Congestive Heart Failure

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General considerations

- The Framingham heart study showed that men in whom clinical symptoms of CHF developed had a 62% probability of dying within 5 years of the onset of symptoms

Definition

- Congestive heart failure (CHF) is a complex clinical syndrome characterized by dysfunction of the left, right, or both ventricles and the resultant changes in neurohormonal regulation

Etiology

- Coronary artery diseases
- Hypertension
- Dilated cardiomyopathy
- Valvular heart disease
- Other

Pathophysiology

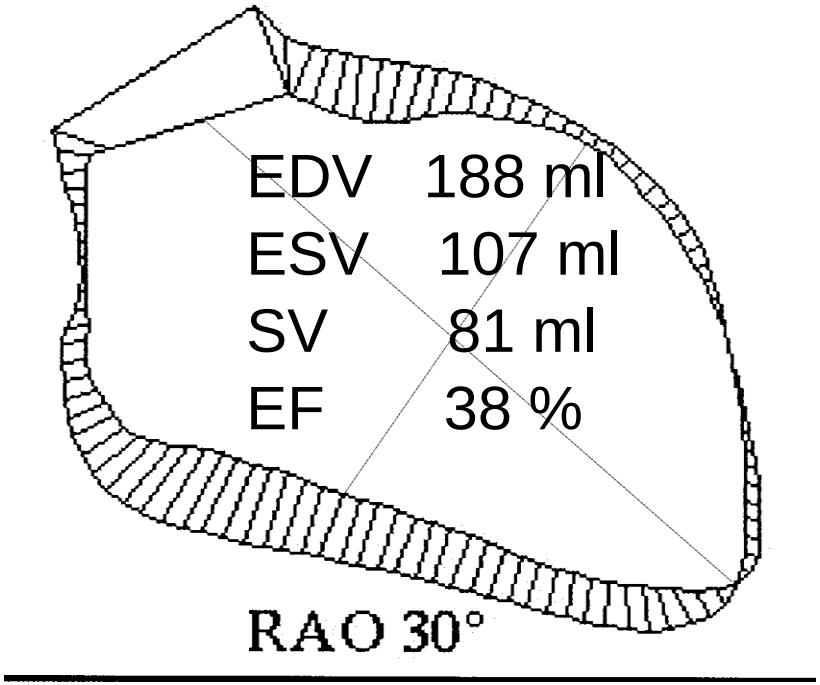
- increased systolic blood pressure (pressure overload) or diastolic volume (volume overload)
- loss of myocardium
- cells hypertrophy
- alterations in biochemistry, electrophysiology, and contractile function lead to mechanical alterations of myocardial function: The rate of contraction slows, the time to develop peak tension increases, and myocardial relaxation is delayed

Pathophysiology - Cardiac remodeling

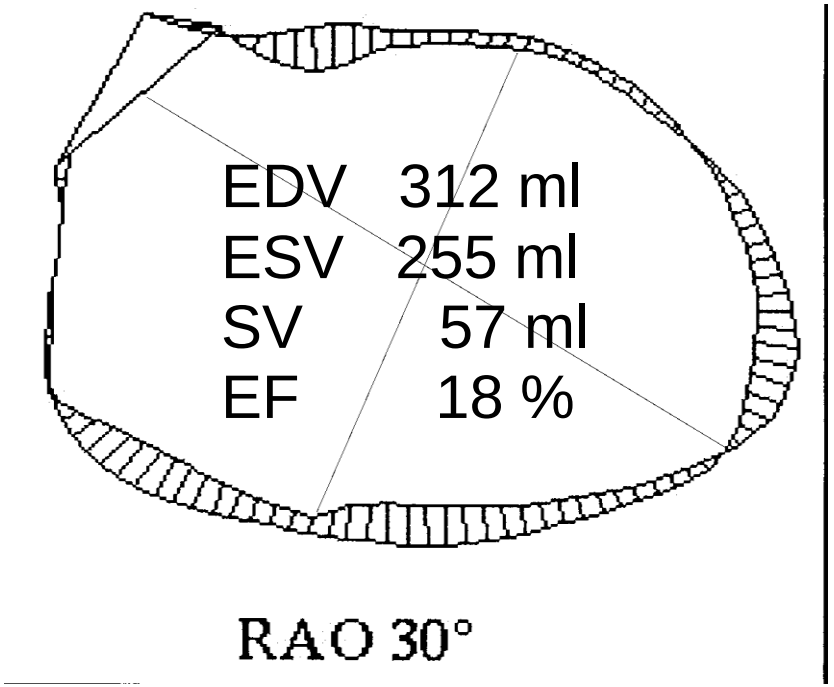
- The force of myocardial contraction is eventually reduced as cell loss and hypertrophy continue, leading to significant geometric ventricular alterations and increased volumes
- Cardiac remodeling - process of chamber **dilatation** or **hypertrophy**

Cardiac remodeling in Heart Failure

Iniţial time



after 2 years



Neurohormonal compensatory mechanisms

- After the initial compensatory phase, the increase in intracavitary volume is usually associated with further reductions in ventricular ejection fraction (progressive systolic dysfunction) and eventually with abnormalities in the peripheral circulation from activation of various neurohormonal compensatory mechanisms

Renin-angiotensin system

- The abnormal neurohormonal responses lead to increased **systemic sympathetic** tone and activation of the **renin-angiotensin system**
- Production of angiotensin increases, causing peripheral vasoconstriction
- The increase in peripheral arterial resistance limits cardiac output during exercise
- Angiotensin II also stimulate release of aldosterone by the adrenal glands, enhancing sodium retention and thus leading to fluid retention and peripheral edema

Acute Heart Failure

the underlying condition develops rapidly or an acute precipitating factor is present, the result may be inadequate organ perfusion or acute congestion of the venous bed draining into the affected ventricle, causing sudden cardiac decompensation, with a concomitant reduction in cardiac output and an acute onset of symptoms

Chronic Heart Failure

- adaptive mechanisms are gradually activated and cardiac hypertrophy develops
- When the onset of left-heart failure is gradual, the right heart develops higher pressures in response to higher pulmonary resistance
- A patient with chronic heart failure may achieve compensation but then experience acute decompensation as a result of a precipitating condition

Precipitating condition of decompensation

- Arithmias: atrial fibrilatie/flutter, paroxismal tahicardias, A-V block hight degree
- 2. Infection: sistemic or cardiac
- 3. Hypertensive emergency
- 4. Myocardial ischemia
- 5.Pulmonary artery embolism
- 6. Anemia

Left- and Right-Sided Heart Failure

- Heart failure is more often limited to one side when the onset is abrupt (eg, in acute MI)
- The disease process may involve only one ventricle initially, biventricular failure usually follows, especially when the left ventricle is the site of initial damage
- Both ventricles have a common interventricular septum, and biochemical changes are not confined to the stressed ventricle but involve the opposite ventricle as well

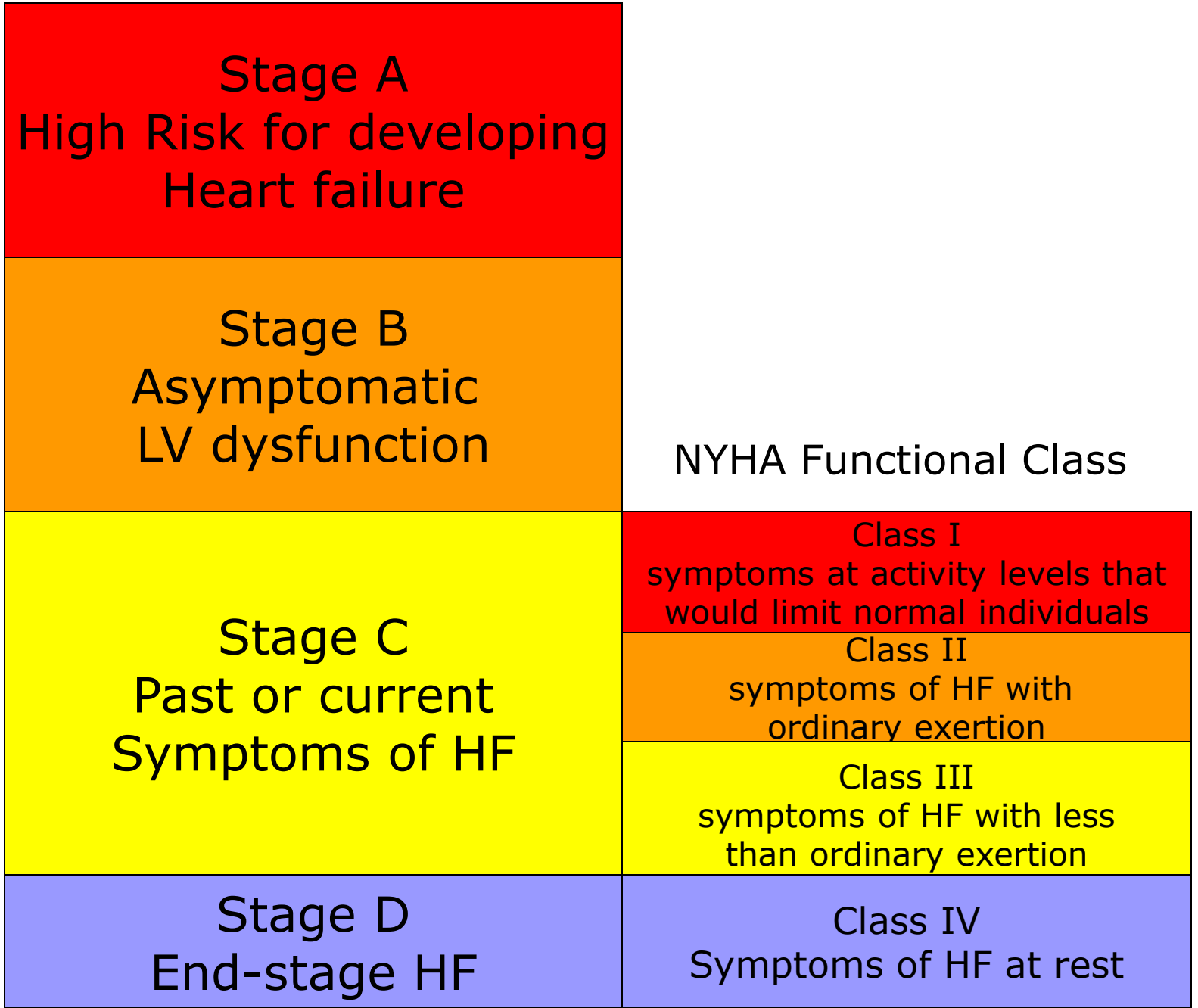
Left- and Right-Sided Heart Failure

	Simptoms	Signs
Left Sided Heart Failure	Dispnee(de effort,de repaus,ortopnee,dispnee paroxistica nocturna) Tuse,hemoptizii Respiratie Astenie,fatigabilitate	Paloare/cianoza, transpiratii Raluri pulmonare subcrepitante simetrice Tahicardie Deplasare laterala soc apexian Cresterea ariei matitatii cardiace Galop protodiastolic de VS (Zg 3) Suflu sistolic apical
Right-Sided Heart Failure	Hepatalgii Balonari, greata, anorexie Edeme periferice	Edeme periferice/generalizate declive Cianoza Subicter/icter Hepatomegalie dureroasa Turgescenta jugulara Reflux hepato-jugular Revarsate lichidiene(pleural, pericardic, ascitic) Galop protodiastolic de VD(Zg 3) Suflu sistolic endapexian(regurgitare tricuspidiana secundara)

High-Output and Low-Output Heart Failure

- Most cases of heart failure are associated with a low-output state
- High-output heart failure, which is less common, is usually associated with a hyperkinetic circulatory state usually due to increased demand on the heart from another condition, such as anemia or thyrotoxicosis

Stages of HF: ACC/AHA



Stages of Heart Failure

COMPLEMENT, DO NOT REPLACE NYHA CLASSES

- **NYHA Classes - shift back/forth in individual patient (in response to Rx and/or progression of disease)**

- **Stages - progress in <u>one</u> direction due to cardiac remodeling**

Symptoms and Signs

Dyspnea, or breathlessness

- **Paroxysmal nocturnal dyspnea** occurs after the patient has been asleep and in the supine position for some time
- **Orthopnea** is defined as dyspnea that occurs—often within a few minutes—when the patient assumes a supine position; sitting up or standing usually improves the symptoms. The most severely affected patients usually sleep sitting upright in a chair
- Orthopnea has the same cause as PND, but it represents more severe cardiac impairment

Cough: dry or nonproductive

- This is due to pulmonary congestion and in patients with heart failure is usually relieved by successful treatment of the heart failure
- Certain drugs used to treat heart failure, such as angiotensin-converting enzyme (ACE) inhibitors, can also cause cough

Fatigue and Weakness

- These symptoms are secondary to low cardiac output with decreased perfusion of skeletal muscles and can occur with exertion or at rest
- they may be worsened after eating because of the increased splanchnic demand for blood flow, which may stress the limited reserve

Nocturia

Nocturia and Oliguria

- Renal filtration of sodium and water is decreased in patients with compromised LV function, in part, because of the redistribution of blood flow away from the kidneys in the upright position and during physical activity
- Oliguria is associated with a markedly reduced cardiac output and is usually a sign of terminal heart failure; it indicates a poor prognosis

Cerebral Symptoms

- Elderly patients with advanced heart failure may have confusion, memory impairment, anxiety, headaches, insomnia, nightmares and, occasionally, disorientation, delirium, and hallucinations
- These cerebral symptoms are predominantly related to a reduced cardiac output and poor perfusion of brain and other neurologic tissues

Abdominal Symptoms

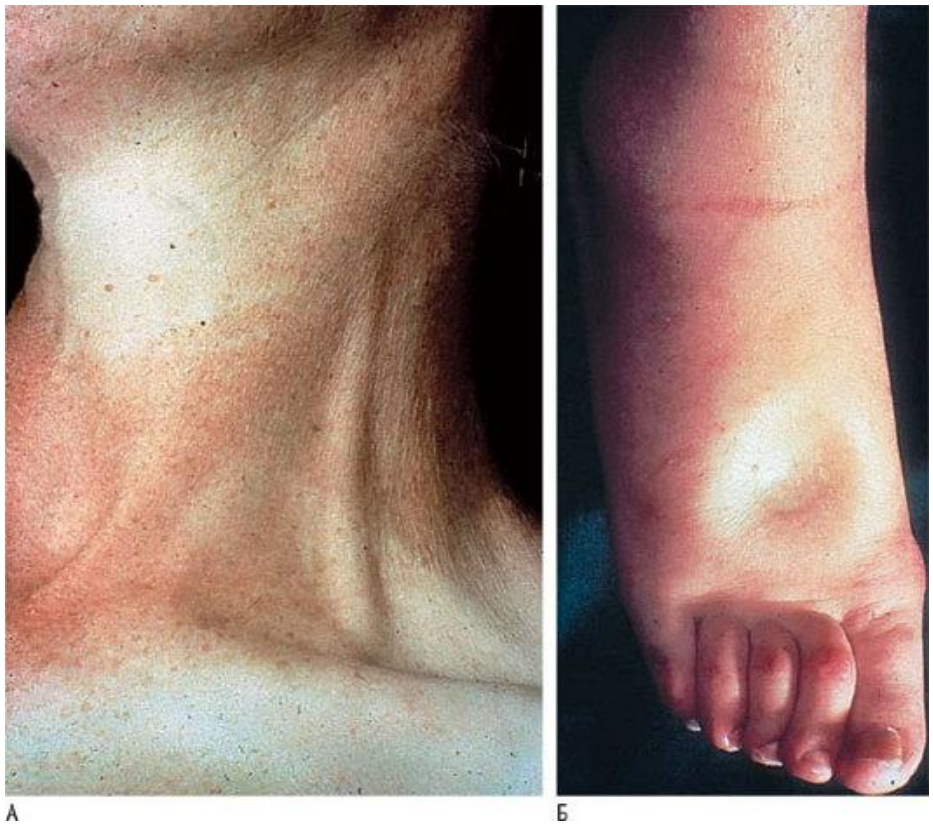
- Gastrointestinal complaints may develop in patients with heart failure as a result of hepatic congestion and edema of the abdominal wall and intra-abdominal organs
- Congestion of abdominal organs may be present with ascites, abdominal fullness and enlargement, early satiety, bloating, anorexia, nausea, vomiting, constipation, and upper abdominal discomfort

Physical Examination: signs

- malnourished, and occasionally cachectic
- pallor and coldness of the limbs and cyanosis of the digits because of vasoconstriction
- diaphoresis and abnormal distention of the superficial veins
- Sinus tachycardia is often observed and usually develops in an effort to maintain the cardiac output when heart failure is decompensated or the stroke volume is significantly decreased

- Hyperpnea and apnea in a smooth crescendo-decrescendo manner (Cheyne-Stokes) can be seen in patients with heart failure
- Moist rales heard initially at the lung bases (transudation of fluid into the alveoli that subsequently moves into the airways)
- In pulmonary edema, coarse bubbling rales and wheezes are heard over both lung fields and may be accompanied by frothy sputum, with or without bloodstaining
- Hydrothorax (pleural effusion) is usually bilateral and can intensify the severity of dyspnea by further reducing vital capacity
- Stony dullness on percussion is characteristic of pleural effusion on one or both sides

- Pitting edema is common, typically noted in the feet and ankles of ambulatory patients and in the sacral area of bedridden ones
- Late in the course of heart failure, edema may become massive and generalized (anasarca), it can involve the upper extremities, the thoracic and abdominal walls, and the genital area
- Occasionally, with acute accumulation of edema or associated trauma, skin rupture and extravasation of fluid can occur



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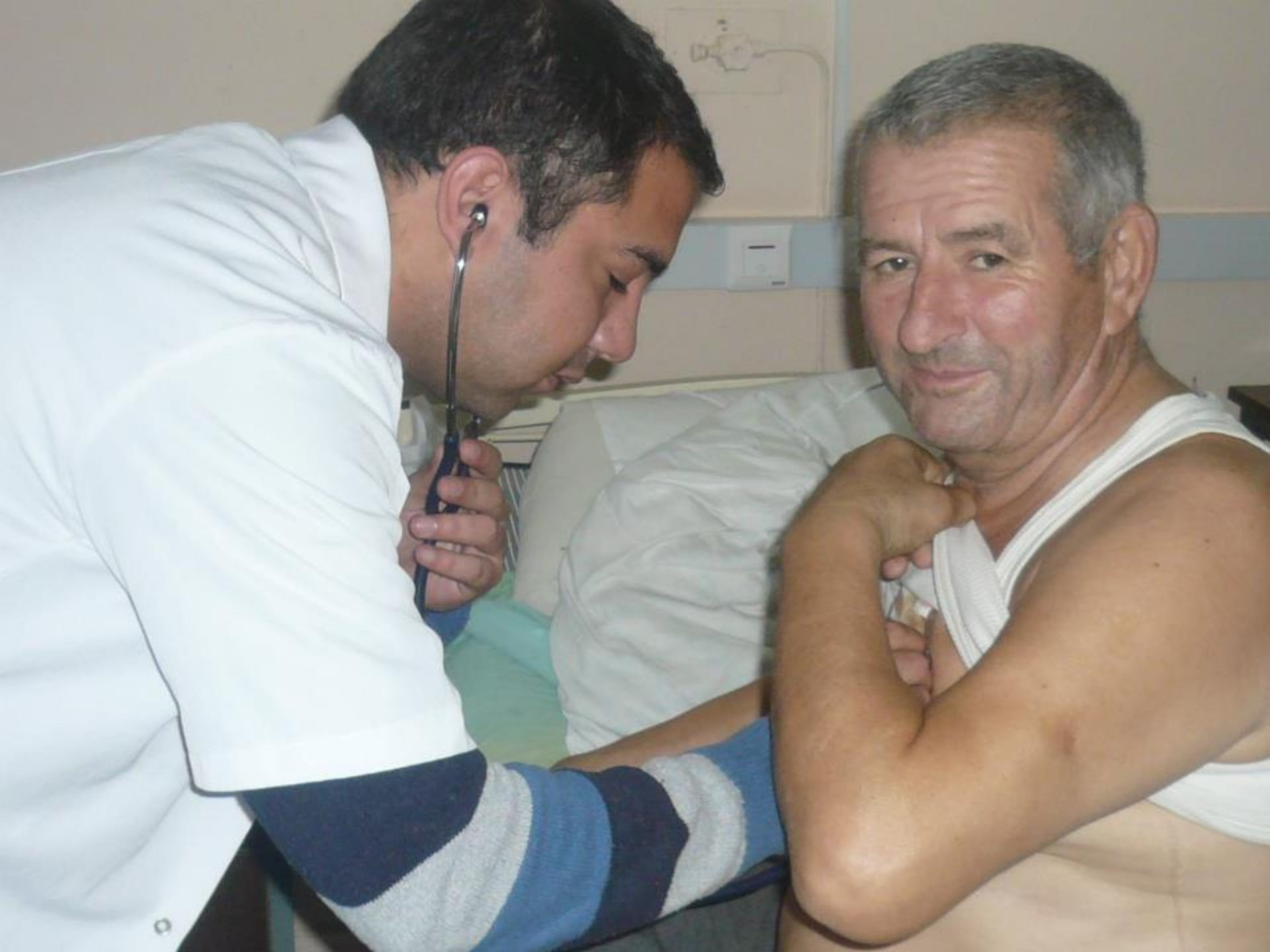
Cardiovascular system

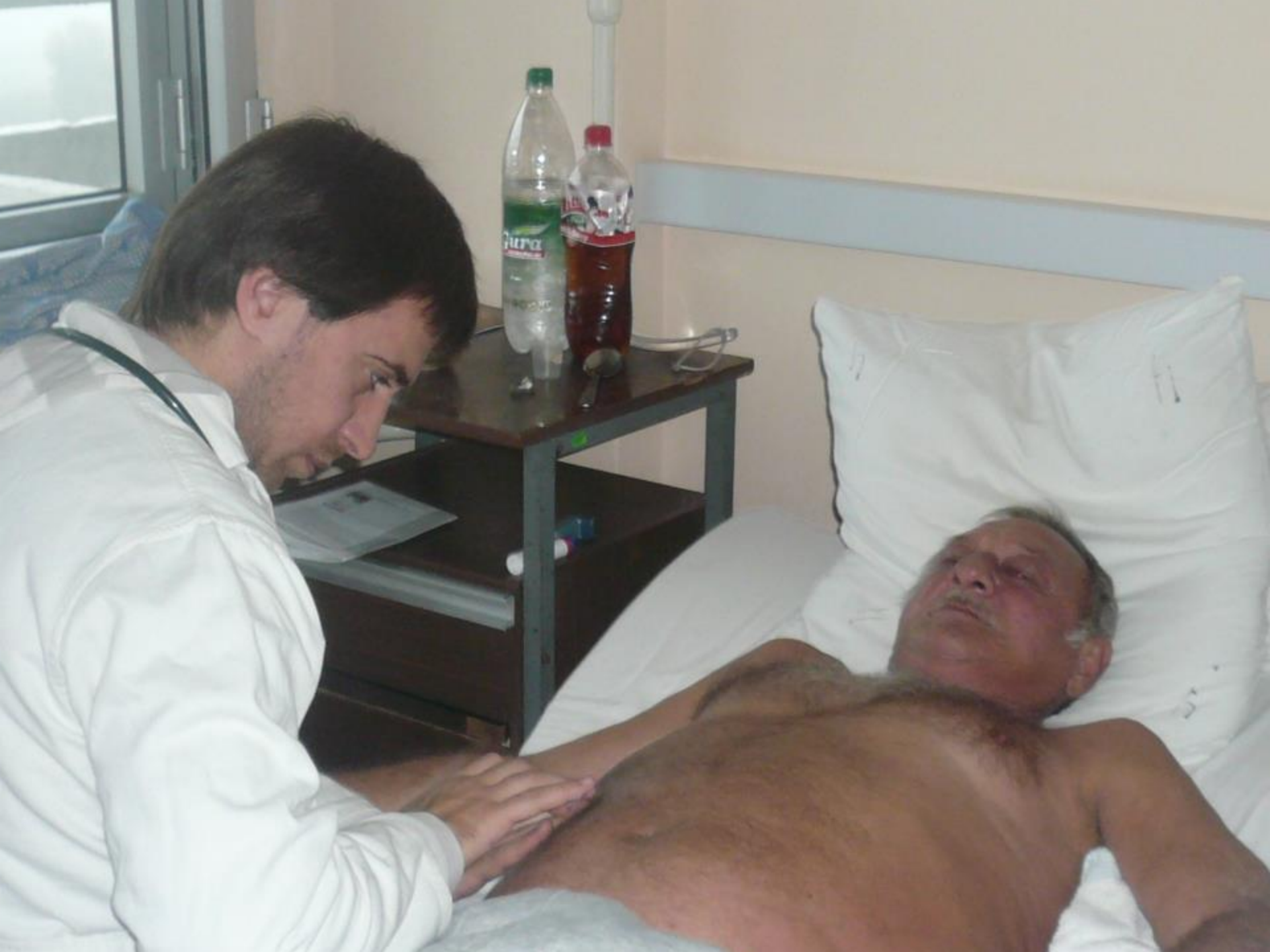
- Cardiomegaly
- Late diastolic atrial sound (S4 gallop)
- A protodiastolic sound (S3 gallop) occurs in patients with more advanced heart failure and is caused by acute deceleration of ventricular inflow after the early filling phase
- Systolic murmurs are common in heart failure and are largely secondary to mitral or tricuspid regurgitation that can result from ventricular dilatation



- Systemic venous hypertension can be detected by abnormal distention of the internal jugular veins

- The jugular venous pressure normally declines on inspiration, it can rise in patients with right-heart failure (Kussmaul sign)

- Liver enlargement and tenderness on palpation are marked by epigastric fullness and dullness to percussion in the right upper quadrant
- These findings may persist after other signs of heart failure have disappeared because it takes longer for hepatic congestion to disappear

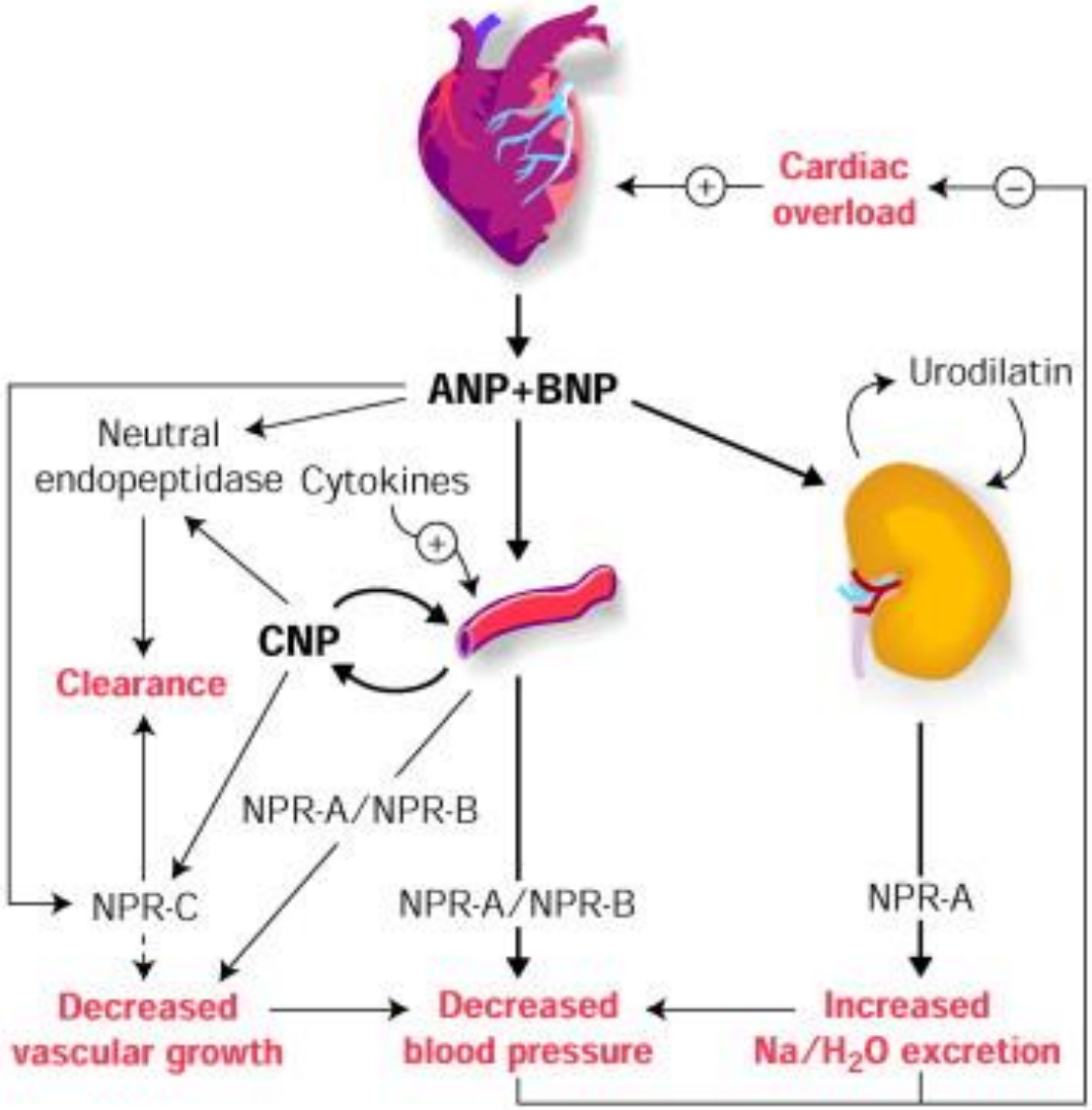


- Pulsus alternans is common in patients with CHF; when severe, it can be detected by sphygmomanometry or by palpation of peripheral pulses, particularly the femoral pulse
- This sign is characterized by a regular rhythm of alternating strong and weak pulsations

Laboratory Findings

- Hyponatremia and other significant electrolyte abnormalities
- Elevated levels of liver enzyme values, particularly serum aspartate aminotransferase, hyperbilirubinemia
- Natriuretic peptide type B

Biomarkeri: Natriuretic peptide type B



Diagnostic Studies

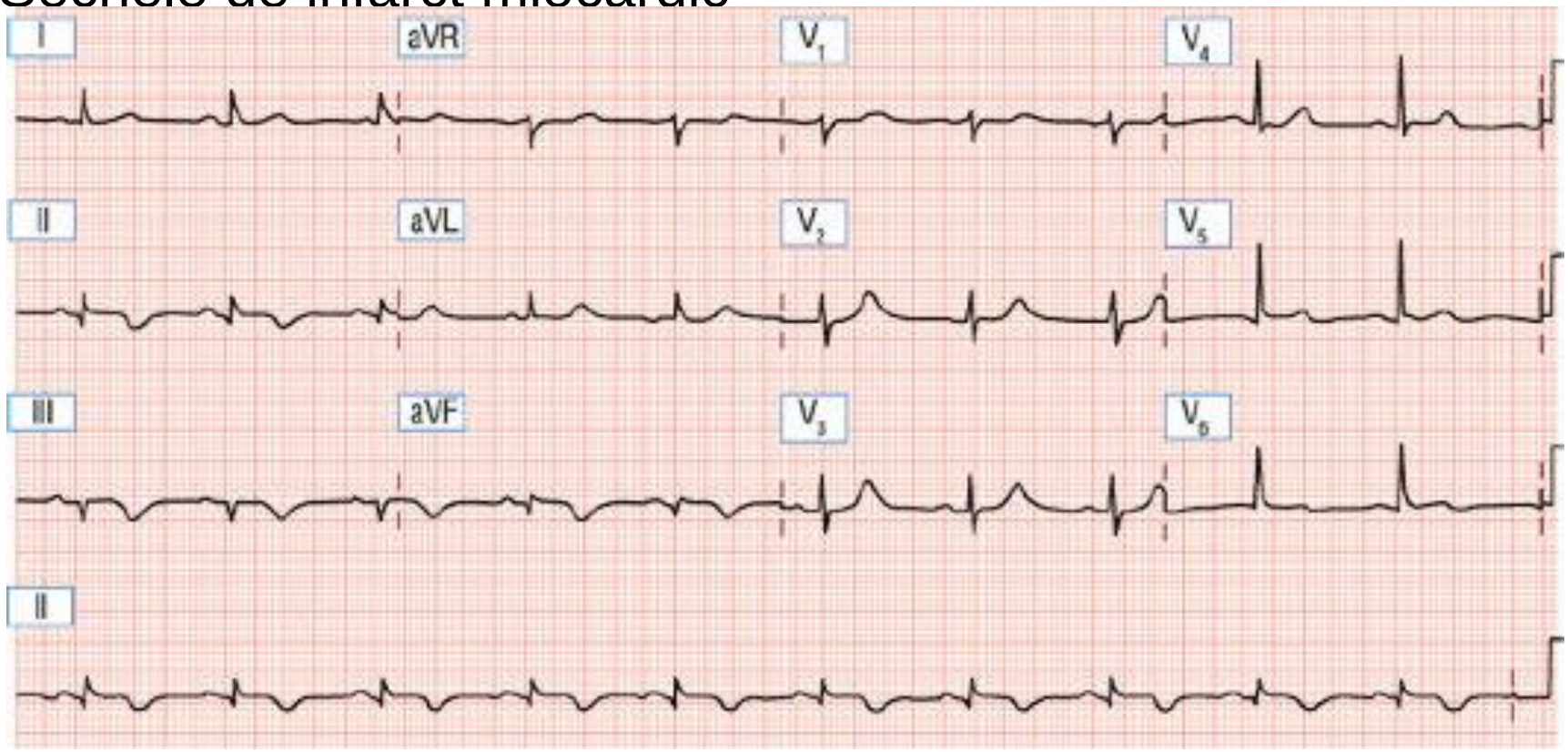
- **ECG** Changes in the 12-lead electrocardiogram (ECG) are generally **nonspecific**
- Sinus tachycardia is usually present
- Isolated premature ventricular beats are common, and complex ventricular arrhythmias can be detected in most patients during prolonged (24- to 48-hour) Holter monitoring
- ECG findings suggestive of atrial and ventricular chamber enlargement may be evident
- Intraventricular conduction delays are also common and include left bundle branch block as well as other, nonspecific repolarization changes

INVESTIGATION: ECG

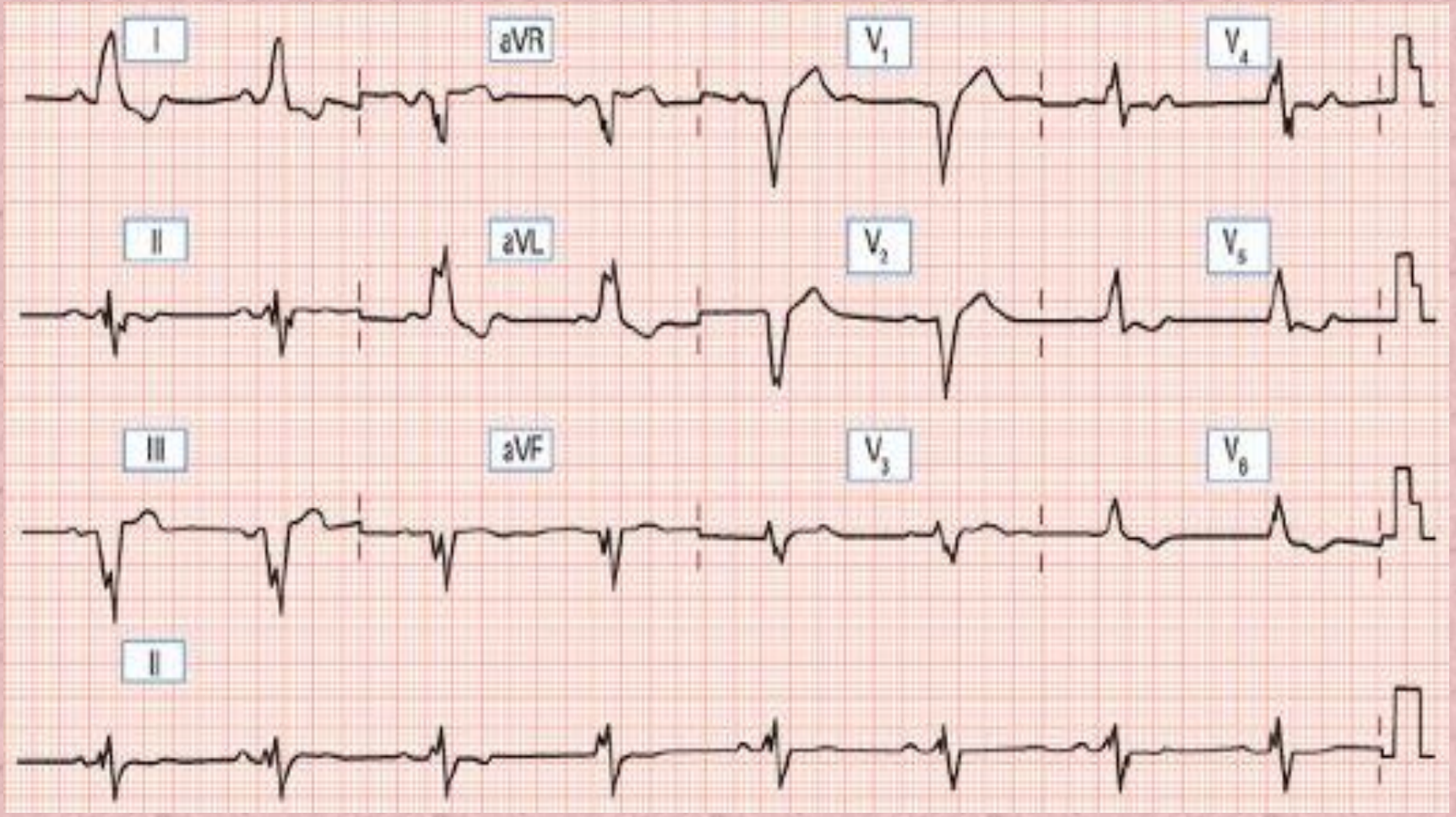
Tulburari de ritm

Largirea complexului QRS (prelungit >130ms)

Sechele de infarct miocardic



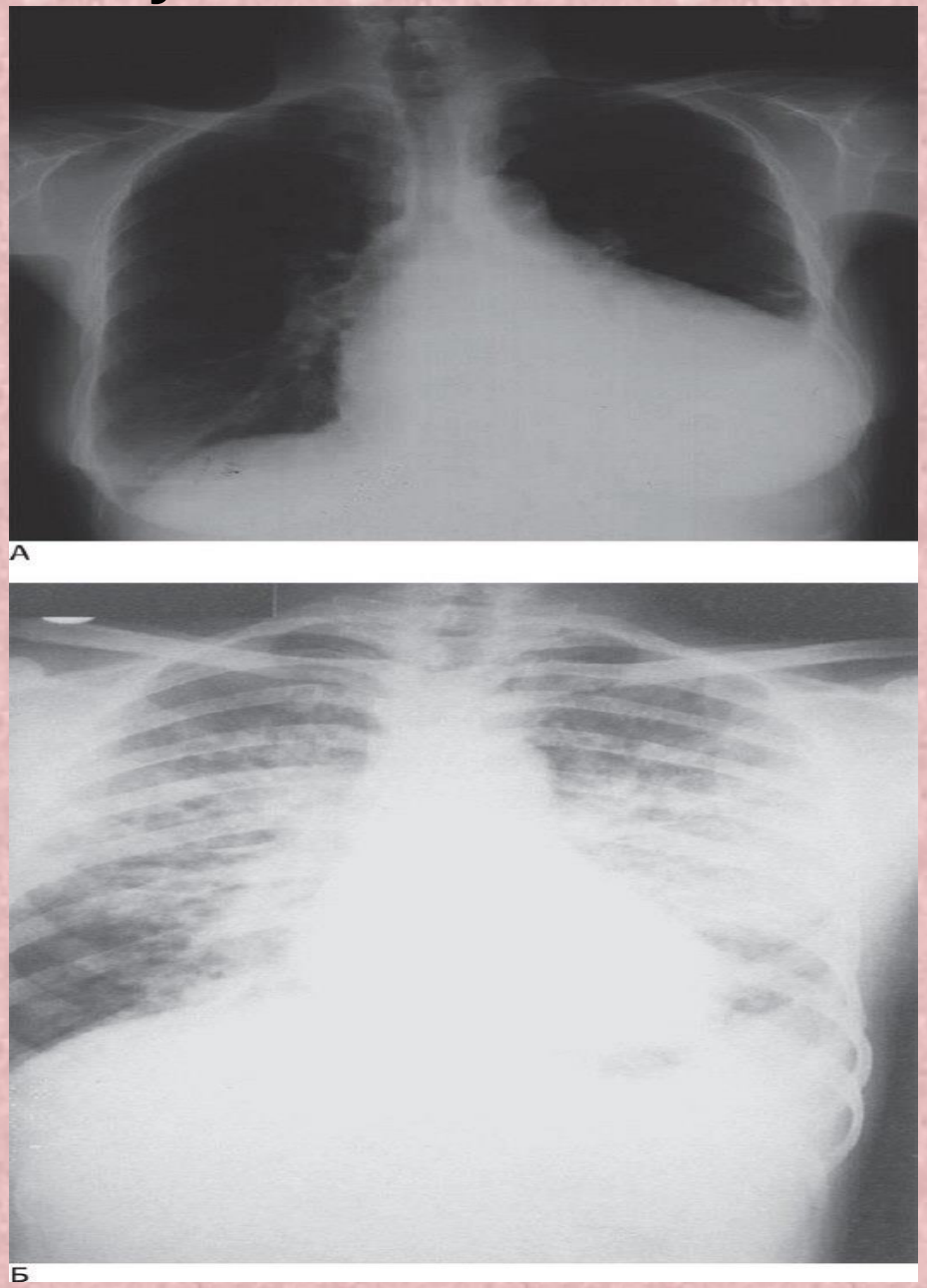
ECG in patient with HF



Chest Radiography

- Cardiomegaly -cardiothoracic ratio > 50% can be found
- Interstitial and perivascular edema develop with acute increases in pulmonary capillary wedge pressure above 20–25 mm Hg
- bronchovascular markings at the bases are prominent
- Interstitial edema can present as perivascular or peribronchial edema (initially in perihilar and then in peripheral zones)
- Kerley lines, spindle-shaped linear opacities at the periphery of the lung bases, occur in the later stages of heart failure
- pleural fluid can produce discrete interlobular-type linear opacities and subpleural fluid accumulation between the lung and adjoining pleura.

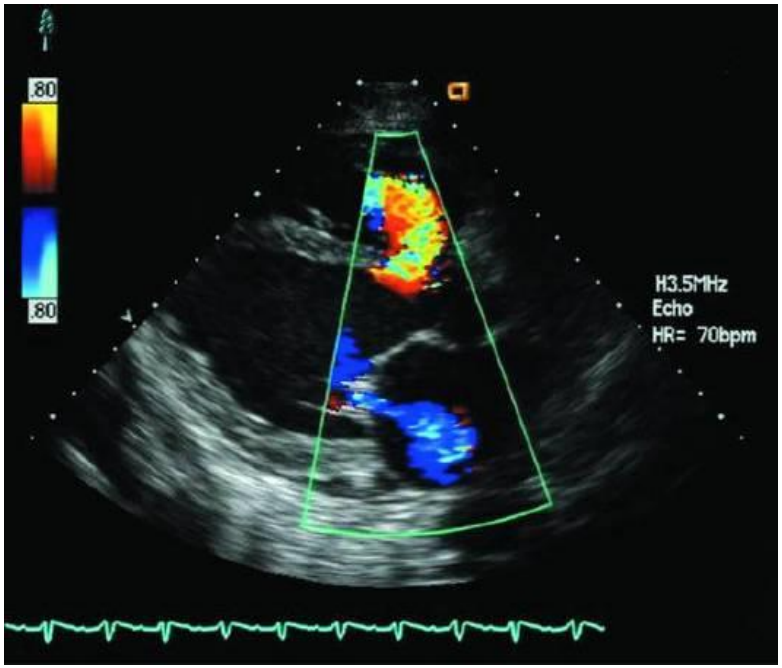
Thoracic X-Ray in HF Pt

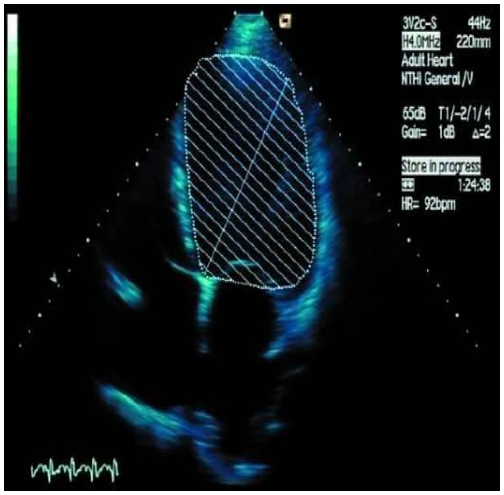


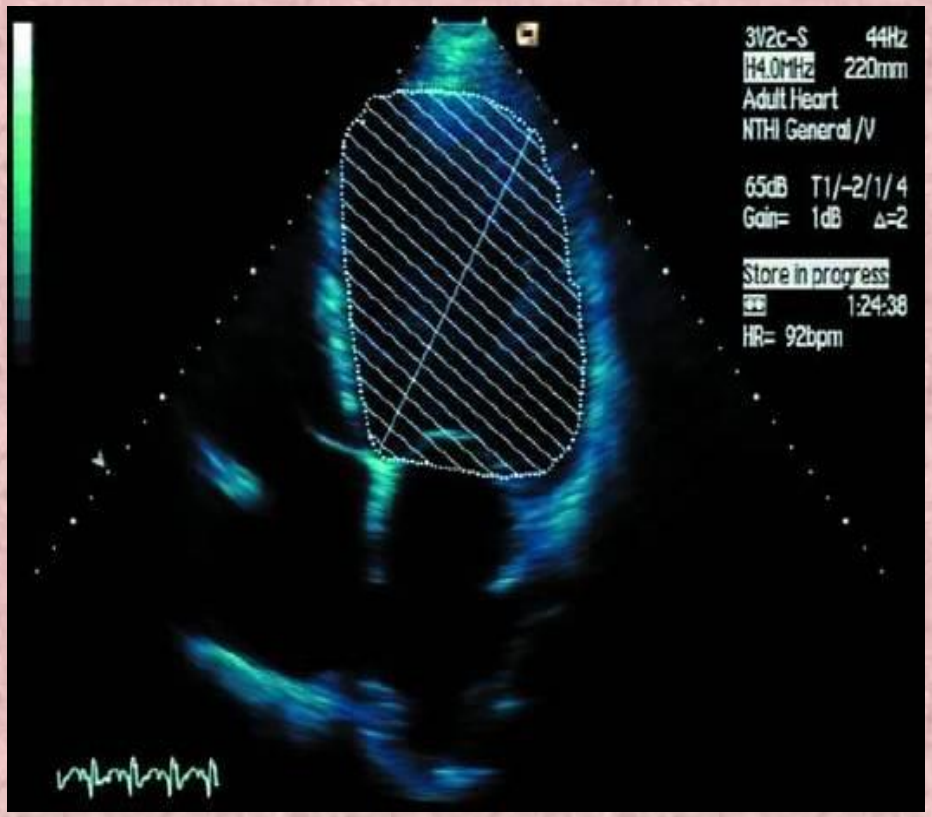
Echocardiography

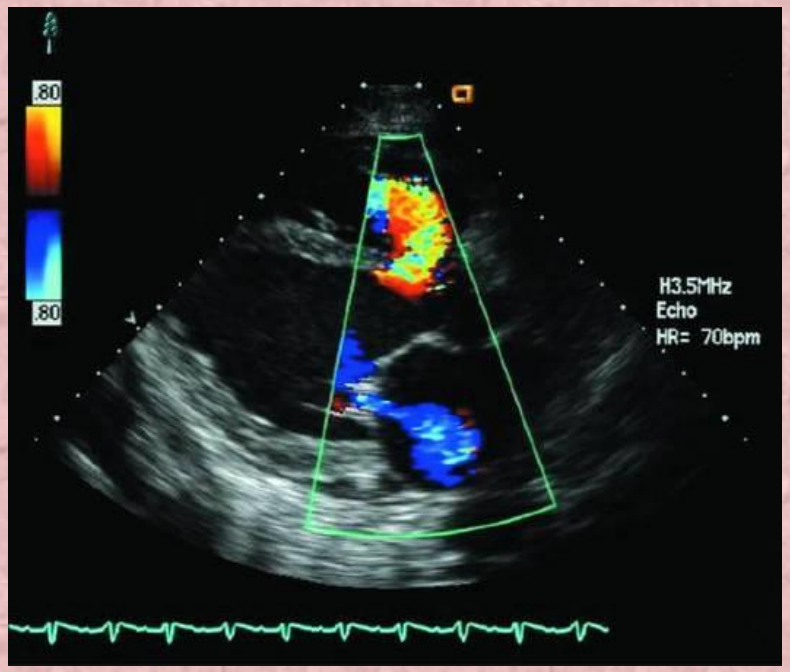
- The Doppler echocardiographic examination is regarded as the most useful test in evaluating patients with heart failure
- in establishing the type of cardiomyopathy (dilated, restrictive, hypertrophic)
- in evaluating the possible primary or secondary causes (valvular disease, LV aneurysm, intracardiac shunts) of heart failure
- provide the information about the size of all cardiac chambers and LV systolic function but also gives information about valvular function, stenotic or regurgitant lesions as well as reasonable estimates of both right- and left-sided pressures

- **Radionuclide Ventriculography**
- **Cardiac Catheterization**






3V2c-S
44Hz
H4.0MHz
220mm
Adult Heart
NTHI General /V
65dB T1/-2/1/4
Gain= 1dB Δ=2
Store in progress
1:24:38
HR= 92bpm


.80
.80
H3.5MHz
Echo
HR= 70bpm

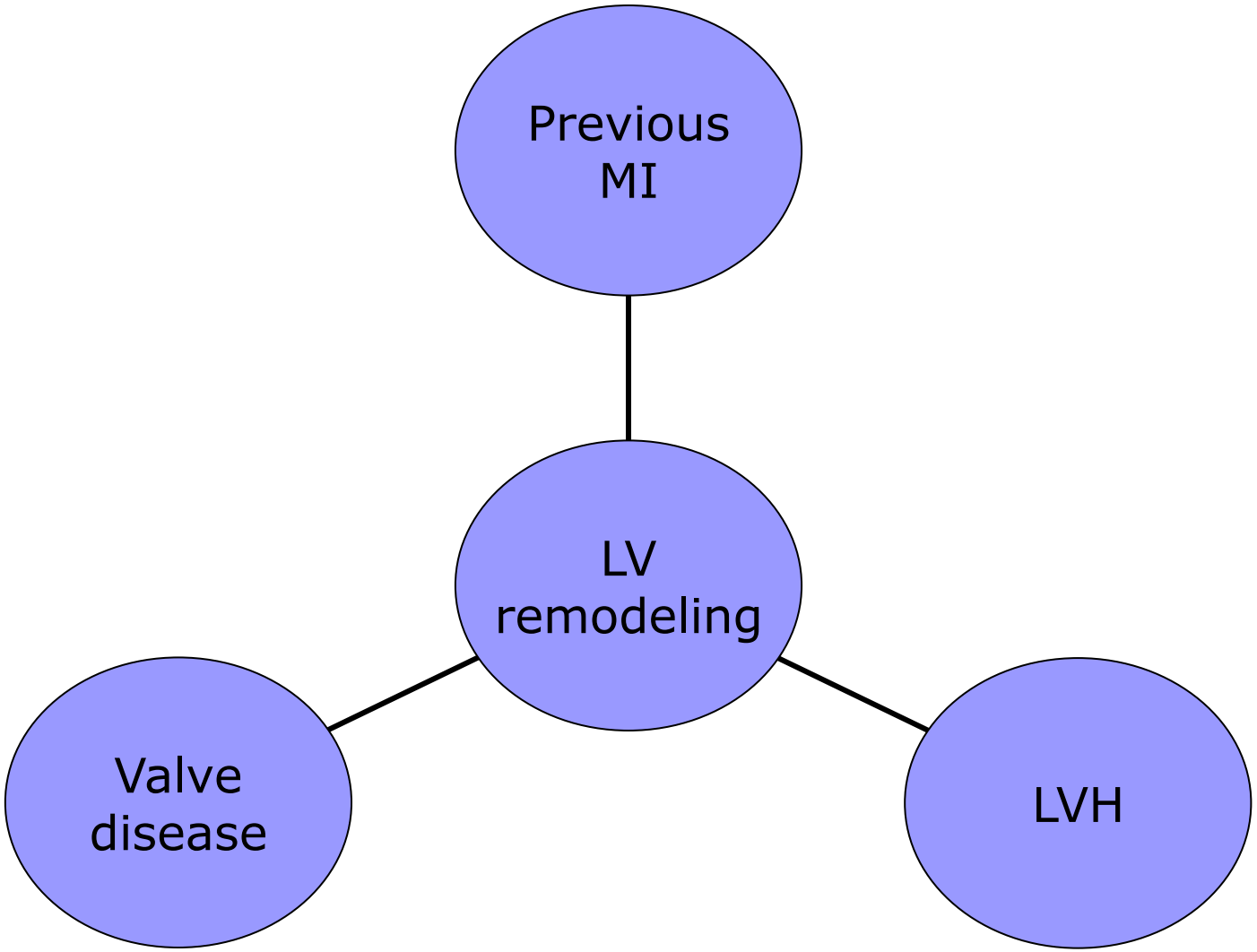
Goals of Therapy

- Improve symptoms and quality of life
- Slow the progression of cardiac and peripheral destruction
- Reduce mortality

Stage A: treatment

- Risk factor modification
- Diet and exercise plans
- Patient and family education
- Tobacco cessation
- Discourage alcohol and illicit drug use
- ACE inhibition in appropriate patient population

Stage B: patients with asymptomatic LV dysfunction



Stage B: therapy

- **General Measures as advised for Stage A**
- **Drug therapy for all patients**
  - **ACEI or ARBs**
  - **Beta-Blockers**
- **Coronary revascularization in appropriate patients**
- **Valve replacement or repair in appropriate patients**

Stage C: patients with past or current Symptoms of Heart Failure - Treatment

- **General measures as advised for Stages A and B**
- **Drug therapy for all patients**
  - **Diuretics for fluid retention**
  - **ACEI**
  - **Beta-blockers**
- **Drug therapy for selected patients**
  - **Aldosterone Antagonists**
  - **ARBs**
  - **Digitalis**
  - **Hydralazine/nitrates**
- **ICDs in appropriate patients**
- **Cardiac resynchronization in appropriate patients**
- **Exercise Testing and Training**

Drug	Initial Daily Dose(s)	Maximum Dose(s)
ACE Inhibitors		
Captopril	6.25 mg 3 times	50 mg 3 times
Enalapril	2.5 mg twice	10 to 20 mg twice
Fosinopril	5 to 10 mg once	40 mg once
Lisinopril	2.5 to 5 mg once	20 to 40 mg once
Perindopril	2 mg once	8 to 16 mg twice
Quinapril	5 mg twice	20 mg twice
Ramipril	1.25 to 2.5 mg once	10 mg once
Trandolapril	1 mg once	4 mg once
Angiotensin Receptor Blockers		
Candesartan	4 to 8 mg once	32 mg once
Losartan	25 to 50 mg once	50 to 100 mg once
Valsartan	20 to 40 mg twice	160 mg twice

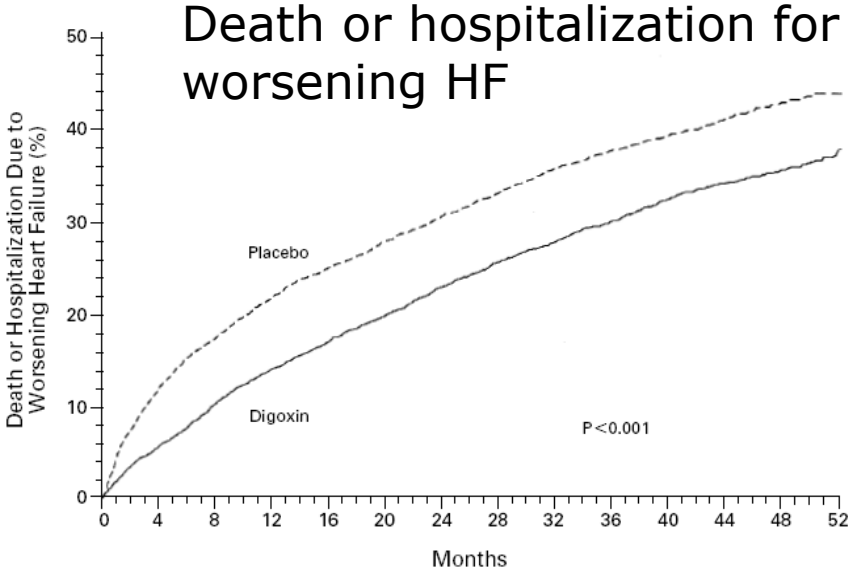
ACE indicates angiotensin converting enzyme; mg, milligrams; and kg, kilograms.

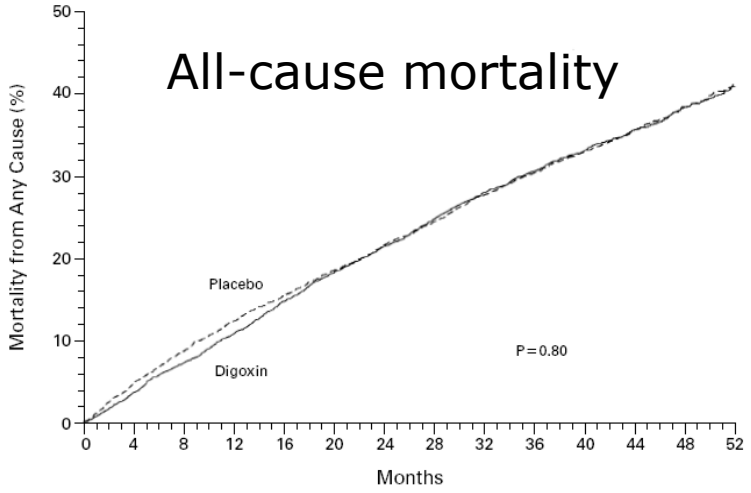
Starting and Target Doses for Beta-blockers in Heart Failure

Agent	Starting Dose	Target Dose <75kg	Target Dose >75kg
Metoprolol CR/XL	12.5-25mg po daily	200mg po daily	200mg po daily
Bisoprolol	1.25mg po daily	5mg po daily	10mg po daily
Carvedilol	3.125mg po twice daily	25mg po twice daily	50mg po twice daily

Digoxin: Improvement in symptoms but not survival

- Digitalis investigation group
- 6800 patients
- EF<45%
- Past or current symptoms of HF
- On ACEI and diuretics



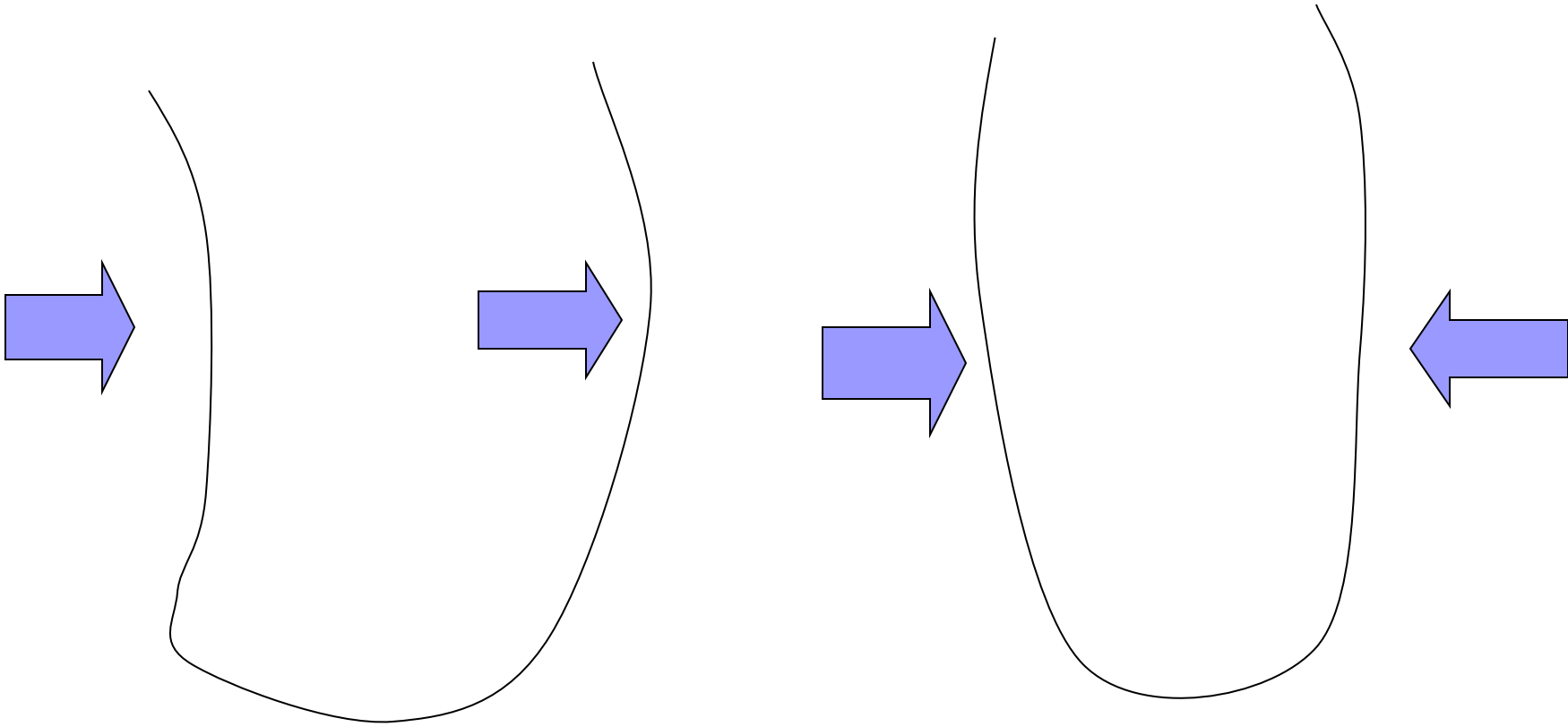


NEJM 1997;336:525.

ICDs for secondary prevention

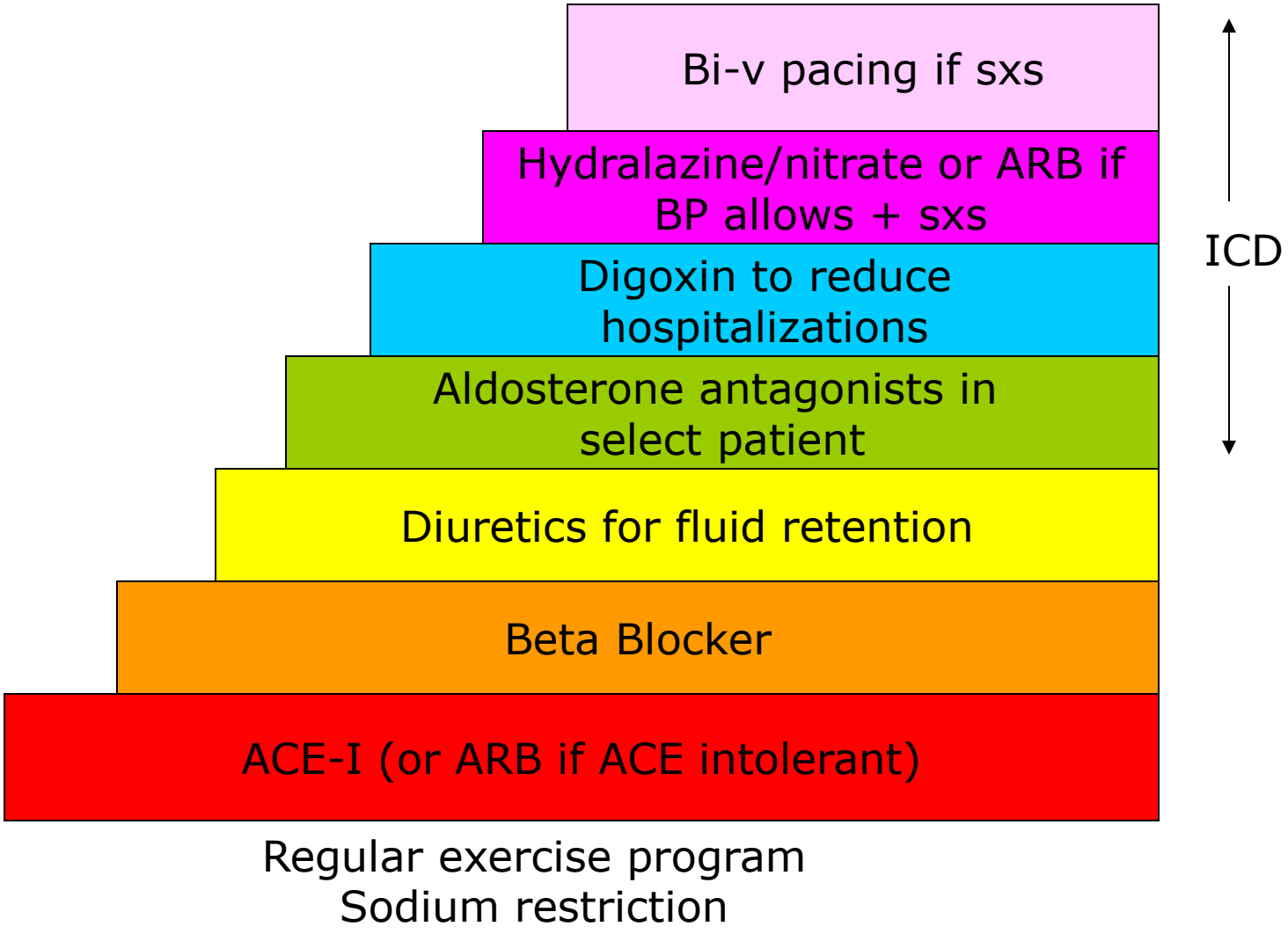
- IA: an ICD is recommended for survivors of cardiac arrest, VF or hemodynamically unstable VT who have low EF and current or prior symptoms

CRT: Cardiac Resynchronization Therapy



Additional recommendations

- Diuretics and salt restriction for fluid retention
- Routine exercise
- Treatment with warfarin if HF + AF, h/o TIA/CVA, h/o DVT/PE, recent anterior wall MI or MI with LV thrombus
- NSAIDS and calcium-channel blockers should be avoided
- Routine combination of ACE-I, ARB and aldosterone antagonist is not recommended


Bi-v pacing if sxs
Hydralazine/nitrate or ARB if BP allows + sxs
Digoxin to reduce hospitalizations
Aldosterone antagonists in select patient
Diuretics for fluid retention
Beta Blocker
ACE-I (or ARB if ACE intolerant)
ICD
Regular exercise program
Sodium restriction

Acute heart failure

- Pulmonary edema
- Cardiogenic Sock
- High-output heart failure with preserved EF (anemia, hipertirioidism, sepsis etc)

Criteria for cardiogenic shock

- 1.Systolic blood pressure of less than 90 mm Hg for at least 30 minutes
- 2. Pulmonary capillary wedge pressure (PCWP) of greater than 15 mm Hg
- 3. Cardiac index less than 2.2 L/min/m2

Causes of Cardiogenic Shock

- I**. Acute myocardial infarction (MI)**
- A. Pump failure
- B. Mechanical complications of acute MI
- 1. Acute mitral regurgitation
- 2. Ventricular septal defect
- 3. Free wall rupture/tamponade
- C. Right ventricular MI
- **II. End-stage, severe cardiomyopathies secondary to**
- A. Valvular disease
- B. Chronic ischemic disease
- C. Restrictive/infiltrative
- D. Idiopathic
- **III. Acute myocarditis: viral/infectious, toxic**
- **IV. Stress cardiomyopathy**
- **V. Endocrine disease (eg, hypothyroidism, pheochromocytoma**)
- A. Bradyarrhythmias
- B. Tachyarrhythmias
- **VII. Secondary to medications**
- **VIII. Post-traumatic**

Pathogenesis

- The principle feature of shock is hypotension with evidence of end-organ hypoperfusion
- In cardiogenic shock, this occurs as a consequence of inadequate cardiac function
- The usual response to low cardiac output is sympathetic stimulation to increase cardiac performance and maintain vascular tone

The pathophysiology of cardiogenic shock due to complications of acute MI

- Acute mitral regurgitation as a consequence of papillary muscle rupture
- Ventricular septal defect (VSD)
- Myocardial free wall rupture leading to cardiac tamponade

The physical examination reveals signs consistent with hypoperfusion

- **1. Vital Signs** – Hypotension is present (systolic blood pressure < 90 mm Hg). The heart rate is commonly elevated, and the respiratory rate is generally increased as a result of hypoxia from pulmonary congestion.

- **2. Neurologic** – Patients may be confused, lethargic, or obtunded as a consequence of cerebral hypoperfusion.

- **3. Pulmonary** – Patients may use accessory muscles of respiration and may have paradoxical respirations. The chest examination in most cases shows diffuse rales, often to the apices. Patients with isolated right ventricular infarction will not have pulmonary congestion.

- **4 Cardiovascular System** –
- Jugular venous pulsations are commonly elevated. Peripheral pulses will be weak
- A third or fourth heart sound suggesting significant left ventricular dysfunction and/or elevated filling pressures may be present
- A mitral regurgitation murmur (holosystolic, usually at the apex) or a VSD murmur (harsh, holosystolic at the sternal border) can help in diagnosing these causes. Patients with a free wall rupture that is partially contained may have a pericardial friction rub.
- Patients with significant right heart failure may have signs on abdominal examination of liver enlargement with a pulsatile liver in the presence of significant tricuspid regurgitation

Management of Cardiogenic Shock

A. Electrocardiogram

B. Chest radiography

C. Laboratory tests (complete blood count, coagulation profile, CK-MB, cardiac troponin, electrolytes + blood urea nitrogen/creatinine, arterial blood gases)

D. Echocardiography

E. Pulmonary artery catheterization (if diagnosis is in question, patient receiving inotropes/vasopressors, or patient is not responding to treatment)

F. Cardiac catheterization

Treatment

A. Oxygen supplementation; intubation, ventilation

B. Vasopressors/inotropes; consider careful intravenous fluids, arterial line and pulmonary artery catheter insertion to guide management; correct underlying causes of acidemia

C. Intra-aortic balloon pump, if needed

D. For suspected acute MI: aspirin, heparin, urgent cardiac catheterization, revascularization (PCI, CABG); fibrinolysis if a delay in PCI is anticipated

Inotropic/Vasopressor Agents

- A variety of drugs are available for intravenous administration to increase the contractility of the heart, the heart rate, and peripheral vascular tone
- It is important to note that these agents also increase myocardial oxygen demand; improvements in hemodynamics and blood pressure therefore come at a cost, which can be deleterious in patients with ongoing ischemia
- Furthermore, -agonists can precipitate tachyarrhythmias and -agonists can lead to dangerous vasoconstriction and ischemia in vital organ beds. When using these agents, attention should be given to the patient as a whole rather than focusing solely on a desired arterial pressure.

DIGOXIN

- Although digoxin benefits patients with chronic congestive heart failure, it is of less benefit in cardiogenic shock because of its delayed onset of action and relatively mild potency (compared with other available agents).

β-ADRENERGIC AGONISTS

- **Dopamine** is an endogenous catecholamine with qualitatively different effects at varying doses
- At low doses (< 3 mcg/kg/min), it predominantly stimulates dopaminergic receptors that dilate various arterial beds, the most important being the renal vasculature
- Intermediate doses of 3–6 mcg/kg/min cause 1-receptor stimulation and enhanced myocardial contractility
- Further increases in dosage lead to predominant - receptor stimulation and increases cardiac output, and its combination of cardiac stimulation and peripheral vasoconstriction may be beneficial as initial treatment of hypotensive patients in cardiogenic shock

Dobutamine is a synthetic sympathomimetic agent that differs from dopamine in two important ways: It does not cause renal vasodilatation, and it has a much stronger 2 (arteriolar vasodilatory) effect. The vasodilatory effect may be deleterious in hypotensive patients because a further drop in blood pressure may occur. On the other hand, many patients with cardiogenic shock experience excessive vasoconstriction with a resultant elevation in afterload (SVR) as a result of either the natural sympathetic discharge or the treatment with inotropic agents, such as dopamine, that also have prominent vasoconstrictor effects. In such patients, the combination of cardiac stimulation and decreased afterload with dobutamine may improve cardiac output without a loss of arterial pressure.

Isoproterenol is also a synthetic sympathomimetic agent. It has very strong chronotropic and inotropic effects, resulting in a disproportionate increase in oxygen consumption and ischemia. It is therefore not generally recommended for cardiogenic shock except occasionally for patients with bradyarrhythmias

Norepinephrine has even stronger and 1 effects than dopamine and may be beneficial when a patient continues to be hypotensive despite large doses of dopamine (more than 20 mcg/kg/min). Because of the intense peripheral vasoconstriction that occurs, perfusion of other vascular beds such as the kidney, extremities, and mesentery may be compromised. Therefore, norepinephrine should not be used for any extended time unless plans are made for definitive treatment.