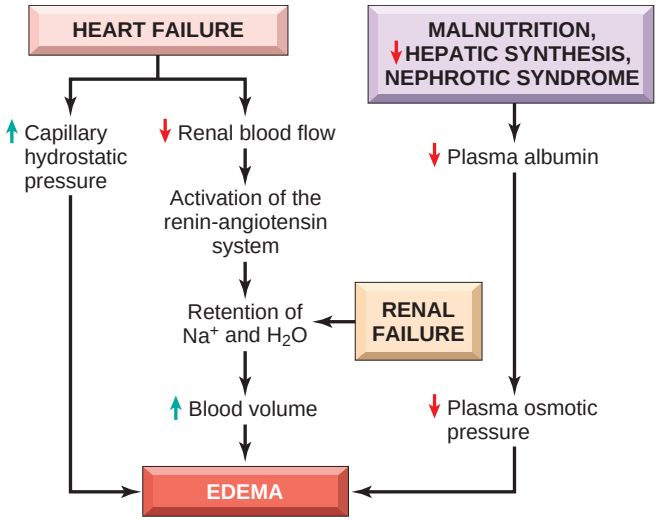

Figure 4-2 Mechanisms of systemic edema in heart failure, renal failure, malnutrition, hepatic failure, and nephrotic syndrome.

MORPHOLOGY

Edema is easily recognized grossly; microscopically, it is appreciated as clearing and separation of the extracellular matrix and subtle cell swelling. Any organ or tissue can be involved, but edema is most commonly seen in subcutaneous tissues, the lungs, and the brain. **Subcutaneous edema** can be diffuse or more conspicuous in regions with high hydrostatic pressures. Its distribution is often influenced by gravity (e.g., it appears in the legs when standing and the sacrum when recumbent), a feature termed **dependent edema**. Finger pressure over markedly edematous subcutaneous tissue displaces the interstitial fluid and leaves a depression, a sign called **pitting edema**.

Edema resulting from **renal dysfunction** often appears initially in parts of the body containing loose connective tissue, such as the eyelids; **periorbital edema** is thus a characteristic finding in severe renal disease. With **pulmonary edema,** the lungs are often two to three times their normal weight, and sectioning yields frothy, blood-tinged fluid—a mixture of air, edema, and extravasated red cells. **Brain edema** can be localized or generalized depending on the nature and extent of the pathologic process or injury. The swollen brain exhibits narrowed sulci and distended gyri, which are compressed by the unyielding skull (Chapter 28).

Effusions involving the pleural cavity (**hydrothorax**), the pericardial cavity (**hydropericardium**), or the peritoneal cavity (**hydroperitoneum** or **ascites**) are common in a wide range of clinical settings. Transudative effusions are typically protein-poor, translucent and straw colored; an exception are peritoneal effusions caused by lymphatic blockage (chylous effusion), which may be milky due to the presence of lipids absorbed from the gut. In contrast, exudative effusions are protein-rich and often cloudy due to the presence of white cells.

Clinical Features

The consequences of edema range from merely annoying to rapidly fatal. *Subcutaneous edema* is important primarily because it signals potential underlying cardiac or renal disease; however, when significant, it can also impair wound healing or the clearance of infections. *Pulmonary edema* is a common clinical problem that is most frequently seen in the setting of left ventricular failure; it can also occur with renal failure, acute respiratory distress syndrome (Chapter 15), and pulmonary inflammation or infection. Not only does fluid collect in the alveolar septa around capillaries and impede oxygen diffusion, but edema fluid in the alveolar spaces also creates a favorable environment for bacterial infection. *Pulmonary effusions* often accompany edema in the lungs and can further compromise gas exchange by compressing the underlying pulmonary parenchyma. *Peritoneal effusions* (*ascites*) resulting most commonly from portal hypertension are prone to seeding by bacteria, leading to serious and sometimes fatal infections. *Brain edema* is life threatening; if severe, brain substance can *herniate* (extrude) through the foramen magnum, or the brain stem vascular supply can be compressed. Either condition can injure the medullary centers and cause death (Chapter 28).

KEY CONCEPTS

Edema

Edema is the result of the movement of fluid from the vasculature into the interstitial spaces; the fluid may be protein-poor (*transudate*) or protein-rich (*exudate*).

Edema may be caused by:

- Increased hydrostatic pressure (e.g., heart failure)
- Decreased colloid osmotic pressure caused by reduced plasma albumin, either due to decreased synthesis (e.g., liver disease, protein malnutrition) or to increased loss (e.g., nephrotic syndrome)
- Increased vascular permeability (e.g., inflammation), which is usually localized but may occur throughout the body in severe systemic inflammatory states such as sepsis
- Lymphatic obstruction (e.g., infection or neoplasia)
- Sodium and water retention (e.g., renal failure)

Hyperemia and Congestion

Hyperemia and congestion both stem from increased blood volumes within tissues, but have different underlying mechanisms and consequences. *Hyperemia* is an active process in which arteriolar dilation (e.g., at sites of inflammation or in skeletal muscle during exercise) leads to increased blood flow. Affected tissues turn red (*erythema*) because of increased delivery of oxygenated blood. *Congestion* is a passive process resulting from reduced outflow of blood from a tissue. It can be systemic, as in cardiac failure, or localized, as in isolated venous obstruction.

As a result of increased hydrostatic pressures, congestion commonly leads to edema. In long-standing *chronic passive congestion,* the associated chronic hypoxia may result in ischemic tissue injury and scarring. In chronically congested tissues, capillary rupture can also produce small hemorrhagic foci; subsequent catabolism of extravasated red cells can leave residual telltale clusters of hemosiderin-laden macrophages.