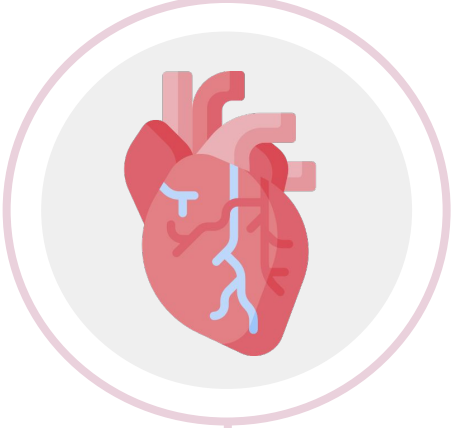
Heart failure (1) (Etiology & Diagnosis)



Objectives :

- ★ Know different classifications of heart failure.
- ★ Know the causes and precipitation factors for heart failure decompensation.
- ★ Describe the Pathophysiology, therapies that improve survival, and prognosis.

Editing file

Color index

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- Text book
- Important
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- Extra

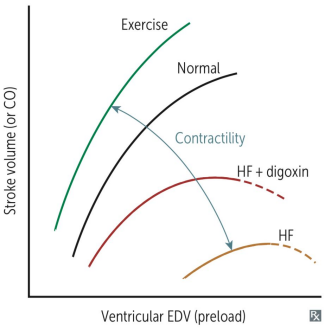
Review of basics

◀ CO variables and Starling curve

- **Cardiac output is determined by SV and HR; SV is determined by the following:**
  - **Preload** (the volume and pressure of blood in the ventricles at the end of diastole)
  - **Afterload** (The pressure that the heart must overcome to eject blood)
  - **Myocardial contractility**

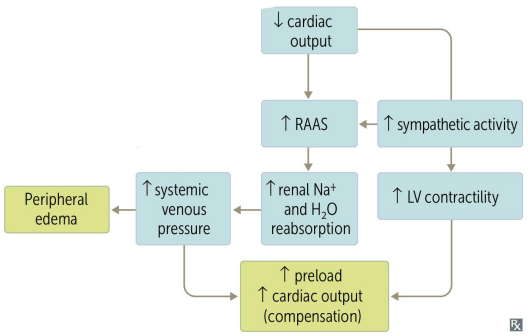
SV increases with ↑Contractility, ↑preload and ↓afterload

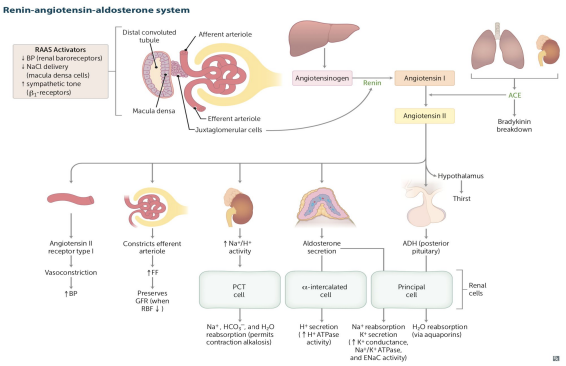
- **What is Starling curve?**
  - Starling's law states that the stroke volume is directly proportional to the diastolic filling (i.e. the preload or ventricular end-diastolic pressure). As the preload is increased, the stroke volume rises (normal). Increasing contractility (e.g. increased with sympathetic stimulation) shifts the curve upwards and to the left . If the ventricle is overstretched the stroke volume will fall . In heart failure the ventricular function curve is relatively flat (Shift to the right) so that increasing the preload has only a small effect on cardiac output.



◀ What happens when CO is decreased?

- **Activation of the SNS** Improves ventricular function by increasing heart rate and myocardial contractility. Constriction of venous capacitance vessels redistributes flow centrally, and the increased venous return to the heart (preload) further augments ventricular function via the Starling mechanism . Sympathetic stimulation, however, also leads to arteriolar constriction, this increasing the afterload which would eventually reduce cardiac output.
- **Activation of RAAS** It is very imp to know how RAAS being activated ,The fall in cardiac output and increased sympathetic tone lead to diminished renal perfusion, activation of the renin–angiotensin system, and hence increased fluid retention. Salt and water retention further increases venous Cardiovascular disease pressure and maintains stroke volume by the Starling mechanism . As salt and water retention increases, however, peripheral and pulmonary congestion causes oedema and contributes to dyspnoea. Angiotensin II also causes arteriolar constriction, thus increasing the afterload and the work of the heart.
- **Natriuretic peptides** These are released from the atria (atrial natriuretic peptide, ANP), ventricles (brain natriuretic peptide, BNP – so called because it was first discovered in the brain) and vascular endothelium (C-type peptide). They have diuretic, natriuretic and hypotensive properties. The effect of their action may represent a beneficial, albeit inadequate, compensatory response leading to reduced cardiac load (preload and afterload).





◀ Ejection Fraction (EF)

- The effectiveness of the ventricles in ejecting blood is described by the ejection fraction, which is the fraction of the end-diastolic volume that is ejected in one stroke volume. Normally, ejection fraction is approximately 0.55, or 55%. The ejection fraction is an indicator of contractility, with increases in ejection fraction reflecting an increase in contractility and decreases in ejection fraction reflecting a decrease in contractility. Thus EF= stroke volume/end-diastolic volume

Introduction to HF

◀ Definition of heart failure فشل القلب

- Heart failure is, **complex** (because it has many types) **clinical syndrome** (not a disease, its a collection of symptoms and signs that resulted from a disease) that can result from any **structural** (valvular heart disease, coronary artery disease) or **functional** (vitamins deficiency, thyroid disease) cardiac disorder that **impairs the ability of the ventricle to fill** (impaired filling{diastolic HF}) (e.g. LVH) **and/or eject (**impaired contractility{systolic HF}) **blood to meet the body demand.**

- An abnormality of cardiac **structure** or **function** leading to failure of the heart to deliver oxygen at a rate commensurate with the requirements of the metabolizing tissues (the patient will feel clammy, fatigued and cold), despite normal filling pressures (or only at the expense of increased filling pressures) (the patient will usually present with fluid overload (peripheral pitting edema, SOB, ascites))
- HF is characterized by signs and symptoms of intravascular and interstitial volume overload and/or manifestations of inadequate tissue perfusion.

Classification of heart failure			
Related to EF **"Most Used classification nowadays"**	(it's important to differentiate between them because they have different management) - HFrEF (reduced ejection fraction: EF<40%) - HFmEF (mildly impaired ejection fraction: EF 40-49%) - HFpEF (preserved ejection fraction: EF≥50%)"If EF is >60% it doesn't mean that the patient is normal" - A normal heart's ejection fraction may be between 50 and 70 percent. You can have a normal ejection fraction measurement and still have heart failure (called HFpEF or heart failure with preserved ejection fraction).		
Related to time-course	- New onset	- Transient	- Chronic
Related to progression	- Acute	- Stable	- Worsening
Related to location	- Left heart	- Right heart	- Combined

◀ The burden of heart failure

- Number of patients: 21 million adults worldwide are living with HF, this number is expected to rise.
- Economic burden: in 2012, the overall worldwide cost of HF was nearly $108 billion.
- Mortality:50% of HF patients die within 5 years from diagnosis.
- Rehospitalization: HF is the number one cause of hospitalization for patients aged >65 years (increase with age due to development of HF risk factors such as hypertension, diabetes and coronary artery disease)
- Comorbidities: the vast majority of HF patients has 3 or more comorbidities.

◀ Prevalence of heart failure

- Prevalence 0.4-2% overall, 3-5 % in over 65s, 10% of over 80s and > 10% also have AF
- Commonest medical reason for admission with an annual mortality of 60% over 80s
- Progressive condition (worsen with time, since HF is incurable, we need a management team to treat risk factors because medications alone are not enough)- median survival 5 years after diagnosis
- Family history is usually positive for patients with HF
- **REMEMBER:** Left ventricular failure is a true life threatening emergency (always assume HF is an emergency until proven otherwise)

Introduction cont.

◄ Etiology of heart failure

- It is a **common endpoint** for many diseases of cardiovascular system.
- **It can be caused by** Inappropriate workload (volume or pressure overload) or Restricted filling or Myocyte loss
- **Heart failure is usually as a result from a chronic process but it may also result from an acute insult** to cardiac function, such as a large myocardial infarction, valvular disease, myocarditis, and cardiogenic shock.

What are the most common causes of left HF? (can be dependant on the region. In south america, chagas disease is one of the most common causes. Alcohol or toxic cardiomyopathy is common in western regions and hypertension is the most common cause in Africa).

1. **Most common:** Coronary artery disease (IHD) مرض الشرايين التاجية
2. **2nd:** Hypertension
3. ★ **3rd:** VHD (e.g. AS) مرض صمام القلب (Regurgitating valve patients are more prone to develop HF)
4. **4th:** Dilated cardiomyopathy اعبلال عصله القلب السوسعى (it is a genetic disorder),
5. Chagas disease in South America
6. Viral myocarditis (due to covid-19) can cause HF

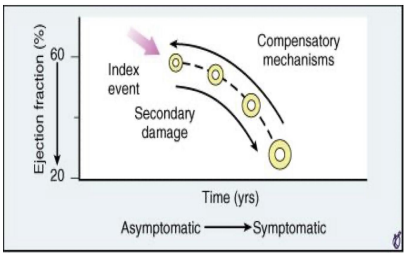
We can **classify the causes** of HF into four main categories according to their pathophysiological alterations/ abnormalities

Volume overload	Pressure overload	Loss of muscles	Restricted filling
1. **Regurgitate valve** (MR,AR) 2. High output status: anaemia, sepsis, thyrotoxicosis, paget's disease, arteriovenous fistula 3. Renal failure 4. Iatrogenic: postoperative fluid infusion	**1. Systemic hypertension** 2. Outflow obstruction	**1. Coronary artery disease** 2. cardiomyopathy 3. **Post MI** **4. Chronic ischemia** 5. Connective tissue diseases 6. Poisons (alcohol, cobalt, Doxorubicin)	1. **Pericardial disease: constrictive pericarditis**(stiff pericardium), pericardial effusion. 2. Restrictive cardiomyopathy 3. Valvular Heart disease: mitral, aortic, tricuspid, pulmonary. 4. Arrhythmia: tachyarrhythmia, atrial, ventricular, bradyarrhythmia, sinus node dysfunction. 5. Endocardial disease: with/without hypereosinophilia, endocardial fibroelastosis 6. Conduction disorders: atrioventricular block

◄ Background of HF pathophysiology

Heart failure pathophysiology:

1. Index event
2. Compensatory mechanisms
3. Maladaptive mechanisms



Heart failure usually begins after an index event (etiology) such as MI that produces a decline in the pumping capacity of the heart, in response to this decline, a variety of compensatory mechanisms are activated that are designed to maintain cardiovascular homeostasis for periods of months to years; during that period, patients tend to remain asymptomatic. When these compensatory mechanisms are excessively activated, they themselves can cause secondary damage to the heart and circulation. It is this secondary damage that drives the disease process of heart failure forward, Largely through the mechanism of cardiac remodeling. As the heart remodels, it not only gets bigger, but the cardiac walls get thinner and the pumping capacity of the heart declines. With the transition from a small heart to a big heart, patients at this stage generally go from asymptomatic to symptomatic(such as orthopnea, SOB, PND, lower limb edema, ascites) heart failure .

Introduction cont.

◀ Changes in HF

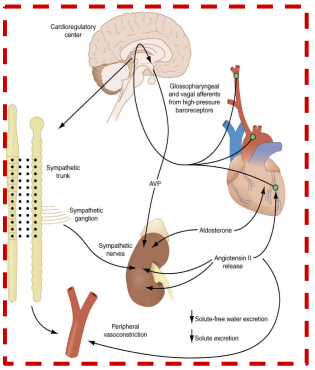
- Injury to myocytes due to MI or other cause (index event) will cause ventricular remodeling by dilating the ventricle which will lead to electrical instability (AF,VT) and reduced EF ultimately causing neurohumoral imbalance.

- **An imbalance occurs in three key neurohumoral systems:**
  1. Renin-Angiotensin- Aldosterone system
  2. Sympathetic nervous system
  3. natriuretic peptide system

- The systemic responses in the renin–angiotensin–aldosterone and sympathetic nervous systems cause further myocardial injury, and have detrimental effects on the blood vessels, and various organs, thereby creating a pathophysiological 'vicious cycle'. The natriuretic peptide system has a protective function, which can counterbalance these detrimental effects.

1 Hemodynamic changes:

Hemodynamic changes associated with HF: MAP, CO(HR*SV) and systemic vascular resistance



- From hemodynamic stand point HF can be secondary to systolic dysfunction or diastolic dysfunction.
- The initial manifestations of hemodynamic dysfunction are a reduction in stroke volume and a rise in ventricular filling pressures under conditions of increased systemic demand for blood flow. This stimulates a variety of interdependent compensatory responses involving the cardiovascular system, neurohormonal systems, and alterations in renal physiology.

2 Neurohormonal changes:

In the short term, these 'neurohormonal' systems induce a number of changes in the heart, kidneys, and vasculature that are designed to maintain cardiovascular homeostasis. However, with chronic activation, these responses result in haemodynamic stress and exert deleterious effects on the heart and the circulation.

N/H changes	Favorable effect	Unfavorable effect
★ **↑ Increased sympathetic activity**	- ↑ HR and contractility - Vasoconstriction⟶ ↑ Venous return, ↑ filling	- ↑ Arteriolar constriction ⟶ ↑Afterload ⟶ ↑ workload ⟶ ↑ O2 consumption
★ **↑ Renin-Angiotensin-Aldosterone**	- Salt & water retention (total body sodium will increase but we will also retain water which will dilute the sodium (the concentration won't change). So if we see low sodium concentration it will be a bad sign) ⟶ ↑ Venous return	- 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 a role in myocyte hypertrophy	- Apoptosis
↑ Endothelin	- Vasoconstriction ⟶ ↑ Venous return	- ↑ Afterload

3 Cellular changes:

- Hypertrophy, loss of myocytes and increased interstitial fibrosis.

Forms of heart failure

HF has different effects on different parts of the heart, there are many types of HF resulting in different symptoms, onsents, etc. For this reason different terms are used to describe HF but the commonest one used nowadays is HFrEF vs HFpEF.

◄ Systolic VS Diastolic Dysfunction

HFpEF: HF with preserved ejection fraction
HFrEF: HF with reduced ejection fraction

Systolic dysfunction فشل القلب الانقباضي (HFrEF)[1]	★ Diastolic dysfunction فشل القلب الانبساطي (HFpEF)[1]

• Owing to **impaired contractility**
• **EF is reduced (<45%)**
• **Causes include:**
  ○ Ischemic heart disease or after a recent MI—infarcted cardiac muscle does not pump blood. Infarction → Dilation → Regurgitation → CHF
  ○ HTN resulting in cardiomyopathy
  ○ Valvular heart disease
  ○ Myocarditis (postviral)
  ○ Less common causes: Alcohol abuse, radiation, hemochromatosis, thyroid disease
Have more worse prognosis than HFpEF
 	
Systolic Dysfunction=dilated cardiomyopathy= Low EF
ALL CAN BE USED INTERCHANGEABLY
	• Owing to **impaired ventricular filling** during diastole (hence decreased cardiac output), because of either: Impaired relaxation or Increased stiffness of ventricle or both.
	• **EF is preserved (>45-50%)**
	• Diastolic dysfunction is less common than systolic dysfunction.
	• HTN leading to myocardial hypertrophy is the most common cause of diastolic dysfunction.
	• **Risk factors:** Age, female, HTN, LVH, ischemia, DM, Obesity, RCM and HCM.
	• **Factors associated with decompensation:** uncontrolled / labile HTN, AF, ischemia, volume overload and extracardiac cause.

What is decapitated blood pressure?

- Once HF is established and, especially, in patients with advanced HF, **SBP is usually low (but high diastolic blood pressure),** even in those who presented initially with HTN. This phenomenon has been called 'decapitated hypertension', that is, patients who have had HTN at the outset, progressively develop normal and even low BP as HF worsens and becomes more severe.

Note: Usually both systolic and diastolic dysfunctions present simultaneously
Based on echo we can differentiate between HFrEF and HFpEF

◄ High VS Low output HF

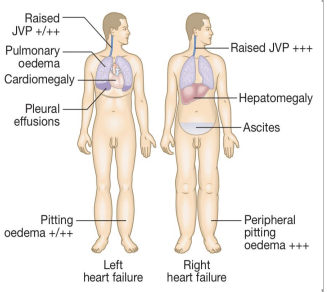
High Output HF فشل القلب عالي النتاج	Low Output HF فشل القلب منخفض النتاج
Certain medical conditions **increase demands on cardiac output,** causing a clinical picture of heart failure due to an excessively high cardiac output. **(e.g. severe anemia, thyrotoxicosis or pregnancy, A/V fistula, Beriberi and Paget's disease)**	**Cardiac output is inadequate** to perfuse the body (i.e ejection fraction <40%), or can only be adequate with high filling pressures.

1- Scenario from doctor:

- **HFPEF**: old 70 year old lady with high BP (190/80) presents with symptoms and signs of heart failure. Echo shows normal EF. we measure the septum and it's 12-13 mm and LV hypertrophy with impaired relaxation. ecg and enzymes will be normal and relatives say she always had this high BP
- **HFREF:** 60 years old, chest pain, shortness of breath physical examination will show edema, crackles and raised JVP, ECG will show STEMI. Patient will be cold bc of vasoconstriction. Echo will show reduced EF Diagnosis will be acute HFREF.
- Total body Na and water are high in HF patient not only water

Right and Left HF

◀ Left VS Right sided HF (Females slides)

	Left heart failure فشل القلب الايسر	Right heart failure فشل القلب الايمن
Patho	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 in turn impairs right ventricular function. (Causes were discussed earlier) **Hallmark: Increased LVEDP**	There is a **reduction in right ventricular output** and an **increase in right atrial and systemic venous pressure.** **The most common cause of right Hf is left HF** (present as congestive HF), other causes include: • Pulmonary HTN and chronic lung disease (cor pulmonale) • Pulmonary embolism and RV infarction • Mitral stenosis and Pulmonic valve stenosis **Hallmark: increased RVEDP and RA**
Symptoms[1]	• **Dyspnea[2]:** Difficulty breathing secondary to pulmonary congestion/edema. Dyspnea (shortness of breath) is the indispensable clue to the diagnosis of HF • **Orthopnea:** Difficulty breathing in the recumbent position; relieved by elevation of the head with pillows" the severity can be determined by number of the pillows" • **Paroxysmal nocturnal dyspnea (PND):** awakening after 1 to 2 hours (time required for the fluid to shift from lower to upper lobe) of sleep due to acute shortness of breath (SOB), usually caused by pulmonary edema. • **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 (legs\ankle edema + sacral edema in bed bound patients):** 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 • **Abdominal symptoms:** anorexia, Nausea, abdominal fullness(ascites), right hypochondrial pain (The pain fibers of Glisson's capsule are stimulated when the capsule is stretched. Thus any diseases that stretches the capsule such as an enlarged liver can cause liver pain.
Signs[1]	• **Displaced** and sustained **PMI[3]** (usually to the left) due to **cardiomegaly** (Normally, the apical impulse is located in the 5th intercostal space, but do to cardiomegaly/LVH it will be displaced to the 6th intercostal space) • **Pathologic S3 (ventricular gallop):** low pitched sound that is heard during rapid filling of ventricle, usually due to **sudden deceleration of blood** as elastic limits of the ventricles are reached leading to vibration of the ventricular wall by blood filling, it's common in children. • **S4 gallop** (at the end of diastole)Exact mechanism is not known, could be due to (forceful) contraction of atrium against stiff (noncompliant) ventricle ★ **Crackles**/rales (inspiratory) at lung bases (Bibasal crepitations)	• **Jugular venous distention (JVD)** • Painful **Hepatomegaly/hepatojugular reflux** • **Ascites** • Cardiac cirrhosis (on the long run) • Right ventricular heave 

Biventricular Heart failure

- In biventricular failure, both sides of the heart are affected. This may occur because the disease process, such as dilated cardiomyopathy or ischaemic heart disease, affects both ventricles: ↑LVEDP → ↑LA pressure → ↑pulmonary capillary pressure → ↑PA pressure → ↑RV pressure → ↑RA pressure → CHF

1- You cannot differentiate between different heart failure classifications using signs and symptoms alone, you need investigations to confirm which type it is
2- Due to pulmonary edema→fluid bulge out of lungs vasculature into alveoli→accumulation of fluid in alveoli will prevent oxygen enter the alveoli
3- Point of maximal impulse, the location at which the cardiac impulse can be best palpated on the chest wall. Frequently, this is at the fifth intercostal space at the midclavicular line. When dilated cardiomyopathy is present, this can be shifted laterally.

Acute and Chronic HF

◄ Acute VS Chronic HF (Females slides)

Acute heart failure فشل القلب الحاد	Chronic heart failure فشل القلب المزمن

• Acute left heart failure presents with a sudden onset of dyspnoea at rest that rapidly progresses to **acute respiratory distress**, orthopnoea and prostration.
• Often there is a clear precipitating factor (e.g. large MI, aortic valve dysfunction, myocarditis, and cardiogenic shock) which may be apparent from the history.
• Patients receive IV diuretics
• **SIgns & Symptoms:**
  ○ Rales
  ○ JVD
  ○ S3 gallop (Most specific)
  ○ Edema
  ○ Orthopnea
	• Patients with chronic heart failure commonly follow a **relapsing and remitting course**, with periods of stability and episodes of decompensation*, leading to worsening symptoms that may necessitate hospitalisation
	• **The clinical picture depends on:**
	  ○ The nature of the underlying heart disease
	  ○ The type of heart failure that it has evoked (e.g. Left/Right HF)
	  ○ The changes in the SNS and RAAS that have developed
	• Low cardiac output causes fatigue, listlessness and a poor effort tolerance; the **peripheries are cold** and the **BP is low**. To maintain perfusion of vital organs, blood flow is **diverted away from skeletal muscle** and this may contribute to fatigue and weakness. Poor renal perfusion leads to **oliguria** and **uraemia**.

★ ***What are the factors that may precipitate acute decompensation of chronic heart failure?**
Infection, ischemia and non compliant patients to dietary regimens ,**uncontrolled HTN**

Events usually leading to rapid deterioration
• Rapid arrhythmia or severe bradycardia/conduction disturbance
• Acute coronary syndrome
• Mechanical complication of acute coronary syndrome (e.g. rupture of interventricular septum, mitral valve chordal rupture, right ventricular infarction)
• Acute pulmonary embolism
• Hypertensive crisis
• Cardiac tamponade
• Aortic dissection
• Surgery and perioperative problems
• Peripartum cardiomyopathy

Events usually leading to less rapid deterioration
• Infection (including infective endocarditis)
• Exacerbation of COPD/asthma
• Anaemia
• Kidney dysfunction
• Non-adherence to diet/drug therapy
• Iatrogenic causes (e.g. prescription of an NSAID or corticosteroid; drug interactions) CCB, BB and antiarrhythmics
• Arrhythmias, bradycardia, and conduction disturbances not leading to sudden, severe change in heart rate
• Uncontrolled hypertension
• Hypothyroidism or hyperthyroidism
• Alcohol and drug abuse

Other factors:
- Dietary indiscretion (eating salty food)
- Iatrogenic volume overload (transfusion, fluid administration)
- Pregnancy
- Exposure to high altitude
- Worsening mitral or tricuspid regurgitation
- COVID-19

Click here for Boston criteria
(Present in females slides only)

◄ Modified Framingham criteria

Major	Minor
1) **PND** 2) **Orthopnea** 3) **Elevated JVP** 4) **Pulmonary rales** 5) **S3** 6) **Cardiomegaly on CXR** 7) **Weight loss ≥4.5kg in 5 days in response to treatment of presumed heart failure.**	1) **Bilateral leg edema** 2) **Nocturnal cough** 3) **Dyspnea on ordinary exertion** 4) **Hepatomegaly** 5) **Pleural effusion** 6) **Tachycardia (heart rate ≥120bpm)** 7) **Weight loss ≥4.5kg in 5 days**

Diagnosis

The diagnosis of HF requires that 2 major **OR** 1 major and 2 minor criteria cannot be attributed to another disease.

Diagnosis

◄ Differential diagnosis of HF signs and symptoms *

What are differential diagnosis of lower limb edema (fluid overload)?

1. Pericardial diseases
2. Liver diseases (common in pediatrics)
3. Nephrotic syndrome
4. Protein losing enteropathy (not very common)

◄ Principles of diagnosis of HF

- ➢ **Consider:** *Medical history, signs, symptoms*
- ➢ **Confirm:** *Natriuretic peptides, Echocardiography*
- ➢ **Assess clinical phenotype**: *HFrEF vs. HFpEF*
- ➢ **Assess etiology:** *Angiography, cMRI, Biopsy*
- ➢ **Risk stratification**
- ➢ **Workup for targeted therapies**

◄ Diagnosis of HFrEF vs HFpEF

- The diagnosis of HFpEF is more difficult than the diagnosis of HFrEF[1]

HFrEF	HFpEF
Symptoms typical of HF	Symptoms typical of HF
Signs typical of HF	Signs typical of HF
Reduced LVEF	Normal or only mildly reduced LVEF and LV not dilated
Measurement of Natriuretic peptide	Measurement of Natriuretic peptide
--	Relevant structural heart disease (LV hypertrophy/LA enlargement) and/or diastolic dysfunction

◄ When we examine CHF patient:

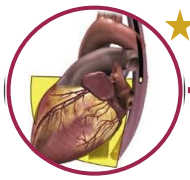
1- Vital signs: Heart rate, blood pressure, temperature and oxygen.
- blood pressure in HF patients can be high and low. High blood pressure is a positive cause of heart failure. Low blood pressure can occur in patient who have MI, cardiomyopathy and aortic stenosis.

2- JVP. If there's a high pressure in the ventricles it will backflow to the atria. In HF the pressure of the RA will be high which will result in raised JVP.
3-Rales 'crepitations" . It can be heard in the chest during inspiration and it is an additional sound. In normal conditions, the lung has no air therefore no sound will be heard. fluid+air = crepitation
4- Displaced apical membrane.
5- Abnormal 3rh heart sound

1- The diagnosis of HFpEF can be challenging, because symptoms are nonspecific and can be explained by several alternative non-cardiac conditions, such as chronic lung disease, anemia, and chronic kidney disease

Diagnosis cont'

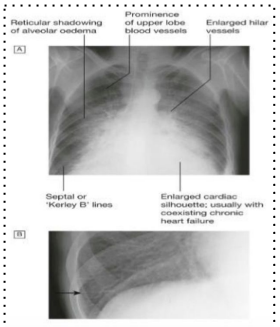
◀ Investigations for ALL patients



★Transthoracic echocardiography

- Echo is unquestionably the **most important** of all tests and should be performed whenever CHF is suspected based on history, examination, or CXR.
- Asses function of both ventricles and motion abnormality that may signify CAD
- ★ Useful in **determining whether systolic or diastolic dysfunction predominates**, and determines the cause of CHF e.g. pericardial, myocardial, valvular process or Intracardiac shunts.
- ★ **Estimates EF:** Patients with **systolic dysfunction (EF <45%)** should be distinguished from patients with **preserved left ventricular function (EF >45-50%).** (Those patients in the grey zone with an LVEF of 40–50% have recently been classified as having heart failure with mid- range ejection fraction (HFmrEF).)
- Assist in planning and monitoring of treatment and to obtain prognostic information.
- Identify patients who will benefit from long-term drug therapy, e.g. ACE inhibitors.
- TEE is more accurate in evaluating heart valve function and diameter. TTE is the best initial test for CHF

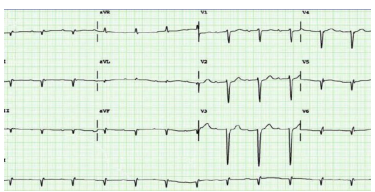
Chest X-ray (CXR) (For fluid overload)



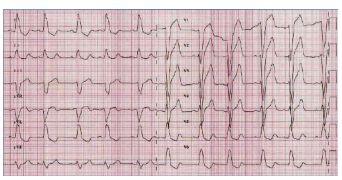
- **Has low sensitivity and specificity.**
- A CXR should be performed in all cases. It's used to detect/exclude certain types of lung disease e.g. cancer (does not exclude asthma/COPD). It's more useful in patient with suspected HF in acute setting
- To check the size and shape of heart (cardiomegaly in Left HF)
- *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 vessel.
- Pleural effusion
- Upper lobe diversion: indicative of HF
- Findings that are seen: (fluid overload, upper lobe redistribution, venous congestion, cardiomegaly)
- Stages of HF seen in X-ray (1- redistribution 2-interstitial edema 3-alveolar edema) **Dr said: someone might ask you about it**

Electrocardiogram (ECG)

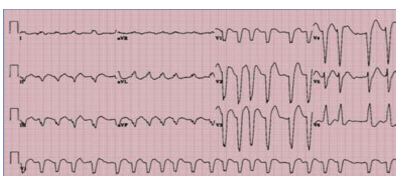
- **Has low sensitivity and specificity** , but can be useful for detecting chamber enlargement and presence of ischemic heart disease, prior MI, arrhythmia, LBBB (may help in management) and some forms of cardiomyopathy are tachycardia related.
- Recommended to determine rhythm, heart rate, QRS morphology, and QRS duration, and to detect other relevant abnormalities. The information also assist in planning of treatment and is of prognostic importance.
- A completely normal ECG makes systolic HF unlikely.



Regular = not AF
Prominent Q wave= old MI



Abnormal ECG showing:
Wide QRS= bundle branch block
Peaked T wave = ischemia or electrolyte imbalance
(the combination of abnormal T wave and wide QRS favors ischemia)



Abnormal ECG showing:
Tachycardia and Irregularity= AF
Wide QRS= left bundle branch block

Diagnosis cont'

◄ Investigations for ALL patients cont'

Optimal NT-proBNP Cut-points

"Rule in"

Age strata	Optimal cut-point	Sensitivity	Specificity	PPV	NPV	Accuracy
All <50 years (n=183)	450 pg/ml	97%	93%	76%	99%	96%
All 50-75 years (n=554)	900 pg/ml	90%	82%	82%	86%	86%
All >75 years (n=519)	1800 pg/ml	85%	73%	92%	55%	83%
Overall average		92%	84%	88%	66%	93%

"Rule out"

	Optimal cut-point	Sensitivity	Specificity	PPV	NPV	Accuracy
Rule out	300 pg/ml	99%	62%	55%	99%	83%

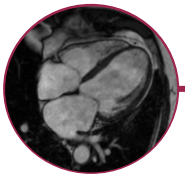
Blood tests

★ **Measurement of Natriuretic peptide** (Natriuretic peptides can be used as **diagnostic** , **prognostic** and follow up .BNP levels are not specific for HF as they can be increased in any condition where atrial pressure is elevated.) **(BNP, NT-proBNP or MR-proANP):**

- BNP >100pg/ml or NT-proBNP >300pg/ml is suggestive of heart failure. (Normal is <100pg/mL). BNP is ordered when the etiology of acute dyspnea is not clear and you cannot wait for echo to be done.
- Normal or reduced BNP level will make HF less likely **(VERY HIGH SENSITIVITY, less specificity)**

- **Liver biochemistry** (may be altered do to hepatic congestion).
- **Electrolytes imbalance** (including Na, K+, Ca and Mg)→ to detect chronic renal insufficiency or hypocalcemia
- **CBC** to look for anemia (causes high output HF) which may exacerbate HF or be an alternative cause of the patient S&S.
- **Blood glucose**, HbA1C. (For diabetes)
- **Lipids, Creatinine** and check **serum ferritin/TIBC level** (to detect hemochromatosis or iron deficiency)
- **Urea** and electrolytes (as a baseline before starting diuretics and ACE inhibitors)
- **Thyroid function tests** to detect hyperthyroidism (in the elderly and those with atrial fibrillation).
- Pre-renal azotemia
- Hemochromatosis Iron overload (mainly seen in heart and kidney)

◄ Investigations for SELECTED patients



Cardiovascular magnetic resonance (CMR) imaging (AKA cardiac MRI)

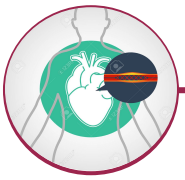
- CMR imaging is recommended to evaluate cardiac structure and function, to measure LVEF, and to characterize cardiac tissue, especially in subjects with inadequate echocardiographic images or where the echocardiographic findings are inconclusive or incomplete (but taking account of cautions/contraindications to CMR)

Myocardial perfusion/ischemia imaging

- Myocardial perfusion/ischemia imaging (echocardiography, CMR, SPECT or PET) should be considered in patients though to have CAD, and who are considered suitable for coronary revascularization, to determine whether there is reversible myocardial ischemia and viable myocardium

Diagnosis cont'

◀ Investigations for SELECTED patients



Cardiac Catheterization قسطرة القلب

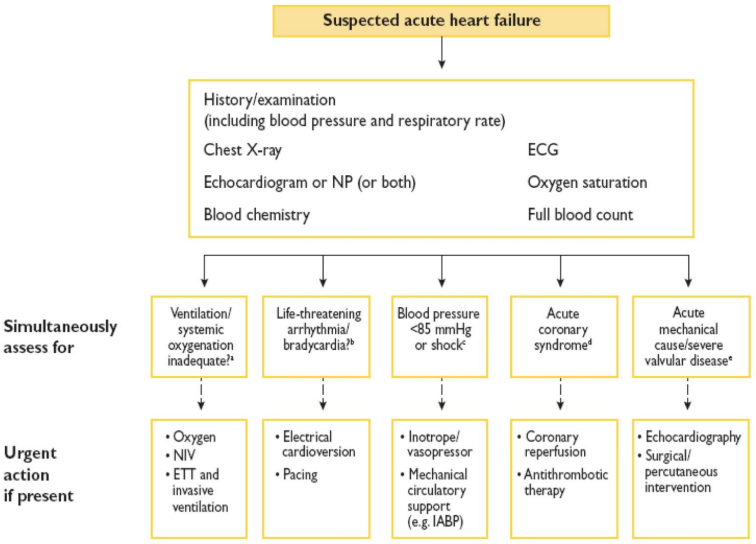
- To evaluate right and left heart function and pulmonary arterial resistance
- Can clarify the cause of CHF if noninvasive test results are equivocal.
- Used when CAD or VHD are suspected
- Recommended in patients being evaluated for heart transplant or mechanical circulatory support.
- Gives precise valve diameter, and detects any septal defects

Exercise testing

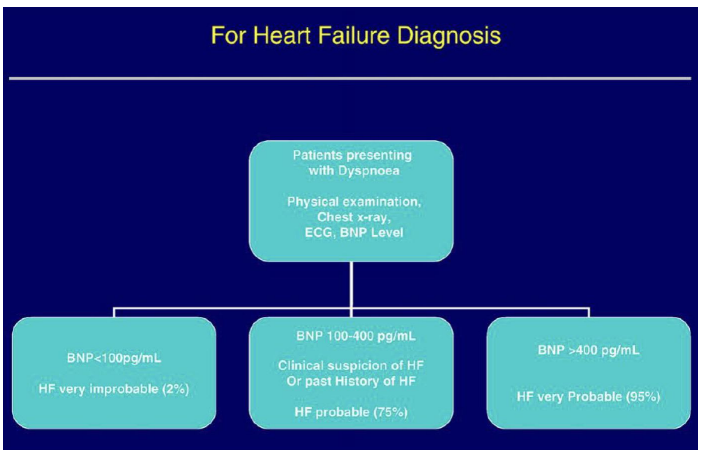
- **Exercise testing should be considered:**
  - To detect reversible myocardial ischemia
  - As part of the evaluation of patients for heart transplantation and mechanical circulatory support
  - To aid in the prescription of exercise training
  - To obtain prognostic information.

Other tests: Metanephrines, endomyocardial biopsy (if infiltrative disease (e.g. sarcoid, amyloid) is considered)

Summary of diagnosis (From Dr slides)



Click here for an algorithm from Kumar



Classification

◀ ACC/AHA Classification

(based on structure and damage to heart)

ACC: American College of Cardiology
AHA: American Heart Association

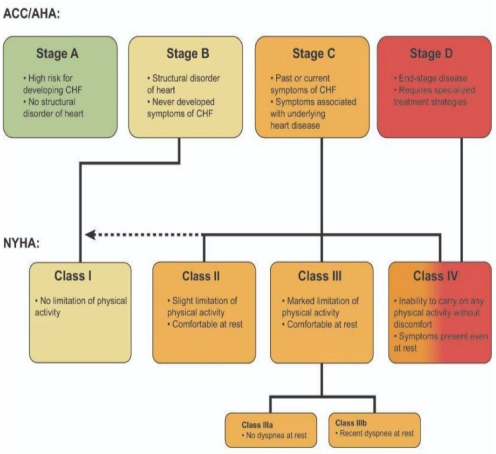
	At risk of HF		HF	
Stage	**Stage A**	**Stage B**	**Stage C**	**Stage D**
Description	**At high risk** for HF but without structural heart disease or symptoms of HF	**Structural heart disease** but without signs or symptoms of HF	**Structural heart disease with** prior or current **symptoms** of HF	**Refractory HF** requiring specialized interventions
Who?	**E.g. Patients with:** - Hypertension - Atherosclerosis - DM - Obesity - Metabolic syndrome **Or patients** - Using cardiotoxins - With family history of CM	**E.g. Patients with:** - Previous MI - LV remodeling including LVH and low EF - Asymptomatic vascular disease	**E.g. Patients with:** - Known structural heart disease and SOB, fatigue and reduced exercise tolerance	**E.g. Patients** who have marked symptoms **at rest** despite maximal medical therapy (e.g. those who are recurrently hospitalized or cannot be safely discharged from the hospital without specialized interventions)
Therapy	**Goals:** - Treat hypertension - Encourage smoking cessation - Treat lipid disorders - Encourage regular exercise - Discourage alcohol intake, illicit drug use - Control metabolic syndrome **Drugs:** - ACEI or ARB	**Goals:** - All measures under stage A **Drugs:** - ACEI or ARB - BB **Devices in selected patients:** - Implantable defibrillators	**Goals:** - All measures under stage A and B - Dietary restriction **Drugs for routine use:** - Diuretics - ACEI - BB **Drugs in selected patients:** - Aldosterone antagonist - ARBs - Digitalis - Hydralazine/nitrates **Devices in selected patients:** - Biventricular pacing - Implantable defibrillators	**Goals:** - Appropriate measures under stages A, B, C - Decisician re: appropriate level of care **Options:** - Compassionate end-of-life care/hospice - Extraordinary measures e.g. heart transplant, chronic inotropes, permanent mechanical support or experimental surgery or drugs

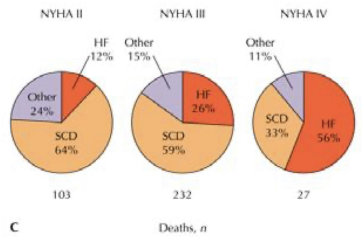
Classification cont'

◀ New York Heart Association (NYHA) Classification

(based on symptoms or physical activity) **Used to assess severity**

Class I	No limitations of activities. Symptoms only **occur with vigorous activities**, such as playing a sport. Patients are nearly asymptomatic.
Class II	Slight or mild limitation of activity. Symptoms **occur with prolonged or moderate exertion**, such as climbing a flight of stairs or carrying heavy packages. Slight limitation of activities.
Class III	Marker limitation if activity.Symptoms **occur with usual activities of daily living**, such as walking across the room or getting dressed. Comfortable at rest.
Class IV	Symptoms **occur at rest**. Incapacitating.





◀ Forrester Classification[1]

Not used clinically anymore

Figure 1. Hemodynamic/Clinical State in Acute Heart Failure

Other signs:
- Narrow pulse pressure
- Pulsus alterations
- Declining serum Na level
- ACEI-related symptomatic hypotension

SIGNS OF LOW PERFUSION
Cool extremities
Low urine output
Altered mental status
Inadequate response to IV diuretic
Prerenal azotemia

Low perfusion at rest?	Congestion at rest? No	Congestion at rest? Yes
No	A *Dry and Warm* Class I	B *Wet and Warm* Class II
Yes	L *Dry and Cold* Class III	C *Wet and Cold* Class IV

SIGNS OF CONGESTION
↑ JVD
+ HJR
And valsalva square wave
And ascites
Peripheral edema
And Loud P2
S_3
DOE/SOA
Orthopnea/PND
Rales
Recent weight gain

↑: 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: No congestion
Wet: Congestion
Warm: No decrease in perfusion
Cold: Decrease in perfusion

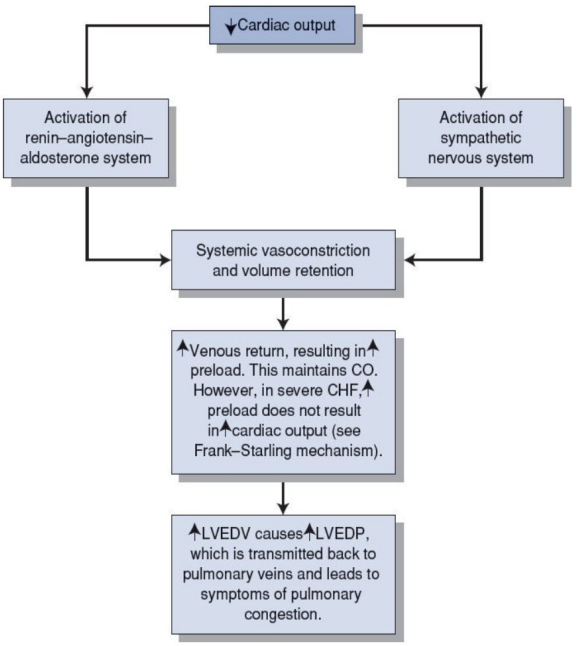
1- first, we look at the congestion state which is the fluid status (is he hypovolemic or overloaded?) and the BP (which is the perfusion status) if he's not congested with normal BP we call him warm & dry, and if he's the opposite then he's Cold & wet (cardiogenic state) . when he's warm & wet when he's perfusing well he's usually hypertensive. If they're not having enough oral intake they're Dry & cold

Summary

Heart failure		
Classification	**Systolic dysfunction (HFrEF)**	**Diastolic dysfunction (HFpEF)**
	• Impaired contractility, EF is reduced. • Causes: IHD, HTN, VHD etc	• Impaired ventricular filling, EF is preserved. • Causes: HTN leading to myocardial hypertrophy.
	High Output HF	**Low Output HF**
	Conditions that increase demand on CO, causing a clinical picture of heart failure due to an excessively high CO e.g. Severe anemia, thyrotoxicosis, pregnancy, A/V fistula, Beriberi and Paget's disease	Cardiac output is inadequate to perfuse the body (i.e. EF <40%), or can only be adequate with high filling pressures.
	Acute HF	**Chronic HF**
	• Acute left heart failure presents with a sudden onset of dyspnoea at rest that rapidly progresses to **acute respiratory distress**, orthopnoea and prostration. • Often there is a clear precipitating factor (e.g. large MI, aortic valve dysfunction, myocarditis, and cardiogenic shock) which may be apparent from the history.	• Patients with chronic heart failure commonly follow a **relapsing and remitting course**, with periods of stability and episodes of decompensation*, leading to worsening symptoms that may necessitate hospitalisation
	Left sided HF	**Right sided HF**
	• **Reduction in left ventricular output** and an **increase in left atrial and pulmonary venous pressure. T**his increases pulmonary vascular resistance and causes pulmonary hypertension, which in turn impairs right ventricular function. • **Hallmark: Increased LVEDP** • **Symptoms:** Dyspnea, Orthopnea, PND • **Signs:** Displaced PMI, Cardiomegaly, S3, S4 and crackles at lung bases.	• **Reduction in right ventricular output** and an **increase in right atrial and systemic venous pressure.** • The most common cause of right Hf is left HF other causes include: Pulmonary HTN and chronic lung disease **(cor pulmonale)** • **Symptoms:** Peripheral edema, Nocturia, Abdominal symptoms • **Signs:** JVD, Hepatomegaly and Ascites

EXTRA

Helpful figures



Box 30.28 Clinical syndromes of acute heart failure (AHF)

Type	Clinical features
Acute decompensated heart failure	Mild features of heart failure, e.g. dyspnoea
Hypertensive AHF	High blood pressure, preserved left ventricular function, pulmonary oedema on chest X-ray
Acute pulmonary oedema	Tachypnoea, orthopnoea, pulmonary crackles, oxygen saturation <90% on air, pulmonary oedema on chest X-ray
Cardiogenic shock	Systolic blood pressure <90 mmHg, mean arterial pressure drop >30 mmHg, urine output <0.5 mL/kg per hour, heart rate >60 b.p.m.
High-output heart failure	Warm peripheries, pulmonary congestion, blood pressure may be low, e.g. septic shock
Right heart failure	Low cardiac output, elevated jugular venous pressure, hepatomegaly, hypotension

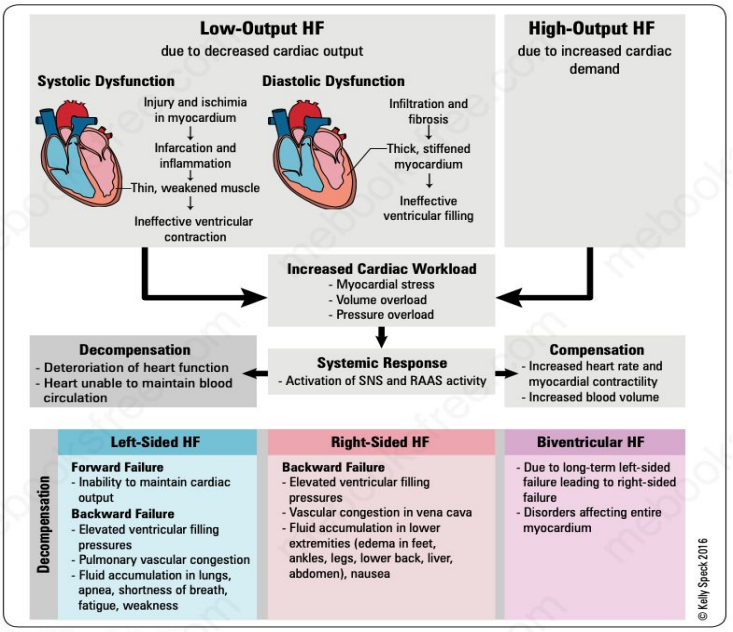


Figure 39. Congestive Heart Failure

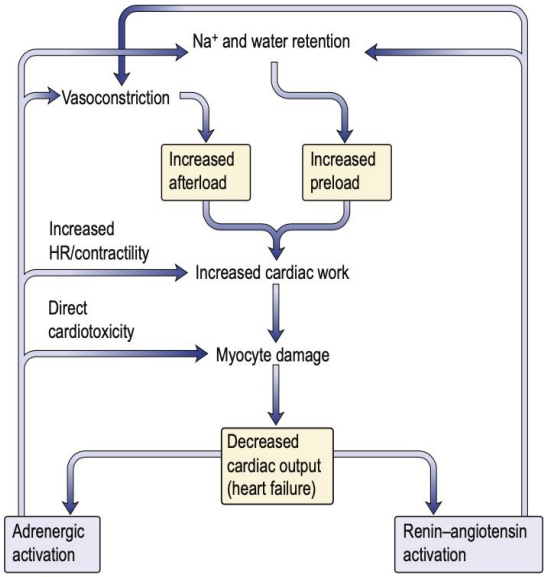


Fig. 30.54 The compensatory physiological response to heart failure. Chronic activation of the renin–angiotensin and adrenergic systems results in a 'vicious circle' of cardiac deterioration that further exacerbates the physiological response. HR, heart rate.

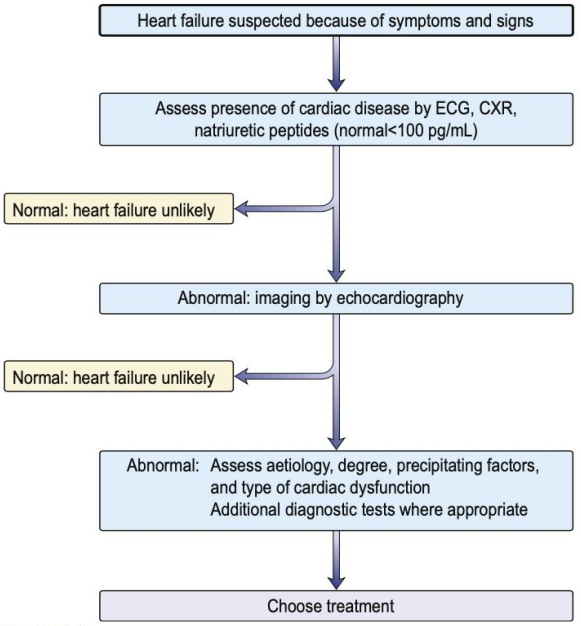


Fig. 30.56 Algorithm for the diagnosis of heart failure. *(Based on the European Society of Cardiology and NICE guidelines.)*

Lecture Quiz

Q1: 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

Q2: 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

Q3: A 71-year-old man is being treated for congestive heart failure with a combination of drugs. He complains of nausea and anorexia, and has been puzzled by observing yellow rings around lights. His pulse rate is 53/minute and irregular and blood pressure is 128/61mmHg. Which of the following medications is likely to be responsible for these symptoms?
A- Lisinopril
B- Spironolactone
C- Digoxin
D- Furosemide

Q4: A 71-year-old woman presents to ambulatory clinic with a chief complaint of dyspnea upon exertion. Over the past few weeks, she has had a chronic cough and shortness of breath when walking more than two city blocks. She has a long history of hypertension that has been poorly controlled in recent years. On physical examination, she has an elevated jugular venous pulse and rales are evident on lung examination. Cardiac enzymes are negative. Which modality is the most appropriate next step in distinguishing systolic from diastolic heart failure?
A- Cardiac catheterization
B- Clinical judgment based on physical examination
C- CT scan of the chest
D- Echocardiography

Q5: A 65-year-old woman with chronic systolic heart failure (left ventricular ejection fraction, 30%) comes for a routine clinic visit. She reports that she is dyspneic climbing one light of stairs and uses two pillows to sleep at night. She has intermittent lower extremity edema, especially after eating a salty meal. Her medications include lisinopril 20 mg daily, carvedilol 25 mg twice daily, spironolactone 25 mg daily, and torsemide 40 mg daily. On examination, she has a heart rate of 70 beats per minute, blood pressure of 110/70 mm Hg, no jugular venous dis- tention, normal heart sounds, a II/VI holosystolic murmur at the apex, and trace-1+ peripheral edema. Her laboratory values are notable for sodium 140 mEq/L, potassium 4.8 mEq/L, blood urea nitrogen 20 mg/dL, and creatinine 1.2 mg/dL. What is the next most appropriate step in her management?
A- Continue her current medications.
B- Increase lisinopril to 30 mg daily.
C- Stop lisinopril and start sacubitril/valsartan 49/51 mg twice daily after 36-hour washout.
D- Increase torsemide to 60 mg daily.

Q6: A 74-year-old man with hypertension, coronary artery disease, GERD, and osteoarthritis presents for follow-up. He had an ST segment myocardial infarction 2 years prior and underwent successful stenting of a complete LAD arterial occlusion. For the past 3 weeks, he has noted worsening dyspnea on light exertion coupled with lower extremity swelling. He has had no recurrent chest pain. His medications include metoprolol, nifedipine, aspirin, and rosuvastatin. On examination, his blood pressure is 126/80 mm Hg. His heart rate is 70 beats per minute. His jugular venous pressure is 14 cm H2O. The first and second heart sounds are normal, and a third heart sound is appreciated. here is lower extremity edema to the knee bilaterally. A stress echocardiogram reveals mild anterior wall hypokinesis at rest, and all walls augment appropriately with stress. he left ventricular ejection fraction at rest is estimated at 40%. In addition to diuresis and discontinuation of nifedipine, what is the most appropriate management?
A- Add hydralazine and isosorbide mononitrate.
B- Add clopidogrel.
C- Add lisinopril.
D- Add spironolactone..

Answers: Q1:D | Q2:A | Q3:C | Q4:D | Q5:C | Q6:C

Answers Explanation File!

GOOD LUCK !

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