



A POCKET GUIDE FOR CLINICIANS DURING HAJJ

**Technical supervisory committee for
hospitals and primary care centers
during Hajj
Ministry of Health
Saudi Arabia**

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Produced by
Technical supervisory
committee for hospitals and
primary care centers during
Hajj
Ministry of Health
Saudi Arabia

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A guide for an effective management
of prevalent medical and surgical
Common Diseases Encountered
during Hajj

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Foreword

I have the pleasure to introduce the fifth edition of this Pocket-sized guide for clinicians who are responsible for the health of pilgrimages.

It lists a variety of prevalent medical and surgical Commonly Encountered conditions during Hajj in a concise and logical approach. It provides the fundamental principles of their emergency management, taking into consideration the massive patients' turnover during Hajj.

This Pocket book guide in its fifth edition was reviewed by the editor after being prepared mainly by the members of technical supervisory committee for hospitals and primary care centers during Hajj.

The contents are also presented just before the start of Hajj to the clinicians working in Arafat and Mena Hospitals and primary care centers, in a four day symposium to make sure that they are familiar with common diseases encountered during hajj and how to deal with them.

I hope that every staff revise this Hajj pocket book especially about life saving procedures, as well as

important drugs; their safe doses, emergency preparedness and disaster plan.

Take precautions to be protected from infectious diseases and what to do if exposed to any of them.

Give the priority to the cases with more risk for seriousness e.g. airway obstruction, shortness of breath and chest pain. Be competent in CPR.

Pay attention to certain problems which may be forgotten e.g. Spine injury in traumatized or comatose patient and how to move and transport these patients.

We wish to thank all of those who worked so diligently on this Pocket book.

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Preface

Of fifth edition

A Guide for Clinicians during Hajj, Presented in its fifth edition in a size of a pocket book provides a practical account of emergency medicine. Concentrates on the common emergencies and diseases encountered during hajj. In addition, aiming for the core information required to make sound and safe treatment decisions in a crowded and high-risk area of clinical practice.

Updates of heat syndromes, Middle East respiratory syndrome coronavirus (MERS-CoV) and Ebola Virus Disease was added in this edition.

We hope that it will be a valuable pocketbook for all health workers serving during Hajj.

Editor

Preface

Of first edition

It is really apparent that increasing number of pilgrimages with increasing need for medical and surgical care to serve the guests of “Allah” , furthermore, the members of the Medical supervisory committee for hospitals during Hajj emphasized that physicians and surgeons serving in hajj must obtain the knowledge and skills to optimally manage the patients coming from almost all countries, with different diseases, Although most of the illnesses may be mild and benign, still during Hajj, number of very critical and seriously ill patients may be faced, because Hajj has a very high potentials regarding emergency cases.

This pocket book is divided into sections, including the most common emergency clinical presentations: the breathless patient, acute chest pain, hypotension/falling blood pressure, disordered consciousness, metabolic emergencies, low urine output, the acute abdomen, the traumatized and the agitated

patient. This book provides limited information related to the pharmacotherapeutic management of the patient.

The information is not referenced; other sources may need to be consulted for medical emergencies not covered in this book.

We hope that “A Pocket guide for clinicians in Hajj” will be usable pocket book providing a concise and practical guide to the management of patients during Hajj season.

Its production was made possible through the effort of the previous and the recent Medical supervisory committees for hospitals during Hajj.

Editor

ETHICS IN HAJJ

Working during Hajj is a religious necessity and a big responsibility, as you will be the first person in contact with the cases which may not be easily diagnosed in its early stages.

It is essential for healthcare workers to have a humble attitude, knowledgeable and appropriately skillful in order to diagnose the seriousness and important cases that need early treatment. Hence, an important fundamental in medicine is to maintain safety and not to cause any harm to the patient.

Although most of the cases you may see will be mild and benign, still during Hajj, you may face a number of very critical and seriously ill patients (Hajj has a very high potentials regarding emergency cases). To note, that a GP in a Primary Health Care center will have different resources for diagnoses and management with that of those available in the hospital.

We are grateful that you have been selected to work during Hajj because you are qualified and expert to render patient care. So, you need to be punctual, productive, responsible, organized in your work, cooperative with your colleagues, honest and Embracing the utmost regard to patient's dignity, feelings,

tenderness and the privacy of his sentiments and body parts.

We hope that you will consider the following ethical principles:

- **Autonomy:** *what the pilgrimage's wishes are, so the pilgrimage is having the* right to accept or refuse the treatment, therefore you need to get the permission from him if he is wise, and respect his Ihram (intention of performing Hajj).
- **Beneficence:** obligation to act in the best interest of the patient unless advanced directives have been issued to relatives.
- **Nonmaleficence:** obligation to do no harm to the patient.
- **Justice:** duty to treat all patients fairly and equitably.

Always remember that you have an honored responsibility by serving the guests of "Allah" the most merciful, so as a healthcare worker in Hajj it is your duty to bring the mercy of Allah unto His guests, therefore consider your work as a worship and charity on top of being a career, and carry out duty in conscientiousness and perfection. perfection that entails that you worship Allah as if you see Him. For even though you don't see Him, He sees you. Hoping for the reward (Thawab) from "Allah" for your proper work and genuine efforts.

AIRWAY

Airway management

Maintaining adequate airway is essential to provide a pathway to the lungs and prevent aspiration In cardiopulmonary resuscitation, anaesthesia, emergency medicine and intensive care medicine. In nearly all circumstances airway management is the highest priority for clinical care. This is because if there is no airway, there can be no breathing, hence no oxygenation of blood and therefore circulation (and hence all the other vital body processes) will soon cease.

Getting oxygen to the lungs is the first step in almost all clinical treatments. The 'A' is for 'airway' in the 'ABC' of cardiopulmonary resuscitation.

How to open the airway?

Manual :- Head tilt / chin lift / jaw thrust
With equipment: - Oro/nasopharyngeal airway, Endotracheal intubation, Laryngeal mask airway (LMA), Combitube.

Manual methods:

Head tilt / chin lift:

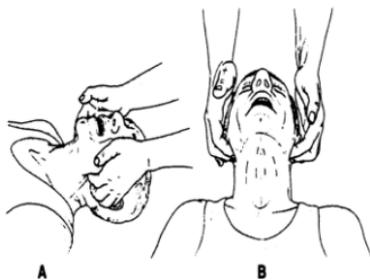
The simplest way of ensuring an open airway in an unconscious patient is to use a head tilt chin lift technique, thereby lifting the tongue from the back of the throat. This is the standard way of clearing an airway.



Jaw thrust:

The jaw thrust is used on patients with a suspected spinal injury and is used on a supine patient.

The practitioner uses their thumbs to physically push the posterior aspects of the mandible upwards to pull the tongue forward and prevents it from occluding the air passage.



Adjuncts to airway management: Oropharyngeal Airways:



S-shaped devices most helpful in the spontaneously breathing patient who is unconscious with no gag reflex and at risk of occluding the airway via tongue and pharyngeal relaxation.

These devices help to do suction of the mouth and throat and prevent the patient from biting and occluding a tracheal tube and keep the airway open during bag-mask ventilation when rescuers tend to push down on the chin.

Nasopharyngeal Airways:

Uncuffed tubes used most frequently for the intoxicated or semi-conscious patient who cannot tolerate an oropharyngeal airway because of severe gag reflex, trismus or massive trauma around the mouth.



The definitive airway:

- ◆ Endotracheal tube.
- ◆ Nasotracheal tube.
- ◆ Surgical airway (cricothyrotomy or tracheostomy).

Indications for definitive airway:

- ◆ Maintenance of patent airway
- ◆ Airway protection from aspiration
- ◆ Pulmonary toilet
- ◆ Control of ventilation
- ◆ Application of positive pressure
- ◆ Maintenance of adequate oxygenation.

Orotracheal intubation:

The most commonly accepted method for securing an airway. When performed by a skilled clinician, it has been shown to have a high rate of success with low rate of complication.



Before an airway is inserted, the patient is placed in the optimal position for alignment of the three anatomic axes, the oral, pharyngeal, and laryngeal;

The “sniffing” position . and preoxygenated via facemask.

First narcotics and then an induction agent is given. Prior to giving neuromuscular blocking agent, it is usually necessary to establish the ability to ventilate. After muscle relaxant takes effect, the patient is mask ventilated before being intubated, After visualization of the vocal cords, the endotracheal tube is inserted. Correct placement of the endotracheal tube may be confirmed by:

- Direct visualization of the endotracheal tube cuff passing the vocal cords
- Presence of ETCO₂ on three consecutive breaths
- Absence of stomach “gurgling” sound made by air entering the stomach. It is important to auscultate over the stomach before the lungs because the stomach may rapidly fill with gases in case of esophageal intubation
- Equal bilateral breath sounds over the lungs
- Fogging of the endotracheal tube
- Refilling of the ventilator bag with expiration
- Rarely, a chest x-ray may be used to confirm placement of tube

Nasotracheal intubation :

Nasotracheal intubation provides another definitive route of securing an airway. In cases such as oral surgery, this route of intubation is preferred. The choice of nostril does not appear to be a factor in the rate of peri-operative complications. The

nostril that the patient breathes more easily through is usually chosen for intubation. After preparation of the nasal mucosa with vasoconstricting nose drops and dilation of the nostril with progressively larger nasal trumpets, the tube is inserted into the nose until visualized in the oropharynx. With the aid of a laryngoscope and Magill forceps, the tube is then advanced into the trachea. Alternatively, the nasal tube may be inserted over a fiberoptic scope. Complications that can occur with this route of intubation include bleeding, infection, laryngospasm, and damage of the turbinates.

Rapid sequence induction:

Rapid Sequence Induction (RSI) is a commonly used technique of intubation in emergent cases and in surgical patients at risk for aspiration. RSI consists of pretreatment, preoxygenation, administering of a short acting induction agent, and the administering of a neuromuscular blocker. Pretreatment also involves the administering of drugs to decrease the cardiovascular response to intubation. Common induction agents are thiopental, propofol, and etomidate. Paralysis is commonly achieved with either succinylcholine or rocuronium.

Surgical airways:

A surgical airway is indicated when other means of establishing an airway fail, or in cases of laryngeal

trauma, facial injuries, or long term need of ventilatory support.

Cricothyroidotomy: is the preferred method of a surgical airway. It involves the opening of the cricothyroid membrane for placement of a tracheal tube. Complications to this technique include bleeding infection, vocal cord damage, and tracheal stenosis. Cases in which a cricothyroidotomy is contraindicated include age <12 years, laryngotracheal disruption, or coagulopathy. When a cricothyroidotomy is contraindicated, a tracheostomy is the preferred approach.

Tracheostomy : Surgical opening of the trachea and insertion of a tracheostomy tube should be performed under controlled conditions in the operating room by a skilled person. Tracheostomies should be performed after the airway has first been secured by a tracheal tube, a translaryngeal catheter, or cricothyrotomy. Tracheostomies are not an appropriate procedure for urgent situations such as airway obstruction or cardiac arrest.

Alternative airway techniques:

Laryngeal mask airway:

When compared to orotracheal intubation, the LMA is considered easier and faster to place correctly. The lubricated LMA is inserted into the hypopharynx until the tip meets the upper esophageal sphincter. The cuff is then inflated. This low-pressure cuff increases the risk of aspiration if vomiting occurs during

ventilation. When using the LMA in this manner the use of muscle relaxants do not necessarily improve the success rate of intubation, but decrease the incidence of coughing and movement. Therefore, muscle relaxants are generally given before LMA placement. Contraindications to the LMA include the need for peak pressure greater than 20cms H₂O, patients at risk for aspiration, and patients with low lung compliance necessitating the need for high-pressure ventilation.

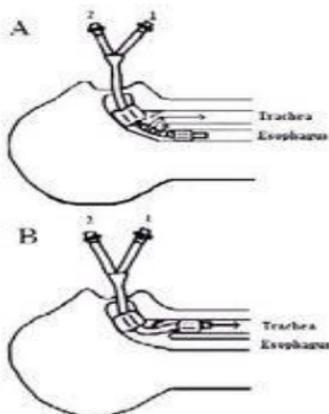
In cases of a difficult airway, the intubating LMA- can be used as a conduit for placement of an endotracheal tube.

Retrograde intubation:

This technique of intubation involves the placement of a guide wire through the cricothyroid membrane and into the pharynx in a retrograde fashion. The guide wire is then used to aid placement of an endotracheal tube.

Combitube:

The combitube is a double lumen tube with one tube serving as an esophageal airway, and the other as a tracheal airway.



Its blind placement into the hypopharynx makes it an important device in emergency airway management. After placement, the longer esophageal tube is ventilated. If no CO₂ is detected with ventilation, the tube is correctly placed in the esophagus.

The ventilator is then attached to the other tube for ventilation into the trachea. Three percent of the blind combitube intubations lead to tracheal placement of the esophageal tube. When this is the case, tracheal tube is ventilated. Placement of the combitube while the patient's neck is in the neutral position allows an advantage for use in the trauma patient. The major contraindication to use of the combitube is esophageal pathology.

Upper Respiratory Tract Infection

More than 200 published papers in the last three decades showed that respiratory symptoms were the most frequent symptoms among pilgrimages and health workers. The most common respiratory tract infection viruses are influenza and rhinoviruses. In hospitalized patients, Pneumonia is a significant cause of admission accounting for 20-50% of such admissions.

Acute rhinitis:

Acute inflammation of the nasal mucosa is called acute rhinitis, which may be viral or bacterial.

Clinical features: These may include, a burning sensation at the back of nose followed by nasal stuffiness, rhinorrhea & sneezing. There may be low grade fever, chills. Initially the nasal discharge is watery and profuse but later may become muco-purulent due to secondary bacterial invasion.

Treatment: Bed rest, Plenty of fluids, Analgesics(preferably non-aspirin) , Oral antihistamines and local nasal decongestants, Antibiotics--required only when secondary bacterial infection supervenes.

Frequent hand washing is very important to prevent the spread of infection.

Acute pharyngitis/tonsillitis:

An inflammation of the pharynx, Which may be due to viral, bacterial, fungal or non-specific causes.

Clinical features: there may be discomfort in the throat, malaise, low-to-high grade fever, dysphagia, headache. Pharynx shows erythema, exudate & Enlargement of tonsils. There may be oedema of soft palate and uvula with the Enlargement of cervical nodes.

Treatment: Bed rest, plenty of fluids, warm saline gargles, Analgesics, Antibiotics- used in case of bacterial pharyngitis for 7-10 days.

Acute laryngitis:

Acute laryngitis may be viral or bacterial or non-infectious, which may be due to vocal abuse, allergy, thermal or chemical burns to larynx due to inhalation.

Clinical features: hoarseness of voice, discomfort or pain in the throat especially after talking, dry & irritating cough, dryness of throat, malaise, fever.

Indirect laryngoscopy will reveal congestion & oedema of epiglottis, aryepiglottic folds, arytenoids, false & true vocal cords.

Treatment: Voice rest, Avoidance of smoking, Steam inhalation, Cough suppressants, Antibiotics--when there is secondary bacterial infection, Commonly used antibiotics are amoxicillin, augmentin, and Erythromycin, Analgesics—to relieve local pain & discomfort and Steroids in laryngitis following thermal or chemical burns.

Acute epiglottitis:

An acute inflammatory condition confined to supraglottic structures i.e. Epiglottis, aryepiglottic folds & arytenoids. Marked oedema of these structures may obstruct the airway. It is a serious condition & children of 2-7 years are usually affected although adults can also get it. Caused by *H.influenzae*.

Clinical features: onset of symptoms is abrupt with rapid progression, there may be sore throat & dysphagia (common in adults) while dyspnoea & stridor (common in children). High grade fever may be present.

Treatment: Hospitalization, Antibiotics: preferably amoxicillin, Steroids, Adequate hydration, Humidification & oxygen, Intubation or tracheostomy—may be required for respiratory obstruction.

Acute laryngo-tracheo-bronchitis:

An inflammatory Condition of the larynx, trachea and bronchi. Usually it starts as viral Infection and secondary bacterial infection soon supervenes, Usually affects the children.

Clinical features: hoarseness of voice, croupy cough, high grade Fever, inspiratory stridor, suprasternal and intercostal recession.

Treatment: hospitalization, antibiotics, humidification, parenteral fluids, steroids, adrenaline (via a respirator), intubation or tracheostomy.

PREVENTIVE MEASURES:

The use of masks may reduce exposure to droplet nuclei, the main mode of transmission of most respiratory tract infections. The practice of social distancing, hand hygiene, and contact avoidance was associated with reduced risk of acquiring respiratory tract pathogens.

Evidence showed that antibiotics do not work for either the common cold or for acute purulent rhinitis and many people are affected by antibiotic side effects.

BREATHING

Sever Air Flow Obstruction

Upper air Way Obstruction:

Major airway obstruction (Above the level of thoracic inlet), that might be caused by:

- Loss of consciousness
- Air way edema (angioedema)
- Trauma to upper Airway
- Foreign Body.
- Infection.
- Tumor.

Management:

- Check responsiveness
- Activate emergency response system
- A = Airway: open the airway
- B = Breathing: check breathing, provide positive-pressure ventilations
- C = Circulation: check circulation, give chest compressions
- D = Defibrillation: assess for and shock VF / pulseless VT.

Small Airway Obstruction:

Air way obstruction at the level of bronchiole.

Types:

- Reversible small air way obstruction (Asthma):
- Irreversible Small Airway Obstruction (COPD):

Acute Asthma Management:

Asthma might present with severe breathlessness, tachypnea, tachycardia, wheeze and even silent chest, cyanosis or collapse.

Patients with SpO₂<92% or other features of life threatening asthma require ABG measurement.

- Give supplementary oxygen to maintain an SpO₂ level of 94-98%.
- Use high dose inhaled β_2 agonists (oxygen-driven) as early as possible. Reserve intravenous β_2 agonists for those patients in whom inhaled therapy cannot be used reliably.
- Give steroids in adequate doses - Continue prednisolone 40-50 mg daily for at least five days or until recovery.
- Add nebulised ipratropium bromide (0.5 mg 4-6 hourly) to β_2 agonist treatment for patients with acute severe or life threatening asthma or those with a poor initial response to β_2 agonist therapy
- Following consultation with senior medical staff consider giving a single dose of IV magnesium sulphate (1.2-2 g IV infusion over 20 minutes) for

patients with acute severe asthma who have not had a good initial response to inhaled bronchodilator therapy or life threatening or near fatal asthma.

- Patients whose peak flow is greater than 75% predicted one hour after initial treatment may be discharged from ED.
- Admit patients with any feature of a severe attack persisting after initial treatment or patients with any feature of a life threatening or near fatal attack.
- Refer to ICU any patient requiring ventilatory support with acute severe or life threatening asthma, failing to respond to therapy, evidenced by:
 - Deteriorating PEF
 - Persisting or worsening hypoxia
 - Hypercapnea
 - ABG analysis showing low pH or high H+
 - Exhaustion, feeble respiration
 - Drowsiness, confusion, altered conscious state
 - Respiratory arrest.

Acute exacerbations of COPD:

The diagnosis of sudden worsening of COPD symptoms should be considered in anyone who has dyspnea, chronic cough or sputum production, and/or a history of exposure to risk factors for the disease such as regular tobacco smoking.

The diagnosis of COPD is confirmed by spirometry, when FEV₁% predicted is < 88% for men, or < 89% for women, On chest x-ray, the classic signs of COPD is

lung (hyperinflation), a flattened diaphragm, increased retrosternal airspace, and bullae. It can be useful to help exclude other lung diseases, such as pneumonia, pulmonary edema or a pneumothorax, ABG may show hypoxaemia and/or Hypercapnea, respiratory acidosis, CBC may show reactive polycythemia.

Acute exacerbation of COPD is treated with supplemental oxygen, Bronchodilators, β_2 agonists, Anticholinergics and Corticosteroids.

The only measures that have been shown to reduce mortality is smoking cessation and supplemental oxygen.

Acute respiratory failure

There are two types of respiratory failure:

Type I: the most common form of respiratory failure is Hypoxemic respiratory failure which is characterized by a PaO_2 of less than 60 mm Hg with a normal or low PaCO_2 . and it can be associated with all acute diseases of the lung, which generally involve fluid filling or collapse of alveolar units. Some examples of type I respiratory failure are cardiogenic or noncardiogenic pulmonary edema, pneumonia, and pulmonary hemorrhage.

type II : Hypercapnic respiratory failure is characterized by a PaCO_2 of more than 50 mm Hg. And commonly associated with Hypoxemia, The pH depends on the level of bicarbonate, which is dependent on the duration of hypercapnia. Common etiologies include drug overdose, neuromuscular disease, chest wall abnormalities, and severe airway disorders (eg, asthma, chronic obstructive pulmonary disease [COPD]).

Manifestations:

- Altered mental status
- Increased work of breathing
 - Tachypnea
 - Accessory muscle use, retractions, paradoxical breathing pattern
- Catecholamine release
 - Tachycardia, diaphoresis, hypertension
- Abnormal arterial blood gas values

Acute Respiratory Failure Management:

- Oxygen supplementation
- Pharmacologic Adjuncts
 - Bronchodilators.
 - β_2 -agonists.
 - Anticholinergics (ipratropium).
 - Corticosteroids.
 - Theophylline .
 - Antibiotics.

Oxygen supplementation:

Nasal Cannula



- 100% oxygen delivered
- Low flow : <math><0.5-5.0</math> L/min
- Low oxygen : $FIO_2 <0.4-0.5</math>$

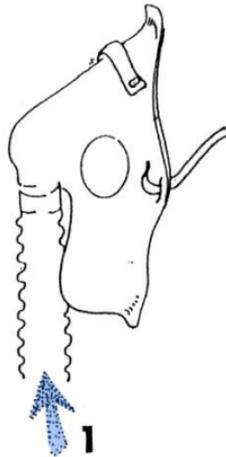
**Air-Entrainment
Face Mask:**

- 100% O₂ + entrainment device
- High flow.
- Variable oxygen : FIO₂ 0.24–0.5

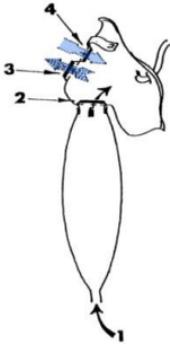


Aerosol Face Mask:

- 100% O₂ + large-bore tubing
- Nebulizer/O₂ blender
- Flow matching: If mist disappears in inspiration, air is entrained
- Moderate-flow, variable FIO₂ device



Reservoir Face Mask:

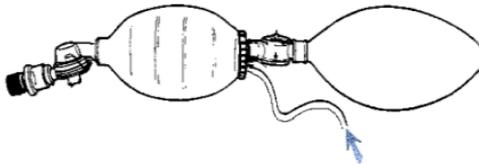


- Reservoir bag filled with 100% O₂
- High oxygen
- High flow

Resuscitation Bag-Mask-Valve Device:

- 100% O₂.
- High flow (> 15 L/min).
- Emergency equipment.

Little to no air entrainment with firm fit.



NIPPV:

- Non invasive ventilatory assistance used for patients with respiratory distress who had moderate to severe dyspnea, as a result of hypoxemic or hypercapnic failure, it is most

effective with alert, oriented and cooperative patient.

- The ventilator is volume or pressure-cycled with unilevel or bilevel pressure support connected to the patient using nasal or face mask with controlled FIO₂
- It is relatively contraindicated in:
 - Decreased level of consciousness.
 - Poor airway protective reflexes
 - Copious secretions
 - Cardiovascular instability
 - Progressive pulmonary decompensation
 - Upper gastrointestinal hemorrhage
- **Initiation of NPPV**
 - Set FIO₂ at 1.00
 - In Hypoxemic failure:
 - Inspiratory pressure (IPAP) 10 cm H₂O
 - Expiratory pressure (EPAP) 5 cm H₂O
 - Titrate EPAP in 2 cm H₂O increments
 - In Ventilatory failure:
 - IPAP 10 and EPAP 2 cm H₂O
 - Titrate IPAP in 2 cm H₂O increments
 - Make changes every 15-30 minutes
 - Monitor vital signs, appearance, pulse oximetry and blood gases
 - Head of bed at 45° angle
 - Consider gastric decompression
 - Intubation if patient deteriorates.

CIRCULATION

Diagnosis and management of Shock

Shock:

- Shock is always a symptom of primary cause
- Shock is an Inadequate blood flow to meet tissue oxygen demand.

Clinical manifestations:

- Signs of Hypoperfusion / inadequate oxygenation :
 - Hypotension
 - Altered mental status
 - Oliguria
 - Metabolic acidosis
 - ↑Lactate
 - Abnormal creatinine, transaminases.
- Compensatory mechanisms:
 - Vasoconstriction
 - Tachycardia
- Signs of Specific etiology.

Shock Categories:

- **Cardiogenic:**
 - Myopathic
 - Arrhythmic
 - Mechanical
- **Hypovolemic:**
 - Hemorrhagic
 - Nonhemorrhagic
- **Distributive:**
 - Septic
 - Neurogenic
 - Adrenal crisis
 - Anaphylactic
- **Obstructive:**
 - Massive pulmonary embolism
 - Cardiac tamponade
 - Tension pneumothorax
 - Constrictive pericarditis

Cardiogenic Shock :

- Decreased contractility.
- Increased filling pressures, decreased LV stroke work, decreased cardiac output.
- Increased systemic vascular resistance – compensatory.

Hypovolemic Shock :

- Decreased cardiac output.
- Decreased filling pressures.
- Compensatory increase in systemic vascular resistance.

Distributive Shock:

- Normal or increased cardiac output.
- Low systemic vascular resistance.
- Low to normal filling pressures.
- The causes are: Sepsis, anaphylaxis, neurogenic, and acute adrenal insufficiency.

Obstructive Shock:

- Decreased cardiac output.
- Increased systemic vascular resistance.
- Variable filling pressures dependent on etiology.
- The causes are Cardiac tamponade, tension pneumothorax, massive pulmonary embolus.

Therapeutic Goals in Shock:

- Increase O₂ delivery.
- Optimize O₂ content of blood.
- Improve cardiac output and blood pressure.
- Match systemic O₂ needs with O₂ delivery.
- Reverse/prevent organ hypoperfusion.

Cardiogenic Shock Management :

- Treat arrhythmias.
- Diastolic dysfunction may require increased filling pressures.
- Vasodilators if not hypotensive.
- Inotrope administration.

- Vasopressor agent needed if hypotension present to raise aortic diastolic pressure .
- Preload and afterload reduction to improve hypoxemia if blood pressure adequate.

Hypovolemic Shock Management :

- Airway and breathing support.
 - Monitor for deterioration of oxygenation.
- Volume resuscitation – Fluid Therapy:
 - Crystalloids Initial choice):
 - Lactated Ringer’s solution.
 - Normal saline (high chloride may produce hyperchloremic acidosis)
 - Colloids
 - Hetastarch
 - Albumin
 - Gelatins
 - Packed red blood cells.
- If traumatic stop the bleeding:
 - Direct pressure
 - Reduce pelvic volume
 - Operation
 - Splint fractures
 - Angio-embolization .
- Monitoring:
 - Correct hypotension first (monitor BP).
 - Decrease heart rate (monitor with continuous ECG)

- Infuse to physiologic endpoints (monitor CVP).
- Correct hypoperfusion abnormalities (eg. Sensorium and urine output)
- Match fluid given to fluid lost (intake output chart)

Distributive Shock Therapy:

- Restore intravascular volume , the patient might remain hypotensive despite volume therapy.
- Vasopressors for MAP < 60 mm Hg.
- Adjunctive interventions dependent on etiology:
 - Sepsis and Anaphylaxis: see following chapters.
 - Neurogenic: fluids, vasopressors and managing the causative factor.
 - Acute adrenal insufficiency: intravenous corticosteroids and dextrose-containing normal saline must be instituted before the diagnosis is confirmed.

Obstructive Shock Treatment:

- Maintain airway and oxygenation.
- Treated according to the etiology (Relieve obstruction):
 - Pericardiocentesis for cardiac tamponade.
 - Tube thoracostomy for tension pneumothorax.
 - Treat pulmonary embolus or pericarditis.

- Temporary benefit from fluid or inotrope administration

Inotropic / Vasopressor Agents:

- Dopamine
 - Low dose (2-3 $\mu\text{g}/\text{kg}/\text{min}$) – mild inotrope plus renal effect
 - Intermediate dose (4-10 $\mu\text{g}/\text{kg}/\text{min}$) – inotropic effect
 - High dose (>10 $\mu\text{g}/\text{kg}/\text{min}$) – vasoconstriction
 - Chronotropic effect
- Dobutamine
 - 5-20 $\mu\text{g}/\text{kg}/\text{min}$
 - Inotropic and variable chronotropic effects
 - Decrease in systemic vascular resistance
- Norepinephrine
 - 0.05 $\mu\text{g}/\text{kg}/\text{min}$ and titrate to effect.
 - Inotropic and vasopressor effects.
 - Potent vasopressor at high doses.
- Epinephrine
 - Both α and β actions for inotropic and vasopressor effects. Increases myocardial O_2 consumption.

Diagnosis and Management of Septic Shock

Definitions:

SIRS: systemic inflammatory response syndrome. Defined as two or more of the following:

- Temperature $> 38^{\circ}\text{C}$ or $< 36^{\circ}\text{C}$
- Pulse rate > 90 beat/min
- Respiratory rate > 20 /min or $\text{Pco}_2 < 32$ mmHg
- WBC > 12000 or < 4000 or $> 10\%$ immature band.

Infection: invasion of naturally sterile tissue by microorganisms.

Sepsis: SIRS due to infection.

Severe sepsis: is sepsis associated with organ dysfunction, hypoperfusion or hypotension and may include but not limited to:

- Lactic acidosis.
- Oligouria.
- Altered mental status.

Septic shock: is severe sepsis induced refractory hypotension ($\text{SBP} < 90$ mmHg or reduced by > 40 mmHg from base line despite adequate fluid resuscitation) and Concomitant organ dysfunction .

Management Steps:

- A. Diagnosis of severe sepsis or septic shock systemic inflammatory response syndrome criteria in addition to:
 - i. Systolic BP < 90mmHg after 20-30 ml/kg crystalloid challenge.
 - ii. Blood lactate 2-4mmol/L..
- B. Insertion of Central line: preferably subclavian or internal jugular line.
 - i. The aim to achieve CVP 8-12 mmHg
 - ii. Resuscitation by 500-1000ml over 30 minutes boluses of crystalloid or Colloid which can be repeated.
 - iii. Resuscitation to clinical end points of arterial MBP (65-70), HR, Urine output (1ml/kg), skin perfusion and mental status, and indices of tissue perfusion as Lactate concentration.
 - iv. Vasopressor agent as necessary to keep mean arterial pressure > 65-70mmHg.
- C. Insertion of Arterial line: preferably radial.
 - i. Accurate measurement.
 - ii. Beat to beat analysis so decision regarding therapy shall be immediate.
- D. Get mixed venous oxygen (SCVO₂) from subclavian catheter:
 - i. Aim to keep SCVO₂ >70%.
 - ii. Optimize CVP to 8-12mmHg.
 - iii. Transfuse packed red blood cells to achieve hematocrit >30%.
 - iv. Dobutamine infusion up to maximum of 20ug /kg/min especially if Cardiac Index <2.5 l/min/m².

- v. If could not achieved, Intubate and mechanically ventilate the patient to keep SCVO₂ >70%.
- E. Achieve cardiac index > 4.5L/min/m²:
- i. Start Vasopressors as soon as possible to maintain mean arterial pressure 65-70mmHg both during and following adequate fluid resuscitation.
 - ii. Norepinephrine the vasopressor of choice. 0.05 ug/kg/min titrated to effect.
 - iii. Dopamine is equal effect but limited due to tachycardia.
 - iv. Vasopressin may be used in patients with refractory shock despite adequate resuscitation and high dose of vasopressors. Dose 0.01-0.04 units/min
- F. Low dose corticosteroid is recommended:
- i. 100mg Hydrocortisone 3 times a day for 7 days if patient is improving or to continue if not improving or showed reduced adrenal function.
 - ii. In the absence of vasopressor requirement steroid should not be used.
 - iii. High dose of steroid is not recommended.
 - iv. Adrenal function test is optional to decide regarding:
 - 1. Weaning of steroid at the end of treatment period.
 - 2. Discontinuing steroids earlier.
 - 3. Addition of oral fludrocortisones.
- G. Precise bacteriological diagnosis before starting antibiotics.
- i. Two to three blood cultures preferably from peripheral veins, different sites.

- ii. Appropriate samples are indicated in particular ventilator associated pneumonia or catheter related infection and soft tissue or in abdominal infections.
- iii. Culture obtained through drains is discouraged.
- H. Early drainage of the source of infection and collection.
- I. Precise Broad Spectrum Antibiotics: according to site of infection and suspected micro-organisms .
- J. Antifungal is not recommended as empirical treatment except when justified as in:
 - i. Gastrointestinal perforation
 - ii. Acute necrotizing pancreatitis
 - iii. Immunocompromized
 - iv. Vascular access devices.
- K. Strict control of blood Glucose level to (80-110 mg/100 ml) using continuous Insulin infusion protocol.

Anaphylactic shock

Anaphylaxis is a life-threatening emergency.

Clinical features:

Early:

- Sensations of warmth, itching especially in axillae and groins, Feelings of anxiety or panic.

Progressive:

- Erythematous or urticarial rash, Oedema of face, neck, soft tissues, Abdominal pain and vomiting, Dyspnoea.

Severe:

(May appear extremely rapid without prodromal features).

- Hypotension (shock), Bronchospasm (wheezing), Laryngeal oedema (stridor, aphonia, drooling), Arrhythmias, cardiac arrest, Hypoxaemia, cyanosis.

Initial management:

- If working alone, call for assistance.
- Stop any suspected medication or diagnostic contrast material, remove allergen from patient's mouth, scrape out bee stings.
- Maintain airway and start oxygen.
- If there is severe respiratory and circulatory collapse or coma, ventilate the patient (Drug-assisted intubation for impending airway obstruction is a very

high-risk procedure and should only be attempted by an expert).

- Establish an intravenous line and rapidly infuse normal saline or Hartmann's solution (20 mL/kg). Continue as necessary.
- Adrenaline:
 - Adrenaline is safe, effective and life-saving and must be used immediately intramuscularly in the lateral thigh.
 - Withholding adrenaline due to misplaced concerns of possible adverse effects can result in deterioration and death of the patient.
 - Adrenaline IM dose – adults
 - 0.5 mg IM (= 500 micrograms = 0.5 mL of 1:1000) adrenaline
 - Adrenaline IM dose – children
 - The recommended doses are based on what is considered to be safe and practical to draw up and inject in an emergency
 - (The equivalent volume of 1:1000 adrenaline is shown in brackets)
 - > 12 years: 500 micrograms IM (0.5 mL) i.e. same as adult dose
 - 300 micrograms (0.3 mL) if child is small or prepubertal
 - > 6 – 12 years: 300 micrograms IM (0.3 mL)
 - > 6 months – 6 years: 150 micrograms IM (0.15 mL)
 - < 6 months: 150 micrograms IM (0.15 mL)

- If necessary, repeat intramuscular dose every 5 minutes. Large doses of adrenaline may be needed, up to a maximum of 5 mL (5 mg).
- If the patient remains shocked after two intramuscular doses, consider an adrenaline infusion to restore blood pressure, If critical care facilities are not immediately available, give the following adrenaline infusion:
 - Mix 1 mg adrenaline (1 ampoule) in 1000 mL of normal saline
 - Start infusion at 5 mL/kg/hour (approx. 0.1 microgram/kg/minute).
 - Titrate rate up or down according to response.
- Some cases are resistant to adrenaline, especially if the patient is taking beta blocking drugs. If adequate doses of adrenaline are not improving the situation, give glucagon 1–2 mg intravenously over 5 minutes.
- Bronchodilator:
 - For bronchospasm, give salbutamol or terbutaline by nebuliser, or aerosol with spacer device.
 - In severe cases use continuously.
- Give hydrocortisone Adults: 20-80 mg PO daily for 2-5 d; Children: 0.5-1 mg/kg PO daily for 2-5 days or (Corticosteroids may modify the overall duration of a reaction and may prevent relapse. However, onset of action will be delayed. Never use these to the exclusion of adrenaline).
- The standard treatment of anaphylaxis should also include antihistamines and corticosteroids. However,

antihistamines have a much slower onset of action than epinephrine, they exert minimal effect on blood pressure, and they should not be administered alone as treatment. Antihistamine therapy thus is considered adjunctive to epinephrine. Administer an H1 blocker and an H2 blocker, because studies have shown the combination to be superior to an H1 blocker alone in relieving the histamine-mediated symptoms. Diphenhydramine and ranitidine are an appropriate combination. Diphenhydramine (Benadryl) - Adults: 25 mg PO q6h for 2-5 d; Children: 1 mg/kg PO q6h for 2-5 d. Second-generation, less-sedating agents may be preferable because of decreased adverse effects. In their adult doses, these include fexofenadine (Allegra) at 180 mg/d, loratadine (Claritin) at 10 mg/d, cetirizine (Zyrtec) at 10 mg/d, desloratadine (Clarinex) at 5 mg/d.

- Observe vital signs frequently and monitor electrocardiogram and pulse oximetry.
- Keep patient in hospital for observation for at least 4–6 hours after the complete resolution of abnormal symptoms and signs, as biphasic reactions may occur.
- Keep patient in hospital longer if there is a history of asthma or previous allergy, or if the patient needed repeated doses of adrenaline.
- All patients must be followed up to investigate possible provoking factors and for further management.

Management of Arrhythmias

Objectives:

1. Correctly identify the arrhythmia.
2. Determine any factors causing the arrhythmia.
3. Appropriately treat the arrhythmia.

Don't Forget Your ABCDs (Adult BLS Algorithm):

- Call for help.
- Check airway and breathing (2 breaths).
- If no pulse, start CPR (30 compressions: 2 breaths) until defibrillator arrives.
- Shock, if appropriate, and resume CPR immediately.
- Check rhythm and pulse every 5 min.

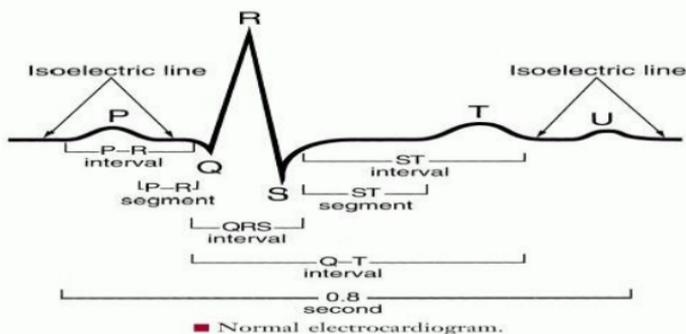
Reversible Causes:

6 H's:

Hypovolemia
Hypoxia
H+ (acidosis)
Hypo-/hyperkalemia
Hypoglycemia
Hypothermia

5T's:

Toxins.
Tamponade (Cardiac).
Tension pneumothorax.
Thrombosis:
Coronary.
Pulmonary.
Trauma:
Hypovolemia.
↑ Intracranial pressure.



When you are faced with an arrhythmic patient, first decide if patient is stable or not based on symptoms and vital signs, then do ECG if unstable then all kind of arrhythmias need to be cardioverted (D/C shock), only the amount of energy is different 50 J for SVT 200J for VT. all synchronized except FV. If the patient is stable then do 12 leads ECG and decide if atrial or ventricular or AV node.

By observing QRS if normal in duration then it is atrial, but if it is wide more than 0.11 seconds then it is ventricular, then decide if regular or irregular then calculate heart rate then, follow the guidelines for each one according to ACLS guidelines:

First degree heart block:



Long P-R interval – Asymptomatic.

Treatment:

- Search for the underlying causes.

Second degree heart block:



2:1 block - Usually symptomatic

Treatment:

- Treat underlying causes
- Atropine
- Pacemakers

SINUS bradycardia:



Underlying causes:

- electrolyte imbalance
- drugs
- hypothyroidism

Treatment:

- B2 stimulants
- Atropine if symptomatic
- Pacemakers ??

Digitalis effect :



Treatment:

- Stop the drug
- Correct potassium
- Digibind

Idioventricular rhythm:



Underlying causes:

- Serious underlying heart disease
- Revascularization

Treatment:

- Treat the cause
- Correct electrolytes
- ?? Pacemakers

Hyperkalemia:



Treatment:

- Calcium gluconate
- Dextrose/insulin
- Dialysis

Premature atrial contractions:



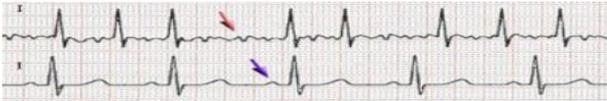
Underlying causes:

- All heart diseases
- Electrolyte imbalance
- Hyperthyroidism
- Excess tea and coffee

Treatment:

- No specific treatment needed
- Correct underlying causes

Atrial fibrillation:



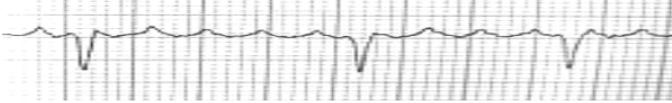
Underlying causes:

- Any disease involving atria
- Lone AF.
- Thyroid disease

Treatment:

- Control rhythm & rate
- Cardioversion

Atrial flutter:



Underlying causes:

- Atrial diseases
- Hypoxia

Treatment:

- Cardioversion
- Ablation

Premature ventricular contractions:



Underlying heart disease:

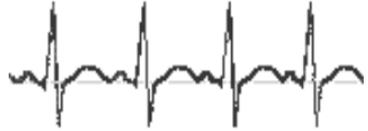
- Hypertension
- Ischemia
- Cardiomyopathy
- failure
- Electrolyte imbalance & hypoxia & drugs
- Reperfusion after thrombolysis

Treatment:

- Correct underlying causes.
- Amiodarone.
- Xylocaine.
- B.blockers.

Supraventricular tachycardia:

- Narrow complex.
- Wide complex.



Underlying causes:

- Any disease affect the heart can cause SVT
- IHD & HTN.
- Electrolyte imbalance.
- Anaemia.
- Hypoxia & stress & thyrotoxicosis.

Treatment::

- Carotid massage.
- Correct underlying causes.
- Adenosine .
- Verapamil & diltiazem
- B.blockers.
- Digoxin.

Ventricular tachycardia:



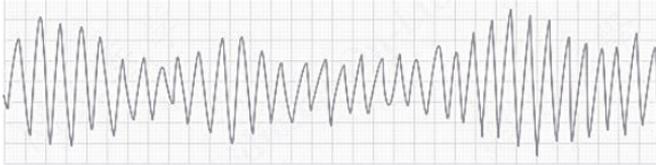
Underlying causes:

- Serious heart disease.
- Severe electrolyte imbalance

Treatment:

- If pulseless V.T: CPR
- If with pulse:
 - Amiodarone
 - Xylocaine
 - Correct causes

Torsade de points:



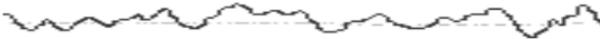
Underlying causes:

- Serious heart disease.
- Electrolyte imbalance.
- Antiarrhythmic drugs.

Treatment:

- Correct the cause
- Magnesium sulphate
- Pacing

Ventricular fibrillation:



Treatment: CPR

Asystole:



Treatment: CPR

Acute Coronary Syndromes

Whenever you are facing a patient with suspected chest pain you should think of the following steps:

- Identify patients with acute coronary syndromes (ACS).
- Diagnosis and acute management of Unstable Angina(UA), Non ST elevation MI(NSTEMI), and ST elevation MI(STEMI).
- Identify reperfusion strategies for STEMI and high-risk NSTEMI/UA patients.

To identify whether the patient is at risk of ACS, think of risk factors:

- Male gender, age, diabetes, hypertension.
- Hyperlipidemia, smoking, family history of MI, obesity, sedentary lifestyle, cocaine/amphetamine use.

First line to diagnosis is the symptoms:

- Character of pain—chest pressure.
- Duration of pain—>1 hour.
- Occurrence in early morning.
- Chest pressure characteristics:
Retrosternal radiating to neck and left shoulder, associated with shortness of breath, diaphoresis, nausea, vomiting, light-headedness, exacerbated by exertion and relieved by rest.

What information is needed to determine the type of ACS?

- Symptoms
- Risk factors
- ECG (initial within 10 minutes and subsequent)
- Cardiac markers

Immediate Emergency Department Assessment (takes less than 10 min):

Check what type of ACS does ECG suggested:

- UA(unstable Angina) or NSTEMI (non ST elevation MI)
- *Immediate General management:*
 - Oxygen,Pulseoxymeter saturation more than 90%, ECG monitor
 - Antiplatelet therapy—aspirin 160-325 mg, preferably chewed (not enteric coated)
 - Antianginal therapy—NTG sublingual, morphine IV,
- Start Adjuvant therapy:-
 - NTG IV,
 - β -blocker
 - (*patients who are not candidates to receive a β -blocker:P <60/min, uncompensated moderate-severe heart failure, shock, AV block >1st degree, systolic BP <100 mm Hg, peripheral hypoperfusion, active bronchospasm*)
 - *Clonidogrel to be given*

- Administered for most high-risk patients if noninterventional approach used.
- Administered if PCI planned.
- Administered for patients who cannot take aspirin.
- *Heparin would be used in:*
 - LMWH preferred (especially enoxaparin) unless surgery planned within 24 hours
- *Calcium channel blocker would be administered to a patient with UA/NSTEMI IN:*
 - Inability to tolerate or receive a β -blocker.
 - Pain not controlled with NTG and β -blocker.
 - Calcium channel blockers do not reduce the risk of MI.
- *Candidates for GP IIb/IIIa inhibitors?*
 - High-risk patients: ST depression, ongoing chest pain, elevated cardiac markers, troponin > 0.1 .
 - Patients with planned PCI
- *Reperfusion considered urgently in UA or NSTEMI in:*
 - *High-risk indicators: such as recurrent angina, elevated troponin, new ST depression or recurrent/ persistent ST deviation, signs of heart failure, PCI within 6 months, prior CABG, Ventricular tachycardia, Hemodynamic instability.*
 - *Transfer If PCI is not available.*
- If Not high risk: Continue ASA, Heparin and other therapies

- Thrombolytics have no efficacy in UA or NSTEMI.

Check what type of ACS does ECG suggest:

- STEMI (ST elevation MI) (*Criteria of >1-mm ST elevation in ≥ 2 contiguous leads*), or new LBBB :
 - Same as General Therapy.
 - Admit to CCU (higher level of care than UA).
 - Same medications but clopidogrel is usually always administered.
 - GP IIb/IIIa inhibitors given if PCI planned.
 - Do not delay reperfusion if less than 12 hours from onset of symptoms, transfer to another institution if PCI is not available.
 - Goals:
 - Door-to-balloon inflation (PCI) goal of 90 min
 - Door-to- needle (fibrinolysis) goal of 30 min
 - PCI preferred in:
 - Contraindication to thrombolytics.
 - Presence of cardiogenic shock.
 - Diagnosis of MI made in cath lab.
 - Higher mortality risk.
 - High risk of thrombolysis .
 - Experienced personnel available with balloon inflation time of ≤ 90 min
 - If PCI is not available and transfer is not possible then consider Thrombolytics if:
 - Presentation within 3 hours of onset of pain.
 - Presentation within 6 hours if PCI is not available.
 - No contraindications to thrombolysis.

- Therapy After Reperfusion:
 - PCI and thrombolysis.
 - Heparin (except with streptokinase).
 - β -Blocker.
 - Nitroglycerin.
 - ACE inhibitor.
 - PCI.
 - Clopidogrel.
 - Glycoprotein Ib/IIIa inhibitor.

Check what type of ACS does ECG suggest:

- *Normal or non diagnostic changes in ST segment or T- wave, intermediate/ low risk UA:*
 - Consider admission to ED chest pain unit or to monitored bed and follow:
 - Serial cardiac markers including troponin
 - Repeat ECG/ continuous ST segment monitorin
 - Consider stress test
 - If develop high or intermediate risk or troponin positive, then treat as UA/ Non- STEMI.
 - If no progression to ischemia or infarction then can be discharged with follow up.

Contraindication to fibrinolysis (any one of the following):

- SBP grater than 180 mmHg.
- DBP greater than 110 mmHg.
- Right vs left arm systolic BP difference grater than 15 mmHg

- History of Structural central nervous system diseases.
- Significant closed head/facial injuries within last 3 months.
- Recent (within 6 weeks) major trauma, surgery (including laser eye surgery), GI/ GU bleeding.
- Bleeding or clotting problems or on blood thinner.
- CPR greater than 10 min. (controversial).
- Pregnancy.
- Serious systemic disease (advanced/ terminal cancer, severe liver or kidney diseases).

Heart failure (HF) & Pulmonary edema

When the Heart is unable to maintain an output adequate to meet the metabolic demands of the body is considered to be in failure

Subdivisions of Heart failure:

- Systolic – characterized by dilated left ventricle with impaired Contractility.
- Diastolic- occurs in normal or intact left ventricle with impaired ability to relax & receive blood return.

Causes of HF:

- Coronary artery diseases (IHD) (the commonest).
- HTN.
- Valvular heart diseases (VHD).
- Congenital heart diseases (CHD).
- Cardiomyopathies.
- Infective endocarditis.
- Myocarditis & others .
- High output failure is associated with high output status such as:
 - Sever anemia
 - Thyrotoxicosis
 - Big AV shunts

HF is often precipitated by cardiac ischemia, dysrhythmias, infection, pulmonary embolism , noncompliance with medications the most sever

manifestation of HF is pulmonary edema, which develops secondary to leakage of fluid from pulmonary capillaries into interstitium & alveoli of the lung.

The functional classification of CHF:

Class I No limitation during ordinary activity

Class II Slight limitation by shortness of breath and/or fatigue during moderate exertion or stress

Class III Symptoms with minimal exertion that interfere with normal daily activity

Class IV Inability to carry out any physical activity.

Clinical manifestations:

The symptoms of HF are:

- Dyspnea upon exertion, then at rest, orthopnea & paroxysmal nocturnal dysnea (PND).
- Cough with pink sputum is highly suggestive of HF.
- Lower limb swelling.
- Non specific symptoms like fatigue ,light headness

Physical examination reveals:

- Tachypnea
- High JVP.
- Wheezing or bilateral basal crepitation.
- Cardiac auscultation may reveals S3 or murmurs.
- Lower extremities edema.

Differential diagnosis:

- Bronchial asthma & COPD.
- Acute respiratory distress syndrome.
- Pneumonia.
- Pulmonary embolism.

Investigation:

- Blood tests may reveal high transaminases & bilirubin.
- CXR to look for cardiomegaly, pleural effusion & perihilar infiltrates.
- ECG to diagnose concomitant ischemia, prior MI, cardiac arrhythmias & chronic HTN.
- Echocardiogram to identify regional wall abnormalities, left ventricular function & VHD.

Management:-

- (ABC). administer supplemental O₂ with face mask, use cardiac monitoring & obtain intravenous access.
- Put patient in setting position.
- Lasix (furosemide) iv 40—80mg to be repeated according to response.
- Metolazone : used as adjunctive therapy in patient initially refractory to lasix.
- IV nitroglycerin 2.5—10ugm/min are particularly useful in patients with acute pulmonary edema.
- Morphine sulfate 2—5mg is an excellent adjunct in acute therapy.
- ACEI or ARBS & B-blockers are considered after stabilizing acute setting.

Hypertensive Crises

Hypertensive Crises Is a severe elevations in BP, often higher than 220/140 mm Hg, complicated by clinical evidence of progressive organ dysfunction. In these conditions, the BP should be lowered aggressively, by 25% of MAP (not necessarily to normal ranges), over minutes to hours to prevent or limit further organ damage.

MAP (mean arterial pressure) = Diastolic pressure + $\frac{1}{3}$ pulse pressure.

Causes:

- Chronic essential hypertension.
- Renal parenchymal disease - Chronic pyelonephritis, primary glomerulonephritis, tubulointerstitial nephritis (accounts for 80% of all secondary causes)
- Systemic disorders with renal involvement - Systemic lupus erythematosus, systemic sclerosis, vasculitides
- Renovascular disease - Atherosclerotic disease, fibromuscular dysplasia, polyarteritis nodosa .
- Endocrine - Pheochromocytoma, Cushing syndrome, ..etc.
- Coarctation of the aorta.
- Preeclampsia/eclampsia .

Clinical Characteristics :

- Duration and severity of the hypertension.
- All current medications including nonprescribed drugs .
- Co- morbid conditions.
- Prior cardiovascular or renal disease.
- Level of compliance with current antihypertensive medications .
- Blood Pressure: Usually $>220/140$ Frequent or continuous monitoring of BP should be established.
- Funduscopic Findings: Hemorrhages, exudates, papilledema , visual loss.
- Neurologic Status: Headache, confusion, somnolence, stupor, coma, dysarthria , seizures, cerebrovascular accident, focal neurologic deficits, Encephalopathy.
- Cardiac Findings: Shortness of breath, chest pain, nocturia, prominent apical pulsation, cardiac ischemia , cardiac enlargement, congestive heart failure, pulmonary edema.
- Renal Symptoms: Azotemia, proteinuria, oliguria, renal insufficiency.
- Gastrointestinal Symptoms: Nausea, vomiting.

Workup:

Laboratory Studies:

- Electrolytes, BUN, and creatinine levels to evaluate for renal impairment
- CBC and smear to exclude microangiopathic anemia

- Urinalysis
- Optional studies:
 - Toxicology screen
 - Endocrine testing
 - Pregnancy test

Imaging Studies:

- Chest radiography is indicated in patients with chest pain or shortness of breath.
 - Cardiac enlargement
 - Pulmonary edema
 - Widened mediastinum
- Head CT and/or brain MRI are indicated in patients with abnormal neurologic examinations
- Chest CT scan, transesophageal echocardiography, or aortic angiography is indicated in cases where aortic dissection is suspected.

ECG .

Acute management of hypertensive crises:

The fundamental principle in determining the necessary ED care of the hypertensive patient is the presence or absence of end-organ dysfunction (EOD).

- If the patient is not in distress:
 - Place the patient who is not in distress in a quiet room and reevaluate the blood pressure measurements twice.
 - Consider the context of the elevated BP (eg, severe pain often causes an increase in BP).
- Screen for end-organ dysfunction.
- Patients without evidence of EOD may be discharged with follow-up.

- Acute lowering of BP in the narrow window of the ED visit does not improve long-term morbidity and mortality rates.
- Patients with EOD usually require admission and rapid lowering of BP using intravenous medications. Suggested medication depends on the affected organ system.
- Even in cases of hypertensive emergencies, the BP should not be lowered to normal levels.
- Excessive blood pressure reduction has been associated with acute deterioration of renal function, ischemic cardiac and cerebral events, and acute blindness.
- A decrease of 20% to 25% in mean arterial pressure from pretreatment blood pressure in the first hour of treatment has been recommended. . If the patient remains stable, the BP should then be lowered to 160/100-110 mm Hg in the next 2-6 hours. the exceptions to this general rule are listed below.
- These BP goals are best achieved by a continuous infusion of a short-acting, titratable, parenteral antihypertensive agent along with constant, intensive patient monitoring.

Rapid BP reduction is indicated in:

Cardiovascular emergencies:

- **Aortic dissection:**
 - Maintain SBP <110 mm Hg, unless signs of end-organ hypoperfusion are present. Preferred

treatment includes a combination of narcotic analgesics (morphine sulfate), beta-blockers (labetalol, esmolol), and vasodilators (nicardipine, nitroprusside). Calcium channel blockers (verapamil, diltiazem) are an alternative to beta-blockers.

- Avoid beta-blockers if there is aortic valvular regurgitation or suspected cardiac tamponade.
- **Acute coronary syndrome**
 - Treat if SBP >160 mm Hg and/or DBP >100 mm Hg. Reduce BP by 20-30% of baseline by Beta-blockers and nitroglycerin.
 - Thrombolytics are contraindicated if BP is >185/100 mm Hg.
- **Acute heart failure**
 - Treatment with vasodilators (ACE inhibitors in addition to diuretics) for SBP \geq 140 mm Hg. IV or sublingual nitroglycerin is the preferred agent.

Neurological emergencies:

- **Acute intracerebral hemorrhage:**
 - Treatment based on clinical/radiographic evidence of increased intracranial pressure (ICP). If signs of increased ICP, maintain MAP just below 130 mm Hg (or SBP <180 mm Hg) for first 24 hours after onset. Patients without increased ICP, maintain MAP <110 mm Hg (or SBP <160 mm Hg) for first 24 hours after symptom onset by Labetalol, nicardipine and esmolol.
 - *Avoid:* Nitroprusside, hydralazine.

- Recent evidence shows that early intensive BP control is well tolerated and can reduce hematoma growth in patients treated within 6 hours after the onset of an ICH. The target systolic BP for these studies was 140 mm Hg and utilized routine intravenous medications. The target SBP was maintained over 7 days.
- **Subarachnoid hemorrhage:**
 - *Avoid:* Nitroprusside, hydralazine.
 - Maintain SBP <160 mm Hg until the aneurysm is treated or cerebral vasospasm occurs. Oral nimodipine is used to prevent delayed ischemic neurological deficits, but it is not indicated for treating acute hypertension. *The Preferred medications:* Nicardipine, labetalol and esmolol.
- **Hypertensive encephalopathy:**
 - Reduce mean arterial pressure (MAP) 25% over 8 hours, by Labetalol, nicardipine and esmolol.
 - *Avoid:* Nitroprusside, hydralazine.
- **Acute ischemic stroke:**
 - Withhold antihypertensive medications unless the systolic blood pressure (SBP) is >220 mm Hg or the diastolic blood pressure (DBP) is >120 mm Hg unless patient is receiving IV or IA fibrinolysis, then the goal BP is SBP <185 mm Hg and DBP <110 mm Hg. After treatment with fibrinolysis, the SBP should be maintained <180 mm Hg and DBP <105 mm Hg for 24 hours. *The Preferred medications are* Labetalol and nicardipine.

Gastrointestinal bleeding

Upper GI bleeding: Any bleeding originating proximal to the ligament of Treitz.

Lower GI bleeding: Any bleeding originating distal to the ligament of treitz.

Presentation:

Hematemesis: vomiting blood (bright red or coffee ground-like).

Melena: black tarry stool, (if 150 to 200 mL of blood in the GI tract), Black stool that is not tarlike (may result from 60 mL of blood from the upper GI tract).

Hematochezia : bloody stool (LGIB, or brisk UGIB with rapid transit time through the bowel).

Causes:

Lower GI bleeding:

Brisk Upper GI bleeding.
Diverticulosis.
Angiodysplasia.
Cancer/polyps.
Rectal disease.
Inflammatory bowel disease.

Upper GI bleeding:

Peptic ulcer disease.
Varices.
Gastric erosions.
Mallory-Weiss tear.
Esophagitis.
Duodenitis.

Management:

Hemodynamically unstable Patients: should undergo the following measures:

- Maintain air way and supplemental oxygen
- Two large-bore peripheral intravenous lines should be placed (minimum 18-gauge).
- Normal saline or lactated ringer should be initiated as a 2L bolus in adults until the patient's vital signs have stabilized or the patient has received 40 mL/kg of fluid.
- place on cardiac and oxygen saturation monitors
- Transfusion:
 - O-positive packed red blood cells (O-negative packed red blood cells in women of childbearing age whose Rh status is unknown) if immediate transfusion is needed.
 - Type O, type-specific, or cross matched blood depending on availability for Patients who remain unstable after 40 mL/kg of crystalloid.
 - Packed red blood cells as soon as they are available for Patients with GI bleeding and clinical or electrocardiogram evidence of myocardial ischemia.

Once stabilized:

History: Specific questions should address:

- Duration and quantity of bleeding.
- Associated symptoms, dizziness, weakness, or syncope.

- Abdominal pain.
- Previous history of bleeding and Surgery.
- Current medications, NSAID and long-term aspirin ingestion.
- Allergies.

Physical examination:

- Vital signs.
- All hypotensive or tachycardic patients should be assumed to have significant hemorrhage.
- Telangiectasia, bruises, or petechiae to assess for vascular diseases or hypocoagulable states
- Pulmonary, cardiac and abdominal findings.
- Rectal and stool examination are often key to making or confirming the diagnosis of GI bleeding.
- Always look for signs of liver disease.

Clinical Laboratory Tests:

- **Blood type** or type and **cross match** should be requested early in the patient's care.
- **Occult Blood** in stool.
- **CBC**, changes in the hematocrit may lag significantly behind actual blood loss. Rapid infusion of crystalloid may cause a decrease in hematocrit by hemodilution.
- **Platelet** counts are used to determine the need for platelet transfusions (i.e., if $<50,000/\text{mm}$).
- **PT**: to determine whether a patient has a preexisting coagulopathy. an elevated PT may

indicate vitamin K deficiency, liver dysfunction, warfarin therapy, or consumptive coagulopathy.

- **Electrolytes, blood urea nitrogen, and creatinine** may be useful in some patient with GI bleeding. Patients with repeated vomiting may develop hypokalemia, hyponatremia, and metabolic alkalosis.
- **Blood urea nitrogen** is elevated in many patients with UGIB as a result of the absorption of blood from the GI tract and hypovolemia causing prerenal azotemia. After 24 hours, hypovolemia is probably the sole determinant of azotemia unless there has been recurrent bleeding.
- **ECG** should be obtained on all patients older than age 50 or symptomatic patient to rule out myocardial ischemia.
- **Abdominal X-ray**

Treatment:

Nasogastric Tube:

NGT is rarely yields information for either diagnosis or risk stratifications so placement of a NG tube is generally not necessary.

Anoscopy / Proctosigmoidoscopy

anoscopy / proctosigmoidoscopy should be performed in patients with mild rectal bleeding who do not have obviously bleeding hemorrhoids.

Endoscopy

Endoscopy is the most accurate diagnostic & therapeutic tool for UGIB.

Colonoscopy is an effective tool for diagnosis and selected treatment of LGIB.

Gastric Acid Secretion Inhibition:

Proton-pump inhibitor (e.g., omeprazole). For all patients with documented peptic ulcer disease.

Octreotide (Somatostatin Analogues):

An intravenous infusion of octreotide at 50 µg/hr for a minimum of 24 hours for Patients with documented esophageal varices.

Sengstaken-Blakemore Tube:

The Sengstaken-Blakemore tube stops hemorrhage in approximately 80% of patients bleeding from esophageal varices. The Linton tube is superior to the Sengstaken-Blakemore tube in patients with bleeding gastric varices.

Surgery:

Surgery is indicated:

- when blood replacement exceeds 5 U within the first 4 to 6 hours or when 2 U of blood is needed every 4 hours after replacing initial losses to maintain normal cardiac output
- For all hemodynamically unstable patients with active bleeding who do not respond to appropriate intravascular volume replacement, correction of any coagulopathy, and endoscopic intervention (if available).

Immediate treatment of burn

The priorities in managing a patient with burn are:

- To assess and manage the patient's ABCDEs
- To stop the burning process (e.g., remove all clothing, jewelry, injurious material, etc.).
- Determine the percentage area of burn (Rule of 9's)
- Good IV access and early fluid replacement.

Manage Airway and Breathing:

- Consider direct thermal or inhalation injury if there are:
 - Carbonaceous sputum.
 - Inflamed oropharynx and hoarseness
 - Face and neck burns
 - Co Hb >10%
 - Hair singeing
 - Carbon deposits
- Establish and maintain patent airway *early* and consider early ET intubation.
- Oxygenate and ventilate.
- Obtain ABGs and CO levels.

Maintain Organ Perfusion:

- Adequate venous access
- Monitor vital signs
- Hourly urine output
 - Adult: 0.5 – 1.0 mL / kg / hour
 - Child: 1.0 mL / kg / hour

- Infant: 2.0 mL / kg / hour

Estimation of burn size :

The adult body is divided into anatomic regions that represent 9%, or multiples of 9%, of the total body surface. The palmar surface (including the fingers) of a patient's hand represents approximately 1% of the patient's body surface; this guideline helps estimate the extent of burns with irregular outlines or distribution.

Depth of burn:

- **First -degree Burn(Superficial):** Superficial burn characterized with erythema and pain.
- **Second-degree Burn(Partial thickness):** Superficial to deep thickness burns characterized with blistering, erythema and severe pain.
- **Third-degree Burn (Full thickness):** Deep burn with a dry, leathery appearance and lack of pain due to damage of nerve endings.

Rate and type of fluids administered:

- 4 mL warmed balanced crystalloid solution / kg / %BSA in first 24 hours (global only)
- Administer $\frac{1}{2}$ in first 8 hours
- Administer $\frac{1}{2}$ in next 16 hours
- Base calculations on time from injury
- Monitor heart rate and urinary output

Other Information:

- AMPLE history (history of the events at the scene of the fire (eg, confined space, and presence of toxic fumes or noxious gases).
- Tetanus status.

Other Management:

- Baseline blood analyses and chest x-ray
- Narcotic analgesia.
- Foley's catheter.
- Antibiotics.
- Wound care Flow sheet documentation.

Management of Chemical Burns:

- Prevent contaminated irrigation solution from running onto unaffected skin.
- Remove contaminated clothes.
- Special situations:
 - If contamination with metallic lithium, sodium, potassium, or magnesium has occurred, irrigation with water can result in a chemical reaction that causes burns to worsen. In these situations, the area should be covered with mineral oil and the metallic pieces should be removed with forceps and placed in mineral oil. If forceps are not available, soak the area with mineral oil and cover it with gauze soaked in mineral oil.
 - If contamination with white phosphorus has occurred, thoroughly irrigate the area with water then cover the area with water-soaked gauze. Keep the area moist at all times. The area can also be covered with petroleum jelly.
 - If eye exposures have not been irrigated, then this should be started immediately. Immediate removal of caustic substances in the eye is critical.

Management of Electrical Burns: (Fascia and muscle damage; may spare overlying skin)

- ABCDE approach.
- Begin fluid resuscitation and titrate to urine output of 0.5-1 mL/kg/h
- Consider furosemide or mannitol for further diuresis of myoglobin.
- Urine alkalinization increases the rate of myoglobin clearance and can be achieved using sodium bicarbonate titrated to a serum pH of 7.5.
- Initiate cardiac monitoring for all patients with anything more than trivial low-voltage exposures.
- Tetanus immunization as indicated
- Wound care
- Measurement of compartment pressures as indicated.

Transfer Criteria for Second and Third-Degree Burns:

- 10% BSA (all ages)
- Third-degree burns > 5% BSA (all ages) with:
Preexisting illnesses, associated injuries
- Unique areas (any size burn) in Face, Eyes, Ears, Hands, Feet, Genitalia, Perineum or Major joints.
- Electrical and chemical burns
- Inhalation injury.

Transfer Procedures:

- Coordinate with burn center physician
- Transfer with: Documentation and Laboratory results.

DISABILITY (NEUROLOGICAL)

Coma & decreased level of consciousness

Coma is the most severe state of impaired consciousness.

- It is a state of unresponsiveness in which the subjects lie with eyes closed they show no understandable response to external stimulus or inner need”.
- Despite the clear descriptions of coma, quantification is difficult. So Glasgow coma scale is almost universally used for this purpose. This measure must be charted from time to time while the patient is under obstruction.

A GCS of 8 or less meet the definition of coma.

Response	Score	Response	Score
Eye Opening		Motor Response	
Spontaneously	4	Obeys verbal commands	6
To verbal command	3	Localizes to painful stimuli	5
To pain	2	Flexion withdrawal	4
None	1	Abnormal flexion	3
Verbal Stimuli		Extension	2
Oriented, converses	5	No response	1
Disoriented, converses	4		
Inappropriate words	3		
Incomprehensible	2		
No response	1		

Glasgow Coma Scale (GCS)

Causes of coma:

- Brainstem lesions- infarction, hemorrhage, encephalitis, abscess, meningitis, bacterial toxemia, tumor, trauma, neurosurgical intervention.
- Cerebral hemisphere lesion with edema and secondary compression of brainstem- infarction, trauma, hemorrhage, hydrocephalus, hypertensive encephalopathy, status epilepticus, cerebral malaria.
- Metabolic abnormalities- diabetes mellitus (hyperglycemia), hypoglycemia, hepatic failure, renal failure, respiratory failure, cardiac failure,

hyponatremia, hypokalemia, hypoxia, hypothyroidism.

- Drugs and physical agents- anesthetic agents, drug overdose and alcohol ingestion, hypothermia and hyperthermia.

Initial Management:

- Airway protection and C-Spine immobilization (for suspected trauma).

In the absence of concerning difficult airway attributes rapid sequence intubation (RSI) is the recommended method, with appropriate modification for elevated intracranial pressure if this is suspected.

- Maintain breathing and Oxygen Delivery.
- Intravenous Access.
- Early use of blood glucose testing or empiric administration of dextrose (25 to 50 g) intravenously is mandatory, even in the presence of focal neurologic findings, to prevent the sequelae from prolonged neuroglycopenia.
- Vital signs including pulse oximetry.
The Blood Pressure is a sensitive indicator of Brain Lesions:
 - Systolic Blood Pressure <90: Brain Lesion is unlikely
 - Systolic Blood Pressure >170: Brain Lesion is likely.

History:

- Abrupt onset, with or without antecedent headache, nausea, or vomiting, suggests CNS hemorrhage.
- Declining mental status over hours to days suggests other disorders (e.g., hyperosmolar nonketotic coma, hyponatremia, infection).
- The patient's state before the onset of coma may provide clues to the underlying cause.
- Coma preceded by delirium suggests hyponatremia, or encephalitis.
- Other crucial elements in the history include patient's medication, antecedent trauma, fever, headache, and any known prior similar episodes.
- Knowing the Past Medical History might help in reaching the diagnosis.

Examination:

- Assessing pupillary reactivity to light .
- Complete rapid neurologic screening (motor and sensory).
- Assess Level of Consciousness (GCS).

Initial investigations:

- Glucose should be obtained in all cases of coma.
- CBC.
- Blood Chemistry: Serum electrolytes & Renal Function to assess patients for acid-base and electrolyte disturbances (i,e hyponatremia, hypernatremia, and uremia....).
- Liver Function Tests.
- Serum Osmolarity.

- Serum Calcium The serum calcium level should be measured in patients with possible metastases.
- Serum Magnesium.
- Urine Toxins Screen.
- Directed drug levels: Digoxin , Theophylline and Phenobarbital level.
- Urinalysis: urine glucose with or without ketones implies hyperglycemia and suggests diabetic ketoacidosis or hyperosmolar coma. The presence of WBCs, nitrite, or bacteria suggests urosepsis, a common cause of altered mental status in the elderly.
- ABG to classify acid-base disturbances
- Rapid Plasmin Reagin (RPR).

Diagnostic studies to consider:

- Electrocardiogram (EKG) and cardiac monitor
- *head CT scan or MRI* should be ordered when an intracranial cause of coma is suspected. Head CT scanning is not necessary if a metabolic cause of coma is identified on initial evaluation.
- Lumbar Puncture .
- C-Spine films (if trauma suspected).
- Chest XRay .
- Peritoneal tap.
- Carboxyhemoglobin level.
- HIV Test .
- Heavy metal screen.

- Vitamin B12 Level.
- Serum Folate Level.

Management of Coma:

- Ensure proper respiration:
 - Oxygen inhalation.
 - Respiratory stimulants like doxapram if needed.
 - ETT and Mechanical ventilation might be indicated in some patients with coma
 - Protect from aspiration by NGT or If not intubated the patient must be nursed in the semi-prone or lateral position.
- Ensure proper circulation:
 - Parenteral fluids intravenous glucose or blood transfusion
 - Vasopressor drug like dopamine of low blood pressure or shock.
- Glucose may have value in suspected cases of hypoglycemia. Thiamine should be added to glucose in empiric treatment of coma patients
- Removal or control of cause- e.g. gastric lavage and diuretics in narcotic poisoning.
- Electrolytes imbalance should be corrected as per degree and type of imbalance.
- *Thyroxine* may be given in comatose patients with characteristic findings consistent with myxedematous skin changes, mild hypothermia, bradycardia, and pseudomyotonic stretch reflexes (delayed relaxation phase).

- Steroids usually are given in advance because the potential for a combined adrenal and thyroid insufficiency exists.
- Control of secondary infection with antibiotics especially in presence of fever.
- Care of skin- frequent change of position in bed, alcohol or spirit rub and powdering of skin and care of mouth.
- Care of bowels and bladder- indwelling catheter, saline or soap water enema.
- Neurosurgical intervention- if coma progression raises the possibility of herniation.

Head Injuries

Evaluation of a patient with head injury should follow the sequence described in the initial assessment:

- Airway patency with cervical spine protection
- Breathing maintained with oxygenation.
- Circulation with hemorrhage control
- Then acute Neurologic Examination:
 - The Glasgow Coma Scale (GCS) is the most widespread scoring method to define altered level of consciousness.
 - Pupillary Examination.
 - Lateralizing signs.
 - Basilar Skull Fractures Signs:
 - Blood in ear canal, Otorrhea.
 - Rhinorrhea.
 - Battle's sign (retroauricular hematoma).
 - Raccoon sign (periorbital ecchymosis).

Minor Head Trauma (GCS of 13 to 15):

- Headache is the most common complaint , Other complaints include Nausea , emesis , Transient disorientation, confusion, or amnesia
- Most patients with low-risk minor head trauma can be discharged from the emergency department with head injury after a normal examination and observation of 4 to 6 hours.

- Few cases deteriorate and require CT scan. And admission.

Moderate Head Trauma (GCS of 9 to 12):

- These patients speak after their head injury but deteriorate to a status of a severe head injury within 48 hours.
- CT scan is essential to avoid delayed diagnosis of traumatic mass lesions or diffuse injury.
- All patients with moderate head injury should be admitted for observation, even with an apparently normal CT scan.

Severe head injuries (GCS of 8 or less):

Clinical pathway & Treatment:

1. Airway and breathing:

- After maintaining patency of the air way and before intubation GCS to be performed if possible.
- Rapid sequence intubation (RSI) to secure the airway in combative or agitated patients.
- Assess breathing, oxygenate and manage thoracic life threatening injuries.

2. Hypotension:

- Cannot be tolerated in the head-injured patient; fluids (crystalloid) or blood transfusion should therefore be delivered to maintain a systolic blood pressure of at least 90 mm Hg.

3. CT scan is essential to avoid delayed diagnosis of traumatic mass lesions or diffuse injury.

4. Severely head-injured patients require admission to an institution capable of intensive neurosurgical care and acute neurosurgical intervention.
5. Hyperventilation: is recommended for brief periods during the acute resuscitation and only in patients demonstrating neurologic deterioration.
6. Osmotic Agents: Mannitol : With deepening coma and deterioration of the neurologic examination, Mannitol (0.25 to 1 g/kg) can effectively reduce cerebral edema. The osmotic effects of mannitol occur within minutes and peak about 60 minutes, may last for 6 hours.
7. Seizure Prophylaxis (Indications):
 - Depressed skull fracture
 - Paralyzed and intubated patient
 - Seizure at the time of injury
 - Seizure at emergency department presentation
 - Penetrating brain injury
 - Severe head injury (Glasgow Coma Scale score ≤ 8)
 - Acute subdural hematoma
 - Acute epidural hematoma
 - Acute intracranial hemorrhage
 - Prior history of seizures
8. Antibiotic Prophylaxis is indicated in penetrating head injury, open skull fractures, and complicated scalp lacerations.

ENVIRONMENTAL EXPOSURE

Heat syndromes

Minor Heat Disorders

Heat Cramps:

Due to salt depletion (in unacclimatized patient).

Presentation:

- Cramps: involving large muscles groups (legs).
- Moist cool skin, a normal body temperature, and minimal distress.

Treatment:

- Rest in cool environment.
- Salt replacement; oral salt solution, as in rehydration solutions, can be made by adding one fourth to one-half teaspoon of table salt (or 2 1-g salt tablets) to 1 L of water. To improve taste, add a few teaspoons of sugar and/or orange juice or lemon juice or IV isotonic saline therapy (rarely required).

Heat Oedema:

- Mild swelling of hands and feet.
- Disappears after acclimatization.

Prickly heat:

- (such as miliaria or heat rash) manifests as small, red, itchy lesions on the skin caused by obstruction of the sweat ducts.
- It is best prevented by wearing light, loose clothing and avoiding heavy, continuous sweating.

Heat Syncope:

(In unacclimatized patients): Due to heat induced peripheral vasodilatation and pooling of blood, with subsequent loss of consciousness.

Treatment:

- Rest in a cool environment.
- Fluid repletion.

Major Heat Disorders:

Heat Exhaustion:

(In unacclimatized patients) Due to loss of salt and water.

Presentation:

- Headache, nausea, vomiting, dizziness, weakness, irritability, and cramps.

- O/E: Sweating, postural hypotension and normal or increased rectal temp < 40 c.

Treatment:

- Rest in a cool environment.
- Fan evaporation and water spraying.
- Salt-containing solutions Orally, if not due to vomiting Give IV isotonic saline one liter bolus then give 1.5 times maintenance rate and titrate to response .

Heat Stroke:

Clinical features:

- Triad of : Rectal Temp > 40.5 c, Altered mental status, Hot skin.
- Other features: tachycardia, hypotension, tachypnea, convulsions, and May have: diarrhea, bleeding tendency and aspiration pneumonia.

Common Laboratory Features;

- Hypokalemia, hyponatremia, hyperglycemia,
- Respiratory alkalosis ends up as metabolic acidosis,
- ARF : high urea and creatinine with hemogranular casts and proteinuria.
- DIC (low platelet, low Hb, high PT, high PTT, high D-Dimer (or FDP), low fibrinogen).

Treatment:

- Establish Airway and maintain Breathing.
- Circulation:
 - Establish an IV line.
 - check blood pressure (BP), if low give IV bolus of NS 500 ml then maintenance IVF NS at 125 ml/hour.
 - CVP line (to monitor fluid input).
 - Cardiac and hemodynamic monitoring.
 - Folly's catheter (to monitor urine output).
 - Monitor; vital signs, rectal temp, O2 saturation.
 - Input output monitoring and charting.
- For hyperthermia (Rectal temp > 40 C)
 - Cooling (MMBCU or fan and body spray) .
 - Stop cooling when rectal temp 39 C
 - No antipyretics should be used as it can exacerbate Heat stroke complications.
- For convulsions:
 - Valium 5-10 mg IV slowly PRN.
 - Phenytoin if repeated.
- For hypotension
 - IV fluid (adequate) .
 - Inotropes if no response to fluid.
- For bleeding:
 - Treat the underlying cause.
 - Packed RBCs, FFP, & Cryoprecipitate (if low fibrinogen) .
- For Acute renal failure:
 - R/O Rhabdomyolysis and treat with IV fluid.
 - Dopamine 3-5 µg/Kg/min.

- o Hemodialysis.
- For Acid base and electrolytes imbalance:
 - o Correct and replace accordingly.
- For hyperglycemia:
 - o Monitor.
 - o No insulin initially unless patient is known to be diabetic.
- For sepsis:
 - o Septic screen and IV antibiotics.
- For diarrhea:
 - o Only fluid replacement.

FLUID, ELECTROLYTES AND METABOLIC DISTURBANCES

Acute renal failure

Acute renal failure (ARF) has traditionally been defined as the abrupt loss of kidney function that results in the retention of urea and other nitrogenous waste products and in the dysregulation of extracellular volume and electrolytes. The loss of kidney function is most easily detected by measurement of the serum creatinine which is used to estimate the glomerular filtration rate (GFR).

Etiological Classification of ARF: Postrenal ARF (obstructive uropathy):

Account of < 5% of ARF , is common cause of ARF in elderly .

Causes of postrenal ARF :

- Upper urinary tract obstruction (stone, Clots ,tumor , external compression).

- Lower urinary tract obstruction (neurogenic bladder, prostatic enlargement, stone, urethral obstruction).

Diagnosis:

- Depends on ultrasound of kidney which reveals hydronephrosis. -

Management:-

- Relief obstruction.
- Lower tract obstruction by Foley's catheter.
- Upper tract obstruction by ureteral stent or percutaneous nephrostomy.

Prerenal ARF

Prerenal ARF is the clinical result of renal hypoperfusion due to decrease in effective arterial blood volume (account of 55-60% of ARF).

Causes of prerenal ARF:

- Volume contraction:
 - Hemorrhage: traumatic, surgical, GIT, and postpartum.
 - Gastrointestinal losses: vomiting, NG suction, diarrhea.
 - Renal losses: drug-induced or osmotic diuresis, diabetes insipidus, adrenal insufficiency.
 - Skin and mucous membrane losses: burns, hyperthermia, and other causes of increased insensible losses.
 - Third-space" losses: pancreatitis, crush syndrome, hypoalbuminemia.

- Prolonged hypotension.
- Low cardiac output like severe heart failure.
- Chronic liver disease (hepatorenal syndrome).

Clinical presentation: Clinically patient has signs of volume contraction like low BP, orthostatic hypotension, low urine output & signs of organ failure e.g. (CLD, CHF).

Investigation:

- High urea and creatinine.
- Low urine Na⁺
- Fraction excretion of Na⁺ < 1%.
- Normal size kidneys & normal urinalysis.

Management:

Treatment of underlying cause & adequate volume expansion (good rehydration) with proper IVF according to the cause.

Intrinsic renal failure:

Intrinsic renal failure is the most serious cause of ARF, accounts of 35-40 %.

Causes of Intrinsic renal failure :

- Disease involving large vessels:
 - Renal arteries: thrombosis, atheroembolism.
 - Renal vein: thrombosis.
- Diseases involving the glomeruli & small vessels:
 - Acute glomerulonephritis.
 - Vasculitis.
 - Thrombotic-thrombocytopenic purpura.
 - Malignant HTN.

- Radiographic contrast.
- Diseases involve renal tubules (ATN):
 - Exogenous toxins (eg. Aminoglycoside , contrast media)
 - Endogenous toxins (e.g. Myoglobin released in rhabdomyolysis , hemoglobin).
- Acute interstitial nephritis (e.g. antibiotic, NSAID...).
- Infection (viral, bacterial...).

Clinical manifestation:

- Clinically patients may be anuric (urine output < 100ml/d), oligouric (urine output <400ml/d) or non oliguric ARF ,also may have pulmonary oedema & high JVP .
- Patient may present with uremic symptoms (e.g.nousa,vomiting, lethargic, shortness of breth ,confusin, convulsion.....) .

Investigations:

- High creat &urea, high K.
- Nephritic or nephrotic range proteinurea.
- Urinalysis ---- many RBC, dysmorphic RBC & RBC cast (acute GN) , Granular cast (ATN), many WBC or WBC cast (AIN).
- Normal size kidneys on US.
- Positive serological tests like ANCA in vasculitis.

Management:

- Depends on underlying cause of ARF.
- Complete fluid intake & output record, daily RFT.
- Frequent assessment of volume status.

- Fluid challenge (NS 500-1000ml) over 1 h if patient is not overloaded .
- Possible diuretic if patient is overloaded.
- Pulse steroid in case of acute glomerulonephritis.
- Hemodialysis if there is:
 - Pulmonary oedema.
 - Sever hyperkalemia
 - Sever metabolic acidosis.
 - uremic pericarditis.
 - Uremic symptoms.
 - Uremic encephalopathy.

Electrolyte imbalance

Principles of Electrolyte Disturbances:

- Electrolyte imbalance implies an underlying disease process.
- Treat the electrolyte change, but seek the cause.
- Clinical manifestations usually not specific to a particular electrolyte change, e.g., seizures, arrhythmias.
- Clinical manifestations determine urgency of treatment, not laboratory values.
- Speed and magnitude of correction dependent on clinical circumstances.
- Frequent reassessment of electrolytes is required.

Hypokalemia

Defined as plasma $K < 3.5\text{Meq/L}$, symptoms seldom occurs unless plasma $K < 3.0$.

Etiologies of hypokalemia:

- Transcellular K shift:
 - Insulin therapy.
 - B-adrenergic agonist.
- Decreased K intake.
- Nonrenal K loss:
 - Profuse diarrhea.
 - Nasogastric suction.
 - Laxative abuse.

- Renal K loss:
 - Diuretic use.
 - Primary hyperaldosteronism.
 - Cushing syndrome.
 - Renal tubular acidosis (type 1&2).

Clinical Manifestations:

Cardiac: Arrhythmia.

Neuromuscular: fatigue, myalgia & muscle weakness of lower limb. More severe hypokalemia may lead to progressive weakness, hypoventilation, complete paralysis, in addition to risk of rhabdomyolysis.

Investigation:

- Plasma K < 3.5Meq/L (Deficit poorly estimated by serum levels).
- Random urine for K (< 20mmol/d--- nonrenal cause, >20 with renal loss).
- ABG to assess for acidosis.

Treatment:

- Oral KCl tab or syrup 600mg tid or Qid depends on severity.
- IV therapy for severe cases, should not be >20meq/hour.
- Titrate administration of K⁺ against serum level and manifestations.
- Correct hypomagnesemia .
- ECG monitoring with emergent administration.
- Treat hypokalemia urgently in acidosis.
- Treat underlying cause.

Hyperkalemia

Defined as plasma K > 5.0mEq/L.

Etiology:

- Decrease renal K excretion:
 - ARF (acute renal failure) .
 - Chronic kidney disease.
 - Addison disease.
 - Renal tubular acidosis Drugs e.g ACE inhibitors, ARB, heparin, aldactone.
- Tissue release & transcellular shift of K:
 - Hemolysis.
 - Rhabdomyolysis.
 - Tumor lysis syndrome.
 - Metabolic acidosis.
 - Drugs e.g. B-blocker.

Manifestations:

The clinical manifestation of hyperkalemia is weakness of lower limb which progress to flaccid paralysis & hypoventilation if respiratory muscle is involved.

The most serious effect of hyperkalemia is cardiac toxicity which does not correlate with plasma K. however there is correlation between s K level & ECG changes.

Mild hyperkalemia: 5.5–6.5 mmol/L: Tall peaked T waves with narrow base, best seen in precordial leads.

Moderate hyperkalemia: 6.5–8.0 mmol/L: Peaked T waves, Prolonged PR interval, Decreased amplitude of P waves and Widening of QRS complex.

Sever hyperkalemia: >8.0 mmol/L: Absence of P wave, Intraventricular blocks, fascicular blocks, bundle branch blocks, QRS axis shift , Progressive widening of the QRS complex, The earliest ECG changes include peaked T wave.



The terminal event is usually ventricular fibrillation or asystole.

Treatment:

- Stop intake.
- Give calcium for cardiac toxicity:
 - Ca gluconate 10ml of 10% infused over 2-3min , the dose can be repeated if no improvement in ECG within 10 min.
- Shift K⁺ into cell:
 - Insulin +D50% (10u regular insulin+ 50ml of D%50) bolus.
 - IV NaHCO₃ (3 ampules in 1L D5%) for sever hyperkalemia associated with metabolic acidosis.
 - Inhaledβ-agonist : ventolin neubilizer.
- Remove from body :
 - Ca resinum (Kayexalate) P.O 15-30gm TID or rectally 50g in 150ml water.
 - Hemodialysis for sever life- threatening not responding to medical therapy.

Hyponatremia

Definition: serum Na < 135meq/L, clinically only significant when it reflect hypo osmolality of the plasma. hyponatremia usually reflect excessive total body water relative to total body Na.

Etiologies and differentiation:

- Hypovolemic hyponatremia:
 - Vomiting, Diarrhea and Third-Space Fluid Loss (U_{osm}>300 mOsm/L , U_{Na}<20 mmol/L, FE Na <1%).
 - Diuretics, Aldosterone Deficiency and Renal Tubular Dysfunction (U_{osm}>300 mOsm/L , U_{Na}>20 mmol/L, FE Na >1%)
- Hypervolemic hyponatremia :
 - Congestive Heart Failure, Cirrhosis and Renal Failure With or Without Nephrosis (U_{osm}>300 mOsm/L, U_{Na}<10-20 mmol/L, FE Na <1%).
- Euvolemic hyponatremia:
 - Polydipsia and Inappropriate Water Administration to Children (U_{osm}<100 mOsm/L, U_{Na}>30 mmol/L).
 - SIADH, Hypothyroidism and Adrenal Insufficiency (U_{osm}>100 mOsm/L (usually >300), U_{Na}>30 mmol/L).

Manifestations of hyponatremia:

When serum $\text{Na} < 125$ meq/l mainly neurological secondary to cerebral edema (headache, confusion, coma if no treatment, tentorial herniation, respiratory depression & death).

Hyponatremia can be acute or chronic:

- Acute hyponatremia (within 48h) is at high risk for developing permanent neurological damage from cerebral edema if it remains uncorrected.
- Chronic hyponatremia is at risk of osmotic demyelination if it is corrected too rapidly.

Treatment:

- Hypovolemic hyponatremia: give normal saline, rule out adrenal insufficiency.
- Hypervolemic hyponatremia: increase free H_2O loss.
- Euvolemic hyponatremia :
 - Restrict free water intake
 - Increase free water loss
 - Normal or hypertonic saline
- Correct slowly due to possibility of demyelinating syndromes.
- Rate of correction of serum Na should not be >12 meq/24h.
- In asymptomatic patients : Fluid restriction is 1st line of therapy.
- Serum Na increased by 0.5 meq/L/hour.
- Symptomatic patients: Need hypertonic saline 3% NaCl. More aggressive correction at rate of 1.5—2

meq/L/hour, in 1st few hours. Serum Na should not exceed >12meq/24h.

- 1ml of 3% NaCl /kg/h - increase serum Na 1meq/l/h.

Hypernatremia

Defined as serum Na > 145meq/L.

Etiologies of hypernatremia:

- Hyponatremia secondary to water loss (commonest cause).
 - Nonrenal water loss e.g. GIT loss , burn.
 - renal water loss e.g. osmotic diuresis like in hyperglycemia , Mannitol & Diabetes insipidus (CDI,NDI).
- Impaired thirst:
 - Intubated patient in ICU.
 - Patient with impaired mental status.
 - Physically handicapped.
- Hyponatremia due to Na gain:
 - In Patient with DKA & osmotic diuresis treated with N saline.
 - Treatment with IV NaHCO₃ during resuscitation.

Manifestations:

- Mainly neurological include altered mental status, weakness, Coma & seizure.
- H₂O deficit (L) = $[0.6 \times \text{wt (kg)}] \times [\text{measured Na} - 1]140$

Treatment:

- Stop ongoing water loss.
- Correct water deficit.

- *Water deficit=plasma Na-140/140 x total body water in L.*
- Consider giving one-half of free H₂O deficit initially
- Water should be corrected slowly over 48-72h, at rate of 0.5meq/L/h, not exceed > 12meq/L/24h. (to avoid Secondary neurologic syndromes with rapid correction such as cerebral edema).
- The safest method to replace deficit through NGT by plain water.
- Alternatively intravascular 1/2 NS or 1/4 NS.
- Reduce Na cautiously: 0.5-1.0 mmol/L/hr.

Other Electrolyte Deficits (Ca, PO₄, Mg)

- May produce serious but nonspecific cardiac, neuromuscular, respiratory, and other effects
- All are primarily intracellular ions, so deficits difficult to estimate.
- Titrate replacement against clinical findings:
 - Hypocalcemia:
 - Calcium chloride or gluconate .
 - Bolus + continuous infusion.
 - Hypercalcemia :
 - Rehydration with normal saline.
 - Loop diuretics.
 - Hypophosphatemia:
 - Replacement iv for level < 1 mg/dL (0.32 mmol/L)
 - Hypomagnesemia :
 - Emergent administration over 5–10 mins.
 - Less urgent administration over 10–60 mins.

Acid Base Disturbances

Analysis of acid-base status is an important monitoring for the patient and requires an organized approach in the following sequence:

- Overall acid-base condition : normal PH 7.4 (low PH suggests acidemia and high PH suggests alkalemia)
- Metabolic or respiratory process: Look at PaCO₂ (respiratory) and HCO₃⁻ (metabolic). Normal HCO₃⁻ is 24 and normal Pco₂ is 40.
 - Metabolic Acidosis : ↓ PH - ↓ HCO₃⁻ - ↓ Pco₂
 - Respiratory Acidosis: ↓ PH - ↑ HCO₃⁻ - ↑ Pco₂
 - Metabolic alkalosis: ↑ PH - ↑ Pco₂ - ↑ HCO₃⁻
 - Respiratory alkalosis: ↑ PH - ↓ Pco₂ - ↓ HCO₃⁻
- Acute or chronic process if respiratory disturbance present for < or > 24 hours.
- Appropriate compensation process present if:
 - Metabolic Acidosis: Hyperventilation → ↑ Co₂ excretion → ↓ Pco₂ → compensatory respiratory alkalosis. Formula to calculate expected PCO₂. ((Pco₂ = (1.5 × HCO₃⁻) + 8 ± 2)).
 - Respiratory Acidosis: : (↑ serum HCO₃⁻)
 - Metabolic alkalosis: Pco₂ ↑ by 6 mmHg for every ↑ in serum HCO₃⁻ by 1 meq/L.
 - Respiratory alkalosis: (↓ serum HCO₃⁻)
- Always calculate Anion gap (AG)

Anion gap = (Na⁺ + K⁺) - (Cl⁻ + HCO₃⁻)

Normal anion gap : 9 ± 3 mEq /L

- Reveal additional information on albumin level.
- Note that an AG acidosis can exist even when the AG is normal; this is particularly true in critically ill patients with low albumin.

Expected AG decreases by 2.5-3 mmol/L for every 1 g/dL decrease in albumin

- Calculate the delta gap if a metabolic acidosis is present.

$\Delta\text{gap} = (\text{deviation of AG from normal}) - (\text{deviation of } [\text{HCO}_3] \text{ from normal})$

- Accurate analysis should lead to early interventions.

Metabolic Acidosis

Mechanisms:

- Increased acid intake or increased acid production, so, exceeding renal acid excretion (e.g. ketoacidosis or lactic acidosis).
- Renal acid excretion fails to match endogenous acid production (e.g. Renal Tubular Acidosis).
- Decreased bicarbonate by GIT loss (e.g. diarrhea or fistula).

Types of metabolic acidosis:

- **Normal anion gap metabolic acidosis:**
 - Causes:
 - RTA (Renal Tubular Acidosis).
 - Loss of HCO_3^- from GIT as in :
 - Diarrhea
 - Uretral diversion
 - ileostomy.

- High anion gap metabolic acidosis.
high anion gap : > 12 mEq /L
 - Causes:
 - Diabetic ketoacidosis (DKA).
 - Alcoholic ketoacidosis .
 - Lactic acidosis.
 - Salicylate intoxication.
 - Ethylene glycol & Methanol intoxication.
 - Advanced renal insufficiency.

Treatment of acute metabolic acidosis:

- Reversal of the underlying cause as :
 - Use of insulin for DKA.
 - Restoration of tissue perfusion in lactic acidosis.
- Alkali administration (Na^+ bicarbonate) :
Only in severe acidemia ($\text{PH} < 7.2$).
- Base deficit must be calculated :
If serum $\text{HCO}_3^- > 10$ mEq /L:
Base deficit = Desired $\Delta\text{HCO}_3^- \times$ body weight (kg) \times 0.5.

Respiratory Acidosis (CO_2 retention)

Hypoventilation \rightarrow CO_2 retention \rightarrow \uparrow $\text{Pco}_2 \rightarrow$ acidosis.

Acute respiratory acidosis: (< 24 hours):

- \uparrow In HCO_3^- by 1 mEq /l for every \uparrow in Pco_2 by 10 mmHg. $\Delta \text{HCO}_3^- = 0.1 \times \Delta \text{Pco}_2$.

Chronic respiratory acidosis:

- \uparrow In HCO_3^- by 4 mEq /l for every \uparrow in Pco_2 by 10 mmHg. $\Delta \text{HCO}_3^- = 0.4 \times \Delta \text{Pco}_2$.

Causes:

- Severe pulmonary disease :
 - COPD (e.g. bronchospasm - emphysema).
 - Adult respiratory distress syndrome.
- Respiratory muscle fatigue :
 - Neuromuscular diseases (e.g. poliomyelitis – myasthenia).
 - Primary muscle disease.
- Depression of the respiratory centre :
 - 1ry depression by drugs, stroke or infection.
 - ↓ Stimulation of the respiratory centre (sleep apnea).

Treatment:

1. Reversal of the underlying cause.
2. Restoration of adequate alveolar ventilation by :
 - Tracheal intubation.
 - Mechanical ventilation if needed.

Metabolic alkalosis

Due to \uparrow serum $\text{HCO}_3^- \rightarrow \uparrow$ pH \rightarrow compensatory hypoventilation \rightarrow CO_2 retention $\rightarrow \uparrow$ PCO_2 .

Causes of \uparrow serum HCO_3^- :

- Exogenous administration of HCO_3^- , or
- Acid loss from the kidney or GIT.

Types of metabolic alkalosis :

- Chloride-responsive metabolic alkalosis
- Chloride-resistant metabolic alkalosis

Chloride-responsive metabolic alkalosis:

(Volume depletion). Characterized by: Low urinary Cl- < 20 meq/L.

Causes:

- **GIT causes:**
 - Vomiting.
 - Cl- losing diarrhea.
 - Naso-gastric suction.
 - Villous adenoma.
- **Renal causes:**
 - Diuretics.
 - Post-hypercapnic state.
 - Hypomagnesemia.
 - Bartter's syndrome.

Manifestations :

Manifestations of volume depletion.

Treatment of Cl- responsive metabolic alkalosis :

- Treatment of the underlying cause :
- Administration of NaCl (normal saline) is sufficient to reverse alkalosis.
- Administration of carbonic anhydrase inhibitors (acetazolamide 250 mg twicedaily) may be used to accelerate renal HCO₃ loss.

Chloride-resistant metabolic alkalosis

(Volume expansion), Characterized by : Normal urinary Cl- excretion > 20 meq/L.

Causes :

- **High rennin**
 - Renal artery stenosis
 - Rennin secreting tumors.

- Estrogen therapy.
 - **Low rennin**
 - 1ry hyperaldosteronism :Adenoma, hyperplasia or carcinoma.
 - Adrenal enzyme defects.
 - Cushing's syndrome.
 - Liddle's syndrome.
- Treatment** of Cl-resistant metabolic alkalosis :
- Spironolactone for 1ry hyperaldosteronism or treatment of the underlying cause for 2ry hyperaldosteronism.
 - Correction of hypokalemia.

Respiratory alkalosis (hypocapnia)

Alveolar hyperventilation → ↑Co₂ output from the lungs
→ ↓PCo₂ → alkalosis.

Acute respiratory alkalosis:

Serum HCO₃ ↓ by 2 meq/L for every ↓ in PCo₂ by 10 mmHg.
 $\Delta \text{HCO}_3 = 0.2 \times \Delta \text{Pco}_2$.

Chronic respiratory alkalosis :

Serum HCO₃ ↓ by 5 meq/L for every ↓ in Pco₂ by 10 mmHg.
 $\Delta \text{HCO}_3 = 0.5 \times \Delta \text{Pco}_2$.

Causes of respiratory alkalosis :

- ↑CNS drive for respiration:
 - Anxiety. CNS infection, infarction or trauma.
 - Drugs : salicylates, nicotine or aminophylline.
 - Pregnancy, progesterone.
 - Fever, sepsis. Liver disease

- ↑ Stimulation of chemoreceptors:
 - Anemia .
 - Carbon monoxide toxicity.
 - Pulmonary edema
 - Emboli or pneumonia.
 - High altitude: ↓ inspired O₂ tension.
- ↑ Mechanical ventilation.
- Iatrogenic.

Treatment of respiratory alkalosis:

The only effective therapy is to eliminate the cause of the hyperventilation.

Diabetic ketoacidosis (DKA)

Diabetic ketoacidosis is a potentially life threatening condition in patients with absolute or relative insulin deficiency. DKA is characterized by the triad of hyperglycemia, anion gap metabolic acidosis, and ketonemia.

PRECIPITATING FACTORS

- New onset type 1 diabetes, in which DKA is a common presentation.
- Underlying infection: (pneumonia, influenza, gastroenteritis, urinary tract infection).
- Poor compliance with the insulin regimen.
- Acute major illnesses such as myocardial infarction, cerebrovascular accident, or pancreatitis.
- Drugs that affect carbohydrate metabolism, including glucocorticoids, higher dose thiazide diuretics, sympathomimetic agents (eg, dobutamine and terbutaline), and second-generation antipsychotic agents.
- Cocaine use, which has been associated with recurrent DKA.

Clinical presentation:

Symptoms: (usually evolve over the period of about 24 hours).

- Predominant symptoms:
 - The earliest symptoms of marked hyperglycemia are polyuria, polydipsia, and weight loss
 - Nausea, vomiting, and abdominal pain that may be severe to the point that an acute abdomen may be suspected.
- In severe DKA:
 - Hyperventilation due to metabolic acidosis.
 - There may be confusion, lethargy, stupor or even coma.

Physical examination:

- Dry mouth and decreased skin turgor.
- If the profound dehydration is enough to cause a decrease in the circulating blood volume, tachycardia and low blood pressure may be observed.
- a fruity odor may be present (due to exhaled acetone and similar to the odor of nail polish remover),
- deep respirations reflecting the compensatory hyperventilation (called Kussmaul respirations).

Investigations:

- Diabetic ketoacidosis is diagnosed when the combination of hyperglycemia (over 300 mg/dL), ketones in serum or urine and an anion gap metabolic acidosis are present.
- Urea and creatinine (kidney function may be impaired as a result of dehydration) .
- Electrolytes.
- Markers of infection (complete blood count, C-reactive protein) and acute pancreatitis (amylase and

lipase) may be measured. Given the need to exclude infection, chest radiography and urinalysis are usually performed.

- Arterial blood gas measurement is usually performed to demonstrate the severity;
 - *Mild*: blood pH mildly decreased to between 7.25 and 7.30 (normal 7.35–7.45); serum bicarbonate decreased to 15–18 mmol/l (normal above 20); the patient is alert
 - *Moderate*: pH 7.00–7.25, bicarbonate 10–15, mild drowsiness may be present
 - *Severe*: pH below 7.00, bicarbonate below 10, stupor or coma may occur. (If cerebral edema is suspected because of confusion, recurrent vomiting or other symptoms, computed tomography may be performed to assess its severity and to exclude other causes such as stroke).

Management:

The main aims in the treatment of diabetic ketoacidosis are replacing the lost fluids and electrolytes while suppressing the high blood sugars and ketone production with insulin. Admission to an intensive care unit for close observation may be necessary.

Fluid replacement:

- The amount of fluid depends on the estimated degree of dehydration. If dehydration is so severe as to cause shock :
 - Administer high volumes of isotonic saline (1-3 L) in the first hour. In the absence of cardiac

compromise, isotonic saline is infused at a rate of 10 to 15 mL/kg lean body weight per hour (about 1000 mL/hour in an average-sized person) during the first few hours, with a maximum of <50 mL/kg in the first four hours.

- The subsequent choice for fluid replacement depends upon the state of hydration, serum electrolyte levels, and the urine output. Most patients are switched at some point to one-half isotonic saline to replace the free water loss induced by the glucose osmotic diuresis. In general, one-half isotonic saline infused at 4 to 14 mL/kg per hour is appropriate if the corrected serum sodium is normal or elevated; isotonic saline at a similar rate is appropriate if the corrected serum sodium is low.
- A special but unusual consideration is cardiogenic shock, where the blood pressure is decreased not due to dehydration but due to inability of the heart to pump blood through the blood vessels. This situation requires ICU admission, monitoring of the central venous pressure, which requires the insertion of a central venous catheter, and the administration of medication that increases the heart pumping action and blood pressure.

Insulin:

- Insulin is given at 0.1 unit/kg per hour to reduce the blood sugars and suppress ketone production. Insulin should be started about an hour after intravenous fluid replacement is started to allow for checking potassium levels and because insulin may

be more dangerous and less effective before some fluid replacement has been obtained.

- When glucose levels have dropped to 250 mg/dL, IV fluids should be switched to D5 /1/2 NSS to prevent hypoglycemia recognizing that insulin is still needed to treat ketonemia.

Potassium:

- Potassium levels can fluctuate severely during the treatment of DKA, because insulin decreases potassium levels in the blood by redistributing it into cells.
- Continuous observation of the heart rate is recommended, as well as repeated measurement of the potassium levels.
- To prevent hypokalemia, potassium chloride (20 to 30 meq/L) is generally added to the replacement fluid once the serum potassium concentration falls below 5.3 meq/L, assuming an adequate urine output (>50 mL/hour). If the patient is hemodynamically stable, one-half isotonic saline is preferred since the addition of potassium to isotonic saline will result in a hypertonic solution that will delay correction of the hyperosmolality. The serum potassium should be maintained between 4.0 and 5.0 meq/L.
- Potassium repletion is more urgent in patients with massive potassium deficits who are hypokalemic prior to therapy. Such patients require aggressive potassium replacement (20 to 30 meq/hour), which usually requires 40 to 60 meq/L added to one-half isotonic saline. Since insulin will worsen the

hypokalemia, insulin therapy should be delayed until the serum potassium is above 3.3 meq/L to avoid possible arrhythmias, cardiac arrest, and respiratory muscle weakness.

Bicarbonate:

Bicarbonate is administered only if the arterial pH is less than 7.35. It is given as 100 meq of sodium bicarbonate in 400 mL sterile water with 20 meq of potassium chloride, if the serum potassium is less than 3.5 meq/L, administered over two hours. The venous pH should be monitored every two hours, and bicarbonate dosed as above, until the pH rises above 7.35.

Detection and treatment of an underlying illness:

- Infection, pancreatitis, cerebrovascular accident (CVA), MI, sepsis, or deep venous thrombosis (DVT).

Consider care for cerebral edema:

If associated with coma, often necessitates admission to intensive care, artificial ventilation, and close observation. Intravenous administration of mannitol (0.25 to 1.0 g/kg) and perhaps from hypertonic (3 percent) saline (5 to 10 mL/kg over 30 min). These approaches raise the plasma osmolality, resulting in osmotic movement of water out of the brain and a reduction in cerebral edema.

Note:

- Use data flow sheets to monitor timing of laboratory tests and therapy.
- Ketoacidosis is not always the result of diabetes. It may also result from starvation.

Hyperosmolar hyperglycemic state

(Non ketotic hyperosmolar hyperglycemia)

In Hyperosmolar hyperglycemic state (HHS), there is little or no ketoacid accumulation, serum glucose concentration frequently exceeds 1000 mg/dL (56 mmol/L), plasma osmolality may reach 380 mosmol/kg, and neurologic abnormalities are frequently present (including coma in 25 to 50 percent of cases). Most patients with HHS have an admission pH >7.30, a serum bicarbonate >20 meq/L, a serum glucose >600 mg/dL (33.3 mmol/L), and test negative for ketones in serum and urine, although mild ketonemia may be present, the clinical features of HHS and DKA overlap and observed simultaneously in as many as one third of cases.

Precipitating Factors:

A common events are infection (often pneumonia or urinary tract infection) and discontinuation of or inadequate insulin therapy. Compromised water intake due to underlying medical conditions, particularly in elderly patients, can promote the development of severe dehydration and HHS.

Other factors include:

- Precipitating event can usually be identified in patients with HHS. The most infections such as pneumonia and urinary tract infections.

- Stroke or heart attack.
- Heat stroke.
- Trauma or severe burns.
- Pancreatitis.
- Medicines that raise blood sugar.
- Poor compliance with the insulin regimen or diabetes medication at all, or taking them incorrectly.
- Undiagnosed diabetes.

Clinical presentation:

Signs and symptoms of HHS usually develop over days or weeks.

- The signs and symptoms that appear first are caused by high blood sugar levels. Blood sugar levels are usually over 600 mg/dL.
 - Blurred vision .
 - Feeling very tired.
 - Frequent urination.
 - Leg cramps.
 - More thirsty than usual.
 - Weight loss.
- Later signs and symptoms are caused by dehydration:
 - Dry eyes or mouth.
 - Weakness.
 - Leg cramps.
 - Dizziness.
 - Seizure
 - Drowsiness or confusion.
 - Coma (HHS was previously termed hyperosmolar hyperglycemic nonketotic coma (HHNC)).

However, the terminology was changed because coma is found in fewer than 20% of patients with HHS).

- Irregular or fast breathing, fast or pounding heartbeat, and low blood pressure

Investigation (Diagnostic features):

- Plasma glucose level of 600 mg/dL or greater
- Effective serum osmolality of 320 mOsm/kg or greater
- Profound dehydration up to an average of 9L
- Serum pH greater than 7.30
- Bicarbonate concentration greater than 15 mEq/L
- Small ketonuria and absent-to-low ketonemia

Emergency treatment:

- Manage the airway as needed, oxygenate and establish intravenous access.
- **fluid resuscitation:**
 - (Fluid deficits in hyperosmolar hyperglycemic state (HHS) are large; the fluid deficit of an adult may be 10 L or more and fluid likely will replenish intravascular volume and correct hyperosmolarity).
 - Administer 1-2 L of isotonic saline in the first 2 hours. A higher initial volume may be necessary in patients with severe volume depletion. Slower initial rates may be appropriate in patients with significant cardiac or renal disease. Caution should be taken to not correct hyponatremia too quickly, as this could lead to cerebral edema.

- After the initial bolus, switch to half-normal saline once blood pressure and urine output are adequate.
- Once serum glucose drops to 250 mg/dL, the patient must receive dextrose in the intravenous fluid. This may decrease the risk of developing cerebral edema.
- **Insulin:**
 - Although many patients with HHS respond to fluids alone, intravenous insulin in dosages similar to those used in DKA can facilitate correction of hyperglycemia.
 - Insulin used without concomitant vigorous fluid replacement increases risk of shock.
- **potassium and magnesium:**
 - Replete potassium and magnesium as needed. Use of insulin may exacerbate hypokalemia.
- **Detection and treatment of an underlying illness** is critical. Antibiotics need to be administered early.
- **Frequent reevaluation** of the patient's clinical and laboratory parameters is necessary. Recheck glucose concentrations every hour. Electrolytes and VBGs should be monitored every 2-4 hours or as clinically indicated.
- All patients diagnosed with HHS require hospitalization, usually to an intensive care unit for close monitoring.

GENERAL PRINCIPLES

Assessment of seriously ill patient

Why should I Identify Patients at Risk of Severe Illness?

- To maximize likelihood of easier management with simpler interventions.
- To prevent further deterioration.
- To provide time for investigation and treatment.

Worrisome Findings:

- Respiratory rate and pulse if increased;
- Increase in pulse pressure with normal or decreased BP.
- O₂ saturation if abnormal, especially on oxygen supplementation.
- Mental status change.
- Bibasilar rales may suggest pulmonary edema.
- Decreased bowel sounds and distension might be consistent with complication of recent surgery.
- Warm extremities would raise a concern initially.

Approaching a seriously ill patient:

Primary survey: which is concerning about:

- What is main physiological problem?
- First minutes of initial contact and resuscitation.

Secondary survey:

- What is underlying cause?
- Subsequent reviews

Primary survey:

Approach is according to ABCDs in primary survey: *Resuscitate any physiological problem hand on hand while evaluating the following:*

Airway:

- ***Maintain airway and oxygenate :***
 - Check for signs of airway obstruction, feel for airflow, presence of cyanosis.
 - Level of consciousness.
 - Oxyhemoglobin saturation.
- **Breathing:**
 - ***Maintain adequate ventilation if required:***
 - Observe chest movement.
 - Respiratory rate and pattern.
 - Tachypnea is the single most important indicator of critical illness.
 - Use of accessory muscles.
- **Circulation:**
 - ***Provide two IV accesses, extract blood for basic investigations, start bolus IV fluid (NS or ringer lactate) and evaluate :***

- Blood pressure.
- Evidence of decreased perfusion: Cool extremities, Pallor and Decreased urine output.
- Abnormal heart sounds.
- Central and peripheral pulses. Rate, quality, regularity and symmetry .
- **(D) Consciousness:** Altered mental status.

Note that the actions and other parts of the assessment will often occur in parallel to Ensure physiological safety:

- Oxygen supplementation and inspired oxygen concentration.
- Intravenous access.
- Institute fluid resuscitation
- Circulatory support.
- Vital signs.
- Fluid balance.
- Invasive parameters (CVP, arterial line)
- Check lactate
- Obtain cultures and consider empiric antibiotics
- Obtain chest radiograph.
- Medications.
- Consider transfer to a higher level of care.
- Call for help.

Any main physiological problem shall be resuscitated before proceeding to secondary survey.

Secondary Survey

1. History:

- Main symptoms
- Coexisting illness (fever, vomiting, etc).
- Type of surgery.
- Severe hemorrhage / transfusion during or after surgery.
- Medications (DVT prophylaxis; chronic meds, such as a diuretic, not given).
- Past hospitalization, chronic diseases.
- Hospital course prior to event (improving, worsening).
- Psychosocial issues (anxiety, drug dependence).
- Allergies.
- Ethical / legal issues.
- Systems review.

2. Physical examination:

The approach is according to system review:

- Respiratory.
- Cardiovascular.
- Abdomen and genitourinary tract.
- Central nervous system.
- Musculoskeletal system.
- Endocrine, hematologic systems.

3. Document review:

- Previous vital signs and pulse oximetry—Is this BP normal or low for the patient? What has been the trend in respiratory rate?
- Were rales present on previous examination?

- Has the patient been confused before?
- What was the prior abdominal examination?
- Urine output
- Medications
- DVT prophylaxis

Adequate documentation is needed to assess changes and trends in patient's condition.

4. Investigations

- Guided by history and physical examination
- Biochemistry, hematology, cultures, radiographs
- Arterial or venous blood gas
- Lactate level
- Metabolic acidosis is an important indicator of critical illness

5. Assess the likely diagnosis and treatments:

- Document diagnosis and treatment rationale.
- Find concern about the new information such as:
 - BP decreases from baseline and pulse increases from baseline.
 - Oxygen saturation deterioration from baseline.
 - WBC count increases.
 - Renal function worsening.
 - ABG documents presence of metabolic acidosis.
 - Determine patient's reserve.
 - Document current events.
- Refine treatment:
 - Assess response to treatment.
 - Provide organ system support.
 - Determine best site for care.
 - Call for advice and assistance.

Disaster Management and Emergency Preparedness

The Aim is *“to deliver The greatest good for the greatest number of pilgrimages”*

Phases of disaster response:

- **Emergency Preparedness:**
 - Phase 1: Preparation:
 - By having a straightforward disaster plan, simple and to train all staff in its application by awareness and repeated drills.
 - Phase 2: Mitigation:
 - Have an Incident Command System which is the organizational structure that provides overall direction for management of the disaster response, with Horizontal and Vertical reporting and to train all staff in its application by awareness and repeated drills.
- **Disaster Management:**
 - Phase 3: Response:
 - Prehospital and Inhospital Care:
 - ◆ Decontaminate every patient: Assume every patient is contaminated. Don't take your facility out of function by

- contaminating the facility. So decontaminate and quarantine if necessary.
- ◆ Disaster triage scheme: Triage categories correspond to color coded tags (see below).
 - ◆ Effective surge capability: Surge capability is the number of additional beds, ventilators, monitors and personnel that can be pressed into service during a disaster response. Plan for a 20% increase in volume. with reliable supply chains
 - ◆ Life-saving procedures: Provide minimal acceptable care for the first 24 hours to maximize the greatest good for the greatest number of patients.
 - ◆ Traffic control system: Control the flow of patients, information, supplies, and personnel to be unidirectional.
 - ◆ Special needs patients: Special needs patients need predetermined response plans. Lost pilgrimages, children, elders, disabled, dispossessed.
 - ◆ Communications : be ready for systems failure and have alternate modes
- Pathophysiology and Patterns of Injury:
- ◆ All natural and most terror disasters cause injury : “Circumstances are extraordinary but care is ordinary”

- ◆ All blast injuries can cause major trauma, burns: Early tracheal intubation, judicious fluid for blast lung and major burn.
- ◆ All chemical agents require decontamination: Decontamination of chemical agents should be done at scene or outside hospital to prevent closure of your facility, Beware of potential contaminants after bomb blast.
- ◆ All radiological agents require decontamination: With radiologic exposure, the risk to providers is minimal. Decontamination should not delay necessary care; Necessary procedures should be done before day 3 (before hematologic effects of radiation exposure develop).
- Phase 4: Recovery: return to normal resources.

Disaster triage scheme

Field triage:

During Hajj the system most widely recognized and used is the **S.T.A.R.T. system** "Simple Triage and Rapid Transport".

TRIAGE System will categorize patients into 4 groups:

1. **RED** (P1 – IMMEDIATE) = patients are those who are critically injured and in need of immediate intervention to correct. In case of Disaster or M.C.I. they are patients with a very good chance to live by a simple

measure (do not need constant intensive care) e.g. hypoxia, shock.

2. **YELLOW** (P2 – DELAYED) = is allowed for care after (P1) and it will not change the outcome and they get a good chance to live.

3. **GREEN** (P3 – AMBULATORY PATIENTS) = Do not need stretcher to shift most of the Disaster victims.

4. **BLACK** (P0) = known also as expectant patients, are deceased or with such catastrophic injuries that they are not expected to survive, to be transported (last to be moved).

The S.T.A.R.T. System directs you to first collect anyone who walk at the incident and tag them as GREEN (Priority 3). Not breathing, open the airway manually if remain apneic 10 seconds, tag them BLACK (Priority Zero).

RED (Priority 1) Breathing < 10 or >30/min. Capillary refill > 2 seconds or absence of radial pulse (BP < 90 mmHg) or mentally cannot follow simple commands such as handgrip.

YELLOW (Priority 2) Breathing > 10 or < 30 min. Capillary refill < 2 seconds or presence of radial pulse (BP ≥ 90 mm Hg) mentally can follow simple commands such as handgrip.

TRIAGE of patient should take < 60 seconds per patient. Pre-hospital stabilization should not delay TRIAGE and should be limited to establishment of airway (oropharyngeal or nasopharyngeal airway), C-spine, pressure dressing to control bleeder. Limbs and spine stabilization and IV insertion.

Once patient are triaged they need to be extricated from their environment and removed to the treatment areas (according to priority), where they should be re-triaged frequently as conditions may be change rapidly.

Hospital or secondary triage is a more refine and specific triage.

Triage in ED:

- **EMERGENCY (RED):** Life threatening conditions:
 - Those who may die without STAT treatment. Patient who's ABCs are compromised.
- **URGENT (YELLOW):**
 - Those with serious conditions who need treatment quickly to prevent further complication.
 - Those whose condition needs investigation and treatment.
 - Should be seen by physician and treatment began within an acceptable time frame, as assessed necessary by triage nurse. Usually 20 to 60 min.
- **ROUTINE (GREEN):**
 - Patients who have conditions that is in no danger if treatment is delayed.
 - Patient who can safely wait to see physician as time permits.
 - According to availability of treatment areas and providers.

Initial management of multiply injured patient

Management of the multiply injured patient requires a co-ordinated multi-disciplinary approach in order to optimise patient outcomes. It is essential to recognize the life-threatening injuries and treat in a timely fashion and that more minor associated injuries are not forgotten. This topic outlines the management of polytrauma patients using the Advanced Trauma Life Support (ATLS) principles.

Initial assessment include:

1. Primary survey (ABCDE).
2. Resuscitation.
3. Secondary survey (head to toe).
4. Definitive care.

Initial assessment could be started by asking the patient his / her name which is a quick, simple way to assess the patient in 10 seconds,

Appropriate response confirms patent airway, sufficient air reserve to permit speech and clear sensorium.

If no response, proceed with primary survey.

1: Primary survey - assessment of ABCDs:

Primary survey and Resuscitation should go hand in hand with each other.

- A. Airway patency with cervical spine protection
- Perform a chin lift or jaw thrust maneuver.
 - Clear the air way of foreign bodies. Insert oropharyngeal/nasopharyngeal air way.
 - Establish a definitive air way (oro-tracheal or nasotracheal intubation / cricothyroidotomy
 - Immobilize & maintain cervical spine in a neutral position.
- B. Breathing : ventilation and oxygenation
- Administer high concentrations of oxygen.
 - Ventilate with Ambo- bag if necessary.
 - Inspect and palpate the neck for tracheal deviation.
 - Auscultate the chest bilaterally.
 - Percuss the chest for presence of dullness or hyperresonance.
 - Needle decompression of pleural space or tube thoracostomy , as indicated.
 - Seal open pneumothorax.
 - Attach the patient to a pulse oximeter .
- C. Circulation with hemorrhage control
- Apply direct pressure to external bleeders.
 - Insert two large caliber IV lines, simultaneously obtain blood for hematologic and chemical analysis, pregnancy test, type and crossmatch.
 - Initiate iv fluid therapy with warmed ringer's lactate solution or normal saline.

- Record Blood pressure.
 - Blood replacement if required.
 - Persistent infusion of large volumes of fluids in an attempt to achieve a normal blood pressure is not a substitute for definitive control of bleeding, so consider presence of internal hemorrhage and obtain surgical consultation and shall be shifted to operating theater if having bleeding.
- D. Disability: brief neurologic examination
- Determine the level of consciousness using GCS. Assess the pupils for size, equality, and reaction.
- E. Exposure / environment:
- Completely undress the patient but prevent hypothermia.
- Reassess the patient ABCDE and consider the need for patient transfer .
- Adjuncts to Primary Survey:
- Vital signs, ABGs, Pulse oximeter and CO₂, ECG, Urinary / gastric catheters unless contraindicated, Urinary output.

2: Resuscitation:

- Airway protected and secured
- Ventilated and oxygenated, ICT inserted.
- External bleeding controlled.
- Vigorous shock therapy
- Protected from hypothermia

3. Secondary survey:

History:

- Allergies.
- Medications currently taken.

- Past illnesses.
- Last meal.
- Events related to injury (*mechanism of injury*).

Examination:

Head and maxillofacial, Cervical spine and neck, Chest, Abdomen, Perineum, rectum, vagina, Back, Musculoskeletal, Neurologic.

Re-evaluate the patient , noting , reporting , and documenting any changes in the patient's condition and responses to resuscitative efforts.

Judicious use of analgesics may be employed only after surgical consultation.

Continuous monitoring of vital signs and urinary output is essential.

4. Definitive care & transfer Begins after:

- Identifying the patient's injuries.
- Managing life - threatening problems.
- Obtaining special studies.

If required, the Patient have to be transferred to the nearest appropriate facility after consulting the concerned surgeon depending on the injuries sustained.

General principles in emergency drugs

ADENOSINE: Antiarrhythmic in PSVT: 6 mg rapid IV push, if no response in 1-2 minutes, administer dose of 12mg, repeat 12 mg dose a second time if required.

AMIODARONE: Antiarrhythmic: Loading dose: 150mg Amiodarone IVP (up to 300mg IVP for cardiac arrest from VF/VT after multiple shocks) for life threatening arrhythmia. 150mg Amiodarone may be mixed in 100cc D5W and infused over 10 minutes or given IVP. Follow with IV infusion upon arrhythmia resolution, IV infusion: 450mg. Amiodarone in 250ml D5W. Initial infusion 1gm/min x 6 hours (33ml/hr x 6 hours). Decrease infusion to maintenance dose of 0.5mg/min (17ml/hr).

ATROPINE: Agent used for symptomatic bradycardia, PEA: 0.5-1 mg IV push, repeat at 3-5 min. Intervals to max. Total dose of .04 mg/kg. May be given via endotracheal route. Stocked 1 mg/10 ml.

DEXTROSE: Antihypoglycemic: 10 to 25 gms. IVP. Repeat dose as required in severe cases depending on symptoms and blood glucose levels.

DOBUTAMINE: Vasopressor: IV infusion: 500 mg Dobutamine in 250 ml IV solution. Usual dose 2-5mcg/kg/min. May titrate to upper dose of 20mcg/kg/min. primarily stimulates B-1 receptors in the heart and is used for inotropic support with mild chronotropic effect. Adequate hydration of patient imperative in blood pressure support. When mixing more than 500mg. Dobutamine in IV solution, equal volume must be removed (e.g. 1gm/40ml Dobutamine, remove 40ml from IV solution).

DOPAMINE: Vasopressor, IV infusion: Usual dose in code situation is 5-20mcg/kg/min. Renal perfusion dosing 2-5mcg/kg/min, increase of cardiac output 5-10mcg/kg/min and peripheral vasoconstriction 10-20mcg/kg/min. As approaching 20mcg/kg/min assess urine output. Extravasation treatment is with phentolamine. Adequate hydration of patient imperative in blood pressure support. Premix drip of 400 mg Dobutamine in 250 ml IV solution.

EPINEPHRINE: Adrenergic agent of choice for cardiac arrest, vasopressor used in Pulsless VT/VF, Asystole and PEA: 1 mg IV every 3-5 min. Or more frequently. May be given endotracheal route, Stocked 1 mg/10 ml 1:10,000. If using for hypersensitivity reaction 0.1-0.25mg SQ, SIVP. IV infusion, mix 1 mg in 250 ml IV solution. Start at 1 mcg/min. (15 cc/hr.), each 15 cc/hr. = mcg/min. Stocked 1 mg/1 ml in 1ml single dose

ampules and 30ml multidose vials. Bristojects are 1mg/10ml.

ETOMIDATE: Induction of rapid hypnosis (non-barbiturate) usual dose 0.2-0.6mg/kg IVP immediately prior or during intubation.

FLUMAZENIL: Antidote for benzodiazepine: Initial dose 0.2mg administered IV over 15 seconds. If desired level of consciousness is not obtained after waiting additional 45 seconds, second dose of 0.2mg may be administered. Repeat at 60-second intervals as necessary to a maximum total dose of 1mg. Individualize the dose based on patient response.

LIDOCAINE: Antiarrhythmic: Bolus: 1-1.5 mg/kg IV push initial bolus. Repeat doses of 0.5-1 mg/kg at 5-10 min. intervals if needed until total dose of 3 mg/kg for cardiac arrest from VF/VT that persists after multiple shocks. May be given via endotracheal route. Follow with IV infusion if effective in arrhythmia resolution. IV infusion: use-premixed bag of 2 Gm in 500 cc D5W. 1-4 mg/min. (1mg/min = 15cc/hr of premix Lidocaine drip).

MAGNESIUM: In cardiac arrest: 1-2 gm. (diluted in 10ml D5W or NS) IVP in torsades de pointes or when it is suspected that the arrhythmia is caused by a hypomagnesemic state.

NALOXONE: Antidote for opiate narcotics: Initial dose of 0.4mg to 2mg. IV; may repeat IV at 2 to 3 minute

intervals. If no response is observed after 10mg has been administered, question the diagnosis of narcotic induced or partial narcotic induced toxicity. Can also be given IM or SQ.

NOREPINEPHRINE: Vasopressor, IV infusion: mix 4 mg in 250-cc IV solution (in D5W only). Usual dose 2-12 mcg/min to titrate for blood pressure control. Monitor urine output. Adequate hydration imperative in blood pressure control.

PROCAINAMIDE: Antiarrhythmic: Bolus: 100 mg IV every 5 min. (or at a rate of 20mg/minute IVSP) until one of the following observed:

- 1) arrhythmia suppressed;
- 2) hypotension ensues;
- 3) QRS complex is widened by 50% of its original width;
- 4) a total of 17 mg/kg. If effective administer IV infusion.

IV infusion: 1 gm Procainamide in 250 ml IV solution
Stocked 1 gm/10 ml. Range: 1-4 mg/min.
(1mg/min = 15cc/hr).

SODIUM BICARBONATE: Should be used, if at all, only after application of definitive and better substantiated interventions and drugs. 1 mEq/kg IV push. Repeat doses are usually 0.5 mEq/kg, based on ABG results. Stocked 1 mEq/1 ml 10ml and 50ml Bristojects.

VASOPRESSIN: Antidiuretic hormone, vasoconstrictor: 40U as a single, one-time bolus. Alternative agent to epinephrine, vasopressin is used in persistent or recurrent VT/VF for vasoconstriction and increasing blood flow to the brain and heart during CPR. Use epinephrine to follow up in 10 minutes if there is no response to vasopressin.

VERAPAMIL: Calcium channel blocker: 5 mg IV push given over 1 min. Repeat in 30 min. if needed, increasing to 10 m.

HAJJ POSSIBLY ENCOUNTERED PROBLEMS

Acute abdomen

Acute abdomen is a general name for presence of signs, symptoms of inflammation of peritoneum; it may indicate a life-threatening intra-abdominal pathology. Determining exact cause is irrelevant in pre-hospital care. But the Important factor is recognizing that acute abdomen is present.

Common conditions:

Appendicitis:

- Usually due to obstruction of appendix with fecolith
- Appendix becomes swollen, inflamed gangrene, possible perforation
- Pain begins periumbilical; moves to RLQ
- Nausea, vomiting, anorexia often present.
- Patient lies on side; right hip, knee flexed
- Tenderness present in RIF.
- Pain may not localize to RLQ if appendix in odd location

Duodenal Ulcer Disease:

- Steady, well-localized epigastric pain
- “Burning”, “gnawing”, “aching”
- Increased by coffee, stress, spicy food, smoking
- Decreased by alkaline food, antacids
- May cause massive GI bleed
- Perforation = intense, steady pain, patient lies still, rigid abdomen

Kidney Stone:

- Mineral deposits form in kidney, move to ureter
- Often associated with history of recent UTI
- Severe flank pain, radiates to groin, scrotum
- Nausea, vomiting, hematuria may be present.
- Extreme restlessness (patient do not find position of comfort)

Pancreatitis:

- Inflammation of pancreas
- Triggered by ingestion of large amounts of fatty foods.
- Nausea, vomiting; abdominal tenderness; pain radiating from upper abdomen straight through to back.
- Signs, symptoms of hypovolemic shock and peritonitis may be present.

Cholecystitis:

- Inflammation of gall bladder
- Commonly associated with gall stones
- More common in 30 to 50 year old females
- Nausea, vomiting; RUQ pain, tenderness; fever
- Attacks triggered by ingestion of fatty foods.

Serious uncommon condition:

Dissecting Abdominal Aortic Aneurysm:

- Localized weakness of blood vessel wall with dilation .
- Pulsating mass in abdomen
- Can cause lower back pain
- If rupture: shock, exsanguination..

Assessment:

History:

- ***Pain:***

- Nature? ○ Site?
- Onset? ○ Radiation?
- How severe?
- New or experienced before?
- Constant/intermittent/colicky
- Relieving/aggravating factors
- Improving or worsening?
- Pain worsened by movement or coughing?

- ***Associated symptoms:***

- Vomiting (undigested food or bile suggests upper GI pathology or obstruction; faeculent vomiting suggests lower GI obstruction).
- Haematemesis ± melaena,
- Stool/urine colour? Urinary symptoms?
- New lumps?
- Eating and drinking ok?
- Constipation? Flatus?
- Any fainting, dizziness or palpitations?
- Fever/rigors? Rash / itching?
- Recent weight loss?

- **Occupation / country?**
- **Past history / medication:**
 - Previous surgery, laparoscopy?
 - Medical conditions?
 - Full medication?
 - Allergies?
 - When was last meal?
- **Gynecological and obstetric history** (in women)
 - Could she be pregnant?
 - Contraception?
 - LMP, STIs/PID?
 - Previous gynae. Surgery or tubal surgery?
 - IUCD use, previous ectopic pregnancy, vaginal bleeding?

Examination:

- Observe the patient for a few seconds:
 - Looking ill, septic or shocked? (arrange any early needed investigations)
 - Lying perfectly still (think peritonitis) .
 - Rolling around in agony? (Think intestinal, biliary or renal colic). In patients with signs of systemic upset or who appear to be shocked or acutely unwell
- **Further assessment :**
 - Pulse, temperature and blood pressure.
 - Respiratory rate and pattern. (Shallow, rapid breaths suggest peritonitis).
 - If altered consciousness check AVPU scale – **A**lert, **V**oice response, **P**ain response, **U**nconscious.

- Anaemia?
- Visible peristalsis or abdominal distension?
- Signs of bruising around the umbilicus (Cullen's sign – associated with haemorrhagic pancreatitis and ectopic pregnancy) or flanks (Grey Turner's sign associated with retroperitoneal haematoma).
- Supraclavicular and groin lymph nodes.
- Skin turgor /dry mucous membranes.
- Absent bowel sounds suggest paralytic ileus, generalised peritonitis or absolute intestinal obstruction.
- High-pitched and tinkling bowel sounds suggest sub-acute intestinal obstruction.
- Abdominal and iliac bruits suspect aortic aneurysm.
- Percuss the abdomen to assess whether swelling might be due to bowel gas or ascites.
- Tenderness to percussion are likely to have generalised peritonitis.
- Palpate the abdomen gently at first, then more deeply, starting away from the pain and moving towards it.
- Feel for masses, tenderness, rebound tenderness, guarding, organomegaly and herniae.
Always examine the scrotum in men as pain may be referred from unrecognised testicular pathology.
- Rectal or pelvic examination.

- Check lower limb pulses if there could be an abdominal aortic aneurysm.
- Examine any other system that might be relevant, eg chest, cardiac.

Investigation:

(non-specific and must be interpreted in concert with the clinical context).

- Blood tests: FBC, U&E, LFT, amylase/lipase, glucose, clotting, and occasionally Ca^{2+} , ABG (pancreatitis)
- Group and Save or crossmatch
- Blood cultures
- Urinalysis and culture if appropriate
- Pregnancy test in a woman of child-bearing age.
- Radiology - AXR (supine), CXR (erect), IVP, CT, US scan.
- Consider ECG if >40yrs
- Peritoneal lavage following trauma if doubtful.

Signs suspecting serious pathology:

- Signs of shock
 - Confusion / impaired consciousness
 - HR more than 100/min.
 - Hypotension
- Systemically unwell / septic-looking
- Signs of dehydration
- Rigid abdomen
- Patient lying very still .
- Absent or altered bowel sounds
- Associated testicular pathology
- Rebound tenderness or involuntary guarding.

- Tenderness to percussion
- Haematemesis / melaena .
- Suspicion of medical cause for abdominal pain

Emergency department care:

- Protect / maintain airway and give oxygen
- NPO and IV fluids, and Consider passing an NG tube if severe vomiting, signs of intestinal obstruction or extremely unwell and danger of aspiration .
- Send blood for group and save/crossmatch
- Antiemetic / Analgesia if needed after full assessment .
- Antibiotics if suspect systemic sepsis, peritonitis, severe UTI. Use IV cephalosporin ± metronidazole in acutely unwell patients
- Arrange urgent surgical consultation.
- **Admit:**
 - Any patient with Signs suspecting serious pathology.
 - If surgery is likely.
 - If the situation has a chance of deteriorating.
 - Severe persistent diarrhoea.
 - If unable to tolerate oral fluids.
 - Co-morbidity such as diabetes or ischaemic heart disease.
 - For pain control or IV antibiotics required.
 - If medical cause is possible.
 - Patients who have no support at home or live alone.

Mechanical intestinal obstruction

The mechanical type of Intestinal Obstruction is encountered during Hajj more than the functional (paralytic ileus).

Causes of small intestinal obstruction:

During Hajj the vast majority of cases are due to strangulated hernia followed by mesenteric vascular occlusion.

The causes can be listed in the following sequence:

- 1- Strangulated hernia.
- 2- Mesenteric vascular occlusion.
- 3- Adhesions.
- 4- Cancer caecum
- 5- Others.

Causes of large intestinal obstruction:

During Hajj, constipation by inspissated feces may be more encountered followed by pseudo obstruction.

The causes can be listed in the following sequence:

- 1- Inspissated stools at rectum.
- 2- Pseudo-intestinal obstruction.
- 3- Colon cancer.
- 4- Diverticulitis.
- 5- Volvulus.

Diagnosis:

- **History:**

- Abdominal distention.
- Colicky abdominal pain.
- Vomiting (More in small intestinal obstruction).
- Absolute constipation (More in large intestinal obstruction).

- **Examination:**

- Abdominal distention.
- High pitched bowel sounds.
- Absent bowel sound in late stages.

- **Investigation:**

- Plain X-ray (erect & supine) will show multiple gas fluid levels and decide whether it is small or large bowel obstruction.

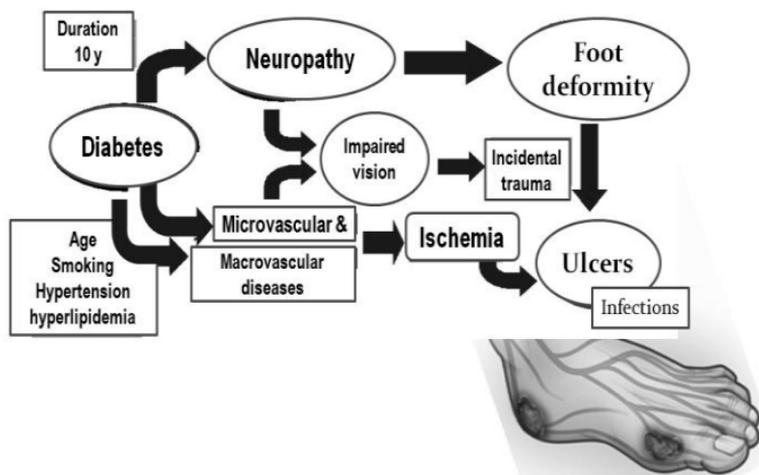
- **Treatment:**

- NPO.
- IV fluids.
- NG tube.
- For small intestinal obstruction, early surgical management is highly recommended during Hajj season. Since that the vast majority of cases are due to strangulated hernia followed by mesenteric vascular occlusion.
- For large intestinal obstruction, conservative approach is recommended During Hajj because constipation by inspissated feces may be more encountered followed by pseudo obstruction.
- Early surgical consultation.

Diabetic foot

Causative factors:

Sandals are part of the pilgrimage dress, which in hot dry weather render the exposed feet to sustain skin trauma especially in areas of crowd.



Some pilgrimages might walk with per feet over the sand or rough hot areas that usually lead to Skin Injury and burn.

Sweating between the toes and Feet being washed five times a day make them wet between the toes that invite fungal infection.

Clinical Presentation:

History:

Patients may or may not have a history of trauma or previous infection.

- Symptoms of peripheral neuropathy include the following:
 - Hyperesthesia, Paresthesia, Dysesthesia, Radicular pain, Anhydrosis.
- Symptoms of Peripheral arterial insufficiency:
 - Most people asymptomatic.
 - Discomfort, cramping, or weakness in the calves or feet, intermittent claudication, ischemic pain at rest, non-healing ulceration of the foot, or frank ischemia of the foot, gangrene.

Physical examination:

- Comprehensive examination of the entire patient (Diabetes is a systemic disease).
- Extremity examination:
 - Diabetic ulcers tend to occur in weight bearing areas.
 - Hypertrophic calluses, Brittle nails, Hammer toes.
 - Fissures.
- Peripheral arterial insufficiency:
 - Absent or diminished peripheral pulses below a certain level indicate the level of occlusion.
 - Absence of both pedal pulses is a specific indicator of peripheral arterial disease.

- Bruit, skin atrophy, loss of pedal hair growth, cyanosis of the toes, ulceration or ischemic necrosis, and pallor of the involved foot followed by dependent rubor after 1-2 minutes of elevation above heart level.
- Peripheral neuropathy:
 - loss of vibratory and position sense, loss of deep tendon reflexes (especially loss of the ankle jerk), trophic ulceration, foot drop, muscle atrophy, and excessive callous formation, especially overlying pressure points such as the heel.

Investigation:

CBC and (ESR), Serum glucose, Urea & creatinine, Electrolytes, Plain x-ray and Doppler study.

Hospital admission is indicated for:

- Acutely infected ulcers.
- Infected gangrene.
- Penetration of digital infections into the forefoot.
- Septic involvement deep to the plantar fascia.
- Uncontrolled diabetes.

Treatment:

- Control of diabetes medically.
- Treatment of infection: second or third generation cephalosporin, in addition to metronidazole 500 mg 8 hourly.
- Wound care with daily saline dressings.
- Removal of necrotic tissues by Debridement and / or amputation.

INFECTIONS

Life-Threatening Infections Diagnosis and Antimicrobial Therapy Selection

Whenever you are dealing with a patient with suspected life- threatening infection, ask yourself the following questions:

1. Does this patient have sepsis or severe sepsis?

- Sepsis: Systemic manifestations of infection.
- Severe sepsis: Sepsis with organ dysfunction, hypoperfusion, or hypotension
- Septic shock: Sepsis with arterial hypotension, despite fluid resuscitation, with organ dysfunction
- *Hypoperfusion abnormalities are:*
 - Acute alteration of mental status.
 - Oliguria.
 - Metabolic acidosis.

- Coagulation abnormalities.
- *Definition of hypotension:*
 - Systolic BP < 90 or decrease > 40 from baseline with adequate fluid resuscitation

2. What information is needed to determine if the patient has an infection?

History:

- epidemiologic setting (home, nursing home, recent hosp stay),
 - More resistant organisms in long-term care facilities, most resistant in hospitals
 - More device-related infections in long-term care and hospitals
 - More comorbidities and severity of illness in hospitalized patients
- Predisposing conditions :
 - Immunosuppression (chemotherapy, steroids, etc)
 - Invasive procedures
 - Prosthetic devices
 - Age
 - Trauma
 - Chronic diseases: cirrhosis, diabetes, malignancy, alcoholism, HIV, etc.

Clinical examination:

- Systemic signs: fever, hypothermia, AMS, hypotension, tachycardia, tachypnea/dyspnea
- Site-specific signs: cough, dyspnea, rales, rhonchi for pulmonary infection, etc.

Laboratory: organ function, CBC

- Increased WBC count, left shift, neutropenia
- Coagulation abnormalities
- Renal/hepatic dysfunction
- Hyperglycemia, hypoglycemia
- Metabolic acidosis, elevated lactate
 - Microbiologic: stains, cultures
- Gram stain—helpful to stain sputum in the case study for pneumonia
- Blood cultures (ask what volume should be obtained—10-15 mL)
- Other fluid cultures
- Toxin assays—*C. difficile*
 - Radiologic: chest Xray, other imaging

3. What interventions should be instituted?

- Resuscitation and hemodynamic stabilization
- Diagnosis of infection
- Control of infection (early source control): antibiotics, removal of device, drainage of abscess.

4. What is the likely source of infection?

- Hospital-acquired,
- Ventilator-associated pneumonia is most likely.

5. What factors influence the choice of antimicrobial agents for this patient?

- Suspected pathogen and site of infection.
- Penetration of antibiotic into site--limited penetration into CNS, abscess, etc.
- Gram stain results.

- Antimicrobial resistance:
 - Longer hospital or ICU stay
 - Prior resistant organism
 - Prior antimicrobial therapy (esp. broad spectrum)
 - Endemic resistant organisms
 - Ongoing outbreak of resistant organism
 - Chronic dialysis
 - Residence in nursing home, etc.
 - Immunosuppressive therapy
- Comorbid conditions—organ dysfunction (renal function, hepatic function), pregnancy.

6. Ask yourself What antimicrobial agent(s) would be appropriate for the following conditions?

Pneumonia:

Community-acquired (immunocompetent)

- β -lactam + macrolide or fluoroquinolone
- Clindamycin for aspiration
- Community-acquired (immunocompromised)
- Trimethoprim-sulfamethoxazole
- Antifungal agent
- Nosocomial and ventilator-associated
- Cephalosporin (3rd/4th generation), β -lactam/ β -lactamase combination, or carbapenem +
- Fluoroquinolone or amino-glycoside
- Vancomycin or linezolid for *S. aureus*
- 2 antipseudomonal agents if *Pseudomonas* suspected

Meningitis:

- Likely community acquired *S. pneumoniae* or *N. meningitidis* meningitis
- Treat initially with 3rd generation cephalosporin (ceftriaxone, cefotaxime) and vancomycin if penicillin resistant *S. pneumoniae* suspected.
- Change to high dose penicillin G if *N. meningitidis*.
- Immunocompromised patient; consider additional coverage for *Listeria monocytogenes* with ampicillin
- Recent neurosurgical procedure increase risk of *S. aureus* and gram negative rods; cover with high dose vancomycin and 3rd or 4th generation cephalosporin.

Suspected Endocarditis:

- Gram positive cocci (*Staph* and *Strep*) most commonly found in general population; choose bactericidal antibiotic (penicillins, 3rd generation cephalosporins ± aminoglycoside, linezolid, etc)

Catheter-related infection:

- Most likely coagulase negative *Staph* and *S. aureus*; remove catheter:
- Vancomycin for MRSA and coagulase negative *Staph* in immunocompromised patient,
- Nafcillin for MSSA,
- 3rd or 4th generation cephalosporin or quinolone if gram negative suspected,
- Fluconazole for *Candida* or caspofungin for more resistant fungi.

Intra-abdominal infection:

- Need to consider gram negative or positive aerobes and anaerobes;
- Involve surgeon,
- Beta-lactam/beta-lactamase inhibitor
- Or carbapenem as monotherapy,
- Cephalosporins/quinolones with metronidazole

Infection in Pregnancy: alters indicated antibiotics

- (avoid quinolones, etc),
- Need to cover gram negative organisms; 3rd generation cephalosporin (safest in pregnancy),
- Ffluoroquinolones,
- Piperacillin/tazobactam,
- Trimethoprim/sulfamethoxazole

Necrotizing fasciitis with polymicrobial infection

- Involve surgeon.
- Vancomycin + beta-lactam/beta-lactamase inhibitor,
- Carbapenem and fluoroquinolone,
- Aminoglycoside and clindamycin

Immunocompromised, neutropenic patient:

- 3rd or 4th generation cephalosporin and aminoglycoside or fluoroquinolone,
- Carbapenem,
- Piperacillin/tazobactam ,
- Vancomycin if gram positive organism likely

C. difficile colitis:

- Oral metronidazole 250-500 mg TID,
- Vancomycin 125-500 mg QID for 10 days.

Community Food Poising

Food poisoning: Defined by 2 criteria:

- Similar illness, often gastrointestinal, in minimum of 2 people.
- Evidence of food as the source.

Causes of food poisoning:

- CDC estimates that 97% due to improper food handling.
- Most common causes:
 - Leaving prepared food at a temperature that allows bacterial growth.
 - Inadequate cooking or reheating.
 - Cross-contamination.
 - Infection in food handlers.
- Bacterial in 75% of outbreaks.

History:

- Duration of the disease.
- Characteristics / frequency of bowel movements.
- Associated abdominal and systemic symptoms.
- Common source.
- Specific food.
- Travel history.
- Antibiotics use.

Physical examination:

- Dehydration.
- Rose spot macules and hepatosplenomegaly (*Salmonella typhi*).
- Erythema nodosum and exudative pharyngitis (*Yersinia*).
- PR: rectal mucosa & blood in stool.

Workup :

Laboratory Studies:

- Gram stain and Loeffler methylene blue stain of stool for WBCs: indicate invasive disease.
- Microscopic exam of stool for ova and parasites.
- Culture for enteric pathogens if invasive disease or symptoms persist for longer than 3-4 days.
- Blood culture if patient is notably febrile.
- CBC, differentials, electrolyte, urea, creatinine to assess inflammatory response and degree of dehydration.
- Assay for *C difficile* to R/O antibiotic-associated diarrhea.

Imaging Studies:

- Flat and upright abdominal radiographs.

Other Tests:

- Sigmoidoscopy/colonoscopy with biopsy in patients with bloody diarrhea.
- Upper GIT endoscopy with duodenal aspirate and biopsy.

Treatment:

- Main objective is adequate rehydration and electrolyte supplementation, achieved with:
 - Oral rehydration solution (ORS) .
 - Intravenous solutions:
 - Isotonic sodium chloride solution
 - Lactated Ringer solution
- Most cases are self-limited, Specific treatment is not necessary.
- Strict personal hygiene should be practiced during the illness..
- < 10% of cases require antibiotic therapy:
- If symptoms persist beyond 3-4 days, specific etiology have to be determined and empiric treatment (ciprofloxacin 500 mg PO bid) to be given.

Malaria

History:

- Most patients live in or have recently traveled to an endemic area; however, a few cases are reported each year in which the patient had no history of such travel (e.g. airport malaria, from imported mosquitoes). Malaria may present over 1 year after travel to an endemic area. Previously infected patients may develop relapsing malaria, a recurrence of the disease after it has been apparently cured; this form is caused by reactivation of hypnozoites (dormant liver-stage parasites) in *P vivax* and *P ovale* infections.
- Determine the patient's immune status, age, allergies, other medical conditions, other medications, and pregnancy status.
- The patient usually remains asymptomatic for a week or more after the infecting mosquito bite.
- Clinical symptoms include the following: Cough, Fatigue, Malaise, Shaking chills, Arthralgia, Myalgia, Paroxysm of fever, shaking chills, and sweats (every 48 or 72 h, depending on species)
- The classic paroxysm begins with a period of shivering and chills, which lasts for approximately 1-2 hours, and is followed by a high fever. Finally, the patient experiences excessive diaphoresis, and the

body temperature of the patient drops to normal or below normal.

- Many patients, particularly early in infection, do not present the classic paroxysm but may have several small fever spikes a day.
- Less common symptoms include the following: Anorexia and lethargy, Nausea and vomiting, Diarrhea, Headache, and Jaundice.

Physical:

- Physical signs that may be noted with malaria include the following: Tachycardia, Fever, Hypotension, Signs of anemia, Splenomegaly, Icterus.

Causes:

- Malaria most often is caused by the bite of a female Anopheles species mosquito that is infected with species of the protozoan genus Plasmodium. The 5 most common species affecting humans are: *P vivax*, *P ovale*, *P malariae*, *P knowlesi*, and *P falciparum* (The most malignant form of malaria is caused by this species)
- Other less common routes of infection are through blood transfusion and maternal-fetal transmission. When *P vivax* and *P ovale* are transmitted via blood, no latent hypnozoite phase occurs and treatment with primaquine is not necessary, as it is the sporozoites that form hypnozoites in infected hepatocytes.

Laboratory diagnosis:

- CBC, electrolyte panel, renal function tests, pregnancy test, urinalysis, free serum haptoglobin, urine and blood cultures, and thick and thin blood smears. For those patients who may receive quinine or primaquine, a G-6-PD test should be ordered. Lumbar puncture may be indicated in patients who have encephalopathy in which the diagnosis is not clear. Rapid HIV testing may also be indicated in select cases.
- Rapid diagnostic tests (RDTs) examples include Para Sight-F test (Becton Dickinson Advanced Diagnostics), ICT Malaria P.f/P.v (Binax Inc), OptiMAL pLDH (DiaMed USA, LLC), Kat-Quick (Katmedical CC), and Rapimal MT Pf Dipstick (Cellabs Pty Ltd) (dipstick tests). Note: This list is not all-inclusive; research individual tests for comparative efficacy and cost.

Emergency Department Care:

- Assess airway, breathing, and circulation; intervene as necessary. If evidence of life-threatening hemolytic anemia is determined, establish large-bore intravenous (IV) lines, initiate fluid resuscitation, and administer transfusion of type-specific packed RBCs.
- Hyponatremia likely reflects continued oral hypotonic fluid intake in the setting of hypovolemia and requires no therapy beyond rehydration. Overly aggressive treatment of hyponatremia may lead to death.

- Consider exchange transfusion for life-threatening complications.
- Monitor and treat hypoglycemia, as needed.
- A reliable, semi-immune, adult patient with a *P vivax*, *P ovale*, or *P malariae* infection may be treated on an outpatient basis. However, special care must be taken if *P malariae* is diagnosed solely on the basis of a blood smear, as it may be confused with the sometimes fatal *P knowlesi*, an infection that would require inpatient treatment. Those treated as outpatients should have adequate follow-up care, including daily blood smears to confirm that the treatment is effective in decreasing parasitemia.
- General hospital admission guidelines are as follows: Patients with suspected or confirmed *P falciparum* or *P knowlesi* infection, Children, Pregnant women, Immunodeficient individuals
- Intensive care unit admission guidelines are as follows: Immediate life-threatening complications present, such as coagulopathy or end-organ failure, Presence of signs and symptoms consistent with cerebral malaria (eg, altered mental status, repeated seizures, coma), Patients who are nonimmune with a *falciparum* parasitemia greater than 2% or who are semi-immune with a *P falciparum* parasitemia greater than 5%, Presence of any other severe malarial complications
- If the infection is caused by an unidentified species or by mixed species, treat it as if it were caused by *P*

falciparum. In the absence of known drug sensitivities, assume that the Plasmodium species in question is chloroquine resistant. If Southeast Asia is the origin of the infection, then assume mefloquine resistance.

- If a patient is diagnosed with *P. falciparum* malaria with a parasitemia greater than 10% or if the patient is experiencing life-threatening complications (ie, coma, respiratory failure, coagulopathy, fulminant kidney failure), then investigate exchange transfusion as a treatment option. If transfusion is undertaken, it should continue until the parasitemia falls below 5%, although the mortality benefit of this intervention has not been proven.

Medications:

Treatment of uncomplicated *P. falciparum* malaria during Hajj (symptomatic malaria without signs of severity or evidence (clinical or laboratory) of vital organ dysfunction):

- Non pregnant adult: Artesunate (AS) plus sulfadoxine-pyrimethamine (SP) *Artecospa*® tab: on day one give 3 tab of SP + 2 tab of AS, on day two and three, give 2 tab of AS each day.
- Pregnancy:
 - First trimester, Quinine plus clindamycin for 7days
 - Second trimester, give *Artecospa*® as non-pregnant adult above.

Treatment of severe *P. falciparum* malaria during Hajj (adult and children):

- Non-pregnant adult: Artesunate 2.4 mg/kg IV or IM at 0, 12h and 24 h, then once daily (See pamphlet for mixing instructions). Note: solution must be used immediately and should not be stored.
- Pregnancy:
 - First trimester, Quinine plus clindamycin for 7 days
 - Second and third trimester, Artesunate as above
- Quinine IV under ICU monitoring is an alternative for both pregnant and non-pregnant.
- IV medication must be given for at least 24h even if able to swallow, then oral *Artecospa*® can be used to complete treatment.

Uncomplicated malaria, *P. vivax* (except Papua New Guinea and Indonesia) or *P. ovale*:

- Chloroquine phosphate 600 mg base (=1,000 mg salt) po immediately, followed by 300mg base (=500 mg salt) po at 6, 24, and 48 hours. Total dose: 1,500 mg base (=2,500 mg salt) PLUS Primaquine phosphate: 30 mg base po qd x 14 days.

Meningitis

Meningitis is a clinical syndrome characterized by inflammation of the meninges

Etiologic Classification:

- Acute bacterial meningitis denotes an acute onset of meningeal symptoms and neutrophilic pleocytosis.
- Fungal and parasitic causes of meningitis are termed according to their specific etiologic agent, such as cryptococcal, *Histoplasma* meningitis, and amebic meningoencephalitis.
- Aseptic meningitis is a broad term that denotes a non-pyogenic cellular response, which may be caused by many different etiologic agents:
 - In many cases, a cause is not apparent after initial evaluation. Patients characteristically have an acute onset of meningeal symptoms, fever, and cerebrospinal pleocytosis that is usually prominently lymphocytic.
 - While viruses cause most cases of aseptic meningitis, it can also be caused by bacterial, fungal, mycobacterial, and parasitic agents.

The mortality:

- The mortality rate for viral meningitis (without encephalitis) is less than 1%.

- Bacterial meningitis was uniformly fatal before the antimicrobial era. With the advent of antimicrobial therapy, the overall mortality rate from bacterial meningitis has decreased but remains alarmingly high (25%).
- The reported mortality rates for specific organism are:
 - 19-26% for *S pneumoniae* meningitis.
 - 3-6% for *H influenzae* meningitis.
 - 3-13% for *N meningitidis* meningitis.
 - 15-29% for *L monocytogenes* meningitis.

History

- The classic presentation of meningitis includes fever, headache, neck stiffness, photophobia, nausea, vomiting, and signs of cerebral dysfunction (e.g., lethargy, confusion and coma).
- The triad of fever, nuchal rigidity, and change in mental status is found in only two thirds of patients. Fever is the most common manifestation (95%), while stiff neck and headache are less common. However, the negative predictive value of these symptoms is high (i.e., the absence of fever, neck stiffness, or altered mental status eliminates the diagnosis of meningitis in 99-100% of cases).
- The classic presentation of acute meningitis is the onset of symptoms within hours to a few days, compared to weeks for chronic meningitis.
- Atypical presentation may be observed in certain groups. Elderly individuals, especially those with

underlying comorbidities and other immunocompromised hosts (e.g., diabetes, renal and liver disease, AIDS), may present with lethargy and an absence of meningeal symptoms.

- Clues in the patient's clinical history may suggest the specific etiologic agent. Detailed epidemiologic and predisposing risks should be assessed.
 - The time of the year is an important variable because many infections are seasonal. Enteroviruses are observed worldwide, and infections occur during late summer and early fall in temperate climates and year-round in tropical regions. In contrast, mumps, measles, and varicella zoster viruses occur more commonly during winter and spring seasons. Arthropod-borne viruses occur during the warmer months.
 - History of exposure to a patient with a similar illness is an important epidemiological clue when determining etiology (eg, individuals who were in close contact with an index case of meningococemia).
 - Elicit a history of sexual contact and high-risk behavior. HSV meningitis is associated with primary genital HSV infection and HIV infection.
 - The intake of unpasteurized milk and cheese predisposes to brucellosis and *L monocytogenes* infection.
 - Animal contacts should be elicited. Patients with rabies could present atypically with aseptic

meningitis, and rabies should be suspected in a patient with a history of animal bite (eg, dog, fox, bat). Exposure to rodents suggests infection with lymphocytic choriomeningitis (LCM) virus and *Leptospira* infection. Laboratory workers dealing with these animals also are at increased risk of contracting LCM.

- Record evidence of systemic viral infection (ie, myalgias, fatigue, anorexia).
 - The presence of exanthemas; symptoms of pericarditis, myocarditis, or conjunctivitis; or syndromes of pleurodynia, herpangina, and hand-foot-and-mouth disease suggest enterovirus infection.
 - A history of recurrent bouts of benign aseptic meningitis suggests Mollaret syndrome, which is caused by HSV.
- The presence of a ventriculoperitoneal shunt and a history of recent cranial surgery should be elicited.
- The presence of cochlear implants with a positioner has been associated with a higher risk of bacterial meningitis.

Physical examination:

- Signs of cerebral dysfunction are common, including confusion, irritability, delirium, and coma. These are usually accompanied by fever and photophobia.
- Signs of meningeal irritation are observed in only approximately 50% of patients with bacterial

meningitis, and their absence certainly does not rule out meningitis.

- Kernig sign: In a supine patient, flex the hip to 90° while the knee is flexed at 90°. An attempt to further extend the knee produces pain in the hamstrings and resistance to further extension.
- Brudzinski sign: Passively flex the neck while the patient is in a supine position with extremities extended. This maneuver produces flexion of the hips in patients with meningeal irritation.
- Nuchal rigidity: Resistance to passive flexion of the neck is also a sign.
- Exacerbation of existing headache by repeated horizontal movement of the head, at a rate of 2-3 times per second, may also suggest meningeal irritation.
- Cranial nerve palsies may be observed as a result of increased ICP or the presence of exudates encasing the nerve roots.
- Focal neurologic signs may develop as a result of ischemia from vascular inflammation and thrombosis.
- Seizures occur in approximately 30% of patients.
- Papilledema and other signs of increased ICP :
 - Coma, increased blood pressure with bradycardia, and cranial nerve III palsy may be present.
 - The presence of papilledema also suggests a possible alternate diagnosis (e.g., brain abscess).

- Systemic findings upon physical examination may provide clues to the etiology.
 - Morbilliform rash with pharyngitis and adenopathy may suggest a viral etiology
 - Macules and petechiae that rapidly evolve into purpura suggest meningococemia (with or without meningitis).
 - Vesicular lesions in a dermatomal distribution suggest varicella-zoster virus. Genital vesicles suggest HSV-2 meningitis.
 - Sinusitis or otitis suggests direct extension into the meninges, usually with *S pneumoniae* and *H influenzae*. Rhinorrhea or otorrhea suggests a CSF leak from a basilar skull fracture, with meningitis most commonly caused by *S pneumoniae*.
 - The presence of a murmur suggests infective endocarditis with secondary bacterial seeding of the meninges.
 - Evidence of parotitis is observed in some cases of mumps meningitis.
 - The presence of a ventriculoperitoneal shunt or a cochlear implant may suggest a bacterial meningitis.
- In contrast to bacterial meningitis, patients with aseptic meningitis syndrome usually appear clinically nontoxic with no vascular instability.

Laboratory investigation:

- Whenever the diagnosis of meningitis is strongly considered, promptly perform a lumbar puncture. Measure the opening pressure and send the fluid for cell count (and differential count), chemistry (i.e., CSF glucose and protein), and microbiology (i.e., Gram stain and cultures). CT scan of the brain may be performed prior to lumbar puncture in patient with a higher risk of herniation. Including those with newly onset seizures, an immunocompromised state, signs suspicious for space-occupying lesions (such as papilledema and focal neurologic signs), and moderate-to-severe impairment in consciousness.
- Special studies, such as serology and nucleic acid amplification, may also be performed depending on clinical suspicion.
- There is increasing data to suggest that serum procalcitonin levels can be used as a guide to distinguish between bacterial and aseptic meningitis in children. The results yielded by a serum procalcitonin, combined with other findings, could be helpful in making clinical decisions.

Treatment:

Bacterial meningitis is a neurological emergency and initiation of empiric antibacterial therapy is essential for better outcome. Institute empiric antimicrobial therapy as soon as possible. This is usually based on the known

predisposing factors and/or initial CSF Gram-stain results.

Recommended Empiric Antibiotics According to Predisposing Factors for Patients With Suspected Bacterial Meningitis:

Predisposing Feature	Antibiotic(s)
Age 0-4 weeks	Ampicillin plus cefotaxime or an aminoglycoside
Age 1-3 months	Ampicillin plus cefotaxime plus vancomycin
Age 3 months to 50 years	Ceftriaxone or cefotaxime plus vancomycin
Older than 50 years	Ampicillin plus ceftriaxone or cefotaxime plus vancomycin
Impaired cellular immunity	Ampicillin plus ceftazidime plus vancomycin
Neurosurgery, head trauma, or CSF shunt	Vancomycin plus ceftazidime

Recommended Empiric Antibiotics for Patients With Suspected Bacterial Meningitis and Known CSF Gram Stain Result:

Gram Stain Morphology	Antibiotic(s)
Gram-positive cocci	Vancomycin plus ceftriaxone or cefotaxime
Gram-negative cocci	Penicillin G
Gram-positive bacilli	Ampicillin plus an aminoglycoside
Gram-negative bacilli	Broad-spectrum cephalosporin plus an aminoglycoside

Pneumonia in hajj

Community-acquired pneumonia

Community-acquired pneumonia develops in people with limited or no contact with medical institutions or settings.

The most commonly identified pathogens:

Streptococcus pneumoniae, Haemophilus influenzae, and atypical organisms (ie, Chlamydia pneumoniae, Legionella sp and Mycoplasma pneumoniae).

Clinical presentation:

Symptoms:

- Malaise.
- Cough typically is productive in older children and adults and dry in infants, young children, and the elderly.
- Dyspnea usually is mild and exertional and is rarely present at rest.
- Chest pain is pleuritic and is adjacent to the infected area.
- Pneumonia may manifest as upper abdominal pain when lower lobe infection irritates the diaphragm.
- Symptoms become variable at the extremes of age; infection in infants may manifest as nonspecific

irritability and restlessness; in the elderly, as confusion and obtundation.

Signs:

- Fever, tachypnea, tachycardia, crackles, bronchial breath sounds, egophony, and dullness to percussion.
- Signs of pleural effusion may also be present.
- Nasal flaring
- Use of accessory muscles.
- Cyanosis is common in infants.
- Fever is frequently absent in the elderly.

No single symptom or sign is sensitive or specific enough to predict the organism. Symptoms are even similar for noninfective lung diseases such as pulmonary embolism, pulmonary malignancy, and other inflammatory lung diseases.

Diagnosis:

- Chest x-ray almost always demonstrates some degree of infiltrate; rarely, an infiltrate is absent in the first 24 to 48 h of illness. In general, no specific findings distinguish one type of infection from another, although multilobar infiltrates suggest *S. pneumoniae* or *Legionella pneumophila* infection and interstitial pneumonia suggests viral or mycoplasmal etiology.
- WBC count and electrolytes, BUN, and creatinine testing to classify risk and hydration status.

- Two sets of blood cultures are often obtained to detect pneumococcal bacteremia and sepsis, because about 12% of all patients hospitalized with pneumonia have bacteremia; *S. pneumoniae* accounts for 2/3 of these cases.
- Pulse oximetry or ABG should also be done.

Probability of Pneumonia Given Chest X-ray Infiltrate:

Assign 1 point each for:

- Temperature > 37.8°C
- Heart rate > 100 beats/min
- Crackles on auscultation
- Decreased breath sounds
- Absence of asthma

Score	Likelihood Ratio	Probability of Pneumonia
0–1	0.3	≤ 1%
2–3	—	3–10%
4–5	8.2	25–50%

Treatment:

- **Antibiotics:**
Suggested antibiotics (as soon as possible):
 - Outpatient, with no underlying disease:
 - Azithromycin 500 mg PO once daily for 5 days.

- Hospitalized patients:
 - Cefuroxime 750mg IV Q 8 h for 7 days &
 - Azithromycin 500mg PO once daily for 5 days.
 - OR Clarithromycin 500 mg PO twice daily for 10 days.
 - OR Erythromycin 500 mg i.v q 6-8 hours.
- Severely ill patients hospitalized in ICU:
 - Piperacilline/Tazobactam (Tazocin) 4.5 gm i.v. 8 hourly.
 - OR Ceftriaxone 2 gm q24 hours and Gentamicin. & Erythromycin 1gm i.v. slowly eight hourly.
- Strongly consider tuberculosis for non-responders and arrange for bronchoscopy.
- Switch to oral therapy if :
 - Fever resolved for 24-48 hours.
 - Cough improved.
 - WBC decreased.
 - Patient able to eat.
- Antivirals for influenza or varicella.
- Admission:
 - Hypoxemia an absolute indication for admission.
 - ICU admission is required for:
 - Patients who need mechanical ventilation.
 - For those with hypotension (systolic BP < 90 mm Hg) that is unresponsive to volume resuscitation.
 - Respiratory rate > 30/min.

- Multilobar pneumonia, diastolic BP < 60 mm Hg, confusion, and BUN > 19.6 mg/dL.
- Supportive measures: includes fluids, antipyretics, analgesics, and for patients with hypoxemia O₂.

Prognosis: is excellent for relatively young or healthy patients, but many pneumonias, especially when caused by *S. pneumoniae* or influenza virus, are fatal in older, sicker patients.

Novel influenza A(H1N1) virus infection

Suspected case :

Any person who has the following symptoms at point of first contact with the hospital.

ILI (Influenza Like Illness), running or stuffy nose, headache, \geq fever 38 °C or more, chills, sore throat, cough, body aches, fatigue, vomiting and diarrhea and/or alarming/ warning signs :

- Difficulty in or shortness of breath.
- Pain or pressure in chest/abdomen.
- Sudden dizziness.
- Hypotension.
- Altered level of consciousness.
- Confusion.
- Hemoptysis.
- ILI symptoms improved but have returned with fever and worse cough.
- Signs and symptoms of pneumonia .
- Fever more than 3 days and not responding to treatment.
- Bluish or gray skin color.
- History of contact with a confirmed / probable or suspected case within last 7 days.

Confirmed case by investigations:

A case with above mentioned symptoms/signs and laboratory investigations positive for A(H1N1).

Incubation period: range from 1 – 7 days.

Infectious period is estimated to be one day prior to and 7 days after the beginning of symptoms. In case of children, infectiousness extends longer up to 10 days.

Transmission : The limited available data on transmission of A(H1N1) virus, indicates transmission as follows:

Person to person transmission primarily through a large particle respiratory droplet (cough, sneezes of infected person and near to susceptible person) It requires close contact < 6 feet.

Contact with contaminated surfaces.

Close contact:

Close contact is defined as having cared for or lived with a person who is confirmed, probable or suspected case of A(H1N1) influenza.

Or having been in a setting where there was a high likelihood of contact with respiratory droplets and/or body fluids of above mentioned.

Examples of close contact include kissing or embracing. Sharing eating or drinking utensils, physical examination.

Or any other contact between persons likely to result in exposure to respiratory droplets.

Antiviral treatment for A(H1N1) influenza:

Treatment with Oseltamivir (Tami flu) 75mg bid for 5days is recommended for:

- All patients with confirmed, probable or suspected novel A(H1N1) Influenza.
- Patients who are at high risk.

Duration for antiviral chemoprophylaxis post – exposure is 10 days after the last known exposure to a confirmed novel A(H1N1) influenza.

Indications for post exposure chemoprophylaxis is upon close contact with a person who is a confirmed, probable or suspected case of A(H1N1) influenza virus begin one day before they develop symptoms to up to 7 days after they get sick.

General guidelines for prevention / protection from A(H1N1)

- Wear mask in crowded area.
- During early signs and symptoms and to avoid spreading the disease to others, a distance of one meter or more should be kept when in contact with others.
- During sneezing and coughing, the nose and mouth should be covered with a tissue paper and disposed off in a covered plastic bag.
- Wash your hands with soap and water many times daily.
- Wash solid items in contact many times daily.

Middle East respiratory syndrome coronavirus (MERS-CoV)

Case definition and surveillance guidance:
Suspect case (patients who should be tested
for MERS-CoV) :

I. A person with fever and community-acquired pneumonia or acute respiratory distress syndrome based on clinical or radiological evidence.

OR

II. A hospitalized patient with healthcare associated pneumonia based on clinical and radiological evidence.

OR

III. A person with:

- 1) acute febrile ($\geq 38^{\circ}\text{C}$) illness, **AND**
- 2) body aches, headache, diarrhea, or nausea/vomiting, with or without respiratory symptoms, **AND**
- 3) unexplained leucopenia ($\text{WBC} < 3.5 \times 10^9/\text{L}$) and thrombocytopenia ($\text{platelets} < 150 \times 10^9/\text{L}$).

OR

IV. A person (including health care workers) who had protected or unprotected exposure to a confirmed or probable case of MERS-CoV infection and who presents with upper or lower respiratory illness within 2 weeks after exposure.

All suspected cases should have nasopharyngeal swabs, and, when intubated, lower respiratory secretions samples collected for MERS-CoV testing.

Patients who meet the criteria for category I or II above should also be evaluated for common causes of community-acquired pneumonia (such as influenza A and B, respiratory syncytial virus, *Streptococcus pneumoniae*, *Hemophilus influenzae*, *Staphylococcus aureus*, and *Legionella pneumophila*). This evaluation should be based on clinical presentation and epidemiologic and surveillance information. Testing for MERS-CoV and other respiratory pathogens can be done.

Probable case:

A probable case is a patient in category I or II above with absent or inconclusive laboratory results for MERS-CoV and other possible pathogens who is a close contact of a laboratory-confirmed MERS-CoV case or who works in a hospital where MERS-CoV cases are cared for.

Confirmed case :

A confirmed case is a suspect case who had Rhinorrhea, sore throat, and/or cough , Shortness of

breath, hypoxemia, or pneumonic infiltration evident on chest x-ray. with laboratory confirmation of MERS-CoV infection.

simultaneously. Positive results for another respiratory pathogen (e.g H1N1 influenza) should not necessarily preclude testing for MERS-CoV because coinfection can occur.

Laboratory tests to exclude other causes of this clinical presentation (e.g., dengue, Alkhumra hemorrhagic fever virus, CMV, EBV, typhoid fever, and malaria) should be simultaneously performed if clinically and epidemiologically indicated.

Confirmatory laboratory testing requires a positive PCR on at least two specific genomic targets (upE and ORF1a) OR a single positive target (upE) with sequencing of a second target (RdRpSeq or NSeq). It is strongly advised that lower respiratory specimens such as sputum, endotracheal aspirate, or bronchoalveolar lavage should be used when possible. If patients do not have signs or symptoms of lower respiratory tract infection or lower tract specimens are not possible or clinically indicated, both nasopharyngeal and oropharyngeal specimens should be collected and combined in a single collection container and tested together. If initial testing of a nasopharyngeal swab is negative in a patient who is strongly suspected to have MERS-CoV infection, patients should be retested using a lower respiratory specimen or, if not possible, a repeat nasopharyngeal and oropharyngeal specimen. For

patients in whom adequate lower respiratory samples are not possible, investigators may also want to consider other types of auxiliary testing such as nasopharyngeal wash for MERS-CoV PCR and paired acute and convalescent sera for serological tests. Collection of additional specimens such as stool, urine, and serum for MERS-CoV PCR is also recommended as the virus has also been demonstrated in these body fluids.

Admission criteria:

Patients suspected to have MERS-CoV infection who have shortness of breath, hypoxemia, and/or clinical or radiological evidence of pneumonia should be hospitalized.

Management of “Patient under investigation” for MERS-CoV infection:

Supplemental oxygen therapy:

Give oxygen therapy to patients with signs of severe respiratory distress, hypoxaemia (i.e. SpO₂ < 90%) or shock. Initiate oxygen therapy at 5 L/min and titrate to SpO₂ ≥ 90%. Pulse oximeters, functioning oxygen systems and appropriate oxygen-delivering interfaces in addition to noninvasive and invasive mechanical ventilation should be available in all areas where patients with syndrome of acute respiratory infection are cared for.

Collect respiratory and other specimens for laboratory testing:

Collect routine clinical specimens (e.g. blood and sputum bacterial cultures) for community-acquired pneumonia, ideally before antimicrobial use. Also collect respiratory specimens from the upper respiratory tract (i.e. nasal, nasopharyngeal and/or throat swab) and lower respiratory tract (i.e. sputum, endotracheal aspirate, bronchoalveolar lavage) for known respiratory viruses (such as influenza A and B, influenza A virus subtypes H1, H3, and H5 in countries with H5N1 viruses).

Empiric antimicrobials to treat suspected pathogens, including community-acquired pathogens:

Although the patient may be suspected to have novel coronavirus infection, administer appropriate empiric antimicrobials as soon as possible for community-acquired pathogens based on local epidemiology and guidance until the diagnosis is confirmed. Empiric therapy can then be adjusted on the basis of laboratory testing results.

Use conservative fluid management when there is no evidence of shock:

Aggressive fluid resuscitation may worsen oxygenation, especially in settings where there is limited availability of mechanical ventilation.

Do not give high-dose systemic corticosteroids or other adjunctive therapies for viral pneumonitis outside the context of clinical trials:

Prolonged use of systemic high-dose corticosteroids can result in serious adverse, including opportunistic infection, avascular necrosis, new health-care-associated bacterial infection and possibly prolonged viral replication. Therefore, corticosteroids should be avoided unless they are indicated for another reason

Closely monitor patients for signs of clinical deterioration, such as severe respiratory distress / respiratory failure or tissue hypoperfusion/shock, and apply supportive care interventions.

Recognize severe cases:

- Even when high oxygen flows (10 to 15 L/min) are delivered through a face mask with reservoir bag, and the concentration of oxygen (FiO₂) is high (between 0.60 and 0.95); patients may continue to have increased work of breathing or hypoxemia because of high intrapulmonary shunt fractions and require mechanical ventilation.
- The type of mechanical ventilation will be determined by the general condition of the patient and the availability and experience with non-invasive ventilation (NIV) (administration of ventilatory support through a mask) or invasive mechanical ventilation administered through an endotracheal tube or tracheostomy tube.

- If NIV is tried, monitor the patient closely in an ICU; if NIV is unsuccessful, do not delay endotracheal intubation.

Use a lung-protective ventilation strategy (LPV) for patients with ARDS:

- Implementing a low-volume, low-pressure ventilation strategy/protocol. To reach LPV targets, allow permissive hypercapnia.
- To reach target SpO₂, use adequate PEEP for the degree of hypoxemia.
- Deep-sedation targets should be considered if unable to control tidal volume.
- Avoid disconnecting the patient from the ventilator. Disconnection results in loss of PEEP and lung collapse. Use in-line catheters for airway suctioning, clamp tube when disconnection is required and minimize transport.

In patients with severe ARDS, consider adjunctive therapeutics early, especially if failing to reach LPV targets:

- Administration of neuromuscular blockade for initial 48 hours has been associated with improved survival and increased time off the ventilator without causing significant weakness .
- Placing the patient in the prone position improves oxygenation and survival but care must be taken to turn the patient safely .

- Delivering a recruitment maneuver and high PEEP improves oxygenation and reduces need for other rescue therapies .
- In case of failure of all previous measures to reach target ventilation and oxygenation ,the use of extracorporeal membrane oxygenator " ECMO" is highly recommended.

Prevent complications associated with critical illness such as: Ventilator-associated pneumonia, Venous thromboembolism, Catheter-related bloodstream infection, Pressure ulcers. stress ulcers and gastric bleeding and ICU-related weakness.

General infection prevention and control precautions:

Standard Precautions:

Standard Precautions, a cornerstone for providing safe health care and reducing the risk of further infection, should always be applied in all health-care settings for all patients.

Standard Precautions include:

- Hand hygiene; includes either washing hands with antiseptic soap and water or the use of an alcohol-based waterless hand sanitizer (waterless hand rub), before touching a patient, before any clean or aseptic procedure, after body fluid exposure, after touching a patient, and after touching a patient's surroundings, including contaminated items or surfaces.

- Use of personal protective equipment (PPE) to avoid direct contact with patients' blood, body fluids, secretions (including respiratory secretions) and non-intact skin.
- Respiratory Hygiene and Cough Etiquette.
- Prevention of overcrowding in waiting and clinical areas is essential to prevent cross infection.
- Environmental ventilation in all areas within a health-care facility.
- Environmental cleaning.
- Prevention of needle-stick or sharps injury.
- Safe waste management.
- Follow standard procedures, per hospital policy and manufacturers' instructions, for cleaning and/or disinfection of surfaces, equipment, Textiles and Food utensils.
- Ensure that cleaning and disinfection procedures are followed consistently and correctly.

Infection prevention and control precautions when caring for patients with suspected, probable, or confirmed MERS-CoV infection:

- For patients with suspected, probable, or confirmed MERS-CoV infection who are not critically ill, standard, contact, and droplet precautions are recommended for management.

- For patients who are critically ill (e.g. pneumonia with respiratory distress or hypoxemia), standard, contact, and airborne precautions are recommended due to the high likelihood of requiring aerosol-generating procedures.
- Standard, contact, and airborne precautions should be used for all (critically or non-critically ill) patients when anticipating or performing aerosolgenerating procedures which may be associated with an increased risk of infection transmission (including both elective procedures such as bronchoscopy, sputum induction, elective intubation and extubation, and emergency procedures such as cardiopulmonary resuscitation, initiation of Bilevel Positive Airway Pressure-BIPAP, emergency intubation, open suctioning of airways, manual ventilation via umbo bagging through a mask before intubation).
- Selected Components of Recommended Precautions for Prevention of MERS-CoV Transmission:
 - Place patients with suspected, probable, or confirmed MERS-CoV infection who are not critically ill in single patient rooms in an area that is clearly segregated from other patient-care areas.
 - Place patients with suspected, probable, or confirmed MERS-CoV infection who are critically

ill (e.g. pneumonia with respiratory distress or hypoxemia) in Airborne Infection Isolation rooms (Negative Pressure Rooms) due to the high likelihood of requiring aerosol-generating procedures.

- When negative pressure rooms are not available, place the patients in adequately ventilated single rooms. When available, a portable HEPA filter, turned on to the maximum power, should be placed at the head side of the patient's bed.
- When single rooms are not available, place patients with the same diagnosis together (cohorting). If this is not possible, place patient beds at least 1 m apart.
- Avoid the movement and transport of patients out of the isolation room or area unless medically necessary. The use of designated portable X-ray, ultrasound, echocardiogram, and other important diagnostic machines is recommended when possible.
- If transport is required:
 - Patients should wear a medical mask to contain secretions
 - Use routes of transport that minimize exposures of staff, other patients, and visitors.
 - Notify the receiving area of the patient's diagnosis and necessary precautions as soon as possible before the patient's arrival.

- Ensure that healthcare workers (HCWs) who are transporting patients wear appropriate PPE and perform hand hygiene afterwards.
- Personal Protective Equipment (PPE) for Healthcare Workers (HCWs)
- The following PPE should be worn by HCWs upon entry into patient rooms or care areas:
 - Gowns (clean, non-sterile, long-sleeved disposable gown)
 - Gloves
 - Eye protection (goggles or face shield) & A medical mask.
 - For patients under airborne precautions, all persons entering the patient's room should wear a fit-tested, seal checked N95 mask instead of a medical mask. For those who failed the fit testing of N95 masks (e.g those with beards), an alternative respirator, such as a powered air-purifying respirator, should be used.
 - Upon exit from the patient room or care area, PPE should be removed and discarded, Except for N95 masks, remove PPE at doorway or in anteroom. Remove N95 mask after leaving patient room and closing door.
 - Remove PPE in the following sequence:
 1. gloves, 2. Goggles or face shield, 3. Gown, and 4. Mask.

- Never wear a medical mask under the N95 mask as this prevents proper fitting and sealing of the N95 mask thus decreasing its efficacy.
- For female staff who wear veils, the medical or N95 mask should always be placed directly on the face behind the veil and not over the veil. In this instance, a face-shield should also be used along with the mask to protect the veil from droplet sprays.
- Perform hand hygiene before and after contact with the patient or his/her surroundings and immediately after removal of PPE.
- If possible, use either disposable equipment or dedicated equipment (e.g. stethoscopes, blood pressure cuffs and thermometers).
- If equipment needs to be shared among patients, clean and disinfect it after each patient use.
- HCWs should refrain from touching their eyes, nose or mouth with potentially contaminated gloved or ungloved hands.
- Clean and disinfect patient-contact surfaces (e.g. bed and machines) after use
- Limit the number of HCWs, family members and visitors in contact with a patient with probable or confirmed MERS-CoV infection.
- To the extent possible, assign probable or confirmed cases to be cared for exclusively by a group of skilled HCWs and housekeepers both for continuity of care and to reduce opportunities for

inadvertent infection control breaches that could result in unprotected exposure.

- Family members and visitors in contact with a patient should be limited to those essential for patient support and should be trained on the risk of transmission and on the use of the same infection control precautions as HCWs who are providing routine care. Further training may be needed in settings where hospitalized patients are often cared for by family members (sitters).
- Additional precautions when performing aerosol-generating procedures:
 - Wear a clean, non-sterile, long-sleeved gown and gloves (some of these procedures require sterile gloves).
 - Wear an impermeable apron for some procedures with expected high fluid volumes that might penetrate the gown;
 - Perform procedures in a negative pressure room.
 - Limit the number of persons present in the room to the absolute minimum required for the patient's care and support;
 - Perform hand hygiene before and after contact with the patient and his or her surroundings and after PPE removal.

Management of health care workers who had contacts with patients with MERS-CoV infection:

- Health care facilities should trace all health care workers who had protected or unprotected contacts with patients with suspected, probable, or confirmed MERS-CoV infection.
- Contacts should not be routinely tested for MERS-CoV unless they develop upper or lower respiratory illness.
- Contacts should continue to work in the hospital unless they develop upper or lower respiratory illness.
- The infection control unit of the facility or equivalent thereof should proactively call by phone all contacts to assess their health on a daily basis for a total of 14 days.
- The Infection Control unit should be notified of all contacts who develop a respiratory illness.
- Symptomatic contacts should be assessed clinically. Nasopharyngeal swabs should be collected and tested for MERS-CoV PCR.
- Symptomatic contacts should be managed as suspected cases.

Duration of isolation precautions for MERS-CoV infection:

- Since the duration of infectivity for MERS-CoV infection is unknown, nasopharyngeal swab should be repeated every 3 days for in-patients to test for viral shedding to assist the decision making particularly in regard to when to stop isolation.
- While standard precautions should continue to be applied always, additional isolation precautions should be used during the duration of symptomatic illness and continued until 48 hours after the resolution of symptoms; AND At least one nasopharyngeal sample is negative for MERS-CoV RNA.

Ebola Virus Disease (EVD)

Case Definition for Ebola Virus Disease:

Suspected Case:

Illness in a person who has both consistent symptoms and risk factors as follows:

Clinical criteria, which includes fever of greater than 38.6°C, and additional symptoms such as severe headache, muscle pain, vomiting, diarrhea, abdominal pain, or unexplained hemorrhage (gingival, nasal, cutaneous [petechiae, bruises, ecchymosis], gastrointestinal, rectal [gross or occult blood], urinary [gross or microscopic hematuria], vaginal, or puncture sites bleeding); AND **Epidemiologic risk factors** within the past 3 weeks before the onset of symptoms, such as contact with blood or other body fluids of a patient known to have or suspected to have EVD; residence in—or travel to—an area where EVD transmission is active; or direct handling of dead or alive fruit bats, monkeys, chimpanzees, gorillas, forest antelope and porcupines from disease-endemic areas. Malaria diagnostics should also be a part of initial testing because it is a common cause of febrile illness in persons with a travel history to the affected countries.

Confirmed Case:

A suspected case with laboratory-confirmed diagnostic evidence of Ebola virus infection.

Laboratory Diagnosis:

Laboratory tests used in diagnosis include:

Timeline of Infection	Diagnostic tests available
Within a few days after symptoms begin	<ul style="list-style-type: none"> • Antigen-capture enzyme-linked immunosorbent assay (ELISA) testing • IgM ELISA • Polymerase chain reaction (PCR) • Virus isolation
Later in disease course or after recovery	<ul style="list-style-type: none"> □ IgM and IgG antibodies
Retrospectively in deceased patients	<ul style="list-style-type: none"> • Immunohistochemistry testing • PCR • Virus isolation

Please refer to the MOH Guidelines for Specimen Collection, Transport, Testing, and Submission for Patients with Suspected Infection with Ebola Virus Disease for details.

Patient Placement:

The patient should be isolated according to the MOH Guidelines for Ebola Virus Disease.

Management of “Patient under investigation” for Ebola Virus Disease:

No specific vaccine or antiviral drug has been proven to be effective against Ebola.

Symptoms of Ebola are treated as they appear. The following basic interventions, when used early, can increase the chances of survival:

- Providing intravenous fluids and balancing electrolytes (body salts)
- Maintaining oxygen status and blood pressure
- Treating other infections if they occur

Timely treatment of Ebola HF is important but challenging because the disease is difficult to diagnose clinically in the early stages of infection. Because early symptoms, such as headache and fever, are nonspecific to ebolaviruses, cases of Ebola HF may be initially misdiagnosed.

Personal Protective Equipment (PPE):

- All persons entering the patient room should wear at least: Gloves, Gown (fluid resistant or impermeable), Eye protection (goggles or face shield), Face mask .
- Additional PPE might be required in certain situations (e.g., copious amounts of blood, other body fluids, vomit, or feces present in the environment), including but not limited to:

- Full body (overall) water-proof suit that covers the whole body from head to ankles.
- Double gloving
- Disposable shoe covers
- Recommended PPE should be worn by HCWs upon entry into patient rooms or care areas.
- Upon exit from the patient room or care area, PPE should be carefully removed and discarded without contaminating one's eyes, mucous membranes, or clothing with potentially infectious materials.
- Hand hygiene should be performed immediately after removal of PPE.

Patient Care Equipment:

- Dedicated medical equipment (preferably disposable, when possible) should be used for the provision of patient care.
- All non-dedicated, non-disposable medical equipment used for patient care should be cleaned and disinfected according to manufacturer's instructions and hospital policies.

Patient Care Considerations:

- Phlebotomy, procedures, and laboratory testing should be limited to the minimum necessary for essential diagnostic evaluation and medical care

- All needles and sharps should be handled with extreme care and disposed in punctureproof, sealed containers.

Aerosol Generating Procedures (AGPs):

- An aerosol-generating procedure (AGP) is defined as any medical procedure that can induce the production of aerosols of various sizes, including small (< 5 micron) particles.
- Aerosol-generating procedures that may be associated with an increased risk of infection transmission includes both elective procedures such as bronchoscopy, sputum induction, elective intubation and extubation, as well as emergency procedures such as cardiopulmonary resuscitation, initiation of Bilevel Positive Airway Pressure-BIPAP, emergency intubation, open suctioning of airways, manual ventilation via umbo bagging through a mask before intubation.
- Avoid AGPs for EVD patients.
- Additional precautions when performing aerosol-generating procedures:
 - Wear N95 masks, and always check the seal.
 - Wear eye protection.
 - Wear a clean, non-sterile, long-sleeved waterproof gown and gloves (some of these procedures require sterile gloves).
 - Wear disposable shoe covers.

- Perform procedures in a negative pressure room, When a negative pressure room is not available, conduct the procedure in a private room.
- Perform hand hygiene before and after contact with the patient and his or her surroundings and after PPE removal.
- Conduct environmental surface cleaning following procedures.

Environmental Infection Control:

- Diligent environmental cleaning and disinfection and safe handling of potentially contaminated materials is paramount, as blood, sweat, emesis, feces and other body secretions represent potentially infectious materials
- HCWs performing environmental cleaning and disinfection should wear recommended PPE (described above) and consider use of additional barriers (shoe and leg coverings, etc.) if needed.
- Face protection (face shield or facemask with goggles) should be worn when performing tasks such as liquid waste disposal that can generate splashes.
 - Follow standard procedures, per hospital policy and manufacturers' instructions, for cleaning and/or disinfection of Environmental surfaces and equipment, Textiles and laundry, Food utensils and dishware.

Duration of Infection Control Precautions:

Duration of precautions should be determined on a case-by-case basis. Factors that should be considered include, but are not limited to: presence of symptoms related to EVD, date symptoms resolved, other conditions that would require specific precautions (e.g., tuberculosis, *Clostridium difficile*) and available laboratory information.

Monitoring and Management of Potentially Exposed Healthcare Workers HCWs:

- Persons with percutaneous or mucocutaneous exposures to blood, body fluids, secretions, or excretions from a patient with suspected EVD should:
 - Stop working and immediately wash the affected skin surfaces with soap and water. Mucous membranes (e.g., conjunctiva) should be irrigated with copious amounts of water or eyewash solution.
 - Immediately contact occupational health/supervisor for assessment and access to postexposure management services for all appropriate pathogens (e.g., Human Immunodeficiency Virus, Hepatitis C, etc.)
- HCWs who develop sudden onset of fever, intense weakness or muscle pains, vomiting, diarrhea, or any signs of hemorrhage after an unprotected exposure

(i.e. not wearing recommended PPE at the time of patient contact or through direct contact to blood or body fluids) to a patient with EVD should:

- Not report to work or should immediately stop working.
 - Notify their supervisor.
 - Seek prompt medical evaluation and testing.
 - Notify public health/infection control departments.
- For asymptomatic HCWs who had an unprotected exposure (i.e. not wearing recommended PPE at the time of patient contact or through direct contact to blood or body fluids) to a patient with EVD:
 - Should receive medical evaluation and follow-up care including fever monitoring twice daily for 21 days after the last known exposure.
 - May continue to work while receiving twice daily fever checks.
 - Asymptomatic HCWs are not allowed to travel by commercial airplane.
 - Local travel for asymptomatic HCWs (e.g. taxi, bus) should be assessed in consultation with local public health authorities.

Hand hygiene

Transmission of health care-associated pathogens from one patient to another via health care workers' hands requires five sequential steps:

1. Organisms are present on the patient's skin, or have been shed onto inanimate objects immediately surrounding the patient.
2. Organisms must be transferred to the hands of health care workers.
3. Organisms must be capable of surviving for at least several minutes on health care workers' hands.
4. Hand washing or hand antisepsis by the health care workers must be inadequate or entirely omitted, or the agent used for hand hygiene inappropriate; and
5. The contaminated hand or hands of the caregiver must come into direct contact with another patient or with an inanimate object that will come into direct contact with the patient.

At present, alcohol-based handrubs are the only known means for rapidly and effectively inactivating a wide array of potentially harmful microorganisms on hands.

Alcohol-based handrubs is recommended based on the following factors:

- Evidence-based fast-acting and broad-spectrum microbicidal activity with a minimal risk of generating resistance to antimicrobial agents.

- Capacity to promote improved compliance with hand hygiene by making the process faster and more convenient.
- Economic benefit by reducing annual costs for hand hygiene, representing approximately 1% of extra-costs generated by HCAI.

Indications for hand hygiene:

- Wash hands with soap and water when visibly dirty or visibly soiled with blood or other body fluids or after using the toilet.
- If exposure to potential spore-forming pathogens is strongly suspected or proven, including outbreaks of *Clostridium difficile*, hand washing with soap and water is the preferred means.
- Use an alcohol-based handrub as the preferred means for routine hand antisepsis in :
 - Before and after touching the patient.
 - Before handling an invasive device for patient care, regardless of whether or not gloves are used.
 - After contact with body fluids or excretions, mucous membranes, non-intact skin, or wound dressings.
 - if moving from a contaminated body site to another body site during care of the same patient after contact with inanimate surfaces and objects (including medical equipment) in the immediate vicinity of the patient.
 - After removing sterile or non-sterile gloves.
- If hands are not visibly soiled. if alcohol-based handrub is not obtainable, wash hands with soap and water.

- Before handling medication or preparing food perform hand hygiene using an alcohol-based handrub or wash hands with either plain or antimicrobial soap and water.
- Soap and alcohol-based handrub should not be used concomitantly.

Hand hygiene technique:

- Apply a palmful of alcohol-based handrub and cover all surfaces of the hands. Rub hands until dry
- When washing hands with soap and water, wet hands with water and apply the amount of product necessary to cover all surfaces. Rinse hands with water and dry thoroughly with a single-use towel. Use clean, running water whenever possible. Avoid using hot water, as repeated exposure to hot water may increase the risk of dermatitis
- Use towel to turn off tap/ faucet
- Dry hands thoroughly using a method that does not recontaminate hands. Make sure towels are not used multiple times or by multiple workers.
- Liquid, bar, leaf or powdered forms of soap are acceptable. When bar soap is used, small bars of soap in racks that facilitate drainage should be used to allow the bars to dry.

Surgical hand preparation

- Remove rings, wrist-watch, and bracelets before beginning surgical hand preparation. Artificial nails are prohibited.
- Sinks should be designed to reduce the risk of splashes

- If hands are visibly soiled, wash hands with plain soap before surgical hand preparation. Remove debris from underneath fingernails using a nail cleaner, preferably under running water.
- Brushes are not recommended for surgical hand preparation.
- Surgical hand antisepsis should be performed using either a suitable antimicrobial soap or suitable alcohol-based handrub, preferably with a product ensuring sustained activity, before donning sterile gloves.
- When performing surgical hand antisepsis using an antimicrobial soap, scrub hands and forearms for the length of time recommended by the manufacturer, typically 2–5 minutes. Long scrub times (e.g. 10 minutes) are not necessary.
- When using an alcohol-based surgical handrub product with sustained activity, follow the manufacturer's instructions for application times. Apply the product to dry hands only. Do not combine surgical hand scrub and surgical handrub with alcohol-based products sequentially.
- When using an alcohol-based handrub, use sufficient product to keep hands and forearms wet with the handrub throughout the surgical hand preparation procedure
- After application of the alcohol-based handrub as recommended, allow hands and forearms to dry thoroughly before donning sterile gloves.

Skin care:

- Provide alternative hand hygiene products for health care workers with confirmed allergies or adverse reactions to standard products used in the health-care setting.
- Provide health care workers with hand lotions or creams to minimize the occurrence of irritant contact dermatitis associated with hand antisepsis or handwashing
- When alcohol-based handrub is available in the health-care facility for hygienic hand antisepsis, the use of antimicrobial soap is not recommended.

Use of gloves

- The use of gloves does not replace the need for hand hygiene by either handrubbing or handwashing.
- Wear gloves when it can be reasonably anticipated that contact with blood or other potentially infectious materials, mucous membranes, or non-intact skin will occur.
- Remove gloves after caring for a patient. Do not wear the same pair of gloves for the care of more than one patient.
- When wearing gloves, change or remove gloves during patient care if moving from a contaminated body site to either another body site (including non-intact skin, mucous membrane or medical device) within the same patient or the environment
- The reuse of gloves is not recommended.
- Keep natural nails short (tips less than 0.5 cm long or approximately ¼ inch).

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