Acute Cardiogenic Pulmonary Edema

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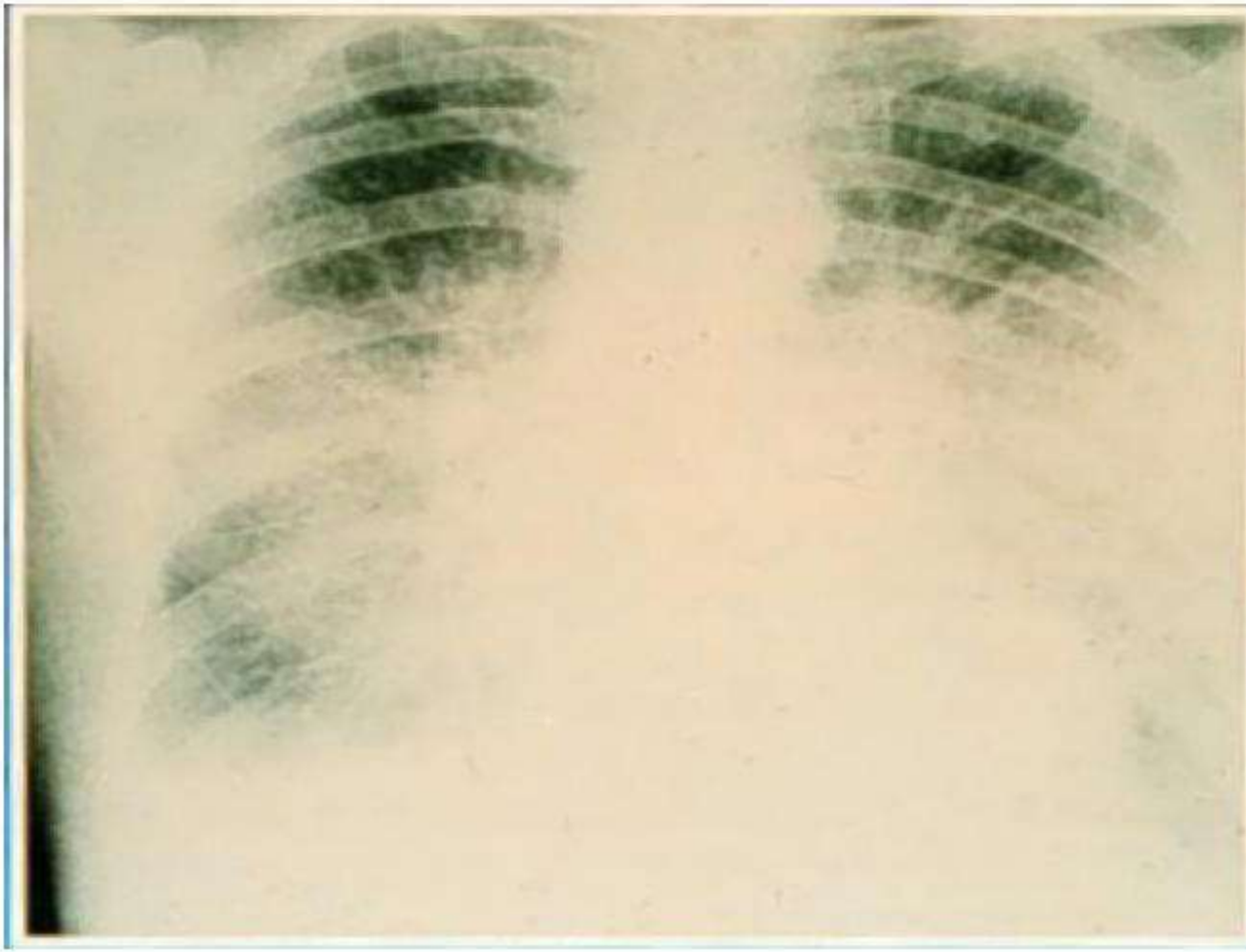
2022

SEVERE ACUTE CARDIOGENIC PULMONARY EDEMA



CardiogenicPulmory Edema:Introduction

- **Cardiogenic Pulmonary edema** is a common and potentially fatal cause of acute heart and respiratory failure.
- The clinical presentation is characterized by the development of **dyspnea** associated with rapid accumulation of **fluid** within the lung's **interstitial and/or alveolar spaces,** which is the result of acutely elevated cardiac filling pressures.
- "**Flash**" pulmonary edema is a term that is used to describe a particulary dramatic form of cardiogenic alveolar pulmonary edema.
  Often,"flash" pulmonary edema is related to a sudden rise in left-sided intracardiac filling pressures in the setting of **hypertensive emergency, acute ischemia, new onset tachyarrhithmia, or obstructive valvular disease.**

Figure 1: Clinical Presentations of Acute Heart Failure

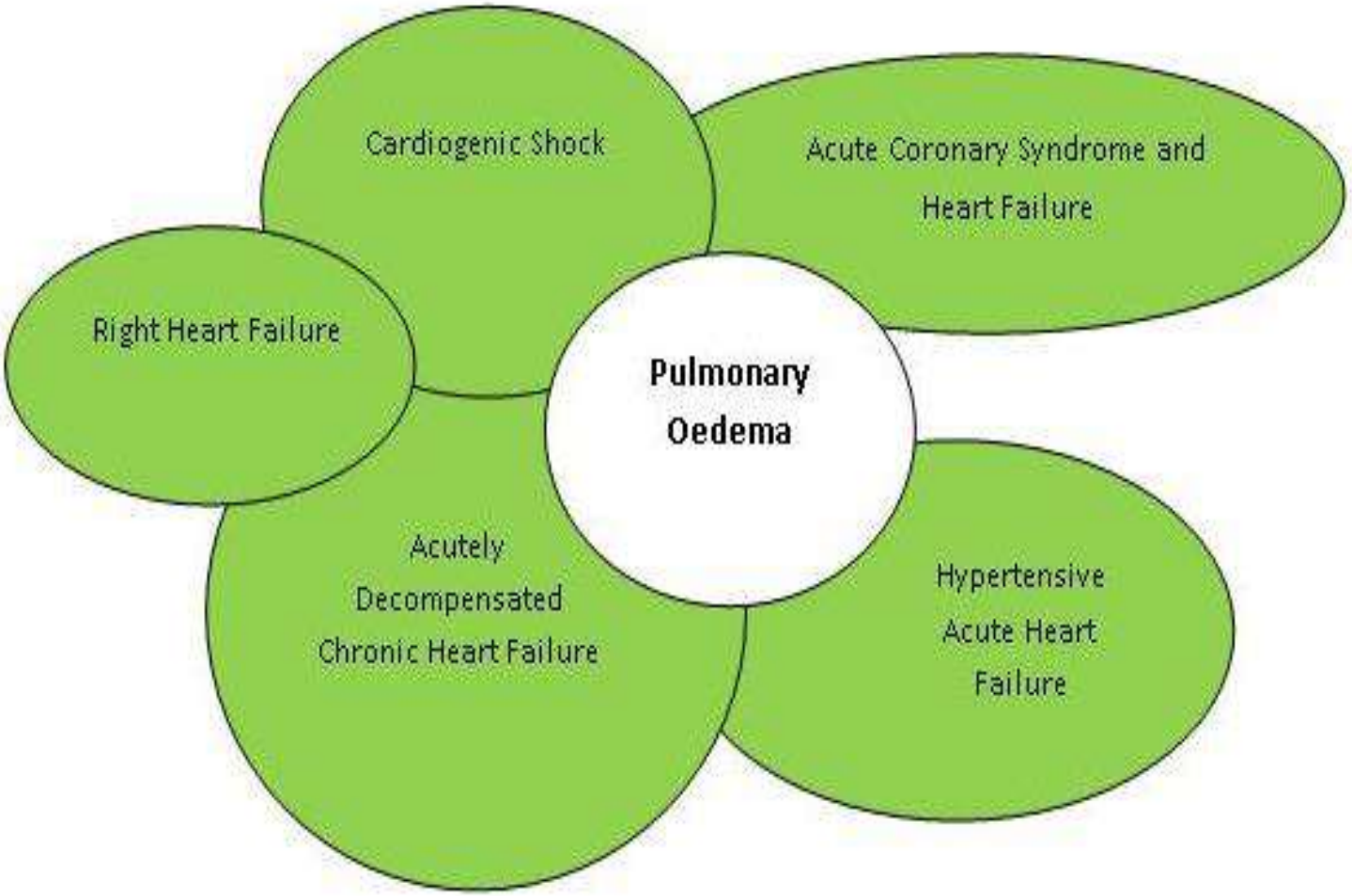
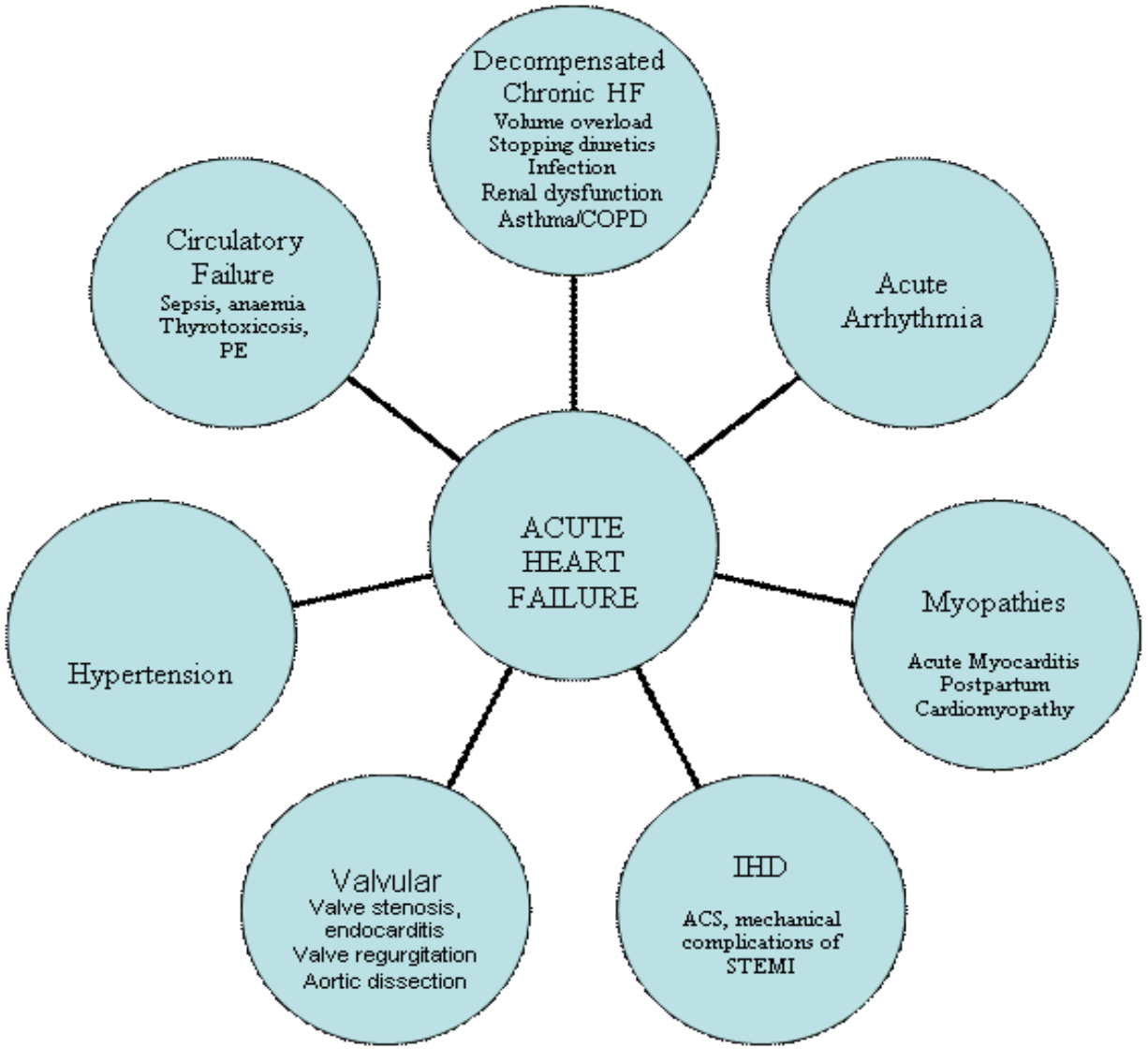


Figure 3: Precipitating Causes of Acute Heart Failure



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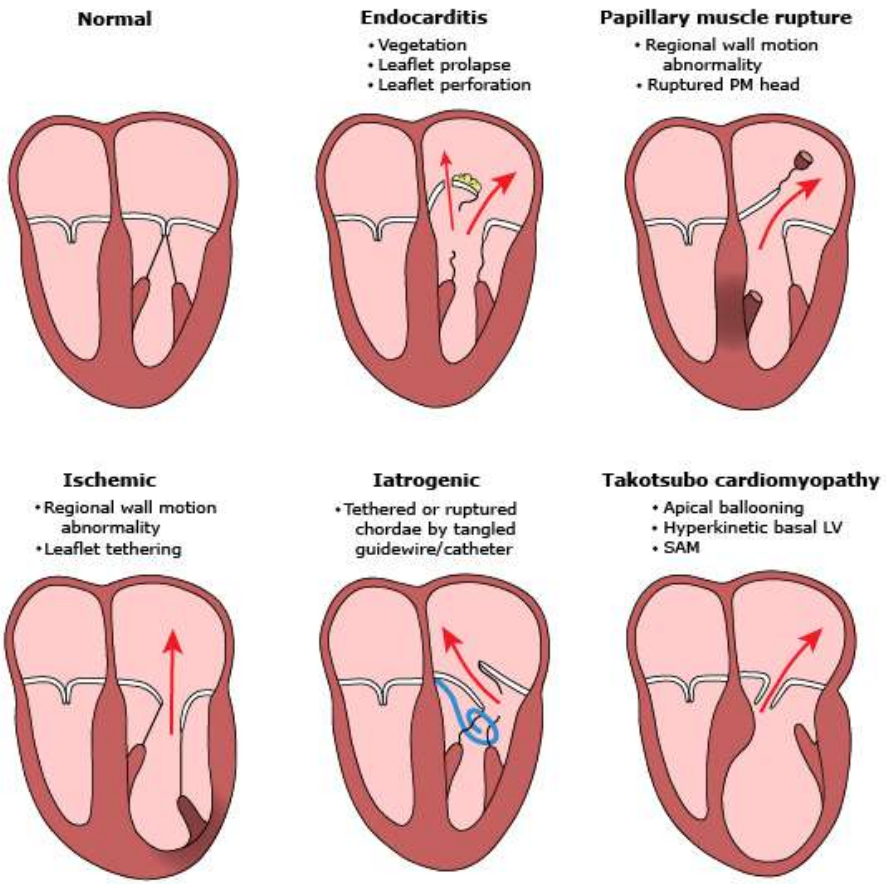
Schematic diagrams of causes of acute mitral regurgitation



MR: mitral regurgitation; PM: papillary muscle; LV: left ventricle; SAM: systolic anterior motion.

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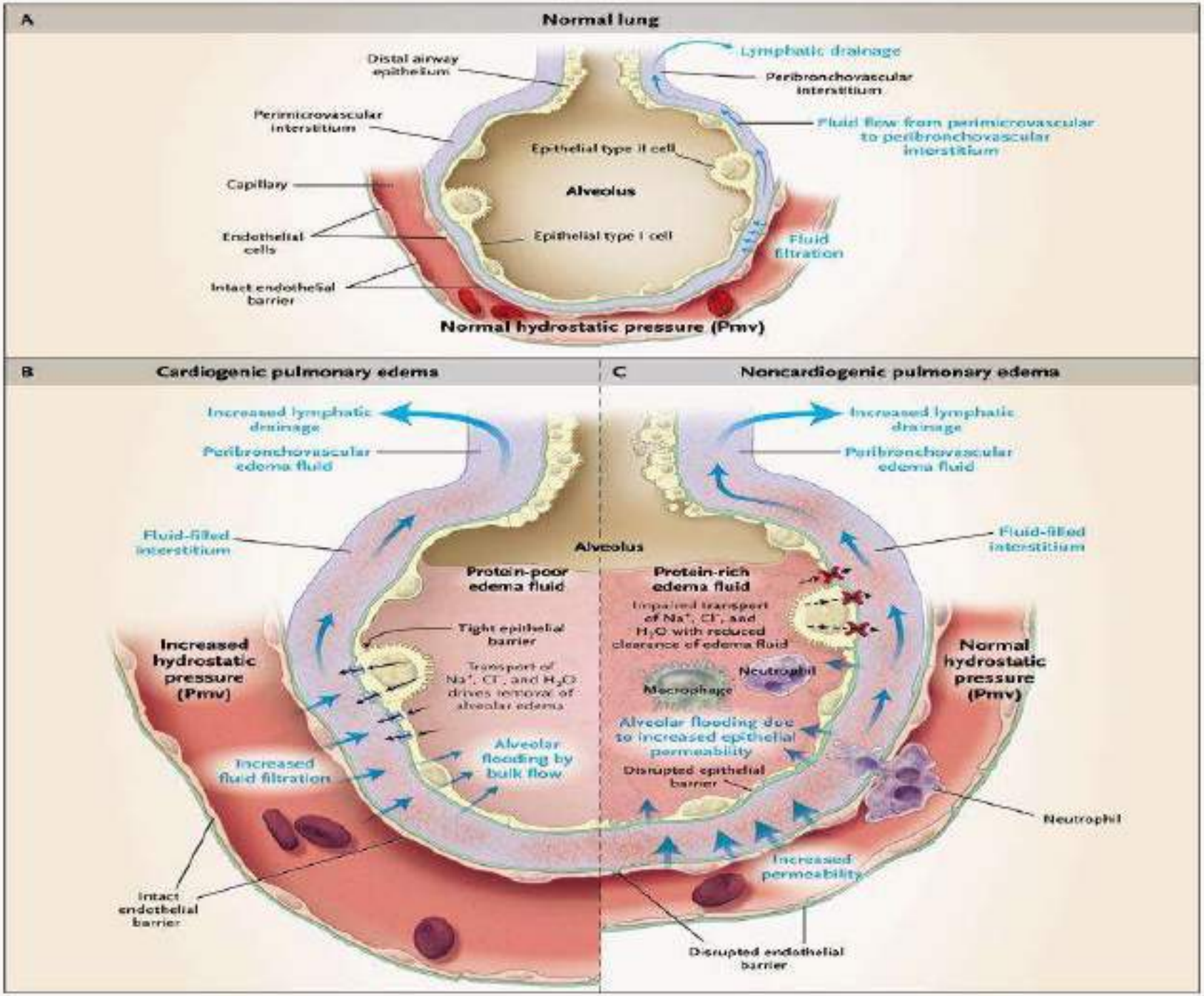
A
Normal lung
Distal airway epithelium
Lymphatic drainage
Peribronchovascular interstitium
Perimicrovascular interstitium
Fluid flow from perimicrovascular to peribronchovascular interstitium
Epithelial type II cell
Alveolus
Capillary
Endothelial cells
Epithelial type I cell
Fluid filtration
Intact endothelial barrier
Normal hydrostatic pressure (Pmv)
B
Cardiogenic pulmonary edema
Increased lymphatic drainage
Peribronchovascular edema fluid
Fluid-filled interstitium
Alveolus
Protein-poor edema fluid
Tight epithelial barrier
Transport of Na+, Cl−, and H2O drives removal of alveolar edema
Increased hydrostatic pressure (Pmv)
Alveolar flooding by bulk flow
Increased fluid filtration
Intact endothelial barrier
C
Noncardiogenic pulmonary edema
Increased lymphatic drainage
Peribronchovascular edema fluid
Fluid-filled interstitium
Protein-rich edema fluid
Impaired transport of Na+, Cl−, and H2O with reduced clearance of edema fluid
Neutrophil
Macrophage
Normal hydrostatic pressure (Pmv)
Alveolar flooding due to increased epithelial permeability
Disrupted epithelial barrier
Neutrophil
Increased permeability
Disrupted endothelial barrier

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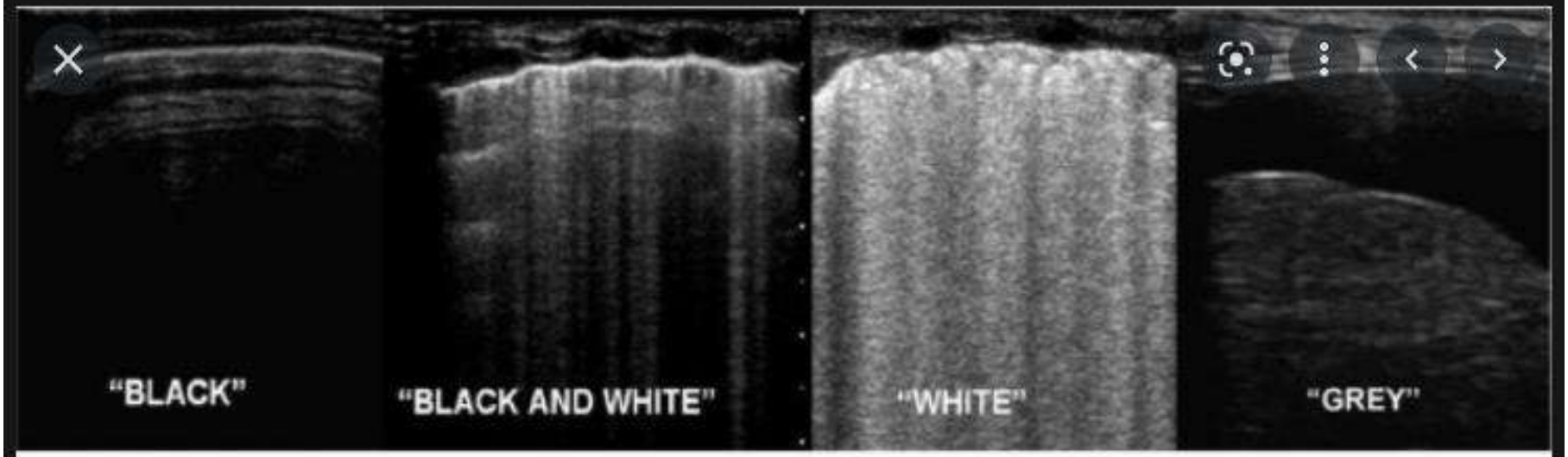

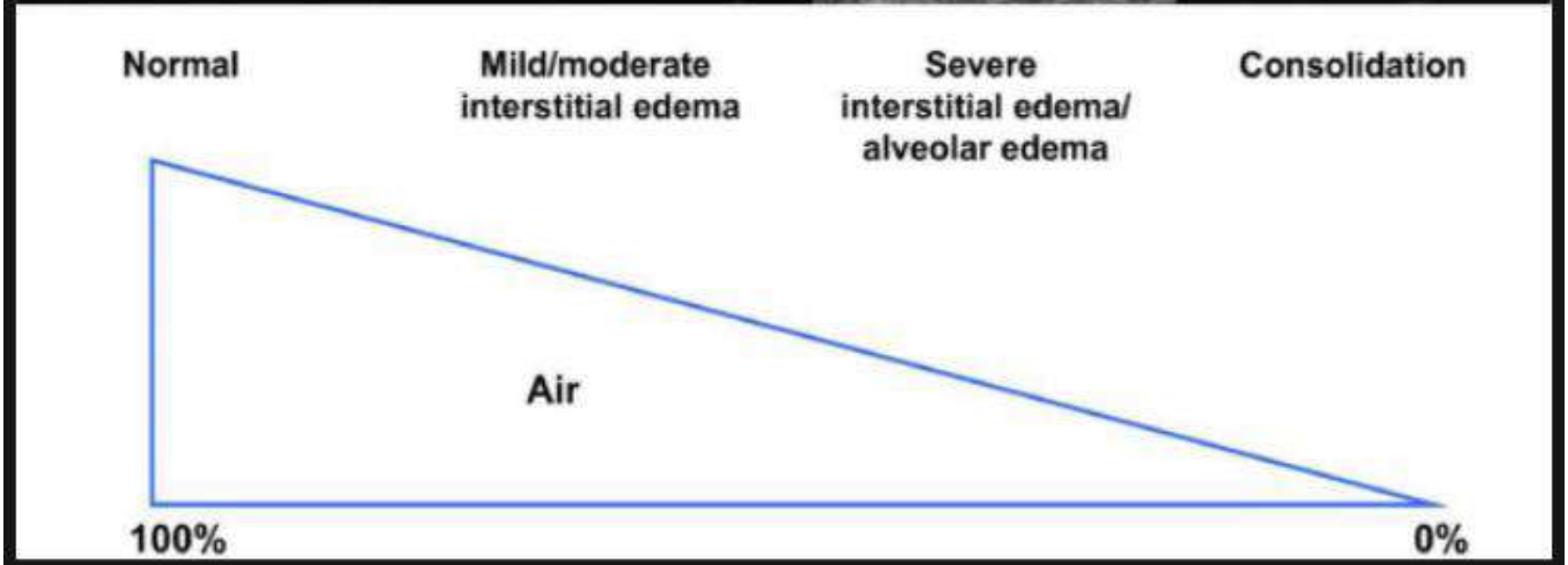

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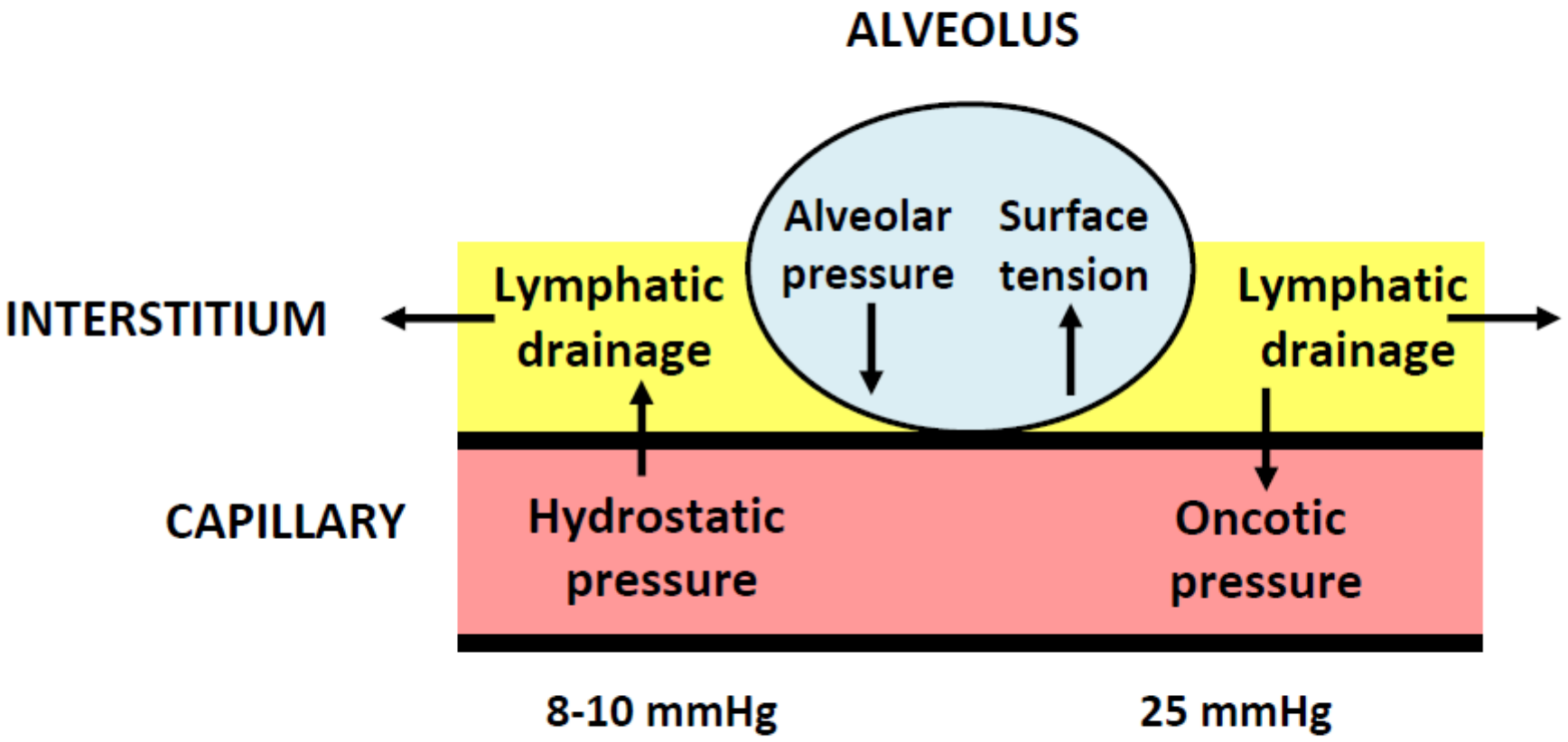
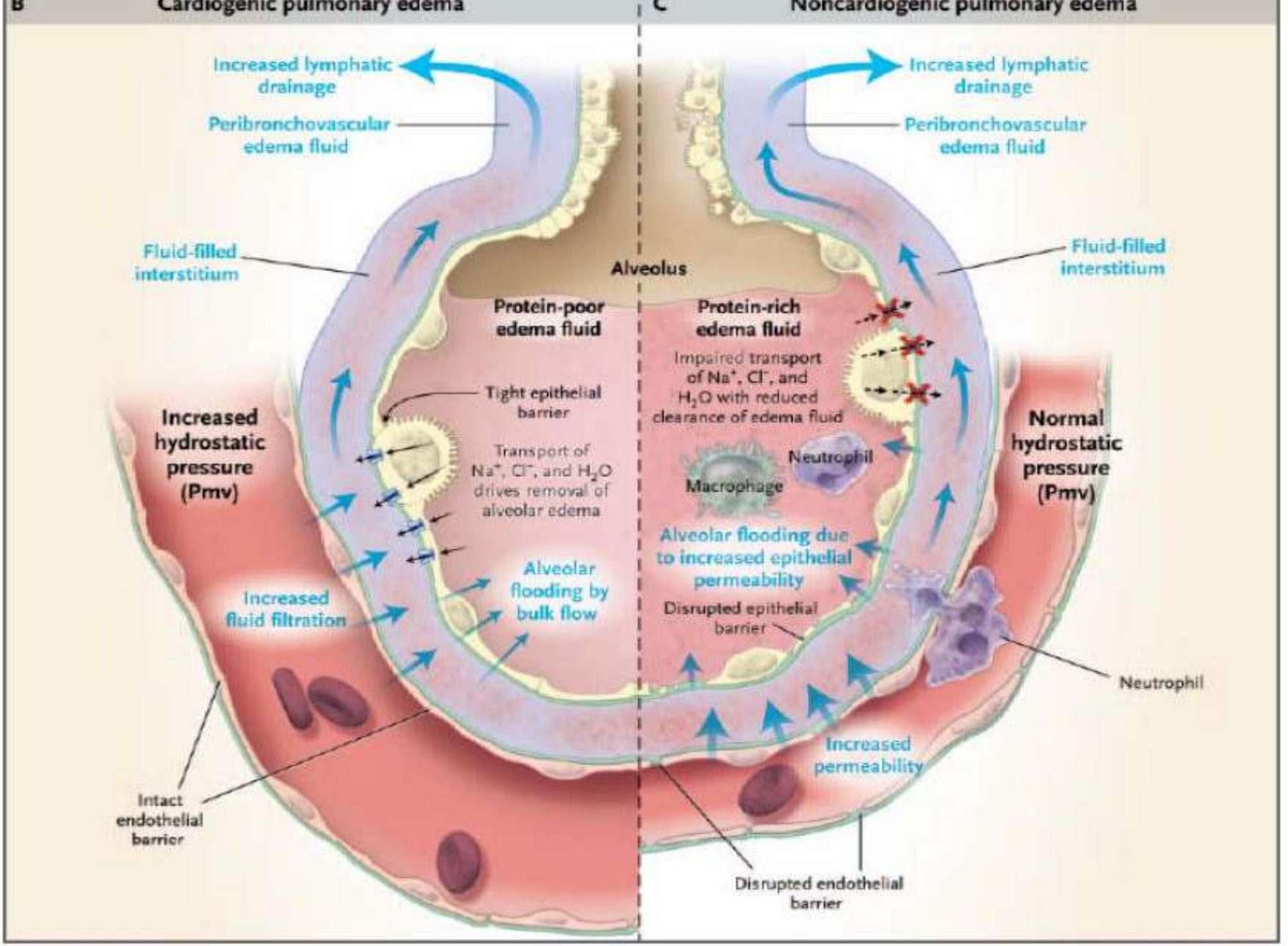
Starling forces involved in APE





Ware L. N Engl J Med 2005

Etiology of acute respiratory distress syndrome*

Etiology	Clinical features	Diagnostic tests
Sepsis	Fever hypotension, leukocytosis, lactic acidosis, infectious source	Appropriate clinical context and positive cultures
Aspiration pneumonitis	Witnessed or risk for aspiration, food, lipid laden macrophages, airway erythema on bronchoscopy	Presumptive diagnosis with negative cultures
Infectious pneumonia (including mycobacterial, viral, fungal, parasitic)	Productive cough, pleuritic pain, fever, leukocytosis, lobar consolidation or bilateral infiltrates in an immunosuppressed patient	Appropriate clinical context and positive respiratory cultures
Severe trauma and/or multiple fractures	History of trauma or fractures within the last week	Diagnosis is apparent
Pulmonary contusion	History of chest trauma (blunt or penetrating), chest pain	Presumptive diagnosis in the correct clinical context, negative cultures
Burns and smoke inhalation	Exposure to fire or smoke, cough, dyspnea, DIC, particulate matter on bronchoscopy, surface burns	Presumptive diagnosis in the correct clinical context, negative cultures
Transfusion related acute lung injury and massive transfusions	History of transfusion, dyspnea during or shortly after transfusion	Diagnosis of exclusion
HSCT¶	History of HSCT	Diagnosis of exclusion
Pancreatitis	Abdominal pain, vomiting, risk actors (eg, gallstones, alcohol, viral infection)	Elevated amylase and lipase, with or without abnormal imaging
Inhalation injures other than smoke (eg, near drowning, gases)	History of inhalation exposure (eg, chlorine gas)	Diagnosis of exclusion
Thoracic surgery (eg, post-cardiopulmonary bypass) or other major surgery	History of surgery, intraoperative ventilation, intraoperative transfusion	Diagnosis of exclusion
Drugs (chemotherapeutic agents, amiodarone, radiation)	New drugs or radiation exposure on history, lymphocytosis on lavage, lavage may have suggestive features of amiodarone toxicity ("foamy macrophages") but is nonspecific	Diagnosis of exclusion, lung biopsy occasionally helpful

ARDS has over 60 etiologies. This is an abbreviated list of the common causes of ARDS.

ARDS: acute respiratory distress syndrome; DIC: disseminated intravascular coagulation; HSCT: hematopoietic stem cell transplant; AEP: acute eosinophilic pneumonia; COP: cryptogenic organizing pneumonia; DAD: diffuse alveolar damage.

* Use of the term ARDS to describe conditions such as AEP or COP is somewhat controversial. However, some experts consider these a "subtype" of ARDS since they present in a similar fashion to ARDS, although the pathology of such entities is different from DAD, which is the classic pathology associated with ARDS. Similarly, while neurogenic pulmonary edema meets the definition of ARDS, since it causes hypoxemia and bilateral infiltrates in the absence of pulmonary edema due to heart failure, the pathology and clinical course is likely different. Similarly, embolism of fat, air, and amniotic fluid may mimic ARDS but it is uncertain as to whether they cause ARDS.

¶ Many patients with HSCT may develop a form of lung injury after transplant but the distinction between this and ARDS due to complications of HSCT (eg, pneumonia) is often unclear.

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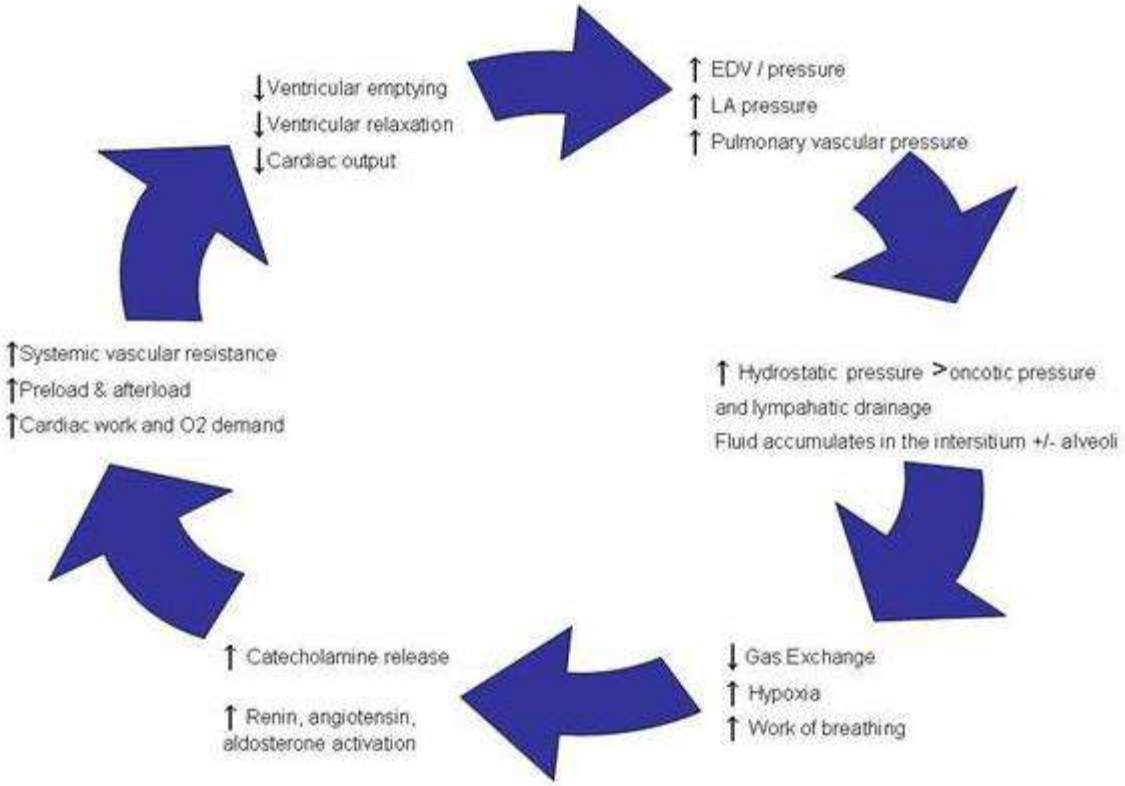
Figure 2: Pathophysiology of CPO



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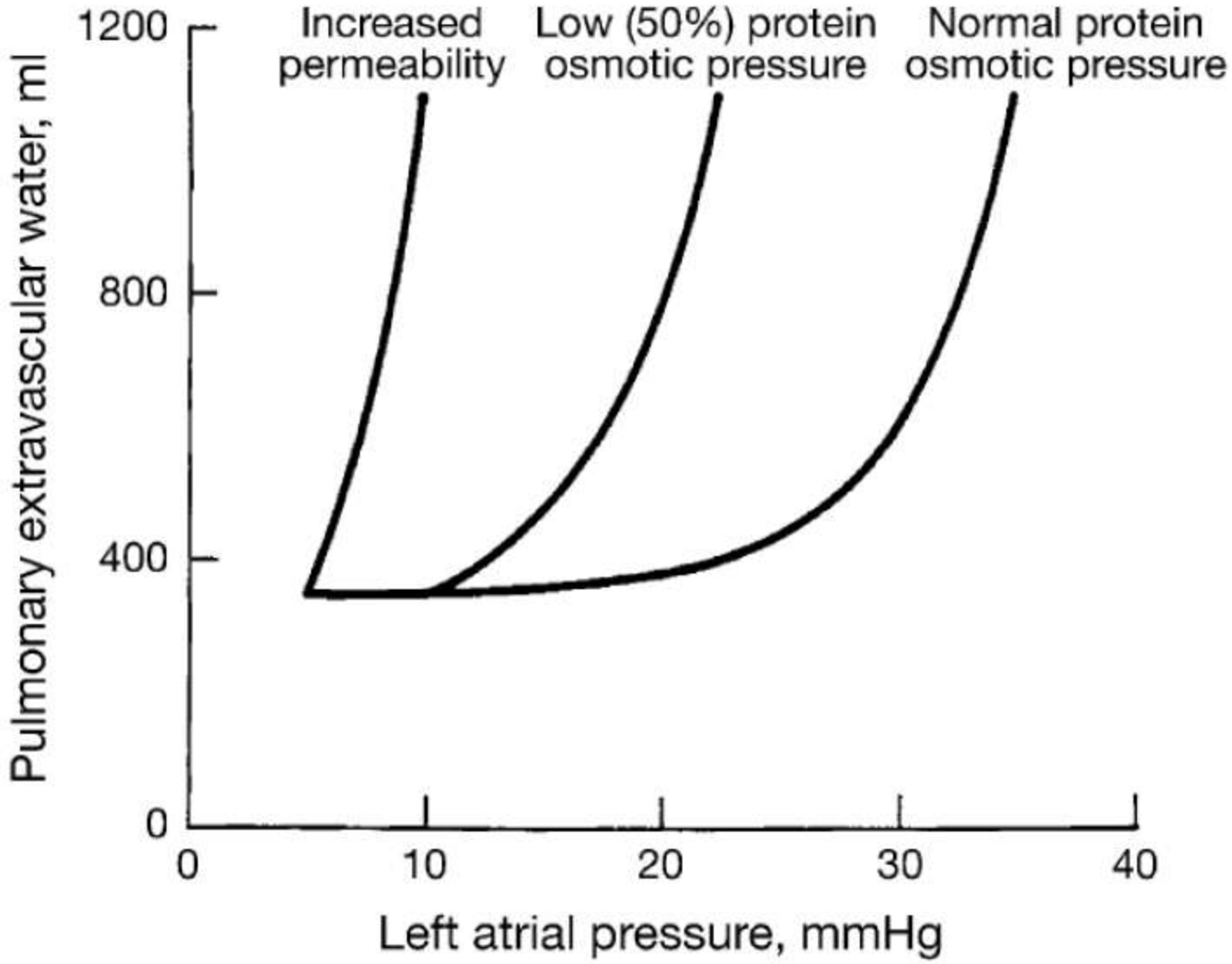

Murral JF- Int J Tuberc Lung Dis 2011

Differentiation of noncardiogenic from cardiogenic pulmonary edema based on clinical data

Noncardiogenic	Cardiogenic
History	
▪ Underlying disease (eg, pancreatitis, sepsis)	▪ Acute cardiac event (eg, myocardial infarction)
Physical examination	
▪ Warm periphery	▪ Cool, mottled periphery
▪ Bounding pulses	▪ Small-volume pulse
▪ Normal-sized heart	▪ Cardiomegaly
▪ Normal JVP	▪ Elevated JVP
▪ S3 absent	▪ S3 present
▪ No murmurs other than innocent flow murmurs	▪ Systolic and diastolic murmurs
ECG	
▪ ECG usually normal	▪ ECG signs of myocardial infarction/ischemia
Chest radiograph film	
▪ Peripheral infiltrates	▪ Perihilar infiltrates
Laboratory test	
▪ BNP <100 mg/mL	▪ BNP >100 mg/mL
Ventilatory needs	
▪ Prolonged need for ventilatory support with high FiO_2 and PEEP to oxygenate	▪ Short duration of need for ventilatory support

JVP: jugular venous pressure; S3: third heart sound; ECG: electrocardiogram; BNP: brain natriuretic peptide; FiO_2: fraction of inspired oxygen; PEEP: positive end-expiratory pressure.

Adapted from: Sibbald WJ, Cunningham DR, Chin DN. Non-cardiac or cardiac pulmonary edema? A practical approach to clinical differentiation in critically ill patients. Chest 1983; 84:452.

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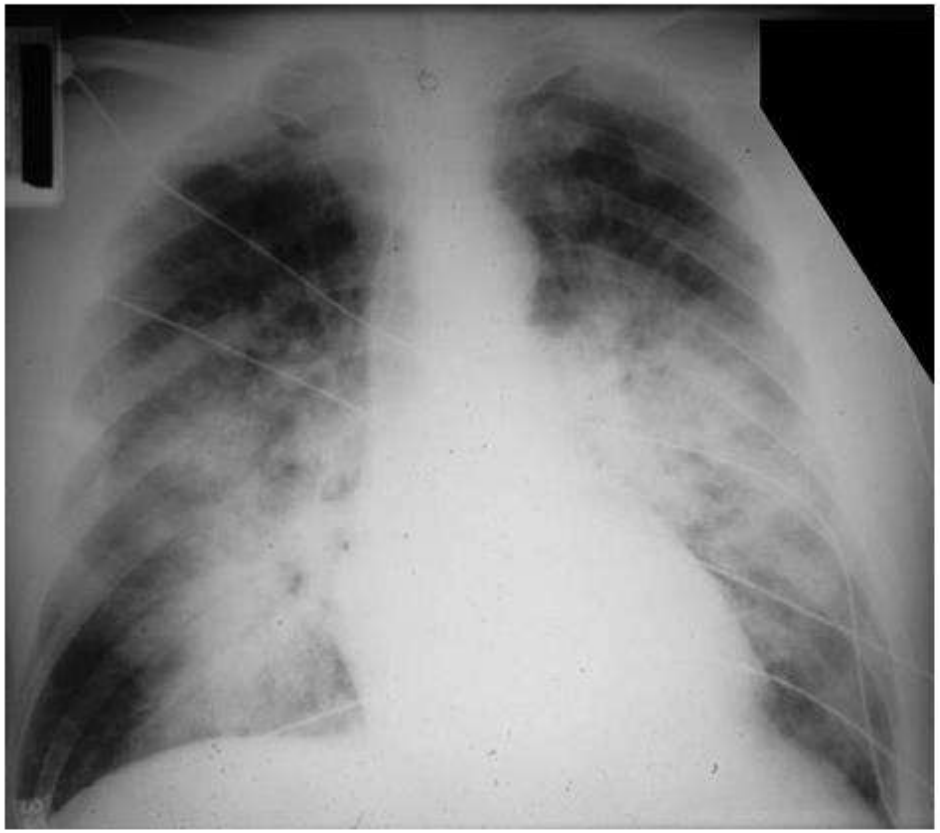
Acute respiratory distress syndrome



Chest radiograph showing diffuse, bilateral, alveolar infiltrates without cardiomegaly in a patient with ARDS.

ARDS: acute respiratory distress syndrome.

Courtesy of Steven E Weinberger, MD.

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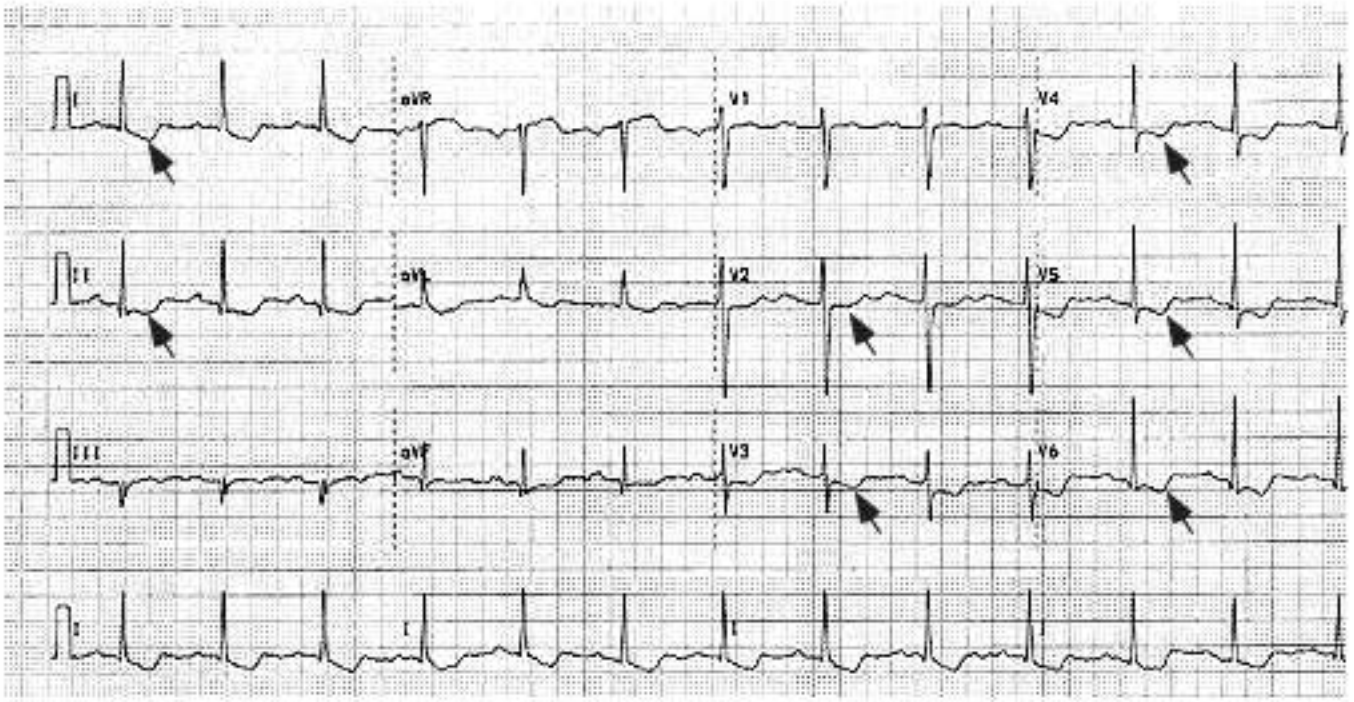
Electrocardiogram in a patient with flash pulmonary edema



The ECG tracing reveals left ventricular hypertrophy (LVH) assoicated with ST segment depression and T wave inversions; these ST-T wave changes (arrows) may also represent subendocardial ischemia, which along with the LVH may be responsible for the episode of flash pulmonary edema.

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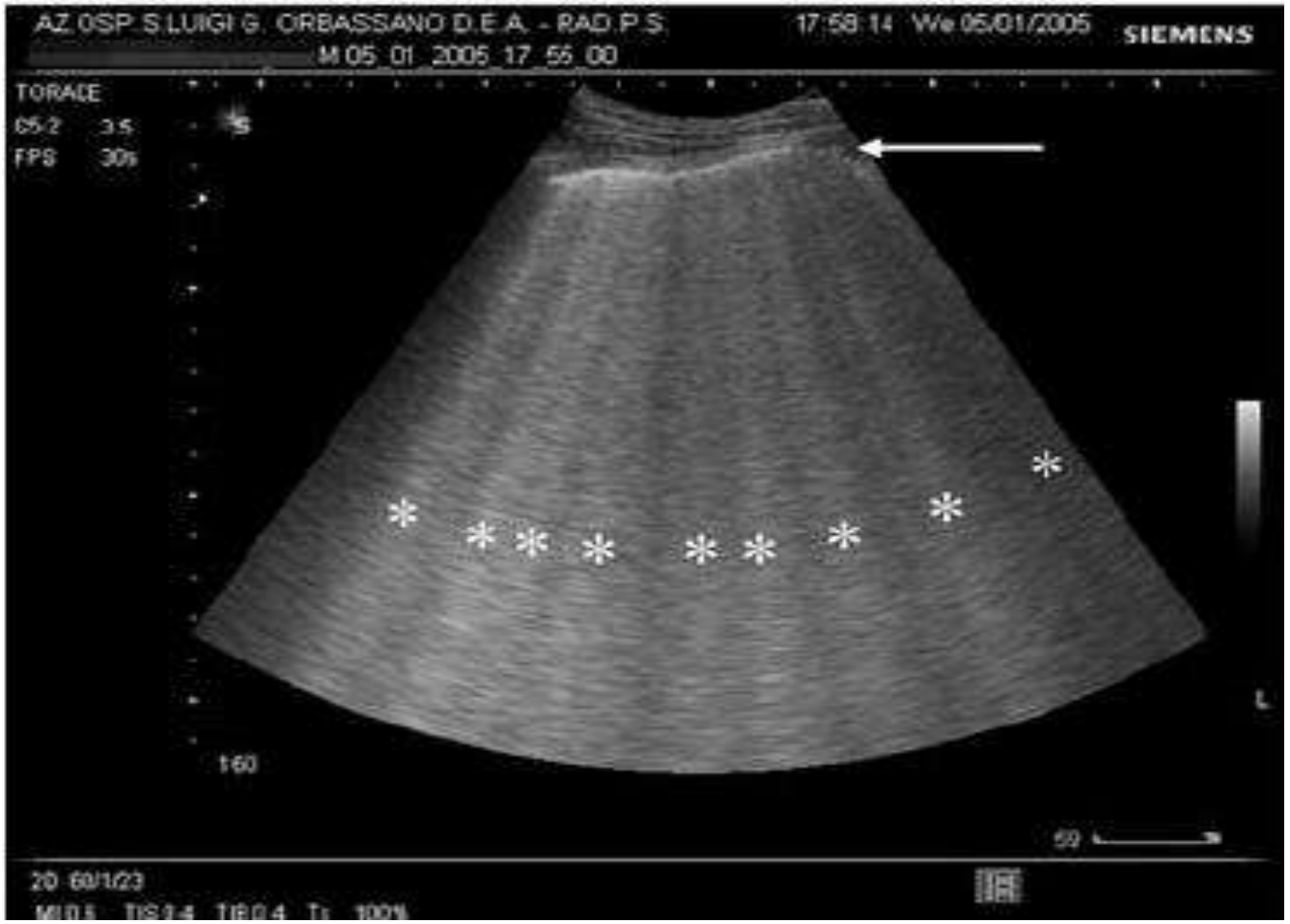

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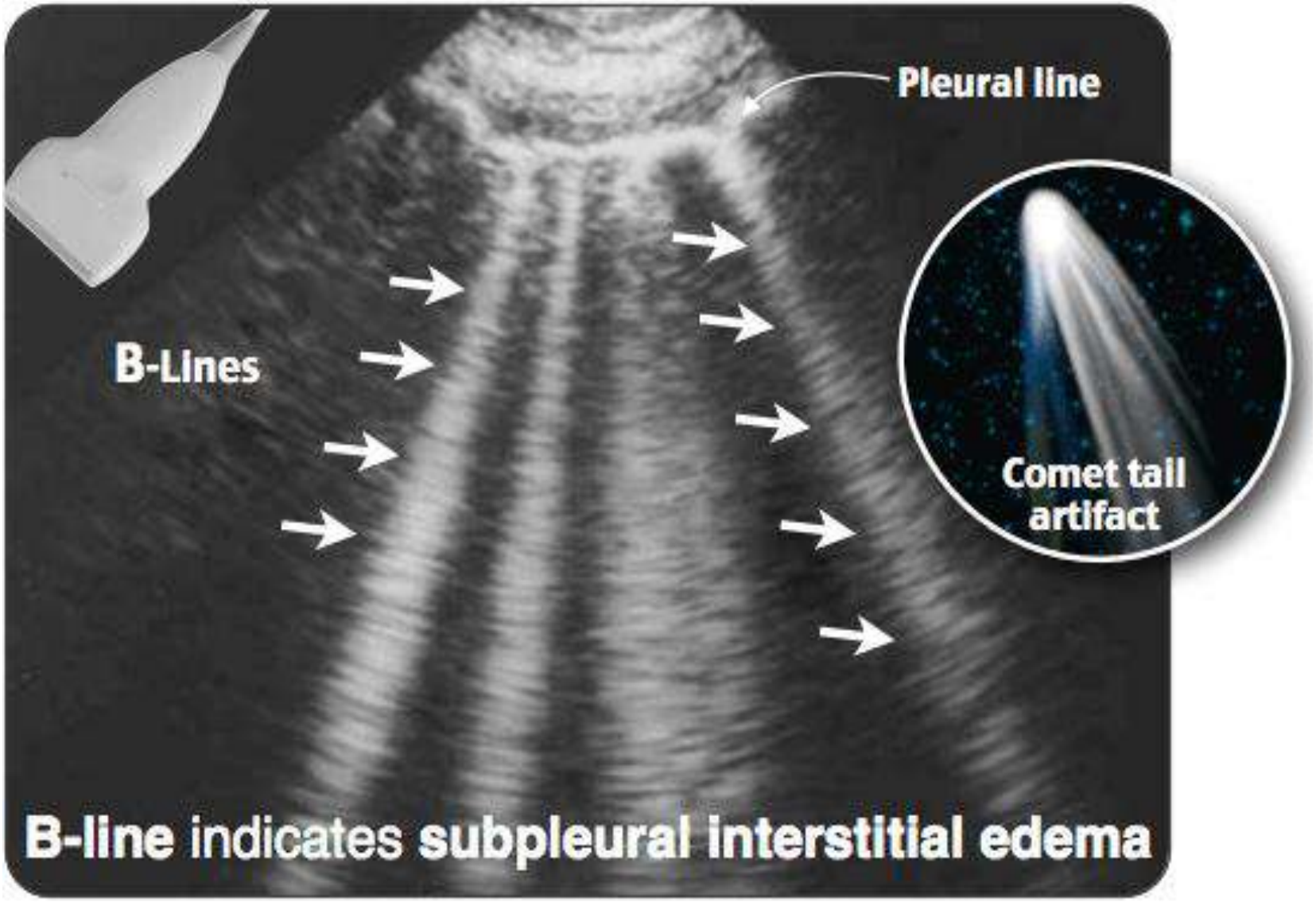

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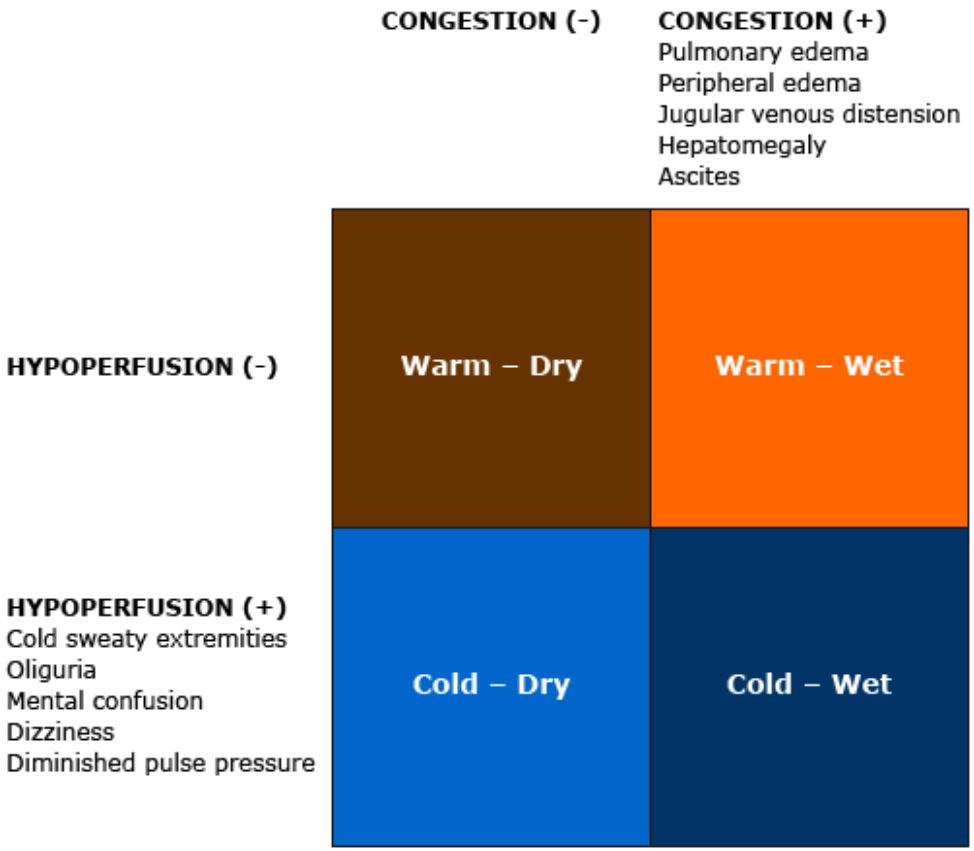
Clinical profiles in acute decompensated heart failure



Adapted from:

1. *Nohria A, Tsang SW, Fang JC, et al. Clinical assessment identifies hemodynamic profiles that predict outcomes in patients admitted with heart failure. J Am Coll Cardiol 2003; 41:1797.*
2. *Thomas SS and Nohria A. Hemodynamic classifications of acute heart failure and their clinical application: an update. Circ J 2012; 76:278.*

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Acute Cardiovascular Care Association

CLINICAL DECISION MAKING TOOLKIT

2018 edition

Instant guidance for diagnosis, risk management and treatment

ESC European Society of Cardiology

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DYSPNEA: Diferential diagnosis

50% have ≥ 2 diagnoses, which may result in acute respiratory failure*!

Basic measures

- BP, HR, respiratory rate, SpO_2 & temperature
- Start oxygen to target SpO_2 94-98%
- Start i.v. line & monitor patient

Criteria for transfer to ICU
(despite treatment for 30 minutes)

- Respiratory rate >35/min
- SpO_2 <85%
- SBP <90 mmHg
- HR >120 bpm

Investigations:

- ECG
- BNP
- Chest X-ray
- Venous BG
- Blood count
- D-dimers if suspicion of PE
- cTn

Acute heart failure

Acute coronary syndrome

Pneumonia

Exacerbated COPD or other chronic lung disease

Pulmonary embolism

Other causes, including

- Asthma
- Severe sepsis
- Tumor
- Pneumothorax
- Pleural effusion/ascites
- Anxiety disorder
- Anemia
- Bronchitis
- Metabolic acidosis
- Neurologic disease

* Defined as ≥1 criterion:
  - Respiratory rate ≥25/min
  - PaO_2 ≤75 mmHg
  - SpO_2 ≤92% in ambient air
  - $PaCO_2$ ≥45 mmHg with arterial pH ≤7.35

Reference: Ray P et al. Acute respiratory failure in the elderly: etiology, emergency diagnosis and prognosis. Critical Care (2006);10 (3):R82.

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DYSPNEA: Acute heart failure (see chapter 4.1)

- **Immediate 12-lead ECG, cardiac monitor, BP, respiratory rate, pulse oximetry**
- **Clinical findings**
  Most commonly: lower extremity edema, jugular venous distension, rales, work up for underlying cardiac disease and triggers
- **Laboratory findings**
  Complete blood count, chemistries, cardiac enzymes, BNP, TSH, ABG as needed
- **Chest X-ray (lung ultrasound)**
- **Echocardiogram**
  During admission (earlier if decompensated aortic stenosis or endocarditis are suspected)
- **Coronary angiography**
  Emergent in patients with ACS; delayed in patients with suspected coronary artery disease

• Positioning	Keep head of bed elevated above level of legs
• Oxygen	Up to 12 l/min via rebreather mask, titrate oxygen saturation to 94%
• Nitroglycerin	1-2 SL tablets or 2-3 patches 10 mg (1st choice). In pulmonary edema with severe shortness of breath: NTG drip 0.05% (100 mg in 200 ml) - Start with 25 µg/min = 3 ml/h, check BP after 5 and 10 min - Increase dose by 25 µg/min at a time as long as SBP >90 mmHg - Additional BP check 5 and 10 min after each increase in dosing - Check BP every 20 min once a steady drip rate is reached
• Furosemide	40-120 mg i.v. (adjust based on kidney function and clinical findings; monitor creatinine)
• Morphine	2 mg i.v. (preceeded by 10 mg i.v. metoclopramide PRN) if patient is in severe dyspnoea
• Consider digoxin	0.5 (-1.0) mg i.v. in patients with atrial fibrillation
• Anticoagulation	Therapeutic dosing in ACS and atrial fibrillation: Enoxaparin 1 mg/kg body weight as 1st dose

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Clinical profiles of patients with acute heart failure

4.1

P.52

Clinical profiles of patients with acute heart failure
based on the presence/absence of congestion and/or hypoperfusion

	CONGESTION (-)	CONGESTION (+) Pulmonary congestion, orthopnoea/paroxismal, nocturnal dyspnoea, peripheral (bilateral) oedema, jugular venous dilatation, congested hepatomegaly, gut congestion, ascites, hepatojugular reflux
HYPOPERFUSION (-)	WARM-DRY	WARM-WET
HYPOPERFUSION (+) Cold sweaty extremities, Oliguria, Mental confusion, Dizziness, Narrow pulse pressure	COLD-DRY	COLD-WET

Hypoperfusion is not synonymous with hypotension, but often hypoperfusion is accompanied by hypotension.

Reference: Ponikowski P et al. Eur J Heart Fail. 2016; 18(8):891-975. DOI: 10.1002/ejhf.592.

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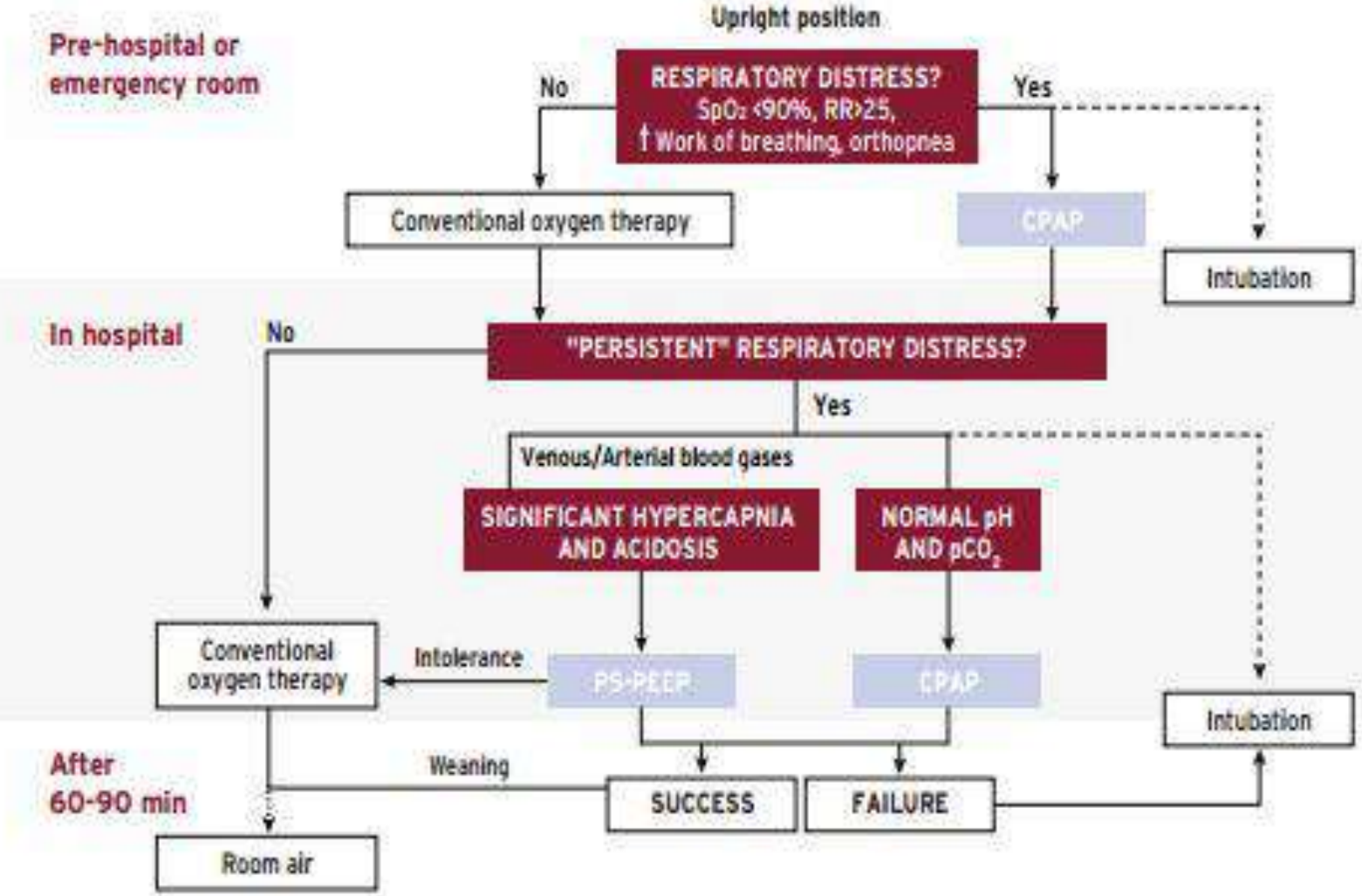
ACUTE HEART FAILURE: Airway (A) and breathing (B)
Oxygen therapy and ventilatory support in acute heart failure



Reference adapted from Mebazaa A et al. Eur J Heart Fail. (2015);17:544-58.

ACUTE HEART FAILURE: Initial diagnosis (CDE)

C - CIRCULATION*

HR (bradycardia [<60/min], normal [60-100/min], tachycardia [>100/min]), rhythm (regular, irregular), SBP (very low [<90 mmHg], low, normal [110-140 mmHg], high [>140 mmHg]), and elevated jugular pressure should be checked.

INSTRUMENTATION & INVESTIGATIONS:

Intravenous line (peripheral/central) and BP monitoring (arterial line in shock and severe ventilatory/gas-exchange disturbances)

Laboratory measures

- Cardiac markers (troponin, BNP/NT-proBNP/MR-proANP)
- Complete blood count, electrolytes, creatinine, urea, glucose, inflammation, TSH
- Consider arterial or venous blood gases, lactate, D-dimer (suspicion of acute pulmonary embolism)

Standard 12-lead ECG

- Rhythm, rate, conduction times?
- Signs of ischemia/myocardial infarction? Hypertrophy?

Echocardiography

a) Immediately in haemodynamically unstable patients
b) Within 48 hours when cardiac structure and function are either not known or may have changed since previous studies

Ventricular function (systolic and diastolic)? Estimated left-and right-side filling pressures? Lung ultrasound? Presence of valve dysfunction (severe stenosis/ insufficiency)? Pericardial tamponade?

ACTIONS:

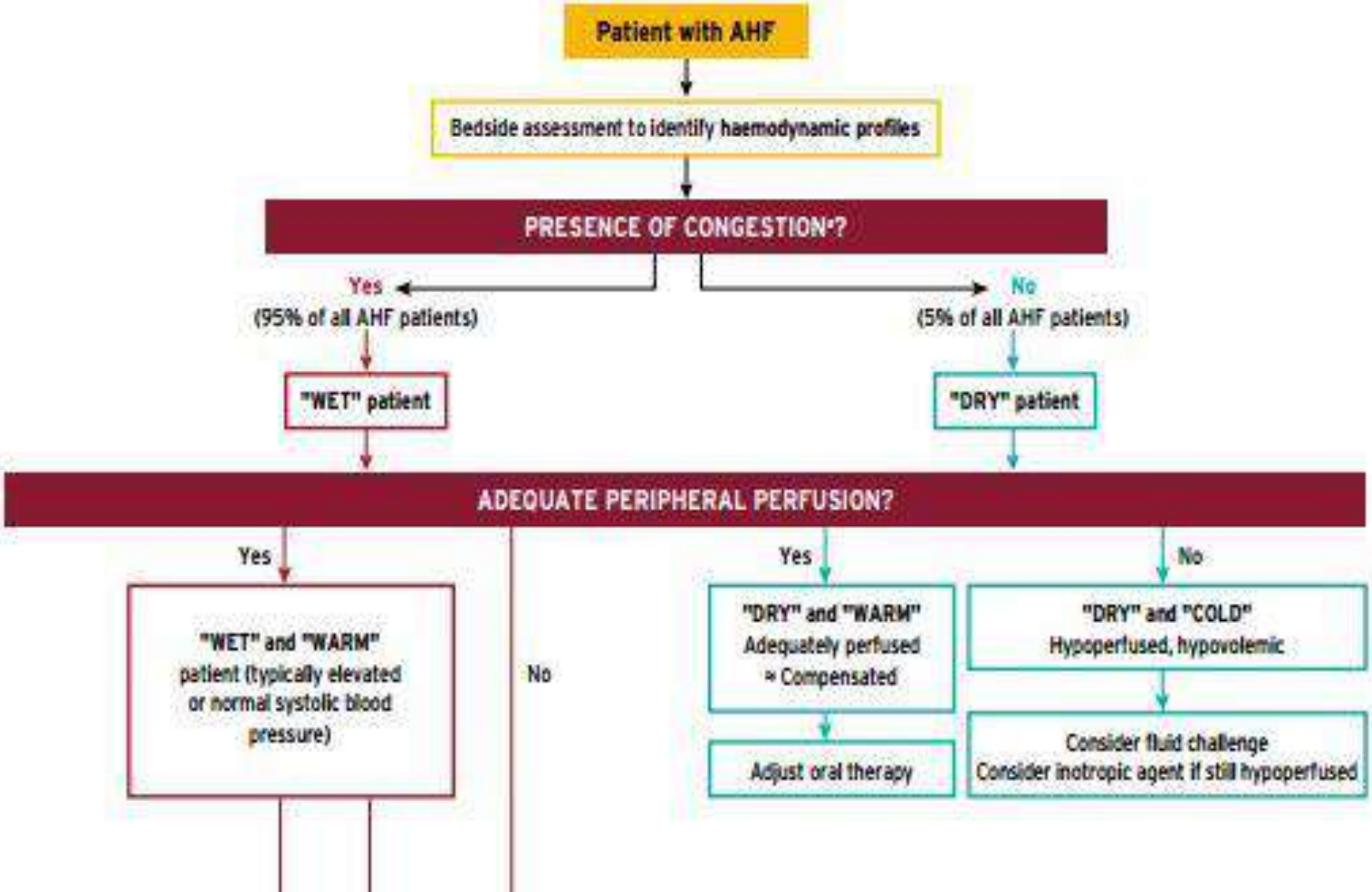
Rule in/out acute heart failure as cause of symptoms and signs

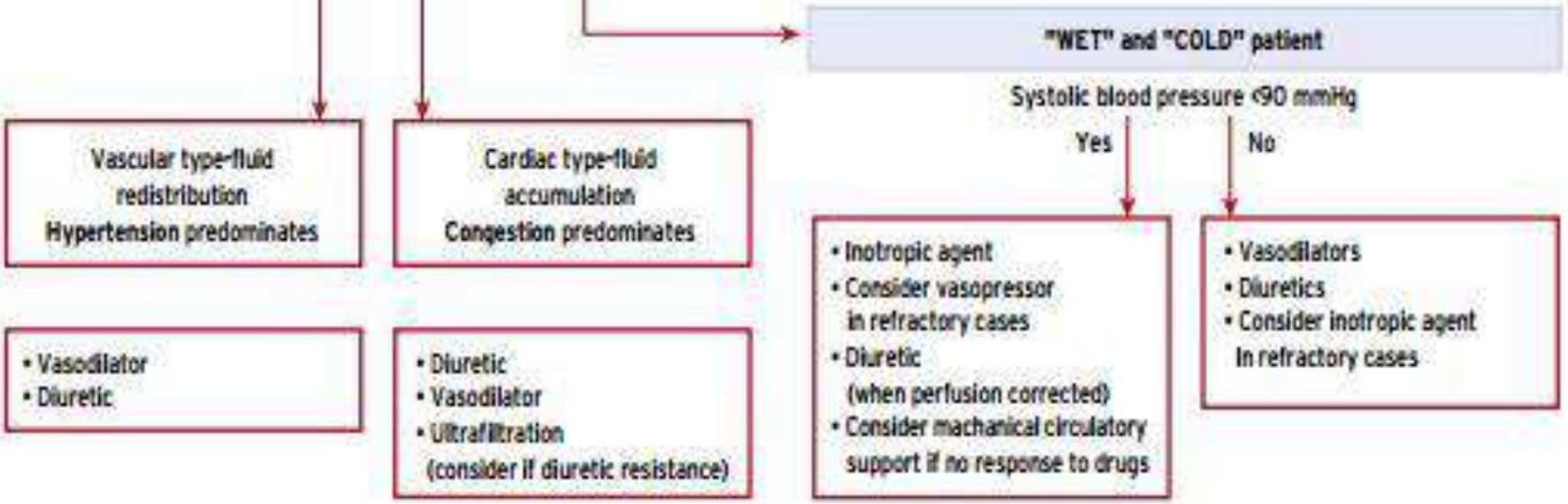
Determine clinical profile

Start as soon as possible treatment of both heart failure and the factors identified as triggers

Establish cause

ACUTE HEART FAILURE: Management of patients with acute heart failure based on clinical profile during an early phase



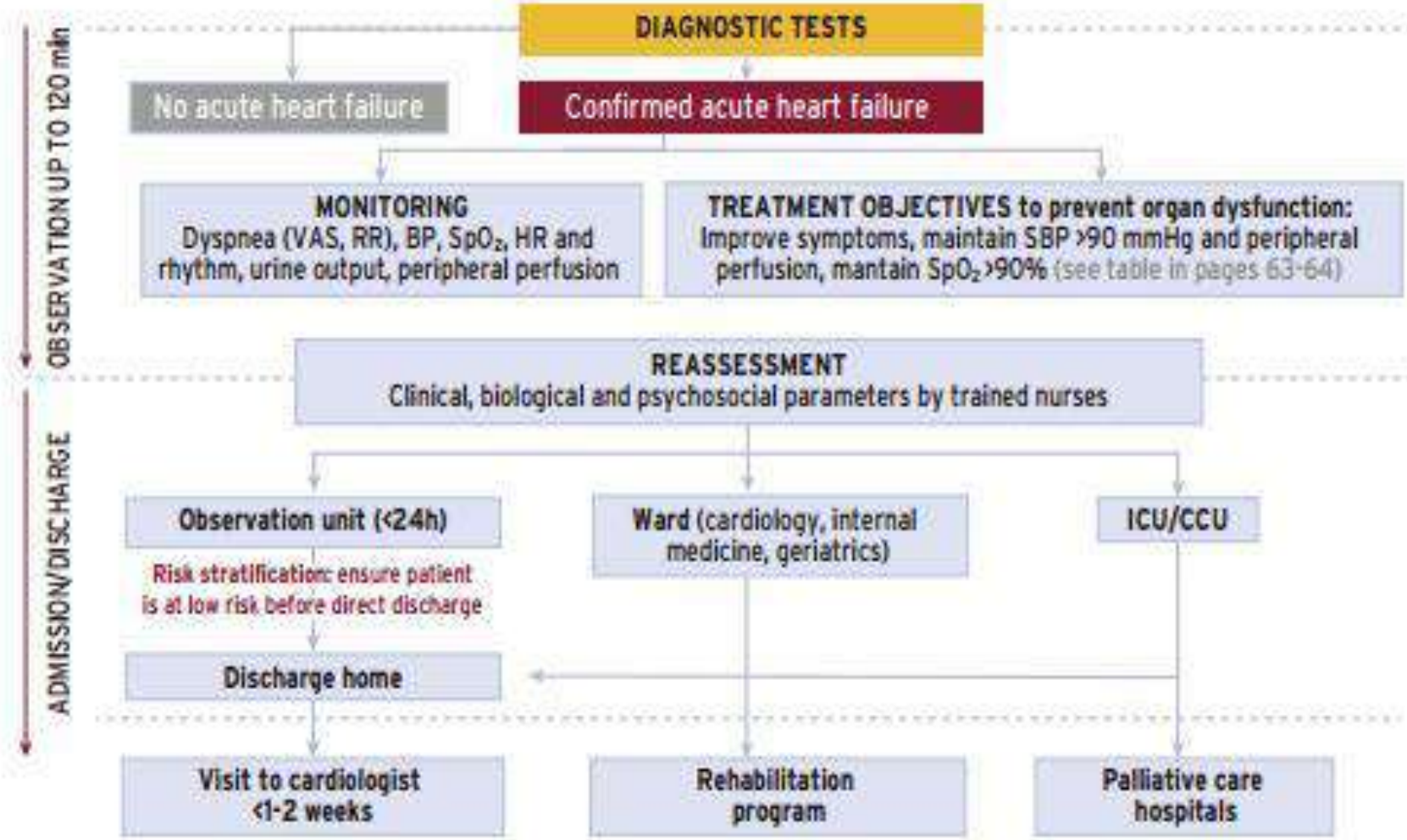


*Symptoms/signs of congestion: orthopnoea, paroxysmal nocturnal dyspnoea, breathlessness, bi-basilar rales, abnormal blood pressure response to the Valsalva maneuver (left-sided); symptoms of gut congestion, jugular venous distension, hepatojugular reflux, hepatomegaly, ascites, and peripheral oedema (right-sided).

For more information on individual drug doses and indications,
SEE CHAPTER 9 **DRUGS USED IN ACUTE CARDIOVASCULAR CARE**

Reference: Ponikowski P et al. Eur J Heart Fail. 2016; 18(8):891-975. DOI: 10.1002/ejhf.592.

ACUTE HEART FAILURE: Management of acute heart failure



Reference adapted from Mebazaa A et al. Eur J Heart Fail. (2015);17: 544-58 and Miró Ò et al. Ann Intern Med (2017); 167:698-705.

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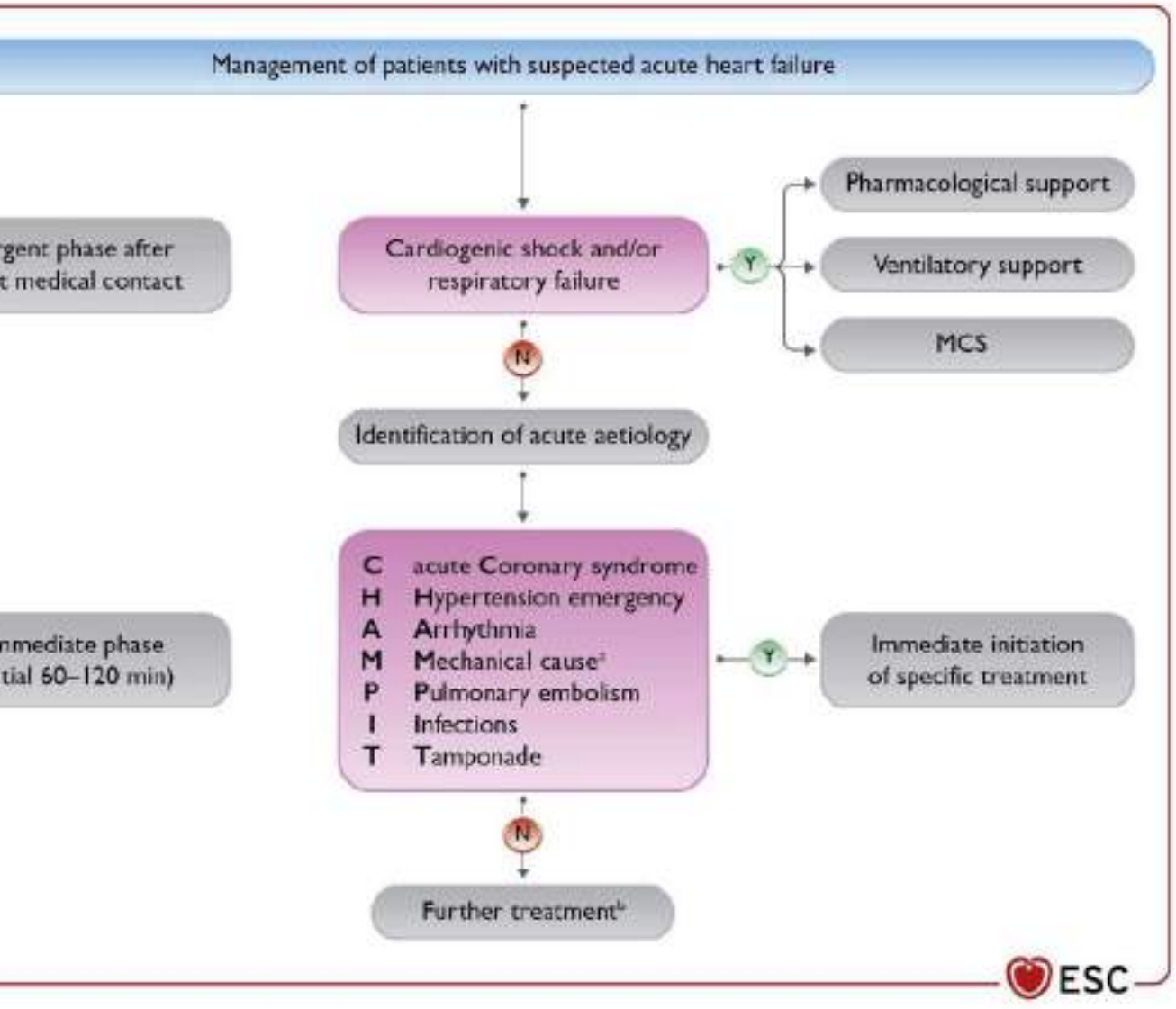
Initial management of acute heart failure



MCS = mechanical circulatory support.
[a]Acute mechanical cause: myocardial rupture complicating acute coronary syndrome (free wall r ventricular septal defect, acute mitral regurgitatio chest trauma or cardiac intervention, acute native prosthetic valve incompetence secondary to endo aortic dissection or thrombosis.
[b]See previous slides for specific treatments accord different clinical presentations.

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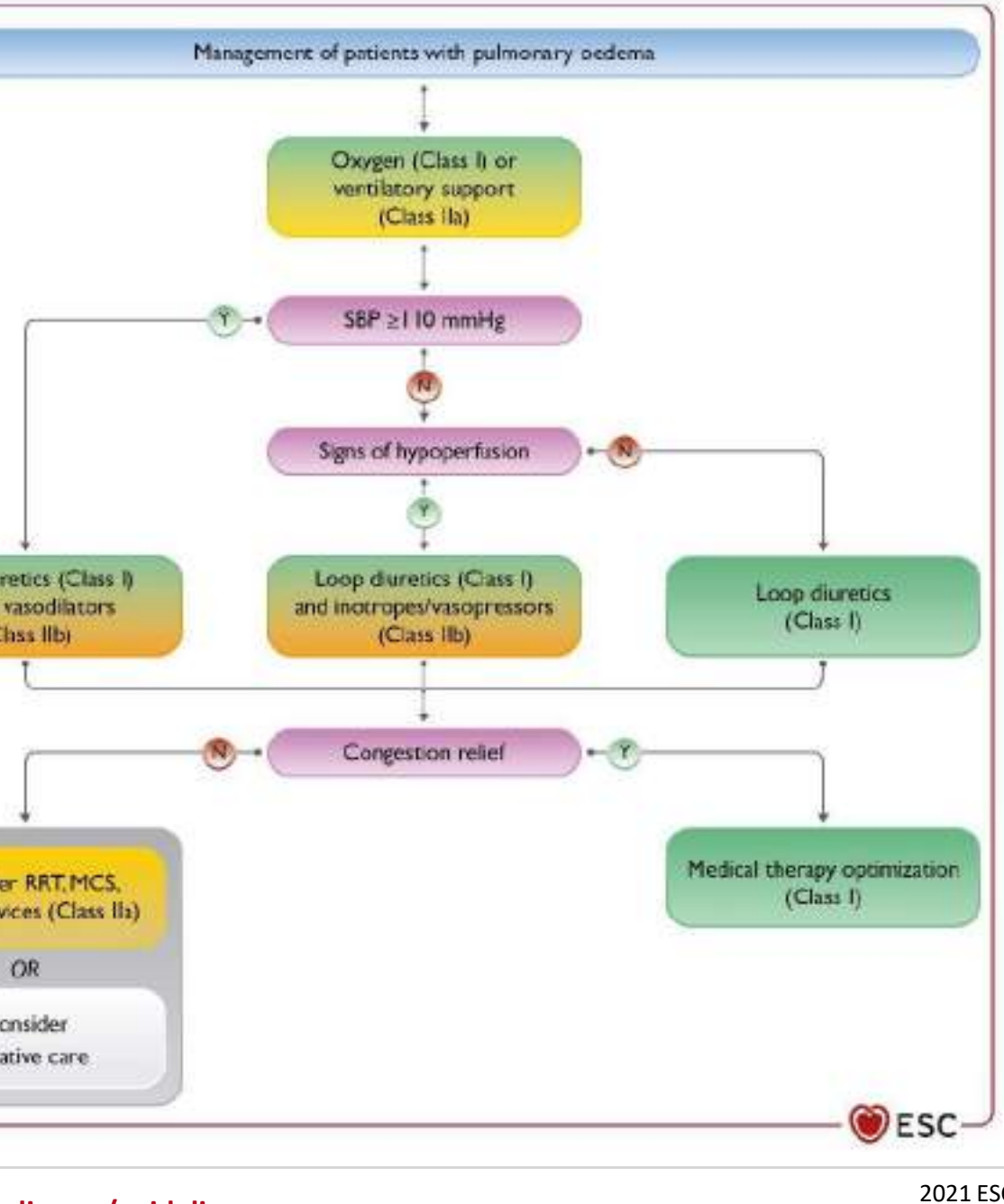
Management of pulmonary oedema



MCS=mechanical circulatory support; RRT= renal replacement thera
SBP=systolic blood pressure.

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mendations for the initial treatment of acute heart failure (1)

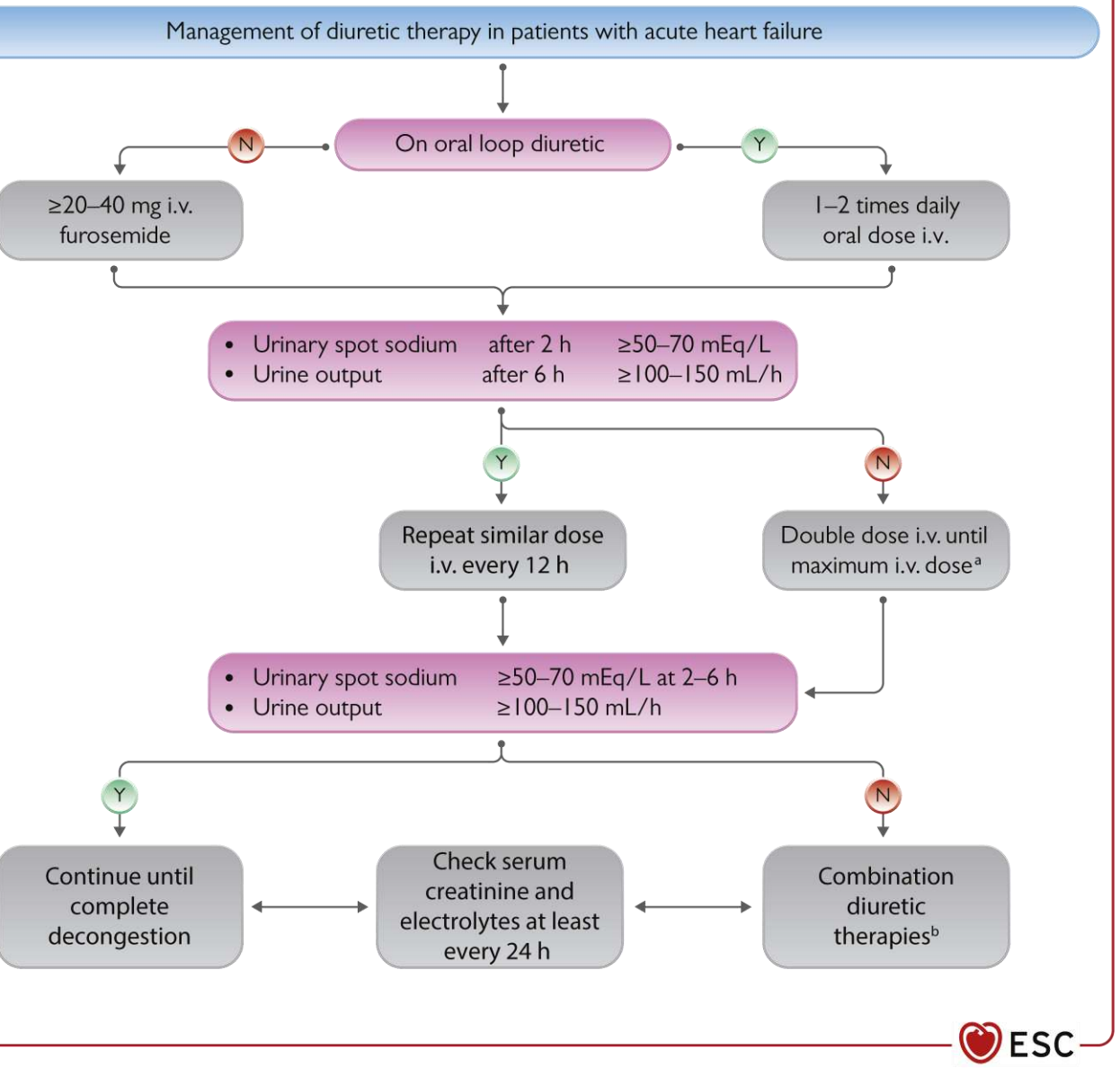
endations	Class	Lev
nd ventilatory support		
s recommended in patients with SpO_2 <90% or PaO_2 <60 mmHg to ypoxaemia.	I	C
n is recommended for progressive respiratory failure persisting in spite of dministration or non-invasive ventilation.	I	C
sive positive pressure ventilation should be considered in patients with ry distress (respiratory rate >25 breaths/min, SpO_2 <90%) and started as possible in order to decrease respiratory distress and reduce the rate of cal endotracheal intubation.	IIa	B

ssure of oxygen; SpO_2 =transcutaneous oxygen saturation.

mendations for the initial treatment of acute heart failure (2)

endations	Class	Lev
ous loop diuretics are recommended for all patients with AHF admitted s/symptoms of fluid overload to improve symptoms.	I	C
tion of a loop diuretic with thiazidetype diuretic should be considered in with resistant oedema who do not respond to an increase in loop diuretic	IIa	B
tors		
ts with AHF and SBP >110 mmHg, i.v. vasodilators may be considered as erapy to improve symptoms and reducecongestion.	IIb	B

failure; i.v. = intravenous; SBP = systolic blood pressure.



Diuretic therapy (furosemide) in acute heart failure

i.v.=intravenous.

[a]The maximal daily dose for i.v. loop diuretics is ge considered furosemide 400–600 mg though up to mg may be considered in patients with severely im kidney function.

[b]Combination therapy is the addition to the loop d of a diuretic with a different site of action, e.g. thia or metolazone or acetazolamide.

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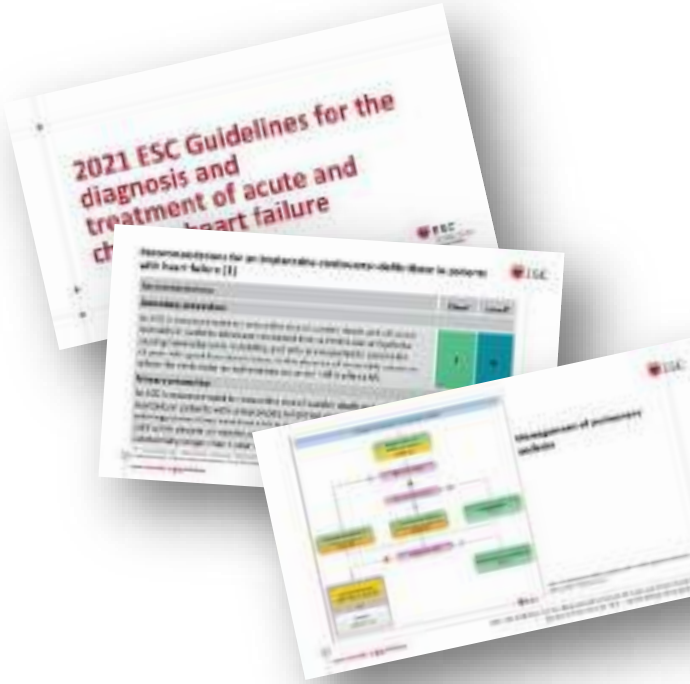
Pocket Guidelines

ESC Pocket Guidelines App

ESC Guidelines Official Slide-set



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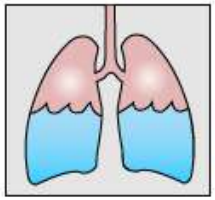
Heart failure action plan - page 1

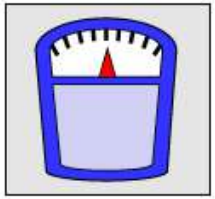
Every morning, when you get up, check how you are doing. Look for:



Changes in breathing

Ask yourself:

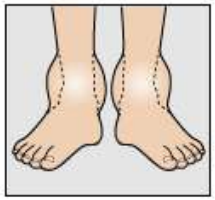
- Can I breathe as well as I usually can?
- Am I getting out of breath doing things I can normally do without a problem?
- Am I coughing more than usual?
- Did I use more pillows than usual to sleep last night?



Changes in weight

Weigh yourself every morning after urinating but before eating. Write down your weight on a calendar. Then ask yourself:

- Has my weight gone up or gone down compared to yesterday? If so, by how many pounds?
- Has my weight gone up or gone down compared to a week ago? If so, by how many pounds?



New or worse swelling

Ask yourself:

- Are my ankles more swollen than usual?
- Do my socks or shoes feel tighter?
- Do my clothes feel tighter at the waist?
- Do my rings fit more snugly?

Changes in your ability to do everyday things

Ask yourself:

- Can I do all the things I normally do, such as get dressed on my own, make meals, or go for walks?
- Do I feel dizzy or more tired than usual?
- Do I have any new symptoms, like pressure or pain in my chest?
- Does my heartbeat feel strange or irregular?
- Do I feel like I might pass out?

See the next page to find out what you should do if any of these changes occur.

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Heart failure action plan - page 2

Symptom	Action
Best weight: _____ If you have: ▪ No trouble breathing ▪ No chest pain ▪ No weight change overnight or over the last week ▪ The usual amount of ankle swelling ▪ No change in ability to be active	Your symptoms are under control. ▪ Keep taking your medications every day, as ordered ▪ Keep weighing yourself every day and writing down your weight ▪ Keep all your medical appointments
If you: ▪ Need more pillows than usual to sleep ▪ Have more trouble breathing when you are active ▪ Have more coughing than usual ▪ Increased shortness of breath with activity ▪ Gain 2 to 3 pounds overnight, or 5 pounds in one week ▪ Have more swelling than usual	You might need to take extra medicine. Call your doctor's office to find out what you should do. Doctor name: _____ Phone #: _____
If you: ▪ Have trouble breathing when you are resting, or you can't stop coughing ▪ Wheeze or feel chest tightness when you are resting ▪ Wake up at night because you can't breathe well ▪ Feel dizzy, very tired, or like you might fall ▪ Gain or lose more than 5 pounds compared to your normal weight	You probably need to **see** a doctor right away. Call your doctor **now**. Doctor name: _____ Phone #: _____
If you: ▪ Have trouble breathing that does not get better no matter what you do ▪ Feel like you can't breathe, or start to turn blue ▪ Cough up frothy or pink saliva ▪ Have pain or pressure in your chest, or you have other signs of a heart attack ▪ Have a fast or uneven heartbeat that will not go away or makes you feel dizzy or lightheaded ▪ Feel very confused ▪ Faint	 Call 9-1-1 for an ambulance **right away**

Adapted from: Heart failure: Heart Failure Action Plan. The National Heart Foundation of New Zealand. Available at: https://www.heartfoundation.org.nz/your-heart/heart-conditions/heart-failure.

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Acute decompensated heart failure: Rapid overview of emergency management

Differential diagnosis: Pulmonary embolism, acute asthma, pneumonia, noncardiogenic pulmonary edema (eg, adult respiratory distress syndrome), pericardial tamponade or constriction
Symptoms and signs
Acute dyspnea, orthopnea, tachypnea, tachycardia, and hypertension are common
Hypotension reflects severe disease, and arrest may be imminent; assess for inadequate peripheral or end-organ perfusion
Accessory muscles are often used to breathe
Diffuse pulmonary crackles are common; wheezing (cardiac asthma) may be present
S_3 is a specific sign but may not be audible; elevated jugular venous pressure and/or peripheral edema may be present
Diagnostic studies
Obtain ECG: Look for evidence of ischemia, infarction, arrhythmia (eg, AF), and left ventricular hypertrophy.
Obtain portable chest radiograph: Look for signs of pulmonary edema, cardiomegaly, alternative diagnoses (eg, pneumonia); normal radiograph does not rule out ADHF.
Obtain: Complete blood count; cardiac troponin; electrolytes (Na^+, K^+, Cl^-, HCO_3^-); BUN and creatinine; arterial blood gas (if severe respiratory distress); liver function tests; BNP or NT-proBNP if diagnosis is uncertain.
Perform bedside echocardiography if the cardiac or valvular function is not known.
Treatment
Monitor oxygen saturation, vital signs, and cardiac rhythm.
Provide supplemental oxygen if hypoxic (SpO_2 <90%), place 2 IV catheters, and position patient upright.
Provide NIV as needed, unless immediate intubation is required or NIV is otherwise contraindicated; have airway management equipment readily available; etomidate is a good induction agent for RSI in ADHF.
Initiate diuretic therapy without delay to relieve congestion/fluid overload:
• Give IV loop diuretic furosemide 40 mg IV or torsemide 20 mg IV; or bumetanide 1 mg IV.
• Higher doses are needed for patients taking diuretics chronically (eg, twice home dose) and in patients with renal dysfunction.
Search for cause of ADHF (including: acute coronary syndrome, hypertension, arrhythmia, acute aortic or mitral regurgitation, aortic dissection, sepsis, renal failure, anemia, or drugs) and treat appropriately.
• Patients with ADHF and AF with rapid ventricular rate often require medication (eg, digoxin) to slow their heart rate.
• Direct current cardioversion is indicated for patients with new onset AF and hemodynamic instability or refractory symptoms despite rate control.
• Obtain immediate cardiac surgery consultation for acute aortic or mitral regurgitation or ascending aortic dissection.
For patients with adequate end-organ perfusion (eg, normal or elevated blood pressure) and signs of ADHF with fluid overload:
• If urgent afterload reduction is required, early vasodilator therapy may be needed: Give nitroprusside* for severe hypertension, or if acute aortic regurgitation or acute mitral regurgitation is present; titrate rapidly to effect (eg, start nitroprusside at 5 to 10 mcg/min and titrate up every 5 minutes as tolerated to a dose range of 5 to 400 mcg/min).
• If response to diuretics to treat congestion/fluid overload is inadequate, give vasodilator to reduce preload: Give IV nitroglycerin in addition to diuretic therapy if persistent dyspnea or as a component of therapy in refractory HF and low cardiac output.¶ Start nitroglycerin* infusion at 5 to 10 mcg/min and titrate every 3 to 5 minutes as needed and tolerated based upon mean arterial blood pressure or SBP to a dose range of 10 to 200 mcg/min.
For patients with known systolic HF (eg, documented low ejection fraction) presenting with signs of severe ADHF and cardiogenic shock, discontinue chronic beta blocker therapy and:
• Give an IV inotrope* (eg, dobutamine or milrinone) and/or mechanical support (eg, intraaortic balloon counter pulsation).
For patients with known diastolic HF (ie, preserved systolic function) presenting with signs of severe ADHF and cardiogenic shock:
• Treat for possible left ventricular outflow obstruction with a beta blocker, IV fluid (unless pulmonary edema is present), and give an IV vasopressor* (eg, phenylephrine or norepinephrine); do not give an inotrope or vasodilator; obtain immediate echocardiogram as needed.
• Consider possibility of acute mitral or aortic regurgitation, or aortic dissection, and need for emergency surgical intervention. Obtain immediate echocardiogram as needed.
For patients whose cardiac status is unknown but present with signs of severe ADHF (ie, pulmonary edema) and hypotension or signs of shock:
• Give an IV inotrope* (eg, dobutamine or milrinone), with or without an IV vasopressor (eg, norepinephrine) and assess need for mechanical support (eg, intraaortic balloon counter pulsation); obtain immediate echocardiogram as needed.

ECG: electrocardiogram; AF: atrial fibrillation; ADHF: acute decompensated heart failure; BUN: blood urea nitrogen; BNP: brain natriuretic peptide; NT-proBNP: N-terminal pro-BNP; IV: intravenous; NIV: noninvasive ventilation; RSI: rapid sequence intubation; SBP: systolic blood pressure.

* Patients receiving vasodilator, vasopressor, or inotrope infusions require continuous noninvasive monitoring of blood pressure, heart rate and function, and oxygen saturation.

¶ Treatment of patients with heart failure with reduced ejection fraction with volume overload unresponsive to diuretics is guided by hemodynamics, which are most commonly imputed from the physical examination with right heart catheterization performed when required for selected cases; refer to accompanying text and separate topic review of management of refractory heart failure.

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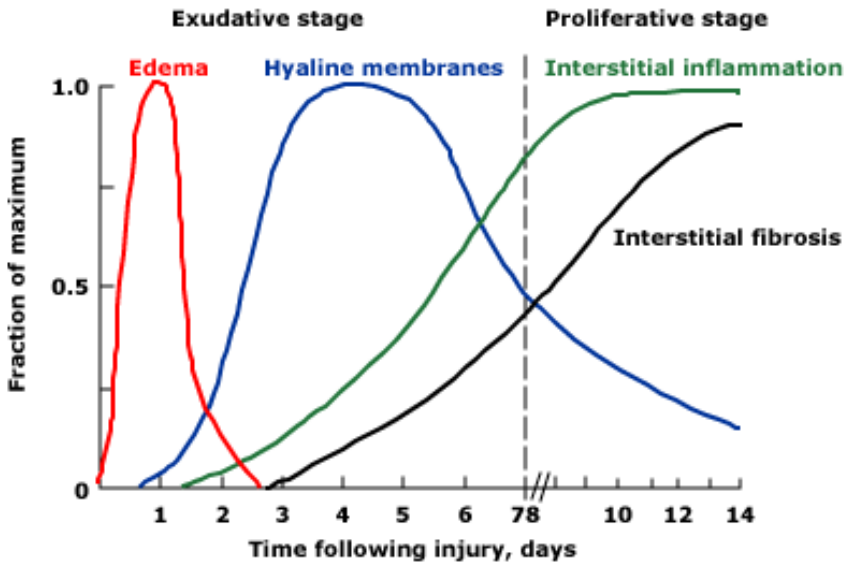
Time course of acute respiratory distress syndrome (ARDS)



Schematic representation of the time course of the acute respiratory distress syndrome (ARDS). During the early (or exudative) phase, the lesion is characterized by high permeability pulmonary edema followed by the formation of hyaline membranes. After seven to ten days, a proliferative phase may develop, with marked interstitial inflammation, fibrosis, and disordered healing.

Redrawn from Katzenstein AA, Askin FB. Surgical Pathology of Non-neoplastic Lung Disease. Saunders, Philadelphia, 1982.

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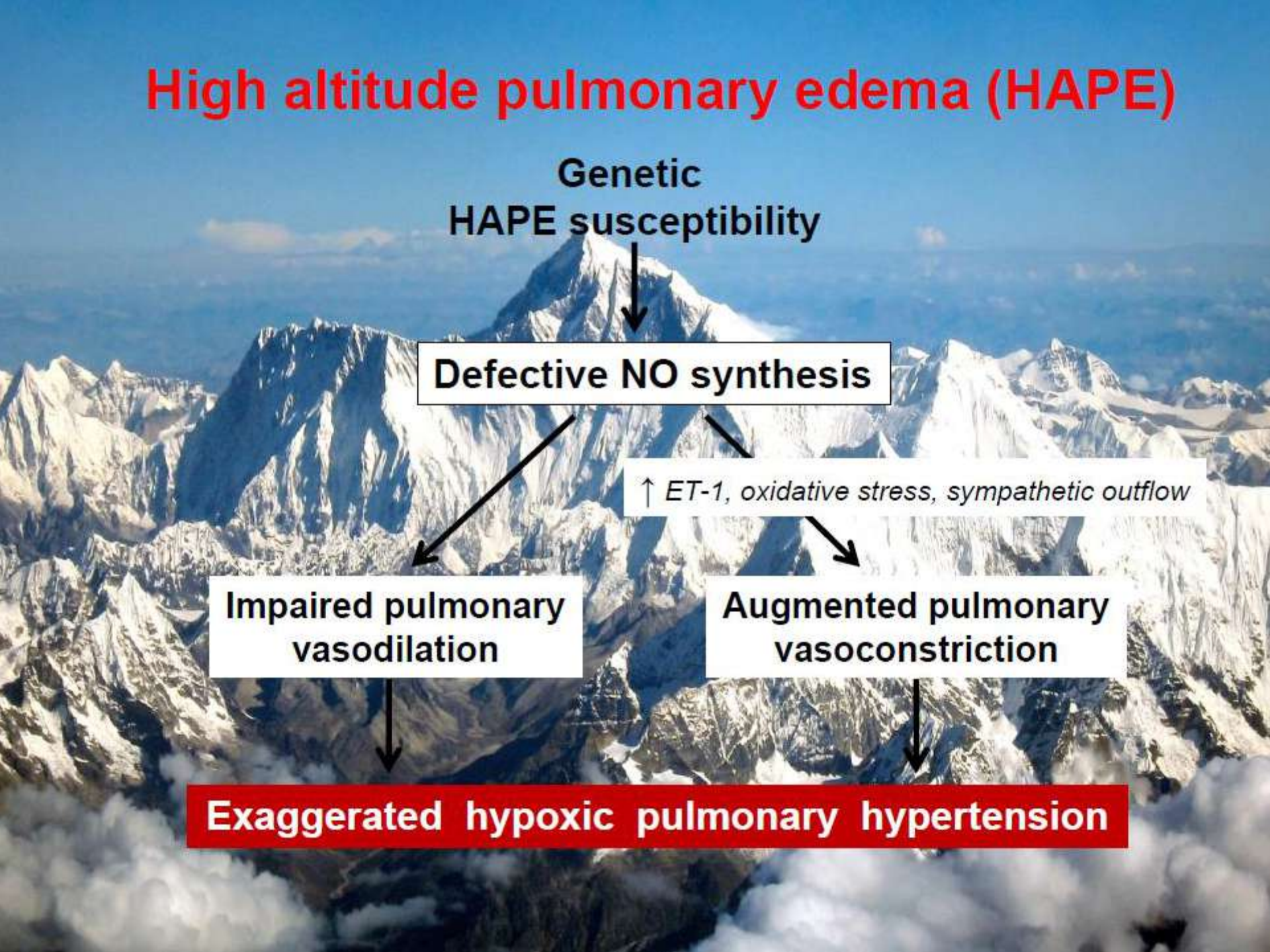

High altitude pulmonary edema (HAPE)
Genetic
HAPE susceptibility
Defective NO synthesis
↑ ET-1, oxidative stress, sympathetic outflow
Impaired pulmonary vasodilation
Augmented pulmonary vasoconstriction
Exaggerated hypoxic pulmonary hypertension

Negative Pressure Pulmonary Edema: *Pathophysiology*

Authors:
Mackenzie Gault
Reviewers:
Arsalan Ahmad
Melinda Davis*
* *MD at time of publication*

Airway Obstruction
Involuntarily biting ET tube or laryngospasm most common

↓ ventilation to lungs

Hypoxia

Detected by peripheral chemoreceptors

Sympathetic stimulation

Patient tries to inspire forcefully against obstruction

Highly negative intrathoracic pressure

Acute ↑ in systemic venous return to right heart

↑ pulmonary blood volume

↑ pulmonary arterial + capillary pressure

↓ pulmonary interstitial pressure

↑ trans-capillary pressure gradient

Fluid pushed out of pulmonary capillaries into the interstitium

Negative Pressure Pulmonary Edema:
Fluid in lungs caused by highly negative intrathoracic pressure

Fluid surrounds alveoli

CXR: diffuse bilateral infiltrates

↓ diffusion of alveolar O_2 into pulmonary capillaries

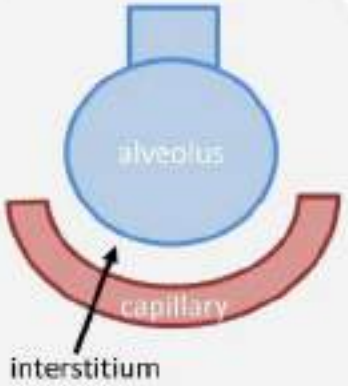
↓ PO_2
↓ O_2 Sats

If *severe*: pressure and fluid build-up damages capillary and alveolar walls

Fluid & red blood cells from capillaries enter alveoli and are coughed up

Frothy pink sputum

Notes:
ET tube: Endotrachial tube
Laryngospasm: spasm of vocal cords; may occur on extubation
CXR: Chest X-Ray



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Common and uncommon precipitating factors associated with hospitalization for ADHF

Dietary and medication related causes
Dietary indiscretion - excessive salt or water intake
Nonadherence to medications
Iatrogenic volume expansion
Progressive cardiac dysfunction
Progression of underlying cardiac dysfunction
Physical, emotional, and environmental stress
Cardiac toxins:
Alcohol
Cocaine
Right ventricular pacing
Cardiac causes not primarily myocardial in origin
Cardiac arrhythmias:
Atrial fibrillation with a rapid ventricular response
Ventricular tachycardia
Marked bradycardia
Conduction abnormalities
Uncontrolled hypertension
Acute myocardial infarction
Myocardial ischemia
Valvular disease:
Progressive mitral regurgitation
Non-cardiac causes
Pulmonary disease - pulmonary embolus, COPD
Anemia, from bleeding or relative lack of erythropoietin or bone marrow suppression
Systemic infection; especially pulmonary infection
Thyroid disorders
Adverse cardiovascular effects of medications
Cardiac depressant medications
Nondihydropyridine calcium antagonists
Type Ia and Ic antiarrhythmic agents
Sodium retaining medications
Steroids
Nonsteroidal anti-inflammatory drugs
Medications that reduce contractility
Anthracyclines and other chemotherapeutic agents

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