Acta Chir Croat 2012; 9: 49-53

HEAD INJURIES

Ozljede glave

Running head: Head injuries

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Introduction

The prognosis of brain injuries is good in patients who respond to simple commands, are not deeply unconscious, and do not deteriorate. The prognosis is grave in patients who are rendered immediately comatose (particularly those sustaining penetrating injury) and remain unconscious for a long period of time. Any subsequent neurological improvement may indicate salvageability and should prompt reevaluation.

Neurosurgical damage control includes

→ early intracranial pressure (ICP) control;
→ cerebral blood flow (CBF) preservation;
→ prevention of secondary cerebral injury from hypoxia, hypotension, and hyperthermia.

A motor examination of the most salvageable severely brain-injured patients will demonstrate localization to central stimulation and these patients will require expedited treatment.

Immediate intubation with adequate ventilation is the most critical first line of treatment for a severely head-injured patient.

Evacuation to the nearest neurosurgical unit, avoiding diagnostic delays, and initiating cerebral resuscitation allow for the best chance for ultimate functional recovery.

Head injury types

1) Blunt (closed head injury).

2) Penetrating.
Penetrating with retained fragments
Perforating
Guttering (grooving the skull)
Tangential
Cranial facial degloving (lateral temple, bifrontal)

3) Blast over-pressure CNS injuries.
A force transmitted by the great vessels of the chest to the brain; associated with unconsciousness, confusion, headache, tinnitus, dizziness, tremors, increased startle response, and occasionally (in the most severe forms) increased ICP. Bleeding may occur from multiple orifices including ears, nose, and mouth.

4) Explosion results in flying fragments, with possible vehicular-collision-associated blunt injuries. Depending on the proximity to the explosion, a blast over-pressure phenomenon may also result. In a severely brain-injured patient, more deficits than indicated by the CT scan may be due to possible underlying injury to brachiocephalic vessels, shear injury or the effects of blast over-pressure with resulting cerebral vasospasm. Plain films, more useful in penetrating than blunt trauma, may reveal a burst fracture of the skull indicating the tremendous perforating force of a penetrating missile. Transventricular bihemispheric fragment tracts portend a poor prognosis.

Severe head injuries are often seen in combination with significant chest, abdomen, and extremity injuries. Very rapid hemorrhage control is the priority in the non-cranial injuries; utilizing damage control concepts and focusing attention on the head injury. All efforts should be directed toward early diagnosis and intervention of the head injury.

Traditional classification of head injuries

1) Closed injuries, seen more often in civilian settings

2) Open injuries are the most commonly encountered brain injuries in combat.
→ Scalp injuries may be closed (e.g. contusion) or open (e.g. puncture, laceration or avulsion).
→ Any scalp injury may be associated with a skull fracture and/or underlying brain injury.
→ Open scalp injuries bleed profusely, even to the point of lethal blood loss, but usually heal well when properly repaired.

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→ Skull fractures may be open or closed, and are described as linear, comminuted or depressed.
→ Skull fractures are usually associated with some degree of brain injury, varying from mild concussion, to devastating diffuse brain injury, to intracranial hematomas.
→ Open skull fractures are prone to infection if not properly treated.

Mechanisms of injury

Primary injury is a function of the energy transmitted to the brain by the offending agent.

→ Very little can be done by healthcare providers to influence the primary injury.
→ Enforcement of personal protective measures (e.g. helmet, seatbelts) by the command is essential prevention.

Secondary injury results from disturbance of brain and systemic physiology by the traumatic event.

Hypotension and hypoxia are the two most acute and easily treatable mechanisms of secondary injury.

Other etiologies include seizures (seen in 30-40% of patients with penetrating brain injuries), fever, electrolyte disturbances (specifically, hyponatremia or hyperglycemia), and infection. All of the above conditions can be treated.

Elevations of ICP may occur early as a result of a space-occupying hematoma or develop gradually as a result of brain edema or hydrocephalus.

Normal ICP is 5–15 mm Hg, with normal cerebral perfusion pressure (CPP = MAP-ICP) usually around 70– 80 mm Hg.

Decreases in perfusion pressure as a result of systemic hypotension or elevated ICP gradually result in alteration of brain function (manifested by impairment of consciousness), and may progress to global brain ischemia and death if not treated.

Patient assessment and triage

During the primary and secondary assessment, attention should be placed on a complete examination of the scalp and neck. Fragments that enter the cranial vault with a transtemple, transorbital or cross midline trajectory should be suspected as having **associated neurovascular injuries**. Wounds are typically contaminated by hair, dirt, and debris and should be copiously irrigated clean with control of scalp hemorrhage, but not at the expense of delaying definitive neurosurgical treatment! Scalp hemorrhage can be controlled with a head wrap, scalp clips or surgical staples; a meticulous plastic surgical closure is only appropriate after intracranial injuries have been ruled out.

Clinical staging of craniocerebral trauma

		Midbrain syndrome				Bulbar-brain syndrome	
		I	II	III	IV	I	II
Conscious		Sleepiness	Coma	Coma	Coma	Coma	Coma
Reaction to	Accoustic irritation	Turns to target	None Untargeted	None Extended limbs	None Extension automatism	None Stretch automatism	None None
Oculomotor	Eye	Normal	Swimming movements	Divergent	Divergent	Fixed Divergent	Fixed Divergent
	Pupils	Mid-open	Mid-open	Below mid-open	Below mid-open	Wide	Wide
	Reaction to light	Prompt	↓	↓	↓↓	None	None
Brainstem reflexes	Oculocephalic reflex	Slight	Puppet-head +	Puppet-head +	Slight	None	None
	Ciliospinal reflex	+	+	-	(+)	None	None
	Vestibulo-ocular reflex	+	+	Tonic reaction to cold irritation	Only ipsilateral	None	None
Body movements	Posture	Normal	Legs nut-stretched	Bend-stretch posture	Extended posture	Extended legs	Sleeping
	Tone	Normal	Legs raised	↑	↑↑	↑	Sleeping
	Spontaneous movements	Bulk movements	Bulk movements of arm	Bend-stretch synergism	Strech synergism	Strech synergism	-
	Babinski	-	(+)	+	+	+	-
Vegetative system	Respiration	Irregular	Possibly Cheyne-Stokes	↑	Mechanical respiration		None
	Pulse	(↑)	↑	↑	↑↑	↑↑	↓
	Blood pressure	Normal	(↑)	↑	↑↑	↑↑	↑
	Temperature	Normal	(↑)	↑	↑↑	↑↑	Normal ↓

The most important **assessment** is the vital signs. Next is the level of consciousness, best measured and recorded by the **Glasgow Coma Scale (GCS)** (see below).

Triage decisions in the patient with craniocerebral trauma should be made based on admission GCS score.

→ GCS ≤ 5 indicates a dismal prognosis despite aggressive comprehensive treatment and the casualty should be considered expectant.

→ A GCS ≥ 8 indicates that a casualty may do well if managed appropriately. In general, neurologically stable patients with penetrating head injury can be managed effectively in the ICU with airway and ventilatory support, antibiotics, and anticonvulsants while awaiting surgery.

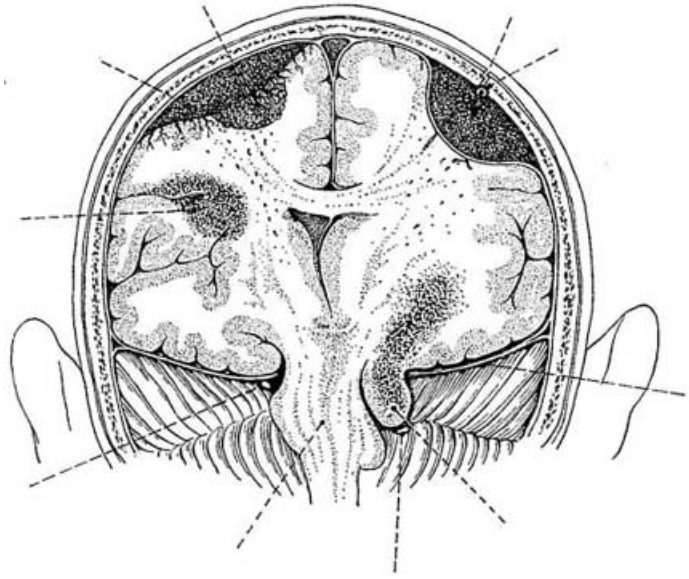
→ An exception to this would be a deteriorating patient with a large hematoma seen on CT - this should be considered a surgical emergency.

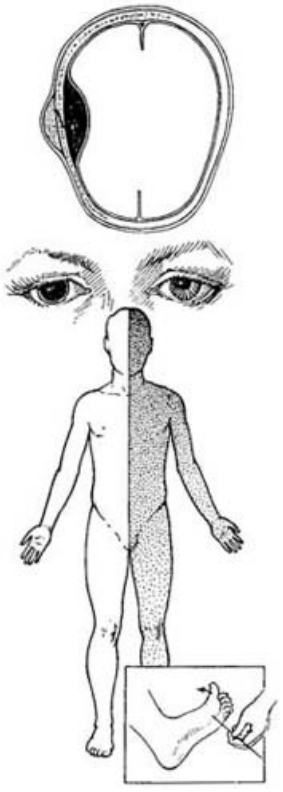
→ Casualties with GCS 6–8 can be the most reversible, with forward neurosurgical management involving control of ICP and preservation of CBF.

Another important assessment is **pupillary** reactivity.

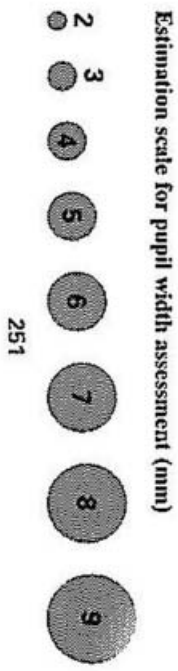
A single dilated or nonreactive pupil adds urgency and implies the presence of a unilateral space occupying lesion with secondary brain shift. Immediate surgery is indicated. The presence of bilateral dilated or nonreactive pupils is a dismal prognostic sign in the setting of profound alteration of consciousness.

Glasgow coma and outcome scales **Glasgow coma scale**		**Points**
Eye opening	Spontaneous	4
	On questioning	3
	On painful irritation	2
	None	1
Verbal reaction	Orientated	5
	Confused	4
	Single words	3
	Sounds	2
	None	1
Motor reaction	After requests	6
	Targeted pain reaction	5
	Flexor mechanism	4
	Atypical flexor reaction	3
	Extensor mechanism	2
	None	1
Maximum points		15
Minimum points		3





(After Lucking, C.H. (1976))



Phases of brain-stem damage	Midbrain syndrome				Bulbar syndrome	
	1	2	3	4	1	2
Vigilance	Sleepiness	Stupor	Coma	Coma	Coma	Coma
Reactivity	Retarded	Reduced	Absent	Absent	Absent	Absent
Spontaneous motor activity						
Motor pain reaction						
Muscle tone	Normal	Arm support	Raised	Raised ++	Normal-lax	Lax
Pupil width						
Pupil reaction to light						
Eye movement	Oscillating	Dysjointed	Absent	Absent	Absent	Absent
Oculocerebral reflex	Ø	+	++	+Ø	Ø	Ø
Vestibular reflex	Normal +	++	Tonic	Dissociated	Ø	Ø
Breathing						
Temperature						
Pulse rate						
Blood pressure	Normal	Normal	Slightly +	Definitely raised	Reduced	Reduced + ·

Management

Medical

Primary tenets are basic but vital; clear the airway, ensure adequate ventilation, and assess and treat for shock (excessive fluid administration should be avoided).

In general, patients with a GCS ≤ 12 should be managed in the ICU. ICU management should be directed at the avoidance and treatment of secondary brain injury.

→ PaO2 should be kept at a minimum of 100 mm Hg.

→ PCO2 maintained between 35 and 40 mm Hg.

→ **The head** should be elevated approximately 30A.

→ **Sedate patient** and/or pharmacologically paralyze to avoid "bucking" the ventilator and causing ICP spikes.

→ **Broad-spectrum** antibiotics should be administered to patients with penetrating injuries (a third-generation cephalosporin, vancomycin or Ancef, Unasyn or meropenen if acinetobacter suspected).

→ **Anaerobic** coverage with metronidazole should be considered for grossly contaminated wounds or those whose treatment has been delayed more than 18 hours.

→ **Phenytoin** should be administered in a 17 mg/kg load, which may be placed in a normal saline

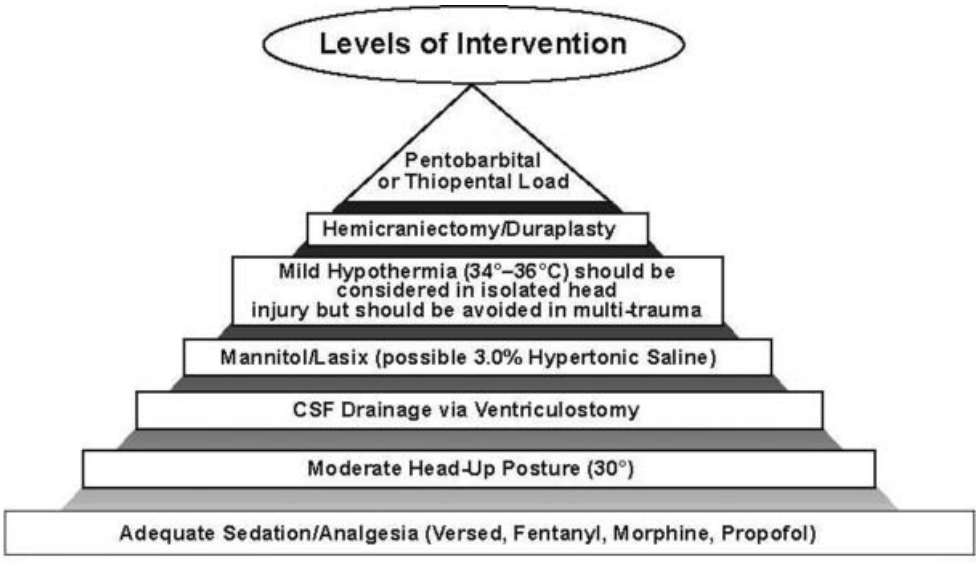
piggyback and given over 20-30 minutes (no more than 50 mg/min, because rapid infusion may cause cardiac conduction disturbances).

→ A maintenance dose of 300-400 mg/d, either in divided doses or once before bedtime, should be adequate to maintain a serum level of 10-20 Hg/L.

→ **Measure** serum chemistries daily to monitor for hyponatremia.

→ **Monitor** and treat coagulopathy aggressively.

→ **Monitoring** of ICP is recommended for patients with GCS< 8 (in essence, it is a substitute for a neurologic examination).



1) Sedation, head elevation, and paralysis.
2) CSF drainage if a ventricular catheter is in place. (Surgery)
3) Hyperventilation to a PCO2 of 30 to 35 mm Hg only until other measures take effect. (Prolonged levels below this are deleterious as a result of small vessel constriction and ischemia.)
   - Refractory intracranial hypertension should be managed with an initial bolus of 1g/kg of mannitol and intermittent dosing of 0.25-0.5 g/ kg q4h as needed.
   - Aggressive treatment with mannitol should be accompanied by placement of a CVP line or even a PA catheter because hypovolemia may ensue.
4) Any patient who develops intracranial hypertension or deteriorates clinically should undergo prompt repeat CT.
5) Mild hypothermia may be considered in isolated head injury, but avoid in the multitrauma patient. Treat hypovolemia with albumin, normal saline, hypertonic saline or other volume expanders to create a euvolemic hyperosmolar patient (290-315 mOsm/L).

Blast over-pressure CNS injuries

Supportive medical therapy is usually sufficient. Only in rare cases is an ICP monitor, ventriculostomy, or cranial decompression necessary.

In the absence of hematomas the use of magnesium has been beneficial. Structures particularly sensitive include optic apparatus, hippocampus, and basal ganglia.

Delayed intracranial hemorrhages have been reported. Additionally, these patients have a higher susceptibility to subsequent injury and should be evaluated at a level 4/5 facility.

Repetitive injury and exposure to blast over- pressure may result in irreversible cognitive deficits.

General medevac rule

Neurosurgical patient after operation is ready for transportation.

Before surgery to the nearest neurosurgical unit only!