A POCKET GUIDE FOR CLINICIANS DURING HAJJ

Technical supervisory committee for hospitals and primary care centers during Hajj

Ministry of Health

Saudi Arabia

وزارة الصحة
Ministry of Health

Seventh Edition
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Produced by
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Foreword

I have the pleasure to introduce the seventh 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 gives the priority to the heat syndromes, which represent the most acute environmental based challenges this season.

I hope that every staff read this Hajj pocket book especially about life saving procedures, related to airway obstruction, shortness of breath and chest pain, as well as the management of heat stroke, heat exhaustion, emergency preparedness and disaster plan. Putting in consideration the priorities in managing seriously ill or traumatized patients, paying 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.

Also this pocket book is a guide for the clinicians to manage the important infectious diseases that might be faced during Hajj, also it contains precautions that help you to be protected from infectious diseases and what to do if exposed to any of them.

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

DR. Abdulaziz Hamid Alghamdi, MD, BP,
Head of Technical supervisory committee for hospitals and primary care centers during Hajj,
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Saudi Arabia 2016 – 1437

Preface
Of Seventh Edition

A Guide for Clinicians during Hajj, Presented in its seventh edition in a size of a pocket book provides a practical account of emergency medicine.

This pocket book concentrates on the common emergencies and diseases encountered during hajj.

The aim of this book is to provide the required information to make sound and safe treatment decisions in a mass gathering and high-risk area of clinical practice.

Heat syndromes were updated, and given the priority, because they represent

The most acute environmental based challenges this season are the Heat illnesses, as the temperatures in Makkah can rise higher than 48ºC, therefor the topic of heat illnesses was updated and elaborated.

In this edition, Yellow haemorhagic fever have been introduced to the infectious diseases that might be faced during Hajj of this season, also the life threatening infections, pneumonia in hajj and definition of sepsis were updated.

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, large 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, 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

Author: DR. Abdulaziz Hamid Alghamdi, MD, ABP, Consultant pediatrician, Director General for Hospitals, Ministry of health, Saudi Arabia.

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 general practitioners 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, co-operative 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 do not see Him, He sees you. Hoping for the reward (Thawab) from "Allah" for your proper work and genuine efforts.

For further reading:

- Professionalism and Ethics Handbook for Residents, Saudi Commission for Health Specialties, Riyadh – 2015

CHAPTER 1
ACUTE ENVIRONMENTAL AND MASS-GATHERING BASED CHALLENGES

Heat illnesses

Heat Stroke
Heat Exhaustion
Heat injury
Heat Syncope
Heat Oedema
Prickly heat
Sunburn
Heat Cramps
Exertion-associated hyponatremia

Disaster Management and Emergency Preparedness

HEAT ILLNESSES

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Heat illnesses are among the leading causes of morbidity and mortality during Hajj, particularly in summer.
Temperatures in Makkah can rise higher than 45ºC. Lack of acclimatization, high intensity physical exertion poor physical fitness, and exposed spaces produce heat illnesses in many pilgrims.
Accordingly, Clinicians, who care for pilgrims with severe heat illness, need to be aware of the basic physiologic principles of thermoregulation, the spectrum of heat illness, strategies for prevention, careful evaluation at the field and hospital, rapid cooling, and must remain with high clinical suspicion to detect developing complications, and treat them as early as possible.

HEAT STROKE

Definition:

Heat stroke is defined as a core body temperature usually in excess of 40ºC. (104ºF) with associated central nervous system dysfunction in the setting of a large environmental heat load that cannot be dissipated.

There are two types of heat stroke:

- **Classic (nonexertional) heat stroke** – Classic heat stroke affects elderly individuals with risk factors that impair thermoregulation, prevent removal from a hot environment, or interfere with access to hydration or attempts at cooling.
- **Exertional heat stroke** – Exertional heat stroke generally occurs in young, otherwise healthy individuals who engage in heavy exercise during periods of high ambient temperature and humidity.

Pathophysiology:

Body temperature is maintained within a narrow range by balancing heat load with heat dissipation. The body's heat load results from both metabolic processes and absorption of heat from the environment. As core temperature rises, the preoptic nucleus of the anterior hypothalamus stimulates efferent fibers of the autonomic nervous system to produce sweating and cutaneous vasodilation.

Evaporation is the principal mechanism of heat loss in a hot environment, but this becomes ineffective above a relative humidity of 75 percent. The other major methods of heat dissipation—radiation (emission of infrared electromagnetic energy), conduction (direct transfer of heat to an adjacent, cooler object), and convection (direct transfer of heat to convective air currents)—cannot efficiently transfer heat when environmental temperature exceeds skin temperature.

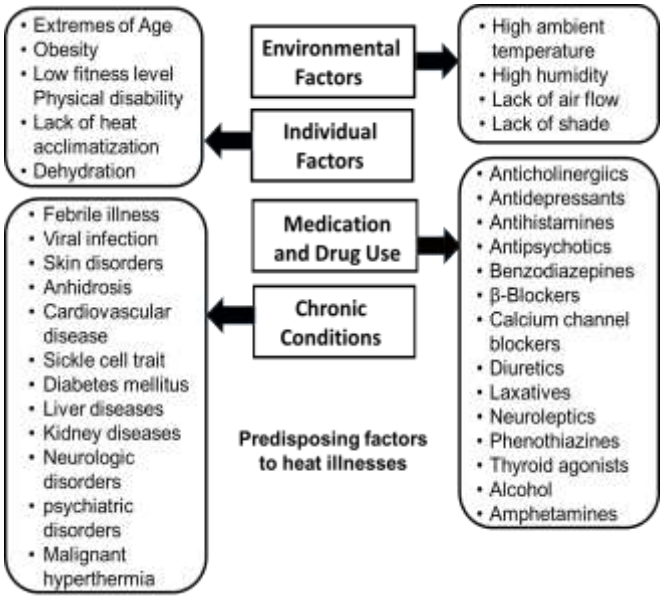
As the temperature, reach 42ºC. The cells of the body start to breakdown and lose their functional capability, resulting in multi-organ system failure and disseminated intravascular coagulation (DIC).

Risk factors:

Conditions that impair thermoregulation, prevent removal from a hot environment, or interfere with access to hydration or attempts at cooling, these conditions include:

- Extremes of age, especially pligrims above 65 years old.

- Cardiovascular disease or hypertension.
- Diabetes.
- Neurologic, psychiatric disorders or Central nervous system medications.
- Liver diseases.
- Kidney diseases or diuretics.
- Obesity.
- Anhidrosis.
- Physical disability.
- The use of recreational drugs, such as alcohol or cocaine.
- Anticholinergic agents.
- Lack of acclimatization.



Acclimatization:

Acclimatization is the body's ability to improve its response and tolerance of heat stress over time, and it is the most important factor that determines how well an athlete withstands extreme heat.

Thus, allowing sufficient time and using optimal training strategies that enable pilgrims to acclimatize are critical for improving performance and mitigating the risk for exertional heat illnesses. Observational studies have found that the first week of athletic practice in high heat and humidity is the period of greatest risk for developing exertional heat illnesses.

Full acclimatization requires at least 10 to 14 days of exercise at an intensity that raises body temperature to at least 38.51C for at least 60 minutes. This can be accomplished in either hot environmental conditions or cooler conditions if clothing or equipment is worn and exercise intensity is high. However, any improved tolerance of heat stress generally dissipates within 2 to 3 weeks of returning to a more temperate environment.

The major physiologic adjustments that occur during heat and humidity acclimatization include:

1. Plasma volume expansion.
2. Improved cutaneous blood flow.
3. Lower threshold for initiation of sweating.
4. Increased sweat output.
5. Lower salt concentration in sweat.
6. Lower skin and core temperatures for a standard exercise.

These adaptations allow for better dissipation of heat during exertion and limit increases in body temperature compared with pilgrims who have not acclimatized.

Risk factors for increased mortality:

Patients who present to the hospital with heat stroke have high mortality. Which is directly correlated to the duration of core temperature elevation, time to initiation of cooling measures, and the number of organ systems affected.

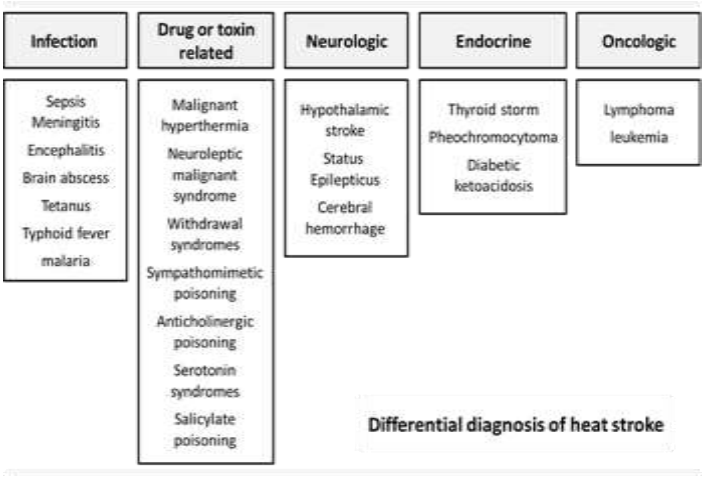
Diagnosis:

Clinical features:

- **History:**
  - History of exposure to severe environmental heat.
  - If they can respond coherently, patients with heat stroke may complain of weakness, lethargy, nausea, or dizziness.
- **Physical findings:**
  - Elevated core body temperature (generally >40ºC [104ºF]), some patients with heat stroke may not exceed 40ºC, particularly if cooling measures were initiated prior to the patient's arrival at the hospital, A thermometer (rectal or esophageal) that is accurate at high temperatures must be used when assessing heat stroke patients.
  - Central nervous system dysfunction (eg, altered mentation, slurred speech, irritability, inappropriate behavior, agitation, ataxia and other signs of poor coordination, delirium, seizures, and coma).
  - Lack another explanation for their hyperthermia (eg, infection).
  - Vital sign abnormalities (eg, tachycardia, tachypnea, hypotension).
  - Other physical findings may include flushing (cutaneous vasodilation), diarrhea, crackles due to noncardiogenic pulmonary edema and aspiration pneumonia.
  - The skin may be moist or dry, depending upon underlying medical conditions, the speed with which the heat stroke developed, and hydration status, not all victims of heat stroke are volume-depleted.
  - The presentation of elder adults with heat stroke may be subtle and nonspecific early in the course of the disease.
  - The severity of a heat illness may not be apparent during the initial presentation.
  - Frequently encountered complications include acute respiratory distress syndrome (ARDS), disseminated intravascular coagulation (DIC), acute kidney injury (i.e. acute renal failure), hepatic injury, hypoglycemia, and rhabdomyolysis.

Differential diagnosis of heat stroke:

The differential diagnosis of severe hyperthermia is extensive and includes infectious, endocrine, central nervous system, toxic, and oncologic etiologies.



Differential diagnosis of heat stroke

Diagnostic evaluation:

No single diagnostic test definitively confirms or excludes heat stroke. Furthermore, laboratory study abnormalities may overlap in patients with heat stroke and with hyperthermia due to other conditions.

- Continuous monitoring of rectal temperature should be obtained in all patients suspected of heat stroke.
- A chest radiograph may demonstrate pulmonary edema.
- The electrocardiogram may reveal dysrhythmias, conduction disturbances, nonspecific ST-T wave changes, or heat-related myocardial ischemia or infarction.

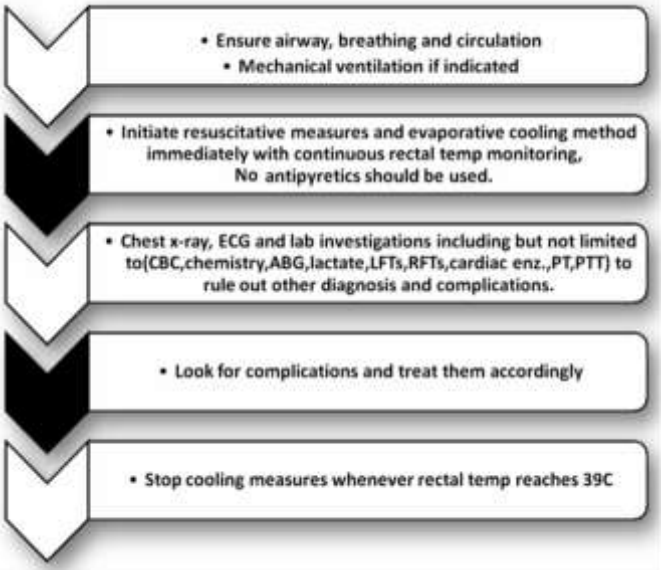
- Laboratory studies to obtain in the patient with heat stroke Including but not limited to Complete blood count, serum electrolyte concentrations, Arterial or venous blood gas, serum lactate, blood urea nitrogen (BUN) and creatinine concentrations, liver function test, cardiac enzymes, Prothrombin time (PT) and partial thromboplastin time (PTT), septic screening. the common laboratory features ;
  - Leukocytosis may reach as high as 30,000 to 40,000/mm^3.
  - Hypokalemia, hyponatremia, hyperglycemia,
  - Metabolic acidosis and respiratory alkalosis are the most common abnormalities.
  - Acute renal failure: high urea and creatinine with hemogranular casts and proteinuria.
  - Features of Rhabdomyolysis: (eg, serum creatine kinase, urine myoglobin) and its complications (eg, hypocalcemia, hyperphosphatemia, myoglobinuria, and BUN and creatinine). Myoglobinuria should be suspected in any patient with brown urine supernatant that is heme-positive and clear plasma. Urinalysis may reveal other evidence of renal injury, including protein, blood, renal tubular casts, and increased specific gravity.
  - Heat-induced liver damage: Hepatic transaminase concentrations. Transaminase concentrations are rarely normal in patients with heat stroke; however, in patients with severe liver injury marked elevations may not appear for 24 to 48 hours.
  - DIC (low platelet, low Hb, high PT, high PTT, high D-Dimer (or FDP), low fibrinogen).
  - Toxicologic screening may be indicated if a medication effect is suspected. Drugs that may contribute to hyperthermia and for which tests are often available include ethanol, amphetamines, cocaine, salicylates, hallucinogens, and lithium.
  - A head CT and analysis of the cerebrospinal fluid should be performed as indicated if central nervous system causes of altered mental status are suspected.

Treatment:

Treatment Goals:

- Normal vital signs.
- Euhydration state.
- Full consciousness.
- Normal investigations.
- Avoid and treat complications.

Algorithm for Management of heat stroke



Resuscitative measures and monitoring:

Establish Airway and maintain Breathing:

- Tracheal intubation and mechanical ventilation are needed for patients unable to protect their airway or with deteriorating respiratory function to overcome the oxygen demand.

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.
- Central venous pressure (CVP) monitoring may be useful for assessing volume status and determining the need for fluid resuscitation. A target CVP of 8 to 12 cm H_2O is an appropriate target. However, CVP readings may be inaccurate if heat-related cardiac dysfunction develops (eg, acute right heart failure). Alpha-adrenergic agonists should be avoided, since the resultant vasoconstriction decreases heat dissipation.
- Cardiac and hemodynamic monitoring.
- Folly's catheter (to monitor urine output).
- Monitor; vital signs, O_2 saturation.
- Input output monitoring and charting.

Rapid cooling:

- For hyperthermia (Rectal temperature > 40º C); Start evaporative Cooling measures (Fan and body water spray). With evaporative cooling, the naked patient is sprayed with a mist of lukewarm water while fans are used to blow air over the moist skin. This Evaporative cooling is the method used most often to treat heat stroke because it is effective, noninvasive, easily performed, and does not interfere with other aspects of patient care. When used to treat elderly patients, evaporative cooling is associated with decreased morbidity and mortality.
- Other effective cooling methods are less commonly used in patients with heat stroke:
  - Special beds called body cooling units have been made for this purpose is an alternative method that allows greater access to the patient, in which the patient is placed supine on a porous stretcher , alternate warm and cold mist with air used till temperature reaches 39ºc.
  - Applying cold compresses to the glabrous (smooth, hairless) skin surfaces of the cheeks, palms, and soles.

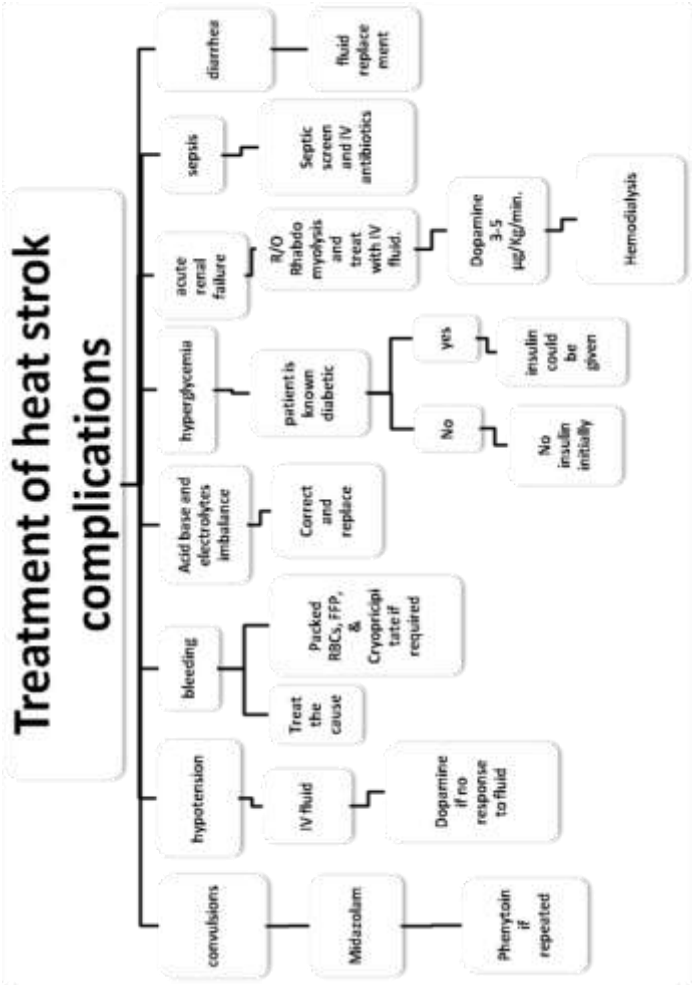
  - Applying ice packs to the axillae, neck, and groin (areas adjacent to major blood vessels, but may be poorly tolerated by the awake patient.
  - Immersing the patient in ice water (cold water immersion) is an efficient, noninvasive method of rapid cooling, but it complicates monitoring and intravenous access, and may be harmful to elder patients.
- Continuous core temperature monitoring rectally and cooling measures should be stopped once a temperature less than 39ºc has been achieved in order to reduce the risk of iatrogenic hypothermia.
- Pharmacologic therapy — Pharmacologic therapy is not required in heat stroke. There is no role for antipyretic agents such as acetaminophen or aspirin in the management of heat stroke, since the underlying mechanism does not involve a change in the hypothalamic set-point and these medications may exacerbate complications such as hepatic injury or disseminated intravascular coagulation (DIC). Salicylates can contribute to hyperthermia by uncoupling oxidative phosphorylation. Dantrolene is ineffective in patients with severe temperature elevation not caused by malignant hyperthermia.

Exclude other causes of hyperthermia:

- Rapid improvement with active cooling suggests that heat stroke is the primary diagnosis. However, clinicians should investigate alternative causes for hyperthermia, and detect heat stroke complications as early as possible.

Treatment of complications:

Severe hyperthermia may lead to a wide range of complications. These often resolve as cooling measures take effect, but this depends upon the degree and duration of hyperthermia. Complications may include the following:


Treatment of heat strok complications
convulsions
Midazolam
Phenytoin if repeated
hypotension
IV fluid
Dopamine if no response to fluid
bleeding
Treat the cause
Packed RBCs, FFP, & Cryoprecipitate if required
Acid base and electrolytes imbalance
Correct and replace
hyperglycemia
patient is known diabetic
No
No insulin initially
yes
insulin could be given
acute renal failure
R/O Rhabdomyolysis and treat with IV fluid.
Dopamine 3-5 µg/Kg/min.
Hemodialysis
sepsis
Septic screen and IV antibiotics
diarrhea
fluid replacement

- **Respiratory dysfunction:**

  Patients with heat stroke often develop pulmonary complications, which can include aspiration, bronchospasm, noncardiogenic pulmonary edema, acute respiratory distress syndrome, pneumonitis, pulmonary infarction, and pulmonary hemorrhage. Tracheal intubation and mechanical ventilation are often necessary to protect the airway and to address increased metabolic demands (ie, provide supplemental oxygen and increased minute ventilation.

- **Convulsions:**

  Seizures are common in patients with heat stroke. Initial treatment consists of short-acting benzodiazepines such as:

  - Midazolam 0.1-0.2 mg/kg IV, to a maximum dose 4 mg, has an onset of action one to five minutes and duration of action of one to six hours.
  - Lorazepam 0.1 mg/kg IV, to a maximum dose 4 mg, is a second-line option, as the duration of action may be prolonged from 12 to 24 hours.
  - Barbiturates should be avoided.
  - Rapid cooling measures.

- **Arrhythmia and cardiac dysfunction:**

  Potential cardiac complications include acute decompensated heart failure and myocardial injury associated with reversible cardiac biomarker increase and ST-segment changes on electrocardiogram (ECG). The biomarker and ECG changes are believed to be caused by an increase in catecholamine levels due to heat stroke, causing a stress-induced cardiomyopathy. Other ECG abnormalities that have been reported in association with heat stroke include sinus tachycardia and other tachyarrhythmias, conduction abnormalities, prolonged QT interval, transient Brugada pattern, and nonspecific ST-T changes.

  - Rapid cooling is essential; cardiac dysfunction and tachyarrhythmias generally resolve with cooling.

- Antiarrhythmics are seldom necessary. and electrical cardioversion should be avoided until cooling is achieved, unless necessary to treat ventricular fibrillation or pulseless ventricular tachycardia.

- **Hypotension:**
  Hypotension associated with heat stroke results from peripheral vasodilation, cardiac dysfunction, and volume depletion. Treatment consists of:
  - IV fluid boluses of isotonic crystalloid solution 250 to 500 ml. then monitor according to vital signs and urine output. Given the risk of pulmonary edema, excessive fluid administration should be avoided.
  - If no response, start Dopamine 3-10 µg/Kg/min and increase up to 20 µg/Kg/min according to vital signs and CVP measurement.
  - Avoid Alpha-adrenergic agonists, which impairs cooling due to vasoconstriction.
- **Bleeding:**
  Disseminated intravascular coagulation (DIC) can develop during the first three days of illness and coagulation studies should be monitored during this period, Replacement of:
  - Lost blood with Packed RBCs.
  - Clotting factors with fresh frozen plasma and platelets, Cryoprecipitate and fibrinogen may be necessary.
- **Acute renal failure:**
  Heat stroke can cause acute kidney injury. Renal function studies and serum electrolyte concentrations should be followed closely over the first few days of illness:
  - Renal replacement therapy or Hemodialysis for overload, may be needed.
  - Correction of hyperkalemia and acidosis.
- **Rhabdomyolysis:**
  The combination of muscle injury, volume depletion, and acute kidney injury can lead to rhabdomyolysis in patients with heat stroke.
  - R/O Rhabdomyolysis by CK and treat with IV fluid.

- **Acid base and electrolytes imbalance:**
  Metabolic acidosis and respiratory alkalosis are the most common abnormalities.
  - Correct and replace accordingly.
- **Hepatic injury:**
  Hepatic injury due to heat stroke is generally self-limited but in some cases may progress to acute liver failure, with a subset of patients requiring liver transplantation.
- **Hyperglycemia:**
  - Monitor RBS and K level.
  - No insulin initially unless patient is known to be diabetic.
- **Sepsis:**
  In cases where the etiology of the patient's hyperthermia is unclear initially and infection remains a possibility, empiric administration of an initial dose of antibiotics, following collection of appropriate cultures, is prudent, while cooling measure is implemented.
- **Diarrhea:**
  - Only fluid replacement.

Discharge criteria:

- Normal vital signs.
- Fully conscious.
- Well hydrated.
- Normal investigations.
- Free of complications.

Follow up:

- Long-term outpatient therapy may be required when chronic renal failure develops and when irreversible damage to the CNS, heart, and liver occurs.

HEAT EXHAUSTION

Definition:

Heat exhaustion is defined as a heat illness with core body temperature elevation, usually less than 40ºC. (104ºF) with normal central nervous system function in the setting of strenuous physical exertion and environmental heat stress.

Pathophysiology:

Inability to maintain adequate cardiac output due to loss of salt and water, in unacclimatized patient.

Clinical features:

- **History:**
  - Most often Heat exhaustion manifests as physical collapse during exercise.
  - Body temperature elevation.
  - Headache, nausea, vomiting, dizziness, weakness and cramps.
- **Physical findings:**
  - Patient is presented with normal or increased core body (rectal) temperature < 40ºc., (milder than with heat stroke or heat injury).
  - Sweating, postural hypotension.
  - The central nervous system is not affected.

Investigations:

- If febrile; including but not limited to CBC, serum chemistry, septic screening and Chest x-ray.
- Common Laboratory Features; Hypokalemia, hyponatremia.

Treatment:

Goals: Normal vital signs, Euhydration state and Normal investigations.

- **Rest in a cool environment.**
  - Remove the patient from the hot place and move him to a shaded or air-conditioned area.
  - Place the patient supine and raise his legs.
  - Remove excess clothing and equipment.
  - Cool the patient until the patient starts shivering by running ice or cool water over him or using evaporative cooling measures.
  - Continuously observe and frequently monitor heart rate, blood pressure, respiratory rate, rectal temperature, and mental status.
- **Transport the patient** to an emergency department if rapid improvement does not occur despite appropriate treatment.
- **Initiate resuscitative measures:**
  - Fluid and salt replacement:
    - Orally; with chilled water or Salt-containing solutions if the patient is not nauseated, vomiting, or manifesting a depressed mental status.
    - If the patient is not tolerating; Check blood pressure (BP), if low; establish an IV line, Give IV bolus of NS 500 ml then maintenance IVF NS at 125 ml/hour and titrate to response.
  - Monitor; vital signs, rectal temperature, O2 saturation.
  - Input output monitoring and charting.
- **Cool the patient**:
  - Any technique used to treat heat stroke may be used; ice packing to axillae and groin, running cool water over him using a shower or hose, or using evaporative cooling measures. (Fan and body water spray).
  - Continuous core temperature monitoring rectally and cooling measures should be stopped once a temperature less than 39^{o}c has been achieved, (The time needed to reach the goal temperature will be much shorter than with heat stroke).
  - Avoid using antipyretic for temperature control as it can exacerbate Heat stroke complications.
  - Exclude other causes of hyperthermia.

Discharge criteria from emergency:

- Pilgrim who recover completely with the treatments described here within one or two hours of presentation may be discharged with a responsible adult provided that he have:
  - Normal vital signs.
  - Well hydrated.
  - No other symptom or sign of illness.

Admission criteria:

- Patient who fail to improve after two hours despite these measures, may be he is a candidate of developing late complications, consistent with possible heat injury, and should be admitted for observation and diagnostic testing.

HEAT INJURY

Definition:

Exertional heat injury is defined as a progressive multisystem disorder with hyperthermia, less than 40ºC. (104ºF) following vigorous activity that is associated with end-organ damage (eg, kidney, liver, muscle) in the absence of significant neurologic injury.

Pathophysiology:

- Exertional heat injury is similar to Heat stroke, but the patient's central nervous system is not affected and core body temperature does not have to exceed 40°C.
- It is unlike heat exhaustion, There is clear evidence of end-organ injury such as rhabdomyolysis, acute kidney injury, disseminated intravascular coagulation, or acute liver failure

Clinical features:

- **History:**
  - Most often Heat injury manifests as physical collapse during exercise.
  - Body temperature elevation.
  - May complain of weakness, lethargy or nausea.
  - No significant neurologic manifestation.
- **Physical findings:**
  - Patient is presented with normal or increased core body (rectal) temperature < 40ºc.
  - Sweating, postural hypotension.
  - The central nervous system is not affected.
  - Lack another explanation for hyperthermia (eg, infection).
  - Vital sign abnormalities (eg, tachycardia, tachypnea, hypotension).
  - The severity of the heat injury may not be apparent during the initial presentation.
  - Frequently encountered complications include acute respiratory distress syndrome (ARDS), disseminated intravascular coagulation (DIC), acute kidney injury (ie, acute renal failure), hepatic injury, hypoglycemia, and rhabdomyolysis.

Differential diagnosis:

The clinical distinction between heat injury and exertional heat stroke is made based upon a careful assessment of central nervous system dysfunction (e.g., seizure, altered mental status, abnormal behavior). In practice, this distinction is often made after the initial treatment of the patient including rapid cooling, and is based on a careful review of the event with the patient, witnesses, and other treating clinicians.

Management plan:

- Rapid cooling: using any of the methods suitable for heat stroke.

- Continuous core temperature monitoring rectally and cooling measures should be stopped once a temperature less than 39ºc has been achieved in order to reduce the risk of iatrogenic hypothermia.
- Monitoring of vital signs and urine output.
- Initial care is largely supportive.
- Laboratory studies including but not limited to urinalysis, urine myoglobin and creatine kinase. Complete blood count, serum electrolyte concentrations, Arterial or venous blood gas, serum lactate, blood urea nitrogen (BUN) and creatinine concentrations, liver function tests (serum aminotransferases (AST, ALT), cardiac enzymes, Prothrombin time (PT) and partial thromboplastin time (PTT), septic screening. And Exclude other causes of hyperthermia.
- Pharmacologic therapy may exacerbate complications such as hepatic injury or disseminated intravascular coagulation (DIC).
- The specific management plan is determined individually on the basis of the patient's presentation. (See "treatment of complications of heat stroke ").
- Organ damage does not always manifest with laboratory abnormalities early in the course of illness and clinicians should admit and monitor patients with possible heat injury closely.
- For patients without severe symptoms or signs and no grossly abnormal initial laboratory results, a reasonable approach is to reexamine the patient and recheck the relevant studies after 24 hours to assess organ function. Once symptoms and signs have resolved and two successive sets of normal laboratory values have been obtained, surveillance may be discontinued, and the patient may gradually return to normal activity

HEAT SYNCOPE

Definition:

Heat syncope is a transient loss or near-loss of consciousness due to the indirect effects of high ambient temperature that generally occurs during the first few days when a pilgrim is exposed to high environmental temperatures, before acclimatization is complete.

Pathophysiology:

Exertion associated collapse in unacclimatized pilgrims, due to heat induced peripheral vasodilatation and pooling of blood, with subsequent loss of consciousness.

Clinical features:

- **History:** Most often Heat syncope manifests as physical collapse during or after exertion, with a feeling of heaviness in the legs, Blurred vision, Confusion, Feeling warm or hot, Lightheadedness, dizziness, a floating feeling, Nausea, Sweating, Vomiting or Yawning.
- **Physical findings:** There may be a drop in blood pressure and weak pulse.

Treatment:

- Move the patient to a shaded area.
- Have the patient lay supine in a cool environment.
- Raise the legs of the patient.
- Give fluids to drink.
- The patient should avoid sudden or prolonged standing until fully recovered.

The patient should recover within 15 to 20 minutes with these maneuvers; failure to improve should prompt further evaluation, including a rectal temperature. Patients at higher risk for dangerous causes or adverse outcomes and those who do not completely recover within 20 minutes should be evaluated in the emergency department using the approach for any patient with syncope.

HEAT OEDEMA

Definition:

Heat oedema is a condition characterized by dependent edema from vasodilatory pooling, due to the indirect effects of high ambient temperature that generally occurs during the first few days when a pilgrim is exposed to high environmental temperatures, before acclimatization is complete.

Pathophysiology:

- Heat in unacclimatized pilgrims induce peripheral vasodilatation and pooling of blood, with subsequent gravitational oedema of hands, feet and legs.

Clinical features:

- **History:**
  - Older adults and People visiting hot climates from colder climates have an increased risk of heat edema, especially if they have other medical conditions that affect their circulation.
- **Physical findings:**
  - Mild swelling of hands and feet.

Treatment:

- Move the patient to a shaded area.
- Have the patient lay supine in a cool environment.
- Raise the legs of the patient.
- Give fluids to drink.

Usually heat edema Disappears after acclimatization.

PRICKLY HEAT

Definition:

Prickly heat, also called miliaria, is a rash that can develop after a person sweats far more than usual and sweat glands become blocked.

Pathophysiology:

Heat rash begins with excessive perspiration, usually in a hot, humid environment. The perspiration makes it easier for dead skin cells and bacteria on the skin to block the sweat glands, forming a barrier and trapping sweat beneath the skin, where it builds up, causing the characteristic bumps. As the bumps burst and sweat is released, there may be a prickly, or stinging sensation that gives this condition its name.

Clinical features:

- Manifests as an itchy rash of small raised red spots with a prickling or stinging sensation.
- Usually affects parts of the body covered by clothes, such as the back, abdomen, neck, upper chest, groin or armpits.

Treatment:

- In most cases, heat rash will clear up on its own in a few days if the affected area is kept cool and dry.
- Calamine lotion and/or hydrocortisone cream can relieve itching and irritation.
- If prickly heat does not go away within a few days, or if an infection developed give antibiotic (cloxacillin).

Prevention:

- Advise to:
  - Wear light, loose clothing.
  - Avoid excessive heat and humidity and avoid heavy continuous sweating by using fans or air conditioning and cool showers to stay cool and let skin air dry.
  - Avoid using any type of oil-based product, which might block sweat glands.

SUNBURN

Definition:
Sunburn is defined as red, painful skin that feels hot to the touch due to exposure to sunshine.

Pathophysiology:
Sunburn usually appears within a few hours after too much exposure to ultraviolet (UV) light from sunshine. Many people don't produce enough melanin to protect the skin well. Eventually, UV light causes the skin to burn, bringing pain, redness and swelling. Sunburn may take several days or longer to fade.

Clinical features:
Signs and symptoms of sunburn usually appear within a few hours after sun exposure:

- Pinkness or redness.
- Skin that feels warm or hot to the touch.
- Pain, tenderness or itching.
- Swelling.
- Small fluid-filled blisters, which may break.
- Headache, fever, chills and fatigue if the sunburn is severe.

Treatment:
- Pain relievers as MEPO gel or calamine lotion.
- Medications that control itching: skin corticosteroids, combined with pain relievers.

Prevention:
- Advise to avoid sun exposure between 10 a.m. and 4 p.m.
- Cover up with white clothes and umbrella.

Heat Cramps

Definition:

Heat cramps (which do not appear to be caused by increased ambient temperatures) are muscle cramps that occur during or after exertion).

Pathophysiology:

Exertion associated cramps in unacclimatized pilgrims, Due to fluid deficiencies (dehydration), electrolyte imbalances, neuromuscular fatigue, or any combination of these factors.

Clinical features:

- Painful involuntary muscle contraction, involving large muscles groups specially legs.
- Moist cool skin, a normal body temperature, and minimal distress.

Treatment:

- Relax in cool environment, stretch and massage the involved muscle to reduce acute discomfort.
- Hydrate the patient and replace sodium losses with oral salt solution, as in rehydration solutions, can be made by adding one fourth to one-half teaspoon of table salt to 1 L of water, to improve taste, add a few teaspoons of sugar and/or orange juice or lemon juice.
- IV isotonic saline therapy (rarely required). However, oral rehydration has consistently been shown to be as effective as IV rehydration when equal amounts of fluids are given.

Persistent or systemic cramping should prompt an assessment of the serum sodium to evaluate for exertional hyponatremia and raise the possibility of sickle cell crisis due to exertion.

Prevention:

Muscle cramps are thought to be prevented best through adequate conditioning, acclimatization, hydration, electrolyte replacement, and appropriate dietary practices.

Exertion-Associated Hyponatremia (EAH)

Definition:

Exertion-associated hyponatremia (EAH) is defined by a serum or plasma sodium concentration below the normal reference range of 135 mmol/L that occurs during or up to 24 hours after prolonged physical activity (usually occur when activity exceeds 4 hours).

Pathophysiology:

Two mechanisms of Low serum-sodium levels are proposed:

- A combination of excessive fluid intake and inappropriate body water retention due to ingesting water or low-solute beverages well beyond sweat losses (also known as water intoxication).
- Sweat sodium losses are not adequately replaced.

Ultimately, the intravascular and extracellular fluid has a lower solute load than the intracellular fluids, and water flows into the cells, producing intracellular swelling that causes potentially fatal neurologic and physiologic dysfunction.

Clinical features:

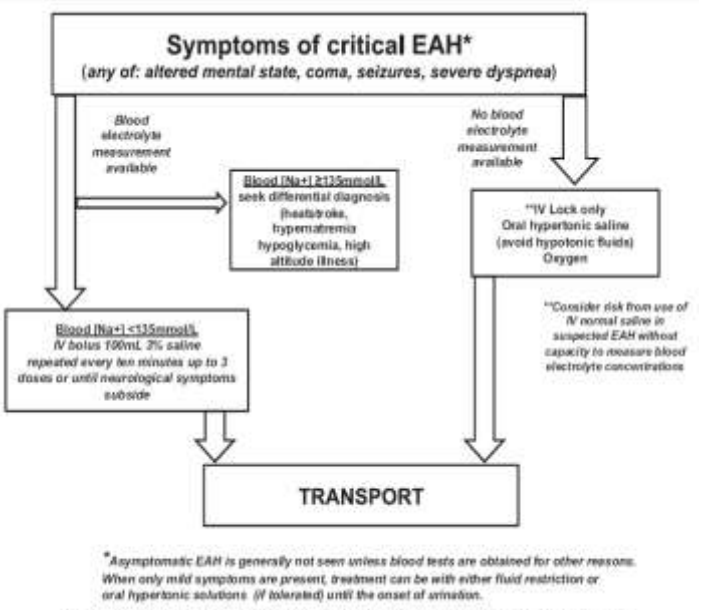
- Affected pilgrims present with a combination of disorientation, altered mental status, headache, vomiting, lethargy, and swelling of the extremities (hands and feet), pulmonary edema, cerebral edema, and seizures.
- Excessive fluid intake with low sweat and urine losses.
- Exertional hyponatremia can result in death if not treated properly.

Management plan:

- In Hajj environments in which patient evacuation to definitive care is often greatly delayed. Rapid recognition and appropriate treatment are essential in the severe form to ensure a positive outcome. Failure in this regard is a recognized cause of event-related fatality.

- It is important to differentiate Exertion-associated hyponatremia from Heat stroke, because the management is deferent, fluid therapy might kill the patient with Exertion-associated hyponatremia.

Algorithm for exertion-associated hyponatremia (EAH) field management:



Algorithm for exertion-associated hyponatremia(EAH) field management.

Acute hospital assessment and management of EAH:

Assessment:

- Urgent measurement of blood sodium by the most rapidly available means .
- Assess for clinical signs suggestive of developing cerebral edema .
- Obtain and store specimens if possible for later analysis of blood serum osmolality and urine sodium and osmolality.

Management:

- Supplemental oxygen to maintain oxygen saturation above 95%
- Restrict fluids (both IV and oral) until onset of urination.
- Avoid IV normal saline until sodium correction is initiated.
- Thereafter normal saline may be required for hypovolemic shock or in renal protection therapy for rhabdomyolysis.
- In severe EAH (signs of cerebral edema or serum sodium less than 125 mmol/L) administer IV 3% hypertonic saline as a 100-mL bolus repeated twice at 10-minute intervals aiming to reverse cerebral edema.
- Aim to increase serum sodium by approximately 4 to 5 mmol/L or until neurological symptoms are reversed by active treatment, then allow the remaining correction to occur spontaneously via urinary free water excretion.

Prevention of Exertion-associated hyponatremia:

Exertion-associated hyponatremia can be prevented by matching fluid intake with sweat and urine losses and by rehydrating with fluids that contain sufficient sodium.

Preventive measures for heat illnesses:

Heat illnesses are often preventable. Important principles for developing a prevention program for exertional heat illnesses and specific measures for reducing risk, including:

- Institute prevention policies, including an emergency action plan.
- Educate staff and pligrims about heat illness.

- Acclimatize gradually to exercising in hot and/or humid conditions; the process of heat acclimatization generally requires 7 days, under a heat stress comparable to the target competition. Training sessions for heat acclimatization should last at least 60 minutes per day, and induce an increase in core and skin temperatures, as well as stimulate sweating.
- Direct sun light exposure should be avoided.
- Remain at air-conditioned places as long as possible.
- Use white– colored umbrella whenever exposed to sun, and take rest at shady areas.
- Provide frequent breaks for hydration and cooling.
- Hydrate before activity, but don't overhydrate, and control hydration by thirst throughout activity.
- Dress light cotton–made clothes and Minimize equipment and clothing that hinder heat loss in hot or humid conditions.
- Avoid activity during severe heat and/or humidity.
- Take all medications regularly.

For further reading:

- Care of the Wilderness and Adventure Athlete, Riana R. Pryor, PhD, ATC, Department of Kinesiology, California State University, published by Wilderness & Environmental Medicine. 26, S69-S75 (2015).
- Centers for Disease Control and Prevention. Extreme Heat: A Prevention Guide to Promote Your Personal Health and Safety. Reviewed May 31, 2012..
- Exertional heat illness in adolescents and adults: Management and prevention, Francis G O'Connor, MD, MPH, FACSM, et al, Nov 04, 2015
- Health risks at the Hajj - Research Gate Qanta A Ahmed, Yaseen M Arabi, Ziad A Memish , Lancet, 2006; 367: 1008–15
- Heatstroke, Robert S Helman, MD; Chief Editor: Joe Alcock, MD, MS, Updated: May 01, 2015,
- Patient information: Heat stroke, UpToDate Patient Information Editors, 2016
- Severe nonexertional hyperthermia (classic heat stroke) in adults, C Crawford Mechem, MD, FACEP, et al , Jun 10, 2015.
- Wilderness Medical Society Practice Guidelines for the Prevention and Treatment of Heat-Related Illness:2014 Update
- Wilderness Medical Society Practice Guidelines for Treatment of Exercise-Associated Hyponatremia: 2014 Update

Disaster Management and Emergency Preparedness

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The Aim is "*to deliver the greatest good for the greatest number of patients"*.

Phases of Emergency Preparedness and disaster Management:

- Phase 1: Preparation:
  - By having a simple and a straightforward disaster plan, with an Incident Command System that provides overall direction for management of the disaster response, and to train all staff in its application by awareness and repeated drills.
- Phase 2: Mitigation:
  - The elimination or reduction of the frequency, magnitude, or severity of exposure to risks, or minimization of the potential impact of a threat or warning.
- 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 situation and replace lost 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** (Priority 1 – 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** (Priority 2 – Delayed) = is allowed for care after (Priority 1) and it will not change the outcome and they get a good chance to live.
3. **GREEN** (Priority 3 – Ambulatory patients) = Do not need stretcher to shift most of the Disaster victims.
4. **BLACK** (Priority Zero – 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 collect initially 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.

For further reading:

- The Fundamental Disaster Management (FDM), third edition, 2009, Society of Critical Care Medicine.
- Advanced Trauma Life Support (ATLS), ninth edition, 2012, American College of Surgeons.

CHAPTER 2
INITIAL ASSESSMENT AND MANAGEMENT

Recognition and evaluation of seriously ill patient

Initial management of multiply injured patient

Recognition and evaluation of seriously ill patient

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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.
- O_2 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.
- **Deficit in level of 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:** Adequate documentation is needed to assess changes and trends in patient's condition including:

- 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

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.

For further reading:

- The Fundamental Critical Care Support (FCCS), fifth edition, 2012, Society of Critical Care Medicine.

Initial management of multiply injured patient

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Management of the multiply injured patient requires a co-ordinated multi-disciplinary approach in order to optimize 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 (orotracheal 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 cross match.
- 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.

For further reading:

- Advanced Trauma Life Support (ATLS), ninth edition, 2012, American College of Surgeons.

CHAPTER 3
AIRWAY AND BREATHING EMERGENCIES

Airway management
Upper Respiratory Tract Infection
Sever Air Flow Obstruction
Acute respiratory failure

Airway management

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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?

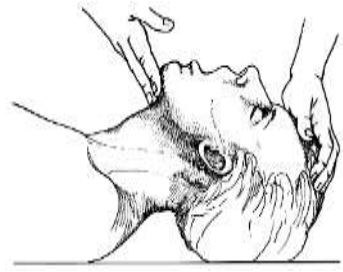
Manual :- Head tilt / chin lift / jaw thrust

With equipment: - Oro/nasopharyngeal airway,
Endotracheal intubation,
Laryngeal mask airway (LMA),
Combitube.

Manual methods:

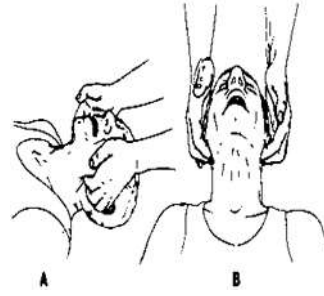
Head tilt / chin lift:

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.



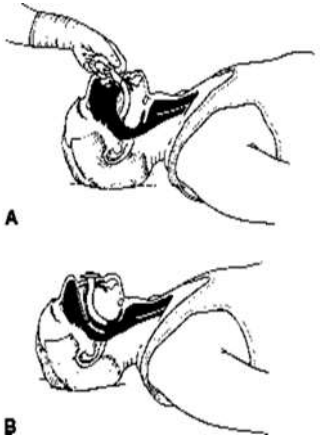
Jaw thrust:

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 semiconscious 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 (cricothroidotomy 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 vasocontricting 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 can be given before LMA placement provided that there is no sign of difficult ventilation or intubation. Contraindications to the LMA include the need for peak pressure greater than 20cms H20, patients at risk for aspiration, and patients with low lung compliance necessitating the need for high-pressure ventilation.

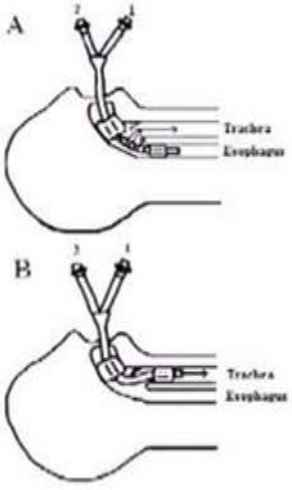
In cases of a difficult airway, the intubating LMA- can be used as a conduit for placement of an endotracheal tube.

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.

For further reading:

- The Fundamental Critical Care Support (FCCS), fifth edition, 2012, Society of Critical Care Medicine.

Upper Respiratory Tract Infection

Author: Dr. Omar Alyahia, MD , SBFM,
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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 were influenza and rhinoviruses. In hospitalized patients, Pneumonia was a significant cause of admission accounting for 20-50% of such admissions.

Acute rhinitis & rhinosinusitis:

Acute inflammation of the nasal mucosa and or sinuses, which may be viral, bacterial or allergic,

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

Treatment:

Bed rest, Plenty of fluids, Analgesics(preferably non-aspirin) , Oral antihistamines and decongestant with or without local nasal decongestants, Antibiotics--required only when secondary bacterial infection supervenes.

Intranasal steroids are of benefit in relieving symptoms in case of allergic rhinitis or rhinosinusitis.

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

Acute pharyngitis (Sore throat):

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, and headache. Pharynx shows erythema, with or without 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.

Most trials showed a beneficial combination of antihistamine with decongestive effect on general recovery as well as on nasal symptoms for adults but not children. (27 trials with over 5000 participants http://www.cochrane.org).

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, and arytenoids, false and true vocal cords.

Treatment: Voice rest, Analgesic, Avoidance of smoking, Steam inhalation, Cough suppressants, Antibiotics--when there is secondary bacterial infection, commonly used antibiotics are amoxicillin, claulanated amoxicillin or Erythromycin.

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 and 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 dyspnea & 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.

PREVENTIVE MEASURES:
The use of masks may reduce exposure to droplet nuclei, the main mode of transmission of most respiratory tract infections .There is a very strong evidence that 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.

For further reading:

- The common cold in adults: Diagnosis and clinical features
- The common cold in adults: Treatment and prevention
- Acute bronchitis in adults
- Acute sinusitis and rhinosinusitis in adults: Treatment
- http://www.moh.gov.sa/endepts/Proofs/Pages/home.aspx

Sever Air Flow Obstruction

Author: Dr. Mohammad Naser, MD, KSF, SBA&ICU, Consultant anesthesiologist, King Fahad Hospital, Jeddah.

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 oxygen to maintain an SpO2 level of 94-98%.
- Use high dose inhaled β2 agonists (oxygen-driven) as early as possible. Reserve intravenous β2 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 nebulized ipratropium bromide (0.5 mg 4-6 hourly) to β2 agonist treatment for patients with acute severe or life threatening asthma or those with a poor initial response to β2 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.

For further reading:

- The Fundamental Critical Care Support (FCCS), fifth edition, 2012, Society of Critical Care Medicine.

Acute respiratory failure

Author: Dr. Mohammad Naser, MD, KSF, SBA&ICU, Consultant anesthesiologist, King Fahad Hospital, Jeddah.

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 : <0.5–5.0 L/min
- Low oxygen : FIO_2<0.4–0.5

Air-Entrainment Face Mask:

- 100% O_2 + entrainment device
- High flow.
- Variable oxygen : FIO_2 0.24–0.5

100% O_2

Aerosol Face Mask:

- 100% O_2 + large-bore tubing
- Nebulizer/O_2 blender
- Flow matching: If mist disappears in inspiration, air is entrained Moderate-flow, variable FIO_2 device

100% O_2 + large-bore tubing

Reservoir Face Mask:

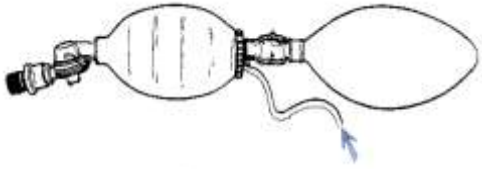
Reservoir bag filled with 100% O_2

- Reservoir bag filled with 100% O_2
- High oxygen
- High flow

Resuscitation Bag-Mask-Valve Device:

- 100% O_2 .
- 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 FIO_2
- 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 NIPPV:**
  - Set FIO_2 at 1.00
  - In Hypoxemic failure:
    - Inspiratory pressure (IPAP) 10 cm H_2O
    - Expiratory pressure (EPAP) 5 cm H_2O
    - Titrate EPAP in 2 cm H_2O increments

- In Ventilatory failure:
  - IPAP 10 and EPAP 2 cm H_2O
  - Titrate IPAP in 2 cm H_2O 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.

For further reading:

- The Fundamental Critical Care Support (FCCS), fifth edition, 2012, Society of Critical Care Medicine.

CHAPTER 4
CARDIAC AND CIRCULATORY EMERGENCIES

Diagnosis and management of Shock
Diagnosis and Management of Septic Shock
Anaphylactic shock
Management of Arrhythmias
Hypertensive Crises
Heart failure (HF) & Pulmonary edema
Acute Coronary Syndromes

Diagnosis and management of Shock

Author: Dr. Mohammad Naser, MD, KSF, SBA&ICU. Consultant anesthesiologist, King Fahad Hospital, Jeddah.

Shock:

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

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

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.

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, and massive pulmonary embolus.

Therapeutic Goals in Shock:

- Increase O_2 delivery.
- Optimize O_2 content of blood.
- Improve cardiac output and blood pressure.
- Match systemic O_2 needs with O_2 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
  - 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).
  - Maintain adequate cardiac output based on direct or indirect measurements.
  - maintain mixed venous oxygen (Spo2) more than 70.

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 μg/kg/min) – mild inotrope plus renal effect.
  - Intermediate dose (4-10 μg/kg/min) – inotropic effect.
  - High dose (>10 μg/kg/min) – vasoconstriction
  - Chronotropic effect.
- Dobutamine:
  - 5-20 μg/kg/min
  - Inotropic and variable chronotropic effects
  - Decrease in systemic vascular resistance
- Norepinephrine
  - 0.05 μg/kg/min and increase to effect (maximum dose 0.25 μg/kg/min).
  - 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.

For further reading:

- Definition, classification, etiology, and pathophysiology of shock in adults
- The Fundamental Critical Care Support (FCCS), fifth edition, 2012, Society of Critical Care Medicine.
- Evaluation of and initial approach to the adult patient with undifferentiated hypotension and shock

Diagnosis and Management of Septic Shock

Author: Dr. Mohammad Naser, MD, KSF, SBA&ICU. Consultant anesthesiologist, King Fahad Hospital, Jeddah.
Dr. Saeed Abdallah Alghamdi, FRCS, ABS, MD, Consultant vascular surgeon, King Abdullah Hospital, Bishah.
ATLS, ATOM, FCCS, FDM instructor.

Definitions:

Systemic inflammatory response syndrome (SIRS) is defined as two or more of the following:

- Temprature > 38C or < 36 C.
- Pulse rate >90 beat/min.
- Respiratory rate > 20/min or Pco2 < 32 mmHg.
- WBC >12000 or <4000 or > 10% immature band.

Infection: invasion of naturally sterile tissue by micro-organisms.

Sepsis is defined as life-threatening organ dysfunction caused by a dysregulated host response to infection.

Septic shock: Septic shock is a subset of sepsis in which underlying circulatory and cellular/metabolic abnormalities are profound enough to substantially increase mortality.

- Patients with septic shock can be identified with a clinical construct of sepsis with persisting hypotension requiring vasopressors to maintain MAP ≥65 mm Hg and having a serum lactate level >2 mmol/L (18 mg/dL) despite adequate volume resuscitation. With these criteria, hospital mortality is in excess of 40%.

Management Steps:

A. Diagnosis of severe sepsis or septic shock systemic inflammatory response syndrome criteria in addition to:
   - Systolic BP < 90mmHg after 20-30 ml/kg crystalloid challenge.
   - Blood lactate 2-4mmol/L.

B. Insertion of Central line: preferably subclavian or internal jugular line.
   - The aim to achieve CVP 8-12 cm H2O
   - Resuscitation by 500-1000ml over 30 minutes boluses of crystalloid or Colloid, which can be repeated.
   - 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.
   - Vasopressor agent as necessary to keep mean arterial pressure > 65-70mmHg.

C. Insertion of Arterial line: preferably radial.
   - Accurate measurement.
   - Beat to beat analysis so decision regarding therapy shall be immediate.

D. Get mixed venous oxygen (SCVO2) from subclavian catheter:
   - Aim to keep SCVO2 >70%.
   - Optimize CVP to 8-12 cm H2O.
   - Transfuse packed red blood cells to achieve hematocrit >30%.
   - Dobutamine infusion up to maximum of 20ug /kg/min especially if Cardiac Index <2.5 l/min/m2.
   - If could not achieved, Intubate and mechanically ventilate the patient to keep SCVO2 >70%.

E. Achieve cardiac index > 4.5L/min/m2:
   - Start Vasopressors as soon as possible to maintain mean arterial pressure 65-70mmHg both during and following adequate fluid resuscitation.
   - Norepinephrine the vasopressor of choice. 0.05 ug/kg/min titrated to effect.
   - Dopamine is equal effect but limited due to tachycardia.
   - 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:

- 100mg Hydrocortisone 3 times a day for 7 days if patient is improving or to continue if not improving or showed reduced adrenal function.
- In the absence of vasopressor requirement steroid should not be used.
- High dose of steroid is not recommended.
- Adrenal function test is optional to decide regarding:
  - Weaning of steroid at the end of treatment period.
  - Discontinuing steroids earlier.
  - Addition of oral fludrocortisones.

G. Precise bacteriological diagnosis before starting antibiotics.
- Two to three blood cultures preferably from peripheral veins, different sites.
- Appropriate samples are indicated in particular ventilator associated pneumonia or catheter related infection and soft tissue or in abdominal infections.
- 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:
- Gastrointestinal perforation.
- Acute necrotizing pancreatitis.
- Immunocompromized.
- Vascular access devices.

K. Strict control of blood Glucose level to (80-110 mg/100 ml) using continuous Insulin infusion protocol.

For further reading:

- The Fundamental Critical Care Support (FCCS), fifth edition, 2012, Society of Critical Care Medicine.
- Evaluation and management of severe sepsis and septic shock in adults
- The Third International Consensus Definitions for Sepsis and Septic Shock, JAMA. 2016;315(8):801-810. doi:10.1001/jama.2016.0287.

Anaphylactic shock

Authors:
Dr. Ali. Ghormallah Alghamdi, MD, SBIM, Consultant physician, King Saud medical city, Riyadh.

Anaphylaxis is a life-threatening allergic reaction.

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), 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:
  - 12 years: 500 micrograms IM (0.5 ml) i.e. same as adult dose and 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 2-5days 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.

For further reading:

- Anaphylaxis: Rapid recognition and treatment - UpToDate

Acute Coronary Syndromes

Authors:
Dr. Abdulmajeed Sulaiman Khan, MD, SBIM, Consultant physician, Hera general hospital, Makkah, Chairman of national CPR committee, Saudi Heart Association.

OBJECTIVES:

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

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, Pulse oxymeter saturation more than 94%, 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)
  - *Clopidogrel 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.
  - *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 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 IIb/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 monitoring
    - 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 more than 180 mmHg.
- DBP greater than 110 mmHg.
- Right vs left arm systolic BP difference more 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).

For further reading:

- Algorithms for Advanced Cardiac Life Support 2015

Management of Arrhythmias

Authors:
Dr. Abdulmajeed Sulaiman Khan, MD, SBIM, Consultant physician, Hera general hospital, Makkah, Chairman of national CPR committee, Saudi Heart Association.

Objectives:

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

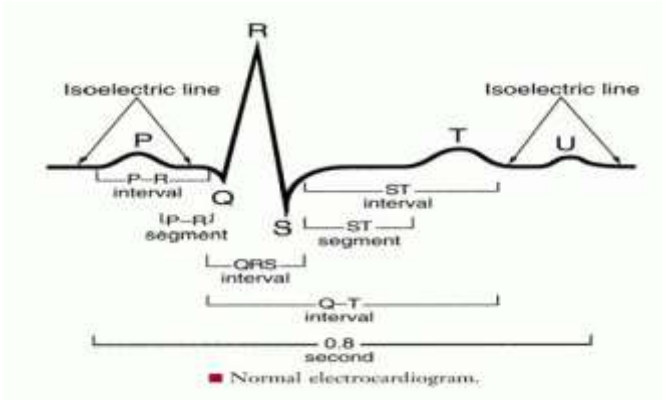
Don't Forget Your ABCDs (Adult BLS Algorithm):

- Check responsiveness, Call for help and AED / defibrillator
- Open airway and check breathing (give 2 breaths if no breathing).
- Check pulse, If no pulse, start CPR (30 compressions:2 breaths) until defibrillator arrives.
- Shock, if appropriately indicated, and resume CPR immediately.
- Check rhythm and pulse every 5 cycle (2 minutes).

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 (synchronized D/C shock), except for VF or pulseless VT you defibrillate(unsynchronized). For cardioversion the amount of energy range from 50 J to 200 J biphasic depends on the type of arrhythmia
(50 J for SVT 200J for VT with pulse).
If the patient is stable then do 12 leads ECG and decide if atrial or ventricular or AV nodal. Simply you look to the relation between the P wave and the QRS complex. Ask yourself, is the P wave present or not ? What is the shape?. Calculate the PR interval is it normal, short or prolonged?. Look to the QRS complex, is every QRS complex preceded by one P wave or more or no P wave? Is it normal in shape? What is the interval of QRS complex? What is the R - R interval? to calculate the rate and its regularity.
The normal intervals:

- P-R interval 0.12 - 0.20 sec.
- QRS complex 0.08 - 0.12 sec
- Corrected QT interval 0.40 - 0.44 sec

Follow the ACLS guidelines according the ECG interpretation.

First degree heart block:

Fixed prolonged P-R interval – Asymptomatic.
Treatment:

- Search for the underlying causes.

Second degree heart block:

2:1 block or fixed ratio (Mobitz type I) / variable ratio (Mobitz type II)- Usually symptomatic
Treatment:

- Treat underlying causes usually ischemia
  - Atropine
  - Pacemakers (IV)temporary or implanted

SINUS bradycardia:

Underlying causes:

- electrolyte imbalance
- drugs
- hypothyroidism

Treatment:

- B2 stimulants
- Atropine if symptomatic
- Pacemakers

Digitalis overdose:

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:

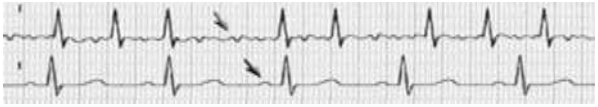
- Calcium gluconate
- Dextrose/insulin
- Sulbutamol nebulizer
- Dialysis

Premature atrial contractions:

Underlying causes:
- All heart diseases
- Electrolyte imbalance
- Hyperthyroidism
- Excess tea and coffee
- Hypoxia

Treatment:
- No specific treatment needed
- Correct underlying causes

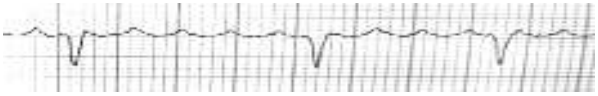
Atrial fibrillation:



Underlying causes:
- Any disease involving atria
- Lone AF.
- Thyroid disease

Treatment:
- Control rhythm & rate
- Cardioversion

Atrial flutter:



Underlying causes:
- Atrial diseases
- Hypoxia
- Hyperthyroidism

Treatment:
- Cardioversion
- Ablation

Premature ventricular contractions:

Underlying heart disease:

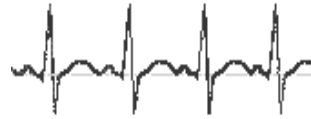
- Hypertension
- Ischemia
- Cardiomyopathy
- failure
- Electrolyte imbalance & hypoxia & drugs
- Reperfusion after thrombolysis

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.
- Cardioversion after premedication

Ventricular tachycardia:

Underlying causes:

- Serious heart disease.
- Severe electrolyte imbalance

Treatment:

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

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.

For further reading:

- Advanced cardiac life support (ACLS) in adults

Heart failure (HF) & Pulmonary edema

Authors:
Dr. Ali. Ghormallah Alghamdi, MD, SBIM, Consultant physician, King Saud medical city, Riyadh.

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, dysrhytmias, 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 headedness

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 (frusemide) 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.

For further reading:

- Overview of the therapy of heart failure due to systolic dysfunction
- Clinical manifestations and diagnosis of diastolic heart failure
- Clinical manifestations and diagnosis of diastolic heart failure

Hypertensive Crises

Authors:
Dr. Ali. Ghormallah Alghamdi, MD, SBIM, Consultant physician, King Saud medical city, Riyadh.

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 + 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, polyarteritisnodosa .
- Endocrine - Pheochromocytoma, Cushing syndrome, ..etc.
- Coarctation of the aorta.
- Preeclampsia/eclampsia .

Clinical Characteristics :

- Duration and severity of the hypertension.
- All current medications including nonprescriped 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
  - Pregnancy test
  - Endocrine testing

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 :

- ECG is indicated to assess for myocardial ischemia and left ventricular hypertrophy.
- Continuous ECG monitoring as therapy is initiated (particularly during the first half-hour; when rapid-acting vasodilators are used.
- Daily ECGs should be obtained for a short period of time to assist in the diagnosis of myocardial necrosis.

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 ≥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.

For further reading:

- Evaluation and treatment of hypertensive emergencies in adults

CHAPTER 5
NEUROLOGICAL EMERGENCIES

Coma & decreased level of consciousness
Head Injuries

Coma & decreased level of consciousness

Author: Dr. Mohammad Naser, MD, KSF, SBA&ICU. Consultant anesthesiologist, King Fahad Hospital, Jeddah.

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.

Glasgow Coma Score

Eye Opening (E)	Verbal Response (V)	Motor Response (M)
4=opens spontaneously	5=normal conversation	6=normal
3=opens to voice	4=disoriented conversation	5=localizes pain
2=opens to pain	3=words, incoherent	4=withdraws from pain
1=none	2=incomprehensible sounds	3=decorticate posturing
	1=none	2=decerebrate posturing
		1=none

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 X-Ray.
- 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.

For further reading:

- The Fundamental Critical Care Support (**FCCS**), fifth edition, 2012, Society of Critical Care Medicine.

Head Injuries

Author:
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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:
  - Pupillary Examination.
  - Lateralizing signs.
  - Basilar Skull Fractures Signs:
    - Blood in ear canal, Otorrhea.
    - Rhinorrhea.
    - Battle's sign (retroauricular hematoma).
    - Raccoon sign (periorbital ecchymosis).
  - The Glasgow Coma Scale (GCS) is the most widespread scoring method to define altered level of consciousness.

Glasgow Coma Score

Eye Opening (E)	Verbal Response (V)	Motor Response (M)
4=opens spontaneously	5=normal conversation	6=normal
3=opens to voice	4=disoriented conversation	5=localizes pain
2=opens to pain	3=words, incoherent	4=withdraws from pain
1=none	2=incomprehensible sounds	3=decorticate posturing
	1=none	2=decerebrate posturing
		1=none

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 and 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.

For further reading:

- Advanced Trauma Life Support (ATLS), ninth edition, 2012, American College of Surgeons.

CHAPTER 6
SURGICAL EMERGENCIES

Acute abdomen
Mechanical intestinal obstruction
Gastrointestinal bleeding
Diabetic foot
Immediate treatment of burn

Acute abdomen

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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. However, 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?
  - Onset?
  - Site?
  - 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 gynecologic 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 and 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.

For further reading:

- History and physical examination in adults with abdominal pain
- Evaluation of the adult with abdominal pain in the emergency department
- Differential diagnosis of abdominal pain in adults
- Acute appendicitis in adults: Clinical manifestations and differential diagnosis

Mechanical intestinal obstruction

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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:

- Strangulated hernia.
- Mesenteric vascular occlusion.
- Adhesions.
- Cancer caecum
- 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:

- Inspissated stools at rectum.
- Pseudo-intestinal obstruction.
- Colon cancer.
- Diverticulitis.
- 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.

For further reading:

- Overview of management of mechanical small bowel obstruction in adults
- Overview of mechanical colorectal obstruction

Gastrointestinal bleeding

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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:	**Upper GI bleeding**:
Brisk Upper GI bleeding.	Peptic ulcer disease.
Diverticulosis.	Varices.
Angiodysplasia.	Gastric erosions.
Cancer/polyps.	Mallory-Weiss tear.
Rectal disease.	Esophagitis.
Inflammatory bowel disease.	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/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).

For further reading:

- Approach to acute upper gastrointestinal bleeding in adults
- Approach to acute lower gastrointestinal bleeding in adults

Diabetic foot

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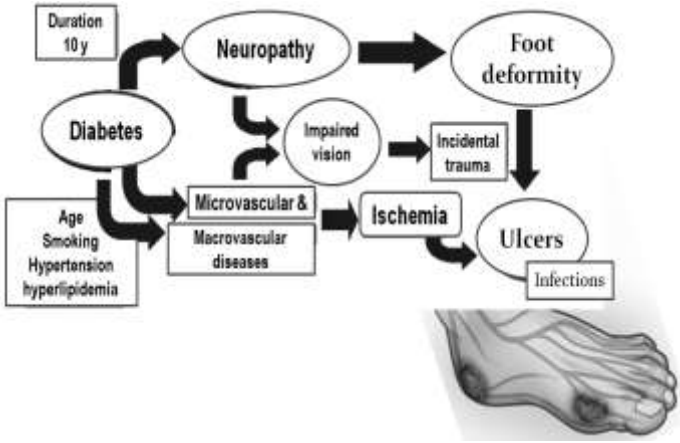
Causative factors:

Sandals are part of the pilgrimage dress, which in hot dray 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.

For further reading:

- Clinical manifestations, diagnosis, and management of diabetic infections of the lower extremities

Immediate treatment of burn

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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.
  - CoHb >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 ½ in first 8 hours.
- Administer ½ 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.

For further reading:

- Advanced Trauma Life Support (ATLS), ninth edition, 2012, American College of Surgeons.

CHAPTER 7
FLUID, ELECTROLYTES AND METABOLIC DISTURBANCES

Acute renal failure
Electrolyte imbalance
Acid Base Disturbances
Diabetic ketoacidosis (DKA)
Hyperosmolar hyperglycemic state - (Non ketotic hyperosmolar hyperglycemia)

Acute renal failure

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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 urinary catheter.
  - Upper tract obstruction by ureteral stent or percutanous 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 sever 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 urinanalysis.

Management:

Treatment of underling 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 glomerulonephritits.
  - 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.

For further reading:

- Brenner and Rector's The Kidney, 9th Edition 2012
- National Kidney Foundation Primer on Kidney Diseases, 6th Edition 2014

Electrolyte imbalance

Author:
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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 < 3.5Meq/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: Arrythemia.

Neuromuscular: fatigue, myalgia & muscle weakness of lower limb. More sever 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. • Metabolicacidosis. • Drugs e.g. B-blocker.

Manifestations:

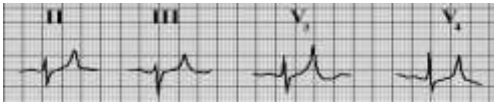
The clinical manifestation of hyperkalemia is weakness of lower limb which progress to flaccid paralysis & hypoventilation if respiratory muscle is involved.

The most serious effect of hyperkalemia is cardiac toxicity which does not correlate will 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 of no improvement in ECG within 10 min.
- Shift K^+ into cell:
  - Insulin +D50% (10u regulare 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:

- Hypvolemic 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 Na< 125 meq/l mainly neurological secondary to cerebral edema (headache, confusion, coma if no treatment, tentorial herniation, respiratory depression and 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 >12meq/24h.
- In asymptomatic patients : Fluid restriction is 1st line of therapy.
- Serum Na increased by 0.5meq/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:

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

Manifestations:

- Mainly neurological include altered mental status, weakness, Coma& seizure.
- H_2O deficit (L) =[0.6× wt (kg)] ×[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 H_2O 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 intravascular1/2 NS or 1/4 NS.
- Reduce Na cautiously: 0.5-1.0 mmol/L/hr.

Other Electrolyte Deficits (Ca, PO_4, 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 minutes.
    - Less urgent administration over 10–60 minutes.

For further reading:

- Brenner and Rector's The Kidney, 9th Edition 2012
- National Kidney Foundation Primer on Kidney Diseases, 6th Edition 2014 Clinical Physiology of Acid-Base and Electrolyte Disorders, 2001

Acid Base Disturbances

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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 24and normal Pco2 is 40.
  - Metabolic Acidosis : ↓ PH - ↓ HCO_3^- - ↓ Pco_2
  - Respiratory Acidosis: ↓ PH - ↑ HCO_3^- - ↑ Pco_2
  - Metabolic alkalosis: ↑ PH - ↑ Pco_2 - ↑ HCO_3^-
  - Respiratory alkalosis: ↑ PH - ↓ Pco_2 - ↓HCO_3^-
- Acute or chronic process if respiratory disturbance present for < or>24hours.
- Appropriate compensation process presentif:
  - Metabolic Acidosis: Hyperventilation → ↑ Co_2 excretion →↓ Pco_2 → compensatory respiratory alkalosis. Formula to calculate expected PCO2. (($Pco2 = (1.5 \times HCO_3^-) + 8\pm2$)).
  - Respiratory Acidosis: : (↑ serum HCO_3^-)
  - 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 = $(Na^+ + K+) - (Cl^- + HCO_3^-)$
  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.
  Δgap = (deviation of AG from normal)– (deviation of [HCO_3] 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 HCO3- 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 academia (PH < 7.2).
- Base deficit must be calculated :
  If serum HCO3- > 10 mEq /L:
  Base deficit = Desired ΔHCO_3^- × body weight (kg) × 0.5.

Respiratory Acidosis
(CO2 retention)

Hypoventilation → Co_2 retention → ↑ Pco_2 → acidosis.
Acute respiratory acidosis: (< 24 hours):

- ↑ In HCO_3^- by 1 mEq /l for every ↑ in Pco_2 by 10 mmHg. Δ HCO_3^- = 0.1 × Δ Pco_2.

Chronic respiratory acidosis:

- ↑ In HCO_3^- by 4 mEq /l for every ↑ in Pco_2 by 10 mmHg. Δ HCO_3^- = 0.4 × Δ 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 ↑ serum HCO3 →↑ pH → compensatory hypoventilation → Co_2 retention → ↑ PCo_2.

Causes of ↑ serum HCO3 :

- Exogenous administration of HCO3, 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 twice daily) 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 → ↑Co_2 output from the lungs → ↓PCo_2 → alkalosis.

Acute respiratory alkalosis:

Serum HCO_3 ↓ by 2 meq/L for every ↓ in PCo_2 by 10 mmHg.

$\Delta HCO_3 = 0.2 \times \Delta Pco_2$.

Chronic respiratory alkalosis :

Serum HCO_3 ↓ by 5 meq/L for every ↓ in Pco_2 by 10 mmHg.

$\Delta HCO_3 = 0.5 \times \Delta Pco_2$.

Causes of respiratory alkalosis :

- ↑CNS drive for respiration:
  - Anxiety. CNS infection, infarction or trauma.
  - Drugs : salicylates, nicotine or aminophyline.
  - 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.

For further reading:

- Brenner and Rector's The Kidney, 9th Edition 2012
- National Kidney Foundation Primer on Kidney Diseases, 6th Edition 2014
- Clinical Physiology of Acid-Base and Electrolyte Disorders, 2001

Diabetic ketoacidosis (DKA)

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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 aciodosis.
  - 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 dieresis. 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 administrated only if the arterial pH is less than 6.90. 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 5.3 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 7.00.

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.

For further reading:

- Diabetic ketoacidosis and hyperosmolar hyperglycemic state in adults: Clinical features, evaluation, and diagnosis
- Diabetic ketoacidosis and hyperosmolar hyperglycemic state in adults: Treatment

Hyperosmolar hyperglycemic state

(Non ketotic hyperosmolar hyperglycemia)

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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 hypernatremia 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.

For further reading:

- Diabetic ketoacidosis and hyperosmolar hyperglycemic state in adults: Clinical features, evaluation, and diagnosis
- Diabetic ketoacidosis and hyperosmolar hyperglycemic state in adults: Treatment

CHAPTER 8
INFECTIONS AND PREVENTIVE MEASURES

Life-Threatening Infections, Diagnosis and Antimicrobial Therapy Selection
Food Poising in Hajj

Malaria

Meningitis

Pneumonia in hajj

Seasonal influenza virus infection

Middle East respiratory syndrome - Coronavirus (MERS-CoV)

Ebola Virus Disease (EVD)

Yellow fever

Hand hygiene

Life-Threatening Infections

Diagnosis and Antimicrobial Therapy Selection

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

1. **Does this patient have sepsis, sever sepsis or septic shock?**
   - Sepsis: organ dysfunction caused by a dysregulated host response to infection.
   - Severe sepsis: Sepsis with organ dysfunction, hypoperfusion, or hypotension
   - Septic shock: is a subset of sepsis in which underlying circulatory and cellular/metabolic abnormalities are profound enough to substantially increase mortality.
   - *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 choose the proper empirical antimicrobial?

History:

- Epidemiologic setting (home, recent hospital stay),
- 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?**

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 does 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.

Pneumonia (see the related article below)

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 inihibitor
- 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),
- Fluoroquinolones,
- 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.

For further reading:

The Fundamental Critical Care Support (FCCS), fifth edition, 2012, Society of Critical Care Medicine.

Food Poising in Hajj

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Food poising is not uncommon in Hajj (based on epidemiological reports) due to difficult logistics in this holy season.

Food poisoning is defined by two criteria:

- Similar illness, often gastrointestinal, in minimum of two 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.

- Movement 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-limiting, 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 has to be determined and empiric treatment (ciprofloxacin 500 mg PO bid) to be given.

For further reading:

- Differential diagnosis of microbial foodborne disease

Malaria

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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 findings:

- 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 (BinaxInc), OptiMALpLDH (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: Artisunate (AS) plus sulfadoxine-pyrimethamine (SP) *Artecospe*® 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 *Artecospe*® as non-pregnant adult above.

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

- Non- pregnant adult: Artisunate 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, Artisunate 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 *Artecospe*® 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 poqd x 14 days.

For further reading:

- Clinical manifestations of malaria
- Diagnosis of malaria
- Treatment of uncomplicated falciparum malaria
- Prevention of malaria infection in travelers
- Overview of malaria in pregnancy

Meningitis

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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 meningococcemia).
    - 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 meningococcemia (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	Ampicillinplus 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

For further reading:

- Clinical features and diagnosis of acute bacterial meningitis in adults

Initial therapy and prognosis of bacterial meningitis in adults

Pneumonia in hajj

Community-acquired pneumonia

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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. pneumoniaeor 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 **(**start as soon as possible):

- ***Previously healthy outpatients; no antibiotic use in past 3months:***
  1. Azithromycin 500 mg PO on day 1 followed by 250 mg q 24 hours on days 2-5.
     OR
  2. Clarithromycin 250 mg PO q12 hours x 7 days.
- ***Outpatients with comorbidities or antibiotic use in past three months:***
  1. Cefuroxime 500 mg PO q12 hours + Clarithromycin 500 mg PO q12 hours.
     OR
  2. Amoxicillin-clavulanate 2 g PO q12 hours + Clarithromycin 500 mg PO q12 hours.

- ***Inpatients, non-ICU:***
  1. Ceftriaxone 2 gm IV q24h + Clarithromycin 500 mg PO q 12 hours.
     OR
  2. Amoxicillin-clavulanate 1 g IV q12 hours + Clarithromycin 500 mg PO q 12 hours.
- ***Inpatients, ICU (with no risk factors for Pseudomonas species):***
  1. Ceftriaxone 1-2g IV q 24 hours + Clarithromycin 500 mg PO q 12 hours + Vancomycin IV loading dose of 25-30 mg/kg then 1g q 8 hours (the regimen should de-escalate in 48-72 hours based on culture result).
     OR
  2. Ceftriaxone 1-2g IV q 24 hours + Azithromycin 500 mg PO q 24 hours + Vancomycin IV loading dose of 25-30 mg/kg then 1g q 8 hours.
- ***Inpatients, ICU (with risk factors for Pseudomonas species):***
  1. Piperacillin/tazobactam 4.5g IV q6h + Gentamycin IV 1 mg/kg q 8 hours + Clarithromycin 500 mg PO q 12 hours.
     OR
  2. Piperacillin/tazobactam 4.5g IV q 6 hours + Gentamycin IV 1 mg/kg q 8 hours + Azithromycin 500mg PO q 12 hours.

- **Considerations:**
  - For penicillin-allergic patients, replace ceftriaxone and Piperacillin/tazobactam with Levofloxacin 750 mg IV q 24 hours.
  - Vancomycin, target trough serum concentration of 15-20 µg/mL
  - Doses above are for adults with normal renal function.
  - 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.

- **When to consider admission to hospital?**
  - Hypoxemia.
  - Confusion
  - Inability to take oral medications
  - 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.

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.

For further reading:

- Diagnostic approach to community-acquired pneumonia in adults
- Treatment of community-acquired pneumonia in adults who require hospitalization

Seasonal influenza virus infection

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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, ≥ fever 38 ^{0}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 influenza.

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: 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 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 influenza:

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

- Patients who are at high risk of influenza related complications.

Duration for antiviral chemoprophylaxis post – exposure is 10 days after the last known exposure to a confirmed influenza.

Indications for post exposure chemoprophylaxis is upon close contact with a person who is a confirmed, probable or suspected

case of influenza virus begin one day before they develop symptoms to up to 7 days after they get sick.

General guidelines for prevention / protection from influenza

- 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.

For further reading:

- Diagnosis of seasonal influenza in adults
- Treatment of seasonal influenza in adults

Middle East respiratory syndrome Coronavirus (MERS-CoV)

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Case definition:

Suspected case (patients who should be tested for MERS-CoV) Adults (> 14 years)

I. Acute respiratory illness with clinical and/or radiological, evidence of pulmonary parenchymal disease (pneumonia or Acute Respiratory Distress Syndrome).

II. A hospitalized patient with healthcare associated pneumonia based on clinical and radiological evidence. III. Upper or lower respiratory illness within 2 weeks after exposure to a confirmed or probable case of MERS-CoV infection.

IV. Unexplained acute febrile (≥38°C) illness, AND body aches, headache, diarrhea, or nausea/vomiting, with or without respiratory symptoms, AND leucopenia (WBC

Suspected case (patients who should be tested for MERS-CoV) Pediatrics (≤ 14 years)

I. Meets the above case definitions and has at least one of the following a. History of exposure to a confirmed or suspected MERS CoV in the 14 days prior to onset of symptoms b. History of contact with camels or camel products in the 14 days prior to onset of symptoms.

II. Unexplained severe pneumonia.

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 who meet definitions 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 suspected case with laboratory confirmation of MERS-CoV infection.

III. Algorithm for managing patients with suspected MERS-CoV [2]

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.
- Patients with suspected MERS-CoV who have no shortness of breath, hypoxemia, or evidence of pneumonia may be cared for and isolated in their home when suitable.

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 aerosol generating 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.
  - High-risk unprotected exposure (Contact with confirmed MERS-CoV case within 1.5 meters for > 10 minutes)
  - Low-risk unprotected exposure (Contact with confirmed MERS-CoV case more than 1.5 meters and/or for < 10 minutes):
- Contacts should also be instructed to report immediately to the Staff Health Clinic or Emergency Room if they develop upper or lower respiratory illness. Symptomatic contacts should be assessed clinically.
- Testing should be done according to the Infection Prevention and Control Guidelines for Middle East Respiratory Syndrome Coronavirus (MERS-CoV) Infection 3rd Edition June 2015.

Duration of isolation precautions for MERS-CoV infection:

- The duration of infectivity for MERS-CoV infection is unknown. However, to stop isolation, two negative deep respiratory sample is required.
- Testing for MERS-Co should be repeated after one week for improving patients in the medical ward and then every 3 days. Discontinue isolation in the hospital or the home setting if the patient is asymptomatic and a single MERS-CoV PCR test is negative. If the sample is still positive, and the patient is well enough to go home, he/she can be allowed to go home with instruction to isolate him/herself at home and come wearing a surgical mask to the clinic for follow up.

For further reading:

- Middle East respiratory syndrome coronavirus

Ebola Virus Disease (EVD)

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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^{O}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	• Antigen-capture enzyme-linked immunosorbent assay (ELISA) testing • IgM ELISA • Polymerase chain reaction (PCR) • Virus isolation
Later in disease course or after recovery	• IgM and IgG antibodies
Retrospectively in deceased patients	• 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 Ebola viruses, 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 puncture proof, 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 water-proof 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.

For further reading:

- Guidelines for Ebola Virus Disease
- Clinical manifestations and diagnosis of Ebola virus disease
- Treatment and prevention of Ebola virus disease

Yellow fever

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Yellow fever is an acute viral haemorrhagic disease transmitted by infected mosquitoes. The "yellow" in the name refers to the jaundice that affects some patients.

- **Transmission**:
  - Yellow fever virus is transmitted by mosquitoes, belonging to the Aedes and Haemogogus species.
  - The different mosquito species live in different habitats - some breed around houses (domestic), others in the jungle (wild), and some in both habitats (semi-domestic)
  - The virus is endemic in tropical areas of Africa and Central and South America.
  - Large epidemics of yellow fever occur when infected people introduce the virus into heavily populated areas with high mosquito density and where most people have little or no immunity, due to lack of vaccination. In these conditions, infected mosquitoes transmit the virus from person to person.
- **Symptoms**:
  - Fever, headache, jaundice, muscle pain, nausea, vomiting and fatigue.
  - A small proportion of patients who contract the virus develop severe symptoms and approximately half of those die within 7 to 10 days.
- **Diagnosis**:
  - Yellow fever is difficult to diagnose, especially during the early stages. More severe disease can be confused with severe malaria, leptospirosis, viral hepatitis (especially

fulminant forms), other haemorrhagic fevers, infection with other flaviviruses (e.g. dengue haemorrhagic fever), and poisoning.
    - Blood tests (RT-PCR) can sometimes detect the virus in the early stages of the disease. In later stages of the disease, testing to identify antibodies is needed (ELISA and PRNT).
- **Treatment**:
    - Good and early supportive treatment in hospitals improves survival rates.
    - There is currently no specific anti-viral drug for yellow fever but specific care to treat dehydration, liver and kidney failure, and fever improves outcomes.
- **Prevention**:
    - **Vaccination**: Yellow fever is prevented by an extremely effective vaccine, which is safe and affordable. A single dose of yellow fever vaccine is sufficient to confer sustained immunity and life-long protection against yellow fever disease and a booster dose of the vaccine is not needed. The vaccine provides effective immunity within 30 days for 99% of persons vaccinated.
    - **Mosquito control:** The risk of yellow fever transmission in urban areas can be reduced by eliminating potential mosquito breeding sites by applying larvicides to water storage containers and other places where standing water collects. Insecticide spraying to kill adult mosquitoes during urban epidemics can help reduce the number of mosquitoes, thus reducing potential sources of yellow fever transmission.

For further reading:

- Yellow fever, Fact sheet, WHO, Updated May 2016

Hand hygiene

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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).

For further reading:

- General principles of infection control.
- Infection control measures to prevent seasonal influenza in healthcare settings.
- Infection control in the outpatient setting.

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