Acute Pulmonary Oedema

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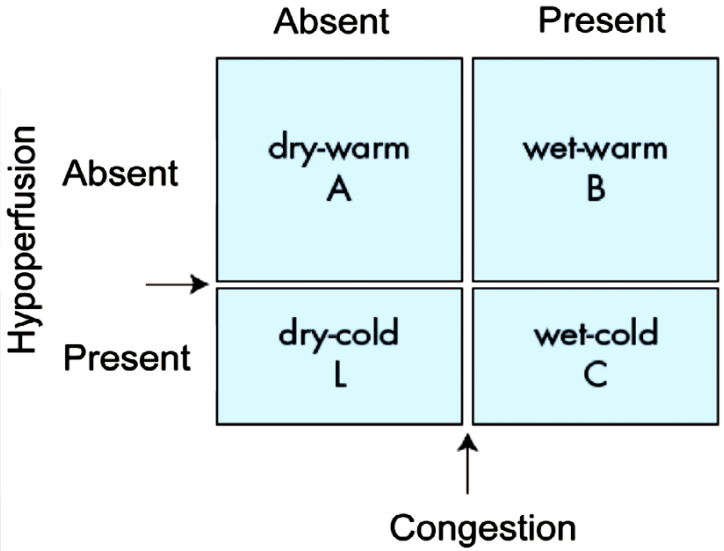
Acute Heart Failure

Rapid onset or change

It is a lifethreatening condition that requires immediate medical attention and usually leads to urgent admission to hospital.

McMurray et al,2012

Clinical Profiles of AHF Patients

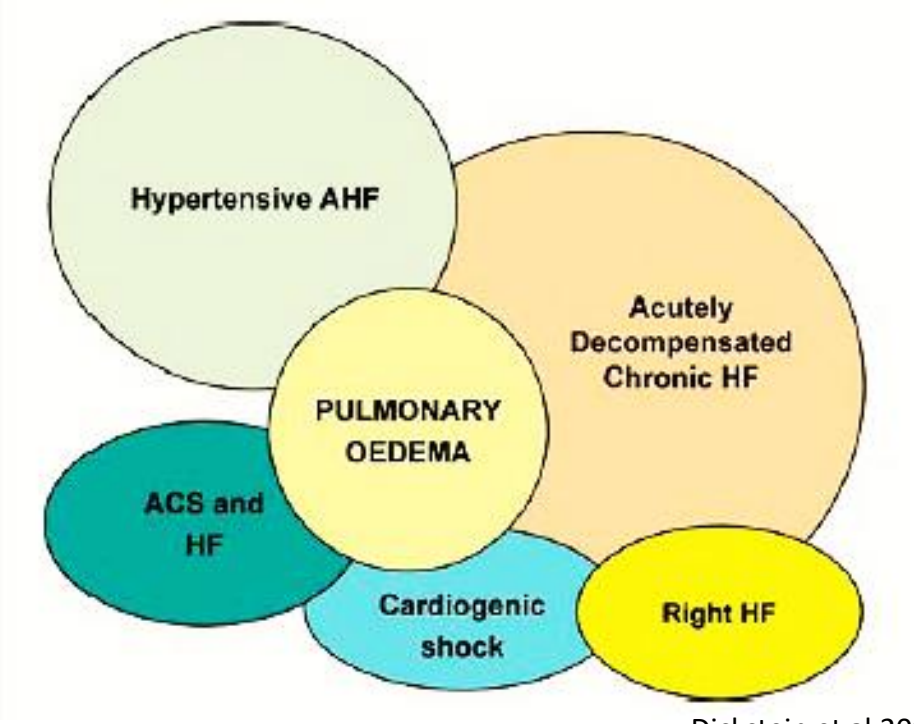


Congestion
- Orthopnea
- Increased jugular venous pressure
- Edema
- Ascites
- Hepatojugular reflux

Hypoperfusion
- Low pulse pressure
- Cold extremities
- Somnolence
- Hyponatremia
- Renal dysfunction

Izumi et al ,2012

Classification of Acute Heart Failure



Dickstein et al,2008

Acute Pulmonary Oedema

- Its a life-threatening emergency that requires immediate improvement of systemic oxygenation and elimination of the underlying cause.
- Patients present with :
  - Severe respiratory distress, tachypnoea, and orthopnoea.
  - Rales over the lung fields.
  - Arterial O2 saturation is usually < 90% on room air prior to treatment with oxygen.

Dickstein et al,2008

- On examination, the patient is tachycardic and may demonstrate cold, clammy skin
- Coughing of “frothy” sputum represent transudation of fluid into the alveoli

Leonard S.Lily,2011

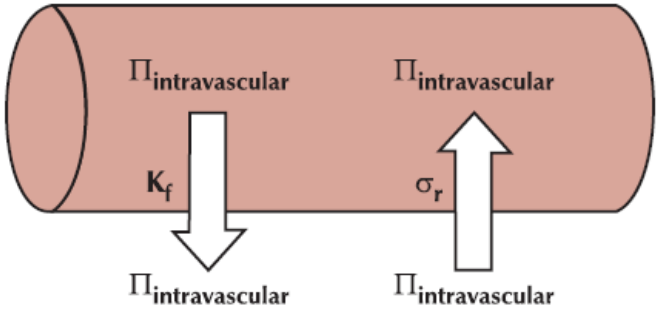
Elements should be considered of importance in the production of pulmonary oedema :

- High Pressure in the Pulmonary Capillaries.
- Increased Permeability of the Pulmonary Capillaries.
- Decreased Osmotic Pressure of the Blood.

Leonard S.Lily,2011

Frank-Starling Law

$$Q_{iv\text{-}int} = K_f(P_{intravascular} - P_{interstitial}) - \sigma_r(\Pi_{intravascular} - \Pi_{interstitial})$$



Margulescu,2007

Classification of Pulmonary Oedema

- **Cardiogenic pulmonary oedema** (hydrostatic or hemodynamic oedema)
- **Non-cardiogenic pulmonary oedema** (increased-permeability pulmonary oedema, acute lung injury, or acute respiratory distress syndrome).

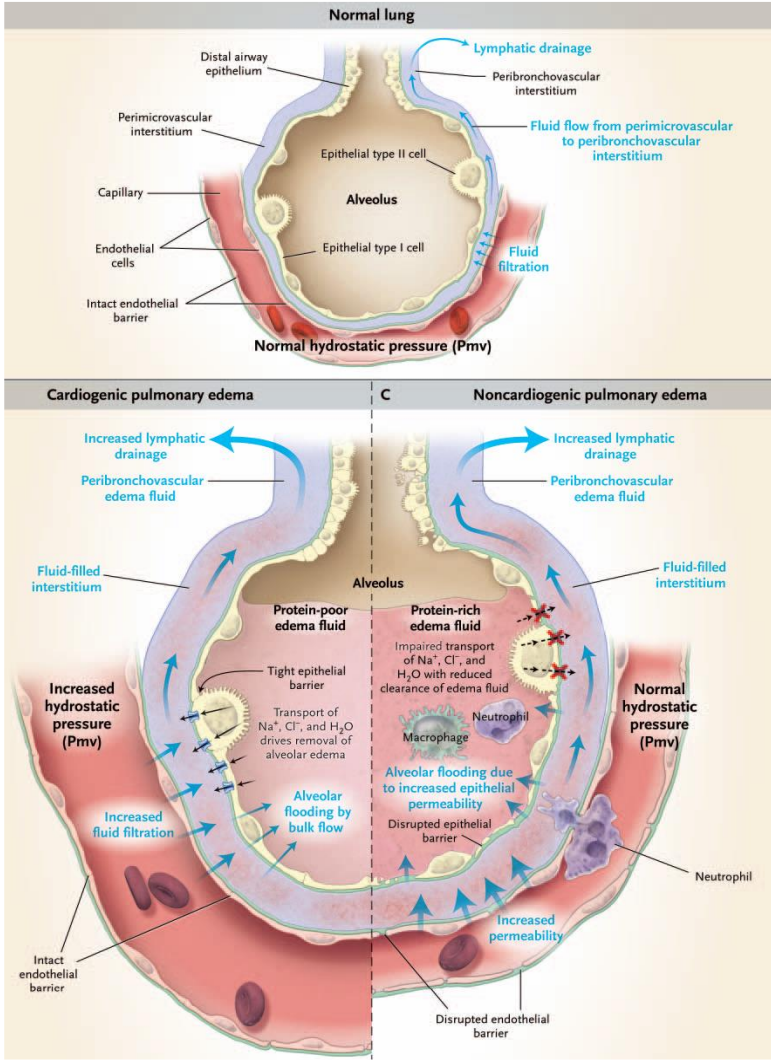
Although they have distinct causes, cardiogenic and non-cardiogenic pulmonary oedema may be difficult to distinguish because of their similar clinical manifestations.

Lorraine et al,2010

Differences of Cardiogenic and Non-Cardiogenic Acute Pulmonary Oedema

Jeremias et al,2010

	Noncardiogenic	Cardiogenic
History	Underlying disease (e.g., pancreatitis, sepsis)	Acute cardiac event (e.g., myocardial infarction)
Physical examination	Warm periphery Bounding pulses Normal-sized heart Normal JVP No S_3 No murmurs	Cool, mottled periphery Small-volume pulse Cardiomegaly Elevated JVP S_3 Systolic and diastolic murmurs
ECG	ECG usually normal	ST segment and QRS abnormalities
Chest x-ray film	Peripheral infiltrates	Perihilar infiltrates
Laboratory test	Normal enzymes BNP <100 mg/mL	Elevated biomarkers
Ventilatory needs	Higher F_{IO_2} and PEEP to oxygenate	Lower F_{IO_2} and PEEP to oxygenate



Etiology

Jeremias et al,2010

Cardiogenic Pulmonary Edema

A. Acute Increase in Pulmonary Capillary Pressure

1. Increased LA pressure with normal LV diastolic pressure
   a. Thrombosed prosthetic mitral valve
   b. Obstructive left atrial myxoma
2. Increased LA pressure owing to elevated LV diastolic pressure
   a. Acute increases in myocardial stiffness or impaired relaxation
      i. Myocardial ischemia
      ii. Acute myocardial infarction
      iii. Hypertrophic heart disease complicated by tachycardia or ischemia
   b. Acute volume load
      i. Acute mitral or aortic regurgitation
      ii. Ischemic septal rupture
   c. Acute pressure load
      i. Hypertensive crisis
      ii. Thrombosed prosthetic aortic valve

B. Exacerbation of Chronically Elevated Pulmonary Capillary Pressures

1. Increase in elevated LA pressure with normal LV diastolic pressure
   a. Mitral stenosis
   b. Left atrial myxoma
2. Increase in elevated LA pressure owing to a further increase in LV diastolic pressure
   a. Further increases in myocardial stiffness or impaired relaxation
      i. Cardiomyopathy complicated by myocardial ischemia or infarction
      ii. Hypertrophic heart disease complicated by tachycardia or ischemia
   b. Volume load imposed on preexisting LV diastolic dysfunction
      i. Worsening mitral regurgitation
      ii. Vigorous postoperative fluid administration
      iii. Dietary indiscretion
   c. Pressure load imposed on preexisting LV systolic dysfunction
      i. Accelerated hypertension

Noncardiogenic Pulmonary Edema

A. Altered Alveolar Capillary Membrane Permeability (Adult Respiratory Distress Syndrome)

1. Infectious or aspiration pneumonia
2. Septicemia
3. Acute radiation or hypersensitivity pneumonitis
4. Disseminated intravascular coagulopathy
5. Shock lung
6. Hemorrhagic pancreatitis
7. Inhaled and circulating toxins
8. Massive trauma

B. Acute Decrease in Interstitial Pressure of the Lung

1. Rapid removal of unilateral pleural effusion

C. Unknown Mechanisms

1. High-altitude pulmonary edema
2. Neurogenic pulmonary edema
3. Narcotic overdose
4. Pulmonary embolism
5. After cardioversion
6. After anesthesia or cardiopulmonary bypass

Radiological Classification

- **Interstitial**

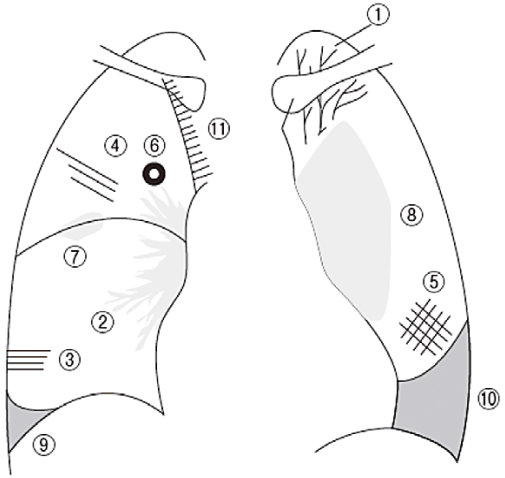
  Oedematous interlobular septa with associated dilated lymph vessels become radiologically recognizable as Kerley A, Band C lines

- **Intraalveolar**

  as fluid accumulates in the alveoli the hilar and basal lung regions assume a finely granular appearance

- **Mixed interstitial and intra-alveolar**

Beyer,1978



① Cephalization: Redistribution of pulmonary blood flow toward the apex (pulmonary venous pressure, 15 to 20 mmHg)
② Perivascular cuffing: Indicating interstitial pulmonary edema (pulmonary venous pressure, 20 to 30 mmHg)
③ Kerley B lines: Indicating interstitial pulmonary edema (pulmonary venous pressure, 20 to 30 mmHg)
④ Kerley A lines: Indicating interstitial pulmonary edema (pulmonary venous pressure, 20 to 30 mmHg)
⑤ Kerley C lines: Indicating interstitial pulmonary edema (pulmonary venous pressure, 20 to 30mmHg)
⑥ Peribronchial cuffing: Indicating interstitial pulmonary edema (pulmonary venous pressure, 20 to 30 mmHg)
⑦ Vanishing tumor: Indicating alveolar pulmonary edema (pulmonary venous pressure, ≥30 mmHg)
⑧ Butterfly shadow: Indicating alveolar pulmonary edema (pulmonary venous pressure, ≥30 mmHg)
⑨⑩ Costophrenic angle blunting: Pleural effusion
⑪ Extrusion of the superior vena cava

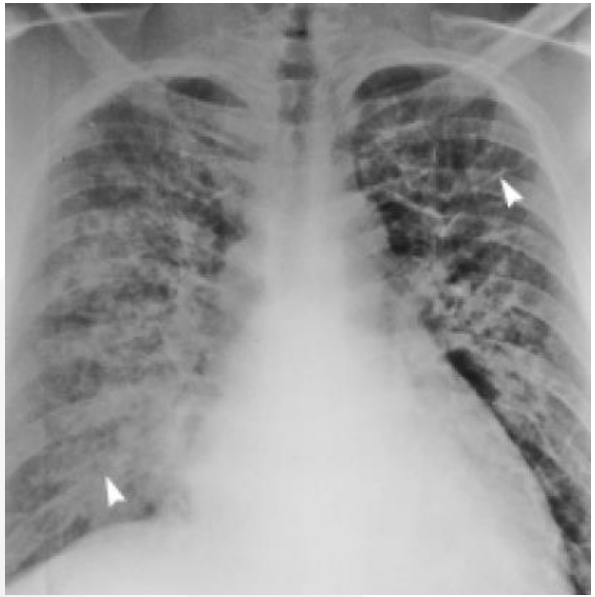
Japanesse Circulation Society,2011

Kerley B lines the best known, most commonly seen, They appear as short, thin sharply defined hairline shadows situated horizontally and extending perpendicularly to the pleural surface, they are most numerous in the lower lung fields, especially in the costophrenic sulci. They are due to oedematous interlobular septa and dilated lymph vessels in the lung periphery

Kerley A lines are longer, somewhat angular hairline opacities best seen in the upper and mid-lung zones and extending towards the hili. They are caused by oedematous interlobular and intersegmental septa and dilated lymph vessels situated more centrally in the lungs.

Kerley C lines are only rarely seen and appear as a fine reticular pattern probably due to superimposition of fine oedematous interlobular septa

Beyer,1978



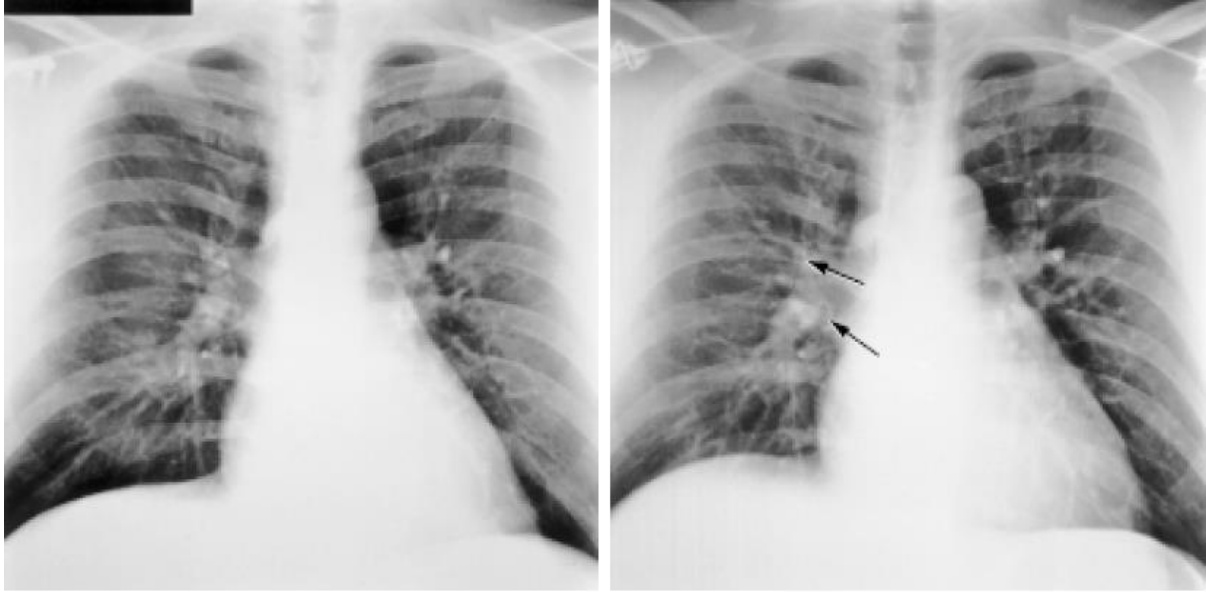


Figure 1. Increased hydrostatic pressure edema in a 33-year-old man with acute myelocytic leukemia who was admitted for fluid overload with renal and cardiac failure. Successive chest radiographs demonstrate progressive lobar vessel enlargement, peribronchial cuffing (arrows in **b**), bilateral Kerley lines (arrowheads in **c**), and late alveolar edema with nodular areas of increased opacity. The fluid overload is confirmed by the increasing size of the azygos vein.

Gluecker,1999

Bat Wing & Kerley B

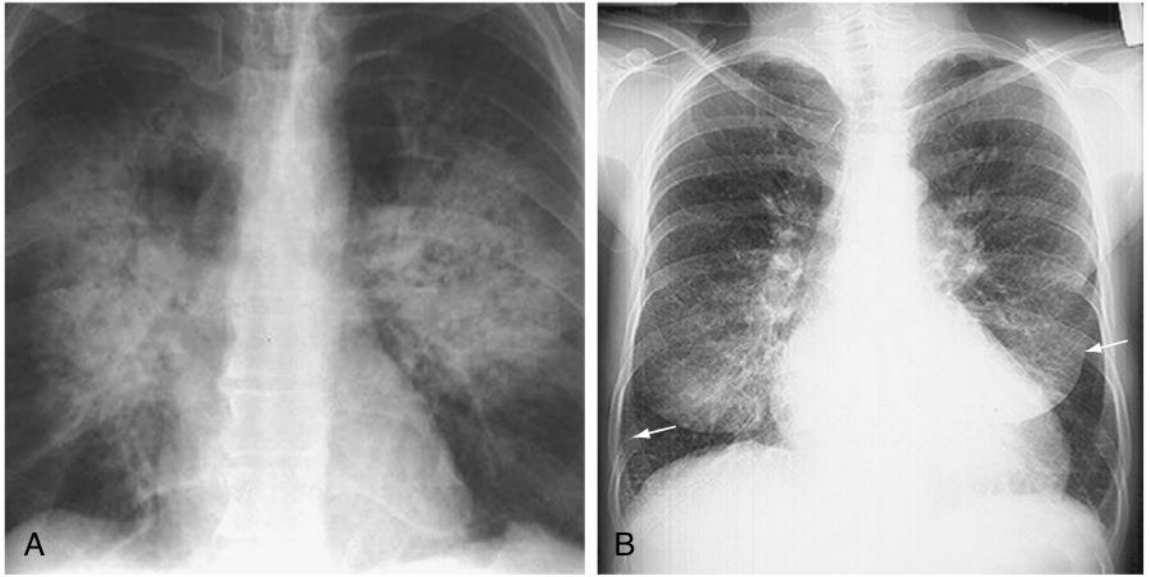
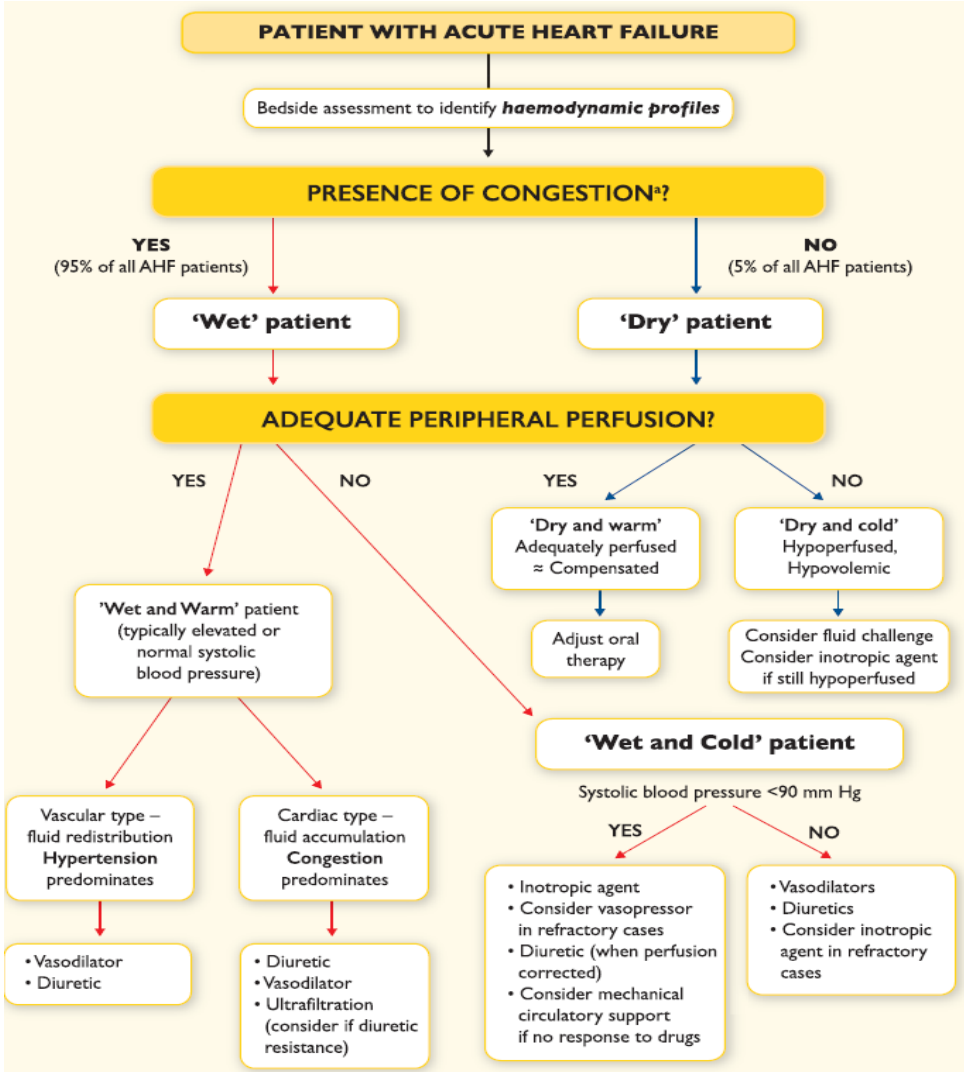


Figure 24-6. Chest x-rays of two patients. **A,** The classic features of acute cardiogenic pulmonary edema. Notice the perihilar alveolar infiltrates. **B,** Marked interstitial changes in the lung bases. Note the Kerley B lines (*arrow*).

Jeremias et al,2010

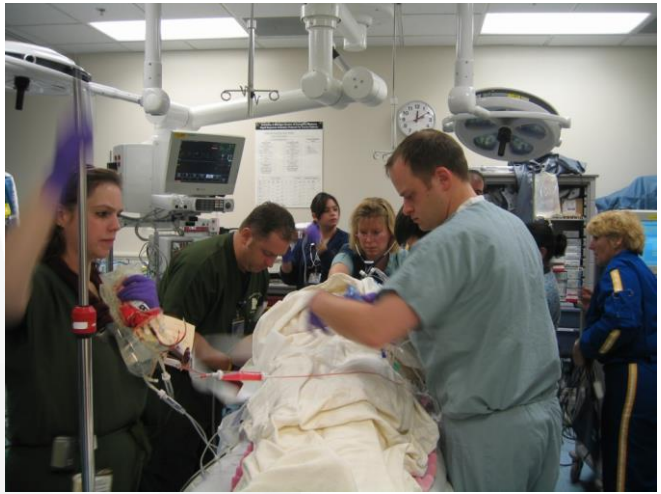
Management of AHF Patients in Early Phase

Ponikowski et al,2016



Treatment of Acute Heart Failure

from Acute to Stabilization Phase



Acute Phase Management

Oxygen

- Oxygen may be given to treat hypoxaemia (SpO2 < 90%, PaO2 <60 mmHg (8.0 kPa)

Diuretic

- The high dose strategy was associated with greater improvement in a number of secondary outcomes (including dyspnoea) but cause more transient worsening of renal function
- Combination between loop diuretic, thiazide for diuretic resistant

McMurray et al,2012

Clinical Scenario	Diuretic	Dose	Goal/Comments
Moderate fluid overload	Furosemide	20-40 mg IV q12h*	Urine output of >200 mL in the first 2 hr after bolus dose
	Bumetanide	0.5-1 mg IV q12h*	
Severe fluid overload	Furosemide	40-80 mg IV q12h† ‡ *or* Bolus of 80 mg IV + continuous infusion at 10-20 mg/hr	Urine output of >400 mL in the first 2 hr after bolus dose and then 150 mL/hr
	Bumetanide	1-2 mg IV q12h†	
Severe fluid overload *and* renal dysfunction (GFR <30 mL/min)	Furosemide	80-200 mg IV q12h *or* Bolus + continuous infusion at 20-40 mg/hr	Urine output of >200 mL in the first 2 hr after bolus dose and then 100 mL/hr
Diuretic resistance	Add chlorothiazide to furosemide	250-500 mg IV 30 min before loop diuretic	Urine output of >200 mL in the first 2 hr after bolus dose and then 100 mL/hr
	Acetazolamide§	0.5 mg IV q12h	

Jeremias et al,2010

Vasodilators

- Reduce preload, afterload & increase stroke volume
- They may relieve dyspnoea or improve other clinical outcomes
- Most useful in patients with hypertension

They should be avoided in patients with a SBP <110 mmHg & used with caution in patients with significant mitral or aortic stenosis

Vasodilator	Dosing	Main side effects	Other
Nitroglycerine	Start with 10–20 µg/min, increase up to 200 µg/min	Hypotension, headache	Tolerance on continuous use
Isosorbide dinitrate	Start with 1 mg/h, increase up to 10 mg/h	Hypotension, headache	Tolerance on continuous use
Nitroprusside	Start with 0.3 µg/kg/min and increase up to 5 µg/kg/min	Hypotension, isocyanate toxicity	Light sensitive
Nesiritide[a]	Bolus 2 µg/kg + infusion 0.01 µg/kg/min	Hypotension	

Opiates (Morphine)

- They reduce anxiety and relieve distress associated with dyspnoea in ALO
- Venodilators, reducing preload, and may also reduce sympathetic drive

Potential adverse effect

Induce nausea & depress respiratory drive, potentially increasing the need for invasive ventilation.

McMurray et al,2012

	Bolus	Infusion rate
Dobutamine	No	2–20 µg/kg/min (β+)
Dopamine	No	<3 µg/kg/min: renal effect (δ+)
		3–5 µg/kg/min; inotropic (β+)
		>5 µg/kg/min: (β+), vasopressor (α+)
Milrinone	25–75 µg/kg over 10–20 min	0.375–0.75 µg/kg/min
Enoximone	0.5–1.0 mg/kg over 5–10 min	5–20 µg/kg/min
Levosimedan[a]	12 µg/kg over 10 min (optional)[b]	0.1 µg/kg/min, which can be decreased to 0.05 or increased to 0.2 µg/kg/min
Norepinephrine	No	0.2–1.0 µg/kg/min
Epinephrine	Bolus: 1 mg can be given i.v. during resuscitation, repeated every 3–5 min	0.05–0.5 µg/kg/min

Inotropes and Vasopressors

- They are given to severe marked hypotension to raise blood pressure and redistribute cardiac output from the extremities to the vital organs.
- Should be restricted to patients with persistent hypoperfusion despite adequate cardiac filling pressures.

McMurray et al,2012

After Stabilization Management

ACE inhibitor/ ARB

- Should be started as soon as possible after stabilization, blood pressure and renal function permitting
- Uptitration as far before discharge

Beta-blocker

- Should be started as soon as possible after stabilization, blood pressure and heart rate permitting
- Uptitration as far before discharge

Mineralocorticoid (aldosterone) receptor antagonist

Digoxin

Non-Pharmacological Treatment

Restrict fluid & sodium intake

Sodium < 2 g/day and fluid intake to < 1.5–2.0 L/day

Ventilation

Non-invasive or invasive ventilation support

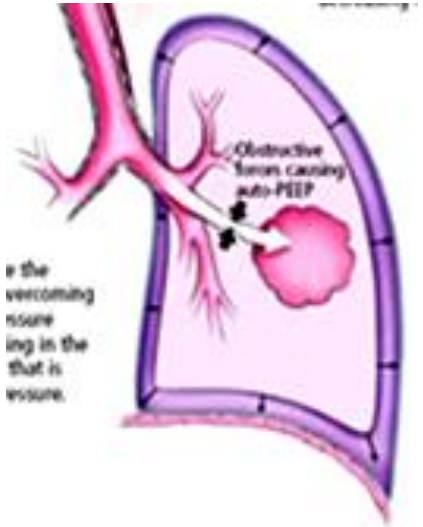
Mechanical circulatory support

- Intra aortic balon pump
- Ventricular assist device
- Ultrafiltration

McMurray et al,2012

Continous Positive Airway Pressure (CPAP)

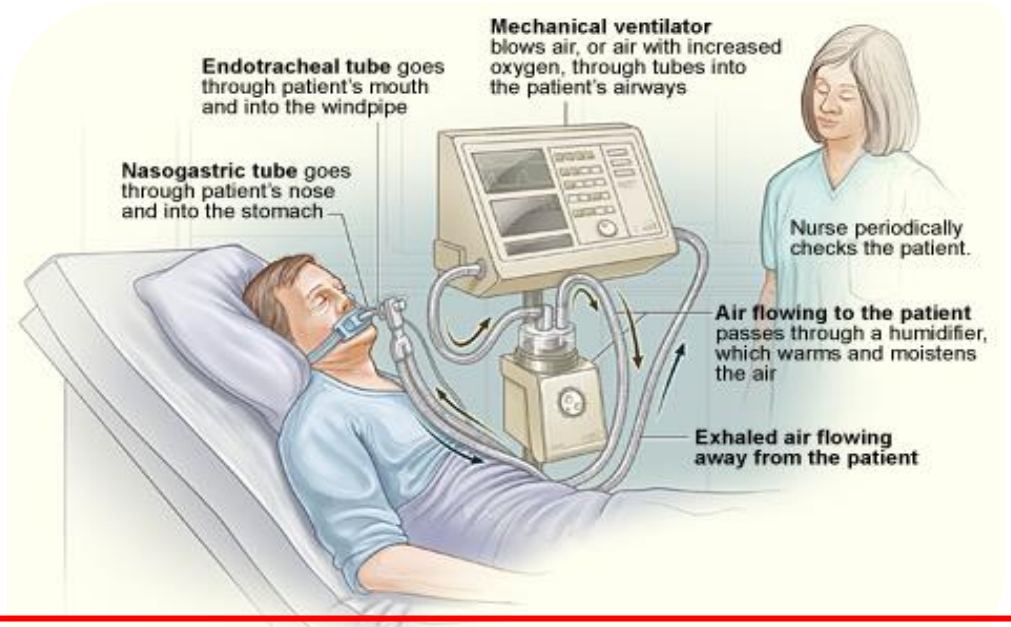
Non-invasive ventilation including CPAP may be used as adjunctive therapy to relieve symptoms in patients with pulmonary oedema and severe respiratory distress or who fail to improve with pharmacological therapy.



Contraindications include hypotension vomiting, possible pneumothorax, and depressed consciousness

McMurray et al,2012

Mechanical Ventilator



The primary indication for endotracheal intubation and invasive ventilation is respiratory failure leading to hypoxaemia, hypercapnia, and acidosis. Physical exhaustion, diminished consciousness, and inability to maintain or protect the airway are other reasons to consider intub-ation and ventilation

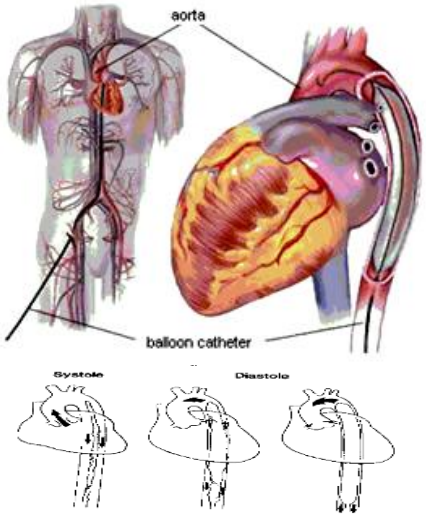
McMurray et al,2012

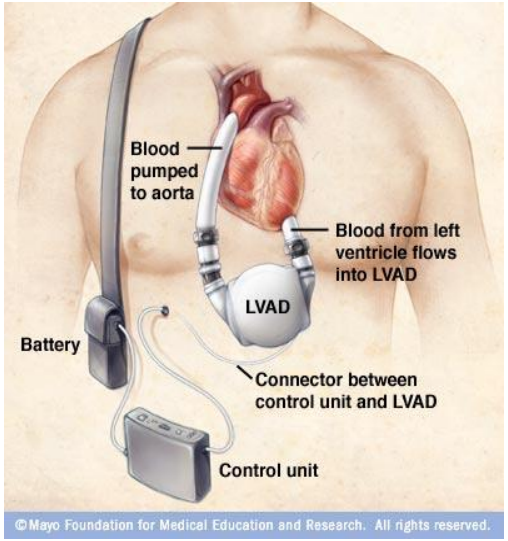
Mechanical circulatory support

Intra Aortic Balon Pump (IABP)

Left Ventricular Assist Device (LVAD)

Ultrafiltration





- Ventricular assist devices and other forms of mechanical circulatory support (MCS) may be used as a ‘bridge to decision’ or longer term in selected patients.
- Ultrafiltration is sometimes used to remove fluid in patients with HF although is usually reserved for those unresponsive or resistant to diuretics.

McMurray et al,2012

Monitoring After Stabilization

- Heart rate, rhythm, blood pressure, and oxygen saturation
- Symptoms relevant to HF (e.g. dyspnoea)
- Fluid intake and output, weight, and the jugular venous pressure and extent of pulmonary and peripheral oedema (and ascites if present)
- Blood urea nitrogen, creatinine, potassium, and sodium

McMurray et al,2012

Thank You