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

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[Color index : Important | Notes | Extra]

Resources:

- 435 slides


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"Medicine is an art, nobody can deny it."

Introduction

NOTE : Based on what doctors said,cardiomyopathy lecture is for your own knowledge, if you are interested check out this file

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%. 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 die within 5 years.

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

Etiology :

Main causes	★ **First : Ischemic heart disease, most common cause** (35–40%) ★ **Second : Cardiomyopathy (dilated)** (30–34%) ★ **Third : Hypertension** (15–20%)
Other causes	• Cardiomyopathies (other than dilated) : hypertrophic, restrictive (amyloidosis, sarcoidosis) • Valvular heart disease (mitral, aortic, tricuspid) • Congenital heart disease (ASD, VSD[2]) • Alcohol and drugs (chemotherapy – trastuzumab, imatinib, Doxorubicin[3]). • Hyperdynamic circulation (anaemia, thyrotoxicosis, haemochromatosis, pregnancy and Paget's disease[4]) "High output status" • Right heart failure (right ventricular infarct, pulmonary hypertension, pulmonary embolism, COPD) • Tricuspid incompetence • 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).

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).

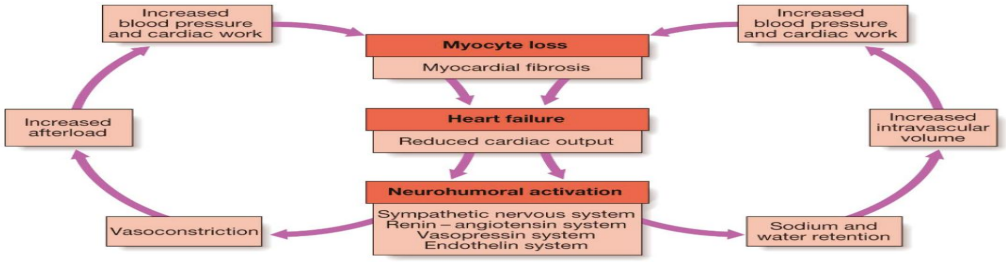
2) **Loss of muscles :** Post-MI (decreased blood supply), chronic ischemia, connective tissue diseases, infections, poisons (alcohol,cobalt and drugs).

3) **Restricted filling :** Pericardial diseases, Restrictive cardiomyopathy, tachyarrhythmias.

Pathophysiology of heart failure

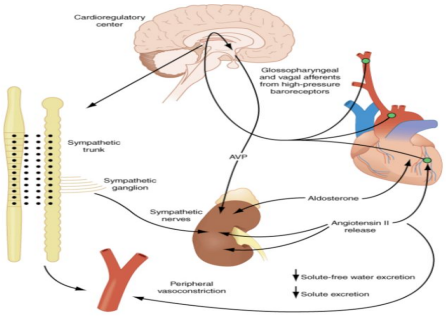
In patients without a valvular disease, the primary abnormality is *impairment of ventricular myocardial function*, leading to a fall in cardiac output. This can occur because of ***impaired systolic contraction, impaired diastolic relaxation, or both***. This activates counter-regulatory neurohumoral 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] Atrial septal defect, ventricular septal defect.
[3] Echo is required before and during the treatment.
[4] Paget disease is a cause of HF (MCQs)



- ***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 *<u>initially</u> sustain cardiac output* through **increased myocardial contractility** (inotropy) **and heart rate** (chronotropy).
	Prolonged sympathetic stimulation :	***<u>Negative effects :</u>*** - Cardiac myocyte apoptosis - Hypertrophy and focal myocardial necrosis. - **Peripheral vasoconstriction** - arrhythmias
Sodium and water retention	**Promoted by :**	➔ **Aldosterone** ➔ **Endothelin-1** (a potent vasoconstrictor peptide with marked effects on the renal vasculature). ➔ Antidiuretic hormone **(ADH)** **"**in severe heart failure**".** ADH = AVP = Vasopressin
	Results in :	Pulmonary and peripheral oedema occurs because of high left and right atrial pressures, respectively; this is compounded by sodium and water retention, caused by impairment of renal perfusion and by secondary hyperaldosteronism.

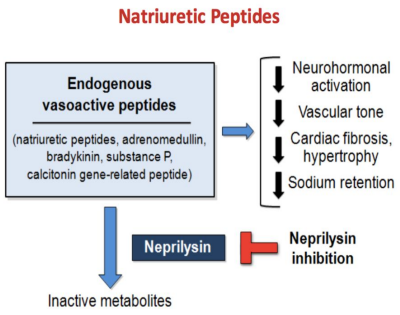
<u>Neurohormonal changes :</u>

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 &TNF α**	May have roles in myocyte hypertrophy	Apoptosis
↑ **Endothelin**	Vasoconstriction→ ↑ VR	↑ Afterload

★ **Natriuretic peptides** are released from the atria in response to atrial stretch, and act as physiological ***antagonists 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 α 1 receptors
  - β 1 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:

Systolic Dysfunction (HFrEF)	Diastolic Dysfunction (HFpEF)
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.
• Usually both systolic and diastolic dysfunctions present simultaneously	

★ High/low output heart failure:

High output heart failure[5]	Low output heart failure[6]
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.

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

★ Acute/Chronic Heart Failure:

<table>
<tr><th colspan="2">Acute heart failure (Acute Pulmonary edema)</th><th>Chronic heart failure (More Common)</th></tr>
<tr><td colspan="2">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 and crepitations are heard at the lung bases.</td><td rowspan="2">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</td></tr>
<tr><td>Management of Acute decompensated Heart Failure</td><td>A. Oxygen
B. Loop diuretics (furosemide): Most important drug that decreases the preload[7]
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.</td></tr>
</table>

★ Left, right and biventricular heart failure :

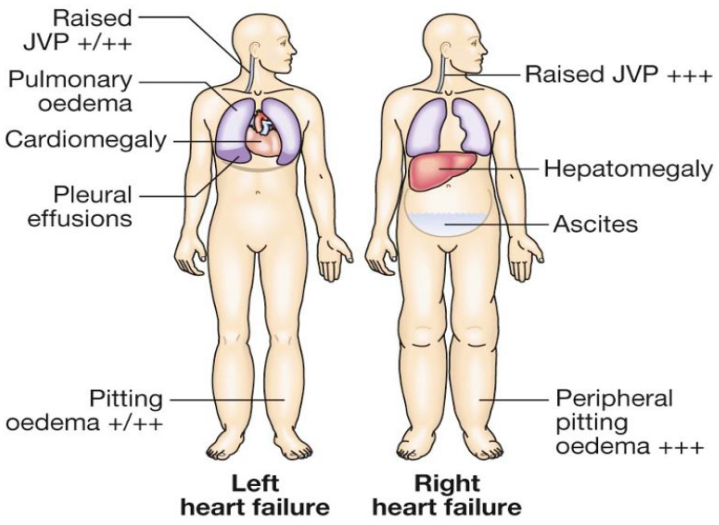
The left side of the heart comprises the functional unit of the LA and LV, together with the mitral and aortic valves; the right heart comprises the RA, RV, and tricuspid and pulmonary valves

Left-sided Heart Failure (reduction in LV output)	Right-sided Heart Failure (reduction in RV output)[8]
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.

[7] Best initial therapy

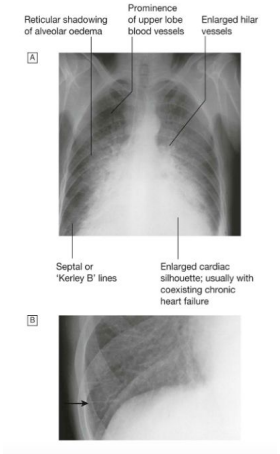
[8] Most common cause of Right-sided HF is left-sided HF.

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 [9]	❖ **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		



[9] Because of Pulmonary Edema

Diagnosis of CHF

Test:	Findings:
Echocardiogram (TTE)	★ **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%). master the boards: What is the most accurate test (in estimating the EF)? Multiple-gated acquisition scan (MUGA) or nuclear ventriculography. - 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) , if normal(<100pg/mL) exclude heart failure (particularly pulmonary edema). - T4 & TSH - Electrolytes imbalance → Chronic renal insufficiency

Management of CHF

Systolic Failure Management	
General lifestyle modification:	❖ Sodium restriction ❖ Fluid restriction ❖ Weight loss ❖ Smoking cessation ❖ Restrict alcohol use ❖ Exercise program ❖ Annual influenza vaccine and pneumococcal vaccine recommended
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 ❖ Thiazide diuretics: **Hydrochlorothiazide**—modest potency
β -Blockers[10]	❖ 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 leads to decreased oxygen consumption. ❖ 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.
Ivabradine	❖ SA nodal inhibitor of "funny Channels" that slows the heart rate. Add it to Systolic dysfunction if the pulse is over 70 bpm or beta blockers can't be used.
Spironolactone (aldosterone antagonist)	❖ It reduces mortality ❖ Monitor serum potassium and renal function ❖ **Prolong survival** in selected patients with CHF ❖ 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.
ACE Inhibitors	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.** ❖ If patient developed hyperkalemia or renal impairment or is pregnant→ **switch from ACEI**

[10] 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).

	to **Hydralazine** (arterial dilator) with isosorbide dinitrate (Venodilator). 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, cough) **but do not replace ACE inhibitors if patient tolerates an ACE inhibitor.**
Digitalis	❖ Positive inotropic agent. ❖ Useful in patients with EF <40%, 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. ❖ For patients with EF <40%, who continue to have symptoms despite optimal therapy (with ACE inhibitor, β -blocker, aldosterone antagonist, and a diuretic). ❖ **Serum levels should be monitored (digoxin toxicity: yellow vision , nausea , vomiting)**
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

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 effects

Medications that have been shown to lower mortality in systolic heart failure: (VERY IMPORTANT)

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

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

1. An ICD 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.

★ **Cardiac transplantation is the last alternative if the above do not control symptoms.**

Summary

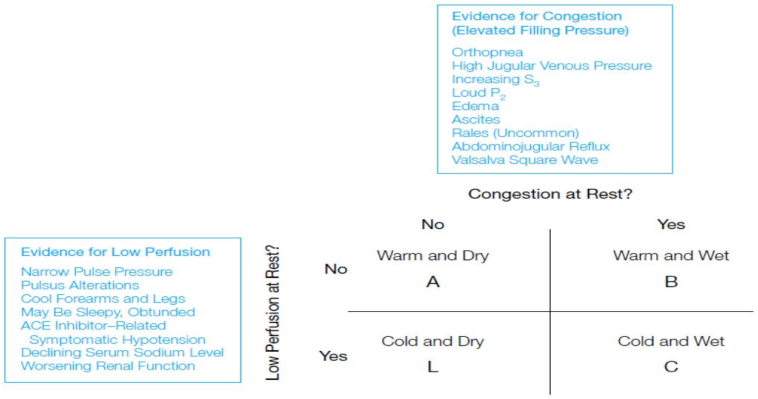
Evaluation :

1 warm and dry: compensated heart failure or normal patient “Well perfusion and not congested”.

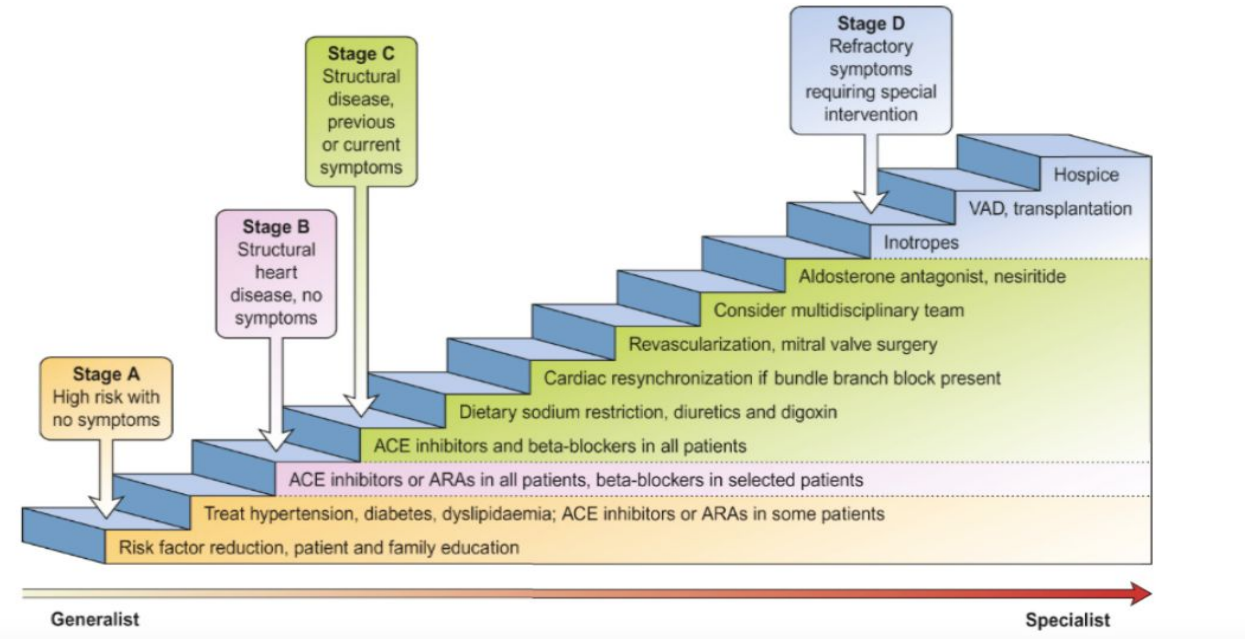
2 Warm and wet: pulmonary edema (diastolic dysfunction) “Well perfusion and congested”

3 cold and dry: hypovolemic “Not congested”

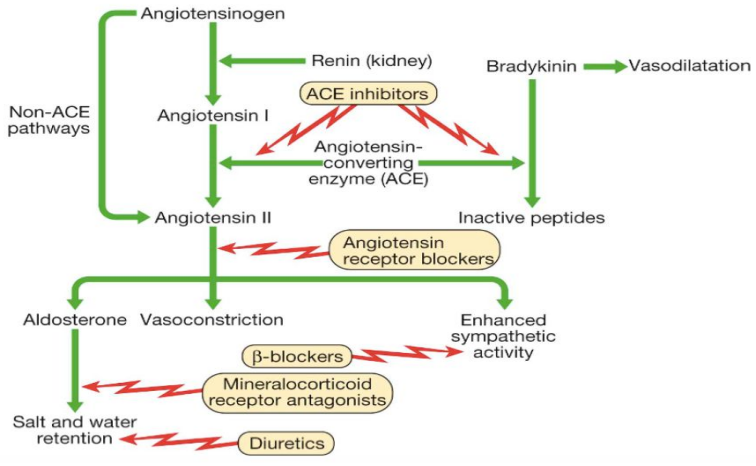
4 cold and wet: cardiogenic shock “congested and not perfusing well”

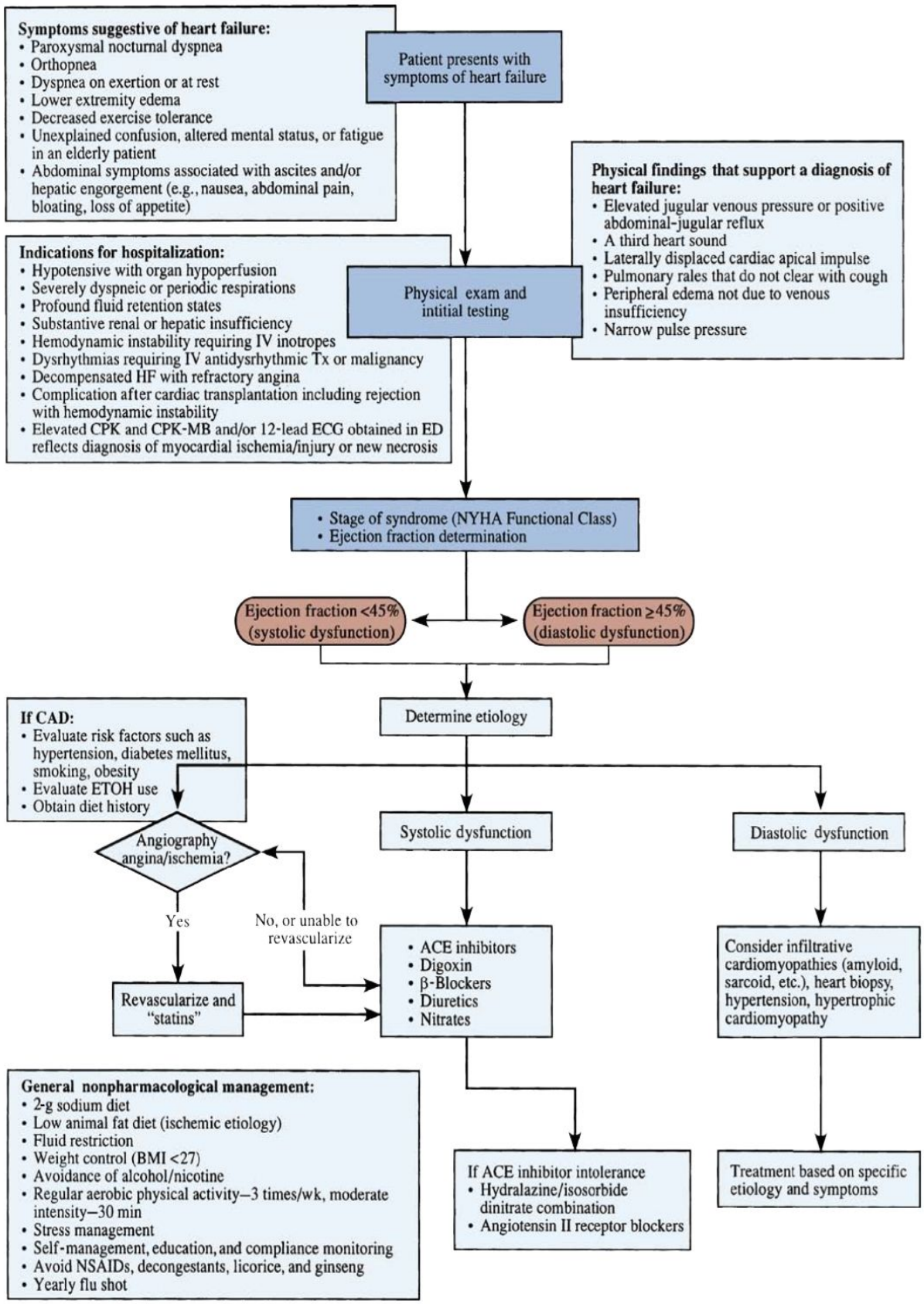


Stages of heart failure and treatment options for systolic heart failure :



Neurohumoral activation and sites of action of drugs used in the treatment of heart failure :




Symptoms suggestive of heart failure:
• Paroxysmal nocturnal dyspnea
• Orthopnea
• Dyspnea on exertion or at rest
• Lower extremity edema
• Decreased exercise tolerance
• Unexplained confusion, altered mental status, or fatigue in an elderly patient
• Abdominal symptoms associated with ascites and/or hepatic engorgement (e.g., nausea, abdominal pain, bloating, loss of appetite)
Patient presents with symptoms of heart failure
Physical findings that support a diagnosis of heart failure:
• Elevated jugular venous pressure or positive abdominal-jugular reflux
• A third heart sound
• Laterally displaced cardiac apical impulse
• Pulmonary rales that do not clear with cough
• Peripheral edema not due to venous insufficiency
• Narrow pulse pressure
Indications for hospitalization:
• Hypotensive with organ hypoperfusion
• Severely dyspneic or periodic respirations
• Profound fluid retention states
• Substantive renal or hepatic insufficiency
• Hemodynamic instability requiring IV inotropes
• Dysrhythmias requiring IV antidysrhythmic Tx or malignancy
• Decompensated HF with refractory angina
• Complication after cardiac transplantation including rejection with hemodynamic instability
• Elevated CPK and CPK-MB and/or 12-lead ECG obtained in ED reflects diagnosis of myocardial ischemia/injury or new necrosis
Physical exam and intitial testing
• Stage of syndrome (NYHA Functional Class)
• Ejection fraction determination
Ejection fraction <45% (systolic dysfunction)
Ejection fraction ≥45% (diastolic dysfunction)
Determine etiology
If CAD:
• Evaluate risk factors such as hypertension, diabetes mellitus, smoking, obesity
• Evaluate ETOH use
• Obtain diet history
Angiography angina/ischemia?
Yes
No, or unable to revascularize
Revascularize and "statins"
Systolic dysfunction
Diastolic dysfunction
• ACE inhibitors
• Digoxin
• β-Blockers
• Diuretics
• Nitrates
Consider infiltrative cardiomyopathies (amyloid, sarcoid, etc.), heart biopsy, hypertension, hypertrophic cardiomyopathy
General nonpharmacological management:
• 2-g sodium diet
• Low animal fat diet (ischemic etiology)
• Fluid restriction
• Weight control (BMI <27)
• Avoidance of alcohol/nicotine
• Regular aerobic physical activity—3 times/wk, moderate intensity—30 min
• Stress management
• Self-management, education, and compliance monitoring
• Avoid NSAIDs, decongestants, licorice, and ginseng
• Yearly flu shot
If ACE inhibitor intolerance
• Hydralazine/isosorbide dinitrate combination
• Angiotensin II receptor blockers
Treatment based on specific etiology and symptoms

Cases

1) **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

2) **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 edema
E. Kerley B lines

3) **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 to128 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
e. Spironolactone

4) **A 55-year-old man is noted to have moderately severe congestive heart failure with impaired systolic function. Which of the following drugs would most likely lower his risk of mortality?**

A. Angiotensin-converting enzyme inhibitors
B. Loop diuretics
C. Digoxin
D. Aspirin

5) A 55-year-old man is noted to have congestive heart failure and states that he is comfortable at rest but becomes dyspneic even with walking to bathroom. On echocardiography, he is noted to have an ejection fraction of 47%. Which of the following is the more accurate description of this patient's condition?

A. Diastolic dysfunction
B. Systolic dysfunction
C. Dilated cardiomyopathy
D. Pericardial disease

Answers

1) **D :** Aortic stenosis will first result in left ventricular failure as a result of increased ventricular pressure as the ventricle tries to pump blood across a narrowed valve. Initially the pressure load will cause a backlog of blood into the lungs, resulting in pulmonary oedema – the first sign of which will be bibasal crepitations (D) before enough fluid accumulates as pleural effusions visible on chest x-ray (A). Earlier signs of pulmonary oedema include upper lobe blood diversion and Kerley B lines as fluid infiltrates the interstitium. If the backlog continues back into the right heart, eventually signs of right-sided heart failure will be evident including raised JVP (B) and bilateral pedal oedema (C). Atrial fibrillation (E) may coexist with aortic stenosis, however it is more commonly associated as a result of mitral stenosis as the enlarged atrium disrupts the normal electrical pathways.

2) **A :** Cardiomegaly (B), bilateral pleural effusions (C), alveolar oedema (D) and Kerley B lines (E) (representing interstitial oedema) are all features that can be seen in a chest x-ray in patients with congestive cardiac failure. Upper lobe diversion is usually seen on chest x-ray and not lower lobe diversion (A).

3) **B :** The answer is b. Inhibition of the renin-angiotensin-aldosterone system by either angiotensin-converting enzyme inhibitors (ACEi) or angiotensin receptor blockers (ARB) has been proven to decrease mortality in patients with symptoms of congestive heart failure and a depressed ejection fraction. All patients with a history of congestive heart failure should be maintained on a beta- blocker and an ACEi or ARB. Most patients will require a diuretic for symptom control. Digitalis glyco- sides decrease rehospitalization rate but have not been shown to improve mortality. Thiazide diuretics are excellent medications for blood pressure control. Our patient, however, has well-controlled blood pressure. The patient is already on a selective beta-blocker and the addition of a non-selective beta- blocker is unlikely to be helpful. Spironolactone provides mortality benefit in patients with NYHA class III or IV heart failure. The patient in this scenario was able to walk two blocks before stopping and would be classified as NYHA class II.

4) A : Angiotensin-converting enzyme inhibitor and beta-blockers decrease the risk of mortality when used to treat CHF with impaired systolic function. For this reason, these agents are the initial choice to treat CHF. They both prevent and can even, in some circumstances, reverse the cardiac remodeling.

5) A : When the ejection fraction exceeds 40%, there is likely diastolic dysfunction, with stiff ventricles. The stiff thickened ventricles do not accept blood very readily. This patient has symptoms with mild exertion which are indicative of functional class III. The worst class is level IV, manifested as symptoms at rest or with minimal exertion. ACE inhibitors are important agents in patients with diastolic dysfunction.

Clinical Pearls

- Congestive heart failure is a clinical syndrome that is always caused by some underlying heart disease, most commonly ischemic cardiomyopathy as a result of atherosclerotic coronary disease or hypertension.
- Heart failure can be caused by impaired systolic function (ejection fraction <40%) or impaired diastolic function (with preserved systolic ejection fraction).
- Chronic heart failure is a progressive disease with a high mortality. A patient's functional class, that is, his or her exercise tolerance, is the best predictor of mortality and often guides therapy.
- The primary goals of therapy are to relieve congestive symptoms with salt restriction, diuretics, digoxin, and vasodilators and to prolong survival with angiotensin-converting enzyme inhibitors or certain beta-blockers.
- Angiotensin-converting enzyme inhibitors together with beta blockers are the agents of choice for CHF.
- Aortic stenosis produces progressive symptoms such as angina, exertional syncope, and heart failure, with increasingly higher risk of mortality. Valve replacement should be considered for patients with symptoms and severe aortic stenosis, for example, an aortic valve area less than 1 cm^2.