Heart failure

LECTURE IN INTERNAL MEDICINE FOR V COURSE STUDENTS

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Plan of the Lecture

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

medicaldevice-network.com/uploads/feature/feature42840/1-heart-failure.jpg

Definition

- (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).
- 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.

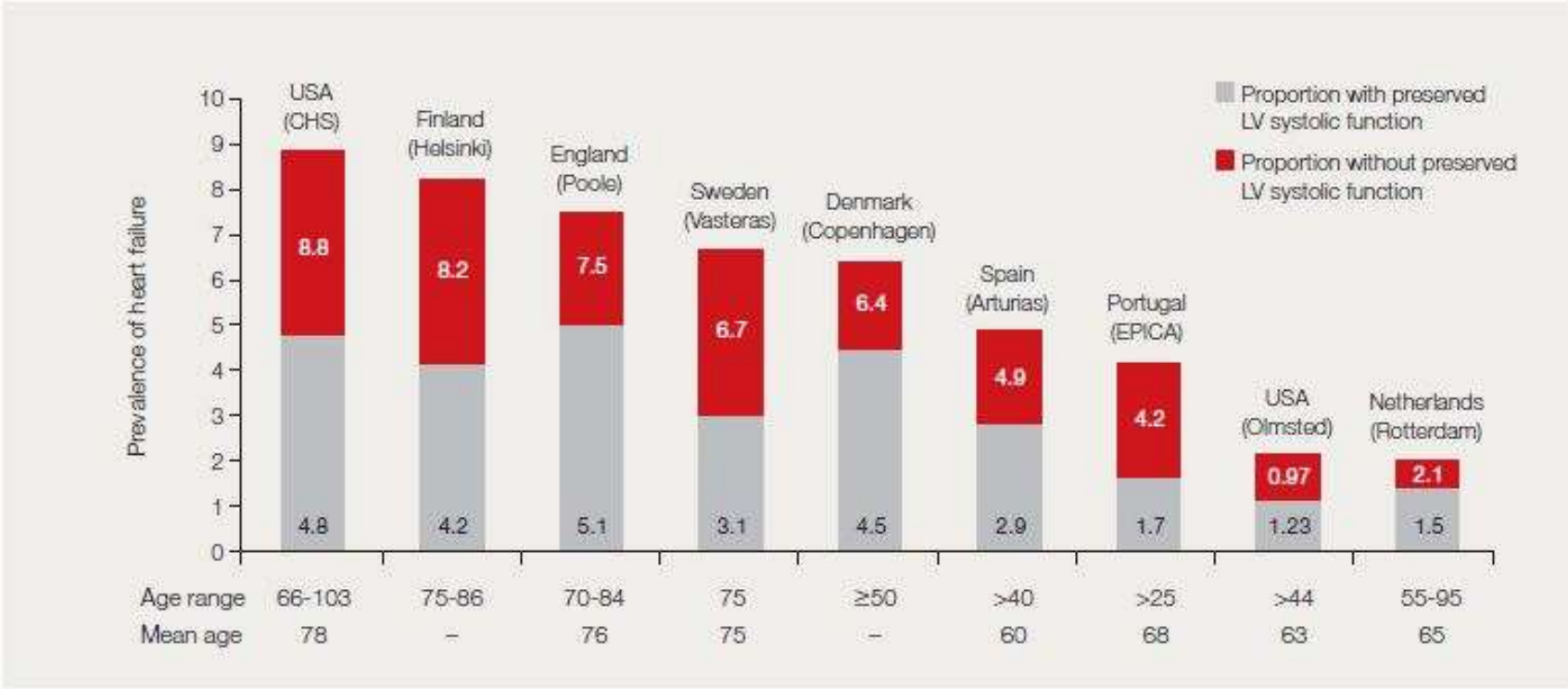
eurheartj.oxfordjournals.org/content/early/2016/06/08/eurheartj.ehw128

Epidemiology
(Hear Failure around the World)

- 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).
- 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.

eurheartj.oxfordjournals.org/content/early/2016/06/08/eurheartj.ehw128

Epidemiology
(The Heart Failure Epidemic)



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

medicographia.com/2012/02/the-heart-failure-epidemic/

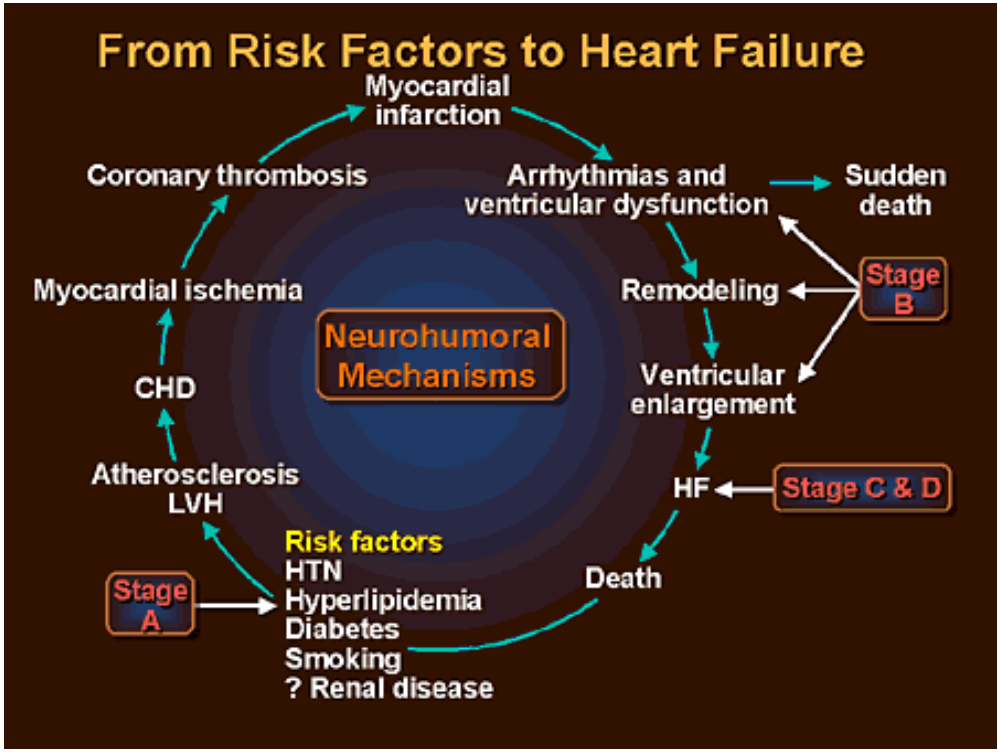
Risk Factors and Etiology
(Underlying Causes of Systolic Heart Failure)

- Coronary artery disease
- Diabetes mellitus
- Hypertension
- Valvular heart disease (stenosis or regurgitant lesions)
- Arrhythmia (supraventricular or ventricular)
- Infections and inflammation (myocarditis)
- 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)

emedicine.medscape.com/article/163062-overview#a4

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

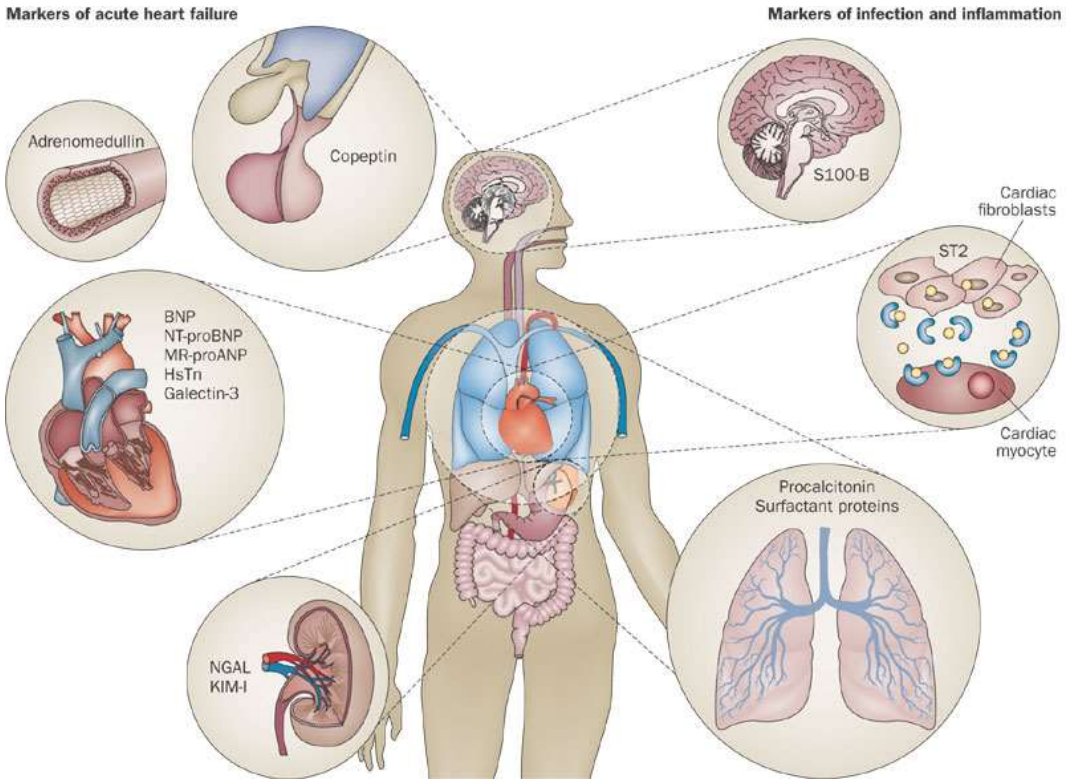
- Coronary artery disease
- Diabetes mellitus
- Hypertension
- Valvular heart disease
- Hypertrophic cardiomyopathy
- Restrictive cardiomyopathy
- Constrictive pericarditis



medscape.org/viewarticle/491741_3

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

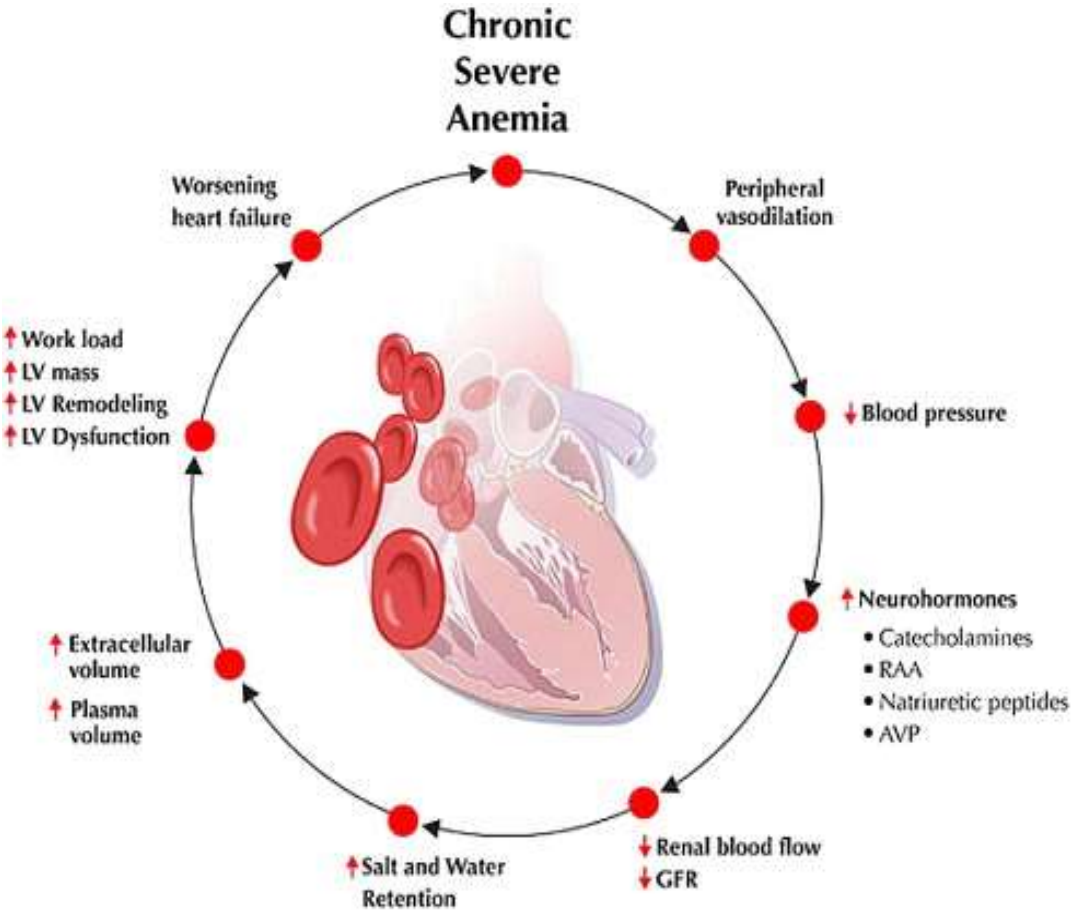


nature.com/nrcardio/journal/v9/n8/fig_tab/nrcardio.2012.60_F1.html

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



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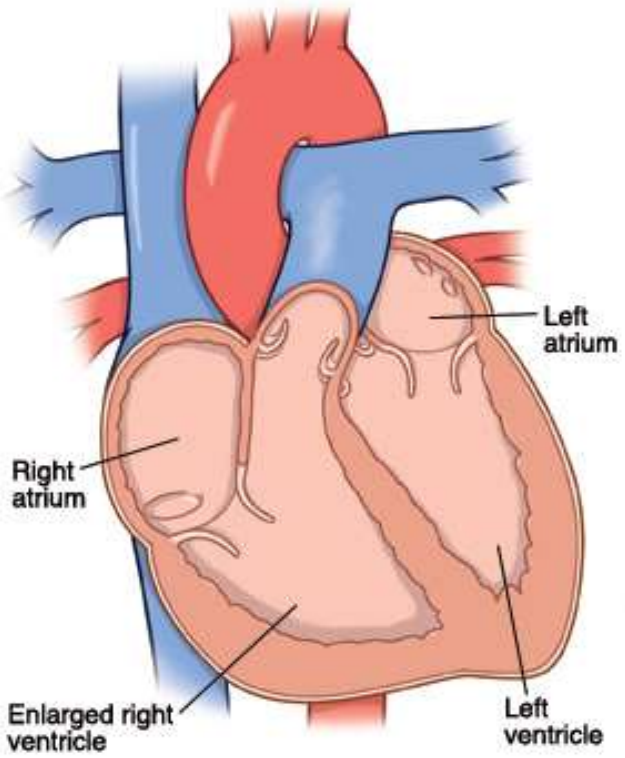
Risk Factors and Etiology
(Underlying Causes of Right Heart Failure)

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



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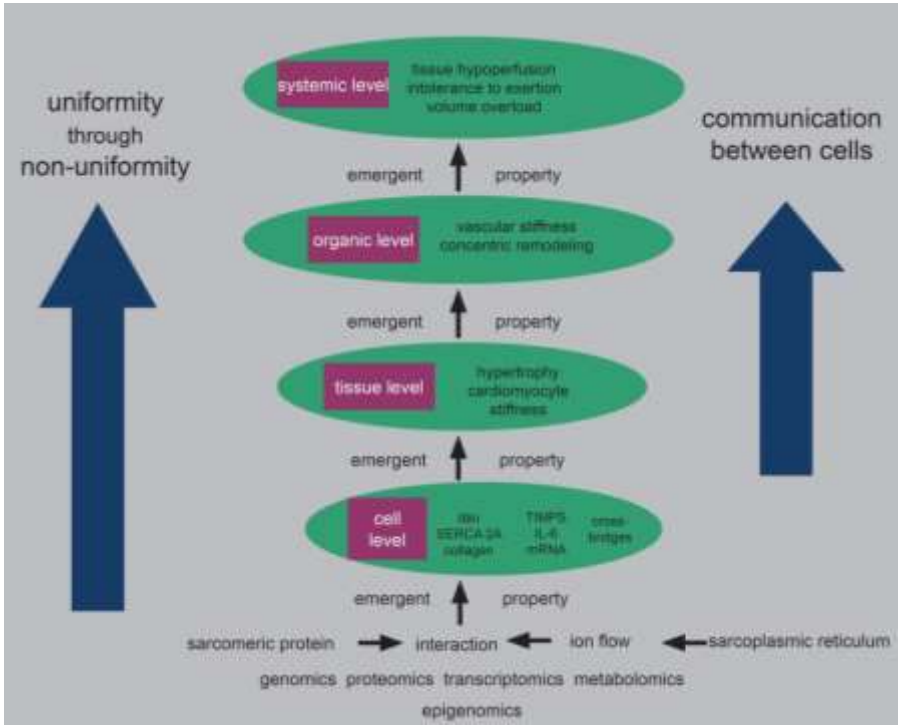
Risk Factors and Etiology
(Fundamental Causes of Heart Failure)

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.



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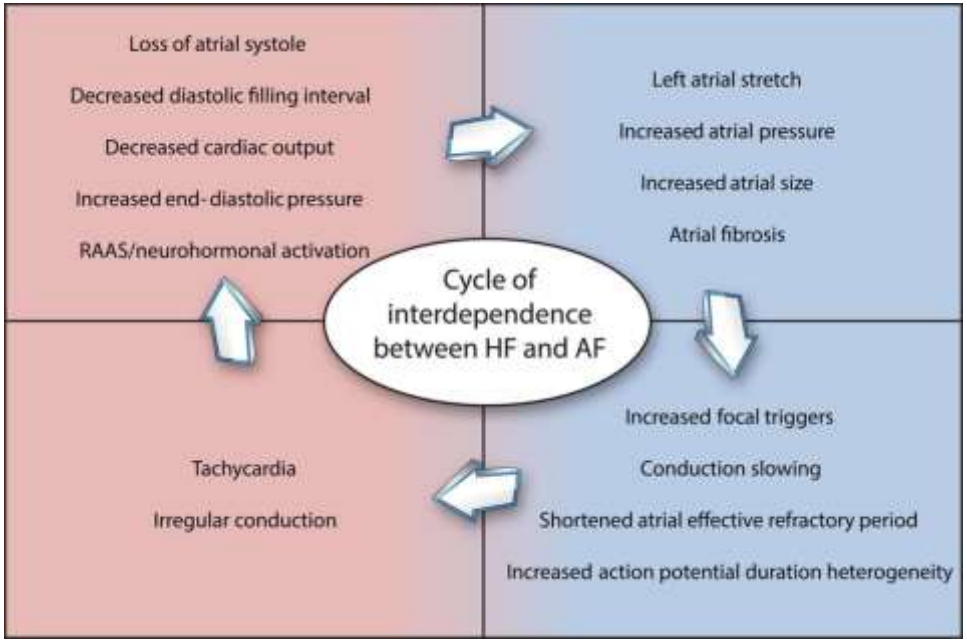
Risk Factors and Etiology
(Precipitating Causes of Heart Failure)

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) that alter the homeostasis of heart failure patients.



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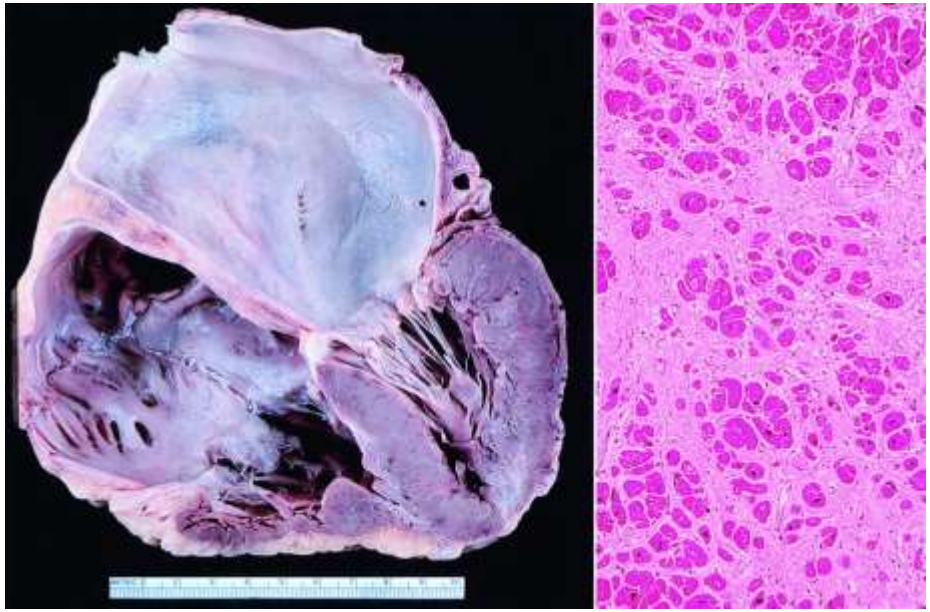
Risk Factors and Etiology
(Genetic Causes of Heart Failure)

- Dilated cardiomyopathy.
- Arrhythmic cardiomyopathy.
- Right ventricular cardiomyopathy .
- Restrictive cardiomyopathy.



Idiopathic Restrictive Cardiomyopathy, marked interstitial fibrosis

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Mechanisms
(The Common Pathophysiologic State)

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

en.wikipedia.org/wiki/Heart_failure#Causes http://emedicine.medscape.com/article/163062-overview#a3

Mechanisms
(Myocytes and Myocardial Remodeling)

- 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.
- 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.

emedicine.medscape.com/article/163062-overview#a3

Mechanisms
(Left Ventricle Stiffness)

- 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.
- 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.

emedicine.medscape.com/article/163062-overview#a3

Mechanisms
(Concentric Left Ventricle Hypertrophy)

- 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.
- 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.

emedicine.medscape.com/article/163062-overview#a3

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.

en.wikipedia.org/wiki/Heart_failure#Causes

Mechanisms
(Diastolic dysfunction)

- 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.
- 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.

en.wikipedia.org/wiki/Heart_failure#Causes

Mechanisms
(Arrhythmias)

- 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.
- 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.

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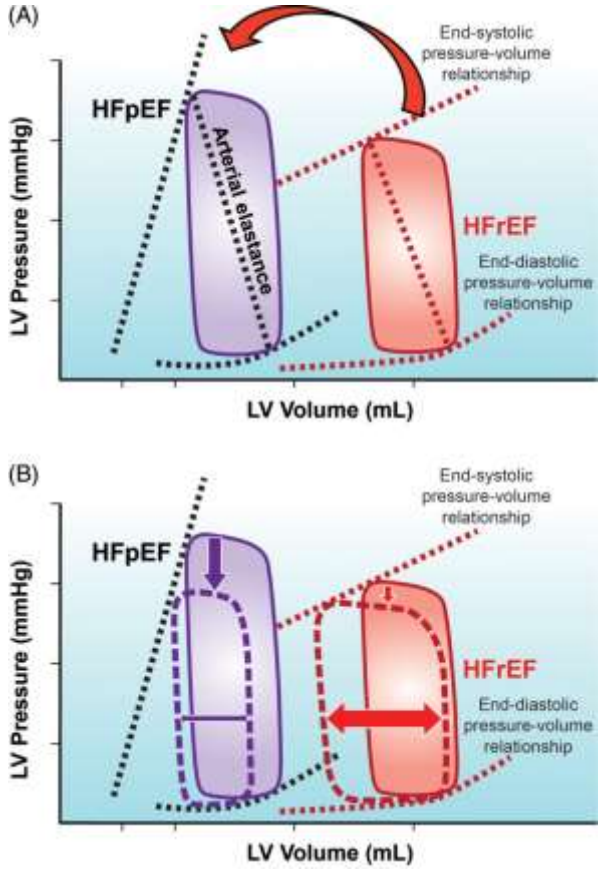
Mechanisms
(Pressure–Volume Loops in Heart Failure)

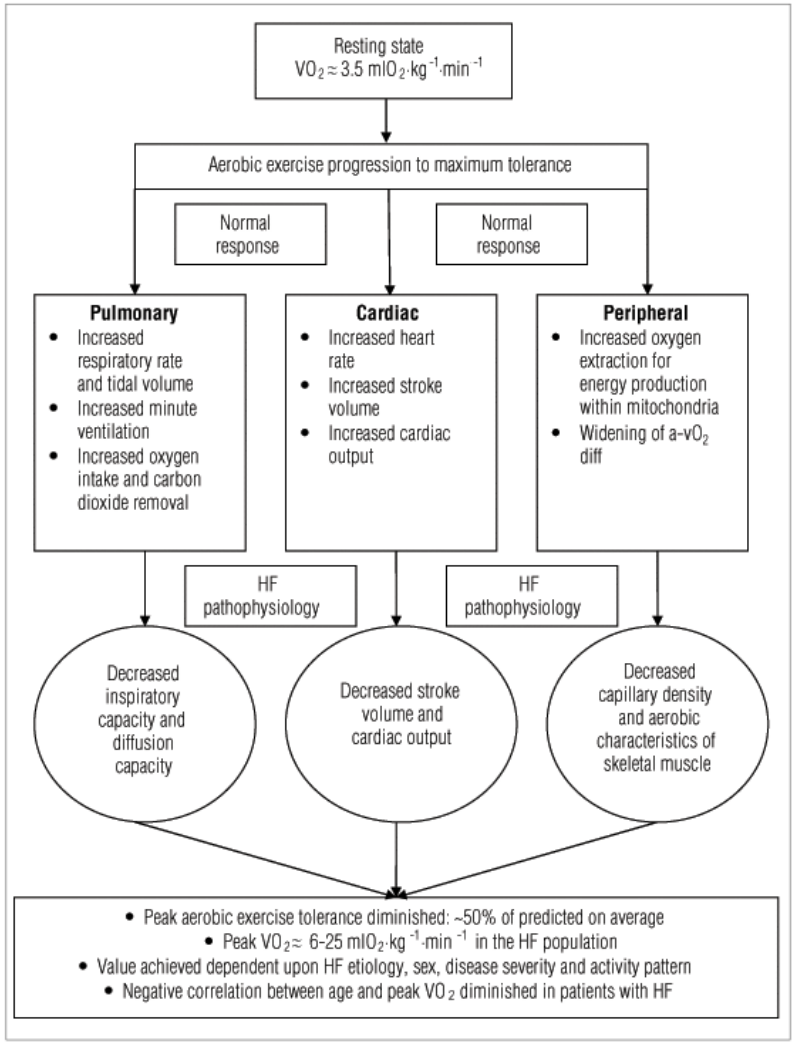


- Pressure–volume loop (PVL)characteristics in HF with preserved (black) ejection fraction (EF)and HF with reduced (red) EF in baseline conditions (*A*), and in response to vasodilators (*B*).
- (*A*) Curved arrow depicts the steeper end-systolic pressure–volume relationship in HF with preserved EF compared with HF with reduced EF.
- (*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.

eurheartj.oxfordjournals.org/content/35/16/1022

Mechanisms

(HF affects the Systems involved in the Physiological Response)



- Severely compromised cardiac function is a primary pathophysiological component in HF.
- Patients with HF frequently present reduced capillary density and intrinsic skeletal muscle abnormalities, primarily in the form of diminished aerobic (mitochondrial) function.

.scielo.br/scielo.php?script=sci_arttext&pid=S1413-35552008000200002

Classification
(International Classification of Diseases (ICD))

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

150 Heart failure

I50.0 Congestive heart failure (congestive heart disease, right ventricular failure (secondary to left heart failure))

I50.1Left ventricular failure (cardiac asthma, left heart failure, oedema of lung, pulmonary oedema)

I50.9 Heart failure, unspecified (cardiac, heart or myocardial failure Not Otherwise Specified (NOS))

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.

en.wikipedia.org/wiki/New_York_Heart_Association_Functional_Classification

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.

heartfailurecenter.com/hfcheartfailurestages.shtm

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%).

heartfailurecenter.com/hfcheartfailurestages.shtm

Clinical Investigation
(Signs and Symptoms)

- 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.
- 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.

en.wikipedia.org/wiki/Acute_coronary_syndrome#Signs_and_symptoms

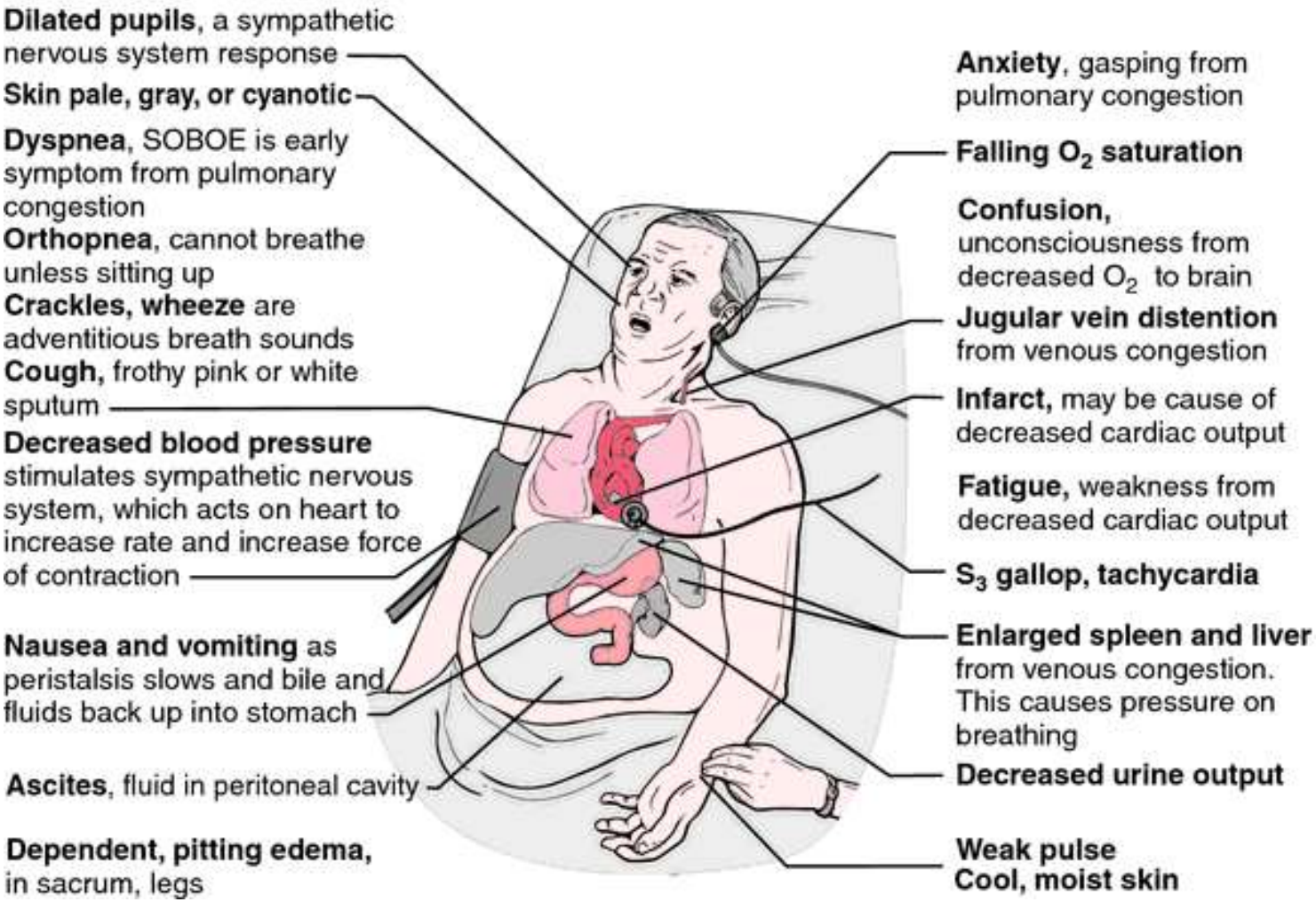
Clinical Investigation
(Price of Signs and Symptoms)

- 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.
- 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

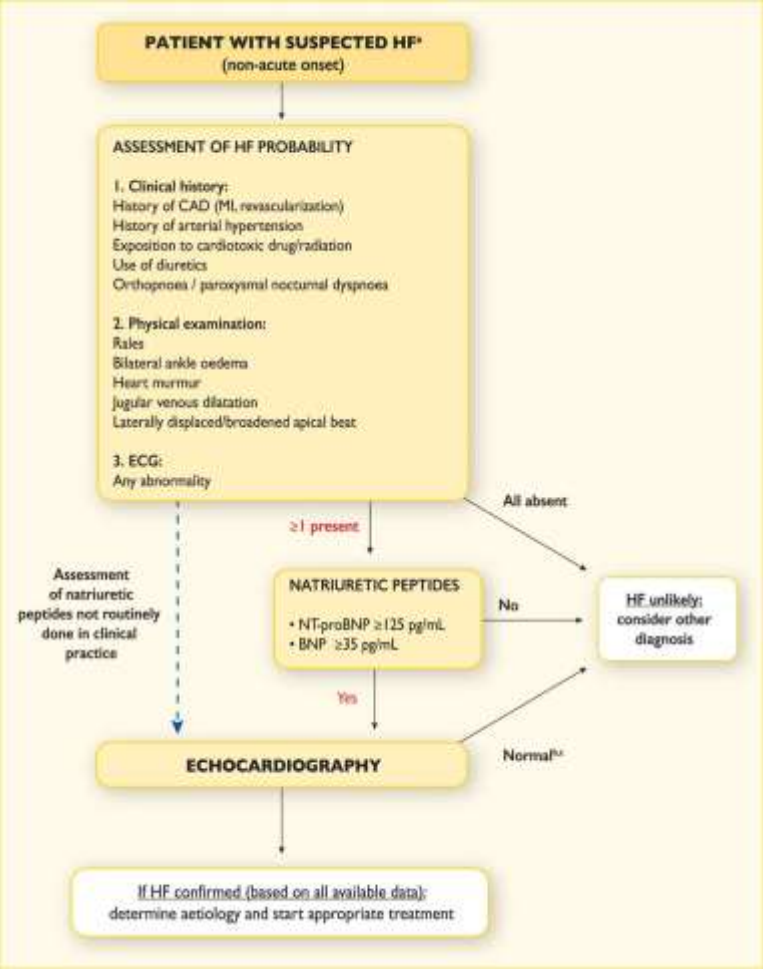
en.wikipedia.org/wiki/Acute_coronary_syndrome#Signs_and_symptoms

Clinical Investigation
(Clinical Portrait of Congestive HF)



medical-dictionary.thefreedictionary.com/heart+failure

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.

eurheartj.oxfordjournals.org/content/early/2016/06/08/eurheartj.ehw128

Diagnosis

(The Framingham Criteria for the diagnosis of HF)

Major criteria

- 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

Minor criteria

- 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

emedicine.medscape.com/article/155919-treatment#d5

Diagnosis
(The Initial Evaluation for Suspected Heart Failure)

- 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
- 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

emedicine.medscape.com/article/155919-treatment#d5

Diagnosis

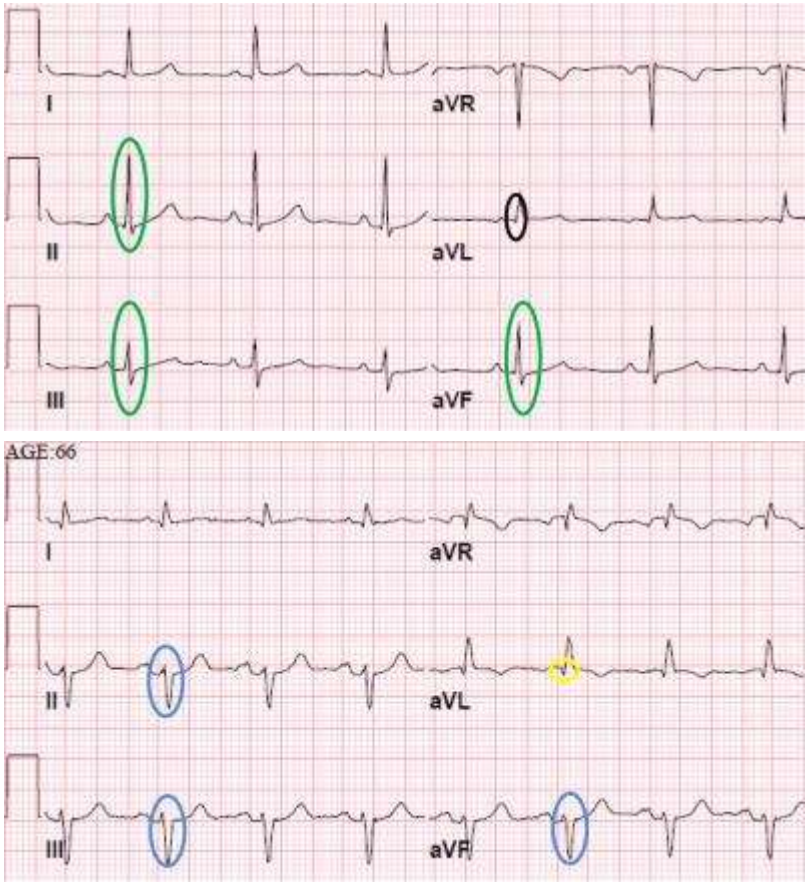
(Natriuretic Peptide Cutoff Values for Acute decompensated HF)

		ACEP recommendation	CKD	BMI >35kg/m^2
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

dovepress.com/cardiopulmonary-laboratory-biomarkers-in-the-evaluation-of-acute-dyspn-peer-reviewed-fulltext-article-OAEM

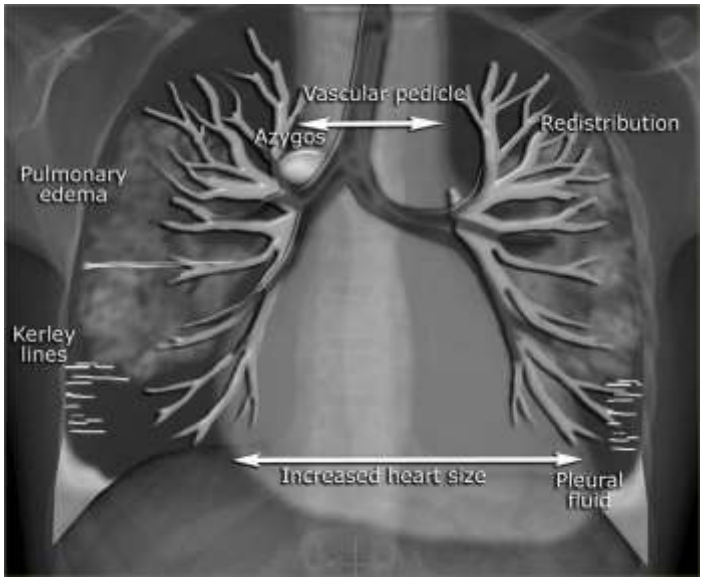
Diagnosis
(Electrocardiography)



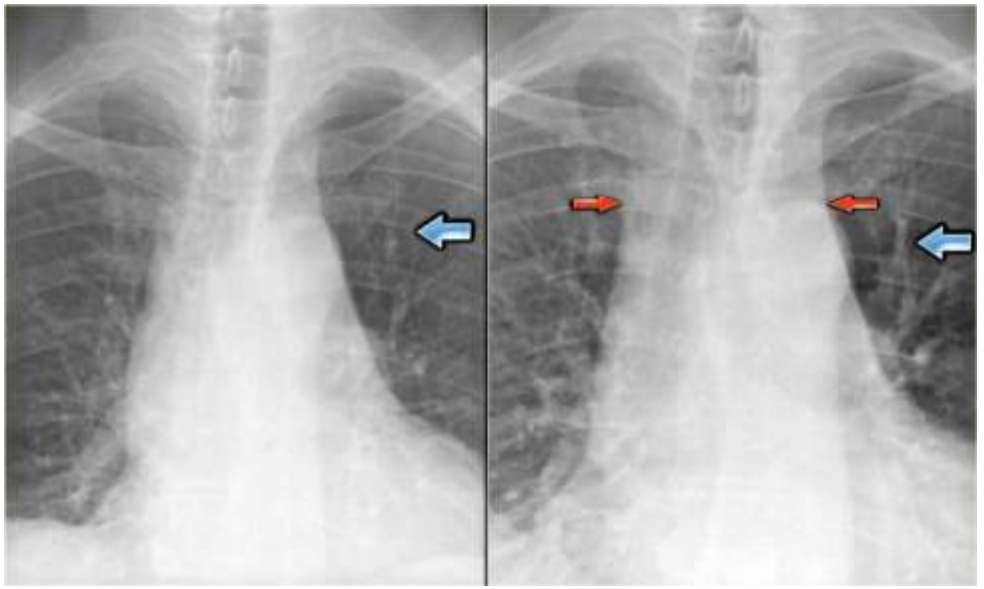
- The top ECG shows a reading of a person with a healthy heart.
- The bottom ECG shows a reading of a person with left anterior fascicular block (LAFB), previously thought to be benign but found to potentially signal a serious HF.

ucsf.edu/news/2013/04/105041/routine-ekg-finding-could-signal-serious-heart-problem

Diagnosis
(Chest Radiography)



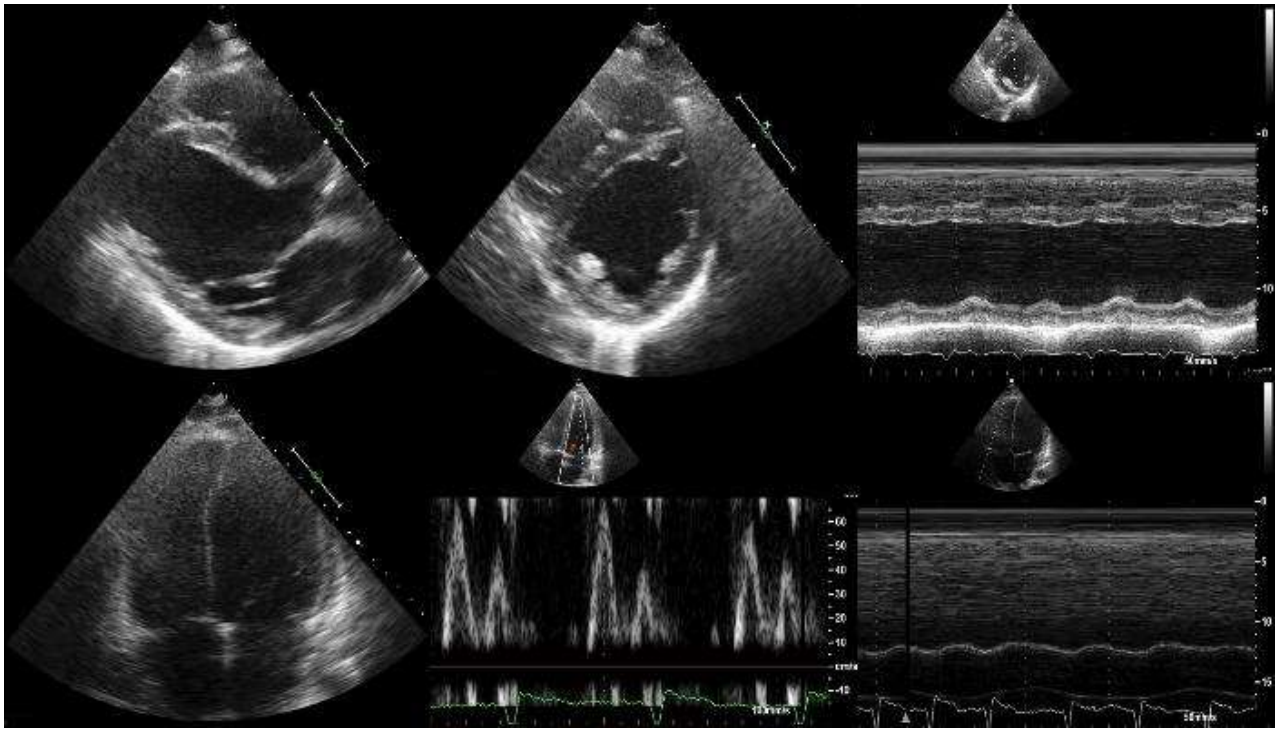
Chest X-Ray signs of HF



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).

radiologyassistant.nl/en/p4c132f36513d4/chest-x-ray-heart-failure.html

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.

elsevier.es/en-revista-revista-portuguesa-cardiologia-english-edition--434-articulo-systemic-sclerosis-a-rare-cause-90443802

Treatment
(General Principles)

- 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.
- 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).

en.wikipedia.org/wiki/Heart_failure#Management eurheartj.oxfordjournals.org/content/early/2016/06/08/eurheartj.ehw128

Treatment
(Lifestyle modification)

- Behavioral modification is a primary consideration in any chronic HF management program, with dietary guidelines regarding fluid and salt intake being of particular importance.
- 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.
- Home visits and regular monitoring at HF clinics reduce the need for hospitalization and improve life expectancy.

en.wikipedia.org/wiki/Heart_failure#Management

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.

en.wikipedia.org/wiki/Heart_failure#Management

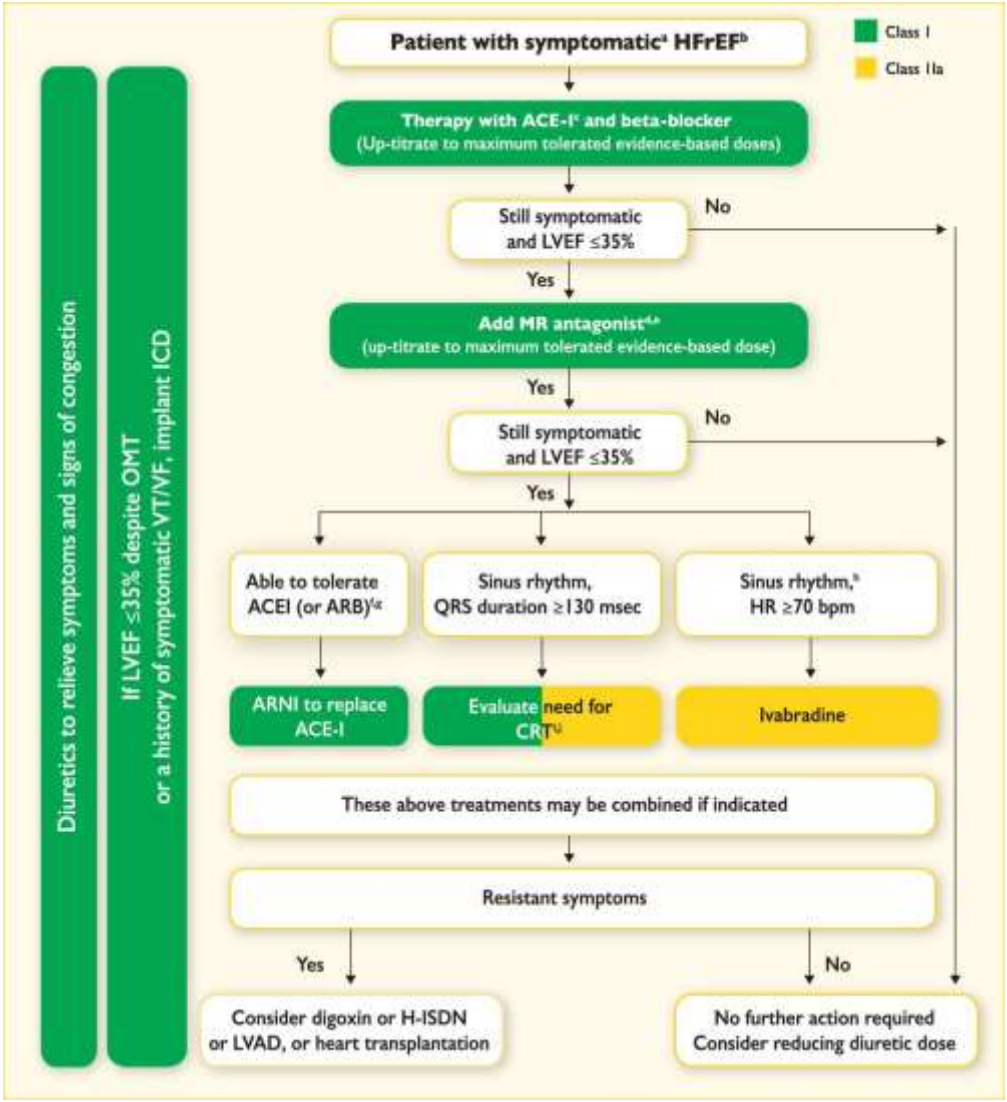
Treatment
(Pharmacological Modalities: 2)

- 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.

en.wikipedia.org/wiki/Heart_failure#Management

Treatment

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



eurheartj.oxfordjournals.org/content/early/2016/06/08/eurheartj.ehw128

Treatment
(Angiotensin-Converting Enzyme Inhibitors)

- 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.
- 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.

eurheartj.oxfordjournals.org/content/early/2016/06/08/eurheartj.ehw128

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.

eurheartj.oxfordjournals.org/content/early/2016/06/08/eurheartj.ehw128

Treatment
(Beta-Blockers)

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

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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.

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Treatment
(Diuretics)

- 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.
- 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.

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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.

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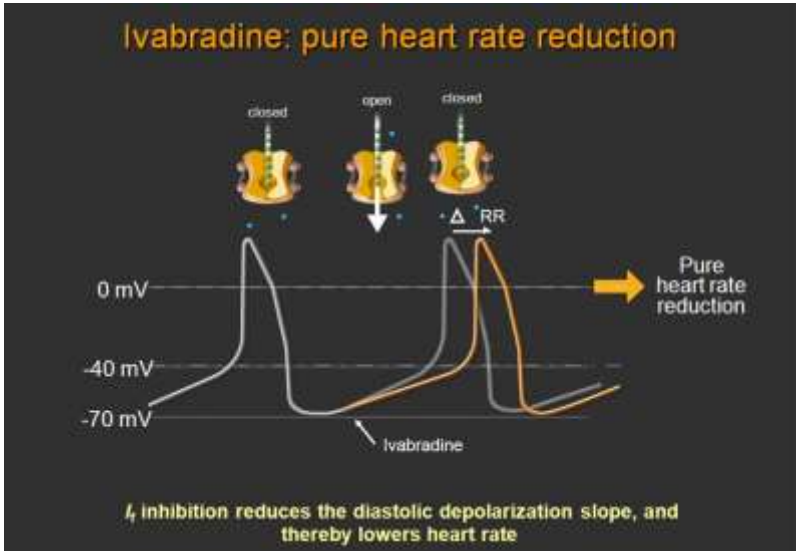
Treatment
(I_f-channel inhibitor)

- 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 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).



eurheartj.oxfordjournals.org/content/early/2016/06/08/eurheartj.ehw128 drsvenkatesan.com/tag/ivabradine/

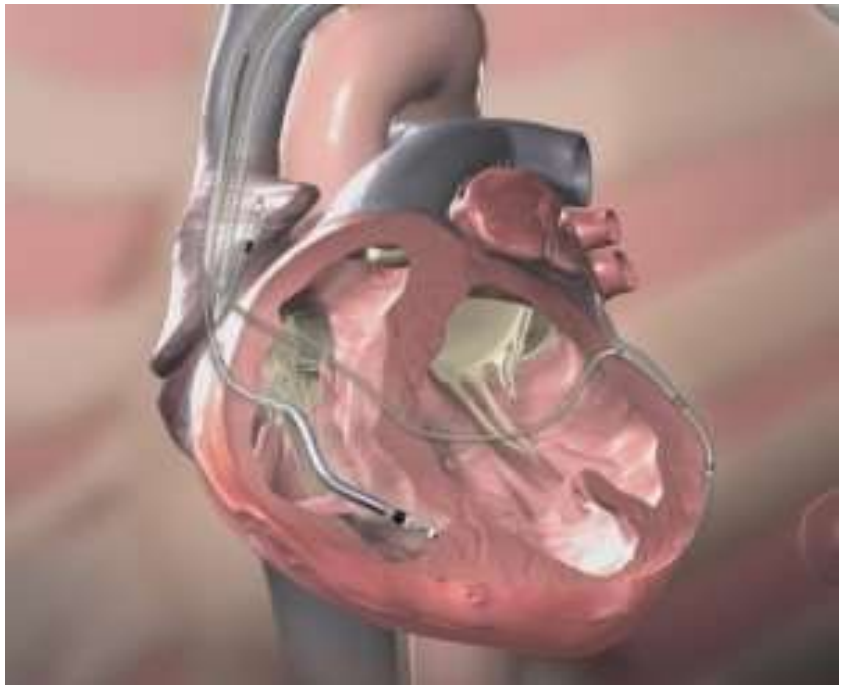
Treatment
(Not Recommended)

- Hydroxy-3-methylglutaryl-coenzyme A reductase (‘statins’).
- Oral anticoagulants and antiplatelet therapy.
- Renin inhibitors.
- Calcium-channel blockers.

eurheartj.oxfordjournals.org/content/early/2016/06/08/eurheartj.ehw128 consumerreports.org/cro/2014/03/treating-high-cholesterol-with-statins/index.htm

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.



eurheartj.oxfordjournals.org/content/early/2016/06/08/eurheartj.ehw128 emedicine.medscape.com/article/163062-overview#a1 www.azheartrhythmcenter.com/procedures/

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.

eurheartj.oxfordjournals.org/content/early/2016/06/08/eurheartj.ehw128 www.eapcnet.eu/Themes/Specificgroups/Heartdisease.aspx

Prognosis

- 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%.
- 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.[

en.wikipedia.org/wiki/Heart_failure#Prognosis

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.

en.wikipedia.org/wiki/Heart_failure#Prevention

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

Europe

- 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

North America

- 2013 ACCF/AHA Guideline for the Management of Heart Failure