HEART FAILURE



HEART FAILURE (HF)

Heart failure is a syndrome manifesting as the inability of the heart to fill with or eject blood satisfactory due to any structural or functional cardiac conditions



HEART FAILURE (HF)

- A state in which the heart cannot provide sufficient cardiac output to satisfy the metabolic needs of the body
- It is commonly termed congestive heart failure (CHF) since symptoms of increase venous pressure are often prominent

ESC GUIDELINES FOR DIAGNOSTIC AND TREATMENT OF ACUTE AND CHRONIC HF

(2016)

HF is a clinical syndrome 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) 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.

HEART FAILURE (HF)

- HF is an imprecise term used to describe the pathological state that develops when the heart cannot maintain an adequate cardiac output or can do so only at the expense of an elevated filling pressure.
- In practice, HF may be diagnosed whenever a patient with significant heart disease develops the signs or symptoms of a low cardiac output, pulmonary congestion or systemic venous congestion.

STATISTICS

- In HF afflicts 2,1% of population
- At 40 years of age, the lifetime risk of developing heart failure for both men and women is 1 in 5
- The number of people experiencing heart failure has increased steadily during the last 2 decades T

HEART FAILURE (HF)

- Final common pathway for many cardiovascular diseases whose natural history results in symptomatic or asymptomatic left ventricular dysfunction
- Cardinal manifestations of heart failure include dyspnea, fatigue and fluid retention
- Risk of death is 5-10% annually in patients with mild symptoms and increases to as high as 30-40% annually in patients with advanced disease

PROGNOSIS

HF is a strong predictor of the sudden cardiac death

The 5-year mortality rate for patients HF is 50 60%

AETHIOLOGY OF HF



The three major contributors are: hypertension, coronary ar tery disease, dilated cardiomyopathy, heart defects, arrhythmias (atrial fibrillation, tachycardia cardiomyopathy, complete AV block) myocarditis other cardiomyopathies (hypertrophic, alcoholic, restrictive)

RISK FACTORS

- Hypertension
- **Diabetes**
- **Age**
- Obesity
- Heart valve problems
- Unhealthy lifestyle (smoking, physical inactivity, etc.)

NORMAL PHYSIOLOGY OF THE HEART

Cardiac output depends on:

1. Contractility

- 2. **Preload** (the volume and pressure in the ventricle <u>at the end of diastole</u>)
- 3. Afterload (the volume and pressure in the ventricle <u>during</u> <u>systole</u>)

Frank-Starlings Law: contractility is related to the degree of myocardial stretching



FRANK-STARLI NGS LAW:



Figure 1. Frank-Starling mechanism. Increasing venous return to the left ventricle increases left ventricular enddiastolic pressure (LVEDP) and volume, thereby increasing ventricular preload. This results in an increase in stroke volume (SV). The "normal" operating point is at a LVEDP of ~8 mmHg and a SV of ~70 ml/beat.



Figure 2. Family of Frank-Starling curves. Changes in afterload and inotropy shift the Frank-Starling curve up or down.

NEUROHUMORAL ACTIVATION

Sympathetic nervous system

Decreased contractility

- Renin-angiotensin system
- Increased release of vasopressin
-) <u>Endothelin</u>

arterial and <u>venous</u> vasoconstriction

increased <u>blood</u> volume.

COMPENSATORY CHANGES IN HEART FAILURE

- Activation of CNS
- Activation of RAS
- Increased heart rate
- Release of ADH
- Release of atrial natriuretic peptide
- Chamber enlargement
- Myocardial hypertrophy

CLASSIFICATION

Heart failure can be classified in several ways
 1 - Acute and chronic HF
 Left , right and biventricular HF
 Systolic and diastolic dysfunction
 Forward and backward HF
 Forward and backward HF
 High-output HF
 Functional classes (NYHA)

ACCF/AHA STAGES OF HF

- Stage A: At high risk for HF but without structural heart disease or symtoms of HF
- Stage B: Structural heart disease but without signs or symptoms of HF
- Stage C: Structural heart disease with prior or current symptoms of HF
- Stage D: Refractory HF Requiring specialized interventions

ACCF/AHA guidelines, 2001

- ESC GUIDELINES FOR DIAGNOSTIC AND TREATMENT OF ACUTE AND CHRONIC HF (2016)
- Definition of heart failure with preserved (HFpEF), mid-range (HFmrEF) and reduced ejection fraction (HFrEF)
 - 1) LVEF < 40% with reduced EF
 - 2) LVEF 40-49% with mid-range EF
 - 3) LVEF > 50 % with preserved EF

ESC GUIDELINES FOR DIAGNOSTIC AND TREATMENT OF ACUTE AND CHRONIC HF (2016) In previous guidelines it was acknowledged that a grey area exists between HFrEF and HFpEF.7 These patients have an LVEF that ranges from 40 to 49%, hence the term HFmrEF. Identifying HFmrEF as a separate group will stimulate research into the underlying characteristics, pathophysiology and treatment of this group of patients. Patients with HFmrEF most probably have primarily mild systolic dysfunction, but with features of diastolic dysfunction

SYSTOLIC AND DIASTOLIC DYSFUNCTION



« FORWARD AND BACKWARD

HF»

In some patients with HF the predominant problem is an inadequate cardiac output (forward HF), whilst other patients may have a normal or near-normal cardiac output with marked salt and water retention causing pulmonary and systemic venous congestion (backward HF).

"HIGH OUTPUT CARDIAC FAILURE"

This can occur from:

- Severe anemia,
- Gram negative septicaemia,
- Beriberi (vitamin B1/thiamine deficiency), thyrotoxicosis,
- Paget's disease,
- arteriovenous fistulae, or arteriovenous malformations.

SKSTEQLIC purpy SELLINGTLON ecrease cardiac output, heart volume per minute decrease arterial pressure increase activity of sympatho-adrenal system, vasoconstriction of renal vessels deterioration of kidneys blood flow activation of renin-angiotensin-aldersone system increase NA reabsorbtion, hyperproducti retention of NA and water, increase ADH circulato ume increase ven turn increase diastolic full of LV ->>> DILATATION of the HEART and decrease cardiac output

SYSTOLIC DYSFUNCTION

- Coronary artery disease (CAD)
 Dilated cardiomyopathy (DCMP)
 Myocarditis
- Anti-cancer drugs (doxorubicin) and some toxins (alcohol)
- Heart valve disorders
- Arrhythmias (atrial fibrillation, tachycardia cardiomyopathy, complete AV block)



DIASTOLIC DYSFUNCTION

- Constrictive pericarditis, cardiac tamponade
- LV hypertrophy (hypertension)
- Restrictive cardiomyopathy

LEFT, RIGHT AND BIVENTRICULAR FAILURE



LEFT-SIDED FAILURE



- Dyspnea and suffocation
- Orthopnea
- Paroxysmal nocturnal dyspnea
- Peripheral cyanosis and coldness
- Tiredness, weakness, anxiety
- A weak, rapid pulse

PERIPHERAL CYANOSIS



RIGHT-SIDED FAILURE

- Fluid accumulation and swelling (edema) in the feet, ankles, legs
- Hepatomegaly
- Enlargement of abdomen (ascitis)
- Jugular vein distention
- A weak, rapid pulse



NEW YORK HEART ASSOCIATION (NYHA) FUNCTIONAL CLASSIFICATION OF CHF

- I class. Patients with cardiac disease but without resulting limitations of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, dyspnoea or anginal pain.
- Il class. Patients with cardiac disease resulting in Slight limitation of physical activity. They are comfortable at rest. Ordinary physical activity results in fatigue, palpitation, dysponea, or anginal pain.
- Ill class. Patients with cardiac disease resulting in marked limitation of physical activity. They are comfortable at rest. Less than ordinary physical activity causes fatigue, palpitation, dysponea or anginal pain.
- IV class. Patients with cardiac disease resulting in inability to can on any physical activity without discomfort. Symptoms of cardiac insufficiency or of the anginal syndrome may be present even at rest. If any physical activity is undertaken, discomfort is increased.

DIAGNOSIS OF HF

- Symptoms (underlying disease + HF)
- Physical examination (pulse, BP, abnormal heart sounds and fluid accumulation in the lungs, an enlarged heart, swollen neck veins, an enlarged liver, and swelling in the abdomen or legs)
- A chest x-ray (an enlarged heart and fluid accumulation in the lungs)
- ECG (tachycardia, low voltage, arrhythmias, blocks, ST depression)
- <u>Echocardiography</u>
- Level BNP
- Other procedures (radionuclide, magnetic resonance, or computed tomography imaging and cardiac catheterization with angiography)



HEART: - ENLARGEMENT OF CARDIAC SILHOUETTE	LUNGS: - RETICULAR SHADOWING - SEPTAL ('KERLEY B' LINES) - ENLARGED HILAR VESSELS - PLURAL EFFUSION
Heart Failure	Normal X-Ray

X-RAY EXAMINATION

Alcoholic Dilated Cardiomyopathy



ECHOCARDIOGRAPHY

2D Echo COCM Diastole



Ejection fraction < 40%

- LA > 40 mm EDV-LV > 55 mm
- EDV-RV > 26 mm

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IVS < 11 mm
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BLOOD TESTS

 B-type natriuretic peptide (BNP) is a specific test indicative of heart failure.

BNP > 35 pg/mL Pro-BNP>125 pg/ml + electrolytes (Na, K), + renal function, + liver function tests, + thyroid function tests, + complete blood count, + C-reactive protein

FRAMINGHAM CRITERIA

requires the simultaneous presence of at least 2 of the following major criteria or 1 major criterion in conjunction with 2 of the following minor criteria:

Major criteria:

- Cardiomegaly on chest radiography
- S3 gallop (a third heart sound)
- Acute pulmonary edema
- Paroxysmal nocturnal dyspnea
- Crackles on lung auscultation
- Central venous pressure of more than 16 cm H2O at the right atrium
- Jugular vein distension
- Positive abdominojugular test
- Weight loss of more than 4.5 kg in 5 days in response to treatment (sometimes classified as a minor criterium[31])

FRAMINGHAM CRITERIA

Minor criteria:

- Tachycardia of more than 120 beats per minute
- Nocturnal cough
- Dyspnea on ordinary exertion
- Pleural effusion
- Decrease in vital capacity by one third from maximum recorded
- Hepatomegaly
- Bilateral ankle edema

THE COURSE OF CHF

- Symptoms of heart failure may begin suddenly, especially if the cause is a heart attack (acute HF)
- Most people have no symptoms when the heart first begins to develop problems.
 Symptoms then develop gradually over days to months or years (chronic HF).
- The latest classification describes transient HF (at the peak of sudden overload with following normalization of function).

TREATMENT OF HEART FAILURE

Acute and chronic management strategies in heart failure are aimed at improving both symptoms and prognosis!

MANAGEMENT OF THE HEART FAILURE

The main purposes:

- 1. To reduce mortality !!!
- 2. To relieve HF symptoms
- 3. To slow down HF progress
- 4. To improve the quality of life (QOL)
- 5. To reduce duration of hospital treatment
- 6. To improve prognosis

GOALS OF TREATMENT

- To improve symptoms and quality of life
- To decrease likelihood of disease progression
- To reduce the risk of death and need for hospitalisation

THE MAIN PRINCIPLES OF HF MANAGEMENT

- To reveal and exclude triggering factors
- To normalise cardiac output
- To eliminate fluid retention in the body
- To reduce peripheral tension
- To reduce sympathoadrenal effects
- To improve blood supply and metabolism of myocardium

METHODS OF HF MANAGEMENT

- Non-medical (changing lifestyle)
- Pharmacotherapy (ACE inhibitors or ARBs, beta-blockers, aldosterone antagonists, diuretics, cardiac glycosides, ivabradine, anticoagulants, antiarrhythmic drugs, statins, cardiometabolic drugs)
- Mechanical (thoracocentesis, paracentesis, dialysis, ultrafiltration)
- Surgical (pace-makers, ICD (implantable cardioverter defibrillator), coronary revascularisation, heart transplantation)

PHARMACOTHERAPY FOR HF

- 1 DRUGS PROVED TO BE ABLE TO REDUCE MORBIDITY AND MORTALITY RATES IN CASE OF CHF EXACTLY
- a) used for all patients (ACE inhibitors or ARBs, beta-blockers, aldosterone antagonists);
- b) used under certain clinical conditions (diuretics, cardiac glycosides, ivabradine, anticoagulants);
- 2 DRUGS NOT INFLUENCING PROGNOSIS FOR CHF BUT RELIEVING SYMPTOMS IN CERTAIN CLINICAL SITUATIONS

(antiarrhythmic drugs, statins, calcium channel blockers (CCB), antiaggregants, cytoprotectants, vasodilators)

MANAGEMENT OF ACUTE LV FAILURE



MANAGEMENT OF ACUTE LV FAILURE

Second line drug treatment

Inotropes: β agonists (dobutamine)

Dopamine (low dose)

Inotropes: phosphodiesterase inhibitors (enoximone)

Intravenous aminophylline

Advanced management

Assisted ventilation

Circulatory assist devices

Increase myocardial contractility

Increases renal perfusion, sodium excretion, and urine flow

Increase myocardial contractility and venodilatation

Weak inotropic effect, diuretic effect, bronchodilating effect

Reduces myocardial oxygen demand; improves alveolar ventilation

Give mechanical support

Treatment of mild HF (Killip class II)

Recommendations		Level
Oxygen is indicated to maintain a saturation > 95%.		С
Loop diuretics, e.g. furosemide: 20-40 mg i.v., is recommended an should be repeated at 1-4 h intervals if necessary.		С
i.v. nitrates or sodium nitroprusside should be considered in patients with elevated systolic blool pressure.	lla	С
An ACE inhitibor is indicated in all patients with signs or symptoms of heart failure and/or evidence of LV dysfunction in the absence of hypotension, hypovolaemia, or renal failure.		A
An ARB (valsartan) is an alternative to ACE inhibitor particularly if ACE inhibitors are not tolerated.	1	в
An aldosterone antagonist (eplerenone) is recommended in all patients with signs or symptoms of heart failure and/or evidence of LV dysfunction provided no renal failure or hyperkalaemia.		В
Hydralazine and isosorbide dinitrate should be considered if the patient is intolerant to both ACE inhibitors and ARBs.	lla	С



European Heart Journal (2012) 33, 2569–2619 doi:10.1093/eurheartj/ehs215

Treatment of mild HF (Killip class III)

Recommendations		Level
Oxygen is indicated.		С
Ventilatory support should be instituted according to blood gasses.	1	С
Loop diuretics, e.g. furomeside: 20-40 mg i.v., are recommended and should be repeated at 1-4 h intervals if necessary.		С
Morphine is recommended. Respiration should be monitored. Nausea is common and an antiemetic may be required. Frequent low-dose therapy is advisable.	T	С
Nitrates are recommended if there is no hypotension.		С
Inotropic agents: • Dopamine;		С
Dobutamine (inotropic);		С
 Levosimendan (inotropic/vasodilator). 		С
An aldosterone antagonist such as spironolactone or eplerenone must be used if LVEF \leq 40%.		в
Ultrafiltration should be considered.		В
Early revascularisation must be considered if the patient has not been previously revascularized.	1	С
European Heart Journal (2012) 33, 2569–2619 doi:10.1093/eurheartj/ehs215		EUROP

CARDIO

Treatment of cardiogenic shock (Killip class IV)

Recommendations	Class	Level
Oxygen/mechanical respiratory support is indicated according to blood gasses.		С
Urgent echocardiography/Doppler must be performed to detect mechanical complications, assess systolic function and loading conditions.		С
High-risk patients must be transferred early to tertiary centres.	1	С
Emergency revascularization with either PCI or CABG in suitable patients must be considered.		В
Fibrinolysis should be considered if revascularization is unavailable.	lla	C
Intra-aortic balloon pumping may be considered.		В
LV assist devices may be considered for circulatory support in patients in refractory shock.		С
Haemodynamic assessment with balloon floating catheter may be considered.		в
Inotropic/vasopressor agents should be considered: • Dopamine;	lla	С
Dobutamine;	lla	С
 Norepinephrine (preferred over dopamine when blood pressure is low). 	llb	В
ww.escardio.org/guidelines European Heart Journal (2012) 33, 2569–2619		EURO

CARDIOLO

GENERAL MANAGEMENT OF CHRONIC HF

- Education of patient and relatives
- Diet: decrease of salt intake, good general nutrition
- Alcohol: elimination
- Smoking: stopping
- Weight: normalization
- Exercise: regular moderate aerobic within limits of symptoms
- Vaccination: influenza and pneumococcal

MANAGEMENT OF CHF <u>WITH SYSTOLIC DYSFUNCTION</u> <u>OF LV</u> (ESC GUIDELINES .2016)

Recommendations	Class ^a	Level ^b	Ref
An ACE-I ^d is recommended, in addition to a beta-blocker, for symptomatic patients with HFrEF to reduce the risk of HF hospitalization and death.	1	A	2, 163 –165
A beta-blocker is recommended, in addition an ACE-I ^d , for patients with stable, symptomatic HFrEF to reduce the risk of HF hospitalization and death.	1	A	167– 173
An MRA is recommended for patients with HFrEF, who remain symptomatic despite treatment with an ACE-I ^d and a beta-blocker, to reduce the risk of HE hospitalization and death	1	A	174, 175

Management of CHF *with systolic dysfunction of LV*

 1. Angiotensin-converting enzyme (ACE) inhibitors Enalapril 2,5-20 mg daily Lisinopril 2,5-20 mg daily Perindopril 5-10 mg daily Ramipril 2,5-10 mg daily
 This group decreases the risk of sudden death and cardiac mortality! (↓30-38%)

.2. Beta-blockers:

Bisoprolol 1,25 – 10 mg daily
Carvedilol 3,125 – 50 mg daily
Metoprolol 6,25 – 150 mg daily
Nebivolol 1,25 – 5 mg daily
This group decreases the risk of sudden death and cardiac mortality!!! (↓41-56%)

N°2 MANAGEMENT OF CHF <u>WITH SYSTOLIC DYSFUNCTION</u> <u>OF LV</u>

- **3.. Aldosterone receptor blockers:**
- Eplerenone 12,5 50 mg daily
- Spironolactone 12,5 25 mg daily
- This group decreases the risk of sudden death and cardiac mortality!!! (↓21-29%)

4.. Angiotensin II receptor blockers:

- Candesartan 4 32 mg daily
- Losartan *12,5 50 mg daily*
- Valsartan 20 320 mg daily
- This group decreases the risk of sudden death and cardiac mortality!!! (↓30%)

. 5. ARNI (Valsartan + Sacubitril) 100mg-200mg

N°3

Management of CHF with systolic dysfunction of LV

6.Diuretcs:Loop diuretics: Furosemide 40-500 mg daily, Ethacrynic acid 25-400 mg daily, Torasemide 10-20 mg daily Thiazide and thiazide-like diure

tics: Hypothiazide 25-75 m

- g daily, Indapamide 2,5
- -5 mg dailyK-sparing diuretics
- Spironolacton 25-100 mg daily, 7.. Digoxin: Tachysystolic form of atrial fib

rillation: 0,25 – 0,5 mg daily Sinus rhythm, CHF II

B-III: 0,125 – 0,25 mg daily8. Inhibitors of If-channels of SA node (Ivabradine) tab. 5-7,5 mg 2 t.dln the SHIFT study, ivabradine significantly reduced the risk of the primary composite endpoint of hospita

ADDITIONAL DRUGS

- Anti-aggregants: aspirin (100-300 mg daily)
- Anti-coagulants: warfarin (3-9 mg daily)
- Statins: atorvastatin (10-80 mg daily)
- Antiarrhythmic: amyodaron (200-400 mg daily) This group decreases the risk of sudden death and cardiac mortality!!! (\ 29%)

MANAGEMENT OF CHF <u>WITH NORMAL SYSTOLIC</u> <u>FUNCTION OF LV</u>

Main group:

- Angiotensin-converting enzyme inhibitors
- Beta-blockers
- Angiotensin II receptor blockers

Reserve drugs:

- 1. Diuretcs
- 2. Ca antagonists

SURGICAL TREATMENT

The following procedures decrease the risk of sudden death and cardiac mortality:

- Implantation of ICD (130%)
- Cardiac resynchronization therapy (CRT)
- Heart transplantation

Contraindications:

- age 65 or older
- another medical condition that could shorten life
- Poor blood circulation
- Personal medical history of cancer
- Mechanical heart support



Take care of your heart!





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