

# 1 QUESTION

## *HOW MUCH FLUID SHOULD BE GIVEN DURING THE INITIAL MANAGEMENT OF ACUTE PANCREATITIS?*

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Regardless of etiology, the pathogenesis of acute pancreatitis results in the extravasation of liters of intravascular fluid into the peritoneum. These losses manifest in the development of pancreatic ascites, hypotension, tachycardia, and further destruction of the pancreas, also referred to as pancreatic necrosis. Impairment of the microcirculation of the pancreas appears to lead to pancreatic necrosis. A vicious cycle develops whereby pancreatic inflammation leads to extravasation of protein-rich intravascular fluid into the peritoneum. The intravascular hypovolemia that accompanies acute pancreatitis subsequently leads to a decrease in pancreatic blood flow. Pancreatic ischemia leads to the activation of inflammatory mediators. The decreased blood flow also causes stasis and the development of thrombi leading to subsequent necrosis which then exacerbates the inflammatory process. The association of hemoconcentration, where the hematocrit rises, with pancreatic necrosis illustrates this process (Figure 1-1).

Vigorous intravenous hydration leads to hemodilution and relief of hemoconcentration. This translates into direct benefits for the patient with acute pancreatitis. A decreased hematocrit is associated with mild disease. Also, a falling hematocrit during the first 24 hours of care leads to a decrease in morbidity. Clinical studies with aggressive plasma volume expansion using intravenous dextran to promote hemodilution have suggested efficacy in preventing severe disease. Although dextran is not used clinically at the present time, isotonic saline is our practical alternative. It appears that vigorous intravenous hydration early in the course of acute pancreatitis can prevent the development of necrosis.

Your goal in managing patients with acute pancreatitis is to decrease the hematocrit, hemodilution, blood urea nitrogen, and creatinine and promote renal blood flow. By preventing intravascular depletion of fluid and promoting pancreatic blood flow, pancreatic perfusion is maintained. By maintaining pancreatic perfusion, pancreatic necrosis and the complications of pancreatitis leading to severe disease are prevented (Figure 1-2).