6

Gastrointestinal Disorders

■ I. GASTROINTESTINAL BLEEDING

A. Classification
   1. Upper gastrointestinal (GI) bleeding is above the ligament of Treitz.
   2. Lower GI bleeding is below the ligament of Treitz.

B. Etiology. The most common causes of acute GI bleeding requiring admission to the intensive care unit (ICU) are depicted in Table 6.1. The most common sources of GI bleeding in the ICU are gastroduodenal stress ulcerations.

C. Diagnostic Evaluation
   1. History
      Although the history and physical assessment in a critically ill patient with acute GI bleeding may be limited by the patient's clinical condition, the following are points that need to be investigated:
      (a) History of hematemesis or melena
      (b) Time of onset
      (c) Amount of blood
      (d) Color and character
      (e) Drug or alcohol use (i.e., nonsteroidal anti-inflammatory drug [NSAID], prednisone, warfarin)
      (f) Past medical history (i.e., cirrhosis, peptic ulcer disease [PUD], inflammatory bowel disease [IBD], etc.)
   2. Physical Examination
      The precise cause of acute GI bleeding is unlikely to be evident from physical examination alone (except in chronic liver disease, Osler–Rendu–Weber syndrome, or hemorrhoids).
      (a) General Appearance: This may vary from the patient in no acute distress to the patient in hypovolemic shock.
      (b) Vital Signs: Tachycardia and postural hypotension. An increase in heart rate of 10–20 beats per minute and drop in blood pressure of >20 mmHg upon assumption of an upright position are generally indicative of significant, acute volume loss.

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Table 6.1. Etiologies of acute GI bleeding

Upper	*Lower*
Esophagus	Small intestine
Mucosal tear	Arteriovenous malformations
Esophageal rupture	Inflammatory bowel disease ischemia
Esophagitis	Meckel's diverticulum
Neoplasms	Neoplasms
Varices	
Stomach	Large intestine
Arteriovenous malformations	Diverticulosis
Gastritis (any etiology)	Hemorrhoids
Neoplasms	Inflammatory bowel disease infections
Peptic ulcer disease	Ischemia
Stress ulcers	Neoplasms
Duodenum	
Arteriovenous malformations	
Neoplasms (*rare*)	
Peptic ulcer disease	

Table 6.2. Bleeding advantages and disadvantages of NG tubes in acute GI

Advantages	*Disadvantages*
1. Document the presence or absence of blood	1. Patient discomfort
2. Monitor rate of bleeding	2. Irritation of esophageal and/or gastric mucosa
3. To lavage and decompress the stomach	3. Increased incidence of sinusitis 4. Possible esophageal or gastric perforation

(c) Other Signs of Hypovolemia: Altered mental status and low urine output.
(d) Associated Findings: Petechiae, jaundice, hepatomegaly, and splenomegaly.
(e) Rectum: Look for hemorrhoids, fissures, etc. Examine stools for blood even if the patient has an upper GI source.

3. A nasogastric (NG) tube should be placed in *all* patients with acute GI bleeding. The major advantages and disadvantages of NG tubes are shown in Table 6.2.

4. Laboratory Evaluation
   All patients admitted to the ICU with GI bleeding should undergo the laboratory tests depicted in Table 6.3.
5. Radiologic Evaluation
   All patients should undergo chest radiograph and abdominal X-rays. These may show evidence of perforation or obstruction and may indicate ischemic changes.
   Contrast studies have a low diagnostic yield and may be hazardous for the critically ill patient. They may also interfere with other diagnostic studies (i.e., endoscopy, angiography). Special tests may be required in the evaluation of acute GI bleeding. These include:
   (a) Selective angiography may be used as a diagnostic as well as therapeutic tool (e.g., embolization). A bleeding rate ≥0.5 mL/min at the time of the procedure is needed for diagnosis.
   (b) Radionuclide scans are sensitive in detecting lesions with lower bleeding rates.
6. Endoscopy is indicated in the vast majority of patients requiring ICU admission for GI bleeding.
   (a) Upper endoscopy is indicated when blood is obtained from the NG tube or when frank hematemesis is present.
   (b) Flexible sigmoidoscopy should be performed initially if lower GI bleeding is suspected. If this is not diagnostic, colonoscopy should be considered.
   (c) Special endoscopic procedures may be required (i.e., wireless video capsule endoscopy, push enteroscopy, double balloon enteroscopy).

D. Initial ICU Management
1. As in any critically ill patient, the management of acute GI bleeding starts with assessment of the airway, breathing, and circulation (ABCs). A low threshold for endotracheal intubation is recommended in the event of clouding of consciousness or overt shock, to prevent aspiration.
2. Insert at least two large-bore (16-gauge) IV catheters.
3. Infuse blood, plasma expanders, and/or normal saline to maintain a mean arterial pressure ≥65 mmHg.
4. Some authors still recommend NG placement in all patients with GI bleeding and lavage of the stomach until the return is clear. This practice is considered useful only in settings where emergency endoscopy is not available.
5. Correction of preexisting coagulopathy (i.e., fresh-frozen plasma [FFP], vitamin K, etc.).
6. Proton pump inhibitors and H_2-receptor blockers to prevent further hemorrhage. Continuous infusions are preferred (i.e., esomeprazole [Nexium™] 20–40 mg/24 h, pantoprazole [Protonix™] 8 mg/h, ranitidine [Zantac™] 150–300-mg/24 h IV infusion if the renal function is normal or famotidine [Pepcid™] 20 mg IV q12 h).
7. Endoscopic and/or angiographic verification of the source of bleeding will allow more definitive therapy (i.e., thermal coagulation, injection therapy, fibrin sealant, endoclips, surgery).

E. Specific Management of Selected Conditions
   1. Variceal Hemorrhage
      (a) Vasopressin Infusion: Start at 0.2–0.4 U/min (up to 1 U/min). Some of the major complications of vasopressin (i.e., myocardial ischemia) can be prevented by the coadministration of nitroglycerin.
      (b) Alternatively, somatostatin analogs can be utilized. Somatostatin inhibits the release of vasodilator hormones, such as glucagon, indirectly causing splanchnic vasoconstriction and decreased portal inflow:
         1. Octreotide is a long-acting analog of somatostatin.
         2. Dosing: 25–50-mcg IV bolus followed by continuous IV infusion of 25–50 mcg/h.
      (c) Sclerotherapy: Indicated at the time of diagnostic endoscopy. Two or three treatments are usually done within a 10-day period.
      (d) Balloon Tamponade: Temporizing measure only. It is usually reserved for hemorrhage that fails to stop after therapy with vasopressin and sclerotherapy. The routine use of Sengstaken–Blakemore or Minnesota tubes has almost disappeared with the advances in endoscopic therapy.
      (e) Surgical Therapy: Every patient with a major esophageal bleed should receive surgical consultation in case an emergent intervention is needed. Indications for surgical therapy include the following:
         1. Child's class A or B patient in whom vital signs cannot be stabilized medically.
         2. Continuous bleeding for ≥48 h despite sclerotherapy and balloon tamponade.
         3. Third acute episode of esophageal bleeding in spite of previous sclerotherapy.
   2. Hemorrhage from Ulcers and Erosive Lesions
      (a) Endoscopy therapy with sclerosing agents, or laser coagulation, or heater probe.
      (b) Surgical intervention is indicated in cases of:
         1. Visible vascular pedicle on endoscopy
         2. Transfusion of 6 U blood in 24 h
         3. Arterial spurting

Table 6.3. Initial laboratory evaluation in GI bleeding

Complete blood count (H/H should be repeated every 4 h until patient is stable or bleeding has been controlled)
BUN, creatinine, and electrolytes
PT, PTT
Type and crossmatch for 2–8 U of PRBCs, FFP
Other tests are ordered according to suspected or known underlying disease (i.e., LFTs, CK, etc.)

BUN blood urea nitrogen, *CK* creatine kinase, *FFP* fresh-frozen plasma, *H/H* hemoglobin/hematocrit, *LFT* liver function tests, *PRBCs* packed red blood cells, *PT* prothrombin time, *PTT* partial thromboplastin time

3. Active Lower GI Bleeding
   (a) If a lesion is reachable with sigmoidoscopy or colonoscopy, local therapy may be attempted (e.g., laser coagulation).
   (b) Arterial embolization is indicated if the above fails.
   (c) All patients with active lower GI bleeding should receive surgical consultation in case an emergent intervention is needed.

■ II. ACUTE MESENTERIC ISCHEMIA

A. Definition. Acute mesenteric ischemia (AMI) is an acute reduction in blood flow to the intestine leading to inadequate perfusion. AMI may be a reflection of generalized poor perfusion, or it may result from local pathology.

B. Epidemiology. The incidence of AMI has increased over the past few decades. The rising incidence may be attributable to advances in medical technology and to new therapies extending the life of critically ill patients who are prone to develop AMI (e.g., elderly). The mortality in AMI is between 55% and 100%.

C. Etiology
   1. Occlusive
      (a) Atherosclerotic narrowing of the mesenteric bed
      (b) Systemic emboli from any source (e.g., endocarditis)
      (c) Vasculitis
      (d) Hypercoagulable states
   2. Nonocclusive
      (a) Splanchnic Vasoconstriction
         1. Hypovolemia
         2. Hypotension
         3. Low cardiac output
         4. Vasopressor agent use

D. Risk Factors for AMI. The most common predisposing conditions are depicted in Table 6.4.

E. Diagnostic Evaluation
   1. History and Physical Examination
      The classic complaint of severe abdominal pain that is out of proportion to the findings of physical examination, in our experience, is rarely seen. If peritoneal

Table 6.4. Risk factors for the development of AMI

Age ≥50 years
Atherosclerotic heart disease
Congestive heart failure
Recent myocardial infarction
Valvular heart disease

signs are present (e.g., rebound tenderness), intestinal infarction is likely to have occurred. Abdominal distention, emesis, and other signs of intestinal obstruction may occur in patients with AMI in situ. Lower GI bleeding may occur.

2. Laboratory Studies
   (a) Leukocytosis in 75% of patients
   (b) Metabolic acidosis
   (c) Elevated amylase, creatine kinase (CK) (6–12 h after infarction has occurred), lactate, and phosphate
3. Radiologic Evaluation. Should be done as soon as the patient has been adequately resuscitated (including measures aimed at relieving acute congestive heart failure and hypotension, correction of hypovolemia, and cardiac dysrhythmias)
   (a) Plain Abdominal X-Rays
      1. Useful in excluding other causes of abdominal pain (i.e., mechanical obstruction, perforation)
      2. Seventy percent of patients will show at least one of the following:
         (a) Ileus
         (b) Ascites
         (c) Small bowel dilation
         (d) Separation of small bowel loops
         (e) Thickening of *valvulae conniventes*
         (f) Thumb printing
   (b) Barium studies are *contraindicated* in these patients and they interfere with arteriography.
   (c) Computed tomography (CT) may be particularly valuable when mesenteric vein thrombosis is being considered. It may show focal or segmental bowel wall thickening or intestinal pneumatosis.
   (d) Arteriography: For adequate study, the patient needs to be hemodynamically stable. Early use of this test is the key to diagnosis. It provides a "road map" for the surgeon.
   (e) Laparotomy.

F. Therapy
1. As in any critically ill patient, the management of AMI starts with assessment of the ABCs.
2. Adequate hydration. If necessary, provide invasive hemodynamic monitoring to maximize cardiac output, oxygen delivery, and volume status.
3. Patients with suspected embolic or thrombotic occlusion should undergo *urgent* laparotomy for possible resection. Heparin and broad-spectrum antibiotic are indicated before surgery. Most patients will undergo a "second-look" operation within 24 h of the initial laparotomy.
4. In those cases with nonocclusive AMI, intra-arterial infusions of vasodilators (e.g., papaverine 30–60 mg/h) are advocated by some.

■ III. FULMINANT HEPATIC FAILURE AND ENCEPHALOPATHY

A. Definition
   1. Acute Fulminant Hepatic Failure
      Acute fulminant hepatic failure (FHF) is defined as acute liver failure associated with the development of hepatic encephalopathy within 8 weeks of the onset of symptoms attributable to hepatocellular dysfunction. This definition assumes that there is no preexisting liver disease.
   2. Hepatic Encephalopathy
      Hepatic encephalopathy (HE) is a complex neuropsychiatric syndrome precipitated by abnormal liver function. This syndrome is a feature of acute and/or chronic hepatocellular failure.

B. Etiology
   Common causes of FHF and HE are depicted in Table 6.5.

C. Diagnostic Evaluation
   1. History
      A detailed history should be obtained from family members. The following points need to be investigated:
      (a) History of preexisting liver disease
      (b) Drug or alcohol use
      (c) Toxin exposure or ingestion
   2. Physical Examination
      This may vary from the patient in no distress to the patient in overt shock.
      (a) Vital signs: Tachycardia, hypotension.
      (b) Associated findings: Petechiae, jaundice, hepatomegaly, splenomegaly.
      (c) The encephalopathy may begin with confusion, disorientation, and irrational behavior. Coma may develop rapidly. (See Table 6.6.)
   3. Laboratory and Radiologic Evaluation
      All patients with HE and/or FHF should undergo the following tests:
      (a) Chest X-ray, abdominal X-rays.
      (b) Blood glucose may reveal hypoglycemia.

Table 6.5. Causes of acute liver failure

1. Viral hepatitis (i.e., A, B, C)
2. Drugs or toxins Acetaminophen Acute alcohol intoxication Carbon tetrachloride Halothane Isoniazid Monoamine oxidase inhibitors Mushroom poisoning
3. Fatty liver of pregnancy
4. Shock of any etiology
5. Massive liver infiltration (i.e., leukemia)
6.Decompensation of chronic liver failure

Table 6.6. Clinical stages of hepatic encephalopathy

Stage	*Neurological findings*
I	Confusion, mild changes in personality, psychometric defects
II	Drowsiness to lethargy
III	Somnolent but arousable
IV	Coma

(c) Serum bilirubin: A value >23 mg/dL is the best predictor of nonsurvival.
(d) AST and ALT have little prognostic value as levels tend to fall as the patient's condition worsens.
(e) Serum albumin: Its decrease reflects poor outcome.
(f) Serum electrolytes.
(g) Complete blood count.
(h) Head computed tomography (CT) scan to rule out a structural lesion (e.g., hemorrhage).
(i) Lumbar puncture needs to be considered and performed if meningitis is suspected.
(j) If the etiology of FHF is unknown, the following need to be ordered:
    1. Acetaminophen level
    2. Hepatitis profile
        (a) Viral hepatitis A is diagnosed by detection of HAV-IgM in the patient's serum.
        (b) Viral hepatitis B is diagnosed by:
            (i) Detection of HBsAg
            (ii) Anti-HB_c IgM
        (c) Viral hepatitis C is diagnosed by detection of anti-HCV.
        (d) Delta virus hepatitis is diagnosed by detection of anti-HDV in a patient coinfected with hepatitis B virus.
    3. Alkaline phosphatase
    4. Amylase
(k) Serum ammonia level.
(l) Electroencephalograms (EEGs) are used to assess clinical response and prognosis in patients with HE.

D. Complications of FHF
When the liver fails acutely, all organ systems are involved to some extent.
1. Central Nervous System (CNS)
Hepatic encephalopathy, cerebral edema
2. Cardiovascular
Dysrhythmias (particularly in patients with advanced FHF), hypotension
3. Pulmonary
Hypoxemia advancing to adult (acute) respiratory distress syndrome (ARDS)

4. Renal
   The development of renal failure with FHF carries a poor prognosis.
   (a) In most instances, the renal failure is related to "prerenal" causes.
   (b) The *hepatorenal syndrome* is a diagnosis of exclusion. It is associated with a normal urine sediment, a urinary sodium concentration of <20 mmol/L, and resolution if liver function improves.
5. Hematologic
   Thrombocytopenia, diminished clotting factors with episodes of severe bleeding
6. Infection
   Susceptibility to infection is increased in patients with FHF.

7. Metabolic
   Hypoglycemia, metabolic acidosis, hypokalemia, hyponatremia

E. Management
1. Supportive Therapy
   (a) As in any critically ill patient, the management of AMI starts with assessment of the ABCs.
   (b) The usual indications for endotracheal intubation and assisted mechanical ventilation apply to these patients.
2. The use of corticosteroids for patients with FHF has not been proven to improve survival and, indeed, may worsen the clinical picture.
3. Some authors suggest avoiding parenteral nutrition, as protein and amino acids may worsen the clinical picture. However, new total parenteral nutrition solutions with "branched-chain" amino acids are probably efficacious and help maintain a positive nitrogen balance.
4. The management of FHF-associated cerebral edema is no different from that for non-hepatic-related causes (see Chap. 9, "Neurologic Disorders"). In recent years, emphasis on the use of therapeutic hypothermia for these patients seems encouraging.
5. Some clinical and experimental evidence shows that the benzodiazepine antagonist flumazenil (Romazicon) may have some role in improving the signs and symptoms of HE.
6. Investigational data have shown some improvement in the hemodynamics of patients with FHF treated with n-acetylcysteine.
7. Liver transplantation may be an alternative form of therapy (in a few specialized transplant centers) for some patients with no known contraindication to the procedure.
8. Liver "dialysis": A few specialized centers are currently exploring this form of therapy.
9. Agents aimed at stimulating ammonia metabolism have also been tried (e.g., ornithine-aspartate, sodium benzoate).

■ IV. PANCREATITIS

A. Definition. Acute pancreatitis is an inflammatory process of the pancreas with a wide range of clinical severity ranging from self-limited to a lethal disease, complicated by multiple organ system failure (10% of cases).

B. Etiology. The most common causes of pancreatitis are:
   1. Alcoholism
   2. Gallstones
   3. Hyperlipidemia
   4. Trauma (blunt or penetrating)
   5. Infections (i.e., mumps, mycoplasma)
   6. Hypoperfusion states (i.e., shock, cardiopulmonary bypass)
   7. Hypercalcemia
   8. Drugs (i.e., sulfonamides, thiazides)

C. Diagnostic Evaluation
   1. History
      Ninety-five percent of patients with acute pancreatitis present with abdominal pain, of which 50% will present with upper abdominal discomfort radiating to the back. Nausea and vomiting are also present.
   2. Physical Examination
      Depending on the severity of the situation, the patient may have overt signs of shock or may be hemodynamically stable. Other findings include the following:
      (a) Abdominal tenderness and distention
      (b) Abdominal ileus
      (c) Low-grade fever (Note: A fever >39 °C should suggest cholangitis, peritonitis, or a pancreatic abscess.)
      (d) Mild jaundice
      (e) Ascites
      (f) Pleural effusion
   3. Laboratory Evaluation
      (a) Complete Blood Count (CBC): Shows marked leukocytosis. Thrombocytopenia may be present in those cases complicated by disseminated intravascular coagulation (DIC).
      (b) Amylase: Elevated initially, but may decrease after 2–3 days if necrosis of the pancreas is widespread. False-positive results may occur in perforation of the esophagus, stomach, intestine, gynecologic disorders, renal failure, severe burns, diabetic ketoacidosis (DKA), salivary gland disorders, and macroamylasemia.
      (c) Lipase: Hyperlipasemia persists longer than hyperamylasemia. However, if necrosis of the pancreas is widespread, these values may be normal.
      (d) Serum calcium is usually low. When levels are <8 mg/dL, the prognosis is poor.
      (e) Other electrolyte imbalances as well as hyperglycemia are usually present.
      (f) Metabolic acidosis may be present.

Table 6.7. Grades of acute pancreatitis by points (Balthazar Score)

Grade of acute pancreas points (Balthazar Score)	*Points*
A = Normal pancreas	0
B = Pancreatic enlargement alone	1
C = Inflammation confined to the pancreas and peripancreatic fat	2
D = One pancreatic fluid collection	3
E = Two or more peripancreatic fluid collections	4

(g) C-reactive protein: Usually elevated.
(h) Urinalysis may reveal proteinuria, casts (25% of the cases), and glycosuria.

4. Radiologic Evaluation
Every patient with suspected acute pancreatitis should get a chest X-ray (to rule out free air under the diaphragm, evidence of pleural effusions, etc.) and an abdominal X-ray (signs of intestinal obstruction, ileus, gallstones, the so-called sentinel loop of pancreatitis, or the colon "cutoff" sign, etc.). In addition, when the diagnosis remains in doubt, especially in the more severely ill, the following can be obtained:
(a) Ultrasonography (US) is the modality of choice in patients with edematous pancreatitis or suspected biliary pancreatitis and to follow up phlegmon or abscesses. Unfortunately, US cannot be accurately performed in obese patients and in those with moderate-to-severe ileus.
(b) CT is the most useful tool in assessing the retroperitoneum. Its use in acute pancreatitis is mainly to follow up on significant complications (i.e., abscess, phlegmon, pseudoaneurysms).
Balthazar CT scoring system was the first and is still in use. This scoring system includes five grades: grade A (normal), grade B (pancreas enlargement), grade C (inflammation of the pancreas and surrounding tissue), grade D (single peripancreatic fluid accumulation), and grade E (two or more peripancreatic fluid accumulation and/or air accumulation). Grade D and E have a mortality of 14% and morbidity of 54% (Table 6.7).
Therefore, contrast CT and necrosis classification, found by Balthazar, were used simultaneously (CT Severity Index): grade 1 (<30%), grade 2 (30–50%), and grade 3 (>50% necrosis) (Table 6.8).

D. Management
1. As in any critically ill patient, the management of acute pancreatitis starts with assessment of the ABCs.
2. Adequate hydration.
3. Correct underlying factors.
4. Control pancreatic enzyme secretion.
(a) Nasogastric suction
(b) Proton pump inhibitors (e.g., pantoprazole 40 mg IV q12 h) or H_2-receptor blocking agents (e.g., ranitidine [Zantac] 300-mg/24 h IV infusion if renal function is normal or famotidine 20 mg IV q12 h).

Table 6.8. Degree of pancreatic necrosis

Degree of pancreatic necrosis	*Points*
No necrosis	0
Necrosis of one-third of pancreas (30 %)	2
Necrosis of one-half of pancreas (50 %)	4
Necrosis of more than one-half of pancreas (>50 %)	6

Table 6.9. Complications of acute pancreatitis

1. Intravascular fluid depletion
   (a) Prerenal azotemia
   (b) Shock
2. ARDS (3–7 days after the onset)
3. Cardiac dysfunction
4. Pancreatic abscess
5. Pancreatic pseudocysts
6. Chronic pancreatitis
7. Permanent diabetes mellitus
8. Multiorgan system failure

(c) Many clinicians use the following agents in acute pancreatitis; however, clinical studies have not supported the routine use of these agents:
   1. Calcitonin (300 IU/24 h)
   2. Somatostatin (250-µg IV bolus, then 250 µg/h as IV drip)
   3. Glucagon

(d) Of interest is the use of intramuscular (IM) clonidine (not yet available in the United States) for patients hemodynamically stable with acute pancreatitis. Preliminary data show encouraging results.

5. Sedation and analgesia: Patients may require substantial amounts of analgesia, usually with meperidine (Demerol).
6. Adequate parenteral nutrition (see Chap. 10, “Nutrition”).
7. Correct hypocalcemia *only* if there is clinical evidence of tetany.

E. Complications. The most common complications of acute pancreatitis are depicted in Table 6.9.
   1. Those patients who demonstrate fever >39 °C with a white blood cell count >20,000/mm^3 should be evaluated for the presence of a pancreatic abscess (with the use of CT). If there are any fluid collections, CT-guided fine-needle aspiration is then indicated (for Gram’s stain and cultures).

2. If the suspected diagnosis is pancreatic abscess, broad-spectrum antibiotics should be started and an emergent surgical consultation obtained.
3. Some authors advocate necrosectomy in patients with necrotizing pancreatitis.

F. Prognosis
1. In assessing the severity of the disease and prognosis, several classifications have been used. The most commonly utilized is the *Ranson's criteria* (initially developed for patients with alcoholic pancreatitis):
   (a) Three or more of the following criteria must be met:
      1. Age >55 years
      2. White blood cell count >16,000/mm^3
      3. Glucose >200 mg/dL
      4. Base deficit >4 mEq/L
      5. Lactic dehydrogenase (LDH) >350 IU/L
      6. AST (serum glutamate pyruvate transaminase [SGPT]) >250 IU/L
   (b) Development of the following within 48 h indicates a worsening prognosis:
      1. Hematocrit drop >10%
      2. Serum urea nitrogen (BUN) rise >5 mg/dL
      3. Partial pressure of O_2 in arterial blood (PaO_2) <60 Torr (mmHg)
      4. Calcium <8 mg/dL
      5. Fluid sequestration >6 L
   (c) Mortality rates correlate with the number of criteria present:
      1. 0–2 criteria, 1% mortality
      2. 3–4 criteria, 16% mortality
      3. 5–6 criteria, 40% mortality
      4. 7–8 criteria, 100% mortality
2. Intensive care management and prompt surgical consultation have lowered the mortality of acute pancreatitis.

■ V. USEFUL FACTS AND FORMULAS

A. *Intestinal Transit*. The normal 24-h *intestinal fluid and electrolyte transport* are depicted in Table 6.10.

B. *Stool Formulas*. As part of the diagnostic workup of patients with diarrhea, *stool osmolal gap* (SOG) is usually calculated utilizing the following formula:

$$\text{SOG} = \text{stool osmolality} - 2 \times \left(\text{stool } Na^+ + \text{stool } K^+\right)$$

Normal stool osmolality is <290 mOsm/L. If the SOG >100, it indicates an osmotic diarrhea.

C. *Liver Facts*. The *Child's classification* for portal hypertension is commonly used in critically ill patients and is depicted in Table 6.11.

Table 6.10. Normal 24-h intestinal fluid and electrolyte transport

Site	*Fluid received (L)*	*Amount absorbed (L)*	*Electrolyte absorption*		
			Na^+	*K^+*	*Cl^-*
Duodenum	9.0	4.0	Passive	Passive	Passive
Jejunum					
Ileum	5.0	3.5	Active	Passive	Passive
Colon	1.5	1.35	Active	Passive	Active

Table 6.11. Child's classification of portal hypertension

Class	*A*	*B*	*C*
Serum bilirubin (mg/dL)	<2	2–3	>3
Serum albumin (g/dL)	>3.5	3–3.5	<3
Ascites	None	Easily controlled	Poorly controlled
Encephalopathy	None	Minimal	Advanced
Nutrition	Excellent	Good	Poor