Lecture 5,6

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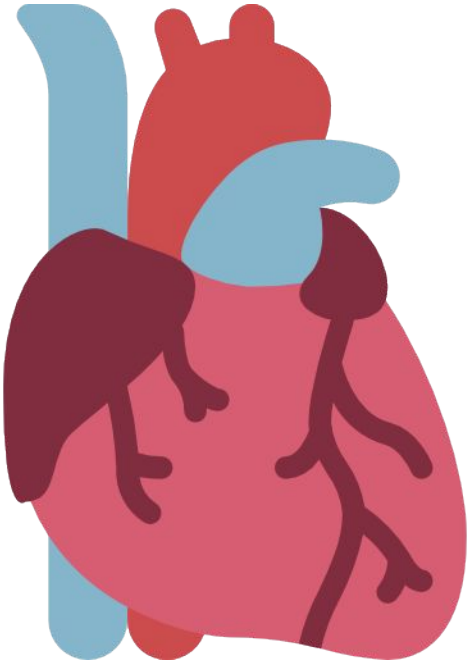

Medicine Team

Reviewed By

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Heart Failure I & II

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.

Color index:

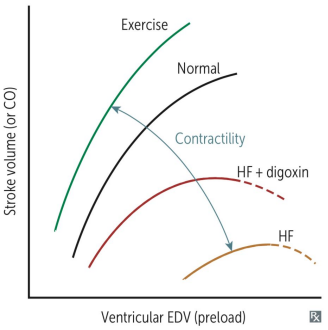
Original text Females slides Males slides
Doctor's notes Text book Important Golden notes Extra

◀ 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**

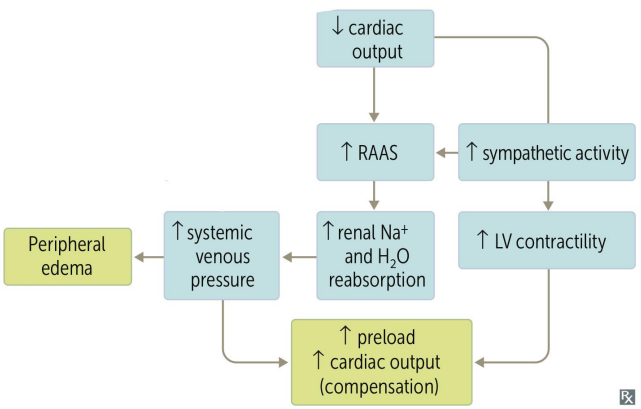
SV increases with ↑Contractility, ↑preload and ↓afterload

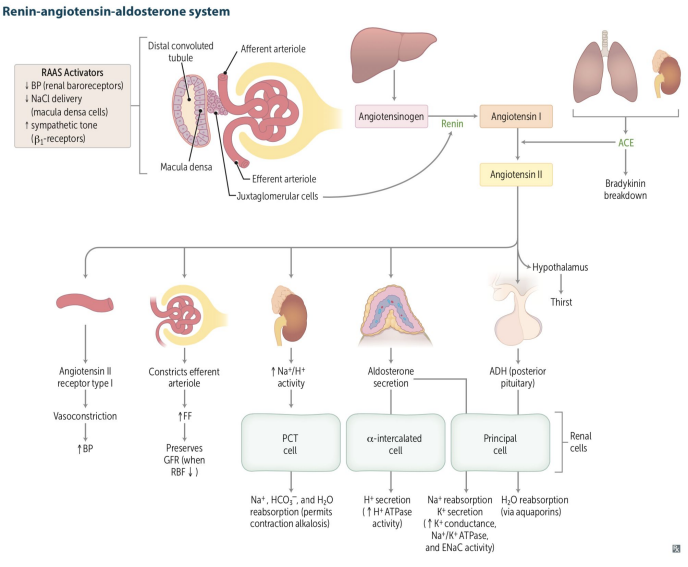
- **What is Starling curve?**
  - Starling's law states that the stroke volume is directly proportional to the diastolic filling (i.e. the preload or ventricular end-diastolic pressure). As the preload is increased, the stroke volume rises (normal). Increasing contractility (e.g. increased with sympathetic 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** 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).





Introduction to HF

◀ What's heart failure?

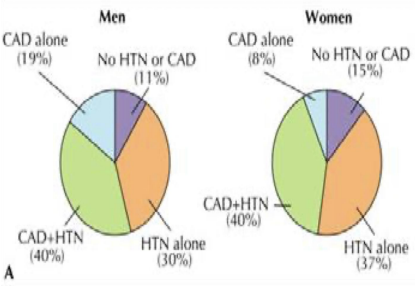
- Heart failure is a complex (clinical) syndrome that can result from any **structural** or **functional** cardiac disorder that **impairs the ability of the ventricle to fill** (e.g. LVH) **and/or eject blood to meet the body demand.**
- HF is characterized by signs and symptoms of intravascular and interstitial volume overload and/or manifestations of inadequate tissue perfusion.
- **Typical presentation of HF:**
  1. Syndrome of decreased exercise tolerance
  2. Syndrome of fluid retention
  3. No symptoms but incidental discovery of LV dysfunction
- 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 - 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

◀ Etiology

- **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?**[1]

1. **Most common:** Coronary artery disease (IHD)
2. **2nd:** Hypertension
3. ★ **3rd:** VHD (e.g. AS)
4. **4th:** Dilated cardiomyopathy



Causes of left ventricular failure

Volume overload	Pressure overload	Loss of muscles	Restricted filling
1. Regurgitate valve 2. High output status	1. Systemic hypertension 2. Outflow obstruction	1. Post MI 2. Chronic ischemia 3. Connective tissue diseases 4. Poisons (alcohol, cobalt, Doxorubicin)	1. Pericardial disease 2. Restrictive cardiomyopathy 3. Tachyarrhythmia

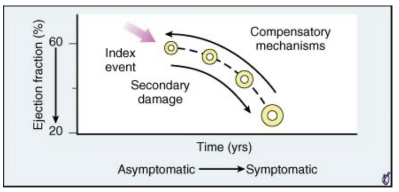
1- can be dependant on the region. In south america, chagas disease is one of the most common causes. And alcohol or toxic cardiomyopathy is common in western regions and hypertension is the most common cause in Africa.

Introduction cont.

◀ Background of HF pathophysiology

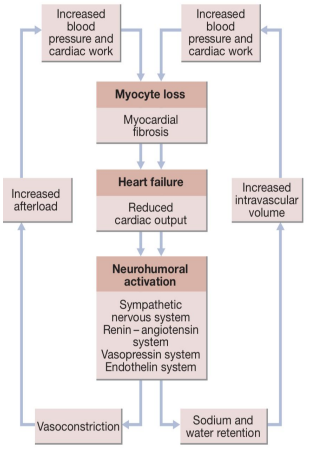
Heart failure pathophysiology:

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



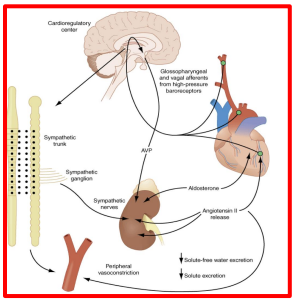
◀ Changes in HF

1 Hemodynamic changes:



Ventricular dysfunction: (Heart failure syndrome)

- The most common cause of heart failure. This can occur because of impaired **systolic** contraction, or **diastolic** dysfunction or both. 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:

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[1] ⟶ ↑ 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.

1- 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.

Systolic and Diastolic HF

◀ Systolic VS Diastolic Dysfunction

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

Systolic dysfunction (HFrEF)[1]	★ Diastolic dysfunction (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.
  ○ HTN resulting in cardiomyopathy
  ○ Valvular heart disease
  ○ Myocarditis (postviral)
  ○ Less common causes: Alcohol abuse, radiation, hemochromatosis, thyroid disease
	• 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.

Note: Usually both systolic and diastolic dysfunctions present simultaneously

◀ High VS Low output HF

High Output HF	Low Output HF
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.

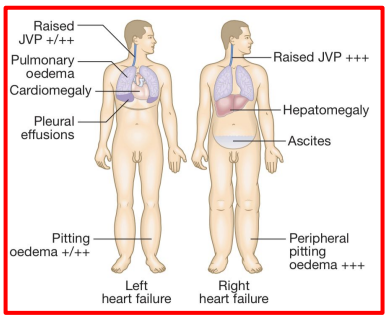
◀ What is decapitated blood pressure?

- 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.

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.

Right and Left HF

◀ Left VS Right sided HF

	Left heart failure	Right heart failure
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:** Difficulty breathing secondary to pulmonary congestion/edema • **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 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, right hypochondrial pain. 
Signs[1]	• **Displaced** and sustained **PMI** (usually to the left) due to **cardiomegaly** • **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) ★ **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

Acute and Chronic HF

◀ Modified Framingham criteria

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

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.

◀ Acute VS Chronic HF

Acute heart failure	Chronic heart failure
• 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. • **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?

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

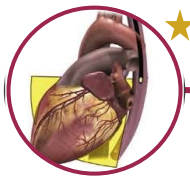
Events usually leading to less rapid deterioration
• Infection (including infective endocarditis)
• Exacerbation of COPD/asthma
• Anaemia
• Kidney dysfunction
• Non-adherence to diet/drug therapy
• Iatrogenic causes (e.g. prescription of an NSAID or corticosteroid; drug interactions) CCB, BB and antiarrhythmics
• 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

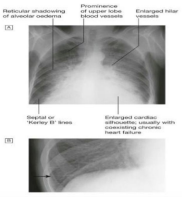
Diagnosis

◀ Investigations for ALL patients



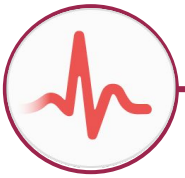
★Transthoracic echocardiography

- 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.



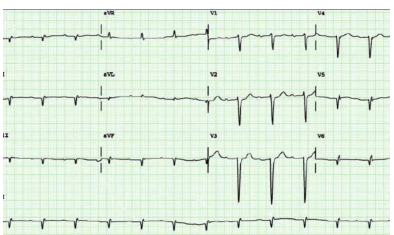
Chest X-ray (CXR)

- **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

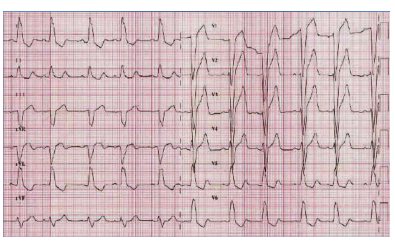


Electrocardiogram (ECG)

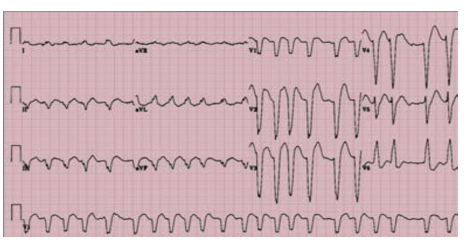
- **Has low sensitivity and specificity** , but can be useful for detecting chamber enlargement and presence of ischemic heart disease, prior MI, arrhythmia, LBBB (may help in management) and some forms of cardiomyopathy are tachycardia related.
- Recommended to determine rhythm, heart rate, QRS morphology, 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



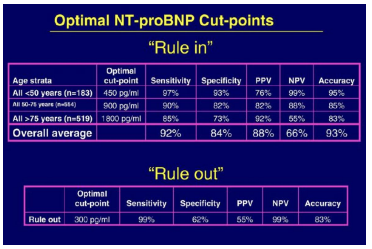
Abnormal ECG showing:
Wide QRS= bundle branch block
Peaked T wave = ischemia or electrolyte imbalance
(the combination of abnormal T wave and wide QRS favors ischemia)



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

Diagnosis cont'

◄ Investigations for ALL patients cont'

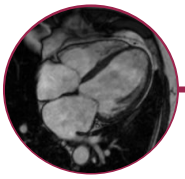


Blood tests

★ **Measurement of Natriuretic peptide**[1] **(BNP, NT-proBNP or MR-proANP):**
- BNP >100pg/ml or NT-proBNP >300pg/ml is suggestive of heart failure. (Normal is <100pg/mL)
- Normal BNP level will make HF less likely

- **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 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).

◄ 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)

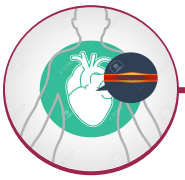
Myocardial perfusion/ischemia imaging

- 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

1- Natriuretic peptides can be used both as **diagnostic** and **prognostic** factors in HF, but they are not specific for HF as they can be increased in any condition where atrial pressure is elevated.

Diagnosis cont'

◀ Investigations for SELECTED patients



Cardiac Catheterization

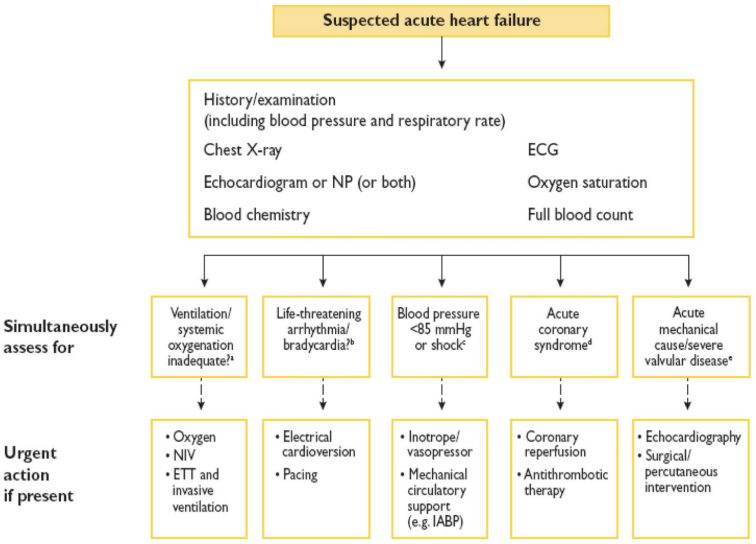
- To evaluate right and left heart function and pulmonary arterial resistance
- Can clarify the cause of CHF if noninvasive test results are equivocal.
- Used when CAD or VHD are suspected
- Recommended in patients being evaluated for heart transplant or mechanical circulatory support.
- Gives precise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

Exercise testing

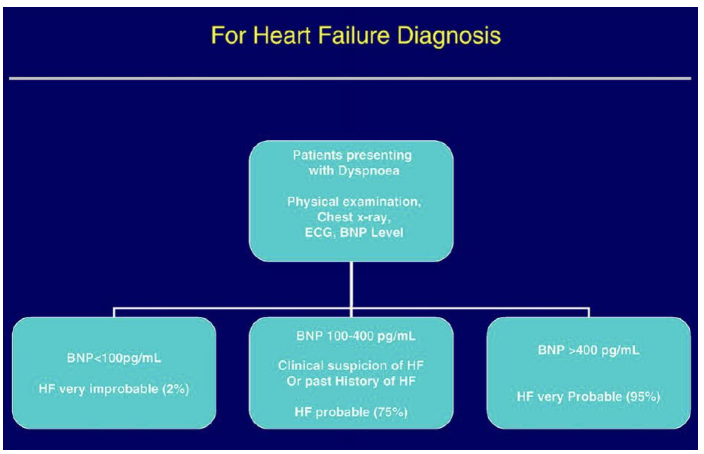
- **Exercise testing should be considered:**
  - To detect reversible myocardial ischemia
  - As part of the evaluation of patients for heart transplantation and mechanical circulatory support
  - To aid in the prescription of exercise training
  - To obtain prognostic information.

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

ACC: American College of Cardiology
AHA: American Heart Association

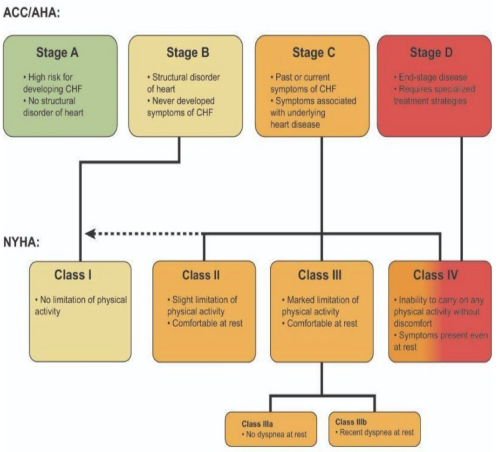
	At risk of HF		HF	
Stage	**Stage A**	**Stage B**	**Stage C**	**Stage D**
Description	**At high risk** for HF but without structural heart disease or symptoms of HF	**Structural heart disease** but without signs or symptoms of HF	**Structural heart disease with** prior or current **symptoms** of HF	**Refractory 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

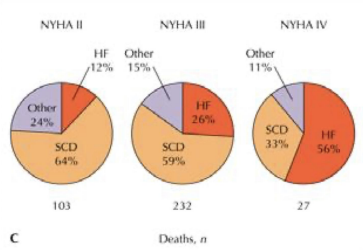
Classification cont'

New York Heart Association (NYHA) Classification

- 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]

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

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

↑: 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.

Differential diagnosis of HF signs and symptoms

1. Pericardial diseases
2. Nephrotic syndrome
3. Liver diseases
4. Protein losing enteropathy

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

Management

◀ Introduction to HF management

- First correct the reversible causes e.g. Ischemia, VHD, thyrotoxicosis, anemia and other high output status, shunts, Arrhythmias (e.g. Afib, atrial flutter, CHB), medications (e.g. CCB, antiarrhythmics, NSAIDS)

Management tools

Lifestyle modification

- Diet[1]
- Fluid intake
- Exercise shows mortality benefit
- Smoke cessation
- Weight reduction

Pharmacological

- Oral medication
- Immunization (flu, pneumococcal and covid-19 (hopefully)

Surgical

- Device therapy
- Assisting devices
- Transplant

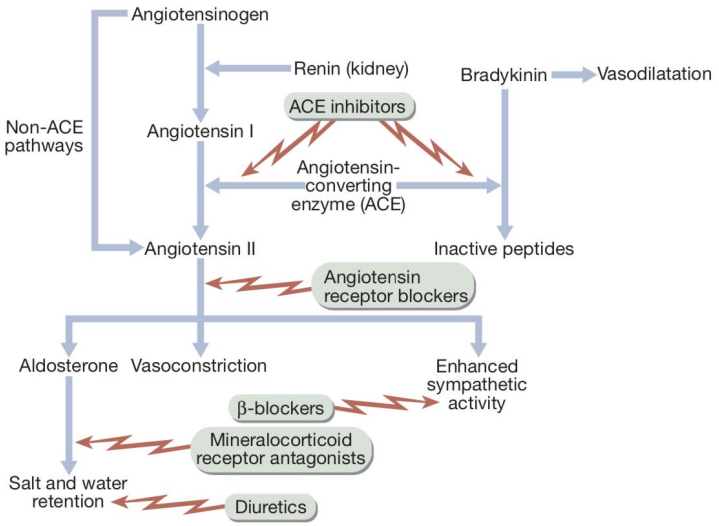
◀ Management of Chronic HF

General measures

- **Education** of patients and families.
- **Physical activity:** reduce during exacerbations to reduce work of the heart. Encourage low-level (e.g. 20- to 30-min walks 3–5 times weekly) with compensated heart failure
- **Diet and social:** weight reduction if necessary (Daily weight "Tailor therapy"), no added salt diet (2g of Na=5g of NaCl), avoid alcohol (negative inotropic effects), stop smoking and fluid restriction (1.5-2L/day, about 8 cups)
- **Vaccine** against pneumococcal disease influenza.
- **Correct aggravating factors**, e.g. arrhythmias, anaemia, hypertension and pulmonary infections
- **Driving:** unrestricted, except symptomatic heart failure disqualifies driving large lorries and buses
- **Sexual activity:** tell patients on **nitrates** not to take phosphodiesterase type 5 inhibitors.

Pharmacological treatment

1. **ACEI/ARB**
2. **β-Blockers**
3. **Diuretic**
4. **Digoxin**
4. **Vasodilators**
5. **SGLT-2 inhibitors**



1- total salt intake should be 2 grams per day. We don't restrict the patient completely from salt. To describe ot to the patient, tell him "don't add any EXTRA salt, don't eat canned food and try to avoid cheese (any kind). And to stay away from restaurant. For fluid they should.t exceed fluids above 1.5 liters

Management cont'

◀ Management of Chronic HF cont.

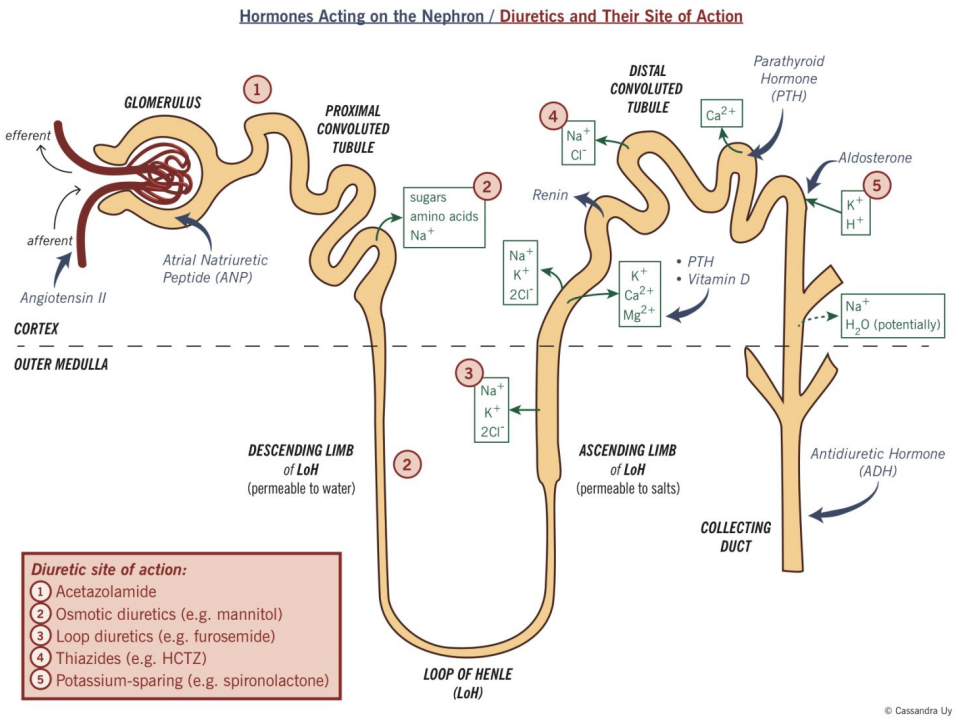
Diuretics		
Group	**Loop diuretics** **Furosemide "Lasix"** (20-320 mg QD) **Bumetanide "Bumex"** (1-8mg) **Torsemide** (20-200mg)	**Thiazide diuretic** **(Hydrochlorothiazides, Chlorthalidone, Metolazone)**
General	• **Most effective in controlling symptoms** (Dyspnea & Peripheral edema), but have **not been shown to reduce mortality** or improve prognosis.	
M.O.A	• Inhibit chloride reabsorption in ascending limb of loop of Henle results in natriuresis, kaliuresis and metabolic alkalosis	• Block Na reabsorption in loop of henle and distal convoluted tubules
Uses	• **Most potent diuretics** used in moderate/severe HF.	• Used for mild HF • Ineffective with GFR < 30 --/min.
ADR	• Pre-renal azotemia[1] • Hypokalemia[2] • Skin rash • Ototoxicity	• Pre-renal azotemia[1] • Skin rashes • Neutropenia • Thrombocytopenia • Hyperglycemia • ↑ Uric Acid • Hepatic dysfunction

Diuretics benefits:

- Improves symptoms of congestion
- Can improve cardiac output
- Improved neurohormonal milieu
- No inherit nephrotoxicity

Diuretics limitations:

- Oral absorption unpredictable
- Excessive volume Depletion
- Electrolyte disturbance



1- in case of Chronic kidney disease they're ineffective
2- Monitor renal function and check for hypokalaemia and hypomagnesaemia

Management cont'

◀ Management of Chronic HF cont.

K+ sparing agents			
Group	**Spironolactone (Aldosterone inhibitor)**	**Eplerenone**	**Triamterene, amiloride**
General	• Relatively weak diuretics with a potassium-sparing action		
M.O.A	• Acts at the collecting duct by competitive inhibition of cytoplasmic aldosterone receptors →↑ Excretion of Na+,Cl & ↓Excretion of K+,H+,NH4	• Binds to the mineralocorticoid receptor and blocks the binding of aldosterone	• Acts on distal tubules to ↓ K secretion
Uses	• Spironolactone (25 mg daily) in combination with conventional treatment **improves survival** in patients with moderate/severe heart failure (Class III & IV), due to its effect on RAAS with subsequent effect on myocardial remodeling and fibrosis.	• **Reduces mortality** in patients with acute myocardial infarction and heart failure.	-
Adverse effects	• **Gynecomastia** (If pt develop this, switch to eplerenone), Hyperkalemia and chest pain. (Monitor renal function)	Hyperkalemia	-

β-Blockers			
Drug name	**Bisoprolol**	**Carvedilol**	**Metoprolol**
Uses	• Has been traditionally contraindicated in pts with CHF • Now they are the main stay in treatment on CHF & may be the only medication that shows substantial improvement in LV function ★ In addition to improved LV function multiple studies **show improved survival**[1] • **"Start low, go slow"**: Bisoprolol 1.25mg od, Carvedilol 3.124mg od • Not rescue therapy. • Consider cardioselective agents in mild to moderate reversible airways disease. • The benefit of beta blockers likely stems from: ○ Anti-Ischemic effect , Decreasing HR (Decrease O2 consumption) Antiarrhythmic effect		
Contraindications	• Severe decompensated CHF (AHF, Pulmonary edema) • Peripheral Vascular Disease (PVD)		

BB trials:

- US Carvedilol studies 1996
  - 65% decrease mortality in carvedilol group
  - 27% reduction in hospitalisations, reduction in progression of CCF
- CIBIS-II – Bisoprolol vs. placebo
  - 34% reduction mortality (42% reduction in sudden death and 32% hospitalisations)
- MERIT-HF - metoprolol
- COPERNICUS
  - NYHA class IV, EF < 25%
  - 35% reduction in mortality with carvedilol
- CAPRICORN - 23% reduction in mortality post MI

1- Beta-blockers are more effective at reducing mortality than ACE inhibitors, with a relative risk reduction of 33% versus 20%, respectively.

Management cont'

◄ Management of Chronic HF cont.

Angiotensin-converting enzyme inhibitors (ACEI)			
Drug name	**Perindopril**	**Lisinopril**	**Quinapril**
M.O.A	• Block the R-A-A system by inhibiting the conversion of angiotensin I to angiotensin II → vasodilation and ↓ Na and water retention.(decrease Afterload and preload) • ↓ Bradykinin degradation ↑ its level → ↑ PG secretion & nitric oxide → Vasodilation • They also ultimately inhibit cardiac remodelling		
Uses	★ They improve symptoms, limit the development of progressive heart failure and **prolong survival**, and **should be given to all patients with heart failure.** • ACE Inhibitors were found to improve survival in CHF patients: ○ Delay onset & progression of HF in pts with asymptomatic LV dysfunction ○ ↓ cardiac remodeling		
Side effects	• Angioedema • Renal insufficiency, Hepatic dysfunction • Rash, Hyperkalemia[1] ★ Cough (If patient develops cough switch to ARBs) • **The major side-effect is first-dose hypotension.** ○ ACEI treatment should be introduced gradually with a **low initial dose and gradual titration** every 2 days to full dose with regular blood pressure monitoring and a check on serum potassium and renal function 1-2 weeks after starting therapy; **creatinine levels normally rise by about 10–15% during ACEI therapy.** • If patient developed hyperkalemia or renal impairment or is pregnant→ **switch from ACEI to Hydralazine with isosorbide dinitrate.**		
Contraindications	• Renal failure • Bilateral renal artery stenosis (RAS) • Aortic stenosis		

Trials for ACEI:
- CONSENSUS 1987 – enalapril vs. placebo – 31% reduction mortality in enalapril group. Confirmed by SOLVD, AIRE, SAVE, TRACE.
- 1995 meta-analysis showed 23% reduction total mortality, 35% in combined mortality/hosp admission

Angiotensin II type 1 receptor blockers (ARBs)			
Drug name	**losartan**	**irbesartan**	**valsartan**
Uses	• **Second-line therapy** in certain conditions when ACE I are contraindicated **(angioneurotic edema, cough)** • Has comparable effect to ACEI. Has mortality benefit.		
ADRs	• Renal dysfunction, hyperkalemia		

1- can be beneficial in offsetting the hypokalemia associated with loop diuretic therapy.

Management cont'

◀ Management of Chronic HF cont.

Angiotensin Receptor - Neprilysin inhibitors (ARNi)	
Drug name	**Combination of Valsartan & Sacubitril**

General info	• Recent FDA approval (2015)
	• The only product available (valsartan/sacubitril)
	  ○ Valsartan = ARB
	  ○ Sacubitril = prodrug for sacubitrilat Inhibit neprilysin which breakdown the vasoactive peptides.

M.O.A	• Inhibits neprilysin which is responsible for the **breakdown of** the endogenous diuretics **ANP** and **BNP**.
Uses	• Used if patient LVEF ≤ 35% and still symptomatic with ACE/ARB • In this specific group of patients it **improves M&M.**

Positive Inotropes		
Drug name	**Digitalis Glycosides (Digitoxin, Digoxin)[1,2]**	
M.O.A	★ +ve inotropic effect by ↑ intracellular Ca & enhancing actin-myosin cross-bridge formation (binds to the Na-K ATPase ⟶ inhibits Na pump ⟶ ↑ intracellular Na ⟶ ↑ Na-Ca exchange • Has Vagotonic effect and Arrhythmogenic effect	
Uses	• Indicated in patients with heart failure and atrial fibrillation. • In patients with severe heart failure (NYHA class III–IV), digoxin reduces the likelihood of hospitalisation for heart failure, it also reduces symptoms, but it has **no effect on long-term survival.** • The role of digitalis has declined somewhat because of safety concern (Narrow therapeutic to toxic ratio)	
Digoxin toxicity	**Cardiac manifestations**	**Non cardiac manifestations**
	• Sinus bradycardia and arrest • A/V block (usually 2nd degree) • Atrial tachycardia with A/V Block • Development of junctional rhythm in patients with AF • PVC's, VT/ V fib (bi-directional VT)	• Anorexia • Nausea, vomiting • Headache • Xanthopsia sotoma (yellow vision) • Disorientation
Digoxin toxicity treatment	**1)** Hold the medications **2)** Observation. **3)** In case of A/V block or severe bradycardia ⟶ atropine followed by temporary PM if needed. **4)** In life threatening arrhythmia ⟶ digoxin-specific fab antibodies. **5)** Lidocaine and phenytoin could be used – try to avoid D/C cardioversion in non life threatening arrhythmia.	

- Positive inotropes are drugs that improve myocardial contractility (β adrenergic agonists, dopaminergic agents, phosphodiesterase inhibitors) e.g. Dopamine, Dobutamine, Milrinone, Amrinone
- Several studies showed ↑ **mortality with oral inotropic agents**
- So the only use for them now is in acute settings as cardiogenic shock

1- **It's ONLY given I.V. NOT Orally** it also has a very narrow therapeutic index and does not decrease mortality. Only improves morbidity
2- Use with caution in renal impairment or conduction disease, and with amiodarone

Management cont'

◀ Management of Chronic HF cont.

Vasodilators		
Drug name	**Hydralazine**	**Nitrate**
M.O.A	• **Reduction of afterload** by arteriolar vasodilation → reduce LVEDP, O2 consumption,improve myocardial perfusion, stroke volume and COP	• **Reduction of preload** By venous dilation → ↓ the venous return → ↓ the load on both ventricles.
Uses	• Valuable in chronic heart failure, when ACE inhibitors or ARBs are contraindicated. • Usually the maximum benefit is achieved by using agents with both action. • **Has mortality benefit.** • Their use is limited by pharmacological tolerance and hypotension.	

Anticoagulants	
Drug name	**Warfarin/NOAC**
Uses	• Atrial fibrillation • H/o embolic episodes • Left ventricular apical thrombus

Antiarrhythmics	
Uses	• Patients with h/o sustained VT or SCD → ICD implant • Patients with non-sustained ventricular tachycardia ○ Correction of electrolytes and acid base imbalance. • **Most common cause of Sudden Cardiac Death (SCD) in HF patients is ventricular tachyarrhythmia.**

hyperpolarization-activated cyclic nucleotide-gated (HCN) channel blockers	
Drug name	**Ivabradine**
M.O.A	• Acts on the I_f inward current in the SA node (phase 4), resulting in reduction of heart rate.
Uses	• Only use it if HR is not controlled by BB and remains > 70 bpm and the patient has sinus rhythm. (It is ineffective in patients with atrial fibrillation.) • In this group of patients **it improve M&M.**

Management cont'

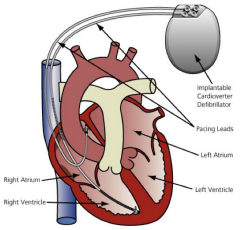
◀ Management of Chronic HF cont.

SGLT-2 inhibitors	
Drug name	**Dabagliflozin, Empagliflozin**
M.O.A	• Work on the proximal convoluted tubule, by inhibiting reabsorption of the glucose. • Reduce the blood glucose, systolic and diastolic blood pressure and work as diuretic.
Uses	• Recent studies showed mortality benefit in patients with heart failure.
ADRs	• UTIs, DKA and osteoporosis

Devices

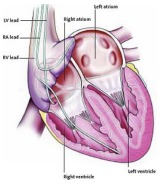
Implantable Cardioverter Defibrillator (ICD)[1]

- 1-3 leads + pulse generator
- Sudden onset criteria
- Stability criteria
- Treatment zones
- Pacing
- Cardioversion
- Defibrillation
- Combined CRT-D available



Biventricular pacemaker[2]

- Used only in patient with Wide QRS complexes & CHF
- Resynchronise ventricles by simultaneous pacing
- NICE guidance published 2007



Other assisting devices:

- Temporary ventricular assist devices.
- Implantable ventricular assist devices.

Cardiac Transplant:[3]

- It has become more widely used since the advances in immunosuppressive treatment.
- Survival rate:
  - 1 year 80% - 90%
  - 5 years 70%

Theses tables show that we should titre the dose of diuretics carefully (Give low dose initially) to avoid excessive volume depletion, which can cause a fall in cardiac output with hypotension (Cardiogenic shock), lethargy and renal failure.

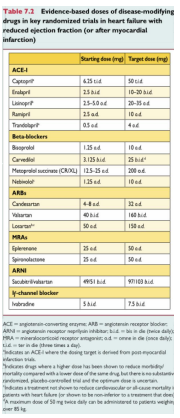


Table 7.2 Evidence-based doses of disease-modifying drugs in key randomized trials in heart failure with reduced ejection fraction (or after myocardial infarction)

	Starting dose (mg)	Target dose (mg)
ACE-I		
Captopril[a]	6.25 t.i.d.	50 t.i.d.
Enalapril	2.5 b.i.d.	10–20 b.i.d.
Lisinopril[a]	2.5–5.0 o.d.	20–35 o.d.
Ramipril	2.5 o.d.	10 o.d.
Trandolapril[a]	0.5 o.d.	4 o.d.
Beta-blockers		
Bisoprolol	1.25 o.d.	10 o.d.
Carvedilol	3.125 b.i.d.	25 b.i.d.[b]
Metoprolol succinate (CR/XL)	12.5–25 o.d.	200 o.d.
Nebivolol[c]	1.25 o.d.	10 o.d.
ARBs		
Candesartan	4–8 o.d.	32 o.d.
Valsartan	40 b.i.d.	160 b.i.d.
Losartan[d]	50 o.d.	150 o.d.
MRAs		
Eplerenone	25 o.d.	50 o.d.
Spironolactone	25 o.d.	50 o.d.
ARNI		
Sacubitril/valsartan	49/51 b.i.d.	97/103 b.i.d.
If-channel blocker		
Ivabradine	5 b.i.d.	7.5 b.i.d.

ACE = angiotensin-converting enzyme; ARB = angiotensin receptor blocker; ARNI = angiotensin receptor neprilysin inhibitor; b.i.d. = bis in die (twice daily); MRA = mineralocorticoid receptor antagonist; o.d. = omne in die (once daily); t.i.d. = ter in die (three times a day).
[a]Indicates an ACE-I where the dosing target is derived from post-myocardial infarction trials.
[b]Indicates drugs where a higher dose has been shown to reduce morbidity/mortality compared with a lower dose of the same drug, but there is no substantive randomized, placebo-controlled trial and the optimum dose is uncertain.
[c]Indicates a treatment not shown to reduce cardiovascular or all-cause mortality in patients with heart failure (or shown to be non-inferior to a treatment that does).
[d]A maximum dose of 50 mg twice daily can be administered to patients weighing over 85 kg.

Table 7.3 Doses of diuretics commonly used in patients with heart failure

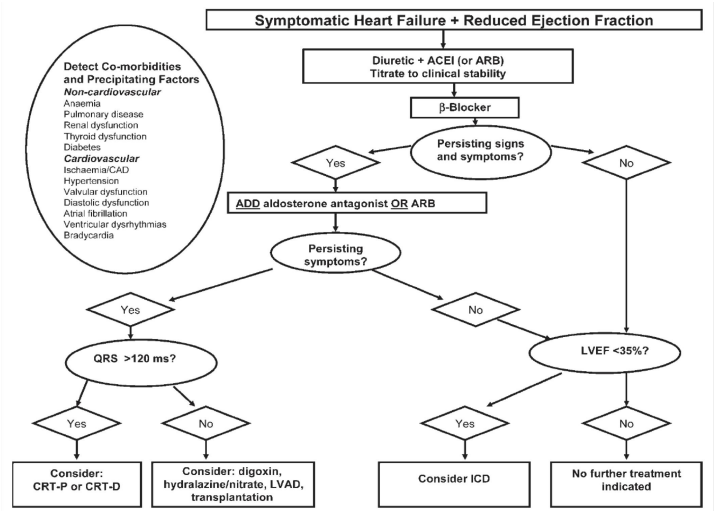
Diuretics	Initial dose (mg)	Usual daily dose (mg)
Loop diuretics[a]		
Furosemide	20–40	40–240
Bumetanide	0.5–1.0	1–5
Torasemide	5–10	10–20
Thiazides[b]		
Bendroflumethiazide	2.5	2.5–10
Hydrochlorothiazide	25	12.5–100
Metolazone	2.5	2.5–10
Indapamide[c]	2.5	2.5–5
Potassium-sparing diuretics[d]		
	+ACE-I/ ARB / -ACE-I/ ARB	+ACE-I/ ARB / -ACE-I/ ARB
Spironolactone/ eplerenone	12.5–25 / 50	50 / 100–200
Amiloride	2.5 / 5	5–10 / 10–20
Triamterene	25 / 50	100 / 200

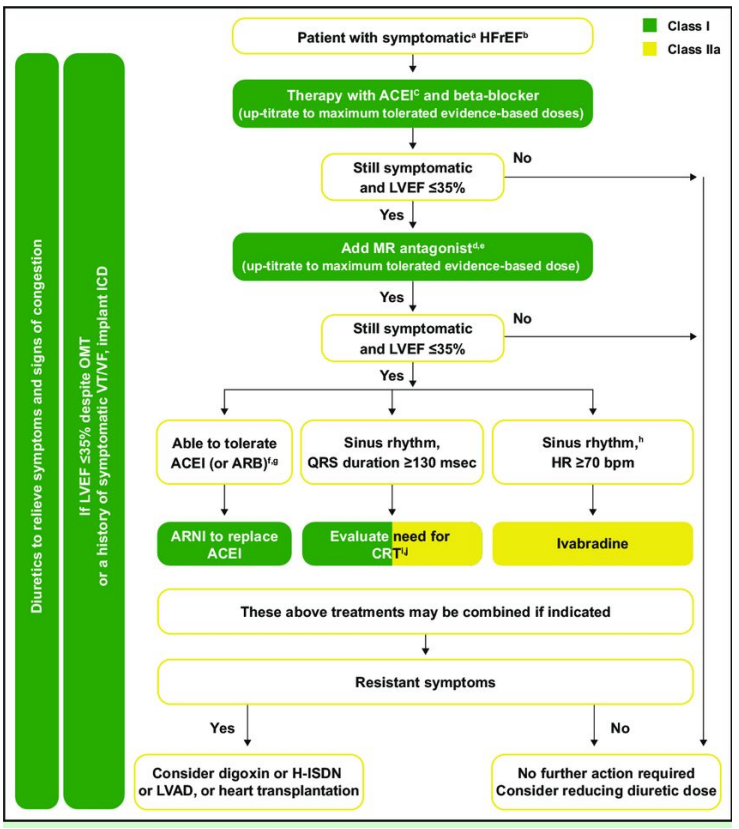
ACE-I = angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor blocker.
[a]Oral or intravenous; dose might need to be adjusted according to volume status/weight; excessive doses may cause renal impairment and ototoxicity.
[b]Do not use thiazides if estimated glomerular filtration rate <30 mL/min/1.73 m², except when prescribed synergistically with loop diuretics.
[c]Indapamide is a non-thiazide sulfonamide.
[d]A mineralocorticoid antagonist (MRA) i.e. spironolactone/eplerenone is always preferred. Amiloride and triamterene should not be combined with an MRA.

1- Used for those with ischemic cardiomyopathy and EF below 30%. Has mortality benefit.
2- Indicated in those with dilated cardiomyopathy and an EF under 35% and a wide QRS >140ms who have persistent symptoms. Has mortality benefit.
3- When maximal therapy (ACEI, BB, spironolactone, diuretics, digoxin) and possibly the biventricular pacemaker fail to control symptoms of CHF, then the only alternative is to seek cardiac transplant.

Management cont'

◄ Management of Chronic HF cont.





Drugs used in (according to MTB)	
Systolic dysfunction	**Diastolic dysfunction**
• ACEI or ARB • BB • Spironolactone • Diuretics • Digoxin	• Spironolactone • Diuretics • Uncertain: ACEI, ARBs **NOT used:** BB, Digoxin

General recommendations

1. An ACE inhibitor should be given to all patients with heart failure unless there are contraindications. In patients intolerant of ACE inhibitors, ARBs are an alternative (level of evidence, A).
2. In symptomatic patients with heart failure, beta-blockers are recommended to reduce mortality rates (level of evidence, A).
3. Aldosterone antagonists are recommended to reduce mortality rates in certain patients with heart failure. These include patients with current or recent history of dyspnea at rest, and patients with recent myocardial infarction who have systolic dysfunction with either clinically significant signs of heart failure or with concomitant diabetes mellitus (level of evidence, B).
4. For persistently symptomatic black patients with heart failure, direct-acting vasodilators reduce overall mortality rates when added to background therapy with ACE inhibitors, beta-blockers, and diuretics (if needed). Direct-acting vasodilators are also an alternative for patients with heart failure who are intolerant of ACE inhibitors (level of evidence, B).
5. For patients with heart failure and volume overload, diuretics are recommended (level of evidence, B).

◄ Contraindicated drugs

Treatments (or combinations of treatments) that may cause harm in patients with symptomatic (NYHA class II-IV) systolic heart failure:

1) **Thiazolidinediones (glitazones)** should not be used as they cause worsening HF and increase the risk of HF hospitalization.

2) **Most CCBs (with the exception of amlodipine and felodipine)** should not be used as they have a negative inotropic effect and can cause worsening HF.

3) **NSAIDs and COX-2 inhibitors** should be avoided if possible as they may cause sodium and water retention, worsening renal function and worsening HF.

4) The addition of an **ARB (or renin inhibitor) to the combination of an ACEI AND a mineralocorticoid antagonist** is NOT recommended because of the risk of renal dysfunction and hyperkalemia

Management cont.

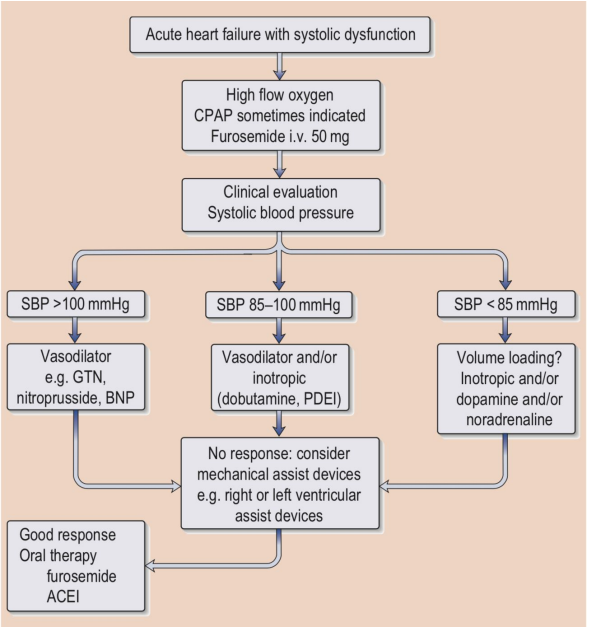
◀ Management of Acute HF

- Acute heart failure with pulmonary oedema is a medical emergency that should be treated urgently.

16.15 Management of acute pulmonary oedema

Action	Effect
Sit the patient up	Reduces preload
Give high-flow oxygen	Corrects hypoxia
Ensure continuous positive airway pressure (CPAP) of 5–10 mmHg by tight-fitting mask	Reduces preload and pulmonary capillary hydraulic gradient
Administer nitrates:* IV glyceryl trinitrate (10–200 µg/min) Buccal glyceryl trinitrate 2–5 mg	Reduces preload and afterload
Administer a loop diuretic: Furosemide (50–100 mg IV)	Combats fluid overload

*The dose of nitrate should be titrated upwards every 10 mins until there is an improvement or systolic blood pressure is <110 mmHg.



Note: If these measures **prove ineffective use dobutamine.**

◀ Prognosis

- Annual mortality rate depends on patients symptoms and LV function.
- 5% in patients with mild symptoms and mild ↓ in LV function.
- 30% to 50% in patient with advances LV dysfunction and severe symptoms.
- **40% – 50% of death is due to SCD.**

◀ What factors indicate poor prognosis in HF?

Clinical	• High NYHA class, hypotension, tachycardia at rest, JVD, S3
Labs	• Hyponatremia, elevated BNP, renal insufficiency
EKG	• QRS >120, LBBB
Echo	• Severe reduction in EF, pulmonary hypertension, diastolic dysfunction, RV function impairment
Associated conditions	• Anemia, AF, DM

Case Summary

A 70-year-old man presents to the emergency department complaining of increased shortness of breath with minimal exercise, cough, and fatigue. These symptoms began 2 weeks ago and have progressed gradually. He reports he used to feel this way “all the time” years ago but that this has not happened much since he began using his inhalers and his “water pill.” He also has a history of chronic obstructive pulmonary disease (COPD), congestive heart failure (CHF), coronary artery disease (CAD), diabetes mellitus, hypertension, and 30-pack-year of smoking. He denies swelling of the extremities, fever or chills, productive cough, chest pain, or palpitations. He cannot remember the names of his medications but says he has not missed any doses. When asked about his diet, he says he has been eating more hot soup since the weather has gotten colder. His temperature is 37.5°C (99.5°F), blood pressure is 135/90 mm Hg, heart rate is 90/min, respiratory rate is 18/min, and oxygen saturation is 94% on room air. Examination of the neck reveals mild jugular venous distention. Examination of the lungs reveals loud crackles throughout the lung fields bilaterally. Examination of the heart reveals a laterally displaced point of maximum impulse with no murmurs, rubs, or gallops. There is mild clubbing of the extremities, as well as pitting edema of the lower extremities to the knee, bilaterally. His plasma brain natriuretic peptide level on rapid bedside assay is 500 pg/mL, and an x-ray of the chest reveals perivascular haziness, interstitial edema, and an enlarged cardiac silhouette.

Q1: What conditions should be included in the differential diagnosis?

CAD, COPD, and CHF each may present with dyspnea on exertion and fatigue. It is of primary importance to distinguish between them when evaluating the presenting symptoms. Etiologies of gradually worsening shortness of breath and fatigue can include both cardiac and pulmonary diseases, including the following: Anemia, Heart failure secondary to ischemia/infarction, dysrhythmia, valvular dysfunction, infection, or volume overload, Lung infections (pneumonia, bronchitis, bronchiectasis), Mechanical impairment of ventilation, Pulmonary edema, Pulmonary embolism, Sepsis.

Q2: What’s the most likely diagnosis?

CHF exacerbation leading to pulmonary edema. This patient’s dyspnea, jugular venous distension, and tachypnea in the presence of crackles, pulmonary edema, elevated brain natriuretic peptide (BNP) level, and cardiomegaly suggest an acute exacerbation of CHF. An exacerbation of COPD is unlikely given that this patient does not have fever, productive cough, or wheezing. Additionally, the patient reported increasing intake of soup, a particularly salty food, which can significantly increase water retention, thereby worsening CHF.

Q3: What are the typical laboratory and imaging findings in this condition?

In addition to an x-ray of the chest that may show pulmonary edema, patients with CHF exacerbations may have the following: Decreased hematocrit (anemia may exacerbate CHF), Increased potassium, creatinine, and blood urea nitrogen levels (renal failure may exacerbate CHF), Increased plasma BNP level, which is usually elevated in CHF exacerbations, A chest radiograph showing cardiomegaly, cephalization of pulmonary vessels, and/or pleural effusion, ECG changes showing left ventricular hypertrophy, arrhythmias, or ischemia or low-voltage or old infarcts (in fact, a normal ECG makes systolic dysfunction highly unlikely), ECG showing abnormal ventricular size (dilated, hypertrophic, or restrictive cardiomyopathy) or function (systolic or diastolic).

Q4: What’s the most appropriate treatment for this patient?

This patient appears to have stage C heart failure. His physical exam and x-ray of the chest show evidence of myocardial hypertrophy, and he is having recurrent symptoms. He should be admitted to the hospital for a trial of intravenous diuresis (which often succeeds when oral diuretics fail). An echocardiogram should be obtained to evaluate for left ventricular structural abnormalities as well as determine an ejection fraction. He should be prescribed an ACE inhibitor or an angiotensin receptor blocker (given his atherosclerosis, hypertension, and diabetes mellitus), a diuretic (given his evidence of fluid retention), and digitalis (if his ejection fraction is less than 25%, as this has been shown to reduce hospitalization). He should also receive frequent blood pressure and weight monitoring, exercise counseling, and possibly an aldosterone antagonist (depending on his ejection fraction). In addition, he should take aspirin and a statin for his CAD.

Summary

Heart failure

Classification

Systolic dysfunction (HFrEF)

- Impaired contractility, EF is reduced.
- Causes: IHD, HTN, VHD etc

Diastolic dysfunction (HFpEF)

- Impaired ventricular filling, EF is preserved.
- Causes: HTN leading to myocardial hypertrophy.

High 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

Low Output HF

Cardiac output is inadequate to perfuse the body (i.e. EF <40%), or can only be adequate with high filling pressures.

Acute 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.

Chronic HF

- 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

- **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.

Right sided HF

- **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

Treatment

Acute HF

1) **Oxygen**
2) **Loop diuretics** (Furosemide)
3) **Nitrate** (IV and Buccal Glyceryl trinitrate)
4) **Morphine:** Can be of value in distressed patients but must be used sparingly, as they may cause respiratory depression and exacerbation of hypoxaemia and hypercapnia.

Note: If these measures **prove ineffective use dobutamine.**

Chronic HF

HFrEF

- ACEI or ARB
- BB
- Spironolactone
- Diuretics
- Digoxin

HFpEF

- Spironolactone
- Diuretics
- Uncertain: ACEI, ARBs
- **NOT used:** BB, Digoxin

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!

THANKS!!

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Send us your feedback:
We are all ears!