The Search for Optimal Management of Head Injury

J. DOUGLAS MILLER, M.D., Ph.D.

Lind Lawrence Professor of Neurosurgery, Medical College of Virginia, Health Sciences Division of Virginia Commonwealth University, Richmond, Virginia

Introduction

Head injuries of sufficient severity to bring the patient to a hospital occur in more than one per hundred of the population every year. The toll in death and disability is staggering; the majority of patients are young wage-earners with families so that the total socio-economic burden can hardly be guessed at. Much emphasis is laid on the concept that physical disruption of neurons in the brain is not a reversible process and that regeneration within the nervous system to the point of functional recovery does not occur; however, this rather dismal view of head injury is at variance with the facts. Most cases of head injury reaching the hospital are associated with relatively minor degrees of primary brain damage from which full functional recovery may be expected even though there are minor residual neurological signs. The major problem for the physician treating head injuries in hospitals is that secondary complications may supervene and change a relatively minor head injury case into a major disability problem or even a fatality. For the management of head injury considerable emphasis should be laid on the prevention of secondary complications; such an approach is outlined in this article.

The Rationale of Head Injury Care

Except with minor trauma, most head injuries result in some observable degree of sudden neurological deterioration followed by a trend towards recovery of neurological function. The limit of that recovery is set by the pretraumatic status of the brain (as affected by previous injury, by degenerative disease, or by congenital defects) and by the amount of structural damage to the brain caused by the primary impact. Immediately after injury, however, and continuing well into the recovery phase for days and even weeks the patient is at risk from a variety of secondary insults to the brain, any one or combination of which may further limit the chance of recovery or may, at worst, transform a trivial head injury into a fatal process.

Early secondary insults stem from possible hypoxic hypoxia due to poor airway care at the scene of the injury or during transport to the hospital, or to arterial hypotension as a result of blood loss, visible or occult. These insults superimpose cerebral hypoxia on an already damaged brain which, due to the injury, may lack the protective physiological mechanisms which normally maintain cerebral energy metabolism during physiologic stress. A further group of secondary insults relate to the formation of secondary intracranial mass lesions (hematoma, swollen contusion) which produce both brain shift with distortion and raised intracranial pressure. Thus, the syndrome of brain compression may soon overshadow the effects of the original head injury. Another group of secondary cerebral insults are infective in origin and occur usually as a result of dural penetration by the injury process, producing meningitis or even an abscess of the brain in epidural, subdural, or intracerebral locations. While the patient remains unconscious he is at risk from chest and urinary tract infection, and from fluid and electrolyte imbalance, all of which may produce neurological deterioration.

The optimal care of patients with head trauma
hinges on a comprehensive and rapid diagnostic assessment, and appropriate surgical and medical management which aims to anticipate rather than just treat secondary insults and complications of the primary head injury. The loss of any patient with head injury who arrived at the hospital able to talk has to be regarded as a failure of such a management regime. Although some such cases continue to occur even with the best care presently available, the goal of head injury management should be the maximum recovery possible consistent with the preexistent state of the brain and the degree of primary brain damage.

Diagnostic Assessment

The process of diagnostic assessment of patients with head injury may be regarded as a search for the answers to six questions, beginning as the patient reaches the hospital emergency room.

1. What is the physiological status of the patient?

During transport to the hospital and on arrival in the emergency room the primary consideration must be the patency of the airway, assessed ideally both by visual inspection and by measurement of arterial blood gases, with immediate relief of any obstructive problem. The next most urgent consideration is the arterial blood pressure. Systemic arterial hypotension is scarcely ever due to head injury, and a low blood pressure must prompt a thorough search for sources of visible or occult blood loss. Correction of these problems may produce dramatic improvement in neurological status.

Thereafter, a rapid assessment must be made of the total extent of injuries which should be carefully noted in writing, preferably supplemented with drawings or diagrams. Special attention must be paid to the possibility of coexistent head and neck injuries. Cervical spine damage must be thought of in all patients with head injury but especially those who have no leg movement yet good facial movement in response to pain. Another pitfall in the assessment of the comatose patient is the soft tissue injury seen early before any substantial swelling has occurred. All major joints should be tested for abnormal mobility and no plans should be made to move the patient until spinal trauma has been excluded. Visual inspection will need to be supplemented by plain x-rays in the emergency room, and after recording the total burden of injuries suffered by the patient, a more accurate assessment can be made of blood and fluid transfusion requirements.

As a general principle, treatment of any injury should be definitive from the outset; there is nothing less impressive than the situation in which it takes four to five days to discover one by one all of the injuries suffered by a patient. In this respect the history of trauma is vitally important as multiple injuries can certainly be expected after a severe car wreck or a fall from a considerable height. The problem of multiple injuries is a common one; about one third of patients with serious head injuries have other injuries as well, and 80% of those patients in car accidents who have multiple injuries have serious head injury as part of the clinical picture. For this reason, patients with serious head injury should always be undressed completely in the emergency room in order to ascertain the full extent of injuries before any move is made to take the patient out of the room for x-ray or other maneuvers.

2. What is the neurological status of the patient, and is it changing?

The neurological evaluation of the comatose patient with a head injury is not difficult if two principles are followed. You can test only what can be tested and you must express your findings in objective terms, as your findings may have to be compared with those of others. The patient must be challenged verbally first of all to reply to questions or if there is no verbal response, to obey commands. Failing such a response a painful stimulus must be used and the response observed. Pain must be applied centrally, to the trunk, and to all four limbs, in turn, so that the best motor response can be determined. The motor response can be graded as obeying commands or localizing painful stimuli, both of these responses being regarded as purposeful. On the next level lower, the arm may flex as a withdrawal type of response to pain which can be classified as a semipurposeful response. Abnormal motor responses consist of those where there is an element of pronation combined with arm flexion, usually called a decorticate response, or arm extension combined with pronation (decerebrate response). Finally, at the extreme, motor responses to painful stimuli may be completely absent and the limbs flaccid.

The pupils should be examined for inequality in size under normal lighting conditions and the response tested both directly and consensually to light to distinguish between optic nerve and oculomotor nerve damage. Oculocephalic or oculovestibular responses should be tested only after ruling out cervical spine and tympanic membrane damage respectively.
Normal findings in awake patients will be suppressed eye movement in the case of the oculocephalic response, or nystagmus in the case of the oculovestibular response. In patients who are unconscious the intact doll's eye response and the ipsidirectional tonic conjugate oculovestibular response (to irrigation of the ear with ice cold saline) both imply that brain stem structures responsible for eye movement are largely intact, while dysconjugate or absent responses to both forms of stimulus point to damage in the brain stem.

In this examination scheme there is no place for the use of terms such as obtunded, stuporous, or semicoma nor nor is there any place for the recording of tendon reflexes or the plantar response. In the hours following head injury funduscopic examination is of value only to detect preexisting disease or possibly subhyaloid hemorrhage in the patient in whom there is doubt concerning the differential diagnosis between head injury and subarachnoid hemorrhage.

The value of serial, objectively expressed, neurological assessment can hardly be overemphasized. In this regard we have found very useful, and strongly recommend, a standardized form such as is illustrated in Figure 1. This form which is filled out by the nursing staff in the intensive care unit has been adapted from one which was developed by Dr. Graham Teasdale at the University of Glasgow, Scotland, after an extensive study to determine that terminology which was least ambiguous and most consistently recorded no matter whether by doctors, students, or nurses.

The neurological findings on admission or shortly after admission to the hospital must be compared with any evidence to suggest a different neurological status shortly after the injury. This is vital information which can be gleaned from witnesses and ambulance men. The patient's relatives must also be questioned to construct a picture of the pre-injury neurological status of the patient and reference to school records is very often of considerable help. Finally, two factors commonly contribute to errors in the clinical diagnostic assessment of patients with head injury. These are epileptic seizures and overuse of alcohol. Early epileptic seizures in the first few hours after injury are oftentimes a cause of dramatically sudden neurological deterioration followed by an equally rapid recovery of consciousness in many cases. Conversely, the cause of coma after head injury is more often attributed wrongly to the ingestion of alcohol. Blood alcohol levels are not usually available until the following day, but in a recent, very large series of head injury patients in whom blood alcohol levels were measured, the conclusion very clearly was that it was wiser at all times to proceed on the assumption that the patient's low conscious level was due to head trauma rather than alcohol consumption.  

3. Does the patient have an intracranial mass lesion?

Traditional teaching on head injury management has laid much emphasis on first looking for evidence of neurological deterioration to suggest that an intracranial hematoma or other mass lesion is developing, then pursuing the appropriate diagnostic measures to localize the mass lesion (Fig 2). With the advent of a new, noninvasive diagnostic technique, computerized axial tomography (CT), this view needs to be modified. The central question in many clinical cases has to be, If this patient deteriorates neurologically from this point, what will his neurological function be? Deterioration in a patient who is already decerebrate would render him flaccid and apneic and cannot be risked.

Some three and a half years ago Dr. Donald Becker instituted a uniform protocol for all patients with head injury who entered the Medical College of Virginia unable to obey simple commands. All such patients were studied immediately by twist drill ventriculography, by angiography, or more recently, by CT scan. In this way some 40% of patients were discovered to be harboring intracranial mass lesions and were taken immediately to surgery to have these removed. Patients who arrive at the hospital after head injury still able to obey commands still have some form of intracranial study within 24 hours of admission. By proceeding directly to these intracranial radiological studies, it is hoped to anticipate neurological deterioration due to expansion of an intracranial mass lesion.

Two criticisms of this approach may be made: first, lesions may be detected which might never have caused any trouble for the patient and second, lesions may develop later which would have been undetected by the early routine studies. Complete answers cannot be given to such questions at this stage as experience with CT scanning is still limited, but our viewpoint is that it is always better to know in advance that a patient has an intracranial mass lesion; whether or not it should always be operated on is another question. We know, however, that the presence of an intracranial mass lesion presents great
Fig 1—Neurological evaluation and progress sheet for intensive care unit. (Fluid balance chart on reverse).
Fig 2—Computerized axial tomograms in patients with head injury. Top left. Extracerebral mass lesion; acute epidural hematoma, underlying edema. Top right. Extracerebral mass lesion; acute subdural hematoma. Bottom left: Dense intracerebral mass; intracerebral hematoma. Bottom right. Large, low density lesion in brain; cerebral edema formation.
danger to the patient during anesthesia, during sleep, or when there is any respiratory obstruction. Even if intracranial pressure is normal under resting conditions, any cerebral vasodilation causes severe intracranial hypertension. Our experience to date is that intracranial hematomas following trauma are virtually always present when the patient is first admitted to the hospital, while brain swelling associated with cerebral contusion undoubtedly develops after admission during the first three to five days following injury. For that reason we believe that follow-up CT scans are also vitally important.

4. Is the dura penetrated?

The importance of this question relates mainly to the risk of infection following injury, but another common complication of penetrating head wounds is the formation of intracerebral hematomas. Dural penetration may be linked to the exterior not only through scalp lacerations and fractures of the skull vault but also through the nose and ears, through fractures of the anterior and middle cranial fossa, and through small puncture wounds in the eye and cheeks with penetration of the base of the skull. These latter injuries are very important to remember as the small puncture wounds may soon become obscured, for example, by swelling of the eyelids. For this reason the history of injury is very important when there is any suggestion that a sharp object has penetrated the head or face for any appreciable distance. In such injuries the patient frequently has never been unconscious so that there is always a real danger of dismissal from the hospital without recognition of the problem.

External examination may immediately reveal that there has been dural penetration; cerebrospinal fluid (CSF) or brain tissue may be seen issuing from a small scalp wound or CSF may be seen coming from the nose or ear. Indirect evidence occurring a few hours after injury is the formation of periorbital bruising (raccoon eyes) or bruising behind the ear (Battle sign) which indicate the presence of fractures of the anterior and middle cranial fossae respectively. These appearances are not in themselves indicators of dural penetration, however, and this may be extremely difficult to judge as CSF leakage may be only transient or may never occur. Plain x-rays of the skull may yield further clues to the presence of dural tearing; the appearance of an aerocele or spontaneous ventriculogram is certain evidence of a dural fistula. When skull x-rays are carried out to detect intracranial air, the crucial film is a brow-up lateral view where the air will be found on the undersurface of the frontal bone or in the frontal horns. Deeply imbedded bone fragments or foreign bodies in the head will also provide evidence of dural penetration, but it must be remembered that many materials such as wood do not appear on x-ray and, in some instances, exploration of the head wounds will have to be based only on a history of deep penetration of the head by some object. It is essential in any case to carry out toilet, debridement, and thorough exploration of compound fractures of the skull, though great care is needed for those fractures which overlie the great venous sinuses.

5. Is the intracranial pressure elevated?

The only way in which this question can be answered is by direct measurement of intracranial pressure, preferably from the lateral ventricles or cerebral epidural or subarachnoid space. Lumbar puncture is not justified as a means of obtaining an answer to this question for two reasons. First, if the patient has a sizable mass lesion in the supratentorial compartment, performance of lumbar puncture may precipitate tentorial or tonsillar herniation. Second, under these same circumstances, the recorded spinal fluid pressure will not nearly approximate the true level of intracranial pressure so that this type of measurement from the lumbar subarachnoid space is not only misleading but dangerous. There are no clinical neurological signs which will indicate a particular level of intracranial pressure. The so-called signs of raised intracranial pressure, pupillary dilation, extensor rigidity, bradycardia, and arterial hypertension are all manifestations of brain herniation, not indicators of any particular level of intracranial pressure. Papilledema which can truly be said to result from intracranial hypertension does not appear early after head injury and is not, in any case, identified with any particular level of intracranial pressure. If the answer to this question is important, therefore, the only solution is for supratentorial pressure to be directly measured. This has been done in a series of 160 patients seen at the Medical College of Virginia over a three-year period and the results from this large series can be reasonably confidently extrapolated to experience with head injuries as a whole. On admission to the hospital, virtually all patients with surgically significant intracranial mass lesions will have some degree of elevated intracranial pressure, at least over 10 mm Hg but going up in some cases as high as 70 mm Hg, particularly in patients with acute subdural hematoma. Of patients with diffuse brain injury...
injury without an intracranial mass lesion more than two thirds will have some degree of intracranial hypertension on admission, although in most cases the increased pressure will be moderate, in the range of 10 to 20 mm Hg. Even if surgical masses are evacuated promptly and patients are ventilated to avoid any hypoxia or hypercapnea and given steroids, raised intracranial pressure will still present a problem in many instances, in approximately half of those patients who have had mass lesions removed, and in approximately one third of those patients with diffuse brain injury. Of patients who die of severe head injury, nearly half do so because of fulminating intracranial hypertension and associated brain shift and ischemia. In the survivors, high intracranial pressure is associated with a poorer outcome.

It is our belief, therefore, that given adequate equipment and personnel, optimal assessment of patients with severe head injury who remain unconscious should include direct measurement of intracranial pressure on a continuous basis for two or more days as indicated. The technique is not without risks, however, and these risks, mainly of infection, must be weighed against the expected benefit of the technique which should be restricted to centers having an intensive care unit with full, round-the-clock, medical and nursing staff.

6. What is the extent of damage to the brain?

In determining the final outcome from head injury the extent of the injuries scattered throughout the brain may be of more importance than the severity of injury at any one place. The neurological examination in the early stages after injury may be more a reflection of the strategic location of the most severe cerebral injury and its capacity to depress the total nervous system than an accurate index of the total extent of areas of brain damage. Nevertheless, the length of time that a patient is unconscious and the length of time until recovery of full continuous memory (the period of posttraumatic amnesia) seem to reflect increasing extent of diffuse brain damage in contrast to persisting hemiplegia which is a manifestation of focal damage in a crucial location. The electroencephalogram (EEG) is of little or no value in this respect and the procedures of ventriculography, angiography, and ultrasonic studies all point only to the most severely involved hemisphere of the brain or to shift of the brain by an extra-axial mass lesion. With the advent of CT, it is possible for the first time to look in a morphological way at the extent of brain damage and it is to be hoped that a clearer prognosis will be possible by the early identification of those patients with, for example, significant bilateral brain lesions.

In the physiological study of the central nervous system (CNS) the use of multi-modality evoked potentials is looking extremely promising, with some capacity to determine the extent of brain damage located both in the brain stem and in the hemispheres. Good correlation has been obtained between persistent focal neurological deficit and local abnormalities in evoked potentials and between prolonged coma and poor outcome and particular combinations of abnormal evoked responses. The main limitation of this technique is that there is no way as yet to perform specific evoked potential responses which will determine function in the frontal lobe of the brain; thus about 50% of the volume of each hemisphere is unavailable for this type of neurophysiological study. Measurements of cerebral blood flow in multiple regions of the brain are now possible and stress tests can be done to see how these areas respond to elevation or lowering of the arterial Pco2 or blood pressure. When such studies are done, sometimes areas of impaired blood flow regulation will be seen in the frontal and temporal parts of the brain, corresponding with those areas which are most frequently affected by cerebral contusions after blunt head injury.

Surgical Management in Head Injury

Surgical procedures are required in only a minority of head-injured patients admitted to hospitals, but it is important that if they are going to be done, they should be done for clear indications as expeditiously as possible and should lean towards being radical rather than inadequate. Burr hole exploration in acute head injury is only an incomplete approach to diagnosis; it is virtually never an adequate therapeutic measure.

There are two main indications for surgical intervention in patients with head injury. The first is intracranial infection for which the debridement and repair of penetrating wounds of the skull is carried out with the aim of forestalling this condition. Thus, the principles in this form of surgical treatment are the same as those for compound wounds elsewhere. The second indication, which is more particular for CNS trauma, is intracranial mass lesions which require surgical evacuation. There is no question that these lesions must be removed immediately if associated with any neurological deterioration, but it is
also our belief that any sizable lesion associated with shift of the intracranial structures should be evacuated even if at that time there are no definite indications of neurological deterioration. In a seriously ill patient any degree of neurological deterioration may push the patient beyond the recoverable stage, and even if the mass itself does not immediately jeopardize the patient, the superimposition of any one of several types of secondary insults may suddenly precipitate the patient into fulminating brain compression. Examples of this are cerebral vasodilation due to hypoxia, hypercarbia, the administration of volatile anesthetic agents, and even a sharp increase in body temperature.\(^4\)

The surgical approach to the penetrating wound is governed of necessity by the site of the damage, but adequate exposure of the wound must be obtained and enough bone removed to permit a clear view of all areas of possible contamination. If the dura is extensively torn, it should be repaired, if necessary, with a graft taken from temporal fascia or from the thigh. All imbedded bone fragments should be removed and x-rays on the operating table are useful to ensure that this end has been satisfactorily achieved. Deeply imbedded metallic fragments such as bullets need not be removed if they are difficult of access surgically as it is considered that the risk of infection from these metallic fragments is much less than from indriven bone. There is a particular risk of intracranial infection in penetrating wounds which involve the frontal sinuses.

In the surgical approach to decompression of intracranial mass lesions there is little place for the use of multiple burr holes once the location of the mass has been established by ventriculography, angiography, or CT scan. In these cases a very large bone flap should be turned which will expose the frontal and temporal lobes and go posteriorly beyond the limits of the frontal lobe (Fig 3). If there is in addition to extracerebral hematoma a pronounced degree of frontal or temporal tip contusion and necrosis, this should be removed. In some cases the swelling is entirely intracerebral and in these instances a frontal or temporal lobectomy of at least 5 cm should be carried out.

Medical Management of Head Injury

The medical management of the patient with head injury is an exercise in maintaining the physiological milieu of the patient as stable as possible. Since abnormal respiration is a common problem soon after injury we believe that all unconscious patients should be artificially ventilated on a volume respirator using a high tidal volume for several days at least after the injury or until they are clearly regaining consciousness. For patients who resist the ventilator small doses of morphine or chlorpromazine may be used or muscle relaxants may be required. In the latter event, however, the neurological evaluation can be done only in those periods of time when the effect of the muscle relaxant has worn off.

We prefer to adjust the minute volume to maintain an arterial PCO\(_2\) level of 25 to 35 mm Hg and also adjust the fraction of inspired oxygen to keep arterial PO\(_2\) well over 70 mm Hg. Although there is no hard evidence that steroids exert a favorable influence on severe acute brain injury, we currently give patients a standard dose of dexamethasone (4 mg every six hours). Recent reports from West Germany have suggested that higher doses of steroids may be efficacious in head injury.\(^{14,15}\) This aspect of head injury care is currently being studied in several centers. It has been our policy to give prophylactic anticonvulsant therapy in the form of phenytoin 100 mg three times daily to all patients with serious head injury, to those patients with penetrating injuries who have dural tearing, and in particular where there has been an intracranial hematoma, infection, or missile wound.

Intracranial pressure is monitored continuously for at least two days and longer as required. In-

![Fig 3—Extent of craniotomy required in patients with mass lesions (acute hematoma).](image)
Increased intracranial pressure is treated by increased hyperventilation, by controlled CSF drainage against a positive pressure, and by intravenous mannitol therapy alone or in combination. Present indications for treatment of intracranial hypertension are an intracranial pressure rise over 30 mm Hg or any increase in intracranial pressure associated with neurological deterioration.

The Outcome of Severe Head Injury

Of the 160 patients with blunt head injury who have been treated with this policy of early diagnosis and evacuation of mass lesions followed by artificial ventilation and intracranial pressure monitoring, the results have been extremely gratifying. Sixty percent of the patients have recovered to the extent of returning home (8%) or remaining in hospitals or nursing homes in a permanent vegetative state (2%). These results compare favorably with results reported in patients with similar degrees of head injury from other centers and of particular interest is that this policy of early and radical surgical decompression has not been associated with an increase in the number of permanent vegetative survivals.

In examining the factors which regulate the outcome from severe injury we find that age is important in that older patients are much more prone to die of infective or other systemic complications of the head injury. Signs which indicate brain stem dysfunction (absent pupillary light response and impaired or absent oculocephalic response) are strongly associated with a bad outcome. The presence of decerebrate or extensor rigidity or loss of the motor response signifies a bad outcome only in those patients without intracranial mass lesions. In the patients with mass lesions a good recovery is still possible despite a combination of all three adverse signs, this being in the small group of patients in whom such signs relate to brain compression rather than to the primary injury and in whom the brain compression is relieved early, before secondary brain dysfunction has become irreversible. Intracranial hypertension occurring on admission or, more important, during the intensive care unit management phase does carry adverse significance for the patient, and should be treated. Increased intracranial pressure is, we believe, related to the extent of brain damage rather than to any particular location.

In the evaluation of outcome of penetrating wounds emphasis must be placed on complete debridement of wounds; bad results are usually due to inadequate surgical debridement and this usually happens because the surgeon is unaware of the extent of the lesion at the time of surgery. For this reason, as complete a diagnostic work-up as possible is also needed in these patients.

Conclusions

Optimum management of head trauma consists largely of making plans to avoid as many of the secondary complications of head trauma as possible. This may be effected through a comprehensive and rapid diagnostic process, by early and adequate surgical decompression, by full debridement and dural repair in penetrating wounds, and by meticulous intensive care. The measure of the success of such a regime will be the small number of patients in whom neurological deterioration occurs after their admission to the hospital.

REFERENCES

9. Langfitt TW, Weinstein JD, Kassell NF, et al: Transmis-


16. BECKER DP, MILLER JD, WARD JD, ET AL: The outcome from severe head injury with intensive management. J Neurosurg, to be published.