**Glasgow Coma Scale**

|  |  |
| --- | --- |
| **Component** | Score |
| **Eye opening****(AVPU)** | 4 – Spontaneous3 – In response to speech2 – In response to pain1 – No eye opening |
| **Verbal response** | 5 – The patient knows who they are, where they are and why, the year, season and month4 – Confused conversation. 3 – Inappropriate speech. Random or exclamatory speech but no conversational exchange2 – Incomprehensible speech. Moaning but no words1 – No speech |
| **Motor response** | 6 – obeys commands. The patient does simple things5 – Localising pain response. E.g. supraorbital pressure. A purposeful movement towards the painful stimulus4 – Withdraws to pain. Pulls limb away from painful stimulus3 – Flexor response to pain. Nailbed pressure causes abnormal flexion of limbs (decorticate posturing). Other signs → arm bend inward to the chest, thumb tucked into a clenched fist, legs extended. **Damage above the red nucleus**2 – Extensor response to pain. Limb extension (adduction, internal rotation of the shoulder, pronation of the forearm). Decerebrate posturing. **Damage below the red nucleus**1 – No movement in response to pain |

**GCS Severity:**

**Minor 🡪 13 to 15 ; Moderate 9 – 12 ; Severe injury <8**

**Coma**

* A state of **unconsciousness** lasting more than **6 hours** in which a person
	+ Cannot be awakened
	+ Fails to respond normally to: pain, light, sound
	+ Lacks a normal sleep-wake cycle
	+ Does not initiate voluntary movement
	+ GCS is normally between 3 and 8
* **Differential**

|  |  |
| --- | --- |
| Classification | Examples |
| **Poisoning** | Drugs (iatrogenic in some cases)AlcoholCarbon monoxide |
| **Traumatic** | ConcussionHaematoma → subdural, exdradural |
| **Vascular** | Stroke → SAH, large infarctions or haemorrhages |
| **Neurological** | Status epilepticus or post-ictal state |
| **Metabolic** | DiabeticUraemiaHepatic |
| **Infective** | MeningitisEncephalitisSepticaemia |
| **Anoxic** | PneumoniaHypoxic-hypotensive encephalopathy → e.g. following MI or profound shock |
| **Structural** | Brainstem herniation (secondary to any cause)Tumours |

**Definitions of Brain death and Persistent Vegetative State**

**Brain Death**

* Irreversible brain damage may have occurred with **permanent destruction of brainstem function** and hence the death of the patient YET CVS function may remain stable and respiration can be maintained by artificial support
* Formal criteria used to decide whether cardiorespiratory support should be withdrawn
	+ **CNS depressant drugs** should not contribute to the clinical start
	+ The patient must be on a **ventilator** due to inadequate spontaneous respiration 🡪 **exclude neuromuscular blocking drugs**
	+ **Hypothermia** and **severe metabolic disorders** must not be the primary cause of the patients condition
	+ The cause of the patients condition must be established and compatible with **irreversible brain damage**
* **Tests must be carried out by 2 doctors with appropriate expertise**
	+ Repeat @ interval 🡪 death certified @ second set of tests
	+ EEG of no value
* **6 tests (test brainstem function)**
	+ No **pupillary response** to light 🡪 pupil fixed and unresponsive
	+ Absent **corneal reflexes**
	+ Absent **vestibule-occular reflex** 🡪 no eye movement following injection of 50ml of ice cold water over 1 minute into external acoustic meatus (normally eyes should deviate away from cold)
		- CONFIRM access to the tympanic membrane by **otoscopy**
	+ No gag or response to **tracheal suction**
	+ No motor response in cranial nerve territory to **painful stimuli**
	+ No **respiratory movement** when the patient is disconnected from a ventilator (PaCO2 rises to above 6.65kPa)

**Persistent Vegetative State**

* Unlike a coma (where awareness and wakefulness are absent)
* **Patients are unaware of the environment** yet **able to breath** spontaneously, with a stable circulation and cycles of eye opening that resembles a **sleep wake cycle**

**Urgent Investigations Required**

* Blood tests (urgent)
	+ FBC, U&E, LFT, Calcium, Glucose, Clotting, ABG
* Toxicology 🡪 paracetamol, salicylates, ethanol
* Imaging 🡪 CXR, CT head
* Lumbar puncture if CT normally

**Complications and their prevention in coma**

|  |  |
| --- | --- |
| Complication | Prevention |
| **Bedsores** | Good nursing care, e.g. **turning the patient every 2-3 hours** |
| **Infection** | **Pneumonia**, usually due to aspiration, lack of gag reflex or from the feeding tube. **Careful feeding and monitoring.** Simples measures, e.g. feeding with the patient tilted up, may help**UTI** → secondary to **long-term catheters** |
| **Atelectasis** | ? chest physio, alteration of ventilator settings |
| **Malnutrition** | NG feeding and careful fluid resuscitation |
| **Contractures** | Physiotherapy may have a role in prevention |
| **Persistent vegetative state** | Coma becomes permanent |

**Prognosis of different Comas**

* Predictors of prognosis in coma include cause, depth, duration and clinical signs
* After head injury, prognosis is directly proportional to the initial GCS
* Infection 🡪 Depends on rapid treatment

|  |  |
| --- | --- |
| Associated with a good prognosis | Associated with a poor prognosis |
| * Drug overdose → high proportion of good recovery if adequately treated
* Metabolic → 25% good recovery
 | * Cerebrovascular disease → 75% die, 3% full recover
* Hypoxic-ischaemia → 60% die, 8% good recover
 |

**Immediate Management of the Unconscious Patient**

* **Airway and C spine**
	+ Assess for signs of obstruction and patency of airway
	+ Establish patient airway; if GCS <8 consider intubation
* **Breathing**
	+ RR, Bilateral chest movement, percussion and auscultation
	+ If no respiratory effort, treat as an arrest and if breathing compromised give O2
* **Circulation**
	+ Assess 🡪 BP, pulse, cap refill, evidence of haemorrhage, IV access
	+ Management 🡪 as for shock
* **Disability**
	+ AVPU (alert, voice, pain, unresp.) , check pupil size and GCS if time
* **Expose** the patient appropriately

**Treatment for reversible causes of unconsciousness**

* **Hypoglycaemia**
	+ **100ml/hr of 10% glucose IV**
* **Alcoholism**
	+ **IV Thiamine**
* **Drug overdose**
	+ **Paracetamol 🡪 N-Acetylcysteine**
		- **Within 4 hours of ingestion**
			* **<1hr activated charcoal**
		- Measure plasma concentrations at **4 hours post ingestion**
		- If levels are above the treatment line on the treatment graph give NAC (**150for60; 50for4; 100for16)(200,500,1L**)
			* **150mg/Kg (maximum 16.5g) in 200mL 5% glucose as IV infusion over 60 minutes**
			* **50mg/Kg (maximum 5.5g) in 500mL 5% glucose as IV infusion over next 4 hours**
			* **100mg/Kg (maximum 11g) in 1L 5% glucose as IV infusion over next 16 hours**
		- **If found within 8-15hrs** of ingestion, start infusion immediately and take blood for concentrations
		- **If found within 15-24hrs** start NAC immediately and take bloods
		- **If @ 24h ;** INR, ABG, Blood and creatinine. If paracetamol concentration <10mg/L then the N-acetylcysteine infusion can be stopped
			* If any of these are abnormal then consider NAC @ 150mg/kg
		- **If presenting after 24h** take blood for paracetamol concentrations and if the patient is asymptomatic and the INR, LFT, venous bicarbonate and plasma creatinine figures are normal then they can be seen as medically fit; if symptomatic/abnormal then discuss
		- **Contact specialist liver centre if** INR post ingestion >2 @24h, >4@48h and >6@72h; other indices of severe hepatotoxicity
	+ **opiates**🡪 **Naloxone** **0.4 – 2mg IV**
	+ **Benzodiazepines** 🡪 **Flumazenil**
	+ **Aspirin 🡪** causes respiratory alkalosis and renal excretion of sodium, potassium and water resulting in metabolic acidosis with dehydration and electrolyte imbalance
		- If patient has tinnitus, it is likely that plasma concentration >400
		- Treat with gastric lavage and 50g activated charcoal IV fluids, and correct electrolyte abnormalities
		- If >500, 1litre of 1.26/1.4 sodium bicarbonate with 40mmol K+ IV over 4 hours
* Infection
	+ **Meningitis**
		- **Ceftriaxone IV 4g OD + Aciclovir IV 10mg/kg + Amoxicillin IV 2g (if immunocompromised)**
	+ **Encephalitis (herpes)**
		- **Aciclovir**

**Complications**

* **Neurological complications**
	+ Early
		- Abnormal consciousness → stupor, coma, PVS, licked-in syndrome
		- Cognitive deficits → memory, judgement, processing, reasoning
		- Post-concussion syndrome
		- Sensory deficits → especially vision
		- Changes in personality EDH/SDH
		- Seizures
	+ Late
		- Chronic sub-dural haematoma
		- Seizures
		- Diabetes insipidus
		- Parkinsonism, Dementia
* Physical complications
* Early: headache, DVT/PE

**Classification of Head Injury**

* **Minor**
	+ Unconscious and posttraumatic amnesia lasting <30 minutes
* **Moderate**
	+ Skull fracture and/or unconsciousness lasting >30mins
* **Severe**
	+ Unconsciousness lasting >24h and/or focal brain damage

**Initial GCS:**

* Mild → 13-15. Mortality 0.1%
* Moderate → 9-12. Mortality 10%
* Severe → 3-8. Mortality 40%

**Pathophysiology:**

* **Primary insult**
	+ Skull fracture 🡪 signs of basal skull fracture (**panda eyes, CSF leak from nose, Battle’s Sign (behind ear), Haemotypanum**
	+ Contusion, Haematoma, Haemorrhage
	+ Axonal shearing
* **Secondary**
	+ Cerebral oedema, Raised ICP, Haemorrhage, Ischaemia, Infection

**Drug Overdose**

* **ABCD**
	+ **+ paracetamol + salicylate blood levels** (4h after ingestion if timing is possible)
	+ **Blood, urine, gastric fluid** for toxicology
	+ **+ capillary blood glucose**
	+ **+ pregnancy test**
	+ **+ U&E, renal and liver function, acid base balance**
		- **Urine output (<400mL/24h)**
	+ **+ body temperature**
* **ECG**
* **CXR** (if aspiration is a possibility)
* **Identify the poison**
	+ History from patient or relatives
	+ Retain tablets or containers found with the patient
	+ **TOXBASE** for info
* **Prevent absorption of drug and poison**
	+ **Gastric Lavage**
		- If presentation is **within 1 hour of ingestion**
		- Contraindications:
			* The patient has depressed conscious level (unless airway is protected by cuffed ET tube
			* If substance is hydrocarbon/corrosive
			* Patient @risk of GI haemorrhage or perforation
	+ **Activated charcoal (50-100g) as a single dose**
		- If presentation is within **1 hour of ingestion** of a potentially toxic amount of drug known to be absorbed by charcoal i.e. antiepileptics, analgesics, cardiac drugs, antidepressants
		- Can take multiple doses i.e. when the blood concentrations in salicylate poisoning are still rising
		- Contraindications:
			* Drugs not absorbed by activated charcoal (metals, alcohols, acids and alkalis)
			* Decreased conscious level unless airway is protected by a cuffed ET tube
		- Complications: aspiration & GI obstruction
	+ **Whole bowel irrigation**
		- Given by mouth/NG tube using **reconstituted polyethylene glycol (4 sachets of Klean-Prep oral powder dissolved in 4 litres of water) at 1500 – 2000ml/hr**
		- Patient should be seated or @ least 45degrees and continue until rectal effluent is clear
		- Life threatening overdose of a sustained release or enteric coated drug (drug not absorbed by activated charcoal)i.e. iron and lithium
		- Also useful for infestion/insertion into lower GI tract of packets of illicit drugs
		- Contraindications:
			* Bowel obstruction, perforation, ileus, GI haemorrhage
			* Haemodynamic instability
			* Compromised, unprotected airway

**Management**

* **CT Head**
* **Involvement of neurosurgeons if**
	+ GCS persistent at <8 after resuscitation
	+ Unexplained confusion >4h
	+ Deterioration in GCS after admission
	+ Progressive focal neurological signs
	+ Seizures without full recovery
	+ Penetrating injury
	+ CSF leak
* **ABCDE**
* **SECONDARY SURVEY**
	+ **Neurological examinations**
		- Chart temperature, pulse, BP, respirations and pupils every 15 minutes
		- Assess anterograde and retrograde amnesia
		- Meticulous airway and bladder care
	+ **Note signs of fractures**
	+ **Check for CSF leak**
	+ **Radiology C-spine**

**Cerebral Contusion**

* **A focal brain injury** caused primarily by impact of the brain surface against bony ridges of the skill
* Most commonly found at frontal, temporal and occipital poles and cerebellar hemispheres
* **Clinical features:** headache, confusion and dizziness, loss of consciousness, N&V, seizures, cognitive difficulties (memory and thinking), vision, hearing or speech may be affected, local signs depend on the area affected.
* **Treatment**
	+ Monitor and examine regularly, treat raised ICP and debridement of affected area if ICP doesn’t go down

**Intracranial Haematoma**

* **Intra-axial -** Bleeding within the brain itself
* **Extra-axial –** Bleeding Within the skull but outside the brain
	+ Extra-dural haemorrhage 🡪 due to rupture of the middle meningeal artery
	+ Subdural haemorrhage 🡪 tearing of veins in the subdural space
	+ Subarachnoid 🡪 from trauma or rupture of an aneurysm

|  |  |  |
| --- | --- | --- |
| **Type**  | **Typical presentation** | **Management Overview** |
| **Intra-axial** | Focal neurological signs or signs of raised ICP, including **vomiting and coma** | Medical → **antihypertensives,** Manage ICP with **mannitol** (reduce ICP) , **elevate head** of the bed, **hyperventilate** (if intubated) and **fluid restrict**Surgical → **catheter embolization of vessels**, aspiration of damaged areas |
| **Extra-dural** | **Initial LOC followed by a lucid interval,** then progressive neurological decline (headache, vomiting, confusion, fits, up going plantar reflexes, ipsilateral pupil dilatation, coma and eventually coming) | Surgical → **surgical evacuation of clot through a burr hole**Managed raised ICP as above |
| **Sub-dural haematoma** | Unlike EDH, these patients have **LOC at the time of injury and do not regain in the interval.**  | Surgical → evacuation via **burr hole** |
| **SAH** | Sudden onset **‘thunderclap headache’** associated with meningism | Medical → **calcium channel antagonists** to reduce vasospasm and maintain cerebral perfusionSurgical → **clipping or embolization of aneurysm** |

**Subdural haematoma**

* No lucid interval as seen in Extradural haematoma
* Findings associated with raised ICP 🡪 N&V, depressed consciousness, false localising signs
* Conservative 🡪 possible for small lesions with no neuro signs
* Medical 🡪 **mannitol, hyperventilation and raising the patients head**
	+ **Antileptics / Coagulopathy**
* **Surgical 🡪 craniotomy**
* **Chronic Subdural haematoma**
	+ Also caused by trauma however this may be mild and could have occurred up to 9 months ago and so may not be remembered
	+ Elderly patients particularly at risk due to cerebral atrophy
	+ Patients with a coagulopathy e.g. Warfarin/Alcoholic liver disease are particularly risk
	+ Typically presents: **fluctuating levels of consciousness,** dementia/confusion, epilepsy, sleepiness, personality change
	+ Signs: raised ICP: Absent venous pulse in retina, false localising signs/localising signs, weakness or sensory changes, incontinence

**Presentation of Acute brain herniation after trauma**

* **Uncal herniation**
	+ **Early signs –** ipsilateral pupillary dilation
	+ **Late – dcccccccddfg- in te brain herniation after trauma s every 15 minutes hours erully fit; if symptomatic/abnormal then discuss i**complete ipsilateral 3rd nerve palsy, LOC, contralateral hemiplegia secondary to mass, ipsilateral hemiplegia due to compression of contralateral cerebral peduncle against the edge of tentorium, flaccid paralysis
* **Tonsilar herniation**
	+ Can be precipitated by LP in the presence of a posterior fossa mass
	+ Signs: head tilt or neck pain, ataxia, 6th nerve palsy and upgoing plantar reflexes, resp arrest, LOC and flaccid paralysis

**Post Traumatic CSF Leak**

* **Conservative**
	+ Elevate patients head to 30 and avoid straining
		- **Laxative, anti-emetics** and **anti-tussives**
* **Surgical**
	+ Lumbar puncture or lumbar drain 🡪 reduces ICP and may allow leak to repair
	+ Operative intervention may required if persistent

**Treatment of skull fractures**

* **Open Skull Fractures**
	+ **Depressed 🡪** significant risk of infection 🡪 operative irrigation, debridement and removal of depressed fragments
	+ **Non depressed 🡪** conservative management (inspection, cleansing and scalp suturing
* **Closed skull fractures**
	+ No specific treatment but monitor for extradural haemorrhage
* **Basilar (**Battle’s sign, racoon sign and hemotympanum**)**
* **Surgery if persistent CSF Leakage** (due to the risk of meningitis)

