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Physiology of the Brain and Related Trauma

Edward W. Shannon*

The scope of this paper will concern itself with the mechanism of various types of head trauma as well as the immediate and late sequelae of the resulting brain injuries. No attempt will be made to discuss therapy.

The seriousness of the problem of trauma can be appreciated when one stops to realize that accidental deaths now exceed the combined deaths from all infectious and communicable diseases excluding chronic diseases. The National Safety Council reports that more than ninety thousand people are killed in accidents every year. Approximately four hundred thousand are permanently disabled and almost ten million are injured severely enough to disable them for more than one day. Another significant figure is that accidents now rank as the first cause of death between the ages of one and thirty-five years and are exceeded in the older age group only by cancer, heart disorders and the so-called degenerative diseases.¹ Motor vehicle accidents over the past few years are responsible for about thirty-eight thousand deaths annually or about four percent of the total of all kinds of accidents. More than one-half of all persons requiring hospitalization for injuries received the trauma in motor vehicle accidents. A significant percentage of the fatal accidents are due to head trauma. In a study of two hundred fatal pedestrian accidents, it was noted that one hundred and twenty-two of these patients had head injuries.² It was significant that in this study many of the persons sustained significant damage to the brain or its covering without any fracture of the skull or prominent injury to the superficial soft tissue to indicate the presence of an underlying brain injury. Actually forty-two of one hundred and sixteen of the cases fell into this class. They also indicated that when skull fracture was present the possibility of a laceration

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of the brain was high. Of seventy-four fatally injured persons with skull fractures, over half had accompanying lacerations of the brain. Of this, twenty-seven per cent were basilar, the others involved the vault. Seventy per cent of head injuries are caused by automobiles and thirty-five per cent of all accidental deaths are due to brain injuries.3

The brain in the adult is encased in a rigid spheroid bony structure known as the skull. The skull varies in thickness from two to six millimeters. The vault of the skull has a smooth interior and is covered by the scalp. The base of the skull is irregular and divides into three fossas. This irregularity of the base accounts for the laceration which may occur in certain types of head injuries to the undersurface of the brain. The brain is separated from the inner table of the bone by the meninges which are composed of three layers of connective tissue. The first of these is the dura which is a tough, membranous structure that is adjacent to the inner table of the brain. Immediately under the dura is a fine spiderweb-like network known as the arachnoid. The pia mater is invested over the sulci, the outer surface of the brain. It is between the arachnoid and the pia membrane that the cerebrospinal fluid circulates on the surface of the brain.

The brain consists of the cerebrum, the brain stem, and the cerebellum, all of which are encased in the bony skull or cranium. The cerebrum is composed of two large halves or hemispheres which occupy the larger part of the cranial cavity. The upper surface of the cerebrum is somewhat convex. The undersurface is irregular, conforming to the depressed spaces of the fossa at the base of the skull. Each hemisphere is a soft whitish mass of tissue deeply grooved with many folds or convolutions. The cerebrum has a narrow frontal pole situated at the brow and a posterior region which extends to the occiput or back of the head. The two hemispheres are separated in the upper half by a space called the longitudinal fissure. The external surface of the brain is enfolded into convolutions which are separated from each other by grooves that are known as sulci. Some of these grooves are short and others are very deep and long, dividing each hemisphere into major sections or lobes. There is a deep cleft beginning in the undersurface of the brain and extending backward and upward. It is known as the fissure

3 Grinker, Neurology 951 (1943).
of Sylvius. This divides the temporal lobe from the frontal lobe. There is another major fissure known as a Rolandic fissure which divides the frontal lobe from the parietal lobe.

The various lobes of the brain have to do with relatively specific functions. It is thought that the intellectual functions are more prominent in the frontal lobes. In the posterior part of the frontal lobe is the voluntary motor control of the body. The right motor area of the brain controls voluntary movements of the left side of the body while the left cerebral motor strip controls the right. The parietal lobe which is immediately in back of the frontal lobe has to do with the reception of sensation. The occipital lobe is the most posterior and has to do with interpretation of visual impulses. The temporal lobe has to do with speech. Speech is a function that is unique to human beings. In a right handed individual the left side of the brain is the dominant hemisphere and it is the dominant hemisphere in which the speech center is controlled. Thus an injury to the left temporal lobe in a right handed individual will result in speech difficulty, whereas the same injury on the opposite side of the non-dominant hemisphere will not affect speech.

The brain stem lies at the base of the brain and extends down to the spinal cord. It is concerned chiefly with the transmission of impulses from the various parts of the brain and to and from the spinal cord. Also in it are located the nuclei of the cranial nerves.

The third main division of the brain is the cerebellum or little brain. This is located in the posterior fossa and chiefly concerned with the coordination of the fine voluntary movements. Within the brain are four cavities that are known as ventricles. The lateral ventricles are each located in the cerebral hemispheres. They join through a small opening to a structure in the mid-line which is known at the third ventricle. This in turn is connected to a small passageway back to the fourth ventricle which is located in the posterior part of the brain and is covered by the cerebellum. Coming off the undersurface of the brain are twelve pairs of nerves that are known as the cranial nerves. These serve specific functions that have to do with the reception of special senses such as smell, sight, hearing, balance, taste as well as furnishing motor power to muscles and the extraocular movements of the eyes. Specific functions of these nerves will be discussed in the injuries which may affect them.
Specific injuries to various parts of the brain result in different clinical syndromes. The end result of a head injury varies from death to complete recovery.

A study by Gurdjian and Webster as to the cause of head injuries in seven hundred and sixteen unselected cases is of some interest.

<table>
<thead>
<tr>
<th>Cause</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Automobile accidents</td>
<td>20.0</td>
</tr>
<tr>
<td>Struck by vehicles</td>
<td>17.0</td>
</tr>
<tr>
<td>Blows</td>
<td>35.1</td>
</tr>
<tr>
<td>Object unknown</td>
<td>14.2</td>
</tr>
<tr>
<td>Fighting and beatings</td>
<td>9.7</td>
</tr>
<tr>
<td>Blunt objects</td>
<td>6.6</td>
</tr>
<tr>
<td>Metal objects</td>
<td>4.9</td>
</tr>
<tr>
<td>Collision in ball play</td>
<td>0.3</td>
</tr>
<tr>
<td>Falls</td>
<td>22.2</td>
</tr>
<tr>
<td>Accidents while drunk</td>
<td>4.3</td>
</tr>
<tr>
<td>Bicycle accidents</td>
<td>0.8</td>
</tr>
<tr>
<td>Attempted suicide</td>
<td>0.1</td>
</tr>
</tbody>
</table>

These causes certainly seem to account for the great majority of head injuries which are seen in a generalized neurological, neurosurgical practice.

Injuries to the head are reflected by certain definite physical signs as well as by symptoms recited by the patient. The type of scalp injury found in a head injury is usually directly related to the object causing the injury. A blunt object may strike the scalp without causing any visible evidence of injury. There may be more extensive contusions resulting in swelling and large hematomas which occur in the scalp. Lacerations are common. The scalp is very vascular and extensive lacerations of the scalp may lead to excessive blood loss. Fracture of parts of the skull are exceedingly common in head injuries. It is important to stress the fact that a patient may have an extensive fracture of the skull without any evidence of damage to the underlying brain. Conversely there are injuries in which the brain is severely damaged without any evidence of bony damage. Skull fractures for practical purposes are divided into different groups. There is a simple linear fracture which is merely a crack in the cranium. There are stellate fractures in which the fracture lines radiate from a central focus. Bony fragments may be depressed below the inner table of the cranium. These are known as depressed skull fractures. They may be simple depressed skull

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4 Gurdjian & Webster, Head Injuries (1958).
fractures in which there is no interruption of the overlying scalp. If the overlying scalp is lacerated, then it is known as a compound fracture. The area of the fracture varies. In some basilar fractures the x-rays often do not reveal the fracture lines. Clinical signs may indicate this such as a leakage of cerebrospinal fluid from the ear or the formation of a hematoma over the mastoid area. The latter is known as “Battle’s sign” and is indicative of a basilar skull fracture. Fractures at the base of the skull may cause damage to various cranial nerves. A linear skull fracture through the temporal region may help to confirm the clinical diagnosis of intracranial bleeding from one of the arteries. The skull itself has no important function other than a protective covering of the brain, so the fact that a fracture occurs in the cranium does not indicate there has been any underlying damage to the brain. As a matter of fact, the force of the blow may have been dissipated by the cracking of the skull and actually save the brain from trauma. The significant point in a patient who has had a skull fracture is that there has been a relatively severe blow to the cranium, enough so to at least cause a fracture line.

As a general rule the severity of a head injury is directly proportionate to the length of time the patient is unconscious. On the other hand it is possible to have a serious head injury yet not lose consciousness. This not infrequently is seen in penetrating wounds of the cranium, such as in bullet wounds. Other things being equal, however, the duration of unconsciousness is the best indicator of the severity of the head injury. Injury of the head may be mild without immediate or late effects or it may be so severe that it is fatal within minutes. Disturbances in the state of consciousness may vary from slight confusion to deep coma with loss of all reflexes. The state of consciousness is an extremely important sign in evaluating head injuries. If a patient remains conscious and well oriented following a head injury, the prognosis is very much more favorable than if they are unconscious for a prolonged period of time. Also if a patient loses consciousness some time after the head injury it is indicative that there is increasing intracranial pressure which may be caused by a multitude of neurological conditions. Extreme disorientation alternating with periods of semi-consciousness may occur in injuries to the tips of the temporal lobes. Generally in the patient who has been unconscious for a prolonged period
of time, the return to a state of rational consciousness is gradual. Amnesia is another manifestation of head injury. It has been estimated by Gurdjian and Webster\textsuperscript{5} that approximately eighty percent of patients who have been unconscious following trauma have a retrograde amnesia for the period immediately preceding the injury and for the accident producing the injury.

Brain injuries may manifest themselves by changes in the vital functions. When there is progressing increased intracranial pressure the pulse becomes progressively slower until a breaking point is reached at which time it becomes extremely rapid and soon after this the patient will expire. Respiration is also a vital function that is disturbed in head injuries. Actually patients that die from head injuries usually die from a respiratory death which is due to damage to the medullary center in which the respiratory controlling mechanism is located. The temperature may also be altered in head injuries. In severe head injuries there may be a progressive elevation of the temperature which may result in the death of the patient, if measures cannot be used to control this hyperthermia. Blood pressure is still another vital function which is affected in head injuries. If there is progressing increased intracranial pressure, the blood pressure will continue to rise while the pulse falls. After a certain critical period however, the blood pressure will fall quite rapidly and the pulse will rise suddenly. When this occurs it is thought that changes have occurred in the brain which are irreversible and the prognosis is extremely poor. Surgical shock which is frequently seen in other types of injury rarely ever occurs in uncomplicated head injuries unless there are other associated injuries. Paralysis of parts of the body may appear immediately after the injury or develop after prolonged periods following the initial injury. When the paralysis appears immediately, it indicates there has been damage to the motor portion of the cortex or to the pathways which transmit voluntary motor control. If however, paralysis or weakness develops some time after the injury, it usually indicates that there is increasing intracranial pressure, generally from a collection of blood within the cranium or collection of cerebrospinal fluid which is known as a Hydroma causing localized pressure over the motor area of the brain. Speech disturbances are seen when damage to the speech center of the dominant hemisphere occurs. Patients may have con-

\textsuperscript{5} Ibid.
vulsive seizures immediately after a head injury. This is a rather low figure however and has been estimated by many writers to be between five and ten per cent. The occurrence of convulsive seizures in the acute phase does not have any prognostic value. Certainly it does not indicate that the patient is prone to post traumatic epilepsy as there is no correlation between post traumatic epilepsy and the occurrence of seizures immediately or soon after an injury to the brain.

Headache is the most common symptom of a head injury. Headache soon following trauma is usually generalized but may be limited to one side of the cranium or to the area of the scalp injured. These headaches usually improve in the course of weeks or months. When there is dizziness associated with headaches, the vertigo usually subsides first. Gurdjian states, “Complaints of headaches which last for a longer time may be based on compensation considerations with financial benefits continuing so long as the complaint is validated.”

Many patients who sustain head injuries will complain of vertigo or a feeling of dizziness or light-headedness. This too clears up with the passage of time.

There are many different classifications of injury to the brain. One of the most common is to divide head injuries into three classifications: (1) concussion; (2) contusion and edema, and (3) laceration of the brain and hemorrhage. A concussion may be defined as a very momentary or short period of unconsciousness following a head injury. It is believed that the blow causes a slight jarring of the brain and a brief slowing of the cerebral circulation. The changes which take place in a concussion are reversible. The damage is transitory and the return to normal is usually complete. The patient who has suffered a mild concussion regains consciousness early, but may show some mental confusion. Frequently they will complain of headache, dizziness, nausea and actually vomit. Usually the length of unconsciousness is directly proportionate to the severity of the initiating trauma. It is pertinent at this time to mention the fact that older individuals do not tolerate head trauma as well as younger ones. The rate of recovery is slower and they are more apt to complain of symptoms for a longer period of time. It is thought that this difference is due to changes which occur in the blood vessels of the brain in advancing age. The period of recovery may vary from a few days to several months, and is
dependent upon the severity of the blow, the period of unconsciousness as well as the age of the individual. If the injury to the brain is more severe, it is classified as a "cerebral concussion." This condition makes itself known by a more prolonged period of unconsciousness. There is also cerebral edema with signs of increased intracranial pressure associated with the cerebral edema. There may be a slowing of the pulse as well as an elevation of the blood pressure. This condition usually responds well to conservative therapy, although the period of convalescence may be much longer.

The next classification of head injuries is the third and most severe. In this type there is actually laceration of the brain substance itself associated with hemorrhage. This frequently results in death of the patient or there may be varying degrees of disability if the patient survives.

In addition to hemorrhages into the brain substance itself, it is possible to have lacerations of the blood vessels which are outside the brain. These result in characteristic syndromes. If there is a collection of blood between the dura mater and the brain itself, this collection of blood is known as a subdural hematoma. The acute subdural hematomas usually are associated with laceration of the brain and have an exceedingly poor prognosis. There is another condition which is known as a chronic subdural hematoma. This is a condition which may be caused by a relatively minor blow on the head. It occurs more commonly in older individuals but is also seen in infants. The blow may be insignificant, such as a patient bumping his head on an overhanging door. Usually these patients are not rendered unconscious. The mechanism of this particular syndrome is that there is a tearing of the veins between the cortex and the venous sinuses. A collection of blood then occurs in the subdural space but inasmuch as the bleeding is from the veins in which there is very little pressure, there are no immediate signs of pressure. The subdural space is unique in that there is no mechanism for the absorption of blood from this region. As a consequence a membrane forms around this collection of blood in the subdural space. The hemoglobin molecule then breaks down. As the hemoglobin molecule breaks down it has an increase in its molecular weight and this in turn causes the osmotic pressure to increase and fluid to be drawn through the membranes from the cerebrospinal fluid. This results in an expanding intra-
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cranial lesion. The symptoms which these patients complain of are progressive headaches, accompanied by drowsiness and they may have convulsive seizures, motor weakness and failing vision or double vision. They closely mimic the symptoms seen in a brain tumor. This is a progressive lesion and results in the eventual death of the patient unless surgical means are used to evacuate the subdural hematoma. The average duration from the initial trauma until the time the patient becomes severely enough incapacitated and requires surgery is between ten and twelve weeks.6 Some men have stated that the symptoms can persist for as long as one year. Gardner7 however has shown that this is not true.

There is another type of bleeding which is much more dramatic and rapid in consequences. This is known as an "epidural hematoma." It is caused by a tear in one of the blood vessels in the temporal region or by a tear through one of the large venous sinuses. The classical picture of this epidural hematoma, in which there is a collection of blood between the inner table of the bone and the dura mater, is an initial blow followed by a temporary period of unconsciousness. The patient may regain consciousness within a short time but complain of severe headaches. They subsequently lapse into coma. This occurs usually within the first twenty-four hours but there have been some reported cases of epidural hematomas occurring as late as twenty-one days after the initial injury. Development of an acute epidural hematoma necessitates immediate surgical intervention as it will lead to death from compression of the brain unless surgery is performed to evacuate the hematoma and stop the bleeding. Another complication occasionally seen in head injuries is leakage of cerebrospinal fluid from the nose or ear. This usually stops spontaneously but rarely surgical measures must be taken to stop a rhinorrhea.

In various types of head injuries the cranial nerves may be injured. Not infrequently the first or olfactory nerve is damaged in fractures through the cribiform plate. This results in the loss of smell. In certain fractures of the basal portion of the brain the optic nerves may be cut. This results in loss of vision. The third, fourth and sixth cranial nerves have to do with the

7 Ibid.
extraocular movements of the eyes. Injuries to these nerves will result in double vision. This is seen not infrequently following head injuries but usually clears up. If, however, the diplopia does not clear within a period of six months, it may be considered to be permanent unless surgical measures are used to correct the muscular imbalance resulting from this injury. The central part of the fifth nerve, which is known as the trigeminal nerve, is rarely injured in trauma to the head. However, peripheral branches are commonly contused or lacerated resulting in numbness to parts of the face and anterior half of the scalp. The seventh or facial nerve also may be injured in basilar skull fractures. This results in a facial paralysis in which the patient is unable to move the side of the face involved or to close the eyelid or wrinkle the forehead on that side. The eighth nerve has to do with hearing and sense of balance. Injuries to this nerve make themselves known by loss of hearing or severe vertigo. The remaining cranial nerves are rarely affected in head injuries.

There are symptoms which may persist from the time of injury on while others may develop from varying periods after head injury. The longer a patient goes following a brain injury without developing any symptoms, the less likelihood there is of his developing complications in the future. Patients who have remained free of symptoms for a period of six months following a head injury can be assumed to have made a complete recovery and not have anything about which to be concerned in the future as a result of the head injury he sustained and from which he recovered.

Following relatively severe head injuries some patients will have multiple complaints. This syndrome is known as a "post traumatic encephalopathy." It is characterized by intermittent headaches, feeling of vertigo, nervousness, decreased intellectual function, difficulty in concentration, decreased tolerance to alcohol and there may be personality changes. This post concussional syndrome follows only relatively severe head injuries and it is not seen following concussion. It is thought the changes responsible for the post traumatic encephalopathy are due to anoxemia resulting in changes in the blood cells.

Patients who receive multiple and repeated trauma to the head, such as in prizefighters, who are frequently knocked out, may show a definite change in personality. The common term for
this is known as being punch drunk and is thought to be due to multiple repeated hemorrhages in various parts of the brain, particularly in the frontal lobe. This may result in personality change of the individual. Neither of the two above-mentioned conditions follows a single concussion.

There are a group of complaints which are classified under the title “post traumatic complaints.” These are thought to be due to emotional responses to the accident. These patients complain of frequent or constant headaches and have a multitude of complaints which are not due to structural changes in the brain. These patients are involved in litigation. So long as the litigation is unsettled they continue to have the symptoms and there is little that can be done for them until the law suit is satisfactorily settled. The symptoms then subside spontaneously.

One cannot help but be impressed by the different complaints seen in patients sustaining similar injuries, such as in the athlete who is knocked out in the football field but comes back again to play the same day or continues playing the season without any further complaints. When one sees a patient who has sustained similar injuries through someone else’s fault and becomes involved in litigation, there are a multitude of complaints which usually develop.

It has been reliably estimated that 75 percent of the impending litigation, exclusive of divorce action, has to do with recovery for personal injuries. This is an astonishing figure when one realizes that most industrial accidents are taken care of by Workmen’s Compensation Boards.

No attempt at this time will be made to discuss the field of post traumatic neuroses, which is an extensive field.

Post traumatic epilepsy may develop following severe head injuries. The incidence of post traumatic epilepsy varies greatly with different authors. Walker\(^8\) states that the frequency of convulsive seizures following head injuries is difficult to assess. He feels there is a direct correlation between the incidence of post traumatic epilepsy and the severity of the initial head trauma. Post traumatic epilepsy is more likely to occur in those cases in which there has been actual laceration of the brain or penetrating injury, such as a bullet wound or compound depressed skull fracture with laceration of the underlying brain.

There are many neurosurgeons with extensive experience,

\(^8\) Walker, Post Traumatic Epilepsy (1949).
such as Gardner⁹ and Hamby¹⁰ who do not feel post traumatic epilepsy results from mild head injuries, such as is seen in a concussion. It is true some patients will develop epilepsy following a blow to the head. This per se does not indicate that the blow was responsible for the epilepsy. It is a feeling of most neurosurgeons that the epilepsy would have developed regardless of whether or not the patient has sustained a head injury.

Epilepsy is an indication of malfunctioning of the brain. In order for trauma to be responsible for the development of epilepsy it is necessary to show that the brain was injured at the time of the accident, also that there were focal symptoms developed and also to have focal evidence in the electroencephalogram.

Brain¹¹ has estimated that less than five percent of patients with severe head injuries develop post traumatic epilepsy. Walker¹² has stated in his book that approximately 75 percent of those patients who develop post traumatic epilepsy have their first seizures within three months after the injury.

Guardjian and Webster¹³ have different figures, however, and in their series state that some patients have developed post traumatic epilepsy as long as eighteen years after the trauma. In evaluating this figure one must realize these patients show definite damage to the brain at the time of injury and resulting abnormal focal electroencephalographic changes and not a mere concussion.

Infections following open head injuries formerly were seen much more commonly than they are now. Due to the development of antibiotics and the better distribution of well trained neurological surgeons these complications are becoming progressively rare.

Osteomyelitis of the skull was formerly an extremely serious problem. This, however, is rarely seen at the present time. Post traumatic meningitis also is becoming progressively rare. Intracranial abscesses may be seen in those cases in which there has been foreign material driven down into the substance of the

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⁹ Gardner, W. J. (Personal communication with the author).
¹⁰ Hamby, W. (Personal communication with the author).
¹¹ Brain, Diseases of the Nervous System 337 (1940).
¹² Supra, n. 8.
¹³ Supra, n. 4.
Brain and the debridement has not been adequate. None of these complications, however, hold the threat of death to the patient as they formerly did before the advent of antibiotics medication.

One of the more rare complications of head injuries is the development of a fistula between a carotid artery and the cavernous sinus. This makes itself known by headaches, exophthalmus, which is protrusion of the eye, and as it progresses there is impairment of vision which may even lead to blindness. There is marked chemosis in the conjunctiva in severe cases and papilledema is usually present. A loud bruit can be heard over the head and the patient complains of a whirling or hissing sound in his head. This is a condition which requires surgical intervention.

Head trauma in the newborn is most frequently the result of injury during the birth process. The injury sustained at this time may vary from severe head injuries resulting in death or permanent disabilities to minor injuries with no permanent sequela. The head may be injured as a result of prolonged contractions of the uterus during labor or in some events by the use of obstetrical forceps.

Intracranial bleeding as the cause of cerebral damage has been thoroughly studied. In infants dying at birth or within two weeks, hemorrhages were found in a large percentage but in only one-third of the cases was the hemorrhage severe enough to be the cause of death.

Roberts showed that in spinal fluid studies of 473 consecutive newborns, blood was found in 60 cases. Of the 60 cases, only 26 had signs of head injury and of these 12 died. The 26 with blood in the cerebrospinal fluid showed no evidence of cerebral involvement at any time subsequent to delivery.

Thus we see it is possible to have bleeding into the cerebrospinal fluid around the brain without necessarily having clinical symptoms develop.

The skulls in infants are soft and pliable. As a result of this, many of them will develop depressed skull fractures at the time of delivery. These depressed fractures however, rarely lacerate the brain or underlying dura and are easily elevated without sequela.

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Infants may develop subdural hematomas as a result of birth trauma or as a result of injuries. The mechanism is identical with that which is found in the adults but clinical symptomotology differ, however, in that the suture lines are not united and these children show progressive enlargement of the head. This condition must be differentiated from hydrocephalus.

It must be remembered that infants and children tolerate head trauma better than adults or older individuals and their prognosis is much more favorable.