



**GUARDIAN TEST PREP**

**Cardiology**

**Workbook**

# CARDIAC ANATOMY

The structure of the heart is what allows the heart to function effectively and efficiently. Understanding the anatomy of the heart is the basis for understanding how the heart functions in pumping blood throughout the entire body to deliver oxygen to the tissues of the body. This then allows us to make sense of what is occurring when the heart is not functioning properly. This module covers the basic anatomy of the heart including its chambers, valves and blood supply.

## Learning objectives

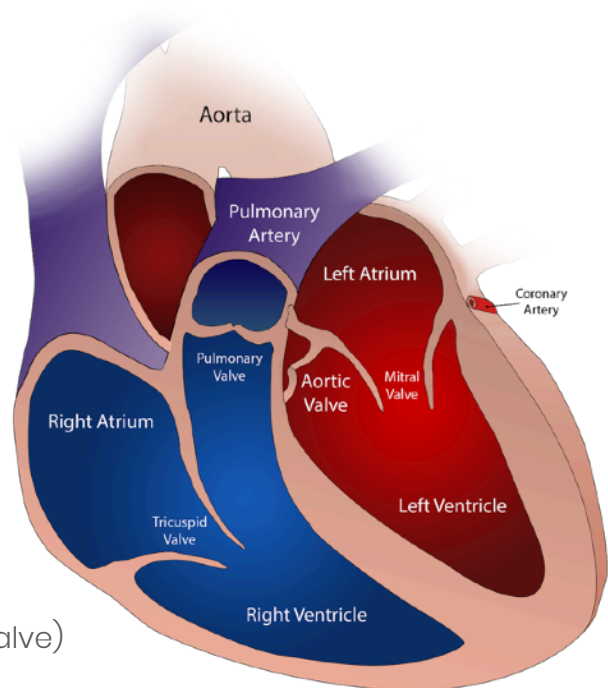
**By the end of this module, you should be able to...**

- ▶ Identify the four chambers of the heart as well as the valves that connect them
- ▶ Follow the route the blood takes through the heart
- ▶ Identify and name the types of vessels that carry blood to and from the heart
- ▶ Identify the layers of the heart muscle tissue
- ▶ List the blood vessels that carry oxygen to the heart muscle itself and what parts of the heart they supply

# Heart Chamber and Valves

## Chambers

1. Right atria
2. Left atria
3. Right ventricle
4. Left ventricle

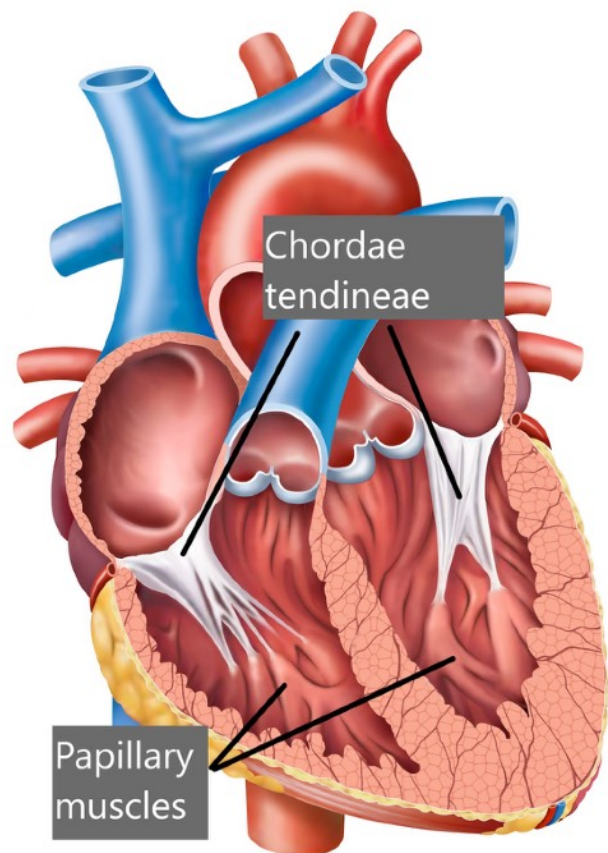


## Valves

1. Atrioventricular valves
  - ▶ Tricuspid
  - ▶ Bicuspid (also called \_\_\_\_\_ valve)
2. Semilunar valves
  - ▶ Aortic
  - ▶ Pulmonic

## Valve supporting structure

1. Chordae tendineae
  - ▶ Tendons attached to valves that pull valve open
2. Papillary muscles
  - ▶ Contract to pull on chordae tendineae and open valve



# Cardiac Blood Flow

## Step 1

Deoxygenated blood returns to heart from the superior and inferior vena cava and empties into the right atria.

## Step 2

Atria contract and blood passes through the \_\_\_\_\_ valve into the right ventricle.

## Step 3

Ventricle contract and blood passes through the \_\_\_\_\_ valve into the pulmonary artery which travels to the lungs where the blood picks up oxygen.

## Step 4

Oxygenated blood return to heart through the pulmonary veins and dumps into the left atria.

## Step 5

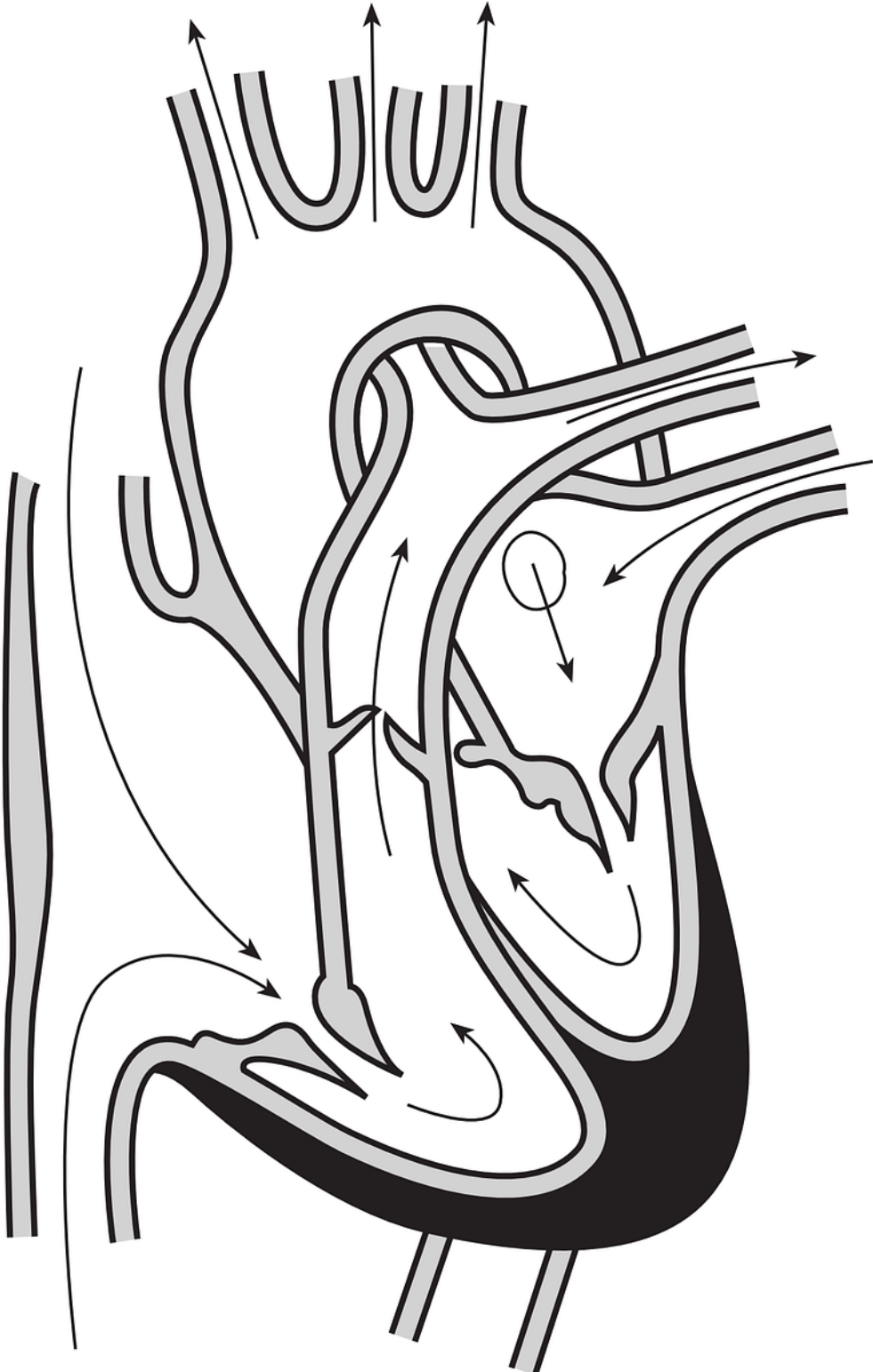
Atria contract and blood passes through the \_\_\_\_\_ valve into the left ventricle.

## Step 6

Ventricles contract and blood passes through the \_\_\_\_\_ valve into the aorta which travels to the body to deliver oxygen to tissues.

(Keep in mind that both the right and left atria contract together, as do the right and left ventricle. Because of this Steps 2 and 5 as well as Steps 3 and 6 are occurring at the same time)

Label the below image with the structures of the heart as you follow the blood through the chambers and valves.



# Blood vessels

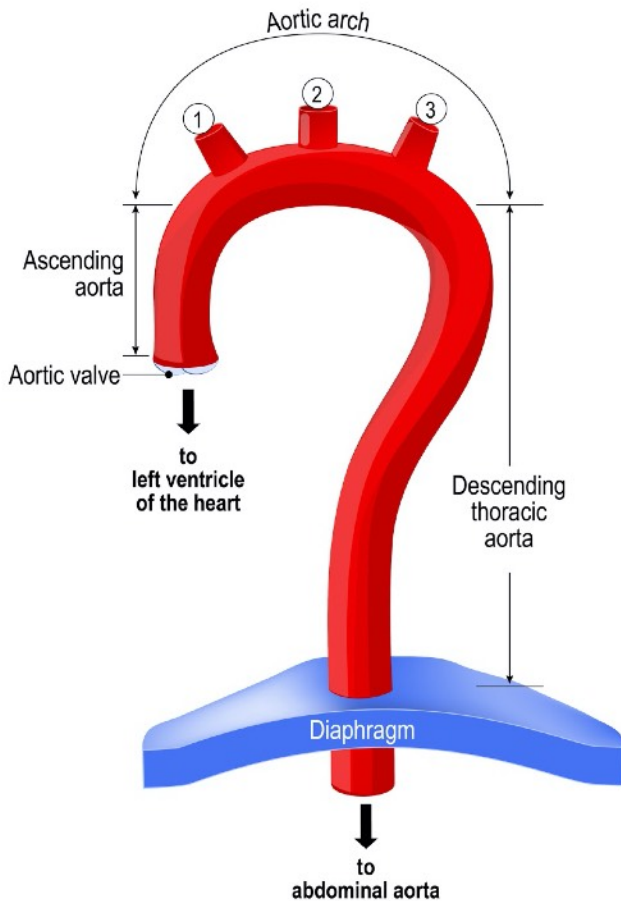
## Aorta

**Ascending aorta** - Part of aorta from where it exits the top of the heart to the aortic arch

**Thoracic aorta** - Part of aorta from the aortic arch to the diaphragm

**Abdominal aorta** - Part of aorta from the diaphragm to where it bifurcates into the left and right femoral arteries

# AORTA



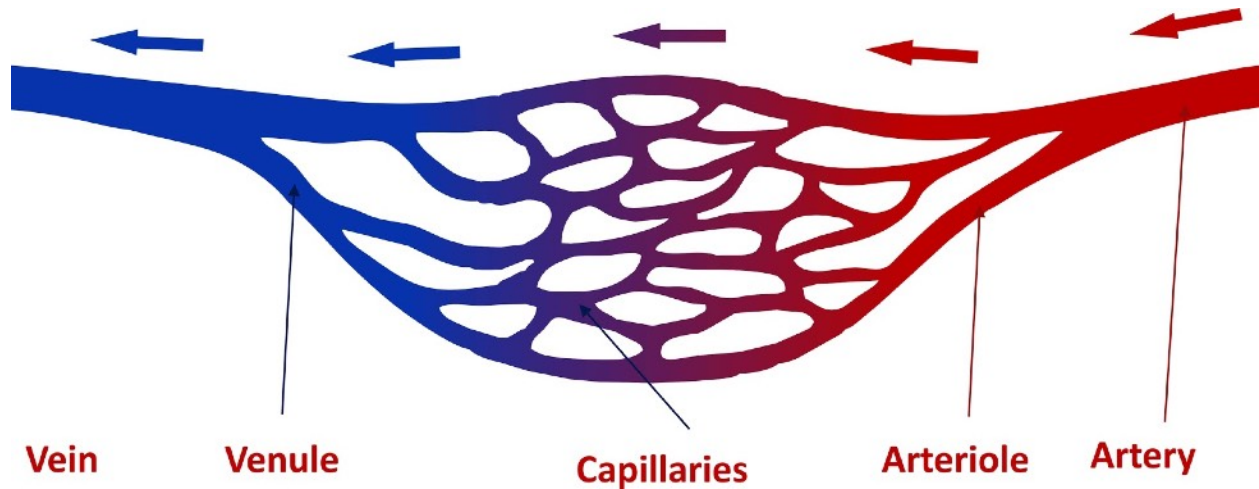
Arteries carry blood \_\_\_\_\_ from the heart.

Veins carry blood \_\_\_\_\_ the heart.

1.Brachiocephalic trunk 2.Left common carotid artery 3.Left subclavian artery

## Blood flow through vessels

Aorta -----> Arteries -----> Arterioles -----> Capillaries -----> Venules -----> Veins -----> Superior and inferior vena cava



- ▶ Capillaries are so small that only one red blood cell can pass at a time.
- ▶ This allows for gas exchange to occur in the tissue capillaries where oxygen is delivered and carbon dioxide and other waste products are picked up.

Question 1:

What ARTERY carries deoxygenated blood away from the heart?

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Question 2:

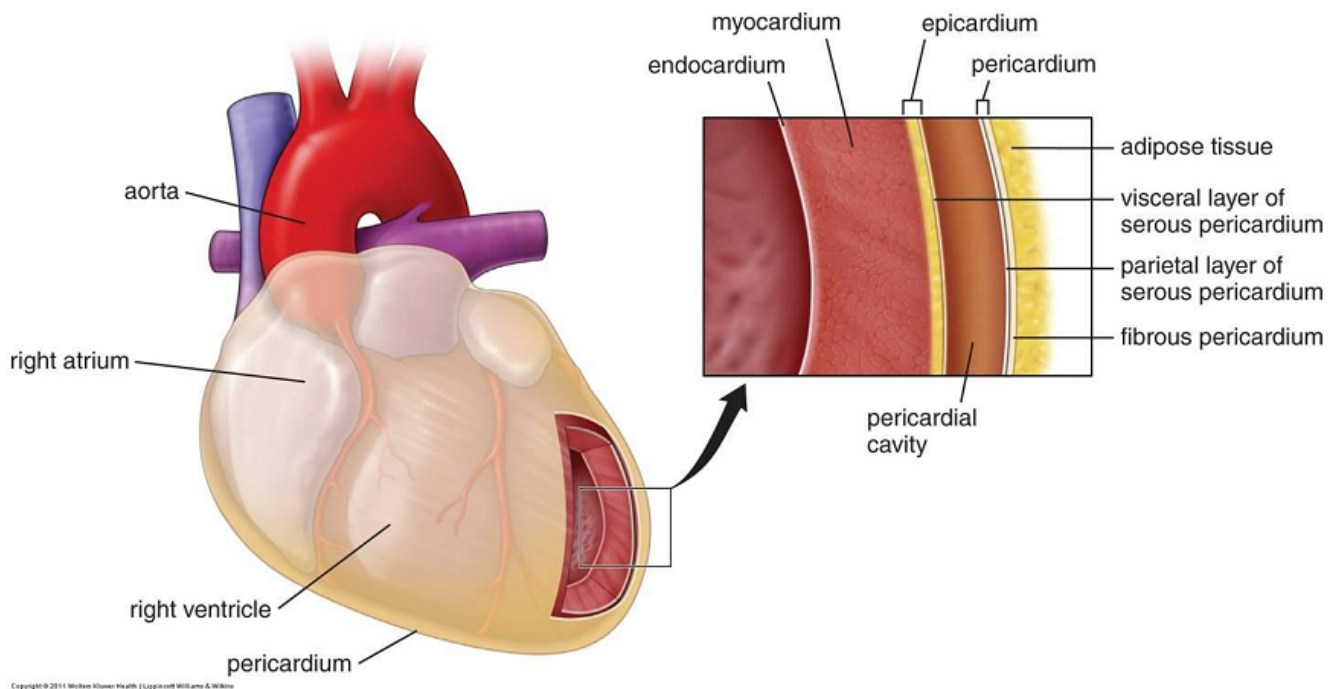
What VEIN carries oxygenated blood to the heart?

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# Layers of Heart Tissue

## Heart Muscle

1. Endocardium
  - ▶ Inner-most layer
2. Myocardium
  - ▶ Middle, muscular layer
3. Pericardium
  - ▶ Visceral pericardium - laying on heart tissue
  - ▶ Pericardial space/sac - space in between two pericardial tissue layers
  - ▶ Parietal pericardium - attached to chest wall in mediastinum





# Myocardial Blood Supply

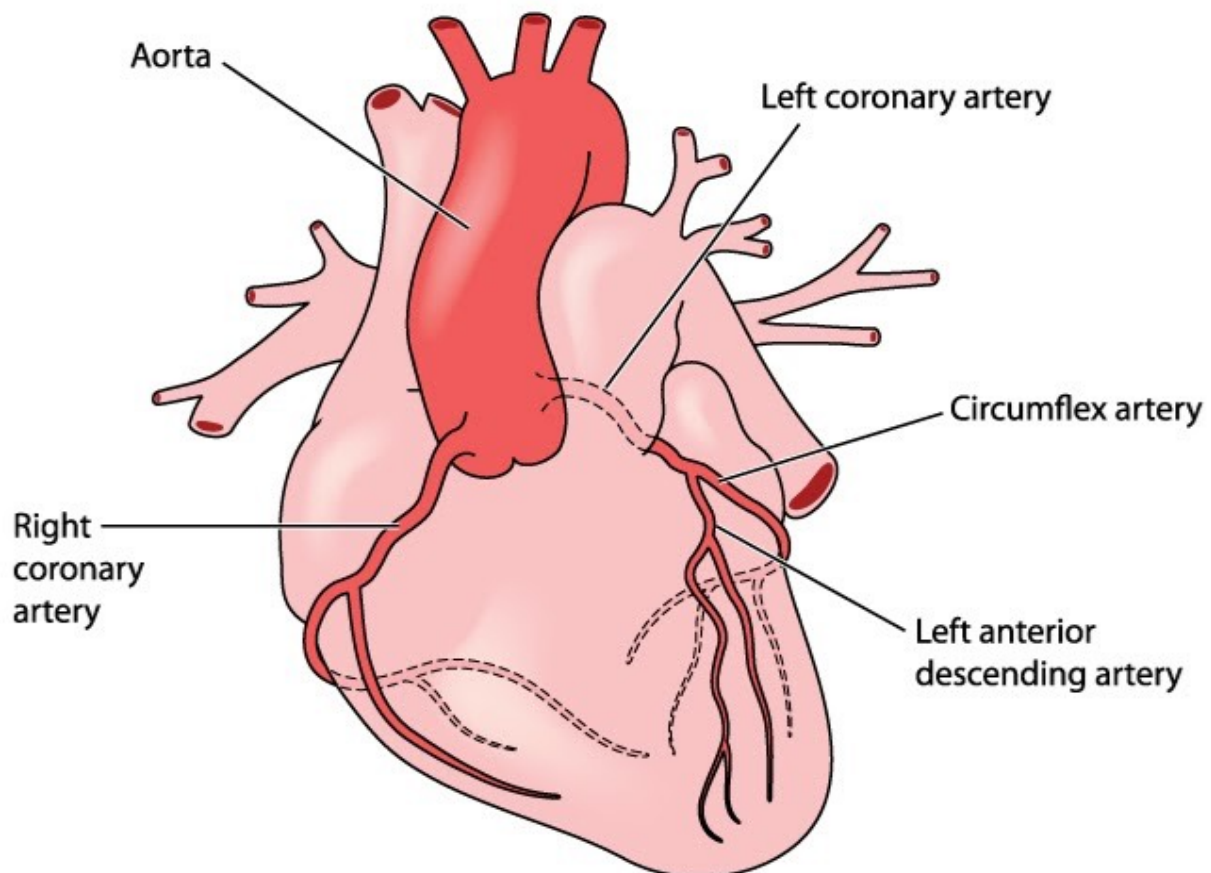
Because the heart is a muscle, it also needs its own blood supply in order for the cells of the heart to receive oxygen and survive.

This oxygen supply is delivered to the cells of the heart through the coronary arteries.

## Coronary Arteries

### 1. Left coronary artery

- A. Left anterior descending artery (LAD)
  - ▶ Supplies anterior left and right ventricles
- B. Circumflex artery
  - ▶ supplies posterior left atria and posterior left ventricle



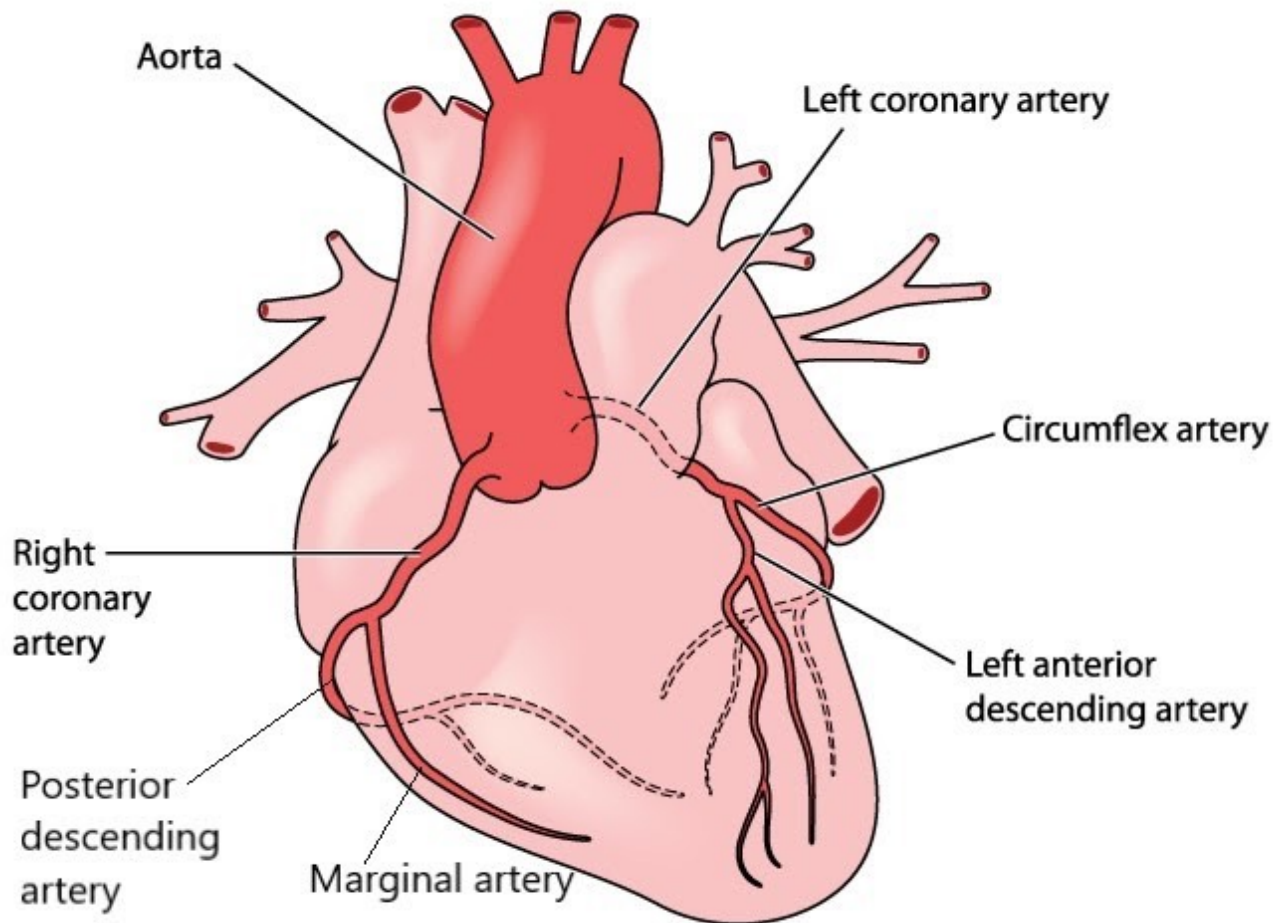
## 2. Right coronary artery

### A. Posterior descending artery

- ▶ Supplies posterior left and right ventricles

### B. Marginal artery

- ▶ Supplies anterior right atrium and right ventricle



# HIGH YIELD REVIEW

- ▶ Atrioventricular valves: tricuspid and bicuspid
- ▶ Bicuspid valve also called mitral valve
- ▶ Semilunar valves: aortic and pulmonic
- ▶ Valves open when papillary muscles contract and pull on the chordae tendineae
- ▶ Arteries carry blood away from the heart and veins carry blood to the heart.  
(pulmonary artery carries deoxygenated blood and pulmonary veins carry oxygenated blood)
- ▶ The diaphragm is the point where the descending thoracic aorta becomes the descending abdominal aorta
- ▶ Left coronary artery branches into LAD and circumflex
- ▶ Right coronary artery branches into posterior descending and marginal arteries

# CARDIAC CYCLE / FUNCTION

The heart functions to pump blood to the entire body in a miraculously efficient and effective way 24/7, 365 days a year for almost 100 years in some people. In this module you will learn how the blood moves through the heart during the cardiac cycle and the phases of contraction and relaxation that take place. You will also discover how the heart's ability to pump blood depends on the pressure outside and inside the cardiac system and how to measure these. The key to understanding how to resuscitate an unhealthy heart is to first understand how the heart was meant to function.

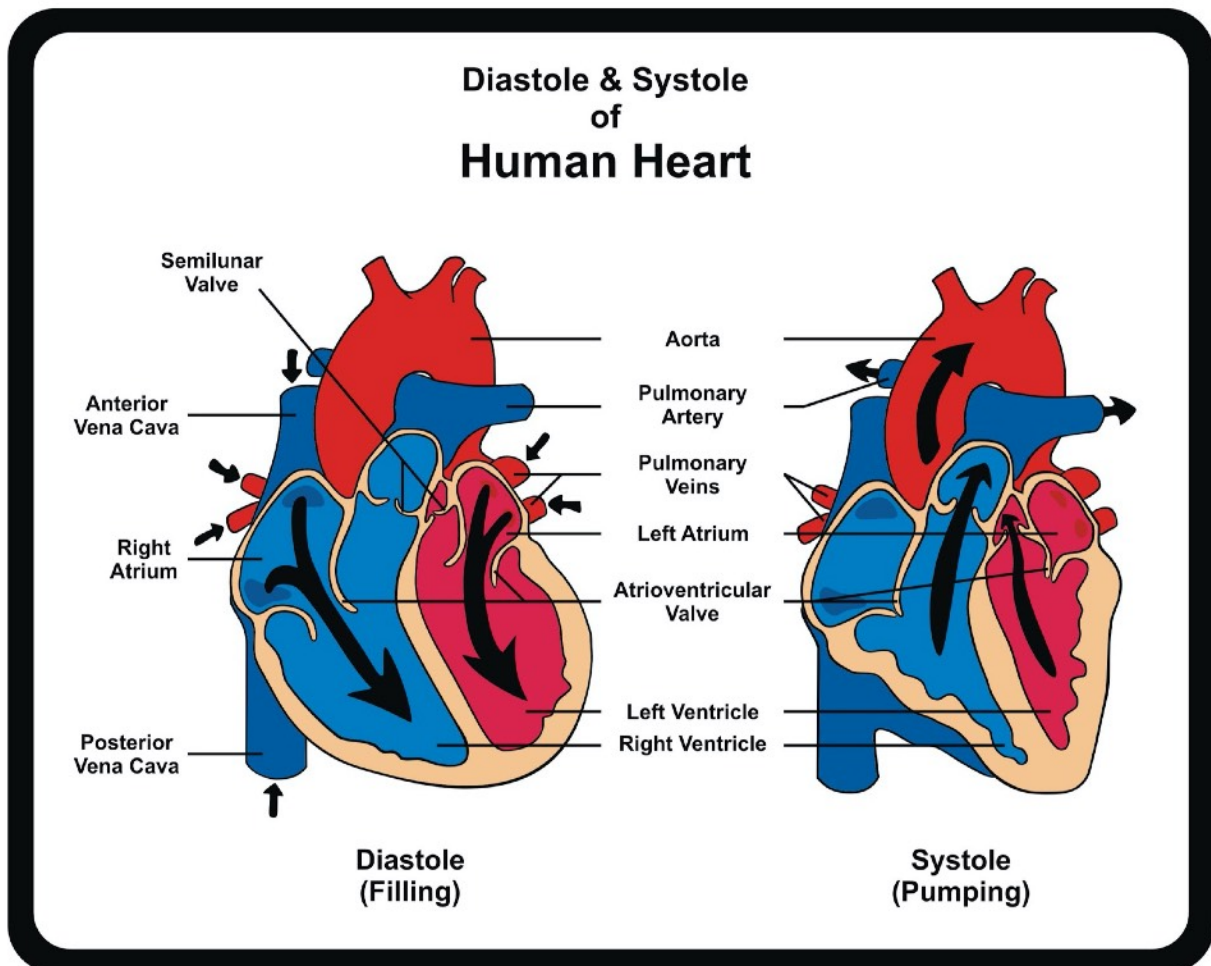
## Learning objectives

**By the end of this module, you should be able to...**

- ▶ Understand how the heart pumps blood through systole and diastole
- ▶ Identify and list the steps of the cardiac cycle
- ▶ Define stroke volume and understand how it affects cardiac output
- ▶ List the factors that can affect stroke volume
- ▶ Understand and define preload and afterload

# Systole and Diastole

- ▶ The cardiac cycle is broken up into systole and diastole
- ▶ Systole = contraction
- ▶ Diastole = \_\_\_\_\_



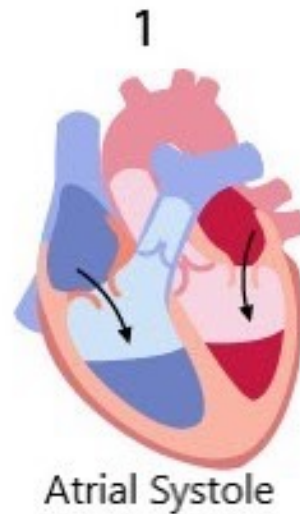
- ▶ The above image shows ventricular diastole and systole, however both the atria and the ventricles go through contraction and relaxation phases.
- ▶ As a general rule, when the atria are contracting (atrial systole) the ventricles are relaxing (ventricular diastole) and vice versa.

# 5 Steps of Cardiac Cycle

1. Atrial systole
2. Isovolumetric contraction
3. Ejection
4. Isovolumetric relaxation
5. Rapid filling

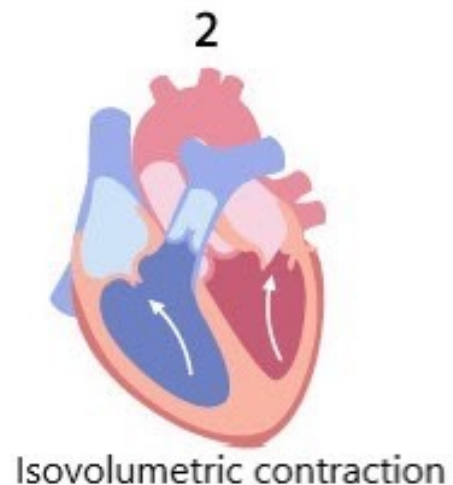
## 1. Atrial systole

- ▶ Atria contract to push blood into ventricles
- ▶ During this time the ventricles are in diastole
- ▶ Tricuspid and Bicuspid valves are open



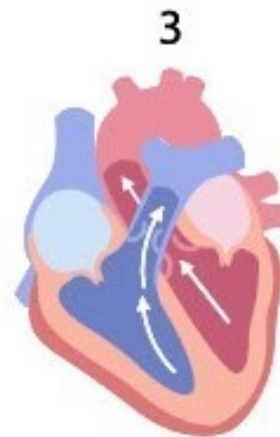
## 2. Isovolumetric contraction

- ▶ Ventricles begin contracting, but no blood is pushed out of the ventricles
- ▶ Pressure builds up as the ventricular muscles begin to contract and this pressure closes the tricuspid and bicuspid valves (S1 heart sound)



### 3. Ejection

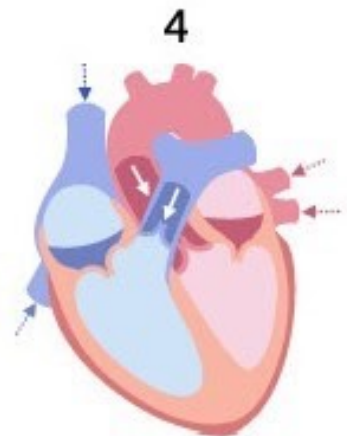
- ▶ Aortic and pulmonic valves open
- ▶ Ventricles contract and push blood into the pulmonary artery and aorta



Ejection

### 4. Isovolumetric relaxation

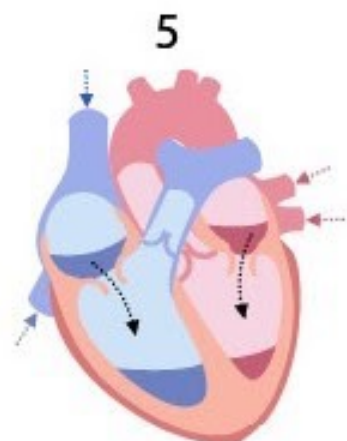
- ▶ This is the beginning of ventricular diastole
- ▶ The ventricles begin to relax but the volume of the ventricles does not change
- ▶ Aortic and pulmonic valves close (S2 heart sound)



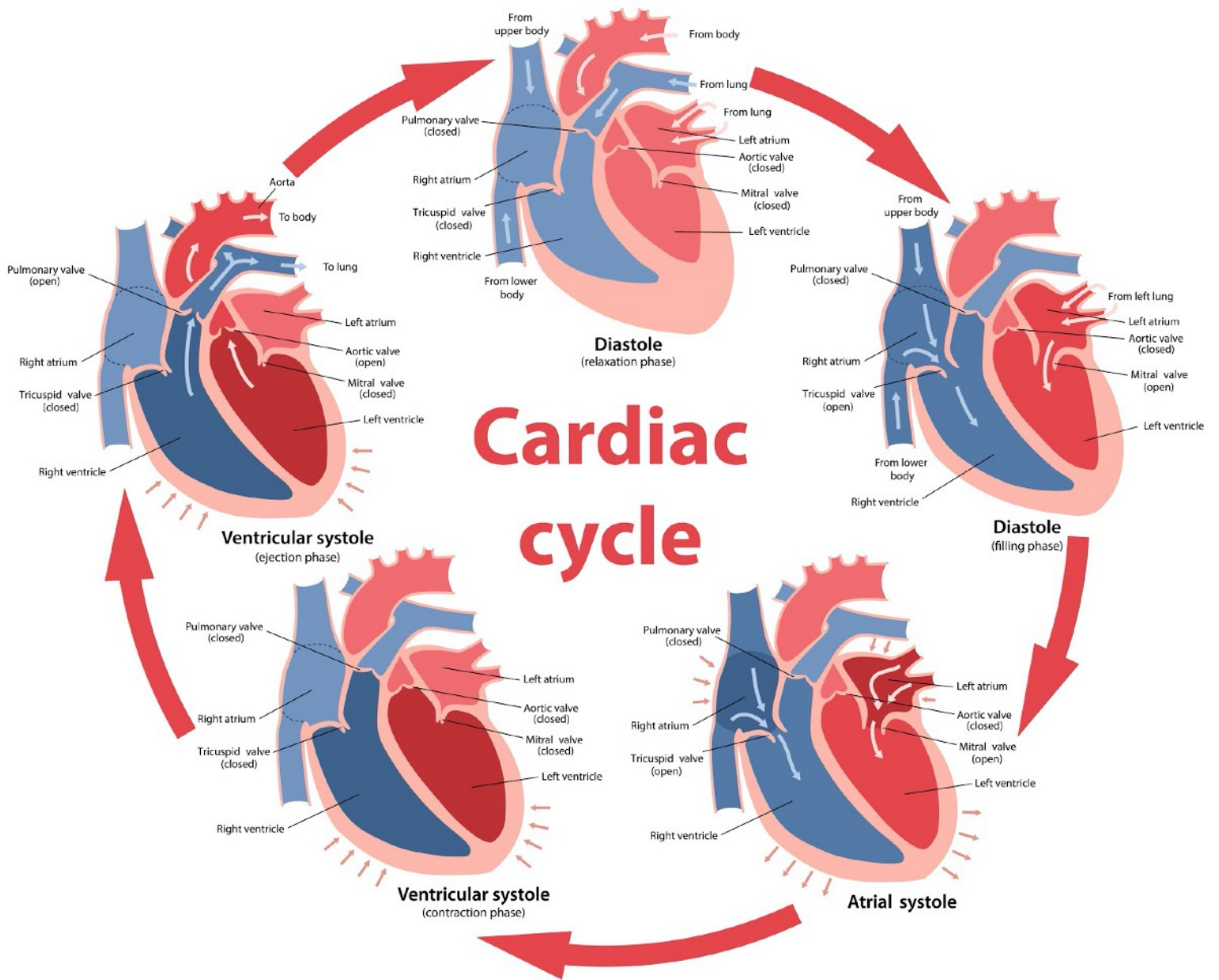
Isovolumetric relaxation

### 5. Rapid filling

- ▶ The tricuspid and bicuspid valves open and blood pours into the ventricles passively
- ▶ Next, the atrial will contract and actively push blood into the ventricles which will be atrial systole and mark the beginning of this cycle again



Rapid filling





# Cardiac Output

Stroke volume (SV) = the amount of blood ejected from the heart during each contraction of the ventricles

SV usually equals \_\_\_\_\_ ml

Heart rate (HR) = the amount of times the heart beats in 60 seconds (one minute)

HR usually equals 60-100 bpm

$$\text{Cardiac output} = \text{SV} \times \text{HR}$$

Cardiac output (CO) = the amount of blood ejected from the heart every minute

CO usually equals about \_\_\_\_\_ L

$$\text{SV} \times \text{HR} = \text{CO}$$

$$70 \text{ ml} \times 70 \text{ bpm} = 4,900 \text{ ml/min} = 4.9 \text{ L/min}$$

- ▶ Anything that affects Stroke volume or Heart rate will affect Cardiac output
- ▶ If SV or HR increase, CO will increase
- ▶ If SV or HR decreases, CO will decrease

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# Stroke Volume

## Factors affecting stroke volume

- ▶ Preload
- ▶ Afterload
- ▶ Contractility

**Preload** = amount of pressure in ventricle before contraction

- ▶ The amount of pressure in the ventricle before contraction depends on the amount of volume in the ventricle before contraction.
- ▶ This means preload can also refer to the amount of blood returning to the heart and filling the ventricle before contraction.

**Afterload** = amount of pressure in vascular system the ventricle must push against

- ▶ This pressure is dependent on how resistant the blood vessels are to the flow of blood, also called systemic vascular resistance

**Contractility** = ability of the heart muscle to contract

- ▶ Anything that weakens or thickens the heart muscle will affect its ability to contract.
- ▶ As heart thickens over time due to high blood pressure and cholesterol, hypertrophy can occur leading to decreased contractility.

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## HIGH YIELD REVIEW

- ▶ Systole = contraction, diastole = relaxation
- ▶ Cardiac cycle includes atrial systole, isovolumetric contraction, ejection, isovolumetric relaxation and rapid filling
- ▶ S1 heart sound is atrioventricular valves closing during isovolumetric contraction
- ▶ S2 heart sound is semilunar valves closing during isovolumetric relaxation
- ▶  $CO = SV \times HR$
- ▶ SV affected by preload, afterload and contractility
- ▶ Afterload is systemic vascular resistance

# HEART FAILURE

According to the American Heart Association, 6 million Americans are living with heart failure and 900,000 new cases are diagnosed each year. If this is the case, as a first responder you will definitely come in contact with patients suffering from heart failure as well as its complications. Knowing the pathophysiology behind heart failure will help you understand how best to treat and support these patients.

## Learning objectives

**By the end of this module, you should be able to...**

- ▶ Define ejection fraction and describe what disease states affect it
- ▶ Understand the difference between right and left sided heart failure and how they relate to one another
- ▶ Describe the main causes of isolated right sided heart failure
- ▶ Recognize the signs and symptoms of congestive heart failure
- ▶ Describe the different treatment modalities for heart failure and their mechanisms of action

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## Ejection Fraction

There are varying degrees of heart failure ranging from mild to severe.

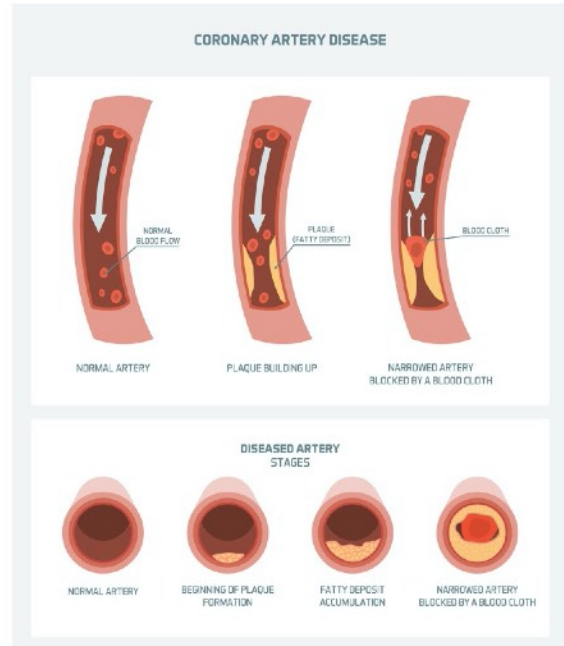
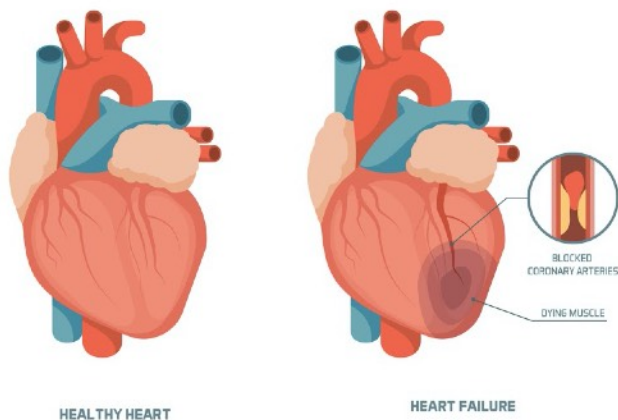
The degree of heart failure is based on a patient's ejection fraction.

**Ejection fraction** = the percentage of blood ejected from the left ventricle during systole.

Normal Ejection fraction in a health heart is between \_\_\_\_\_% and \_\_\_\_\_%.

## Disease leading to decreased ejection fraction

1. Hypertension
  2. Coronary artery disease
  3. Myocardial infarction
- ▶ Hypertension - requires heart to pump against high afterload which over time weakens the heart muscle.
  - ▶ Coronary artery disease - decreased blood flow to the heart requires the heart better harder and faster due to lack of oxygen leading to weakening of the heart tissue.
  - ▶ Myocardial infarction - death of tissue due to ischemia leads to areas of weakened heart muscle.

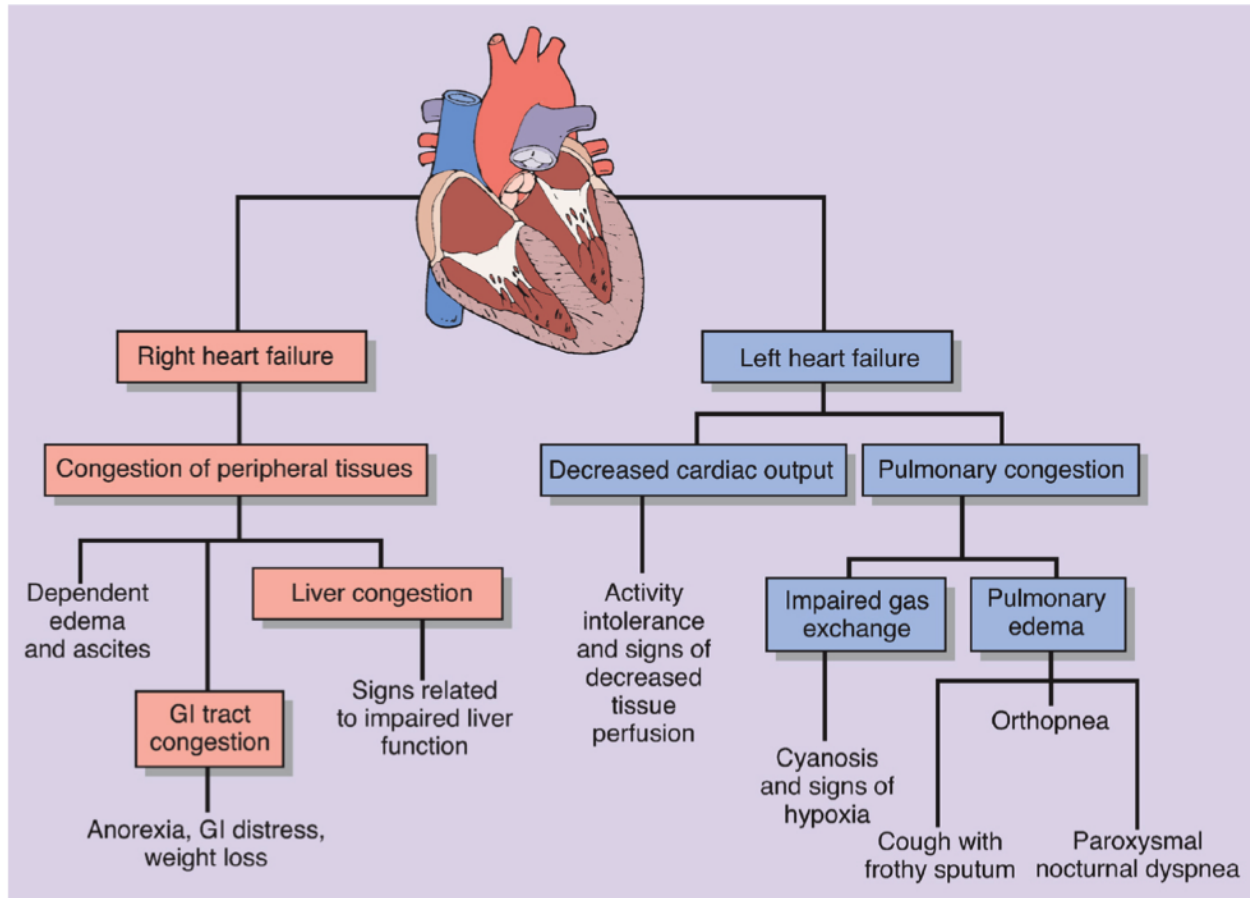


## Types of Heart Failure

1. Left sided heart failure
  - ▶ Ejection fraction typically refers to ejection fraction of left ventricle, however, heart failure can involve both sides of the heart, not just the left side
2. Right sided heart failure

The most common cause of right sided heart failure is \_\_\_\_\_

- ▶ Weakening of the left ventricle leads to back up of fluid into the right side of the heart. Over time, this increased pressure and fluid weakens the right side of the heart as well.



## Isolated Right Heart Failure

- ▶ Though right sided heart failure is usually secondary to left sided heart failure, there are circumstances where right sided heart failure can be found on its own.

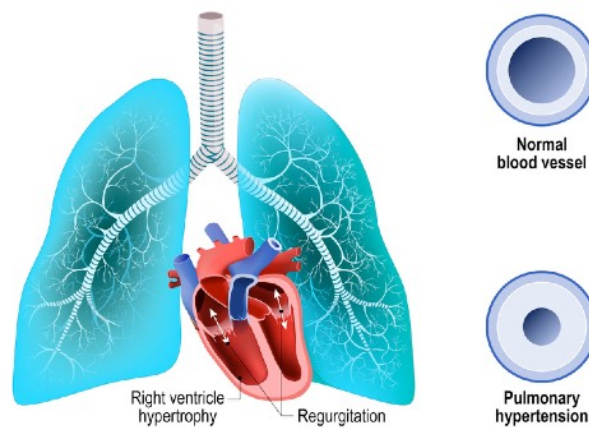
1. COPD
2. Pulmonary embolism
3. Pulmonary hypertension

**COPD** – in COPD, inflammation in the lungs causes increased pressures leading to right sided heart failure.

**Pulmonary embolism** – a blood clot in the vessels of the lungs creates an increased pressure that the right heart has to push against leading to right sided heart failure

**Pulmonary hypertension** – hypertension that develops in the lung vessels leading to increased pressures and right sided heart failure.

## PULMONARY HYPERTENSION



Right sided heart failure is also sometimes referred to as \_\_\_\_\_

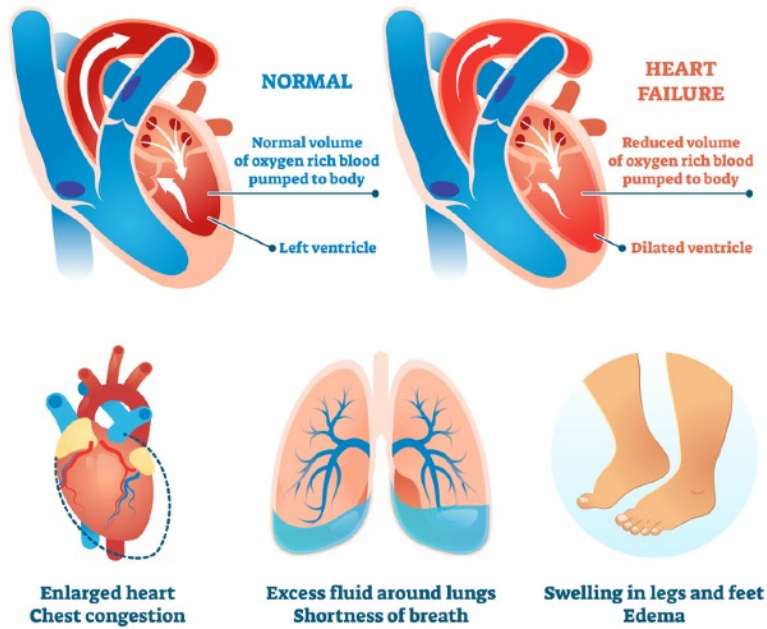
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## Congestive Heart Failure

### Signs and Symptoms

1. Hx of HTN, CAD, MI
2. Edema
3. SOB
4. Cyanosis

# CONGESTIVE HEART FAILURE



## Treatment

1. Supplemental O<sub>2</sub>
2. Consider CPAP
3. Lasix
4. Nitroglycerin and beta blockers

# HIGH YIELD REVIEW

- ▶ Ejection fraction = percentage of blood ejected from left ventricle during contraction
- ▶ Normal ejection fraction is between 55% and 70%
- ▶ HTN, CAD and MI are main contributors to heart failure
- ▶ Most common cause of right sided heart failure is left sided heart failure
- ▶ Isolated right sided heart failure typically caused by COPD, PE and pulmonary hypertension

# ACUTE CORONARY SYNDROMES

Because of the prevalence of cardiovascular disease in America, most of our population is at an increased risk for Heart attack and stroke. In this module we concentrate on acute coronary syndromes. These are syndromes causing lack of O<sub>2</sub> to the heart muscle, preventing its function, and in a worse case scenario, leading to heart attack, and even death. Unstable angina, myocardial ischemia and myocardial infarction fall into the category of acute coronary syndromes.

## Learning objectives

**By the end of this module, you should be able to...**

- ▶ Understand the pathophysiology behind coronary artery disease
- ▶ Identify the difference between stable and unstable angina
- ▶ Identify the difference between ischemia and infarction
- ▶ Understand the different treatments utilized in acute coronary syndrome and their mechanisms of action

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## Acute Coronary Syndrome

### Pathophysiology

ACS involves heart muscle being starved of oxygen. This is typically secondary to plaque build up and thrombus formation in the coronary vessels.

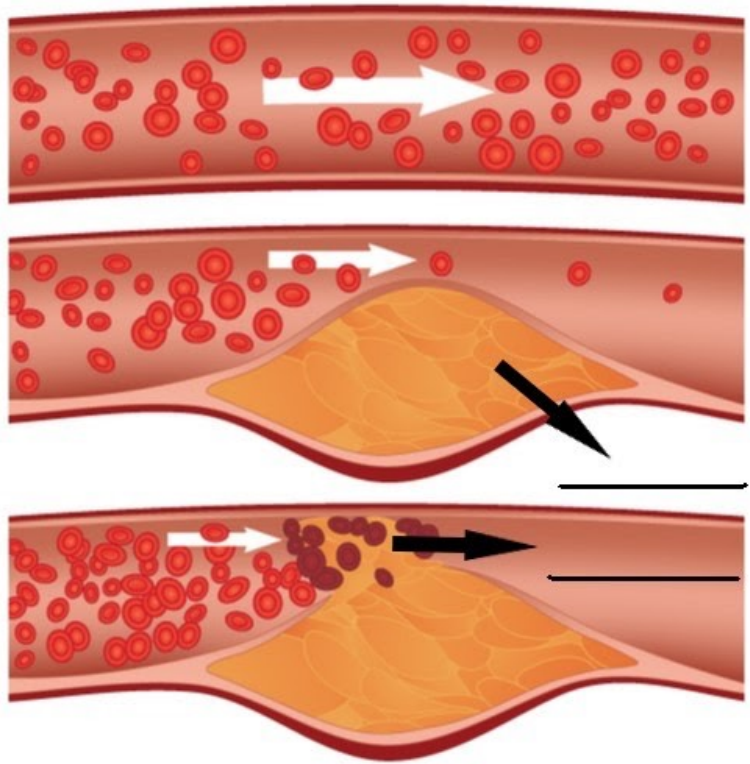


## Types of ACS

- ▶ Stable Angina
- ▶ Unstable Angina
- ▶ Myocardial infarction

## Signs and Symptoms

**CHEST PAIN** - there is no way of clinically determining whether chest pain is benign or represents stable angina, unstable angina or myocardial infarction. Because of this, treatment is the same for anyone experiencing chest pain.

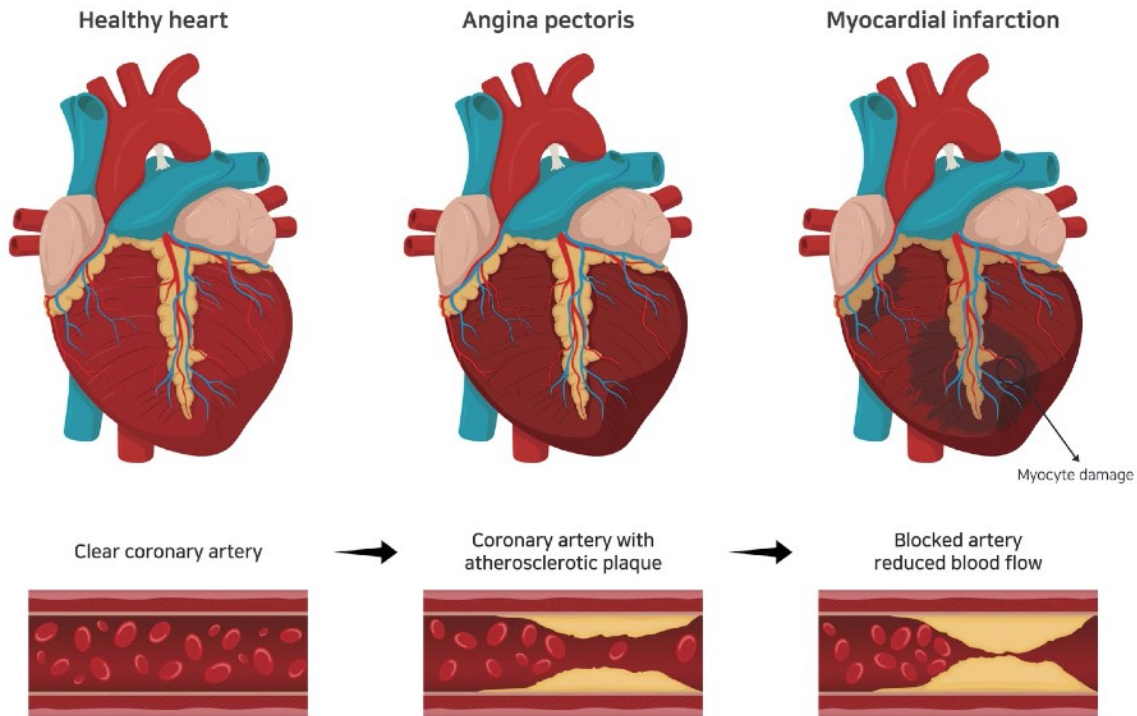


## Ischemia vs Infarction

Ischemia = \_\_\_\_\_

Infarction = death of tissue

- ▶ Plaque build up and thrombus formation leads to narrowing vessels, decreased perfusion and lack of oxygen. This is ischemia.
- ▶ When cells and tissues die, we call this infarction. Even if the vessel is opened back up and blood flow returns, that tissue will not function.



## Stable Angina vs Unstable Angina

### Stable Angina

- ▶ Occurs with \_\_\_\_\_
- ▶ Goes away with rest
- ▶ Perfusion is unable to keep up with increased O<sub>2</sub> demand because of vessel blockage

### Unstable Angina

- ▶ Occurs at \_\_\_\_\_
- ▶ Does not go away and leads to increased ischemia
- ▶ Blockage is severe enough that there is decreased perfusion even with O<sub>2</sub> demand is normal.

## ACS Treatment

Chest pain treatment is the same for stable angina, unstable angina and myocardial infarction since we don't know the underlying pathology on physical exam and assessment alone.

1. **Supplemental O<sub>2</sub>**

Provides more oxygen to tissues

2. **Aspirin**

Anti-platelet medication

Prevent worsening clot formation

3. **Nitroglycerin**

Dilates vessels to allow for more blood flow to tissue

4. **Morphine**

Reduces pain and discomfort -----> less O<sub>2</sub> requirement

Dilate vessels to allow for more blood flow to tissue

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## HIGH YIELD REVIEW

- ▶ There are three different types of ACS: Stable angina, unstable angina and myocardial infarction
- ▶ The pathophysiology of ACS is plaque build up and thrombus formation
- ▶ Ischemia = lack of O<sub>2</sub>, Infarction = death of tissue
- ▶ Stable angina occurs with exertion and goes away with rest. Unstable angina occurs at rest
- ▶ Treatment for chest pain is O<sub>2</sub>, Aspirin, Nitro and Morphine

# VASCULAR DISORDERS

As important as the heart is in pumping blood to the body to deliver oxygen, without the vascular system, delivery would be impossible. Disorders of the vasculature can lead to decreased perfusion despite adequate cardiac output and heart health. In this module we discuss some of the most common vascular issues as well as rarer but emergent conditions that the first responder must be able to recognize and treat.

## Learning objectives

**By the end of this module, you should be able to...**

- ▶ Define atherosclerosis and understand what leads to plaque build up in the vessels
- ▶ Define a thrombus and an embolism and understand their differences
- ▶ Recognize the signs and symptoms of deep vein thrombosis and arterial thrombosis as well as their treatments
- ▶ Understand aortic aneurysm and its risk factors as well as complications
- ▶ Recognize the signs and symptoms of aortic dissection and know how to treat it

# Atherosclerosis

Atherosclerosis is the thickening and narrowing of blood vessels

## Risk Factors and Contributing Factors

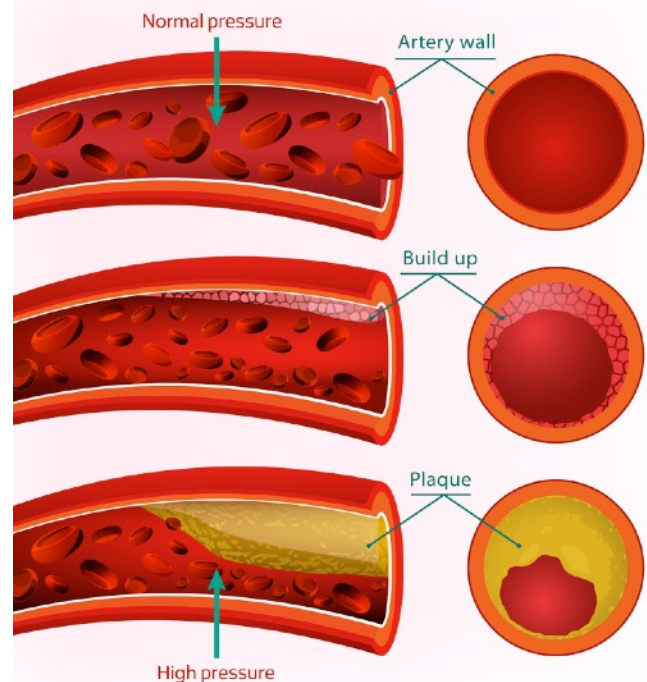
1. \_\_\_\_\_
2. \_\_\_\_\_
3. \_\_\_\_\_

Hypertension - high blood pressure puts continuous strain on the vessel walls which leads to injury of the inner lining of the blood vessels

- ▶ Damage to the inner lining of the vessels leads to formation of fatty deposits, or plaques, which thicken and narrow the vessels

High cholesterol - increased cholesterol levels in the blood stream leads to increased deposit of cholesterol into plaques, making them more solid and thicker which further narrows the vessels.

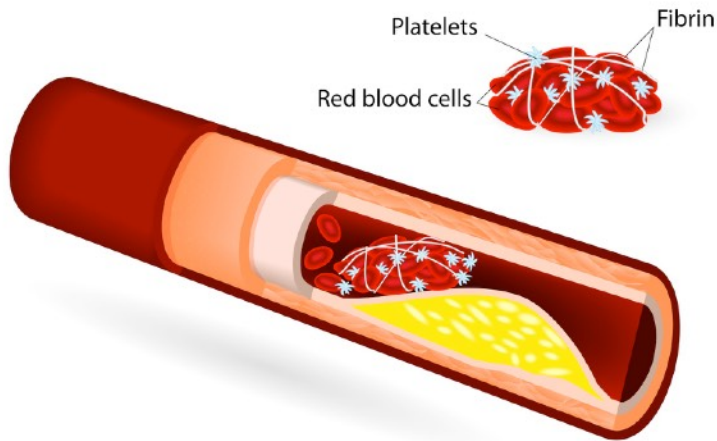
Smoking - smoking increases cholesterol levels. The nicotine and carbon monoxide in cigarette smoke also causes damage to the vessel walls



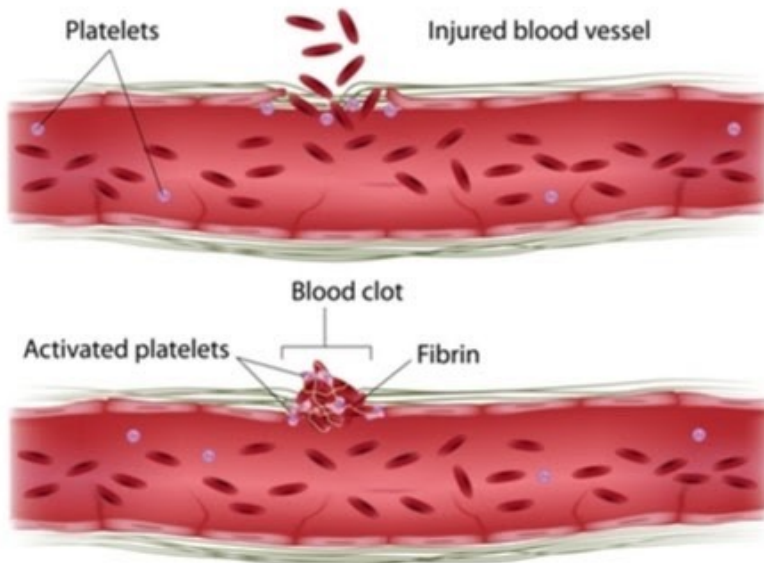
# Thrombus vs Embolus

**Thrombus** = a blood clot forms on the inner lining of the blood vessel

## THROMBUS

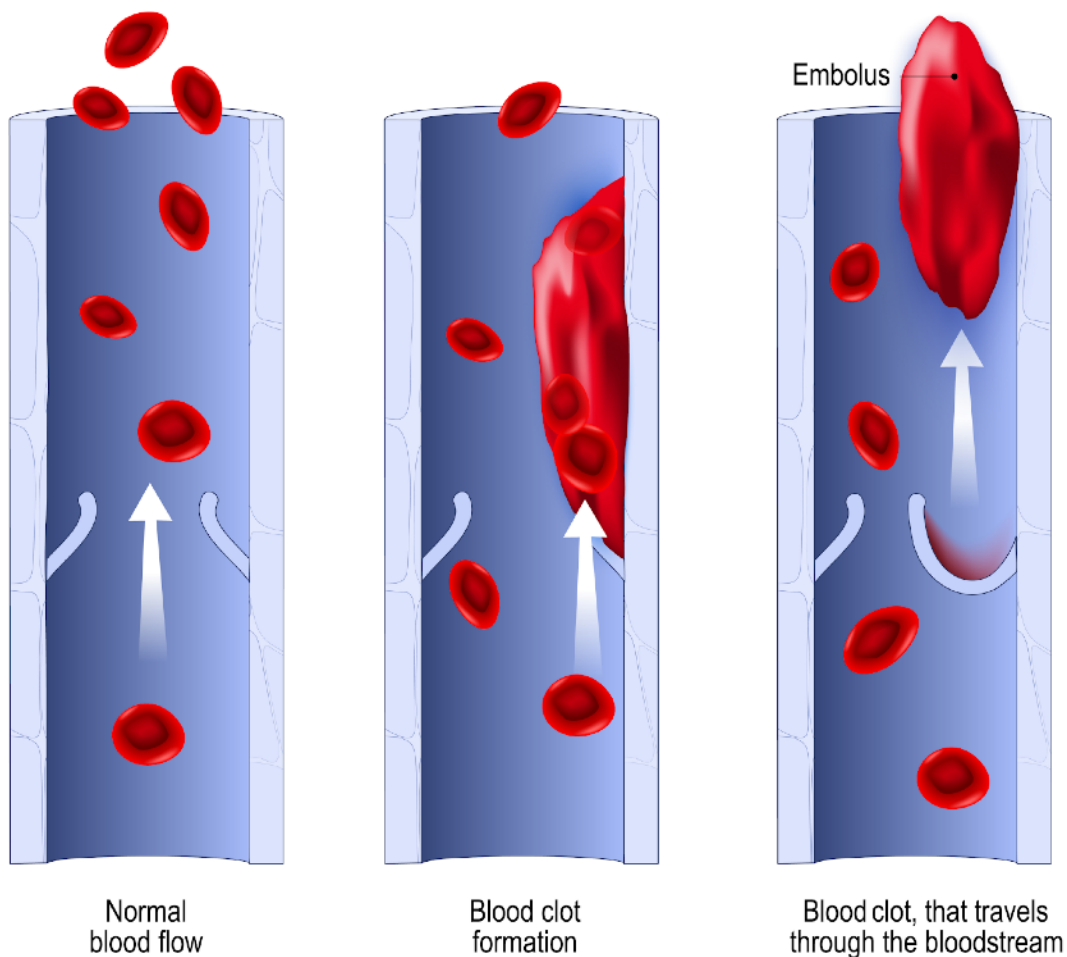


A thrombus will form where the blood vessel wall is disrupted. Examples include a tear in the vessel wall, on top of a plaque, or on top of an indwelling catheter such as an IV



**Embolism** = any foreign material that lodges inside blood vessels and blocks flow

- ▶ An embolism can be a blood clot (thrombus), broken off piece of plaque, fat globule or air.
- ▶ A thromboembolism is an embolism that is a thrombus, as seen in DVT's that break loose and travel to the lungs.
- ▶ A fat embolism is where a fat globule enters the bloodstream and obstructs flow. This can happen after long bone fractures such as femur fractures.
- ▶ An air embolism can occur due to diving injuries, or peripheral infection of air into the vessels.



# Deep Vein Thrombosis (DVT)

## Signs and Symptoms

1. Swelling
  2. Pain
  3. Edema
- ▶ Superficial vein thrombosis exists but these resolve on their own and are self absorbed by the body
  - ▶ Extremities with DVTs continue to have pulses and remain warm and pink because arterial blood flow is intact

## Treatment

1. Blood thinners
  2. Surgical removal
- 

# Arterial Occlusion

\_\_\_\_\_ = TRUE EMERGENCY!!!

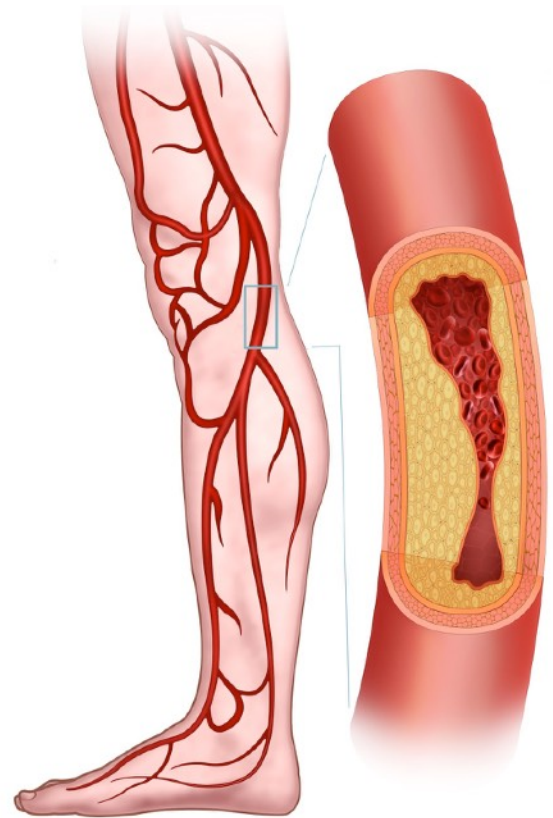
## Causes

1. Atherosclerosis - Gradual onset
2. Embolism - Sudden onset

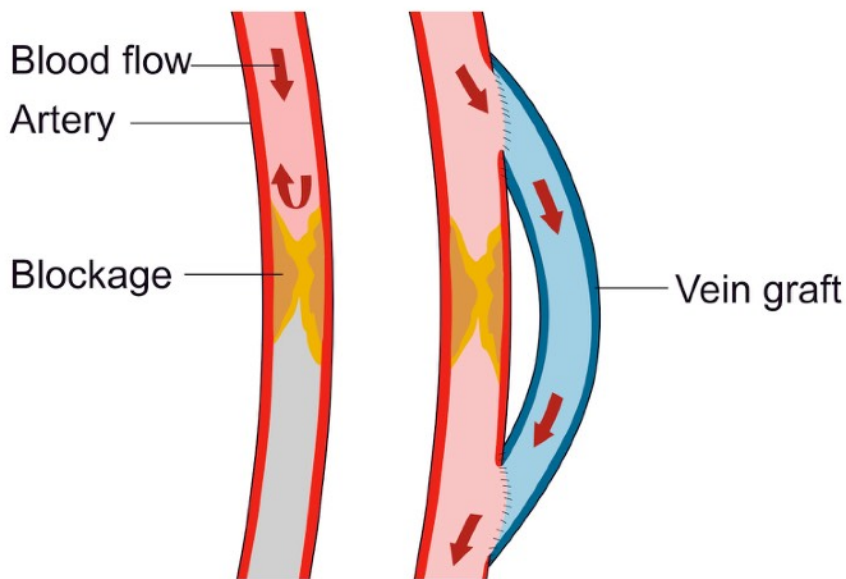


### 5Ps of Arterial Occlusion

1. \_\_\_\_\_
2. \_\_\_\_\_
3. \_\_\_\_\_
4. \_\_\_\_\_
5. \_\_\_\_\_



### BYPASS SURGERY



### Treatment of arterial occlusion

1. Bypass surgery
2. TPA (clot busting drug)
3. Surgical removal of embolism

# Aortic Aneurysm

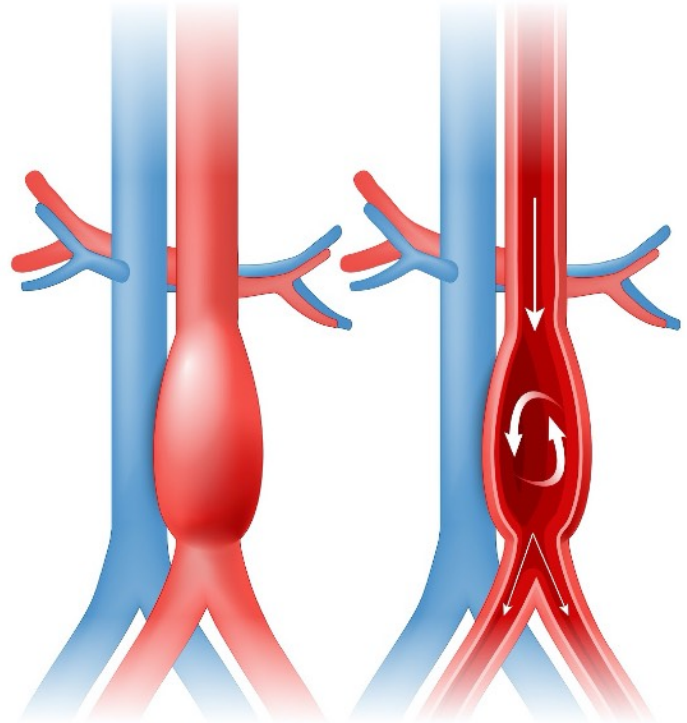
## Risk Factors

1. HTN
2. High cholesterol
3. Smoking
4. Trauma
5. Connective tissue disorders

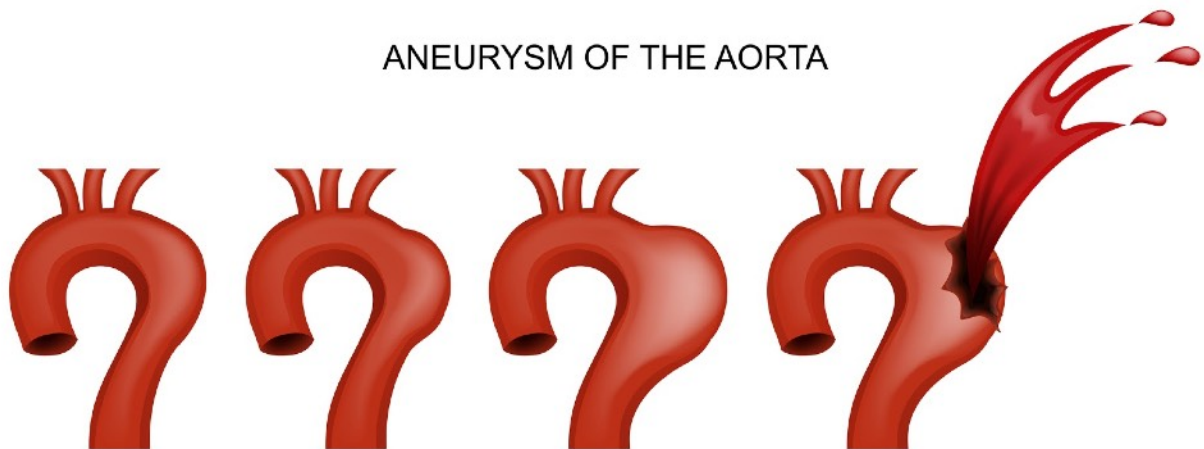
Blood flow through the aorta is typically linear. When aneurysm occurs, the flow becomes more turbulent which puts more pressure on the aneurysm walls leading to more ballooning.

An aneurysm does not cause symptoms. However aneurysms can continue to balloon and grow and lead to rupture or dissection.

# Aortic Aneurysm



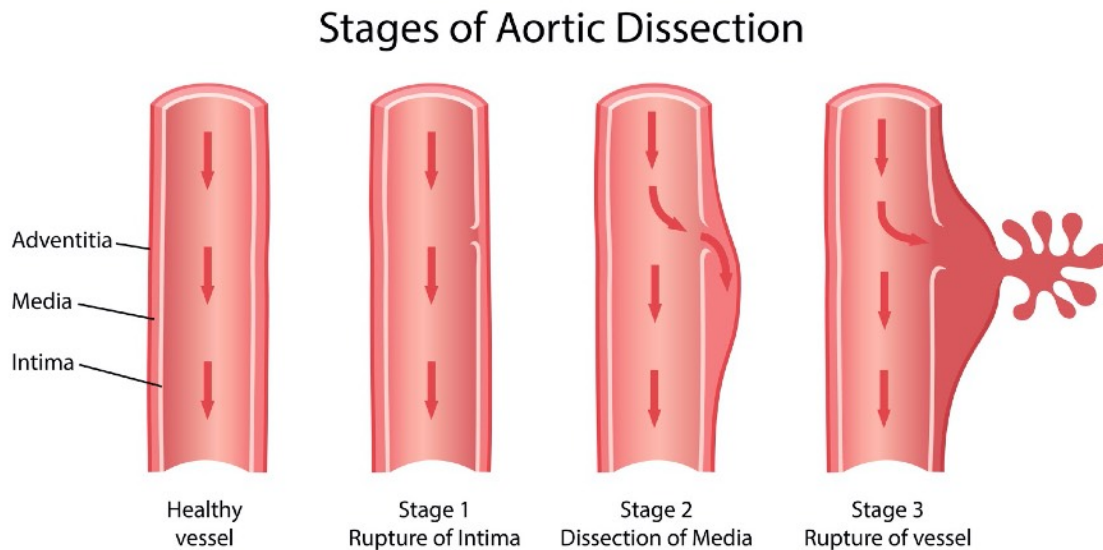
## ANEURYSM OF THE AORTA



- ▶ A ruptured aorta will lead to massive blood loss and drop in BP. Almost 100% mortality if this occurs.
- ▶ Emergent surgery is the only treatment for aortic rupture but most patients won't make it to the hospital in time.

# Aortic Dissection

- ▶ In an aortic dissection, a tear occurs in the vessel wall but does not tear completely through.
- ▶ This tear allows blood to flow into the wall of the vessel, creating a false tract.
- ▶ Ongoing pressure and flow of fluid into this space leads to continual tearing and eventually can lead to rupture.



- ▶ The layers of the aorta wall are the intima, media and adventitia. A tear occurs through the intima and allows blood to flow into the media.

## Signs and Symptoms

1. Tearing chest pain radiating to back - caused by tearing of the aortic wall.
  2. Uneven BP and/or pulses - dissection may extend into one of the large vessels of one arm.
  3. Hypertension - dissection creates a loss of blood into the false tract leading to hypertension as the body compensates.
- ▶ The increased blood pressure found in dissection leads to further tearing of the vessel wall.
  - ▶ Prehospital treatment and Emergency department treatment is centered around lowering blood pressure to prevent further dissection and buy time for patients to go to the operating room.

## Treatment

1. Treat \_\_\_\_\_
  2. Lower HR and BP
    - ▶ Medications such as esmolol, which lower both HR and BP, are ideal, however protocols may call for a different medication such as nitroglycerin to lower BP.
- 

# HIGH YIELD REVIEW

- ▶ Contributing factors to atherosclerosis = HTN, High cholesterol and smoking
- ▶ Thrombus is a blood clot attached to a vessel wall. Embolus is foreign material that travels through vessel and than lodges and blocks blood flow
- ▶ Embolus may be blood clot, pieces of plaque, fat globule or air
- ▶ Pulseless limb = true emergency
- ▶ 5Ps of arterial occlusion; pain, pallor, pulselessness, pain, paresthesias
- ▶ Aortic aneurysms are asymptomatic but can lead to dissection or rupture
- ▶ Dissections create false tract in aortic wall and elevated HR and BP worsen the condition
- ▶ Treatment of dissection centered around lowering blood pressure

# SHOCK AND RESUSCITATION

The role of emergency medicine, both prehospital and in-hospital is to recognize and resuscitate the unstable patient. Stability of a patient is dependent on a few different factors, but one of the most important is perfusion. Perfusion is the delivery of blood, and thus oxygen, to the tissues and is required to sustain life. Patients who suffer from hypoperfusion enter into a state known as shock and without early identification and intervention will continue to decompensate. In this module we discuss the pathophysiology of this state, how it presents, and the best treatment modalities to prevent further decompensation of our patients.

## Learning objectives

**By the end of this module, you should be able to...**

- ▶ Identify shock and its signs and symptoms
- ▶ Define hypovolemic shock, its causes and its treatments
- ▶ Define cardiogenic shock, its main causes and its treatments
- ▶ Define obstructive shock, its main causes and its treatments
- ▶ Define distributive shock, its main causes and its treatments

---

## Shock

**Shock** - a state in which the body is hypoperfusing and decompensating.

- ▶ Hypoperfusion is a decreased, and inadequate, supply of blood and oxygen to the tissues and organs of the body.
- ▶ Low blood pressure can be a sign of hypoperfusion and thus shock, however low pressure is a late sign of shock and other signs and symptoms of hypoperfusion may present first.

**On your exam however, if they want you to recognize shock they will most likely give you a low BP.**

# Signs and Symptoms of Shock

1. Low blood pressure (late finding)
2. Ischemic chest pain
3. Altered mental status
4. Signs of heart failure

## Types of Shock

1. \_\_\_\_\_
2. \_\_\_\_\_
3. \_\_\_\_\_
4. \_\_\_\_\_

---

# Hypovolemic Shock

**Hypovolemic = low volume**

## Causes

1. Loss of blood
2. Loss of body fluid
  - ▶ Vomiting
  - ▶ Dehydration

## Treatment

1. Blood products
2. IV fluid

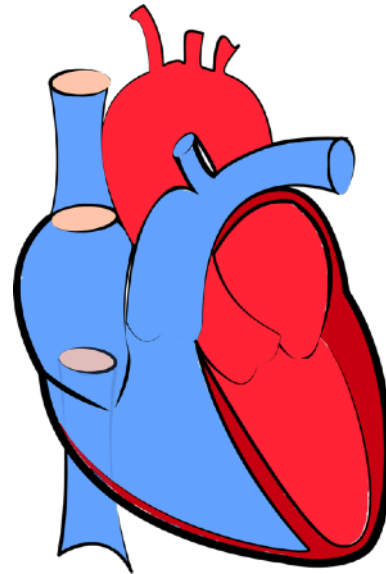


# Cardiogenic Shock

**Cardiogenic shock = pump problem**

## Causes

- ▶ Heart failure
- ▶ Myocardial infarction
- ▶ Cardiac infection
- ▶ Valvular disorder



## Treatment

1. Limit IV fluids
  - ▶ All shock should be initially treated with fluids, but if the heart is not pumping adequately as in cardiogenic shock, too much fluid could worsen heart failure
2. Vasopressors
  - ▶ Vasopressors are medications such as dopamine, epinephrine and norepinephrine which cause the vessels to constrict and also increase the ability of the heart to squeeze

# Obstructive Shock

**Obstructive shock = block of blood flow**

## Causes

- ▶ \_\_\_\_\_
- ▶ \_\_\_\_\_
- ▶ \_\_\_\_\_

## Treatment

1. Cardiac tamponade = pericardiocentesis
2. Pulmonary embolism = TP
3. Tension pneumothorax = needle decompression

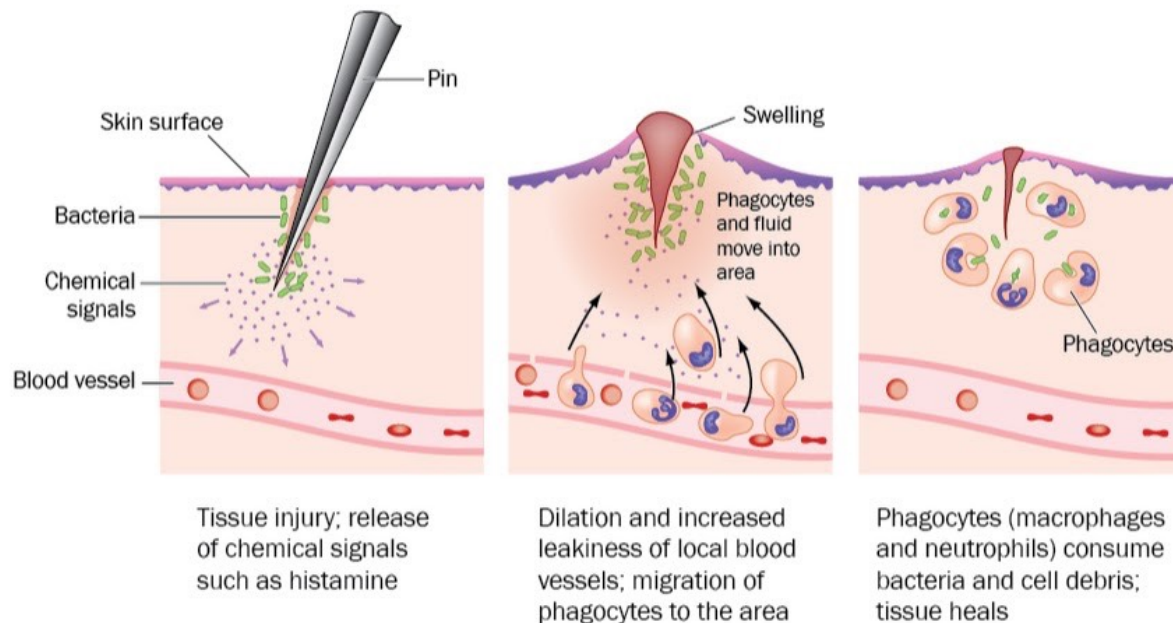
**TENSION  
PNEUMOTHORAX**



# Distributive Shock

## Distributive shock = systemic vasodilation

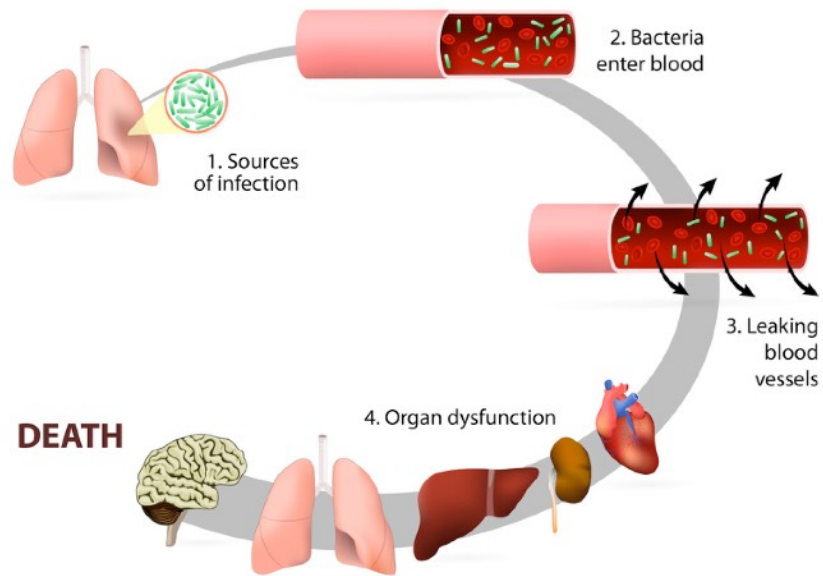
- ▶ In distributive shock the volume of blood is distributed over too large of an area so that no organ or tissue is getting adequate perfusion.
- ▶ Pressure is inversely proportional to volume. So as the volume of the vessels increases, the pressure inside the vessels decreases leading to lack of perfusion.



1. In local trauma, infection or other stress, the blood stream mobilizes white blood cells (WBCs) that travel to the site of stress.
  2. Vessels then dilate in order for the WBCs to leak through the vessel wall.
  3. WBCs heal damage and fight off infection at the local site.
- ▶ In distributive shock, the same thing occurs but on a systemic level. When all of the vessels of the body dilate in response to stress, infection or trauma, overall perfusion decreases, leading to shock.



# Sepsis



## Types of Distributive Shock

1. \_\_\_\_\_
2. \_\_\_\_\_
3. \_\_\_\_\_

## Distributive Shock Treatment

1. Fluids
2. Vasopressors
3. Treat underlying causes
  - ▶ Sepsis = antibiotics
  - ▶ Anaphylaxis = epinephrine
  - ▶ Neurogenic = supportive care

# HIGH YIELD REVIEW

- ▶ Four types of shock; hypovolemic, cardiogenic, obstructive and distributive
- ▶ Classic signs of all shock = low BP, ischemic chest pain, altered mental status
- ▶ Low BP can be a late finding but on your exam will be your clinical clue for shock
- ▶ First line treatment for all shock is IV fluids, however in cardiogenic shock care must be taken to limit these fluids as not to make the problem worse
- ▶ Distributive shock includes, septic shock, anaphylaxis and neurogenic shock
- ▶ Distributive shock is marked by systemic vasodilation leading to overall low perfusion

# CARDIAC TAMPONADE

Cardiac tamponade is a rare but fatal condition that, if left undiagnosed, can lead to swift decompensation and death of the patient. In this module we discuss the pathophysiology of cardiac tamponade as well as its presentation and treatments so that you will be well prepared to recognize this life threatening condition in the field and act quickly to provide the patient with the care they need.

## Learning objectives

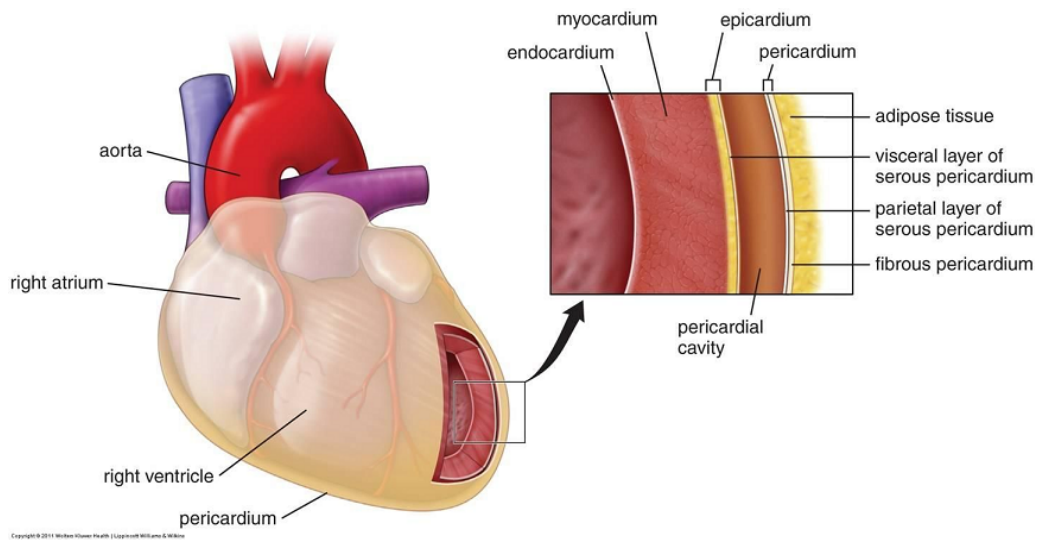
**By the end of this module, you should be able to...**

- ▶ Review the layers of heart tissue and identify the pericardial space
  - ▶ Explain cardiac tamponade and recognize its signs and symptoms
  - ▶ Describe and identify the two ECG findings that can appear with cardiac tamponade
  - ▶ Recall the definitive treatment of cardiac tamponade as well as risk factors for its development
- 

## Layers of Heart Wall

### Heart Wall

1. Endocardium
2. Myocardium
3. Pericardium
  - a. Visceral pericardium
  - b. Parietal pericardium



- ▶ The visceral pericardium sits on the heart, while the parietal pericardium attaches to the chest wall
- ▶ The space between the visceral pericardium and parietal pericardium is the pericardial cavity/sac
- ▶ A thin layer of fluid resides in the pericardial sac that allows the heart to move smoothly inside the chest wall as it beats
- ▶ Too much accumulation of fluid in the pericardial sac leads to filling difficulties and pressure build up on the heart

## Cardiac Tamponade

### Cardiac tamponade:

- ▶ Impaired diastolic filling due to increased outside pressure on the heart
- ▶ Heart contracts without difficulty but then does not fill appropriately because the pressure from the fluid in the pericardial sac

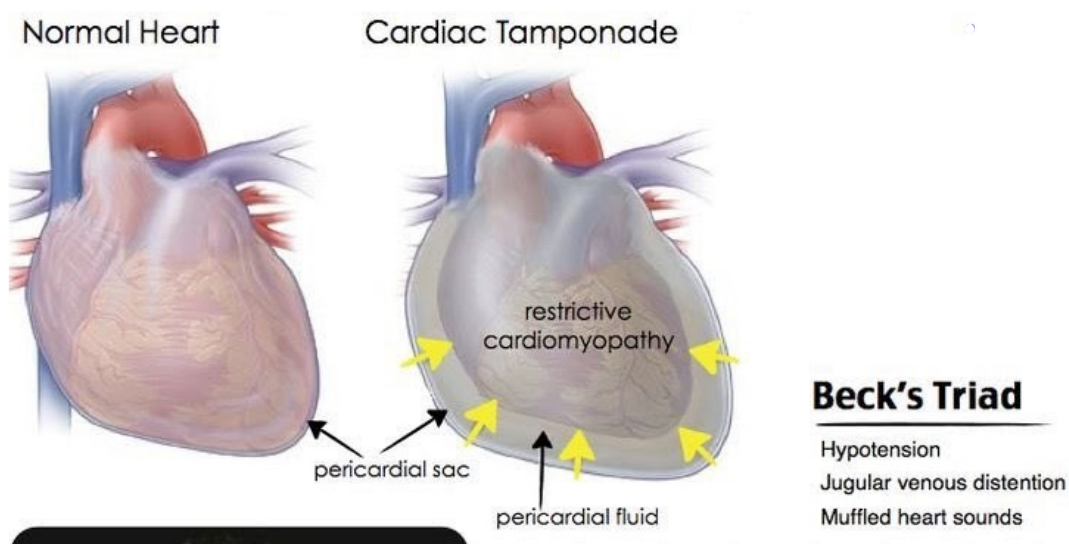
### Signs and Symptoms

1. Jugular venous distention (JVD)
2. Low blood pressure
3. Muffled heart sounds

**Jugular venous distention** – Fluid returning from the superior vena cava meets resistance as it enters the heart due to the increased pressure. This fluid backs up into the superior vena cava and jugular veins causing them to bulge and distend.

**Low blood pressure** – Because the heart cannot adequately fill, there is a decrease in cardiac output leading to low blood pressure.

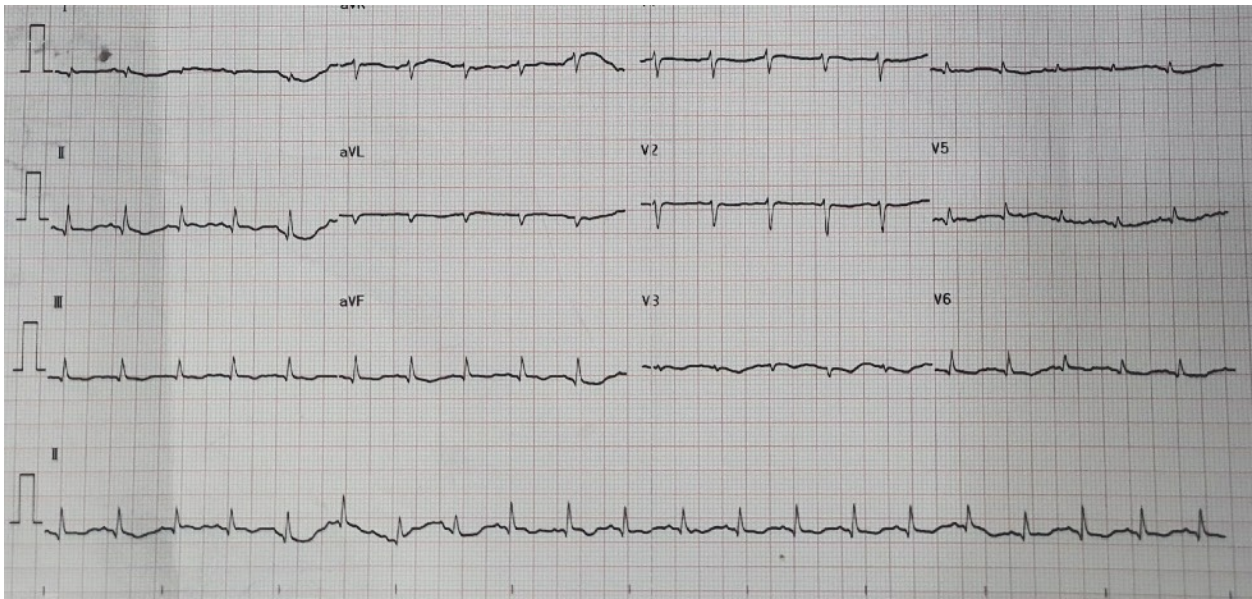
**Muffled heart sounds** – When listening with the stethoscope, the heart sounds must travel through the pericardial sac and accumulated fluid there. This muffles the heart sounds as if listening to them underwater.



**Beck's Triad = JVD, Low BP and muffled heart sound**

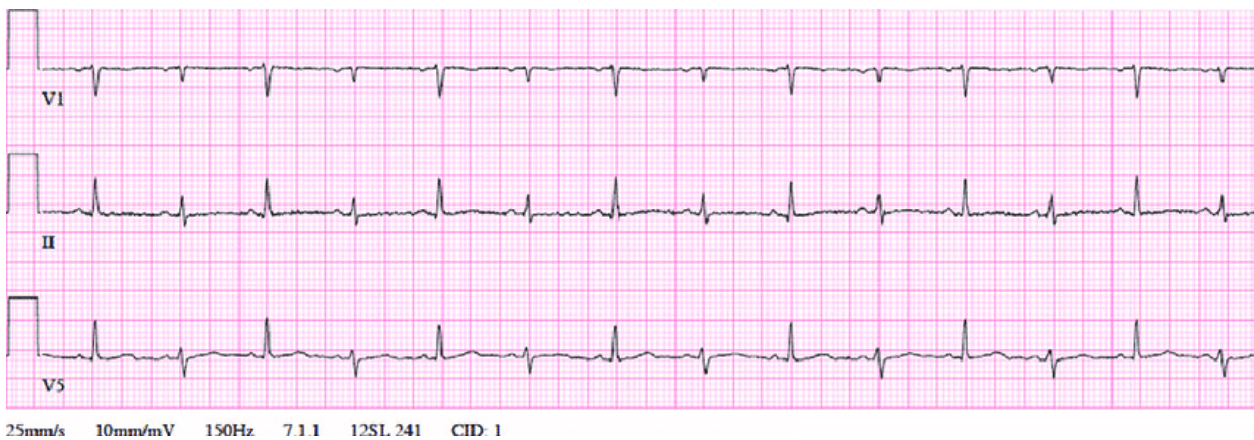
## ECG Findings

1. Low Voltage
  - ▶ Just as heart sounds are muffled because of sound moving through the fluid in the pericardial sac, the electrical activity must travel through the fluid as well. This leads to what appears to be low voltage on the ECG



## 2. Electrical alternans

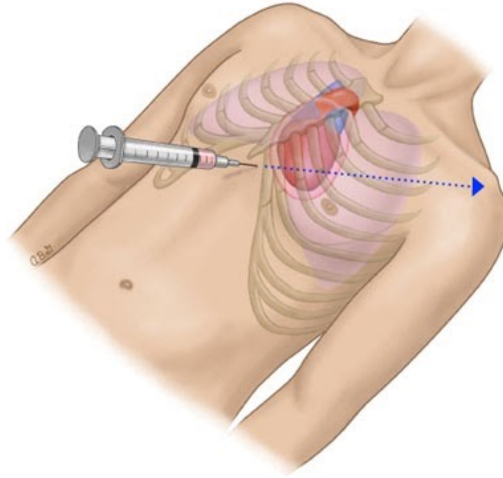
- ▶ Electrical alternans occurs because the heart starts swinging back and forth inside the fluid filled pericardial sac. As it swings closer to the ECG leads, voltage appears amplified and as it swings away, voltage appears diminished. The voltage alternates between high and low.



**ECG findings are not always noted but their presence is a clue to the diagnosis**

# Management and Causes

Definitive management of cardiac tamponade is \_\_\_\_\_



## Causes

1. Trauma
  2. Cancers
  3. Infections
  4. Kidney Problems
- ▶ Trauma can lead to bleeding in the pericardial sac.
  - ▶ Cancers and infections can sometimes lead to overdevelopment of fluid from inflammation.
  - ▶ Kidney failure may lead to inadequate filtering of fluid and back up of fluid in the body including the pericardial sac.

**It is not the amount of fluid that accumulates in the pericardial sac that determines if cardiac tamponade will occur, but rather the rate at which it accumulates. Even small amounts of fluid in the pericardial sac can lead to tamponade if it accumulates quickly and the parietal pericardium cannot stretch and accommodate.**

# HIGH YIELD REVIEW

- ▶ Cardiac tamponade is due to fluid accumulation in the pericardial sac
- ▶ It is not the amount of fluid, but the rate of accumulation that leads to tamponade
- ▶ Beck's triad = JVD, low BP and muffled heart sounds
- ▶ Low voltage and electrical alternans can be seen but are not always present
- ▶ Definitive treatment is pericardiocentesis



# CARDIAC INFECTIONS

Infection of the cardiac tissue is an often overlooked etiology for fever and sepsis. Though not as common as pneumonia and urinary tract infections, infections of the cardiac tissues can occur and lead to poor outcomes if undiagnosed. In this module we will cover the different types of cardiac infections, however keep in mind that many of these patients will present similarly. Patients who have general infectious symptoms such as fever and fatigue without an obvious source should be considered to be possible cases of cardiac infections. If their infectious symptoms are combined with signs and symptoms of heart failure, the chance of a cardiac infection becomes much higher. An understanding of how these infections occur and present will help prevent the first responder from overlooking cardiac infections as a possible diagnosis in their patients.

## Learning objectives

**By the end of this module, you should be able to...**

- ▶ Identify the risk factors for cardiac infections
- ▶ Recognize the signs and symptoms of endocarditis
- ▶ Recognize the signs and symptoms of myocarditis
- ▶ Recognize the signs and symptoms of pericarditis
- ▶ Recognize the signs and symptoms of rheumatic heart disease and its causes

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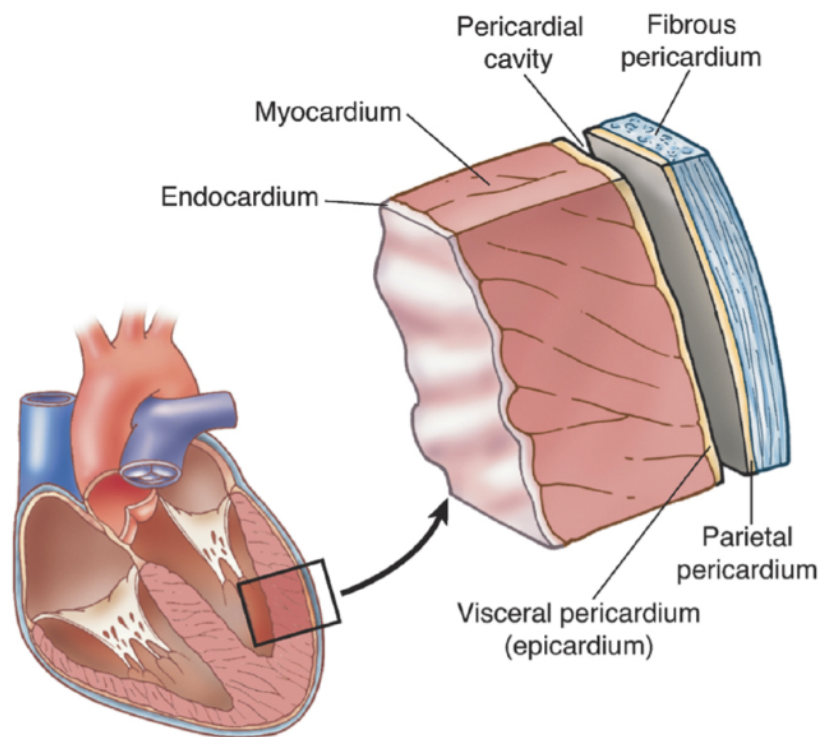
## Cardiac Infections

### Risk Factors

1. IV drug use
2. Dental surgery
3. Heart surgery
4. Viral syndromes

## The three layers of the heart wall

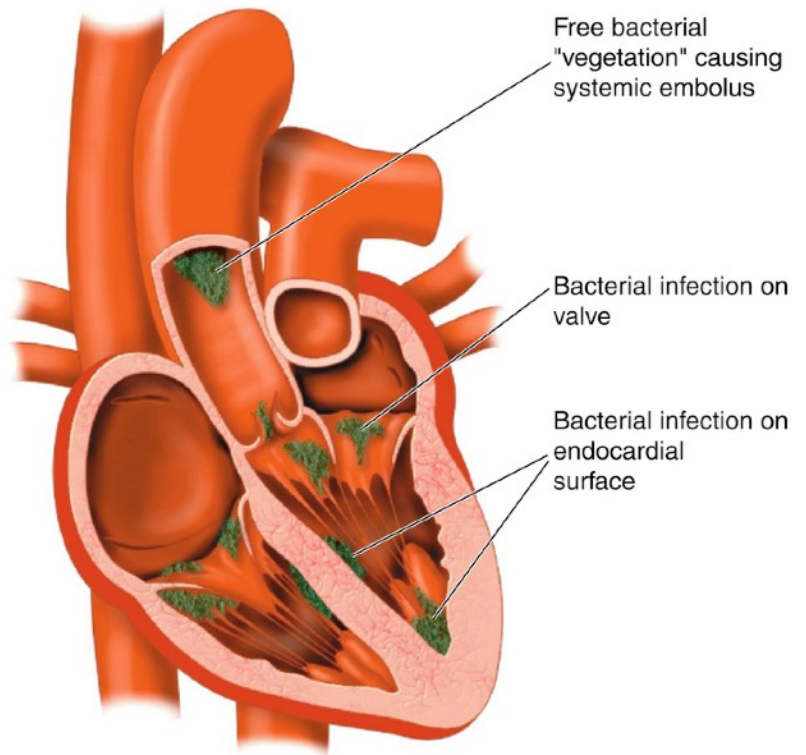
1. \_\_\_\_\_
2. \_\_\_\_\_
3. \_\_\_\_\_



Infection and inflammation can occur in any of these layers. There is no specific reason why some infections affect one layer over another, but all require prompt diagnosis and treatment to decrease morbidity and mortality.

# Endocarditis

- ▶ Endocarditis is infection of the innermost layer of heart tissue; the endocardium
- ▶ Infection is usually due to bacterial growth
- ▶ Because the endocardium is the innermost layer, endocarditis will affect the valves as well
- ▶ Infection on the valves can lead to emboli of infected material that break off and travel to the lungs. We call these types of emboli \_\_\_\_\_



## Type

- ▶ Bacterial Infection

## Location

- ▶ Endocardium

## Classic Symptoms

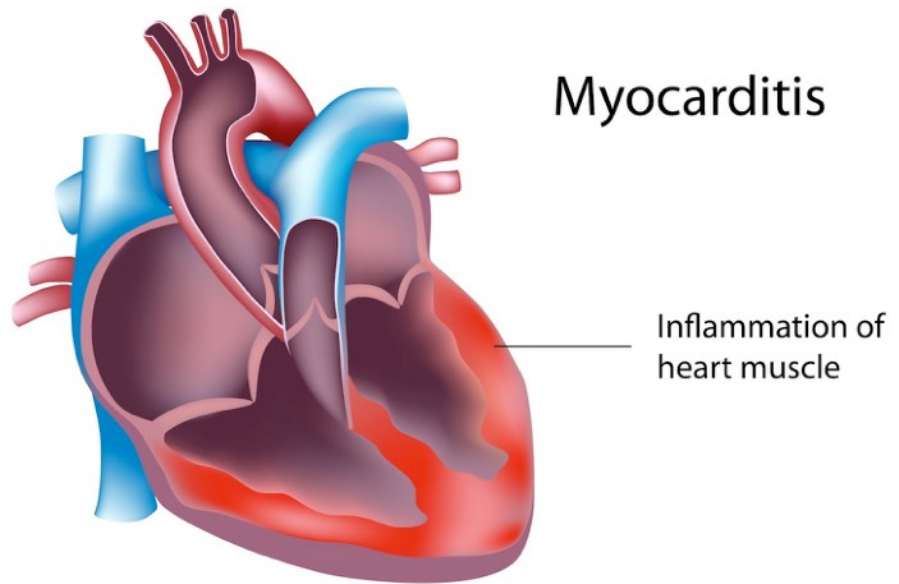
1. \_\_\_\_\_
2. \_\_\_\_\_

## Treatment

- ▶ IV Antibiotics

# Myocarditis

- ▶ The infection and inflammation of the middle layer of heart tissue; the myocardium.
- ▶ The myocardium is the muscular layer of the heart and the layer that the electrical system of the heart runs through.
- ▶ Because the conduction system runs through this layer, patients may present with arrhythmias as this pathway is disrupted.



## Type

- ▶ Viral Infection

## Location

- ▶ Myocardium

## Classic Symptoms

1. Fever
2. Flu-like symptoms
3. Arrhythmias
4. Heart failure

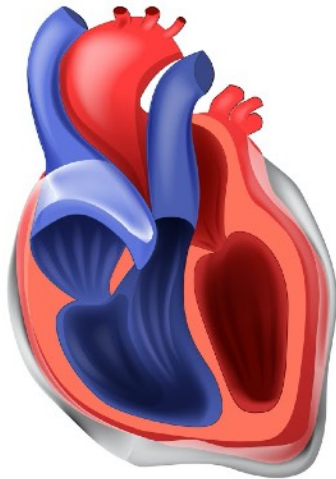
## Treatment

- ▶ Supportive
- ▶ Treat complications (heart failure, arrhythmias etc)

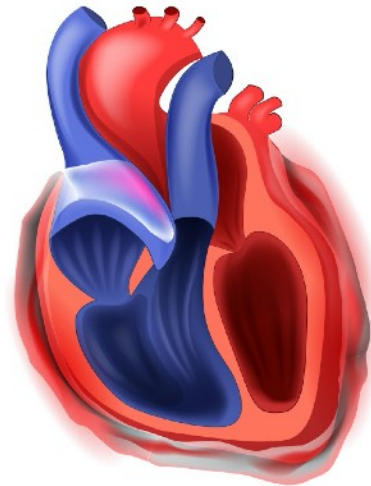
# Pericarditis

- ▶ Pericarditis is infection and inflammation of the outermost layer of the heart tissue; the pericardium.
- ▶ The pericardium is in contact with the mediastinum and irritation can cause chest pain as well as a rubbing sound when the heart is auscultated. This is called a “friction rub”.
- ▶ Like myocarditis, pericarditis is typically viral and self limited.

## PERICARDITIS



a healthy pericardium



pericarditis

### Type

- ▶ Viral Infection

### Location

- ▶ Pericardium

### Classic Symptoms

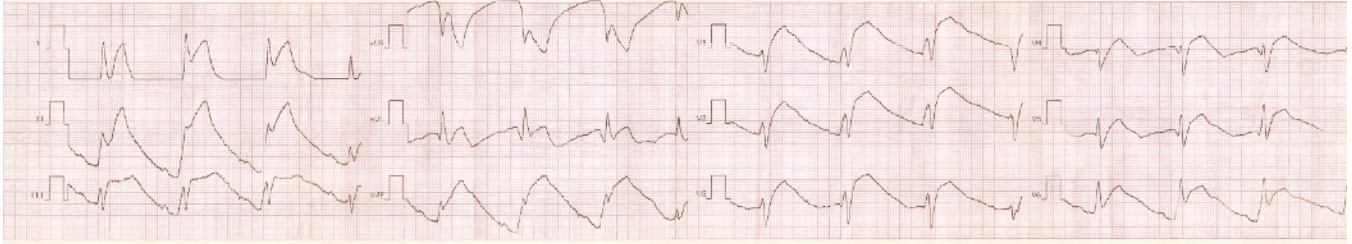
1. Fever
2. Flu-like symptoms
3. Heart Failure
4. Diffuse ST segment elevation

### Treatment

- ▶ Supportive
- ▶ Treat complications

Pericarditis can show up on ECG as \_\_\_\_\_.

This is because the inflammation can spread throughout the entire pericardium and entire heart causing irritation.

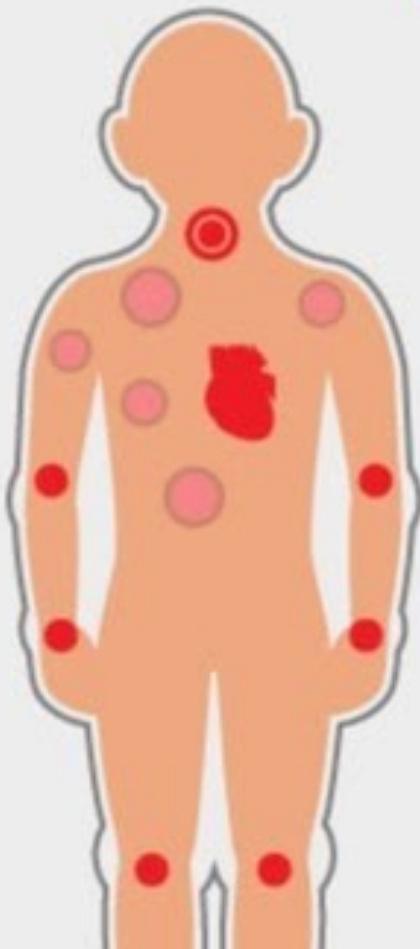


## Rheumatic Heart Disease

- ▶ Rheumatic heart disease is a complication of rheumatic fever, which is a complication of Group A strep.
- ▶ Group A strep can present as Strep throat or Scarlet Fever. Scarlet fever presents as rash, fever and sore throat whereas strep pharyngitis presents as fever, sore throat and pus on the tonsils.
- ▶ If left untreated, Group A strep can lead to rheumatic fever. Rheumatic fever typically occurs after the group A strep infection has subsided and in children between 5 and 15 years old.
- ▶ Signs and symptoms of rheumatic fever include inflammation of the heart and heart valves, joints and the central nervous system.
- ▶ The inflammation of the heart and heart valves in rheumatic fever can lead to ongoing and lifelong issues with the heart which is defined as rheumatic heart disease.
- ▶ Rheumatic heart disease may not show up for 10-20 years after rheumatic fever.



## RHEUMATIC HEART DISEASE



Rheumatic heart disease (RHD), is damage caused to the heart's valves by rheumatic fever, which then do damage to the heart, particularly the heart valves. Although rheumatic fever can strike people of all ages, it is most common in children between 5 and 15 years old.

Rheumatic fever symptoms:

- 🎯 strep throat
- swollen joints
- skin sores
- scars in the heart valves

# HIGH YIELD REVIEW

- ▶ The three layers of the heart are the endocardium, myocardium and pericardium
- ▶ Risk factors for cardiac infections include IV drug use, dental surgery, heart surgery, viral syndromes
- ▶ Endocarditis typically presents with fever and new heart murmur
- ▶ Myocarditis and pericarditis are typically viral
- ▶ Pericarditis may presents with diffuse ST elevation on ECG
- ▶ Rheumatic heart disease is a complication of rheumatic fever from untreated group A strep infection
- ▶ Rheumatic fever is usually seen in children 5-15 with fever, inflammation of joints, heart and heart valves



# CONGENITAL HEART DEFECTS

Congenital defects are caused by genetic alterations leading to changes in normal anatomy and function at birth. Typically these alterations cause changes in multiple organ systems, however this module focuses on the heart defects that can occur and how they will present. The focus here is not to memorize the different defects but to recognize findings and risk factors that should raise suspicion for such conditions in infants and children to allow for early identification and treatment.

## Learning objectives

**By the end of this module, you should be able to...**

- ▶ Identify common risk factors for congenital defects
- ▶ Understand the signs and symptoms of cyanotic heart defects and the most common types of defects that call into this category
- ▶ Understand the signs and symptoms of non cyanotic heart defects and the most common types of defects that fall into this category.

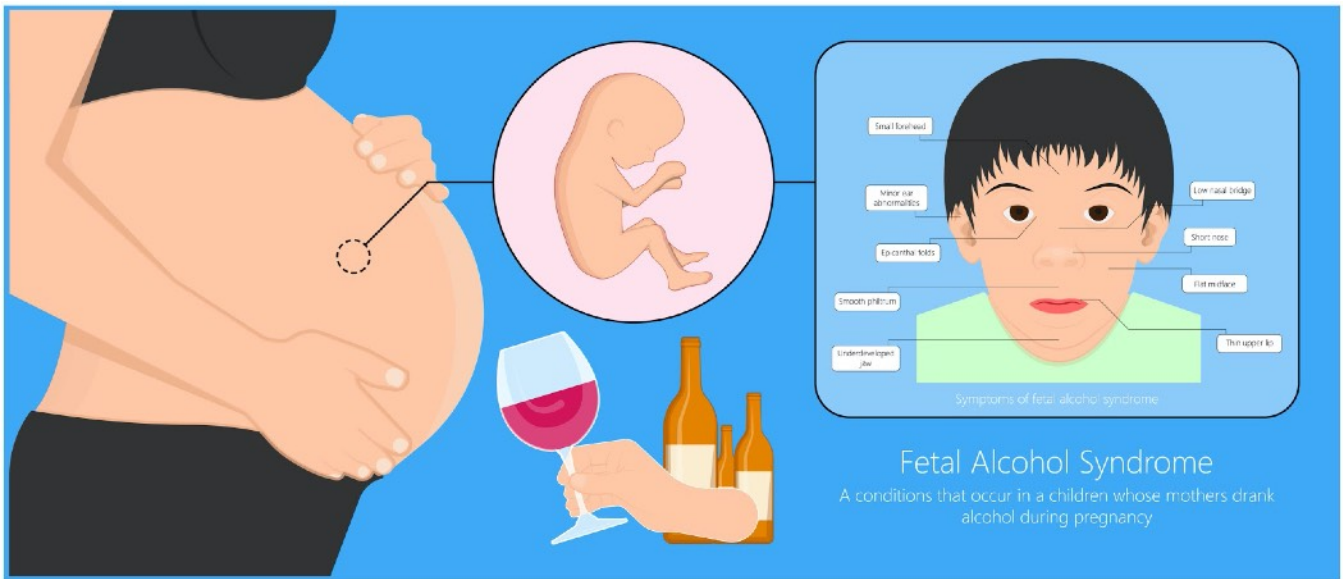
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## Congenital Defects

Congenital defects are birth defects that arise because of genetic alterations prior to birth. Congenital defects may affect multiple systems and their physiology.

Risk Factors for Congenital Defects

1. Genetic mutations
2. Maternal alcohol use
3. Maternal infection (ex rubella)
4. Maternal drug use
5. Certain medications if taken during pregnancy



## Cyanotic Heart Defects

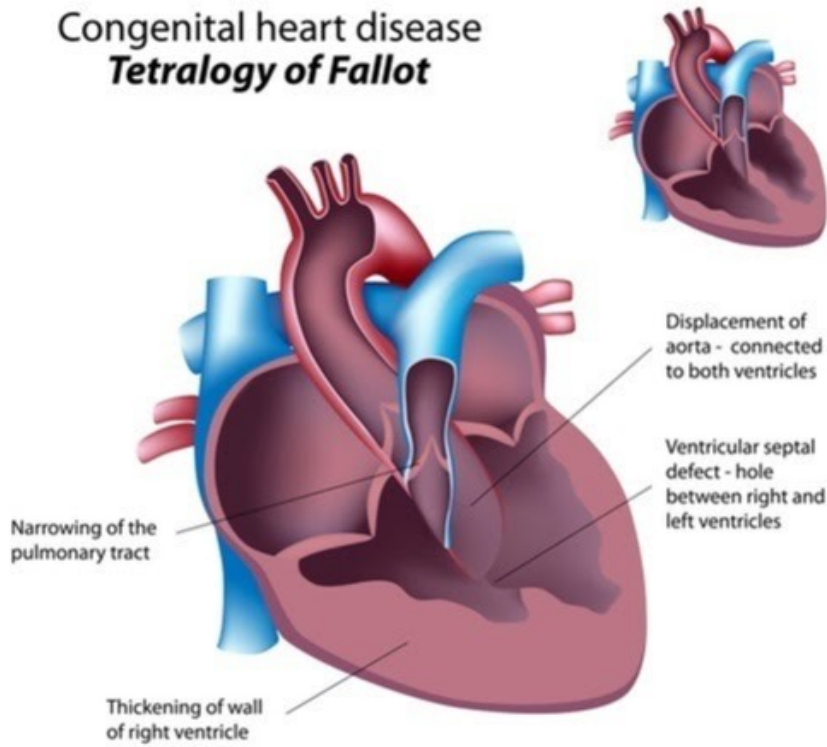
Right to left shunting of blood in the heart that bypasses the lungs

Presents: (Circle One) Infancy / Childhood

### Signs and Symptoms

- ▶ Cyanosis: blue lips, fingers, toes
- ▶ Hypoxia

## Congenital heart disease **Tetralogy of Fallot**



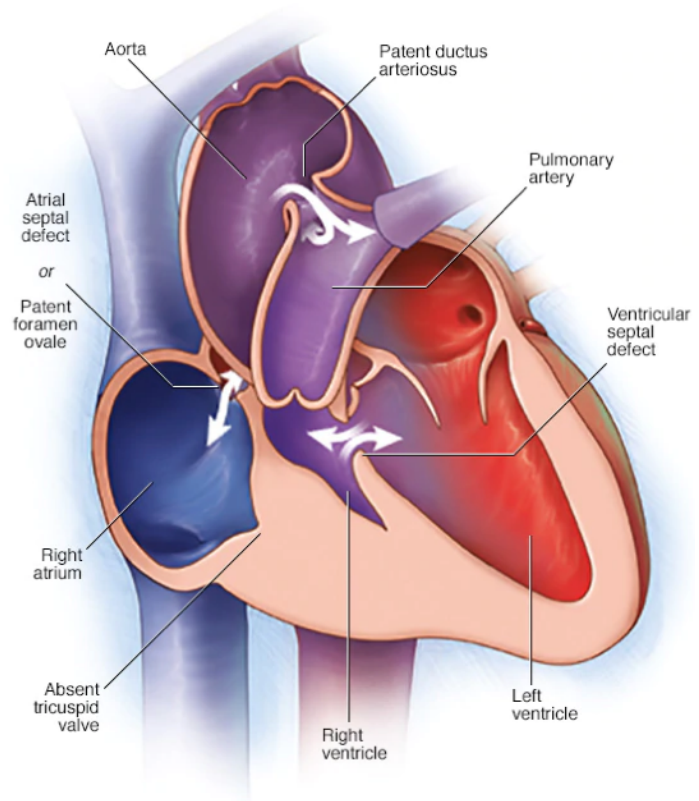
### Examples:

#### 1. Tetralogy of Fallot

- ▶ Ventricular septal defect
- ▶ Displacement of aorta
- ▶ Narrowing of pulmonary tract
- ▶ Thickening of right ventricular wall

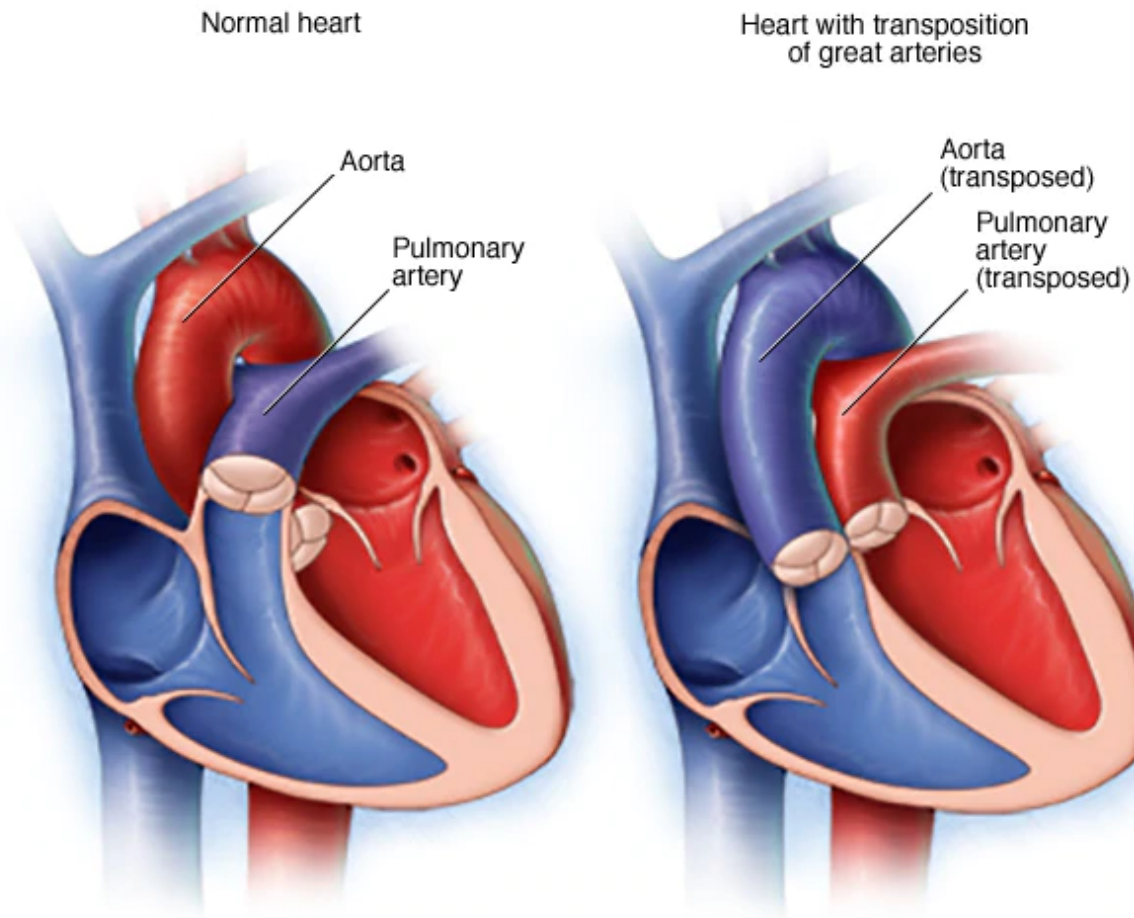
#### 2. Tricuspid atresia

- ▶ Tricuspid valve does not form correctly



## 2. Transposition of the great vessels

- ▶ Switching of aorta and pulmonary vessels



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## Non Cyanotic Heart Defects

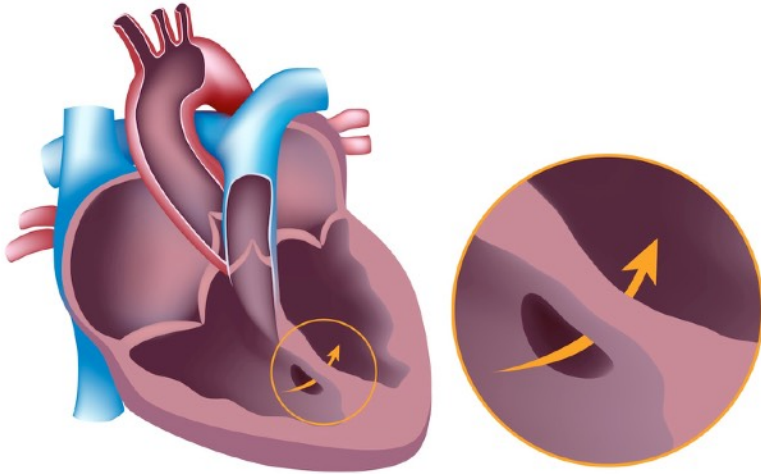
Left to right shunting of blood (mixing of oxygenated and deoxygenated)

Presents: (Circle One) Infancy / Childhood

### Signs and Symptoms

- ▶ SOB
- ▶ Fatigue and syncope with exertion

Congenital heart disease  
***Ventricular septal defect***

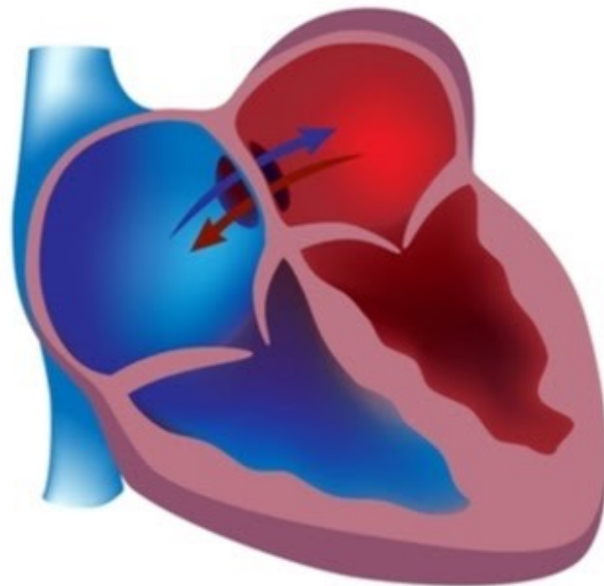


Examples:

1. Ventricular septal defect

Congenital Heart Disease  
***Atrial Septal Defect***

2. Atrial septal defect



**VSD and ASD are the most common of all congenital abnormalities**

# HIGH YIELD REVIEW

- ▶ Cyanotic heart diseases present in infancy, non cyanotic heart diseases present in childhood
- ▶ Remember the T's for cyanotic heart disease: Tetralogy, Tricuspid atresia, Transposition of the great vessels
- ▶ Cyanotic heart diseases are right to left shunting. Non cyanotic are left to right shunting
- ▶ Ventricular septal defects and atrial septal defects (non cyanotic heart defects) are the most common of all birth defects