Hemodynamic Disorders
Normal fluid homeostasis requires vessel wall integrity as well as maintenance of intravascular pressure and osmolarity within certain physiologic ranges. Increases in vascular volume or pressure, decreases in plasma protein content, or alterations in endothelial function can result in a net outward movement of water across the vascular wall. Such water extra-vasation into interstitial spaces is called edema.
Normal fluid homeostasis also means maintaining blood as a liquid until such time as injury necessitates formation of a clot. Absence of clotting after vascular injury results in hemorrhage.

more extensive hemorrhage can result in hypotension (shock) and death.
inappropriate clotting (*thrombosis*) or migration of clots (*embolism*) can obstruct tissue blood supplies and cause cell death
EDEMA

The term *edema* signifies increased fluid in the interstitial tissue spaces; fluid collections in different body cavities are variously designated *hydrothorax, hydropericardium*, or *hydroperitoneum* (the last is more commonly called *ascites*). *Anasarca* is a severe and generalized edema with profound subcutaneous tissue swelling.
Edema results from any of the following conditions:

1. Increased hydrostatic pressure, caused by a reduction in venous return (as in heart failure).
2. Decreased colloid osmotic pressure, caused by reduced concentration of plasma albumin.
Subcutaneous edema can be diffuse or more prominent in regions with high hydrostatic pressures; the ultimate distribution depends on the underlying etiology.
PULMONARY EDEMA

- is a common clinical problem most frequently seen in the setting of left ventricular failure, but it also occurs in renal failure, acute respiratory distress syndrome, pulmonary infections, and hypersensitivity reactions.
EDEMA OF THE BRAIN

- may be localized to sites of focal injury (e.g., infarct, abscesses or neoplasms) or may be generalized, as in encephalitis, hypertensive crises, or obstruction to the brain's venous outflow. Trauma may result in local or generalized edema, depending on the nature and extent of the injury.
HYPEREMIA AND CONGESTION

The terms *hyperemia* and *congestion* both indicate a local increased volume of blood in a particular tissue. The affected tissue is redder than normal because of engorgement with oxygenated blood.

- Hyperemia is an *active process* resulting from augmented blood flow due to arteriolar dilation.
- Congestion is a *passive process* resulting from impaired venous return out of a tissue. The tissue has a blue-red color (*cyanosis*), especially as worsening congestion leads to accumulation of deoxygenated hemoglobin in the affected tissues.
HEMORRHAGE

- Hemorrhage is extravasation of blood from vessels into the extravascular space.
- Hemorrhage can be external or can be confined within a tissue; any accumulation is referred to as a hematoma.
- Hemorrhages into skin, mucous membranes, or serosal surfaces are called petechiae, associated with locally increased intravascular pressure, low platelet counts, defective platelet function, clotting factor deficiencies.
Slightly larger hemorrhages are called *purpura* and can be associated with many of the same disorders that cause petechiae; in addition, purpura can occur with trauma, vascular inflammation (*vasculitis*), or increased vascular fragility.

Larger subcutaneous hematomas (bruises) are called *ecchymoses*. 
Large accumulations of blood in one or another of the body cavities are called *hemothorax*, *hemopericardium*, *hemoperitoneum*, or *hemarthrosis* (in joints).

Patients with extensive hemorrhages occasionally develop jaundice from the massive breakdown of red blood cells and systemic increases in bilirubin.
Normal hemostasis is a consequence of tightly regulated processes that maintain blood in a fluid, clot-free state in normal vessels while inducing the rapid formation of a localized hemostatic plug at the site of vascular injury. The pathologic form of hemostasis is thrombosis; it involves blood clot (thrombus) formation in uninjured vessels.
THROMBOSIS

There are three primary influences on thrombus formation (called Virchow's triad):

- (1) Endothelial injury,
- (2) Stasis or turbulence of blood flow, and
- (3) Blood hyper-coagulability.
FATE OF THE THROMBUS

- *Propagation.* Thrombi accumulate additional platelets and fibrin, eventually causing vessel obstruction.
- *Embolization.* Thrombi dislodge or fragment and are transported elsewhere in the vasculature.
- *Dissolution.* Thrombi are removed by fibrinolytic activity.
- *Organization and recanalization.* Thrombi induce inflammation and fibrosis (*organization*). These can eventually *recanalize*, or they can be incorporated into a thickened vessel wall.
EMBOLISM

- An embolus is a detached intravascular solid, liquid, or gaseous mass that is carried by the blood to a site distant from its point of origin. Virtually 99% of all emboli represent some part of a dislodged thrombus, hence the term *thromboembolism*.

- Rare forms of emboli include fat droplets, bubbles of air or nitrogen
PULMONARY THROMBOEMBOLISM

- Most pulmonary emboli (60% to 80%) are clinically silent because they are small.
- They eventually become organized and become incorporated into the vascular wall.
- Sudden death, right ventricular failure, or cardiovascular collapse occurs when 60% or more of the pulmonary circulation is obstructed with emboli.
- Embolic obstruction of medium-sized arteries can cause pulmonary hemorrhage.
**Systemic Thromboembolism**

- Systemic thromboembolism refers to emboli in the arterial circulation.
- Very small fraction of systemic emboli appear to arise in veins but end up in the arterial circulation.
In contrast to venous emboli, which tend to lodge primarily in one vascular bed (the lung), arterial emboli can travel to a wide variety of sites; the site of arrest depends on the point of origin of the thromboembolus and the relative blood flow through the downstream tissues. The major sites for arteriolar embolization are the lower extremities (75%) and the brain (10%), with the intestines, kidneys, and spleen affected to a lesser extent.
**Fat Embolism**

- Microscopic fat globules can be found in the circulation after fractures of long bones (which contain fatty marrow).
- *Fat embolism syndrome* is characterized by pulmonary insufficiency, neurologic symptoms, anemia, and thrombocytopenia; it is fatal in about 10% of cases. Typically, the symptoms appear 1 to 3 days after injury, with sudden onset of tachypnea, dyspnea, and tachycardia.
Air Embolism

- Gas bubbles within the circulation can obstruct vascular flow may enter the circulation during obstetric procedures or as a consequence of chest wall injury. Generally, more than 100 mL of air are required to produce a clinical effect.
**Amniotic Fluid Embolism**

- Amniotic fluid embolism is uncommon complication of labor and the immediate postpartum period (1 in 50,000 deliveries). If the patient survives the initial crisis, pulmonary edema typically develops. The underlying cause is entry of amniotic fluid (and its contents) into the maternal circulation via a tear in the placental membranes and rupture of uterine veins. Classically, there is marked pulmonary edema.
The End