Heart failure

Objectives:

- Different classifications of heart failure.
- Causes and precipitating factors for heart failure decompensation.
- Pathophysiology, therapies that improve survival, and prognosis.
- Diagnostic tests of HF.
- Different treatment of HF.
- Side effects of medication of HF.
- Management of cardiac risk factors for HF.
- Role of devices and lifestyle in HF treatment.

Team members: Essam Alshahrani, Fahad Alzahrani, Mohammed Bagais, Rana Barasin.

Team leader: Nora AlSahli.

Revised by: Maha AlGhamdi.

Resources: 435 team + Davidson + Kumar + Step up.

- Editing file
- Feedback

Introduction

NOTE: Based on what doctor Khalid said, cardiomyopathy part is for your own knowledge.

★ General principles: (watch this)

- **Ejection Fraction (EF)** is a measurement of how much blood the left ventricle pumps out with each contraction, Normally between 50% and 70%. (EF= SV/EDV) An ejection fraction of 60 percent means that 60 percent of the total amount of blood in the left ventricle is pushed out with each heartbeat.
- **Preload is the end-diastolic volume (EDV)** at the beginning of systole. It's The amount of ventricular stretch at the end of diastole.
- **Afterload is the ventricular pressure at the end of systole (ESP)**. The amount of resistance the heart must overcome to open the aortic valve and push the blood volume out into the systemic Circulation.
- **Cardiac output** is simply the amount of blood pumped by the heart per minute. Necessarily, the cardiac output is the product of the **heart rate**, which is the number of beats per minute, and the **stroke volume**, which is amount pumped per beat: **CO = HR X SV** (Note : SV = EDV - ESV). Cardiac output at rest is about 5 liters/minute (4900 ml/min). Cardiac output is determined by preload (the volume and pressure of blood in the ventricles at the end of diastole), afterload (the volume and pressure of blood in the ventricles during systole) and myocardial contractility; this is the basis of *Starling's Law.*

• Heart Failure:

- ➔ Heart failure is **a complex syndrome**[1] that can result from any structural or functional cardiac disorder that impairs the ability of the ventricle to fill with or eject blood.
- ➔ Heart failure describes the state that develops when the heart cannot maintain an adequate cardiac output or can do so only at the expense of elevated filling pressures. In mild to moderate forms of heart failure, cardiac output is normal at rest and only becomes impaired when the metabolic demand increases during exercise or some other form of stress.
- ➔ In practice, heart failure may be diagnosed when a patient with significant heart disease develops the signs or symptoms of a low cardiac output, pulmonary congestion or systemic venous congestion (signs and symptoms of interstitial volume overload and/or manifestations of inadequate tissue perfusion).
- ➔ Almost all forms of heart disease can lead to heart failure. An accurate aetiological diagnosis is important because treatment of the underlying cause may reverse heart failure or prevent its progression. Approximately 50% of patients are dead within 5 years.

Etiology:

Main causes	★ **First: Ischemic heart disease, most common cause** (35–40%) ★ **Second: Cardiomyopathy (dilated)** Such as in Peripartum Cardiomyopathy (30–34%) ★ **Third: Hypertension** (15–20%) ★ **Valvular heart disease (mitral, aortic, tricuspid).**
Other causes	• Cardiomyopathies (other than dilated): hypertrophic, restrictive (amyloidosis, sarcoidosis) • Congenital heart disease (Atrial septal defect, ventricular septal defect).

[1] HF is a syndrome as a result of many diseases of the heart. Complex clinical syndrome: because there are many mechanisms that will result in HF signs and Sx.

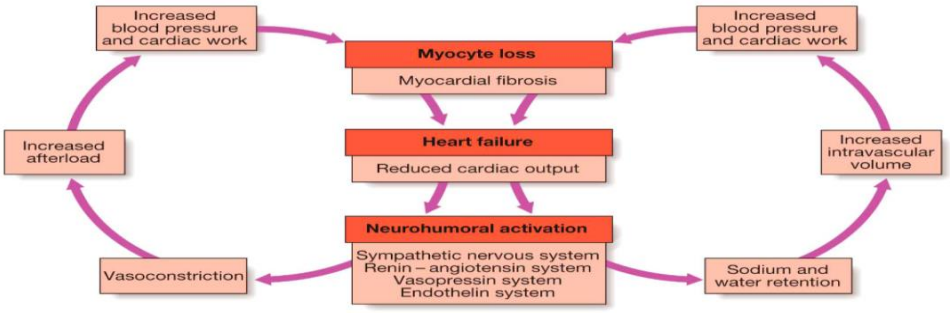
	• Alcohol and drugs (chemotherapy – trastuzumab, imatinib, Doxorubicin[2]). • Hyperdynamic circulation (anaemia*, thyrotoxicosis*, pregnancy* and Paget's disease[3]) "High output status^" • Haemochromatosis[4], radiation. • Right heart failure (right ventricular infarct, pulmonary hypertension, pulmonary embolism, COPD, Pneumonia, Interstitial lung disease). • Tricuspid incompetence. • Obesity* Any factor that increases myocardial work (*) may aggravate existing HF or initiate failure. • Arrhythmias* (atrial fibrillation, bradycardia (complete heart block, sick sinus syndrome)) • Pericardial disease (constrictive pericarditis, pericardial effusion) if the pericardium is calcified there will be impaired filling which in turn lead to HF. • Infections (e.g. myocarditis due to Chagas' disease), (Coxsackieviruses). • Sleep apnea.

In general, we can conclude that HF could be caused by:

1) **Inappropriate workload:**
   - *Volume overload:* Regurgitate valve (Aortic, mitral), High output status (mentioned above^).
   - *Pressure overload:* Systemic HTN, Outflow obstruction (Aortic stenosis).
2) **Loss of muscles:** Post-MI (decreased blood supply), chronic ischemia, connective tissue diseases, infections, poisons (alcohol,cobalt and drugs).
3) **Restricted filling:** Pericardial diseases, Restrictive cardiomyopathy, tachyarrhythmias.

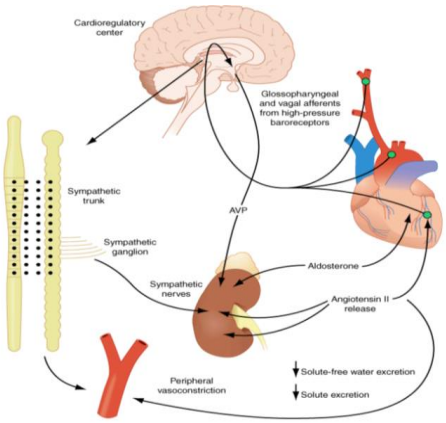
Pathophysiology of heart failure

In patients without a valvular disease, the primary abnormality is *impairment of ventricular myocardial function*, leading to a fall in cardiac output. This can occur because of ***impaired systolic contraction, impaired diastolic relaxation, or both***. This activates counter-regulatory neurohumoral mechanisms that, in normal physiological circumstances, would support cardiac function but, in the setting of impaired ventricular function, can lead to a deleterious increase in both afterload and preload . A vicious circle may be established because any additional fall in cardiac output will cause further neurohumoral activation and increasing peripheral vascular resistance.



[2] Echo is required before and during the treatment.
[3] Paget disease is a cause of HF.
[4] Storage disorder causes restrictive cardiomyopathies.

- ***Stimulation of the renin–angiotensin–aldosterone system*** *(mediated by* ***angiotensin II****, a potent constrictor of arterioles, in both the kidney and the systemic circulation)* ***leads to:***
    1) **Vasoconstriction**
    2) **Sodium and water retention.**
    3) **Sympathetic nervous system activation.**



After MI, cardiac contractility is impaired and neurohumoral activation causes hypertrophy of non-infarcted segments, with thinning, dilatation and expansion of the infarcted segment (remodelling). This leads to further deterioration in ventricular function and worsening heart failure.

Activation of SNS	**Initially:**	Activation of the sympathetic nervous system may *initially sustain cardiac output* through **increased myocardial contractility** (inotropy) **and heart rate** (chronotropy).
	Prolonged sympathetic stimulation:	***Negative effects :*** - Cardiac myocyte apoptosis - Hypertrophy and focal myocardial necrosis. - **Peripheral vasoconstriction** - arrhythmias
Sodium and water retention[5]	**Promoted by:**	➔ **Aldosterone** ➔ **Endothelin-1** (a potent vasoconstrictor peptide produced by the vascular endothelium with marked effects on the renal vasculature). ➔ Antidiuretic hormone **(ADH) "**in severe HF**".** ADH = AVP = Vasopressin
	Results in:	Pulmonary and peripheral oedema occurs because of high left and right atrial pressures.

Neurohormonal changes:

Neurohormonal changes	Favorable effect	Unfavorable effect
↑ **Increased sympathetic activity**	● ↑ HR and contractility ● Vasoconstriction→ ↑ Venous return, ↑ filling	↑ Arteriolar constriction → After load → ↑ workload → ↑ O_2 consumption
↑ **Renin-Angiotensin- Aldosterone**	Salt & water retention → ↑ Venous return (preload).	● Angiotensin-II will lead to Arteriolar constriction ↑ Afterload. ● Increased salt & water retention → peripheral and pulmonary edema.
↑ **Vasopressin**	Same effect	Same effect
↑ **Interleukins & TNFα**	May have roles in myocyte hypertrophy	Apoptosis

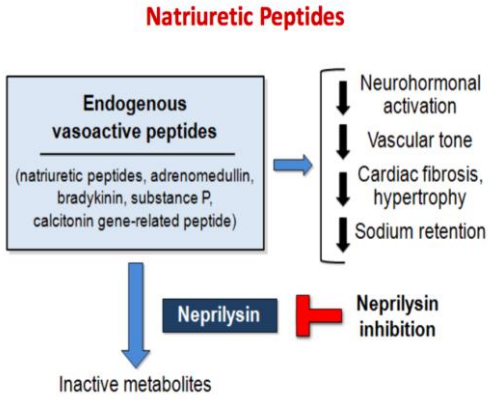
[5] caused by impairment of renal perfusion and by secondary hyperaldosteronism.

↑ Endothelin	Vasoconstriction→ ↑ Venous return	↑ Afterload

★ **Natriuretic peptides** are released from the atria in response to atrial stretch, and act as physiological ***ant-agonists to the fluid-conserving effect of aldosterone***. There are four different groups NPs identified till date [atrial natriuretic peptide (ANP), B-type natriuretic peptide (BNP), C-type natriuretic peptide (CNP) and dendroaspis natriuretic peptide, a D-type natriuretic peptide (DNP)].

➔ ***Atrial natriuretic peptide (ANP)*** *is released from atrial myocytes in response to stretch. ANP induces diuresis, natriuresis, vasodilatation and suppression of the renin–angiotensin system. Levels of circulating ANP are increased in congestive cardiac failure and correlate with functional class, prognosis and haemodynamic state.*

➔ Administration of synthetic natriuretic peptides has not improved outcomes in acute HF but modulation of the natriuretic system through inhibition of the enzyme that degrades natriuretic (and other vasoactive) peptides, ***neprilysin***, has proven to be successful.



Cellular changes: Last thing to happen, irreversible.

• Changes in Ca^{+2} handling *(In heart failure, there is a prolongation of the calcium current in association with prolongation of contraction and relaxation).*

• Changes in adrenergic receptors:

- Slight ↑ in α1 receptors
- β1 receptors desensitization → followed by down regulation

• Changes in contractile proteins.

• Program cell death (Apoptosis).

• Increase amount of fibrous tissue.

Classifications of heart failure

There are different classifications of heart failure:

★ Diastolic/Systolic failure: (MOST IMP. CLASS.)

Systolic Dysfunction (HFr[6]EF)	Diastolic Dysfunction (HFp[7]EF)
Owing to impaired contractility • The abnormality is decreased EF • Causes include: 1. **Ischemic heart disease** or after a recent MI—infarcted cardiac muscle does not pump blood (decreased EF) 2. HTN resulting in cardiomyopathy 3. Valvular heart disease 4. Myocarditis (postviral) 5. Less common causes: Alcohol abuse, radiation, hemochromatosis, thyroid disease	Owing to impaired ventricular filling during diastole, because of either: 1. Impaired relaxation 2. Increased stiffness of ventricle or both - EF is preserved - Diastolic dysfunction is less common than systolic dysfunction. - HTN leading to myocardial hypertrophy is the most common cause of diastolic dysfunction. whom have HFrEF will end up with HFpEF but not usually the other way around.
• Usually both systolic and diastolic dysfunctions present simultaneously • Around 20-33% of HF cases have normal EF	

★ High/low output heart failure:

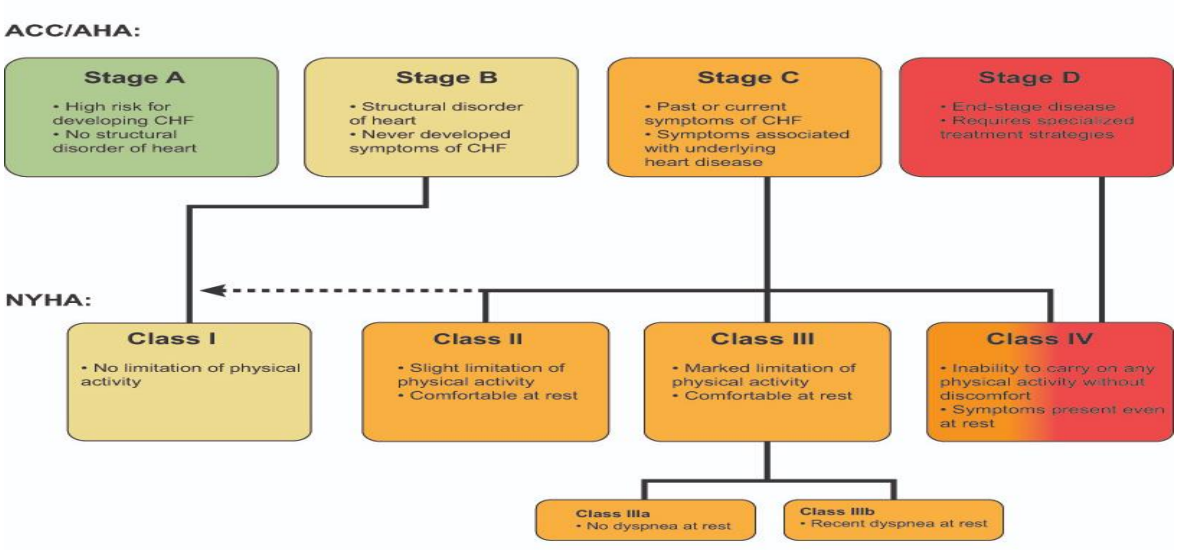
High output heart failure[8]	Low output heart failure[9]
Certain medical conditions **increase demands on cardiac output**, causing a clinical picture of heart failure due to an excessively high cardiac output. (**ex; severe anemia, thyrotoxicosis or pregnancy**)	Cardiac output is inadequate to perfuse the body (ie ejection fraction <40%), or can only be adequate with high filling pressures.

★ New york heart association classification (Important):

Class I	No limitation during ordinary activity. Normal physical exercise doesn't cause symptoms (fatigue,dyspnea or palpitations).
Class II	Slight/Mild limitation during ordinary activity. Comfortable at rest but normal physical exercise causes symptoms
Class III	Marked limitation. Comfortable at rest but gentle physical activity produces symptoms
Class IV	Symptoms of heart failure occur at rest, and exacerbated by any physical activity.

[6] reduced
[7] preserved

[8] الجسم يطلب Output عالي من القلب عشان يلبي احتياجه
[9] المشكلة مو في احتياج الجسم العالي، المشكلة إن القلب مو قادر يشتغل بشكل طبيعي

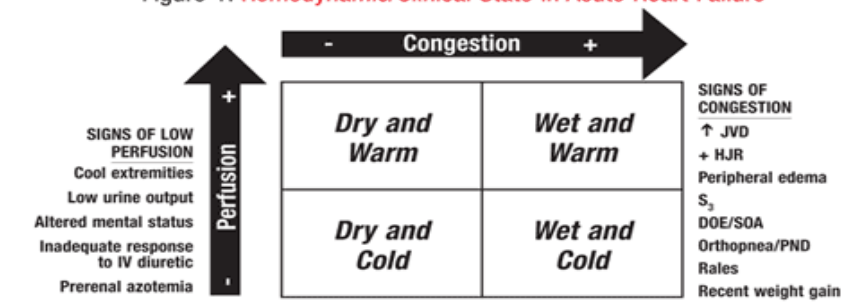


★ Acute/Chronic Heart Failure:

Acute heart failure (Acute Pulmonary edema)		Chronic heart failure (More Common)
This usually presents with sudden-onset dyspnoea at rest with acute respiratory distress, orthopnoea . A precipitant (e.g. acute MI, valvular disease, myocarditis, and cardiogenic shock) may be apparent from the history. The peripheries are cool to the touch, the pulse is rapid and there is JVD. The apex is not displaced, as there has been no time for ventricular dilatation. Auscultation may reveal S3 'gallop' and crepitations are heard at the lung bases.		This commonly follows a relapsing and remitting course, with periods of stability interrupted by episodes of decompensation. A low cardiac output causes fatigue, listlessness and a poor effort tolerance; the peripheries are cold and BP is low Clinical picture depends on: 1. The underlying heart disease 2. Type of heart failure 3. Neurohormonal changes that developed. Sometimes associated with: - Weight loss (cardiac cachexia) - Poor tissue perfusion - Skeletal muscle atrophy
Management of Acute decompensated Heart Failure	A. **Oxygen** B. **Loop diuretics** (furosemide): Most important drug that decreases the preload[10] C. **Nitrate (IV)** : that decrease the afterload D. **Morphine** can be used Note: If pulmonary edema continuous despite these 4 > dobutamine is added (increased contractility & decrease afterload) *ACE inhibitors and β-Blockers are not used in acute settings.	

★ Forrester Classification:

Figure 1. Hemodynamic/Clinical State in Acute Heart Failure



↑: increased; +: positive; -: negative; DOE: dyspnea on exertion; HJR: hepatojugular reflux; JVD: jugular venous distention; PND: paroxysmal nocturnal dyspnea; S_3: ventricular filling murmur; SOA: shortness of air.
Source: References 10, 11.

Dry and cold worse prognosis.

- ★ Dry: no congestion.
- ★ Wet: congestion.
- ★ Warm: no decrease in perfusion.
- ★ Cold: decrease in perfusion.

[10] Best initial therapy

★ Left, right and biventricular heart failure:

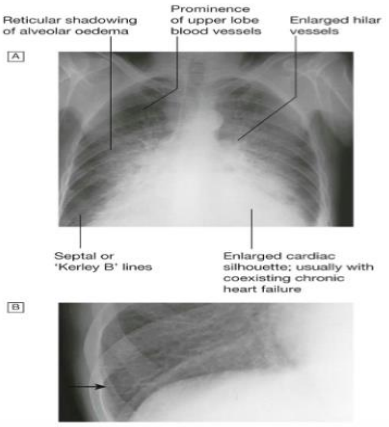
The left side of the heart comprises the functional unit of the LA and LV, together with the mitral and aortic valves; the right heart comprises the RA, RV, and tricuspid and pulmonary valves

	Left-sided Heart Failure (reduction in LV output)	Right-sided Heart Failure (reduction in RV output)[11]
	***Left-sided heart failure*:** There is a reduction in left ventricular output and an increase in left atrial and pulmonary venous pressure. An acute increase in left atrial pressure causes *pulmonary congestion or pulmonary oedema*; a more gradual increase in left atrial pressure, as occurs with mitral stenosis, leads to reflex pulmonary vasoconstriction, which protects the patient from pulmonary oedema. This increases pulmonary vascular resistance and causes pulmonary hypertension, which can, in turn, impair right ventricular function.	***Right-sided heart failure*:** There is a reduction in right ventricular output and an increase in right atrial and systemic venous pressure. Causes of isolated right heart failure include chronic lung disease (cor pulmonale), pulmonary embolism and pulmonary valvular stenosis.
Symptoms:	❖ **Dyspnea**: Difficulty breathing secondary to pulmonary congestion/edema ❖ **Orthopnea**: Difficulty breathing in the recumbent position; relieved by elevation of the head with pillows ❖ **Paroxysmal nocturnal dyspnea (PND)**: awakening after 1 to 2 hours of sleep due to acute shortness of breath (SOB) ❖ **Nocturnal cough (nonproductive)**: worse in recumbent position (same pathophysiology as orthopnea) ❖ **Confusion and memory impairment:** occur in advanced CHF as a result of inadequate brain perfusion ❖ **Diaphoresis and cool extremities at rest**: Occur in desperately ill patients (NYHA class IV)	❖ **Peripheral pitting edema**: Pedal edema lacks specificity as an isolated finding. In the elderly, it is more likely to be secondary to venous insufficiency ❖ **Nocturia**: Due to increased venous return with elevation of legs
Signs	❖ **Displaced PMI** (usually to the left) due to cardiomegaly ❖ **Pathologic S3 (ventricular gallop)** *"low pitched sound that is heard during rapid filling of ventricle"* ❖ S4 gallop ❖ Crackles/rales at lung bases [12]	❖ **Jugular venous distention (JVD)** ❖ Painful Hepatomegaly/hepatojugular reflux ❖ Ascites ❖ Right ventricular heave
Biventricular failure (Both sides)		
Example: dilated cardiomyopathy or ischaemic heart disease, affects both ventricles or because disease of the left heart leads → chronic elevation of the left atrial pressure→ pulmonary hypertension → right heart failure		

[11] Most common cause of Right-sided HF is left-sided HF.

[12] Because of Pulmonary Edema

Diagnosis of CHF

Test:	Findings:
Transthoracic Echocardiogram	★ **Initial test of choice:** should be performed whenever CHF is suspected based on history, examination, or CXR. - Useful in determining whether systolic or diastolic dysfunction predominates, and determines whether the cause of CHF is due to a pericardial, myocardial, or valvular process. - **Estimates EF (very important)**: Patients with systolic dysfunction (EF <40%) should be distinguished from patients with preserved left ventricular function (EF >40%). - Shows chamber dilation and/or hypertrophy. - Identify patients who will benefit from long-term drug therapy, e.g. ACE inhibitors.
Chest X ray (CXR)	High pulmonary venous pressure in left-sided heart failure first shows on the chest X-ray as an abnormal distension of the upper lobe pulmonary veins (with the patient in the erect position). The vascularity of the lung fields becomes more prominent, and the right and left pulmonary arteries dilate. Subsequently, interstitial oedema causes thickened interlobular septa and dilated lymphatics. These are evident as horizontal lines in the costophrenic angles (septal or 'Kerley B' lines). More advanced changes due to alveolar oedema cause a hazy opacification spreading from the hilar regions, and pleural effusions.  - **Cardiomegaly** - ***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 - **Pleural effusion**
ECG	- **Nonspecific**, but can be useful for detecting chamber enlargement and presence of ischemic heart disease or prior MI.
Catheter	- Precise valve diameter, septal defects (when CAD or valvular suspected or if heart transplant is indicated)
Blood tests	- CBC for→ anemia - Liver biochemistry(may be altered do to hepatic congestion) - Brain natriuretic peptide (BNP) or (Pro BNP), if normal(<100pg/mL) exclude heart failure (particularly pulmonary edema). - T4 & TSH - Electrolytes imbalance → Chronic renal insufficiency

Management of CHF

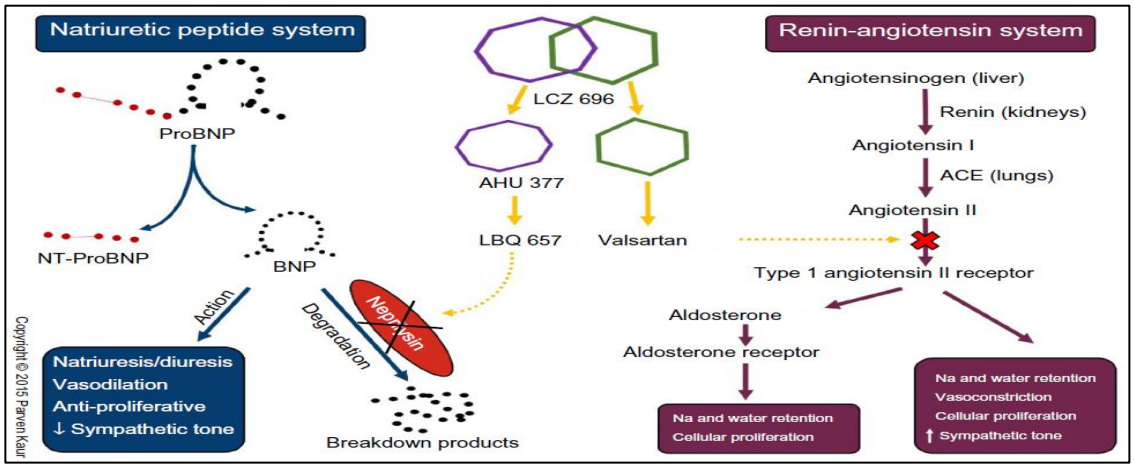
Management of any disease we think in three categories: 1- life modification. 2- Medical management. 3- Surgical intervention.

✦ Acute presentation treats only by 1- Diuretics. 2- Ventilator support if needed.

Systolic Failure Management	
General lifestyle modification:	❖ Sodium restriction (2 gram Na = 5 gram NaCl) ❖ Fluid restriction (1.5 Liter = 8 cups) ❖ Weight loss ❖ Daily weight (tailor therapy). (diuretics) نقول للمريض راقب وزنك اذا شفته زاد زود الجرعة ❖ Smoking cessation ❖ Restrict alcohol use ❖ Exercise program (to increase heart contractility function) ❖ Annual influenza vaccine and pneumococcal vaccine recommended because influenza virus has mortality in chronic diseases one of them chronic heart failure.
Diuretics	❖ Most effective means of providing symptomatic relief to patients with moderate to severe CHF ❖ Recommended for patients with systolic failure and volume overload ❖ Have not been shown to reduce mortality or improve prognosis, ***just for symptom control.*** Goal is relief of signs and symptoms of volume overload (dyspnea, peripheral edema) ❖ Loop diuretics: **Furosemide (Lasix)**—most potent (Most common use. Sometimes we use (furosemide + metolazone (thiazide[13]) 'work in distal convoluted tubule') ❖ Thiazide diuretics: **Hydrochlorothiazide**—modest potency ❖ Side effect of these drugs: Pre-renal azotemia, Skin rashes, Neutropenia, Thrombocytopenia, Hyperglycemia, ↑Uric Acid, Hepatic dysfunction ✦ High stage of heart failure needed high dose comparison to early stage of heart failure.
β-Blockers[14]	❖ The combination of B blockers and an ACE inhibitors required for patient with LVEF less than 40% either **symptomatic or asymptomatic** ❖ Proven to decrease mortality in patients with post-MI heart failure. ❖ β-Blockers also have antiarrhythmic and anti-ischemic effect. ❖ Reported to improve symptoms of CHF; may slow progression of heart failure by slowing down tissue remodeling. The decrease in heart rate(antiarrhythmic) ar leads to decreased oxygen consumption (anti ischemic effect) ❖ Should be given to stable patients with mild to moderate CHF (class I, II, and III) unless there is a noncardiac contraindication. ❖ Not all β-blockers are equal. There is evidence only for metoprolol, bisoprolol, and carvedilol.
Spironolactone (aldosterone antagonist)	❖ It reduces mortality ❖ Monitor serum potassium and renal function ❖ **Prolong survival** in CHF patients with subsequent effect on myocardial remodeling and fibrosis. ❖ Monitor serum potassium and renal function ❖ Spironolactone is proven effective only for more advanced stages of CHF (classes III and IV) ❖ Eplerenone is an alternative to spironolactone (does not cause gynecomastia). If the patient developed gynecomastia, impotence (cause its structurally similar to progesterone) switch to eplerenone.

[13] Not effective with GFR < 30/min

[14] When a patient comes to ER with very high HR don't give him beta-blocker! Because you're blocking the mechanism that increase his cardiac output! Think, why he is having tachycardia? because of activation of sympathetic NS, now you want him to relax so give him oxygen or diuretics > HF will improve > HR back to normal. (YOU DON'T TREAT NUMBERS! YOU DEAL WITH PATHOPHYSIOLOGY).

ACE Inhibitors (Benazepril, Captopril, Enalapril, Fosinopril. Lisinopril)	Cause venous and arterial dilation, decreasing preload and afterload. ❖ **The combination of a diuretic and an ACE inhibitor should be the initial treatment in most symptomatic patients.** ❖ ACE inhibitors reduce mortality, prolong survival, and alleviate symptoms in mild, moderate, and severe CHF. ❖ Indicated for left ventricular systolic dysfunction (LV ejection fraction less than 40%). ❖ All patients with systolic dysfunction should be on an ACE inhibitor **even if they are asymptomatic.** ❖ Side effects of ACE inhibitors: Angioedema (rare occurring but when it happen it is scary we should stop this medication and start alternative one which is ARBs and give patient steroid), Hypotension, Renal insufficiency (afferent arteriole constriction), Rash , Cough (increase in Bradykinin) ❖ If patient developed hyperkalemia or renal impairment or is pregnant→ **switch from ACEI to Hydralazine** (arterial dilator)(decrease afterload) with isosorbide dinitrate (Venodilator)(decrease preload). Always start at a low dose to prevent hypotension, Monitor BP, potassium, BUN, and creatinine.
Angiotensin II receptor blockers (ARBs)	Used in patients unable to take ACE inhibitors due to side effects (eg, angioneurotic edema, cough) **but do not replace ACE inhibitors if patient tolerates an ACE inhibitor.**
Angiotensin Receptor-Neprilysin inhibitor (ARNi)	❖ Recent FDA approval (2015). ❖ The only product available (valsartan/sacubitril). ❖ Valsartan = ARB. ❖ Sacubitril = prodrug for sacubitrilat. ❖ Inhibit neprilysin which breakdown the vasoactive peptides. ❖ Used if patient LVEF <= 35% and still symptomatic with ACE/ARB. In this specific group of patients it improves mortality and morbidity. 
If - Channel blocker[15]:	❖ Ivabradine; Inhibit the Na inflow during the SA node action potential phase 4. ❖ Decrease the heart rate. ❖ Only use it if HR not controlled by B-blocker and remains > 70 bpm and the patient has sinus rhythm. In this group if patients it improve Morbidity and Mortality.

[15] Funnel channel Na in SA node.

Digitalis	❖ Positive inotropic[16] agent. Has vagotonic[17] & arrhythmatic effects. ❖ Useful in patients with EF <40%, who continue to have symptoms despite optimal therapy (with ACE inhibitor, β-blocker, aldosterone antagonist, and a diuretic), severe CHF, or severe AFib. ❖ Provides short-term symptomatic relief (used to control dyspnea and will decrease frequency of hospitalizations) but has not been shown to improve mortality. ❖ **Serum levels should be monitored (digoxin toxicity: yellow vision, nausea, vomiting)** ❖ Neither works on RAAS nor improves patient survival.

Diastolic Failure Management

Notes	Few therapeutic options available; patients are treated symptomatically (NO medications have proven mortality benefit) 1.β-Blockers have clear benefit and should be used 2.Diuretics are used for symptom control (volume overload) 3.ACE inhibitors and ARBs (Digoxin and spironolactone should NOT be used).

✓ **The standard treatment of systolic dysfunction is:**	Diuretics + ACE inhibitor + β blockers.
→ **The initial treatment for symptomatic patient is:**	Diuretics + Vasodilatation (ACEI, ARB or Hydralazine with isosorbide[18]).

★ The following medications are contraindicated in patients with CHF:

1. Metformin—may cause potentially fatal lactic acidosis.
2. Thiazolidinediones—causes fluid retention.
3. NSAIDs may increase risk of CHF exacerbation.
4. Some antiarrhythmic agents that have negative inotropic effect.

★ Medications that have been shown to lower mortality in systolic heart failure: (imp.)

1. ACE inhibitors and ARBs
2. β-Blockers
3. Aldosterone antagonists (spironolactone)
4. Hydralazine and nitrate (Nitroglycerin)

★ The following devices have been shown to reduce mortality in selected patients:

1. **An ICD**[19] lowers mortality by helping prevent sudden cardiac death (which is the most common cause of death in CHF). It is indicated for patients at least 40 days post-MI, EF <35%, and class II or III symptoms despite optimal medical treatment.

2. **Cardiac resynchronization therapy (CRT):** This is biventricular pacemaker indications are similar to ICD except these patients also have prolonged QRS duration >120 msec. Most patients who meet criteria for CRT are also candidates for ICD and receive a combined device.

3. **Revascularization.**

   ★ **Cardiac transplantation is the last alternative if the above do not control symptoms.**
   Advance stage of heart failure (stage D) management Cardiac Transplant (best)

[16] Affect the strength of contraction of heart muscle (myocardial contractility).
[17] Overactivity or irritability of the vagus nerve, adversely affecting function of the blood vessels, stomach, and muscles.
[18] is in the class of drugs called nitrates that are used for treating and preventing angina.
[19] An implantable cardioverter-defibrillator (ICD) or automated implantable cardioverter defibrillator (AICD) is a device implantable inside the body, able to perform cardioversion, defibrillation, and (in modern versions) pacing of the heart

Summary

★ Heart Failure:

Any cardiac disorder that causes the heart to fail in ejection of blood or failure to adequately fill with blood (low cardiac output).

★ Main Pathophysiologies:

1- Sympathetic nervous system: makes cardiac output better at first, but then later causes vasoconstriction.

2- RAS, Aldosterone (& vasopressin): cause sodium and water retention which eventually cause peripheral and pulmonary edema.

3- Atrial Natriuretic Peptides: released from the atria to antagonize aldosterone, therefor decrease sodium and water retention.

4- Cellular changes: Changes in calcium, adrenergic receptors, and contractile proteins.

★ Classifications:

1- Systolic or Diastolic:

a. Systolic dysfunction: impaired contractility, ejection fraction is decreased. (more common)

b. Diastolic dysfunction: impaired ventricular filling, ejection fraction is preserved.

2- High or Low Output:

a. High Output: increase in demands cause excessively high cardiac output

b. Low Output: inadequate tissue perfusion, unless there is high filling pressure.

3- Class I to IV of New York Heart Association:

Depends on whether the symptoms occur at rest, or at different intensities of physical activities.

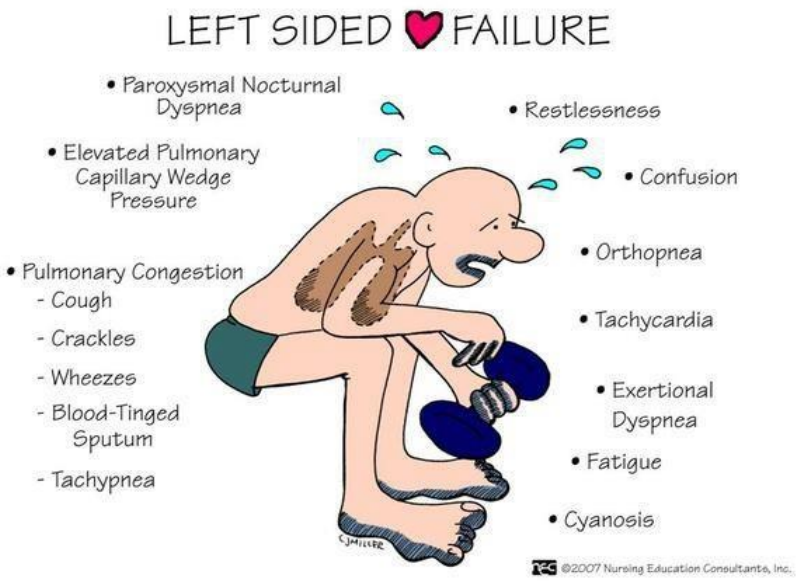
4- Acute or Chronic:

a. Acute pulmonary edema: Usually a sudden presentation of SOB and orthopnea, with Jugular Venous Distention, an S3 Gallop, but without apex beat displacement.

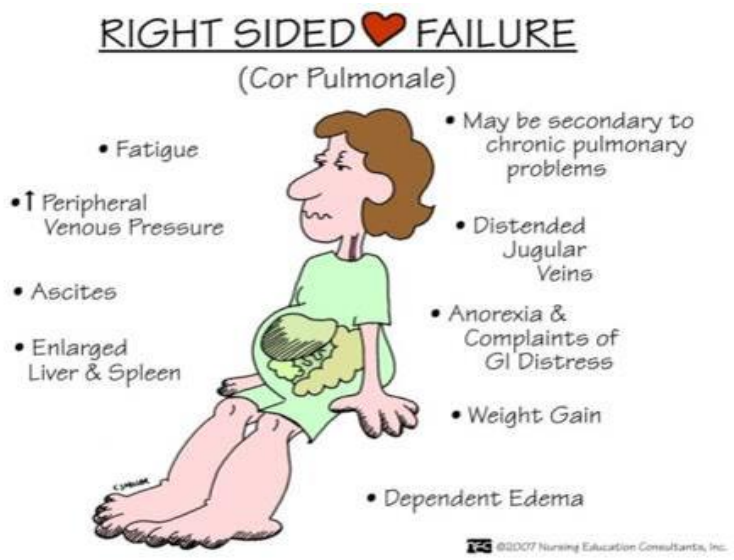
b. Chronic heart failure: Usually has a relapsing and remitting course, the signs and symptoms vary depending on the underlying pathologies.

5- Left sided or Right Sided or Biventricular:

a. Left sided heart failure: Reduction in Left Ventricle output, either with a sudden increase in Left Atrial (pulmonary) venous pressure which causes pulmonary edema, or gradual increase in Left Atrial pressure which protects from it. But still, this gradual increase causes pulmonary vasoconstriction which can eventually lead to Right Ventricular failure. It presents with the same signs of acute pulmonary edema, but the PMI here is displaced without JVD.



b. Right sided heart failure: Reduction in Right Ventricle output and increase in Right atrial (systemic) venous pressure. It presents with JVD, painful hepatomegaly, and ascites.



c. Biventricular heart failure: either due to a disease that affects both ventricles, or a disease of the left heart which eventually affects the right.

★ Diagnosis:

1- Transthoracic Echocardiogram: Determines whether systolic or diastolic (& determining EF).

2- Chest X Ray: Shows the important signs of pulmonary edema such as Kerley B Lines.

3- ECG 4- Catheter 5- Blood tests: the levels of BNP can exclude the diagnosis of heart failure.

★ Management (main differences between systolic and diastolic):

- → Systolic: Drugs that decrease mortality (B-Blockers, ACE Inhibitors, ARBs, Spironolactone) and to improve symptoms (Diuretics, Digitalis).
- → Diastolic: the drugs do not decrease mortality, they just treat the symptoms. (B-Blockers, ACE Inhibitors, ARBs, Diuretics).

Questions:

1- The term 'orthopnoea' refers to breathlessness (dyspnoea) in a particular situation. Which answer below describes that situation?

A. After several hours of sleep
B. Due to asthma
C. Immediately on lying flat
D. On exertion
E. On sitting upright

2- Which of the following physical signs is associated with left ventricular failure?

A. A gallop rhythm with a fourth heart sound
B. A gallop rhythm with a third heart sound
C. A loud second heart sound
D. A quiet first heart sound
E. Fixed splitting of the second heart sound

3- What relationship does Starling's Law of the heart describe?

A. Between blood pressure and cardiac output
B. Between cardiac filling and blood pressure
C. Between cardiac filling and cardiac output
D. Between heart rate and blood pressure
E. Between heart rate and cardiac output

4- What underlying pathophysiological changes is chronic cardiac failure associated with?

A. Activation of the renin–angiotensin– aldosterone system (RAAS)
B. Inhibition of the RAAS
C. Inhibition of the sympathetic nervous system
D. Reduced production of brain natriuretic peptide (BNP)
E. Systemic vasodilatation

5- Neuroendocrine system activation is a feature of heart failure. Abnormalities of which hormone can cause heart failure rather than result from heart failure?

A. Aldosterone
B. Angiotensin II
C. Catecholamines
D. Thyroxine
E. Vasopressin (antidiuretic hormone, ADH)

6- A 78-year-old woman is admitted with heart failure. The underlying cause is determined to be aortic stenosis. Which sign is most likely to be present?

A. Pleural effusion on chest x-ray
B. Raised jugular venous pressure (JVP)
C. Bilateral pedal oedema
D. Bibasal crepitations
E. Atrial fibrillation

7- A 78-year-old woman is admitted to your ward following a 3-day history of shortness of breath and a productive cough of white frothy sputum. On auscultation of the lungs, you hear bilateral basal coarse inspiratory crackles. You suspect that the patient is in congestive cardiac failure. You request a chest x-ray. Which of the following signs is not typically seen on chest x-ray in patients with congestive cardiac failure?

A. Lower lobe diversion
B. Cardiomegaly
C. Pleural effusions
D. Alveolar oedema
E. Kerley B lines

8- A 70 years old female presented to the ER with SOBOE, LL swelling for 2 weeks. BP 180/100. JVP high. LL oedema. Chest crackles. ECO was done, EF = 55%. What is the patient expected to have?

A. HfpEF
B. HfrEF
C. LSHF
D. RSHF

9- A 42 English man presented to the ER with shortness of breath, fatigue, lower limb swilling. A climical dignosis of HF. on examination the patient was found to have hip pain, deafness, LL numbness, varus. What is the expected underlying cause?

A. ASD
B. Pericarditis
C. Paget's disease
D. Anemia.

10- A patient presented with dyspnea, fatigue, palpitations at rest. He is NYHA class..

A. I
B. II
C. IV
D. III

11- 50 year old man is note to have severe congestive heart failure what drug of the following can prolong survival:

A. Furosemide
B. Hydrochlorothyzide
C. Spironolactone.
D. Digitalis.

12- What is the initial treatment for asymptomatic patients with systolic dysfunction?

A. β blockers and Diuretics
B. Diuretics + vasodilatation (ACEI or ARBs) + β blockers
C. Diuretics + vasodilatation (ACEI or ARBs)
D. β blockers and vasodilatation (ACEI or ARBs)

13- What is the standard treatment for patients with diastolic dysfunction?

A. β blockers and Diuretics
B. Diuretics + vasodilatation (ACEI or ARBs) + β blockers
C. Diuretics + vasodilation (ACEI or ARBs)
D. β blockers and vasodilatation (ACEI or ARBs)

14- 45 years old known CHF, he's on (Diuretics, ACE inhibitors, beta blockers) Recently he develops a dry cough. Which one of the following drugs caused this side effect?

A. Carvedilol (beta-blockers)
B. Enalapril (ACE inhibitors)
C. Losartan (AIIR blockers)
D. Furosemide (loop diuretic)

15- Which one of the following drugs reduce the morbidity rate (hospitalization, HF symptoms) but does not affect the mortality rate?

A. Ivabradine
B. Spironolactone
C. Digoxin
D. Captopril

16- 55-year-old patient presents to you after a 3-day hospital stay for gradually increasing shortness of breath and leg swelling while away on a business trip. He was told that he had congestive heart failure, but is asymptomatic now, with normal vital signs and physical examination. An echocardiogram shows

an estimated ejection fraction of 38%. The patient likes to keep medications to a minimum. He is currently on aspirin and simvastatin. Which would be the most appropriate additional treatment?

A. Begin an ACE inhibitor and then add a beta-blocker on a scheduled basis.
B. Begin digoxin plus furosemide on a scheduled basis.
C. Begin spironolactone on a scheduled basis.
D. Begin furosemide plus nitroglycerin.

17- 56-year-old man, diagnosed with dilated cardiomyopathy with ejection fraction less than 25%, NYHA class II dyspnea, BP: 112/68, HR:82, JVP: 7cm water [normal], soft S3 and grade 2 pansystolic murmur, chest is clear, no lower limb edema, warm extremities.

1. According to perfusion and congestion, how do we classify this patient?

A. Warm and dry
B. Cold and dry
C. Warm and wet
D. Cold and wet

2. How do we treat this patient?

A. Diuretics
B. ACEI and beta-blockers
C. Inotropes
D. No treatment

18-You are caring for a 72-year-old man admitted to the hospital with an exacerbation of congestive heart failure. Two weeks prior to admission, he was able to ambulate two blocks before stopping because of dyspnea. He has now returned to baseline and is ready for discharge. His preadmission medications include aspirin, metoprolol, and furosemide. Systolic blood pressure has ranged from 110 to 128 mm Hg over the course of his hospitalization. Heart rate was in 120s at the time of presentation, but has been consistently around 70/minute over the past 24 hours. An echocardiogram performed during this hospitalization revealed global hypokinesis with an ejection fraction of 30%. Which of the following medications, when added to his preadmission regimen, would be most likely to decrease his risk of subsequent mortality?

A. Digoxin
B. Enalapril
C. Hydrochlorothiazide
D. Propranolol

1-C 2-B 3-C 4-A
5-D 6-D 7-A 8-A
9-C 10-C 11-C
12-D 13-C 14-B
15-C 16-A 17(1)-A
17(2)-B 18-B