Frank-Starling mechanism

- Definition: a law that describes the relationship between end-diastolic volume and cardiac stroke volume
  - Cardiac contractility is directly related to the wall tension of the myocardium.
    - An increase in end-diastolic volume (preload) will cause the myocardium to stretch (↑ end-diastolic length of cardiac muscle fibers), which increases contractility (↑ force of contraction) and results in increased stroke volume in order to maintain cardiac output.

Underlying mechanism of reduced cardiac output

- Heart failure with reduced ejection fraction (HFrEF)
  - Reduced contractility → systolic ventricular dysfunction → decreased left ventricular ejection fraction (LVEF) → decreased cardiac output
  - Causes include:
    - Damage and loss of myocytes (e.g., following myocardial infarction, coronary artery disease, dilated cardiomyopathy)
    - Cardiac arrhythmias
    - High-output conditions
- Heart failure with preserved ejection fraction (HFpEF)
  - Decreased ventricular compliance → diastolic ventricular dys function → reduced ventricular filling and increased diastolic pressure → decreased cardiac output (while the left ventricular ejection fraction remains normal)
  - Causes include:
    - Increased stiffness of the ventricle (e.g., long-standing arterial hypertension with ventricular wall hypertrophy, restrictive cardiomyopathy)
    - Impaired relaxation of the ventricle (e.g., constrictive pericarditis, pericardial tamponade)
- Left-sided heart failure (HFrEF and/or HFpEF)
  - Increased left ventricular afterload: increased mean aortic pressure; outflow obstruction
  - Incre ased left ventricular preload: left ventricular volume overload
- Right-sided heart failure
  - Increased right ventricular afterload: increase in pulmonary artery pressure
  - Increased right ventricular preload: right ventricular volume overload

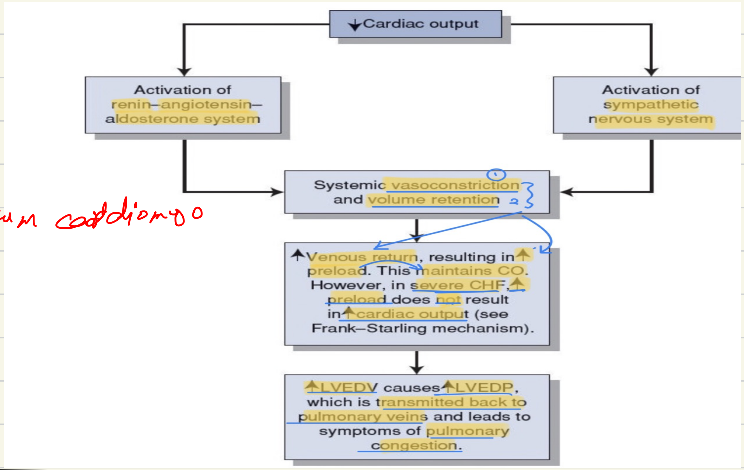
Congestive heart failure:- (CHF)

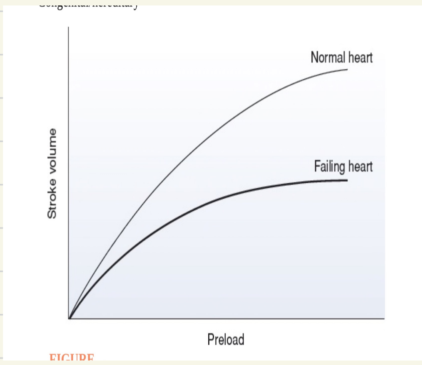
→ HF with reduced EF
→ HF with preserved EF

heart's inability to meet the body's circulatory demands under nL physiology condition

Often, both systolic and diastolic dysfunctions are present simultaneously.

Frank–Starling relationship
In a normal heart, increasing preload results in greater contractility.
When preload is low (at rest), there is little difference in performance between a normal and a failing heart. However, with exertion a failing heart produces relatively less contractility and symptoms occur





high out put HF: an increase in CO is needed for the requirement of peripheral tissues for O_2

Causes
- chronic anemia
- Pregnancy → paripartum cardiomyo
- Hyperthy
- Paget di
- MR
- aortic insufficiency
- AV fistula
- wet beriberi (caused by thiamine deficiency)

Consequences of decompensated heart failure

- Forward failure: reduced cardiac output → poor organ perfusion → organ dysfunction
- Backward failure
  - Left ventricle:
    increased left-ventricular volumes or pressures → backup of blood into lungs → increased pulmonary capillary pressure → cardiogenic pulmonary edema (presenting with orthopnea) and increased pulmonary artery pressure
  - Right ventricle:
    increased pulmonary artery pressure → reduced right-sided cardiac output → systemic venous congestion → peripheral edema and progressive congestion of internal organs (e.g., liver, stomach)
  - Nutmeg liver: the macroscopic appearance of the liver which resembles a nutmeg seed due to ischemia and fatty degeneration from hepatic venous congestion

Systolic dys. HFrEF

EF < 40
: impaired contractility

Causes
- IHD / after MI recently
- idiopathic
- HTN
- Myocarditis (Postviral, giant cell, AI)
- Alcohol, cocaine, methamphetamine, anthracycline trastuzumab
- infiltrative di (amyloidosis, sarcoidosis, hemochromatosis, wilson di
- Radiation therapy
- Thyroid di
- peripartium cardiomyopathy
- infections di (Chagas di, HIV, endocarditis causing valvular di)
- Valvular heart di (MR (usually), AS, AR)
- High out put heart failure
- congenital / heredity

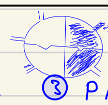
HFPEF diastolic dys function

EF > 50
→ impaired vent. filling during diastole (impaired relaxation or increased stiffness of vent. or both)

Echocardiogram shows impaired relaxation of left ventricle

causes
1. HTN leading to myocardial hypertrophy (m/c of diastolic dys func.)
2. valvular di (AS, AR, MS)
3. Restrictive cardiomyopathy (Amyloidosis, sarcoidosis, hemochromatosis)
3. hypertrophic cardiomyopathy

left vent failure

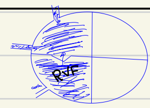


1. Dyspnea 2. orthopnea 3. PND
4. impairment memory and confusion (advanced)
5. diaphoresis and cool extremities (desperately ill)
6. cardiac asthma — Increased pressure in the bronchial arteries → airway compression
7. nocturnal cough (nonproductive)

Signs:
1. Displaced point of maximal impulse
2. gallop (S3)
   - Rapid filling phase “into” a noncompliant left ventricular chamber
   - May be normal finding in children; in adults, usually associated with CHF May be difficult to hear, but is among the most specific signs of CHF Heard best at apex with bell of stethoscope
   - The sequence in the cardiac cycle for S3: S3 follows S2
3. gallop (S4)
   - Sound of atrial systole as blood is ejected into a noncompliant, or stiff, left ventricular chamber
   - Heard best at left sternal border with bell of stethoscope
   - The sequence in the cardiac cycle for S4: S4 precedes S1
- crackles / rales at lung bases
  - Caused by fluid spilling into alveoli; indicates pulmonary edema
  - Rales heard over lung bases suggest at least moderate severity of left ventricular heart failure
- Dullness / decreased tactile fremituse of the lower lung field caused by pleural effusion
- coolness and pulor of lower extermities
- Increased intensity of pulmonic component of second heart sound suggests pulmonary HTN (heard over left upper sternal border)

S/S? syndrome stress induce cardio myopathy

Rt. vent failure

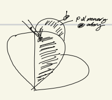


1. peripheral pefting edema
   pedal edema lacks specificity as an isolated finding. In the elderly, it is more likely to be secondary to venous insufficiency
2. Hepatic venous congestion symp
3. nocturia due to increased venous return with elevation of legs
4. jugular venous distension
5. hepatomegaly / hepatojugular reflex
6. Rt vent. heaves (pHTN)
7. acites
8. kussmaul sign

Given enough time, left-sided heart failure can lead to right-sided heart failure (most common cause right-sided heart failure)
a. Patients may present with sign/symptoms of both right-and left-sided HF

General features of heart failure
- Nocturia
- Fatigue
- Tachycardia,various arrhythmias
- S3/S4 gallop
- Pulsus alternans
- Cachexia

NYHA class I: Symptoms only occur with vigorous activities, such as playing a sport. Patients are nearly asymptomatic
NYHA class II: Symptoms occur with prolonged or moderate exertion, such as climbing a flight of stairs or carrying heavy packages. Slight limitation of activities.
NYHA class III: Symptoms occur with usual activities of daily living, such as walking across the room or getting dressed. Markedly limiting
NYHA class IV: Symptoms occur at rest. Incapacitating.



CHF → symptoms التشخيص

① CBC
② BNP (assess renal injury due to volume overload or cardiorenal syn.)
③ cardiac enz to rule out MI
④ BNP
⑤ CXR (pulmonary edema, cardiomegaly, rule out COPD)
⑥ ECG
⑦ Echo (estimate EF, rule out pericardial effusion)

DX:

① Chestx-ray(CXR)
a. Cardiomegaly
b. Kerley B lines are short horizontal lines near periphery of the lung near the costophrenic angles, and indicate pulmonary congestion secondary to dilation of pulmonary lymphatic vessels c. Prominent interstitial markings d. Pleural effusion

② Echocardiogram(transthoracic)
a. Initial test of choice—should be performed whenever CHF is suspected based on history, examination, or CXR.
b. Useful indetermining whether systolic or diastolic dysfunction predominates, and determines whether the cause of CHF is due to a myocardial, valvular, or pericardial process.
c. Estimates EF(veryimportant):Patients with systolic dysfunction (EF<40%) should be distinguished from patients with preserved left ventricular function (EF >40%).
d. Shows chamber dilation and/or hypertrophy.

③ B- type natriuretic peptide is released from the ventricles in response to ventricular volume expansion and pressure overload.

- BNP > 100 pg/mL → decompensated CHF
- BNP → differentiating between dyspnea caused by CHF and COPD
- N-terminal pro-BNP (NT-pro BNP) → depends on age; < 300 virtually excludes the Dx of HF.
- falsy low in obese

④ ECG is usually nonspecific but can be useful for detecting chamber enlargement and presence of ischemic heart disease or prior MI.

⑤ Radionuclide ventriculography using technetium-99m("nuclear ventriculography"). Also called multigated acquisition (MUGA) scan.

⑥ Cardiac catheterization can provide valuable quantitative information regarding diastolic and systolic dysfunction, and can clarify the cause of CHF if noninvasive test results are equivocal. Consider coronary angiography to exclude CAD as an underlying cause of CHF.

⑦ Stress testing
a. Identifies ischemia and/or infarction
b. Quantifies level of conditioning
c. Can differentiate cardiac versus pulmonary etiology of dyspnea
d. Assesses dynamic responses of HR,heart rhythm,and BP

HTN is a common cause of CHF and should be treated. Goal is to reduce preload and afterload.

Monitoring a patient with CHF:
Weight—unexplained weight gain can be an early sign of worsening CHF
Clinical manifestations (exercise tolerance is key); peripheral edema
Laboratory values (electrolytes, K, BUN, creatinine levels; serum digoxin level, if applicable)

Mild CHF (NYHA Classes I to II)
Mild restriction of sodium intake (no-added-salt diet of 4-g sodium) and physical activity.
Start a loop diuretic if volume overload or pulmonary congestion is present.
Use an ACE inhibitor as a first-line agent.

Mild to Moderate CHF (NYHA Classes II to III)
Start a diuretic (loop diuretic) and an ACE inhibitor.
Add a β-blocker if moderate disease (class II or III) is present and the response to standard treatment is suboptimal.

Moderate to Severe CHF (NYHA Classes III to IV)
Can add digoxin (to loop diuretic and ACE inhibitor) for the relief of symptoms in patients with systolic dysfunction. (It does not improve mortality.)
Add spironolactone or eplerenone if EF <35%

Ventricular assist device (VAD) may be used to support the left ventricle (LVAD), right ventricle (RVAD), or both ventricles (BiVAD).

The pump is implanted in the abdominal cavity with cannulation to the heart. The system controller and battery are worn externally.

These devices may allow for a patient requiring continuous hospitalization to eventually be discharged with relatively straightforward outpatient follow-up. Patients may live with these devices for a year or more.

Lifelong anticoagulation with heparin or warfarin is essential without exceptions as these devices are very thrombogenic.

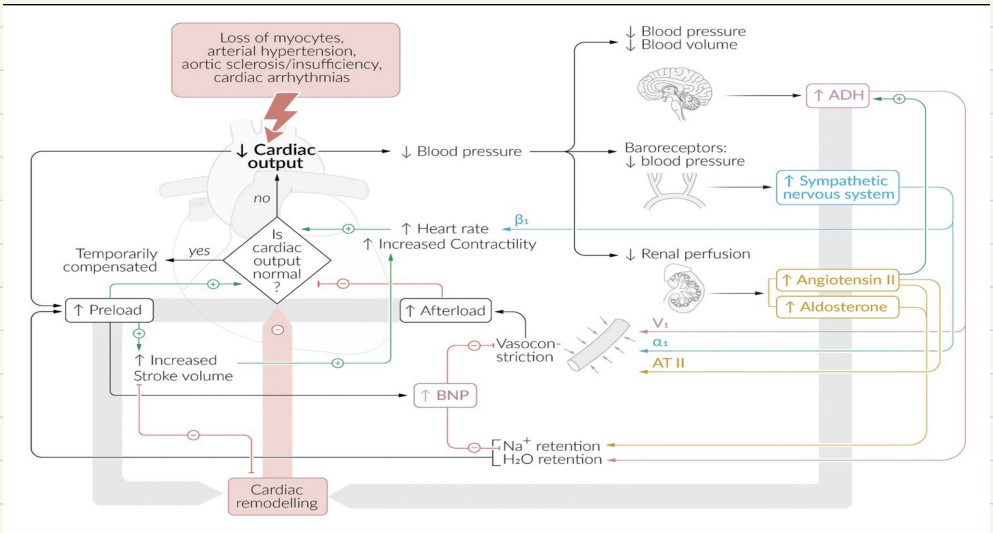
VADs may also be used for hemodynamic support related to the acute treatment of a STEMI or other cardiac pathology (i.e., while recovering from a severe case of myocarditis).

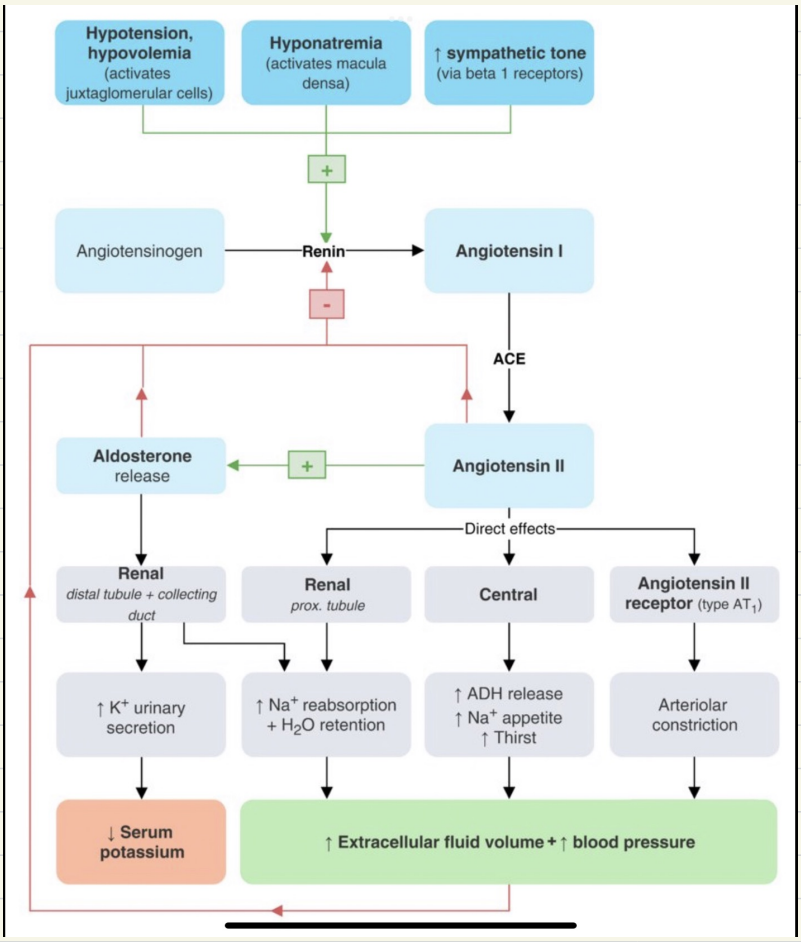
Acute Decompensated Heart Failure

A. Acute dyspnea associated with elevated left-sided filling pressures, with or without pulmonary edema.
B. Most commonly due to LV systolic or diastolic dysfunction.
C. Flash pulmonary edema refers to a severe form of heart failure with rapid accumulation of fluid in the lungs.
B. Diagnosis
A. Differential includespulmonaryembolism,asthma,andpneumonia,allofwhich can cause rapid respiratory distress.
B. Diagnostic tests include ECG,CXR,ABG,B-type natriuretic peptide(BNP), echocardiogram, and possible coronary angiogram if indicated.

Treatment ←

Natriuretic peptide levels in the diagnosis of heart failure [8]		
	Heart failure unlikely	Heart failure likely
BNP (in pg/mL)	< 100	> 400
NT-proBNP (in pg/mL)	< 300	> 900


Loss of myocytes, arterial hypertension, aortic sclerosis/insufficiency, cardiac arrhythmias
↓ Cardiac output
↓ Blood pressure
↓ Blood pressure
↓ Blood volume
↑ ADH
Baroreceptors: ↓ blood pressure
↑ Sympathetic nervous system
no
Is cardiac output normal ?
yes
Temporarily compensated
↑ Heart rate
↑ Increased Contractility
β1
↓ Renal perfusion
↑ Angiotensin II
↑ Aldosterone
↑ Preload
↑ Afterload
V1
α1
AT II
Vasocon-striction
↑ Increased Stroke volume
↑ BNP
Na+ retention
H2O retention
Cardiac remodelling


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