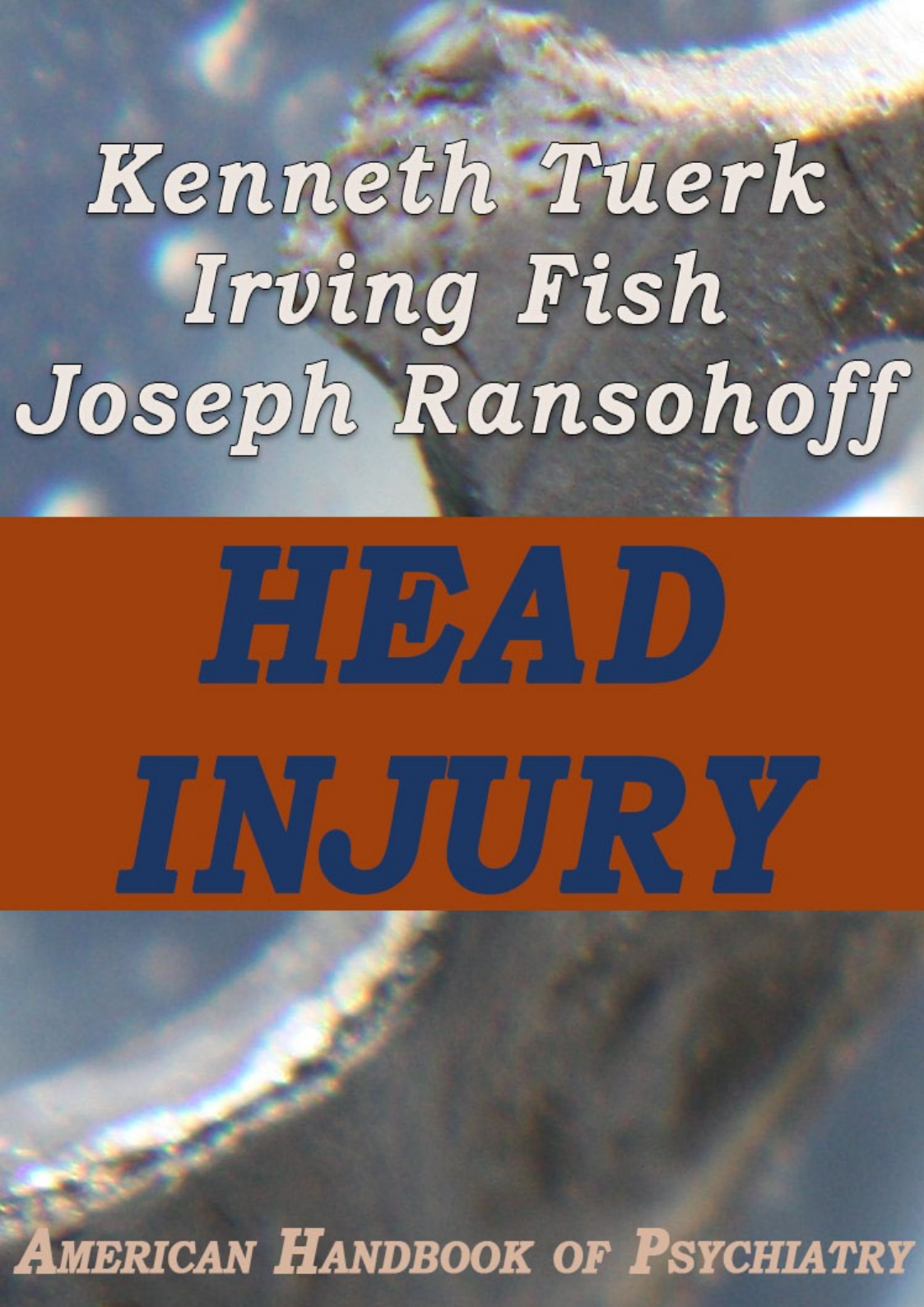
Kenneth Tuerk
Irving Fish
Joseph Ransohoff
HEAD INJURY
American Handbook of Psychiatry

HEAD INJURY

Kenneth Tuerk, Irving Fish, and Joseph Ransohoff

e-Book 2015 International Psychotherapy Institute
From American Handbook of Psychiatry: Volume 4
edited by Silvano Arieti

Table of Contents

HEAD INJURY

The increasing incidence of serious head injuries in the United States is a byproduct of the rapid pace of urban life, as well as of the increasing use of high-speed transportation. Precise statistics are not available but the estimates are staggering. It is well known that accidents are by far the leading cause of death under the age of thirty, over 100,000 annually in the United States. Approximately 50,000 deaths occur each year as a result of automobile accidents, and of these, it is estimated that 60 percent of the victims (or 30,000) died directly as a result of head injury. Vehicular accidents account for about 3 million head injuries yearly, 750,000 concussions, 150,000 skull fractures and 150,000 significant brain injuries (Figures 7-1 and 7-2).

Improved facilities, better understanding of the pathology of head trauma, and advancements in therapy have led to more successful treatment. Mortality has decreased, but as increasing numbers of patients are saved, the morbidity rate has increased. Not all patients are fortunate enough to recover without neurological sequelae.

Evaluation of a posttraumatic patient can be quite complex. Certain deficits, such as hemiparesis, and hemianopsia are obviously of an organic nature. Subtle mental changes may be either organic or functional, or represent a combination of both factors. At times, the symptoms may be only those of mild mental changes,

such as irritability or forgetfulness, with or without such vague symptoms as headache and light-headedness. The skills of both the neurologist and psychiatrist may be taxed in evaluating and treating these patients with the ultimate goal of rehabilitation and return to a normal, productive position in society.

In evaluating the head injury patient, the examining physician should be aware of the possibility of preexisting disease of the central nervous system which may not come to light until an episode of trauma brings it to the patient's or the physician's attention. A slowly developing homonymous hemianopsia may go totally unnoticed by the patient, for example, until such time as he is involved in an auto accident when the other vehicle approaches from his "blind side." Similarly, the patient with developing cerebellar ataxia may not be conscious of his gait disturbance until a fall. Subsequently he may date all of his difficulties to that dramatic episode.

Similarly, in evaluating the patient for possible mental changes after head trauma, one must not only determine that trauma actually occurred, but also that it was significant, and that there was a cause-and-effect relationship between the trauma and the symptoms.

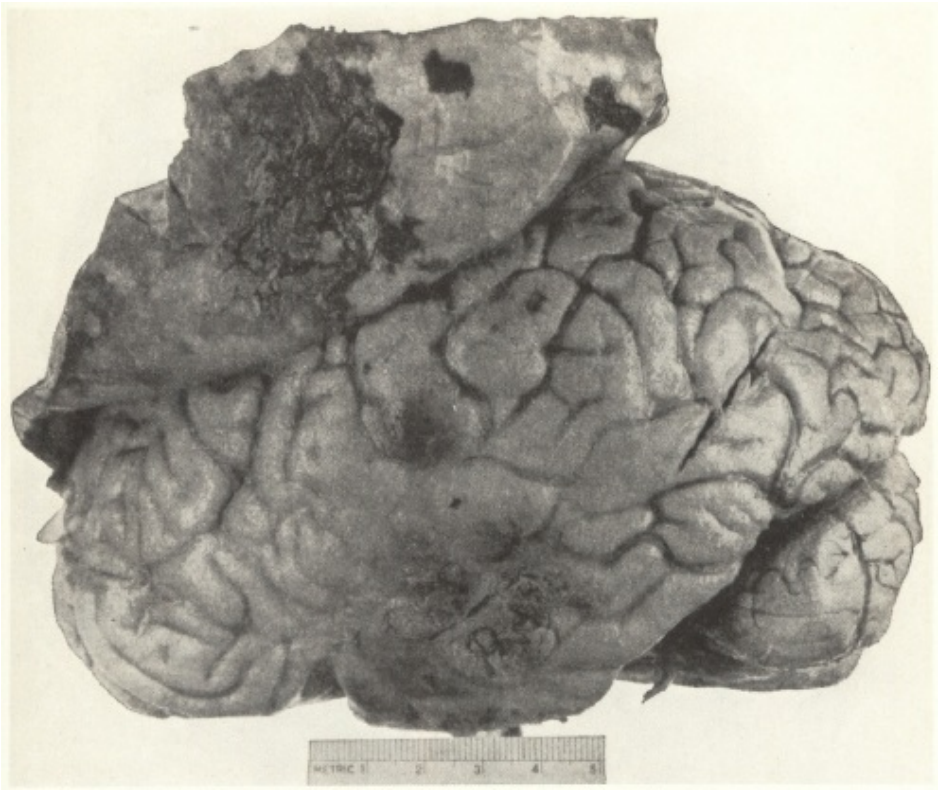

Figure 7—1.

The brain of a thirty-year old male in a traffic accident. The dura is reflected from the left hemisphere to show a subdural hematoma. In the rolandic operculum there is contusion of the brain with subpial bloody discoloration of the cortex. Below in the temporal lobe there are contusion and laceration of the brain. The cut in the occipital lobe is an autopsy artifact. (Courtesy of Dr. Paul I. Yakolev, Warren Anatomical Museum, Harvard University.)

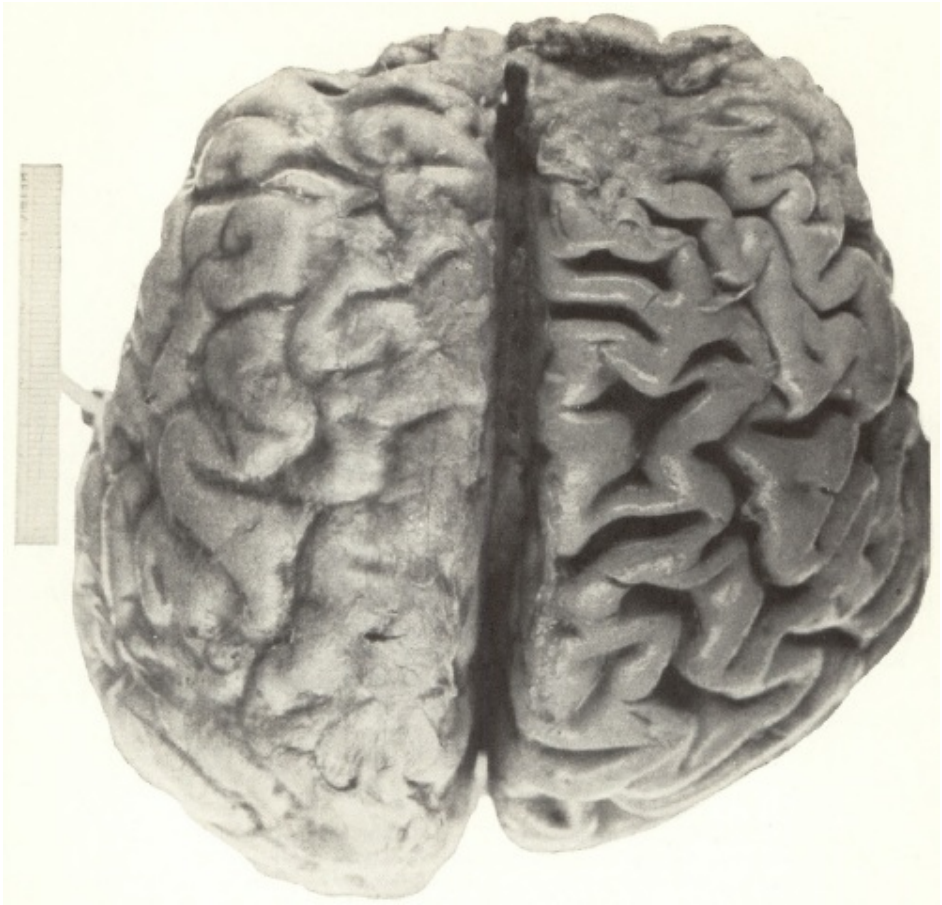

Figure 7—2.

The brain of a middle-aged man who suffered a head injury in an automobile accident several years prior to his death. He showed both intellectual and social deterioration and died in a state hospital. The frontal lobes show old traumatic destruction of convolutions, with atrophy and scarring of the brain. The pia-arachnoid is stripped off from the right hemisphere to show extensive corticomeningeal adhesions over the areas of the old cerebral trauma. (Courtesy of Dr. Paul I. Yakolev, Warren Anatomical Museum, Harvard University.)

Significant Head Trauma

Significant head trauma is generally thought to be that which is followed either by alteration in the state of consciousness, focal neurological signs, or a skull fracture. Although the patient may appear absolutely normal in all respects, any of these conditions are grounds for hospitalization for at least a twenty-four-hour observation period.

In arriving at these criteria, one must remember that while injuries to the soft tissues of the head are not of neurological significance per se, they do afford some indication of the forces applied to the head during the trauma, and can serve to alert us to the possibility of underlying brain damage. During the twenty-four-hour period of observation, evidence of acute intracranial changes generally appears. We must remember, however, that while these guidelines are helpful, it is certainly possible for delayed deterioration to occur after a period of observation, as in the incidence of chronic subdural hematoma, and can even occur without meeting any of the above criteria for admission to the hospital for observation. Figures 7-3 to 7-5 show various stages of brain damage after cerebral trauma.

Trauma, severe enough to cause alteration or loss of consciousness, is obviously significant. Indeed, the severity of the brain injury can be correlated with the length of time the patient remains unconscious. Loss of consciousness for a few seconds to an hour may be empirically classified as mild, one to twenty-four

hours as severe. This, however, is a rough guide and does not mean that a patient who was unconscious for only a few seconds (or not at all) cannot develop dangerous complications.

Figure 7-3.

Cerebral Trauma. Contusion; two days old. Cortex. Hemorrhagic extravasate in the right lower corner of the field; hyperemia; perivascular infiltration ("cuffing") with inflammatory cells. Cresyl violet stain (X 80). (Courtesy of Dr. Paul I. Yakolev, Warren Anatomical Museum, Harvard University.)

Patients with a history of unconsciousness often have a period of pre- and

posttraumatic amnesia. Pretraumatic amnesia is defined as the time interval from the last moment remembered before injury to the time of injury. Posttraumatic amnesia is defined as the time interval from the moment of injury to the time when the patient remembers awakening. Again, a rough correlation can frequently be made between the severity of the intracranial injury, the length of unconsciousness and the extent of pre- and posttraumatic amnesia.

Following head injury, the patient may be seen in either the acute or chronic stage. Although most patients will not be seen by a psychiatrist until the chronic stage, a brief discussion of the acutely injured patient is important to an understanding of the posttraumatic psychiatric sequelae.

Concussion

The definition and implications of the term “concussion” have undergone much change since the description of Wilfred Trotter in 1925. He felt that it was an “essentially transient state due to head injury which is of instantaneous onset, manifest widespread symptoms of purely paralytic kind, does not as such comprise any evidence of structural cerebral injury and is always followed by amnesia for the actual moment of the accident.”

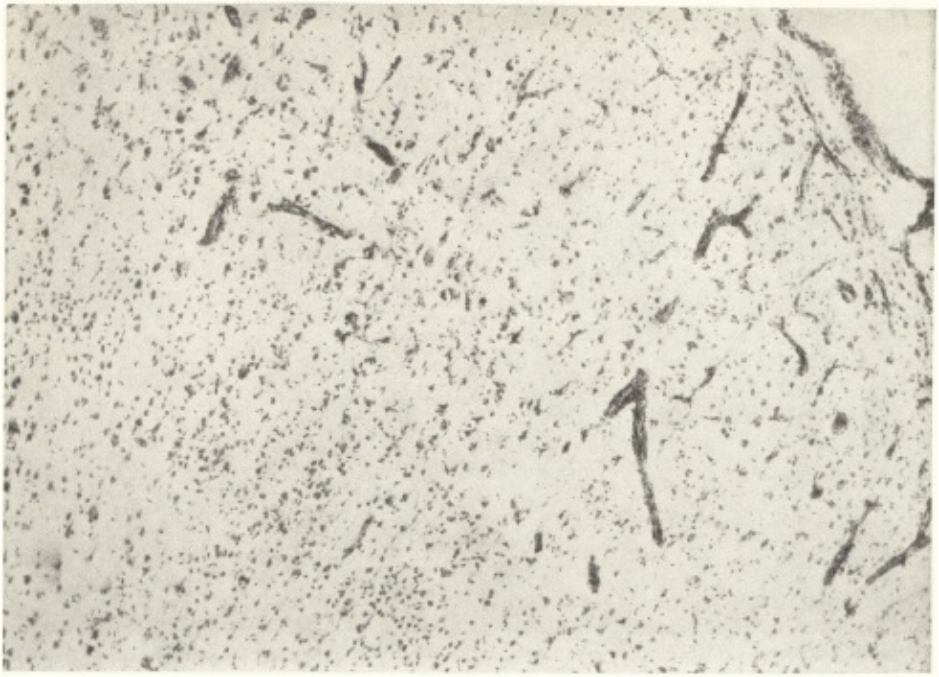

Figure 7-4.

Cerebral Trauma. Contusion; six days old. Cortex. Intense proliferation of new capillaries; patchy areas of nerve-cell loss; beginning organization of the damaged tissue into future scar. Cresyl violet stain (X 80). (Courtesy of Dr. Paul I. Yakolev, Warren Anatomical Museum, Harvard University.)

Some authors have included those patients who are dazed and confused, as well as those who have actually lost consciousness. Clinically, a concussion with loss of consciousness is accompanied by flaccidity, abnormal brain stem reflexes, bradycardia, and apnea. If this state is prolonged, death may ensue, and autopsy may show few or no structural changes. These phenomena can be explained in only two ways: widespread simultaneous neural dysfunction or focal dysfunction in areas concerned with levels of consciousness, as well as respiratory and cardiac reflexes.

It has been shown by French and Magoun that destruction of the reticular formation can produce coma. It has also been shown by Foltz and Schmidt that, following concussion in monkeys, evoked potentials from peripheral stimulation are abolished in the reticular formation but not in other ascending pathways. Finally, electrical action in the brain decreases markedly in many areas after concussion, but the most marked depression almost always occurs in the medial reticular formation. These physiological data, with marked dysfunction of the reticular formation, seem adequate to explain the clinical events during and after a concussion.

A search for a structural pathological explanation is still in progress. It is felt,

at least by some schools of neuropathology, that structural alterations do occur at the time of concussion and include changes in neurons as well as axons. However, cause and effect have not been proved and these changes may be concomitant rather than causative. Indeed, it seems difficult to explain the phenomenon of concussion as anything other than brain-stem dysfunction and the absence of significant lesions is not surprising, as structural changes in this area are rarely compatible with survival. In any case, the term concussion comprises a convenient clinical category for the multitude of patients who have a head injury, a brief alteration in the state of consciousness and complete return to neurological normalcy after a relatively short period of time.

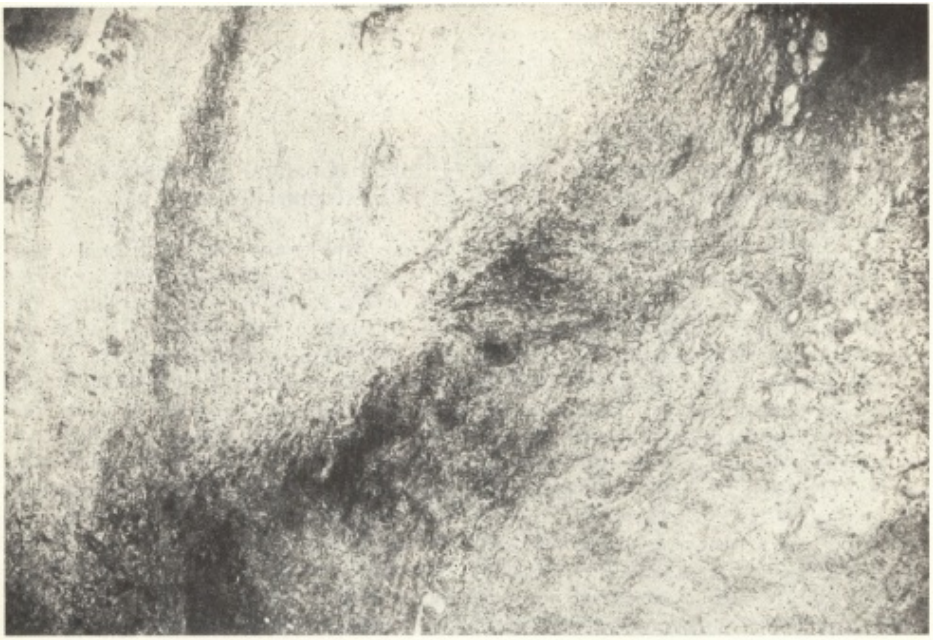

Figure 7-5.

Cerebral Trauma. Contusion; several weeks old. Cortex. Organized scar; proliferation of mesodermal and neuroglial cicatricail tissue; the scar tissue is relatively avascular. Hematoxylin and eosin stain (X 40). (Courtesy of Dr. Paul I. Yakolev, Warren Anatomical Museum, Harvard University.)

Posttraumatic Syndrome

The arguments pro and con structural damage take on real significance in the consideration of the posttraumatic syndrome. This entity is defined as headaches, a feeling of unsteadiness, at times true vertigo, and mental changes. These symptoms may occur alone or in combination, and they may or may not be associated with other more objective neurological signs. They may occur following minimal trauma as well as after a more severe injury. However, the degree of injury does not necessarily correlate with the severity of the posttraumatic syndrome. Headache is the most prominent symptom and is a complaint of most patients in the postconcussion state. Giddiness and/or vertigo are about equally frequent, being present in approximately 50 percent of patients. True vertigo usually indicates pathology and, when associated with postural nystagmus, is objective evidence of damage to the vestibular system. Finally, the many mental symptoms which occur are the most difficult to characterize. Although a mild organic mental syndrome may, indeed, be present, normal results on formal testing are the rule. The usual complaints are nervousness, irritability, impaired memory, and difficulty in concentration.

Although Russell found a slightly higher incidence of this syndrome in more severely injured patients, there is truly no consistent relationship between the severity of postconcussion state and the extent of the injury. A mildly dazed patient may be disabled for years, while a comatose patient may recover without

developing this syndrome. Taylor argues convincingly for an organic etiology, expressing the opinion that this syndrome is present in the majority of patients following head injury and, if this is a functional disorder, then the majority of all postconcussion patients have disabling functional disorders. On the other hand, it is quite clear that there is a high incidence of this syndrome in industrial as opposed to recreational accidents, and a higher incidence of occupational incapacitation in cases involved in compensation or litigation actions. These facts, however, may be due to secondary gain rather than being related to the organic or functional aspects of the syndrome. In the follow-up study after the Korean War, 60 percent of the patients with long-term persistence of posttraumatic syndromes (four to six years) were competing as well as or better than before the injury. Brenner et al., Symmons, and Miller, have all concluded that symptoms which persist for a long period of time are at least in part due to a significant functional overlay. Many investigators have felt that secondary gain plays an important role in the persistence of symptoms and that when financial matters are settled, the syndrome will subside. Two long-term follow-up studies, however, do not bear this out and in addition, the incidence of persistent and disabling symptoms is significantly higher in patients over the age of fifty. This suggests that this syndrome cannot be totally accounted for on a functional basis. It would seem likely, in conclusion, that an organic substrate of a vasomotor nature affecting brain-stem functions plays an important part in the posttraumatic state. It also seems clear that there are non-organic functional factors affecting those

individuals in whom the syndrome persists for months or years.

Mental Changes Associated with Structural Injury

Patients with gross structural damage may have either contusions, lacerations, or hematomas. The hematomas may be either epidural, subdural, intracerebral or, commonly, combinations thereof. Brain swelling (cerebral edema) occurs in a high percentage of instances in association with these lesions and accounts for a significant degree of their morbidity and mortality. Neurological and neurosurgical evaluations are imperative and dictate the immediate medical or surgical treatment. The condition of the patient and his mental state will be determined by the extent of the injury to the brain, the intracerebral location of the injury and, finally, the degree of increased intracranial pressure.

Increased intracranial pressure will be relieved by either medical decompression with mannitol and/or steroids, or by surgical decompression. Early symptoms of increased intracranial pressure may include headache and increasing drowsiness, as well as progressive focal neurological deficits.

Once the pressure is controlled, the patient's clinical status will be a function of the extent and location of the intracerebral damage. Whether the insult be a contusion, laceration or hematoma, the degree of dysfunction should be maximal soon after the injury. At this point, the cerebral dysfunction is a result of: (1) dead and dying cells; (2) injured cells and connections which are not functioning, but will recover and function again; and (3) cells which are not functioning well

because of pressure from either edematous brain or hematoma. Recovery begins soon after injury, as the edema subsides and the repair processes ensue. The final result, assuming optimal recovery, will depend on the amount of unavoidable damage which was sustained at the time of injury. Thus, whether the patient is first seen while comatose, lethargic, or normal, he should remain stable or improve. Any further significant deterioration in the clinical status heralds the onset of a new pathological process other than the original trauma. This may be a systemic problem such as hypoxia or sepsis, or a neurological one such as seizures, meningitis, or hydrocephalus. In any case, one should be alerted to possible deterioration in a patient who was stable or improving. The cause is often treatable and the deterioration reversible.

In addition to the extent of the injury, the mental status can also be determined by the location of the lesion. A very small lesion, strategically placed in the brain stem, produces a greater deficit than a much larger lesion in one of the "silent" areas of the brain. Other focal lesions may produce a neurological deficit, such as hemiparesis or hemianopsia, without any change in mental status.

Although contusions, lacerations, and hematomas may occur at any site, the anterior temporal lobes and the frontal lobes are most common, owing to the sharp bony prominence of the sphenoid ridge and the roof of the orbits.

Focal Injury

There are two areas of focal injury of particular interest to the psychiatrist. The first is the area of the dominant hemisphere, where a lesion may produce disturbances in comprehension manifest by some degree of aphasia, either expressive or receptive. See Chapter 11, entitled “Aphasia,” for a detailed description of these symptoms. When severe, there is no question about the diagnosis and its organic substrate. However, a minimal degree of receptive dysfunction following recuperation from a head injury may bring the patient to the attention of a psychiatrist. The presenting complaint may be “personality change” or “inability to function as before” the injury. Awareness of the condition and appropriate examination will demonstrate the true etiology and the simple “personality change” may become a focal deficit in expression or understanding or both.

The second area of focal lesions, which may require psychiatric consultation, are the frontal and anterior temporal lobes, particularly when the orbital and medial portions are injured. As noted before, the frontal and temporal lobes are preferred sites for injury in head trauma. The first, and perhaps still one of the best, descriptions of such a patient is that of Phineas T. Gage, who, in 1848, was injured with a crowbar in the left frontal lobe. He recovered and lived for many years, but underwent an extreme change in personality and behavior which was followed and reported by his physician. Other reports have since appeared, but

most of the information on the effects of frontal lobe ablation has come from patients on whom it was surgically performed for therapeutic reasons. This technique was first employed by Moniz in 1936. The patients with frontal lobe ablation are characterized by apathy and lack of foresight. Their affect is flat, anxiety is reduced, and there is a lack of concern for the consequences of their actions, both verbal and physical. This leads to inappropriate behavior, tactlessness, and a lack of concern for environment and personal appearance.

Extensive testing has been performed on patients before and after ablation. Speech is particularly affected. There is a significant reduction in spontaneous speech. Verbal response to questions is delayed and reduced to phrases and short sentences. Intelligence tests are difficult to evaluate. They show definite reduction in verbal scores. Performance scores are about the same and may actually improve after frontal-lobe ablations. There is decreased facility for abstract thinking. However, part of the reduction in scores may be attributed to damages in personality.

Organic Mental Syndrome (OMS)

The most common and disabling change in mental status after severe head injury is an organic mental syndrome. This is a condition resulting from any of a number of organic pathological processes, whether traumatic or not. The syndrome thus is a final common pathway of many organic diseases, one of which is trauma and its sequelae.

The hallmarks of the syndrome are dementia and loss of recent memory. In its mildest form, there are subtle changes in personality such as increased instability, decreased attention span, reduction in capacity for abstract thinking and, out of proportion to all other signs, a decrease in recent memory and recall. If the organic mental syndrome is more severe, an outright dementia may occur with disorientation, lack of personal care, incontinence, and severe lability of affect. The mood may be either manic or depressed but this is governed to some extent by the pre-morbid personality. Focal neurological deficits may or may not be present. However, attempts to correlate the OMS with a specific localization of brain injury have failed. It is best correlated with diffuse, bilateral cerebral dysfunction. The first and most common etiology of an OMS following head injury is diffuse, bilateral brain damage as a result of the trauma and secondary edema.

As his state of consciousness improves, the patient may pass through a stage of traumatic delirium characterized by excessive motor activity and confusion. He may scream, try to climb out of bed, and alternate between periods of

spontaneous but inappropriate speech and sleep. Sedation and restraints may be necessary to prevent the patient from inflicting further injury upon himself.

Finally, continuing on the path of recovery, the patient manifests a severe organic mental syndrome which gradually becomes milder and finally disappears.

Depending on the severity of the injury, patients may pass through these stages in a few minutes, days, or months. Generally, the more severe the injury, the longer the period of unconsciousness and the more extended and gradual the recovery period. After very brief concussions, there may only be a few minutes of confusion before return to normal, and all these stages may not be evident.

Unfortunately, not all patients recover. Those with a severe deficit may be considered for institutionalization. Those with a milder deficit may be discharged from the hospital only to arouse anxiety in the family by even moderate changes in their behavior and personality. This may create havoc within the family unit.

The first step in the evaluation of such patients is to confirm the diagnosis of organic mental syndrome, i.e., dementia and loss of recent memory. At times, psychometric evaluation may be helpful in establishing the diagnosis. In cases of a posttraumatic organic mental syndrome, the crucial questions to be asked are whether the process is the end result of the traumatic event, whether further recovery will occur and, finally, whether the arrest in recovery is the result of some intervening, superimposed disease or complication. These patients are often

young and ambulatory with a long life expectancy. Therefore, particularly as the rate of recovery of mental function is decreasing or when the condition has stabilized, the physician must be absolutely sure that nothing further can be done to advance recovery before offering a final diagnosis and prognosis to the patients and their families.

Chronic Subdural Hematoma

A chronic subdural hematoma usually manifests itself by increased intracranial pressure and progressive focal neurological deficit. At times, and particularly in an injured brain, a progressive organic mental syndrome may be the earliest sign. If not corrected, this progression may continue until the patient becomes progressively obtunded and, finally, comatose. Headache, although usually present, may easily be overlooked. Focal neurological signs may not be present until late and papilledema may be present in 25 percent of cases. Skull X-ray may show the pineal gland to be shifted off the midline. Brain scan will be positive in 90 percent of cases. Lumbar puncture may show xanthochromia and increased protein in 50 percent of cases. Lumbar pressure is usually elevated, but a normal pressure does not exclude the diagnosis of hematoma. Echogram may show a shift of the midline structures, and cerebral angiography is diagnostic.

Early diagnosis and treatment is quite gratifying, with most patients returning to their previous neurological status. However, the diagnosis may be missed in a slowly deteriorating patient until coma and herniation ensue. Emergency surgery at this time, while often successful, does not always carry the same favorable prognosis.

Communicating Hydrocephalus

Another complicating condition, which may produce an organic mental syndrome, is a secondary communicating hydrocephalus. It has been known for some time that head trauma with bloody CSF (cerebrospinal fluid) may produce a subacute hydrocephalus in the weeks following the injury. Moritz and Wartman, in 1938, discussed three cases with postmortem changes of arachnoiditis. In 1956, Foltz and Ward reported the successful treatment of hydrocephalus after subarachnoid hemorrhage of diverse etiologies, including trauma. In their cases, onset was between the second and tenth week after the hemorrhage. The important factor in all of these cases appears to be the arachnoidal reaction to the blood in the spinal fluid, producing an obstruction to the normal flow of fluid in the subarachnoid space. The obstruction is usually at the level of the tentorium but may, at times, be over the cerebral convexity. This obstruction results in communicating hydrocephalus with its attendant large ventricles and signs of diffuse cerebral dysfunction. Although this was once thought to be a rare occurrence, studies have shown that some degree of hydrocephalus, although not always clinically significant, is quite common after head injury with bloody CSF.

The clinical course of posttraumatic hydrocephalus may vary. In its mildest state the diagnosis may be an incidental finding when contrast studies are performed for other reasons. In other cases, there may be a mild organic mental syndrome or deterioration of a previously improving neurological state. After a

few weeks, the mechanisms of production and resorption of spinal fluid equilibrate and the process may resolve spontaneously. Finally, in its most severe expression, the hydrocephalus is progressive and relentless, causing not only a plateau of mental recovery, but retrogression, leading at times to severe obtundation. Neurological and neurosurgical consultations are imperative as the situation may become reversible after a shunting procedure.

In 1965, Adams et al. reported the syndrome of "normal-pressure hydrocephalus" (occult hydrocephalus, low-pressure hydrocephalus). These patients present with a progressive OMS, unsteady gait, urinary incontinence, and normal pressure on lumbar puncture. There is nothing particularly characteristic about the OMS but the gait disturbance is quite unusual. It is somewhat broad based and unsteady, although no cerebellar signs are present. There appears to be some difficulty with the initiation of walking movements and some patients have stated that "it feels as if my feet are glued to the floor." This has been characterized as "a magnetic gait." Onset of these symptoms may be months and even years after the injury. Skull X-ray and lumbar puncture are normal. Definitive diagnostic tools are RISA (radioactive iodinated serum albumin) cisternogram and pneumoencephalogram. In the former, the I salt-free albumin is injected via lumbar puncture. Normally, the follow-up brain scans show activity in the basal cisterns in four hours. By twenty-four hours, there is diffuse activity over the cerebral convexities and sagittal sinus. In occult hydrocephalus, the activity enters the lateral ventricles within six hours and, even at the end of forty-eight hours,

there is no activity over the convexities. The pneumoencephalogram shows enlarged ventricles with little or no air in the supratentorial subarachnoid space, indicating a block of the CSF pathways at the level of the tentorium. This condition must be differentiated from hydrocephalus ex-vacuo, i.e., cerebral atrophy. In this situation, the RISA scan is normal and, although the ventricles are large as a result of atrophy, air does pass over the convexities and demonstrates widened sulci and atrophic gyri. Although there are two contrary reports, most observers feel that shunting is of no value in these patients, as the hydrocephalus is secondary to atrophy, in contrast to the results of shunting in patients with obstructive hydrocephalus secondary to trauma.

In addition to trauma, other etiologies accounting for occult hydrocephalus include subarachnoid hemorrhage, meningitis, and intracranial surgery. The idiopathic cases are thought to be due to unrecognized or forgotten trauma or infection. The few pathologic studies performed have confirmed the presence of adhesions at the base of the brain.

Posttraumatic Epilepsy

Posttraumatic epilepsy is a disorder which, at first, may appear to be functional but is, on close examination, an organic sequela of head injury. The incidence is quite low in closed head injuries, whether or not the patient has been unconscious. However, when the dura is penetrated, the incidence rises to about 50 percent.

Posttraumatic seizures can be divided into two groups, depending on whether the onset is early or late. Of the patients who develop seizures, about 10-15 percent have their ictus within one month, often within the first forty-eight hours after injury. These seizures are secondary to an irritative process set off by cerebral contusion, laceration, or edema. The prognosis is good and after appropriate therapy, the seizures usually subside. Although the long-term prognosis is good, maintenance anticonvulsant therapy should be initiated and a baseline EEG (electroencephalogram) obtained. Therapy may be discontinued after two years if the EEG has reverted to normal.

About 85 percent of patients with posttraumatic epilepsy have a delayed onset. Of those who develop seizures, 50 percent do so within six months and 75 percent in two years. Because of the high incidence of posttraumatic seizures in penetrating wounds and the social stigmata accompanying these seizures, we feel that all such patients should be placed on prophylactic anticonvulsant therapy for at least two years. The medication may then be discontinued if the EEG is normal.

The prognosis of all types of posttraumatic seizures is good.

The seizure itself, with its period of relative hypoxia and postictal confusion may interrupt the convalescence of the head-injured patient, particularly if repeated. Focal neurological deficits may become more pronounced postictally and, with lesions of the dominant hemisphere, aphasia may become exaggerated.

Approximately 80 percent of posttraumatic seizures have a focal component. At times, the seizure arising from a temporal lobe focus consists entirely of psychomotor phenomena. These patients, more than others, may be brought to psychiatric attention. The dreamy state, periods of amnesia, episodes of deja-vu and sudden emotional outbursts may easily be mistaken for a functional disorder.

Boxing Encephalopathy

Boxing encephalopathy (chronic progressive traumatic encephalopathy of boxing) has been associated with the injuries received in the ring. The disease is characterized by a progressive OMS with both pyramidal and extrapyramidal signs. Symptoms do not usually occur until fifteen to twenty-five years after the onset of the boxing career. Only a few pathological examinations have been performed and these show atrophy with widespread areas of gliosis. The etiology is contested. It was long felt to be secondary to repeated trauma with each episode causing a bit more permanent damage. This hypothesis is contradicted by the fact that symptoms may not begin until long after retirement and are then progressive. Alcoholism has been invoked as a possible etiology but retrospective studies have shown that a significant number of these patients are not alcoholic. The exact etiology remains to be clarified. It may simply be that the repeated episodes of trauma leave only a marginal reserve. As these patients become older, they develop an OMS at an early age.

Head Trauma in Children

If the incidence of head trauma is high in adulthood, it is virtually 100 percent in childhood, multiple minor head injuries being a frequent occurrence in this age group. Approximately 200,000 children each year have head trauma severe enough for them to be hospitalized. About 5-10 percent of these exhibit neurologic findings at the time of admission.

The differences in the sequelae of head trauma in children as compared to adults, is related to two main factors: (1) The state of closure of the sutures of the skull at the time of injury, and (2) The state of development of the brain at the time of injury.

Sutures

At birth the sutures of the skull are not closed. This allows for the growth of the skull to accommodate the enlarging brain comfortably, without increasing the pressure within the cranium. The period of the most rapid postnatal brain growth is the first six months of life. At about six to nine months, the sutures begin to fuse. The anterior fontanelle is the last to close at about eighteen months of age. Fusion between the bones of the skull gradually becomes more firm over several years.

The nonfused or partially fused skull of the infant and young child is apparently better able to absorb the energy transmitted to it by external forces,

molding or distorting with the blow, and protecting the underlying brain. The young brain with its higher water content is also better able to tolerate external forces than the older, more solid adult brain. Every summer we see in our large-city hospital emergency rooms innumerable instances of survival following falls from open windows four or five floors above the street. This is another evidence for the tolerance of the infant to head trauma.

Thus, the dynamics of increased intracranial pressure are different in early childhood than in adulthood, and the younger the child, the greater the difference. It was mentioned previously that brain dysfunction following head trauma is caused by (1) direct injury to the brain, e.g., laceration or contusion; (2) by edema; and (3) by increased intracranial pressure secondary to edema or blood which may be intracerebral or extracerebral (subdural or epidural hematoma). Increased intracranial pressure in adults causes downward herniation in the cerebrum through the tentorium, causing pressure on the brain stem which results in disturbances of consciousness, cranial-nerve dysfunction and abnormalities in cardiovascular and respiratory function. This threatens life and must be treated immediately by decompression as mentioned previously. Otherwise permanent brain-stem dysfunction can result, although the brain stem may not have been injured directly. In young children, with an open fontanelle and sutures that are not closed, supratentorial blood or edema causes enlargement of the skull and accommodation of the increased contents which result from the injury. Consequently, the brain has less of a tendency to “herniate down” into the

brain stem. However, this can occasionally take place if the sudden increase in intracranial volume exceeds the expansion capacity of the skull. Obviously, the older the child, the more the dynamics approach those of adults, and therefore, the more danger of injury secondary to herniation.

Hydrocephalus may follow head trauma because of obstruction of the extraventricular spinal fluid pathways along the base of the brain. This results in an enlarged head and pressure on the white matter surrounding the ventricles. Treatment consists of shunting the fluid from the ventricles into another body cavity such as the atrium of the heart, the peritoneum, or the pleural space.

If subdural hematomas are very large, they may also cause enlargement of the head and pressure on the gray matter of the cerebrum. If the subdurals are small, they may resorb on their own without causing pressure sequelae. Sometimes repeated paracentesis of the subdural fluid is enough to prevent signs and symptoms. If the subdurals are very large, shunting of the fluid into another body cavity may be required.

Brain Development

The second major factor which differentiates the prognosis and sequelae of head trauma in children from adulthood is the state of development of the brain at the time of injury. The newborn brain has fewer cells, less myelin, and a less well developed dendritic system than that of adults. The immature injured brain is able

to compensate better. It is said to have more plasticity. Intact parts of the brain can “take over” an injured or destroyed area’s function. For example, in an adult, an injury to the dominant hemisphere in the speech centers results in aphasia. In children under two this never happens, and even up to the age of five, “compensation” by the opposite side is frequently complete or almost complete. As the patient gets older, the ability to compensate decreases.

All of this means that a child with a brain injury secondary to head trauma has a far better chance for complete or satisfactory recovery than an adult with a comparable head injury. Also, the younger the child, the better the chances of satisfactory mental and motor recovery. Unfortunately, this does not mean that every child recovers satisfactorily from a head injury. Approximately 5-10 percent of childhood head injuries, which result in hospitalization, are severe. About 30 percent of children with severe head injury who survive, remain with neurological and/or mental dysfunction.

The most accurate gauge as to whether a child will remain with a brain dysfunction is the same as that with adults, namely the longer the posttraumatic amnesia (PTA), the more likely the chance for a permanent deficit. However, at every phase of recovery, the degree is much better in childhood.

Richardson studied ten children who were comatose from seven to forty-seven days after head injury. The PTA varied between twenty-five and sixty-five

days. Only one patient was so incapacitated that he could not care for himself. Six others had motor or movement abnormalities. All had some intellectual and behavioral changes. Improvement in these patients continued for up to four years after the head injury.

An interesting study of patients who suffered less severe head trauma and were evaluated an average of ten years later, was performed by Dencker in Sweden. These patients were twins, and the twin was used as control. She failed to find any significant differences in intellectual, personality, EEG, or performance between the "head injured" and the control groups.

Sequelae of Head Trauma in Childhood

The common neurologic and psychologic sequelae of head trauma may be listed in six categories.

Motor and Movement Disorders

Focal damage in the pyramidal system gives rise to spastic hemiplegia or hemiparesis. If the lesion is bilateral, quadraparesis results. If the lesion involves the postcentral gyrus, sensory abnormalities result. Sensory impairment is usually incomplete. Destruction of the extrapyramidal system, such as the lenticular nuclei, may result in choreiform or athetoid movements, tremors, and rigidity.

Posttraumatic Syndrome

The posttraumatic syndrome, seen so extensively in adults, occurs in only about 1-6 percent of children following head injury. In children, headaches predominate, with giddiness and aesthenia being quite rare. The vast majority of patients with this syndrome have a self-limited course, usually lasting less than six months. The very few cases which persist beyond six months are usually early adolescents or preadolescents. The etiology and pathogenesis of this syndrome is discussed earlier in this chapter (p. 171).

Posttraumatic Epilepsy

Posttraumatic epilepsy occurs in about 10 percent of children. The lesions are usually located in the medial portion of the temporal lobe, the posterior frontal, or the anterior parietal lobes. Those children who suffer a seizure at the time of or very shortly after a head injury are *not* more likely to have posttraumatic epilepsy. Even an EEG which shows epileptiform activity soon after injury is an unreliable prognosticator, for over a period of several weeks the epileptiform activity gradually disappears. If permanent seizures occur, it is generally within the first two years after the acute head injury.

Minimal Cerebral Dysfunction Syndrome

Although many patients recover full motor and sensory functions, they may

remain with signs and symptoms of minimal cerebral dysfunction. These include hyperactivity, easy distractibility, short attention span, visual motor-perception deficits, cognitive dysfunction, clumsiness, spatial disorientation, and learning disability. This syndrome is discussed fully in Chapter 8. Suffice it to say here that head injury is occasionally etiologic in this syndrome. Caution must be shown in ascribing head trauma as the etiology in too many patients with minimal cerebral dysfunction. Almost every child has had a significant bump on the head at one time or another. This does not mean that the etiology of the minimal cerebral dysfunction is necessarily related to the head trauma.

Mental Retardation

Mental retardation resulting from head injury is a result of diffuse brain injury. Generally, the more extensive the injury, the more severe the retardation. There are usually associated motor and/or movement disorders associated with the mental retardation secondary to head trauma. In these patients the head injury is usually severe and the PTA prolonged.

Personality Changes

Occasionally children have profound changes in personality that are beyond those which fit into the category of minimal cerebral dysfunction. In addition to hyperactivity and short attention span they become aggressive, destructive, lacking in judgment, and emotionally extremely labile. This can be severely

incapacitating to the child and his family. These children usually respond poorly to psychotherapy and medication. It may or may not be associated with motor dysfunction.

Natural History of Children Suffering Head Trauma

As mentioned previously, most children who suffer head trauma do not show any adverse sequelae. When evaluating the patient who has deficits (neurologic, intellectual, or behavioral), we must keep in mind the fact that recovery can take place for a period of up to four years after the injury. This means that we must not "give up" on a patient early or because he has severe deficits soon after head trauma. For example, passive exercises to prevent contractures should be continued. Educational and environmental manipulation should be instituted when necessary. There should be frequent reassessment because gradual improvement is the rule and what may have been appropriate educational placement at the time of initial evaluation may be inappropriate six or eight months later.

Finally, evaluation of therapy must be assessed in the light of the spontaneous improvement. Heroic claims have been made for several elaborate modes of therapy. They must be assessed with the natural history of spontaneous improvement of head injuries in children in mind.

Bibliography

Adams, R. D. "Further Observations on Normal Pressure Hydrocephalus," *Proc. R. Soc. Med.,* 59 (1966), 1135-1140.

Adams, R. D., C. M. Fisher, S. Hakim et al.

"Symptomatic Occult Hydrocephalus with 'Normal' Cerebrospinal Fluid Pressure: a Treatable Syndrome," *N. Engl. J. Med.,* 273 (1965). 117-126.

Barber, H. O. "Postural Nystagmus, Especially after Head Injury," *Laryngoscope,* 74 (1964), 891-944.

Brenner, C., A. P. Friedman, H. H. Merritt et al. "Post-Traumatic Headache," *J. Neurosurg.,* 1 (1944), 379-391.

Caverness, W. F. "Onset and Cessation of Fits Following Craniocerebral Trauma," *J. Neurosurg.,* 20 (1963), 570-583.

_____. "Post-Traumatic Sequelae," in W. F. Caverness and A. E. Walker, eds., *Head Injury Conference Proceedings,* pp. 209-219. Philadelphia: Lippincott, 1966.

Courville, C. B. *Commotio Cerebir.* Los Angeles: San Lucas Press, 1953.

Critchley, M. "Medical Aspects of Boxing, Particularly from a Neurological Standpoint," *Br. Med. J.,* 1 (1957), 359.

Crown, S. "Psychological Changes Following Prefrontal Lobotomy. A Review," *J. Ment. Sci.,* 97 (1951), 49-83-

Dencker, S. J. "Closed Head Injury in Twins," *Arch. Gen. Psychiatry,* 2 (1960), 569.

DeVivo, D. C. and P. R. Dodge. "Diagnosis and Management of Head Injury," *Pediatrics,* 48 (1971), 129.

Foltz, E. L. and R. P. Schmidt. "The Role of the Reticular Formation in the Coma of Head Injury," *J.*

Neurosurg., 13 (1956), 145-154.

Foltz, E. L. and A. J. Ward, Jr. "Communicating Hydrocephalus from Subarachnoid Bleeding,"*J. Neurosurg.,* 13 (1963), 546-566.

Freeman, W. and J. W. Watts. *Psychosurgery,* 2nd ed. Springfield, Ill.: Charles Thomas, 1950.

French, J. D., and H. W. Magoun. "Effects of Chronic Lesions in the Cephalic Brain Stem of Monkeys," *Arch. Neurol. Psychiatry,* 68 (1952), 591-604.

Friedman, A. P., C. Brenner, and Denny-Brown, "Post-Traumatic Vertigo and Dizziness," *J. Neurosurg.,* 2 (1945); 36-46.

Gordon, N. "Post-Traumatic Vertigo with Special Reference to Postural Nystagmus," *Lancet,* 1 (1954), 1126.

Heinz, E. R., D. O. Davis, and H. R. Karp. "Abnormal Isotope Cisternography in Symptomatic Occult Hydrocephalus. A Correlative Isotope Neuroradiology Study in 130 Subjects," *Radiology,* 95 (1970), 105-120.

Johnson, J. "Organic Psychosyndrome Due to Boxing," *Br. J. Psychiatry,* 115 (1969), 45-53.

Kilberg, J. K. "Head Injury in Automobile Accidents," in W. F. Caverness and A. E. Walker, eds., *Head Injury Conference Proceedings,* pp. 27-36. Philadelphia: Lippincott, 1966.

Lindenberg, R. "Trauma of Meninges and Brain," in J. Minckles, ed., *Pathology of the Nervous System,* Vol. 2, pp. 1705-1765. New York: McGraw-Hill, 1971.

Mealey, J., Jr. *Pediatric Head Injuries.* Springfield, Ill.: Charles C. Thomas, 1968.

Miller, H. "Accident Neurosis," *Br. Med. J.,* 1 (1961), 919, 992.

Moniz, E. "Essai d'un Traitment Chirurgical de Certaines Psychoses," *Bull. Acad. Med. Paris,* 115 (1936), 385-392.

Moritz, A. R. and W. B. Wartman. "Post-Traumatic Internal Hydrocephalus," *Am. J. Med. Sci.,* 195

(1938), 65-70.

Ojeman, R. G., C. M. Fisher, R. D. Adams et al. "Further Experiences with the Syndrome of 'Normal' Pressure Hydrocephalus," *J. Neurosurg.,* 31 (1969), 279-294.

Petrie, A. *Personality and the Frontal Lobes,* Philadelphia: Blakiston, 1952.

Phillips, G. "Traumatic Epilepsy after Closed Head Injury," *J. Neurol. Neurosurg. Psychiatry,* 17 (1954), 1-10.

Rabe, E. F., R. E. Flynn, and P. R. Dodge. "Subdural Collections of Fluid in Infants and Children," *Neurology,* 18 (1968), 559.

Richardson, F. "Some Effects of Severe Head Injury. A Follow-up Study of Children and Adolescents after Protracted Coma," *Dev. Med. Child Neurol.,* 5 (1963), 471.

Russell, W. R. "The After Effects of Head Injury," *Trans. Med. Chir. Soc. Edinburgh,* 113 (1933-1934), 129-144.

Russell, W. R. and A. Smith. "Post-traumatic Amnesia in Closed Head Injury," *Arch. Neurol.,* 5 (1961), 4-17.

Salmon, J. H. "Senile and Presenile Dementia. Ventriculoatrial Shunt for Symptomatic Treatment," *Geriatrics,* 24 (1969), 67-72.

Salmon, J. H. and J. L. Armitage. "Symptomatic Treatment of Hydrocephalus Ex-vacuo. Ventriculoatrial Shunt for Degenerative Brain Disease," *Neurology,* 18 (1969), 1223-1226.

Spillane, J. D. *Five Boxers, Br. Med. J.,* 2 (1962), 1205-1210.

Steegman, A. T. "Dr. Harlow's Famous Case. The Impossible Accident of P. T. Gage," *Surgery,* 52 (1962), 952-958.

Stritch, S. J. "Shearing of the Nerve Fibers as a Cause of Brain Damage in Head Injury. A Pathological Study of 20 Cases," *Lancet,* 2 (1961), 443-448.

Symonds, C. P. “Concussion and Contusion of the Brain and their Sequelae,” in S. Brock, ed., *Injuries of the Brain and Spinal Cord and Their Coverings,* 4th ed., pp. 69-117. Baltimore: Williams & Wilkins, 1960.

_____.“Concussion and Its Sequelae,” *Lancet,* 1 (1962), 1-5.

Symonds, C. P. and W. R. Russell. “Accidental Head Injuries: Prognosis in Service Patients,” *Lancet,* 1 (1943), 7-14.

Taylor, A. R. “Post-Concussional Sequelae. *Br. Med. J.,* 3 (1967), 67-71.

Trotter, W. “On Certain Minor Injuries of the Brain,” *Lancet,* 1 (1924), 933-939.

Vital Statistics of the United States, Vol. 2, *Mortality,* Washington, D.C.: Nat. Center for Health Statistics, 1967.

Walker, A. E. *Post-Traumatic Epilepsy.* Springfield Ill.: Charles C. Thomas, 1949.