Ministry of Higher Education and Scientific Research

Middle Euphrates Technical University

College of Health and Medical Technology

Anaesthesia Techniques Department

Teaching package for anaesthesia techniques

.Subject: Practical Intensive Care Unit, 4th stage

Intensive Care Management After Neurovascular Surgery

The immediate postoperative period following neurovascular surgery represents a highly vulnerable phase during which rapid clinical deterioration can occur. This 24-72 hour window demands vigilant monitoring and proactive intervention to prevent secondary brain injury and optimize outcomes.

Core Principles:

1. Brain Protection - Minimize secondary insults

2. Early Detection - Recognize complications before irreversible damage
3. Physiologic Optimization - Maintain cerebral homeostasis

4. Multidisciplinary Coordination - Seamless neurocritical care team approach

Immediate Postoperative Phase (First 24 Hours)

A. Airway and Ventilation Management

Management of the airway, including early extubation when possible, or mechanical ventilation to maintain normocapnia (preventing increased ICP).

Indications for Continued Intubation:

• Glasgow Coma Scale (GCS) ≤ 8

• Significant posterior fossa swelling

• Inadequate airway reflexes (absent gag or cough reflex)

• Anticipated neurological deterioration

• Need for controlled hyperventilation

Ventilation Strategy:

Oxygenation: Maintain PaO_2 > 100 mmHg to ensure adequate cerebral oxygenation.

Ventilation: Maintain normocapnia ($PaCO_2$ 35-40 mmHg) unless hyperventilation is required for acute management of intracranial hypertension.

Positive End-Expiratory Pressure (PEEP): Use ≤10 cm H_2O to avoid impaired cerebral venous return.

Mode: Volume or pressure control ventilation for consistent ventilation during sedation.

Special Considerations:

• Hyperventilation: Reserve for acute herniation syndromes (target $PaCO_2$ 30-35 mmHg)

• Weaning: Only consider when neurological status has been stable for ≥12 hours

B. Hemodynamic Management

Tight blood pressure control is crucial to prevent re-bleeding (low BP) or cerebral edema/hemorrhage (high BP). For subarachnoid hemorrhage this includes managing cerebral vasospasm.

Blood Pressure Targets by Pathology:

After Aneurysm Clipping:

• Systolic blood pressure: 120-140 mmHg

• Mean arterial pressure: 70-90 mmHg

• Special consideration: Avoid hypertension to prevent rebleeding

After Arteriovenous Malformation Resection:

• Systolic blood pressure: 110-130 mmHg

• Mean arterial pressure: 65-85 mmHg

• **Special consideration**: Prevent normal perfusion pressure breakthrough

Acute Ischemic Stroke:

• Systolic blood pressure: 140-180 mmHg

• Mean arterial pressure: 85-105 mmHg

• Special consideration: Permissive hypertension unless thrombolysis administered

Cerebral Vasospasm:

• Systolic blood pressure: 160-200 mmHg

• Mean arterial pressure: 90-110 mmHg

• Special consideration: Induced hypertension to improve collateral flow

Clinical Considerations for Agent Selection:

Post-aneurysm clipping: Preferred labetalol and nicardipine

Cerebral vasospasm: Preferred phenylephrine and norepinephrine.

Concurrent myocardial ischemia: Preferred esmolol, labetalol, and nitrates.

Renal impairment: Preferred nicardipine and fenoldopam.

Monitoring Parameters During Therapy:

1. Continuous arterial blood pressure, HR, and cardiac rhythm monitoring

2. Neurological examination every 15-30 minutes initially

3. Serum electrolytes and renal function every 4-6 hours

4. For nitroprusside: Acid-base status and thiocyanate levels if prolonged use

C. Neuromonitoring

In addition to invasive arterial pressure monitoring, advanced monitoring may include intracranial pressure (ICP) sensors, transcranial Doppler (for vasospasm), and continuous EEG to detect seizures

Intracranial Pressure Management Algorithm:

For intracranial pressure > 20-25 mmHg:

❖ Step 1: Head elevation to 30°, maintain midline head position

❖ Step 2: Ensure adequate sedation and analgesia

❖ Step 3: Osmotherapy: o Mannitol 0.25-1 g/kg intravenous bolus OR o Hypertonic saline (3%) 250 mL intravenous bolus

❖ Step 4: Cerebrospinal fluid drainage (if ventricular catheter present)

❖ Step 5: Moderate hyperventilation (target $PaCO_2$ 30-35 mmHg)

❖ Step 6: Barbiturate coma (pentobarbital) for refractory cases

C. **Fluid & Electrolyte Balance:**

Maintaining euvolemia using isotonic crystalloids is standard. Strict monitoring for sodium imbalances (such as Diabetes Insipidus or Cerebral Salt Wasting) is essential, especially after pituitary or hypothalamic surgeries.

D.Temperature & Metabolic Control: Avoiding hyperthermia (which increases brain metabolic demand) and managing blood glucose levels to prevent secondary brain injury.

E.Pain & Sedation: Multimodal analgesia is used to control pain, which, if unmanaged, can cause hypertension and increased ICP. Sedation is often used to facilitate ventilation and reduce metabolic rate.

F.Thromboprophylaxis: Mechanical methods (e.g., pneumatic compression devices) are started immediately. Pharmacological prophylaxis (e.g., heparin) is typically initiated 24-48 hours postoperatively, following a CT scan confirming stability.

G.Positioning: The head of the bed is usually elevated to 30° to promote venous drainage and reduce ICP.

Management of common complications

Vasospasm/Delayed Cerebral Ischemia: Particularly in SAH, treated with nimodipine.

Postoperative Hemorrhage: Requires immediate identification and, in some cases, surgical evacuation

Hydrocephalus/Increased ICP: Managed with cerebrospinal fluid (CSF) drainage (EVD) or pharmacological agents (mannitol, (hypertonic saline

Seizures: Prophylactic anticonvulsants may be used.

Pulmonary Complications:

Ventilator-Associated Pneumonia Prevention:

• Head of bed elevation ≥30°

• Oral care every 4 hours with chlorhexidine

• Daily sedation holidays

• Peptic ulcer prophylaxis

Transition Planning and De-Escalation of Care Intensive Care Unit
Discharge Criteria:

• Neurologically stable for ≥24 hours

• Intracranial pressure normal without therapy for ≥24 hours

• No vasopressor requirements

• Airway protective reflexes intact

• Seizure-free (or well-controlled on medications)

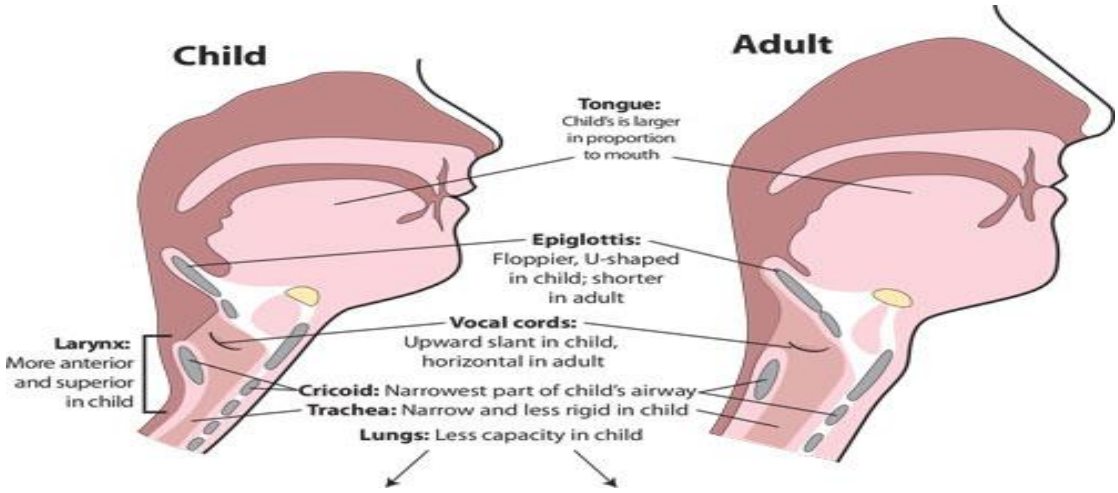
• No active intracranial complication on imaging

Airway Management Of Pediatric

How are children different?

•Narrower airways- they are more easily obstructed by oedema, blood foreign bodies and external compression. Even mild narrowing can lead to rapid increases in airway resistance and subsequent work of breathing.

•Larger tongue and adenoids - increased risk of obstruction, along with increased difficulty of laryngoscopy and visualising the vocal cords.



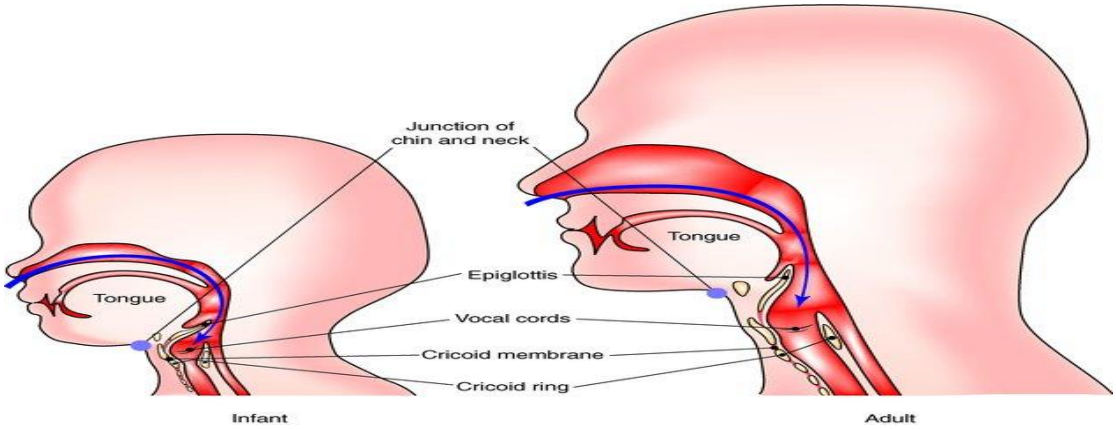
•The soft structures of the pediatric upper airway are also more prone to injury from multiple attempts at intubation

•Higher anterior larynx.

•Shorter trachea. Chest x-ray is required to confirm ETT position

•Increased metabolic requirements and reduced functional residual capacity lead to rapid desaturation

•Longer floppy epiglottis so a straight blade laryngoscope may be preferred for intubation.



Mechanisms of airway obstruction

Direct trauma to the airway or surrounding structures. This may include: Maxillo-facial / laryngeal / tracheal injury or compression due to anterior neck haematoma

•Burn-associated oedema of mouth, pharynx, larynx.

•Contamination of the airway due to material - for example with vomitus / blood / teeth or other foreign bodies

•Loss of pharygeal tone - due to head injury or intoxication with drugs/alcohol

Signs of airway obstruction

Respiratory distress which may be characterised by:

•An increase in respiratory rate

•Paradoxical movement of the chest and abdomen

•Use of accessory muscles with sternal, intercostal and subcostal recession

•Intrathoracic obstruction (of trachea or bronchi) may present with wheeze or prolonged expiration

•Extrathoracic obstruction may present with

•Stridor / hoarseness or voice change (laryngeal or upper tracheal injury),

•gurgling (contamination of the oro-pharynx)

•Snoring (loss of pharyngeal tone due to reduced level of consciousness leading to occlusion of airway).

Look for!

•Cyanosis / Low SpO_2

•Visible swelling of the tongue, pharynx or neck

•External signs of injury to face, mouth, mandible or neck

•Nonspecific effects on other organ systems - tachycardia / decreased conscious state / restlessness

Airway Assessment

You should examining the anterior neck

•**Tracheal deviation** - may be caused by a tension pneumothorax or a massive haemothorax (with tension)

•**Wounds** - blunt or penetrating wounds to the neck may directly injure the airway, or lead to swelling that will progressively obstruct it

•**Emphysema** (subcutaneous) - indicates a pneumothorax

•**Venous distension** - associated with obstructive shock secondary to a tension pneumothorax or cardiac tamponade

In trauma, always use **jaw thrust**. (Not head tilt or chin lift) (Place fingers behind the angles of the mandible and push anteriorly (towards the tip of the nose

If an obstruction persists

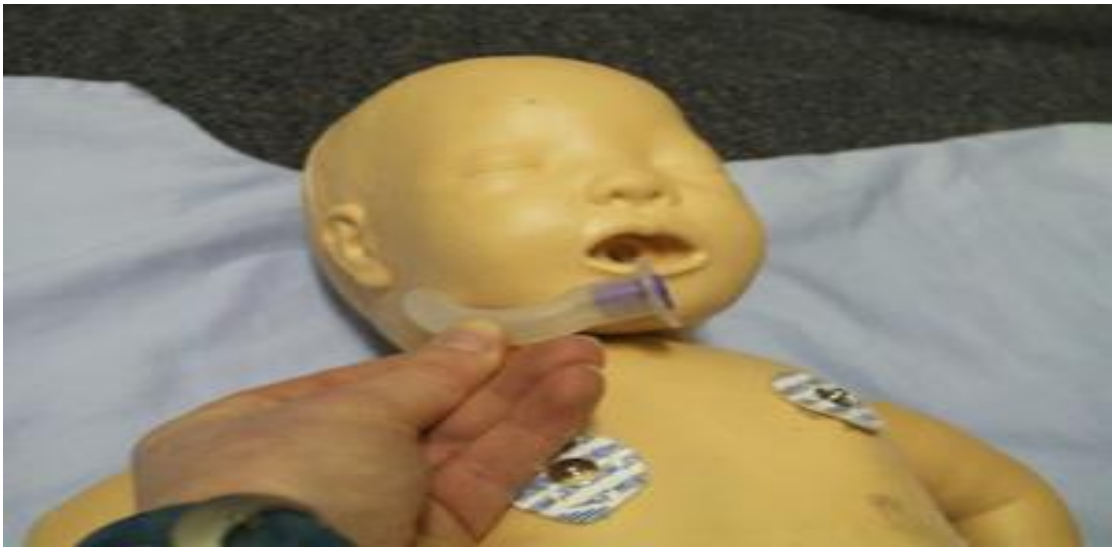
•Optimise the head position, and reopen the airway using a jaw thrust.

•Rapidly examine the mouth with a laryngoscope.

•Remove any visible foreign body using Magill forceps or Yankauer sucker.

•If the airway is adequate during laryngoscopy but inadequate at other times, insert an oropharyngeal airway.

Choosing the correct size of Oropharyngeal airway for child is important, the right size reaches from the central incisors to the angle of the child's mandible.



Oral Airway

•Child more than 8 years: As for an adult: concave side up; pass to the back of the hard palate, then rotate 180^{o} to concave side down

•Only in unconscious children use a tongue blade to facilitate insertion<8y

•DO NOT INSERT AND ROTATE 180 degrees—this maneuver can tear the soft palate and cause bleeding.

❖If the airway remains inadequate: consider tracheal intubation if the facilities are available to do this safely and quickly .

Preparation for Endotracheal intubation

Pre-Oxygenate:

•Pre-oxygenate with T-piece and high flow oxygen

•Deliver positive airway pressure breaths and positive end-expiratory pressure

•Aspirate air from the stomach via a small bore gastric tube

•**Laryngoscope:** Have 2 available; check they are working

•**Suction:** Check it is working

•Verify the suction device is a Yankauer, Check it is next to the child's head .

Drugs: Drawn up and labeled:

Ketamine 1-2mg/kg depending on the degree of physiologic un-wellness (**lower** dose in acute circulatory failure)

Rocuronium

1.2-1.6mg/kg depending on the degree of physiologic unwellness ,Saline flush 10 ml IV cannula + 3-way

Endotracheal intubation

Tube diameter should be the size of the child’s 5th finger

New born 3.5mm, 1 year 4.0mm, 2 years 4.5mm,

>2 years 4.5 + age/4.

Oral: Always use oral, never nasal, intubation in a child with a head injury (because of the risk of meningitis, or of entering the cranial cavity if an undiagnosed fracture of the skull base is present).

Preparation for Endotracheal intubation

Laryngoscopy:

Hold laryngoscope in your left hand. And Be gentle.

Don't lever on the teeth. And don't jam the lip between blade and teeth.

Up to 1year: Straight blade

It is recommended that only an experienced clinician should attempt to intubate a child - unless the procedure is immediately required to save a life.

NEUROMUSCULAR WEAKNESS SYNDROMES

Normal Physiology at the Neuromuscular Junction

The following physiological events occur during neuromuscular transmission:

1. A nerve action potential propagates down the axon and depolarizes the presynaptic nerve terminal.

2. Voltage-gated calcium channels open in response to depolarization, allowing an influx of calcium ions into the nerve terminal.

3. Synaptic vesicles fuse with the presynaptic membrane and release acetylcholine (ACh) into the synaptic cleft.

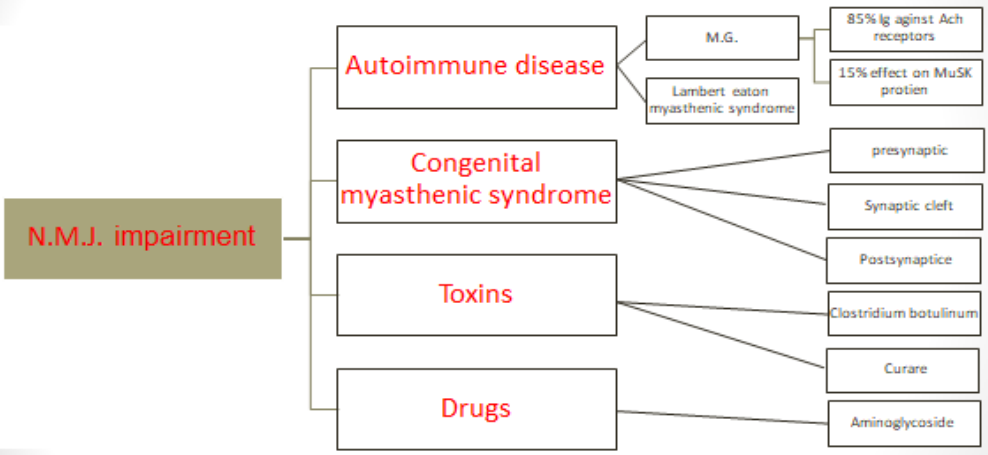
4. ACh molecules bind to acetylcholine receptors (AChR) on the postsynaptic membrane, inducing a conformational change that opens the associated ion channel.

5. Membrane conductance to Na^+ increases (Na^+ influx, K^+ efflux), depolarizing the endplate region and generating an endplate potential (EPP).

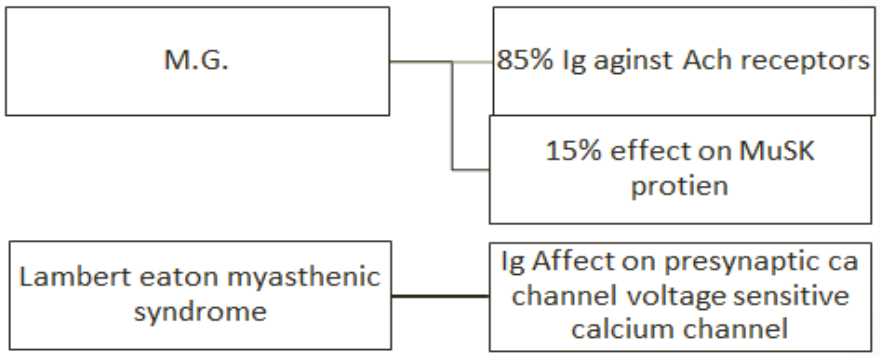
6. If the EPP is sufficient to depolarize the adjacent muscle membrane to threshold, an action potential is generated in the muscle fiber. If insufficient, no muscle action potential occurs.

❖ The neuromuscular weakness syndromes that deserve attention include myasthenia gravis, Guillain-Barré syndrome, and critical illness neuromyopathy.

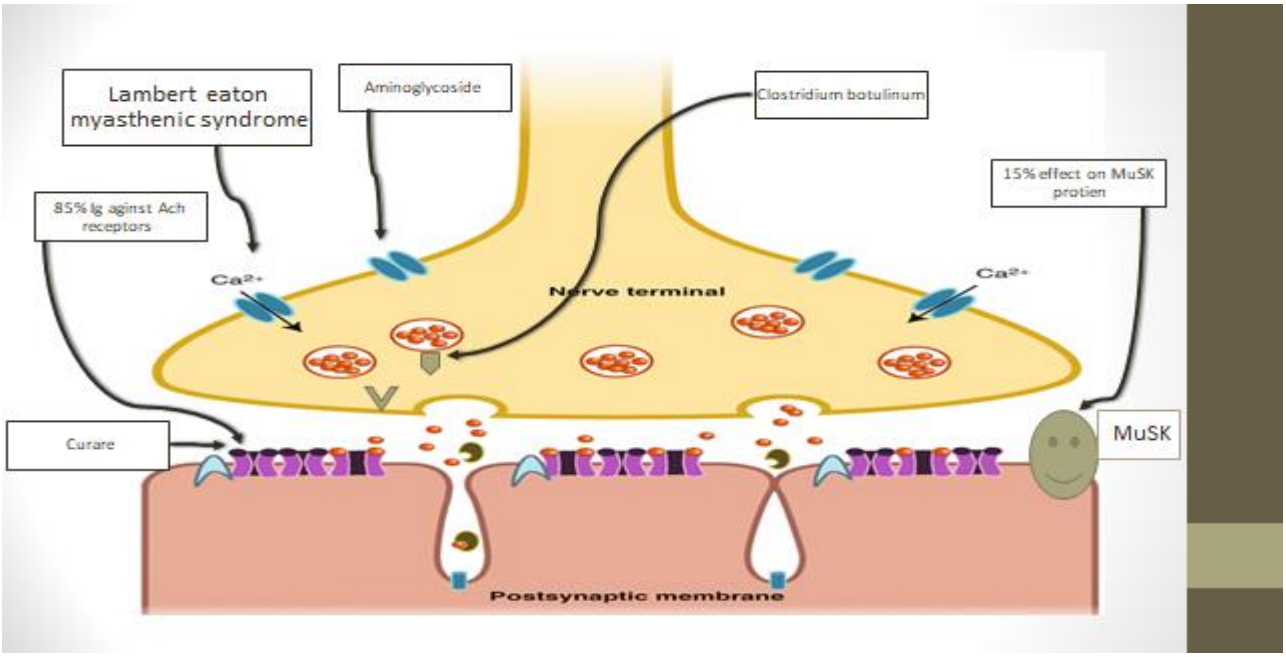
neuromuscular junction impairment



Autoimmune disease



❖ Muscle-specific kinase (MuSK) is a single-pass transmembrane protein that has a critical role in signaling between motor neurons and skeletal muscle



A. Myasthenia Gravis Myasthenia gravis (MG) is an autoimmune disease caused by antibody mediated destruction of acetylcholine receptors at neuromuscular junctions.

Etiology

MG is a chronic autoimmune process. No specific cause has been found for MG. However, the current thought is that a virus or bacteria may initiate the autoimmune process. Genetics may also play a role. Disorders of the thymus gland are often associated with MG. Thymomas or tumors on the thymus gland can account for the malfunction of the immune system that initiates the autoimmune process.

Clinical Features

• Weakness increases with activity and improves with rest.

• Weakness typically begins in the eyelids and eye muscles, followed by limb weakness in 85% of cases.

• Progression can involve the chest wall and diaphragm. A rapid decline to respiratory failure (myasthenic crisis) occurs in 15–20% of patients.

• Weakness is purely motor; deep tendon reflexes remain normal.

Diagnosis

• Suggested by weakness that worsens with repeated use of muscles (especially in the eyes).

• Confirmed by: Improvement in strength after administration of edrophonium (a test drug).

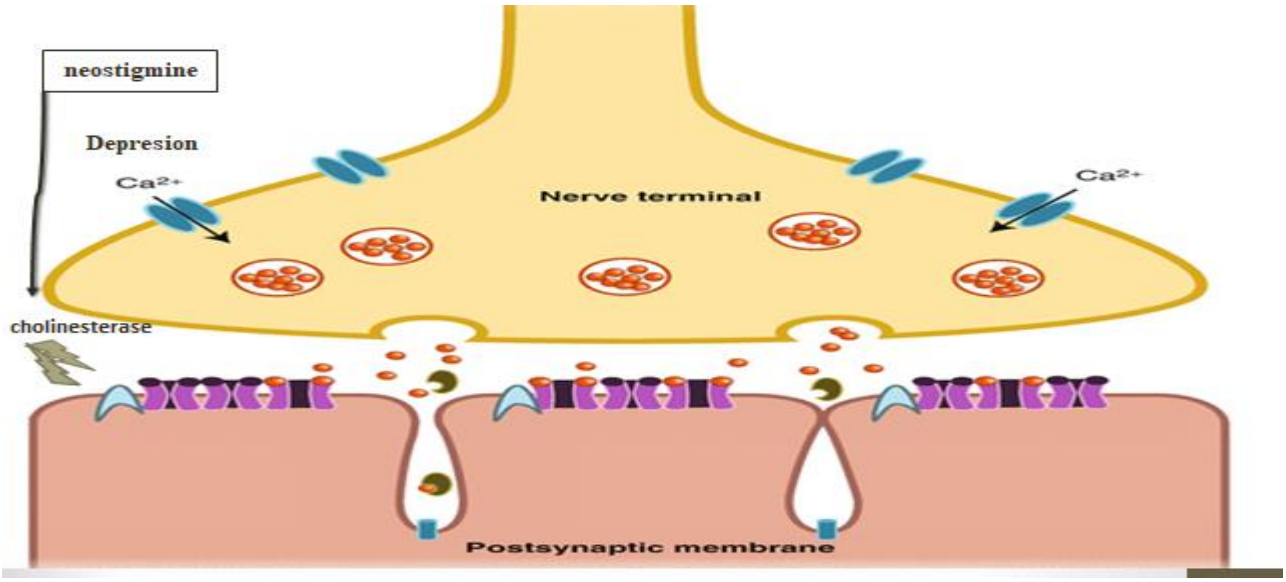
•Detection of acetylcholine receptor antibodies in the blood (present in 85% of patients).

Treatment

• First-line: Acetylcholinesterase inhibitors (e.g., pyridostigmine), started orally and adjusted as needed. Intravenous forms are used for crisis.

• Immunotherapy: Added if needed, using drugs like prednisone, azathioprine, or cyclosporine.

• Thymectomy: Often recommended for patients under 60 to reduce the need for long-term immunosuppression and advanced Cases (requiring mechanical ventilation)

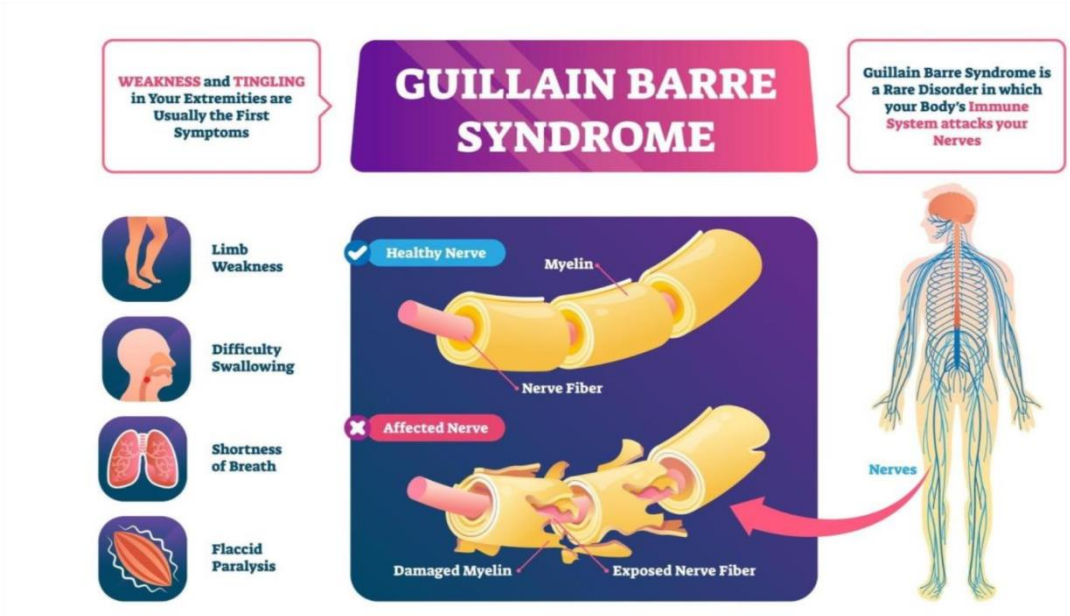


Plasmapheresis: Removes harmful antibodies from the blood; acts quickly.

• Intravenous Immunoglobulin (IVIG): Neutralizes harmful antibodies; often preferred due to easier administration.

B. Guillain-Barré Syndrome (GBS)

GBS is a subacute inflammatory demyelinating polyneuropathy, often occurring 1–3 weeks after an acute infection. An immune cause is suspected.



Clinical Features

• Presents with tingling in the hands/feet and symmetric limb weakness that develops over days to weeks.

• Progresses to respiratory failure in 25% of cases.

• Autonomic nervous system instability can occur in advanced cases.

• Most patients (80%) recover spontaneously, but residual weakness is common.

Diagnosis

Based on the clinical picture, nerve conduction studies (showing slowed signals), and cerebrospinal fluid analysis (showing elevated protein).

Treatment

Primarily supportive. For advanced cases with respiratory failure, plasmapheresis or IV immunoglobulin are equally effective for short-term improvement. IVIG is often preferred for ease of use.

ACUTE STROKE

I. Definitions and Classification

A. Stroke

An acute brain disorder of vascular origin accompanied by neurological dysfunction that persists for longer than 24 hours.

B. Transient Ischemic Attack (TIA)

An acute episode of focal neurological dysfunction lasting less than 24 hours, with no apparent cause other than vascular origin. Critically, up to one-third of TIAs are associated with cerebral infarction on imaging, highlighting the urgency of evaluation.

C. Stroke Classifications

Type	Prevalence	Subtypes & Notes
Ischemic	87%	- **Thrombotic (80%)**: Arising from local thrombosis. - **Embolic (20%)**: Often from cardiac sources (e.g., AF, MI) or paradoxical embolism via a Patent Foramen Ovale (PFO).
Hemorrhagic	13%	- **Intracerebral Hemorrhage (97%)** - **Subarachnoid Hemorrhage (3%)** *Note: Epidural/Subdural hematomas are not classified as strokes.*

II.Initial Evaluation: "Time is Brain"

Each minute of cerebral infarction destroys **1.9 million neurons**. the evaluation must be swift and systematic.

A.Bedside Neurological Evaluation

The clinical presentation localizes the area of brain injury.

Mental Status: Most unilateral infarctions do not cause coma. Coma with focal deficits suggests intracerebral hemorrhage, brainstem infarction, or seizures.

Aphasia: Indicates injury to the dominant (usually left) hemisphere.

Stroke Mimics: Up to 30% of suspected strokes are mimics. Common ones include nonconvulsive seizures, sepsis, and metabolic encephalopathy.

B.NIH Stroke Scale (NIHSS)

A standardized 11-item scale (score 0-41) to quantify stroke severity. A score ≥22 generally indicates a poor prognosis.

C. Echocardiography

Indicated to identify an embolic source (e.g., AF, mural thrombus, endocarditis) or a Patent Foramen Ovale (PFO) in the context of a cryptogenic stroke.

III. Reperfusion Therapy: Thrombolysis and Thrombectomy

A. Thrombolytic Therapy with tPA

The goal is to administer tPA within 4.5 hours of symptom onset. Time of onset must be accurately identified.

B. Mechanical Thrombectomy

Can be performed up to 8 hours (sometimes longer) from symptom onset.

Does not preclude concurrent tPA therapy; they are often used together

IV. Supportive Care and Protective Measures

A. Blood Pressure Management

General Principle: Do not aggressively lower BP in the first 24 hours unless for tPA eligibility or if SBP >220 / DBP >120, as this may extend the ischemic penumbra.

B. Oxygen Therapy

•**Do not** administer supplemental oxygen if O_2 saturation is ≥94%.
•Unnecessary oxygen can cause cerebral vasoconstriction and increase oxidative stress.

C. Antipyretic Therapy

• Aggressively treat fever with antipyretics and search for an underlying infection.

D. Glycemic Control

•Hyperglycemia aggravates ischemic injury and maintain plasma glucose between 140–180 mg/dL. Avoid hypoglycemia, which is also detrimental.

V. Mechanical Ventilation in Acute Stroke (Anesthesia Focus)

Mechanical ventilation in stroke patients **aims**

protect the brain

maintain adequate cerebral perfusion,

avoid secondary injury.

A. Indications for Intubation in Stroke

Intubate if any of the following are present:

•Decreased consciousness (GCS ≤8 or inability to protect airway)

• Loss of gag or cough reflex

• Severe agitation interfering with imaging or treatment

• Respiratory failure (hypoxia or hypercapnia)

• Impending herniation signs

• Need for general anesthesia during mechanical thrombectomy

B. Ventilation Goals

1. Oxygenation

Maintain SpO_2 94–98%

• Avoid unnecessary high FiO_2 (hyperoxia → cerebral vasoconstriction)

2. CO_2 Management (VERY IMPORTANT)

CO_2 strongly influences cerebral blood flow (CBF)

Target: $PaCO_2$ 35–40 mmHg

CO_2 LEVEL	EFFECT ON BRAIN	CLINICAL USE
HYPOCAPNIA ($PACO_2 < 35$)	Cerebral vasoconstriction → ↓CBF	**Avoid** in ischemic stroke (worsens infarct)
NORMOCAPNIA ($PACO_2$ 35–40)	Stable CBF	**Primary target** in most strokes
MILD HYPERVENTILATION ($PACO_2$ 30–35)	Temporary ↓ICP	Use **only in acute herniation**, avoid prolonged use

C. Ventilation Strategy by Stroke Type

1. Ischemic Stroke

• Goal: maximize cerebral perfusion and prevent secondary ischemia by choosing the correct setting of ventilators ,mode (VC or PC) and maintain normocapnia (PaCO2 35-40) also avoid high peep(may reduce cerebral perfusion),with SpO2≥94%.

• Avoid hypotension at induction—use etomidate or ketamine cautiously

2. Hemorrhagic Stroke (ICH or SAH)

Goals: control ICP and prevent rebleeding

•Slightly higher PEEP acceptable (8–10 cmH_2O) if needed for oxygenation and maintain $PaCO_2$ 35–38 mmHg with avoid hypercapnia → ↑ICP

• Avoid high airway pressures and control BP to prevent rebleeding

D. Sedation Strategy (ICU/OR)

Preferred Agents

• Propofol: fast, titratable, decreases ICP

• Dexmedetomidine: good for neurological exams

• Remifentanil: short-acting, useful in procedures

Avoid

•Benzodiazepines (prolong sedation, mask neuro exams)

• Ketamine is controversial; modern data suggests it’s safe and does not increase ICP, but use cautiously in ICH.

F. Extubation Criteria in Stroke Patients

•Conscious, able to follow commands

• Strong cough and gag reflex

• No significant bulbar dysfunction

• Adequate gas exchange

• No large cerebral edema or mass effect

• Hemodynamically stable

BRAIN DEATH

The irreversible cessation of either circulatory and respiratory functions or all functions of the entire brain, including the brainstem.

Diagnostic Criteria and Prerequisites

The diagnosis of brain death is clinical, based on the demonstration of irreversible coma, the absence of brainstem reflexes, and apnea.

Coma: The patient must exhibit irreversible, unresponsive coma with a known cause sufficient to explain the loss of brain function.

Absence of Brainstem Reflexes:

This includes no pupillary, corneal, oculocephalic (doll's eyes), oculovestibular (caloric), gag, or cough reflexes.

• No pupillary light reaction

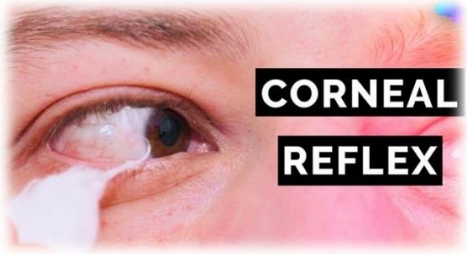
• No corneal reflex

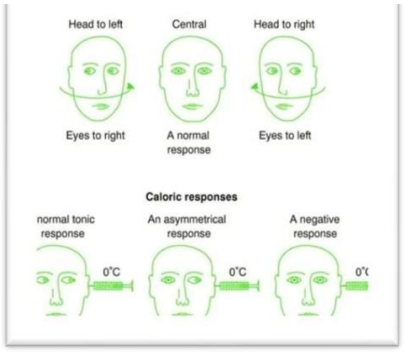
• No oculocephalic reflex (doll’s eyes)

• No oculovestibular reflex (caloric testing)

• No gag reflex

• No cough reflex on tracheal suction





Apnea: The absence of spontaneous respiratory effort during a formal CO2 challenge (apnea test).

The clinical examination must be performed under the following prerequisite conditions to exclude confounding factors:

•**Hemodynamic Stability:** Systolic blood pressure ≥100 mm Hg. Normothermia: Core body temperature >36°C.

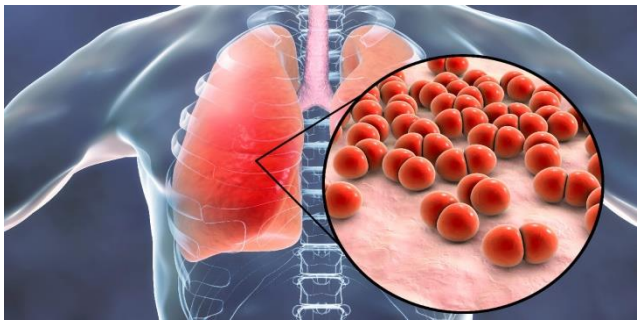
•**Absence of CNS Depressants:** No evidence of sedation or the presence of neuromuscular blocking agents

•**Metabolic Stability:** Euglycemia and normal thyroid function

Note: A single neurologic examination is sufficient for diagnosis in most U.S. states, though some require two separate examinations.

VENTILATOR-ASSOCIATED PNEUMONIA

Pneumonia is the most common nosocomial infection in ICU patients, and more than 90% of these pneumonias occur during mechanical ventilation. However, the prevalence of VAP is overstated, because post-mortem studies have shown that over half of the cases of VAP are false-positive diagnoses



Causes & Risk Factors

Microbial Invasion: Bacteria from the mouth or environment bypass the body's defenses due to the endotracheal tube ,the most responsible pathogens in VAP are gram negative aerobic bacilli and Staphylococcus aureus

Contaminated Equipment: Ventilator circuits, humidifiers, or contaminated respiratory care practices.

Compromised Airway: Intubation allows oral/gastric secretions to enter lower airways.

Timing: Late-onset VAP (after 4 days) often involves antibiotic-resistant bacteria

Assessment and Diagnostic Methods

• Primarily history, physical examination

• Chest x-rays, blood and sputum cultures, Gram stain

A. Diagnostic Accuracy

The traditional clinical criteria for the diagnosis of VAP include:
(a) fever or hypothermia

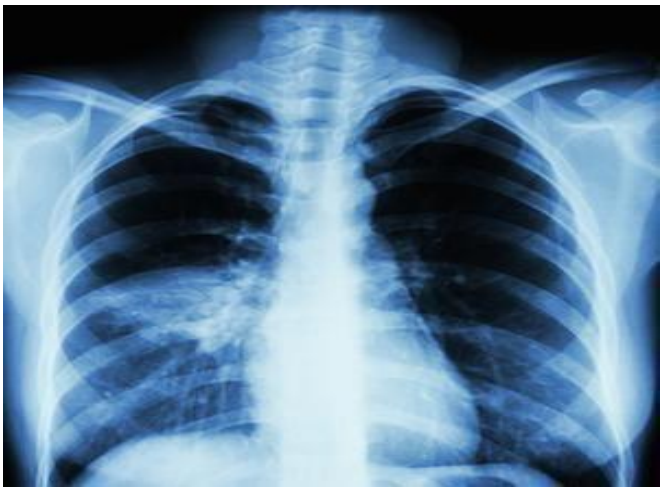
(b) leukocytosis or leukopenia

(c) an increase in volume of respiratory secretions or a change in character of the secretions

(d) a new or progressive infiltrate on the chest x-ray

B. Chest Radiography

Portable chest x-rays perform poorly in the diagnosis of ventilator-associated pneumonia, with an overall accuracy of only about 49%. This is largely because they have a low sensitivity of approximately 38%, meaning they often fail to detect actual pulmonary infiltrates even when pneumonia is present.



Chest radiograph showing ventilator-associated aspiration pneumonia

C. Lung Ultrasound

Ultrasound examination of the lungs is a more reliable method for detecting pulmonary consolidation than portable chest x-rays

Management of VAP

•Antibiotics are prescribed on the basis of Gram stain results and antibiotic guidelines

•Supportive treatment includes hydration, antipyretics, antitussives medications, antihistamines, or nasal decongestants.

•Bed rest is recommended until infection shows signs of clearing.
•Oxygen therapy is given for hypoxemia.

•Respiratory support includes high inspiratory oxygen concentrations, endotracheal intubation, and mechanical ventilation.

•Treatment of atelectasis, pleural effusion, shock, respiratory failure

•For groups at high risk for CAP, pneumococcal vaccination is advised

Mechanical ventilation management in VAP

Mechanical ventilation management plays a crucial role in both the pathophysiology and the supportive care of patients with ventilator-associated pneumonia (VAP).

A. Lung-Protective Ventilation Strategy

1. Low Tidal Volume Ventilation: Use tidal volumes of 6–8 mL/kg of predicted body weight

2. Plateau Pressure Limitation: Maintain an end-inspiratory plateau pressure (Pplat) ≤30 cm H_2O

3. Positive End-Expiratory Pressure (PEEP): Apply adequate PEEP to prevent alveolar collapse at end-expiration

B. Management of Airway Secretions and Hygiene

1. Endotracheal Suctioning: Perform suctioning only as needed, based on audible or visible secretions, increased peak airway pressures, or desaturation

2. Bronchial Hygiene Adjuncts: Chest Physiotherapy and mechanical Insufflation-Exsufflation may be beneficial in select patients with lobar consolidation and difficulty mobilizing secretions.

C. Ventilator Weaning and Spontaneous Breathing Trials (SBTs)

1.Daily Assessment for Readiness to Wean

• Criteria for SBT Consideration:

1. Evidence of infection improvement

2. Adequate gas exchange (e.g., PaO_2/FiO_2 >150-200, PEEP ≤8 cm H_2O, FiO_2 ≤0.5).

3. Hemodynamic stability without significant vasopressors.

4. Ability to initiate spontaneous breaths.

2. Conducting the SBT

• **Method**: A 30–120-minute trial on a low level of pressure support (e.g., 5–8 cm H_2O) with PEEP (5 cm H_2O) or on a T-piece.

Failure Criteria: Development of tachypnea (RR >35), hypoxia (SpO_2<90% ,tachycardia/bradycardia, hypertension/hypotension, diaphoresis, or signs of increased work of breathing and agitation.

D. Sedation and Mobility

Sedation Strategy: Use a protocol of light sedation or a "no sedation" strategy where feasible, deep sedation increases aspiration risk, promotes atelectasis, and delays weaning.

• **Early Mobility**: Implement early, progressive mobilization as soon as the patient is hemodynamically and respiratory stable. This improves secretion clearance, reduces atelectasis, and decreases ventilator days.

Diabetic Ketoacidosis (DKA)

Diabetic ketoacidosis (DKA) is a metabolic emergency caused by absolute or relative deficiency of insulin. It is characterized by hyperglycemia (>300mg/dL), metabolic acidosis (pH <7.30 and low carbonate level<15mEq/L

Clinical Manifestations

•Polyuria and polydipsia (increased thirst).

•Orthostatic hypotension in patients with volume depletion.

•Gastrointestinal symptoms, such as anorexia, nausea/vomiting, and abdominal pain.

•Acetone breath (fruity odor).

•Kussmaul respirations: hyperventilation with very deep, but not labored, respirations.

•Mental status varies widely from patient to patient (alert to lethargic or comatose).

Complications of Diabetic Ketoacidosis

• Hypokalemia

• Hypophosphatemia

• Metabolic acidosis

• Hypoglycemia

• Cerebral edema

• Thromboembolism (due to dehydration and sluggish perfusion)

Assessment and Diagnostic Findings

•Blood glucose level: 300 to 800 mg/dL (may be lower or higher).

•Low serum bicarbonate level: 0 to 15 mEq/L.

•Low pH: 6.8 to 7.3 and low PaCO2: 10 to 30 mm Hg.

•Sodium and potassium levels may be low, normal, or high depending on amount of water loss (dehydration).

•Elevated creatinine, blood urea nitrogen (BUN), and hematocrit values may be seen with dehydration.

• ECG: Assess for cardiac events or electrolyte abnormalities (hypo /hyperkalemia).

Management of DKA

In addition to treating hyperglycemia, management of DKA is aimed at correcting dehydration, electrolyte loss, and acidosis ,insulin treatment & fluid replacement are the mainstay of treatment.

❖**Airway management** is the primary concern in any patient with a significantly lowered level of consciousness. Breathing and circulatory stability should also be established before proceeding to specific management

Rehydration

•Patients may need as much as 6 to 10 L of IV fluid (0.9% normal saline is administered at a high rate of 0.5 to 1 L/h for 2 to 3 hours) to replace fluid loss caused by polyuria, hyper ventilation, diarrhea, and vomiting.

•Hypotonic (0.45%) NS solution may be used for hypertension or hypernatremia and for those at risk for heart failure. This is the fluid of choice (200 to 500 mL/h for several additional hours) after the first few hours, provided that blood pressure is stable and sodium level is not low.

•When the blood glucose level reaches 300 mg/dL (16.6 mmol/L) or less, the IV solution may be changed to dextrose 5% in water (D5W) to prevent decline in the blood glucose level.

Plasma expanders may be used to correct severe hypotension that does not respond to IV fluid treatment.

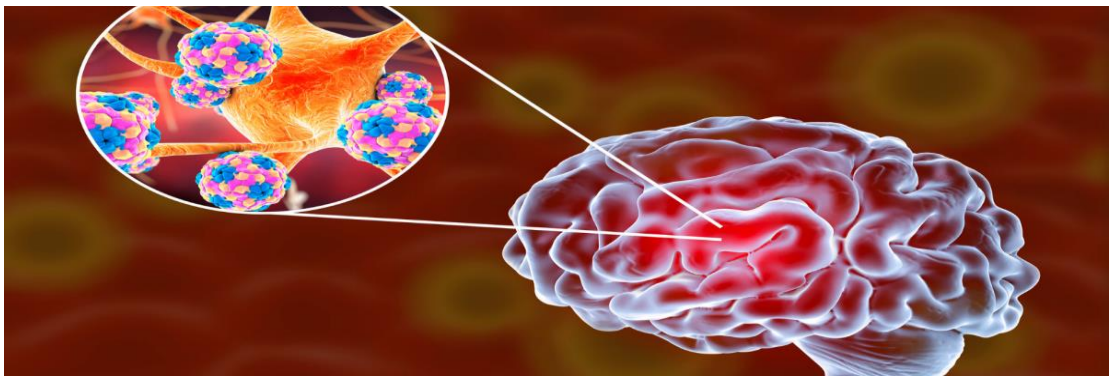
Restoring Electrolytes

Potassium is the main electrolyte of concern in treating DKA. Cautious but timely replacement of potassium is vital for avoiding severe cardiac dysrhythmias that occur with hypokalemia

❖ The **Insulin infusion** should be continued until the acidosis resolves, i.e., until the pH and anion gap are normal, even if the blood glucose levels are normal

Central Nervous System Infections

CNS infections involving the brain, spinal cord, meninges, or surrounding structures with the most significant entities being meningitis, encephalitis, brain abscess, and other parameningeal foci of infection.



•These disease processes have distinct pathophysiological features, their clinical presentations often exist on a spectrum with considerable overlap.

•Classic meningitis manifests with headache, neck stiffness, and fever, whereas encephalitis is characterized by disturbances in cerebral function, and brain abscesses typically present as space-occupying lesions.

Acute Bacterial Meningitis

Acute bacterial meningitis the most definitive emergency in infectious diseases. Delayed or inadequate treatment substantially increases the risk of death or permanent neurological impairment

Etiology of Bacterial Meningitis

•**Bacterial meningitis** is predominantly caused by five pathogens. Streptococcus pneumoniae is the most common cause in adults.

•**Risk factors** include CSF leaks, alcoholism, asplenia, and cochlear implants.

•Neisseria meningitidis, though its incidence has declined due to vaccination, still causes over 10% of cases and it is notable for epidemic potential.

•Listeria monocytogenes ,Haemophilus influenzae type B.

•Group B Streptococcus (Streptococcus agalactiae) remains the leading cause of neonatal meningitis and is increasingly recognized in adults.

•Other important pathogens include gram-negative bacilli, Staphylococcus aureus and various skin flora and coagulase-negative staphylococci.

Diagnosis of Acute Bacterial Meningitis

•**Classic symptoms** include fever, headache, nuchal rigidity, and altered mental status.

•In the ICU, red flags include unexplained altered consciousness, new-onset seizures, or fever in a neurosurgical or immunocompromised patient.

•**High-risk groups** include the elderly, alcoholics, asplenic individuals, those with complement deficiency, and patients with CSF shunts or cochlear implants.

Diagnostic Findings

•**Computed tomography** (CT) scan or magnetic resonance imaging (MRI) scan to detect a shift in brain contents (which may lead to herniation) prior to a lumbar puncture.

•**Key diagnostic tests**: bacterial culture and Gram staining of CSF and blood

Management of acute Bacterial Meningitis

•Immediate actions in the ICU follow a structured approach. First, securing the airway, breathing, and circulation is paramount, as patients can deteriorate rapidly; anesthesiologists must be prepared for rapid sequence intubation if the GCS score drops.

•Second, two sets of blood cultures should be drawn prior to antibiotic administration to aid pathogen identification.

•Third, empiric antibiotic therapy—along with dexamethasone for suspected pneumococcal meningitis—must be initiated without delay.

•Common empiric regimens include ceftriaxone plus vancomycin, with the addition of ampicillin for patients over 50 or those who are immunocompromised to cover Listeria.

•ICU practitioners must anticipate and manage several complications: increased intracranial pressure managed with head elevation, osmotherapy, and possibly CSF drainage

•Seizures, requiring readily available anticonvulsants

•Hyponatremia from SIADH or cerebral salt wasting, necessitating careful sodium monitoring

Septic shock, which may require vasopressor support.

•Airway compromise, mandating intubation for patients with a GCS below 8.

ENCEPHALITIS

Encephalitis is an inflammatory process of the brain parenchyma that causes neurological dysfunction. Its presentation overlaps significantly with meningitis, leading to the term meningoencephalitis when both are present.

Etiologies are diverse, including infectious and noninfectious causes. Viruses are responsible for most infectious cases, but the cause remains unidentified in over 50% of patients.

Even without a specific agent, distinguishing infectious from noninfectious causes provides important prognostic and epidemiological insight.

Diagnosis and management of encephalitis

I. Diagnostic Approach

The goal is early identification of treatable causes, focusing on (1) HSE, (2) nonviral pathogens, and (3) autoimmune etiologies. Over 50% of cases remain undiagnosed despite investigation.

II. Clinical manifestations

The classic presentation is encephalopathy with diffuse or focal neurologic symptoms, including the following:

•Behavioral and personality changes, with decreased level of consciousness

•Neck pain, stiffnes ,Photophobia , Lethargy

•Generalized or focal seizures (60% of children with CE)

•Acute confusion or amnestic states and flaccid paralysis.

III. Diagnostic Workup – ICU Protocol

CSF analysis is essential ,measure opening pressure

•Typical CSF shows <1000 cells/μL(lymphocytic), mildly elevated protein and normal glucose. RBCs suggest hemorrhagic encephalitis (e.g., HSE); eosinophils suggest parasites/fungi.

IV. Imaging & Neurophysiology

MRI is recommended for all patients; it may show temporal lobe involvement (suggestive of HSE) and rule out abscess, tumor, or vasculitis. Repeat in 3–7 days if suspicion remains high.

•EEG can provide early clues; 80% of HSE cases show temporal foci. Normal EEG correlates with better prognosis.

•Brain biopsy is a last resort for progressive deterioration with unknown etiology.

V. Therapy

•Empiric: Start acyclovir (10 mg/kg IV q8h) immediately in all suspected cases while awaiting HSV PCR.

Specific:

• HSE: Acyclovir IV for 14–21 days; repeat CSF PCR before stopping if symptoms persist.

• VZV: Acyclovir.

• CMV/HHV-6: Ganciclovir ± foscarnet (in immunocompromised).

• Rickettsial/Ehrlichial: Empiric doxycycline in summer months.

• Autoimmune: Treat underlying cancer if paraneoplastic; use immunotherapy (plasma exchange, IVIG, immunosuppressants) for antibody-mediated forms.

Supportive Care in ICU

• Airway: Intubate if GCS<8 or respiratory failure

•Seizures: Have anticonvulsants available; monitor for status epilepticus.

•Cerebral edema: Elevate head of bed; use osmotherapy if herniation suspected; maintain euvolemia.

• High-risk groups: Patients >65 have worse outcomes; immunocompromised hosts require aggressive workup and management.

Toxicological Emergencies in Critical Care

Toxicological emergency = life-threatening condition due to exposure, ingestion, inhalation, or injection of a toxic substance requiring immediate critical care.

Initial ICU Approach (ALWAYS FIRST)

Primary Survey – Immediate Life-Saving Priorities (ABCDE)+F+TOX

- Airway
- Breathing
- Circulation
- Disability (Neurologic Assessment
- Exposure
- Full documentation & Further management
- TOX: history, containers, timing, dose **(**How and why did exposure happen, What substance was taken, When did the exposure occur)

Signs and Symptoms in One-Line Diagnostic Tips for Major Toxicological Emergencies (Clinical Pattern Recognition)

▪Pinpoint pupils + respiratory depression + dry lungs → Opioids overdose

▪Pinpoint pupils + wet lungs + muscle fasciculations → Organophosphate poisoning

▪Tinnitus + hyperventilation → Salicylate toxicity

▪Unexplained bradycardia with hypotension ± hypoglycemia → β-blocker overdose

▪Normal pulse oximetry + headache/confusion ± multiple exposed patients → CO poisoning

▪Mild symptoms followed by marked transaminase elevation (24–72 h) → Paracetamol overdose

Specific Poisonings

1. Paracetamol (Acetaminophen) Toxicity

2. Aspirin (Salicylate) Toxicity

3. Opioid Toxicity

4. Beta-Blocker Toxicity

5. Organ phosphorus Poisoning

6. Carbon Monoxide (CO) Poisoning

Paracetamol (Acetaminophen) Toxicity

Toxic doses

▪Paracetamol acute overdose >150 mg/kg → Potentially toxic acute dose.
▪Gradual intake >150 mg/kg/24h → High risk of toxicity

ICU Indications

Transfer to ICU is required for signs of acute liver failure which include:

¤ Hepatic Encephalopathy (altered mental status)

¤ Coagulopathy with INR > 2.0

¤ Metabolic Acidosis (pH < 7.3 after resuscitation), especially if refractory

¤ Hypoglycemia, renal failure, or hemodynamic instability.

Management

▪**Activated Charcoal:** Activated charcoal reduces paracetamol absorption from the gastrointestinal tract when given early within 1–2 hours of ingestion thereby reducing the amount of hepatotoxic metabolite formed and decreasing liver injury risk.

▪**N-Acetylcysteine (NAC) as Antidote:** NAC works by replenishing glutathione stores and acting as an alternative substrate to bind NAPQI، preventing liver injury

Opioid Toxicity

Opioids are drugs bind to opioid receptors in the central and peripheral nervous system to produce analgesia, sedation, and euphoria, and can also cause respiratory depression, miosis, & dependence.

Types of opioids:

▪Natural (Opiates): Morphine, Codeine

▪Semi-synthetic :Heroin, Oxycodone, Hydrocodone

▪Synthetic :Fentanyl, Methadone, Tramadol

Mechanism of opioids toxicity

▪Excessive μ-opioid receptor activation → CNS depression and respiratory depression

Management of opioids toxicity

Airway:protection is priority

Naloxone: Continuous infusion for long-acting opioids toxicity (titrate to ventilation, not arousal) Dose: 0.4-2 mg IV bolus, followed by infusion of 0.4-2 mg/hour, titrated torespiratory rate.

Observe:for re-sedation

Carbon Monoxide (CO) Poisoning

Carbon monoxide is a colorless, odorless, tasteless, non-irritant gas produced by incomplete combustion of carbon-containing fuels. Because it is undetectable by human senses, exposure often occurs unknowingly.

▪CO is highly toxic due to its ability to bind hemoglobin with high affinity, impairing oxygen delivery and causing systemic cellular hypoxia, particularly affecting the brain and heart.

Mechanism of CO Toxicity

▪CO binds hemoglobin with ~200× the affinity of oxygen → formation of carboxyhemoglobin (COHb) → impaired oxygen delivery

▪Inhibition of mitochondrial oxidative phosphorylation → cellular hypoxia→ shift to anaerobic metabolism → lactic acidosis

▪Causes direct myocardial depression and arrhythmias

Management of Carbon Monoxide toxicity

▪100% oxygen via non-rebreather oxygen mask delivery

▪Hyperbaric oxygen if:

- COHb >25% (or >15% in pregnancy)
- Depressed level of consciousness with preserved airway reflexes

▪Intubation not yet indicated

- Cardiac ischemia
- Severe metabolic acidosis

Hyperbaric Oxygen Therapy (HBOT): Administration done by placing the patient in 100% O2 chamber pressurized to 2-3 times the atmospheric

Thank You…….