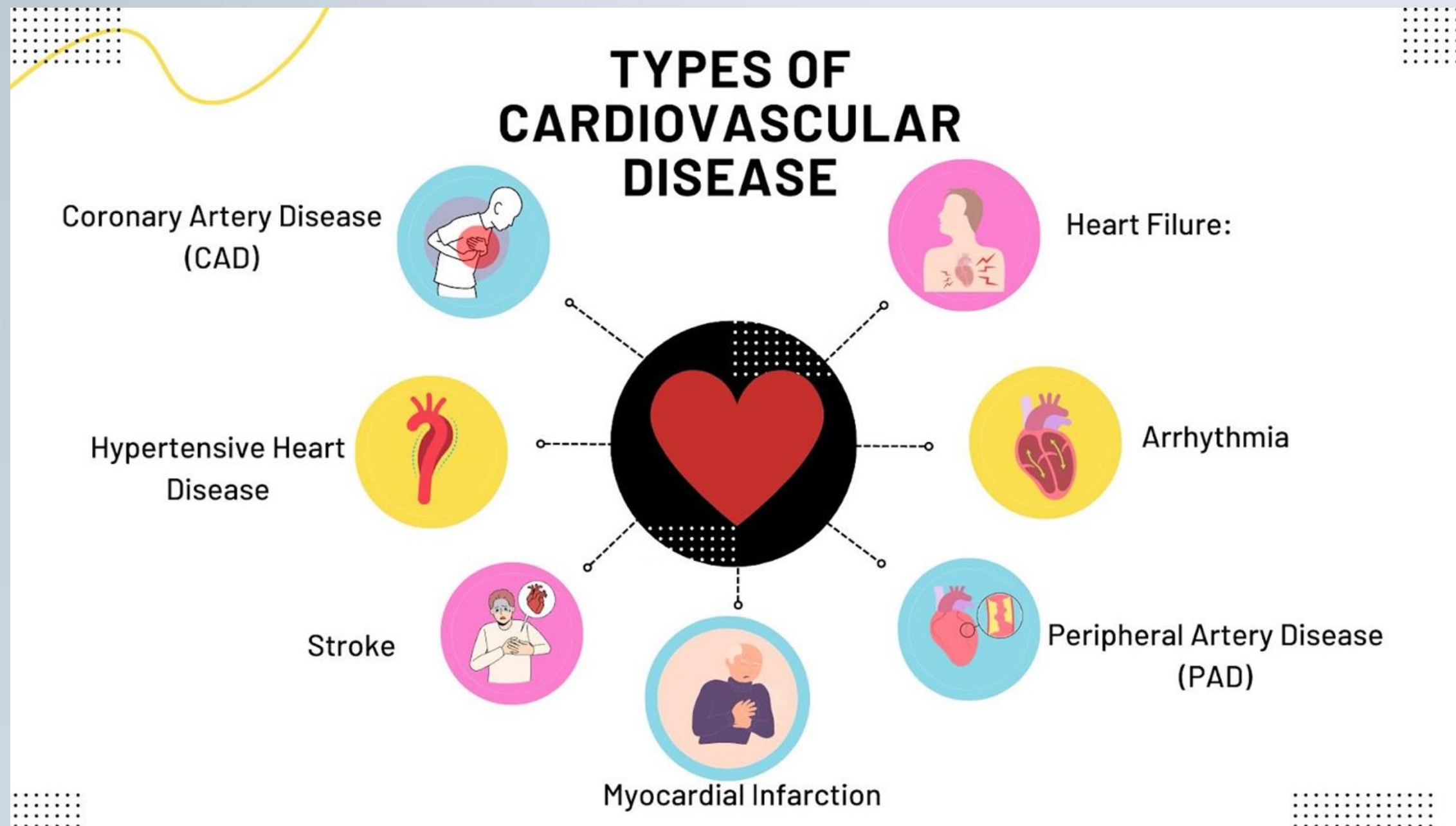


DISORDERS OF CARDIOVASCULAR SYSTEM



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HYPERTENSION

- *Hypertension* is defined as persistently elevated arterial blood pressure (BP).
- Hypertension, also known as high blood pressure, is a chronic medical condition in which the force of blood against the artery walls is consistently too high, increasing the risk of heart disease, stroke, and other health complications.

WHO Definition of Hypertension

According to the World Health Organization (WHO), hypertension is defined as:

Systolic Blood Pressure (SBP) $\geq$ 140 mmHg

Diastolic Blood Pressure (DBP) $\geq$ 90 mmHg

Based on repeated measurements under standardized conditions.

Risk factors

Obesity	A sedentary lifestyle
Diabetes mellitus	High intakes of salt or alcohol
Family history	Stress
Cigarette smoking	Low birth weight

Hypertension is described as essential (primary, idiopathic) or secondary to other diseases.

Essential hypertension

1. A condition of persistently elevated blood pressure without a known secondary cause, often associated with genetic predisposition and lifestyle factors.

Benign (chronic) hypertension

1. The rise in blood pressure is usually slight to moderate and continues to rise slowly over many years.
2. Sometimes complications, such as heart failure, cerebrovascular accident or myocardial infarction are the first indication of hypertension, but often the condition is symptomless and is only discovered during a routine examination.

Malignant (accelerated) hypertension

1. This is a rapid and aggressive acceleration of hypertensive disease.
2. Diastolic pressure in excess of 120 mmHg is common.
3. The effects are serious and quickly become apparent, e.g. haemorrhages into the retina, papilloedema (oedema around the optic disc), encephalopathy (cerebral oedema) and progressive renal disease, leading to cardiac failure.

Secondary hypertension

Hypertension resulting from other diseases accounts for 5% of all cases.

I. Kidney disease

- a. Raised blood pressure is a complication of many kidney diseases. In kidney disease, there is salt and water retention, sometimes with excessive renin activity.

II. Endocrine disorders

I. Adrenal cortex

- a. Secretion of excess aldosterone and cortisol stimulates the retention of excess sodium and water by the kidneys, raising the blood volume and pressure.

II. Adrenal medulla

- a. Secretion of excess adrenaline (epinephrine) and noradrenaline (norepinephrine) raises blood pressure, e.g. phaeochromocytoma .

III. Stricture of the aorta

- a. Hypertension develops in branching arteries proximal to the site of a stricture, e.g *congenital coarctation*.

IV. Drug treatment

- a. Hypertension may be a side-effect of some drugs, e.g. corticosteroids and oral contraceptives.

Effects and complications of hypertension

1. Heart

- i. The rate and force of cardiac contraction are increased to maintain the cardiac output against a sustained rise in arterial pressure.

2. Brain

1. Stroke, caused by cerebral haemorrhage, is common, the effects depending on the position and size of the ruptured vessel.
2. When a series of small blood vessels rupture, e.g. microaneurysms, at different times, there is progressive disability.
3. Rupture of a large vessel causes extensive loss of function or death.

3. Kidneys

- i. Essential hypertension causes kidney damage.
- ii. If sustained for only a short time recovery may be complete. Otherwise the kidney damage causes further hypertension owing to activation of the renin– angiotensin– aldosterone system, progressive loss of kidney function and kidney failure.

4. Blood vessels

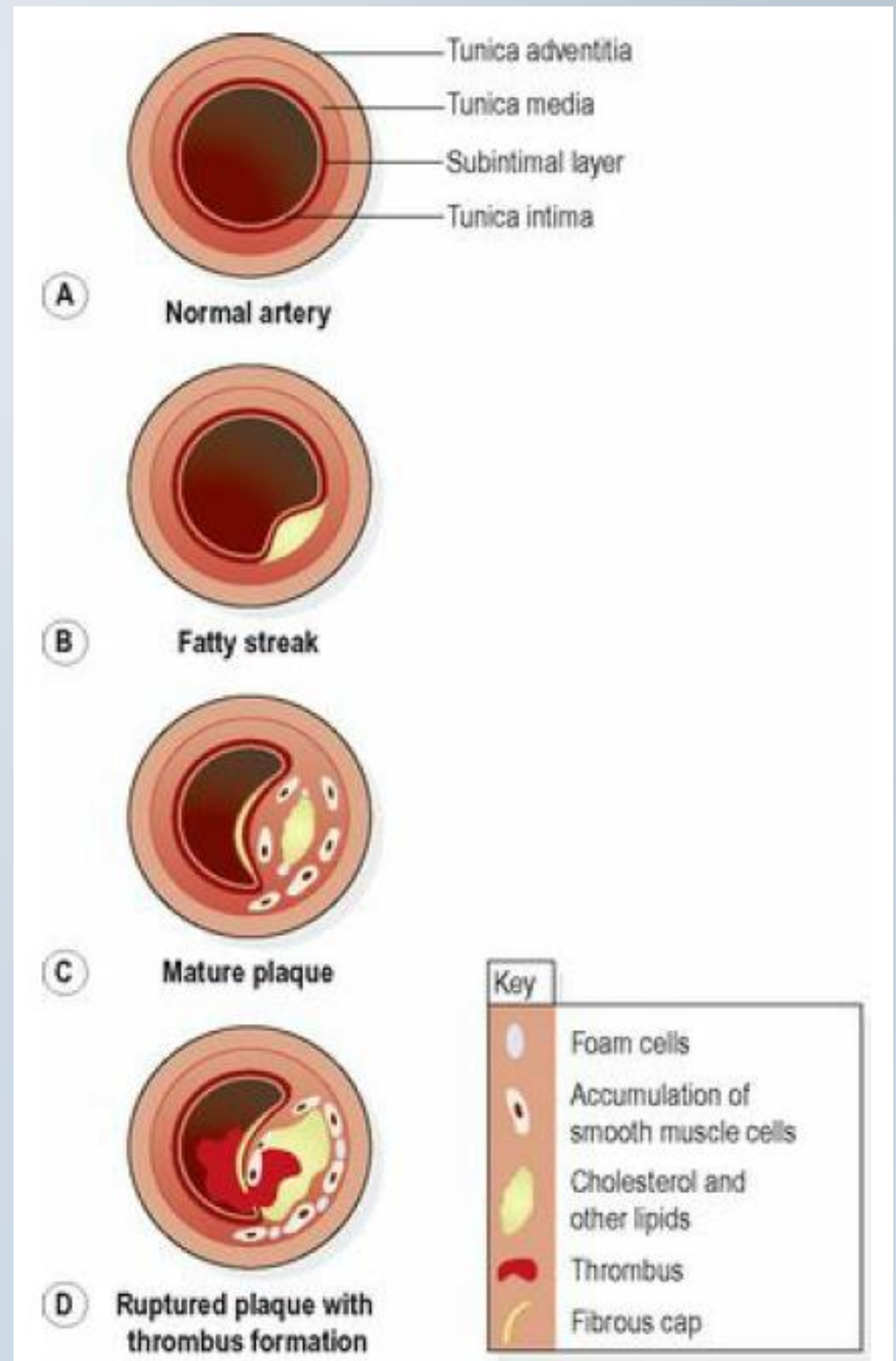
1. High blood pressure damages blood vessels.
2. The walls of small arteries become hardened, and in larger arteries, atheroma is accelerated.
3. The vessel wall may become so badly weakened by these changes that an aneurysm develops, and as the blood vessels become progressively damaged and less elastic, hypertension worsens.
4. The capillaries of the retina and the kidneys are particularly susceptible to the effects of chronic hypertension, leading to retinal bleeding and reduced renal function.

HYPOTENSION

1. Hypotension is characterized by abnormally low BP.
2. This usually occurs as a complication of other conditions, such as shock or Addison's disease.
3. Low blood pressure leads to inadequate blood supply to the brain.
4. Depending on the cause, unconsciousness may be brief (fainting) or more prolonged, possibly causing death.
5. Postural hypotension is an abrupt fall in blood pressure on standing up suddenly from a sitting or lying position. It causes dizziness and occasionally syncope (fainting).

ATHEROSCLEROSIS / ATHEROMA

1. Atherosclerosis is a chronic, progressive disease characterized by the thickening and hardening of arterial walls due to the buildup of plaque (composed of fat, cholesterol, calcium, and other substances) inside the arteries.
2. This leads to narrowing and reduced elasticity of the arteries, which can restrict blood flow and increase the risk of cardiovascular diseases.
3. *Atheromatous plaques* are patchy changes that develop in the tunica intima of large and medium-sized arteries.
4. Initial changes show a fatty streak in the artery wall.
5. Mature plaques consist of accumulations of cholesterol and other lipid compounds, excess smooth muscle and fat-filled monocytes (foam cells).
6. The plaque is covered with a fibrous cap.



7. As plaques grow they spread along the artery wall forming swellings that protrude into the lumen.
8. Eventually the whole thickness of the wall and long sections of the vessel may be affected.
9. Plaques may rupture, exposing subintimal materials to the blood. This may cause thrombosis and vasospasm and will compromise blood flow.
10. Arteries most commonly involved are those in the heart, brain, kidneys, small intestine and lower limbs.

Predisposing factors in atherosclerosis

- | | |
|---|-----------------------------------|
| 1. Heredity – family history | |
| 2. Obesity | |
| 3. Gender – males are more susceptible than females, until after the female menopause | |
| 4. Diet – high in refined carbohydrates and/or saturated fats and cholesterol | |
| 5. Increasing age | |
| 6. Smoking cigarettes | |
| | 7. Diabetes mellitus |
| | 8. Excessive emotional stress |
| | 9. Hypertension |
| | 10. Sedentary lifestyle |
| | 11. Hyperlipidaemia |
| | 12. Excessive alcohol consumption |

Complications of atheroma

I. Thrombosis and infarction

- 1.If the fibrous cap overlying a plaque breaks down, platelets are activated by the damaged cells and a blood clot forms, blocking the artery and causing ischaemia and infarction.
- 2.Emboli may break off, travel in the bloodstream and lodge in small arteries distal to the clot, causing small infarcts.

II.Haemorrhage

- 1.Plaques may become calcified, making the artery brittle, rigid and unresponsive to rises in blood pressure.
- 2.They may rupture, causing haemorrhage.

III.Aneurysm

1. When the arterial wall is weakened by spread of the plaque between the layers of tissue, a local dilation (aneurysm) may develop.

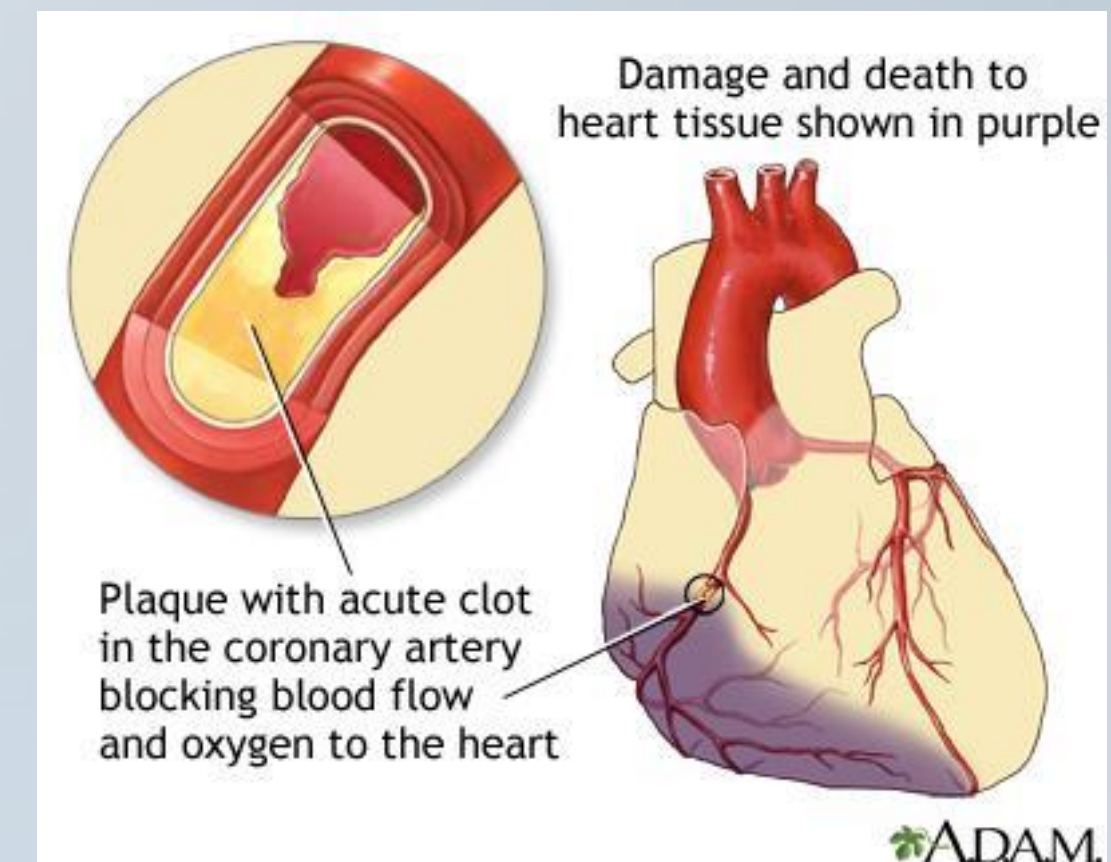
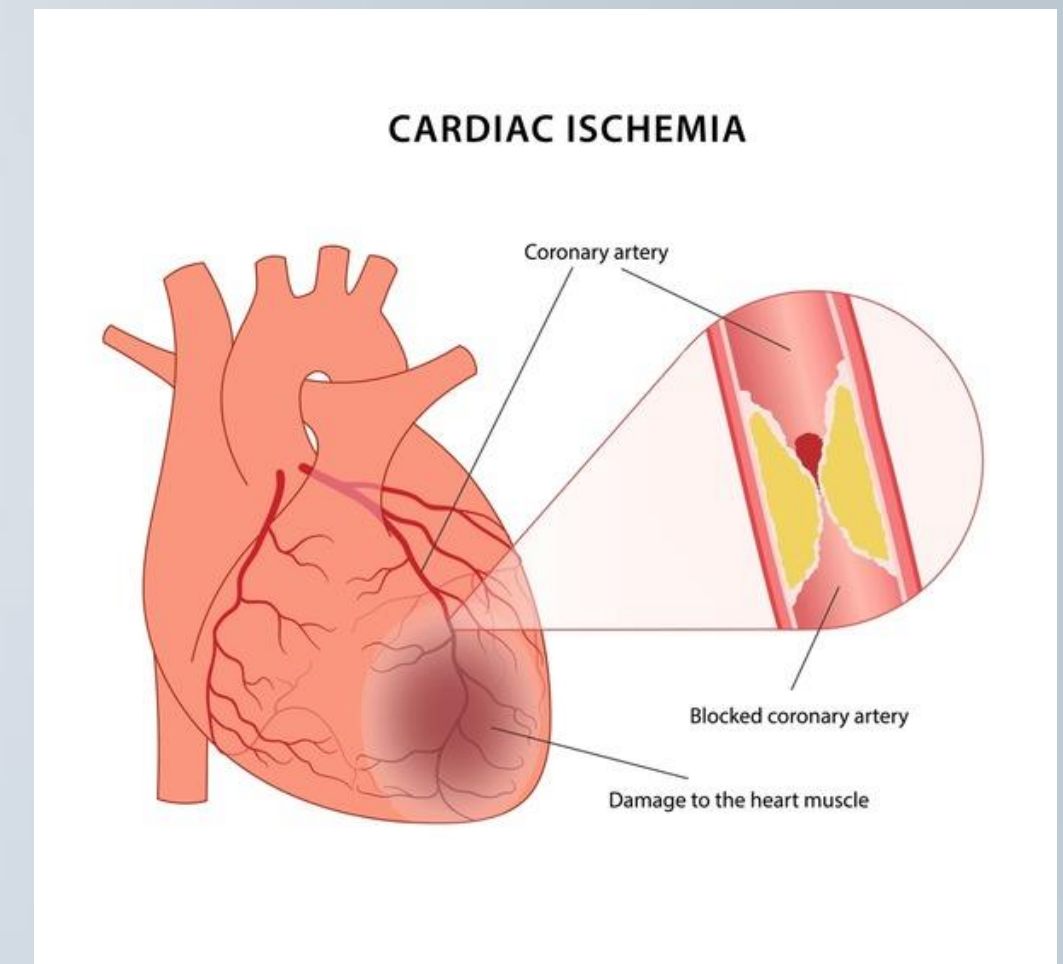
This may lead to thrombosis and embolism, or the aneurysm may rupture causing severe haemorrhage.

ARTERIOSCLEROSIS

1. This is a progressive degeneration of arterial walls, associated with ageing and accompanied by hypertension.
2. In large and medium-sized arteries, the tunica media is infiltrated with fibrous tissue and calcium.
3. This causes the vessels to become dilated, inelastic and tortuous.
4. Loss of elasticity increases systolic blood pressure, and the *pulse*.
5. When small arteries (arterioles) are involved, their lumen is reduced because of a deposition of a substance called *hyaline material*, which also reduces the elasticity of the vessel wall.
6. Because these arteries are the main determinants of peripheral resistance, this narrowing increases peripheral resistance and blood pressure.
7. Damage to small vessels has a disproportionate effect on blood flow, leading to ischaemia of tissues supplied by affected arteries.
8. In the limbs, the resultant ischaemia predisposes to gangrene, which is particularly serious in people with diabetes mellitus.

ISCHAEMIC HEART DISEASE

1. Ischaemia is due to the effects of atheroma, causing narrowing or occlusion of one or more branches of the coronary arteries.
2. Atheromatous plaques cause narrowing.
3. Occlusion may be by plaques alone, or plaques complicated by thrombosis.
4. The overall effect depends on the size of the coronary artery involved and whether it is only narrowed or completely blocked.
5. Narrowing of an artery leads to *angina pectoris*, and occlusion to myocardial infarction.
6. When atheroma develops slowly, a *collateral arterial blood supply* may have time to develop and effectively supplement or replace the original.
7. This consists of the dilation of normally occurring anastomotic arteries joining adjacent arteries.
8. When sudden severe narrowing or occlusion of an artery occurs, the anastomotic arteries dilate but may not be able to supply enough blood to meet myocardial needs.



ANGINA PECTORIS

1. According to the **WHO**, angina pectoris is:

"A condition characterized by episodes of chest pain or discomfort due to transient myocardial ischemia, typically precipitated by exertion and relieved by rest or nitrates, without causing permanent heart muscle damage."

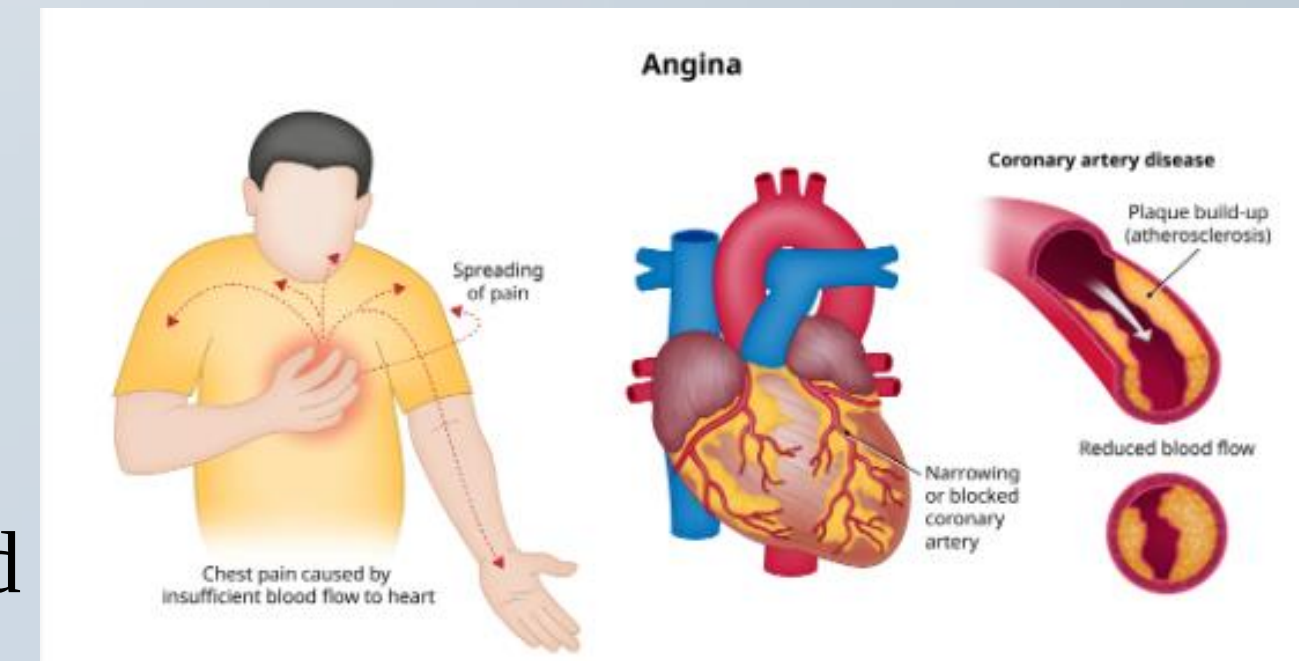
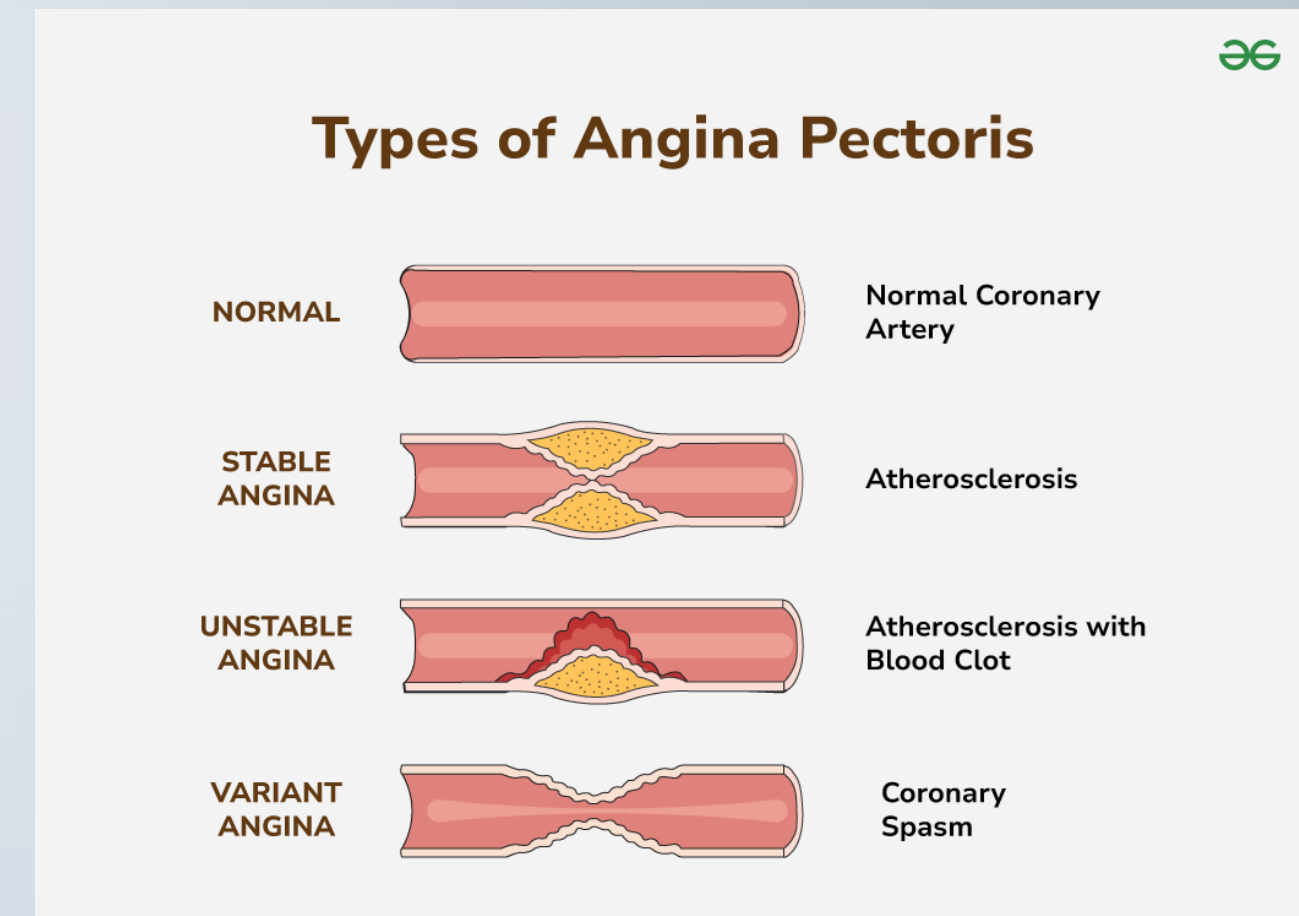
2. Types of Angina :

a. Stable Angina: Predictable, occurs with exertion, relieved by rest.

b. Unstable Angina: Occurs at rest or with minimal exertion; more severe and may indicate an impending heart attack.

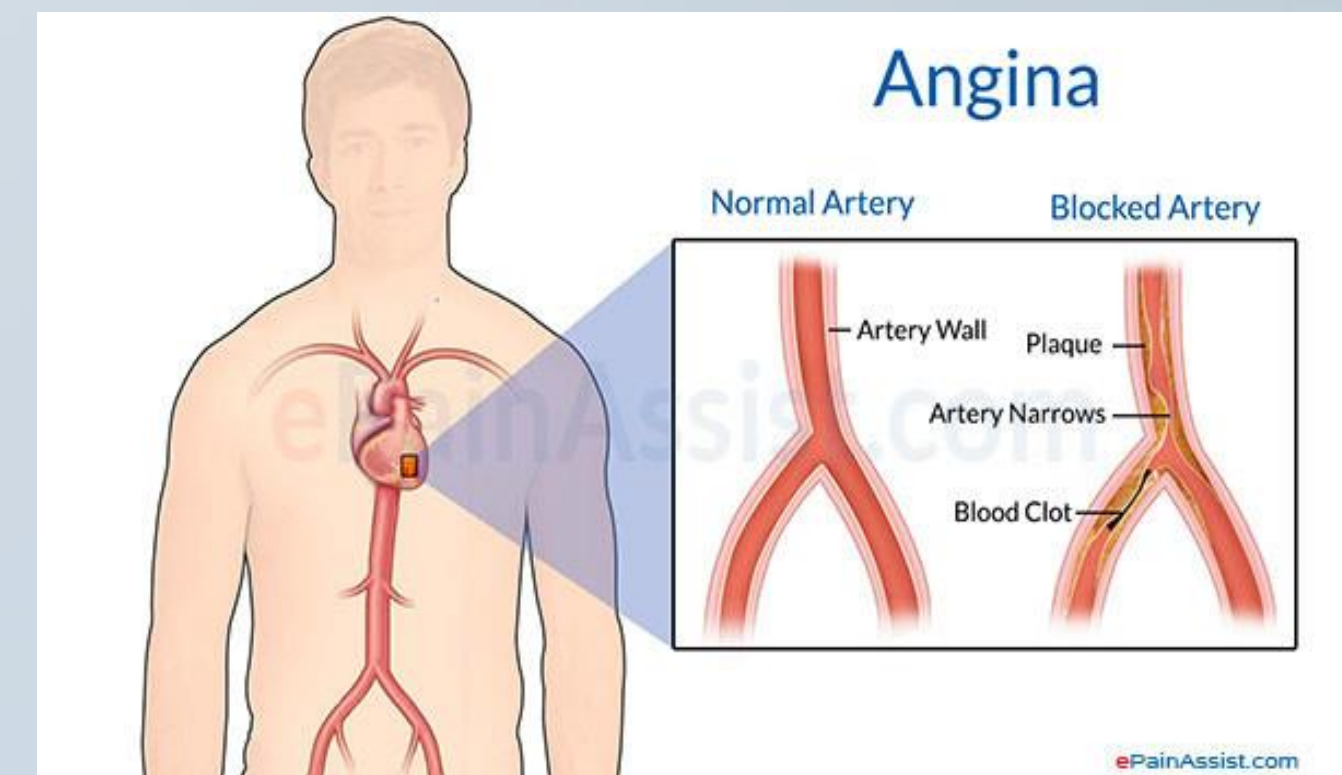
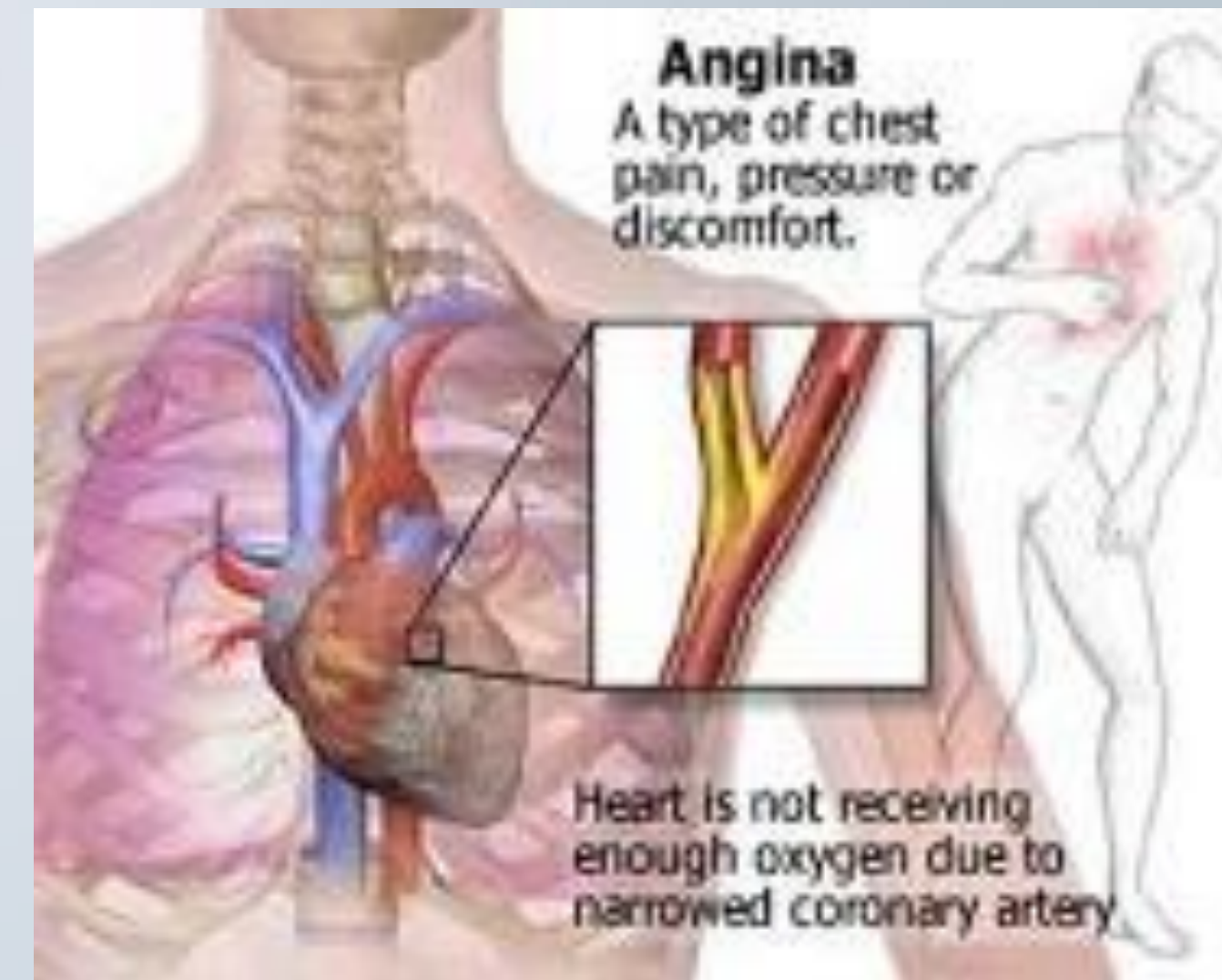
c. Variant (Prinzmetal's) Angina: Caused by coronary artery spasms, occurs at rest, and is unrelated to exertion.

3. This is sometimes called *angina of effort* because the increased cardiac output required during extra physical effort causes severe chest pain, which may also radiate to the arms, neck and jaw.



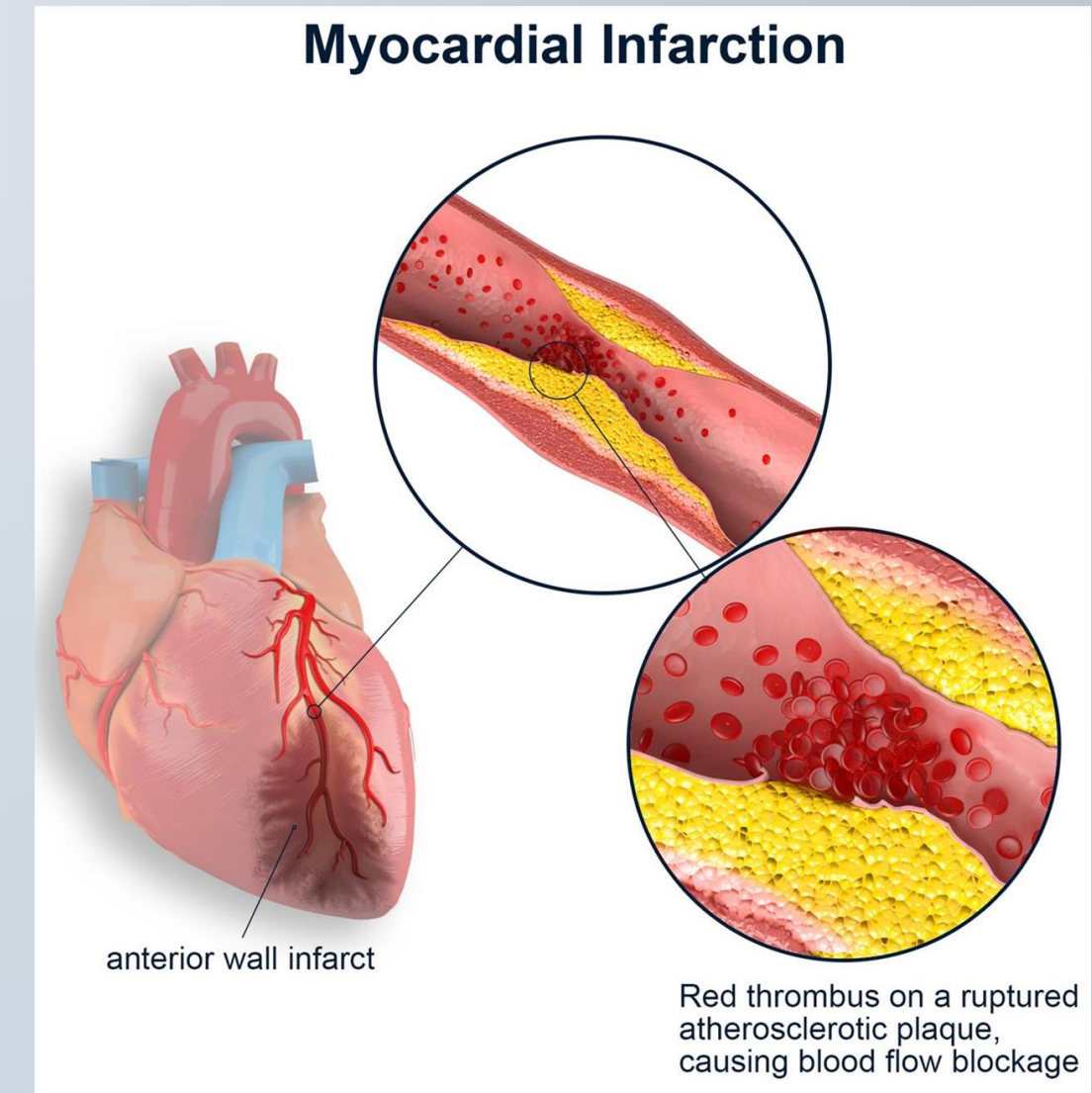
4. Other factors that may precipitate angina include cold weather and emotional states.
5. A narrowed coronary artery may supply sufficient blood to the myocardium to meet its needs during rest or moderate exercise but not when greatly increased cardiac output is needed, e.g. walking may be tolerated but not running.
6. The thick, inflexible atheromatous artery wall is unable to dilate to allow for the increased blood flow needed by the more active myocardium, which then becomes ischaemic.

In the early stages of angina, the chest pain stops when the cardiac output returns to its resting level soon after the extra effort stops.



MYOCARDIAL INFARCTION

1. The myocardium may infarct when a branch of a coronary artery is blocked.
2. The commonest cause is an atheromatous plaque complicated by thrombosis.
3. The extent of myocardial damage depends on the size of the blood vessel and site of the infarct.
4. The damage is permanent because cardiac muscle cannot regenerate, and the dead muscle is replaced with non-functional fibrous tissue.
5. Speedy restoration of blood flow through the blocked artery using clot-dissolving (thrombolytic) drugs can greatly reduce the extent of the permanent damage and improve prognosis, but treatment must be started within a few hours of the infarction occurring.
6. The effects and complications are greatest when the left ventricle is involved.
7. Myocardial infarction is usually accompanied by very severe crushing chest pain behind the sternum which, unlike angina pectoris, continues even when the individual is at rest.
8. It is a significant cause of death in the developed world.



Complications

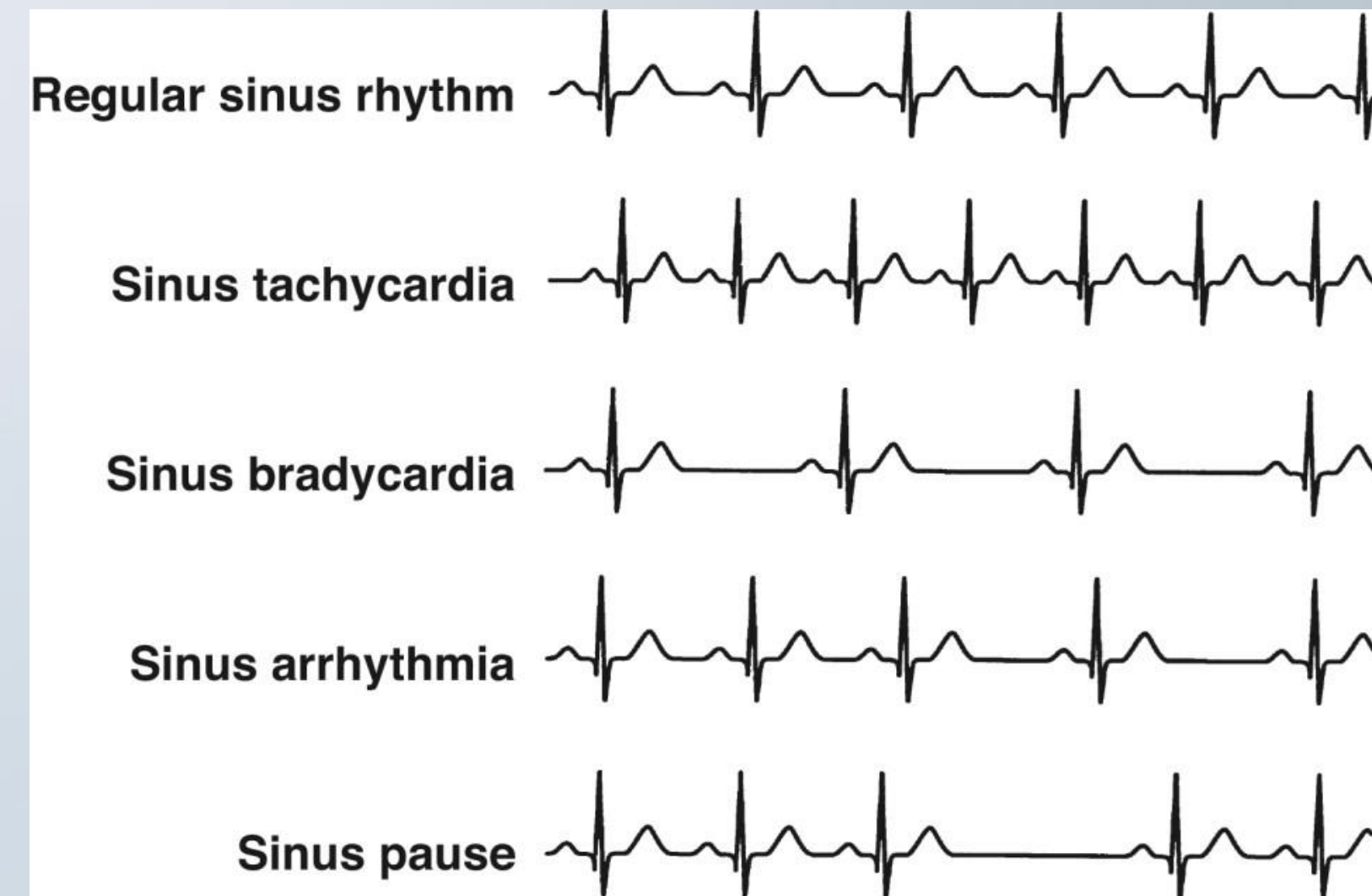
These may be fatal and include:

1. **Severe arrhythmias**, especially *ventricular fibrillation*, due to disruption of the cardiac conduction system acute heart , caused by impaired contraction of the damaged myocardium,
2. **Cardiogenic shock** rupture of a ventricle wall, usually within 2 weeks of the original episode pulmonary or cerebral embolism originating from a mural clot within a ventricle, i.e. a clot that forms inside the heart over the infarct pericarditis angina pectoris recurrence.

CARDIAC ARRHYTHMIAS

1. The heart rate is normally determined by intrinsic impulses generated in the SA node.
2. The rhythm is determined by the route of impulse transmission through the conducting system. The heart rate is usually measured as the pulse, but to determine the rhythm, an electrocardiogram (ECG) is required.
3. A *cardiac arrhythmia* is any disorder of heart rate or rhythm, and is the result of abnormal generation or conduction of impulses.
4. The normal cardiac cycle gives rise to *normal sinus rhythm*, which has a rate of between 60 and 100 bpm.

ECG traces: **A.** Normal sinus rhythm. **B.** Life-threatening arrhythmias.



I. Sinus bradycardia

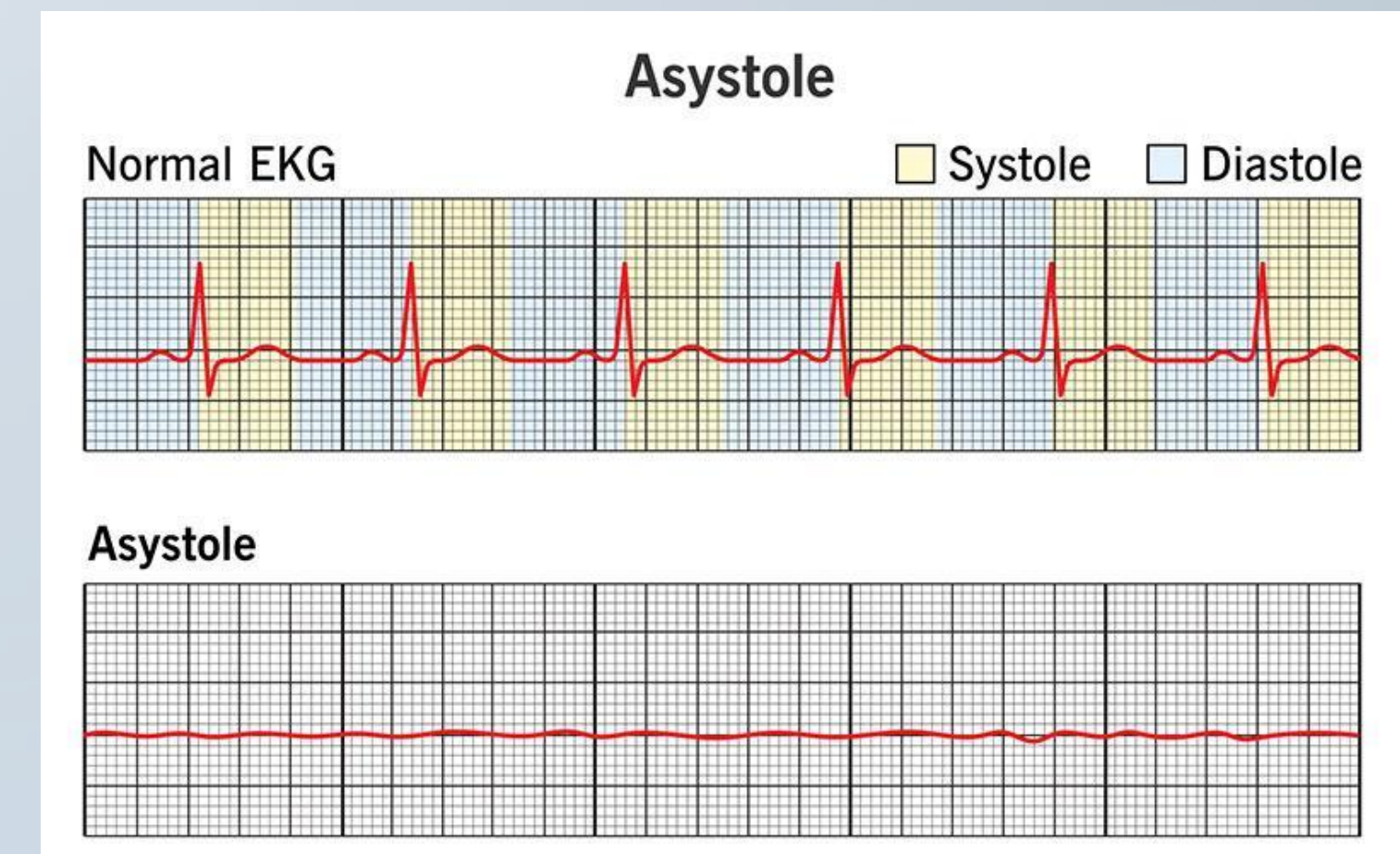
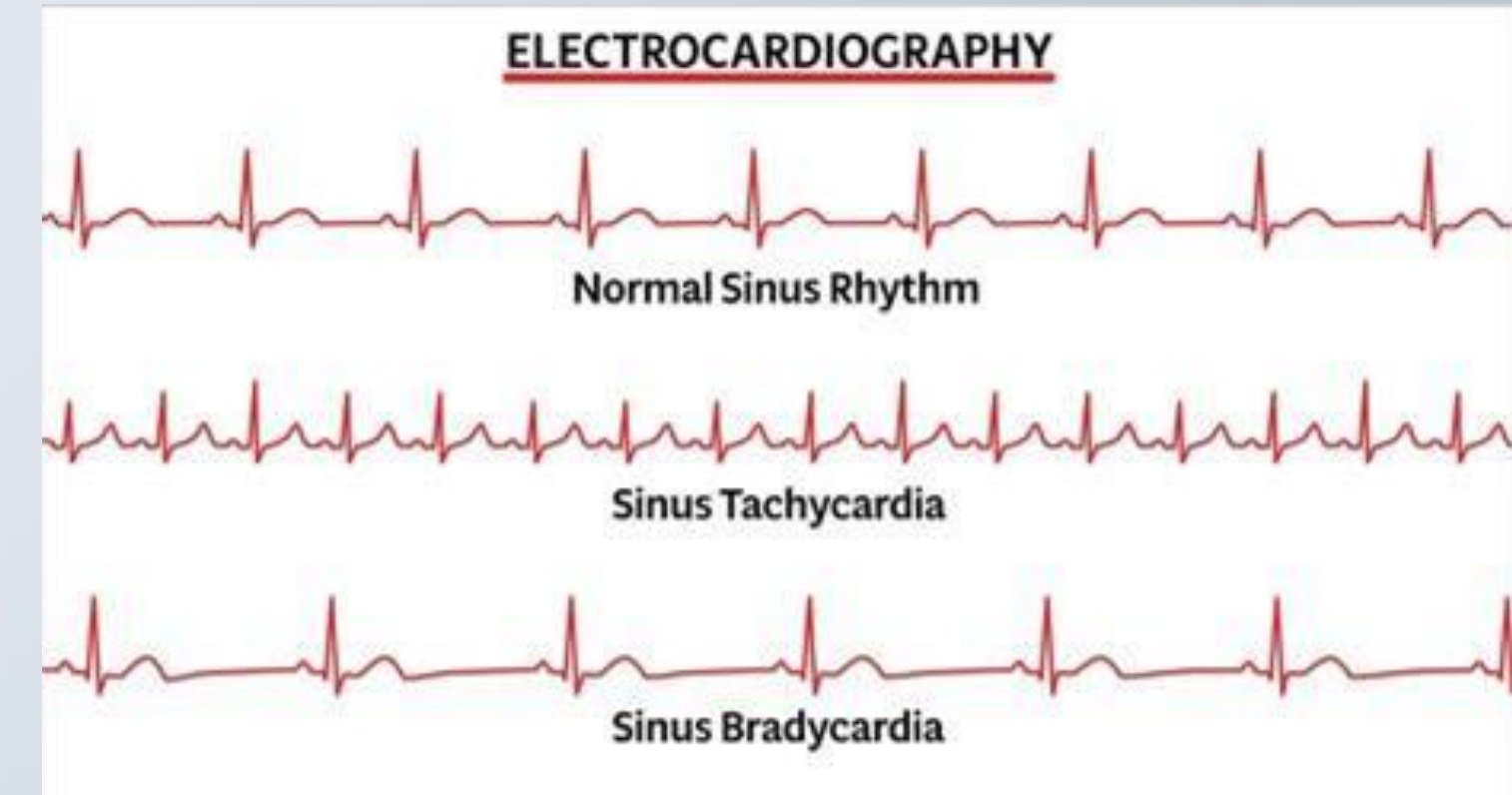
1. This is normal sinus rhythm below 60 bpm. This may occur during sleep and is common in athletes.
2. It is an abnormality when it follows myocardial infarction or accompanies raised intracranial pressure.

II. Sinus tachycardia

1. This is normal sinus rhythm above 100 bpm when the individual is at rest.
2. This accompanies exercise and anxiety, but is an indicator of some disorders, e.g. fever, hyperthyroidism, some cardiac conditions.

III. Asystole

1. This occurs when there is no electrical activity in the ventricles and therefore no cardiac output. The ECG shows a flat line.
2. Ventricular fibrillation and asystole cause sudden and complete loss of cardiac output, i.e. *cardiac arrest* and death.



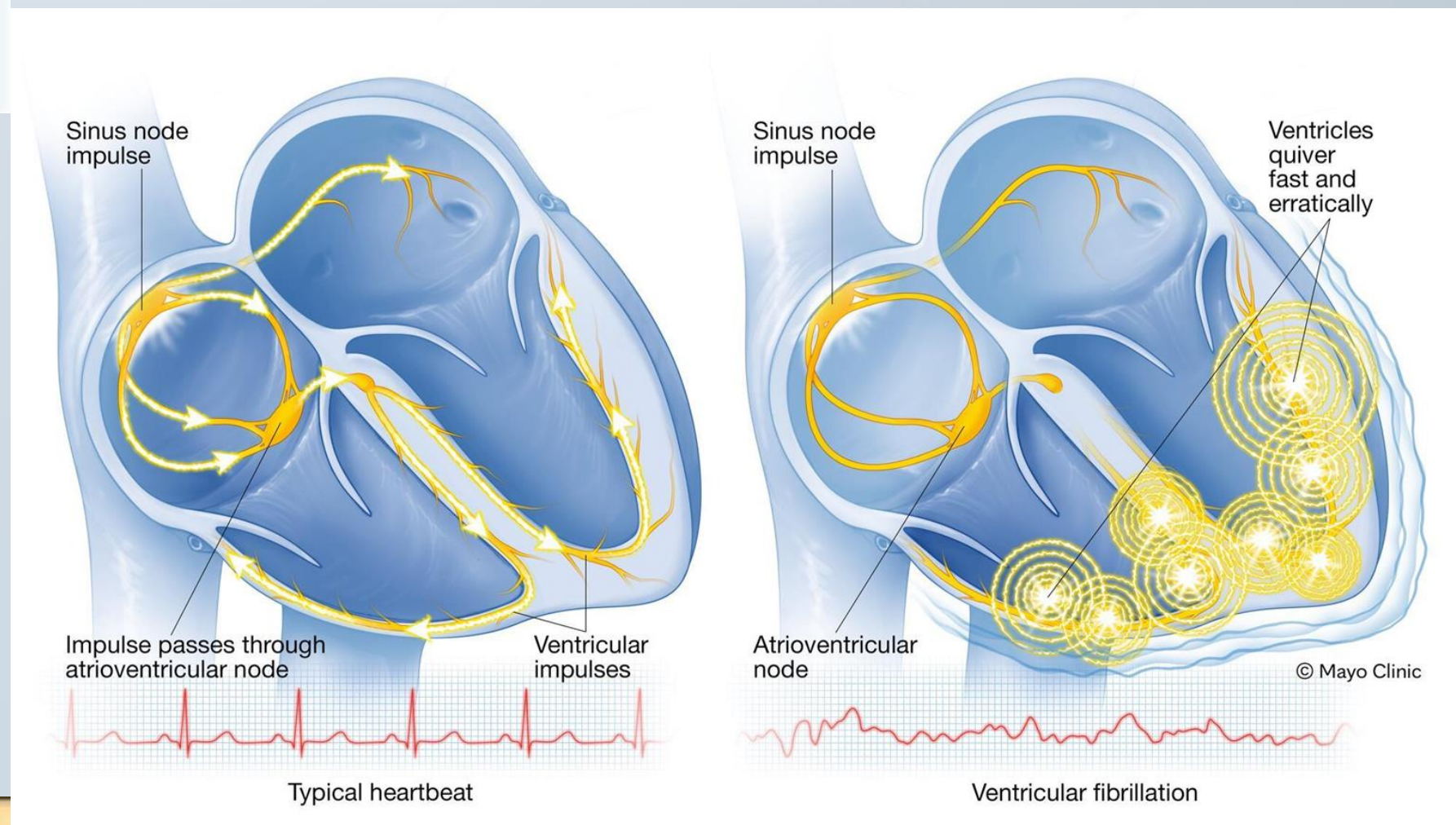
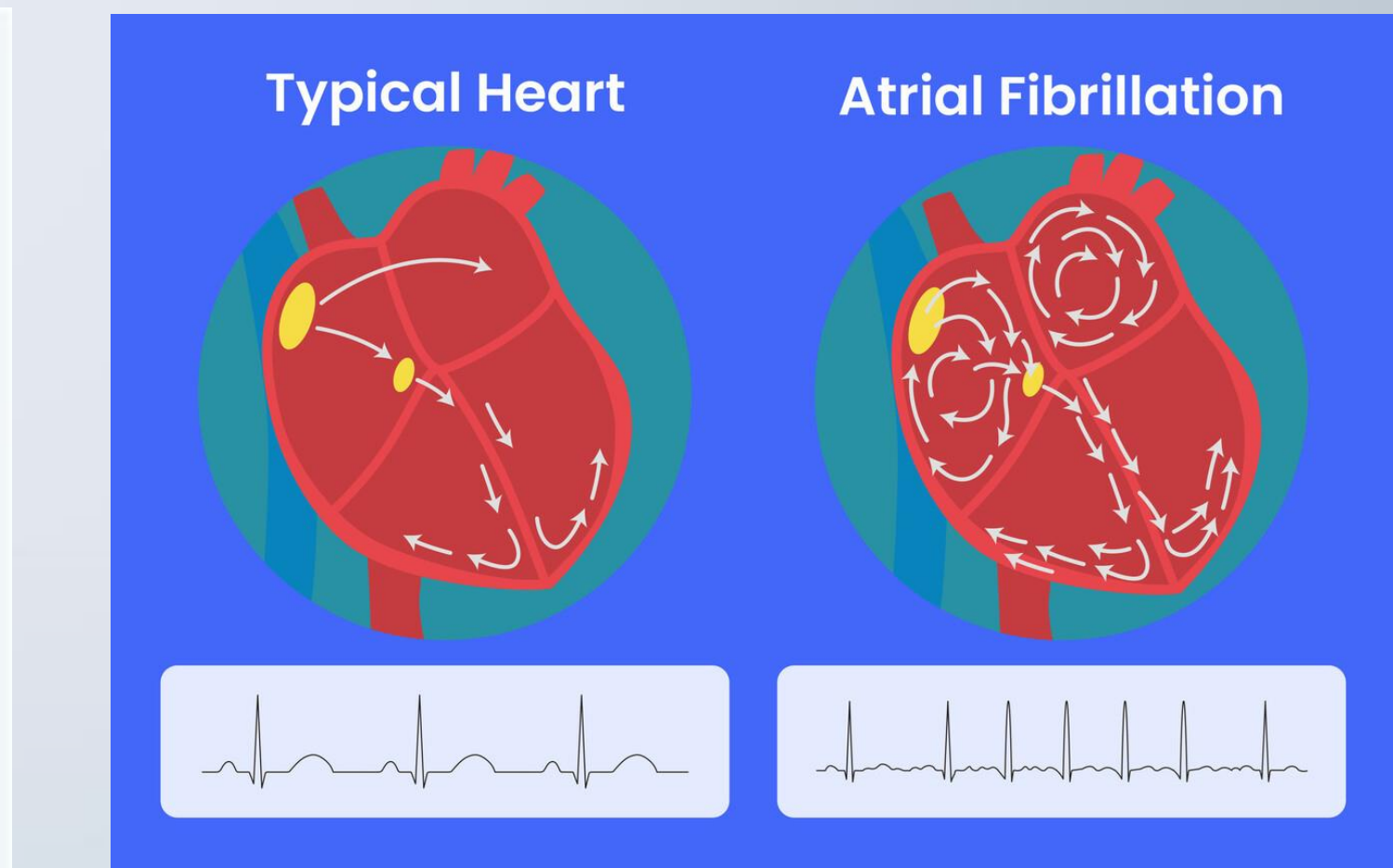
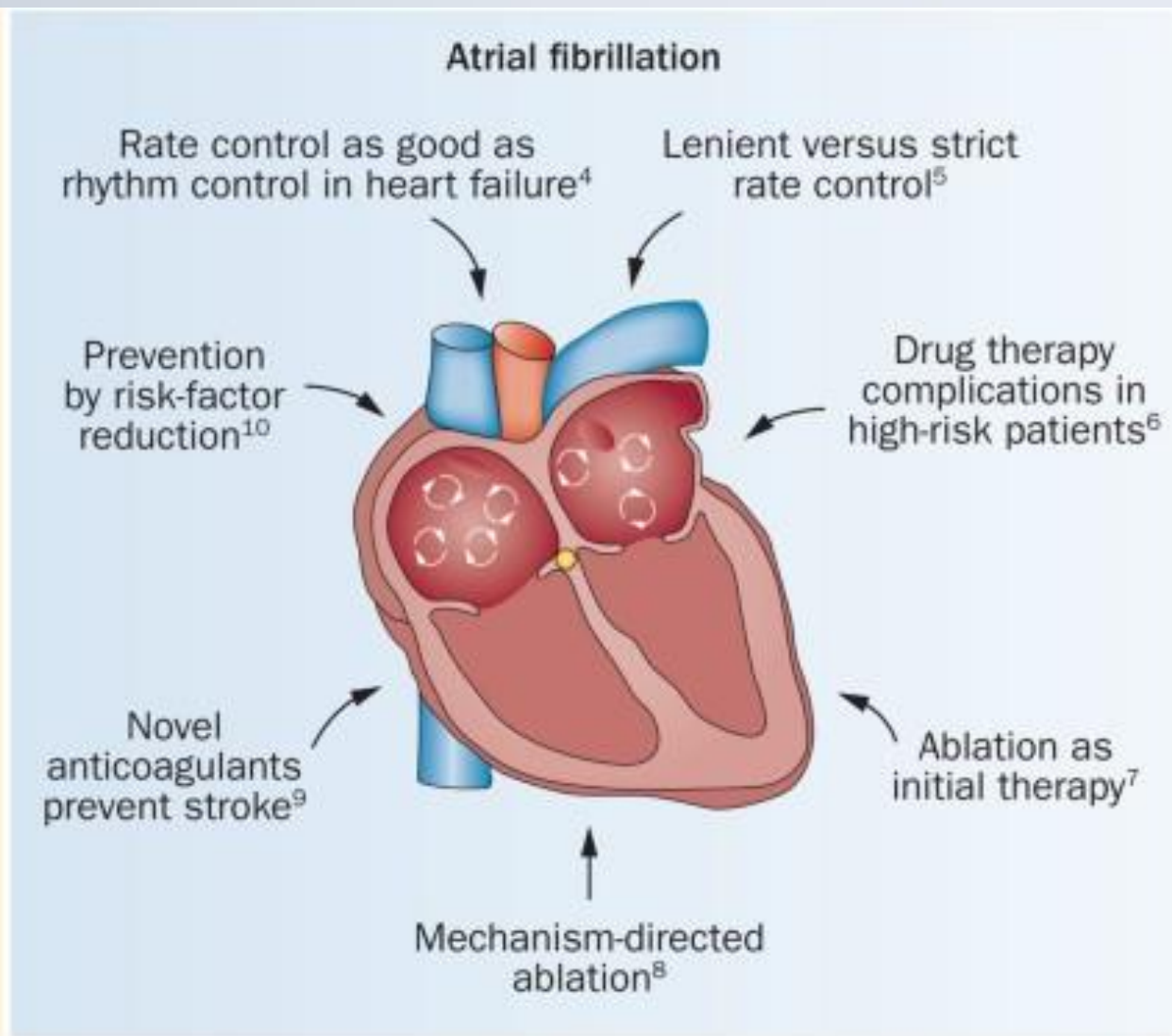
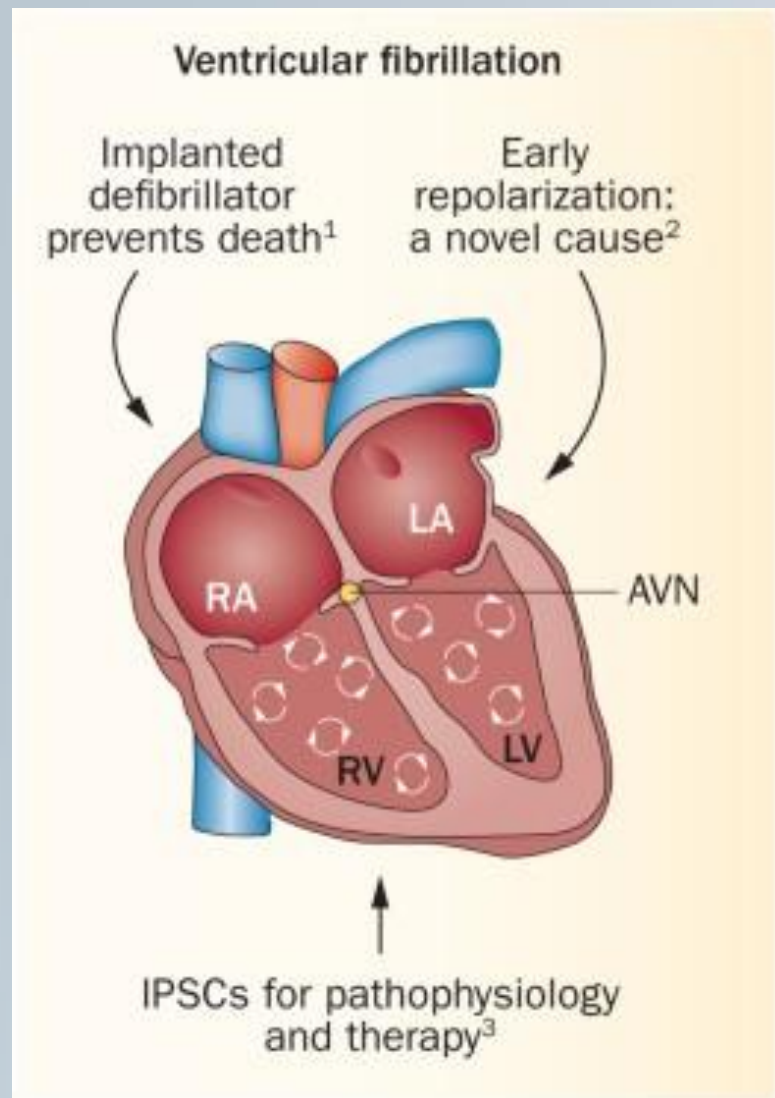
IV.Fibrillation : This is the contraction of the cardiac muscle fibres in a disorderly sequence. The chambers do not contract as a coordinated unit and the pumping action is disrupted.

Atrial fibrillation,

1. Contraction of the atria is uncoordinated and rapid, pumping is ineffective and stimulation of the AV node is disorderly.
2. Ventricular contraction becomes rapid and rhythm and force irregular; although an adequate cardiac output and blood pressure may be maintained, the pulse is irregular.
3. The causes of increased excitability and disorganised activity are not always clear but predisposing conditions include: ischaemic heart disease degenerative changes in the heart due to old age thyrotoxicosis, rheumatic heart disease.

Ventricular fibrillation

1. It is a medical emergency that will swiftly lead to death if untreated, because the chaotic electrical activity within the ventricular walls cannot coordinate effective pumping action (cardiac arrest).
2. Blood is not pumped from the heart into either the pulmonary or the systemic circulation.
3. No pulses can be felt, consciousness is lost and breathing stops.
4. The ECG shows an irregular chaotic trace with no recognisable wave pattern.



I. Heart block

1. Heart block occurs when normal impulse transmission is blocked or impaired.
2. A common form involves obstruction of impulse transmission through the AV node.
3. When the AV node is involved, the delay between atrial and ventricular contraction is increased. The severity depends on the extent of loss of stimulation of the AV node.
4. In *complete heart block*, ventricular contraction is entirely independent of impulses initiated by the SA node.
5. Freed from the normal pacing action of the SA node, the ventricles are driven by impulses generated by the pacemaker activity of the AV node, resulting in slow, regular ventricular contractions and a heart rate of about 30 to 40 beats per minute.
6. In this state the heart is unable to respond quickly to a sudden increase in demand by, for example, muscular exercise.
7. The most common causes are: acute ischaemic heart disease myocardial fibrosis following repeated infarctions or myocarditis drugs used to treat heart disease, e.g. digitalis, propranolol.
8. When heart block develops gradually there is some degree of adjustment in the body to reduced cardiac output but, if progressive, it eventually leads to death from cardiac failure and cerebral anoxia.

Heart Blocks

Constant P-R interval

Variable P-R Interval

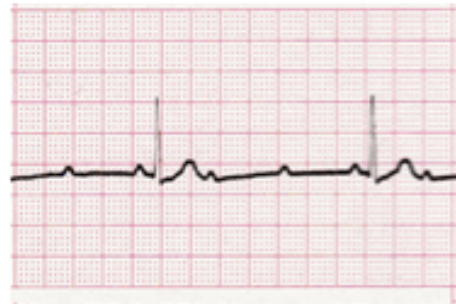
First Degree Heart Block



Second Degree Heart Block Type I



Second Degree Heart Block Type II

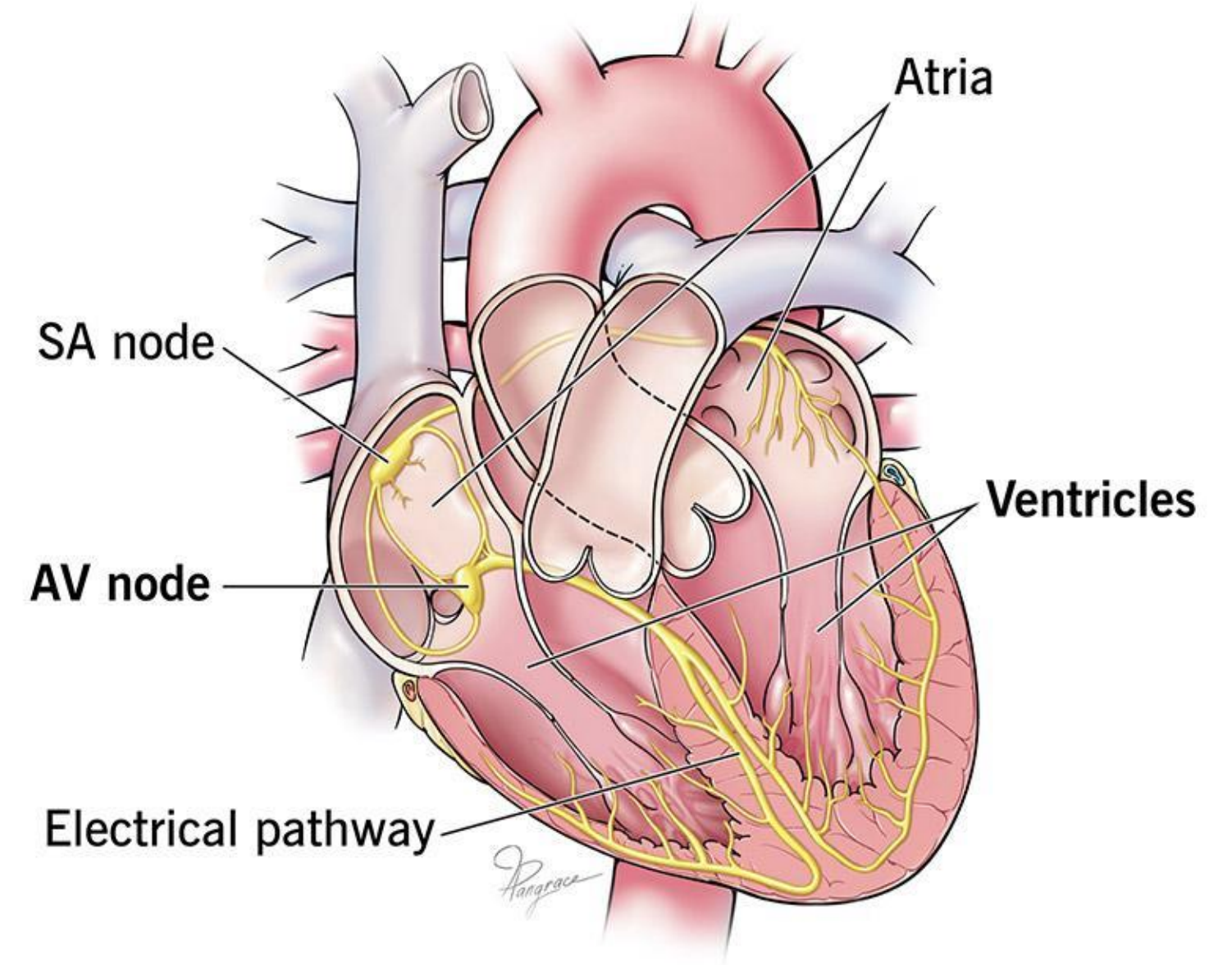


Third Degree Heart Block

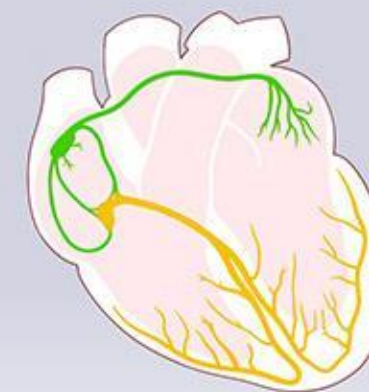


Heart block

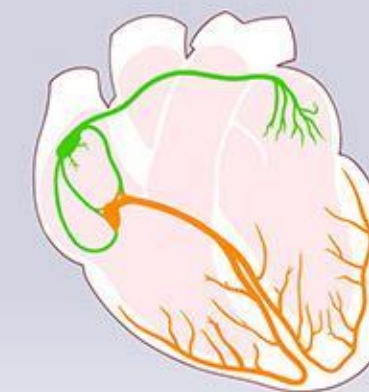
Issue with heartbeat signal reaching the ventricles



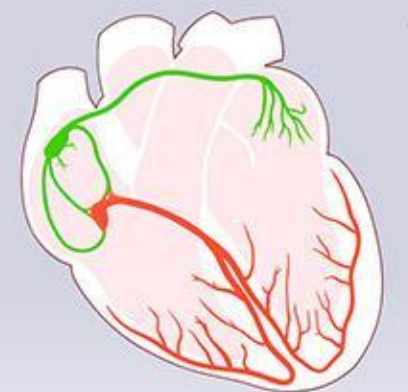
Types of heart block



First degree
Slow signal



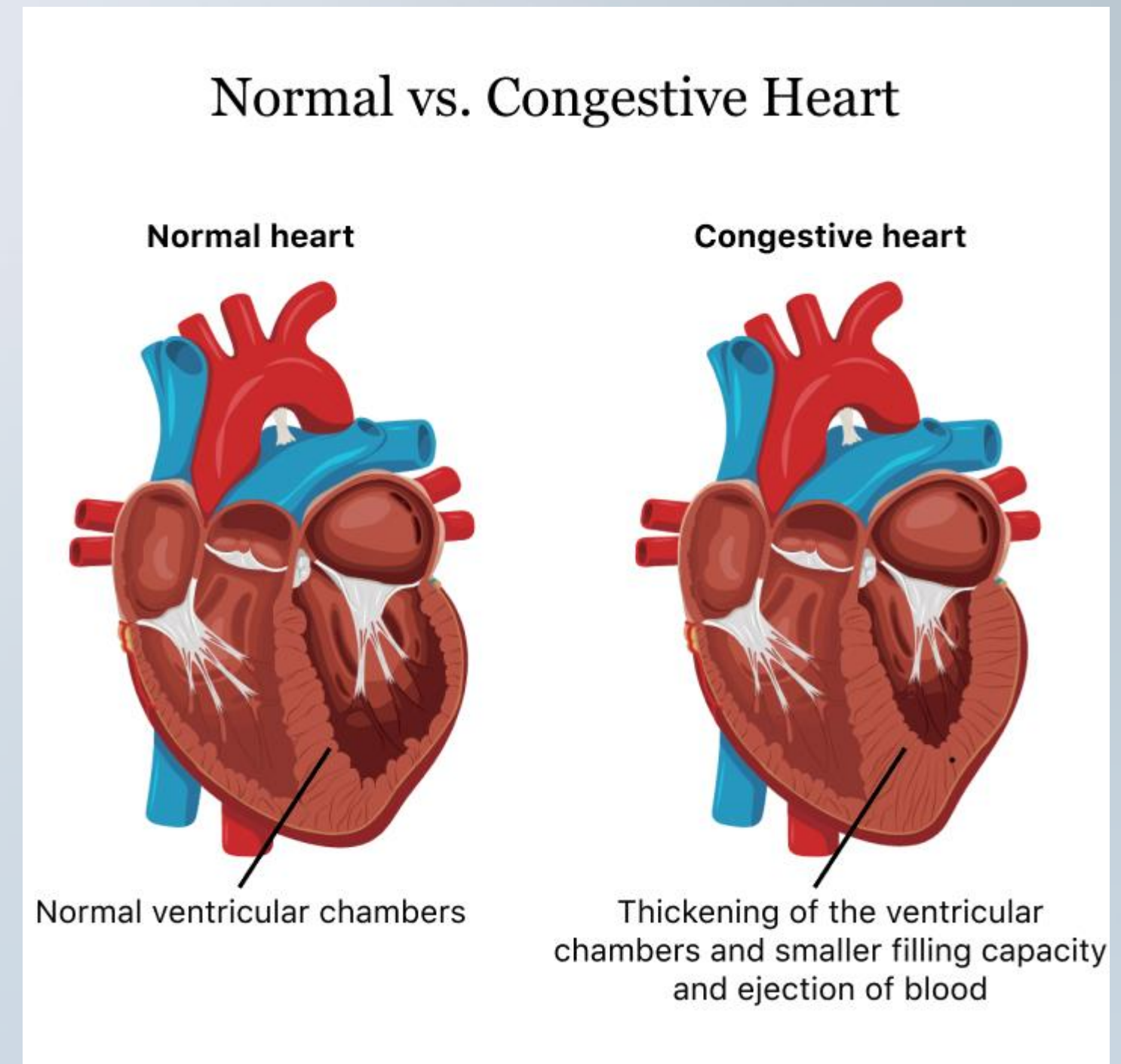
Second degree
Sporadic signal



Third degree
Blocked signal

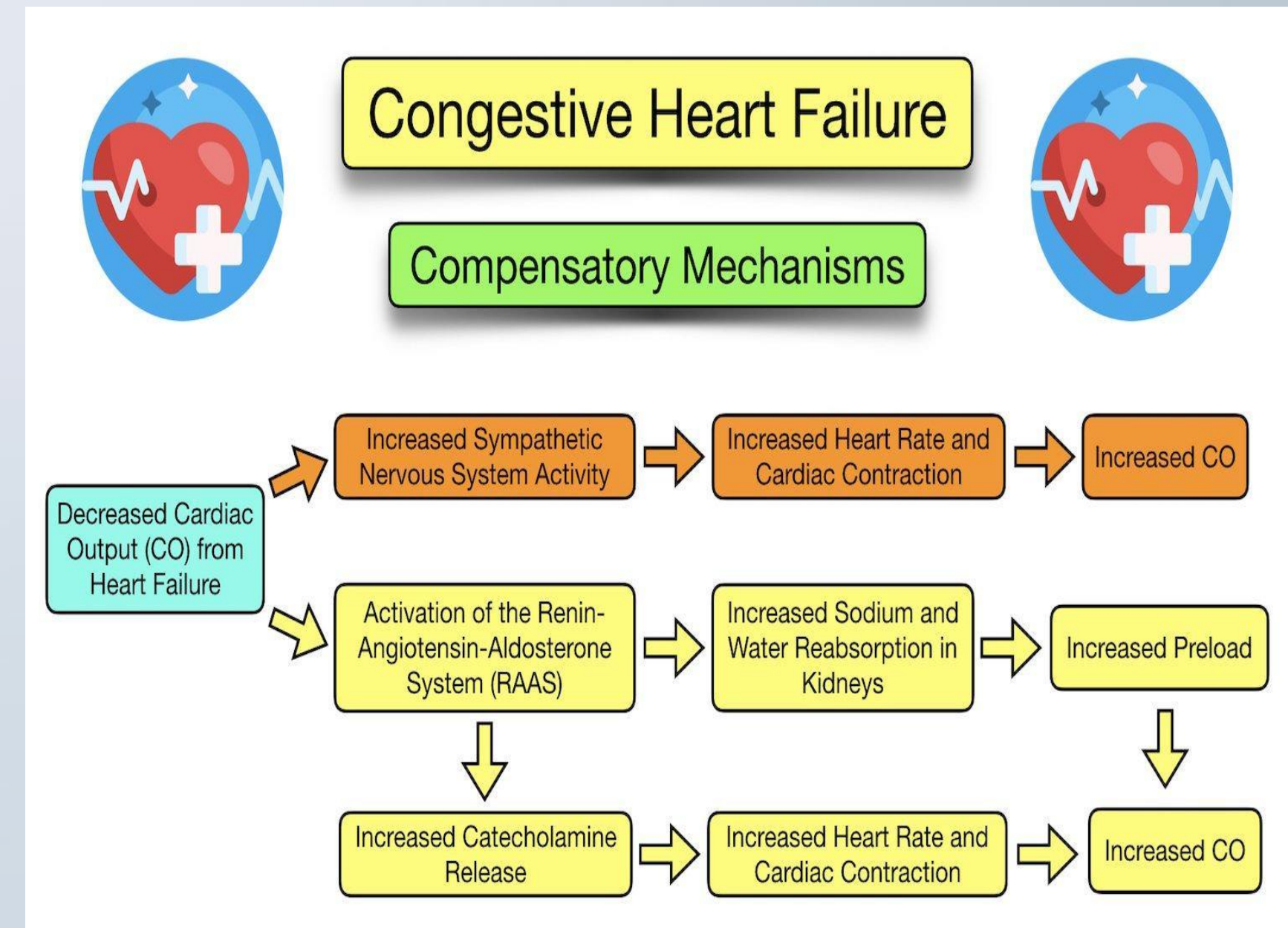
HEART (CARDIAC) FAILURE

1. A complex clinical syndrome that results from any structural or functional impairment of ventricular filling or ejection of blood, leading to inadequate blood supply to organs and tissues.
2. The heart is described as *failing* when the cardiac output is unable to maintain the circulation of sufficient blood to meet the needs of the body.
3. In mild cases, cardiac output is adequate at rest and becomes inadequate only when increased cardiac output is required, e.g. in exercise.
4. Heart failure may affect either side of the heart, but since both sides of the heart are part of one circuit, when one half of the pump begins to fail it frequently leads to increased strain on, and eventual failure of, the other side.
5. The main clinical manifestations depend on which side of the heart is most affected. Left ventricular failure is more common than right, because of the greater workload of the left ventricle.



Compensatory mechanisms in heart failure

1. When heart failure happens acutely, the body has little time to make compensatory changes, but if the heart fails over a period of time the following changes are likely to occur in an attempt to maintain cardiac output and tissue perfusion, especially of vital organs:
 - a. the cardiac muscle fibres enlarge and increase in number, which makes the walls of the chambers thicker
 - b. the heart chambers enlarge decreased renal blood flow activates the renin–angiotensin–aldosterone system, which leads to salt and water retention. this increases blood volume and cardiac workload.
 - c. The direct vasoconstrictor action of angiotensin 2 increases peripheral resistance and puts further strain on the failing heart.



Acute heart failure

1. A sudden decrease in output of blood from both ventricles causes acute reduction in the oxygen supply to the tissues.
2. Recovery from the acute phase may be followed by chronic failure, or death may occur due to anoxia of vital centres in the brain.
3. The commonest causes are: myocardial infarction, pulmonary embolism, blocking blood flow through the pulmonary circulation, severe cardiac arrhythmia,

Chronic heart failure

1. This develops gradually and in the early stages there may be no symptoms because compensatory changes occur as described above.
2. When further compensation is not possible there is a gradual decline in myocardial efficiency.
3. Underlying causes include degenerative heart changes with advancing age, and many chronic conditions, e.g. anaemia, lung disease, hypertension or cardiac disease.

Right-sided (congestive cardiac) failure

1. The right ventricle fails when the pressure developed within it by the contracting myocardium is not enough to push blood through the lungs.
2. When compensation has reached its limit, and the ventricle can no longer empty completely, the right atrium and venae cavae become congested with blood and this is followed by congestion throughout the venous system.
3. The organs affected first are the liver, spleen and kidneys.
4. *Oedema* of the limbs and *ascites* (excess fluid in the peritoneal cavity) usually follow.
5. This problem may be caused by increased vascular resistance in the lungs or weakness of the myocardium.

Left-sided (left ventricular) failure

1. This occurs when the pressure developed in the left ventricle by the contracting myocardium is not enough to force blood into the aorta and the ventricle cannot then pump out all the blood it receives.
2. Causes include ischaemic heart disease, which reduces the efficiency of the myocardium, and hypertension, when the heart's workload is increased because of raised systemic resistance.
3. Failure of the left ventricle leads to dilation of the atrium and an increase in pulmonary blood pressure.
4. This is followed by a rise in the blood pressure in the right side of the heart and eventually systemic venous congestion.
5. Congestion in the lungs leads to pulmonary oedema and dyspnoea, often most severe at night.
6. This paroxysmal *nocturnal dyspnoea* may be due to raised blood volume as fluid from peripheral oedema is reabsorbed when the patient slips down in bed during sleep.

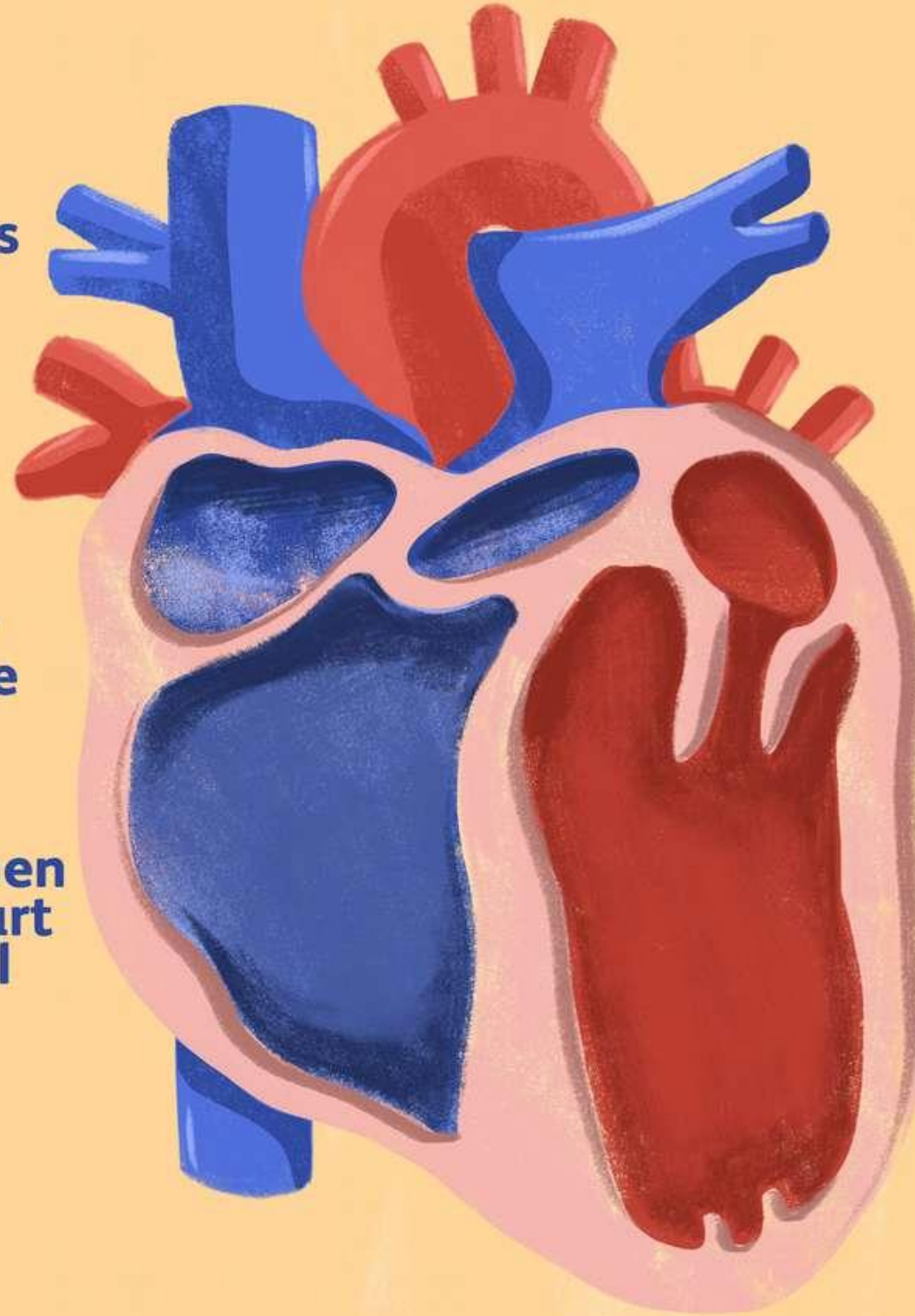
Left-Sided vs. Right-Sided Heart Failure

Right-Sided

Heart loses some of its ability to move oxygen-depleted blood to the lungs to pick up new oxygen

Most often caused by left-sided heart failure

Can also occur even when the left side of the heart is apparently normal



Left-Sided

Most common type of heart failure

Heart loses some of its ability to pump blood out to your body after it's been re-oxygenated

Usually caused by coronary artery disease