جامعة الملك سعود
King Saud University

Lecture 9
Heart failure and venous pulse

- Red: important
- Black: in male / female slides
- Pink: in female slides only
- Blue: in male slides only
- Gray: extra information

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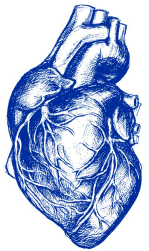
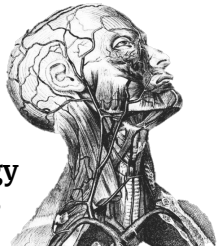


Physiology
MED438



Objectives:

- Define Jugular Venous Pressure.
- Identify different transmitted waves in the JVP record.
- Define and classify heart failure (HF).
- Understand the etiology of heart failure.
- Summarize clinical picture of left-sided and right-sided failure.
- Recognize the factors aggravating HF
- Understand the pathophysiology & compensatory mechanisms of Heart Failure.

Jugular Venous Pulse (JVP)

Reflection of:

The hemodynamics of the right sided heart:
Phasic pressure changes in the RA during systole & the RV during diastole.

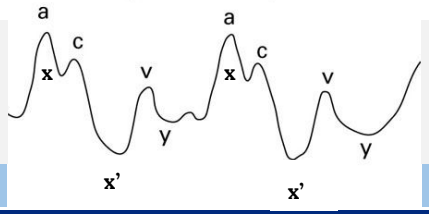
Best measured from the Right Internal Jugular Vein (IJV). Why?

- It is in **direct continuation** & **anatomically closer** to the RA.
- Extend in an almost **straight line** through innominate vein to the superior vena cava & RA.
- Is **less likely affected** by extrinsic compression from other structures in the neck.
- Has no or **less number of valves** than the External Jugular Vein (EJV) which favors transmission of the RA pressure.
- **Less impact of vasoconstriction** (sympathetic activity) on IJV than EJV.

Transmission of pulsations:

Pulsations produced in **the central veins** transmitted → **jugular veins**, producing pulsations and pressure waves in the jugular veins.

a - c and v
= 1 Cardiac cycle =
0.8 sec
3 Positive Wave

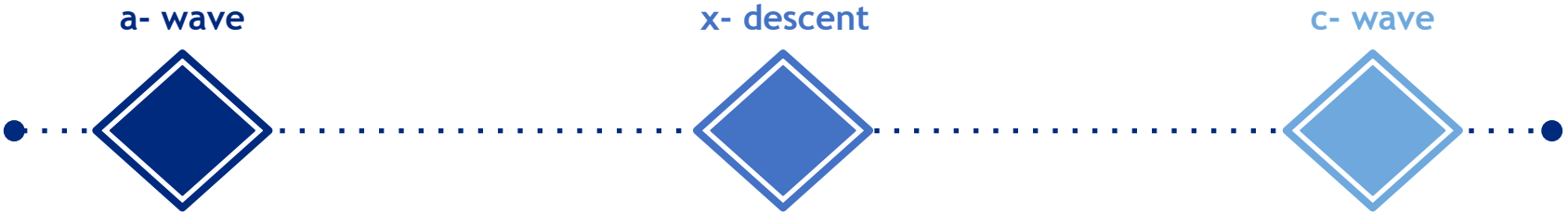


x - x' and y
(downward deflections)
3 Descents

After y- descent (during **diastasis**) there is **h-wave**:
+ve wave seen in long diastole (slow HR)

Causes of JVP waves

Female slides



Atrial systole

- **First** +ve presystolic wave due to ↑ in atrial pressure during effective **RA systole.**
- Results in **retrograde blood flow** in to the SVC & jugulars.
- Dominant wave & is > v- wave.
- It follows the **P- wave in ECG.**

It precedes:

- Upstroke of the carotid pulse.
- The first heart sound (**S1**).

Atrial diastole

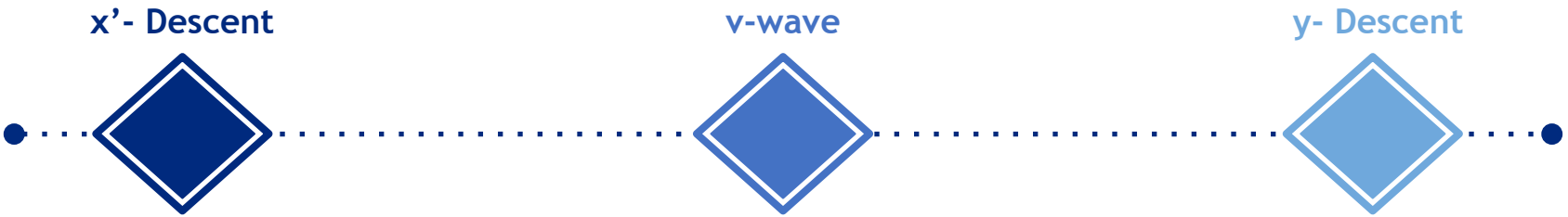
- Begins during V.systole & ends just before the second heart sound (**S2**). (Systolic collapse)
- **Most prominent motion** of normal JVP (especially during inspiration).
- It is > y- descent

Isovolumic ventricular contraction

- **Second** +ve wave recorded in JVP which **interrupts** the x-descent.
- Produced by:
  I. **Upward bulging** of closed tricuspid valve **(TV)** into the RA during **isovolumic ventricular contraction.**
  II. Carotid artery impact on JVP.

Causes of JVP waves cont

Female slides



Early RV systole

- It is systolic trough after c-wave.

Due to:

- **Downward pulling** of the TV by contracting right ventricle. **(rapid ejection phase).**
- **Descent of RA floor** by contracting RV.
- **Fall of RA pressure** during early RV systole.

Late RV systole

- **Third** +ve wave in JVP which begins in late systole & ends in early diastole.
- Rise in the RA pressure due to continued (venous return) during ventricular systole when **tricuspid valve closed.**
- It is roughly **synchronous** with carotid upstroke & corresponds to the second heart sound **(S2).**

RV Diastole

- Diastolic collapse wave (down slope).
- It begins & ends during **diastole** & after second heart sound **S2.**
- Decline of RA pressure due to RA emptying during early diastole when **tricuspid valve opens.**

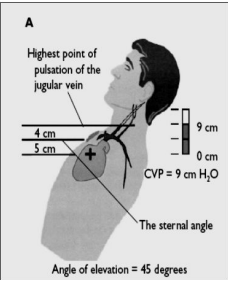
Initial y- descent → corresponds to the **rapid RV filling.**
later part → produced by **continued diastolic inflow** in to RV.

Internal Jugular vein & Carotid Pulses

Female slides

IJV	Carotid pulse
• **Superficial** and **lateral** in the neck. • Better **seen** than felt. • Has **two peaks** and **two troughs.** • Descents > obvious than crests. • Digital compression: **abolishes** venous pulse. • JVP **falls** during inspiration & standing. • Abdominal compression ↑ jugular pressure.	• **Deeper** and **medial** in the neck. • Better **felt** than seen. • Has **single upstroke** only. • Upstroke brisker and visible. • Digital compression: has **no effect.** • Do **not change** with respiration or standing. • Abdominal compression has **no effect.**

JVP Measurement



1 Patient lies supine and at 45 degrees.

2 Two scale method:
A. **horizontal scale** at the top of the oscillating venous column in IJV cuts.
B. **vertical scale** at the sternal angle.

3 **JVP=** Vertical distance that you measured (normally= 4cm) + distance between midpoint of RA & sternal angle= 5cm.

> 4cm (elevated JVP)

4 Thus, 4cm + 5cm = 9cm
By conversion → normal mean JVP (9 cm column of water/1.3 =6.9 mmhg

Abnormalities of jugular venous pulse

Male slides

A- Low jugular venous pressure

- Hypovolemia.

B- Raised Jugular Venous Pressure

- Increased right ventricular filling pressure

  e.g in heart failure fluid overload.

- Obstruction of blood flow from the right atrium to the right ventricle

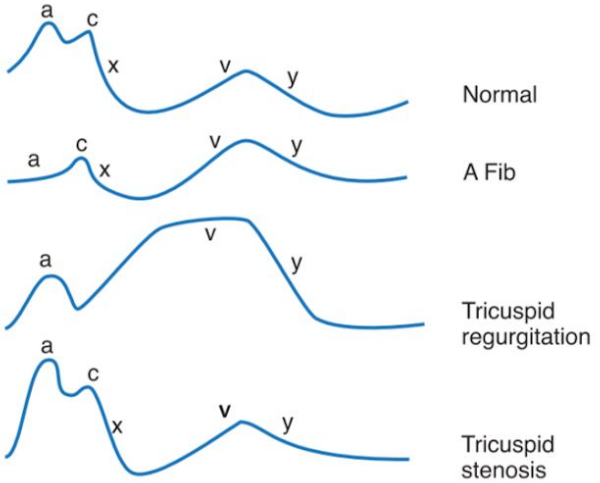
  e.g tricuspid stenosis.

- Superior vena caval obstruction

  e.g retrosternal thyroid goiter.

- Positive intrathoracic pressure

  e.g pleural effusion, pneumothorax.



The JVP usually drops on inspiration along with intrathoracic pressure

Heart Failure

Definition

A condition that exist when the heart is **unable to pump sufficient blood** to meet the metabolic needs of the body.
It is not a heart attack and not a death sentence.

Forms of Heart Failure:

- Systolic/Diastolic dysfunction.
- Left/right heart failure.
- High/low output failure.
- Acute/chronic failure.

Acute heart failure	Chronic heart failure
• Occurs within hours or days • Sudden serious abnormalities are presented. **E.g**: arrhythmia, infarction, valve rupture. • Can be lethal because the heart does not have time to activate its compensatory mechanisms. • If left alone, cardiogenic shock develops	• Occurs within months or years • Caused by the heart going through its compensatory responses. **E.g:** - Hypertrophy - Dilation

Causes of Right vs Left- Sided HF

- Occurs when either side of the heart can't keep up flow of blood.
- Can involve left or right side of the heart or both.
- Usually, the left side is affected first.

Right sided HF

Most common cause is left sided failure.

Other causes included:

- Pulmonary embolism.
- Pulmonary hypertension.
- RV infarction.
- Mitral + Pulmonary stenosis.
- COPD
- Interstitial lung disease
- Adult respiratory distress
- Chronic infections

Left sided (LV) HF

Volume/Pressure overload:

- Regurgitate valve (volume overload)
- High output status (volume overload)
- Systemic HTN (pressure overload)
- Outflow obstruction (pressure overload)

Restricted filling :
Pericardial diseases, restrictive cardiomyopathy, tachy arrhythmia

Loss of muscles :
Post MI, chronic ischemia, connective tissue diseases, infection, poisons.

Presents with pulmonary congestion.

Left and Right sided Heart failure

Clinical Picture	Left sided HF	Right sided HF
Pitting edema (legs)	Mild to moderate	Moderate to severe
Fluid retention	Pulmonary edema	Abdomen
Organ enlargement	Heart	Liver
Neck vein (JVP)	Mild to moderate	Severe
Breath shortness	Prominent dyspnea	Present but not prominent
GIT	Present but not prominent	Loss of appetite, constipation, etc...

In **left** sided HF, blood gets backed up into the pulmonary veins, causing pulmonary edema. In some cases, it can back up into the pulmonary artery, going to the right atrium and causing right sided HF and secondary pulmonary HT (Congestive HF).
In **right** sided HF, blood gets backed up into the systemic circulation, causing systemic edema

Clinical signs of Heart Failure

Left Heart Failure:

(Mostly respiratory)

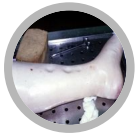
- Restlessness
- Confusion
- Tachycardia
- Exertional dyspnea
- Fatigue
- Cyanosis
- Cough
- Crackles
- Blood-tinged sputum
- Paroxysmal nocturnal dyspnea
- Orthopnea

Heart failure affects the heart in 2 ways:

1. not enough blood circulates, this leads to **fatigue**
2. fluid congests behind the heart because it's not being pumped, squeezing the lungs and the rest of the body. This causes **swelling and dyspnea**

Right Heart Failure:

- Fatigue
- Swelling of hands
- Hepato/splenomegaly
- GI distress
- **Ascites**
- Increased peripheral venous pressure
- **Pitting edema**



- Anorexia
- **Distended jugular veins**

- Secondary to chronic pulmonary issues

Think **FACES...**
Fatigue
Activities limited
Chest congestion
Edema or ankle swelling
Shortness of breath

Systolic dysfunction

Impairment of left ventricular contraction as a result from loss of the intrinsic inotropy (contractility.)

Causes:

- Myocardial infarction
- Transient myocardial ischemia
- Dilated cardiomyopathy
- Chronic volume overload

Leads to:

- Impaired LV contraction
- Decrease in ejection fraction(<40%)
- Dilated LV
- Reduction in Stroke volume (SV) for any given end-diastolic volume (EDV)

Diastolic dysfunction

Impaired ventricular diastolic relaxation as the ventricle becomes less compliant (stiffer.)

Causes:

- LV hypertrophy
- Transient myocardial ischemia
- Hypertrophic cardiomyopathy
- Obstruction of LV filling
- Restrictive cardiomyopathy

Leads to:

- Stiffer LV
- Normal ejection fraction
- Contracted LV
- Reduction in Stroke volume (SV) for any given end-diastolic volume (EDV)

Diastolic Dysfunction

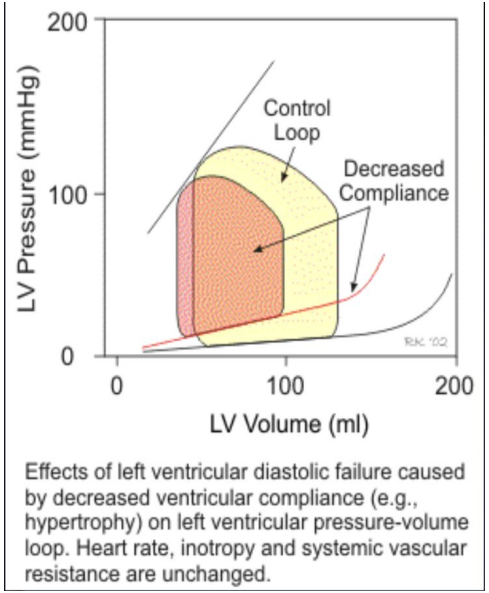
↓ EDV (end diastolic volume)

↓SV (stroke volume)

↓or = EF (ejection fraction)

↓Work

↓EDP (end diastolic pressure)



Effects of left ventricular diastolic failure caused by decreased ventricular compliance (e.g., hypertrophy) on left ventricular pressure-volume loop. Heart rate, inotropy and systemic vascular resistance are unchanged.

Systolic Dysfunction

↑ EDV

↓SV

↓EF

↓Work

↓EDP

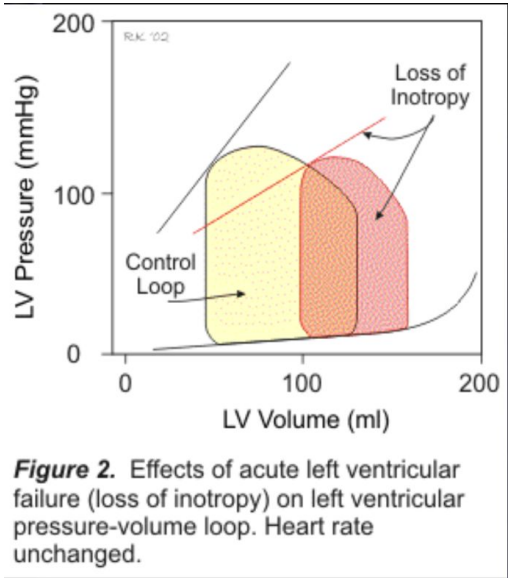


Figure 2. Effects of acute left ventricular failure (loss of inotropy) on left ventricular pressure-volume loop. Heart rate unchanged.

Etiology of Heart Failure

Female slides

Congestive Heart Failure (Chronic)

Is a clinical syndrome which is developed as a result of accumulation of the blood before the left or right parts of the heart.

Myocardial Failure (Chronic-Acute)

Myocardial abnormalities that can lead to its inability to fulfill its function.

Circulatory Failure

Abnormality in circulation that can be responsible for the inadequacy in tissue perfusion.

e.g. decrease in blood volume, changes in vascular tone(eg.hypersensitivity), disorder in heart function. (sudden changes)

Inappropriate work-load	Restricted filling	Impaired myocardial function/ Damage/ Loss
• Hypertension / Severe hypotension. • Pulmonary embolism (Cor-pulmonale). • Pregnancy, anemia, thyrotoxicosis, A-V fistula. • Valvular heart disease.	• Pericarditis. • Myocarditis. • Cardiomyopathy.	• Myocardial ischemia (Coronary artery disease). • Myocardial infarction. • Myocardial death.

Risk Factors of HF

Female slides

- Diabetes Mellitus
- High cholesterol
- Obesity
- Severe anemia
- Abnormal heart rhythm (Arrhythmias.)
- Congenital heart defects.
- Conditions associated with metabolic demand: (e.g. thyrotoxicosis, pregnancy, excessive physical activity)
- Old age
- Alcohol
- Severe lung disease
- Medication (noncompliance)
- Inter-current illness (e.g. infection)
- Dietary: excess sodium, excess fluid intake.
- Administration of drug(s) with negative inotropic properties or fluid retaining properties (e.g. NSAIDs, corticosteroids)

Pathophysiology of Heart Failure

Female slides

Hemodynamic changes

- Heart failure can be secondary to systolic or diastolic dysfunction.
- This will result in a decrease in cardiac output (CO), as a result of a decline in stroke volume (SV).
- Reduced “Ejection Fraction”:
  - Healthy heart ≥ 60%
  - Heart failure ≤ 40%
- Remaining healthy parts of the heart tries to remodel & compensate for the loss in the pumping function.

Neurohormonal changes

Cellular changes

- Change in Ca handling
- Change in contractile proteins
- Apoptosis
- More fibrous tissue
- Changes in adrenergic receptors:
  - ↑ a1 receptors
  - b1 receptors:

  desensitization → down regulation

Pathophysiology of Heart Failure

(Neurohormonal changes)

Female slides

N/H changes	Favorable effect	Unfavorable effect
↑Sympathetic adrenergic activity	↑HR,↑contractility, vasoconstriction>↑VR and filling	Vasoconstriction> ↑After load>↑workload>↑ O2 Consumption.
↑Circulating catecholamines	↑HR,↑contractility, vasoconstriction>↑VR & filling	Vasoconstriction> ↑After load>↑workload>↑ O2 Consumption.
↑Renin angiotensin aldosterone system activation	Salt & water retention>↑VR & vasoconstriction	Vasoconstriction> ↑After load>↑BP
↑Vasopressin (ADH)	water retention>↑VR & vasoconstriction	Vasoconstriction> ↑After load>↑BP
↑Interleukins & TNF alpha	May have roles in myocyte hypertrophy.	Apoptosis (programmed cell death)
↑Endothelin	Vasoconstriction>↑VR	Vasoconstriction> ↑After load>↑BP

Female slides

Neurohormonal Compensatory Changes in Heart Failure:

- Increase in sympathetic nervous activity (tone).
- Increase in circulating catecholamines.
- Increase in Renin-angiotensin-aldosterone.
- Increase in vasopressin.
- Increase in atrial natriuretic factor.

Main Compensatory Changes for Decreased Cardiac Output in Heart Failure:

- Increased sympathetic nervous system activity (tone) and catecholamines:
  - ↑ HR,
  - ↑ Contractility,
  - vasoconstriction to ↑ BP.
  - ↑ Venous return will ↑ SV.
- Activation of Renin-Angiotensin aldosterone System (RAAS) to ↑ BP.

Myocardial Remodeling as Compensatory Changes for HF

Female slides

01

Concentric Hypertrophy

(Lumen Changes)

- Myocardial thickening without dilation of ventricular lumen.
- Increase ratio of wall thickness to cavity radius.
- Thickening of myocytes by parallel apposition of sarcomeres.
- tension with an unchanged extent of shortening.
- Increased LVEDP.
- Increased incidence of backward failure.
- Decreased wall stress at expense of increased oxygen demand & increased LVEDP.

02

Eccentric Hypertrophy

(lumen doesn't change)

- Cavity dilation & hypertrophy.
- Prolongation of myocytes.
- Less internal work expended than in pressure overload.
- Increased stroke volume at the expense of increased wall stress, oxygen

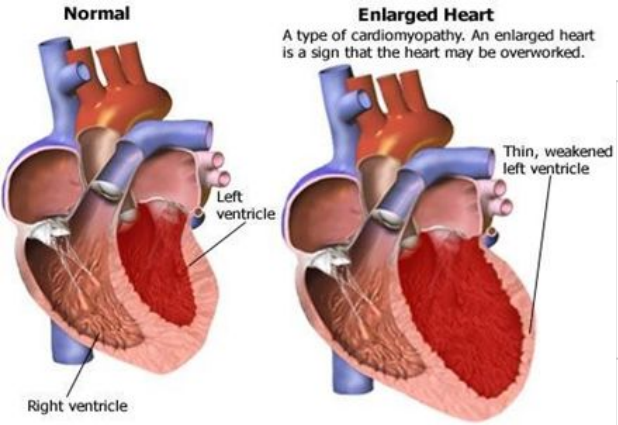
03

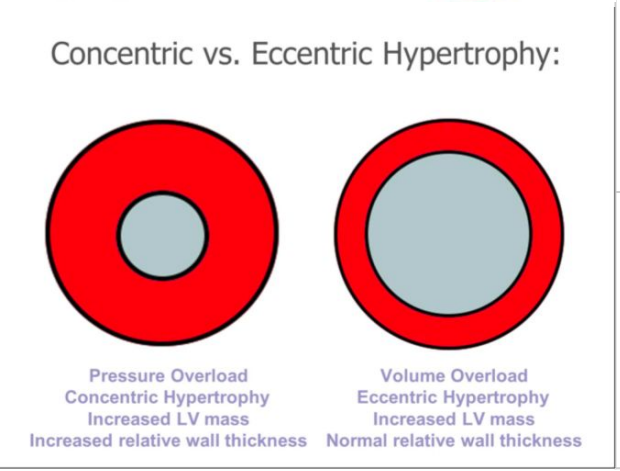
Ventricular dilation

More & more stretched out & weaker No change in the wall (thin)

Female slides

Ventricular Remodeling

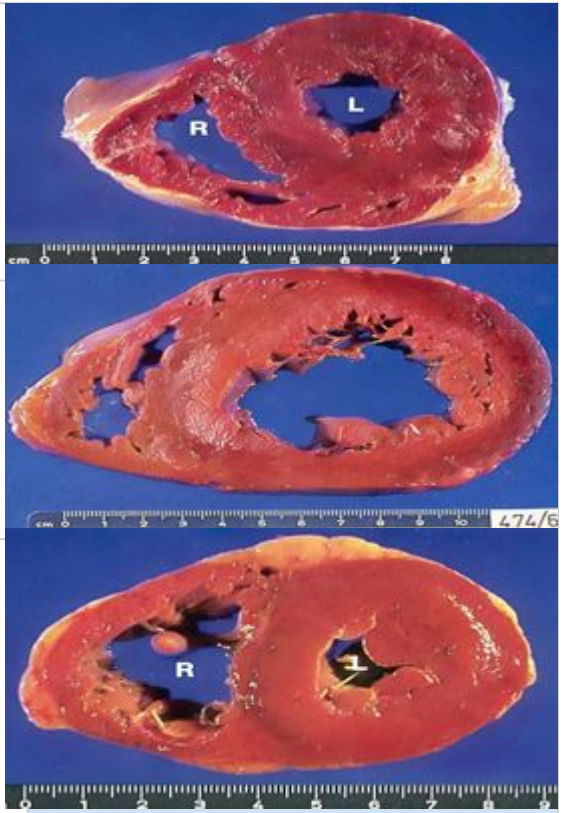




Normal heart
(cross section)

Eccentric hypertrophy (hypertrophy & dilatation) of left ventricle:
Seen in volume overload states, after acute MI (postinfarction remodeling), Seen in HTN heart disease

Concentric hypertrophy of the left ventricle:
Seen in pressure overload states as in HTN & aortic stenosis



New York Heart Association (NYHA) Functional Classification of Heart Failure

Class	% of patients	Symptoms
I	35%	Patient with cardiac disease but with no limitations in ordinary physical activity (no symptoms)
II	35%	Mild symptoms and slight limitation during ordinary activity
III	25%	Marked limitation in activity even during minimal activity. Comfortable only at rest
IV	5%	Severe limitation. Experiences symptoms even at rest

AHA/ACC Stages of Heart Failure

I - Asymptomatic with no heart damage.
- High risk for development of heart failure.

II - Structural damage or heart disease.
- No symptoms of heart failure.

III - Symptomatic heart failure.

IV - End-stage heart failure.

Diagnosis of heart failure:

Heart failure can be diagnosed by the following:

- Ejection fraction
- Blood tests
- Physical exam
- **Other tests:** Female slides

Echocardiogram

Look for:

- ventricular dysfunction.
- wall motion abnormality that may signify coronary artery disease (CAD).
- any valvular abnormality.
- any intra-cardiac shunts.

Electrocardiogram (ECG)

Look for:

- arrhythmia
- Old or recent MI.
- Left bundle branch block.
- Cardiomyopathy.

Chest X-ray

Look for:

- size & shape of the heart.
- evidence of pulmonary venous congestion.
- pleural effusion.

Cardiac Catheterization

Performed:

- when coronary artery disease (CAD) is suspected.
- if heart transplant is indicated.

Female slides

Treatment Options of heart failure:

The more common forms of heart failure cannot be cured, but can be treated:

Lifestyle Changes

- low-sodium +low fat diet.

-Fluid restriction.

-Stop smoking.

-Lose weight + exercise

-Avoid alcohol.

-Reduce stress.

-Keep track of symptoms, weight & report any changes or concern to the doctor.

Medications Used to Treat Heart Failure:

Male slides

-**Diuretics** (to reduce swelling)

-**Digoxin** (Increase contractility)

-**ACE Inhibitors** (vasodilation,↓ BP, block RAAS)

-**β Blockers** (↓ HR, ↓BP.)

•Combination of medications has been proven to save lives & keep people out of hospital.

Surgery & Other Medical Procedures:

•Not often used unless there is a correctable problem.

-Coronary artery bypass.

-Angioplasty.

-Valve replacement.

-Defibrillator implantation.

-Left ventricular assist device (LVAD.)

-Heart transplantation.

Physiological adaptations (compensatory mechanisms)

1- Decreased firing of carotid sinus baroreceptor

➡ ↑sympathetic stimulation

- Vasoconstriction of veins and arteries (increased preload and afterload, **respectively**)
- Increased HR and Contractility.
- Increased CO and BP.

2- Decreased renal perfusion:

activation of renin-angiotensin system (RAAS) which has many effects including raising BP

3- Decreased effective circulating blood volume:

posterior pituitary releases ADH which increases H2O reabsorption. (Increases blood volume)

Basically, this all leads to:

- ↑ SV + EDV
- ↑ Blood volume
- ↑ Contractility
- ↑ HR
- ↑ H2O retention
- ↑ Sympathetic firing

Complications of prolonged compensatory measures

1. **Prolonged sympathetic activation:** leads to down regulation of adrenergic receptors
2. **Vasoconstriction of arterioles:** resistance to blood flow is increased (increased afterload)
3. **Hypertrophy:** there's a difference between O2 supply and need -> land without O2 there is decreased ability to generate force
4. **Excessive salt and H2O retention:** as a result of RAAS
5. **Over-distended**(swollen) **ventricles:** has to use more energy and more wall tension to reach ejection pressure.

Quiz

1. Which of the following is a Characteristic of JVP?

A. Increased in right sided heart failure
B. Decreased in right sided heart failure
C. Increased during diastole
D. Decreased during systole

2. In right-sided heart failure, the organ that gets enlarged is:

A. Heart
B. Kidney
C. Liver
D. Lungs

3. Which of the following is a feature of eccentric hypertrophy?

A. Decreased wall stress
B. Dilated cavity only.
C. Thickening of ventricle with dilated cavity.
D. Hercules the great roman hero

5. Which of the following is a clinical sign of left sided heart failure?

A. Anorexia
B. orthopnea
C. Ascites
D. Edema

5. Bulging of the tricuspid valve causes which of the following waves?

A. C- wave
B. A- wave
C. V- wave
D. X- wave

SAQ:

1- How can mitral stenosis cause right-sided heart failure?

Blood builds up in left atrium, then goes into pulmonary vessels until it reaches the right side.

2- Why does the liver get enlarged in RHF

In RHF, blood will congest in the systemic circulation. Since the liver is a huge blood reservoir, it will most definitely get enlarged

Answers: A - C - C - B - A

Leaders

Sedra Elsirawani Abdulrahman Alhawas

Members

- Lama AlZamil
- Arwa AlEmam
- **Noura AlTurki**
- Ghada AlSadhan
- Nouf AlShammari
- Nouf AlHumaidhi
- Taibah AlZaid
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