University of Al- Ameed - College of Dentistry
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Lectures on Medical Physiology

Lectures 11-13

CARDIOVASCULAR PHYSIOLOGY

Functional anatomy of the heart

The heart has four chambers (**two atria and two ventricles**). *Pulmonary trunk* carries blood from the right ventricle to the lungs. Four *pulmonary veins* return blood from the lungs back to the left atrium (**pulmonary circulation**).

Aorta routes blood from left ventricle to the body. *Superior vena cava* collect blood from regions superior to diaphragm and *inferior vena cava* collect blood from regions inferior to diaphragm. Both pour their blood into the right atrium (**systemic circulation**).

Right and left **coronary arteries** provide blood supply to the heart and **coronary sinus** drains blood from the heart and pours into the right atrium (**coronary circulation**).

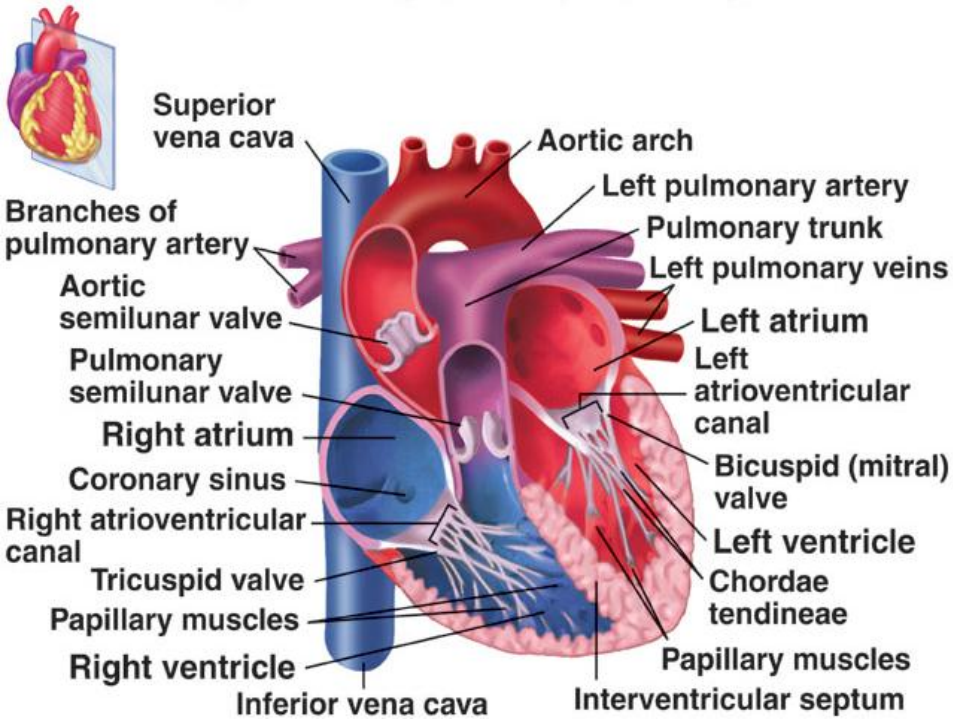
Within the lining of ventricles are crossbars called **trabeculae carneae** and **papillary muscles** project into heart cavity and hold the cusps of heart valves with **chordae tendineae**.

Heart valves

A. **Atrioventricular (AV)** valves are two (right and left) valves between atria and ventricles. They prevent backflow into atria. They are closed during ventricular contraction (**systole**). Right AV valve is **tricuspid** (with three cusps which are reinforced endocardium). Left AV valve is **bicuspid** (or mitral valve).

B. **Semilunar valves (SL)** are also two.

- **Aortic valve** between left ventricle and aorta.
- **Pulmonary valve** between right ventricle and pulmonary trunk.



Clinical considerations

A. **Angina pectoris**: Temporary deficient blood flow to the heart with severe chest pain.

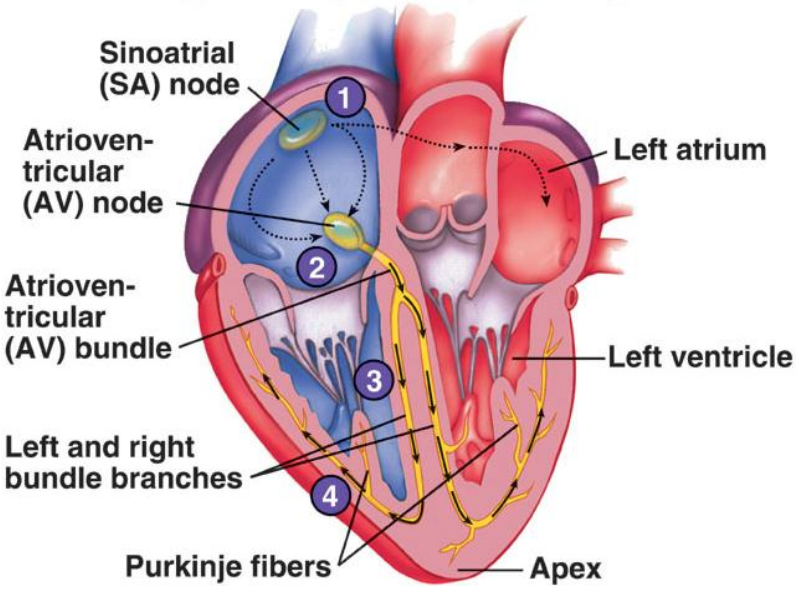
B. **Myocardial infarction (MI) or heart attack**: O2 deficiency causes necrosis (cell death). Cells that die are replaced by non-contractile scar tissue.

C. **Stenosis of heart valves** (mitral, tricuspid, aortic or pulmonary): Narrowing of one or more of heart valves with dynamic or fixed obstruction of blood flow passing through.

D. **Regurgitation** or **reflux** (of mitral, tricuspid, aortic or pulmonary): The blood backflows in the opposite direction due to valvular insufficiency.

Rhythmical excitation of the heart

Cardiac muscle fibers are branched, short, and interconnected. They are functionally connected by **intercalated discs** and **gap junctions**. As a result of gap junctions, entire myocardium acts as a single unit (**functional syncytium**). Cardiac muscle is self-excitable (i.e., **autorhytmic**) which initiate action potentials (AP) independent of nervous innervation. Presence of plateau and long refractory period after AP prevents tetanic contractions. There are specific non-contractile cardiac cells called **pacemaker** cells that form 1% of heart muscle and depolarize spontaneously to initiate and distribute impulses. Autorhythmic fibers are contractile muscle fibers depolarize in response to pacemaker cell activities.



Location of autorhythmic cells

1. **Sinoatrial (SA) node**: Has the fastest rate of depolarization. Sinus rhythm is the characteristic rhythm of the heart (gallop rhythm). Located in right atrial wall. After depolarization is initiated, depolarization wave sweeps via gap junctions throughout atria

2. **Atrioventricular (AV) node**. Depolarization wave initiated by SA node reaches AV node. AV node is located in interatrial septum near tricuspid valve. Diameter of fibers is smaller which slows impulse conduction (0.1 s) to permit completion of atrial contraction. Impulse passes to bundle of His

3. **Atrioventricular bundle (bundle of His)** permits functional passage of impulse from atria to ventricles. Located in inferior interatrial septum. It is very short and branches to form bundle branches
4. **Bundle branches** course interventricular septum toward apex of heart
5. **Purkinje fibers** reach apex then branch superiorly into ventricular walls. Impulses in fibers moves faster than cell to cell contact to ensure greater pumping efficacy

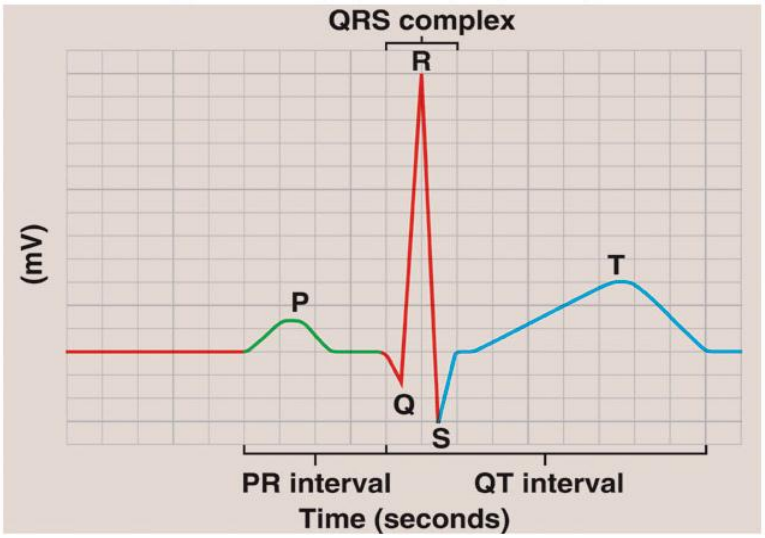
Clinical considerations

A. **Arrhythmias**: Uncoordinated contractions of atria and ventricles
B. **Fibrillation**: Rapid, irregular contractions (*atrial or ventricular fibrillation*).
C. **Ectopic focus**: Excitable tissue other than SA node controls heart contractions
D. **Heart block**: Damage to AV node so that impulse cannot reach ventricles

Extrinsic control of the heart (brain based control)

1. **Cardioaccelatory center** in medulla provides sympathetic NS control to innervate SA and AV nodes.
2. **Cardioinhibitory center** provides parasympathetic NS control via vagus nerve to SA and AV nodes to slow heart rate.

Electrocardiogram



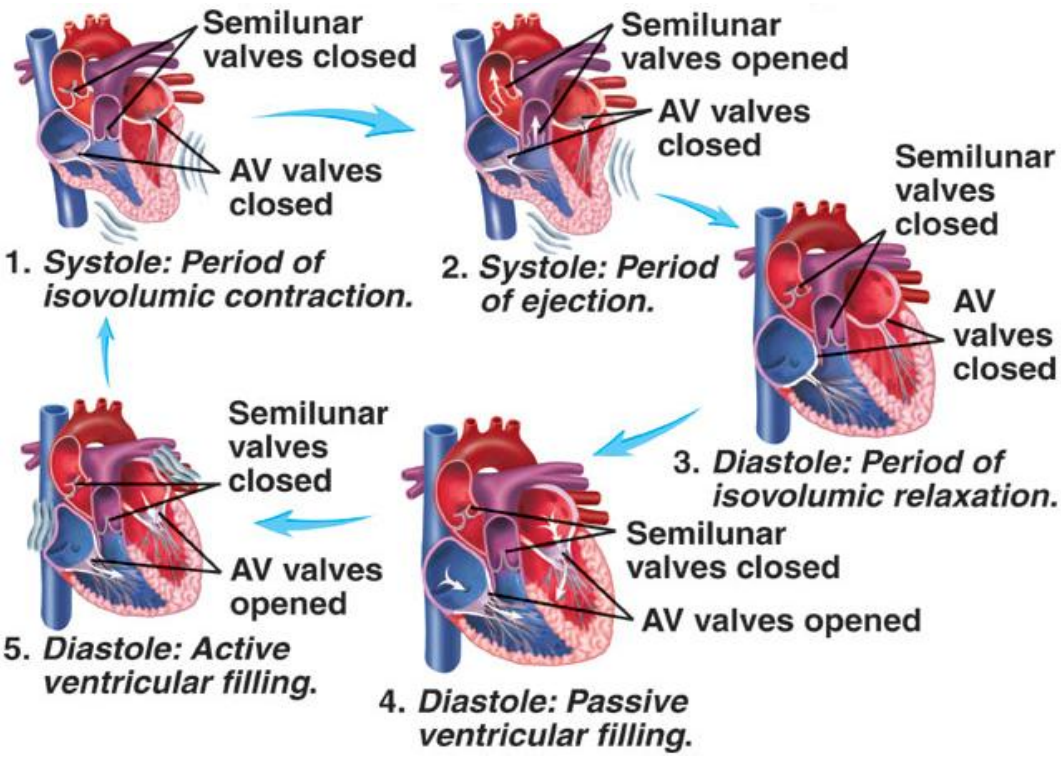
Three deflection waves and two intervals:

1. **P wave**: Depolarization moving from SA node through atria
2. **QRS complex**: Ventricular depolarization precedes contraction
3. **T wave**: Ventricular repolarization occurs more slowly than depolarization so it spreads out much than QRS
4. P-R: Interval from beginning of atrial excitation and ventricular excitation 0.16 sec.
5. Q-T: Interval from ventricular depolarization through repolarization.

Mechanics and regulation of heart pump

Cardiac cycle includes systole (contraction) and diastole (relaxation) of total 0.8 seconds. Atrial systole lasts 0.1 s and ventricular systole lasts 0.3 s while quiescent period lasts 0.4 s. Mechanical events are:

1. **Start point**: Atria and ventricles are relaxed.
2. **Ventricular filling**: AV valves are open, semilunar valves are closed, ventricles begin to fill (70% occurs before atrial contraction and 30% after atrial systole).
3. **Ventricular systole**: Contraction begins while AV valves and semilunar valves are closed. This period is called **isovolumetric contraction** phase. After that **Ventricular ejection phase** occurs when semilunar valves open and blood is propelled out of ventricles. Atria begin to fill with blood.
4. **Isovolumetric relaxation** Ventricles relax while semilunar and AV valves are closed.
5. **Quiescent period**: Start of another cycle when AV valves open.



Heart Sounds

They are associated with closing of heart valves. Sounds of separate valves can be differentiated. The sounds occur in the following sequence:
[1]Mitral, [2]Tricuspid, [3]Aortic semilunar, and lastly [4]Pulmonary semilunar.

Sound 1: AV valves close, onset of systole, louder and longer than sound 2
Sound 2: SL valves close, beginning of ventricular diastole, short, sharp sound
Pause: Quiescent period

Dynamics of blood flow through the circulation

Blood Flow (F) = ∆P/PR

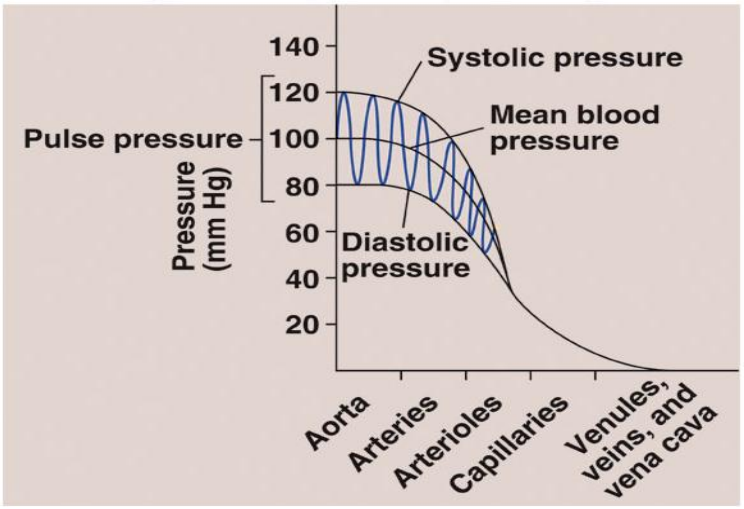
Flow: Is the volume flowing through a given structure per unit time (ml/min).
Pressure: Is the force per unit area (mm Hg).
Resistance: Opposition to flow in the systemic circuit (PR = peripheral resistance)

Sources of resistance (PR):
1. *Blood viscosity*: Is the thickness related to formed elements
2. *Total vessels length*: Longer vessels result in greater resistance
3. *Diameter*: Flow is inversely related to vessels diameter; larger diameter results in less resistance (**$1/r^4$**). In healthy humans, smaller diameter is the greatest source of resistance

Systemic BP:

Heart pumping generates blood flow. Pressure results when flow is opposed by resistance. Blood flows along a pressure gradient from higher to lower pressure. It is highest in aorta and lowest in right atrium.

Pulse pressure (PP) = Systolic BP _ Diastolic BP
or (PP=SBP_DBP).
Mean arterial pressure (MAP) = DBP + 1/3 PP



Venous pressure is very low. It cannot promote adequate venous return. So, it needs additional functional modifications. These functional modifications are:

a. **Respiratory pump**: During expiration, abdominal pressure increases squeeze local veins while backflow is prevented by vein valves. So, blood is forced toward the heart. At the same time, chest cavity pressure decreases and thoracic veins expand. So, blood enters right atrium.

b. **Muscular pump** (more important): Contraction of skeletal muscle surrounding veins compress vein and, again, backflow is prevented by valves. So, blood moves in direction of heart.

Cardiac output (CO)

Cardiac output is the amount of blood pumped by each ventricle per minute.

CO = Stroke volume * Heart rate

Stroke volume (SV)= volume of blood pumped out of each ventricle per beat =70 ml.
Heart rate (HR) is the number of **b**eats **p**er **m**inute (**BPM**) = 72 BPM

CO = 70 ml * 72 BPM = about 5 L/min

Factors that affect SV

1. **Preload**: Much stretch before contraction increases SV.
2. **Contractility**: Increase in contractile strength increases SV.
3. **Afterload**: Increase in arterial blood pressure (BP) decreases SV.

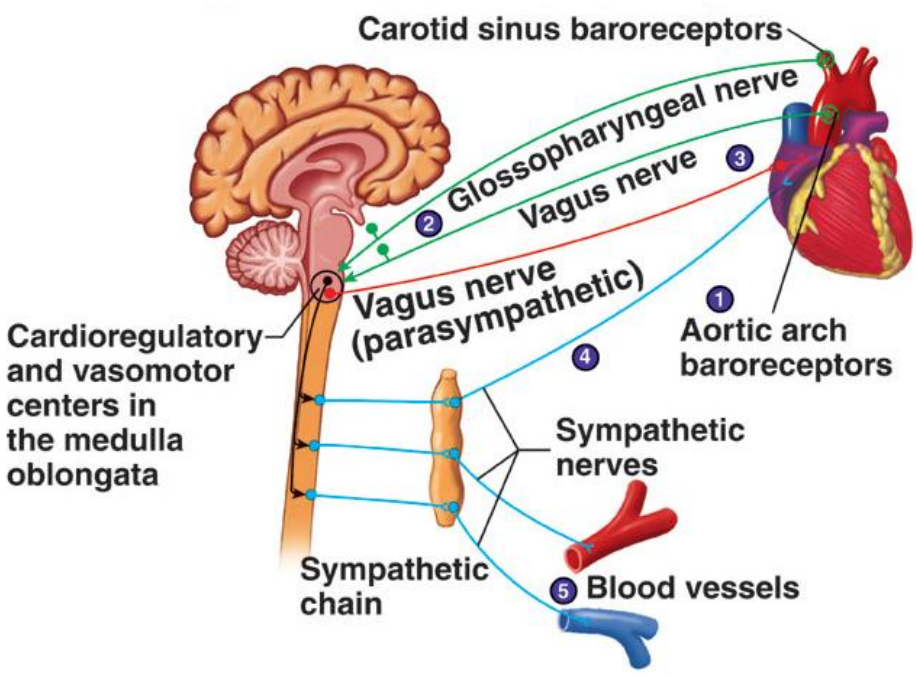
Regulation of systemic arterial pressure

BP = CO * PR

1. Reduced parasympathetic (vagal) control increases HR, CO and BP.
2. Increased sympathetic activity increases contractility of heart, SV and HR and also stimulates adrenal medulla to release epinephrine (EPI) hormone which increases HR.
3. Increased activity of respiratory and muscular pumps: Increases venous return and SV.

Neural control of blood pressure (short-term mechanisms):

Nervous control of peripheral resistance alters blood distribution and/or alters blood vessel diameter via *vasomotor center, baroreceptors and chemoreceptors*:



Chemical control of BP (short term mechanisms)

a. Adrenal medulla hormones: Norepinephrine (NE) is a vasoconstrictor increases PR while EPI increases cardiac muscle contractility and CO. Both increase BP.

b. ANP: Reduces BP by increasing water excretion from kidney.

c. Antidiuretic hormone (ADH): Increases BP by increasing water absorption. At high concentrations, it causes vasoconstriction (which increases PR and BP).

d. Angiotensin-II: Causes vasoconstriction of systemic arterioles (which increases PR and BP). It also causes release of aldosterone which increases reabsorption of water by kidney tubules. This will increase blood volume, venous return, SV, CO and BP.

e. Endothelium-derived factors: Endothelin and prostaglandin are vasoconstrictors (increase PR and BP) while nitrous oxide (NO) is a fast acting local vasodilator (decreases BP)

f. Inflammatory chemicals (vasodilators): e.g. histamine which also increases capillary permeability and, hence, decreases blood volume and BP.

g. Alcohol: Inhibits ADH release and increases vasodilation (of skin capillaries) by depressing vasomotor center. Both will result in decreased BP.

Renal regulation of BP (long-term mechanisms for BP regulation)

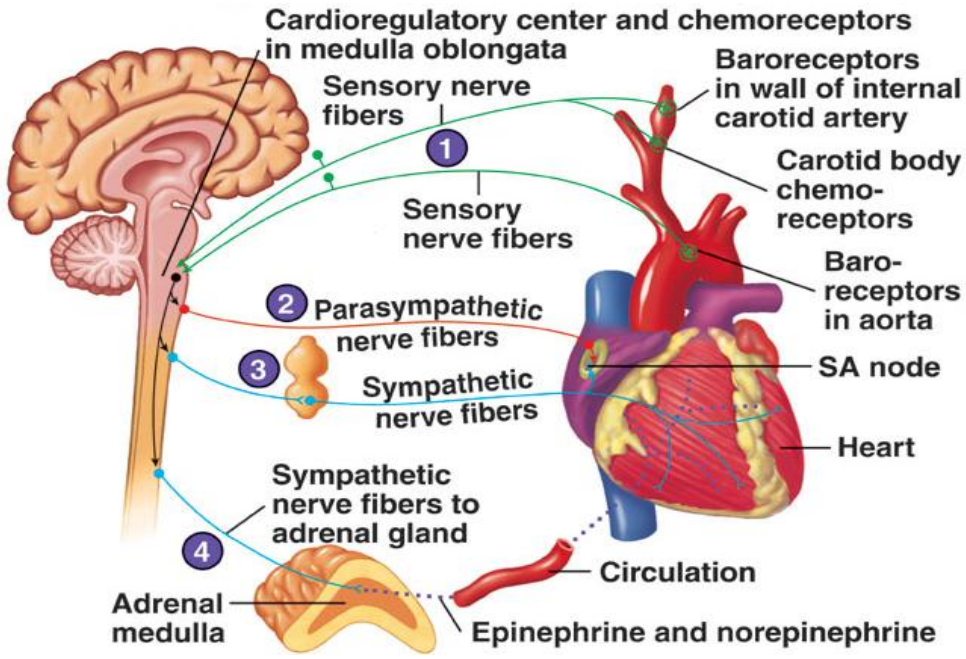
Kidney controls blood volume by regulating water loss in urine *directly* or *indirectly*. **Direct action** is to control the rate of fluid filtration from blood stream to kidney tubules. **Indirect actions** are by secretion of renin and aldosterone. Renin is converted to angiotensin-I in the liver. Angiotensin-I is converted to angiotensin-II in the lungs. Angiotensin-II stimulates adrenal cortex to secrete aldosterone and both increase Na^+ reabsorption. Aldosterone also causes posterior pituitary to release ADH (water reabsorption).

Regulation of CO (A. Neural, B. Chemical, and C. Physical mechanisms)

A. **Neural mechanisms**: Autonomic nervous system:

i. Sympathetic division: Increases HR, increases contractility, increases SV.

ii. *Parasympathetic division*: Opposes the effects of sympathetic division and decreases heart rate. Sympathetic and parasympathetic divisions are continuously active. Effect of parasympathetic division predominates (vagal tone). It reduces HR 25 beats/min.



B. **Chemical regulation**:

1. Hormones of adrenal medulla: Epinephrine and nor epinephrine).
2. Decreased Ca^{2+} concentrations cause depressed heart function and increased Ca^{2+} concentrations cause heart irritability

C. **Physical factors**:

1. Age: Inverse relation.
2. Gender: Female faster.
3. Exercise: Increases HR, SV and muscle mass.
4. Body temperature: HR lowered when cold.

Clinical considerations: Hypertension

A mean arterial pressure greater than 110 mm Hg (normal is about 90 mm Hg) is considered to be hypertensive. (This level of mean pressure occurs when the diastolic blood pressure is greater than about 90 mm Hg and the systolic pressure is greater than about 135 mm Hg.) In severe hypertension, the mean arterial pressure can rise to 150 to 170 mm Hg, with diastolic pressure as high as 130 mm Hg and systolic pressure occasionally as high as 250 mm Hg.

Heart failure

The heart fails to pump enough blood to the body. The cause usually is decreased contractility of the myocardium resulting from diminished coronary blood flow. However, failure can also be caused by damaged heart valves, external pressure around the heart, or other causes.

Circulatoryshock

1- **Cardiogenic shock**. It is due to decreased ability of the heart to pump blood. These include especially myocardial infarction but also toxic states of the heart, severe heart valve dysfunction, heart arrhythmias, and other conditions.
2- **Hypovolemic shock**. Factors that decrease venous return also decrease CO. The most common cause of decreased venous return is diminished blood volume, but venous return can also be reduced as a result of decreased vascular tone, especially of the venous blood reservoirs (larger veins and sinuses), or obstruction to blood flow at some point in the circulation, especially in the venous return pathway to the heart.
3- **Vasovagal syncope**: Normal fainting which is often caused by fear and anxiety. Decreased HR and BP result in less blood flow to the brain. The blood pools in lower extremities. In dental clinics, the dentist needs to take more time explaining the dental procedure to them, thus allaying their fears. Other patients may require sedation. Inhalation sedation (nitrous oxide/oxygen) may be ideal for some patients, while enteral sedation or even general anesthesia may be more appropriate for others. When syncope occurs, the patient must be laid in supine position with his legs level higher than his body to ensure blood return to the heart and then to the brain.