The State of Consciousness is Immediately Assessed in A Patient with A Head Injury

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Abstract
When an injured person with a head injury arrives at the hospital, doctors and nurses first check for life signs: heart rate, blood pressure, and breathing. The patient’s state of consciousness and memory is immediately assessed. The basic functions of the brain are also examined by checking the size of the pupils and their reaction to light, assessing the reaction to sensations such as heat and needle sticks, and examining the ability to move the arms or legs. To determine a possible brain injury, Computed Tomography (CT) or Magnetic Resonance Imaging (MRI) is needed.

Keywords: Head; Injuries; CT; MRI; GCS

Introduction
The most important guide to the management of the patient with head trauma is the mental status and its serial reevaluation [1]. In patients with normal or relatively normal blood pressure and adequate oxygenation, significant abnormalities of mental status not responding to intravenous 50% dextrose, naloxone, and thiamine must be assumed to be secondary to injury to the brain and be treated as such. Mental status should be investigated in the usual manner, along with the routine neurologic examination, but the physician must recognize those subtle changes in personality reported by the family of the patient, such as combativeness or unusual aggressiveness. Also, subtle degrees of emotional lability may accompany important cerebral injuries and may be the first and often the only clue to their presence.

Clinical Problem
Head injuries are a complex clinical problem with a wide spectrum from the mild concussion to the severe brain injury resulting in death [2]. A thorough understanding of the head injuries should be based on the identification of the pathophysiological processes that developed as a result of the injury. This can be achieved by classifying the injuries according to the following specific criteria:

- Type of underlying tissue damage
- Mechanism of the injury
- Severity of the injury
- Extent of the tissue damage (focal or generalized)

The following classifications of head injuries in various types are very useful in clinical management and also in forensic practice. Depending on the type of damage they are classified as:

Scalp injury
- Skull fracture
- Extradural haematoma
- Subdural haematoma
- Intracerebral haematoma
- Brain contusion
- Cerebral oedema
- Cranial nerve damage

Identification
Head injury is common with an incidence of 0.3% of the population and a mortality rate of 25 per 100 000 in North America and 9 per 100 000 in the UK [3]. Severe head injury is associated with a high mortality rate (30% - 50%) and many survivors will have persistent severe neurological disability. Prompt identification and appropriate early management of such patients is necessary to ensure optimal long-term outcome. Managing such injuries in the field can pose many challenges. Patients may require extrication from vehicles, may be agitated and combative, or may require advanced airway management in a difficult environment. Coexisting injuries are common in patients with a severe head injury. Coordination between the various emergency services is essential to ensure that the patient is managed in a timely fashion.

All patients should have their Glasgow Coma Score (GCS) calculated as part of their disability assessment. The motor score is the most powerful predictor of outcome (except in cases of paralysis - therapeutic or traumatic). Abnormal posturing can be indicative of severe brain injury. Decorticate posturing, where the upper limbs flex and the lower limbs extend involuntarily, reflects injury at the level of midbrain or above (cerebral hemispheres, internal capsule, thalamus). Decerebrate posturing, where both the upper and lower limbs extend involuntarily reflects injury at the midbrain or below. The pupils...
should be examined for signs of equality and reactivity. Any difference between the left and right side should raise a suspicion for significant head injury. Signs of basal skull fracture should be actively sought.

**GCS**

Establish a GCS for any patient with a head injury [4]. The scale measures eye opening, speech, and motor response with total scores ranging from 3 (no response in all categories) to 15 (completely normal) provides a reliable way for physicians to assess the degree of neurologic dysfunction and communicate findings to other clinicians. Repeat the GCS periodically during reassessment. In addition, measure pupillary response and symmetry and also consider doll's eye (oculocephalic) movements (unless cervical spine injury has not been excluded) and caloric stimulation (oculovestibular) tests, if needed, to gauge the patient's level of cortical and brainstem functioning. Note any asymmetry in neurologic examination or focal neurologic findings. In an unresponsive patient, motor response may be elicited by nail bed pressure. If motor responses are asymmetric, the best response is a more accurate predictor of outcome and should be used for calculating the GCS. It is particularly important to document initial neurologic examination findings prior to administering sedative or paralytic agents, if possible.

As for any patient with altered mental status, the clinician is advised to check for and treat any easily reversible causes of decreased level of consciousness including hypoglycemia (bedside fingerstick blood glucose), hypoxemia (pulse oximetry), narcotic overdose (naloxone administration), and, in malnourished or alcoholic patients, Wernicke encephalopathy (thiamine administration).

**Hyponatremia**

Hyponatremia is not a usual finding in patients with isolated head injuries except when massive bleeding from the scalp occurs or as a terminal event; other injuries to the chest and abdomen must therefore be presumed to be present and excluded [1]. Spinal shock may result in hyponatremia, although it is generally not profound, and this must remain a diagnosis of exclusion; when compared with hemorrhagic shock, the extremities are warm and the pulse remains within the normal range. In these patients, other evidence of spinal cord injury is usually present, including flaccid paralysis, a flaccid rectal sphincter, a sensory level, apnea, or diaphragmatic breathing, and, occasionally, priapism.

**CT and MRI**

In disorders with acute onset, like head trauma or stroke, an initial CT scan, when available, is the most powerful method of showing the mechanism and the acuteness of the injury [5]. In the case of head trauma, the presence of acute hemorrhage (white on CT scans) allows proof that the event in question, not some other event, caused the injury. Later examinations do not contain this information because the fractures heal, swelling resolves, blood is broken down and injured brain tissue is resorbed. The later scans, particularly MRI, show the final outcome of the head injury, effectively demonstrated by sequential MRI scans that show the loss of brain tissue. This is also true for stroke, or for tracking any type of lesion over time. Methods also demonstrate generalized atrophic changes of the brain when present, important for assessing degenerative diseases where mental competency may be questioned.

Conventional imaging, such as standard CT and MRI scans, show the qualitative effects of focal and diffuse brain damage, but do not address such questions as, 'How much tissue has been lost?' 'How has this brain changed?' 'How may these changes be quantified, and how do they relate to medical, neurobehavioral and neurocognitive outcome in someone with a neurological condition?' The conventional imaging report represents the radiologist's qualitative impression. As an adjunct to this qualitative evaluation of the scans, there are new and reliable quantitative methods that can also be applied to radiographic interpretations. Quantitative analyses of neuroimaging findings provide additional objectivity to the qualitative radiological report.

**TBI**

Head injury (Traumatic Brain Injury [TBI]) is a leading cause of morbidity and mortality [6]. The incidence of hospitalization for TBI is 75-200/100,000 population. TBI occurs among all ages, peaking in 15 to 24-year-old males. Head injury is very frequent in poly trauma patients managed by trauma surgeons or emergency physicians. Thorough familiarity of the basics of care is therefore highly desirable.

Motor vehicle accidents are the most frequent cause of TBI in the developed world, accounting for 30% - 50% of all serious head injuries. Falls and recreational injuries account for about 10% - 15% of TBI. Inflicted injury (assault) accounts for about 10% - 20% of injuries in adult patients. Age and mechanism or injuries are related, as assaults occur mostly to very young infants (child abuse) and to young adults ages 18-24. Falls are the most common source of head injury in patients over 80 years of age. Response to injury also appears to be age dependent. Young people, particularly young men, are more likely to suffer a brain injury, but the chances of dying from that injury are much higher in the elderly. Head injury is the leading cause of death among all patients suffering traumatic injury.

Head injury can be divided on clinical grounds into mild, moderate, and severe forms. About 80% of injuries are mild, including most concussions. Many patients with these injuries do not require hospitalization. Moderate and severe injuries account for about 10% each of the total injury burden and all of these patients are hospitalized. The death rate from head injury is estimated at 20-30/100,000. Traumatic brain injuries are classified as primary or secondary, focal or diffuse [7].

The initial brain injury producing irreversible damage which occurs immediately following the trauma is known as the primary injury. In children with AHT who have abnormal radiological investigations, diffuse axonal/sheer injury and cortical contusions are the most common primary injuries followed by intracranial haemorrhage and vascular and penetrating injuries. Pathophysiological responses to the primary injury, such as cerebral congestion, fall in cerebral blood flow, shock and cerebral vasospasm may occur, resulting in secondary injury (e.g., oedema, swelling, herniation and hypoxic ischaemia).

Hypoxia further contributes to brain injury and arises from a variety of mechanisms, including impaired brainstem function. Squeezing of the chest during shaking may restrict respiratory effort and reduce ventilation. Carotid occlusion with reduced cerebral blood flow can occur with direct strangulation and forceful neck movements. Suffocation by blocking the nose and/or mouth to suppress crying,
occlusion of airways by regurgitated stomach contents and seizures can also contribute to hypoxia.

Persons with affective disorders may underestimate their behavioral and cognitive capabilities and over perceive their functional difficulties. Individuals with conversion or somatizing disorders may believe they are brain impaired when they are not, or that they are more incapable or impaired than they are, and they may misperceive normal behavior as indicative of disorder, skewing their self-reports [8]. False-positive diagnoses may lead examinees to mistakenly believe that they are brain-damaged and to greatly overestimate the frequency of neuropsychological difficulties, and false-negative diagnoses may lead to the opposing types of errors in self-perception and self-reporting. Some individuals reconstruct an overly positive image of pre-accident functioning and may misperceive their present normal shortcomings as pathological or as representing a change in status. Consequently, they may describe a long list of “symptoms” secondary to their injury.

People can easily form false attributions about the causes of their problems (if these judgments were always so easy, there would be little need to consult highly trained specialists to determine etiology). The patient with dementia who has started down the path of progressive decline may suddenly come to the attention of service providers after a mild head injury causes a temporary diminution in cognitive functioning, with subsequent problems blamed entirely on the car accident. The patient who shows persisting symptoms may attribute them to medication side effects rather than the head trauma, the patient who cannot concentrate at work may blame the problem on exposure to toxins rather than a sleep disorder, and so on. Clinicians usually ask patients to discuss possible precipitating factors and may give great weight to their self-reports, sometimes above all other information. (It would be fascinating to study the frequency with which patients draw correct conclusions about the causes of their conditions when there is no incentive to mislead but powerful incentives for accuracy.) Considering the many ways examinees can inadvertently mislead themselves and others, it would be outrageous to assume that any type of misrepresentation provides strong evidence of malingering; this is exactly why we cannot overlook the element of intentionality.

Mild head injury

Most patients after head injury complain for a time of headache and dizziness, and sometimes of poor concentration and memory, and of fatigue and irritability [9]. These symptoms comprise the postconcussional syndrome and because they commonly occur in patients who have had only a few minutes of posttraumatic amnesia or none at all, it was once believed that they were psychological in origin, and not related to organic dysfunction of the brain. That has now been disproved, because for some 2 weeks to 3 weeks after such a mild injury it can be shown that all patients have some impaired processing of information when formal psychological tests are carried out. There may also be disorders of the vestibular apparatus controlling balance and an increased sensitivity to noise (hyperacusis). If these various subjective complaints are ignored by doctors, instead of the patient being reassured that they are likely to be temporary and are not evidence of serious damage, the symptoms may become a source of anxiety to the patient and this may make matters worse. These symptoms may be exaggerated or prolonged if the patient returns to work too soon, particularly if this involves paper work and intellectual effort. Usually these complaints do not persist for more than a month but there are a few patients who, for reasons that are often not obvious, continue to complain for months and a few who find it difficult to adjust to normal life again. This has been termed accident neurosis and if it is suspected then a skilled psychiatric opinion should be obtained. In view of the clear organic background to these symptoms in the early stages, it is unwise to assume that the patient is malingering or exaggerating. The post-traumatic stress syndrome, related to recalling the frightening circumstances of the accident, is not a feature of patients with a significant head injury, as they are amnesic for these events.

Children

As any parent knows, pediatric head injury is common, but fortunately the vast majority of events do not result in any significant brain injury [10]. Pediatric head injury can be a consequence of impact head injury, rotational head injury or penetrating head injury. Typical mechanisms include falls and motor vehicle accidents, with assaults being much less common in children when compared with adults. Much of pediatric head injury mirrors that seen in adult practice, although specific entities not seen in adult neurotrauma are perinatal head injury and abusive head trauma.

Pathologies associated with pediatric head injury include focal lesions, such as skull fracture, extradural (epidural), and subdural hemorrhage, cortical contusions, and focal pathologies associated with secondary processes (e.g. infarctions from herniation); and diffuse lesions, such as brain swelling, diffuse traumatic axonal injury and global hypoxic-ischemic brain injury.

The concept of abusive brain injuries in children has been known for many years, and has had several names, such as battered baby syndrome and, more commonly, shaken baby syndrome. This latter name reflected the proposed mechanism resulting in the typical triad described in these cases of acute subdural hemorrhage, usually a thin film and bilateral, retinal hemorrhages, and ischemic encephalopathy. However, a biomechanical study suggested that shaking alone was unlikely to generate the forces required to produce the typical pathology, and the term “shaking-impact syndrome” was suggested. Biomechanical data have subsequently been conflicting as to whether shaking alone can cause a fatal injury. Nonaccidental trauma or nonaccidental head injury are two commonly used terms, although, currently, the term “abuse head trauma” is recommended by the American Academy of Pediatrics: all three terms avoid attributing injuries to any specific mechanism, recognizing that different mechanisms can cause the variety of injuries observed in this context, although even these terms do imply intent.

Emergencies

Facial injuries are among the most common emergencies seen in an acute care setting [11]. They range from simple soft tissue lacerations to complex facial fractures with associated significant craniofacial injuries and soft tissue loss. The management of these injuries generally follows standard surgical management priorities but is rendered more complex by the nature of the numerous areas of overlap in management areas, such as airway, neurologic, ophthalmologic, and dental. Also, the significant psychological nature of injuries affecting the face and the resultant aftermath of scarring can have devastating and long-lasting consequences. Despite the fact that these injuries are exceedingly common, they are cared for by a large group of different specialists and as such have a remarkably heterogeneous presentation and diverse treatment
schema. Nonetheless, guiding principles in the care of these injuries will provide the basis for the best possible outcomes. The following questions will guide general management and provide a framework for understanding the principles in the acute care of patients with facial injuries and trauma.

Despite the extremely common presentation of such injuries, there remains little standardization on repairing and then caring for the wounds or lacerations. There is great variation in the repair of lacerations as well as the different materials used to repair them. This is again because of the numerous different specialties involved in the care of the injuries and their desires to provide the best possible outcome with regard to scarring. Pediatricians, emergency department personnel, and surgeons may not all agree on the best modalities for repair. Placements as well as type of dressing are also controversial.

The timing of facial skin laceration closure is the same as that of any open wound. The presence of contaminating factors in the management of wound would generally not allow closure after six hours and would favor delayed closure. However, clinical practice is slightly more variable with facial lacerations because of the uniquely sensitive nature of facial scarring. Although we generally ascribe to experimental data regarding timing of closure, in practice the six-hour rule is often overlooked with an attempt to be vigorous in cleaning the wound. The presence of exceptionally rich blood supply in the face is also deemed of benefit in extending the six-hour rule.

Forensics

Many forensic autopsy reports have specific sections dealing with the external examination and the internal examination [12]. Included in the external examination are various physical characteristics of the body (height, weight, eye color, gender, skin pigmentation, identifying marks and scars, medical therapy, etc.), as well as clothing and jewelry, and any evidence of postmortem change. Many forensic pathologists choose not to include evidence of injury within this section, and instead opt for a separate section that specifically deals with injuries.

The internal examination section provides a description of the organ systems, the body cavities, and the other tissues of the body. Usually, each system is described separately, and weights are provided for most of the organs. As with the external examination, many forensic pathologists choose not to include evidence of injury in this section, preferring to have a separate injury section.

Many forensic pathologists include a separate “evidence of injury” section within their autopsy report. The rationale is that it seems rather disjoined to include injury descriptions interspersed amongst the descriptions of normal or deceased organs or tissues. A separate evidence of injury section allows descriptions of external injuries and internal injuries in a relatively concise fashion. For some injury types, such as blunt force head injuries, it is appropriate to start superficially (at the skin surface) and describe different depths of injury (skin, scalp, skull, dura, arachnoid, brain). For other injury types (gunshot wounds), it is more appropriate to describe the pathways of the injuries. When there is more than one specific injury of a particular type (such as gunshot wounds), it is appropriate for the pathologist to number the wounds for descriptive purposes. It is important to note that such numbering does not necessarily correspond to the sequence of injury occurrence. Many pathologists attempt to describe different injury types within different sections within the evidence of injury section. For example, a specific case may have gunshot wounds, sharp force injuries, and blunt force injuries. It is appropriate to have subsections for each injury type within the evidence of injury portion of the autopsy report. Sometimes within a certain injury type, further separation is provided based on body region. For example, with blunt force injuries, there may be separate sections for head and neck injuries, trunk injuries, and extremity injuries.

Conclusion

Severe head injury can break, cut, or rupture nerves, blood vessels, and tissues in or around the brain. Nerve pathways may be interrupted and bleeding or severe swelling may occur. Bleeding, swelling, and fluid formation have a similar effect to massive growth inside the skull. Because the skull cannot expand, increased pressure can damage or destroy brain tissue. Due to the position of the brain in the skull, the pressure tends to push the brain down. The upper part of the brain can be pushed into the opening that connects it to the lower part, or into a condition called herniation. A similar type of hernia can pinch the cerebellum and brainstem through a skull-based opening into the spinal cord. Herniations can be life-threatening because the brainstem manages such vital functions as heart rate and respiration.

References


