- Epithelial cells, tissue macrophages and dendritic cells, leukocytes, and other cell types express receptors that sense the presence of microbes and damage. Circulating proteins recognize microbes that have entered the blood.
- The outcome of acute inflammation is either elimination of the noxious stimulus followed by decline of the reaction and repair of the damaged tissue, or persistent injury resulting in chronic inflammation.

Acute Inflammation

Acute inflammation has three major components: (1) dilation of small vessels leading to an increase in blood flow, (2) increased permeability of the microvasculature enabling plasma proteins and leukocytes to leave the circulation, and (3) emigration of the leukocytes from the microcirculation, their accumulation in the focus of injury, and their activation to eliminate the offending agent (Fig. 3-1). When an individual encounters an injurious agent, such as an infectious microbe or dead cells, phagocytes that reside in all tissues try to eliminate these agents. At the same time, phagocytes and other sentinel cells in the tissues recognize the presence of the foreign or abnormal substance and react by liberating cytokines, lipid messengers, and other mediators of inflammation. Some of these mediators act on small blood vessels in the vicinity and promote the efflux of plasma and the recruitment of circulating leukocytes to the site where the offending agent is located.

Reactions of Blood Vessels in Acute Inflammation

The vascular reactions of acute inflammation consist of changes in the flow of blood and the permeability of vessels, both designed to maximize the movement of plasma proteins and leukocytes out of the circulation and into the site of infection or injury. The escape of fluid, proteins, and blood cells from the vascular system into the interstitial tissue or body cavities is known as *exudation* (Fig. 3-2). An *exudate* is an extravascular fluid that has a high protein concentration and contains cellular debris. Its presence implies that there is an increase in the permeability of small blood vessels triggered by some sort of tissue injury and an ongoing inflammatory reaction. In contrast, a *transudate* is a fluid with low protein content (most of which is albumin), little or no cellular material, and low specific gravity. It is essentially an ultrafiltrate of blood plasma that is produced as a result of osmotic or hydrostatic imbalance across the vessel wall without an increase in vascular permeability (Chapter 4). *Edema* denotes an excess of fluid in the interstitial tissue or serous cavities; it can be either an exudate or a transudate. *Pus*, a *purulent* exudate, is an inflammatory exudate rich in leukocytes (mostly neutrophils), the debris of dead cells and, in many cases, microbes.

Changes in Vascular Flow and Caliber

Changes in vascular flow and caliber begin early after injury and consist of the following.

- **Vasodilation is induced by the action of several mediators, notably histamine, on vascular smooth muscle.**

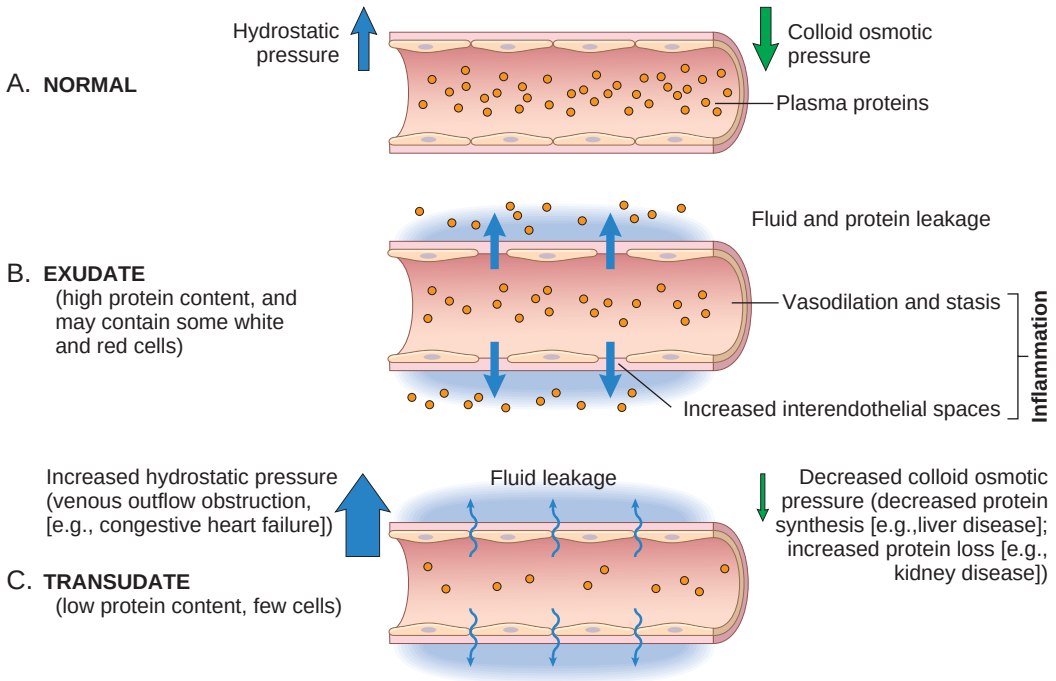

Figure 3-2 Formation of exudates and transudates. **A,** Normal hydrostatic pressure *(blue arrow)* is about 32 mm Hg at the arterial end of a capillary bed and 12 mm Hg at the venous end; the mean colloid osmotic pressure of tissues is approximately 25 mm Hg *(green arrow)*, which is equal to the mean capillary pressure. Therefore, the net flow of fluid across the vascular bed is almost nil. **B,** An exudate is formed in inflammation, because vascular permeability increases as a result of increased interendothelial spaces. **C,** A transudate is formed when fluid leaks out because of increased hydrostatic pressure or decreased osmotic pressure.