Physiology Team 434

Cardiac Output | Stroke Volume | Venous Return | Their Regulation & Heart Failure

Color index

- Important
- Further Additions
- In boys' slides

Only in Boys' Slides

Only in Girls' Slides

Explained in:
-Guyton Ch.9, P.110 & Ch.20
-Linda's Ch.4, P.151

Physiology Team 434
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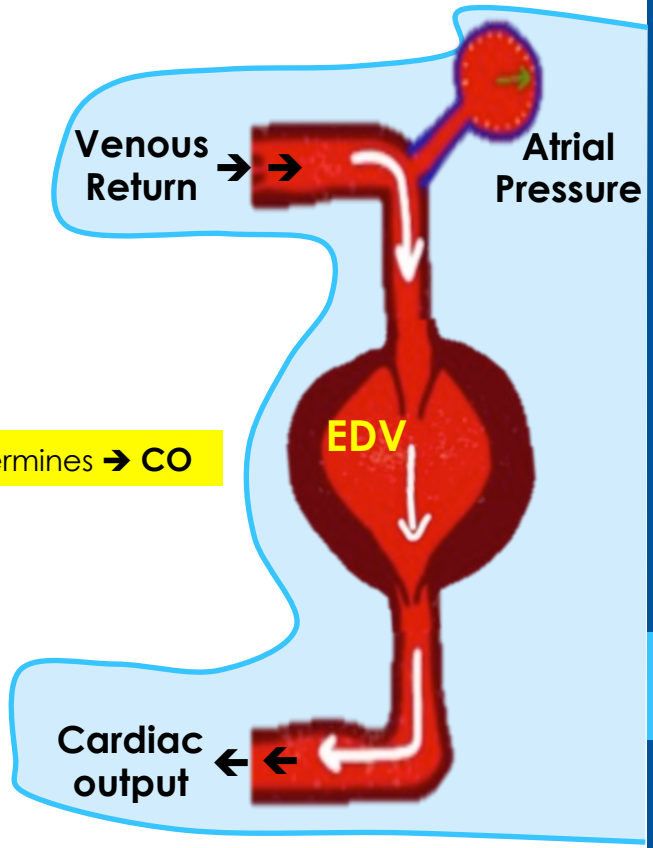
Story Time..

You may think the titles of these lectures are confusing!
Indeed they are a BIT confusing but when you understand the relation between those titles everything will make perfect sense!

Let me tell you a story:

Cardiac output (CO) is the volume of blood pumped by the heart per minute and one of the most important factors determining CO is left ventricular **end diastolic volume (EDV)** which depends on **venous return (VR)**, which also determines **right atrial pressure (AP)**. Thus, it follows that there is not only a relationship between CO and EDV but also a relationship between CO and AP. CO and VR each can be examined separately as a function of right AP.



Simply

VR depends mainly on ➔ right AP which will determines ➔ EDV which will determines ➔ **CO**

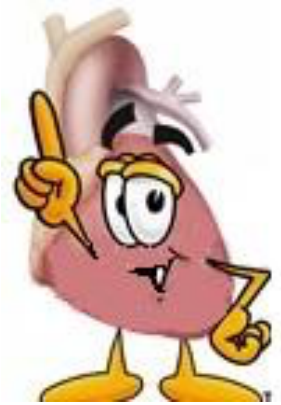
Where is the stroke volume in all this?
Stroke volume is the amount of blood pumped per beat!
While CO is amount of blood pumped per minute!

We will come to know the details of each one of them and the factors that regulates it..
Don't you worry, just set back read and enjoy the journey..☺

Additional Slide
But READ it for a better understanding...

Definitions:

- **Cardiac Output** (CO) [mL/min] :
  Amount of blood pumped by each ventricle per minute. = (5L/min)
- **Stroke Volume** (SV) [mL/beat]:
  Volume of blood is ejected by each ventricle per beat. = (70mL/beat)
- **End-diastolic volume** (EDV) [mL]:
  Amount of blood remaining in the heart by the end of diastole. =(140mL)
- **End-systolic volume** (ESV) [mL]:
  Amount of blood remaining in the heart by the end of systole. = (70mL)
- **Ejection fraction** (EJ) [%]:
  The percentage of blood that is pumped out of a filled ventricle as a result of a heartbeat.



- ✓ CO = SV **x** HR | UNIT : ml/~~beat~~ **x** ~~beat~~/min = **ml/min**
- ✓ SV = EDV **-** ESV
- ✓ EJ = SV / EDV
- ✓ Cardiac Index = CO / BSA[1] | UNIT is litres per minute per square metre **L/min/m²**

This allows direct comparison of the cardiac index of large and small patients

1: Body surface area

Cardiac Index

Is a haemodynamic parameter that relates the cardiac output(CO)from left ventricle in one minute to body surface area (BSA),thus relating heart performance to the size of the individual.

Cardiac Output

Cardiac output is the blood flow generated by each ventricle per minute (i.e., the blood pumped by each ventricle per minute).

- ✧ The cardiac output is equal; to the volume of blood pumped by one ventricle per beat (stroke volume) times the number of beats per minute (heart rate)

$$CO = SV \times HR$$

- ✓ CO is well regulated according to tissue metabolic demands.
- ✓ Accordingly, if the metabolic rate is increased, the CO and VR are increased to maintain optimal O2 supply to the active tissues.

- ✧ The cardiac output at rest is approximately 5 L/min.
- ✧ The body's blood volume averages 5 to 5.5 liters.
- ✧ Thus, each ventricle pumps the equivalent of the entire blood volume each minute.
- ✧ During exercise, the CO can increase to 20 to 25 liters/min and to as high as 40 liters/min in well trained athletes.
- ✧ The difference between the resting CO and the maximum volume of blood the heart is capable of pumping per minute is known as the cardiac reserve

CO = SV x HR

Stroke Volume = EDV - ESV

Recall! Stroke volume is Volume of blood is ejected by each ventricle per beat. (mL/beat)

Affected by changes in

- The heart's ability to contract.
- The force of contraction.
- The volume of blood available to be pumped.
- Preload (venous return).
- Afterload.

Other Variables

- Resistance within the circulatory system.
- Severe hemorrhage.
- Shock.
- Heart damage.
- Extreme infection.

Any factor effects the stroke volume will affect cardiac output as well.

Heart Rate (Beats/min)

↑ INCREASE	↓ DECREASE
Due to *Stimulation *Excitation by activity, drugs, medications...	Due to *Electrical abnormality *Some medications
Increase ↑CO	Decrease ↓CO
Excessively Fast!!↑↑	**Too slow!!↓↓**
Not have enough time to adequately fill with blood between beats.	↓CO decreased significantly.
Decrease ↓CO	Innervations required: Implanted pacemakers.

✧ **To increase the Cardiac output:**

✓ **Increase Stroke volume**

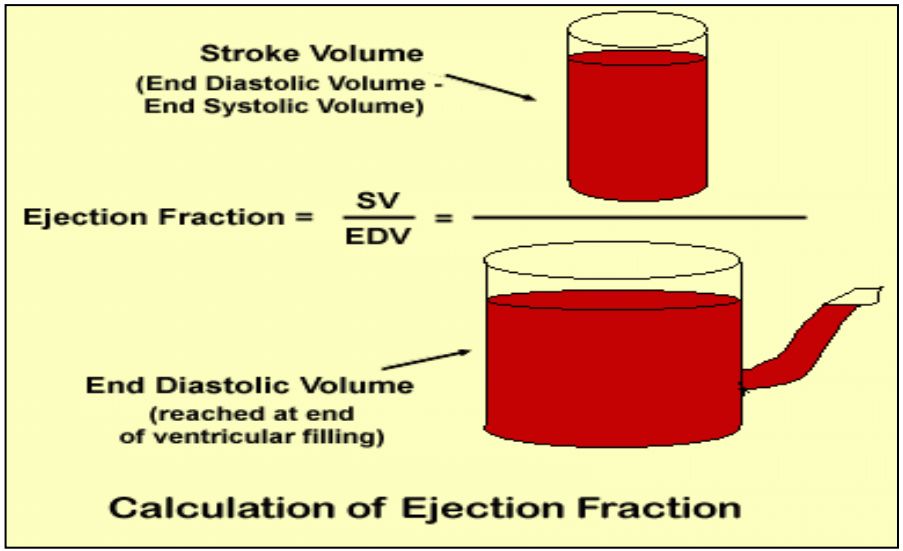
✓ **Increase Heart rate** up to a limit

✓ **Increase both** like in exercise

Ejection fraction: = SV / EDV

The percentage of blood that is pumped out of a filled ventricle as a result of a heartbeat.

- ✓ The heart **does not eject all the blood** in the ventricle.
- ✓ Only about ⅔ of the blood is normally pumped out with each beat.
- ✓ That fraction is referred to as the **Ejection Fraction**.
  it's decrease when the cardiac muscle becomes diseased...



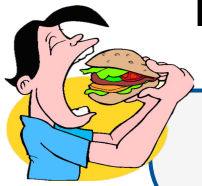
Variations of Cardiac output

Physiological Variations	Pathological Variations
Increases CO ↑	
Exercise (700%)	Fever
Eating (30%)	Hyperthyroidism
High Environmental Temperature	Anaemia
Pregnancy	**Decreases CO ↓**
Anxiety (50-100%)	Hypothermia
Sympathomimetics 'Epinephrine'	**Myocardial Diseases** eg. Infarction, failure..etc
Decreases CO ↓	Rapid Arrhythmias
Sitting Or Standing From Laying Position (20-30%)	Hypothyroidism

Arrow toward center = Decreases
Arrow away from center = Increases

Variations of Cardiac Output


Exercise


Eating

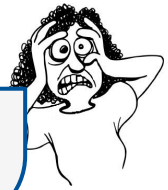

High Temperature

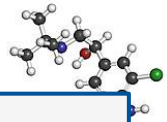

Hyperthyroidism
Hypothyroidism


Myocardial diseases


Rapid arrhythmias


Pregnancy


Anxiety


Sympathomimetics


Sitting or standing form laying position


Fever & Hypothermia


Anaemia

Factors Affecting Cardiac Output

CO = Stroke Volume **x** Heart Rate

↑ Preload → ↑ SV (↑ EDV)
↑ Afterload → ↓ SV (↑ ESV)
↑ Inotropy → ↑ SV (↓ ESV)

An increase or decrease in heart rate will affect the cardiac out put as said previously

Stroke volume with all the factors affecting it (mentioned previously) will affect CO But most important factors are:

Preload

=

Venous Return

Contractility

Afterload

=

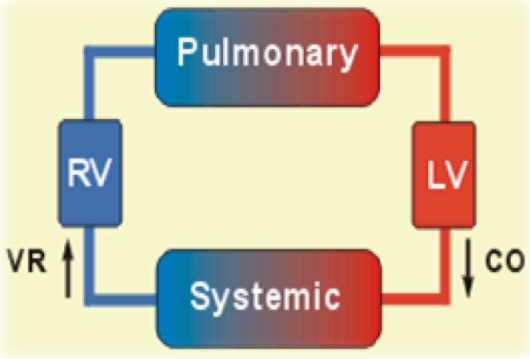
Arterial Blood Pressure

Venous Return (Preload)

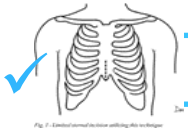
It represent the amount of blood returning to the heart per minute [Ventricular filling]

✧ **Relationship between Cardiac output & Venous Return**

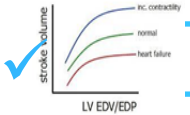
Because the cardiovascular system is a closed loop.
If they're not equal ➔ blood accumulate in either:
the systemic or pulmonary circulation.



✧ Its controlled by many factors:



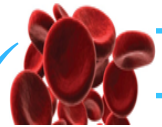
✓ Thoracic pump



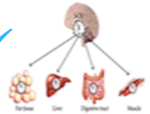
✓ Frank-starling's mechanism

✓ Muscle pump

✓ Gravity



✓ Blood volume



✓ Tissue metabolism

✓ Valsalva maneuver

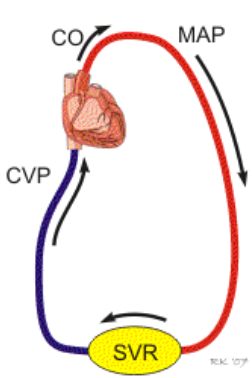
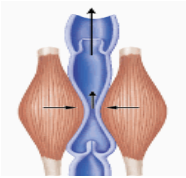
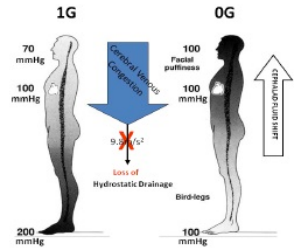
✓ Mean systemic pressure

✓ ↑ Atrial Contractility
✓ ↑ Aortic Pressure
✓ ↑ Ventricular Compliance
✓ ↑ Central Venous Pressure By:
- ↑ Blood Volume
- ↓ Venous Compliance

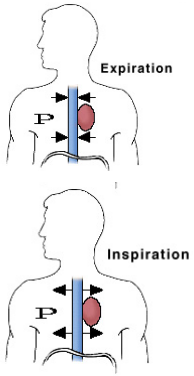
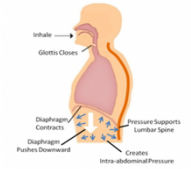
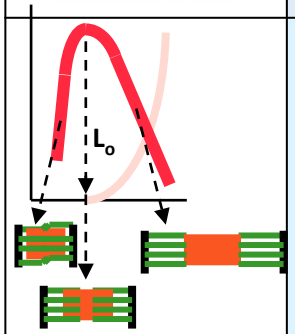
✓ ↓ Heart Rate

Will Increase the ↑ Preload

Factors Affecting Venous Return (Preload)

	**Mean Systemic Filling Pressure**	-Sympathetic fibers supply veins (sympathetic vasoconstrictor tone) -This is important to maintain venous pressure and hence creates pressure gradient (from high to low)to push blood to the right atrium. - Acute venous dilatation can lead to significant drop in VR and so cardiac output.
	**Skeletal Muscle Pump**	-When the skeletal muscle compress the veins, they force the blood to the heart. -Valves in the veins prevent backflow of blood .
	**Gravity**	-The amount of blood push upward decreased and VR decreased. -CO decreased and that decreased tissue perfusion in brain then that will cause unconsciousness.

Factors Affecting Venous Return (Preload)

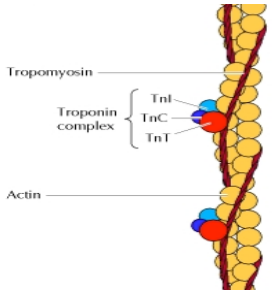
	**Thoracic Pump**	-Affect blood flow to the heart. **During Inspiration:** Thoracic pressure become more(-) Pressure inside the abdomen is (+) that push blood through the vessels from abdomen to the thoraxes . **During Expiration :** The thoracic pressure here is (+) Pressure inside the abdomen is(-)
	**Valsalva Maneuvear**	-It is a forced expiration against closed glottis. (Straining) -Increase intrathoracic positivity. -Significant compression of the veins.
	**Frank-starling's Law of the heart**	-Ability of the heart to change it force of contraction and therefore stroke volume in response to changes in venous return. -Ability of the heart to pump all blood coming to it, without allowing systemic stasis within limits. -Good filling of the Heart with blood (VR) ➔ Cardiac stretch ➔ ↑Cardiac contraction➔ ↑CO -The greater stretch of the myocardial fibers ,the stronger the force of the contraction. مثل المطاط.

Mechanism of Starling's Law

*قدرة القلب على التكيف مع كمية الدم القادم اليه

- Increase In preload
- Increase in sarcomere length
- Increase troponin C calcium sensitivity
- Increase rate of cross-bridge attachment and detachment
- Amount of tension developed by the muscle fibers (force of contraction)

Increase the stroke volume

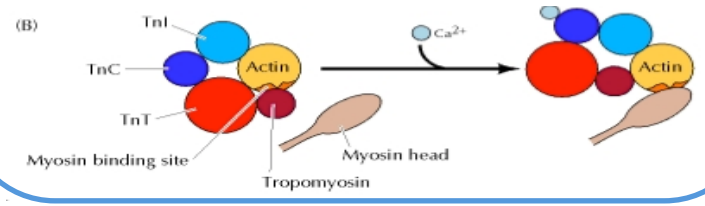


REMEMBER:

-Sarcomere:
Between 2 Z lines & its the unit of contraction

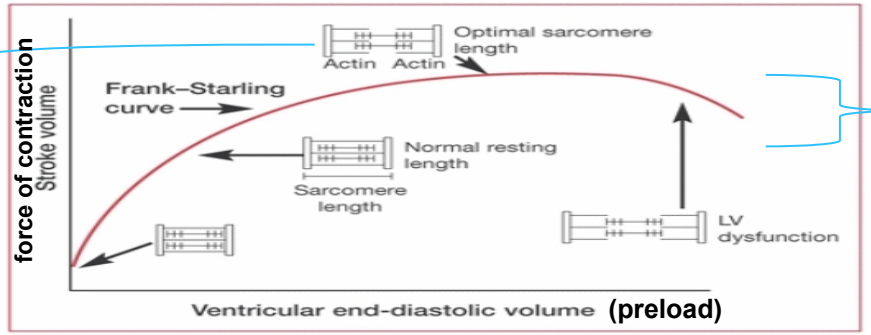
-Troponin c:
The part of troponin where calcium bind.

-Cross bridge: attachment of the actin & myosin



The troponin and myosin have to be at optimum interdigitation (التشابك الامثل)

The maximum stretch leads to stronger contraction



Drop of force of the cardiac contractility & stroke volume.

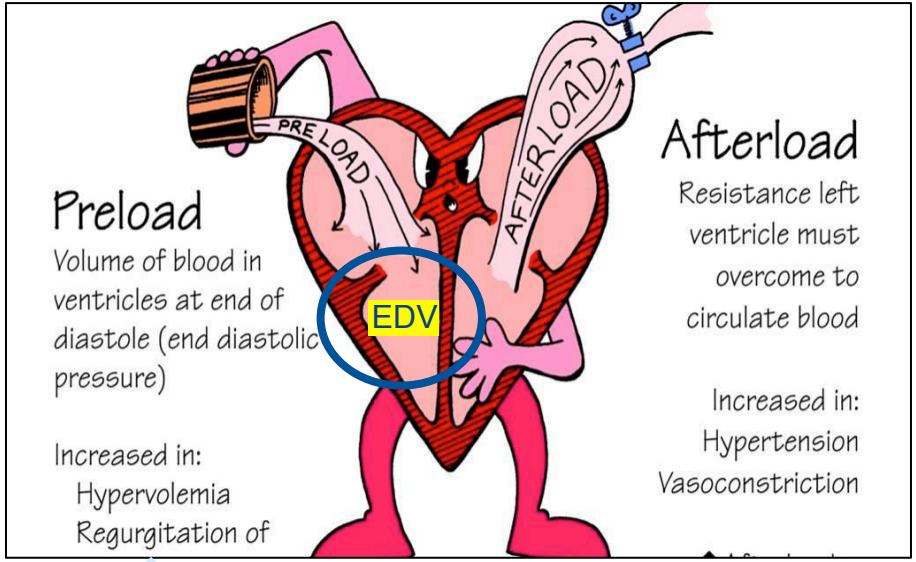
Over stretch that leads to loss of interdiction & can lead to muscle tear

End Diastolic Volume (EDV)

Recall! EDV is the amount of blood remaining in the heart by the end of diastole =(140mL)

Factors Affecting

Increase EDV ↑
Stronger Atrial Contraction↑
Increased Total Blood Volume↑
Increased Venous Tone↑ The tone that is present in the whole of the veins as a result of The sympathetic discharge that comes to it from the cardiovascular centre.
Increased Skeletal Muscle Pump↑
Increased -ve Intrathoracic Pressure↑
Decrease EDV ↓
Standing
Increased Intrapericardial Pressure↑
Decreased ventricular compliance ↓



The end diastolic volume (affected by the same factor that control the venous return

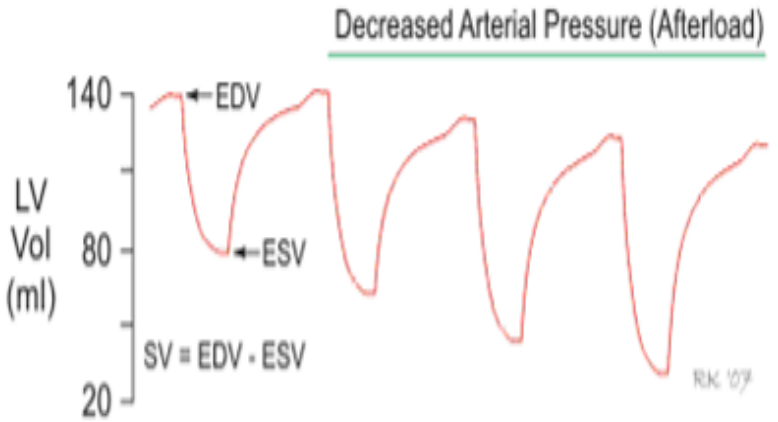
(Previously discussed)

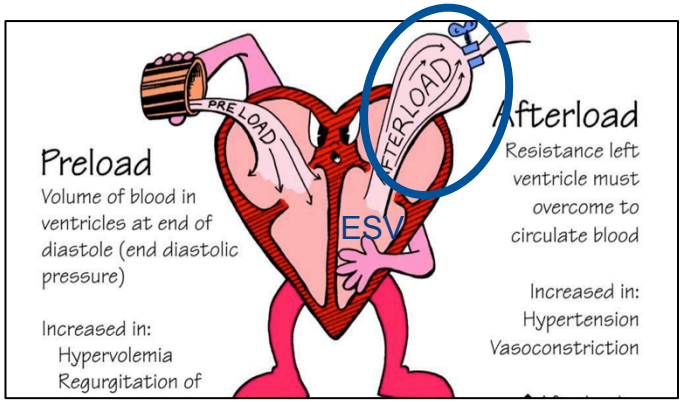
Afterload

Can be defined as the (load) that the heart must eject blood against

✓ The afterload is closely related to the **aortic pressure.**

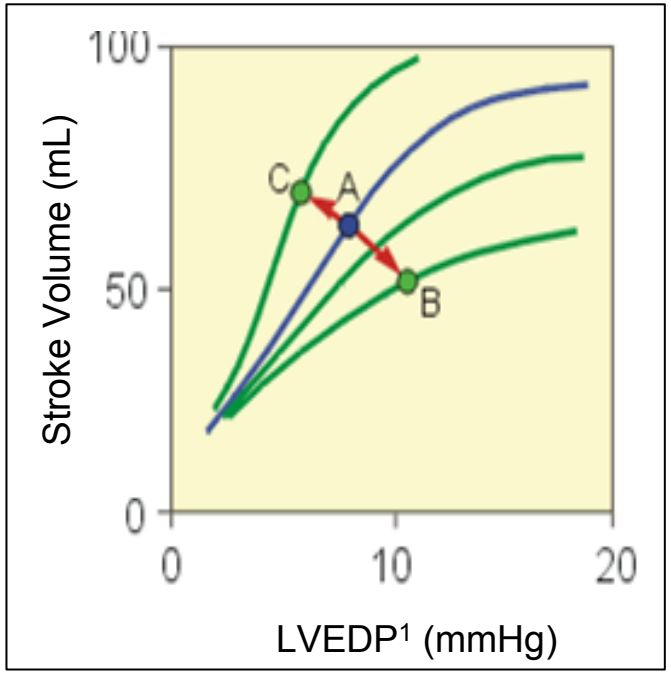
- When **Arterial pressure** is reduced ↓
- The **Ventricle** can eject blood more rapidly ↑
- Increases the **Stroke volume** ↑
- Decreases the **End-systolic volume** ↓





What is The Effect of an Increase in Afterload on Stroke Volume ?

- When afterload increases
- An increase in End-systolic volume ↑
- Decrease in Stroke volume ↓
- Shifts the frank-starling curve down and to the right (from A to B)



1: Left ventricular end diastolic pressure

Explanation: an increase in afterload decreases the velocity of fibre shortening, This reduces the rate of volume ejection so that more blood is left within the ventricle at the end of systole (increase end-systolic volume)

A decrease in afterload shifts the Frank-Starling curve up and to the left (A to C).

Factors Affecting Afterload

- ✓ **Vascular tone** (and therefore blood pressure)
- ✓ **Aortic stiffness**
- ✓ **Myocardial tension** (affected by hypoxia, volume overload)
- ✓ **Preload**
- ✓ **Valvular regurgitation**

Afterload is increased when..

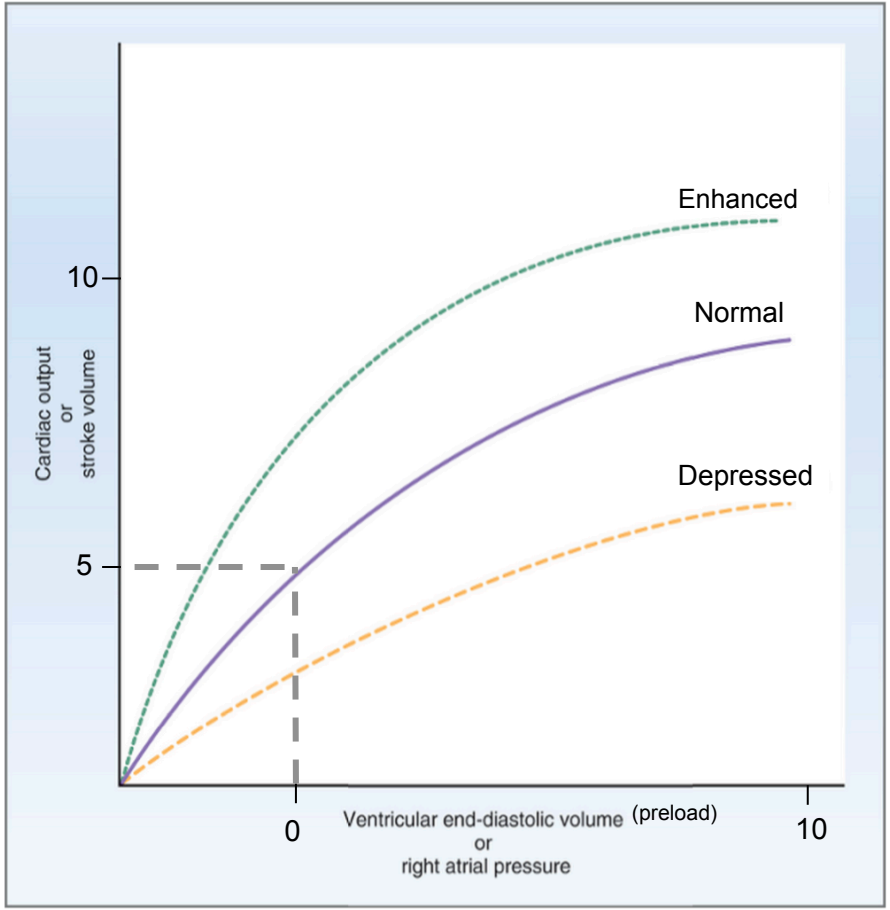
- ✓ Aortic stenosis
- ✓ Arterial hypertension

Extrinsic Regulation of Cardiac output :

- ✧ **Chemicals**
  - ✓ Potassium
  - ✓ Catecholamines
  - ✓ Calcium
  - ✓ Thyroxin
- ✧ **Nervous:**
  - ✓ Sympathatic
  - ✓ Parasympathatic

Cardiac Function Curve

If in a controlled experimental model right **atrial pressure**[1] is varied (independent variable) المتغير.
and the **cardiac output** measured (dependent variable) المتغير التابع.



- ✓ You will find that as RAP is increasing ➔ the CO increases.
- ✓ When the PRA[2] is about 0 mmHg (note that PRA normally fluctuates with atrial contraction and respiration), the cardiac output in an adult human is about 5 L/min.
- ✧ Because of the steepness انحدار of the cardiac function curve, very small changes in PRA (just a few mmHg) can lead to large changes in CO

1: Venous Return (Preload) ➔Right atrial pressure ➔EDV

2: PRA = Pressure of Right Atrial

Measurement of Cardiac Output

Old Way

Frick Principle

$CO = VO_2 / (O_2 art - O_2 ven)$

- VO_2 : Body Oxygen consumption.
  measured by **analyzing expired air**.
- O_2 art : Arterial Oxygen contents.
- O_2 ven : Mixed venous
  measured by **blood sample from each Ven. & Art.**

The blood content of O_2 are expressed as :
mL O_2 /mL blood

VO_2 expressed as:
ml O_2/ min

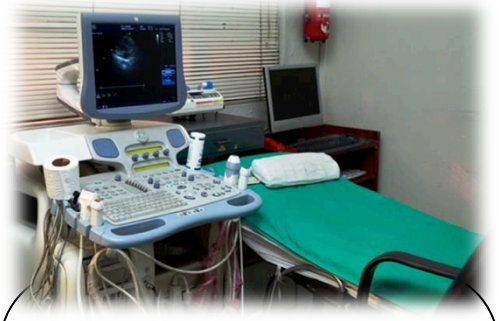
if O_2 art & O_2 ven content are (0.2 mL) & (0.15 mLO_2/ml blood) respectively, and VO_2 is 250 ml O_2/min.
How much will be CO?
CO = 250 ml O_2/min / (0.2 ml - 0.15 mlO_2/min)
CO = **5000** ml/min or **5** L/ min .

Example

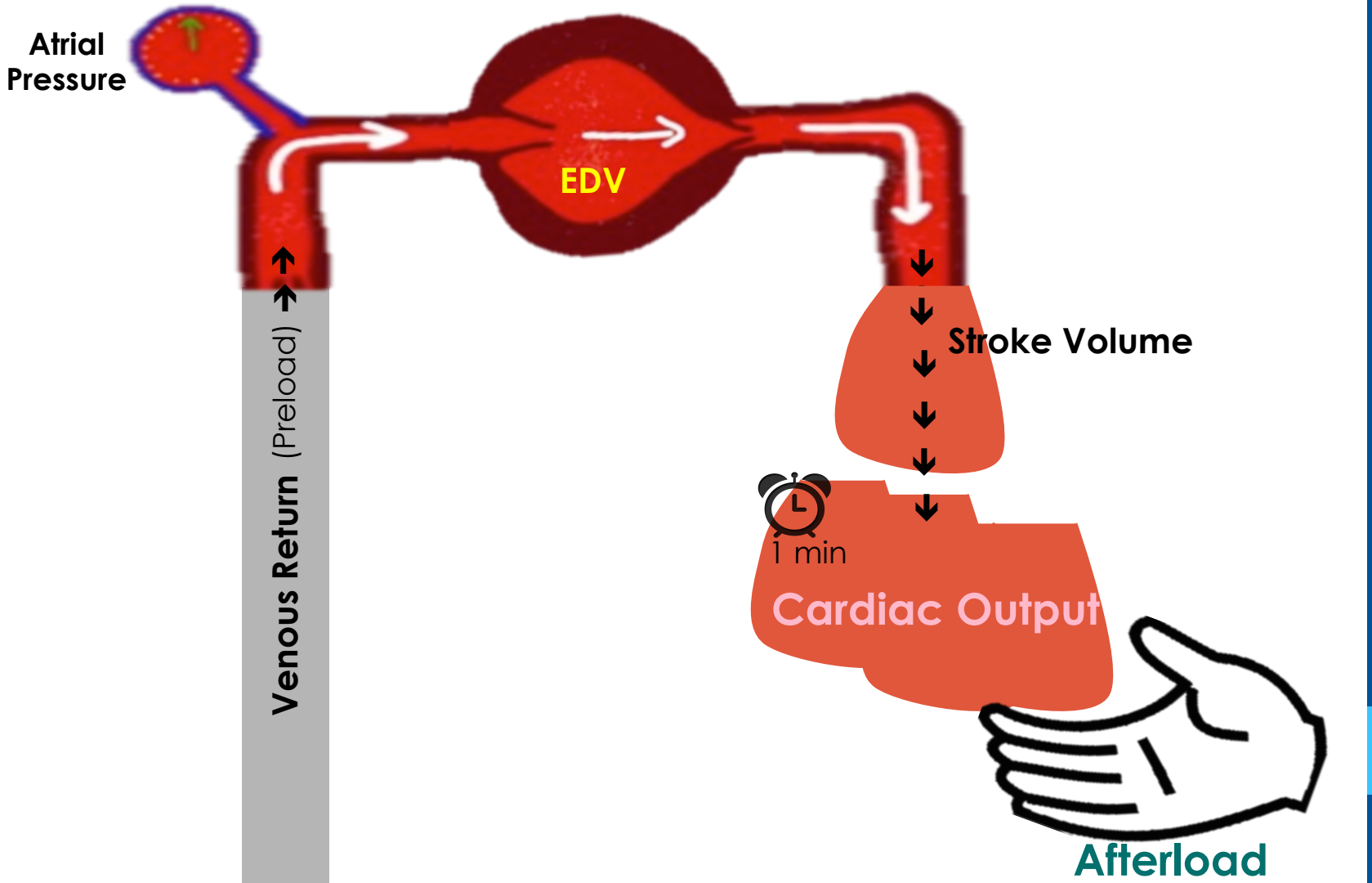
Ventricular Stroke Volume : CO / HR
Heart rate will be given then just multiply the HR by the CO

New Way

Echocardiography



- Record Real-Time changes in ventricular dimensions, thus computing Stroke Volume.
- Which when multiplied by the heart rate, gives CO.



Simple illustration

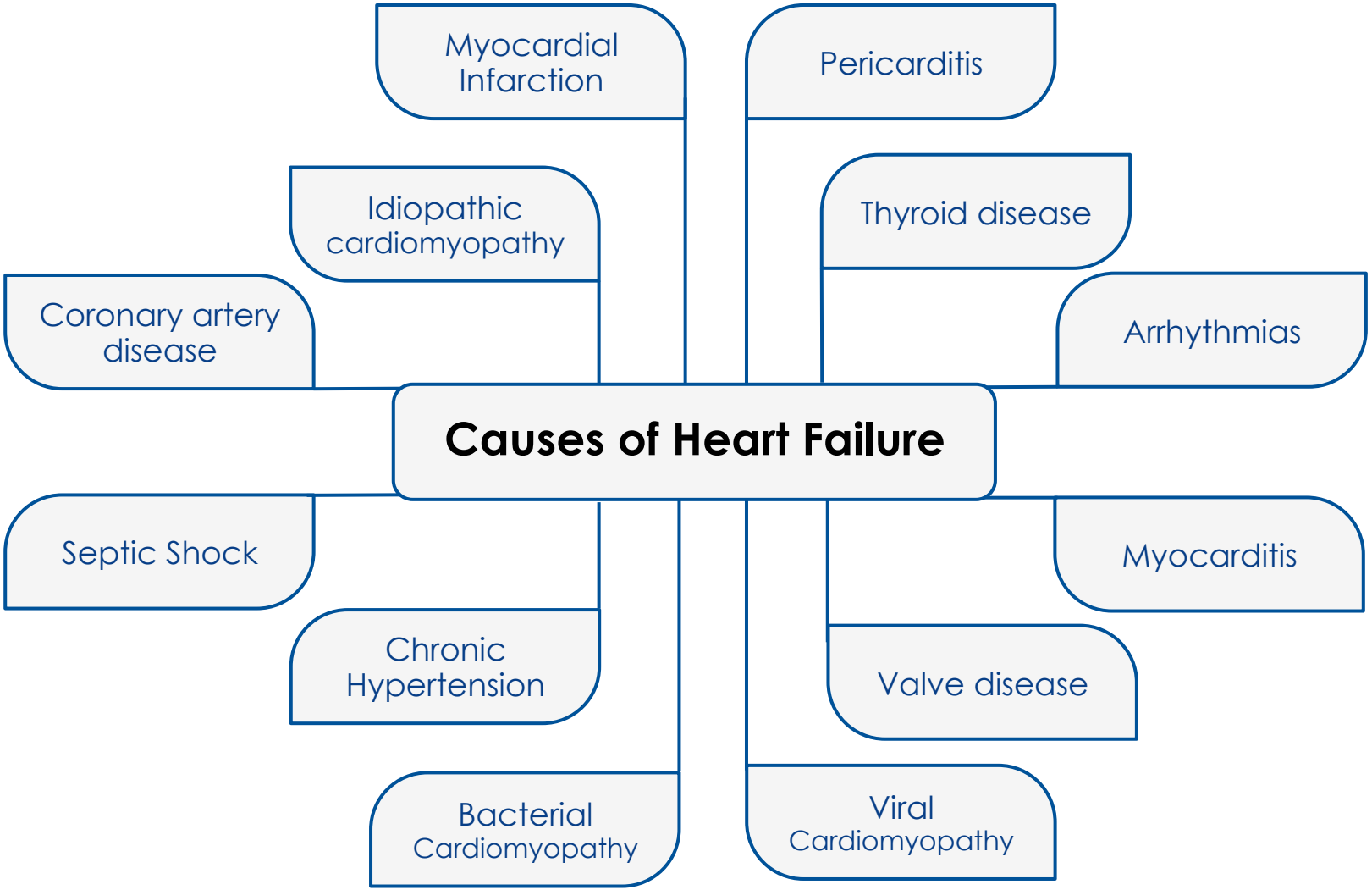
Heart Failure

(Congestive Heart Failure)

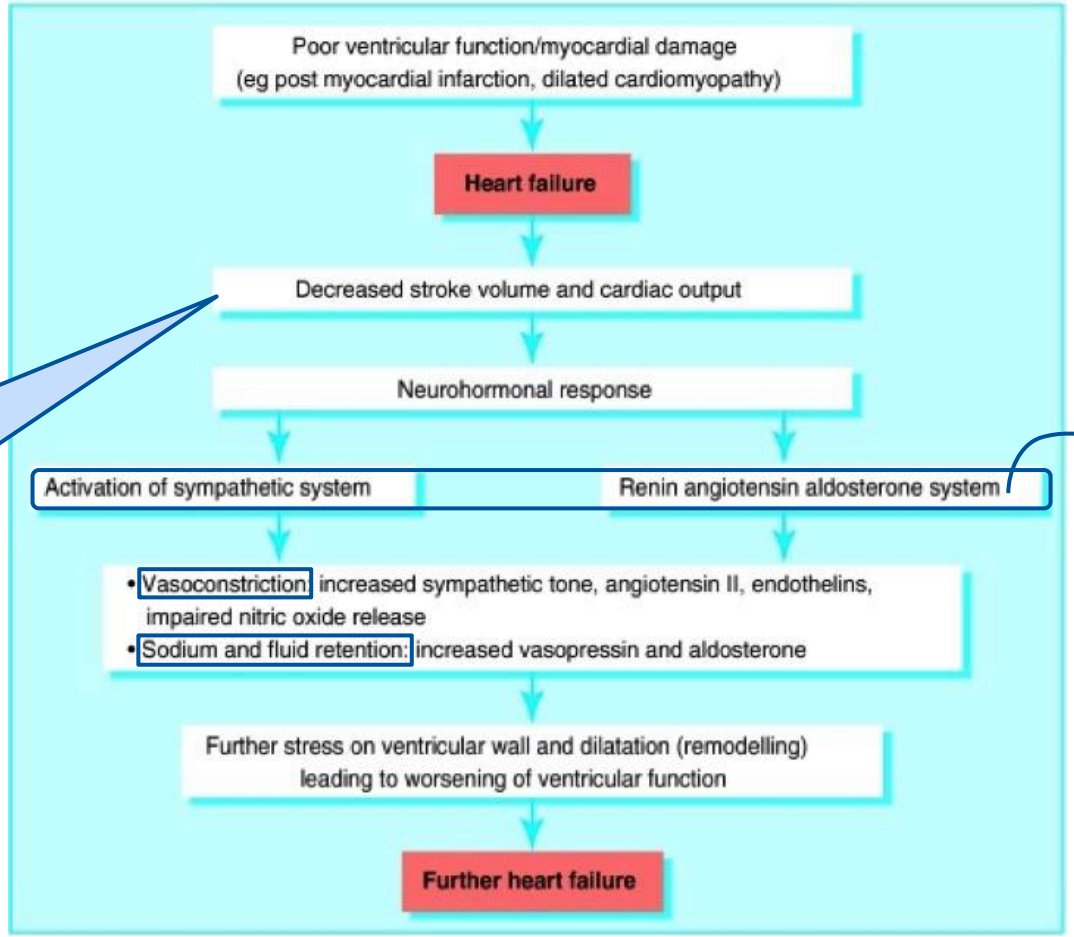
The inability of the heart to pump adequate output for the body metabolism needs

- ✧ **Manifested mainly by:**
- ✓ Inadequate cardiac output
- ✓ Build-up of blood in veins behind left heart or right heart (increased venous pressure)

	Acute Heart Failure	Chronic Heart Failure
Development	Rapid	Long-term
Duration	Hours/Days	Months/Years
Causes	1-Cardiopulmonary Bypass Surgery. 2-Acute infection (Sepsis). 3-Acute Myocardial infarction. 4-Severe Arrhythmias.	-The most common cause of CHF is a heart attack which causes damage to the muscles of left ventricle.
Does it undergo Adaptive Response?	life threatening because the heart does **not have time to undergo compensatory adaptations.**	Associated with the heart **undergoing Adaptive Responses** (E.g. Dilation, Hypertrophy), however it can be deleterious.


Myocardial Infarction
Pericarditis
Idiopathic cardiomyopathy
Thyroid disease
Coronary artery disease
Arrhythmias
Causes of Heart Failure
Septic Shock
Myocarditis
Chronic Hypertension
Valve disease
Bacterial Cardiomyopathy
Viral Cardiomyopathy

Overall Mechanism Route of Heart Failure



In response to these changes the body will immediately release hormones (Compensatory mechanisms) that will activate the Sympathetic and RAS[1] systems.

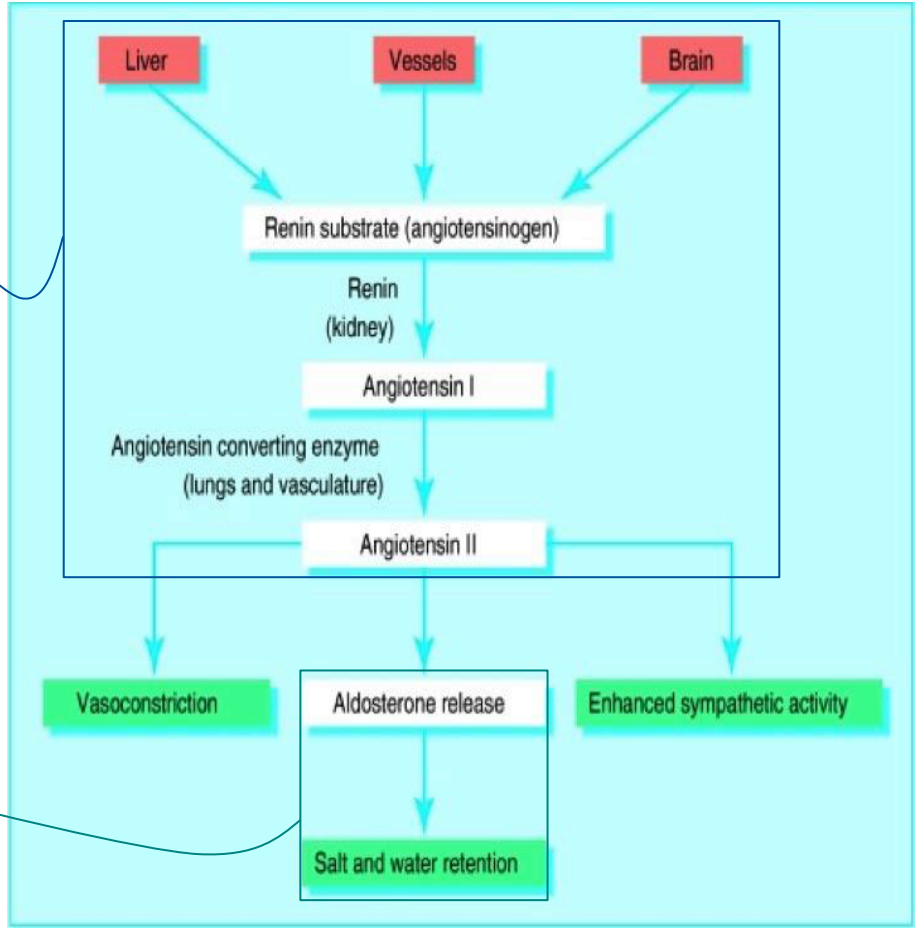
Causes of the reduced renal output of urine during cardiac failure.

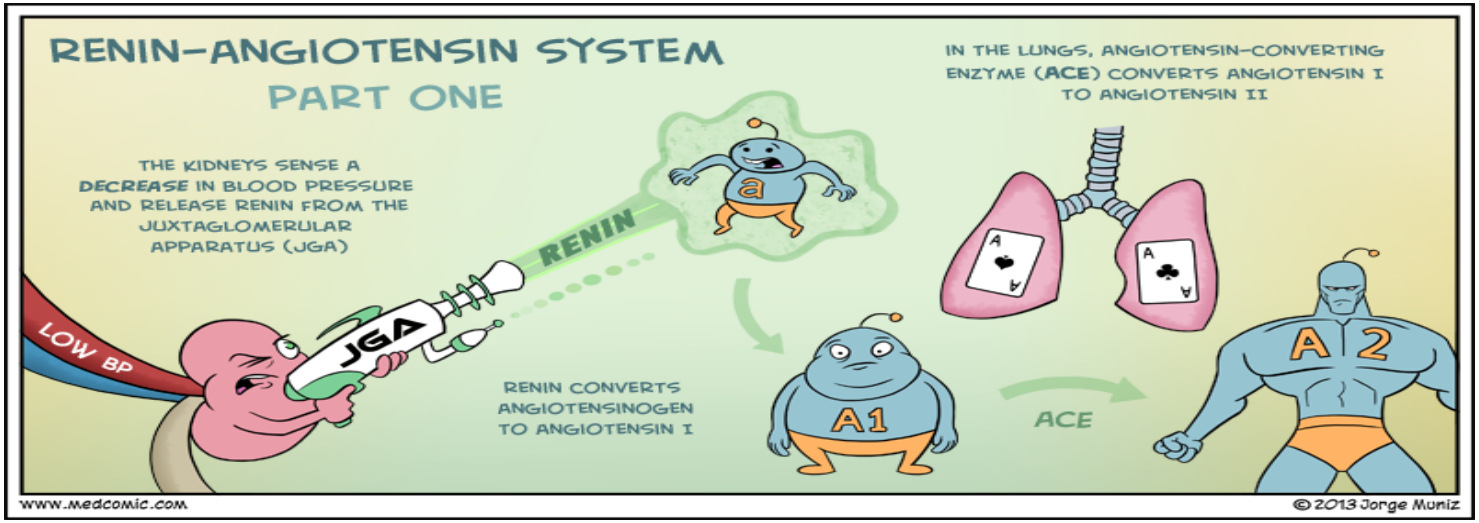
1: Renal Artery Stenosis

RAS System Effect on Heart Failure

Reduced blood flow to the kidneys causes marked increase in *renin secretion.* this in turn increases the *formation of* ***angiotensin II.***

In the chronic stage of heart failure, large quantities of **aldosterone are secreted** by the adrenal cortex. Which results by the effect of angiotensin.
This elevation of aldosterone level increases the reabsorption of sodium and water from the renal tubules.




RENIN-ANGIOTENSIN SYSTEM
PART ONE
THE KIDNEYS SENSE A DECREASE IN BLOOD PRESSURE AND RELEASE RENIN FROM THE JUXTAGLOMERULAR APPARATUS (JGA)
LOW BP
JGA
RENIN
a
RENIN CONVERTS ANGIOTENSINOGEN TO ANGIOTENSIN I
A1
IN THE LUNGS, ANGIOTENSIN-CONVERTING ENZYME (ACE) CONVERTS ANGIOTENSIN I TO ANGIOTENSIN II
ACE
A2
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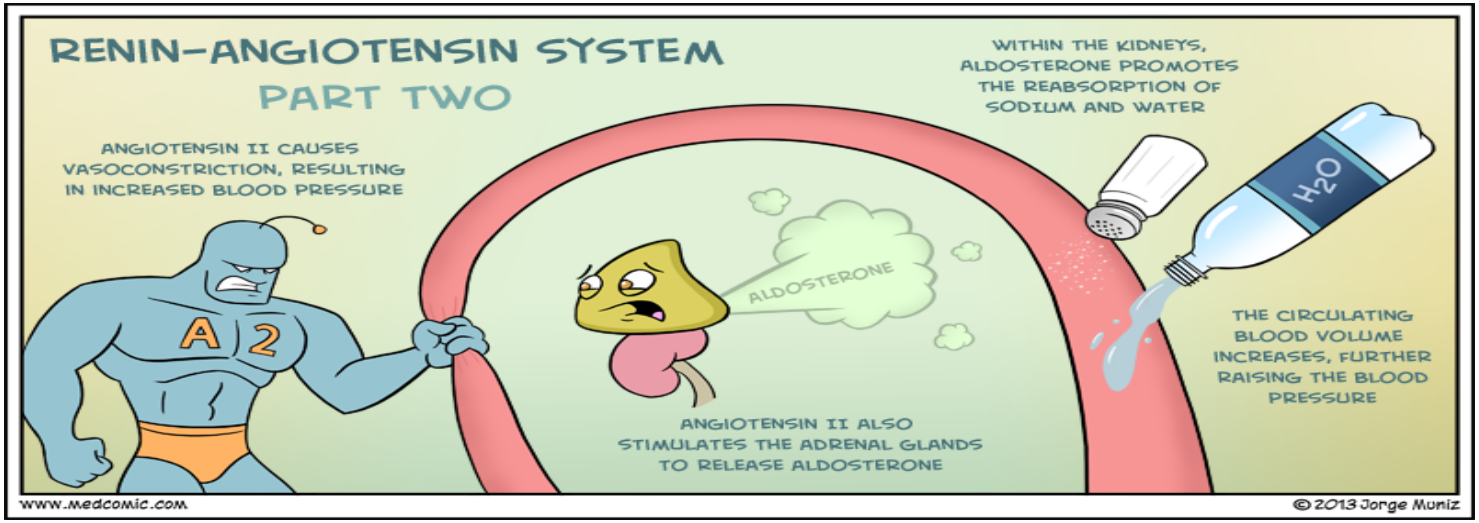
RENIN-ANGIOTENSIN SYSTEM
PART TWO
ANGIOTENSIN II CAUSES VASOCONSTRICTION, RESULTING IN INCREASED BLOOD PRESSURE
A2
ALDOSTERONE
ANGIOTENSIN II ALSO STIMULATES THE ADRENAL GLANDS TO RELEASE ALDOSTERONE
WITHIN THE KIDNEYS, ALDOSTERONE PROMOTES THE REABSORPTION OF SODIUM AND WATER
H2O
THE CIRCULATING BLOOD VOLUME INCREASES, FURTHER RAISING THE BLOOD PRESSURE
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Additional

The significance of Natriuretic Peptides

They are a family of peptides that share a common ring structure.

ANP[1]: is a hormone released by the **atrial walls** of the heart when they become stretched

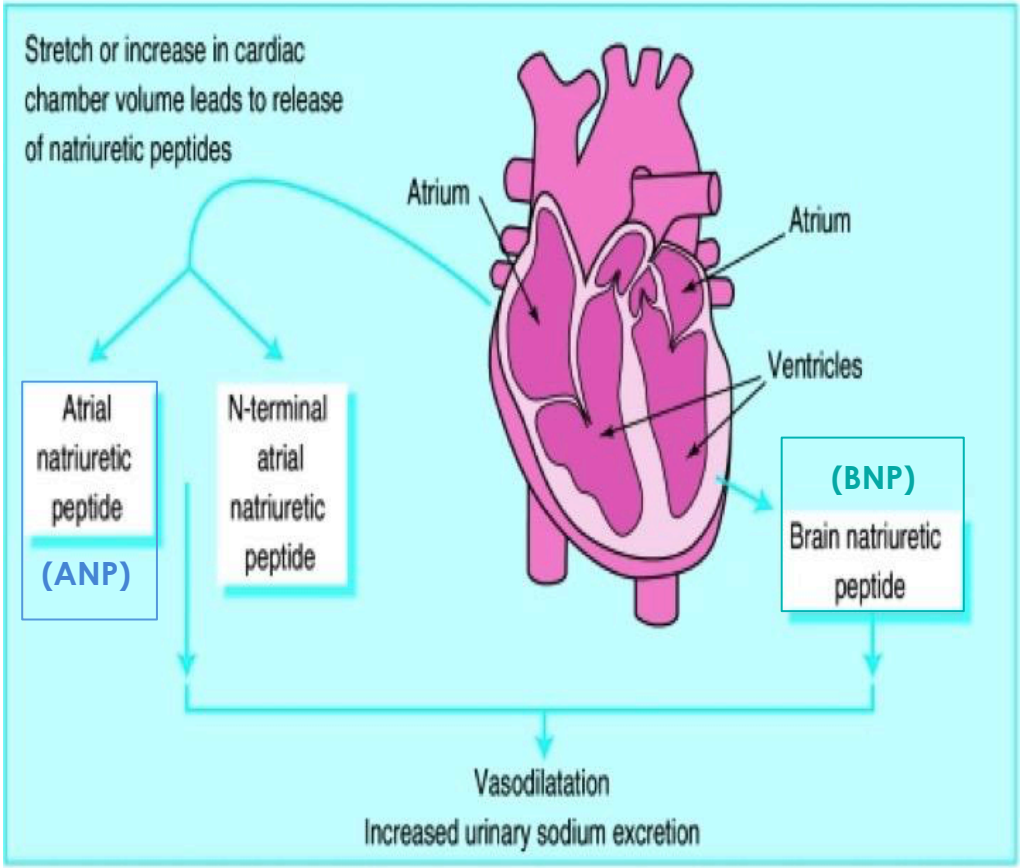
It increases in the blood by 5- to 10-fold in severe heart failure.

✓ It has a direct effect on the kidneys to increase greatly their excretion of salt and water.

✓ ANP plays a natural role to help **prevent extreme congestive symptoms** during cardiac failure.

BNP[2]: Present in high concentrations in cardiac tissues, particularly the ventricles.

✓ An elevated BNP level is a marker of **increased LV filling pressure and LV dysfunction**

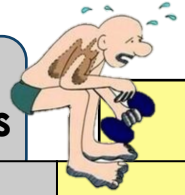


Types of Heart Failure

	Left-Sided HF	Right-Sided HF
Pathogenesis	Increase Aortic pressure causes backward pressure travels to Left Ventricle ➔Then Pulmonary Veins ➔Finally goes to the lung ➔Sort of accumulation of the blood in the lung that can not go back to the heart, Causing **Pulmonary Edema** present as area of consolation on the X-ray imgings.	Increase Right Ventricle pressure causes backward pressure on Right Atria ➔ Increasing right atrial pressure ➔ Stagnation of the blood On the: ✓ Superior Vena Cava ➔ Increased Jugular venous pressure ✓ Inferior Vena Cava ➔Ascites[1]
Signs, Symptoms & (manifestations)	✧ **Respiratory manifestations:** ✓ **Tachypnea:** increased work of breathing ✓ **Crackle sound:** it is a sign of development of pulmonary edema, heard initially in the lung bases, however if severe it can be heard throughout the lung fields. ✓ **Cyanosis**: which suggests severe hypoxemia, is a late sign of severe pulmonary edema. ✧ **Cardiac manifestations:** ✓ **Lateral displacement of apex beat** (Which occurs in the case of cardiomegaly –enlargement) ✓ **Gallop rhythm :** (additional heart sounds) may be heard as a marker of increased blood flow, or increased intra-cardiac pressure.	✧ **Symptoms**: ✓ **Ascites** (backward pressure of Inferior vena cava on the hepatic circulations) ✓ **Increased Jugular venous pressure** (backward pressure of Superior vena cava on the hepatic circulations) ✓ **Hepatomegaly** (backward pressure of Inferior vena cava on the hepatic circulations) ✓ **Pitting peripheral edema**

1: An accumulation of fluid in the peritoneal cavity. (Abdomen)

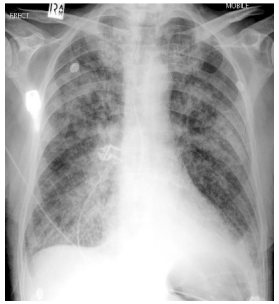
Types of Heart Failure (Signs & Symptoms)



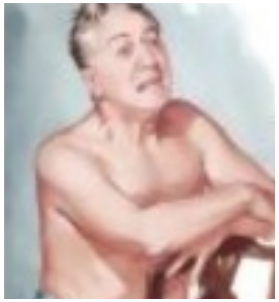
Signs & Symptoms	Left-Sided HF	Right-Sided HF
Pitting Edema (legs and hands)	Mild to moderate	Moderate to sever
Fluid Retention	Pulmonary edema (fluid in lungs) Pleural effusion (fluid around lungs)	Abdomen (Ascites)
Organ Enlargement	Heart	Liver (Mild jaundice may be present)
Neck Veins	Mild to moderate raised in jugular venous vein pressure (JVP)	Severe raised in jugular venous vein pressure (JVP), Neck veins visibly distended
Shortness of breath	Prominent dyspnea Paroxysmal nocturnal dyspnea (PND)	Dyspnea present but not as prominent
Gastrointestinal	Present but not as prominent	Loss of apatite Bloating (any abnormal general swelling) Constipation Symptoms are more prominent

Comparison

Left-Sided HF

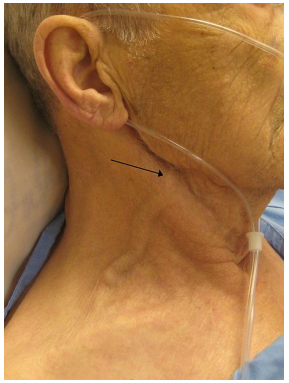


Pulmonary Edema
As area of consolation

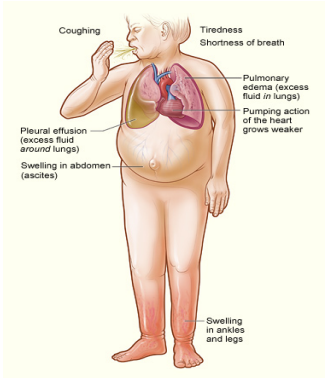


Dyspnea

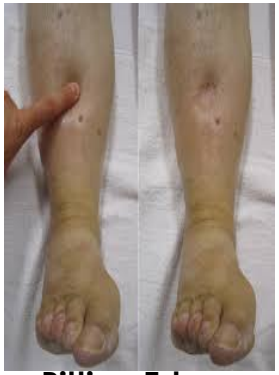
Right-Sided HF



Elevated JVP



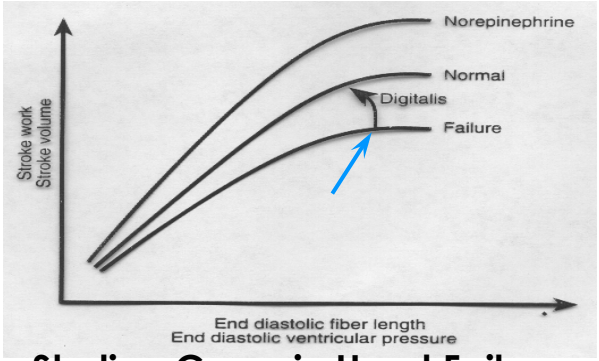
Ascites



Pitting Edema

Physiological adaptation to Heart Failure
(compensation mechanisms)

- ✧ **Reduction of cardiac workload, including both preload and afterload.**
  - ✓ Increased sympathetic stimulation:
    - *Vasoconstriction
    - * ↑HR
    - * ↑BP
- ✧ **Enhancement of myocardial contractility.**
  - ✓ Increased sympathetic stimulation:
    - *↑Force of contraction
- ✧ **Control of excessive retention of salt and water.**
  - ✓ Greater blood volume → peripheral and pulmonary edema



Starling Curve in Heart Failure

Complications of Progressive HF

- ✧ Prolonged sympathetic activation to the heart
- ✧ Hypertrophied heart
- ✧ Excessive salt and water retention:
- ✧ Over-distended ventricle

MCQs

1- In normal conditions Venous return (preload) must be:
A. Equal to cardiac output
B. Less than cardiac output
C. Greater than cardiac output

2- Excessive increase in heart rate:
A. Increase cardiac output
B. Decrease cardiac output

3- Which one of the following causes an increase in stroke volume?
A. Increase preload
B. Decrease preload
C. Gravity

4- 44 years old women came to the ER having a Heart Failure and her physical examination reveals pitting peripheral edema, ascites, and hepatomegaly. Which mostly type of Heart failure is she having ?
A. Left-sided heart failure
B. Right-sided heart failure

1.A
2.B
3.A
4.B

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