Federal Ministry of Health

National Emergency Treatment Protocol

Emergency and Critical Care Service Directorate
November 2016

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Acronyms and Abbreviations

ACLS	Advanced Cardiac Life Support
AED	Automated External Defibrillators
AF	Atrial Fibrillation
AION	Anterior Ischaemic Optic Neuropathy
AKI	Acute Kidney Injury
AML	Acute Myeloid Leukaemia
aPTT	Activated Partial Thromboplastin Time
ARDS	Acute Respiratory Distress Syndrome
AS	Aortic Stenosis
ATN	Acute Tubular Necrosis
Bi PAP	Bi-level Positive Airway Pressure
BLS	Basic Life Support
BRAO	Branch Retinal Artery Occlusion
BUN	Blood Urea Nitrogen
CHF	Congestive Heart Failure
CML	Chronic Myeloid Leukemia
CPAP	Continuous Positive Airway Pressure
CPR	Cardio Pulmonary Resuscitation
CRAO	Central Retinal Artery Occlusion
CRP	C- Reactive Protein
DIC	Disseminated Intravascular Coagulation
DPL	Diagnostic Peritoneal Lavage
DVT	Deep Vein Thrombosis
ECG	Electro Cardiograph
ESR	Erythrocyte Sedimentation Rate
ETT	Endotracheal Tube
EVD	Ebola Virus Disease
GCS	Glasgow Coma Scale
HIT	Heparin Induced Thrombocytopenia
HOCM	Hypertrophic Obstructive Cardiomyopathy
IO	Intraosseous
IVIG	Intravenous Immunoglobulin
JVP	Jugular Venous Pressure
LMWH	Low Molecular Weight Heparin
MDI	Metered Dose Inhaler
MERS-CoV	Middle East Respiratory Syndrome Coronavirus
MI	Myocardial Infarction
NAION	Nonarteritic Ischemic Optic Neuropathy
NGT	Nasogastric Tube
PaCO2	Partial Pressure of Carbon Dioxide in Arterial blood
PEA	Pulseless Electrical Activity
PEEP	Positive End Expiratory Pressure
PET CO2	End Tidal Carbon Dioxide Pressure
PCI	Percutaneous Coronary Intervention

PCWP Pulmonary Capillary Wedge Pressure
PTE Pulmonary Thromboembolism
ROSC Return of Spontaneous Circulation
SA Sino Atrial
SaO2 Saturation of Oxygen
SARS Severe Acute Respiratory Syndrome
SAH Subarachnoid Haemorrhage
SCD Sudden Cardiac Death
SVT Supra Ventricular Tachycardia
TIA Transient Ischaemic Attacks
UAO Upper Airway Obstruction
UGIB Upper Gastrointestinal Bleeding
UFH Unfractionated Heparin
VF Ventricular Fibrillation
Vtac/VT Ventricular Tachycardia
List of Tables and Figures

Tables

Foreword

The Federal Ministry of Health's Emergency and Critical Care Directorate has been working to improve emergency medical services in the country. To date, although health facility based operating procedures exist, there is no standardized treatment protocol on how to manage emergency cases commonly encountered in the emergency room. To address this need, an easily accessible and practical reference protocol has been developed to help health professionals to make timely decisions and give appropriate treatment. It is envisaged that the development of this emergency treatment protocol will play a vital role in improving the quality of emergency patient care at health facilities by decreasing unnecessary delays in the provision of emergency treatment. This treatment protocol is intended to be used by all health care professionals at all the three levels of the health care-tier system and for both public and private health institutions throughout the country.

Finally, I would like to extend my gratitude to all individuals and institutions for their valuable contributions in the development of this treatment protocol.

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Section 1 Cardiopulmonary Resuscitation (CPR) Related Procedures and Arrhythmias

1.1. Cardiac arrest

Definitions:
Cardiac Arrest: is the abrupt cessation of cardiac pump function, which is mostly due to sudden cardiac death in adults. Sudden Cardiac Death (SCD) –is death due to cardiac causes which is heralded by abrupt loss of consciousness in an individual with/without known preexisting heart disease.

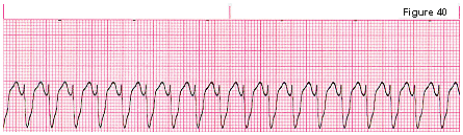
Causes of Cardiac Arrest:
1) Cardiac origin (SCD): Ventricular Fibrillation (VF), Ventricular Tachycardia (Vtac), pulse less tachyarrhythmia due to ischemic heart disease, shock, stroke, etc
2) Respiratory origin: upper airway obstruction, drowning, smoke inhalation, drug overdose, trauma, etc

3) Metabolic disturbances: electrolyte imbalance, acid base imbalance, etc
Always think of reversible causes for cardiac arrest: the 5 H's (hypovolemia, hypoxia, hydrogen ion (acidosis), hypo/ hyperkalemia, hypothermia) and 5 T's (tension pneumothorax, tamponed-cardiac, toxins, thrombosis-pulmonary/coronary. Most of the time the final even of SCD is due to arrhythmia like ventricular fibrillation, ventricular tachycardia, pulseless electrical activity, and asystole.

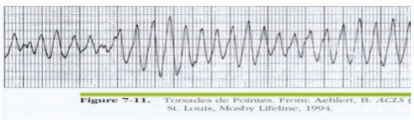
1.2 Arrhythmia associated with cardiac arrest

Ventricular Tachycardia (Vtac) is a wide complex tachycardia originating in the ventricle, and can be stable pulseless (absent central pulse like carotid pulse), unstable (low blood pressure (BP) or shock) and stable (normal BP).It is classified in to monomorphic and polymorphic based on the morphology of the rhythm.

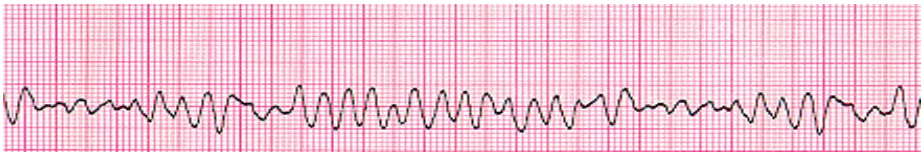
Monomorphic Ventricular Tachycardia



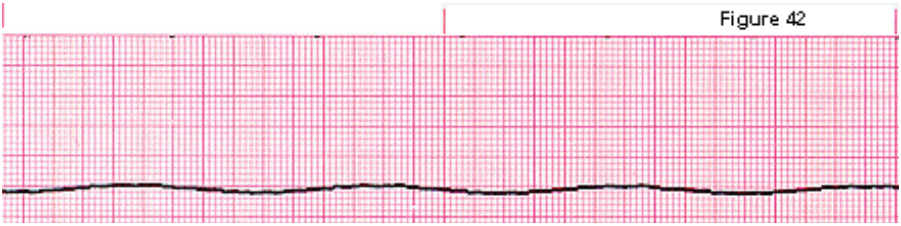
Polymorphic V tac (Torsade-pointes)



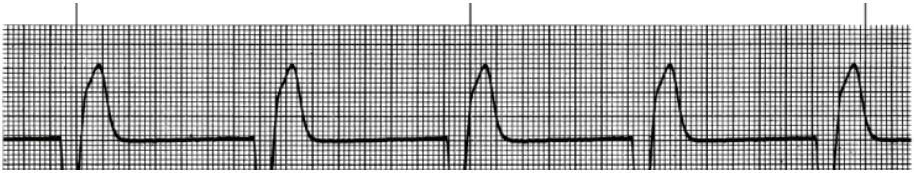
Ventricular Fibrillation



Asystole: is a cardiac arrest rhythm associated with no discernable electrical activity



Pulseless Electrical Activity (PEA): rhythm with organized electrical activity (other than VF or VT) without a central pulse.



PEA

Consequences of cardiac arrest

After cessation of blood flow there will be depletion of oxygen in vital organs. And after 4-6 min of cardiac arrest brain damage starts to occur. Early CPR within 4 min and rapid ACLS with defibrillation within 8 min are essential in improving survival and neurological recovery.

Early intervention is essential.

After 4-6 min of cardiac arrest, brain damage can occur. Early—within 4 minutes—cardiopulmonary resuscitation (CPR) and rapid advanced cardiac life support (ACLS) with defibrillation within 8 minutes time are essential in improving survival and neurological recovery.

1.3 Cardiopulmonary Resuscitation

Cardiopulmonary resuscitation (CPR) is a skill which includes providing artificial respiration to provide oxygen to the lungs and artificial circulation to maintain blood flow through the body enough to give a person a chance for survival. It is

performed when there is cardic or repiratory arrest. There are two levels of arrest resuscitation, namely: basic life support and advanced cardiac life support.

1.3.1 Adult Basic Life Support

1-Basic Life Support (BLS)

It is an initial airway, breathing, circulation (ABC) assessment and management of arrested patients which is also called cardiopulmonary resuscitation (CPR). It can be provided by trained non-medical or medical workers, until definitive medical treatment can be accessed.

When a patient is found unconscious or suspected to have cardiac arrest the following steps should be followed:

- Determine un-responsiveness by touching and shouting
- Call for help
- Position the victim and start CPR
- Act as a team and have a team leader that guides the quality of the CPR and for decision making

Sequences of CPR (In cardiac arreast is CAB sequence)

1. Circulation

- If the carotid artery pulse is absent, start CPR (carotid artery palpation has been found inaccurate in untrained rescuers and absence of spontaneous breathing including agonal breaths is now taken as a sign of cardiac arrest).
- Start chest compression:
- Depress and release the chest rhythmically,
- Press the heels of the hands straight down on the center of the chest.
- The pressure and release phases should take the same time,
- Give compressions at a rate of 100 per min (count compressions out loud),
- Give 30 compressions to 2 breaths whether with ONE rescuer or TWO
- Depress the chest 2.5-5cm depth
- After every 5 cycle/2min. check for spontaneous breathing and circulation for 5 sec

2. Airway

- In suspected trauma stabilize the neck before airway opening maneuver, use jaw thrust in patients with suspicion of cervical spine injury
- To open the airway, use head tilt and chin lift maneuver: push backward on the forehead and lift the chin if the possibility of cervical spine injury is less
- In order to check for breathing, look for chest movement, listen for sounds of breathing, feel for breath.

3. Breathing

- In cardiac arrest give 30 chest compressions immediately before any rescue breaths are attempted and give two rescue breaths with bag valve mask (BVM). Do not over inflate the chest; connect the BVM with oxygen source. Avoid interruptions during CPR.

If the chest does not rise when you blow air:

- Reopen the airway by tilting the head and lifting the jaw, see and remove any foreign bodies or secretions. Then try ventilation again.

Box A Protocol and skills in CPR (see also the algorithm)

Assessment: If victim is unresponsive check for breathing movement of the chest and carotid Pulse.

CPR: If breathing effort is absent or agonal, and there is no carotid pulse - start chest compression and artificial ventilation. If pulse is present, but there is no breathing, provide rescue breathing - every 5 - 6 seconds)

For high quality CPR, effective chest compression skills are very essential.

- **Push hard & fast (100/min) using heel of both hands, one on the other, at the lower half of sternum (2.5-5 cm depth)**
- **Allow complete chest recoil after each compression.**
- **Compression to ventilation ratio is 30:2 (for both one and two rescuer). Avoid rapid & forceful breaths (to prevent hyperventilation).**
- **Minimize interruptions in compression (No pause in compression for checking the pulse). Any interruption should be less than 10 seconds**
- **If advanced air way inserted, 100 compressions per minute; and ventilate 8- 10 breaths/min while compression is maintained, meaning no need of sequencing.**

1.3.2 Advanced Cardiac Life Support (ACLS)

Advanced Cardiac Life Support (ACLS) is a continuation of BLS but necessitates better setup and expertise. Although the same CPR protocol is followed it is done by skilled persons and equipment such as a defibrillator and other resources.

In addition to the basic CPR defibrillation, pharmacologic treatment, advanced airway management is included. Survival from cardiac arrest is highly dependent on high quality CPR, ACLS and post- cardiac arrest care. These steps and care are also described as chain of survival.

Chain of Survival for effective resuscitation includes:
1. Early access to the patient or victim
2. Early CPR initiation
3. Early defibrillation
4. Early & effective post resuscitation care

The Protocol in Shockable Rhythms (Ventricular Fibrillation or Pulseless VT):

1. Immediate defibrillation with 200 J (most new machines models are biphasic and start with 200J, in older monophasic defibrillators 360 J is used), then follow with immediate effective CPR for 2 min and evaluate rhythm

2. Defibrillation 200 biphasic or 360 monophasic then immediate effective CPR for 2 min and evaluate rhythm.

3. Adrenaline 1 mg IV and repeat every 3 – 5 min (every second cycle) and evaluate rhythm

4. Defibrillation 200 J biphasic or 360 J monophasic and effective CPR for 2 min evaluate

5. Amiodarone 300 mg I/V push or Lidocaine 1mg/kg Evaluate rhythm

6. Defibrillation 200 biphasic or 360 monophasic and Adrenaline 1 mg every 3 min.

7. Can use Mg 2g IV in suspected Torsade de Pointes, alcoholism or malnutrition

Protocol in non-shockablerhythms (asystole or pulseless electrical activity)
1. Continuous CPR and find and correct possible causes
2. Adrenaline 1 mg every 3 minutes for the duration of resuscitation
3. If asystole – Flat line protocol: Check monitor/ power, check leads

Defibrillation and cardioversion: is passage of an electrical current of sufficient magnitude to depolarize a critical mass of myocardium and restoration of coordinated electrical activity from the SA node. It is indicated for VF and pulseless ventricular tachycardia.

• Defibrillation: non-synchronized delivery of energy, defibrillation is part of it i.e., the shock is delivered randomly during the cardiac cycle.

• Cardio version refers to delivery of energy that is synchronized to the QRS complex to eliminate the risk of VF (in unstable SVT/AF or monomorphic VT).

In this condition the patient is responsive and hence, ask for consent and consider sedation, prepare the machine and attach monitor leads of the defibrillator, and then touch the synchrony button on the defibrillator; before discharge make sure everybody, including yourself, is clear from the patient (i.e. nobody is touching the patient or the bed). Finally, discharge the selected amount of energy which is smaller in cardioversion(50-100J)

In witnessed arrest, defibrillation should be attempted as early as possible, immediately after starting the CPR & repeated as per the algorithm.

Box B Defibrillator Use

- **Steps in defibrillator use:**
- **Selection of proper energy, proper mode (asynchronous vs Synchronous),**
- **proper position of the paddles or electrode pads,**
- **adequate contact between paddles and skin,**
- **no contact with anyone other than the victim,**
- **rhythm assessment.**

(Preparationàselects energyà chargeà discharge)

Automated External Defibrillators (AED): turn onàapply the padàwait while the machine analyzes the rhythmàthen the machine advices shock if it detects a shockable rhythm.
Precordial thump: forceful heating of the heart at the lower half of sternum may produce about 7 joules and might be attempted when there is no defibrillator.
Precordial thump: forceful heating of the heart at the lower half of sternum may produce about 7 joules and might be attempted when there is no defibrillator.

Other Procedures

Artificial Ventilation: for basic mouth to mouth, bag valve ventilation and advanced airway with mechanical ventilation (Refer to breathing)
Intravenous access is needed for IV drug administration like epinephrine, atropine, etc

Strategies in post cardiac resuscitation care: Therapeutic hypothermia (32-34°C);Hemodynamic optimization(hypotension management) ; Ventilation optimization (SaO2≥94%);Immediate coronary reperfusion with percutaneous coronary intervention (PCI) for AMI; Glycemic control: maintain glucose levels between 140-180 mg/dl to avoid hypoglycemia.

Box C ACLS Pharmacology

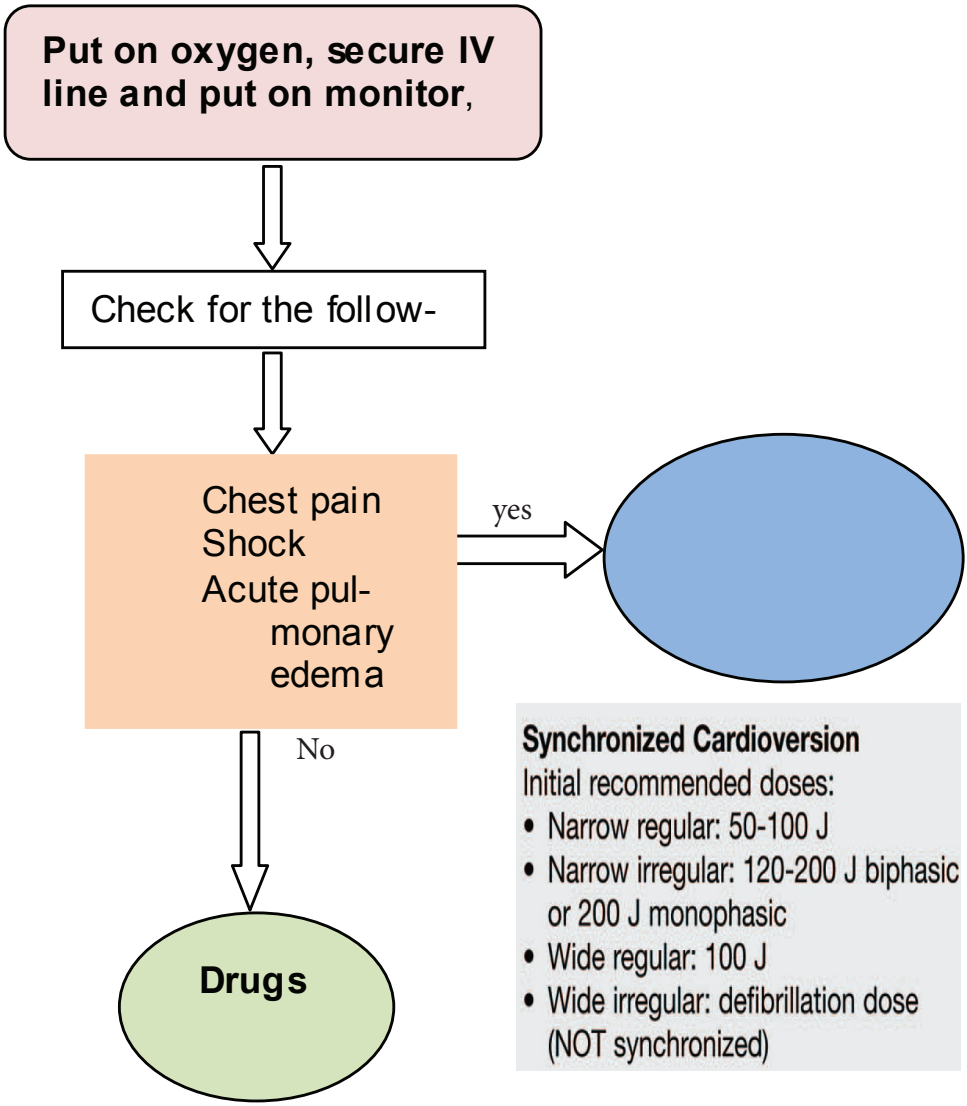
Vasopressors

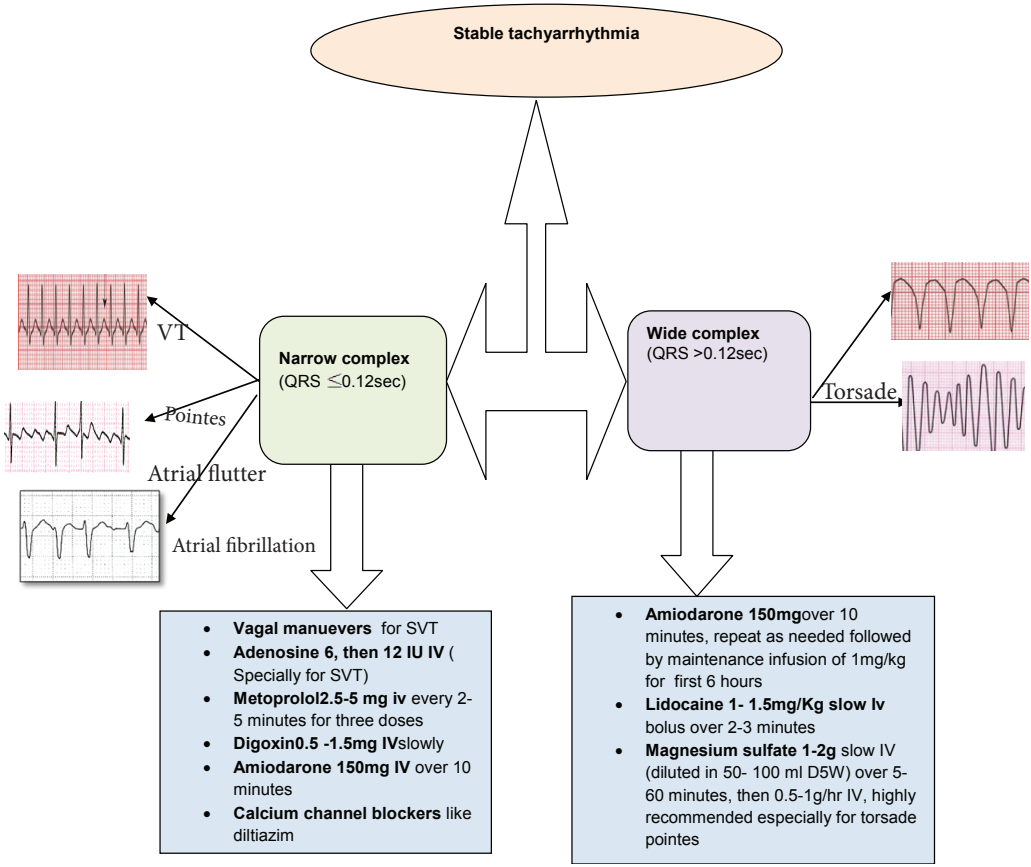
- **Epinephrine= indication:VF, Pulseless VT, Asystole, PEA, Symptomatic bradycardia Dose: 1mg IV/IO Q 3-5 minutes during CPR, followed with 20 ml flush with fluids. Antiarrhythmic:**
- **Amiodarone= indication: VF/Pulseless VT unresponsive to CPR, Vasopressor or Shock; Polymorphic VT, adjunct to cardioversion of SVT/PSVT. Dose: Cardiac Arrest: 300 mg IV/IO with 20-30 ml D5W push. If no response,150 mg IV push; repeat in 3-5 minutes.(for stable wide complex tachy-150mg iv)**
- **Lidocaine: alternative to amiodarone in cardiac arrest from VF/VT, stable monomorphic VT with preserved LV function. Dose : 1-1.5mg/kg IV; if VF/VT persists 0.5-0.75mg/kg IV at 5-10 min.(max 3mg/kg)**
- **Magnesium sulphate: for VF/Pulseless VT associated with torsade's de pointes (Irregular/Polymorphic VT associated with long QT interval); hypomagnesaemia or digitalis toxicityDose: IV/IO bolus of 1-2 gram diluted in 10 ml D5W over 5-20 minutes**
- **Adenosine=first drug for most forms of narrow tachyarrhythmia's. Dose:6 mg rapid IV push, follow with 20 ml N/s; repeat dose of 12 mg in 1- 2 minutes, up to 3rd dose of 12 mg. Side Effects: hypotension, chest discomfort .C/I : Asthma, 2nd or 3rd degree AV block**
- **Diltiazem/Verapamil= indication: control ventricular rate in Atrial- fibrillation and atrial flutter. Dose: Diltiazem-15-20 mg IV over 2mins or Verapamil- 5mg may repeat in 15 munities at 20-25 mg.**
- **Digoxin= for Atrial- fibrillation / flutter rate control. Dose: loading 0.25mg Q 2hr until 1mg total; then**

1.4. Arrhythmia

1.4.1. Tachyarrhythmia

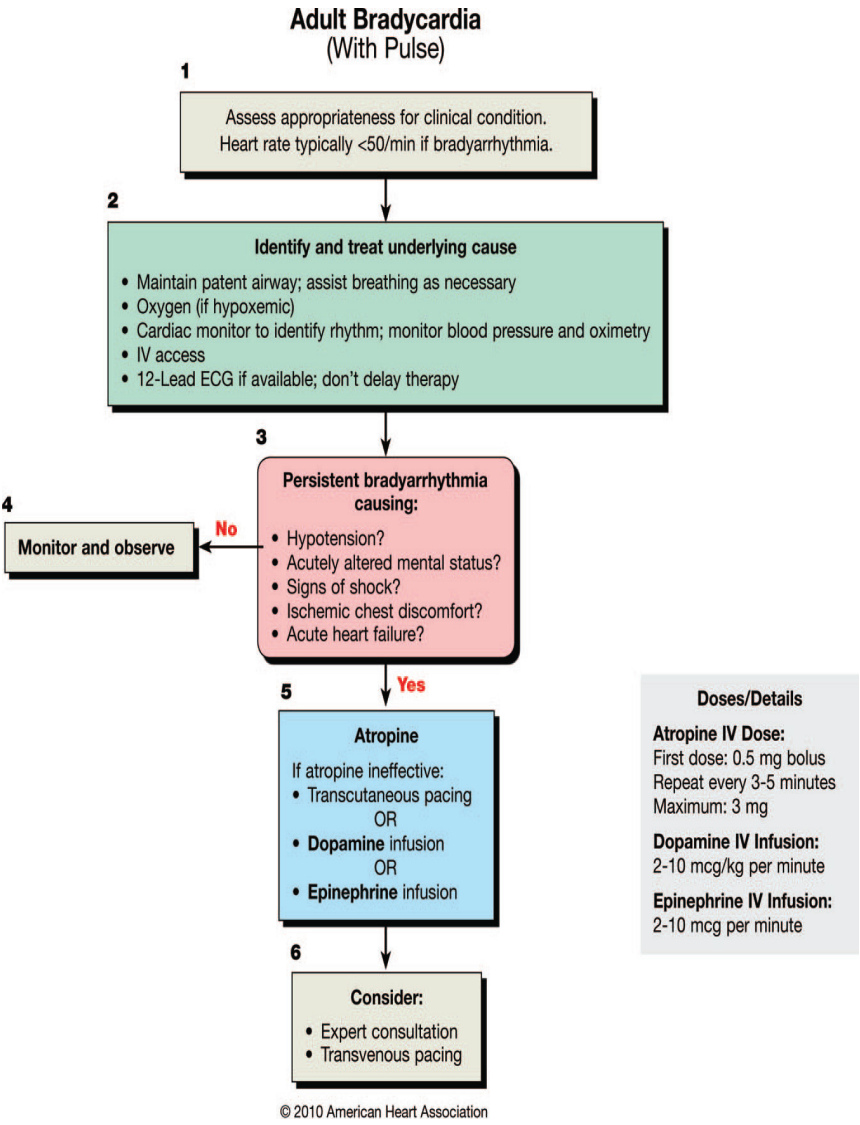
- Tachyarrhythmia is defined as a heart rate greater than 100 beats per minute. Patients become symptomatic when the heart rate is greater than 150 beats per minute. Tachyarrhythmia can occur in patients with normal or abnormal cardiac function.
- In patients with tachyarrythmia check for signs of instability. If stable, give drugs. If unstable, do synchronized cardioversion. Check algorithm.
- Adenosinecan be administered for a patient with unstable tachyarrhythmia because it is fast onset and has a brief duration of action. Many drugs are contraindicated in ventricular dysfunction but digoxin and amiodarone also can be used.


Put on oxygen, secure IV line and put on monitor,
Check for the follow-
Chest pain
Shock
Acute pul-
monary
edema
yes
No
Drugs
Synchronized Cardioversion
Initial recommended doses:
• Narrow regular: 50-100 J
• Narrow irregular: 120-200 J biphasic or 200 J monophasic
• Wide regular: 100 J
• Wide irregular: defibrillation dose (NOT synchronized)


Stable tachyarrhythmia
VT
Pointes
Atrial flutter
Atrial fibrillation
Narrow complex
(QRS ≤0.12sec)
Wide complex
(QRS >0.12sec)
Torsade
Vagal manuevers for SVT
Adenosine 6, then 12 IU IV (Specially for SVT)
Metoprolol2.5-5 mg iv every 2-5 minutes for three doses
Digoxin0.5 -1.5mg IVslowly
Amiodarone 150mg IV over 10 minutes
Calcium channel blockers like diltiazim
Amiodarone 150mgover 10 minutes, repeat as needed followed by maintenance infusion of 1mg/kg for first 6 hours
Lidocaine 1- 1.5mg/Kg slow Iv bolus over 2-3 minutes
Magnesium sulfate 1-2g slow IV (diluted in 50- 100 ml D5W) over 5-60 minutes, then 0.5-1g/hr IV, highly recommended especially for torsade pointes

1.4.2. Bradycardia

This bradycardia algorithm focuses on management of clinically significant bradycardia (i.e., bradycardia that is symptomatic).



Section 2 Adult Airway Problems

2.1. Adult Upper Airway problems

The Upper airway is the conducting passage extending from the nose or mouth to the main carina.
Airway obstruction results in hypoventilation (decrease breathing exercise), increased work of breathing and impaired gas exchange, with development of hypercarbia (high concentration of CO2 in the body) and hypoxemia (O2 deficiency) if left untreated.

Airway Obstruction may be partial or complete, depending on the mechanism or cause.

- Complete airway obstruction will rapidly cause hypoxia and cardiac arrest
- Partial obstruction –patients may have different degree of breathing difficulty or maintain their spontaneous breathing but they are still at high risk to develop complete obstruction if left untreated

Causes

- Obstruction of the pharynx by the tongue, secretions and foreign body in the unconscious patient usually with GCS <8/15
- Foreign bodies in the oropharynx, trachea, or esophagus (commonly large pieces of meat or food);
- Allergic swelling of upper airway structures ("angioedema");
- Chemical burns; inhalation injuries;
- Infectious causes: epiglottitis, and retropharyngeal or peri- tonsillar abscess.
- Trauma: of face, neck, upper tracheal or laryngeal injury

Signs and symptoms of upper airway obstruction

- Snoring – is a sound heard during inspiration and expiration, main causes are: secretions, tongue falling to the back of oropharynx
- Stridor– is a sound heard most of the time during inspiration and it is audible accompanied with use of respiratory accessory muscles, caused by obstruction at or above the laryngeal level, due to spasms or edema of the larynx
- Expiratory wheeze–caused by obstruction of lower airways,
- Different degree of respiratory distress- shallow, fast, labored, with respiratory muscle retractions
- CNS manifestations– Irritability, agitation, convulsion, and coma
- 'Tripod positioning' – standing up and leaning forward position for maximal air passage to lung

Investigations:

- Every investigation done outside the emergency suite has to be considered only following stabilization of life threatening condition of the patients
- If the condition allows, plain and lateral neck and chest films may be useful as screening tests by identifying tracheal deviation, extrinsic compression, or radiopaque foreign bodies.
- High-resolution CT of neck and chest can help identify intrinsic and extrinsic tumors, vascular structures, and foreign bodies.
- Rigid or flexible bronchoscopy with direct visualization is the most effective tool in establishing diagnosis and frequently provides the best way to correct UAO.
- Blood gas analysis and end tidal CO2 tension detects a respiratory acidosis, with a high carbon dioxide tension (PaCO2) reduced pH that reflects alveolar hypoventilation.

General approach and disposition

- Triage and ABCD evaluation to determine presence of complete or partial upper airway problem signs and symptoms

2.1.1. Complete airway obstruction

- Complete airway obstruction is defined as severe respiratory distress or no breathing activity. To respond to this:
- mobilize the emergency team and dispose to resuscitation area,
- open the mouth with head tilt chin lift maneuver or jaw thrust if trauma is suspected, suction secretions, see for foreign body and remove if it is visible and accessible with Magill forceps
- insert oropharyngeal or naso-pharyngeal airway if the cause of obstruction is loss of consciousness or the tongue is obstructing the pathway
- Administer oxygen using appropriate oxygen delivery device, according the severity of symptoms. Bag-valve-mask (ambubag) with 100% oxygen if the breathing effort is not adequate or no breathing effort, Consider oral airway if the chest is not expanding well during bag-valve-mask assistance. For adults, ventilate once every 5 to 6 seconds (10 to 12 times per minute). If patient has breathing effort and on distress administer O2-10-15 litres/min via face mask and monitor with pulseoxymeter, and capnogram
- Obtain appropriate history related to event, recent infectious history (fe-

ver, cough, etc.) or exposure to allergens while managing the airway or ABCD evaluation

- If patient has no breathing effort and you couldnot ventilate or facing difficulty to ventilate with an Ambu bag, use Laryngeal Mask Airway (LMA) OR Combitube. Activate experienced team for definitive airway management using intubations or surgical methods such as crico-thyroidotomy or tracheostomy
- Before intubation, attempting rapid airway evaluation/assessment is mandatory
- According your airway assessment, if you suspect difficult airway such as: airway assessment of Malapati III-IV, trauma on the face and neck, obesity, deformity of the face, inadequate mouth opening, severe hypoxia not resolving with high flow of oxygen then avoid rapid sequence inductions
- Indications for Rapid Sequence Induction (RSI) includes: all emergency and trauma patients, pregnant women, diabetic patients, patients with signs and symptoms of gastric and bowel obstruction. For the technique of RSI see annex.
- Following intubations confirm for proper position of the Endotracheal Tube (ETT), secure the tube properly, continue ventilation manually using ambubag and 100% of oxygen and transfer to ICU for mechanical ventilation support after stabilization of the condition.
- The need for an immediate surgical airway (crico-thyroidotomy) must be evaluated considering the potential difficulties associated with emergency intubation in cases of laryngotracheal trauma, foreign body lodged in the pharyngolaryngeal area, or severe anatomic deformity caused by trauma or congenital
- Determine patient's hemodynamic stability and symptoms. Continually reassess level of

consciousness, ABCs and Vital Signs. If cardiac arrest is diagnosed, use CPR protocol

- Monitor and record vital signs and respond as you find
- Consult appropriate specialty for definitive management.

2.1.2. Partial obstruction

Partial airway obstruction is define as when the patient is conscious, might have difficulty of breathing, coughing, drooling, and difficulty of breathing.

Action

- Encourage him/her to cough, forcefully and close observation if the suspicion is foreign body aspiration or chocking
- Place patient in position of comfort, avoid upper airway stimulation for infection of the epiglottis
- Administer O2, 10-15liters/min via facemask
- Racemic epinephrine is used to treat postextubation laryngeal edema or laryngotracheobronchitis
- Steroids, epinephrine, anti-histamine reduce airway edema
- Helix, a helium–oxygen gas mixture, is effective in reducing the work of breathing by decreasing airway resistance for conditions such as post extubating laryngeal edema, tracheal stenosis or extrinsic compression, and angioedema

2.2 Specific airway problems

2.2.1 Unconscious patients

2.2.1.1 Unconscious with adequate breathing Glasgow Coma Scale (GCS) >8/15

- Put them on recovery/left lateral position
- Consider supplemental oxygen to maintain O2 saturation > 93%.
- Give oxygen via nasal cannula at 2 to 5 liter per minute - OR –
- Use facemask at 10 to 15liters per minute.
- Insert nasopharyngeal airway to facilitate suctioning and to support the airway patency
- Use pulseoxymeter and capnometre to measure oxygen saturation and end tidal CO2, record and act accordingly
- Remember C- spine precaution if patient has history of trauma

2.2.1.2 Unconscious with inadequate breathing:

- Patients with a reduced conscious level with GCS of 8/15 or below are often considered unable to maintain their airway patency and clear their secretions due to their airway reflexes (gag reflex, swallowing reflex, and cough reflexes) are suppressed. And they are at risk of aspiration and alveolar hypoventilation, with development of hypercarbia and respiratory acidosis.

a. Open airway- use head tilt chin lift for none trauma patients and jaw thrust for trauma patient
b. Consider placement of oropharyngeal airway in deeply unconscious ptients (GCS<8)
c. Supplemental oxygen at 10 to 15 liter per minute with Face mask OR
d. Bag-valve-mask (ambubag) with 100% oxygen if the breathing effort is not adequate. Consider oral airway if the chest is not expanding well during bag-valve-mask assistance.
e. For adults, ventilate once every 5 to 6 seconds (10 to 12 breaths per minute).
f. If signs of airway obstruction present (no chest movement) reassess the ptient for possible presence of foreign body or tongue is falling back
g. Suction if there is excessive secretion, checkfor any foreign body and remove if it is accessible, proceed to use oral air way, get an assistant and use you both hands for jaw trust and ventilate with Ambu bag. If saturation is above 92% continue bagging.
h. If you could not ventilate use Laryngeal Mask Airway (LMA) OR Combitube
i. Prepare for definitive airway management using ETT intubation.
j. Use rapid sequence induction and intubation method if there are no signs of difficult airway and ventilate with BVM or Mechanical ventilator
k. Use pulseoxymeter and capnogram to measure oxygen saturation and CO2 tension, record readings and act accordingly
l. Remember C- spine precaution if patient has history of trauma

2.2.2 Obstruction due to a foreign body

In order to treat a choking patient it is important to recognize the patient's condition. Try to ascertain what material is causing the obstruction.

Signs and symptoms

- Choking has a sudden onset
- Patient will be obviously distressed and agitated.
- The face is likely to be congested (red),

- The patient may be holding their throat (universal sign).
- The patient may be standing up and leaning forward.
- Difficulty in breathing and/or speaking.
- Cyanosis (late sign, blueness around the lips).
- Unconsciousness (late and pre-arrest sign).

Treatment

Conscious /partial obstruction

a. Reassure the patient

b. Encourage him/her to cough, forcefully and provide close observation.

c. If the patient is not improving and has developed signs of complete obstruction, use the management measures for complete airway ob struction

Conscious with complete obstruction

a. Stand to the side and slightly behind the patient.

b. Support the chest with one hand; lean the patient forwards so that if the object becomes dislodged it will not go further down to the windpipe but be ejected.

c. Give FIVE firm blows with theheel of the palm of the hand between the shoul der blades.

d. If this fails, abdominal trusts should be used. Stand behind the patient and place both arms around the upper part of the abdomen, just below the ribcage

e. Hold one fist within the other hand and pull sharply inwards and upward up to FIVE times.

f. If unsuccessful, return to 5 back blows and alternate with abdominal thrusts until the obstruction is removed or patient becomes unconscious.

g. If patient loses consciousness, position carefully on his or her back at tempt to conduct five times abdominal thrust see the object in the mouth and if visible remove and proceed to give two rescue breaths. Continue such ma neuvers until the foreign body is dislodged and patient resumes spontaneous breathing

h. If cardiac arrest is eminent, conduct CPR according the adult CPR guide line

For technical application please refer to diagram provided in the annex section.

If unconscious

- If the patient has no pulse commence CPR.
- Loss of consciousness will lead to a relaxation of the muscles in the throat and this may allow some air to pass beyond the obstruction and into the lungs, therefore give 2 rescues breathing during the CPR. See CPR protocol.
- If the object is visible, attempt finger sweep or remove using Magill forceps.

2.2.3 Burn or smoke inhalation and trauma on the face and neck

- In the case of burns or history of smoke inhalation, adopt a high index of suspicion for airway injury.
- Signs such as carbonaceous sputum, dust around the face and mouth, and burned hair warrant urgent airway intervention
- An anesthesia professional or senior emergency physician must always assess these patients early, as these are dynamic situations that may rapidly progress from one of stability to one of a threatened and compromised airway.
- Early intubation is undertaken in these at-risk groups.
- External compression from an adjacent pathology, e.g. trauma on the face and neck, goiter, lymphadenopathy, tumor and hematoma may be dangerous or rapid in onset, therefore early identification of patients at risk is vital.

2 2.4 Airway problem due to Tracheostomy tube obstruction

In a patient with tracheostomy tube, obstruction may be developed due to:

- Dry secretions or crust or hematoma if the tube insertion is fresh
- Dislodged from the site of insertion and the opening is covered by tissue

Action

1. Wipe neck opening with gauze
2. Attempt to suction tracheostomy tube with saline
3. Remove tracheostomy inner tube and suction well if it is metallic
4. If the tracheostomy tube is plastic and has cuff, deflate the cuff
5. Once the tracheostomy is open, give oxygen as necessary and reassess and monitor
6. If no improvement, call your senior

2.3 Rapid sequence intubation technique and organization

Preparation

- Make sure your emergency unit is equipped with ABCD resuscitation drugs and equipment. Have short list of these equipment and design a scheme for daily checking and refill.
- Organize these resuscitation equipment and drugs in one area/trolley, so they are easily accessible for emergency periods
- Have a corner or a room for resuscitation with the above arrangements Equip this area also with monitoring devices such as cardiac monitor, pulseoxymeter, o2, O2 administration devices, suction machine and defibrillator When a patient is identified as having respiratory failure,first consider high flow oxygen with face mask and non-invasive- CPAP, or BiPAP if it is appropriate
- If your decision is to intubate and ventilate, assume emergency patients as having full stomach and rapid sequence should be the intubation technic used
- Before giving any drug for intubation, rapid assessment of the airway is mandatory to predict difficult airway.
- If you expect difficult airway, consult senior personnel for alternatives if failed.
- Put the patient on cardiac monitor, and pulseoxymeter
- Mobilize 3-4 colleagues who can assist you and describe for them your plan and how they are going to assist you
- Draw ketamine 1-2mg/kg, or Thiopental 3-4mg/kg, suxamethonium 1-2mg/kg, Atropine 04-1mg

Pre- oxygenation: Preoxyginate with 100% oxygen for 4-5min, or if patient is cooperative advise him/her to take 5big breathing

Premedication: Administer the anesthetic drugs and relaxants as the same time

Position: Hold face mask tightly, position on sniffing position, and avoid bagging
Pressure: Tell your assistant to Apply cricoid pressure on the cricoid curtilage
Placement: Do direct laryngoscopy and intubate, and inflate the ETT balloon
Post intubation care: Check for proper placement of the tube by direct visualization of the cords during ETT insertion, observation of clouding of the tube, auscultation of the chest and if possible with End tidal CO2 measurement after insuring the proper placement of the tube secure properly the tube with tape and bandage.

Section 3 Selected Respiratory Emergencies

3.1 Acute Severe Asthma

Asthma is a recurrent airway disease affecting bronchus with reversible airflow obstruction. It is due to hyperresponsiveness of airways, broncho-constriction and inflammation.

Clinical features and severity grading
Table1. Classification of severity of an asthma attack

Parameter	Mild	Moderate	Severe	Imminent respiratory arrest
Breathless	Walking Can lie down	Talking Prefers to sit up	At rest Hunched forward	
Talks in	Sentences	Phrases	Words	Unable to speak
Alertness	May be agitated	Usually agitated	Always agitated	Drowsy or confused
Respiratory rate	Increased	Increased	Often > 30/min	
Accessory muscles and suprasternal retractions	Usually not	Usually	Usually	Paradoxical thoracic and abdominal movements
Wheeze	Moderate, often only end -expiratory	Loud	Usually loud	Absence of wheeze
Pulse rate	100	100-120	>120	Bradycardia
PEF after inhalation of salbutamol	>80%	60-79%	<60% or (< 100l/min in adults)	Impossible to measure

Treatment

1. Oxygen administration: high flow with mask with reservoir bag, keep saturation ≥ 95% Ventilator support is provided when there is severe respiratory distress or failure.
2. Begin reliever drugs immediately: depending on availability of the drugs and equipment one of the following reliever drugs can be administered and repeated every 20 minutes..
    1. High dose salbutamol (6-10 puffs), repeat every 20' during the first hour (Ipratropium bromide-Anticholinergis-0.5mg inhalation/MDI can be given together if available). OR
    2. Salbutamol 2.5-5 mg by a nebulizer, with oxygen every 20'.(continue 5mg every 1-4 hours till recovery) OR
    3. Aminophylline: a loading dose of 3-5 mg/kg in D/W over 10-20 minutes (Maintenance dose with dextrose 5% at a dose of 0.6mg/kg/hour) OR Severe
    4. Adrenaline 0.3-0.5 cc subcutaneously, repeats after 20 min if no i prove ment.

cases

3. Begin Controller drugs immediately

- Prednisolone, 40-60 mg P.O. should be started immediately, Po steroids is preferable, and it should be continued at least for a minimum of 5-7 days(Salvage Therapy) and no need to tapper.
- If patient cannot take P.O. prednisoloneintravenous hydrocortisone, 4-6 mg/kg or 200 mg should be given every 4-6 hours, change to P.O. when possible.
- Symbicort smart (Symbicort and formoterol) have both reliever effect of formoterol and controller effect of symbicort, hence can be given immediately and replace other drugs.

4. Adjunct therapy :if the response is poor to the above regimen-magnesium sul phate 1-2gm IV in 5minutes

Technique of using inhaler drugs

- Test the inhaler: shake well and release one puff into the air
- Breathe out gently & place the mouth piece in the mouth and close lips around it
- Tilt head slightly backwards, breathe in slowly and press down the canister to release one dose
- Remove the inhaler and hold breath for 10 seconds and breathe out slowl

Mechanical ventilation in severe asthma

- It is indicated only in severe respiratory distress with deteriorating mental status or coma, respiratory or cardiac arrest, cyanosis and hypoxemia on O2,PaCO2 greater than 50 mmHG and rising > 5mmHg/hr., minimal chest movement/air exchange and Pneumothorax.
- Type of mechanical ventilation-non-invasive positive pressure ventilation (NIPPV) like CPAP can be used in patients who are conscious cooperative but in more severe cases intubation and invasive ventilation might be needed.

Special conditions in ventilator support of severe asthma

- Experienced personnel should intubate
- Ketamine is the good choice for intubationif there is no contraindication such as hypertension, increased ICP
- permissive hypercapnia or "controlled hypoventilation" is preferred which is achieved through low tidal volume(5-7ml/kg),lower respirator rate, lower inspiratory time, low PEEP and prolonged expiratory time.

Treatment of mild to moderate Attack

- Salbutamol 4-8 puffs every 20 minutes for the first hour
- Oral prednisolone =40-60mg Po per day for 5-10 days (no tapering) –may not be given in mild attack.
- The patient is then reassessed
  - o Complete response –disappearance of clinical signs (and PEF> 80%
    - -The patient is kept for one more hour. If stable, the patient can be discharged to continue treatment at home.
  - o No response/incomplete response – no or incomplete disappearance of clinical signs (or PEF<80%)
    - - After the first or second hour, the patient should be treated as for a severe attack and be kept in the emergency room for at least 6 hours to continue treatment.

Assessment of response

Clinical improvement

- Patient is less distressed
- Decreased respiratory rate and heart rate
- Able to talk in sentences
- Louder breath sounds on auscultation (may be more wheeze)
- Pulse oximeter- aim O2 saturation of 90-93%
- Monitor heart rate and Oxygen saturation continuously and measure BP frequently

Discharge plan and ED Education

1. Inhaled corticosteroid should be started or dose adjusted before discharge
2. Appropriate continuum of care should be established.
3. Education on how to take medications during exacerbations and routine use.
4. An action plan to recognize early signsand home self-management of asthma exacerbations
5. Appropriate intensification of therapy in case of deterioration and Prompt communication between patient and clinician during serious deterioration.

3.2 Pneumonia

Pneumonia is an acute infection of the pulmonary parenchyma that is associated symptoms and signs of acute infection and acute infiltrates on a chest radiograph consistent with pneumonia.

Classification for pneumonia based on acquisition environnent

- **Community acquired pneumonia:** Acute pulmonary infection in a patient who is not hospitalized or residing in a long-term care facility 14 or more days before presentation
- **Hospital-acquired pneumonia:** New infection occurring 48 or more hours after hospital admission
- **Ventilator-acquired pneumonia:** new infection occurring 48 or more hours after endotracheal intubation
- **Health care–associated pneumonia patients**

  - Residence in a nursing home or extended-care facility;Home infusion therapy (including antibiotics) within the past 30 days;Long-term dialy sis within the past 30 days;Home wound care within the past 30 days; receiving chemotherapy ;Immuno-compromised patients

Diagnostic strategies

- History and physical examination
- Investigations: CBC, CXR, Organ function tests serum electrolytes (in critical patients)
- Blood culture : Indications for blood culture
  - o Those admitted to the intensive care unit (ICU), patients with severe sepsis and septic shock, those with leukopenia, cavitary lesions, severe liver dis ease, alco hol abuse and asplenia

- For para pneumonic effusion: do pleural fluid analysis incluing cells with differential count, glucose, protein, LDH, Gram stain, AFB,and cytology.

Treatment

First decide whether the patient is a candidate for admission or not. To decide admission , among different criteria, here CURBS-65 is used.

The CURB-65 criteria (Confusion, Urea>7mmol/L, Respiratory rate >30, Blood pressure <90/60, Age >65)

- For all patients, the CURB-65 score should be interpreted in conjunction with clinical judgment. Patients with a CURB-65 or CRB-65 score of >2 patient should be admitted

Outpatient management of uncomplicated pneumonia

- Azithromycin 500 mg po/d for 3 days first lin
- Doxycycline 100 mg po BID for 10-14 days(2nd line)

Therapy for outpatient management of patients with severe co morbidities: (chronic heart, lung, liver, or renal disease, diabetes mellitus, alcoholism, malignancies, asplenia)

- Amoxicillin-clavulanate 2 gm P.O. BID for 7-10 days + Azithromycin 500 mg po/d for 3 days
- Encourage fluid intake and analgesics (paracetamol) if needed
- In patient therapy of non-ICU patients with severe pneumonia
- Assess oxygenation status of patient(maintain SaO2 94% and above)
- Assess fluid status of the patient and resuscitate with crystalloids
- Antipyretics/ analgesics if needed
- Ceftriaxone 1gm IV BID +Azithromycin 500 mg po/d

Empirical Therapy for Patients with Suspected Health Care-Associated Pneumonia

- Assess oxygenation status of patient(maintain SaO2 94% and above)
- Assess fluid status of the patient and resuscitation
- Antipyretics/ analgesics if needed and monitor vital signs and urine output.
- Cefepime/ceftazidime 1-2 gm every 8-12hr + ciprofloxacin 400 mg IV TID + Vancomycin 1 gm IV BID OR Meropenem 1gm IV TID + Ciprofloxacin 400 mg IV TID + Vancomycin 1 gm IV BID

Inpatient Therapy for Intensive Care Unit Patients in Community acquird pneumonia:

- Ceftriaxone 1gm IV BID + azithromycin 500 mg po/d

In patient therapy for ICU patients with pseudomonas risk:

- Prolonged hospital or long-term care facility stay, Structural disease of lung (e.g. heart failure, bronchiectasis), Steroid treatment (>10 mg prednisone / day), Broad-spectrum antibiotics for >7 days in the past 1 month, AIDS especially CD4 < 50/ml, Neutropenia (ANC <500/dL)
  - Meropenem /ceftazidime/cefepime 1 gm IV TID + Ciprofloxacin 400 mg IV TID

Therapy for aspiration pneumonia

- Ceftriaxone 1g IV BID + Metronidazole 500mg IV/PO TID OR
- Clindamycin 600mg Po/IV TID

Disposition plan

- Monitor: Temperature, pulse, respirator rate, and blood pressure, mental status, oxygen saturation and urine output
- Discharge if: Temperature <37.8°C, heart rate <100/min, respiratory rate <24/min, systolic blood pressure >90 mm Hg, oxygen saturation >90% without oxygen supplement and ability to tolerate oral intake.
- For those patients who are candidate for discharge IV antibiotics changed to P.O.antibiotics (Amoxicillin-clavunate acid 2 gm po BID for 7 days.

Indication for ICU admission:

- Septic shock;Respiratory failure, Adult Repiratory Disteress Syndrome(ARDS); Coma;Acidosis and significant electrolyte disturbance

3.3.Pulmonary thromboembolism

Pulmonary thromboembolism (PE) is occlusion of pulmonary arteries by emblus disloged from thrombus at a distant site,mostly deep vein of the lower limb,and is one of the potentially life threatening illnesses with a mortality rate of approximately 30 percent without treatment. Therapy with anticoagulants decreases the mortality rate to 3 to 8 percent. ED visit related to weakness, shortness of breath, dizziness or syncope, pain, extremity discomfort, or nonspecific malaise or functional deterioration could represent a potential PE.The presence or absence of sudden onset of symptoms neither increases nor decreases the probability of PE.

Diagnosis

CXR seldom provides specific information but is useful to suggest alternative diagnoses, such as pneumonia, CHF and Pneumothorax. Unilateral basilar atelectasis on the chest radiograph increases the probability of PE. Pleural based opacity (Hampton's hump), dilated pulmonary artery (Pala's sign) unilateral lung oligemia (Westermark's sign) are rare radiographic manifestation of PE.

ECG provides more information about the presence of alternative diagnoses, such as pericarditis or cardiac ischemia. The most common ECG signs are rapid heart rate, symmetrical T-wave inversion in the anterior leads (V1-V4), S1Q3T3 pattern, and incomplete or complete right bundle branch block. Any one of these findings approximately doubles the probability of PE in a symptomatic patient

Spiral CT- is diagnostic in 98 percent of patients with PE.

Risk factor assessment: Wells criteria is one of risk strafication methods which is described beow:

Table 2. Wells criteria and modified Wells criteria: clinical assessment for pulmonary embolism

Clinical symptoms of DVT (leg swelling, pain with palpation)	3.0
Other diagnosis less likely than pulmonary embolism	3.0
Heart rate >100	1.5
Immobilization (≥3 days) or surgery in the previous four weeks	1.5
Previous DVT/PE	1.5
Hemoptysis	1.0
Malignancy	1.0
Probability	Score
Traditional clinical probability assessment (Wells criteria)	
High	>6.0
Moderate	2.0 to 6.0
Low	<2.0
Simplified clinical probability assessment (Modified Wells criteria)	
PE likely	>4.0
PE unlikely	≤4.0

TREATMENT IN THE ACUTE PHASE
1 ABC APPROACH, RESPIRATORY AND HEMODYNAMIC SUPPORT

- Modest fluid challenge for patients with PE.
- Vasopressors for patients who are not responding to fluid challenge, nor epinephrine is preferred if available.
- Administer oxygen for hypoxaemia. When Mechanical Ventilation is indicated, give low tidal volume (6 ml/kg) and low PEEP; end- inspiratory plateau pressure 30cm of H2O
- Empiric anticoagulation for patients with suspected pulmonary embolis

Heparin administration and monitoring

- The dose of UFH is 5000 IV bolus followed by 1000hourly , but if continuous infusion is not possible as 10,000-18500 IU BID can be given(SC anticoagulant effect is delayed for acute VTE)
- LMWH like Enoxaparin 1mg/kg SC Bid (or 1.5mg/kg daily) is preferred over UFH as they carry low risk of inducing major bleeding and HIT (Heparin induced thrombocytopenia).
- aPTT should be checked 1 hr. after bolus of heparin and every 6 hrs ,if difficult at least once a day targeting aPTT 1.5 to 2 times the patient's baseline
- In patients with severe renal insufficiency or renal failure diagnosed with acute PE, UFH is favored over LMWH because of its short half-life and rapid reversal.

Dispose to resuscitation room and Intensive care units if,

- Massive PE is with a SBP of <90 mm Hg for >15 minutes, or <100 mm Hg in a patient with a history of hypertension, or a >40% reduction in baseline SBP.
- Sub massive PE is characterized by a normal or near-normal BP, but with other evidence of cardiopulmonary stress

Special Population: Pregnant Women

- An effort should be made to reduce radiation exposure by shielding and limiting testing where possible.
- Warfarin is better avoided during pregnancy.
- UFH and LMWH are safe during pregnancy, and can be started in suspected patients

3.4 Hemoptysis

Hemoptysis is coughing of blood that originates from the respiratory tract below the level of the larynx.

- Its causes can vary from simple bronchitis to life threatening pulmonary embolism
- It can be used as an alarm symptom for possible underlying lung cancer in elderly smokers
- It requires emergent intervention results from bleeding from the bronchial vessels 90% of the time

Clinical features

- Can be classified as mild (<20ml/24hours), moderate (20 to 600ml/24 hrs), severe to massive (>600ml/24 hours). Classes are arbitrary.
- Since exact quantification is difficult in the ED, it can be classified as speckled or blood streaked sputum, gross hemoptysis with consistency of clotted or unclotted blood, and massive hemoptysis interfering with oxygenation and/or ventilation
- It is often difficult to know the etiology based on signs and symptoms
- An abrupt onset of cough with bloody purulent sputum, with or without fever, suggests acute pneumonia or bronchitis
- A chronic, productive, blood streaked cough suggests chronic bronchitis, Tuberculosis, bronchiectasis or neoplasm.
- Hemoptysis can be the only initial presentation of a neoplasm
- Alveolar hemorrhage syndromes from vacuities present with dyspnea and mild hemoptysis associated with renal disease and hematuria.
- In children with hemoptysis, ask about choking spell.
- Rhinitis and upper respiratory tract inflammation can cause friable mucosa and post nasal bleeding that may confuse hemoptysis
- Patients with mitral stenosis can present with hemoptysis
- True hemoptysis is usually bright red in color with frothy appearance, alkaline PH and macrophages in it unlike GI bleeding which is usually acidic and dark red .

Diagnosis

- Evaluation and intervention should be done simultaneously in unstable patients
- Chest X-ray: Is a good initial diagnostic tool, may show abnormality in 70-85%.
- Bronchoscopy: Diagnostic/therapeutic is recommended within 48 hours of symptoms.
- Chest CT: non-invasive modality which more effectively peripheral lesions, sensitive in delineatingbronchiectasis and nodular lesions

Treatment

- The ABC of life is a priority. And Prone positioning is recommended
- If airway is compromised intubate using a large sizeET tube for the particular age and sex to allow bronchoscopy
- If persistent bleeding, continue resuscitation with fluids and blood products and consult surgical team and pulmonology team.
- Considerfresh frozen plasma or platelet transfusion for identified coagulopathies or for those taking warfarin without waiting for INR
-

Section 4 Cardiovascular Emergencies

4.1 Acute Heart Failure

Acute Heart Failure is the sudden worsening of the signs and symptoms of heart failure, which typically includes difficulty breathing (dyspnea), leg or feet swelling, and fatigue and is mostly due to acute decompensation of Chronic Heart Failure (CHF), but can occur as a first occurrence. The clinical manifestation may vary from mild decompensation to Acute Cardiogenic Pulmonary Oedema and Cardiogenic Shock.

Chronic Rheumatic heart disease with valvular heart disease is the most important cause in Ethiopia but other important causes are congenital heart disease, hypertensive disorders, Ischemic heart disease, pericardial disease, cardiomyopathy and arrhythmia.

Diagnose Heart Failure Exacerbation

History & Physical exam -Dyspnea on exertion, Paroxysmal Nocturnal Dyspnea, orthopnea, pink frothy sputum, JVP raised > 4cm and Neck vein distention, Leg edema, S3 gallop, displaced Point of Maximal Impulse, Basal Crackle .
Initial Management

- Evaluate for ABC, put on cardiac monitor , place in semi sitting position,and open IV
- Identify if patient is stable or not using the following important clues:

  - BP <90/60(Hypotension)
  - RR > 35 and other signs of respiratory distress,O2 sat < 90%
  - Severe increasing work of breathing /sweating)

Acute heart failure could be stable or unstable (cardiogenic shock or cardiogenic pulmonary edema

Management of Acute heart failure in different scenarios:

Cardiogenic Pulmonary edema - Normal BP>90/60 but in severe respiratory distress,
fatigued, then endotracheal intubation and mechanical ventilation is necessary.

- Oxygen administration with appropriate system (Support Breathing)-5 to 6 liters / minute by mask, to achieve oxygen saturation of more than 93%.

Other option to support breathing when saturation is not achieved:

- If noninvasive positive pressure ventilation system is available con sider Continuous Positive Airway Pressure [CPAP] or Bi-level Posi tive Airway Pressure [BiPAP])
- Should the oxygen saturation be inadequate or the patient develops respiratory muscle fatigue invasive ventilation might be necessary

- Furosemide(Lasix)40 mg IV or <0.5mg/kg and
- Escalate the dose of furosemide every hr. with 40 mg increment or doubling the dose depending on response to achieve urine output ≥0.5ml/kg/hr. The maximum recommended dose of Furosemide as a single IV bolus is 160-200. (chest auscultation, Sao2)

Note: there is no standing dose of furosemide that is universal for every patient and decision on frequency and dosage must be individualized depending on renal status, BP and response. When there is no response a perfuser can be used to administer continuous Lasix infusion at 10-40mg/hr. If the urine output remains below 1ml/kg/hr., the infusion rate can be increased each hour as necessary till a maximum dose of 80-160 mg/hr.

- Nitroglycerine 0.4 mg sublingual 3 doses every 5 minutes or isosorbide dinitrate 10mg, repeat every based on response. If pulmonary edema persists and no evidence of shock IV Nitroglycerin 5-10 microgram/min infusion
- Morphine 2-4mg IV boluses can be used in anxious pts(for reduction of preload and anxiety)
- If available and increased work of breath, trial of CPAP 12/6
- If after 1 hr. CPAP SaO2 is low and work of breathing is significant then intubate if mechanical ventilator is available [use high PEEP 10-15]
- Monitoring: includes Resolution of symptoms (dyspnea, orthopnea),V/S and SaO2,urin output and fluid balance ,Serum electrolytes, upper border of crepitation to see for resolution of lung congestion, BUN, Creatinine,
- If improved admit to wards, if not ICU

Cardiogenic Shock (BP<90/60 and features of hypo perfusion)

- Put the patient on cardiac monitor stabilize the ABC
- Fluid challenge with N/S 200- 500 cc IV fluid bolus(5 - 10 ml/kg) for restoring vascular volume and preload, depending on the level of crepitations on the chest
- When invasive monitoring is possible: if CVP is < 15 mmHG fluid is given and if it is >15 mm HG innotropes/pressers are used.
- Consider vassopressors/innotropes- if SBP is around 90mmHG consider

dobutamine, if SBP is 70-90mmHG consider dopamine (3-10 mg/kg/min – 50 mg/kg/min) or if SBP<70mmHG consider adrenaline 2-10 microgram/min or escalate the dose of dopamine gradually. If other pressor are not available adrenaline 2-10 microgram/min with escalation can be used.

- Diuress with furosemide 20-40mg IV bolus after inotropic support if there are signs and symptoms of pulmonary edema when BP is elevated to low normal.
- Reduce work of breathing or desaturation through Facemask O2 or undertake ventilator support like using CPAP/BiPAP.
- Look for treatable causes: If tachyarrhythmia is contributing to cardiogenic shock in heart failure- Amiodarone or digoxin, pain management in AMI.
- Admit to ICU

Stable Heart Failure

- Lasix 40mg -120 mg based on previous close and renal function status.
- Start ACEI like enalapril or Lisinopril 2.5 mg Po stat if not started.
- Consider digoxin.
- Consider Spironolactone in severe disease with inadequate response to Lasix.
- Discharge with follow up visit and advise patient on lack of salty diet, serial weight measurement, exercise, smoking cessation and need for outpatient B-blocker therapy.

NB: In all cases identify and treat precipitating factor and underlying cause for the failure.

Precipitating factors are summarized with the Pneumonic – HEART FAILES

H- Hypertension, E-Endocarditis, A- Arrhythmia- Rheumatic fever /Myocarditis,

T- Thyrotoxicosis and pregnancy, F- Fever (infection), A-Anemia, I-Infarction, L-Lack of compliance,

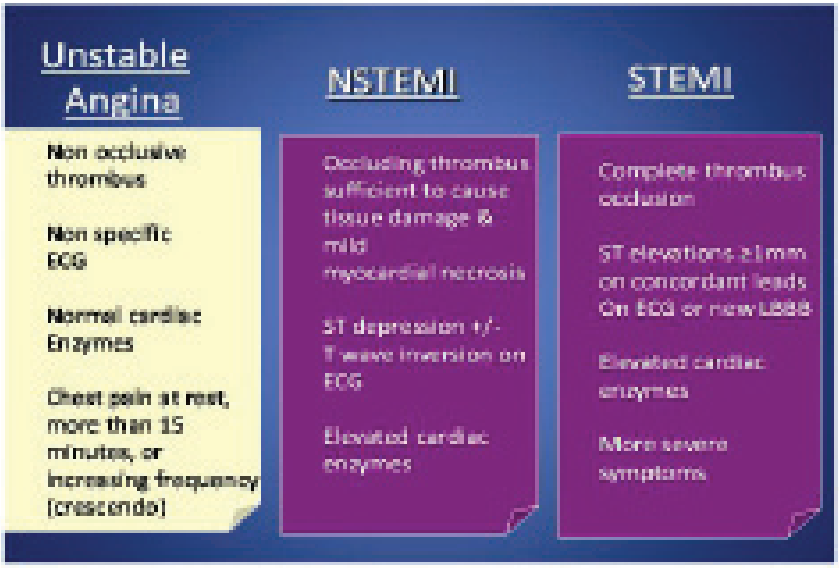
E-Embolism (Pulmonary), S-Stress (emotional, dietary, fluid excess, physical)

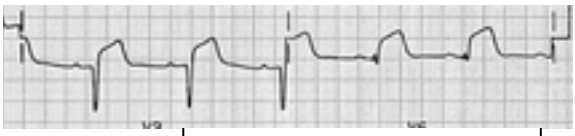
4.2. Acute coronary syndrome

Acute coronary syndrome defines a spectrum of clinical presentations that is related to acute coronary diseases. This involves unstable angina, ST elevation myocardial infarction and non ST elevation myocardial infarction. The symptoms of acute coronary syndrome can range from no chest pain to severe chest pain that is significantly distressing to the patient. The Chest pain/Discomfort Pain lasting for more than 20 minutes, Radiating to the arms, neck or jaw. It can also be Epigastric pain and Associated Nausea, sweating or diaphoresis, Shortness of breath, fatigue. Atypical symptoms are common in diabetic patients, elderly and women.

Patients with risk factors are those with Age> 55 for female,>45 for male, male sex, smoking, family history, Diabetes, Hypertension, Hypercholestromia.
Diagnosis:
It is made based on typical clinical features, ECG findings and cardiac markers (CKMB and troponin I)

Diagnosis:





Eg.ST elevation on ECG

Diagnostic tests:
1. ECG: done at presentation;repeat requently to see changes

*UA/NSTEMI= ST depression/transient elevation or deep T inversion (>=0.3mV)
* STEMI=New ST elevation in 2 contiguous leads >= 2 mm/2 small squars in men or 1.5 mm in women in leads V2-3 and/or 1mm in other leads OR new LBBB . ECG must also be analyzed for arrhythmias.

2. Cardiac biomarkers: testing at presentation & 6–12 h after sx onset *Cardiac troponins (T&I)-rise 20 -50 Xs Upper normal limit in acute MI; rise 4-8 hr after injury; may remain elevated for 7-10 days; more Specific& Sensitive than CK-MB .Creatinekinase (CK)-rises in 4–8 hr; normalize by 48–72 h.

3. Echocardiography:may show new regional wall motion abnormality,RBS, lipid profiles

5. CXR: to look for pulmonary edema; R/o other DDx like PTE, pericarditis.
Management of ACS: should focus on stabilizing the patient's condition, relieving ischemic pain, and providing antithrombotic therapy to reduce myocardial damage and prevent further ischemia. The goal is early revascularization.

1) General measures: Continuous ECG monitoring for arrhythmia & ST changes

- V/S: frequent until stable, then Q 2- 4hr & as needed
- O2 (2-4 lit/min) keep SaO2>93%
- Bed rest,Sedation with diazepam 5mg po BID,VTE(venous thromboembolism) prophylaxis
- NPO except for fluid diet until stable; IV fluid – eg. for inferior MI
- Glycemic control-goal is RBS of 140-180mg/dl(if > 180mg/dl,give regular insulin-1-2IU for each 50mg/dl increase above 180mg/dl, by measuring RBS Q 6hrs)

2) Medications:

- Nitroglycerin (NTG) : sublingual 0.4 mg Q 5 min for three doses as needed or chest pain (C/I= low BP, sildenafil use)
- Morphine sulphate: 2–5 mg IV , may be repeated Q 5–30 min as needed to relieve chest pain (can also use pethidine 25-50mg IM/IV or tramadol 100mg IM)
- Aspirin(ASA): loading:162–325 mg chewed immediately, then 75–162 mg/d plus

Clopidogrel-loading: 300mg Po, then 75 mg/d for at least 1 yr (but ASA lifelong)

- Metoprolol: 25–50 mg PO q 6 h ,target HR=60-80/min (If HTN, ongoing pain,tachycardia: give IV over 1–2 min by 5mg increments) or Atenolol 25-50mg PO. C/I- CHF,bradycardia
- Un Fractionated Heparin: Bolus 60–70 U/kg (max 5000 U) IV then infusion of 12–15 U/kg/ h (initial maximum 1000 U/h) titrated to aPTT 50–70 s * If no perfuser,12,500U SC BID is possible; OR LMWH (Enoxaparin):1 mg/kg

SC Q 12 h(if GFR< 30,1mg/kg once daily)

- Warfarin: initially 2.5mg titrated to INR goal of 2-3(eg. for extensive anterior STEMI with severe left ventricular dysfunction,CHF,atrial fibrillation or LV thrombus)
- Statins: atorvastatin 80mg po/d is preferred. Others options are pravastatin/ simvastatin/lovastatin 40mg Po/day
- ACEIs: start low dose eg. Enalapril/lisinopril 2.5 to 5mg po/d OR captopril 6.25-12.5mg TID; then escalate gradually to clinically effective dose.

Invasive therapy in ACS: for high risk patients who present early, referral to a better set up is recommended (If the patient can afford)

- STEMI : fibrinolysis Vs PCI/CABG: Recommended door to PCI time is 90 minutes while door to thrombolytic in 30minutes.
- In Unstable angina/NSTEMI: PCI/ CABG; but fibrinolysis is not indicated. Com

Monitoring:

- o Patient should be on monitor to look for electrical complications like Vtac,Vfib , AV nodal blocks and atrial fibrillation(manage using ACLS algorithm)
- o ECG should be repeated every 15-30 minutes. First ECG is diagnostic only in 50%.
- o Vital signs: Every 15 minutes until stabilization
- o Cardiac markers: every 6 hours, CK MB and troponin will rise in 4-6 hours .
- o Manage chest pain: and follow for clinical improvement.
- o Watch for mechanical complications like heart failure, myocardial wall rap ture, papillary wall rapture, septum rupture, ventricular aneurysm, post myo car dial infarction pericarditis and Dressler's syndrome, and manage accord ingly.
- o Blood sugar should be determined and preferably maintained at less than 180mg/dl.

Disposition

- o All suspected patients with acute coronary syndrome should be admitted to resuscitation
- o Patients with STEMI should be assessed for eligibility and transferred to a facility where reperfusion therapy can be provided as soon as possible.
- o Patients with unstable angina or NSTEMI with persistent symptoms and com plications should be considered for PCI in coronary care unit.

o All patients with diagnosed acute coronary syndrome should be admitted to intensive care units at least for the first 48 to 72 hours.

4.3. Severe Hypertension

Hypertensive urgency (severe asymptomatic hypertension) is defined as blood pressure measurement ≥180/120 mmHg without any evidence of end organ damage.

Malignant hypertension: It is a type of hypertensive emergency with a sudden increase in blood pressure with associated hemorrhages, exudates, papilledema and fibrinoid necrosis in small arteries of the kidney, brain, and retina. It presents clinically as impaired renal function, retinopathy, and encephalopathy.

Hypertensive emergency: Blood pressure ≥180/120 mm Hg with evidence of end organ damage. It must be noted that in some patients with newly diagnosed hypertension, end organ damage can even be present at diastolic pressure as low as 100 mm Hg.

End organ damages in hypertensive emergency: These are complications of severe hypertension such as acute left ventricular failure, dissecting aneurysm, acute coronary syndromes, hypertensive encephalopathy, subarachnoid haemorrhage, haemorrhagic stroke, acute renal failure and eclampsia

Clinical Evaluation

The evaluation of these patients is as in any other patient with hypertension should include a thorough history and physical examination, particularly looking for signs of acute target organ damage and causes of secondary hypertension.

Work up

Look for any evidence of end organ damage. By performing Urinalysis, BUN and Creatinine, Peripheral blood morphology, ECG and Chest x-ray. When stroke is suspected get CT of the brain. Further investigations to look for secondary causes is necessary when there is clue for Renal parenchyma, Systemic disorders with renal involvement, Renovascular, Endocrine, Drugs, Coarctation of Aorta, preeclampsia/eclampsia.

Treatment of Hypertensive Crisis

The degree of end organ damage determines the rapidity of lowering the blood pressure because precipitous lowering of blood pressure has deleterious effects. The brain has an auto regulatory mechanism which allows it to adapt to a wide range of blood pressures. This auto regulatory set point is shifted to a higher level in patients having hypertension in attempt to maintain normal cerebral blood flow.

Hypertensive emergency

- Admit patient to emergency department or ICU. The initial goal is to reduce the blood pressure relatively quickly, by about 25% in the first 2-3 hours or to a level around 160/100 mmHg. This gives time for healing of necrotizing vascular lesions in the end organs without increasing the risk of ischemic events.

Choice of drug: An ideal drug to achieve this should be parenteral, rapid onset, and short acting to help adjust the dose based on response.(see table below) Oral antihypertensive are not preferred due to their slower onset and longer action and difficulty to monitor response minute to minute. Oral or sublingual drugs with rapid onset of action can result in an uncontrolled drop in blood pressure leading to stroke, myocardial ischemia.

- Once the blood pressure is controlled, oral therapy can be initiated with a goal to gradually reduce the diastolic pressure to 85 – 90 mmHg over weeks to months. Following stabilization of the patient's BP, subsequent management is tailored towards achieving optimal control using a combination of medications.
- Patient education on the problem, the medications, compliance and life style modification before discharge.

Table 3. List of Anti hypertensive drugs and dosage-

Drug	Dose	Onset/Duration	Remark
Labetalol	50 mg over 1 minute repeat every 5 minute if necessary to a max of 200 mg then 2 mg/min	5 min/3 – 6 hours	
Hydralazine	5 – 10 mg IV, repeat after 20-30 minutes if needed, Maintenance infusion also can be given 50-150 µ / min	20 – 30 min/3 – 8 hours	Caution in acute coronary syndromes, cerebrovascular accidents and dissecting aneurysm *Available in Ethiopia
Sodium nitroprusside	0.25 – 10 µ /kg/ min	1 min/1 – 5 minutes	Ideal drug in Hypertensive urgency but it is not available and needs thorough monitoring
Nitroglycerine	5 – 100 µ /min	2 – 5 min/3-5mins	Preferred in acute coronary syndromes and acute pulmonary oedema
Esmolol	i.v. bolus 250 – 500 µ /kg over 1 min i.v. 50 – 200 µ / kg/min for 4 min. May repeat sequence	1-2min/3-10min	Used in peri-operative situations and tachyarrhythmia

In pregnancy, 200 mg labetalol in 50 ml normal saline and start infusion at 4 ml/hour.
- * In pregnancy, the initial dose is 25 mg/min IV infusion (25 mg in 500 ml normal saline at 30 ml/hour

Hypertensive urgency (severe asymptomatic hypertension)

- Goal is to reduce the blood pressure to <160/100 mmHg over hours to days. There is no benefit of rapidly reducing the blood pressure and it rather has deleterious effects. Dangers of rapid reduction in blood pressure - rapid reduction of BP (within minutes to hours) in an asymptomatic severe hypertension or hypertensive urgencies is best avoided as it may precipitate ischemic events

o Counsel on adherence and restart initial antihypertensive if noncompliant

o add another drug for difficult to control with one drug and enroll to chronic care
o Newly diagnosed: Initiate oral antihypertensive and enroll to chronic care clinics.

Any of the long acting antihypertensive can be used for the management of these patients. The patient should be observed for a few hours to ascertain that the blood pressure is improving. They can then be enrolled into chronic hypertension care clinic.
Examples of oral drugs used in hypertensive urgencies

-Nifedipine 20-40 mg Po BID
-Labetalol 200mg-800mg Po BID

4.4 Cardiac Tamponade

The accumulation of fluid in the pericardial space in a quantity sufficient to cause serious obstruction to the inflow of blood to the ventricles results in cardiac tamponade which causes results in obstructive cardiogenic shock.

Etiology

- Trauma; gunshot or stab wounds, blunt trauma to the chest
- Inatrogenic accidents: perforation during cardiac catheterization, central line, etc
- Pericarditis (TBC, Pyogenic, SLE etc)
- Malignant pericardial effusion
- A ruptured aortic aneurysm
- Radiation to the chest
- Other causes: hypothyroidism, uremia,AMI,etc

Diagnosis

Clinical: Beck's triad is clinical clue for diagnoses, it includes:

- Low blood pressure and weak pulse, and pulsus paradoxus because of low cardiac output.
- Raised JVP and distended neck veins because ofelevated R atrial pressure.
- Faint/ muffled heart sounds and tachycardia

ECG characteristics: reduction in amplitude of the QRS complexes, and electrical alternans of the P, QRS, or T waves.
Echocardiographic diagnostic criteria
One or more of the following in the setting of moderate to large effusion and symptoms or a paradoxical pulse:

- Right atrial systolic collapse and Right ventricular diastolic collapse
- Reciprocal respiratory ventricular inflow(25% reduction mitral inflow velocity in inspiration)
- Inferior vena cava plethora

Treatment

Cardiac tamponade is a medical emergency and patients should be closely monitored and obtain urgent management:

- Oxygen via nasal prong or face mask
- Volume expansion with isotonic saline ,blood, plasma, as necessary, to maintain adequate intravascular volume in order increase in cardiac output after volume expansion
- Bed rest with leg elevation in order to facilitate venous return
- Inotropic drugs (e.g, dobutamine) - These can be useful because they increase cardiac output without increasing systemic vascular resistance
- Pericardiocentesis: use subxiphoid or anterior approach depending on the pool of fluid and drain as much as possible fluid in tamponade for therapeutic and diagnostic purpose.
- After pericardiocentesis, leave the intrapericardial catheter in place after secur ing it to the skin using sterile procedure and attaching it to a closed drainage system, periodically check for reaccumulation of fluid, and drain as needed.

Indication of Emergency pericardiocentesis

Emergency pericardiocentesis is indicated in cardiac tamponade which is diagnoses using clinical features of instability and echocardiography signs.

Contraindications

Relative contraindications are Pericardial effusion of less than 2 cm width and presence of Bleeding disorder (including platelet count <50 × 109/L, INR >1.5), in that case consultation is necessary with cardiology expertise.

Complications of Pericardiocentesis: Puncture of the heart, laceration of liver/stomach or colon in the subxiphoid approach, pneumothorax, and puncture of internal mammary artery.

Section 5 Endocrinological Emergencies

5.1 Hypoglycemia

Blood glucose level<70 mg/dL

Clinical Features: Divided into two broad categories:

A) Neuroglycopenic (CNS) symptoms: - alterations in consciousness, seizures, focal neurologic deficits, and unresponsiveness.

B) Autonomic symptoms: - irritability, nausea, vomiting, palpitations, and tremor, sweating, changes in pupil size, bradycardia, and salivation.

Diagnosis: Use three criteria - referred to as "Whipple's triad"

A. Signs and symptoms of hypoglycemia
B. Documentation of low blood glucose when the signs and symptoms occur
C. Disappearance of the signs and symptoms when blood glucose levels are raised.

Treatment

- In patients with altered mental status,
- 50% dextrose in water is administered IV as a bolus dose of 50 mL.
- When regains consciousness,
- Oral feeding should be continued to prevent recurrence of hypoglycemia if able to take /
- IV infusion of 10% dextrose in water at a rate to maintain the serum glucose >100 mg/dL [5.55 mmol/L].
- Determine RBS Q30 minutes for 2 hours
- Failure to respond to IV glucose administration
- Consideration of other causes : sepsis, toxin, insulinoma, hepatic failure, or adrenal insufficiency
- Glucagon may be used SC or IM in the absence of IV access.
- 1-2mg glucagon IM or SC: may repeat q20min.

DISPOSITION AND FOLLOW-UP

- Patients can be discharged from ER when, sign & symptoms are **resolved, cause is isolated & addressed.**

5.2 Thyroid Emergencies

5.2.1. Myxoedema coma

Clinical presentation – hypothermia, altered conscious level, bradycardia, hypotension, delayed tendon reflexes in a known hypothyroid patient or with deranged TFTs.

Precipitating factors such as: Infections, surgery or trauma, acute myocardial infarct, stroke, hypothermia, sedative-hypnotic or narcotics drugs.

Investigations

- CBC – macrocytic anaemia, leukocytosis
- Blood glucose - hypo glycaemia
- Arterial blood gases – hypoxaemia
- Thyroid function tests – hypothyroid picture
- Serum electrolytes – low Na+
- Blood urea and serum creatinine
- Infections screen – blood and urine culture
- Others – CXR, ECG

Treatment:

- Treat the precipitating event.
- Usually treatment started empirically.

Supportive measures

- Give oxygen according the severity of hypoxia using nasal prongs or face mask. If no improvement, consider MV (mechanical ventilation)
- Correct electrolytes to normal.
- Ensure adequate hydration.
- Hypothermia should be corrected.

Steroids –

Thyroid hormone replacement- there is controversy over whether T4 or T3 is better in the treatment of Myxoedema coma. Both the oral or intravenous form of T4 or T3 can be used.

I. T4 - a dose of 0.5 mg daily

II. Maintenance doses daily of 0.1 mg – 0.2 mg one week later.

T3 - low dose T3 at 20 micrograms every 12 hours. T4 may be substituted for T3 when the patient is improving.

5.2.2 Thyrotoxic Crisis

It is a state of decompensated thyrotoxicosis, with failure of organs to cope with the additional metabolic demands.

Clinical presentations:

- Fever, sweating, altered mental state such as restlessness, delirium, coma, and myopathy.
- cardiac - arrhythmias, heart failure,
- gastrointestinal - diarrhea, abdominal pain,
- liver failure – jaundice

Look for the precipitating factors such as: infections, surgery, trauma, pulmonary embolism, drugs (e.g. Amiodaron), acute myocardial infarct,and diabetic ketoacidosis

Investigations

- Full blood count
- blood glucose
- thyroid function tests
- serum electrolytes
- blood urea and serum creatinine
- infections screen – blood and urine culture
- others (to look for causes) – CXR, ECG

Treatment:

- **Treat the precipitating cause.**
- Give sedation such as intravenous Benzodiazepines.
- Supportive measures: Oxygen, ensure adequate hydration, reduce fever
- Treat cardiac failure and arrhythmias.
- Give vasopressors infusion if hypotensive.
- Tepid sponging and paracetamol for fever. (Aspirin is contraindicated as it displaces bound thyroxine from its carrier protein.)
- Give intravenous vitamin B complex.

Specific measures

1. Inhibition of thyroid hormone formation

Propylthiouracil 150 - 300 mg given 6 hourly for first 24 hours and then reduce dosage to 100 – 200 mg 8 hourly. OR

Carbimazole 15 – 30 mg 6 hourly for first 24 hours and then tapper dosage .

2. Receptor blockade – Propranolol 40–60 mg orally every 4 h or IV propranolol 2 mg 6 hourly OR use other alternatives like metoprolol 50mg PO QID

3. Steroids –dexamethasone 2mg IV QID.

4. A saturated solution of potassium iodide (5 drops SS KI every 6 h)

5. Endocrinology consultation.

5.3. Adrenal Crisis

Adrenal insufficiency: is deficiency of adrenal gland hormone production in the cortex.

Primary adrenal insufficiency, or Addison's disease, is due to intrinsic adrenal gland dysfunction and results in decreased cortisol, aldosterone, and sex hormone production.

Secondary adrenal insufficiency: is due to hypothalamic-pituitary dysfunction with failure to secrete corticotrophin-releasing hormone and/or adrenocorticotropic hormone. This disorder results in cortisol deficiency only.

Adrenal crisis: is a life-threatening exacerbation of adrenal insufficiency when an increased demand fails to increase hormone production.

Clinical Features: severe hypotension refractory to vasopressors, severe abdominal pain, nausea, and vomiting, confusion,

Consider adrenal crisis in patients with a history of glucocorticoids therapy; those with acquired immunodeficiency syndrome, tuberculosis, autoimmune disease, or severe head trauma; those with a history of chronic fatigue and hyper pigmentation; and those with disorders known to cause acute adrenal crisis.

Treatment

- **Administer IV fluids for hypotension**
- Give steroids: Hydrocortisone 100mg IV/ Dexamethasone 4mg bolus
- Consider vasopressors: if unresponsive to aggressive resuscitation & steroids
- Consider steroid supplementation: lifelong glucocorticoids+_ mineralocorticoid +_ androgen supplementation may be needed.
- Determine underlying cause
- Optimizing maintenance dosage of steroids

Section 6 Renal and Urologic Emergencies

6.1. Acute Kidney Injury (AKI)

Acute kidney injury is the abrupt decrease in renal function resulting in the accumulation of nitrogenous compounds such as urea and creatinine. Increase in Cr by >0.3 mg/dl within 48 hours; or Increase in Cr by >1.5-fold above baseline, which is known or presumed to have occurred within 7 days; or Urine volume <0.5 ml/kg/h for 6 hours.

Etiologies
Pre-renal (55-60%), Post renal (<5%), Renal (35-40%)

Diagnosis: Clinical evaluation and diagnostic work up is essential.

Clinical evaluation
- Oliguria in 50 percent of patients
- Anuria : Can occur in Bilateral cortical necrosis, RPGN, Bilateral renovascular obstruction (arterial /venous), Bladder outlet obstruction, Obstruction in a solitary kidney
- Ask for risk factors:Prerenal and ATN(Acute Tubular Necrosis) have renal hypoperfusion states and / or use of nephrotoxic medications like NSAID , Aminoglycosides
- Glomerulonephritis : nephritic syndrome proteinuria , hematuria edema
- Post renal – prostatism, renal colic, anuria
- Often times it is incidental detected rise in BUN or Creatinine

Various clues favouring Chronic Renal Failure (CRF) instead of AKI
- History : Chronic symptoms- vomiting, nocturia, itching
- Known risk factors : DM, HTN, collagen vascular disease, etc
- Previous abnormal BUN or Creatinine
- BilateralSmallkidney and cortical thinning in ultrasound exam
- Severe unexplained anaemia
- Renal osteodytrophy (20hyperparathyroidism with hyperphosphatemia and hypocalcemia)
- ACUTE CRF -CRF presenting in uremic emergency

NB: prerenal Vs ATN definitive Diagnosis is by response to treatment only
Laboratory Work up
- RFT Creatinine rises after 50 percent of GFR falls
- Urine analysis : protein, sediments
- Ultrasound for kidney size and obstruction
- Electrolytes especially Potassium, Calcium
- Haemoglobin
- Kidney Biopsy
- Work-up in line of considered diagnosis

Treatment

Non-dialytic supportive management

- Care for ABCD as any emergency care
- Fluid overload -Diuresis (furosemide 80-160mg IV stat), limit fluid/salt intake,
- Hyperkalemia (See Hyperkalemia guideline, page----)
- Acidosis- limit protein intake, HCO3 adminstration .Hco3 adminstration (50ml, 50mq, 8.4meq/l in 30 min) is needed only when other mesures do not control acidoses and PH is 7.1.
- Hyponatremia:Restriction of P.O.free water intake, minimization of hypotonic IV dex trose
- Hypokalemia: Calcium carbonate or calcium gluconatecan be given

Dialysis

Indications for dialysis

- UREMIC SYMPTOMS :pericarditis ,neurologic-encephalopathy , bleeding-coagulop athy
- SEVERE FLUID OVERLOAD
- REFRACTORY ELECTROLYTE DISORDERS: hyperkalemia
- SEVERE REFRACTORY ACIDOSIS
- TOXINS----ethylene glycol, methanol, lithium

Specific Etiology Directed

Pre-renal AKI

Correct volume deficit with IV isotonic fluids, severe acute blood loss should be treated with packed red blood cells. Optimization of cardiac function in the cardio renal syndrome (i.e., renal hypoperfusion from poor cardiac output) may require use of inotropic agents, preload- and afterload-reducing agents, and antiarrhythmic drugs

- Post-renal AKI Prompt recognition and relief of urinary tract obstruction
- Transurethral or suprapubic bladder catheterization and Urology expert for possible nephrostomy in ureteric obstruction or other decisions consultation

NB-be aware of post –obstructive diuresis

Intrinsic AKI

- Acute glomerulonephritis/RPGN or vasculitis may respond to immunosuppressive agents like prednisolone 2mg/kg for 6weeks and tapering.
- Allergic interstitial nephritis due to medications requires discontinuation of the offend ing agent
- Rhabdomyolysis-Early and aggressive volume repletion-who may require 1 0 L of fluid per day
- Renal expert consultation

Prevention

• Aminoglycoside/Gentamycin – use when needed and do RFT on initiation and through the course of therapy

• Radiocontrast do prior RFT

Monitoring

• V/S,input/ output,BUN,Creatinine ,Serum electrolyte

Disposition

Table 4. Admission and Discharge Criteria for Renal Cases

ADMISSION CRITERIA	DISCHARGE
1. Electrolyte abnormality	1. Improvement in symptoms
2. Fluid over load and AKI	2. No vital sign derangement
3. Patient require Renal replacement therapy	3. No electrolyte abnormality
4. Correction of underlying cause	4. Improvement in renal function
5. Workup for cause of AKI	5. Appropriate outpatient follow up arranged
6. Poor follow up	

6.2. Testicular pain

Testicular pain refers to pain or discomfort that is felt in one or both testicles.

The pain may originate from the testicle itself, or it may be the result of other conditions affecting the scrotum, groin or abdomen.

Though there are numerous medical conditions that can cause testicular pain, it is important to understand that a few of them constitute medical emergencies that require immediate medical attention in order to prevent impairment or loss of testicular function.

Etiology

Testicular pain can be an acute (short-term) or chronic (long-term) condition. The differential causes of testicular pain

Acute

• Torsion of testis or appendages

• Trauma

• Infection/inflammation:

• epididymo-orchitis

Chronic

- Intra-scrotal tumors
- Systemic diseases:
  - o Idiopathic lymphedema
  - o Henoch-Schonlein purpura
- Hernia
- Idiopathic scrotal edema
- Hydrocele
- Varicocele
  - o In the acute testicular pain our main goal is to detect or exclude a testicular torsion

Approach to a patient with testicular pain

- History
  - – timing of onset: acute or insidious onset
  - – associated symptoms or prior episodes
  - – age at presentation
  - – trauma
  - – fever
  - – sexual activity
- Physical examination
  - – general appearance
  - – observation of the patients gait and resting position
  - – natural position of the testis in the scrotum while standing
  - – presence or absence of cremasteric reflex (this is absent in torsion)
  - – palpation of lower abdomen, inguinal canal and cord
  - – palpation of scrotum and contents, compare with unaffected hemiscrotum
  - – trans illumination
  - – Is the swelling reducible?
  - – lie of testes(to differentiate between torsion and epididymo orchitis), scrotal skin, fluid collection,
  - – testes or epididymis tenderness

Investigation:

- **Urinalysis: bacteria,** WBC's, crystals
  - – commonly in epididymitis
- **Obtain urine culture** (why? If pt. have +ve culture with epidedmytise R/O congenital anomaly by US or MCUG (in pediatrics)
- **CBC may be helpful**
- **Radiographic studies**
  - – Ultrasonography , Nuclear Scan
  - – Doppler US :-helps to differentiate between epidedmytis and torsion , the first we will see high blood supply in the affected site(infection) while in the second decrease blood supply(torsion)

Testicular Pain Management in Adults

- Testicular pain is initially triaged based on history and physical exam as low or high risk

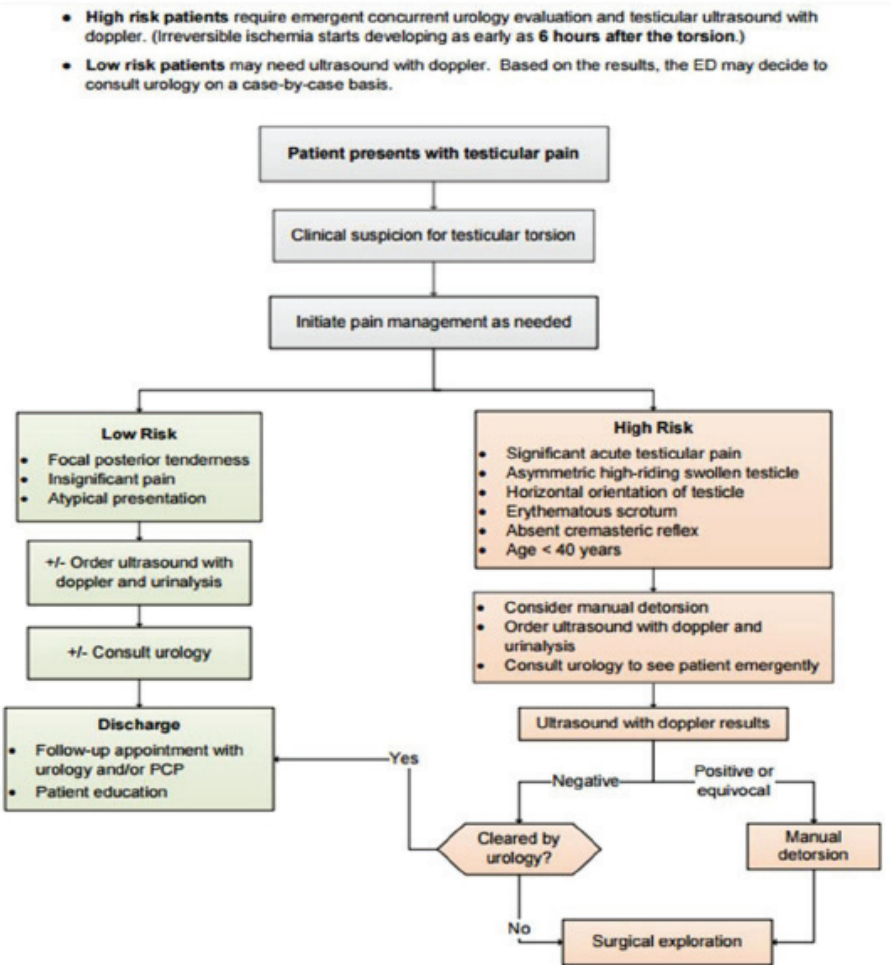


Figure 1. Testicular Pain Management

6.3 Urolithiasis

Urolithiasis is the condition where urinary stones are formed or located anywhere in the urinary system.

- Kidney stones
- Ureteral stones
- Bladder stones
- Urethral stones

Etiology:-

A. Disorders of urinary tract:

- congenital abnormalities those favor to apostasies;
- obstructive processes;
- neurogenic duskiness of the urinary tract;
- inflammative and parasitogenic damages;
- foreign bodies of urinary tract;
- Traumatic injuries.

B. Endocrine diseases

- hyperparathyreoidism;
- hyperthyroidism;
- hypopituitaric diseases

C. Metabolism disorders.

- essential hypercalciuria;
- disorders of membranes for colloid substances diffusion;
- renal rickets

Diagnostic Approach

- History and Physical
- Urine sediment
- Serum Chemistries
- Appropriate imaging
- 24 hour urine stone risk profilets, etc

Treatment

Emergency Renal Colic

- o IV access to allow :
  - Fluid
  - Analgesics:
    - Paracetamol
    - NSAID
    - Opioid
- Antiemetic

o In case of infection:
- Urine culture
- Blood culture accordingly e.g. febrile
- Antibiotics
- The primary indications for surgical treatment include:

– Pain
– Infection
– Obstruction
– Indications for urgent intervention:
– Obstruction complicated by evident infection
– Obstruction complicated by acute renal failure
– Solitary kidney
– Bilateral obstruction

Approach Considerations

o In emergency settings what should be kept in mind is the small percentage suffering renal damage or sepsis.

- These include:

- Evident infection with obstruction
- A solitary functional kidney
- Bilateral ureteral obstruction
- Renal failure

The most morbid and potentially dangerous aspect of stone disease is the combination of urinary tract obstruction and upper urinary tract infection.

- Pyelonephritis
- Pyonephrosis
- Urosepsis

o E**arly recognition and immediate surgical drainage are necessary in these situations**

o **Hospital admission is clearly necessary when any of the following is present:**

- Oral analgesics are insufficient to manage the pain.
- Intractable vomiting
- Ureteral obstruction from a stone occurs in a solitary or transplanted kidney.
- Bilateral ureteral obstruction
- Ureteral obstruction from a stone occurs in the presence of

o a urinary tract infection (UTI)
o Fever
o Sepsis
o Pyonephrosis

Section 7 Hematologic Emergencies

7.1 Anemia

Anemia is a common clinical manifestation that is characterized by reduced concentration of red blood cells (RBCs) from an individual's base line level. Anemia

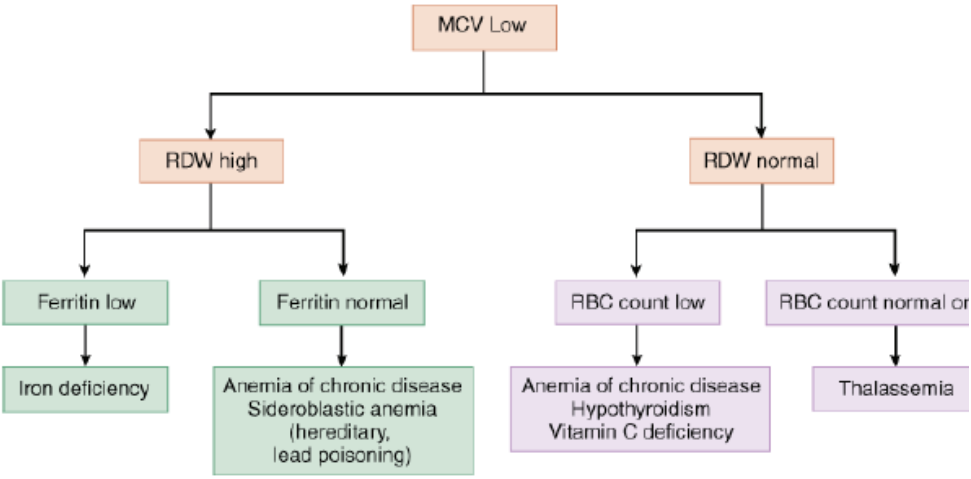
- can be caused by decreased RBC production, increased rate of RBC destruc tion or blood loss.
- signs and symptoms of anemia depend on several factors including the rate o f development, the extent of anemia and underlying medical condition includ ing the age of the patient.

Clinical features

- Clinical manifestations may range from no symptoms to severe hypoxia and acidosis.
- Symptoms may include weakness, fatigue, lethargy, shortness of breath with mild exertion, headache, tinnitus, palpitation, orthostatic symptoms and in severe cases restlessness and altered mental status.
- Signs include tachycardia, pallor, ejection systolic murmur, bounding pulse and wide pulse pressure.
- Jaundice and hepatosplenomegaly suggest hemolysis while unusual skin ulcera tions and peripheral neuropathy suggest nutritional anemia.

Diagnosis

- Is made by finding decreased RBC count, hematocrit and hemoglobin
- Work up for specific cause of anemia should be started in the ED.
- Look and investigate for GI and uterine causes of bleeding
- If no obvious bleeding, look for RBC indices (especially MCV), reticulocyte count (to look for bone marrow response) and peripheral blood smear.
- Consider sending for serum ferritin level if iron deficiency anemia is suspected.

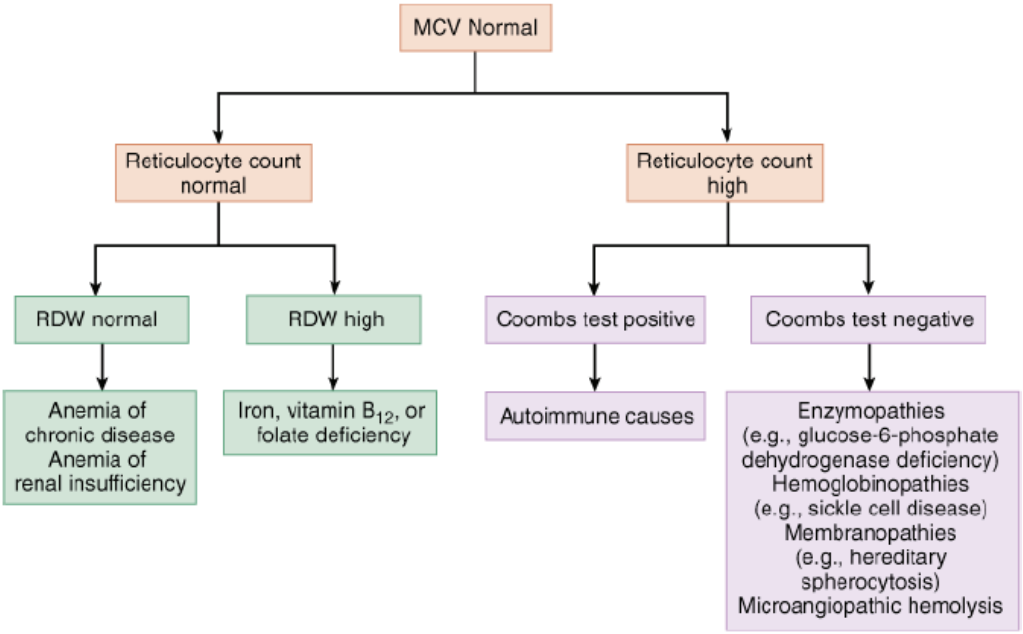


Source: Tintinalli JE, Stapczynski JS, Ma OJ, Cline DM, Cydulka RK, Meckler GD: *Tintinalli's Emergency Medicine: A Comprehensive Study Guide, 7th Edition*: http://www.accessmedicine.com

Evaluation of microcytic anemia. MCV = mean corpuscular volume; RBC = red blood cell; RDW = red distribution width.

Figure 226-3.

Figure 2a. Differential diagnosis of Anemia using MCV



Evaluation of normocytic anemia. MCV = mean corpuscular volume; RDW = red cell distribution width.

Figure 2b. Differential diagnosis of Anemia using MCV

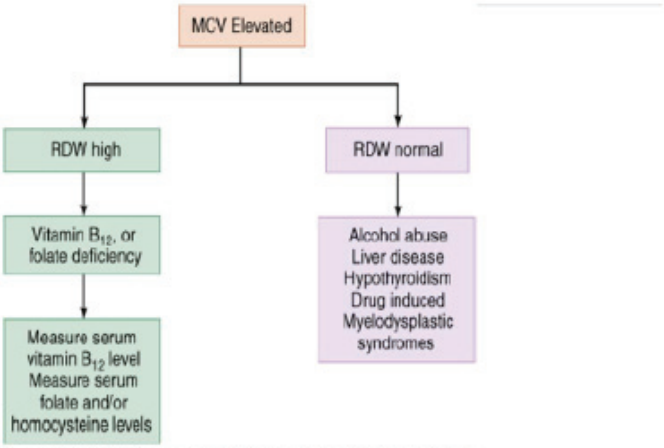


Figure 2c. Differential diagnosis of Anemia using MCV

Treatment

- Put the patient on oxygen if there is any suspicion for hypoxia. Don't rely on pulse oximetry.
- Cross matched blood should be prepared for patients with ongoing blood loss and anemia.
- Transfusion should be individualized based on the patient's clinical condi tion.
- Packed RBC is the recommended blood product for correcting anemia
- Most patients with acute blood loss will require transfusion when the hemoglobin is ≤ 7mg/dl.
- Transfusion for chronic anemia should be considered when patients are hemodynamically unstable, hypoxic, and present with acidosis or have ongo ing is chemic chest pain.
- Consider consulting hematology unit before transfusing a patient with suspected hemolytic anemia.

Disposition

- Those with ongoing bleeding should be kept and managed in the emergency room
- If the cause of anemia is identified, treat the underlying cause
- If other cell lines are also affected, consider medical ward admission
- After stabilization, refer the patient to hematology clinic for follow up

7.2 Acquired bleeding disorders

Normal coagulation is a complex process involving platelets and coagulation factors. Bleeding secondary to platelets disorders usually manifests as mucosal bleeding and pethicae while coagulation problems present with spontaneous and excessive bleeding.

Acquired platelet disorders

Platelet disorders can be qualitative or quantitative. See Table for mechanisms.

Mechanism	Associated Clinical Conditions
Decreased platelet production	Marrow infiltration (tumor or infection)
	Viral infections (rubella, HIV, others)
	Drugs (**Table 228-2**)
	Radiation
	Vitamin B12 and/or folate deficiency
Increased platelet destruction	Idiopathic thrombocytopenic purpura
	Thrombotic thrombocytopenic purpura
	Hemolytic uremic syndrome
	Disseminated intravascular coagulation
	Viral infections (HIV, mumps, varicella, Epstein-Barr virus)
	Drugs (heparin, protamine)
Platelet loss	Excessive hemorrhage
	Hemodialysis, extracorporeal circulation
Splenic sequestration	Sickle cell disease, cirrhosis

Figure 3. Pathophysiology of acquired thrombocytopenia

7.2.1 Thrombocytopenia from decreased production

A number of disease processes can be associated with decreased production of platelets. These may include

- Bone marrow suppression from different disorders affecting all hematologic cell lines
- Drug related thrombocytopenia – Leading to either decreased production or in creased destruction
- Chronic alcohol intake- which usually resolves if a person abstains from alcohol for 7 days

Table 5 Drugs that produce thrombocytopenia or impair normal function of platelets

Produce Thrombocytopenia	Impair Function (prolong bleeding tim
Heparin 4+	Aspirin
Gold salts 4+	NSAIDs
Sulfa-containing antibiotics 4+	Glycoprotein IIb–IIIa agents: ticlopidine an
Quinine and quinidine 4+	
Ethanol (chronic use) 4+	Penicillins and cephalosporins
Aspirin 3+	Calcium channel blockers
Indomethacin 3+	β-Adrenergic blockers: propranolol
Rifampin 2+	Nitroglycerin
Abciximab and eptifibatide 2+	Antihistamines
Thiazides and furosemide 2+	Phenothiazines
Acyclovir 2+	Cyclic antidepressants
Procainamide 2+	
Digoxin 2+	
Cimetidine and ranitidine 2+	
Phenytoin and valproate 1+	
Penicillins/cephalosporins 1+	

Thrombocytopenia from increased platelet destruction

- Can be immune mediated (e.g. ITP) or non-immune mediated.

7.2.2 Idiopathic thrombocytopenic purpura (ITP)

An acquired autoimmune disease that is characterized by production of auto antibodies that lead to platelet destruction by the reticuloendothelial system. It can be acute or chronic.

- Acute ITP usually resolves in a month or two. It commonly affects children and both sexes equally
- Chronic ITP lasts more than 3 months, has a female predilection and rarely remits with treatment or spontaneously.

Clinical features

- Most patients will present with petechiae, epistaxis or gum bleeding
- Menorrhagia is common in women of child bearing age
- Physical examination is usually normal except for the bleeding manifestations

Diagnosis

- CBC usually reveals low platelet count
- Mild anemia with normal RBC indices might be observed in patients with bleed ing
- Peripheral morphology shows few, large, well granulated platelets
- Work up for retroviral infection, hepatitis C virus, H pylori test should be sent depending the patients to look for secondary ITP
- Screening for SLE, Antiphospholid antibody syndrome and others should also be considered.

Treatment

- Assess and manage the ABC of life
- Asymptomatic patients with platelet count >50,000 need no further treatment
- For patients with platelet count between 20,000- 30,000 or less, consider initiating steroids (prednisolone 60-100mg) in consultation with hematology unit
- For patients with very low platelet and bleeding give 1g/Kg/day ofIV immuno globulin for two days or a single dose of Anti D 50micorgram/Kg onlyfor Rh+ve individuals (watch for hemolysis).
- For life threatening bleeding, give methylprednisolone 30mg/Kg/day IV for 3 days with immunoglobulin or anti D and platelet transfusion (preferably given after the first dose of methyl prednisolone or immunoglobulin).
- For patients with treatment failure for steroids, spleenectomy can be considered.

Disposition

- Patients with platelet count less <20,000 or those with mucosal bleedings should be admitted.
- Admit patients when treatment compliance is doubtful or when there are other bleeding risk factors.
- Adults with platelet counts >20,000 and with no symptoms can be managed as an outpatient.

7.2.3 Platelet sequestration

An enlarged spleen can sequester platelets and result in thrombocytopenia. Platelet counts as low as 40,000/mm3 are common. Rarely splenectomy is indicated.

7.2.4 Qualitative platelet disorders

In myeloproliferative disorders, the platelets might be dysfunctional. Patients may develop prolonged bleeding times or significant bleeding. Raising the platelet count up to 50,000 should be considered.

Other conditions may include uremia, liver disease, DIC, dysproteinemias and Von Willebrand disease.

7.3 Acquried coagulation disorders

Acquired coagulation disorders can result from medications, underlying medical disease and autoimmune factor inhibitors.

7.3.1.Liver disease

- Acute or chronic liver disease might be associated with coagulopathies
- The bleeding can be from the combination of thrombocytopenia from poratal hypertension and decreased coagulation factors production from the liver
- It is difficut to differentiate coagulopathy from liver disease from DIC
- Patients can have prolonged coagultion profiles, hypofibrinogenemia and low platelet count might be observed.
- PO or IV vitamine K should be administered for all patients with bleeding and liver disease
- Fresh frozen plasma can be considered for temporary management
- Cryoprecipitate can be considered for patients with low fibrinogen levels (<100mg /dl)

7.3.2. Renal disease

- Is usually secondary to coagultion factors abnormalities or qualitative or quantitative platelet problems.
- Active bleeding is treated with dialysis, transfusion of red blood cells, desmopressin, conjugated steroids and cryoprecipitate
- Platelete transfusion and cryoprecipitate are only resereved for life threatening bleedings.

7.3.3. Disseminated intravascular coagulation

Disseminated intravascular coagulationis an acquired disorder characterized by inappropriate and widespread activation of the coagulation system. There is unregulated thrombin formation system. It can be associated with a number of disorders.

Clinical features

- Depends on the underlying disease leading to disseminated intravascular coagulation
- Bleeding, thrombosis, purpura fulminans and multiorgan failure can be seen
- Mental status change, Oligouria and ARDS might be observed
- Purpura fulminans occurs when there is wide spread arterial and ve nous thrombosis and is most commonly seen with significant bacteremia.

Table 6: Common conditions associated with the development of disseminated intravascular coagulation

Infection Bacterial Viral Fungal	Probably the most common cause of DIC; 10%–20% of patients with Gram-negative sepsis have DIC; endotoxins stimulate monocytes and endothelial cells to express tissue factor; Rocky Mountain spotted fever causes direct endothelial damage; DIC more likely to develop in asplenic patients or cirrhosis; septic patients are more likely to have bleeding than thrombosis.
Carcinoma Adenocarcinoma Lymphoma	Malignant cells may cause endothelial damage and allow the expression of tissue factor as well as other procoagulant materials; most adenocarcinomas tend to have thrombosis (Trousseau syndrome), except prostate cancer tends to have more bleeding; DIC is often chronic and compensated.
Acute leukemia	DIC most common with promyelocytic leukemia; blast cells release procoagulant enzymes, there is excessive release at time of cell lysis (chemotherapy); more likely to have bleeding than thrombosis.
Trauma	DIC especially with brain injury, crush injury, burns, hypothermia, hyperthermia, rhabdomyolysis, fat embolism, hypoxia.
Organ injury Liver disease Pancreatitis	May have chronic compensated DIC; acute DIC may occur in the setting of acute hepatic failure, tissue factor is released from the injured hepatocytes. Pancreatitis can activate the coagulation cascade.
Pregnancy	Placental abruption, amniotic fluid embolus, septic abortion, intrauterine fetal death (can be chronic DIC); can have DIC in *h*emolysis-elevated *l*iver enzymes-*l*ow *p*latelets (HELLP) syndrome.
Vascular disease	Large aortic aneurysms (chronic DIC can become acute at time of surgery), giant hemangiomas, vasculitis, multiple telangiectasias.
Envenomation	DIC can develop with bites of rattlesnakes and other vipers; the venom damages the endothelial cells; bleeding is not as serious as expected from laboratory values.
Acute lung injury or adult respiratory distress syndrome	Microthrombi are deposited in the small pulmonary vessels, the pulmonary capillary endothelium is damaged; 20% of patients with ARDS develop DIC and 20% of patients with DIC develop ARDS.

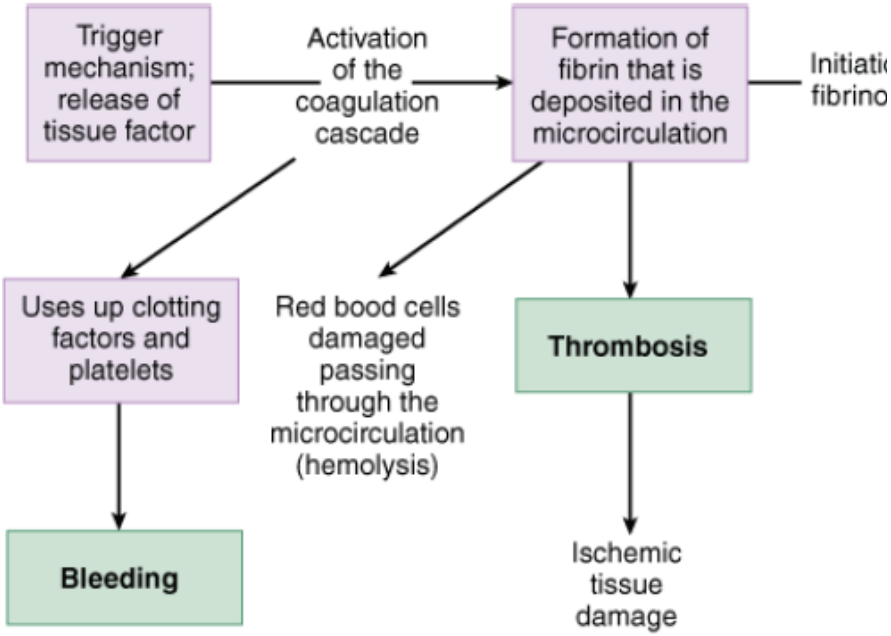


Figure 4. Pathophysiology of Disseminated Intravascular Coagulation

Table 7. Laboratory features for Disseminated Intravascular Coagulation

Studies	Result
Most Useful	
Prothrombin time	Prolonged
Platelet count	Usually low, or dropping
Fibrinogen level	Usually low (fibrinogen is an acute phase reactant, so may actually start out elevated) fibrinogen level <100 milligrams/dL correlates with severe DIC
Helpful	
Activated partial thromboplastin time	Usually prolonged
Thrombin clotting time	Prolonged (not sensitive)
Fragmented red blood cells	Should be present (not specific)
Fibrin degradation products and D-dimer*	Elevated
Specific factor assays	Extrinsic pathway factors are most affected (VII, X, V, and II)

Treatment

- ABC of life and supportive care
- Treat the underlying condition
- If no bleeding or thrombosis, no specific treatment for DIC is needed
- Administer FFP or cryoprecipitate if there is hypofibrinogenimia and bleeding.

Target fibrinogen level should be 100-150mg/dl

- Transfuse with platelets if platelet count is <10,000- 20,000 with out bleeding and when platelet count is <50,000 with bleeding
- Heparin (unfractionated or LMWH) should be considered in patients with DIC and thrombotic complications like purpura fulminans or chronic DIC with thrombosis secondary to solid tumors
- Anti fibrinolytic agents should be used in cation

Dispostion

- Ward admission should be considered

7.4 Blood Product Transfusion

Indications for emergencyPacked Red Blood Cells (PRBC) transfusion are:

- Acute blood loss resulting in shock
- Profound anemia with impaired oxygen delivery
- Unstable trauma patients
- Hgb of 7 g/dL in symptomatic patients

Clinical evaluation includes appearance, mentation, heart rate, blood pressure, and the nature ofthe bleeding (active, controlled, and uncontrolled).

Laboratory evaluation include Hgb, Hct, platelets, and clotting functions.

General Indications for Platelet Transfusion

- Platelet count <10,000/mm3 in asymptomatic patients (unless due to ITP, TTP or HIT)
- Platelet count <15,000/mm3 with a coagulation disorder or minor bleeding
- Platelet count <20,000/mm3 with major bleeding
- Platelet count <50,000/mm3 with an invasive procedure (thoracentesis, paracen tesis) or general surgery required or during massive transfusion
- Platelet count <100,000/mm3 with neurologic or cardiac surgery

General Indications for Fresh Frozen Plasma

- Rapid reversal of warfarin over-anticoagulation
- Bleeding and multiple coagulation defects
- Correction of coagulation defects for which no specific factor is available
- Transfusion of more than one blood volume with active bleeding and coagulopa thy

Table 8. Characteristics of Blood produc

Component	Volume/ml	Dose	Dosage effect
PRBC	250-350	2units or 15ml/kg	1 Hg by 2gm/dl
Platelets	50-60	6units or 5ml/kg	Platelets by 50,000/mm3
Fresh frozen plasma	200-250	4units or 15ml/kg	Coagulation factors by 20%
Cryoprecipitate	20-50	10units or 1units/5kg	Fibrinogen by 75mg/dl

Massive transfusion

Massive transfusion is the replacement of one blood volume or approximately 10 units of PRBCs within a 24-hour period.

Indications for platelet or coagulation factor replacement during massive transfusion

- if the platelet count is <50,000/mm3, give platelet
- if the INR is >1.5, FFP may be given
- if the fibrinogen level is <100 milligrams/dL, it may be replaced with cryoprecipi tate

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Bedside safe transfusion principles

- Do not collect the blood from blood bank before you are ready to trans fuse the patient
- Do not keep a blood for more than 30minutes out of refrigerator
- Inspect the blood consistency and make sure the blood is not hemolized or has big debris
- Make sure the right blood unit for the right patient (check the xmatch paper for expiry date x-match number, group and RH)
- Have new IV line for the transfusion
- Warming of blood is not necessary if you are going to transfuse 1-2units. If you are going to transfuse fast and more blood have appropriate warmer in your unit
- Check and document VS before start of the tra nsfusion
- Start slowly the transfusion especially for the first 15minutes
- Monitor every 5minutes for the first 15minutes to detect transfusion reactions
- If patient is unconscious make sure he /she is catheterized and urine volume and and color is monitored
- When you observe transfusion reaction stop transfusion start normal saline and contact your senior/physician and act according to the reac tion severity and type

Table 9. Transfusion Reactions

Reaction Type	Signs and symptoms	Management	Remark
Acute intravascular hemolytic reaction	Fever, chills, low back pain, dyspnea, tachycardia, shock	ABC of life Stop transfusion monitor input and out put Hydration with dieresis, bronchodilators…	Retype and repeat cross match Direct and indirect comes test CBC, creatinine, PT, aPTT, Urine for hemoglobin
Acute extravascular hemolytic reaction	Less severe symptoms than intravascular hemolytic reaction	ABC of life Stop transfusion Hydration with diuresis	
Febrile transfusion reaction	Fever, chills	Stop tranicfarnsi ABC of life actaminophine	
Allergic reaction	Urticaria, pruritis, dyspnea, bronchospasm, hypotension, tachycardia, shock	Stop transfusion diphenhydramine	
Transfusion-related acute lung injury	Dyspnea, tachycardia, hypoxemia	ABC of life, supportive care	

Other complications include hypocalcaemia, hypokalemia, or hyperkalemia, hypervolemia and infection transmission.

7.5. Febrile neutropenia

Neutropenic fever is defined as a single oral temperature of >38.3°C (101°F) or a temperature of >38.0°C (100.4°F) sustained for >1 hour in patients with severe neutropenia, usually defined as an ANC <500 cells/microL or an ANC that is expected to decrease to <500 cells/microL over the next 48 hours. Neutropenic patients are unable to mount robust inflammatory responses. In such patients, fever is often the only sign of infection.

- The ANC can be calculated by multiplying the total white blood cell (WBC) count by the percentage of polymorphonuclear cells (PMNs) and bands

Risk of serious complications-The initial clinical evaluation focuses on assessing the risk of serious complications.

- Low-risk patients are defined as those who are expected to be neutro penic (absolute neutrophil count [ANC] <500 cells/microL) for ≤7 days and those who have no active comorbidities or evidence of significant hepatic or renal dysfunction.
- High-risk patients are those who are expected to be neutropenic (ANC <500 cells/microL) for >7 days or having ongoing comorbidities like evidence of significant hepatic or renal dysfunction

Clinical presentation

- The clinical presentation of patients with neutropenic fever might be subtle. Pa tients may present with minimal cough with normal chest x-ray findings.
- They may luck pyuria despite having urinary tract infection.
- Oral cavity, perineal area and intravenous insertions sites should be evaluated thoroughly
- Digital rectal examination is a relative contraindication.
- Patients presenting with evidence of new organ dysfunction (altered mental sta tus, hypotension, or hypoxia) should be managed emergently for severe sepsis.

Diagnostic evaluation

- Blood culture at least from two sites should be obtained including central lines
- Urinalysis, urine culture and CXR should be sent
- Sputum, stool and wound drainage gram stain and culture should be done de pending on the patient's presentation

Empiric therapy

- ABC of life and resuscitation
- Broad-spectrum antibacterial should be given as soon as possible (within 60 min utes of triage).
- Antibiotic recommendations for high risk and low risk patients are summarized below.

High risk patients:

First-line monotherapy: This must include an agent with antipseudomonal activity. The following antibiotics are appropriate as monotherapy:

- Piperacillin-tazobactam 4.5 g IV q6h or
- Cefepime 2 g IV q8h or
- Meropenem 1 g IV q8h or
- Imipenem-cilastatin 500 mg IV q6h

Second-line dual therapy: The use of dual therapy in high-risk patients is indicated for complicated cases (hypotension or pneumonia) or suspected or proven antimicrobial resistance. Appropriate antibiotic regimens in this setting include the following:

- Piperacillin-tazobactam 4.5 g IV q6h plus an aminoglycoside or
- Cefepime 2 g IV q8h plus an aminoglycoside or
- Meropenem 1 g IV q8h plus an aminoglycoside or
- Imipenem-cilastatin 500 mg IV q6h plus an aminoglycoside

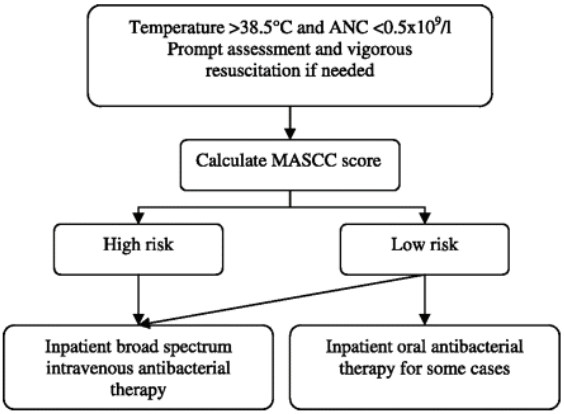
Indications for the empiric addition of vancomycin (15 mg/kg IV q12h) to drug regimens listed above:

- Clinically suspected serious catheter-related infections (e.g, bacteremia, cellulitis)
- Known colonization with penicillin- and cephalosporin-resistant pneumococci or methi cillin-resistant Staphylococcus aureus (MRSA)
- Blood culture positive for gram-positive bacteria
- Hypotension
- Severe mucositis, if prior fluoroquinolone prophylaxis provided

Low-risk patients

Regimens include the following:

- Amoxicillin-clavulanate 500 mg/125 mg PO q8h plus ciprofloxacin 500 mg PO q12h
- Moxifloxacin 400 mg PO daily
- If penicillin allergic, substitute clindamycin 300 mg PO q6h for amoxicillin-clavu lanate



MASCC scoring index

Characteristic	Score
Burden of illness: no or mild symptoms	5
Burden of illness: moderate symptoms	3
Burden of illness: severe symptoms	0
No hypotension (systolic BP >90 mmHg)	5
No chronic obstructive pulmonary disease	4
Solid tumor/lymphoma with no previous fungal infection	4
No dehydration	3
Outpatient status (at onset of fever)	3
Age <60 years	2

Scores ≥21 are at low risk of complications.

Points attributed to the variable 'burden of illness' are not cumulative. The maximum theoretical score is the 26.

Figure 2. Algorithm for management of patients with neutropenic fever

Disposition

- High risk patients should be admitted to the hospital

There is a place for managing low risk patients as an outpatient, which should be decided in consultation with the Hematologist/Oncologist.

Section 8 Oncologic Emergencies

8.1. Malignant spinal cord compression

Up to 20 % of patient with neoplastic involvement of the vertebral column and 3 to 6% of all patients with cancer will develop spinal cord compression.

- Most are secondary to metastasis and few, from primary spinal cord tumors and local spread.
- The most commonly involved area is the thoracic spine
- The most common metastatic cancers are from lung, breast and prostate
- Should be suspected in patients presenting with back pain specially if the patient had history of malignancy

Clinical features

- Up to 90% present with back pain that is usually unrelenting, progressive, worse in supine position and located in the thoracic area.
- Other symptoms include muscular weakness (proximal), sensory changes, impo tence and or, bowel or bladder dysfunction (late signs)

Diagnosis

- Plain x-ray can be used as initial diagnostic imaging. It can detect abnormalities in approximately 80% of patients with painful vertebral metastasis.
- MRI is the imaging modality of choice
- CT scan with or without myelography can be used if there are contraindication for MRI
- The whole spinal column should be scanned because multiple involvements are common. Cervical spine may not be scanned (rare), unless there is strong suspi cion.

Treatment

- Manage pain using WHO pain management ladder or use your pain management protocol
- Bowel bladder care
- Steroids: Dexamethasone 10mg IV followed by 4mg IV or PO every 6 hours
- Communicate with oncology unit for Emergency Radiotherapy (beneficial re sponse in 70% and 90% of those who can walk at the time of treatment, remain ambulatory after wards)
- Surgery

Disposition

- Transfer to oncology unit when possible

8. 2 .Superior vena cava syndrome

Superior vena cava syndrome describes clinical effects of elevated venous pressures in the upper body from obstruction of venous blood flow through the superior venna cava.

Etiologies

- Malignant tumors like lung cancer and lymphoma
- Benign conditions
- Intravascular thrombosis (in patients with indwelling catheters and pacemakers…)

Clinical presentations

- Facial swelling, dyspnea, cough and arm swelling
- Less common symptoms include hoarse voice, syncope, headache and dizziness
- In rare cases there can be signs of increased ICP from the venous congestion like visual changes, dizziness, confusion, seizures and obtundation
- Physical examination may reveal swollen face, plethora and distended chest and neck veins

Diagnosis

- Chest x-ray: to look for mediastinal mass
- CT of the chest with intravascular contrast: the recommended imaging modality
- MRI: if contrast cannot be administered
- Contrast venography: rarely needed, performed when diagnosis is uncertain or for interventional purposes
- Tissue diagnosis

Treatment

- Positioning: Head elevation
- Oxygen: to reduce the work of breathing
- Steroids: proven benefit only in lymphoma
- Diuretics: no evidence for clinical improvement
- Communicate with oncology for possible emergency radiation therapy (improvement in 75%)
- Consider transferring patient for intravascular stenting with or without angioplasty specially for those not responsive to radiotherapy and chemotherapy like mesothilioma, fibrosing mediastinitis and intravascular thrombosis associated with indwelling catheters
- Chemotherapy: specially for lymphoma and small cell lung cancer (80% response) , non-small cell lung cancer (40% response)
- Thrombolytic therapy and anticoagulation with removal of stent : for intra vascular thrombosis
- Surgical graft can be considered

8.3. Tumor lysis syndrome

A metabolic crisis form massive cytolysis and release of intracellular components into systemic circulation

- Commonly seen post treatment for hematologic malignancy, malignancies with rapid cell turn over, bulky tumor mass and high sensitivity to antineoplastic agents
- Rarely seen in solid tumors prior to therapy
- Laboratory abnormalities include hyperuricemia, hyperkalemia, hyperphos phatemia and hypocalcemia
- Renal failure is the most common cause of death in tumor lysis syndrome from precipitation of uric acid crystals in the renal tubules
- Cardiovascular complications of hyperkalemia may result in cardiac arrest

Treatment

- Admit to resuscitation area
- Put on monitor and follow for arrhythmia
- Secure IV line and hydrate aggressively
- Allopurinol should be initiated
- Manage hyperkalemia with beta two agonists, insulin and dextrose, sodium bicar bonate and Sodium potassium binding resins
- Avoid CALCIUM administration for the management of hyperkalemia unless there is cardiovascular instability or neuromuscular irritability like seizures. It can cause metastatic precipitation of calcium phosphate
- Hyperphosphatemia is managed with phosphate binding resins or with insulin and dextrose
- Hemodialysis should be considered for severe case

Disposition

- Consider admission to the wards or ICU depending on the severity of symptoms

8.4. Hyper leukocytosis syndrome

Total leukocyte blood cell count greater than100,000 micro

- Leukostasis is a medical emergency most commonly seen in patients with AML or CML with blast crisis characterized by symptoms of decreased tissue perfusion due to white cell plug in the microvasculature.
- Diagnosed empirically when a patient with leukemia and hyper leukocytosis pre sents with respiratory or neurological distress.

Clinical presentation- dyspnea, hypoxia, visual changes, headache, dizziness, tinnitus, gait instability, confusion, somnolence, coma

Treatment

- Adequate fluid resuscitation—4-5L N/S per day
- Cytoreduction
  - o Hydroxyurea given at a total dose of 50- 100mg/kg per day orally
  - o Induction chemotherapy
  - o Allopurinol 100mg P.O. tid per day
- Monitor urine output , renal function test, CBC after 24hrs of the hydroxyuria

Disposition --- admission is required

Section 9 Infectious Diseases

9.1. Infectious Diseases

Infectious disorders are common in the emergency care and high index of suspicion is needed to have etiologic diagnoses. If there is no prompt diagnosis, lethal but treatable febrile illnesses could be missed like severe malaria, meningitis, relapsing fever...etc. Furthermore, early diagnoses of localized infection or internal abscess detection are very important for early antibiotics treatment, early drainage & surgical consultations.

Through appropriate evaluation of a febrile patientlife threatening causes of fever in the emergency department (ED) will be categorized into:

1. Non-localized source like acute febrile illness (AFI)or sepsis
2. Localized infections: peritonitis, meningitis, pneumonia, pyelonephritis, etc.

9.2. Approach to a Febrile Patients in the Emergency department

History:

a) The nature of fever and associated features: a febrile patient may present as non-specific symptoms like headache, joint pain, chills, rigor, sweating, and anorexia and flu-like symptoms. A patient may have change in sensorium or coma as in malaria, meningitis, relapsing fever, sepsis,and encephalopathy.

b) Clues for etiology search:History of travel, similar family history, any localizing symptoms, underlying disease, medications, transfusions, animal exposures, etc.

c) Localizing features and complications: like pulmonary, CNS, GI, etc features

Physical examination: Assessthe vital signs and thorough systemic evaluation is worth while.

HEENT and lymphoglandular (anemia, jaundice, bleeding tendencies and lymphadenopathies), Respiratory system(tachypnea, crepitations, signs of pleural effusion),CVS (for new murmers,features of myo/pericarditis), GIT (abdominal swelling, guarding, tenderness, rebound tenderness, perianal area assessment, abdominal fluid tap if present),GUS (Cost vertebral angle and suprapubic tenderness), Musculo Skeletal system(for joint / bone swelling, spine tenderness),dermatology(phlebitis, cellulites, rash), CNS(nuchal rigidity, mental status tests).

Diagnostic Workup:

- CBC with ESR including differential: for leukocytosis and neutropenia
- Culture &sensitivity: blood, urine, CSF, discharge-Sputum Gram stain, AFB-Blood chemistries
- Blood film: for hemoparasites (malaria, Borrelia, ;eishmaniasis)
- Urinalysis: for WBCs,hematuria,casts : for UTI/Pyelonephritis, Stool examination: for ova or parasites , CSF analysis for cell count, organisms (Gram stain, AFB, Indian ink)
- Hepatitis serology (if LFTs abnormal) ,HIV test
- CXR, abdominal ultrasound(if indicated)

Diagnostic procedures might be needed to get sample: Lumbar puncture: for suspected meningitis, Aspiration & drainage of infected collections / abscesses

9.3. Sepsis

Sepsis is considered present if infection is highly suspected or proven and two or more of the following systemic inflammatory response syndrome (SIRS) criteria are met:

- Heart rate > 90 beats per minute (tachycardia)
- Body temperature < 36 °C (96.8 °F) or > 38 °C (100.4 °F) (hypothermia or fever)
- Respiratory rate > 20 breaths per minute or, on blood gas, a PaCO2 less than 32 mm Hg (tachypnea or hypocapnia due to hyperventilation)
- White blood cell count < 4000 cells/mm^3 or > 12000 cells/mm^3 (< 4 x 109 or > 12 x 109 cells/L)

Stage of Sepsis Syndrome and its complications

- Bacteremia: is presence of viable bacteria in the blood.
- Sepsis (Septicemia): is clinical evidence of infection accompanied by systemic response such as tachypnea, tachycardia, fever, leukocytosis etc.
- Severe Sepsis: Organ dysfunction secondary to Sepsis.e.g. hypoperfusion, hypotension, acute lung injury
- Septic Shock::Hypotension secondary to Sepsis that is resistant to adequate fluid administration
- Refractory septic shock-Resistant to ressors
- Multiple end organ damage: is involvement two or more organs by sepsis.

Clinical aspects

In the early fever, chills are common but patients in the extremes of age can be hypothermic. Generalized weakness is common and the skin is warm. The other features are related with the site of infection when it is identified and specific complications like DIC.On physical examination, hypotension, tachycardia, and tachypnea, high or low temperature are important clues.

Investigations

Depending on the scenario and level of complication the following tests can be done:
Laboratory tests: Full blood count, Blood cultures (before antibiotics are given), Tissues / fluid / pus culture,
U/A andculture, Renal and liver functions, Blood film, Lumbar puncture and CSG analysis, Disseminated intravascular coagulation (DIC) screen
Imaging: CXR, abdominal X-ray, ultrasound, CT scan

Initial Resuscitation of Sepsis

- **Diagnosis of sepsis:** Obtain appropriate cultures (e.g 2 or more sets of blood cultures, tissues, pus or other sites as clinically indicated) before starting antibiotics provided this does not significantly delay antibiotic administration.
- **Fluid Therapy**- Open two large bore IV lines fluid resuscitation with colloids or crystalloids. Target a CVP of > 8mmHg (> 12mmHg if mechanically ventilated) whenever CVP measurement is not possible monitor the urine output and make sure the volume is >0.5ml/min, pulse rate is decreasing, mentation improving and auscultate the chest for early detection of fluid overload
- **Vasopressor –** Maintain mean arterial pressure (MAP) > 65mmHg or systolic pressure of more than 90 mmHg. Dopamine or norepinephrine is the initial vasopressor of choice. Doses of dopamine often required are 0.5–25µg/kg per minute. If the response is inadequate, norepinephrine at dose of 0.01–0.5 µg /kg per minute should be started. Doses are gradually titrated according the response of the patient. Once the blood pressure and perfusion have been stabilized, always use the lowest dosage that maintains blood pressure in order to minimize the complications of vasoconstriction. (In ideal situation monitoring needs Inserting an arterial catheter following intrarterial pressure)
- **Steroid:** consider low dose hydrocortisone (100 mg IV tid) for adult septic shock when hypotension remains poorly responsive to adequate fluid resuscitation.
- **Antibiotic therapy:** Begin antibiotic as early as possible (within one hour of recognizing sepsis). Antimicrobial therapy is often an empiric choice and generally broad spectrum antibiotics (one or more agents) are used.
- **Source identification and control:** A specific anatomic site of infection should be established as rapidly as possible and control measures (e.g abscess drainage, debridement, and removal of intravascular access device if potentially infected) as soon as possible following successful initial resuscitation.

(Adapted from Surviving Sepsis Campaign: International guidelines for management of sepsis and septic shock Intensive Care Med (2008) 34:17-60)

Other Supportive Therapies –In special conditions with complications consider:

- Respiratory support - maintain oxygen saturations above 90 %. The rough oxygen with face mask or intubation and mechanical ventilation is often needed in almost all patients with ARDS.
- Monitoring for acute renal injury-it can complicate patients with severe sepsis due to hypoperfusion and hypotension. Normally urine output should be of greater than 30 ml per hour (0.5ml/kg/hr.). Insert urinary catheter to monitor urine output.
- Glucose control - hyperglycaemia is common in sepsis. Blood glucose must be monitored. Continuous insulin infusion may be necessary to maintain target blood glucose levels.
- Sedation and analgesia – Use sedation protocols with a sedation goal for critically

ill mechanically ventilated patients.

- Deep vein thrombosis (DVT) prophylaxis – use either low dose unfractionated heparin or low molecular weight heparin (LMWH).
- Stress ulcer prophylaxis with proton pump inhibitors (PPI) agent such as omepra zole,
- Blood products – Give red blood cells when hemoglobin is < 7.0 g/dL, to target hemoglobin of 7.0-9.0 g/dL in adults.
- Replace deficient haemostatic factors in decompensated disseminated intravascu lar coagulation (DIC) using cryoprecipitate, fresh frozen plasma or platelets. A re ferral to a haematologist may be necessary as both anticoagulants and factors re placement therapy is potentially dangerous and should be used with caution.

9.4. Malaria

Malaria is a parasitic infection causing acute febrile infection with Ethiopia being one of hyperendemic areas in the world. There are four main species known to affect humans, namelyP.falciparium, P.vivax, P.ovale, and P.malariae. The most serious and life-threatening disease occurs from Plasmodium falciparum infection.

Diagnosis can be co confirmed by demonstration of malaria parasites in the blood to estimate the degree of parasitemia, which is extremely useful not only to predicate severity but follow response to treatments.

Clinical features of severe malaria.

Withsevere and complicated malaria, in addition to the diagnoses of malaria, it is important to diagnose and monitor the presence of one or more of the following conditions:

- Cerebral malaria with impairment of consciousness and/or seizures
- Hematologic complications: severe anemia, spontaneous bleeding, DIC
- Acute renal failure,
- Pulmonary edema, or adult respiratory distress syndrome,
- Metabolic: Hypoglycemia, Metabolic acidosis
- Circulatory collapse or shock,
- Macroscopic hemoglobinuria or black water fever

Treatment of severe and complicated malaria

Non – drug treatment and monitoring:

- Clear and maintain the airway and position semi – prone or on side.
- Make rapid clinical assessment for hypoglycemia, dehydration and shock.
- Measure and monitor urine output, if necessary insert urethral catheter.
- Take blood for diagnostic smear, monitoring of blood sugar & HCT
- Plan first 8h of intravenous fluids including diluents for anti-malarial drug, glu cose therapy and blood transfusion.
- Consider other infections and if proved treat concurrently.
- Consider need for anti-convulsing treatment in seizures.

Drug Treatment:
Currently the management of severe P.falciparium malaria is done with either by quinine or artesunate family drugs.

Quinine: Loading Dose: 20 mg/kg in 500 ml (10ml/kg) of isotonic saline or 5% dextrose over 3-4 hours. Maintenance dose: should be given 8 hours after the loading dose -10 mg/kg to be given 8 hourly diluted in 500 ml of isotonic saline or 5% dextrose over 3-4 hours and Po dose Quinine 10mg/kg every 8hrs When patient tolerates N.B. If IV infusion is not possible, quinine can be given IM.

Artesunate Family drugs
Artesunate 3:2 mg/kg (loading dose) IM, followed by 1.2 mg/kg at 12 and 24 hours, then 1.2 mg/kg daily for 6 days. If the patient is able to swallow, the daily dose can be given orally.
Or
Artemether: 3.2 mg/kg (loading dose) IM, followed by 1.6mg/kg daily for 6 days. If thepatient is able to swallow, the daily dose can be given orally.

Suppository Forms
If parenteral administration is not possible suppositories may be given.

Artemisininsuppositories: 40 mg/kg (loading dose), then 20 mg/kg 24,48 and 72 hours later, followed by an oral antimalarial drug. Or

Artesunate suppositories: 200 mg at 0, 12,24,36,48 and 60 hours.
Coartem 3 tabs two times daily for 3 days.

Chloroquine- resistant malaria or sensitivity not known
Quinine (adults20 mg dihydrochloride salt/kg of body weight (loading dose) diluted in 10 ml isotonic fluid /kg by IV infusion over 4 hours: then 8 hours after the start of the loading dose, give a maintenance dose of quinine, 10 mg salt/ kg, over 4 hours.

This maintenance dose should be repeated every 8 hours, calculated from the beginning of the previous infusion, until the patent can swallow, then quinine tablets. 10 mg salt/kg. 8-hourly to complete a 7-day course of treatment, or a single dose of 25 mg/kg sulfadoxine and 1.25 mg/kg pyrimethamine (maximum 1500 mg sulfadoxine- 75 mg pyrimethamine).

9.5. Relapsing fever

Relapsing fever is an acute febrile illness found in Ethiopia. It is caused by Borrelia recurrentis and is transmitted to through human body lice. It presents with abrupt onset of severe headache and fever with chills, arthralgia myalgia, etc. There are notably two phases: a chill phase, characterized by rigors, rising temperature, and hypermetabolism, and a flush phase of falling temperature, diaphoresis, and a decreased effective circulating blood volume.A diagnosis is by BF showing Borrelia spp., which spiral organism.

Treatment

- Antibiotics such as- Doxycycline 100mg ,TTC 500mg,Erythromycin 500mg or Procaine penicillin(PPC) 600,000 IU IM (all single dose).The advantage of PPC is the destruction of the organism might be delayed Jerisch, Herx, Hemir (JHH) reaction could be milder, whileit may not destroy the whole organisms. Hence after PPC one may give other PO single dose agents.
- Antibiotics administration, can cause release of cell wall proteins and serious endotoxic shock which is called ,characterized by chills/rigors phase and a flush phase characterized by diaphoresis and can be complicated by hypotension.
- If JHH reaction occurs and causes shock follow septic shock management guideline (isotonicsaline, using inotropes / vasopressors such as dopamine). Fever can be controlled by the use of paracetamol or a cooling blanket and ice packs and by sponging of the patient with tepid water and alcohol in severe condition.
- Management of Complications: if it is complicated thorough monitoring and ICU admission.
    - o Bleeding abnormalities: manifest with petechial rush, ecchymosis, subconjunctivalhemorrhage, jaundice: platelet transfusion, FFP, Vitamin K can be given.
    - o Myocarditis and heart dysfunction: includes a third heart sound (S3), prolonged QT, interval, elevated central venous pressure, arterial hypotension, and pulmonary congestion with crepitation on auscultation of the lung -digoxin can be given to slow the heart rate.

9. 6. Typhus

- Typhus is a rickettsia disease which causes an acute febrile illness characterized by an abrupt onset of high grade fever, severe headache and prostration.
- The human body louse (Pedicels humanus corporis) lives in clothing and is most commonly present in areas that are cold, impoverished and have poor hygienic conditions, Lice acquire R. prowazekii when they ingest blood from a rickettsemic patient. Brill-Zinsser disease is a recrudescent illness occurring years after acute epidemic typhus, probably as a result of waning immunity.

- Clinical Feature: After an incubation period of 1 week, the onset is abrupt, with prostration, severe headache, and fever rising rapidly to 38.8°–40.0°C with a prominent cough in most patients. There is notably severe myalgia (Back breaking).A rash begins on the upper trunk on the 5th day in untreated people with pale skin, and then becomes generalized, involving the entire body except the face, palms, and soles. Initially, this rash is macular; it becomes maculopapular, petechial, and confluent. The complications like CNS involvement and confusion, coma, encephalopathy and vasculitis with gangrene, skinnecrosis, and neurological deficits can be seen.
- Diagnoses: there is no commercial test to diagnose typhus, and hence high index of suspicion based on the clinical scenario and the type of host which from underprivileged living condition like poverty ,overcrowding ,prison,etc.
- Treatment: Doxycycline 100mg P.O.bid for 5-7 days or Tetracycline, 250mg, qid P.O. for 5-7 days.

9.7. Typhoid Fever

- Typhoid Fever is an acute febrile illness caused mainly by Salmonella typhi and the mode of transmission is via contaminated food or water.
- It is clinically characterized by a gradual increase in body temperature associated with headache, malaise and chills and symptoms are usually non-specific.

Diagnosis: it depends mainly on clinical features which can be supported.

- Confirmed by positive culture of blood, Bone marrow, stool or urine for the bacillus.
- Serological examination, such as the Widal test less commonly indicated as validity of the test has been very low so clinical suspicion is very important.

Complications: Bacteremia with septic shock, Intestinalhemorrhage, andIntestinal perforation

Treatment

- Symptomatic Treatment like using antipyretics, e.g. paracetamol to control fever
- Drug treatment
  - Ciprofloxacin, 500mg P.O., twice daily for 7 days (IV in critical pts),or azithromycin 1gm po stat and then 500mg po/d for 6 days, or Ceftriaxone, 1g daily as a single dose or 2 divided doses i.m. or iv for 5-7 days. OR
  - For severe cases: admit at ER or wards and open IV line and then Start IV medications every 6 hrs, until 48 hrs after fever has settled followed by p.o. rugs.

Localized Infectious Problems/Localized Fever

The following section deals problems which affect specific organ or system of the body which identified by the clinical feature .

9.8. Pyogenic Meningitis

- Pyogenic meningitis is inflammation of meninges, the outer cover of the brain due to infection by organisms such as bacteria, viruses, mycobacterium tuberculosis fungus, etc. Ethiopia is found in the sub- Saharan meningococcal meningitis belt where this infection is endemic and epidemic is happening cyclically. Meningitis can cause brain damage and as a result prompt treatment is needed.
- Clinical features – fever, neck stiffness, headache, altered status of consciousness, nausea and vomiting, nuchal rigidity. If there is meningococcal sepsis (fulminant Meningococcemia) the course will be very rapid manifesting with shock, petechial rash, lung injury and hypoxemia, disseminated intravascular hemolysis, etc, which evens kills patients within 12 hours.
- Laboratory diagnoses: WBC and deferential and do lumbar puncture. Whenthere are signs of ICP rise like altered state of consciousness,papilledema or focal neuro logical deficit LP can be after CT scan – CSF analysis: CSF is turbid, and rise in the number of WBC and protein while glucose level gets lower. In addition, CSF gra stain shows causative organisms and culture may be positive.
- Complications – Brain abscess, hydrocephalus, raised intracranial pressure, deaf ness, etc

Treatment of bacterial meningitis
Supportive Measures: regular monitoring of vital signs and neurological state.

Drug treatment
A) Community acquired infection (when bacterial etiology unknown)
1. Ceftriaxone, 2g IV every 12 hourly for 10 days(add vancomycin when skin is breached like in head injury patients).Crystalline penicillin.20 – 24 IU.day i.v. in 4-6 divided doses for 7 – 10 days is an is an alternative choice for ceftraxone

2. Elderly patients with Meningitis (L.Monocytogenes is possibility)-Ceftraxone and Ampicillin

3. In immunocompromised host and hospital acquired infections-consider pseudomonas and ceftazidime (pseudomonas coverage) or/and carbapenemis (in critical patients).

N.B. Treatment of Complication

1. When there is significant rise in ICP and or hydrocephalus apply ICP reduction methods (ex. Mannitol)

2. Sepsis and septic shock (See guideline for severe sepsis and septic shock)

3. Brain abscess-long term antibiotics-for 6-8weeks

4. Seizure should be relieved by immediate benzodiazepines and controlled by long acting ones (ex Phenytoin)

- Third generation cephalosprines are effective against N.Meningidiits,Pneumoc and H.Influenzae spp but hospitals' susceptibility pattern has to be regul checked.
- IV Dexamethasone is administered with the first dose of empiric antibiotics w there is moderate to severe alteration in mental status, focal neurologic defici papiledema (there are proven evidences of advantage especially in pneumoco meningitis in adults)

9.9. Urinary Tract Infection (UTI) - Pyelonephritis, cystitis, urethritis

- It is inflammation of the urinary tract, which includes the renal parenchyma (pyelonephritis), the bladder (cystitis), the prostate in males (prostatitis) and the urethra (urethritis).
- *Escherichia coli cause approxi*mately 75-80 % of acute infections in patients without catheters, stone or other urologic abnormalities. With urologic abnormality, manipulations or hospital origin organisms likeklebsiella, enterobacteria,proteus, serratia and pseudomonasare more prevalent.
- Usually complication of structural defects, obstruction or instrumentation such as Cystoscopy or inserting urinary catheters. It is characterized by fever, flank pain, costovertebral angle tenderness, dysuria, frequency and urgency of micturition in bladder & urethral involvement. The range of symptoms caused by UTI from asymptomatic to symptoms referable to the lower urinary tract (e.g. dysuria and frequency), to symptoms indicative of an upper UTI (e.g. loin pain and costo-vertebral angle tenderness), to full-blown septic shock. The majority of acute symptomatic infections occur in young women but lesser in young males and asymptomatic bacteriuria is very common in elderly men

and women.

- Complications: Sepsis and septic shock, abscess, stone formation
- Diagnosis: clinical and laboratory evidences. In complicated and /or recurrent UTI imaging study, by ultrasound is necessary
  - o U/A: urine has to be examined in the morning and the sample has to be clean catch, i.e. midstream. Abnormal findings with many WBC, WBC casts, RBCcan be seen while urine culture shows growth organisms.
  - o Gram stain of the urine showing pyuria and bacteriuria
  - o Urine culture and sensitivity (specimen should be sent before antibiotics initia tion). Bacterial colony count of 105organisms per milliliter or greater in urine in dicates urinary tract infection.

Treatment

A**) UTI in women**

Acute, Uncomplicated Upper UTI (Pyelonephritis) in women:

- Quinolones (Norfloxacin, 400mg ciprofloxacin 500mg P.O. BID) or Sulfamethoxazole+trimethoprim, 800 mg/160 mg P.O. BID, Amoxicillin, 250-500 mg P.O. TID for 7-10 days.
- In severe cases, patients should be admitted and antibiotics should be given paren tally until fever gets down for the first 48-72 hours. In such conditions Third genera tion cephalosporines like ceftraxone 1gm IV Bid can be given.
- Besides aminoglycosides like Gentamycin can be combined in severe infections when renal function is normal

Acute, Uncomplicatedlower UTI in women:

- Quinolones (Norfloxacin, 400mg ciprofloxacin 500mg P.O. BID) or Sulfamethoxazole+trimethoprim, 800 mg/160 mg P.O. BID. Amoxicillin, 250-500 mg P.O. TID for 3-5days.

B) UTI in Men:

Quinolones (Norfloxacin, 400 mg P.O. BID or ciprofloxacin 500mg P.O. BID), for 10-14 days or **Sulfamethoxazole+trimethoprim,** 800 mg/160 mg P.O. BID, for 10-14 days. Amoxicillin, 250-500 mg P.O. TID for 10-14 days.

C) For children: Amoxicilin 20-40mg/kg/day in 4 divided doses.

D) For recurrent and resistant cases of UTI-refer for urologic and nephrologic evaluation. Structural lesions should be ruled out and antibiotics based on C/S.

9.10. Peritonitis

- It is infection and inflammation of the peritoneum and is categorized into primary and secondary.
- Primary peritonitis is found without clear gastrointestinal risk factor while sec ondary peritonitis occurs due to complication of acute abdomen such as appendi citis, salpingitis, cholecystitis, perforated discus like typhoid fever, etc. It is usually a mixed infection and the gram negative Bacilli(E.Coli,klebsilea,pseudomonas,pro teus,etc) ,gram positive Coci like pneumococcus and gastrointestinal anaerobes like bacteroides fragilis are the most important organisms involved.
- Clinical features – Fever, abdominal pain, guarding, tenderness and rebound ten derness.
- Complications – localized or generalized abscess, sepsis /septic shock
- Diagnosis is clinical with underlying conditions mostly are clear.

Treatment-

- Make patient NPO
- Open two large bore IV lines and start resuscitation as needed
- Antibiotics with coverage of Gram positives, negatives and anaerobes
- Input output
- Insert NGT
- Determine hemoglobin, Rh, CBC, urine analysis, RFT if patient has vomiting de termine electrolyte
- Consult surgical side

Enteric Infections

Toxigenic diarrhea:

- Exotoxins extracted from microorganisms like staphylocolus aureus stimulate hyperse cretion of intestines and as a result cause nausea and vomiting with in 6h-12hrs. of ex posure to contaminated food, but if toxin is formed in the intestine it takes time to produce symptoms.
- Stabilizing the patient with fluid replacement and symptomatic therapy is adequate.

Infectious causes

Etiology

1. Bacterias

Shigella, Salmonella, Campylobacter

Cause invasive infections and so cause fever and diarrhea with fecal WBC & RBC

Salmonella typhi can cause generalized acute febrile illness in addition to Local symptoms.

2. Protozoans –

Amoeba and Giardia commonly cause watery diarrhea but invasive Amoebiasis can cause dysentery (fever, fecal leukocytosis)

3. Viruses – Rota, Norwalk

Treatment

General treatment

Fluid replacement:

In mild to moderate ones oral hydration is enough.In severe case IV fluid hydration (N/S, R/L)

In severe gastroentestes such as in cholera the loss may be very high that need many liters, the first liter in 20 minutes.

9.11. Tetanus

It is a neurologic syndrome cause by a neurotoxin elaborated by Clostridium tetani at the site of injury. It can largely be prevented by appropriate immunization, but as a result of inadequate coverage we still have cases of tetanus in our country.

The most common and important clinical features include trismus (lockjaw) localized or generalized muscular rigidity and spasms. The presence of arrhythmia, extreme fluctuation in blood pressure, diaphoresis, laryngeal spasm and urinary retention suggest autonomic dysfunction occurrence as a complication.

Diagnosis is usually established on clinical grounds and severity has to be graded.
It is mild if there is no spasms but only rigidity, moderate if there are few spasms, and severe if there are frequent spasms and has autonomic dysfunctions.

Table 10. Assessment of severity in adults with tetanus

Severity score	Symptoms/signs
Mild	Mild/moderate trismus, spasticity, no respiratory embarrassment
Moderate	Moderate trismus, rigidity, mild to moderate spasms
Severe	Severe generalized spasticity, prolonged spasms, respiratory embarrassment
Very severe	As above, plus autonomic dysfunction, labile hypertension, tachyarrhythmia, bradycardia, sweating

Treatment

General care:

- Admit patients to a quiet place, and in severe cases, to ICU if possible for a con tinuous cardio-pulmonary monitoring.
- Assess for ABC and intervene accordingly: airway care including Intubations or tracheotomy could be need in severe cases.
- Wound care which includes thorough cleansing and debridement
- Attention should be given to adequate fluid therapy. Nutrition should be given through NG tube feeding, or parenteral feeding.

Drug treatment

A. Control of Spasm-

Diazepam 5-10 mg IV q4- 6h, alternating with chlorpromazine 25-50 mg IV q4- 6h is given to control spasms and reduce respiratory complications. Large doses as much as 250 mg a day could be used.

In uncontrolled spasms to the above drugs give magnesium sulphate 2gm IV push.

B. Antibiotic Treatment:

Metronidazole, 500n mg p.o. or IV tid for 7-10 days or crystalline penicillin

C. Neuromuscular blockade

Suxamethonium, 20-100 mg i.v. depending on the effect with mechanical ventilation may be employed in patients with severe laryngeal spasm. But remember if you para lyze the patient with suxamethonium or other drugs, patient is completely dependent on the machine functions and during any malfunctioning of the machine or electrical interruption patient is at risk of arrest due to apnea caused by the drug, so the ICU staff has to be aware and ready to bag with ambubag in such situations

D. **Neutralize toxin by Tetanus immunoglobulins**

Tetanus anti toxin shortens the duration of illness and may reduce severity. Admin ister tetanus anti toxin 5,000 - 10,000 IU IV after skin test. Equine anti toxin may be complicated by severe allergic reactions. Hyper immune human tetanus immunoglob ulin 5000-10,000 IU is preferable if available/affordable.

E. C**ontrol of Autonomic Dysfunction:** labile hypertension and supra-ventricular tachy cardia can be treated with Beta-blockers like IV labetalol 0.25-1 mg/min or IV pro pranolol 1mg may be used cautiously, or Propranolol, 60 mg P.O. daily in three-divided dose

F. **Active immunization:** All patients recovering from tetanus should be given tetanus toxoid, two doses, 0.5 ml IM, on discharge.

G. **Reduce respiratory complications**

In patients with severe tetanus and respiratory embarrassment, tracheostomy should be performed electively before development of severe laryngeal spasm paralysis of patients and mechanical ventilation significantly improves outcomes in severe cases.

Prevention/treatment of other complication

- Secondary bacterial pneumonia, urinary tract infections may compli cate the course of tetanus, and should be treated accordingly when they occur.
- Thromboembolic complications due to prolonged immobilization may require heparin therapy.
- In rare cases rhabdomyolysis and vertebral compression fracture may occur.

Common Opportunistic Infections associated with HIV in the Emergency Room

9.12. Toxoplasmosis (CNS)

CNS Toxoplasmosis is an infection of the central nervous system by the protozoan Toxoplasma gondii. The disease develops in individuals with underlying immunodeficiency, usually occurring as a reactivation. Patients with cerebral toxoplasmosis typically present with headache confusion, fever and/or signs of focal neurological deficit.

Diagnosis:

- Clinical and Neuro-imaging (CT scan or MRI of the brain).
- Serologic test for anti-toxo Ig-G antibody. If negative, it may help to exclude the diagnosis. On the other hand positive test or high titer for Ig-M would suggest a more recent infection.With empirical treatment for toxoplasmosis, nearly 90% of patients will demonstrate Clinical improvement within days of starting therapy. Radiological evidence of improvement is usual after 14 days of treatment.

Treatment: Initial therapy of choice consists of a combination of pyrimethamine, sulfadiazine and leucovorin. Since these drugs are not available and not usedin Ethiopia,

1. Sulfadoxine/pyrimethamine (Fansidar): 500 mg/ 25 mg po b.i.d for two days, followed by once daily both for four (4) weeks is given together with Folic acid (10 mg daily)

2. Cotrimoxazole -15 mg /Kg Trimethoprim in three divided doses daily when Fansidar cannot be administered safely is alternative regimen in Ethiopia .

3. Corticosteroids (dexamethasone 4mg PO or IV q6hrs) is used if cerebral oedema present, but should be discontinued as soon as clinically feasible.

9.13. Pneumocystis Carinii Pneumonia

Pneumocystis carinii pneumonia (PCP) frequently causes pneumonia among immuno-compromised individuals. It is caused by the fungus pneumocystis caranii(jiroveci). The onset is typically subacute over 2 to 4 weeks, with prominent symptoms of low grade fever, non-productive cough, progressive dyspnoea exacerbated by exertion, and fatigue. Patients will have an increasing tachypnea, tachycardia and cyanosis as the disease progresses.

Diagnosis: Presumptive diagnosis of PCP is based on clinical judgement and typical chest X-ray findings revealing a perihilar interstitial infiltrationwith tendency to spread Outwards. Chest X-ray can be normal in 20% of patients. Definitivediagnosis of PCP is based on demonstration of the organism from an induced sputum Sample using special stains like Giemsa or methylamine silver stains, but not routinely done here.

Treatment

Supportive treatment:

In moderate and severe cases oxygen throughappropriatemeans should be given, in critical patients with respiratory failure assisted ventilation and oxygen through ventilators might be needed.

Drug treatment:

Treatment: use Trimethoprim 15-25 mg/Kg, which amounts to cotrimoxazole 3- 4 single-strength tablets three or four times daily for 21 days (3-4 tablets/Tid or QID for 21days).

Adjuvant treatment

In moderate and severe cases, prednisolone is given with a dosage of 40 mg BID for 5 days, 20 mg BID for 5 days, 20 mg QD until therapy is complete (for 11 days) and no tapering from the 20 mg dose is necessary.

Alternative regimens for mild to moderate cases of PCP include:

1. Clindamycin 600 mg qid plus primaquine 15 mg bid nor
2. Clindamycin 600 mg qid plus dapsone 100 mg daily.

Secondary prophylaxis: after completion of the course of treatment withcotrimoxazole should be started. In addition, the patient has to be prepared for ART as s/he is automatically eligible for ART.

9.14. Cryptococcal Meningitis

The majority of cases are observed among patients with CD4 lymphocyte counts of <50 cells/μL.

Clinical Manifestations:

Cryptococcosis among patients with AIDS most commonly occurs as a sub-acute meningitis or meningoencephalitis with fever, malaise, and headache. Classic meningeal symptoms and signs (e.g., neck stiffness or photophobia) occur in approximately one

fourth to one third of patients. Certain patientsmight present with encephalopathic symptoms e.g. altered mentation, personality changes and memory loss.

Disseminated disease also can occur. Skin lesions and pulmonary manifestations are common.

Diagnosis: depends on laboratory evidence of infection in the CSF and rise of pressure. The CSF usually have a mildly elevated serum protein, normal or slightly low glucose, and a few lymphocytes and numerous organisms. The opening pressure in the CSF is elevated (with pressures >200mm of water) in the majority of patients. Indian ink staining of CSFdemonstrates theorganism inup to 60% of cases. Cryptococcal antigen is almost invariably detected in the CSF at high titer in patients with meningitis.

Treatment:
First-line treatment:
Induction phase - amphotericin B is 0.1 mg/kg testing dose to be escalated to 0.6-1 mg/Kg for 2 weeks, usually combined with flucytosine, for 2 weeks. (Lipid formulations of amphotericin B appear effective and safe but very expensive.)

Consolidation phase: fluconazole alone for 8 weeks, CSF should be checked if it is sterile. Alternative treatment: Therapy with fluconazole alone at a dose of 400-800 mg/daily is effective for treating AIDS-associated cryptococcal meningitis, especially when it is not severe.

Increased intracranial pressure :it might cause clinical deterioration despite a microbiologic response, probably reflects cerebral oedema, and is more likely if the CSF opening pressure is >200mm H_2O. The opening pressure should always be measured when a lumbar puncture is performed.

The principal initial intervention for reducing symptomatic intracranial pressure is repeated daily lumbar punctures. *Corticosteroids -no beneficial effect.

Disposition: Admit patients to the ward

Box D CSF values

Normal values of CSF typically range as follows:

Pressure: 70 to 180 mm H20, Appearance: clear, colorless, CSF Cells : 0-5, CSF total protein: 15 to 60 mg/100 mL, CSF glucose: 50 to 80 mg/100 mL (or >2/3 of blood sugar level), Cells:0-5.

9.15. Pulmonary Tuberculosis

Mycobacterium tuberculosis is the leading cause of morbidity and mortality among people living with HIV/AIDS (PLWHA)worldwide. In Ethiopia the co-infection rate is 20-50% creating a dual epidemic of symptomatic HIV infection and tuberculosis. Tuberculosis enhances progression of HIV infection by inducing immune activation, and HIV increases the risk of infection as well as reactivation of latent tuberculosis. Hence it is conceivable that tuberculosis can occur across the clinical spectrum of HIV infection. Tuberculosis can cause pulmonary and/or extra-pulmonary symptoms depending on the degree of immune suppression. In patients with good immunity, tuberculosis typically involves the apical lung fields and causes either cavitations or fibrosis visible in a chest X-ray. However, in patients with CD4 count below 200 cells/ μL, tuberculosis is often atypical, with lower zone infiltration on chest X-ray and tends to be extra-pulmonary; e.g. pleural effusion, scrofula, meningitis and other form of tuberculosis.

A TB suspect is a patient with persistent cough and/or sputum production of longer than two weeks with constitutional symptoms including fever, weight loss and anorexia. This mode of presentation indicates pulmonary tuberculosis, which is the major form in both HIV positive and negative patients. Symptoms of extra pulmonary disease are more common in patients with HIV co-infection particularly when the CD4 count is low. Systemic symptoms like fever, sweating, weight loss and cough are non-specific manifestations of many complications of HIV, but the health worker should consider tuberculosis as a first possibility. Offer HCT for all TB suspects and refer co-infected individuals to HIV care and treatment. Diagnosis: tuberculosis is likelywhen anyof the following isdetected singlyor in combination:

1. Specific radiological findings on chest X-ray, which include apical fibrosis or cavity lesion.
2. Typical findings of epithelioid cells with gaseous formation on cytologi cal or histological examination of tissue sample or bacteriologic tests, such as smear and culture, from infected sample

Definitive diagnosis of tuberculosis is; however, based on culture.

In Ethiopia, pulmonary tuberculosis is mostly diagnosed when two sputum samples are positive for acid fast bacilli or one sample is positive for AFB and the chest X-ray shows typical radiological features. Tuberculosis can present as immune reconstitution syndrome following commencement of HAART. These patients need to be treated for tuberculosis and continue taking ART, but drug

toxicity and interactions should be monitored strictly; Treatment: TB treatment has two phases: an intensive phase of 8 weeks followed by a continuation phase of 4-6 months, depending on the regimen used. Selection of the regimen depends on the treatment category of the patient (refer to TB-leprosy manual for details). A new TB patient co-infected with HIV belongs to category I and accordingly the following drugs are used:

1. Intensive phase: rifampicin, isoniazid, ethambutol and pyrazinamide for 8 weeks. Follow patients for clinical evidence of hepatitis. Bear in mind that drug interactions of Nevirapine with rifampin can occur and poten tial additive toxicity to the liver when ARVs are used with anti-TB drugs during this phase.
2. Continuation phase: INH with ethambutol for six months.

9.16. Emerging infections

Background
Throughout human history different contagious infections have causes lots morbidities andmortalities. After the era of antibiotics and vaccines there has been a hope to control communicable diseases. Nevertheless, recently, with emergency of new viral species and epidemics, humanity and health care system has faced newest challenges. SARS, MERS and Ebola virus diseases are described here to alert emergency rooms professionals to be vigilant on detection of index cases, control and prevention of the infections and give appropriate management of cases.

9.16.1 Severe Acute Respiratory Syndrome (SARS)

Severe acute respiratory syndrome (SARS) is a viral respiratory illness caused by a coronavirus, called SARS-associated corona virus (SARS-CoV). The first cases of SARS occurred in China in November 2002.

It is manifested with a high fever, headache, and body aches. There might mild respiratory symptoms and diarrhea. Within a week time some of these patients may develop a dry cough followed with pneumonia and acute respiratory distress syndrome.SARS spreads through close person-to-person contact. The virus is thought to be transmitted by respiratory droplets (droplet spread) produced when an infected person coughs or sneezes. Droplet spread can happen when droplets from the cough or sneeze of an infected person are propelled a short distance (generally up to 3 feet) through the air and deposited on the mucous membranes of the mouth, nose, or eyes of persons who are nearby. The virus

also can spread when a person touches a surface or object contaminated with infectious droplets and then touches his or her mouth, nose, or eye(s). In addition, it is possible that the SARS virus might spread more broadly through the air (airborne spread) or by other ways that are not now known.Except supportive care there was no specific therapy when transmission was stopped in 2004.

9.16.2 Middle East respiratory syndrome coronavirus (MERS-CoV)

Middle East respiratory syndrome (MERS) is a viral respiratory disease caused by a coronavirus (MERS-CoV) that was first identified in Saudi Arabia in 2012.

Epidemiology and Transmission:
The majority of human cases of MERS have been attributed to human-to-human infections; camels are likely to be a major reservoir host for MERS-CoV and an animal source of MERS infection to humans. Close contact such as providing unprotected care to an infected patient is suggested to be significantly linked with the transmission to another person. There have been clusters of cases in healthcare facilities, where human-to-human transmission appears to be more probable, especially when infection prevention and control practices are inadequate. Most of these infections are believed to have been acquired in the Middle East, and then exported outside the region. The outbreak in Republic of Korea had the largest outbreak outside of the Middle East.

Clinical Features:
The clinical spectrum of MERS-CoV infection ranges from asymptomatic or mild respiratory symptoms to severe acute respiratory disease and death. Typical MERS symptoms include fever with or without chills, cough and shortness of breath and occasionally diarrhea has been seen. Most patients have had severely pneumonia and acute respiratory distress syndrome, and among them many needed mechanical ventilation and in some MERS-CoV infected patients acute kidney injury and DIC was seen. Approximately 36% of reported patients with MERS have died and MRS become fatal in elderly and people with co morbidities like chronic lung disorders, hypertension,diabetes, kidney diseases and those with weakened immune system.

9.16.3 Ebola virus disease

Ebola virus disease (EVD) is a severe contagious viral illness caused by Ebola virus, often fatal to humans.

Epidemiology and Transmission: The virus is transmitted to people from wild animals and spreads in the human population through human-to-human transmission. The virus is excreted in the human body fluids like blood, urine, sweat, diarrhea and vomiting, etc, and all can be infectious when a contact has. Many people are also infected through burial procedures of deceased people and sexual contacts.

The first EVD outbreaks occurred in remote villages in the DRC around Ebola River, near tropical rainforests, and so far 24 outbreaks had happened. But the most recent outbreak in West Africa has involved major urban as well as rural areas and Liberia, Guinea and Sierra Leone were severely affected and Nigeria had also limited number of cases. There have been more cases and deaths in this outbreak than all others combined.

Clinical Features:
The incubation period is 2 to 21 days and patients will become contagious only after they develop symptoms. Clinically it manifest with sudden onset of fever fatigue, muscle pain, headache and sore throat. This is followed by vomiting, diarrhea, rash, symptoms of impaired kidney and liver function.In some cases hemorrhagic features with internal and external bleeding (e.g. oozing from the gums, blood in the stools) is common.

The average EVD case fatality rate is around 50%. Case fatality rates have varied from 25% to 90% in past outbreaks.

Diagnoses of emerging viral infections
High index of suspicion and identifying the index case is very important. Different tests are like PCR and serological tests are used depending on the virus.

Treatment:
There is no established treatment in many of the viral syndromes but supportive care, including the nursing care is crucially important and is lifesaving.

Prevention and control:

The rules in these problems are case detection, supportive care, prevention and control of transmission. For these to be effective the following important issues should be addressed:

- High index of suspicion in the emergency rooms and out patients departments or wards when unusual symptoms appear. History travel or any connection with people who have travelled to other regions or countries is important in many viral syndromes.
- Health facility should adhere to standard precaution methods of infection prevention in all circumstances and when there is specific suspicion specific methods of contact, droplet or airborne precaution with usage of specific barriers become practical.
- Cases management with supportive care, like hydration, pain management, oxygenation and ventilator support, managing secondary infections, etc
- Surveillance and contact tracing with isolation of contacts in problems like Ebola.
- Community engagement and raising awareness of risk factors is very important.
- Community should be cooperative in prevention and cases tracing and also avoid harmful practice. For instance safe burial practice is very important in Ebola epidemic control and may need modification or alteration of culture of the community.

- A good laboratory service is essential to confirm or rule out these problems timely.
- Emergency departments should work very closely with the FMOH, specifically the public health emergency directorate section for notifying cases, involving in surveillance; participate in rapid response team and case management.

9.17. Infection Prevention/Control Procedures

Routes of Transmission

- Contact: Infections spread by direct or indirect contact with patients or the patient-care environment(e.g., shigellosis, typhoid, staphylococcal)
- Droplet: Infections spread by large droplets generated by coughs, sneezes, etc. (e.g., Neisseria meningitides,)
- Airborne (droplet nuclei): Infections spread by particles that remain infectious while suspended in the air (TB, measles)

Box E Emergency Department Infection Prevention and Control Rule

All faculty physicians or nurses, residents, staff nurses, and students in any categories should be trained on IP guidelines before practicing in the department and should abide by the following standard precaution principles

Standard Precaution:

Hand hygiene

- Perform Hand hygiene should be performed before and after any direct contact with a patient, after contact with blood, body fluids, secretions and excretions and after contact with items contaminated with blood, body fluids, secretions and excretions, including respiratory secretions

Use alcohol-based hand rub or wash hands with soap and water

- Wash hands if visibly soiled

Respiratory hygiene

- Source control measures (e.g., cover cough to prevent dissemination of infectious droplets)
- Spatial separation (> 1 meter distance between patient and other contacts)
- Ventilation
- Hand hygiene
- Isolate the patient in a single room and Limit patient movement
- Education of patients, visitors and staff

Use of personal protective equipment (PPE

- Gloves – protect hands
- Gowns/aprons
- Masks and respirators
- Goggles – protect eyes
- Face shields – protect face, mouth, nose, and eyes

Prevention of needle sticks/sharps injuries

- Do not recap, cut, or bend used needles.
- Dispose of sharps in a sharps container.
- Use a face shield, pocket mask, or other airway adjunct for resuscitation

Cleaning and disinfection of the environment and equipment

- o Environmental cleaning: Use adequate procedures for the routine cleaning and disinfection of environmental and other frequently touched surfaces.
- o Waste disposal: Treat waste contaminated with blood, bodily fluids, secretions and excretions as clinical waste, in accordance with local regulations.

E**mergency Department Infection Prevention Quality Improvement work**

1. The Department considers infection control to be a priority for the safety of our staff and our patients.
2. There is a designated IP focal person from nurses and physicians who will monitor weekly the FMOH IP standards compliance of the department.
3. The coordinator nurses and IP focal person will conduct IP monitoring rounds before daily work starts.
4. The department will get IP report weekly and act accordingly.
5. There will be bi-yearly department quality summit and IP progress will be discussed.

Section 10 Fluid and Electrolyte Management

10.1 Sodium disorders

10.1.1 Hyponatremia

- Hyponatremia: - is defined as a serum [Na+]Serum Na <135
- **Classification:** Mild if it is between 130-135meq/l, moderate 125-130meq/l and below 125meq/l is severe Rate of change and the clinical features are more important than actual value
- **Causes of hyponatremia:** hypovolemic states (as in GI, renal, skin losses and edematous states), no hypovolemic states of ADH excess (as in Syn drome of Inappropriate ADH secretion, cortisol deficiency and hypothy roidism) and renal failure.
- **Clinical Features:** neurologic dysfunction caused by hypo osmolality the most important features. Severity and rapidity of onset of hyponatremia are two important factors in the development of the clinical manifestations. Initial symptoms of Nausea and malaise are earlier symptoms and occur as plasma Na acutely falls below 125 mEq/L. Between 115 and 120 mEq/l headache, lethargy and obtundation may occur although those with chronic hyponatremia will have few if any symptoms. With plasma Na less than 110 to 115 more severe changes like seizures and coma appear.
- **Diagnosis:** The history (possibly of vomiting, diarrhea, diuretic therapy, one of the causes of SIADH) and physical exam (findings of volume depletion or edema) can provide important clues to the cause of hyponatremia. The initial lab evaluation should include measurement ofplasma osmolality and plasma Na as well as K, Cl, HCO3, BUN/CR and glucose.

Recommendations regarding the correction of hyponatremia:

If asymptomatic

- Hypovolemic: Give isotonic fluid (0.9NS)
- hypovolemic: Fluid restrict, maximize renal perfusion
- Euvolemic and SIADH : Fluid restriction

If symptomatic, need aggressive therapy: 3% saline

- Treatment:

In symptomatic patients or severe hyponatremia like plasma Na is less than 115mEq/L(especially in acute circumstance) administration of hypertonic saline i.e. 3% NaCl is needed, since these are the settings in which irreversible neurologic damage and death can occur.

Box F Important tips

- **Rapid correction can lead to osmotic demyelination (central pontine myelinolysis).**
- **Cerebral adaptation: Within first day of acute hyponatremia, brain secretes osmolytes, lowering volume towards normal. In this setting, rapid correction of severe hyponatremia can lead to central pontine myelinolysis.**
- **Plasma sodium rise should be moderate like to 10-12 meq/L per day(to 120-125meq/l).**
- **Asymptomatic:increase the sodium level by no more than 0.5-1 meq/L/h to a maximum increase of 12 meq/L per day**
- **Symptomatic: (Na<120 meq/L) Increase the sodium level by no more than 1meq/L per hour until the serum Na level reaches 130 meq/L or neurologic symptoms are improved**
- **If very symptomatic (seizures/severe neurologic problems, can correct: 1.5-2.0 meq/L per hour for first few hours until symptoms resolve.**

Na deficit is calculated first as follows:

For instance measured Na =105meq/l and the target is to elevate it to 115meq/l in 24 hrs in 70kg person, then Na deficit= Na deficit per liter (115-105) x volume of distribution of Na (70kgx0.6) =420mmol. 420mmol is to be given in 24 hrs .

Box G Important clues on calculation

- **Volume of distribution is 60 and 50% of body weight respectively in men and women and the Na deficit per liter is the target Na- actual Na**
- **Concentrations: Isotonic Saline(0.9%Nacl=154meq/l, 3%Nacl=514meq/l**
- **Volume needed per 24hrs=calculatedmeqx1000/514=420x1000/514=81ml.**
- **Calculation For 1hr,the goal in the example is to increase serum Na to 10meq(total)=817ml,then 1meq=81.7ml per hr. Hence the first hours usually 100-150 ml of 3% Nacl is given hourly for 4 hrs).But in seizure and special CNS threat 3-5ml/kg bolus can be given.**

10.1.2 Hypernatremia (Hyperosmolar states)

Hypernatremia represents hyperosmolality with plasma Na >145meq/l. In this condition cellular dehydration is responsible for the neurologic symptoms associated with hypernatremia. It occurs in patients who cannot express thirst and hence poor ADH release which keeps plasma osmolality within narrow limits .Hypernatremia primarily occur in children or unconscious adults with diabetes insipidus. Patients are more likely to be symptomatic when hypernatremia develops acutely, <48 hrs.

Symptoms: The symptoms of hyponatremia are primarily neurologic. Lethargy, weakness and irritability are the earliest findings which can then progress to twitching, seizures and coma.

Treatment:

- Calculate the water deficit which is equal to TBW×(Plasma Na/140—1)
- In volume depleted patients, intravascular volume restoration takes precedence, with isotonic fluid.
- In symptomatic acute hypernatremia patients give oral or IVwater (5% D/W or hypotonic saline or P.O. water untilwater deficitcorrected)
- Give vasopressin if there is central diabetes insipidus
- When it is chronic, rapid correction should be avoided to prevent cerebral oedema and treatment should be directed to the underlying cause
- Rate of reduction should be no more than 0.5-1mmol/hr. and not exceed 12 mmol in the first 24 hrs. After symptoms resolve slower correction rate.
- Only half of the water deficit should be corrected in the first 24 hours; in most clinical situations, the total deficit will require replacement over the next 24 - 72 hours
- RISK of CEREBRAL EDEMA WITH RAPID CORRECTION
- In the volume overloaded water repletion + solute removal (diuretics).

10.2 Potassium disorders

10.2.1 Hyperkalaemia

Plasma potassium depends on the balance between intake, excretion and the distribution of potassium across cell mem¬branes and excretion is normally controlled by the kidneys. The causes are: reduced renal excretion (e.g. chronic renal failure, ad¬renal insufficiency, diabetes, potassium sparing diuretics). Intracellular potassium release (e.g. acidosis, rapid transfusion of old blood, cell lysis including rhabdo¬myolysis, haemolysis, and tumourlysis and potassium (K)+ channel openers.

Evaluation and diagnosis of Hyperkalemia:

The first step is to rule out pseudohyperkalemia—delayed processing of specimen, hemolysisLeukocytosis 100,000/dl, Thrombocytosis 1000, 000/dl) and severity determination. When K level >7mmol/dl or there are advanced ECG changes urgent measure is needed. The earlier ECG features are prominent T wave and fattening or P wave. When it is severe there will be QRS prolongation, sinus wave and asystole/PEA features.

Treatment

• General: avoid K + intake ,K+binding resins , Diuresis, Discontinuation of potassium-sparing diuretics, ACE inhibitors, ARBs, NSAIDs ,and dietary K restriction

• Specific: follow the following hyperkalemia management protocol

Treatment of moderate to severe hyperkalemia

- Give 50 ml of 50% glucose which is 25g with 10 units of soluble insulin IV over 15-30 min in less severe cases (K<7, earlier ECG features). The onset time is 15 minutes, peak effect with 1hr and the effect stays 3-4 hrs. Check blood glucose after glucose/insulin has been given to exclude rebound hypoglycemia.
- Give 10 ml of calcium gluconate/chloride 10% IV over 5 min when K level is >7mmol/dl, there is muscle weakness and there are wide complex QRS or sinus wave or Asystole/PEA. This can be repeated every 5 min up to a total dose of 40 ml. Calcium chloride is more toxic to veins than calcium gluconate, but provides more calcium per ampoule.
- Give50 mmol (50 ml of 8.4% solution) bicarbonate IV over 30 min preferably via a central line If hyperkalemia is associated with a severe metabolic acidosis (arterial pH <7.2).
- Stop potassium supplements or any drugs (e.g. ACE inhibitors, potassium-retaining diuretics)
- If hyperkalemia is due to acute renal failure, dialysis may need to be started to prevent a recurrence: Consult renal experts
- Start potassium removing resins if available like calcium polystyrene. Restriction of dietary potassium intake.

Table 11: Treatment modalities of Hyperkalemia

Drug/Procedure	Mechanism of Action	Dosage	Onset time	Peak effect	Duration of action	Remark
Calcium gluconate/chloride	Improves membrane stability	10ml of 10% solution	5 minutes	-		Repeat after 5 minutes
Insuin/ Glucose(I/G) Infusion	Shift K to the cell	10iu insulin(I) and 50ml of 50% glucose(25gm),10'	15minutes	60 min	3-4 hrs	Continuous infusion of I/G possible In severe metabolic acidosis
NaHCO3	Shift K to the cell	50 ml of 50mmol ,8.4 meq/l with in 30'	15minutes	30-60 min		
Cation(K) binding resins	Bind K in the Gut -remove	15-30gm in 2-3 divided doses orally				
Hemodialysis						

10.2.2 Hypokalemia

Introduction:
Hypokalemia occurs when potassium<3.5mmol/l and its etiology can be increased losses of K (from the GI, through the urine or sweat), decreased dietary intake or increased shift into cells.

It is mostly asymptomatic.When it is severe, K <3.0 it can cause muscleweakness, paralytic ileus, cardiac arrhythmias, rhabdomyolysis and renal dysfunction.

Evaluation and Diagnoses:
When hypokalemia is suspected determine K and other electrolytes determination including RBS.When correction of hypokalemia is a problem determine magnesium level too. ECG can be done which can be manifested with ST depression, flattening of T waves and abnormal U waves.

Treatment

- Oral supplementation with a total intake of 80–120 mmol/day,KCL tabs 2-3 bid or tid, including nutritional input is preferred route when there are no clinical features.
- Slower intravenous replacement (20 mmol over 1 -2 h) is recommended dose in symptomaticpatients without arrhythmias and K dilution should not exceed 60mm/l. (Example-2 ampoules of IV kcl=40mmol/l is given in2- 4 hrs).NO bolus or undiluted K administration.
- When there is a clinically signif¬icant arrhythmia, largerdose of 20 mmol over 30 min can be attempted but should be intravenous (via cen¬tral line) and ECG monitoring.
- Mg++ level determination and replacement is necessary in resistant hypokalemia (hypomagnesemia impairs renal K retention) and wherever possible, the cause of potassium loss should be treated.

Monitoring

- Severe hypokalemia, likein DKA patients has to reassessed Q4-6 hours while mild to moderate has to be daily.

10. 3 Calcium disorders

Calcium

- Normal range: 8.8-10.1 with half bound to albumin
- Ionized (free or active)calcium: 4.4-5.4 – relevant for cell function
- Majority is stored in bone

10.3.1 Hypercalcemia

Clinical presentation:

- Groans: constipation
- Moans: psychic moans (fatigue, lethargy, depression)
- Bones: bone pain
- Stones: kidney stones
- Psychiatric overtones: depression & confusion
- Fatigue, anorexia, nausea, vomiting, pancreatitis
- ECG: short QT interval, widened T wave

Medical management of hypercalcemia

- Administer high volume of NS0.9% to dilute the serum and increase urine output
- Phosphate may be given as it increase calcium excretion
- Lasix rarely given as it increase excretion
- Also rarely Calcitonin may be given as it move calcium from the blood to the bone.
- Glucocorticoids
- Dialysis

10.3.2 Hypocalcemia

- Causes
- Eating disorder
- Hungry bone syndrom Ingestion: mercury, excessive Mg
- Chelation therapy EDTA
- Absent of PTH
- Ineffective PTH: CRF, absent or ineffective vitamin D, pseudo hypoparathy roidism
- Deficient in PTH: acute hyperphos: TLS, ARF, Rhabdo
- Blood transfusion

Clinical presentation

- Neuromuscular irritability
- Paresthesias: oral, perioral and accrual, tingling or pin & needles
- Tetany (Chvostek & Trousseau signs)
- Hyperreflexia
- Laryngospasm
- Jittery, poor feedings or vomiting in newborns
- ECG changes: prolonged QT intervals

Treatments

- Supplements
- IV: gluconate or chloride with EKG change
- Oral calcium with vitamin D
- Medical management
- Increase dietary intake(milk, green leafy vegetables, canned salmon, sardines, and oyster
- IV supplement as calcium gluconate, or calcium chloride
- Vitamin D therapy (increase absorption from the GIT

Magnesium:-

- Normal range: 1.5-2.3
- 60% stored in bone
- 1% in extracellular space
- Necessary cofactor for many enzymes
- Renal excretion is primary regulation

10.4.1 Hypermagnesemia

Causes

- Hemolysis
- Renal insufficiency
- DKA, adrenal insufficiency, hyperparathyroidism, lithium intoxication

Clinical presentation

- Weakness, nausea, vomiting
- hypocalcemia ,hypotension
- Arrhythmia and asystole
- 4.0 mEq/L hyperreflexia
- >5 prolonged AV conduction
- >10 complete heart block
- >13 cardiac arrest

Treatments

- Calcium infusion
- Diuretics
- Dialysis

10.4.2 Hypomagnesemia

Causes

- **Alcoholism**: malnutrition + diarrhea; Thiamine deficiency
- **GI causes:** Crohn's, UC, Whipple's disease, celiac sprue
- **Renal loss:** Bartter's syndrome, post obstructive diuresis, ATN,
- DKA
- Drugs
    - Loop and thiazide diuretics
    - Abx: aminoglycoside, ampho B, pentamidine, gent, tobra
    - PPI
    - Others: digitalis, adrenergic, cisplatin, cyclosporine

Clinical presentation

- Weakness, muscle cramps
- Cardiac arrhythmias
    - Prolonged PR, QRS & QT
    - Torsade de pointes
    - Complete heart block & cardiac arrest with level >15
    - CNS: irritability, tremor, athetosis, jerking, nystagmus
    - Hallucination, depression, epileptic fits, HTN, tachycardia, tetany

Treatments

- Oral or IV supplement
- Correct on going loss

Section 11 Gastrointestinal Emergencies

11.1 Acute upper gastrointestinal bleeding (UGIB)

Acute upper gastrointestinal bleeding(UGIB) can be defined as bleeding from any site along the gastrointestinal tract (GIT) that is above the ligament of treitz.

Etiology

- Peptic ulcer is the commonest cause of acute upper gastrointestinal bleeding (UGIB).
- Consider variceal bleeding in patients with
    - history of alcohol ingestion
    - Chronic hepatitis (especially hep. B and C)
    - previous variceal bleed Presence of stigmata for chronic liver disease (e.g. palmar erythema, spider naive, etc), or portal hypertension (e.g ascites, splenomegaly, caput medusa).

- **Clinical presentation**
    - presents with either haematemesis or melaena or both

- Investigations
    - Urgent full blood count
    - Blood grouping and cross matching
    - Coagulation screen
    - Blood urea and electrolyte
    - Liver function tests.
    - *Stool for O/Occult blood*
    - *Endoscopy (diagnostic) – performed within 24hrs in most patients.*

Treatment

ABC Resuscitation- for emergency cases, when patient presents with an ongoing blood loss, in shock or unconscious.

- A- Clear airway:- Suctioning in cases of haematemesis

Left lateral position to prevent aspiration.

- B- Ensure breathing: - *Oxygen therapy for patients in shock, for those who are confused, agitated or elderly.*
- **C- Circulation:** -*Establish one or more intravenous accesses with wide bore cannulae, if bleeding is brisk and massive.*
    - *Blood transfusion with fresh whole blood*
    - *Crystalloid (like N/S) until blood becomes ready (with target haemo globin of 7-9mg/dl).*

-continue monitoring pulse and BP
-avoid circulatory overload
Consider endoscopy

Specific treatment

- Early upper gastrointestinal endoscopy after resuscitation (within 12 to 24 hours) is the cornerstone of management of upper gastrointestinal bleed (UGIB). Early endoscopy has 3 major roles, which is for diagnosis, treatment and risk stratification.
- High dose intravenous proton pump inhibitors (e.g i.v. Omeprazole or Pantoprazole 80 mg stat followed by an infusion of 8 mg hourly for 72 hours) should be commenced.
- Interventional radiology (embolization therapy) or surgery should be considered when bleeding is unresponsive to endoscopic hemostasis or failure of endoscopic visualization of the bleeder due to profuse hemorrhage or inaccessibility.
- In patients with variceal bleeding,

-Terlipressin is administered as IV injection of 2 mg bolus and 1 mg every four to six hours for 48 hours. Alternatively, Octreotide is administered as a bolus injection of 50 mcg followed by an infusion at a rate of 50 mcg per hour.
- Antibiotic prophylaxis (such as quinolones or third generation cephalosporines) should be given for 7 days.
- If endoscopy is unavailable, consider balloon tamponade with a Sengstaken–Blakemore tube and referral to the nearest tertiary center.
- When there is failure to control bleeding, consider repeating endoscopy, surgical intervention or transjugular intrahepatic portasystemic shunts (TIPS).
- Fresh frozen plasma may be given if the prothrombin time is at least 1.5 times higher than the control value.

Monitoring and evaluation should include:

- Frequent measurement of blood pressure, pulse rate and oxygen satura tion. (Postural hypotension of 10 mmHg or more usually indicates at least 20% reduction in blood volume).
- Level of consciousness
- Severity (amount) of bleeding
- Urine output
- Consider securing Central venous pressure (CVP) monitoring in pa tients with profound shock or organ failure and in elderly patients with significant co-morbidity.

- Initial hemoglobin or haematocrit level obtained in a patient with acute bleeding may not be reflective the degree of blood loss. This is due to haemoconcentration.
- Consider intubation for airway protection in severe uncontrollable bleeding, encephalopathy and inability to maintain oxygen saturation adequately and to prevent aspiration.
- Continuous haematemesis or persistent hypovolemia despite aggressive resuscitation suggest bleeding is still active. Passage of "fresh" Malena, which is maroon colored or passage of bright red visible clots suggest active bleeding.

Consultations: Consultation with a Gastroenterologist and surgeon should be considered for all patients with gastrointestinal hemorrhage.
Disposition: Admit to ICU if the patient is unstable

11.2 Acute abdomen

- Acute abdomen represents the rapid onset of severe symptoms that may indicate potentially life-threatening intra-abdominal pathology that requires urgent surgical intervention.
- Abdominal pain is usually a feature, but a pain-free acute abdomen can occur, particularly in older people, in children, immunocompromised and in the last trimester of pregnancy.

Etiologies

The differential diagnosis is extremely wide and definitive diagnosis is often difficult, particularly in primary care. This is due to the many different organs within the peritoneal cavity and the potential for referred pain.

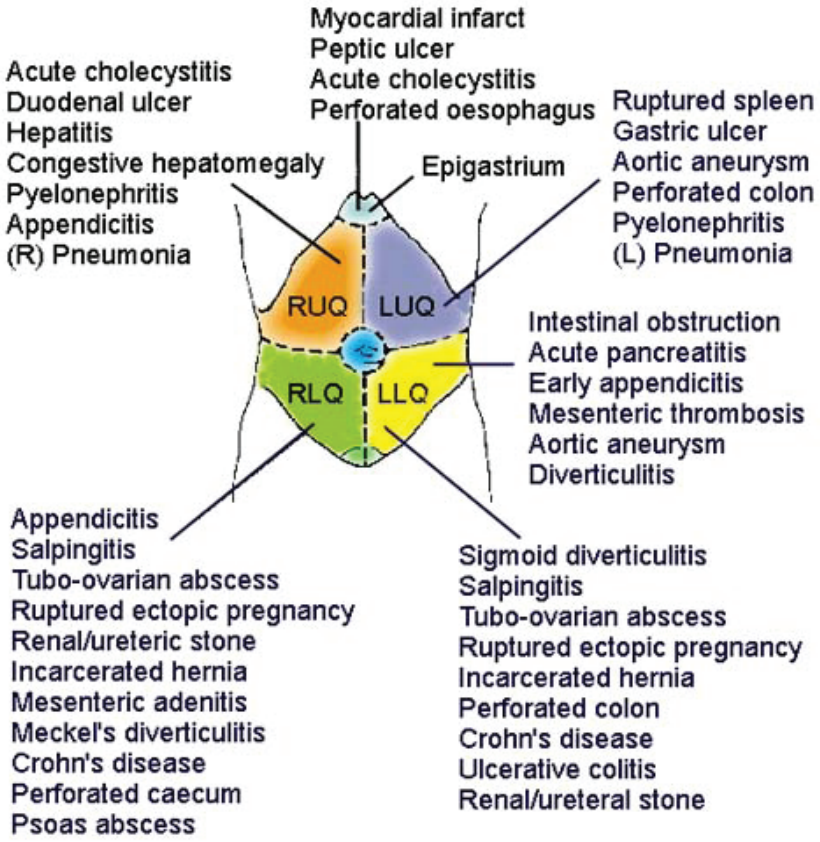


Figure 6. Classification of causes according to site of pain

Assessment
Initial impression/observation
Note whether the patient looks ill, septic or in shock.

Note whether they are lying still (think peritonitis) or rolling around in agony (think intestinal, biliary or renal colic).

Assess and manage Airway, Breathing and Circulation (ABC) as a priority.
In an emergency department setting: if there are signs that the patient is shocked or acutely unwell assess quickly but carefully and arrange any early investigations.

In a community setting: make arrangements for rapid transfer to hospital for further assessment.

History
This should cover the following points:
Demographic details, occupation, recent travel, history of recent abdominal trauma.

Pain: Onset (including whether new pain or previously experienced), Site (ask the patient to point), localised or diffuse, Nature (constant/intermittent/colicky), Radiation, Severity, Relieving/aggravating factors (e.g., if worsened by movement/coughing, suspect active peritonitis; pancreatitis is relieved by sitting forward).

Associated symptoms: vomiting and the nature of vomitus (undigested food or bile suggests upper GI pathology or obstruction; faeculent vomiting suggests lower GI obstruction).

Haematemesis or melaena.
Stool/urine color.
New lumps in the abdominal region/groins.
Eating and drinking - including when the patient's last meal occurred.
Bowels - including presence of diarrhoea, constipation and ability to pass flatus.
Fainting, dizziness or palpitations.
Fever/rigors.
Rash or itching.
Urinary symptoms.
Recent weight loss.
Past medical and surgical history/medication.

Gynaecological and obstetric history:
Contraception (including intrauterine contraceptive device (IUCD) use).
Last menstrual period.
History of sexually transmitted infections/pelvic inflammatory disease.
Previous gynaecological or tubal surgery.
Previous ectopic pregnancy.
Vaginal bleeding.
Drug history and allergies - including any complementary medication.

Examination
Pulse, temperature and blood pressure.
Assess respiratory rate and pattern. Patients with peritonitis may take shallow, rapid breaths to reduce pain.
If there is altered consciousness, check Glasgow Coma Scale (GCS) or AVPU (Alert, responds to voice, responds to pain, unresponsive).

Inspection:
Look for evidence of anemia/jaundice.
Look for visible peristalsis or abdominal distension.
Look for signs of bruising around the umbilicus (Cullen's sign - this can be present in haemorrhagic pancreatitis and ectopic pregnancy) or flanks (Grey Turner's sign - this can be present in retroperitoneal haematoma).
Assess whether the patient is dehydrated (skin turgor/dry mucous membranes).

Auscultation:
Auscultate the abdomen in all four quadrants.
Absent bowel sounds suggest paralytic ileus, generalized peritonitis or intestinal obstruction. High-pitched and tinkling bowel sounds suggest subacute intestinal obstruction.
Intestinal obstruction can also present with normal bowel sounds.
If there is reason to suspect aortic aneurysm, listen carefully for abdominal and iliac bruits.

Percussion:
Percuss the abdomen to assess whether swelling/distension might be due to bowel gas or ascites.
Patients who display tenderness to percussion are likely to have generalized peritonitis and this should act as a red flag for serious pathology.
Assess for shifting dullness and fluid thrill.

Palpation:
Palpate the abdomen gently, then more deeply, starting away from the pain and moving towards it.
Feel for masses, tenderness, involuntary guarding and organomegaly.
Test for rebound tenderness.
Examine the groins for evidence of hernia.
Always examine the scrotum in men, as pain may be referred from unrecognized testicular pathology.
Check supraclavicular and groin lymph nodes.

Further examination:
Perform rectal or pelvic examination as needed, with an appropriate chaperone in attendance.
Check lower limb pulses if there could be an abdominal aortic aneurysm.
Dipstick urine and send for culture if appropriate.
In a woman of childbearing age, assume that she is pregnant until proven otherwise - perform a pregnancy test.
Examine any other system that might be relevant - eg, respiratory, cardiovascular.

Emergency department care of suspected acute abdomen
Keep the patient nil by mouth.
Apply oxygen as appropriate.

Intravenous (IV) fluids: set up immediately if the patient is in shock

Send blood for group and save/cross match and other blood tests as appropriate.

Consider passing a nasogastric (NG) tube if severe vomiting occurs, there are signs of intestinal obstruction or the patient is extremely unwell and there is danger of aspiration.

Analgesia: the previous practice was to withhold analgesia until surgical review, but a surgical abdomen is very painful and is likely only to be adequately relieved by parenteral opiates - e.g., morphine.

Antiemetic: avoid using this as a symptomatic treatment without considering a diagnosis in a community setting.

Antibiotics: if systemic sepsis, or peritonitis, or severe urinary tract infection (UTI) is suspected. IV cephalosporin plus metronidazole are commonly used in acutely unwell patients in whom peritonitis is suspected.

Arrange urgent surgical/gynaecological review as appropriate.

Arrange investigations such as ECG if a medical cause is likely.

Admit: if surgery is considered likely, if the patient is unable to tolerate oral fluids, for pain control, if a medical cause is possible or if IV antibiotics are required.

Investigation
The following tests are often used but can be nonspecific and must be interpreted in the clinical context and with appropriate medical/surgical expertise:

Blood tests: FBC, U&Es, LFTs, amylase, glucose, clotting, and occasionally calcium; arterial blood gas (pancreatitis).
'Crossmatch
Blood cultures.
Pregnancy test in women of childbearing age.
Urinalysis.
Radiology - CXR (erect looking for gas under the diaphragm), intravenous pyelogram (IVP), CT scan and ultrasound, as appropriate.
Consider ECG and cardiac enzymes.
Peritoneal lavage if there is a history of abdominal trauma.
Red flags that raise suspicion of serious pathology
Hypotension.
Confusion/impaired consciousness.

Signs of shock
Systemically unwell/septic-looking.

Signs of dehydration
Rigid abdomen, patient lying very still or writhing, absent or altered bowel sounds, associated testicular pathology, marked involuntary guarding/rebound tenderness, tenderness to percussion, history of haematemesis/melaena or evidence of latter on examination per rectum (PR and suspicion of a medical cause for abdominal pain.

Special situations
Children: Pain etiology varies with age; history and examination can be difficult.

Pregnancy: Always consider ectopic pregnancy in women of childbearing age. Causes of acute abdomen in late pregnancy are different and require expert evaluation on for obstetric, gynaecological and surgical conditions.

Elderly: Tend to show less specific symptoms and signs.
Tend to present later in the course of their illness.

Morbidity and mortality is high

Diagnosis requires higher index of suspicion of serious pathology

Aortic aneurysm and bowel ischaemia are more prevalent in the elderly.

Angiodysplasia of the colon is more common and can cause GI haemorrhage.

Medical causes of abdominal pain are encountered more frequently.

The 'Top 5' medical causes of an acute abdomen to consider in older patients are Inferior myocardial infarction.

Lower-lobe pneumonia/pulmonary embolism causing pleurisy.

Diabetic ketoacidosis or hyperosmolar hyperglycemic states.

Pyelonephritis.

Inflammatory bowel disease.

Biliary tract disease, including cholecystitis, is the most common indication for surgery in older patients with abdominal pain. This is thought to be due to age-related changes in the biliary tract.

Disposition:-
Admit to ICU if the patient is unstable

Dispose to OR who need urgent surgical intervention

11.3 Acute diarrhea and vomiting

11.3.1 Acute diarrhea

The passage of three or more loose or watery stool in 24 h, or passage of one or more bloody stool.

- Acute diarrhea refers to illness not lasting longer than 14 days.
- Usually self-limited

Types of diarrhea

- Watery Diarrhea: 3 or more liquid or watery stools in 24 h
- Dysentery: Presence of blood and/or mucus in stools
- Persistent Diarrhea: Diarrhea lasting for 14 days or more

Etiology:-

- Can be caused by viral and bacterial pathogens.
- Persistent diarrhea suggests an enteric pathogen other than viral, such as bacterial or protozoan.
- Chronic diarrhea usually is associated with noninfectious causes and re quires further testing to determine the etiology.
- Normally, the small and large bowels absorb 99% of gastrointestinal tract secretions. Any pathologic state that reduces water absorption by 1% can cause diarrhea.

Emergency Assessment and Stabilization:-

- An immediate assessment should be made of the patient's stability, (ABC o f life), with particular attention to volume status.
- Tachycardia, orthostatic hypotension, poor skin turgor /color, diaphoresis, and mental status changes all are characteristic of hypovolemia and hy poperfusion.
- Associated septic shock may contribute to the hypotension and general or gan hypoperfusion, and diarrhea may be a manifestation of toxic shock syndrome.
- A diarrhea-associated acid-base disorder should be suspected in patients with Kussmaul respirations, a significant anion gap on basic metabolic panel reflecting a lactic acidosis from significant volume loss or a non–anion gap metabolic acidosis associated with massive bicarbonate loss.
- After stabilization, a secondary survey may elucidate the potential cause of the diarrhea and direct for further evaluation and treatment

Secondary Survey

- The physical examination should assess the patient's overall health, toxicity, fever, volume status, signs of a surgical abdomen, and determine the pres ence of blood in the stool.

- Young healthy adults may maintain a normal blood pressure and heart rate even with significant dehydration.
- In patients who are taking antiarrhythmic or beta-blocker medications or have conduction disease or fixed-pace rhythms, heart rate may not be a reliable indicator of volume status.
- Signs of volume depletion and impending shock include dry mucosa, poor skin turgor, decreased urine output and mental status changes.
- Children will present with sunken eyes, depression of the fontanel, decrease in urine output (number of wet diapers) and decrease in alertness and ac tivity.
- Particular attention should be given to the abdominal examination. Focal abdominal pain with peritoneal findings may be due to an acute surgical ab domen with symptoms mimicking those of severe gastroenteritis.
- A rectal examination should be performed to detect fecal impaction, me lena, or hematochezia.

Investigation

- Stool culture for bacteria: may be warranted in patients who are febrile, toxic-appearing, immunocompromised, at the extremes of age, experienc ing a prolonged course or if no response to conventional treatment
- Stool examination for ova and parasites: The assessment of stool for ova and parasites is not routinely recommended. This study is used in patients with chronic diarrhea (E. histolytica, Cryptosporidium); patients with a his tory of travel to endemic areas
- Urinalysis: A urinalysis and a urine pregnancy test should be obtained only when urinary tract infection is a possibility, a gastrointestinal origin for the symptoms is not clear, or pregnancy is suspected.
- Radiographic studies: Plain radiographs and contrast computed tomogra phy (CT) may be indicated for patients thought to have a surgical abdomen and to identify pathologic abnormalities, such as tumor, obstruction, free air, fistulas, blind loops, and those associated with Crohn's disease.

Treatment:-

- Initial treatment of any patient begins with the ABC of life.
- Rehydration of severely dehydrated patients should begin immediately af ter large-bore IV access is achieved
- Unless the patient is comatose or severely dehydrated, oral rehydration with a glucose-based electrolyte solution should be initiated in patients without associated nausea or vomiting.
- The World Health Organization recommends a solution with a higher so dium concentration is well suited to more severe dehydration.

- Mildly dehydrated patients should aim to drink 30 to 50 mL/kg over the next 4 hours. For moderate dehydration, patients should drink 100 mL/kg over the next 4 hours
- Lactose and caffeine should be avoided
- Encourage patients to attempt early solid food intake following the above restrictions to expedite recovery from diarrheal illnesses..
- Treatment is dictated by the differential diagnosis.
- Treatment hinges on treating or excluding the life-threatening causes of diarrhea.
- Avoid ant motility agents in patients with dysentery or other evidence of invasive bacterial or protozoan disease

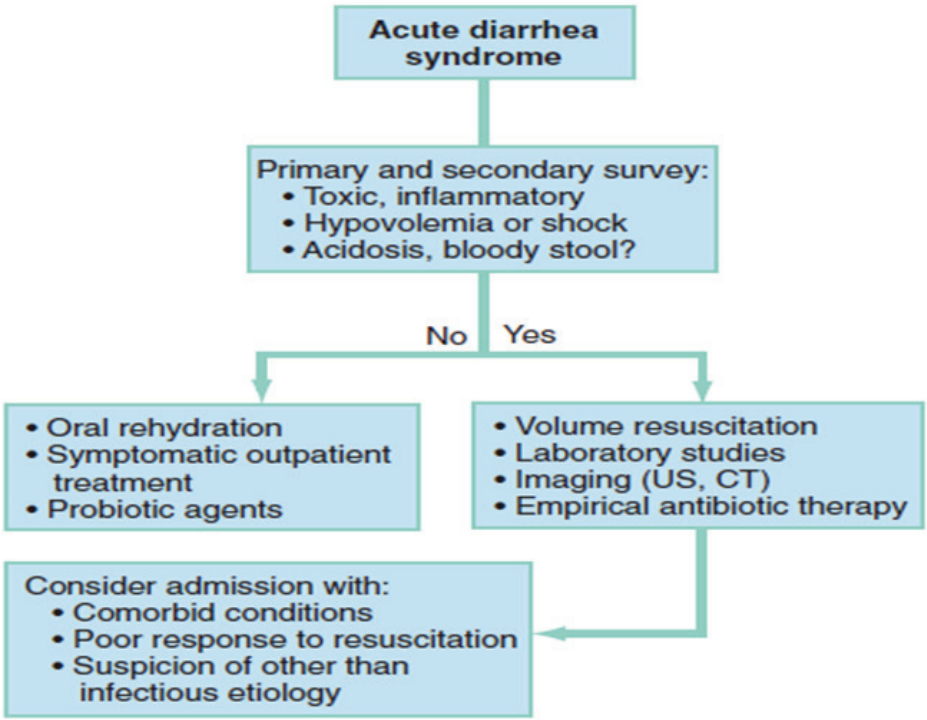


Figure 7: Algorithm for the approach and management of acute diarrhea syndrome

11.3.2 Vomiting

Vomiting is the expulsion of gastric contents.

- The etiology of vomiting should be identified prior to treatment initiation
- Symptoms may be the direct result of primary GI disorders or represent other pathologies likeincreased intracranial pressure, tumor, psychiatric conditions (bulimia nervosa, anxiety), endocrine or metabolic abnormalities (diabetic ketoacidosis, hyponatremia), or iatrogenic causes (medications, toxins).
- Might also occur as the result of severe pain, myocardial infarction, sepsis, or other systemic illnesses.
- A comprehensive history and physical examination, as well as the use of various diagnostic modalities, are needed to determine the cause and its complications.
- In young women, pregnancy is an important consideration

Clinical evaluation

History:-

- Identify the onset and duration of the symptoms.
- Chronic symptoms are defined as those symptoms present for >1 month.
- If the problem is chronic, asking the patient the results of, if any, tests that have already been performed will help narrow the diagnostic possibilities.
- Frequency and interval between episodes is helpful to gauge the severity o f illness
- Timing of the episodes, such as increased number of episodes in the morning, may suggest pregnancy or a CNS cause, whereas an increased number of episodes postprandial may suggest gastroparesis or gastric outlet obstruction
- The content of the vomitus may be helpful to determine if an obstruction is present and its location.
- Esophageal disorders produce vomitus with undigested food particles.
- Bile is often associated with a small bowel obstruction.
- Large bowel obstruction often is composed of feculent material and a foul odor
- The presence or absence of associated symptoms like abdominal pain is a focal starting point.
- If pain is present, elicit its location and quality. Pain preceding the nausea and vomiting is most particularly associated with an obstructive process. Fever or possibly, diarrhea suggests gastroenteritis.

- Ask about sick contacts or ingestion of food suspicious for a foodborne illness.
- A history of recent weight loss is associated with a malignancy or psychiatric component.
- Any CNS sign, such as headache, visual changes, vertigo, or neurologic deficits, may suggest a central cause for the nausea and vomiting.
- Obtain a thorough past medical history.
- Always ask about prior abdominal surgeries because the patient is at risk for increased rate of bowel obstruction secondary to adhesion.
- Review the patient's medication list to identify a medication with the common side effect of nausea and vomiting.
- Other medications at toxic levels are known to cause nausea and vomiting. Examples include acetaminophen, salicylates, and digoxin.

The physical examination

- Should focus on determining whether a critical, life-threatening condition exists.
- Assess vital signs for hypotension and tachycardia. Observe skin turgor, mucous membrane hydration, and capillary refill to assess for dehydration.
- The abdominal examination is particularly important to assess for an emergent problem as well as to help narrow the differential diagnosis to a possible GI cause. Inspect, auscultate, and palpate the abdomen

Laboratory

- Obtain serum and urine studies to help determine the cause of the symptoms and to evaluate for complications. Most often, a complete blood count as well as electrolyte testing is part of the basic evaluation.
- Obtain a pregnancy test in a woman in childbearing years.
- Obtain liver function tests and a se**rum lipase in patients with epigastric, r**ight upper quadrant pain, or jaundice.
- Check thyroid function tests for suspected thyrotoxicosis
- Obtain specific drug levels for possible ingestions or suspected toxicity.
- A urinalysis may be beneficial in several ways. The specific gravity may be used to help determine the degree of dehydration. The presence of ketones may suggest not only dehydration, but also diabetic ketoacidosis, or hyperemesis gravidarum in a pregnant patient.

- The presence of bilirubinuria may suggest a biliary tract obstruction. Nitrites, leukocyte-esterase, bacteria, and white blood cells may indicate an upper or lower urinary tract infection. Also, red blood cells, in the proper clinical picture, may support a diagnosis of kidney stones

- Lastly, an erythrocyte sedimentation rate may be evaluated to search out an inflammatory cause
- There are two common radiographic tests ordered in the ED for evaluation of nausea and vomiting.
    - Flat and upright abdominal films are often ordered to diagnose a mechanical obstruction. Because varying degrees of partial small bowel obstruction exist, these films may be normal or show only nonspecific changes in 22% of patients.
    - CT with PO and IV contrast is superior to plain films to diagnose a mechanical obstruction and also may detect the cause of the obstruction.
- An abdominal ultrasound is helpful to evaluate a patient with associated right upper quadrant or epigastric pain to diagnose possible gallbladder, hepatic, or pancreatic pathology.

Treatment:

- Stabilize the acutely ill, vomiting patient in a fashion similar to any patient with a potentially life-threatening illness. Attention should be paid to the ABC's:
- Intubate patients who are vomiting and unable to protect their airway.
- Dehydration
    - For mild to moderate dehydration: give ORS by mouth or NGT
    - If severe dehydration or shock, give abolusof 20ml/kg NS with two further bolus if further signs of shock. Monitor response to therapy.
    - Antiemetic(metoclopramide,prochlorpromazinepromethazine,on dandestron)
- Once the patient has been stabilized, provide symptomatic relief. Definitive management will often require disease-specific treatment

11.4 Foreign body in the GI

- Foreign body (FB) ingestion is one of the most common indications for emergency endoscopy.
- It is usually accidental and more than 85-90% of ingested foreign bodieswill pass spontaneously
- Symptoms depend on several factors and in a percentage as high as 50% can be asymptomatic.
- Hematemesis, peritonitis or intoxication symptoms are less frequent but alarming symptoms.

Patient at Risk of Foreign Body Ingestion

- Children between 6 months and 6 years
- Elderly patients
- Impaired vision patients
- Mentally impaired patients
- Patient with esophageal motility disorder

Symptoms

Symptoms may also vary depending on the following:

(1) Localization of FB
(2) Size/Shape of FB
(3) Composition and content of FB
(4) Time elapse from the ingestion
(5) Onset of Complications.

Symptoms may include

Pain
Dysphagia,
Drooling
Regurgitation
Coughing
Hematemesis
Airway obstruction signs
Fever
Mediastinitis
Peritonitis
Intestinal obstruction

Diagnosis

• History is the first step of the diagnostic process. Pre-existing GI pathologies can increase the risk of foreign body impaction ,thus previous dysphagia, previous GI surgery or reflux esophagitis should be carefully recorded

• The presence of dental hardware, type and content of recent meals (like chicken or fish that may conten bones) as well as positive history of psychiatric disorders may also provide important information.

• Biplane radiography of neck, chest and abdomen is useful in case of radiopaque foreign bodies.

• CT scan may provide information in case of small FB foreign bodies, which could not be detected by radiography, still it can miss totally translucent FB.

• Endoscopy is the most common and useful procedure for the detection and localization of FB especially radiopaque objects. It also states the presence of possible

• The indication for urgent endoscopic removal is based not only on patients clinical conditions or symptoms, but also on the localization, shape and content or composition of the ingested FB.

Foreign body(FB) Localization

• Impaction occurs most often in areas where GI tract shows physiological narrowing, angulations or pathological stenosis, upper esophageal sphincterand pylorus are common sites.

• FB in the oesophagus must be removed as soon as possible if they cause obstruction and/or the patient is symptomatic. Coins or non sharp objects localized in the distal oesophagus might spontaneously progress into the gastric lumen.

• If the patient is asymptomatic, endoscopic retrieval may be delayed for 12-24 hours unless the FB is a button battery, which will be discussed later.

• Stomach FB must be removed urgently only if they are potentially harmful. If not, most FB will spontaneously transit into the duodenum and eventually will pass into the colon. Patient should be monitored with daily radiographs and clinical

evaluation. Surgical intervention is required if FB fails to progress or complications like hematemesis, melena, GI obstruction, peritonitis arise.

- Due to their dimensions some FB may not pass the pylorus. If they are still in the gastric lumen after 48-72 h endoscopic removal can be scheduled.

FB shape

- Sharp or acuminated objects should be immediately detected by radiography or endoscopy if radiography is negative.
- Those which are located in the oesophagus should be immediately removed.

- Sharp objects that reach the gastric lumen will most often pass spontaneously.

- There is an increased risk of complications (up to 35%) due to the FB transit and they should be retrieved if the endoscopic removal can be done safely.

- Gastric FB which does not pass the pylorus also needs to be removed.

Food Bolus

- Food bolus is the most common foreign body in adults.
- Food impaction is more common in elderly patients or patients with underlying oesophageal diseases (Eosinophilic esophagitis, peptic or neoplastic strictures, motility disorders)
- Radiography should be performed to identify the presence of bones which could increase the risk of perforation in case of delayed diagnosis or during removal manueuvres

Coins

- Coins are the most frequent FB in paediatric population. In 10-16% of cases ingestion can be asymptomatic

- Diagnosis should be ruled out or confirmed by biplane radiography.

- Coins impacted in the upper oesophagus may cause partial or total obstruction, drooling, pain and should be immediately removedendoscopic retrieval should be performed if coin fails to pass into the stomach after 12-24 hr.

- Coins located in the stomach do not need endoscopic removal unless they remain in the gastric lumen for more than 2 or 3 weeks

Disk Batteries

- Disk battery (DB) ingestion is worth a special consideration.

- DB ingestion can cause mucosal damage due to: (1) leakage of caustic substances (alkali); (2) direct pressure; (3) generation of an electric current which can cause liquefaction necrosis and perforation.

- There is an absolute indication to immediate endoscopic removal of DB located in the oesophagus.

- Once in the stomach, the risk of tissue damage is less high, still an endoscopic evaluation is necessary in order to point out possible lesions due to previous lodging of the battery in the oesophagus especially when the exact time of ingestion is unknown.

Section 12 Trauma management

12.1 Trauma assessement and Stablization

Trauma is a common and important contributor of morbidity, disability and mortality. Its burden is also increasing in the world, and notably so in low income countries. In trauma care the first hour is said to be the 'golden hour' as most deaths occur during this period. Hence, trauma care needs an organized approach which is called primary survey and secondary survey.

Primary Survey

Primary survey is the immediate assessment of key areas Airway, Breathing, Circulation, Disability and Exposure (ABCDE) and stabilization when there is any life threatening emergency before going to meticulous secondary survey and detailed investigations.

- Mobilize a trauma team if available or have more hands/medical personnel

Table 12. Primary survey

	Assessment	Intervention
Airway	Assess patency	Maintain airway patency (do NOT insert a naso-pharyngeal airway if there is any possibility of a fractured base of skull or nasal bone fracture) Stabilise the C-spine with in-line immobilisation and apply a semi-rigid cervical collar or other appropriate material
Breathing	Respiratory rate and effort.	Assist ventilation if required Apply O2 via non-rebreather mask to maintain SpO2 greater than 92% Tension pneumothorax – requires immediate chest decompression with a needle thoracentesis or chest tube insertion Cover with non-porous dressing taped on 3 sides only – remove immediately if respiratory status deteriorates
Circulation	Respiratory rate and effort. SpO2 Asymmetrical chest movement, unilateral decreased breath sounds, tracheal deviation ,subcutaneous emphysema Open sucking chest wound	-Control external bleeding using direct pressure/ elevation/ pressure dressing and have HCT, blood group and cross match -compression bandage, or a haemostatic dressing to control active external bleeding. -For exsanguinating extremity injury, apply a tourniquet Involve a surgeon as soon as possible IV/IO Normalize saline or ringer lactate Solution 20 mL/kg bolus to maintain SBP >90 mmHg
Disability	AVPU/GCS + Pupils Monitor GCS frequently ,RBS	patient will require endotracheal intubation by MO to protect the airway from aspiration Consider LMA insertion if GCS equals 3 and airway difficult to maintain Correct if there is glycemic abnormality.
Exposure	Undress the patient completely for thorough examination,prevent hypothermia,Log roll for complete exposure of the back and spine. .	Keep the patient warm

Adjuncts to Primary Survey

1. Do E-FAST (Extended Focused Abdominal Sonogram in Trauma) to rule out intraperitoneal,pericardial and pleural cavity collections.If needed insert foley catheter and NAsogastric tube(NGT).
2. Administer prophylactic antibiotics for all open injuries, based on the institu tion's antibiotics of choice. There is strong evidence suggesting antibiotics given within 3hours of open injury significantly lower infection risk.
3. Blood works and trauma series x-rays (lateral cervical, chest and pelvic AP x-rays) in the ED with portable x-ray should be obtained.
4. Reassess for end points of resuscitation during primary survey weather to proceeding to secondary survey or continue addressing the primary survey.

Secondary Survey

- The secondary survey is a rapid but thorough head-to-toe examination for injuries.
- Do not start the secondary survey until basic functions have been corrected in conjunction with the primary survey (airway, breathing, circulation, disability, exposure) and resuscitation has been initiated.
- The secondary survey can help set priorities for ongoing evaluation and management. Frequent reassessment of the patient's blood pressure, pulse rate, and response to interventions should continue during this period.
- Scalp lacerations can bleed profusely. Bleeding should be controlled
- Inspect the tympanic membranes for hemotympanum and repeat the pupil examination.
- Repeat the examination of the neck and thorax for any changes.
- When there is facial trauma or evidence of basilar skull fracture, insert the gastric tube through the mouth rather than the nose.
- Inspect the urinary meatus, scrotum, and perineum for the presence of blood, laceration.
- Perform a rectal examination, noting sphincter tone, gross blood, and prostatic bogginess or displacement. The rectal examination is no longer routinely performed in alert patients without evidence of pelvic or spinal injury.
- If the prostate is normal and there is no blood at the urethral meatus, a urinary catheter can be

placed in the bladder. If meatal blood is present or the prostate is displaced, which suggests a urethral injury, perform retrograde urethrography before Foley catheter.

- If there is vaginal bleeding, perform a manual and speculum examination to identify a possible vaginal laceration in the presence of a pelvic fracture.

- Evaluate the extremities for fracture and soft tissue injury, with attention to peripheral pulses. Perform a more thorough neurologic examination, carefully checking motor and sensory function.

- The most frequently missed conditions are orthopedic. Careful consideration of orthopedic extremity injuries is essential in patients with multisystem trauma.

- A tertiary survey has been recommended in patients with multisystem trauma within the first 24 hours to lessen the risk of missed injury.

- If any deterioration while doing the secondary survey return to primary survey ie ABC assessment and management

12.2 Traumatic Brain Injury

- Traumatic brain injury is brain function impairment that results from external force.

Clinical Feature

- The clinical manifestations represent a broad constellation of symptoms from brief confusion to coma, severe disability, and/or death.
- The underlying pathology ranges from temporary shifts in cellular ionic concentrations to permanent structural damage.
- Traumatic brain injury (TBI) is classified as mild, moderate, and severe based on the Glasgow Coma Scale (GCS) score.

o **Mild** (GCS 14 to 15)
o **Moderat**e TBI(GCS 9 to 13)

- Mortality rates for patients with isolated moderate TBI are <20%, but long term disability can be higher.
- 40% of patients with moderateTBI have an abnormal finding on CT scan, and 8% will require neurosurgical intervention.

o Severe TBI (GCS 3 to 8),

- Mortality rate approaches 40%, with most deaths occurring in the first 48 hour s after injury.
- Fewer than 10% of patients with severe TBI experience good recovery

Diagnosis is based on clinical evidence, pre contrast Brain CT.

Treatment of TBI

The main aim of management of traumatic brain injury is identification of surgically treatable conditions (epidural and subdural hematomas) while preventing secondary brain insults (Hypotension, Hypoxia, Hyperglycemia/Hypoglycemia, Hyperthermia)

Evaluate and manage the patient according to primary survey and secondary survey principle

o Airway

C-spine protection has to be performed and suctioning should be done frequently in comatose patients. Rapid Sequence Intubation (RSI) is modality of intubation in the following situation:

- Failure to oxygenate and ventilate
- Failure to maintain/protect airway
- GCS <=8 or Potential to deteriorate (GCS decrease by two points or more)

Facilitation of transport/treatment (increased ICP, urgent surgical intervention)

Refer to the topic on RSI (Rapid Sequence intubation)

o Breathing

Evaluate for possible presence of hemopnuemothorax and consider chest tube insertion if present

If GCS >8/15 with adequate breathing:

- Put on recovery/left lateral position
- Consider supplemental oxygen to maintain O2 saturation > 93%.
- Give oxygen via Nasal cannula at 2 to 5 liter per minute - OR –
- Use Facemask at 10 to 15liter per minute.
- Insert nasopharyngeal airway to facilitate suctioning and to support the airway patency
- Use pulseoxymeter and capnometre to measure oxygen saturation and end tidal
- CO2, record and act accordingly
- Remember C- spine precaution

If patient intubated:

- Confirm tube is in appropriate position by auscultation of the chest and ETCO2measurement of CO2
- Use minimal PEEP, not more than 5cmH2O
- Keep the ETCO2 value between 30-32cmH2O
- Keep oxygen saturation 93-100% and
- Make sure patient is well sedated and appropriate analgesia is given inorer that pressure will not elevate (the patient to fight with the machine)

o Circulation

Isolated TBI rarely causes hypotension,hence assess and control for any haemorrhage including scalp. A single episode of hypotension/hypoxia during initial resuscitation is associated with significant mortality rise.

- Adequately resuscitate the patient to get better GCS
- Keep the head/neck in neutral position and 30degree up
- Management of ICP (refer to coma management)
- Keep patients in euvolemic state and avoid dextrose IV unless hypoglycemic
- Measure glucose level at least QID and act accordingly
- Seizure prophylaxis-Seizures increase metabolic demands of brain significantly and anticonvulsants decrease incidence of early seizures. Use a 7 day course of prophylactic phenytoin or valporic acid if :
    - o GCS < 8,Cortical contusion, depressed skull fracture,
    - o Subdural, epidural ,Intracerebral hematoma, penetrating head injury
    - o Seizure within 24 h of injury
- GI prophylaxis: like providing cimetidine
- Prevent and treat fever aggressively
- Initiate enteral feeding as soon as possible if there is no contra indication
- Pain medications
    - o Even though TBI patients are not aware of their environment they feel pain
    - o Paracetamol 1gm P.O. qid plus opioids (tramadol P.O. or IV)
    - o Avoid NSAID's and ASA since they might exacerbate bleeding and cause GI ulcers

Indication of CT scanning in Head injury (according Canada CT head)

GCS < 15 at 2 hours post trauma, suspected open or depressed skull fracture, Any sign of basal skull fracture, More than one episode of vomiting, Retrograde amnesia >30 min, Dangerous mechanism (fall >3 ft. or struck as pedestrian), Age> 65 years,

TRANSFER PROTOCOL FOR TBI PATIENTS

1. Patients who can be managed in general hospitals.

Patients with:

- mild TBI
- superficial scalp lacerations.
- unstable vital signs explained by other injuries.

2. Patients who need transfer to EDs with Neurosurgical facilities or ICU are those with:

- moderate and severe TBI
- skull fractures (depressed, basal)

- penetrating head injuries
  - Patients with severity signs like raised ICP, seizure, deteriorating GCS or vital signs.

12.3 Pulmonary Trauma

By mechanism of injury it can be classified as Blunt vspenetrating

- Blunt thoracic injuries
  - o Blunt trauma produces damage by direct injury, compression, and forces of acceleration or deceleration. Patients with significant blunt injur may require intubation and mechanical ventilation and invasive procedures such as tube thoracostomy.
  - o Injuries that do not violate the pleura usually can be managed with con servatively.
- **Penetrating injuries**
  - o That violate the pleura typically result in pneumothorax, with an accompanying hemothorax in most cases. Treatment is generally supportive care after tube thoracostomy.
  - o Penetrating chest injuries in the "cardiac box" an area bounded by the sternal notch, xiphoid process, and nipples, should be presumed cardiac or great vessel injuries until proven otherwise.

General treatment

Initial Resuscitation

- Perform initial resuscitation and airway management
- If the patient is making little or no respiratory effort, consider CNS dysfunction due to head trauma, intoxication, or spinal cord injury.
- In patients with respiratory effort but with little or no air movement, suspect up per airway obstruction.
- Absent or abnormal breath sounds may indicate flail chest, hemopnuemothorax, diaphragmatic injury, or parenchymal lung damage.
- Suspect, diagnose, and treat specific life-threatening pulmonary injuries during the primary survey(pneumothorax, massive hemothorax, and open pneumothorax).

Ventilatory Support

- **Maintaining adequate oxygenation and ventilation** in the acute chest trauma pa tient is essential.
- Monitor all trauma patients by continuous noninvasive pulse oximetry to assure ad equate oxygen saturation.
- **In patients with severe chest trauma** or respiratory compromise, an arterial blood gas is helpful to monitor metabolic status and adequate oxygenation and ventilation.
- Considerations for early ventilator assistance after thoracic trauma if there is
  1. Altered mental status
  2. Hypovolemic shock

3. Multiple injuries
4. Multiple blood transfusions
5. Elderly patient
6. Pre-existing pulmonary disease
7. Respiratory rate >30–35 breaths/min despite appropriate resuscitation and pain management
8. Vital capacity <10–15 mL/kg
9. Negative inspiratory force <25–30 cm H2O
10. Metabolic acidosis with insufficient respiratory compensation is an indication for ventilator support.
11. Trauma patients who continue to exhibit impaired ventilation despite measures to relieve chest wall pain and evacuate hemopnuemothorax

Potential Causes of Cardiac Arrest or worsening of the condition after Endotracheal Intubation

1. Inadequate preoxygenation
2. Esophageal intubation
3. Intubation of the right or left main stem bronchus
4. Tension pneumothorax
5. Systemic air embolism
6. Decreased venous return due to excessive ventilatory rate or pressures
7. Vasovagal response

Life Threatening Pulmonary Injuries

Tension Pneumothorax

Diagnose tension pneumothorax clinically, before the chest x-ray is obtained.

- Perform immediate needle decompression.
  - o The most common approach to needle decompression is to introduce a 14-gauge IV needle or the available large bore IV needle and catheter into the pleural space in the mid-clavicular line just above the rib at the second intercostal space
  - o An alternative site is the fourth to fifth intercostal space at the anterior axillary line.

Massive Hemothorax

- Common causes of massive hemothorax include injury to the lung parenchyma, intercostal arteries, or internal mammary arteries.
- Each hemithorax can hold 40% of a patient's circulating blood volume. A massive hemothorax is defined in the adult as at least 1500 mL. Massive hemothorax is life threatening by three mechanisms.
- Treatment is tube thoracostomy and replacement of blood products as clinically indicated.

Open Pneumothorax

- Open pneumothorax is a communication between the pleural space and surrounding atmospheric pressure. This is sometimes referred to as a "sucking chest wound,"
- Avoid complete occlusion, as this may convert the injury into a tension pneumothorax.
- Do not insert a chest tube through the trauma wound, as it is likely to follow the missile or knife tract into the lung or diaphragm.

Systemic Air Embolism

- Systemic air embolism is an acute complication of severe chest trauma and presents with disastrous circulatory and cerebral complications.
- Patients with penetrating chest wounds who require positive-pressure ventilation are at risk for developing air embolus.
- High ventilatory pressures, especially >50 cm H2O, may force air from an injured bron chus into an adjacent injured vessel.
- Air embolus may lead to severe dysrhythmias or CNS deficits. Patients presenting with hemoptysis in the setting of penetrating chest trauma are at particular risk for this seri ous complication.
- If systemic air embolism is suspected or diagnosed,
  - o place the patient in a flat supine position with 100% oxygen applied,
  - o There is no evidence to support the theoretical benefit of theTrendelenburg (head down) position in arterial air embolism.
  - o Hyperbaric oxygen therapy, if available, helps to decrease size and increase resorption of air bubbles.
  - o Airway management of patients at risk for systemic air embolism should include ma neuvers that can selectively ventilate each lung. In unilateral lung injury,
  - o isolating and ventilating the uninjured lung can, in theory, be used to prevent systemic air embolism.
  - o In the event of circulatory collapse, treatment begins with
- cardiopulmonary resuscitation protocols
- An immediate thoracotomy to clamp the injured area of lung.
- followed by air aspiration from the heart and ascending aorta
- Open cardiac massage with clamping of the ascending aorta may help push air through the coronary arteries. Initiate cardiopulmonary bypass promptly, if available.

12.4 Abdominal Trauma

- Abdominal trauma accounts for 15% to 20% of all trauma deaths.
- Although the liver is the most frequently injured abdominal organ, the spleen is the most frequently injured intra-abdominal organ from sports accidents

Blunt Abdominal Trauma

- The most common mechanism for blunt abdominal trauma is a motor vehicle collision
- Penetrating Abdominal TraumaStab and gunshot wounds produce injury as the foreign

object passes through tissue.
- With gunshot wounds, there may be additional injury from the transmitted energy of the blast. Furthermore, gunshot wounds create secondary missiles such as fragmented bone that may increase the traumatic burden.
- Assume any penetrating injury to the lower chest, pelvis, flank, or back to have pen etrated the abdominal cavity until proven otherwise.

Clinical Features
- Clinical signs may be obvious (such as evisceration) or occult.

Physical Examination
- Inspect the abdomen for external signs of trauma (e.g., abrasions, lacerations, contusions, seatbelt marks).
- A normal-appearing abdomen does not exclude serious intra-abdominal injury. Cul len's sign and GreyTurner's sign (periumbilical and flank ecchymosis) generally repre sent delayed findings of intraperitoneal bleeding.
- making note of tenderness, tympany, or rigidity
- Reliance on physical exam alone, particularly with a worrisome mechanism of injury, may result in an unacceptably high misdiagnosis rate. As many as 45% of blunt trauma patients thought to have a benign abdomen on initial physical exam are later found to have a significant intra-abdominal injury.

Abdominal Wall Injuries
Contusions of the abdominal wall musculature may result either from a direct blow or indirectly via a sudden muscular contraction.

Solid Organ Injuries
Signs and symptoms of a solid organ injury are generally due to blood loss.
- An increase in pulse pressure may be the only clue to loss of ≤15% of total blood volume. As blood loss continues, heart and respiratory rate increase.
- Hypotension may not occur until a 30% decrease in circulating volume occurs.
- Delayed rupture can occur in splenic and hepatic injuries.
- Splenic injuries may cause referred pain into the left shoulder or arm.
- Patients with liver injuries may complain of right shoulder pain.
- Pregnancy and mononucleosis are conditions that may predispose a patient to splenic injuries.

Hollow Viscous and Mesenteric Injuries
- In blunt abdominal trauma, the incidence of blunt bowel occurs in about 5% of patients.
- Hollow viscus injuries produce symptoms from the combination of blood loss and peri toneal contamination by GI contents.
- Hemorrhage from a mesenteric injury may be minimal and not be obvious on physi cal exam. Chemical irritation of the peritoneum from gastric acid contents may produce immediate pain, although bacterial contamination of the abdominal cavity may result in delayed signs and

symptoms.

- Delays in diagnosis and operative management are associated with an increase in mor tality

Retroperitoneal Injuries

- The retroperitoneal structures discussed in this chapter include the pancreas (excluding the tail) and duodenum.
- Pancreatic injuries are present in approximately 4% of patients with abdominal trauma and are associated with significant morbidity and mortality
  - o There are no specific signs and symptoms of pancreatic injury, but mechanism of injury provides some clues to diagnosis. Pancreatic trauma often occurs from rapid deceleration. Unrestrained drivers who hit the steering column or bicyclists who fall against a handlebar are at risk for pancreatic injuries.
  - o Initial symptoms may be delayed if the injury is minor.

- **Duodenal injuries**
  - o may be relatively asymptomatic on presentation, and a small hematoma of the duodenum may go undiagnosed.
  - o As a duodenal hematoma expands, however, signs and symptoms of gastric outlet obstruction develop (abdominal pain, distention, and vomiting).
  - o Rupture occurs following high-velocity deceleration events where the intraluminal pressure of the pylorus and proximal small bowel rapidly increases. The ruptured contents are generally contained within the retroperitoneum and may be missed with studies that investigate the peritoneum exclusively. For patients with a delayed presentation, fever and leukocytosis herald the development of an abscess or sepsis.

Diaphragmatic Injuries

- o The diaphragm may spasm secondary to a direct blow to the epigastrium. The patient will experience difficulty breathing as the diaphragm loses its ability to relax and allow the lungs to expand. This process is sometimes referred to as "getting the wind knocked out." As the diaphragm relaxes, symptoms abate.
- o Diaphragmatic rupture may result from a penetrating injury or blunt force mechanism. The condition is uncommon (0.8% to 5% of patients with thoracoabdominal injury) and is almost exclusively a left-sided phenomenon.
- o Signs and symptoms are nonspecific and may be attributed to associated injuries.
- o Failure to diagnose and treat diaphragmatic rupture may lead to delayed herniation or strangulation of abdominal contents through the diaphragmatic defect.

Diagnosis

Is done with a combination of careful physical exam, attention to the mechanisms and circumstances of injury, and judicious selection of diagnostic studies is used for diagnosis.

- o Hemodynamic instability may limit the utilization of some diagnostic testing before

definitive treatment is initiated (such as laparotomy or transfer to a trauma
Ultrasonography The focused assessment with sonography for trauma (FAST) examination is a widely accepted primary diagnostic study

- The greatest benefit of FAST is the rapid identification of free intraperitoneal fluid in the hypotensive patient with blunt abdominal trauma.

Diagnosis in Penetrating Trauma
The same diagnostic tools are available for evaluation of intraperitoneal injury in the patient with penetrating trauma (CT, US, and DPL). Mandatory exploration for patients sustaining a stab wound to the abdomen has yielded unacceptably high rates of nontherapeutic laparotomy, yet physical exam alone can miss important intra-abdominal injuries.

Locally explore anterior abdominal stab wounds to assess for violation of the peritoneum. Patients with transabdominal gunshot wounds almost always have intra-abdominal injuries. In the hemodynamically stable patient with penetrating trauma, CT can help guide the surgeon for operative versus non-operative management.
Treatment

- ABCD according the ATLS protocol
- Open two large bore IV line
- Do not increase the blood pressure to normal value or maintain only to low normal
- Do CBC, blood group and Rh, consider cross matching
- Consult surgical side without delay
- Definitive management LAPAROTOMY

NONOPERATIVE MANAGEMENT OF BLUNT TRAUMA should be decided with knowledge of the trauma surgeon or general surgeon

Section 13 Common Orthopedic Injuries

13.1 Prehospital care of patients with orthopaedic injury

Effective splinting of an injured extremity reduces pain, continued bleeding, further tissue damage including damage to nerves and vessels, chance of inadvertently converting a closed fracture to an open one, fat embolism, especially in young patients with multiple long bone fractures; and facilitates transportation of patients to a health facility. Long backboard immobilization, hard cervical collar, rigid splinting, and transport in position of comfort are acceptable course of care for majority of trauma patients.

Prehospital assessment and transport

All trauma patients should undergo initial assessment:

Primary survey (immediately)

- Scene size-up: Standard precautions, scene hazards, number of patients, need for more help or equipment, mechanism of injury
- Initial assessment:
    - o general impression of the patient,
    - o level of consciousness,
    - o control cervical spine and airway,
    - o breathing
    - o circulation: pulse, skin, major bleeding scan and direct bleeding control
- Rapid trauma survey Vs. focused exam: based on mechanism of injury- if generalized or unknown---Rapid trauma survey; localized injury---focused exam

Secondary survey: while transporting

Ongoing exam: Every 5minutes for critical, every 15-30minutes for stable, each time patient moved, with each intervention, if conditions get worse

N.B. Prehospital management should not delay transportation of critically injured patients.

Pre-hospital immobilization or splinting devices and measures

Principles of orthopaedic immobilization

- For diaphyseal fractures- Immobilize the joint above and below the fracture site.
- For metaphyseal fractures- immobilization of two-third of the long bone suffices usu ally
- Limbs should be immobilized in position of comfort in pre-hospital scenarios, for pain control. While for definitive immobilization - Joints should be in functional position, to function better if the joint goes stiff.
- The bony prominences should be well padded.
- Immobilize two-third of the bone below and above when immobilizing joints.

Cervical collar

- Spinal immobilization only recommended for patients with known or suspected c-spine injury or spinal cord injury, if experienced personnel evaluation done.
- Not recommended in fully awake and communicable patients that are not intoxicated, without neck pain or tenderness, neurologically intact and without distracting injuries.
- Rigid collar and supportive blocks on spine board with straps is preferred method.
- Limits c-spine motion, and should be used till c-spine is cleared properly.

N.B. Routine collar application in all trauma patients is not necessary.

Pelvic binder/sheet

- Should be applied in hemodynamically compromised patients with mechanism of in jury suggestive of pelvic injury or evidence of pelvic injury.
- Provides stability and allows clot formation, preventing ongoing hemorrhage and trau ma induced coagulopathy.
- Should be applied circumferentially centered over the greater trochanter.

Sling and Swath - involves applying a sling, then binding the affected arm to the thorax with a gauze wrap.

- Works well for suspected injuries to the shoulder, humerus, or elbow.
- For injuries of the wrist or forearm, consider using a sling to supplement the splint, as sling helps keep the elbow at rest.

Spinal board (long spine board- LSB or Long Backboard)

- Traditionally used to provide spinal motion restriction (SMR) and rigid support during pre-hospital transport.
- Prolonged time on LSB can be deleterious by hindering respiratory effort, increase risk of aspiration or result in pressure sores. Hence, LSB should primarilyconsidered as an extrication device, whose purpose is to allow transfer to a transport stretcher.
- Appropriately applied SPM is considered in the following trauma scenarios:
    - o Spinal deformity, pain or tenderness
    - o Blunt trauma and altered level of consciousness
    - o High energy mechanism of injury with drug or alcohol intoxication
    - o Focal neurologic complain

Splints

- Some injuries warrant special splints, such as traction splints for femur shaft fractures.
- Mid-thigh trauma/ suspected femur shaft fractures: are better immobilized with traction splints, as immobilizing hip is not easily achievable. Traction component of the apparatus makes hip joint immobilization unnecessary. E.g. Thomas splint.

- If a traction device is not available, then both the hip and knee should be immobilized. One method of accomplishing this is to bind the lower limbs together, then bind the patient to a backboard from ankles to thorax with folded sheets or towels beneath the lower legs (but not the feet) to ease pressure on the heels and neurovascular status of the

limbs. A pillow can be kept in between the two limbs for comfort.

- **Wrist and ankle injuries:** Inflatable plastic splints, may be used for injuries to the ankle or wrist but sometimes are used inappropriately for fracture of the humerus or femur. Because these devices normally do not extend sufficiently proximally, they provide inadequate immobilization for such injuries. Also, overinflating the device may impair circulation. Hard board carton splints could be used in resource-limited settings as alternatives.
- N.B. If the inflatable splint cannot be dented by moderate thumb pressure, it is probably overinflated. Inflatable splints should not be applied over clothing, because wrinkles in the clothing may cause pressure sores in swollen and vulnerable tissue.

- Well-paddedmalleable aluminum splints: can be made to conform more closely to the contour of the extremity, even accommodating some degree of deformity. These splints should be removed promptly once a fracture is diagnosed or ruled out. If a fracture is confirmed, replace the splint with an alternative immobilization dressing before the patient leaves the ED, because aluminum splints may cause pressure sores even when padded. Padded Kremer wires can also be used for similar purposes.

Reducing deformity in the field

- Prehospital reduction of joint deformity for an injured extremity is not universally recommended, as injudicious manipulation may convert pure dislocation to a fracture-dislocation.
- One circumstance in which prehospital reduction of obvious fractures or dislocation maybe justified is a non- palpabledistal pulse as seen in knee dislocation with compromised peripheral circulation.

13.2 Diagnosis and general principles of management

History and Physical Examination

- Knowing the precise mechanism of injury may be the key to diagnosing some fractures or dislocations.Mechanism of injury is also the key to guide on what part of the body to focus during evaluating trauma patients. E.g. fall from a height is classically associated with calcaneus, distal tibia articular fractures (or pilon fractures), pelvic fractures and dislocation, hip area as well as spine injuries; Dashboard injury is classically associated with patella and femur fracture, knee posterior dislocation or subluxation with posterior cruciate ligament tear, simple or complex hip dislocations associated with acetabular fractures.
- **Exquisite tenderness to palpation or pain on weight bearing or passive range of motion suggests the possibility of an occult or easily missed fracture**
- General medical history should be obtained because it may have implications for further workup, the potential for complications, or ultimate prognosis for recovery of function.
- The pain of a fracture or a dislocation may be referred to another area, and hence, imaging decisions should be based not only on the chief complaint, but also on systematic

palpation, observation of subtle deformity or significant point tenderness, and mechanism of injury.

- Essential components of the examination for musculoskeletal trauma are:

a) Look for: - gross deformity of long bone and joints, abnormal mobility, fracture end crepitations, open fractures visible on necked eye are sure signs of a fracture. Loss of range of motion, and severe pain at rest suggest the presence of a dislocation or fracture near the joint.

b) Feel for: areas of bony step-off and the precise location of point tenderness. Only a meticulous palpation examination may protect the clinician from being misled by referred pain and missing a crucial diagnosis.

c) Neurovascular Assessment - Neurologic deficit is important to document early, particularly before the patient has undergone any significant manipulation or reduction maneuvers.

- Sensorimotor testing should be performed on the basis of peripheral nerve function for appendicular skeleton injures, and nerve root and dermatomal distribution for spinal and pelvic injuries.
- In the upper extremity, the radial (wrist and finger extension, median (fist), and ulnar nerves (abduction and adduction of fingers) should be tested.
- When the shoulder is anteriorly dislocated, two additional nerves, the axillary (supplying sensation to the lateral aspect of the shoulder) and the musculocutaneous (supplying sensation to the extensor aspect of the forearm), also should be checked.

- In the lower extremity, examination of the saphenous (sensory only), peroneal (dorsiflexion of foot and toes), and tibia nerves (plantar flexion of foot and toes) should be performed.

Assess vascular status early. The sooner circulatory compromise is identified and addressed, the better the chance of avoiding tissue ischemia or necrosis.

- Injuries commonly associated with vascular injury are:
    - o dislocation of the knee (tibiofemoral joint),
    - o fracture-dislocation of the ankle, and
    - o displaced supracondylar fracture of the elbow in children

Imaging

- Follow principles of fracture imaging (i.e. rule of two):
    - o Two joints: joints above and below a fracture should generally be imaged because injury/dislocation at the proximal or distal joint may coexist with longbonefractures.
    - o Two occasions: Obtain radiograph before and after reduction.
    - o Two views: obtain two orthogonal radiographs to see all possible displacements.
    - o Two sides: when doubtful about the anatomy of one side, obtain contralateral

x-ray. Children who have sustained trauma at or near a joint may need comparison studies of the opposite extremity to differentiate fracture lines from normal epiphyseal plates or ossifying growth centers.

- a negative radiologic report does not exclude significant injury. (e.g. Fracture of undisplaced femur neck or radial head, scaphoid, or some stress fractures may be undetectable on radiographs initially, even when special views are taken.)
- Delayed radiographs taken 1-2weeks after initial imaging can show evidence of fractures.
- CT or MRI may allow early diagnosis of fractures that are not radiographically evident.

Facture Characterization
Characterizing fractures makes communication among health care providers, and is based on the following;

Skin integrity: Open Vs. Closed
Location of the Fracture: proximal third, shaft/mid third or distal third;
Joint involvement: articular vs extra articular

Pattern of fracture:

Simple: spiral, oblique, transverse

Complex: wedge fragment, segmental and comminuted

Bone name: femur, humerus…

Displacement

Leg length discrepancy: Shortening or lengthening

Translation:
Anteroposterior (AP) view-shows medial or lateral translation;
Lateral view- AP translation

Angulation:

AP view - apex medial or lateral angulation;
Lat- apex anterior or posterior angulation.

Rotation: internal or external rotation.

Presence of joint dislocation: Fracture combined With Dislocation or Subluxation

Paediatric patterns: Physical injuries, Torus/buckle, plastic deformity/bowing, and

greenstick fracture patterns

Management principles

- The basic principles of fracture management are:
- Life saving measures
- Limb preserving intervention
- Function restoring: which follow the following principles
    - o Reduction
    - o Immobilization
    - o Rehabilitation
- Control pain and swelling
- Initiate measures to reduce swelling early (application of cold and elevation)
- Give analgesics as necessary.
- Withhold oral intake
- Any patient who might be a candidate for prompt surgical fixation, manipulation, or any other procedure under general anesthesia or procedural sedation should not be al lowed to eat or drink from the moment of arrival until the need for, and timing of, such a procedure has been ascertained.
- Reduce fracture deformity
- short-term benefits to reducing deformity early:
    - o alleviating pain,
    - o relieving the tension on nerves or vessels that may be stretched as they pass along the deformity,
    - o eliminating or significantly minimizing the possibility of inadvertently convert ing a closed fracture to an open one when the skin is tented by a sharp bony frag ment, and
    - o restoring circulation to a pulseless distal extremity.Reduce dislocations follow ing principles for each dislocated joint. (see "subluxation and dislocations" topic below for details)
- Initial management of open fractures- (See "Open fracture" topic for details)

13. 3: Common Orthopaedic Injuries

13.3.1 Emergency Management of Pelvic Ring Injuries

In a patient who sustained sever pelvic ring injury; exsanguinating hemorrhage is the major cause of death in the first 24hours. Hence, the management of acute pelvic ring injury is carried out as "C" component of the ATLS (attention to Circulation) .

Diagnosis

History and Physical examination - High-energy injury (e.g. motor vehicle collusions, fall from height and construction injuries are the common causes in our set up). Physical examination should focus of evaluation of mechanical and hemodynamic instability.

Concealed or obvious external perineal bleeding, asymmetric pelvis, gross lower extremity rotational deformity and leg length discrepancies are clues to unstable pelvic injuries.

The senior and experienced physician available in ED should evaluate mechanical stability. Rotational stability: can be assessed by grabbing each anterior superior iliac spine to apply internal and external rotation forces, and when found unstable apply circumferential pelvic sheet/wrap/ binder centered on the greater trochanter. Vertical stability: assistant/ examiner applies gentle traction of the leg by the ankle while keeping one hand on the ASIS to assess for vertical translation, and when found unstable apply longitudinal traction (preferably by distal femur skeletal pin traction). Pelvic stability should be done only once by experienced physician, and it is not necessary to do stability test if the patient has been x-rayed or CT scanned.

Examine the urethral meatus for blood, and when present is suggestive of possibility of urethral injury. Retrograde urethrogram rules out presence of injury.
Perform digital rectal examination noting perianal sensation, sphincter tone, gross blood, and prostatic bogginess or displacement.

Digital vaginal examination should be done to assess occult bleeding, not to miss open fractures. Speculum evaluation by gynecologist is warranted if there is vaginal bleeding.
Neurovascular examination is critical in these patients. L5 (big toe extension), S1 (Ankle plantar flexion), S2-4 nerve roots (bowel, bladder, sexual function) are commonly affected.
Imaging - obtain AP pelvis as part of ATLS trauma series imaging. Inlet and outlet pelvic views as well as CT scan should only be obtained if the patient is hemodynamically stable. Presence of pelvic gas on the CT scan is suggestive of internal open pelvic fracture. Imaging should not delay urgent intervention required.

Initial Management

- Follow principles of ATLS for hemodynamic resuscitation. (refer to ATLS section)
- Apply pelvic packing by unwrapped sterile roll bandage; ends of roll bandage can be tied together if one roll bandage is not sufficient to control externally bleeding pelvic wound.
- Apply circumferential sheet centered over the greater trochanter for rotationally unstable pelvis.
- Apply longitudinal traction (distal femur skeletal traction) for vertically unstable pelvis.
- Suprapubiccystostomy might be indicated if transurethral catheter could not be passed. When indicated, put it far away from area of symphysis pubis and pelvic injury side as putting cystostomy with in close distance to the symphysis compromises future definitive surgical intervention for the pelvic/acetabulum injury. (Insert it as close to the umbilicus as possible)

- Appropriate antibiotic coverage for open wounds started as soon as possible. (Initiating antibiotics within 3hours has been shown to significantly lower infection risk in open fractures).
- Emergency surgical interventions include: pre-peritoneal pelvic packing (or angio-embolization by intervention radiologist if available) for non-responding bleeders, laparotomy for viscous injury
- DVT prophylaxis according to the center's protocol shall be initiated after 12-24hours and once the patient is hemodynamically stable. Aspirin 81mg po daily or Heparin are safe anticoagulants depending on institutions protocol after 24hours of the injury.

Definitive treatment

Centers with expertise and the set up to deal with these complex injuries should be communicated for prompt definitive management referral. These patients should be transferred to these centers once properly resuscitated and as soon as feasible. Delay in referral could make the definitive surgery more complex; and it increases pelvic sheet related complications when sheet is kept on for more than 24-48hours.

13.3.2 Acute Compartment Syndrome (ACS)

Compartment syndrome occurs when increased pressure within a limited space compromises the circulation and function of the tissues within that space.

The normal pressure within a compartment is <10 mm Hg. Anoxia and muscle death occurs with prolonged elevated pressure. Tissue pressures exceeding 30mm Hg have traditionally been thought to be toxic if left untreated for several hours.

Absolute as well as "Delta pressure" (difference between diastolic and intra compartmental pressure) measurement of 30 mm Hg is commonly used to diagnose acute compartment syndrome.

Etiologies

- Orthopedic (Tibial fractures, Forearm fractures)
- Vascular Ischemic-reperfusion injury Hemorrhage
- Iatrogenic (Vascular puncture in anticoagulated patients, IV/intra-arterial drug injection, Constrictive casts)
- Soft tissue injury (Prolonged limb compression, Crush injury, Burns)
- Hematologic (Hemophilia, adverse effects of anticoagulants (warfarin))

Diagnosis

History and Physical Examination– diagnosis is mainly clinical.

- Pain out of proportion and aggravation by passive stretching of muscles in the compartment in question are the most sensitive (and often the only) clinical find

ings before the onset of ischemic dysfunction in the nerves and muscles.

- The six P's (pain, paresthesia, pallor, pulselessness, paralysis, poikilothermic) are signs and symptoms of florid syndrome.
- Compartment pressure measurement is only necessary in doubtful scenarios, as in borderline cases and patients who are not mentally clear.

Treatment

Is tailored to avoid sequels of Volkmann's ischemic contracture and gangrenes.

a) Insure adequate airway protection and oxygenation
b) Keep the affected limb at the level of the heart, to avoid further ischemia by elevation.
c) Maintain circulation aggressively since reduction in MAP and significantly compromise tissue perfusion.
d) Remove circumferential dressings, split and bivalve circular casts as soon as ACS is suspected.
e)Urgent surgical consultation and fasciotomy is the main stay of treatment.

13.3.3 Dysvascular LIMB/ Ischemic Limb

Diagnosis

High index of suspicion is the key to timely diagnosis by noting whether any hard signs are present (i.e. active hemorrhage; large, expanding or pulsatile hematoma; bruit or thrill over wound; absent palpable pulse distally; and signs of distal ischemia (the 6P's).

Arteriography- immediate arteriography is warranted in extremity trauma patient with any hard sign. If cannot be done, consult vascular surgeon before any further investigation to avoid delay.

There is no clear role for non-invasive testing in initial evaluation (Doppler pressure or signals, duplex ultrasound).

Treatment

Tissues do not tolerate warm ischemia time of more than 6 hours; hence any injury associated with vascular compromise should be addressed as soon as possible on an emergency basis.

a) Promptly correct gross deformity as soon as these patients are encountered, and attempt reduction of dislocations before any other further investigation and imaging.
b) In some cases, reducing a deformity by means of longitudinal traction is all that is necessary to restore circulation or nerve function.
c) Direct pressure dressing, elevation, ligation or clamping the bleeding vessel are the preferred measure of hemorrhage control. To minimize ischemic insult and

reperfusion syndrome, avoid tourniquet application except for exsanguinating bleedings.
d) Commercially available plastic instrumental shunts (plastic IV tubing (IV line set) are alternatives in resource limited settings, after irrigation with heparinized saline before use) can be used as vascular shunts. The ends of the tubing are placed in the distal and proximal segments of the injured artery, secured by a silk tied around the vessel over the shunt and then also tied directly on the shunt itself to prevent dislodgement. This is an option especially when patients need to be referred long distances.
e) Refer/consult vascular surgeon promptly without delay for any reason, especially if circulation remain compromised after reduction of the joint or gross fracture deformity.
f) When imaging is not possible, immediate surgical exploration of the vessel at risk must be done in presence of hard signs.

N.B

- Gross deformity correction or reduction of dislocation should not be delayed for radiographic reasons when peripheral circulation is compromised.
- Surgical intervention should not be delayed for purpose of investigation, or imaging.
- Immediate surgery without imaging may be undertaken when clear vascular injury picture is present (i.e. absent pulse, cold ischemic foot/hand) in high risk injury patters such as supracondylar humerus fractures and posterior knee dislocations.

13.3.4 Open Fractures

An open fracture is a break in continuity of a bone associated with a communication between the fracture site or fracture hematoma and epithelial lines surface (i.e. the environment, gastrointestinal lining as seen in cases of pelvic fractures).

Diagnosis

History and Physical Examination– Presence of wound overlying a fractured bone is the clue for further evaluation of the wound. This warrants further evaluation of the wound and the fracture. Basedon the severity, Gustilo and Anderson graded open fractures into three:

a) Grade I: <1cm wound, low energy injury, simple fracture pattern, clean wound. Usually the communication occurs from within by fracture ends.
b) Grade II: Between 1-10cm wounds, moderate energy injury, moderately comminuted fractur Section 14 Procedural Sedation and Analgesiae, with moderate contamination of the wound.
c) Grade III: >10cm wound, high-energy injury, comminuted fracture, with a grossly contaminated wound. Further sub categorized as
d) Grade IIIA: Adequate soft tissue coverage of the bone possible
e) Grade IIIB: Bare bone, with no coverage to stripped periosteal lining of the bone
f) Grade IIIC: Circulation impaired, with major vascular injury without repair it leaves the limb viability is at stake.

N.B. High velocity bullet injuries, farmyard injuries are grade III irrespective of other criteria, and should be managed as such.
Imaging – patients should be sent for imaging only if hemodynamically stable and after initial dose of prophylactic antibiotics. Follow principles of fracture imaging.

Initial Management
Amputations, infection, and non-unions are the major complications of open fracture; hence the management is geared towards preventing these.

a) Sterile gauze dressing of wounds.
b) Temporarily immobilize the fracture site following principles of fracture immobilization. (e.g. well-padded Kremer wire for tibia fractures, Thomas splint for femur fractures...)
c) Prophylactic antibiotics: first generation cephalosporin (e.g. Cefazolin) for Gustilo-Anderson types I and II. (Ciprofloxacin P.O. or Cloxacillin IV can be considered as alternatives); Gentamycin should be added for Gustillo-Anderson type III open fractures. Add crystalline penicillin or Metronidazole for gross and farmyard contaminations.
d) Tetanus Toxoid after skin test, update vaccination accordingly.
e) ED (OPD) irrigation of wounds should be practiced only for gross contaminations; proper irrigation and debridement (I &D) should be done in the operating room.

N.B It should be emphasized that administration of prophylactic antibiotics within three hours of the injury is the single most important factor to minimize risk of infection. Hence, make sure the patient receives antibiotic before sending him for imaging.

Definitive Management
Patients should be promptly refer/consulted to a center where there is an orthopaedic or general surgeon who is well versed with management of these injuries.

13.3.5 Subluxation and Dislocation

Definition
Subluxation is a condition in which the articular surfaces of a joint are nonconcentric, with some part of the articular surface remaining in contact.
Dislocation is when the articular surfaces of the bones that normally meet at the joint are completely out of contact from one another.

Diagnosis
History and Physical Examination–mechanism of injury usually depicts the type and location of a dislocation (e.g. dashboard injuries are associated with posterior knee and hip dislocations; fall on outstretched hand can cause elbow dislocation, extreme abduction

and external rotation can cause anterior shoulder dislocation…). Acute dislocations are usually very painful, and present with classic deformities. (e.g. posterior hip dislocation presents with hip in flexion, adduction and internal rotation; while anterior shoulder dislocation presents with their arm held by the normal side, while elbow is in extension, and arm in slight abduction…)

Imaging –orthogonal x-ray views of the affected joint are usually diagnostic, and should be obtained before attempt of reduction. Obtaining two orthogonal views might not be feasible because of pain, so alternative views should be considered when diagnosis is not clear. Dislocations with vascular compromise and recurrent dislocations can be reduction before obtaining radiographs. Proper orthogonal radiographs will show direction of dislocation; weather the dislocation is simple or complex (associated with fracture)…

Initial Management

The urgency of reducing a dislocation is to restore neurologic or circulatory compromise; the longerthat a joint has been dislocated, the more difficult it may be to reduce and the less table the reduction is likely to be by non-operative measures; and to avoid avascular necrosis of the affected part of the joint. (e.g. AVN of femoral head)and well as cartilage necrosis.

a) Follow ATLS principles if a dislocation occurs following high-energy injury.
b) Administer appropriate ED sedation and relaxation before any attempt of re duction (refer/consult "Procedural sedation" topic for details).
c) Follow specific joint reduction maneuvers. Physician working in emergency department should be familiar with reduction maneuvers of specific joint dislo cations. If not experienced or comfortable promptly refer/consult the patient to a nearby health facility where there is expertise. Knee dislocations warrant an attempted reduction because of high risk of associated neurovascular insult.
d) After care involves:
    - Assessment for clinical stability of reduction
    - Assess and document neurovascular status before and after reduction
    - Obtain post reduction orthogonal view radiography to assess congru ency of reduction and look for possible associated fractures.
    - If dislocation is simple, and when reduction is stable and concentric, proper immobilization of the reduced joint should be done following principles of immobilization. (for shoulder- body strap/arm sling; el bow- long arm posterior gutter; hip- knee immobilizer in extension; knee- external fixator might be the best option and hence better if referred to Orthopaedic surgeon)

N.B.Do not delay reduction in limbs with obvious vascular Impairment, correct gross deformity with compromised vascular circulation warrant attempted reduction before radiographs.

Definitive management

Promptly refer/consult dislocations,

a) which are complex
b) irreducible
c) with vascular compromise
d) found unstable after reduction
e) non-concentric reductions

Recurrent dislocations also warrant referral on non-urgent basis.

Section 14 Procedural Sedation and Analgesia

14.1 Procedural sedation and analgesia (PSA)

Procedural Sedation: is administration of different type of sedative and/or analgesic agents to induce a state that allows the patient to tolerate stressful and/or painful procedures while maintaining cardio respiratory function, and airway protective reflexes.

- Minimal sedation and analgesia: mild anxiolysis or pain control. Pa tients respond normally to verbal commands. Example: changing burns dressings
- Moderate sedation and analgesia: patients are sleepy but also aroused by voice or touch. Example: direct current cardioversion
- Deep sedation and analgesia: patients require painful stimuli to evoke a purposeful response. Airway or ventilator support (or both) may be needed. Example: major joint reduction

Choice of depth of sedation and analgesia depends on the type of procedure, extent of painful intervention, general condition of patients

Indications for procedural sedation: Incision and drainage of large abscesses, wound debridement, reduction of fractures, dislocations and prolapsed, viscera (hernias), chest tube insertion, repair of complicated lacerations, especially in children, diagnostic studies (US, CT or MRI)in children and irritable patients, Lumbar puncture in non-cooperative individuals, cardioversion, removal of embedded foreign bodies, painful or anxiety-inducing procedures (e.g., pelvic, examination in a young rape victim)

Preparation for PSA

- Have a corner or a room for PSA, equip at least with ABCD resuscitation materials (ambubag, different size of airways, face mask, and suction machine and tubes)
- Monitoring equipment (cardiac monitor, pulseoximeter, capnogram)
- Oxygen, oxygen administration devices
- Emergency drugs such as adrenalin and atropine
- Have always open IV line with normal saline
- Secure antidotes for severe side effects of benzodiazepines and narcot ics
- Determine the desired depth of sedation.
- Select drugs that provide adequate analgesia and use drugs that is very familiar to you and available in your seating
- Duration of the procedure also influences the choice of drugs and the route of delivery.
- Individual response for different drugs, even when the dose is calculat ed according to their weight is different. Careful titration produces the optimal response.

- Intravenous (IV) administration is preferable, since repeated oral doses are unpredictable and usually slow to onset, and repeated intramuscular (IM) injections are not acceptable.

Patient assessment and selection

- Always determine whether the patient is able to tolerate PSA or not
- Always assess the airway for possibility of presence of difficult airway
- Candidates for procedural sedation in the ED or outside the operating suite mus t fall into either ASA class II or I.
    - o ASA-I Normally healthy patient. The pathologic process for which the procedure is to be performed is localized and not a systemic disturbance.
    - o ASA- II Mild systemic disease under control (e.g., controlled HPN, DM, and asthma).
    - o ASA- III Severe systemic disease from any cause (e.g. poorly controlled HPN, DM, asthma)
    - o ASA- IV Severe systemic disease that is a constant life-threat, not always correctable by the operative procedure.
    - o ASA- V Moribund patient who is not expected to survive without the operation
- Patients with higher ASA classifications and difficult airways are better served in a more controlled environment, such as the operating room (OR) or intensive care unit (ICU)

Procedural sedation (PSA) needs having NPO: Before giving a drug for sedation and analgesia you have to make sure patients, last hour of feeding and always consider emergency trauma and non-trauma patients are considered as full stomach due to stress therefore proper preparation is mandatory

Children up to 6 months old:

- 2 hours, for clear liquids, 4 hours fast for breast milk and other formula

Children 6 months–3 years:

- 3 hours fast for clear liquids, and 6 hours fast for milk and solid food

Children3 years old and above :

- 3 hours fast for clear liquids, and 6–8 hours fast for milk, solid food

Adults:

- 2 hours fast for clear liquids and 6 hours fast time for solids

Drug selection

Ideal drug for PSA have to have:

- Predictable action
- Rapid onset
- Easily adjustable
- Short recovery time
- If possible have both amnestic and analgesic properties
- Also have minimal hemodynamic effects and
- Noor minimal respiratory depression

Widely used drugs for sedation and analgesia:

- Combination of sedatives and analgesics are recommended for pain full proce dures and
- if the patient is going to have non-painful procedure such as for calming of ir ritable adult patients and children for CT, MRI, and US only amnesic or sedative drugs can be used without combining of analgesic drugs

o Midazolam0.05-0.1mg/kg plus fentanyl 1-3mcg/kg/IV, onset of action 1-3min, duration of action up to one hr. midazolam is potent amnesic and muscle relaxant and fentanyl analgesic. Has to be given slowly titrated upon the response of the patient and desired level of sedation. Side effects respiratory depression and hypotension, which mandates close and con tinuous monitoring.

o Valium0.1mg/kg plus Fentanyl 1–3 mcg/kg IV. Onset of action within 1–2 min, potent rapidly acting analgesic. Hemodynamically stable. But may cause res piratory depression, bradycardia, and apnea. Push and flush slowly; monitor closely. Dose can be titrated up to the desired level of sedation and analgesia. Remember opioids and benzodiazepines has synergistic effect

o Valium 0.1mg/kg and Morphine 0.01MG/kg titrate slowly according patient's response and desired level of sedation and analgesia. Onset of action 3-5min, duration of action up to 30min. important side effects includes respiratory de pression, bradycardia, and hypotension. The recovery time for the combination of benzodiazepines and fentanyl/Morphine is generally longer than that for keta mine, and propanol or etomidate, and can be used for log procedures

o Ketamine 0.5-1.0mg/kg and propanol 1-2mg/kg, IV slowly. Onset rapid dura tion of action for10-15min. give additional boluses with titration, if the proce dure requires longer time

o Ketamine 0.5mg/kg and Etomidate 0.1-0.3mg/kg. Onset of action rapid, dura tion of action 10-15min. titrate additional dose according the patients response. May cause myoclonic muscle jerk and pain on the injection site

Antidotes

o Flumazenil antidote for benzodiazepines side effects. Adult: 0.2 mg IV, onset of action 1–2 min, duration of action 20 min Used for the reversal of benzodiaz epine-induced prolonged sedation. Administer slowly. Pediatric: 0.01 mg/kg IV. Duration of action shorter than midazolam. Repeat doses may require following initial reversal

o Naloxone Adult: 5–10mcg/kg IV/ET/IM, onset within 1–2 min, duration of ac tion 30-60min. Titrate to desired effect. Upon the or reversing the effects of opioids

14.2 Acute Pain Management

Pain management is an essential component of care for any patient. However, it is often overlooked and many patients are found to have moderate and severe pain and are suffering needlessly.

What is Pain?

Acute pain is a result of tissue damage, which can result from an injury, infection or other issues such as degenerative illness or a metabolic condition. Pain is both a sensory and emotional experience and previous experience can effect a patient's perception.

Effect of pain

- o Acute pain causes a stress response to the body, which causes the blood pressure and heart rate to rise.
- o Increase systematic vascular resistance,
- o Impair immune function and altered release of pituitary, neuroendocrine and other hormones. This response could limit recovery to injury.
- o Untreated pain can lead to acute sensitivity on subsequent occasions.

Pain Assessment – 5th vital sign

Pain assessment is considered as the 5th vital sign and should be a part of the patient's initial assessment. Pain should be assessed and reassessed throughout the patient's stay. There are various pain assessment tools that can be used (see Appendices).

Pain Management

Immediately after diagnosis, active pain management should be initiated. The aim is to manage the patient's pain and to prevent any physical and psychological responses to acute pain.

General Principles of Acute Pain Relief

- Assess and determine the severity of pain before treatment and after treatment according the drugs half-life or action time
- Identify any allergic history to analgesic drugs
- Reassure and explain on the treatment you are going to give
- Use analgesic drugs you are familiar on their actions, side effects and complica tions
- Analgesic selection is based on the pathophysiological mechanism of the pain and its severity.
- Non-opioids are preferred for mild pain.
- Opioids may be required for moderate to severe pain.
- Combined treatment with opioids and non-opioids can be appropriate; non-opioids can be used to reduce the opioid dose requirement.
- Non-pharmacological treatments may be helpful but should not preclude phar macological treatment.

- The first 24-48hrs treating pain in regular base is effective and reduces high dose requirement (avoid pain management on PRN base)
- Do not combine drugs with same class, dose by the clock, do not wait for pain to recur
- Mild pain is considered 1-3acording the numeric pain scaling method
- Non-pharmacologic pain treatment such as psychological reassurance, physical treatment has to be considered
- NSAIDs reduce the flow of blood to the kidneys and impair function of the kidneys. The impairment is most likely to occur in patients who already have impaired function of the kidney or congestive heart failure, and use of NSAIDs in these patients should be cautious. People who are allergic to other NSAIDs, including aspirin, should not use ibuprofen. Individuals with asthma are more likely to experience allergic reactions to ibuprofen and other NSAIDs. Blocks the enzyme that makes prostaglandins (cyclooxygenase), resulting in, ulceration can occur without abdominal pain due to the bleeding, and the only signs or symp toms of an ulcer are: black, tarry stools, weakness, and dizziness upon standing (orthostatic hypotension)
- Ibuprofen-the usual adult dose is 200 or 400 mg every 4 to 6 hours. Side effects and contra indication on the NSAID above
- Paracetamol- Mild to moderate analgesic and antipyretic. Side effects; Liver damage in overdose. Dose: Neonates: 10-15mg/kg 6-hourly (5mg/kg if jaun diced) Max 60mg/kg/d; Pediatric- PO/PR: 20mg/kg 6-hourly Rectal loading dose 30-40mg/kg; Adult PO:0. 5-1g QID slow IV:0. 5-1g QID
- Tramadol- Opioid analgesic thought to have less respiratory depression, con stipation, euphoria, or abuse potential than other opioids. Have opioid and non-opioid mechanisms of action. Side effects: Nausea, dizziness, dry mouth. In creased side effects in conjunction with other opioids. Dose: adult, PO: 50-100mg 4-hourly. Slow IV/IM: 50-100mg 4-hourly, pediatric 1-2mg/kg 6-hourly
- Morphine is an opioid pain medication. Morphine is used to treat moderate to severe pain. Short-acting formulations are taken as needed for pain. The extend ed-release form of this medicine is for around-the-clock treatment of pain. This form of morphine is not for use on an as-needed basis for pain. Oral, Sublingual, or Buccal: 5 to 30 mg every 3 to 4 hours as needed IM or subcutaneous: 2.5 to 20 mg every 3 to 4 hours as needed IV: 4 to 15 mg every 3 to 4 hours as needed. Give very slowly over 4 to 5 minutes.
- For combination of drugs and step wise treatment see WHO Analgesic Ladder for pain management below.

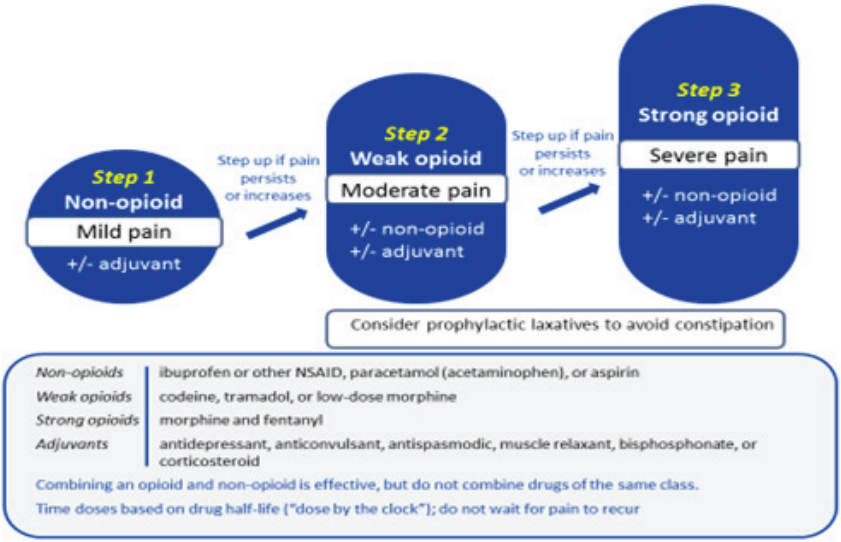


Figure 8. WHO Analgesic Ladder for pain management

Section 15 Neurologic Emergencies

15.1 Coma and Altered Mental Status

Dementia is failure of the content portions of consciousness with relatively preserved alerting functions.
Delirium is arousal system dysfunction with the content of consciousness affected as well.
Coma is failure of both arousal and content functions.
Etiology

Table 13. Etiology of Coma and Altered Mental Status

Primary Neurologic (usually with focal signs)	Systemic
Stroke Seizure (postictal, status, non-convulsive) Infection: meningoencephalitis, abscess Epidural/subdural hematoma Concussion Hydrocephalus Complicated migraine Venous thrombosis Transient global amnesia CNS vasculitis TTP	Cardiac: severe CHF, HTN encephalopathy Pulmonary:TPaO2,cPaCO2 GI: liver failure, constipation, Wilson's Renal: uremia, hyponatremia and hypernatremia Endocrine:Tglc, DKA, HHNS,cCa, hypothyroidism or hyperthyroidism, Addisonian crisis ID: pneumonia, UTI, sepsis Hypothermia and hyperthermia Medications (espec. opiates & sedatives) Alcohol & toxins

Initial Evaluation
Primary Survey; ABCDE, if there is sign of trauma A is with C-spine protection.
Secondary Survey

History; typically from others): previous or recent illnesses, including underlyingdementia or psychiatric disorders; head trauma; meds, drug or alcohol use

P/E; evaluate for signs of trauma, skin lesions, stigmata of liver disease, astrexsis, nuchal rigidity
Neurologic Examination;

- Calculate GCS, Observation for response to stimuli, papilledema, spontaneous move ments
- Pupil size & reactivity:
    - o Pin point; opiates, pontine lesions, organophosphates;
    - o Mid position & fixed; midbrain lesion;

o Fixed & dilated; severe anoxic encephalopathy, herniation
- ICP: head ache, vomiting, HTN,THR, papilledema, unilateral dilated pupil
- Motor response in the extremities to noxious stimuli, noting purposeful vs. posturing; (decerebrate arms extended; decorticate arms flexed; both with legs extended Deep tendon reflexes, Babinski response)

Initial treatment

- Control airway, monitor vital signs, IV access
- Immobilization of C-spine if concern for cervical trauma
- Thiamine (100 mg IV) prior to or with dextrose to prevent exacerbation. of Wer nicke's encephalopathy
- Dextrose (50 g IV push) (~ 2 ampules of 40% dextrose)
- Naloxone 0.01mg/kg if opiates suspected; flumazenil 0.2 mg IV if benzos sus pected
- If concern for ICP and herniation:
- Elevate head of bed 30 degree;
- Mannitol IV 1gm/kg loading then 0.25 g/kg q6h;
- Hyperventilation;
- Dexamethasone; if mass lesion with edema
- Consider emergent surgical decompression
- If patient is agitated/combative and endangering oneself or others sedation may be needed.
- Haloperidol at a dose of 5 to 10 milligrams PO, IM, or
- Reduce dosing 1 to 2 milligrams in the elderly.
- Repeat at 20- to 30-minute intervals as needed.
- Benzodiazepines such as diazepam, 0.5 to 2.0 milligrams PO, IM, or IV, may be used in combination with haloperidol in doses of 1 to 2 milligrams, with the dose varying widely depending on the age and size of the patient and the degree of agitation

Diagnostic studies

- Head CT; radiographs to r/o C-spine fracture; CXR to r/o PNA (in elderly)
- Blood film to r/o malaria, relapsing fever
- Laboratory: electrolytes, BUN, Cr, ABG, LFTs, CBC, PT, PTT, NH3, tox screen, TSH, U/A
- Lumbar puncture to r/o meningitis
- EEG to r/o non-convulsive seizures

15.2 Stroke

Stroke is a sudden focal neurological deficit attributable to a specific vascular territory secondary to obstruction or bleeding. It is characterized with rapidly developing signs of focal or global disturbance of cerebral functions lasting more than 24 hours.

Transient Ischaemic Attacks (TIA) is a clinical syndrome characterized by an acute loss of focal cerebral or ocular function with symptoms lasting less than 24 hours.

Cerebral infarction from thrombosis or embolism accounts for 85% of strokes. Cerebral haemorrhage account for the remainder.

Ischemic stroke: The mechanism of cerebral infarcts may be due to insitu thrombosis or embolism. Embolism may be from a cardiac source or artery to artery emboli. Causes are due to risk factors for atherosclerosis. Systemic hypo perfusion also may contribute.

Cerebral haemorrhage: Primary Intracerebral Haemorrhage is associated with rupture of Charcot-Bouchard aneurysms in patients who usually have hypertension.

Subarachnoid haemorrhage (SAH) is a haemorrhage from a cerebral blood vessel, aneurysm or vascular malformation into the subarachnoid space. It is characterized by sudden onset of headache, and vomiting, with or without loss of consciousness.

Assessment:
The first step is to conduct immediate assessment and stabilization of ABCD.It will be followed with subsequent assessment in order to diagnosis through careful history taking, examination and investigation. The assessment and investigation should include identification of possible underlying cardiovascular causes and the initial neurological assessment should document the localization of the likely cerebral area affected.

Investigation of acute stroke
Immediate RBS to exclude hypoglycaemia, RFT/LFT, Lipid profile/A, CXR, ECG should be done.If cardiac source of embolism is likely e.g. atrial fibrillation an echocardiogram is needed.
CT scan should be requested immediately in all patients or at least within 24 hrs .If the diagnosis of stroke is in doubt after CT scan, MRI should be considered

INDICATIONS FOR IMMEDIATE CT SCANNING

- Coma or reducing/fluctuating conscious level.
- features of increased ICP or SAH like severe headache, papilloedema, neck stiffness or fever
- Likelihood of important non-stroke diagnosis (e.g. subdural hematoma, SAH).

- A known bleeding tendency, patient on or requiring anticoagulants.
- Patient eligible for thrombolysis.
- Unusual presentation? Basilar artery thrombosis.
- Unexpected deterioration be considered to identify intracranial complica tions, e.g hydrocephalus or hemorrhagic transformation

Treatment:

General Measures: focuses on stabilization of ABCD, careful nursing care and early physiotherapy.

- Check for blockage of airways and clear if needed with suctioning or oral airway. In altered state of consciousness positioning is also important to prevent aspira tion and rise in ICP.
- Check breathing status and give oxygen if oxygenation is inadequate or patient is in distress. Intubate patient if necessary-in significant distress not corrected with appropriate non-invasive oxygenation or GCS is low.
- Check pulse/BP and put the patient on cardiac monitor and start isotonic fluids: intravenously if unable to feed and maintain electrolytes within normal range. Hypotension should be handled with fluid bolus, pressor agents and treatment of primary cause of shock like hypovolemia or sepsis.
- Maintain good glycaemic control: hypoglycemia or hyperglycemia has to be managed accordingly.RBS should be in between 90-140mg/dl.
- Manage intracranial pressure if there is a rise and anticonvulsant for seizure activity
- Reduce pyrexia.
- treatment of associated illnesses e.g pneumonia
- DVT prophylaxis and early physical and speech support.
- Check for swallowing in order to prevent aspiration
- admit a patient to ICU or an acute stroke unit

Nursing Care

- Coma care: nursing care to prevent bedsores, hypostatic pneumonia, contractures, exposure keratitis .Nutritional support to maintain adequate caloric intake.
- Patients should be mobilized as soon as possible.
- Change position at least every two hours: for the prevention of bedsores.
- Bladder and bowel care: catheterize if patient is incontinent or unconscious.
- Feeding: insert NG tube for patients who have swallowing problems or are at risk of aspiration (e.g. pseudobulbar palsy) and those unconscious.

Specific treatment

Ischemic stroke: management includes handling high BP, antiplatelet/anthrobotic medications, thrombolytic and statins.

Box H. BP Goals in management of Stroke

- **Ischemic Stroke: cerebral perfusion is depending on higher BP and do not lower BP unless it is very severe like >220/120.When there is extracranial or intracranial stenosis- require a slower reduction in blood pressure**
- **Ischemic stroke with hypertensive encephalopathy, IHD, Aortic dissection: treat it with BP target is 140/90 but it is harmful to the brain and use short act ing drugs like esmolol.**
- **Ischemic stroke with thrombolytic plan:BP should be reduced below 185/110mmHG.**
- **Hemorrhagic stroke-lower SBP up to 140mmHg if well tolerated and it re duces hematoma expansion**
- **Sub arachnoid hemorrhage (SAH)-BP goal is to keep SBP between 140-160mmHG and initiate drugs if it is >170mmHG.**

When treatment is indicated- blood pressure lowering by approximately 15-20% during the first 24 hours after stroke onset and it is suggested that antihypertensive medications should be restarted at approximately 24 hours after stroke onset in patients with preexisting hypertension who are neurologically stable

If pharmacologic therapy is given, intravenous labetalol is generally the drug of choice. labetalol (10-20 mg IV for 1-2 min) should is given, and may be repeated or doubled every 10 minutes to a maximum dose of 300 mgIn our setup, since it is not available, reduction of BP cautiously with the available agents like Hydralazine 10mg iv every 20-30 minutes with careful monitoring of BP.

Antiplatelet and Anti-thrombotic treatment:

1. Aspirin (300 mg) Po, enterally or suppository should be given as soon as possible after the onset of stroke symptoms once a diagnosis of primary haemorrhage has been excluded. Then aspirin (150- 300 mg) should be continued indefinitely. Following thrombolysis it should be delayed for 24 hours.

2. Early anticoagulation: may precipitate secondary haemorrhage and it is advisable to start anticoagulation only after one to two weeks in large artery territorial infarcts.

Thrombolysis improves outcome of patients with cerebral ischemia, though with a high-risk treatment and should only be administered by personnel trained in its use, in a center equipped to investigate and monitor patients appropriately

Intracranial hemorrhage:

The approach to blood pressure management must take into account the potential benefits and risks of BP lowering:

- Reducing the BP in patients with either ICH or SAH may be beneficial by mini mizing further bleeding and continued vascular damage

- Patients with an intracranial hemorrhage due to ICH or SAH- have increased intracranial pressure (ICP) due to blood within the cranium.

- Intracerebral hemorrhage: blood pressure can be lowered by at least 15 percent without causing ischemia in the perihemorrhage tissue

- Subarachnoid hemorrhage: The optimal therapy of hypertension in SAH is not clear. Elevated blood pressure can worsen an SAH and lowering the blood pres sure may decrease the risk of rebleeding from an untreated aneurysm. However, reducing the BP can result in ischemia due to an inadequate cerebral perfusion pressure and the concurrent cerebral vasospasm.

- The patient's cognitive status may be a useful guide. If the patient is alert, then CPP is adequate and lowering the blood pressure may decrease the risk of re-rupture.

- In contrast, antihypertensive therapy is generally withheld following a SAH in those with a severely impaired level of consciousness since the impairment may be due to increased ICP with a reduced CPP.

- In patients with SAH who do not have marked hypertension volume expansion is recommended in an attempt to minimize cerebral ischemia. Avoidance of vol ume depletion is also desirable to prevent hyponatremia from possible cerebral salt wasting.

Recommendations: With ICH, intravenous labetalol or nicardipine should be given if the systolic pressure is above 170 mmHg.

- The goal in this setting is to maintain the systolic pressure between 140 and 160 mmHg and to carefully monitor the patient for signs of cerebral hypoperfusion induced by the fall in blood pressure

- Nimodipine 60 milligrams PO every 4 hours to decrease risk of vasospasm and should be initiated within 96hrs and phenytoin for seizure prophylaxis

- In SAH, in the absence of ICP measurement, antihypertensive therapy is often withheld unless there is a severe elevation in blood pressure because of concern about cerebral ischemia and the frequent compensatory nature of acute hyper tension.
- Correct any identifiable coagulopathy with fresh frozen plasma, vitamin K, pro tamine, or platelet transfusions.

15.3 Seizure and Status Epilepticus

Seizuredefined as abnormal, paroxysmal, excessive discharge of CNS neurons; occurs in5–10% of the population; clinical manifestations can range from dramatic to subtle

Epilepsyis recurrent seizures due to an underlying cause; 0.5–1.0% of population

Generalized seizures (involves brain diffusely)

- Tonic-clonic(grand mal): tonic phase (10–20 sec) with contraction of muscles (causing expiratory moan, cyanosis, pooling of secretions, tongue biting) clonic phase (30 sec) with intermittent relaxing and tensing of muscles
- Absence(petit mal): transient lapse of consciousness w/o loss of postural tone
- Myoclonic(infantile spasms & juvenile myoclonic epilepsy): sudden, brief contraction

Partial or focal seizures (involves discrete areas, implies a focal, structural lesion)

- Simple: without impairment of consciousness; may be motor, sensory, or autonomic
- Complex: with impairment of consciousness automatisms
- Partial with secondary generalization: starts ad focal and becomes diffuse

Etiologies

- Alcohol withdrawal, illicit drugs, medications
- Brain tumor or penetrating trauma
- Cerebrovascular disease, including subdural hematomas, hypertensive encephalopathy
- Degenerative disorders of the CNS (e.g., Alzheimer's)
- Electrolyte (hyponatremia) & other metabolic (e.g., uremia, liver failure , hypoglycemia
- Infectious (meningitis, encephalitis, malaria etc)

Patients with Active Seizures

Typically, little is required during the course of an active seizure other than supportive and *patient protective measures.*

- If possible, turn the patient to the side to reduce the risk of aspiration
- Once the attack subsides, clear the airway.

- Suction and airway adjuncts should be readily available.

- It is not necessary or recommended to give IV anticonvulsant medications dur ing the course of an uncomplicated seizure. Any unnecessary sedation at this point will complicate the evaluation and result in a prolonged decrease in level of consciousness.

- Seizures that fail to abate after 5 minutes are considered status epilepticus and require more aggressive medical interventions (see "Status Epilepticus" section, below).Patients with a History of Seizures Proper management of a patient with a well-documented seizure disorder who presents after one or more seizures depends on the particular circumstances of the case.

- Identify and correct potential precipitants that may lower the seizure threshold. Many seizures occur because of failure to take anticonvulsant medication as prescribed(send sample for serum anticonvulsant level if available).

- If anticonvulsant levels are very low, a loading dose is appropriate, and the regular regimen can be restarted or adjusted.

- If anticonvulsant levels are adequate and the patient has had a single attack, specific treatment may not be needed and consultation with the patient's primary care physician or neurologist may be sufficient.

- If the maintenance dose is increased, ensure follow-up within 1 to 3 days.

Patients with a First Unprovoked Seizure

- In general, patients with a first unprovoked seizure who have a normal neuro logic examination, no acute or chronic medical comorbidities, normal diag nostic testing including non-contrast head CT, and normal mental status can safely be discharged from the ED without initiating medication.
- Patients with provoked (secondary) seizures due to an identifiable underlying condition often require admission and should generally be treated to minimize seizure recurrence.
    - o The ideal initial antiepileptic regimen is a single-drug therapy that controls seizures with minimum toxicity.

o If treatment is initiated, drug selection is based on the type of seizure

- **Generalized tonic-clonicseizure**
    - First line- Valporic acid, lamotigine, topiramate
    - Alternatives- Phenytoin, carbamazepine, Phenobarbital
- **Focal seizure**
    - First line- carbamazepine, phenytoin, lamotrigine
    - Alternative- valporic acid, Phenobarbital

- **Absence, myoclonic, tonic, clonic**
  - First line- valporic acid

*Treatment of Status Epilepicus*The goal of treatment is seizure control as soon as possible and within 30 minutes of presentation

Examination;

- Identification of potential causes;
  - o Checking the airway, breathing, and circulation; and treatment all begin simultaneously.
  - o Direct a focused history and physical examination toward possible causes and subsequent injuries..
  - o Place the patient on oxygen, a cardiac monitor, a pulse oximeter, and end-tidal capnography.
  - o Establish large-bore IV access and determine a bedside glucose
  - o Thiamine 100mg IV
  - o Glucose 50gm Iv
  - o Diazepam 0.2 mg/kg or adult dose 5-10mg Iv stat/ lorazepam 0.1- 0.15mg over 1-2min and repeat dose if no response after 5 min
  - o Phenytoin 20mg/kg IV@ 50mg/min or for phenytoin 20mg/kg @ mg/min
  - o If seizure continues repeat phenytoin 7-10mg/kg@ 50mg/kg or for phenytoin 7-10mg/kg @150mg/min
  - o In established status epilepticus, consider endotracheal intubation for airway protection, oxygenation, and ventilation.
  - o If a paralytic agent is used for intubation, use a short-acting agent so as not to mask ongoing seizure activity.
  - o Arrange for continuous EEG monitoring as soon as possible after paralytic agents have been used.
- Initial laboratory evaluation includes blood glucose, a metabolic panel including calcium and magnesium, lactate, and if appropriate, a pregnancy test, a toxicology screen, and anticonvulsant levels.
- Monitor temperature continuously, and treat hyperthermia with passive cooling.
- Place a urinary catheter to monitor urine output, and insert a nasogastric tube to help prevent aspiration.
- If toxic ingestion is suspected as the cause of seizures, proceed with GI decontamination (as appropriate).
- Do not attempt lumbar puncture during status epilepticus. If bacterial meningitis or encephalitis is suspected, start empiric antibiotic or antiviral therapy.

Refractory Status Epilepticus

Refractory status epilepticus is defined as persistent seizure activity despite the IV administration of adequate amounts of two antiepileptic agents and usually exceeds 60 minutes.

Pharmacologic Treatment

- o Propofol infusion at typical rates of 2 to 10 milligrams/kg/h and titrated up to effect seizure cessation.
  - o At higher doses (>40 milligrams/kg/h), patients are at increased risk for hemo dynamic instability, including hypotension, as well as Propofolinfusion syndrome.
  - oMidazolam infusion at 0.05 to 0.4 milligram/kg/h and is titrated up to seizure cessation.
  - o Phenobarbital (up to 20 milligrams/kg IV) or pentobarbital may be considered as third-line drugs in patients whose seizures are not controlled despite full loading doses of benzodiazepines and other agents.
  - o Ketamine may bolus dose of 0.5 to 4.5 milligrams/kg or as an infusion up to 5 milligrams/kg/h.

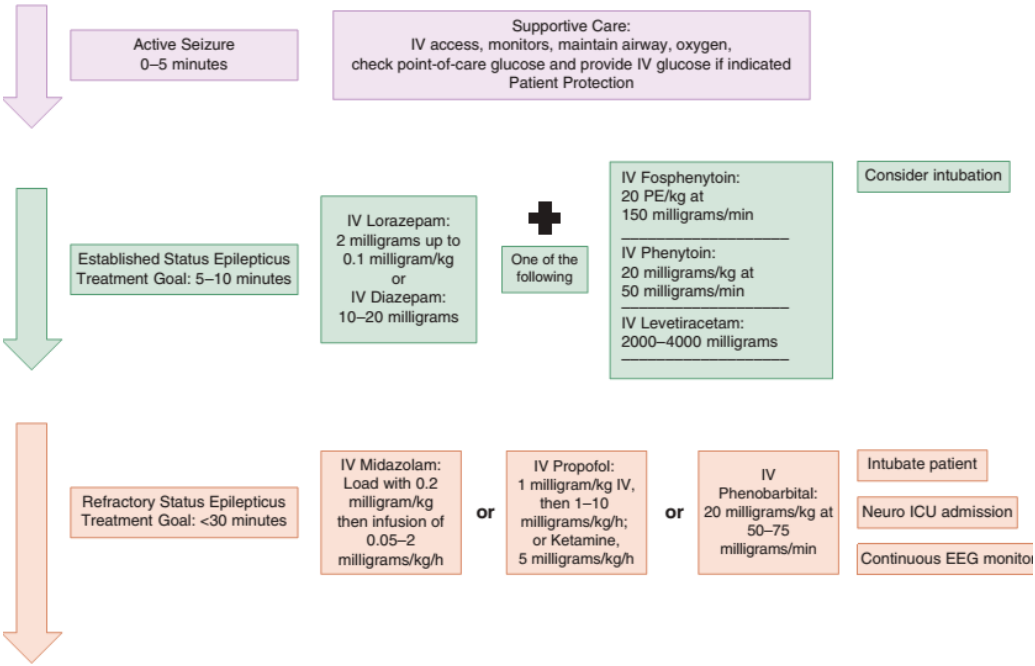


Figure 9 Treatment of status epilepticus

Section 16 General Management of Poisoned Patients

- Poisoning-Poisoning occurs when exposure to a substance adversely af fects the function of any system within an organism. The setting of exposure may be occupational, environmental, recreational, medicinal or suicidal. Poisoning may result from varied portals of entry including inhalation, ingestion, cutaneous exposure and injection.
- Try Identify the substance
- Obtaining the original toxic substance and the amount
- Containers found near or on patient
- Through accurate history

Approach to poisoned patient at the bedside

- ABC
- Oxygen, monitors, IV access
- Full set of vitals including O2 sat
- Gather history and collateral information
- Check glucose
- Physical exam: skin, cardio, resp, GI, neuro
- Disability : GCS, pupils
- Drugs: Consider universal antidotes
- Decontamination
- Draw Labs

*Contact poison centre *

- Specific antidotes and supportive care

Decontamination: Terminating topical exposures

- Contaminated clothing should be removed and safely disposed of.
- Wash skin and hair with soap and water while wearing gloves.
- Eye exposures: irrigate with copious amounts of water or saline for 10-15 minutes.

Gastric Lavage:

- *Indication:* ingestion of large amounts of tablets and capsules with a high inherent toxicity with 2 hrs.

- **Method:**
  - Insert a large bore or gastric tube(32-40 F)
  - Place patient head down, left lateral decubitus
  - Aspirate fluid from stomach prior to fluid lavage
  - Install water or saline into stomach: 200-300ml
  - Aspirate fluid back & repeat till aspirate clears

- Risks: pulmonary aspiration, epistaxis, laryngospasm, hypoxia, sinus bradycardia & mechanical injury

Contraindications: patients with decreased LOC, unprotected airway, ingestion of corrosives, volatile substances and GI hemorrhage.

Early post-ingestion activated charcoal:

- *Minimizes* systemic absorption from the GIT
- *Indication:* consider use within 1hr of ingestion of the poisonous substance.
- *Method:*
    - *Route:* oral or instill via NG tube
    - *Adult dose:* 50-100g (1-1.5g/kg) as a slurry in 400-800ml water

Shake vigorously to ensure adequate dispersion

- Risks: no systemic effects, but may induce vomiting, constipation, diarrhea
- Contraindicated: decreased level of consciousness or unprotected airway.
- Avoid (no value): strong acids, alkali, corrosives, heavy metals, cyanides, lithium, hydrocarbons (paraffin), methanol and ethylene glycol.

Multi-dose activated charcoal

- Enhance elimination of drugs already absorbed
- Interrupting enterohepatic circulation.
- Indication: ingestion of large doses of Carbamazepine, dapsone, phenobarbitone, quinine, theophylline, Salicylates, sustained release formulations.
- *Method:* after first dose of charcoal, follow up dose of 25g every 2hrs or 50g every 4hrs,

Until clinical conditions and lab parameters improve.

Contraindications: diminished bowel sounds, proven ileus or small bowel obstruction.

Enhanced Elimination

- Whole Bowel Irrigation: Instillation of large volumes of polyethylene glycol in osmotically balanced electrolyte solutions promotes rapid, mechanical elimination of ingested toxins.
- Urinaryalkalinisation: Infusion of sodium bicarbonate to raise urinary pH to enhance clearance of toxins excreted by kidneys

- 1-2 mEq/kg NaHCO3 IV push
- 3 ampules of NaHCO3 in 850 cc of D5W at 1.5X maintenance fluid rate
- Target urinary pH 7.5-8.5
- Monitor electrolytes
- **Extracorporeal Removal:**
- **Hemodialysis**
- Less effective when toxin has large volume of distribution (>1 L/kg), has large molecular weight, or highly protein bound.
- Acetone, Barbiturates, Bromide, Ethanol, Ethylene glycol, Salicylates (Aspirin), Lithium
- **Peritoneal Dialysis**
- Alcohols, long acting salicylates, Lithium.

Investigations

- Serum electrolytes
- Blood glucose
- Renal & liver function tests
- Urinalysis
- Coagulation studies
- ECG
- Arterial blood gases
- Osmolality

Plasma Concentrations: Are essential for: CO poisoning, Paracetamol, Salicylates, theophyline, TCA, Lithium, digoxin and metals.

Universal antidotes:

- Thiamine
- Oxygen
- Naloxone
- Glucose

Table 14. The most common toxidromes with presentation and examples

Toxidromes	Common Causes	Sign & Symptom
Anticholinregic	Antihistamines, Atropine, Scopalamine	Sedation, Hallucinations, Mydriasis, Dry skin, Dry mucous Membrane, Decreased bowel sounds & urinary retention
Sedative – Hypnotics	Baributrates, Benzodiazepins	Sedation, normal pupils, respiratory depression
Sympathomimetic	Cocaine, Amphetamine	Agitation, mydriasis, tachycardia, hypertension, hyperthermia, diaphoresis
Cholinergic	Organophosphates& Carbametes	Altered mentation, seizure, mitosis, lacrimation, urination, diaphoresis, bronchospasm, bronchorrhea, vomiting, bradycardia
Opoids	Meperidine, Codeine	Sedation, meiosis, decreases bowel sounds, respiration depression

Table 15: List of Common antidotes

Agent	antidote
Paracetamol	N- acetylcysteine, 140 mg/kg PO, then 70 mg/kg q4h for up to 17 do 150 mg/kg IV load over 1 hr with 50 mg/kg over 4 hr followed by 100 mg/kg over 16 hr
Cardiac glycosides	Digoxin immune Fab, 10–20 vials if patient in ventricular fibrillation
Cyanide	Hydroxycobalamin, 5 mg in 100 ml of NS over 15 min
Iron	Deferoxamine , 15 mg/kg/hr IV
Lead	EDTA, 75 mg/kg/day by continuous infusion
Opiods	Naloxone 2 mg; less to avoid narcotic withdrawal, more if inadequa response
Benzodiazepins	Flumazenil, 0.2 mg, then 0.3 mg, then 0.5 mg, up to 5 mg

16.1 Organophosphate poisoning management

Organophosphates	Atropine, Test dose, 1–2 mg IV in adults, titrate to drying of pulmonary secretions
TCA	Bicarbonate, 44–88 mEq in adults, 1–2 mEq/kg
Anticholinergics	Physostigmine 1–2 mg IV in adults, for anticholinergic delirium, seizures, or dysrhythmia
β- blockers	Glucagon, 5–10 mg in adults, then infusion of same dose per hour
Sulfonylureas	Octreotide, 50 µg SC q12h, 5–10 µg/kg/24 hr IV

16.1 Organophosphate poisoning management

Organophosphate compounds are the insecticides most commonly associated with systemic illness.

Organophosphate compounds bind irreversibly to acetylcholinesterase, thus inactivating the enzyme through the process of phosphorylation andacetylcholine at nerve synapses and neuromuscular junctions, resulting in overstimulation of acetylcholine receptors

Clinical presentation:

S	Salivation
L	Lacrimation
U	Urinary incontinence
D	Defecation
G	GI pain
E	Emesis
D	Defecation
U	Urination
M	Muscle weakness, miosis
B	Bradycardia, bronchorrhea, bronchospasm
E	Emesis
L	Lacrimation
S	Salivation
"Killer B's"	Bradycardia, bronchorrhea, bronchospasm

Four distinct syndromes can occur:

- acute poisoning,
- intermediate syndrome,
- chronic toxicity,
- organophosphate induced delayed neuropathy

Of these intermediate syndromesis the most feared one withparalysis of neck flexor muscles, muscles innervated by the cranial nerves, proximal limb muscles, and respiratory muscles; respiratory support may be needed and occurs within 1 to 5 days of initial symptoms and in up to 40% of poisonings.

Table 16: Acute organophosphate poisoning severity grading table (Tintinallis 8thed)

Severity	Butyrylcholinesterase Activity (% normal) Measured from Plasma	Acetylcholinesterase Activity (% normal) Measured from Red Blood Cells	Clinical Features	Typical Initial Atropine Amount to Control Symptoms
Mild	40–50	50–90	Lightheadedness, nausea, headache, dyspnea Lacrimation, rhinorrhea, salivation, diaphoresis	<2 milligrams IV/IM
Moderate	10–40	10–50	Restless, confusion, vomiting, diarrhea, drowsiness Autonomic instability: bradycardia or tachycardia, hypotension or hypertension, miosis or mydriasis Muscle fasciculations Bronchorrhea and bronchospasm	2–10 milligrams IV/IM
Severe	<10	<10	Coma, seizures, flaccid paralysis, urinary or fecal incontinence, respiratory arrest	>10 milligrams IV/IM

*The correlation between cholinesterase activity, clinical symptoms, and atropine dose is inconsistent, and treatment should be guided predominately by clinical symptoms.

Diagnosis and treatment are based on history(people may bring substance itself) and the presence of a suggestive toxidromes; laboratory cholinesterase assays and reference laboratory testing for specific compounds take time and have limitations, and waiting for results delays administration of potentially life-saving therapy.

Meiosis (papillary constriction) and muscle fasciculation are the most reliable signs of organophosphate toxicity and help in diagnosis.

Treatment:

- airway control
- intensive respiratory support
- general supportive measures, decontamination
- prevention of absorption, and the administration of antidotes

- During intubation since Succinylcholine is metabolized by plasma butylcho linesterase, and therefore, prolonged paralysis may result, a non-depolarizing agent should be used when neuromuscular blockade is needed.

Table 17: Treatment approaches summary of acute organophosphate poisoning with medication dosages (Tintinallis 8thed)

Decontamination	Protective clothing must be worn to prevent secondary poisoning of healthcare workers. Handle and dispose of all clothes as hazardous waste. Wash patient with soap and water. Handle and dispose of water runoff as hazardous waste.
Monitoring	Cardiac monitor, pulse oximeter, 100% oxygen.
Gastric lavage	No proven benefit (see text).
Activated charcoal	No proven benefit (see text).
Urinary alkalinization	No proven benefit (see text).
Atropine	1–3 milligrams IV in an adult or 0.01–0.04 milligram/kg IV (but never <0.1 milligram per dose) in children. Repeat every 5 min until tracheobronchial secretions attenuate. Followed by continuous infusion to maintain the anticholinergic state. Dose varies from 0.4 to 4 milligrams/h IV infusion in adults.
Pralidoxime	No proven benefit (see text). 1–2 grams for adults or 20–40 milligrams/kg IV (up to 1 gram) in children, mixed with normal saline and infused over 5–10 min. Followed by continuous infusion: 500 milligrams/h in adults or 5–10 milligrams/kg per hour in children.
Seizures	Benzodiazepines IV.

- Pralidoxime is single most important treatment in nicotinic effect of or ganophosphate poisoning and used for intermediate syndrome within 48 hours which is lifesaving.

Disposition and follow up:

Minimal exposures may require only decontamination and 6 to 8 hours of observation in the ED to detect delayed effects.

- Admission to the intensive care unit is necessary for significant poisonings.
- Most patients respond to Pralidoxime therapy with an increase in acetylcho linesterase levels within 48 hours.
- The end point of therapy is determined by the absence of signs and symptoms on withholding Pralidoxime therapy.

- Death from organophosphate poisoning usually occurs in 24 hours in untreated patients, usually from respiratory failure secondary to paralysis of respiratory muscles, neurologic depression, or bronchorrhea.

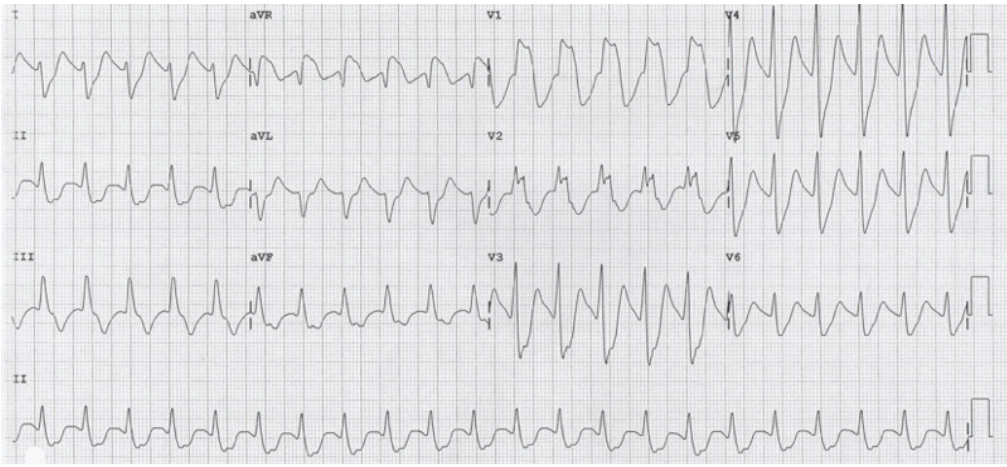
16.2 Cyclic antidepressants poisoning

- **Consists of:**
  - amitriptyline,clomipramine,desipramine,dothiepin,doxepin,imipramine,lofepramine,nortipytline,protripytline,trimipramine,amoxapine and maprtiline.Life-threatening ingestion at >10mg/kg, and fatalities at >1gm for adults (therapeutic dose 5-10mg/kg/day).
  - Prolonged GI absorption due to anticholinergic effects.

Mechanisms of action; involves Na+ channel, K+ efflux, Alpha1 adrenoceptor, Muscarinic & Histamine receptors blockade. Inhibition of NE & 5HT reuptake as well as GABA antagonism.

- Clinical features : Prolonged QRS (>100msec), Right-axis deviation in terminal 40msec, Decreased inotropy, Arrhythmias – ectopy, heart blocks, VT, Brugada pattern (ST elevation in V1-2), Hypotension. (all from Na+-channel blockade).
- Prolonged QT interval, prone to torsade de Points (anticholinergic toxidromes)

Na+-channel blockade-ECG



- QRS >100 ms in lead II, Right axis deviation of the terminal QRS, Terminal Rwave >3 mm in aVR, R/S ratio >0.7 in aVR.

- Others: Wide pulse pressure, Mycoses, bradycardia, agitated delirium and seizures (anticholinergic Toxidromes).

Management of overdoses

1. ABCs

- Monitored setting, full set of vital signs, serial ECG
- Airway equipment available
- Intubate if low LOC or respiratory depression
- Hyperventilate to pH of 7.50-7.55
- Provide high-flow oxygen
- Start IV line If hypotensive, give 30cc/kg NS bolus
- Consider vasopressors if necessary (norepinephrine)
- Do NOT treat hypertension

2. Universal antidotes

"TONG"

- Thiamine
- Oxygen
- Naloxone (opioid antagonist)
- Glucose

3. GI Decontamination

- If within 1hr of ingestion but also later if severe toxicity
- Gastric lavage (intubated patient)
- Activated charcoal -1g/kg (50g) through NG tube

4. Further history and physical exam

- Confirm ingestion
- Toxidrome-Anticholinergic

5. Others

- Alkalization and Na loading for dysrhythmia by:
  - Bolus of 1-2amps NaHCO3 until BP improves andQRS narrows, or serum pH reaches 7.50-7.551-2mEq/kg of 8.4% NaHCO3 (50mEq(50ml)/amp)
  - Maintenance rate with 3amps of NaHCO3 in 1L ofD5W (for at least 4-6hrs)
  - Hypertonic NACI (3%,7%)
  - Also consider hyperventilation (palm/cerebral edema)
- Treat seizures with:
  - IV benzodiazepines(diazepam 5-10mg)
  - May use phenobarbital, Propofol
  - Avoid phenytoin (Na+-channel blocker)

6. Definitive management

- Observation for >6hrs
- Admission to hospital (to ICU if comatose and/or cardiac dysrhythmias ,hypo tension)

- Psychiatric consultation for suicidal attempts.

16.3 Carbon monoxide poisoning

- Carbon monoxide (CO) is an odor less, tasteless, colorless, non-irritating gas formed by HC combustion. Atmospheric concentration is generally below 0.001%, but higher in urban areas and closed environments.

- CO poisoning is one of the leading causes of poisoning deaths. Smoke inhalation is responsible for most inadvertent cases of CO Poisonings.

    - CO binds to Hb with much higher affinity than oxygen (240) forms carboxy hemoglobin and tissue oxygenation is impaired. Clinical findings are harshly variable and largely nonspecific, headache, malaise, nausea and dizziness com monly seen. We should also ask about loss of consciousness.
    - Patients may manifest symptoms ranging from mild confusion to coma.
    - Acute myocardial injury is common among CO poisoned patients & is associated with increased mortality.

Diagnosis: is based on a compatible Hx and physical exam in addition with an elevated carboxyhemoglobin level measured by cooximetry of a blood gas sample

Management: The most important interventions in the management are removal from the source and administration of oxygen by face mask.

- Comatose patients should be intubated & mechanically ventilated using 100% oxygen
- Hyperbaric oxygen: involves exposing patients to 100% oxygen under supra atmospheric conditions to decrease the half-life of carboxyhemoglobin, used in potentially severe intoxication.

16.4: 2-4 D poisoning:

- These compounds are herbicides used as weed killers on lawns and grain crops.
- Metabolic pathway or mechanism of toxicity is unknown,
- Toxicity results from dermal contact, inhalation or ingestion.
- Local exposure leads to eye and mucous membrane irritation, ingestion leads to nausea, vomiting and diarrhea.
- Hypotension, tachycardia, dysrhythmias and also tachypnea form pulmonary edema are seen, muscle toxicity manifests as muscle tenderness, fasciculation, myotonia and rhabdomyolysis.
- Diagnosis is based on history of exposure
- Treatment is supportive, including decontamination, and respiratory support.

16.5: Caustic ingestions

Caustics are substances that cause both functional and histologic damage on contact with body surface.

C**ommon caustic compounds**- Alkali-NaoH (industrial chemicals), KoH/lithium hydroxide (batteries), sodium triphosphate (detergents), sodium hypochlorite (bleach)

Acids- sulfuric acid(automobile batteries, fertilizer), hydrochloric acid(metal cleaning, chemical production), hydrofluoric acids(rust remover, petroleum industry), nitric acids (fertilizers), phosphoric acid (metal cleaners, disinfectants)

Alkali injuries- deep tissue injury called liquefaction necrosis.

- The most common house hold alkali is bleach (3%-6%) sodium hypochlorite solution-rarely cause beyond grade 1 esophageal injuries.

Acids- produce coagulation necrosis and forms protective eschar.

Clinical features

History: type and amount of caustic ingested, presence of co-ingestants

Examination:

- Priority to ABCS
- Oral mucosa, eyes, skin on face or chest (dribble burns)
- Drooling, stridor, wheezes, dysphonia
- epigastric tenderness

Lab: Complete blood count and blood group, electrolyte panel, liver enzymes, coagulation profile

- ECG
- Radiology: CXR (erect), CT-Scan (noncontract) – viscus perforation, ingestion of strong caustic
- contrast esophagogram, abdominal sonography
- Endoscopy: between 12-24 hrs post ingestion.

Treatment

- The first priority is airway maintenance
- Intubation(direct laryngoscopy or fibrotic endoscopy)
- Prepare surgical back up
    - Dexamethasone 10mg IV in adults , 0.6 mg/kg(max 10 mg) in airway edema
- Large bore IV access and fluid resuscitation with crystalloids
- Standard decontamination precautions
- Immediate and adequate irrigation of the eyes and skin

- Dilutional therapy (beyond 30 min more harmful than beneficial), water but with restricted amount to avoid gastric dilatation.

- Inducing emesis contraindicated
- Activated charcoal relatively contraindicated
- Gastric emptying with gentle suctioning of nasogastric tube can be attempted for acidic ingestions<30 min and no spontaneous emesis but contraindicated for alkalis
    - Except: zinc chloride(ZnCl2) and mercury chloride(HgCl2) -- severe systemic toxicity, aggressive decontamination with gentle nasogastric tube aspiration and administration of activated charcoal
- Systemic steroids: cannot be recommended as standard care
-]Prophylactic antibiotics: no evidence to support the use
- Analgesia
- Antacids(PPIs, H2 antagonists)
- Surgery: if peritoneal signs or free intraperitoneal air, esophageal perforation

    - Stricture: esophageal dilatation

Disposition and Follow up based on endoscopic grading

Grade1 injuries- discharge after endoscopy if they tolerate oral fluid and food

Grade 2A injuries- admission to ward

Grade 28 and 3 injuries-intensive care unit recommended

16.6: Barbiturates

- From the group of `sedative -hypnotics` that lower excitement and induce sleep.
- Most commonly used are amobarbital, butabarbital, pentobarbital &phenobarbital which is long acting.
- Have a narrow therapeutic index
- Clinical features f acute intoxication includes slurred speech, in coordination, unsteady gait and impaired attention or memory.
- Severe overdose leads to coma
- Most common vital sign abnormalities are hypothermia, respiratory depression and hypotension.

Treatment

- Airway stabilization
- Intubation in severe overdose before gastrointestional decontamination.
- Volume expansion by rapid infusion of 1 – 2 L of isotonic fluid
- Gastric lavage if within 1hr of ingestion.
- activated charcoal in multiple doses to reduce serum concentration
- Forced diuresis with fluid loading and diuretic therapy is most effective for Phenobarbital
- Hemodialysis and hemoperfusion are used to maximize barbiturate elimination reserved for patients who are deteriorating despite institution of aggressive supportive care.

Section 17 Environmental Emergencies

17.1: Hypothermia

A state of low body temperature, specifically a low CORE temperature (< 35° C or < 95° F).

- Primary Hypothermia: Direct environmental exposure to cold, No underlying medical condition

- Secondary Hypothermia: Complication of any medical condition: Such as sepsis, hypothyroid, adrenal insufficiency, hypoglycemia, Ethanol or carbon monoxide intoxication, etc.

- Classification of Hypothermia: Based on CoreTemperature severity and associated clinical features.

- Mild hypothermia: 32 to35°C (90 to 95°F) : Confusion, slurred speech, ataxia "cold diuresis", increased heart, rate, hyperventilation, shivering.

- Moderate hypothermia: 28 to 32°C (82 to 90°F):Central nervous system depression: lethargy, hallucinations, decreased reflexes, decreased renal blood flow, decreased heart rate, arrhythmias: A. fib, junctional bradycardia , hypoventilation, decreased/loss of shivering

- Severe hypothermia: < 28°C (82°F): coma, areflexia, oliguria, pulmonary edema, apnea, hypotension, bradycardia, ventricular arrhythmias, cardiac arrest/ asystole, pseudo rigor mortis"à may appear dead.

- Treatment for Hypothermia:
  - Remove patient from cold environment
  - Remove wet garments
  - Protect against further heat loss and wind chill
  - Maintain patient in horizontal position
  - Avoid rough handling
  - Monitor the core temperature
  - Monitor the cardiac rhythm.
  - Active Internal (Core) Rewarming: Warm/humidified Oxygen, Warm IV fluids (42° C), Padded warm packs to major areas (neck, axilla, groin), Pleural/Peritoneal Irrigation with warm saline, GI/bladder irriga tion with warm saline,extracorporeal rewarming.

17.2: Hyperthermia

Hyperthermia iselevated core bodytemperature above 38.5° Celsius.

Mechanisms of Heat Illness:

- **Heat Cramps:** inadequate replacement of salt from loss through sweating à low Naà muscle cramps

- **Heat Tetany:** hyperventilation àrespiratory alkalosisàparesthesia

- **Heat Exhaustion:** salt water depletion from sweat loss: Normal to mildly elevated temp, extreme fatigue, headache, nausea, vomiting, diarrhea , increased HR + RR, mental status intact.

- **Heat Stroke:** heat stress or endogenous heat production from exertion: Increased temp > 40.5deg Celsius, HA, N/V, weakness, CNS symptoms àataxia, confusion, seizures, Hot + Dry skin, Increased HR, renal failure, etc.

Hyperthermia Treatment

- **Heat Cramps:** Oral or IV fluid, Electrolyte Replacement, Rest in cool environment with gradual return to normal environment.

- **Heat Tetany:** Remove from heat, Rebreathe expired air (bag-breathing),R/o electrolyte abnormalities.

- **Heat Exhaustion:** Bed rest in cool environment, Rapid IV fluid replacement, Electrolyte replacement, Labs (CBC, glucose, lytes, etc).

- **Heat Stroke:** Immediate rapid cooling à to <40deg C within 30 min from beginning of cooling, Remove patient from heat source, Undress patient completely, Apply tepid water (notice) to the skin andfan, apply ice packs to axillae and groin, immersion in ice water also effective but may precipitate seizures.

17.3: Snake Bites

The clinical manifestations of patients bitten by snakes are very different depending on species of snakes.

When patients are bitten by snakes, it may just cause a wound (dry bite), may produce bacteria and spores, tetanus and secondary infections or may inject venoms with systemic effects.

There are three main groups of envenomations:

- Cytotoxic: causes tissue loss & swelling, may lead to compartment syndrome.
- Neurotoxic: causes descending type of paralysis, patient may die of respiratory depression.
- Hematotoxic: Causes DIC, uncontrolled bleeding and organ dysfunction.
- Or a combination of the above groups

Knowledge of toxicity profiles of local snake species is vital.

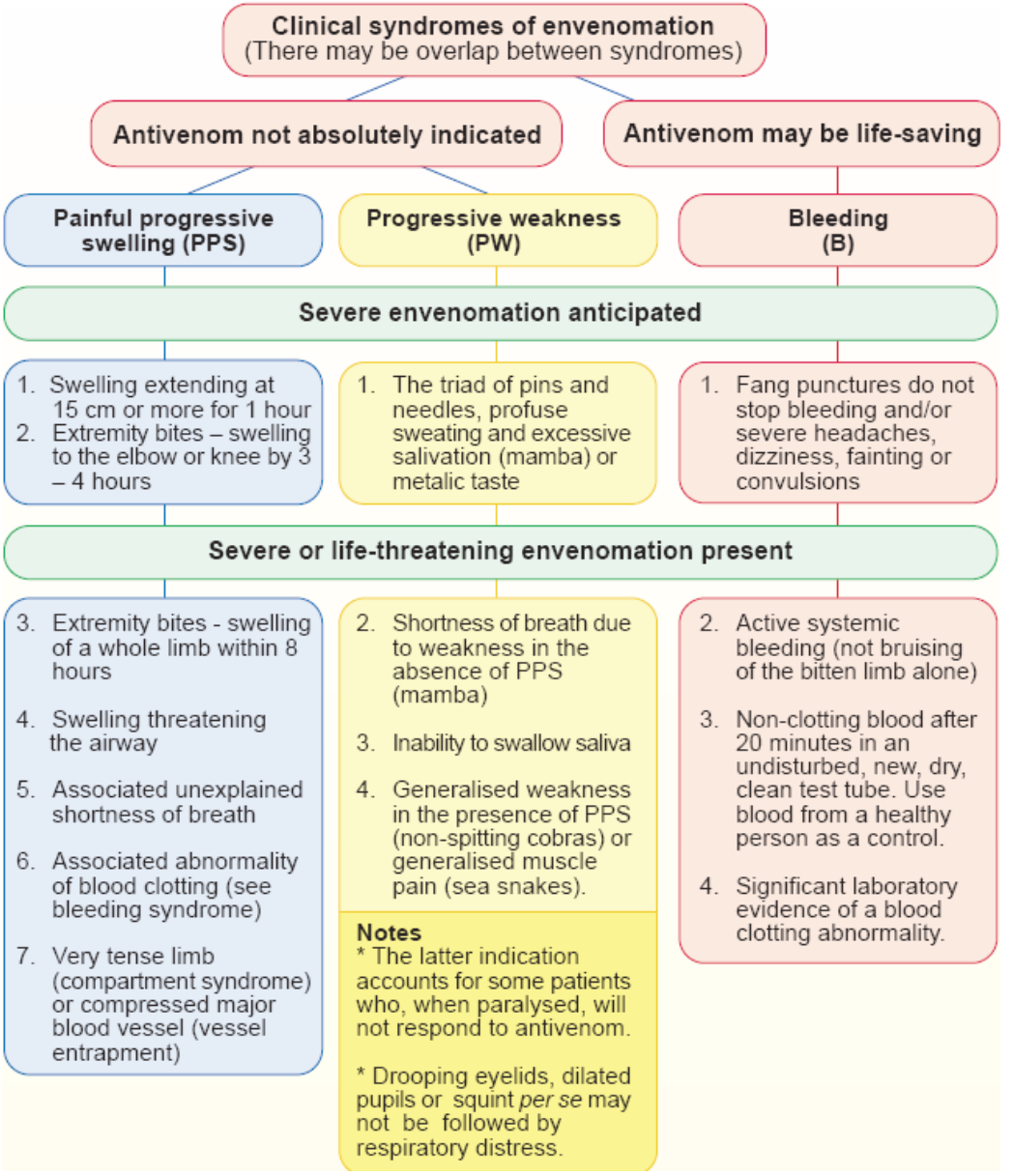
Diagnosis:

Diagnosis of snake bite is based on the presence of fang marks and a history consistent with exposure to a snake.

Snake envenomation involves the presence of snakebites plus evidence of tissue injury.

Management

- Primarily assess the ABCs and then a complete head to toe exam
  - o Airway: may be unable to clear secretions and exhibit hyper salivation
  - o Breathing: may be compromised by respiratory muscle weakness
- Assess for shock and signs of unusual bleeding (hematuria/ oozing from the wound)
- Neurological examination and cranial nerves
- Neurovascular status of limb, swelling and dermal necrosis.
- Immobilisation of the extremity and place at rest.
- Most snake bites can be treated without antivenoms with supportive care only.
- Antivenoms are indicated based on the clinical syndrome and poisonous species identified.
- **DO NOT:** try to suck out the venom, cut open the area around the bite, apply ice to the bite area, rub or inject any substance in the bite area.



FFigure 10. Clinical Syndromes of Envenomation.
Source: Blaylock RS. The identification and syndromic management of snakebite in South Africa: SA FamPract 2005; 47(9):48-53

Section 18 Ophthalmic Emergencies

18.1: Emergencies in Glaucoma

18.1.1 Acute angle closure glaucoma (AACG)

Acute angle closure glaucoma is a type of glaucoma that occurs when intraocular pressure (IOP) rises suddenly as a result of blockage of the aqueous humor outflow. It is due to appositional attachment of the iris with lens surface that in turn causes peripheral appositional closure of the angel system.

Clinical Features

Risk factors: age above 40, females, eyes with shallow anterior chamber and hyperopic eyes.

Symptoms: It is typically manifested by sudden onset ocular pain, headache, blurred vision, rainbow-colored halos around lights, nausea, and vomiting. During the attack, the intraocular pressure (IOP) is quite high that causes corneal edema and the other symptoms.

Signs: Red eye, reduced visual acuity, corneal edema and a mid-dilated and sluggish/ unreactive pupil. The affected eyeball is hard or firm on digital palpation (through the eyelid) compared to the unaffected second eye.

Examination: IOP measurement- It can be estimated by digital ballottement or using one of the tonometers, like Applanation, Tonopen or Shiotz tonometer. The IOP is typically above 30 mmHg (normal range: 10- 20 mmHg).The eye can be grossly assessed using hand held torch light and if available slit lump microscope for detailed examination of the eye.

Treatment:

Treatment focuses on lowering the IOP, breaking the obstruction and controlling inflammation.

Start with Acetazolamide 500 mg po stat then 250mg P.O. TID or QID for five days unless contraindicated (Sulphur allergy, renal pathology...)

Pilocarpine drops 1% or 2% to constrict the pupil. Administer 1 drop and repeat after 15minutes. It is also advisable to give drop to the contralateral eye (for prophylaxis).

Topical β-blocker (e.g., Timolol 0.5%) eye drop BID, (caution with asthma or COPD patients).

Steroid drops like Dexamethasone QID.

The second eye is also to high risk of acute attack, if prophylactic peripheral iridotomy is not done.

Refer urgently to a nearby eye center where there is Ophthalmologist (s).

Prognosis
AACG if not diagnosed as early as possible and not managed as an emergency, the affected eye cold loss vision permanently.

Untreated eye may further develop retinal vascular occlusion and neovasculazation that will further complicate the eye to be not only blind, but also painful.

Remember: Vision lossfrom glaucoma is not reversible.

18.1.2 Childhood Glaucoma

Childhood glaucoma (CHG) refers to glaucoma that occurs during the first three years of life.
It can be primary, the commonest type, developmental or acquired.

The primary CHG, often genetic in origin, is due to developmental abnormality of the anterior chamber angle, the site for the aqueous drainage system.

Developmental glaucoma is associated with presence of other eye anomalies.

Acquired or secondary childhood glaucoma is related to other causes like trauma, inflammation, tumor or surgery.

Clinical features
Primary CHG is a bilateral disease in 60% of the cases, and males are more affected than females.

Common among communities with consanguineous marriage.

60% in the first six months of age and 80% in the first year of life have the typical symptoms.

Symptoms: Tearing, blepharospasm (inability to open the eyelids) and photophobia are the three cardinal features of childhood glaucoma.

Other features include big eye ball(buphthalmos), large and/or cloudy cornea.

Signs: Elevated eye pressure, enlarged eye ball and cornea, corneal edema or opacification and glaucomatous optic nerve head.

Treatment
Because of its impact on visual development, all forms of congenital glaucoma need urgent intervention.

The definitive treatment is surgery (trabeculotomy or goniotomy).

Till the surgery, medical therapy is recommended. (Timolol 0.25% drops BID)
CHG requires referral to a tertiary eye center as an emergency case.

Prognosis
CHG leads to lifelong blindness if not detected and managed early.

The surgical management can be curative, if done successfully and in the first six months of age.
It requires lifelong follow-up.

18.1.3 Phacomorphic glaucoma

Phacomorphic glaucoma is an acute form of secondary glaucoma resulting from blockage of the pupil by an anteriorly displaced swollen or intumescent cataracts lens. It occurs in eyes with mature or hypermature cataract. Eyes with shallow anterior chamber are at more risk for the attack.

Clinical Feature
Patients who have had poor vision of the affected eye from cataract, present with pain, redness and further reduction of vision.

Examination: high intraocular pressure (IOP), inflamed eye, edematous cornea, shallow anterior chamber, pupil blocked by cataractous lens and either shallow or closed anterior chamber angle.

Treatment
Reduction of the IOP with medical treatment: Diamox 250 -500 mg TID or QID, Oral glycerol 2mg per, topical-Timolol, dorzalamid, or brimonidine.

Control inflammation with topical steroid drops- Dexamethasone or prednisolone drops every 4 hours.

Surgery: the definitive treatment is extraction of the cataract. If the angle is closed from delayed presentation, combined cataract extraction and glaucoma surgery is required.

Start treatment with the aforementioned drugs and refer the patient to eye center as an emergency case for the surgical management.

Prognosis
Early presentation, diagnosis and management reverse and cure the condition
Delayed presentation, misdiagnosis and mismanagement lead to permanent vision loss.

18.2 The Acute Red Eye

18.2.1 Ophthalmianeonatorum

Neonatal conjunctivitis (Ophthalmianeonatorum) is defined as conjunctival inflammation developing within the first month oflife. It is the most common infection of any kind in neonates, occurring in up to 10%.

Because of its association with serious local and systemic complications, it remains a significant cause of ocular morbidity, blindness, and even death in medically underserved areas around the world.

Gonococcal conjunctivitis (caused by Neisseria gonorrhoeae) is by far the most common cause and it is often the result of infection transmitted from mother to infant during delivery.

Other causes include Herpes simplex infection, chlamydial infection and chemical irritation.

Clinical features
Gonococcal and chlamydial neonatal conjunctivitis are characterized by mucopurulent/ purulent discharge from one or both eyes with diffuse conjunctival injection.

There may be associated eyelid swelling.

The child may also have associated systemic features, like pneumonitis.

Treatment of gonococcal conjunctivitis

Copious saline irrigation of the conjunctival discharge

Give a stat dose of ceftriaxone 25-50mg/kg iv/im and refer to ophthalmic center.

Prognosis

- Early and appropriate treatment leads to excellent prognosis.
- Delay in treatment of bacterial conjunctivitis in neonates can lead to corneal involvement (with subsequent ulceration and endophtalmitis) and systemic spread.

18.2.2 Endophtalmitis

Endophtalmitis is an infection of the contents of the eye. It is typically seen 2-5 days after an intraocular surgery (cataract, retinal detachment repair...) or an intraocular injection. It also follows ocular trauma. It is usually painful and associated with a red eye and reduced vision. The inflammation is typically so severe that white cells precipitate in the lower portion of the anterior chamber giving rise to a hypopyon (pus in the anterior chamber). This is a very significant sign.

Endophtalmitis is the most feared complication of intraocular procedures and is potentially devastating (the eye can be lost). These cases need urgent referral.

In cases where there is no history of recent surgery, or trauma, a hypopyon may be the result of severe uveitis (often HLA-B27-associated anterior uveitis). These cases should be referred on a routine basis to an ophthalmologist.

18.2.3 Uveitis

Uveitisis inflammation primarily of the uveal tissue of the eye and may involve the anterior segment, posterior segment or both. Patients may have history of prior episodes, with history of use of eye drops/ injections. On examination, patients typically have ciliary injection (redness around the limbus), photophobia, mildly reduced acuity and an aching discomfort in and around the eye.

These patients should be referred to an ophthalmologist as routine practice.

18.2.4 Subconjunctival hemorrhage

It is confluent bleed that obscures the conjunctival bloodvessels. It will be patchy and less dense at its edges. Seen with trauma but may also occur with Valsalva, vomiting, coughing.No treatment is needed unless the conjunctiva is bulging or prolapsing between theclosed lids. If this is the case then an eye ointmentwill prevent desiccation/ulceration (e.g. Tetracycline QID).

- No referral needed ifspontaneous.

18.2.5 Bacterial keratitis

This condition presents as a painful red eye (usually with a foreign body sensation).

Predisposing factors – Trauma associated with agricultural activity is by far most common risk factor in the developing world.

Other predisposing factors include ocular surface diseases, like dry eye syndrome, trichiasis, blepharitis etc.

Local and systemic immune suppression andcontact lens wear are other risk factors for bacterial keratitis.

Clinical Features
Patients present with pain, photophobia, red eye and decreased vision.

The characteristic finding is a small creamy-white spot on the surface of the cornea, usually only a millimeter or so in diameter. This "infiltrate" is a solitary colony of bacteria (like what we see on an agar plate). If patients present late, there may be a wider area of whitish cornea (ulceration or abscess formation).

Treatment
Start on topical antibiotic eye drops (e.g., 3% ciprofloxacin) using one drop every 15minutes for the first 2 hours then hourly until advised otherwise. Never give topical anesthetics or steroid eye drops to patients for use at home! Patients need immediate referral to ophthalmologist.
Prognosis – unless managed properly, bacterial keratitis can end up with corneal perforation or scarring.

18.2.6 Herpes simplex keratitis

Herpes simplex keratitis is a recurrent condition that has a very characteristicappearance with fluorescein staining. The eye will have sharp pain, it will often appear mildly injected and the visual acuity may be normal or slightly reduced. Cornealsensation will be reduced when compared to the fellow eye.

The hallmark of this condition is a "dendritic ulcer": a thin, linear, branching epithelialulceration with club-shaped terminal bulbs at the end of each branch. (Best appreciated using blue light after fluorescein stain).

Do not treat this with steroids as this will provoke rapid progression and acorneal melt! Never give topical anesthetic eye drops to patients for use at home!

Treatment
Treat with 3% acyclovir ointment 5 times day or oral anti-viral agents. Patients need routine referral.

18.2.7 Herpes zoster ophthalmicus (shingles):

With Herpes simplex zoster (shingles) a dermatomal vesicular rash is characteristic. Start a suitable oral antiviral straight away. If the disease is bilateral or if a patient under 40 years is affected then it may represent the first manifestation of HIV.Patients need routine referral to check for signs of ocular involvement.

18.2.8 Scleritis

Scleritis is an intensely painful inflammation of the sclera (often woken from sleepat night). The affected area is usually a brawny red and acutely tender to any pressure.
Vision is usually impaired. Unlike episcleritis (which is not usually painful), there will be no blanching of the blood vessels in the affected area after topical phenylephrine 2.5%. 50% of patients have systemic disease (often a connective tissue disease). These patientsneed routine referral.

18.3. Chemical Injuries

Chemical injuries range in severity from superficial punctuates keratitis to corneal opacification with limbal ischemia. Acids and irritants can damage the cornea and conjunctiva. However, the most feared chemical insult to the cornea is an alkali burn, and these injuries should be managed with particular care.

The severity of a chemical injury is related to the properties of the chemical, the area of affected ocular surface, duration of exposure (including retention of particulate chemical on the surface of the globe or under the upper lid) and related effects such as thermal damage. Alkalis tend to penetrate more deeply than acids, as the latter coagulate surface proteins, forming a protective barrier; the most commonly involved alkalis are ammonia, sodium hydroxide and lime. Powders and other solids can be retained under the lids or deep in the conjunctivalfornices. They are therefore harder to remove and much more likely to cause serious damage than liquids.

Treatment

A chemical burn is the only eye injury that requires emergency treatment without formal clinical assessment. Immediate treatment is as follows:

- Copious irrigation is crucial to minimize duration of contact with the chemi cal and normalize the pH in the conjunctival sac as soon as possible, and the speed and efficacy of irrigation is the most important prognostic factor following chemical injury. Topical anesthetic should be instilled prior to irrigation, as this dramatically improves comfort and facilitates cooperation. A lid speculum may be helpful. Tap water should be used if necessary to avoid any delay, but a sterile balanced buffered solution, such as normal saline or Ringer lactate, should be used to irrigate the eye for 15–30 minutes or until the measured pH is neutral.

- Double-eversion of the upper eyelid should be performed so that any retained particulate matter trapped in the fornices is identified and removed.

- Debridement of necrotic areas of corneal epithelium should be performed at the slit lamp to promote re-epithelialization and remove associated chemical residue.

- Further management –Depending on the degree of the ocular injury patients may need hospitalization and further medical management in ophthalmic center.

18.4 Visual Phenomena

This refers to "seeing things" that the patient knows are not really there.

Flashing lights & floaters
Patients commonly present with white flashes of light in their temporal visual field in the affected eye. These aremoreobvious when it is dark. In the light they may complain of looking through a "veil" or a "cobweb" and there is often a new largefloater that may be described as a spider or a ring that moves about as they look from one side to another.Visualacuity should not be significantly impaired (must be documented).Thisis a common acute presentationin ophthalmology and is usually caused by abenignposterior vitreousdetachment. These events can precipitatea retinaldetachment so these patients need to be seen by ophthalmology on a routine basis to rule out retinal pathology.

18.4.1 Migraine aura

No referral is needed to the ophthalmologist if the patient has established migraine with visual symptoms that are typical for them.

18.4.2 Diplopia

Monocular: usually due to cataract, corneal or retinal pathology.
- Elective referral.

Binocular: due to misalignment of the visual axes of the two eyes. (Always consider the possibility of giant cell arteritis.

Cause includes: Third cranial nerve paresis
- This is a potential medical emergency.

The signs may include ptosis, pupil dilation and poor/absent adduction, elevation and depression of the affected eye (leaving the eye exotropia).

If the patient is over 40 with identifiable cardiovascular risk factors, a complete third nerve palsy and no other signs or symptoms then a microvascular etiology is likely.Such patients need routine referral.All other patients ("partial" third nerve paresis, patients without cardiovascular risk factors, young patients and patients with signs or symptoms that may suggest an intracranial bleed) need an emergent CT or MR angiography to exclude an intracranial aneurysm (usually of the posterior communicating artery). If an aneurysm is found then refer to neurosurgery. Patients with no intracranial pathology need a routine referral to ophthalmology.

18.5 Sudden Visual Loss

History

For any patient with sudden visual loss, the following information should be obtained:

- Age
- Duration of visual loss or changes
- Whether one eye or both eyes affected
- History of trauma
- Prior episodes/ophthalmologic history
- Symptoms - Photophobia, headache, pain ,photopia, floater ,curtaining

It is important to ask about comorbid conditions such as hypertension, hypercholesterolemia, collagen vascular disease, hematological disorders, cancer, or drug use.

Physical

- Inspect the extraocular area and assess visual acuity, visual fields, extraocular movements, and pupil reactivity
- Inspect the eye: A non-injected eye may be painful owing to optic neuritis, clus ter headaches, sinusitis, or dental pain. Normal findings on this examination eliminate extraocular causes of visual loss.
- Red and painful eyes should be examined with a slit lamp, (see acute red eye above)
- The anterior chamber should be evaluated for hyphemia, cells, and flares
- Visual field testing with hand movement is used to assess if central or peripheral field vision deficiency is present.
- Careful fundus examination is part of a complete ophthalmic assessment. On funduscopy, a detached retina appears gray and detached.

The examination should also include complete cardiac and neurologic evaluation, including murmurs and carotid bruits.

Causes of painless sudden visual loss

- Ocular Ischemic Syndromes

  Persistent eye ischemia can be classified into central retinal artery occlusion (CRAO), branch retinal artery occlusion (BRAO), or ischemia of the optic nerve, which is caused by involvement of the posterior choroidal blood supply of the nerve (anterior ischemic optic neuropathy [AION]).

  - CRAO is sudden, painless blindness with persistent visual loss. Perception of hand movement or light can be preserved in parts of the visual field. Diagnosis is confirmed by ophthalmoscopy, which shows attenuated retinal arteries and veins (very early only), and a cloudy whitening of the retina (i.e., edema) with the consequent cherry-red spot in the macula in a patient who has lost vision in

one eye. If the occlusion lasts more than 1 hour, the retina becomes irreversibly infarcted.

- BRAO, visual defect and retinal ischemia are more focal and have an altitudinal, lateral, or scotomatous quality.

- AION, the patient usually develops painless visual loss in the eye, which is noted on awakening in the morning without worsening thereafter. The degree of loss is variable but most often incomplete. Ophthalmoscopy shows edema of the op tic disc and splinter hemorrhages at the disc margins. Subsequent involvement of the other eye is common.

- In nonarteritic ischemic optic neuropathy (NAION), the patient develops pain less visual loss in the eye, decreased central visual acuity, peripheral visual field loss, or both. The etiology of NAION is unknown, but pallid swelling of the optic disc is observed. Patients are at risk during the next 5 years to develop involve ment of the other eye; Spontaneous improvement of vision may occur.

- BRVO involves one of the branch retinal veins. Most involve the superior or infe rior temporal arcades and occur at an arteriovenous crossing where the vein is compressed by a sclerotic artery. The superior or inferior temporal arcades cause macular vein occlusion with profound visual deficit.

- Hemispheric vein occlusion involves the venous drainage of either the superior or inferior retina.

- CRVO involves occlusion of the main central vein, which usually occurs at the level of the lamina cribrosa. This occlusion interferes with the drainage of the whole retina.

- CRVO has 2 types: nonischemic and ischemic. These types are characterized by the severity of the retinal vein ischemia, although both have very similar oph thalmological findings. Nonischemic is the more common form and occurs when blood flow and oxygen delivery are restored following vein blockage but Is chemic CRVO patients have sudden, painless vision loss with count fingers or see hand movement.

- **Retinaldetachment:** the patient develops floaters, photopia followed by curtain ing and painless visual loss.

- **Angle-closure glaucoma:** (See above Acute angle closure glaucoma)
- **Papilledema/neoplasm:** Intracranial hypertension causes persisting visual loss by mechanically compressing or physiologically destroying the optic nerve. Anddyschromatopsia.

- **Intraocular foreign bodies:** Usually follows trauma.
- **Hysteria/malingering:** The patient with hysterical blindness or loss of vision will, despite alleged loss of vision, still be capable of maneuvering in a room. The pupillary reactions are normal. The loss of vision is a subconscious conversion symptom. A purely functional loss of vision can be assumed when the visual field is markedly constricted, orientation when walking is intact, and pupillary reactions to light are normal.

Treatment of Sudden Visual Loss

Medical Care

- Medical care for patients with sudden visual loss includes the following:
- Aspirin is believed to be beneficial in patients with no hemodynamically signifi cant disease of the carotid artery (i.e., greater than 1 mm residual lumen) or in those who are poor surgical candidates.
- Cases with Retinal detachment should be urgently referred to Ophthalmologist
- Optic Neuritis is treated by high dose intravenous/oral steroids.
- In casesof CRAO, no effective treatment.

For patients with nonischemic CRVO are need treatment with intraocular anti-inflammatory agents and therefore should be referred to an Ophthalmologist.

Consultations

Ophthalmic consultation is prudent in any case of sudden visual loss that cannot be easily and confidently explained and managed by emergency department physicians.

Cardiac and neurologic consultation is recommended. A complete cardiac and neurologic examination, including murmurs and carotid bruits, should be performed.

18.6 Orbital & Peri-Orbital Swelling

The commonest cause of orbital and peri-orbital swelling is cellulitis. An important distinction exists between infections arising in the orbit and infections that arise in the skin (Orbital cellulitis Vs perceptual cellulitis – respectively). Pre-septal cellulitis is no different from cellulitis elsewhere in the body. This usually occursfollowing local trauma, or insect bites.

Infection in the orbit (orbital cellulitis) is far more serious as it poses a risk of vision loss (through increased intra-orbital pressure) and ofposterior spread (which can lead to meningitis and brain abscess formation). Orbital cellulitis usually occur secondary to sinusitis with direct spread of infection to the orbit.Despite the differing severity, the two conditions can be very hard to discern clinically asconditionspresent with marked lid swelling (often unable to open the eye), redness, warmth of the affected tissues and tenderness.

The key to differentiating the two is to look for signsof orbital disease:

- Reduced visual acuity
- Limited eye movements which may give rise to binocular diplopia
- Proptosis
- A relative afferent pupillary defect
- Red desaturation (a red target appears darker or less saturated with the affected eye)

All the above are signs of orbital disease and therefore point towards orbital cellulitis.

Management

- Blood should be sent for CBC and culture prior to the administration of any antibiotics.
- A CT scan of the orbits can detect the presence of sinus disease & identify whe ther ornot an intraorbital abscess is present.

Treatment

If orbital cellulitis is suspected then IV antibiotics should be administered after sending blood samples for cultures. Ceftriaxone is a reasonable empiric choice based on the likely sinus pathogens. Patients need to be referred for further management and follow up.Other causes of orbital swelling exist such as thyroid eye disease (Graves' disease),

Carotid-cavernous fistulae and orbital tumors. These would also warrant routine referral.

18.7 Eye injuries

18.7.1 Eyelid trauma

In all eye traumas always consider the need for tetanus immunization if there is an open wound.Any sharp injury near the eye may alsohave involved the globe or even the brain. Apparently innocent lid lacerations may be the only sign of a penetrating eye injury or injury to the frontal lobe. The mechanism of injury must be considered. Small wounds of the eye lid that are uncomplicated, that do not involve the lid margin and that are well away from the lacrimal system may be treated in the Emergency Department.Indications for referral of patients with lid laceration include:

1. Involvement of the lid margin.
2. Extensive lid lacerations associated with tissue loss.
3. Lacerations associated with orbital fat prolapse.
4. Lacerations involving thepunctum orcanaliculi.
5. Suspicion of associated globe/brain injury.

18.7.2. Ocular foreign bodies

18.7.2.1 Superficial foreign body

The history is usually diagnostic. Foreign bodies on the conjunctiva or in the fornix can usually be wiped away with a cotton-tipped applicator. A foreign body on the cornea isusually more adherent and usually requires removal under slit lamp using a 27G needle. Always ensure that there is not another foreign body in the other eye or hidden under aneyelid. The upper lid should always be everted to check for this. It is also important to be suspicious about the possibility of an intraocular foreign body in thesecases. The history should be a good guide.In cases where the injury occurred when "metal hit metal" (e.g. hammer on chisel), thereis a serious risk of penetrating injuries from high velocity fragments that carry sufficient momentum to pass readily into the orbit or the eye. It is important that thesecases must have plain x-rays of the orbits to look for an intraocular or intraorbital foreign body. No referral is needed if a superficial foreign body is successfully removed and there are no other concerns. Prescribe an antibiotic eye ointment q.i.d. for 5 days. Never give topical anesthetic eye drops to patients for use at home!

18.7.2.2 Sub tarsalforeign bodies

Foreign bodies that do not embed into the eye will usually end up beneath the upper eyelid. This causes pain on blinking and gives rise to a very characteristic pattern of fluorescein staining. If you see this you must evert the upper lid. The foreign body will most likely be close to the margin of the eyelid (near the base of the lashes) and can usually be wiped away with a cotton-tipped applicator. Give an antibiotic ointment q.i.d. for 5 days. Never give topical anesthetic eye drops to patients for use at home.

18.7.2.3 Suspected intraocular foreign body

If the mechanism of injury suggests the possibility of an intraocular foreign body (IOFB) then you must do plain x-rays of the orbits to investigate the possibility.

Plain films should include a PA & 2 laterals; the laterals should be taken in up and downgaze to look for signs of a foreign body inside the eye (an IOFB will move in the 2 lateral views whereas intraorbital foreign bodies will stay still).A CT with fine slices through the orbits is the best investigation for localizing a foreign body and should be requested if the x-ray is suspicious.

IOFBs are a form of penetrating eye injury and are therefore potentially devastating. They warrant an emergent referral. Do not administer any topical medication; the patient must have an eye shield over the eye (to ensure that there is no pressure on the globe).NB. If there is evidence of an open globe or there is suspicion that there may be an open globe thendo not apply any eye drops or ointment and cover the eye with a shield. This shield should ensure that nothing can place any pressure on the globe.

18.7.3 Ruptured Globe

Presentation - decreased vision, subconjunctival haemorrhage, hyphemia and irregular shaped pupil. A traumatic cataract and full-thickness scleral or corneal lesion may also be present. The lens may become subdued and vitreous haemorrhage may also be present.

Management

TAT injection

Protect the eye with eyeshield.

- Start systemic antibiotics, analgesics
- Avoid topical medications!
- If the patient has nausea, give antiemetics to prevent expulsive haemorrhage.
- Immediate referral to ophthalmologist

18.7.4 Orbital Blow-out Fracture

Traumatic blow-out fractures usually follow a direct trauma by a fist or by a material with a diameter larger than the orbital diameter. Patients usually present with pain that increases upon vertical eye movement, binocular diplopia and crepitusafter nose blowing. Epistaxis and ecchymosis may also be present. Enophthalmos may develop after the oedema hasresolved. Traumas without a blow-out fracture may present with similar signs, in which case they usually resolve spontaneouslyin a week.An examination of the orbital contents as described above should be performed. Signs of subcutaneous emphysema shouldbe noted. The globe should be evaluated carefully for rupture, hyphemia, inflammation, iridodialysis, and retinal or choroidal injury.

Treatment

Nasal decongestant sprays, broad-spectrum oral antibiotics, and ice packs should all be administered. Surgical repair isemergent within 24 h if CT shows entrapped muscle or tissue with signs of diplopia and gastrointestinal (nausea/vomiting) orcardiovascular symptoms (heart block, bradycardia or syncope) or if the patients is a child.All patients with suspected blow out fractures should be immediately referred to ophthalmologist.

18.8: Retrobulbar Haemorrhage

Retrobulbar haemorrhage is bleeding in the orbit behind the globe (intraconal space). It is a sight–threatening condition unless managed immediately.Common following fall accidents, trauma to the head or periocular region or it could occur as iatrogenic orbital pain, tight eyelids (especially lower eyelid with subconjunctival haemorrhage and proptosis resistingretropulsion following trauma are the hallmarks. Decreased vision, eyelid ecchymosis, limited extraocular motility and increased IOP may also be present.

Treatment

Intervention comes before further investigations.If vision is threatened, do canthotomy (a 5mm long lateral incision of the lateral canthus deep to the orbital rim) and cantholysis (cutting the lateral attachment of the lower lid). Success is achieved when the lower eyelid is relaxed and moving freely.

Do not repair the wound after problem is relieved.Refer patient to ophthalmologist for further investigation and management.If IOP is dangerously increased or vision is threatened, urgent surgical intervention in the form of a lateral canthotomy with orwithout cantholysis is required and can often prevent permanent visual loss.

18.9: Pediatric considerations

In addition to the ocular disorders mentioned above, children with the following conditions warrant urgent referral for ophthalmologist evaluation and management.

1. Lecocoria – A condition in which there is awhitish reflex through the pupil of the eye. It is more noticeable at night. This could be a sign of a grave condition like a retinoblastoma or other embryogenic conditions like congenital cataract. Retinoblastoma can be treated if the condition is detected early enough, but it can quickly spread to brain and other organs if there is any delay in treatment. Parents should be counseled for urgent treatment!

2. Squint- squinting eyes could be a sign of a serious ocular problem or neurologic disorder. These eyes are also at risk of developing amblyopia.Therefore they need urgent referral.

Section 19 Ear, Nose and Throat Emergencies

19.1: Epistaxis

Epistaxis (nasal bleeding) is a common condition that presents as an emergency. It may be very severe and life threatening but in most cases is trivial and easily controlled. Epistaxis is a sign and not a disease per se and an attempt should always be made to find cause.

Location of Epistaxis
Epistaxis can be classified into anterior and posterior locations. Anterior source of epistaxis is 90 % of the time.

Anterior

- The most common anterior Epistaxis location is the Kiesselbach's plexus area, also known as "Little area". This is a region in the anteroinferior part of the nasal septum where four arteries have anastomotic connections.

Posterior

- Posterior epistaxis location is bleeding that occurs from beyond the middle turbinate or at the posterior and superior aspects of the nasal cavity. The vessels most responsible for posterior epistaxis are branch of the sphenopalatine artery and the anterior ethmoid artery

Etiologies of Epistaxis
Most common etiologies of Epistaxis (however, not entirely conclusive) include the following

Local causes

- Idiopathic (85%)
- Traumatic (fractures, foreign body, nose picking)
- Inflammatory (rhinitis ,sinusitis)
- Neoplastic (tumors of the nose, sinuses and nasopharynx)
- Environment (
- Iatrogenic (surgery , steroid nasal spray)

General causes

- Anticoagulant use (most common)
- Disease of the blood (haemophilia,leukaemia)
- Familial haemorrhagic telangiectasia
- Palate defect (thrombocytopenia)
- Hepatic insufficiency and alcohol abuse

Evaluation

The initial evaluation of epistaxis should focus on airway assessment, cardiovascular stability, fluid refutation and emergent otolaryngology consultation can be necessary in severe epistaxis.

History

The following questions are not conclusive in the full evaluation of the patient's history

- Important aspects of the patient's history
    - o Is the bleeding primarily anterior or is the patient mainly coughing out or spit ting up blood ?Note:The later may suggest a posterior bleed.
    - o Is the patient taking aspirin, warfarin or nonsteroidal anti-inflammatory (NSAIDs) medication?

Presentation

Anterior Epistaxis

Usually the blood exits almost entirely from the anterior portion of the nose

Posterior Epistaxis

Most of the bleeding occurs in the nasopharynx and mouth although some blood can exit through the anterior nose as well.

Posterior Epistaxis is often more severe and difficult to control

Physical Examination

If the site of bleeding is not readily apparent on direct visual examination then a nasal endoscopy needs to be done to further evaluate the nasal cavity

Investigation

Investigation should include a full blood count (FBC)(check Hb, white cell count and platelets),clotting studies and blood for group and cross-match if necessary

Management

The aims are to arrest the haemorrhage and to treat the underlying cause. The bleeding is usually stopped by one of the following methods:

- **Pressure** on the nostril (can be supplemented with ice-cold packs and sucking ice cubes)
- **Local** cautery (chemical or electro coagulation)
- **Anterio**r nasal packing (paraffin gauze, BIPP, Marcel)
- **Packing** of posterior space (gauze, Foley 's catheter,
- **Surgical** intervention
    - o Endoscopic approach. For exposure and ligation of the sphenopalatine artery
    - o Ligation of maxillary artery in the pterygomaxillary fossa
    - o Anterior ethmoid artery ligation
    - o External carotid artery ligation

o Embolization of vessels under radiographic control

PROCEDURE PROTOCOL: SILVER NITRATE CAUTERIZATION FOR EPISTAXIS

Indication

- Anterior nosebleed from the Kiesselbach's plexus area

Cautery

- If active bleeding is present, hemostasis must be achieved first by applying topi cal vasoconstrictor in combination with anterior nasal compression for 5 to 10 minutes
- The silver nitrate should be applied with firm pressure for 5 to10 seconds

- After the successful procedure is complete, the patient is instructed to use anti biotic ointment in the nose twice a day for 1 week; if possible the patient should not use aspirin or NSAIDs products for this duration of time.

PROCEDURE PROTOCOL: NASAL PACKING FOR EPISTAXIS

Nasal packing is generally reserved for those cases where external pressure and cauterization fail to control the bleeding. First try and effectively evacuate clots from the nasal cavity there are two types of packing – anterior and posterior nasal packing. The packs stay in place for a minimum of 2 to 5 days. The patient is placed on a prophylactic antistaphylococcal antibiotic.

Table 18. Type of Packing Used for Nasal Epistaxis

Anterior	Posterior
Paraffin gauze	NOTE: Posterior packs are extremely uncomfortable and patients requiring postnasal pack should always be hospitalized • Foley's catheter can be use, the bulb I inflated with saline and pulled forward so that choana is blocked and then anterior nasal pack is kept in the usual manner
Marcel nasal tampon expands when saline is injected over the sponge after inserted into the nasal cavity	• These days nasal balloons are available
Rapid rhino balloon tampon acts as a palate aggregator and forms a lubricant upon contact .deflate prior to removal	

Complication of anterior or posterior packing

- Pressure necrosis of surrounding structure
- Sinus infection
- Toxic shock syndrome

19.2: Nasal Bone Fracture

The nose may be injured in varies forms of sport, impersonal assaults and in traffic accidents. Injury to the nose may result in one or a combination of the following

- Fractures of the nasal bone
- Epistaxis
- Fracture or dislocation of septum
- Septal haematoma

Fracture of the nose

Nasal fracture can be open, close or both. A fracture of nose is considered "open" (compound) if laceration of the overlying skin exposes the underlying nasal bones.

History

- Mechanism of trauma. What was the direction of force and the nature of striing objects?
- History of previous facial trauma or surgery
- Other than swelling, is the nose appearance different than before the trauma
- Does the patient perceive a functional impairment in breathing compared to before the trauma
- Ask – the nasal fracture related to an assault or motor vehicle accident? if so there may be medical-legal aspects to address including documentation of nasal fracture through ordering a nasal x-ray or ct scan

Evaluation

- Asses the airway first
- Immediate nasal bleeding after a blow to the nose means nasal bone fracture (90% probability)
    - o X- ray of the nose are optional
    - o The best way to evaluate nasal fracture is by waiting for swelling to subside for a week and comparing with a pre- injury photo.
- At the time of injury, always check for septal hematoma
    - o A hematoma causes the septum to appear defected to both sides of nose at the same place
    - o If a haematoma is missed and infection occurs, the top of the nose will collapse (saddle nose)
- Consideration in other facial trauma , check for
    - o Numbness (floor of orbit fracture)
    - o Diplopia (ocular muscle trapped in fracture)
    - o Trismus (mandibular fracture)

Treatment of nasal fracture

- If there is a nondisplaced or minimally fracture that is cosmetically acceptable, it is ap propriate to wait and observe as the swelling diminishes before considering if surgery is needed

- Surgical management of nasal fracture is divided into close reduction and open reduc tion
- The window of opportunity to obtain the best result in early reduction of the nasal frac ture is to do the close reduction within 2 to 3 hours before the onset of significant oedema. The second best window of opportunity is 5 to 10 days after injury.

Closed reduction	Open reduction
• Closed reduction involves manipulation of the nasal bone without incisions and gen erally preferred choice for treating nasal fracture • Close reduction can be done under local anesthesia or general anesthesia	• Open reduction may include a range of techniques including septoplasty or septorhinoplasty

Septal hematoma

- Septal hematoma have high risk of complication if left untreated
- Needle drainage as soon as possible is indicated for all nasal septal hematoma
- If left untreated, septal haematoma abscess can result in the spread of bacteria into the paranasal sinus and intracranial structure leading to intracranial abscess, cavernous sinus thrombosis

19.3: Foreign bodies

19.3.1 Foreign body in the nose

- Nasal foreign bodies are most commonly found in 2 to 3 year old children
- Types of foreign bodies are:
    - o Inorganic foreign bodies such as metal,plastic from toys and stones
    - o Organic foreign bodies such as sponge, paper, peas and nuts
- Foreign bodies are irritants and its presence usually generates an inflammatory reaction in the nasal mucosa causing a nasal discharge. This is initially mucoid, but will eventu ally become mucopurulent and finally odiferous muco pus.Confirmation of the presence of the foreign body is from the history and examination of the child

Treatment

- Removal is best accompanied with a wax hook. It is passed point downwards above the foreign body, which is brought to the floor of the nose and raked anteriorly.
- Cupped forceps are preferable for the removal of thin objects
- In every case the nasal cavity must be examined afterwards as there may be a second foreign body more posteriorly
- The child should be discharged with antibiotic if there is any obvious infection

19.3.2 Foreign body in the ear

Foreign bodies in the ear common problem at school age children and cotton wool is common in adults.

Types of foreign bodies are

- Inorganic foreign bodies such as metal,plastic from toys and stones
- Organic foreign bodies such as sponge, paper, peas and nuts

Treatment

Foreign body in the external ear canal is usually easily seen on otoscopy. Removal may appear to be easy, but usually requires the skills and facilities of a specialist. It is sometimes possible to remove the foreign body in the clinic, but a general anaesthesia may be required for children and sensitive adults

Treatment

- As general rule, most foreign bodies can be removed by syringing. Organic for eign bodiesare hygroscopic and should not syringed.
- Suction or a fine hook may be used to remove material of vegetable origin and large objects which lie superficial to the isthmus
- Forceps should be used for soft material such as paper, cotton or sponge
- Insects should be killed before syringing by instilling spirit drops into the ear canal

19.3.3 Foreign body in the pharynx

Sharp and irregular foreign bodies may become impacted in the tonsil, base of tongue, vallecular or pyriform fossa. Fish bone is usually lodged in the tonsil.

Treatment

The first step of management is to get the patient try to localize by pointing to where they feel the bone. A thorough examination with light, a tongue depressor and laryngeal mirror should reveal the offending bone. If the patient cannot tolerate indirect laryngoscope, then examination with a nasendoscopy is useful. In some patients there will be no abnormal findings and a lateral soft- tissue radiograph is indicated, if this too is normal they should be reassured and reviewed 2 days later. By this time the sensation will usually have passed, but if there are persistent symptoms the patient should be re-examined.

Treatment

- After the foreign body has been visualized, the pharynx should be anaesthized with lignocaine spray and use an anaesthetic laryngoscope to depress the tongue and a suitable forceps to try and grasp it and remove it.

- General anaesthesia is required to remove a foreign body from the pharynx if the patient is young or unable to tolerate the above maneuvers.

19.3.4 Foreign body in the oesophagus

Impaction of foreign body depends on the size and shape of the object. The commonest objects are coins in children and fish or meat bones in adults. Impaction is commonest at the level of the cricopharyngeus muscle, but may also occur at the level where the oesophagus is crossed by the left main bronchi

Clinical feature

Adults are usually aware of having swallowed something and are able to fairly accurately the level at which it is impacted. Children and psychiatric patients may not be able to do so reliably. Discomfort or pain in the oesophagus and difficulty in swallowing are the cardinal symptoms. The foreign body may cause coughing and excessive salivation.

Investigation

- Lateral and antero-posterior soft – tissue radiographs of the neck and are man datory.
- The inexperienced may confuse calcification on the laryngeal cartilage with opaque of foreign body

Treatment

- If the obstruction is due to an impacted food bolus, the safest treatment is to admit the patient and give a dose of intravenous hyoscine and diazepam. This will usually allow the oesophagus to relax and permit the passage of the bolus.
- If there is a sharp or bony object, the patient requires an oesophagoscopy as soon as possible.
- The patient should remain in the hospital for 24 hours postoperatively and receive nil by mouth for the first 4 hours and only water for the next 4 hours
- Perforation of the oesophagus should be treated with intravenous antibiotic and nasogastric feeding

19.4. Injury to the tympanic membrane

The tympanic membrane, being deeply placed, is well protected from injury. .Damage does occur, may be direct or indirect

- Direct trauma is caused by poking in the ear with sharp, such as hair grips, in an attempt to clean the ear
- Indirect trauma is usually caused by pressure from a slap with an open hand or blast injury

Symptom

- Pain, acute at time of rupture, usually transient
- Deafness, not usually severe, conductive type, cochlear damage may occur from excessive movement of the stapes
- Tinnitus, may be persistent- this is cochlear damage
- Vertigo, rarely

Signs

- Bleeding from the ear
- Blood clot in the meatus
- Visible tear in the tympanic membrane
- Physical examination - On physical examination the following should be documented
- Facial nerve palsy
- If nystagmus exits
- External auditory canal appearance
- Tympanic membrane perforation location

Treatment

- If perforation is traumatic tend to close spontaneously,healing may take up to 3 months
- Instruct patient to use Vaseline impregnated cotton to seal ear during showers, otherwise leave it open
- Do not clean out the ear or syringe
- Prescribe antibiotics drops only if ear is actively discharging and if the injury has been caused by direct t

Section 20 Dermatologic Emergencies

20.1: Stevens-Johnson Syndrome/Toxic Epidermal Necrolysis

Stevens-Johnson syndrome (SJS)/toxic epidermal necrolysis (TEN) are life-threatening severe mucocutaneous hypersensitivity reactions, usually caused by a drug are the most common causes. If the body surface area involved is<10%, it considered as SJS and if > 10% of the body surface area is involved, it is TEN while involvement of 15 to 30% of the body surface area is SJS/TEN overlap. Cases that are not caused by drugs are attributed to infection (mostly Mycoplasma pneumonia), vaccination and Graft-vs. hostdisease. The incidence, severity of both disorders may be higher in bone marrow transplant recipients. In Pneumocystis jiroveci- infected HIV patients, in patients with Systemic Lupus(SLE) and in patients with other chronic rheumatologic diseases

Diagnosis

Diagnosis of SJS/TEN requires clinic pathologic correlation. The appropriate clinical findings include targeted and atypical targeted skin lesions and at least two mucosal surfaces involved (ocular, oral, genital, etc.) Nikolsky's sign, which refers to epidermal detachment occurring with lateral pressure adjacent to bullae, is present and can be a clue to diagnosis which can be confirmed through skin biopsy. Labeling as either SJS or TEN depends on the amount of epidermal detachment present.

Treatment

The mainstay of treatment for SJS/TEN includes:

- Immediate discontinuation of the offending agent
- Supportive care: involves maintenance of thermal regulation, fluid/volume re placement, maximizing protein nutrition, and meticulous wound care which is similar to a burn care.
- Ophthalmology should be consulted to assess for ocular involvement, as ocular sequelae are common
- Systemic corticosteroids,IV immunoglobulins

20.2 Staphylococcal Scalded Skin Syndrome

Staphylococcal scalded skin syndrome (SSSS), also called Ritter's disease, is a superficial skin blistering disorder caused by toxin mediated exfoliative disease, occurring commonly in neonates and young children. It is very rarein adults.Adults are less commonly affected and tend to have significant comorbidities such as renal failure, immunosuppression, alcoholism, malignancy, or HIV infection.Although mortality is quite low in neonates and in children, in adults it can be as high as 50%.

Diagnosis

The diagnosis is generally clinical. The diagnosis of SSSS is based on the presence of three criteria: (1) appropriate clinical presentation with erythroderma, skin desquamation, or bullae; (2) isolation of S. aureus strain that produces ET; (3) characteristic histopathology of intraepidermal cleavage at the granular layer.Blood cultures are usually negative in children, but may be positive in adults.

Treatment

- Patients should be hospitalized and, depending on severity P.O. or IV antibiotics should be started immediately. Penicillinase-resistant penicillin like IV Cloxacil lin is the treatment of choice to eradicate the infectious focus.
- Supportive care

20.3 Necrotizing Fasciitis

Necrotizing fasciitis (NF) is a severe, insidiously advancing soft-tissue infection characterized by wide spread fascial necrosis. The organisms most closely linked to necrotizing fasciitis aregroup A beta hemolytic streptococcus, although it may be caused by other bacteria or different streptococcal serotypes. The three most important causes are as follows: Type I Polymicrobial,Type II group A streptococcal and Type III – gas gangrene or clostridial myonecrosis

The frequency of necrotizing fasciitis is on the rise because of an increase in immunocompromised patients with diabetes mellitus, cancer, alcoholism, vascular insufficiencies, organ transplants, HIV infection and neutropenia. Fournier gangrene is a form of necrotizing fasciitis that is localized to the scrotum and perianal area.

Diagnosis

NF is an infection of the superficial fascia and develops as organisms extend from the subcutaneous tissue and travel along fascial planes. It can affect the trunk, groin, head, or extremities. In many cases of NF, antecedent trauma or surgery can be identified.

Diagnosis of NF can be difficult and requires a high index ofsuspicion, as misdiagnosis and delay in appropriate treatment is considered the most important factor in fatal cases. The hallmark symptoms of NF are fever, intense pain and erythema,edema and tenderness over the involved skin and underlying muscle. Pain out of proportion to the physical examination, rapidly progressing erythema, associated myalgia, history of a puncture injury, and bullae formation. Late clues include crepitus, hypoesthesia of skin, prominent bullae and skin necrosis. Eventually septic shock with hypotension and multi-organ failure occurs.

Treatment

The treatment protocol includes surgical debridement of necrotic tissue, antimicrobial therapy for polymicrobial infection, fluid, nutritional support and IVIG. Urgent and extensive surgical debridement of all necrotic and affected tissue is the mainstay of effective treatment.

20. 4 Erysipelas and Cellulites

Ersipelas and cellulites refers to spreading bacterial infection of the skin.

Cellulitis involves the deep subcutaneous tissue while Erysipelas is an acute superficial form of cellulitis and involves the dermis and upper subcutaneous tissue

Causes -Streptococcus pyogenes and Staphylococcus aureus

Presentation: Most common in the lower limbs clinically. Local signs of inflammation – swelling (tumor), erythema (rubor), warmth (calor), pain (dolor); may be associated with lymphangitis

Systemically has fever, malaise or rigors, particularly with erysipelas.Erysipelas is distinguished from cellulitis by a well-defined, red -raised border

Treatment

- Antibiotics (Cloxacillin or Amoxicillin/amoxiclav/procaine penicillin)
- Supportive care including rest, leg elevation, sterile dressings and analgesia

Section 21 Psychiatric Emergencies

21.1 Principles of management in emergency setting

Different behavioral and psychic conditions lead to emergency presentation. The following are few examples:

- Aggression & violence, Suicidal behavior
- Restlessness, hyperactivity
- Mute, not eating & drinking
- Sever anxiety like panic attack
- Confusion state
- Intoxication or withdrawal and others

Any psychiatric condition can result in emergency presentation. The common once are Mania, acute psychotic state, suicide, catatonia, panic attacks, delirium and delirium tremens. It is very important that these disorders are to be considered after excluding non-psychiatric medical conditions as it is equally possible that they present with psychological and behavioral symptoms. The emergency health care provider must have high index of

suspicion for organic etiology:

- Very acute onset of symptoms
- Fluctuating levels of consciousness
- Disorientation to time and place, short-term memory difficulties
- Concurrent medical illness with physical findings, and
- History free of previous psychiatric episodes

Principles of management in emergency setting

- Safety issues – to patient, staff and other people
- Talking down the patient
- Restrain – mainly chemical restraint; physical restraint as a last resort
- Consider and R/o underlining emergency medical conditions, diagnostic inves tigation
- Admission decision, mostly involuntary. Based on psychiatric assessment deci sion is made as to whether to admit or discharge patient.
    - o **DISCHARGE –** Patient is deemed both medically and psychiatrically safe to be discharged to home setting. Follow-up with outpatient psy chiatry needs to be arranged if deemed necessary. If discharge prescrip tion is written, it should not be for duration longer than follow-up ap pointment.
    - o **ADMIT –** Patient is deemed unsafe or too impaired by current mental health condition to cope in home setting. The following decisions need to be made with regards to admission: 1) Does the patient need stabiliza tion in ER ward; 2) What ward will the patient be eventually admitted to; 2) Will the patient be a voluntary or involuntary admission.
- **Mechanical Restraints –** Avoid the use of mechanical restraints whenever possible. If restraints are used, least-restraint policy should be initiated. The least amount and du

ration of restraints should be utilized. If physically restrained, the patient needs to be assessed by attending physician within 30 minutes and frequently assessed thereafter to determine if he/she could be taken out of restraints.

21.2 Aggressive and violent behavior

Many different factors result in aggressive and violent behavior. The most common implicated psychiatric disorders are

- Schizophrenia
- Post convulsion confusion in epilepsy
- Acute organic syndrome specially alcohol intoxication, drug intoxication and delirium
- Chronic organic brain syndrome
- Manic states when frustrated
- Catatonic excitement
- Aggressive psychopaths

Treatment

- Never take patients by surprise, explain all the procedures you are going to do
- Quick organized intervention to avoid injury to self and others
- Understand why a patient is aggressive before resorting to restraining methods.
- Use minimum restraint when necessary and discontinue when it is no longer necessary
- Never approach an aggressive mental patient alone
- In case of organic brain syndrome, identify the physical cause and manage it accordingly
- Pharmacological Treatment – Standing medications should be initiated based on medication reconciliation. Typical as needed (PRN) medication for agitation, aggression and violence are listed below:

•Young and Healthy	**Antipsychotic Treatments:** Haloperidol 2-5mg PO/IM every hour as needed up to a max of 15mg/24h Risperidone 1-2mg PO every hour as needed up to a max of 6-8mg/24h Olanzapine 10mg PO every hour as needed up to a max of 20mg/24h **Benzodiazepine Treatment:** Diazepam 5-10 mg every 1-2 hour as needed up to a max 40mg/24h **Extrapyramidal Side-Effect Treatment:** Trihexiphenidyl(artane/benzxexol) 2-5mg PO 2-4 times daily up to a maximum of 20 mg/24h
• Elderly, Frail or Neuroleptic Naïve	**Antipsychotic Treatments:** Haloperidol 0.5-2mg PO/IM/IV every hour as needed up to a max of 5mg/24h Risperidone 0.25-0.5mg PO every hour as needed up to a max of 2mg/24h Olanzapine 2.5-5mg PO every hour as needed up to a max of 20mg/24h **Benzodiazepine Treatment:** Diazepam 5-10 mg every 1-2 hour as needed up to a max 20mg/24h **Extrapyramidal Side-Effect Treatment:** ***Trihexiphenidyl(artane/benzxexol) 2-5mg PO 2-4 times daily up to a maximum of 10 mg/24h***

21.3 Suicide

Suicide is an intentional self-inflicted death. Not a random or pointless act. It is a way out of a crisis that causes intense suffering (psychological pain, financial loss, illness etc). It is associated with unfulfilled needs, feelings of hopelessness & helplessness, ambivalent conflicts between survival and unbearable stress, a narrowing of perceived options, and a need for escape.

History of current or past psychiatric disorder is the most important risk factor for suicide but not all suicidal people have a psychiatric condition. The SAD PERSON acronym is of a fairly good suicide risk assessment tool: Sex (male sex), Age (the very young and old), Depression or any psychiatric diagnosis, Previous suicidal attempt, Ethanol abuse, Rational thought loss, Social supports lacking, Organized plan, No spouse and Sickness. Other factors that also increase suicide risk are available means/weapons, recent life-altering events, an acute psychosocial stressor e.g. public humiliationcommand hallucinations, religious preoccupation and persistent hostile environment.

The following are factors identified to deter suicidal behaviour:

- Children in the home
- Sense of responsibility to family
- Pregnancy
- Religiosity
- Life satisfaction
- Reality testing ability
- Positive coping skills
- Positive problem-solving skills
- Positive social support
- Positive therapeutic relationship

Asking about suicide
Has there been a period

- where you have had trouble sleeping;
- where you have felt depressed, sad, or lost interest in things;
- where you have felt worthless, simple or guilty
- Any thought of death, wish to go to sleep and never to wake up?
- Sometimes people think of committing suicide, did it ever cross your mind?
- Any plan or preparation? Access to the means?

When the person presents after a suicidal attempt

- ABC of life
- Patient may be defensive, feel shame or anger. There mayalso a negative reaction by medical personnel and the public towards the act.

- Start by asking physical symptoms
- Then ask "What & how did it happen"
- "Why"
- Intent, lethality and patient's understanding of lethality
- Reaction for being rescued & any future plan

Documentation - the screening and any other conversation about suicide with a patient should be clearly documented.

Treatment

Avoiding thinking of the behavior in a derogatory way (e.g., that the patient is being "manipulative" or has made a "suicidal gesture")

In-patient vs. Out-patient – for any moderate to severe suicidal risk, inpatient management should be seriously considered. Before sending the person home imagining the place and situation that the individual will be returning to after the evaluation – availability and adequacy of social support? Impulsive behaviour? Suicidal plans?

Remove or treat risk factors

- underlying medical and psychiatric problems
- physical (in unpredictable and impulsive patients) &/or chemical restraint (anxiolitics, antidepressants, neuroleptics)
- Psychotherapy (supportive, family therapies)
- Electroconvulsive therapy (ECT)
- REFERE the patient where he/she can be best helped for follow-up

21.4 Delirium

Delirium is a syndrome characterized by a disturbance of consciousness and a change in cognition that develops over a short time.Associated clinical features include disorganization of thought processes, perceptual disturbances, psychomotor hyperactivity and hypoactivity, disruption of the sleep-wake cycle, mood alteration and altered neurological function.It is almost always caused by one or more systemic or cerebral derangements that affect brain function.Advanced age is a major risk factor for the development of delirium. Other predisposing factors are preexisting brain damage, a history of delirium, alcohol dependence,diabetes, cancer, sensory impairment, and malnutrition.

Treatment

Management focuses on ensuring safety from behavioral disturbances while simultaneously assessing for a probable etiology and definitive treatment.

Primary goal in treatment of delirium is to treat the underlying cause.

Safety can best be addressed by combining environmental, behavioral, and pharmacologic means.

Environmental support: Patients with delirium should be neither sensory deprived nor overly stimulated by the environment.

They are usually helped by having a friend or relative in the room or by the presence of a regular sitter.

Regular orientations to person, place, and time help make patients with delirium comfortable.

Pharmacotherapy: The two major symptoms of delirium that may require pharmacological treatment are psychosis and insomnia.

A well-studied and commonly used drug for psychosis is haloperidol (Haldol), a first generation antipsychotic drug. Depending on a patient's age, weight, and physicalcondition, the initial dose may range from 2 to 6 mg intramuscularly, repeated in anhour if the patient remains agitated.

As soon as the patient is calm, oral medication should begin. Two daily oral doses should suffice, withtwo-thirds of the dose being given at bedtime.

*Phenothiazines (e.g. Chlorpromazine) should be avoided in delirious patients because these drugs are associated with significant anticholinergic activity.

***Use of second-generation antipsychotics,** such as risperidone (Risperdal), clozapine, olanzapine (Zyprexa), has limited data supporting it.

***The avoidance of benzodiazepines except for particular indications** (e.g., alcohol withdrawal delirium, delirium related to seizures) continues to be a recommendation.

Section 22 Pediatric Emergencies

22.1: Pediatric Basic Life Support for Healthcare Providers

In Pediatric Basic Life support the rescuer should first:

- Determine quickly if the victim is unresponsive
- If the victim is not breathing or is only gasping

If the victim is not breathing, only gasping, or is unresponsive, with sudden collapse:

- A lone rescuer should activate the emergency medical response service and retrieve the automated external defibrillator.
- If the victim did not experience "sudden" collapse, the rescuer should perform CPR for 2 minutes prior to seeking a defibrillator.
- Check the victim's pulse for no longer than 10 seconds.
- In children aged one to adolescence, the pulse should be checked at the carotid artery.
- In infants, the brachial pulse should be assessed.

If there is a palpable pulse within 10 seconds, then a rescue breath should be given every 3 -5 seconds

- Breaths should last one second and the chest should be observed for visible rise.

If the pulse is less than 60/minutes, or if the victim has signs of poor perfusion:

- The provider should begin chest compressions.
- 30 high quality compressions followed by two breaths.
- If two healthcare providers are available, the cycle of compressions to breaths should be 15:2

High quality compressions in CPR should be:

- A minimum of 1/3 to 1/2 the AP diameter of the chest, or
- Approximately 1 ½ inches in infants (4 cm) and 2" in children from age one to adolescence.
- The rate of compressions should be 100 per minute.
- Chest recoil should be complete between compressions.

Pediatric compression is performed:

- With the head of one hand over the lower ½ of the sternumbetween the nipples
- In infants, use two fingers, or use the thumb encircling technique if multiple providers are available.

After two minutes have passed when the AED is available:

- The victim's rhythm should be quickly assessed.
- Shockable rhythms include
    - Ventricular fibrillation
    - Pulseless ventricular tachycardia.
- One shock should be given to the victim, with resumption of CPR immediately after the shock.
- CPR should continue for 2 minutes.
- A dose attenuator should be used on infants if available, but if not, adult pads can be used.

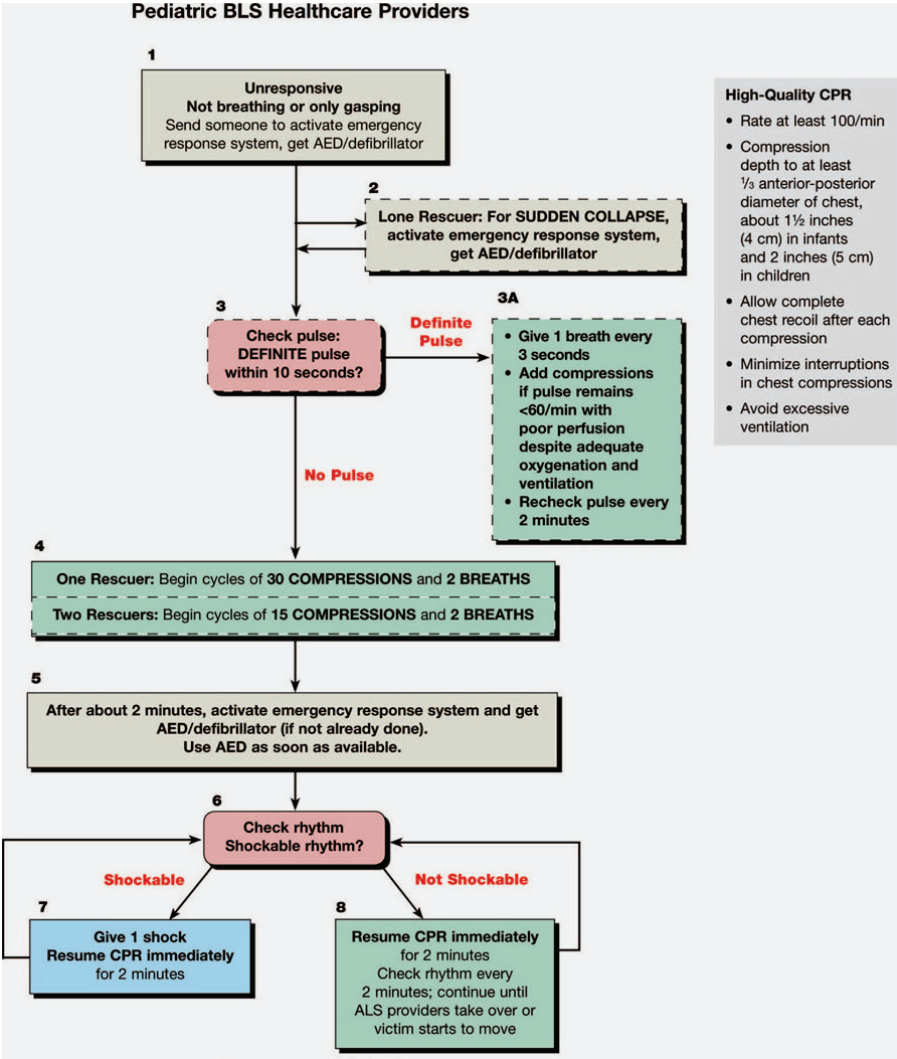


Figure 11. Pediatric BLS Healthcare Providers

22.2 Respiratory Emergencies

22.2.1 Croup

- Hoarseness
- Barky Cough
- Inspiratory Stridor
- Non-Toxic Appearance
- Common Age Group 6months - 6years

Triage considerations:

If severe stridor or retractions at rest, cyanotic/pale, hypoxic, obtunded or lethargic:

- Notify ED physician
- Transfer patient to resuscitation room
- Start immediate airway management
- Humidified oxygen to keep SaO2> 92%
- Start racemic epinephrine a 1:1 mixture at a dose of 0.25-0.5ml of 2.25% in 3ml of NS nebulizer and after each racemic epinephrine nebulizer, calculate and document Croup Score
- Avoid PO intake if respiratory rate > 60

Laboratory studies:

- In critical illness, consider ABG
- Imaging: Consider CXR or neck plain films if concern for foreign body, other obstruction

Medications/interventions:

- Dexamethasone (all patients, mild to severe)
- 0.6 mg/kg PO (preferred), IM, or IV; max 12 mg stat
- May give IV form orally for improved palatability

Croup Score (Westley croup score

	0	1	2	3	4	5
Stridor	None	With agitation	At rest			
Retractions	None	Mild	Moderate	Severe		
Air entry	Normal	Decreased	Markedly decreased			
Cyanosis	None				With agitation	At rest
Consciousness	Normal including sleep					Disori-ented

Mild <3
Moderate 3-7
Severe >7

22.2.2 Pneumonia

Fever, acute respiratory symptoms and signs, plus parenchymal infiltrate on chest X-ray in a previously healthy child due to a community-acquired infection (danger signs: cyanosis, apnoea, convulsions, impaired consciousness).

Indications for • hospitalization

- All patients with danger signs.
- Toxic appearance.
- Hypoxemia (Oxygen Saturation < 90%).
- Severe respiratory distress (Apnoea, grunting, chest in drawing, head nodding).
- Dehydration with vomiting or poor oral intake.
- Immunocompromised patients.
- Pneumonia refractory to oral antibiotics.
- Unreliable home environment.

Investigations:

CXR, WBC, CRP, ESR, and blood culture only positive in 30-40%

General management:

- Supportive: lowering temperature, adequate hydration and feeding (oral).
- Humidified oxygen via face mask or nasal prongs.
- Continuous monitoring of vital signs and saturation.

Management of infants under one year

(Common Bacteria in the first three months of age: Escherichia coli, Group B streptococci, Listeria monocytogenes, Haemophilius influenza type B, Staph aureus).

- Admit all newborns / infants with danger signs.
- Antibiotic regimen (consider antibiotic combinations).
    - o For the first 3 months Ampicillin + Gentamycin combination, beyond this age crystalline penicillin is the first line drug followed by ceftriax one or cefotaxime
    - o Ampicillin 50-200 mg/kg divided q12 hours.
    - o Gentamycin 2.5 mg/kg repeated q8-12 hours.
    - o Crystalline Penicillin 50,000 – 75,000 I.U/kg/dose q4-6 hours
    - o Cefotaxime 100-150 mg/kg divided q8 hours.
    - o Ceftriaxone 50 – 75 mg / kg / day IV BID

Organisms requiring additional antibiotic coverage

- Methicillin Resistant Staphylococcus Aureus (MRSA) Vancomycin.

Outpatient (if febrile without respiratory distress)

- Amoxicillin 50-90mg/kg/day.
- Amoxicillin – Clavulanic Acid 50-90 mg / kg / day.
- Erythromycin 30 – 40 mg / kg / day PO divided q6 hour's ×10d.
- Azithromycin 10 mg / kg day for children older than 6 months

Management of children aged > 1 year

(Bacterial causes: S.pneumonia, Chlamydia pneumonia)

Inpatient

- o Crystalline Penicillin 50,000 – 75,000 I.U/kg/dose q4-6 hours
- o Cefotaxime 100 mg / kg / day IV or
- o Ceftriaxone 50 – 75 mg / kg / day IV BID

Outpatient (if febrile without respiratory distress)

- o Amoxicillin 50 – 90 mg / kg / day.
- o Amoxicillin – Clavulanic Acid 50 – 90 mg / kg / day.
- o Erythromycin 30 – 40 mg / kg / day.
- o Azithromycin 10 mg / kg day.
- o Clarithromycin 15mg /kg/day

General management:

- Supportive: lowering temperature, adequate hydration and feeding (oral).
- Humidified oxygen via face mask or nasal prongs.
- Continuous monitoring of vital signs and saturation.

Management of infants under one year

(Common Bacteria in the first three months of age: Escherichia coli, Group B streptococci, Listeria monocytogenes, Haemophilius influenza type B, Staph aureus).

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o Ampicillin 50-200 mg/kg divided q12 hours.

o Gentamycin 2.5 mg/kg repeated q8-12 hours.

o Crystalline Penicillin 50,000 – 75,000 I.U/kg/dose q4-6 hours

22.2.3 Acute bronchiolitis

Viral induced reactive airways disease and the first episode of asthma are clinically indistinguishable.

Clinical features

Cough, fever, runny nose, tachypnoea, tachycardia, intercostal retraction, crackles and wheeze.

Diagnostic studies

Rapid antigen detection for RSV from nasopharyngeal secretions.

Management

- Humidified oxygen via face mask or nasal prongs.
- Hydration.
- Continuous monitoring.
- Consider mechanical ventilation if, impending respiratory failure, severe
- hypoxia unresponsive to noninvasive oxygen therapy

22.2.4 Acute asthma

A disease characterized by recurrent attacks of breathlessness and wheezing, which vary in severity and frequency from person to person.

Clinical features:

- Tachycardia.
- Restlessness.
- Tachypnea and prolonged expiration.
- Wheeze (could be audible) and mainly expiratory.
- Chest deformity: pigeon chest, barrel chest, Harrison Sulcus
- Hyper resonant chest.

Diagnostic studies:

- Lung function test :
- Reduced FEV1 by 20%.
- Reduced PEFR by 20%.
- Response (FEV1, PEFR) to bronchodilator by > 15%.Blood gases (Sat O2> 90%).

Treatment

- Humidified oxygen via face mask or nasal prongs
- Hydration
- Nebulized Salbutamol 2.5 mg (for children < 5 years) & 5.0 mg (for children >5 years) in 3ml saline to be nebulized over 5 minutes using face mask OR
- Salbutamol by Metered Dose Inhaler (MDI) 6 – 8 puffs via spacer OR
- Epinephrine 1: 10,000 subcutaneously 0.1 mg / kg.
- Reassess for restlessness, wheeze, RR, PR and air entry
- If no response, repeat Nebulized Salbutamol after 30 minutes.
- Reassess after another 30 minutes.
- If no response: repeat Nebulized Salbutamol and start steroids (Hy drocortisone 100 – 300 mg IV), start Prednisolone 2 – 4 mg / kg stat; continue Prednisolone 2 mg / kg / day for three days.
- A child who does not respond to 3 doses of nebulized salbutamol given should be considered as acute severe asthma (status asthmaticus).
- Continuous monitoring.

22.2.5. Acute severe asthma/life threatening asthma

- Acute severe asthma/life threatening asthma is an acute asthma that does not respond to the (usual) outpatient treatment or did not respond to three doses of nebulized Salbutamol within two hours.
- Life – threatening asthma is asthma that endangers life (cyanosis, drows iness, and silent chest)

Clinical features:

- Severe respiratory distress.
- Inability to talk or drink.
- Tachypnoea and Severe tachycardia.
- Impaired consciousness.
- Pulsus paradoxus.
- Exhaustion.

Diagnostic tests:

- Lung function test.
- Reduced FEV1 by > 20%.
- Reduced PEFR by > 20%
- Blood gases (Sat O2 < 90%).

Treatment

- Admit to I.C.U or high care area (continuous monitoring).
- Humidified Oxygen via face mask.
- Continuous nebulization of Salbutamol nebulized solution 0.25 mg / kg / hr.I.V Hydrocortisone (2 – 4 mg / kg / dose 4 hourly)
- Nebulized ipratropium hydrochloride (15 mcg in 3 ml saline over 5 – 7 minutes), 4 – 6 hourly.
- Subcutaneous adrenaline (0.5 ml (1: 10000) half – to – one hourly (three doses).
- If there is no raised HR start with aminophylline 4mg/kg per dose
- I.V. magnesium sulphate (50 – 100 mg / kg) slowly 20 minutes
- Consider lsoprenaline infusion.
- Reassess half – hourly.
- Consider mechanical ventilation.

22.2.6 Respiratory failure

Respiratory failure is the inability of body to adequately oxygenate and / or ventilate.

Common Causes:

Upper airway obstruction

- Croup syndrome
- Retropharyngeal abscess,
- Diphtheria, epiglottitis.
- Foreign – body aspiration.

Lung disease

- Acute severe asthma.
- Bronchiolitis.
- Severe pneumonia.
- Pulmonary edema.
- Near drowning.
- Sepsis.

Central.

- CNS infection.
- Drug overdose.
- Stroke.
- Traumatic brain injury.

Clinical features:

- Type 1 respiratory failure (hypoxemia): Anxiety, severe tachypnoea, tachycardia, and pallor.
- Type II respiratory failure (hypoxemia and hypercarbia),
- Cyanosis, bradycardia, altered level of consciousness, and cardiac arrest.
- **Diagnostic studies:**

•

Type I

ABG (pa CO2<40 mm Hg, pa O2<80-90 mm Hg, or arterial oxygen saturation less than 90%)

• Type II

ABG (paCO2 >50 mm Hg, paO2<60 mm Hg, or arterial oxygen saturation less than90%).

Treatment

- Admission to the ICU.
- Ensure ABC.
- Intubation and mechanical ventilation.
- Ensure adequate oxygenation.
- Determine and treat the underlying cause.

22.3: Circulatory Emergency

22.3.1 Shock

Cold extremities, feeble pulse and delayed capillary refill (>3 seconds)
Investigation (after immediate resuscitation)

- Blood tests - ABG/electrolytes/urea/creatinine/bloodglucose
- CBC & differential
- Coagulation screen(PT,PTT,fibrinogen)
- Blood Group/ cross match
- Blood culture

Apply monitors – ECG/oximeter/non-invasive blood pressure, monitor level of Consciousness/pupils/hourly urine output (catheterize)

Treatment

- Bolus of fluids
- Crystalloid: 20mls/kg (0.9% saline) continue to monitor and assess.
- Give up to 80ml/kg Contact PICU
- Consider Intubation and ventilation

Antibiotics: if the cause of shock is sepsis or distributive shock
Neonates (less than 6/52): ampicillin (to cover Listeria) and cefotaxime orGentamycin.

- Cefotaxime: 75 mg/kg: 12 hrly;
- Ampicillin:75 mg/kg 4-6 hrly (max 2 g /24)
- Gentamycin: 2.5 mg/kg: for premature babies: 12hrly

Inotropes:

- Warm shock:
  - Dopamine 2 – 20 mcg / kg / min (30 X wt. in kg = no of mg to be added to 5 or 10 % dextrose to make up to 50 mls), 1 ml = 10 mcg / kg / min or
  - Adrenaline 0.05-1mcg /kg /min (0.3 X wt. in kg = no of mg to be added to 5 or 10 % dextrose to make 50 mls), 1 ml /hr. = 0.1 mcg /kg .min.
- **Cold shock:**
  - Dobutamine 2.5 – 20 mcg / kg / min (made as dopamine).
- If in doubt start Adrenaline 0.05-1 mcg /kg /min (0.3 X wt. in kg = no of mg to be added to5 or 10 % dextrose to make 50 mls), 1 ml /hr. = 0.1 mcg / kg .min.

Nor adrenaline 0.05 - 1 mcg /kg/min (made up as adrenaline) .

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22.4: CNS Emergencies

22.4.1 Coma

Coma in a child is defined as any child with a Glasgow coma score less than 15 or responding only to voice, pain or being unresponsive on the AVPU score. Coma is a symptom, not a diagnosis.

Immediate treatment

The aim of immediate management is to minimise any ongoing neurological damage whilst making a definitive diagnosis. Elements of the history, examination, investigation and treatment will therefore occur simultaneously.\

- Attend to airway, breathing and circulation. (ABCs)
- If trauma cause is possible immobilise cervical spine and arrange urgent neurosurgeryinvolvement.
- Insert I.V. line.
- Put the child in a recovery postion.
- Perform blood glucose; if glucometer < 2.5 mmol/l (45mg/dl) in a non-diabetic, administer I.V. dextrose (10%)
- Assess and monitor pulse, respiratory rate, BP, temperature, pulseoxime try and ECG monitoring and level of consciousness.
- Look carefully for subtle signs of a continuing convulsion.

History and physicalexamination Onset and duration of symptoms

- Past history – seizures, diabetes, adrenal. Insufficiency, infection, car diac, previous similar episodes (metabolic conditions).

In the presence of the mentioned signs consider:

- Scalp bruising or haematoma head injury
- Inconsistent history, retinal haemorrhage non-accidental injury
- Fever, seizures meningitis, encephalitis
- Focal neurological signs
- Focal seizures
- Papilloedema
- Asymmetric pupils
- Focal intracerebral pathology, e.g. Tumor
- Shunted hydrocephalus Blocked shunt
- Renal disease Hypertensive encephopathy

Investigations

- Full blood examination, urea and electrolytes, glucose, liver function test, arterial blood gas
- Urine drug metabolic screen, culture of blood and urine, ammonia
- cortisol coagulation screen, ECG

Ongoing care

Will be determined by the diagnosis, level of consciousness and degree of ventilatory and circulatorysupport needed.

22.4.2 Febrile seizure

Convulsion occurring in a child who:

- Is 6 months to 5 years of age and febrile.(Temp. >380c)
- Has no evidence of intracranial infection.
- Has no other defined metabolic disease.
- Is otherwise neurologically normal.
- Has no Past history of seizures.

Simple febrile convulsion:

- Primary generalized convulsion.
- Lasts less than 15 minutes.
- Is not repeated within 24 hours.

Emergency management:

- A-B-C-D-E.
- Maintain vital functions.
- Control the fever.
- Control the convulsion.
- Identify precipitating factors.

Approach to convulsing child

- Breathing- RR, intercostal retractions, accessory muscle use, grunting, chest expansion.
- Oxygen saturation if available
- High flow oxygen (2-4L/min)-via face mask with reservoir- support breathing with bag-valve maskdevice and consider intubation if needed.
- Circulation:-Monitor heart rate, pulse volume, capillary refill time, blood pressure, skin temperature and color.

- Calculate the dose 0f diazepam 0.5mg/kg Per rectum.
- Do not ever forget blood glucose – do a bedside glucometer test as a guide, if ≤ 45mg/dl in a well-nourished or <54mg/dl in a malnourished child, give 5ml/kg of 10% dextrose as soon as iv access is established.

If the child starts convulsing at the heath facility:

- A B C D E
- If seizure continues for> 5 min give diazepam (0.5mg/kg PR) or buccal midazolam at thesame dose.
- Observe for another 5 mins, if the seizure abates put in recovery position-providedbreathing is satisfactory

.

- Management of fever
- Remove excess clothing.
- Antipyretics:-Paracetamol 10-15mg/kg PO or PR 4-6 hrly
- Non -steroidal anti- inflammatory drugs such as Ibuprofen (5mg/kg) 8 hrly
- Ensure adequate fluid intake and correct dehydration
- Cold compression….
- Do not give empirical antibiotics if you are confident about the diagnosis of • simplefebrile seizures and there is no obvious bacterial focus.

Status Epilepticus

- Seizure lasts 30 min without regaining of consciousness
- Airway & Oxygen Breathing & Circulation Glucose.
- Diazepam 0.3mg/kg IV. Repeat same dose if the seizure does not stop
- Give a loading dose of phenobarbital 20mg/kg
- Midazolam 150-200micoro-g/kg tobe followed by midazolam infusion 2 micro grams/kg/min.
- You can increase the dose of midazolamby 4 micro-g/kg every 30 minutes according to patient's response. Maximum dose 0.5 ml/kg.
- Look for the underlying cause for the status epilepticus

22.5: Congestive Cardiac/heart failure (CC/HF)

Respiratory distress, tachycardia, hepatomegaly and cardiomegaly, oedema, raised jugular venous pressure and basal crepitation (in older patients).

- Depends on underling condition), skin perfusion and temperature, urine output), oedema.
- Chest x ray: helps confirm diagnosis and assess severity.

- Echo to know the cause of HF rather than diagnose it.
- Do not delay management till after echo.

Management, monitoring and follow up:

Supportive management:

- O2
- Bed-rest in cardiac position.
- Fluids: 2/3 of maintenance NGT/IV. (only if they do not take po, it is better to avoid IV fluids as much as possible)
- Blood/PRBC transfusion: patients with HF and HB below 8gm% small volumes over 4 hours with monitoring.
- Antibiotics in patients with suspicion of infection.

Specific treatment:

Mild-moderate:

- Diuretics: Start with Furosemide 1-3 mg/kg/day orally /IV.
- Use angiotensin converting enzyme inhibitor as second line after diuretics(Captopril/enalapril) captopril dose: 0.2 mg/kg/dose BD/TDS increasegradually to 4 mg/kg/day.Monitor blood pressure especially after the first few doses.
- Do not use ACE inhibitors in patients with obstructive lesions like AS(aortic stenosis) and HOCM(hypertrophic cardiomyopathy).
- If the patient is still significantly symptomatic or tachycardia Digoxin can bead ded, dose: 3-5 micrograms/kg/dose orally 12-24 hourly.
- Potassium supplement is not needed with the above combination (Lasix,captopril) unless the serum potassium is low.
- Add K-sparing diuretic (e.g. spironolactone) in cases of refractory heart failure/oedema.

Heart Failure in Cardiomyopathies/Myocarditis:

- Same treatment as above.

Consult cardiologist before further treatment

- Use of IVIG for acute myocarditis: IVIG still debatable but can give ben efitof doubt if history suggests acute disease. Dose 2gm/kg/dose over 12hours.
- Add beta blockers (carvedolol dose 0.2mg/kg/dose) increase gradually according to the response.
- Add aspirin (3-5 mg/kg/d) for patients with EF<30.
- Add warfarin in patients with H/O cerebrovascular accident or left ventric leclot seen on echo, monitor INR.

Heart Failure in Acute Rheumatic Carditis:

- Diagnosis is clinical applying the modified Jones' Criteria plus lab evi denceof streptococcal infection.
- In mild-moderate carditis: Bed rest, penicillin, aspirin (75-100mg/kg/d) 6hourly and anti-heart failure medications.

Indications for steroids:

- In severe or refractory carditis.
- Moderate- large pericardial effusion.

Dose:

- P.O. Prednisone 2 mg/kg/d for 2 weeks then aspirin 60mg/kg/d is added.
- Steroids tapered over a week and aspirin alone continued.
- Aspirin is tapered gradually guided by ESR.
- Penicillin prophylaxis 3 weekly continued for life in case of carditis withresidual valve lesion and for 18-25 years if there is no cardiac involvement.
- SBE prophylaxis on indications.

Heart Failure in Patients with established Rheumatic Valvular Disease:
Is it a new episode of rheumatic fever??
(ESR, ASO, ECG, repeat ECHO).

You need to have 2 minor criteria plus evidence of strep infection to diagnose ARF in this category.

- Diuretics +/- captopril combination in patients with MR, AR.
- Assess the ventricular dimensions and EF periodically.
- Add Propranolol or digoxin in patients with atrial fibrillation.

22.6: Endocrine Emergencies

22.6.1 Diabetic ketoacidosis

Diabetic ketoacidosis (DKA) is defined as significant hyperglycemia (blood glucose > 17 mmol/L or 300 mg/dl), ketonemia, andmetabolic acidosis (pH < 7.3, HCO3 < 15 mmol/L) , coupled with severe disturbance in fluid andelectrolytes balance. (Severe DKA = PH < 7.1 HCO3 < 5 mmol/L), in absence of these lab facilitiesconsider the patient to have DKA if he is symptomatic for diabetes and is dehydrated with hyperglycemiaand glycosuria with ketonuria.

Diagnosis and Assessment:

- Think of DKA in a known diabetic child or any child who presents with either of or acombination of the following.
- Classical symptoms of diabetes.
- Acute abdomen, provided that other causes have been ruled out
- Dehydration.
- Acidotic breathing.
- Disturbed level of consciousness.

Principles of Management:

- Treatment of shock.(they do not usually go into shock)
- Correction of dehydration and replacement of losses with provision of maintenance.
- Correction of electrolyte deficit.
- Correction of hyperglycemia.
- Correction of acidosis.
- Treatment of precipitating factors including sepsis.
- Observation for and treatment of cerebral oedema.
- Prevention of further attacks.

Treatment

Immediate:

- Coma care if child is comatose (gastric tube if abdomen distended).
- Assess and control breathing and circulation
- Start two IV lines. Line one is for fluid and electrolyte replacement and line 2 for insulin infusion.

Laboratory:

- Blood for: glucose, urea, creatinine, electrolytes gases, CBC+ diff, (cul ture: ifindicated), others if needed e.g. BF for malaria.
- Urine: urinalysis + culture (if indicated).

Notes:

- If a child is comatosed, urine can be obtained by catheter. In infants you can squeezethe napkin urine.
- Both hyperglycemia (using glucometer) and glycosuria and ketonuria (with strip)should be performed by the doctor in the ER without waiting for the laboratory results to take action.

Fluids:

- If patient is shocked give 20 ml/kg of normal saline (or ringer lactate) as fast as possible. Repeat these doses till circulation is restored in theemergency room. This should not later be subtracted from fluid therapy.
- If not shocked or once circulation is restored, start IV fluids as men tioned below.
- No need to give bolus saline if the patient is not shocked or hypotensive.

Insulin:

- No need to give IV insulin bolus but start I.V. insulin infusion 0.1 unit/ kg/hour or 0.3units/kg/s/c. . - 1 hour after starting of fluids.

Disposition:

- PICU: Those with coma, young infants, cardiovascular instability, and those whodeteriorate in the ward.
- Ward: other cases.
- Home: Some known cases with mild ketoacidosis or ketonuria can be managed at home, if there is reliable care taker.

Fluid Replacement

- Fluid replacement hould extend over 48 hours to achieve a slower correction of serum hyperosmolalityto prevent cerebral oedema. Therefore deficit should be given over 48 hours.

Deficit:

- For practical purposes, the usual deficit in most DKA patients is 10%.

Rate of Infusion:

- Add 24 hours maintenance to half of the calculated deficit and divide by 24 to obtain the hourly rate.
- After the intial fluid resuscitation, the 48 hour fluid is calculated as 85ml/kg (which is the defict) + maintenance fluid – bolus given in the first hour over 47 hours.

Fluid used:

- Use normal saline till blood glucose reaches 14-17 mmol/L (250-300 mg/dl) then change to 5%dextrose with 0.45 normal saline.

Potassium:

- Commenced when the child starts to pass urine (practically after the firs hour)) or if he is alreadypassing urine and or K is below 5 mmol/L).
- Add 40 mmol/L of potassium chloride (i.e. 20 mmol/bottle of 500 ml).
- Monitor by ECG, clinically & biochemically (if available).
- If serum potassium is > 6 mmol/L withhold potassium temporarily till potassium is < 6 mmol/L.

Bicarbonate:

- Give only if pH is less than 7 and there is circulatory un stability gener ally it is preferable to avoid it completely.
- Dose: 1-2 mmol/kg; given over 60 minutes.
- Check blood gas (venous or capillary sample in non-shocked patient) every 6 hours.
- Unless child is critically ill avoid giving bicarbonate during first hour or two of resuscitation thenrepeat blood gas if PH is still < 7 offer bicar bonate (if necessary).

Insulin Therapy:

Preparation: use regular (or rapid acting) insulin only. Infuse into a separate IV line using a syringe pump. Add 100 units in 100 ml (or 50 units in 50 ml) of normal saline in a syringe pump (or burette of the normal infusion pump) eachml will contain 1 unit/ml. This solution should be changed every 6 hours.

- Infusion rate: 0.1 unit/kg/hour i.e. 0.1 ml/kg/hour of the above men tioned preparation. Aiming to reduce blood glucose at rate of 4-5 mmol/ hr. (80 – 90 mg/hour). Usually there is a rapid drop after one hour of starting i.v. fluids.
- Continue this insulin infusion till acidosis is cleared i.e. either pH > 7.3, HCO3 > 15 mmol or normal anion gap (Na+ - (cl + HCO3) normal = 12 + 2 mmol/L).

Note: **Discontinuation of insulin infusion is not dictated by blood sugar level, but by clearance of acidosis.**

- If there is no facility to monitor blood gasses or serum bicarbonate continue infusion until the patient is clinically stable (fully conscious, well hydrated, does not look acidotic and taking orally well) and urine is containing no or trace of ketone.
- If there is no facility for a pump initially give regular insulin 0.3 units/kg s/c as start dose then 0.1units/kg subcutaneously hourly or 0.15 – 0.2 units s/c 2 hourly till acidosis is cleared. Monitoring:

- Blood glucose with a meter hourly during insulin infusion (at least hourly for 1st 4-6 hours then 2hourly if needed) then every 6 hours thereafter.
- Blood gases, blood glucose, urea and electrolytes 4 - 6 hourly (if available) and urine for ketone is2 hourly or on each voided urine.
- Vital signs (ECG monitor if available) and neuro observation (initially hourly till stable then every 4-6 hours). Also watch for headache, vomiting or behavior change and other signs of cerebral edema.
- Flow sheet to record: blood workup, intake and output, doses of insulin, and urinalysis for glucose and ketone. (Adequate urine output = > 1.5 ml/kg/h)

Problem Solving During Monitoring:

- After resuscitation, the typical aim of rate of blood glucose fall is 4-5 mmol/hour. (80 – 90 mg/dl).
- When blood glucose falls to 14-17 mmol/L (250 – 300 mg) change fluid to 5% dextrose with 0.45saline to maintain blood glucose in the desired range of 120-200 mg/dl.
- If blood glucose rises again above 17 mmol/L (300 mg/dl) change the fluid to NS
- If blood glucose falls below 100mg/dl or falls too rapidly increase the concentration of glucose to7.5% (or more).
- The insulin infusion rate should only be decreased if blood glucose levels remains below thetarget range despite glucose supplementation.
- Do not stop insulin infusion or hourly s/c if the patient is still acidotic.
- Blood sugar freshly voided urinary ketones before main meals, and midnight.
- Electrolyte once daily (if necessary).
- Vital sign and neuro observation 4 - 6 hourly at least for the first 24 hours.

Complications: Cerebral Edema

Diagnostic criteria:

- Abnormal motor or verbal response to pain.
- Decorticate or decerebrate positive.
- Cranial nerve palsy (especially 3, 4, and 6).
- Abnormal respiration (chine stokes, apnoea).

Major criteria:

- Abnormal mentation/function of level of consciousness.
- Sustained heart rate deceleration (decrease more than 20 beats/minute) not attributable to sleepor improved intravascular volume.
- Age inappropriate incontinence.

Minor criteria:

- Vomiting.
- Headache.
- Lethargy or not easily arousable.
- Diastolic B.P. > 90 mm.
- Age < 5 yrs.

Action:

- Exclude hypoglycemia. Give immediate mannitol 1 gm/kg over 20 min utes (i.e. 0.5 ml/kg of20% solution) (3% saline 5-10 ml/kg I.V over 30 minutes can be used • if no mannitol is available.
- Halve rehydration infusion rate until situation improves.
- Elevate the head.
- Move to PICU
- If assisted ventilation required maintain PCO at 23.5 K pa (25 – 30 mmHg).
- Consider continuation of mannitol at 0.25 gm/kg every 6 hours to pre vent rebound increase inICP or repeat bolus every 4 - 6 hours.

22. 7: Severe complicated malaria

Severe malaria is malaria due to P. falciparum that sufficiently serious to beimmediate threat to life. It is a medical emergency which requires hospitalization.
A patient is regarded as having severe malaria if he or she has one or more (mostly seen incombination) of the following conditions:

- Prostration.
- Respiratory distress.
- Repeated convulsions within 24 hours.
- Severe anaemia
- Pulmonary oedema.
- Impaired conciousness
- Shock
- Jaundice
- DIC

If severe malaria is suspected, the following key aspects of assessment should be followed:

- Assess level of consciousness follow Glasgow scale or Blantyre coma scale.
- Vital signs:

- Pulse rate.
- Respiratory rate (look for acidotic breathing deep and rapid).
- Blood pressure.
- Temperature.
- Pallor.
- Assess hydration status.

Immediate management:

- Start resuscitation particularly maintenance of a patent airway.
- Abort convulsion by giving diazepam (see protocol of fits) 0.5 mg/kg PR.
- Establish IV line.

Manage fever by the following actions:

- Remove excess clothing.
- Put fan on if available.
- Paracetamol orally or PR 15 mg /kg 4-6 hourly.
- Correct dehydration.

Correct hypo glycaemia:

Do bedside glucometer test (if available) if blood glucose is less than 3 mmol/L give 5ml/kg of 10% dextrose as soon as an IV access is estab lished.

If level of consciousness is disturbed:

Insert nasogastric tube if the patient is unconscious or in coma and fix indwelling catheter.

Immediate tests should include the following:

- Thick and thin blood film for malaria.
- PCV.
- Hg.
- Blood glucose.
- Lumber puncture if indicated.

Specific management:

Artesunate

2.4mg/kg IV or IM given on admission (time=0) then at 12 and 24 hours, then once a day for 6 days or until the child tolerates po medication. If the patient tolerates after 24 hours, the medication can be changed to po Artesunate- Lumefantrine for the next three days.

Quinine:
Should be given initially by intravenous infusion, preferably in 5%glucose. The dose is 10 mg salt/kg body weight administered 8 hourly until the patient can tolerate orally, then continue the same dose to complete the course duration for 7 days. If IV notpossible, quinine (the same dose)
can be given intramuscularly diluted with normal saline ordistilled water to a concentration of 60 mg/ml into both anterior upper thighs.

Artemether:
Artemether injection is another alternative. The dose for children is 1.6mg/kg twice in the1st 24 hour (12 hours apart). Followed by 1.6 mg/kg daily for 6 days (8 days in total) .
- Anticonvulsants. (Ifneeded).

22. 8: Tetanus

An acute often fatal infectious disease caused by the bacterium clostridium tetani, which usually enters the body through a wound and produce a toxin that affect nerve conduction

Principles of management:

- Eradication of clostridium tetani.
- Neutralization of tetanus toxin.
- Control of seizures and respiration.
- Palliation and provision of supportive care.
- Prevention of recurrences.

Treatment:

- Surgical wound excision and debridement to remove the foreign body after administration of human globulin (HTIG) and antibiotics. Remov al of umbilical stump in neonate is not recommended.
- Single injection of 500 U of TIG I.M to neutralize the toxin. But doses as high as3000-6000 U are also recommended. If TIG is not available I.V human immunoglobulin or tetanus antitoxin (TAT) in dose of 50.000 – 100.000 U I.M (check for sensitivity is needed).
- Penicillin G 100.000 u/k 6 hourly for 10 days + Metronidazole + Genta mycin in neonate.
- Diazepam for both seizure and relaxation 0.1- 0.2 mg/kg every 3 - 6 hour I.V for2 - 5 weeks + chlorpromazine 50-100mg/kg/day alternate

with diazepam every 4-6 hours.

- Keep the Pt. in dark quiet room, maintenance of fluid and electrol needs.

Careful nursing to mouth, skin, bladder, and bowel function is needed to avoid ulceration and infection.

NB: don't insert nasogastric tube unless the patient is fully sedated.

Prevention:

- Active immunization (DPT) 6 weeks, 10 weeks and 14 weeks. Booster at 4years every 10 years (DP).
- Immunization of women with tetanus toxoid prevents neonatal tetanus with atleast 2 doses.

22. 9: Fluids and Electrolytes

Fluid deficit occurs if there is fluid loss from the body secondary to diarrhea, burn, renal loss etc

Goals of management:

- Estimate fluid and electrolyte deficits, maintenance requirements, and ongoing losses.
- Select and administer appropriate fluids.
- Monitor the management.
- Treat the specific cause.

Maintenance requirements:

- First 10kg 100ml/kg
- Second 10kg 50ml/kg
- Each additional kg 20ml/kg
- Sodium 2-4mmol/kg
- Potassium 2-3mmol/kg
- Chloride 2mmol/kg

Ongoing losses:

The maintenance fluid volume given above includes the total fluid requirement under normal conditions including the insensible losses, plus essential urine output and moderate state of diuresis. Under certain pathological conditions, you might need to calculate the exact water and electrolyte losses.

22.9.1 Acute diarrhea and severe dehydration

Assessment of dehydration:

Severe dehydration

If any two of the following signs are present:

- Lethargic or unconscious
- Deeply sunken eyes
- Not able to drink or drinking poorly
- Skin pinch goes back very slowly >2 seconds

Some dehydration

If any two of the following signs are present:

Restlessness, irritability,
Sunken eyes, Drink eagerly, Skin recoil in <2 seconds

No dehydration

No signs of dehydration

Treatment of Severe Dehydration:

- First year of age: give 30ml/kg in 01 hour, then give 70ml/kgin infants (under 12Months) 1hour 5 hours
- Children (12 months upto 5 years) 30ml/kg over 30minutes and 70ml/kg over 2 and ½ hours.

NB: be sure to add ongoing losses to maintenance + deficit fluids and electrolytes:

- The patient should be reevaluated every 1-2 hours by checking vital signs, clinical signs, ongoing losses and urine output.
- Reassess the hydration status after 6hrs to choose the appropriate plan for subsequent management.

Treatment of some dehydration

75ml/kg of ORS over 4 hours

Treatment of no dehydration

Replacement treatment,
Age upto 2 years = 50-100ml ORS after each loose stool
2 years or more= 100-200ml ORS after each loose stool.

Treatment of dehydration in malnourished patients:

- Use Resomal (rehydration solution for malnutrition).
- Give 5ml/kg every 30 min for first 2 hrs orally or by NG tube.

- Then 5 – 10ml/kg hrly in the next 4 – 10 hrs.
- Strict monitoring as they are prone to fluid overload

22.9.2 Electrolyte Disturbances

22.9.2.1 Hyponatremia:

Serum Na+ less than 130 mmol\L

Causes of Hyponatremia:

Sodium loss (diarrhea, Burn, third space loss, hyperglycemia etc)

Treatment:

- Formula for correction of hyponatremia (Deficit)
- (Desired Na+-actual Na+)*body wt.\kg*0.6
- If Na+ is >=105 mmol\L correct to125-130mmol\L
- If Na+ is<105 mmol\L correct by20 mmol\L maximum
- Rate of rising Na+ should not exceed 2-4mmol\L, 4 hourly or 20mmol\L every 24hours.
- For symptomatic hyponatremia (seizers) correct serum Na+ to 125 mmol\L over 2hours.
- Use hypertonic saline 3% if Na less than 120 mmol/each ml will contain 0.5 mmol

22.9.2.2 Hypernatremia:

Serum Na+ more than 150 mmol\L.

Cause of hypernatremia:

Diabetes, insipidus, Increased, insensible loss, High Na intake, NaHCO3 therapy etc.

Management guidelines:

- Give maintenance plus 1\2 deficit (or 75% maintenance + deficit over 24 hours) (Subtract boluses)
- Use 1\5th or 1\2 NS (Not water & Normal Saline)
- Lower serum Na+ by 10 – 15 mmol\day or (0.5 –0.75 mmol\hour)
- Normal hydration should be achieved over 36 – 48 hours and perhaps 72 hours if the initial plasma Na is > 170 mmol/L
- Monitor electrolytes 4-6 hourly & adjust fluid accordingly
- Treat hypo glycaemia& hypocalcaemia.

Complication of treatment:

- Cerebral oedema & seizure: 3% NaCl 4 ml\kg or mannitol.
- Pulmonary oedema: give diuretics
- Hypocalcaemia: add calcium gluconate 10 ml 10%
- Renal tubular injury & uremia.

22.9.2.3 Hypokalarmia

Serum K+ below 3.5 mmol\L.

Cause of Hypokalemia:

- Diuretics, Skin burn, G.I loss, Malnutrition etc

Clinical:

- Muscle weakness (paralysis), Smooth muscle (intestinal ileus, ureteric dilatation),
- Cardiac (arrhythmia and ECG changes: prolonged QRS, flat T-wave, ST depression, U -wave in ECG)

Treatment:

- In acute emergences (e.g. cardiac arrhythmia) give 0.5mmol\kg\hour in 20 mL of 5% dextrose over 30 min -1hour. Concentration should not exceed 80 mmol/L
- Otherwise put 30-40mmol\L in I.V fluids
- For oral therapy dose is 2-4 mmol\kg\24hours Bid or Qid

22.9.2.4 Hyperkalaemia

Serum k+ >5.5mmol\L in non-haemolyzed sample:

Cause of hyperkalemia:

- Transfusion of old blood,Tumor lysis syndrome,poisoning, Decreased Renal
- ECG changes: peaked T wave (tenting), wide QRS, ST depression

Management guidelines

- Ca gluconate 10%(2ml/kg/dose)max.100mg/min
- NaHCO3 1-2mmol/kg iv over 30 min, repeat if needed Insulin with glu cose 0.1Unit\kg With 0.5gm glucose\kg (2mlof 25%/kg).Over 30min utes followed by continues infusion of 0.1unit\kg\hour with dex trose 25% 1-2ml\kg\ hour or 10%dextrose 4ml\kg\hour
- Kayexalate 1gm\kg\dose POQ 6\hourly

22.9.2.5 Hypocalcaemia:

Total serum calcium below2mmol/l=8mg/dl in term newborns and older children or ionized calcium below 1 mmol/l IN PRETERMS; total below1.75 mmol/l

Cause of hypocalcaemia

- NEONATAL EARLY(0-72 HRS)preterm, LBW, RDS, asphyxia, acidosis, infants of diabetic mothers
- NEONATAL L ATE (3-7 DAYS)VD deficiency,Maternal hyperpara,Mg deficiency

- LATER IN CHILDHOOD VD deficiency, VD metabolism problems, hypoparathyroidism, calcium deficiency, hypomagnesaemia, high phos phate

Investigations

- Ca. phosphate alkaline phosphatase
- Radiology
- PTH(WITH SAME Ca sample)
- Vitamin D metabolites(draw separate and freeze)till senior or endo con sult

Treatment

Symptomatic

- 10%Calcium gluconate 2ml/kg iv Dilute 1:4 with 5%dextrose or water Give over 30 minutes ECG monitor(if possible) or clinically for bradycardia or even arrest
- Then start infusion of 1mmol/kg /24hours in 5%dex+1/5 saline or give the total daily dose divided as 6hourly infusions Monitor pt and calcium 6hourly

22.10: Neonatal Emergency

22.10.1 Neonatal resuscitation

- Risk factors are poor predictors of birth asphyxia.
    - o Up to half of newborns who require resuscitation have no identifiable risk factors before birth.
- Preparation is very important for any delivery
    - o Equipment
    - o Personnel
    - o Environment

Equipment

- A self-inflating ambubag (newborn size)
- Two infant masks (for normal and small newborn),
- A suction device (mucus extractor),
- A radiant heater (if available), prewarmed towels, a blanket
- Cord tie
- A clock

Personnel

- There should be a person to call for help who knows how to do neona tal resuscitation mostly when the mother delivers there is a need that helps the mother as well as the baby.
- The heath personnel should wash their hands, there should be also a commu nication system to get ambulance call phones or the next person to be called

Environment

- The room or ambulance doors and windows should be closed,
- It should be heated in advance

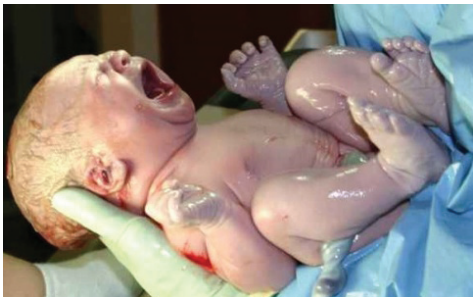


Figure 12. Normal newborn drying and warming

Figure 13. Floppy baby

Positioning:

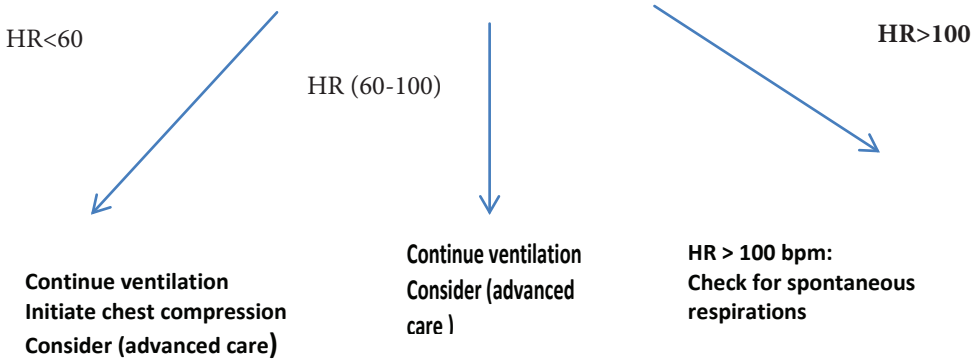
- Sniffing position, these bring the posterior pharynx, larynxand trachea inline and facilitate air entry.
- Put towel or blanket upper part of the shoulder
- Avoid hyperextension and or flexion of the newborn
- Clearing the airway with a transparent suction devise from mouth then \ to nose.
- It should be gentle not vigorous otherwise deep suctioning of the oro pharynx will induce respiratory depression

Bag Mask Ventilation

- Clearing the airway with a transparent suction devise from mouth then to nose.
- It should be gentlenot vigorous otherwise deep suctioning of the oro pharynx will induce respiratory depression

- Adequacy of ventilation is assessed by observing the chest movements
- After effectively ventilating for about 1 minute, stop briefly but do not remove the mask and bag and look for spontaneous breathing
- If there is none or weakspontaneous breathing, continue ventilating until spontaneous cry/breathing begins.
- If there is no chest rise, check the position of the baby or if there is any secretion blocking the airway, or if your mask is fitting well,

Ventilate for 30 seconds:
Rate: 40-60 /min
Pressure: Visible rise and fall of chest



- If the child is not breathing spontaneously and heart rate >100beats.
- Continue bag mask till advanced care arrives or for 20 minute

Chest compression

- If the AHR is <60 bpm despite adequate assisted ventilation
- In newborns with persistent bradycardia (heart rate <80/min and fall ing) despite adequate ventilation
- The (2-thumb, encircling-hands method) is preferred, with a depth of compression one third the anterior-posterior diameter of the chest and sufficient to generate a palpable pulse.
- 3 compression for 1 breath Two people are needed for effective chest compres sion and ventilation

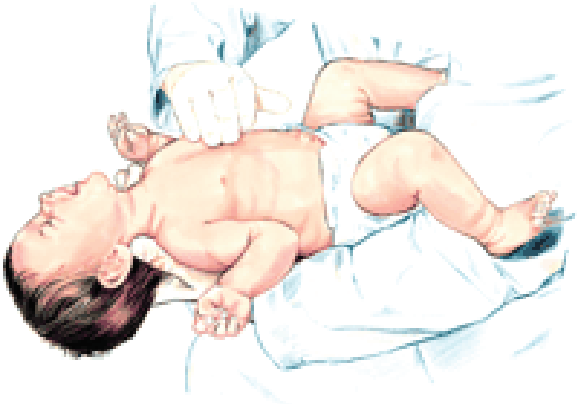


Figure 14. Two finger cardiac compression for single rescuer

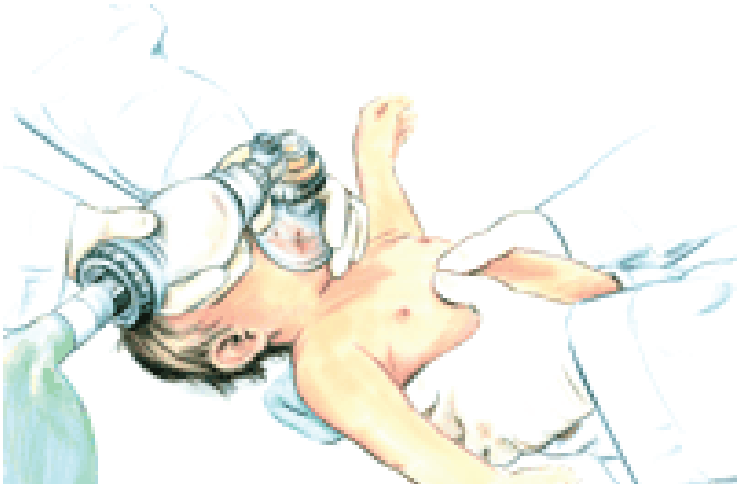


Figure 15. Thumb encircling for two rescuers 22.10.2 Neonatal seizures
Paroxysmal alteration in neurologic function (i.e. behavioural, motor,autonomic function).

22.10.2 Neonatal seizures

Paroxysmal alteration in neurologic function (i.e. behavioural, motor,autonomic function).

Etiology:'

- Hypoglycaemia
- Hypocalcaemia
- Hypomagnesaemia or refractory hypocalcaemia

Anticonvulsants:

Phenobarbitone:

- Loading dose: 20-30 mg/kg IV or IM over 15-30 min.
- Maintenance does: 2.5 - 4 mg/kg /day od or in 2 divided doses.

[For neonates <30 weeks 1-3 mg/kg/day]

Phenytoin:

Loading dose: 15-20 mg/kg/at a rate not>0.5 mg/kg/min.

Maintenance dose: 5-8mg/kg/day divided q 12-24h.

- P.O.: highly variable:-5-8 mg/kg/day to 8 mg/kg q 12h

Pyridoxine:

- Dose: 100-200 mg IV under EEG control. The seizure will abruptly stop and the EEG will normalize within few hours.

22.10.3 Respiratory distress syndrome (RDS)

Etiology:

- Prematurity
- Perinatal asphyxia
- Caesarean section
- Maternal diabetes

Clinical Signs:

RDS presents within four hours of birth:

- Sternal retraction, intercostal &sub costal recessions.
- An expiratory grunt.
- Tachypnoea above 60/min.
- Bilateral or unilateral decreased air entry

So common to all definitions of the disease is that the signs should present before fourhours of age, should still be there at four hours of age and should persist for someperiod beyond four hours of age.

Diagnosis:

- History
- Clinical signs
- Investigations:
- Radiology:
- **Atelectasis:** documented by the ground-glass pattern.
- Air bronchogram
- In severe causes the lungs cannot be separated from the cardiac border.
- Hb%, packed cell volume (PCV), WBC&Platelets count.
- Electrolytes, Creatinine, calcium (establishing base line).
- Blood gases (initiating treatment).
- Blood group and cross-match.
- Serum albumin.

Treatment:

The aim of treatment is to keep the newborn alive and in good condition until he starts tosynthesize his own surfactant 36-48 hours after birth. This means avoiding:

- Hypoxia.
- Acidemia.
- Hypothermia, which inhibits surfactant synthesis
- Put newborn on CPAP, maintenance fluid and start antibiotics

22.11: Poisoning

The clinician should maintain a high index of suspicion to be able to arrive at theoften difficult diagnosis of poisoning. Strongly consider ingestion in any patient withan unexplained loss of consciousness, especially if it is of a sudden onset in apreviously healthy child.

Management priorities:

Emergency stabilization of the patient comes first.

- Start by treating the patient, not the poison.
- C-A.B.s of resuscitation then add "D" for drugs used for relief of other symptomslike convulsions.
- Perform a brief neurological exam, establish the level of consciousness (Glasgowcoma scale), and determine pupillary size and reactivity.

- Institute an IV line, fluid therapy, drug therapy, oxygen, dextrose, as indicated.
- Consider decontamination: ocular, dermal, Gl, etc.

History:

- Symptoms complexes (toxidromes) may give clues to an unknown poisoning.

History aims to:

- Identify substance or substances including ingredients made in house.
- Identify the maximum possible amount (number in bottle originally, number left).
- Estimate ingestion, usually grossly under-estimated.
- Estimated time of ingestion.
- Symptoms.
- Home treatment.
- Glue exposure, recurrent episodes, etc.

Examinations:

- Vital signs.
- Level of consciousness (GCS), motor function.
- Eyes (pupils, EOM, fundi).
- Mouth (lesions, odor).
- Heart (rate, rhythm).
- Lungs (rate, pattern).
- Skin odors (breath, clothing).
- Can the patient maintain the airway? Does the patient have a gag?
- Major modes of presentation are coma, disturbances, and seizures.

Investigations:

- Blood glucose.
- LFT.
- CBC.
- Blood urea, creatinine & electrolytes (including calcium).
- Urine and blood for TOX screen and drug levels if intoxicant is known.
- Serum Osmolarity.

Treatment

Elimination of the poison:

- Inhaled poisons:

If smoke, gas or fumes have been inhaled carry the victim to fresh air.

- Poisons on the skins:

Remove the clothing and flood the involved parts with water. Wash withsoapy water and rinse thoroughly.

- Poisons on the eye:

Rinse out the eyes with plain tap water for 15-20 minutes Do not try to Neutralize acids or alkalis.

- Swallowed poisons:

Eliminate poison by:-

- Gastric lavage:

With the contraindications of induction of emesis, gastric lavage may beperformed after the introduction of a cuffed endotracheal tube. Gastric lavage is useful if it is performed within 1-2 hours of ingestion. Use a large28-36° F. Nasogastric tube is recommended, since smaller tubes are less effective. Lavage is best done with warm isotonic saline to avoid hypothermia especially ininfants. The amount instilled should approximate the amount removed. Emesis and lavage will remove about 30% of the amount of poison ingested.

Prevention of absorption of poisons:

This is best done by giving activated charcoal: Dose is 1-2 gm/kg (maximum100 gm), it might be repeated every 2-6 hours untilcharcoal is passed per rectum. Prepare charcoal as slurry of a ratio of 1: 4 charcoal to water.Consider for all significant toxic ingestions. It is poorly bound to iron and lithium, soit is not recommended for them. Do not use with caustic ingestion since it is poorlybound to them and it renders endoscopy difficult.

Enhancement of excretion:

This is achieved by:

- Forced diuresis

Used with pH modifications and it needs close monitoring for fear of toxicity.

- 1/2 -2 X maintenance (3000 cc/m2/day) of fluids is to be given.
- Urine output should approach 3-6 cc/kg/hr.
- 1-Alkalinization of urine is used with ingestions of Phenobarbital and salicylate use 0.5 -2 mg/kg/hour of IV $NaHCO_3$ titrate to keep urine pH 7.5-8.0.2-
- Acidification of urine :

Used for ingestions of amphetamine, chloroquine, lidocaine quinidine.

- Use ammonium chloride 75 mg/kg/day q 4-6 p.o. (contraindication, he patic insufficiency).
- Keep urine PH between 5.5 and 6.

o Dialysis(consult nephrology)

Dialysis has been used for many substances, some of which are:

Ammonia, amphetamines, anilines, antibiotics, barbiturates, boric acid, bromides,calcium, chloral hydrate, ethylene glycol, fluorides, iodides, isoniazid, meprobamate,methanol, paraldehyde, potassium, quinidine, quinine,salicylates, strychnine,thiocyanates.Dialysis is preferably by hemodialysis or peritoneal dialysis if hemodialysis is notavailable. It is part of the supportive care if the child is having any of the followingcriteria:-

Clinical criteria:

- Potentially life threatening toxicity caused by a dialyzable drug that can not bedealt with conservatively.
- Severe hypotension which is not correctable by adjusting circulatory vol ume.
- Marked hyperosmolarity or electrolyte or acid base disturbances not re sponding totherapy.
- Marked hypothermia or hyperthermia not responding to therapy.
- Dialysis is also indicated when: coma is deeper than level 3

Immediate dialysis:

- Is to be considered in ethanol and methanol poisoning if acidosis isrefrac tory or blood ethanol level is constantly above 100mg/dl.

Antidotes:

- Use of specific antidotes is invaluable, unfortunately few poisons have an tidotes.
- Contact poison control for specific antidotes and doses.

Disposition:

- May involve medical and/or psychiatric follow-up (psychiatric treatment may benecessary in certain patients, especially those with suicidal attempt

22.11.1 Hydrocarbon poisoning

Compound contains carbon and hydrogen Absorbed by:

- Skin
- Lung

- Gastrointestinal tract
- Kerosene ingestion:

o Risk of aspiration
o GIT & Respiratory effects.
o Burning sensation, nausea, belching and diarrhea
o Cough, chocking, gagging and grunting.
o CXR 2-8 hrs later: Pulmonary infiltrates or perihilar densities.
o pneumatoceles, pleural effusion or pneumothorax and bacterial superin fection
o Resolution 2-7 days.

Treatment:

- Do not induce vomiting
- Do not attempt gastric lavage
- Risk of aspiration outweighs any benefit from removal of substance
- CXR around 2-4 hrs "not before 2hrs"
- Observe in ER for 6-8 hrs if no symptoms è discharge.
- May develop pneumonia after 24 hours so appoint for follow up

22.11.2 Organophosphate

Primary toxic effects involve the autonomic nervous system, neuromuscular junction, and central nervous system (CNS)

- The parasympathetic nervous system is particularly dependent on acetyl choline regulation
- Both the autonomic ganglia and the parasympathetic nervous system are regulated by nicotinic and muscarinic cholinergic receptor subtypes, re spectively
- The muscarinic signs can be remembered by use of one of two mnemon ics: SLUDGEBB –(salivation, lacrimation, urination, defecation, gastric emesis, bronchospasm, bradycardia)
- DUMBELS (defecation,urination,miosis, bradycardia,emesis,lacrimation ,salivation)
- Stimulation of nicotinic receptors

Release of epinephrine and nor epinephrine, muscleweakness, fasciculation hypertension, central respiratory depression, lethargy convulsion and coma

Clinical presentation

- Depends on the balance between stimulation of muscarinic and nic otinic respecter
- The balance depend on the
    - o Type of organophosphate
    - o Dose
    - o Route and rate of absorption
    - o Individual factor

Laboratory abnormalities

- Direct measurement of RBC acetylcholinesterase (RBC AChE) activity provides a measure of the degree of toxicity
- Hypokalemia, hyperglycemia,leukocytosis, proteinuriaglycosuria
- ECG sinus tachycardia
- Depressed RBC cholinesterase level

Diagnosis

- The diagnosis of organophosphate poisoning is made on clinical grounds
- If doubt exists as to whether an organophosphate has been ingested, a trial of atropine 0.01 to 0.02 mg/kg may be employed
- The absence of signs or symptoms of anticholinergic effects following atropine challenge strongly supports the diagnosis of poisoning

Treatment

- Atropine 0.02 mg / Kg IV. Repeat as needed and titrate to respiratory secretions. It will likely take massive doses!!
- Pralidoxime (2-Pam) 20-40 mg / Kg bolus followed by 10-20 mg / Kg / hour infusion.
- Remember to send RBC and Plasma Cholinesterase levels upon arrival and daily.

22.12 Snake Bite

- More than 400 different species of snakes occurring in the African con tinent, only 90of these has venomous bites, of them only 30 different species are known to havecaused death.
- Snake bites can be deadly. It's important to react quickly to bites.
- It is worth knowing that different snakes have different systemic effects and thecause of death is mainly respiratory depression.

Pathophysiology:

- Venom is mostly water. Enzymatic proteins in venom impart its destructiveproperties. Proteases, collagenase, and arginine ester hydrolase have beenidentified in pit viper venom.
- Neurotoxins comprise the majority of snake venom.

Specific details are known for several enzymes as follows:

- Hyaluronidase allows rapid spread of venom through subcutaneous tissuesby disrupting mucopolysaccharides.
- Phospholipase A2 plays a major role in hemolysis secondary to the osteolytic effect on red cell membranes and promotes muscle necrosis.
- Thrombogenic enzymes promote the formation of a weak fibrin clot, which,in turn, activates plasmin and results in a consumptive coagulopathy and itshemorrhagic consequences.

Laboratory tests:

- Baseline and serial laboratory tests are critical.
- B. Group and cross match of blood; complete blood and platelet counts; Prothrombinand partial thromboplastin times; fibrinogen and fibrin degradation products; bloodurea, creatinine, electrolytes and creatinine phosphokinase.

Clinical Effects:

- Clinical manifestations depend on many variables including victim (age, generalhealth and size), snake (species, glands and fangs) and bite (number, location, depthand amount injected poison).
- Envenomation grading determines the need for antivenin in victims of snakeenvenomations. Grades are defined as mild, moderate, or severe.

Mild envenomation is characterized by

- local pain, edema, no signs of systemic toxicity
- Normal laboratory values.
- Moderate envenomation is characterized by severe local pain; edema largerthan 12 inches surrounding the wound; and systemic toxicity includingnausea, vomiting, and alterations in laboratory values (e.g. , decreasedhematocrit or platelet count).
- Severe envenomation is characterized by generalized petechiae,ecchymosis, blood-tinged sputum, hypotension, hypoperfusion, renaldysfunction

,changes in prothrombin time and activated partialthromboplastin time, and other abnormal test results defining consumptivecoagulopathy.

The crude clotting time is helpful and practical.
In most severe cases there is generalized oedema, shock, cardiac arrhythmias and death.

Evaluation and Treatment:
ABC's FIRST!! And evaluating the patient for signs of shock (e.g., tachypnoea,tachycardia, dry pale skin, mental status changes, and hypotension).

Important warnings:

- Do not try to suck out the venom.
- Do not attempt to cut open the area around the bite.
- Do not apply ice to the bite area.
- Do not rub any substances into the bite.
- Do not give anything orally to the victim.
- Do not inject anything, including antivenom unless you arequalified to do so. Anyone prone to allergies and asthma may gointo anaphylactic shock.

First aid manoeuvers should attempt to impede local lymphatic flow, the patient should be:

- Placed at rest with local pressure and immobilization of the extremity.
- Close monitoring and large-bore IV access should be established
- Start a broad spectrum antibiotic.

Antivenin:

- The decision to use antivenin should be based on the severity or rapidprogression of the symptoms. It is most effective when given within 4 hours of thebite.
- Surgical assessment is essential as it focuses on the injury site and concern for thedevelopment of compartment syndrome which may need fasciotomy, monitoring of compartment pressure and follow up of the necrotic tissue.

Section 23 Obstetric and Gynecologic Emergencies

23.1: Hypertensive Disorders during Pregnancy

Hypertension(HTN): Systolic blood pressure (SBP) >140 mmHg or diastolic blood pressure (DBP) > 90 mmHg on two occasions 6 hours to 7 days apart; Or one BP > 160/110 mmHg.

Proteinuria: 24-hour urine specimen with 0.3 g protein or >1+ dipstick or >100 mg/dl two random urine sample, collected at least 4 hours apart.
Diagnosis, Classification and Clinical features:

1. **Gestational HTN:** HTN without proteinuria or other signs of preeclampsia

2. **Preeclampsia:** HTN and proteinuria after 20 weeks. Can be mild or severe. Severe if:
BP>160/110mmHg or urine protein > +3 or >5 gm /24 hr. urine
Severe symptoms: headache, visual changes, epigastric pain, severe edema, IUGR, abruption placenta, oliguria, DIC (bleeding, petechia), HELLP syndrome (hemolysis, elevated liver enzymes and low platelets), severe nausea and vomiting, low platelets < 100,000/mm3, altered liver and renal function tests.

3. **Eclampsia:** grand mal seizures or coma in a woman with preeclampsia. Hypertension in pregnancy includes eclampsia, a medical emergency. In eclampsia, mobilize all staff, make a rapid ABC assessment, control hypertension, collect relevant information, start MgSo4 to stop convulsions and prevent subsequent convulsion. Rule out other causes of convulsions.

4. **Chronic HTN:** HTN antedates pregnancy. Get superimposed preeclampsia/ eclampsia if:new proteinuria after 20 weeks GA; or sudden increase in preexisting proteinuriaan exacerbation of blood pressure to the severe range or severe symptoms.

Diagnostic tests and Procedures
Helps in diagnosis and determine severity of the disease.Hematocrit, urine protein, serum creatinine, uric acid, ALAT, ASAT, bilirubin, platelet count, LDH, PT, aPTT, type and crossmatch non-stress test and ultrasound.

Management of different stages of PIH

Gestational Hypertension

Check blood pressure and protein. Refer for outpatient management. If BP remains in mild range, proceed with normal labour and childbirth before post term.

Table 20. Eclampsia and Pre-eclampsia

	Mild/Severe Pre-eclampsia	Eclampsia
When to deliver	If mild, deliver at 37 weeks (refer for outpatient follow up) If severe, GA > 34 weeks or end organ damage: deliver immediately. If < 34 weeks: give steroids, observe as inpatient, deliver at 34 weeks	Immediately (should be in < 12 hours); deliver by C-section if vaginal delivery not possible in this time Transfer patient if needed
Initial Measures	ABCs	ABCs; apply oxygen; give prophylactic IV antibiotics
When to give seizure prophylaxis	During evaluation, labour and continued for 24 hrs after delivery	Immediately; continue until 24 hours after delivery or last seizure
General Measures when admitted	Monitor urine output (goal >30ml/hr.), fluid balance; vital signs, FHB, reflexes and signs of pulmonary edema. ,	
Acute blood pressure control (goal DBP 90-100 mmhg and SBP 140-150 mmHg)	1st line: Hydralazine: 5-10 mg IV every 30 minutes until DBP <110mmhg. Repeat if not controlled (or 12.5mg IM every 2hrs). 2nd line: Nifedipine: 5-10 mg sublingually, then 5-10mg in 30 minutes if response inadequate. Then 10-20 mg PO every 6 hours (10-40mg PO bid maintenance). 2nd line: Labetalol: 20 mg IV push over 2 minutes. Repeat as needed every 10 minutes, doubling the dose up to 80 mg for desired effect. Maximum total cumulative daily dose is 300mg IV.	
Seizure Prophylaxis: Note:Magnesium (MgS04) is critical to stopping and preventing convulsions.	Magnesium (MgS04) Before administration: ensure RR > 16/minute, DTRs present Loading dose: MgS04 (20% solution) 4 g IV over 5 minutes. Then 10 g 50% MgS04 IM, 5 g in each buttock. If convulsions recur after 15 minutes, give 2 g MgS04 (50% solution) IV over 5 minutes. Maintenance: 5 g MgS04 (50% solution) + 1 mL lignocaine 2% IM every 4 hours into alternate buttocks If respiratory arrest: assist ventilation, give calcium gluconate 1 g (10 mL of 10% solution) IV slowly until respiration begins. Diazepam: Second line. Risk fetal or maternal respiratory depression Loading dose: 10 mg IV over 2 minutes, if convulsions recur, repeat loading dose and 40 mg in 500 ml IV fluids; drops titrated to keep woman sedated but arousable Can give rectally when IV access is difficult: Loading dose of 20 mg followed by maintenance dose of10mg/hr.	

23. 2: Management of Labor and Delivery

Rhythmic uterine contraction leading to cervical dilatation and expulsion of the fetus.

Diagnosis: History and Physical Examination

History: Ask about uterine contractions, leakage of fluid, vaginal bleeding, fetal movement. Take vital signs, check uterine contraction and FHR. Gravidity, parity, GA (in weeks), time contraction started, frequency; leakage of liquor, bleeding, past medical illnesses.

Physical: Leopold maneuver for GA and fetal presentation; sterile digital vaginal examination if no vaginal bleeding to assess the stage of labor.

Investigation

If not already done, Hgb, Blood group and Rh, U/A and microscopy, RVI

Management during 1st stage

Record all observations and findings on the partograph.

Maternal wellbeing monitoring

If patient in 1st stage or has high risk pregnancy (requiring obstetrician), immediately transfer to OB delivery unit. If unable to transfer or imminent delivery, do the following:

Vital signs: every half an hour.

Pain management - continuous emotional support, use analgesia safe for mother and fetus without effects on progress of labour.IV, IM or SC opioids or epidural analgesia.

Fetal Wellbeing monitoring

FHR: Pinnard stethoscope or continuous FHR monitoring. Do for 1 min after contraction; every 30 min if low risk pregnancy, 15 min if high risk.Monitor liquor for meconium: if moderate meconium, likely fetal distress; if thick, definite fetal distress.

Monitor Progress of labour:
1. Uterine contractions – track frequency, duration and intensity (by palpation or toco-dynamometer); monitor q1 hr. for latent phase and q30 min for active phase.

2. **Descent of fetal head:** determine by abdominal palpation and vaginal exam q4 hours.

3. **Vaginal examinatio**n (every 4 hours). Assess: cervical dilation, (normally progresses ≥1 cm/hr.), station, position, caput and molding.Management of 2nd stage

Normal duration of 2nd stage: Nullipara: < 2hrs without or 3hrs with epidural anesthesia. Multipara: < 1hrs without or 2hrs with epidural anesthesia. If longer, is prolonged 2nd stage.

Maternal wellbeing monitoring
Evaluate: general condition, pain, hydration, vital signs every 30 minutes
Empty bladder, avoid early pushing, LLP untilhead is visible.

Fetal Wellbeing Monitoring
Every 15 min and 5 minutes for low and high risk fetuses respectively.

Monitoring of Labour Progress
Evaluate uterine contraction every 10 minutes and the descent every 1 hour.

Assistance of spontaneous delivery
Do episiotomy only if perineal resistance or if expedited delivery indicated.

Timing: When head distends vulva 2-3cms. Types: median or mediolateral (mediolateral is preferable). Local anesthesia used.

Assist delivery of head using modified Ritgen's maneuver if extension does not occur easily i.e., hand protected with sterile towel placed on the perineum, fetal chin palpated and pressed upward gently effecting extension.

Check for cord around neck: if present gently move over the head. If not reducible: deliver without reduction or clamp at two sites and cut in between.

After delivery of head: wipe mouth and oro-pharynx (routine suctioning not recommended). Allow for restitution of head. Then, place a hand on each parietal eminence. Apply gentle downward pressure of the head toward the maternal sacrum to deliver anterior shoulder. Then, deliver posterior shoulder by upward traction.

Put fetus at level of introitus for 3 min. Immediately dry the body. Clamp cord 4-5 cm from fetal umbilicus. Delayed clamping (after a few minutes) associated with increased neonatal hematocrits. Take cord blood if indicated.

If second stage prolonged, manage the causes:

Poor maternal pushing effort: consider vacuum or forceps delivery.
Poor uterine contraction: oxytocin.

CPD: cesarean delivery.
If cord prolapse is detected at any stage of labor and the cord is pulsating:
Put the mother in knee chest position, support the fetal head with one hand in the vagina during transportation until the women is on the operating theater table and ready for C/S

Management of third stage of labour.
Provide active management of third stage of labour (AMTSL) for all patients:

Administer Uterotonic agents immediately after fetal delivery (1st choice: oxytocin 10 IU IM; 2nd choice: Misoprostol 600 mcg orally; 3rd choice: ergometrine 0.2mg IM or syntometrine (1 ampoule) IM). Apply controlled cord traction and use uterine massage (immediately after the placental delivery).

23.3: Shoulder dystocia

Shoulder dystocia is inability to deliver the shoulders after the fetal head has been delivered despite the performance of routine obstetric maneuvers. It is an acute obstetric emergency requiring prompt, skillful management to avoid significant fetal damage and death. Higher risk of occurrence during delivery of macrosomic (>4kg) babies, but shoulder dystocia may not be predicted. Be prepared for

shoulder dystocia at all deliveries, especially if a large baby is anticipated and in women with diabetes mellitus, previous history of macrosomic babies and obesity. Asphyxia, birth injuries, injury to the brachial plexus and maternal PPH are some of the complications.

Diagnosis

The fetal head is delivered but remains tightly applied to the vulva, the chin retracts and depresses the perineum. Traction on the head fails to deliver the shoulder, which is caught behind the symphysis pubis.

Management

In the lithotomy position, ask the woman to open and flex both thighs, bringing her knees as far up as possible towards her chest. Ask two assistants to push her flexed knees firmly up onto her chest (McRobert's maneuver).Apply firm (not excessive), continuous traction downwards (towards the floor) on the fetal head while an assistant simultaneously appliessuprapubic pressure downwards to assist delivery of the anterior shoulder. If not successful, continue with maneuvers below. Episiotomy should be performed at any point if needed to create adequate space for maneuvers.

Insert a hand into the vagina and apply pressure on the back surface of the anterior shoulder in the direction of the baby's sternum to rotate the shoulder and decrease the width of the shoulders; if needed, apply pressure to the back of the posterior shoulder in the direction of the sternum (Rubin's maneuver).

Alternatively, one can try applying pressure to the anterior surface of the posterior shoulder until the baby turns and the anterior shoulder emerges from underneath the pubic symphysis. (Woods screw maneuver)

If the shoulder still is not delivered despite the above measures, insert a hand into the vagina; grasp the humerus of the posterior arm. Keeping the arm flexed at the elbow, sweep the arm across the chest. This will provide room for the shoulder that is anterior to move under the symphysis pubis.

If the posterior arm cannot be reached, apply traction with a hook in the axilla to extract the shoulder that is posterior; this may bring the posterior arm down sufficiently to be grasped and delivered.

In patients with only local or pudendal anesthesia, an effective initial maneuver may be to rotate the woman to a position of hands and knees and attempt delivery in this manner. (Gaskin all-fours maneuver)

If all of the above measures fail, the last option is to fracture the clavicle (will decrease the width of the shoulders and free the anterior shoulder); apply traction with a hook in the axilla to extract the arm that is posterior. Replacing the head and doing C/S is rarely successful.

After this is done, repeat all above maneuvers with the newly shortened bi-acromial diameter.

23.4: Breech Presentation and Delivery

Fetus presenting by the buttock and/or the lower extremity.

Diagnosis: History and Physical Examination
The history may reveal discomfort under the rib (due to the hard head). On abdominal palpation (Leopold's maneuvers), may feel a round, hard, smooth mass (head) occupying the fundus. A soft, broad, indefinite and non ballotable mass (the breech) occupying the lower pole of the uterus. FHB loudest just above the umbilicus.On vaginal examination: three types of breech presentation can be identified:

1. Frank breech: buttocks in the pelvis, both legs extended.

2. Complete breech: one or both feet are felt alongside the buttock.

3. Footling breech: one or both feet are inferior to the buttock.
Ultrasound confirms the clinical diagnosis of breech and predisposing factors.

Investigation
Hgb and Blood group and Rh, Ultrasound

Treatment Plan
If preterm and not in labour, refer to OBY for expectant management; may turn cephalic. If GA > 37 weeks of gestation and if there is no contraindications for external cephalic version (ECV), consider ECV. If the ECV fails, consider breech

vaginal delivery. ECV: baby manipulated through mother's abdominal wall into cephalic presentation.If in advanced labour do breech vaginal delivery. Do cesarean delivery only for footling breech (increased risk cord prolapse + arrest head) or obstetric indications like APH, etc.

Induction or augmentation of labour is contraindicated in breech presentation.

Types of vaginal breech delivery:

1. Spontaneous breech delivery: The baby is expelled entirely spontaneously.

2. Total breech extraction: Deliver by extracting the entire body of the fetus from the uterus; rarely indicated except to expedite the emergent delivery of a second twin.

3. Assisted vaginal breech delivery: Partial breech extraction if no cord prolapsed or entanglemen

Delivery of the breech, abdomen and shoulder:

Keep in lithotomy position and empty her bladder. Provide emotional support, pudendal block and local infiltration, do episiotomy when the fetal anus is visible, instruct the mother to bear with contraction and allow the buttocks delivered spontaneously and shoulder blades are seen (no other manipulation until delivered up to umbilicus)

If the legs are not delivered spontaneously, assist delivery of one leg at a time, by lateral rotation of thighs and flexion of knees using a finger.

Hold the baby by the hips with towel (not by the flanks or abdomen as this may cause organ damage) with fingers over anterior superior iliac spine and the sacrum. Apply gentle, steady downward traction with good maternal pushing until the lower half of the scapula is delivered.

Allow the arms to disengage spontaneously one by one; only assist if necessary. After spontaneous delivery of the first arm, lift the buttock towards the mother's abdomen to enable the second arm to deliver spontaneously. If the arm is not spontaneously delivered, place one or two fingers in the elbow and bend the arm, bringing the hand over the baby's face.

If arms are stretched above the head or folded around the neck, use the Lovett's maneuver.

Delivery of the head:
Mauriceau Smellie Veit Maneuver (MSV): Lay the baby face down with the length of its body over your hand and arm, with the mid and forefingers of your hand on the baby's cheekbones. Pull down to flex the head while the other hand grasps the baby's shoulders. With two fingers and middle finger placed and pushing over the suboccipiut (hooking round the neck), gently flex the baby's face towards the chest, while applying down ward pressure on the shoulder. Pull gently downward until the hairline is visible while an assistant applies suprapubic pressure. Once the head gets into the pelvis, following the pelvic curve, raise the baby until the mouth and nose are delivered.

Forceps delivery if MSV fails.

23. 5: Obstructed Labor and Ruptured Uterus

Obstructed labour (OL) is failure of descent of the fetus in the birth canal for mechanical reasons in spite of good uterine contractions. OL is a neglected labour and should not occur in a labour ward. OL is an emergency condition and requires a concerted team approach.

Causes
Cephalopelvic disproportion (CPD): small/abnormal pelvis or large fetus. Congenital fetal abnormalities, locked twins, shoulder dystocia, fetal malpresentations and positions, or abnormal reproductive tract.

Diagnosis
History of labour for days, maternal exhaustion, anxiety, confusion, or unconsciousness, low BP, rapid PR and RR.

Abdominal examination:
Fetal head above the pelvic brim, abnormal or no fetal heart tone. Distended and tender abdomen, Bandl's ring. The 'three tumor abdomen" indicates an impending uterine rupture.

In ruptured uterus (common n multiparous women), the findings include: Shock, hemoperitoneum, tender abdomen, easily palpable fetal parts. On vaginal examination: foul smelling discharge, significant capute and molding, edema of the vulva and the causes of OL.

Complications:
Maternal: PPH, sepsis, urinary and rectal fistula, nerve injuries, uterine rupture, etc.Fetal/neonatal: fetal death, asphyxia, sepsis, etc.

Treatment
Simultaneously start resuscitation while identifying the cause and treating infection In case of ruptured uterus, laparotomy is indicated.Once OL is diagnosed, C/S is the rule. No place for instrumental vaginal delivery. Destructive delivery (craniotomy) is an exception used only when the fetus is dead, there is no sign of uterine rupture or impending rupture, OL is in 2nd stage with engaged head, and a place with no operating room facility.

23.6: Emergency Cesarean Section(C/S)

Cesarean section is the delivery of the fetus (es), placenta and membranes through an incision of the abdominal and uterine wall at ≥ 28 weeks of gestation. It can be elective or emergent. Uterine incision either lower (most common) or upper segment. Abdominal incision either midline (for fast delivery) or Pfannelstein.

Indications: can be obstetric or non-obstetric.

Anesthesia: usually regional (spinal/epidural); general with certain indications. If neither available nor safe, local analgesic infiltration may be used.

Procedure
Skin incision. Types include:
Pfannensteil incision and Subumbilical midline incision. Make a small incision over the fascia with a scalpel; extend to the whole length of fascia with scissors. Dissect the rectus and pyramidalis muscles by sharp instrument followed by blunt dissection.

Elevate the peritoneum at the upper edge of the incision by holding it with two artery forceps about 2 cm apart. Palpate the tent of peritoneum to check for omentum or bowel. If present, release the artery forces and grasp again.

Incise between the two artery forceps with scalpel to open the peritoneal cavity. Check for adhesion of the peritoneum or dense infiltration by inserting a finger and palpating up and down the peritoneal opening. Extend the peritoneal opening with scissors up to the upper border of the incision and downward to the reflection of the bladder checking for adhesions.

Correct the uterus if dextro-rotated. Insert moistened packs on each side of uterus and insert bladder retractor.

Lower segment transverse cesarean section:
Grasp the peritoneal flap at the site of the reflection with forceps and incise with scissors. Dissect the peritoneal flap at the reflection site be inserting the scissors between the serosa and myometrium. Open up your instrument to dissect the peritoneum and then cut moving to the left and right side of the uterus (The assistant moves the bladder retractor to the side you are moving your scissors). Push the peritoneum downwards with gauze on a holder or using your fingers.

Incise transversely over the exposed uterine lower segment for about 2 cm with a scalpel. The incision should be just enough to cut through the myometrium but should not reach the fetal parts. Extend the incision bluntly with your index fingers of the two hands laterally and upwards. Rupture the amniotic membrane if encountered.

Remove the bladder retractor. Then insert your right hand between the symphysis pubis and the presenting part and elevate the head (in case of vertex, brow or face presentation) gently through the incision assisted by gentle abdominal pressure. Wipe the nares and mouth once the head is delivered. Deliver the rest of the body. The anesthetist administers uterotonics (oxytocin 10 IU IM or 20 IU in 1000ml NS).

Clamp the cord at two sites and cut in between. Hand over the neonate to the midwife for immediate newborn care.

Give prophylactic antibiotics: One dose of a broad spectrum antibiotic (ampicillin 2gm IV or a first-generation cephalosporin) IV is given immediately after the cord is clamped.

Deliver the placenta by cord traction. Clean the uterine cavity with pack to ensure completeness of the placenta and membranes.

Clamp the edges of the uterine incision and any briskly bleeding sites with green armitage or ring forceps. Lift the uterus out of the abdominal cavity and cover the fundus with moist pack. Close the uterine incision with two layers of continuous inverting stitches starting from the edge the first bite just behind the edge with

Chromic 1- or 0-catgut or polyglycolic (Vicryl). Replace back the uterus into the abdominal cavity. Make sure hemostasis is secured and uterus is well contracted. Dry the abdominal cavity with gauze pack if there is grossly contaminated amniotic fluid or meconium.

Close the fascia with continuous Vicryl no 2, approximate the subcutaneous layer with chromic 2-0 catgut, close the skin with continuous subcuticular stitch or interrupted silk as needed. Ensure uterine contraction and clean any clot in the vagina.

23. 7: Antepartum Haemorrhage (APH)

Antepartum haemorrhage is vaginal bleeding that occurs after 28 weeks of gestational age and before the delivery of the fetus.

Diagnosis: History and physical Examination
Estimate gestational age, assess amount of visible bleeding. No digital vaginal or speculum exam until placenta previa ruled out. Vital signs, ask about the duration, amount and antecedent event.

Investigation
Hgb, Blood group and Rh, Ultrasound exam
Specific Causes and Management
1. Placental causes: Abruptio placentae, Placenta previa, Vasa previa(rarely).

2. Uterine rupture

3. Local causes of the cervix, vagina and vulva

4. Preterm labour/bloody show

5. Indeterminate: no cause identified even after delivery and examining the placenta.

Abruptio Placentae
Abruptio placenta is a premature separation of the whole or part of a normally implanted placenta. Complications include: hemorrhagic shock, DIC, and utero-placental insufficiency (UPI) that may lead to IUGR, fetal distress or IUFD.
Diagnosis (Table 20)

Treatment
Secure IV with crystalloid. Delivery is the definitive management. In milder abruption and remote from term expectant management (admission, steroid adminstration) to prevent prematurity.Moderate and severe abruption (irrespective of gestational age) and abruption at term (irrespective of degree) immediate delivery. Mode of delivery is vaginal. Cesarean section is indicated for severe bleeding endangering maternal life and fetal distress, when vaginal delivery seems unlikely within a reasonable time and for other obstetrics indication. Coagulation defect must be corrected early.

Placenta Previa
Placenta previa is the presence of placental tissue lying adjacent to or overlying the internal cervical.

Placenta previa is classified based on nearness of the placental edge to internal-os of the cervix: Low lying placenta, Marginal placenta previa, and Major placenta previa (may be partial or total).

Diagnosis: Clinical (See table 20) and ultrasound if available.

Treatment
Delivery is the definitive management of placenta previa. In cases of mild and non-recurrent bleeding, do conservative management to prevent prematurity. Mode of delivery: Cesarean section. Vaginal delivery may be considered if low lying placenta.

Local Causes
All local causes of APH have minimal spotting or bleeding. An exception to such a presentation is the occasional profuse bleeding of ruptured vaginal varicose vein. Once placenta previa is excluded, digital and speculum examination may confirm the specific local cause.

Table 21: Clinical Findings in Placenta Previa and Abruptio Placenta

Clinical Findings	Placenta Previa	Abruptio Placentae
Vaginal bleeding	Painless Causeless Recurring(often) Bright red	Painful Presence hypertension, trauma, etc Non-recurring Menstrual like
Hypotension	Proportion to vaginal blood loss	Degree of hypotension out of proportion to amount of vaginal bleeding
Uterus	Quite or relaxed between labour contractions	Irritable, not relaxing between labour contractions (tetanic contraction),
Fetal presentation	Mal-presentation (transverse, breech), unengaged head	Difficult to palpate fetus Engaged head
Fetal condition	Usually normal fetal Condition	Fetal distress, Fetal death, IUGR

23. 8: Post-Partum Haemorrhage (PPH)

Post-partum haemorrhage is vaginal bleeding > 500ml after singleton vaginal delivery of >28 weeks (if cesarean delivery or multiple vaginal birth, bleeding >1000ml).

Diagnosis: History and Physical Examination
Vital signs, estimate the blood loss, uterine size and extent of contraction, completeness of the placenta.

Atonic Uterus (Uterus not contracted)
The most common cause of primary PPH.

Hypotonic uterus leads retention of the placenta and excessive bleeding.

Diagnose: if soft, not contracted uterus with fundus above the umbilicus.
Retained placenta

The common cause of placental retention is poor uterine contraction.
In retention of the placenta without bleeding, pathological adherence (accreta,

increta and percreta) should be considered. Manual removal of the placenta has to be done in the operating room with all the preparation for laparotomy and possible hysterectomy.

Traumatic causes
Risk factors for tears of the birth canal (including uterine rupture) and PPH: Fetopelvic disproportion (leading to obstructed labour), instrumental deliveries and scarred uterus.

Diagnosis: bright red (arterial) bleeding despite a contracted uterus.
Coagulation defects

Risk factors: abruption placenta, intrauterine fetal death, infection etc.

Physical examination: gross haemostatic failure is revealed.

Bed side clotting tests and deranged laboratory coagulation profiles support the diagnosis.

Acute inversion of the uterus
The uterus may rarely turn inside-out during delivery. Causes shock by bleeding or neurogenic shock due to increased vagal tone from stretching of the pelvic parasympathetic nerves.

With the placenta detached, is described as cherry red mass.

Management of PPH
Once PPH is diagnosed, shout for help and gather the team, immediately initiate resuscitation, and perform diagnostic and treatment activities promptly. For all cases ABC, wide bore IV on both hands, run crystalloids fast, cross match blood.

1. Retained placenta: PPH with undelivered placenta:

Apply controlled cord traction (CCT)

If CCT fails, manual removal of the placenta in operating room

Consider laparatomy for possible pathological adherence if both fails.

2. Atonic uterus: if PPH with delivered placenta and atonic uterus: Stimulate contraction by massaging the uterus.Start oxytocininfusion (20IU/1000ml, 30drops/minute).If there is no response, perform bimanual compression of the uterus; consider compression of the abdominal aorta. Administer other uterotonics such as misoprostol (800mcg sublingual) or ergometrine 0.4mg IM. If persistent bleeding, consider uterine tamponade with intrauterine balloon or condom tamponade (condom tied to end of Foley catheter, inserted into uterus, and filled with 350ccs NS). If there is no response subsequent management involves laparotomy uterine or utero-ovarian artery ligation, or hysterectomy.

3. Genital trauma: if PPH with delivered placenta and well contracted uterus:Explore the genital tract manually and using speculum and repair vaginal/cervical tear; if uterine rupture detected laparatomy is indicated.

4. Clotting abnormality: Correct with fresh frozen plasma or whole blood.

5. PPH after acute inversion of the uterus: under appropriate analgesia, apply:Immediate gentle upward transvaginal pressure. The Johnson technique calls for lifting the uterus and the cervix into the abdominal cavity with the fingers in the fornix and the inverted uterine fundus on the palm. Gently push the fundus back through the cervix. The operator's hand should be kept in the uterus until the fundus begins to climb up. If the placenta is still attached, it should not be removed until after the uterus is replaced through the cervix. Tocolysis may be used. Oxytocin only after successful replacement. If this fails unsuccessful (in delayed recognition) laparotomy for abdominal replacement is indicated.

23.9: Vaginal Discharge

Presence of vaginal discharge different from the normal cyclic discharge.Vaginal discharge is a common complaint. Emergency physicians must identify dangerous causes of discharge, and provide appropriate treatment for all types of vaginal discharge.

Diagnosis: History and Physical Examination
Is there abdominal pain, itching, or dysuria? Last menstrual period?Sexual history?Oral contraceptive use? Bleeding with intercourse? History of diabetes or HIV?Any recent antibiotic use (risk factor for candida)?Check for: vital signs, abdominal pain, cervical motion tenderness (CMT), flank pain, ulcerations or

lesions on the genitalia, discharge on speculum exam (physiologic discharge: thin, clear or white, without foul odour; pain, or pruritis). If haemodynamically unstable, place IV and give fluids. Consider pelvic inflammatory disease (PID), ectopic pregnancy, and other causes of shock. Children: do external genitalia exam. Check for signs of abuse or foreign body.

Possible Causes and Differential Diagnosis

Infection: Bacterial Vaginosis (overgrowth of vaginal bacteria); candidiasis (overgrowth of yeast); trichomoniasis (sexually transmitted protozoal infection); cervicitis or pelvic inflammatory disease (PID) (often due to gonorrhoea or chlamydia). Other: retained foreign body; atrophic vaginitis; allergic or contact dermatitis; chemical irritation; severe condylomata acuminate; malignancy. In children: above causes, or nonspecific vulvovaginitis, pinworm, enteric flora, or foreign body; sexually transmitted diseases may occur due to sexual abuse.

Investigations

Urine pregnancy test; urinalysis if dysuria or urinary frequency. Vaginal pH: apply pH stick to vaginal side wall (not posterior secretions), pH of normal secretions in menstruating females 4-4.5. Microscopic analysis of discharge KOH Whiff test: apply KOH to discharge. Fishy odor = bacterial vaginosis. Vaginal discharge culture for GC; PCR for Chlamydia (do not delay treatment for resul

Treatment:

Table 22. Treatment of different causes of vaginal discharge

Condition	Clinical Features	Management
Pelvic inflammatory disease	Patients may be febrile and tachycardia, or have normal vitals. May have associated vomiting or pain. CMT with adenexal or uterine tenderness;	If haemodynamically unstable: give IV fluids Antibiotics: Ceftriaxone 250 mg IM x 1, cefexime or ciprofloxacin for gonorrhoea (ceftriaxone preferred if high local resistance) AND– Doxycycline 100 mg PO BID x 14 days for Chlamydia –AND– Metronidazole 500 mg TID x 14 days for anaerobes
Cervicitis	purulent cervical discharge, friable cervix on exam, bleeding of cervix with intercourse or exam, or partner with vaginal discharge	Treat for both gonorrhoea and Chlamydia: Gonorrhea: ceftriaxone 250 mg IM x1, cefexime or ciprofloxacin (ceftriaxone better if high resistance) Chlamydia: Azithromycin 1000 mg PO x1 or Doxycycline 100 mg PO BID x 14 day
Candidiasis	Vaginal itching + discharge (white, curd like) without abdominal pain; budding yeast or pseudohyphae on microscopy	fluconazole 150 mg PO x 1 or topical and vaginal suppository of antifungal (eg - nystatin or miconazole) x 3-7 days follow up if symptoms do not resolve; Needs HIV testing if recurrent
Bacterial vaginosis (bv) and trichaomoniasis	Non-purulent discharge without pain; pH > 4.5 Microscopy: clue cells in bv; mobile trichomonads in (trichamoniasis)	Metronidazole 2000 mg PO x1 Follow up if not resolved in 1 week Recommend STD testing for patient and partner
Foreign body	Discharge and itching; foreign body on exam	Remove foreign body; children may need sedation; antibiotics not indicated

Disposition

Discharge home if tolerating oral liquids and hemodynamically stable. Counsel on condom use; refer for HIV and other sexually transmitted disease (STD) testing. If not tolerating oral liquids or hemodynamically unstable, admit.

23.10: Acute Pelvic Pain

Acute pelvic pain is the sudden onset lower quadrant pain, unilateral or bilateral that requires medical attention. A good interpretation of acute pelvic pain is often difficult for health personnel because of the multitude of potential causes. In low resource settings, a meticulous history and physical exam of the patient is the most important thing. Pelvic pain can be simple or put the life of the patient in danger, and may demand an emergent surgical intervention.

Diagnosis: History and Physical Examination

Characteristics of pain: time course, progression, location, radiation, intensity, medications or other treatments used at home for pain, similar pain in past?Last menstrual period and type of contraception used, if any?Vaginal bleeding, recent or current pregnancy, miscarriage, or abortion?Is there vomiting, diarrhea (frequency, presence of blood or mucous), dysuria, urinary frequency or urgency, fever, chills? What is the appearance of urine?Abdominal exam: assess for masses, tenderness and location of tenderness, discoloration or ecchymosis, prior surgical scars, guarding, rebound tenderness. Speculum exam: evaluate for vaginal lacerations or discharge (if present, note color and odor), evaluate cervix for color, friability, discharge; evaluate if os is open or closed. Bimanual exam: cervical motion tenderness, uterine and adnexal tenderness, discharge.

Possible Causes and Differential Diagnosis

Obstetrics: Early pregnancy, ectopic pregnancy, miscarriage.

Gynecologic: pelvic inflammatory disease [diagnose if discharge, cervical motion tenderness, uterine or adenexal tenderness], torsion [sudden onset severe pain, adenexal tenderness and/or mass on exam], endometriosis [recurrent, cyclical pain], ovarian cyst, fibroids [irregular, enlarged uterus], tubo-ovarian abscess [fever, pelvic inflammatory disease, positive ultrasound].

Gastrointestinal: Gastroenteritis, diverticulitis, appendicitis (right lower quadrant pain, may have associated fever, nausea, anorexia, or no associated symptoms), small bowel obstruction (abdominal pain + vomiting or nausea), inflammatory bowel disease.

Urologic: Nephrolithiasis (suspect if blood in urine, colicky pain), urinary tract infection (dysuria or frequency + positive urinalysis).

Hernia: inguinal, femoral, umbilical (diagnose by exam).

Other: Sickle cell crisis, neurogenic, mesenteric thrombus, parasites, DKA.

Investigations

Urinalysis, Urine HCG, vaginal smear, hematocrit, white blood cell count, stool evaluation, ultrasound to evaluate for ectopic pregnancy, ovarian cyst, free fluid (if present, consider

ectopic or ruptured cyst), tuboovarian abscess, fibroid, torsion, CT scan.

Treatment
Check vital signs and ABCs. Give IV fluids if hypotension. Check for signs of dehydration (skin tenting, sunken eyes, dry mucous membranes) or trauma. Ask about hemorrhage. If dehydrated or in shock, place IV, give 1-2 liters IV fluids (0.9% NaCl or lactated ringer's) and control pain with analgesia.

Disease specific
Ectopic Pregnancy, threatened miscarriage: see chapter on 1st trimester vaginal bleeding Pelvic Inflammatory Disease: doxycycline 100 mg PO BID x 7 days PLUS ciprofloxacin PO 500 mg x1 or ceftriaxone 250 mg IM x 1 PLUS Metronidazole 500 mg PO TID x 7 days; if pregnant: erythromycin 500 mg QID x 7 days, ceftriaxone 250 mg IM x 1; metronidazole if after 1st trimester. Torsion: laparatomy, Urinary tract infection: ciprofloxacin 500 mg BID x 3 days in women; 14 days if pyelonephritis, Endometriosis: oral contraceptives and NSAIDs, follow up with gynecology, Parasites: mebendazole or other locally available treatment, ovarian cyst: Pain control with tramadol, NSAIDs, or other available analgesia. If persistent pain, consult gynecology or transfer.Appendicitis, diverticulitis: see appropriate chapter.

Disposition
Depends on underlying condition. Hospitalize if peritoneal signs, unable to take oral fluids, severe pain, concern for surgical etiology.

23.11: First trimester Vaginal Bleeding

Bleeding in the first three months (first 12-14 weeks) of pregnancy. Vaginal bleeding in early pregnancy is common, but may indicate life-threatening disease. Women of child-bearing age with abdominal pain or vaginal bleeding should be presumed pregnant.

Diagnosis: History and Physical Examination
LNMP, events during current pregnancy, degree and duration of bleeding, abdominal pain, cramps, fever, dizziness, syncope.

Ectopic risk factors: tubal surgery, PID, prior abortion, IUD.

Exam: abdominal tenderness; internal cervical os open or closed, clots or products of conception (POC), **adnexal mass, adnexal or uterine tenderness** or masses. Obtain vital signs. If haemo dynamically unstable (HR>100, SBP<90), give 1-2 L crystalloids.

Possible Causes and Differential diagnosis
Ectopic pregnancy: Gestational sac outside of uterus. Peritonitis or abnormal vitals suggest rupture (life threatening). Rule out in all pregnant women with vaginal bleeding. Unlikely

if IUP on ultrasound (US). Incidence of heterotopic pregnancy (IUP + ectopic) is 1/4000.

Recent induced abortion or complications
Spontaneous abortion: May be inevitable (bleeding with open internal os), Incomplete (open os, POC visualized), complete (closed os, fetus and placental materials fully expelled).

Septic abortion: fever, abdominal pain; can complicate any abortion.

Threatened abortion: bleeding, closed internal os, US with IUP.Risk of loss of pregnancy 35-50%.Molar pregnancy: Abnormal trophoblastic tissue. Bleeding at 12-16 weeks, uterus larger than expected, passage of grape-like material, US with 'snowstorm' pattern.Non-pregnancy-related vaginal or cervical pathology: laceration, ulcer, cervical mass

Investigations:
Laboratory: urine HCG, Hb/hct, type and Rh screen, cross match if heavy bleeding/haemo dynamic instability, quantitative serum HCG if possible,

Pelvic ultrasound: transvaginal or transabdominal

Diagnostic of IUP: intrauterine double gestational sac, fetal pole or heart activity

Diagnostic of ectopic: pregnancy outside uterus, ectopic fetal pole or heart activity

Suggestive of ectopic: moderate to large free fluid or adnexal mass without IUP

Indeterminate: empty uterus, nonspecific fluid, single gestational sac/"pseudo sac". Normal pregnancy unlikely if no gestational sac on transabdominal US with HCG>6500 or transvaginal US with HCG>3000 (6-7 weeks)

Culdocentesis: If US not available. Detects hemoperitoneum, but less sensitive and specific. If positive, treat as ruptured ectopic.

Examine passed uterine contents: rinse and place in saline/tap water. Blood dissolves, chorionic villi will be fluffy and finger like. Presence of villi excludes ectopic.

Treatment
General
Intra venous fluid/blood as needed for hemodynamic instability, **anti-D immunoglobulin if Rh-negative** (50ug in first trimester, 300ug after first trimester), analgesics

Ectopic: consult obstetrics or transfer

Laparotomy / Laparoscopy: Unstable, peritoneal signs, evidence of rupture

Medical management (rarely, for unruptured): Methotrexate (50mg/m2 BSA IM x 1) only if definite ectopic by ultrasound, tubal mass <3.5cm, no fetal cardiac activity, no evidence of rupture, minimal pain, HCG<5000. Needs close follow up for serial HCG levels until zero (2-3 months)

If ultrasound is indeterminate or not available, admit for serial exams. Or, if patient is pain free, stable, HCG< above values or pregnancy <6 weeks, and resources available, can discharge for repeat HCG in 48 hours and ultrasound in 7 days.

Spontaneous abortion:
If heavy bleeding, perform haematocrit, transfuse as needed.

Gently remove fetal tissue if visualized in cervical os then MVA if <14 weeks; E and C if >14 weeks

If febrile, doxycycline 100mg, ceftriaxone 1 gm IV and Metronidazole 500 mg IV or ampicillin 2 gm IV, Gentamicin 2mg/kg IV and Metronidazole 500 mg IV

Threatened abortion: bleeding with live IUP on ultrasound, closed os

Expectant management; close follow up with OB

Molar pregnancy

Consult OB, Evacuation, ensure OB follow up to trend hCG

Disposition
Admit for ruptured ectopic, haemodynamic instability, falling haematocrit, severe pain, fever

Discharge if abortion with minimal blood loss and good OB follow up. Instructions to return for fever, worsening abdominal pain, or significant increase in bleeding

23.12: Emergency Treatment of Sexual Violence

Sexual violence can be verbal, physical or emotional and occurs without the victims consent. It includes rape, attempted rape, sexual harassment, incest, touching, and child molestation. Sexual violence occurs in all societies and can take place anywhere: in schools, homes, workplaces, churches and public places. It can be perpetrated by anyone, and it can happen to anyone, regardless of their social status. Sexual assault is an emergency; timelines to start ARV, STI, and pregnancy prophylaxis are crucial to the life of the patient. Management is multidisciplinary and should be integrated with the social and judicial system. Medical certificates or other required legal documents are essential for victims.

Diagnosis: History and physical exam
Conduct in a safe, private environment; explain everything, reassure the patient, determine date and time of the assault, number of assailants, type of assault, and ask about: other physical injuries, date of last menstrual cycle and if on contraception.

On exam: be careful not to force or pressure the patient at any point. Note vital signs, do head to toe examination, identify and describe any injuries (location, size, type, shape); note position patient was examined in. Genital examination (including anal margin): describe injuries in clockwise direction: location, old or new, evaluate other sites, including lips and breasts, and photograph injuries per local evidence laws with patient permission.

Investigations
HIV and pregnancy test, hepatitis B, screen for STI, if assailant testing available, obtain HIV, STD testing, Hepatitis B, Viral load and CD4, imaging or other laboratory studies directed to associated traumatic injuries.

Treatment
Ensure patient is haemo dynamically stable without life threatening trauma. Reassure patient about her safety.Treat traumatic injuries. Complete medical certificate or local legal documentation for all patients; if patient does not want it, place in chart in case she eventually needs it.

Evidence Collection
Collect in accordance to local laws and capabilities, change gloves at each step; remember the patient can refuse any step, place all samples in labelled evidence envelopes, do not add other substances, and collect all biological material for DNA testing if available: saliva, hair, semen, skin, blood, substances under nails, secretions; conserve patient's clothes

Disease Prophylaxis: if anal or vaginal penetration or body fluid exposure

Table 23: Disease Prophylaxis Regimens for sexual violence

	Non-pregnant adults	Pregnant women	Children
Syphilis	Penicillin benzathine 2.4 million IU IM x 1	Penicillin benzathine 2.4 million IU IM x 1	Penicillin benzathine 50,000 IU/kg IM x1
Gonorrhea	ceftriaxone 250 mg IM x1 or ciprofloxacin 500 mg PO x1	ceftriaxone 250 mg IM x1	Cefexime 8 mg/kg PO x1
Chlamydia	Doxycycline 100 mg PO BID x 7 days	Erythromycin 500 mg PO QID x 7 days	Erythromycin 12.5 mg/ kg QID x 7days
Trichomonas & BV	Metronidazole 2 g PO x 1	Metronidazole 2 g PO x1 (if after 1st trimester)	Metronidazole 5 mg/ kg PO TID x 7 days
Pregnancy: if ≤ 5 days after assault	Levonorgestrol 1.5 mg PO x1; or 0.75 PO q12 hours x2 or IUD insertion within 5 day	None	If post-menarche: adult dosing Pre-menarche: none
HIV: if ≤ 72 hours after assault	28 day course. Per national guidelines or if unknown or low risk source + low local resistance: 2NRTI + 1 PI or 1 NRTI + 1 NtRTIs +1 NNRTI; if source on ARVs or high local resistance: 2NRTIs + boosted PI (e.g. – zidovudine + lamivudine + lopinavir + ritonavir boost); Note: no Efavirenz in pregnancy		
Tetanus	Last tetanus vaccination ≤ 10 years: none Last vaccination > 10 years + open wound: booster dose of tetanus IM No vaccination + open wound: antitoxin + 1st vaccine in the other arm		
Hepatitis B	If patient not already vaccinated: 500UI immunoglobulin + vaccine		

Follow up testing: HIV at 6 weeks, 3 and 6 months; HbV at 3 months.

Psychological Treatment

Ideally by psychologist, but can also be done by doctor or nurse. Follow up psychological care is important. Potential techniques include: maintenance clinic, talkgroup, expressive techniques and art therapy, and mind-body techniques.

Legal

Encourage and support patient with reporting the assault to authorities. Justice must be impartial. Social assistance, including emotional support and housing assistance may be needed. Referral to institutions or organizations that support victims of violence is essential.

Section 24 Emergency management of burn injuries

Burn is defined as an injury to the skin or other organ and tissue primarily caused by heat or due to radiation, radioactivity, electricity, friction or contact with chemicals.The burns patient has the same priorities as all other trauma patients. **Ignore the burn wound and search for life threatening conditions.**

Do Initial Assessment
Airway, Breathing-(beware of inhalation injury and rapid airway compromise), Circulation-fluid replacement, Disability-compartment syndrome and Exposure-percentage area of burns.

First aid
-Drench the burn thoroughly with cool water to prevent further damage and remove all burned clothing

-If the burn area is limited, immerse the site in cold water for 30 minutes to reduce pain and edema and to minimize tissue damage

-If the area of burn is large,after it has been doused with cool water, apply clean wraps about the burned area [or the whole patient]to prevent systemic heat loss and hypothermia

-First 6 hours following injury are critical,transport the patient with severe burns to a hospital as soon as possible

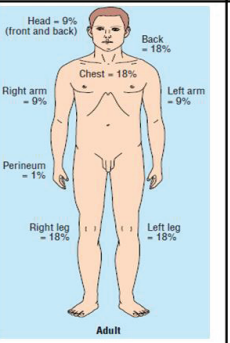
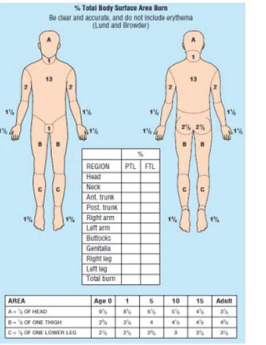
The severity of burn is determined by:
A. Burned surface area

B. Depth of burn

C. Other considerations, Part of the body burned and patient risk factor, age and comorbidities

- Morbidity and mortality rises with increasing burned surface area.it also rises with increasing age so that even small burns may be fatal in elderly people.

Table 24. Evaluation of total surface area and depth of burn

Depth			**Surface area of the burn**	
Type of burn	Character	Usual causes	Rule of nine	Lund-Bruder chart (for children)
1° burn(superficial epidermal burn)	Erythema, pain, absence of blisters	Sun-burn		
2° burn(partial thickness burn)	Red or mottled	Contact with hot liquid		
3° burn(full thickness burn)	Dark and leathery	Flame, electricity, lighting	N.Bfor small burn area, we can use the outstretched palm and fingers which approximates to 1% of the body surface area.	

- It is common to find all three types within the same burn wound
- Burns greater than 15% in an adult, greater than 10%in a child, or any burn occurring in the very young or elderly are serious.
- It is important to estimate the depth of the burn to assess its severity and to plan future wound care.

Emergency room care for burn patients

Airway:

Suspect inhalation injury in flame burns or anyone burned in a closed space. Signs of inhalational injury include hoarseness and expiratory wheezes, copious mucus production and carbonaceous sputum needs early intubation

Fluid resuscitation

Place one or two large bore cannula and administer crystalloid solution/ringer lactate/

Use Parkland formula for fluid resuscitation- 4ml*TBSA * wt. (Kg).
½ to run over the first 8 hrs and ½ to run over the next 16 hours

Place a Foley catheter in the bladder to monitor the effectiveness of fluid resuscitation
30 ml/hr. in Adult And 1 ml/kg/h in children indicate adequate resuscitation

Tetanus prophylaxis

Both active and passive immunization based on patient immunization status

Gastric decompression; NGT

Pain control: IV opiates

Psychosocial care

Determine the need for special procedures

- Do escharotomy for full thickness burns involving the extremities or the chest
- Debride any open blisters
- Fingers and toes should be wrapped individually separating the digits

Initial treatment of Burn wound
Initially burns are sterile. Focus the treatment on steady healing and prevention of infection.

Except in small burns, debride all bullae. Excise adherent necrotic (dead) tissue initially and debride all necrotic tissue over the first several days.

After debridement, gently cleans the burn with 0.25% chlorhexidine solution, 0.1%, cetrimide solution, or another mild water based antiseptic.

Do not use alcohol based solutions.
Gentle scrubbing will remove the loose necrotic tissue. Apply a thin layer of Antibiotic cream.

Moderate burns requiring hospitalization:

-Partial thickness burn involving 15-20 %TBSA in an adult
- Partial thickness burn involving 10-20 % TBSA in a child
- Full thickness burn involving 2-10 % TBSA
Major Burns which require admission to burn unit
-Partial thickness burn > 25% TBSA in Adults
--Partial thickness burn >20% TBSA in children
-Full thickness burn > 10 % TBSA
-Burns of special region; face, hands, feet, perineum
-Any burn in the very young, the elderly or the infirm
-Inhalation injury
-Chemical burns, High voltage electrical burn
-Associated trauma or significant pre burn illness e.g diabetes

Special consideration with chemical burns
1. Remove all clothing

2. Brush powdered chemicals off the wound, and then flush chemical burns for a minimum of 30 minutes using copious volumes of running water

3. Determine which chemical caused the injury

Special consideration with electrical injuries

1. Differentiate between low voltage/<1000 v/and high voltage/>1000 v/injuries

2. Attach a cardiac monitor, treat life threatening dysrhythmias as needed

3

. Assess for associated trauma, Assess central and peripheral neurological function

4. Administer ringer lactate: titrate fluids to maintain adequate urineoutput or to flush pigments through the urinary tract.

5. Using pillow elevate burned extremities above the level of the heart. Monitor distal pulses.

Annexes

Annex 1. Heimlich maneuver

Heimlich maneuver

Step 1. Determine whether the airway is blocked. Ask, "Are you choking? Can you speak? Can I help you?"

Step 2. If you determine the airway is blocked:

- Stand behind the person and wrap your arms around the person's waist. Locate the navel (bellybutton).
- If the person is obese or pregnant, wrap your arms around the chest.

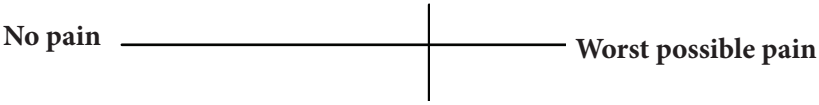
Annex 2: Pain management

Pain Scales

- **Numerical Scale: - It has 0 at one end meaning no pain and 10 at the other end meaning worst imaginable pain**

(No pain)0 1 2 3 4 5 6 7 8 9 10 (Worst possible pain)

- **Visual Analogue Scale (VAS): - A100 mm scale with no pain at one end worst imaginable pain at the other is particularly used in clinical trials, as it is more amenable to statistical analysis than numerical scale. It is a simple line on which the patient marks X to denote how strong their pain is**

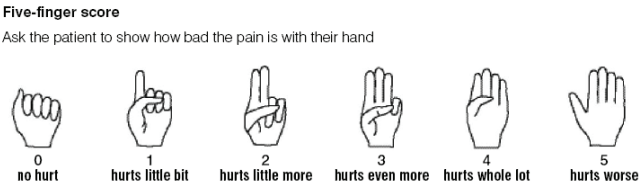


- **Palms Pain Scale or Five-finger score (0-5)**

Figure 1- Five-finger score

The hand scale

Show on your fingers how severe is pain ….5 is most severe



The hand scale ranges from a clenched hand (which represents 'No hurt') to five extended digits (which represents 'Hurts worst'), with each extended digit indicating increasing levels of pain. Note: it is important to explain this to the patient as a closed fist could be interpreted as worst possible pain in some cultures.

- **For children: The Faces pain scale**

Figure 2- The Faces pain scale for children

(The Faces pain scale has been revised so that the scale is from zero to ten).

- Use with children who can talk (usually 3 years and older)
- Explain to the child that each face is for a person who feels happy because he has no pain, or a little sad because he has a little pain, or very sad because he has a lo t of pain
- Ask the child to pick one face that best describes his or her current pain intensity
- Record the number of the pain level that the child reports to make treatment decisions, follow-up, and compare between examination

•For children: FLACC Scale

For use in children less than three years of age or older non-verbal children
Use like an Apgar score: evaluating each item and arriving at a total score out of

DATE/TIME						
Face 0 – No particular expression or smile 1 – Occasional grimace or frown, withdrawn, disinterested 2 – Frequent to constant quivering chin, clenched jaw						
Legs 0 – Normal position or relaxed 1 – Uneasy, restless, tense 2 – Kicking, or legs drawn up						
Activity 0 – Lying quietly, normal position, moves easily 1 – Squirming, shifting back and forth, tense 2 – Arched, rigid, jerking						
Cry 0 – No cry (awake or asleep) 1 – Moans or whimpers, occasional complaint 2 – Crying steadily, screams or sobs, frequent complaints						
Consolability 0 – Content, relaxed 1 – Reassured by occasional touching, hugging or being talked to, distractible 2 – Difficult to console, comfort						
TOTAL SCORE						

For Elderly-Pain Assessment in Advanced Dementia (PAINAID)

Items	0	1	2	Score
Breathing independent of vocalization	Normal	Occasional labored breathing. Short period of hyperventilation	Noisy labored breathing. Long periods of hyperventilation. Cheyne-Stokes respiration	
Negative vocalization	None	Occasional moan or groan. Low level speech with a negative or disapproving quality	Repeated troubled calling out. Loud moaning or groaning. Crying	
Facial expression	Smiling or inexpressive	Sad. Frightened. Frown	Facial grimacing (an ugly or disapproving facial expression)	
Body language	Relaxed	Tense. Distressed pacing. Fidgeting	Rigid. Fists clenched. Knees pulled up. Pulling or pushing away. Striking out	
Consolability	No need to console	Distracted or reassured by voice or touch	Unable to console, distract, or reassure	

Annex 3: Glasgow Coma Scale

Observation	Response	Score
Eyes	Open spontaneously	4
	Open to speech	3
	Open to painful stimulus	2
	No response (no eye opening)	1
Verbal	Responds sensibly	5
	Confused	4
	Inappropriate words	3
	Incomprehensible sounds	2
	No response (silent)	1
Motor	Obeys commands	6
	Points (localizes) to pain	5
	Withdraws from pain	4
	Bends limbs in response to pain (flexion)	3
	Straighten limbs in response to pain (extension)	2
	No response	1

Annex 4: AVPU Scale

AVPU scale

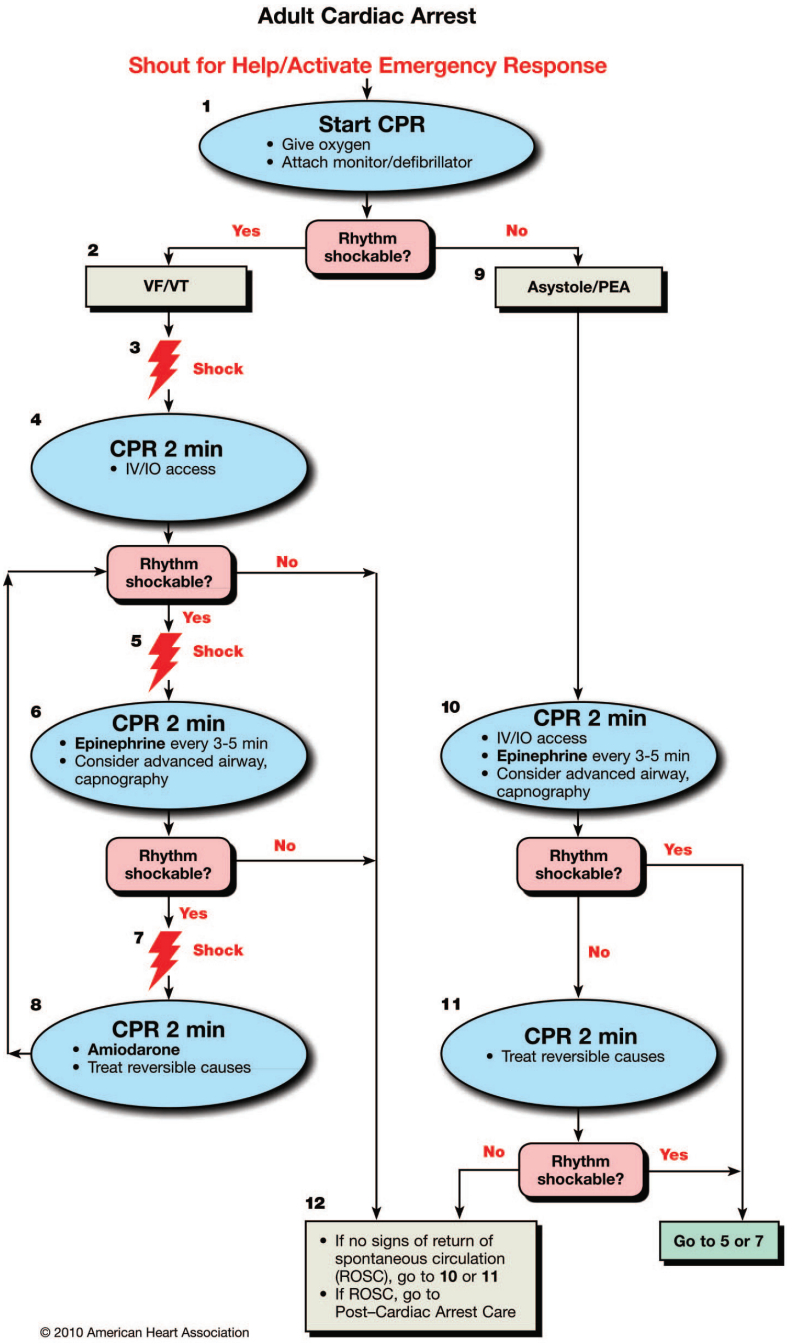
A	**Patient is A lert**
V	**Patient responds to V oice**
P	**Patient responds to P ain**
U	**Patient is U nresponsive**

Annex 5: Adult Triage scales(Adopted from Early Warning Scores(EWS))

ADULT TRIAGE SCORE								
	3	2	1	0	1	2	3	
Mobility				Walking	With Help	Stretcher/ Immobile		Mobility
RR		less than 9		9-14	15-20	21-29	more than 29	RR
HR		less than 41	41-50	51-100	101-110	111-129	more than 129	HR
SBP	less than 71	71–80	81-100	101-199		more than 199		SBP
Temp		less than 35		35-38.4		38.5 or more		Temp
AVPU				Alert	Reacts to Voice	Reacts to Pain	Unresponsive	AVPU
Trauma				No	Yes			Trauma
over 12 years / taller than 150cm								

Colour	RED	ORANGE	YELLOW	GREEN	BLUE
TEWS	7 or more	5-6	3-4	0-2	DEAD
Target time to treat	Immediate	less than 10 mins	less than 60 mins	less than 240 mins	
Mechanism of injury		High energy transfer			
Presentation		Shortness of breath - acute		ALL OTHER PATIENTS	DEAD
		Coughing blood			
		Chest pain			
		Haemorrhage - uncontrolled	Haemorrhage - controlled		
	Seizure - current	Seizure - post ictal			
		Focal neurology - acute			
		Level of consciousness reduced			
		Psychosis / Aggression			
		Threatened limb			
		Dislocation - other joint	Dislocation - finger or toe		
		Fracture - compound	Fracture - closed		
	Burn – face / inhalation	Burn over 20%	Burns - other		
		Burn - electrical			
		Burn - circumferential			
		Burn - chemical			
		Poisoning / Overdose	Abdominal pain		
	Hypoglycaemia – glucose less than 3	Diabetic - glucose over 11 & ketonuria	Diabetic - glucose over 17 (no ketonuria)		
		Vomiting - fresh blood	Vomiting - persistent		
		Pregnancy & abdominal trauma or pain	Pregnancy & trauma		
			Pregnancy & PV bleed		
Pain		Severe	Moderate	Mild	
	Senior Healthcare Professional's Discretion				

Annex 6: Adult CPR Algoristhm (adopted from 2010 American Heart Association)



CPR Quality
- Push hard (≥2 inches [5 cm]) and fast (≥100/min) and allow complete chest recoil
- Minimize interruptions i compressions
- Avoid excessive ventilati
- Rotate compressor eve 2 minutes
- If no advanced airway, 30:2 compression-ventilation ratio
- Quantitative waveform capnography
  - If P_{ETCO_2} <10 mm Hg attempt to improve CPR quality
- Intra-arterial pressure
  - If relaxation phase (diastolic) pressure <20 mm Hg, attempt to improve CPR qual

Return of Spontaneous Circulation (ROSC)
- Pulse and blood pressure
- Abrupt sustained increase in P_{ETCO_2} (typically ≥40 mm Hg)
- Spontaneous arterial pressure waves with intra-arterial monitoring

Shock Energy
- **Biphasic:** Manufacturer recommendation (eg, initial dose of 120-200 J); if unknown use maximum available Second and subsequen doses should be equiva lent, and higher doses may be considered.
- **Monophasic:** 360 J

Drug Therapy
- **Epinephrine IV/IO Dos** 1 mg every 3-5 minutes
- **Vasopressin IV/IO Dos** 40 units can replace first or second dose of epinephrine
- **Amiodarone IV/IO Dos** First dose: 300 mg bolus Second dose: 150 mg.

Advanced Airway
- Supraglottic advanced airway or endotracheal intubation
- Waveform capnography to confirm and monitor ET tube placement
- 8-10 breaths per minute with continuous chest compressions

Reversible Causes
- **H**ypovolemia
- **H**ypoxia
- **H**ydrogen ion (acidosis
- **H**ypo-/hyperkalemia
- **H**ypothermia
- **T**ension pneumothorax
- **T**amponade, cardiac
- **T**oxins
- **T**hrombosis, pulmonary
- **T**hrombosis, coronary


Emergency
EMERGENCY

Federal Ministry of Health

National Emergency Treatment Protocol

Emergency and Critical Care Service Directorate

November 2016