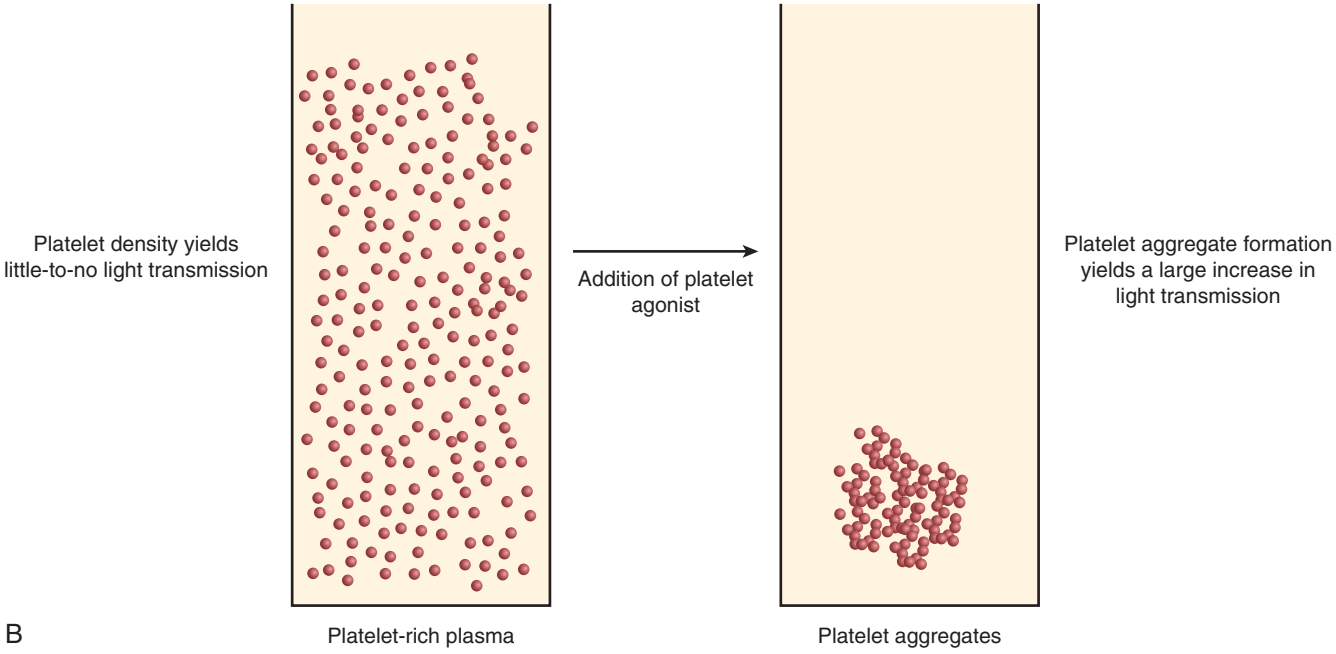

FIGURE 51-5 Methodology underlying light transmission aggregometry. **A,** Typical laboratory light transmission aggregometer. **B,** Platelet function is directly proportional to light transmission in this assay. Platelet-rich plasma, which prevents light transmission, is exposed to various agonists (i.e., adenosine diphosphate, epinephrine, collagen, arachidonic acid, and ristocetin). As platelets begin to aggregate or agglutinate, light transmission increases over time and is typically reflected as a primary or secondary wave of aggregation for most agonists. Low or no increase in light transmission typically correlates with diminished platelet function.

irreversible inhibitors, but this action should allow newly produced platelets to be free of drugs effects and to function appropriately at the site of an injury.

Beyond stopping the offending drug, bleeding caused by aspirin or other antiplatelet agents may be addressed by infusion of 1-deamino-(8-D-arginine)-vasopressin (DDAVP). This agent has been effective in decreasing the bleeding time of platelet anticoagulated patients. Occasionally, platelet transfusion is appropriate. In most cases, a single platelet transfusion of 4 to 6 random donor units (or one apheresis unit) contributes enough normal platelets (>10% of total circulating number) to restore primary hemostasis. Platelet dysfunction and bleeding caused by other drugs is similarly treated by discontinuing the drug and providing platelet transfusions when needed (Table 51-6).

Uremic Platelet Dysfunction

Renal insufficiency can be associated with the accumulation of toxic proteins such as guanidinosuccinic acid, which induces high levels of nitric oxide formation by vascular endothelial cells to inhibit platelet function. The uremic state can also suppress platelet secretory pathways and platelet adhesion to exposed endothelium through mechanisms that are not well understood.