Med. J. Malaysia Vol. XXX No. 4 June, 1976

Craniocerebral Injuries

by *Abbott J. Krieger*
M.D., D. Med. Sc.

Director, Division of Neurological Surgery College of Medicine and Dentistry of New Jersey, New Jersey Medical School, Newark, and Chief, Neurosurgery Section, Veterans Administration Hospital, East Orange, New Jersey, 07019, U.S.A.

Abstract

Definitive diagnostic procedures and appropriate therapy have been developed for approximately twenty-five percent of patients with craniocerebral injuries of sufficient severity to warrant their hospitalization. The remaining seventy-five percent represent those with damage of the brain resulting from blunt injury followed by drowsiness;-stupor;-coma; as consequence of cerebral contusion-laceration, multiple small hemorrhages, axial distortion, cerebral edema, and increased intracranial pressure. Many of these pathophysiological alterations present unsurmountable therapeutic problems. Increases to an undesirable level of intracranial tension may be effectively managed, and this in turn have a beneficial bearing on some of the other undesirable pathological processes.

THE DEVELOPMENT OF various modes of rapid transportation and the relatively unrestrained manner of deportment of the citizenry of most industrialized nations, has resulted in a progressively increasing number of bodily injuries. Multiplicity of lesions of traumatic origin, in particular those sustained in motor car accidents, is the rule and not the exception. In sophisticated medical institutions groups of surgeons have been formed to accord the injured skillful diagnostic and therapeutic considerations. This widespread development of the Surgery of Trauma has had general acceptance by both the laity and the medical profession. It has resulted in substantial decrease in the morbidity and mortality directly related to injury. Interestingly enough, the care of patients with the sole or principal traumatic lesions being located in the intracranial cavity, remains the responsibility of the neurosurgeon. They have made advances in definitive diagnoses and surgical treatment, however, there still remains many unknowns concerning the pathophysiology of these intracranial lesions of traumatic origin.

Presented as an invited lecturer at the Tunku Abdul Rahman Institute for Neurological Sciences, August 1975.

As obtains in most segments of medicine, the interpretation of the clinical features of patients with craniocerebral injuries and their correlation with the pathological state constitutes our basic knowledge of those with this type of lesion. It has become conventional to classify these for facility of presentation, however, it is recognized that more often than not the classes fuse or meld. Moreover, some of these categories in common use are based on abnormal clinical features alone whereas others are derived from demonstrable anatomical derangements.

A. Laceration of Scalp

B. Simple Fracture of Skull (linear or depressed)

C. Compound Fracture of Skull (vault and base)

D. Penetrating Missile Wound of Head

E. Closed Head Injuries
   1. Mild Cerebral Injury (concussion)
   2. Moderate Cerebral Injury (cerebral contusion)
   3. Severe Cerebral Injury (cerebral laceration)

F. Intracranial Hemorrhage:
   1. Extradural Hemorrhage
   2. Subdural Hemorrhage
   3. Subarachnoid Hemorrhage
   4. Intracerebral Hemorrhage
   5. Intraventricular Hemorrhage

In some instances the location and extent of the intracranial lesion or lesions is evident upon routine examination, whereas in others the pathophysiological factors are obscure. Under the latter circumstances in particular, one should accord "little things" full consideration. A few examples: the determination of the site of impact to the head by the inflicting object is quite important. Here there is frequently transitory deformation of the skull, with or without resultant linear fracture but sufficient to detach the dura mater. Crossing vascular channels from the diploic to the dural circulation are interrupted thus setting the stage for the development of an extradural hematoma; the relatively rapid appearance of a swollen upper eyelid with the overlying skin having a blue tint and this associated with evidence of a blow to the occipital scalp commonly signifies multiple parallel linear fractures in the bony roof of an orbit and a concomitant homolateral contusion-laceration of the frontotemporal region of the brain; ataxic breathing associated with stupor and paucity of muscular movements strongly suggest impaired medullary function resulting from pressure by a hematoma within the posterior cranial fossa; excessive sweating associated with generalized stiffness of the extremities, but not evident decerebration, appearing shortly after injury suggests intraventricular hemorrhage. In all events, every abnormal feature that can be demonstrated in each patient should be assessed and recorded even though it may require repetative examinations to determine the relative significance of these in the total clinical picture.

A cursory appraisal followed by cerebral arteriographic examination is to be condemned. This somewhat definitive examination does serve as an excellent diagnostic adjunct but should be used with discretion. It serves as an aid in establishing the presence of or excludes the possibility of a mass lesion of surgical significance. It is to be recognized that only about one-fifth of the total group of patients who have sustained some type of craniocerebral trauma are in need of surgical therapy. These consist of those with open wounds of the vault of the skull, an occasional instance of compound fracture involving the nasal cavity and/or ears, a variety of blood collections or hematomas of the intracranial compartments, and focal contusion of the brain with edema adjacent to the lesion. Even though surgical elimination of such focal lesions may be considered adequate, this frequently resolves only a segment of the total problem. It is, therefore, necessary to maintain an attitude of expectancy interlarded with repeated observations, throughout the patient's illness.

Information regarding the details of the accident, obtained on entry to the hospital, is often fragmentary, second handed, and frequently unreliable. The victim, commonly a young to middle aged male, is transported by ambulance from the site of accident to the hospital. Even the attendant in charge of the ambulance may not be able to give an accurate account of the sequence of events that ensued enroute to the emergency area of the hospital, in particular changes in the state of awareness or consciousness. It is, therefore, mandatory that the doctor in attendance be familiar with abnormal physical features that may indicate the site of dysfunction of the brain. He should also be sufficiently versatile to make a determination of the nature and extent of damaged parts other than the head and assign those so injured to appropriate members of the surgical team.

Those with lacerations of the scalp, compound fractures of the vault of the skull and the patients who have had a period of unconsciousness followed by a lucid interval and subsequent drowsiness to stupor, should have definitive diagnostic procedures as indicated and prompt surgical attention. The remaining group consists of those with stupor to coma without abnormal physical features suggesting a focal lesion.

There are three important abnormal findings to be looked for during the initial as[essment: degree of alterations in the conscious state, the status of the eyes, especially the pupils and alterations, usually diminution, in movements of the extremities. Among these three the state of consciousness of awareness stands preeminently. Conventionally five grades have been recognized, i.e. alert, drowsy, stuporous, coma, and moribund, however, sharp lines of distinction do not separate these stages. A complete understanding of the changes in the complex cerebral mechanism that results in loss of awareness and contact with surroundings remains obscure. The accumulated evidence indicates that the function of the reticular formation of the upper brain stem is in part at least to regulate the state of consciousness. The clinical evidence in the human suggests more widespread nuclear areas of influence. The conscious state varies from patient to patient. In general the more profound the loss of awareness the more severe the cerebral injury. Furthermore improvement is usually estimated by the patient becoming more

responsive to all types of stimuli, remaining on a plateau when reaction to surrounding remained unchanged and worsening if the altered state of unconsciousness become more profound. Consequently the clinician relies heavily on this index in estimating the extent of the cerebral damage. In some patients of the moderately to severely injured group treatment is instituted immediately upon entry before embarking on the examination. This therapeutic urgency is posited on the presence of excessive bloody secretions which partially obstruct the airway. This complication may be corrected by positioning the patient on one side and complete toilet of the nasopharynx by suction. If this is not successful, tracheostomy should be performed forthwith.

After mandatory emergency issues have been resolved, a careful and thoughtful examination should be made. Usually drowsiness to stupor prevails. The status of the pupils is determined. The central pathways for both pupilloconstrictor and pupillodilator effects course through the upper brain stem. Lesions rostral to the diencephalon have little influence on pupillary and ocular functions. More caudally situated lesions are not infrequently associated with significant pupillary changes. Importantly, among these are the dilatation of a pupil secondary to compression of an oculomotor nerve by laterally located hematoma and constriction of the pupils in patients with pontile lesions. Observation of other oculomotor nerve dysfunctions may aid in diagnosis, in particular a laterally rotated optic bulb. In addition oculocephalic and oculovestibular reflexes may furnish information regarding the neural level of the traumatic lesion implicating the mesencephalic-hindbrain complex.

The third of the aforementioned trio of areas to be critically examined namely the motor components of the extremities frequently show unilateral dimunution of spontaneous and/or limited movements subsequent to noxious stimuli. Hemiparesis to paralysis is not difficult of demonstration. Stretch reflexes are variable consequently alteration of these may not represent valid evidence of damaged myoneural connections, however, the sign of Babinski if present on the side of suspected paresis, is an important finding. Mild to moderate rigidity of all extremities suggests impending decerebration secondary to uncal herniation. The frequently quoted "flexion posture" of the upper extremities representing decortication and the "extension posture" with internal rotation of these parts as indicative of decerebration does not hold in clinical practice. One of these attitudes may alternate with the other in a matter of seconds in patients who subsequently are shown to have tentorial herniation of the mesial posterior temporal lobe of the brain.

If after careful assessment of the evidence derived from repeated observations and examinations a decision regarding surgical therapy cannot be reached, arteriography should be performed. Although a subdural collection is demonstrated by the examination this does not necessarily settle the issue of therapy. It is to be remembered that in most instances the subdural hematoma represents a complication of a surface brain lesion and that its surgical evacuation in some way not be followed by appreciable change in the course of the patient's illness. Surgical mortality remains high in the group with so-called acute subdural hematoma consequently serious consideration should be accorded the possible effectiveness of this therapeutic approach during the early phase of illness. This viewpoint does not hold for extradural hemorrhage which should receive surgical attention prior to the appearance of decerebration or fragments of this posture. Evacuation of the hematoma in this anatomical position should be followed by prompt recovery.

After resolving a part of the total problem of some patients by surgical evacuation of a blood clot and/or this in combination with removal of contused and/or macerated brain, factors which are instrumental in the production of stupor to comatose in many with closed head injury still persist. The two outstanding ones are damage to important cell masses of the brain stem and cerebral edema. Dehydrating chemicals may be beneficial in some instances, however, in others the effects are transitory at best. The major problems are said to be brain swelling and cerebrovascular congestion associated with an increase in intracranial pressure. Accumulated experimental and clinical evidence indicates that uncontrolled intracranial hypertension is a major contributing cause of death in patients with craniocerebral trauma. The anatomicophysiological basis for this is that the intracranial volume is essentially fixed. These constituants are blood, brain, and cerebrospinal fluid consequently an increase in the volume of one is at the expense of the others. Swelling of the brain following cerebral injury results from brain edema. As a consequence of this, intracranial tension is increased thus imposing cerebrovascular congestion which in turn adds to the total intracranial volume. At some point the intracranial pressure rises beyond that compatable with life. Imposing a relatively high degree of intracranial pressure on a normally functioning brain is quite different from creating a comparable set of intracranial circumstance in a patient with

contusion of the brain implicating the upper brain stem. Adding even small increments of pressure may produce medullary failure. Untreated compression of the brain by an extradural hematoma resulting in a fatal outcome has been cited by some as an example of death due to intracranial hypertension. This oversimplified examination does not take into account the concomitant edema of the white matter underlying the area of compressed brain, the axial shift of the upper brain stem with the herniation of the uncus through the incisura tentorii and the resultant mesencephalic hypoxia. No doubt the accompanying increase in intracranial pressure under the conditions aforementioned should be regarded as a component of the complex mechanism that resulted in death, however, several pathophysiological mechanisms are brought into play. Since our knowledge is grossly defective regarding the interplay of these pathophysiological processes, treatment of a relatively large segment of patients with closed head injuries is largely empirical. There is ample clinical evidence to support the notion that dysfunction of the brain consequent to blunt trauma of the head becomes less in evidence if the cerebrospinal fluid pressure is maintained within normal limits. During the past six decades various methods have been developed and used to effect this end result, however, none of these have proved to be a therapeutic answer to the multiple problems presented. For the past fifteen years, it has been recognized that continuous recording of the intracranial tension furnishes a more knowledgeable approach to maintaining the intracranial pressure at a normal or physiologically acceptable level.

Reference

1. Lundberg, N., Troupp, H., and Lorin, H.: Continuous recording of the ventricular-fluid pressure in patients with severe acute traumatic brain injury. A preliminary report, J. Neurosurg., 22: 581–590, 1965.