HEAD INJURY

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Preface

This book was prompted by the realization that no up-to-date, comprehensive text on head injuries and their treatment is currently available. We believe that a practical handbook on this subject will be of great help to many physicians, including neurosurgeons, particularly to those who treat patients with head injuries away from university hospital trauma centers, where the most recent developments in this field are constantly monitored.

The latest technical advances are included here, as well as the classic descriptions of signs and symptoms that have remained essentially unchanged. We have dealt with our subject comprehensively, since we felt it would be an insult to the intelligence of our readers to present a mere "how to" syllabus. On the other hand, an all-encompassing volume that would include much of what has been written on head injuries and that would present all unproved theories and therapies would be poorly suited to practical use. We also felt that a somewhat authoritative text is needed for the use of those physicians who are involved in medicolegal litigation, a fertile and still expanding field.

Many aspects of the treatment of head injuries are still debated, and even time-honored customs and methods are up for scrutiny in the light of recent studies by an increasing number of experts. We have attempted to include impartial presentations of controversial but reasonable views.

The first fully documented head injury was in a Neanderthal man, whose skull was found in a Turkish cave, and from prehistoric times to the present, head injuries have continued to take a tremendous toll. In spite of the sophisticated methods of treatment now in use, many people with severe brain injury still become disabled or die. This fact is a challenge to all neurosurgeons and traumatologists.

Our reliance on literary sources had to be limited and arbitrary, but we have included representative contributions up to 1978.

Mr. Dennis Atkinson performed the difficult task of preparing the illustrations, including the black-and-white rendition of colored slides, which in many instances turned out better than the originals. The editing, typing, and many retypings of the text were accomplished by the tireless efforts of Joy DesGeorges and Camille Everingham.

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L.B.
F.E.G.

Contents

1. Incidence of Head Injuries

Head injuries in the United States, as in all industrialized countries, are frequent. Many of the victims are in the prime of life; their expectancy in terms of both life and productivity is long. According to Caveness [2], in 1974, an estimated 8.1 million Americans had head injuries, of which about 1.9 million (23 percent) were associated with the danger of brain damage. These severe head injuries caused 9.6 million days lost from work and 6.6 million days of hospitalization. Later figures indicate a similar number of injuries, 21 percent of which involve contusion of the scalp and face, 62 percent, laceration of the head, and 16 percent, cerebral contusion or intracranial hematomas. In general, 20 percent of the head injuries were classified as major and potentially life-threatening.

Among men, accidents were the chief cause of death at ages under 35 years and were responsible for more than 50 percent of deaths in the age range 15 to 24. Among women, accidents were the leading cause of death in those under 25 years of age and accounted for more than 40 percent of deaths from age 15 to 19. The more serious head injuries constitute about 3 percent of overall injuries at work, in the home, or elsewhere. However, they make up 15 percent of all injuries incurred from motor vehicles [2]. They are also the major cause of accidental death.

Similar data are available from other industrially advanced countries. It is estimated that the number of head injuries in Europe is 1 million per year, 25 percent of which are of industrial or other origin and 75 percent, the result of traffic accidents.

Of people injured in traffic accidents in the United States in 1966, 70 percent had head injuries. Of these, 66 percent were classified as mild, 24 percent as moderately severe, and 5 percent as severe, with a 5 percent mortality.

An immediate question arises: Who should take care of patients with serious head injuries? Certainly not the family physician, who may know the medical history of the patient in detail and who may have established a lasting rapport with the family. However, neither will help the physician or the patient in an acute emergency. The primary physician caring for a patient with a severe head injury has to be someone who knows how to manage such a patient. And this, in North America, means a neurosurgeon.

Although hematoma is usually completely remediable when handled promptly, Bucy [1] has pointed out that not too many years ago, any patient in the United States outside the large cities in whom an extradural hematoma developed, died. The great increase in the number of neurosur-

geons and their general dispersal throughout the country has now made such deaths far less likely.

Much has been said and written about the treatment of severe head injuries by general surgeons in other countries, particularly in Great Britain. From all available evidence, such treatment is erratic at best. According to such an expert as Jennett [3], a large number of patients with head injuries are not receiving optimal care in Great Britain in spite of the generally high medical standards in that country. The cause seems to be a dearth of neurosurgeons and neurosurgical units or centers.

Under our system of postgraduate medical education, most general surgeons have either no exposure to neurosurgery at all or training that is inadequate for handling a serious situation independently. We are in full agreement with Bucy [1], who believes that improved education and training for all surgeons in the care of patients with head injuries is desirable, but that the creation of second-rate neurosurgeons must be avoided if one is to avoid second-rate care. Furthermore, no amount of preliminary training is likely to protect even the best intentioned general surgeons against malpractice suits in this country if they do not treat head injuries on a regular basis. It will not take much effort on the part of the attorney for the plaintiff to prove that this means ipso facto that the surgeon is not experienced. General surgeons are reluctant to perform neurosurgical procedures for trauma in the United States precisely for this reason.

It is generally considered that the total number of neurosurgeons in this country has reached and perhaps even passed the saturation point. However, large areas, particularly rural areas, are still not being adequately served by neurosurgeons. As a result, patients with head injuries still die unnecessarily for want of expert treatment. Rather than drastically reducing the total number of neurosurgeons, efforts should be made to establish a more even distribution throughout the country.

The era of the solo neurosurgeon is rapidly fading. However, the creation of regional treatment centers—or, at least, of practice groups—is still possible, given encouragement and support. It is unfair and unwise to place the responsibility for the care of patients with significant head injuries in untrained and inexperienced hands.

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2. Mechanism of Head Injuries

GENERAL PRINCIPLES

In trauma to the head, the hair and scalp have some damping effect on the impact. However, the brunt of the blow is delivered to the skull, which is elastic enough to be flattened or indented when struck with a blunt object. The maximum depression occurs instantly and is followed within a few milliseconds by several oscillations. A severe blow to the skull actually results in generalized deformation by flattening in the direction of the impact, with a corresponding widening of the diameter at right angles to the impact line. When the skull is bent beyond its elastic tolerance, it breaks. This may result in a simple linear fracture extending from the center of the impact toward the base. A more severe blow results in a stellate fracture, and an even more forceful blow can lead to a depressed fracture.

The type of fracture depends not only on the velocity of the blow, but also—and more important—on the size and surface of the object striking the skull. A pointed object may perforate the skull, while, on the other hand, an object with a large, blunt end descending on the head at the same velocity may cause a depression. Rarely, fractures can occur opposite the site of impact, such as in the vault or floor of the anterior fossa on occipital or vertex impact [5].

The skull travels faster under the impact than does the brain. Although the brain is frequently contused by the inbending skull at the site of impact, severe surface injuries occur in the brain when it is hurled by inertial pressure against the internal table, particularly against its rough, bony prominences, the crista galli, the major sphenoid wings, or the petrous bones. It is customary to see the frontal poles, temporal poles, undersurface of the temporal lobes, and, less frequently, the occipital poles contused or pulped as a result of these encounters. Similar damage may also be caused by the edges of the relatively unyielding falx and tentorium.

With frontal impacts the brain moves anteroposteriorly; with occipital impacts it moves posteroanteriorly. Impact on the vertex causes movement of the brain along the vertical axis, while lateral impacts cause the brain mass to move from side to side. However, the range of these movements is not the same. The experiments of Pudenz and Shelden [9] revealed that in monkeys, in which the skull and brain are shaped very much like those of humans, the parieto-occipital brain movements are the greatest, irrespective of the direction of the blow.

Pressure gradients at the craniospinal junction result in relative movement of the brain stem. Deeper contusions in the substance of the brain

are due to inertial stresses from relative movements of portions of the brain and pressure gradients.

While so-called coup lesions of the brain occur at or close to the point of impact, the contrecoup lesions develop at approximately the opposite end of the brain.

Gurdjian [5] characterizes the biomechanics of "coup-contrecoup" contusions as follows:

1. Coup contusions are caused by the "slapping" effect of inbending bone during impact.
2. Contrecoup contusions are caused by the movements of the brain against irregular and rough bony surfaces.
3. When the head is relatively fixed, a blunt impact causes a coup lesion with no contrecoup effect.
4. When the head is free to move, a blunt impact will cause contrecoup lesions but little or no coup effect.

The relative frequency of coup and contrecoup lesions under impact at various sites has been summarized by Lindgren [7]. With impacts to the occipital region, coup lesions are rare; the lesions are almost exclusively contrecoup. When the head is struck in the frontal region, lesions occur on the impact site in half the patients, and both coup and contrecoup lesions occur in the other half. In blows to the temporal region, marked impact-site lesions are infrequent (12 percent), but contrecoup lesions are common (68 percent). When the head is struck in the vertex, contrecoup lesions are often orbital. There are also frequent lesions in the corpus callosum [10].

Lindgren [7] explains the rarity of occipital contrecoup lesions in frontal blows by the damping effect of the facial tissues that are very often involved. Our own experience does not corroborate this assumption, since occipital contrecoup lesions in our patients have been just as rare in frontal impact delivered above the facial structures.

The frequent subfrontal brain lesions in cases of frontal impact are not real contrecoup lesions, but rather are the result of deformation changes in the base of the skull [7].

Dural tears, extracerebral and intracerebral hematomas, and surface contusions can occur at either the coup or contrecoup site. They are much more frequent on the side of the impact in depressed fractures. Epidural hematomas arising from a torn meningeal artery, with or without fracture, are almost invariably on the impact side, but contralateral subdural hematomas develop in about one-third of all patients.

The kinetics of coup and contrecoup damage to the brain are complicated and still not completely resolved, despite a great deal of sophisticated research conducted and summarized by Gurdjian [5], Lindgren [7],

and Unterharnscheidt and Sellier [14], to name only a few of the most prominent investigators. After blunt impacts, relative movement of the brain occurs with translational and rotational acceleration [4]. Translational movements are associated with high intracranial pressure (ICP) around the point of impact and negative pressure at the antipole. Many authors emphasize the importance of the negative pressure in the brain opposite the site of impact, the area where contrecoup lesions, frequently more severe than the coup lesions, develop. This negative pressure results in cavitation bubbles, and they or their collapse cause local tissue damage. When gas and vapor pressure increase above the level of hydrostatic pressure, bubble formation occurs, and when the water breaks due to tension or suddenly lowered hydrostatic pressure, cavitation results [7].

Although the cavitation theory is attractive, it has never been demonstrated within a shadow of a doubt in in-vivo experiments. Furthermore, the negative pressure is usually followed within milliseconds by high positive-pressure waves that can also result in tissue damage.

The concomitant development of both positive and negative pressures in different parts of the intracranial cavity are responsible for many controversial reports on general ICP in acute head injuries immediately or very shortly after the impact. There certainly seems to be an immediate increase in ICP on impact. However, there is also a second increase that develops rapidly as a second phase several minutes after the injury. The increase at the time of impact results from acceleration or deceleration of the head and deformation of the skull, the former being more significant than the latter. Lately, it has been proposed that negative pressure can cause damage, not only at the site of the contrecoup, but also at the site of the impact, the latter resulting from the outbending of the skull following the initial inbending during elastic deformation. However, the cerebral damage caused by cavitation as contrasted with the slapping effect of the inbending skull is not yet clearly understood. The development of deep lesions of the brain substance could be explained either by negative pressure and cavitation from the increase in the transverse diameter of the skull on frontal or occipital impact [14], or by the relative movements caused by inertial stresses and pressure gradients in the brain [5].

In addition to linear or translational acceleration, rotational acceleration also plays an important part in injuring the brain. It has been demonstrated that the brain oscillates in a rotary fashion after any significant blow. Furthermore, it has been shown that without significant head impact not only cerebral concussion but also gross contusions and hemorrhages over the surface of the brain can be produced by rotational displacement of the head on extreme motion of the neck [8].

It is still somewhat hypothetical whether or not some types of tissue damage can be attributed to one force and other types to other forces, although attempts have been made to classify certain injuries according to

the various accelerations. According to Unterharnscheidt and Sellier [14], certain cortical contusions are caused by translational acceleration, and primary traumatic cortical hemorrhages are produced by rotational acceleration. With translational acceleration there is a systematic pattern of contusions arranged in a cylindrically symmetrical outline. With rotational acceleration there is a radially symmetrical pattern of hemorrhagic lesions located near the midline. With rotational acceleration the traumatic lesions of the cortex are associated with venous tears rather than with arterial and capillary tears. These tears mainly involve vessels crossing at right angles to the cortical surface. Shearing stresses resulting from rotational acceleration are usually tangential to the surface and tend to rupture radially arranged structures such as penetrating arteries and draining veins. Bridging veins leading to the superior sagittal sinus are also susceptible to tearing. The same mechanism is responsible for the tearing of axons in the white matter.

During impact, the cerebrospinal fluid (CSF), particularly that contained in the major basal cisterns, offers some protection to the brain. However, this protective layer is insufficient in the shallow subarachnoid space around the frontal and temporal lobes, the most frequent sites of contusion. The relative fluid-brain movement is negligible because of the close density of the brain tissue and CSF.

SPECIFIC INJURIES

MOTOR VEHICLE INJURY

Motor vehicle accidents constitute one of the largest single causes of severe head injuries. However, there are considerable differences among the injuries suffered under various kinds of accidents.

Automobile Injury to Pedestrians

As Ryan [11] points out, pedestrians are not "run over" so much as "run under." The standing adult pedestrian's center of gravity is at about the level of the umbilicus and is usually higher than the top of the hood of a car. The impact from the front of a car causes the pedestrian's head and trunk to rotate toward the car. The head strikes the hood or—if the car is traveling fast enough—the windshield and roof (Fig. 2-1). Children, whose center of gravity is below the top of the hood, are pushed over at impact. They can suffer severe head injury when hitting the pavement.

Motorcycle Injuries

Head injuries are frequent, usually from hitting the pavement. Although Ryan's data [11] indicated that the incidence of concussion and of skull fracture was the same for both those wearing and those not wearing helmets, more recent studies have revealed that protective headgear reduces the risk of serious head injury by 50 percent.

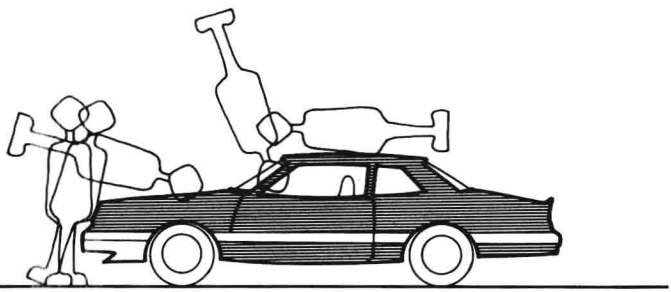

FIGURE 2-1. *Sequence of pedestrian impact in collision with the front of the car (From G. A. Ryan. Injuries in traffic accidents. Reprinted by permission from the* New England Journal of Medicine *276:1069, 1967.)*

Injuries to Car Occupants

Head injuries are by far the commonest injury to car occupants, among whom 70 percent of those injured sustain some head injury, and 26 percent sustain moderate to fatal head injuries [11]. Also, 75 percent of persons with head injuries have injuries to the soft tissues of the face, and 20 percent suffer soft-tissue scalp injuries.

An unrestrained person in a head-on collision continues forward in the vehicle essentially at the velocity at which the car is traveling and collides with the interior front structures of the vehicle when the car has stopped. This is particularly true for the front-seat passenger who is not driving because his or her forward movement is not damped by the steering wheel. It is generally believed that the leading source of injury to the head is the steering wheel or the dashboard, resulting in fracture of the frontal sinus and frontal bones, as well as laceration of the eyelid and eyebrow. However, this occurs mostly in collisions at relatively low speed and in short people or children, in whom the head is low in relation to the interior of the car. It also applies to persons who wear seatbelts. In fully grown adults not wearing seatbelts in a head-on collision at moderate or high speeds, the instrument panel is struck by the knees [11]. Then the torso pivots about the knees and travels forward and upward, the head striking the upper part of the windshield.

In side impacts the side of the occupant strikes the inside of the door. The head hits the upper part of the window glass and frame and the adjacent roof. Consequently, head injuries are primarily caused by impact with the areas of the car interior that are at about head level, such as the upper part of the side door, window frame and glass, windshield, and the rearview mirror.

In fatal traffic accidents, intracranial air is frequently present either in the subdural or CSF spaces, or in the venous sinuses [1]. The association of head and neck injuries is common. The vast majority of the injuries are

localized to a single level at the craniocervical junction, or else they involve the upper two cervical vertebrae.

SPORTS INJURIES

Contact sports can result in severe brain damage when a blow is directed against the head, which occurs, for example, in boxing. Although some of these injuries are relatively mild, resulting in a severe concussion or a mild contusion, their cumulative effect can be devastating, as seen in the punch-drunk boxer after a long career in the ring [3]. Significant injuries to the brain are rare in football, considering the great number of people playing the game. However, they are particularly heartbreaking because they involve young people, usually in perfect health. They are also largely preventable.

Head injuries suffered at football games are closely related to the expertise and physical development of the players, their training, and the degree and quality of supervision. They are most frequent in improvised (sandlot) games and in high school games. They are rare among members of college teams and almost unheard of among professional players.

Alley [2] found that during a single year, 1 percent of all high school football players suffered a head injury, amounting to 4.2 percent of all injuries. Most were mild, requiring no hospitalization or specific treatment, and only 5.6 percent were diagnosed as severe concussion or possible contusion. It is important to note that in over one-third of the more severe injuries, some fundamental football rule was violated, including "spearing" (the use of the head against the body of an opponent), which is as much the fault of the player as that of the coach. In over one-half of the players injured the helmet was not properly fitted.

The most common head injuries sustained while playing football are extradural and subdural hematomas, cortical contusions, and intracerebral clots [13]. In addition, severe cerebral ischemia may result from direct trauma to blood vessels, such as the internal carotid artery, the vertebral artery, the dural sinuses, and important arterial or venous tributaries [12].

Although many severe head injuries in football result from extreme blows to the head, which is often in extreme flexion or extension, with the blow delivered by the opponent's knee or elbow, this is not always the mechanism involved. Severe injury can be caused by violent motion of the neck without any direct blow to the head. How severe brain injury can be caused by extreme motion of the cervical spine without direct injury to the head was illustrated recently by one of our patients, a high school football player who suffered irreversible brain damage without any direct impact to the head. Movie film taken at the game clearly revealed that he was injured when tackled, without falling to the ground. There was an extreme rotational movement of his neck with relatively mild extension of the cervi-

cal spine. Ten minutes later he lapsed into deep coma with decerebrate posturing and almost immediately stopped breathing. Autopsy revealed extensive contusion of the frontal and temporal cortex bilaterally, massive midbrain hemorrhage, and a moderate amount of bilateral subdural hematoma, although the patient had worn a properly fitted helmet. There was no evidence of soft-tissue injury to the scalp and no skull fracture.

PROTECTION AGAINST HEAD INJURIES

It is obvious that no head gear offers satisfactory protection against overwhelming force, such as that encountered by a motorcyclist who hits the ground or any solid object traveling at 60 miles per hour. Helmets worn in contact sports offer considerable protection against head injuries, provided that they are properly designed, constructed, and fitted. Helmets used in one sport may not be satisfactory in another. The basic data for the construction of protective headgear based on measurements, accelerations, and so forth have been presented by Gurdjian and his associates [6].

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3. Pathophysiology of Head Injury

CEREBRAL METABOLISM

Energy is normally produced in the brain almost entirely by an oxidative mechanism. Despite the brain's dependence on this mechanism, it does not have the capacity to store oxygen. Thus, the lack of adequate blood flow of even short duration is incompatible with brain function. Under normal conditions, cerebral metabolism is fueled by glucose, a continuous large supply of which is required to keep the neurons in working condition. Actually, the brain consumes 25 percent of total body glucose. When the plasma level drops to 70 mg/100 ml, the first signs of cerebral dysfunction develop; at levels under 20 mg/100 ml, coma ensues. Glucose is converted into energy in the form of high-energy phosphate bonds and stored as adenosine triphosphate.

When the brain is significantly hypoxic, anaerobic glucose metabolism occurs. However, this mechanism is not sufficiently effective to supply the brain's energy requirement. Anaerobic glucose metabolism results in an increased production of lactate. This is a compensatory mechanism instrumental in supplying the brain with more oxygen, because lactate dilates the blood vessels and increases the delivery of oxygen to the central nervous system (CNS). Severe concussion results in greatly increased lactate production. This increase in cerebral lactate production results from anaerobic glucose metabolism, which in turn is caused by traumatic or hypoxic damage to the normal aerobic pathway using the Krebs cycle [23].

Not much is known as yet about changes in the various enzyme systems of the brain in head injuries, although such changes may have a considerable bearing on cerebral dysfunction, particularly in relatively mild trauma. During the first hour after concussion, in the neurons of the structures close to the site of the impact, there is a transient increase in succinic dehydrogenase activity, associated with mitochondrial functions [21]. Alkaline phosphatase virtually disappears from the walls of small cerebral blood vessels for 2 days after concussion [21].

CEREBRAL BLOOD FLOW

Normally, the rate of cerebral blood flow (CBF) is between 50 and 60 ml/min/100 gm of brain tissue, representing 15 percent of the cardiac output—a remarkable supply of blood for such a relatively small organ as the brain. Under normal circumstances the flow is regulated by metabolic activity. It

almost doubles in the motor cortex on vigorous use of the muscles of the corresponding side of the body.

There are practical ways to determine the blood flow in humans not only through the entire brain and its hemispheres, but also through circumscribed regions (regional cerebral blood flow) (rCBF). Inert radioactive gases (^{85}krypton, ^{133}xenon) are inhaled by the patient or injected in a saline solution into the carotid artery. The use of collimated scintillation detectors allows flow measurement in small individual areas of the brain. Regional differences in blood content can also be depicted on a computed tomography (CT) scan [26].

The physiology of CBF was recently summarized by Lassen and Christensen [19]. Under normal circumstances CBF is constant, apart from functional regional variations that occur despite any change in perfusion pressure. This constant flow is maintained by an autoregulatory mechanism that is based on active vascular responses. The arterioles constrict when the perfusion pressure increases and dilate when the pressure decreases. It is assumed that this autoregulation is mediated by the response to stretch of the smooth-muscle cells of the arteriolar wall rather than by chemical factors or by the influence of the vegetative nervous system. This does not mean, of course, that the blood vessels of the brain are free of nervous or chemical influences. The cerebral arteries and arterioles are supplied with a rich network of sympathetic and parasympathetic fibers. Although their role in maintaining vasomotor tone is presently minimized, they simply must have some kind of function to justify their existence. Recent investigations revealed that even the capillaries are innervated and capable of active constriction. However, maximal stimulation of the sympathetic nerves reduces CBF by only 5 to 10 percent. Further, vasodilation on parasympathetic stimulation is not very striking.

Changes in cerebral arterial O_2 and CO_2 concentration influence blood flow. When the P_{O_2} is low, the flow increases markedly. On the other hand, a decrease of P_{CO_2} results in alkalosis, constriction of the small arteries, and reduction in CBF. Elevated P_{CO_2} levels cause acidosis, vasodilation, and an increase in CBF.

The pressure in the major arterial branches of the brain, such as the anterior, middle, or posterior cerebral arteries, is equal to that in the internal carotid artery, i.e., the systemic arterial pressure. However, in cortical arteries 0.5 to 1.0 mm in external diameter, the pressure is already 20 percent less [6]; further reduction occurs in the smaller intracerebral arteries.

A relatively normal range of arterial blood pressure is required for autoregulation of CBF. The system does not work properly when the perfusion pressure falls below 60 mm Hg (torr). When the pressure rises above 150 mm Hg in an otherwise normotensive subject, the arterioles become dilated beyond their autoregulatory constrictive capacity. This action re-