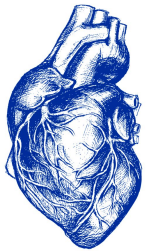
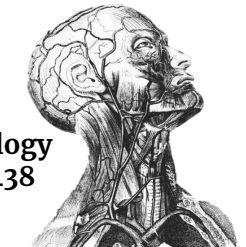
جامعة الملك سعود
King Saud University

Lecture 7-8
Cardiac output
&
Venous Return

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

Editing file





Physiology
MED438

Objectives:

- Define Cardiac Output and its normal values.
- Define stroke volume, End- systolic volume, and end- diastolic volume.
- Identify factors affecting and determining Cardiac Output.
- Role of stroke volume and heart rate regulation on Cardiac Output regulation.
- Understand the role of venous return on Cardiac Output.
- Understand factors affecting the EDV (venous return) and the end- systolic volume (ESV).

Definitions

01

End-Diastolic Volume (EDV):

Volume of blood in the ventricles at the end of diastole (before ejection).

- Volume = 110-130 ml

02

Stroke Volume (SV):

Amount of blood pumped/ejected from ventricles (out of ventricles) per beat.

- Volume = 70 - 80 ml/beat
- EDV - ESV = SV

03

End-Systolic Volume (ESV):

Amount of blood left/remained in ventricles at the end of systole (after ejection).

- Volume = 40-60 ml
- Increase in SV → decrease ESV

04

Ejection fraction (EF):[1]

The percentage (Fraction) of ventricular EDV which is ejected with each stroke (beat), it's around 60-65%.

- It's a good index of ventricular function.
- EF = SV (EDV-ESV) / EDV X 100

1. Ejection fraction is important in emergencies, for example, when a patient is having an operation, the value must be checked before starting the operation, if the value is (60-65%) then he's fit for the operation, if not (e.g. 50%) then the doctor will make sure the operation is done under general anesthesia ONLY, because anesthesia decrease contractility therefore decrease the ejection fraction.
If the fraction is even 40% or less, then he's on heart failure, no operation should be done unless it's emergency.

Definitions

05

Cardiac Output (CO):

It's the volume of blood pumped by the ventricle per minute. (while SV pear beat)

- In adults at rest = 4-6 L/min
- In normal condition the blood volume will circulates through the body once each minute.[1]
- It is a function of: Heart rate (HR) & Stroke volume.
- CO (5 L/min) = SV (70 ml/beat) X HR (70-75 beat/min)

06

Cardiac Index (CI):

It's Cardiac Output per Square Meter of Body Surface Area.

- It's relating heart performance to size of individual, Since CO vary with Size of individual, Age & Gender.
- CI = CO/m2

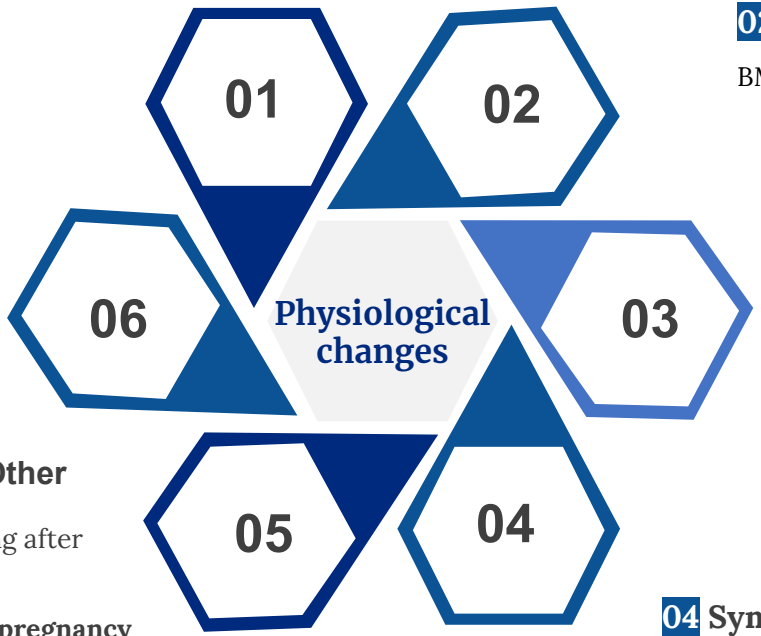
07

Cardiac Reserve:

- It's the difference between resting cardiac output & maximum volume of blood the heart capable of pumping per minute.
- **During exercise**, the cardiac output can increase up to 20-25 L/min (2-4 times), And up to 35-40 L/min (7 times) in **well trained athletes**.[2]

1. ↑Heart rate → ↓stroke volume, because there is less time for filling (it happen in case of no adaptation).
2. For example, my cardiac output was 4 at rest, if I ran, the maximum performance of the heart can reach 9 during exercise, this difference is called the Cardiac Reserve. And It's different for each individual.

Factors affecting CO



01 Sex & Age

Adults have more volume, hence more CO. Females have smaller bodies, meaning less CO(4.9L) than males(5.6L).

02 Height & weight

BMI affects CO

03 Metabolism

After a meal, CO is increased by 30% to enhance blood flow to the intestines

Metabolic demand Increases CO.

04 Sympathetic activity

sympathetic activity during anxiety and exercise enhances the CO up to 50% - 100%

E.g. ↑ sympathetic activity → ↑ force of ventricular contraction → ↑ SV → ↑ CO.

05 Other

- **Body position** affects CO. I.e, standing after lying down decreases it by 20-30%
- CO increases during later months of **pregnancy** due to ↑uterine blood flow.
- Increase in **body temperature** shows an increase in CO At environmental T > 30 °C, the CO is ↑due to ↑skin blood flow, Also at ↓environmental T, CO is ↑due to shivering that ↑blood flow to the muscles.

06 Hyperthyroidism

Due to ↑ peripheral O2 needs, ↑ cardiac contractility & ↑ HR.

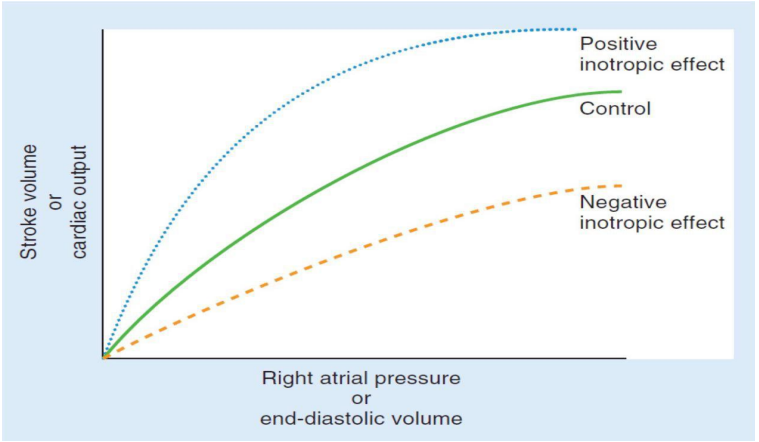
Pathological Changes

1. **Low Venous Return**
   (Haemorrhage)
2. **Hyperthyroidism** (High CO)
   the increase in the CO is due to the high metabolic rate → vasodilatation → CO increase to 50%+ of control.
3. **AV Fistulas** (High CO)
4. **Fever** (High CO)
5. **Anxiety** (High CO)
6. **Anemia** (High CO)
7. **Tachyarrythmias**
   (atrial fibrillation & ventricular tachycardia)
8. **Reduced Contractility**
   (Heart failure)

Increase	Moderate Exercise	Severe Exercise
CO	to 240% (12L) to 2-4 folds of resting (up to 20-25 L/min)	to 500% - 700% (25-35 L) to 7 times of resting (up to 35-40 L/min)
HR	to 200% of resting (140 bts/min)	to 300% of resting (200 bts/min)
SV	to 120% (85ml)	to 175% (125ml)

Cardiac output regulation

- The Frank-Starling law states that the more the heart stretches, the stronger its contractility will be.
- When venous return increases, the stroke volume increases with it due to the change in EDV. this consequently increases CO.
- Also, as venous return increases, the heart will stretch more than usual, causing an increase in contractility. **This shows how EDV can affect CO and SV.**



Determinants of Cardiac Output:

CO = SV X HR

CO = EDV - ESV (Myocardial contraction (Inotropic state)) **X HR** (Chronotropic state)

- **Stroke Volume** (ventricular myocardium) **is regulated by:**

1. **EDV:** preload/Venous Return
2. **Contractility:** Starling's law & Sympathetic innervation
3. **Total peripheral resistance:** afterload (inversely proportional)

- **Heart Rate** (SA node) **is regulated by:**

1. Autonomic nervous system
2. Hormones and Drugs

Parasympathetic nerves have no effects on the stroke volume because they don't innervate the ventricles.

Heart Rate

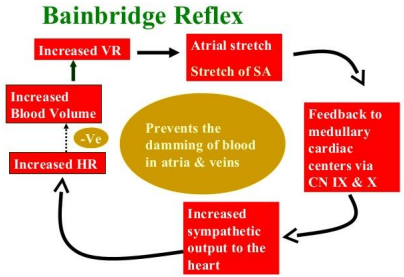
- Normal heart rate (60-100 beats/min), > 100 beats/min is **Tachycardia**, < 60 beats/min is **Bradycardia**. .
- Increased HR up to 180 bpm will make the ventricular filling be adequate and CO will increase, but at very high HR (>180 bpm), filling may be compromised to a degree that CO falls.
- The HR has an influence on cardiac contractility as well (Frequency-Force Relation.)

Heart Rate (HR) is regulated by

1- **Hormones and Drugs:** Epinephrine & thyroxine increase HR + Increased calcium level in blood causes prolonged contraction + Reduced calcium level in blood decrease HR.

2- **Physical factors:** Age (Resting HR is faster in fetus) + Gender (HR is faster in females) + Temperature (Heat increases HR as occurs in high fever, Cold has the opposite effect)
+ Exercise (Increases HR)

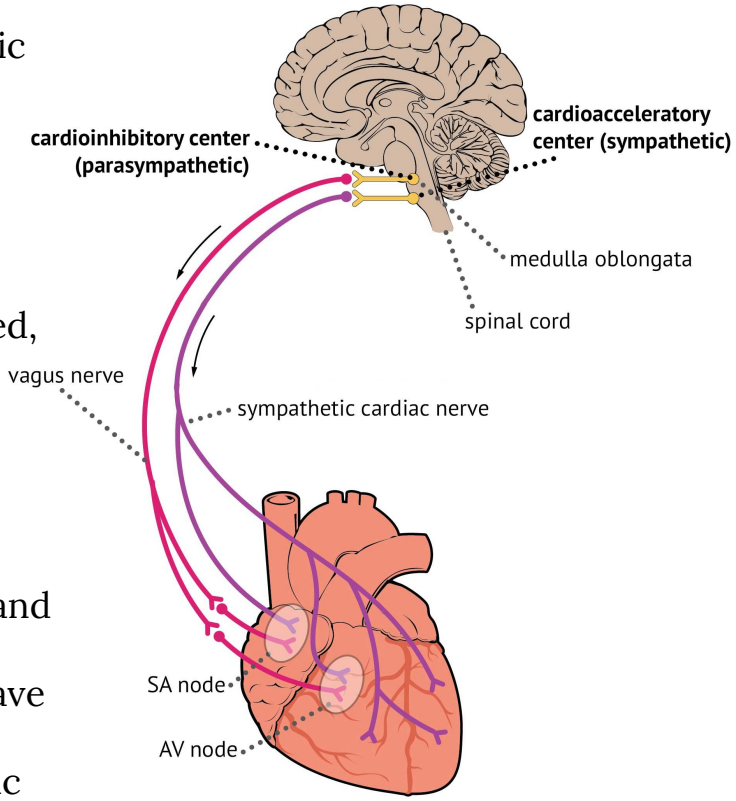
3- **Blood volume:** Atrial reflex (Bainbridge reflex).
This reflex adjusts Heart rate in response to venous rate.
Increased blood volume is detected by stretch receptors located in right atrium, which will triggers increase in HR through increased sympathetic activity.



Regulation of heart rate

4- **Autonomic nervous system** through cardiac control centers in medulla oblongata in the brain stem:

- Cardiac-accelerator Center / vasomotor center → Sympathetic nerve fibers →increases HR & contractility.
- Cardiac-inhibitor Center →Parasympathetic nerve fibers → (vagus nerve) slow HR.



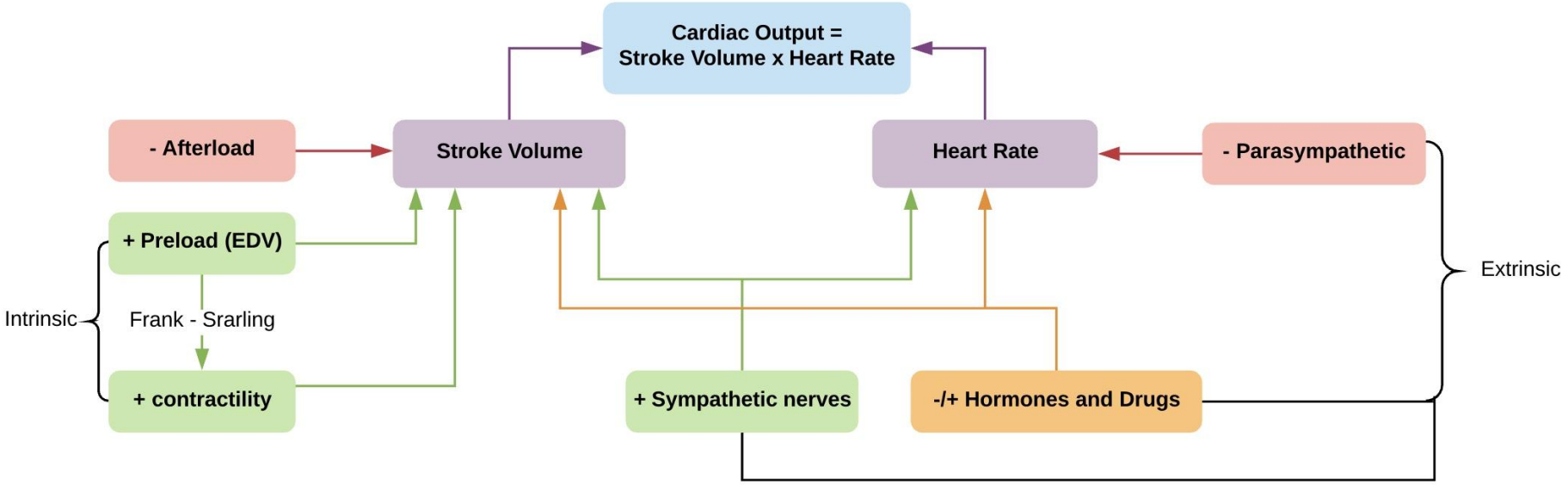
Sympathetic effect:

- Sympathetic nerves innervate the whole heart, when stimulated, they increase heart rate. (and contractility).
- Sympathetic nerves release noradrenaline (adrenaline), which stimulates heart β1 - receptors.

Parasympathetic effect:

- Parasympathetic nerves innervate the SA, AV nodes, the atria and Purkinje system, and do not innervate most of the ventricular myocardium. When stimulated, they slow the heart rate but have little inotropic action.
- Parasympathetic nerves release ACh that stimulates muscarinic (M2) receptors.

Determinants of Cardiac Output



Factors affecting contractility

- End-Diastolic Volume (Starling's law of the heart)
- Cardiac innervation (SNS ↑, PNS ↓)
- Oxygen supply (Hypoxia →↓ contractility)
- Calcium ↑ & potassium ions ↓ contractility.
- Physical factors (Warming →↑ , Cooling →↓)
- Mechanical factors (syncytium, cannot be tetanized)
- Hormonal & chemical factors. (Drugs)

Positive inotropic:	Negative inotropic:
• Adrenaline • Noradrenaline • Alkalosis • Digitalis • Ca2+ • Caffeine	• Acetylcholine • Acidosis • Ether • Chloroform • Some bacterial toxins (e.g. diphtheria toxins) • K+

Effect of Ions and Temperature

Male slides

Ions:

- Increased K in the extracellular fluid causes the heart to become dilated and flaccid slowing down the heart rate. ↑ K decreases the resting membrane potential in the cardiac muscle fibers, the intensity of the action potential also decreases, which makes contraction of the heart progressively weaker
- Increased Ca causes spastic contraction. This is caused by a direct effect of calcium ions to initiate the cardiac contractile process.
- Decreased Ca cause flaccidity.

Temperature:

- An increase in temperature can result in an elevated heart rate.
- A decrease in temperature has the opposite effect, dropping the heart rate severely in some cases.

Frequency-force relationship

- **The frequent stimulation of the myocytes can probably be owed to the accumulation of Ca ions within them, which is caused by:**

1. More depolarizations per minute, which leads to more Ca entry.
2. An increase in the intracellular stores of Ca can be noted due to a higher magnitude in the Ca current.

- Both effects positively influence the availability of Ca in the sarcoplasmic reticulum.

Measurement of cardiac output

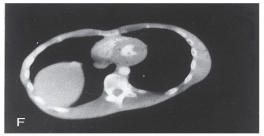
Can measure changes in ventricular diameter at several depths to estimate changes in ventricular volume.

Fick's principle states that total oxygen consumption= delivered oxygen - returned oxygen.

$$\frac{\text{Total O2 consumption}}{\text{Arterial O2- mixed venous O2}} = CO$$

Records real-time changes in ventricular dimensions during systole and diastole.It computes stroke volume, which when multiplied by heart rate, gives the cardiac output.

1



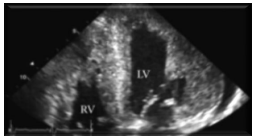
Ultra fast computer tomography

2

Fick's Principle

2-D Echocardiography

3



Definitions:

08

Mean Circulatory Filling Pressure

- the value for right atrial pressure at which venous return is zero.
- The pressure that exists in the circulatory system when there is no blood motion

10

Preload: (Venous Return)[1]

It is the amount of blood that returns to the heart from veins.

- Preload is the load on the muscle in the relaxed state.
- Increase in venous return → increase EDV

09

Veins

Capacitance vessels that hold 70% of blood. Their thin, stretchy walls lets them hold more blood without a major increase in pressure, which is usually 0–10 mmHg.

11

Venous capacity

the volume of blood that the veins can accommodate.

12

Afterload (Resistance)

It is the resistance against which the ventricles contract.

- afterload increases →SV decreases.

1. Preload = venous return, but venous return doesn't equal EDV, there's different between them. For example, if we say that in College of Medicine, the total number of students was around 500 students (venous return), and there was a hall that can take up only 200 students (ventricle), if we told the students to come into the hall, all the students will try to come in but only 200 students will be able to make it (which is EDV). So basically EDV is the blood which got into the ventricle!

Venous Return

- Normally **venous return** must equal **cardiac output** when averaged over time because the cardiovascular system is essentially a closed loop, Otherwise blood would accumulate in either the systemic or pulmonary circulations.
- **Venous return** is determined by the difference in pressure between the venous pressure nearest to the tissues (mean systemic filling pressure; mean circulatory pressure; MCP) and the venous pressure nearest to the heart (CVP).

Central venous pressure (CVP)

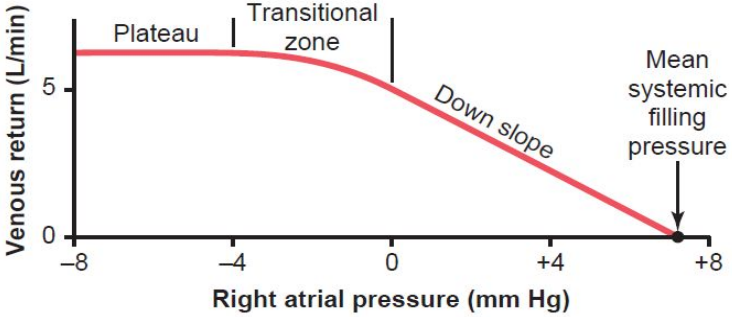
- It's the pressure in the right atrium and the big veins of the thorax. (right atrial pressure (RAP) = jugular venous pressure).
- Measured with a catheter inserted in SVC.
- The normal range of the CVP = 0 - 4 mmHg.
- It's the force responsible for cardiac filling.
- Used clinically to assess hypovolemia and during IV transfusion to avoid volume overloading.
- Raised in right-sided heart failure.

Pressure curves

- ## Venous Return Curve

This curve represents the relationship between venous return and RAP. When RAP drops below 0, increase in VR ceases, and that is due to the collapse of veins entering the chest.

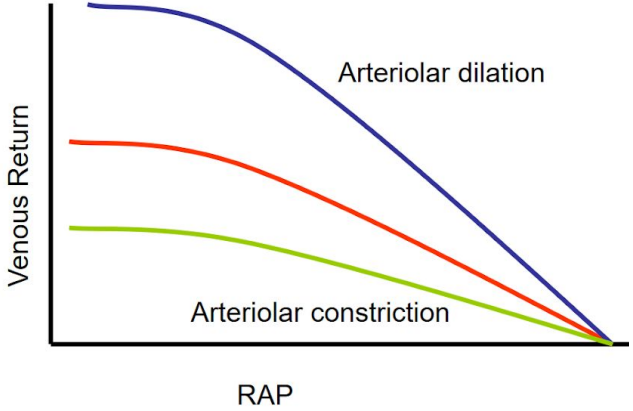
However, if VR hits 0 (which happens when the heart is shocked and all circulation stops and pressure throughout the body is equal) , the RAP at that point (Usually 7 mmHg) is called Mean Systemic Filling pressure.



- ## Vascular Function Curve Male slides

This curve shows the effect of arterial dilation and constriction on venous return.

- As the arterioles (resistance vessels) dilate, venous return increases because there is less pressure restricting the blood from going to the venous side, and vice versa.



Factors affecting preload

- **Total blood volume**: At constant venous capacity as the blood volume increase → the Mean Circulatory Pressure increase → venous return increase. And the same will happen when the blood volume decrease.
- **Pressure gradient**: The higher the difference, the more venous return there is. Basically, if the pressure gradient is low, not a lot of blood will be pushed to the right atrium, dropping venous return.
- **Gravity**: When a person initially stands, right atrial pressure & ventricular EDV falls, which decreases stroke volume. Consequently, arterial pressure drops, causing a disturbance in the systemic blood flow. This affects overall CO.
- **Venoconstriction:** By sympathetic stimulation which will increase the venous return.
- **The presence of valves:** Permit blood to move forward towards the heart & prevent it from moving back toward the tissues.

Factors affecting preload

- **The skeletal muscle pump:** Rhythmical contraction of limb muscles (as occurs during walking, running or swimming) → increase the venous return by the muscle pump mechanism that squeeze the blood vessels between muscle fibers.
- **Respiratory activity:** Inspiration will increase venous return because of a decrease in right atrial pressure (RAP). In Valsalva maneuver (forceful expiration against a closed glottis), intrapleural pressure become positive which is transmitted to the large veins in the chest which will decreases the venous return
- **Right atrial pressure:** RAP and preload are inversely proportional. When RAP drops below 0, venous return enters a plateau phase. And when venous return hits 0, the pressure at that point (Usually 7mmHg) is known as mean systemic filling pressure. MSFP is affected by the volume of blood and venous capacity.
- **Contractility of the heart.**

EDV and ESV

Factors Affecting End - diastolic Volume

EDV is increased with:

1-Increased total blood volume.

2-Increased venous return.

3-Increased venous tone.

4-Increased skeletal muscle pump (exercise).

5-Increased negative intrathoracic pressure.

6-Stronger atrial contraction.

EDV is decreased with:

1-Standing.

2-Decreased venous return.

3-Increased intrapericardial pressure.

4-Decreased ventricular compliance.

Factors Affecting End - systolic Volume

- **Cardiac contractility:**

Increase in contractility →increase in stroke Volume →decrease in ESV.

Decrease in contractility →decrease in stroke Volume →increase in ESV.

- **Afterload: Vasoconstriction:**

Increase in resistance →decrease in stroke volume →increase in ESV

Decrease in resistance →increase in stroke volume →decrease in ESV

Afterload

It represents the load against which the heart must eject blood.

It is affected by any factor that acts on arterial blood flow, like:

1. **arterial blood pressure:**

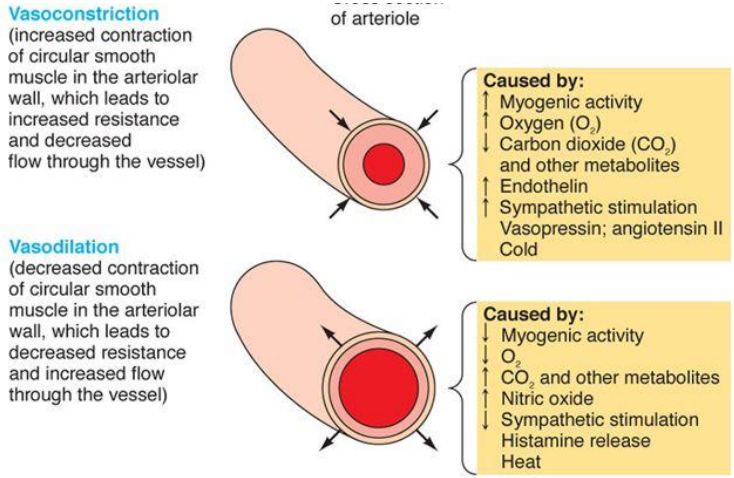
   A change in blood pressure means a change in afterload, keeping in mind that they are directly proportional. So, in cases of hypertension, an increase in afterload would be noted.

2. **Total peripheral resistance:**

   Vasodilation decreases resistance, which will in turn decrease afterload, and vice versa for vasoconstriction.

3. **Valves:**

   The condition of the aortic valve could have an effect. In cases of aortic stenosis, an increase in afterload can be seen



Angiotensin II is a STRONG vasopressin

Ventricle pressure – volume loop

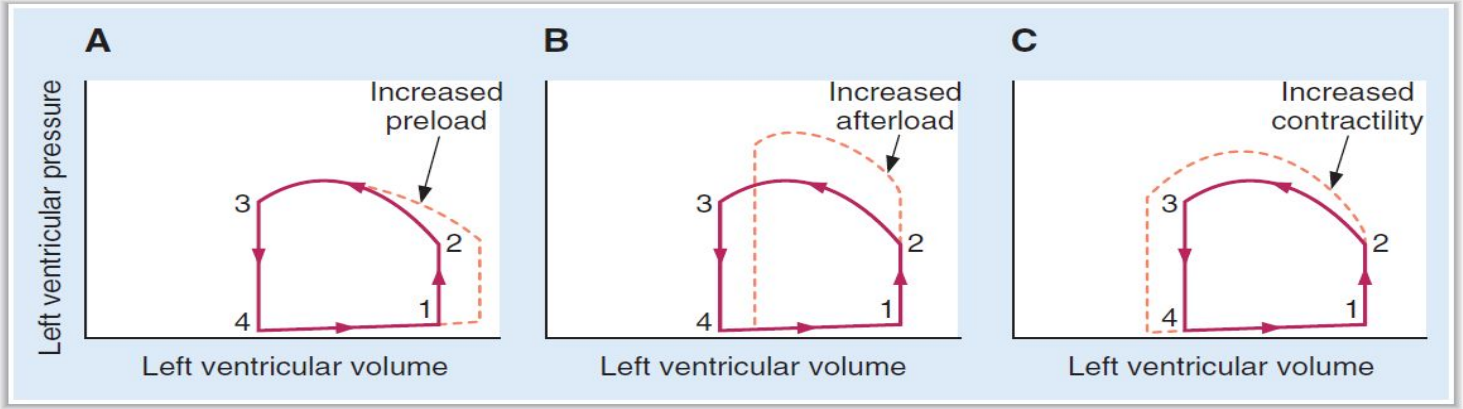


FIGURE 3-10 Effects of changes in **(A)** preload, **(B)** afterload, and **(C)** contractility on the ventricular pressure–volume loop.

A.Increased preload: ↑ venous return → increase in SV based on the Frank–Starling relationship, reflected in ↑ width of the PV loop.

B.Increased afterload: due to an increase in aortic pressure → decrease in stroke volume, reflected in ↓ width & ↑ height of the PV loop.

C.Increased contractility: ↑ width & ↑ height of the PV loop.

Quiz

1. Venous return is increased when:

A. RAP increases
B. The pressure gradient increases
C. Valsalva maneuver
D. The nervous circulatory reflexes are absent

2. What is the cause of the plateau when RAP falls below zero:

A. Closing of the aortic valve
B. Collapse of veins entering the chest
C. Stopping of the heart
D. Closing of the pulmonary valve

3. Which of the following is known as resistance vessels?

A. Veins
B. Capillaries
C. venules
D. Arterioles

4. A patient with thyrotoxicosis came into the clinic for a check up, and the nurse was asked to take his vitals. Which of the following findings will most likely be noticeable?

A. Increased heart rate
B. Decreased heart rate
C. No noticeable changes
D. High WBC level

5. Calculate the cardiac output using the following:
ESV= 50 ml, EDV=130 ml, HR= 70 BPM

A. 3.5 L/min
B. 5 L/min
C. 5.6 L/min
D. 1 L/min

SAQ:

1- explain what happens to CO when heart rate is severely increased.

Filling is compromised and CO falls

3- Explain the relationship between arteriolar dilation/constriction and venous return.

Answers: B - B - D - A - C

Leaders

Sedra Elsirawani Abdulrahman Alhawas

Members

- Lama AlZamil
- Arwa AlEmam
- Noura AlTurki
- Ghada AlSadhan
- Nouf AlShammari
- Nouf AlHumaidhi
- Taibah AlZaid
- **Ajeed AlRashoud**
- Reem AlGarni
- Raghad AlKhashan
- Leen AlMazroa
- Nouran Arnous
- Maha AlNahdi
- Badr Almuhanna
- Abdulrahman Almezaini
- Omar Aldosari
- Omar Alghadir
- **Ibrahim Alshaqrawi**
- Abdullah Aldawood
- Abdullah Shadid
- Meshari Alzeer
- Mohammed Alhamad
- Abdullah Alassaf
- Khalid Alkhani
- Amjad Albaroudi
- Mohammed Alhuqbani