Chapter 7

Pediatric Emergencies

CONTENTS :

Cardiopulmonary Resuscitation (CPR)

1. The diagnosis of cardiopulmonary arrest (CPR) must be made rapidly (absent pulse and respiration). Prompt and orderly resuscitative efforts are essential, and assignment of responsibilities is mandatory.

2. The team of resuscitation includes:

- The leader (the most experienced person), makes all therapeutic decisions, assigns others their roles, and continually reassesses the quality of the resuscitation.
- Vascular access should be accomplished by the next available person.
- Appropriate doses of medications and times of giving them are recorded.
- Historian,One person must obtain a history from the parents or caretakers.

3. Stages of CPR:

CPR consists of 3 successive stages: basic life support (BLS), advanced life support (ALS), and prolonged life support (PLS).

1. Basic life support (BLS): aims to provide emergency oxygen delivery to vital organs especially the brain and the heart. It includes the ABCs of resuscitation:

A. Airway control:

- Immobilize the cervical spine if spinal cord injury is a possibility.
- Clear the oropharynx with a suction catheter. Avoid blind finger sweeps.
- The head tilt –chin lift maneuver removes obstruction caused by the tongue.
- The head must be in the midline, in the “sniffing” position by placing a folded towel under the occiput.
- Oral airway placement may improve airway status.

B. Breathing support:

Begin mouth-to-mouth or bag-to-mouth ventilation with 100% oxygen. Assess the adequacy of ventilation by observing chest expansion. If chest wall expansion is insufficient, endotracheal tube (ETT) placement is indicated.

C. Circulation support:

- The patient is placed on a hard, solid surface, and external cardiac compressions are started immediately.

- Ventilation/compression ratio is 2:30 with the rate of compression is 100 per minute for infants and children.

- For infants, the two-finger technique is used where two fingers of one hand is used. The hand-encircling technique is preferred, where the infant is grasped with the fingers supporting the back and the thumbs over the middle third of the sternum. For toddlers, the heel of one hand is used and in the older child, two hands interlaced are used; in both cases, compressions are done two finger-breadths above the xiphoid.

2. Advanced life support (ALS): aims to restore spontaneous circulation by supporting circulation and treatment of the life-threatening arrhythmias.

A. Vascular access and fluid administration:

Peripheral venous access should be attempted. Proceed quickly to intraosseous needle placement at the proximal tibia if peripheralaccess fails. Central access (femoral) is best achieved using the catheter-over-a guide wire technique. Start infusion with volume expanders as Ringer's lactate or normal saline in an amount of 20 ml/kg over 10 minutes.

B. ECG monitoring and treatment of life-threatening arrhythmias:

- Bradycardia is treated with atropine.
- Asystole is treated with epinephrine and atropine,

- Ventricular fibrillation or pulseless ventricular tachycardia is treated by:

* Electrical defibrillation at a dose of 2 Joules/kg, repeated up to 3 times if needed.

* Epinephrine to change fine fibrillations into coarse ones.

* lidocaine

- Bicarbonate is used in prolonged arrest and for documented acidemia.

Drugs used in resuscitation:

Drug	Dose	Route	Indications
Atropine	0.02 mg/kg	IV,ETT	Bradycardia
Bicarbonate	1-2 mEq/kg	IV	Metabolic acidosis
Dextrose	0.5 gm/kg	IV	Hypoglycemia
Epinephrine	0.1 ml/kg (of 1:10.000)	IV, ETT	Asystole, bradycardia, hypotension
Lidocaine	1-2 mg/kg	IV, ETT	Ventricular ectopy
Naloxone(Narcan)	0.01 mg/kg	IV	Opiate intoxication

3-Prolonged life support (PLS): aims to identify and treat the cause of arrest and to promote recovery of the brain, heart and other vital organs.

A. Recognition and treatment of the cause: such as hypothermia, tension pneumothorax, cardiac tamponade, hypovolemia, metabolic imbalance, toxin ingestion or closed head injury.

B. Monitoring:

- Cardiovascular system: (by ECG, blood pressure, and central venous pressure).
- Pulmonary system: (by arterial blood gases and chest x-ray).
- CNS: (observe for seizures and signs of increased intracranial pressure).
- Renal system :(for renal function).
- Hematological system: (for DIC).

C. Multiple system support:

- Cardiovascular support: (volume expanders, inotropes).
- Respiratory support: (oxygen therapy, mechanical ventilation).
- Neurologic support :(control of convulsions, reduction of increased intracranial pressure).
- Metabolic support: (correction of temperature abnormalities, water imbalance, acid-base and electrolyte disturbances, blood sugar abnormalities).
- Hematologic support: (correction of anemia and DIC).

Poisoning

Ingestion of toxic products by children is a common occurrence. Children under 5 years of age account for 80% of recorded cases of poison ingestion.

1. Poison identification:

- The initial history should include the identification of the product ingested (containers or bottles should be brought).

- Physical examination will often reveal supporting evidence for a particular ingestion.

- When the nature of the substance ingested is unknown, the list of common symptoms or signs is presented in the followingtable.

- The specific substance causing a poisoning should be confirmed by qualitative analysis performed on blood or urine. Gastric fluid analysis will be of value if done within 2-3 hours of ingestion.

2. Supportive therapy:

a) **Cardiopulmonary support:**The ABCs items of cardiopulmonary resuscitation are applied for poisoned child.

b) **Fluid support:** Replace the previous and ongoing fluid losses while correcting electrolyte disturbances.

c) **Hematologic support:** Correction of hemolytic anemia with packed RBCs or exchange transfusion.

d) **CNS support:** For control of seizures and prolonged care of comatosed child.

e) **Renal support:** Renal function is monitored and hemodialysis is instituted as needed.

3. Gastrointestinal decontamination:

A) Gastric evacuation: it is the cornerstone of intervention after a toxic ingestion. Its efficacy falls when it is instituted more than one hour after ingestion. Ipecac syrup is the method of choice for gastric emptying, where it induces emesis within 15 minutes of intake. Orogastric lavage is as effective as ipecac and offers the advantage of speed and the prompt administration of adsorbent and cathartic.

Common symptoms and signs of toxic exposures (Toxidromes)	
System involved	**Substance involved**
Central nervous system:	
- Depression and coma	- Sedatives, narcotics, tranquilizers, tricyclic antidepressants, anticonvulsants, alcohol, hypoglycemic agents, aromatic hydrocarbons, lead, mercury, lithium and CO.
- Convulsions	- Amphetamines, xanthines, sympathomimetics, psychotropics, cocaine, ergot, strychnine, lead, organophosphates,
- Hallucinations	- Amphetamines, psychotropics, alcohol withdrawal, antihistamines, cocaine, tricyclic antidepressants.
- Hyperpyrexia	- Atropine, salicylates.
Cardiovascular system:	- Digitalis, quinidine, tricyclic antidepressants, cocaine
- Arrhythmias	
- Tachycardia	- Amphetamines, xanthines, sympathomimetics, cocaine, tricyclic antidepressants
- Bradycardia	- Beta blockers, cardioglycosides, quinidine, calcium-channel blockers.
- Hypotension	- Antihypertensive agents, tricyclic antidepressants, narcotics
Gastrointestinal system:	
- Nausea, vomiting, and diarrhea	- Almost any toxic substance can produce these symptoms and signs.
- Increased salivation	- Insecticides
- Decreased salivation	- Antihistaminic, antimuscarinic agents
Respiratory system:	
- Hypoventilation	- CNS-depressant agents
- Hyperventilation	- Salicylates, cocaine, nicotine, CO_2
Ocular system:	
- Mydriasis	- Atropine, sympathomimetics, psychotropics, cocaine
- Miosis	- Narcotics, organophosphate insecticides, parasympathomimetics
Cutaneous system:	
- Cyanosis	- Nitrites, aniline dyes
- Jaundice	- Carbon tetrachloride, benzene, phenothiazines

B) Adsorbents: activated charcoal forms a stable complex with the toxin, thus preventing its absorption. It is not given before ipecac and is not effective against metals, alcohols, hydrocarbons, or caustics. It is given in a dose of 1gm/kg in water orally.

C) Cathartics: as magnesium citrate and sorbitol. They hasten transit of gastrointestinal contents, thus decreasing systemic absorption of the toxin.

4. Elimination enhancement: methods of enhancing excretion of poisons include;

a. **Fluid and osmotic diuresis** by intake of hypertonic fluid.

b. **Diuretics,** such as furosemide (2 mg/kg/dose) are used to increase urine output.

c. **Ionized diuresis;** excretion of acidic compounds, such as salicylates and barbiturates, is enhanced by alkalinization of urine which is accomplished by IV sodium bicarbonate.

d. **Extracorporeal poison removal,** such as by hemodialysis, peritoneal dialysis and exchange transfusion.

5. Antidotes: the number of ingestions for which there is a specific antidote is small. The following table shows antidotal therapy of some specific poisoning:

Poison	Antidote	dose
Carbon monoxide	Oxygen	100% or hyperbaric O_2
Chlorpromazine	Diphenhydramine	0.5 -1 mg/kg, IV or IM.
Cyanide	Na nitrite, Na thiosulphate	depends on hemoglobin level.
Organic phosphorous	Atropine	0.1 mg/kg, IV every 10-30 min until papillary dilatation.
Opiates, narcotics	Naloxone (Narcan)	0.1 mg/kg, IV, may be repeated twice
Iron	Deferoxamine	10–15 mg/kg/hr, (IV infusion)
Isoniazide	Pyridoxine (B_6)	5 gm, IV
Methemoglobinemia	Methyline blue	1-2 mg/kg, IV over 10 min
lead	EDTA	250 mg/M^2/dose, IM, every 4 hrs.

Respiratory failure

Definition:

Respiratory failure is defined as significant alterations in the arterial PO2 and PCO2 due to alterations in the respiratory functions.

Pathophysiology:

Gas exchange alterations in respiratory failure results from abnormalities in:

* The mechanical properties of the lungs and chest wall,

* The function of the respiratory muscles or their innervation, or

* The respiratory control.

Types of respiratory failure:

* **Peripheral (type I = lung) failure,** due to poor arterial oxygenation, develops due to causes of respiratory distress, and present clinically with respiratory distress, with blood gases showing arterial hypoxemia± hypoventilation and acute metabolic acidosis.

* **Central (type II= respiratory pump)failure** , due to alveolar hypoventilation, develops due to causes of respiratory pump failure and present clinically with shallow breathing, cyanosis, coma or paralysis. with blood gases showing hypoventilation ± arterial hypoxemia and acute respiratory acidosis.

Etiology:

1) Lung diseases:

A] Airway obstruction:(central or peripheral):Tracheomalacia, subglottic stenosis, epiglotitis, croup, vocal cord paralysis , FB aspiration, Bronchiolitis, and bronchial asthma .

B] Pulmonary diseases:Aspirations, pneumonia, ARDS, pulmonary edema , pulmonary hemorrhage or embolism, and massive lung collapse.

2) Neuromuscular diseases: Guillain-Barre syndrome, botulism, birth trauma, werdnig Hoffman disease, poliomyelitis, and brainstem disorders.

Clinical manifestations:

* Respiratory failure should be anticipated rather than recognized, so that alteration in gas exchange can be prevented.

* Assessment should include respiratory rate, signs of respiratory distress, cyanosis,consciousness, and signs of respiratory obstruction.

* Symptoms and signs of the underlying disease.

* Symptoms of acute hypoxemia and hypercapneia include headache, lower back ache, restlessness, dizziness and impaired consciousness.

* Multisystem complications of acute respiratory failure include GIT hemorrhage, cardiac arrhythmia, renal failure and malnutrition.

Diagnosis:

* Underlying disease.
* Clinical manifestations of the patient.
* Radiological assessment of the cause of respiratory failure.
* Pulse oxymetry and capnography
* PaCO2 over 50 mmHg ---------- imminent respiratory failure.
* PaCO2 over 60 mmHg ---------- respiratory failure.
* PaO2 below 55 mmHg in room air.
* Metabolic and or respiratory acidosis.

Treatment:

1] Oxygen therapy.

2] Ventilatorysupport;The objective of mechanical ventilation is to provide adequate gas exchange.

3] Treatment of the underlying disease.

4] Extracorporeal membrane oxygenation (ECMO)

5] Inhaled nitric oxide.

SHOCK

Definition: A clinical state in which there is inadequate tissue perfusion to meet metabolic demands.

Types and causes of shock:

Hypovolemic	Distributive	Cardiogenic	Obstructive	Septic
▪ Dehydration ▪ Hemorrhage ▪ Burns	▪ Anaphylaxis ▪ Neurogenic ▪ Drug toxicity ▪ Early septic shock	▪ Acute heart failure ▪ Ischaemic heart disease ▪ Traumatic ▪ Toxic in late septic shock	**Venous:** ▪ Pneumothorax ▪ Pulmonary embolus: clot, fat or air ▪ Cardiac tamponade. **Arterial:** ▪ Critical aortic stenosis. ▪ Critical aortic coarctation ▪ Critical pulmonary stenosis.	• Fulminant sepsis without localization • Secondary to focal infection

Pathophysiology:

A. Early compensated shock:

- During this initial stage, the body compensatory mechanisms develop to maintain perfusion to vital tissues.
- This occurs through stimulation of the baroreceptors, chemoreceptors, ending in excessive release of endogenous catecholamines.
- These in turn augment the mean arterial blood pressure through increasing heart rate and contractility and peripheral vasoconstriction.
- This results in selective redistribution of blood to vital (brain, heart, lungs, kidneys) at the expense of non-vital organs (skin and extremities).

Thus, during this stage, **clinical manifestations** of shock include:

1. The clinical manifestations of the cause of shock (e,g, dehydration).
2. Tachycardia.
3. Normal blood pressure.
4. Signs of poor peripheral perfusion that include:
    - Cold extremities, and increased core/skin temperature difference (> 2°C).
    - Slow capillary refill time over finger nails (> 5 seconds).
    - Skin mottling and peripheral cyanosis**.**

B.Established shock: With continued hypoperfusion, failure of compensatory mechanisms occurs with subsequent hypotension.

The **clinical triad** of tachycardia, hypotension and poor peripheral perfusion becomes evident. **In addition**, manifestations of **hypoperfusion of vital organs** start to appear:

1. Metabolic acidosis (deep & rapid respiration),
2. Renal hypoperfusion (oliguria or urine flow less than 1ml/kg/hour)
3. Brain hypoperfusion (irritability followed by drowsiness and confusion).

C.Advanced decompensated shock: Manifestations of acute **failure** of different systems occur with a variable severity and different combinations.

Manifestations of multiple organs system failure:

- **Kidneys:** Acute renal failure (Oliguria, metabolic acidosis).
- **Lungs:** Adult respiratory distress syndrome (ARDS)**.**
- **GIT:** Ischemia, stress ulcers, hemorrhage, ileus.

- **Liver:** Acute hepatic failure.
- **Blood:** DIC, thrombocytopenia.
- **Metabolic:** Metabolic acidosis
- **Brain:** Hypoxic ischemic encephalopathy with coma.
- **Heart:** Myocardial ischemia, serious arrhythmias.

D. Irreversible (refractory) shock:

- Clinically, **myocardial ischemia** (serious arrhythmias) and **brain ischemia** (deep coma) are well evident.
- **Metabolic acidosis** is severe or profound and is refractory to therapy.

Treatment:

1. ABC's,
2. Cardiovascular system support,
3. Other systems support,
4. Specific treatment,
5. Monitoring.

I- Always Start With The ABC'S:

1)Airway: keep airway open as explained in resuscitation.

2)Breathing: oxygenation through different routes as mentioned in resuscitation.

3)Circulation: establish an IV line or other access for volume resuscitation.

II- Cardiovascular system support:

1 . Oxygen therapy: 100% oxygen is given by a facemask and the concentration can be gradually decreased over the next few hours. Endotracheal intubation and mechanical ventilation should be considered in case of marked distress.

2 . Preload augmentation: Expansion of intravascular volume with volume expanders (crystalloids and colloids) is initially indicated in all types of shock to improve tissue perfusion:

- **A crystalloid** (Ringer lactate or saline): It is initially given I.V. in an amount of 20 ml/kg over 10 - 15 minutes. The dose can be repeated once or even twice in case of poor response (persistent poor peripheral perfusion and/or hypotension).
- **A colloid** (albumin or plasma): It may be also given in an amount of 10 ml/kg, IV over a period of 15 minutes. It has the advantages of maintaining oncotic pressure and less tendency to leak into the interstitial spaces.
- **Whole blood transfusion**: 10 - 20 ml/kg can be also given in hemorrhagic shock or when hemoglobin level is very low.

Failure of response to 50 - 70 ml/kg of volume expanders over the first I - 2 hours should suggest cardiogenic shock or obstructive shock.

3. Contractility augmentation:

Drugs	Dose (µg/kg/min)	Effects
Dopamine	Low (0.5 – 4) Medium (5-10) High (11 – 20)	Renal vasodilator Inotropic Peripheral vasoconstrictor
Dobutamine	2 – 20	Inotropic Peripheral vasodilator Pulmonary vasodilator

4. Afterload reduction: The use of afterload reducing agents (e.g. nitroprusside or nitroglycerine) should be considered to improve myocardial performance in patients with severe cardiogenic shock not adequately responding to inotropic drug support.

Drugs	Dose (µg/kg/min)	Effects
Nitroprusside	0.5 – 10	Arterial dilatation (+++) Venous dilatation (+)
Nitroglycerin	1 – 20	Venous dilatation (+++) Arterial dilatation (+)
Amrinone	1 – 20	Vasodilator Inotropic

6. Treatment of arrhythmias:Initial treatment of any acute arrhythmias should include correction of hypoxia, acidosis and electrolyte disturbance (hypocalcemia, hypomagnesemia, hypokalemia or hyperkalemia). Antiarrhythmic drugs for bradyarrhythmias include atropine and isoproterenol. Supraventricular tachyrrhythmias is treated with adenosine, verapamil or propranolol. Lidocaine is the main drug for ventricular tachyarrhythmias

III- Multisystem Support:

• Respiratory support:

-Early oxygen therapy in all cases to prevent or delay respiratory fatigue.

-Endotracheal intubation and CPAP for pulmonary edema.

-Endotracheal intubation and CPAP or mechanical ventilation for ARDS.

-Hyper oxygenation and hyperventilation for acute pulmonary hypertension.

• Renal Support:

-Keep urine output above 1 ml / kg /hour.

-Give volume expanders, diuretics and low dose dopamine in oliguria.

-Consider peritoneal dialysis in severe cases.

• Metabolic support:

-Correct hypothermia and hyperthermia (both increase metabolic demands).

-Correct metabolic acidosis with sodium bicarbonate.

-Correct electrolyte disturbances (hypocalcemia, hypomagnesemia or hyperkalemia).

-Correct hypoglycemia or hyperglycemia.

- **Gastrointestinal support:**

-Antacids. cimetidine and cold saline wash for gastric stress ulcers.

-Intestinal decontamination may be considered to prevent gut translocation of bacteria. Rest of GIT in ileus (give maintenance IV. fluids, parenteral nutrition)

- **Hematological support:**

-Correct coagulopathies with vitamin K, fresh frozen plasma and platelets.

-Consider heparnization if peripheral gangrene occurs.

IV- Specific Treatment:

- **In sepsis or septic shock,** Early combined parenteral antibiotic therapy is essential.
- **In hypovolemic shock:** Specific replacement of the lost fluid (water, plasma, blood) is essential.
- **In obstructive shock**, management depends on the cause of obstruction.
- **In cardiogenic shock**, specific treatment of the underlying cause, if available, should be instituted. For instance, arrhythmias should be promptly corrected and rheumatic carditis should receive anti-inflammatory therapy including corticosteroids.
- **In anaphylactic shock:** Early drug therapy with adrenaline, hydrocortisone and antihistamines is important.

V- Monitoring:

Key items to monitor: Electrolytes, glucose, blood gases (pH and oxygenation), central, venous pressure, hemodynamic, coagulation status, urine output, and neurologic status.

ALTERED CONSCIOUSNESS AND COMA

Definition of Consciousness:

A conscious individual is:

a. Aware of himself and environment.

b. Capable of responding correctly to verbal and mechanical stimuli.

c. Able to recall past events.

Physiology of Consciousness:

Normal consciousness requires perfect functions of:

1. The reticular activating system (RAS), which is a collection of nuclei in the reticular formation of the brain stem.
2. Both cerebral hemispheres.

***Normally**, increased activity of the RAS, produces the alert conscious state whereas, decreased activity of the RAS reduces the activity of the cerebral cortex and produces sleep.

*Altered states of consciousness:

Interruption of the state of consciousness may occur at one or both these levels:

1. RAS**:** a small lesion is sufficient to produce coma.
2. Cerebral cortex: an extensive lesion is necessary to produce coma.

Assessment of the state of consciousness: The state of consciousness can be assessed by Glasgow coma scale.

The **Glasgow coma scale** is a useful tool for the grading of the degree of altered consciousness and the severity of CNS insult. Glasgow coma scale is used for adults and older children and its modification is used in infants and young children. The scale is simple, easy, can be applied at bed side and does not need any investigations

Glasgow Coma Scale (GCS)

ACTIVITY	BEST RESPONSE		
	Adults/Older Children	Infants (modified GCS)	Score
Eye Opening	1. Spontaneous	1- Spontaneous	4
	2. To speech	2- To speech	3
	3. To pain	3- To pain	2
	4. None	4- None	1
Verbal	1. Appropriate speech	1- Coos, babbles	5
	2. Confused speech	2- Irritable, cries	4
	3. Inappropriate words	3- Cries to pain	3
	4. Incomprehensible or none specific sounds	4- Moans to pain	2
	5. None	5- None	1
Motor	1. Obeys commands	1- Normal spontaneous movement	6
	2. Localizes pain	2- Withdraws to touch	5
	3. Withdraws to touch	3- Withdraws to pain	4
	4. Decorticate to pain	4- Decorticate to pain	3
	5. Decerebrate to pain	5- Decerebrate to pain	2
	6. None	6- None	1

Significance of Glasgow coma scores:

(1) Diagnosis of different grades of altered consciousness:

- Fully conscious child or infant is given GCS of 15.
- Mild to moderate grades of altered consciousness are given GCS from 14 – 11 as with lethargy, confusion and delirium.
- Severe grades as with stupor are given GCS from 9 – 10.
- As **coma** is defined as no eye opening (score 1), not obeying commands (score 5), and no recognizable words uttered (score 2), so the sum of G.C.S. = 8 . So, patients whose score = 8 or less are in coma. The lower the score the deeper is the coma.

(2) Follow – up of comatose children: Glasgow coma scores helps in follow-up of patients and in assessment of response to therapy.

(3) Prediction of prognosis of comatose child: Glasgow coma scores on admission can be used to predict mortality e.g. score ≤ 5 or less on admission, the probability of death is 90%, and the probability of death is decreasing to 1% with a score of ≥ 10.

Etiologic Classification of Coma:

I. Local CNS causes:

A.Head trauma may produce:

- Cerebral concussion.
- Cerebral contusion.
- Cerebral laceration.
- Brain edema.
- Extra and subdural hematoma.

B.Vascular: Hemorrhage, thrombosis, and embolism.

C. Epilepsy (Post-convulsion coma).

D.Central nervous system infections:

- Meningitis.
- Encephalitis.
- Brain abscess.
- Cerebral malaria.

F.Brain tumors.

II. Systemic causes:

1-Metabolic disorders:

- Metabolic acidosis: Diabetic ketoacidosis and acidosis complicating diarrheal diseases.
- Hypoglycemia: Infants of diabetic mothers and insulin over dosage in diabetic patients.

- Hypoxemia: Congestive heart failure, blue spells in infants with congenital cyanotic heart disease, acute respiratory failure and carbon monoxide poisoning.
- Hypernatremia and hyponatremia.

2-Renal failure.

3-Hepatic failure.

4-Severe systemic sepsis .

5-Heat stroke and hypothermia.

6-Hypertensive encephalopathy.

III. Drugs & Poisons:

Drugs	Poisons
▪ Atropine overdose. ▪ Barbiturates, benzodiazepines. ▪ Salicylate poisoning. ▪ Narcotics as morphia. ▪ Antihistamines overdose. ▪ Theophylline overdose.	▪ Lead poisoning. ▪ Kerosene poisoning. ▪ Carbon mono-oxide poisoning. ▪ Bango, hashish and other addicts. ▪ Inhalation of carbon tetrachloride, gasoline or other cleaning fluids. ▪ Organophosphrus poisoning ▪ Rubbing alcohol sponging

IV. Psycho - neurological problems: Hysteria or functional coma: rare, recognized by exclusion.

Diagnostic Approach ofa Child with Alerted Consciousness:

1. History:

- **Head trauma**.
- **Medication overdose, toxin ingestion**.
- **Seizures,** recent illness or exposure to infection suggestive of meningitis or cerebral malaria.
- **Chronic liver, renal, respiratory, metabolic diseases** especially diabetes, congenital and rheumatic heart disease.

- **Exposure to physical insults** (sun-stroke or hypothermia).
- **Failure to thrive with vomiting** in metabolic and degenerative diseases of the C.N.S.
- **Similar episodes in the past.**
- **Otitis media or ear discharge** (brain abscess).

2. Mode of onset:

- **Acute onset** with: trauma, toxins, cerebro-vascular accidents due to neurovascular malformations.
- **Subacute onset** with thrombosis.
- **Gradual onset** with progressive course with brain tumors or degenerative brain disease.

3. Family history:

- Family history of neonatal deaths, may suggest inherited metabolic disorders.

4. Clinical examination: Clinical examination of a comatose child aims at:

(1)Assessment of depth of coma: through GCS as mentioned before.

(2) Detection of causes of coma: e.g. head trauma, hepatomegaly, heart lesion, otitis media, etc…..

(3)Early detection of signs of increased intracranial pressure: (life-threatening brain herniation) through examination of papillary size & reactivity, ocular movements, breathing pattern and motor response to stimuli.

Laboratory investigations:

If the cause of coma is unknown or uncertain, a number of laboratory studies should be considered:

1. Blood examination:

- Blood: glucose, urea, creatinine, ammonia, bilirubin, and liver enzymes.
- Blood gases and pH: for metabolic disturbances.
- Blood picture: leukocytosis in bacterial infection, lymphocytosis in viral infection.
- Blood culture: in infectious causes.
- Blood coagulation studies: in bleeding disorders.
- Blood film for malaria.

2. Serum:

- Serum electrolytes: (Na+, K+).
- Osmolarity: high in diabetic ketoacidosis.
- Neutralization and complement fixation studies if there is possibility of viral encephalitis.
- Toxic screen: serum lead, serum salicylate, and other poisoning.

3. Urine: In addition to toxic screen, urine must be examined for characteristic odor, reducing substances, specific gravity, pH, ketones, glucose, RBCs, casts, and albumin.

4. C.S.F. examination: bloody in subarachnoid hemorrhage, purulent in bacterial meningitis. The lumber puncture is contraindicated if there are signs of increased intracranial pressure or if the patient is shocked.

5. Gastric lavage: examination of gastric aspirate is indicated if there is a possibility of poisoning.

6. Radiological investigation:

- Plain x- ray: Skull, spine, chest and heart.
- Brain imaging. (CT and MRI).

7. Electro-encephalogram: indicated in cases with unexplained stupor or coma.

8. Electro-cardiogram: and **Echocardiographic studies** may detect the cause of coma (e.g. embolism from chronic valvular lesion of the heart).

Management of the comatose patient:

I. Adequacy of the child's airway, breathing, and circulation ("the ABCs"):

- The child's neck should be immobilized carefully in neutral position unless it is clear that a cervical spine injury has not occurred.
- Insert an oral airway, clear vomitus and blood from pharynx by suctioning.
- Place your patient in lateral position.
- 100% supplemental oxygen should be given initially.
- Check gag reflex early in the course of care and the airway promptly protected by intubation if this reflex is inadequate.
- Hemodynamic status then should be assessed rapidly, paying attention to heart rate, blood pressure, and peripheral perfusion (pulses, capillary refill, and skin temperature of distal extremities).
- Establish an intravenous line.
- Insert an indwelling urinary catheter. This is important for the assessment of the fluid management and for obtaining urine samples.

II. Continuous monitoring:

- The conscious level by regular estimation of GCS.
- The vital signs.
- The blood electrolytes and blood gases.
- The arterial O_2 saturation should be monitored by pulse oximetry.
- The intracranial pressure to assess cerebral perfusion (CP),

  (C.P. = mean arterial BP – mean intracranial pressure).

- Development of bed sores with long-standing coma.
- Prevention of corneal ulcers by covering both eyes.
- Urinary catheterization with urine retention.

III. Immediate therapeutic intervention:

1. Treatment of reversible causes of coma:
   - Hypoglycemic coma: by IV D10-25W and run to give 4-6 mg glucose /kg/min and increase to keep glucose within the normal range (above 3 mmol/L).
   - DKA coma: by insulin and fluid therapy.
   - Narcotic overdose: Naloxone 0.5 mg IV.
2. Treat hyperthermia: by sponging with tepid water, antipyretics, and using a cooling blanket. Hypothermia requires careful re-warming of the patient.
3. Treatment of seizures: using diazepam IV 0.3 mg/kg/dose followed by phenytoin 10 mg/kg IV slowly. Maintenance phenytoin dose is then given 5 mg/kg divided in 2 doses.
4. Treatment of shock: as mentioned in chapter of shock.

IV. Prevention and urgent treatment of increased intracranial pressure:

Avoidance of factors that could increase ICP:

- Painful stimuli.
- Physiotherapy without sedation.
- Tracheal suctioning without sedation.
- Movements.
- Excessive fluid intake: Keep fluid at 80 ml/kg/day.
- Keep serum osmolality between 280-300 mosml/L
- Prevent hyperpyrexia.

Treatment of increased intracranial pressure by:

- Keep head in mid-position; elevate the head 30 degrees to augment venous drainage.
- Mannitol: 0.5 gm/kg over 20 minutes, followed by 0.25 gm/kg to be repeated as required and furosemide 1-2 mg/kg IV.
- Mechanical Ventillation: Aiming at reducing $PaCO_2$ between 20-25 mmHg. This will reduce brain blood flow and reduce brain edema.

Prognosis of Comatose Child:

This depends upon the following factors:

- **The etiology of the condition;** diabetic ketoacidosis has a more favorable outlook than anoxia and severe encephalitis.
- **Paralysis and placement on the respirator.**
- **Severity of the coma** (by Glasgow coma score).
- **The EEG** is also useful to estimate the potential for neurologic recovery.
- **Neurophysiologic studies** have also been used to make a prognosis for comatose children, including brain stem auditory, visual, and somatosensory evoked potentials (SEPs). Generally, the absence of all waveforms in these three modalities is associated with brain death or severe neurologic residua.