HEART FAILURE
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Group 4

HEART FAILURE

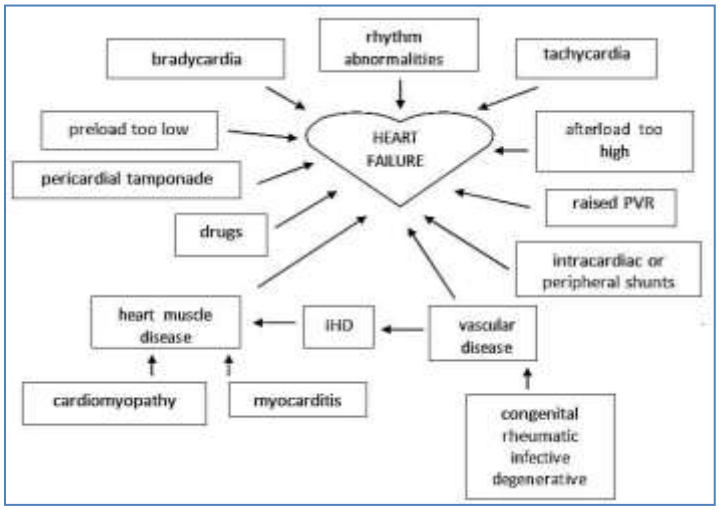
- A complex clinical syndrome that can result from any structural or functional cardiac disorder that impairs the ability of the ventricle to fill with or eject blood ACC/AHA Guidelines

Heart failure is characterized by generalized adrenergic activation and parasympathetic withdrawal

Clinical syndrome could either be structural or functional

- Final common pathway for many cardiovascular diseases whose natural history results in symptomatic or asymptomatic left ventricular dysfunction
- When you diagnose heart failure, identify the cause of heart failure (ex. valvular, myocardial)
- 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
- Heart failure vs. Congestive heart failure: not all patients have VOLUME OVERLOAD at the time of initial or subsequent evaluation, the term "heart failure" is preferred over the older term "congestive heart failure"
- NOT a disease but a manifestation of a disease

Main Causes:



- **Coronary Artery Disease**

Patients with underlying CAD are at risk due to myocardial ischemia. Even in patients for whom acute ischemia is not a precipitating factor, the substrate of hibernating or stunned myocardium may play a major pathophysiologic role because such patients maybe more susceptible to myocardial injury as a result of the AHF episode or treatment.

- **Hypertension** – also a risk factor for CAD

The increase in BP is most likely driven by an increased LV filling pressure and further activation of the sympathetic nervous system and RAAS.

Reactive hypertension – an indirect measure of cardiac reserve. It a relatively rapid normalization and improvement of blood pressure in response to diuretic therapy.

Severe hypertension may be the cause rather than a result of acute heart failure and may precipitate pulmonary edema. This "acute hypertensive emergency" occurs most frequently in patients with susceptible underlying substrate (e.g., diastolic dysfunction due to LV hypertrophy).

- **Valvular heart disease** – all valvular disease can lead to heart failure
  - In valvular regurgitant lesions, the heart will eventually fail as the patient is chronically volume overloaded. Intervene when the heart starts to fail, not too late nor not too early since changing to metallic valves will subject the patient to chronic prolonged anticoagulation.
  - Anticoagulation is a risk for bleeding.
  - Warfarin excess -> cerebral bleed
  - Antifungals generally have an interaction with Coumadin, prolonged protime.
- **Cardiomyopathy**

Patients with chronic HF and reduced ejection fraction are known to have variable degrees of viable but dysfunctional myocardium (VDM). It could still be salvageable through therapy such as beta blockers and revascularization, since the cells has not still loss the cell membrane and mitochondrial integrity and still exhibit preserved glucose metabolism and contractile reserve.

- **Cor pulmonale** – R-sided heart failure due to a primary pulmonary disease

At night, there is a shift of fluid from intracellular to extravascular space thus, more fluid volume. Proteins are increased in ARDS (acute respiratory distress syndrome) which cause pulmonary edema.

FORMS OF HEART FAILURE

1. Systolic vs. Diastolic Failure

- systolic and diastolic failure coexist in most patients with HF

Systolic Heart Failure

- Inability of the ventricle to contract normally with symptoms resulting from inadequate cardiac output
- Ejection Fraction: <40% (as seen in echo and cardiac catheterization)
- Failure of the heart to pump blood at the rate commensurate with the requirements of the metabolizing tissues
- PE: prominent S3 (time when the ventricle has a rapid filling)
- Ventricles contract very weakly

Causes:

- Contractile dysfunction:
  - Ischemic Heart disease
  - Cardiomyopathy
- Volume overload
  - aortic regurgitation
  - mitral regurgitation
  - VSD

There is a problem in contractility. If a muscle has ischemia, there loss of heart muscle because of infarction

Cardiomyopathy- due to weakening, may be because of infection, causes include myocardial endocarditis, chronic alcoholism, pregnancy

There is eccentric hypertrophy - dilatation of the heart muscle occurs so wall become thinner and cavity becomes bigger—bigger in xray

Sustained neurohormonal activation results in transcriptional and posttranscriptional changes in the genes and proteins that regulate excitation-contraction coupling and cross-bridge interaction.
Collectively these changes impair the ability of the myocyte to contract and therefore contribute to the depressed LV systolic function observed in patients with HF.

Diastolic Heart Failure

- Inability of the ventricle to relax and fill normally with symptoms from elevated filling pressures
- Ejection Fraction: >50%
- Increased resistance to ventricular filling leading to elevated ventricular pressures in a ventricle of normal dimensions

Systolic function is normal but the ventricle is stiff therefore it is not able to relax and fill normally. With symptoms from elevated filling pressure. Clinically, they may be difficult to distinguish.

PE: S4 (atrium has to contract very strongly to counter the stiff ventricles)

There may be concentric hypertrophy

An increase in heart rate disproportionately shortens the time for diastolic filling, which may lead to elevated LV filling pressures, particularly in non-compliant ventricles.
Elevated LV end-diastolic filling pressures result in increases in pulmonary capillary pressures, which can contribute to the dyspnea experienced by patients with diastolic dysfunction. Importantly, diastolic dysfunction can occur alone or in combination with systolic dysfunction in patients with HF.

Diastolic Heart Failure	
Increased resistance to atrial emptying	Mitral stenosis, Tricuspid stenosis
Increased resistance to ventricular inflow, restrictive pericardial compliance	Constrictive pericarditis
Reduced ventricular compliance, Hypertrophic HD	Hypertension, Aortic stenosis, Hypertrophic cardiomyopathy
Restrictive cardiomyopathy	Loeffler's syndrome
Specific Heart muscle disease	Amyloidosis, Hemachromatosis
Rate of ventricular Relaxation	Ischemic heart disease

Parameters	Systolic	Diastolic
History		
CHD	++++	+
HTN	++	++++
DM	+++	+
Valvular Heart disease	++++	-
Paroxysmal dyspnea	+++	+++
Physical Examination		
Cardiomegaly	+++	+
Soft heart sounds	++++	+
S3 gallop	+++	+
S4 gallop	+	+++
HTN	++	++++
MR	+++	+
Rales	++	++
Edema	+++	+
Venous distention	+++	+

Chest Roentgenogram		
Cardiomegaly	+++	+
Pulmonary congestion	+++	+++
ECG		
Low voltage	+++	-
LVH	++	++++
Q waves	++	+
Echocardiogram		
Low EF	++++	+
LV dilation	++	+
LVH	++	++++

2. Low-output vs. high-output HF

<u>Low-output HF</u>

- Cardiac output at rest < 2.2L/min per m^2 (lower limit of normal) and fails to increase normally with exertion
- Seen after MI, HTN, dilated cardiomyopathy, valvular or pericardial disease
- Often accompanied by vasodilation and warm extremities

<u>High-output HF</u>

- CO>3.5 L/min/m^2 or upper limit of normal (before development of HF)
- Seen in hyperthyroidism, anemia, pregnancy, AV fistula, beriberi, Paget's disease, usually with underlying heart disease *(common examination question! High vs. low output HF)*

3. Left-sided vs. Right-sided HF

<u>Left-sided HF</u>

- Left ventricle is hemodynamically overloaded and/or weakened, resulting in pulmonary congestion (exertional dyspnea, orthopnea, paroxysmal nocturnal dyspnea)

<u>Right-sided HF</u>

- Abnormality primarily affecting RV, resulting in edema (high JVP), congestive hepatomegaly, and systemic venous distention (ascites, pedal edema, anasarca)

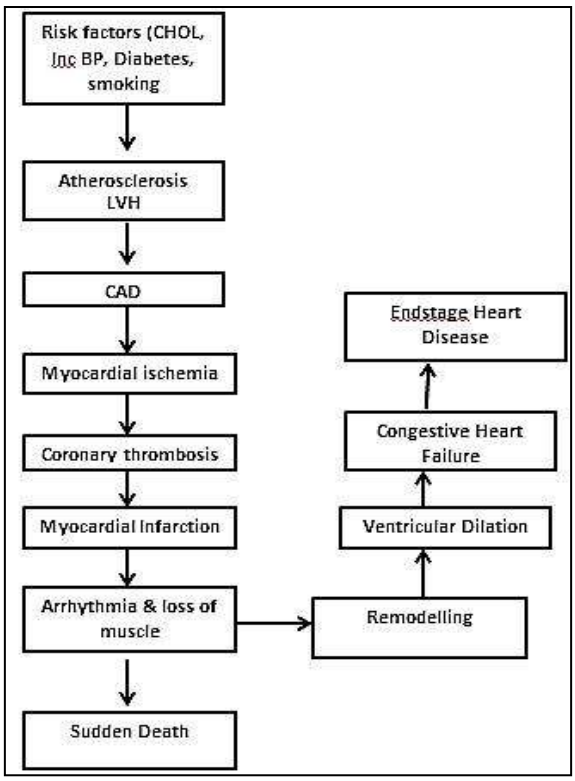
Common cause: LEFT SIDED HEART FAILURE - rare to see RSHF without LSHF, example in patients with pulmonary hypertension due to COPD or pulmonary vascular problem (Pulmonary embolism) causing COR PULMONALE, a RSHF due to a pulmonary problem, not LSHF

Note: before diagnosing Primary Pulmonary Hypertension always exclude all other possible causes.

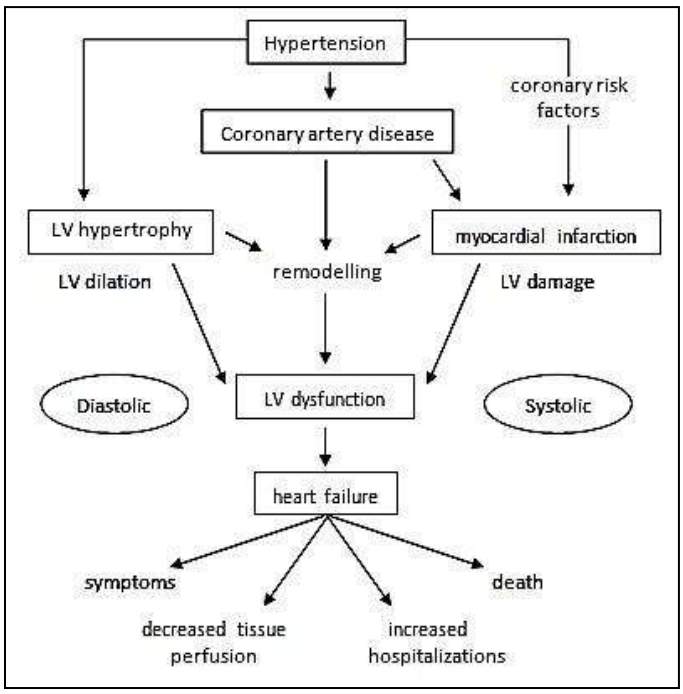
- Example: undiagnosed PDA presenting as RSHF with HTN; end stage continuous murmur and usual findings disappear, Eisenmenger syndrome

Eisenmenger Syndrome - any untreated congenital cardiac defect with intracardiac communication that leads to pulmonary hypertension, reversal of flow, and cyanosis. The previous left-to-right shunt is converted into a right-to-left shunt secondary to elevated pulmonary artery pressures and associated pulmonary vascular disease)

Cardiovascular Continuum Focusing on CAD as Cause of Heart Failure



"Chronic ischemia alone can weaken the heart in time, no need for repeated MI (or recurrent heart attack)"

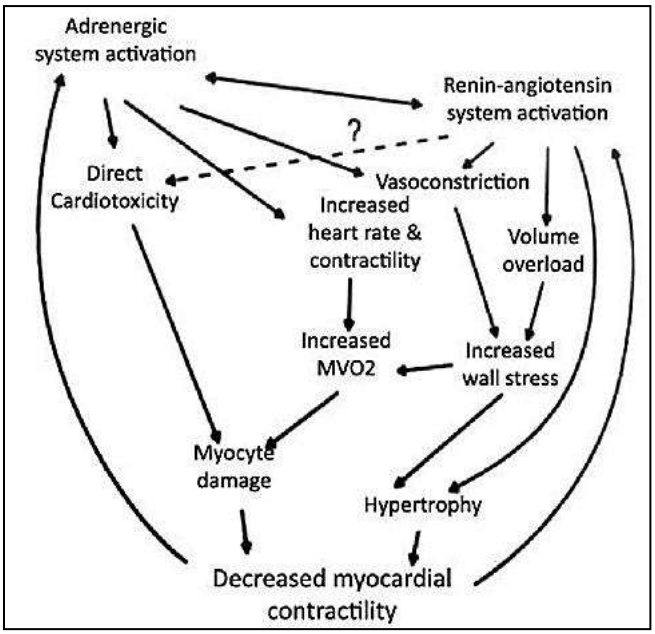


Compensatory Changes in HF:

- Activation of sympathetic nervous system
- Activation of renin-angiotensin system
- Release of anti-diuretic hormone
- Release of atrial natriuretic peptide
- Chamber enlargement
- Myocardial Hypertrophy

While they are compensatory at first, later they will also worsen the condition. It is a vicious cycle.

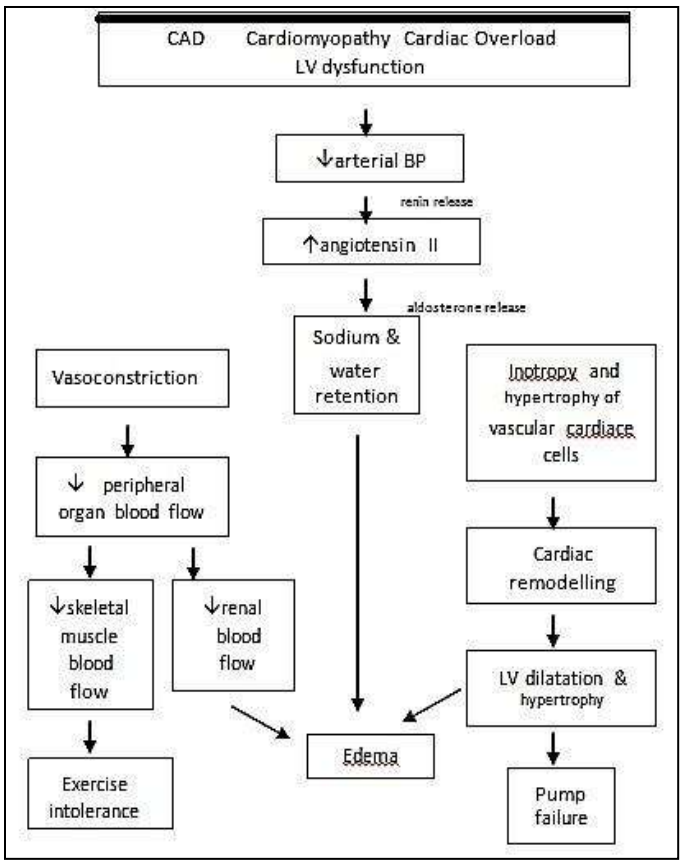
Heart Failure Compensatory Mechanisms



Effect of SNS activation in heart failure:

- Dysfunction/death of cardiac myocytes
- Provokes myocardial ischemia
- Provokes arrhythmias
- Impairs cardiac performance
- These effects are mediated via stimulation of β and α1 receptors.

Role of Angiotensin II in the Progression of Heart Failure



NYHA Classification of HF

- Class I: No limitation of physical activity
- Class II: Slight limitation of physical activity
- Class III: Marked limitation of activity
- Class IV: Unable to carry out physical activity without discomfort

Stages of Heart Failure

At risk for Heart failure

- Stage A: High risk for developing HF
- Stage B: Asymptomatic LV dysfunction

Heart Failure:

- Stage C: Past or current symptoms of HF
- Stage D: End-stage HF

- Designed to emphasize PREVENTABILITY of HF
- Designed to recognize the PROGRESSIVE NATURE of LV dysfunction
- Complement, do not replace NYHA classes
- NYHA classes - shift back/forth in individual patient (in response to Rx and/or progression of disease)

Factors Aggravating Heart Failure:

- Myocardial ischemia or infarct
- Dietary sodium excess
- Excess fluid intake
- Medication noncompliance
- Arrhythmias

AF can cause congestion

- Intercurrent illness(i.e. infection)
- Conditions associated with increased metabolic demand

Pregnancy, px with HF are not advised to get pregnant because the heart cannot cope with the metabolic demands of pregnancy

Thyrotoxicosis

Excessive physical activity

- Administration of drug with negative inotropic properties or fluid retaining properties

NSAIDS and steroids

- Alcohol

Upon cessation of alcohol intake there is a dramatic improvement

Precipitating factors in chronic heart failure:

- Noncompliance (diet)
- Noncompliance (drugs)
- Noncompliance (both diet and drugs)
- Myocardial infarction
- Pulmonary infection
- Inadequate therapy
- Arrhythmias
- Hypertension
- Others

SIGNS AND SYMPTOMS

- Dyspnea with exertion(early) or at rest (late)
- Orthopnea
    - Dyspnea when recumbent; relief with sitting upright or use of several pillows
- Paroxysmal nocturnal dyspnea
    - Coughing and wheezing often persist even with sitting upright
    - Cardiac asthma: nocturnal dyspnea, wheezing and cough due to bronchospasm
- Fatigue and weakness
- Abdominal symptoms
    - Anorexia

    - Nausea
    - Abdominal pain and fullness – may be a sign of congestive hepatomegaly
- Cerebral symptoms
    - Altered mental status due to reduced cerebral perfusion
    - Confusion
    - Difficulty concentrating
    - Impaired memory
    - Headache
    - Insomnia
    - Anxiety
- Nocturia

Common because of the shift of fluid to the intravascular space, thus more renal perfusion

PHYSICAL FINDINGS

- **Pulmonary rales with or without expiratory wheeze**

Result from the transudation of fluid from the intravascular space into the alveoli. In pulmonary edema, rales maybe heard widely over both lung fields and may be accompanied by expiratory wheezing. Rales are frequently absent in patients with chronic HF.

- **Lower extremity edema**
- **Hydrothorax** (pleural effusion)

Result from the elevation of pleural capillary pressure and the resulting transudation of fluid into the pleural cavities. Since the pleural veins drain into both the systemic and pulmonary veins, pleural effusions occur most commonly with biventricular failure.

- **Ascites**

Most common in constrictive pericarditis and tricuspid valve disease. Ascites, a late sign, occurs as a consequence of increased pressure in the hepatic veins and the veins draining the peritoneum

- **Congestive hepatomegaly**

Hepatomegaly is an important sign in patients with HF. When present, the enlarged liver is frequently tenderand may pulsate during systole if tricuspid regurgitation is present.

- **Positive abdominojugular reflex**

In the early stages of HF, the venous pressure may be normal at rest but may become abnormally elevated with sustained (~1 min) pressure on the abdomen (positive abdominojugular reflux.

- **Jugular venous distention**
- **S3 and S4 heart sounds**, often present but not specific

An S3 (or protodiastolic gallop) is most commonly present in patientswith volume overload who have tachycardia and tachypnea, and it oftensignifies severe hemodynamic compromise. A fourth heart sound (S4) is not a specific indicator of HF but is usually present in patients with diastolic dysfunction.

- **Elevated diastolic arterial pressure**

If LV filling is delayed because LV compliance is reduced (e.g., from hypertrophy or fibrosis), LV filling pressures will similarly remain elevated at end diastole. An increase in heart rate disproportionately shortens the time for diastolic filling, which may lead to elevated LV filling pressures, particularly in noncompliant ventricles.

- **Depression**
- **Sexual dysfunction**
- **Findings in late severe HF**
- **Diminished pulse pressure**
- **Pulsus alternans**
    - Regular rhythm w/ alternation in strength of peripheral pulses
    - Most common in cardiomyopathy, HTN, IHD
- **Jaundice**

A late finding in HF, results from impairment of hepatic function secondary to hepatic congestion and hepatocellular hypoxia, and is associated with elevations of both direct and indirect bilirubin.

- **Decreased urine output**
- **Cardiac cachexia**

Although the mechanism of cachexia is not entirely understood, it is likely multifactorial and includes elevation of the resting metabolic rate; anorexia, nausea, and vomiting due to congestive hepatomegaly and abdominal fullness; elevation of circulating concentrations of cytokines such as TNF; and impairment of intestinal absorption due to congestion of the intestinal veins. When present, cachexia augers a poor overall prognosis.

DIFFERENTIAL DIAGNOSIS

HF resembles but should be distinguished from:

1. Conditions in which there is circulatory congestion secondary to abnormal salt and water retention but in which there is no disturbance of cardiac structure or function (e.g., renal failure) and

2. Noncardiac causes of pulmonary edema (e.g., acute respiratory distress syndrome). In most patients who present with classic signs and symptoms of HF, the diagnosis is relatively straightforward.

- Pulmonary disease with dyspnea
- Obstructive airway disease
- Diffuse parenchymal lung disease
- Pulmonary vascular occlusive disease
- Disease of chest wall and respiratory muscles
- Cardiac asthma: wheezing secondary to bronchospasm occurring at night
- Other conditions leading to peripheral edema:
  - Varicose veins, cyclic edema, or gravitational effects: no jugular venous HTN
  - Renal disease: abnormal renal function tests, urinalysis
  - Elevation of venous pressure is uncommon
- Hepatic cirrhosis
  - Enlargement of liver
  - Ascites
  - Normal Jugular venous pressure
  - Negative abdominojugular reflex

DIAGNOSTIC APPROACH

Approach to patient:

- Detailed history and clinical examination (basic in any medical problem: go clinical before any laboratory)
- 2D-Echo with Doppler studies
- ECG
- Chest radiography
- Brain Natriuretic Peptide (BNP) measurement - help you identify if dyspnea is pulmonary or cardiac in origin

FRAMINGHAM CRITERIA FOR CHF

- Usually used for academic purposes
- To establish a clinical diagnosis of CHF, at least 1 major and 2 minor criteria are required:

Major Criteria

- Paroxysmal nocturnal dyspnea
- Neck vein distention
- Rales
- Cardiomegaly
- Acute pulmonary edema
- S gallop
- Increased venous pressure
- Positive hepatojugular reflux

Minor Criteria

- Extremity edema
- Night cough
- Dyspnea on exertion
- Hepatomegaly
- Plueral effusion
- Vital capacity reduced to one-third from normal
- Tachycardia (greater than or equal to 120 bpm)

Major or Minor Criteria

- Weight loss of greater than or equal to 4.5 kg over 5 days of treatment

Framingham is a town in Massachusetts where they started to study the population there since 1940s and traced these population through the years until now, thus able to identify the risk factors for coronary heart disease

LABORATORY TESTS

- ECG
  - Aids in determining etiology e.g. abnormal Q waves in old MI, LVH in hypertension
- BNP Measurement
  - >200 pg/ml supports diagnosis
  - <40 pg/ml rarely seen in HF
  - Useful in diagnosis, prognosis, and monitoring therapy
  - Helps in differentiating between cardiac and pulmonary causes of dyspnea
- Urinalysis
  - Albuminuria (renal function)
  - High specific gravity
  - Low sodium level

- Renal Function
  - Prerenal azotemia
- Electrolytes
  - Hypokalemia from thiazide diuretics
  - Hyperkalemia from potassium-retaining diuretics
  - Dilutional hyponatremia in late HF

To monitor imbalances e.g. due to medications

- Liver Function Testing
  - Hepatic enzymes frequently elevated
  - Elevated direct and indirect bilirubin levels (late finding)
- 2-Dimensional Echocardiography with Doppler Flow
  - To determine underlying causes
  - To assess severity of ventricular systolic and/or diastolic dysfunction and valvular dysfunction
  - Question diagnosis if all cardiac chambers normal in volume, shortening and wall thickness

Some ER like in heart center has echocardiogram already disadvantage: patient is not examined anymore

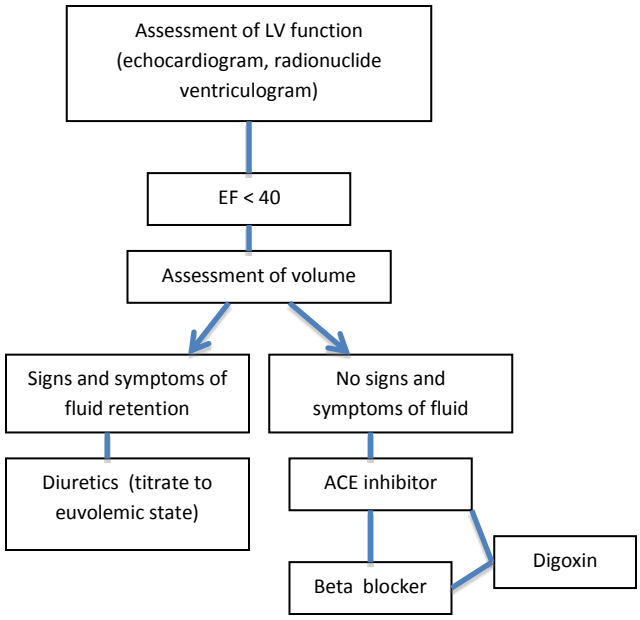
- Chest Radiography
  - To detect cardiomegaly and pulmonary congestion

To rule out concomitant pulmonary problem e.g in patient with HCM. Thick myocardium as in hypertension but usually patient is not hypertensive. This is used as diagnosis when thickness of myocardium cannot be explained by the presence of hypertension and aortic stenosis.

Dilated myocardiopathy –normal heart thickness but dilated cavity

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 hospitalization



THERAPY BY DISEASE STAGE

STAGE A

- Treat hypertension
- Prescribe ACE inhibitor especially in hypertension
- Encourage smoking cessation *(change unhealthy lifestyle)*
- Treat lipid disorders
- Encourage regular exercise
- Discourage alcohol intake and illicit drug use

Use of antihypertensives depends on comorbidities "compelling indications", example: hypertensives with LV dysfunction ACEI is #1 drug, if you can't use it, use ARB, also in patient with DM prioritize use of ACEI (protects kidney). ACEI also delays remodeling in patient post MI.

STAGE B

- All measures under stage A
- Add beta-blocker (because px is already symptomatic)

STAGE C

- All measures under stages A and B
- Add diuretic
- Add digitalis in systolic HF (DIGOXIN)
- Add spironolactone *(an aldosterone antagonist)*
- Restrict fluid and salt to <2 g/d (eliminate salt-rich foods and added salt in cooking or at table)

STAGE D (end stage heart disease)

- All measures under A, B, and C
- Dietary salt restriction to < 1 g/d
- Mechanical assist devices *(advise patient for heart transplant)*
- Heart transplantation
- Continuous intravenous inotropic infusions for palliation (does not prolong life) example: DOBUTAMINE drip
- Hospice care (make patient comfortable before death)

SPECIFIC TREATMENTS: GENERAL MEASURES

- Treat hypertension
- Treat lipid disorders
- Encourage smoking cessation
- Discourage alcohol intake and illicit drug use
- Recommend influenza and pneumococcal vaccines
- Achieve optimal weight
- Activity
    - **Compensated HF:** regular isotonic exercise in compensated HF
    - **Moderately severe chronic HF:** additional rest on weekend, scheduled naps or rest periods, avoidance of strenuous exertion. Avoid extremes and tiring trips

Regular isotonic or aerobic exercise – ideally 3-5 times a week for 30 minutes to 1 hour, ex. walking, jogging, swimming, biking.

Not isometric – muscles become big e.g. weightlifting – not the kind of exercise that is good for the heart (good for body figure only)

- Diet - reduce sodium intake (normal diet contains 6-10 g of sodium daily)
    - Intake can be halved by excluding salt-rich foods and eliminating salt table
    - Can be reduced to one- quarter with above measures and omitting salt from cooking
    - In severe HF - limit to 1 g/day
    - Late in course – often, both sodium and water intake must be restricted

- DIGITALIS: has no role in diastolic dysfunction
- Lanoxin – has been proven in many studies that improves survival because of its inotropic effect but has no role in diastolic dysfunction.

DIGOXIN

- Enhances LV function, normalizes baroreceptor-mediated reflexes and increases cardiac output at rest and during exercise
- Recommended to improve clinical status of patients with heart failure due to LV dysfunction and should be used in conjunction with diuretics, ACE inhibitors and beta-blockers
- Also recommended in patients with heart failure who have atrial fibrillation
- Digoxin initiated and maintained at a dose of 0.25 mg daily
- Adverse effects induce cardiac arrhythmias, GI symptoms and neurological complaints (e.g. visual disturbances, confusion)

Is digitalis helpful in heart failure with normal sinus rhythm? YES
How about ACEI – no doubt that it has an important role in heart failure, especially after MI

META-ANALYSIS OF ACEI TRIALS IN CHF (32 trials, 7105 patients)

- ACEI significantly reduce death and hospitalization for CHF
- Trend towards reduction in sudden death, fatal MI and stroke
- Benefit a class effect
- Reduction in mortality and hospitalization consistent regardless of age, sex, etiology and NYHA FC
- The lower the EF, the greater the benefit
- The earlier the better
- The higher the dose the better

STUDY	HOSPITALIZATION CAUSE	PATIENT'S HOSPITALIZED		RR REDUCTION
		PLACEBO	BETA BLOCKER	
MDC	Heart failure-related hospitalization	37%	28%	24%
CIBIS (I)	Heart failure-related hospitalization	28%	19%	32%
ANZ	All cause	58%	48%	23%
US Carvedilol	All cause	27%	19%	29%
	Heart Failure	09%	06%	38%
CIBIS (II)	All cause	39%	33%	20%
	Heart Failure	18%	12%	36%
MERIT-HF	Cardiovascular	39%	33%	16%
	Heart Failure	23%	16%	30%

YUSUF et.al. JAMA 1995

ACE INHIBITORS

PHYSIOLOGIC BENEFITS:

- Arteriovenous Vasodilation
  - decrease pulmonary arterial diastolic pressure
  - decrease pulmonary capillary wedge pressure
  - decrease left ventricular end-diastolic pressure
  - decrease systemic vascular resistance
  - decrease systemic blood pressure
  - decrease maximal oxygen uptake (MVO2)
  - increase LV function and cardiac output
  - increase renal, coronary, cerebral blood flow
  - no change in heart rate or myocardial contractility
  - no neurohormonal activation
  - resultant diuresis and natriuresis

CLINICAL BENEFITS:

- Increases exercise capacity
- Improves functional classification
- Attenuation of LV remodeling post MI
- Decrease in the progression of Chronic HF
- Decreased hospitalization
- Enhanced quality of life
- Improved survival

GUIDELINES TO ACE INHIBITOR THERAPY:

- Contraindications
  - Renal artery stenosis
  - Renal insufficiency (relative)
  - Hyperkalemia
  - Arterial hypotension
  - Cough
  - Angioedema (if they are sensitive)
- Alternatives
  - Hydralazine + ISDN, ARB
- All patients with symptomatic heart failure and those in functional class I with significantly reduced left ventricular function should be treated with an ACE inhibitor, unless contraindicated or not tolerated
- ACE inhibitors should be continued indefinitely
- It is important to titrate to the dosage regimen used in the clinical trials, in the absence of symptoms or adverse effects on end-organ perfusion
- In very severe heart failure, hydralazine and nitrates added to ACE inhibitor therapy can further improve cardiac output

BETA-BLOCKERS

- Reduce myocardial O2 demand with its negative inotropic effect
- Protect myocardium against catecholamine-induced damage
- Increased myocardial B-receptors density

META-ANALYSIS OF BETA BLOCKERS TRIAL IN CHF
(17 trials, 3039 patients)

- Greater treatment affect for non-sudden cardiac death
- Similar mortality reduction with ischemic and non-ischemic cardiomyopathy *(early studies was only about non-ischemic cardiomyopathy)*
- Greater survival benefit with carvedilol beta-blocker reduced all-cause mortality

Heidenreich et.al. JACC 1997

MANAGEMENT OF COMPLICATIONS:

- You can manipulate the medications, vasodilator and diuretics can be given but be careful with bradycardia. Also, be careful of bronchial obstruction with beta blockers.
- Diuretic is very important tool. Urination of more than 1 liter has a very dramatic improvement, ex. patient with fluid retention.

Next thing to do if patient shows no improvement? Heart transplant

Left ventricular device – attached to the patient before he undergoes heart transplant.

All heart transplantation performed in the Philippines: all patients died

- Transient worsening of heart failure (e.g. increasing dyspnea, decreasing exercise capacity)
- Increase dose of diuretics and/or ACE inihibitor
- If necessary, reduce carvedilol dose and/or prolong titration interval
- Search for other possible causes (e.g. thyroid malfunction, infection, non-compliant drug intake, excessive liquid intake, etc.)

- Vasodilatory Symptoms (dizziness, light headedness, symptomatic hypotension)
- Decrease diuretic dose and, if necessary, ACE inhibitor dose
- If the cessation of both is not successful, reduce carvedilol dose and/or prolong titration interval

- Bradycardia (pulse rate below 55 beats per minute)
- Check and eventually reduce digitalis dose
- If necessary, reduce carvedilol dose and/or prolong titration interval
- Withdraw carvedilol only in the event that hemodynamics are affected

- Symptoms of Bronchial Construction
- Search for other possible causes (e.g., concurrent infection, subacute pulmonary edema)
- Reduce dose or withdraw, carvedilol only after possible causes for symptoms have been ruled out

- Diuretics
- Indicated in patients with symptoms of heart failure who have evidence of fluid retention
- Enhance response to other drugs in hearts failure such as beta-blockers and ACE inhibitors
- Therapy initiated with low doses followed increments in dosage until urine output increases and weight decreases by 0.5-1kg daily

SUMMARY OF DRUG TREATMENT FOR CHF		
ASYMPTOMATIC LV DYSFUNCTION	MILD TO MODERATE CHF	MODERATE TO SEVERE CHF
ACE Inhibitor	Digoxin	Digoxin
Beta blocker	Diuretics	Diuretics
	ACE Inhibitor	ACE Inhibitor
	Beta blocker	Spironolactone

- References: Harrison's IM, Braunwald's, JAX notes, audio, powerpoint slides
- Compiled by: Alcantara, Catague, Dizor, Lumasag, Iwag, Sameon