MKAIC

Muhimbili-Karolinska
Anaesthesia &
Intensive Care Collaboration

Intensive Care
Muhimbili National Hospital

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- Three Most Common Admissions to MNH ICU

- CEREBRAL VASCULAR ACCIDENT
    - Haemorrhagic type
- PERITONITIS
- COMPLICATIONS FOLLOWING SPINAL ANAESTHESIA

- Hemorrhagic stroke occurs when a blood vessel bursts inside the brain.
- **The brain is very sensitive to bleeding and damage can occur very rapidly**.
- Bleeding irritates the brain tissue, causing swelling.
- Bleeding:
  - collects into a mass a hematoma.
  - increases pressure on the brain and presses it against the skull.

Classification

- Grouped according to location of the blood vessel:
  - INTRACEREBRAL HAEMORRHAGE:Bleeding in the brain
  - SUBARACHNOID HAEMORRHAGE: Bleeding in the area between the brain and the thin tissues that cover the brain
  - Hemorrhagic stroke is most often due to high blood pressure, which stresses the artery walls until they break.
    - Other causes of hemorrhagic stroke include:
      - Aneurysms which create a weak spot in an artery wall, which can eventually burst
      - Abnormal connections between arteries and veins, such as arteriovenous malformation (AVM)
      - Cancer, particularly cancer that spreads to the brain from distant organs such as the breast, skin, and thyroid
      - Cerebral Amyloid Angiopathy, a build up of amyloid protein within the artery walls in the brain, which makes bleeding more likely
      - Conditions or medications (such as aspirin or Warfarin) that can make one bleed excessively
      - Illicit drugs, such as cocaine

SYMPTOMS

- The symptoms vary depending on the location of the bleed and the amount of brain tissue affected.
- Symptoms usually develop suddenly, without warning, and often during activity.
- They may come and go (be episodic) or slowly get worse over time
- Symptoms may include:
- Change in alertness (level of consciousness)
  - Apathetic
  - Coma
  - Lethargy
  - Sleepiness
  - Stupor
  - Unconsciousness
  - Withdrawn
- Difficulty Speaking or understanding others
- Difficulty Swallowing
- Difficulty writing or reading
- Headache

INVESTIGATIONS

- A neurologic exam is almost always abnormal.
- The patient may look drowsy and confused.
- An eye examination may show abnormal eye movements and changes in the back of the eye.
- The patient may have abnormal reflexes. (these findings do not necessarily mean a person is having a brain hemorrhage, and could be due to another medical condition).
- **A Computed Tomography (CT scan) of the brain is the most important test used to confirm a brain hemorrhage**. (It should be done without delay).
- A brain Magnetic Resonance Imaging (MRI) scan can be done later to better understand what caused the bleeding.
- Conventional angiography may be done in some cases to identify aneurysms or an arteriovenous malformation although CT and MRI are more often used.

- Other tests may include:
- Complete Blood Count (FBC)
- Blood clotting studies -Prothrombin time (PT) and Partial Thromboplastin Time (PTT)
- CSF (cerebral spinal fluid examination)

MANAGEMENT

- Hemorrhagic stroke requires prompt medical attention.
- It can develop quickly into a life-threatening situation.
- Goals of treatment ‘ABCDE’ are to:

  SAVE THE PATIENT’S LIFE

  – Relieve symptoms
  – Repair the cause of bleeding
  – Prevent complications
  – Start rehabilitation therapy as soon as possible

TREATMENT

- Treatment is ideally given in a hospital's intensive care unit (ICU), **where complications can immediately be detected**.
- The health care team will pay careful attention to **breathing because sometimes bleeding in the brain can cause breathing to stop or become irregular**.
  - A person having a hemorrhagic stroke may be unable to protect their airway when they cough or sneeze because of impaired consciousness.
  - Saliva or other secretions may go "down the trachea," which may cause potentially serious aspiration pneumonia.
    - An **Endotrachea tube may needed to treat or prevent these problems**.

- Carefully controlling blood pressure, which can be too high or too low
- Drugs to control brain swelling – Mannitol, Dexamethasone, VPshunt
- Medications to relief headaches
  - (but should be used with caution because they may reduce alertness and give the wrong impression that the patient is getting worse)
- Seizure medications such as phenytoin, (Midazolam, Diazepam, Thiopental Na)
- The patient will need to rest in bed avoiding activities that may increase the intracranial pressure, such as:
  - Bending
  - Lying flat
  - Sudden position changes
  - Straining during bowel movement (**stool softeners or laxatives may be prescribed**)
- Nutrients and fluids may be necessary, especially if the person has swallowing difficulties.
  - These may be given through a vein (intravenously) or a feeding gastrostomy tube in the stomach.
  - Swallowing difficulties may be temporary or permanent.

? SURGERY

- **Sometimes**, surgery is needed to save the patient's life or to improve the chances of recovery.
- The type of surgery depends upon the specific cause of brain bleeding.
- A hemorrhage due to an aneurysm or AVM requires special treatment
- Other types of bleeding, removal of the hematoma may occasionally be needed, especially when bleeding occurs in the back of the brain.
- One common problem related to brain bleeding is hydrocephalus, which is the build up of fluid within the brain - ventriculostomy may be needed to drain the fluid.

LONG TERM TREATMENT

- ***The goal of long-term treatment is to help the patient recover as much function as possible and prevent future strokes***.
- The recovery time and need for long-term treatment differs from person to person.
- Depending on the symptoms, rehabilitation may include:
  - Occupational therapy
  - Physical therapy
  - Speech therapy
  - Therapies such as repositioning and range-of-motion exercises can help prevent complications related to stroke, such as infection and bed sores.
  - Those who have had a stroke should try to remain as active as physically possible.

PREVENTION

- Prevention is extremely important because treatment for hemorrhage-related brain injury often cannot reverse brain damage.
- Most cases of hemorrhagic stroke are associated with specific risk factors such as high blood pressure, smoking, or cocaine use.
- Controlling blood pressure and avoiding smoking and cocaine can reduce the risk for brain bleeding.
- Surgery to correct blood vessel abnormalities such as aneurysms or AVMs is sometimes recommended to prevent bleeding.

PERITONITIS

- An inflammation of the peritoneum, the thin membrane that lines the abdominal wall and covers the organs inside.
- The inflammation is caused by a bacterial or fungal infection of this membrane.
- There are two major types of peritonitis.
  - **Primary** peritonitis is caused by the spread of an infection from the blood and lymph nodes to the peritoneum. Rare -- less than 1% of all cases of peritonitis.
  - **Secondary** peritonitis, the **more common** type of peritonitis, happens when the infection comes into the peritoneum **from the gastrointestinal or biliary tract**.
- Both cases of peritonitis are very serious and can be life threatening if not treated quickly.

- Tertiary Peritonitis:
    - Late peritonitis like syndrome due to disturbance in immune response of the patient
    - Peritonitis without evidence of pathogen
    - Peritonitis with fungi
    - Peritonitis with low-grade pathogenic bacteria

CAUSES:

- Primary peritonitis is usually caused by liver disease.
  - Fluid builds up in the abdomen, creating an environment for bacteria to grow.
- Secondary peritonitis is caused by other conditions that allow bacteria, enzymes, or bile into the peritoneum from a hole or tear in the gastrointestinal or biliary tracts.Such tears can be caused by:
    - Pancreatitis,
    - Ruptured appendix,
    - Stomach ulcer,
    - Crohn's disease,
    - Diverticulitis.
    - Peritoneal dialysis, which uses the blood vessels in the abdomen to filter waste from the blood renal diseases causing peritonitis.

RISK FACTORS:

- Risk for primary peritonitis:
  - Liver disease (cirrhosis)
  - Fluid in the abdomen
  - Weakened immune system
  - Pelvic inflammatory disease
- Risk for secondary peritonitis include:
  - Appendicitis (inflammation of the appendix)
  - Stomach ulcers
  - Torn or twisted intestine
  - Pancreatitis
  - Inflammatory bowel disease, such as Crohn's disease or ulcerative colitis
  - Injury caused by an operation
  - Peritoneal dialysis
  - Trauma

SIGNS AND SYMPTOMS

- Swelling and tenderness in the abdomen with pain ranging from dull aches to severe, sharp pain
- Fever and chills
- Loss of appetite
- Thirst
- Nausea and vomiting
- Reduced urine output
- Not being able to pass gas or stool

DIAGNOSIS

- Peritonitis can be life threatening
- **Palpation** (feel and press) of the abdomen to find any swelling, tenderness as well as signs that fluid has collected in the area.
- **Auscultation** for bowel sounds, check for difficulty breathing, low blood pressure, and signs of dehydration.
- **Laboratory**:
  - Blood tests -- to see if there is bacteria in your blood
  - Samples of fluid from the abdomen -- identifies the bacteria causing the infection
- **X-rays** -- detect air in the abdomen, which indicates that an organ may be torn or perforated
- **CT scan** -- identifies fluid in the abdomen, or an infected organ

MANAGEMENT

- Peritonitis is a medical emergency that calls for hospitalization for treatment.
- Surgery may be needed to get rid of the source of infection, such as an inflamed appendix, or to repair a tear in the walls of the gastrointestinal or biliary tract.
- Antibiotics are used to control infection.
- Secondary peritonitis is the most frequent form of peritonitis, characterized by a high disease burden and a high mortality rate.
- Choice of adequate antibiotics is an independent factor for survival.
- **Complementary therapies may be used along with conventional medicine when recovering from peritonitis**.

SUPPORTIVE TREATMENT:

- Supportive measures:
  - To combat hypovolemia, shock and to maintain adequate tissue oxygenation
  - To treat bacteria not eliminated by surgery with antibiotics
  - To support failing organ systems
  - To provide adequate nutrition

SURGICAL REPAIR:

- To repair and/or control source of infection
- Evacuate bacterial inoculum, pus and adjuvants
- Treat abdominal compartment syndrome
- Prevent or treat persistent and recurrent infections or verify repair and purge

STAGED ABDOMINAL REPAIR

- Critical patient condition, due to haemodynamic instability, precluding definitive repair
- Excessive peritoneal oedema preventing abdominal closure without under tension
- Abdominal compartment syndrome, pulmonary, cardiac, hepatic or renal dysfunction, decreased visceral perfusion
- IA >15mmHg
- Massive abdominal wall loss
- Impossible to eliminate or control source of infection
- Incomplete debridement of necrotic tissue
- Uncertainty on the viability of remaining bowel tissue
- Uncontrolled bleeding (the need for packing)

ACUTE ‘ABCDE’ MANAGEMENT

- **Nursing Diagnosis**: Deficient fluid volume related to intravascular fluid shift to the peritoneal space and inability to ingest oral fluids.
- **Outcome Criteria**
- Central venous pressure 2 to 6 MM Hg
- BP 90 to 120 mm Hg
- Mean arterial pressure 70 to 105 mm Hg
- Pulmonary artery systolic 15 to 30 mm Hg
- Pulmonary artery diastolic 5 to 15 mm Hg
- HR 60 to 100 beats/min
- Urine output 30 ml/hr
- **Patient Monitoring**
- Obtain pulmonary artery pressure and central venous pressure and monitor mean arterial pressure hourly or more frequently if the patient’s hemodynamic status is unstable.
- Not the patient’s response to all therapy.
- Monitor fluid volume status by measuring urine output hourly and measure nasogastric and other bodily drainage.
- Determine fluid balance every 8 hours.
- Continuously monitor ECG fir dysrhythmias resulting from electrolyte disturbances.

- **Patient Assessment**
- Assess tissue perfusion. Note level of consciousness, skin color and temperature, pulses, and capillary refill.
- Assess hydration status: note skin turgor on inner thigh or forehead, condition of buccal membranes, and development of edema or crackles.
- Assess the patient's abdomen for resolution of rigidity, rebound tenderness, and distention. Auscultate bowel sounds.
- **Diagnostic Assessment**
- Review serum sodium and potassium levels, which may become depleted with nasogastric suctioning or fluid shifts.
- Review serial WBC count and differentiated to evaluate the course of action.
- **Patient Management**
- Administer crystalloid or colloid solutions to improve intravascular volume.
- Replace potassium as ordered; validate adequate urine output before administration.
- Keep the patient NPO during acute phase and before evaluation by a surgeon.
- Provide nutritional support as indicated; most patient will benefit from postpyloric delivery of early enteral nutrients at a minimal hourly rate to prevent bacterial translocation and sepsis.
- Administer antibiotics as prescribed after appropriate cultures obtained.

REGIONAL ANAESTHESIA COMPLICATIONS

Regional Anaesthesia Complications

Major complications of regional anaesthesia in descending order of severity and ascending order of frequency

Complications in order of frequency	Estimated frequency	Comments
1= Direct nerve damage	1:10,000 – 1:30,000	No effective treatment Most improve over 1–6 months
1= Spinal haematoma	1:150,000 – 1:220,000	Requires urgent evacuation May cause paraplegia
1= Spinal infection	1:100,000 – 1:150,000	Evacuation and aggressive antibiotic therapy required. May cause paraplegia
4= Drug error	Unknown	Avoidable. May be fatal
4=Systemic toxicity	Unknown	May be fatal unless treated promptly
6= Respiratory depression	Unknown	Beware neuraxial opioids
6= Hypotension	Common with epidural/spinal	Treat effectively to avoid complications (see text)
8 Confusional states	Common in the elderly	Beware neuraxial opioids
9 Pruritus/urinary retention/nausea	Up to 16% incidence	Treat effectively
10 Technical failure	5–25% for different techniques	Consider alternative strategy

- Major regional anaesthetic techniques produce physiological consequences arising from the motor, sensory and autonomic blockade.
- It is important to distinguish side-effects from potential complications to prevent the former becoming the latter.
- Side-effects are predictable and can be beneficial
- complications are usually unexpected and may cause harm even when recognized and treated promptly.

- Key note

- The effects of motor, sensory and proprioceptive block are minimized by adequate support for the affected limbs, avoidance of joint hyperextension or flexion and protective padding of pressure areas, especially heels and elbows.
- Autonomic nerve dysfunction may require the urinary bladder to be catheterized until the block regresses and the pulse rate and blood pressure may need supporting with intravenous fluids, inotropic and vagolytic drugs.
- In healthy patients without cardiorespiratory risk factors, the systolic blood pressure may drop by 30% from its preoperative value without any increase in risk to the patients.
- In older patients, or those with cardiac risk factors, a reduction of 20–25% is the maximum before treatment should be initiated to restore it towards the normal value for the individual patient.
- In patients under 45 years (especially fit young men), spinal anaesthesia may produce bradycardia without significant hypotension and, in this patient group, heart rates of less than 50 beats/min may be associated with sudden cardiac arrest.
- It is sensible to maintain the heart rate above 50 beats/min in all patients undergoing spinal or epidural blockade.

TECHNIQUE RELATED COMPLICATIONS

- **Failure:**
  - many regional blocks are technically demanding and failure rates range from 5 to 25% for different techniques.
  - One of the most significant risk factors for serious neurological damage is multiple attempts at a regional technique.
  - If technical difficulties are encountered, a rapid risk assessment is necessary to evaluate whether to continue with the procedure, to seek more experienced assistance or to consider an alternative anaesthetic technique.
- As a general rule, two to three unsuccessful attempts are an indication to seek help or use an alternative technique.
  - This particularly applies to central neuraxial blocks and brachial plexus blocks.
  - The 'failed spinal' is a specific example where a repeat injection should not be made.
  - If CSF is aspirated and local anaesthetic injected but an adequate spinal block does not occur, **repeated local anaesthetic injection can cause neurological damage, probably because of maldistribution of the drug in the CSF and localized nerve toxicity resulting from high concentration of local anaesthetic around a few nerve roots**.

TOTAL SPINAL

- **T**he inadvertent injection of a large (20–30 ml) volume of local anaesthetic into the CSF is a particular hazard of epidural blocks but can happen with cervical plexus, interscalene brachial plexus, paravertebral and psoas sheath blocks.
- The spinal nerve root dural cuffs may extend beyond the intervertebral foraminae and local anaesthetic can be injected into CSF with these techniques.
- **The effects of a total spinal can be catastrophic unless rapid and effective treatment is instituted**.
- All anaesthetists performing such blocks must be able to recognize the signs and symptoms of a total spinal and be able to treat them appropriately.
- Treatment is aimed at maintaining adequate oxygenation, protecting and securing the airway in the event of unconsciousness, restoring stable haemodynamics, and supporting the patient until the effects of the total spinal regress

- The signs vary depending on the volume injected and the spinal level at which the injection is made.
- Typically there is rapid onset of profound motor blockade and high dermatomal sensory block (within 1–5 minutes) as the local anaesthetic spreads rostrally in the CSF.
- It may reach the cervical spine and produce motor weakness of the upper limbs and respiratory embarrassment or even respiratory failure as the phrenic nerves are blocked.
- If the local anaesthetic reaches the cerebral CSF, unconsciousness, respiratory arrest, severe hypotension and pupillary dilatation occur.

- Key Note

C O M A

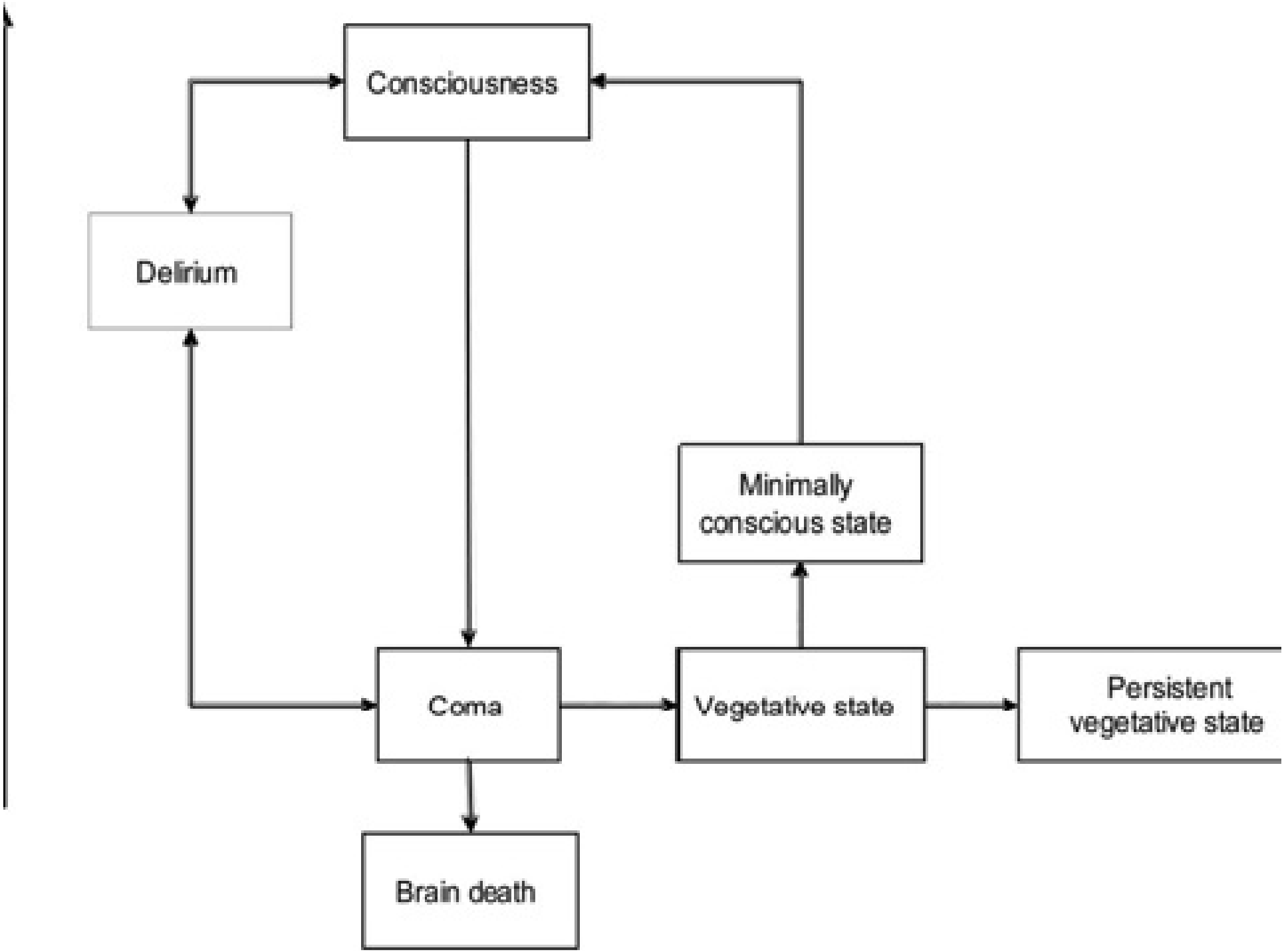
- As opposed to states of transient unconsciousness such as syncope or concussion, coma must last >1 hr.
- Comatose patients have no eye opening and do not speak or move spontaneously.
- They do not follow commands, and when provoked by a noxious stimulus their eyes remain closed, vocalization is limited or absent, and motor activity is absent or abnormal and reflexive rather than purposeful or defensive .
- Sleep-wake cycles are lacking.
- **Coma is typically a transitional state, evolving toward recovery of consciousness, the vegetative state, the minimally conscious state, or brain death**.
- Coma is associated with injury to or functional disruption of bilateral cortical structures or of the RAS.
- Lesions involving the brainstem portion of the RAS frequently coexist with oculomotor findings and pathologic breathing patterns.

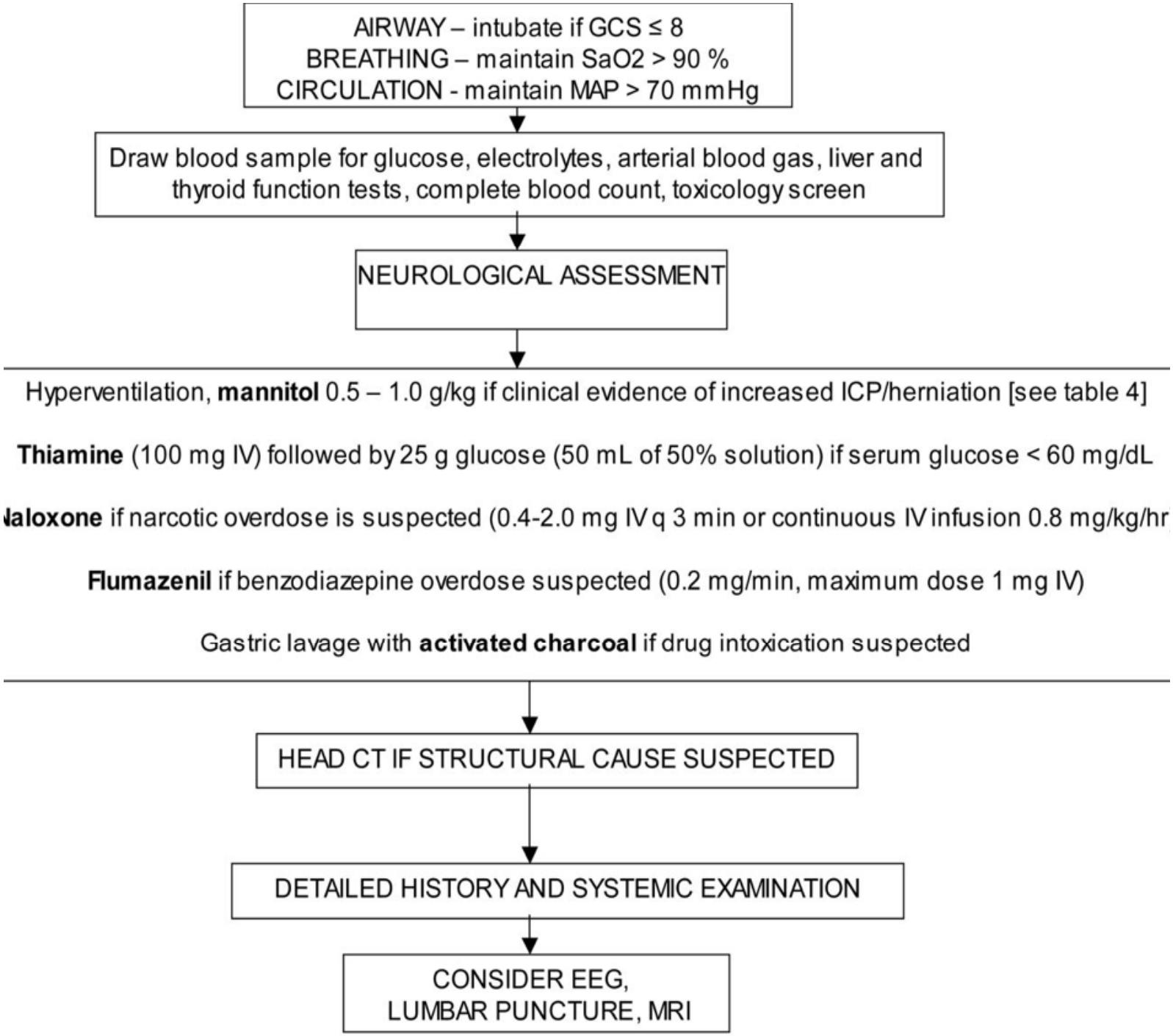
VEGETATIVE STATE

- The vegetative state(VS) is notable for preserved arousal mechanisms associated with a complete lack of self or environmental awareness.
- Patients in a VS open their eyes spontaneously:
  - there is no evidence of sustained visual pursuit (tracking) or visual fixation.
- They do not follow commands and do not move in any meaningful or purposeful manner.
- They evolve through temporal cycles of increased and decreased arousal akin to a “sleep-wake” pattern.
- Cardiovascular regulatory function, breathing patterns, and cranial nerves are usually intact.
- Some patients regain partial or complete consciousness, others remain for extended periods without significant changes in their neurologic state, prompting the term “persistent vegetative state” (PVS).
- The VS is caused by widespread damage to bilateral cerebral hemispheres with sparing of the brainstem;
- trauma and hypoxic-ischemic encephalopathy are the most common acute causes of VS.

MINIMALLY CONSCIOUS STATE

- The minimally conscious state (MCS) describes a subset of patients who do not meet the criteria for coma or VS.
  - Patients in MCS have a severe alteration in consciousness but demonstrate wakefulness and cyclic arousal and intermittently demonstrate self or environmental awareness, such as following of commands, the ability to signal yes/no (regardless of accuracy), intelligible speech, or purposeful behavior .
  - Emergence from the MCS to higher states of consciousness is signaled by the ability to communicate reliably or use objects functionally.
  - Although data are limited, it is believed that the MCS represents a greater likelihood of recovery compared with the VS.
  - As in the VS, lesions or dysfunction associated with the MCS involve the cerebral hemispheres, with possibly greater sparing of corticocortical and cortico-thalamic connective fibers.


Consciousness
Delirium
Minimally conscious state
Coma
Vegetative state
Persistent vegetative state
Brain death


AIRWAY – intubate if GCS ≤ 8
BREATHING – maintain SaO2 > 90 %
CIRCULATION - maintain MAP > 70 mmHg
Draw blood sample for glucose, electrolytes, arterial blood gas, liver and thyroid function tests, complete blood count, toxicology screen
NEUROLOGICAL ASSESSMENT
Hyperventilation, **mannitol** 0.5 – 1.0 g/kg if clinical evidence of increased ICP/herniation [see table 4]
Thiamine (100 mg IV) followed by 25 g glucose (50 mL of 50% solution) if serum glucose < 60 mg/dL
Naloxone if narcotic overdose is suspected (0.4-2.0 mg IV q 3 min or continuous IV infusion 0.8 mg/kg/hr
Flumazenil if benzodiazepine overdose suspected (0.2 mg/min, maximum dose 1 mg IV)
Gastric lavage with **activated charcoal** if drug intoxication suspected
HEAD CT IF STRUCTURAL CAUSE SUSPECTED
DETAILED HISTORY AND SYSTEMIC EXAMINATION
CONSIDER EEG,
LUMBAR PUNCTURE, MRI

- The prognosis of coma is determined in large part by the underlying etiology.
- It is widely believed that toxic/metabolic coma carries a better prognosis than coma of structural origin;
  - direct evidence demonstrating this is limited.
  - The better prognosis of traumatic vs. anoxic coma is supported in a number of studies and two recent systematic reviews.
  - In adults with PVS secondary to TBI who were reevaluated at 1 yr, the proportion with a good recovery was 7%, moderate disability 17%, severe disability 28%, PVS 15%, and dead 33% (96). In patients with nontraumatic PVS (principally HIE), these outcomes were significantly worse (respectively 1%, 3%, 11%, 32%, and 53%) (96).
- Younger age and better general health of TBI patients may account for some of this difference

- Consciousness disorders must be distinguished from brain death,
  - the irreversible loss of all brain and brainstem function,
  - clinically diagnosed by:
    - demonstrating absence of consciousness,
    - lack of motor response to noxious stimulus,
    - the disappearance of:
      - brainstem reflexes
      - respiratory drive.
- Before this determination, pharmacologic, physiologic, and metabolic causes of coma should be excluded.

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Management of physiological hypotension, bradycardia and the 'total spinal'

Physiological hypotension and bradycardia

Treatment	*Timing*	*Duration*
Crystalloid, 500–1000 ml	When block is performed	15–20 min
Crystalloid, 500–1000 ml	As block develops	20–30 min
Colloid, 500–1000 ml	During surgery	Dependent on blood pressure
Ephedrine, 3 mg boluses: total 30 mg	As block develops	Dependent on blood pressure
Atropine, 0.3 mg boluses or Glycopyrrolate, 200 μg boluses	If heart rate < 50 beats/min	Dependent on heart rate

Total spinal anaesthesia

- Intravenous fluids as above but over shorter timescale. Rapid administration of crystalloid, 1000 ml, then colloid, 500 ml. Repeat as necessary to maintain systolic pressure > 100 mm Hg
- Respiratory support (100% oxygen via a face mask progressing to assisted manual ventilation)
- Tracheal intubation if unconsciousness occurs
- Atropine, increments of 0.3 mg, used to treat bradycardia
- Ephedrine, 10 mg increments to a total of 30 mg, to restore systolic pressure > 100 mm Hg
- Intravenous infusion of epinephrine may be necessary to maintain this level of blood pressure until the block begins to wear off
- Slight head-down tilt (10–20°) and slight lateral tilting may be used to maximize venous return
- Maintain supportive measures, until level of spinal block regresses