Heart failure

LECTURE IN INTERNAL MEDICINE FOR V COURSE STUDENTS

M. Yabluchansky, L. Bogun, L. Martymianova, O. Bychkova, N. Lysenko, M. Brynza
V.N. Karazin National University Medical School' Internal Medicine Dept.

US MLE TEST

A 65-year-old man with a history of ischemic cardiomyopathy, congestive heart failure, diabetes mellitus, and chronic kidney disease presents to the emergency room with progressive dyspnea on exertion and weight gain for 8 days. Vitals signs are T 99.0, HR 110, BP 130/90, RR 24, SpO2 94% on room air. Physical examination reveals an S3 gallop, 2+ peripheral pitting edema, and marked jugular venous distention. Laboratory results show a serum creatinine of 1.2 mg/dL compared with the patient's normal baseline value of 1.1 mg/dL. The patient's chest radiograph is shown in Image A. A serum troponin is drawn and found to be 0.04 ng/mL. Which of the following medications is indicated first in the care of this patient?

1. Enalapril, 2. Furosemide, 3. Dobutamine, 4. Hydralazine, 5. Dopamine

US MLE TEST

Correct Answer 2: This patient is volume-overloaded and requires a diuretic to decrease preload to his heart. Furosemide, a loop-diuretic, will decrease preload and is the best choice. (Note that the other drugs listed are used for chronic CHF management and/or in the case of CHF refractory to furosemide treatment.)

Incorrect Answers:

1: Enalapril is an ACE inhibitor that decreased mortality when used in the treatment of systolic heart failure. It is not the most appropriate first line agent for ADHF in this patient., 3: Dobutamine is sympathomimetic used in the treatment of heart failure, but furosemide is a more appropriate first line agent in this patient., 4: Hydralazine is used in the treatment of hypertension and is not the most appropriate first line agent in the treatment of this patient., 5: Dopamine is an ionotropic and chronotropic drug used in the treatment of heart failure. However, furosemide is a more appropriate first line agent in this patient.

Plan of the Lecture

- Definition
- Epidemiology
- Risk factors
- Etiology
- Mechanisms
- Classification
- Clinical investigation
- Diagnosis
- Treatment
- Prognosis
- Prophylaxis
- Abbreviations
- Diagnostic and treatment guidelines
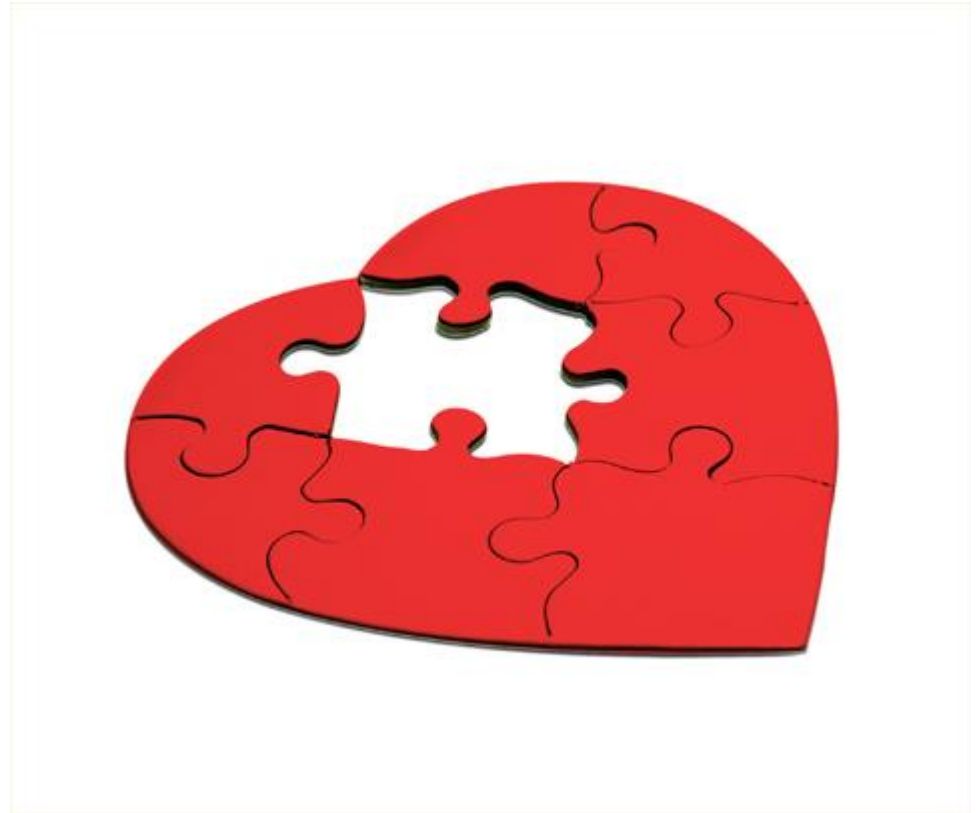
Definition 1

- (Congestive) heart failure (HF) is a clinical syndrome caused by a structural and/or functional cardiac abnormality, resulting in a reduced cardiac output and/or elevated intracardiac pressures at rest or during stress, characterized by typical symptoms (e.g. breathlessness, ankle swelling and fatigue) that may be accompanied by signs (e.g. elevated jugular venous pressure, pulmonary crackles and peripheral oedema).

Definition 2

- Before clinical symptoms become apparent, patients can present with asymptomatic structural or functional cardiac abnormalities (asymptomatic systolic left ventricle (LV) dysfunction), which recognition as precursors of HF is important for starting its treatment as soon as possible in deal to reduce patients mortality.

Epidemiology
(Hear Failure around the World) 1

- The prevalence of HF depends on the definition applied, but is approximately 1–2% of the adult population in developed countries, rising to ≥10% among people >70 years of age.

- Among people >65 years of age presenting to primary care with breathlessness on exertion, one in six will have unrecognized HF (mainly HFpEF).

Epidemiology
(Hear Failure around the World) 2

- The lifetime risk of HF at age 55 years is 33% for men and 28% for women.

- The proportion of patients with HFpEF ranges from 22 to 73%, depending on the definition applied, the clinical setting (primary care, hospital clinic, hospital admission), age and sex of the population, previous myocardial infarction and the year of publication.

Epidemiology
(The Heart Failure Epidemic)
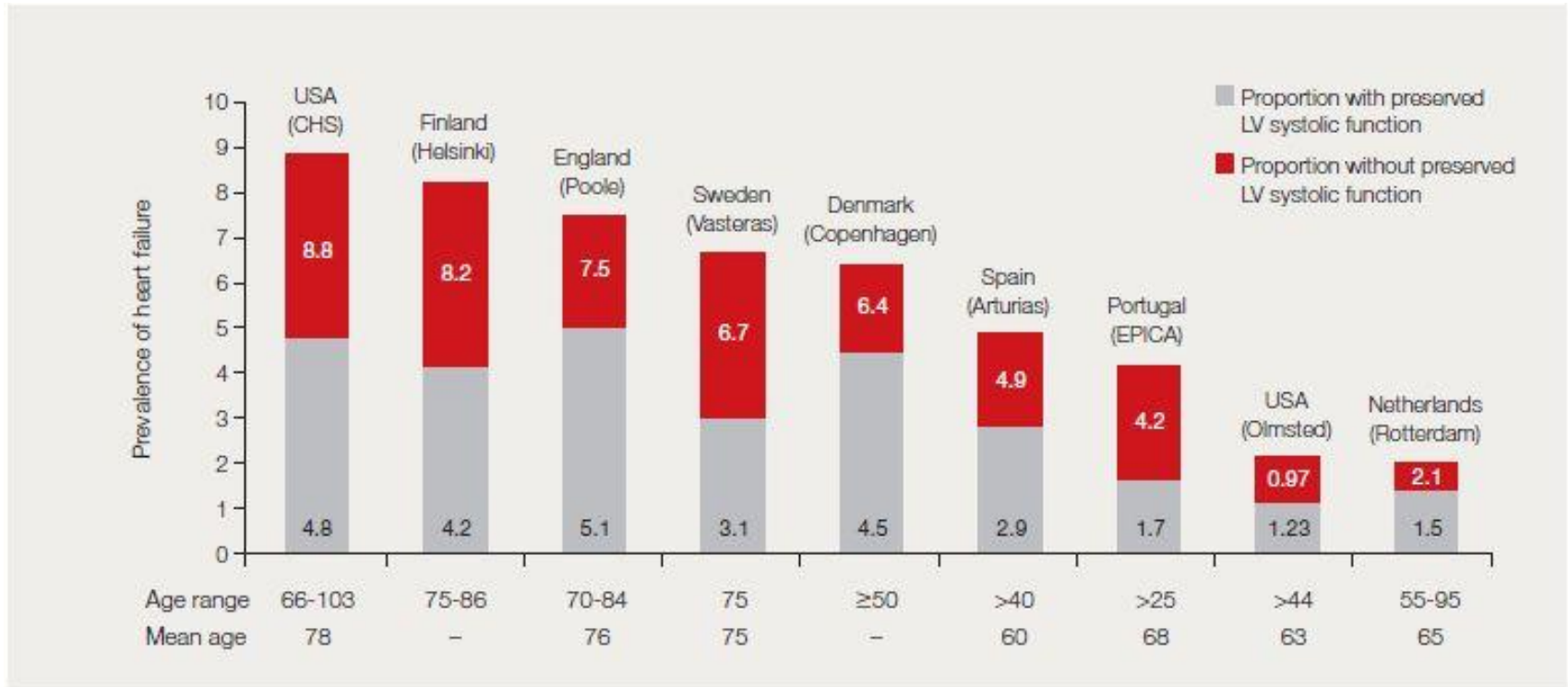
The relative proportion of cases with (grey bars) and without (red bars) preserved LV systolic function.

Risk Factors and Etiology
(Underlying Causes of Systolic Heart Failure) 1

- Coronary artery disease

- Diabetes mellitus

- Hypertension

- Valvular heart disease (stenosis or regurgitant lesions)

- Arrhythmia (supraventricular or ventricular)

- Infections and inflammation (myocarditis)
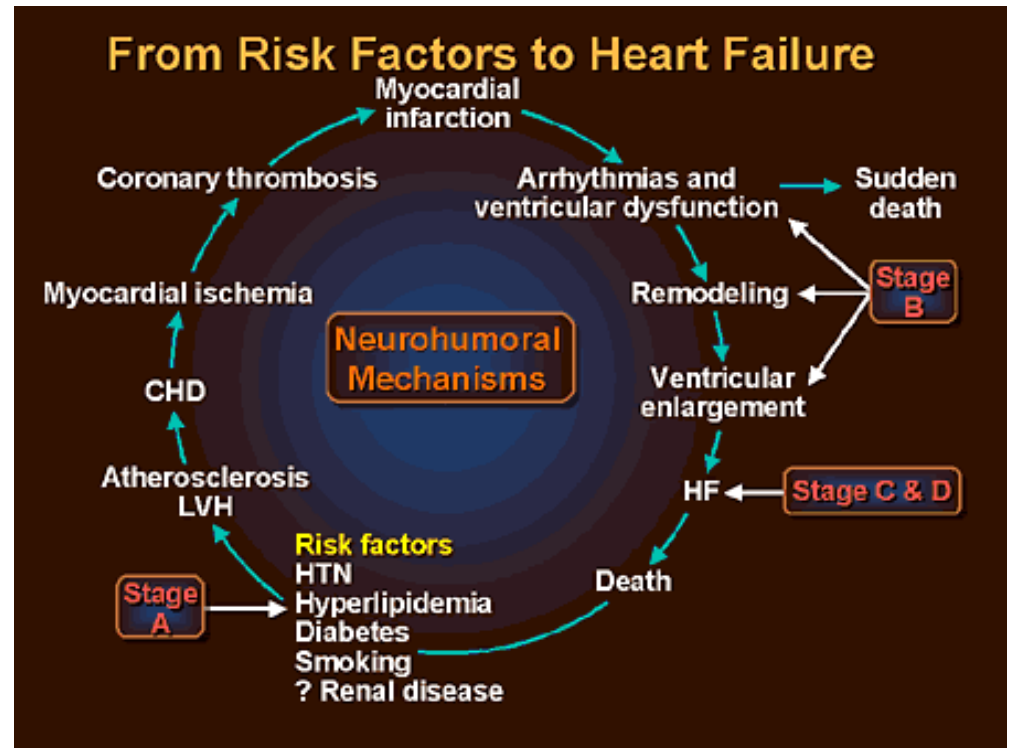
Risk Factors and Etiology
(Underlying Causes of Systolic Heart Failure) 2

- Peripartum cardiomyopathy

- Congenital heart disease

- Drugs (either recreational, such as alcohol and cocaine, or therapeutic drugs with cardiac side effects, such as doxorubicin)

- Idiopathic cardiomyopathy

- Rare conditions (endocrine abnormalities, rheumatologic disease, neuromuscular conditions)

Risk Factors and Etiology
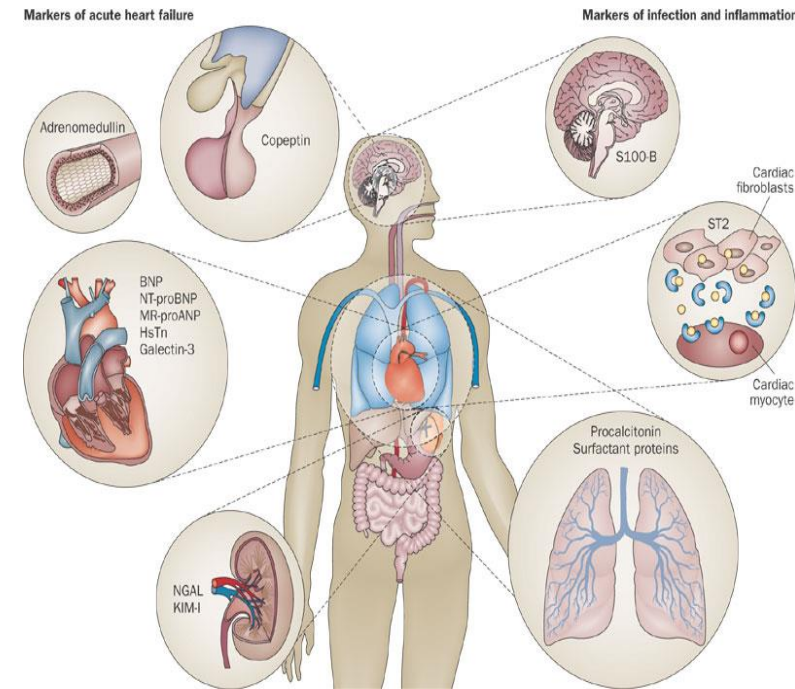## (Underlying Causes of Diastolic Heart Failure)

- Coronary artery disease
- Diabetes mellitus
- Hypertension
- Valvular heart disease
- Hypertrophic cardiomyopathy
- Restrictive cardiomyopathy
- Constrictive pericarditis

From Risk Factors to Heart Failure

Risk Factors and Etiology
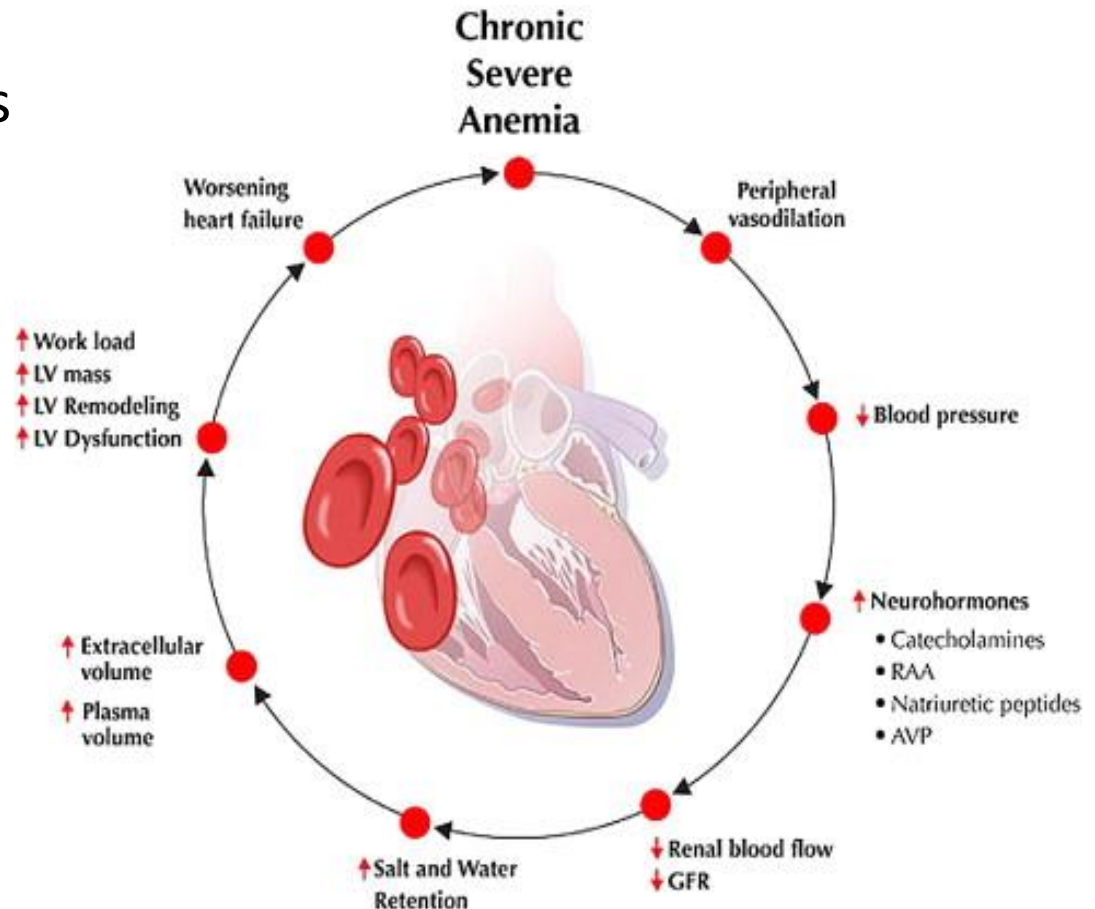## (Underlying Causes of Acute Heart Failure)

- Acute valvular regurgitation

- Myocardial infarction

- Myocarditis

- Arrhythmia

- Drugs (e.g., cocaine, calcium channel blockers, or beta-blocker overdose)

- Sepsis

Risk Factors and Etiology
## (Underlying Causes of High-Output Heart Failure)

- Anemia

- Systemic arteriovenous fistulas

- Hyperthyroidism

- Beriberi heart disease

- Paget disease of bone

- Fibrous dysplasia

- Multiple myeloma

- Pregnancy

- Glomerulonephritis

- Polycythemia vera

- Carcinoid syndrome

Chronic Severe Anemia

↑Work load
↑LV mass
↑LV Remodeling
↑LV Dysfunction

Worsening heart failure

Peripheral vasodilation

↓Blood pressure

↑Neurohormones
• Catecholamines
• RAA
• Natriuretic peptides
• AVP

↑Extracellular volume
↑Plasma volume

↑Salt and Water Retention

↓Renal blood flow
↓GFR

Risk Factors and Etiology
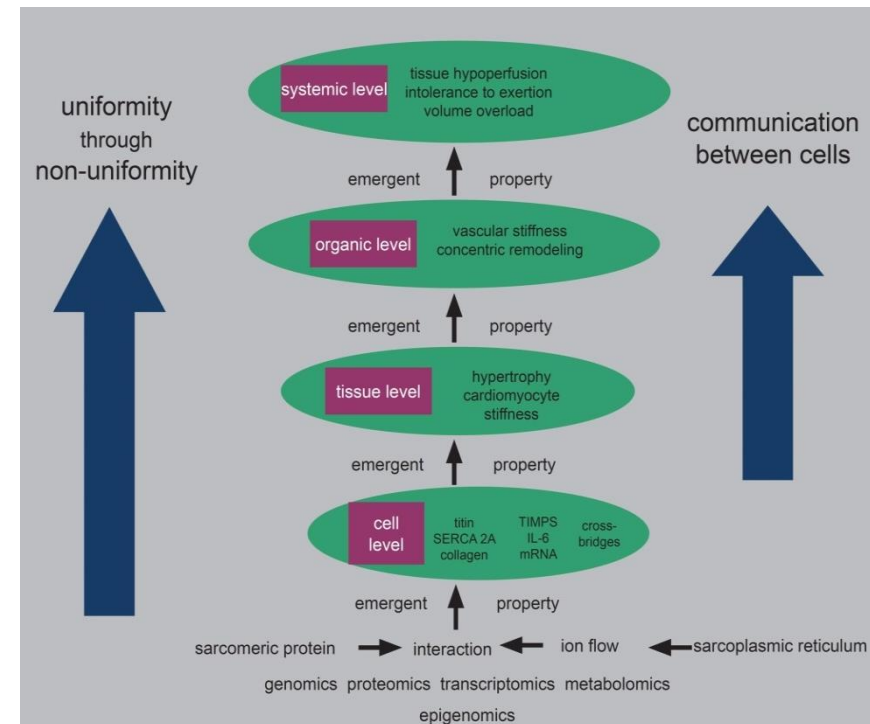## (Underlying Causes of Right Heart Failure)

- Left ventricular failure
- Coronary artery disease (ischemia)
- Pulmonary hypertension
- Pulmonary valve stenosis
- Pulmonary embolism
- Chronic pulmonary disease
- Neuromuscular disease

Left atrium

Right atrium

Enlarged right ventricle

Left ventricle
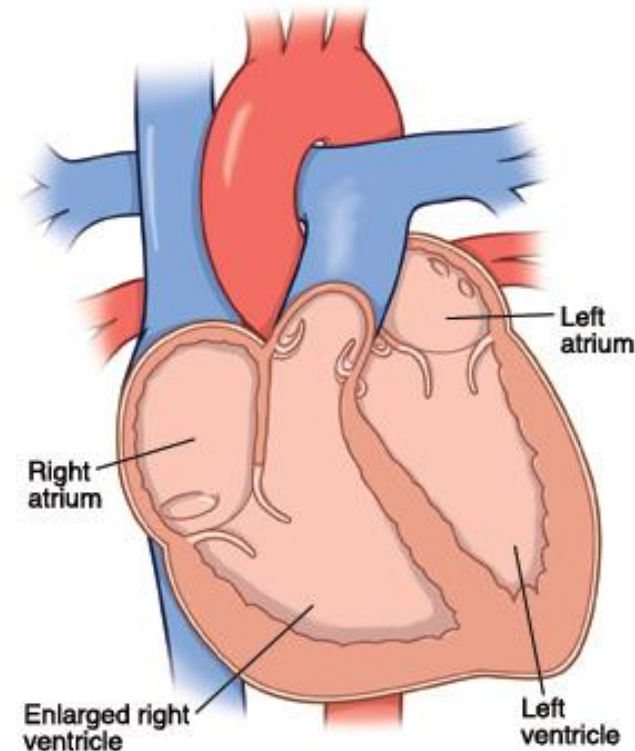
Risk Factors and Etiology
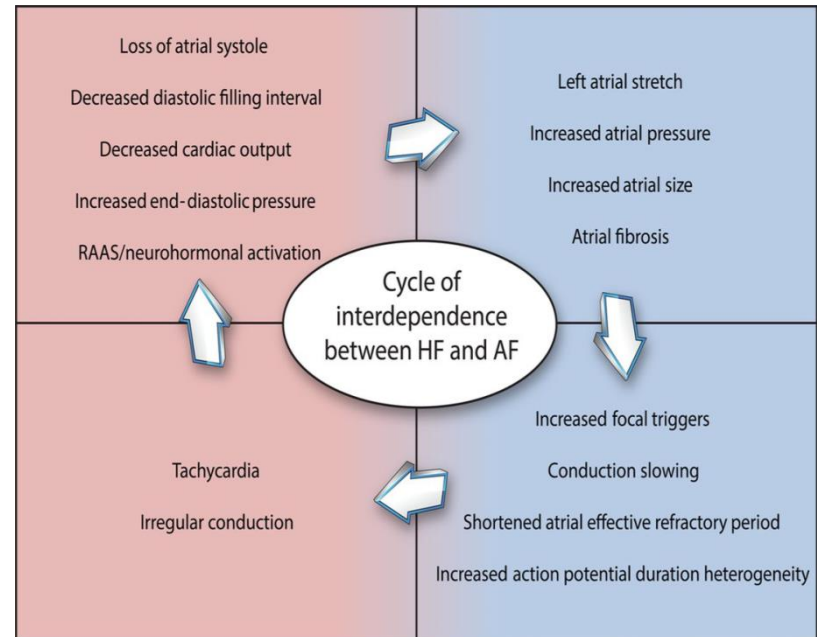## (Fundamental Causes of Heart Failure)

Fundamental causes include the biological mechanisms, through which either an increased hemodynamic burden or a reduction in oxygen delivery to the myocardium results in impairment of myocardial contraction.

Risk Factors and Etiology
## (Precipitating Causes of Heart Failure)

Overt heart failure may be precipitated by progression of the underlying heart disease (e.g., further narrowing of a stenotic aortic valve or mitral valve) or various conditions (fever, anemia, infection) or medications (chemotherapy, NSAIDs).

Loss of atrial systole
Decreased diastolic filling interval
Decreased cardiac output
Increased end-diastolic pressure
RAAS/neurohormonal activation

Left atrial stretch
Increased atrial pressure
Increased atrial size
Atrial fibrosis

Cycle of interdependence between HF and AF

Increased focal triggers
Conduction slowing
Shortened atrial effective refractory period
Increased action potential duration heterogeneity

Tachycardia
Irregular conduction

Risk Factors and Etiology
(Genetic Causes of Heart Failure)

- Dilated cardiomyopathy

- Arrhythmic cardiomyopathy

- Right ventricular cardiomyopathy

- Restrictive cardiomyopathy.
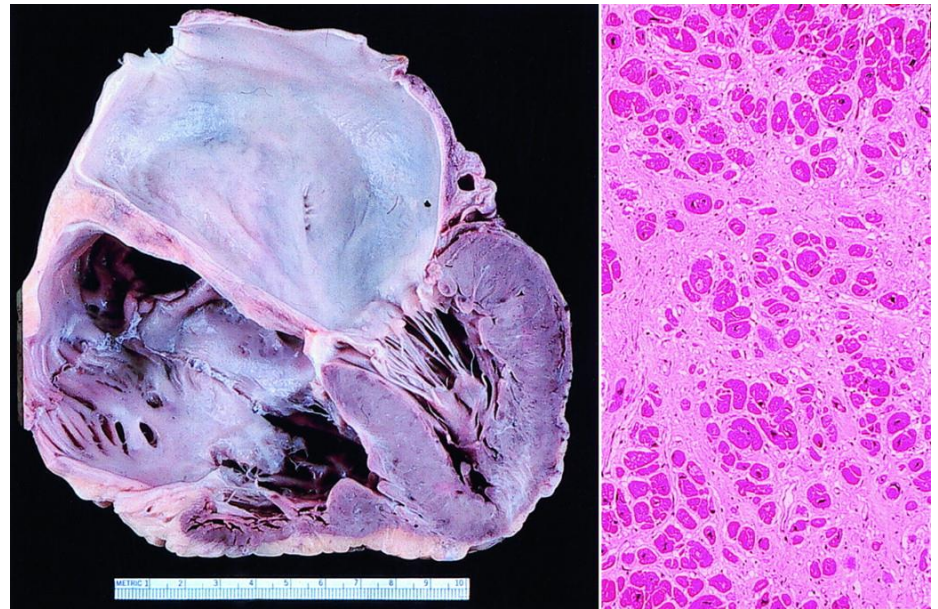
Idiopathic Restrictive Cardiomyopathy, marked interstitial fibrosis

US MLE TEST

A 60-year-old Caucasian male presents to your office complaining of shortness of breath on exertion. He undergoes an echocardiogram and is found to have an ejection fraction of 35%. Which of the following classes of drugs would improve mortality in this patient?

1. Cardiac glycosides
2. Calcium channel blockers
3. Thiazide diuretics
4. ACE inhibitors
5. Nitrates

US MLE TEST

Correct Answer 4: The patient in the question stem has systolic heart failure as evidenced by his decreased ejection fraction. Beta blockers, angiotensin converting enzyme (ACE) inhibitors, and angiotensin receptor blockers (ARBs) have been shown to decrease mortality in the treatment of systolic heart failure. Spironolactone and eplerenone have additionally been shown to provide a mortality benefit.

Incorrect Answers:

Although all of these drug classes may provide symptomatic relief in the treatment of systolic heart dysfunction, of the choices listed, only ACE inhibitors improve mortality.

Mechanisms
(The Common Pathophysiologic State)   1

- The heart in HF may have a reduced force of contraction due to overloading of the ventricle with fails the Frank–Starling law.

- A reduced stroke volume (SV) may occur as a result of a failure of systole, diastole or both, that contributes to the exercise intolerance commonly seen in HF.

Mechanisms
(The Common Pathophysiologic State)   2

- A common finding in HF is an increased sympathetic and renin-angiotensin-aldosterone system (RAAS) activity with other neurohumoral adjustments that leads to salt and water retention, resulting in deep disturbances of heart function and structure with enlargement of the ventricles and their remodeling.

Mechanisms
(The Common Pathophysiologic State)   3

- This increases the risk of cardiac arrest (specifically due to abnormal ventricular heart rhythms), and reduces blood supply to the rest of the body.

Mechanisms
(Myocytes and Myocardial Remodeling) 1

- In the HF, increased myocardial volume is characterized by larger myocytes approaching the end of their life cycle and as more myocytes drop out, an increased load is placed on the remaining myocardium, and this unfavorable environment is transmitted to the progenitor cells responsible for replacing lost myocytes.

Mechanisms
(Myocytes and Myocardial Remodeling) 2

- Progenitor cells become progressively less effective as the underlying pathologic process worsens and myocardial failure accelerates.

- This results in cellular proliferation, adverse myocardial remodeling, and antinatriuresis, with total body fluid excess and worsening of heart failure symptoms.

Mechanisms
(Left Ventricle Stiffness)   1

- An increase in LV stiffness occurs secondary to any one of, or any combination of, the following 3 mechanisms:
  - rise in filling pressure,
  - shift to a steeper ventricular pressure-volume curve,
  - decrease in ventricular distensibility.

Mechanisms
(Left Ventricle Stiffness)   2

- A shift to a steeper ventricular pressure-volume curve results, most commonly, not only from increased ventricular mass and wall thickness but also from infiltrative, endomyocardial fibrosis, and myocardial ischemia.

- Parallel upward displacement of the diastolic pressure-volume curve is generally referred a decrease in ventricular distensibility that caused by extrinsic compression of the ventricles.

Mechanisms
(Concentric Left Ventricle Hypertrophy) 1

- Pressure overload that leads to concentric LV hypertrophy (LVH), shifts the diastolic pressure-volume curve to the left along its volume axis and ventricular diastolic pressure is abnormally elevated, although chamber stiffness may or may not be altered.

Mechanisms
(Concentric Left Ventricle Hypertrophy) 2

- Increases in diastolic pressure lead to increased myocardial energy expenditure, remodeling of the ventricle, increased myocardial oxygen demand, myocardial ischemia, and eventual progression of the maladaptive mechanisms of the heart that lead to decompensated HF.

Mechanisms
(Systolic dysfunction)

- HF caused by systolic dysfunction is characterized by a decreased ejection fraction (less than 45%).

- The strength of ventricular contraction is attenuated and inadequate for creating an adequate stroke volume, resulting in inadequate cardiac output.

- Because the ventricle is inadequately emptied, ventricular end-diastolic pressure and volumes increase and this is transmitted to the atrium.

- On the left side of the heart it causing pulmonary edema and on the right side of the heart it resulting in dependent peripheral edema.

Mechanisms
(Diastolic dysfunction) 1

- HF caused by diastolic dysfunction is generally described as the backward failure of the ventricle to adequately relax and typically denotes a stiffer ventricular wall with inadequate filling of the ventricle, and therefore inadequate SV.

Mechanisms
(Diastolic dysfunction) 2

- Diastolic dysfunction can be caused by processes similar to those that cause systolic dysfunction, particularly causes that affect cardiac remodeling.

- The patient may be completely asymptomatic at rest, but is exquisitely sensitive to increases in heart rate, and sudden bouts of tachycardia may result in flash pulmonary edema.

Mechanisms
(Arrhythmias)   1

- Arrhythmia imparts a significant burden in all forms of HF.

- The most significant of all rhythms associated with HF are the life-threatening ventricular arrhythmias.

- Structural substrates for ventricular arrhythmias regardless of the underlying cause, include ventricular dilatation, myocardial hypertrophy, and myocardial fibrosis.
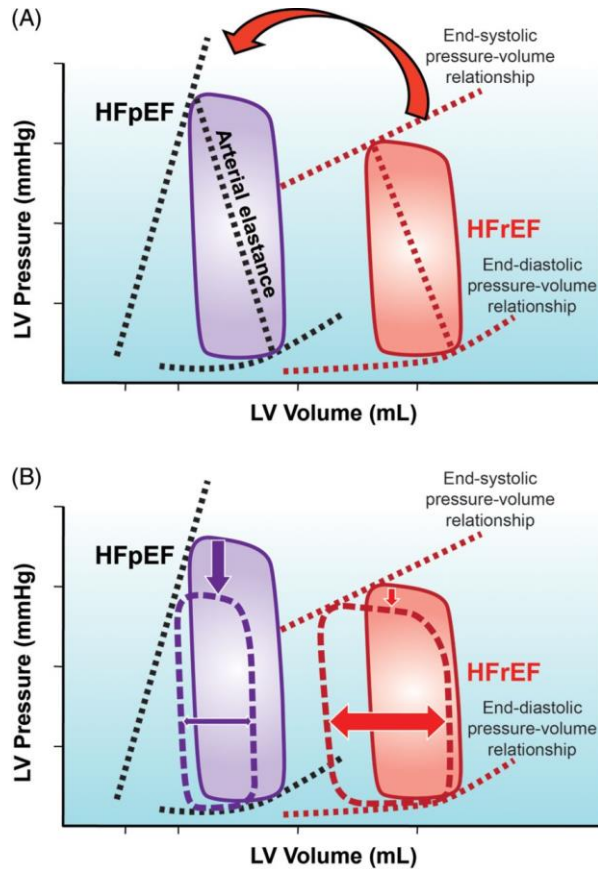
Mechanisms
(Arrhythmias)   2

- At the cellular level, myocytes may be exposed to increased stretch, wall tension, catecholamines, ischemia, and electrolyte imbalance.

- The combination of these factors contributes to an increased incidence of arrhythmogenic sudden cardiac death in patients with heart failure.
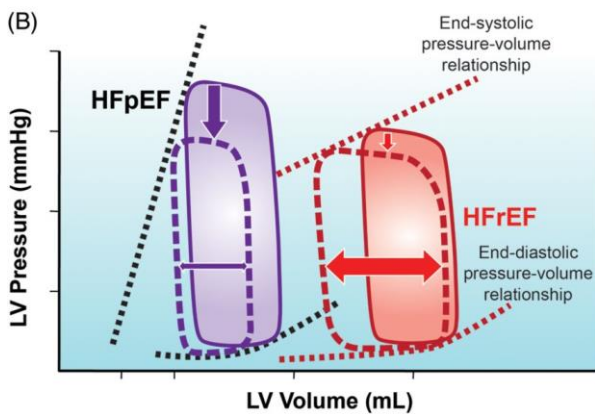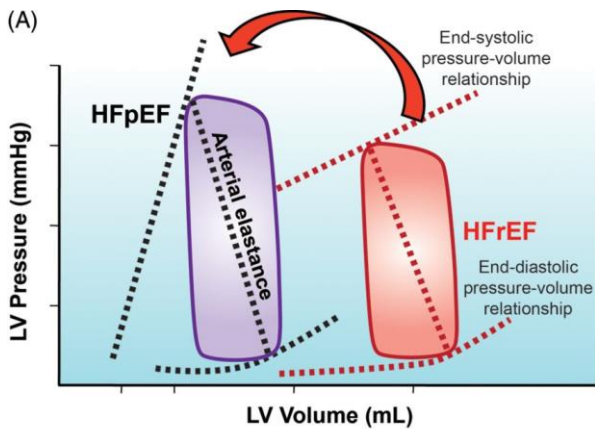
Mechanisms
(Pressure–Volume Loops in Heart Failure) 1

- Pressure–volume loop (PVL)characteristics in HF with preserved (black) ejection fraction (EF)and HF with reduced (red) EF in baseline conditions (*A*), and in response to vasodilators (*B*).

- (*A*) Curved arrow depicts the steeper end-systolic pressure–volume relationship in HF with preserved EF compared with HF with reduced EF.
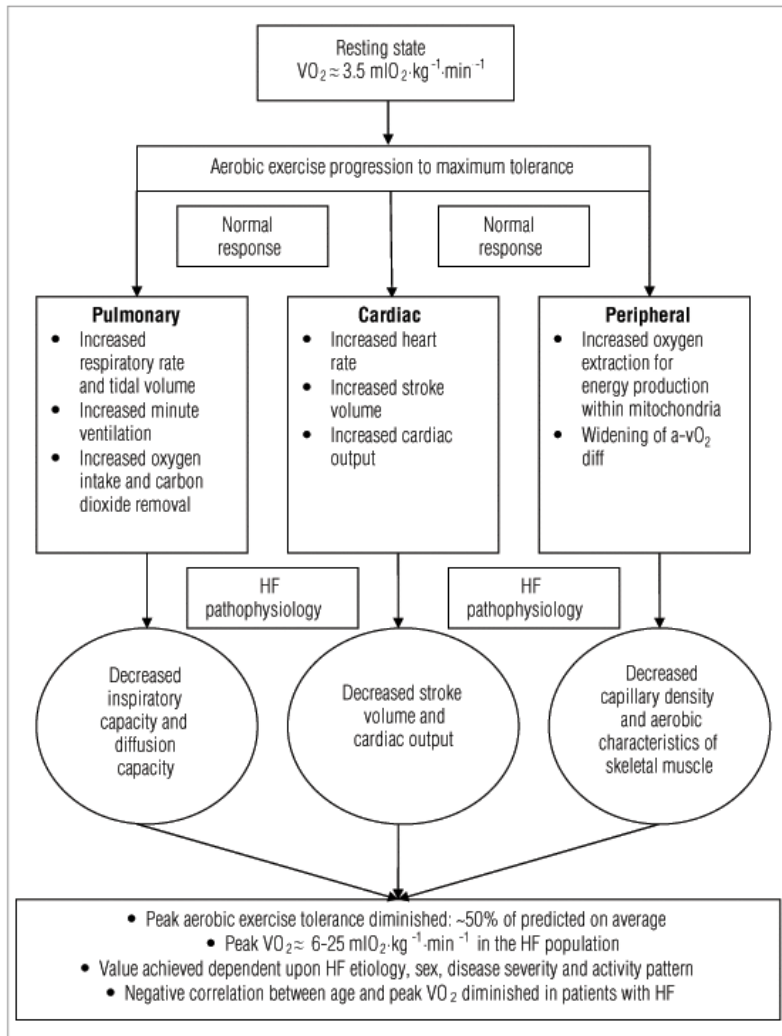
Mechanisms
(Pressure–Volume Loops in Heart Failure) 2

- (*B*) PVL before (solid) and after (dotted) administration of vasodilators.

- Arrows contrast the drop in blood pressure and changes in stroke volume between HF with preserved EF and HF with reduced EF in response to vasodilators.
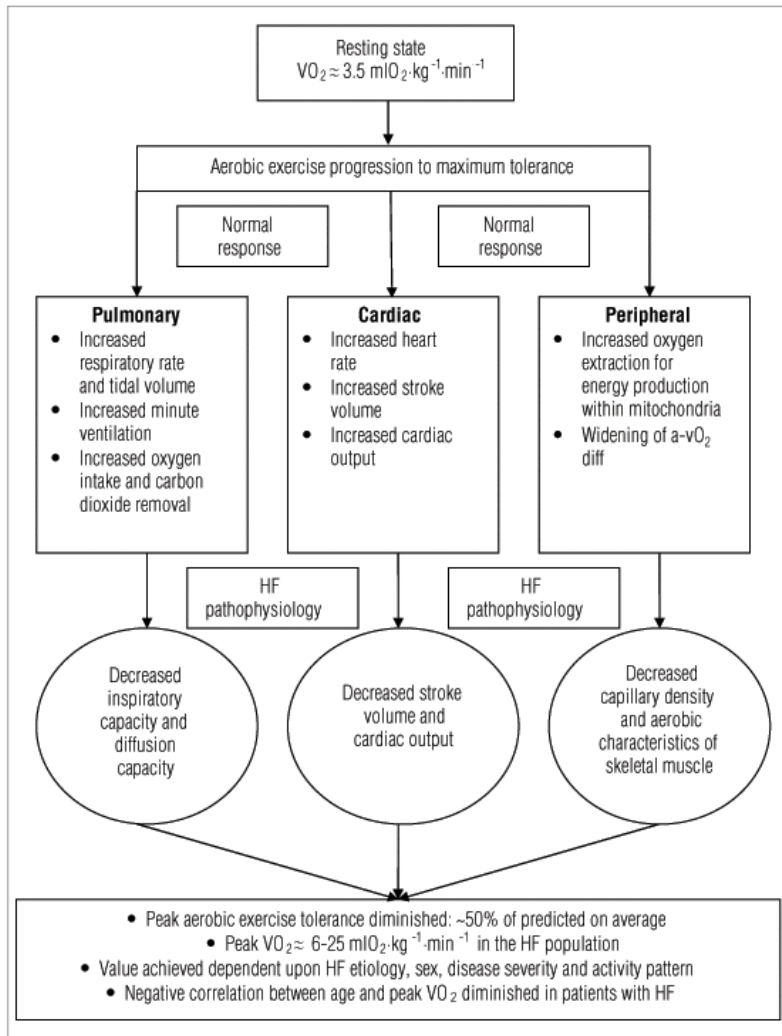
Mechanisms

(HF affects the Systems involved in the Physiological Response) 1

- Severely compromised cardiac function is a primary pathophysiological component in HF.

Mechanisms

(HF affects the Systems involved in the Physiological Response) 2

- Patients with HF frequently present reduced capillary density and intrinsic skeletal muscle abnormalities, primarily in the form of diminished aerobic (mitochondrial) function.

US MLE TEST

A 68-year-old male suffered a myocardial infarction two weeks ago. Since this incident, he has reported increased shortness of breath with both activity as well as when lying flat; he has also noted increased swelling in his ankles. Physical exam is significant for an S3 gallop on cardiac auscultation, bibasilar crackles on lung auscultation, and 2+ edema of the bilateral ankles. Which of the following sets of cardiac parameters would most likely be associated with this patient's current condition? (Cardiac index = CI; Systemic vascular resistance = SVR; Left ventricular end diastolic pressure = LVEDP)

1. Decreased CI, decreased SVR, increased LVEDP, 2. Decreased CI, increased SVR, decreased LVEDP, 3. Decreased CI, increased SVR, increased LVEDP, 4. Increased CI, decreased SVR, increased LVEDP, 5. Increased CI, increased SVR, decreased LVEDP

US MLE TEST

Correct Answer 3: This patient is suffering from systolic heart failure. He would be expected to have a decreased cardiac index, increased systemic vascular resistance, and increased left ventricular end diastolic pressure.

Incorrect Answers:

1: Decreased systemic vascular resistance occurs due to systemic vasodilation, which may be a sequela of sepsis (most common), pancreatitis, cirrhosis, adrenal insufficiency, head injury, or beriberi., 2: Decreased LVEDP occurs in mitral stenosis., 4 & 5: Increased cardiac index may be seen in patients suffering from high-output cardiac failure; causes of this may include AV fistula, anemia, hyperthyroidism, beriberi, renal disease, hepatic disease, or sepsis.

Classification
(International Classification of Diseases (ICD))

Chapter IX
Other forms of heart disease
(I30-I52**)**

      150 Heart failure

          I50.0 Congestive heart failure, right ventricular failure

          I50.1Left ventricular failure

          I50.9 Heart failure, unspecified

Classification
(New York Heart Association Functional Classification)

NYHA Class	Symptoms
I	Cardiac disease, but no symptoms and no limitation in ordinary physical activity, e.g. no shortness of breath when walking, climbing stairs etc.
II	Mild symptoms (mild shortness of breath and/or angina) and slight limitation during ordinary activity.
III	Marked limitation in activity due to symptoms, even during less-than-ordinary activity, e.g. walking short distances (20–100 m). Comfortable only at rest.
IV	Severe limitations. Experiences symptoms even while *at rest*. Mostly bedbound patients.

Classification
(Stages of Heart Failure)

HF Stages	Symptoms
A	No objective evidence of cardiovascular disease. No symptoms and no limitation in ordinary physical activity.
B	Objective evidence of minimal cardiovascular disease. Mild symptoms and slight limitation during ordinary activity. Comfortable at rest.
C	Objective evidence of moderately severe cardiovascular disease. Marked limitation in activity due to symptoms, even during less-than-ordinary activity. Comfortable only at rest.
D	Objective evidence of severe cardiovascular disease. Severe limitations. Experiences symptoms even while at rest.

Classification
(Killip classification for Acute Myocardial Infarction)

Killip Class	Symptoms
I	Individuals with no clinical signs of heart failure (mortality 6%).
II	Individuals with rales or crackles in the lungs, an S_3, and elevated jugular venous pressure (mortality 17%).
III	Individuals with frank acute pulmonary edema (mortality 38%).
IV	Individuals in cardiogenic shock or hypotension, and evidence of peripheral vasoconstriction (mortality 67%).

Clinical Investigation
(Signs and Symptoms) 1

- Exertional dyspnea and/or dyspnea at rest.

- Orthopnea.

- Acute pulmonary edema.

- Chest pain/pressure and palpitations.

- Tachycardia.

- Fatigue and weakness.

- Nocturia and oliguria.

- Anorexia, weight loss, nausea.

Clinical Investigation
(Signs and Symptoms) 2

- Exophthalmos and/or visible pulsation of eyes.

- Distention of neck veins.

- Weak, rapid, and thready pulse.

- Rales, wheezing.

- S_3 gallop and/or pulsus alternans

- Increased intensity of P_2 heart sound.

- Hepatojugular reflux.

- Ascites, hepatomegaly, and/or anasarca.

- Central or peripheral cyanosis, pallor.

Clinical Investigation
(Price of Signs and Symptoms) 1

- Symptoms are often non-specific and do not help discriminate between HF and other problems.

- Symptoms due to fluid retention may resolve quickly with diuretic therapy.

Symptoms	Signs
Typical	**More specific**
Breathlessness Orthopnoea Paroxysmal nocturnal dyspnoea Reduced exercise tolerance Fatigue, tiredness, increased time to recover after exercise Ankle swelling	Elevated jugular venous pressure Hepatojugular reflux Third heart sound (gallop rhythm) Laterally displaced apical impulse
Less typical	**Less specific**
Nocturnal cough Wheezing Bloated feeling Loss of appetite Confusion (especially in the elderly) Depression Palpitations Dizziness Syncope Bendopnea[53]	Weight gain (>2 kg/week) Weight loss (in advanced HF) Tissue wasting (cachexia) Cardiac murmur Peripheral oedema (ankle, sacral, scrotal) Pulmonary crepitations Reduced air entry and dullness to percussion at lung bases (pleural effusion) Tachycardia Irregular pulse Tachypnoea Cheyne Stokes respiration Hepatomegaly Ascites Cold extremities Oliguria Narrow pulse pressure

Clinical Investigation
(Price of Signs and Symptoms) 2

- Signs, such as elevated jugular venous pressure and displacement of the apical impulse, may be more specific, but are harder to detect and have poor reproducibility.

- Etc.

Symptoms	Signs
Typical	**More specific**
Breathlessness Orthopnoea Paroxysmal nocturnal dyspnoea Reduced exercise tolerance Fatigue, tiredness, increased time to recover after exercise Ankle swelling	Elevated jugular venous pressure Hepatojugular reflux Third heart sound (gallop rhythm) Laterally displaced apical impulse
Less typical	**Less specific**
Nocturnal cough Wheezing Bloated feeling Loss of appetite Confusion (especially in the elderly) Depression Palpitations Dizziness Syncope Bendopnea[53]	Weight gain (>2 kg/week) Weight loss (in advanced HF) Tissue wasting (cachexia) Cardiac murmur Peripheral oedema (ankle, sacral, scrotal) Pulmonary crepitations Reduced air entry and dullness to percussion at lung bases (pleural effusion) Tachycardia Irregular pulse Tachypnoea Cheyne Stokes respiration Hepatomegaly Ascites Cold extremities Oliguria Narrow pulse pressure

Clinical Investigation
(Clinical Portrait of Congestive HF)

Dilated pupils, a sympathetic nervous system response

Skin pale, gray, or cyanotic

Dyspnea, SOBOE is early symptom from pulmonary congestion
Orthopnea, cannot breathe unless sitting up
Crackles, wheeze are adventitious breath sounds
Cough, frothy pink or white sputum

Decreased blood pressure stimulates sympathetic nervous system, which acts on heart to increase rate and increase force of contraction

Nausea and vomiting as peristalsis slows and bile and fluids back up into stomach

Ascites, fluid in peritoneal cavity

Dependent, pitting edema, in sacrum, legs

Anxiety, gasping from pulmonary congestion

Falling O$_2$ saturation

Confusion, unconsciousness from decreased O$_2$ to brain

Jugular vein distention from venous congestion

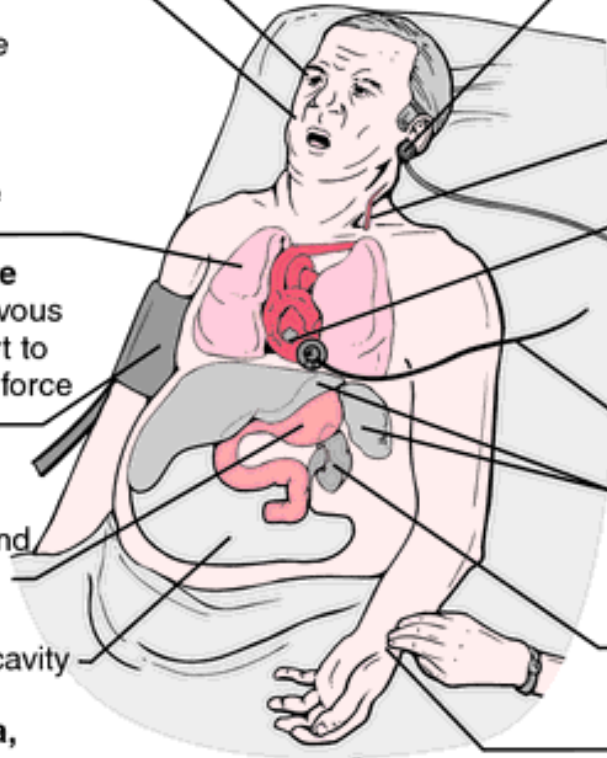**Infarct,** may be cause of decreased cardiac output

Fatigue, weakness from decreased cardiac output

S$_3$ gallop, tachycardia

Enlarged spleen and liver from venous congestion. This causes pressure on breathing
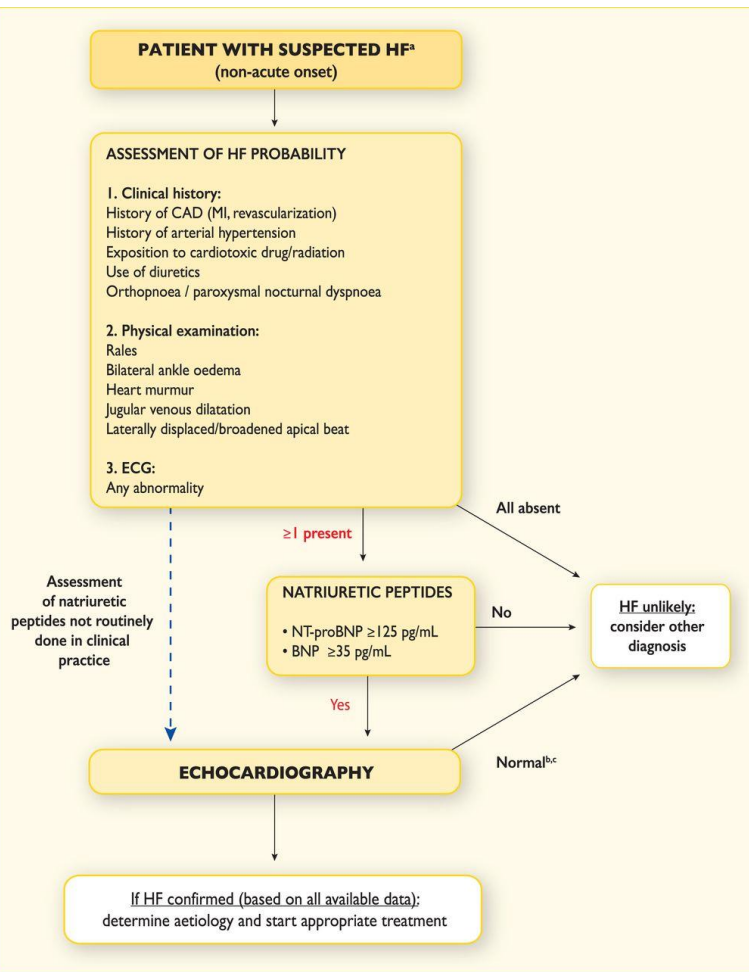
Decreased urine output

Weak pulse Cool, moist skin

Diagnosis
(Diagnostic algorithm of Non-Acute HF)

- Diagnostic algorithm for a diagnosis of HF of non-acute onset

- BNP - B-type natriuretic peptide; CAD - coronary artery disease; MI - myocardial infarction; NT-proBNP - N-terminal pro-B type natriuretic peptide.

Diagnosis

(The Framingham Major Criteria for the Diagnosis of HF)

- Paroxysmal nocturnal dyspnea
- Weight loss of 4.5 kg in 5 days in response to treatment
- Neck vein distention
- Rales
- Acute pulmonary edema

- Hepatojugular reflux
- S_3 gallop
- Central venous pressure greater than 16 cm water
- Circulation time of 25 seconds
- Radiographic cardiomegaly
- Pulmonary edema, visceral congestion

Diagnosis

(The Framingham Minor Criteria for the Diagnosis of HF)

- Nocturnal cough

- Dyspnea on ordinary exertion

- A decrease in vital capacity by one third the maximal value recorded

- Pleural effusion

- Tachycardia (rate of 120 bpm)

- Bilateral ankle edema

Diagnosis
(The Initial Evaluation for Suspected Heart Failure) 1

- Complete blood count (CBC)

- Urinalysis

- Electrolyte levels

- Renal and liver function studies

- Fasting blood glucose levels

- Lipid profile

- Thyroid stimulating hormone (TSH) levels

- B-type natriuretic peptide levels

Diagnosis
(The Initial Evaluation for Suspected Heart Failure) 2

- N-terminal pro-B-type natriuretic peptide

- Electrocardiography

- Chest radiography

- 2-dimensional (2-D) echocardiography

- Nuclear imaging

- Maximal exercise testing

- Pulse oximetry or arterial blood gas

Diagnosis
(Natriuretic Peptide Cutoff Values for Acute decompensated HF)

	ACEP recommendation	CKD	BMI >35kg/m²
Exclude			
BNP	<100	<200[5]	54
NTproBNP	<300	<300[21]	NA
Identify			
BNP	>500		NA
NTproBNP			
<50 years	>450	>1,200[6]	NA
50–75 years	>900	>4,502[6]	NA
>75 years	>1,800		NA

ACEP -American College of Emergency Physicians, CKD – chronic kidney disease, BMI - body mass index
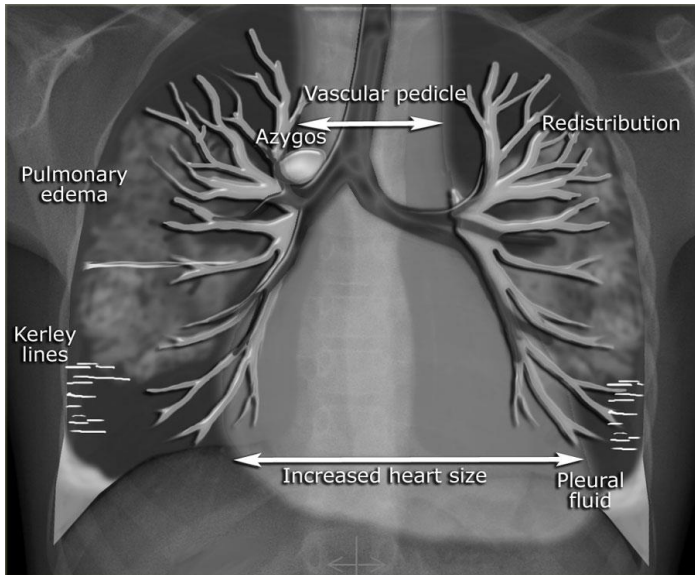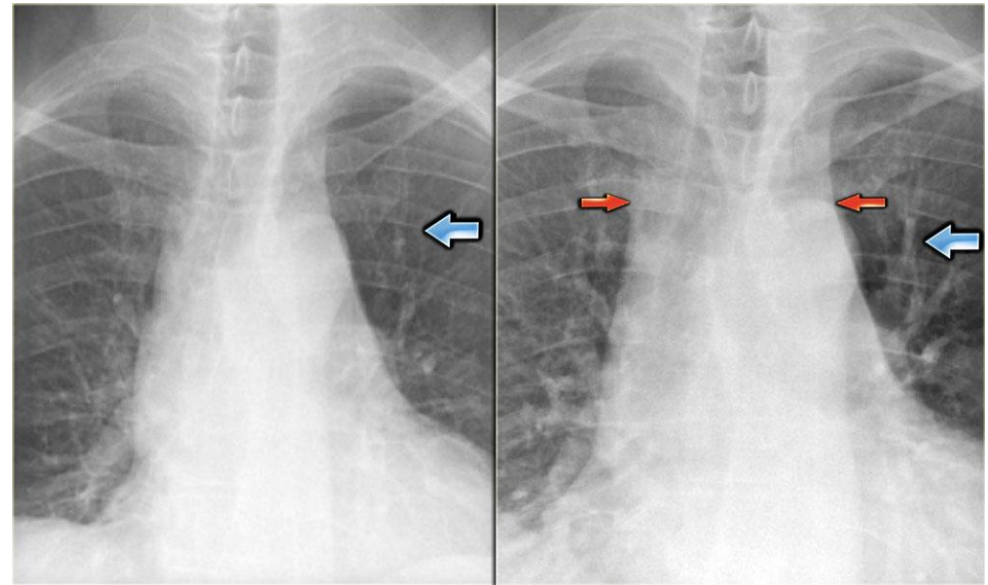
Diagnosis
(Electrocardiography)
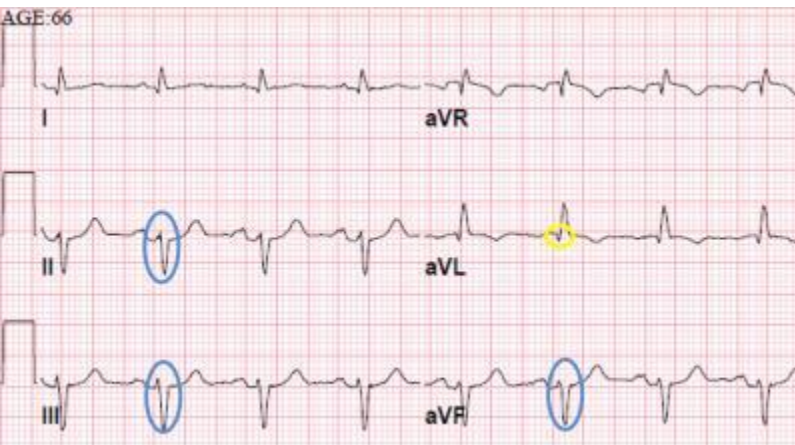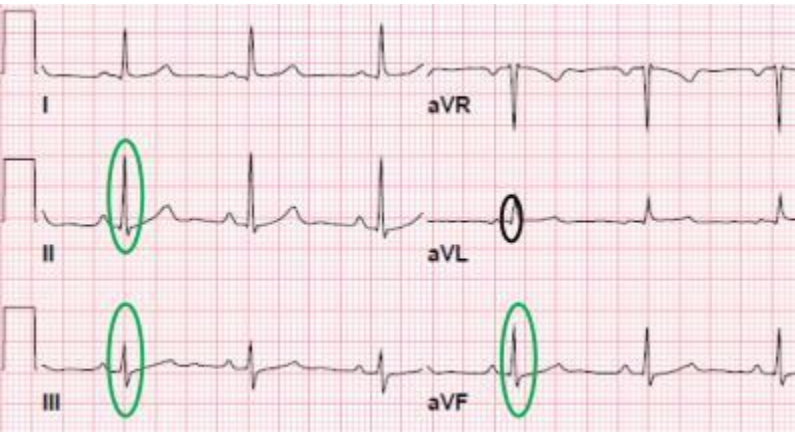
- The top ECG shows a reading of a person with a healthy heart.

- The bottom ECG shows a reading of a person with left anterior fascicular block (LAFB), previously thought to be benign but found to potentially signal a serious HF.
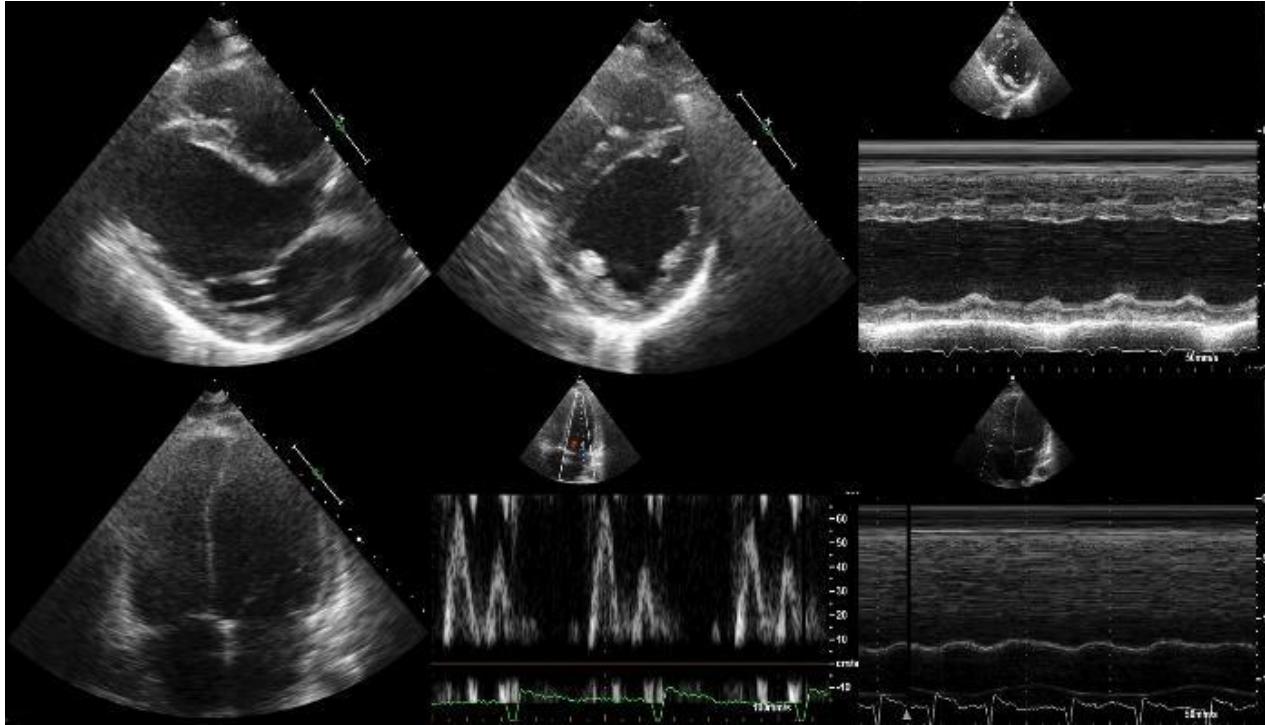
Diagnosis
(Chest Radiography)

Chest X-Ray signs of HF

Views of the upper lobe vessels of a patient in good condition (left) and during a period of CHF (right). Notice also the increased width of the vascular pedicle (red arrows).

Diagnosis
(Transthoracic Echocardiography)

Mild LV dilation (end-diastolic diameter 55 mm, end-systolic diameter 49 mm), normal LV thickness, asynchronous LV wall motion, LV ejection fraction, LV diastolic dysfunction (restrictive pattern), right ventricular (RV) dilation and impaired RV systolic function (tricuspid annular plane systolic excursion 9 mm), without evidence of significant pulmonary hypertension.

US MLE TEST

A 69-year-old is hospitalized for worsening dyspnea at rest. Physical examination is notable for crackles at both lung bases and 2+ edema at the ankles bilaterally. Current medications include losartan, metoprolol, furosemide and spironolactone. An EKG and echocardiography are ordered. Which of the following results would serve as the best indication for placement of an implantable cardioverter defibrillator (ICD) in this patient?

1. Supraventricular tachycardia on EKG
2. Atrial fibrillation on EKG
3. Reduced diameter of aortic valve on echocardiography
4. Reduced left ventricular ejection fraction on echocardiography
5. Left ventricular hypertrophy on echocardiography

US MLE TEST

Correct Answer 4: The single best indication for placement of an ICD in an adult is an ejection fraction less than 35%.

Incorrect Answers:

1: Supraventricular tachycardia on EKG should be converted to sinus rhythm in the acute setting.

2: Atrial fibrillation indicates a need for anticoagulation therapy in patients with heart failure.

3: Aortic stenosis as a cause of heart failure is an indication for valve replacement.

5: Left ventricular hypertrophy is commonly seen in heart failure, but is not in itself an indication for ICD placement.

Treatment
(General Principles) 1

- The goals of treatment in patients with HF are to improve their clinical status, functional capacity and quality of life, prevent hospital admission and reduce mortality.

- Treatment include lifestyle and pharmacological modalities with occasionally various forms of device therapy and rarely cardiac transplantation.

Treatment
(General Principles) 2

- In acute decompensated HF, the immediate goal is to re-establish adequate perfusion and oxygen delivery to end organs, that involve some combination of vasodilators, diuretic, and possibly non invasive positive pressure ventilation (NIPPV).

Treatment
(Lifestyle modification) 1

- Behavioral modification is a primary consideration in any chronic HF management program, with dietary guidelines regarding fluid and salt intake being of particular importance.

Treatment
(Lifestyle modification) 2

- Exercise should be encouraged and tailored to suit individual capabilities: the inclusion of regular physical conditioning as part of a cardiac rehabilitation program can significantly improve quality of life and reduce the risk of hospital admission for worsening symptoms however there is no evidence for a reduction in mortality rates as a result of exercise.

Treatment
(Lifestyle modification) 3

- Home visits and regular monitoring at HF clinics reduce the need for hospitalization and improve life expectancy.

Treatment
(Pharmacological Modalities: 1)

- First-line therapy due to reduced systolic function should include angiotensin-converting enzyme (ACE) inhibitors (ACE-I) or angiotensin receptor blockers (ARBs), and beta-adrenergic blocking agents (beta blockers).

- In people who are intolerant of ACE-I and ARBs or who have significant kidney dysfunction, the use of combined hydralazine and a long-acting nitrate; it is especially beneficial in African-Americans.

- In patients with markedly reduced ejection fraction in addition to beta blockers and ACE-I, should be used of an aldosterone antagonist.
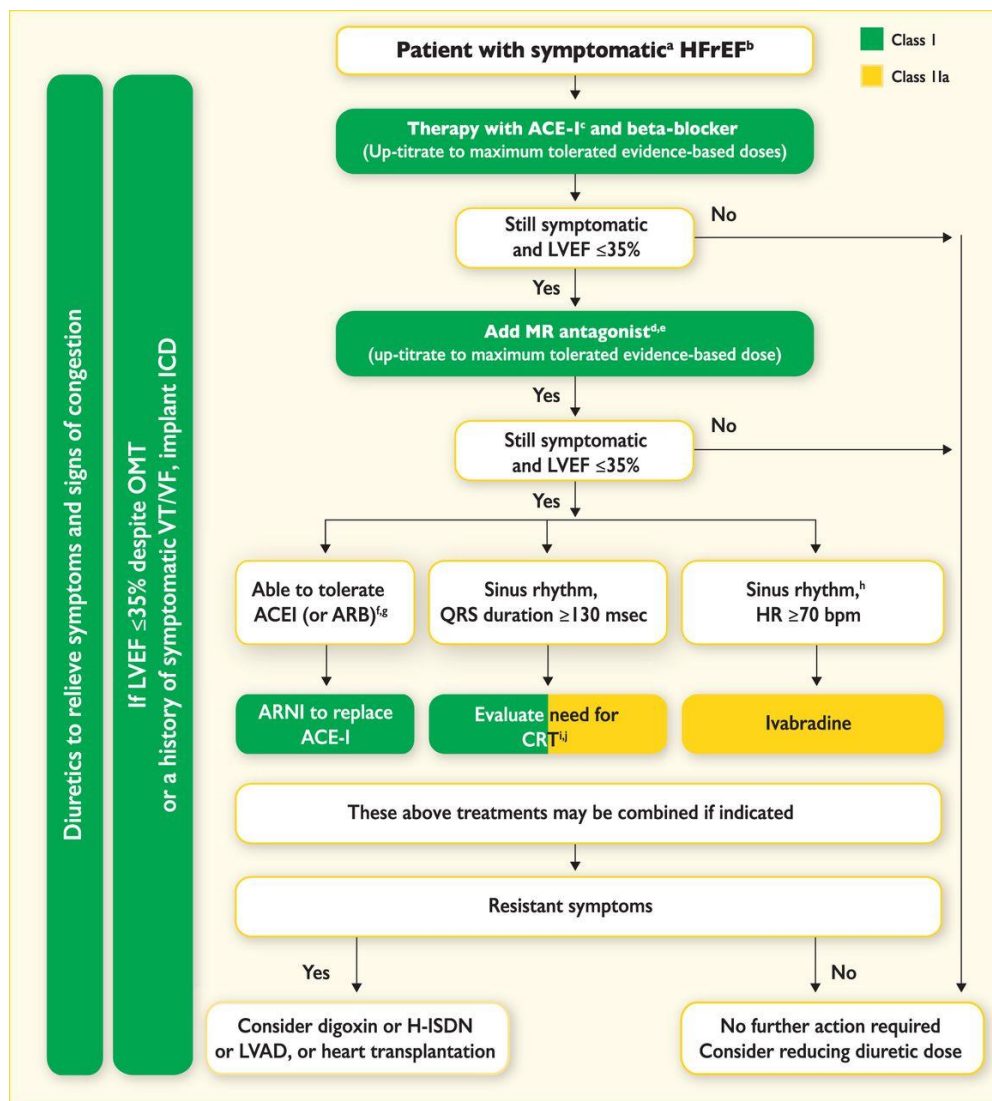
Treatment
(Pharmacological Modalities: 2)

- Second-line drug HF digitalis do not confer a mortality benefit.

- Diuretics have been a mainstay of treatment for patients with fluid accumulation, and include diuretics classes such as loop diuretics, thiazide-like diuretic, and potassium-sparing diuretic.

- A new therapeutic class of agents: angiotensin receptor neprilysin inhibitor (ARNI), I_f-channel inhibitor (ivabradin.

- Treating with parenteral iron if anemia is found.

Treatment
(Algorithm for a Patient with Symptomatic HF and reduced EF)

Treatment
(Angiotensin-Converting Enzyme Inhibitors) 1

- ACE-I have been shown to reduce mortality and morbidity in patients and are recommended unless contraindicated or not tolerated in all symptomatic patients.

- ACE-I should be up-titrated to the maximum tolerated dose in order to achieve adequate inhibition of RAAS.

Treatment
(Angiotensin-Converting Enzyme Inhibitors) 2

- There is evidence that in clinical practice the majority of patients receive suboptimal doses of ACE-I.

- ACE-I are also recommended in patients with asymptomatic LV systolic dysfunction to reduce the risk of HF development, HF hospitalization and death.

Treatment
(Angiotensin II type I receptor blockers)

- ARBs are recommended only as an alternative in patients intolerant of an ACE-Is.

- The combination of ACEI/ARBs for HF was reviewed by the European Medical Association, which suggested that benefits are thought to outweigh risks only in a select group of patients in whom other treatments are unsuitable.

Treatment
(Beta-Blockers) 1

- There is consensus that beta-blockers and ACE-I are complementary, and can be started together as soon as the diagnosis of HF with reduce EF is made.

- Beta-blockers should be initiated in clinically stable patients at a low dose and gradually up-titrated to the maximum tolerated dose.

- Beta-blockers should be considered for rate control in patients with HF and AF, especially in those with high heart rate.

Treatment
(Beta-Blockers) 2

- Beta-blockers are recommended in patients with a history of myocardial infarction and asymptomatic LV systolic dysfunction to reduce the risk of death

Treatment
(Mineralocorticoid/Aldosterone Receptor Antagonists)

- Mineralocorticoid/aldosterone receptor antagonists (spironolactone and eplerenone) block receptors that bind aldosterone and, with different degrees of affinity, other steroid hormone (e.g. corticosteroids, androgens) receptors.

- Spironolactone or eplerenone are recommended in all symptomatic patients (despite treatment with an ACE-I and a beta-blocker) with HF and LVEF ≤35%, to reduce mortality and HF hospitalization.

- Regular checks of serum potassium levels and renal function should be performed according to clinical status.

Treatment
(Diuretics) 1

- Diuretics are recommended to reduce the signs and symptoms of congestion in patients with HF.

- Loop diuretics produce a more intense and shorter diuresis than thiazides, although they act synergistically and the combination may be used to treat resistant oedema.

- The aim of diuretic therapy is to achieve and maintain euvolemia with the lowest achievable dose.

Treatment
(Diuretics) 2

- The dose of the diuretic must be adjusted according to the individual needs over time.

- Patients can be trained to self-adjust their diuretic dose based on monitoring of symptoms/signs of congestion and daily weight measurements.
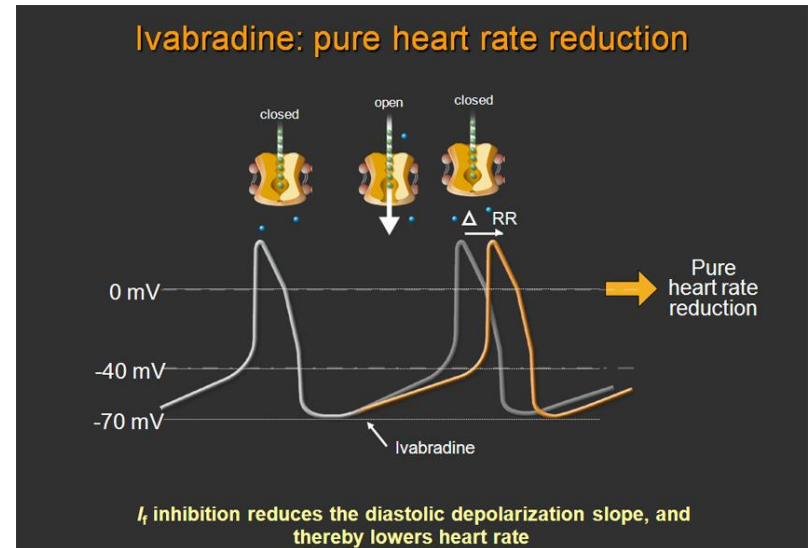
Treatment
(Angiotensin Receptor Neprilysin Inhibitor)

- Angiotensin receptor neprilysin inhibitor (ARNI) is a new class of agents acting on the RAAS and the neutral endopeptidase.

- The first in class is LCZ696, which is a molecule that combines the moieties of valsartan and sacubitril (neprilysin inhibitor) in a single substance.

- Neprilysin inhibiting enhancing diuresis, natriuresis and myocardial relaxation and anti-remodelling.
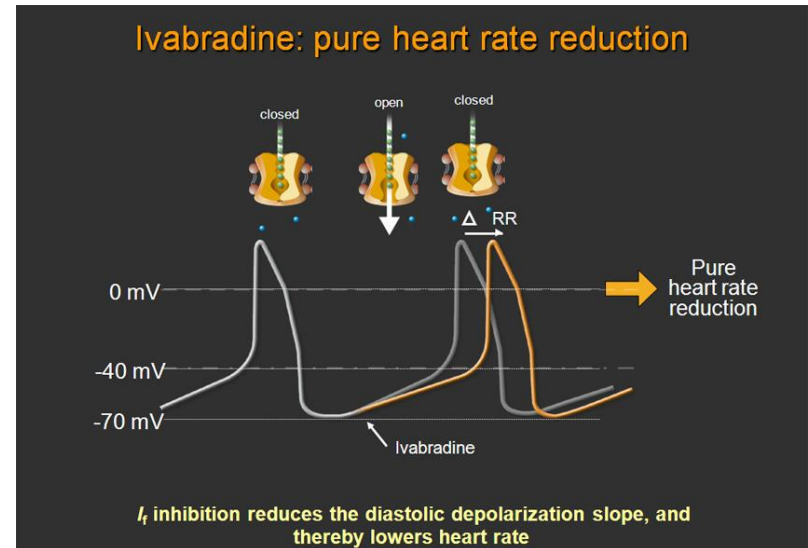
Treatment
(I_f-channel inhibitor) 1

- Ivabradine slows the heart rate through inhibition of the I_f channel in the sinus node and therefore should only be used for patients in sinus rhythm.

Ivabradine: pure heart rate reduction

I_f inhibition reduces the diastolic depolarization slope, and thereby lowers heart rate

Treatment
(I$_f$-channel inhibitor) 2

- Ivabradine reduced the combined endpoint of mortality and hospitalization in patients with symptomatic HF and LV EF ≤35%, in sinus rhythm and with a heart rate ≥70 beats per minute (bpm).

Ivabradine: pure heart rate reduction

I_f inhibition reduces the diastolic depolarization slope, and thereby lowers heart rate
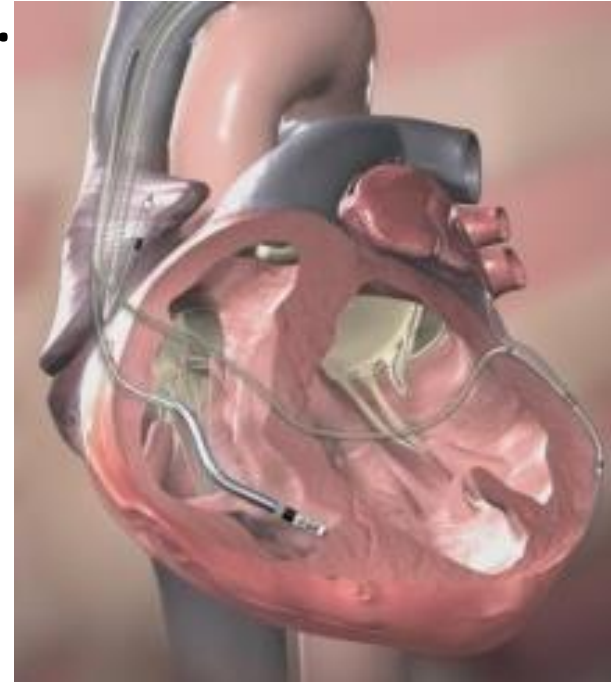
Treatment
(Not Recommended)

- Hydroxy-3-methylglutaryl-coenzyme A reductase ('statins').

- Oral anticoagulants and antiplatelet therapy.

- Renin inhibitors.

- Calcium-channel blockers.

Treatment
(Interventional Treatment)

- Implantable cardioverter-defibrillator.

- Cardiac resynchronization therapy.

- Revascularization procedures.

- Valve replacement/repair.

- Ventricular restoration.

- Extracorporeal membrane oxygenation.

- Ventricular assist devices.

- Heart transplantation.

- Total artificial heart.

Treatment
(Palliative Care)

The growing number of patients with Stage IV HF (intractable symptoms of fatigue, shortness of breath or chest pain at rest despite optimal medical therapy) should be considered for palliative care or hospice, according to American College of Cardiology/American Heart Association guidelines.

US MLE TEST

A 69-year-old male presents to the emergency department with shortness of breath. The patient has presented three times this past month with similar complaints. The patient sees no primary care physician and is currently not taking any medications. The patient states his shortness of breath started when he was walking from his car to a local restaurant. His temperature is 99.5°F (37.5°C), pulse is 100/min, blood pressure is 130/90 mmHg, respirations are 18/min, and oxygen saturation is 96% on room air. On physical exam you note a fatigued appearing gentleman. Cardiovascular exam reveals an additional heart sound after S2. Pulmonary exam is notable for bilateral crackles. Abdominal exam reveals an obese abdomen without pain in any of the quadrants. Lower extremity pitting edema is noted bilaterally. Which of the following sets of lab values most likely corresponds to this patient's presentation?

1. High BNP, high ADH, high sodium, high potassium, 2. High BNP, low ADH, normal sodium, low potassium, 3. High BNP, high ADH, low sodium, low potassium, 4. Low BNP, high ADH, low sodium, low potassium, 5. Low BNP, low ADH, normal sodium, normal potassium

US MLE TEST

Correct Answer 3: This patient is presenting with symptoms of congestive heart failure (CHF). The most likely laboratory abnormalities are elevated brain natriuretic peptide (BNP), high anti-diuretic hormone (ADH), low sodium, and low potassium.

Incorrect Answers:

1: High BNP, high ADH, high sodium, and high potassium does not reflect the changes that would be seen in CHF. Answer 2: High BNP, low ADH, normal sodium, and low potassium does not reflect the appropriate increase in ADH and subsequent decrease in sodium that would be seen in CHF., 4: Low BNP, high ADH, low sodium, and low potassium does not reflect the finding of elevated BNP that is classically found in the dilated ventricles of CHF., 5: Low BNP, low ADH, normal sodium, and normal potassium reflects the findings in a healthy patient.

Prognosis 1

- Prognosis in HF can be assessed in multiple ways including clinical prediction rules and cardiopulmonary exercise testing.

- HF is associated with significantly reduced physical and mental health, resulting in a markedly decreased quality of life.

- Although some people survive many years, progressive disease is associated with an overall annual mortality rate of 10%.

Prognosis 2

- Approximately 18 of every 1000 persons will experience an ischemic stroke during the first year after diagnosis of HF.

- As the duration of follow-up increases, the stroke rate rises to nearly 50 strokes per 1000 cases of HF by 5 years.[

Prophylaxis

- A person's risk of developing HF is inversely related to their level of physical activity.

- Those who achieved at least 500 MET-minutes/week (the recommended minimum by U.S. guidelines) had lower HF risk than individuals who did not report exercising during their free time; the reduction in heart failure risk was even greater in those who engaged in higher levels of physical activity than the recommended minimum.

Abbreviations

- ACE - angiotensin converting enzyme
- ACE-Is - angiotensin converting enzyme blockers
- ACEP -American College of Emergency Physicians
- ARBs - angiotensin receptor blockers
- ARNI -angiotensin receptor neprilysin inhibitor
- BMI - body mass index
- BNP – Brain (B-type) natriuretic peptide
- Bpm - beats per minute
- CAD - coronary artery disease
- CKD – chronic kidney disease
- ECG – electrocardiography
- EF - ejection fraction
- HFpEF – HF with preserved EF

- HR - heart rate
- HF – heart failure
- PVL - pressure–volume loop
- ICD - International Classification of Diseases
- LV - left ventricle
- LVH - LV hypertrophy
- MI - myocardial infarction
- NOS - Not Otherwise Specified
- NT- proBNP - N-terminal pro-B type natriuretic peptide
- PVL - pressure–volume loop
- RAAS - renin-angiotensin-aldosterone system
- RV - right ventricle
- SV - stroke volume

Diagnostic and treatment guidelines

- [2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure](#)
- [Treatment of heart failure in adult congenital heart disease: a position paper of the Working Group of Grown-Up Congenital Heart Disease and the Heart Failure Association of the European Society of Cardiology](#)
- [2013 ACCF/AHA Guideline for the Management of Heart Failure](#)