confirmed, aggressive fluid resuscitation is initiated, intravenous analgesics are administered, severity is assessed, and a search for etiologies that may impact acute care is begun. Patients who do not respond to aggressive fluid resuscitation in the emergency ward should be considered for admission to a step-down or intensive care unit for aggressive fluid resuscitation, hemodynamic monitoring, and management of necrosis or organ failure.

**Fluid Resuscitation and Monitoring Response to Therapy** The most important treatment intervention for acute pancreatitis is safe, aggressive intravenous fluid resuscitation. The patient is made NPO to rest the pancreas and is given intravenous narcotic analgesics to control abdominal pain and supplemental oxygen (2 L) via nasal cannula.

Intravenous fluids of lactated Ringer's or normal saline are initially bolused at 15–20 cc/kg (1050–1400 mL), followed by 3 mg/kg per hour (200–250 mL/h), to maintain urine output >0.5 cc/kg per hour. Serial bedside evaluations are required every 6–8 h to assess vital signs, oxygen saturation, and change in physical examination. Lactated Ringer's solution has been shown to decrease systemic inflammation and may be a better crystalloid than normal saline. A *targeted resuscitation strategy* with measurement of hematocrit and BUN every 8–12 h is recommended to ensure adequacy of fluid resuscitation and monitor response to therapy, noting less aggressive resuscitation strategy may be needed in milder forms of pancreatitis. A rising BUN during hospitalization is not only associated with inadequate hydration but also higher in-hospital mortality.

A decrease in hematocrit and BUN during the first 12–24 h is strong evidence that sufficient fluids are being administered. Serial measurements and bedside assessment for fluid overload are continued, and fluid rates are maintained at the current rate. Adjustments in fluid resuscitation may be required in patients with cardiac, pulmonary, or renal disease. A rise in hematocrit or BUN during serial measurement should be treated with a repeat volume challenge with a 2-L crystalloid bolus followed by increasing the fluid rate by 1.5 mg/kg per hour. If the BUN or hematocrit fails to respond (i.e., remains elevated or does not decrease) to this bolus challenge and increase in fluid rate, consideration of transfer to an intensive care unit is strongly recommended for hemodynamic monitoring.

**Assessment of Severity and Hospital Triage** Severity of acute pancreatitis should be determined in the emergency ward to assist in patient triage to a regular hospital ward or step-down unit or direct admission to an intensive care unit. The Bedside Index of Severity in Acute Pancreatitis (BISAP) incorporates five clinical and laboratory parameters obtained within the first 24 h of hospitalization (Table 371-3)—BUN >25 mg/dL, impaired mental status (Glasgow coma score <15), SIRS, age >60 years, and pleural effusion on radiography—that can be useful in assessing severity. Presence of three or more of these factors was associated with substantially increased risk for in-hospital mortality among patients with acute pancreatitis. In addition, an elevated hematocrit >44% and admission BUN >22 mg/dL are also associated with more severe acute pancreatitis. Incorporating these indices with the overall patient response to initial fluid resuscitation in the emergency ward can be useful at triaging patients to the appropriate hospital acute care setting.

In general, patients with lower BISAP scores, hematocrits, and admission BUNs tend to respond to initial management and are triaged to a regular hospital ward for ongoing care. If SIRS is not present at 24 h, the patient is unlikely to develop organ failure or necrosis. Therefore, patients with persistent SIRS at 24 h or underlying comorbid illnesses (e.g., chronic obstructive pulmonary disease, congestive heart failure) should be considered for a step-down unit setting if available. Patients with higher BISAP scores and elevations in hematocrit and admission BUN that do not respond to initial fluid resuscitation and exhibit evidence of respiratory failure, hypotension, or organ failure should be considered for direct admission to an intensive care unit.

**Special Considerations Based on Etiology** A careful history, review of medications, selected laboratory studies (liver profile, serum triglycerides, serum calcium), and an abdominal ultrasound are recommended in the emergency ward to assess for etiologies that may impact acute management. An abdominal ultrasound is the initial imaging modality of choice and will evaluate the gallbladder and common duct and assess the pancreatic head.

***GALLSTONE PANCREATITIS*** Patients with evidence of ascending cholangitis (rising white blood cell count, increasing liver enzymes) should undergo ERCP within 24–48 h of admission. Patients with gallstone pancreatitis are at increased risk of recurrence, and consideration should be given to performing a cholecystectomy during the same admission or within 4–6 weeks of discharge. An alternative for patients who are not surgical candidates would be to perform an endoscopic biliary sphincterotomy before discharge.

***HYPERTRIGLYCERIDEMIA*** Serum triglycerides >1000 mg/dL are associated with acute pancreatitis. Initial therapy may include insulin, heparin, or plasmapheresis. Outpatient therapies include control of diabetes if present, administration of lipid-lowering agents, weight loss, and avoidance of drugs that elevate lipid levels.

Other potential etiologies that may impact acute hospital care include *hypercalcemia*, *autoimmune pancreatitis*, *post-ERCP pancreatitis*, and *drug-induced pancreatitis*. Treatment of hyperparathyroidism or malignancy is effective at reducing serum calcium. Autoimmune pancreatitis is responsive to glucocorticoid administration. Pancreatic duct stenting and rectal indomethacin administration are effective at decreasing pancreatitis after ERCP. Drugs that cause pancreatitis should be discontinued. Multiple drugs have been implicated, but only about 30 have been challenged (Class 1A) and found to be causative.

**Nutritional Therapy** A low-fat solid diet can be administered to subjects with mild acute pancreatitis after the abdominal pain has resolved. Enteral nutrition should be considered 2–3 days after admission in subjects with more severe pancreatitis instead of total parenteral nutrition (TPN). Enteral feeding maintains gut barrier integrity, limits bacterial translocation, is less expensive, and has fewer complications than TPN. The choice of gastric versus nasojejunal enteral feeding is currently under investigation.

**Management of Local Complications (Table 371-4)** Patients exhibiting signs of clinical deterioration despite aggressive fluid resuscitation and hemodynamic monitoring should be assessed for local complications, which may include necrosis, pseudocyst formation, pancreas duct disruption, peripancreatic vascular complications, and extrapancreatic infections. A multidisciplinary team approach is recommended including gastroenterology, surgery, interventional radiology, and intensive care specialists, and consideration should also be made for transfer to a pancreas center.

***NECROSIS*** The management of necrosis requires a multidisciplinary team approach. Percutaneous aspiration of necrosis with Gram stain and culture should be performed if there are ongoing signs of possible pancreatic infection such as sustained leukocytosis, fever, or organ failure. There is currently no role for *prophylactic antibiotics* in necrotizing pancreatitis. It is reasonable to start broad-spectrum antibiotics in a patient who appears septic while awaiting the results of Gram stain and cultures. If cultures are negative, the antibiotics should be discontinued to minimize the risk of developing opportunistic or fungal superinfection. Repeated fine-needle aspiration and Gram stain with culture of pancreatic necrosis may be done every 5–7 days in the presence of persistent fever. Repeated CT or MRI imaging should also be considered with any change in clinical course to monitor for complications (e.g., thromboses, hemorrhage, abdominal compartment syndrome).

In general, *sterile necrosis* is most often managed conservatively unless complications arise. Once a diagnosis of *infected necrosis* is established and an organism identified, targeted antibiotics should be instituted. Pancreatic debridement (necrosectomy) should be considered for definitive management of *infected necrosis*, but clinical decisions are generally influenced by response to antibiotic treatment and overall clinical condition. Symptomatic local complications as outlined in the revised Atlanta criteria may require definitive therapy.