	20.3	72	17.3	39	26.4	39	22.4
Test not done or unsatisfactory	14	1.9	1	.2	7	4.7	6	3.4

Table 59.—*Relationship of Inability to Carry Out Serial 7 Test to Severity of Wounding*

Serial 7 test	Total		R–I group							
			1		2		3		4	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Total	739	100.0	99	100.0	182	100.0	362	100.0	96	100.0
0–2 errors	537	72.7	78	78.8	135	74.2	270	74.6	54	56.2
≥3 errors	163	22.1	20	20.2	44	24.2	71	19.6	28	29.2
Test not done or unsatisfactory	39	5.3	1	1.0	3	1.6	21	5.8	14	14.6

Table 60.—*Correlations—Memory and Serial 7 Test With Wechsler-Bellevue*

Errors	Total number	Percent	Wechsler-Bellevue Intelligence Scale							
			<100		100–119		≥120		Abnormally deteriorated	
			Number	Percent	Number	Percent	Number	Percent	Number	Percent
Memory:										
0	189	100.0	5	2.6	115	60.8	69	36.5	15	7.9
1	232	100.0	15	6.5	179	77.2	38	16.4	32	13.8
2	115	100.0	17	14.8	86	74.8	12	10.4	32	27.6
3	62	100.0	19	30.6	39	62.9	4	6.5	23	37.1
≥4	78	100.0	29	37.2	47	60.3	2	2.6	39	50.0
Serial 7:										
0	314	100.0	13	4.1	212	67.5	89	28.3	30	9.6
1	120	100.0	8	6.7	91	75.8	21	17.5	22	18.3
2	69	100.0	5	7.2	54	78.3	10	14.5	13	18.8
3	50	100.0	11	22.0	37	74.0	2	4.0	15	30.0
≥4	98	100.0	38	38.8	58	59.2	2	2.0	43	43.4

E. APHASIA

That disturbances of speech result from cerebral injuries has long been known and is usually attributed to damage to the dominant hemisphere. Yet in young individuals the ability of the cerebrum to compensate for impairment in speech functions seems remarkable, for a relatively small number of patients sustaining head injuries, even of the dominant hemispheres, complained of aphasic troubles, and even fewer had overt manifestations. Of 636 patients who did not complain of aphasic disturbances, only 6 were found to have clinical evidence of speech impairment, and of the 103 who complained of trouble, approximately one-third had no clinical evidence of aphasia. Thirty-seven patients complained of difficulties in all aspects of speech (global aphasia) and 26 of a specific anomia.

The changes in aphasia with the passage of time are also of interest. There were 99 patients noted to be aphasic at the time of debridement; of these, 57 had apparent speech disturbances at the time of discharge approximately 9 months after injury. Seven to 8 years later only 34 of them were noted to have clinical manifestations of aphasia. On the other hand, of the 42 men with initial aphasia, which was not observed at the time of discharge, 11 were said by the examiner to have aphasia at follow-up. It seems reasonable to believe that these men who were aphasic at the time of injury and also at follow-up were probably so affected at the time of discharge, even though the records do not note it. On this assumption, the following is found:

Aphasic at injury, 99 (100 percent)
Still aphasic at 9 months, 68 (68.7 percent)
Still aphasic at follow-up, 45 (45.5 percent)

On the other hand, there are some patients whose aphasic symptoms develop or become aggravated after debridement, perhaps as the result of the operative procedures, or vascular or infective complications. This apparently happened to 25 patients in this series; that is, there were 25 who were not noted to be aphasic at the time of injury, but who were found to be so at follow-up. In 11 of the 25, speech difficulties were already noted at discharge, while for 14 men, evidence of speech difficulty was at no time noted in the Army clinical records.

The evidence, then, is of marked improvement and even recovery in more than half of patients who were aphasic immediately after a head wound. Nearly a third recovered during the first 6 to 8 months, and nearly a quarter more improved during the subsequent 7 to 8 years.

The side of the lesion was predominantly left in aphasic right-handed patients, although some of these men had their major wound on the right side (table 61). However, the 7 left-handed persons suffering head wounds who had aphasia also had left-sided lesions. This would seem to emphasize the fact that left-handedness is not necessarily associated with dominance of the right hemisphere, a fact which has been noted previously (43). It further lends support to the concept that unilateral (usually left) representa-

tion of speech is the most prevalent form of cerebral organization in sinistrals. In general, wounds of the left hemisphere were more disabling than comparable wounds of the right side of the brain; of 286 patients with right hemispheral wounds, 61 percent had no immediate neurological deficit, whereas of 312 patients with left hemispheral wounds, only 48 percent were without deficit.

Table 61.—*Correlation of Handedness, Side of Wounding, and Aphasia*

Handedness	Side of injury	Number	Percent aphasic
Right	Right	286	5.9
Right	Left	312	21.2
Left	Right	20	0
Left	Left	27	25.9
Ambidextrous and unknown	Right	11	27.3
Ambidextrous and unknown	Left	7	14.3
	Bilateral	76	5.3
Total		739	13.3

It is obvious that the severity of injury (and all factors relating to it) was strongly correlated with the presence or absence of an aphasia (table 62). However, the location of the wound did not seem to be a very important determinant of aphasic difficulties (table 63). Global aphasia was much more common in patients with foreign bodies within the brain than was a nominal type of aphasia (table 64).

That the speech mechanisms are of great importance in mental functions, even in formal intelligence testing, is apparent from the scores on the Wechsler-Bellevue tests. Those patients having aphasia tended to be in the lower range of intelligence, and fewer of them had superior IQ's as compared to nonaphasic patients. Moreover, almost half (46 percent) of the 87 aphasic patients had abnormal deterioration as compared to 17.6 percent of the 592 patients with normal speech. As a further corollary of the above generalization is the fact that speech is essential to most occupations, and its impairment is a serious handicap to business success. In fact, as mentioned before, a speech defect may be a greater handicap than a hemiplegia or monoplegia. This hindrance became apparent in the analysis of the occupational status. The aphasic men had difficulty in their work so that almost half (42 percent) regressed and only 29 percent advanced, while the corresponding percentages for the nonaphasic were 17 percent and 57 percent. Similarly, aphasia seemed to have considerable influence on the social adjustment, some 46 percent of aphasics not having a satisfactory adjustment compared to 19 percent of the normally speaking men. However, economic and home adjustments were not significantly influenced by speech impairment.

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Table 62.—*Correlation of Subjective Speech Disturbances With Severity of Injury*

Speech	Total		R–I group							
			1		2		3		4	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Total	739	99. 9	99	100. 0	182	99. 9	362	99. 9	96	100. 0
Normal	641	86. 7	99	100. 0	172	94. 5	312	86. 2	58	60. 4
Anomia	26	3. 5	0	. 0	4	2. 2	15	4. 1	7	7. 3
Global	37	5. 0	0	. 0	1	. 5	15	4. 1	21	21. 9
Other and unknown	35	4. 7	0	. 0	5	2. 7	20	5. 5	10	10. 4

Table 63.—*Correlation of Subjective Speech Disturbances With Location of Wounding*

Speech	Location of wounding							
	Frontal		Parietal		Temporal		Occipital	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Total	251	100.1	372	100.0	187	100.0	176	99.9
Normal	214	85.3	297	79.8	152	81.3	153	86.9
Anomia	8	3.2	20	5.4	10	5.3	5	2.8
Global	18	7.2	29	7.8	14	7.5	9	5.1
Other and unknown	11	4.4	26	7.0	11	5.9	9	5.1

Table 64.—*Correlation of Subjective Speech Disturbances With Presence of Intracranial Foreign Bodies*

Speech	Total		Foreign bodies							
			None		All removed		Bone or metal remained		Other and unknown	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Total	739	99.9	297	100.0	195	100.1	210	100.1	37	100.0
Normal	641	86.7	281	94.6	162	83.1	165	78.6	33	89.2
Anomia	26	3.5	5	1.7	9	4.6	8	3.8	4	10.8
Global	37	5.0	2	.7	12	6.2	23	11.0	0	.0
Other and unknown	35	4.7	9	3.0	12	6.2	14	6.7	0	.0

F. APRAXIA

There were 27 patients with apraxic difficulties, whereas 708 patients seemed to have no such disturbances. Those patients having apraxic difficulties were all in the more severe group of cerebrally wounded, that is, in groups 3 and 4 (table 65), and the majority had a marked hemiparesis. The disturbances occurred in 24 right-handed men, with involvement in 10 instances of the right and 12 instances of the left hemisphere.

G. OLFACTORY DISTURBANCES

Olfactory disturbances were uncommon in this series, less than 6 percent of patients having any complaints referable to smell (table 66). Although wounds of the frontal region were more frequently associated with olfactory disturbances than injuries of other regions, the later also were accompanied by impairment of smell (table 67), perhaps as the result of contrecoup laceration of the orbitofrontal region. As might be expected, other factors of wounding did not seem to be related to olfactory alterations except that unilateral or bilateral anosmia was associated with severe injuries. These findings are similar to Leigh's (77) observations in a series of 1,000 head-injured patients. He found impairment of smell in 72 cases and stated that recovery occurred in only 6 cases. No observations were available on recovery in this series. Parosmia was noted in 12 of Leigh's cases and in 11 percent of patients in this series. This symptom may develop some time after injury.

H. VISUAL DISTURBANCES

Visual disturbances are rather common in head-injured patients. As seen in table 68, a field defect occurred in almost 10 percent of cases. The presence or absence of a visual disturbance correlated well with the severity of the injury, the mildly injured patients being unlikely to have symptoms referable to the visual system (table 69). Although diplopia and blurred vision did not seem to be related to wounding of any particular part of the brain, blindness, as would be expected, was much more commonly found with wounds of the frontal region since these implicated the optic nerve and tract; hemianoptic defects were found in patients having injuries of the posterior parietal, temporal, and particularly the occipital region (table 70). Nearly 24 percent of the cases of occipital wounds had lasting field defects.

The course of patients having visual disturbances is of interest. Of the 55 patients observed to be hemianoptic at the time of debridement, only 36 were noted in the records to have visual field disturbances at discharge, while at the time of examination 38 had contralateral, 1 an ipsilateral, and 1 a bilateral hemianopsia. It would, therefore, seem that regression of a hemianoptic defect may occur in approximately 30 percent of cases. Eighteen men were recorded at discharge as having field disturbances which were not noted at debridement, so that a total of 54 men had field defects when separated from service, and 5, or 9.3 percent, of these had no visual signs at follow-up, evidnce that late recovery may sometimes occur.

Table 65.—*Relationship of Apraxia to Severity of Injury*

Apraxia	Total		R–I group							
			1		2		3		4	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Total	739	100.0	99	100.0	182	100.0	362	100.0	96	100.0
None	708	95.8	99	100.0	182	100.0	345	95.3	82	85.4
Present	27	3.7	0	.0	0	.0	15	4.1	12	12.5
Unknown	4	.5	0	.0	0	.0	2	.6	2	2.1

Table 66.—*Olfactory Symptoms at Follow-up*

Symptom	Number of men	Symptom	Number of men
None	684	Unknown	6
Parosmia	11		
Anosmia	25	Total	739
Other	13		

Table 67.—*Correlation of Olfactory Disturbances With Location of Wounding*

Symptom	Location of wounding							
	Frontal		Parietal		Temporal		Occipital	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Total	251	100.0	372	100.0	187	100.0	176	100.0
None	223	88.8	344	92.5	176	94.1	164	93.2
Parosmia	6	2.4	7	1.9	1	0.5	2	1.1
Anosmia	17	6.8	7	1.9	5	2.7	3	1.7
Other and unknown	5	2.0	14	3.8	5	2.7	7	4.0

Table 68.—*Visual Disturbances*

Subjective complaint	Number of men	Objective findings	Number of men
None	578	Normal	645
Diplopia	18	Blind [2]	7
Blurred vision—bilateral	38	Vision impaired	23
Blindness [1]	27	Homonymous hemianopsia, contralateral	63
Scotomata	6	Homonymous hemianopsia, ipsilateral	3
Field defect	55	Other	5
Other	17	Unknown	3
Unknown	5		
Total number of cases	739	Total number of cases	739

[1] Includes patients who had one eye enucleated.
[2] Excludes patients who had an eye enucleated.

Table 69.—*Correlation of Visual Disturbances With Severity of Injury*

Disturbance	Total		R–I group							
			1		2		3		4	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Total	739	99.9	99	99.9	182	99.9	362	100.0	96	100.0
No symptoms	578	78.2	92	92.9	158	86.8	270	74.6	58	60.4
Diplopia	18	2.4	0	0.0	3	1.6	11	3.0	4	4.2
Blurred vision	38	5.1	3	3.0	13	7.1	17	4.7	5	5.2
Blindness	27	3.7	1	1.0	2	1.1	18	5.0	6	6.2
Field defect	55	7.4	0	.0	2	1.1	30	8.3	23	24.0
Others and unknown	23	3.1	3	3.0	4	2.2	16	4.4	0	.0

Table 70.—*Correlation of Visual Disturbances With Location of Wounding*

Disturbance	Location of wounding							
	Frontal		Parietal		Temporal		Occipital	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Total	251	100.0	372	100.0	187	100.0	176	100.0
No symptoms	197	78.5	289	77.7	146	78.1	116	65.9
Diplopia	6	2.4	12	3.2	5	2.7	2	1.1
Blurred vision	13	5.2	18	4.8	8	4.3	9	5.1
Blindness	22	8.8	9	2.4	10	5.3	2	1.1
Field defect	7	2.8	33	8.9	12	6.4	42	23.9
Other and unknown	6	2.4	11	3.0	6	3.2	5	2.8

Injury to the peripheral visual structures, the optic nerve or bulb, is not uncommon in head injuries. Turner (127) found 25 cases of optic nerve injury in 1,550 head injuries, and Russell (108) found 8 such cases in 600 patients. Brändle (21) gives an incidence of 0.7 percent in head injuries. In this series some 25 patients had an eye enucleated because of injury to the orbit, 7 patients were blind in 1 eye, and 23 patients had some visual impairment. On ophthalmoloscopic examination 19 of these patients with impaired eyesight had optic atrophy in one or both eyes. Over a period of time considerable improvement may occur in the vision of patients with traumatic damage to the optic nerve. Turner (127) states that if recovery is going to occur, light perception begins to return about the fourth day and the recoverable vision has returned by the end of a month. Bryson (25) noted that the prognosis is poor if light perception has not returned in 2 months, if the pupil is inactive, and if there is early disc pallor. Askenasy et al. (9) conclude that an immediate amaurosis will not be benefited by surgery. Hooper (68) points out that some cases of traumatic blindness are caused by a contusion with small petechial hemorrhages in the optic nerve and that recovery may occur, although, in general, the prognosis is poor. Landolt (75) found fractures in the sphenoidal ridge in 75 percent of his series of traumatic optic nerve lesions.

Blurred vision does not seem to correlate with specific factors of wounding nor with objective evidence of impaired vision since 29 of 39 patients so complaining had normal vision. However, it may be a manifestation of the posttraumatic syndrome, for every one of the 39 men complaining of blurred vision had posttraumatic symptoms, and 29 had the full-blown syndrome.

I. ACOUSTIC DISTURBANCES

Complaints referable to hearing were quite common (table 71), although objective auditory defects were less frequent. Tinnitus was the most common complaint, and impaired hearing was almost as frequent. These com-

plaints, however, were not correlated with the severity of injury or any of its component factors (table 72). The fact that wounds in the mastoid area were more frequently associated with auditory disturbances (table 73) than were wounds of other regions would fit in with the recognized occurrence of eighth nerve lesions as the result of basal temporal fractures.

The chances of recovery from tinnitus are not very good. Of the 24 men who were reported to have had noises in one or both ears at the time of separation from service, only 5 had no auditory symptoms at follow-up. Of the 715 men without tinnitus at discharge, 62.5 percent were without auditory symptoms at follow-up; 126 (19.0 percent) complained of tinnitus. If one accepts Mygind's (91) figures as applying generally, approximately 50 percent of head-injured men, if examined within the first week of injury, would have otoscopic changes such as tears in the drum, and hemorrhages behind the tympanic membrane. Hence, many of these changes must resolve, leaving the patient asymptomatic.

Table 71.—*Auditory Disturbances*

Disturbances of hearing	Subjective complaints	Objective findings
None	456	587
Tinnitus:		
Contralateral to side wounded	36	.....
Ipsilateral to side wounded	80	.....
Bilateral	81	.....
Impaired hearing:		
Contralateral to side wounded	32	27
Ipsilateral to side wounded	71	89
Bilateral	26	19
Deafness:		
Contralateral to side wounded	5	5
Ipsilateral to side wounded	20	9
Bilateral	1	1
Other and unknown	5	4
Total number of cases	739	739

Table 72.—*Correlation of Auditory Findings With Severity of Wounding*

Finding	R–I group							
	1		2		3		4	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Total	99	100.0	182	100.0	362	100.0	96	100.0
None	83	83.8	147	80.8	282	77.9	74	77.1
Impaired hearing	15	15.2	30	16.5	69	19.1	19	19.8
Deafness	1	1.0	5	2.7	7	1.9	2	2.1
Other and unknown	0	.0	0	.0	4	1.1	1	1.0

Table 73.—*Correlation of Auditory Findings With Location of Wounding*

Finding	Location							
	Frontal		Parietal		Temporal		Occipital	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Total	246	100.0	372	100.0	163	100.0	176	100.0
None	209	85.0	294	79.0	116	71.2	131	74.4
Impaired hearing	32	13.0	70	18.8	38	23.3	38	21.6
Deafness	2	.8	4	1.1	6	3.7	4	2.3
Other and unknown	3	1.2	4	1.1	3	1.8	3	1.7

The tinnitus may be associated with normal hearing. Only 41.7 percent of the patients with aural noises complained of some degree of impairment of hearing, as compared to 20.3 percent of the group without tinnitus, and 58.3 percent were found to have some auditory disability, as compared to 19.4 percent of the group with no aural symptoms.

Late symptoms referable to the vestibular portion of the acoustic nerve were rare, for, as Linthicum and Rand (80) state, they tend to disappear early. Although dizziness is a common complaint, rarely is it due to vestibular involvement. If true vertigo is present, as Baumoel and Marks (11) note, abnormalities in the caloric responses may persist for some weeks after the whirling sensation has disappeared. Grove (59) emphasizes asymmetrical vestibular sensitivity as a source of symptoms. Positional vertigo, Gordon (56) states, following a head injury usually clears in a few months. However, dizziness, as a more general complaint, is prone to persist indefinitely after a head injury. In this series approximately two-thirds of the cases had some such symptoms over 7 to 8 years. Rawling (100) reported 77 percent of his cases complained of this disturbance, Gliddon (54) that 58 percent of head-injured men had dizziness, some as long as 19 years after their injury, and, in Germany, Brun (24) noted that 47.9 percent of men with open head injuries suffered dizziness for 9 years or more.

J. OTHER CRANIAL NERVES

Nerves to the extraocular muscles were rarely involved; in 18 cases only was there such impairment—14 times in the ipsilateral and 4 times in the globe contralateral to the injury (table 74). There were eight examples of sixth cranial nerve weakness, six being on the side of wounding. Anisocoria was also relatively uncommon, being found in 39 cases, of which the contralateral pupil was the larger in 21 instances. In spite of an anisocoria, the light response was normal in both eyes in 12 of the cases. When the reflex was impaired it usually occurred in the ipsilateral eye.

Although facial sensation and movement were examined and coded with the cranial nerves, their impairment in this series was usually supranuclear and hence, strictly speaking, not the result of involvement of a cranial nerve.

Impairment of facial sensation to pinprick and touch was noted in 80 cases, usually associated with a hemihypesthesia; in 13 of these cases there was an impairment of sensation ipsilateral to the side of injury not associated with an apparent peripheral nerve injury. In some additional 27 cases ipsilateral sensory impairment seemed to be due to an injury of the peripheral branches of the trigeminal nerve.

Facial movement was impaired on the side opposite cerebral wounding in 75 cases and on the same side in 27 cases, but 16 of the latter seemed to be due to extracranial peripheral injuries of the facial nerve. In two cases there was bilateral facial paresis.

The 9th and 10th cranial nerves were involved in only two cases in this series, causing impairment of deglutition.

Table 74.—*Impairment of Other Cranial Nerves*

Nerve and type of impairment	Number of men	Nerve and type of impairment	Number of men
Ocular motor nerves:		Contralateral to wound....	7
Impairment extraocular movement:		Fifth cranial nerve:	
		Trigeminal hypesthesia (central):	
Ipsilateral to wound.....	14	Ipsilateral to wound.....	13
Contralateral to wound....	4	Contralateral to wound....	67
Anisocoria:		Seventh cranial nerve:	
Ipsilateral larger.....	18	Facial paresis (central):	
Contralateral larger.....	21	Ipsilateral to wound.....	11
Impairment light reflex:		Contralateral to wound....	75
Ipsilateral to wound.....	20		

K. DISTURBANCES OF THE MOTOR SYSTEM

It is of interest that most patients who complained of weakness had objective paresis upon examination. At follow-up, 513 patients had no symptoms of motor impairment. Of these, 485 had normal power and another 9 patients had weakness due to a peripheral wound so that only 19 (3.7 percent) had paresis attributable to the head wound. Complaints of monoparesis were less likely to be associated with a demonstrable weakness than symptoms of hemiparesis. Of the 76 patients complaining of isolated paresis of face, arm, or leg, 33 (43.4 percent) had no impairment of strength on examination; of the 70 who complained of unilateral arm and leg weakness, 13 (18.6 percent) had no paresis; and of the 32 complaining of hemiparesis, only 1 had no demonstrable impairment.

Motor disturbances usually were referred to as weakness of the extremities opposite the wound, as may be seen in table 75. However, some patients complained of weakness on the ipsilateral side and a few had weakness of both legs. The degree of weakness, both subjectively and objectively, was correlated with the severity of injury and with all factors which modify that aspect of wounding (table 76). Reflecting this is the fact that paresis occurred in approximately 25 percent of patients having primary healing and in over 50 percent of patients with secondary healing. Weakness was more common in parietal lobe wounds, although present in wounds of all parts of the brain (table 77). Perhaps as a corollary of this,

Table 75.—*Impairment of Motor Power*

Weakness	Subjective complaints	Objective findings
None	513	546
Arm:		
Contralateral to side wounded	37	34
Ipsilateral to side wounded	6	7
Leg:		
Contralateral to side wounded	18	22
Ipsilateral to side wounded	3	2
Arm and leg:		
Contralateral to side wounded	66	55
Ipsilater l to side wounded	4	9
Entire side:		
Contralateral to side wounded	32	20
Ipsilater l to side wounded	3	1
Weakness not due to head injury	34	32
Other and unknown	23	11
Total	739	739

Table 76.—*Correlation of Motor Disturbances With Severity of Wounding*

Motor disturbance	R–I group							
	1		2		3		4	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Total	99	100.0	182	100.0	362	100.0	96	100.0
Subjective:								
No symptoms	92	92.9	158	86.8	237	65.5	26	27.1
Contralateral weakness	3	3.0	10	5.5	89	24.6	65	67.7
Objective:								
Normal	92	92.9	163	89.6	258	71.3	33	34.4
Contralateral weakness	2	2.0	6	3.3	78	21.5	55	57.3

impairment of motor power was generally associated with some loss of superficial sensibility (table 78).

At debridement 148 patients were noted to have weakness; at the time of follow-up only 27 of these (18 percent) did not complain of weakness, and another 14 were found to have no paresis on examination. It may, therefore, be concluded that in penetrating wounds of the head, a hemiparesis noted just after wounding has about a 25-percent chance of clearing up almost completely. However, motor impairment at the time of wounding was either overlooked at that examination or developed later, for 160 patients were noted to have some paresis at the follow-up examination.

Table 77.—*Correlation of Motor Disturbances With Location of Wounding*

Motor disturbance	Location of wounding							
	Frontal		Parietal		Temporal		Occipital	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Total	246	100.0	372	100.0	163	100.0	176	100.0
Subjective:								
No symptoms	176	71.5	207	55.6	102	62.6	133	75.6
Contralateral weakness	51	20.7	135	36.3	40	24.5	29	16.5
Objective:								
Normal	185	75.2	226	60.8	120	73.6	134	76.1
Contralateral weakness	43	17.5	120	32.3	31	19.0	27	15.3

Table 78.—*Correlation of Motor Findings With Impairment of Superficial Sensation*

Finding	Total	Superficial sensation		
		Impaired	Other	Normal
Paralyzed	34	32	.....	2
Impaired	124	83	1	40
Other	2	2	.....	.....
Normal	578	38	2	538
Unknown	1	.....	1	.....
Total	739	155	4	580

Of the 148 men stated to have paresis at debridement, 96 were noted in the discharge records to have some weakness at that time. At follow-up 84 percent of these were found to have motor impairment. Of the 52 who were considered normal at discharge, 21 had paresis at the time of the follow-up examination. Fourteen men had weakness at discharge and at follow-up, but were not so noted at the time of debridement, while 47 other men had weakness at follow-up which was not noted in any of the Army records.

It was hoped that an analysis with reference to the status at follow-up, of the 148 patients noted to have a hemiparesis at the time of wounding, would give some indication of factors favorable and unfavorable in prognosis. For the purpose of this study, a residual weakness at follow-up of at least 20 units difference in dynamometer readings of the 2 grips, or a difference of 10 units in favor of the nondominant hand, was arbitrarily taken to be the

criterion for residual weakness. Patients were considered to have no residual weakness if the difference of dynamometer readings of the 2 grips was less than 10 units. This residual nonparalyzed group consisted of only 24 men, or 16 percent of the men with an initial hemiparesis. This figure is a little lower than might be expected from the 25-percent recovery rate estimated above. Russell (110), however, noted recovery of motor function in more than half of the initially paralyzed cases.

There is no apparent or statistically significant difference in the residual paralyzed and nonparalyzed groups relative to the factors of wounding—site of wounding, size of cranial defect, and depth of wound—or to the factors associated with the initial debridement—time of debridement and skill of surgeon. It is of interest, although not significant statistically, that the group with no residual weakness had a higher percentage (83.3 percent) of men without complications of debridement than the hemiparetic group (67.4 percent of 86 patients).

The factors associated with cranioplasty are the same in the two groups. There is no difference in the pneumoencephalographic abnormalities of the two series. In the early electroencephalographic studies there are both more focal (62.2 percent) and generalized (42.2 percent) abnormalities in the paralyzed group than in the nonparalyzed group (27.2 and 18.1 percent). However, in the follow-up electroencephalograms this difference is not so apparent, the proportions that were normal being 41.9 percent and 47.8 percent, respectively. The two groups seem to have the same type and frequency of posttraumatic symptoms. Aphasia is more common in the group with residual weakness (34 percent) than in the second group (17 percent), although the latter contains a higher percentage of left-sided wounds (83.3 percent vs. 52.6 percent). Epilepsy seems to be more frequent in the paralyzed than in the nonparalyzed group (53.7 percent vs. 33.3 percent). The two classes do not seem to be distinguishable on the basis of intelligence or personality.

This suggests that the factors favoring recovery were either not those examined or that the influence of several factors together was important. Certainly there is no evidence that factors in treatment within the range of the present study modify the course appreciably.

Disturbances of the motor system may be manifested by weakness, spasticity, atrophy, or changes in reflex activity. Because in wounds of the brain the involvement of the motor system is of the upper motor neuron type, spastic paresis with increased deep reflexes and pathological superficial reflex responses is usually found. However, the degree of involvement of each of these does not run absolutely parallel so that, although paresis may be used as a prototype for correlations with other phenomena, it is worthwhile considering some of the other characteristics of upper motor lesions in relationship to the degree of paresis.

The tone of the paretic muscles varied from hypotonic to hypertonic. About half of the patients with objective weakness had normal tone, but of the more severely paralyzed men, 28 of 34 had hypertonia (table 79). Augmentation of the appropriate tendon reflexes, usually considered a manifestation of increased tone, was present in 67 of the 70 hypertonic patients and in a number of patients in whom increased tone could not be

demonstrated clinically. It was, however, not present in all patients having paresis. In fact, decreased reflexes were noted in paretic extremities having normal (five cases) or decreased tone (three cases). Alterations in tone were frequently accompanied by abnormalities of deep sensation: 54 of 70 men with hypertonic paresis had hypesthesia, as did 8 of 13 men with hypotonic limbs.

Table 79.—*Correlation of Motor Power With Tone*

Finding	Tone, side contralateral to wound					
	Hypotonia	Hypertonia	Rigidity	Normal	Other and unknown	Total
Paralyzed	3	28	2	1	.....	34
Impaired	9	39	3	73	.....	124
Normal	1	1	.....	574	2	578
Other	.....	2	.....	.....	.....	2
Unknown	.....	.....	.....	.....	1	1
Total	13	70	5	648	3	739
	Tone, side ipsilateral to wound					
Paralyzed	1	.....	.....	33	.....	34
Impaired	7	5	.....	112	.....	124
Normal	.....	1	.....	575	2	578
Other	2	.....	.....	.....	.....	2
Unknown	.....	.....	.....	.....	1	1
Total	10	6	.....	720	3	739

Muscular atrophy was less commonly found than hypertonicity or hyperreflexia, being noted in only 62, or approximately one-fourth of the paretic patients (table 80). It was present, however, in 32 of the 34 patients with severe weakness. Usually there was an associated alteration in tone: Of some 70 patients with hypertonic and paretic limbs, 46 had atrophy, and of 13 patients with hypotonic weakness, 8 had atrophy. Although somewhat more commonly seen in parietal lobe lesions, it did occur with lesions of other regions (table 81). Atrophy was generally associated with superficial sensory impairment (56 of 63 men), although the reverse was not true. (Only 56 of 154 men with superficial sensory disturbances had atrophy.)

L. SOMATOSENSORY DISTURBANCES

Paresthesias, such as numbness or feeling of the member being dead, are the sensory symptoms mentioned by about 15 percent of the patients in this series. These disturbances, as may be seen in table 82, are usually on the side contralateral to the wound, but may involve one member or one side of the face. The distal type of sensory impairment (ulnar or radial dis-

Table 80.—*Correlation of Motor Power With Atrophy*

Finding	Atrophy				
	None	Present	Other	Unknown	Total
Paralyzed	2	32	.....	.....	34
Impaired	94	28	2	.....	124
Normal	576	.....	.....	2	578
Other	.....	2	.....	.....	2
Unknown	.....	.....	.....	1	1
Total	672	62	2	3	739

Table 81.—*Relationship of Atrophy to Site of Wounding*

Atrophy	Location of wounding							
	Frontal		Parietal		Temporal		Occipital	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Total	251	100.0	372	100.0	187	100.0	176	100.0
Contralateral extremities	21	8.4	54	14.5	14	7.5	8	4.5

Table 82.—*Somatosensory Disturbances*

Location	Paresthesias	Impaired superficial sensation	Impaired deep sensation	Impaired stereognosis
None	584	561	618	634
Face:				
Contralateral	8	.....	.....	.....
Ipsilateral	4	.....	.....	.....
Arm:				
Contralateral	34	28	23	56
Ipsilateral	4	4	1	3
Leg:				
Contralateral	18	13	18	5
Ipsilateral	2	3	2	0
Arm and leg:				
Contralateral	33	77	64	27
Ipsilateral	5	10	5	3
Entire side:				
Contralateral	27	.....	.....	.....
Ipsilateral	3	.....	.....	.....
Other	16	5	4	2
Unknown	4	4	5	7
Total number of cases	739	739	739	739

680802—62——6

tribution), although present in some patients shortly after wounding, was not noted in this series at the follow-up; stocking or glove paresthesias did occur. The presence of such paresthesias correlated positively with the severity of wounding, being present in practically 50 percent of the patients with a severe head wound (table 83). All factors relating to severity of wounding, such as brain laceration, diameter of wound, depth of wounding, complications, debridement, period of unconsciousness, neurological deficit, residual foreign body, and type of healing, influence the incidence of paresthesias. Wounds of the parietal area are more likely to give rise to sensory disturbances than those of other regions of the head (table 84).

Table 83.—*Correlation of Sensory Disturbances With Severity of Wounding*

Disturbance	R–I group							
	1		2		3		4	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Total	99	100.0	182	100.0	362	100.0	96	100.0
Contralateral:								
Paresthesia	1	1.0	13	7.1	62	17.1	44	45.8
Hypesthesia	3	3.0	10	5.5	54	14.9	51	53.1

Table 84.—*Correlation of Sensory Disturbances With Location of Wounding*

Disturbance	Location of wounding							
	Frontal		Parietal		Temporal		Occipital	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Total	251	100.0	372	100.0	187	100.0	176	100.0
Contralateral:								
Paresthesia	35	13.9	96	25.8	37	19.8	25	14.2
Hypesthesia	41	16.3	99	26.6	33	17.6	25	14.2

Paresthesias may or may not be associated with demonstrable sensory alterations. In none of the patients having facial paresthesias alone could sensory changes be demonstrated in the face. Paresthesias of the arm or leg alone were associated with sensory disturbances in approximately 50 percent of cases, but if both were involved the incidence rose to 75 percent. Of the

30 patients having paresthesias of the face, arm, and leg, 28 had impairment of sensation.

The association of paresthesias and motor disturbances was of the same order as for paresthesias and sensory alterations. About half of the patients with paresthesias did not complain of weakness. Dysesthesias of the face or of both arms or both legs were rarely associated with paresis (2 of 13 cases in each category). Paresthesias of the arm or leg were accompanied by weakness in 75 percent of cases. Of the 27 patients having paresthesias of the entire side, 26 had some weakness.

The presence of paresthesias was not significantly associated with abnormal findings in the personality inventory. It is apparent that disturbances of deep sensibility and astereognosis closely parallel those of the superficial sensibilities. Of 154 men with superficial sensory disturbances, 101 had impaired deep sensibility, and only 11 with normal superficial sensation had impairment of deep sensibility. Of the 154 men with superficial sensory disturbances, 84 had astereognosis. Of the 113 men with impaired deep sensibility, 82 had astereognosis as contrasted with only 12 of 621 men with normal deep sensation. Astereognosis was also closely associated with motor impairment. Of 123 patients with some degree of weakness, 52 had astereognosis, as did 28 of 32 paralyzed patients. It was also closely correlated with disturbances of tone, being present in 50 of 70 hypertonic patients and in 7 of 13 hypotonic patients.

M. URINARY DISTURBANCES

Urinary disturbances were encountered in only 21 cases. In the early stages of a cerebral injury, bladder disturbances are common, but they usually disappear in a few weeks. They may consist of an urgency or inability to urinate. Goldstein (55) and others have discussed the cortical centers mediating bladder function. The lateral cortical representation in the arm and leg area does not seem to be so important in producing urinary disturbances as the paracentral region which, if injured bilaterally, gives rise to a paralytic bladder with overflow incontinence.

N. SEXUAL DISTURBANCES

Relatively few patients complained of sexual disturbances. Impotence occurred in 60 cases and some increase in sexual desire was reported by 4 men. Various other complaints referable to sexual appetite were mentioned in 14 instances. The great majority of patients, some 641, maintained that there was no change in their sexual appetite. There seemed to be a definite relationship betwen the severity of the injury and the presence of decreased potency (table 85). However, although a slightly greater percentage of patients having frontal lobe involvement suffered from sexual complaints than those having injuries of other regions, the difference was not statistically significant.

It is noteworthy that the presence of sexual disturbances correlates well in the personality inventory only with psychasthenia. Sexual disturbances have been noted in previous reports of cerebral injuries, but few detailed reports are available. Usbeck (128) mentions such difficulties in his series. Meyer (87) discusses the problem, indicating that decreased erection, weak

Table 85.—*Correlation of Sexual Disturbances With Severity of Wounding*

Disturbance	Total		R–I group							
			1		2		3		4	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Total	739	99. 9	99	100. 0	182	99. 9	362	100. 0	96	100. 0
No symptoms	641	86. 7	96	97. 0	160	87. 9	314	86. 7	71	74. 0
Impotence and impaired libido	60	8. 1	2	2. 0	20	11. 0	24	6. 6	14	14. 6
Increased desire	4	. 5	0	. 0	1	. 5	2	. 6	1	1. 0
Other and unknown	34	4. 6	1	1. 0	1	. 5	22	6. 1	10	10. 4

orgasm, or premature ejaculation are the most common causes of poor sexual relationships. He reports that 81 of 100 head-injured patients experienced these difficulties. Perhaps the explanation of the relatively few cases from military wounds lies in the fact that these disturbances are much more frequent in older people. Meyer believes that they are more common following wounds of the orbitofrontal or hypothalmic regions than other parts of the brain.

O. DISCUSSION

Although wounds of the head and brain are generally considered as local or focal cerebral lesions, it is emphasized in this analysis that such violence has a much wider distribution than merely to the area below the site of cranial impact. The relative frequency of effects on a part of the brain remote from the violence, and the failure to demonstrate the correlation of site of wounding with cerebral functions which appear to be represented in small cortical foci, indicate a widespread effect of the head trauma.

This generalized effect of the craniocerebral injury is as important in the sequelae as the local damage. In fact, certain disabling symptoms seem to be more dependent upon the general than the local injuries. Thus the posttraumatic syndrome, the mental disturbances, certain cranial nerve impairments, and sexual alterations, correlate with general rather than local factors.

On the other hand, the disturbances of sensation and motor power are related more specifically to regional damage, and to a lesser extent with general factors of cerebral impairment.

The degree of recuperation of a patient from a head wound, while dependent upon the severity of wounding, the location of the injury, and factors in repair, is also bound up with the preinjury mental and personality status of the patient. It is of great interest that individuals of low intelligence and neurotic adjustment do not recover from a brain injury as rapidly or as completely as more intelligent and better adjusted individuals. Although intelligence and mature personality may not necessarily be related, in general they seem to be associated and are strong factors in combating the stresses involved in cerebral injuries. As a rule, men with higher intelligence and nonneurotic personalities were able to effect satisfactory occupational, economic, and social adjustments, even with a severe neurological deficit, while patients of subnormal intelligence and neurotic makeup were unable to adjust well in any sphere, even in the absence of an overt neurological deficit.

It is of interest that none of the head-injured men in this study have developed extrapyramidal diseases, brain tumors, multiple sclerosis, etc., which have occasionally been attributed to trauma. As Leonhardt (78) has emphasized, if rigid criteria must be met before suggesting a traumatic etiology for such conditions, the number of cases which might be so considered dwindles.

Chapter IV

ELECTROENCEPHALOGRAPHY, PNEUMOENCEPHALOGRAPHY, AND PSYCHOMETRY

A. INTRODUCTION

The functions of the brain are commonly described in terms of the results of observations of nervous activity, psychological reactions, or cerebral morphology. Although none of these special examinations may indicate the degree of specific functioning of the brain, each or all may be disturbed by injuries of the head. It is recognized that these diagnostic procedures do not analyze or measure the same or even similar properties of cerebral function. Those most commonly employed are the electroencephalogram, the pneumoencephalogram, and psychometric examinations. In this chapter it is planned to analyze the influence of the neurological deficit on these yardsticks and to determine, as much as possible, the sensitivity of these criteria in evaluating the neurological deficit. Having done that, it is proposed to examine the factors which may influence recovery and which may give certain prognostic indications as to the amount of recovery which might be expected from a neurological deficit.

B. ELECTROENCEPHALOGRAMS AND NEUROLOGICAL DEFICIT

1. Variability in Interpretation

The electroencephalograms have been read by independent examiners in the four centers. That the interpretation of an electroencephalogram is not entirely objective but is influenced to a variable extent by the reader's subjective impressions which are not always based upon tangible clues, is generally recognized.

When the study was organized, the desirability of having all tracings interpreted uniformly (possibly by several readers independently) was not fully appreciated. However, analysis of the results of examination revealed obvious gross differences between readers at the several centers. Thus, the proportion of tracings which were considered to manifest generalized abnormalities varied between 10.1 percent and 27.1 percent at the four centers. At one center, no less than 88.2 percent of the tracings which were called "generalized abnormality" were diagnosed paroxysmal slow, while the highest such percentage in any of the other three centers was 34.1 percent and the lowest 22.2 percent. Similar differences, fortunately of smaller magnitude, were seen in the diagnoses of types of focal abnormalities.

It was considered desirable, in the face of these findings, to have all tracings interpreted by a single reader, but this proved to be impossible since many of the records had been destroyed. However, in order to gain some idea of the amount and nature of the variability between different readers, 140 tracings obtained from other than the Baltimore center were independently reread by Dr. Curtis Marshall and the results were compared. These

findings are presented here for their general interest and as background for the discussion of correlations between electroencephalograms and other observations.

In table 86 data are presented for the comparison of the summary diagnoses. Here generalized and focal abnormalities are considered independently, so that tracings judged to show both kinds of abnormalities are counted under both headings. Of the tracings considered by reader B to show generalized abnormality, reader A considered 60 percent to be normal or borderline. Correspondingly, of the 12 that reader A considered to have generalized abnormality, reader B called 5 normal. The situation with respect to focal abnormalities was a little better: The proportions of each such reader's diagnoses called normal by the other reader were 46.2 percent and 26.9 percent, respectively.

The types of generalized abnormalities diagnosed were, not surprisingly, somewhat different. Reader A observed 2 instances of dysrhythmia and 10 of slow waves. Reader B saw no generalized abnormalities in half of these tracings. There were but six tracings which both readers thought to show generalized abnormalities, and the comparative diagnoses were:

	Reader A	Reader B
4 cases	Slow	Slow.
1 case	Slow	Paroxysmal slow.
1 case	Dysrhythmia	Paroxysmal slow.

Types of focal abnormalities were as inconsistently diagnosed by the 2 readers even in the 18 tracings for which they agreed that focal abnormalities existed. Diagnoses were identical in seven cases: two of asymmetry, three of paroxysmal slow waves, and one each of asymmetry combined with paroxysmal slow waves and of slow waves. The other 11 were quite varied:

	Reader A	Reader B
1 case	Irregular	Asymmetry.
1 case	Irregular	Asymmetry plus other, unspecified.
1 case	Paroxysmal slow	Asymmetry.
1 case	Paroxysmal slow	Paroxysmal slow plus spikes.
1 case	Paroxysmal slow	Asymmetry plus slow.
1 case	Spikes	Paroxysmal slow plus spikes.
5 cases	Other, unspecified	Asymmetry.

The findings presented above indicate that the art of interpretation of electroencephalographic tracings is quite subjective. Without decrying the usefulness of such tracings in the clinical management of patients, or even in diagnosis, it seems clear that the state of the art is not entirely satisfactory for the purposes of general surveys, since there is apparently wide disagreement as to interpretive criteria. Nevertheless, affected as they are by subjective considerations, it turned out that correlations did exist between the

Table 86.—*Comparison of Independent Interpretations of EEG Tracings*

Reader A	Reader B							
	Total		Normal or borderline		Generalized abnormality		Focal abnormality	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Normal or borderline	107	76. 4	81	89. 0	12	60. 0	18	46. 2
Generalized abnormality	12	8. 6	5	5. 5	6	30. 0	3	7. 7
Focal abnormality	26	18. 6	7	7. 7	5	25. 0	18	46. 2
Total	140	100. 0	91	100. 0	20	100. 0	39	100. 1

interpretations originally reported by the four centers and certain other observations.

2. Characteristics of the Wound

In the following discussion the EEG results are presented in three classes: those electroencephalograms which were normal or borderline, those which showed a generalized abnormality, and those which showed a focal abnormality. The last two groups are not mutually exclusive, and men with both generalized and focal abnormalities are, except in table 87, counted in both classes.

Slightly more than half of the patients examined had a normal electroencephalogram, and 9 percent had an electroencephalogram which was borderline (table 87). In terms of age of the patients, it is apparent that older men were more likely to have normal electroencephalograms than younger men, the latter having more of both focal and generalized abnormalities (table 88). Statistically this is significant, and possibly may be correlated with a certain instability of the younger nervous system, which would appear to be more easily disturbed by the stress of a head injury.

Table 87.—*General Characteristics of Follow-up Electroencephalogram*

Interpretation of follow-up EEG	Number	Percent
Normal	333	56.0
Borderline	54	9.1
Generalized abnormality only	73	12.3
Focal abnormality only	98	16.5
Generalized and focal abnormalities	37	6.2
Total with EEG	595	100.1
No EEG at follow-up	144	.....
Total examined	739	.....

Table 88.—*Relation of EEG at Follow-up to Age at Injury*

Age at injury	Number done	EEG at follow-up					
		Normal or borderline		Generalized abnormality		Focal abnormality	
		Number	Percent	Number	Percent	Number	Percent
Total	595	387	65.0	110	18.5	135	22.7
18–20	107	67	62.6	21	19.6	28	26.2
21–23	157	87	55.4	37	23.6	46	29.3
24–26	131	84	64.1	27	20.6	30	22.9
27–29	90	66	73.3	13	14.4	15	16.7
30–32	65	48	73.8	8	12.3	10	15.4
≥33	45	35	77.8	4	8.9	6	13.3

The severity of wounding, which may be examined in terms of a number of factors, obviously influences the electroencephalogram in a very positive fashion (table 89). It is worthy of note that the proportion of men with generalized abnormalities is much less affected by the overall severity of the injury (as measured by the R–I group) than the proportion with focal abnormalities. Groups 1 and 2 (which are alike in injury score but differ as to source, whether from Army hospital admissions or VA disability rosters) were quite similar with respect to electroencephalographic results. These groups have, therefore, been combined to examine in more detail the nature of the EEG abnormality in relation to severity of injury.

Table 89.—*Abnormalities of Follow-up EEG in Relation to R–I Group*

Interpretation of follow-up EEG	Total		R–I group					
			1 and 2		3		4	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Total done	595	100.0	234	100.0	285	100.0	76	100.0
Normal or borderline	387	65.0	182	77.8	175	61.4	30	39.5
Generalized abnormality	110	18.5	32	13.7	56	19.6	22	28.9
Irregular (dysrhythmia)	27	4.5	7	3.0	17	6.0	3	3.9
Slow (including paroxysmal)	88	14.8	29	12.4	39	13.7	20	26.3
Other, including fast or spiky	20	3.4	3	1.3	12	4.2	5	6.6
Focal abnormality	135	22.7	24	10.3	77	27.0	34	44.7
Irregularity	6	1.0	1	.4	4	1.4	1	1.3
Asymmetry	62	10.4	11	4.7	35	12.3	16	21.1
Hypersynchrony	4	.7	1	.4	2	.7	1	1.3
Paroxysmal slow waves	65	10.9	8	3.4	37	13.0	20	26.3
Paroxysmal fast waves	12	2.0	4	1.7	6	2.1	2	2.6
Spikes, spikes and waves	15	2.5	3	1.3	7	2.5	15	6.6
Other	14	.7	1	.4	3	1.1	.....	.....

It is also apparent from table 89 that to the extent that generalized abnormalities are more frequent in severely injured men, the cause is an increase in the frequency of slow waves (including paroxysmal slow waves) and in patterns characterized as fast or spiky. The types of focal abnormalities which are more frequent in the severely injured men are asymmetry and paroxysmal slow waves, which are almost wholly responsible for the increased number of focal abnormalities previously noted. Focal spikes, or spike and wave complexes, are also increased, but the total frequency of these abnormalities is small. Irregularity, hypersynchrony, and paroxysmal fast waves are apparently not altered in frequency by the severity of injury, and it is perhaps justified to conclude that these abnormalities do not result from trauma to the brain.

It is apparent that men with penetrating wounds have a much higher percentage of abnormal EEG's than other men (table 90). This difference

is quite significant. The penetrating and perforating wound groups have an excess of men with generalized abnormalities, but the really impressive difference is in the high proportion of focal abnormalities—only 5.8 percent for uncomplicated fractures but 30.1 percent for compound comminuted fractures, and 42.9 percent for perforating wounds.

The region wounded does not seem to be highly correlated with the electroencephalogram. There is a suggestion that wounds of the parietal and occipital regions are somewhat more likely to produce focal abnormalities than wounds of other regions. That the electroencephalographic abnormalities coincide with the site of wounding is not entirely correct, since it is apparent that the electroencephalographic abnormalities may be present in sites both adjacent to and some distance from the actual site of wounding.

Table 90.—*Relation of EEG at Follow-up to Type of Wound*

Type of wound	Number done	EEG at follow-up					
		Normal or borderline		Generalized abnormality		Focal abnormality	
		Number	Percent	Number	Percent	Number	Percent
Total	595	387	65.0	110	18.5	135	22.7
Uncomplicated fractures	52	41	78.8	8	15.4	3	5.8
Compound comminuted fracture, penetrating wound	345	199	57.7	75	21.7	104	30.1
Perforating wound	21	8	38.1	5	23.8	9	42.9
Other, excluding fractures	177	139	78.5	22	12.4	19	10.7

The fact that the severity of injury, as reflected in the R–I groups, was strongly correlated with EEG abnormalities leads one to expect that the specific observations from which the injury score was built would also show close relationships with the EEG, and this is, in fact, true. The size of the cranial defect and the depth of the head wound (table 91) are both well reflected by the EEG results. Superficial wounding (of the scalp), penetration of the cranium, or even of the dura mater, are relatively similar in the proportions attended by EEG abnormalities, but the brain waves of men in whom there was penetration of the brain are distinctly worse than those of men with more superficial wounds, while those of men with penetration to the ventricles seemed still worse. The difference between the last 2 groups mentioned is not statistically significant (only 22 men were coded as having penetration to the ventricles), but the first difference mentioned is highly significant.

Complications of wounding which would tend to aggravate the damage to the brain might be expected to increase the proportion of abnormal electroencephalograms, but such is not the case with the possible exception of fungus cerebri. Men with ulcerating cerebral herniations have a 56-percent

incidence of EEG abnormalities, chiefly (46 percent) focal. The period of unconsciousness, which indicates only roughly the severity of wounding, as has been noted in chapter III, is but weakly related to the changes in the electroencephalogram. Men unconscious more than 1 day do have a statistically significant, albeit small, excessive frequency of abnormality as compared with men unconscious 2 hours or less . There is, however, no significant difference between the EEG abnormalities in the men who did not show any impairment of consciousness and those who showed even considerable impairment (2 to 24 hours of unconsciousness).

Table 91.—*Relation of EEG at Follow-up to Depth of Head Wound*

Depth of wound	Number done	EEG at follow-up					
		Normal or borderline		Generalized abnormality		Focal abnormality	
		Number	Percent	Number	Percent	Number	Percent
Total	588	381	64.8	109	18.5	135	23.0
Scalp	179	138	77.1	24	13.4	20	11.2
Cranial	109	83	76.1	14	12.8	14	12.8
Dural	16	11	68.8	3	18.8	2	12.5
Brain	262	141	53.8	62	23.7	88	33.6
Ventricle	22	8	36.4	6	27.3	11	50.0

Men who had an immediate neurological deficit of any character have significantly more abnormal EEG's than men with no deficit (table 92). All deficits are accompanied by elevation of the proportions, especially of focal abnormalities. Men with hemianopsia seem even worse from the standpoint of EEG abnormalities than men with other deficits, and with respect to men with aphasia the difference is statistically significant.

The presence of intracranial foreign bodies is also an indication of the severity of wounding as reflected in the EEG (table 93). In the classes in which a foreign body was present before debridement there was a higher proportion of abnormal EEG's than in the class in which there were no bone or metal fragments. The differences are all significant except for the class of incomplete removal of metal, where the difference is relatively small. This class (in which metal only remained) differs significantly from the class in which both bone and metal remained. In summary, then, remaining bone fragments seem to be of greater significance than remaining metallic foreign bodies. It is possible that the reason for this lies in the greater average severity of the wound in men with remaining bone fragments.

Complications after debridement also apparently aggravate the severity of wounding, for those men who had complications were more likely than men without complications to have abnormal EEG's at follow-up (table 94). In particular, frank infection, fungus cerebri, and cerebrospinal fluid

Table 92.—*Relation of EEG at Follow-up to Immediate Neurological Deficit*

Neurological deficit at time of wound	Number done	EEG at follow-up					
		Normal or borderline		Generalized abnormality		Focal abnormality	
		Number	Percent	Number	Percent	Number	Percent
Total	589	382	64.9	110	18.7	134	22.8
None	336	250	74.4	47	14.0	48	14.3
Hemiplegia	122	58	47.5	33	27.0	45	36.9
Hemianopsia	42	15	35.7	12	28.6	21	50.0
Aphasia	80	46	57.5	19	23.8	23	28.8
Other	96	52	54.2	22	22.9	30	31.2

Table 93.—*Relation of EEG at Follow-up to Intracranial Foreign Bodies*

Foreign bodies	Number done	EEG at follow-up					
		Normal or borderline		Generalized abnormality		Focal abnormality	
		Number	Percent	Number	Percent	Number	Percent
Total	484	308	63.6	88	18.2	120	24.8
No foreign bodies	167	126	75.4	23	13.8	21	12.6
Complete removal bone and metal	147	87	59.2	29	19.7	42	28.6
Incomplete, bone removal	54	28	51.9	9	16.7	23	42.6
Incomplete, metal removal	66	45	68.2	12	18.2	14	21.2
Incomplete, bone and metal removal	50	22	44.0	15	30.0	20	40.0

leaks seem to be associated with a relatively high probability of abnormal EEG.

Men who have had cranioplasty tend to have fewer normal EEG's than men without cranioplasty, and the abnormalities tend to be of the focal type, but the time of cranioplasty relative to the injury seems to be unrelated to the probability of EEG abnormalities (table 95). In large part, the correlation between cranioplasty and EEG abnormalities is related to the greater average severity of wound in those who had cranioplasty. Whether the patient had one or two or more cranioplasties seems to have virtually no effect upon the EEG.

Table 94.—*Relation of EEG at Follow-up to Complications After Debridement*

Complication	Number done	EEG at follow-up					
		Normal or borderline		Generalized abnormality		Focal abnormality	
		Number	Percent	Number	Percent	Number	Percent
Total	496	314	63.3	94	19.0	121	24.4
None	424	280	66.0	76	17.9	94	22.2
Frank infection	19	5	26.3	6	31.6	8	42.1
Meningitis	19	10	52.6	6	31.6	7	36.8
Fungus cerebri	10	4	40.0	3	30.0	5	50.0
Cerebrospinal leak	14	4	28.6	4	28.6	7	50.0
Other	23	16	69.6	5	21.7	5	21.7

3. Neurological Deficit

The neurological deficit as indicated both by the subjective complaints of the patient and the objective findings at the time of discharge definitely seems to be related to abnormality in the electroencephalogram. The symptoms of impaired memory and impaired mentation in particular are significant in correlation with the abnormalities of the electroencephalogram, while the other symptoms are not apparently reflected by the electroencephalogram (table 96).

Most of the objective neurologic findings at the time of discharge from service appear to have significance with respect to the follow-up EEG findings (table 97). Exceptions are personality changes and the miscellaneous

Table 95.—*Relation of EEG at Follow-up to Cranioplasty*

Time of cranioplasty after injury	Number done	EEG at follow-up					
		Normal or borderline		Generalized abnormality		Focal abnormality	
		Number	Percent	Number	Percent	Number	Percent
Total	595	387	65.0	110	18.5	135	22.7
No cranioplasty	355	264	74.4	48	13.5	52	14.6
≤3 months	59	29	49.2	16	27.1	21	35.6
3–6 months	122	66	54.1	29	23.8	39	32.0
6–48 months	59	28	47.5	17	28.8	23	39.0

findings grouped under the heading "other and unknown." In particular the men with field defects (chiefly hemianopsia) show a high frequency of focal abnormalities: 25 of 42, or almost 60 percent. On the other hand, men with cortical sensory disturbances seem to have generalized EEG

Table 96.—*Relation Between EEG at Follow-up and Neurological Symptoms at Discharge From Service*

Neurological symptoms at discharge from service	Number done	EEG at follow-up					
		Normal or borderline		Generalized abnormality		Focal abnormality	
		Number	Percent	Number	Percent	Number	Percent
All men	595	387	65.0	110	18.5	135	22.7
None	252	173	68.7	38	15.1	54	21.4
Headache	192	124	64.6	42	21.9	40	20.8
Dizziness	79	47	59.5	19	24.1	20	25.3
Impaired mentation, lack of concentration	14	5	35.7	7	50.0	6	42.9
Impaired memory	22	4	18.2	9	40.9	13	59.1
Tinnitus	19	13	68.4	2	10.5	5	26.3
Irritability, fatiguability, insomnia, etc.	36	21	58.3	9	25.0	7	19.4
Posttraumatic syndrome	97	67	69.1	16	16.5	18	18.6

Table 97.—*Relation Between EEG at Follow-up and Neurological Abnormalities at Discharge From Service*

Neurological abnormality at discharge from service	Number done	EEG at follow-up					
		Normal or borderline		Generalized abnormality		Focal abnormality	
		Number	Percent	Number	Percent	Number	Percent
All men	595	387	65.0	110	18.5	135	22.7
None	267	188	70.4	46	17.2	44	16.5
Hemiplegia or hemiparesis	85	35	41.2	23	27.1	38	44.7
Paraparesis	4	2	50.0	1	25.0	1	25.0
Aphasia	54	21	38.9	17	31.5	22	40.7
Spasticity	24	11	45.8	5	20.8	8	33.3
Field defect	42	11	26.2	12	28.6	25	59.5
Cortical sensory disturbances	32	15	46.9	12	37.5	8	25.0
Personality changes	17	11	64.7	4	23.5	3	17.6
Visual disturbances	13	7	53.8	1	7.7	5	38.5
Other and unknown	202	141	69.8	32	15.8	40	19.8

abnormalities more frequently than men not so afflicted, although the difference is not statistically significant. This group is rather noteworthy in that it is quite exceptional in this material for a group of men to exhibit an increased frequency of generalized EEG abnormalities in the absence of an increased frequency of focal abnormalities of even greater magnitude.

4. Pneumoencephalogram

Among the 595 men for whom EEG results at follow-up were available, only 88 pneumoencephalograms (PEG) were reported in the Army clinical records for the hospital admission directly following occurrence of the head wound. These were not done at a uniform time, but almost all (86 of 88) were within a year of injury; 30 were within 3 months and another 44 within the period 3 to 6 months, so that five-sixths were within 6 months of injury.

It seems plain (table 98) that early PEG abnormalities of unilateral or local type (ventricular dilatation or absence of air) are fairly well reflected by focal EEG abnormalities several years later, at follow-up. Unfortunately, the samples are so small that the differences do not achieve statistical significance; however, the probability (one-tailed) comes to between .05 and .10, and in view of the low power of the significance test (because of the paucity of cases) we may regard the data as quite consistent with the existence of a moderately strong relationship between early local PEG abnormalities and late focal EEG abnormalities.

Table 98.—*Relation Between Follow-up EEG and Pneumoencephalogram in Army Hospital*

Pneumoencephalogram in Army hospital	Number done	EEG at follow-up					
		Normal or borderline		Generalized abnormality		Focal abnormality	
		Number	Percent	Number	Percent	Number	Percent
Total done	88	44	50.0	20	22.7	33	37.5
Normal	26	17	65.4	5	19.2	6	23.1
Abnormal	62	27	43.5	15	24.2	27	43.5
Generalized ventricular dilatation	21	12	57.1	4	19.0	6	28.6
Unilateral ventricular dilatation on side of lesion	17	6	35.3	4	23.5	8	47.1
Local ventricular dilatation on side of lesion or over cortex	22	10	45.5	4	18.2	10	45.5
Local absence of air in subarachnoid space	8	4	50.0	1	12.5	4	50.0
Inadequate filling of ventricle	11	5	45.5	3	27.3	4	36.4
Other abnormality	13	4	30.8	4	30.8	7	53.8

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5. Early Electroencephalograms

It has been seen that the correspondence between independent readings of the same tracings is far from good; it is, therefore, not surprising that the correlations are quite low between the readings of EEG's made and interpreted in Army hospitals within 6 months of injury and those made and interpreted at follow-up (table 99).

Of 80 men called abnormal at follow-up, 50 were called abnormal at 6 months, while of 104 normal at follow-up, 41 were abnormal at 6 months.

Table 99.—*Correlation of EEG Findings at 6 Months After Injury and at Follow-up*

Electroencephalogram within 6 months of injury	Total	EEG at follow-up					
		Normal or borderline		Generalized abnormality		Focal abnormality	
		Number	Percent	Number	Percent	Number	Percent
Total	184	104	56.5	44	23.9	56	30.4
Normal or borderline	93	63	67.7	18	19.4	18	19.4
Generalized abnormalities	53	23	43.4	15	28.3	24	45.3
Focal abnormalities	62	26	41.9	19	30.6	27	43.5

Similarly, if the EEG findings at 6 to 12 months, 12 to 24 months, and more than 24 months after injury are compared to the findings at follow-up, the relationships are not very strong. Table 100 shows the correlation of the follow-up EEG and those made at varying times after injury. The numbers of readings are not very large, but plainly the correlations between early and late EEG's are disappointingly small. Admittedly, the men chosen for repeated early EEG examination were probably highly selected.

It is concluded that the variation of interpretation of tracings by different electroencephalographers is probably so great as to interdict any attempt to follow temporal changes utilizing the different interpreters at different times, even if the interpretation be described simply as normal or abnormal.

6. Rehabilitation

An examination of the electroencephalogram in terms of rehabilitation does not show impressive relationship. The men who were not regularly employed exhibited a significant excess of focal abnormalities over those who were employed. Similarly, men coded for "regression" in general work progress were significantly more likely to have focal abnormalities than men coded "no work progress" or "school and advancement" (table 101). Both the home and social adjustment seem to be uncorrelated with the electroencephalogram.

Table 100.—*Correlation of Follow-up and Early EEG's*

Follow-up EEG	Total	Fractions called abnormal at specified time after injury			
		≤ 6 months	6 to 12 months	12 to 24 months	≥ 25 months
Abnormal	80	50/80 (62 percent)	32/55 (58 percent)	26/35 (74 percent)	15/27 (56 percent)
Normal or borderline	104	41/104 (39 percent)	11/35 (31 percent)	11/35 (31 percent)	17/37 (46 percent)

Table 101.—*Relation Between EEG and Vocational and Social Rehabilitation*

Adjustment	Number done	EEG at follow-up					
		Normal or borderline		Generalized abnormality		Focal abnormality	
		Number	Percent	Number	Percent	Number	Percent
Total	595	387	65.0	110	18.5	135	22.7
Work status:							
Unemployed or occasional jobs	94	56	59.6	16	17.0	30	31.9
School	35	23	65.7	6	17.1	7	20.0
Employed	458	304	66.4	85	18.6	96	21.0
Work progress:							
None	165	114	69.1	26	15.8	29	17.6
Regression	121	68	56.2	23	19.0	44	36.4
Advancement (including school)	300	200	66.7	59	19.7	60	20.0
Home adjustment:							
Depressed, restless, unhappy	69	46	66.7	8	11.6	15	21.7
Social adjustment:							
Has difficulty	133	84	63.2	23	17.3	35	26.3

7. Clinical Abnormalities

The relation between the EEG and clinical symptomatology is not strong. None of the various symptom groups exhibits a significantly high percentage of focal abnormalities (table 102). However, men with abnormal

Table 102.—*Relation Between EEG and Clinical Symptomatology at Follow-up*

Symptom	Number done	EEG at follow-up					
		Normal or borderline		Generalized abnormality		Focal abnormality	
		Number	Percent	Number	Percent	Number	Percent
All men	595	387	65.0	110	18.5	135	22.7
None	55	40	72.7	8	14.5	9	16.4
Headache	154	99	64.3	29	18.8	36	23.4
Dizziness	40	30	75.0	4	10.0	6	15.0
Irritability or nervousness	56	37	66.1	10	17.9	13	23.2
Impaired memory, mentation, concentration	37	18	48.6	13	35.1	13	35.1
Easy fatiguability or insomnia	30	14	46.7	11	36.7	10	33.3
Posttraumatic syndrome	333	214	64.3	60	18.0	75	22.5
Not stated	2	1	.....	1	.....	1	.....

findings upon neurological examination (table 103) have a significantly high proportion of abnormal electroencephalograms. Every specific neurological abnormality is accompanied by a significantly increased frequency of focal abnormalities on EEG. For men with hemianopsia, the most extreme class, the proportion with focal abnormalities is almost 60 percent, nearly four times the incidence in men without neurological abnormalities. On the other hand, generalized abnormalities are much less closely correlated with the neurological findings. The elevations of the proportions of generalized EEG changes in men with neurological abnormalities are always smaller, usually considerably, than the corresponding elevations in the proportions of focal abnormalities. Moreover, in only four instances do the elevations above the incidence of generalized EEG changes in normal men prove to be statistically significant at the 5-percent level: for men with hemiplegia, with hemianopsia, with cortical sensory impairment, and with mental impairment. It is perhaps significant that this last class shows the greatest elevation in the proportion with generalized abnormalities on EEG.

Table 103.—*Relation Between EEG and Neurological Status at Follow-up*

Neurological finding	Number done	EEG at follow-up					
		Normal or borderline		Generalized abnormality		Focal abnormality	
		Number	Percent	Number	Percent	Number	Percent
Total	595	387	65.0	110	18.5	135	22.7
Normal	280	207	73.9	41	14.6	44	15.7
Hemiparesis of hemiplegia	128	63	49.2	32	25.0	48	37.5
Hemianopsia	52	15	28.8	15	28.8	31	59.6
Cortical sensory impairment	128	64	50.0	31	24.2	46	35.9
Aphasia	48	24	50.0	11	22.9	17	35.4
Mental impairment	79	36	45.6	24	30.4	29	36.7
Cranial nerve palsy	142	86	60.6	23	16.2	37	26.1
Cerebellar syndrome	4	1	25.0	1	25.0	3	75.0
Other	13	10	76.9	2	15.4	1	7.7

8. Epilepsy

The electroencephalograms made at the time of follow-up seem to correlate reasonably well with epilepsy (table 104). In each of the injury groups, the men with fits are much more likely to have focal EEG abnormalities than those without fits; in group 4—the most severely injured men—fits are also accompanied by an increased proportion of generalized abnormalities. This increase is caused by a relatively large number of tracings characterized as slow or paroxysmal slow. On the other hand, even in those group 4 men who had epilepsy, nearly one-third were classed as having normal or borderline EEG's.

Abnormalities in no single area seem to be associated with epilepsy. The most frequent alteration in all groups with or without epilepsy are asymmetry and paroxysmal slow waves. The type of focal abnormality does not seem to be related to an epilepsy. Spikes, commonly considered as related to seizures, were noted in only 10 of 168 men who had epilepsy, or 6.0 percent. Among men who did not have seizures, the relative frequency was 5 in 427, or 1.2 percent. This difference, although statistically significant, is not impressive. Neither is there any abnormality characteristic of epilepsy induced by hyperventilation.

The correlation between the EEG and type of epilepsy (table 105) emphasizes the high ratio of focal abnormalities to generalized abnormalities

Table 104.—*Correlation of EEG With Epilepsy*

R–I group and presence of epilepsy	Number done	EEG at follow-up					
		Normal or borderline		Generalized abnormality		Focal abnormality	
		Number	Percent	Number	Percent	Number	Percent
Group 3:							
Epilepsy absent	192	125	65.1	35	18.2	45	23.4
Epilepsy present	93	50	53.8	21	22.6	32	34.4
Group 4:							
Epilepsy absent	26	14	53.8	5	19.2	9	34.6
Epilepsy present	50	16	32.0	17	34.0	25	50.0

Table 105.—*Correlation of Type of Epileptic Attack and EEG at Follow-up*

Type of attacks	Total	EEG at follow-up					
		Normal or borderline		Generalized abnormality		Focal abnormality	
		Number	Percent	Number	Percent	Number	Percent
Total with fits	168	80	47.6	44	26.2	63	37.5
Minor attacks	32	15	46.9	10	31.3	11	34.4
Focal attacks	29	10	34.5	9	31.0	14	48.3
General (grand mal)	68	34	50.0	17	25.0	27	39.7
Psychomotor	9	5	55.6	3	33.3	1	11.1
Other types	5	3	60.0	.....	.....	2	40.0
Focal and general attacks	29	12	41.4	10	34.5	11	37.9
General and minor	8	4	50.0	1	12.5	3	37.5
Other combinations and unknown	7	3	42.9	2	28.6	2	28.6

for most types. This is true not only of the group having focal attacks but also of those having generalized attacks. The differences between men having different kinds of fits do not attain statistical significance. If the type of epilepsy is a determinant factor in the electroencephalographic abnormality, its influence is little reflected in these data.

The groups categorized by aura seem relatively homogeneous with respect to abnormalities of the brain waves.

For a further study of the sensitivity of the electroencephalogram relative to epilepsy, a comparison was made between men who continued to have attacks and those whose attacks ceased. No difference could be discerned in the electroencephalograms made at 6 months or at follow-up between men whose fits were controlled for at least 2 years prior to follow-up and those who were continuing to have attacks. This is true for all aspects of the brain waves—generalized abnormalities, location or type of focal abnormalities, and alterations induced by hyperventilation.

9. Intelligence

Finally, the Wechsler-Bellevue IQ seems to have little relationship to the EEG's, although the deterioration as indicated by this test does seem to be so related in that men with abnormal deterioration tend to have abnormal EEG's more frequently than men with no deterioration (table 106). Moreover, the excess of abnormal EEG's is wholly confined to the category of focal abnormality. The statistical test of significance applied to the difference in the proportions with focal abnormality shows this difference to be significant at the 5-percent (one-tail) level.

Table 106.—*Relation Between EEG and Wechsler-Bellevue Intelligence Test*

Wechsler-Bellevue test	Number done	EEG at follow-up					
		Normal or borderline		Generalized abnormality		Focal abnormality	
		Number	Percent	Number	Percent	Number	Percent
Total	562	366	65.1	101	18.0	129	23.0
Intelligence scale:							
≤89	78	51	65.4	14	17.9	17	21.8
90–119	388	257	66.2	65	16.8	90	23.2
≥120	96	58	60.4	22	22.9	22	22.9
Abnormal deterioration:							
Present	121	70	57.9	24	19.8	36	21.1
Absent	441	296	67.1	77	17.5	93	29.8

10. Discussion

Electroencephalography was heralded as a means of determining and, to some degree, of measuring the function of the brain. While experience has shown the fallaciousness of this as an exact principle, it has demonstrated

that abnormalities of the brain waves do correlate with cerebral damage in a general way. Because the electrical potentials are related to functional rather than purely anatomical substrata, it should be understandable that a brain, damaged in one area, may have a normal electrical pattern assuming that:

a. The area is not so large that a diminution of potentials will be reflected in the total picture.
b. The area is electrically relatively silent and not giving rise to abnormal discharges.
c. The cortical margins of the area are functioning in an electrically normal fashion.
d. The area is not essential in the activation of subcortical structures which may influence the focal or general activity of the cerebral cortex.

That these conditions are met in 20 percent of cases of penetrating head wounds is apparent from an examination of table 107. Moreover, Williams' (138) serial examinations of head-wounded men indicate that there is a tendency for the electrical patterns of the damaged brain to revert to normal over a period of months. In the present series, this tendency is not so apparent; at 6 months after injury 52 percent of men had normal EEG's, and at follow-up, 6 or more years later, this had only increased to 57 percent. Allowing for some variation due to different readers, this can indicate only mild improvement. Perhaps this result should be expected since Williams reports rather rapid reversion to normal in 3 to 6 months; at later times the record may change little. Dow et al. (37) likewise found that the records of patients with closed head injuries returned to normal within a few weeks. Puech et al. (99) also found that the EEG's of head-injured (both closed and open) patients tended to become normal in about 3 months, with little change in the percentage of normal records (80 percent) after that time. They indicate a rather slower return to the normal state for patients developing epilepsy, but since their series is small (19 cases) and the factor of severity of the injury is not controlled, the differences are probably not significant. In cases of cerebral contusion characterized clinically by focal neurological signs after a closed head injury,

Table 107.—*EEG Findings After Dural Penetrating Head Wounds*

Author	Year	Number of cases	Percentage	
			Normal	Abnormal
Laufer and Perkins (76)	1946	81	19.7	80.3
Aita (2)	1947	100	10	90
Busse (26)	1947	103	19	81
Cramer et al. (31)	1949	442	21	79
Kaufman and Walker (70)	1949	[1] 83	22.9	77.1
		[2] 241	8.7	91.3
Hilterhaus and Bayer (64)	1955	300	14	86

[1] Without convulsions.
[2] With epilepsy.

Meyer-Mickeleit (88) states that the diffuse EEG abnormalities disappear in 6 months, at which time the alpha, previously slowed, regains its normal frequency. Focal alterations may persist for 2 years (25 percent of cases) or more (5 years in 15 percent of cases).

The prediction of posttraumatic epilepsy is generally admitted to be difficult and uncertain. Only a few authors have asserted that an epilepsy can be prophesied on the basis of electroencephalograms. Roseman and Woodhall (102) suggested that the development of spike foci over a period of time might be correlated with an impending convulsive diathesis. But Williams (138) observed such changes to occur over a period of 3 to 6 months in patients who did not develop seizures. Jasper and Penfield (69) similarly conclude that "the EEG cannot be depended upon, however, to predict the development and course of a potentially epileptogenic lesion of the brain, since regressive as well as progressive lesions are encountered."

Assuming that clinical manifestations of an epilepsy have occurred, does the EEG give information as to the posttraumatic origin of that convulsive state? There is more evidence concerning this point, although much of it is poorly controlled or uncontrolled.

Williams (138) found that there was a sharp decline in the percentage of EEG abnormalities of an unselected group of head-injured patients (both open and closed) in the first 2 months. The length of posttraumatic amnesia was found to correlate closely to the percentage of abnormal records—those men being unconscious for 1 week or more have 67 percent of abnormal records. The EEG was abnormal in 55.4 percent of the men with posttraumatic symptoms and in 8 percent of those symptomless. Williams concluded that the character of the changes seen in the EEG in traumatic epilepsy appear to be the same as in idiopathic epilepsy, and prediction of traumatic epilepsy does not appear possible by electroencephalographic examination during the period of recovery from a head injury. Moreover, he noted that clinical recovery is followed closely by improvement in the EEG.

Clark and Harper (29) while noting a pronounced change from "normal" to "focal" EEG changes as one moved from "closed" to "open penetrating head wounds," admitted that the EEG did not reveal with any degree of certainty those patients who had had convulsions.

Based on a comparison of 2 series—one of 102 cases of severe head injury (both open and closed) in both children and adults, and one of 175 posttraumatic epileptic patients, the criteria for which is not clearly defined, Gibbs et al. (52) conclude that focal paroxysmal disturbances are 21 times as common among posttraumatic epileptics as among head-injured men without epilepsy. Because of these generalities in the population, the rather specific conclusions drawn by the authors require considerable reservation. This is particularly true since Heppenstall and Hill (63) have demonstrated that children and adults respond differently to head injury.

Roseman and Woodhall (102) reported on serial EEG studies of 75 men with brain laceration, stating that in 4 cases the onset of the convulsive state was predicted following the gradual alteration from delta activity into "one or more characteristic types of epileptogenic encephalographic foci," but admit that similar findings were present in 15 cases who did not develop

seizures. They concluded that certain broad predictions could be made regarding the resolution of cerebral damage or its metamorphosis into a focus of epileptic activity.

Jasper and Penfield (69) state that in posttraumatic epilepsy the electroencephalogram revealed (in 90 percent of cases) a focal area of random spikes or sharp waves usually upon a background of random delta waves. Slow waves alone were not considered a reliable guide to an epileptogenic focus. They conclude that the EEG cannot be depended upon to predict an epilepsy. Similarly, Puech et al. (99) state that posttraumatic epilepsy does not have a constant electrical record. Kaufman and Walker (70) in 1949 showed that in comparable wounds the EEG findings were essentially the same in patients sustaining penetrating head injuries with and without complicating seizures.

Finally, the question has been raised regarding cerebral trauma as a causative factor in petit mal. Most authors [Williams (138), Gibbs (52), Hilterhaus and Bayer (64), and Kaufman and Walker (70)] whose material has been composed largely of adults, have not seen a typical three per second spike and wave pattern as a sequel of a head injury. Kellaway (71) believes that such a pattern requires considerable time to develop, and concludes "if generalized spike wave patterns are seen in the acute phase of head injury or without antecedent abnormality of other type, the chances are that they predated the injury." Jasper and Penfield (69) report three cases of petit mal or petit mal variant in their posttraumatic series. In their discussion the authors admit that all patients having this type of abnormality were under 14 years of age, and that "until such posttraumatic lesions (at the base of the brain or large areas of contused cortex) have been shown to be present in cases with these EEG findings, one may assume that the probabilities are greatest that they are essential rather than posttraumatic epilepsy." Meyer-Mickeleit and Schneider (89) also report that "three confirmed posttraumatic epilepsies of childhood showed three per second bilateral symmetrical spike and wave discharges with clinical 'absences'." These authors believe that the age of the injured patient is more important in the type of epilepsy than the etiology. It would seem that in children no positive statement can be made regarding the role of trauma in petit mal, but in adults there is no evidence that cerebral injuries cause such a convulsive state.

One may conclude that the evidence of a single EEG is not very substantial whether positive or negative, but a series of tracings, especially if one of them antedates the trauma, is of great value both from the diagnostic and prognostic standpoints. Since EEG's are so commonly done, in the future it may not be difficult to obtain records made before a head injury occurred.

C. PNEUMOENCEPHALOGRAPHY

1. Findings

The results of air studies were originally classified as normal, generalized ventricular dilatation, unilateral ventricular enlargement, localized ventric-

ular dilatation at site of lesion, abnormal air over cortex, absence of air in subarachnoid space, inadequate ventricular filling, and combinations of these and other abnormalities. However, so few PEG's were performed during the original Army hospitalization that, for the purpose of analysis, it seemed necessary to combine the findings into fewer categories as:

a. Normal.
b. Generalized abnormality, including generalized ventricular dilatation, with or without localized ventricular dilatation, and abnormal air over cortex.
c. Localized abnormality, including unilateral ventricular dilatation, localized ventricular dilatation.
d. Other, including abnormal air over cortex, absence of air over cortex.

As might be expected, air studies were more frequently done on the more seriously injured men. Of the total of 128 men with PEG's, 46 were in R–I group 4. Only 3 of the 99 men in R–I group 1 had PEG's as contrasted with 48 percent of those in R–I group 4. In R–I groups 2 and 3 the proportions with PEG's were 5 and 15 percent, respectively.

The frequency of abnormal findings might be expected to be affected by factors relating to greater severity of wounding, such as increasing neurological disability, high incidence of epilepsy, etc., but since the number of men in groups 1 and 2 for whom PEG's were reported is so small, such correlations cannot be well demonstrated in this material.

Only the wounds of the temporal region seem to have a significantly larger percentage of normal air studies (44 percent) than those of other regions. Degree of severity of wounding, in terms of size and depth of the wound correlates with the pneumoencephalographic abnormalities (table 108). The immediate complications did not seem to influence the abnormalities of the PEG except for the cases with fungus cerebri, of whom only 1 person in 20 had a normal air study. Localized abnormalities in patients having cerebral fungi were almost twice as common as in the total group.

The criterion of unconsciousness following injury correlates with the presence of abnormality: Few men unconscious more than 2 hours had normal air studies. The neurological deficit at the time of wounding, too, was related to PEG abnormalities in that men with hemiplegia had less than half as many normal air studies as the entire group, and had a corresponding increase in localized abnormalities. Except that the absence of intracranial foreign bodies was likely to be associated with a high percentage of normal air studies, the presence of intracranial bone or metal seemed to influence the air studies little.

Those patients whose cranial defects required repair had more ventricular distortions than those whose heads did not require cranioplasty; while 77 percent of the first group had abnormalities, only 50 percent of the latter were so recorded. The abnormality was usually a localized ventricular distortion.

The pneumoencephalographic findings bear a direct relationship to the subjective complaints of the patient at follow-up, by which time few men were asymptomatic and three-fifths had a traumatic syndrome (table 109).

Table 108.—*Correlation of PEG and Characteristics of Wounding*

Characteristic	Total number of men	Normal (percent)	Generalized abnormality (percent)	Localized abnormality (percent)	Other abnormality (percent)
Total	129	31.8	21.7	26.4	20.2
Mean diameter of cranial defect:					
No cranial defect	15	53.3	26.7	.....	20.0
≤2 cm	20	40.0	10.0	30.0	20.0
2 – 4 cm.	52	36.5	23.1	23.1	17.3
>4 cm	23	13.0	26.1	43.5	17.4
Other (including gutter wounds) and unknown	19	15.8	21.1	31.6	31.6
Depth of wound:					
Scalp, cranium, or dura	35	51.4	20.0	14.3	14.3
Brain or ventricle	94	24.5	22.3	30.9	22.3
Period of unconsciousness:					
None	19	52.6	15.8	21.1	10.5
≤2 hours	22	50.0	22.7	4.5	22.7
>2 hours	32	12.5	25.0	31.3	31.3
Other and unknown	56	28.6	21.4	33.9	16.1

Table 109.—*Correlation of PEG and Clinical Symptomatology at Follow-up*

Symptom	Total number of men	Normal (percent)	Generalized abnormality (percent)	Localized abnormality (percent)	Other abnormality (percent)
Total	101	29.7	22.8	23.8	23.8
Posttraumatic syndrome	60	26.7	26.7	21.7	25.0
Other symptoms	33	27.3	18.2	27.3	27.3
No symptoms	8	62.5	12.5	25.0	.....

A neurological deficit of almost any type decreased the probability of a normal air study, and increased the chances of a localized ventricular distortion. It is noteworthy, however, that of the men having no detectable neurological abnormalities, almost half were recorded as having pneumoencephalographic abnormalities. No one category of defect was particularly apt to be accompanied by PEG abnormalities (table 110).

With regard to employment at follow-up, perhaps the most interesting point is that of the 67 men who were working, only 40.3 percent had a normal pneumonecephalogram.

The occurrence of epilepsy was likely to be associated with abnormalities of the pneumoencephalogram. Only 12 percent of men who had multiple fits were recorded as having normal air studies (table 111).

Finally, although general intelligence, as measured by the Wechsler-Bellevue test, did not seem to be correlated with morphological changes

in the brain, abnormal deterioration was associated with pneumoencephalographic changes (table 112).

2. Discussion

That both closed and open head injuries will produce ventricular changes easily demonstrable by pneumoencephalography has been recognized for decades (34). Such changes in the ventricular size and shape may occur within 10 days of a head injury and continue to become more pronounced over a period of months. Browder and Hollister (23) made serial examinations after head injuries, noting not only progressive ventricular dilatation but also that such changes might go on during clinical recovery from a hemiplegia. Troland et al. (126) also emphasized the early occurrence of ventricular alterations. Even mild head injuries may produce dilatation of the ventricles; Klaue (73) reported that 46 percent of concussed patients had slight ventricular enlargement.

The late changes in the ventricular system, noted early in Foerster's clinic in Breslau, were described as "the wandering ventricle"—a dilation and deviation of the ventricle toward the side of the lesion. Schwab (115) and Bielschowsky (17), in papers from this clinic, emphasized not only the

Table 110.—*Correlation of PEG and Neurological Abnormalities at Follow-up*

Abnormality	Total number of men	Normal (percent)	Generalized abnormality (percent)	Localized abnormality (percent)	Other abnormality (percent)
Total	101	29.7	22.8	23.8	23.8
None	28	53.6	28.6	3.6	14.3
Hemiparesis or hemiplegia	40	17.5	20.0	45.0	17.5
Hemianopsia	17	17.6	17.6	35.3	29.4
Cortical sensory impairment	36	19.4	16.7	41.7	22.2
Aphasia	20	10.0	20.0	35.0	35.0
Mental impairment	24	16.7	20.8	33.3	29.2
Cranial nerve palsy	33	12.1	30.3	27.3	30.3
Other and unknown	3	.....	.....	66.7	33.3

Table 111.—*Correlation of PEG and Epilepsy*

Epilepsy since injury	Total number of men	Normal (percent)	Generalized abnormality (percent)	Localized abnormality (percent)	Other abnormality (percent)
Total	101	29.7	22.8	23.8	23.8
None	55	38.2	20.0	20.0	21.8
One definite attack	3	66.7	33.3	.....	.....
>1 definite attack	41	12.2	26.8	31.7	29.3
Borderline attacks only	2	100.0	.....	.....	.....

Table 112.—*Correlation of PEG and Intelligence*

Wechsler-Bellevue test	Total number of men	Normal (percent)	Generalized abnormality (percent)	Localized abnormality (percent)	Other abnormality (percent)
Total	101	29. 7	22. 8	23. 8	23. 8
Intelligence scale:					
≤89	14	28. 6	28. 6	14. 3	28. 6
90–119	61	36. 1	18. 0	23. 0	23. 0
≥120	18	22. 2	38. 9	22. 2	16. 7
Unknown or not done	8	.....	12. 5	50. 0	37. 5
Abnormal deterioration:					
Present	22	13. 6	27. 3	22. 7	36. 4
Absent	79	34. 2	21. 5	24. 1	20. 3

ventricular enlargement, but also the sulcal prominence and in some cases, the failure of the ventricles to fill. Each noted that although there was an obvious correlation between the clinical and pneumoencephalographic findings in many cases, in some this did not hold. Subsequent authors (48, 62, 121) considered that air studies allowed a differentiation between patients with organic and patients with functional brain disturbance. However, most authors deprecated this view because of the many cases of ventricular abnormality without clinical concomitants and of abnormal neurological findings with a normal ventricular system. This study, too, finds little correlation between posttraumatic findings and degree of ventricular abnormality.

Since the early descriptions, many authors have discussed and illustrated the ventricular distortions. There is much emphasis upon the changes noted but little effort expended upon the proof that the changes were secondary to the injury and not due to some other factor. True, a few authors such as Fischer (46) and Kennedy (72) sound this warning, observing that similar changes can be found in patients without a history of head injury. To control this factor, Falk and Silfverskiöld (44) compared the ventricular changes in a series of patients with cephalalgia and with traumatic encephalopathy. Although they found a much higher proportion of abnormal pneumoencephalograms in their posttraumatic group than in the headache series (52/72 vs. 19/58), they noted that the percentage was almost as high in the cases which had no loss of consciousness (14/22) as in those insensible for some period of time.

It must be admitted that pneumoencephalography is not an accurate measure of either pathological alterations or normal functioning of the brain. As an indicator of function, it is particularly unreliable, and sensitive only in the sense that function correlates to some extent with morphology.

D. PSYCHOMETRY

The mental processes may be subjected to analysis in many ways. For the purpose of this study two formal tests were employed—the Wechsler-Bellevue and the Minnesota Multiphasic Personality Inventory. It was

hoped that the first of these tests might give evidence not only of the immediate intelligence of the individual, but an indication of deterioration which might have resulted from cerebral damage. The evaluation of the personality was considered of particular pertinence because of the presumed "functional" elements which enter into posttraumatic cerebral phenomena.

1. Intelligence as Determined by the Wechsler-Bellevue Test

"Intelligence" as discussed in this section is based upon the formal IQ obtained from the Wechsler-Bellevue test. This test is not a measurement of the native endowment of men with brain injuries, since such factors as aphasia, apraxia, and even hemiplegia, may influence the scores of certain categories. Emphasis will be placed upon these changes wrought by the brain injury—the deterioration score.

From the simple breakdown into low, intermediate, and high IQ, little data of clinical significance is obtainable (table 113). It will be noted that while half of the men with IQ under 90 were abnormally deteriorated, the proportion was less than 2 percent for men with IQ of 120 or more. This might be interpreted as showing either that low intelligence at follow-up was often caused by abnormal deterioration, perhaps as a result of the head wound, or alternatively, that "abnormal deterioration" only in part measures what its name implies and that the scoring rules are such that persons with low IQ often tend to be scored as having abnormal deterioration simply as an artifact of the way the test is constructed.

Table 113.—*IQ as Determined by Wechsler-Bellevue Test*

IQ	Number	Percent	Abnormally deteriorated	
			Number	Percent
≤89	88	12.9	44	50.0
90–119	470	68.8	99	21.1
≥120	125	18.3	2	1.6
Total	683	100.0	145	21.2

In order to learn more about the meaning of the "abnormal deterioration" score, military records were searched for results of the Army General Classification Test (AGCT) administered to most recruits during their first few weeks of training. This test has much in common with the Wechsler-Bellevue. Unfortunately, it turned out that the majority of the records had been destroyed, but it was possible to obtain AGCT scores for 153 men for whom Wechsler-Bellevue test results were also available at follow-up. A gross comparison of the AGCT prior to trauma and the Wechsler-Bellevue, made several years later, is presented in table 114 and discloses several interesting features. First, the correlation between the two scores is reasonably good: The correlation coefficient is +.65 and the mean Wechsler-Bellevue IQ increases regularly with increasing AGCT for

the whole sample and also for the subgroup of 102 men in whom no abnormal deterioration was noted. Of the 60 men with AGCT score less than 100, 25 (41.6 percent) were scored by the Wechsler-Bellevue for abnormal deterioration for age, while only 26 (28.0 percent) of the 93 men with AGCT scores of 100 or more were so scored. This clearly implies that the deterioration noted by the Wechsler-Bellevue, whatever it may be, was often at least implicit at the time of the soldier's entry into service, and, hence, cannot be considered a measure of deterioration from that point of time forward.

Table 114.—*Relation Between Wechsler-Bellevue IQ at Follow-up and Army General Classification Test at Entry Into Service*

AGCT	Total	Abnormal deterioration		Total	Abnormal deterioration	
		Absent	Present		Absent	Present
	Number of scores			Mean Wechsler-Bellevue score at follow-up		
≤69	9	4	5	90	94	86
70–79	13	10	3	93	95	87
80–89	16	9	7	98	98	97
90–99	22	12	10	106	107	104
100–109	30	17	13	108	112	103
110–119	26	18	8	112	113	108
120–129	28	24	4	117	119	104
130+	9	8	1	119	121	104
Total	153	102	51	107	110	101

On the other hand, clearly the relation between the AGCT and the Wechsler-Bellevue was affected by the head wound. An analysis of covariance in terms of the four R–I groups showed significant differences between the regressions of the Wechsler-Bellevue scores on the AGCT scores within groups. The correlation coefficients for the four groups were: group 1, +.72; group 2, +.86; group 3, +61; and group 4, +.45. It also seems significant that if attention be resricted to men whose AGCT scores were 90 or more, then, within the subgroup of those who were scored as having abnormal deterioration, there was no apparent relation between the AGCT and the Wechsler-Bellevue IQ. This would at least be consistent with the idea that for these men an extraneous influence (perhaps the head wound) was disturbing the relation between the AGCT and the Wechsler-Bellevue, and was being recognized by the latter test as "abnormal deterioration."

This rather complicated situation may be understood a little more clearly by reference to table 115. Here it can be seen that among those men whose original AGCT scores were 100 or more, abnormal deterioration was scored on the Wechsler-Bellevue almost entirely among men with wounds that penetrated the brain (15 out of 18). On the other hand, men with low

AGCT scores were equally likely to be scored for deterioration whether the wound extended to brain substance or not. It can be concluded that abnormal deterioration may be attributed to the head wound with confidence only for men whose AGCT scores were 100 or more.

Table 115.—*Relation Between Army General Classification Test Score, Wechsler-Bellevue Deterioration, and Depth of Wound*

AGCT score	Depth of wound					
	Scalp, cranium or dura or not shown			Brain or ventricle		
	Total number	With abnormal deterioration		Total number	With abnormal deterioration	
		Number	Percent		Number	Percent
Total.....	77	13	16.9	76	24	31.6
≤79.....	14	4	28.6	8	1	12.5
80–89.....	6	2	33.3	10	4	40.0
90–99.....	15	4	26.7	7	4	57.1
100–109.....	14	2	14.3	16	7	43.8
110–119.....	12	1	8.3	14	3	21.4
120+.....	16	.....	.....	21	5	23.8

A glance through the correlations of the Wechsler-Bellevue with factors of wounding reveals two main tendencies:

a. The relative independence of the IQ itself to all factors.
b. The close relationship between increasing deterioration and most factors contributing to the severity of injury.

Although the type of wound changes the proportions of the classes of intelligence only moderately, it reflects itself strongly in the abnormally deteriorated (table 116). The proportion of men abnormally deteriorated increases slowly as diameter of defect increases, but the effect is not strong. There is little apparent correlation with the IQ itself. The probability of deterioration also increases with increasing depth of wounding; little relationship to the IQ itself is seen (table 117). There is a tendency for men having a second wound to be deteriorated more frequently than men with but a single head wound. This difference, although it seems natural enough, is not statistically significant.

Men who were unconscious more than 2 hours are more frequently deteriorated than men never in coma or who were so for less than 2 hours. Moreover, there seems to be a definite shift in the proportions of low and high IQ's between men who had long unconsciousness and those who had little or none. However, within the group of men in coma for more than 2 hours, duration of unconsciousness seems not to affect the scores on the Wechsler-Bellevue (table 118).

Table 116.—*Correlation of Intelligence and Type of Wound*

Type of wound	Number tested	Wechsler-Bellevue IQ					Abnormally deteriorated	
		Number			Percent			
		≤89	90–119	≥120	≤89	≥120	Number	Percent
Total	683	88	470	125	13	18	145	21
Perforating wounds	22	7	12	3	32	14	10	45
Penetrating wounds	419	41	294	84	10	20	93	22
Wounds with no overt evidence of brain damage	246	42	166	38	17	15	43	17

Table 117.—*Relation of Intelligence to Depth of Wound*

Depth of wound	Number tested	Wechsler-Bellevue IQ					Abnormally deteriorated	
		Number			Percent			
		≤89	90–119	≥120	≤89	≥120	Number	Percent
Total	683	88	470	125	13	18	145	21
Scalp	185	29	127	29	16	16	30	16
Cranial	130	18	81	31	14	24	21	16
Dural	19	3	10	6	16	32	4	21
Brain	314	29	233	52	9	17	80	25
Ventricle	25	8	13	4	32	16	9	36
Unknown	10	1	6	3	10	30	1	10

It is clear that "hemiplegia or hemiparesis" or "aphasia" at the time of the wound is of considerable prognostic significance both for deterioration and for the general IQ. However, hemianopsia seems not to have the same influence. Men with combinations of deficits of different kinds do worse on the Wechsler-Bellevue than men with single deficits, and they also have a higher probability of exhibiting abnormal deterioration (table 119).

There seems little doubt that men with intracranial foreign bodies at the time of wounding are more frequently deteriorated than men without, but the significance of the type of foreign body and its removal on the intelligence is less clear (table 120).

The only neurological symptom at discharge which was significantly correlated with the Wechsler-Bellevue score was "Impaired Memory":

Table 118.—*Relation of Intelligence to Period of Unconsciousness*

Unconsciousness	Number tested	Wechsler-Bellevue IQ					Abnormally deteriorated	
		Number			Percent		Number	Percent
		≤89	90–119	≥120	≤89	≥120		
Total	683	88	470	125	13	18	145	21
None	141	13	94	34	9	24	21	15
≤2 hours	151	15	104	32	10	21	23	15
>2–24 hours	52	10	33	9	19	17	16	31
>1–3 days	40	13	22	5	33	13	10	25
>3–7 days	35	4	29	2	11	6	9	26
>1 week	15	7	7	1	47	7	8	53
Other and unknown	249	26	181	42	10	17	58	23

More than half (57 percent) of the 23 men so coded were abnormally deteriorated as opposed to 23 percent for men with no symptoms. Moreover, not a single one of the 23 men with this symptom had a general IQ of 120 or more, although 18 percent of men generally were so scored.

Many neurological abnormalities at discharge are associated with a significantly increased proportion of men who are abnormally deteriorated at follow-up: Hemiplegia or hemiparesis, aphasia, and sensory disturbance are all marked by a highly significant elevation of the proportion abnormally

Table 119.—*Relation of Intelligence to Neurological Deficit at Time of Wounding*

Neurological deficit	Number tested	Wechsler-Bellevue IQ					Abnormally deteriorated	
		Number			Percent			
		≤89	90–119	≥120	≤89	≥120	Number	Percent
Total	683	88	470	125	13	18	145	21
None	385	41	262	82	11	21	61	16
Hemiplegia or hemiparesis	83	13	60	10	16	12	24	29
Hemianopsia	35	3	26	6	9	17	3	9
Aphasia	40	7	28	5	18	13	12	30
Combination of 2 or more	57	12	38	7	21	12	21	37
Other and unknown	116	17	81	18	15	16	36	31

deteriorated. Spasticity and hemianopsia are characterized by elevations which are significant, although not highly so. Men with personality changes exhibit an elevated proportion with deterioration, but the number of cases is not large enough to achieve significance (table 121).

Table 120.—*Relation of Intelligence to Intracranial Foreign Bodies at Time of First Debridement*

Intracranial foreign bodies	Number tested	Wechsler-Bellevue IQ					Abnormally deteriorated	
		Number			Percent			
		≤89	90–119	≥120	≤89	≥120	Number	Percent
Total	683	88	470	125	13	18	145	21
No intracranial foreign bodies	173	28	119	26	16	15	23	13
Foreign bodies, all removed	179	17	122	40	9	22	39	22
Retained bone fragments	68	10	44	14	15	21	17	25
Retained metal fragments	74	8	51	15	11	20	12	16
Retained bone and metal fragments	57	7	45	5	12	9	24	42
Other and unknown	132	18	89	25	14	19	30	23

Table 121.—*Relation of Intelligence to Neurological Abnormalities at Discharge*

Abnormality	Number tested	Wechsler-Bellevue IQ					Abnormally deteriorated	
		Number			Percent			
		≤89	90–119	≥120	≤89	≥120	Number	Percent
Total	683	88	470	125	13	18	145	21
None	299	24	200	75	8	25	48	16
Hemiplegia or hemiparesis	101	21	69	11	21	11	38	38
Paraparesis	5	.....	3	2	.....	40	1	20
Aphasia	60	15	40	5	25	8	28	47
Spasticity	30	5	20	5	17	17	11	37
Hemianopsia or other field defect	48	7	35	6	15	13	15	31
Sensory disturbances	37	11	22	4	30	11	15	41
Personality changes	22	4	14	4	18	18	7	32
Visual disturbances	17	5	11	1	29	6	3	18
Other and unknown	235	38	167	30	16	13	52	22

On the other hand, certain factors of wounding or repair appear to have no relationship to either the IQ or deterioration. The region wounded, complications of the wound, or time of occurrence of cranioplasty have no apparent relationship to the IQ or the degree of deterioration.

The intelligence as determined by the Wechsler-Bellevue test seems to have similar relationships to the findings at follow-up as to those at the time of wounding. The intelligence *per se* in the groups is little affected, but the deterioration bears a significant relationship to factors indicating neurological dysfunction.

The complaints referable to the nervous system at the time of follow-up are more numerous than at discharge. The significant variations in the fractions of men deteriorated are the elevations for those with impaired mentation or memory and with posttraumatic syndrome. The fact that the fraction of asymptomatic men who were deteriorated is lower than the corresponding fraction of men with headache, dizziness, irritability, or nervousness is suggestive, but the differences are not significant (table 122).

Almost every neurological abnormality is marked by a corresponding elevation of the fraction with deterioration, sometimes of large degree. The only statistically nonsignificant elevations are those for men with cranial nerve palsy and cerebellar syndrome. It seems anomalous that mental impairment should have been coded in 4 patients having an IQ of 120 or more. All neurological abnormalities (except the small group with cerebellar syndrome) were accompanied by significantly high proportions of men with quite low IQ, that is, under 90 (table 123).

Table 122.—*Relation of Intelligence to Neurological Symptomatology at Follow-up*

Symptom	Number tested	Wechsler-Bellevue						
		IQ					Abnormally deteriorated	
		Number			Percent			
		≤89	90–119	≥120	≤89	≥120	Number	Percent
Total	683	88	470	125	13	18	145	21
None	58	1	36	21	2	36	4	7
Headache	177	12	117	48	7	27	26	15
Dizziness	45	4	24	17	9	38	7	16
Irritability or nervousness	61	7	38	16	11	26	7	11
Impaired mentation	22	4	15	3	18	14	11	50
Impaired memory	20	3	14	3	15	15	9	45
Easy fatiguability	26	2	17	7	8	27	3	12
Insomnia	9	1	6	2	11	22	2	22
Posttraumatic syndrome	389	66	278	45	17	12	102	26
Unknown	1	.....	1	.....	.....	.....	.....	.....

Table 123.—*Relation of Intelligence to Neurological Abnormalities*

Abnormality	Number tested	Wechsler-Bellevue IQ Number ≤89	90–119	≥120	Percent ≤89	≥120	Abnormally deteriorated Number	Percent
Total	683	88	470	125	13	18	145	21
Normal	320	25	218	77	8	24	47	15
Hemiparesis or hemiplegia	144	32	95	17	22	12	47	33
Hemianopsia	61	14	40	7	23	11	20	33
Cortical sensory impairment	142	29	98	15	20	11	48	34
Aphasia	51	16	30	5	31	10	21	41
Mental impairment	93	31	58	4	33	4	51	55
Cranial nerve palsy	161	25	116	20	16	12	34	21
Cerebellar syndrome	6	1	5	.....	17	.....	1	17
Other and unknown	13	2	10	1	15	8	6	46

2. Minnesota Multiphasic Personality Inventory

Unlike the Wechsler-Bellevue test, the personality inventory seems little related to the degree of organic neurological deficit (table 124). Perhaps this was to be expected since the two examinations measure quite distinct aspects of an individual's mental functions. The personality inventory, unaffected by the factors related to severity of wounding—diameter of defect, depth of wound, neurological deficit, presence of epilepsy, electroencephalographic abnormality—does, however, correlate with certain factors, which will be presented in detail. By examination of the first row of table 124, the proportion of elevated scores can be seen for each scale in the whole group with valid tests (485 men). Substantial proportions (about one-third) were elevated above the norms on Hs, D, and Hy; Pt and Sc were elevated for about one-sixth of the men, while small fractions (at most 6.6 percent) were elevated on the remaining form scales.

There appears to be a moderate correlation with the type of wound (table 125). It seems noteworthy that the Hs scale is elevated for men with scalp lacerations and closed wounds and for men with fractures without brain penetration as compared with men having penetrating wounds. This may be related to the fact that, of scalp lacerations and closed wounds, a disproportionate number came from compensation rosters. On the other hand, although their differences test as statistically significant, this may not be clinically significant for there are a great many other possible comparisons in this table, none of which seems remarkable. It must be remembered that a certain fraction of comparisons even in homogeneous data can be expected to turn out to be significant by chance.

Table 124.—*Relation of Personality Inventory and Neurological Deficit at Time of Wounding*

Deficit	Number of men	Minnesota Multiphasic Personality Inventory								
		Hs	D	Hy	Pd	Mf	Pa	Pt	Sc	Ma
		Percent with elevated scores								
Total	485	36.9	28.7	31.3	6.6	2.5	1.9	15.5	16.5	4.9
No deficit	289	39.4	24.2	30.8	6.6	2.4	2.1	14.9	12.8	4.2
Hemiplegia or hemiparesis	77	33.8	36.4	32.5	9.1	3.9	1.3	20.8	24.7	5.2
Hemianopsia	32	21.9	28.1	18.8	6.3	....	....	3.1	6.3	9.4
Aphasia	45	35.6	37.8	31.1	4.4	....	....	22.2	26.7	6.7
Other and unknown	42	38.1	35.7	42.9	4.8	4.8	4.8	11.9	23.8	4.8

Table 125.—*Relation of Personality Inventory and Type of Wound*

Type	Number of men	Minnesota Multiphasic Personality Inventory								
		Hs	D	Hy	Pd	Mf	Pa	Pt	Sc	Ma
		Percent with elevated scores								
Total	485	36.9	28.7	31.3	6.6	2.5	1.9	15.5	16.5	4.9
Closed head wound without fracture, scalp laceration	130	43.1	26.2	36.2	6.9	.8	3.1	15.4	13.8	3.1
Fractures, not penetrating	39	53.8	33.3	38.5	7.7	5.1	2.6	17.9	25.6	10.3
Penetrating wounds	302	31.5	28.8	28.1	6.0	2.6	1.3	15.2	15.6	5.0
Perforating wounds	14	50.0	35.7	35.7	14.3	7.1	....	14.3	35.7	7.1

The period of unconsciousness seems to bear some relationship to the MMPI (table 126). The Hs, D, Hy, and Sc scales are elevated more frequently for men unconscious 2 hours or more than for men not unconscious or only briefly so (under 2 hours).

Social maladjustment bears a relationship to certain elevations in the personality inventory. Those individuals classified as depressed, restless, or unhappy have generally high scores in all categories of the personality inventory. Similarly, the men who consider themselves social misfits have elevated scores (table 127). Perhaps as a corollary of this, those who have not advanced or have regressed in their work have higher scores in most factors than those men who advanced.

In the follow-up examinations organic abnormalities such as cranial nerve impairment, motor, sensory, or reflex abnormalities had little or no influence upon the elevations in the personality scores, whereas subjective neurological

Table 126.—*Relation of Personality to Period of Unconsciousness*

Unconsciousness	Number of men	Minnesota Multiphasic Personality Inventory								
		Hs	D	Hy	Pd	Mf	Pa	Pt	Sc	Ma
		Percent with elevated scores								
Total	485	36. 9	28. 7	31. 3	6. 6	2. 5	1. 9	15. 5	16. 5	4. 9
None	116	31. 9	25. 9	23. 3	7. 8	3. 4	1. 7	19. 0	12. 9	1. 7
≤2 hours	124	37. 1	29. 0	29. 8	8. 1	3. 2	1. 6	14. 5	14. 5	4. 0
>2 hours	88	48. 9	43. 2	43. 2	8. 0	3. 4	2. 3	22. 7	27. 3	8. 0
Other and unknown	157	33. 8	22. 3	31. 8	3. 8	0. 6	1. 9	9. 6	14. 6	6. 4

Table 127.—*Relation of Personality to Home, Social, and Work Adjustment*

Adjustment	Number of men	Minnesota Multiphasic Personality Inventory								
		Hs	D	Hy	Pd	Mf	Pa	Pt	Sc	Ma
		Percent with elevated scores								
Total	485	36. 9	28. 7	31. 3	6. 6	2. 5	1. 9	15. 5	16. 5	4. 9
Unsatisfactory home adjustment (depressed, restless, unhappy)	37	51. 4	56. 8	43. 2	32. 4	8. 1	5. 4	32. 4	56. 8	16. 2
Unsatisfactory social adjustment (feels out of place, asocial, antisocial)	80	50. 0	43. 8	43. 8	12. 5	6. 3	5. 0	28. 8	33. 8	7. 5
Unsatisfactory work progress, (no progress, regression)	189	47. 6	38. 6	36. 5	9. 5	1. 6	2. 6	20. 6	24. 3	6. 3

complaints were associated with high scores generally (table 128); only in the Mf scale did this not hold.

It is interesting to note that men with low intelligence as measured by the Wechsler-Bellevue tests tended to have elevations of most scales of the personality inventory. On the contrary, those with supranormal intelligence had few elevated scores. Along the same line it should be noted that intellectual deterioration was associated with elevation of the Hs, D, Hy, and Sc scales of the personality inventory—precisely those elevated in men with prolonged unconsciousness (table 129).

It seems on the basis of this study that the deterioration indicated by the Wechsler-Bellevue test is particularly related to the severity of organic brain damage, although it is also associated with high scores on most of the scales

Table 128.—*Relation of Personality to Clinical Symptomatology at Follow-up*

Symptom	Number of men	Minnesota Multiphasic Personality Inventory								
		Hs	D	Hy	Pd	Mf	Pa	Pt	Sc	Ma
		Percent with elevated scores								
Total	485	36.9	28.7	31.3	6.6	2.5	1.9	15.5	16.5	4.9
No symptoms	48	6.2	8.3	10.4	4.2	6.2	....	2.1	4.2	2.1
Headaches	149	26.8	14.1	24.2	5.4	2.7	1.3	9.4	9.4	.7
Dizziness	33	33.3	21.2	30.3	3.0	3.0	3.0	18.2	12.1	3.0
Irritability or nervousness	51	23.5	13.7	17.6	3.9	2.0	....	3.9	7.8	2.0
Impaired mentation or memory	31	32.3	19.4	12.9	9.7	3.2	....	12.9	22.6	6.5
Easy fatiguability, insomnia	30	30.0	23.3	30.0	3.3	3.3	....	6.7	16.7	6.7
Posttraumatic syndrome	244	51.6	43.4	43.0	7.8	1.6	2.9	23.4	23.4	7.8

Table 129.—*Relation of Personality Inventory to Intelligence*

Wechsler-Bellevue test	Number of men	Minnesota Multiphasic Personality Inventory								
		Hs	D	Hy	Pd	Mf	Pa	Pt	Sc	Ma
		Percent with elevated scores								
Total	485	36.9	28.7	31.3	6.6	2.5	1.9	15.5	16.5	4.9
Intelligence scale:										
≤89	35	60.0	48.6	42.9	8.6	....	2.9	31.4	31.4	5.7
90–119	323	37.5	30.3	32.5	7.4	2.2	2.2	14.6	16.1	5.6
≥120	112	29.5	19.6	26.8	3.6	3.6	.9	15.2	14.3	3.6
Abnormal deterioration for age	75	53.3	37.3	44.0	8.0	2.7	1.3	18.7	21.3	5.3

of the personality inventory. The Minnesota Multiphasic Personality Inventory, on the other hand, is only slightly affected by organic brain damage, but is significantly elevated in almost all scales by aspects of neurological affections generally considered to be "functional." Although these differences acquire statistical significance, it must be admitted that, in an isolated case, the criteria may not be entirely reliable.

3. The Goddard Form Board Test

To investigate sensory motor performance in the absence of exteroceptive clues, the Goddard Form Board was used in those patients having sufficient motor and sensory abilities to carry out the test. Only a few correlations

will be examined. The presence of neurological symptoms is related to this performance task. The test does not seem to be very specific, but in all symptomatic categories the performance is impaired as compared to the groups having no clinical symptoms (table 130). The memory score in this analysis would seem to be somewhat more sensitive than the other components of the test although the differences are not impressive. A neurological deficit is correlated with impaired scores in this examination. The performance test is definitely deficient in those patients having motor,

Table 130.—*Goddard Test in Relation to Clinical Symptomatology*

Symptom	Total number of men tested	Total time ≥16 minutes (percent)	Dominant hand ≥6 minutes (percent)	Recessive hand ≥6 minutes (percent)	Boths hands ≥6 minutes (percent)	Memory score ≤4 items (percent)	Location score ≤4 items (percent)
Total	576	29.5	44.1	29.3	7.5	17.0	70.3
None	48	20.8	[1] 27.1	16.7	4.2	6.2	[2] 52.1
Posttraumatic syndrome	329	31.6	[1] 47.4	32.2	9.4	19.8	[2] 76.6
Other symptoms or unknown	199	28.1	[1] 42.7	27.6	5.0	15.1	[2] 64.3

[1] Variation is statistically significant at the P ≤.05 level.
[2] Variation is statistically significant at the P ≤.01 level.

Table 131.—*Goddard Test in Relation to Summary of Neurological Abnormalities*

Abnormality	Total number of men tested	Total time ≥16 minutes (percent)	Dominant hand ≥6 minutes (percent)	Recessive hand ≥6 minutes (percent)	Boths hands ≥6 minutes (percent)	Memory score ≤4 items (percent)	Location score ≤4 items (percent)
Total	576	29.5	44.1	29.3	7.5	17.0	70.3
None	302	24.8	39.1	24.8	6.0	15.2	67.2
Hemiparesis or hemiplegia	68	[2] 50.0	[1] 52.9	[2] 50.0	[1] 14.7	23.5	77.9
Hemianopsia	42	31.0	52.4	31.0	7.1	23.8	71.4
Cortical sensory impairment	71	[1] 40.8	50.7	[1] 40.8	8.5	23.9	66.2
Aphasia	23	[2] 56.5	[1] 65.2	52.2	[1] 21.7	30.4	82.6
Mental impairment	61	[2] 55.7	[2] 68.9	[2] 49.2	[1] 14.8	24.6	[1] 82.0
Cranial nerve palsy	130	25.4	42.3	26.2	10.0	16.9	72.3
Cerebellar syndrome	5	20.0	40.0	20.0	20.0	40.0	80.0
Other and unknown	9	55.6	77.8	55.6	11.1	22.2	77.8

[1] Percentages differ significantly from the percent in normal men at the P≤.05 level.
[2] Percentages differ significantly from the percent in normal men at the P≤.01 level.

sensory, or mental impairment (table 131). The poor scores in the aphasic group are probably related to the coincident motor or sensory disturbance in most of these patients. Other neurological defects do not seem to influence the performance scores significantly.

Intelligence seems definitely related to the ability to manipulate the form board. In all components of the performance test lower intelligence is associated with significantly impaired scores, being perhaps reflected most sharply in the memory score. As further confirmation of the effect of intelligence on the performance test is the finding that abnormal deterioration is associated in all categories with decreased performance, although statistical significance is reached only in three components (table 132).

In summary, one might conclude that the Goddard Form Board serves as an additional check on general neurological function, and that it seems to fluctuate with other measures of neurological abnormality.

Table 132.—*Correlation of Goddard Test and Intelligence*

Wechsler-Bellevue test	Total number of men tested	Total time ≥16 minutes (percent)	Dominant hand ≥6 minutes (percent)	Recessive hand ≥6 minutes (percent)	Both hands ≥6 minutes (percent)	Memory score ≤4 items (percent)	Location score ≤4 items (percent)
Total	576	29.5	44.1	29.3	7.5	17.0	70.3
Intelligence scale:							
≤89	61	[2] 47.5	[2] 57.4	[2] 47.5	[1] 13.1	[2] 52.5	[2] 90.2
90–119	391	[2] 30.2	[2] 46.8	[2] 30.9	[1] 8.2	[2] 15.9	[2] 74.4
≥120	113	[2] 17.7	[2] 26.5	[2] 14.2	[1] 1.8	[2] 3.5	[2] 43.4
Unknown	11	27.3	54.5	27.3	9.1	.0	90.9
Abnormal deterioration for age	100	[2] 45.0	[2] 63.0	[2] 42.0	8.0	24.0	75.0

[1] Statistically significant at the $P \leq .05$ level.
[2] Statistically significant at the $P \leq .01$ level.

4. Discussion

There has been much discussion of the sensitivity of psychometric tests in differentiating organic from functional alterations of the nervous system. The present study, in line with previous investigations, suggests that, as a group, people with predominant neurotic disturbances react differently in mental tests than people with predominant organic deficits. Individual cases may not always be differentiated. Sands and Price (112) believe that the digit span and symbol test of the Wechsler-Bellevue test is sensitive to organic deficit as contrasted to epileptic and functional groups. Halstead (60) relies upon his category tests for such differentiation. Many psychologists prefer the Rorschach responses as a basis of judgment. However, Lynn et al. (81) believe that a battery of tests is necessary to cover the complete range of potentially deviant cerebral responses in late head injuries.

It is of interest to consider that cerebral function may be impaired both by the elimination of cerebral substance and by the abnormal activity of damaged cortex. Dailey (33) has examined the thesis that continued interference with normal brain function by pathological tissue may have more adverse psychological effects than those produced by the elimination of that tissue. In head-injured patients, psychological testing indicated that the performance was not reduced and might be improved by such excisions.

Chapter V

EPILEPSY

A. INTRODUCTION

The occurrence of paroxysmal alterations in the state of consciousness following a head injury is very common. Even shortly after a blow producing only a momentary loss of consciousness, the victim is likely to feel dizzy, lightheaded, and to black out upon assuming an erect position. These minor lapses are generally considered as due to vasomotor instability producing a temporary cerebral ischemia. Such lightheaded spells or blackouts usually occur only in the first few days after cerebral concussion, but may be present for weeks or months, especially with more severe head injuries. Are they simply to be considered vasomotor phenomena or may they be epileptic? It is difficult to define the border between vasomotor and epileptic episodes. Only time, which usually eliminates the former and elaborates the latter, may allow a differentiation. Clinicians for years have noted this difficulty. In one of the oldest descriptions of posttraumatic sequelae, von Bergmann (131) noted that the incidence of epilepsy as the result of head wounds in the war of 1870–71 was 4.7 percent, but if the dizziness and blackout episodes were considered epilepsy, the frequency was 26.7 percent.

The classification of the epileptic seizures in the present study was not a simple matter. Although most major attacks could usually be recognized by their description and most focal seizures were readily identified, there remained a number of minor episodes which did not fit into any of the usual types. It is noteworthy that typical petit mal was not seen in these patients. In fact, not a single patient presented its usual clinical picture with the spike and dome pattern which characterizes its electroencephalogram. Some authors have referred to posttraumatic petit mal attacks (Elvidge, 18 of 58 cases) without describing their characteristics. If occurring as posttraumatic sequelae, they must be rare. In some 33 cases, episodes which superficially resembled petit mal occurred. On reviewing these cases, 13 patients were noted to have spells of faintness, dizziness, weakness, wobbly feeling, or headache and dizziness, but without true impairment of consciousness. The other 20 had similar episodes, epigastric feelings, staring, or blank spells associated with definite loss of consciousness for as long as a minute so that 10 of them fell down but did not convulse. These latter 20 cases would seem properly classified in the convulsive group, although the type of epilepsy is not clear from the history or from the electroencephalograms, even though 4 had abnormalities in the brain waves.

The epileptic nature of minor attacks which preceded major episodes seems clear. Some 28 patients had minor attacks which sometimes progressed to convulsive seizures. Such minor episodes were described in

the same terms as given above by patients who did not have definite epileptic manifestations at any one time.

These difficulties in establishing the diagnosis of epilepsy following a head wound are insurmountable at the present stage of our knowledge of convulsions. For practical purposes, certain arbitrary standards must be established as a working basis. In this study the cases were classified as shown in table 133.

The 12 cases labelled "borderline attacks only" had attacks of 2 kinds. The first and most common of these types consisted of attacks of dizziness, lightheadedness, faint or dazed feeling without definite loss of consciousness, with few exceptions associated with a normal electroencephalogram. These episodes seem correctly placed in the borderline group falling into Gowers' vasovagal class. The second type consisted of dizzy or dazed feelings associated with a short period of loss of consciousness usually called a "blackout" but not accompanied by a fall. Again, in these cases the electroencephalograms were sometimes normal and sometimes not, but in no case were they typical of petit mal or psychomotor epilepsy. The classification of these latter cases as borderline rather than epileptic might be contested. However, they are so few that their exclusion can introduce no significant error.

B. TYPES OF ATTACKS

Although typical generalized convulsions were the most common type of attack (table 134), many other varieties occurred. Focal, or apparently focal, manifestations were associated with generalized seizures in 39 instances. In 31 patients focal convulsions occurred, never progressing to grand mal, and in 26 cases minor attacks such as described previously were never followed by generalized seizures. It is interesting to note that only 12 patients were considered to have psychomotor seizures, although this type of attack is now thought to be the most common in adult life.

Table 133.—*Occurrence of Epilepsy*

Classification	Number of patients	Classification	Number of patients
No epilepsy	532	Borderline attacks only	12
One definite attack	26		
>1 definite attack	169	Total	739

The focal manifestations ran the gamut of cerebral functions. Thus the aurae were of many types (table 135). In the first place, it is interesting to note that although the epilepsy was presumably due to a focal cerebral injury, more than one-fourth of the patients had no warning of their attacks. This is not a new observation; all writers on the subject have noted that 25 to 69 percent of patients with localized cerebral injuries have major epilepsy without obvious focal onset (83). Apparently, such attacks may occur from wounds of any part of the brain (table 136) but somewhat more commonly of the frontal region.

Table 134.—*Type of Epilepsy*

Type	Number	Percentage	
		All men	Men with fits
Total number of men examined	739	100.0	
Total number with fits	207	28.0	100.0
Minor attacks	26	3.5	12.6
Focal	31	4.2	15.0
Grand mal	72	9.7	34.8
Psychomotor	12	1.6	5.8
Focal and grand mal	39	5.3	18.8
Minor attacks and grand mal	15	2.0	7.2
Other combinations	7	.9	3.4
Type undetermined	5	.7	2.4

The most common aura in this series was a motor phenomenon—jerking, tonic movement, or an impairment of motor ability. This type of aura was noted in 45 patients. The phenomenon might begin in almost any part of the body but most commonly in the face, hand, or leg. Frequently the muscular jerking was associated with a sensory aura in the same part; 17 of the 45 patients with motor aurae also had sensory manifestations. Although motor warnings were more common with parietal and frontal lobe lesions, they also occurred with wounds of other regions. Somatosensory aurae occurred in 40 cases, and were associated with motor phenomena in 17 cases. The frequency of combined sensory and motor aurae is probably only an indication of the closeness of the cortical representation of these two functions.

It is of interest to note that of the five patients with auditory aurae, three had no auditory symptoms, one had tinnitus unlocalized, and one had tinnitus and impaired hearing in the left ear but the aura of ringing in the right ear!

The somatosensory warnings were commonly referred to as numbness, but many other terms were used, such as "pumping sensation," "hot feeling," "smarting," "funny sensation," "sharp pain," "throbbing pressure,"

Table 135.—*Aurae in Men With Epilepsy*

Aura	Number	Percent	Aura	Number	Percent
Total number of men with epilepsy	207	100.0	Somatosensory	40	19.3
			Gastric	10	4.8
			Motor	45	21.7
			Vertiginous	19	9.2
None	58	28.0	Dreamy state	4	1.9
Olfactory	5	2.4	Automatism, etc	3	1.4
Auditory	5	2.4	Other and unknown	39	18.8
Visual	19	9.2			

Table 136.—*Correlation of Aurae With Location of Wound in Men With Epilepsy*

Aura	Location of wound			
	Frontal region, sinuses, and orbit	Parietal region	Temporal region	Occipital region
Total number of men with epilepsy	86	114	41	35
None	38. 4	25. 4	24. 4	28. 6
Olfactory or gustatory	3. 5	. 9	7. 3	.....
Auditory	4. 7	2. 6	7. 3	2. 9
Visual	7. 0	11. 4	4. 9	20 0
Sensory	20. 0	27. 2	29. 3	20. 0
Gastric	5. 8	4. 4	7. 3	5. 7
Motor	25. 6	32. 5	17. 0	25. 7
Vertiginous	8. 1	10. 5	4. 9	8. 6
Dreamy state	3. 5	3. 5	4. 9	2. 9
Automatism, etc	.....	1. 8	.....	2. 9
Other and unknown	5. 8	7. 9	17. 0	11. 4

"burning," "sticking feeling," etc. As with the motor phenomena, they were usually referred to the arm, hand, face, entire side, or least commonly the leg. Besides being associated with motor phenomena, visceral, visual, vertiginous, or olfactory sensations were accompaniments.

The visual aurae sometimes took the form of negative (blurred or dark spots) or positive (photic hallucinations) scotomata, usually in the field contralateral to the injury.

In this series vertiginous aurae were common. Generally, they were described as a lightheaded or dizzy feeling, although two patients spoke of their warning as spinning from one side to the other, which would suggest a true vertigo. It is interesting that more than half of the patients with vertiginous aurae had other phenomena at the beginning of the attack, usually of a sensory or motor nature but occasionally visual or autonomic.

In three patients, automatisms ushered in their attacks, all of which were associated with other phenomena such as a dazed feeling, scintillating scotomata, or paresthesias. A dreamy state at the beginning of the attack was noted by three patients, associated in two cases with vertigo and a gastric sensation. Epigastric feelings occurred in some 10 patients, being described in various terms, such as "butterfly feeling in the stomach," a peculiar sensation which would rise from the epigastrium, a desire to void, etc. These aurae were often associated with other sensory manifestations.

Olfactory aurae occurred in five cases, in three of which the smells were said to be unpleasant, in two cases that of burning material, and in one case that of gasoline.

Various other aurae were experienced by some 16 patients; a feeling of weakness, a sudden headache, dazed feeling, saliva accumulating in the mouth, a funny feeling, a tightening up, wobbly feeling, lightheadedness, unsteadiness.

The postictal phenomena are not particularly enlightening. Most of the patients had such nonspecific phenomena as headache or confusion; only 19 had a Todd's paralysis (table 137). It is interesting and not unexpected that the majority of these 19 had motor aurae.

C. INCIDENCE OF EPILEPSY

The overall incidence of convulsive episodes in the 739 men is 28 percent, but only 22.9 percent of the men had more than 1 definite attack. As is shown in table 138, the incidence of epilepsy in 267 men without dural penetration is 14.2 percent, and in 472 men with dural penetration, 35.8 percent. If only multiple attacks be considered, the figures are 9.7 percent and 30.3 percent, respectively.

These figures are not out of line with those presented by most other authors regarding war-wounded men (table 139). Discrepancies may well be accounted for by the relatively low percentage of follow-up in most series and by the selection of cases.

Table 137.—*Postictal Phenomena in Men With Epilepsy*

Postictal phenomena	Number	Percent	Postictal phenomena	Number	Percent
Total number of men with epilepsy	207	100.0	Confusion	87	42.0
			Todd's paralysis	19	9.2
			Headache	67	32.4
			Other states	12	5.8
None	66	31.9	Other and unknown	22	10.6

Table 138.—*Incidence of Posttraumatic Epilepsy in Relation to Type of Wound*

Type of wound	Number of men		With 1 or more attacks		With multiple attacks			
					Any type		Focal	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Total	739	100.0	207	28.0	169	22.9	65	8.8
No fracture	207	100.0	30	14.5	20	9.7	1	.5
Fracture without dural penetration	60	100.0	8	13.3	6	10.0	3	5.0
Total without dural penetration	267	100.0	38	14.2	26	9.7	4	1.5
Dural:								
Penetrating	448	100.0	157	35.0	132	29.5	58	12.9
Perforating	24	100.0	12	50.0	11	45.8	3	12.5
Total with dural penetration	472	100.0	169	35.8	143	30.3	61	12.9

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Table 139.—*Incidence of Posttraumatic Epilepsy*

Authors	Source and date	Number of cases (percent followed-up)	Follow-up		Epilepsy percentage			
			Length (years)	Technique	Closed	Scalp and cranium	Dural penetration	Mixed
von Bergmann (131)	Civil War pension lists, 1861–65.	98	?	R				9
		69	?	R			20	
Eguchi (41)	Franco-Prussian pension lists, 1870–71.	8, 985 (? percent)	?	R	0. 75			
		517 (? percent)	?	R				4. 3
Allen et al. (5)	Franco-Prussian	571						4. 3
Eguchi (41)	Russo-Japanese, 1904–05	1, 245 (? percent)	?	R				3. 0
Holbeck (66)	Russo-Japanese, 1904–05	65	2	?			29	
Alajouanine et al. (4)	French Army, 1914–18	602 (? percent)	12–14	?		14. 7	37	23
Gamberini (50)	1914–18	138 (51 percent)	3–5	Examination			61. 9	
Wagstaffe (132)	British Army, 1914–18	820 (71 percent)	11	L				
		17			0			
		180				2		
		176					18. 7	
Rawling (100)	British Army, 1914–18	750 (60 percent)	2	L				
		47			9			
		119				13		
		226					33. 2	
Sargent (113)	British Army, 1914–18	18, 000	?	R				4. 5
Stevenson (119)	British Army, 1914–18	17, 300	?	R				1. 5
Ascroft (8)	British pensioners, 1914–18	540 (60 percent)	4+	R				
		170				23		
		129					45	

Béhague (15)		3, 623	?					12. 1
Vogeler (130)	German, 1914–18	500 (80 percent)	10+	Personal				26. 5
Credner (32)	1914–18	1, 990	10±					38. 9
		1, 234					49. 5	
		417				20. 3		
		244			19. 7			
Baumm (10)	1914–18	955						22. 2
Steinthal and Nagel (118)	1914–18	639	10±	?				28. 9
								35. 5
		348					39. 8	
		169				25		
Gliddon (54)	Canadian pensioners, 1914–18.	500	18	R				
		363				5. 5		
		137					18. 9	
Russell and Whitty (111)	World War II	500 (? percent)	? 2–3	?	6			
Penfield and Shaver (96).	Civilian patients	1, 612 (25 percent)	2–14	L	(*)			2. 7
Rowbotham (105)	Civilian	1, 000 (43 percent)	5+	L	2. 5			
Elvidge (42)	Civilian	362	Short					1. 93
Watson (136)	World War II, Personal	500 (57 percent)	3	L	(**)		41. 6	
Walker and Jablon (135).	World War II, Army and veteran rosters.	932 (80 percent)	7–8	Interviews and examinations.	14. 5	13. 3	35. 8	28
Wilson (139)	New Zealand World Warr II	196	6	?			17. 3	
Faust (45)	World War I, civilian (selected)	80	3–35				44	Frontal lobe involvement.
Phillips (98)	Closed head injury	500 (? percent)	?	?	6. 2			
Dubitscher (38)	World Wars I and II	1, 000	?				33. 9	

R = Examination of records.
L = From letter.
* =Recurrent fits.
** = Any after first 4 weeks.

Table 140.—*Type of Epilepsy in Relation to Type of Wound*

Type of wound	Number with epilepsy	Minor attacks only		Major attacks	
		Number	Percent	Number	Percent
Total	207	67	32.4	140	67.6
Without dural penetration	38	15	39.5	23	60.5
No fracture	30	12	40.0	18	60.0
Fracture without dural penetration	8	3	37.5	5	62.5
With dural penetration	169	52	30.8	117	69.2
Penetrating	157	52	33.1	105	66.9
Perforating	12	.....	.....	12	100.0

There is no doubt that epilepsy, irrespective of how it is defined, is much more common when the dura mater is violated, although the type of attack appears to be the same whether or not the cortex is overtly damaged. Perforating wounds, however, seem to favor major attacks (table 140).

D. TIME OF FIRST ATTACK

In table 141 the time of the first attack of any kind is given. It is obvious that within 9 months about half of the patients who will develop epilepsy within the next 8 years have already had their first attack, and 75 percent have had their initial attack within the first 2 years. After the second year, for the next 5 to 10 years the chances of developing an epilepsy are slightly more than 1 percent per year. This is in agreement with other reports (table 142). It is of interest to note that only 17 patients (8.3 percent) had seizures (about equally divided between major and minor) in the first week after their injury (table 143). It is also plain from table 143 that whether a man would have minor or grand mal seizures was quite unrelated to the time of the first attack.

Some authors do not consider early attacks as indicative of epilepsy, but what constitutes an early attack is not clearly defined. Penfield and Shaver (96) presumably consider 11 days within this period (their case 6 was considered to have early seizures at least 11 days after injury). Denny-Brown (35) states that attacks beginning in the first month respond well to treatment and tend to disappear.

Braun (20) does not give a precise time but refers to von Bergmann's concept that early epilepsies are due to local cortical wounding, so that one may infer he is thinking in terms of hours, or at the most days—not weeks or months. Specifically, he states that they occur most frequently 2 to 3 days or 8 days after trauma, seldom later. Tönnis (125) believes that the highest incidence corresponds to the phase of bleeding and edema about the seventh day; he states that the favorable prognosis of these early epilepsies has yet to be confirmed.

Goldstein (55) notes that 50 percent of all patients who had attacks in the first week did not acquire permanent epilepsy, while the prognosis for patients having attacks 2 to 3 years after injury was bad.

Table 141.—*Time of First Attack for Men Who Ever Had Epilepsy*

Time interval from injury	Number of men having first fit in this interval	Annual rate [1] of development of fits per 100 not previously attacked	Accumulated [1] percent of examined men
Less than 1 week	17	70.2	2.3
1 week to <3 months	30	16.8	6.4
3 to <6 months	33	17.8	10.8
6 to <9 months	30	17.0	14.9
9 to <12 months	16	9.8	17.0
12 to <15 months	16	10.0	19.2
15 to <18 months	9	5.9	20.4
18 to <21 months	4	2.7	21.0
21 to <24 months	3	2.0	21.4
2 to <3 years	17	2.9	23.7
3 to <4 years	10	1.8	25.0
4 to <5 years	5	.9	25.7
5 to <6 years	6	1.2	26.6
6 to <7 years	5	1.2	27.5
7 to <8 years	4	1.7	28.8
Unknown	2	.....	.....

[1] The rates are based on the 205 men for whom the time of first attack could be determined.

Table 142.—*Proportion of Men Having Onset of Posttraumatic Epilepsy in Specified Time Intervals From Injury*

Authors	Time interval from injury						
	0–1 month	1.1–6 months	6.1–12 months	Second year	Third year	Fourth year	Fifth year
	(Percent)						
Ascroft (8)	6	10	8	5	2	2	0.5
Baumm (10)	.....	.....	[1] 11	5	2	1.5	1.5
Gliddon (54)	2	.....	[2] 2.5	1.5	.4	.8	.1–2.5
Russell and Whitty (111)	5	14	11	9	1	1	1
Walker and Jablon (135)	.....	[3] 10.8	6.2	4.4	1.3	.7	.9

[1] First year.
[2] 1.1 to 12 months.
[3] 0–6 months.

Russell (109) states that a focal or generalized fit in the first 2 to 3 weeks is not significant, nor are such patients more likely to develop epilepsy after recovery. Symonds (122) notes that the occurrence of fits in the early stages (within 48 hours) does not mean they will recur later, but Wagstaffe (132) believes that the likelihood is greater than in uncomplicated head injuries. Elvidge (42) noted 1.93 percent of "immediate" seizures (up to 24 hours of injury) in a series of 362 personally treated cases. He further noted an incidence of 6.9 percent of "immediate" and 13.9 percent of early (up to 4 weeks) epilepsy in a series of 43 cases of late posttraumatic epilepsy.

Table 143.—*Time of First Attack in Relation to Type of Epilepsy*

Time interval from injury	Men having first fit in this interval				
	Total	Major and/or minor attacks		Minor attacks only [1]	
		Number	Cumulative percent	Number	Cumulative percent
Total	207	140	.....	67	.....
<1 week	17	10	7.1	7	10.8
1 week to <3 months	30	20	21.4	10	26.2
3 to <6 months	33	24	38.6	9	40.0
6 to <9 months	30	23	55.0	7	50.8
9 to <12 months	16	9	61.4	7	61.5
12 to <15 months	16	11	69.3	5	69.2
15 to <18 months	9	6	73.6	3	73.8
18 to <21 months	4	2	75.0	2	76.9
21 to <24 months	3	2	76.4	1	78.5
2 to <3 years	17	13	85.7	4	84.6
3 to <4 years	10	7	90.7	3	89.2
4 to <5 years	5	4	93.6	1	90.8
5 to <6 years	6	5	97.1	1	92.3
6 to <7 years	5	2	98.6	3	96.9
7 to <8 years	4	2	100.0	2	100.0
Unknown [2]	2	.....	.....	2	.....

[1] The percentages are based on the 65 men for whom the time of first attack was known.
[2] One man had attacks within the first 3 months, but it could not be determined whether they did or did not originate in the first week; one man had his first attack some time during the second year after injury.

The previous literature regarding the time of first attack is somewhat confused by reason of the varied concepts of early epilepsy. These convulsions, generally agreed to be more common with closed than open head injuries and present with basal skull fractures rather than convexity wounds, seem to occur in about 5 percent of cases (74). The majority of such patients are said to have no further attacks, but if this is not the case, between these attacks in the first days and those said to be more significant of a chronic recurring epilepsy, an interval of several months or years may elapse. Redlich (101) believed that wounds of the central region were associated with attacks later than those in other regions, but Tilmann (124) concluded the reverse.

From several sources, data is available to indicate that about 25 to 33 percent of patients who ever will develop posttraumatic epilepsy will do so 2 years or more after injury. If one assumes an incidence of 35 percent for posttraumatic epilepsy in open head wounds (table 139), 9 to 12 percent of the exposed population will develop fits 2 years or more after a blow to the head. From the 2d to the 10th year the frequency of new cases would seem to be 1 percent per year, or a total of 8 percent. This would leave 1 percent to 4 percent of the population to have attacks beginning later than 10 years.

E. ANTECEDENT FACTORS IN EPILEPSY

Because all patients receiving a cerebral wound of a given intensity in a given region do not develop convulsive seizures, the suggestion has frequently been made that some predisposing factor, personal or genetic, might be responsible for seizures in those so afflicted. Factors peculiar to the individual might be related to physical stresses applied to the head at birth or during development.

Within the relatively narrow limits of the age range in the present series, the age of the patient at the time of injury does not seem to be related to the probability of epilepsy (table 144).

Although the birth histories are probably unreliable, in the small group which admit abnormal deliveries there is no unusual incidence of epilepsy (table 144).

Table 144.—*Epilepsy in Relation to Age at Injury, Birth History, and Birth Order*

Characteristic	Number of men	With epilepsy		With multiple focal attacks	
		Number	Percent	Number	Percent
Total	739	207	28.0	65	8.8
Age at injury:					
18–20 years	133	34	25.6	11	8.3
21–23 years	193	58	30.1	12	6.2
24–26 years	162	54	33.3	16	9.9
27–29 years	109	25	22.9	5	4.6
30–32 years	73	24	32.9	6	8.2
≥33 years	69	12	17.4	5	7.2
Birth history:					
Norm l	701	199	28.4	63	9.0
Other and unknown	38	8	21.1	2	5.3
Birth order:					
First born	186	53	28.5	14	7.5
Second born	182	54	29.7	14	7.7
Third born	145	41	28.3	19	13.1
Fourth or over	216	56	25.9	16	7.4
Unknown	10	3	30.0	2	20.0

Since the first labor is usually more difficult and longer than subsequent ones, the baby of a primipara might be subject to more cerebral anoxia and molding of the head than those of multiparas. For that reason it has been suggested that the first-born is more susceptible to seizures than later children. Nielsen and Butler (92) concluded from a survey of an epileptic colony that 40 percent of all cases of epilepsy occurred in first children, and that the first-born was twice as susceptible to epilepsy as children born subsequently to the same mother. Alstrom (6) came to a somewhat similar conclusion. However, Orr and Risch (93), in reviewing the subject, point out that these authors included cases of "the only child" and that if such were excluded from the series, the incidence of epilepsy was not any higher in the first than in the second, third, fourth, or subsequent children. In

Alstrom's series, if the individuals who were "only children" are excluded, the incidence of epilepsy is 22.9 percent in the first-born as opposed to 21.0 percent in the last-born. Moreover, Orr and Risch (93) in a series of 158 veterans found that epilepsy seemed to occur more frequently in the last-born of a series of children than in the first-born. In the present series, the number of the patient in birth order has no relationship to the development of the epilepsy (table 144).

The rate of physical and mental development is said to be normal in all but four men, all of whom fall in the epileptic groups. That this is significant is doubtful. Certainly it could not be considered a very important factor in the entire epileptic group since it has a relative frequency of only 2 percent. Previous systemic illnesses seem unrelated to the occurrence of epilepsy, and head injuries in early life before wounding are equally well represented in the epileptic and nonepileptic men (table 145). The incidence of 10.6 percent of previous head injuries agrees well with Pennington and Mearin's (97) figure of 13 percent in male naval inductees of World War II. There is clearly no correlation between antecedent factors in the personal history and the occurrence of epilepsy after a head wound.

Table 145.—*Relation of Epilepsy to Previous Illnesses and Head Injuries*

History	Number of men	With epilepsy		With multiple focal attacks	
		Number	Percent	Number	Percent
Total	739	207	28.0	65	8.8
Previous illnesses:					
Uncomplicated childhood diseases	635	188	29.6	59	9.3
Complicated childhood diseases	18	3	16.7	1	5.6
Allergic conditions	44	8	18.2	2	4.5
Enuresis	28	9	32.1	3	10.7
Temper tantrum, breath holding	4	3	75.0	.....	.....
Syphilis, other diseases or unknown	18	3	16.7	1	5.6
Previous head injuries:					
None significant	657	186	28.3	58	8.8
Knocked out once or twice	61	14	23.0	3	4.9
Knocked out more than twice	11	5	45.5	2	18.2
Severe head injury	7	2	28.6	1	14.3
Unknown	4	1	25.0	1	25.0

A genetic factor might be indicated by an abnormally high incidence of nervous or mental disorders in relatives of the posttraumatic epileptic. An analysis of the frequency of nervous disorders in the immediate family indicates no correlation with the development of epilepsy (table 146). In the immediate family (parents and siblings) of the nonepileptic head-injured man there is an incidence of epilepsy of 1:191,[3] and in the family of the posttraumatic epileptic a frequency of 1:107.[4] In the more remote relatives

[3] Seventeen epileptics in 3,240 relatives of nonepileptic veterans.
[4] Eleven epileptics in 1,182 relatives of the epileptic veterans.

of the nonepileptic the incidence of epilepsy was computed at 1:452,[5] and in the relatives of the epileptic it is 1:369.[6] These high ratios strongly suggest that the veterans tended to be unaware of or to ignore fits in more distant relatives. The difference in the ratios for immediate family members as between epileptics and nonepileptics is not large enough to be statistically significant. However, the subgroup of veterans with epilepsy who had multiple attacks, not of focal type, show a ratio of only 1:64 in the immediate family.[7] This ratio does differ significantly from the ratio for nonepileptic veterans ($X^2=6.32$, $P<.02$), suggesting that genetic predisposition may have played a role in at least some of the veterans who developed recurrent epilepsy not of focal type.

Table 146.—*Relation of Epilepsy to Nervous Disorders in Family*

Disorder	Number of men	With epilepsy		With multiple focal attacks	
		Number	Percent	Number	Percent
Total	739	207	28.0	65	8.8
None	597	173	29.0	54	9.0
Fainting	20	5	25.0	2	10.0
Migraine	22	5	22.7	3	13.6
Neurosis	37	8	21.6	1	2.7
Psychosis	30	9	30.0	2	6.7
Psychopathic traits	5	2	40.0	.....	.....
Other nervous diseases	25	2		1	.....
Unknown	11	5	45.5	3	27.3

F. FACTORS AT THE TIME OF WOUNDING WHICH MAY MODIFY THE PROBABILITY OF EPILEPSY

It has been known for the past century that certain characteristics at wounding influence the likelihood of the development of epilepsy. In general, these are related to several basic factors: (*1*) severity, (*2*) location, and (*3*) repair of the wound.

1. Severity of Wounding

There are a number of factors directly related to the severity of wounding. These include the dimensions of the wound, the period of unconsciousness, the neurological deficit, and the presence of foreign bodies within the head. It is apparent that a greater likelihood of epilepsy is associated with increasing dimensions of the wound in all planes. Both the diameter of defect (table 147, panel A.) and depth of wounding (panel B.) seem to influence the incidence of epilepsy, especially multiple focal attacks, in a positive manner. Probably reflecting the severity of wounding in a somewhat different way, the duration of unconsciousness (table 148) also seems to

[5] Twelve epileptic relatives in 5,423 relatives of nonepileptic veterans.
[6] Five epileptics in 1,846 relatives of the epileptic veterans.
[7] Nine epileptics in 576 relatives.

Table 147.—*Relation of Epilepsy to Diameter of Cranial Defect and Depth of Wound*

Diameter of defect or depth of wound	Number of men	With epilepsy		With multiple focal attacks	
		Number	Percent	Number	Percent
	A. Diameter of defect				
Total	739	207	28.0	65	8.8
No defect	264	38	14.4	4	1.5
≤2 cm	128	43	33.6	10	7.8
2–4 cm	186	66	35.5	24	12.9
>4 cm	85	32	37.6	14	16.5
Other (including gutter wounds)	21	11	52.4	6	28.6
Unknown	55	17	30.9	7	12.7
	B. Depth of wound				
Total	739	207	28.0	65	8.8
Scalp	207	31	15.0	2	1.0
Cranium	139	24	17.3	6	4.3
Dura mater	20	3	15.0	.....	.....
Brain	338	136	40.2	50	14.8
Ventricle	25	13	52.0	7	28.0
Unknown	10	.....	.....	.....	.....

Table 148.—*Relation of Epilepsy to Period of Unconsciousness*

Period	Number of men	With epilepsy		With multiple focal attacks	
		Number	Percent	Number	Percent
Total	739	207	28.0	65	8.8
None	146	29	19.9	8	5.5
≤2 hours	166	34	20.5	8	4.8
2–24 hours	57	18	31.6	6	10.5
1–3 days	42	14	33.3	6	14.3
3–7 days	35	16	45.7	8	22.9
>7 days	15	8	53.3	3	20.0
Unconscious, time unknown	142	58	40.8	16	11.3
Unknown if unconscious	136	30	22.1	10	7.4

correlate with the incidence of epilepsy. In a similar sphere of reference, the amount of neurological impairment (table 149) is an important factor in the probability of epilepsy, but there is no evidence that any one type of impairment is a more potent determinant than another. The role of

intracranial foreign bodies in the genesis of epilepsy is not clear, but there is no doubt that their presence in this series is strongly associated with epilepsy (table 150). This is in agreement with the previous reports of most authors, but contrary to Ascroft's (8) conclusion that the presence or absence of foreign bodies had no bearing on the incidence of convulsive sequelae.

Table 149.—*Relation of Epilepsy to Neurological Deficit at Time of Wound*

Deficit	Number of men	With epilepsy		With multiple focal attacks	
		Number	Percent	Number	Percent
Total	739	207	28.0	65	8.8
None	341	76	22.3	11	3.2
Hemiplegia or hemiparesis, sensory or motor	148	74	50.0	38	25.7
Hemianopsia	55	20	36.4	8	14.5
Aphasia	99	47	47.5	19	19.2
Other	125	42	33.6	15	12.0

Table 150.—*Relation of Epilepsy to Intracranial Foreign Bodies and Their Removal at Debridement*

Intracranial foreign bodies	Number of men	With epilepsy		With multiple focal attacks	
		Number	Percent	Number	Percent
Total	739	207	28.0	65	8.8
No intracranial foreign bodies	297	52	17.5	8	2.7
Bone fragments only, removed	144	36	25.0	11	7.6
Bone fragments only, not all removed	52	22	42.3	7	13.5
Metal foreign bodies and bone fragments (if present) removed	51	22	43.1	6	11.8
Metal foreign bodies, not all removed; bone fragments (if present) removed	82	31	37.8	15	18.3
Bone fragments not all removed, metal foreign bodies removed	16	6	37.5	4	25.0
Bone fragments and metal foreign bodies remained	60	26	43.3	10	16.7
Unknown	37	12	32.4	4	10.8

2. Location of the Wound

The location of wounding may be apparent as in compound wounds of the brain or may have to be inferred as in closed head wounds where the major damage is thought to be in the brainstem, basal frontal regions, or tip of the temporal lobe. From table 151 it is apparent that the variation

in incidence of epilepsy by region is not striking. Only the occipital wounds are associated with significantly less posttraumatic epilepsy than injuries of the parietal, temporal, and frontal regions. If, as mentioned above, the scalp lacerations and wounds produced by blunt objects be considered as closed head injuries in which the site of wounding is ordinarily brainstem or orbitotemporal cortex, then it is apparent that men with such wounds have not only a lower frequency of fits but also a different type (table 152) for none of the men with scalp lacerations had multiple focal seizures, whereas this was the most sensitive criterion in wounds of the convexity. Major seizures without aurae would seem to be characteristic of wounds affecting the brainstem, basal frontal lobe, and temporal tip.

Most authors (35, 54, 100, 105) have emphasized that parietal lobe wounds are more frequently associated with epilepsy than wounds of other regions. Krause and Schum (74), in reviewing previous studies, concur in this impression. Ascroft (8) states that his figures, "for what they are worth," indicate that wounds in or about the Rolandic area are most often followed by fits, although he admits that the cortex giving rise to fits is not necessarily directly beneath the wound. Russell (109) suggests that injury to the suppressor areas may play a role, but admits that his evidence was not statistically valid, even if the concept of suppressor areas could be considered correct. Neither his figures of the site of wound nor those of Schum (74) suggest more than a slight predominance of central lesions.

Table 151.—*Relation of Epilepsy to Location of Wound*

Region	Number of men	With epilepsy		With multiple focal attacks	
		Number	Percent	Number	Percent
Frontal	246	83	33.7	24	9.8
Parietal	372	134	36.0	53	14.2
Temporal	163	53	32.5	18	11.0
Occipital	176	42	23.9	13	7.4

Table 152.—*Epilepsy in Relation to Type of Wound*

Type of wound	Number of men	With epilepsy		With multiple focal attacks	
		Number	Percent	Number	Percent
Total	739	207	28.0	65	8.8
Closed head injuries	50	9	18.0	2	4.0
Perforating and penetrating wounds	472	169	35.8	61	12.9
Compound fractures	56	6	10.7	2	3.6
Scalp laceration	158	23	14.6	.....	.0
Other	1	.....	.0	.....	.0
Unknown	2	.....	.0	.....	.0

3. Repair of the Wound

Finally, all factors tending to delay the healing of the wound increase the likelihood of epilepsy. However, late primary debridement (table 153) is seen to be associated with little or no increase in epilepsy.

The use of chemotherapeutic agents at the first debridement seems not to be related to the subsequent development of epilepsy, nor does the scalp closure with or without drainage appear to be so related (table 154). Although secondary healing increases the incidence of seizures, this seems to follow directly from the greater probability of secondary healing occurring in severely wounded men, for within the individual R–I groups there is no significant difference in the incidence of epilepsy in patients whose wounds heal primarily or secondarily (table 155), a finding previously noted by Steinthal (117). Complications which may impair healing or increase the extent and severity of wounding are favorable to the development of epilepsy (table 156). The presence of a dural graft for closure adds slightly to the incidence of epilepsy (table 157), but this may be related to the greater severity of wounding in the grafted cases, particularly in R–I group 3.

In general, the development of epilepsy tended to follow wounds of greater severity and with longer reparative processes. The location of the

Table 153.—*Relation of Epilepsy to Time of Primary Debridement (R–I Groups 3 and 4 Only)*

Time	Number of men	With epilepsy		With multiple focal attacks	
		Number	Percent	Number	Percent
Total	455	174	38.2	62	13.6
≤24 hours	332	119	35.8	42	12.7
1–3 days	74	32	43.2	10	13.5
≥3 days	31	12	38.7	7	22.6
Unknown	18	11	61.1	3	16.7

Table 154.—*Relation of Epilepsy to Scalp Closure After Debridement*

Closure	Number of men	With epilepsy		With multiple focal attacks	
		Number	Percent	Number	Percent
Total	634	190	30.0	64	10.1
None	16	4	25.0	2	12.5
Tight	489	153	31.3	49	10.0
Drain	36	12	33.3	5	13.9
Other	5	1	20.0	.....	.....
Unknown	88	20	22.7	8	9.1

wound may be of greater importance in determining the type of seizure, although it does influence to some lesser degree the incidence of epilepsy.

It is interesting to note that the skill of the operator, within groups of injuries of equal severity, has no influence upon the occurrence of epilepsy. It would seem that the more experienced operators were prone to tackle the severely wounded men, and accordingly their patients were more likely to

Table 155.—*Relation of Epilepsy to Wound Healing*

Healing after debridement	Number of men	With epilepsy		With multiple focal attacks	
		Number	Percent	Number	Percent
	R–I group 3				
Total	356	111	31.2	30	8.4
Primary	285	94	33.0	25	8.8
Secondary	51	12	23.5	5	9.8
Unknown	20	5	25.0	.....	.....
	R–I group 4				
Total	96	61	63.5	33	34.4
Primary	41	25	61.0	17	41.5
Secondary	38	25	65.8	11	28.9
Unknown	17	11	64.7	5	29.4

Table 156.—*Relation of Epilepsy to Complications of Debridement*

Complication	Number of men	With epilepsy		With multiple focal attacks	
		Number	Percent	Number	Percent
Total number with debridement	634	190	30.0	64	10.1
None	520	139	26.7	44	8.5
Intracranial hemorrhage	11	5	45.5	2	18.2
Frank infection	25	11	44.0	5	20.0
Abscess	15	11	73.3	5	33.3
Meningitis	9	4	44.4	1	11.1
Fungus cerebri	13	6	46.2	2	15.4
Cerebrospinal rhinorrhea	3	1	33.0	.....	.....
Cerebrospinal otorrhea	3	.....	.....	.....	.....
Cerebrospinal fistula	11	5	45.5	.....	.....
Other	27	10	37.0	4	14.8
Unknown	13	7	53.8	3	23.1

Table 157.—*Relation of Epilepsy to Dural Closure*

Dural closure	Number of men	With epilepsy		With multiple focal attacks	
		Number	Percent	Number	Percent
	R–I group 3				
Total	263	90	34.2	24	9.1
No closure	22	8	36.4	1	4.5
Tight	80	24	30.0	4	5.0
Drain	6	2	33.3	.....	.....
Dural graft	94	41	43.6	15	16.0
Other and unknown	61	15	24.6	4	6.6
	R–I group 4				
Total	84	51	60.7	28	33.3
No closure	14	9	64.3	7	50.0
Tight	12	6	50.0	3	25.0
Drain	2	2	100.0	1	50.0
Dural graft	38	24	63.2	13	34.2
Other and unknown	18	10	55.6	4	22.2

develop epilepsy than the slightly injured men debrided by surgeons of lesser experience (table 158).

It has been suggested by Gardner (51) that cranioplasty carried out early, even at the time of primary debridement, will decrease the pulsations at the site of a cranial defect and thus minimize the stresses to the cortex. This, it is argued, will lessen the likelihood of an epilepsy. In this series, in the few instances in which cranioplasty was carried out at a primary debridement, there is no evidence that it had any effect, positive or negative, upon the probability of epilepsy (table 159). The time between wounding and cranioplasty may bear some relationship to the development of an epilepsy in that a long delay may be associated with a greater likelihood of seizures (table 160), but since cranioplasty was deferred in more seriously injured patients, it is unclear just what was the role of the time of cranioplasty *per se*.

The majority of plates used for cranioplasty were of tantalum, and so few other materials (acrylic and bone) were employed that no valid conclusions can be made as to their influence on epilepsy (table 161). It is of interest to note that relatively few plates had to be removed (table 162) and that subsequent cranioplasties seemed to play no role in the development of epilepsy.

G. POSTTRAUMATIC FACTORS IN THE PROBABILITY OF EPILEPSY

Certain factors relative to the habits of the patient may play a role in the development of epilepsy. Two of these factors have been prominently

Table 158.—*Relation of Epilepsy to Neurosurgical Training of Debriding Surgeon*

Qualification	Number of men	With epilepsy		With multiple focal attacks	
		Number	Percent	Number	Percent
	R–I group 3				
Total	211	72	34. 1	25	11. 8
Neurosurgical specialist, certified	45	15	33. 3	8	17. 8
Neurosurgical specialist, in training	93	37	39. 8	13	14. 0
General surgical, specialist, certified	10	1	10. 0	1	10. 0
General surgical specialist, in training	46	13	28. 3	2	4. 3
Other	17	6	35. 3	1	5. 9
	R–I group 4				
Total	68	44	64. 7	17	25. 0
Neurosurgical specialist, certified	21	14	66. 7	6	28. 6
Neurosurgical specialist, in training	34	21	61. 8	6	17. 6
General surgical specialist, certified	3	2	66. 7	.....	.....
General surgical specialist, in training	7	4	57. 1	4	57. 1
Other	3	3	100. 0	1	33. 3

Table 159.—*Relation of Epilepsy to Cranioplasty Performed at Debridement* [1]

Time	Number of men	With epilepsy		With multiple focal attacks	
		Number	Percent	Number	Percent
	R–I group 3				
Cranioplasty done at debridement	9	4	44. 4	1	11. 1
Cranioplasty done later	181	62	34. 3	19	10. 5
	R–I group 4				
Cranioplasty done at debridement	6	3	50. 0	2	33. 3
Cranioplasty done later	59	39	66. 1	22	37. 3

[1] Three at first, 10 at second, and 2 at third debridement.

Table 160.—*Relation of Epilepsy to Time of Cranioplasty*

Time	Number of men	With epilepsy		With multiple focal attacks	
		Number	Percent	Number	Percent
	R–I group 3				
Total	362	115	31.8	30	8.3
No cranioplasty	145	38	26.2	8	5.5
Cranioplasty:					
Within 3 months of injury	60	19	31.7	5	8.3
3–6 months after injury	112	45	40.2	10	8.9
>6 months after injury	45	13	28.9	7	15.6
	R–I group 4				
Total	96	61	63.5	33	34.4
No cranioplasty	17	11	64.7	6	35.3
Cranioplasty:					
Within 3 months of injury	10	3	30.0	2	20.0
3–6 months after injury	35	23	65.7	12	34.3
>6 months after injury	34	24	70.6	13	38.2

Table 161.—*Relation of Epilepsy to Type of Plate*

Type of plate	Number of men	With epilepsy		With multiple focal attacks	
		Number	Percent	Number	Percent
Total with cranioplasty	307	128	41.7	49	16.0
Tantalum	283	122	43.1	46	16.3
Plastic	15	6	40.0	3	20.0
Other	9	.....	.....	.....	.....

Table 162.—*Relation of Epilepsy to Secondary Operations on Plate*

Operation	Number of men	With epilepsy		With multiple focal attacks	
		Number	Percent	Number	Percent
Total with cranioplasty	307	128	41.7	49	16.0
No secondary operations	280	115	41.1	44	15.7
Secondary operation	27	13	48.1	5	18.5
Plate removed	21	11	52.4	4	19.0

mentioned off and on from earliest times, namely, (*1*) the abuse of alcohol and (*2*) constipation.

An examination of the drinking habits of the men in this series brings out several interesting facts (table 163). From the top panel of this table, it can be seen that while the relative frequency of heavy drinkers is the same in men with and without epilepsy, a larger proportion of the men with epilepsy (27.5 percent) than of the men without fits (17.7 percent) were abstainers. From the breakdown by severity of injury, it can be seen that the situation is quite complex, and that in all probability all three of the factors here considered affect each other mutually. Thus, among the slightly injured men and those whose wounds were only moderately severe, alcohol consumption and epilepsy seem to be unrelated. However, among the most severely injured patients (R–I group 4), the proportion of abstainers is elevated among men with epilepsy (as contrasted to men without fits), and the proportion of heavy drinkers is depressed, although the differences are not statistically significant. A significantly larger proportion of severely injured men were abstainers than were those with slight or moderately severe wounds, and this is true both of men with and without

Table 163.—*Relation of Drinking Habits of Patients to Severity of Wounding and Epilepsy*

Drinking habits R–I groups	Total		With epilepsy		Without epilepsy	
	Number	Percent	Number	Percent	Number	Percent
AU R–I groups						
Total	739	100.0	207	100.0	532	100.0
Abstainer	151	20.4	57	27.5	94	17.7
Occasional drinker	506	68.5	127	61.4	379	71.2
Frequent or bouts	78	10.6	22	10.6	56	10.5
Unknown	4	.5	1	.5	3	.6
R–I groups 1 and 2 (slight wounds)						
Total	281	100.0	31	100.0	250	100.0
Abstainer	49	17.4	6	19.4	43	17.2
Occasional drinker	204	72.6	22	71.0	182	72.8
Frequent or bouts	28	10.0	3	9.7	25	10.0
R–I group 3 (moderately severe wounds)						
Total	362	100.0	115	100.0	247	100.0
Abstainer	68	18.8	26	22.6	42	17.0
Occasional drinker	250	69.1	77	67.0	173	70.0
Frequent or bouts	40	11.0	11	9.6	29	11.7
Unknown	4	1.1	1	.9	3	1.2
R–I group 4 (severe wounds)						
Total	96	100.0	61	100.0	35	100.0
Abstainer	34	35.4	25	41.0	9	25.7
Occasional drinker	52	54.2	28	45.9	24	68.6
Frequent or bouts	10	10.4	8	13.1	2	5.7

fits. While interpretation must be largely conjectural, it seems most reasonable to conclude that alcohol consumption had little to do with whether a patient ever had fits or not (although epilepsy may have been exacerbated by drinking) but that severe impairments caused men to cut down on their drinking.

Constipation seems to be definitely and significantly associated with epilepsy of the focal type (table 164). The relationship is found in all R–I groups, hence is not merely a concomitant of other disorders. If R–I groups 3 and 4 are combined (since the frequency of constipation is not

Table 164.—*Relation of Epilepsy to Constipation*

Constipation	Number of men	With epilepsy		With multiple focal attacks	
		Number	Percent	Number	Percent
All men					
Total	739	207	28.0	65	8.8
None	561	147	26.2	43	7.7
Occasional difficulty	124	37	29.8	8	6.5
Chronic	49	20	40.8	13	26.5
Unknown	5	3	60.0	1	20.0
R–I groups 3 and 4					
Total	458	176	38.4	63	13.8
None	348	127	36.5	42	12.1
Occasional difficulty	74	31	41.9	7	9.5
Chronic	31	15	48.4	13	41.9
Unknown	5	3	60.0	1	20.0

Table 165.—*The Course of Posttraumatic Epilepsy in 7- to 8-Year Period*

Frequency of attack	Type of attack					
	Any		Major		Minor	
	Number	Percent	Number	Percent	Number	Percent
Total	207	100.0	149	100.0	132	100.0
Continuing attacks (more than 2 per year)	91	44.0	44	29.5	60	45.5
Rare attacks (1 to 2 per year)	46	22.2	37	24.8	24	18.2
No attacks:						
For at least 2 years	70	33.8	67	45.0	46	34.8
For at least 5 years	44	21.3	36	24.2	34	25.8
Unknown	.....	.....	1	.7	2	1.5

dependent upon R–I groups), and if the total epilepsy group is divided into those with focal attacks and those with other types, the lower panel of table 164 is obtained, whence it is apparent that at least 3 times as large a proportion of men with chronic constipation have multiple focal attacks as do other men.

H. COURSE OF THE EPILEPSY

The occurrence of one or more convulsive episodes following a head injury does not indicate that there will be recurring attacks. If the series of 207 men having posttraumatic epilepsy is analyzed for the occurrence of attacks in the 2- and 5-year period before follow-up, one-third of the group is found to have had no seizures for at least 2 years (table 165). Another one-fifth has had rare attacks, one or two per year. Less than half has had more than two attacks of any type per year. If only major attacks are considered, the results are still more favorable for the patient; only 30 percent of the men who ever had major fits have had such attacks more than twice a year, and 45 percent have had none for 2 years.

This favorable course has been known for patients having their initial epileptic manifestations within a few days of a head injury. These convulsions have generally been considered to result from the reaction to the acute stage of hyperemia, edema, necrosis, and glial reaction of the brain, whereas the later epilepsies are thought to be related to the chronic scarring of the brain; Röttgen (103) believes they are related to a connective tissue scar. There is some evidence from an analysis of this series to support the concept of a more favorable prognosis for the early epilepsies. If the course of the epilepsies occurring (*1*) within the first week, (*2*) from the first week to the third month, and (*3*) later than the third month, within the first year after injury, are plotted in terms of epilepsy or no epilepsy for each year, it is apparent that the early epilepsies have a definitely lower incidence each year

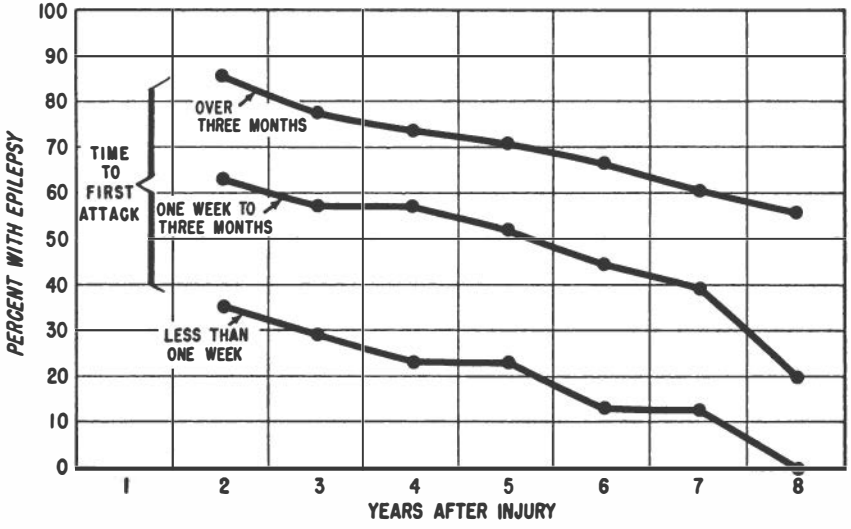

Figure 1.—*Relation of the Course of Epilepsy to Time of Initial Attack Within the First Year*

than the later developing cases (fig. 1). It must be admitted that there is no sharp distinction between early and late epilepsies. Just as there is a continuum in the graph showing time of onset (figs 2–4), there is a progressive deterioration in prognosis as the time of onset increases at least up to a year.

To analyze further prognostic factors in the course of the epilepsy, the series was broken into two groups.

Group 1: 58 men having one or more attacks in the first 4 years after injury but free of any type of attack after the fourth year.

Group 2: 126 men having one or more attacks in the first 4 years and continuing to have attacks.

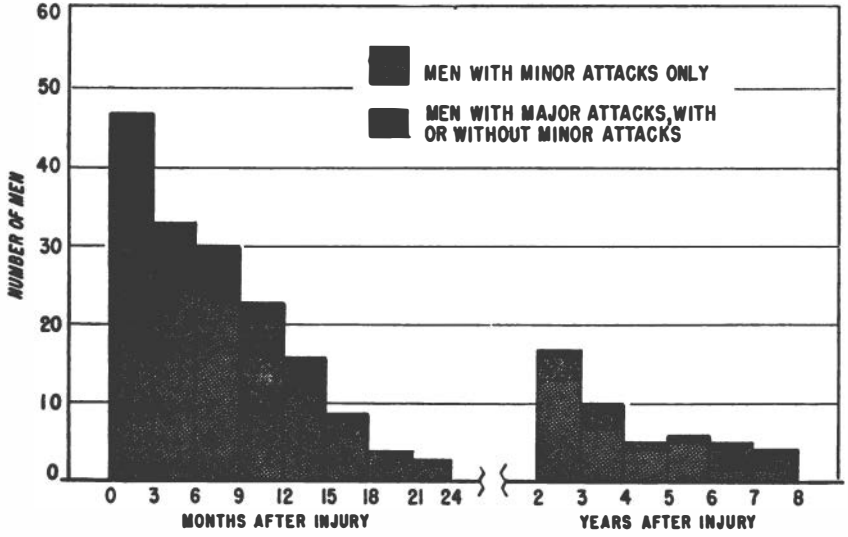

Figure 2.—*Time of First Epileptic Attack*

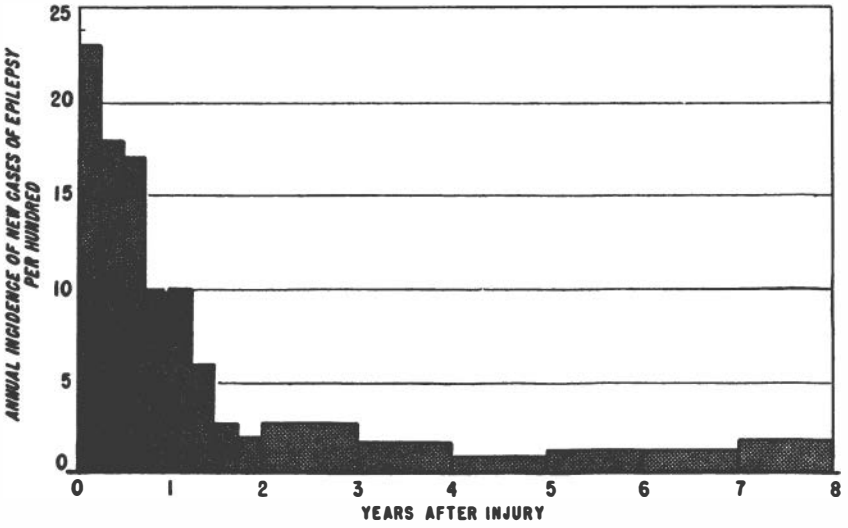

Figure 3.—*Annual Incidence of New Cases of Epilepsy by Time From Injury*

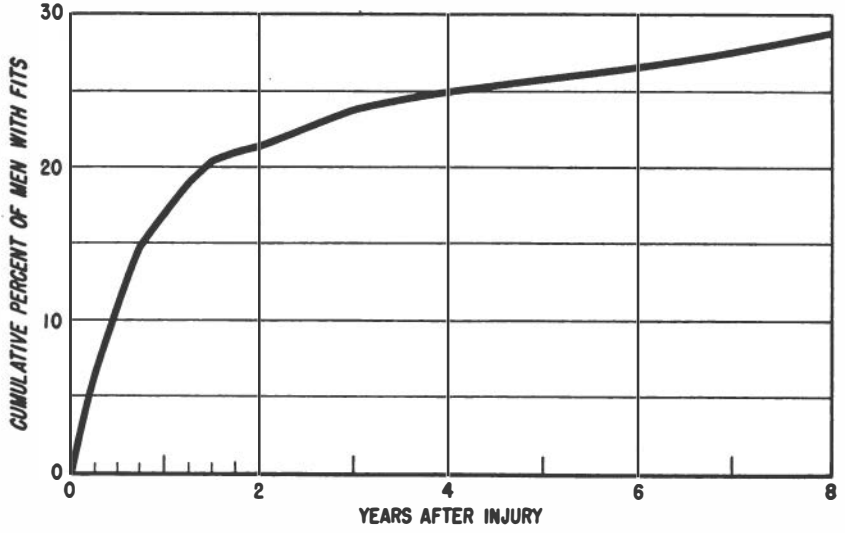

Figure 4.—*Cumulative Percent of Men With Fits as a Function of Time From Injury*

These two groups were compared with regard to location of wound, depth of wound, time of debridement, complications, neurological deficit, handedness, types of attacks, and other factors, without finding any significant difference between the two groups. Only in two respects were differences of statistical significance noted.

The seizure-free group had a lower incidence of the posttraumatic syndrome (table 166). There is, in general, a lower intelligence in the seizure group than in the seizure-free group (table 167). Both of these phenomena may be the result of the seizures rather than the cause.

There have been a few references in the literature to the favorable prognosis of some posttraumatic epilepsies, although many authors, as Penfield (95), have concluded that, once established, the prognosis was bad. Alajouanine et al. (4) pointed out that 16 percent of their patients were free

Table 166.—*Relation of Clinical Symptomatology to Seizure-Free Patients (R–I Group 1) and Patients Continuing to Have Seizures (R–I Group 2)*

Symptom	Group 1 [1]		Group 2 [1]	
	Number	Percent	Number	Percent
None	5	8.6	7	5.6
Posttraumatic syndrome	28	48.3	88	69.8
All other	25	43.1	31	24.6
Total	58	100.0	126	100.0

[1] The difference between the two distributions is statistically significant ($P < .05$).

Table 167.—*Relation of Wechsler-Bellevue Intelligence Test to Patients Seizure-Free (R–I Group 1) and Patients Continuing to Have Seizures (R–I Group 2)*

Wechsler-Bellevue test	Group 1 [1]		Group 2 [1]	
	Number	Percent	Number	Percent
≤89	5	8.9	25	21.4
90–119	39	69.6	78	66.7
≥120	12	21.4	14	12.0
Total	56	100.0	117	100.0
Unknown	2	.....	9	.....

[1] The difference between the two groups is statistically significant ($P < .05$).

Table 168.—*Medication Received by Men Having Major Attacks 3 to 6 Years After Injury*

Medication	Year after injury			
	Third	Fourth	Fifth	Sixth
Number of men with major attacks in this year	83	85	77	67
	Number			
Medication—total number of men	50	55	51	45
Phenobarbital ≤3 grs	12	12	10	10
Phenobarbital >3 grs	11	12	7	6
Dilantin ≤4½ grs	11	15	16	13
Dilantin >4½ grs	9	11	10	10
Phenobarbital ≤3 grs. and Dilantin ≤4½ grs	18	17	15	15
Other drugs	1	3	3	1
	Percent			
Phenobarbital ≤3 grs	24.0	21.8	19.6	22.2
Phenobarbital >3 grs	22.0	21.8	13.7	13.3
Dilantin ≤4½ grs	22.0	27.3	31.4	28.9
Dilantin >4½ grs	18.0	20.0	19.6	22.2
Phenobarbital ≤3 grs. and Dilantin ≤4½ grs	36.0	30.9	29.4	33.3
Other drugs	2.0	5.5	5.9	2.2

of attacks. Steinthal (116, 118) stated that of his 185 cases of typical epilepsy, 49 percent became cured, although he does not define the term "cured," nor does he give the precise time of onset of the attacks. Wilson (139) observed, in his series of head injuries in servicemen of World War II, that half the patients who had epileptic attacks had no attacks for at least the last 2 years of a 6-year follow-up. Walker (134) has noted a similar tendency in a group of 246 posttraumatic epileptics of World War II. Some 46 percent of the patients had no convulsive episodes in the 5th to 10th year after injury.

I. TREATMENT OF EPILEPSY

Retrospective surveys are notoriously poor instruments for evaluation of the effectiveness of therapy. Investigators have found, time and again, that in such material, intensity of treatment is associated with worsening of prognosis. Hence, there is no intent here to try to evaluate the efficacy of various medications in the treatment of epilepsy. It is thought, however, that it may be of interest to know which medications were being employed, and how many men received medication. The data are shown in tables 168 and 169 for men with major and minor attacks, respectively.

Table 169.—*Medication Received by Men Having Minor Attacks 3 to 6 Years After Injury*

Medication	Year after injury			
	Third	Fourth	Fifth	Sixth
Number of men with minor attacks in this year	80	80	76	59
	Number			
Medication—total number of men	37	37	45	35
Phenobarbital ≤3 grs	13	12	13	11
Phenobarbital >3 grs	7	6	8	5
Dilantin ≤4½ grs	5	6	9	6
Dilantin >4½ grs	8	9	9	7
Phenobarbital ≤3 grs. and Dilantin ≤4½ grs	11	11	10	9
Other drugs	.....	2	2	.....
	Percent			
Phenobarbital ≤3 grs	35.1	32.4	28.9	31.4
Phenobarbital >3 grs	18.9	16.2	17.8	14.3
Dilantin ≤4½ grs	13.5	16.2	20.0	17.1
Dilantin >4½ grs	21.6	24.3	20.0	15.6
Phenobarbital ≤3 grs. and Dilantin ≤4½ grs	29.7	29.7	22.2	25.7
Other drugs	.....	5.4	4.4	.....

J. DISCUSSION

It is apparent as the discussion of the epileptic manifestations is read that their relation to the localization of wounding does not correspond exactly with our present concepts of localization of cerebral function, for occipital lobe wounds are associated with focal motor seizures and frontal wounds with seizures having visual aurae. Either current thoughts on cerebral functions must be modified or it must be admitted that the site of cranial wounding may not correspond precisely with the site of cerebral injury. The latter would seem the most logical course as contrecoup and polar contusions resulting from rotation of the brain are well known. Thus a wound of the frontal region on one side may be accompanied by a contusion of the ipsilateral temporal tip, contralateral occipital pole, or brainstem, any one of which sites might serve as a focus for an epilepsy. This multiplicity of cerebral wounding has been recognized for years, but its importance in the genesis of seizures having focal manifestations referable to cerebral aurae quite remote from the wound, even on the contralateral hemisphere, has not been adequately emphasized. The fact that a relatively small contusion rather than a large cerebral scar may be the epileptic focus demands that special care be taken to analyze every case, both clinically and electroencephalographically.

That constitutional or systemic factors play a role in the initiation or recurrence of the attacks seems possible from the observed data relative to constipation, alcohol consumption, etc., but it is difficult to separate cause and effect. Is the constipation the result of the attack or attacks, or related in any way causally to them? Similarly, the intolerance to alcohol poses a question which cannot be answered by a retrospective research design.

Chapter VI

SOCIAL AND ECONOMIC REHABILITATION

In the final analysis, the complete rehabilitation of the head-injured patient is the goal of the therapist. How well this can be accomplished, and the factors modifying its achievement, are the subject of this chapter.

It is immediately apparent that the severity of injury plays an important role in certain spheres of rehabilitation (table 170). The rehabilitation of the badly injured men is related to severity of wounding; this factor seems to affect more strongly the readjustment in work and social circles than in economic and home spheres. Unemployment is progressively more frequent as the severity of wounding increases. Similarly, the less severely wounded are more likely to advance in their jobs, whereas those more impaired tend to regress. These observations, coupled with the relatively satisfactory economic and home adjustments, might suggest that the compensation being received goes far to meet the immediate needs of the head-injured so that relatively few have excessive worries.

The development of epilepsy, like the severity of injury, may play a role in the work and social rehabilitation, but seems to influence less the home and economic adjustment. Particularly, the occurrence of multiple focal seizures decreases the likelihood of employment and advance in work (table 171). In fact, in all respects, the epileptic is a little worse off than the nonepileptic head-injured man. Moreover, the presence of epilepsy appears to have an influence on rehabilitation, which is independent of the fact that the men with epilepsy were, on the average, more severely wounded than the men without epilepsy (table 172). Even within fixed injury groups, men with epilepsy are more frequently unemployed, have advanced in work less frequently, and have made unsatisfactory home and social adjustments more often (table 173).

On the other hand, the electroencephalogram (table 174), which might be considered as indicating an epileptic diathesis, is not well correlated with the work, social, economic, or home status. Only men unemployed or regressing in work have significantly different EEG's from other men, in that they are more likely to have focal abnormalities.

The intelligence of the head-injured men is an important determinant in the work status (table 175). In the group of men who were unemployed, had odd jobs, or were hospitalized at the time of follow-up, the proportion of deteriorated persons was higher than in the other groups. Of 145 deteriorated men, 48, or 33.1 percent, had no steady employment, as against 56 of 538, or 10.4 percent, for nondeteriorated men. On the other hand, only a low proportion of men advancing in their jobs were deteriorated or had IQ's of 89 or less. However, the intelligence of the individual, at least as measured by the Wechsler-Bellevue test, seems to be a less important factor in home and social adjustment (table 176).

Table 170.—*Relation of Vocational, Social, and Economic Status at Follow-up to Severity of Injury*

Status	R–I group							
	1		2		3		4	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Total	99	100.0	182	100.0	362	100.0	96	100.0
Vocational:								
Unemployed or occasional jobs	2	2.0	23	12.6	55	15.2	30	31.3
Attending school	4	4.0	4	2.2	22	6.1	11	11.5
Returned to former job	7	7.1	22	12.1	26	7.2	6	6.3
Advanced at former job	23	23.2	23	12.6	39	10.8	2	2.1
New work	63	63.6	109	59.9	215	59.4	42	43.8
In hospital	.....	.....	1	.....	3	.8	4	4.2
Unknown	.....	.....	.....	.....	2	.6	1	1.0
Regression in work	7	7.1	30	16.5	77	21.3	36	37.5
Unsatisfactory adjustment:								
Home	6	6.1	26	14.3	37	10.2	14	14.6
Social	13	13.1	46	25.3	65	18.0	33	34.4
Economic	3	3.0	32	17.6	43	11.9	14	14.6

Table 171.—*Relation of Vocational, Social, and Economic Status at Follow-up to Epilepsy*

Status	Without epilepsy		With epilepsy		With multiple focal attacks	
	Number	Percent	Number	Percent	Number	Percent
Total	532	100.0	207	100.0	65	100.0
Vocational:						
Unemployed or occasional jobs	64	12.0	46	22.2	21	32.3
Attending school	25	4.7	16	7.7	10	15.4
Returned to former job	43	8.1	18	8.7	6	9.2
Advanced at former job	71	13.3	16	7.7	2	3.1
New work	325	61.1	104	50.2	23	35.4
In hospital	3	.6	5	2.4	2	3.1
Unknown	1	.2	2	1.0	1	1.5
Regression in work	85	16.0	65	31.4	28	43.1
Unsatisfactory adjustment:						
Home	53	10.0	30	14.5	11	16.9
Social	97	18.2	60	29.0	20	30.8
Economic	57	10.7	35	16.9	9	13.8

The personality seems to play a role in the work adjustment, for unemployed men have a higher proportion of elevated Hs, D, Hy, Pt, and Sc scores than employed men. Similarly, those scores are elevated in a higher percentage of men showing no progress or regressing in their work (table 177) than in men advancing in their jobs.

In addition to having a higher proportion of men with high scores in the above categories, men poorly adjusted at home frequently tend to have elevated Pd and Ma, while the social misfits exhibit a smaller degree of elevation of thePd score and no significant elevation of the Ma scale.

There would, then, seem to be a number of factors other than the neurological deficit which may modify the rehabilitation of the head-injured patient. For this reason the therapy of these patients might profitably be preceded by a series of psychological tests, which may indicate the chances of a successful therapeutic outcome. The individual with an IQ below 90, having evidence of mental deterioration and personality aberrations, has relatively little chance of returning to a useful vocation. On the other hand, time and effort expended on the supernormal individual without personality abnormalities, even if he has a severe neurological handicap, may well result in a successful rehabilitation.

Table 172.—*Relation of Vocational Status at Follow-up to Epilepsy and R–I Group*

Status	R–I group			
	3		4	
	Number	Percent	Number	Percent
	No epilepsy			
Total	247	100. 1	35	100. 1
Hospitalized	1	0. 4	1	2. 9
Unemployed or occasional jobs	33	13. 4	8	22. 9
Attending school	12	4. 9	5	14. 3
Employed	200	81. 0	21	60. 0
Other and unknown	1	0. 4	.....	.....
Regression in work	41	16. 6	11	31. 4
	Epilepsy			
Total	115	100. 0	61	99. 9
Hospitalized	2	1. 7	3	4. 9
Unemployed or occasional jobs	22	19. 1	22	36. 1
Attending school	10	8. 7	6	9. 8
Employed	80	69. 6	29	47. 5
Other and unknown	1	0. 9	1	1. 6
Regression in work	36	31. 3	25	41. 0
	Multiple focal attacks			
Total	30	99. 9	33	100. 1
Hospitalized	.....	.....	2	6. 1
Unemployed or occasional jobs	6	20. 0	15	45. 5
Attending school	7	23. 3	3	9. 1
Employed	16	53. 3	13	39. 4
Other and unknown	1	3. 3	.....	.....
Regression in work	12	40. 0	16	48. 5

Table 173.—*Relation of Home, Social, and Economic Adjustment to Epilepsy and R–I Group*

Adjustment	R–I group			
	3		4	
	Number	Percent	Number	Percent
	No epilepsy			
Total	247	100.0	35	100.0
Unsatisfactory:				
Home	22	8.9	3	8.6
Social	39	15.8	10	28.6
Economic	23	9.3	3	8.6
	All epilepsy			
Total	115	100.0	61	100.0
Unsatisfactory:				
Home	15	13.0	11	18.0
Social	26	22.6	23	37.7
Economic	20	17.4	11	18.0
	Multiple focal attacks			
Total	30	100.0	33	100.0
Unsatisfactory:				
Home	4	13.3	7	21.2
Social	7	23.3	13	39.4
Economic	3	10.0	6	18.2

Table 174.—*Relation of Vocational, Social, and Economic Status to Electroencephalogram*

Status	Total	Electroencephalogram			
		Generalized abnormality		Focal abnormality	
		Number	Percent	Number	Percent
Total	595	110	18.5	135	22.7
Vocational:					
Unemployed or occasional jobs	94	16	17.0	30	[1] 31.9
Attending school	35	6	17.1	7	20.0
In hospital	5	2	40.0	2	40.0
Employed	458	85	18.6	96	21.0
Unknown	3	1	(*)	.....	(*)
Regression in work	121	23	19.0	44	[2] 36.4
Unsatisfactory adjustment:					
Home	69	8	11.6	15	21.7
Social	138	24	17.4	35	25.4
Economic	72	11	15.3	22	30.6

[1] Differs significantly from percent in total group ($P \leq .05$).
[2] Differs significantly from percent in total group ($P \leq .01$).
*Not calculated.

Table 175.—*Relation of Wechsler-Bellevue Intelligence Test to Work Status*

Status	Number of men	Wechsler-Bellevue			
		Intelligence Scale			Abnormally deteriorated
		≤89	90–119	≥120	
	Number				
Total	683	88	470	125	145
Hospitalized	4	1	3	.....	
Unemployed or occasional jobs	100	37	60	3	45
Attending school	39	4	17	18	7
Employed	537	46	387	104	90
Unknown	3	.....	3	.....	.....
Regression in work	135	34	98	3	53
	Percent				
Total	100.0	12.9	68.8	18.3	21.2
Hospitalized	100.0	25.0	75.0	.....	75.0
Unemployed or occasional jobs	100.0	37.0	60.0	3.0	[1] 45.0
Attending school	100.0	10.3	43.6	46.2	17.9
Employed	100.0	8.6	72.1	19.4	16.8
Regression in work	100.0	25.2	72.6	2.2	39.3

[1] Differs significantly from percent in total group ($P \leq .01$).

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Table 176.—*Relation of Wechsler-Bellevue Intelligence Test to Home, Social, and Economic Adjustment*

Adjustment	Number of men	Wechsler-Bellevue			
		Intelligence Scale			Abnormally deteriorated
		≤89	90–119	≥120	
	Number				
Total	683	88	470	125	145
Unsatisfactory:					
Home	75	19	49	7	23
Social	138	22	98	18	39
Economic	88	22	53	13	23
	Percent				
Total	100.0	12.9	68.8	18.3	21.2
Unsatisfactory:					
Home	100.0	25.3	65.3	9.3	30.7
Social	100.0	15.9	71.0	13.0	28.3
Economic	100.0	30.8	53.8	15.4	30.8

Table 177.—*Relation of Adjustment to Minnesota Multiphasic Personality Factors*

Status	Number of men	Scale								
		Hs	D	Hy	Pd	Mf	Pa	Pt	Sc	Ma
		Percent with abnormally elevated score								
Total	485	36. 9	28. 7	31. 3	6. 6	2. 5	1. 9	15. 5	16. 5	4. 9
		Work status								
Unemployed or occasional jobs	48	[1]54. 2	[2]47. 9	[1]45. 8	6. 2	6. 2	.....	[2]33. 3	[2]35. 4	10. 4
School	31	29. 0	29. 0	22. 6	12. 9	[2]12. 9	.....	16. 1	22. 6	3. 2
Employed	401	35. 4	26. 2	30. 2	6. 2	1. 0	2. 2	13. 2	13. 7	4. 5
		Work progress								
None	120	[2]48. 3	[2]39. 2	36. 7	10. 8	1. 7	4. 2	[1]22. 5	[2]25. 0	5. 0
Regression	69	46. 4	37. 7	36. 2	7. 2	1. 4	.....	17. 4	23. 2	8. 7
Advancement, including school	287	30. 0	22. 0	27. 5	4. 9	2. 8	1. 4	12. 2	11. 5	4. 2
		Home adjustment								
Depressed, restless, unhappy	37	51. 4	[2]56. 8	43. 2	[2]32. 4	8. 1	5. 4	[2]32. 4	[2]56. 8	[1]16. 2
		Social adjustment								
Has difficulty	80	[1]50. 0	[2]43. 8	[1]43. 8	[1]12. 5	6. 3	5. 0	[2]28. 8	[2]33. 8	7. 5

[1] Differs significantly from percent in total group ($P \geq .05$).
[2] Differs significantly from percent in total group ($P \geq .01$).

Bibliography

1. Adler, A.: MENTAL SYMPTOMS FOLLOWING HEAD INJURY; A STATISTICAL ANALYSIS OF 200 CASES, A. M. A. Arch. Neurol. & Psychiat. *53*: 34–43, 1945.
2. Aita, J. A.: MODERN CONSIDERATIONS OF THE MAN WITH BRAIN INJURY, J. Neurosurg. *4*: 240–254, 1947.
3. Akerlund, E.: BEHANDLINGEN AV COMMOTIO CEREBRI DIRIGERAD EFTER EVIPANPROVET, Nord. Med. *56*: 1617–1619, 1956.
4. Alajouanine, T., Maisonnet, J., and Petit-Dutaillis, D.: SUITES ÉLOIGNÉES DE LA TRÉPANATION DU CRÂNE POUR LÉSIONS TRAUMATIQUES, J. chir. (Par) *32:* 397–432, 1928.
5. Allen, D. P., Sanford, H. L., and Dolley, D. H.: TRAUMATIC DEFECTS OF THE SKULL: THEIR RELATION TO EPILEPSY. A CLINICAL AND EXPERIMENTAL STUDY OF THEIR REPAIR, Boston M. & S. J. *CLIV*: 396–405, 1906.
6. Alstrom, C. H.: A STUDY OF EPILEPSY IN ITS CLINICAL, SOCIAL AND GENETIC ASPECTS, Acta Psychiat. et Neurol. (Suppl. 63) pp. 1–284, 1950.
7. Aschaffenburg, G.: ZUR FRAGE DER PSYCHOGENEN REAKTIONEN UND DER TRAUMATISCHEN NEUROSEN, Deut. med. Wchnschr. *52*: 1594–1595, 1926.
8. Ascroft, P. B.: TRAUMATIC EPILEPSY AFTER GUNSHOT WOUNDS OF THE HEAD, Brit. M. J. *1*: 739–744, 1941.
9. Askenasy, H. M., Herzberger, E. E., and Wijsenbeek, H.: TRAUMATIC LESIONS OF THE OPTIC NERVES AND CHIASMA, Folia Psychiat. Neerl. *57*:1–16, 1954.
10. Baumm, H.: ERFAHRUNGEN UBER EPILEPSIE BEI HIRNVERLETZTEN, Ztschr. f.d. ges. Neurol. u. Psychiat. *127*:279–311, 1930.
11. Baumoel, S., and Marks, M. I.: EVALUATION OF VERTIGO FOLLOWING HEAD INJURIES, Arch. Otolaryng. *33*: 204–215, 1941.
12. Bay, E.: DIE TRAUMATISCHEN HIRNSCHÄDIGUNGEN, IHRE FOLGEZUSTÄNDE UND IHRE BEGUTACHTUNG, Fortschr. Neurol. Psychiat. Stuttgart *21:* 151–181, 1953.
13. Beckett, W.: PRACTICAL SURGERY, ILLUSTRATED AND IMPROVED, London, C. Corbett, 1740.
14. Beebe, Gilbert W., and DeBakey, Michael E.: BATTLE CASUALTIES. INCIDENCE, MORTALITY, AND LOGISTIC CONSIDERATIONS, Springfield, Thomas, 1952.

15. Béhague, P.: ETUDE SUR L'ÉPILEPSIE TRAUMATIQUE, (Thése de Paris, Arnette), Par., 1919.
16. Berengarius, J.: TRACTATUS DE FRACTURA CALVAE SIVE CRANEI A CARPO EDITUS. Bononiae, per H. de Benedictus, 1518.
17. Bielschowsky, P.: STÖRUNGEN DES LIQUORSYSTEMS BEI SCHÄDELTRAUMEN. Ztschr. f.d. ges. Neurol. u. Psychiat. *117*: 55–117, 1928.
18. Birkelo, C. C., Chamberlain, W. E., Phelps, P. S., Schools, P. E., Zacks, D., and Yerushalmy, J.: TUBERCULOSIS CASE FINDING; COMPARISON OF EFFECTIVENESS OF VARIOUS ROENTGENOGRAPHIC AND PHOTOFLUOROGRAPHIC METHODS, J. A. M. A. *133*: 359–366, 1947.
19. Bouchet et Cazauvieilh: DE L'ÉPILEPSIE CONSIDÉRÉE DANS SES RAPPORTS AVEC L' ALIÉNATION MENTALE. RECHERCHES SUR LA NATURE ET LE SIÈGE DE CES DEUX MALADIES. Arch. Gén. Méd. *9:* 510–542, 1825; *10*: 5–50, 1826.
20. Braun, W.: EPILEPSIE NACH KOPFVERLETZUNGEN, Neue Deut. Chir. *18*: 93–207, 1916.
21. Brändle, K.: DIE POSTTRAUMATISCHEN OPTICUSSCHÄDIGUNGEN (INSBESONDERE DIE OPTICUSATROPHIE), Confinia Neurol., Basel *15*: 169–208, 1955.
22. Breasted, J. H.: THE EDWIN SMITH SURGICAL PAPYRUS, IN FACSIMILE AND HIEROGLYPHIC TRANSLITERATION WITH TRANSLATION AND COMMENTARY IN TWO VOLUMES, Chicago, University of Chicago Press, 1930.
23. Browder, J., and Hollister, N. R.: AIR ENCEPHALOGRAPHY AND VENTRICULOGRAPHY AS DIAGNOSTIC AIDS IN CRANIOCEREBRAL TRAUMA, A. Research Nerv. & Ment. Dis. Proc. *24*: 421–436, 1945.
24. Brun, R.: KATAMNESTISCHE UNTERSUCHUNGEN ÜBER VERLAUF UND SPÄTFOLGEN VON SCHÄDEL—UND GEHIRNTRAUMEN AUF GRUND EINES KRANKENGUTES VON 1648 FÄLLEN, Zschr. Unfallmed., Zür. *49:* 3–45, 1956.
25. Bryson, J. G.: OCULAR MANIFESTATIONS OF INTRACRANIAL INJURY, Mil. Surgeon *104*: 348–358, 1949.
26. Busse, E. W.: ELECTROENCEPHALOGRAM ASSOCIATED WITH POST-TRAUMATIC HEADACHES (IN PATIENTS WITH SKULL DEFECTS), Dis. Nerv. System *8*: 299–306, 1947.
27. Cedermark, J.: ÜBER VERLAUF, SYMPTOMATOLOGIE UND PROGNOSE KRANIOZEREBRALER VERLETZUNGEN, Acta Chir. Scand. *86*: Suppl., 75: 199 pp., 1942.
28. Charcot, J. M.: LEÇONS DU MARDI, À LA SALPÊTRIÈRE, POLICLINQUES 1887–1888. NOTES DE COURS DE M. M. BLIN., CHARCOT (FILS) ET COLEN. Paris, A. Delahaye and E. Lecosniec, 638 pp., 1888.

29. Clark, E. C. and Harper, E. O.: ELECTROENCEPHALOGRAPHIC FINDINGS IN 186 CASES OF CHRONIC POST-TRAUMATIC ENCEPHALOPATHY, Electroencephalog. & Clin. Neurophysiol. *3:* 9–14, 1951; correction 3: 224, May 1951.
30. Courville, C. B.: COMMOTIO CEREBRI; CEREBRAL CONCUSSION AND THE POST-CONCUSSION SYNDROME IN THEIR MEDICAL AND LEGAL ASPECTS, Los Angeles, San Lucas Press, 161 pp., 1953.
31. Cramer, F., Paster, S., and Stephenson, C.: CEREBRAL INJURIES DUE TO EXPLOSION WAVES—"CEREBRAL BLAST CONCUSSION;" PATHOLOGIC, CLINICAL AND ELECTROENCEPHALOGRAPHIC STUDY, A.M.A. Arch. Neurol. & Psychiat. *61*: 1–20, 1949.
32. Credner, L.: KLINISCHE UND SOZIALE AUSWIRKUNGEN VON HIRNSCHÄDIGUNGEN, Ztschr. f.d. ges. Neurol. u. Psychiat. *126*: 721–757, 1930.
33. Dailey, C. A.: PSYCHOLOGIC FINDINGS FIVE YEARS AFTER HEAD INJURY, J. Clin. Psychol. *12*: 349–353, 1956.
34. Davies, H., and Falconer, M. A.: VENTRICULAR CHANGES AFTER CLOSED HEAD INJURIES, J. Neurol. & Psychiat. *6*: 52–68, 1943.
35. Denny-Brown, D.: SEQUELAE OF WAR HEAD INJURIES, New England J. Med. *227*: 771–780, 1942.
36. Denny-Brown, D.: DISABILITY ARISING FROM CLOSED HEAD INJURY, J. A. M. A. *127*: 429–436, 1945.
37. Dow, R. S., Ulett, G., and Raaf, J: ELECTROENCEPHALOGRAPHIC STUDIES IN HEAD INJURIES, J. Neurosurg. *2:* 154–169, 1945.
38. Dubitscher, F.: FESTSTELLUNGEN BEI 1000 HIRNVERLETZTEN AN HAND DER VERSORGUNGSAKTEN, Monatsschr. Unfallh. *56*: 65–82, 1953.
39. Echeverria, M–G.: DE LA TRÉPANATION DANS L'ÉPILEPSIE PAR TRAUMATISMES DU CRÂNE. Arch. Gén. Méd. *142*: 529–554, 652–676, 1878.
40. Eckstein, O. G.: THE HISTORY OF THE LAW OF PENSIONS TOGETHER WITH A SUMMARY OF THE PENSION LAWS OF THE UNITED STATES. IN THE JOHNSON PRIZE ESSAYS FROM VARIOUS LAW SCHOOLS. Ed. by G. W. Pepper, Philadelphia, T. & J. Johnson & Co., 1890.
41. Eguchi, T.: ZUR KENNTNIS DER TRAUMATISCHEN EPILEPSIE NACH KOPFVERLETZUNGEN IM JAPANISCH-RUSSISCHEN KRIEGE. Deut. Zschr. f. Chir. Leipz. *121:* 199–230, 1913.
42. Elvidge, A. R.: THE POSTTRAUMATIC CONVULSIVE AND ALLIED STATES. *In Brock, S.* (ed.): *INJURIES OF SKULL, BRAIN AND SPINAL CORD,* Baltimore, Williams & Wilkins Co., 1940.
43. Ettlinger, G., Jackson, C. V., and Zangwill, O. L.: CEREBRAL DOMINANCE IN SINISTRALS, Brain *79*: 569–588, 1956.

44. Falk, B., and Silfverskiöld, B. P.: PNEUMOENCEPHALOGRAPHIC CHANGES IN THE CHRONIC POSTCONCUSSION SYNDROME AND NON-TRAUMATIC CEPHALALGIA, Acta Psychiat. et Neurol. Scandinav. *29*:161–171, 1954.
45. Faust, C.: ZUR SYMPTOMATIK FRISCHER UND ALTER STIRNHIRNVERLETZUNGEN. ZUGLEICH EIN BEITRAG ZUR POSTTRAUMATISCHEN, ORGANISCHEN WESENSVERÄNDERUNG, Arch. Psychiat., Berl. *193*:78–97, 1955.
46. Fischer, M.: ENCEPHALOGRAPHISCHE BEFUNDE BEI SCHÄDELVERLETZUNGEN, Arch. f. Psychiat. Berl. *82*:403–421, 1927.
47. Friedman, A. P., Brenner, C., and Carter, S.: SYMPTOMATIC TREATMENT OF CERTAIN TYPES OF CHRONIC HEADACHE, J. A. M. A. *139*:195–198, 1949.
48. Friedman, E. D.: HEAD INJURIES: EFFECTS AND THEIR APPRAISAL; ENCEPHALOGRAPHIC OBSERVATIONS, A. M. A. Arch. Neurol. & Psychiat. *27*:791–810, 1932.
49. Friedmann, M.: ÜBER EINE BESONDERE SCHWERE FORM VON FOLGEZUSTÄNDEN NACH GEHIRNERSCHÜTTERUNG UND ÜBER DEN VASOMOTORISCHEN SYMPTOMENCOMPLEX BEI DERSELBEN IM ALLGEMEINEN, Arch. f. Psychiat. Berl. *23*:230–267, 1891.
50. Gamberini, C.: SULL 'EPILESSIA POSTTRAUMATICA, Bologna, L. Capelli, 1921. (Referred to by Krause & Schum, p. 45.)
51. Gardner, W. J.: CLOSURE OF DEFECTS OF SKULL WITH TANTALUM, Surg. Gynec. & Obst. *80*:303–312, 1945.
52. Gibbs, F. A., Wegner, W. R., and Gibbs, E. L.: ELECTROENCEPHALOGRAM IN POST-TRAUMATIC EPILEPSY, Am. J. Psychiat. *100*:738–749, 1944.
53. Glasson, W. H.: HISTORY OF MILITARY PENSION LEGISLATION IN THE UNITED STATES. V.13 STUDIES IN HISTORY, ECONOMICS, AND PUBLIC LAW, New York, The Columbia Univ. Press, 1900.
54. Gliddon, W. O.: GUNSHOT WOUNDS OF THE HEAD (REVIEW OF AFTER-EFFECTS IN 500 CANADIAN PENSIONERS FROM GREAT WAR, 1914–1918), Canad. M. A. J. *49*:373–377, 1943.
55. Goldstein, K.: AFTER-EFFECTS OF BRAIN INJURIES IN WAR; THEIR EVALUATION AND TREATMENT; THE APPLICATION OF PSYCHOLOGIC METHODS IN THE CLINIC, New York, Grune and Stratton, 244 pp. 1942.
56. Gordon, N.: POST-TRAUMATIC VERTIGO WITH SPECIAL REFERENCE TO POSTURAL NYSTAGMUS, Lancet *1*:1216–1218, 1954.
57. Grant, F. C., and Norcross, N. C.: REPAIR OF CRANIAL DEFECTS BY CRANIOPLASTY, Ann. Surg. *110*:488–512, 1939.
58. Grantham, E. G., and Landis, H. P.: CRANIOPLASTY AND THE POST-TRAUMATIC SYNDROME, J. Neurosurg. *5*:19–22, 1948.

59. Grove, W. E.: OTOLOGIC OBSERVATIONS IN TRAUMA OF THE HEAD; A CLINICAL STUDY BASED ON 42 CASES, Arch. Otolaryngol. *8*:249–299, 1928.

60. Halstead, W. C.: BRAIN AND INTELLIGENCE. A QUANTITATIVE STUDY OF THE FRONTAL LOBES, Chicago, Univ. Chicago Press, XIII, 206 pp. 1947.

61. Hammond, W. A.: A TREATISE ON THE DISEASES OF THE NERVOUS SYSTEM, 7th ed., New York, D. Appleton & Co., 1881.

62. Hauptmann, A.: DIE OBJEKTIVIERUNG POSTKOMMOTIONELLER BESCHWERDEN DURCH DAS ENCEPHALOGRAMM, Arch. Psychiat. *96*:84–94, 1932.

63. Heppenstall, M. E., and Hill, D.: ELECTROENCEPHALOGRAPHY IN CHRONIC POST-TRAUMATIC SYNDROMES, Lancet *1*:261–263, 1943.

64. Hilterhaus, H., and Bayer, S. L.: ÜBER EEG.—VERÄNDERUNGEN NACH ALTEN SCHÄDELTRAUMEN, Zentrabl. Neurochir. *15*:93–98, 1955.

65. Hines, F. T.: MEDICAL CARE PROGRAM OF VETERANS ADMINISTRATION, Ann. Am. Acad. Polit. and Social Sc. *239:* 73–79, 1945.

66. Holbeck, O.: DIE SCHUSSVERLETZUNGEN DES SCHÄDELS IM KRIEGE. BEOBACHTUNGEN UND ERFAHRUNGEN WÄHREND DES RUSSISCH-JAPANISCHEN KRIEGES 1904–1905, 10 pl. Berlin, A. Hirschwold, 1912.

67. Hollerius, Jacobus: OMNIA OPERA PRACTICA, DOCTISSIMIS EJUSDEM SCHOLIIS ET OBSERVATIONBUS ILLUSTRATA: DEINDE LUD. DURETI, IN ENUNDEM ENARRATIONIBUS ANNOTATIONIBUS, ET. ANTONII VALETII. ACCESSIT ETIAM AD CALCEM LIBRI THERAPIA PUERPERARUM I. LE BON. LIBRI DUO IN VOLUMEN UNUM CONJUNCTI. 7, p. 1., 584, 9 1 roy 80, Genevae, J. Stoer., 1623.

68. Hooper, R. S.: ORBITAL COMPLICATIONS OF HEAD INJURY, Brit. J. Surg. *39:* 126–138, 1951.

69. Jasper, H., and Penfield, W.: ELECTROENCEPHALOGRAMS IN POSTTRAUMATIC EPILEPSY; PRE-OPERATIVE AND POST-OPERATIVE STUDIES, Am. J. Psychiat. *100:* 365–377, 1943.

70. Kaufman, I. C., and Walker, A. E.: ELECTROENCEPHALOGRAM AFTER HEAD INJURY, J. Nerv. and Ment. Dis. *109:* 383–395, 1949.

71. Kellaway, P.: HEAD INJURY IN CHILDREN, Electroencephalog. & Clin. Neurophysiol. *7:* 497–502, 1955.

72. Kennedy, F.: HEAD INJURIES: EFFECTS AND THEIR APPRAISAL; EVALUATION OF EVIDENCE, Arch. Neurol. & Psychiat. *27:* 811–814, 1932.

73. Klaue, R.: ZUR BEURTEILUNG HIRNTRAUMATISCHER FOLGEZUSTÄNDE NACH STUMPFEM SCHÄDELTRAUMA MIT BESONDERER BERÜCKSICHTIGUNG ENCEPHALOGRAPHISCHER BEFUNDE, Deutsche Ztschr. Nervenh. *164:* 259–302, 1950.

74. Krause, F., and Schum, H.: DIE SPEZIELLE CHIRURGIE DER GEHIRNKRANKHEITEN. 2: DIE EPILEPTISCHEN ERKRANKUNGEN, Neue Deut. Chir., Stuttgart, F. Enke, 49a. 1931.

75. Landolt, E.: ZUR OPTICUSSCHÄDIGUNG BEI SCHÄDELTRAUMA, Acta neurochir. *4:* 128–142, 1955.

76. Laufer, M. W., and Perkins, R. F.: STUDY OF ELECTROENCEPHALOGRAPHIC FINDINGS IN 209 CASES ADMITTED AS HEAD INJURIES TO ARMY NEUROLOGICAL-NEUROSURGICAL CENTER, Proc. Neuropsychiat. Conf., Sixth Serv. Command, pp. 118–124, 1945.

77. Leigh, A. D.: DEFECTS OF SMELL AFTER HEAD INJURY, Lancet *1:* 38–40, 1943.

78. Leonhardt, W.: HIRNTRAUMA UND EXTRAPYRAMIDALE ERKRANKUNGEN, Fortschr. Neurol., Psychiat. u. *21:* 341–354, 1953.

79. Leuret, M.: RECHERCHES SUR L'ÉPILEPSIE. Arch Gén. Méd. *2:* 32–50, 1843.

80. Linthicum, F. H., and Rand, C. W.: NEURO-OTOLOGICAL OBSERVATIONS IN CONCUSSION OF THE BRAIN, Arch. Otolaryngol. *13:* 785–821, 1931.

81. Lynn, J. G., Levine, K. N., and Hewson, L. R.: PSYCHOLOGIC TESTS FOR THE CLINICAL EVALUATION OF LATE "DIFFUSE ORGANIC," "NEUROTIC," and "NORMAL" REACTIONS AFTER CLOSED HEAD INJURY, A. Research Nerv. & Ment. Dis. Proc. *24:* 296–378, 1945.

82. Maltby, G. L.: PENETRATING CRANIOCEREBRAL INJURIES; EVALUATION OF LATE RESULTS IN A GROUP OF 200 CONSECUTIVE PENETRATING CRANIAL WAR WOUNDS, J. Neurosurg. *3:* 239–249, 1946.

83. Marburg, O.: DIE TRAUMATISCHEN ERKRANKUNGEN DES GEHIRNS UND RÜCKENMARKS, In Handb. Neurol. *11:* 1–177, 1936. (Bumke & Foerster), Berlin, 1936.

84. McConnell, A. A.: PROLONGED POST-TRAUMATIC AMNESIA FINDINGS AT OPERATION, Lancet *1:* 273–274, 1944.

85. McConnell, A. A.: ON CERTAIN SEQUELAE OF CLOSED HEAD INJURIES; PATHOLOGICAL BASIS OF "POST-TRAUMATIC SYNDROME," Brain *76:* 473–484, 1953.

86. McConnell, A. A.: CEPHALIC POST-TRAUMATIC SYNDROME. PATHOLOGICAL OBSERVATIONS, J. Ment. Sc. *102:* 330–335, 1956.

87. Meyer, J. E.: DIE SEXUELLEN STÖRUNGEN DER HIRNVERLETZTEN, Arch. Psychiat. *193:* 449–469, 1955.

88. Meyer-Mickeleit, R.: THE EEG IN CLOSED HEAD INJURIES, Electroenceph. Clin. Neurophysiol. *5:* 119, 1953.
89. Meyer-Mickeleit, R. W., and Schneider, E.: TRAUMATIC EPILEPSY IN CHILDREN AND ADOLESCENTS AND THEIR RELATION TO EPILEPSY, Electroenceph. Clin. Neurophysiol. *9:* 349–350, 1957.
90. Minkowski, M.: UNFALLNEUROSE, TRAUMATISCHE ENZEPHALOPATHIE ODER KOMBINIERTER ORGANISCH-NEUROTISCHER FOLGEZUSTAND NACH TRAUMA, Schweiz. Arch. f. Neurol. u. Psychiat. *27:* 108–124, 1931.
91. Mygind, S. H.: TRAUMATIC VESTIBULAR DISEASES, Acta Otolaryng. (Stockh) *1:* 515–526, 1918.
92. Nielsen, J. M., and Butler, F. O.: BIRTH PRIMACY AND IDIOPATHIC EPILEPSY, Bull. Los Angeles Neurol. Soc. *13:* 176–178, 1948.
93. Orr, J. K., and Risch, F.: IS THE ORDER OF BIRTH A FACTOR IN EPILEPSY? Neurology *3:* 679–683, 1953.
94. Penfield, W.: CHRONIC MENINGEAL POST-TRAUMATIC HEADACHE, AND ITS SPECIFIC TREATMENT BY LUMBAR AIR INSUFFLATION; ENCEPHALOGRAPHY, Surg. Gynec. & Obst. *45:* 747–759, 1927.
95. Penfield, W., and Erickson, T. C.: EPILEPSY AND CEREBRAL LOCALIZATION; A STUDY OF THE MECHANISM, TREATMENT AND PREVENTION OF EPILEPTIC SEIZURES, Springfield, Ill., Charles C. Thomas, 1941.
96. Penfield, W., and Shaver, M.: INCIDENCE OF TRAUMATIC EPILEPSY AND HEADACHE AFTER HEAD INJURY IN CIVIL PRACTICE, A. Research Nerv. & Ment. Dis. Proc. (1943) *24:* 620–634, 1945.
97. Pennington, L. A., and Mearin, R. J.: FREQUENCY OF CRANIOCEREBRAL INJURIES IN RELATION TO MILITARY SCREENING EXAMINATIONS, War Med. *4:* 465–470, 1943.
98. Phillips, G.: TRAUMATIC EPILEPSY AFTER CLOSED HEAD INJURY, J. Neur., Lond. *17:* 1–10, 1954.
99. Puech, P., Brun, M., Lairy-Bounes, G. C., Morice, J., and Perrin, J.: TRAUMATISMES CRANIO-CÉRÉBRAUX, Paris, A. Legrand & Cie., 1950.
100. Rawling, L. B.: GUNSHOT WOUNDS OF THE HEAD, Brit. J. Surg. *10:* 93–126, 1922.
101. Redlich, E.: ZUR PATHOLOGIE DER EPILEPSIE NACH SCHÄDELSCHUSSVERLETZUNGEN, Zschr. f.d. ges Neurol. und Psychiat. *48:* 8–110, 1919.
102. Roseman, E., and Woodhall, B.: ELECTROENCEPHALOGRAM IN WAR WOUNDS OF THE BRAIN, WITH PARTICULAR REFERENCE TO POST-TRAUMATIC EPILEPSY, A. Research Nerv. & Ment. Dis. Proc. (1944) *25:* 200–219, 1946.
103. Röttgen, P.: ERFAHRUNGEN AN FRISCHEN UNKOMPLIZIERTEN HIRNWUNDEN, Arch. klin. Chir. *274:* 388–396, 1953.

104. Roussy, G., and Lhermitte, J.: THE PSYCHONEUROSES OF WAR. Tr. W. B. Christopherson, London, Univ. Press, 1918; Paris, Masson & Co., 1918.
105. Rowbotham, G. F.: ACUTE INJURIES OF THE HEAD, THEIR DIAGNOSIS, TREATMENT, COMPLICATIONS AND SEQUELS, Baltimore, Williams & Wilkins Co., 1945.
106. Ruesch, J., and Bowman, K. M.: PROLONGED POST-TRAUMATIC SYNDROMES FOLLWING HEAD INJURY, Am. J. Psychiat. *102:* 145–163, 1945.
107. Russell, W. R.: CEREBRAL INVOLVEMENT IN HEAD INJURY. A STUDY BASED ON THE EXAMINATION OF TWO HUNDRED CASES, Brain *55*:549–603, 1932.
108. Russell, W. R.: MEDICAL ASPECTS OF HEAD INJURY, Brit. M. J. *2*:521–523, 1942.
109. Russell, W. R.: THE ANATOMY OF TRAUMATIC EPILEPSY, Brain *70*:225–233, 1947.
110. Russell, W. R.: DISABILITY CAUSED BY BRAIN WOUNDS; REVIEW OF 1,166 CASES, J. Neurol., Neurosurg. and Psychiat. *14*:35–39, 1951.
111. Russell, W. R., and Whitty, C. W. M.: STUDIES IN TRAUMATIC EPILEPSY; FACTORS INFLUENCING INCIDENCE OF EPILEPSY AFTER BRAIN WOUNDS, J. Neurol., Neurosurg. and Psychiat. *15*:93–98, 1952.
112. Sands, H., and Price, J. C.: A PATTERN ANALYSIS OF THE WECHSLER-BELLEVUE ADULT INTELLIGENCE SCALE IN EPILEPSY, Res. Publ. Ass. Nerv. Ment. Dis. *26*:604–615, 1946.
113. Sargent, P.: SOME OBSERVATIONS ON EPILEPSY, Brain *44*:312–328, 1921.
114. Schilder, P.: NEUROSES FOLLOWING HEAD AND BRAIN INJURIES. *In Brock, S.* (ed.): *INJURIES OF SKULL, BRAIN AND SPINAL CORD,* Baltimore, Williams & Wilkins Co., 1940.
115. Schwab, O.: ENCEPHALOGRAPHIE, LIQUORPASSAGE—UND LIQUORRESORPTIONSPRÜFUNGEN IM DIENSTE DER BEURTEILUNG VON SOGENANNTEN COMMOTIONS-NEUROSEN, Ztschr. f. d. ges. Neurol. u. Psychiat. *102*:294–312, 1926.
116. Steinthal K: TRAUMATISCHE EPILEPSIE NACH HIRNSCHÜSSEN UND BERUFLICHE LEISTUNGSFÄHIGKEIT, München med. Wchnschr. *73*:1393–1395, 1926.
117. Steinthal, K.: DIE EPILEPSIE INSBESONDERE DIE TRAUMATISCHE EPILEPSIE UND DIE ERGEBNISSE IHRER CHIRURGISCHEN BEHANDLUNG, Ergebn. d. Chir. u. Orthop. *22*:222–257, 1929.
118. Steinthal, K., and Nagel, H.: DIE LEISTUNGSFÄHIGKEIT IM BÜRGERLICHEN BERUF NACH HIRNSCHÜSSEN MIT BESONDERER BERÜCKSICHTIGUNG DER TRAUMATISCHEN EPILEPSIE, Beitr. z. Klin Chir. *137:* 361–400, 1926.
119. Stevenson, W. E.: EPILEPSY AND GUNSHOT WOUNDS OF THE HEAD, Brain *54*:214–224, 1931.

120. Strauss, I., and Savitsky, N.: HEAD INJURY. NEUROLOGIC AND PSYCHIATRIC ASPECTS, A. M. A. Arch. Neurol. & Psychiat. *31*:893–955, 1934.

121. Swift, G. W.: HEAD INJURIES OF MODERATE DEGREE; A REVIEW OF ONE HUNDRED CASES INCLUDING FIFTY VENTRICULAR STUDIES, Surg. Gynec. & Obst. *52*:576–578, 1931.

122. Symonds, C. P.: CONCUSSION AND CONTUSION OF THE BRAIN AND THEIR SEQUELAE. *In Brock, S.* (ed.): *INJURIES OF SKULL, BRAIN AND SPINAL CORD,* Baltimore, Williams & Wilkins Co., pp. 69–111, 1940.

123. Symonds, C. P., and Lewis, A.: DISCUSSION ON DIFFERENTIAL DIAGNOSIS AND TREATMENT OF POST-CONTUSIONAL STATES, Proc. Roy. Soc. Med. *35*:601–614, 1942.

124. Tilmann, Prof. von: ZUR PATHOGENESE DER EPILEPSIE, Virchows Arch. f. Path. Anat. *229*:40–60, 1920.

125. Tönnis, W.: VERLETZUNGEN IN M. KIRSCHNER AND O. NORDMANN (ED.): DIE CHIRURGIE. EINE ZUSAMMENFASSENDE DARSTELLUNG DER ALLGEMEINEN UND DER SPEZIELLEN CHIRURGIE. *3*:801–880, 1948.

126. Troland, C. E., Baxter, D. H., and Schatzki, R.: OBSERVATIONS ON ENCEPHALOGRAPHIC FINDINGS IN CEREBRAL TRAUMA, J. Neurosurg. *3*:390–398, 1946.

127. Turner, J. W. A.: INDIRECT INJURIES OF THE OPTIC NERVE, Brain *66*:140–151, 1943.

128. Usbeck, W.: UEBER STATISTICHE ERHEBUNGEN BEI UNSEREN ALTEN OFFENEN UND GEDECKTEN HIRNVERLETZUNGEN, Psychiat. Neur. med. Psychol., Lpz. *6*:300–306, 1954.

129. Valescus de Tharanta: PHILONIUM. AUREUM AC PERUTILE OPUS PRACTICE MEDICINE OPERAM DANTIBUS: PHILONIUM APPELLATUR, CONSUMATISSIMI MEDICI. Venice, Heirs of Octavianus Scotus, 1521.

130. Vogeler, K.: DAS SPÄTSCHICKSAL DER SCHÄDELSCHUSSVERLETZTEN, Deutsche Ztschr. Chir. *234*:245–300, 1931.

131. von Bergmann, E.: DIE LEHRE VON DEN KOPFVERLETZUNGEN, Stuttgart, F. Enke, 1880.

132. Wagstaffe, W. W.: THE INCIDENCE OF TRAUMATIC EPILEPSY AFTER GUNSHOT WOUND OF THE HEAD, Lancet *2:* 861–862, 1928.

133. Walker, A. E.: A HISTORY OF NEUROLOGICAL SURGERY, Baltimore, Williams and Wilkins Co., 1951.

134. Walker, A. E.: PROGNOSIS IN POST-TRAUMATIC EPILEPSY. A TEN-YEAR FOLLOW-UP OF CRANIOCEREBRAL INJURIES OF WORLD WAR II, J. A. M. A. *164*:1636–1641, 1957.

135. Walker, A. E., and Jablon, S.: A FOLLOW-UP OF HEAD-INJURED MEN OF WORLD WAR II, J. Neurosurg. *16*:600–610, 1959.

136. Watson, C. W.: INCIDENCE OF EPILEPSY FOLLOWING CRANIOCEREBRAL INJURY; THREE-YEAR FOLLOW-UP STUDY, A. M. A. Arch. Neurol. & Psychiat. *68*:831–834, 1952.
137. Watts, J. W., Wiley, W. B., and Groh, R. H.: RELATION OF CONTUSION OF SCALP TO POST-TRAUMATIC HEADACHE AND DIZZINESS, A. Research Nerv. & Ment. Dis. Proc. (1943) *24*:562–577, 1945.
138. Williams, D.: ELECTROENCEPHALOGRAM IN CHRONIC POST-TRAUMATIC STATES, J. Neurol. & Psychiat. *4*:131–146, 1941.
139. Wilson, D. M.: HEAD INJURIES IN SERVICEMEN OF 1939–1945 WAR, N. Zealand M. J. *50*:383–391, 1951.

Appendix 1

POSTTRAUMATIC HEADACHE

by

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A. INTRODUCTION

Chronic headache as a sequela of head injury may follow all types and degrees of head trauma. The incidence of headaches following head injuries varies in the literature from 28 percent to 65 percent. As emphasized by Denny-Brown (36) and his associate (1), it is evident that the complaints of the patient following head injury will vary not only with the manner and degree of injury but with the personality structure, compensation factors, and the environmental status.

The present report is based on a series of 169 patients with posttraumatic headache seen at the Montefiore Hospital as a part of the follow-up study. More than one-half of the patients were of Latin and Semitic extractions, and less than 1 percent (only 1) was Negro. The family histories were not significant in their psychiatric aspects. Only five (3 percent) gave a history of any mental illness. From our inquiries, 146 patients (75 percent) had no neurotic or habit disturbances in infancy or childhood; enuresis and nail biting were reported by 12 patients (7 percent).

One hundred and thirty-five patients (80 percent) were in active combat at the time of trauma, and 22 (13 percent) were in a combat area.

During the first week following injury, 71 patients (42 percent) had no complaints, 17 (10 percent) were torpid, 9 (5 percent) were confused, and 5 (3 percent) were stuporous.

Some 152 patients (90 percent) had fair or good school adjustment. Of this group, 44 (26 percent) were high school graduates, 59 (35 percent) had 1 or 2 years of high school, 20 (12 percent) had 1 or more years of college, and 11 (7 percent) had professional training.

In this series it was found that 159 patients (94 percent) were headache-free prior to injury. Of the 10 (6 percent) who had pretraumatic headaches, 7 (4 percent) were classified as suffering from tension headaches. The onset of headaches was immediate in 25 patients, within 1 week in another 25, and within 1 month following injury in another, a total of 93 men (55 percent). Ten percent of the total group developed headache between 1 and 6 months and 13 percent after the sixth month and up to the fifth year. In the others, the time of onset is unknown. In over 50 percent of the patients (86), the onset of headache was abrupt.

In 118 patients (70 percent) the duration of posttraumatic headache was over 5 years. In eight (5 percent) it was less than 2 months. In 85 (50 percent) severity of headache was indicated as moderate, in 42 (25 percent) it was severe, and in the remaining 25 percent it was variable.

Thirty-four patients (20 percent) had generalized headaches, 56 (33 percent) had localized polar headaches (bifrontal, bitemporal, etc.), 56 (33 percent) had localized unilateral headaches, while the others were variously located.

The most predominant quality was "dull and pressing," but the headaches varied from patient to patient and in each patient, and were described as sharp, pressing, stabbing, aching, burning, etc. Fifty-one (30 percent) had pain localized to the site of scalp injury.

One hundred and thirty-five patients (80 percent) reported no spread of their headache, while 15 percent reported spread to the head, neck, or upper face.

B. DURATION AND FREQUENCY

In 12 patients (7 percent) the headaches were constant, and of the remaining 157 (93 percent) with periodic headaches, 51 (30 percent of the entire group) complained of one or more attacks per day, and 90 (53 percent) reported a minimum of 1 to 4 attacks per month. In the remaining group (10 percent) the attacks occurred approximately three times a year. In those patients with periodic headaches, attacks lasted from 30 minutes to 4 hours in approximately half of the patients, and less than 30 minutes in 10 percent.

C. PRODROMATA

No prodromata were noted in 159 patients (94 percent). In the remaining 10 the most common complaints were visual (i.e., scotoma, photophobia, blurring), gastric (nausea and anorexia), and emotional (difficulty in concentration, irritability, etc.).

D. ASSOCIATED MANIFESTATIONS

During an attack of headache the most common associated manfestation was photophobia, which was noted by 37 patients (22 percent). Thirty-four (20 percent) complained of gastric disturbances, i.e., anorexia or nausea, and of this group 12 (7 percent) had vomiting. Neurological complaints as associated manifestations were not noteworthy; a few complained of tinnitus. Twenty-five (15 percent) complained of vasomotor phenomena, which included chilly feelings, sweating, coldness, warmth, etc. Fifty-four (32 percent) did not have any associated mental or emotional symptoms with their headaches. Of the remaining 115, 106 reported irritability, 52 had difficulty in concentration, and 35 reported anxiety with depression. Of the group, 110 men (65 percent) had dizziness without true vertigo associated with the headache or as part of the posttraumatic syndrome. Such feelings were described as "unsteadiness" by a majority of the patients, but attacks of dizziness did not consistently occur with the headache, even in these men. Thirty percent of the patients were free of such symptoms and 5 percent had both dizziness and vertigo. Other associated manifestations were not particularly significant except that 34 (20 percent) reported general weakness and hypersensitivity to noise.

E. TERMINATION OF ATTACKS

One hundred and fifty-six patients (92 percent) reported that they had no symptoms accompanying the end of the headaches. The remaining 13 (8 percent) had a variety of complaints, including sleepiness, frequency of urination, etc.

F. CONTRIBUTING FACTORS

One hundred and one patients (60 percent) reported that mental or physical effort or fatigue precipitated the headache. Change in temperature and weather was a predominant physical factor which the patients said was responsible for precipitating their attacks. In one-half of the patients emotional disturbances alone were responsible for producing an attack. One hundred and eight patients (64 percent) reported that rest and quiet would relieve their attacks, and 60 (15 percent) reported that lying down gave relief. In a small number of patients relief was reported after eating, mental effort, or application of heat. One-third of the patients reported that no specific maneuver helped their headaches. In one-half of the patients, headaches were aggravated by movement of the head.

G. RESPONSE TO TREATMENT

Only 22 patients (13 percent) received psychotherapy as prophylactic treatment for their headaches. Fifteen failed to respond, and 7 had moderate success.

Symptomatic Treatment

Coal tar derivatives were moderately effective in approximately 60 percent of the patients. Opiates, used in a few patients, gave good results. Sedatives, which were used in a very small number of patients, were effective in only one-third. There are no data available on the use of combinations of sedatives and coal tar derivatives. Data on prophylactic treatment by pharmacological methods were not available.

Appendix 2

CODE SHEETS

A detailed code for the acute injury was printed in full detail on several code sheets, both to facilitate statistical processing and to provide the follow-up examiner with the essential data abstracted from Army clinical records. The code sheets themselves, with their detailed classifications, are too bulky to reproduce but the information they contain is outlined below. The more important classifications in the code appear in tables throughout the text.

Outline of Code for Acute Injury

I. Case number and race

II. The wound

- A. Age in years at injury
- B. Type of wound
- C. Agent
- D. Most important single head wound
  - Location
    - Regions wounded
    - Entrance
      - Region
      - Side
    - Exit or lodgement
      - Region
      - Side
    - Mean diameter of cranial defect
    - Depth
- E. Second head wound
  - Location
    - Regions wounded
    - Entrance
      - Region
      - Side
    - Exit or lodgement
      - Region
      - Side
    - Mean diameter of cranial defect
    - Depth
- F. Location of other wounds
- G. Immediate complications
- H. Period of unconsciousness
- I. Period of confusion
- J. Neurological deficit at time of wound

III. Debridement

A. First debridement done
   1. Time from injury
   2. Chemotherapy
      Drug
      Mode

B. First debridement reported
   1. Operator's MOS and level of training
   2. Time from injury
   3. Extent
   4. Intracranial foreign bodies removed
   5. Closure
      Scalp
      Dural
   6. Chemotherapy
      Drug
      Mode
   7. Complications
   8. Healing

C. Second debridement

D. Third debridement

IV. Cranioplasty

A. Time after injury

B. Time of cranioplasty relative to debridement

C. Healing and infection after cranioplasty

D. Type of plate

E. Convulsions following cranioplasty

F. Occurrence of epilepsy relative to time of cranioplasty

G. Further cranioplasty

V. X-rays (Army only)

A. Result of intracranial X-rays

B. Time of injury to last Army X-ray

VI. Epilepsy in service

A. Time from injury to first episode recorded

B. Typical episodes
   1. Type of attack
   2. Aura
   3. Post-ictal phenomena

C. Petit mal attacks

D. Grand mal attacks

E. Anti-convulsive medications in 3-month periods after injury

VII. Cortical resection

VIII. Discharge from service

A. Time from injury to discharge
B. Neurological symptoms at discharge
C. Neurological abnormalities at discharge
D. Reason for discharge

IX. Pneumoencephalograms

A. Time from injury to each PEG (1st, 2d, 3d)
B. Type of each PEG (1st, 2d, 3d)
C. Time since last PEG (1st to 2d, 2d to 3d)
D. Change between PEGs (1st to 2d, 2d to 3d)

X. Electroencephalograms

A. Number of EEGs made
B. EEG within 6 months of injury
    1. Time from injury to EEG
    2. Type
    3. General characteristics
    4. Generalized abnormalities
    5. Focal abnormalities
        a. Location
        b. Type
    6. Activated EEG
    7. Focal activated changes
        a. Location
        b. Side
C. Six to 12 months after injury
    (Same as B)
D. Twelve to 24 months after injury
    (Same as B)
E. More than 24 months after injury
    (Same as B)

XI. Cortical operations

A. Cause
B. Type
C. Findings
D. Type of cortical procedure
E. Dural repair
F. Cranial repair
G. Postoperative healing
H. Postoperative sequelae
I. Postoperative neurological state
    1. Convulsions
    2. Status
J. Time from injury

Appendix 3

EXAMINATION BOOKLET

The examiner was furnished with a booklet in which he entered the results of his examination. The booklet was designed on the basis of a draft code, so that his entries might have the desired specificity and he might have the responsibility for making the proper choice of alternative classifications. At the right of each page the examiner was given a generous blank space in which to write explanatory notes clarifying his entries, etc. Only after all the booklets had been received and edited by both authors could the booklet be amended to constitute a technically adequate code for punch-card use. Such amendments may be exemplified as follows: (*1*) a single-choice classification is changed to a multiple-choice one; (*2*) an additional combination of findings is recognized in the classification; (*3*) a central review (e.g., EEG) requires a new classification of its own; (*4*) a classification is extended to include findings not originally recognized, and (*5*) a center makes some additional type of observation that requires its own classification.

As used, the booklet for the follow-up examination extended to 47 pages and has seemed too detailed for reproduction here except in outline form. As in appendix 2, therefore, the outline specifies every item of information obtained but provides none of the detail of the classification of information applied to each item. However, many of the more important classifications appear in the tables throughout the text.

Outline of Booklet for Follow-up Examination

I. Identification
- A. Center and type of roster
- B. Study number within center, and race

XII. Time from injury to follow-up examination

XIII. Personal history
- A. Birth history
- B. Birth number in family
- C. Development
- D. Previous head injuries
- E. Previous illnesses
- F. Immediate family
  1. Number in family
  2. Number of epileptics in family
- G. Relatives
  1. Number of aunts, uncles, and first cousins
  2. Number of aunts, uncles, and first cousins that were epileptics

H. Other nervous disorders in immediate family, excluding epileptics and self
I. Alcohol habits of patient
J. Constipation

XIV. Rehabilitation

A. Work status at time of report
B. General work progress
C. Marital status during period from injury
D. Children
E. Mode of patient's living
F. Home adjustment
G. Social adjustment
H. Economic adjustment

XV. Clinical symptomatology

A. General
B. Speech
C. Visual
D. Auditory
E. Somatosensory
F. Motor
G. Olfactory
H. Sexual
I. Abnormalities of other systems

XVI. Neurological examination

A. General summary
B. Mental status
C. Memory
D. Serial 7 test
E. Aphasia
F. Apraxia

XVII. Cranial nerves

A. General
B. Olfaction
C. Vision
    1. General
    2. Funduscopic
    3. Extraocular movements
    4. Pupils
D. Face
    1. Facial sensation
    2. Facial movement
E. Hearing
F. Swallowing

XVIII. Motor status

- A. Power
- B. Tone
  - 1. Contralateral side
  - 2. Ipsilateral side
- C. Atrophy
- D. Coordination
- E. Alternating movements and general status
- F. Grip dynamometer
  - 1. Right
  - 2. Left

XIX. Sensory status

- A. Superficial: pain or touch or temperature
- B. Deep: position or vibratory or 2 point
- C. Stereognosis

XX. Reflex status

- A. Tendon reflexes
- B. Abdominal reflexes
- C. Plantar reflexes

XXI. Miscellaneous

- A. Station and gait
- B. Handedness and side wounded
- C. Scalp and skull
- D. Summary of neurological examination
- E. Summary of physical examination

XXII. Epilepsy since injury

- A. Incidence of attacks
- B. Type of attacks
- C. Aura
- D. Post-ictal phenomena
- E. Number of minor or focal attacks in each 12-month period after injury
- F. Number of grand mal attacks in each 12-month period after injury
- G. Anti-convulsive medication in each 12-month period after injury

XXIII. Summary of electroencephalogram

- A. Hospital EEG number
- B. Time from injury
- C. Type
- D. General characteristics
- E. Generalized abnormalities
- F. Focal abnormalities
  - 1. Location
  - 2. Type

G. Activated EEG
H. Focal activated changes
    1. Location
    2. Side

XXIV. Wechsler-Bellevue Intelligence Scale

A. Time
B. General IQ
C. Percentage deterioration
D. General information
E. Comprehension
F. Arithmetic
G. Digit span
H. Similarities
I. Vocabulary
J. Picture arrangement
K. Picture completion
L. Object assembly
M. Block design
N. Digit symbols

XXV. Minnesota Multiphasic Personality Inventory

A. Time required
B. Query score
C. Lie score
D. K score
E. F (validity) score
F. Profile chart
    1. Hs scale
    2. D scale
    3. Hy scale
    4. Pd scale
    5. Mf scale
    6. Pa scale
    7. Pt scale
    8. Sc scale
    9. Ma scale

XXVI. Fluoroscopy of skull

XXVII. Visual fields, results

XXVIII. Goddard Tactual Performance Test

A. Time in minutes for test
B. Dominant hand, time
C. Recessive hand, time
D. Both hands, time
E. Memory score (drawing)
F. Location score

Appendix 4

LETTERS TO VETERANS

FORM LETTER NO. 1

National Research Council
Project 4
Building 113
Veterans Administration Center
Los Angeles 25, California

Mr. John Smith
1234 Fifth Street
City, State

Dear Mr. Smith: The National Research Council is conducting a study of the present condition of a selected group of men who were hospitalized during military service for head injuries.

It is hoped to learn through this study many things about this type of injury, such as the extent to which they improve as time goes on, how effective different kinds of treatment have been, and so on.

This study is a purely medical one and has nothing to do with any of your relationships with the Veterans Administration. All of the results will be kept strictly confidential by the National Research Council, and no information about any single individual will be released unless you give written consent.

I have been asked to check thoroughly your present condition to see how you are. This will involve no cost to you and does not require your admission to the hospital. You will be reimbursed for the expense you incur in travel (coach rate) and for your lunch.

Would you be kind enough to call my office, Arizona 12345, for a definite appointment sometime before _____, or fill in the enclosed card telling me when it would be convenient for you to come in. My hours are from 9 a.m. to 4 p.m., Monday through Friday. However, a special appointment can be made for Saturday. Parking facilities are adjacent to my office at a distance of about 50 yards and there are two steps into the building. The examination will take from four to six hours including X-rays of the skull.

The results of my examination will be as confidential as if it had been done by your family doctor. I shall be pleased to discuss your condition with you and the results of our examination when completed, if you so desire. Of course, there is to be no charge for this examination.

Sincerely yours,

J. Brown, M.D.

Enclosure.

FORM LETTER NO. 2

National Research Council
Project 4
Building 113
Veterans Administration Center
Los Angeles 25, California

Mr. John Smith
1234 Fifth Street
City, State

Dear Mr. Smith: I have had no reply to my letter of ----------, sent to you regarding the follow-up examinations which I am conducting on veterans with head injuries.

An encouraging number of men have already responded to this invitation and have been quite satisfied with the results. This examination, as you know, is for the purpose of determining what effect the injury may have had on you over a period of time, the effectiveness of the treatment you had, and what has been your adjustment to this.

I should like to remind you that this examination will have no relation to your VA compensation rating and that you will be reimbursed for your transportation and lunch. Nor does it require your admission to the hospital.

It is my feeling that this investigation will be of considerable value to you. I shall be glad to answer any questions about the medical findings and discuss your condition with you. Of course, there is no charge for this.

May I then urge you to make an appointment? Write me or call Arizona 12345, and I am sure that a time can be arranged that is suitable to you. I am enclosing a card for your convenience and reply.

Sincerely yours,

J. Brown, M.D.

Enclosure.

FORM LETTER NO. 3

NATIONAL RESEARCH COUNCIL
PROJECT 4
Building 113
Veterans Administration Center
Los Angeles 25, California

Mr. JOHN SMITH
1234 Fifth Street
City, State

DEAR MR. SMITH: The National Research Council study of veterans who had head injuries during World War II is in progress, and I should like to remind you that the opportunity still remains for you to have a thorough checkup on your physical condition at no cost whatever.

This study is a purely medical one and has nothing to do with your relationship with the VA. All of the results will be kept strictly confidential by the National Research Council, and no information about any single individual will be released to any person or agency without your permission.

Through this study it is hoped to learn many things about the treatment of head injuries and how to improve the treatment in the future. The examination is not a lengthy or tedious one and does not require your staying in the hospital. Usually the complete checkup takes only four hours, and there is no red tape or waiting around involved. Also, there are no blood tests to be taken and no needle punctures.

Already over 90 veterans from Los Angeles, Long Beach, the San Fernando Valley—and some from San Bernardino, Riverside, and Santa Barbara, too—have come in for this examination, and all of them have been quite pleased. We explain everything we do, answer any questions we can, and inform the men of the results, upon their request, as soon as it is completed. There is no cost for this checkup and we will reimburse you for your travel expenses at the rate of five cents per mile to and from the hospital.

Although you may have had a number of physical examinations through the Veterans Administration, I should like to say that the Armed Forces are particularly interested in the results of our study and are anxious to have information which can be used now in the treatment of our injured soldiers in Korea. So your return for this checkup would be of great value to the military services.

If you can foresee a few free hours anytime in the near future, I am sure you would not regret this opportunity. Any day of the week (except Sundays) would be satisfactory to us. If you would write to me at the above address, or call my office at Arizona 12345, I am sure an appointment can be arranged to suit your time. I am enclosing a card for your convenience and reply.

Sincerely yours,

J. BROWN, M.D.

Enclosure.

Author Index

Subject Index