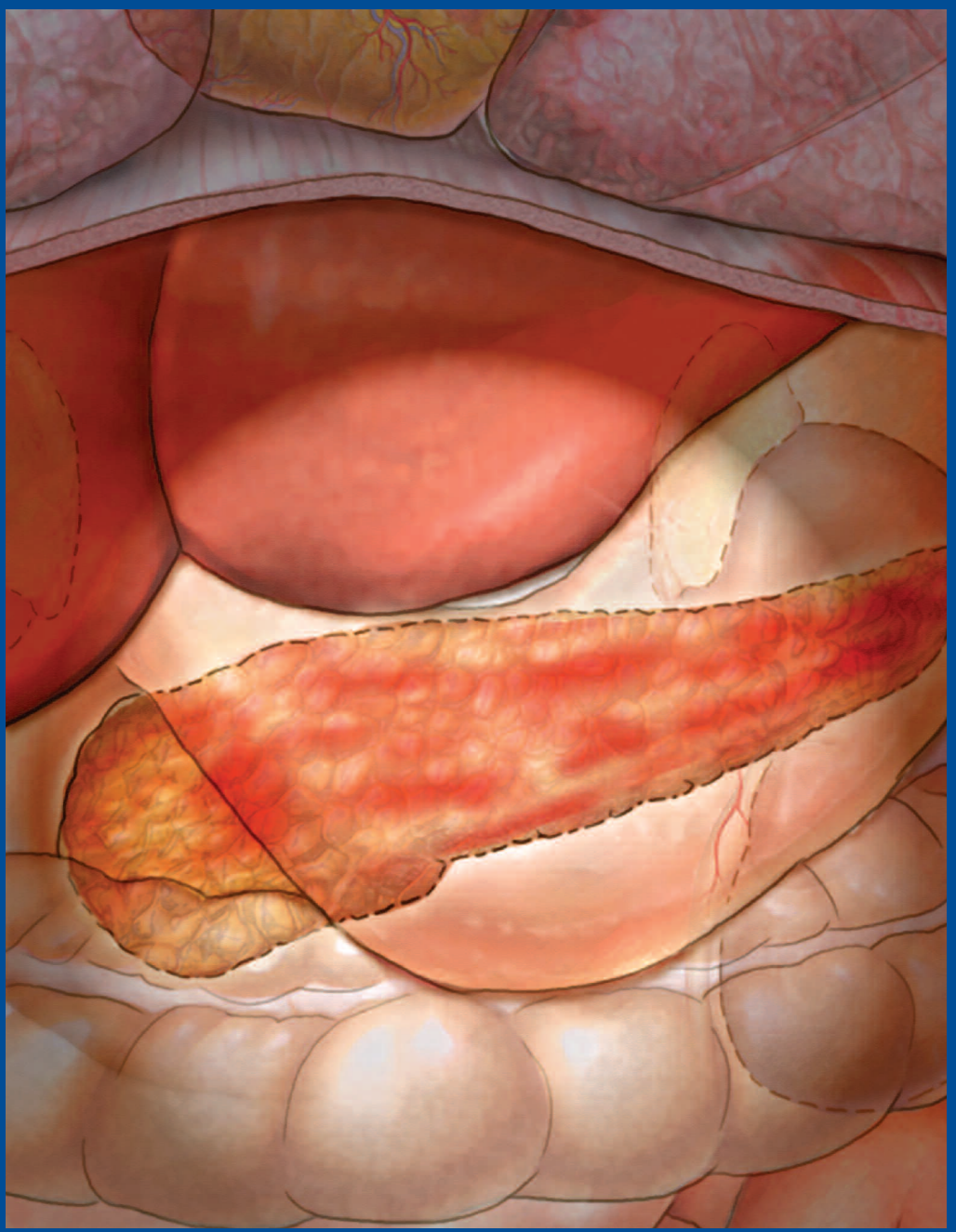
Acute pancreatitis

Inflammation gone wild

CE
2.5
ANCC/AACN
CONTACT HOURS



An attack of acute pancreatitis can leave your patient seriously ill; it can even kill him. In this article, we'll help you understand what happens inside the body when the pancreas is attacked, how pancreatitis is diagnosed, and what you can do to help your patient.

REBECCA A. PHILLIPS, RN, PHD
Assistant Professor • College of Nursing • University of Oklahoma
Oklahoma City, Okla.

The author has disclosed that she has no significant relationships with or financial interest in any commercial companies that pertain to this educational activity.

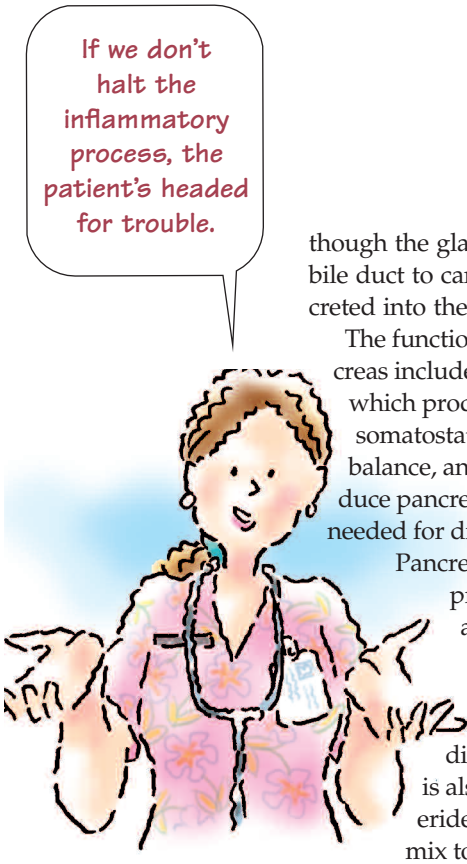
EVER CARE FOR a patient with pancreatitis who died despite giving him every antibiotic in the book? When nothing worked, I'll bet you wondered just what had happened. And even if your patient survived, you were probably pretty alarmed by just how serious this condition can be.

Pancreatitis can be frightening—for you and for the patient—because so much can be going wrong and you may feel powerless to stop it. But not every patient with acute pancreatitis dies, and there's a lot you can do to help patients recover. In this article, I'll get into the details on this potentially deadly condition, including signs and symptoms of pancreatitis, risk factors for the disease, its diagnosis and treatment, and nursing considerations you should know.

Let's start with a brief review of the anatomy and physiology of the pancreas.

Two organs in one

The pancreas is both an endocrine and an exocrine gland. The organ is divided into a head, body, and tail. The head joins the common bile duct where it drops down behind and into the descending duodenum. The body of the pancreas forms a shelf where the stomach rests; it's also where veins and arteries for the duodenum separate. The tail forms a shelf for the spleen to rest on. The pancreatic duct runs transversely left to right



though the gland, joining with the common bile duct to carry pancreatic juices to be secreted into the duodenum.

The functional components of the pancreas include the islets of Langerhans, which produce insulin, glucagon, and somatostatin to keep blood glucose in balance, and the acinar cells, which produce pancreatic juices and bicarbonate needed for digestion.

Pancreatic juices contain the inactive protease form of trypsinogen and chymotrypsinogen. These activate in the duodenum to become trypsin and chymotrypsin, which help digest protein. Pancreatic lipase is also released to digest triglycerides, and amylase is added to the mix to digest starches. This combination turns the food we eat into substances the body can use for energy.

Now that you know what goes on when everything's working, let's look at what happens when things start going wrong.

Wildfire

Acute pancreatitis can take two forms: edematous (or interstitial) pancreatitis or necrotizing pancreatitis.

- ***Edematous pancreatitis*** causes fluid accumulation and swelling. It's usually mild and self-limiting.
- ***Necrotizing pancreatitis*** is more severe, as its name suggests. It causes cell death and tissue damage, with serious systemic complications.

No matter which form it takes, at its core, pancreatitis is an inflammatory process that begins within the acinar cells. If not checked, it can affect multiple systems and cause them to fail. The severe form of pancreatitis is an aggressive hyperinflammatory reaction; it's caused by release of chemical mediators, called cytokines, that are associated with inflammation (see *At the cellular level*). The severity of acute pancreatitis and the risk of mortality are predicted by using Ranson's criteria (see *How bad is it?*).

The patient's response determines whether pancreatitis becomes chronic. Chronic pancreatitis is diagnosed when progressive, recurring episodes of inflammation cause structural changes within the pancreas (see *What about chronic pancreatitis?*).

Stones, toxins, and who knows?

The wide-angle view of the causes of acute pancreatitis includes obstruction, genetic

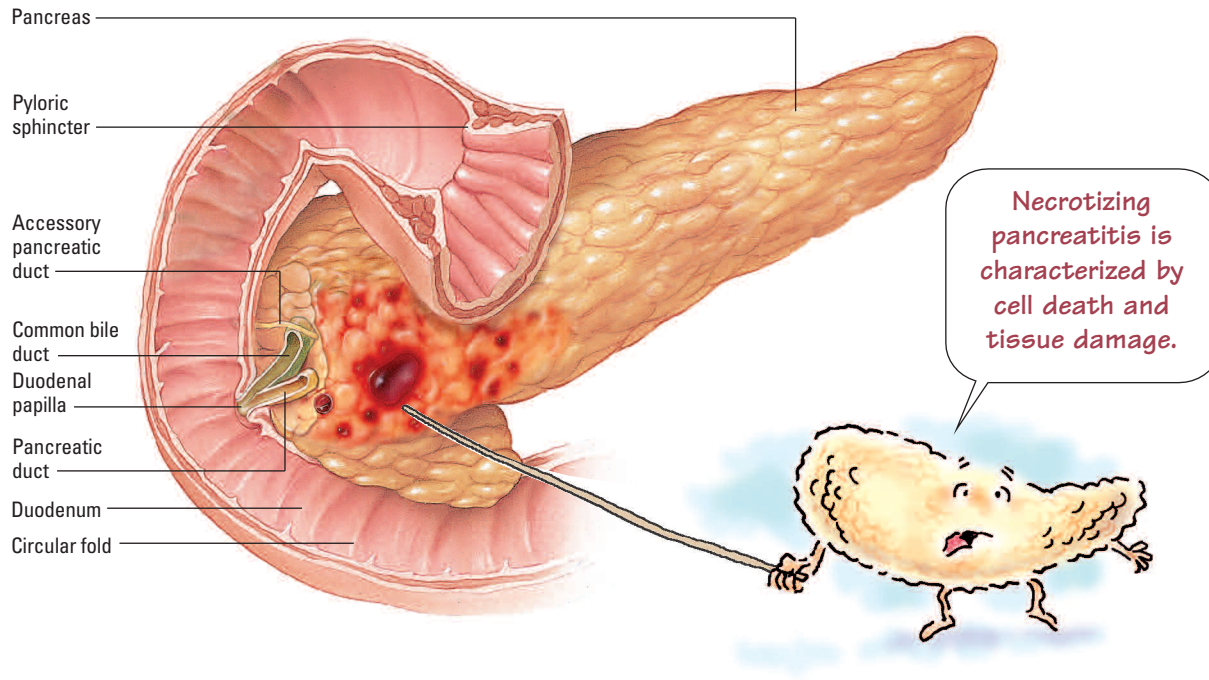
At the cellular level

The inflammatory process is in full swing in a patient with pancreatitis, regardless of what caused the condition. The process begins in the acinar cells of the pancreas when they secrete, among other things, platelet-activating factor (PAF), cytokines, and acute phase proteins (APPs) in response to various triggers.

PAF stimulates the hyperinflammatory response that can result in multiorgan failure. It's released by the polymorphonuclear white cells, which are usually mildly to moderately elevated at first. PAF also signals for the release of histamine, which causes the cells of the blood vessel walls to begin to loosen their hold on each other and creates a tiny gap for fluid to leak through and out into the tissues.

Cytokines are released from monocytes and signal the liver, which is already releasing cytokines from its Kupffer cells, to synthesize and release APPs. Release of APPs triggers the coagulation, fibrinolytic, kallikrein-kinin, and complement cascades. The end point for the kallikrein-kinin and complement cascades is further production and release of cytokines. And so the cycle continues.

A view of acute pancreatitis



predisposition, toxic metabolic processes, hypertriglyceridemia, infectious agents, and idiopathic origin.

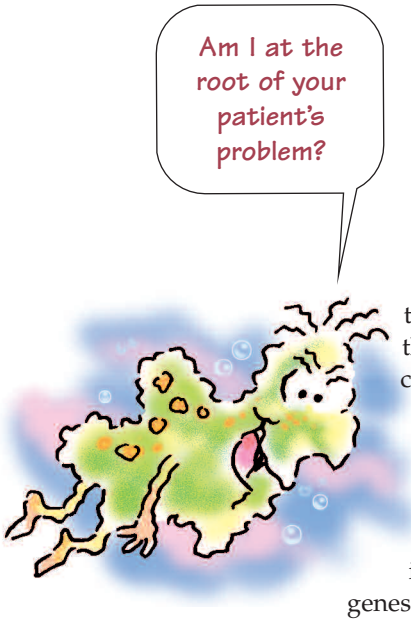
Obstruction can be caused by gallstones, stenosis of the sphincter of Oddi, neoplasms, pancreatic divisum, and trauma. Obstruction most commonly occurs from gallstones that migrate into the lower bile duct, where they block passage of bile into the duodenum. They may also settle in the pancreatic duct before it joins the common bile duct, blocking the bile passage higher up in the drainage system.

Repeated exposure to stones may cause stenosis of the sphincter of Oddi. The sphincter's job is to regulate the forward flow of bile and pancreatic juices into the duodenum while preventing reflux. So it's easy to see how obstruction could lead to an attack of acute pancreatitis.

Any lesion, such as a neoplasm, that occupies space in the pancreatic parenchyma will obstruct the outflow of juices and cause obstructive autodigestion and pancreatitis.

Pancreatic divisum is a congenital anomaly occurring when the dorsal and lateral ducts of the pancreas fail to fuse during the second month of gestation. This causes up to 95% of the pancreatic juice to flow through the dorsal duct, which happens to be the one with the small, minor papillary orifice. Although this rarely causes problems, you can see how it could create an obstruction that would lead to an acute pancreatitis.

Trauma may be caused by injury during endoscopic retrograde cholangiopancreatography related to multiple contrast injections, high injection pressures, contrast shot into the acinar cell cluster, or an inexperienced operator performing the procedure. Patients who've had upper abdominal, renal, or cardiovascular surgeries may develop pancreatitis because of injury to



the pancreas or obstruction of the bile pathway during the procedure. And any episode of prolonged ischemia could lead to pancreatitis.

Genetic predisposition relates to the genes for cationic trypsinogen and serine protease inhibitor. Mutation of these genes allows trypsinogen to convert to trypsin in the pancreas instead of the duodenum, setting the stage for autodigestion and pancreatitis.

How bad is it?

The severity of acute pancreatitis is determined by the existence of certain criteria, called Ranson's criteria. The more criteria met by the patient, the more severe the episode of pancreatitis—which increases the risk of mortality.

Mortality is less than 1% among patients who meet fewer than three of the criteria. It rises to 16% when three to four criteria are met and to 40% with five or six criteria. Mortality is 100% when the patient meets seven or eight criteria.

The health care provider establishes the severity of the disease on admission and during the first 48 hours after admission by evaluating the patient for the following criteria:

On admission

- patient older than age 55 years
- white blood cell count > 16,000/mcL
- serum glucose level > 200 mg/dL
- serum lactate dehydrogenase level > 350 units/liter
- aspartate aminotransferase level > 250 units/liter

After admission

- 10% decrease in hematocrit
- blood urea nitrogen level increase > 5 to 8 mg/dL within 48 hours of admission
- serum calcium level < 8 mg/dL
- base deficit > 4 mEq/liter
- partial pressure of arterial oxygen < 60 mm Hg
- estimated fluid sequestration > 6 liters.

Toxic metabolic processes include ethanol abuse and certain drug regimens. Ethanol abuse is classified as a chronic, daily intake of 100 to 150 grams/dL. Ethanol sets up a transient but severe drop in blood flow to the pancreas, triggering a vicious cycle of repeated ischemic episodes resulting in cellular damage. The more cells destroyed by the ischemic event, the sooner pancreatitis will occur and the more severe the attack will be.

Immunosuppressant drug regimens, including azathioprine (Imuran), mercaptopurine (Purinethol), and didanosine (Videx), may also cause acute pancreatitis. Why's that? Suppression of the immune system increases the risk of infection, which is one of the causes of acute pancreatitis.

Hypertriglyceridemia occurs when large amounts of cytotoxic free fatty acids are released into the pancreatic circulation.

When the triglyceride level exceeds 1,000 mg/dL, lipase in the pancreas binds triglycerides to albumin. Once albumin is saturated, the pancreas releases triglycerides as free fatty acids that are toxic to the acinar cells. In addition, red blood cells become sluggish and plug capillaries. Capillary plugging and stasis of blood flow result in vascular endothelial damage, pancreatic ischemia, acidosis, activation of trypsinogen, and the flare-up of acute pancreatitis.

The patient's blood becomes so lipemic that serum amylase can't be used to measure pancreatic dysfunction. Urine amylase must be checked to find out how the pancreas is doing.

Infectious agents that can cause acute pancreatitis include viruses, bacteria, and parasites. Most of the viruses you know fall into this category: measles, mumps, rubella, coxsackie B, Epstein-Barr, cytomegalovirus, and the viruses that cause the different types of hepatitis. Bacterial sources include *Legionella*, *Mycoplasma pneumoniae*, *Mycobacterium tuberculosis*, and *Campylobacter jejuni*, to name a few. Parasites that can cause acute pancreatitis include *Ascaris* and *Clonorchis*.

What about chronic pancreatitis?

Structural changes within the pancreas, resulting from progressive, recurring episodes of inflammation, are at the root of chronic pancreatitis. Damage to the functional capabilities of the organ usually starts with the exocrine side, causing weight loss from an inability to digest and absorb nutrition from the intestinal tract.

If pancreatitis causes damage to the organ's islets of Langerhans, the patient typically develops diabetes mellitus. This is usually a late symptom of chronic pancreatitis. As a complication of the inflammatory process, calculi develop within the pancreas in up to 60% of the cases. These calculi can cause stenosis of the common bile duct and portal hypertension.

The causes of chronic pancreatitis differ from those that trigger an acute attack and include the following:

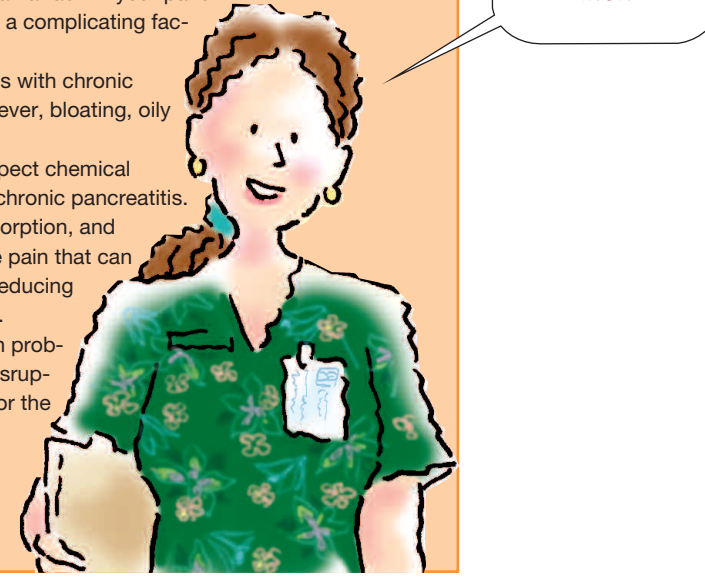
- ***Ethanol intake*** creates a progressive, calcifying form of pancreatitis and causes 80% of cases of chronic pancreatitis. It occurs with a daily intake of 80 grams/dL or more for a period of 35 years or longer. Ethanol-induced attacks of acute pancreatitis are likely to end up in chronic pancreatitis, but this outcome depends on how much the ethanol has destroyed the underlying parenchyma.
- ***Protein and trace element malnutrition*** is referred to as tropical chronic pancreatitis, or kwashiorkor, and it's associated with toxin uptake that causes calcifications in the pancreas.
- ***Hereditary*** chronic pancreatitis is an autosomal disorder described in over 100 cases to date.
- A ***lowered trypsinogen level*** due to a mutation in the activation peptide can lead to chronic episodes of pancreatitis.
- ***Juvenile idiopathic*** chronic pancreatitis is caused by an imbalance of proteases and antiproteases in the early decades of life.
- ***Idiopathic*** causes remain unclear, but may be related to inflammation and fibrosis of the pancreas.
- ***Recurrent acute episodes*** can lead to a chronic state because of the underlying calcification of the pancreas in acute episodes.

Patients with chronic pancreatitis aren't immune to acute pancreatitis. How can you tell if your patient's having an acute attack? Generally, not by the pain that's typical of acute pancreatitis: Up to half of patients with chronic pancreatitis won't experience that pain during an acute episode. It seems that the longer pancreatitis exists, the more the gland is destroyed and the less likely the patient is to have severe pain—or any pain—related to an attack. If your patient with chronic pancreatitis does have pain, expect to see a complicating factor, such as pseudocysts or cholestasis.

Other acute symptoms, however, do occur in patients with chronic pancreatitis, such as nausea and vomiting, along with fever, bloating, oily malodorous stool, and weight loss.

Because ethanol abuse is the number one cause, expect chemical dependency therapy to be offered to your patient with chronic pancreatitis. Also, give enzymes to aid digestion and encourage absorption, and recommend dietary changes that will help decrease the pain that can be associated with early chronic pancreatitis, such as reducing fat intake and consuming smaller, more frequent meals.

As with all chronic illnesses, depression is a common problem that will need to be treated to bring the chemical disruption back into balance and provide emotional stability for the patient.



Why do these infectious agents cause acute pancreatitis? Here's one theory: Pathogenic organisms are thought to trigger proteolytic enzymes (trypsinogen, chymotrypsinogen, protease) to become activated within the pancreas instead of within the intestine. The enzymes digest pancreatic tissue, resulting in pancreatitis.

Idiopathic is listed far more frequently than it probably should be as the reason for pancreatitis. It's the catchall for "I don't know what caused this." As many as one-quarter of pancreatitis episodes are labeled idiopathic following a workup that includes a thorough history; routine lab studies such as liver function tests, calcium level, triglycerides, globulin level, and serum amylase and serum lipase levels; and noninvasive studies such as ultrasound.

When these studies fail to identify the cause, most health care providers stop looking and call the etiology idiopathic. However, health care providers should take advantage of the diagnostic technologies available before labeling an episode idiopathic. These include abdominal and chest X-rays (which show bowel dilation and ileus, as well as pleural effusion), computed tomography scan (which can visualize pancreatic abscesses, pseudocysts, and an enlarged pancreas with fluid collection), cholangiopancreatography (which visualizes bile duct stones), and special lab studies such as gene analysis.

did you know?

Why do you sometimes see fever in patients with acute pancreatitis even though they don't have an infection? Pancreatic ascites fluid, obtained from a swollen and boggy pancreas, is rich in the cytokines interleukin-1β, and tumor necrosis factor-α, powerful pyrogenic inflammatory mediators. They cause the hypothalamus to raise body temperature, resulting in a fever.

Knowing the cause of the disease increases the likelihood that patients will receive the most appropriate treatment—which means they'll be less likely to suffer a recurrence.

Detecting pancreatitis

The patient with acute pancreatitis will complain of nausea, vomiting, and upper abdominal pain, usually with an abrupt onset and a characteristically steady, boring pain located in the periumbilical area and epigastrium. This pain may radiate to the back and may be more intense when the patient is walking or lying supine. It may be relieved by sitting up and leaning forward.

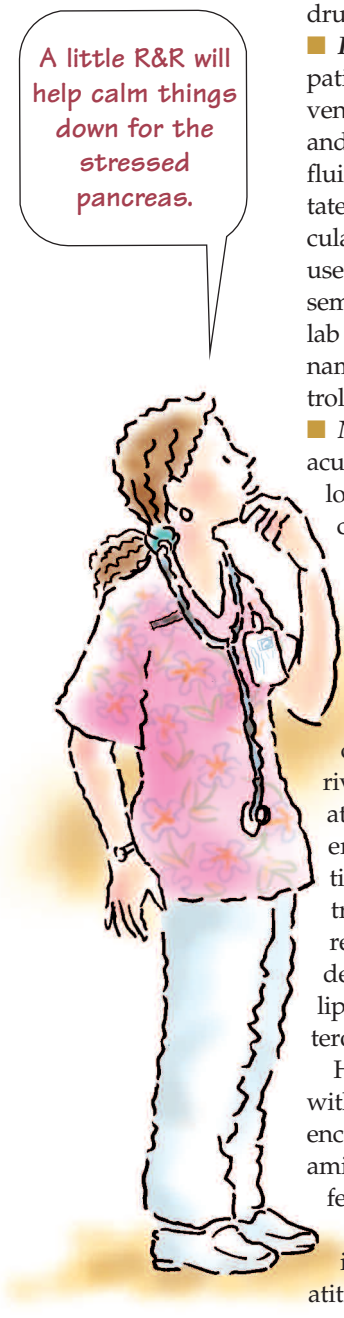
Other signs and symptoms of acute pancreatitis may include tachycardia, tachypnea, hypotension, abdominal distension, abdominal rigidity, mild jaundice, diminished bowel sounds, and occasional muscle spasms due to hypocalcemia. In more severe cases, you may see the Grey Turner sign (discoloration of the flank area) or the Cullen sign (discoloration of the periumbilical area related to hemorrhagic pancreatitis).

The trick is to be sure the symptoms aren't caused by some other abdominal disorder. The health care provider will order serum amylase and lipase levels, as well as a urine amylase level. If you see a serum amylase level four times the top of the reference scale, you can be sure your patient has acute pancreatitis. Elevated lipase and urine amylase levels seal the diagnosis.

Rest, drains, and drugs

Treatment goals for patients with acute pancreatitis include resting the pancreas and bowel, relieving pain, replacing fluids and electrolytes, providing nutritional support, and, in some cases, reducing hypertriglyceridemia and administering antibiotics. Some patients may even need surgery.

- ***Rest and relief of pain.*** If no other organ systems are involved and if there's no sign of necrosis or infection, keep the patient



N.P.O. so pancreatic juices are suppressed and the pancreas can take a rest. A nasogastric tube may be placed to relieve nausea and vomiting, keep the stomach empty, and reduce pain. Administering medications for pain relief is a nursing priority for patients with acute pancreatitis. Opioids are the drug of choice in this situation.

■ ***Fluid and electrolyte replacement.*** Most patients with acute pancreatitis need intravenous (I.V.) replacement of fluid, protein, and electrolytes. You'll generally give I.V. fluids like 0.9% sodium chloride and lactated Ringer's solution to increase intravascular volume. Electrolyte replacements are used to treat hyocalcemia, hypermagnesemia, and hypokalemia based on serum lab values. Monitor the patient's hemodynamic status, fluid balance status, and electrolyte values.

■ ***Nutritional support.*** If your patient with acute pancreatitis has to remain N.P.O. for a long period of time, or if he develops complications from pancreatitis, you can expect to administer total parenteral nutrition. You won't usually give lipids, though; they can raise the triglyceride level, which can exacerbate the inflammatory process in acute pancreatitis.

■ ***Hypertriglyceridemia.*** The health care provider may order fibric acid derivatives like gemfibrozil (Lopid) as a first attempt to reduce the patient's triglyceride level. These drugs have three actions: reduce the liver's ability to produce triglycerides through fatty acid uptake, reduce exchange between very low-density lipoproteins and high-density lipoproteins, and stimulate reverse cholesterol transport.

However, fibric acid derivatives aren't without adverse effects. Patients may experience elevated liver enzymes (aspartate aminotransferase and alanine aminotransferase), myalgia, gallstones, or, in rare cases, rhabdomyolysis. Closely monitoring hepatic function during acute pancreatitis is important anyway; it's even more so with these types of drugs.

Plasma exchange has also been used to reduce triglyceride level, but only lipoprotein aphaeresis should be used. This form of exchange removes the large molecules from the plasma while retaining immunoglobulins, albumin, and clotting factors. It significantly lessens the adverse effects of aphaeresis therapy by lowering the potential for bleeding and infection.

patient pointer

Instruct a patient being treated for acute pancreatitis to avoid high-fat foods and alcohol after he's discharged. They can trigger another acute attack.

■ ***Antibiotics.*** Many health care providers feel that prophylactic antibiotic therapy with imipenem-cilastatin (Primaxin) is the best way to avoid infection-related mortality during pancreatitis. Imipenem is one of the third-generation cyclosporine antibiotics that effectively diffuse into the pancreatic tissues, giving it the best chance of killing any invading organism.

■ ***Surgery.*** When surgery is needed, the aim is to debride the necrotic tissue of the pancreas and provide adequate drainage for any remaining debris. If the patient also has organ failure, the mortality rate rises. Postoperative management includes high-volume retroperitoneal lavage and repeat debridement as needed.

■ ***Easing the tension.*** While you're busy giving treatments and monitoring the situation, don't forget to take time to provide psychological support to patients and their families.

A shocking development

Local and systemic complications can occur with pancreatitis. The major complication of acute pancreatitis is systemic inflammatory response syndrome, a hyperinflammatory state that the body creates to help defend itself against an invasion of some type. This

defense attempt goes awry, leading to complications such as acute respiratory failure, acute respiratory distress syndrome, and shock.

Lung injury during an attack of acute pancreatitis is caused by the rapid infiltration of neutrophils as soon as 3 hours after the illness is triggered. As the blood travels through the portal circulation and the liver, alveolar macrophages leap into action. But they end up making the lung injury worse by helping to create pleural effusions, atelectasis, and pneumonia, all of which reduce oxygen uptake and carbon dioxide release within the capillaries.

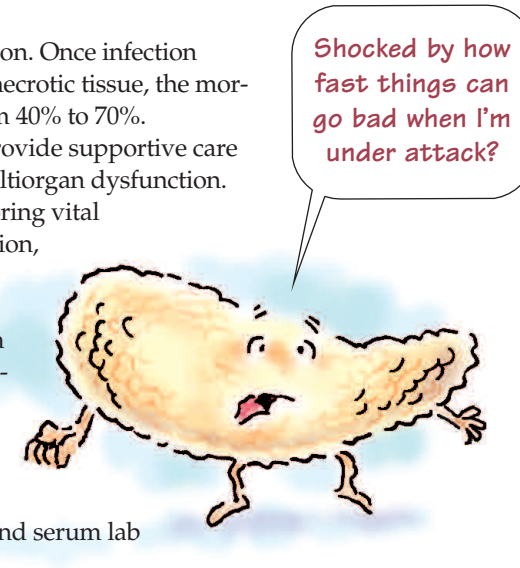
The hypoperfusion of shock further injures the sick pancreas as blood flow through the capillary bed slows. The pancreas reacts by releasing a substance known as myocardial depressant factor, which further adds to the systemic complications by reducing cardiac contractility, cardiac output, and perfusion pressure. During this low-flow state, stasis in the capillary beds leads to pancreatic ischemia and necrosis, as well as pooling of cytokines. Once perfusion pressure is raised, the cytokine pool is free to circulate, dumping a fresh load of chemical mediators within the circulating system and causing the cycle to continue.

Shock needs to be aggressively treated with volume replacement, vasopressors, and contractility agents as indicated by the patients' hemodynamic state. Lactated Ringer's solution or albumin is used to correct for loss of fluids. Blood and clotting factors should also be replaced as needed.

Once volume has been replaced, you may be adding vasopressors to help support blood pressure. Contractility agents are used when it becomes apparent that the cardiac output is falling because the heart is no longer beating strongly enough.

The development of pancreatic necrosis, and the probable infection that follows, is the most significant variable for patient mortality and relates to the progressive advance of multiorgan dysfunction. Once infection occurs in the face of necrotic tissue, the mortality rate ranges from 40% to 70%.

You'll primarily provide supportive care for a patient with multiorgan dysfunction. That includes monitoring vital signs, oxygen saturation, and hemodynamic parameters. Provide supplemental oxygen and prepare for endotracheal intubation and mechanical ventilation as necessary. Monitor the patient's fluid balance status and serum lab values.



Bowel rest is ordered, but in the face of the hyperinflammatory reaction, an inactive bowel allows for translocation of intestinal flora and pancreatic infection. Usual organisms cultured are *Escherichia coli*, *Klebsiella*, *Enterococcus*, *Staphylococcus*, and *Pseudomonas*. When the fungus *Candida albicans* is cultured, the outcome is grim.

Outcome options

The goal of treating an attack of pancreatitis is complete recovery. To get to that goal, it's important to correct the cause of the attack. If the patient has gallstones, for instance, a cholecystectomy will be needed to remove the stones. If hypertriglyceridemia caused the problem, working to control the triglycerides will help reduce the number and perhaps the severity of attacks, although most of these patients will develop chronic pancreatitis.

Severe attacks, with prolonged periods of hypoperfusion and pancreatic necrosis and infection, will end in death 70% of the time. Little can be done when the hyperinflammatory state leaves the patient profoundly hypoperfused and refractory to therapy.

Stay positive

Don't be discouraged after reading the last section. Yes, some patients with acute pan-

creatitis die, but others live and can lead productive, happy lives. Give your patients the best chance for survival by being alert to the possibility of pancreatitis, reacting quickly when it occurs, and watching closely for complications. You just may save someone's life. ■

Learn more about it

Ahmed SA, et al. Chronic pancreatitis: Recent advances and ongoing challenges. *Current Problems in Surgery*. 43(3):127-238, March 2006.

Connor S, Neoptolemos JP. Surgery for pancreatic necrosis: "Whom, when and what." *World Journal of Gastroenterology*. 10(12):1697-1698, June 2004.

Khoury G, Deeba SS. Pancreatitis. **http://www.emedicine.com/EMERG/topic354.htm**. Accessed June 16, 2006.

Kim H, et al. Idiopathic acute pancreatitis. *Journal of Clinical Gastroenterology*. 37(3):238-250, September 2003.

Porth CM. *Essentials of Pathophysiology: Concepts of Altered Health States*. Philadelphia, Pa., Lippincott Williams & Wilkins, 2006.

Smeltzer SC, Bare B. *Brunner & Suddarth's Textbook of Medical-Surgical Nursing*, 10th edition. Philadelphia, Pa., Lippincott Williams & Wilkins, 2003.

Society for Surgery of the Alimentary Tract. *Treatment of Acute Pancreatitis*. Manchester, Mass., Society for Surgery of the Alimentary Tract, 2004. **http://www.guideline.gov/summary/summary.aspx?ss=15&doc_id=5512&nbr=3755**. Accessed June 16, 2006.

Straight A's in Pathophysiology. Philadelphia, Pa., Lippincott Williams & Wilkins, 2005.

Tierney LM, et al. (eds). *Current Medical Diagnosis & Treatment*, 45th edition. New York, N.Y., McGraw-Hill, 2006.

Whitcomb DC. Clinical practice. Acute pancreatitis. *The New England Journal of Medicine*. 354(20):2142-2150, May 18, 2006.

Yadav D, Pitchumoni C. Issues in hyperlipidemic pancreatitis. *Journal of Clinical Gastroenterology*. 36(1):54-62, January 2003.

CE CONNECTION

Earn CE credit online:
Go to http://www.nursingcenter.com/CE/nmie and receive a certificate *within minutes*.

INSTRUCTIONS

Acute pancreatitis: Inflammation gone wild

TEST INSTRUCTIONS

- To take the test online, go to our secure Web site at **www.nursingcenter.com/ce/nmie**.
- On the print form, record your answers in the test answer section of the CE enrollment form on page 55. Each question has only one correct answer. You may make copies of these forms.
- Complete the registration information and course evaluation. Mail the completed form and registration fee of $22.95 to: **Lippincott Williams & Wilkins, CE Group**, 2710 Yorktowne Blvd., Brick, NJ 08723. We will mail your certificate in 4 to 6 weeks. For faster service, include a fax number and we will fax your certificate within 2 business days of receiving your enrollment form. Deadline is October 31, 2008.
- You will receive your CE certificate of earned contact hours and an answer key to review your results. There is no minimum passing grade.

DISCOUNTS and CUSTOMER SERVICE

- Send two or more tests in any nursing journal published by Lippincott Williams & Wilkins together and deduct $0.95 from the price of each test.
- We also offer CE accounts for hospitals and other health care facilities on nursingcenter.com. Call **1-800-787-8985** for details.

PROVIDER ACCREDITATION:

Lippincott Williams & Wilkins, the publisher of *Nursing made Incredibly Easy!,* will award 2.5 contact hours for this continuing nursing education activity. Lippincott Williams & Wilkins is accredited as a provider of continuing nursing education by the American Nurses Credentialing Center's Commission on Accreditation. This activity is also provider approved by the California Board of Registered Nursing, Provider Number CEP 11749 for 2.5 contact hours. Lippincott Williams & Wilkins is also an approved provider by the American Association of Critical-Care Nurses (AACN 00012278, CERP Category A), Alabama #ABNP0114, Florida #FBN2454, and Iowa #75. Lippincott Williams & Wilkins home study activities are classified for Texas nursing continuing education requirements as Type 1. Your certificate is valid in all states.

CE 2.5
ANCC/AACN CONTACT HOURS

Acute pancreatitis: Inflammation gone wild

GENERAL PURPOSE: To provide registered professional nurses with an overview of the causes, signs and symptoms, diagnosis, and nursing care for patients with acute pancreatitis. **LEARNING OBJECTIVES:** After reading the article and taking this test, you should be able to: 1. Describe the anatomy, physiology, and pathophysiology of the pancreas. 2. Discuss the causes, signs and symptoms, and diagnosis of acute and chronic pancreatitis. 3. Identify appropriate nursing interventions for acute and chronic pancreatitis.

---

1. Which statement about the pancreas is correct?
a. It's comprised of four lobes.
b. It's both an endocrine and exocrine gland.
c. Its tail forms a shelf where the stomach rests.

2. Which statement about pancreatic juices is correct?
a. Amylase helps digest protein.
b. Lipase helps digest triglycerides.
c. Trypsinogen helps digest starches.

3. The development of pancreatitis begins with the
a. islets of Langerhans.
b. pancreatic head.
c. acinar cells.

4. The hyperinflammatory response that occurs with pancreatitis is stimulated by
a. chymotrypsin.
b. somatostatin.
c. platelet-activating factor.

5. Ethanol abuse damages the pancreas by causing
a. ischemia.
b. autodigestion.
c. insulin resistance.

6. Which class of medication is most likely to cause acute pancreatitis?
a. antidiabetic agents
b. immunosuppressants
c. lipid-lowering agents

7. A patient with acute pancreatitis is most likely to report pain that
a. has an insidious onset and is relieved by walking.
b. can be relieved by sitting up and leaning forward.
c. is peristaltic and felt in the mid to lower abdomen.

8. All of the following are possible signs or symptoms of acute pancreatitis *except*
a. flat, soft abdomen.
b. abdominal rigidity and distension.
c. mild jaundice and diminished bowel sounds.

9. The Cullen sign can be recognized by
a. discoloration of the periumbilical area.
b. petechiae covering the abdomen.
c. ecchymosis of the flank area.

10. Which laboratory test result is *not* consistent with acute pancreatitis?
a. triglycerides level > 1,000 mg/dL
b. urine amylase below normal levels
c. serum amylase level four times the top of the reference scale

11. A priority nursing intervention for a patient with acute pancreatitis is
a. administering total parenteral nutrition (TPN) with lipids.
b. offering small, frequent meals.
c. administering opioid analgesics.

12. A patient with acute pancreatitis is most likely to receive which intravenous fluid?
a. 5% dextrose in water
b. lactated Ringer's solution
c. 5% dextrose in ½ normal saline

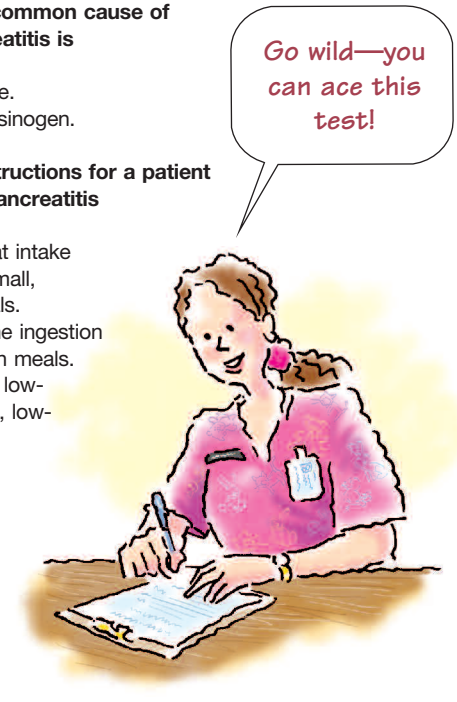
13. Which drug may be ordered for hypertriglyceridemia in a patient with acute pancreatitis?
a. gemfibrozil (Lopid)
b. atorvastatin (Lipitor)
c. pravastatin (Pravachol)

14. Which antibiotic should you expect to administer to a patient with acute pancreatitis?
a. ciprofloxacin (Cipro)
b. imipenem-cilastatin (Primaxin)
c. trimethoprim and sulfamethoxazole (Bactrim DS)

15. An initial clinical finding associated with chronic pancreatitis is
a. diabetes mellitus.
b. jaundice.
c. weight loss.

16. The most common cause of chronic pancreatitis is
a. kwashiorkor.
b. ethanol abuse.
c. elevated trypsinogen.

17. Dietary instructions for a patient with chronic pancreatitis include
a. decreasing fat intake and eating small, frequent meals.
b. eliminating the ingestion of liquids with meals.
c. consuming a low-carbohydrate, low-protein diet.



Turn to page 55 for the CE Enrollment Form.