Surgery of the Pancreas

By

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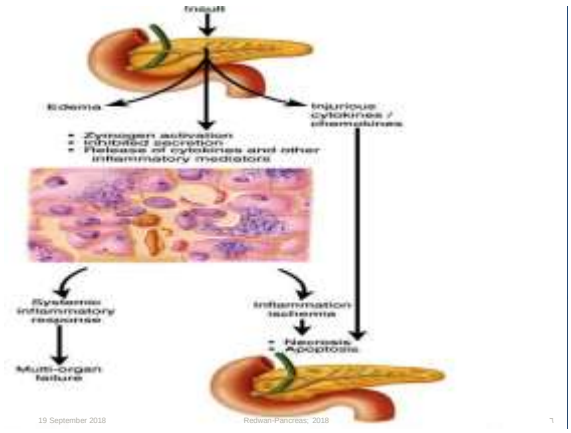
Acute Pancreatitis

Etiologic Factors in Acute Pancreatitis
METABOLIC
Alcohol
Hyperlipoproteinemia
Hypercalcemia
Drugs
Genetic
Scorpion venom
MECHANICAL
Cholelithiasis
Postoperative
Pancreas divisum
Post-traumatic
Retrograde pancreatography
Pancreatic duct obstruction: pancreatic tumor, ascaris infestation
Pancreatic ductal bleeding
Duodenal obstruction
VASCULAR
Postoperative (cardiopulmonary bypass)
Periarteritis nodosa
Atheroembolism
INFECTION
Mumps
Coxsackie B
Cytomegalovirus
Cryptococcus

Etiologies of Acute Pancreatitis
Alcohol
Biliary tract disease
Hyperlipidemia
Hereditary
Hypercalcemia
Trauma
External
Surgical
Endoscopic retrograde cholangiopancreatography
Ischemia
Hypoperfusion
Atheroembolic
Vasculitis
Pancreatic duct obstruction
Neoplasms
Pancreas divisum
Ampullary and duodenal lesions
Infections
Venom
Drugs
Idiopathic



Grey Turner sign in a patient with severe necrotizing pancreatitis.

Ranson's Prognostic Signs of Pancreatitis

At admission	During the initial 48 h
Criteria for acute pancreatitis not due to gallstones	
Age >55 y	Hematocrit fall >10 points
WBC >16,000/mm³	BUN elevation >5 mg/dL
Blood glucose >200 mg/dL	Serum calcium <8 mg/dL
Serum LDH >350 IU/L	Arterial PO_2 <60 mm Hg
Serum AST >250 U/dL	Base deficit >4 mEq/L
	Estimated fluid sequestration >6 L
Criteria for acute gallstone pancreatitis	
Age >70 y	Hematocrit fall >10 points
WBC >18,000/mm³	BUN elevation >2 mg/dL
Blood glucose >220 mg/dL	Serum calcium <8 mg/dL
Serum LDH >400 IU/L	Base deficit >5 mEq/L
Serum AST >250 U/dL	Estimated fluid sequestration >4 L

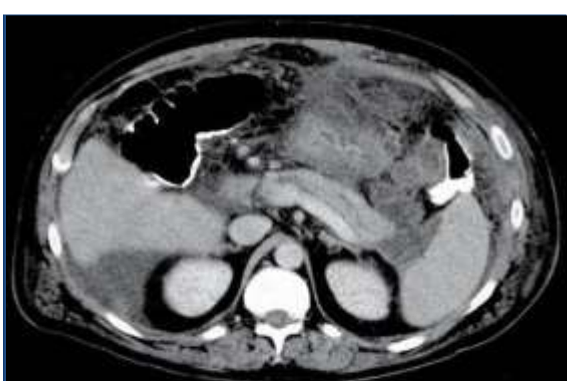
Imrie (Glasgow) prognostic grading system for acute pancreatitis (all within 48 h).

- Age > 55 years
- White cell count > 15 × 10^9/L
- Blood glucose > 10 mmol/L and patient not diabetic
- Serum albumin < 32 g/L
- Blood urea > 16 mmol/L with no response to i.v. fluids
- Lactate dehydrogenase > 600 U/L
- Aspartate aminotransferase/alanine aminotransferase > 100 U/L
- Serum calcium < 2.0 mmol/L

The APACHE II system allocates three sets of points: A, B and C.

- A: assessment of clinical parameters, e.g. vital signs, electrolytes, arterial blood gases, etc.
- B: points allocated in accordance with age.
- C: points added for comorbid disease or chronic health of patient.

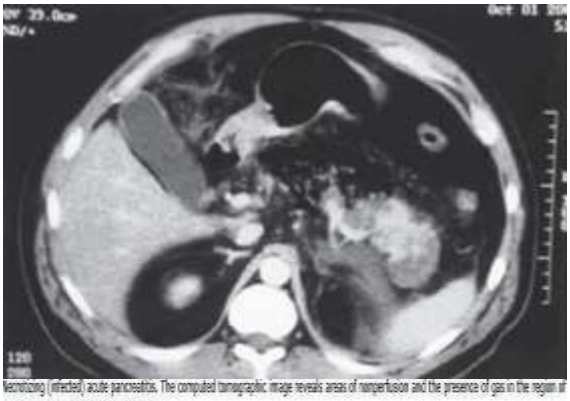
The APACHE II score is the sum of A, B and C; if this exceeds 9, the patient has severe acute pancreatitis. Mortality is very high if the score increases after admission.



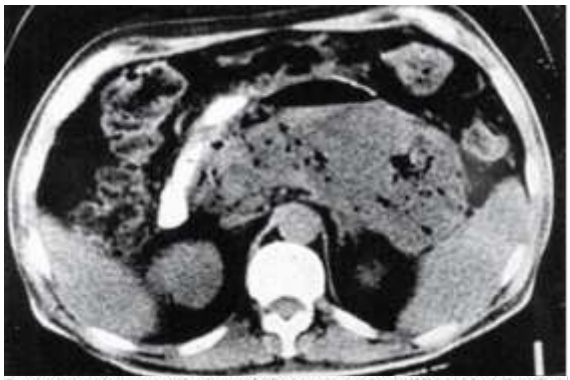
Contrast-enhanced abdominal CT scan in a 47-year-old man with acute pancreatitis. Relevant findings include significant fat stranding of the peripancreatic tissues, with a fluid collection at the tail of the pancreas measuring approximately 4 x 4 cm. Pancreatic parenchyma enhances with IV contrast, with no evidence of pancreatic necrosis. (Reprinted from Clancy TE, Benoit EP, Ashley SW. Current management of acute pancreatitis. J Gastrointest Surg 9:440–452, copyright © 2005, with permission from Elsevier.)

Contrast-enhanced abdominal CT scan in the same 47-year-old man with a second episode of acute pancreatitis. Scan shows stranding of peripancreatic fat consistent with acute pancreatitis. Most notable is the near complete absence of pancreatic enhancement, diagnostic of pancreatic necrosis. (Reprinted from Clancy TE, Benoit EP, Ashley SW. Current management of acute pancreatitis. J Gastrointest Surg 9:440–452, copyright © 2005, with permission from Elsevier.)

Nonnecrotizing acute pancreatitis. The computed tomographic image reveals edema (large arrow) and fluid (small arrow) but intact vascularization of the pancreas overall.

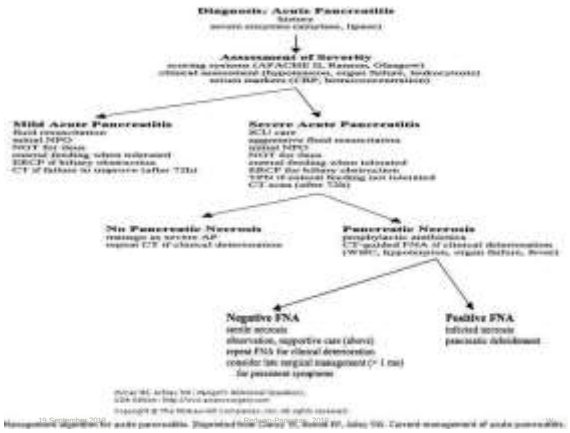


Necrotizing (infected) acute pancreatitis. The computed tomographic image reveals areas of nonperfusion and the presence of gas in the region of severe necrosis, which indicates infection.



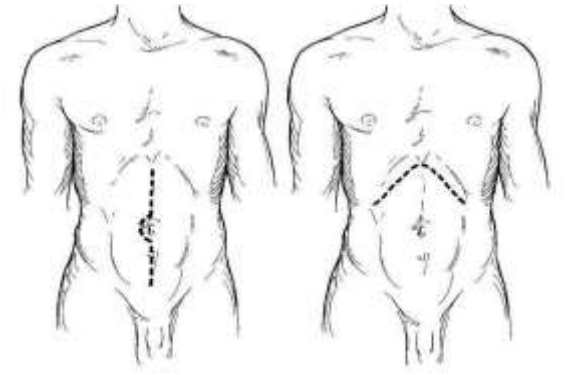
CT scan demonstrating emphysematous pancreatitis, pathognomonic for infected pancreatic necrosis. Operative debridement is indicated without additional confirmation of pancreatic infection.

CT-guided percutaneous fine-needle aspiration of the pancreatic tail. The aspiration area had been identified previously as necrotic in the contrast-enhanced CT shown in Figure 36–2. Gram's stain and cultures were negative for organisms, consistent with sterile pancreatic necrosis. (Reprinted from Clancy TE, Bansal VP, Adler DA. Current management of acute pancreatitis. J Gastrointest Surg 5:445–452, copyright © 2001, with permission from Elsevier.)



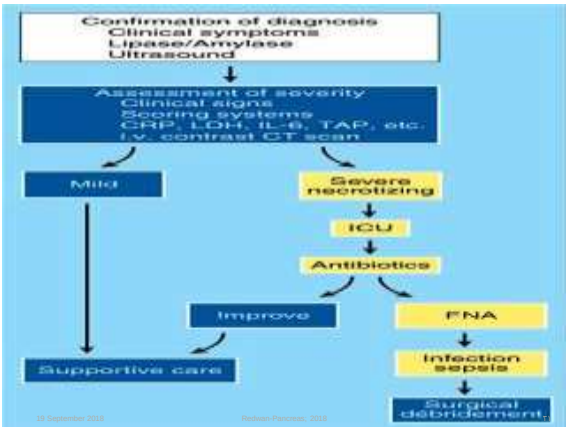
Management algorithm for acute pancreatitis. (Reprinted from Clancy TE, Bansal VP, Adler DA. Current management of acute pancreatitis.

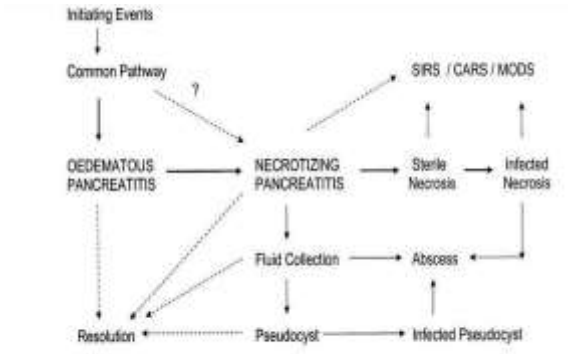
Table 36–5. Definitions Proposed by the International Symposium on Acute Pancreatitis (The Atlanta Symposium), 1992[29]	
Acute pancreatitis	Acute inflammatory process of the pancreas with variable involvement of other regional tissues or remote organ systems.
Severe AP	Association with organ failure and/or local complications, such as necrosis, abscess, or pseudocyst.
Acute fluid collection	Occurs early in the course of AP, located in or near the pancreas, always lacking a wall of granulation or fibrous tissue; bacteria variably present; occurs in 30–50% of severe AP; most acute fluid collections regress, but some progress to pseudocyst or abscess.
Pancreatic necrosis	Diffuse or focal area(s) of nonviable pancreatic parenchyma, typically associated with peripancreatic fat necrosis, diagnosed by CT scan with intravenous contrast enhancement.
Acute pseudocyst	Collection of pancreatic juice enclosed by a wall of fibrous or granulation tissue, which arises as a consequence of AP, pancreatic trauma, or chronic pancreatitis; formation requires 4 or more weeks from onset of AP.
Pancreatic abscess	Circumscribed intra-abdominal collection of pus usually in or near the pancreas, containing little or no pancreatic necrosis, arises as a consequence of AP or pancreatic trauma.



Operative approaches to open pancreatic debridement. Either a midline or bilateral subcostal approach is acceptable.

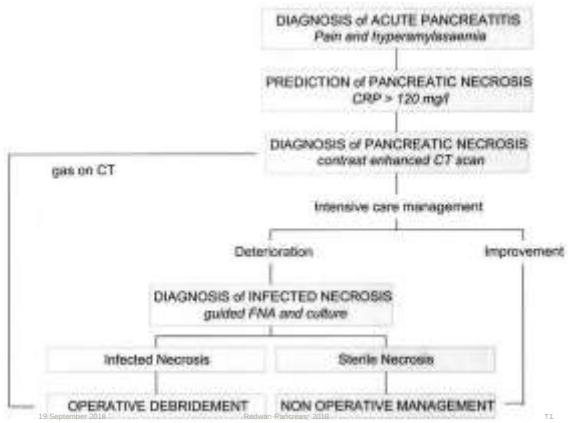
Operative view of infected acute pancreatitis. Peripancreatic infection, characterized by mucopurulent exudate, extends far beyond the boundaries of the pancreas in the retroperitoneum.

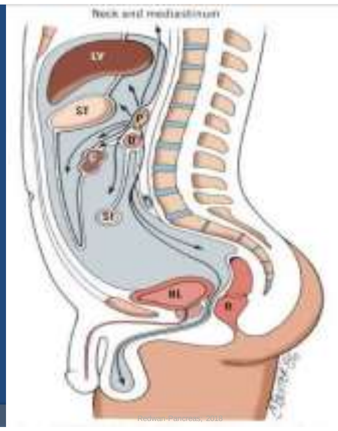


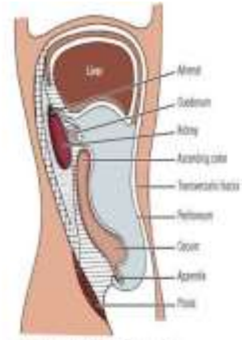


The protean nature of acute pancreatitis and related complications. SIRS = systemic inflammatory response syndrome; CARS = compensatory anti-inflammatory response syndrome; MODS = multiple-organ-dysfunction syndrome.2018 TT

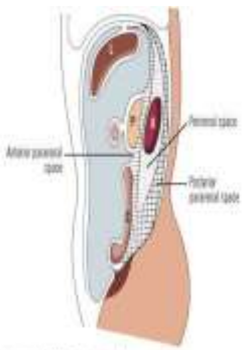
The Complications of Acute Pancreatitis	
Local	Fluid collections
	Pancreatic ascites/pleural effusion
	Pancreatic pseudocyst
	Pancreatic necrosis
	Infected pancreatic abscess
	Hemorrhage/pseudoaneurysm
Regional	Venous thrombosis
	Paralytic ileus
	Intestinal obstruction
	Intestinal ischemia/necrosis
	Cholestasis
Systemic	Systemic inflammatory response syndrome
	Multiple-organ-dysfunction syndrome
	ARDS/pulmonary failure
	Renal failure
	Cardiovascular complications
	Hypocalcemia
	Hyperglycemia
	Disseminated intravascular coagulopathy
	Protein calorie malnutrition
	Encephalopathy







Three extraperitoneal compartments. Stripes, anterior pararenal space; stipples, perirenal space; cross hatches, posterior pararenal space. (Modified from Meyers MA. Radiologic features of the spread and localization of extraperitoneal gas and their relationship to its source: An anatomical approach. Radiology 1974;111:17-26; with permission.)



Relationships and structures of three extraperitoneal spaces on left. Sigmoid colon in continuity with posterior and anterior pararenal compartments. L, liver; P, pancreas; K, kidney; C, colon. (Modified from Meyers MA. Acute extraperitoneal infection. Semin Roentgenol 1973;8:445-464; with permission.)

Spaces and peritoneal reflections of abdomen. Spaces: A, right subphrenic; B, left subphrenic; C, right subhepatic; D, lesser sac; E, perisplenic; F, right paracolic; G, left paracolic; H, pelvic; I, left infracolic; K, right infracolic. Reflections: 1, coronary ligaments and bare area; 2, duodenum; 3, small intestine; 4, colon; 5, stomach; 6, phrenicocolic ligament; 7, urachus; 8, gastrosplenic ligament; 9, hepatogastric ligament; 10, falciform ligament. Possible pathways of pancreatic drainage: W, left paracolic space to scrotum; X, perforation into duodenum or colon; Y, perforation into perisplenic or left paracolic spaces; Z, perforation through diaphragm to pleura and lung. (Modified from [illegible] Anatomical Complications in General Surgery. New York: McGraw-Hill, [illegible]; with permission.)

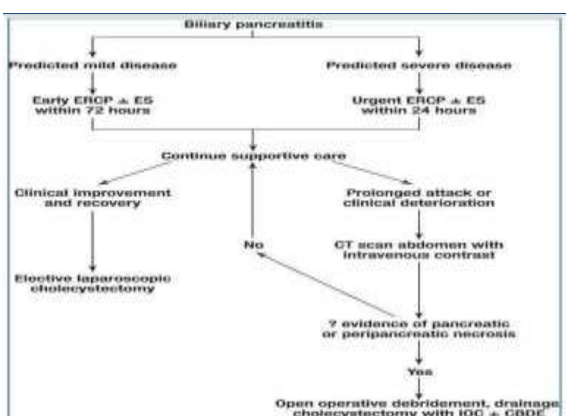
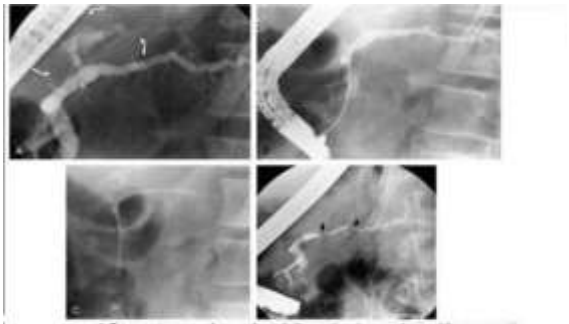
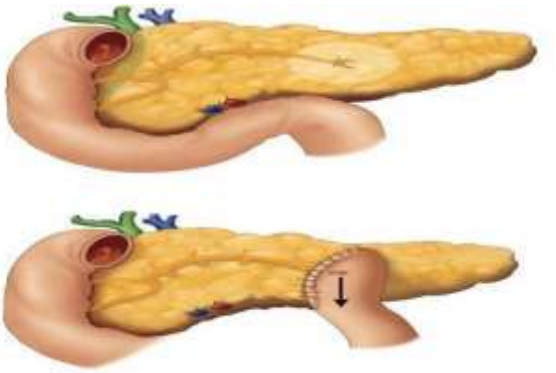


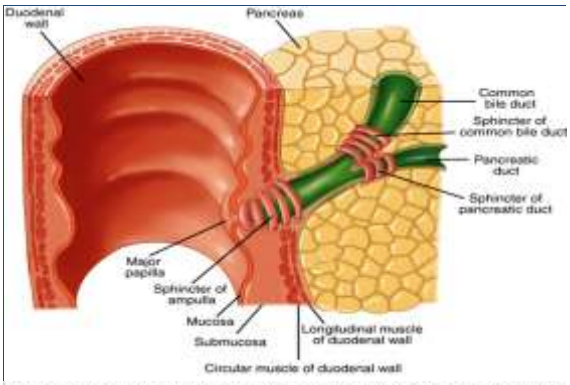
Table 37–4. Open and Minimally Invasive Approaches to the Treatment of Pancreatic Necrosis
Open surgery approaches
Pancreatic resection
Necrosectomy + wide tube drainage
Necrosectomy + relaparotomy (staged reexploration)
Necrosectomy + drainage + relaparotomy
Necrosectomy + laparostomy ± open packing
Necrosectomy + drainage + closed continuous lavage
Minimally invasive approaches
Laparoscopic necrosectomy
Laparoscopic intracavity necrosectomy
Laparoscopic assisted percutaneous drainage
Laparoscopic transgastric necrosectomy
Percutaneous necrosectomy and sinus tract endoscopy
MRI–radiologically assisted necrosectomy
Translumbar extraperitoneal retroperitoneoscopy
Video-assisted retroperitoneal débridement



A, Pancreatogram shows ductal disruption in a patient with pancreatic ascites. *Circled area* and *arrows* depict the site of duct disruption. *B*, Guidewire is passed beyond the site of ductal leakage. *C*, Pancreatic duct stent. After stent placement and a single paracentesis, the patient left the hospital after an overnight stay with complete ascites resolution. *D*, Main duct strictures *(arrows)*, possibly stent-induced, were noted 2 months later at stent retrieval.



Internal drainage for leaking pancreatic duct. A Roux-en-Y pancreaticojejunostomy is performed at the site of duct rupture to accomplish internal drainage of the pancreatic duct leak.



Schematic diagram of the ampullary, biliary, and pancreatic duct sphincters. The point of merger of the bile duct and pancreatic duct is highly variable, and a true sphincter of the pancreatic duct may be poorly developed.

Zinner MJ, Ashley SW: Maingot's Abdominal Operations, 11th Edition: http://www.accessurgery.com

Detail of ERCP showing the bile duct and pancreatic duct entering the duodenum.

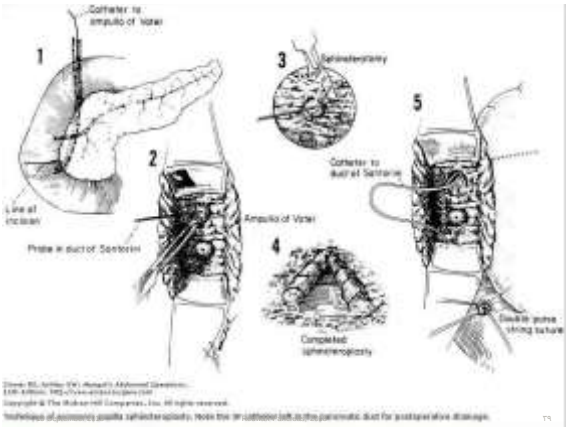
A

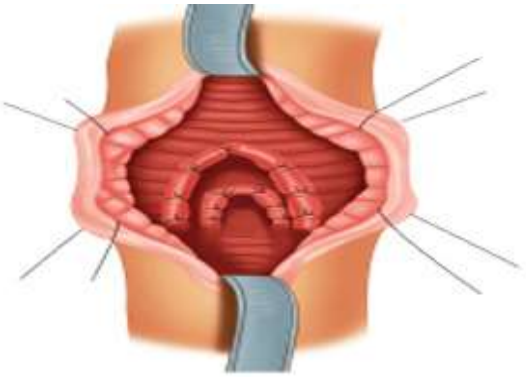
Zinner MJ, Ashley SW: Maingot's Abdominal Operations, 11th Edition: http://www.accessurgery.com

Two examples of ampullary stenosis. **A**, Dilated bile and pancreatic ducts in a man with recurrent discrete attacks of acute pancreatitis. **B**, Narrowed ampullary segments of the bile duct (*open arrow*) and pancreatic duct (*closed arrow*) in a woman with persistent pain, normal serum amylase, and a positive secretin-ultrasound test. Both were cured by transduodenal sphincteroplasty and transampullary septectomy.

Pancreatogram via the major ampulla and duct of Wirsung, showing typical pancreas divisum. The duct is short (2–3 cm) and ends in a fine tapered arborization.

Pancreatogram showing a dominant dorsal duct. The dorsal duct in this case drains the entire pancreas, including the head. There is no duct of Wirsung.





Operative sphincteroplasty of the biliary and pancreatic duct. The ampullary and bile duct sphincters are divided, as is the pancreatic duct sphincter, with suture apposition of the mucosal edges of the incision.

Laparoscopic necrosectomy for infected pancreatic necrosis.