High Dependency Unit

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1. ACUTE FEBRILE ENCEPHALOPATHY

1.1 Learning objectives

After completion of this section the participants should be able to

- Identify a case of acute febrile encephalopathy
- Identify patients with intracranial hypertension
- Emergency resuscitation and stabilization
- Managing intracranial hypertension
- When to give empirical treatment in acute febrile encephalopathy
- Know when and how to refer?

1.2 What is acute febrile encephalopathy'!

Acute onset of fever and a change in mental status (confusion, disorientation, coma, or inability to talk) AND/OR new onset of seizures (excluding simple febrile seizure: refer box 1.1) is labelled as acute febrile encephalopathy (AFE)

BOX 2.1 Features of simple febrile seizure

Fever in a child aged 6 months to 5 years with single generalized seizure lasting less than 15 minutes

The child is otherwise neurologically healthy and without neurologic abnormality by examination or by developmental history

It is a medical emergency with both diagnostic and therapeutic challenge
Various causes of AFE are enumerated in Box 1.2

Box 1.2: Etiology of acute febrile encephalopathy

- Pogenic meningitis
- Viral meningoencephalitis
- Cerebral malaria
- Tubercular meningitis
- HSV encephalitis
- Scrub encephalitis
- Japanese encephalitis
- Dengue encephalitis

1.3 what are the neurocritical problems anticipated or seen in patient with AFE?

Raised intracranial pressure (ICP) or hypertension
Seizures or status epilepticus (refer section D, Chapter 2)

1.4 Intracranial hypertension

Monroe Kelley doctrine states

There is a pressure – volume relationship between volume of CSF, blood, and brain tissue in our non-complaint skull. Any increase in one component is compensated by decrease in the other, until a particular limit is reached, following which raised intracranial pressure manifests.

ICP varies with age. Normal reange of ICP is 5 -15 mmHg

Pediatric literature has shown as association between ICP of >20 mm Hg and poor outcome. Hence, treatment of ICP is recommended at a ICP threshold of 20 mm Hg or when clinical sings and symptoms of ICP are present.

1.5 How to identify a child with raised ICP? (Refer box 1.3)

BOX 1.3: clinical features or raised ICP

Symptoms	Signs
Acute raised ICP: Comatose/obtunded Decerebrate posturing Unexplained irritability, persistent shrill cry in infants Chronic raised ICP: Headache (worse in morning) Projectile vomiting Gradual deterioration in sensorium	Low GCS with or without posturing Neurogenic hyperventilation Bulging anterior fontanel in infants Anisocoria, Papilloedema Cushing's triad: Hypertension, bradycardia, irregular, respiration

1.6 How to manage?

Step I. Initial stabilization (The ABCDE approach)

A. Airway
    - Children are at risk for airway instability due to :
    - Depressed sensorium
    - Loss of tone in oropharyngeal muscles
    - Falling back of tongue which blocks airway
    - Pooling of secretions & risk of aspiration
    - Action

- Positon _ head-tilt-chin- lift, jaw thrust (refer appendix)
- Suction
- Insertion of nasopharyngeal/oral airway (refer appendix0
- Intubation if required (refer box 1.4)

Box 1.4 indications of intubation

Encephalopathy i.e. GCS<8
Unable to protect airway/aspiration
Impaired respiratory drive

Seizures
Raised ICP
Neuromuscular weakness – Hypoventilation – Respiratory failure
Inadequate ventilation/oxygenation

B. Breathing

Monitor SpO2 and breathing
Start supplemental oxygen by delivery device that is available
Start Bag & Mask Ventilation (BMV) in case of inadequate chest movements efforts.
Intubate If – severe hypoxemia, raised ICP, refractory status epilepticus failure of or prolonged BMV

C. Circulation

Adequate systemic perfusion is crucial to maintain adequate cerebral perfusion
Secure IV access
Monitor pulse, perfusion, blood pressure and urine output. Maintain adequate hemodynamic parameters using fluids or vasoactive drugs

D. Desability

Monitor neurological status by Glasgow coma scale (refer appendix)
Treat seizures (refer status epilepticus)
Identify signs of raised ICP (refer Box 1.3)
Correct hypoglycemia, hypocalcemia, hyponatremia

E. Exposure

Look for fever or hypothermia, any rash

Step 2. First tier management of raised ICP

Head in midline with 15-30 elevation
Ensure normoxia SpO2 > 94%
Hypercarbia causes cerebral vasodilatiation thus causing raised ICP
Mild hyperventilation for few minutes (Target PCO2 = 30-35 mm Hg) canc be used to manage acute ICP spikes (refer box 1.5)
Prolonged and /or aggressive hyperventilation should be avoided
Hyperventilation is not useful to achieve long lasting ICP reduction

Ensure normovolemia by assessing hydration status (heart rate, pulse pressure, pulse volume, CFT, blood pressure and urine output)
Maintain blood pressure at 50th centile.
Prevent events that increase ICP

Fever : Ensure normothermia by using round the clock antipyretics
Use sedoanalgesia for pain
Sedation: Midazolam 0.05 – 0.2 mg/kg over 2-3 min followed by 1-2 ug/kg/min (max 6 ug/kg/min) as infusion
Analgesia: Fentany 1-3 ug/kg/hr or Morphine 0.1 mg/kg/dose 4-6 hrly as needed
Paralysis; Use neuromuscular blocker, cecureonium 0.1 mg/kg/dose as required
Avoid noxious stimulation, premedicate with lignocaine prior to ET suctioning (nebulized (4% lidocaine mixed in 0.9% saline) or intravenous (1-2 mg/kg as 1% solution) given 90sec prior to suctioning
Start antiepileptics in case symptomatic seizures and post trauma patient
Maintain normoglycemia: RBS 80-120 mg/dl
Haemoglobin: Maintain around 10 gm/dl

Box 1.5: indication of acute mild hyperventilation
Mild short-term hyperventilation (Target a PaCO2 ˜mmHg)
Should be undertaken if danger of herniation are present
Unequal pupils
Posturing
New onset deterioration in sensorim

Step 3. Pharmacologic therapy of intracranial hypertension
Osmotherapy: Causes movement of water from parenchyma to circulation and also reduces blood visocity and cerebral blood volume to reduce ICP
Agents used are 3% NaCl (hypertonic saline) or mannitol
3% NaCl is preferred osmotherapy indicated in all cases of raised ICP especially in children with hypotension
3% NaCl: Dose: 5-10 ml/kg bolus followed by 0.1-1ml/kg/hr to maintain serum sodium between 145-155 mEq/L
Sodium monitoring has to be done every 6 hourly till infusion is ongoing followed by 12 hourly
Side effects: acute kidney injury

Mannitol: Indicated if signs of impending herniation are there.
Patient should be Normotensive as mannitol can precipitate hypotension.
Initial bolus – 0.25-1g/kg followed by 0.25-0.5 g/kg every 2-6 hourly as per requirement.

Side effects: Hypotension, rebound rise in ICP, hypokalemia, hemolysis and renal failure

Steroids: Dexamethasone (0.15mg/kg/dose 6 hrly) is indicated in
Tubercular meningitis
Intracranial tumours with perilesional oedema

Acetazolamide (20-100 mg/kg/day 8 hrly, max 2 g/day) is used in hydrocephalus (in infants with hydrocephalus ventricular tap can be attempted if anterior fontanel is open)

Barbiturate coma is reserved for refractory raised ICP. It causes decrease in cerebral blood flow and cerebral metabolic rate thus decreasing ICP. Thiopentone: 3-5mg/kg loading dose followed by 1-5mg/kg/hr is used for barbiturate coma. This drug however needs ICP and EEG monitoring. The most dreaded side effects is hypotension.

Step 5: Refractory intracranial hypertension (second tier therapy)
Defined as no improvement despite osmotherapy and acute mild hyperventilation

Modalities use are

Barbiturate coma
Mild hypothermia (core temperature 32-34 C)
Need to begin within 8 h of injury, and maintained for 48 h
Done by surface cooling
Speed of rewarming should be less than 1 C every 4-6 hr

Decompressive cranectomy: On rare occasions when all other measures fail, decompressive craniectomy may be valuable.

1.7 Specific therapy
Choice of empirical therapy depends on the geographical location, season and the local microbiological data

Box 1.6

Etiology	Clinical clues	Treatment
Bacterial meningitis	Acute febrile encephalopathy with or without seizure	Ceftriaxone (100 mg/kg/day in 2 divided doses)
HSV encephalitis	Fever with progressive deterioration of consciousness, focal seizures or focal neurological abnormalities in the absence of any other cause	Acyclovir (30 mg/kg/day in 3 divided doses)
Cerebral malaria	AFE with pallor, icterus hepatospleonomegaly	IV Artesunate (3 mg/kg/dose in < 20

		kg; 2.4 mg/kg/dose in >20 kg
Scrub typhus	AFE with organomegaly, capillary leak, eschar	IV doxycycline (4.5mg/kg/day 12 hourly)
Tuberculous meningitis	Subacute/chronic history of encephalopathy with h/o possible koch's contact	SATT after appropreiate investigations
Enteroviral encephalitis	Preceding loose stools and rash- summer/monsoon	Supportive treatment
Japanese encephalitis	Altered sensorium, changing neurological signs, extrapyramidal movements	Supportive treatment
Vaircella encephalitis	Vesicular rash, cerebellar signs	Supportive treatment
Rabies encephalitis	H/o dog-bite, bulbar signs	Supportive treatment

1.8 How to monitor?

Child should be monitored by a nurse at least every 2 hourly and by a doctor at least 4 hourly

What to look for?
GCS, pupillary size and reaction, seizures, new signs
Any other new signs of worsening
Hemodynamic and respiratory parameters

1.9 How to investigate?

- Complete blood count
- Serum electrolytes, renal function test, liver function test
- Peripheral smear for malaria and/or rapid malaria antigen test: In endemic areas with symptoms & signs: pallor, organomegaly
- Dengue serology: In endemic areas with signs & symptoms: capillary leak with bleeding manifestations
- Rickettsial serology: Endemic areas with eschar, organomegaly, capillary leak
- Lumber puncture: Only in hemodynamically stable patients with no signs of raised ICP
- Neuroimaging : CT brain is indicated in
  - Focal neurological deficit

- Aferbrile cause of encephalopathy
- History of trauma
- Unexplained pallor
- If lumbar puncture is contraindicated

1.10 When to refer?

- Cases with persistent raised ICP
- Non improvement in sensorium within 3-5 days
- Need for neurosurgical procedures like ventriculo-peritoneal shunt, evacuation of bleed or decompressive craniotomy
- Refractory status epilepticus

1.11How to refer

Secure airway, breathing and circulation
Ensure 2 patent IV lines and fluids
Premedicate with lignocaine, midazolam, morphine before transfer to prevent excessive movement of patient
Ensure resuscitation kit and a BLS trained provider through transport
Monitor: Vitals, sensorium and pupils
Have anti seizure mediations also readily available

Remember:

- In a child with acute onset sltered encephalopathy with or without pupillary changes or brisk reflexes, assume raised ICP until proven otherwise
- Target normothermia, normoxia, normovolemia, normoglycemia
- No role of prophylactic hyperventilation.
- Mild short-term hyperventilation (target a $PaCO_2$ ≈30-35 mmHg) is indicated if signs of herniation are present
  - Unequal pupils
  - Posturing
  - New onset deterioration in sensorium

Protocol for Acute Febrile Encephalopathy

Identify case of acute febrile encephalopathy
Recgnise signs and symptoms of raised ICP

Indications of acute mild hyperventilation

Mild short – term hyperventilation (Target PaCo2≈ 30-35 mmHg) should be under
Taken if danger of herniation are present
Unequal pupils
Posturing
New onset deterioration in sensorium

Immediate measures

A.Maintain Airway
B. Assisted breathing in a child with apnea, bradypnea or irregular brething
C. Adequate circulation to maintain cerebral perfusion with the help of fluid or vasoactive drugs
D. Dextrose check & correction of hypoglycemia
E. Look for fever/hypothermia

Supportive measures

First tier Measures

Head in midline with elevation 15-30
Ensure normoxia Spo2 > 94 %
Ensure normovolemia by assessing hydration Status (heart rate, pulse pressure)
Maintain BP at 50th centile
Prevents events that increase ICP; fever, pain, noxious stimulation,seizures,
Maintain euglycemia: RBS 80-120 mg/dl
HB: Maintain around 10 gm/dl
If any surgical cause of encephalopathy then refer to higher centre where neurosurgical facilities are available
Specific therapy (refer box 1.6)

Pharmocotherapy

- 3% NaCl is preferred osmotherapy indicated in all cases of raised ICP especially with hyptotension
- Dose: 5-10 ml/kg bolus followed by 0.1-1 ml/kg/hr
- Side effects: AKI
- Mannitol: Used in case of impending herniation if 3% Nacl is unavailable provided patient is normotensive
- Intial bolus – 0.25 – 1 g/kg followed by 0.25-0.5 g/kg every 2-6 h as per requirement side effect:
- Hypotension, rebound rise in ICP, hypokalemia, hemolysis and renal failure
- Steroids: Dexamethasone (0.15mg/kg 6hrly)
- Indicated in TBM,m Intracranial lesions with surrounding oedema
- Acetazolamide (20-100 mg/kg/day 8 hrly) in hydrocephalus
- In infants with hydrocephalus ventricular tap can be attempted in anterior fontanelle open

Second tier measure
Possible only with ICP monitoring

Barbiturate coma:
Thiopental or pentobarbital
Moderate Hypothermia (32-34 C)
Decompressive cranectomy

2. STATUS EPILEPTICUS

2.1 Learning objectives

After completion of this section, the participants should be able to

- Identify a case of status epilepticus
- Provide adequate first line antiepileptics to abort seizures
- Stabilise and manage ABC's
- Send appropriate investigations to detect the cause of seizures
- Look for reversible causes and treat them
- Escalate anti epileptic therapy based on need
- Know when and how to refer

2.2 What is status epilepticus?

- A seizure lasting for more than 30 minutes or recurrent seizures for more tham 30 minutes during which time the patient does not regain consciousness.
- Operational Definition:
  - Prolonged seizure activity 9>5mins) or persistent, repetitive, seizure activity without recovery of consciousness in between episodes.
  - Any child who is brought seizing to the emergency room should be treated as status epilepticus.
  - A child with epilepsy should be considered in status if the seizure persists for more than twice the usual duration of the previous seizures.

2.3 Steps in management

Start immediate stabilization (ABCD approach)

A. Airway

Position the child's head to one side and suction secretions (oral followed by nasal)

Place an oropharyngeal airway if required, to be avoided in conscious patients

Endotracheal intubation (Refer box 2.3)

Box 2.3 Indications of intubation

- Severe hypoventilation and hypoxia
- Failure of bag and mask ventilation
- Prolonged requirement of bag and mask ventilation
- Raised ICP

B. Breathing

- Monitor SpO2 and breathing.
- Start supplemental oxygen by delivery device that is readily available.
- Start bag & mask ventilation (BMV) in case of tachypnea, inadequate chest movements, poor air entry.

C. Circulation

- Secure IV access, monitor pulse, blood pressure and perfusion
- Correct reversible causes like hypoglycemia, Hypocalcemia, hyponatremia (Refer box 2.4)
- Begin continuous cardio – respiratory monitoring
- Abort seizure with escalating antiepileptic drugs and doses (Refer to algorithm)

Box 2.4 Correction of reversible factors

Calcium (if ionic value not available then do ECG): 2 ml of calcium gluconate in equal dilution with D5 over 20 mins under heart rate monitoring (ceiling dose of 10 ml)
Sodium: 5 ml/kg hypertonic saline (3%) bolus
Glucose: Remember product of volume and concentration of destrose should be 50

- 2 ml/kg of 25% dextrose
- 5ml/kg of 10% dextrose
- 10ml/kg of 5% dextrose

2.4 What are the causes of status epilepticus?

Box 2.1: Etiology of status epilepticus

- Acute:
  - CNS infections (meningitis, meningoencephalitis)
  - Febrile convulsions
  - Vascular episodes
  - Trauma
  - Metabolic
  - Poisonings

- With or without and underlying neurologic disorder
  - Non – compliance or withdrawal of anti-epileptic therapy

2.5 What are the conditions that mimic seizures?

Box 2.2 Non epileptic events
Syncope
Usually preceded by blurred vision, dizziness and pallor

Gastroesophageal reflux
Result in an arched back position with crying
No loss of consciousness & events associated with feeding
Cyanotics breath holding spells
Day dreaming
Pseudoseizure
Tetanus (patient will be conscious)

2.6 Investigations

Blood Glucose
Serum electroluytes (serum sodium, potassium, chloride)
Serum calcium (If ionic calcium not available them do ECG and calculate QTc)
If febrile: hemogram, lumber puncture
Neuroimaging: CT scan (refer section D, Chapter 1)

2.7 Monitoring

- Child should be monitored by a nurse at least every 4 hourly and by a doctor atleast 6 hourly
- What to look for?
  - Recurrence of seizure
  - Neurological status 2 hourly
  - Hemodynamic status 2 hourly

2.8 When to refer?

- Refractory status Epilepticus: Seizures that persist even after the adequate treatment with benzodiazepine, phenytoin or any second line agent.
- History of trauma, developmental delay, prolonged fever
- Examination suggestive of meningeal irritation, neurocutaneous marker

2.9 How to refer?

Refer only after stabilization, ensuring adequate airway, breathing and circulation
Proper documentation regarding the presentation of the child, details of resusciatative measure taken, interventions done during resuscitation and reasons for referral including name and contact number of referring physician
A doctor/paramedic trained in Pediatrics Advanced life Support/ Basic Life Support to accompany the patient
Transporting ambulance should have sufficient O2 supply and Resusciatation equipment

Inform the referral centre prior to sending the patient regarding the diagnosis of the child, indication for referral, current status and approximate time to arrival
Counsel the family of the child regarding the need for transfer and risks during transfer . Obtain written, signed consent for the same

Remember:

- If IV access not available
- Buccal/nasal midazolam 0.2-0.3 mg/kg (max 5mg) or
- Per rectal Diazepam 0.5 mg/kg (max 10mg) or
- IM Midazoloam 0.2 mg/kg (max 5 mg)

- If the patient is already on phenytoin, then administer mini loading 10 mg/kg of phyenytoin s the initial anticonvulsant
- If the patient is already on valproate, the administer 10 mg/kg as the loadind dose. The infusion is continued until seizure free for 6 h, then tapered off @ of 1 mg/kg/h every 2 hourly
- Midazolam infusion is preferred over valproate in children less then 2 years of patients with liver failure
- Calcium infusion should be given only after hypocalcemia is confirmed or clinical examination
- How to calculate QTc interval?

Normal QTc – 0.44 sec, borderline QTc -0.44-0.46 sec, prolonged QTc -0.46 sec

- Thiamine 100 mg IV push: pyriodoxine 100 mg IV push can be tried in refractory in children < 3 years

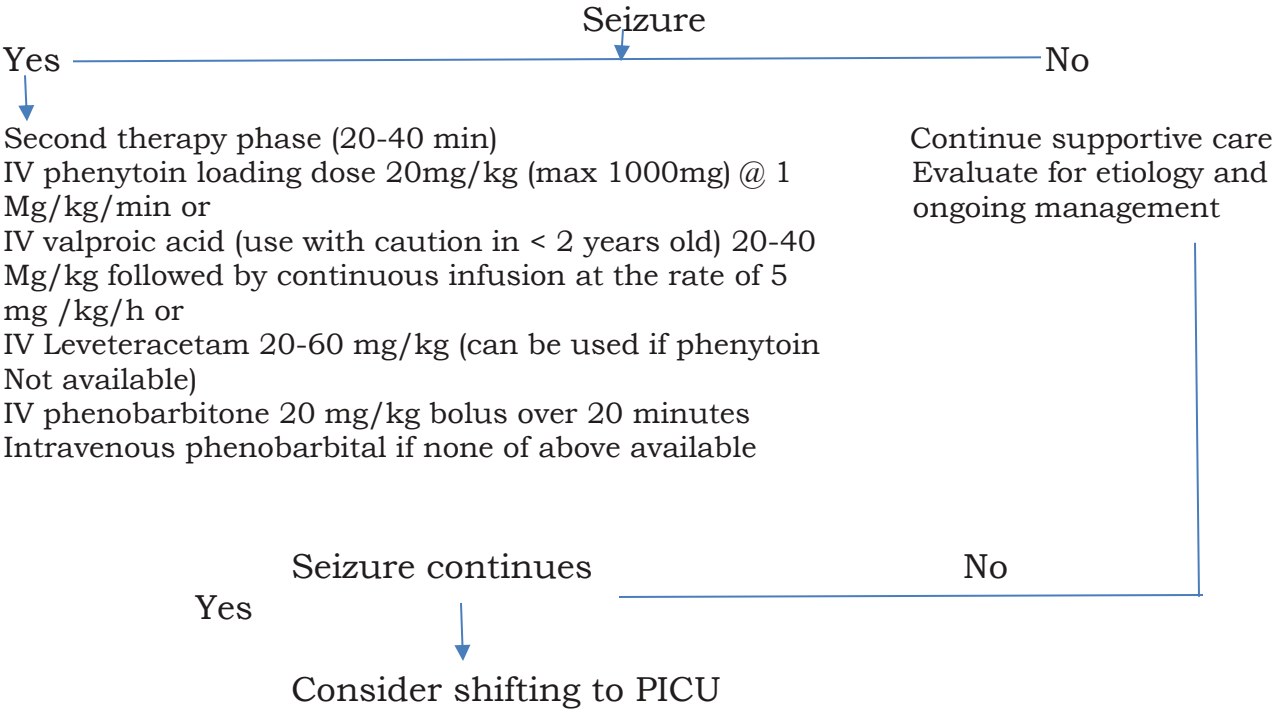
Identify a case of status epilepticus

Stabilisation phase (0-5 min)

- Initial stabilization (0-5 mins) (ABCDE).
- Check airway patency, clear secretions
- Supplemental oxygen
- Immediate intubation if indicated
- IV/IO access, correct reversible cause

Initial therapy phase (5-20min)

IV lorazepam (can repeat once, max 2 doses) or
IV diazepam (can repeat once, max 2 doses) or
Inj Midazolam 0.15 – 0.2 mg/kg IV (max 5mg)
If IV access cannot be established.
IM midazolam or Instransal/Buccal midazolam or rectal diazepam



Third therapy phase

Repeat second line therapy: Use drugs not used in second line
If not controlled then start midazolam 0.2mg/kg bolus followed by 0.05-02 mg/kg/hr
If not controlled then consider pentobarbital, or propofol (all with continuous EEG monitoring).
In children below 2 years of age, pyridoxine (100 mg intravenously) may be tried

**Midazolam infusion needs to be considered if seizure not controlled in 60 min.

1. ACUTE POISONING

1.1 Learning Objectives

After completion of this section the participant should be able to

- Identify a child with acute poisoning
- Identify different toxidromes
- Steps in emergency stabilization of every case
- Decontamination and specific antidotes required
- Know when and how to refer?

1.2 Poisoning in children

- Most cases of poisoning in children are accidental and involve a single toxic substance
- Most common route of poisoning in ingestion followed by dermal, inhalational and ocular exposure
- Most cases are due to non-pharmaceutical substances like cosmetics, cleaning solution, pesticides, hydrocarbons, plants etc.
- Commonly ingested pharmaceutical substances include analgesics, topical preparations, anticonvulsants, psychiatric medications, anti-diabetic drugs, vitamin preparations, etc.

1.3 How to identify a child with acute poisoning?

- Suspect acute poisoning if circumstantial history is suggestive, e.g:
    - Child becomes sick during/after group play
    - Child is unsupervised at home
    - Younger sibling becomes ill while playing with older sibling
    - Drugs/wrapper/household products lying on the floor
    - Missing quantity of drugs.
- History and physical examination clues to diagnosis of a specific toxin ingestion
- Most importantly identify toxidrome. A toxidrome is aconstellation of characteristic signs and symptoms associated with exposure to particular substance or class of substances (refer box 1.1, 1.2, 1.3)

Box 1.1 Common toxidromes

Toxin	Syndrome
Cholinergic syndrome (organphosphates)	Diarrhea, diaphoresis Urination Miosis Bradycardia, bronchorrhea, bronchospasm Emesis (vomiting) Lacrimation Salivation Others: weakness, fasciculation
Anticholinergic syndrome	Flushing (red as a beet) Dry skin and mucosa (dry as a bone) Hyperthermia (hot as a hare) Delirium (mad as a hatter) Mydriasis (blind as a bat) Tachycardia Urinary retention
Sympathomimetic agents (Amphetamines)	Mydriasis Anxiety Diaphoresis Hyperthermia Tachycardia Hypertension
Opioids	Altered sensorium, respiratory depression, pinpoint pupils

Box 1.2: Common clinical signs in poisoning and their causes

Clinical signs	Toxins
Miosis	C Cholinergics, clonidine **O** Opiates, Organophosphates P Phenothiazine, Pilocarpien, Pontine bleed S Sedative- hypnotics
Mydriasis	A Antihistaminics A Antidepressants A Anticholonergics, Atropine S Sympathomimetics (Cocaine, Amphetamines)
Hyperthermia	Salicylates, Anticholinergic
Hypothermia	Opioids, Barbiturates
Tachypnea	Hydrocarbons, Salicytes, Cyanide, Ethylene glycol, Iron
Bradypnea	Opiods, Barbiturates, Benzodiazepines
Hypertension	Sympathomimetic, Withdrawal syndrome
Bradycardia	Digitals, Beta-blockers, Calcium channel blockers
Depressed sensorium	Opioids, Barbiturates, Benzodiazepines, Hydrocarbons
Seizures	Organophosphates, hydrocarbons, Tricyclic antidepressants, Anti-diabetic drugs, sympathomimetic, Camphor, Ethanol, lithium, Lead
Delirium	Anticholinergic, Dhatura, Amphetamines
Excessive sweating	S Sympathomimetic O Organophosphates A Amino Salicylic Acid
Fasciculation	Organophosphates
Blue skin (cyanosis)	Methemoglobinemia
Specific breath odors **Garlic** **Bitter almonds** **Fruity** **Rotten eggs** **Mothballs**	 Organophosphates, Arsenic Cyanide Alcohols Sulphur dioxide, Hydrogen sulphide Camphor

Box 1.3: Common toxins and their clinical features

Common Agents	Clinical features
Acids and alkalis	Airway, esophageal and gastric burns
Anti-diabetic drugs	Hypoglycemia, seizures
Antimalarials (Chloroquine)	Seizures, dysrhythmias
Camphor	Seizures
Hydrocarbons (Kerosene etc.0	Respiratory distress, altered sensorium
Laundry detergents	Respiratory distress, altered sensorium
Lindane (benzene hexachloride)	Seizures, dysrhythmias
Tricyclic antidepressants	Altered sensorium, seizures, dysrhythmias, hypotension

box 1.4 goals of management

- emergency stabilization (PRIORITY)
- Post stabilization management
- Appropriate decontamination
- Administration of spefic antidotes

Steps in management of a child with poisoning

The goals of management are as outlined in Box 1.4

1.4 Emergency Stabilization: ABCD3E approach

A: Ensure clear and patent airway – intubate if indicated (refer box 1.5)

B: Assisted breathing by AMBU bag if apnea, bradypnea or irregular breathing present.

C: Ensure stable circulatory parameter to secure cerebral perfusion. Use fluids or vasoactives as indicated.

D:

D1: Check dextrose, correct hypoglycaemia if present

D2: Disability assessment. Check neurological status and control seizures. Benzodiazepines are the drug of choice. IV Phenytoin is usually avoided for seizures due to toxins and it may intensify seizures in cocaine, lindane, theophyline overdose

D3: Decontamination

E: Expose the child completely and look for residual toxins/burns/scalds. Wash if caustic/dermal toxins suspected. Control hyperthermia with cooling methods.

BOX 1.5: Indications for intubation
GCS<8
Apnea or bradypnea
Inability to maintain patent airway (pooling of secretions despite repeated suctioning or depressed sensorium)
Prolonged seizures
Airway injury: Corrosive ingestion

1.5 Decontamination

This is most effective within first hour (golden hour) after exposure, however, can be used later also

- **Skin:** Remove contaminated clothing, wash skin thoroughly with soap and water for 15 mins
- **Eyes:** Irrigate with normal saline for at least 15 mins
- **Gastrointestinal tract:**
  - **Emesis:** It has no role
  - **Gastric lavage**
    - Done during first hour (golden hour) of toxin ingestion
    - 15 ml/kg (max 200-400 ml/cycle) normal saline is used
    - Administered through largest bore nasogastric tube with patient lying on side and head end lowered. Wide bore tube may be required in case of iron tablets ingestion
    - Contraindications: corrosives, hydrocarbons, absent protective reflexes, depressed sensorium
    - Complications: Pulmonary aspiration
  - Activated charcoal;
- Effective when given within first hour of ingestion
- Airway should be intact/protected before administering charcoal
- Dose: upto 1 year: 1g/kg, 1-12 years: 25-50 g (1 tablet = 50gm)
- Mix required amount in glassful of water or saline and give orally or by nasogastric tube
- Contraindications: paralytic ileus, intestinal obstruction, peritonitis
- Toxins not adsorbed to charcoal: corrosives, hydrocarbons, alcohol, metal like iron, lithium

  - Whole bowel irrigation:
    - Decontaminates entire gut
    - Used for agents not bound to activated charcoal and in ingestion of significant amount of iron and sustained release preparations
    - 20 ml/kg/hr polyethylene glycol is given via nasogastric tube till there is a clear rectal effluent
    - Contraindications: ileus, intestinal obstruction, perforation, gastrointestinal bleed

1.6 Enhacement of elimination

- Active elimination techniques have limited role, restricted to any of the following situations:
  - Hemodynamic instability despite supportive measures
  - Intractable seizures
  - Organ failure

- Various techniques used are forced diuresis, urinaryalkalinisation, hemodialysis, peritoneal dialysis, hemoperfusion, exchange transfusion, palsmapheresis

Box 1.6: Specific antidotes

Toxin	Antidote	Dose
Acetaminophen (Paracetamol0	N-Acetyl Cysteine	Oral: Loading Dose Of 140 Mg/Kg IV: 150 Mg/Kg Loading Over 15 Min Followed By 50 Mg/Kg Over 4 Hours Then 100 Mg/Kg Over 16 Hours Dilute In NS Or 5% Dextrose without causing fluid overload
Anticholinergics	Physostigmine	0.5 mg slow IV over 5 minutes, repeat every 10 minutes till max 2mg
Anti-diabetic drugs (sulphonylureas)	Dextrose Octreotide	2ml/kg 25% Dextrose; 5ml/kg 10% D 1-1 µ/kg every 6 hrs for 24 hrs
Benzodiazepines	Flumazenil	0.2 mg IV over 30 sec, repeat every minute till max 0.5 mg
Beta-blockers	Glucagon	0.05 mg/kg IV bolus followed by 0.05/kg/hr IV infusion
Calcium channel blockers	Calcium, Intropes, Glucagon (myocardial depletion), Insulin	Calcium gluconate 2ml/kg (max 10 ml) Glucagon loading dose 50µ/kg followed by 50-100 µ/kg/hr infusion Glucose loading 0.5 g/kg followed by regular Insulin 0.1 µ/kg bolus followed by

		0.1 to 0.2 µ/kg/hr infusion
Carbon Monoxide	Oxygen	100 % oxygen preferably hyperbaric (2-2.5 atm)
Cyanide	Amyl nitrate/ sodium nitrate/ Sodium thiosulphate/ Hydroxyc obalamin	1 vial (40 mg of Fab) binds 0.6 mg digitals infused over 30 min
Ethylene glycol/Methanol	Ethanol Fomepizole	Load 750 mg/kg followed by a maintenance of dose of 80-150 mg/kg/h infusion Load 15 mg/kg IV maintenance 10mg/kg 12 hrly IV (4 doses)
Iron	Desferoxamine	15mg/kg/hr IV infusion diluted in 5% dextrose (max 360 mg/kg upto a total of 6g)
Isoniazid	Pyridoxine	1 g of IV pyridoxine per g of INH ingested
Lead and other heavy metals	BAL DMSA Calcium disodium EDTA	500 mg/sqm/24hours 1500 mg/sqm/24 hrs
Methemoglobinemia (nitrates, nitrites)	Methylene blue (1%)	1-2ml/kg/dose IV may be repeated 6 hrly
Opioids	Naloxone	0.1mg/kg/IV, max 2 mg
Organophosphates	Atropine/Pralidoxine	(Refer section C, Chapter 2)
Salicylates/Tricyclic antidepressants/Other Sodium channel blocker	Sodium bicarbonate	IV 1-2 mEq/kg Titrate to maintain urine pH >8

1.7 When to discharge a patient with suspected poisoning?

- When the child is stable without any need for life – support measures for at least 24 hours and no further delayed manifestation are expected
- Discharge only during daytime
- Child safety and parental education mandatory before all discharges
- Mandatory psychiatric review if self-harm is suspected before discharge

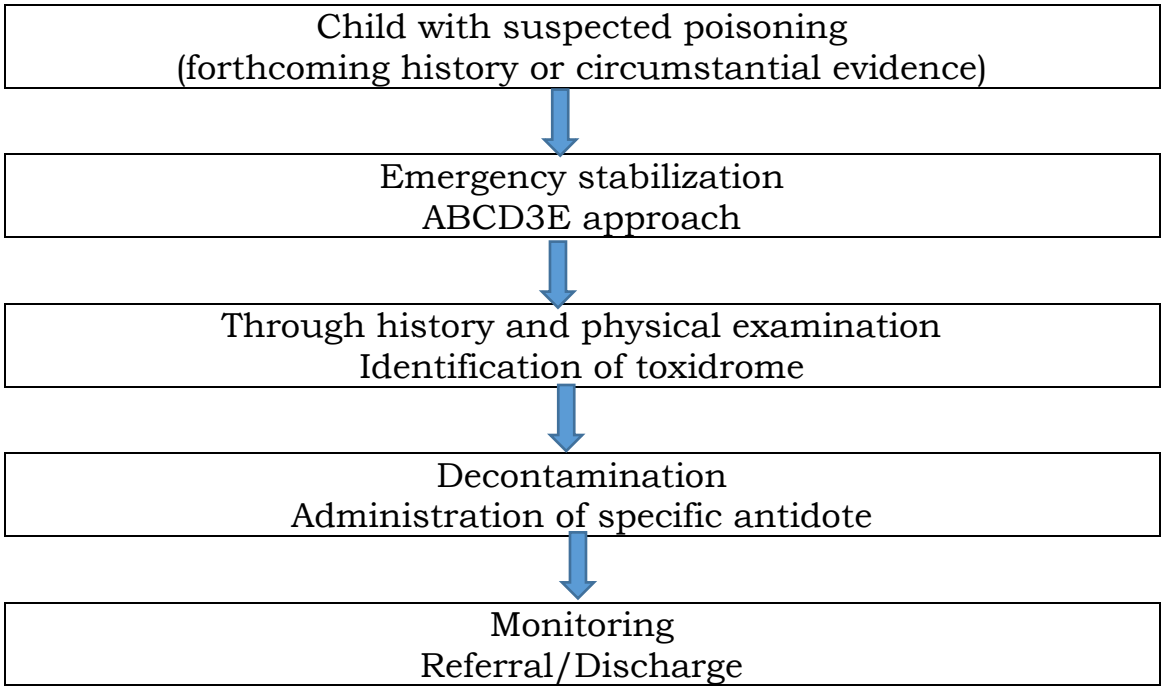
1.8 When to refer?

- Persistent altered sensorium
- Requirement of assisted ventilation
- Hemodynamic instability
- Need for renal replacement therapy
- Non-availability of appropriate antidote/s
- Need for ICU care and monitoring

1.9 How to refer?

- Secure airway, breathing and circulation
- Ensure resuscitation kit and a BLS trained provider during transport
- Child's peripheral venous access should be secured and patent
- Child's details, history, examination findings, vitals events which occurred and treatment given should be legibly written in the referral letter
- The parents should be counselled regarding the child's condition and need for referral
- Written, informed consent should be taken from the parents/guardian's prior to referral

Management Algorithm



Remember:

- High index of suspicion for poisoning in presence of circumstantial evidence of vague history
- If unknown poison, assume worst case scenario
- No toxic panel is uniformly helpful
- Resuscitation and stabilization must precede all other measures
- Basic supportive care saves more lives than all the antidotes put together

2. Hydrocarbon Poisoning

2.1 Learning objectives

After completion of this section the participants should be able to

- Identify child with suspected hydrocarbon poisoning
- Perform emergency stabilization
- Supportive management
- Know when and how to refer?

2.2 Hydrocarbon poisoning

- Hydrocarbon includes all compounds composed predominantly of carbon and hydrogen
- Ingestion of hydrocarbons is quite common in Pediatrics toxicological emergencies.
- Common products with hydrocarbon includes kerosene, petrol, diesel oil, turpentine, furniture polish, household cleansers, propellants in mosquito repellent liquids.
- Types of hydrocarbon compounds:
  - Aliphatic: petroleum distillates, and we are most common type of hydrocarbon exposure
  - Halogenated: Commonly found is solvents, dry-cleaners, degreasers, vehicles for paints, varnishes
  - Aromatic: Exposure is usually in occupational setting hence rare in Pediatric Patients
- Mortality and morbidity is maximum in patients younger than 5 years of age.
- Exposure can occur by unintentional ingestion or inhalation, intentional recreational abuse in adolescents and dermal exposure or oral ingestion in a suicide attempt.

2.3 Toxicokinetics of hydrocarbon poisoning

- The toxic potential of hydrocarbons is directly related to dose and physical properties of the compound
- Physical properties (refer box 2.1)
  - Volatility is the compound's ability to vaporize. The higher the volatility, the easier the compound is to inhale.
  - Viscosity is the compound's resistance to flow. The aspiration potential of the compound decreases as the viscosity increases
  - Surface tension: This property allows the substance to spread over the contacted surface

Box 2.1: Properties of hydrocarbon

Physical Properties	**Aliphatic**	**Aromatic**	**Halogenated**
Examples	Kerosene	Benzene, industrial solvent	Carbon tetrachloride, dry clean or degreasing solvents, Fire extinguishers
Viscosity	Low	Less	High
Volatility	High	High	High
Surface tension	Low	Low	Low
Clinical features	Respiratory features secondary to aspiration	Respiratory and CNS features	Respiratory features Systemic features (CNS, Cardiac, hepatic and renal systems) as readily absorbed from GIT

2.4 What are the clinical features of hydrocarbon poisoning? (refer box 2.2)

Box 2.2 Clinical features

Symptoms	Signs
• Respiratory symptoms develop within 30 min of aspiration • Choking, coughing, difficulty in breathing • Kerosene odour in breath • Fever • Irritability, drowsiness, seizures	• Tachypnea, retractions, cyanosis • Wheeze, crackles

2.5 How to investigate?

- Chest X-ray
  - All symptomatic children with suspected aspiration should have a CXR
  - Radiographic changes usually occur 2 to 72 hours after hydrocarbon exposure.
  - It is advisable to wait for 4 to 6 hours before performing the CXR in asymptomatic patients

- Radiographic changes can be unilateral or bilateral lower lobe densities, consolidation, atelectasis, and rarely pneumatocele and pneumothorax

- Chest X-ray finding correlate poorly with clinical symptoms

- Arterial blood gas analysis if available
- Complete blood count may reveal leucocytosis
- Direct tissue inflammation may cause leucocytosis and fever early in the course of ingestion
- Toxicological analysis for hydrocarbons has no value in clinical management

2.6 Treatment

- Emergency stabilization (refer Section F, Chapter 1, 1.4)
- Keep all the cases under observation in the emergency department
- Whom to admit? (refer box 3.3)
- Decontamination
  - Skin: Remove all clothing and wash thoroughly with soap and water
  - Eyes: Rinse with normal saline
  - Gastric lavage and usage of activated charcoal are contraindicated in hydrocarbon ingestion because procedure increase the risk of aspiration.
  - Gastric lavage may however be indicated in special situations
    - Large volume ingestion
    - Hydrocarbon mixed with another poison
    - Toxin with potential systemic toxicity (halogenate0

- In these cases it should be done only after airway is protected adequately with cuffed endotracheal tube

- Supportive treatment is more important as there is no specific antidote for hydrocarbon ingestion

  - Maintaining SpO2>94 with supplemental oxygen is the priority
  - Children not responding to supplemental oxygen may be started on CPAP or invasive ventilation as indicated
  - Bronchospasm can be treated with aerosolized β2 agonist.
  - No proven benefit of corticosteroids

- Antibiotics are not routinely indicated in hydrocarbon poisoning in the first 48 hours. Persistent or new onset fever at 48 hours may warrant antibiotics for a superadded secondary infection. This cover should contain an anti

Box 2.3 Indications of admission

- Symptomatic child
- Worsening symptoms over the 6 hours observation period
- Sings of toxicity: hypoxia, altered mental status
- Abnormal CXR
- Suicidal intent
- Asymptomatic child who has received lavage outside

2.7 When to discharge?

- Asymptomatic or mildly symptomatic patients who remain stable over the period of 6 hrs of observation can be safely sent home.
- Parental education on proper storage of hydrocarbon products and adequate child proofing is of paramount importance to prevent further episodes of exposure

2.8 When to refer?

- Worsening hypoxemia or respiratory failure requiring ventilation
- Persistent encephalopathy
- Multi organ dysfuntion

2.9 How to refer?

- Secure airway, breathing and circulation
- Ensure resuscitation kit and a BLS trained provider during transport
- Child's peripheral venous access should be secured and patent
- Child's details, history, examination findings, vitals, events which occurred and treatment given should be legibly written in the referral letter
- The parents should be counselled regarding the child's condition and need for referral
- Written, informed consent should be taken from the parents'/guardian's prior to referral

3. ORGANOPHOSPHATES POISONING

3.1 Learning objectives

After completion of this section the participants should be able to

- Identify child with suspected organophosphorus poisoning
- Steps in emergency stabilization
- Decontamination and antidotes
- Know when and how to refer?

3.2 What are different organophosphorus poision?

- Organophosphates are the most widely used insecticide in agriculture, gardens, and veterinary practice.
- Different organophosphorus compounds are chlorpyrifos, cabonphenothion, endotion, malathion, and triamiphos

3.3 What is the mechanism of action of organophosphorus compounds?

- They exert their toxic effects by long-lasting inhibition of hcolinesterases at many sites in the body.
- This results in accumulation of acetylcholine in the synapses and neuromuscular junctions, leading to overstimulation of cholinergic (i.e. muscarinic and nicotine) receptors.
- Hence spectrum of CNS, autonomic nervous system and neuromuscular symptoms are seen depending on the type of receptor affected

3.4 How to identify child with suspected organophosphorus poisoning?

- Circumstantial evidence of exposure
- Playing outside in the fields just freshly sprayed with pesticides
- Farmer's family with pesticides at home
- Licking coloured powder smeared on floor/fields.
- Identification of the toxidrome (refer box 3.1)

Box 3.1 Organophosphorus toxidrome

- Muscarinic manifestations: excessive secretions

(Mnemonic SLUDGE or DUMBELS)

Salvation, sweating	Diarrhoea
Lacrimation	Urination
Urination	Miosis
Defecation	Bradycardia, bronchospasm, bronchorrhea
Gut nausea	Emesis
Emesis	Lacrimation
Salivation	

- Nicotinic manifestations: fasciculations, cramps, weakness respiratory failure (diaphragmatic weakness). Weakness of

neck flexors is an early sign of significant muscle weakness and may be useful in prediction onset of respiratory failure
- Central: Central anxiety, restlessness, confusion, emotional lability, ataxia, tremors, seizures, coma

3.5 Treatment

- Emergency stabilization (refer Section F, Chapter 1: 1.4)
- Decontamination
  - Skin: Remove all clothing and wash thoroughly with soap and water
  - Eyes: Rinse with normal saline
  - Gastric lavage: If child presents within 1-2 hours of exposure, and after adequate airway protection. Repeat every 2-3 hourly return of clear and odourless fluid
- Specific Antidote:

a. Atropine

- Give 0.05mg/kg IV every 10 minutes tills signs of atropinisation appear (refer Box 2.2)
- Then give 10-20% of total atropinisation dose as hourly infusion for 24-48 hours and withdraw gradually.

b. Pralidoxime (PAM)

- Recycles acetylcholinesterase enzyme
- Should be used in patients with nicotine receptor manifestations
- Should be used early before irreversible inhibition of acetylcholinesterase enzyme occurs
- 30 mg/kg intravenous loading dose over 10-20 minutes followed by 8-10 mg/kg/hr infusion till clinical recovery
- Continue up to 12-24 hours after atropine is no longer required

Box 3.2: End-points of atropinisation
- Reduced secretions
- Clear chest on auscultation (most important)
- Dry axilla and oral mucosa

*Tachycardia & mydriasis should not be considered as the end-points

3.5 When to refer?
- When facilities for monitoring are not available
- When mechanical ventilation is required

3.6 How to refer?

- Initial stabilization and decontamination to be done at receiving facility before referral
- Peripheral venous access to be secured
- Clinical details and treatment given to be legibly noted on referral slip
- Person trained in resuscitation to accompany the child while referring

Algorithm for Organophosphorus poisoning

Suspected organophosphate poisoning
(History of exposure with suggestive clinical features)

Emergency stabilization
ABCD3E

Decontamination

Establish 2 IV cannula, take samples for complete blood counts, serum electorlytes, renal functions test and plasma cholinesterase levels, if available

Specific antidote: Atropine
Give 0.05mg/kg IV every 10 minutes till signs of atropinisation appear (Box 3.2)
Then give 10-20% of total atropinisation dose as hourly infusion for 24-48 hours and withdraw gradually

If atropine toxicity (tachycardia, hyperthermia, ileus) → taper infusion

Pralidoxime (PAM): recycles acetylcholinesterase enzyme 30mg/kg intravenous loading dose over 10-20 minutes followed by 8-10 mg/kg/hr infusion till clinical recovery
(up to 12-24 hours after atropine is no longer required)

Consider discharge if stable for 24 hours after stopping oxime

4. SNAKE ENVENOMATION

4.1 Learning Objectives

After completion of this section, the participants should be able to:

- Identify a case of snake envenomation
- Differentiate between non-venomous and venomous bites
- Know the syndromic approach to envenomation
- Provide on the scene first aid management
- Initial stabilization and resuscitation
- Specific management with antivenin
- Supportive management
- Know when and how to refer precautionary measures to take before referral

4.2 Snake bites

- The 4 most important venomous snakes in India include India Cobra (Najanaja), Indian krait (Bungaruscaeuleus), Russels vipers (Daboiarusselii) and saw scaled viper (Echiscarinatus).
- Many bites are inflicted by non-venomous species and may be mistaken for venomous species resulting in unnecessary, expensive, risky and wasteful anti venom treatment.
- Also about 50% of venomous snake bites are DRY BITES and do not result in envenomation

4.3 How to differentiate between venomous and non-venomous snakes? (refer box 4.1)

Venomous	**Non venomous**
• Triangular shaped head, neck is narrower than the head • Pits on head • Elliptical pupils • Fangs present • Undivided anal plate	• Head is rounded and more continuous with body • No pits on head • Round pupils • No fangs • Divided anal plate

4.4 Different types of venomous snakes: (Refer Box 4.2)

Box 4.2: Types of venomous snakes

Elapidae	Viperadae
• Cobra and kraits • Short fixed fangs • Large smooth symmetrical scales on dorsum head • Cobras, raise a hood • Neurotoxic	• Vipers • Longer fangs • Flat against upper jaw, erected on strike • Small rough scales on dorsum • Colored marking • Vasculotoxic

4.5 How to clinically identify a venomous snake bite?

A. Neurotoxic envenomation (predominant neuroparalytic manifestations)

Box 4.3 Features of neurotoxic envenomation

<1hr	1-3 hrs	>3hrs
•Headache •Nausea •Vomiting •Abdominal pain	Ptosis Diplopia External ophthalmoplegia ▪Dysphonia ▪Dysphagia ▪Myopathic facies	paralysis of trunk and limbs Respiratory failure Shock Hypoxemia Cyanosis

- How to differentiate between a kraitand cobra bite? (refer box 4.4)

Box 4.4: Difference in krait and cobra bite

Points of difference	Krait	Cobra
Circumstances of bit	Indoor	Outdoor
Time	Nocturnal	Daytime or nocturnal
Local reaction	Minimal/no	++++
Onset and progression	Rapid	Slow
Mechanism of action	Presynaptic blockade	Post synaptic blockade

B. Vasculotoxic envenomation (predominant hemorrhagic manifestation):

Box 4.5: Features of vasculotoxic envenomation

Local signs	Spontaneous systemic bleeding
• Fangs marks • Local pain & bleeding • Bruising • Lymphangitis (within 1hr) & lymph node enlargement • Swelling, blistering • Local infection, necrosis, abscess formation • Traumatic bleeding from recent wounds and old partly-healed wounds	• Skin bleed • Gums • Epistaxis • Intracranial haemorrhage • GI bleed • Hematuria

C. Syndromic approach to snake bit (refer box 4.6)

Box 4.6;

Local swelling + neuroparalysis → Cobra or king cobra

No or minimal local swelling + Neuroparalysis → Krait

Intense local swelling and necrosis with bleeding → Viper

4.6 Management guidelines:

A. At the scene of snake bite:

Box 4.7: First Aid – Do's and Don'ts

Do's	Don'ts
• Reassure and calm the victim • Immobilize the patient, especially the bitten limb, as any movement increases the venous and lymphatic absorption of the venom • Accelerated transport to medical care, ideally in the recovery position • Monitor the perfusion of the limb regularly. • The tourniquet should be tight enough to block venous drainage and not block the arterial supply of the limb	• No incision on the bite site • No sucking of the venom • No tight tourniquet • Avoid interference with the bite wound • Don't try to get the live/dead snake to the hospital as it may still be venomous – endangering other healthcare professionals.

B. Specific management after reaching a hospital: Rapid clinical assessment and resuscitation: The ABCDE approach

- Airway: Maintain open and stable airway
- Breathing: Check for respiratory movements and need for oxygen/ventilation
- Circulation: Check hemodynamic parameters and establish I.V access
- Disability; Check the level of consciousness/ptosis/neuroparalysis
- Exposure: Examine the local site, remove tight tourniquets after antivenin
- Species diagnosis: Expert identification of the snake by mobile phone picture, infer species from patient's description, circumstances of the bite9 nocturnal bite while sleeping on ground indicates Krait bite) or by a clinical syndrome.

4.7 Investigation:

- 20 minutes Whole Blood Clotting Time (WBCT);
  - Requires new, clean, dry, ordinary glass tube, bottle, vial or syringe
  - Positive test (non-clotting) indicates severe consumption coagulopathy and need immediate antivenin treatment.
  - Blood; Coagulogram, complete hemogram, Renal Function tests, Electrolytes
  - Urine for haemoglobin, Myoglobin, proteinuria or RBC

4.7 Antivenom administration:

A. Antivenom is the only specific antidote for snake vemom. It should be given only if indicated clinically and to patients in whom its benefits are considered likely to exceed its risks. Since it is costly and often in limited supply, it should not be used indiscriminately.

B. Indications of antivenom: Box 4.8

Systemic signs	Local signs
• Hemostatic (bleeding), WBCT positive • Neurological • Ptosis, opthalmoplegia, paralysis • Hypotension, shock, acute kidney injury, hemoglobinuria, dark brown urine • MODS	• Local swelling involving more than 50% of bitten limb withing 48 hours • Swelling of digits • Rapid extension of swelling within few hours • Development of enlarged tender lymph nodes draining bitten limb.

C. How much to give?

- Initially start with 10 vials and administer over one hour
- Criteria for repeating the dose
    - Repeat dose in neurotoxic envenomation is based on 1-2 hours rule
    - Deteriorating neurotoxic sign beyond 1 hour.
    - Bleeding beyond 1-2 hrs.
    - Maximum recommended dose is 20 vials.
    - Repeat dose in vasculotoxic is based on 6 hours rule
    - Repeat 20 minute –WBCT after 6 hours in uncoagulable
    - Generally no benefit after 20 vials

D. How to administer ASV?

- Snakes inject same dose of venom into child & adult hence ASV dose is same in all
- It is given as intravenous infusion only.
    - Reconstituted ASV diluted in 5-10 ml/kg of DNS
    - IV infusion over 1 hour
- No test dosing is recommended

E. Reactions to antivenom:

- Early (within few hours) or late (5 days or more).
- If reaction occurs:
  - ASV must be temporarily suspended
  - Adrenaline 0.1% solution (1 in 1000) 0.1 ml/kg is given intramuscularly.
  - Hydrocortisone (2mg/kg, IV) and antihistaminic can be given

F. When will ASV response be seen?

- Spontaneous bleeding usually stops within 15-30 min
- 20 minute – WBCT normalize usually in 3-9 hours
- Patient in shock: BP may increase in 30-60 min and arrhythmias may resolve

4.9 Monitoring

- Child should be checked by nurses every two hourly and by doctor at least 6 hrly
- Monitor Sensorium and vitals along with local site mapping

4.10 Supportive Care

- A trial of anticholinesterase should be given in every patient with neurotoxic envenomation.
  - Patients with cobra bite (post synaptic neuromuscular blockade) will show improvements.
- I.M Neostigmine (0.04 mg/kg/dose) followed by IV Atropine (0.05 mg/kg0 is given and response observed over the next 30-60 minutes. If response is seen, the same doses are repeated every 30 minutes, for 8 hours.
- Acute kidney injury
  - Correct hypovolemia, severe acidosis and promote alkaline diuresis until there is evidence that rhabdomyolysis is decreasing.
  - Indications of Dialysis: Uremia, fluid overload, metabolic acidosis.
- Wound management and fasciotomy in case of compartment syndrome

(refer box 4.9)

Box 4.9 Compartment Syndrome

When to suspect compartment syndrome?	• Disproportionately sever pain • Weakness of intracompartmental muscles • Pain on passive stretching of intracompartmental muscles • Hypoaesthesia of areas of skin supplied by nerves running through the compartment • Obvious tenseness of the compartment on palpation
Treatment of choice	Early administration of anti-venom (prevents necrosis of muscles in the compartment)
Criteria for fasciotomy	After haemostatic abnormalities have been corrected (anti-venom+/- FFP) • Clinical evidence of compartment syndrome • Intra-compartmental pressure> 30 mm Hg

4.11 When to refer?

- Patient with respiratory failure requiring ventilation
- Multiorgan dysfunction requiring intensive care e.g. dialysis
- Non availability of antivenom

4.12 How to refer?

- Most important point during referral is immobilization of the limb and giving ASV prior/during transport.
- Secure airway, breathing and circulation
- Child's peripheral venous access should be secured.
- Child's details, history, examination findings, vitals, events occurred and the treatment given should be legibly written in the referral letter.
- Child's parents should be counselled regarding patient's condition
- Ensure resuscitation kit and a BLS trained provider through transport

Remember:

- First aid: Do it RIGHT
  - R. : Reassure
  - I.:Immobilize, like you would, a fracture
  - G.H.: Get a hospital
  - T.: Tell the doctor about any symptoms
- Heparin has no role and can increase bleeding
- Botrophase not to be used

APPROACH TO A CASE OF TROPICAL FEVER

1.1 Learning objectives:

After completion of this section, participants should be able to

- Assess and identify a case of tropical fever
- Classify them into syndromic patterns
- Identify the physiological impairment and MODS
- Initiate stabilisation and broad empirical cover
- Know the point of care testing that should be ordered
- Initiate specific therapy based on etiological diagnosis
- Counsel parents regarding home management in non severe cases
- Know when and how to refer?

1.2 What are tropical fevers?

- Tropical fevers are defined as infections prevalent in, or unique to tropical and subtropical regions
- Most of these infections cluster during monsoon and post monsoon period
- Most are vector borne and transmitted by an insect bite
- There is a significant overlap in clinical presentation of these infections making it difficult to arrive at a specific etiological diagnosis
- Hence simplified syndromic approach in an emergency setting is better than and individual disease approach
- This strategy helps in timely stabilisation and management.

1.3 How to identify a case with tropical fever?

- Any febrile child presenting to emergency department with the following
  - Duration of fever <2weeks
  - Most of them will cluster around rainy and post rainy season
  - Non-specific symptoms: headache, myalgia, arthralgia, retro-orbital pain, photophobia
  - Skin rash
  - Multiorgan involvement including encephalopathy, seizures, jaundice hepatosplenomegaly, respiratory distress and renal failure.
  - No specific localisation to a single organ
- Common tropical infections include
  - Malaria
  - Dengue

- Scrub typhus
- Leptospirosis
- Typhoid

1.4 How to approach a case of suspected tropical fever?

- The primary focus should be on identification of physiological impairments and stabilizing them
- Identify the presence of associated symptoms along with fever
- Classify the constellation into a syndrome to guide initial empiric therapy in a critically ill child (refer box 1.1)
- Remember children with MODS like encephalopathy, rash, thrombocytopenia, ARDS, jaundice, bleeding and acute kidney injury need urgent treatment
- Proceed with first line point of care investigation (refer box…….)
- Initiate a broad empirical cover while awaiting laboratory results.
- Depending on the result proceed with further management.

Box 1: Syndromic approach to tropical fevers in emergency room

Fever with rash/thrombocytopenia	Dengue Malaria Rickettsial infections Typhoid Leptospirosis
Fever with encephalopathy	Scrub typhus Cerebral malaria Typhoid encephalopathy Meningitis Encephalitis
Fever with ARDS	Scrub typhus P. Faliciparum malaria Leptospirosis Influenza (H1N1)
Fever with jaundice	Malaria Leptospirosis Dengue
Fever with multiorgan dysfunction	Falciparum malaria Leptospirosis Scrub typhus Dengue Bacterial sepsis

Box 2: Point of care test in suspected tropical fevers

Box 1.2: Baseline investigations

- Complete blood count with peripheral smear
- Serum electrolytes with renal function test
- Liver function test
- Urine analysis
- Rapid diagnostic test
  - Malaria card test
  - Dengue card test
  - Dengue NSI antigen

Malaria

- Suspect malaria in any case of tropical fever spectrum with pallor, hepatosplenomegaly, multiorgan involvement
- Confirm the diagnosis with either microscopy or rapid diagnostic test
- Rule out malaria if two negative RDT
- Identify any signs of severe malaria (refer box 3)
- Patient with no features of severe malaria is defined as having uncomplicated malaria
- Treatment of uncomplicated malaria is ACT for 3 days
  - Artemether + Lumefanthrine
  - Artemether + amodiaquine
  - Artemether + mefloquine
  - Artemether + sulfadoxine – pyrimethamine: is safe and effective for uncomplicated P. Falciparum malaria.
  - Chloroquine is safe and an effective for uncomplicated P. vivax malaria.

Box 3. Features of severe malaria

Features of severe malaria

- Multiple convulsions>2 in 24 hours
- Impaired consciousness
- Severe anemia, Hb<5g/dl
- Hypoglycemia, RBS <60mg/dl
- Metabolic acidosis, HCO3<15mmol/l or base deficit>8 meq/l
- Acute renal failure (serum creatinine>3mg/dl)
- Jaundice (serum bilirubin>3mg/dl)
- ARDS
- Shock (“algid malaria)
- DIC
- Hemoglobinuria
- Hyperparasitemia(>5%)

Treatment of severe malaria

1. Stabilize ABCDE:

- Start supplemental oxygen for children with sever pallor, tachypnea and/or shock
- Those with impaired (GCS≤8) on, ALI/ARDS, shock, pulmonary edema, and require endotracheal intubation and mechanical ventilation
- Management of septic shock follows standard guidelines using fluids and vasoactive drugs. Fluid boluses should be used with extreme caution (due to severe anemia) and only if there is frank hypotension.
- Adequate seizure control and management of raised intracranial pressure in cerebral malaria.
- Rapid detection and correction of hypoglycaemia is extremely important. A bolus (5ml/kg) of 10% dextrose solution be given by a rapid IV push. RBS should be rechecked after 30 minutes. It is important to give 10% dextrose in normal saline or Ringer's lactate for maintenance infusion to prevent hypoglycaemia. Routine blood glucose monitoring is mandatory.
- Urgent blood transfusion is indicated if haemoglobin is below 7g%.
- Children presenting with high grade fever and shock should receive IV broad- spectrum antibiotics to cover for coexistent bacterial sepsis.

2. Specific treatment:

- Two important groups of antimalarial drugs are available – artemisinin derivatives (artesunate, artemether), and cinchona alkaloids, (quinine, quinidine, chloroquine).
- Artemisinin derivatives are more effective, simpler, and safer than cinchona alkaloids.
- For pre-referral administration, intramuscular (IM) artesunate is more effective than rectal artesunate In children > 6 years, rectal artesunate is not recommended.
- The combination drugs available in artemisinin combination therapy (ACT) are given in box 4.
- The specific therapy of malaria is summarized in box 5.

Box 4: Combination drugs used in Artemisinin combination therapy (ACT)

- Artesunate + sulfadoxine – pyrimethamine
- Artesunate + amodiaquine
- Artesunate +lumefantrine
- Dihydroartemisinin + piperaquine
- Artesunate + mefloquine (avoided in cerebral malaria)
- Artesunate + doxycycline or clindamycin

- Drug of choice : Artesunate
  Dose: 3 mg/kg/dose in children < 20 kg : 2.4 mg/kg/dose in children> 20 kg
  ACT must be given parenteral for at least 24 hours\
  Once patient is able to take orally complete treatment with 03 days of ACT

Box 5:	Uncomplicated	Severe
P vivax	Areas With Chloroquine Or ACT Areas With Chloroquine Resistance – ACT Primaquine 0.25 -0.5 mg/kg/d for 14 d Except in < 6m & know G6PD deficiency G6PD deficiency: 0.75 mg/kg/w for 8 w G6pd status unknown 7 unavailable – individualize	ACT + Primaquine
P Falciparum	ACT + single dose of primaquine in low transmission areas (0.25 mg/kg – no G6PD testing required	ACT + single dose of primaquine in low transmission areas (0.25 mg/kg – no G6PD testing required
Mixed	ACT + Primaquine (as for vivax)	ACT + Primaquine (as for vivax)
Not know	As for uncomplicated P falciparum	As for complicate P falciparum

3. Management of complications

- Cerebral edema: Management is largely supportive and includes treatment of raised ICP, and maintenance of euthermia and euglycemia. Short term hyperventilation to achieve PCO2 ~ 30mm Hg can be done if signs of impending herniation are present.
- Hyperparasitemia and role of exchange transfusion: Exchange transfusion (ET) rapidly reduces the parasitic index (PI) and mortality. It may be used in complicated malaria with Hyperparasitemia or multiorgan dysfunction which fail to respond to chemotherapy alone, preferably in and intensive care setting.
- Acute kidney injury: Loop diuretics can convert an oliguric to non-oliguric renal failure and reduce the risk of volume overload. Presence of AKI or severe acidosis unresponsive to fluids is and indication fore renal replacement therapy.

SCRUB TYPHUS

- Suspect scrub typhus in any case of tropical fever spectrum with rash, hepatosplenomegaly, encephalopathy or multiorgan involvement.
- An eschar, when present is a valuable clue to the diagnosis and should be meticulously looked for in every child.
- As it is a potentially fatal but relatively easy to treat, keep a low threshold for empirical treatment.
- Confirm the diagnosis with IgM ELISA.
- Identify organ involvement and need for organ support
- Drug of choice is doxycycline.

1. **Stabilize ABCDE:**

- Start supplemental oxygen for children with tachypnea and/or shock
- Those with persistent hypoxemia and/or severe encephalopathy (GCS≤8) require endotracheal intubation and mechanical ventilation
- Management of septic shock follows standard guidelines using fluids and vasoactive drugs
- Adequate seizure control and management of raised intracranial pressure is crucial

2. **Specific therapy:**

- Currently, doxycycline remains the first choice even for children younger than 8 years
- Early treatment with doxycycline (5mg/kg/day in 2 divided doses, IV or oral shortens the disease course and reduces mortality

- Duration of therapy
    - 3-5 days after defervescence in uncomplicated cases
    - 7-14 days ins severe scrub typhus with organ failure
- Azithromycin (10mg/kg/day for 07 days) is an effective alternative

3. Management of complications

- Complications include ARDS, septic shock, myocardotos. Amd encephalopathy and raised intracranial pressure.
- Acute respiratory failure due to ARDS responds well to non-invasive ventilation (NIV) if started early. In delayed presentation, intubation and ventilation will be required.
- Myocardial dsyfuntion should be actively looked for (if possible) in all children with fluid –refractory shock. Addition of an inodilator like dobutamine or milrinone may be required to improve cardiac output. Continuous EKG monitoring is desirable to detect arrhythmias.
- Children with acute encephalitis syndrome require seizure control, sedation and analgesia and management of raised intracranial pressure (ICP_
- Renal replacement for Acute Kidney Injury (AKI) may be required in small proportion of patients.
- Platelet transfusion is required in symptomatic thrombocytopenia. Platelets recover with antiobiotics and defervescence.

4. Acute encephalitis

- Acute encephalitis is a medical emergency where stabilization, management of comlications, investigations and specific treatment all need to be performed simultaneously.
- Airway stabilization and hemodynamic support are the initial priorities.
- Raised ICP and seizures/status epilepticus are the 2 main complications that need aggressive management.
- Lumbar puncture is essential and should be performed after initial stabilization unless contraindicated.
- Neuroimaging is not required in all cases.
- Dengue fever, scrub typhus and cerebral malaria can present like an acute encephalitis's syndrome.
- Identifying treatable causes and empirically start therapy is essential.

1. **Stabilize ABCDE:**

- Assessment of airway and stabilization
- Hemodynam assessment and resuscitation if needed
- Assessment of level of consciousness using a quantitative scale like GCS. GCS below 8 and clinical signs of raised ICP are indications for rapid sequence intubation and ventilation.
- Management of complications like seizures and raised intracranial pressure (ICP).
- Correct reversible factors like hypoglycaemia, hypo or hypernatremia and hypcalcemia

2. Specific therapy:

- Empiric treatment pending neuroimaging and/or lumbar puncture includes antibiotics for bacterial meningitis and acyclovir for herpes simplex viral (HSV) ncephalitis.
- Remember that other tropical infections like dengue, malaria and scrub typhus can also present like an "encephalitis syndrome" and may need empiric treatment (Box.…)
- Hsv is the most common cause for sporadic encephalitis and one which is potentially treatable. When to give and when not to give acyclovir are enumerated in Box 6.

Box 6. When to give and when not to give acyclovir

Indications for Acyclovir

- Acyclovir should be initiated in all patients with suspected encephalitis, pending results of diagnostic studies.
- Fever with progressive deterioration of consciousness, focal seizures or focl neurological abnormalities in the absence of any other cause
- Dose: 20mg/kg/dose TDS as 1 hour infusion

When not to give acyclovir

- Child with simple febrile convulsions
- Seizures without documented fever or history of fever (unless immunocompromised)
- Other obvious cause for symptoms, e.g. blocked VP shunt, epilepsy (exacerbation with febrile illness)
- Acute head injury, drug overdose
- CSF and clinical picture are highly suggestive of bacterial meningitis

Box 7: Empirical therapy in acute encephalitis syndrome

Antibiotics	Ceftriaxone
Antivirals	Acyclovir
Antimalarials	Artesunate
Others	Doxycycline Azithromycin

Management of complications:

The most important neurological complications of encephalitis are raised ICP and status epilepticus, therefore supportive management is needed for both. Patient positioning prevention of bed sores, nutrition and physiotherapy should be meticulously taken care off.

Dengue Fever

- Suspect dengue fever in all tropical fevers with rash, features of capillary leak or multiorgan dysfunction.
- Rapid WHO approved caerd tests using a combination of NS-1 antigen and Igm antibody have food sensitivity and specificity.
- Fluids are the mainstay of therapy and normal saline is the fluid of choice.
- Colloids may be needed in some cases.
- Therapeutic endpoints ofr dengue shock are different from those of septic shock
- Titrate fluids with close monitoring of hemodynamic parameters and haematocrit.
- Identifying occult bleeding is important and blood transfusion may be life saving
- All other transfusions and procedures may be more harmful than beneficial.
- Titrate therapy based on natural course of illness; overdoing in terms of fluids, drugs and transfusions can be fatal.

1. Stablize ABCDE:
   - Start supplemental oxygen for children with tachypnea and/or shock
   - Circulation should be classified as hemodynamically stable with danger signs, compensated shock (hypoperfusion with normal blood pressure), and hypotensive shock as this will decide further management.
   - Supportive therapy with supplemental oxygen and initial evaluation with a chest X-ray should be done.

Fluid resuscitation

(i) hemodynamically stable with danger signs:

- Start IV crystalloids, normal saline (NS) or Ringer's lactate (RL) at 5-7 ml/kg/hr for 1-2 hours.
- Titrate fluids according to clinical improvement and HCT change.
- Clinical improvements and decreasing HCT, fluid rate should be decreased to 3-5 ml/hr for 2-4 hours and then 2-3 ml/hr
- Clinical worsening and increasing haematocrit, fluid rate should be increased to 5-10 ml/hr
- Clinical worsening and falling haematocrit, suspect bleeding, which can be a occult gastrointestinal haemorrhage. Urgent blood product transfusion will be required in such cases.

(ii) Compensated Shock:

- Start on NS or RL at 5-10 ml/kg over one hour.
- If no improvements and static or increasing haematocrit, start on IV bolus of RL/NS – 10-20 ml/kg over 1 hour.
- If there is no improvement, give another bolus of crystalloid of colloid.
- Non response to 40 ml/kg of NS/RL should warrant check for haematocrit and occult bleed. Blood transfusion is indicated if there is fall in haematocrit without clinical improvement.
- Fluids should be tapered as soon as child is clinically improving and shock is passive.

(iii) Hypotensive shock:

- Rapid crystalloid 20 ml/kg over 15 minutes under close monitoring
- If no improvement give second bolus preferably colloid 10-20 ml/kg over 30 minutes to 1 hours which can be repeated if need be
- Bleeding should be rules out
- In fluid non-responders, suspect underlying myocardial dysfunction (diastolic or systolic). Initiate inotropic support and manage shock guided by serial HCT and echocardiography (if available).
- In persistent fluid refractory shock, actively look for occult severe bleeding, metabolic derangements (acidosis hypoglycaemia, hypocalcemia), myocarditis and cardiac dysfunction, abdominal compartment syndrome, obstructive shock due to massive pleural/pericardial effusion and coexistent bacterial sepsis.

(iv) Indications for blood transfusion:

- Shock non-responsive to 40-60 ml/kg fluids

- Fall in haematocrit associated with hemodynamic instability
- Hypotensive shock with low/normal HCT
- Persistent or worsening metabolic acidosis along with abdominal tendereness or distension.
- In the above situations, 5-10 ml/kg of fresh packed red cell or 10-20 ml/kg of fresh whole blood can be given. Current literature does not support prophylactic platelet transfusions for thrombocytopenica; platelet transfuisons may be considered for children with severe bleeding. FFP may be considered in children with significant coagulopathy.

2. Specific treatement:

There is no specific treatment for dengue fever. Supportive therapy is the mainstay of management.

Management of complications

- Fluid overload: Children with dengue are predisposed to fluid overload due to excessive fluid administration during leaky phaseuse of hypotonic fluids during resuscitation resorption of fluid from extravascular to intravascular space at end of critical pahse fluid rate should be decreased timely once hemodynamic stability is achieved to prevent overload.

Treatment of established fluid overload includes fluid restriction, respiratory support in form of continuous positive pressure and removal of excess fluids by diuretics or continuous renal replacement therapy (CRRT), hemodialysis (HD) or peritoneal dialysis. CRRT is preferred because it causes mininmal hemodynamic instability. One has to be very cautious with diuretics during active capillary leakage as it can further worsen intravascular hypovolemia.

- Neurological complications Seizures and encephalopathy are commonest neurological manifestation and their treatment is primarily supportive
- Renal Failure: Renal replacement therapy modes useful during acute infection are CRRT, PD and HD. Bleeding diathesis complicate utilization of anticoagulation in CRRT and HD.
- Abdominal compartment syndrome: Excessive third spacing, fluid overload, intra-abdominal bleed can contribute to intra-abdominal hypertension (more than 10 mm Hg in children) and abdominal compartment syndrome (ACS). This can lead to worsening renal function, shock and respiratory distress. Management includes treatment of fluid overload with diuretics or

renal replacement, gastrointestinal decompression through nasogastric tube and flatus tube and in sever cases peritoneal drainage.

- Respiratory failure: Early positive pressure support though non invasive modes will be helpful.

ENTERIC FEVER

- Suspect enteric fever in any case of tropical fever spectrum with hepatosplenomegaly, PUo or multiorgan involvement
- Confirm the diagnosis with blood culture
- Common complications are gastrointestinal an dneurological.
- Decide on ambulatory vs. hospital based management.
- Accordingly, oral cefixime or IV 3rd generation cephalosporin is the drug of choice.

1. Stabilize ABCDE:
   - Children with persistent vomiting, abdominal distention, and poor oral acceptance or severe diarrhoea or any complications must be hospitalized
   - Start supplemental oxygen for children with tachypnea and/or shock
   - Those with persistent hypoxemia and/or severe encephalopathy (GCS≤8) require endotracheal intubation and mechanical ventilation
   - Management of septic shock follows standard guidelines using fluids and vasoactive drugs

2. Specific therapy

- Most patients with uncomplicated disease improve with home-based treatment comprising appropriate oral antibiotics, hydration, and supportive care.
- Parenteral antibiotics are indicated in children with persistent gastrointestinal symptoms, poor oral acceptance and those with systemic complications.
- Third –generation cephalosporins are the first line antibiotics of choice for hospitalized patients. Parenteral antibiotics should be given for a period of at least 5 days after defervescence or for a total duration of 14 days.
- Oral cefixime (20mg/kg/d) is recommended for ambulatory patients.
- For uncomplicated quinolone sensitive strains, defixime or fluoroquinolones are the initial antibiotics of choice

- Alternatively, azithromycin can be used in cases of strains resistant to cephalosporins.
- Multi drug resistant strains should be suspected in children presenting with short duration of illness, serious complications, failure to respond to first line antibiotics or a known household contact or during an apidemic of MDR typhoid fever.
- Children with enteric fever have severe anorexia along with spiking fever, necessitating adequate hydration, liberal use of antipyretics and early resumption of nutrition.

3. Treatment of complication:

- Indications for PICU admission include:
- Hemodynamic monitoring for shock
- Neurological monitoring for encephalopathy
- Monitoring for abdominal complications
- Fluid and electrolyte balance
- Bleeding diathesis die to thrombocytopenia
- Disseminated intravascular coagulation and multiorgan failure

Organ supportive therapy

- Children presenting with shock or enteric encephalopathy should be treated with dexamethasone, initial dose of 3mg/kg followed by 1 mg/kg every 6 hours for total 8 doses.
- Intestinal haemorrhage requires intensive monitoring, volume resuscitation and blood transfusion.
- Patients with suspected intestinal perforation should be stabilized first followed by urgent surgical intervention.
- Secondary peritonitis with E.coli and Klebsiella is common after perforation and may lead to secondary organ dysfunction.
- Acute respiratory distress syndrome requires mechanical ventilator support.
- Myocarditis presenting as cardiogenic pulmonary edema and/or shock with ST segment, and T wave changes requires supportive care including ventilation, fluid restriction and inotropic support.

When to refer a child with suspected tropical fever?

- When child appears sick or toxic and there are signs of hemodynamic instability.
- When additional assistance for diagnosis and management for any of the diseases is required.

How to refer?

- Child's peripheral venous access should be secured
- Child's details history, examination findings, vitals, events occurred and the treatment given should be legibly written in the referral letter.

PEDIATRIC HIGHER CENTER TRANSFER GUIDELINES

1.1 Learning objectives.

After completion of this section the participants should be able to

- Recognize clinical conditions that require referral to higher centre for further care
- Identify the nearby higher center which is appropriate for management of such a child
- Outline need for referral
- Enlist perquisites for referral
- Maintain transport checklist
- Pre-transport stabilization of ABCD

1.2 Why is transport to higher center required?

- Sometimes critically ill patients cannot be managed at resource limited setting due to inadequacy of available resources
- To overcome this gap in management transfer to higher centre is required
- The outcome is best when this transfer is done by adequate planning and careful risk benefit analysis

1.3 Points to considered while deciding transfer to higher centre

- Urgency of transfer
- Benefit of transfer
- Risk involved in transfer
- Child's clinical status before transfer and his/her ability to sustain transfer
- Child should be adequately assessed, resuscitated and optimal stabilized before transfer

1.4 What are the perquisites for transfer?

- Communication and coordination between the referring and referred team (centre where patient will be transferred)
- Appropriate equipment for monitoring and intervention if required
- Trained personnel to deliver life sustaining treatment during transport

1.5 How to ensure proper communication and co-ordination?

- Contact the referred facility doctor and confirm availability of resource (e.g. ventilator, CT) for which patients needs to be the transferred
- Confirm availability of bed for the patient
- Brief the doctor regarding patient history and present clinical status
- Summarize the treatment given along with all the relevant laboratory and radiological reports (attach copy with transport form)

1.6 What are the conditions requiring higher centre transfer? (refer box 1.1)

Box 1.1 indications for Pediatric Transfer/Referrals

• Medical	Trauma	Burns (Thermal or chemical)
• Depressed or deteriorating neurologic status • Status epilepticus • Severe respiratory distress responding inadequately to treatment and accompanied by any one of the following: • Grunting or gasping respirations • Cyanosis • Apnea • Retractions (moderate to severe) • Stridor (moderate to severe0	• Fractures and deep penetrating wounds to and extremity complicated by neurovascular or compartment injury • Fracture to two or more major long bones (i.e. femur, humerus0 • Fracture of the axial skeleton • Spinal cord or column injuries • Traumatic amputation of an extremity with potential for replantation	• and 3 burns>10% of the body surface area in children <10 years • and 3 burns >20% of the body surface area in children >10 yrs • burns > 5% of the body surface area for any age group • Signs or symptoms of inhalation injury • Burns involving face, mouth, throat and • Ears (serious full thickness burns

• Children requiring endotracheal intubation and/or ventilator support • Serious cardiac rhythm disturbance • Post cardiopulmonary resuscitation care • End organ failure (Cardiac, renal, hepatic) Shock responding inadequately to treatment Severe electrolyte imbalances Severe metabolic disturbances Severe dehydration Severe hypothermia or hyperthermia Need for invasive monitoring Arterial pressure Monitoring Central venous pressure Intracranial pressure monitoring	• Head injury when accompanied by any of the following: • Cerebrospinal fluid leaks • Open head injuries • (excluding simple scalp injuries) • Depressed skull fracutures • Significant penetrating wounds to the head, neck, thorax, abdomen or pelvis • Major pelvic fractures • Significant blunt injury to the chest or abdomen	or burns involving the ear canal or drums), Deep burns of the hands, feet, genitalia, major joints, or perineum ○ Electrical injury or burns (including lightning)

1.7 How to stabilize the patient before transportation? (ABCDE approach)

- Airway
  - Maintain the airway patent
  - If child's airway is unstable it is safer to intubate
  - Indications of intubations before transfer (refer box 1.2)
  - Fix the endotracheal tube properly since any change in position can cause displacement in tube and accidental extubation

- Breathing
  - Maintain adequate oxygenation
  - Continuous SPO2 monitoring
  - Insert nasogastric tube in children with ileus and on bag and tube ventilation
- Circulation
  - Insert two large bore IV cannula; if IV access is difficult the secure IO access
  - Assess the need for fluid boluses (refer box 1.3)
  - Label the infusions properly
  - Catheterize every child with hemodynamic instability for strict fluid management.

- Disability
  - Assess the sensorium, pupillary size and its reaction to light
  - Control seizures and raised ICP
  - Adequate sedation and analgesia in intubated patients

- Exposure: Maintain temperature and glucose

Box 1.2 Indications for intubation before transfer

- GCS <8
- Respiratory failure (Spo2<94% despite oxygen therapy with any oxygen delivery devices)
- Unequal pupils, decerebrate posturing
- Fluid refractory or dopamine refractory shock
- Unstable airway (pooling of secretions, severe stridor)

Box 1.3 Indications for fluid bolus

Unexplained tachycardia with no features of CCF
Prolonged CFT, cold peripheries or poor peripheral pulses
Tachycardia with flash CFT, wide pulse pressure with bounding pulse (warm septic shock)
Hypotension: systolic BP<70 + (age x 2)

1.8 Accompanying personnel

Minimum of one doctor and one nurse along with the vehicle operator must accompany a critically ill child

The doctor should be trained in Advanced Life Support skill and should be well versed with emergency procedures like establishment of IV/IO access, endotracheal intubation, suctioning.

He/she should be able to manage any complication during transport like seizures, hypoglycaemia
Similarly and advanced life support trained nurse well versed with emergency management should accompany the doctor

1.9 Monitoring during transport

- Continuous SPO2 and ECG monitoring
- Vitals monitoring at regular intervals
- In intubated patients note the endotracheal tube position and identify any signs of displacement

1.10 Transport Check list

This is required to ensure compliance during transfers

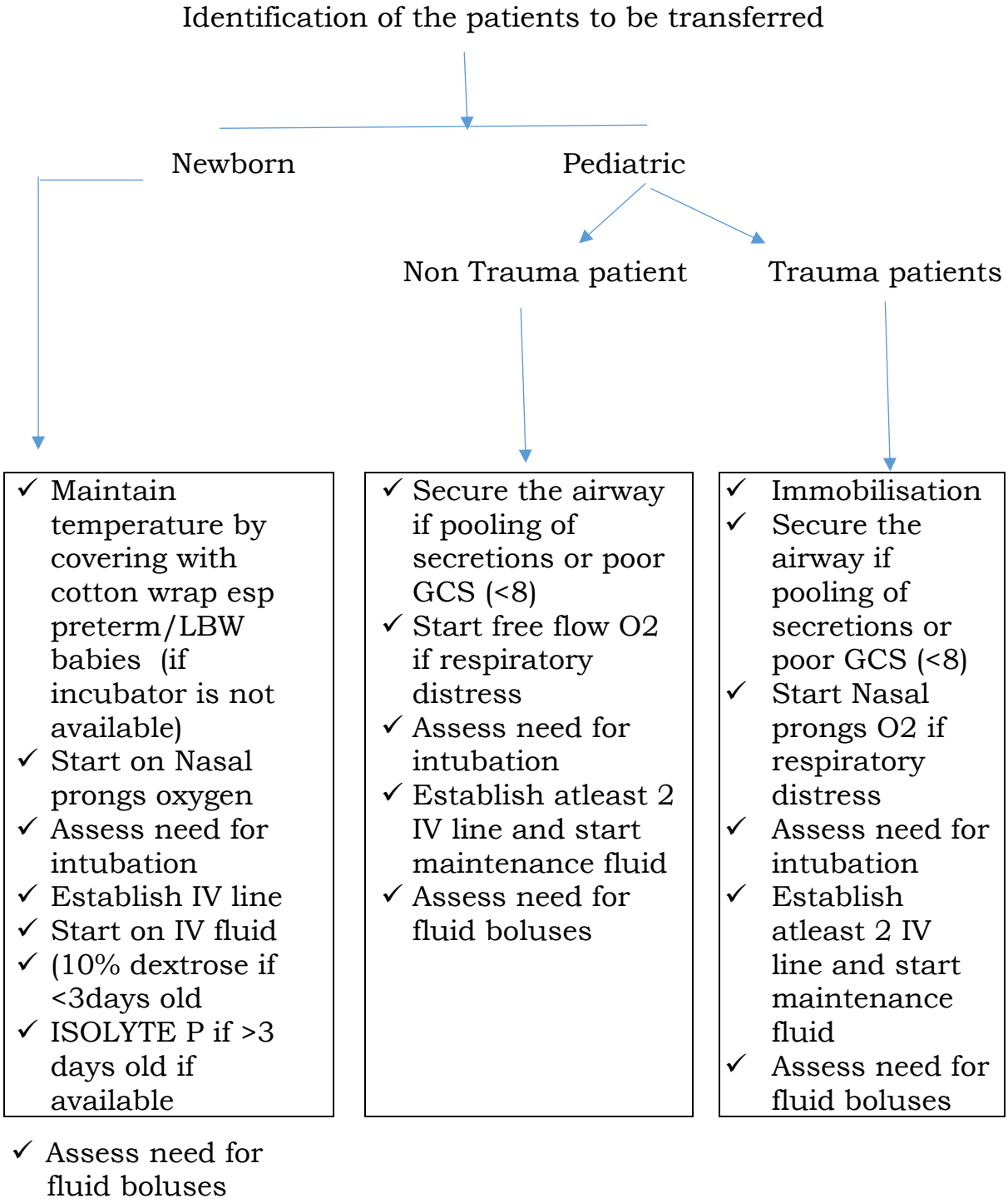
- Document intial evaluation and stabilization
- Indication for transfer
- Informed consent
- Name of the referred hospital
- Appropriate staff
- Monitoring equipment
- Pulse oximeter
- Glucometer

- Essential equipment
  - Airway bag: Endotracheal tube, laryngoscope, mask, AMBU, tape
  - Fluids for boluses
  - Oxygen tubing
  - Suction
  - Defibrillator
  - Catheter/IO needles/IV cannula/NG tubes

- Medications
  - Adenosine
  - Atropine
  - Calcium
  - Dextrose
  - Dopamine
  - Dobutamine
  - Dobutamine
  - Epinephrine
  - Lidocaine

- Mannitol
- Dextrose
- Dopamine
- Dobutamine
- Epinephrine
- Lidocaine
- Mannitol/3% NaCl
- Magnesium sulfate

Algorithm for transfer of critically ill Pediatric patient



1. APPROACH TO A CHILD WITH BREATHING DIFFICULTY

1.1 Learning objectives

- After completion of this section, the participants should be able to
- Identify a child with breathing difficulty
- Differentiate between respiratory distress and respiratory failure
- Localise level of respiratory pathology based on clinical signs
- Resuscitate and initiate appropriate stabilisation measures
- Escalate respiratory support as indicated
- Identify likely etiology and initiate specific therapy
- Know when and how to refer?

1.2 How to differentiate respiratory distress and respiratory failure?

- Any pathology in the respiratory system will first cause respiratory distress (increased rate and retractions)
- Clinically the severity of respiratory impairment is classified as respiratory distress or failure
- By increasing respiratory rate and efforts, the child tries to maintain adequate gas exchange. This is body's compensatory mechanismto allow the child to maintain oxygenation within normal limits (SpO2>94%)
- Hypoxemia is defined as SpO2 ≤ 94%
- Respiratory failure ensues when child gets tired and compensatory mechanisms fail
  - This result in inadequate oxygenation (SpO2≤94%) and/or inability to eliminate carbon dioxide (PCO2>50mm Hg)
  - Respiratory failure can be acute (Pneumonia, pulmonary oedema) or chronic (chronic lung disease, heart disease or neuromuscular disorders)
- Based on the inciting mechanism and arterial blood gases, acute respiratory failure can be categorised into two broad categories (refer box 1.1)

Box 1.1: Types of respiratory failure

- Type I Respiratory failure: Occurs due to any condition that leads to hypoxemia, SpO2≤94% or PaO2 <60mm Hg eg. Pneumonia
- Type II Respiratory failure: Occurs due to any condition that causes hypoventilation resulting in hypercarbia (PCO2>50mm Hg) eg. Neuroparalytic snake envenomation

1.3 What is the stepwise approach to a child with breathing difficulty?

- Step 1 : Assessment: Begins with a quick visual and auditory evaluation using Pediatric Assessment Triangle followed by Primary assessment pentagon ABCDE (Refer section A)
- Step 2 : Classify severity and localize level of respiratory pathology
- Step 3 : Initiate stabilization measures
- Step 4: the etiology of respiratory distress by meticulous history and focused physical examination

History should include SAMPLE: signs and symptoms, Allergies, Medications, Past medical history, Last meal, and Events leading to the present illness

- Onset (Acute, recurrent,chronic) and nature of progression
- Associated symptoms: cough, fever, rash, chest pain
- Preceding events: choking, foreign body inhalation, trauma, exposure to chemical or environmental irritants.
- Family history: exposure to infections, tuberculosis, atopy

Focused physical examination should include

Clubbing, lymphadenopathy

Tracheal position and adequacy of chest movements

Adventitious sounds: stridor, wheeze, grunt (suggest alveolar disease causing loss of functional residual capacity), crepts

Detailed assessment of other systems like cardiovascular and neurological

- Step 5: Appropriate investigations
- Step 6: Initiate specific and adjuvant therapy (e.g antibiotics, fluids etc)
- Step 7: Reassessment and monitoring to decide need for escalation or de-escalation of therapy

1.4 How to localise organ system involved based on breathing pattern and understand the pathology by the pattern of breathing? (refer box 1.2 and 1.3)
Box 1.2

System	Characteristics	Examples
Respiratory	Tachypnea with increased efforts: subcostal, intercostal, subxiphoid retractions Abnormal sounds: stridor, grunting, wheezing, crepts	Croup, diphtheria, bronchiolitis, asthma pneumonia, pulmonary oedema
Cardiovascular	Disproportionate tachycardia, soft muffled heart sounds, cardiac murmurs, cardiomegaly, hepatomegaly, raised JVP, basal crepitations	Congestive cardiac failure, myocarditis, arrhythmia, pericarditis
Metabolic	Acidotic breathing: tachypnea without retractions, deep and sighing breathing and clear chest	Diabetic ketoacidosis, acute renal failure, inborn error of metabolism

Neurogenic hyperventilation	Increased rate and depth without retractions, and clear chest. Associated altered senosorium and features of raised intracranial pressur	Acute encephalitic syndrome, traumatic brain injury
Neurogenic	See-saw or paradoxic breathing (chest moving inwards with inspirations)	Neurotoxic snake envenomation, GuillianBarre syndrome, CNS depression

BOX 1.3: How to localise pathology and level within respiratory system?

Retractions + stridor	Upper airway obstruction	Croup, diphtheria, foreign body
Retractions + wheeze	Lower airway obstruction or small airway obstruction	Bronchiolitis, asthma
Retraction + grunt+ crepitations	Parenchymal disease	Pneumonia, pulmonary oedema, hemorrhage

1.5 Steps in stabilisation

- Goals of therapy are
  a. To relieve hypoxemia
  b. To give rest to overworked respiratory muscles
  c. To enhance CO2 wash out
  d. To support other organ system
  e. To treat underlying etiology
- Ensure and maintain an open and patient airway by
  - Proper position (gentle extension of the neck, rolled towel under shoulder)
  - Suction oropharyngeal secretions
  - Consider oropharyngeal airway if patient is unconscious, to prevent tongue falling back over airway (refer appendix)
  - Endotracheal intubation/tracheostomy in emergent conditions

  (refer box 1.4,1.5)

Deliver supplemental oxygen without increasing agitation

- By and delivery device that is available
- Choice between low flow or high flow oxygen delivery device depends on severity of hypoxemia, patient's flow demand and tolerability, availability and familiarity with device (refer appendix)
- Monitor for target SpO2 and escalate respiratory support accordingly

- Patients not responding to increasing FiO2 (SPO2≤94%) can be put on non invasive respiratory support, e.g. Bubble CPAP, Ventilator CPAP, or HFNC (refer appendix)

- If spontaneous breathing is absent or inadequate then initiate
  - Assisted ventilation with bag and mask ventilation
  - Endotracheal intubation as soon as adequate expertise and equipment are available

- Optimize all circulatory parameters
  - Establish intravenous or intraosseous access (if venous access is not feasible)
  - If patient is in shock initiate shock protocol (refer section C, Chapter 1)

- Once stabilization measures have been instituted, proceed to established the etiological diagnosis so as to initiate specific therapy (e.g. antibiotics for bacterial pneumonia or diuretics for congestive cardiac failure)

BOX 1.4 Indications of emergent endotracheal intubation

- Children with marked respiratory distress despite adequate FiO2
- Gasping, bradypneic or apneic
- Cyanosis with decreased respiratory efforts
- CNS signs of hypoxia (lethargic, unresponsive, obtunded, seizures, coma)
- Cardiovascular signs of hypoxia (marked tachycardia, bradycardia, or hypotension)
- Children who worsen on CPAP or other non invasiverespiratory support

Box 1.5 Indications of emergent tracheostomy

- Diphtheria
- Upper airway obstruction
- Facial trauma
- Unable to intubate

1.6 What is the likely etiology? How to investigate?

- Chest X ray PA view (once the child is stabilized)
- Arterial blood gas analysis (if available)

- Sepsis Work-up
  - Blood counts (TLC, DLC)
  - C-Reactive protein
  - Blood culture if available

1.7 How to monitor for response?

Monitoring is required to assess response to therapy and detect worsening/ complications

- What is monitor?
  - Temperature
  - Vitals: HR,RR,SPO2, perfusion, BP
  - Work of breathing and use of accessory muscles
  - Cyanosis
  - Signs of exhaustion such as somnolence, confusion, and seizure

1.1 When to refer?

- Persistent hypoxemia/respiratory failure requiring ventilation
- Presence of shock/multiorgan dysfunction

1.2 How to refer?

- Secure airway, breathing and circulation
- Ensure resuscitation kit and a BLS trained provider during transport
- Child's peripheral venous access should be secured and patent
- Child's details, history, examination findings, vitals, events which occurred and treatment given should be legibly written in the referral letter
- The parents should be counselled regarding the child's condition and need for referral
- Written, informed consent should be taken from the parents/guardian's prior to referral

2. COMMUNITY ACQUIRED PNEUMONIA

2.1 Learning objectives

After completion of this section, the participants should be able to

- Identify a case of community acquired pneumonia (CAP)
- Grade severity and stabilize
- Assess the etiology and risk factors
- Specific management of all cases of pneumonia
- Identify causes of non response
- Know when and how to refer?

2.2 What is CAP? How to identify?

- Any child presenting with fever, cough and difficult or rapid breathing should be considered as a case of pneumonia (WHO definition)
- Tachypnea is defined with age specific cut offs (refers box 2.1)
- CAP should be differentiated from other common respiratory illnesses such as

BOX 2.1: WHO Age specific criteria for tachypnea		
Age	Approximate normal respiratory rate (breaths/min)	Tachypnea threshold (breaths/min)

a b r Onchiolitis and upper airway

infections.

2-12 Months	25-40	50
1-5 Years	20-30	40
>5 Years	15-30	30

BOX: - 2.2 Risk factors for community acquired pneumonia

- Malnourished state
- Age< 6 months
- Post-measles state
- Absence (or inadequate) breastfeeding
- Solid fuel use

2.3 Clinical features and etiology

The symptomatology and etiology is determined by the age of the patient (refer box

BOX 2.3: Age-wise clinical features and etiology of pneumonia

	Neonates	Infants	Preschool Children
Symptoms	Hypothermia Poor feeding Irritability Fever and cough may be absent	History of antecedent URI Cough prominent	History of antecedent URI Cough Post-tussive vomiting Chest pain, if pleural involvement Abdominal pain, if lower lobe involved

Signs	Tachypnea, grunting, nasal flaring, retractions	Tachypnea, grunting, and retractions	Tachypnea, grunting, retractions and chest pain
Etiology	Group Streptococcus Escherichia coli Klebsiella Cytomegalovirus	**Febril:** Respiratory syncytial virus S. Pneumoniae H. influenza (type b) Staphylococcus aureus, Mycoplasma Pneumonia **Afebrile:** Chlamydia Trachomatis Mycoplasma hominis Cytomegalovirus	Respiratory viruses, S. Pneumoniae H. Influenzae (type b) Staphylococcus aureus Mycoplasma Pneumoniae Chlamydia pneumonia, Group A Streptococcus

2.4 How to assess severity?

The management of pneumonia is guided by severity of disease (Box No. 2.4)

BOX 2.4 WHO severity criteria (6 months – 5 years)	
Cough and cold (No pneumonia)	No fast breathing No chest in drawings
Pnuemonia (Not severe)	Fast breathing (>age specific threshold) No chest is drawings
Severe pneumonia	Tachypnea + chest indrawing + danger signs (lethargy, refusal to feed, convulsion, central cyanosis, severe malnutrition, grunting)

2.5 How to investigate?

Hemogram with total and differential leukocyte count

CRP – bacterial pneumonia> viral pneumonia

Blood culture: Positive in 10%-20% of children with pneumonia, may be done if available

CSF (if feasible) in case of

Newborn

Infants presenting with altered sensorium

Seizures

Irritability out of proportion to illness

Chest X ray (Refer Box 2.4)

BOX 2.5: Indication for chest X-ray

- When the diagnosis is in doubt (bronchiolitis, asthma, developmental malformation, foreign body inhalation, aspiration pneumonia)
- Asymmetrical findings on chest examination
- Suspected complications of pneumonia (pleural effusion, empyema, lung abscess)
- Know case of recurrent respiratory illness (asthma, cystic fibrosis, immunodeficiency disorders)
- Severe pneumonia

Arterial blood gas: Not routinely indicated. If available in a given set up then can be done for following indications

Box 2.6: Indications of ABG

- Sever pneumonia
- Hypoxemia on pulse oximentry (SpO2 <94% on 40% FiO20
- Cyanosis

2.6 Steps in the management of pneumonia

Grade the seventy and classify (Refer Box 2.4)

Resuscitate and stabilize ABC

- Airway: Maintain open and stable airway; suction if secretions are present. Indications of intubation (Refer box 2.7)
- Breathing: start oxygen by any delivery device, depending on availability free flow oxygen/nasal prongs, at flow rate 1-5L/min (depending on age) if child has lower chest wall, indrawing or SpO2≤94%.
- Circulation: Maintain normal circulatory parameters – correct dehydration; use vasoactives if there is shock despite adequate fluids; think of septic shock

Supportive care

Hydration (intravenous or nasogastric tube feed)

If oral acceptance is good – allow orally

Oral acceptance poor (but feeding not contraindicated) – nasogastric or orogastric feed

Severe distress (feed contraindicated): Start fluids 0.45 Saline in 5% dextrose as 2/3rd to 3/4th maintainance

Check blood glucose by dextrostix. Correct hypoglycaemia and maintain euglycemia (blood glucose between >60mg/dl – 100mg/dl

Treat electrolyte imbalance (refer section E, Chapter 3,4)

Treat fever with oral paracetamol 15mg/kg/dose, can be given 6 hourly if need be

Box 2.7 Indications of endotracheal intubation

- Children with marked respiratory distress despite adequate FiO2
- Gasping, bradypneic or apneic
- Cyanosis with decreased respiratory efforts
- CNS signs of hypoxia (lethargic, unresponsive, obtunded, seizures, coma)
- Cardiovascular signs of hypoxia (marked tachycardia, bradycardia, or hypotension
- Children who worsen on CPAP or other non invasive respiratory support

2.7 Antibiotics

Choice of Antibiotic therapy is guided by multiple factors (Refer Box 2.6)

BOX 2.8 Guidelines for antibiotic choice

- Age of the child (refer to box 2.3)
- Severity of pneumonia (refer to box 2.4)
- Associated clinical features suggesting specific eitology e.g. pustules suggesting staphylococcal infection
- Immune-suppressed or immunocompromised state (such as PEM, post-measles state, chronic steroid therapy)
- Underlying chronic lung disease e.g. cystic fibrosis
- Radiographic pointers towards a specific etiology
- Presence of complications such as pneumothorax/empyema

- Pneumonia (non severe) can be treated at home with oral antibiotics in most cases
  - Amoxicillin (40mg/kg/dose) in 2 divided doses for 3-5 days
  - Follow-up after 48 hours to reassess for improvement (general well being, respiratory rate, retractions, appetite)

Severe Pneumonia

  - IV ampicillin (50mg/kg/dose,max 12gm/day) 6 hourly + IV gentamicin 7.5 mg/kg/dose 24 hourly
  - Add cloxacillin (100-200mg/kg/day in 4 four divided doses) if clinical & radiographic features suggest staphylococcal infection (pustules, post- measles state, severe malnutrition, empyema, pneumatoceles, necrotizing pneumonia or air leaks)

- Add azithromycin if CXR suggests atypical pneumonia
- Reasess after 48 hours

2.8 How to proceed after reassessment at 48 hours of therapy?

- In case of (non severe) pneumonia hospitalise urgently if danger signs appear (inability to suck/drink, impaired sensorium, convulsions, grunting, cyanosis)
- If no danger signs but persistence of difficulty in breathing then
- Change to amoxicillin-clavulanic acid (80-90 mg/kg of amoxicillin) in 2 divided doses for 5 days or
- Add azithromycin 10 mg/kg for 5 days if features suggest atypical pneumonia
- In severe pneumonia
- If improved and patient is able to take orally then shift to oral amoxicillin and continue for 5 more days
- If not improved or deteriorated: go over a check list of non- response (refer box 2.9)
- If above are tackled then upgrade antibiotic to IV ceftriaxone (100 mg/kg/day, max 4gm) 12 hrly
- Change antibiotics as per cultures

2.9 How to monitor?

- Child should be monitored by nurses every hourly and by doctors at least twice a day
- Look for heart rate, respiratory rate, retractions sensorium, SPO2 and circulatory parameters

2.10 What is non response and how do we identify?

- Persistently raised respiratory rate at 48 hours
- Danger signs at any time during the illness, such as inability to suck/drink, impaired sensorium, conculsions, grunting, cyanosis.
- Causes of non response are enumerated in Box 2.7

BOX 2.9 : Causes of Non response

- Asthma/reactive airway disease/ wheeze associated LRTI
- Underlying cardiac disease
- Underlying obstructed bronchus or collapse
- Foreign body
- Pulmonary malformation
- Thrombophlebitis
- Inadequately drained source e.g. empyema

- Incorrect choice and dosing of antibiotics e.g non-staphylococcal cover for post measles pneumonia

2.11 When to refer?

- Severe pneumonia
- Hypoxemia/respiratory failure requiring ventilation
- Presence of shock/multiorgan dysfunction

2.12 How to refer?

- Secure airway, breathing and circulation
- Ensure resuscitation kit and a BLS trained provider during transport
- Child's peripheral venous access should be secured and patent
- Child's details, history, examination findings, vitals, events which occurred and treatment given should be legibly written in the referral letter
- The parents should be counselled regarding the child's condition and need for referral
- Written, informed consent should be taken from the parents/guardian's prior to referral

Protocol for management of CPAP

Identify case of pneumonia (symptoms & signs)
Rule out other causes

Assess severity
WHO classification

Pneumonia

Treat at home with oral antibiotics
Amoxicillin (40 mg/kg/dose0 in 2 divided doses for 3-5 days
Reassess after 48 hrs for
General danger sings (inability to suck/drink, impaired sensorium, convulsions grunting, cyanosis) if present hospitalise urgently
If child has persistent tachypnea but no indication for admission

- Change to amoxicillin-clavulanic acid (80-90 mg/kg of amoxicillin) in 2 divided doses for 5 days if
- features suggest atypical pneumonia.

Advise to return immediately if the child develops any danger signs

Sever Pneumonia

Resuscitation and stabilisation
A: Maintain open and stable airway; Suction secretions
B: Support breathing: Start oxygen by any delivery device, everity, demand, and availability of pro-mpts.
pO2> 94%
C: Maintain normal circulatory parameter – ct dehydration; use vasoactives if there is shock despite adequate fluids, think of septic shock
If oral acceptance is good – Hydration (intravenous or nasogastric tube
Oral acceptance poor (but feeding not contraindicated) – Nasogastric or orogastric feed
Severe distress (feed contraindicated); Start fluids 0.45 saline in 5 % dextrose as 2/3rd to 3/4th maintenance

First dose of antibiotics** (as early as
Maintain target S
feed)

Maintain euglycemia (blood glucose)
Treat electrolyte imbalance

** Antibiotics in severe pneumonia

- IV ampicillin (50 mg/kg/dose) 6 hourly + IV gentamicin 7.5mg/kg/dose 24 hourly
- Add cloxacillin (100-200 mg/kg/day in 4 four divided doses if clinical & radiographic features suggest staphylococcal infection (pustules, post – measles state, severe malnutrition, empyema, pneumatocles, necrotizing pneumonia or air leaks)
- Add azithromycin if CXR suggests atypical pneumonia

Monitor and assess after 48 hours

- If improved and patient able to take orally then shift to oral amoxicillin for 5 more days

If not improved in 48 hours or deteriorated, check list of non response (box 2.9)
If above are tackled, upgrade to IV ceftriaxone
Change antibiotic as per cultures

- Monitor and assess after 48 hours

If improved: complete 10-14 days of antibiotics

3. ACUTE EXACERBATION OF ASTHMA

3.1 Learning objectives

After completion of this section the participants should be able to

Identify a case of acute exacerbation of asthma
Assess the severity based on clinical evaluation
Initiate appropriate bronchodilator therapy to relieve airway obstruction
Identify red flag signs of acute severe asthma
Initiate appropriate respiratory support
Monitor therapeutic response
Know when, how to refer and precautions before referral

3.2 What is acute exacerbation of asthma?

Acute exacerbation of asthma is defined as episodes of coughing (particularly in the night/early morning), wheezing, and/or breathlessness (with or without fever) due to diffuse inflammation and airflow obstruction of the lower airways. This is reversible either spontaneously or with treatment.

Acute severe asthma is defined as a severe asthma exacerbation that does not respond to repetitive or continuous administration of inhaled short-acting β2 – adrenergic receptor agonists (SABAs) in an emergency setting

3.3 How to identify an acute attack of asthma?

- Family and past history of reactive airway disease/atopy/eczema
- Correlation with triggers: dust, mold, specific food, animal hair
- History of recurrent symptoms, seasonal predilection
- History of cough, fever, respiratory distress, noisy breathing, whistling sounds
- Therapeutic response to bronochodilators

Differential diagnosis as given in Box 3.1

Box 3.1 Differential diagnosis

- Wheeze associated lower respiratory tract infection

Bacterial pneumonia: usually a sick looking child, with moderate to high grade fever, bronchial breathing and crepitations

Viral Pneumonia: associated with an upper respiratory prodrome

- Congestive cardiac failure (secondary to myocarditis or congenital heart disease): associated with suck-rest-suck cycle, sweating, tachycardia, hepatomegaly, basal crepitations, murmurs and cardiomegaly on chest radiography.
- Bronchiolitis: As described in section B, Chapter 4
- Anatomic and functional abnormalities : will cause recurrent episodes of wheezing

Extrinsic airway anomalies: vascular ring/sling

Intrinsic airway anomalies: congenital lobar emphysema, lung sequestration, cystic adenomatoid malformation

Gastroesophageal reflux

- Foreign body: Sudden respiratory worsening, unilateral findings with or without history of choking
- Mucociliary clearance disorders: Recurrent episodes of wheezing with failure to thrive
  - Cystic fibrosis
  - Primary ciliary dyskinesia

3.4 How to grade severity?

- Severity of asthma can be graded by the clinical Assessment severity Score (CASS) (refer box 3.2). This score should be used for initial assessment and to monitor response to therapy

Box 3.2 Clinical Asthma Severity Score (CASS)

Score	RR	Room air SpO2	Asuscultation (wheeze)	Retractions	Dyspnea
0	<30	97-100	None	None	None
1	31-45	94-96	End expiration	+/-	Full sentences
2	46-60	91-93	All expiration	++	Partial sentences
3	>60	<91	Inspiration & Expiration	+++	Single words/ grunts

Maximum score is 15; mild <4, moderate 4-7, severe >7

3.5 Steps in management?
Grade severity (Refer Box 3.2)
Start oxygen
Maintain SpO2 >94%
Use humidified oxygen via free flow, nasal prongs or through face mask @6L/min
Supportive care
Encourage oral fluids in mild distress
Give restricted fluids (80%) in moderate and severe cases
Treat pyrexia with oral paracetamol 15 mg/kg/dose
Start initial nebulizations
Salbutamol (<5yrs – 2.5mg, >5yrs – 5mg) every 20 min, 3 times OR MDI salbutamol (100µg/puff) 6 puffs with spacer with/without mask (if the child can use the MDI) Budesonide (800µg/dose) 3 times every 20 mins
Ipratropium 250µg/dose or 2 puffs (80µg/puff) 3 times every 20 mins
Mix all three in the nubulizations chamber and dilute in normal saline of required to make a volume of 3-4 ml and give it within one hour
Steroids: indications of steroids are enumerated in Box 3.3
Oral: prednisolone 02 mg/kg/day x 5 days OR
IV hydrocortisone 10 mg/kg followed by 5 mg/kg 6 hrly x 5 days

Box 3.3 Indications of steroids
Acute severe asthma
Previous history of life-threatening attack or severe attacks requiring System steroids
Child on regular oral steroids or high dose inhaled steroids

3.6 How to monitor?
Child should be monitored by a nurse atleast every 1 hourly and by a doctor atleast 4 hourly
Monitor CASS score (Refer Box 3.2)

3.7 How to define adequate or no response? (Refer Box 3.4)
Box 3.4: Response to treatment

Adequate response (improving)	No response (worsening)
No distress/dyspnea Response sustained 60 mins after the last treatment No wheeze SPO2 > 94 % No pulses paradoxus	HR/RR – increase Persistent dyspnea Decreased air entry Accessory muscle usage severe SPO2 < 94 % Pulses paradoxus > 15mmHg

3.8 How to escalate therapy in non-responders to initial nebuliations?

In case of severe asthma not responding to initial therapy alternative drugs may be required

Magnesium sulphate: Causes bronchodilation and decreases neutrophilic burst associated with inflammation

Dose: Intravenous infusion: 50 mg/kg/dose in 30 ml NS with 5 % dextrose

Over 30 min, can be given 6 hrly (maximum 4 doses)

Monitor adequate urine output

Aminophylline: increases endogenous catecholamine release an has anti-inflammatory action

Dose: 5 mg/kg IV loading f/b 0.9mg/kg/hr infusion

Side effects: Tachycardia, hypokalemia

Terbutaline: Beta agonist

Dose: 10 mcg/kg loading dose followed by 0.1 mcg/kg/min continuous infusion (can be hiked every 30 min 0.1-0.2 mcg/kg/min max 10 mcg/kg/min

Side effects: Hypokalemia, arrhythmia, hypotension and myocardial ischemia.

3.9 How to investigayte?

Laboratory studies are generally not indicated in a routine acute exacerbation, unless child is unusually ill with suspicion of infection

Chest radiographs are nor also routinely indicated except as outlined in box 3.5

Arterial blood gases (if available)

Box 3.5 indications of CXR
If clinical examination suggests the possibility of pneumothorax or pneumonia
Suspecting other causes of wheeze such as airway foreign body, anatomic abnormality etc.

3.10 When to give antibiotics?

Not routinely indicated except in following conditions

High grade fever without any signs of viral prodrome

Chest radiograph showing a patch of consolidation

Which antibiotics to be given? (Refer section on pneumonia)

3.11 What is the check list in case of non-responders?

Think of alternative diagnosis (Box 3.6)

Box 3.6: Non responders
Foreign body inhalation
Anatomic abnormalities
Underlying cardiac disease
Secondary bacterial infection
Wheeze associated lower respiratory tract infection
Gastro oesophageal reflux disease

Interstitial lung disease

3.12 Whom to admit? (Refer box 3.6)

Box 3.7 Indications of admission Severe distress as per CASS (refer Box 3.2) Inadequate or no response to supportive first hour treatment in mild or moderate cases Requiring > 10 puffs of salbutamol Suspicion of alternative diagnosis

3.13 When to refer?

Any child with signs of life-threatening attack
Poor or no response to therapy in emergency department
Development of signs of respiratory fatigue

3.14 How to refer?

Refer only after stabilization, ensuring adequate airway, breathing and circulation
Proper documentation regarding the presentation of the child, details of resuscitative measures taken, interventions done during resuscitation and reasons for referral including name and contact number of referring physician.
A doctor/paramedic trained in Pediatric Advanced Life Support/Basic Life Support to accompany the patient
Transporting ambulance should have sufficient O2 supply and Resuscitation equipment
Inform the referral centre prior to sending the patient regarding the diagnosis of the child, indication for referral, current status and approximate time to arrival
Counsel the family of the child regarding the need for transfer and risks during transfer. Obtain written, signed consent for the same.

Remember: Red flags: An anxious asthmatic child indicates severe obstruction and probably hypoxia Silent chest indicates inadequate air exchange and severe airway Obstruction Unilateral diminished breath sounds (in a case of asthma) indicates severe obstructive atelectasis or a pneumothorax, and needs urgent attention. A calm patient usually denotes, at the most, mild distress Do not interrupt oxygen supply during nebulisation CXR is not routinely indicated Intubation is seldom required in asthma. Positive pressure ventilation can worsen hemodynamic and respiratory parameters in lower airway obstruction. Hence decision should be taken cautiously

4. ACUTE BRONOCHIOLITIS

4.1 Learning objectives:

After completion of this section, the participants should be able to

Identify a case of acute bronochiolitis

Categories severity

Rule out other causes of wheezing in an infant

Initial stabilization and other therapies

Familiarize with non-invasive respiratory support modalities like bubble CPAP and high flow Nasal cannula (HFNC)

Monitor for response

Know when and how to refer and what precautionary measure to taken before referral?

4.2 What is Bronchiolitis?

- It refers to inflammation of peripheral small airways
- It is the most common cause of wheezing in young infants
- Smaller children are more prone to wheezing due to following reasons:

- In children <5 yr old, small-calibre peripheral airways contribute up to 50% of the total airway resistance hence even a marginal (slight) additional narrowing causes further flow limitation and wheeze.
- With the very complaint newborn chest wall, the inward pressure produced in expiration subjects the intrathoracic airways to collapse.
- Differences in tracheal cartilage composition and airway smooth muscle tone, further increase airway resistance in comparison to older children.
- Infant who presents with mild to moderate grade fever, upper respiratory symptoms, with wheeze and respiratory distress, without previous such episodes, bronchiolitis is the most likely diagnosis.

Box 4.1 Differential diagnosis:
First episode of asthma: family history or past history of atopy (nasal allergy, atopic dermatitis, eczema or asthma) and good response to bronchodilators
Wheeze associated lower respiratory tract infection:
Bacterial Pneumonia : Usually a sick looking child, with moderate to high grade fever, bronchial breathing and crepitations
Viral Pneumonia: Associated with an upper respiratory prodrome.
Congestive cardiac failure (secondary to myocarditis or congenital heart disease): Associated with suck-rest-suck cycle, sweating, tachycardia, hepatomegaly, basal crepitations, murmurs and cardiomegaly on chest radiography.

4.3 How to assess severity?

The severity can be assessed by the modified Respiratory Distress Assessment (RDAI) Score.

Box 4.2 Modified Respiratory Distress Assessment (RDAI) Score
Mild: 0-4, Moderate: 5-8, Severe: 9-12

Clinical parameter	Score 0	Score 1	Score 2	Score 3
Respiratory rate (per min0	< 40	40-60	60-70	>70
Use of accessory muscles	None	1 accessory muscle used	2 accessory muscles used	3 or more accessory muscle used
Colour/Cyanosis	No cyanosis in room air/pink in room air	Cyanosed when crying	Cyanosed in room air/Pink with oxygen	Cyanosed with oxygen or cardio-respiratory arrest
Auscultatory findings	Normal	Decreased air entry, no rhonchi	Decreased air entry, rhonchi heard	Silent chest

4.4 Treatment of Bronchiolitis:

- Grade severity (refer box 4.2)
- Start oxygen

  - Maintain Spo2>94%. Continue oxygen until no signs of hypoxemia are present (lower chest retractions, tachypnea for age)
  - Use humidified oxygen: Free flow/Nasal prongs/oxygen hood/non rebreathing mask depending on severity, availability and patient's tolerance
  - In moderate and severe distress: Preferable to five some PEEP by using:
  - Indigenous bubble CPAP/high flow nasal cannula. PEEP helps to splint the small airways and keep them patent (refer appendix)

- Supportive Care:
  - Encourage breastfeeding and oral fluids in mild distress
  - Give resitricted fluids (80%) in moderate and severe cases
  - Treat fever with oral paracetomal 15mg/kg/dose
  - Ensure regular and gentle suctioning if any thick secretions present in nose and mouth

- Nebulizations

  - Hypertonic (3%) saline decreases airway inflammation, mucus plugging thus improving the mucus clearance.
  - Dose : 4 ml of hypertonic saline (3%) without dilution
  - Nebulized epinephrine 3-5 ml (1:1000) without dilution has also been shown to improve symptoms
  - Hence a trial of both drugs should be considered but there is nor role of round the clock nebulisations
  - Continue oxygen during nebulisation at a flow rate of 6-8 L/min.
  - Monitoring: (Refer box 4.3)
  - Child should be monitored by a nurse at least every 3 hourly and by a doctor at least twice a day
  - Look for respiratory rate, any retractions, sensorium SPO2

Box 4.3 How to judge response: Decrease in respiratory reate/work of breating Decrease in heart rate SpO2 ≥94%

Non responders: (Refer Box 4.4)

Inadequate oxygen delivery; Device malfunction or improper oxygen supply via cylinder or central source Inadequate drug delivery: Inappropriate dose/inadequate delivery Associated pathology Multiple small atelectasis (collapse) on CXR Secondary bacterial infection: sick child

First episode of asthma: family history of atopy, may require a trial of bronchodilators Underlying heart disease and congestive cardiac failure

When to intubate a case of bronchoiolitis?
Indications for intubation in bronchiolitis are
Severe respiratory distress not maintaining saturation on CPAP or HFNC
Patient progressing to respiratory failure.

When to five antibiotics to a case of bronchiolitis?
Prolonged illness of >7 days (usually bronchiolitis improves within 3-4 days)
Persistent high grade fever > 48 hrs.
Inadequate response to therapy requiring escalation of respiratory support
Chest x-ray suggestive of lobar consolidation, necrotizing lesions

Which antibiotics to give?
Refer section B, Chapter 2
4.5 When to refer?
• No improvement with supportive treatment
• Ventilator support required

4.6 How to refer?
• Child' peripheral venous access should be secured.
• Child's details, history, examination findings, vitals, events occurred and the treatment given should be legibly written in the referral letter.
• Parents should be counseled regarding patient's condition
• A person trained in BLS should accompany the child during referral.
• If child is being referred for ventilator support, then it is preferable to intubate and transport

Remember:
• Anxiety/ tachycardia may be due to hypoxemia; Do not sedate
• Humidification gases is critical
• Nebulization with epinephrine can be given maximum twice" should not be given round the clock '
• Do not interrupt oxygen supply while nebulizing
• No role of antibiotics in Management of simple bronchiolitis
• Non invasive modes of respiratory support like CPAP or HFNC are very useful and help avert intubation
Positive pressure ventilation can worsen hemodynamic and respiratory parameter in lower airway obstruction. Hence, the decision to intubate should be taken cautiously.

Protocol for management of acute bronchiolitis

Protocol Management of Acute Bronchiolitis

Identify case of bronchiolitis
Rule out other causes

Mild di tress
Humidified oxygen
Free flow/nasal prongs/

oxygen hood (*depending on availability and patient's tolerance)
Oral feed : If child accepting orally

* Nebulizations:

3ml hypertonic saline (3%) followed by 3ml (1:1000) epinephrine(1: 1000)

Moderate distress
Humidified oxygen:
Non rebreathing mask

Preferable to give PEEP by using: Indigenous bubble CPAP*/high flow nasal cannula
IV fluids : 80% restricted

* Nebulizations:

3ml hypertonic saline (3%) followed by 3ml

epinephrine (1:1000)

Assess response

Severe distress
Humidified oxygen

with PEEP
Indigenous bubble

CPAP*/ High flow nasal cannula

* IV fluids: 80% restricted

Nebulizations

3ml hypertonic saline

(3%) followed by

3 ml epinephrine

Adequate
Inadequate

Continue supportive support Management through check

responders

Escalate oxygen

after going

list of non-

* Hypertonic saline and epinephrine has to be given without dilution
Continue oxygen at flow rate of 6-8I/min during nebulization

PROTOCOL FOR MANAGEMENT OF A CASE OF ACUTE EXACERBATION OF ASTHMA

Identify case of acute exacerbation of asthma & grade severity (refer box 3.2)

Mild Case

Humidified oxygen: Free flow or through face Mask 6L/min
or
Nebulization:
Salbutamol (0.15 mg/kg) diluted in 3 mL NS, every 20 min, 3 times or MDI salbutamol (100 µg/dose) every 20
2 times every 20 mins
MDI
Steriods:
spacer
Oral: Prednisolone 2 mg/kg/day in 2 divided doses x MDI)
5 days
times

Moderate or severe case

Admit the patient
Humidified oxygen: Free flow through face mask 6 L/min
Nebulization:
Salbutamol (0.15mg/kg) I mins, 3 times withing 1 hr OR
Salbutamol 6 puffs with and mask (if child can use
Budesonide (800 µg/dose) 3 every 20 mins
Iptatropium 250 µg/dose 3 times

every 20 mins
Steroids:
Oral: Prednisolone 2

mg/kg/day in 2

divided doses x 5 days
OR
IV hydrocortisone 10mg/kg

f/b 5mg

Per kg 6 hrly x 5 doses

Assess response at 1hour : RR, SpO2 , pulses paradoxus, CASS score

Improving

Worsening

Mild

Moderate

- Discharge the patient
- Continue steroids, if salbutamol
- Initiated (5 days) steroids
- Use salbutamol as
- Needed
- Patient education in 30
- Follow up after 48 hr

Repeat salbutamol 1 dose & give 2 doses 2

hrly

Observe for 6 hrs & discharge if improved

If not improved then treat like

Severe

Continue oxygen
Continue inhaled

Continue systemic

Monitor parameters
IV magnesium

(50%) 50 mg/kg/dose

ml NS with 5%

Over 30 min, can be

given 6

hrly (maximum 4

doses)

Monitor adequate

urine output

worsening

Indications for intubation:

- Severe hypoxemia
- Respiratory fatigue
- Rapid deterioration in the
- Child's mental state
- Cardiopulmonary arrest

Shift to PICU
Continue inhaled salbutamol continously
Continue Magnesium sulphate 6 hrly max 4 doses
Consider IV aminophylline 5 mg/kg IV loading f/b

0.9/kg

/hr infusion OR
IV ternutaline 10 mcg/kg loading dose f/b 0.1

mcg/kg/min

Continuous infusion (can be hiked every 30 min

0.1 – 0.2

Mcg/kg/min max 10 mcg/kg/min

If no response then consider intubation and ventilation

*mix all three in the nebulization chamber and dilute in normal saline if required to make a volume of 3-4 ml and give it within one hour

5. ACUTE UPPER AIRWAY OBSTURCTION

5.1 Learning objective

After completion of this section, the participants should be able to

- Identify a case of acute upper airway obstruction (UAO)
- Initial assessment and severity classification
- Immediate stabilization measures
- Etiology and differential diagnosis of UAO
- Specific management of coup
- Management of other causes of acute UAO
- Know when and how to refer and what precautionary measure to take?

5.2 **How to identify a case of acute upper airway obstruction in ER?**

- Upper airway obstruction is defined as obstruction in the respiratory tract above the level of vacal words.
- Clinical clues to upper airway obstruction include
- Stridor

- Suprasternal supraclavicular retractions
- Drooling of salive
- Dysphagia
- Change in voice
- Extension of neck in very small infants

5.3 **How to score severity of UAO?**

The severity of UAO can be assessed by using scoring systems. Though the croup score was devised originally for patients with croup, it guides in the monitoring and management of other causes of UAO also (refer box 5.1)

Box 5.1 Clinical score for croup severity

Features	**Mild**	**Moderate**	**Severe**	**Impending respiratory failure**
Barky cough	Occasional	Frequent	Frequent	Often not prominent due to fatigue
Stridor	None of minimal at rest	Easily audible to rest	Prominent inspiratory and occasional expiratory	Audible at rest but may be quiet or hard to hear
In drawing suprasternal and/or intercostal	None to mild	Visible to mild	Visible at rest	May not be marked
Distress/agitation /lethargy (CNS hypoxia)	None	None to limited	Substantial lethargy may be present	Lethargy or decreased level of consciousness
Cyanosis	None	None	None	Dusky or cyanotic without supplemental oxygen

****** Chan A, Langley J, Leblanc J. Interobserver variability of croup scoring in clinical practice. Pediatric Child Health. 2001:6 (6) : 347-51.

5.4 Management

- Initial stabilization: ABCD approach
- Keep the child in a position of maximum comfort (mother's/caregiver's lap in infant or sitting position in older child)
- Administer oxygen without causing agitation preferably by blow by method (a plastic tube held by the mother within a few centimeters of the baby's nose and mouth

- If child is unable to maintain a patent airway then intubate (refer box 5.2)
- Maintain normal hemodynamics by fluids and vasopressors
- Monitor sensorium to identify signs of hypoxia
- Identify the cause and manage accordingly (points 5.5 and 5.6)

Box 5.2 Indication of intubation

- Marked tachypnea
- Cyanosis with decreased respiratory efforts
- Decreased mental status (lethargic, unresponsive)
- Bradypnea to apnea, in severe cases poor or absent air entry
- Cardiovascular signs of hypoxia (marked tachycardia, bradycardia, or hypotension)

Box 5.3 Remember

- Perform immediate intubation in cases of impending airway obstruction as delay may lead to complete obstruction
- When in doubt it is always better intubate.

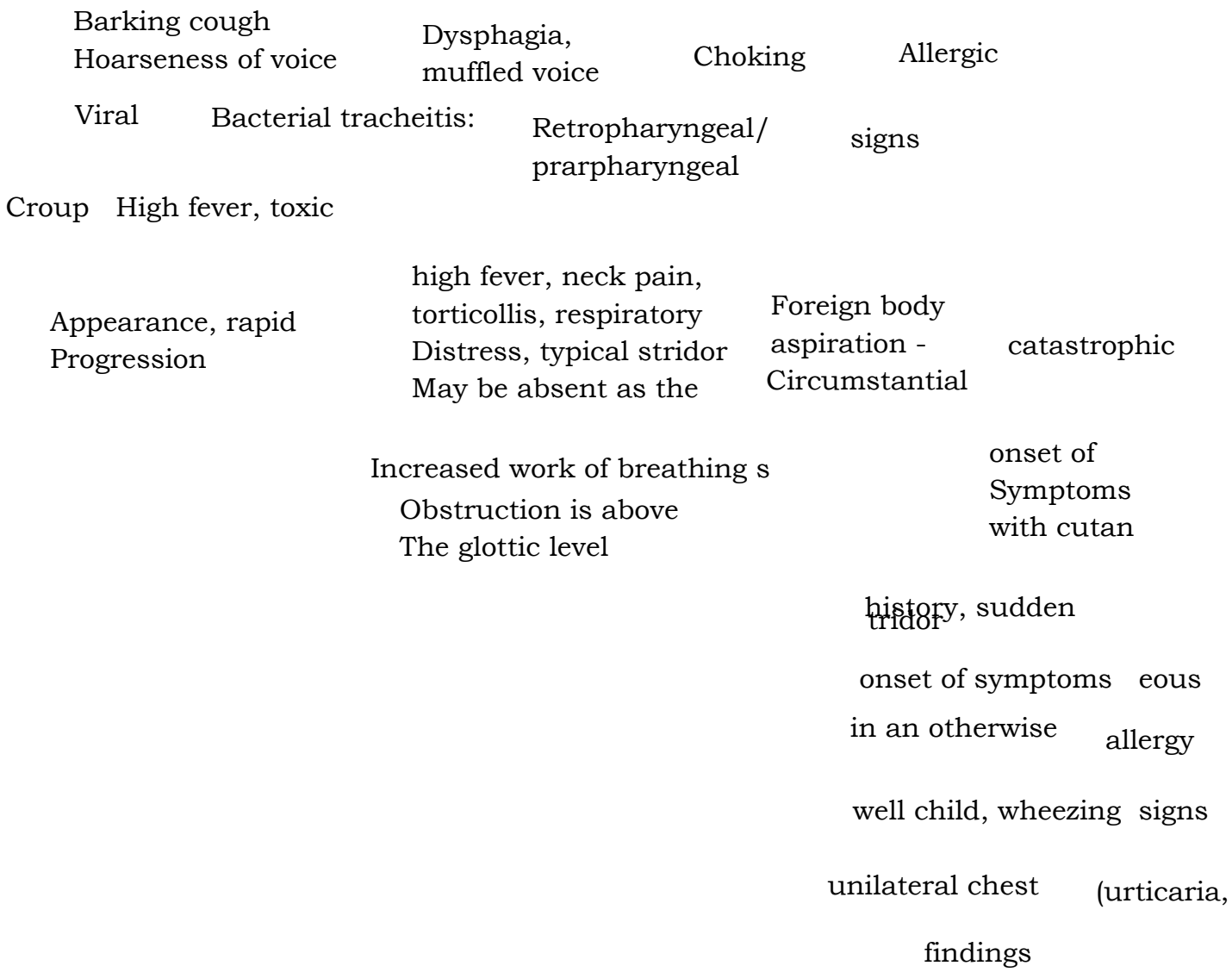
5.5 DON'T'S of upper airway obstruction

- Insert tongue depressor
- Attempt intravenous access till airway is secure
- Prone position
- Agitate child with nasal prongs or mask
- Sedate and anxious child
- Imaging for establishing diagnosis
- Transport for any investigations before airway is secure

5.6 Etiology and differential diagnosis

- Causes of acute upper airway obstruction
- Viral croup
- Bacterial Tracheitis
- Epiglottitis
- Retropharyngeal/parapharyngeal abscess
- Foreign body aspiration
- Acute anaphylaxis
- Differential diagnosis of acute upper airway obstruction in enumerated in fig. 1

Figure 1 – Causes of acute upper airway obstruction



5.7 Croup

After initial stabilization, assess the severity (refer box 5.1)

Specific management includes corticosteroids and nebulized epinephrine

Corticosteroids

Dexamethasone should be given to all cases of croup (mild to severe)

Dose: 0.6mg/kg/oral/im/iv single dose (maximum 8 mg), dose may e repeated once more in severe cases

Improvement generally begins within 2 to 3 hours after a single oral dose of dexamethasone and persists for 24 to 48 hours

Nebulished Epinephrine

Recommended for moderate – severe croup

Dose ; 3-5 ml of 1:1000 alrenaline without dilution

Clinical response is sustained for at least 1 h and wanes off by 2 h of administration

Repeat dose is indicated if respiratory distress persists

- Whom to admit?
- Impeding respiratory failure
- Moderate and severe croup

- When to discharge?
- Observation of 3-4 hours is required to see for recurrence of symptoms
- During observation child should not have stridor at rest or chest wall indrawing

5.8 Specific treatment of other causes of UAO

Diagnostic tests should not precede initial stabilization

Bacterial tracheitis	Antibiotics are mandatory IV Ceftriaxone + IV Cloxacillin for 10-14 days Bronchoscopy may be needed in severe cases
Deep neck abscess	Antiobiotics Ceftriaxone + Cloxacillin + Clindamycin Referral to higher centre for imaging and source control
Foreign body	Referral to higher centre for immediate bronchoscopic removal Remember do and don't during referral

Acute anaphylaxis	Correct hypotension Place in Trendelenburg position Give IM epinephrine 0.1 ml/kg (1:1000 dilution) immediately And repeat 2-3 times every 5-15 minutes if needed Nebulise with salbutamol for bronchospasm Cosider Hydrocortisone 10mg/kg (max 100 mg)

5.9 When to refer?

- Patients requiring mechanical ventilation
- In croup, if no improvement after two doses of steroid consider alternative diagnosis
- For imaging and bronchoscopy

5.10 How to refer?

- Secure airway, breathing and circulation
- Ensure resuscitation kit and a BLS trained provider during transport
- Child's peripheral venous access should be secured and patent
- Child's details, history, examination findings, vitals, events which occurred and treatment given should be legibly written in the referral letter
- The parents should be counselled regarding the child's condition and need for referral
- Written, informed consent should be taken from the parents/guardian

- **Remember DON'T'S in UAO**

Protocol for management of acute croup

Identify a case of Croup

- Initial stabilization
- Place child on parent's lap
- Provide position of comfort
- Provide free flow oxygen if required

Assessment of severity (refer box 5.1)

- Immediate airway management in impending respiratory failure

Mild | Moderate | Severe

Give oral Dexamethasoe 0.6 mg/kg

Nebulized adrenaline (1:1000) 3-5 mL once
Five oral/im dexamethasone (0.6 mg/kg)

Send home
Educate parents
About danger signs

Improved
Observe for 4-6 hrs
Discharge if stable

No imprvoment/ worsening
Repeat neb adrenaline (1:1000) 5 ml once
Repeat Dexamethasone single Dose

If still no improvement think of Alternative cause

Pediatrics trauma at primary care level

1.1 Learning objectives

After completion of this section, the participants should be able to

- Perform primary survey of trauma patients
- Initiate resuscitation and stabilization
- Identify the system involved by focused secondary assessment
- Initiate systematic targeted approach in management
- Identify patients who can be managed at primary health care level
- Identify patients who need early referral
- Know how and when to refer?

1.2 How to do initial assessment in the emergency room?

Primary survey: ABCDE approach

Box 1.1 Initial assessment

Airway	Assess the following parameter • Position of the tongue • Stridor yes or no • Trauma to the airway/facial trauma • Secretions/blood/foreign body/dislodged teeth
Breathing	Assess the following parameters • Respiratory rate • Pattern of breathing • Retractions and work of breathing • Auscultate for differential air entry and added sounds like wheeze, stridor, grunt • SPO2 • Chest wall injuries/contusion/rib fracture/flail chest
Circulation	Assess the following parameter • Heart rate • Central and peripheral pulses • Blood pressure

	• Temperature of peripheries • Any active bleeding
Disability	Assess sensorium using Glasgow coma scale Look for focal neurological deficit (refer appendix)
Exposure	Look for any external bleeding, pallor, visible fractures, spinal and neck injury

1.3 What are the steps in initial stabilization?

Airway	• Position patient on a firm surface • Assume cervical injury in every case of trauma and stabilize-spine (refer box 1.2) • Do not use head tilt, chin lift to open airway, use jaw thrust maneuver instead • Suction secretions if present • Insert oropharyngeal airway to prevent tongue fall • Remove visible foreign bodies/dislodged teeth • If above measures fail then intubate (refer box 1.3)
Breathing	• Provide adequate oxygen support (preferable non-rebreathing mask) • Identify tension pneumothorax and perform needle decompression (refer box 1.4)
Circulation	• Apply direct pressure to external bleeding site • Obtain 2 wide bore venous accesses/intraosseous access if IV not possible • Initiate IV fluid therapy (20ml/kg bolus) with warmed crystalloid solution (NS/RL) • If no improvement after first bolus suspect ongoing haemorrhage due to internal organ injury • Give second and if required third fluid bolus but suspicion of internal bleed becomes stronger • Consider blood transfusion if child worsens or is non responder to fluid resuscitation • Transfuse 10mL/kg of type-specific or O-negative warmed pRBCs

	• Maintain urine output • Prevent hypothermia

Box 1.2: Stabilization of cervical spine
Better to assume cervical spine injury in every case of trauma

- Steps of stabilize
  - Immobilization of cervical spine is of utmost importance
  - Move the head and body together
  - Use cervical collar
  - Place pad under shoulder and back

Box 1.3 Indications of intubation

- Unconscious patient of GCS<8
- All manoeuvres to maintain open airway are unsuccessful
- Significant hypovolemia with depressed sensorium
- Perform rapid sequence intubation:
  - Pre-oxygention
  - Atrophine (only in infants) 0.01mg/kg
  - Sedation: Midazolam – 0.1mg/kg
  - Paralysis: Succinylcholine [2mg/kg(<10kg), 1mg/kg (>10kg] or
  - Vecuronium 0.1mg/kg

Box 1.4 Tension pneumothorax

Clinical clues

- Signs of respiratory failure
- Tracheal deviation away from the side of injury
- Hyper-resonant note on percussion
- Unilateral absence of breath sounds
- Cyanosis (late manifestation)
- Obstructive shock

How to treat?
Under water needle decompression: Insert needle just over the top of the third rib in the midclavicular line using a 14-18 gauge needle catheter in infants and small children

1.4 Secondary survey:

- The secondary survey begins once the primary survey (ABCDEs) is completed and normalization of vital functions has been demonstrated with resuscitation.
- Involves two components:
  Appropriate history taking from patient or family or any prehospital personnel detailed head to toe assessment (refer box)

Box 1.5: Components of Secondary Survey

Parameters to assess	**Methods**	**Interpretation**
Levels of consciousness	GCS	<8 : indication for intubation
Pupils	Size, symmetry and reaction to light	Unequal with no reaction to light : CNS injury, bleed (herniation due to mass effect)
Head	Any laceration, depressed fracture	-
Maxillofacial	Any plapable crepitation, mal – occlusion	Facial fracture Soft tissue injury
Neck	Inspect and palpate for any crepitation Auscultate for bruit Palpate cervical spine	Subcutaneous emphysema secondary to airway injury Hematoma-injury to major vein Brut-injury to carotid artery Pain and tenderness of cervical spine – suspect

		injury and stabilize cervical spine
Thorax	Respiratory and cardiovascular examination	Chest wall tenderness and palpable crepitation – subcutaneous emphysema Paradoxical chest movement – Flail chest due to rib fracture Decreased air entry – Pneumothorax Muffled heart sounds with raised JVP – Cardiac tamponade
Abdomen	Per abdomen examination	Generalized tenderness – peritonitis secondary to perforation Visceral injury – splenic hematoma
Pelvis	Symphysis pubis examination Palpate pelvic bone Inspect perineum	Rectal, vaginal or vulval injury Hematuria – urethral injury
Spinal cord	Neurological examination for tone and power	Paresthesia/hyperasthesia Paraplegia/quadriplegia
Vertebral Column	Palpate for tenderness, lateralizing signs	Fracture or dislocation
Extremities	Visual inspection and palpation	Swelling, bruising Crepitation Absent and diminished pulses

1.5 Which cases can be treated in primary health care facility?

- Trauma with minimal abrassions

- Fracture of single bone which can be managed conservatively and the child is hemodynamically stable.
- Trauma in which the child is hemodynamically stable and has normal sensorium.

1.6 When to refer immediately?

- Impaired sensorium
- Need for mechanical ventilation
- Fractures requiring immediate fixation
- Disproportionate pallor suggestive of concealed haemorrhage
- Refractory shock not responding to fluid boluses suggestive of ongoing losses
- Fractures and deep penetrating wounds to an extremity complicated by neurovascular or compartment injury
- Anticipated need for surgical intervention.

1.7 How to refer?

- Ensure secured airway
- Provide adequate oxygen support
- Ensure hemodynamic stability
- Immobilize fractures
- With proper written consent obtained from relatives and explained about the sickness level
- In an ambulance with life savings equipment, drugs and monitors
- With a health care personnel who has been trained in basic life support management.
- Provide appropriate analgesia
- Ensure adequate continuous monitoring facility

1. SHOCK

Learning objectives
After completion of this Section, the participants should be able to
• Identify shock based on clinical parameters
• Differentiate between compensated rind decompensated shock
• Classify based on peripheral perfusion and etiology.
• Initiate fluids and vasoactive therapy as per underlying etiology
• Monitor therapeutic end points
• Look for causes of non-response in refractory shock
• Know when and how to refer?

1.2 What is shock?
• Shock is a clinical syndrome arising out of mismatch between oxygen supply and demand of the tissues

1.3 How to classify shock?
- Shock is classified based on both severity (Box l.1) and etiology (Box 1.2)

Box 1.1: Classification of shock based on severity

Compensated	Decompensated / Hypotensive
Tachycardia Impaired perfusion (cold or warm) Decreased urine output Normal blood pressure (BP>5th centile for age) 5th centile systolic BP is given as Neonates = 65 mmHg Infants (<1 year) = 70 mmHg 1-10 years = 70 + (age x 2) mmHg • > 10 years = 90 mmHg	Tachycardia lmpaired perfusion (Cold and warm) • Decreased urine output Hypotension (BP< 5th centile for age) 5th centile systolic BP is given as • Neonates = 65 mmHg • Infants (<I year) = 70 mmHg • 1·10 years = 70 + (age x 2) mmHg • > 10 years = 90 mmHg.

Box 1.2: Classification of shock based on etiology

Type	Pathophysiology	Disease states
Hypovolemic	Decrease in effective Circulating blood volume (Preload)	Actual intravascular volume loss (diarrhea dehydration, haemorrhage) apparent intravascular volume loss (capillary leak, third space loss; redistribution
Cardiogenic	Myocardial pump failure	Impaired cardiac contractility (myocarditis, cardiamvopathy)
Distributive	Loss of vascular tone and vasdilation	Sepsis (increased capillary leak with loss of intravascular volume)

		Neurogenic shock (acute spinal injury, CNS catastrophe)
Obstructive	Obstruction to cardiac output	RV outflow obstruction (pulmonaryembolism, lung hyperinflation, pericardial effusion) LV outflow obstruction due to critical aortic stenosis

1.4 How to recognize shock?

The recognition of shock is always clinical. The ABCDE approach during primary assessment helps identify shock (Refer Box 1.3)

Box 1.3: Clinical recognition of shock

Airway	Patency	Airway open and maintainable not maintainable	
Breathing	Respiratory rate Respiratory effort Breath sounds	Increased Increased/labored Normal or crepts	
Circulation		Warm Shock	Cold Shock
	Skin	Warm suffused skin	Pale, cool skin
	Heart rate	Tachycardia Bounding pulses	Tachycardia
	Peripheral pulse	Flashed CFT	Weak pulse delayed CFT (>2 sec) Maintained
	Systolic BP	Maintained in compensated, decreased in hypotensive shock	In compensated, Decreased in hypotensive Shock
	Pulse pressure	Increased	Decreased
	Urine output	Decreased	Decreased
Disability	Consciousness	Imitable early/Lethargic late	
Exposure	Temperature	Variable	
	Blood glucose Rash Jaundice		

*Adopted from IAP-ALS handbook

1.5 What are the steps in management of shock?

• Airway: Suction secretions
if patient is obtunded and unable to maintain airway, start bag and mask ventilation, followed by intubation

• **Breathing**: Start oxygen (any delivery device that is available), preferably 100% Fi02.
o In spontaneously breathing patient start with non- rebreathing mask at flow rate of 10-15 L/min
o In apneic or bradypneic patient start bag and mask ventilation followed by intubation.
• **Circulation:**
o Secure immediate vascular access: Select a large vein close to the heart
o Avoid delay in getting a vascular access;establishintraosseous access if there is inability to secure a peripheral venous accessin 3 attempts or 90 seconds (whichever is earlier) (see appendix)

A) Fluids:
• Fluids are given to augment the preload and optimization of preload is the first consideration in therapy of shock, irrespective of etiology.
• Fluids of choice are always crystalloid either Normal Saline/Ringer's Lactate (whichever is available readily).

• **Rate of infusion:**
- If hypotensive: Administer fluids rapidly through push and pull method
- If in compensated shock: Administer fluids over a period of 15 to 20 mins by gravity method
- Be very cautious with fluids in severe malnutrition, anemia and underlying cardiac disease (refer box 1.4)

Volume:
- 20 ml/kg in hypovolemic and septic shock (may require upto 40-60 ml/kg)

Smaller aliquots (5ml/kg) in suspected myocardial dysfunction

• Watch for signs of fluid overload (Increase in tachycardia and liver span new onset crepitations or worsening tissue perfusion)
• Response to fluids is monitored by looking for therapeutic end points **(refer box 1.5)**

Box 1.4 Fluid therapy in specific conditions

- Severe Malnutrition:
- 15 ml/kg over 1 hour
- RL with 5% D, 0.45% Nacl with 5% D

- Cardiogenic Shock
- 5-10 ml/kg slow boluses
- Start on early ionotropic support
- Accept just 5th centile blood pressure

Box 1.5: Therapeutic end points of shock
- Decreased HR, improved tissue perfusion
- Increasing urine output> 1ml/kg/hr

- Normal capillary refill ≤ 2 sec
- Normal peripheral pulses
- Warm peripheries
- Normal level of consciousness
- Reversing hypotension

B) Optimizing cardiac output: Vasoactives are required to augment cardiac output (inotrope) or improve Systemic vascular resistance (SVR). Any child requiring >60 ml/kg fluids (fluids refractory shock) or develops signs of fluids overload during resuscitation requires vasoactive therapy. Children with septic shock require vasoactive therapy of underlying myocardial dysfunction and vasodilation(refer box 1.6).

1.6 How to prepare infusions of vasoactive drugs? (refer appendix section)
BOX 1.6 : Choice of vasoactive therapy

Cold shock		**Warm shock with hypotension**
With normal BP	With hypotension	
• Low dose epinephrine (<0.3 µ/kg/min) • Dobutamine (5-20 µ/kg/min) • Milrinone (50 µ/kg loading over 10-16 min followed by 3-0.75 µ/kg/min)	• Dopamine (5-20 µ/kg/min) • Epinephrine (0.13 -1.0 µ/kg/min) • Norepinephrine (0.01 – 0.1 µ/kg/min) + Milrinone (0.3 – 0.75 µ/kg/min)	• Norepinephrine (0.1 - 20µ/kg/min • Vasopressin (0.0001) – 0.000251 IU/kg/min)

1. 7 How to monitor?
a) Child should be monitored by a nurse atleast every 2 hourly and by a doctor atleast 6hourly
b) What to assess? (Look for all therapeutic end points Box 1.5)

• Heart rate
• Pulses central and peripheral)
• Capillary refill time
• Blood pressure
• Temperature
• Leyel of consciousness
• Urine output

1.8 What is the checklist for non- response to vasoactive therapy (refer box 1.7)

Box 1.7 : Checklist for non response
- Uncorrected preload (volume)
- Recheck: dose, infusion, rate
- Correct dyselectrolytemia : Ca++, Mg+, Glucose

- Calcium (if ionic value not available them do ECG): 2ml/kg of calcium gluconate in equal dilution with D5 over 20 mins under heart rate monitoring (ceiling dose of 10 ml)
- Glucose: Remember product of volume and concentration of dextrose should be 50

- 2ml/kg of 25 % dextrose
- 5ml/kg of 10% dextrose
- 10ml/kg of 5% dextrose

1.9 Specific treatment of shock (refer box 1.8)

BOX 1.8: Specific treatment of shock

Hypovolemic	a) Hemorrhagic • Control external bleeding 20ml/kg NS/RL bolus, repeat as needed • Transfuse PRBC • Look for hidden bleeding source if shock is uncontrolled b) Non hemorrhagic (e.g. diarrhoea, dehydration) • 20mllkg NS/RL bolus, repeat as needed
Cardiogenic	• NS/RL bolus 5-10mllkg over 10-20min (small bolus) • Assess need for positive pressure ventilation Start early inotropes Dopamine/ dobutamine/ epinephrine (can be safely started through peripheral line)
Obstructive	• Tension pneumothorax: Needle thoracentesis (2nd intercostal space) • Cardiac tamponade: Pericardiocentesis Pulmonary embolism: Thrombolytics Duct dependentcirculation: Prostaglandin E1 infusion (0.01 – 0.2µ/kg/min)
Distributive	a) Septic: • As per algorithm b) Anaphylactic: • 0.01 mL/kg of intramuscular epinephrine (1:1000), repeat after 10 – 15 min in severe cases. • Fluid bolus as required • Anthihistamines, steroids

	• If hypotension persists, start epinephrine infusion (0.05 mcg/kg/min) c) Neurogenic: Fluid bolus Vasopressor (norepinephrine/epinephrine)

1.10 What are the causes of refractory shock? (refer Box 1.9)

Box 1.9 Unrecognized morbidities in refractory shock

- Ongoing blood loss and hypovolemia
- Hypoxia
- Acidosis
- Hypoglycemia
- Electrolyte disturbances
- Hypocalcemia
- Pericardial effusion
- Pneumothorax
- Hypoadrenalism
- Inadequate source control (loculated pus, empyema etc)
- Intrabdominal catastrophe (surgical abdomen, abdominal compartment syndrome)

1.11 When to refer?

• Need for mechanical ventilation
• Fluid refractory catecholamine resi stant shock - if PICU not available
• Cardiogenic shock which requires advanced support and diagnostics

1.12 How to refer?

• Refer only after stabilization, ensuring adequate airway, breathing and circulation
• Proper documentation regarding the presentation of the child, details of resuscitative measures taken, interventions done during resuscitation and reasons for referral including name and contact number of referring physician
• A doctor/paramedic trained in Pediatric Advanced Life Support/Basic Life Support to accompany the patient
• Transporting ambulance should have sufficient 02 supply and Resuscitation equipment
• Inform the referral Centre prior to sending the patient regarding the diagnosis of the child, indication for referral, current status and approximate time to arrival
• Counsel the family of the child regarding the need for transfer and risks during transfer. Obtain written, signed consent for the same.

Protocol for management of septic shock

0-5 min	Evalute & Identify	Recognise septic shock fever for hypothermia, Tachycardia, Prolonged/brisk CRT, Altered Mental status with or witout Hypotension	Maintain high index of sus picion
	Intervene Airway Breathing	Start O@ by NRM/HFNC/any other device bag and mask: if apnea/bradypnea plan for early intubation	If grunting/ Severe retraction PEEP will help
	Circulation	Establish IV/IO access Draw samples for CBC, ABG, glucose, electrolytes, culture Administer first dose of antibiotics	As per local epidemiology and antibiogram
	Disability	Establish IV/IO access Draw samples for CBC, ABG, glucose, electrolytes, culture Administer First dose of antibiotics	As per local epidemiology and entibiogram
15-20 Min	Fluids	Start NS/RL 20ml/kg strict monitoring of HR/RR/CRT/BP/SPO2/liver size/chest Signs/UOP/CNS/Temp	if hypotensive: use pull push method if compen-sated shock: Infuse Over 15-20 mins By gravity method
40 mins	Reassess	End Points Reached	Therapeutic Goals Decreased HR, RR, improved perfusion BP>5th Centile, Improved mental status, improved urine output	End points not reached & No fluid overload	S/o fluid overload - Worsening Tachycardia Worsening tachy -pnea Increasing liver size Increased crepts
	Intervene	Continue Monitoring			
			More fluids 2nd bolus 20 ml/kg @ 15-20 min give upto 40-60 ml/kg		Gallop rhytm
		End points reached			Caution with fluid if - Anemia Severe malnutri tion Underlying heart disease
	Reassess Evaalute Identity		End points not reached		
		Persistent shock/Refractory to fluids			
	Intervene Myocardial Support	Start vasoactives Epinephrine for cold shock Nor-epinephrine for warm shock			if S/o fluid overload - stop fluid - intubate & ventilate - start inotropes - UOP monitoring
At 60 Min		Vasoactives Can be given through peripheral line Dopamine can also be started in cold shock with normal BP High dose dopamine if NE is not available			

2. ACUTE GASTROENTERITIS

2.1 Learning objective~

After completion of this section, the participant should be able to

- Identify case or acute diarrhoea
- Grade, severity of dehydration
- Understand principles of fluid therapy
- Counsel parents regarding home management in non- severe cases
- Understand diarrhoea can be a manifestation of systemic illness
- Know when and how to refer?

2.2 What is diarrhoea?

Diarrhoea: Passage of unusually loose or watery stools with an increase in frequency
Consistency of stools is more important than frequency
Dysentery: Diarrhoea with blood in stools associated with abdominal cramps and fever

2.3 What is not diarrhoea?

- Passage of frequent well formed stools
- Passage of pasty stools in breast fed infants
- Passage of stool during or immediately after feeding due to gastrocolic-reflex
- Passage of frequent loose greenish yellow stools on the 3rd and 4th day of life called as transitional stools

2.4 What are the steps in management ?

a) Grade the severity of dehydration: The severity of dehydration can be assessed clinically as given in Box 2.1

BOX 2. Grades of dehydration

Characteristics	No dehydration	Some dehydration	Severe dehydration
1. Condition	Well, alert	Restless & irritable	Lethargic
2. Eyes	Normal	Sunken	Very sunken & dry
3. Tears			
4. Tongue	Present	Absent	Absent
5. Thirst	Moist	Dry	Very dry
6. Skin turgor	Drinks normally	Thirst & drinks eagerly	Unable to drink
7. Extrmities			
8. Pulse quality			
9. Urine output	Normal	Delayed	Very Delayed
10. Estimated	Normal	Prolonged	Prolonged
Weight loss %	Normal	Cold	Weak & thread
ml/kg			Anuria for 6 hr
11. Treatment plan	Normal	Decreased	
	<5%		
	<50 ml/kg	5-10%	>10%
	Plan A	50-100 ml/kg	>100 ml/kg
		Plan B	Plan C

Remember there is an alternate classification of mild, moderate and severe dehydration to assess the severity of dehydration

b) Fluid therapy in diarrhoea has 4 components
a. Resuscitate (Intravascular volume expansion)
b. Rehydrate (Deficit correction)
c. Replace (Ongoing losses)
d. Maintain (Maintenance therapy)

c) Severe dehydration: Children, With severe dehydration and shock need resuscitation followed by replacement. Immediate fluid resuscitation. (For more details refer section B, chapter: 1, point 1.8)

20ml/kg of Ringer's Lactate (RL) or Normal Saline (0.9%) IV or intraosseous (IO) till perfusion improves (quality of central and peripheral pulses, CFT and *blood* pressure) In children with no signs of shock or in those with severe dehydration after adequate resuscitation start Plan C: 100ml/kg RL or NS. This needs to be given over a period of 6 hrs in <12months and 3hrs in > 12 month old child as shown below

	30ml/kg	70ml/kg
< 12 mon	1 hour	5 hrs
1- 5 yr	1/2 hour	2.5hrs

Replace losses:
For <2 years: 50 to 100 ml ORS per loose motion
For >2years- 10 years: 100 to 200 ORS per loose motion
If unable to take orally then give by nasogastric tube or as IV 0.45% NS in 5 % dextrose in same dose

d) Some dehydration: Children with some dehydration need rehydration followed by replacement, Plan B. ORS 75ml/kg over 4 hrs is used as rehydrating fluid.
Replace losses:
For <2 years: 50 to 100 ml ORS per loose motion
For >2years- 10 years: 100 to 200 ORS per loose motion
e) No dehydration: Children just need replacement of losses Plan A. Most can be treated on ambulatory basis at home while some may need a brief period of observation. The indication for observation in Emergency department are listed in

Box 2.2

Box 2.2 Indications of observation in Emergency department
Stable newborns and infants with diarrhea without features of toxicity and dehydration
Moderate dehydration but accepting well orally
Malnourished children with mil dehydration
Patient with diarrhea without dehydration but with decreased oral mistake.

Box 2.3 Indications for hospitalization

- Severe dehydration
- New born and infants <3 months of age with dehydration
- Malnourished children with moderate/severe dehydration
- Toxic appearance, changing mental status (GCS<11) or seizures
- High output diarrhea (>10 large volume stool/day)
- Persistent vomiting, or decreased/no oral intake
- Suspected surgical cause: localizing findings and enterocolitis need surgical consult
- Suboptimal or no response to oral rehydration therapy (ORT) or further deterioration
- Inability of caregivers to administers ORS replacement

g) Adjuvant therapy: Zinc decreases the severity and duration of dehydration
o Dose:< 6 months- 10mg /d for 14 days
o Dose: >6months - 20mg/d for 14 days

h) Nutrition: Continue breast feeding, home based fluids (rice water, soup, yoghurt drink). Avoid drinks sweetened with sugar (e.g. commercial carbonated beverages, fruit juices, sweetened tea, coffee) as they can cause osmotic diarrhoea and hypernatremia

i) Antibiotics are not routinely recommended, unless indications as enumerated in box 2.4 are there.
Box 2.4 indications for antibiotics
Clinical signs of sepsis (toxic look, leukocytosis, fever>38.5 C, septic shock)
Severe malnutrition
Neonates and very young infants (<3 mo) with fever
Dysentery (bloody stools)

2.5 How to monitor?
• Child needs to be monitored by nurse atleast every 4 hourly
• What to monitor?
o Hemodynamic status
o Sensorium
o Hydration status
o Number of stools passed
o Urine output
o Oral intake
o Adequacy of care by mother /caretaker

2.6 When to discharge patient?
• Once rehydration therapy is initiated, assess patient after 2 hours
• Discharge only if -
o Clinical status improving
o Accepting orally
o Normal sensorium
o Adequate urine output
o No indication for observation or admission

2.7 When should mother/caretaker return to health care facility?
• Stool frequency> 10 times/day
• Has sunken eyes, fever
• Not accepting orally
• Not passed urine for 6 hrs
• Abnormal sensorium

2.8 What is parental diarrhoea?
Few systemic illnesses can have diarrhoea as the presenting symptom (refer box 2.5)

Box 2.5 Causes of parenteral diarrhea

- Otitis media
- Bacterial Pharyngitis
- Urinary tract infection
- Pneumonia
- Meningitis
- Bacterial sepsis
- Toxic shock syndrome
- Acute surgical abdomen: Colicky abdominal pain vomiting, lethargy, bloody
- Diarrhea, abdominal distention, and palpable mass suggests intussusception, or malrotation

2.9 When to refer?
• Diarrhoea with fluid refractory shock
• Diarrhoea associated with decreased urine output, abdominal distension, encephalopathy, seizures or any other complications
• Diarrhoea as presenting symptom of systemic illness (Box 2.5)

2.10 How to refer?

- Refer only after stabilization, ensuring adequate, breathing and circulation
- Proper documentation regarding the presentation of the child, details of resuscitative measures taken, interventions done during resuscitation and reasons for referral including name and contact number of referring physician.
- A doctor/paramedic trained in Pediatrics Advanced Life Support/Basic life Support to accompany the patient
- Transporting ambulance should have sufficient O2 supply and Resuscitation equipment.
- Inform the referral centre prior to sending the patient regarding the diagnosis of the child, indications for referral, current status and approximate time to arrival
- Counsel the family of the child regarding the need for transfer and risks during

Remember

- Fluid therapy in diarrhea has 4 domains
  - Resuscitate (intravascular volume expansion
  - Rehydrate (Deficit correction)
  - Replace (ongoing losses)
  - Maintain (Maintenance therapy)

- Use oral route when available
- In a child not responding to fluids first re-check
  - Quantity of fluids
  - Compliance to ORS
  - Ongoing losses
  - Associated sepsis

Transfer. Obtain written, signed consent for the same.

Protocol for management of Acute Diarrhoea

Child with diarrhea

Grade severity of dehydration

No dehydration

Replace losses orally
<2 years: 50 – 100 ml ORS per loose Motion > 2 years – 10 years: 100 to 200 ORS per loos Motion

Supportive treatment:
Nutrition
Ensure adequate calorie intake
Continue breast feeding
Age appropriate normal diet
Zinc supplementation:
Dosage: < 6 months – 10 mg/d, >6 months-20mg/d
Duration 14 days

Some dehydration

Rehydration
75 ml/kg ORS over 4hrs
Replace losses

Severe dehydration

Resuscitation:
20 ml/kg IV/IO RL/NS if in shock till perfusion improves
If no signs of shock then give 100 ml/kg RL
Divided as below

	30ml/kg	70 ml/kg
<12 mon	1 hour	5hrs
1-5 yr	½ hour	2.5 hrs

Replace losses
If unable to take orally then give by nasogastric tube or give IV 5% Dextrose in ½ NS in Same dose

3. CARDIAC ARREST

3.Learning Objectives
After completion of this section, the participants should be able to
• Understand the sequence of events leading to a cardiac arrest
• Early identification of pre-arrest risk factors
• Timely intervention to prevent cardiac arrest
• Immediate identification of cardiac arrest and the contributory rhythms
• Components of high quality CPR
• Algorithmic management of cardiac arrest

3.2 What are the causes of cardiac arrest?
• Most common cause of arrest in infants, children and adolescent is hypoxia resulting from respiratory failure or shock
    o These usually occur with prior warning signs (such as signs of respiratory distress and/or shock).
    o Initially the child will compensate for hypoxia by increasing respiratory rate, work of breathing ,heart rate and cardiac output
    o Once these compensatory mechanism get exhausted, thereis a rapid downhill progression to cardiopulmonary failure and cardiac arrest
    o Therefore it is important to recognize and treat children in respiratory distress and shock before they progress to cardiopulmonary failure and cardiac arrest
• Rarely cardiac arrest may occur without prior warning signs as in cardiac arrhythmias *(VF/*pulseless VT). The risk of arrhythmias increases in the following conditions
    o Congenital heart defects
    o Myocarditis and coronary artery abnormalities
    o Electrolyte abnormalities
    o Long QT syndrome

3.3 How to indentify conditions that cause cardiac arrest ? (refer box 3.1)
a) Certain finding in the assessment need to be identified and corrected immediately so that cardiac arrest is prevented.

Box 3.1

Parameters	**Finding**
Airway	Not maintainable, complete or severe airway obstruction
Breathing	Bradypnea, tachypnea, gasping, irregular breathing, seesaw breathing Increased respiratory effort (severe chest retractions, head bobbing) Stridor, grunting SPo2 <94% in room air, SPO2 <90% on 100 % O2
Circulation	Bradycardia, tachycardia, irregular rhythm Weak/absent peripheral or central pulses CRT>3 sec Cool, pale, dusky, mottled extremities Hypotension
Disability	Decreased level of consciousness (GCS), hypotonia, generalized seizures, abnormal pupillary response
Exposure	Trauma, significant bleeding manifestation

b) Similarly some reversible causes (refer box 3.2) always need to be looked at and corrected for prevention as well as during treatment of cardiac arrest

Box 3.2 : Reversible causes of cardiac arrest

H's	T's
Hypovolemia Hypoxia Hydrogen ions (acidosis) Hypoglycemia Hyperkalemia/Hypokalemia Hypothermia Hyperthermia	Tension pneumothorax Tamponade Toxins (drugs, poisons, anaphylaxis) Thrombosis in pulmonary artery Trauma Prolonged QT interval

3.4 How to indentify a child in cardia arrest?
Unresponsiveness
Not breathing or gasping and
No central pulses (should not take more than 10 seconds to assess the pulse)

3.5 How to manage cardiac arrest?

Goal is to achieve return of spontaneous circulation (ROSC) which is described as resumption of organized cardiac electrical rhythm with palpable central pulses

This can be achieved by

High quality CPR which includes chest compressions and assisted breaths (refer box 3.3)

- Advanced life support measures (refer box 3.4)
- Advanced airway management
- Establishment of vascular access
- Defibrillation
- Medications
- Continuous monitoring

Box 3.3 Components of high quality CPR

- Push hard – at least 1/3rd of anterior – posterior chest diameter, about 1.5 inches (4cm) in infants and 2 inches (5cm) in children
- Push fast – at least 100 compressions/min
- Allow complete recoil of chest after each compression
- Minimize interruptions - <10 seconds for ventilation
- Avoid hyperventilation

To identify rhythms those have caused cardiac arrest. There are four rhythms which can be cause pulseless arrest

Asystole
Pulseless Electrical Activity (PEA)
Pulseless ventricular Tachycardia (pVT)
Ventricular Fibrillation (VF)

Of these the first two are treated with effective CPR and epinephrine and hence known as non shockable rhythms whereas the last two require effective CPR and defibrillation (unsynchronized shock) and known as shockable rhythms

Advanced airway and breathing management	After opeining the airway provide effective ventilation using bag and mask and consider using advanced airway i.e. endotracheal tube or Laryngeal mask (refer appendix airway adjuncts)
Establishing vascular access	To deliver drug directly into circulation Routes used in order of preference are Intravenous Intraosseous (refer appendix) Endotracheal (ET): Used only if any of the above access is unavailable Durgs given by ET route; Vasopressin, Lidocaine, Epinephrine, Atropine and Naloxone (V-LEAN) Deliver the drug when chest compressions are being performed followed by 5 ml normal saline flush every time. Withhold the compressions during ET administration of the drug followed by 5 ml normal saline flush and provide 5 rapid manual breaths.
Defibrillation	Refer appendix
Medications	Drugs used in treating cardiac arrest: Epinephrine Amiodarone, Lidocaine and Magnesium sulfate Calcium, atropine and sodium bicarbonate are not recommended in cardiac arrest as they do not improve survival
Continuous monitoring	At least heart rate, respiratory rate, SpO2, blood pressure, rhythm

3.6 How to manage different rhythms in Cardiac arrest ?
Every patient in cardiac arrest, must be connected to a monitor to identify rhythm. The EEG will show one of the four rhythms as described above. The Management of cardiac arrest follows an algorithm as shown in Fig E.

A. Asystole:
• It is defined as complete absence of electrical activity of *heart* and is reflected as a flat line on ECG (Fig *A).* It should be always corroborated by an absent central pulse.
• It is managed with CPR and epinephrine

A: Asystole

B. Pulseless electrical activity:
• PEA is defined as some electrical activity seen on the ECG/ monitor (Fig B) other than VF/VT but without a palpable central pulse
• In other words, there is some electrical activity but no myocardial contractions and cardiac output; hence no central pulse is felt
• The ECG rhythm may be
o Normal or wide QRS complex (with lower than normal rate)
o Prolonged PR or QT interval or
o Low or high amplitude T waves
o Complete heart block

• This condition is always secondary to underlying reversible causes
• Management includes effective CPR, advanced airway, epinephrine and identifying and treating the underlying cause

Fig B: Pulseless electrical activity

C. Ventricular Fibrillation:
• It is a life-threatening cardiac arrhythmia characterized by disorganized myocardial contractions that results in absent cardiac output and central pulse.

• ECG does not have identifiable QRS complex. Instead small or coarse fibrillatory waves are seen (Fig C)
• This rhythm is uncommon in children and more common in adults with myocardial infarction
• Management includes effective CPR and prompt defibrillation (unsynchronized shock)

Fig C: Ventricular Fibrillation

D. Pulseless Ventricular Tachycardia:
• Ventricular Tachycardia(VT) is a wide QRS arrhythmia and may nor may not be associated with a central pulse
• Management includes effective CPR and prompt defibrillation. .'
(unsynchronized shock

3.7 When to terminate CPR?

- It can be discontinued if there is no ROSC at any time during 30 minutes f cumulative advanced life support.
- Resuscitation efforts can be extended in the following conditions
- Drug overdose
- Sever pre-arrest hypothermia (e.g. near -drowning in cold water)
- Toxin exposure
- Electrolyte abnormalities

Remember

• Most common cause of arrest in infants, children and adolescent is hypoxia resulting from respiratory failure or shock
• Early identification and effective treatment of respiratory failure and shock can prevent cardiac arrest
- Once cardiac arrest is identified, start high quality CPR
- Always look for and correct reversible factors
- Cardiac arrest does not always mean asystole., there are other rhythms associated with cardiac arrest e.g VF, pVT and PEA
- Asystole & PEA require effective CPR & epinephrine (non-shockable rhythms)
- Pulseless VT & VF require effective CPR & defibrillation (Shockable rhythms)

Algorithm For management of cardiac arrest

1. Verify scene safety. Assess responsive & look for evidence of etiology e.g. drug, poisoning or other
2. If unresponsive, shout for help (Activate emergency response system if witnessed collapse)
3. Look for normal breathing and check carotid/brachial pulse simultaneously within 10 second
4. Start CPR if cardiac arrest (unresponsiveness, not breathing or only gasping and no central pulses felt
5. Attach and power on AED or manual defibrillator. Check rhythm

6. VF/pVT
8. Give shock

9. Resume CPR immediately Without pulse check. Give CPR for 2 min. Give epinephrine every 3 to 5 min interval.
10. Check rhythm
11. Give shock if VF/pVT

13. Resume CPR immediately without pulse Check. Give CPR for 2 min Give Amidarone

7. Asystole/PEA

14. Resume CPR immediately. Give CPR for 2 min give Epinephrine every 3 to 5 min interval.

12. Organized rhythm: Pulse present :ROSC Give post cardiac arrest care

Medications

Epinephrine:

IV/IO	Endotracheal
1:10,000 Strength 0.1 ml/kg. Repeat every 3 to 5 min.	1:1000 strength 0.1 ml/kg

Amiodarone:

IV/IO	Endotracheal
5 mg/kg bolus. May be repeated up to 2 times for refractory VF/pVT	Not recommended

Lidocaine:

IV/IO	Endotracheal

Important Instructions:

- During CPR, Bag and mask ventilation should
- Preferably by given using 100 % oxygen.
- Take vascular access – IV/IO
- Prepare and consider for advanced airway if multiple rescuers available.
- Identify the reversible cause. 8H's and 8T's
- Talk to relatives if additional staff available

AIRWAY INTERVENTIONS

AIRWAY ADJUNCTS

Learning objectives

- Familiarise with types of airway adjuncts
- Indications and insertion of different airway adjuncts

I. Oropharyngeal airway:

- Pediatric patients have a relatively larger tongue for the size of the oral cavity hence airway obstruction is common especially if child is comatose
- Orapharyngeal airway prevents airway obstruction by displacing the tongue forward and keeping soft hypopharyngeal structures away from the posterior pharyngeal wall
- It provides a conduit for airflow from mouth to pharynx
- It facilitates suctioning of the pharynx
- It also prevents teeth grinding, tongue biting that causes occlusion of endotracheal or oral gastric tubes.

Indications:

- Used in unconscious children with absent cough and gag reflex to maintain a clearand unobstructed airway

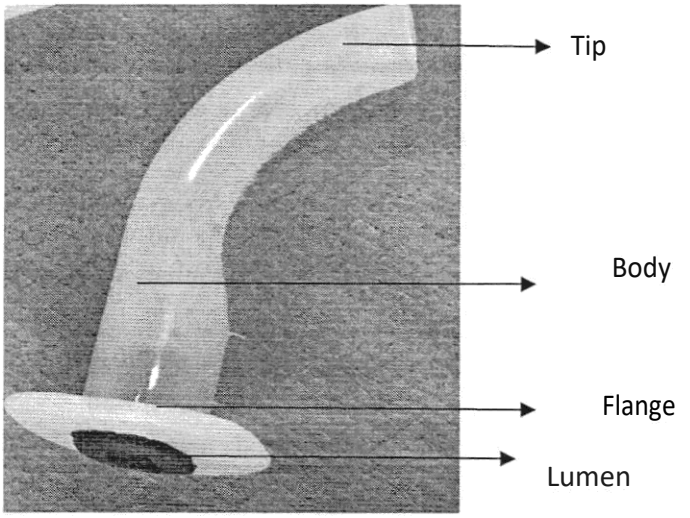
Contraindications :

- If placed in an awake patient it will induce vomiting and aspiration

Parts of an Oral Airway:

The oral airway has four parts:

1. **Flange:** It protrudes from the mouth and rests on the lips. The design pro .ects against aspiration into the airway.
2. **Body:** It is the part that curves over the tongue.
3. **Tip:** It is the distal most part of the airway toward the base of the tongue.
4. **Lumen :** It enables passage of a suction catheter through the central core



'Oropharyngeal airway

Size of the Oral Airway:

- Measure the distance from central incisors to angle of mandible to select the appropriate size of oral airway
- Various available sizes are 0, 1, 2
    - Size 0 1 to 3 years of age
    - Size 1 upto 6 years of age
    - Size 2 upto 12 years of age and above
- Too large airway — May obstruct the larynx and traumatize laryngeal structures
- Too small airway - Can push the tongue posteriorly and obstruct the airway

Steps of insertion of an oropharyngeal airway (OPA)

1. **Position patient:** Supine or semi fowler position is preferred unless contra-indicated. Extend the patient's neck with head tilt, chin lift or jaw-thrust maneovere
2. Use a tongue blade to hold the tongue on the floor of the mouth
3. Remove any foreign body if visible in the mouth.
4. Hold oral airway with concave side downward
5. Gently glide airway following curvature of the tongue
6. It can also be introduced upside down and gently rotated to proper position, to pull the baseof the tongue forward
7. Do not exert pressure on palate
8. Secure the airway with tape and reassess the respiratory status of the child.

9. **Confirm the correct placement** by checking the air entry
10. **For removal :** pull the oral airway out and downward towards patient's chin in one swift movement without rotating

ii. Nasopharyngeal airway (NPA)

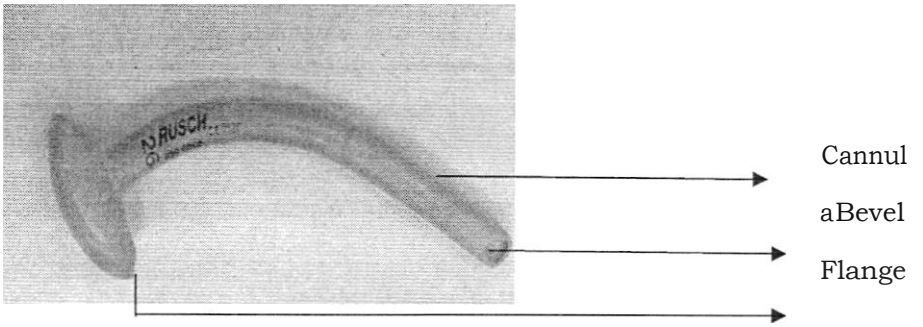
- NPA is flexible and designed to open a channel between the nostril and the nasopharynx
- Purpose is to bypass upper airway obstruction at the level of the nose, nasopharynx or baseof the tongue.
- It primarily acts as a splint and maintains patency of the airway and prevents airway obstruction by keeping the tongue from falling back on the posterior pharyngeal wall.
- It requires adequate training and practice more than OPA

- **Indications :**
  - Maintain open airway
  - Support the upper airway post trauma or surgery
  - In children with congenital maxillofacial abnormalities, syndromic craniosynotosis,mid-facial hypoplasia

- **Contraindications:**
  - Patient prone to epistaxis
  - Patient with obstructed nasal passage
  - Facial or head trauma where basilar skull fracture or cranial vault communication is suspected.

- **Advantages of NPA:**
  - Increased tolerance in conscious patients
  - Stable airway positioning for long periods of time.
  - Decreased incidence of gag-reflex stimulation.
  - Insertion in clenched-jaw situations.
  - Minimal incidence of mucosal trauma during frequent suctioning.

- **Parts of naso-pharyngeal airway**
  - **Flange:** Wide trumpet-like end that prevents further slippage into the airway.
  - **Cannula :** Hollow shaft of cannula that permits airflow into hypo-pharynx.
  - **Bevel (Tip):** Opening at distal end of tube that will be seen resting posterior to the base of the tongue.



- **How to determine size of naso-pharyngeal airway:**
  - External diameter of naso-pharyngeal airway should be slightly smaller thanthe patient's external nares.
  - Length is determined by measuring distance from tip of the nose to the tragus of the ear.
  - In case of non availability of a naso-pharyngeal airway, endotracheal tube of similar diameter size can serve the purpose
  - NPA one size smaller should always be kept ready

- **Insertion of a nasopharyngeal airway**
  - Position patient: Supine or high-fowler position is preferred unless contra-indicated.
  - Prefer right nostril unless contraindicated
  - Lubricate the tip and outer cannula of NPA with water-soluble lubricant.
  - Remove excess secretions from nares.
  - Insert NPA gently into nostril and thread the tube along the floor of the nasal passage perpendicular to the face until the flange is at the tip of the nose.
  - In case of resistance rotate the tube and continue gentle forward pressure ,do not force
  - If resistance persists withdraw tube and try the other nostril.
  - Verify patency of airway
  - Secure the naso-pharyngeal airway with tape

- If unable to insert the NPA, consider:
  - Use of smaller size NPA
  - Insertion in OT under direct vision and general anaesthesia

- **Immediately after insertion observe for**
  - Excessive bleeding from the nostril

- Patency with suctioning
- Improvement of respiratory symptoms

III. Endotracheal intubation:

Indications:

- Maintain open airway
  - Obstructive pathology : Severe croup, diphtheria, trauma
- Protect from aspiration in patients with impaired glottis reflexes
  - Altered mental status
- Provide ventilation

Checklist for ET intubation
:

- Universal precautions —Gloves, masks, eye protection
- Monitors : Cardiac monitor, pulse oximeter, blood pressure monitoring
- Intravenous and intraosseous infusion equipment
- Oxygen supply, bag — mask (appropriate size)
- Suctioning equipment
- Oral/nasopharyngeal airways
- ET tubes with stylets (cuffed/uncuffed)
- Laryngoscope :No definite rule for blade selection
  - Straight blade: preferred in <2 yrs and difficult airway
  - Curved blade: > 2yrs
- Position patient :
  - Sniffing position : mouth ,pharynx, trachea aligned for direct visualization ofglottis
  - a < 2 yrs a towel or roll under the shoulders might be required i/v/o large occiput
- 3,5,10 ml syringes to inflate cuff
- Adhesive

Selection of tube:

Age of the patient	• >8 yrs : Cuffed tubes • <8 yrs : Uncuffed tubes • Correct tube placement prevents air leak around the tube
Size	• Uncuffed tubes: Age in years/4 + 4 • Cuffed tube: Age in years/4 + 3 • Two or more ET tubes with internal diameters of ID 0.5 mm smaller and bigger than age appropriate must be kept ready during intubation
Depth of insertion	• (Age in years/2) +12 • Internal diameter x 3
Cuff pressure	Maintain cuff inflation pressure < 20 cms HCO
Confirmation of ET tube placement	**Primary confirmation -** • Symmetrical chest rise • Mist in the tube with breaths • **Bilateral equal breath sounds** • Absence of breath sounds over upper abdomen **Secondary confirmation** - • Capnographs: electronic monitors that display o Change color in the presence of COC o Yellow in the presence of CO2. and purple or bluecolor when CO_2 is not detected. • Improvement or sustained good oxygen saturation

Red flags:

What to think in case of sudden deterioration in an intubated patient:

- Displacement of tube
- Obstruction of tube by secretions, mucus or kinking of tube
- Pneumothorax
- Equipment failure: Leak in ventilator circuit, malfunction in power supply

Intervention to be done:

- ✓ Support oxygenation and ventilation : start bag and tube ventilation if child is on ventilator
- ✓ Assess : Symmetrical chest movements ,heart rate
- ✓ Tube block : Try suction or remove the tube and start bag and mask ventilation
- ✓ Sedative and analgeiscs to reduce child's agitation should be used only afterruling out correctable causes
- ✓ If pneumothorax is detected —Needle thoracocentensis

Head Tilt — Chin Lift Maneuver

Place one hand on the child's forehead and gently tilt the head back into a natural position, the neck should be slightly extended at the same time, place the fingertips of your other hand under the bony part of the child lower jaw near the point of the chin, lift the mandible upward and downward to open the airway.

Be careful not to close the lips and mouth or push on the soft tissues under the chin which might obstruct the airway

If secretions, vomitus or a foreign body is visible, remove it.

Jaw Thrust Maneuver

When neck or head injuries are suspected, the jaw thrust maneuver with spinal imrrobilization should be used to open the airway.

Place two or three fingers under each side of the lower jaw at the angle and lift the jaw upward and outward. Your elbow may rest on the surface on which the victim is lying.

BREATHING INTERVENTIONS

BAG AND MASK VENTILATION

- Bag and mask ventilation is an essential emergency skill
- This basic airway technique allows oxygenation and ventilation of patients until more definitive airway can be established

- **Indication:**
    - Child with apnea, bradypnea or irregular breathing despite having an open orpatent airway
    - Respiratory failure
    - Failed intubation

- **Contraindications:**
    - Presence of complete airway obstruction
    - Relatively contraindicated after paralysis and induction due to increase riskof aspiration
    - Spontaneously breathing patient

Equipment required:

1. Face mask —
    - Appropriate size creates a good seal and aids in effective ventilation
    - Mask should have soft rim and extend from bridge of the nose to the cleft of the chin and cover nose and mouth but not eyes
    - It should be transparent so that color of the child's lips, condensation and regurgitation can be observed
2. Ventilation bag

a. Self-inflating bag (Ambu bag)

- Can provide positive pressure ventilation without a compressed gas source
- Volume: Comes in various sizes
    - 300ml —neonates
    - 550 ml - infants and young children.
    - 1000 -1500 ml adolescents and older children,
- Flow rate
    - 10-15 L/min- pediatric bag

- Atleast 15L/min - adult bag

- **FiO2 delivered:**
  - Without an oxygen reservoir-30-80%
  - With an oxygen reservoir- 60-95%.

Different valves :

- Intake valve — allows bag to fill with oxygen or room air. This valve closes when the bag is compressed thus restricting the flow back from the inlet
- Outlet valve allows oxygen to flow out if pressure is excessive
- Non-re breathing valve- directs flow of oxygen to the patient and prevents exhaled gas re-entering the bag
- Pressure release valve : is set at 30 -40 cm of H20 and should always be tightened
- Contraindicated in a spontaneously breathing patient

B. Flow inflating bags (anaesthesia bag) —

- Needs more expertise to operate therefore should be used only by trained and experienced providers
- Requires flow of oxygen from a compressed source and also has flow control valve to regulate pressure inflation
- Can inflate only if tight face mask seal is maintained

- **Technique of bag and mask ventilation:**

- One hand required for positioning head, maintaining airway patency and maintainingface seal, other hand for ventilation
- Position the child
- Sniffing position if cervical spine is normal to open the airway
- Do not hyperextend the neck
- Infants & toddlers - may require padding under the shoulder
- Children >2 years of age — may require padding under occiput
  - Maintain the face mask seal using thumb and index finger (E-C technique)
  - Place middle finger under mandbular symphysis
  - Ring and little finger under angle of mandible
  - If possible keep mouth open inside the mask, either manually or by using an oropharyngeal airway
  - Ventilation: deliver breaths by squeezing the bag. Deliver each breath over 1 second.
  - If patient has intrinsic respiratory drive assist patient's breath
  - Ventilate with low pressure and low volume to avoid gastric distention

- Assessment
  - Chest rise with each breath
  - Good bilateral air entry
  - Heart rate
  - 0_2saturation
  - Exhaled CO_2

❖ **Corrective measures to be tried in case of poor ventilation**

- Reposition/reopen airway
- Verify mask size and ensure tight seal
- If required place an oropharyngeal or nasopharyngeal airway
- Suction and clear airway
- Check O_2 source & flow

❖ **Complication due to excessive ventilation**

- Decreased venous return and reduced cardiac output, coronary perfusionand cerebral blood flow
- Volutrauma and barotrauma in children with small airway obstruction
- Increased risk of aspiration

Insert pie of bag and mask ventilation being done or any other relevant

OXYGEN DELIVERY DEVICES

Oxygen delivery system consists of:

1. Oxygen source
2. Pressure regulator and flow meter
3. Oxygen delivery device
4. Patient

Oxygen delivery system:

- Administers, regulates, and supplements oxygen to patient to increase the arterial oxygenation
- It takes atmospheric air and oxygen to prepare a fixed concentration required for administration
- Tubing carries the oxygen from the regulator/flow meter to the delivery device
- Oxygen delivery devices are classified into low flow and high flow whether
    - Oxygen flow through the delivery device is able to(high flow) or unable to(low flow) meet the inspiratory flow requirement of the patient
    - Air is entrained (low flow) or not entrained (high flow) from surrounding tomeet the flow requirement of the patient, respectively.

Low Flow Oxygen Delivery Systems:

Salient features:

- Used when child requires low FiO2 and is relatively stable
- Oxygen flow to the delivery device is less than the child's inspiratory flow rate
- This results in entrainment of atmospheric air , mixing and dilution of the deliveredFiO2
- Provides a variable inspired oxygen concentration of about 22% - 60°â
- Cannot be used in patient with poor efforts, apnea , severe hypoxia

Devices :

a. Nasal cannula :

- ❖ Delivers 24-44% FiO2
- ❖ Acceptable flow rate

- 2 LPM in infants/children < 2 years of age
- 4 LPM for children > 2 years of age
- With the above flow rates, humidification is not usually required

❖ **Indications:**

- Low to moderate oxygen requirement in a patient with no or mild respiratory distress

Remember:

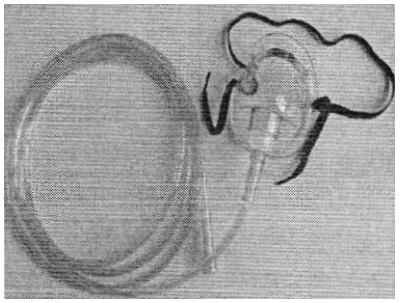
The prong size should be approximately half the diameter of the nares

Available in different sizes: Infant, intermediate infant , pediatric ,adult

Secure the nasal prongs on the patient's face with adhesive tape

b. Simple mask :

- Delivers 40-60% oxygen
- Flow rate required is 6 -101/min
- Flow < 4 L /min results in re breathing and carbon dioxide retention
- Perforations in mask act as exhalation ports
- Vents in the mask allow for the dilution of oxygen
- Indications:
  - Medium flow oxygen desired, mild to moderate respiratory distress



Remember:

Select a mask which best fits from the child's bridge of nose to the cleft of jaw, and adjust the nose clip and head strap to secure in place

No pressure point or damage to eyes

High flow oxygen delivery system:

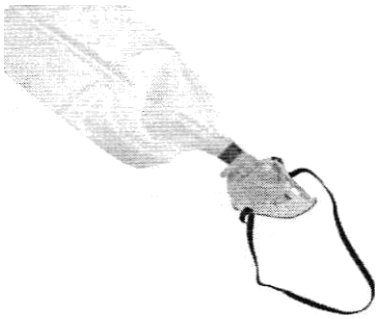
Salient features:

- Deliver an oxygen concentration >60%
- Flow rate should be at least 10 L/min
- High flow systems must be used in emergency settings : respiratory distress or shock
- Suitable for spontaneously breathing patients with severe hypoxia

Devices:

A. Non re breathing mask:

- Delivers 90-100 N o oxygen
- Flow rate required is 10-15 I/min
- Face mask + oxygen reservoir , a valve at exhalation port , a valve between reservoir and mask
- Patient inhales oxygen from the bag and exhaled air escapes through valves on the side of the mask



Remember:
Oxygen flow into the mask is adjusted to prevent the collapse of the reservoir (12 L/min)
Flow rate must be sufficient to keep bag 1/3 to 1/2 inflated at all times
A valve in one or both exhalation port(s) to prevent entrainment of room air during inhalation
Do not use with humidification system as this can cause excessive 'rain out' in the reservoir bag

B. Oxygen hood :

- Delivers FiO2: 80-90%
- Flow rate @ 10-15 L/min
- Adequate flow rate to prevent re breathing of CO2
- Ensure the head box has a gap all around the child's neck

Remember

- 3-4 sizes are available
- Too big will dilute the oxygen;
- Too small will cause discomfort and CO, retention

NEBULISATION

- Nebulisation is a method of converting a medicine or solution into an aerosol, which is inhaled directly into the lungs.
- Nebulizers are indicated in
    - Too ill or too young to use handheld devices
    - In cases where large drug doses are necessary

❖ **Indications**

- Acute severe asthma (β2 agonists, anticholinergics)
- Croup (Epinephrin e, Steroids)
- Cystic fibrosis (Antibiotics)
- Before physiotherapy to loosen secretions
-

❖ **Equipment**

- Type of nebulizer : Jet or ultrasonic
- Parts of nebulizer sets:
    - Chamber- where solution should be put
    - Mouthpiece or mask
    - Plastic tubing connected to oxygen/gas souce

❖ **Technique**

- **Position:** Child should sit upright or in head up tilt position.
- Calculate dose of drug
- Unscrew the top of the chamber and fill with the drug and saline to make a total volume of 3-4 ml
- Connect the nebulizer mask/ T piece

  Connect the plastic tubing to oxygen source at flow rate of 6-8 L/min especially if patient is oxygen dependant or in acute respiratory distress
- Treatment is continued for 8-10 mins maximum 15 min till the chamber is empty and no mist flows from the mask/T piece.

a When the aerosol becomes less apparent, but still some solution is left in the chamber, gently tap the chamber

❖ **Post-procedure cleaning of equipment**

- Disconnect the nebulizer set from tubing and flow meter
- Take apart the chamber and mask and ensure all residual solution is discarded
- Wash the three parts of the chamber in warm water
- Rinse all three parts well and remove excess water

- Leave the nebulizer parts in a clean area to air dry

❖ **Disadvantages**

- Expensive for home
- treatment Longer set-up and delivery time
- Decreased portability
- Variable nebulizer performance
- Need for a source of compressed air or oxygen in very sick patients

PULSE OXIMETRY

Non invasive method of measuring oxygen saturation (SpO_2) i.e haemoglobin saturated with oxygen

❖ **Indications:**

- All children with respiratory distress or failure should be continuously monitored with pulse oximetry
- During transport of critically ill child

❖ **Parts of an oximeter:**

- Probe which is attached to the child's finger, toe or ear lobe
- Other end of the probe connected to the monitor (oximeter)
- The unit displays SpO_2 in percentage
- Most monitors make an audible sound for each pulse beat and display heart rate

❖ **Limitations:**

- Erroneous low readings:
  - Hypoperfusion of the extremity : severe shock
  - Hypothermia
  - Motion, shivering

- Nail polish
- Incorrect sensor application misalignment of sensor with light source

- Gives no information about ventilation and CO2 status
- Gives no information regarding blood oxygen content; Severe anemia : though hemoglobin is 100% saturated, total blood oxygen content will be less
- Not a substitute for blood gas as it does not detect pH ,bicarbonate, Co2 levels

Remember :

- Pulse oximetry should always be used in conjunction with clinical assessment and signssuch as respiratory rate, respiratory effort and level of consciousness
- If heart rate displayed on the monitor does not correspond to the child's heart rate, the oximeter may not be functioning properly

CIRCULATORY INTERVENTIONS

INTRAOSSEOUS LINE

- The marrow cavity contains a non-collapsible venous plexus which can be utilizedfor resuscitation during emergency situations, when peripheral veins are collapsed
- Intraosseous(IO) access can be used for delivery of fluids, medications during pediatric resuscitation
- It is safer, faster, requires less skill and associated with fewer complications,

❖ **Indications:**

- Recommended technique for vascular access in cardiac arrest
- In decompensated shock , if vascular access is not getting rapidly established (3 attempts or > 90 sec for IV access)

❖ **Contraindications :**

- Infection at the proposed puncture site (relative contraindication)
- Osteopetrosis
- Osteogenesis imperfecta
- Ipsilateral fracture of the extremity } Can cause extravasation
- Attempted placement in same site } & risk of compartment syndrome

Equipment

- Intraosseous (IO) needle:
  - Should have a needle stylet to reduce clogging of needle by bony spicules or clot
  - Should have means to gauge distance to which needle can be penetrated,either with markings on the shaft or a covering flange
  - Once the bony cortex has been penetrated, the needle usually need not be advanced more than 1 cm to provide stability and access to the m arrow cavity.
  - Manual or electric needles (especially available for intraosseous insertion)
  - 18G needle with trocar (at least 1.5 cm in length)

- Bone marrow biopsy/aspiration needle can be used
  - Alcohol swabs
  - 5 ml ,20 ml syringe

❖ **STEPS OF INSERTION:**

- Prepare skin
- Identify appropriate site
  - Proximal tibia: Anteromedial surface, 2-3 cm below the tibial tuberosity
  - Distal tibia: Proximal to the medial malleolus
  - Distal femur: Midline, 2-3 cm above the external condyl
- Local anaesthesia may be required if patient is conscious.
- Insert needle through skin, and then with a screwing motion perpendicularly / slightlyaway from the growth plate into the bone. There is a give as the marrow cavity is entered
- Remove trocar
- Confirm position

I. Needle stands erect

II. Aspiration of bone marrow particles (sometimes may not be possible)

III. Fluids can be flushed through it

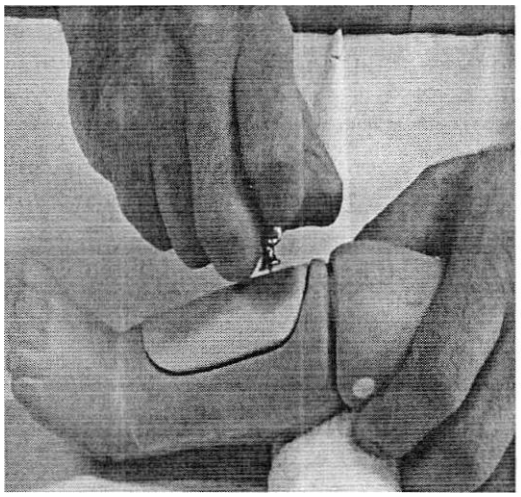
Failure of effective IO placement (check list)

- Incorrect identification of landmarks
- Clogging of needle ,can be avoided by frequent flushing of the needle
- Through-and-through penetration, fractures caused by excess force leading toextravasations and compartment syndrome
- Incomplete placement causing subcutaneous or subperiosteal infiltration

Complications :

- Pain and Local hematoma
- Local infection (proportional to duration of use)
- Compartment syndrome secondary to fluid extravasations

- Very rare : Fat embolus ,Bone embolus, growth plate injury



Remember:
In practice, needle should be removed as soon as another means of vascular access is available, ideally within 6-12 hours.
Longer the needle remains in place, the greater the risk of infection and dislodgment

CARDIOVERSION AND DEFIBRILLATION

- Cardioversion and defibrillation are important skills needed as a part of resuscitation.
- It is a method of delivering electrical shock (energy) to the heart through the chest wall which produces simultaneous depolarization of mass myocardial cells and restore's heart's normal rhythm.
- A defibrillator is required to deliver electric shock, two types of shock are :

A. Synchronized cardio version:

- Shock is delivered to coincide with R wave of the patient's QRS complex this corresponds to ventricular depolarization phase.
- Cardio version requires less energy dose (0.5J/kg first dose) as there is already an organized electrical activity going on and central pulse is present
- Indicated in:
  - I. Tachyarrhythmia with pulse
  - II. SVT with pulse
  - III. VT with pulse Technique

- Technique (Cardio version):

- Use universal precautions. Always wear gloves
- Gain access to the patient's chest by cutting off their upper clothing
- Pads position
  - I, Anterior-lateral position: One pad over middle of the anterior chest nndother pad over apex.
  - II. Anterio posterior position in infants and smaller children
- As soon as the pads have been applied adjust doses to be delivered

B. Defibrillation (Unsynchronized shock):

- There is no synchronization with the QRS complex. In fact such rhythms willnot have organized electrical activity
- Patient will be in pulse less arrest. Dose therefore required will be higher (2J/kg first dose)

- Indicated in pulseless arrest due to ventricular fibrillation and ventriculartachycardia

❖ **Technique :**

- Use universal precautions. Always wear gloves
- Gain access to the patient's chest by cutting off their upper clothing.
- Position defibrillation pads correctly on patient by applying conductive gel
    - Use anterior-lateral position.
    - One pad over middle of the anterior chest and other pad over apex.
- Press charge on the defibrillator. Ensure no one is touching the patient (this means that paddles are charged and ready to be used)
- Once the defibrillator is fully charged, it will alarm.
- State firmly in a clear and loud voice
- Compressions cease
- Everyone clear, oxygen clear
- Shock on count of three
- On the count of three after ensuring that everyone is clear, press the shock button or press both paddle discharge buttons simultaneously. Hold paddles in place till shock is delivered.
- Check monitor, if tachycardia persists, prepare for cardio version again by increasing the energy dose.
- In case of defibrillation (pulse less arrest) resume high quality CPR immediately after shock

TYPES OF FLUIDS

Composition of different crystalloids

Crystalloids	Concentration in mEq/L								g/100 ml	mOsm/L
	Na+	K+	Cl-	Ca2+	Mg2+	Lactate	Acetate	Gluconate	Dextrose	Osmolarity
0.9% NaCl	154	0	154	0	0	0	0	0	0	308
Lactated Ringer's	131	5	111	2.7	0	29	0	0	0	273
Hartmann's	129	5	109	4	0	29	0	0	0	278
Ringer's Acetate	130	5.4	112	0.9	1	0	27	0	0	276
PlasmaLyte	140	5	98	0	3	0	27	23	0	280
Dextrose 5%	0	0	0	0	0	0	0	0	5	250

*Adapted from IAP-ALS Handbook, First edition 2018

Preparation of IV fluids:

N/2 saline	50 ml NS + 50ml distilled water
N/4 saline	25 ml NS +75 ml distilled water
N/6 saline	10 ml NS +50 ml distilled water 18.5 ml NS+81.5 ml distilled water
N/2 -5%dextrose	50 ml NS + 50 ml 10% dextrose
N/2- 7.5 % dextrose	50 ml NS + 30 ml 25 % dextrose + 20 ml distilled water
N/2- 10% dextrose	50 ml NS + 40 ml 25 % dextrose + 10 ml distilled water
N/2 — 12.5 % dextrose	50 ml NS + 50 ml 25 % dextrose
N/4 -10% dextrose	25 ml NS + 50 ml 10 % dextrose + 20 ml 25% dextrose +5ml distilled water 50 ml N/2 -5% dextrose +30 ml 25% dextrose +20 ml distilled water
N/4 -7.5% dextrose	25 ml NS + 75 ml 10 % dextrose 50 ml 25% dextrose +50 ml 10% dextrose
N/3 -5% dextrose	33 ml NS + 50 ml 10 % dextrose + 17ml distilled water
N/4 **5% dextrose**	25 ml NS + 25 ml 10 % dextrose + 10 ml 25% dextrose + 40 ml distilled water

VASOACTIVE MEDICATIONS

Infusions rate

- Rule of 6
    - 6 x body weight (kg) equals the amount of drug in mg that should be added to 100 mI of solution
    - The infusion volume in (ml/hour) will then equal the mcg/kg/minute dose ordered
- To prevent fluid overload, to deliver with a 50 ml syringe via infusion pump and to prevent drug wastage, the above equation is modified as
    - 3 x body weight(kg) equals the amount drug in mg that should be added to 50 of solution

Vasoactive infusions

Drugs	Dose	Dilution to volume	Infusion rate	Dose range
Epinephrine	0.3 mg/kg	50mI D5 or NS	0.1-10 ml/hr	0.01-1mcg/kg/min
Dobutamine	30mg/kg	50ml D5 or NS	0.5-2 mI/hr	5-20 mcg/kg/min
Dopamine	30 mg/kg	50 ml D5 or NS	0.5-2mI/hr	5-20 mcg/kg/min
Norepinephrine	0.3 mg/kg	50 ml D5 or NS	0.1-10 ml/hr	0.01-1mcg/kg/min
Vasopressin	1.5 IU/kg	50 ml D5 or NS	0.2- 0.5 mI/hr	0.0001-0.00025IU/kg/min
Milrinone	1.5mg/kg	50 mI D5 or NS	0.6-1.5 ml/hr	0.3-0.75 cg/kg/min

"Adapted from IAP ALS Handbook, First edition 2018

Table 5.7.3:Mechanism of action, doses and side effects ofvasoactive drugs

Drug	Mechanism of action	Dosage	Side effects	Remarks
Dopamine Natural precursor of norepinephrine (NE). Acts by NE release.	Alpha and beta adrenergicreceptor agonist Dose dependent action: S 10 ug/kg/min stimulates beta-1 receptors and increases inotropy and chronotropy 10-20 mcg/kg/min stimulates alpha receptors and increases vasoconstriction and SVR	5—20 pg/kg/ min	Tachycar dia, arrhythmias	Use of dopamine for renc› protection is no longer recommended (1-5 pg/kg/min) Due to tachyphylaxis, response decreases ovei time and hence frequent dose titration is required
Dobutamine	Predominantly beta-1 adrenergic receptor agonist. Inotropy and some chronotropy Minimal beta-2 effect causes vasodilatation	2-20 pg/kg/ min	Tachyarrhythmi as, hypotension	Agent of choice for car Jiogenic shock with low cardiac output and normal blood pressure
Epinephrine	Potent beta-1 and moderate beta-2 and alpha-1 receptor effects. Lower doses (0.1pg/kg/min) increases cardiac output due to beta action causing inotropy and chronotropy Higher doses (>0.2\Jg/kg/min), stimulate alpha-activity increasing SVR in addition to augmentation of CO	0.05-0.3 pg/kg/ min	Tachyarrhythmi as, ischemia	Is fii st line therapy for cold hypodynamic septic shock Ag‹*nt of choice for anaphylactic shock
Norepinephrine	Stimulates both alpha-1 and beta-1 adrenergic receptors, producing potent vasoconstriccion	0.1-2 pg/kg/ min	Tachycardia, arrhythmias	Fir',t line therapy in warm septic shock (decreased SVR) Anaphylaxis Neurogenic shock
Milrinone Phosphodieste ra se inhibitors	Non-adrencrgic drug with inotropic and vasodilatory actions. Clinical response similar to dobutamine buI more potent and with less incidence of dysrhythmias Onset: 2 to 5 mins Peak: 10 min	0.125 0.75 pg/kg/ min	Hypotension, ventricular arrhythmias	Indicated in cardiogenic sh›ck refractory to other agiznts Pr«ferred agent for postopera tive low cardi xc ou tput state

”Adapted from IAP-ALS Handbook, First edition 2018

DISABILITY INTERVENTIONS

NEUROLOGICAL ASSESSMENT

Glasgow coma scale (GCS)

Behavior	Response	Response in children	Score
Eye opening response	Spontaneously To speech To pain No response	Spontaneously To speech To pain No response	4 3 2 1
Best verbal response	Oriented to time ,place and person Confused Inappropriate words Incomprehensible words No response	Coos, babbles Irritable cry Cries to pain Moans to pain No response	5 4 3 2 1
Best motor response	Obeys commands Moves to localized pain Withdrawal from pain Abnormal flexion (decorticate) Abnormal extension (decerebrate) No response	Obeys commands Moves to localized pain Withdrawal from pain Abnormal flexion (decorticate) Abnormal extension (decerebrate) No response	6 5 4 3 2 1
Total score	Best response Comatose Totally unresponsive		15 <8 3

AVPU scale:

Category	Stimulus	Response type	Reaction
Alert	Normal environment	Appropriate	Normal intractability for age
Verbal	Simple command/ sound stimulus	Appropriate Inappropriate	Responds to name- Non specific or confused
Painful	Pain	Appropriate Inappropriate Pathological	Withdraws from pain Sound or motion without purpose or localization of pain Posturing
Unresponsive	No perceptible response to any stimulus	Pathological	No response

Ready reckoners

Stock up your circulatory cart :

Intraosseous line

Vasoactive infusions

Defibrillation

Fluids composition

Blood pressure centile charts

Stock up your respiratory cart :

Airway Adjuncts

Bag and mask ventialion

Nebulization

Pulse oximetry

Oxygen delivery devices

1. DIABETIC KETOACIDOSIS

1.1 Learning objectives

After completion of this section the participant should be able to

- Identify a case of diabetic ketoacidosis (DKA)
- Assess the severity of DKA
- Initiate golden hour management of DKA
- Know when, how to refer and precautionary measures to be taken before referral?

1.2 What is DKA?

- DKA is an acute life threatening complication of diabetes characterized by features enumerated in Box 1.1

1.3 Why does DKA happen?

- **It** is a complex disordered metabolic state occurring as a consequence of absolute or relative insulin deficiency accompanied by an increase in counter-regulatoryhormones (glucagon, cortisol, growth hormone. epinephrine)
- This hormonal imbalance causes hepatic gluconeogenesis, glycogenolysis resulting in hyperglycemia and lipolysis that causes ketogenesis

Box 1.1: Features of DKA

Clinical	• Dehydration • Acidotic breathing (deep, sighing, no recessions) • Lethargy + drowsiness • Abdominal pain + vomiting • Polyuria and polydipsia
Laboratory	• Hyperglycemia (>200 mg/dl) • Ketosis (Blood > 3 mmol/ and/or Urine > 2+ with ketostix • Metabolic acidosis (pH <7.3) or bicarbonate <15 mmol/L (Note: If blood gas is not available, acidotic breathing can be taken as acidosis)

1.4 When to suspect diabetic ketoacidosis (DKA)?

In new onset diabetes, (diabetes is diagnosed during the first episode of DKA) suspect DKA in case of following presentation

- Unexplained drowsiness
- Unexplained vomiting & abdominal pain
- Unexplained dehydration but preserved urine output
- **History** of polyuria and polydipsia (history needs to be taken carefully as parents quite often ignore these symptoms)
- Deep sighing respiration
- All the above may or may not be associated with fever

In a **known case of diabetes on insulin therapy,who presents with DKA, the following risk factors need to be assessed:**

- Omission of insulin, infection, psychological stress as they can precipitate DKA
- Poor metabolic control
- Puberty and adolescence

1.5 Approach to a child with DKA

- Assess the severity (refer box 1.2)

Box 1.2: Severity of DKA

Parameter	**Mild**	**Moderate**	Severe
Arterial pH	7.3 - 7.2	7.2 - 7.1	< 7.1
Serum bicarbonate (mmo1/1)	10- 15	5- 10	<5
Level of consciousness	Alert	Alert/drowsy	Stupor/coma

- Goals of management

  Correct fluid loss (dehydration):

- Calculate fluid requirements:

Deficit (A) + Maintenance over 36-48 hours(**B)**

- **Deficit:** 5% in mild & moderate; 8.5% in severe cases
- A+ B has to be given as hourly infusion
- Normal saline (0.9%) is the initial fluid of choice for first hour management. Subsequently the fluid can be changed to half normal saline (0.45%) and continued till BG<250 mg/dl
- If plain 0.45% NS is not available, then normal saline can be continued
- The fluid is changed to 0.45% NS with 5%Dextrose once the BG is <250 mg/di

ii) Correct hyperglycemia by starting low dose insulin infusion : 0.05 to 0.1 u/kg/hr(refer box1.3)

iii) Correct electrolyte disturbances

- Hyperkalemia can occur at presentation due to acidosis (refer to management section E, Chapter 4)
- Hypokalemia is a known complication of therapy in DKA. Once fluids and insulin are instituted, hypokalemia occurs due to intracellular shift of potassium. Add potassium at 40mmo1/1 of ICI after assessing adequate urine output

iv) Correct precipitating factors like infection. Start antibiotic if the child has clinical features of infection like UTI, sepsis etc

Box 1.3 : Insulin administration

- How to prepare insulin infusion?
  - Regular insulin (1ml = 40U) 1 ml of regular insulin in 39 ml of NS (1ml = 1U)
- How to administer:
  - Dilute this further in 10 ml of NS and infuse @ 1ml/hr will give 0.1 U/hour
  - If infusion pump is not available then use dial flow. In this case dilute in 100 ml infuse @10ml/hr
- Never mix other drugs along with insulin in the infusion set
- It is preferable to use a separate IV line for insulin

1.6 Investigations:

- Blood Glucose (capillary glucose with glucometer or venous blood glucose)
- ECG for potassium, if serum electrolytes are not available
- Blood gas & serum electrolytes
- Blood ketones and/orurine ketones

1.7 Complications

- Cerebral edema: it is the most dreaded complication of DKA and carries a high mortality if not detected and treated early
    - Features: Clinical signs of neurological deterioration (headache, irritability, drowsiness, seizures, coma, hyperventilation, pupillary inequality, bradycardia and bradypnea)
    - Treatment

- Reduce fluids by 113rd to half
- Immediate hyperosmolar therapy — 0.5 gm/kg Mannitol or 5m1/kg 3% saline
- Intubation, and initiate anti-raised ICP measures(refer section D, chapter 1)

- Hypokalemia :
    - Potassium is mainly an intracellular ion, and there is always massive depletion of total body potassium although initial plasma levels may be low, normal or even high. Levels in the blood will fall once insulin is commenced
    - Treatment
        - Early potassium replacement, after ruling out AKI
        - Rapid correction (0.3-0.5mEq/kg/hour) for hypokalemia which presents with ECG changes(refer section E, chapter 4)
        - Maintenance potassium at 40-60mEq/L to keep serumpotassium between 3.5-5mEq/L

- Hypoglycemia :
    - Malnutrition is an important risk factor for hypoglycemia
    - Aggressive monitoring of blood glucose is required during insulin therapy
    - Early introduction **of dextrose containing fluids**

1.8 Non responders (refer box 1.4)

Box 1.4: Checklist for non-response

- Check patency of IV lines
- Improper insulin dose, dilution and rate of infusion
- Incorrect administration: flush the IV line completely before starting insulin to saturate insulin binding sites
- Underlying infection
- Uncorrected dehydration (check fluid charting)
- Rarely, there may be associated lactic acidosis/ or renal compromise

1.9 When to refer?

- Neurological deterioration: seizures, encephalopathy.
- Acidosis not improving
- Persisting hyperglycemia
- Decreased urine output

1.10 How to refer?

- Child's peripheral venous access should be secured:
- Child's details, history, examination findings, vitals, events occurred and the treatment given should be legibly written in the referral letter.
- Parents should be counseled regarding the patient's clinical status
- A person trained in BLS should accompany the child during referral

Remember:

- Acidosis not getting corrected , think of following possibilities
    - Insufficient insulin to switch off ketones
    - Inadequate resuscitation
    - Sepsis
    - Hyperchloremic acidosis
    - Salicylate or other prescription or recreational drugs
- Think about sepsis in a child with DKA who has any of the following:
    - fever or hypothermia
    - hypotension
    - refractory acidosis
    - lactic acidosis
- Ensure that all fluids (except any initial bolus) contain 40 mmo1/1 potassium Chloride, unless there is evidence of renal

Approach to DKA

Confirm diagnosis
Assess Clinical

Shock Altered sensorium

Resuscitation
Airway
Breathing-100% oxygen With NRM
Circulation- 10ml/kg 0.9%
Saline bolus
Once shock resolves then go to DKA fluid

Clinically dehydrated no shock, drowsy

Intravenous therapy
Calculate fluid requirements:
Deficit + management over over 36-48 hours
(Deficit: 5% in mild & moderate; 8.5% in severe cases)
Use NS in hourly infusion rate till BG<250 mg/dl
Change to 0.45% NS with 5% D later
Add potassium at 40mmol/l of KCI after assessing adequate urine output
Insulin: 0.05 to 0.1 u/kg/hr

Dehydration <5% clinically well & tolerating orally, Alert

Allow oral feeds
Subcutaneous insulin
lu/kg/day regular insulin in 4

Monitoring:
Input output charting
Blood glucose hourly
Neurological status hourly
Serum electrolytes 2hrs after start of IV therapy then 4 hourly ECG monitoring if SE not available

No improvement

Checklist Box 1.4

Reduce insulin <0.05u/kg/hr if acidosis start

Neurological deterioration
Headache
Irritability
Drowsiness, seizures
Bradycardia

Exclude hypoglycemia
Cerebral edema?
Management
5ml/kg 3% NaCl or 0.5g/kg mannitol

Blood glucose <250mg/dl

Changes to 0.45NS with 5% D continue insulin @ 0.1u/kg/hr

Resolution of DKA
Clinically well, tolerating food
pH Normal (7.30)
* **Urine ketones may be positive**

Start subcutaneous insulin then stop IV after one hour

2. **Acute kidney injury**

2.1 Learning Objectives:

After completion of this section, the participant should be able to

- Define acute kidney injury
- Identify cases at risk for AKI
- Identify clinically children with AKI
- Classify based on severity
- Know the common etiology of AKI
- Steps in management of AKI
- Know when and how to refer?

2.2 Acute kidney **injury (AKI)**

- **It is** a spectrum of acutely compromised renal function that result in impaired fluid balance, dyselectrolytemia, and raised urea and creatinine.

2.3 How to define AKI?

In 2012, KDIGO Kidney Disease Improving Global Outcomes (KDIGO) developed a harmonized child-adult definition of AKI based on serum creatinine, eGFR, and urine output.

Remember in most situations AKI will be diagnosed based on urine output criteria only

- Urine volume <0.5 ml/kg/hour for six hours **OR**
- Increase in serum creatinine by >0.3 mg/dL from baseline within 48 hours., OR
- Increase in serum creatinine to >1.5 times baseline within the prior seven days

2.4 Which patients are at high risk for developing AKI?

BOX 1.1 High risk states

In community setting

- History of kidney disease
- Hypotension
- Dehydration
- Gastrointestinal losses
- Dark, concentrated urine
- Ongoing sepsis
- Exposure to potential nephrotoxins

In hospital setting

- Multiple organ failure
- Nephrotoxic medication exposure
- Post operative state

2.5 How to identify patients with AKI?

- Identify high risk cohort (refer box 1.1)
- Monitor urine output to quantify oliguria /anuria
- Other clinical features
    - Odema
    - Gross or microscopic hematuria
    - Hypertension
    - Anemia
    - Laboratory parameters (refer box 1.2)

2.6 **Investigations**

BOX 1.2

To confirm o	**Associated metabolic problems**	**Ascertain etiology**
Urea Creatinine	Sodium , Potassium Acidosis Calcium, phosphorus	Urine routine examination Complete blood count Peripheral smear C3 Reticulocyte count LDH C3 USG (KUB)

Caveats of serum creatinine

- Change in creatinine is a delayed phenomenon. Therefore serum creatinine may be normal in early stages of renal failure
- Serum levels depend on muscle mass
- Young children, malnourished children with less muscle mass will have lower creatinine for the same degree of AKI
- Fluid overload may falsely lower creatinine levels due to dilutional effect

Etiology of AKI can be divided into three categories as summarized in box 1.3