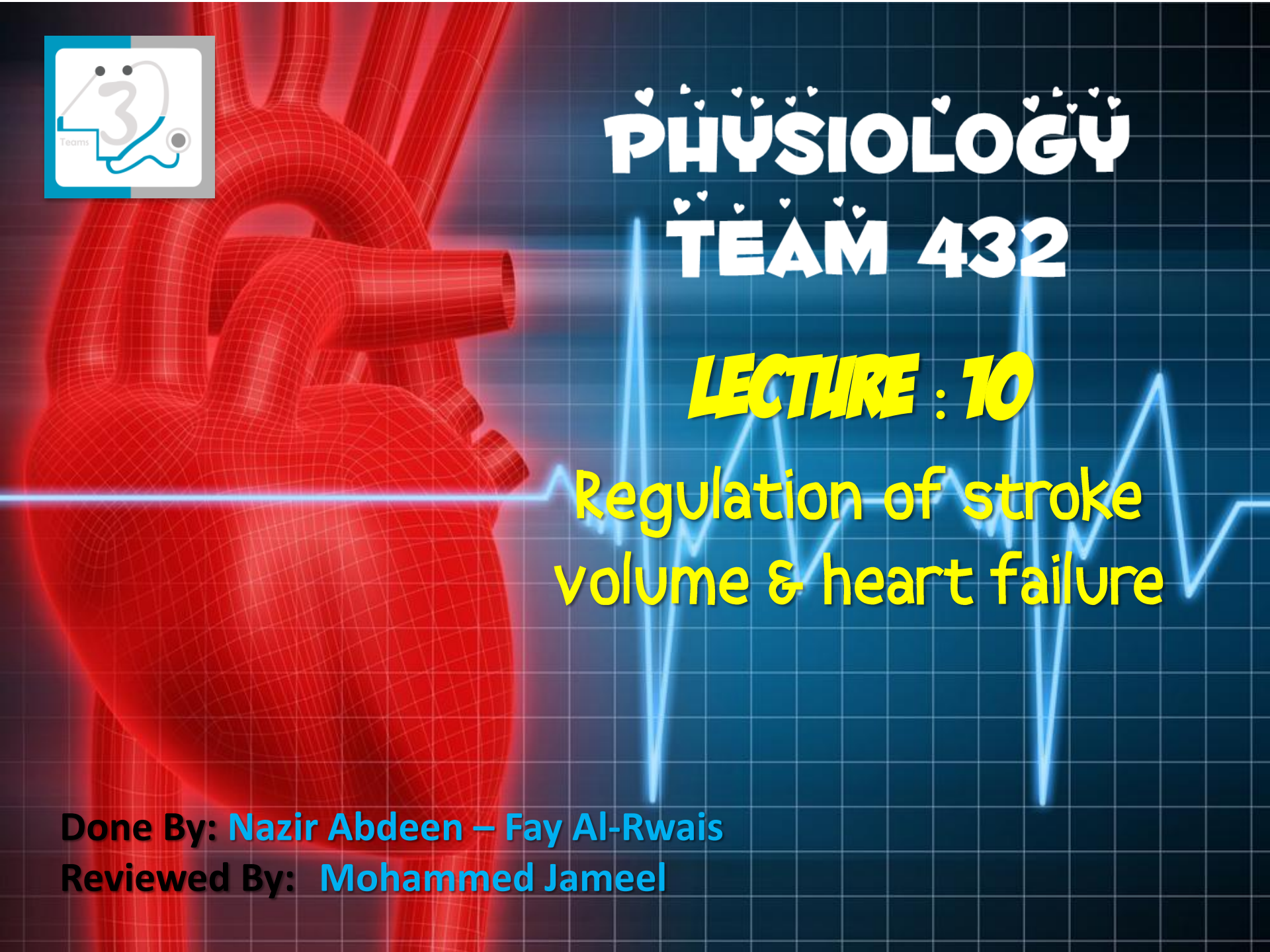

PHYSIOLOGY TEAM 432

LECTURE : 10

Regulation of stroke volume & heart failure

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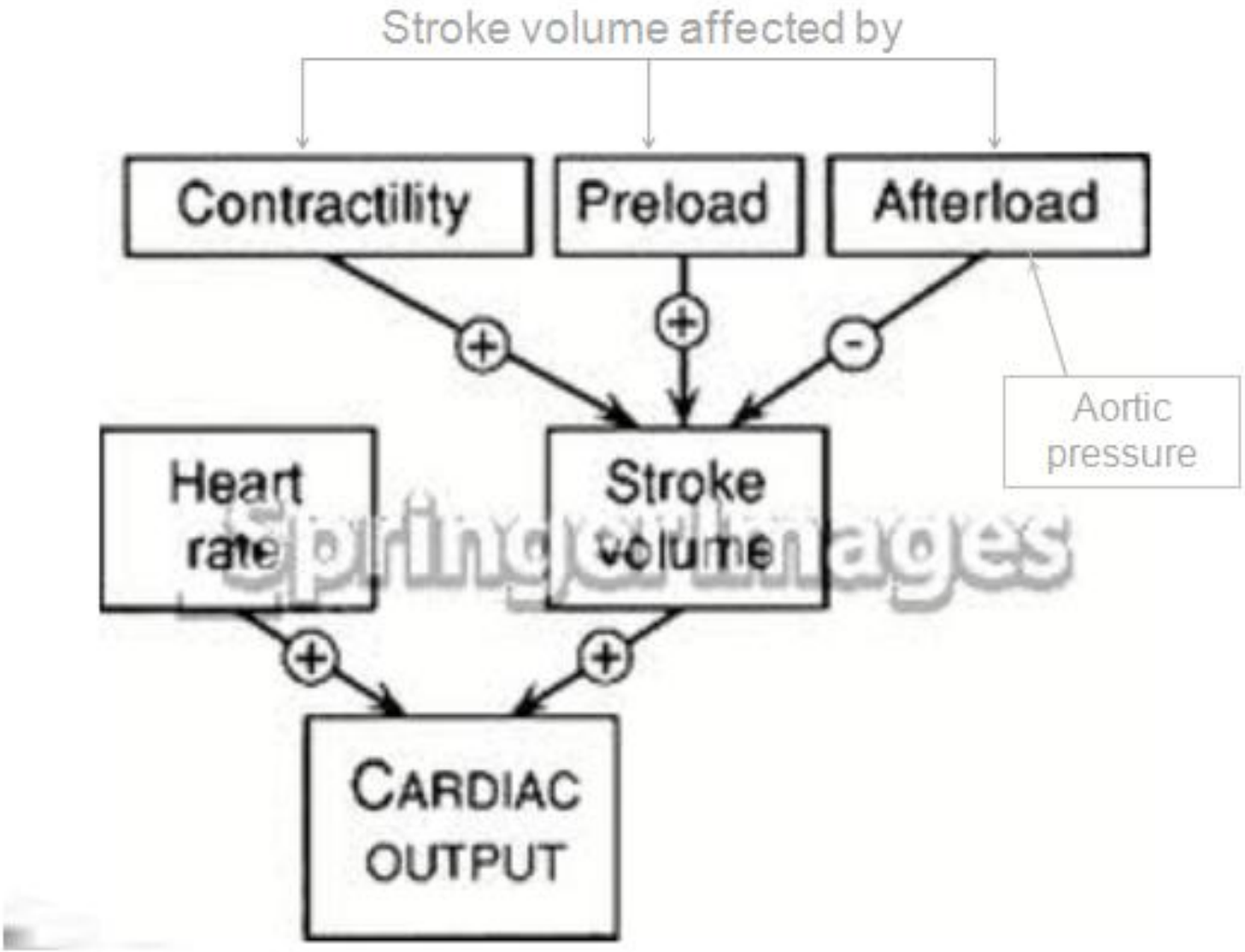
Reviewed By: Mohammed Jameel

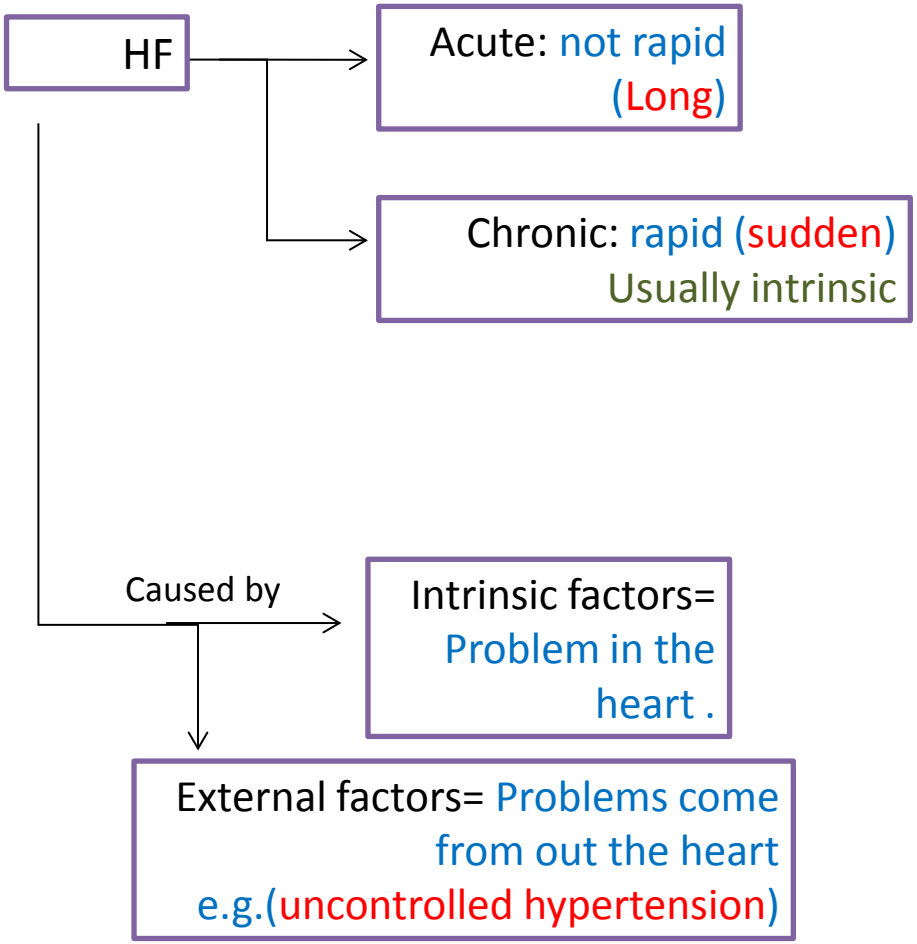
OBJECTIVES

By the end of this lecture the students are expected to:

A. Define cardiac output, stroke volume, end-diastolic and end-systolic volumes.
B. Define physiological conditions affecting CO
C. List causes of high and low output pathological states.
D. Define venous return and describe factors controlling venous return


Stroke volume affected by
Contractility
Preload
Afterload
Aortic pressure
Heart rate
Stroke volume
CARDIAC OUTPUT

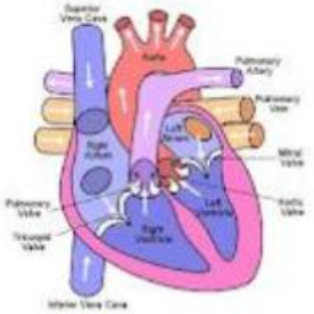
MIND MAP


HF
Acute: not rapid (Long)
Chronic: rapid (sudden) Usually intrinsic
Caused by
Intrinsic factors= Problem in the heart .
External factors= Problems come from out the heart e.g.(uncontrolled hypertension)

Lecture outline

- Cardiac output is the amount of blood pumped by each ventricle per minute (5L/min). It varies physiologically with age, body mass index, physical activity, sleep, meals, pregnancy, etc.. But there are pathological conditions that lead to a significant increase in CO including hyperthyroidism, anemia and conditions decreasing CO as myocardial infarction. CO is well controlled and regulated by Many Factors: venous return, ABP , blood volume and nervous regulation. This lecture will focus on Venous return as an important factor determining CO. Venous return represents the amount of blood returning to the heart per minute. Venous return is controlled by many factors:1) Frank-Starling's mechanism, 2) mean systemic filling pressure, 3) tissue metabolism, 4) thoracic pump, 5) Gravity, 6) Muscle pump, 7) blood volume.

Definitions



- **Cardiac output:**
- Amount of blood pumped by each ventricle per minute.
- **Stroke volume (SV):**
- Volume of blood ejected by each ventricle/beat.
- **CO = SV x HR**
- Stroke volume is expressed in ml/beat and heart rate in beats/minute. Therefore, cardiac output is in ml/minute. Cardiac output may also be expressed in liters/minute.

End-diastolic volume (EDV):

Amount of blood remaining in the heart by the end of diastole.

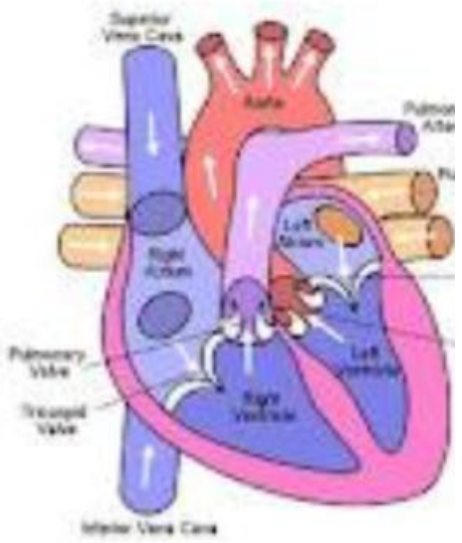
140 mL

End-systolic volume (ESV):

Amount of blood remaining in the heart by the end of systole.

=70 mL

SV = EDV-ESV

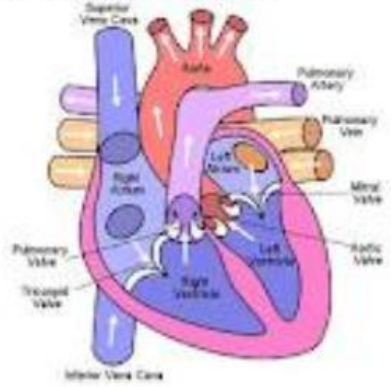


Stroke volume

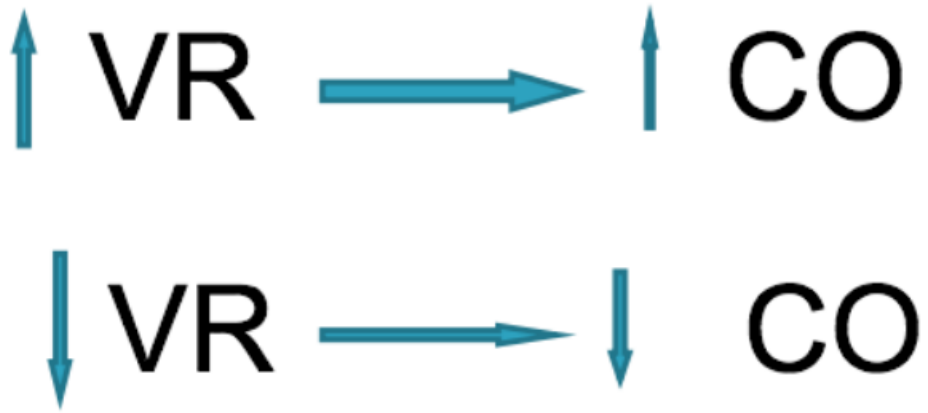
SV = EDV - ESV.

This measurement can be affected by changes in the heart's ability to contract, the force of contraction, the volume of blood available to be pumped or other variables such as resistance within the circulatory system that can affect or alter these factors. Severe hemorrhage or shock, heart damage or extreme infections can change the heart's ability to pump effectively.

It is affected by cardiac contractility, preload, and afterload.



VR & CO



Cardiac contractility and stroke volume

Systolic function of the heart is controlled by

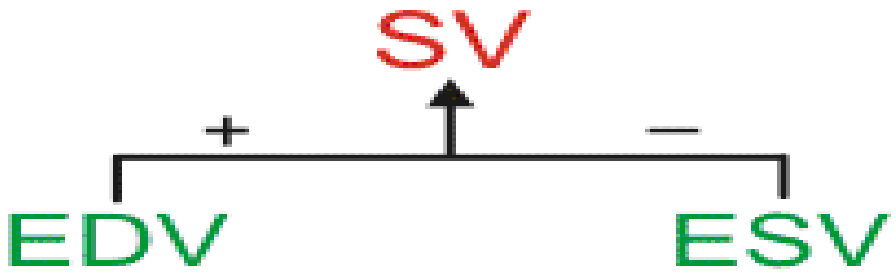
- Contractile state of the myocardium.
- Preload of the ventricle.
- Afterload applied to the ventricle.
- Heart Rate.

Preload (VR) :

increase in venous return to the heart, increases the filled volume (EDV) of the ventricle

Afterload (Aortic pressure) :

related to the pressure that the ventricle generate to eject blood into the aorta.



↑ Preload → ↑ SV (↑ EDV)

↑ Afterload → ↓ SV (↑ ESV)

↑ Inotropy → ↑ SV (↓ ESV)

Heart Failure

What is Heart Failure؟

It is a pathological process in which *systolic and /or diastolic function of the heart is impaired s a result ‹*

CO is low and unable to meet the metabolic demands of the body.

Pathophysiology of heart failure

Heart failure can be caused by factors originating from within the heart (i.e., intrinsic disease or pathology) or from external factors that place excessive demands upon the heart.

Intrinsic factors:
dilated cardiomyopathy and hypertrophic cardiomyopathy، myocardial infarction.

External factors:
-long-term, uncontrolled hypertension, - increased

stroke volume:
(volume load; arterial-venous shunts), hormonal disorders such as hyperthyroidism.

Causes of Heart Failure

- Myocardial infarction
- Coronary artery disease
- Valve disease
- Idiopathic cardiomyopathy
- Viral or bacterial cardiomyopathy
- Myocarditis
- Pericarditis
- Arrhythmias
- Chronic hypertension
- Thyroid disease
- Septic shock
- Aneamia

Acute HF

Acute heart failure develops rapidly and can be immediately life threatening because the heart does not have time to undergo compensatory adaptations. Acute failure (hours/days) may result from cardiopulmonary by-pass surgery, acute infection (sepsis), acute myocardial infarction, severe arrhythmias, etc.

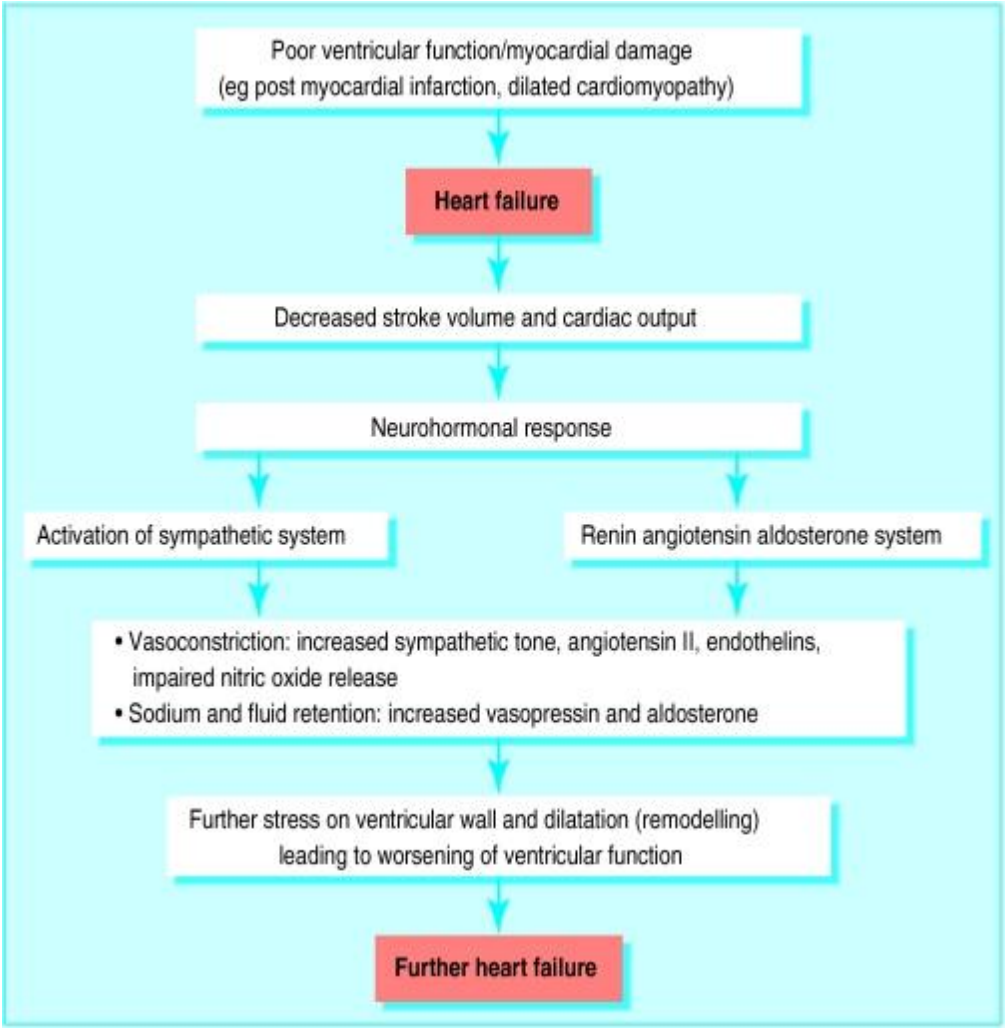
Acute heart failure can often be managed successfully by pharmacological or surgical interventions.

Chronic HF

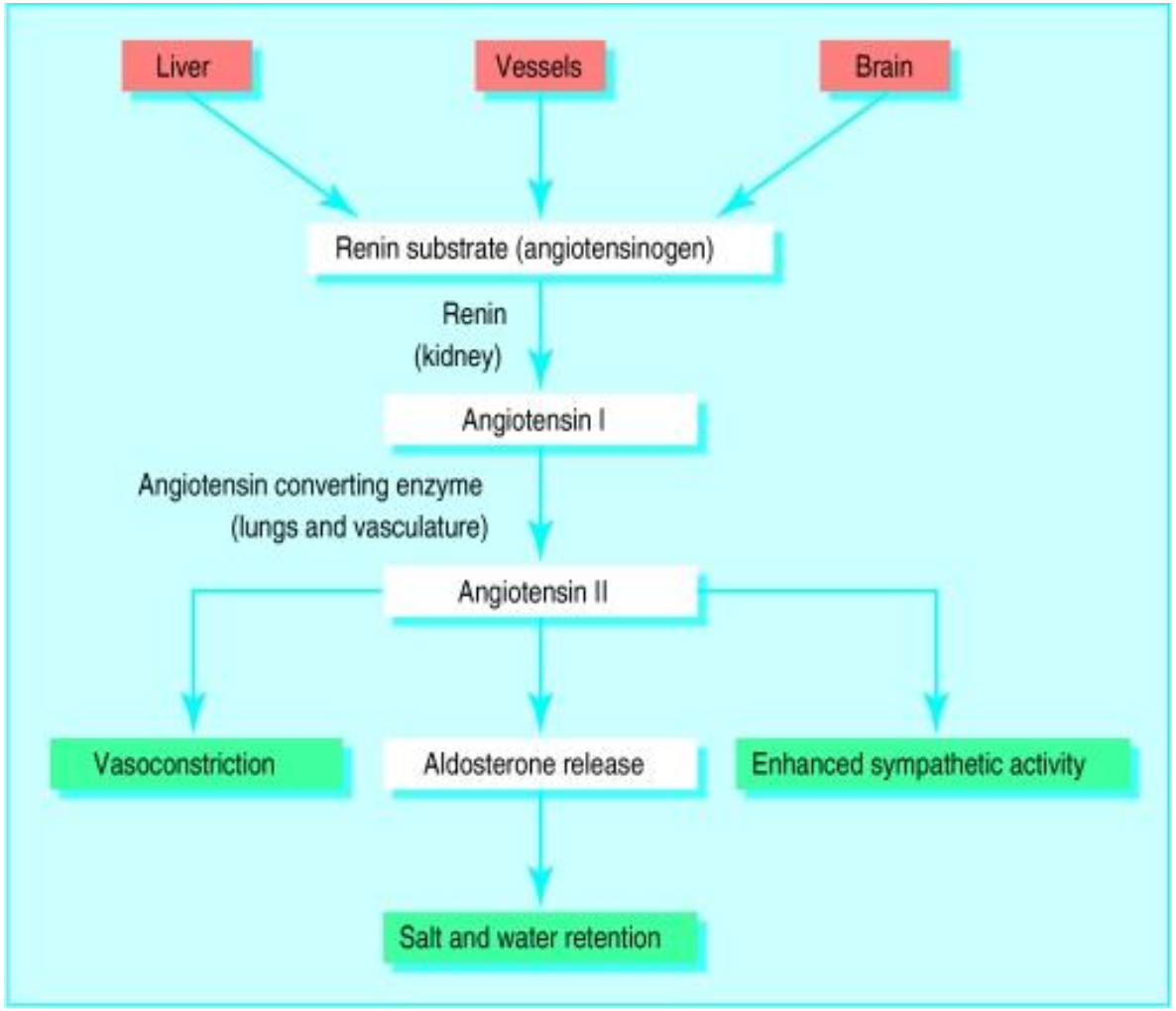
Chronic heart failure is a long-term condition (months/years) that is associated with the heart undergoing adaptive responses (e.g., dilation, hypertrophy) .These adaptive responses, however, can be deleterious؟

Neurohormonal mechanisms and compensatory mechanisms in heart failure

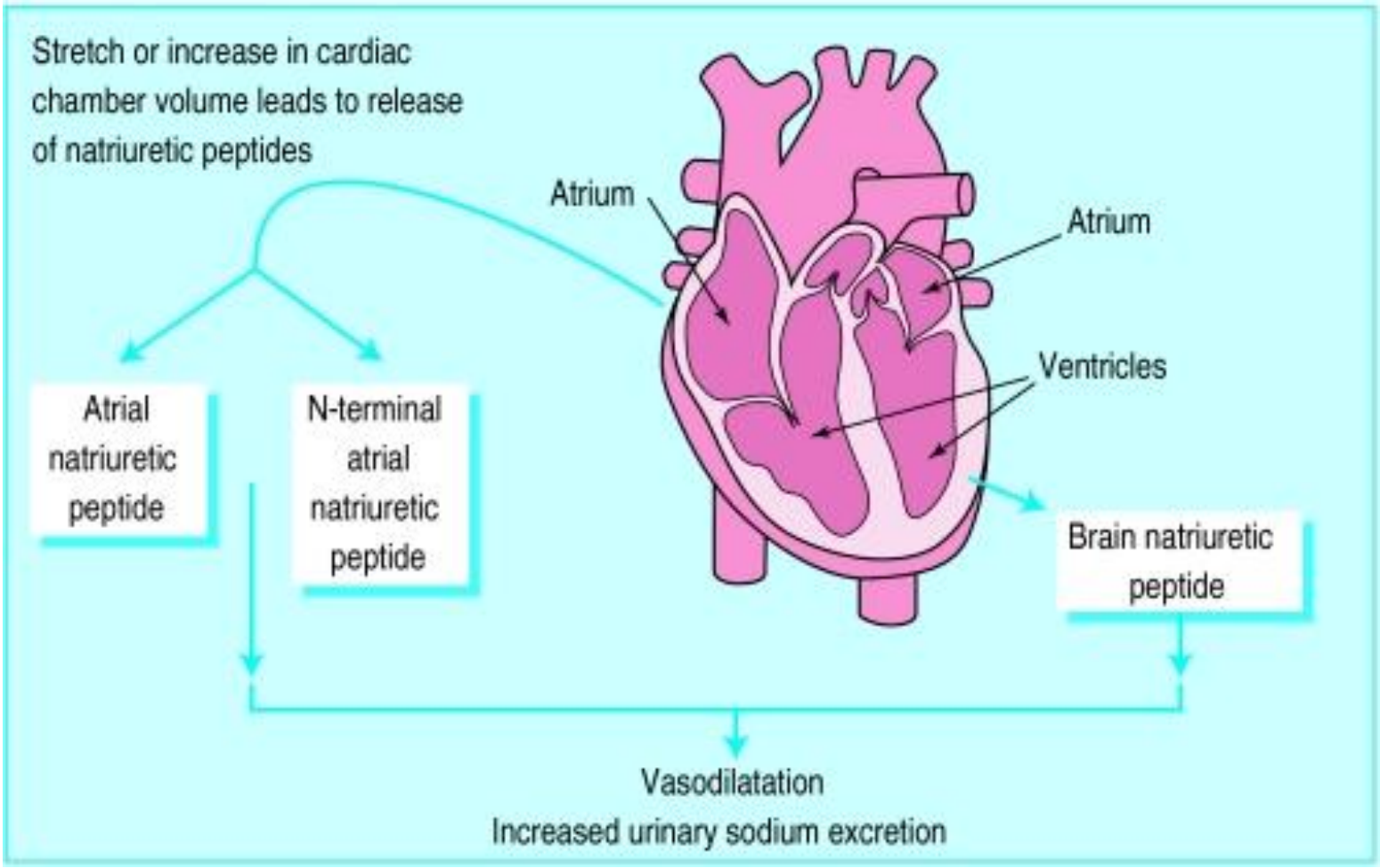
HF patients always have tachycardia Because of sympathetic system (adaptation mechanism)



Renin-angiotensin-aldosterone system in HF



Effect of natriuretic peptide



natriuretic peptide

Na+

Exerted in urine

natriuretic peptide

from

Atrium

Released when

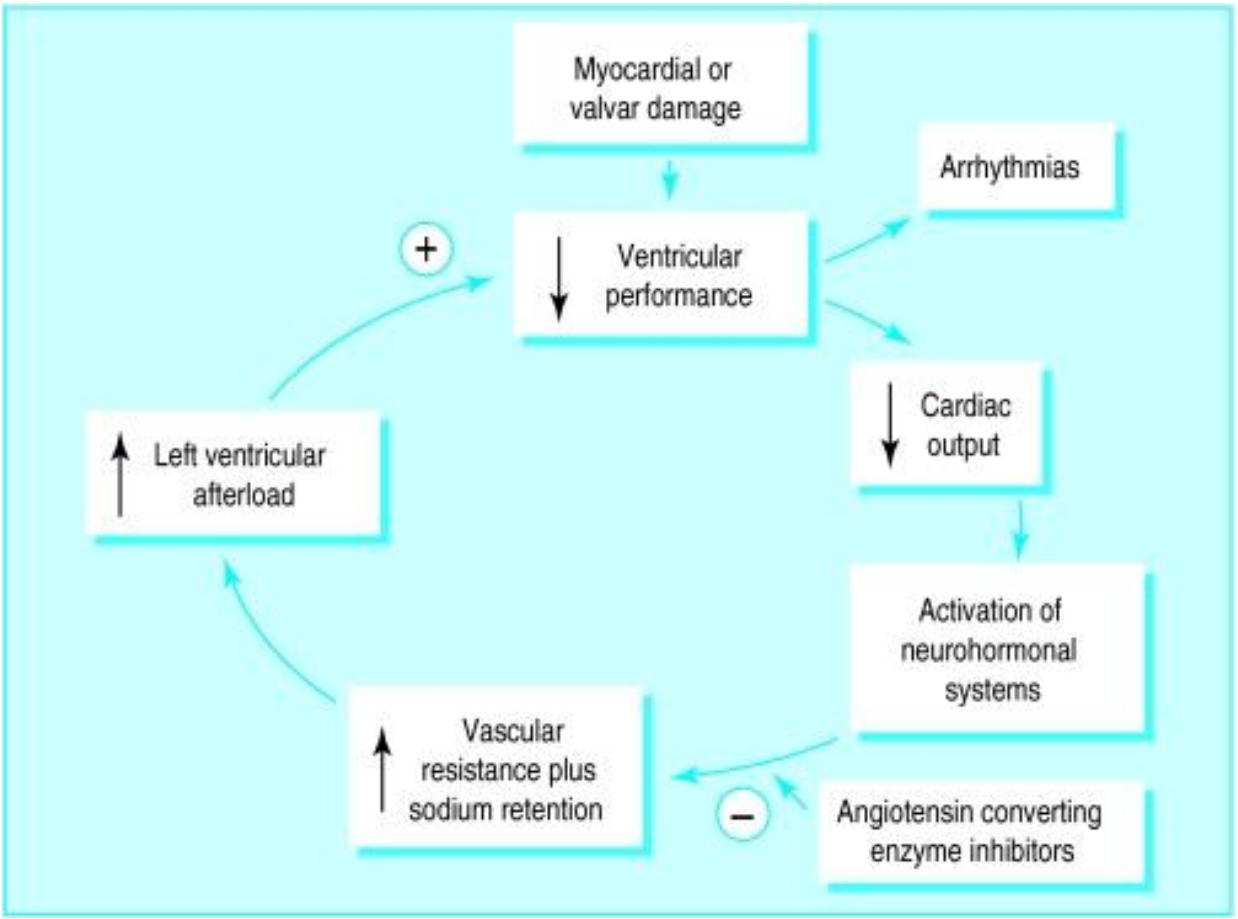
Blood volume increased

Heart is endocrine organ because it release hormones

Summary of the consequences to the neurohormonal responses to impaired cardiac performance

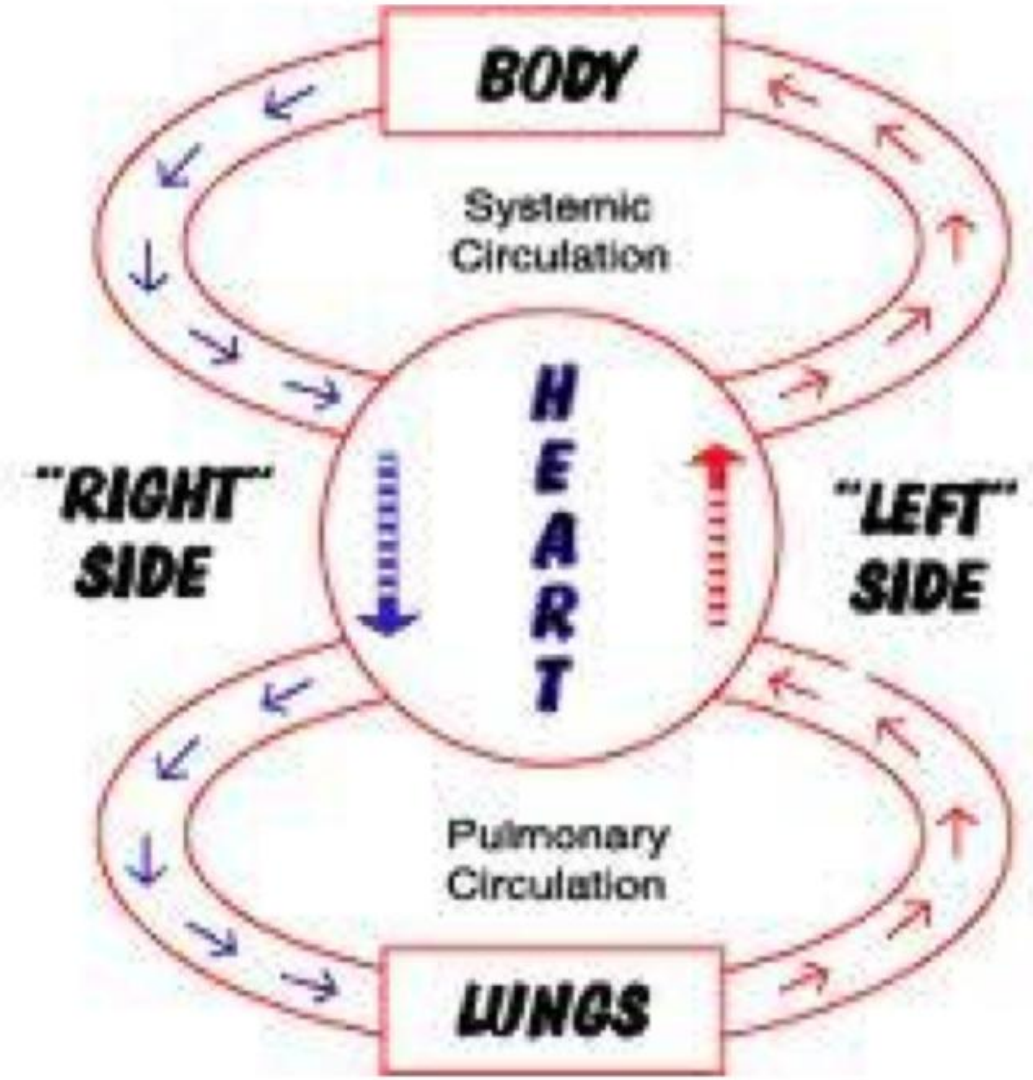
Responses	Short-term effects	Long-term effects
Salt & water retention	Increase preload	Pulmonary congestion Systemic congestion
Vasoconstriction	Maintain BP for perfusion of vital organs	Exacerbate pump dysfunction by increasing afterload Increase cardiac energy expenditure
Sympathetic stimulation	Increase heart rate and ejection	Increase energy expenditure, Risk of dysrrhythmia, Sudden death

Summary & effect of ACE inhibitors

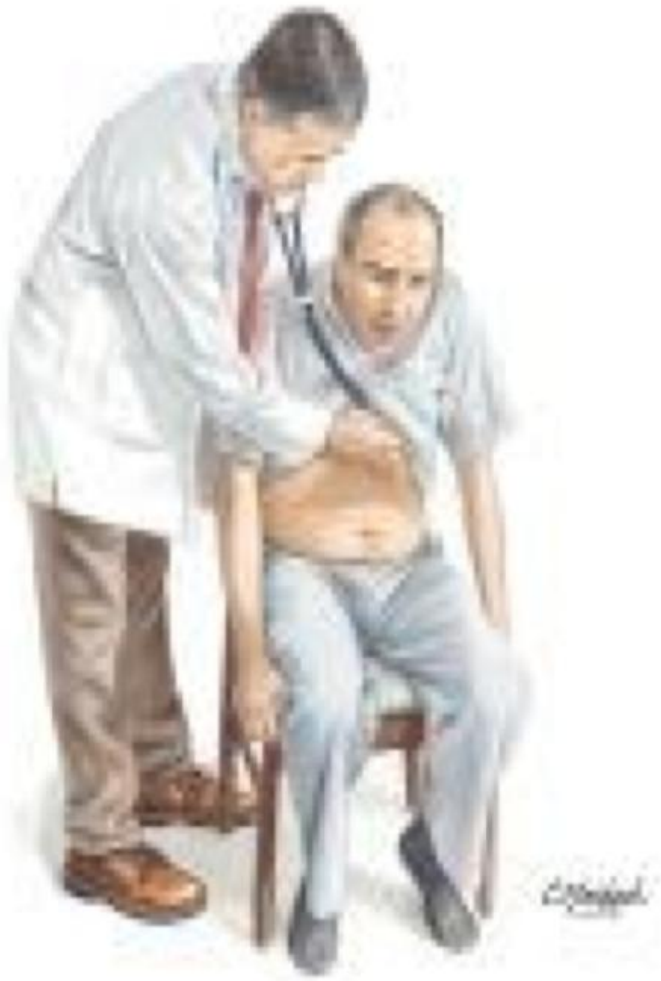


Left-sided failure

- **Common respiratory signs :**
- **Signs & symptoms are due to pulmonary congestion and low CO**
- **Tachypnea** (increased *rate* of breathing) and increased *work* of breathing (non-specific signs of respiratory distress).
- **Rales or crackles**, heard initially in the lung bases, and when severe, throughout the lung fields suggest the development of **pulmonary edema** (fluid in the alveoli).
- **Cyanosis** which suggests severe hypoxemia, is a late sign of extremely severe pulmonary edema.


BODY
Systemic Circulation
"RIGHT" SIDE
HEART
"LEFT" SIDE
Pulmonary Circulation
LUNGS

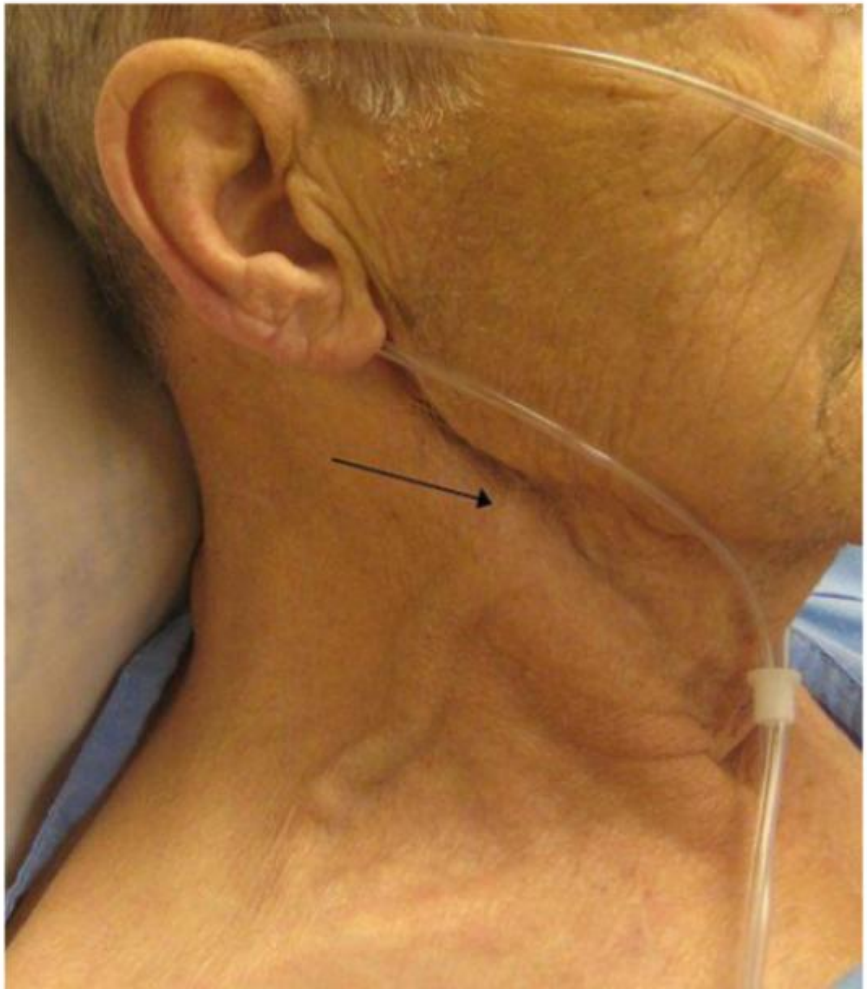
Left-sided HF



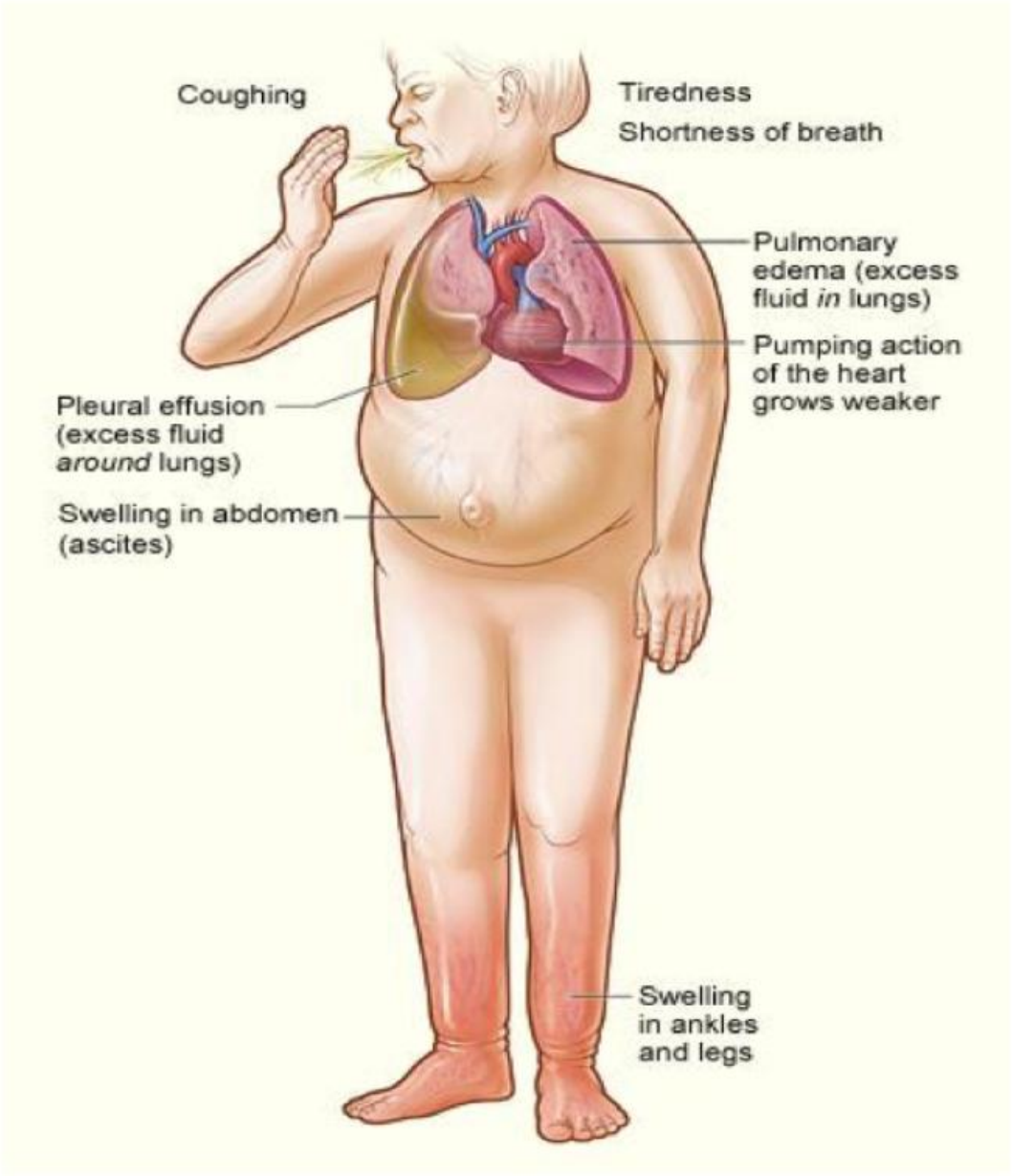
Right-sided failure

- Pitting peripheral edema
- Ascites
- Hepatomegaly
- Jugular venous pressure is frequently assessed as a marker of fluid status, which can be accentuated by the hepatojugular reflux. If the right ventricular pressure is increased, a parasternal heave may be present, signifying the compensatory increase in .contraction strength

Elevated JVP, in a patient with congestive HF



HF


Coughing
Tiredness
Shortness of breath
Pulmonary edema (excess fluid *in* lungs)
Pumping action of the heart grows weaker
Pleural effusion (excess fluid *around* lungs)
Swelling in abdomen (ascites)
Swelling in ankles and legs

Left vs Right HF

Signs/Symptoms	Left-Sided Heart Failure	Right-Sided Heart Failure
Pitting Edema (Legs, Hands)	Mild to moderate.	Moderate to severe
Fluid Retention	Pulmonary edema (fluid in lungs) and pleural effusion (fluid around lungs).	Abdomen (ascites).
Organ Enlargement	Heart.	Liver. Mild jaundice may be present.
Neck Veins	Mild to moderate raised jugular venous pressure (JVP).	Severe jugular venous 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 appetite. Bloating. Constipation. Symptoms are significantly more prominent than LVF

Treatment

The control of congestive heart failure symptoms, can be divided into three categories:

1- reduction of cardiac workload, including both preload and afterload;

2- control of excessive retention of salt and water; and

3- enhancement of myocardial contractility.

THE END

If there are any problems or suggestions
Feel free to contact:

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THANK YOU

Actions speak louder than Words