Congestive heart failure

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Definition

Congestive heart failure (CHF): a **clinical syndrome** in which the heart is unable to pump enough blood to meet the metabolic needs of the body; characterized by ventricular dysfunction that results in **low cardiac output**

Systolic dysfunction: CHF
with **reduced** stroke volume and **ejection fraction** (EF)
Diastolic dysfunction: CHF with
reduced stroke
volume and **preserved ejection fraction**

Right heart failure (RHF): CHF due to right ventricular dysfunction; characterized by **backward heart failure**

Left heart failure (LHF): CHF due to left ventricular dysfunction; characterized by **forward heart failure**

Biventricular (global) CHF: CHF in which both the left and right ventricle are affected, resulting in **simultaneous backward and forward CHF**

Chronic compensated CHF:
clinically compensated CHF; the patient has signs of CHF on echocardiography but is asymptomatic or symptomatic and stable (see “Diagnostics” below)
Acute decompensated CHF: sudden deterioration of CHF or new onset of severe CHF due to an acute cardiac condition (e.g., myocardial infarction)

Epidemiology

Systolic heart disease is the most common form of CHF overall.

Etiology

	Systolic dysfunction (reduced EF)	Diastolic dysfunction (preserved EF)
General causes	• Coronary artery disease, myocardial infarction • Arterial hypertension • Valvular heart disease • Diabetes mellitus (diabetic cardiomyopathy) • Renal disease • Infiltrative diseases (e.g., hemochromatosis, amyloidosis)	
Specific causes	• Cardiac arrhythmias • **Dilated cardiomyopathy** (e.g., Chagas disease, chronic alcohol use, idiopathic) • Myocarditis	• Constrictive pericarditis • **Restrictive or hypertrophic cardiomyopathy** • Pericardial tamponade
Further risk factors	• Obesity • Smoking • COPD • Heavy drug (recreational and prescription) and alcohol abuse	

Systolic ventricular dysfunction (most common) due to: Reduced contractility: Damage and loss of myocytes reduce ventricular contractility and stroke volume.

Diastolic ventricular dysfunction due to: Decreased ventricular compliance: increased stiffness or impaired relaxation of the ventricle

Consequences of systolic and diastolic dysfunction

Forward failure: reduced cardiac output → **poor organ perfusion** → organ dysfunction (e.g., hypotension, renal dysfunction)

Backward failure

Increased left-ventricular volume and pressure → backup of blood into lungs → increased pulmonary capillary pressure → **cardiogenic pulmonary edema**

Reduced cardiac output → systemic venous congestion → **edema** and progressive **congestion** of internal organs

Compensation mechanisms

Aim: maintain cardiac output if stroke volume is reduced

- **↑ Adrenergic activity** → increase in heart rate, blood pressure, and ventricular contractility

- Increase of renin-angiotensin-aldosterone system activity (**RAAS**): activated following decrease in renal perfusion secondary to reduction of stroke volume and cardiac output
  - ↑ Angiotensin II secretion → vasoconstriction → ↑ systemic blood pressure → ↑ afterload
    - Kidney: vasoconstriction of the efferent arterioles and, to a lesser degree, the afferent arterioles → ↓ net renal blood flow and ↑ intraglomerular pressure to maintain GFR
  - ↑ Aldosterone secretion → ↑ renal Na^+ and H_2O resorption → ↑ preload

- **Brain natriuretic peptide (BNP)**:
  ventricular myocyte hormone released in response to increased ventricular filling and stretching
  - ↑ Intracellular smooth
    muscle cGMP → vasodilation → hypotension and decreased pulmonary capillary wedge pressure
- CHF is characterized by reduced cardiac output that results in venous congestion and poor systemic perfusion!

General features of heart failure
•Nocturia •Fatigue •Tachycardia, various arrhythmias •Heart sounds: **S^3/S^4 gallop** •**Pulsus alternans**

Clinical features of left-sided heart failure

Pulmonary symptoms dominate

- **Dyspnea**, orthopnea
- **Pulmonary edema** in severe cases or acute decompensated heart failure (see below)
- Bilateral basilar rales may be audible on auscultation.
- **Paroxysmal nocturnal dyspnea**: nocturnal bouts of coughing and acute shortness of breath
- **Cardiac asthma**: increased pressure in the bronchial arteries results in airway compression, leading to bronchospasm [10]
- Laterally displaced apical heart beat (precordial palpation beyond the midclavicular line)

- **Forward failure**: cool extremities, cerebral and renal dysfunction, sweating (NYHA IV)

Clinical features of right-sided heart failure

Symptoms of fluid retention (backward failure) dominate

- **Peripheral pitting edema**
- Signs of increased central venous pressure (CVP)
  - **Jugular venous distention**: visible jugular venous congestion, also seen in biventricular heart failure
  - **Hepatojugular reflux**: jugular venous congestion induced by exerting manual pressure over the patient's liver → ↑ volume load on the right side of the heart → right heart is unable to pump additional blood volume → visible jugular venous distention persists for several seconds
- **Hepatic venous congestion**
  - Hepatosplenomegaly
  - Abdominal pain
  - Jaundice
  - Ascites
- Congestion of other organs, e.g., congestive gastritis or gastropathy (nausea, loss of appetite), renal congestion

- Forward failure less pronounced

High-output heart failure

Definition: heart failure **secondary** to conditions associated with a high-output state, in which cardiac output is elevated to meet the demands of peripheral tissue oxygenation

Etiology:

conditions that lead to increased cardiac demand (high-output state)

- Anemia
- Systemic arteriovenous fistulas
- Sepsis
- Hyperthyroidism
- Multiple myeloma
- Glomerulonephritis
- Polycythemia vera
- Wet beriberi (vitamin B_1 deficiency)
- Physiological causes: pregnancy, fever, exercise

Clinical features

Symptoms of low-output CHF; particularly tachycardia, tachypnea, low blood pressure, and jugular distention with an audible hum over the internal jugular vein

Pulsatile tinnitus

Bounding peripheral **pulses**

Laterally displaced apical heart beat

Midsystolic murmur, **S_3 gallop** (indicates rapid ventricular filling)

Diagnostics

Primarily a clinical diagnosis

X-ray and echocardiography: cardiomegaly

Therapy

Manage heart failure: symptom relief, hemodynamic stabilization

Treat underlying condition

Stages

NYHA functional classification

The NYHA (New York Heart Association) functional classification system is used to assess the patient's functional capacities (i.e., limitations of physical activity and symptoms) and has prognostic value.

NYHA class	Characteristics
Class I	No limitations of physical activity; **no symptoms** of CHF
Class II	Slight limitations of moderate or prolonged physical activity (e.g., symptoms after climbing 2 flights of stairs or heavy lifting); comfortable at rest
Class III	Marked limitations of physical activity (symptoms during daily activities like dressing, walking across rooms); comfortable only at rest
Class IV	Confined to bed, discomfort during any form of physical activity; **symptoms present at rest**

American Heart Association (AHA) Classification (2013)

The AHA classification system classifies patients according to their stage of disease. It takes objective findings (patient history, diagnostic findings) as well as symptoms of CHF into account.

Stages	Objective assessment	Corresponding NYHA functional class
Stage A	High risk of developing heart failure (e.g., pre-existing arterial hypertension, CAD, diabetes mellitus); no structural cardiac changes	No corresponding NYHA class
Stage B	Structural damage to the heart (e.g., infarct scars, dilatation, hypertrophy), without signs or symptoms of heart failure	NYHA I
Stage C	Structural damage to the heart + signs or symptoms of heart failure	NYHA I, II, III, IV
Stage D	Heart failure at its terminal stage	NYHA IV

Diagnostics

The presence of CHF means severe pathology and advanced etiology .

Heart failure is primarily a clinical diagnosis. Laboratory tests and imaging tests, including a chest x-ray and echocardiogram, are useful for evaluating the severity and cause of the condition.

Diagnostic approach

1. Medical history, including pre-existing conditions and history of alcohol and recreational or prescribed drug use
2. Initial evaluation involves a range of routine laboratory tests and a test for BNP level, ECG, and chest x-ray.
3. Echocardiography is the gold standard tool for assessing cardiac morphology and function, as well as investigating the underlying cause of CHF.
4. Other procedures (exercise testing, angiography) may be required for further investigation.

Laboratory analysis

- **Elevated BNP and NT-pro BNP**
  - High levels of BNP in patients with classic symptoms of CHF confirm the diagnosis (high predictive index).

	CHF unlikely	CHF likely
BNP (pg/mL)	< 100	> 500
NT-pro BNP (pg/mL)	< 300	> 450

- Elevated atrial natriuretic peptide (ANP):
- **Complete blood count**: may show **anemia**
- **Serum electrolyte levels**: hyponatremia → indicates a poor prognosis
- **Kidney function tests**: ↑ **creatinine**, ↓ sodium
- **Urine analysis**: rule out concurrent renal impairment
- **Fasting glucose**: to screen for diabetes mellitus, which is a common comorbidity
- **Fasting lipid profile**: to detect dyslipidemia associated with a higher cardiovascular risk

Electrocardiogram (ECG)

- ECG abnormalities in CHF are common, but are mostly nonspecific and nondiagnostic.
- Signs of left ventricular hypertrophy
  - ↑ QRS voltage (in the left chest leads and limb leads I and aVL) → positive Sokolow-Lyon index
  - ↑ QRS duration (incomplete or complete left bundle branch block)
  - Left axis deviation
- Assessment of prior or concurrent heart conditions
  - Previous or acute MI: see ECG changes in STEMI
  - Arrhythmias (e.g., atrial fibrillation, ventricular arrhythmias, sinus tachycardia or bradycardia, AV block)
- Signs of pericardial effusion and tamponade: low voltage ECG

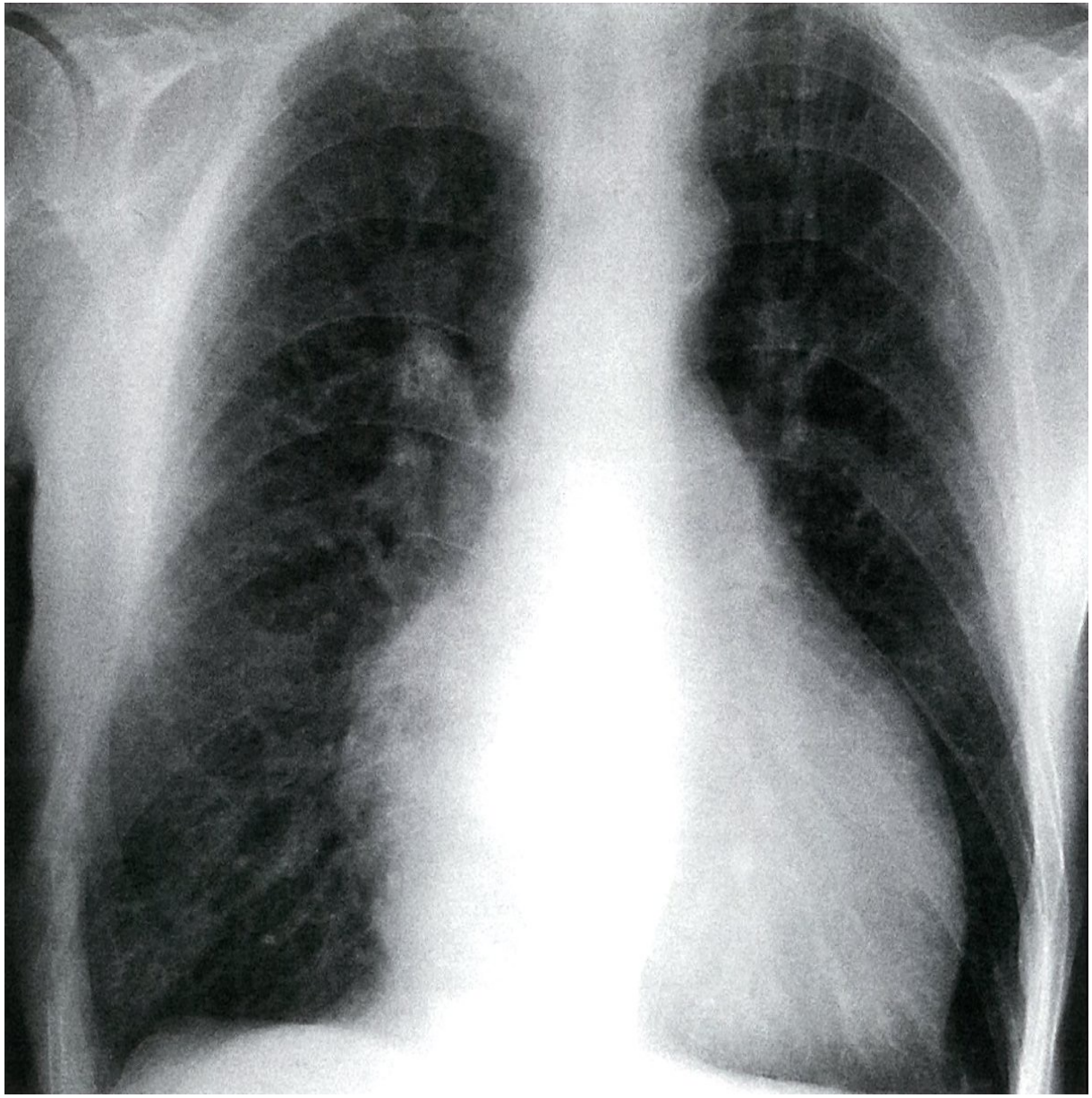
Chest x-ray

Useful diagnostic tool to evaluate a patient with dyspnea and differentiate CHF from pulmonary disease

Signs of cardiomegaly

- Cardiac-to-thoracic width ratio > 0.5
- **Boot-shaped heart** on PA view (due to left ventricular enlargement)

Assess pulmonary congestion (see x-ray findings in pulmonary congestion)

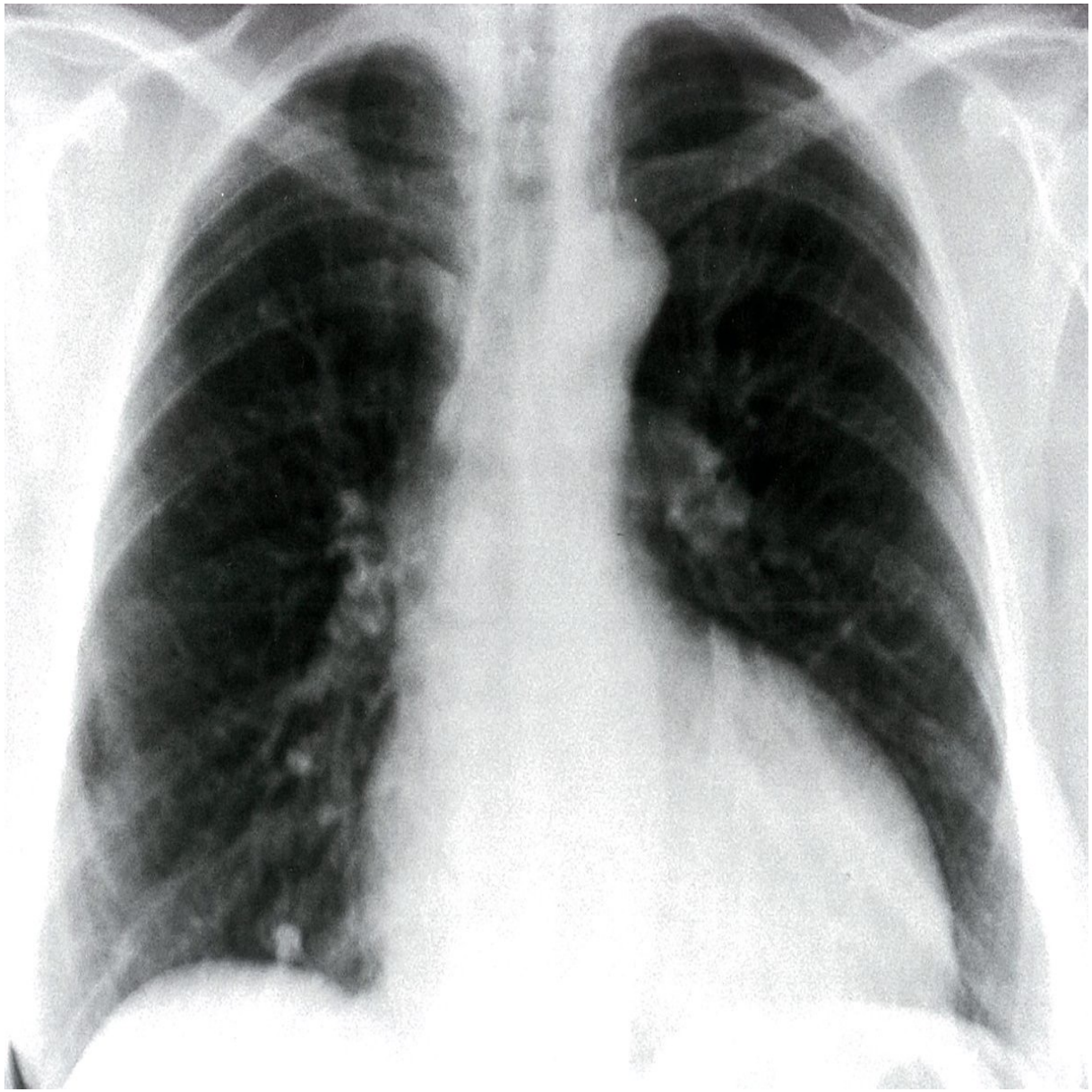


Left ventricular enlargement in aortic valve stenosis

Chest x-ray (PA view)

Enlargement of the heart at its left border can be seen (hatched overlay).

This enlargement is consistent with left ventricular enlargement, which leads to a cardiac-to-thoracic width ratio > 0.5.

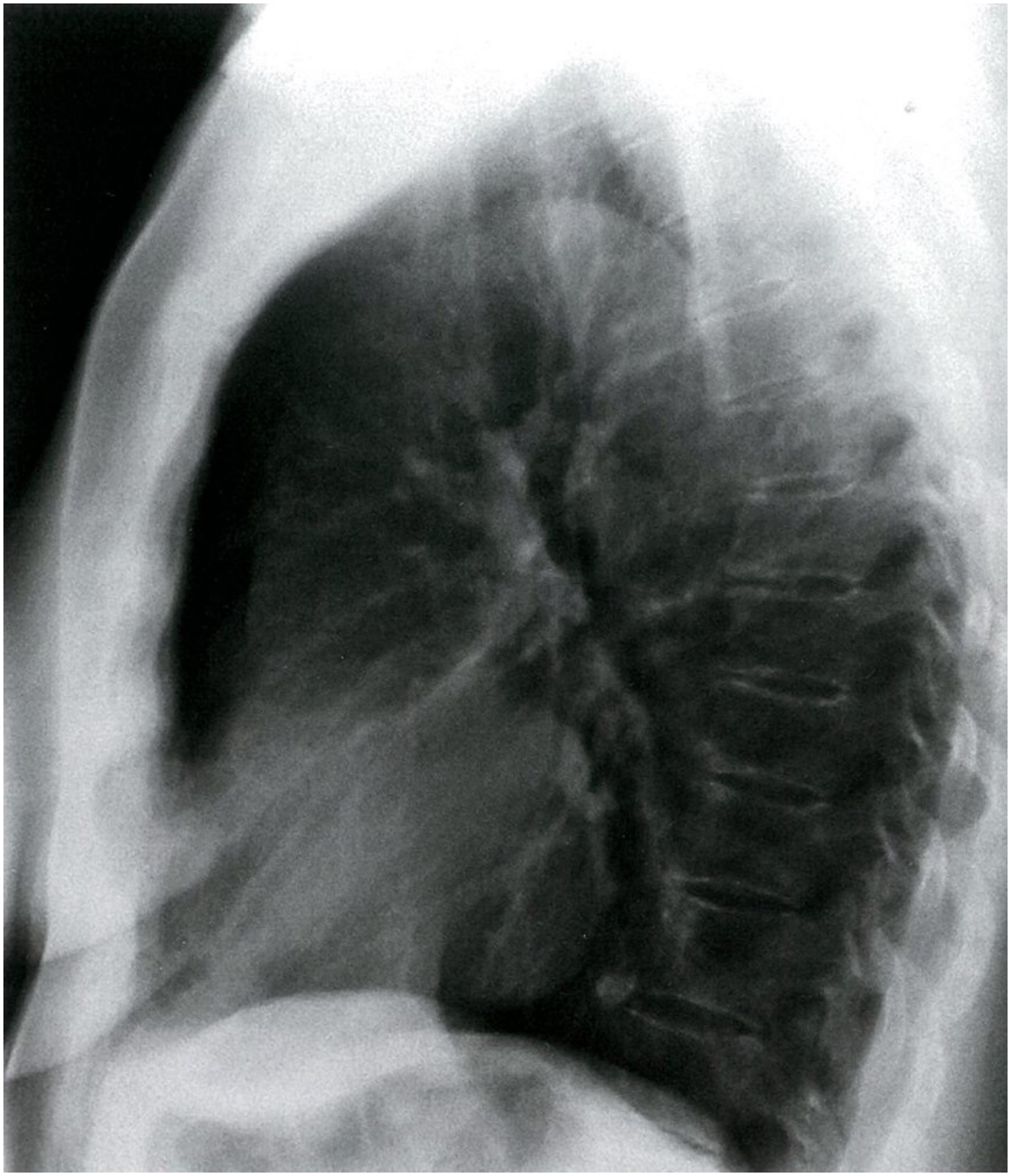


Left ventricular hypertrophy

Chest x-ray (PA view)

Compared to the normal heart size (green line), the heart appears to be enlarged at the left border, and there is widening of the cardiac base against the diaphragm (green overlay).

These findings are consistent with left ventricular hypertrophy.

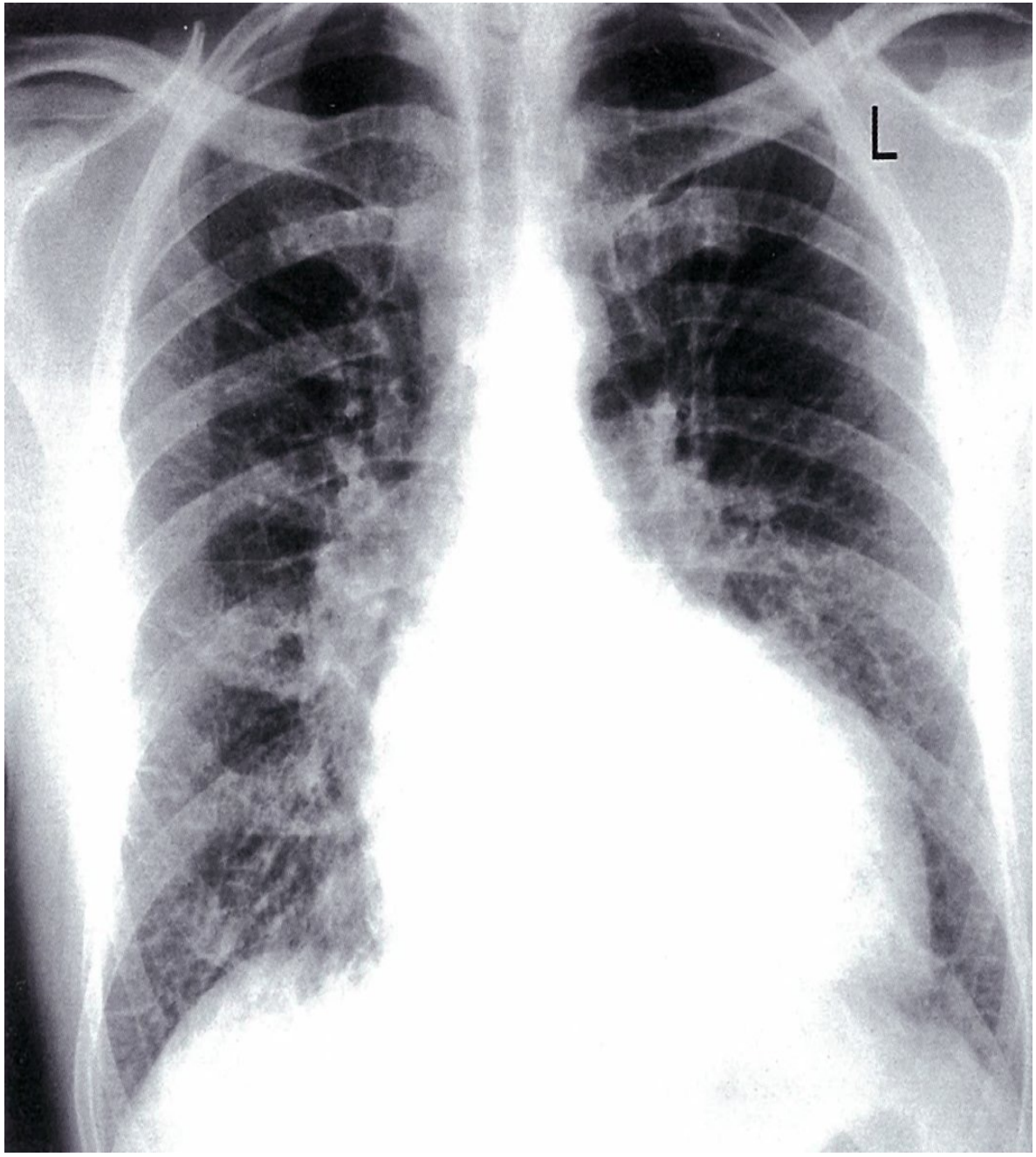


Left ventricular enlargement

Chest x-ray (lateral view)

There is convex enlargement of the dorsal cardiac border (green overlay), reducing the size of the retrocardiac space. Additionally, the ascending aorta appears to be dilated (white overlay and arrowheads).

These findings may be due to enlargement of the left ventricle, which forms the dorsal cardiac border on lateral view.



Pulmonary edema secondary to left-sided congestive heart failure.

Chest x-ray (PA view)

The left border of the heart appears enlarged (hatched green overlay), increasing the width of the cardiac silhouette (green arrow). There is increased opacity of the hilar region, indicating pulmonary vascular congestion (green overlay). Diffuse vascular markings, known as Kerley B lines, can be seen in the basal segments on both sides (dotted green lines).

These findings are characteristic of pulmonary edema secondary to heart failure.

Investigate etiology

Valvular heart disease

Wall motion abnormalities (indicate prior or acute MI)

Right ventricular strain

Further tests

Cardiac stress test (exercise tolerance test): to assess the functional impairment due to CHF or other conditions (particularly CHD!)

Radionuclide ventriculography : indicated to assess left ventricular volume and ejection fraction (LVEF)

Cardiac MRI: particularly useful for assessing cardiac morphology and function

- Cardiac size and volumes, wall thickness, valvular defects, wall motion abnormalities

Coronary angiography (left heart catheterization): indicated to detect/confirm CHD and possible percutaneous coronary intervention

Right heart catheterization: if pulmonary hypertension is suspected, to assess the severity of systolic dysfunction, and/or to differentiate between types of shock

- SvO_2: will be low in decompensated heart failure

Endomyocardial biopsy: may be performed if a specific diagnosis is suspected in patients with rapidly progressive clinical CHF or in case the results would alter the management of the patient, e.g., in amyloidosis

Treatment

General measures

Lifestyle modifications

- Salt restriction (< 3 g/day)
- Fluid restriction in patients with edema and/or hyponatremia
- Weight loss and exercise
- Cessation of smoking and alcohol consumption
- Immunization: pneumococcal vaccine and seasonal influenza vaccine

Patient education

- Self-monitoring and symptom recognition
- Daily weight check
  - Weight gain > 2 kg within 3 days: consult the doctor
- Monitoring of potential side effects (e.g., hypotension caused by ACE inhibitors, hyperkalemia caused by aldosterone-antagonists, sensitivity to sunlight caused by amiodarone)

Treat any underlying conditions and contributing comorbidities.

Drug	NYHA stages				Indications	Contraindications and important side effects	Benefits
	I	II	III	IV			
First-line drugs							
Diuretics (loop diuretics and thiazide diuretics)	(✓)	(✓)	✓	✓	•Begin treatment with loop diuretics (furosemide) to **treat volume overload** •Thiazides may be added for a synergistic effect	•Monitor for hypokalemia and hyponatremia, weight gain, and volume status	•Improve symptoms

Drug	NYHA stages				Indications	Contraindications and important side effects	Benefits
	I	II	III	IV			
ACE inhibitors	✓	✓	✓	✓	•Initiate treatment with ACE inhibitors to reduce preload, afterload, and improve cardiac output •If the patient does not tolerate drug (e.g., dry cough develops) → substitute with AT2-receptor blocker	•Monitor for hyperkalemia, hypotension, ↑ creatinine (renal impairment)	•**Improve symptoms and prognosis**
Beta blockers	(✓)	✓	✓	✓	•Add a beta blocker once the patient is **stable** on ACE inhibitor •Particularly beneficial for patients with hypertension and post-myocardial infarction	•Contraindicated in acute decompensated heart failure!	
Aldosterone antagonists		(✓)	✓	✓	•In select patients, an aldosterone antagonist may be beneficial. •If EF < 35%, and after myocardial infarction •Spironolactone; eplerenone as an alternative	•Monitor for hyperkalemia	

Drug	NYHA stages				Indications	Contraindications and important side effects	Benefits
	I	II	III	IV			
Second-line drugs							
Ivabradine		(✓)	(✓)	(✓)	•If the highest tolerable dose of beta blocker is reached and the patient is still symptomatic or if the patient has a contraindication to beta-blocker use •If EF < 35% and the patient has a sinus rhythm with a resting heart rate > 70/min	•Contraindicated in severe bradycardia	•Improves symptoms •Reduces hospitalization rate

Drug	NYHA stages				Indications	Contraindications and important side effects	Benefits
	I	II	III	IV			
Hydralazine plus nitrate			(✓)	(✓)	•If EF < 40%; particularly beneficial for African-American patients •Alternative if ACE inhibitors and AT1 blockers are not tolerated	•Monitor for volume depletion and hypotension	•Improves symptoms; may improve prognosis

Drug	NYHA stages				Indications	Contraindications and important side effects	Benefits
	I	II	III	IV			
Digoxin		(✓)	(✓)	(✓)	•In heart failure with reduced ejection fraction •If symptoms persist despite treatment with beta blocker, ACE inhibitor, diuretics, and aldosterone antagonists •May be given to control ventricular rate in atrial fibrillation (if beta blockers are contraindicated)	•Contraindi-cated in severe AV block	•Improves symptoms •Reduces hospitalization rate

Drug	NYHA stages				Indications	Contraindications and important side effects	Benefits
	I	II	III	IV			
ARNI (angiotensin receptor-neprilysinihibitor)	(✓)	(✓)	(✓)	(✓)	•Persistent or worsening symptoms despite adequate treatment regimen with first-line drugs •Administered as combination **valsartan-sacubitril**	•Angioedema, hypotension, hyperkalemia, and progression of chronic renal disease(↑ creatinine)	•Improves prognosis •Reduces hospitalization rate

Drug	NYHA stages				Indications	Contraindications and important side effects	Benefits
	I	II	III	IV			
Nesiritide (BNP derivative)					•Acute decompensated heart failure •Rarely used today due to side effects and longer half-life compared to other vasodilators (e.g., nitroglycerin)	•Contraindicated in patients with hypotension and/or cardiogenic shock •Adverse effects include hypotension and decrease in pulmonary capillary wedge pressure	
(✓): see “Indications” column for detailed information							

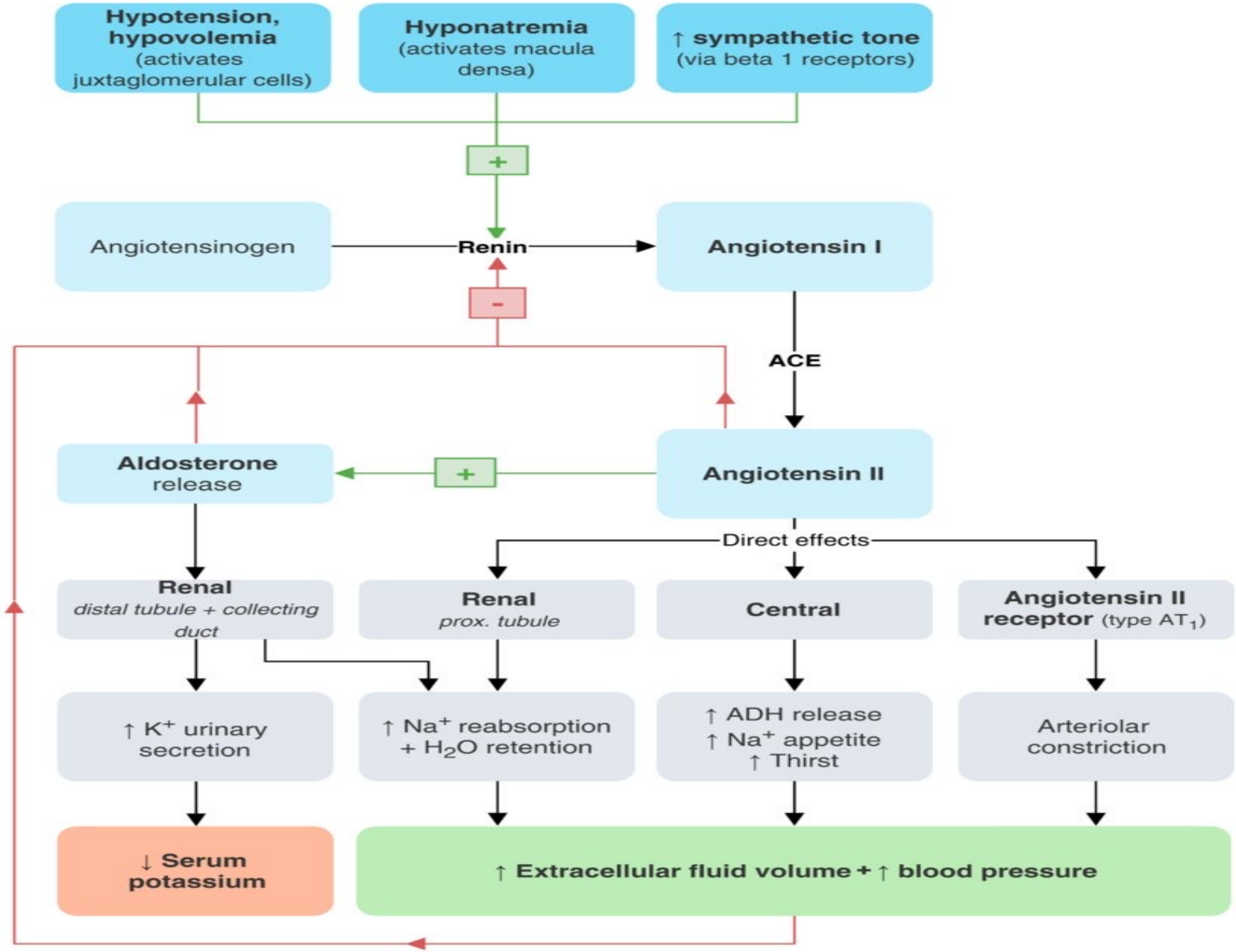
Drugs that **improve prognosis**: beta blockers, ACE inhibitors, and aldosterone antagonists, valsartan- sacubitrel !

Recently , FDA approved SGLT2 inhibitors for adults with heart failure with reduced ejection fraction to reduce the risk of cardiovascular death and hospitalization for heart failure.

Iron deficiency is a major finding that is present in about 50% of patients with heart failure irrespective of the left ventricular function and independent of anaemia.The current guidelines for treatment of chronic and acute heart failure acknowledge importance of iron deficiency correction and recommend (intravenous) iron supplementation for its treatment, iron deficiency remains frequently undertreated and insufficiently diagnosed in setting of the heart failure.

Drugs
that **improve symptoms**: diuretics and digoxin (significantly reduce the number of hospitalizations)!

Conducting regular blood tests to assess electrolyte levels (potassium and sodium) is mandatory if the patient is on diuretics!


Hypotension, hypovolemia (activates juxtaglomerular cells)
Hyponatremia (activates macula densa)
↑ sympathetic tone (via beta 1 receptors)
+
Angiotensinogen
Renin
Angiotensin I
-
ACE
Aldosterone release
+
Angiotensin II
Direct effects
Renal distal tubule + collecting duct
Renal prox. tubule
Central
Angiotensin II receptor (type AT1)
↑ K+ urinary secretion
↑ Na+ reabsorption + H2O retention
↑ ADH release
↑ Na+ appetite
↑ Thirst
Arteriolar constriction
↓ Serum potassium
↑ Extracellular fluid volume + ↑ blood pressure

Contraindicated drugs

- **NSAIDs**
  - Worsen renal perfusion (see "Side effects" of NSAIDs)
  - Reduce the effect of diuretics
  - May trigger acute cardiac decompensation
- **Calcium channel blockers (verapamil and diltiazem)**: negative inotropic effect; worsen symptoms and prognosis
- **Thiazolidinediones**: promote the progression of CHF (↑ fluid retention and edema) and increase the hospitalization rate
- **Moxonidine**: increases mortality in CHF with reduced ejection fraction (systolic dysfunction)

Invasive procedures

- **Implantable cardiac defibrillator** (ICD): prevents sudden cardiac death
  - Primary prophylaxis indications
    - CHF with **EF < 35%** and prior myocardial infarction/CHD
    - Increased risk of life-threatening cardiac arrhythmias
  - Secondary prophylaxis indications: history of sudden cardiac arrest, ventricular flutter, or ventricular fibrillation

- **Cardiac resynchronization therapy** (biventricular pacemaker): improves cardiac function
  - Indications: CHF with **EF < 35%**, dilated cardiomyopathy, and left bundle branch block
  - Can be combined with an ICD
- **Coronary revascularization** with PCTA or bypass surgery may be indicated if CAD is present.
- **Valvular surgery** if valvular heart defects are present
- **Ventricular assist devices**: may be implanted to support ventricular function; may be indicated for temporary or long-termsupport (e.g., to bridge time until transplantation) of decompensated CHF
- **Cardiac transplantation**: for patients with end-stage CHF (NYHA class IV), ejection fraction < 20%, and no other viable treatment options

Complications

- Acute decompensated heart failure
- Cardiorenal syndrome
- Cardiac arrhythmias
- Central sleep apnea syndrome
- Cardiogenic shock

- Stroke; increased risk of arterial thromboembolisms (especially with concurrent atrial fibrillation)
- Chronic kidney disease
- Cardiac cirrhosis (congestive hepatopathy): Cirrhosis due to chronic hepatic vein congestion in patients with right-sided heart failure.
- Venous stasis, leg ulcers

Acute decompensated heart failure

Cardiac decompensation is the most common reason for hospital admissions and is the most important complication of congestive heart failure.

Etiology

ADHF typically occurs in patients who have a history of CHF or other cardiac conditions in which an acute cause precipitates the deterioration of cardiac function.

- Exacerbation of congestive heart failure (e.g., through pneumonia, anemia, volume overload, medication noncompliance)
- Acute myocardial infarction

- Atrial fibrillation, severe bradycardia, and other arrhythmias
- Myocarditis
- Hypertensive crisis
- Pulmonary embolism
- Pericardial tamponade
- Aortic dissection
- Cardiotoxic substances
- Renal failure
- Cardiodepressant medication (e.g., beta blockers, CCBs)

Clinical features

- Rapid exacerbation of symptoms of CHF
  (see symptoms of left heart failure and symptoms of right heart failure)

- **Pulmonary congestion** with:
  - Acute, severe dyspnea and orthopnea; worse when supine
  - Cough (occasionally with frothing, blood-tinged sputum)
  - Cyanosis
  - Auscultation of the lungs: rales accompanied by wheezing
  - Flash pulmonary edema: rapid, life-threatening accumulation of fluid associated with the risk of acute respiratory distress
- Weakness, fatigue, and cold, clammy skin

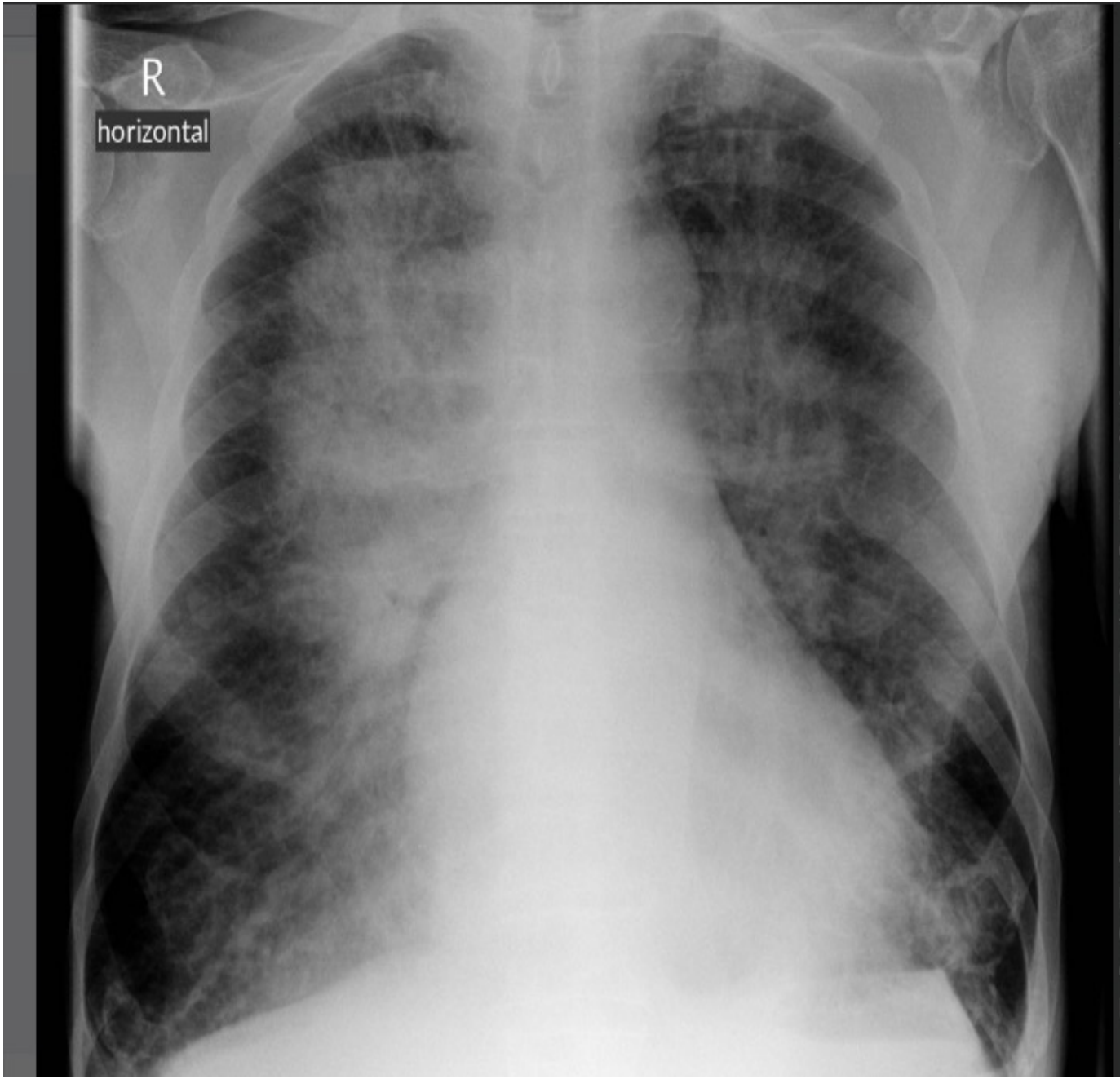
Diagnostics

- **X-ray findings in pulmonary congestion**
  - Cardiomegaly
  - **Prominent pulmonary vessels** and perihilar pulmonary edema (butterfly or "**bat's wings**" appearance of the hilar shadow)
  - **Kerley B lines**: visible horizontal interlobular septa caused by pulmonary edema
  - Basilar edema
  - Bilateral pleural effusions

- **Sputum analysis**: **heart failure cells** (hemosiderin-containing cells)

- **Thoracentesis**
  - Indicated if the etiology of the pleural effusion is unclear
  - Pleural fluid analysis: Transudate effusions are typical of cardiogenic causes.

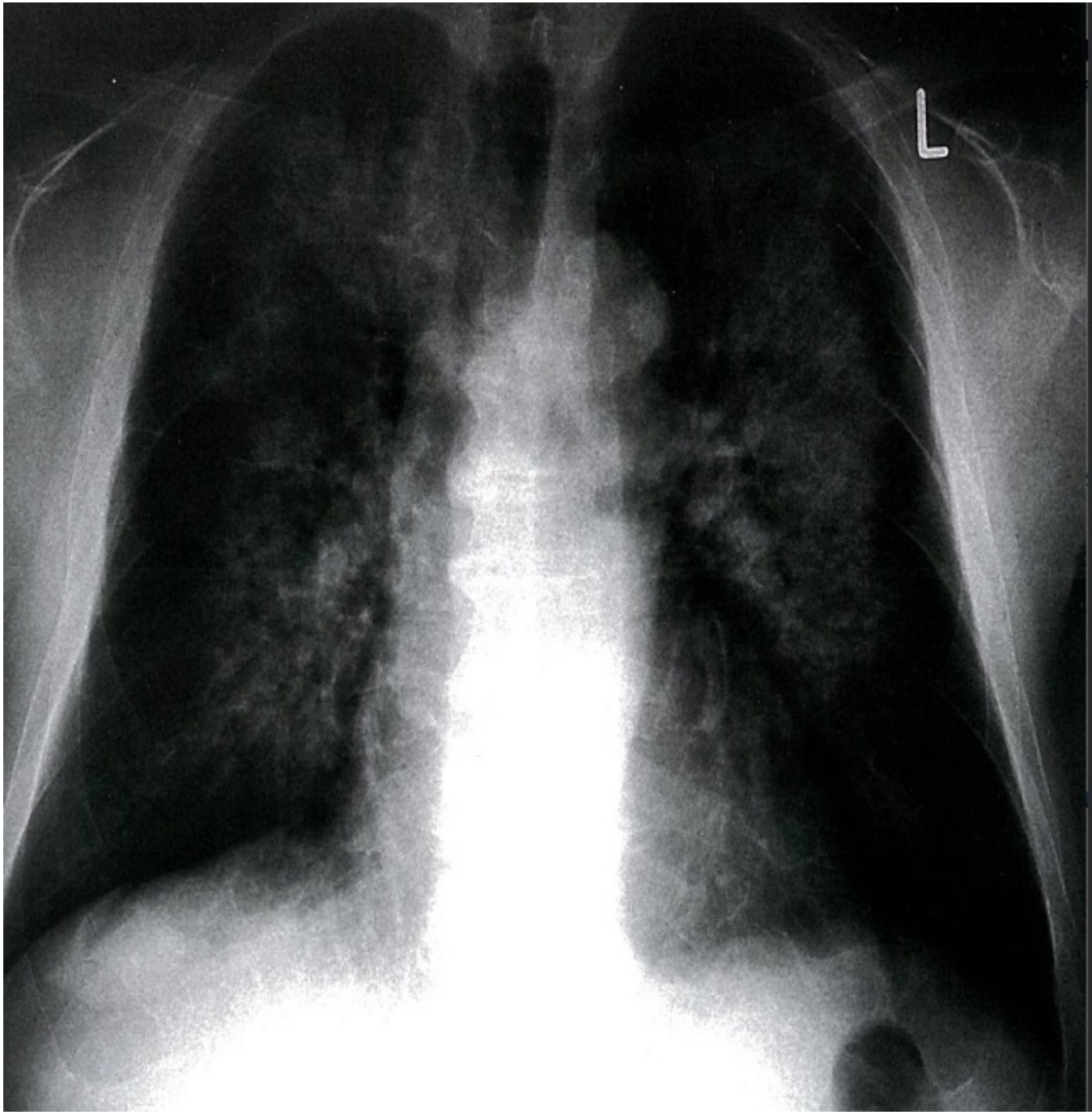
The radiologic signs of **pulmonary congestion** can be remembered with "**ABCDE**": A = **A**lveolar edema (bat's wings), B = Kerley **B** lines (interstitial edema), C = **C**ardiomegaly, D = **D**ilated prominent pulmonary vessels, and E= **E**ffusions!



Perihilar pulmonary edema in acute left-sided heart failure

Chest x-ray (AP view)

Perihilar, interstitial opacities can be seen, which give the hilar shadow a butterfly or “bat wing” appearance. The cardiac silhouette appears bilaterally enlarged, indicating cardiomegaly (hatched green overlay). The costophrenic angles, although not shown in their entirety, appear to be covered by an are of increased opacity (green arrows), suggesting pleural effusion. There are thin linear opacities radiating from the hilum to the apex (orange dotted lines) and from the hilum to the lower periphery of the lungs (white dotted lines). These opacities are consistent with Kerley A and Kerley B lines.



Diffuse bilateral pulmonary edema

Chest x-ray (PA view)

There are patchy areas of increased opacity bilaterally (green overlay).

The finding of symmetrical opacities in both lungs is referred to as Bat wing opacities. These suggest pulmonary edema secondary to cardiac decompensation.

Differential diagnosis of pulmonary edema and respiratory distress

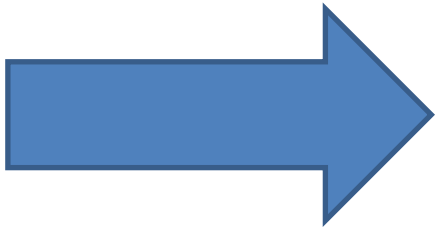
Noncardiogenic pulmonary edema due to ARDS, pulmonary embolism, transfusion-related acute lung injury, high altitude

Asthma

Pneumonia

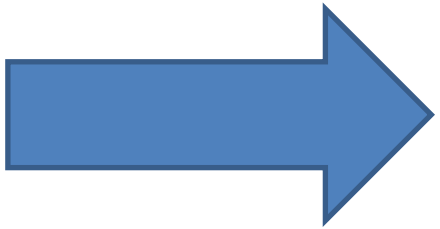
Treatment

- Sufficient oxygenation and ventilation ; assisted ventilation as needed (e.g., CPAP).
- Fluid management:
  - Aggressive diuresis (e.g., IV furosemide) to reduce volume overload
  - Vasodilators: (e.g., IV nitroglycerine) can be considered as adjunct treatment in patients without hypotension.



- Hemodynamic stabilization: **inotropes (e.g., dobutamine)** in case of systolic dysfunction
- Treat the cause of decompensation.
- Hemodialysis if volume overload is symptomatic (pulmonary edema, pleural effusion, ascites) and resistant to treatment
- ECLS may temporarily substitute pulmonary function.
- Ventricular assist devices (see “Treatment of heart failure” above)

Beta blockers must be used cautiously in decompensated heart failure!

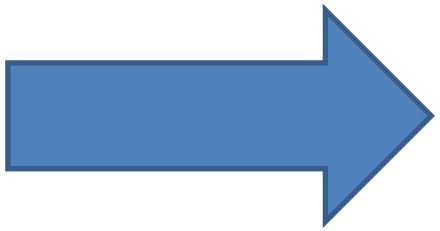


Management of **ADHF** can be remembered with "**LMNOP**": L = **L**asix (furosemide), M = **M**orphine, N = **N**itrates, O= **O**xygen, P = **P**osition (with elevated upper body).

Cardiorenal syndrome

Cardiorenal syndrome is a complication of acute heart failure and CHF.

Definition: a complex syndrome in which renal function progressively declines as a result of severe cardiac dysfunction; occurs in ~ 20–30% of cases of ADHF

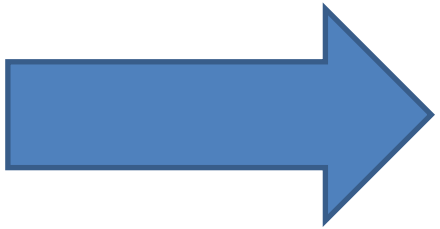


Pathophysiology

Cardiac forward failure → renal hypoperfusion → prerenal kidney failure

Cardiac backward failure → systemic venous congestion → renal venous congestion → decreased transglomerular pressure gradient → ↓ GFR→ worsening kidney function

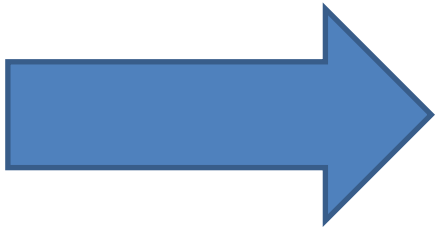
RAAS activation → salt and fluid retention, hypertension → hypertensive nephropathy



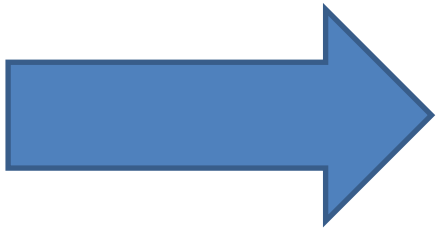
Diagnosis: ↓ GFR, ↑ creatinine that cannot be explained by underlying kidney disease
Treatment: treat heart failure; manage renal failure (see treatment of acute renal injury)
Prognosis: CHF with reduced GFR is associated with a poor prognosis.

Prognosis

- The prognosis depends on the patient, type and severity of heart disease, medication regimens, and lifestyle changes.
- The prognosis for patients with preserved EF is similar to or better than for patients with decreased EF
- Risk stratification scales may be used to evaluate the prognosis (e.g., CHARM and CORONA risk scores).



- **Factors associated with worse prognosis**Elevated BNP
- Hyponatremia
- Systolic BP < 120 mm Hg
- Diabetes
- Anemia
- Weight loss or underweight
- S_3 heart sound
- Implantable cardioverter-defibrillator use
- Frequent hospitalizations due to CHF



1-year survival according to NYHA stage

- Stage I: ~ 95%
- Stage II: ~ 85%
- Stage III: ~ 85%
- Stage IV: ~ 35%