Fig. 4.1. Blood platelet concentrations are established mostly by the relative rates of platelet production, consumption, and destruction, and by the shifting of platelets to and from the circulation. In pathologic states, combinations of these factors often contribute to abnormal platelet concentrations.

- Production: Most megakaryopoiesis and thrombopoiesis occur in the bone marrow, and new platelets enter systemic circulation via marrow sinusoids. Some megakaryocytes appear to become lodged in the lungs after release from the bone marrow. These megakaryocytes release platelets into the blood, but the overall importance of this process is not known. Platelets produced by splenic hematopoietic tissue may contribute to the circulating platelet mass in health and disease. Hematopoietic foci may also arise in the liver and contribute to the circulating platelet mass.
- Consumption: Platelets may be removed from circulation during normal maintenance of vascular integrity or during accelerated consumptive states (e. g., thrombotic disease and vasculitis).
- Destruction: Macrophages (Mϕ), primarily in the spleen and liver, may destroy platelets that carry surface-associated antibodies or complement. Aged or damaged platelets may be similarly destroyed.
- Redistribution: Splenic platelet sequestration (reversible) may reduce the circulating platelet mass, and splenic contraction may increase it. Pulmonary sequestration of platelets has been associated with severe hypothermia and endotoxemia.
- Massive transfusion with blood products or other fluids may cause a dilutional decrease in platelet concentration.