- **Thromboembolic Disease**
- **Shock**

Hemodynamic Disorders

- Edema (increased fluid in the ECF)
- Hyperemia (INCREASED flow)
- Congestion (INCREASED backup)
- Hemorrhage (extravasation)
- Hemostasis (opposite of thrombosis)
- Thrombosis (clotting blood)
- Embolism (downstream travel of a clot)
- Infarction (death of tissues w/o blood)
- Shock (circulatory failure/collapse)

EDEMA

- 2/3 of body water is INTRA-cellular,the rest is INTERSTITIAL,only 5% is INTRA-vascular
- **EDEMA is SHIFT to the INTERSTITIAL SPACE**

  -THORAX, -PERICARDIUM, -PERITONEAL

- **<u>ONLY 4 POSSIBILITIES!!!</u>**
  - Increased Hydrostatic Pressure
  - Reduced Oncotic Pressure
  - Lymphatic Obstruction
  - Sodium/Water Retention

INCREASED HYDROSTATIC PRESSURE

- Impaired venous return
- Congestive heart failure
- Constrictive pericarditis
- Ascites (liver cirrhosis)
- Venous obstruction or compression
- Thrombosis
- External pressure (e.g., mass)
- Lower extremity inactivity with prolonged dependency
- Arteriolar dilation
- Heat

REDUCED PLASMA ONCOTIC PRESSURE (HYPOPROTEINEMIA)

- Protein-losing glomerulopathies (nephrotic syndrome)
- Liver cirrhosis (ascites)
- Malnutrition
- Protein-losing gastroenteropathy

LYMPHATIC OBSTRUCTION (LYMPHEDEMA)

- Inflammatory
- Neoplastic
- Postsurgical
- Post irradiation

Na+ RETENTION

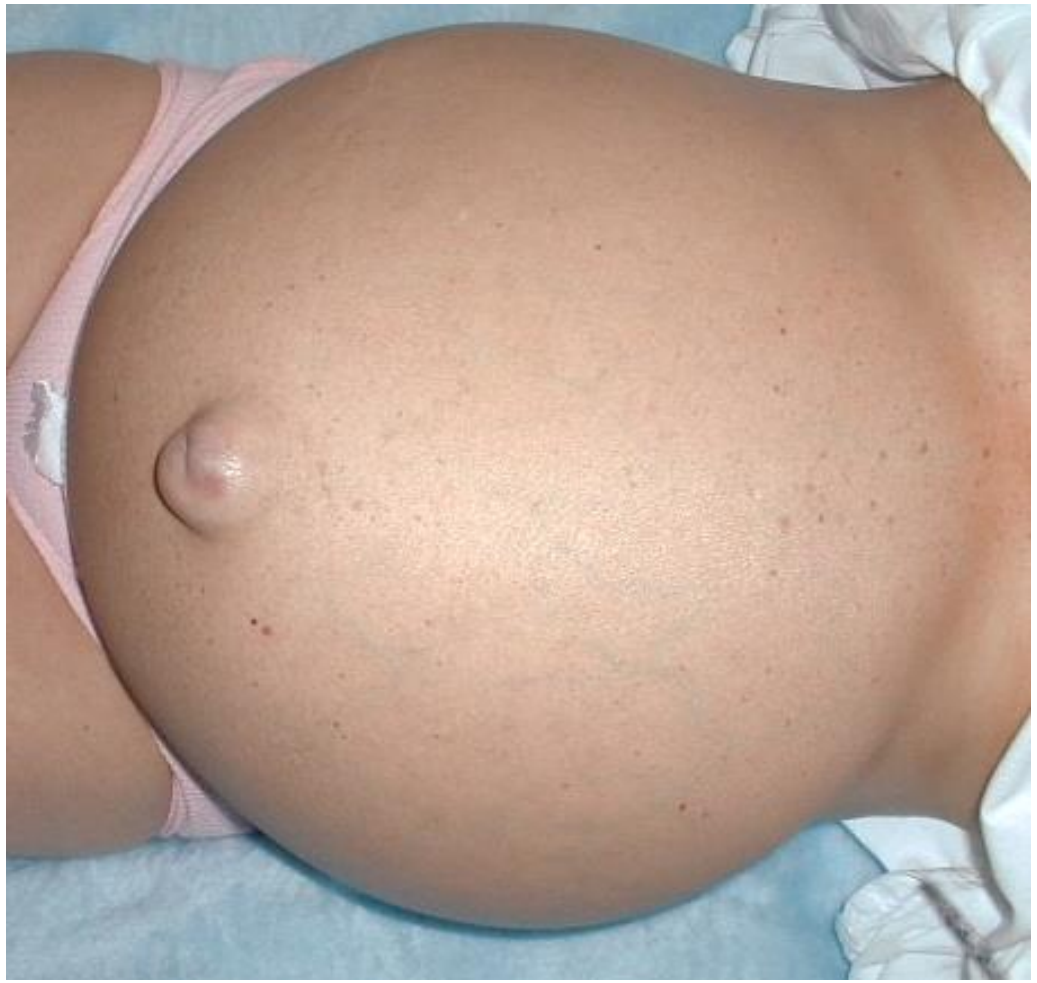
- Excessive salt intake with renal insufficiency
- Increased tubular reabsorption of sodium
- Renal hypo perfusion→ Increased renin-angiotensin-aldosterone secretion

CHF EDEMA

- INCREASED VENOUS PRESSURE DUE TO FAILURE
- DECREASED RENAL PERFUSION, triggering of RENIN-ANGIOTENSION-ALDOSTERONE complex, resulting ultimately in SODIUM RETENTION

HEPATIC ASCITES

- PORTAL HYPERTENSION
- HYPOALBUMINEMIA



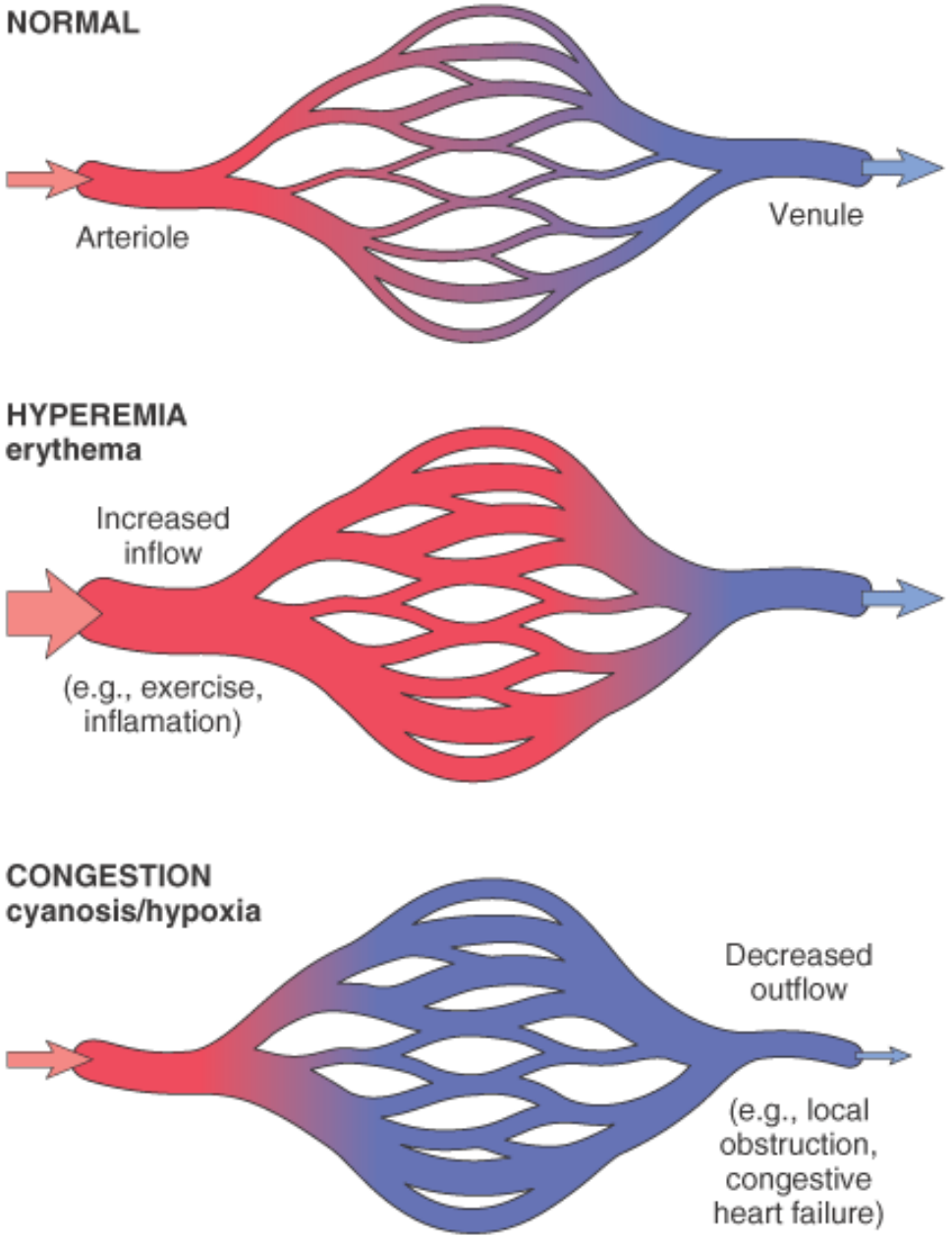
RENAL EDEMA

- SODIUM RETENTION
- PROTEIN LOSING GLOMERULOPATHIES (NEPHROTIC SYNDROME)

Transudate vs Exudate

- Transudate
  - results from disturbance of Starling forces
  - specific gravity < 1.012
  - protein content < 3 g/dl, LDH LOW
- Exudate
  - results from damage to the capillary wall
  - specific gravity > 1.012
  - protein content > 3 g/dl, LDH HIGH

HYPEREMIA/(CONGESTION)



HYPEREMIA

Active Process

CONGESTION

Passive Process

Acute or Chronic

CONGESTION

- LUNG
  - ACUTE
  - CHRONIC
- LIVER
  - ACUTE
  - CHRONIC
- CEREBRAL

HEMORRHAGE

- EXTRAVASATION beyond vessel
- “HEMORRHAGIC DIATHESIS”
- HEMATOMA (implies MASS effect)
- “DISSECTION”
- PETECHIAE (1-2mm) (PLATELETS)
- PURPURA <1cm
- ECCHYMOSES >1cm (BRUISE)
- HEMO-: -thorax, -pericardium, -peritoneum, HEMARTHROSIS
- ACUTE, CHRONIC

EVOLUTION of HEMORRHAGE

- ACUTE→ CHRONIC
- PURPLE→ GREEN→ BROWN
- HGB→ BILIRUBIN→ HEMOSIDERIN

HEMATOMA vs. “CLOT”

Hemostasis PLAYERS

- ENDOTHELIUM
- PLATELETS
- COAGULATION “CASCADE”

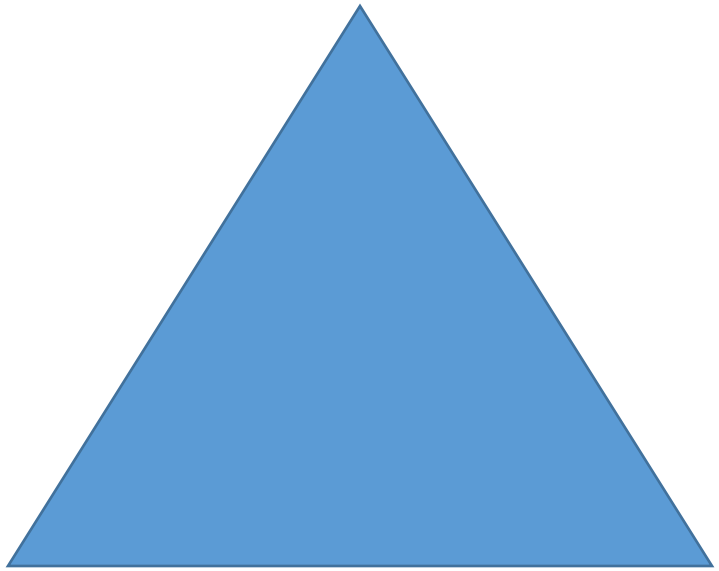
THROMBOSIS

- Pathogenesis
- Endothelial Injury
- Alterations in Flow
- Hypercoagulability
- Morphology
- Fate
- Clinical Correlations
- Venous
- Arterial (Mural)

THROMBOSIS

•Virchow's TRIANGLE

ENDOTHELIAL INJURY

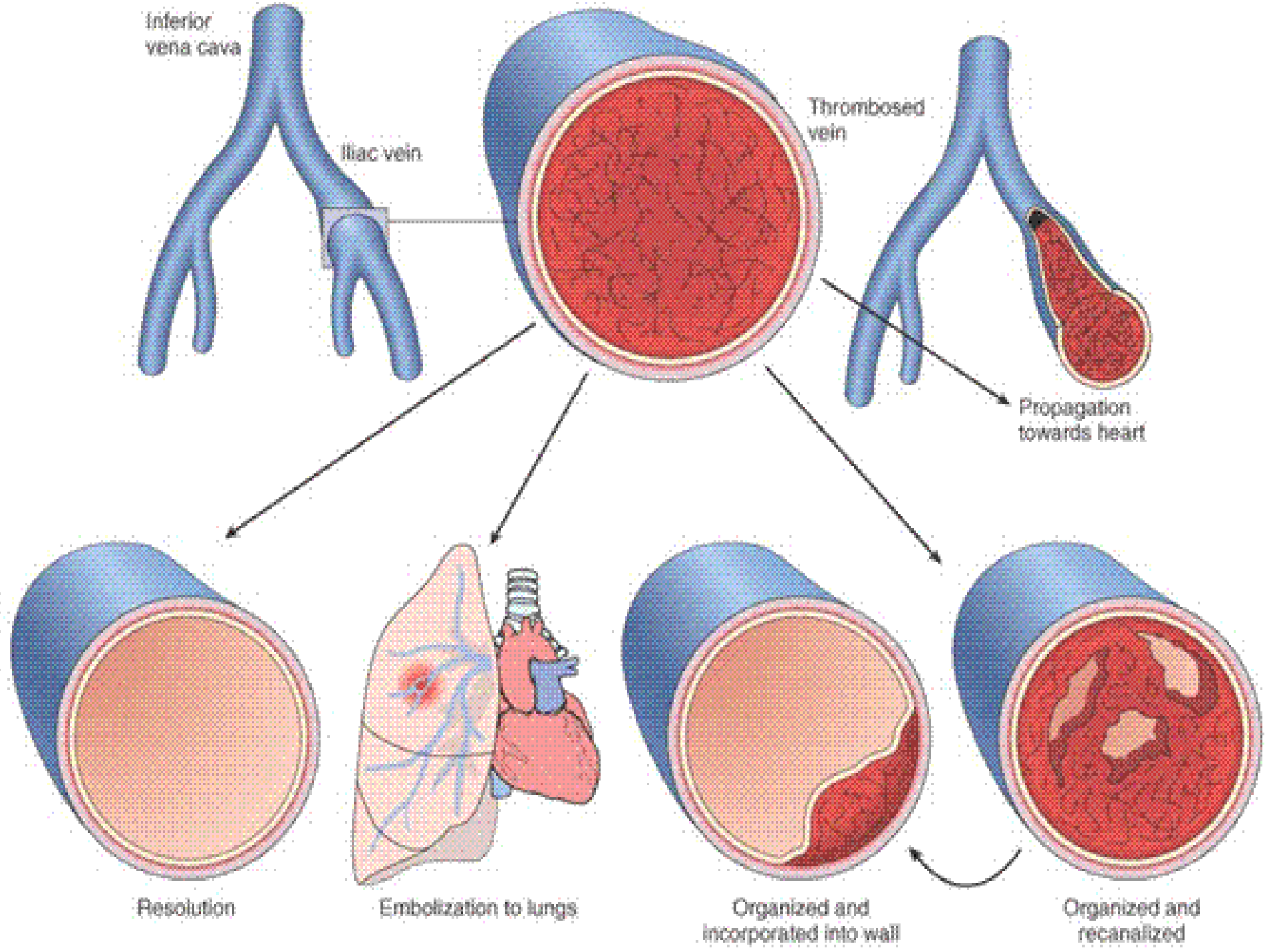


ABNORMAL FLOW (NON-LAMINAR)

HYPER-COAGULATION

FATE of THROMBI

- PROPAGATION (Downstream)
- EMBOLIZATION
- DISSOLUTION
- ORGANIZATION
- RECANALIZATION


Inferior
vena cava
Iliac vein
Thrombosed
vein
Propagation
towards heart
Resolution
Embolization to lungs
Organized and
incorporated into wall
Organized and
recanalized

EMBOLISM

- Pulmonary
- Systemic (Mural Thrombi and Aneurysms)
- Fat((long bone fx's, also bones with marrow)
- Air
- Amniotic Fluid(very prolonged or difficult delivery, high mortality)

SYSTEMIC EMBOLI

- “PARADOXICAL” EMBOLI
- 80% cardiac/20% aortic
- Embolization lodging site is proportional to the degree of flow (cardiac output) that area or organ gets, i.e., brain (15%), kidneys (~25%), legs, splanchnic (~25%), liver (~25%)

INFARCTION

- Defined as an area of necrosis* secondary to decreased blood flow
- HEMORRHAGIC vs. ANEMIC
- RED vs. WHITE
  - END ARTERIES vs. DUAL ARTERY SUPPLY

- ACUTE→ORGANIZATION→FIBROSIS

SHOCK

- Definition: CARDIOVASCULAR COLLAPSE

- Common pathophysiologic features:
  - INADEQUATE CARDIAC OUTPUT and/or
  - INADEQUATE BLOOD VOLUME

GENERAL RESULTS

- INADEQUATE TISSUE PERFUSION
- CELLULAR HYPOXIA
- If UN-corrected, a FATAL outcome

TYPES of SHOCK

- CARDIOGENIC: (Acute, Chronic Heart Failure)
- HYPOVOLEMIC: (Hemorrhage or Leakage)
- SEPTIC: (“ENDOTOXIC” shock, #1 killer in ICU)
- NEUROGENIC: (loss of vascular tone)
- ANAPHYLACTIC: (IgE mediated systemic vasodilation and increased vascular permeability)

CARDIOGENIC shock

- MI
- VENTRICULAR RUPTURE
- ARRHYTHMIA
- CARDIAC TAMPONADE
- PULMONARY EMBOLISM (acute RIGHT heart failure or “cor pulmonale”)

HYPOVOLEMIC shock

- HEMORRHAGE, Vasc. compartment→H2O
- VOMITING, Vasc. compartment→H2O
- DIARRHEA, Vasc. compartment→H2O
- BURNS, Vasc. compartment→H2O

SEPTIC shock

- OVERWHELMING INFECTION
- “ENDOTOXINS”, i.e., LPS (Usually Gm-),Gm+ and FUNGAL

CLINICAL STAGES of shock

- NON-PROGRESSIVE
  (compensatory mechanisms)
- PROGRESSIVE
  (acidosis, early organ failure)
- IRREVERSIBLE

NON-PROGRESSIVE

- COMPENSATORY MECHANISMS
- CATECHOLAMINES
- VITAL ORGANS PERFUSED

PROGRESSIVE

- HYPOPERFUSION
- EARLY “VITAL” ORGAN FAILURE
- OLIGURIA
- **ACIDOSIS**

IRREVERSIBLE

- HEMODYNAMIC
- CORRECTIONS of no use

CLINICAL PROGRESSION of SYMPTOMS

- Hypotension →
- Tachycardia →
- Tachypnea →
- Warm skin→ Cool skin→ Cyanosis
- Renal insufficiency→
- Obtundance
- Death