THE CARDIAC OUTPUT

- **The cardiac output (COP) is the volume of blood pumped by each ventricle per minute**; Normally, it is about 5 litres/minute during rest, and is equal for both ventricles.
- **Stroke volume (SV): Is the volume of blood pumped by each ventricle per beat. = 70 ml**.

Variations in the cardiac output:

- The COP is subject to **both physiological and pathological variations.**

I. Physiological variations:

1. **Posture:** The COP, is about 25% less in the standing than in the recumbent position **due to the effect of gravity which decreases the VR.**
2. **Digestion:** Increases the COP by **about 30% due to increased blood flow to gastrointestinal tract (GIT) during digestion**.
3. **Temperature:** Exposure to warm weather increases the COP by **about 30% due to vasodilatation of peripheral arterioles, leading to increased venous return**. Cold weather has the opposite effect.

4. **Excitement: increases the COP 2 times due to sympathetic stimulation.**

5. **Pregnancy: increases the COP 2 times due to V.D of blood vessels of the uterus and placenta.**

6. **Muscular exercise:** increases the COP 5-7 times.

II- Pathological variations:

Pathological increase in COP:

1. Anaemias: cause VD of arterioles → ↑ VR →↑ COP.

2. Increased metabolic rate: as in fever and hyperthyroidism, **due to peripheral vasodilatation → ↑ VR → ↑ COP.**

3. Aortic incompetence (regurgitation): increases the EDV(end diastolic volume) → increase of the COP.

- **Pathological decrease in COP:**

1. **Pathological increase or decrease in heart rate**, e.g. in paroxysmal tachycardia or in complete heart block respectively.
2. **Decreased coronary blood flow** as in myocardial ischemia or infarction.
3. **Decreased circulating blood volume as in shock**, whether hemorrhagic, traumatic or histamine shock.
4. Heart failure, due **to decreased strength of ventricular contraction**.
5. Valvular lesion as in mitral stenosis, regurgitation and aortic stenosis.

Factors Affecting the Cardiac Output

1- Venous return (preload)

- The COP is determined mainly by **the volume of blood returned by the veins from the tissues** i.e. by the metabolic activity of the tissues **particularly the voluntary muscles. So, ↑ VR → ↑ COP**

Factors that influence the venous return:

1- The pumping action of the heart (most important)

2- Pressure gradient:

The mean circulatory pressure (MCP):

Is the blood pressure in peripheral venules and veins in the recumbent position and is normally about 7-8 mm Hg.

The right atrial pressure (RAP):

- It is the pressure in the right atrium. **In the recumbent position it is 2 mm Hg.**
- In the recumbent position, (MCP) **is 7-8 mm Hg and (RAP) is 2 mm Hg, this leads to a venous return of about 5 liters/minute**

3- Respiratory movements:

- Venous return increases with inspiration and decreases with expiration.

a- During inspiration:

- **Normal inspiration makes the intrathoracic pressure more negative and the intraabdominal pressure more positive** → blood is sucked from extra to intrathoracic veins →↑VR.

b- During expiration:

- **During normal expiration the negativity inside the thorax is decreased**, and so the VR is less than during expiration

4- Gravity:

- In the recumbent position, **gravity has no effect on VR.**
- In the erect position**, gravity helps the VR from parts above the level of the heart**, but reduces it from parts below the level of the heart. However, **this effect is counteracted by some factors as:**

a- The suction power of the –ve intrathoracic pressure.

b- The pumping action of the muscle tone and muscle contraction.

c- The presence of valves in the veins that divide the large column of blood to small segments.

- **Therefore, all these factors help the flow of blood towards the heart and prevent stagnation or pooling of blood in the veins of lower limbs.**

5- Vascular system:

a- Diameter of arterioles:

- Normally, the tone of the arterioles leads to partial VC.
- **VD of arterioles → ↑VR and vice versa.**

b- Diameter of capillaries:

- Normally about 10% only of the capillaries are opened and the other 90% are collapsed during rest.
- **If all the capillaries of the body are widely dilated, as by injection of histamine,** the whole blood volume will be retained in them → no VR, no COP, **→shock and death i.e. "histamine shock". Thus VD of capillaries → ↓VR → ↓ COP.**

c- Diameter of veins:

- Venous tone prevents full distension of veins with blood. So, it maintains VR.
- **VD of veins → stagnation blood into veins →↓ VR and COP and vice versa.**

d- Arterial pulsation:

- In most situations the veins run parallel to the arteries. The **arterial pulsations are mechanically conducted to the venous wall** → drive of blood towards the heart → increasing the VR and the COP.

6- Skeletal muscle contraction (muscular pump):

- ***During muscular contraction,*** **the muscle fibers compress the blood vessels lying in between them,** squeezing the blood into veins towards the heart, so increasing the VR and COP.
- ***During muscular relaxation,*** **the blood does not regurgitate back to the muscles because the veins possess very efficient valves which direct the blood stream towards the thoracic veins.**

7- Blood volume:

- Increased blood volume (**e.g. during muscular exercise due to contraction of blood reservoirs)** →↑VR and COP.
- Decreased blood volume **→↓VR and COP e.g during haemorhage.**

II- Arterial blood pressure (after load)

Changes in ABP have no effect on the COP provided the VR is kept constant.

III- Heart rate (HR)

The effect of changes of heart rate on COP is influenced by **both the amount of VR and the extent of changes in heart rate.**

1- Changes of heart rate with constant VR:

a) Physiological or moderate changes of HR:

- If the venous return is kept constant, moderate changes in HR has no effect on COP, it affects only the SV.

b) Pathological or excessive changes in HR:

If the venous return is kept constant marked acceleration or slowing of the heart decreases the COP.

2- Changes of HR with change of VR:

In muscular exercise:

- When the VR is increased, **the acceleration of the heart becomes of fundamental importance in increasing the COP.**

IV- Strength of ventricular contraction

- **The SV and consequently the COP are directly proportional to contractility, i.e. when the ventricles contract more strongly, they pump more blood**.
- **Starling's law of the heart** represents an important mechanism of cardiac reserve power.
- **In normal heart within limits, when the VR increases as in muscular exercise**, → the ventricle dilates i.e. increases the initial length of the cardiac muscle fiber during diastole → **↑force of ventricular contraction → ↑SV and COP i.e. the heart perform more work.**

ARTERIAL BLOOD PRESSURE

Definition: the lateral force exerted by the moving column of blood on the lateral wall of arteries.

Systolic blood pressure:

- **This is maximum pressure created inside the arteries during ventricular systole** due to rapid ejection of blood into the aorta.

It normally ranges between 90-140 mmHg, at rest

Diastolic blood pressure: **minimum pressure created inside the arteries during ventricular relaxation**

Mean systemic ABP:

This is the average pressure in the systemic arteries throughout the cardiac cycle

Factors that determine and maintain the ABP

1. Cardiac output (COP):

- ABP is directly proportional to COP provided all other factors affecting ABP remain constant.
- **Because, COP = SV x HR, so changes in any of its components (SV & HR), affect the ABP in a particular way:**

a. Stroke volume:

Changes in stroke volume, **with HR kept constant, affect the systolic more than the diastolic pressure**

b. Heart rate:

- Changes in the HR, **with constant SV affect the diastolic more than the systolic BP,**

2. Peripheral resistance:

- **It is the resistance which the blood meats during its passage through the peripheral arterioles.**
- As mentioned before: ABP = COP x PR ➔ So the ABP is directly proportional to PR.

Resistance at different parts of the circulatory system is calculated by measuring BP before and after crossing this area:

	Arteries	Arterioles	Capillaries	veins
Pressure difference (mmHg)	100 ➔ 85	85 ➔ 35	35 ➔ 10	10 ➔ 0
Resistance (% of total)	15%	50%	25%	10%

Changes of PR affect directly the diastolic more than the systolic BP.

- **Factors that determine the PR:**
- They are detected from **Poiseuille's equation** which states that:

$$PR = \frac{8\eta L}{\pi r^4}$$

- **Where R is the resistance, η is the viscosity, L is the length of the vessel and r is the radius.**

a. The diameter of arterioles:

- **The PR inversely proportional to the arteriolar diameter**.
- The diameter of the arterioles is **under the control of VMC, and other chemical factors**

i- **Arteriolar VC →↑ diastolic more than the systolic BP**

ii- **arteriolar VD produces opposite effects.**

b. Viscosity of blood:

- **The PR is directly proportional to the degree of blood viscosity (η).**
- **The whole blood is about 3 times more viscous than water or saline.**

The viscosity of blood is due to:

- **Volume of suspended corpuscles (RBCs, WBCs & platelets).**
- **Amount of plasma proteins.**

c. Length of the blood vessels:

- The PR is directly proportional to the length of the blood vessels (L).
- **The main factor that determines the PR is the diameter of arterioles.**

How to calculate the peripheral resistance:

- **As ABP = COP x PR So PR =** $\frac{\text{ABP}}{\text{COP}}$

- ***During rest:***

i-PR in the systemic circulation:

$$\textbf{PR} = \frac{\text{ABP}}{\text{COP}} = \frac{90}{5} = \textbf{18 mmHg/liter/minute.}$$

i.e. **18 mmHg of pressure energy is needed to drive one liter of blood/minute through the peripheral resistance offered by the systemic circulation.**

During exercise in the systemic circulation:

$$PR = \frac{ABP}{COP} = \frac{15}{3} = 6 \text{ mmHg / L/ min. (i.e. 1/3 the value at rest).}$$

- ***In a hypertensive patient:***

$$\mathbf{PR} = \frac{ABP}{COP} \quad \frac{150}{25}$$

3. Elasticity of arteries:

- **The elasticity of the aorta and its large branches buffer excessive changes in the ABP during systole and diastole.**

4. The total blood volume in relation to the capacity of the circulatory system:

a. Changes in blood volume:

i-Moderate changes in blood volume do not affect ABP, due to compensatory changes in the capacity of the circulatory system

ii-Excessive changes in blood volume → variations in ABP, inspite of the compensatory mechanisms.

Hemorrhage

- Hemorrhage means loss of blood from the cardiovascular system.
- **It may be external (revealed) e.g. bleeding from open wound or internal (concealed) e.g. intra-abdominal, intracranial....etc. Each of them may be further classified:**

1. According to the amount of blood loss:

- Loss of about 20% of the total blood volume could be compensated by various physiological mechanisms of the body itself.
- **If the loss is more than 20%, it must be aided by transfusion of blood or any substitute**, as early as possible, otherwise irreversible shock will occur.

2. According to the rate of blood loss:

- **Sudden loss of a large amount of blood is more dangerous than small repeated bleeding over a long period of time** e.g. from piles, parasitic infestation.

I. Immediate (compensatory) effects of hemorrhage

1. Cardiovascular changes:

a. Cardiac acceleration:

Importance: increase DBP ➔ **increase mean systemic ABP which determine BF through cerebral and coronary blood vessels.**

b. Increased vasoconstrictor discharges:

- It causes generalized vasoconstriction except in the cerebral and coronary vessels.
- **This helps the venous return and maintains the cardiac filling, COP and ABP**.

2. Contraction of blood reservoirs:

- **The most important blood reservoir is the spleen** which contracts driving its content of blood into the general circulation. .

3. Respiratory changes:

a. **There is VC of the pulmonary vessels which helps in reduction of the circulatory capacity and maintenance of blood volume**.

b. There is acceleration of respiration → more oxygenation of blood, and also helps in increasing the venous return.

4. Blood changes:

a. Coagulation of blood → stops hemorrhage.

b. **Vasoconstriction of the injured vessels → helps the blood clot to seal the vessels.**

- c. Formation of erythropoietic factor →↑ RBCs by the bone marrow.
- **5- Kidney changes**

  Decreased formation of urine, so reduces the amount of water loss.

- Release of erythropoietin→ stimulation bone marrow to produce RBCs.

6- Hormonal changes:

a- Increased Catecholamines (adrenaline and noradrenaline)

b- ACTH and glucocorticoids:

- **Haemorrhage stimulates the hypothalamus which activates the anterior pituitary gland to secrete ACTH.** → release glucocorticoids from the adrenal cortex.
- This hormone plays an important role in restoring homeostasis after hemorrhage.

c- Aldosterone:

- **Causes Na and water retention.**

d- ADH:

- Release: from posterior pituitary by ➔

  Effect:

- **Vasoconstriction**
- **Increase water reabsorption from renal tubules.**
- Thus, ABP, increase to normal

f- Erythropoietin: see before.

II. Delayed effects of hemorrhage:

1. Restoration of plasma volume: This is done by:

a. **Withdrawal of fluid from interstitial and intracellular compartments into the blood.**

b. Increased water reabsorption from the distal renal tubules by the effect of aldosterone and antidiuretic hormones.

c. **Intake of external fluid caused by thirst sensation as a result of tissue dehydration.**

2. Replacement of plasma proteins:

a. Rapid replacement from labile proteins in the liver and other tissues

b. Slow mechanism by new synthesis

A diet containing proteins of high biological value accelerates the synthesis.

3. Restoration of RBCs:

a- Rapid restoration of RBCs by splenic contraction due to adrenaline release and sympathetic stimulation.

b- **Slow restoration of RBCs by the bone marrow. This takes 3-5 weeks**

4. Tissue repair:

- To restore **the integrity of the damaged tissues.**

5. Recanalization of the closed blood vessels:

- **By removal of the clots inside the vessels by fibrinolytic system.**

Circulatory shock

Shock means inadequate tissue perfusion or insufficient blood flow to supply tissue requirement and remove waste products.

Types:

1. Hypovolemic shock:

- Hypovolemia means diminished blood volume, and hemorrhage is perhaps the most common cause of hypovolemic shock.
- **Hypovolemic shock is characterized by:**
- Hypotension.
- Rapid weak pulse.
- Pale, cold, sweaty skin.
- Rapid respiration.
- Intense sense of thirst.
- Anxiety and restlessness.

Examples:

1. Hypovolemic shock caused by blood loss

2. Hypovolemic shock caused by plasma loss:

- **Severe loss of plasma even without loss of whole blood can cause typical hypovolemic shock.**
- This occurs in the following conditions:

a. Intestinal obstruction:

- **The resulting distension of the intestine causes fluid to leak from the intestinal capillaries** into the intestinal walls and intestinal lumen.
- Thus, the plasma volume is markedly diminished.

b. Severe burns:

- **Severe loss of plasma through the burned areas so that the plasma volume is markedly reduced.**
- **c. Trauma:**
- Trauma to the body often produces shock, simply due to hemorrhage.

But contusion of the body can also damage the capillaries sufficiently to allow excessive loss of plasma into the tissues.

d. Dehydration:

- Dehdyration means loss of fluid from all fluid compartments of the body. **This can reduce the blood volume and cause hypovolemic shock, e.g.:**
- Excessive sweating.

Severe diarrhea or vomiting. .

3-Low-resistance shock:

- **This type of shock is caused by widespread vasodilatation**
- So the venous return, the cardiac output and the ABP are decreased → shock.
- **The low-resistance shock has the same manifestations as hypovolemic shock except that the skin is warm**.

Types of low-resistance shock:

a. Neurogenic shock:

- **It occurs by strong emotions, severe pain and irritation of the trigger zones. It leads to**:
- Inhibition of VCC leading to VD and decrease ABP.

Inhibition of CAC leading to bradycardia.

b. Anaphylactic shock:

- **This is a severe allergic reaction that sometimes occurs if the person is exposed to an antigen to which he has been sensitized**.
- The allergic reaction **releases large amounts of histamine → massive vasodilatation.**

c. Septic (endotoxin) shock:

- **It is due to** invasion of the blood by bacteria or their toxins (endotoxin).

It is manifested by high fever and marked vasodilatation throughout the body due to excessive release of nitric oxide.

III. Cardiogenic shock:

- This type of shock is caused by **severe depression of the pumping action of the heart as in myocardial infarction→↓ COP and ABP.**

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Haemorrhagic shock:

1- Reversible shock:

It occurs if the amount of blood loss is less than 20% of total blood volume-

2-Irreversible shock:

- **It o2ccurs if the amount of blood loss is more than 30% of total blood volume.**
- **The compensatory mechanisms failed to restore the ABP to its normal level.**

The mechanisms that lead to irreversible shock:

1. Positive feed-back death cycles:

- These are dangerous cycles that eventually lead to circulatory failure and death, which are:

Death cycle: Tissue ischemia → ↑ VD metabolites → VD

↑ ↓

↓ Tissue blood flow ← ↓ ABP ← ↓ COP ← ↓ VR. 2-Cerebral ischemia:

- **Haemorrhage →↓ ABP → cerebral ischemia → depression of vasomotor center & cardiac centers** → V.D & bradycardia →↓ ABP→cerebral ischemia, and so on.

Management of shock:

1. Treatment of the cause of shock.

2. General measures:

   a. Warming the patient, but avoid over-heating as it causes VD that leads to further drop of ABP.

   b. Raising the foot of the bed to help the venous return and improves the cerebral blood flow.

3. Restoration of adequate level of tissue perfusion by transfusion of blood**, plasma or saline as early and rapidly as possible to provide adequate blood flow to the vital organs i.e. brain, heart.**

4. Drugs used in the treatment of shock:

- **Adrenaline and noradrenaline** to produce arterial vasoconstriction and to increase the force of ventricular contraction.
- **Glucocorticoids** **a**s they decrease the permeability of the capillaries.
- **Sedatives** should be given in small doses as they inhibit the vasoconstrictor and cardio stimulator centers.

N.B.: *Alcohol and overheating are very dangerous in shock as they produce peripheral vasodilatation and further drop of ABP.*