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## Disorders of cardiovascular system

### Hyperemia& Congestion

Both refer to increase volume of blood with in a tissue but they have different underlying mechanisms:

Hyperemia: is an active process from augmented tissue inflow due to arteriolar dilatation.

Congestion: is passive process from impair outflow of blood from tissue

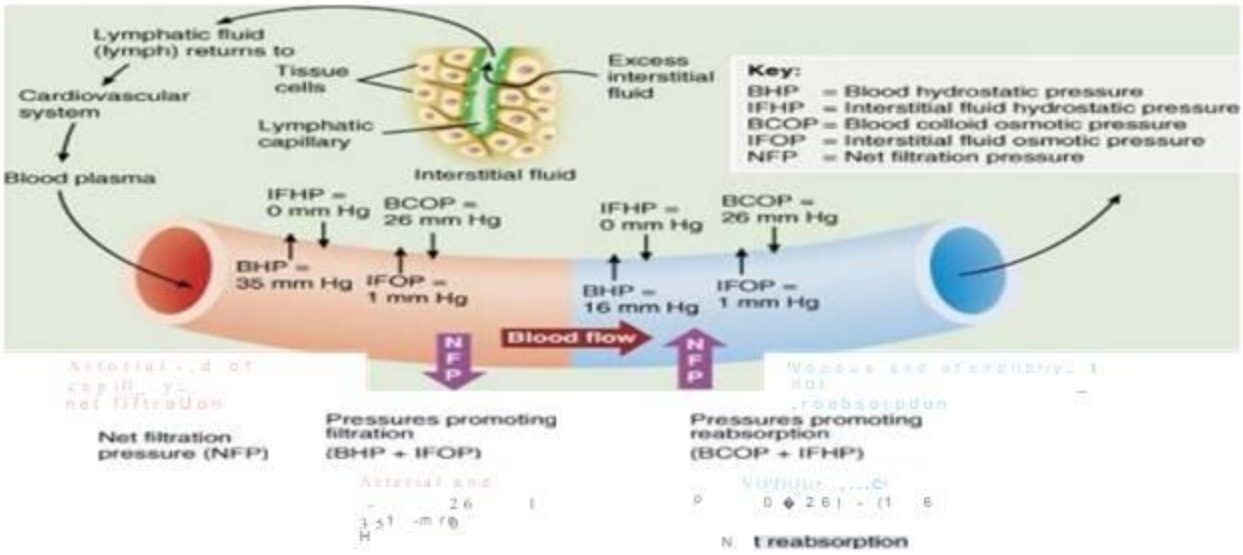
| characteristics | Hyperemia | CONGESTION |
|---|---|---|
| **Mechanism** | Active | Passive |
| **Cause** | Arterial dilatation | Venous obstruction |
| **Tissue color** | Tissues are red (erythema( owing to engorgement with oxygenated blood | Tissues are blue-red (cyanosis( due to an accumulation of deoxyhemoglobin |
| **Type** | .1Localized<br>•Physiological (exercises(<br>•Pathological (inflammation(<br>.2Generalized<br>-Physiological (hot weather(<br>-Pathological -Fever | .1Generalized systemic<br>e.g. right ventricular failure<br>2Pulmonary venous congestion<br>e.g left ventricular failure.<br>-3Localized venous congestion:<br>eg DVT(deep venous thrombosis( of leg. |

### *EDEMA*

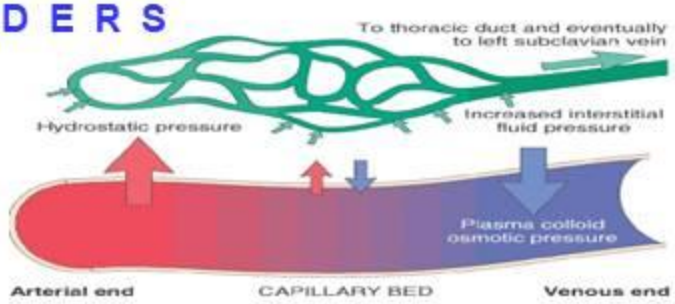
is an accumulation of interstitial fluid within tissues marked by profound swelling of subcutaneous tissues or the accumulation of fluid occurred in body cavities.such accumulations are often referred to collectively as effusions, . Examples in the pleural cavity (hydrothorax), the pericardial cavity (hydropericardium), or the peritoneal cavity

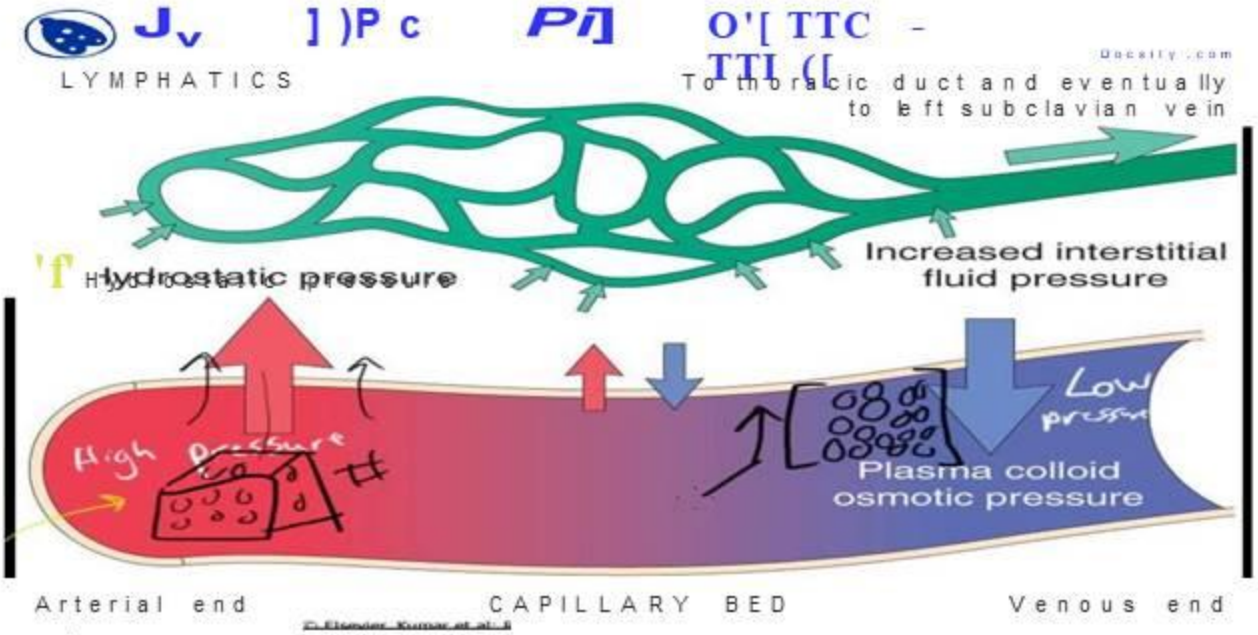
)hydroperitoneum or ascites.(

## Forc s involved c llary Exch ange




## H E I V I O D V N A I V I I C D I S O R D E R S




- 60% of body weight is water.
- Two third of which is intracellular"

- ❖ one third is extra cellular mostly as interstitial fluid" .
- ❖ only 5% of total body water is in blood plasma.

## Causes of edema:

.1 Hydrostatic pressure increased:
   a. Localized increase in venous pressure e g (deep venous thrombosis DVT( with edema of the affected limb.
   b. Generalized increase in venous pressure with systemic edema occur in congestive heart failure

.2 Increased vascular permeability (e.g., inflammation(

.3 Decreased colloid osmotic pressure, due to reduced plasma albumin
   a. decreased synthesis (e.g. liver disease, protein malnutrition(
   b. increased loss (e.g., nephritic syndrome(

.4 lymphatic obstruction (e.g inflammation or neoplasia). Usually localized result from:
   - inflammatory e.g filariasis which causes lymphatic obstruction& lymph node fibrosis inguinal region (ELEPHANTLASIS(
   - Cancer of breast treated by surgery or irradiation with resection of lymphatic there is edema of the arm.
   - In CA breast infiltration &obstruction of superficial lymphatics will cause edema of skin.

**.5** Sodium retention renal failure (eg, renal failure(

## HEMORRHAGE:

The extravasation of blood from vessels, is most often the result of damage to blood vessels or defective clot formation. (internal) include:

- Hematoma encased within tissue
- Petechiae Minute hemorrhage in skin membranes & serosal surfaces.
- Ecchymosis 1-2 cm subcutaneous hematoma and between them called purpura

## Causes of Hemorrhage:

.1Traumas e.g penetrating wounds to the heart.

.2Abnormalities of blood vessel wall:
   a. Inflammation lesion vessels (vasculitis.(
   b. Neoplastic invasion e.g. carcinoma of tongue with invasion of lingual arteries.
   c. Other vascular diseases e.g. (Severe atherosclerosis may so weaken the wall of the abdominal aorta.(

.3High pressure e.g systemic within blood vessel hypertension leading to hemorrhage at sites of arterial weakness.

## Effects of acute hemorrhage:

Depend on volume & rate of blood loss, the effect is slight if less than 20% of blood volume, while sudden loss of 33% of blood volume leads to death. Gradual loss (within 24hour) more than 50% blood volume is not necessarily fatal but it is serious.

## THROMBOSIS:

a blood clot that ouccured to limits bleeding from an injured vessel. The pathologic thrombosis, the formation of blood clot (thrombus) within non traumatized, intact vessels. The primary abnormalities that lead to intravascular thrombosis are Pathogenesis: (causes(

.1 Endothelial injury (e.g., by toxins, hypertension, inflammation or metabolic products(
.2 abnormal blood flow stasis or turbulence (e.g., due to aneurysms, atherosclerotic plaque.(
.3 Increased coagulability of the blood primary (e.g factor V Leiden, increased synthesis, antithrombin deficiency) or secondary (eg, bed rest, tissue damage malignancy(

## Types of thrombi:

| Characteristics | . 1Arterial thrombus | .2venous thrombus |
|---|---|---|
| effects | Usually occlusive | Almost occlusive |
| Sites | Effects Coronary cerebral & Femoral arteries | %95of lower limb |
| Gross features | Gray White, firmly adhere to the wall | Red, soft & gelatinous |
| Microscopic features | Platelets, fibrin, RBC& Degenerated WBC | Contains more RBC (red thrombi( |

## Fate of thrombus:

.1 Dissolution means total lyses of recent thrombus by activation of fibrinolytic system.
.2 Organization recanalization: Ingrowth of endothelial cells, smooth muscle cells& fibroblasts into fibrin rich thrombus will create conduits from one end of thrombus to other & re-establish continuity of original lumen
.3 Thrombus accumulates more platelets & fibrin & lead to obstruction.
.4 Embolization : Thrombus dislodged & transported to other sites.

## Embolism:

An embolus is a detached intravascular solid, liquid, or gaseous mass that is carried by the blood from its point of origin to a distant site, where it often causes tissue dysfunction or infarction.

- **Pulmonary emboli** derive primarily from lower-extremity deep vein thrombi their effects depend the size of the mainly on embolus and the location in which it lodges. Consequences may include heart failure pulmonary hemorrhage pulmonary infarction or sudden death.
- **Systemic emboli** derive primarily from cardiac mural or valvular thrombi, or plaques; whether an embolus causes tissue infarction depends on the site of embolization and the presence or absence of collateral circulation.

**Types of emboli according to the constituents: Solid liquid, gaseous**

- **Solid emboli:** it can be
  i. a fragments detached from a thrombus, this is called thromboembolic phenomenon
  ii. some fragments of an ulcerated atherosclerotic plaque.
  iii. tumor fragments.
  iv. droplets of fat as in fracture of long bone (fat embolism(
- **Fluid emboli Amniotic fluid embolus.**
- **Gaseous emboli: as in air embolism.**

## INFARCTION

- **INFARCT:** are area of ischemic necrosis most commonly caused by arterial occlusion (typically due to thrombosis or embolization); venous outflow obstruction is a less frequent cause.
- Infract caused by venous occlusion or occurring in spongy tissues typically are hemorrhagic (red). those caused by arterial occlusion in compact tissues typically are pale white(
- Whether not vascular occlusion causes tissue infarction is influenced by collateral blood supplies, the rate which an obstruction develops susceptibility to ischemia injury and decreased blood oxygenation.

## Shock:

define as a state of system tissue hypo perfusion reduced cardiac output and/or reduced effective circulating blood volume. The consequences are impaired tissue perfusion and cellular hypoxia.

Shock is the final common pathway for several potentially lethal events including extensive trauma burns, myocardial infarction, pulmonary embolism, and sepsis.

**Classification:**

-1Hypovolemic shock: in which there is a real decrease in blood volume.

Causes: "Hemorrhage, Fluid loss as in severe vomiting, diarrhea & burns.

Mechanism of development is inadequate blood or plasma volume.

-2 Cardiogenic shock:(Pumping failure(

Causes: Myocardial infarction. Rupture of the heart. Arrhythmias.

-3 Septic shock Causes: overwhelming bacterial infection (gram negative septicemia or endotoxic shock) or gram positive septicemia.

-4 Neurogenic shock Causes: Anesthesia & spinal cord injury

-5 Mechanism shock: peripheral vasodilatation.

**Stages of shock:**

-1**Non progressive phase:** which is the compensatory phase .In this stage a compensatory

mechanisms operate to maintain cardiac output &blood pressure near normal levels The compensatory mechanisms include:

a- Arteriolar constriction leading to increase blood pressure.

b- Increase heart rate &cardiac output.

c- Retention of fluid through increase secretion of ADH & activation of rennin angiotensin aldosterone axis to retain fluid

-2**Progressive phase:** when an additional factor is added like extensive burn complicated by bacterial infection. In this stage, despite the compensatory mechanisms, there is progressive decline in blood pressure &cardiac output. Clinically observed increase in respiratory rate &decrease in urine output reflecting pulmonary &renal hypoperfusion.

-3Irreversible phase result from irreversible injury to the cell membrane as manifested by paralysis of sodium-potassium pump & defect in cell membrane so cell contents go to outside .The reduction in blood flow to the vital organs such as brain, heart, kidney lead to ischemic cell death in these organs.

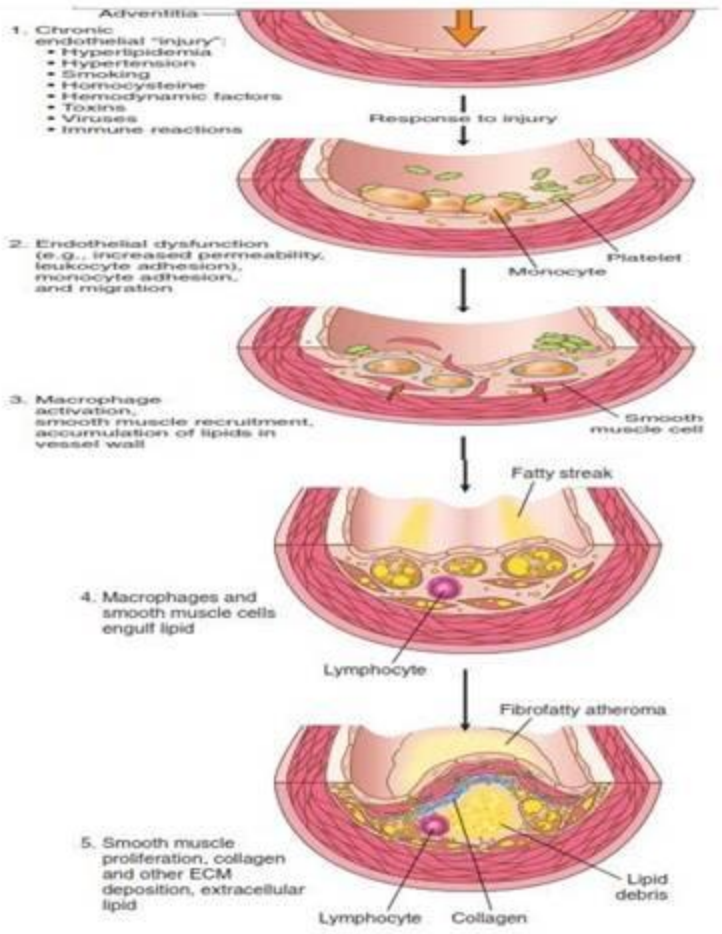
**Arteriosclerosis**: Is a generic term for thickening & Loss of elasticity of arterial Walls, it occurs in three forms.

1- **Arteriolosclerosis:** affects small arteries and arterioles and may cause downstream ischemic injury. The two variants hyaline and hyperplastic arteriolosclerosis. Most often in patient with hypertension & diabetes mellitus

-2 **Atherosclerosis** the most frequent& important pattern

-3 **Monckeberg medial calcific sclerosis** : characterized by calcific deposits in muscular arteries persons older in than 50 years, (Radiological visible & often palpable calcification.(

-4 **Fibromuscular intimal hyperplasia:** is a non-atherosclerotic process that occurs in muscular arteries larger than arterioles. This is predominantly an SMC- and ECM-rich lesion driven by inflammation (as in a healed arteritis or transplant-associated arteriopathy

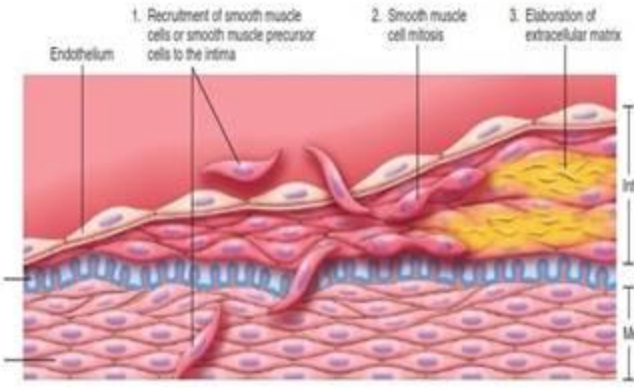



## Atherosclerosis ATH :

characterized by intimal lesion called Atheromas, atheromatous and fibrofatty plagues that obstruct vascular lumen and can rupture to cause sudden occlusion.

### Pathogenesis of ATH

The currently held view of pathogenesis is embodied in the



response-to-injury hypothesis. This model views atherosclerosis involves interaction of modified lipoproteins, monocyte derived macrophages, T lymphocytes, and the cellular constituents of the arterial wall as a chronic inflammatory response of the arterial wall to endothelial injury.

**Pathogenic events:**

.1EC injury—and resultant endothelial dysfunction leading to increased permeability, leukocyte adhesion, and thrombosis

.2Accumulation of lipoproteins (mainly oxidized LDL and cholesterol crystals) in the vessel wall

.3Platelet adhesion

**.4**Monocyte adhesion to the endothelium, migration into the intima, and differentiation into macrophages and foam cells

.5 Lipid accumulation within macrophages, which respond by releasing inflammatory cytokines

.6 SMC recruitment due to factors released from activated platelets, macrophages, and vascular wall cells

.7 SMC proliferation cells, including SMCs, macrophages, and T cells and ECM production CM, including collagen, elastic fibers, and proteoglycans; And intracellular and extracellular lipid.

**Morphology of ATH: ATH passes in 3 stages:**

-1**Fatty streaks** characterized by intimal thickening &lipid accumulation, this is the precursor of atheromas.

-2**Atheromatous Plaques:** These are raised focal lesions within the intima, also called (Fibrofatty or Fibrolipid Plaques) These plaques consist of two parts:

i. Core: soft, yellow, core. E Consist of cholesterol & cholesterol esters.

ii. Cap: firm, white, fibrous cap.

These plaques are varied in their size from 0.3 to 1.5cm in diameter sometimes they are coalesce to form large masses in lumen.

-3 **Advanced atheromatous lesions:**

- ❖ Atheromatous lesions usually involve the arterial wall only partially& ‹ patchy around its circumference (eccentric lesions.(
- ❖ Then Atheromatous lesions become more & more numerous & diffuse as the disease is advanced

**Disterbution of ATH (site:(**

-1Lower abdominal aorta: mast common site of involvement mainly around the ostia of its major branches

-2Then coronary arteries popliteal , internal carotid artery branches circle of willingness.

**Complication of ATH lesion:**

-1 Hemorrhage:
Hemorrhage into the plaque( mainly in coronary arteries).may result in formation of Hematoma within the plaques that increase the risk of rupture.

-2 Focal rupture, ulceration,& erosion of the luminal surface of atheromatous plaque &cholesterol emboli formation.

-3 superimposed thrombosis: This is a most feared complication: usually follow the rupture ulceration, & erosion of luminal surface of plaque

-4 Aneurysmal dilatation

## Risk Factors of ATH:

**-1major risk factors: this include**

**A. Unmodified risk factors:**

i. Age is a dominant at middle age or later.
ii. Sex: Male more prone to ATH than the female (due to hormonal causes(
iii. Familial predisposition
**iv.** Genetic abnormalities**.**

**B. Modifiable Major risk factors (Potentially controllable:(**

- **Hyperlipidemia:**

❖ LDL is a major components lipid profile that are associated with increase of ATH (LDL precipitate the cholesterol vascular wall(

❖ HDL is protective & lipid against (HDL mobilize cholesterol from existing atheroma & transport cholesterol to the liver for excretion in the bile.(

-2**Hypertension** : Those with blood pressure 170 /95 mmHg have 5 folds greater risk of IHD(ischemic heart disease) than those blood pressure of 140/80 mmHg.

**-3Cigarettes smoking**

**-4Diabetes Mellitus**

## C. Less & Uncertain quantitative risk factors:

.1obesity (because obesity increases risk hypertension, diabetes mellitus hypertriglyceridemia, & decrease HDL(
.2Physical inactivity.
.3Stressful lifestyle (type A personality(
.4High carbohydrate intake.
.5postmenopausal estrogen treatment.
.6Unsaturated fat intake.
.7Chlamydia pneumoniae.

## Hypertension:

A common disorder affecting 25% of the population; it is a major risk factor for atherosclerosis، congestive heart failure, and renal failure. It may be primary )idiopathic) or less commonly secondary to an identifiable underlying condition. Sustained diastolic pressure in excess of 90 mmHg or a sustained systolic pressure in excess of 140 mmHg is considered as hypertension.

**Causes of hypertension:**

- ❖ 95% of cases of hypertension are idiopathic or essential these cases about 95% of this cases are benign hypertension. It is a complex, multifactorial disorder, involving both environmental influences and genetic polymorphisms that may influence resorption, aldosterone pathways, the adrenergic nervous system, and the renin-angiotensin system, & 5% malignant or accelerated hypertension(
- ❖ cases are secondary hypertension

## Causes of secondary hypertension

**.1Renal causes** like acute glomerulonephritis

**.2Endocrine causes:** like

- Cushing syndrome (increase steroid hormones(
- Exogenous steroid intake.
- Pheochromocytoma (adrenalin & noradrenalin secreting tumor(
- Acromegaly ( increase level of growth hormone(
- hypothyroidism & hyperthyroidism
- pregnancy induced hypertension.

**.3Cardiovascular causes.** Coarctation of aorta

**.4Neurologic** causes increased intracerebral pressure.

## ANEURYSM

Congenital or acquired dilations of the heart or blood vessels that involve the entire wall thickness. Complications are related to rupture, thrombosis, and embolization. Aneurysms are congenital or acquired dilations of the heart or blood vessels that involve the entire wall thickness. Dissections occur when blood enters the wall of a vessel and separates the various layers. Complications are related to rupture, thrombosis, and embolization. Aneurysms and dissections result from structural weakness of the vessel wall caused by loss of SMCs or weakening of the ECM, which can be a consequence of ischemia, genetic defects, or defective matrix remodeling.

- True aneurysm (when aneurysm is bounded by arterial wall components or the attenuated wall of the heart(
- False aneurysm, (pseudoaneurysm) is a beach in the vascular wall leading to an extravascular space.

Examples on true aneurysm are Atherosclerotic, syphilitic, & congenital vascular aneurysm. Examples on false aneurysm are post-myocardial rupture within a pericardium

## Causes of aneurysm:

-1 ATH -2Cystic medial degeneration arterial medial.

-3 Trauma. -4Congenital defect (Berry aneurysm(

-5 Infections (Mycotic aneurysm).or Syphilis. -6Vasculitis