

Fig. 19.1. Pleural fluid formation and removal. Forces of Starling's law move protein-poor fluid from capillaries to the parietal and pulmonary interstitial spaces. The parietal capillary beds and parietal pleura are relatively impermeable to proteins, so the fluid that diffuses from the interstitium to the pleural space has a lower [TP] (≈ 1.0 g/dL). Most pleural fluid is drained by the parietal lymphatic vessels, with small amounts entering the pulmonary lymphatic vessels via stomata. The drainage is powered by the lymphatic pump that creates a negative pressure to pull fluid from the interstitium. Evidence indicates the visceral pleural barrier is impermeable to H_2O and solutes in most mammals.

Fig. 19.2. Schematic drawing of the five major pathogenesises of pleural and peritoneal effusions.

1. Transudates form when there is increased vascular hydraulic pressure with or without decreased plasma oncotic pressure. The transudate formed from increased hydraulic pressures in hepatic sinuses and alveolar capillaries (lungs not shown) have relatively higher protein concentrations because the vessels are more permeable to plasma proteins.
2. Exudates form when increased vascular and mesothelial permeability enables protein-rich fluid to escape from the capillaries to the interstitium and then to the cavity.
3. Damage to blood vessels enables blood to escape to create a hemorrhagic effusion.
4. Effusions develop when there is decreased drainage of the fluids by the lymphatic vessels either because of increased pressure within the lymphatic vessel or because cells (e.g., neoplastic) are blocking pathways. Damage to lymphatic vessels enables lymph to escape to create a lymphocyte-rich effusion. If the lymph contains chylomicrons, then a chylous effusion forms.
5. Damage to viscera enables contents of those structures to enter the body cavity (e.g., a uroperitoneum). The contents released from damaged alimentary, biliary, or urinary tissues will initiate an inflammatory reaction and thus exudation.