Cardiovascular Physiology

Jugular Venous Pressure & Heart Failure

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Lecture Outcomes

- To define Jugular Venous Pressure.
- To identify different transmitted waves in the JVP record.
- To identify what is meant Heart Failure.
- To recognize causes of Heart Failure.
- To understand the pathophysiology & compensatory mechanisms of Heart Failure.

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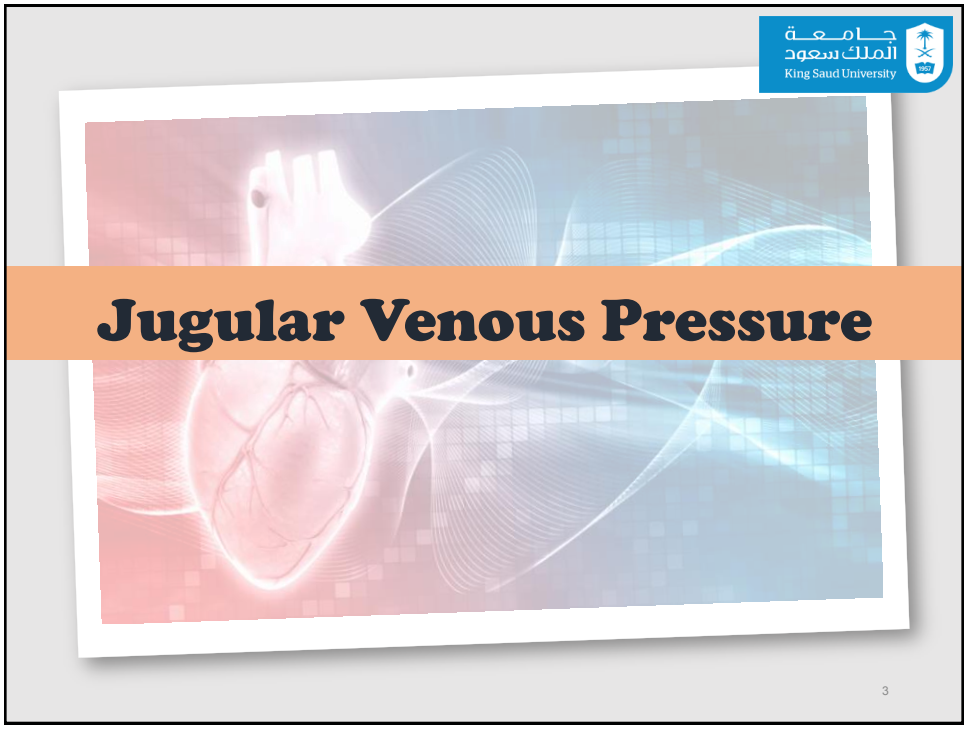
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Jugular Venous Pressure
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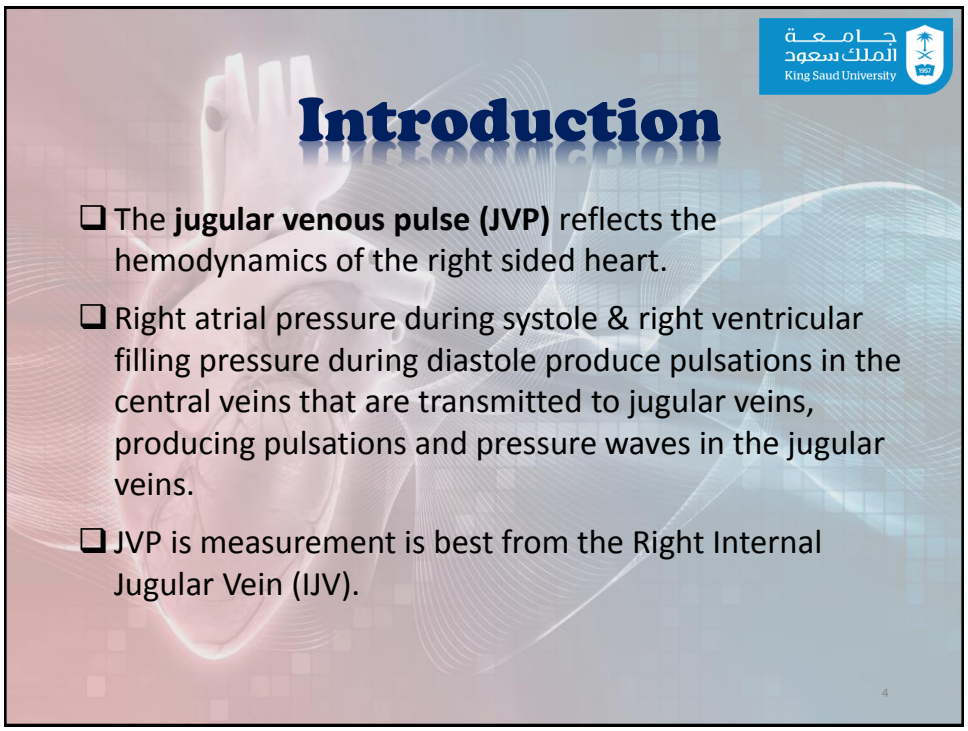
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Introduction
The jugular venous pulse (JVP) reflects the hemodynamics of the right sided heart.
Right atrial pressure during systole & right ventricular filling pressure during diastole produce pulsations in the central veins that are transmitted to jugular veins, producing pulsations and pressure waves in the jugular veins.
JVP is measurement is best from the Right Internal Jugular Vein (IJV).
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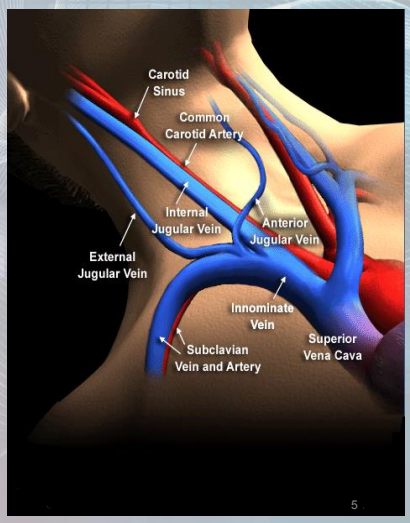
Why Right Internal Jugular Vein ?

- It is in direct continuation to the right atrium (RA).
- Is 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.



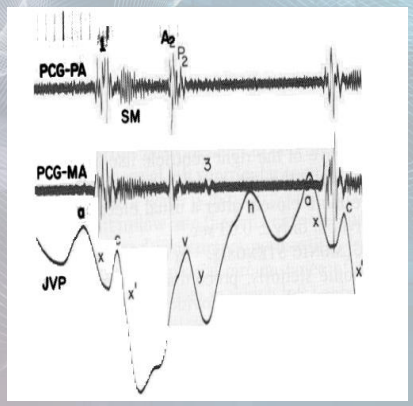
Difference Between IJV and Carotid Pulses

- 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
- Jugular venous pressure falls during inspiration
- Abdominal compression elevates jugular pressure
- Mean jugular venous pressure falls during standing

- 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
- Abdominal compression has no effect on carotid pulse
- Carotid pulse do not change when standing

Normal Jugular Venous Pulse

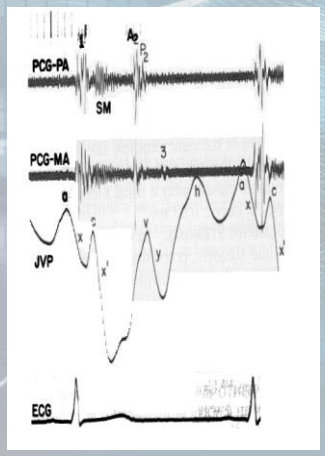
- Normal JVP reflects phasic pressure changes in the RA during systole & the RV during diastole.
- Consists of:
  - **3** positive waves: **a, c, & v**
  - **3** descents (downward deflections): **x, x' (x prime) & y**
- The 3 wave (a, c, & v) are equal to ONE cardiac cycle = 0.8 sec.



Causes of JVP waves:

a- Wave

- First positive presystolic a- wave is due to increase in atrial pressure during right atrial contraction (**atrial systole**).
- Results in retrograde blood flow in to the Superior vena cava (SVC) & jugulars.
- Effective RA contraction is needed for visible a- wave
- Dominant wave in JVP & is larger than v-wave.
- It precedes:
  - upstroke of the carotid pulse.
  - The first heart sound (S1).
- It follows the P- wave in ECG.



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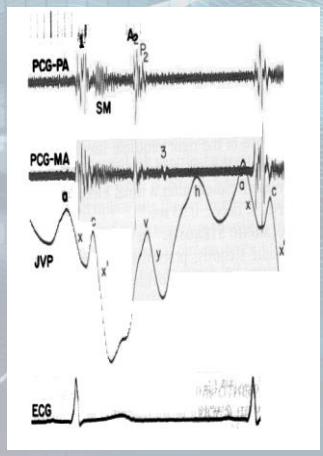
Causes of JVP waves: x- Descent

- Systolic x- descent (systolic collapse) is due to **atrial relaxation** during atrial diastole
- X- descent is most prominent motion of normal JVP which begins during systole & ends just before the second heart sound (S2).
- It is larger than y- descent.
- X- descent is more prominent during inspiration.



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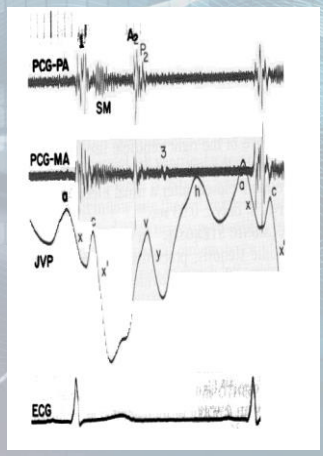
Causes of JVP waves: c- Wave

- Second positive wave recorded in JVP which interrupts the x-descent.
- Produced by:
  - **upward bulging** of closed tricuspid valve (TV) into the RA during **isovolumic contraction phase** of the ventricle.
  - carotid artery impact on JVP?



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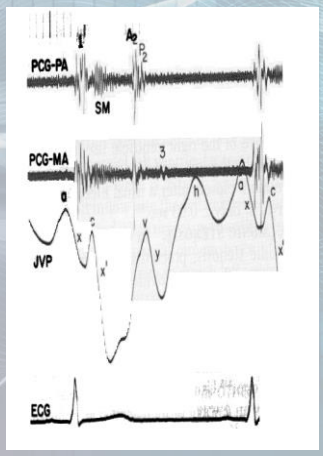
Causes of JVP waves: X'- Descent

- x`descent is systolic trough after c-wave
- Due to:
  - **downward pulling of the TV** by contracting right ventricle.
  - descent of RA floor by contracting RV.
  - fall of right atrial pressure during early RV systole.



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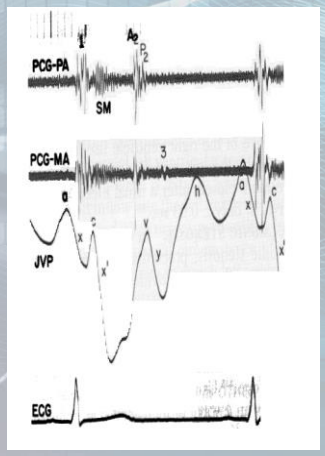
Causes of JVP waves: v- Wave

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



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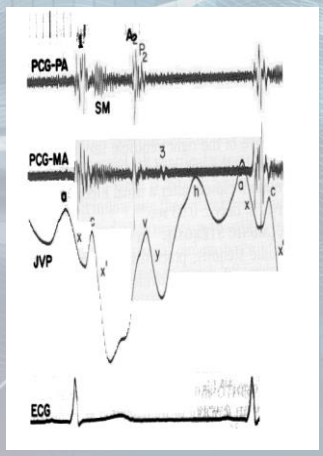
Causes of JVP waves: y- Descent

- Diastolic collapse wave (down slope v- wave).
- 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 of the y- wave is produced by continued diastolic inflow in to RV.



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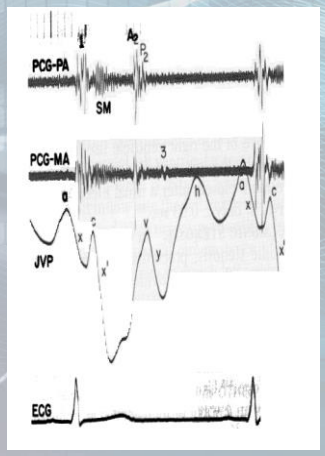
Causes of JVP waves: h- Wave

- Small brief positive wave following y- descent just prior to a- wave **during period of diastasis**.
- Described by Hieschfelder in 1907.
- It **usually seen when diastole is long** (as in slow heart rates).
- With increasing heart rate, y- descent immediately followed by next a- wave .



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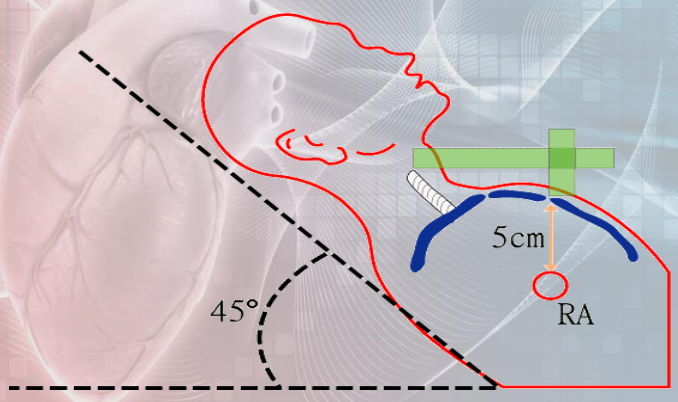
JVP Measurement



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JVP Measurement

- Two scale method is commonly used
- A horizontal scale at the top of the oscillating venous column in IJV cuts the vertical scale at the sternal angle gives JV pressure in cm of water
- Normally JV pressure does not exceed 4 cm above the sternal angle
- Since RA is approximately 5 cm below the sternal angle , the jugular venous pressure (RA mean pressure) is corresponds to 9 cm
- By way of conversion , normal mean JV pressure does not exceed 7 mm Hg (9 cm column of water / 1.3 =6.9)
- Elevated JVP : JVP of >4 cm above sternal angle .

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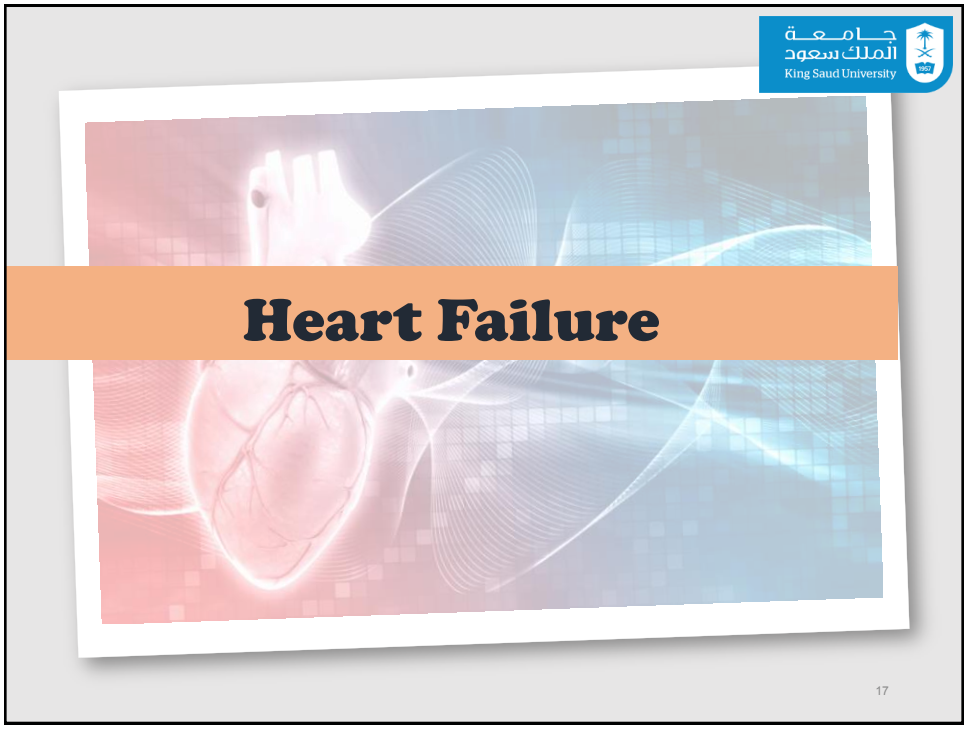

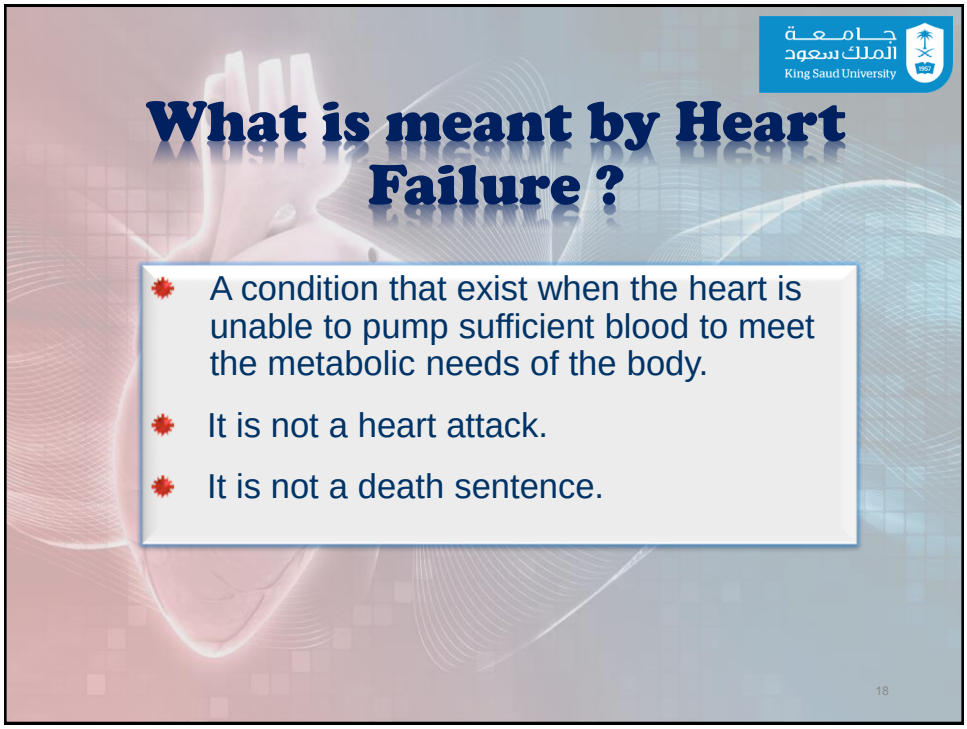

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Etiology of Heart Failure (HF)

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

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Heart Failure Terminologies

- **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, disorder in heart function.
- **Myocardial Failure**
  - Myocardial abnormalities that can lead to its inability to fulfill its function.
- **Congestive Heart Failure**
  - Is a clinical syndrome which is developed as a result of accumulation of the blood before the left or right parts of the heart.

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Types/Forms of Heart Failure

- Systolic HF, or systolic dysfunction
- Diastolic HF, or diastolic dysfunction

- Right- sided HF
- Left- sided HF

- Acute HF: (e.g. large MI, aortic valve dysfunction)
- Chronic HF

- High output failure:
  (e.g. pregnancy, anemia, thyrotoxisis, A/V fistula)
- Low output failure

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Causes of Right vs Left- Sided HF

- Occurs when either side of the heart can't keep up w 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 (HTN).
  - RV infarction.
  - Mitral stenosis (MS).

✓ **Usually presents with:**

Lower limb edema, sacral edema, ascites, pleural effusion, hepatic congestion, hepato-splenomegally, elevated JVP, cardiac cirrhosis.

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

✓ **Presents with pulmonary congestion.**

Systolic Dysfunction

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

Causes:

Myocardial infarction, transient myocardial ischemia, chronic volume overload, dilated cardiomyopathy, pressure overload (aortic stenosis, uncontrolled hypertension.)

It will lead to:

- Impaired left ventricular contraction.
- Reduction in Stroke volume (SV) for any given end-diastolic volume (EDV).
- Decrease in Ejection Fraction (EF):
  - When EF reduce to ≤ 40%, the heart will have systolic failure.
  - EF = SV/EDV
- ? Dilated left ventricle.

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Diastolic Dysfunction

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

Causes:

Left ventricular hypertrophy, hypertrophic cardiomyopathy, restrictive cardiomyopathy, transient myocardial ischemia, obstruction of left ventricular filling (mitral stenosis, pericardial constriction or tamponade.)

What will happen?

- A reduction of ventricular filling rate & extent of filling / or a normal extent of filling in associated with an inappropriate rise in ventricular diastolic preassure.
- Ejection Fration (EF) is maintained.
- Increased stiffness of the left ventricle.
  - Left ventricular hypertrophy (LVH).
  - Left ventricular (LV) fibrosis.
  - Restrictive or infiltrative cardiomyopathy.
- ? Small left ventriclular diameter.

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Factors Aggravating HF (Risk Factors)

- Diabetes Mellitus (DM).
- High cholesterol.
- Obesity.
- Severe anemia.
- Conditions associated with ↑ metabolic demand: (e.g. thyrotoxicosis, pregnancy, excessive physical activity.)
- Abnormal heart rhythm (Arrhythmias.)
- Congenital heart defects.

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Factors Aggravating HF (Risk Factors) … (Cont.)

- Inter-current illness (e.g. infection.)
- Old age.
- Dietary: excess sodium, excess fluid intake.
- Severe lung disease.
- Medication (noncompliance.)
- Administration of drug(s) with negative inotropic properties or fluid retaining properties (e.g. NSAIDs, corticosteroids.)
- Alcohol.

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Pathophysiology of Heart Failure

- **Hemodynamic changes**
- **Neuro-hormonal changes**
- **Cellular changes**

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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.

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Neurohormonal Changes

N/H changes	Favorable effect	Unfavorable effect
↑ Sympathetic adrenergic activity	↑ HR ,↑ contractility, vasoconstriction → ↑ Venous return (VR), ↑ filling.	Vasoconstriction constriction → ↑ After load →↑ workload →↑ O_2 Consumption.
↑ Circulating catecholamines	↑ HR ,↑ contractility, vasoconstriction → ↑ Venous return (VR), ↑ filling.	Vasoconstriction constriction → ↑ After load →↑ workload →↑ O_2 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 &TNFα	May have roles in myocyte hypertrophy.	Apoptosis (programmed cell death.)
↑Endothelin	Vasoconstriction→↑ VR.	↑ After load → ↑ BP

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Compensatory Mechanisms for HF

Remaining healthy parts of the heart tries to compensate for the loss in pumping function:

Neuro-hormonal compensation.

Remodeling.

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Neuro-hormonal Compensatory Changes in Hearf Failure

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

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Main Compensatory Changes for Decreased Cardiac Output in Hearf Failure

- Increased sympathetic nervous system activity (tone) and catecholamines:
  ↑ HR, ↑ contractility, & vasoconstriction to ↑ BP.
  - Frank-Starling mechanism:
    ↑ Venous return will ↑ LVEDP = ↑ SV.
- Activation of Renin-Angiotensinaldosterone System (RAAS) to ↑ BP.

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Myocardial Remodeling as Compensatory Changes for Heart Failure

- **Myocardial hypertrophy: (more muscle mass)**
  - **•Concentric hypertrophy:**

-Myocardial thickening without dilatation 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.

  - **•Eccentric hypertrophy:**

-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 demand & LVEDP.

- **Ventricular dilation:** More & more stretched out & weaker.

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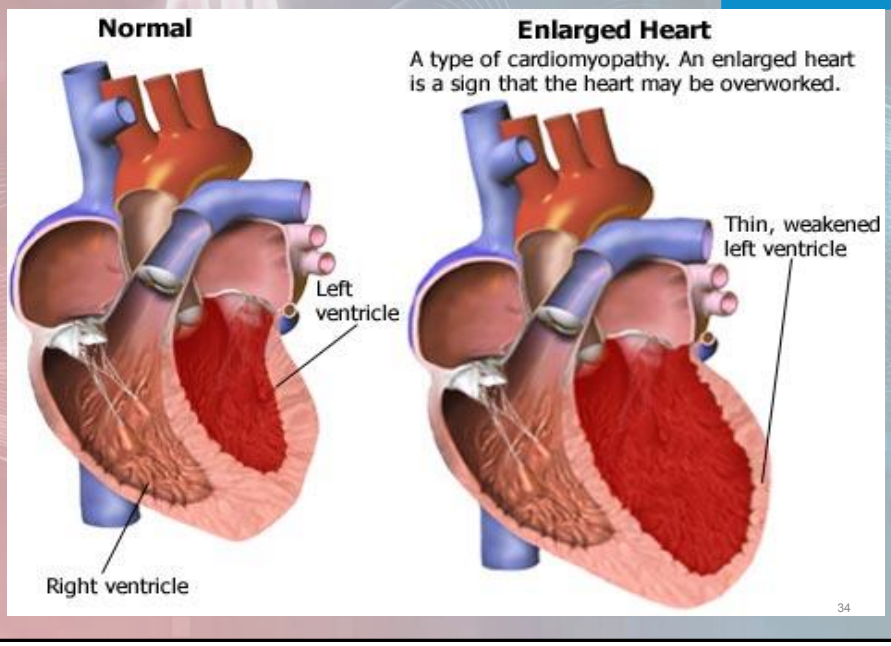

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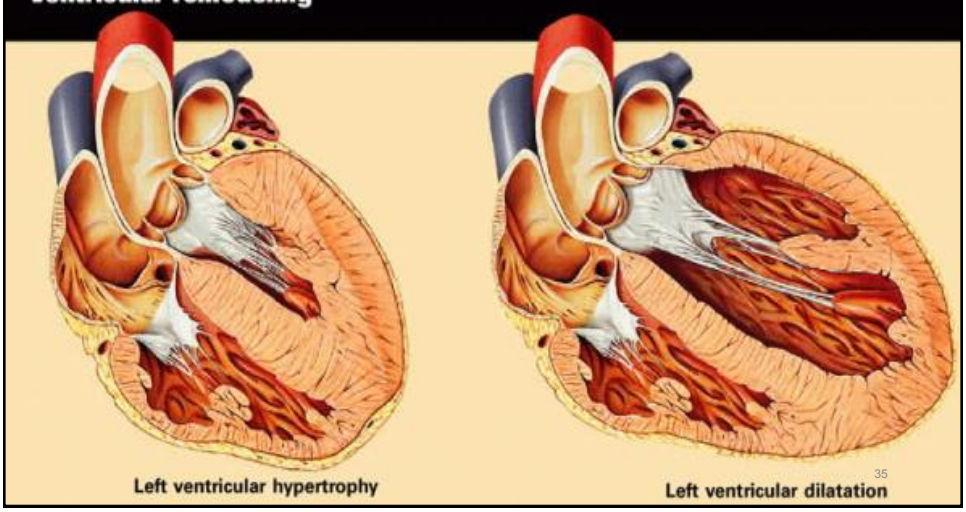
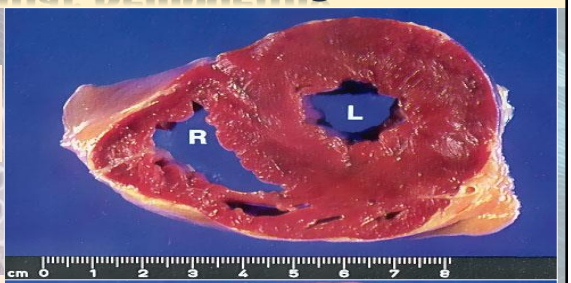
Ventricular Remodeling



Ventricular Remodeling

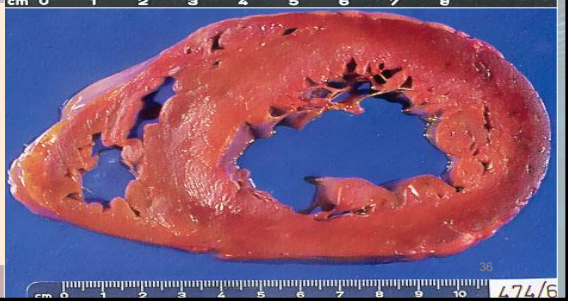
Normal heart

(cross section)



Eccentric hypertrophy (hypertrophy & dilatation) of left ventricle:

Seen in volume overload states, after acute MI (post-infarction remodeling), ? Seen in HTN heart disease.



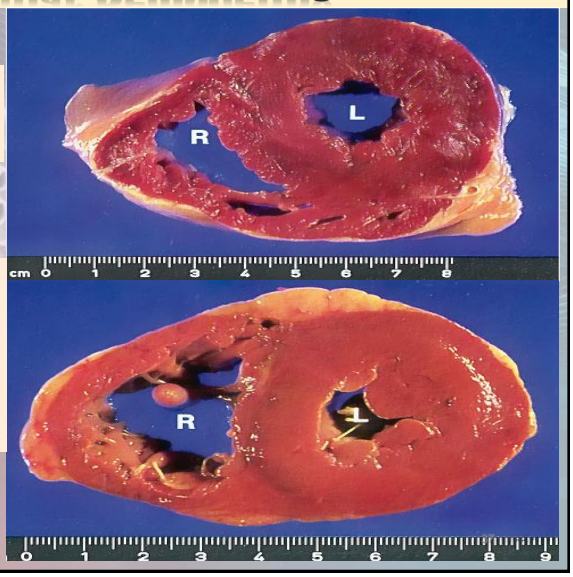
Ventricular Remodeling

Normal heart

(cross section)

Concentric hypertrophy of the left ventricle:

Seen in pressure overload states as in HTN & aortic stenosis.



Cellular Changes

- **Changes in Ca^{+2} handling**
- **Changes in adrenergic receptors:**
  - Slight ↑ in α_1 receptors.
  - β_1 receptors desensitization → followed by down regulation.
- **Changes in contractile proteins**
- **Program cell death (Apoptosis)**
- **Increase amount of fibrous tissue**

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
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

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AHA/ACC Stages of Heart Failure

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

Stage B
- Structural damage or heart disease.
- No symptoms of heart failure.

Stage C
- Symptomatic heart failure.

Stage D
- End-stage heart failure.

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Clinical Manifestations of Heart Failure

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How Does HF Affect the Body ?

- **Commonly termed congestive heart failure (CHF):**
  - Since symptoms of increase venous pressure are often prominent, as a result of two major problems:
    1) Not enough blood to circulate → fatigue.
    2) Fluid builds up behind the heart, since the blood is not squeezed well out of the heart, creating congestion into the lungs & the rest of the body → shortness of breath & swelling.

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Symptoms of HF

Think **FACES**...

Fatigue

Activities limited

Chest congestion

Edema or ankle swelling

Shortness of breath

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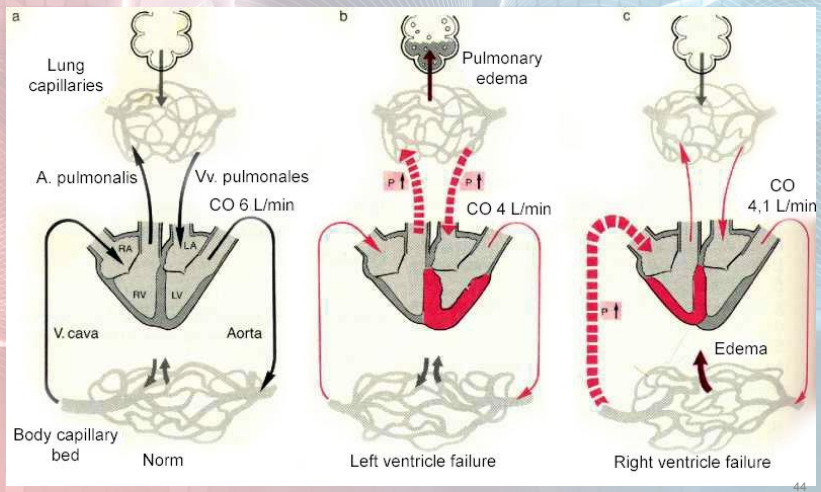
Organismic Consequences of HF

"Forward" & "backward" failure:



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Symptoms

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Physical Signs of Heart Failure

- Tachycardia, tachypnea.
- Jugular venous distension (JVD.)
- High diastolic BP & occasional decrease in systolic BP (decapitated BP.)
- Edema.
- Pulmonary rales (inspiratory.)
- Pleural effusion.
- Ascites.
- Hepato/splenomegaly.
- Displaced & sustained apical impulses (Cardiomegaly.)
- S3 gallop, ? S4.
- Pale, cold sweaty skin.

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Jugular venous distension (JVD)

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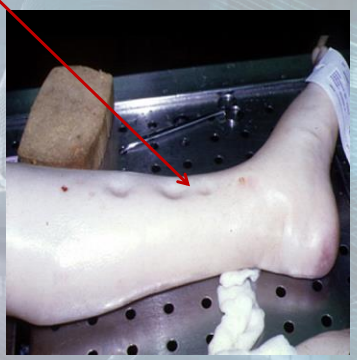
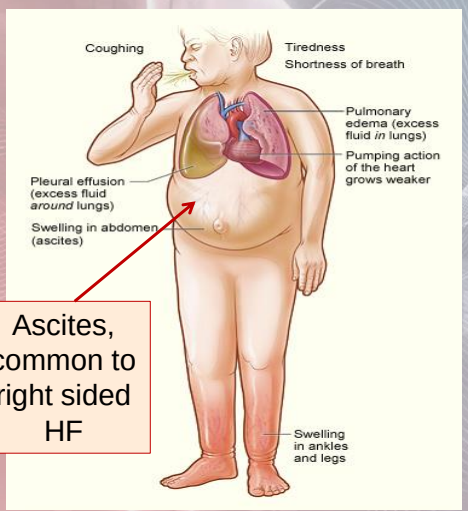
Pitting edema, common to right sided HF





Ascites, common to right sided HF

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Diagnostic Investigation

- **Echocardiogram**
- **Electrocardiogram (ECG)**
- **Chest X-ray**
- **? Cardiac Catheterization**

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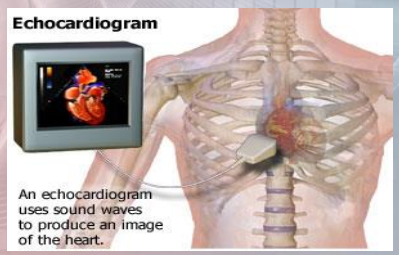
Echocardiogram

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



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Electrocardiogram (ECG)

- Look for arrhythmias:
  - Old or recent MI.
  - Left bundle branch block (LBBB).
- ? Some forms of Cardiomyopathy.

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Chest X- ray

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

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Cardiac Catheterization

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

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Is there a Cure for Heart Failure ?

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Currently, there is NO cure for heart failure

- **However, early diagnosis & proper treatment can:**
  - Slow the progression of disease.
  - Keep out of the hospital.
  - Save life!

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Goal of Treatment

- To improve symptoms & quality of life.
- To decrease likelihood of disease progression.
- To reduce the risk of death & need for hospitalization.

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Treatment Options of heart failure ?

- **The more common forms of heart failure cannot be cured, but can be treated:**
  - Lifestyle changes.
  - Medications.
  - Surgery.

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Lifestyle Changes .. (Diet & activities)

- Salt restriction … low-sodium diet.
- Fluid restriction.
- Stop smoking.
- Loose weight … daily weight (tailor therapy).
- Eat a low-fat.
- Exercise … gradual exertion program.
- Avoid alcohol.
- Reduce stress.
- Keep track of symptoms, weight & report any changes or concern to the doctor.
- See the doctor more frequently.

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Medications Used to Treat Heart Failure :

- **Experts recommend:**
  - **Diuretics** … (to reduce swelling)
  - **Digoxin** … (to increase heart contractility)
  - **ACE Inhibitors** … (in case of high blood pressure: to cause vasodilatation & ↓ blood pressure & block renin-angiotensin aldosterone system.)
  - **β Blockers** … (in case of high blood pressure: to ↓ heart rate & ↓ blood pressure.)
- Combination of medications has been proven to save lives & keep people out of hospital.

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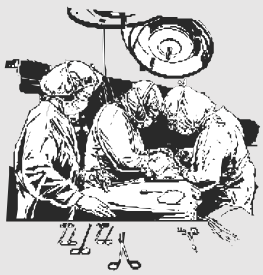
Surgery & Other Medical Procedures :

- **Not often used in heart failure unless there is a correctable problem.**
  - Coronary artery bypass.
  - Angioplasty.
  - Valve replacement.
  - Defibrillator implantation.
  - Left ventricular assist device (LVAD.)
  - Heart transplantation.



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Cardiac Transplant

- **Become more widely used since the advances in immunosuppressive treatment.**
- **Survival rate:**
  - 1 year 80% - 90%
  - 5 years 70%

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Prognosis

- **Annual mortality rate depends on patients symptoms & LV function.**
  - 5% … in patients w mild symptoms & mild ↓ in LV function.
  - 30% – 50% … in patient w advances LV dysfunction & severe symptoms.
  - 40% – 50% of death … due to sudden cardiac death (SCD.)

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Can a Person Live with Heart Failure?

YES!

- **See physician regularly.**
- **Limit salt & water intake.**
- **Weigh each day.**
  - Contact physician if his weight changes > 2-3 pounds in one day.
- **Take medications.**
- **Exercise at levels recommended by physician.**

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