HEART FAILURE SIMPLIFIED

by

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Heart failure: failure of the heart to act as an efficient pump and to maintain body metabolic needs

Types of heart failure:

- **According to function:**

  1- Systolic heart failure : failure to pump blood efficiently during systole القلب غير قادر علي ضخ الدم بكفاءة

  2- Diastolic heart failure : failure to relax and fill with blood during diastole القلب يفتقد القدرة علي استقبال الدم اثناء الانبساط

- **According to duration:**

  1- Acute heart failure: occurs suddenly and can be corrected with reversal of the cause e.g. Acute myocardial infarction, arrhythmias, acute mitral regurge

  2- Chronic heart failure: has long duration due to non-corrected cause e.g. cardiomyopathy

- **Anatomical :**
  1. Left side heart failure
  2. Right side heart failure

Aetiology (causes) of heart failure:

القلب عبارة عن عضلة وصمامات وشرايين وكهرباء وعليه غطاء وأي خلل شديد بهذه المكونات يؤدي الي فشل في وظائف القلب

1. **Cardiac muscle disease: e.g.** dilated cardiomyopathy, myocarditis, rheumatic carditis
2. **Valvular heart disease:** severe mitral stenosis or regurge, severe aortic stenosis or regurge, severe tricuspid regurge (leads to right side heart failure
3. **Coronary artery disease :** occluded coronary arteries lead to cardiac muscle damage (infarction) and if sizeable area is affected it may lead to heart failure or cardiogenic shock
4. **Arrhythmias:** rapid atrial fibrillation may cause hypotension and pulmonary edema. Pulseless ventricular tachycardia leads to

cardiac arrest. Severe bradycardia may lead to hypotension and may be cardiogenic shock

5. **Pericardium:** severe pericardial effusion can cause hypotension due to cardiac tamponade.

Clinical picture:

A. History of the cause

B. History of symptoms

1. **<u>Symptoms of left side heart failure:</u>**

   Dyspnea: difficulty in breathing during exercise and may be at rest in severe cases

   Orthopnea: dyspnea that occurs when the patient lies flat in bed

   Paroxysmal nocturnal dyspnea: the patient wakes up after 1-2 hours of sleep with severe cough and respiratory discomfort

   Pallor and easy fatigue

   Cyanosis may be present

   Palpitations: due to tachycardia or arrhythmias

   Syncopal attacks: especially with severe aortic stenosis or recurrent ventricular arrhythmia

2. **<u>Symptoms of right side heart failure:</u>**

   Lower limb edema

   Congested liver leads to abdominal discomfort

   Congested stomach and intestine leads to dyspepsia and refusing food

   Ascites

C. Signs of heart failure:

Pulse: tachycardia

Lips: pallor or cyanosis

Congested neck veins

Cardiac murmurs

Chest: wheezy or basal crepitation on both lungs

Abdomen: enlarged liver (congestive hepatomegaly)

Ascites may be present

Lower limb edema

Investigations:

1. Imaging
   ECG
   Chest X ray
   Echocardiography
2. Laboratory:
   Cardiac enzymes if chest pain or suspected infarction
   Electrolytes including sodium, potassium, ca, ph, Mg
   Renal functions
   Thyroid profile
   Investigation of the cause

Treatment:

a. General measures:

Oxygen if hypoxic to reach oxygen saturation to 94%
Semi-sitting position
Decrease salt intake
Morphine if chest pain due to infarction

b. Specific treatment:

Diuretics to relieve congestion and edema
Angiotensin converting enzyme inhibitors: captopril , enalapril, and ramipril to reduce cardiac afterload
Nitrates to relieve ischemia and decrease preload
Beta-blockers are added after treatment of congestion to decrease heart rate and reduce cardiac work: bisoprolol, carvedilol
Aldactone: diuretic that decreases salt and water retention

C. Treatment of the cause:

Treatment of ischemic heart with coronary angiography and stenting
Or surgery (CABG)

Treatment of metabolic problems: e.g. thyrotoxicosis, hemochromatosis

Acute respiratory distress syndrome (ARDS)

Definition: acute severe inflammation of lung parenchyma that leads to acute severe refractory hypoxia and respiratory failure

Berlin definition 2011: defines ARDS according to

1- Onset: respiratory distress within 1 week of precipitating cause (trauma, sepsis, burn or aspiration)

2- Radiology: bilateral lung infiltration in chest x ray or CT chest

3- Refractory hypoxemia: PO2/ FiO2 < 300

كل زيادة ٢٠% في نسبة الاكسجين المتنفس يوازيها زيادة ١٠٠mmHg في الاكسجين بالدم يعني لو انسان سليم وتم اعطاءه اكسجين بنسبة ٤٠% ساعتها يكون PO2 = 200 وستكون المعادلة كالآتي: 200/0.4 = 500

4- Absence of cardiac cause of pulmonary edema by echocardiography or other methods

Pathophysiology of ARDS:

الالتهاب قد يأتي من خارج الرئة (trauma and burn, sepsis)

أو من داخلها (pneumonia and aspiration)

Acute inflammation leads to activation of neutrophils. Neutrophils produce inflammatory mediators=➔ increased alveolar capillary permeability➔ alveoli become filled with fluids (non-cardiogenic pulmonary edema)

Alveolar epithelial cells when destructed with intra-alveolar fibrin deposition leading to severe hypoxemia due to impaired gas exchange

Diagnosis:

Picture of the cause

Progressive dyspnea and respiratory distress

Examinations shows bilateral crepitations on both lungs

Investigations:

Laboratory investigations:

CBC, CRP, cultures in cases of infection and sepsis

Radiological investigations:

Chest X ray

CT chest

Echocardiography

Treatment of ARDS:

A- General treatment:

1-Adequate oxygenation

2-Adeuate nutrition

3- Avoid excessive fluids to decrease systemic and lung tissue edema

4- Treatment of the cause

B- Mechanical ventilation:

Protective lung ventilation is recommended:

Low tidal volume to avoid alveolar injury

High PEEP to reach oxygen saturation ≥ 88%

Avoid high airway pressures

Prone position in severe cases: in severe cases of hypoxia with high PEEP level and high FiO2, prone position is recommended to improve hypoxia

Severe cases may require ECMO support (extra-corporeal membrane oxygenation)

الايكمو هو جهاز يتم توصيل المريض به عن طريق قسطرة وريدية ليدخل الدم عبار الجهاز ويتم تحميله بالأكسجين ثم يعود الدم مرة أخري عن طريق قسطرة وريدية أخري

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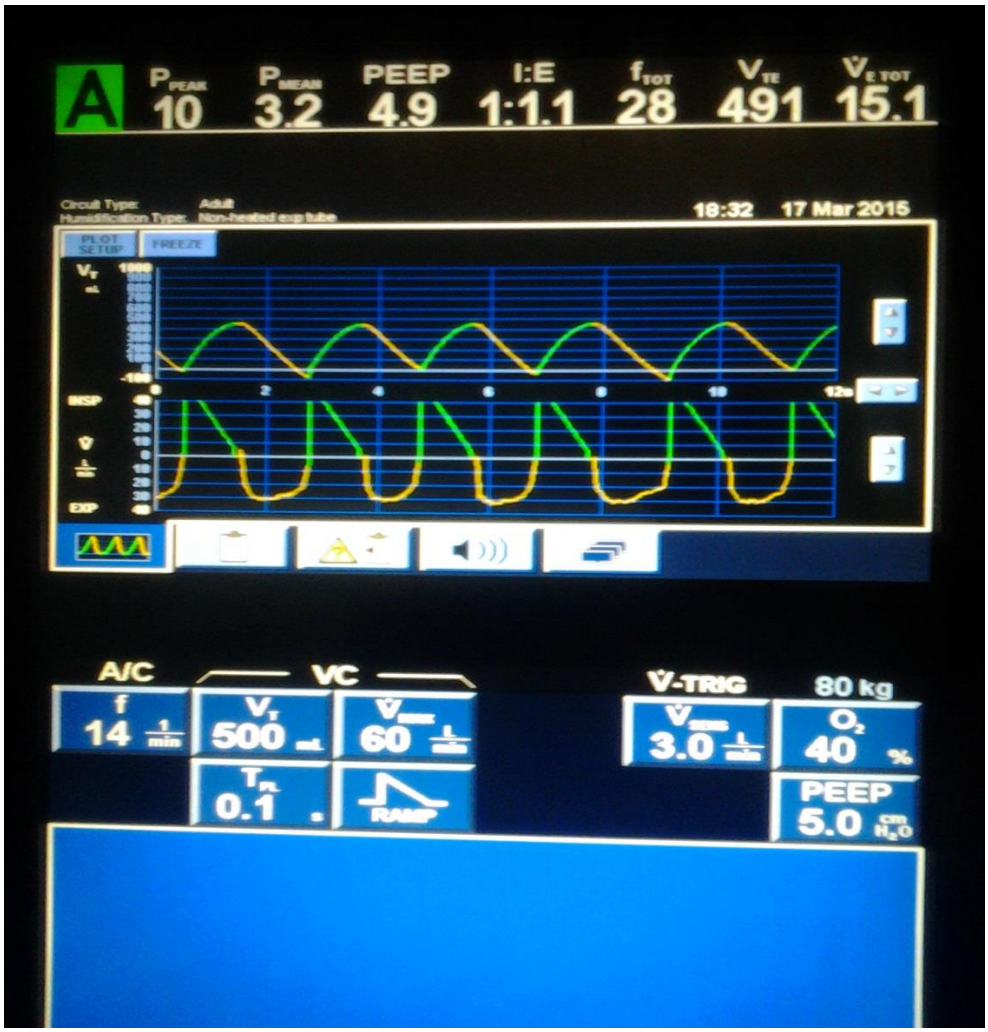

Graphic of mechanical ventilator panel in assist control mode

Respiratory failure

Definition: failure of the respiratory system to provide adequate oxygen or to eliminate CO2 or both

Most common types of respiratory failure are 2:

Type I: the patient suffers hypoxia but no hyper-capnia (usually PO2 < 60 mmHg)

Type II: the patient has hypoxia and hypercapnia (usually PCO2 > 55 mmHg)

Causes of type I respiratory failure:

a. abnormality in lung tissue:

Pneumonia

Heart failure that causes pulmonary edema

Lung fibrosis

Acute respiratory distress syndrome

b. interrupted blood flow to the lungs: pulmonary embolism

Causes of type II respiratory failure:

هات من فوق لتحت: مركزالتنفس ثم الفقرات العنقية ثم عضلات الصدر ثم الضلوع ثم الغشاء البللوري ثم نسيج الرئة

1- Respiratory center depression: due to trauma, drugs, cerebral hemorrhage, massive or brain stem stroke

2- Cervical spine trauma

3- Fracture ribs: flail chest: 2 or more ribs fractured at 2 lines

4- Pneumothorax: collection of large amount of air in the pleural space

5- Massive pleural effusion: large amount of fluid or blood in the pleural space

6- Respiratory muscle weakness or paralysis:

Myasthenia gravis: autoimmune disease that leads to severe muscle weakness

Poliomyelitis شلل الاطفال

7- Airway obstruction:

Laryngeal obstruction: due to edema, stridor or foreign body

Tubal obstruction in mechanically ventilated patient

Acute severe asthma

Chronic obstructive pulmonary disease (COPD)

7- End stage lung disease

Clinical picture:

Picture of the cause

Dyspnea

Cyanosis

Fatigue and confusion or coma

Investigations (to define the cause)

Arterial blood gases (ABG)

Chest x ray

CT chest

Echocardiography to exclude cardiac cause

CT pulmonary angiography to diagnose pulmonary embolism

Treatment:

Oxygen

Intubation and mechanical ventilation

Treatment of the cause

Acute kidney injury (AKI)

Definition: acute rise of serum creatinine (≥ 0.3 mg within 48 hrs) or decreased urine output < 0.5 ml/kg/ hr for 6 hours or more or both of conditions

Causes of AKI:

1-Pre-renal causes: due to decreased renal blood flow

كمية الدم والسوائل التي تصل إلي الكليتين لا تكفي لإرواء النسيج الكلوي وافراز البول

Dehydration

Shock

Hemorrhage

2-Renal causes: The kidney tissue includes glomeruli, renal tubules and interstitium

Glomerular disease: glomerulonephritis

Tubular damage: by drugs (vancomycin, amikacin) and toxins

Interstitial disease: interstitial nephritis by drugs like analgesics (declofenac, ibubrufen)

3-Post-renal disease:

Due to obstruction of urine flow by urine retention as in enlarged prostate or bladder neck obstruction

Clinical picture:

1-picture of the cause: dehydration, shock, drug intake

2-symptoms of uremia:

There may be headache, vomiting and hiccup

Tachypnea due to metabolic acidosis

3-Signs of volume overload: pulmonary edema, generalized edema

Investigations:

Laboratory investigations:

Urea (normally below 40 mg/dl)

Creatinine (normally 0.5-1.4 mg/dl)

Sodium, potassium, calcium, magnesium, phosphorus

Urine analysis

Urine sodium: low in dehydrated patients (pre-renal failure)

Radiological investigations:

Abdomino-pelvic ultrasound:

To detect causes of urinary tract obstruction

To examine renal tissue echogenicity

Plain x-ray urinary tract: to show urinary tract stones

CT urinary tract

Treatment: according to the cause

1-pre-renal injury:

Dehydration; give intravenous fluids

Cardiogenic shock: give vasopressors, inotropes and treatment of cardiac ischemia

Septic shock: fluids, vasopressors and antibiotics

2- Renal injury:

Stop nephrotoxic drugs

Give corticosteroids or immunosuppressive if auto-immune disease

3- Postrenal cause:

Urinary catheter to treat urine retention

Urology consultation

4- Treatment of electrolyte disturbance:

Hyperkalemia: give slow IV calcium gluconate, glucose insulin infusion

Hypernatremia: strart with intravenous bolus of 2 l saline 0.9% if still hypernatremia (Na> 150 meq/l) then give half normal saline

Hyponatremia: usually corrected with saline 0.9 %

5- Renal replacement therapy (Hemodialysis): is required when persistent life threatening one or more of the following is present:

1-persistent oliguria or anuria with volume overload
2-severe metabolic acidosis
3-Severe hyperkalemia even with giving anti-hyperkalemic treatment
4-uremic encephalopathy: coma due to effect of retained metabolites on brain functions
5-Pulmonary congestion with oliguria
6-Drug toxicity not treated with antidotes

Cardiac Arrest

This chapter presents the essential elements of cardiopulmonary resuscitation (CPR) and post-CPR care, including criteria for predicting a poor neurologic outcome after cardiac arrest. The material in this chapter is based on the most recent clinical practice guidelines on CPR from the American Heart Association.

I. BASIC LIFE SUPPORT

The essential components of basic life support (BLS) are:

(a) chest compressions

(b) airway opening (i.e., establishing a patent oropharynx)

(c) periodic lung inflations.

A. Chest Compressions

1. The original mnemonic ABC (*A*irway, *B*reathing, *C*irculation) for the components of BLS has been rearranged to CAB (*C*irculation, *A*irway, *B*reathing), reflecting the shift in emphasis to chest compressions in the resuscitation effort. The rationale for this shift was the realization that cardiac arrest is primarily a circulatory (not ventilatory) disorder.

2. The recommendations for chest compressions in the BLS guidelines are shown in Table 15.1. Early and uninterrupted chest compressions are a major emphasis of the guidelines.

B. Airway Opening

Airway opening refers to the act of establishing a patent oropharynx, which can become obstructed by a flaccid tongue in comatose patients who are supine. The “head tilt/chin up” maneuver (which hyperextends

the neck and moves the lower jaw forward) is designed to pull the tongue away from the
posterior oropharynx and relieve any obstruction from a floppy tongue

C. Ventilation

1. Prior to endotracheal intubation, ventilation can be delivered with a face mask that is connected to a self-inflating ventilation bag (e.g., Ambu bag) that fills with oxygen. The bag is compressed by hand to deliver the breath, and 2 breaths are provided for every 30 chest compressions (as in Table 15.1).
2. After an endotracheal tube is in place, lung inflations should be delivered at 6-second intervals (10 breaths /min) while chest compressions continue uninterrupted.

3. Avoid large inflation volumes and rapid rates:

Large inflation volumes are common during CPR, resulting in hyperinflation of the lungs, which can decrease cardiac filling and reduce the effectiveness of chest compressions. The recommended inflation volume during “bagged breathing” is 6–7 mL/kg , or about 500 mL for an average-sized adult.

II. ADVANCED LIFE SUPPORT

Advanced cardiovascular life support, or ACLS, includes a variety of interventions, such as

- Airway intubation
- Mechanical ventilation
- Defibrillation

- Administration of circulatory-support drugs .

Three types of cardiac arrest rhythm are known:

1- Cardiac arrests associated with ventricular fibrillation (VF) or pulseless ventricular tachycardia(VT)
2- Asystole
3- Pulseless electrical activity (PEA).

A. VF or Pulseless VT

The outcomes in cardiac arrest are most favorable when the initial rhythm is VF or pulseless VT, which are “shockable” arrhythmias.

1. Defibrillation

Electrical cardioversion is the most effective resuscitation measure for cardiac arrest associated with VF or pulseless VT. However, the survival benefit from defibrillation is *time-dependent*

a. IMPULSE ENERGY: the maximum effective energy level (about 200 J for biphasic and 360 J for monophasic shocks) should be selected for the initial shock.

2. Protocol

a. Three defibrillation attempts are allowed, if needed, using the same impulse energy.

b. After each shock is delivered, 2 minutes of uninterrupted chest compressions are advised before checking the post-shock rhythm (to prevent repeat shocks in rapid succession, which prolongs the interruption of chest compressions)

c. If a second defibrillation attempt is required, bolus injections of *epinephrine* are started (1 mg IV, or intra-osseous, every 3–5 min, for the duration of the resuscitation effort).

d. If a third defibrillation attempt is required, *amiodarone* is administered IV or intra-osseous using a dose of 300 mg, which can be followed by a second dose of 150 mg, if needed.

e. Failure to terminate VF/VT with two defibrillation attempts carries a poor prognosis.

B. Asystole or PEA (pulseless electrical activity)

The major intervention is epinephrine injections (same regimen as used for VF and pulseless VT), and there are no defibrillation attempts unless the rhythm changes to VF or VT. لا يتم إعطاء صدمة كهربائية إلا في وجود نشاط كهربي

سريع مسبب لتوقف القلب مثل ارتجاف او تسارع البطين

1. Reversible Causes of PEA

There are four potentially reversible causes of PEA, each sharing the letter T: i.e.,

Tension pneumothorax

Tamponade (raid accumulation of fluid or blood in the pericardium causing compression of cardiac chambers)

Thromboembolism of pulmonary vessels

Thrombotic occlusion of the coronary arteries (myocardial infarction)

C. ACLS Drugs

1. Epinephrine

In the doses used for cardiac arrest (1 mg IV bolus every 3–5 min), the systemic vasoconstriction it produces is intense enough to increase coronary
perfusion pressure (the difference between aortic and right atrial relaxation pressures, which occur between chest compressions). However, epinephrine also produces β-receptor-mediated cardiac stimulation, which could erase the benefit of the increased coronary perfusion. Epinephrine use is associated with an increased rate of *return to spontaneous circulation (ROSC)*, but the mortality rate is unchanged

a. INJECTION: In the rare instance when intravenous or intraosseous access is not available, epinephrine can be injected into the upper airway through an endotracheal tube. The dose for endotracheal injection is 2–2.5 times the IV dose

2. Amiodarone

Amiodarone is the preferred antiarrhythmic agent for VF/pulseless VT that is refractory to defibrillation and epinephrine (2).

3. Lidocaine

Lidocaine is the original antiarrhythmic agent used for shock-resistant VF and pulseless VT, but it is now recommended as an alternative to amiodarone.

III. POST-RESUSCITATION PERIOD

A. Post-Cardiac Arrest Syndrome

The post-cardiac arrest syndrome has 3 major features:

(a) Brain injury due to hypoxia

(b) Cardiac dysfunction which causes hypotension due to systolic and diastolic dysfunction

(c) Systemic inflammation: fever, high TLC