The glomerular membrane, normally impermeable to albumin and other proteins, becomes permeable to proteins, especially albumin, that leak through the membrane and are lost in urine (**hyperalbuminuria**). This reduces the serum albumin level (**hypoalbuminemia**), decreasing the colloidal osmotic pressure in the capillaries. As a result, the vascular hydrostatic pressure exceeds the pull of the colloidal osmotic pressure, causing fluid to accumulate in the interstitial spaces (**edema**) and body cavities, particularly in the abdominal cavity (**ascites**). The shift of fluid from the plasma to the interstitial spaces reduces the vascular fluid volume (**hypovolemia**), which in turn stimulates the renin–angiotensin system and the secretion of antidiuretic hormone and aldosterone. Tubular reabsorption of sodium and water is increased in an attempt to increase intravascular volume. The elevation of serum lipids is not fully understood. The sequence of events in nephrotic syndrome is diagrammed in Fig. 26-6.