Hemodynamic Disorders

Edema: Edema refers to “increased fluid in the interstitial tissue spaces.” Hydrothorax, hydropericardium, or hydroperitoneum (ascites) refer to fluid collection in the respective body cavity. Anasarca is a severe and generalized edema with profound subcutaneous tissue swelling.

Mechanisms of edema: Either increased capillary pressure or diminished colloid osmotic pressure can result in increased interstitial fluid. Excess interstitial edema fluid is removed by lymphatic drainage, ultimately returning to the bloodstream via the thoracic duct; clearly, lymphatic obstruction (e.g., due to scarring or tumor) can also impair fluid drainage and cause edema. Finally, sodium retention (with its obligatory associated water) due to renal disease can also cause edema.

The edema fluid may be either a transudate or exudate. A transudate occurs with volume or pressure overload, or under conditions of reduced plasma protein; it is typically a protein-poor fluid. An exudate occurs with increased vascular permeability, due to inflammation (inflammatory edema); it is a protein-rich fluid.

The principal causes of edema are:

- Increased Hydrostatic Pressure: This is either localized or generalized (systemic):
  - Localized edema: this is due to localized increase in intravascular pressure.
  - Generalized edema: this is due to generalized increases in intravascular pressure, occurs most commonly in congestive heart failure, with involvement of the right ventricular cardiac function.

- Reduced Plasma Osmotic Pressure: Reduced osmotic pressure occurs when there is reduced synthesis or increased loss of albumin from the circulation. Albumin loss is exemplified by the nephrotic syndrome (glomerular capillary walls become leaky) that is associated with generalized edema. Reduced albumin synthesis occurs in the setting of diffuse liver diseases (e.g., cirrhosis) or protein malnutrition.

- Lymphatic Obstruction: Impaired lymphatic drainage and consequent lymphedema is usually localized; it can result from inflammatory or neoplastic obstruction. Cancer of the breast can be treated by resection and/or irradiation of the associated axillary lymph nodes; the resultant scarring and loss of lymphatic drainage can cause severe upper extremity edema. In breast carcinoma infiltration and obstruction of superficial lymphatics can also cause edema of the overlying skin, the so-called peau d’orange (orange peel) appearance. Such a finely pitted surface results from an accentuation of depressions in the skin at the site of hair follicles.

- Sodium and Water Retention: Increased salt-with the obligate accompanying water-causes both increased hydrostatic pressure (due to expansion of the intravascular volume) and reduced vascular osmotic pressure. Salt retention can occur with any impairment of renal function, as in poststreptococcal glomerulonephritis and acute renal failure.

Pathologic features of edema: Edema is most easily recognized grossly. Microscopically, edema fluid is reflected primarily as a clearing and separation of the extracellular matrix elements.
Although any organ or tissue in the body may be involved, edema is most commonly encountered in subcutaneous tissues, lungs, and brain.

**Subcutaneous edema:** This can be diffuse or more prominent in regions with high hydrostatic pressures; the ultimate distribution depends on the underlying etiology. Finger pressure over significantly edematous subcutaneous tissue displaces the interstitial fluid and leaves a finger-shaped depression, so-called **pitting edema.**

**Pulmonary edema:** This is a common clinical problem that is encountered with:
A. Left ventricular failure (most frequent association).
B. Renal failure.
C. Acute respiratory distress syndrome (ARDS).
D. Pulmonary infections.
E. Hypersensitivity reactions.

**Gross features:**  ● The lungs typically heavy.  ● Sectioning reveals frothy, sometimes blood-tinged fluid representing a mixture of air, edema fluid, and extravasated red cells.

**Microscopic features:**  ● The alveolar spaces are filled with pale pink edema fluid.  ● There is congestion of the capillaries within the alveolar walls due to the increase in venous pressure.

**Brain Edema:** This 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.

**Gross features**  ● With generalized edema, the brain is grossly swollen with narrowed sulci and distended gyri showing signs of flattening against the unyielding skull.

**Hyperemia and Congestion:** The terms hyperemia and congestion both indicate a local increased volume of blood in a particular tissue. *Hyperemia* is an active process resulting from augmented blood flow due to arteriolar dilation (e.g., at sites of inflammation or in skeletal muscle during exercise). The affected tissue is redder than normal because of engorgement with oxygenated blood. *Congestion* is a passive process resulting from impaired venous return out of a tissue. It may occur systemically, as in cardiac failure, or it may be local, resulting from an isolated venous obstruction. The tissue has a blue-red color (cyanosis), especially as worsening congestion leads to accumulation of deoxygenated hemoglobin in the affected tissues.

In long-standing congestion, called chronic passive congestion, the stasis of poorly oxygenated blood causes chronic hypoxia, which in turn can result in degeneration or death of parenchymal cells and subsequent tissue fibrosis. Capillary rupture at such sites of chronic congestion can also cause small foci of hemorrhage; phagocytosis and catabolism of the erythrocyte debris can result in accumulations of hemosiderin-laden macrophages.

**Gross features**  ● Cut surfaces of hyperemic or congested tissues are red and wet.

**Microscopic features:**

**Acute pulmonary congestion**  
● This is characterized by engorgement of alveolar capillaries with blood  
● Alveolar septal edema and/or focal minute intra-alveolar hemorrhage may also occur.

**Chronic pulmonary congestion**  
● The septa become thickened and fibrotic  
● The alveolar spaces may contain numerous hemosiderin-laden macrophages ("heart failure cells").

**Chronic passive congestion of the liver**  
● The liver is enlarged & firm  
● Thus each lobule presents a dark brown center (congestion), and a light yellow periphery (fatty degeneration).  
● The central regions of the hepatic lobules are grossly red-brown (congested) and are accentuated against the surrounding zones of uncongested tan, sometimes fatty, liver ("nutmeg liver").
**Microscopic features:** ● There is centrilobular necrosis with hepatocyte drop-out, hemorrhage, and hemosiderin-laden macrophages. ● In long-standing, severe hepatic congestion (most commonly associated with heart failure), hepatic fibrosis ("cardiac cirrhosis") can develop.

**Hemorrhage:** Hemorrhage is the escape of blood from the vasculature into surrounding tissues, a hollow organ or body cavity, or to the outside. Hemorrhage is most often caused by trauma.

**Hematoma:** This localized hemorrhage occurs within a tissue or organ.

**Hemothorax, hemopericardium, hemoperitoneum, and hemarthrosis:** Hemorrhage may occur in the pleural cavity, pericardial sac, peritoneal cavity, or a synovial space, respectively.

**Petechial hemorrhages, petechiae, or purpura:** These small, punctate hemorrhages occur in the skin, mucous membranes, or serosal surfaces.

**Ecchymosis:** This diffuse hemorrhage is usually in skin and subcutaneous tissue.

**Thrombosis:** Normally the blood is kept in a fluid state with rapid formation of a plug at the site of injury. This is normal hemostasis.

**Thrombosis** is the formation of a blood clot inside a blood vessel.

**Both hemostasis and thrombosis involve three components:**
1. Vascular wall.
2. Platelets.
3. Coagulation cascade.

**Pathogenesis:** There are three predisposing factors for thrombus formation (Virchow's triad):
1. **Endothelium injury:** This is a dominant predisposing factor, since endothelial loss by alone can lead to thrombosis. It is particularly important for thrombus formation occurring in the heart or in the arterial circulation, where the normally high flow rates might otherwise interfere with clotting by preventing platelet adhesion & diluting coagulation factors. It is important to note that endothelium need not be denuded or physically disrupted to contribute to the development of thrombosis; any disturbance in the balance of the prothrombotic and antithrombotic activities of endothelium can influence local clotting events. Thus, dysfunctional endothelium may elaborate greater amounts of procoagulant factors or may synthesize reduced amounts of anticoagulant effectors. Significant endothelial dysfunction (in the absence of endothelial cell loss) may occur through
   1. Hypertension.
   2. Turbulent flow over scarred valves.
   3. Bacterial endotoxins.
   5. Hypercholesterolemia.
   6. Radiation.
   7. Products absorbed from cigarette smoke.

2. **Alterations in Normal Blood Flow:** Turbulence contributes to arterial and cardiac thrombosis by causing endothelial injury or dysfunction, as well as by forming countercurrents and local pockets of stasis.

**Stasis** is a major contributor to the development of venous thrombi. Normal blood flow is laminar, such that platelets flow centrally in the vessel lumen, separated from the endothelium by a slower moving clear zone of plasma.

**Stasis and turbulence therefore:**
1. Disrupt laminar flow and bring platelets into contact with the endothelium.
2. Prevent dilution of activated clotting factors by fresh-flowing blood.
3. Retard the inflow of clotting factor inhibitors and permit the buildup of thrombi.
4. Promote endothelial cell activation.

**Turbulence and stasis contribute to thrombosis in several clinical settings:**
● Ulcerated atherosclerotic plaques not only expose subendothelial ECM but also cause turbulence
● Abnormal dilations (aneurysms) of the aorta & other arteries create local stasis and consequently a fertile site for thrombosis.
Acute myocardial infarction results in focally noncontractile myocardium; ventricular remodeling after more remote infarction can lead to ventricular aneurysm formation. In both cases cardiac mural thrombi form more easily because of the local blood stasis.

Mitral valve stenosis (e.g., after rheumatic heart disease) results in left atrial dilation. In conjunction with atrial fibrillation, a dilated atrium is a site of profound stasis and a prime location for development of thrombi.

Hyperviscosity syndromes (such as polycythemia) increase resistance to flow and cause small vessel stasis.

The deformed red cells in sickle cell anemia cause vascular occlusions, with the resultant stasis also predisposing to thrombosis.

3. Hypercoagulability: Hypercoagulability generally contributes less frequently to thrombosis but is, however, an important component in the equation. It is defined as any alteration of the coagulation pathways that predisposes to thrombosis, and it can be divided into primary (genetic) and secondary (acquired) disorders.

Causes of the primary (inherited) hypercoagulable states include most commonly mutations in the factor V gene and the prothrombin gene. Unlike the hereditary disorders, the pathogenesis of acquired thrombotic diatheses is frequently multifactorial & include

- Cardiac failure or trauma: stasis or vascular injury may be most important.
- Oral contraceptive use & pregnancy: probably related to the hyperestrogenic state that is associated with increased hepatic synthesis of coagulation factors and reduced synthesis of antithrombin III.
- Disseminated cancers, release of procoagulant tumor products predisposes to thrombosis.
- Advancing age: is associated with hypercoagulability & this has been attributed to increasing platelet aggregation and reduced endothelial PGI2 release.
- Smoking and obesity promote hypercoagulability by unknown mechanisms.

Pathological features of thrombosis: Thrombi can develop anywhere in the cardiovascular system (e.g., in cardiac chambers, on valves, or in arteries, veins, or capillaries).

The size and shape of a thrombus depend on the site of origin and the cause.

Arterial or cardiac thrombi typically begin at sites of endothelial injury or turbulence; venous thrombi characteristically occur at sites of stasis. Thrombi are focally attached to the underlying vascular surface; arterial thrombi tend to grow in a retrograde direction from the point of attachment, while venous thrombi extend in the direction of blood flow (thus both tend to propagate toward the heart).

The propagating portion of a thrombus tends to be poorly attached and therefore prone to fragmentation, generating an embolus. Thrombi can have grossly (and microscopically) apparent laminations called lines of Zahn; these represent pale platelet and fibrin layers alternating with darker erythrocyte-rich layers. Such lines are significant only in that they represent thrombosis in the setting of flowing blood; their presence can therefore potentially distinguish antemortem thrombosis from the bland nonlaminated clots that occur in the postmortem state.

Thrombi occurring in heart chambers or in the aortic lumen are designated mural thrombi. Abnormal myocardial contraction (resulting from arrhythmias or myocardial infarction) or endomyocardial injury (caused by myocarditis, catheter trauma) promotes cardiac mural thrombi, while ulcerated atherosclerotic plaques and aneurysmal dilation promote aortic thrombosis.

Arterial thrombi are frequently occlusive and are produced by platelet and coagulation activation; they are typically a friable meshwork of platelets, fibrin, erythrocytes, and degenerating leukocytes. Although arterial thrombi are usually superimposed on an atherosclerotic plaque, other vascular injury (vasculitis, trauma) can be involved.

Venous thrombosis (phlebothrombosis) is almost invariably occlusive, and the thrombus can create a long cast of the lumen; venous thrombosis is largely the result of activation of the coagulation cascade, and platelets play a secondary role. Because these thrombi form in the sluggish
venous circulation, they also tend to contain more enmeshed erythrocytes and are therefore called red, or stasis, thrombi. The veins of the lower extremities are most commonly affected (90% of venous thromboses); however, venous thrombi can occur in other parts of the body.

Postmortem clots can sometimes be mistaken at autopsy for venous thrombi. However, postmortem "thrombi" are gelatinous, with a dark red dependent portion where red cells have settled by gravity, and a yellow "chicken fat" supernatant, and they are usually not attached to the underlying wall. In contrast, red thrombi are firmer and are focally attached, and sectioning reveals strands of gray fibrin.

**Postmortem clots**

Thrombi on heart valves are called vegetations. Bacterial or fungal blood-borne infections can cause valve damage, subsequently leading to large thrombotic masses (infective endocarditis).

**Fate of the Thrombus:** In the ensuing days or weeks after the formation of thrombi, they undergo some combination of the following four events:

1. **Propagation:** thrombi accumulate additional platelets and fibrin, eventually causing vessel obstruction.
2. **Embolization:** thrombi dislodge or fragment and are transported elsewhere in the vasculature.
3. **Dissolution** is the result of fibrinolytic activation, which leads to rapid shrinkage and even total lysis of recent thrombi. With older thrombi, extensive fibrin polymerization renders the thrombus substantially more resistant to proteolysis, and lysis is ineffective.
4. **Organization:** Older thrombi become organized by the ingrowth of endothelial cells, smooth muscle cells, and fibroblasts into the fibrin-rich clot.

5. **Recanalization:** Capillary channels are eventually formed, to can create conduits along the length of the thrombus and thereby re-establish the continuity of the original lumen. Occasionally, instead of organizing, the center of a thrombus undergoes enzymatic digestion, presumably because of the release of lysosomal enzymes from trapped leukocytes and platelets.

**Venous Thrombosis (Phlebothrombosis):** Most venous thrombi occur in the superficial or deep veins of the leg.

**Superficial venous thrombi** usually occur in the saphenous system, particularly when there are varicosities. Such superficial thrombi can cause local congestion, swelling, pain, and tenderness along the course of the involved vein, but they rarely embolize. Nevertheless, the local edema and impaired venous drainage do predispose the overlying skin to infections from minor trauma and to the development of varicose ulcers. **Deep thrombi** in the larger leg veins at or above the knee joint (e.g., popliteal, femoral, and iliac veins) are more serious because they may embolize.

**Deep venous thrombosis can complicate**

1. Advanced age, bed rest, and immobilization regardless of the specific clinical setting, increase the risk of deep venous thrombosis because reduced physical activity diminishes the milking action of muscles in the lower leg and so slows venous return.
2. Cardiac failure is an obvious reason for stasis in the venous circulation.
3. Trauma, surgery, and burns usually result in reduced physical activity, injury to vessels, release of procoagulant substances from tissues, and/or reduced t-PA activity.
4. Peripartum and postpartum states; in addition to the potential for amniotic fluid infusion into the circulation during parturition (see below), late pregnancy and the postpartum period are associated with hypercoagulability.
5. Hypercoagulable states.
6. Disseminated cancers: tumor-associated procoagulant release is largely responsible for the increased risk of thromboembolic phenomena.

**Cardiac and Arterial Thrombosis**

**Atherosclerosis** is a major initiator of thromboses, because it is associated with loss of endothelial integrity and abnormal vascular flow.
Myocardial infarction may be complicated by cardiac mural thrombi as a result of dyskinetic myocardial contraction as well as damage to the adjacent endocardium. Rheumatic heart disease can cause atrial mural thrombi due to mitral valve stenosis, followed by left atrial dilation and concurrent atrial fibrillation. Arterial aneurysms (e.g. aortic) are frequently filled by thrombi. In addition to the obstructive consequences, cardiac and aortic mural thrombi can also embolize peripherally. Virtually any tissue can be affected, but brain, kidneys, and spleen are prime targets because of their large volume of blood flow.

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.

Forms of emboli
1. Thromboemboli: representing a dislodged thrombus or part of it. This type virtually constitutes 99% of all emboli.
Rare forms of emboli include:
2. Fat emboli: consisting of fat droplets.
3. Air emboli: consisting of bubbles of air or nitrogen.
4. Atherosclerotic emboli (cholesterol emboli): consisting of atheromatous debris
5. Tumor emboli: made up of fragments of a tumor.
7. Foreign body emboli: as bullets or shrapnel.

Inevitably, emboli lodge in vessels too small to permit further passage, resulting in partial or complete vascular occlusion. The consequences of thromboembolism include ischemic necrosis (infarction) of downstream tissue. Depending on the site of origin, emboli may lodge anywhere in the vascular tree.

Pulmonary Thromboembolism
In more than 95% of cases, venous emboli originate from deep leg vein thrombi above the level of the knee such as the popliteal, femoral, or iliac veins. These emboli are carried through progressively larger channels and pass through the right side of the heart before entering the pulmonary arterial circulation. Depending on its size, the embolus may settle within
1. The main pulmonary trunk.
2. Across the bifurcation (saddle embolus).
3. The main pulmonary arteries.
4. The medium sized pulmonary arteries.
5. Pass out into the smaller branching arteries or arterioles.

Frequently, there are multiple emboli, perhaps sequentially, or as a shower of smaller emboli from a single large thrombus. Rarely, an embolus can pass through an interatrial or interventricular defect, thereby entering the systemic circulation (paradoxical or crossing embolism).

Systemic Thromboembolism: This refers to emboli in the arterial circulation. Sources include:
1. Intracardiac mural thrombi (80%) that complicate:
   A. Infarction of the left ventricular wall (70%).
   B. Dilated left atria (e.g., secondary to mitral valve disease) (25%).
   The remainder (5%) originates from thrombi complicating:
2. Aortic aneurysms.
3. Ulcerated atherosclerotic plaques.
4. Valvular vegetations.
5. A very small fraction of systemic emboli appear to arise in veins but end up in the arterial circulation, through interventricular defects. These are called paradoxical emboli.
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:**

1. The lower extremities (75%).
2. The brain (10%).
3. The intestines (mesenteric), kidneys, and spleen.
4. The upper limbs are the least common sites.

**Fat & bone marrow embolism:** Microscopic fat globules can be found in the circulation after fractures of long bones (which contain fatty marrow) or after soft-tissue trauma. Fat enters the circulation by rupture of the marrow vascular sinusoids or rupture of venules in injured tissues. Although fat and marrow embolism occurs in some 90% of individuals with severe skeletal injuries, only about 10% of such patients show any clinical findings. The pathogenesis of fat embolism syndrome probably involves both mechanical obstruction and biochemical injury. Fat globules within the pulmonary circulation squeeze through into the systemic circulation to reach the brain & kidney.

**Air Embolism:** Gas bubbles within the circulation can obstruct vascular flow and cause ischemic injury. Air 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; bubbles can coalesce to form frothy masses sufficiently large to occlude major vessels.

**Amniotic Fluid Embolism:** The underlying cause is entry of amniotic fluid into the maternal circulation via a tear in the placental membranes with the fluid gaining access into ruptured uterine veins. Classically, there is marked pulmonary edema and diffuse alveolar damage.

**Infarction:** This is defined as “localized area of ischemic cell necrosis in a living organ or tissue, resulting most often from sudden reduction or cessation of its arterial blood supply or occasionally its venous drainage”

**Causes of vascular obstruction:**

1. Nearly 99% of all infarcts result from thrombotic or embolic events, and almost all result from arterial occlusion.
2. Uncommon causes include:
   1. Expansion of atheromatous plaques by intraplaque hemorrhage.
   2. Spasm of coronary arteries.
   3. Pressure on a vessel from outside: ● Tumor. ● Fibrous adhesions. ● Narrow hernial sac.
   4. Twisting (torsion) of the pedicle of mobile organ e.g. loop of small intestine (volvulus), ovary and testis.
3. External pressure and torsion (causes 4 and 5) usually interfere with venous drainage, since veins are more readily compressed than arteries.

**Gross features:** Infarcts are classified on the basis of their color (reflecting the amount of hemorrhage) into red (hemorrhagic) or white (pale, anemic). They are also classified according to the presence or absence of microbial infection into septic or bland.

- **Red infarcts** occur:
  1. With venous occlusions (such as in ovarian torsion)
  2. in loose tissues (such as lung) that allow blood to collect in the infarcted zone
  3. in tissues with dual circulations such as lung and small intestine, permitting flow of blood from an unobstructed parallel supply into a necrotic area (such perfusion not being sufficient to rescue the ischemic tissues)
  4. in tissues that were previously congested because of sluggish venous outflow
  5. When flow is re-established to a site of previous arterial occlusion and necrosis (e.g., fragmentation of an occlusive embolus or angioplasty of a thrombotic lesion).
● **White infarcts** occur with arterial occlusions or in solid organs (such as heart, spleen, and kidney), where the solidity of the tissue limits the amount of hemorrhage that can seep into the area of ischemic necrosis from adjoining capillary beds.

● All infarcts tend to be wedge shaped, with the occluded vessel at the apex and the periphery of the organ forming the base; when the base is a serosal surface there can be an overlying fibrinous exudate.

● At the outset, all infarcts are poorly defined and slightly hemorrhagic. The margins of both types of infarcts tend to become better defined with time by a narrow rim of congestion attributable to inflammation at the edge of the lesion (line of demarcation).

● In solid organs, the relatively few extravasated red cells are lysed, with the released hemoglobin remaining in the form of hemosiderin. Thus, infarcts resulting from arterial occlusions typically become progressively paler and sharply defined with time. In spongy organs, by comparison, the hemorrhage is too extensive to permit the lesion ever to become pale. Over the course of a few days, however, it does become firmer and browner, reflecting the accumulation of hemosiderin pigment.

● Infarcts within the brain could be pale or hemorrhagic. An embolus may break into smaller emboli that are pushed more distally; this causes reopening of the already closed major artery that pours blood into the soft area of infarction. Thus, an extensive hemorrhage occurs into what had been initially a pale infarction.

**Microscopic features:** The dominant histologic characteristic of infarction is **ischemic coagulative necrosis**. The brain is an exception in that ischemic tissue injury in the central nervous system results in liquefactive necrosis.

An inflammatory response begins to develop along the margins of infarcts within a few hours and is usually well defined within 1 to 2 days. Eventually the inflammatory response is followed by a reparative response beginning in the preserved margins.

In stable or labile tissues, parenchymal regeneration can occur at the periphery, where underlying stromal architecture is spared. However, most infarcts are ultimately replaced by scar. This, depending on the size of the infarct, may take several months.

**Septic infarctions** occur when bacterial vegetations from a heart valve embolize, when microbes seed an area of necrotic tissue, or when infarction occurs in an already infected area. In these cases the infarct is converted into an abscess, with a correspondingly greater inflammatory response.

**Factors That Influence Development of an Infarct:**

Vascular occlusion can have no or minimal effect, or can cause death of a tissue or even the individual. The major determinants of the eventual outcome include

1. **Nature of the Vascular Supply:** The most important factor that determines whether occlusion of a vessel will cause damage is the presence or absence of an alternative blood supply. For example, lungs have a dual pulmonary and bronchial artery blood supply; thus, obstruction of small pulmonary artery or arterioles does not cause infarction in an otherwise healthy individual with an intact bronchial circulation.

2. **Rate of Development of Occlusion:** Slowly developing occlusions are less likely to cause infarction because they provide time for the development of alternative perfusion pathways i.e. collateral vessels.

3. **Vulnerability to Hypoxia:** The susceptibility of a tissue to hypoxia influences the likelihood of infarction. Neurons undergo irreversible damage when deprived of their blood supply for only 3 to 4 minutes. Myocardial cells, though more resistant to hypoxic damage than neurons, are also quite sensitive and die after only 20 to 30 minutes of ischemia. In contrast, skeletal muscles & fibroblasts within a limb may remain viable after many hours of ischemia.

4. **Oxygen Content of Blood:** The partial pressure of oxygen in blood also determines the outcome of vascular occlusion. Partial flow obstruction of a small vessel in an anemic or cyanotic patient might lead to tissue infarction, whereas it would be without effect under conditions of normal oxygen tension.