also common findings. Red cell fragmentation is often detected but at a lower degree than in acute DIC.

Differential Diagnosis Distinguishing between DIC and severe liver disease is challenging and requires serial measurements of the laboratory parameters of DIC. Patients with severe liver disease manifest laboratory features including thrombocytopenia due to platelet sequestration, portal hypertension, or hypersplenism; decreased synthesis of coagulation factors and natural anticoagulants; and elevated levels of D-dimer. However, in contrast to DIC, these laboratory parameters in liver disease do not change rapidly.

Although microangiopathic disorders such as acquired thrombotic thrombocytopenic purpura present with acute onset accompanied by thrombocytopenia, red cell fragmentation, and multiorgan failure, the clinical presentation and laboratory findings such as an inhibitor to ADAMTS13 levels assist in making the microangiopathic disorder diagnosis **(Chap. 115)**.

TREATMENT

Disseminated Intravascular Coagulation

The morbidity and mortality associated with DIC are primarily related to the underlying disease. Management of the underlying disease is required to control and eliminate DIC; however, support with platelets and coagulation factors may be needed until the inciting cause is under control. Many patients with overt DIC are critically ill, usually requiring management in the intensive care unit to treat shock physiology and other manifestations of the underlying illness.

MANAGEMENT OF HEMORRHAGIC SYMPTOMS

Patients with active bleeding or at high risk of bleeding during invasive procedures or after chemotherapy require transfusion support; however, transfusion solely to correct mildly to moderately abnormal coagulation parameters is not indicated. Platelet transfusion for platelet counts <10,000–20,000/μL and replacement