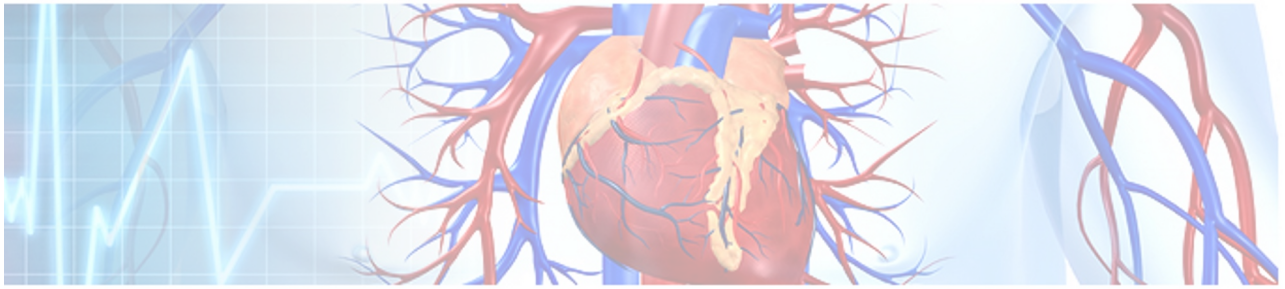


Dr. Gary Mumaugh
Campbellsville University

Vascular Disorders

Basic Vascular Structure: Arteries

Tunica Intima

- Innermost layer of flattened endothelial cells
- **Basement Membrane**- Layer of glycoproteins that keeps the endothelial cells in place
- Layer of fibrous connective tissue below BM

Basic Vascular Structure: Arteries

Tunica Media

- Thickest part of arterial wall
- Elastic connective tissue
- **Internal elastic lamina**- thin, strong elastic tissue separating tunica interna from media
- Surrounded by the **external elastic lamina**- another layer of elastic connective tissue

Basic Vascular Structure: Arteries

Tunica Adventitia

- Outermost of three layers
- Protective layer of loosely organized fibrous connective tissue

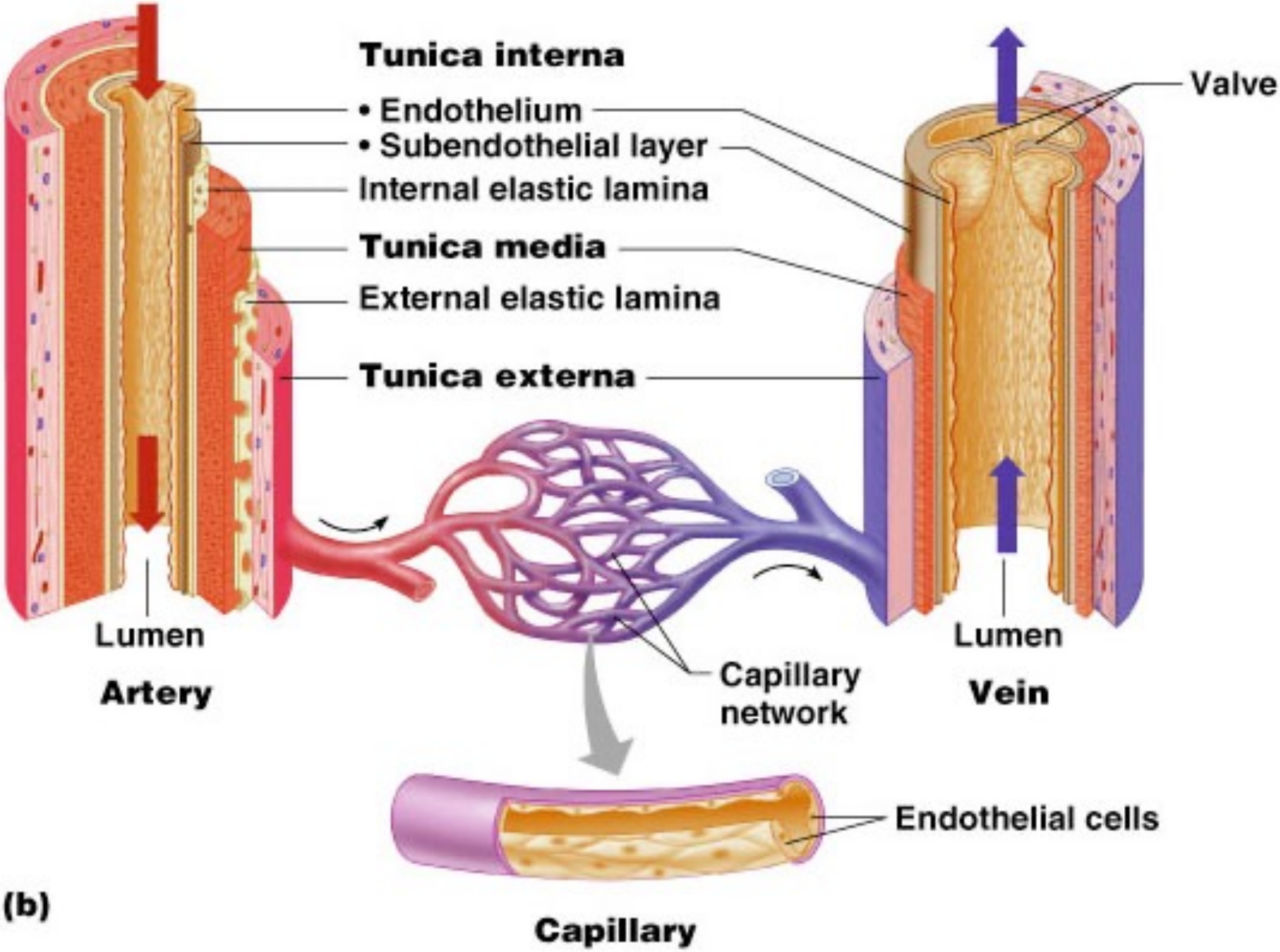
Vasa Vasorum

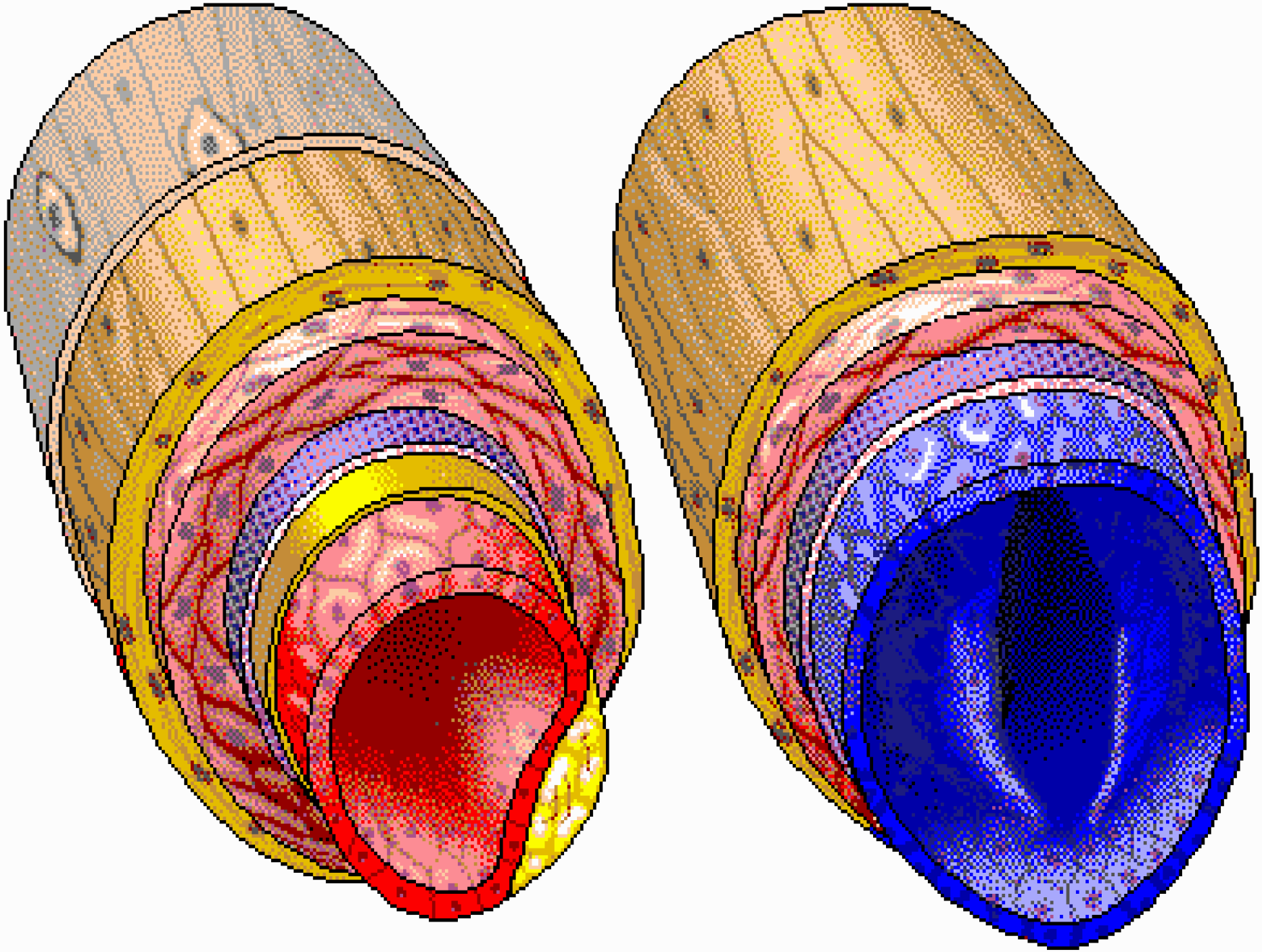
- Network of small vessels outside tunica adventitia
- Supply blood to cells that make up outer layers of large arteries

All layers can become more rigid (i.e. higher collagen content) with age

Basic Vascular Structure: Veins

- **Have thinner walls and larger luminal spaces**
  - No internal or external elastic lamina
  - Tunica intima has thinner connective tissue layer outside of it
  - Tunica media is thinner
- **More "anastomosed" throughout the body**
- **Many have valves to prevent backflow**
  - Especially veins in extremities
  - Projections from the Tunica intima wall


Tunica interna
• Endothelium
• Subendothelial layer
Internal elastic lamina
Tunica media
External elastic lamina
Tunica externa
Valve
Lumen
Artery
Capillary network
Lumen
Vein
Endothelial cells
(b)
Capillary



Vascular Organization

Large elastic arteries

- (e.g., aorta, arch vessels, iliac and pulmonary arteries).
- In these vessels, elastic fibers alternate with smooth muscle cells throughout the media, which expands during systole (storing some of the energy of each cardiac contraction), and recoils during diastole to propel blood distally.

Vascular Organization

Medium-sized muscular arteries

- Coronary and renal arteries
- The media is composed primarily of smooth muscle cells, with elastin limited to the internal and external elastic lamina.
- Smooth muscle cells are circularly or spirally arranged around the lumen.
- Regional blood flow is regulated by smooth muscle cell contraction (vasoconstriction) and relaxation (vasodilation).

Vascular Organization

- **Small arteries** (2 mm or less in diameter) **and arterioles** (20 to 100 μm in diameter) **that lie within the connective tissue of organs.**
- The media in these vessels is mostly composed of smooth muscle cells.
- Arterioles are where blood flow resistance is regulated.

Vascular Organization

- As pressures drop during passage through arterioles, the velocity of blood flow is sharply reduced, and flow becomes steady rather than pulsatile.
- Because the resistance to fluid flow is inversely proportional to the fourth power of the diameter (i.e., halving the diameter increases resistance 16-fold), small changes in arteriolar lumen size have profound effects on blood pressure.

Vascular Organization

- **Capillaries have lumen diameters that approximate those of red cells** (7 to 8 μm).
- Collectively, capillary beds have a very large total cross-sectional area and a low rate of blood flow.
- With their thin walls and slow flow, capillaries are ideally suited to the rapid exchange of diffusible substances between blood and tissue.

Endothelial Cells

- Endothelium is a continuous sheet of cells lining the entire vascular tree that regulates many aspects of blood and blood vessel function.
- Endothelial cells maintain a blood-tissue interface, modulate inflammation , and affect the growth of other cell types, particularly smooth muscle cells.
- Endothelial cells influence the vasoreactivity of the underlying smooth muscle cells by producing both relaxing factors (e.g., nitric oxide [NO]) and contracting factors (e.g., endothelin).

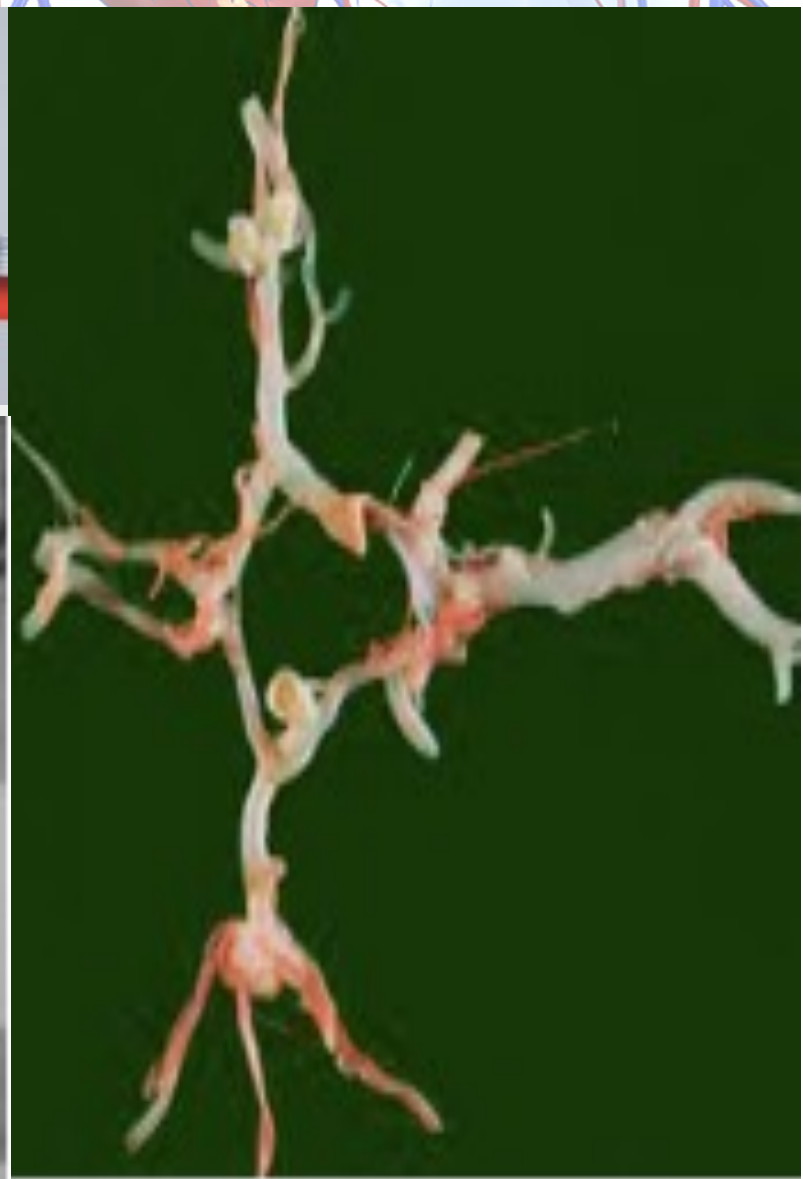
Congenital Anomalies

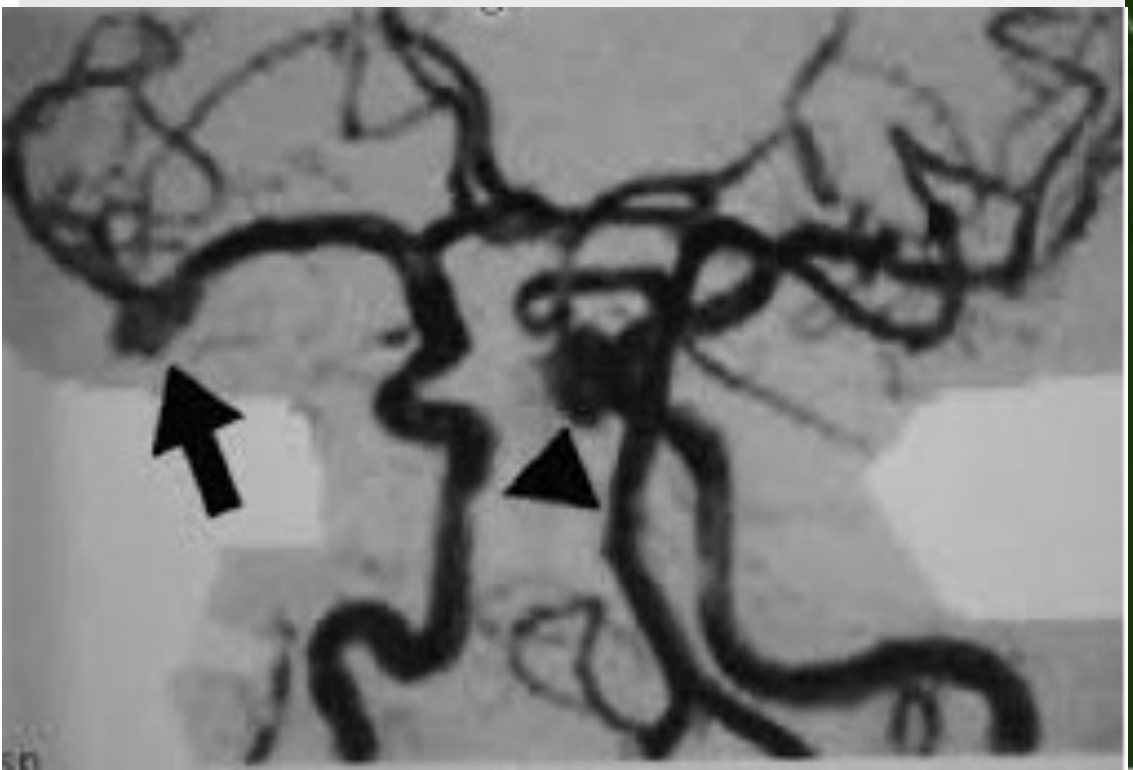
- Although rarely symptomatic, unusual anatomic variants in the vascular supply can cause complications during surgery, such as when a vessel in an unexpected location is injured.
- Cardiac surgeons and interventional cardiologists also must be familiar with coronary artery variants. Among the other congenital vascular anomalies, three deserve further mention:
  - **Berry aneurysms**
  - **Arteriovenous (AV) fistulas**
  - **Fibromuscular dysplasia**

Berry Aneurysms

- A berry aneurysm, which looks like a berry on a narrow stem, is **the most common type of brain aneurysm**. They make up 90 percent of all brain aneurysms
- Thin-walled arterial outpouchings in cerebral vessels, classically at branch points around the circle of Willis
- They occur where the arterial media is congenitally attenuated and can spontaneously rupture causing fatal intracerebral hemorrhage.

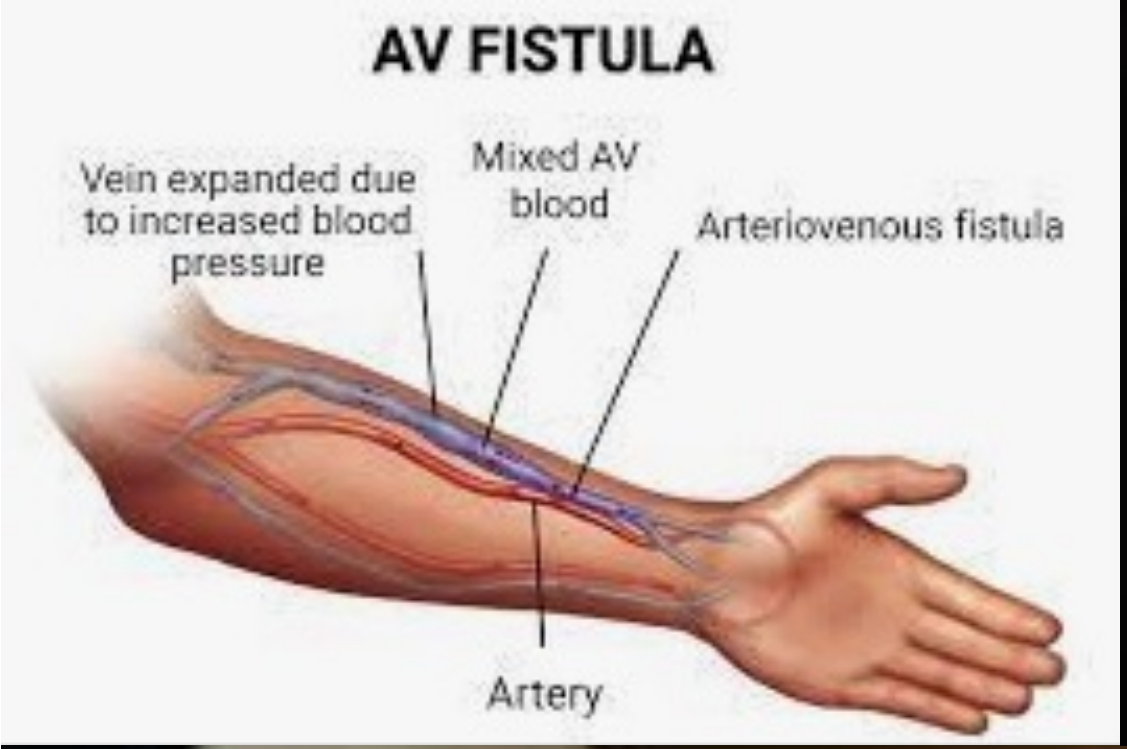
Berry Aneurysms

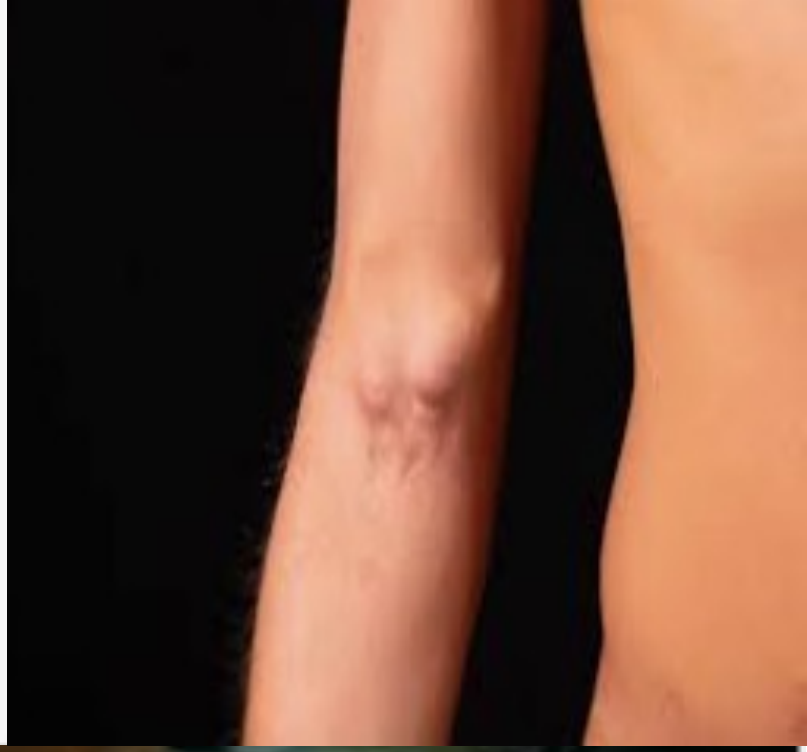


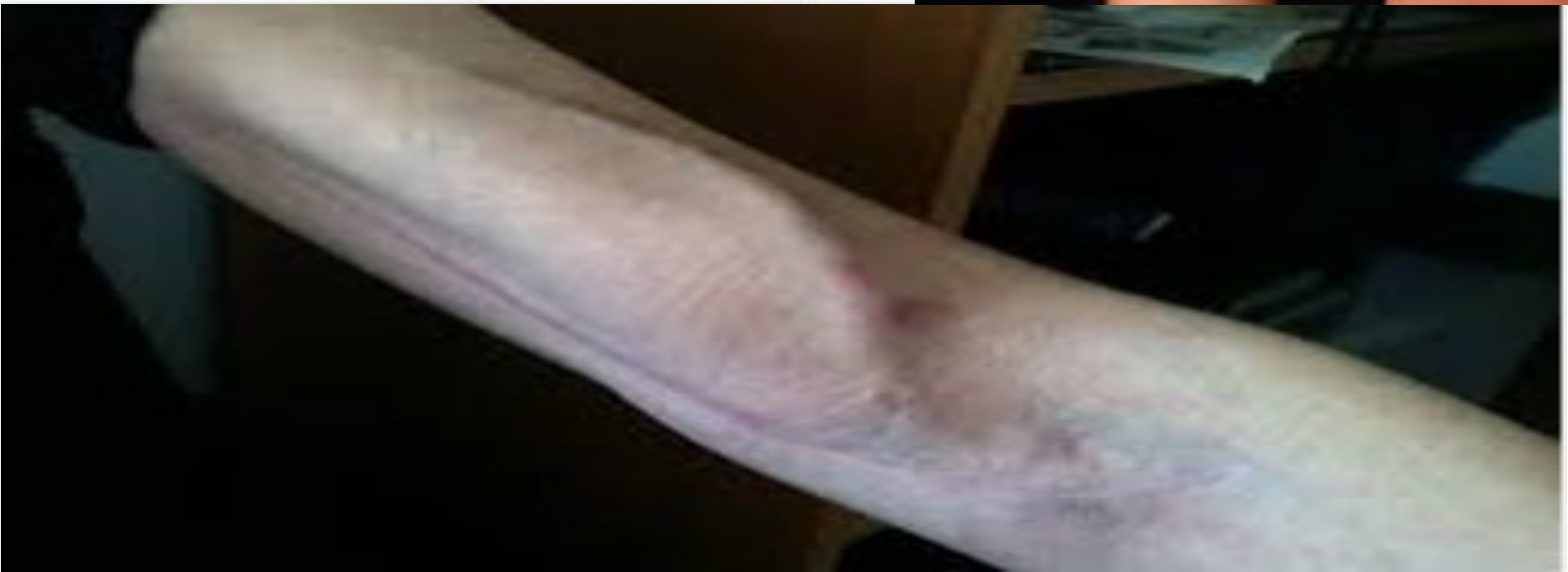


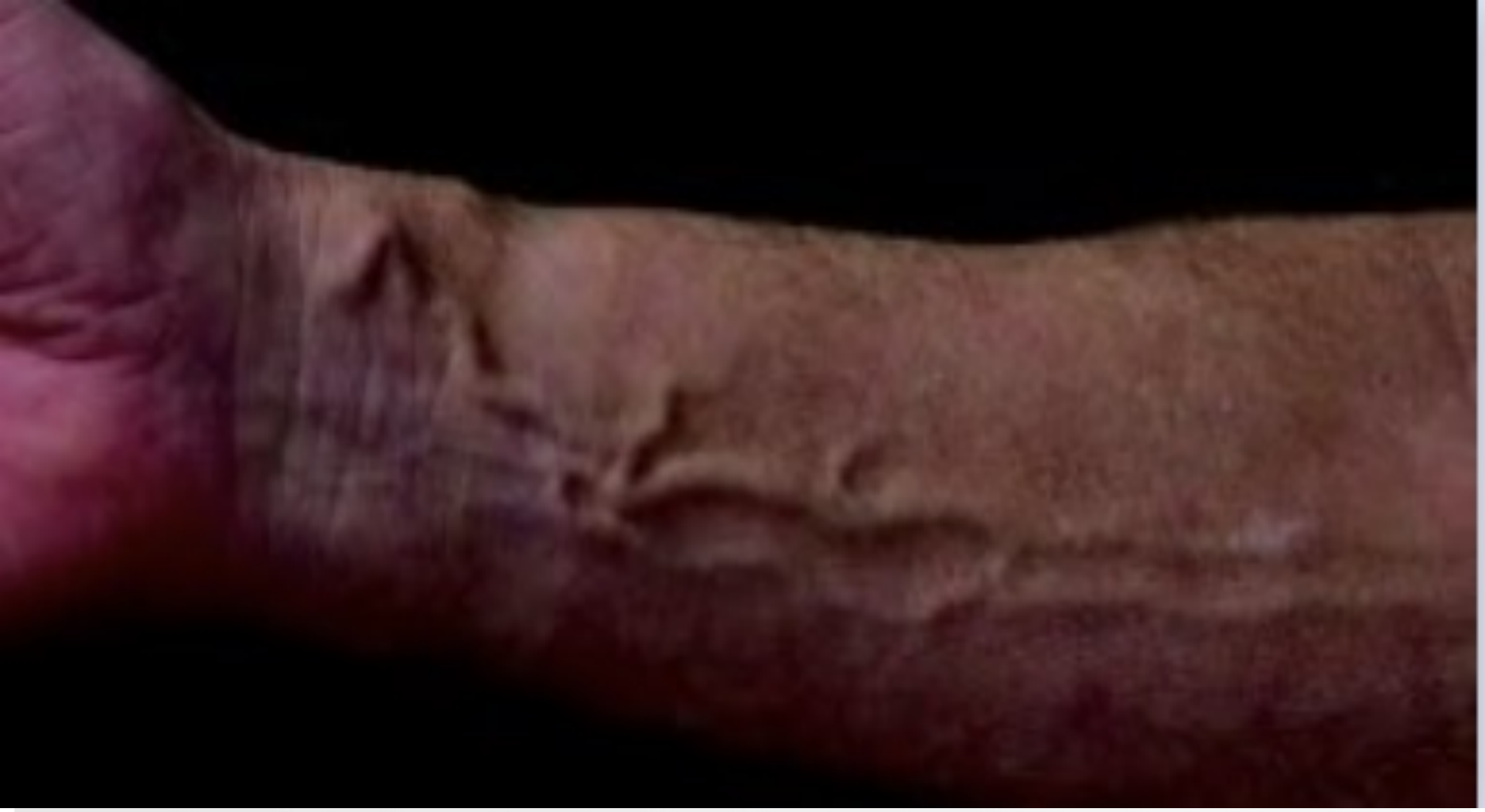
Arteriovenous (AV) Fistulas

- An arteriovenous (AV) fistula is **an irregular connection between an artery and a vein**. Blood flow avoids tiny blood vessels (capillaries) and moves directly from an artery into a vein.
- Abnormal connections between arteries and veins without an intervening capillary bed.
- Can be Iatrogenic.


AV FISTULA
Vein expanded due to increased blood pressure
Mixed AV blood
Arteriovenous fistula
Artery



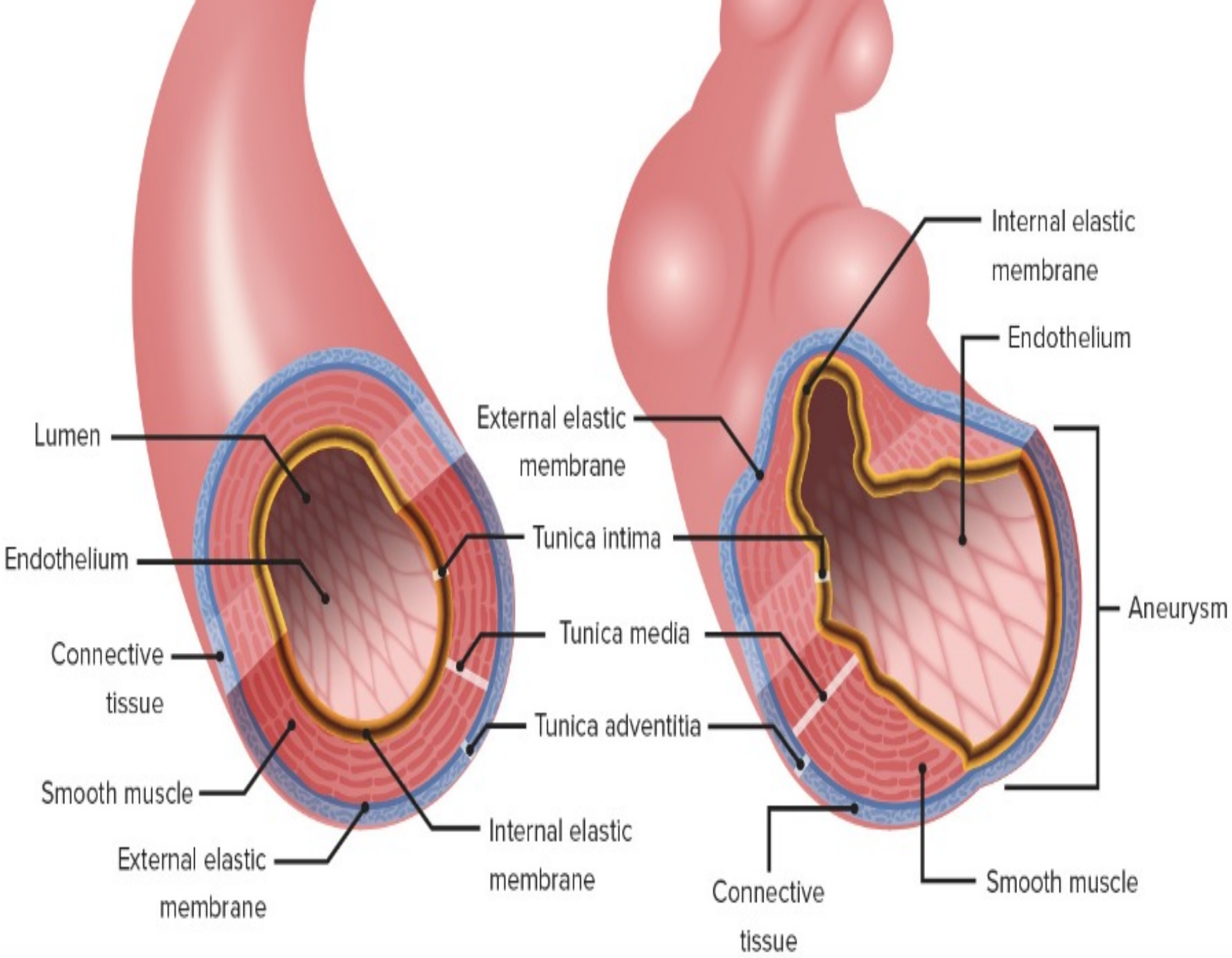




AV Fistula after cardiac catherization

Fibromuscular Dysplasia

- Fibromuscular dysplasia (FMD) is a rare blood vessel disorder in which some of the strong, flexible cells of arteries are replaced with cells that are more fibrous.
- Fibrous cells are less strong and also less flexible.
- This change in composition of the arteries leads to their becoming stiffer and more prone to damage.
- It can manifest at any age but occurs most frequently in young women.


Lumen
Endothelium
Connective tissue
Smooth muscle
External elastic membrane
Internal elastic membrane
Tunica intima
Tunica media
Tunica adventitia
Internal elastic membrane
Endothelium
External elastic membrane
Aneurysm
Connective tissue
Smooth muscle

Hypertensive Vascular Disease

- Hypertension is a major health problem in the developed world.
- Although it occasionally manifests in an acute aggressive form, high blood pressure is much more often asymptomatic for many years.
- This insidious condition is sometimes referred to as benign hypertension, but it is in fact far from harmless.

Hypertensive Vascular Disease

- Besides increasing the risk of stroke and atherosclerotic coronary heart disease, hypertension can lead to cardiac hypertrophy and heart failure (hypertensive heart disease), aortic dissection, multi-infarct dementia, and renal failure.

Epidemiology of Hypertension HTN

- Like height and weight, blood pressure is a continuously distributed variable, and the detrimental effects increase continuously as the pressure rises; no rigidly defined threshold reliably predicts who will suffer ill effects.
- Sustained diastolic pressures greater than 90 mm Hg, or sustained systolic pressures in excess of 140 mm Hg, are associated with an increased risk of atherosclerosis and are therefore used as cutoffs in diagnosing hypertension in clinical practice.

HTN

- By these criteria, some 25% of persons in the general population are hypertensive.
- As noted however, these values are somewhat arbitrary, and in patients with other cardiovascular risk factors (e.g., diabetes), lower thresholds may be applicable.

HTN

- The prevalence of pathologic effects of high blood pressure increases with age and is also higher in African Americans.
  - Without appropriate treatment, some 50% of hypertensive patients die of ischemic heart disease (IHD) or congestive heart failure, and another third succumb to stroke.

HTN

- Reduction of blood pressure dramatically reduces the incidence and clinical sequelae (including death) of all forms of hypertension-related disease.
- Detection and treatment of asymptomatic hypertension constitute one of the few instances in which “preventive medicine” has a major demonstrated health benefit.

HTN

- A small percentage of hypertensive patients (approximately 5%) present with a rapidly rising blood pressure that, if untreated, leads to death in within 1 to 2 years.
- Such malignant hypertension usually is severe (i.e., systolic pressures over 200 mm Hg or diastolic pressures over 120 mm Hg) and associated with renal failure.

HTN

Essential Hypertension

- Accounts for 90% to 95% of all cases

Secondary Hypertension

- Renal
- Cardiovascular
- Neurologic
- Sleep apnea
- Acute stress, including surgery
- Endocrine

Secondary HTN

Cardiovascular

- Coarctation of aorta
- Polyarteritis nodosa
- Increased intravascular volume
- Increased cardiac output
- Rigidity of the aorta

Renal

- Acute glomerulonephritis
- Chronic renal disease
- Polycystic disease
- Renal artery stenosis
- Renal vasculitis

Secondary HTN

Neurologic

- Psychogenic
- Increased intracranial pressure

Sleep apnea

Acute stress, including surgery

Endocrine

- Exogenous hormones
- Hypothyroidism
- Hyperthyroidism
- Acromegaly

Two main pathophysiology occurring with arteries

- **Arteriosclerosis** is the stiffening or hardening of the artery walls.

- **Atherosclerosis** is the narrowing of the artery because of plaque build-up. **Atherosclerosis** is a specific type of **arteriosclerosis**.

Diseases of the Arteries & Veins

Arteriosclerosis

- Chronic disease of the arterial system
  - Abnormal thickening and hardening of the vessel walls
  - Smooth muscle cells and collagen fibers migrate to the tunica intima

Arteriosclerosis

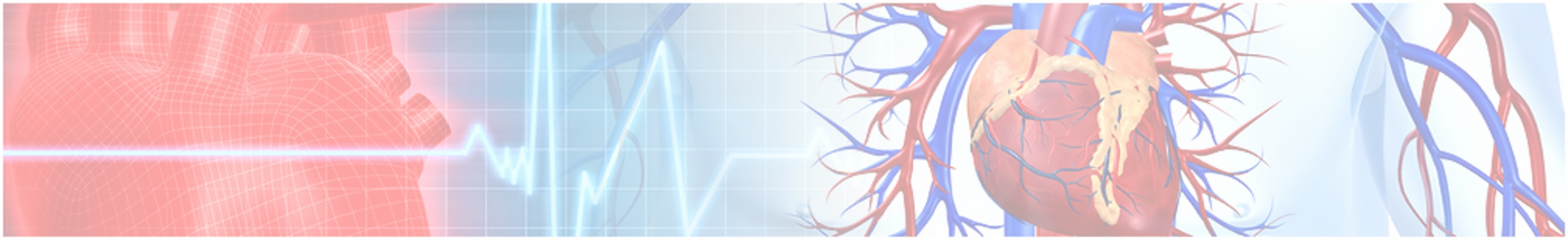
Arteriosclerosis is often referred to as Hypertensive Vascular Disease

- Also called **Arteriosclerosis** (very common)
- Usually found in smaller arteries/arterioles
- Arterial walls get thicker- looks much like hyalinization
- Caused by deposition of proteins, lipids, and BM components in the Tunica intima and media

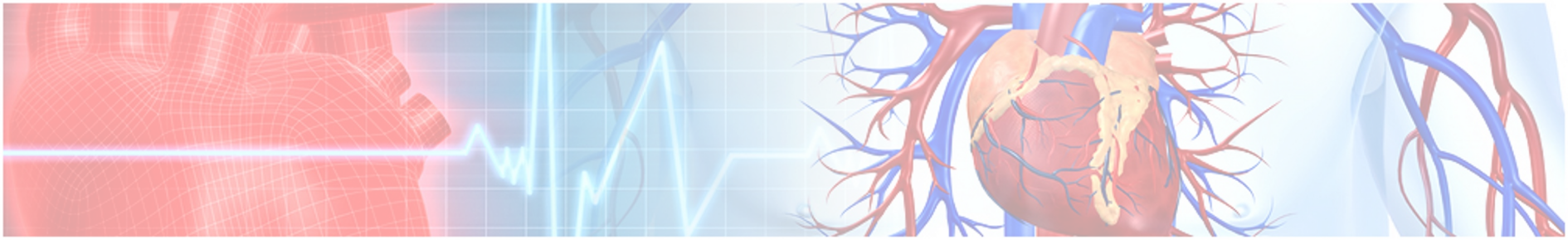
Atherosclerosis

Atherosclerosis

- Form of arteriosclerosis
- Thickening and hardening caused by accumulation of lipid-laden macrophages in the arterial wall
- Plaque development
- Atherosclerosis is virtually ubiquitous among most developed nations but is much less prevalent in Central and South America, Africa, and parts of Asia.



- In the Western world, morbidity and mortality rates for atherosclerosis are higher than for any other disorder, with roughly half of all deaths attributable to this entity.
- Because coronary artery disease is an important manifestation of atherosclerosis, epidemiologic data related to atherosclerosis mortality typically reflect deaths caused by ischemic heart disease (IHD).
- Myocardial infarction is responsible for almost one fourth of all deaths in the United States.



- The mortality rate for IHD in the United States is among the highest in the world, approximately five times higher than that in Japan.
  - However, IHD is increasing in Japan, where it is now the second leading cause of death.
  - Furthermore, Japanese emigrants who come to the United States and adopt American life styles and dietary customs acquire the same atherosclerosis risk as for U.S.-born persons, emphasizing the important etiologic role of environmental factors.

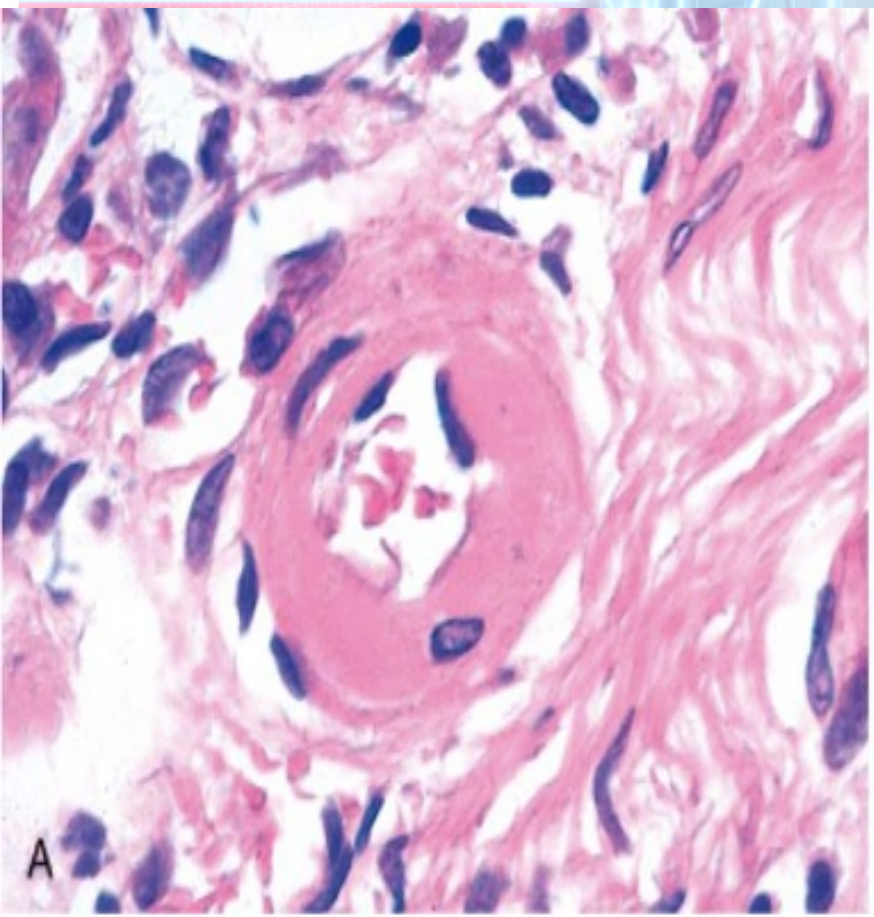
Nonmodifiable Rick Factors (Constitutional)

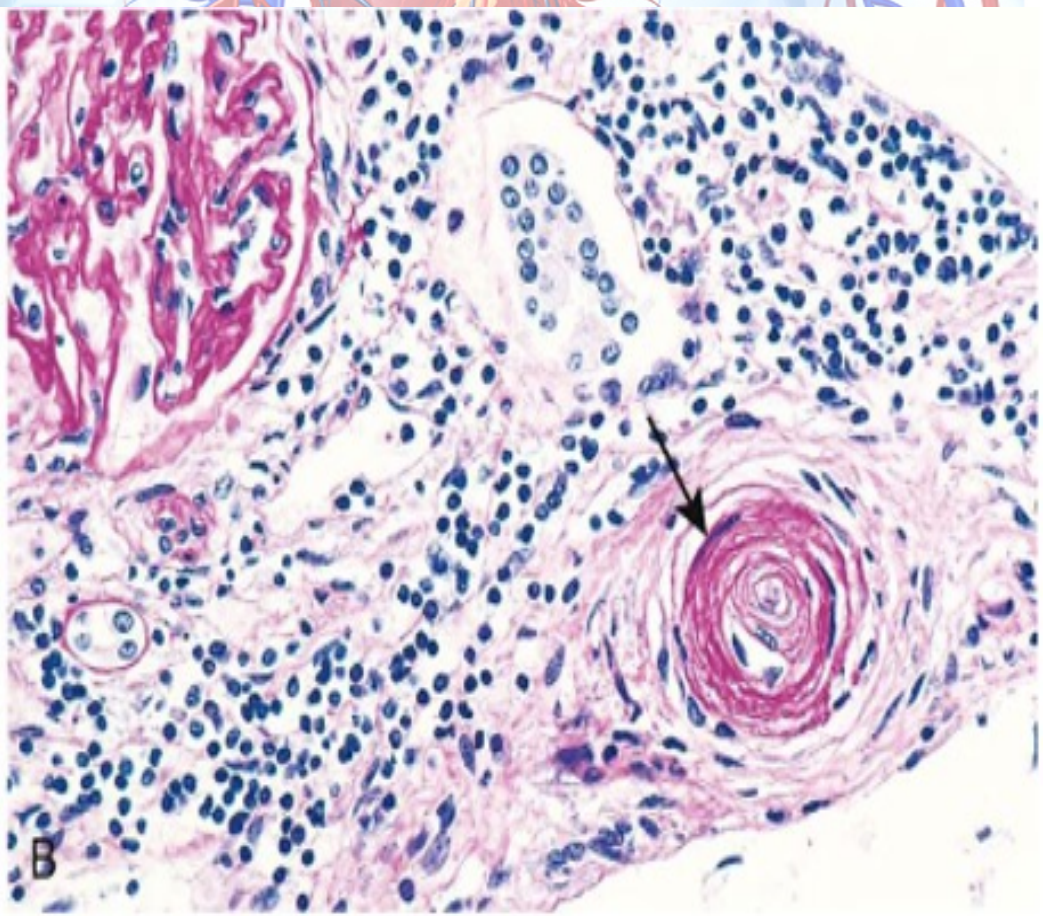
- Genetic abnormalities
- Family history
- Increasing age
- Male gender

Modifiable Risk Factors

- Hyperlipidemia
- Hypertension
- Cigarette smoking
- Diabetes
- Inflammation

Hypertensive Vascular Disease





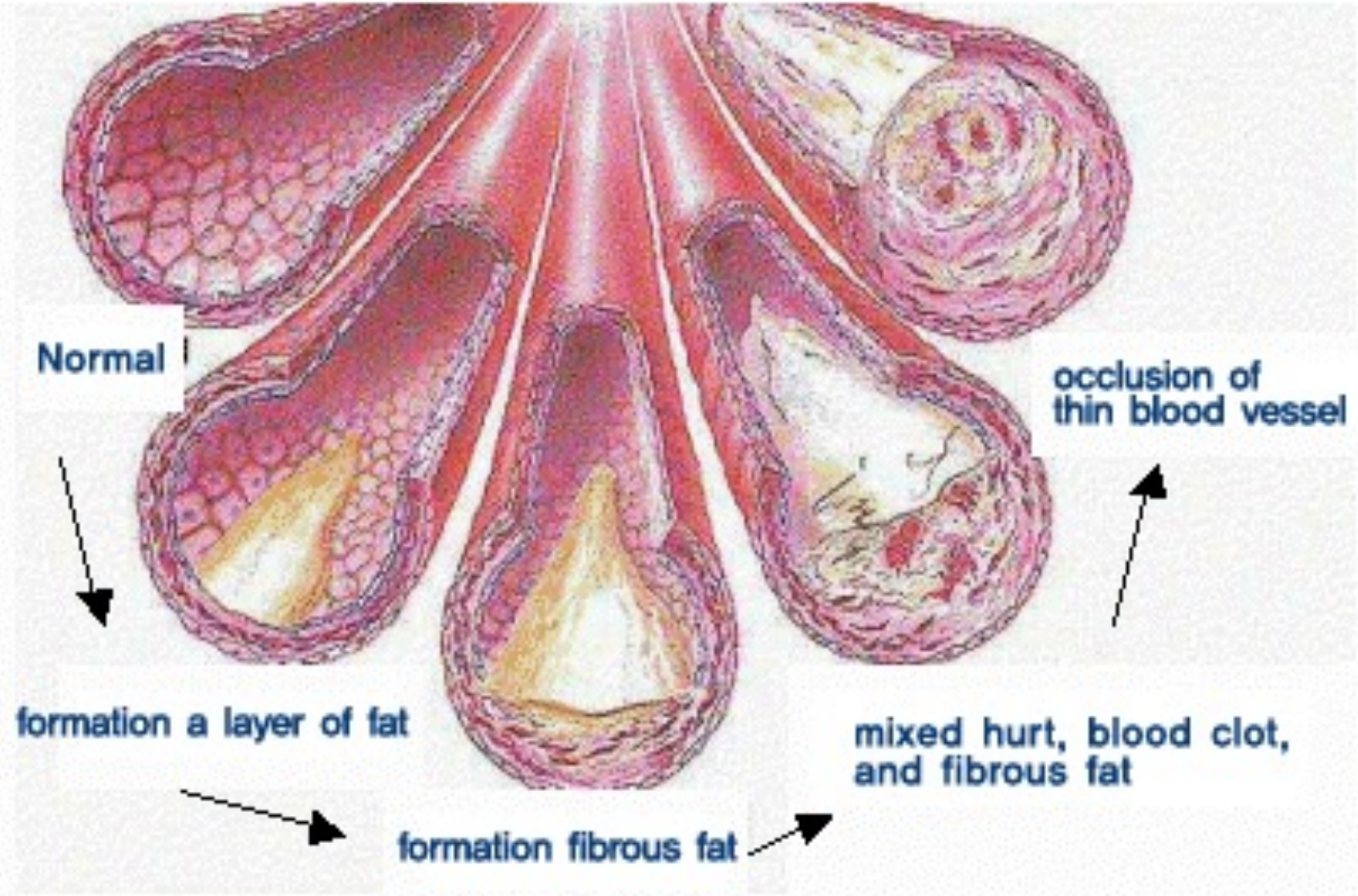
Hyaline arteriosclerosis on left showing thick hyaline around lumen. **Hyperplastic sclerosis** on the right showing an onion skinning which nearly obliterates the lumen,

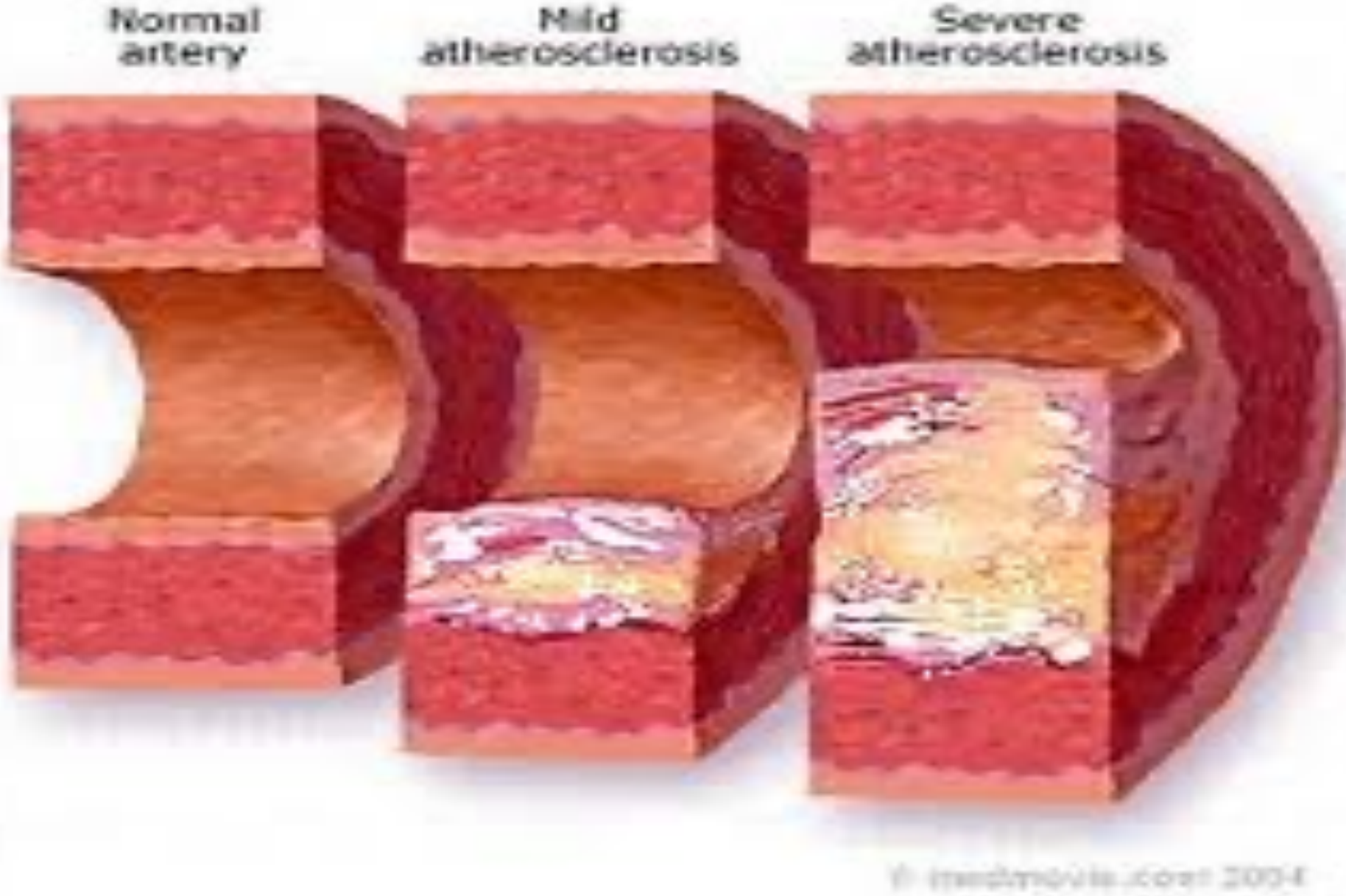
Diseases of the Arteries & Veins

Atherosclerosis

- Progression
  - Inflammation of endothelium
  - Cellular proliferation
  - Macrophage migration and adherence
  - LDL oxidation (foam cell formation)
  - Fatty streak
  - Fibrous plaque
  - Complicated plaque
- Result in—inadequate perfusion, ischemia, necrosis

Progress of arteriosclerosis




Normal artery
Mild atherosclerosis
Severe atherosclerosis

Atherosclerosis Risk Factors

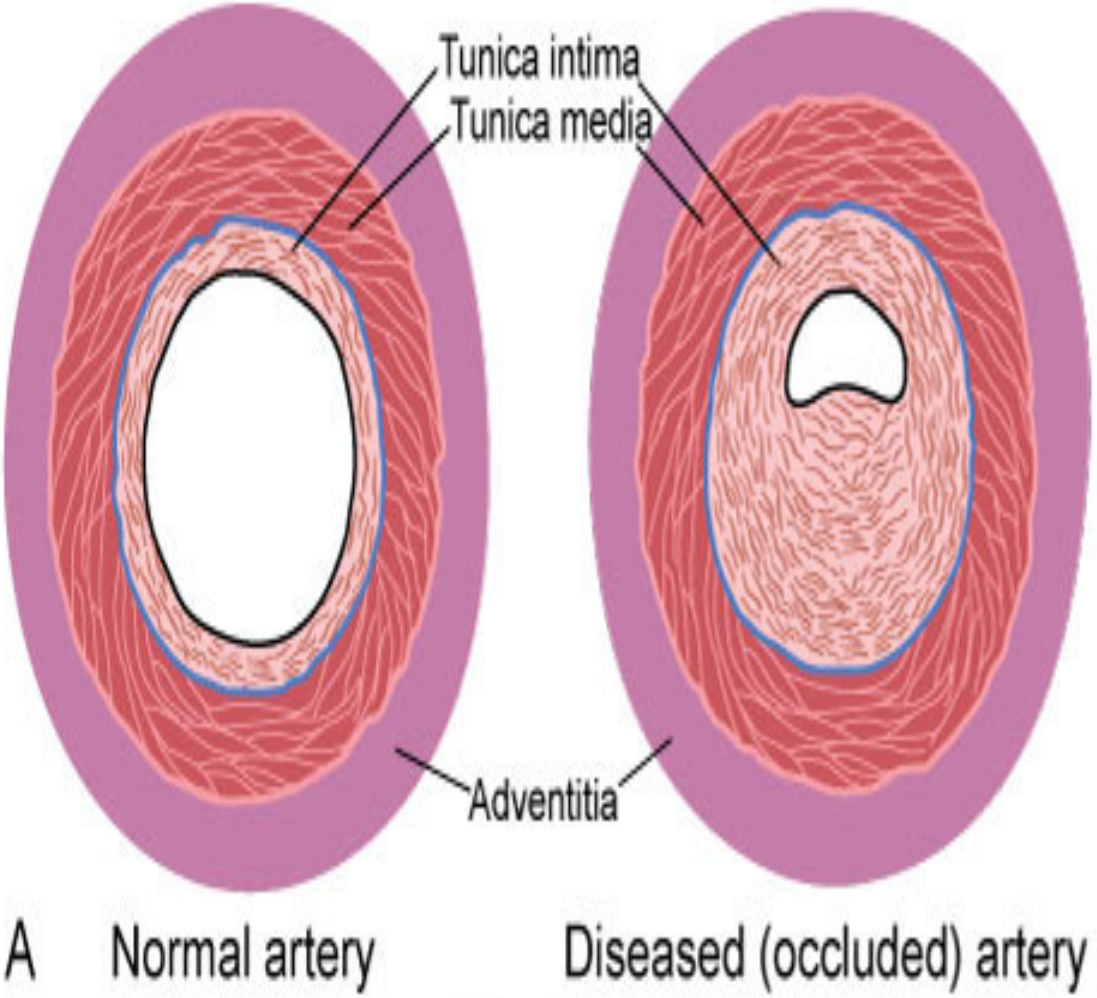
Hyperlipidemia- elevated levels of fat in the blood

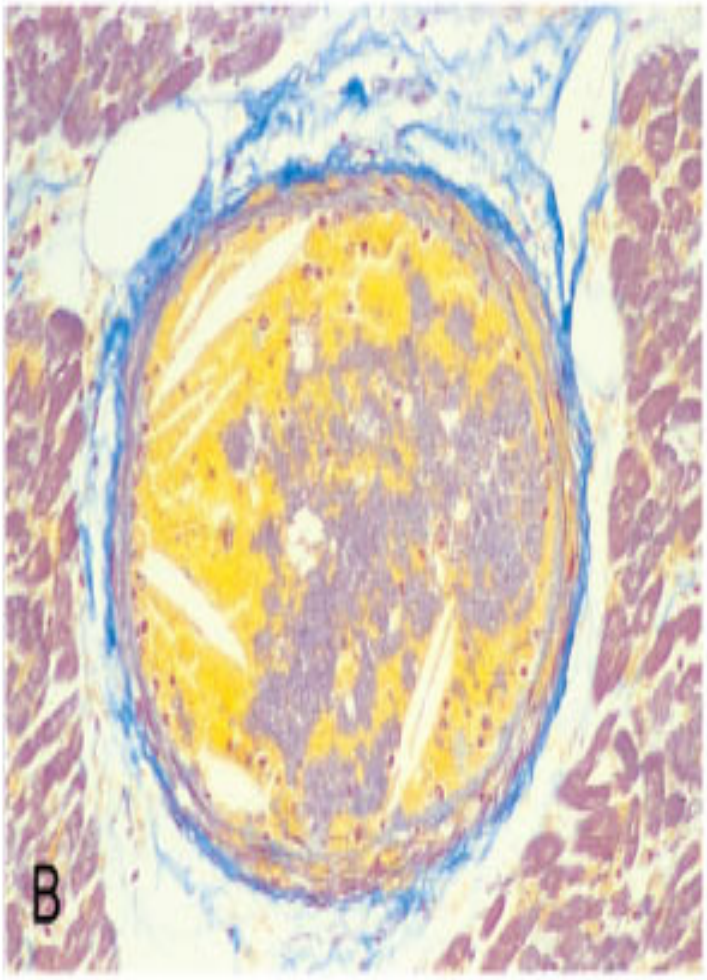
- High Blood LDL (**Low Density Lipoproteins**)- cholesterol carrying particles between liver and other tissues that metabolize cholesterol
- **Familial Hypercholesterolemia-** elevated plasma cholesterol due to defective LDL receptors

Atherosclerosis Risk Factors

- Hypertension
- Smoking
- Diabetes Mellitus
- Gender (males more predisposed)
- Genetic Predisposition
- Secondary factors- obesity, stress, inadequate exercise, excessive (or no) alcohol use

Arteriosclerosis





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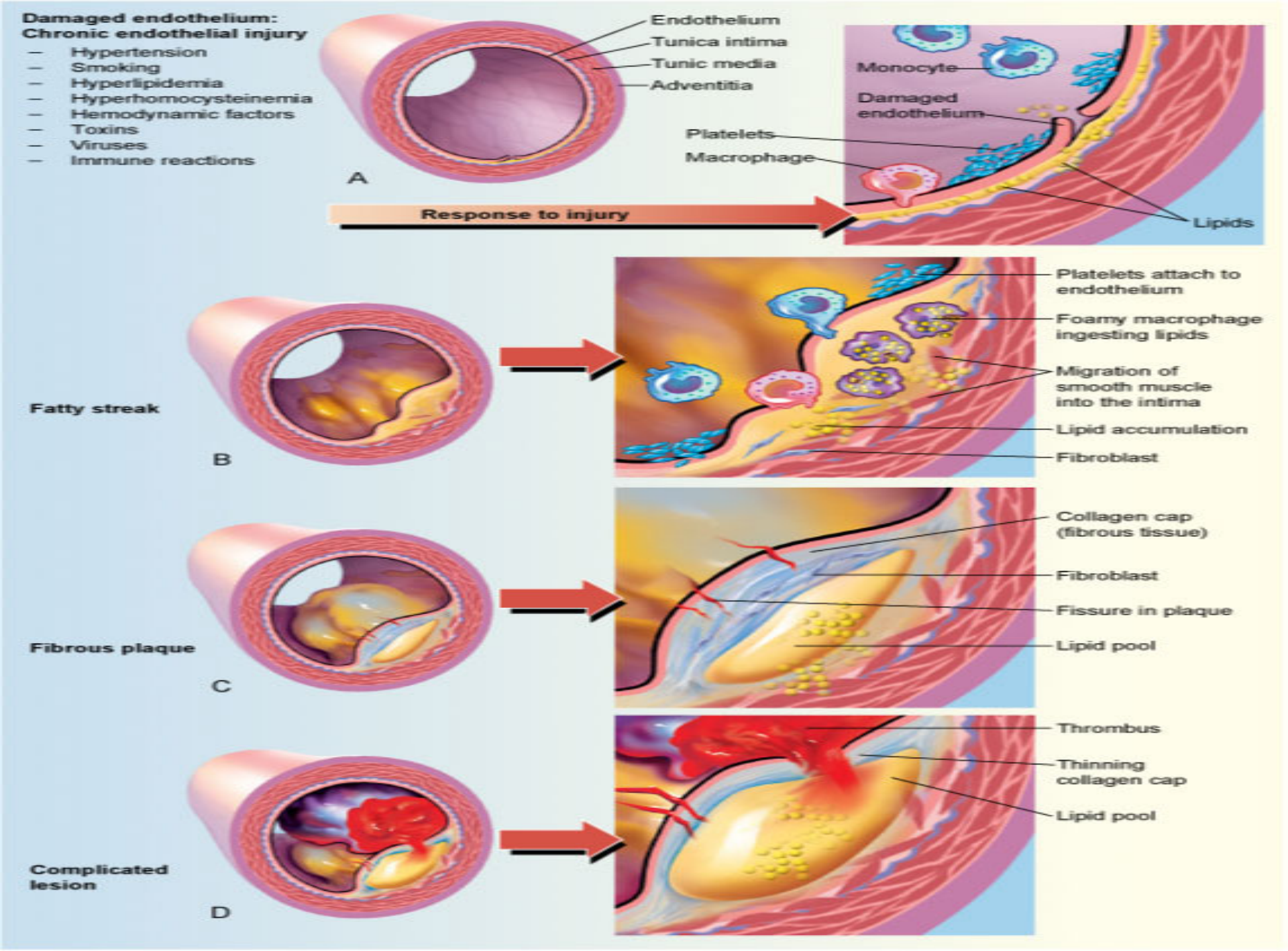
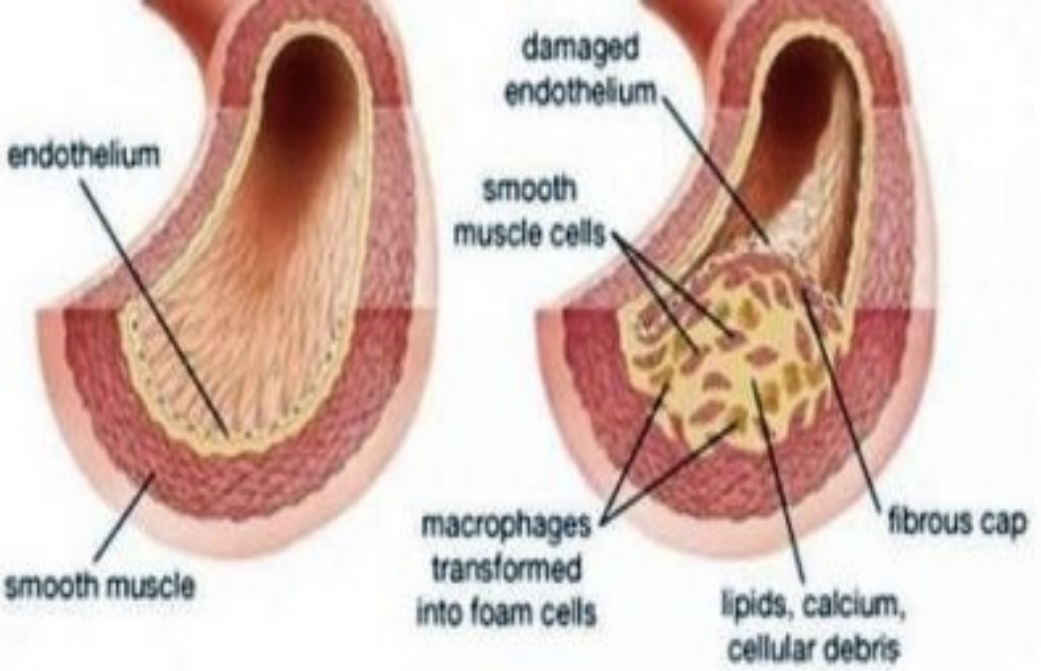
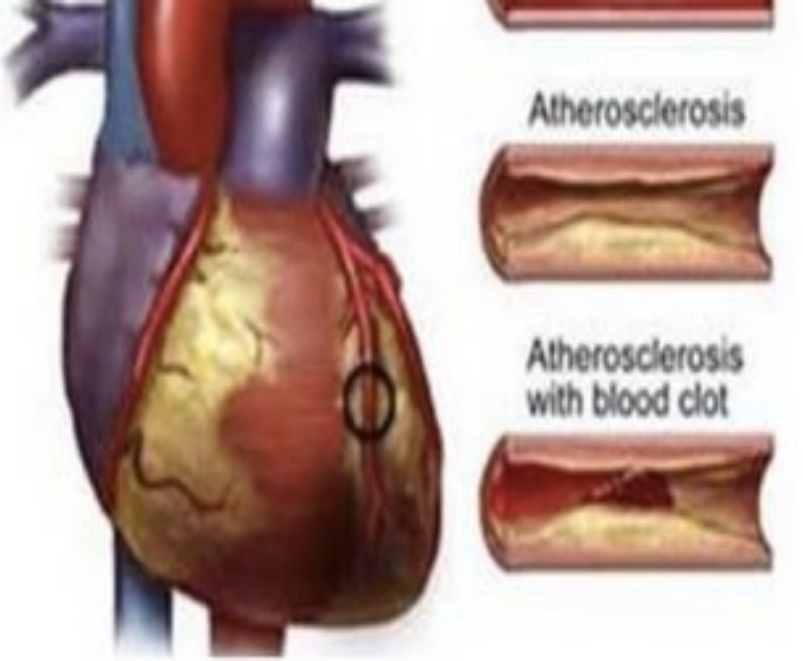
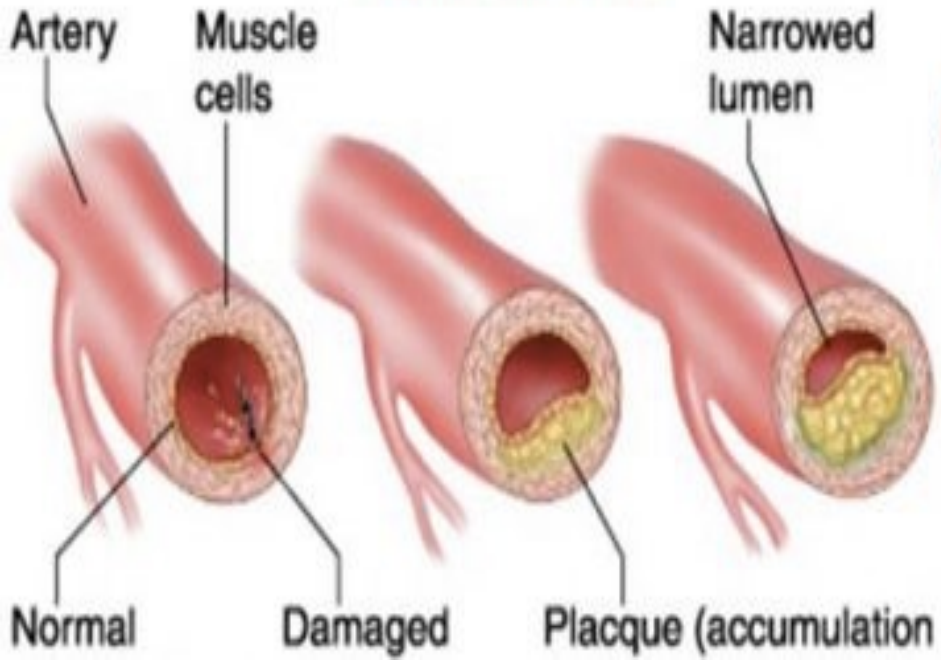


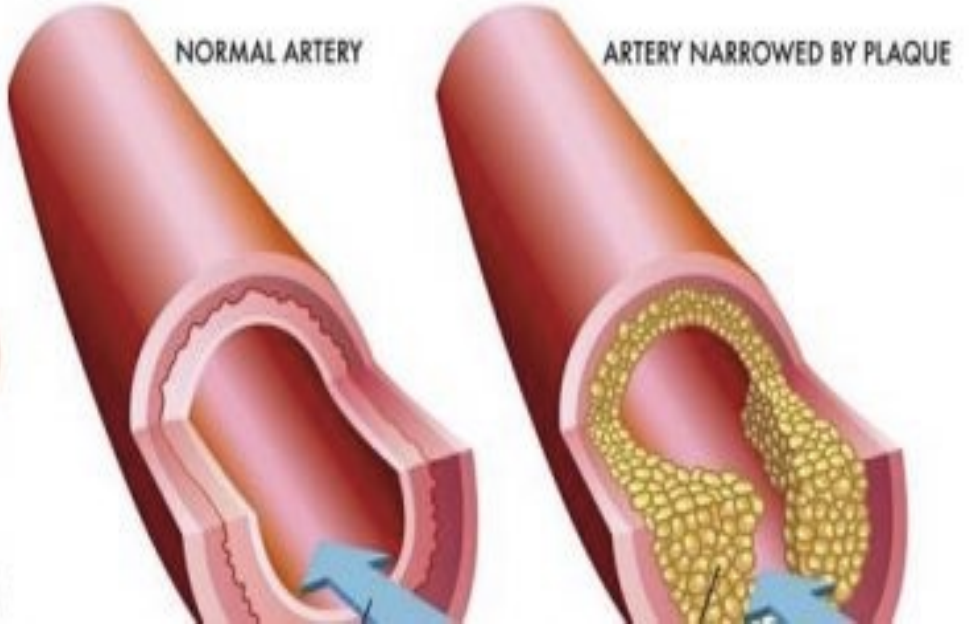


Arteriosclerosis



ATHEROSCLEROSIS





Clinical Consequences of Atherosclerotic Disease

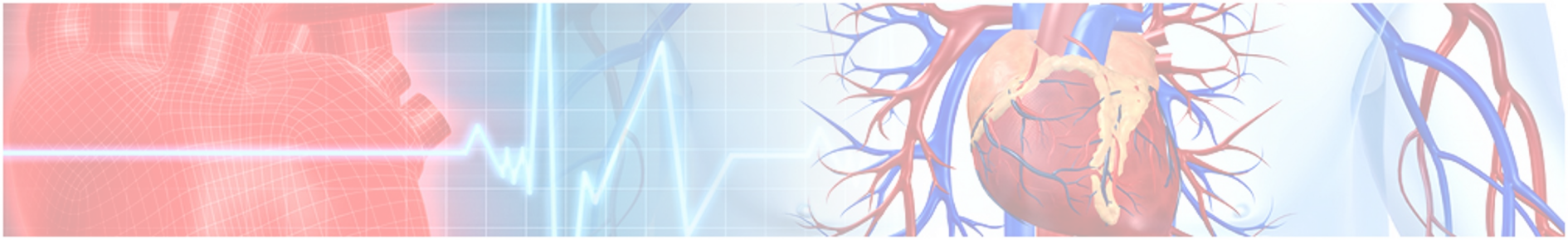
- Large elastic arteries (e.g., aorta, carotid, and iliac arteries) and large and medium-sized muscular arteries (e.g., coronary, renal, and popliteal arteries) are the vessels most commonly involved by atherosclerosis.
- Accordingly, atherosclerosis is most likely to present with signs and symptoms related to ischemia in the heart, brain, kidneys, and lower extremities.

Clinical Consequences of Atherosclerotic Disease

- Myocardial infarction (heart attack), cerebral infarction (stroke), aortic aneurysms, and peripheral vascular disease (gangrene of extremities) are the major clinical consequences of atherosclerosis.

Three Complications of Atheroma

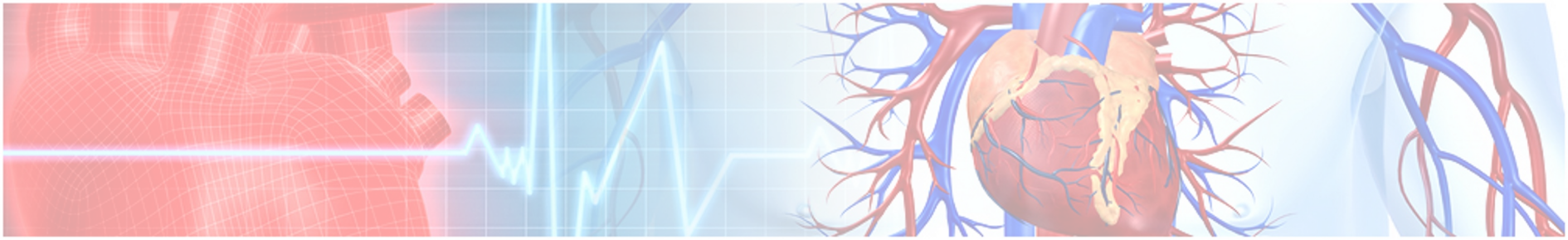
- **Ischemia**
  - The lumen of the artery narrows and distal vascular flow is lowered
- **Thrombus**
  - The presence of the atheroma increases the clot risk, which forms a thrombus
  - This can close off the lumen causing an infarct – tissue death



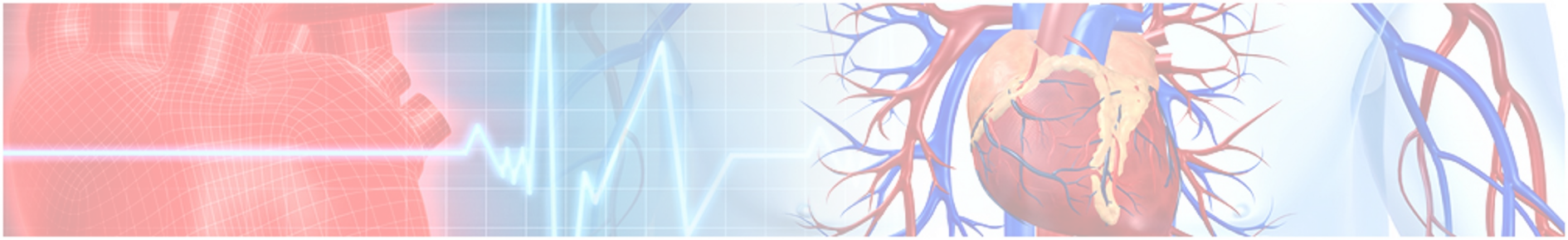
- **Aneurysm**
  - The atheroma can weaken the arterial wall causing a ballooning of the wall which can burst

Acute Plaque Change

- Plaque erosion or rupture typically triggers thrombosis, leading to partial or complete vascular obstruction and often tissue infarction. Plaque changes fall into three general categories:
  - Rupture/fissuring, exposing highly thrombogenic plaque constituents
  - Erosion/ulceration, exposing the thrombogenic subendothelial basement membrane to blood
  - Hemorrhage into the atheroma, expanding its volume



- It is now recognized that plaques responsible for myocardial infarctions and other acute coronary syndromes often are asymptomatic before the acute event, which superimposes thrombosis on a lesion that previously did not produce significant luminal occlusion.
- The worrisome conclusion is that large numbers of asymptomatic persons are at risk for a catastrophic coronary event.



- Factors extrinsic to plaques also are important.
- Adrenergic stimulation (as with intense emotions) can increase systemic blood pressure or induce local vasoconstriction, thereby increasing the mechanical stress on a given plaque.

- One explanation for the pronounced circadian periodicity in the onset of heart attacks (peak incidence between 6 AM and 12 noon) is the adrenergic surge associated with waking and rising, which is sufficient to cause blood pressure spikes and heightened platelet reactivity.

Hemodynamic Disorders

- Any disorder that arises from an interruption in blood flow in the body

- **Thrombus -** Mass of platelets, RBCs, WBCs, and fibrin
- **Thrombosis -** Thrombus forming in an inappropriate place (i.e. vessel)

Ischemia

- Reduced blood supply results in decreased perfusion of cells, tissues and organs distal to the narrowed lumen
- The ischemia causes the tissues to become hypoxic, which makes it hard for the cells and tissues to carry on normal cell metabolism.
- This causes aerobic (In the presence of oxygen) metabolism to be converted into anaerobic (without oxygen) metabolism.
- The anaerobic metabolism causes and accumulation of waste products to accumulate in the tissues.

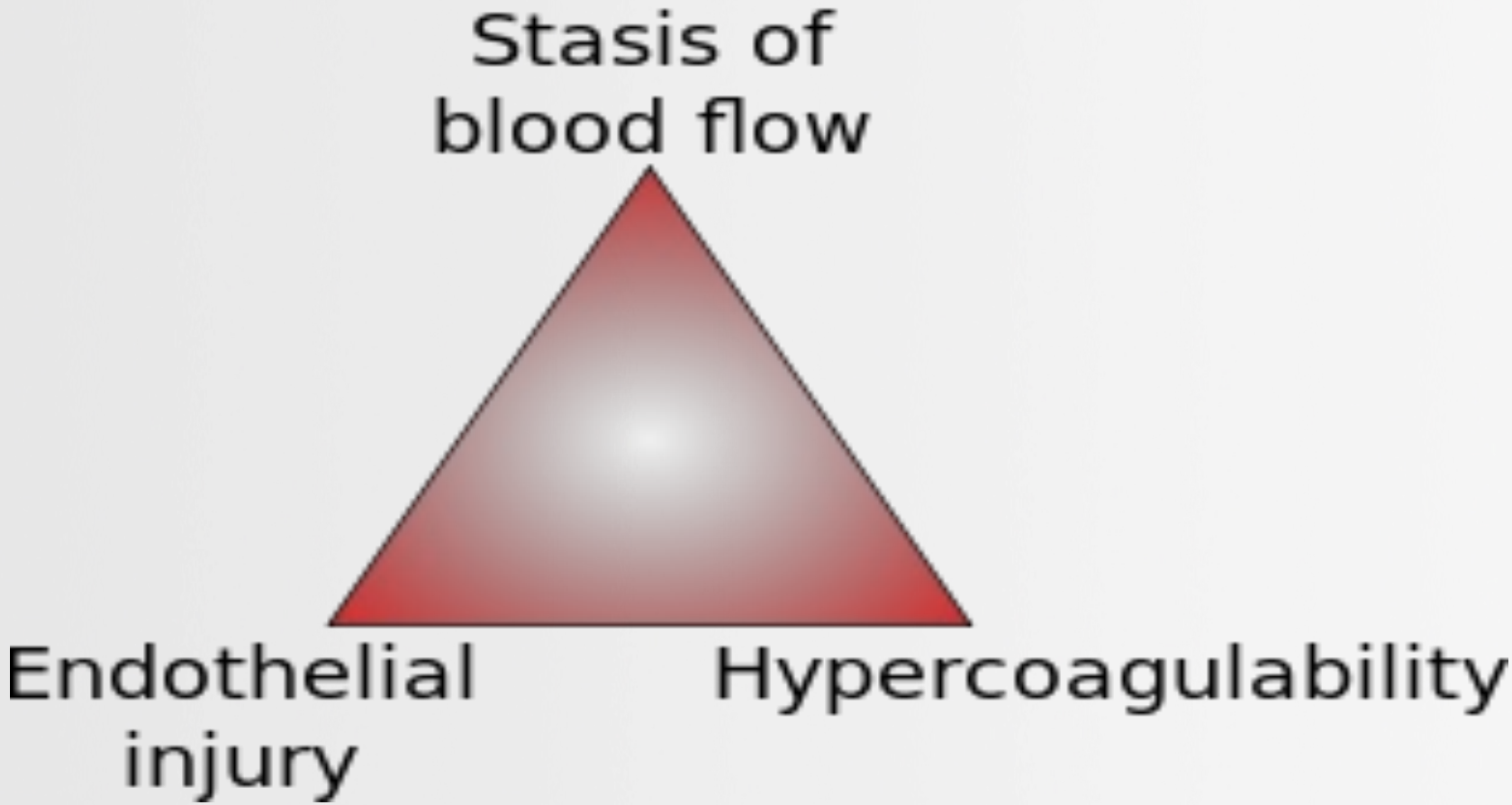
Effects of lactic acid on arterial walls

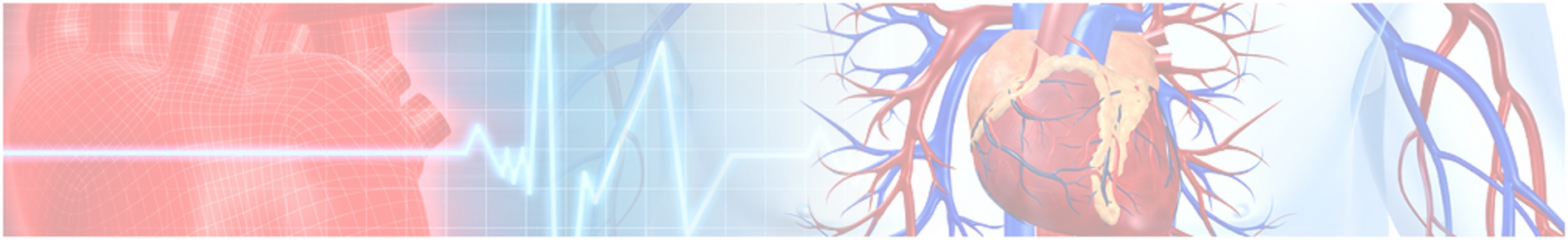
- The anaerobic metabolism is only intended to be a short term emergency measure in the body and was not meant to be a long term fix.
- If it is not washed away by sufficient blood flow, it tends to stick to the walls which causes more hypoxia.
- When this occurs in muscles it causes cramping and pain.
- Smooth muscles around organs can cramp and close off.
- When a smooth muscle cramps around a hollow organ it is referred to as colic.
- Lactic acid accumulation in the heart is called angina.

Thrombus

- Because of the platelets, blood is meant to clot to protect us from hemorrhaging
- Blood inside the lumen is liquid and when it clots it is a thrombus, which is stationary
- There are three main causes of thrombus formation in the arterial walls:
  - Disorders of the endothelial lining
  - Decreased rates of blood flow
  - Increased coagulability of blood

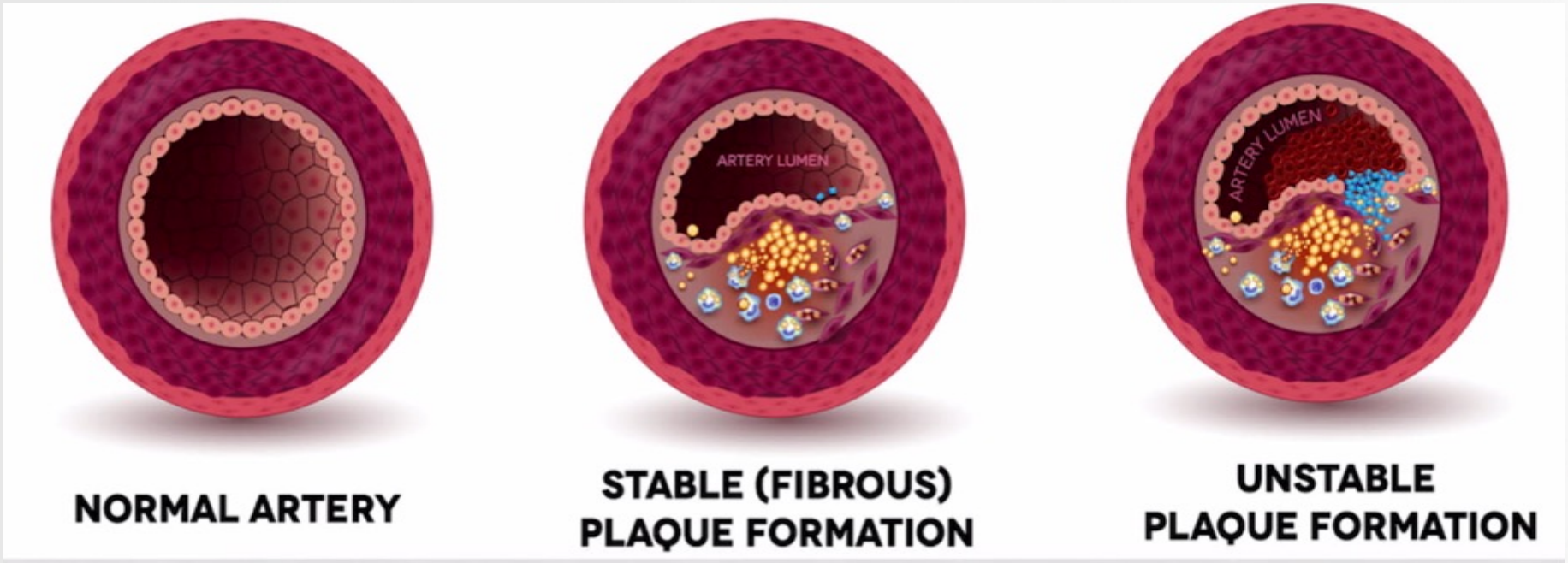
Virchow's Triad

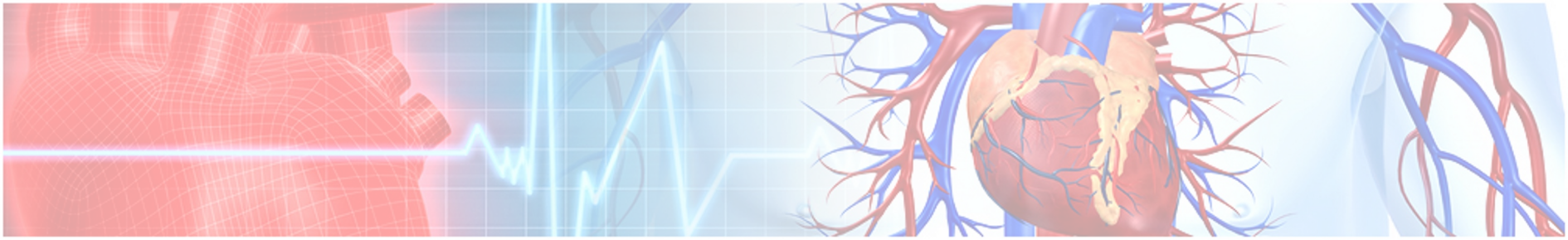




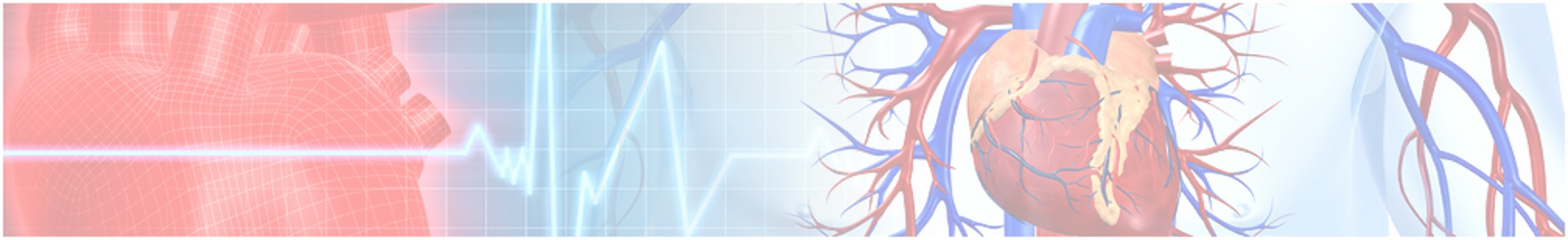
- **Disorders of the endothelial layer**
  - The atheroma under the endothelial lining greatly increases the risk of a thrombus development or plaque formation
    - A stable or simple plaque has an intact fibrous cap and a layer of vascular endothelium separating the plaque from coming in contact with the blood. This makes a thrombus formation unlikely because platelets and other clotting factors do not get to the core

- An unstable plaque occurs when there is a crack or fissure or ulcer of the endothelial lining and clotting factors can get to the core. Inflammatory chemicals can also cause this

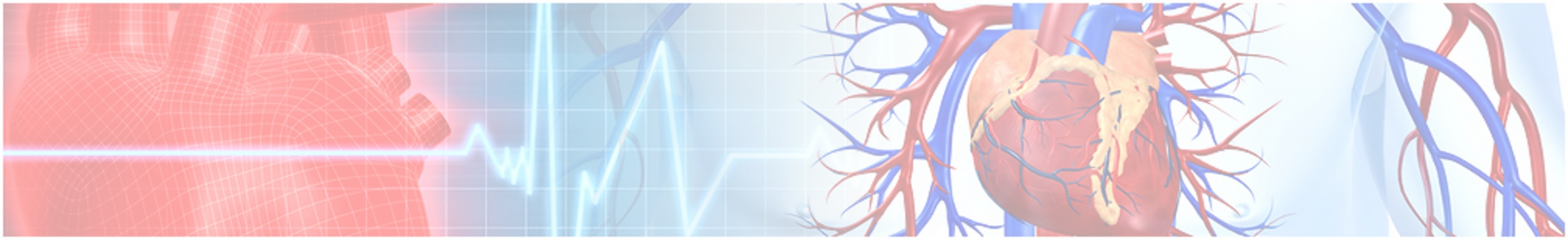




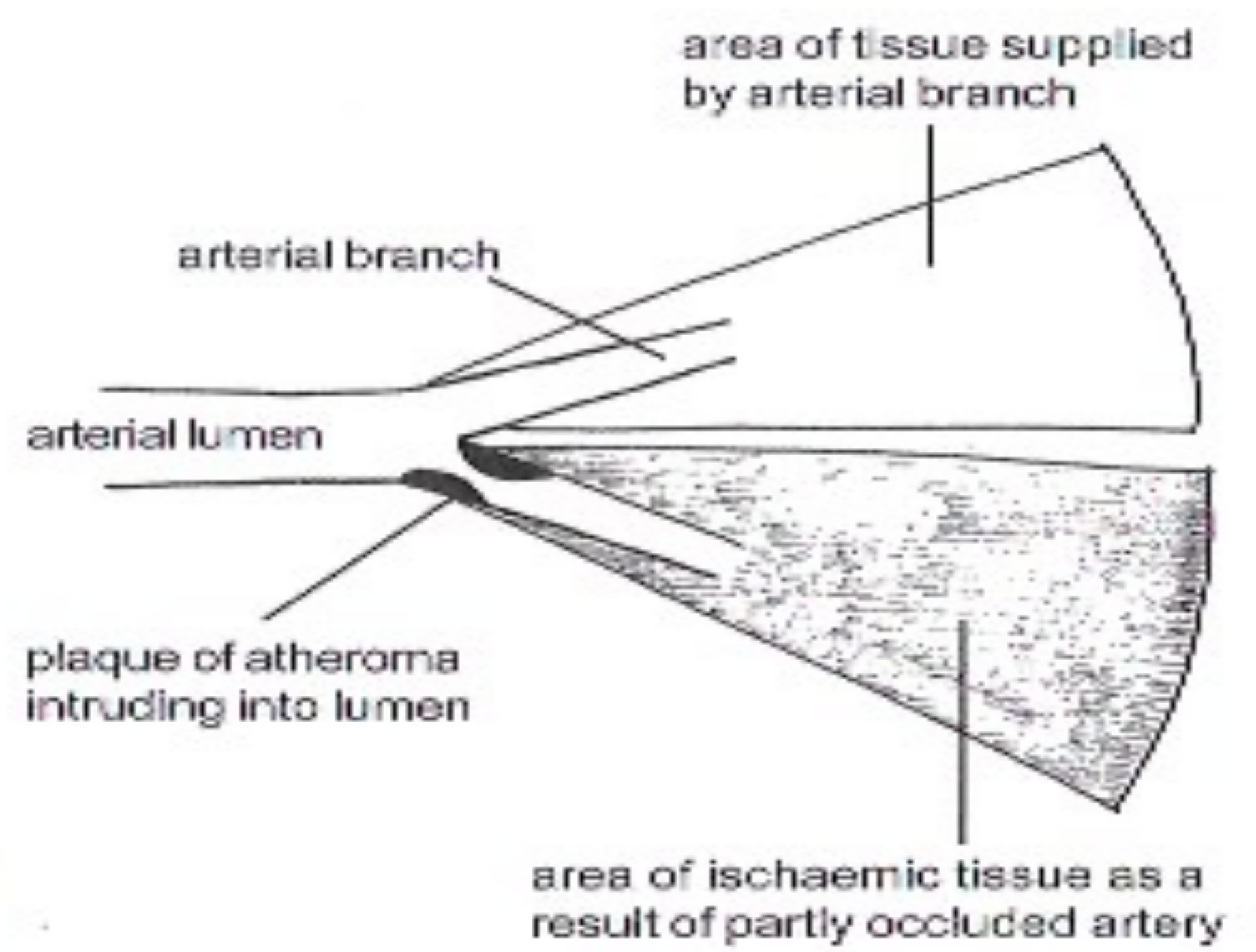
- **Sluggish or abnormal blood flow**
  - This is a risk because when the blood moves slower, it allows platelets and other clotting factors more time to stick to the arterial walls.
  - In the legs, this can cause DVT – deep vein thrombosis
  - In AF, atrial fibrillation, the atria do not fully contract and some blood stays behind and can form clots



- **Increased blood coagulability**
  - Anything that changes the viscosity of blood to being thicker increases the risk of thrombus formation.
  - Can be caused by dehydration or at high altitudes, where more RBC are needed to survive.

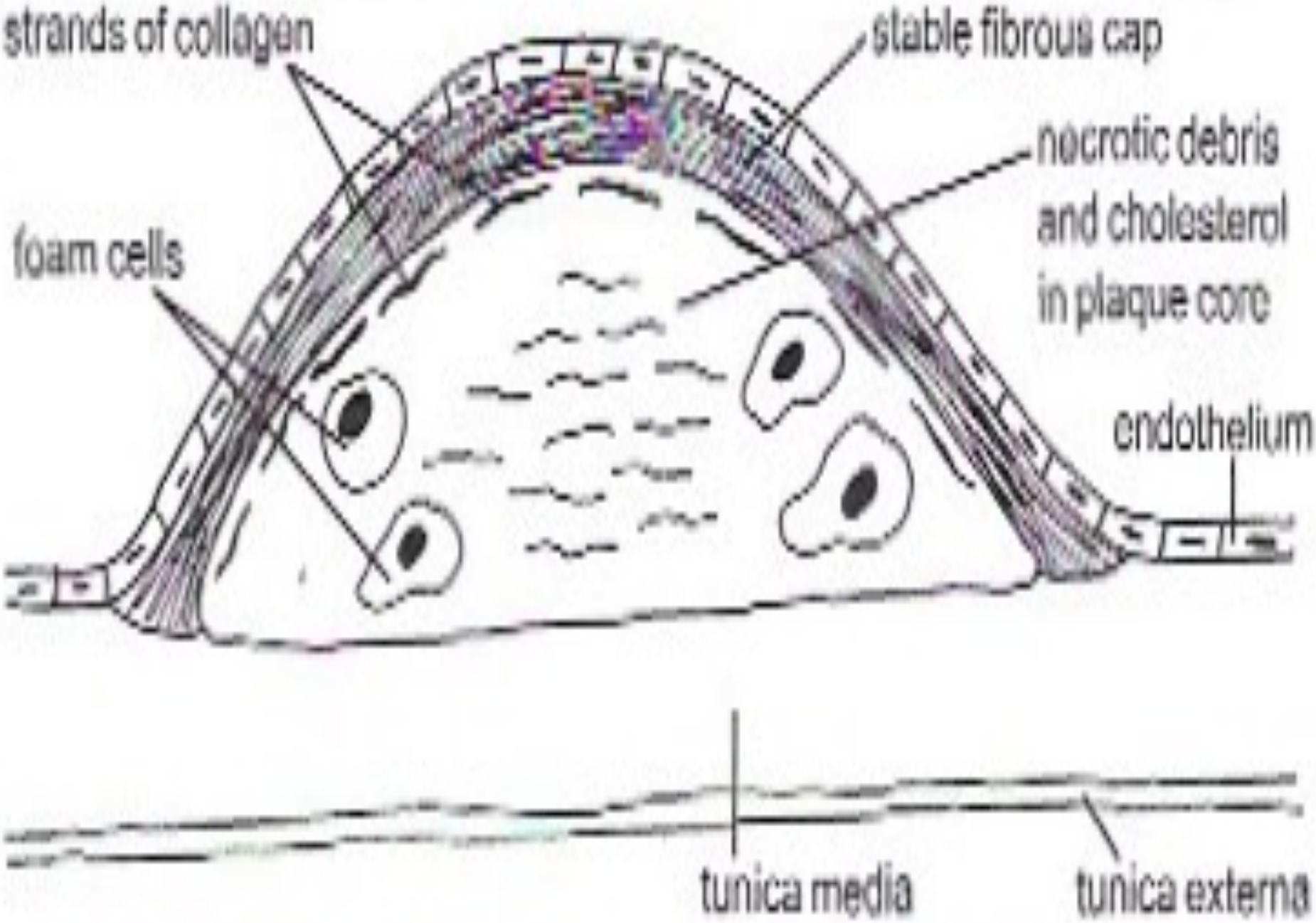


- Smokers produce more RBCs because the hemoglobin in inactivated in the presence of carbon monoxide.
- Thrombocytosis – increased platelet count
- Whenever there is a hemorrhage (surgery, trauma, childbirth), more young platelets are released from the spleen. Young platelets are more likely to stick to arterial walls.

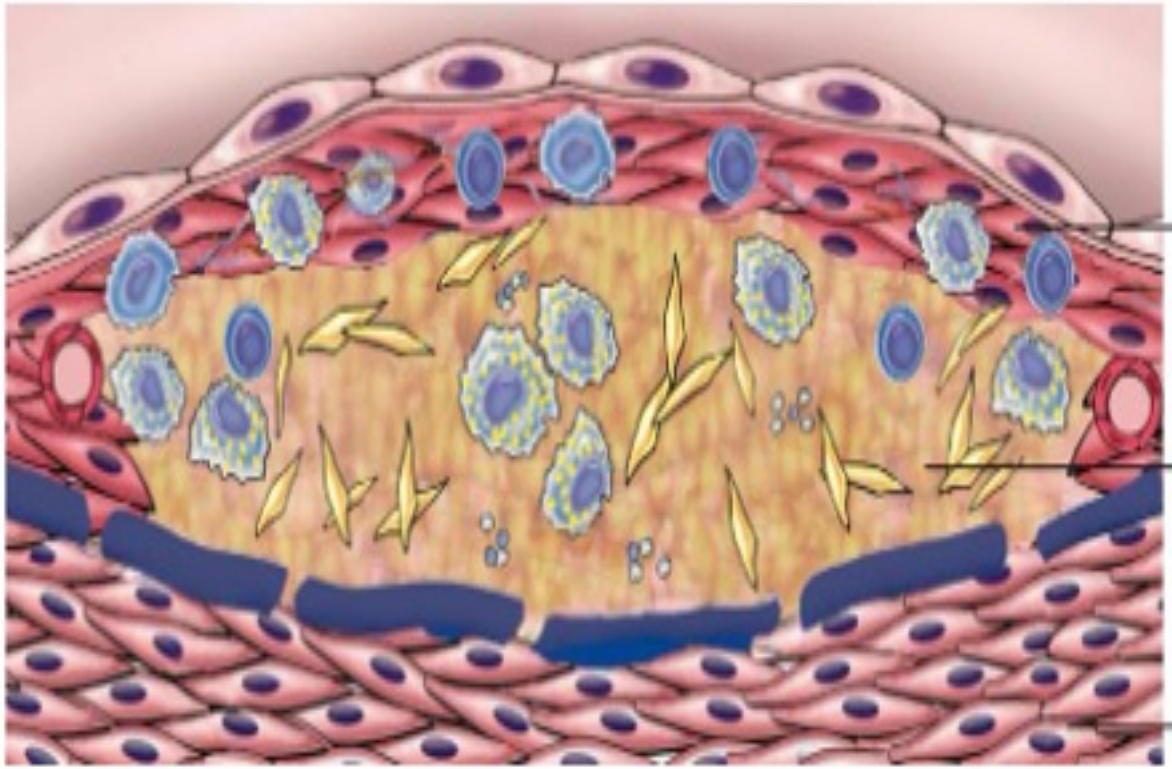

area of tissue supplied by arterial branch
arterial branch
arterial lumen
plaque of atheroma intruding into lumen
area of ischaemic tissue as a result of partly occluded artery

Stable Thrombus Plaque

- The thrombogenic core and the blood are kept apart by the presence of a stable fibrous cap and an intact layer of vascular endothelial cells. The central core contains foam cells, which are macrophages full of LDL, cholesterol, and necrotic cell debris.

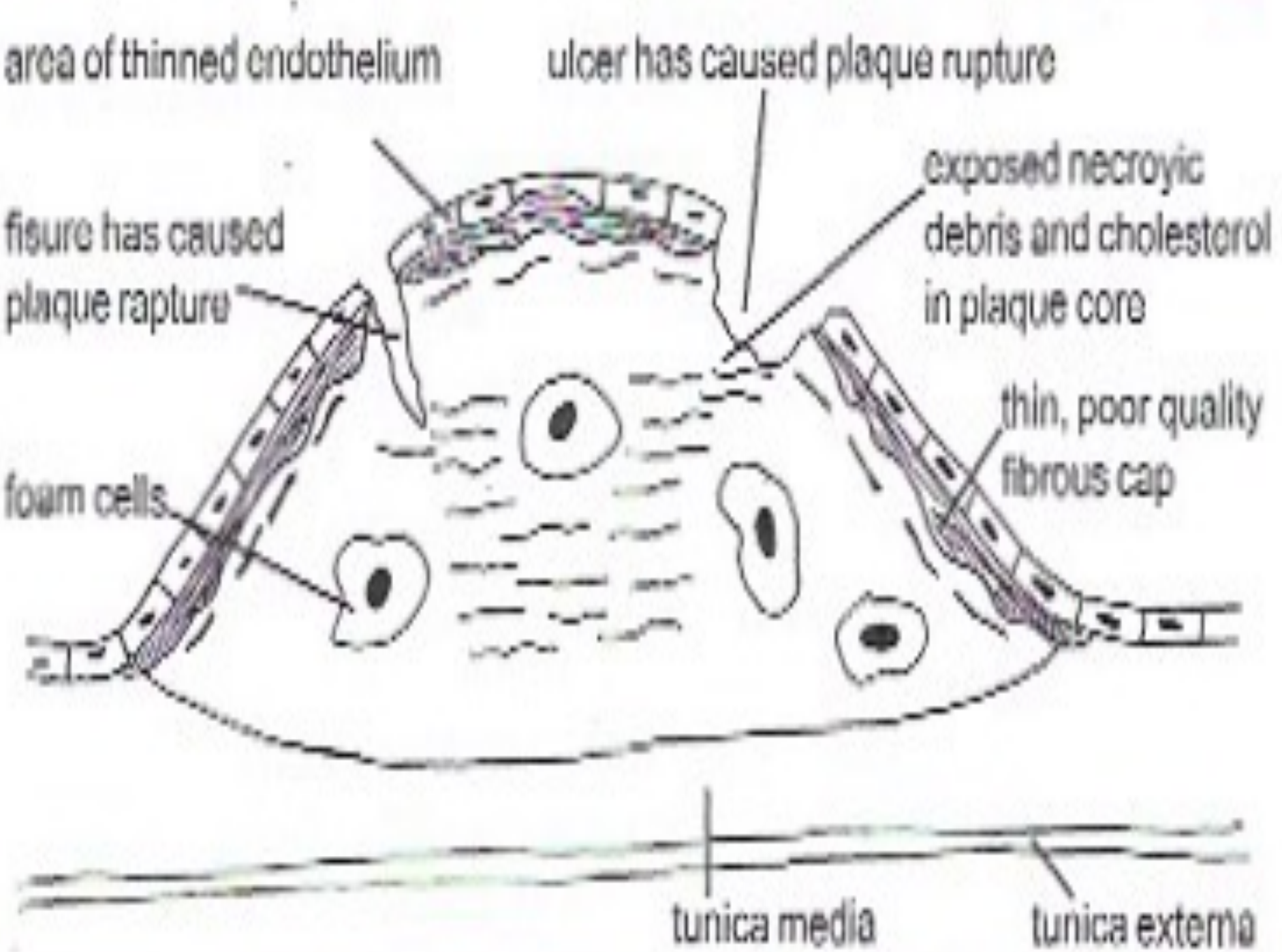

strands of collagen
stable fibrous cap
necrotic debris
and cholesterol
in plaque core
foam cells
endothelium
tunica media
tunica externa

Basic structure of a plaque

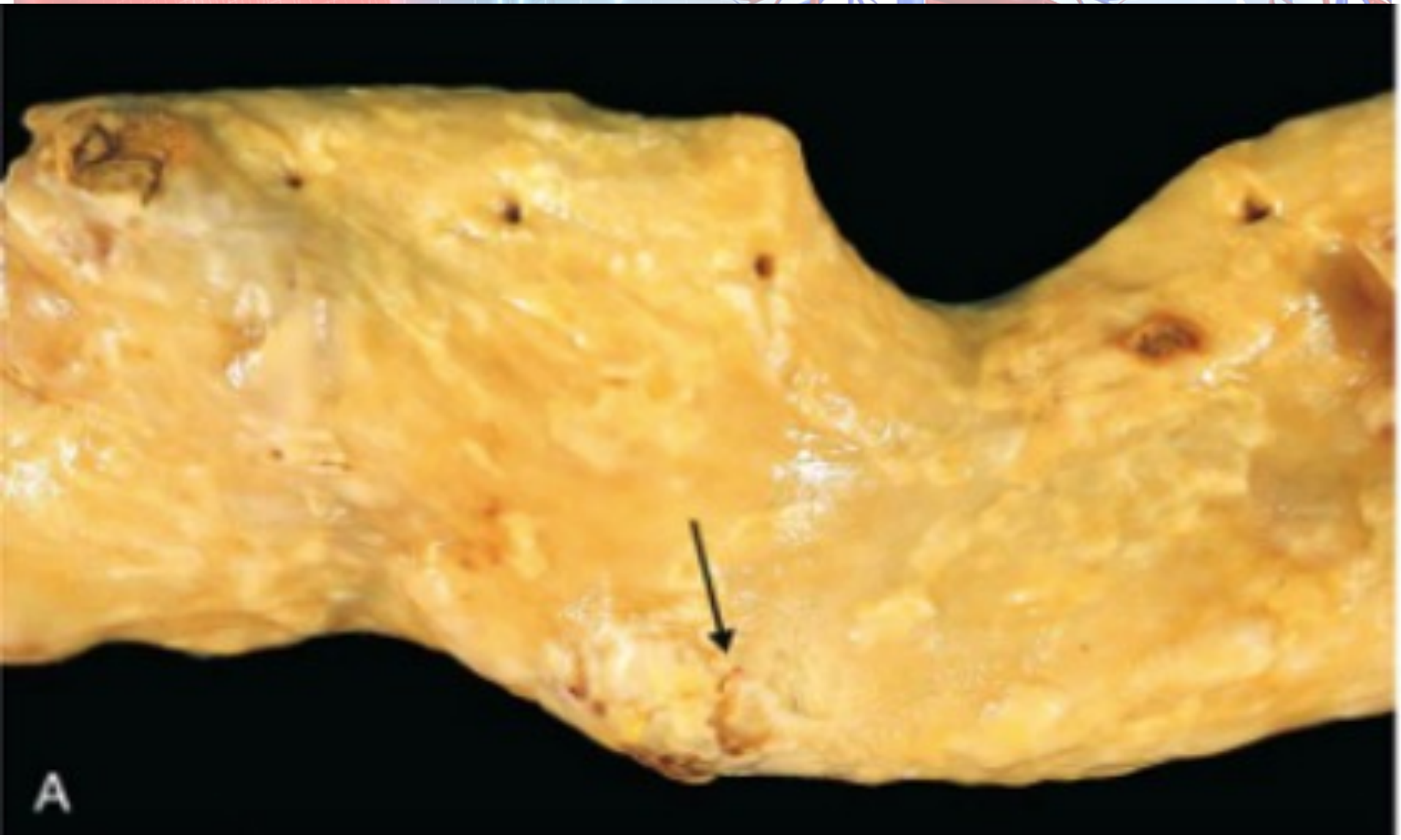


Unstable Thrombus Plaque

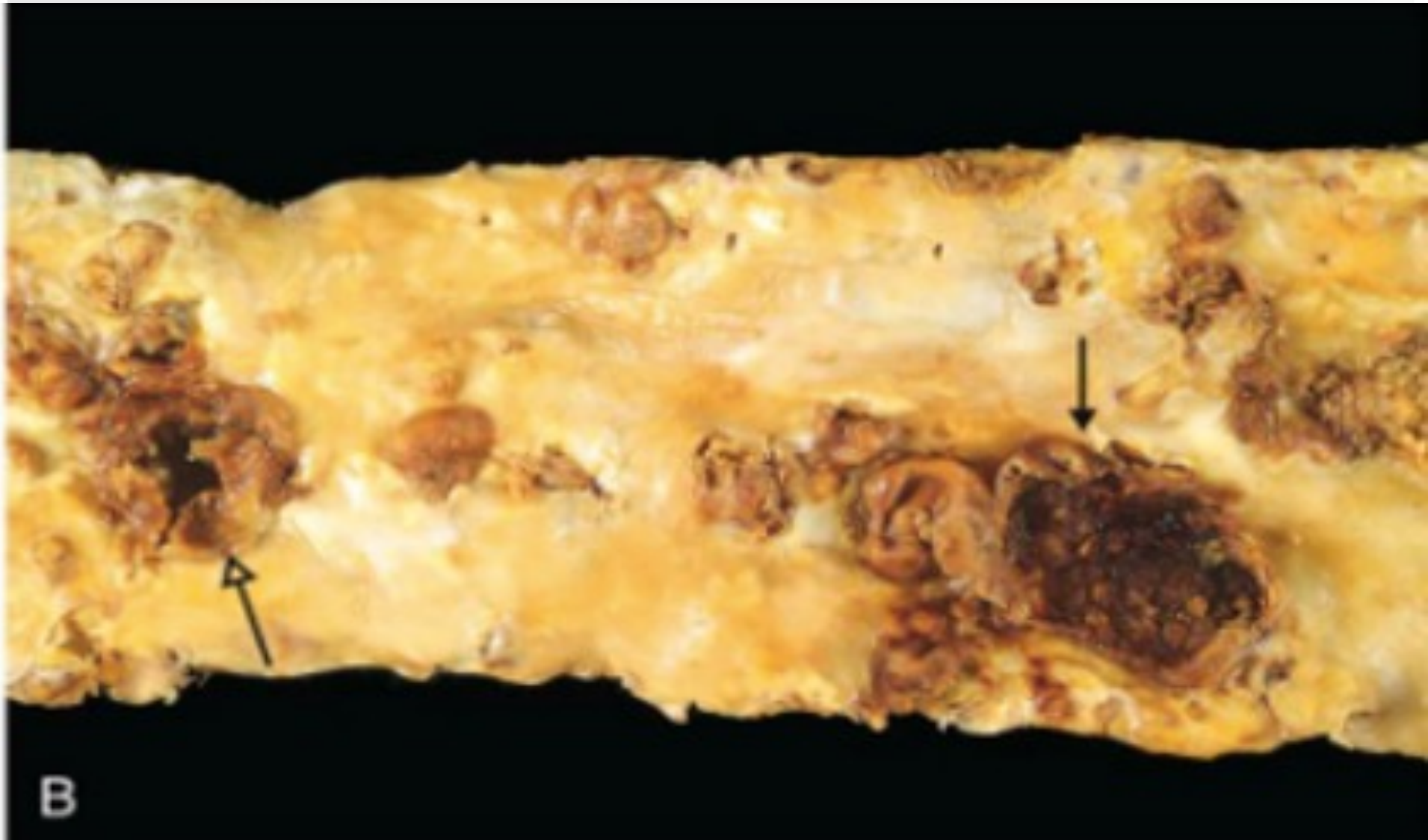
- In the unstable (complicated) thrombus plaque, inflammatory chemicals have thinned the fibrous cap and caused it to become unstable. This can result in the plaque rupturing. As a result, the blood comes into contact with thrombogenic collagen and a clotting factor produced from the foam cells.
- Statin drugs have anti-inflammatory properties as well as lowering LDL cholesterol. As a result, statins prevent anti-inflammatory processes thereby preventing inflammatory thinning of the fibrous cap.


area of thinned endothelium
ulcer has caused plaque rupture
exposed necroyic debris and cholesterol in plaque core
fisure has caused plaque rapture
thin, poor quality fibrous cap
foam cells
tunica media
tunica externa

Atherosclerotic Aorta with fibrous plaques

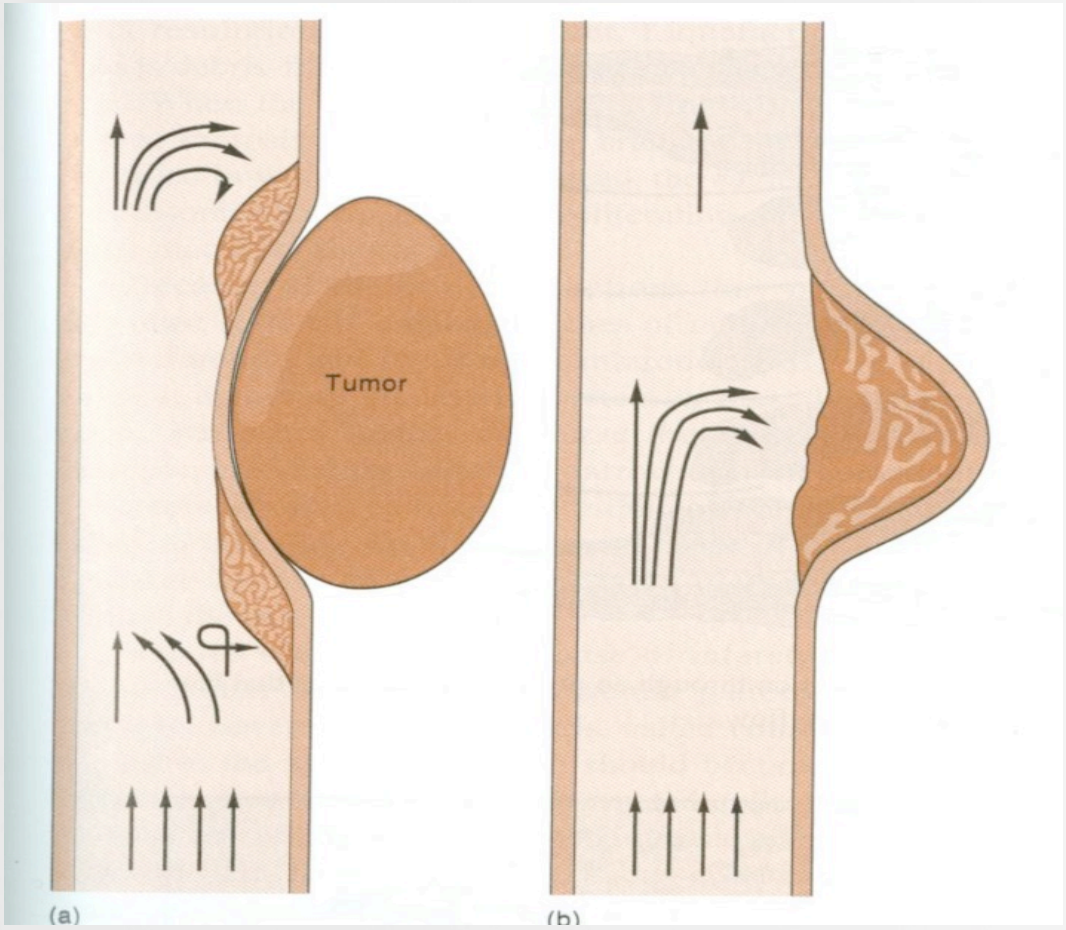


Atherosclerotic Aorta with complications of ulcerated plaque on left and thrombus on right



Vessel Distortions

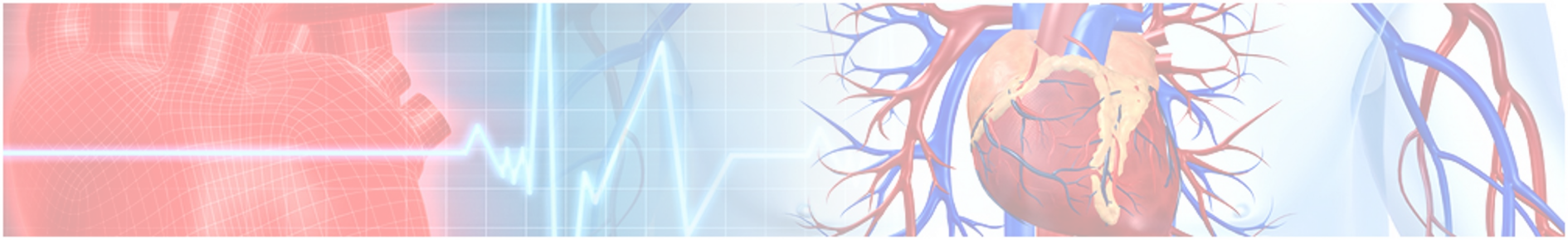
Pressure on vessel wall by a tumor



Thrombosis Sequela

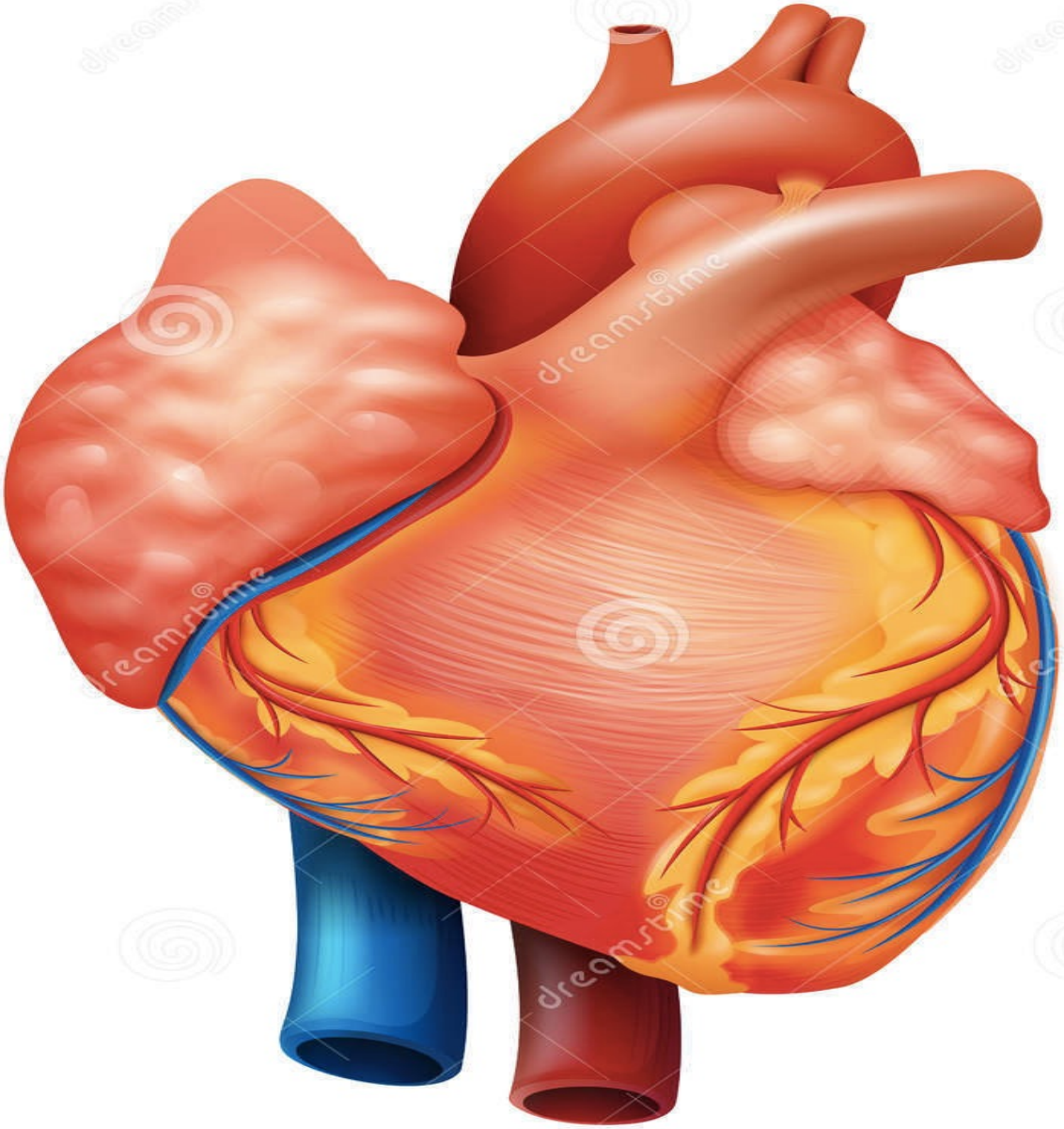
- **Resolution**
  - Thrombus doesn't continue to develop (stable)
- **Organization**
  - *Phagocytes* digest thrombus 2-3 days after it forms
  - *Platelets* and *fibrin* are replaced by fibrous connective tissue
  - Endothelium forms over the tissue
  - Recanalization - Small channels form in a thrombus to allow blood flow, reducing ischemia

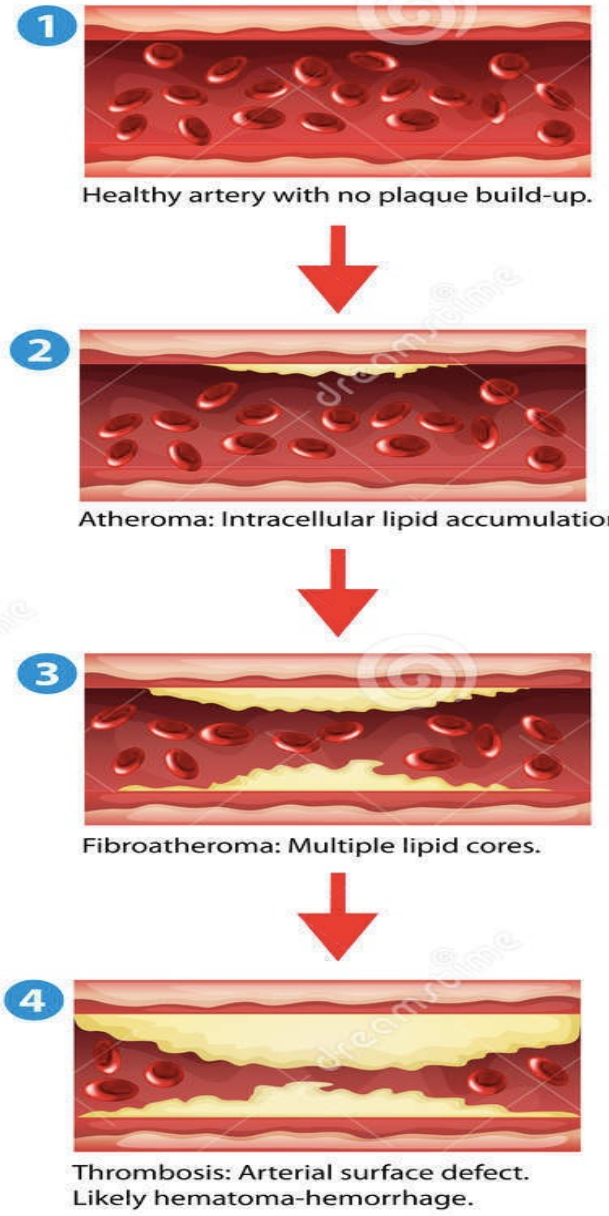
- **Recanalization of a thrombus.**
  - Newly formed thrombus partially restores flow.
- **Propagation**
  - Thrombus extends along the vessel and may obstruct other vessels
- **Infarction**
  - *Infarct* - A region of necrosis near a thrombus caused by ischemia
  - Necrotic tissue broken down and replaced by scar tissue
  - More serious in arteries because veins are branched



- **Embolism**
  - Cause: Blood flow is suddenly stopped by an embolus, an abnormal mass in the bloodstream
  - Embolism is a common cause of infarction

Process of Arteriosclerosis



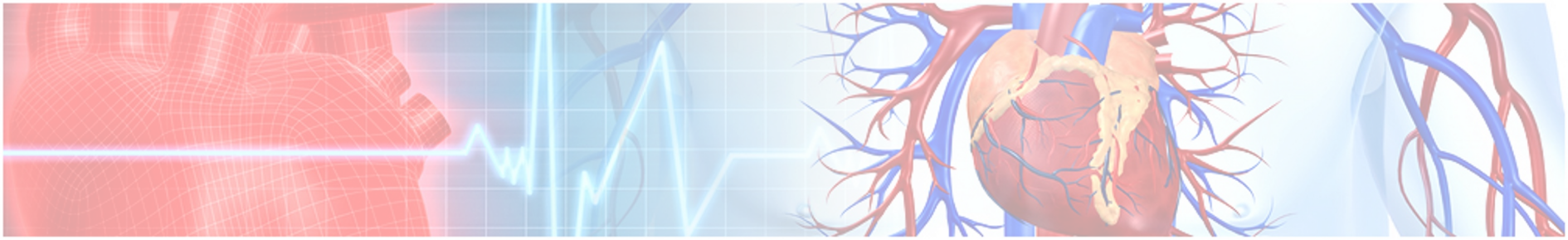


Types of Embolism

- Thromboembolism
- Fat Embolism
- Air Embolism
- Foreign Body Embolism
- Amniotic Fluid Embolism (rare)

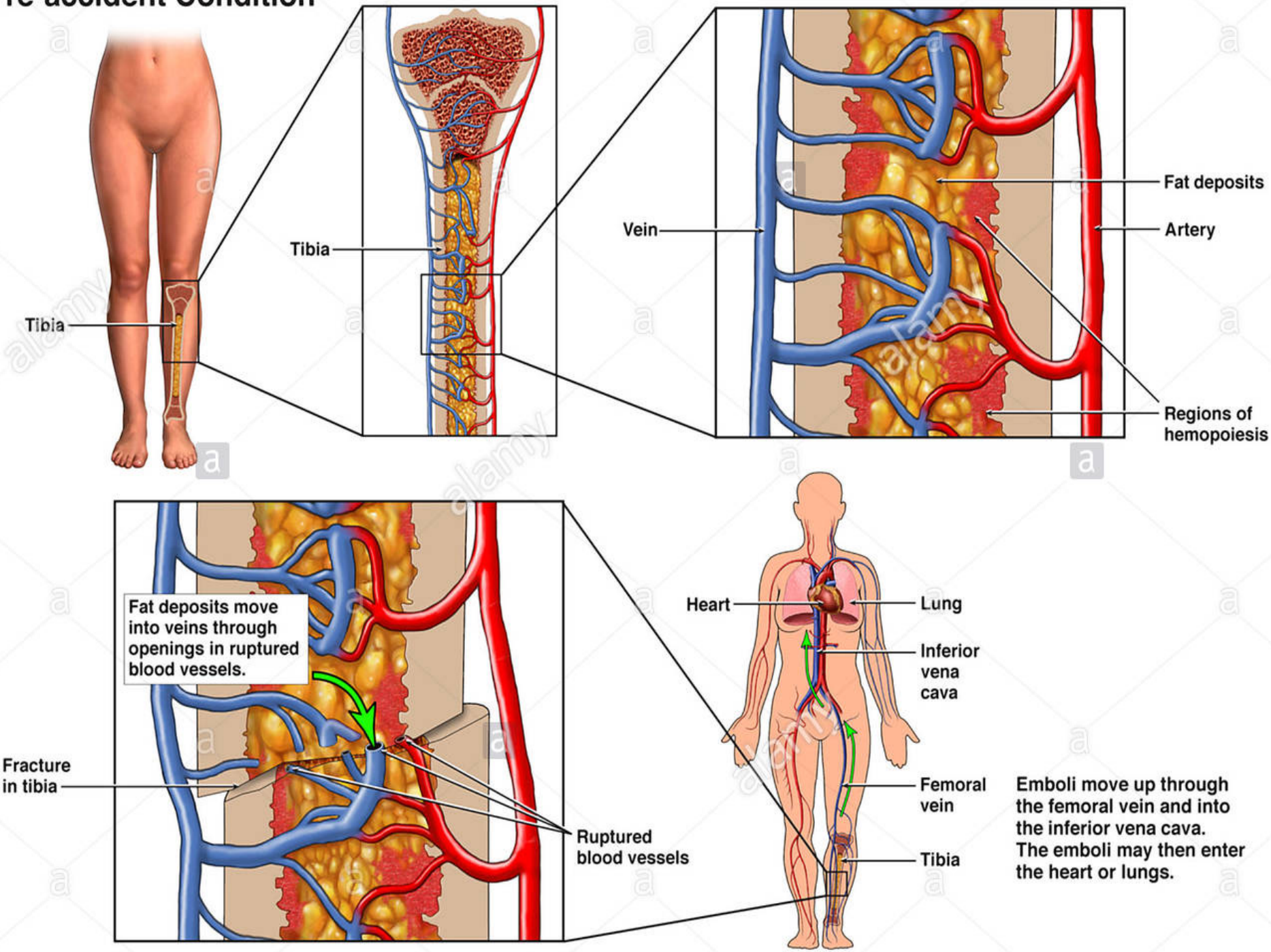
Thromboembolism

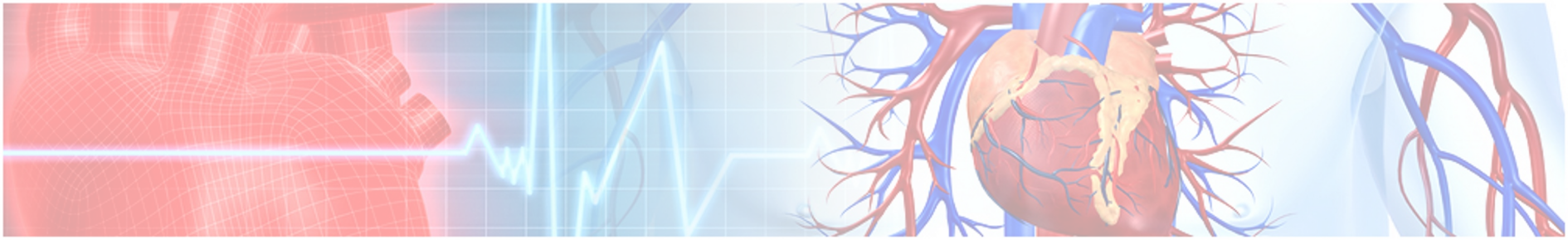
- Embolus is a *thrombus* or *fragmented thrombus* that breaks away from its source
- Cause of fragmentation: Temporary pressure change from resuming activity after a period of inactivity
- Two types of thromboemboli:
  - Arterial Thromboemboli - Thromboembolus in arteries is carried to smaller vessels until it becomes lodged
  - Venous Thromboemboli - Thromboembolus in veins is carried to larger vessels until it becomes lodged in the heart or lungs



- **Fat Embolism**
  - Forms when fat-rich marrow gains access to blood
  - Causes: Long bone fractures and liver damage

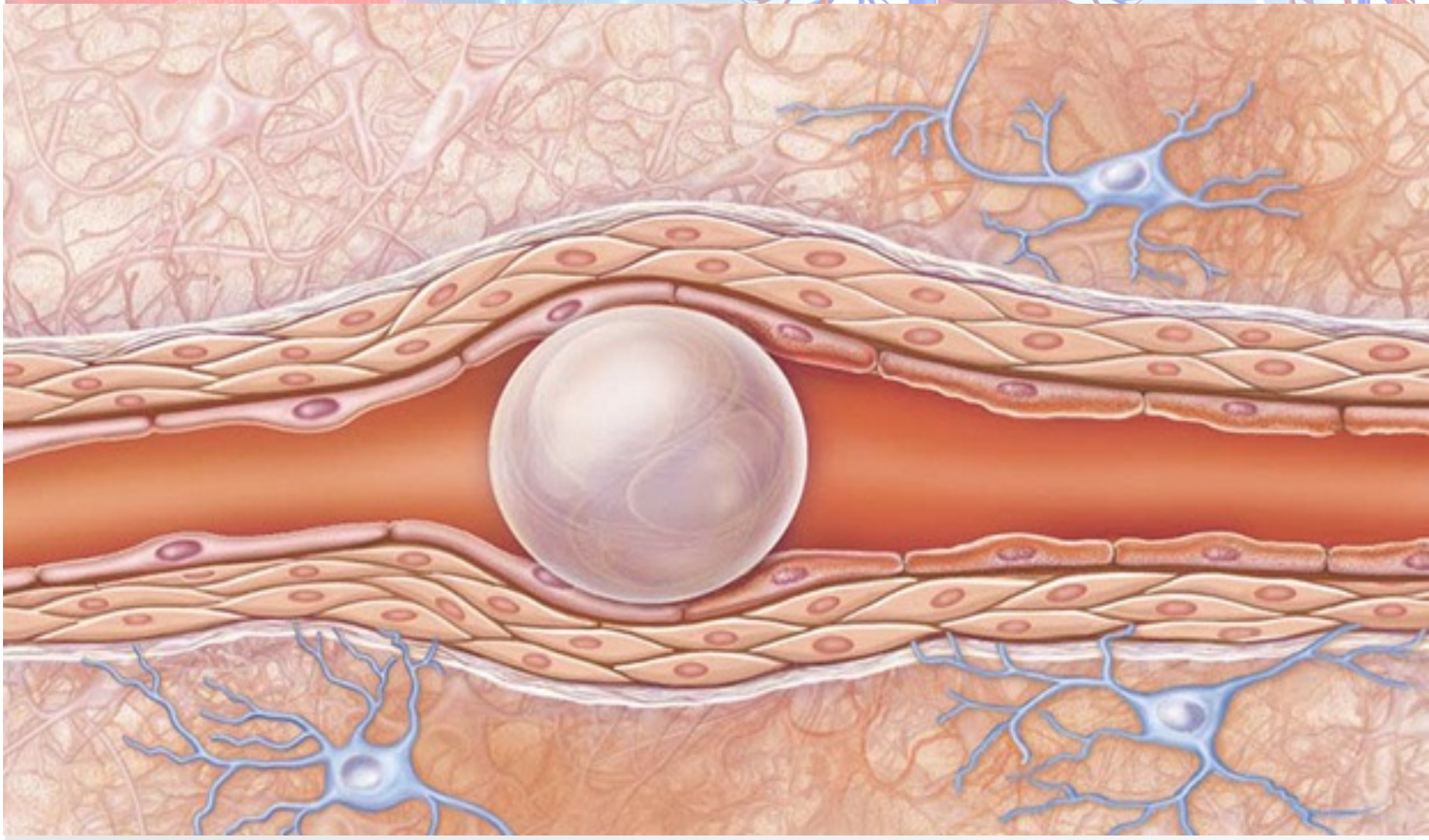
Pre-accident Condition

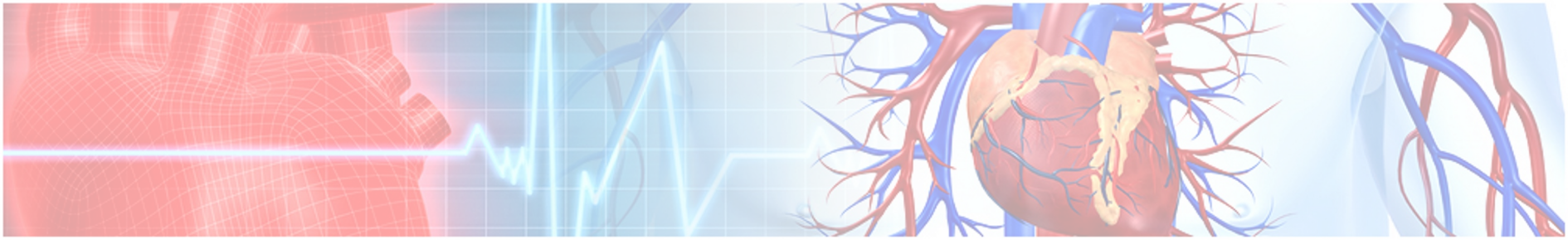




- **Air Embolism**
  - *Air* or *gases* form bubbles in the blood
    - Volume over 100 mL is dangerous
    - Heart is damaged by volume of 300 mL
    - Type of air embolism: The Bends
    - *Nitrogen gas* leaves blood and forms emboli, resulting in quick reduction in pressure

Air Embolism



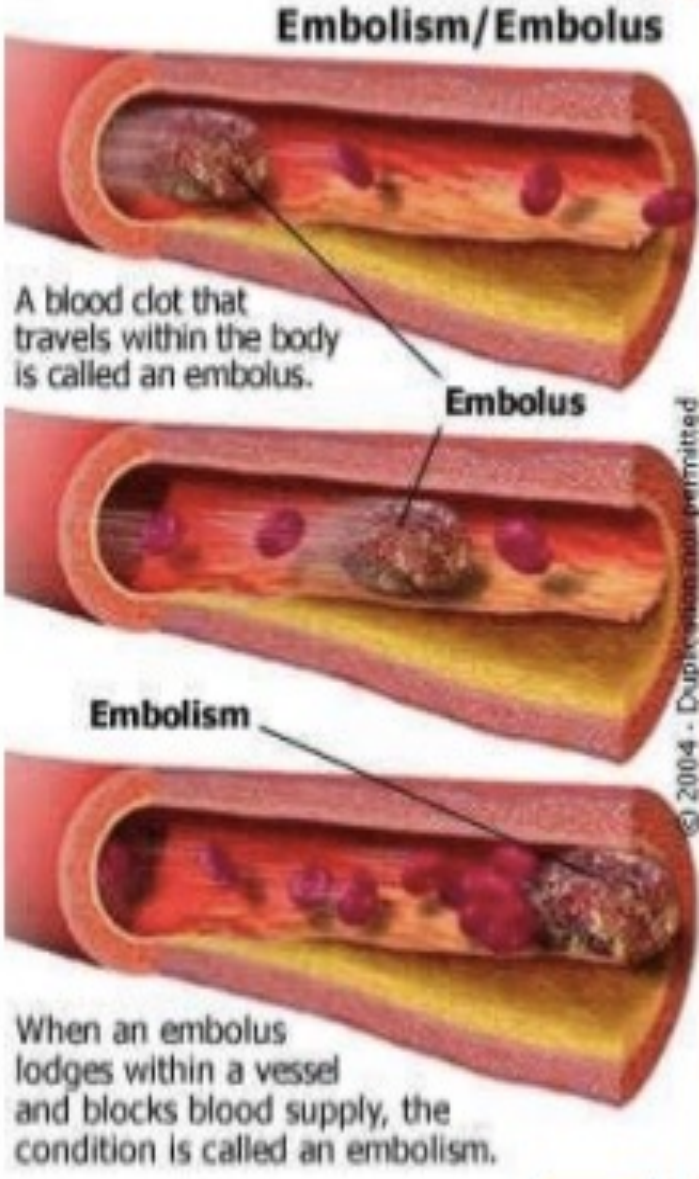


- **Foreign Body Embolism**
  - Foreign material impedes blood flow
- **Amniotic Fluid Embolism (rare)**
  - Infant's cells are shed into amniotic fluid and enter circulation via cuts in the placental membrane

Fat Emboli: Fat particles or droplets that travel through the circulation

Fat Embolism: A process by which fat emboli passes into the bloodstream and lodges within a blood vessel.

Fat Embolism Syndrome (FES): serious manifestation of fat embolism occasionally causes multi system dysfunction, the lungs are always involved and next is brain

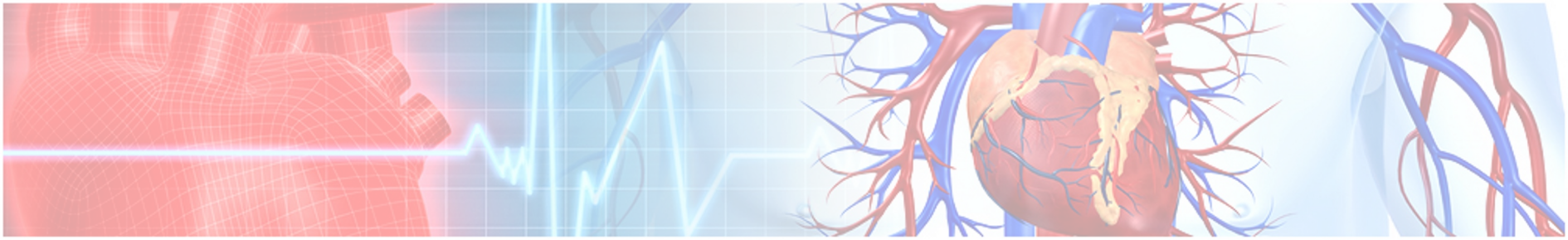


Thromboembolism Therapy

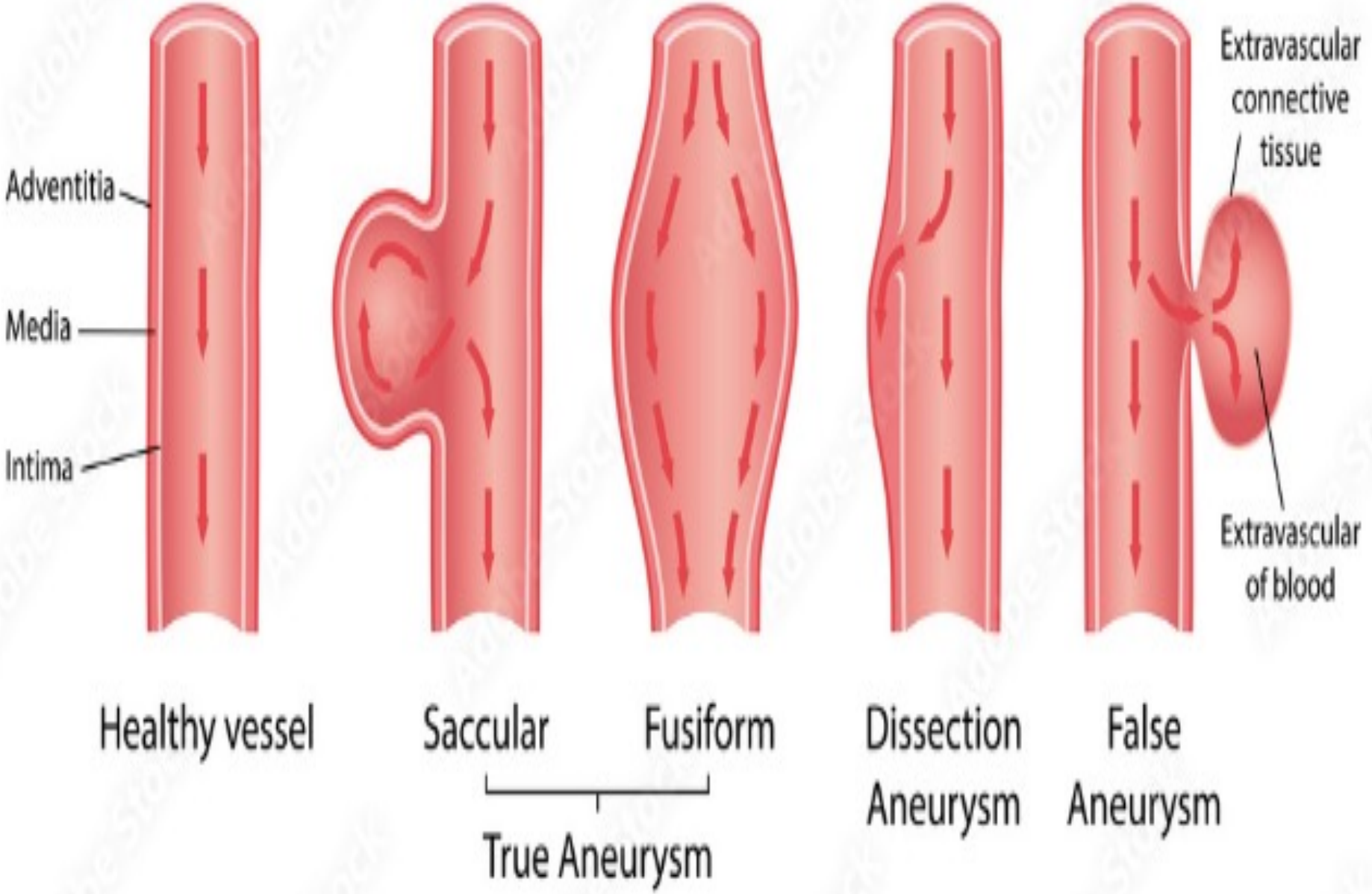
- **Anticoagulant Therapy**
  - Interferes with blood coagulation
  - Example: *Heparin* blocks thrombin and fibrin formation
- **Activation of the Fibrinolytic System**
  - Promotes breakdown of clotting factors
  - Example: *t-PA (tissue plasminogen activator)*
- **Antiplatelet Therapy**
  - Decreases platelet aggregation and activation
  - Example: *Aspirin*

Aneurysm

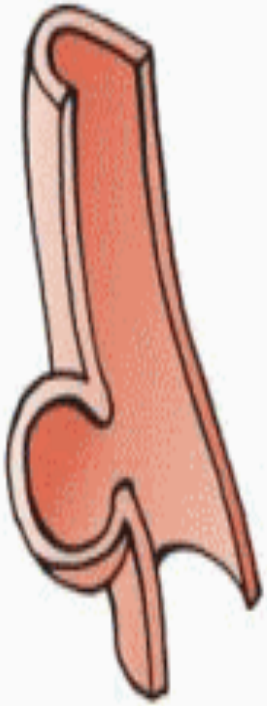
- Enlargement of the artery or ballooning of the wall
- The growing atheroma and thinning of the wall causes the arterial pressure to increase, which only further weakens the wall
- Two causes of aneurysm are increased blood pressure and weakened arterial walls



- True” aneurysms involve all three layers of the artery (intima, media, and adventitia) or the attenuated wall of the heart; these include atherosclerotic and congenital vascular aneurysms, as well as ventricular aneurysms resulting from transmural myocardial infarctions.
- False aneurysm results when a wall defect leads to the formation of an extravascular hematoma that communicates with the intravascular space (“pulsating hematoma”).

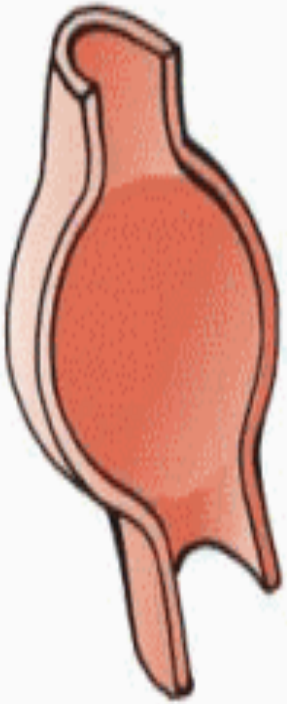

Adventitia
Media
Intima
Extravascular connective tissue
Extravascular of blood
Healthy vessel
Saccular
Fusiform
True Aneurysm
Dissection Aneurysm
False Aneurysm

Saccular aneurysm



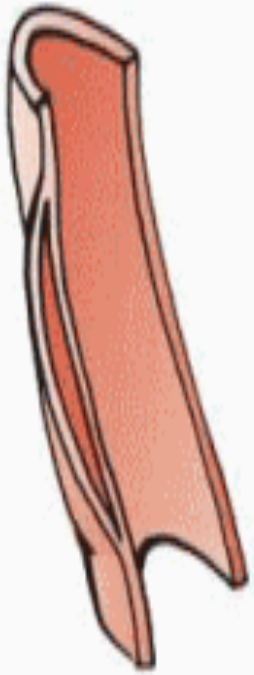
Unilateral pouchlike bulge with a narrow neck

Fusiform aneurysm



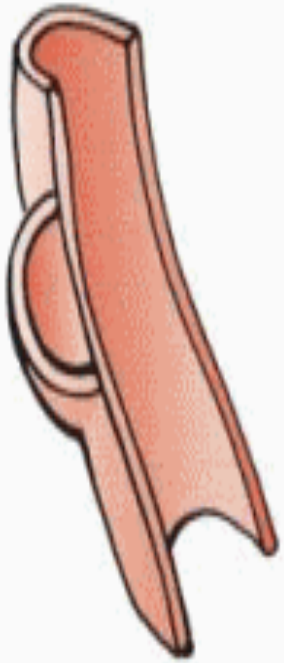
Spindle-shaped bulge encompassing the entire diameter of the vessel

Dissecting aneurysm



Hemorrhagic separation of the medial layers of the vessel wall creating a false lumen

False aneurysm



Pulsating hematoma resulting from trauma and commonly mistaken for an abdominal aneurysm

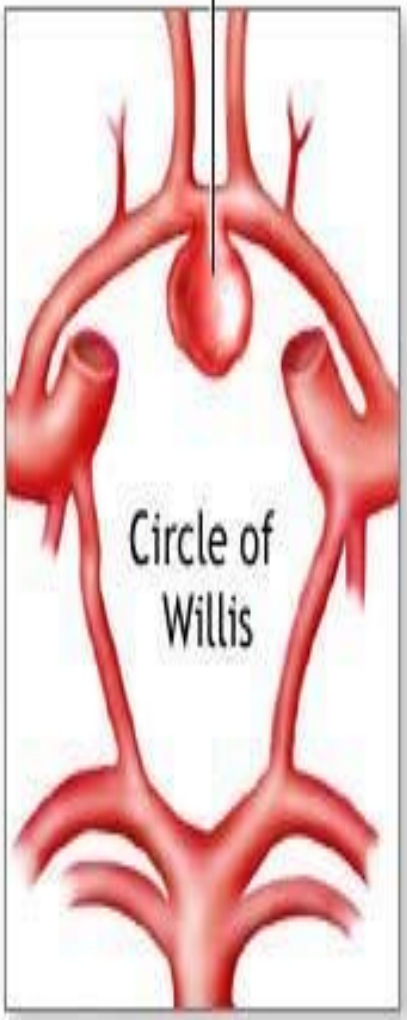
Saccular Aneurysm

Fusiform Aneurysm

Ruptured Aneursym

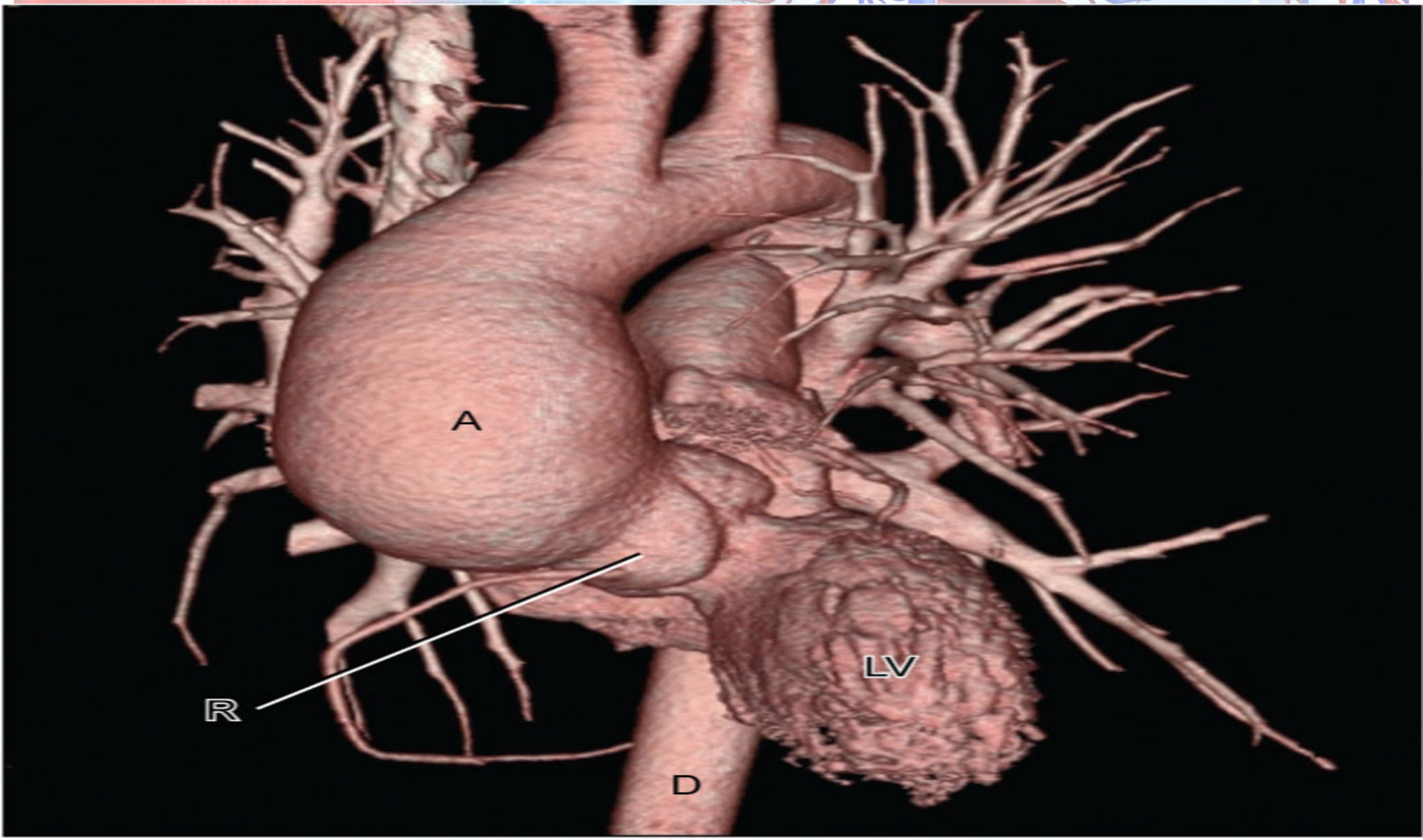
Bottom view of brain and major arteries of the brain

Berry aneurysm on the anterior communicating artery of the brain

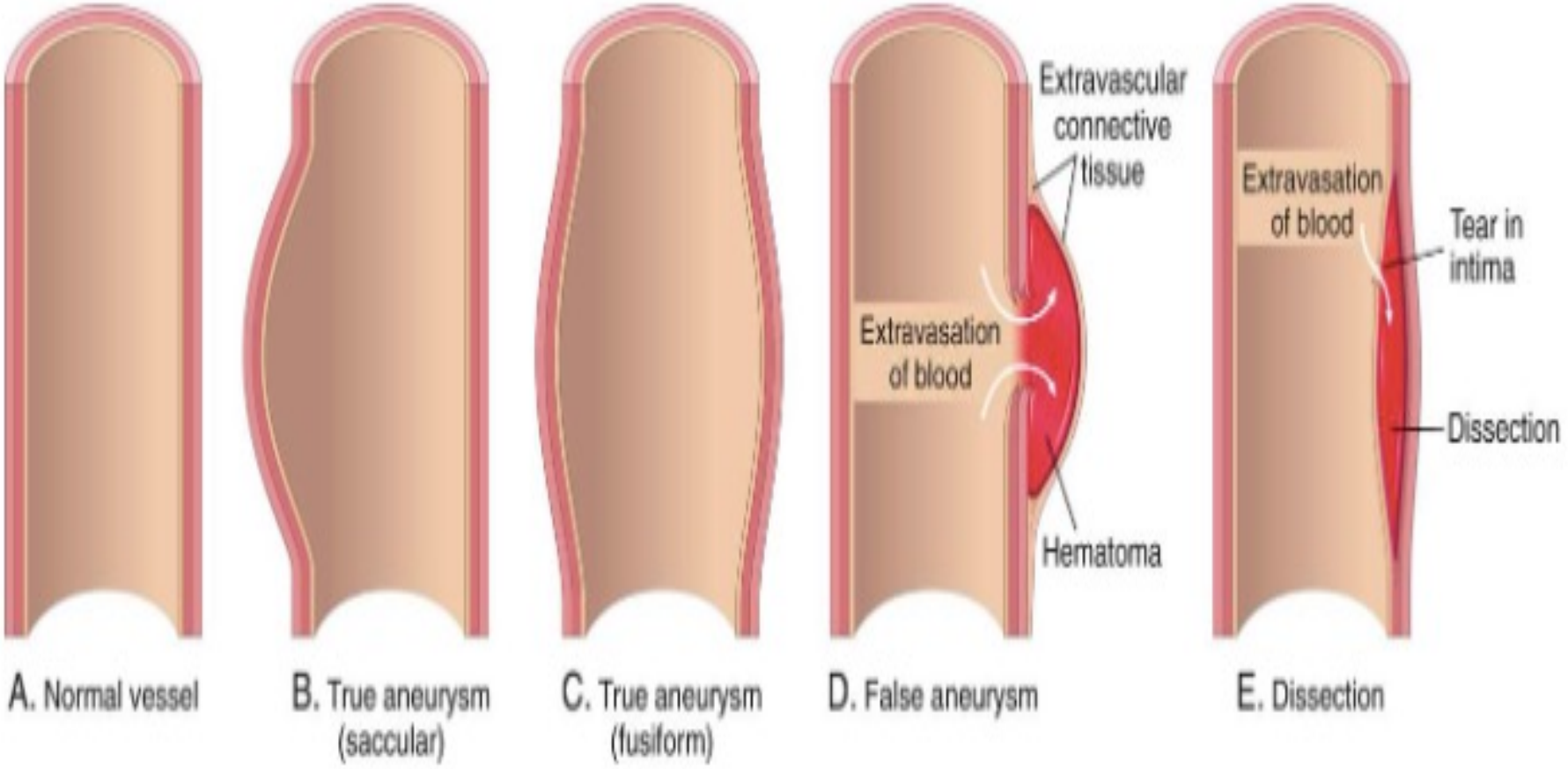


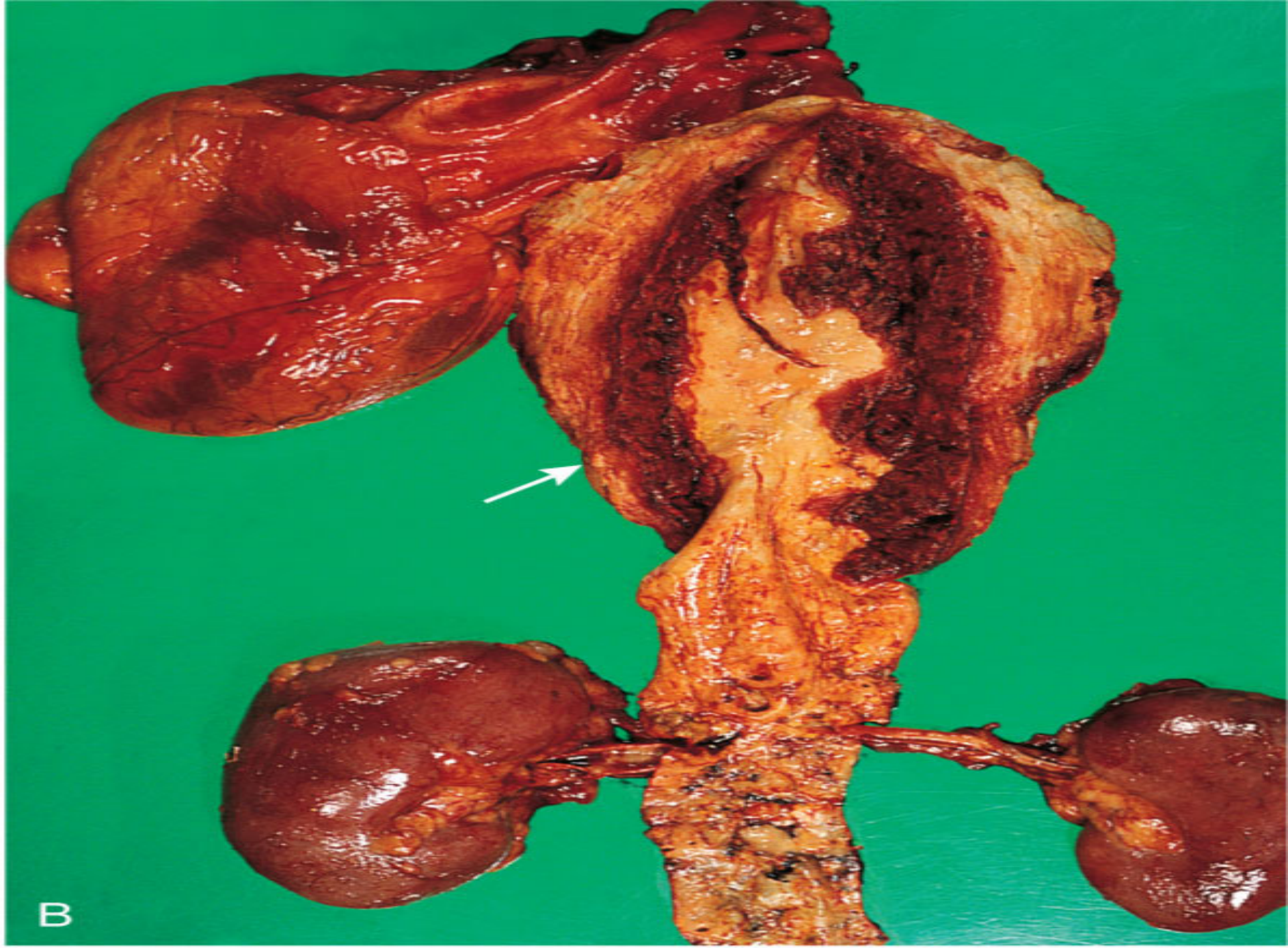
An aneurysm can occur in any blood vessel which supplies the brain

Aneurysm



Aneurysm


Extravascular connective tissue
Extravasation of blood
Hematoma
Extravasation of blood
Tear in intima
Dissection
A. Normal vessel
B. True aneurysm (saccular)
C. True aneurysm (fusiform)
D. False aneurysm
E. Dissection



(**B** from Damjanov I, Linder J, editors: *Anderson's pathology*, ed 10, St Louis, 1996, Mosby.)

Abdominal Aortic Aneurysm

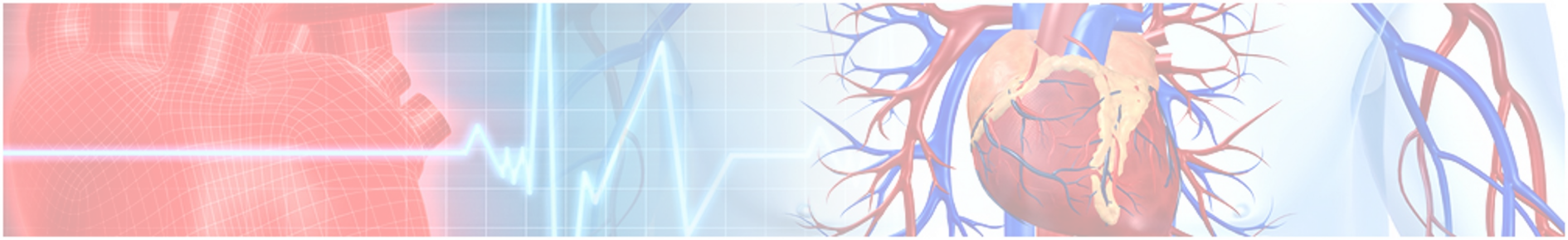
- Atherosclerotic aneurysms occur most frequently in the abdominal aorta, but the common iliac arteries, aortic arch, and descending thoracic aorta can also be involved.
- Abdominal aortic aneurysm (AAA) occurs more frequently in men and in smokers and rarely develops before the age of 50 years.
- Atherosclerosis is a major cause of AAA.

Clinical Consequences of AAA

- Obstruction of a vessel branching off the aorta (e.g., the renal, iliac, vertebral, or mesenteric arteries), resulting in distal ischemia of the kidneys, legs, spinal cord, or gastrointestinal tract.
- Impingement on adjacent structures, e.g., compression of a ureter or erosion of vertebrae by the expanding aneurysm
- An abdominal mass (often palpably pulsating) that simulates a tumor

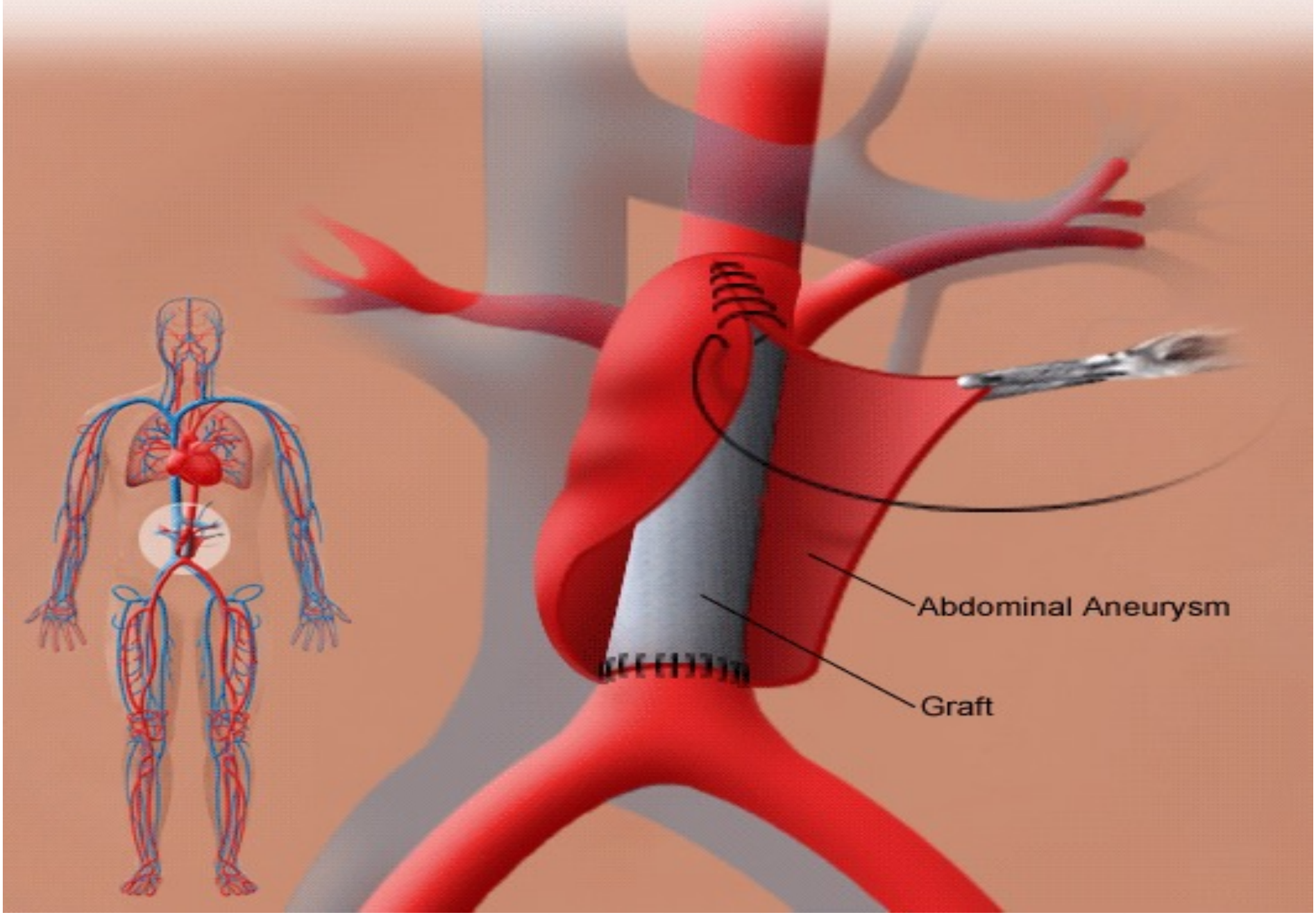
Clinical Consequences of AAA

- Rupture into the peritoneal cavity or retroperitoneal tissues, leading to massive, often fatal hemorrhage
- The risk of rupture is determined by size.
  - AAAs 4 cm or less in diameter almost never burst, while those between 4 and 5 cm do so at a rate of 1% per year.
  - The risk rises to 11% per year for AAAs 5 to 6 cm in diameter, and to 25% per year for aneurysms greater than 6 cm in diameter.

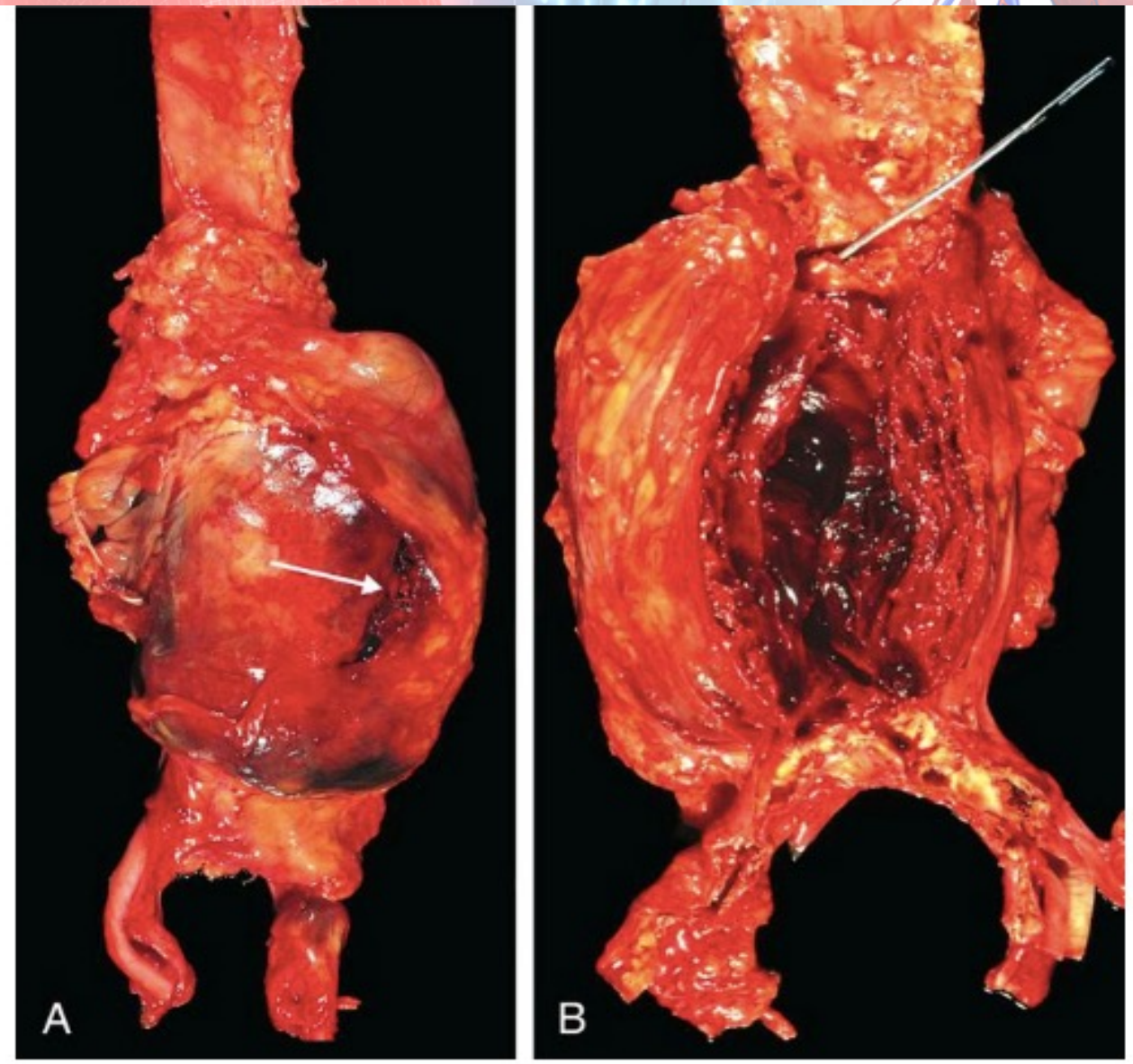


- Thus, aneurysms 5 cm in diameter or larger are managed surgically, either by open placement of tubular prosthetic grafts or with endoluminal insertion of stented grafts (expandable wire frames covered by a cloth sleeve).
- Timely intervention is critical, because the mortality rate for elective procedures is approximately 5%, whereas the rate for emergency surgery after rupture is roughly 50%.

Abdominal Aortic Aneurysm (AAA) Open Surgical Repair



Abdominal Aortic Aneurysm



- A rupture is seen on the left
- A large thrombus is seen on the right

Aortic Dissection

- Aortic dissection occurs when blood splays apart the laminar planes of the media to form a blood-filled channel within the aortic wall; this development can be catastrophic if the dissecting blood ruptures through the adventitia and escapes into adjacent spaces.
- Aortic dissection occurs mainly in two age groups:
  - (1) men aged 40 to 60 hypertension (more than 90% of cases)
  - (2) younger patients with connective tissue abnormalities that affect the aorta (e.g., Marfan syndrome).

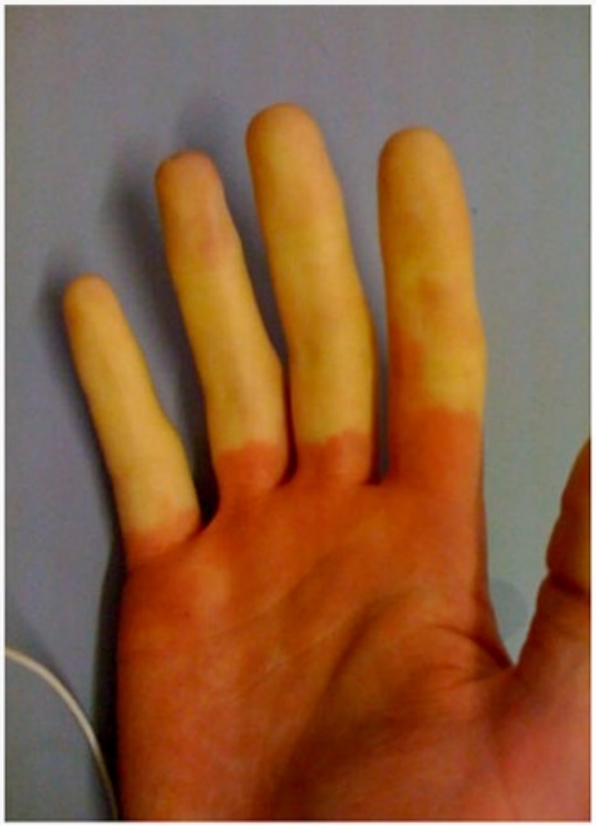
- The classic clinical symptom of aortic dissection is the sudden onset of excruciating tearing or stabbing pain, usually beginning in the anterior chest, radiating to the back between the scapulae, and moving downward as the dissection progresses.
- The most common cause of death is rupture of the dissection into the pericardial, pleural, or peritoneal cavity.
- Common clinical presentations with cardiac involvement include tamponade, aortic insufficiency, and myocardial infarction.

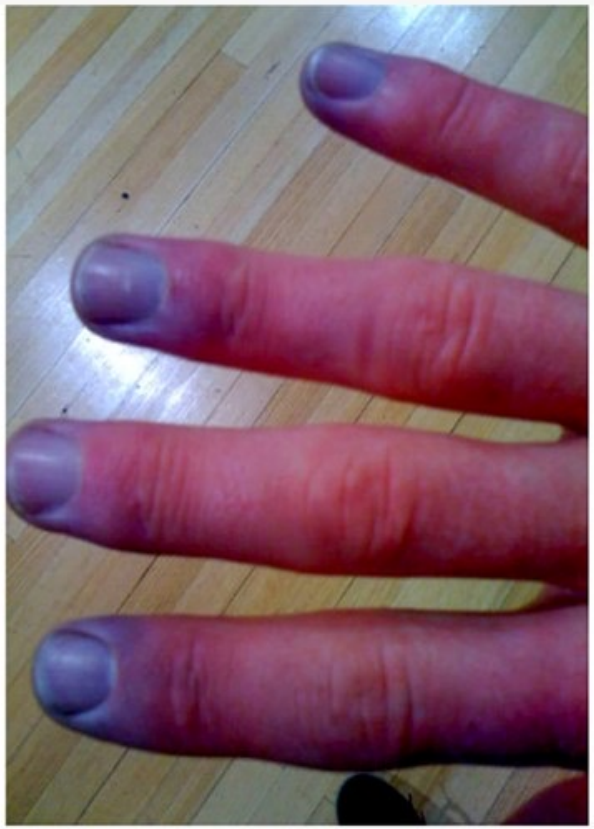
Other vascular disorders caused by disrupted arterial circulation

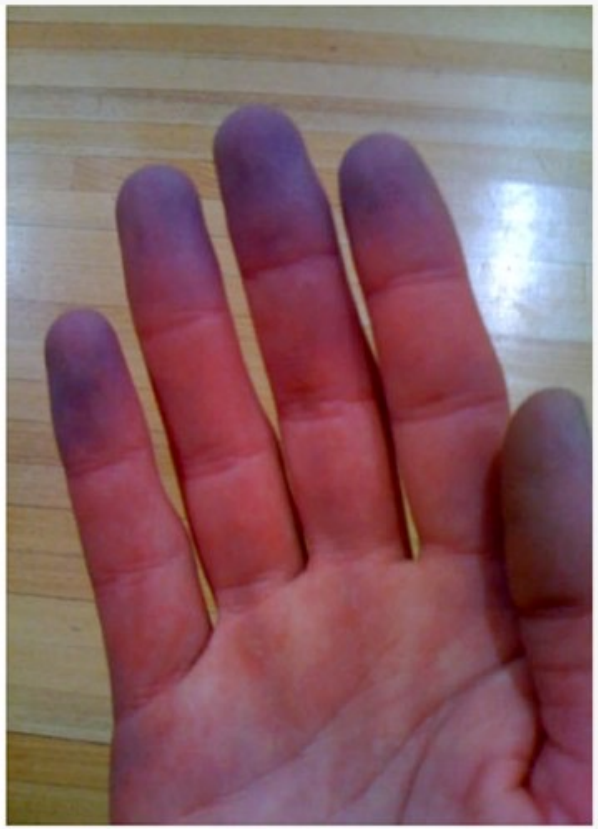
- **Gangrene –** necrotic dead tissue
- **Pressure sores**
- **Coronary arterial disease**
- **Strokes – cerebral vascular accidents**

- **Raynaud's disease**
  - Excessive constriction of small arteries and arterioles
  - Usually seen in fingers and toes
  - Affects about 5% of population
  - The restricted blood flow induces paroxysmal pallor or cyanosis; involved digits characteristically show "red-white-and-blue" color changes from most proximal to most distal, reflecting proximal vasodilation, central vasoconstriction, and more distal cyanosis, respectively.

Raynaud's



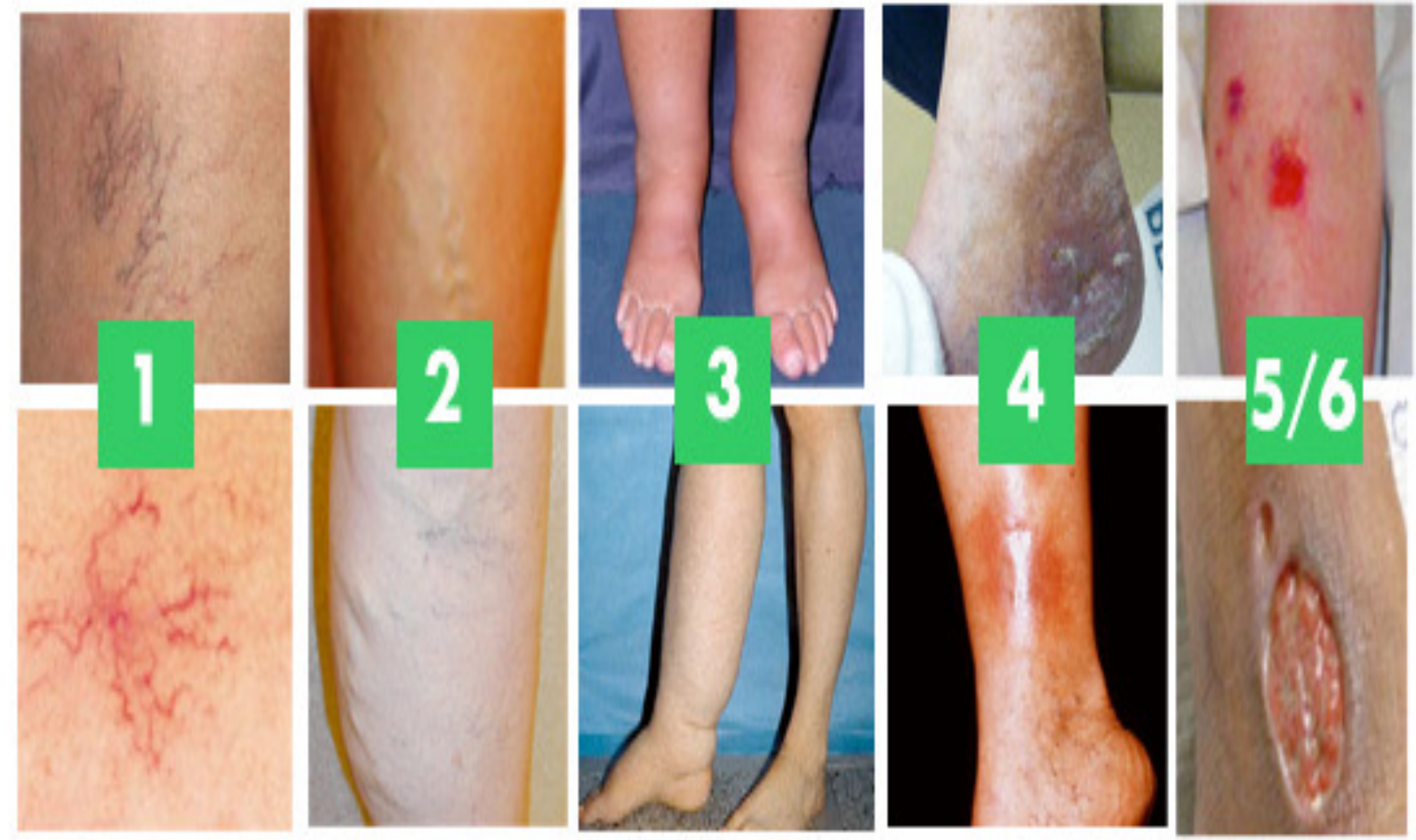


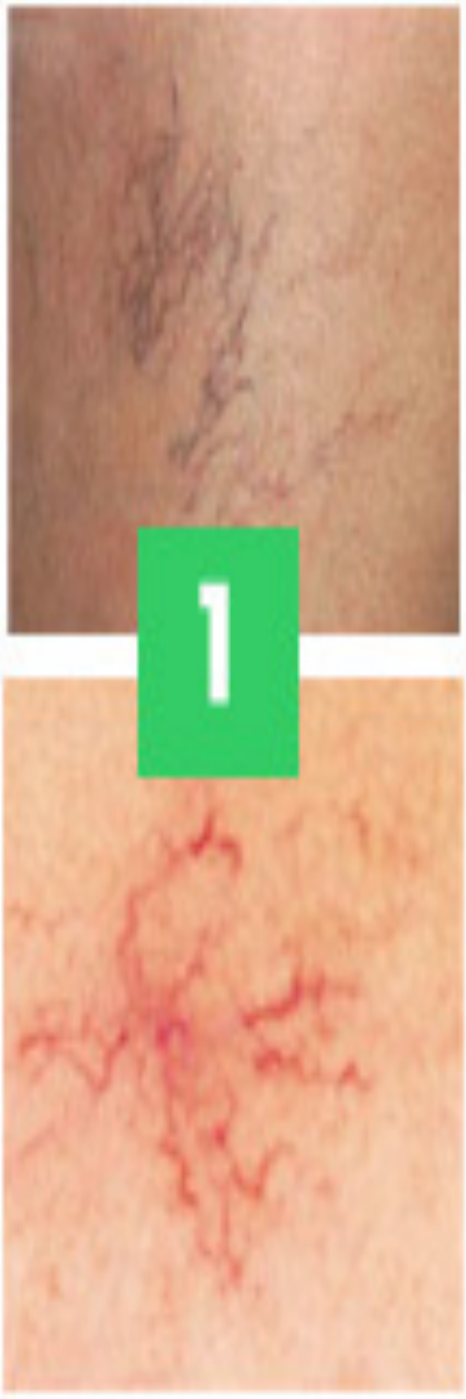


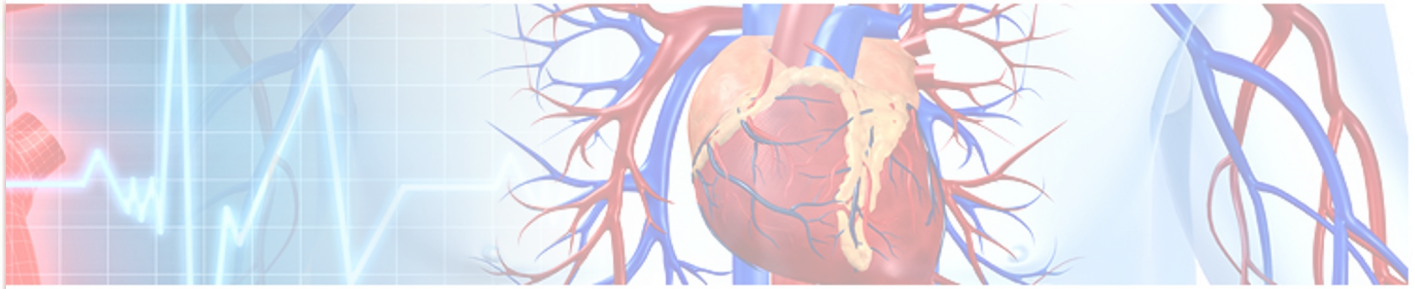
Vein Disorders

- Common, but usually not life-threatening
- **Thrombophlebitis**
  - Inflammation of a vein as a result of thrombosis (usually in deep leg veins)
  - Risk of thromboembolism

Vein Disease Stages from Spider Veins to Ulcers





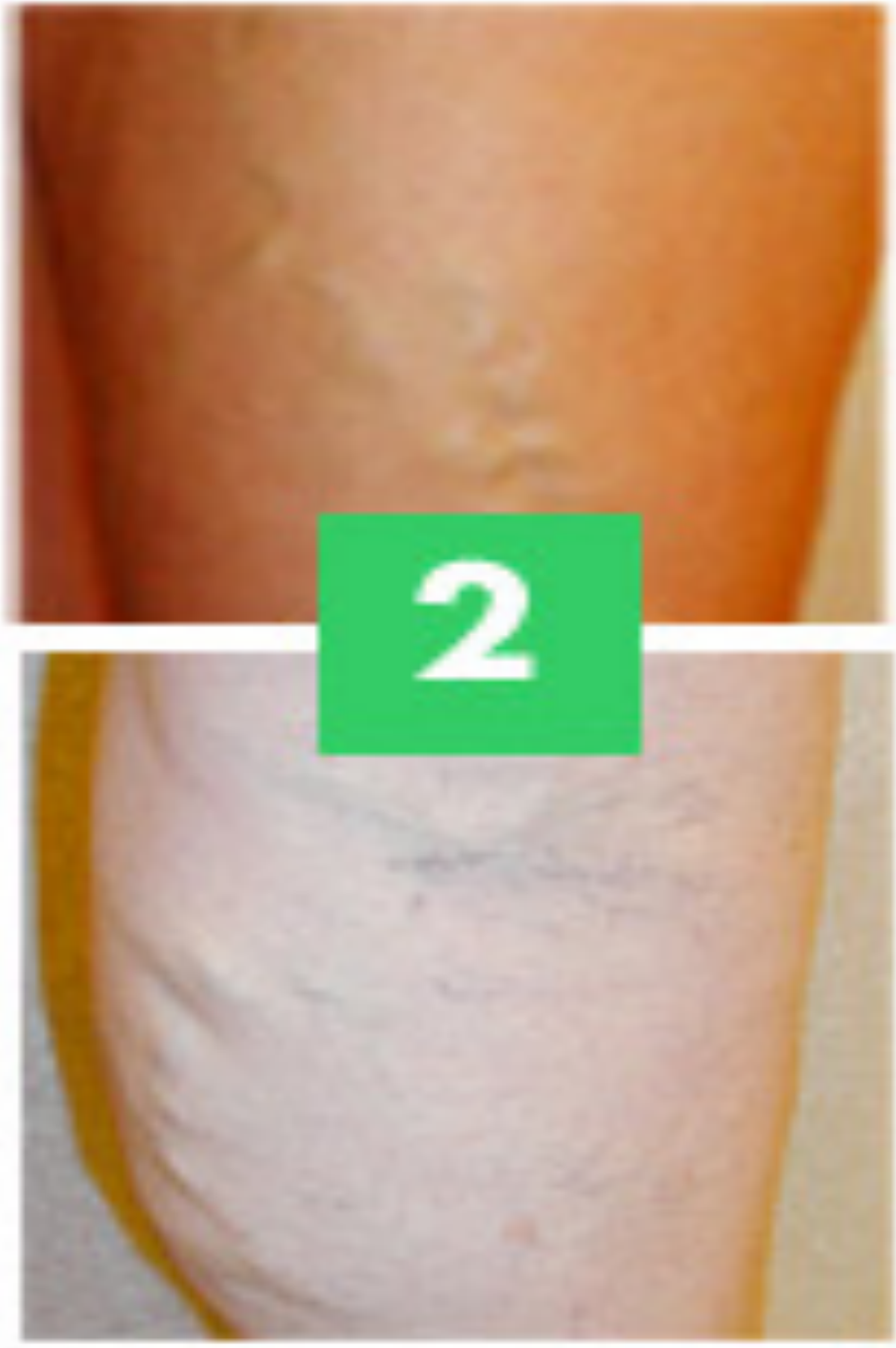


Class 1 - Vein Disease: Spider Veins (Telangectasias)

- This describes diseases that occur in tiny veins in the skin
- The dilated veins and capillary vessels are called telangectasis or spider veins.
- Occurs in 40% of woman and 18% of men. Over half of the population has this form of vein disease

Vein Disease Stages from Spider Veins to Ulcers

- **Class 2 – Vein Disease: Varicose Veins**
  - Often referred to as dilated superficial veins
  - Affects 15% of the population
  - Varicose veins are veins that are tortuous and dilated because of leaky valves
  - This is the stage in which people often become aware of vein disease.
  - 75% of patients at this stage states that it interferes with daily activity.
  - Heredity, prolonged standing, obesity, pregnancy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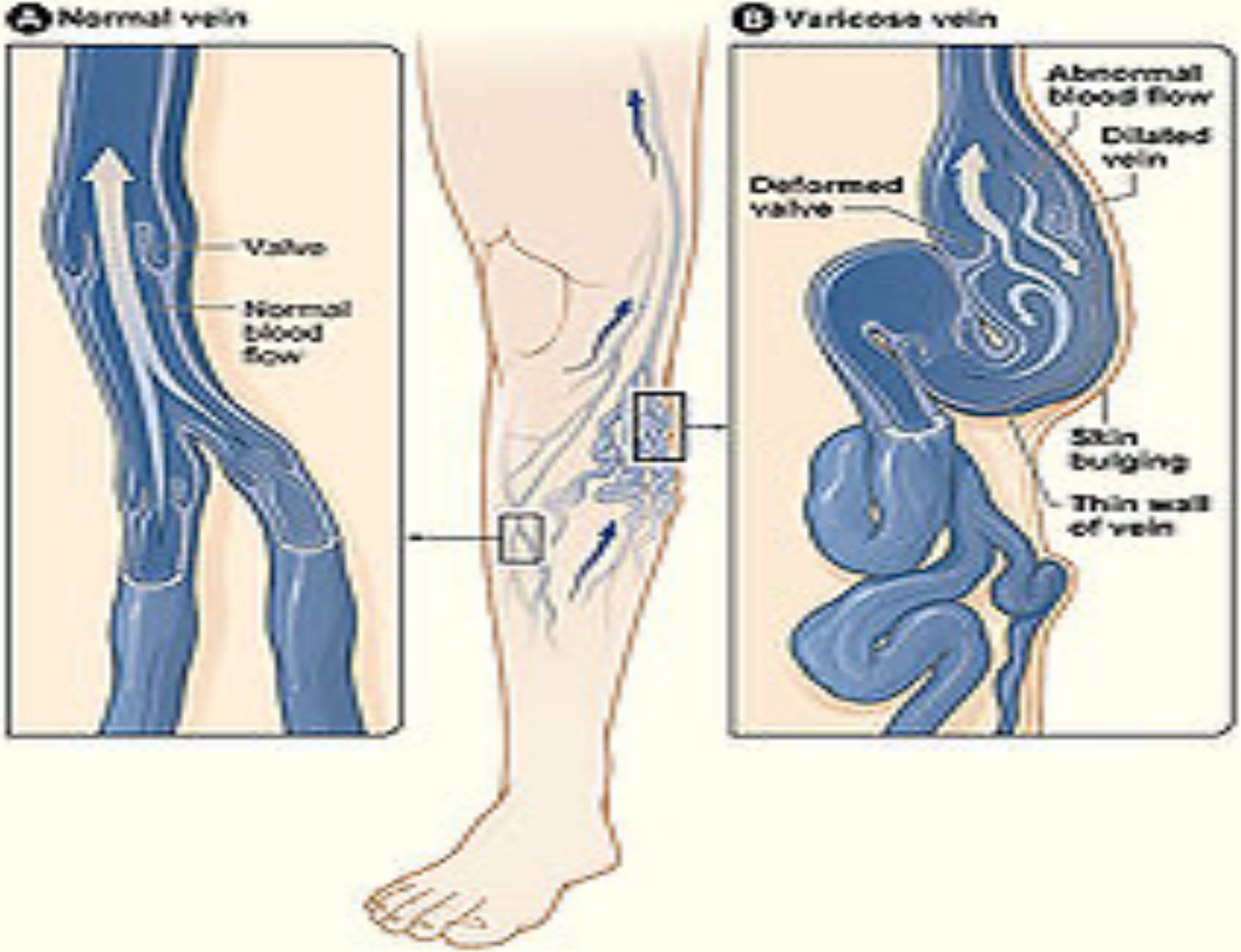
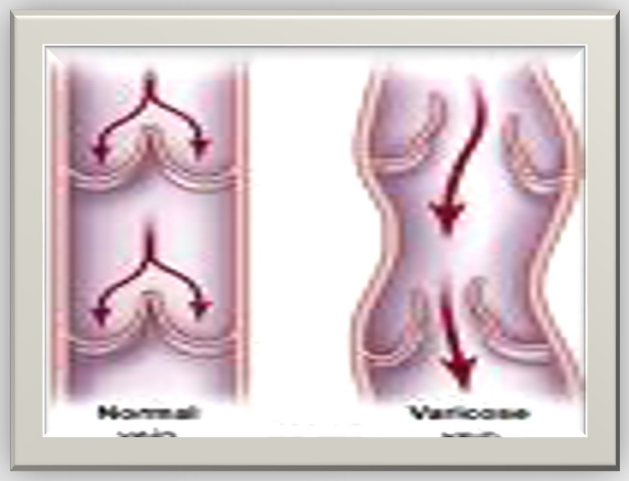
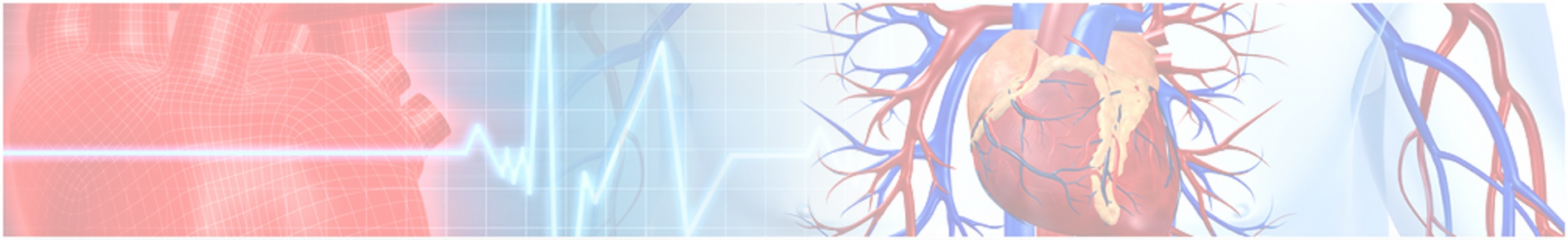
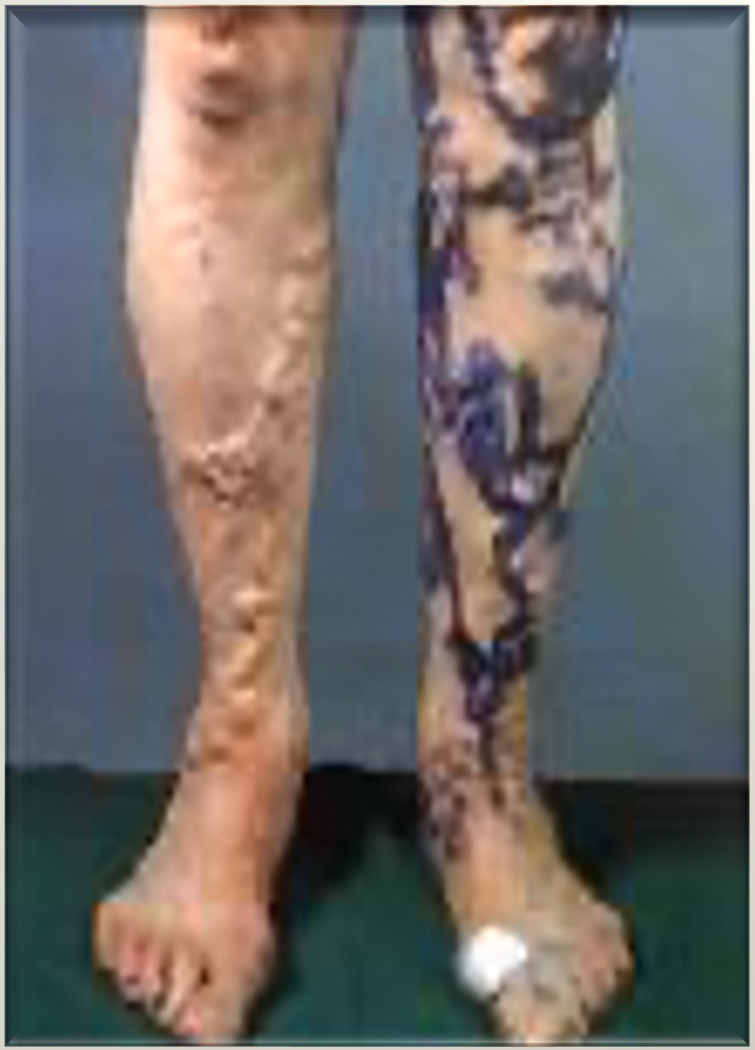

A Normal vein
Valve
Normal blood flow
B Varicose vein
Abnormal blood flow
Dilated vein
Deformed valve
Skin bulging
Thin wall of vein

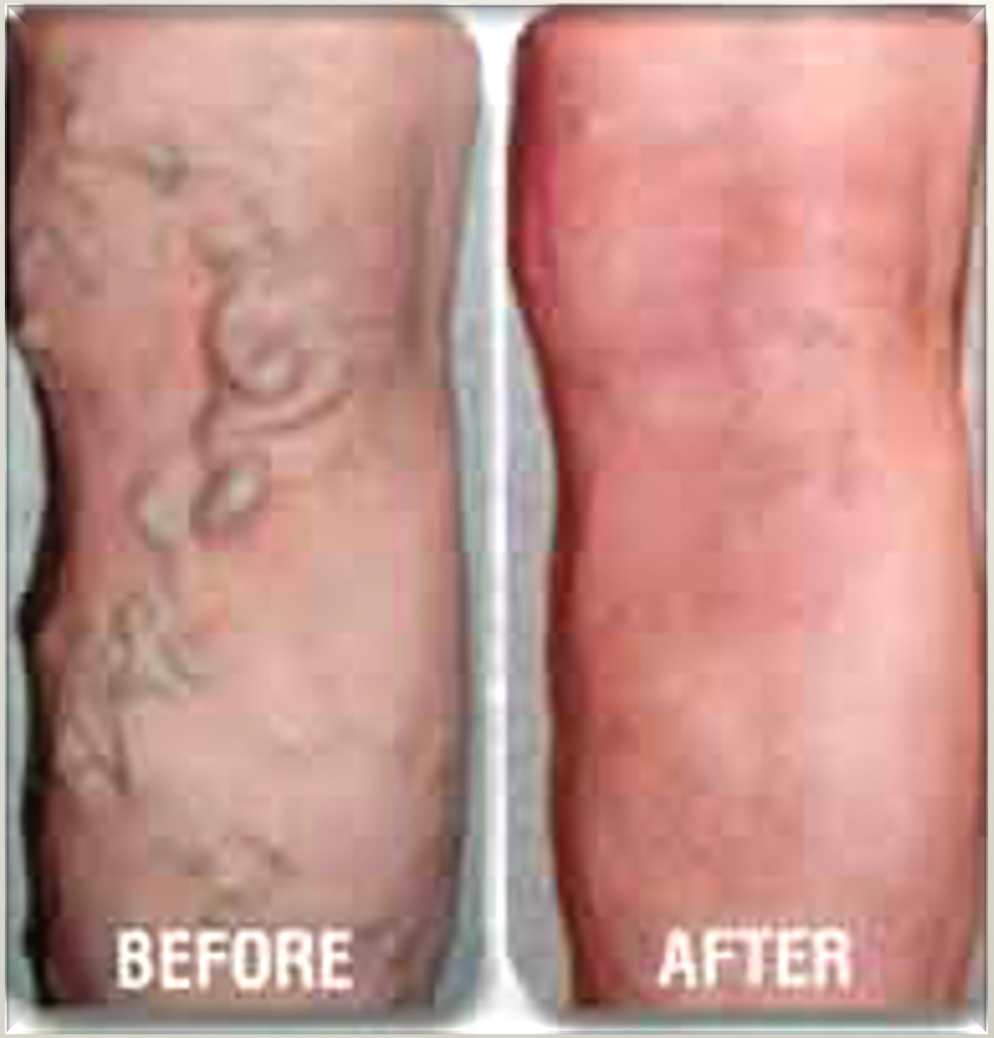
Varicose Veins

- Varicose veins are veins that are tortuous and dilated because of leaky valves
  - 15% of adult population
  - Heredity, prolonged standing, obesity, pregnancy

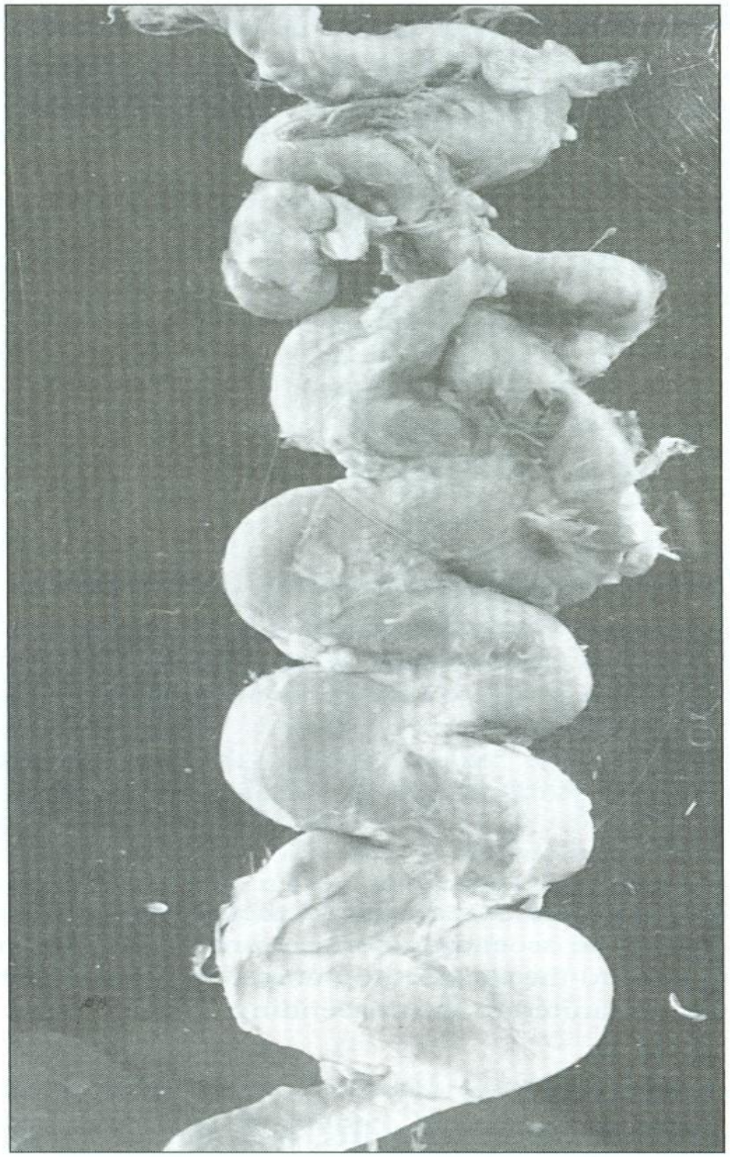


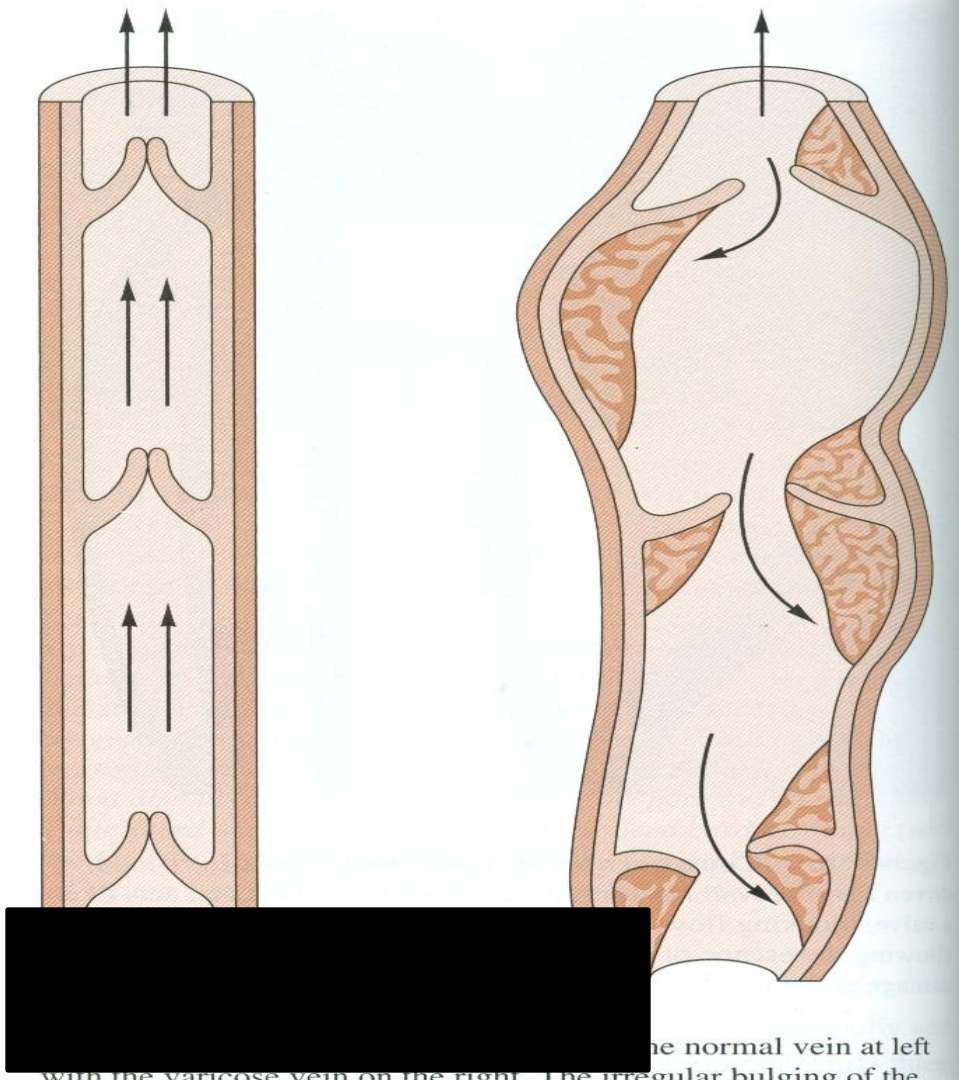





BEFORE
AFTER

Varicose Veins





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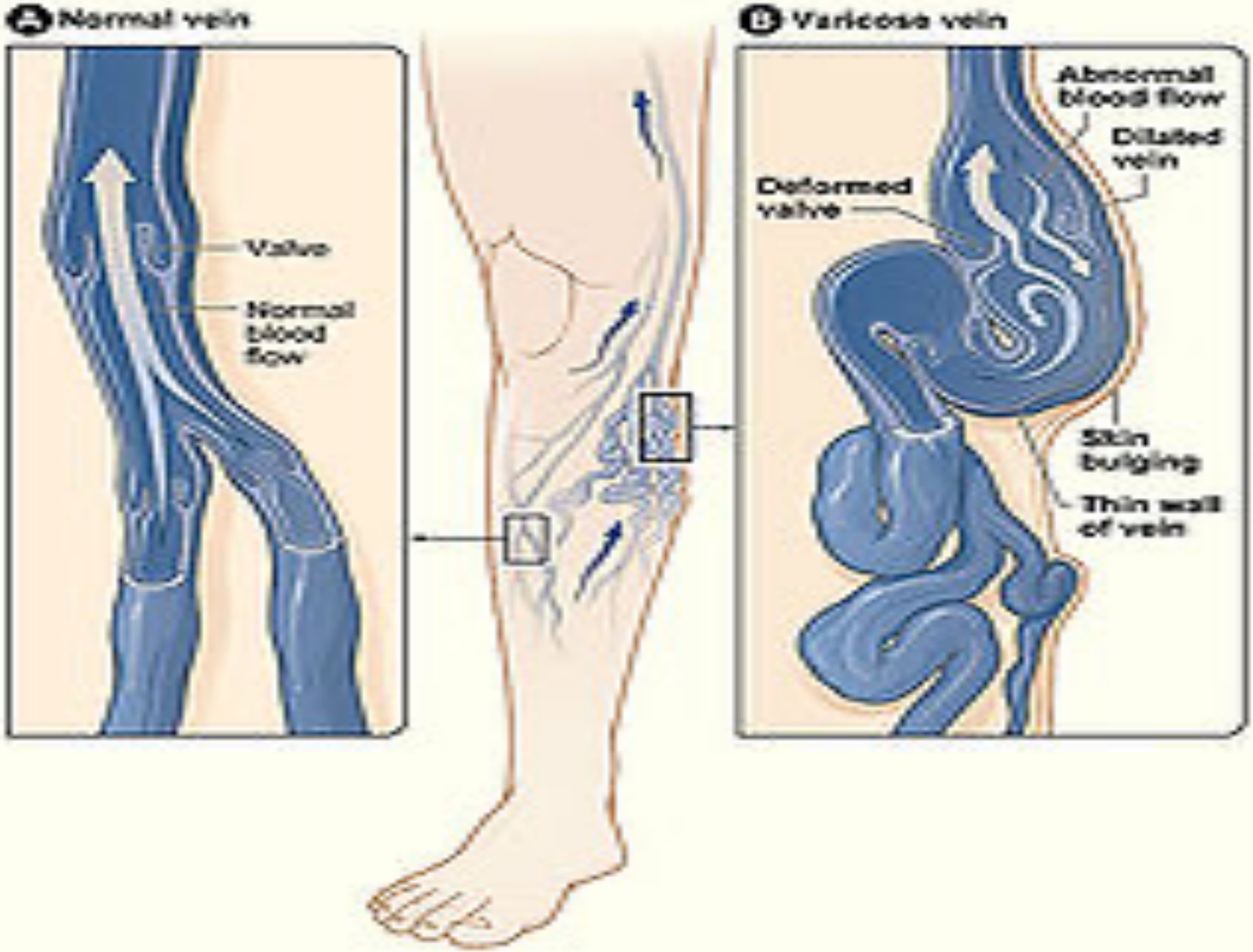
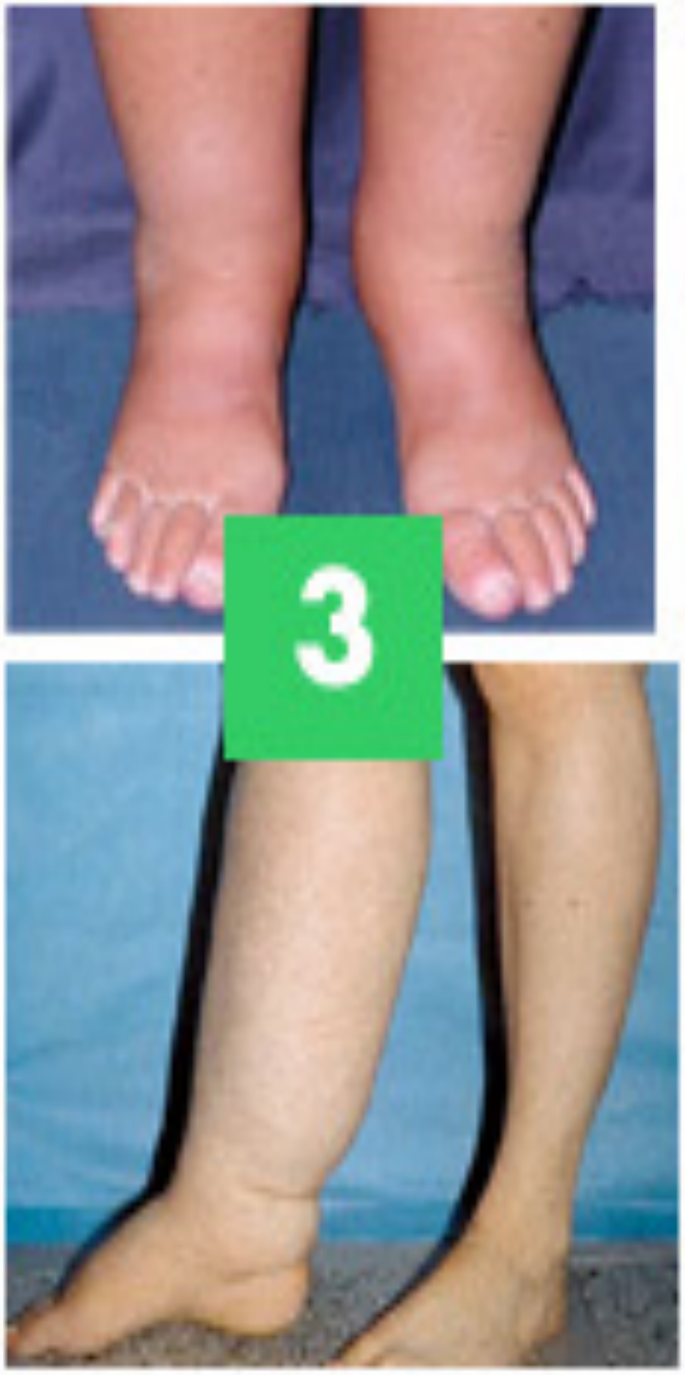
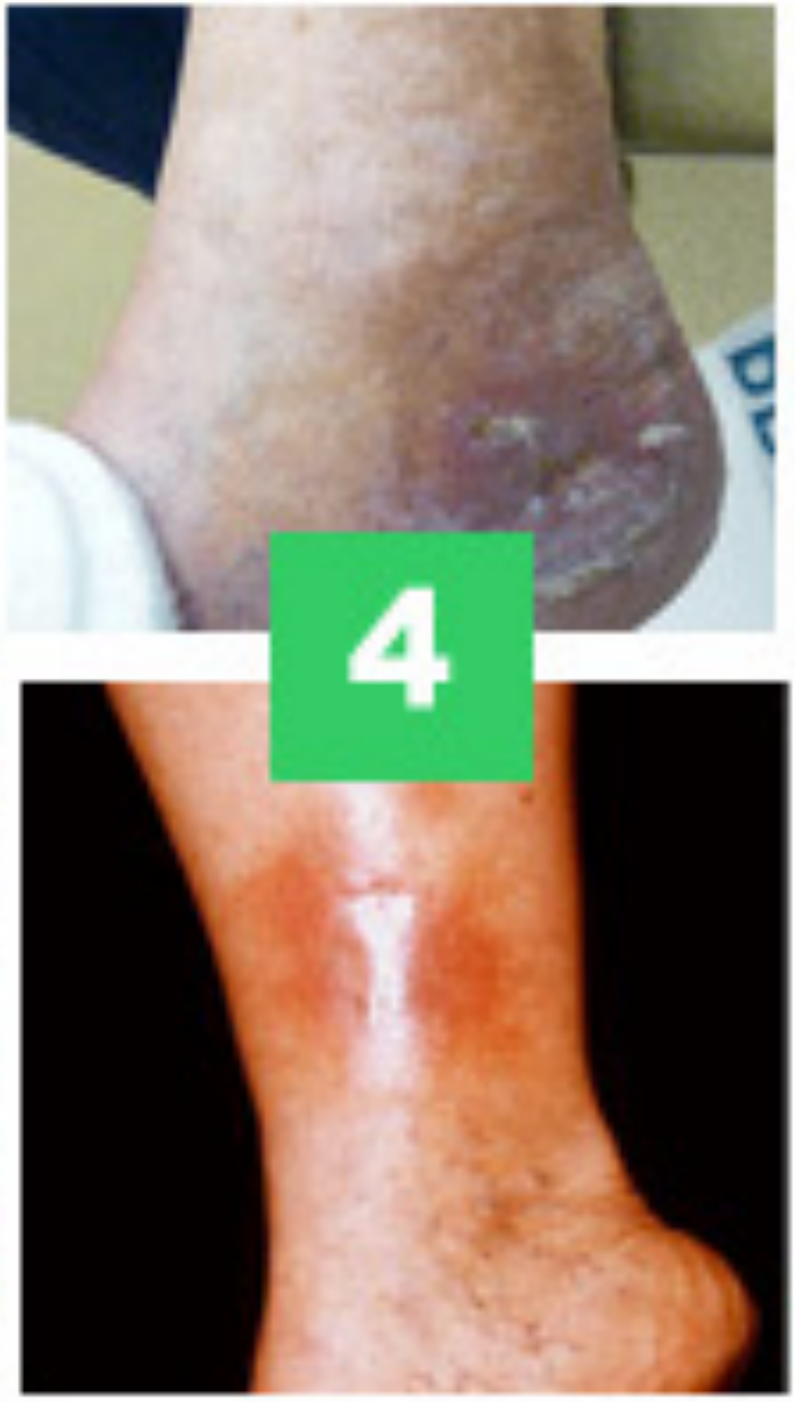
ne normal vein at left with the varicose vein on the right. The irregular bulging of the varices limits complete valve closure and allows some backflow. Note also the developing thrombi, whose formation is favored by

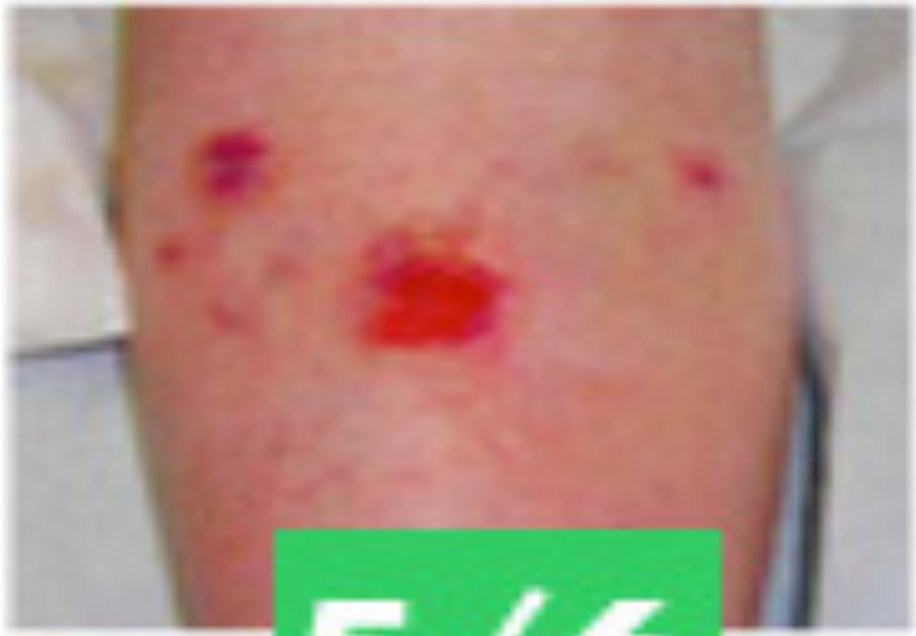

A Normal vein
Valve
Normal blood flow
B Varicose vein
Abnormal blood flow
Dilated vein
Deformed valve
Skin bulging
Thin wall of vein

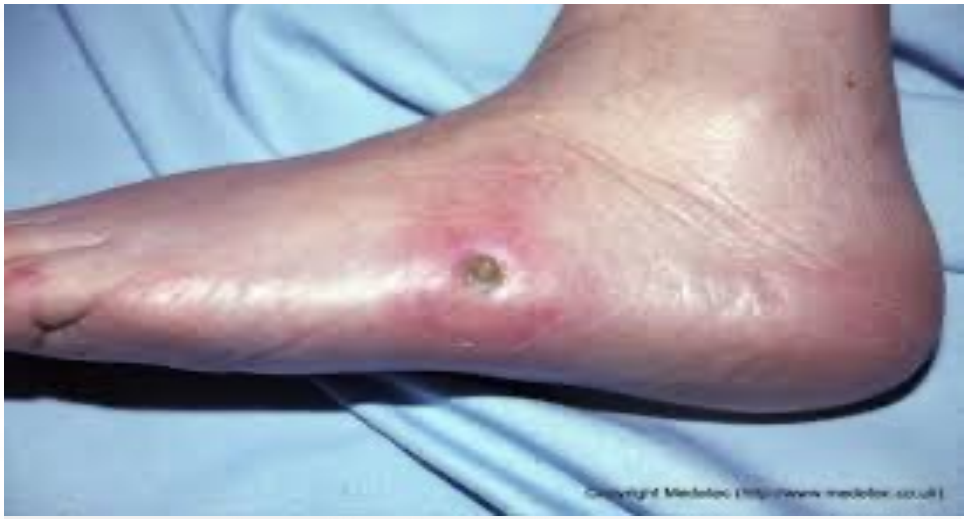


- **Class 3 – Vein Disease: Leg Edema**
  - Slightly more advanced vein disease causes swelling of the legs.
  - The backflow in the venous system interferes with the body's ability to reabsorb fluid leading to swelling of the leg.
  - This swelling resolves at the end of the day when legs are elevated.
  - Vein disease is the most common cause of leg swelling.

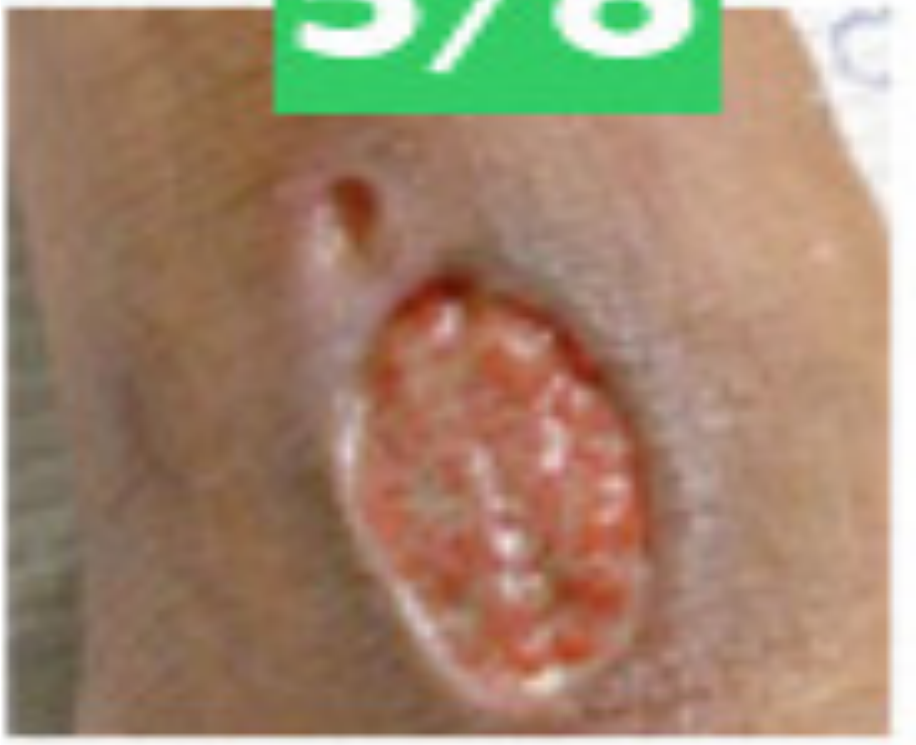


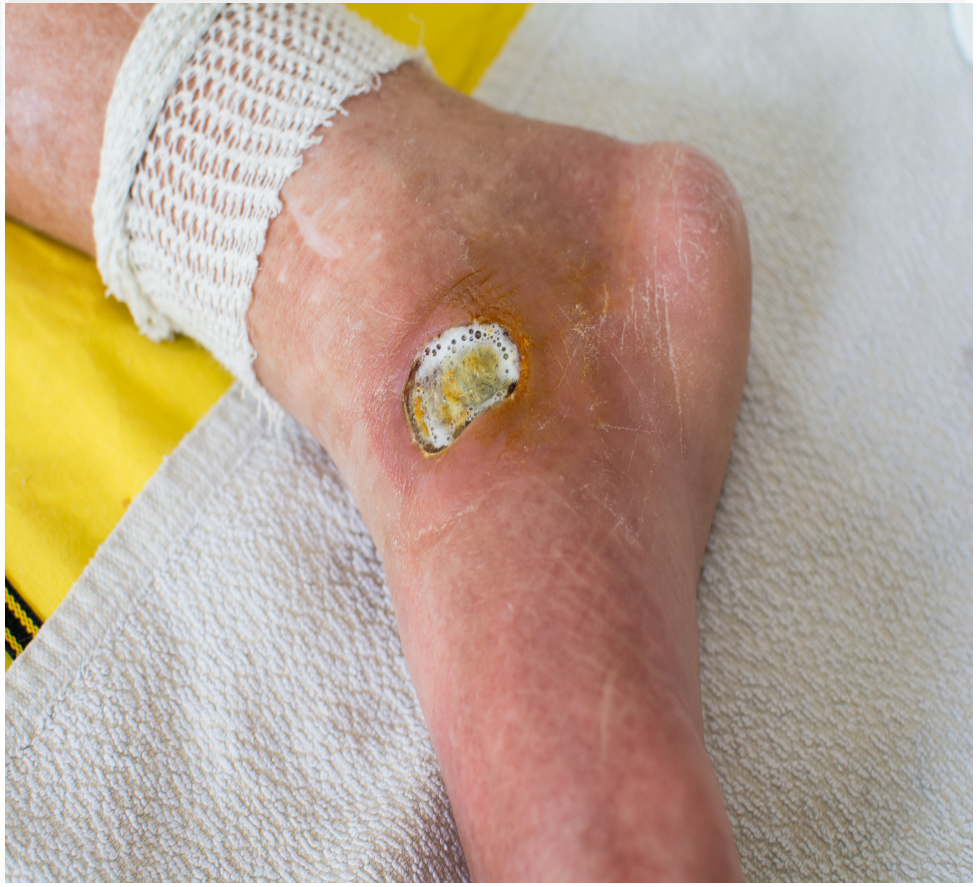
- **Class 4 - Vein Disease: Skin Changes**
  - Over time the venous congestion leads to changes in the skin.
  - The skin becomes thinner and **discolored to reddish brown or whitish** in color.
  - At this vein disease stage the skin is easily injured and heals slowly.





5/6

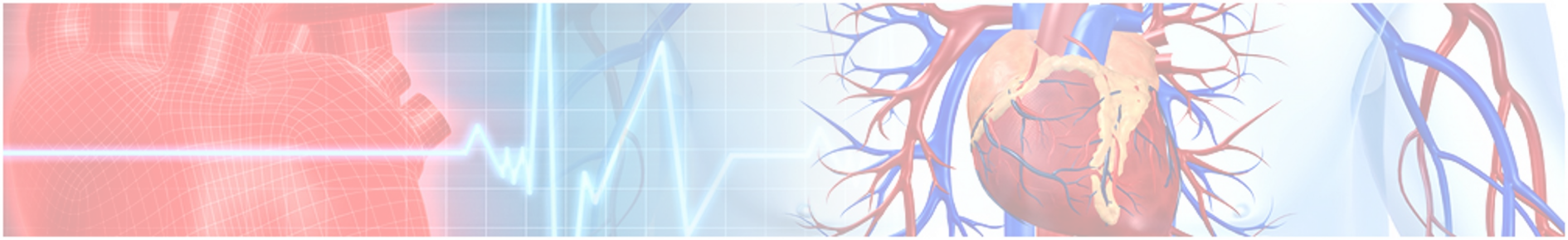




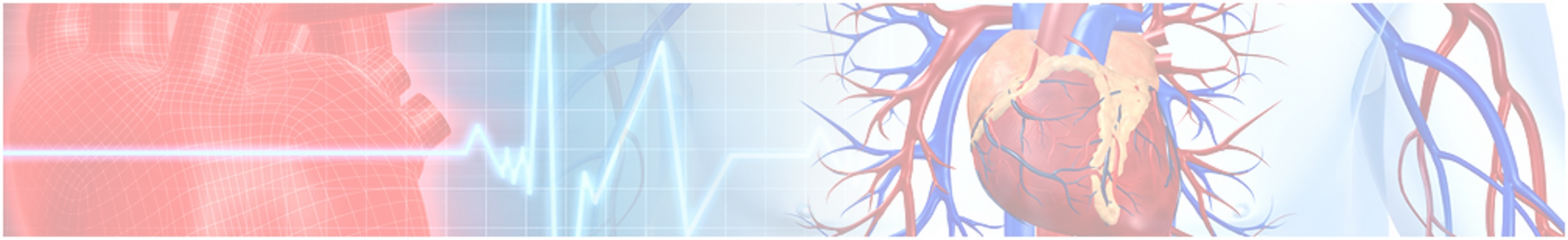
- **Class 5 & 6 – Vein Disease: Leg Ulcers**
  - These are the most advanced forms of superficial vein disease.
  - Venous ulcers are caused by venous congestion that has progressed to point that it interferes with ability for blood flow to provide nutrition to skin. This makes any skin injury heal very slowly, if at all.
  - Without treatment of the underlying vein disease, 20% of these ulcers remain unhealed at 2 years.
  - Class 5 Vein is used to describe legs with healed venous ulcers.
  - Class 6 Vein Disease is used to describe legs with active ulcers.
  - Venous disease is the most common cause of ulcers on the leg.

Deep Vein Thrombosis

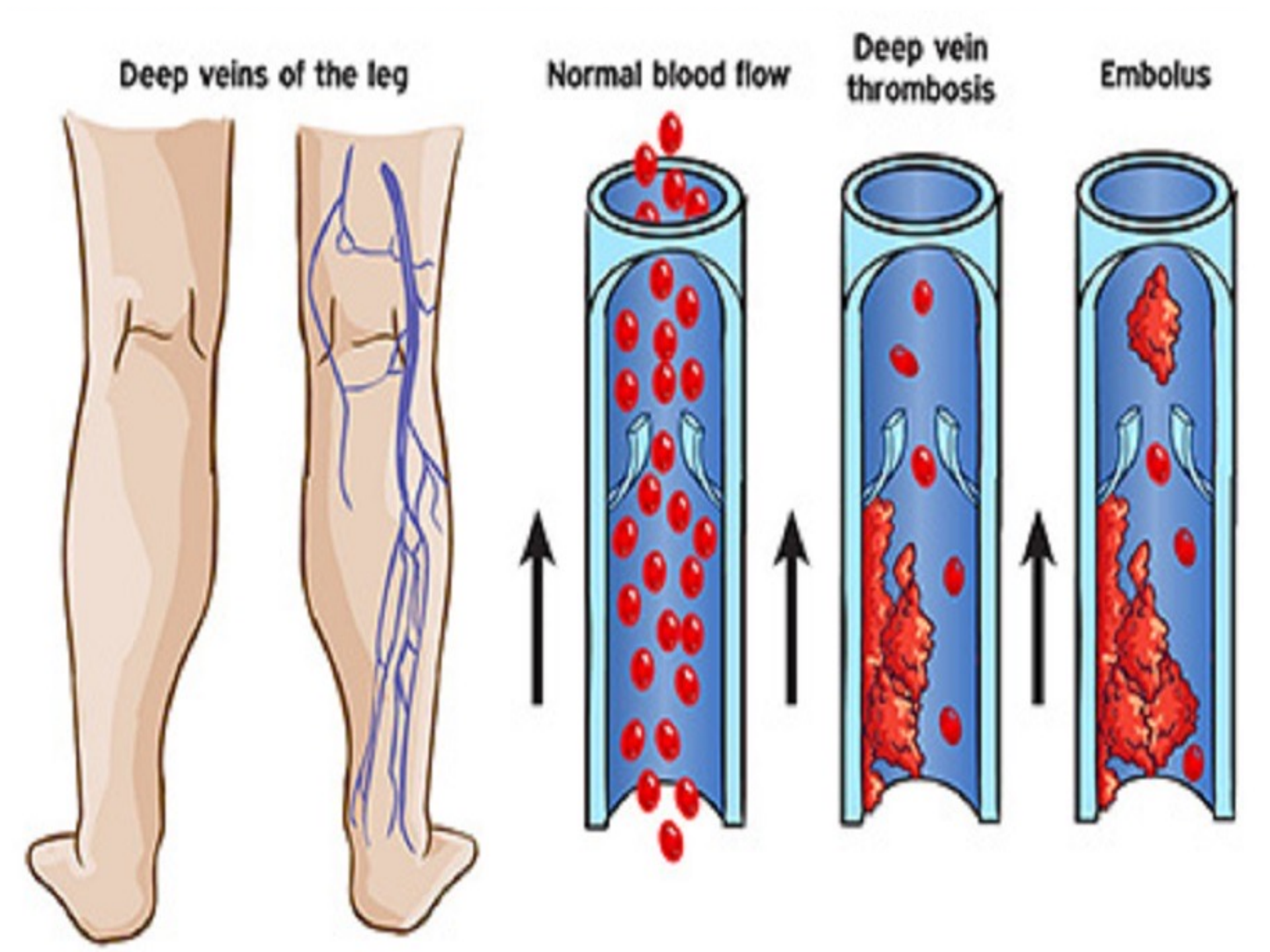
- The formation of a blood clot in a deep vein
- Symptoms may include pain, swelling, redness or warmth of the area
- Half of the cases have no symptoms.
- Complications may include pulmonary embolism, as a result of detachment of a clot which travels to the lungs, and post-thrombotic syndrome.



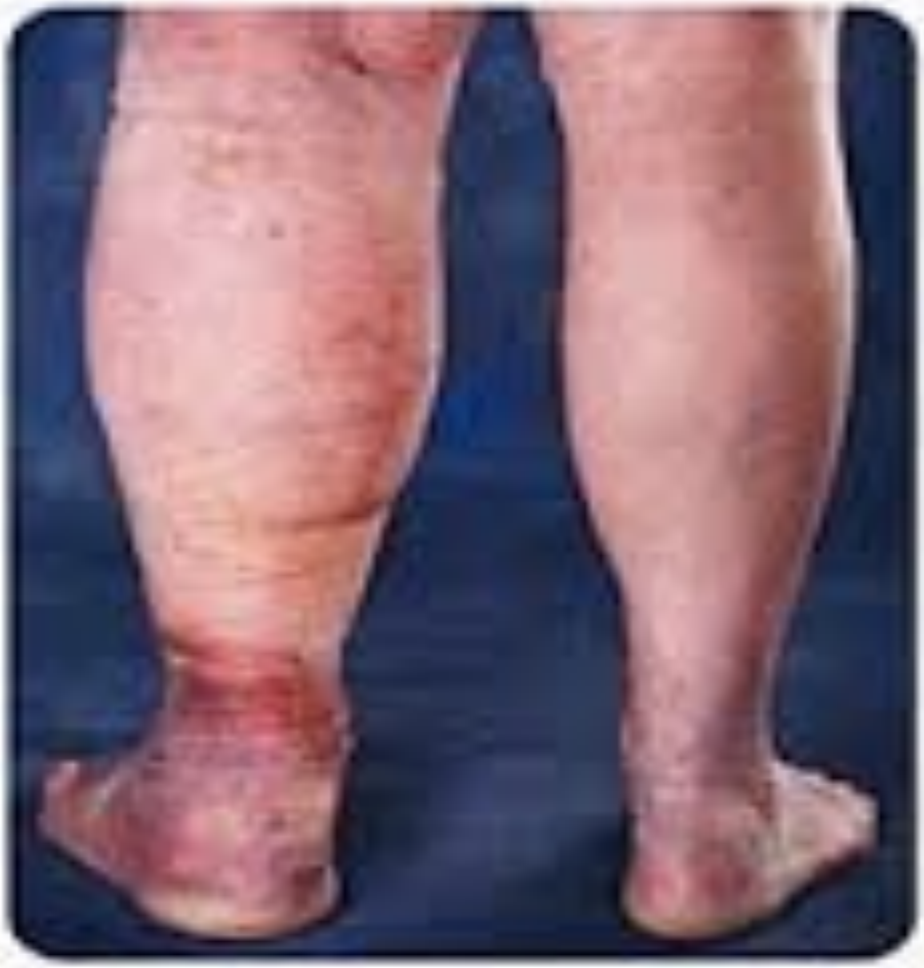
- Risk factors include recent surgery, cancer, trauma, lack of movement, obesity, smoking, hormonal birth control, pregnancy, and the period following birth.
- The underlying mechanism typically involves some combination of decreased blood flow rate, increased tendency to clot, and injury to the blood vessel wall.



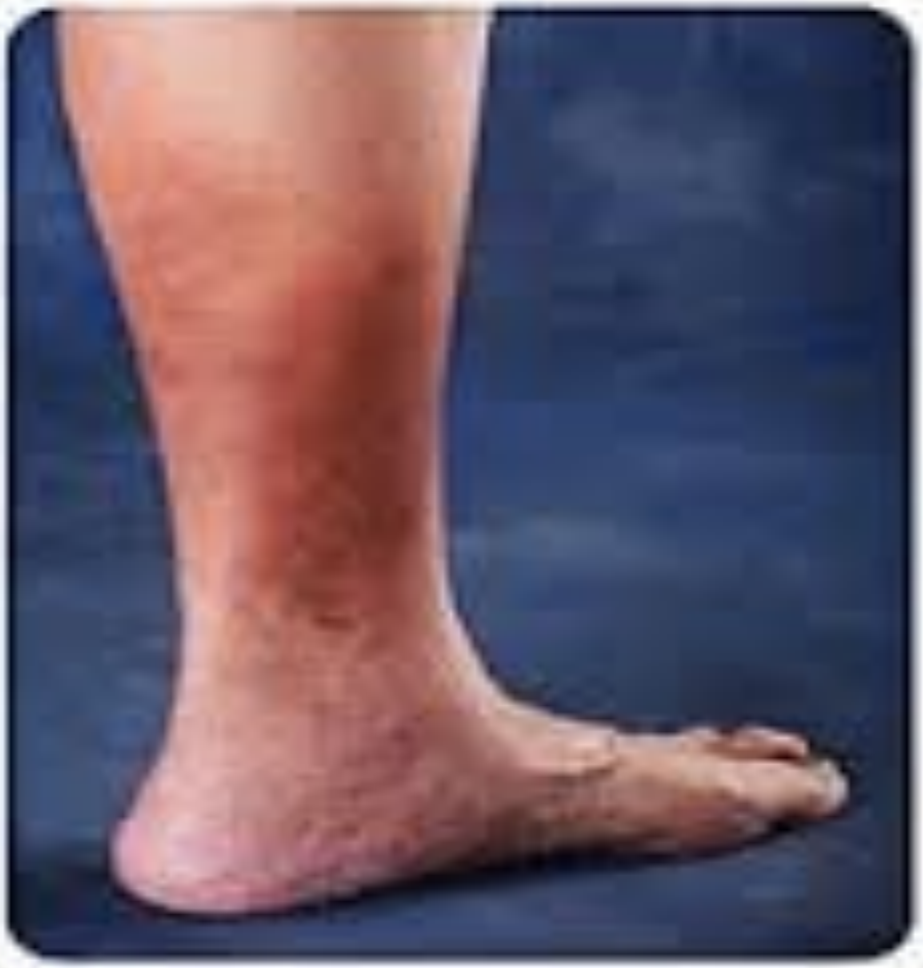
- The three factors of Virchow's triad — Venous stasis, hypercoagulability, and changes in the endothelial blood vessel lining (such as physical damage or endothelial activation)— contribute to DVT and are used to explain its formation


Deep veins of the leg
Normal blood flow
Deep vein thrombosis
Embolus

DVT - Deep Vein Thrombosis



Swelling



Skin Changes

Vein Disorders

Common, but usually not life-threatening

Thrombophlebitis

- Inflammation of a vein as a result of thrombosis (usually in deep leg veins)
- Risk of thromboembolism

Thrombophlebitis