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Heart Failure

★ Objectives:

1. Definition of heart failure (HF)
2. Know Pathophysiology of HF
3. Diagnosis of HF
4. Describe the Causes of HF
5. Know the Classification of HF
6. Diagnostic tests of HF
7. Different treatment of HF
8. Side effects of medication of HF
9. Management of cardiac risk factors for HF
10. Role of devices and lifestyle in HF treatment

★ Resources Used in This lecture:

Slides, Davidson's, Master The Board, Step-up, Kaplan CK, CLASS NOTES

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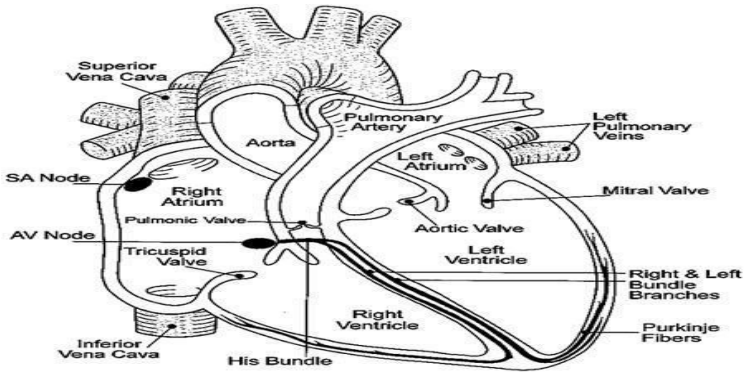
Introduction

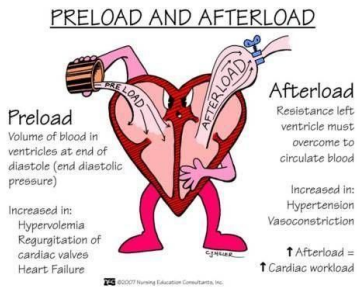
Anatomy:

- The heart is a hollow muscular organ specialized for pumping blood.
- Blood supply from the right and left coronary arteries. These arteries branch off from the aorta just above the Aortic Valve.
- The nerve supply from two sets of nerves originating in the medulla of the brain. One set, the branches from the vagus nerve, keeps the heart beating at a slow, regular rate. The other set, the adrenergic nerves(T1-T4), speeds up the heart.



Physiology:

- Stroke volume is the volume of blood ejected from heart per heartbeat.
- Cardiac output(CO) (stroke volume x heart rate) is determined by your body demand normally 4-8 L/Min. It increases during exercise and stress.
- Ejection Fraction (EF) 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 (Think of it as the heart loading up; more load → the greater the stretch → greater stroke volume; this is called Frank-starling mechanism).
- 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.



Blood From IVC & SVC to the right atrium → to the right ventricle → ventricle contract →to the lungs → back to the left atrium → to the left ventricle → ejection → to the aorta →to the whole body.

Congestive Heart Failure

It is a complex syndrome that can result from any structural or functional cardiac disorder that impairs the ability of the heart to maintain sufficient cardiac output (impaired filling or ejection) to maintain the demand of the body.

- Most common presentation in cardio. Because it's the final common pathway of cardiac diseases.
- Progressive disease → leads to death. (Median mean of survival after diagnosis 5 years.)

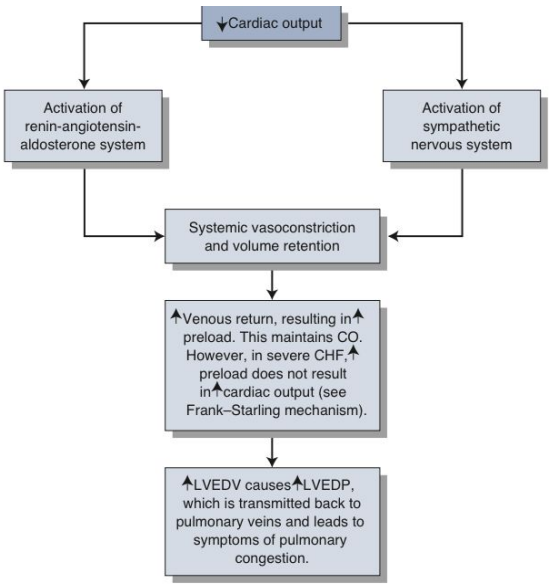
Note: It's not a disease it's → a Syndrome (clinical signs & symptoms) that result from an underlying disease.

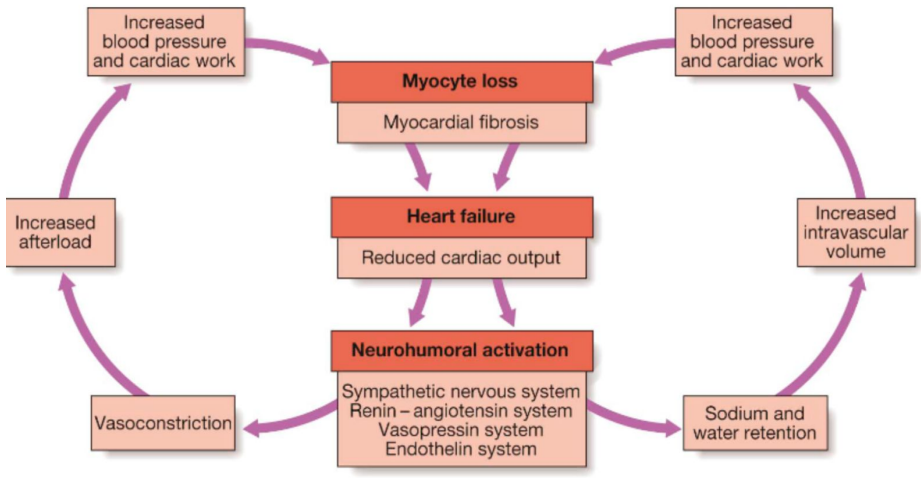
Pathophysiology:

In patients without 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.

- Reduce in CO→ will cause Neurohormonal activation → to increase blood pressure and cardiac work.
- Neurohormonal activates the following systems, 1-Sympathetic nervous system 2- Renin-angiotensin system 3- vasopressin system 4- Endothelin.
- Causing either vasoconstriction → increasing the afterload **or/and** sodium and water retention → increasing intravascular volume.

These hemodynamic changes will trigger a compensatory mechanism,but with time these mechanisms will be overwhelmed and become pathophysiological affecting both preload and afterload.

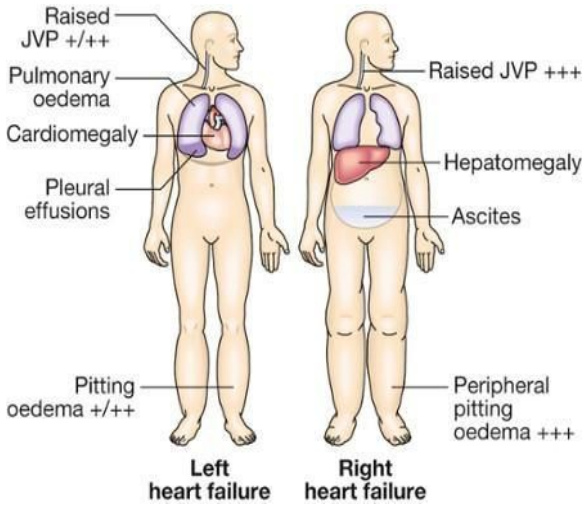




N/H changes	Favorable effect	Unfavorable effect
↑ Sympathetic activity	• ↑ HR and contractility, • Vasoconstriction→ ↑ Venous return filling	Arteriolar constriction ↑ → After load ↑ workload → ↑ O_2 consumption
↑ Renin→ → ↑ Angiotensin → ↑ Aldosterone.	Salt & water retention ↑ Venous return.	• Angiotensin-II will lead to Arteriolar constriction ↑ After load. • Increased salt & water retention → peripheral and pulmonary edema.
↑ vasopressin	Same effect	Same effect
↑ Interleukins &TNFa	May have roles in myocyte hypertrophy	Apoptosis
↑ Endothelin	Vasoconstriction→ ↑ Venous return	↑ After load

Clinical Feature:

- Shortness of breath (dyspnea)/paroxysmal nocturnal dyspnea
- Orthopnea[1]
- Fatigue and weakness (low CO)
- Lower extremity edema
- Abdominal symptoms with or without → Ascites and/or hepatomegaly (volume retention) (Nausea,vomiting,pain, loss of appetite)
- Decrease exercise tolerance
- Unexplained confusion,altered mental status in elderly (Low CO)



Physical findings

- Jugular Venous distension (JVD)
- 3rd heart sound
- Lateral displacement of cardiac apical impulse
- Pulmonary rales
- pleural effusion
- Pale,cold sweaty skin

Three main etiology for CHF :

1.Myocyte loss 2. Inappropriate workload 3. Restricted filling

[1] Sensation of breathlessness in the lying position, relieved by sitting or standing.

Main Etiology	Cause	Examples	Features
1.Myocyte loss Post MI, chronic ischemia, C.T infection, poison.	A. Reduced ventricular contractility  Remember left ventricular failure is a life threatening emergency.	MI (segmental dysfunction) *CAD most common cause.	• Heart muscle→ weak pumping ability and often leaving permanent damage. • It can lead to a weakened heart muscle.
		Myocarditis/cardiomyopathy[2] (global dysfunction)	Cause progressive ventricular dilatation.
2. Inappropriate workload	A. ventricular outflow obstruction (pressure overload)	• Hypertension, • aortic stenosis (left heart failure) • Pulmonary hypertension, • pulmonary valve stenosis (right heart failure)	**Initially,** concentric ventricular hypertrophy **Later,** ventricular dilatation and rapid clinical deterioration.
	B. ventricular inflow obstruction	Mitral stenosis, tricuspid stenosis.	Ex: Atrial fibrillation is common and often causes marked deterioration because ventricular filling depends heavily on atrial contraction.
	C. ventricular volume overload	• Left ventricular volume overload (e.g. aortic regurgitation) • Ventricular septal defect[3] • Right ventricular volume overload (e.g. atrial septal defect) • Increased metabolic demand (high output).	Dilatation and hypertrophy ad help to maint a normal cardiac output. However, secondary changes in the myocardium lead to impaired contractility and worsening heart failure
	D. Arrhythmia	Atrial fibrillation	Tachycardia does not allow for adequate filling of the heart, →low cardiac output and back pressure.
		Tachycardia cardiomyopathy	Causes myocardial fatigue.
		Complete heart block	Bradycardia limits cardiac output, even if stroke volume is normal.(affecting HR)
3. Restricted filling (Heart here is preserved)	A. Diastolic dysfunction	Constrictive pericarditis	Inflammation in this part of the heart causes scarring, thickening, and muscle tightening, or contracture.
		Restrictive cardiomyopathy	Bi-atrial enlargement (restrictive filling pattern and high atrial pressures).
		Left ventricular hypertrophy and fibrosis Cardiac tamponade	Good systolic function but poor diastolic filling.

Chronic disease of heart muscle→ many causes, including infections, alcohol abuse and the toxic effect of drugs, such as[2] cocaine or some drugs used for chemotherapy

[3] a hole in the heart, is a common heart **defect** that's present at birth (congenital). The hole occurs in the wall that separates the heart's lower chambers (**septum**) and allows blood to pass from the left to the right side of the heart.

Classifications:

1. Diastolic/Systolic dysfunction:

Diastolic dysfunction	Systolic dysfunction
-Poor ventricular filling and high filling pressures(either impaired relaxation or increase stiffness of ventricle or both). -Preserved ejection fraction. -No s4 gallop,No apex beat displacement	-Impaired myocardial contraction. -Impaired Ejection Fraction → Low. -S4 gallop -apex beat displacement

★ **Systolic and diastolic dysfunction often coexist, particularly in patients with coronary artery disease.**

2. High/Low Output Failure:

High output Failure	Low output Failure
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.

3. NYHA[4] CLASSIFICATION OF HF:

Class I:	Class II:	Class III:	Class IV:
No limitation of activities; no symptoms with ordinary activities.	Mild limitation of activity; comfortable with rest or mild exertion.	Marked limitation of activities; comfortable at rest only.	Symptoms of HF occur at rest, any physical activity brings discomfort.

4. Acute/ Chronic

Acute/Acute Pulmonary edema[5]	Chronic (ex;progressive valvular dysfunction)
De novo: -Sudden onset of dyspnea→ acute respiratory distress. -inappropriate brady or tachycardia -JVP raised & 3rd heart sound is heard - No Ventricles dilatation no apex-beat displacement. **Acute on chronic:** will have additional feature of long standing CHF, the cause commonly upper resp. Tract infection, atrial Fib,PE or inappropriate cessation of diuretics.	- Patient follow relapsing & remitting course with periods of stability in between. -Clinical picture depends on the underlying heart disease, type of heart failure, neurohormonal changes that developed. Sometimes associated with: -Weight loss (cardiac cachexia) -Poor tissue perfusion & -Skeletal muscle atrophy

[4] New York Heart Association Functional Classification
[5] is the worst & most severe form of CHF. It's rapid accumulation of fluid in the lungs.

5. Left/Right & biventricular sided Heart failure:

1. Left-sided heart failure	*2. Right-sided heart failure*
• Reduction in left ventricular output. • Increase in left atrial and pulmonary venous pressure. • **Acute**→ pulmonary edema. • **Gradual**, ex; mitral stenosis, leads to reflex pulmonary vasoconstriction→ causes pulmonary hypertension, which can, in turn, impair right ventricular function. Simply: Here blood is not going normally to the system instead it's getting compressed back to the lungs, causing fluid from BV to lung.	• Reduction in right ventricular output. • Increase in right atrial and systemic venous pressure. • Causes of isolated right heart failure include chronic lung disease (cor pulmonale[6]), pulmonary embolism and pulmonary valvular stenosis. Simply: blood stays in the system can't move to the lungs, here fluid from BV to the lower limbs and congest the liver.
	

3. Biventricular heart failure[7]
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.

[6] Alteration in the structure and function of the right ventricle (RV) of the heart caused by a primary disorder of the respiratory system such as pulmonary hypertension.
[7] Failure of the left and right heart.

Differential Diagnosis:

- Pericardial disease
- Liver disease
- Nephrotic syndrome
- Protein losing enteropathy

Diagnostic Tests:

Echocardiography *Gold standard

- Best initial → TTE[8]

-It assist the function of both ventricles, valvular abnormality, intracardiac shunts, wall motion abnormality (signify CAD).
-Most important because it's the **only** way to distinguish systolic from diastolic failure.
-It evaluate Ejection Fraction.

Note: -TEE[9] is more accurate for evaluating heart valve function and diameter, but not for evaluating CHF.
- Most accurate to assess EF→ Nuclear ventriculographyrarely needed.

Other tests are used not to diagnose CHF, They are used to diagnose the cause of CHF:

Tests to know the etiology	
EKG	MI (old or recent) ,Heart block,Arrhythmia, LBBB
Chest X-ray	Dilated cardiomyopathy, pleural effusion,(Initial and best for pulmonary edema)
Cardiac catheter	Precise valve diameter, septal defects (when CAD or valvular suspected)
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 - Hemochromatosis
Endomyocardial Biopsy	Rarely done; to exclude infiltrative disease such as sarcoid or amyloid, for unexplained CHF.

[8] Transthoracic Echo
[9] Transesophageal Echo

Management

Systolic Dysfunction:

★ **loop diuretics(furosemide,torsemide , bumetanide)**: Initial therapy used to relieve the symptoms by ↓ preload. Does not reduce the mortality.

Note : not giving to asymptomatic patient

★ **ACE inhibitors :** initial therapy used to ↓ afterload by vasodilatation and ↓ preload by absorption of the fluid and it reduces the mortality. ADRs → angioedema + cough

Note: If patient developed cough switch from ACEi to ARB.

Note: If patient developed hyperkalemia or renal impairment or pregnant→ switch from ACEi to Hydralazine(arterial dilator) with isosorbide dinitrate (Venodilator).

Both Hydralazine with isosorbide dinitrate and ARB reduce the mortality

★ **B-Blockers (metoprolol . bisoprolol . carvedilol)** :anti-ischemic , antiarrhythmic , decreased the heart rate (decrease oxygen consumption increasing filling time) & reduced remodeling of the heart → It decreases mortality.. used in type 1,2 and 3

Note : The combination of B blockers and an ACE inhibitors required for patient with LVEF less than 40% either symptomatic or asymptomatic

★ **spironolactone(potassium sparing diuretic that is used in chronic CHF)** : aldosterone antagonist used in advance stage 3 and 4 only and it reduces the mortality.

Note : if the patient developed gynecomastia, impotence (cause its structurally similar to progesterone) switch to eplerenone.

Digoxin : + inotropic effect used only relieve symptoms with EF<40%have been continues despite (ACEi , BB , LD and spironolactone) and does not improve the mortality. *Adrs: Vagotonic, Arrhythmogenic.*

lifestyle modification : such as Sodium and fluid restriction , Weight loss ,smoking cessation ,Restrict alcohol and use Exercise program

Correction of reversible causes such as Ischemia Valvular heart disease Thyrotoxicosis, Shunts, Arrhythmia

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)

★ **devices** : performed only when patient not get benefit from medication and it has been shown reduce the mortality

- **An ICD (implantable cardiovascular defibrillator)**it's indicated for patients at least 40 days post-MI, EF <35%, and class II or III.
- **Cardiac resynchronization therapy (CRT):** biventricular pacemaker indications are similar to ICD except these patients also have prolonged QRS duration >120 msec

★ **Cardiac transplantation** : last choice if the medication and devices can't control symptoms.

Diastolic Dysfunction:

The standard treatment for diastolic dysfunction is β blockers and Diuretics

Digoxin and spironolactone should NOT be used.

ACE inhibitors and ARBs—benefit is not clear for diastolic dysfunction.

No medication have proven the mortality in diastolic dysfunction

Management of Acute decompensated Heart Failure (Acute Pulmonary edema)

A. **Oxygen**
B. **loop diuretics (furosemide):** most important drug that decreases the preload *Best initial therapy
C. **Nitrate (IV) :** that decrease the afterload
D. **Morphine can be used**

Note : If pulmonary edema continuous despite these 4 so should added dobutamine **(increased contractility & decrease afterload)**

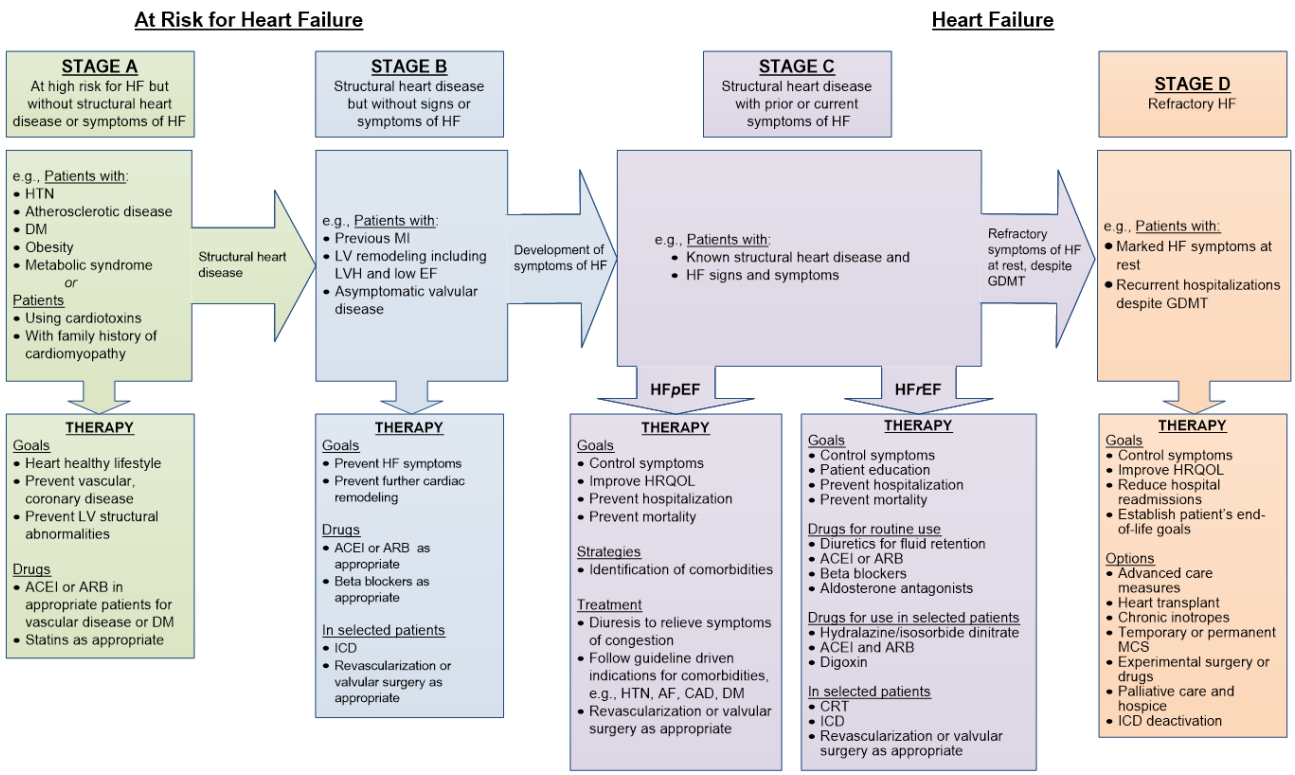
Remember: - We don't give β blockers in Acute cases (only in stable patient).

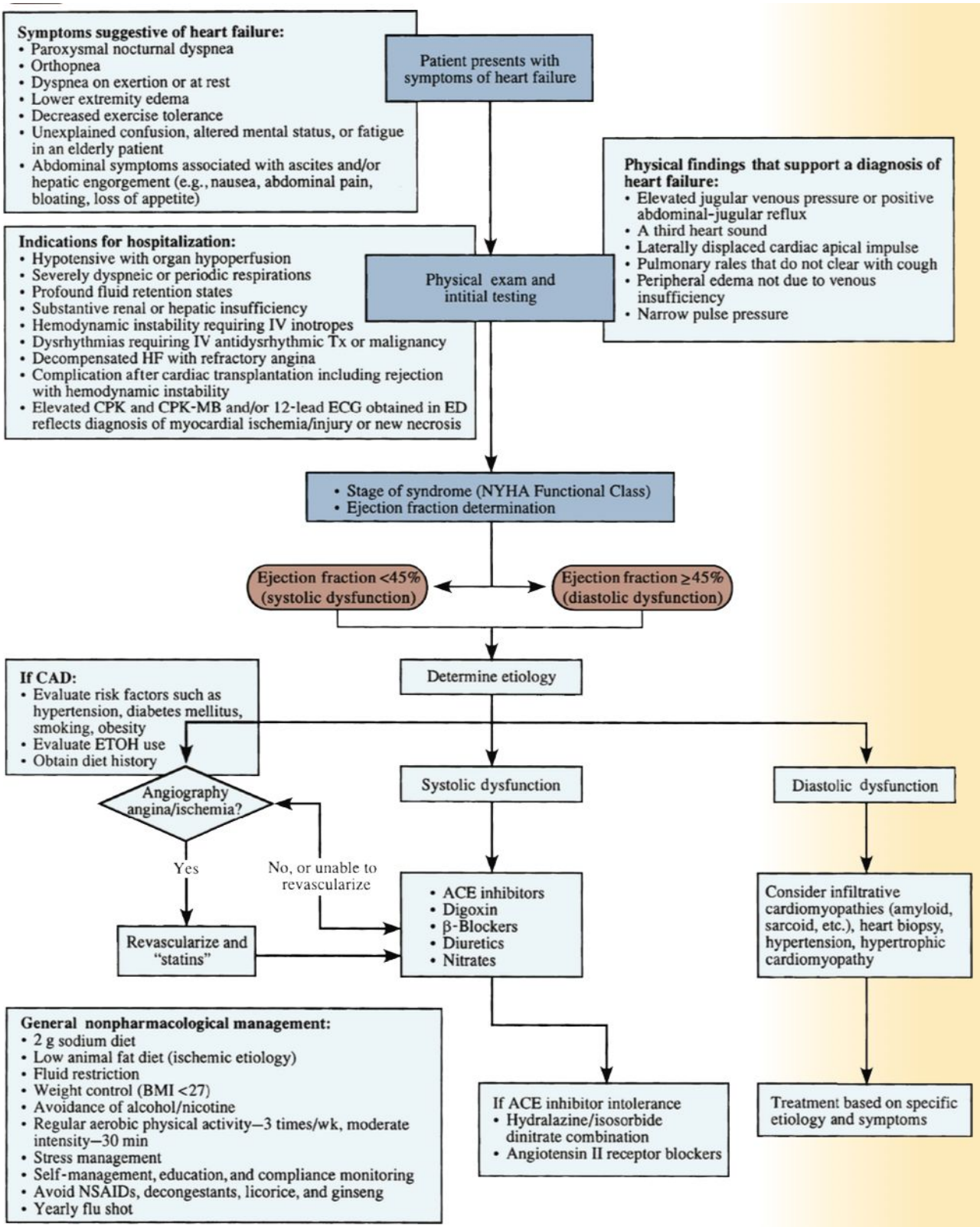
- **Digoxin** (not used in acute settings, usually used if there was AFib).

Contraindicated Medication on CHF

★ **Metformin :** may cause potentially fatal lactic acidosis.
★ **Thiazolidinediones (glitazone):** causes fluid retention.
★ **NSAIDs :** may increase risk of CHF exacerbation.
★ **some of Calcium Channel blockers :** may raises mortality.

Summary :




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

MCQ's:

1. A 60-year-old female cigarette smoker complains of fatigue and dyspnea. The most specific evidence for congestive heart failure in this patient would be?

A. Ankle edema
B. Wheezes
C. S3 gallop
D. Weight gain

2. Which of the following is a precipitating cause of high-output cardiac failure?

A. Alcoholic cardiomyopathy
B. Hypertension
C. Myocardial infarction
D. Multiple PVCs
E. Thyrotoxicosis

3. Which one of these drugs is Contraindicated Patients with HF?

A. NSAIDs
B. Spironolactone
C. Penicillin
D. Digoxin

4. What is the most common cause of death from CHF ?

A. Pulmonary edema
B. Myocardial infarction
C. Arrhythmias/sudden death
D. Emboli

5. 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)

6. 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 + vasodilatation (ACEI or ARBs)
D. β blockers and vasodilatation (ACEI or ARBs)

7. CHF patient on medication, suddenly he develops Nausea, vomiting, Headache, ECG shows Atrial tachycardia with A/V Block. Which one of the following drugs caused this side effect?

A. Carvedilol
B. Spironolactone
C. Digoxin
D. Enalapril

ANSWERS:
1.C
2.E
3.A
4.C
5.D
6.
7.C