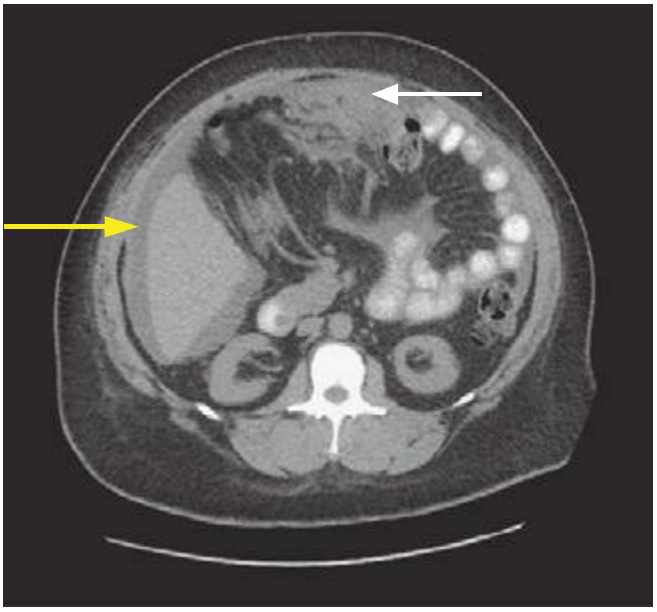

FIGURE 59-2 CT of a patient with peritoneal carcinomatosis (white arrow) and ascites (yellow arrow).

carcinoma or melanoma (Fig. 59-2). The tumor cells lining the peritoneum produce a protein-rich fluid that contributes to the development of ascites. Fluid from the extracellular space is drawn into the peritoneum, further contributing to the development of ascites. Tuberculous peritonitis causes ascites via a similar mechanism; tubercles deposited on the peritoneum exude a proteinaceous fluid. Pancreatic ascites results from leakage of pancreatic enzymes into the peritoneum.

CAUSES

Cirrhosis accounts for 84% of cases of ascites. Cardiac ascites, peritoneal carcinomatosis, and "mixed" ascites resulting from cirrhosis and a second disease account for 10–15% of cases. Less common causes of ascites include massive hepatic metastasis, infection (tuberculosis, *Chlamydia* infection), pancreatitis, and renal disease (nephrotic syndrome). Rare causes of ascites include hypothyroidism and familial Mediterranean fever.

EVALUATION

Once the presence of ascites has been confirmed, the etiology of the ascites is best determined by *paracentesis*, a bedside procedure in which a needle or small catheter is passed transcutaneously to extract ascitic fluid from the peritoneum. The lower quadrants are the most frequent sites for paracentesis. The left lower quadrant is preferred because of the greater depth of ascites and the thinner abdominal wall. Paracentesis is a safe procedure even in patients with coagulopathy; complications, including abdominal wall hematomas, hypotension, hepatorenal syndrome, and infection, are infrequent.

Once ascitic fluid has been extracted, its gross appearance should be examined. Turbid fluid can result from the presence of infection or tumor cells. White, milky fluid indicates a triglyceride level >200 mg/dL (and often >1000 mg/dL), which is the hallmark of *chylous ascites*. Chylous ascites results from lymphatic disruption that may occur with trauma, cirrhosis, tumor, tuberculosis, or certain congenital abnormalities. Dark brown fluid can reflect a high bilirubin concentration and indicates biliary tract perforation. Black fluid may indicate the presence of pancreatic necrosis or metastatic melanoma.

The ascitic fluid should be sent for measurement of albumin and total protein levels, cell and differential counts, and, if infection is suspected, Gram's stain and culture, with inoculation into blood culture bottles at the patient's bedside to maximize the yield. A serum albumin level should be measured simultaneously to permit calculation of the *serum-ascites albumin gradient* (SAAG).

The SAAG is useful for distinguishing ascites caused by portal hypertension from nonportal hypertensive ascites (Fig. 59-3). The SAAG reflects the pressure within the hepatic sinusoids and correlates with the hepatic venous pressure gradient. The SAAG is calculated by subtracting the ascitic albumin concentration from the serum albumin level and does not change with diuresis. A SAAG ≥1.1 g/dL reflects the presence of portal hypertension and indicates that the ascites is due to increased pressure in the hepatic sinusoids. According to Starling's law, a high SAAG reflects the oncotic pressure that counterbalances the portal pressure. Possible causes include cirrhosis, cardiac ascites, hepatic vein thrombosis (Budd-Chiari syndrome), sinusoidal obstruction syndrome (veno-occlusive disease), or massive liver metastases. A SAAG <1.1 g/dL indicates that the ascites is not related to portal hypertension, as in tuberculous peritonitis, peritoneal carcinomatosis, or pancreatic ascites.

For high-SAAG (≥1.1) ascites, the ascitic protein level can provide further clues to the etiology (Fig. 59-3). An ascitic protein level of ≥2.5 g/dL indicates that the hepatic sinusoids are normal and are allowing passage of protein into the ascites, as occurs in cardiac ascites, early Budd-Chiari syndrome, or sinusoidal obstruction syndrome. An ascitic protein level <2.5 g/dL indicates that the hepatic sinusoids have been damaged and scarred and no longer allow passage of protein, as occurs with cirrhosis, late Budd-Chiari syndrome, or massive liver metastases. Pro-brain-type natriuretic peptide (BNP) is a natriuretic hormone released by the heart as a result of increased volume and ventricular wall stretch. High levels of BNP in serum occur in heart failure and may be useful in identifying heart failure as the cause of high-SAAG ascites.

Further tests are indicated only in specific clinical circumstances. When secondary peritonitis resulting from a perforated hollow viscus is suspected, ascitic glucose and lactate dehydrogenase (LDH) levels can

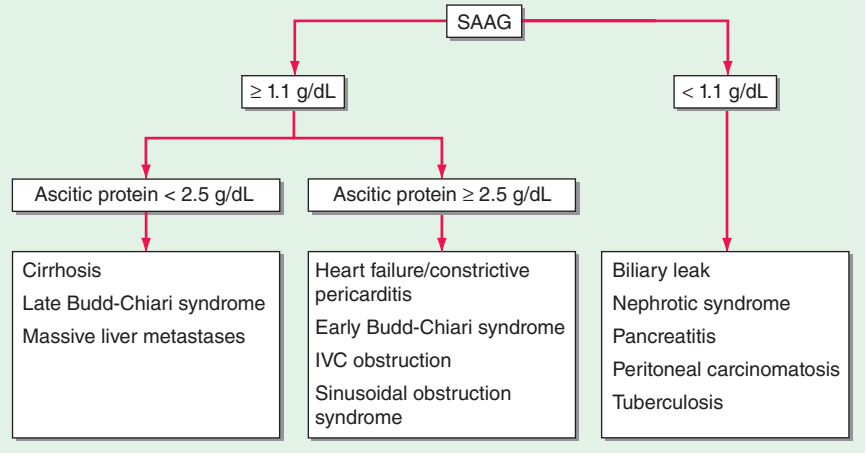

FIGURE 59-3 Algorithm for the diagnosis of ascites according to the serum-ascites albumin gradient (SAAG). IVC, inferior vena cava.