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PRACTICAL MANAGEMENT OF HEAD INJURY

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ABSTRACT

For an effective practical management of head injury, a clear knowledge of the various causes and mechanism of head injury is essential. The concept of the brain in a rigid cranial cavity makes the pathophysiological mechanism of head trauma unique. Most problems occur due to poor handling of patients at the site of trauma or lack of adequate resuscitation before radiological investigation at the emergency department. With adequate management of intracranial pressure and nursing of unconscious patient, most patients can be managed without neurosurgical consultation. Timely referral and surgical intervention can prevent the catastrophic effects of rapidly expanding intracranial haematoma. This paper discusses practical management head injury management and highlighted some pit falls in management.

Key words: Head injury, management, pitfalls

INTRODUCTION

Head injury is a major cause of death in children and young adults, in both developed and developing countries[1]. Majority of the patients are males, [3] and it accounts for 50% of trauma deaths. Most patients with head injury are managed by general practitioners and generals surgeons and, only few are referred to the neurosurgeon. An understanding of the mechanism and pathophysiology of head trauma is necessary for optimal management of these patients.

CAUSES OF HEAD INJURY

Road traffic accidents account for 27% of minor head injuries and for 70% of severe head injuries.[3] Pedal cycles account for many injuries in childhood, [2,7] Assaults, Firearms, Interpersonal Violence, fall, Fits and Recreational Accidents, are another important causes of head injury. Many causes of head injury are associated with injuries to other parts of the body and this contributes, significantly to the overall outcome.

MECHANISM OF HEAD INJURY

Most head injuries are due to blunt acceleration-declaration injuries, [2] that is the rapid declaration when a moving head strikes a static surface or acceleration when a static head is struck by a moving object. This result in local (Coup) or an opposite

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(Counter Coup) injury to the brain.[7] This usually causes diffuse brain damage. In contrast, penetrating injuries cause local damage especially when due to low velocity agents such as a sharp objects and low velocity bullets. High velocity ballistic missiles at close range cause extensive brain damage.[3] Crushing injuries usually cause extensive scalp and skull injuries with minimal damage to the brain.

PATHOLOGY

Primary injuries are those injuries that occur at the time of impact. They range from minor concussion injury to severe brain damage. Secondary injuries may follow any time after the impact and are potentially preventable. (Table 1).

Minor injury causes physiological disturbance, while moderate to severe moderate to severe injury causes neural, axonal and small vessels damage, which leads to prolonged period of unconsciousness.[5]

Table 1: Primary head injuries

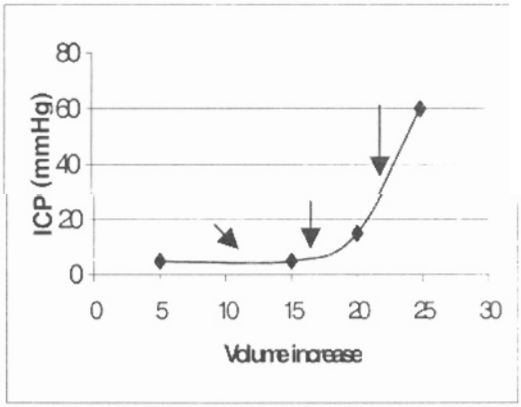
Site	Types of injuries
Scalp	Contusion, Lacerations, Avulsion, Haematomas (Subcutaneous, Subgaleal, Subperiosteal).
Skull	Simple (closed) – Linear, Depressed, or Comminuted Fractures Compound (open) fractures (skull base fractures are also compound from within as it communicate with one of the air sinuses or auditory canal).
Brain	Contusion, Diffuse Axonal damage, Laceration
Meningeal	Laceration, Vessel rapture

Secondary injuries include cerebral hypoxia, metabolic disorders, infection, epilepsy and pneumocephalus.[1, 2,3] These cause increasing neurological damage and can be corrected by timely intervention.

INTRACRANIAL PRESSURE AND CEREBRAL PERFUSION

Intracranial pressure (ICP) is governed by 3 factors within the fixed confines of the skull, i.e. brain parenchyma, cerebrospinal fluid and cerebral blood volume. [1] Following trauma and subsequent brain swelling, there is an increase in ICP and except there is a compensatory decrease in volume in the other compartments, when these compensatory mechanisms fail small volume increase have a relatively large effect on ICP (Figure 1).

Figure 1: Intracranial pressure and volume curve (= *Compensatory phase,* = *Decompensation but reversible,* = *Decompensation and irreversible)*



THE CONCEPT OF CEREBRAL PERFUSION PRESSURE (CPP)

CPP is the pressure required to perfuse the brain, and is dependent on the difference of mean arterial blood pressure (MBP) and the intracranial pressure (CPP = MBP- ICP). Increase in ICP as happens in head injury results in reduced CPP, which results in cerebral ischaemia. In addition, there is lack of naturally existing autoregulatory mechanism in head injury. This also results in brain oedema.

PRE-HOSPITAL CARE

At the site of injury the patient should be taken away quickly from the source of trauma to prevent further injury. Airway is cleared using cloth, handkerchief or fingers. Patients is placed in the left lateral position. The patient is assumed to have spiral injury and should be moved in one piece. Arrangements should be made for quick transport to the hospital. In areas where ambulance services are available, intravenous fluid, rigid cervical support and even intubations and ventilation can be commenced by the trained ambulance crew.

EMERGENCY DEPARTMENT CARE

PRIMARY SURVEY

As the patient is brought to the emergency unit, a quick history is taken from the relations, police, ambulance crew or people that brought the patient, as to what happened and if the patient talked before becoming unconscious. As these questions are being asked a check is made of the airways and the cervical spine is placed in line with the rest of the spine. Next the breathing pattern and adequacy is assessed. The circulation and areas of haemorrhage are noted and the disability is noted on AVPU scale (Alert, Responding to Voice only, Responding to Pains only and Unresponsive). The pupil's are quickly checked for dilatation and response to light. The patient is then completely exposed to allow adequate examination but protection is made against hypothermia. All these should be accomplished within the first three minutes of admission.

RESUSCITATION

Primary survey and resuscitation should go on simultaneously.

1. The airway is cleared of secretions, vomitus or other materials. The unconscious patient with a lot of secretions or craniofacial injuries may require tracheal intubation.
2. Cervical spine is immobilized until cervical X-ray excludes spinal injury.
3. The breathing pattern and adequacy is assessed. Breathing can be maintained using an ambu-bag with oropharyngeal airway or endotracheal tube.
4. Circulatory support and control of haemorrhage: Intravenous fluid (Normal saline, Ringer's lactate, Dextrose saline but not 5% Dextrose) is set up in a large vein and blood taken for biochemical, haematological test and grouping and cross matching.
5. Nasogastric tube and urethral catheter are inserted except if contraindicated.
6. Assessment is made of the response to the resuscitative measures using the pulse, blood pressure, capillary refill and urine output.

SECONDARY SURVEY

Neurological assessment is made using the Glasgow Coma Scale (GCS) (Table 2) and external signs of injury to the head. The best response is recorded.

It is based on eye opening, verbal response and motor response. The score ranges between 3 and 15. It is simple, and has only minimal inter observe error. The stimulus consists of firm pressure over the supra-orbital margin for localization of pain, sternal pressure for flexion to pain and nail-bed pressure for flexion-withdrawal, abnormal flexion and extension. Side to side differences should be recorded separately.

Table 2: The Glasgow coma scale

Score	Eye opening	Verbal response	Motor response
6	///////	//////////	Obeys Commands
5	///////	Oriented	Localizes Pain
4	Spontaneous	Confused	Flexion Withdrawal
3	To Speech	Inappropriate Word	Abnormal Flexion
2	To Pain	Incomprehensive Sounds	Extension
1	None	None	None

Glasgow Coma Score (GCS) = E+V+M+ = 3 – 15
Severe head injury GCS = <9
Moderate head injury GCS = 9 – 13
Mild head injury GCS = 14 – 15

Glasgow paediatric score

The parameters exist for children (Tables 3 and 4), as in adults but the aggregate score is 12 for less than one year and 14 for those 1 – 5 years. Assessment is difficult due to difficulty in communication and expression.[5, 10]

Table 3: Less than 1 year (total = 12)

Score	Eye opening	Verbal response	Motor response
5	///////	Smiles/ Cries appropriately	//////////
4	Spontaneous	Cries	Localizes
3	To shout	Inappropriate crying	Flexion pain
2	To pain	Grunting	Extension
1	None	None	None

Table 4: 1 – 5 years (total = 14)

Score	Eye opening	Verbal response	Motor response
5	///////	Oriented / Appropriate	Obeys command
4	Spontaneous	Disoriented	Localized pain
3	To command	Cries/Inappropriate words	Flexion to pain
2	To pain	Grunting	Extension to pain
1	None	None	None

The GCS should be recorded before and after resuscitation. Other evaluations include;

1. Papillary size and response to light is also assessed. Unilateral dilatation indicates an expanding intracranial haematoma. Local orbital injury should be excluded.
2. Other neurologic signs include:
   - Haematoma around the mastoid tubercle "Battle Sign" indicate middle cranial fossa injury.
   - CSF leak through the nose or ear indicate fracture base of skull with dural tier.
   - Scalp lacerations, skull fractures and spinal tenderness are assessed.
   - Motor deficits such as hemiparesis and quadriparesis are also noted.
3. Detailed history is now taken and a record made of the vital signs i.e. pulse, blood pressure, respiratory rate and temperature.
4. Systemic examination from head to toe is made to exclude other associated injuries.

INVESTIGATIONS

Patients should be adequately resuscitated before radiological investigations.

1. **Skull X-ray**: This provides useful information about skull fracture and possible underlying brain injury. Essential views are antero-posterior and lateral.[6] X-ray of the cervical spine should also be included, which must show C7/T1 before it is regarded as satisfactory.

Indication for skull x-ray

- Loss of consciousness
- Focal neurologic signs
- Penetrating injury
- Palpable or visible skull deformity
- CSF rhinorrhoea or otorrhoea
- Scalp bruising, haematoma or laceration
- Persisting headache and vomiting
- Difficulty in clinical assessment in alcoholics, drug intoxications, epilepsy, children or elderly.

The presence of a skull fracture with loss of consciousness is associated with 1:4 chances of intracranial haematoma.[2, 3] It also indicates the possible site of an extradural haematoma in a rapidly deteriorating patient. The presence of air (pneumocephalus) can also be detected.

2. **CT brain scan**: The introduction of computed tomography (CT) in 1973 opened new opportunities in the investigation of head injury and it is the investigation of choice.[6] It demonstrates the presence of space occupying lesions, such as haematoma, infarction or brain oedema as the cause of the mass effect (Figures 1 – 3). The indications for CT brain scan include:

I. GCS <9 following adequate resuscitation.
II. Focal neurologic signs especially with presence of a skull fracture.
III. Neurological deterioration e.g. a drop of 2 points or more in the GCS.
IV. Persistent headache and vomiting (signs of raised ICP)
V. CT scan should be repeated if the patient deteriorates even after a previously normal scan. This is most important especially if the scan was done in the firsts 4 – 6 hours of injury.[5]

3. **Angiography:** CT has largely replaced angiography except in patients with pre-existing intracranial aneurysm, traumatic carotico-carvenous sinus fistula or

injury to neck vessels leading to cerebral infarction.[6]

4. **Magnetic resonance imaging (MRI):** The role in head injury is limited by availability and problems of using of life support system in magnetic field.[6] The images although with better resolution are no more diagnostic than CT scan.

Figure 1: CT appearance of a left extradural haematoma

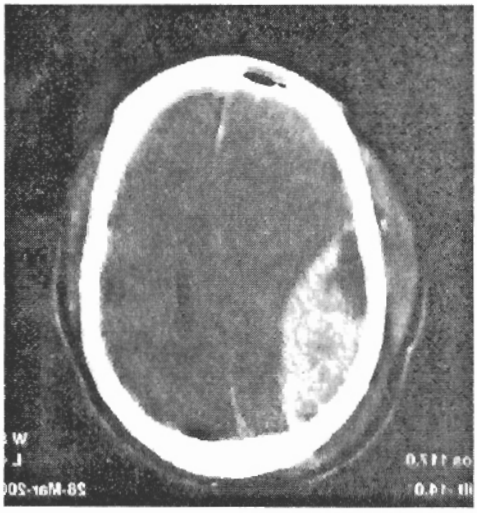


Figure 2: CT appearance of a right subdural haematoma

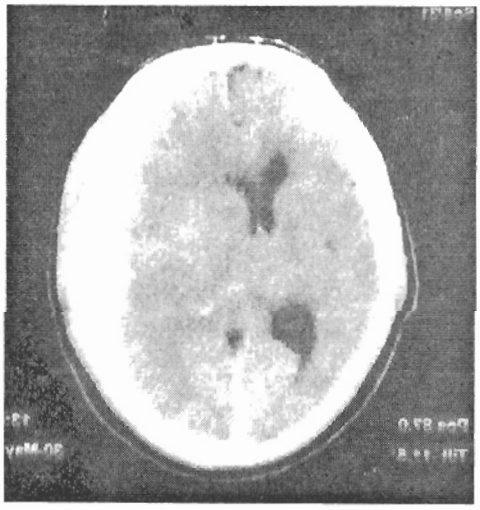
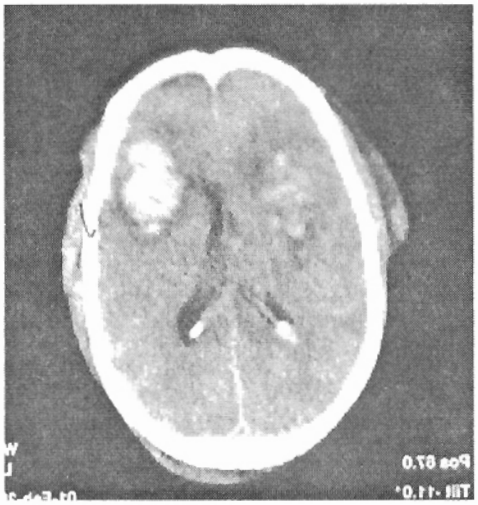


Figure 3: CT appearance of a right intracerebral haematoma with contre coup injury



DEFINITIVE CARE

Following resuscitation and investigation, a decision should be made about further management, or transfer, and also management of other associated injuries. The indications for admission include:

1. Altered level of consciousness.
2. Focal neurologic deficit.
3. Persistent headache and vomiting.
4. Post traumatic epilepsy.
5. Skull fracture or abnormal CT brain Scan.
6. CSF rhinorrhea or otorrhea.
7. Difficulty in clinical assessment especially in alcoholics, children and elderly.
8. Associated medical conditions such as bleeding disorders, diabetes mellitus etc.
9. Inadequate supervision at home.

Most patients can be managed without neurosurgical consultation. The following are indications for neurosurgical consultation or transfer. Deteriorating level of consciousness of two or more points following adequate

resuscitation.
2. Development of focal neurologic signs.
3. Penetrating injury.
4. Persistent CSF rhinorrhoea.
5. Compound or depressed skull fracture.

The patient should be stabilized before transfer. A trained medical personnel with knowledge and experience in cardiopulmonary resuscitation should accompany the patient.

TREATMENT MODALITIES

In an unconscious patient hypoxia and hypotension interfere with cerebral oxygenation. Attention should be paid to the airways. The patient is intubted and ventilated if the airway is compromised. Oxygen by facemask or nasal catheter also improves the oxygenation. The patient is nursed head up (30°) to improve cerebral venous return from the valveless intracerebral sinuses and jugular veins.[7]

Urine output is monitored and attention is paid to pressure points, oral hygiene and the genital region.

INTRAVENOUS FLUID

Resuscitation should be done with isotonic solutions such as Ringer's lactate solution or Normal saline. There after normovolaemia is maintained with 2 to 2.5litres of dextrose saline. 5% dextrose should be avoided because it may cerebral oedema. Dehydration and over hydration should be avoided. Physiologic response to raised ICP includes raised blood pressure and a slowing of pulse rate (Cushing's reflex). This may mask the effect of shock. However, head injury alone is not usually associated with hypovolaemic shock except in children or patients with extensive scalp lacerations. Serum electrolyte and urea, and blood sugar levels should be monitored.

TREATMENT OF RAISED ICP

Active treatment of intracranial pressure should only be undertaken when there is evidence of neurological deterioration due to intracranial causes, e.g. pupillary dilatation or deteriorating motor function.[3, 4,8]

1. **Intravenous Mannitol:** Mannitol should be avoided unless adequate resuscitation has been carried out. A bolus of 0.5 to 1mg/kg body weight over 20 minutes is given and arrangements are made for operative interventions or transfer to a neurosurgical center.[9] Fluid loss through diuresis should be replaced. Furosemide can be alternatively given at a dose of 40 mg or 1mg/kg body weight in a child. These measures improve or at least keeps the patients condition for at least 1 to 2 hours before definitive treatment or investigations. Mannitol a has rebound effect on raise ICP, [9] so if there is a prolonged delay further smaller bolus of mannitol can be given at four hours interval.
2. **Ventilation:** It is important to avoid hypoxia and raised $PC0_2$ as they cause a rise in the intracranial pressure.[5] Patients with GCS < 8 and poor respiratory function should be intubated and ventilated. Controlled hyperventilation causes cerebral vasocontriction, thereby reducing cerebral blood volume and ICP. Further decrease of $PaC0_2$ to less than 30mmHg can result in cerebral ischaemia and should be avoided.[2]
3. **Corticosteroids:** These are not recommended. They have not been found useful in head injury.[4, 7] They could further cause fluid retention, stress ulcers and decrease patient's immunity.[5]

ANTIBIOTICS
Patients with contaminated wounds or compound skull fracture should be given appropriate antibiotics.

TETANUS PROPHYLAXIS
Tetanus prophylaxis in the form of tetanus toxoid, 0.5mg should be given to patients with open wounds. Tetanus immune globulin (or antitetanus serum if the immune globulin is not available) should also be given if the wound is grossly contaminated.

ANTICONSVULSANTS
Anticonvulsants should not be given routinely, or because the patient is restless. The following are indications for anticonvulsant therapy. [7]

1. Early posttraumatic epilepsy (within one week).
2. Late posttraumatic epilepsy (after the first week).
3. Depressed skull fractures especially those with focal signs, dural laceration or posttraumatic ammesia more than 24hours.
4. Penetrating injury
5. Compound skull fracture with brain herniation.
6. Following surgery for intracranial haematoma.
7. Patient with previous history of epilepsy.

Acute seizure should be controlled with intravenous phenitoin 150 to 200mg slowly due to its cardiotoxic effect. Phenobarbitone at a daily dose of 1 to 2.5mg/kg could also be use.
Maintenance close of phenytoin is 4 – 8mg/kg.

ANALGESICS
Non-opiate analgesics should be given to restless patient with GCS of 9 and above, as pain causes an increase in intracranial pressure.

SEDATION
The head injury patient should not be sedated as this could cause respiratory depression and hypoxia. A restless patient should be restrained and the cause of restlessness such hypoxia, full bladder, pain, dehydration, hypoglycaemia or constipation identified.

INTENSIVE CARE MANAGEMENT
A GCS score of 8 or lower after resuscitation and following removal of haematoma are indications for admission to an ICU if available. The focus of ICU management is prevention of secondary injury and maintenance of cerebral oxygenation.

- Controlled hyperventilation, via endotracheal tube or tracheostomy is maintained.
- $PaC0_2$ is maintained at 30mmHg and $Pa0_2$ above 80mmHg.[3]
- Intra-arterial blood pressure monitoring and blood gases could also be carried out.
- ICP monitoring devices can be attached.[8]
- Total parenteral nutrition to prevent negative nitrogen balance possible.
- Tracheobronchial toileting can be done.

SURGERY
Emergency surgical treatment for a rapidly deteriorating patient with extradural haematoma can be done if transfer to a neurosurgical unit is not possible or require prolonged time. The site of the burr hole is indicated by CT scan or if not available, by the site of skull fracture on X-ray. Scalp swelling or lacerations and the side of pupilary dilatation also indicate the site of the haematoma. If no external sign exists, the site for bifurcation of middle meningeal vessel i.e. the temporal region 2cm above the zygomatic arch and 2cm in front of external auditory meatus is the most probable site.[4, 7]

Next a frontal and parietal burr hole are made if the haematoma is not found. The opposite side could also be treated similarly. Usually the extradural haematoma is solid and craniectomy around the burr hole is recommended for effective evacuation. However, the neurosurgeon prefers raising a flap so that bleeding vessels can be effectively coagulated.

PHYSIOTHERAPY

The Physiotherapist should be involved early to give chest physiotherapy and later the limbs to prevent wasting and improve early return of function.

COMMON PITFALLS AND MISTAKES

1. The GCS should be assessed at the time of admission and after adequate resuscitation.
2. Primary resuscitation for hypoxia and hypotension should be initiated without delay.
3. Primary survey and resuscitation should be carried out simultaneously.
4. Delay in initiating definitive neurosurgical care especially for the rapidly deteriorating intracranial haematoma.
5. Failure of early prevention of craniocerebral infection and institution of tetanus prophylaxis.
6. Abnormal neurosurgical signs such as abnormal pupilary size and reaction can be due to local eye injury.
7. Motor dysfunction can be due to cervical injury or abnormal response to trauma especially in the very young or elderly.
8. Large volume of crystalloids can result in cerebral oedema. Care should also be taken in the presence of renal or cardiopulmonary disease.
9. Head injury alone without extensive scalp injury does not cause hypovalaemic shock. So spinal injury, haemothorax, or haemoperitonum should be looked for.
10. Patients should be fully resuscitated before sending them for X-ray or CT scan. Rapid deterioration may require an immediate operation rather than risk delay in performing CT scan.
11. In intubated patient who are ventilated care should be taken to avoid pneumocephalus and pneumothorax.
12. Osmotic diuretics should only be used if the patient is well hydrated to prevent renal shut down.
13. Sedatives should be avoided in head injured patient to avoid respiratory depression.
14. Assessment of small children with head injury is usually difficult. Over hydration must be avoided.
15. In the presence of other injuries analgesia are recommended under close supervision.

CONCLUSION

Most head injured patient can be managed if they are well assessed and resuscitated and a simple guide is followed to prevent development of secondary injuries to the brain.

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