Increased Hydrostatic Pressure

Increases in hydrostatic pressure are mainly caused by disorders that impair venous return. If the impairment is localized (e.g., a deep venous thrombosis [DVT] in a lower extremity), then the resulting edema is confined to the affected part. Conditions leading to systemic increases in venous pressure (e.g., *congestive heart failure*, Chapter 12) are understandably associated with more widespread edema.

Reduced Plasma Osmotic Pressure

Under normal circumstances albumin accounts for almost half of the total plasma protein; it follows that conditions leading to inadequate synthesis or increased loss of albumin from the circulation are common causes of reduced plasma oncotic pressure. Reduced albumin synthesis occurs mainly in severe liver diseases (e.g., end-stage cirrhosis, Chapter 18) and protein malnutrition (Chapter 9). An important cause of albumin loss is the *nephrotic syndrome* (Chapter 20), in which albumin leaks into the urine through abnormally permeable glomerular capillaries. Regardless of cause, reduced plasma osmotic pressure leads in a stepwise fashion to edema, reduced intravascular volume, renal hypoperfusion, and secondary hyperaldosteronism. Not only does the ensuing salt and water retention by the kidney fail to correct the plasma volume deficit, but it also exacerbates the edema, because the primary defect—a low plasma protein level—persists.

Table 4-1 Pathophysiologic Categories of Edema

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
Neurohumoral dysregulation
Reduced Plasma Osmotic Pressure (Hypoproteinemia)
Protein-losing glomerulopathies (nephrotic syndrome)
Liver cirrhosis (ascites)
Malnutrition
Protein-losing gastroenteropathy
Lymphatic Obstruction
Inflammatory
Neoplastic
Postsurgical
Postirradiation
Sodium Retention
Excessive salt intake with renal insufficiency
Increased tubular reabsorption of sodium
Renal hypoperfusion
Increased renin-angiotensin-aldosterone secretion
Inflammation
Acute inflammation
Chronic inflammation
Angiogenesis

Modified from Leaf A, Cotran RS. Renal pathophysiology, 3rd ed. New York, Oxford University Press, 1985, p 146.

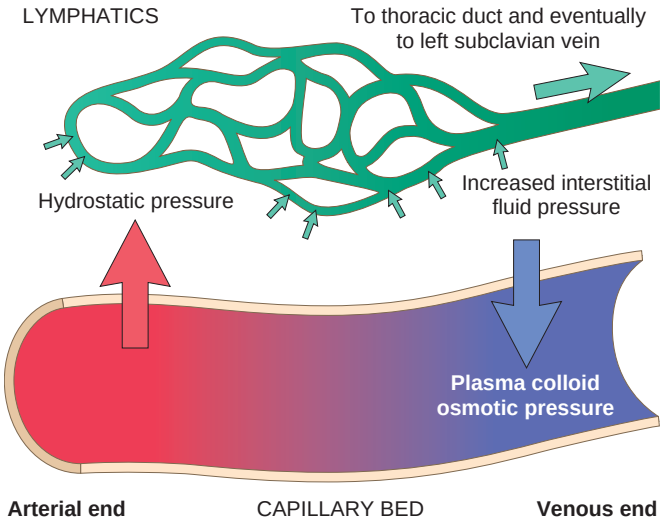

Figure 4-1 Factors influencing fluid movement across capillary walls. Normally, hydrostatic and osmotic forces are nearly balanced so that there is little net movement of fluid out of vessels. Many different pathologic disorders (Table 4-1) are associated with increases in capillary hydrostatic pressure or decreases in plasma osmotic pressure that lead to the extravasation of fluid into tissues. Lymphatic vessels remove much of the excess fluid, but if the capacity for lymphatic drainage is exceeded, tissue edema results.

Sodium and Water Retention

Increased salt retention—with obligate retention of associated water—causes both increased hydrostatic pressure (due to intravascular fluid volume expansion) and diminished vascular colloid osmotic pressure (due to dilution). Salt retention occurs whenever renal function is compromised, such as in primary kidney disorders and in cardiovascular disorders that decrease renal perfusion. One of the most important causes of renal hypoperfusion is congestive heart failure, which (like hypoproteinemia) results in the activation of the renin-angiotensin-aldosterone axis. In early heart failure, this response is beneficial, as the retention of sodium and water and other adaptations, including increased vascular tone and elevated levels of antidiuretic hormone, improve cardiac output and restore normal renal perfusion. However, as heart failure worsens and cardiac output diminishes, the retained fluid merely increases the hydrostatic pressure, leading to edema and effusions.

Lymphatic Obstruction

Trauma, fibrosis, invasive tumors, and infectious agents can all disrupt lymphatic vessels and impair the clearance of interstitial fluid, resulting in *lymphedema* in the affected part of the body. A dramatic example is seen in parasitic *filariasis*, in which the organism induces obstructive fibrosis of lymphatic channels and lymph nodes. This may result in edema of the external genitalia and lower limbs that is so massive as to earn the appellation *elephantiasis*. Severe edema of the upper extremity may also complicate surgical removal and/or irradiation of the breast and associated axillary lymph nodes in patients with breast cancer.