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Pancreatitis is a heterogeneous inflammatory disease with a broad spectrum of clinical manifestations. Acute pancreatitis can present as a mild, self-limited illness with no sequelae or as fulminant multiple organ failure with circulatory collapse. Chronic pancreatitis may be clinically silent, marked by recurrent episodes of pain, or may cause endocrine and exocrine insufficiency. The outcome for an individual patient is difficult to predict at presentation. Despite improved diagnosis and treatment of complications, both acute and chronic pancreatitis carry significant morbidity and mortality.[1] Knowledge of common etiologies, especially medications, can lead to the early and accurate diagnosis of pancreatitis in the emergency department (ED). Additionally, goal-directed therapy and recognition of potential complications can be life-saving. This review outlines the common causes of pancreatitis and provides a rational approach to diagnosis and management in the ED.

—The Editor

Emergency Department Management of Pancreatitis

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Epidemiology

The incidence of acute pancreatitis varies worldwide and between patient populations. In England and the Netherlands the incidence is 5-10 cases per 100,000 inhabitants; in Scotland and Denmark it is 25-35 per 100,000; and in the United States and Finland it is 70-80 per 100,000 inhabitants. The incidence appears to be increasing, but this may reflect better imaging and improved methods of diagnosis.[2-4] The male to female ratio of acute pancreatitis is 1:0.6, and incidence increases with age.[5] For unknown reasons, the incidence in black men is three times greater than in white men.[6] The rate of chronic pancreatitis varies with the degree of alcohol abuse in the population studied, but in many parts of the world is in the range of 3-10 per 100,000 population.[7] Biliary tract disease and alcohol account for the vast majority of acute pancreatitis cases in the United States. Gallstones, the most common etiology, are encountered more frequently in females,

patients older than age 50, and in the community hospital setting.[8] Alcohol abuse predominates in males, younger patients, and in the inner city. Alcohol abuse also is the most common cause of chronic pancreatitis.

Acute pancreatitis is more prevalent in certain patient groups, such as those with HIV, hypertriglyceridemia, or hypercalcemia.[9,10] In children, abdominal trauma is the most common cause of acute pancreatitis, while cystic fibrosis is the most common cause of chronic pancreatitis.[11,12]

Although the majority of patients with acute pancreatitis have a mild, self-limited course, 15-25% will have a severe and complicated course, and there remains a 5% overall mortality.[13]

Pathophysiology

Acute Pancreatitis. There are a number of commonly identified precipitants of acute pancreatitis. Gallstones and alcohol abuse account for nearly 80% of cases. Despite a century of research, however, the pathogenesis of acute pancreatitis remains a matter of debate. Mechanical obstruction of the pancreatic duct or the ampulla of Vater by a biliary stone is thought to induce pancreatitis through ductal hypertension and subsequent bile reflux into the pancreatic duct.[14,15] Similarly, edema at the ampulla of Vater due to a passed stone may produce pancreatic ductal hypertension. Alcohol is thought to be a direct cellular toxin, leading to intracellular accumulation of pancreatic enzymes and decreased bicarbonate and trypsin inhibitor concentrations. This enhances the formation of protein plugs and pancreatic outflow obstruction.

Regardless of the initiating force, premature activation of pancreatic enzymes is the central event in the pathogenesis of acute pancreatitis.[16] The cellular mechanisms responsible for intrapancreatic activation of digestive enzymes, particularly trypsin, are unclear. Once activated, trypsin activates many other proteolytic and vasoactive enzymes, including elastase, phospholipase A_2, and chymotrypsin.[4] Release of these proteolytic enzymes from acinar cells leads to acute inflammation of the pancreatic parenchyma and surrounding tissues. This early phase, known as acute edematous pancreatitis, involves peripancreatic fat necrosis and interstitial edema. Trypsin also activates enzyme cascades such as coagulation, complement, kallikrien, and fibrinolysis.[1] This may lead to further autodigestion of pancreatic tissue or to coagulation necrosis of the gland and the surrounding fatty tissue. In this more severe form of pancreatitis, known as acute hemorrhagic or necrotizing pancreatitis, there is parenchymal necrosis, hemorrhage, and impairment of endocrine and exocrine function.[17]

Local inflammation of the pancreas can lead to systemic circulation of pancreatic enzymes and inflammatory mediators such as platelet activating factor, tumor necrosis factor, and other cytokines. As a result, patients with severe acute pancreatitis may develop a systemic inflammatory response syndrome (SIRS) marked by renal failure, acute respiratory distress syndrome (ARDS), pleural effusions, and myocardial depression.[1] Further complications from vasoactive substances include systemic vasodilation, increased vascular permeability, and disseminated intravascular coagulation.

Bacterial infection occurs in 30-70% of cases of acute necrotizing pancreatitis and likely is due to translocation of enteric bacteria through a compromised gastrointestinal barrier.[18] Local and systemic infection is a major source of mortality in pancreatitis.[19]

Chronic Pancreatitis. Unlike acute pancreatitis, where the gland returns to normal histologic and biochemical function between attacks, chronic pancreatitis is characterized by irreversible morphological change and progressive loss of function. While alcohol is the most common and most widely studied etiology of chronic pancreatitis, approximately 30% of cases are idiopathic and numerous other rare causes exist. The reason that fewer than 10% of patients with alcoholism develop chronic pancreatitis is unknown.

Ingestion of alcohol in large quantities over a prolonged period causes increased protein secretion into pancreatic ducts and decreased fluid and bicarbonate production from ductal epithelial

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cells. Protein plugs and viscous fluid leads to progressive ductal obstruction, chronic inflammation, cellular atrophy, calcium deposition, and fibrosis.[20] These progressive and irreversible changes cause a range of clinical findings, including abdominal pain, steatorrhea, malabsorption, and diabetes.[2]

Clinical Presentation

Acute Pancreatitis. The hallmark of acute pancreatitis is severe, unremitting, deep abdominal pain. Pain is present at the onset and is the typical reason for seeking care. It usually is severe enough for patients to seek care directly at the ED. The pain classically is described as steady, located in the upper abdomen, and worse when supine. The exact location is variable, although it is epigastric more often than left-sided. Band-like radiation to the back is present in approximately half of cases.[21] Patients with acute pancreatitis can present with peritonitis. There are rare reports of cases presenting without pain (5%) although this is not the authors' experience.[22]

Alcoholic pancreatitis tends to begin six hours to three days after a binge or cessation of drinking. Gallstone pancreatitis can be preceded by biliary colic and should be suspected in patients who present with abdominal pain after a large or fatty meal. The pain usually lasts longer than the pain of biliary colic, which is limited to 6-8 hours.

Nausea and vomiting usually are present in patients with acute pancreatitis. These symptoms can continue for hours, and patients often are described as restless, agitated, and requiring large amounts of analgesia to control their symptoms. Dyspnea may occur as pulmonary complications (i.e., pleural effusion or ARDS) develop.

The physical examination in pancreatitis is variable, depending on the severity and time of presentation. The authors found no studies evaluating the utility of the physical exam in diagnosing acute or chronic pancreatitis. Fever and tachycardia can be present in moderate cases that do not have acute necrotizing pancreatitis or in severe cases with shock. Progression to shock, with hypotension, tachypnea, and respiratory failure, is a hallmark of severe necrotizing pancreatitis.

Abdominal tenderness often is epigastric and initially may be minimal, as the pancreas is a retroperitoneal organ. The retroperitoneal location may explain, in part, why patients often describe their pain as deep. In more advanced cases, peritoneal signs can be present even without infection, as the release of pancreatic enzymes directly irritates the peritoneum. Abdominal distension develops as fluid leaks into the retroperitoneum. The intra-abdominal irritation also frequently results in a small bowel ileus with diminished bowel sounds. Grey-Turner's sign is ecchymosis in the flank; Cullen's sign is periumbilical ecchymosis. These classic signs rarely are present (1% of cases), reflect intra-abdominal hemorrhage, and herald severe disease with poor prognosis.[22]

Respiratory signs and symptoms are common in acute pancreatitis. Patients may exhibit tachypnea and splinting due to diaphragmatic irritation even before pulmonary complications develop. If the patient is in true respiratory distress, it may represent ARDS, the most severe complication of pancreatitis. If the disease has progressed to acute necrotizing pancreatitis, shock or coma may occur. Examination may reveal signs of the systemic complications of acute necrotizing pancreatitis: sepsis, disseminated intravascular coagulation (DIC), and ARDS.

Other rare physical findings that may help to identify the etiology of pancreatitis include parotid swelling associated with mumps, hepatomegaly in alcoholic patients, and xanthomas in hyperlipidemic patients.

Later in the course of the disease, complications may become evident on physical examination. An abdominal mass representing a pseudocyst may become palpable. Panniculitis representing subcutaneous fat necrosis can occur. These are tender, erythematous nodules 1-2 cm in size, usually in the distal extremities. Thrombophlebitis in the legs can develop.

Chronic Pancreatitis. The major clinical features of chronic pancreatitis are abdominal pain and symptoms associated with pancreatic exocrine dysfunction. Abdominal pain is the usual reason for seeking care in the ED. The pain classically is described as severe, dull, and radiating to the back, but its history is variable.[23] In one prospective cohort of 207 patients with chronic pancreatitis, two usual patterns emerged. One group had brief painful episodes (usually fewer than 10 days) separated by pain-free periods lasting from months to more than a year. The second group was characterized by long periods with daily pain necessitating repeated hospitalizations.[24] Patients frequently avoid eating, as it may precipitate their pain. Nausea and vomiting frequently are associated with painful attacks.

Abdominal pain may be absent. In one retrospective study, 20% of patients with chronic pancreatitis initially presented to their doctors with symptoms of pancreatic dysfunction without pain.[25] It is likely that the percentage of patients who seek care in the ED solely with symptoms of painless pancreatic dysfunction is substantially less.

Patients with pancreatic insufficiency present with steatorrhea, protein deficiency, or glucose intolerance. Symptoms associated with pancreatic insufficiency usually occur later in the disease, as more than 90% of pancreatic function must be lost before it becomes clinically apparent.[26] Steatorrhea tends to occur prior to protein deficiency, as lipolysis usually is lost before proteolysis. Clinical complaints of steatorrhea are foul-smelling, loose, and greasy stools that are difficult to flush away. Glucose intolerance occurs frequently in the course of chronic pancreatitis, but true diabetes mellitus is a late complication. The destruction of pancreatic function leaves patients without insulin or its counteregulatory hormone glucagon. These patients require insulin, although ketoacidosis is rare. They are "brittle" diabetics who are at risk for hypoglycemia due to inability to produce glucagon. This combination especially is dangerous if patients continue to drink alcohol, with hypoglycemic coma a known cause of death.

The physical examination in chronic pancreatitis is nonspecific. Patients generally appear well-nourished, but in chronic alcoholic patients with advanced disease, weight loss and malnutrition may be apparent, or one may see signs of coexistent chronic alcoholic liver disease. Abdominal tenderness usually is upper abdominal and non-peritoneal, but no clear pattern exists. The findings of an abdominal mass, representing a

Table 1. Causes of Acute Pancreatitis

OBSTRUCTION
- Gallstones (40%)
- Biliary sludge
- Ampullary or pancreatic tumors
- Foreign body or worm-obstructing papilla
- Choledochocele
- Hypertensive sphincter of Oddi

TOXINS
- Ethanol (40%)
- Drugs (2-5%) (*see Table 2 for full list*)
- Methanol
- Organophosphate insecticides
- Scorpion venom

TRAUMA
- Penetrating more common than blunt
- Iatrogenic injuries: ERCP (5%) or intraoperative

METABOLIC ABNORMALITIES
- Hypertriglyceridemia (1-2%)
- Hypercalcemia

INHERITED CONDITIONS
- Cystic fibrosis
- Genetic mutations

INFECTIOUS
- Viral (mumps, coxsackie B, Epstein-Barr, cytomegalovirus, rubella, hepatitis)
- Bacterial (mycoplasma, *M. tuberculosis*, *M. avium* complex, Leptospira, Legionella),
- Parasitic (ascaris, clonorchiasis, toxoplasma), fungal aspergillus)

VASCULAR ABNORMALITIES
- Hypoperfusion (intraoperative or due to hemorrhagic shock)
- Atherosclerotic emboli
- Vasculitis (SLE, PAN)

MISCELLANEOUS CONDITIONS
- Perforating peptic ulcer
- Hypothermia
- Reye's syndrome

IDIOPATHIC (10%)
- Many of the cases previously labeled idiopathic now are diagnosed using ERCP. The most frequent diagnosis is biliary sludge.

Note: For a full discussion with references, see reference 22. Percentage of cases is approximate, and is rare if not noted.

Key
ERCP = endoscopic retrograde cholangiopancreatography;
SLE = systemic lupus erythematosus; PAN = polyarteritis nodosa

pseudocyst, or a palpable spleen from splenic vein thrombosis are rare.

Etiologies

Acute Pancreatitis. Many causes of acute pancreatitis have been reported, but a few causes account for most cases. (*See Table 1.*) The two most frequent causes of acute pancreatitis in the United States are gallstones and alcohol abuse. Together these account for approximately 80% of all cases of acute pancreatitis.[27] Exact percentages depend on the patient population. Gallstones clearly have been shown to cause acute pancreatitis, although the mechanism is debated. Approximately 35% of cases of acute pancreatitis are caused by gallstones, but only 3-7% of patients with gallstones ever develop acute pancreatitis.[28,29] The cause and effect relationship has been proven, as cholecystectomy has been shown to prevent gallstone-induced pancreatitis.[29]

Alcoholic pancreatitis develops in drinkers with a history of prolonged, heavy alcohol consumption (at least 5-10 years), not after an occasional binge in a patient without a drinking history.[27,30] About 10% of chronic alcoholics will develop attacks of acute pancreatitis that are clinically indistinguishable from other etiologies.

Many drugs have been reported to cause acute pancreatitis. Although there are numerous case reports showing association, causality is difficult to prove. Overall, the incidence of drug-induced pancreatitis is low, as illustrated by a comprehensive German report of pancreatitis cases from 45 centers. Only 22 of 1613 cases (1.4%) were related to medication use.[31] Three recent review articles have evaluated the case reports.[32-34] Although systems of classifying a medication's association to pancreatitis have been developed (classifying a medication's association to pancreatitis as definite, probable, or possible) the data behind the classifications are variable, leading reviewers to disagree about many drugs. Table 2 lists drugs strongly associated with pancreatitis in at least two of the three recent reviews of this subject. A recent analysis indicates that newer antipsychotic drugs may have a greater likelihood of causing pancreatitis than older agents.[35] Textbooks, review articles, or on-line databases can be consulted for extensive lists of drugs with possible associations.

Pathogenesis of drug-induced pancreatitis is variable. In some cases it is due to a direct toxic effect (sulfonamides), in other cases it is due to an allergic reaction (6-mercaptopurine), while in the case of angiotensin-converting enzyme inhibitors, it is thought to be due to angioedema of the pancreas. Prognosis is excellent if the cause is identified and removed, with case series reporting lower than average rates of necrotizing pancreatitis and mortality.[31]

Traumatic pancreatitis can be divided into idiopathic and non-idiopathic cases. As the number of endoscopic retrograde cholangiopancreatography (ERCP) procedures has increased during the past 30 years, the percentage of pancreatitis cases related to ERCP has risen to 3-5%. The risk is higher when the procedure involves therapy (i.e., sphincterotomy or stents) and can occur in up to 25% of cases when sphincter of Oddi manometry is performed.[36] Post-operative pancreatitis is seen most frequently after liver or renal transplantation and coronary

Table 2. Drugs Strongly Associated with Acute Pancreatitis[32-34,121]

CLASS	DRUG
ACE-inhibitors	Lisinopril
Anti-inflammatory drugs	Sulindac, salicylates
Antimicrobial agents	Metronidazole, stibogluconate, sulfonamides, tetracycline
Diuretics	Furosemide, thiazides
Drugs used for inflammatory bowel disease	Sulfasalazine, 5-ASA
HIV and AIDS therapy	Didanosine (ddI), pentamidine
HMG-CoA reductase inhibitors	Simvastatin
Immunosuppressive agents	L-asparaginase, azathioprine, 6-mercaptopurine
Neuropsychiatric agents	Valproic acid
Others	Calcium, estrogen, tamoxifen

Table 3. Differential Diagnosis of Acute Pancreatitis

- Biliary colic or acute cholecystitis
- Renal disease (especially renal colic)
- Peptic ulcer disease
- Lung disease (pleurisy or pneumonia)
- Acute liver conditions
- Cardiac ischemia/inferior MI
- Closed loop bowel obstruction
- Dissecting aortic aneurysm
- Perforated hollow viscous
- Ectopic pregnancy
- Ischemic bowel
- Connective tissue disorders with vasculitis

MI = myocardial infarction

artery bypass grafting. The prognosis in post-operative pancreatitis is worse than with other causes.[37] Blunt and penetrating trauma both can cause pancreatitis, although it is a rare complication. A nine-year review of 16,188 trauma cases at a Level 1 trauma center showed a 0.4% incidence of pancreatic trauma, of which two-thirds were due to penetrating trauma, and almost all were diagnosed at laparotomy.[38]

Up to 30% of patients with pancreatitis will have no identifiable cause by history, laboratory tests, and ultrasound. Historically, 10% of acute pancreatitis cases were designated idiopathic after the workup. More recently, patients with idiopathic pancreatitis have undergone more extensive workups, including ERCP. These often reveal a treatable cause. One study showed that a presumed cause was found in 100 of 126 patients with idiopathic pancreatitis, and therapy was possible for a significant number of these patients.[39] One frequently identified cause is biliary sludge, seen in 66-74% of these patients. Sludge is a viscous suspension in gallbladder bile that can contain small stones and usually is asymptomatic. Although no prospective studies have shown that treatment is curative, several retrospective studies suggest that cholecystectomy and papillotomy reduce future attacks.[40,41] It is debated whether all patients with a first attack of pancreatitis and no identifiable cause should undergo extensive testing (including ERCP), as only a small percentage will develop another case of pancreatitis in the next three years.[42]

Both hypertriglyceridemia and hypercalcemia are uncommon but significant causes of pancreatitis. Infection frequently has been reported in association with pancreatitis, but, like drug-induced pancreatitis, the data for causality are weak with many of the reported organisms. Pregnancy is not a cause of pancreatitis, as shown in a 10-year case control study that failed to show an increased incidence in pregnant women compared to age-matched controls.[43]

While the etiology of pancreatitis is similar in children and adults, the overall incidence is lower in children. Blunt trauma and infection (such as mumps and other viral illnesses especially), congenital anomalies, and multi-system disease are the leading causes, with many cases ultimately classified as idiopathic.[27]

Chronic Pancreatitis. Compared to acute pancreatitis, there are fewer causes of chronic pancreatitis. The majority of cases are due to alcohol abuse (70-80%), with the risk related to duration and amount consumed.[44] There are other important factors in the development of chronic pancreatitis in alcoholics, as only 5-10% of alcoholics will develop chronic pancreatitis.[45] Ductal obstructions, hereditary pancreatitis, systemic diseases (e.g., systemic lupus erythematosus [SLE] or cystic fibrosis), and tropical pancreatitis are other major causes of chronic pancreatitis.

Differential Diagnosis

Pancreatitis must be differentiated from the large number of intra-abdominal, pulmonary, and cardiac processes that can present with acute abdominal pain, nausea, and vomiting. (*See Table 3.*) Historical features are most useful in prioritizing the differential diagnoses, as many patients will describe a history of heavy alcohol use or of gallstones, which together cause 80% of acute pancreatitis cases. The severity and length of the attack will help further differentiate these potential diagnoses. If the patient presents late in the course of pancreatitis, it may be easier to clinically exclude other processes, as acute pancreatitis can cause constant abdominal pain for days without relief or progression, unlike many other conditions.

Patients with a history of chronic pancreatitis may present with symptoms similar to previous flares and self-diagnose an exacerbation, but a thorough assessment usually is indicated.

Diagnostic Workup

The diagnosis of pancreatitis usually is made by history with confirmation by laboratory testing. Radiography is helpful for determining etiology and prognosis, but rarely is the sole means of diagnosis.

Laboratory. Many biochemical tests have been studied in their ability to diagnose and prognosticate pancreatitis. The tests that most frequently are used in the ED include amylase, lipase, transaminases, and non-specific markers of inflammation (i.e., C-reactive protein [CRP] and erythrocyte sedimentation rate [ESR]). As no perfect test to diagnose pancreatitis exists, emer-

gency physicians must know the properties of each test and be able to apply the best available test.

Studies evaluating laboratory tests for pancreatitis have several weaknesses that have clouded debate regarding what is the ideal test. First, there is no clear gold standard for diagnosing pancreatitis. Early studies often used the laboratory test in question (usually amylase) as the sole marker or as one of the diagnostic criteria of pancreatitis.[46-48] This artificially inflates the sensitivity of the test in question. Better constructed studies use a number of factors, including lab tests, computed tomography (CT) results, surgical findings, and discharge diagnoses, to define the population with pancreatitis. Second, the exact lab techniques used have varied between studies. Third, "normal limits" are difficult to define. They usually are defined in a sample of healthy young men that may be very different from the population at risk for pancreatitis. Fourth, sensitivity and specificity depend on the prevalence of pancreatitis in the study population and on the cut-off level chosen. This said, there are several well-conducted studies that can guide the emergency physician's choice of diagnostic tests.

Lipases are enzymes secreted from pancreatic acinar cells that hydrolyze triglycerides into metabolic substrates. Normally, 99% is secreted into the pancreatic ductal system, while less than 1% makes its way into the serum through lymphatics and capillaries.[49] This gives lipase the theoretical advantage of being more specific than amylase because it has less tissue distribution. Serum lipase increases within 4-8 hours of acute pancreatitis, peaks at around 24 hours, and decreases within 1-2 weeks.[49] Traditionally, serum lipase was a difficult test to perform rapidly and accurately, was more costly, and was not widely available. This has been overcome with development of a radioimmunoassay for lipase and inclusion of co-lipase in commercially available kits.[50,51] Lipase assays now are comparable to amylase in terms of cost, access, and speed.[50] Sensitivity is quoted at between 85% and 100%, and specificity is reported at between 60% and 100%.[52] The test characteristics depend on where the cut-off for a positive is set. At five times the upper limit of normal (ULN), the specificity of lipase was 100%, but sacrificed sensitivity to only 60%.[53,54] Other conditions reported in association with elevated lipase include acute cholecystitis, bowel obstruction or infarction, duodenal ulcer, and diabetic ketoacidosis.[52]

Amylase is a small enzyme that cleaves starches into smaller carbohydrates. There are two major sources of amylase, the pancreas and salivary glands, and several minor sources. Although there are two different isoenzymes of amylase, they both are picked up by common techniques.[55] Newer tests are being developed to test for amylase isoenzymes, but these are only in preliminary stages. There are many physiologic and pathologic causes of amylase elevations. (*See Table 4.*) The varied sources of hyperamylasemia give amylase a poor specificity, which is its major limitation. As a small protein, amylase is cleared renally and has a half-life of approximately two hours.[55] In patients with acute pancreatitis, amylase rises within 6-24 hours, peaks at 48 hours, and normalizes in 5-7 days.[46] Amylase levels normalize more rapidly than lipase levels. Many studies have shown that amylase can be normal in between 19% and 32% of patients with acute pancreatitis.[52] This is most likely in alcoholic pancreatitis and acute flares of chronic pancreatitis.[56,57] Reported sensitivities for amylase range between 60% and 98%, with specificities between 40% (ULN as cut-off) and 70-100% (five times ULN used as cut-off). By raising the cut-off value, specificity is gained at the expense of sensitivity.

The question for emergency physicians is which is the superior test to diagnose acute pancreatitis. Although there are theoretical limitations to each test, the studies that have compared amylase and lipase in patients with acute pancreatitis all conclude that lipase is better or equal. One study retrospectively looked at 352 cases that had laboratory tests in the ED and a CT-verified diagnosis of pancreatitis. They found that lipase was more sensitive: 19% had normal amylase levels, and of these two-thirds had elevated lipase.[58] Another study compared a cut-off of three times the ULN in 306 adults with abdominal pain of whom 48 had pancreatitis. Amylase was 54% sensitive while lipase was 77% sensitive.[59] A third study compared enzymes in two studies. In the first, they used a cut-off of three times ULN and found lipase to have a sensitivity of 100% and specificity of 99%, while amylase

Table 4. Causes of Increased Serum Amylase Activity[55]

PANCREATIC DISEASES
• Acute pancreatitis
• Complications of pancreatitis
• Acute exacerbation of chronic pancreatitis
• Pancreatic tumors, cysts
OTHER INTRA-ABDOMINAL PROCESSES
• Acute cholecystitis
• Common bile duct obstruction
• Intestinal ischemia or infarction
• Intestinal obstruction or perforation
• Acute appendicitis
• Acute gynecologic conditions such as ruptured ectopic pregnancy and acute salpingitis
DISEASES OF SALIVARY GLANDS
• Mumps
• Effects of alcohol
TUMORS
• Ovarian cysts or papillary cystadenocarcinoma
• Lung carcinoma
RENAL INSUFFICIENCY
MACROAMYLASEMIA
MISCELLANEOUS
• Endoscopy
• Sphincter of Oddi stenosis or spasm
• Anorexia nervosa
• Head trauma with intracranial bleeding
• Diabetic ketoacidosis

was 75% sensitive and 99% specific.[60] In a second study comparing alcoholics with and without pancreatitis, amylase performed poorly, with a sensitivity of 45%, while lipase was 100% sensitive.[56] Another study reported 57 episodes of acute pancreatitis and found amylase to have a sensitivity of 79% and lipase 96%, while both tests had similar specificities greater than 95%.[57]

Other investigators have looked into whether combinations of tests improve diagnostic accuracy. A few individual studies have recommended use of both amylase and lipase. Others have recommended use of a lipase-to-amylase ratio greater than 2 to suggest an alcoholic etiology to acute pancreatitis.[61] Larger studies and meta-analyses conclude that neither of these strategies is more accurate than the use of lipase alone.[52]

A single prospective study evaluated a urinary screening dipstick for trypsinogen-2 in 447 ED patients with acute abdominal pain. The test showed a sensitivity of 94%, picking up 50 of 53 patients with acute pancreatitis, and all seven patients who had severe pancreatitis. It was 96% specific. It was more accurate than serum amylase, but serum lipase results were not reported.[62] This test currently is not commercially available or in widespread use in the United States.

A recent meta-analysis has shown that an ALT (alanine aminotransferase) concentration greater than 150 IU/L is 96% specific for gallstone pancreatitis, but only 48% sensitive. Therefore, a value greater than 150 IU/L strongly suggests gallstones as the cause, but a lower value does not rule it out.[63]

Serum tests have been recommended as part of the workup for traumatic pancreatic injury in selected patients in the Advanced Trauma Life Support guidelines. Recent studies reveal that serum tests are neither sensitive nor specific for pancreatic injury in the setting of trauma.[38,64,65]

Therefore, when evaluating patients in whom there is a clinical suspicion for acute pancreatitis, lipase should be used as the initial serum test. If lipase is not available, amylase should be used. The use of serum tests for pancreatitis in abdominal trauma should be clinically directed, not routine. For a comprehensive review of amylase and lipase utilization, see the review by Vissers et al.[52]

Radiography

Radiography is not highly sensitive for acute pancreatitis in the ED, so it should play an adjunctive role to clinical examination and laboratory testing.

X-rays often are ordered in patients with acute pancreatitis to evaluate for other potential diagnoses. Abdominal flat and upright and chest radiographs are most common. The abdominal series may show nothing, a focal ileus of a small intestine segment ("sentinel loop"), the colon cut-off sign, gallstones, or pancreatic calcifications of chronic pancreatitis. The chest film may show a pleural effusion, atelectasis, or signs of ARDS. Most plain films in patients with acute pancreatitis will have abnormalities, but the findings are nonspecific.[66]

Ultrasonography is only 67% sensitive for acute pancreatitis when gallstones are the cause, and is less sensitive for other causes.[67] Ultrasound diagnosis of pancreatitis often is limited by operator technique and the inability to visualize the pancreas due to its retroperitoneal location and overlying intestinal gas. In addition, ultrasound availability may be limited. These factors limit the use of ultrasound in the ED. There is an important role for ultrasound in determining the etiology of acute pancreatitis, although this is not an emergent concern.

There are three indications for CT scan in acute pancreatitis: first, to exclude other serious intra-abdominal conditions, such as a posterior perforated ulcer, renal colic, or a dissecting aortic aneurysm; second, to stage the severity of acute pancreatitis; and third, to look for complications such as pancreatic pseudocyst, hemorrhage, or necrosis of other adjacent organs.[68,69] Dynamic, spiral CT with oral and intravenous contrast is the test of choice, as this increases the sensitivity of prognostic pancreatic findings and does not damage the pancreas.[70,71] Pancreatic necrosis may not appear until 48-72 hours after the onset of the disease, resulting in CT scan sensitivity as low as 70% for acute pancreatitis.[69] The CT scoring system (*see Table 5*) developed by Balthazar is useful for prognosis, as grades A-C suffer low morbidity and mortality, while grades D and E have mortality up to 15%.[72,73] Balthazar recommends waiting three days for the initial abdominal CT if the patient is not showing signs of serious pancreatitis (i.e., fever, peritonitis, or shock).[69]

Magnetic resonance cholangiopancreatography (MRCP) is better able to visualize intrahepatic and intraductal stones compared to all other diagnostic techniques and, thus, has a role in defining etiology in acute pancreatitis.[74,75] Pancreas MRI now is being studied for prognosis. If available, this technique could determine prognosis, presence of gallstones, and complications from biliary tract disease. These techniques currently have a limited role in the ED.

ERCP is useful in determining the etiology of acute pancreatitis, pancreatic and biliary anatomy, and the presence of common bile duct stones. It also is useful therapeutically for removal of

Table 5. CT Severity Index[73]

GRADES OF INFLAMMATION	POINTS
A: Normal pancreas consistent with mild pancreatitis	0
B: Focal or diffuse enlargement of the gland, including contour irregularities and inhomogeneous attenuation but without peripancreatic inflammation	1
C: Grade B plus peripancreatic inflammation	2
D: Grade C plus associated single fluid collection	3
E: Grade C plus ≥ 2 peripancreatic fluid collections or gas in the pancreas or retroperitoneum	4
DEGREE OF NECROSIS	
None	0
< 33 %	2
33-50%	4
> 50%	6

Reprinted with permission from: Balthazar EJ, Robinson DL, Megibow AJ, et al. Acute pancreatitis: Value of CT in establishing prognosis. *Radiology* 1990;174:331-336.

common bile duct stones and sphincter of Oddi papillotomy pancreatitis. It is beyond the scope of ED evaluation.

The authors recommend the following for the use of radiography in patients with acute pancreatitis: Plain abdominal films should be obtained as needed to evaluate for other potential causes of abdominal pain and vomiting (i.e., perforation or bowel obstruction); CT scan should be performed in the ED in cases of severe acute pancreatitis (e.g., peritonitis, shock, or ARDS) or when complications are suspected (i.e., necrotizing pancreatitis, abscess, or pseudocyst); other radiographic testing can be done as an inpatient; and notably, all patients with pancreatitis should receive an abdominal ultrasound within one day of admission to determine if they have gallstones.

Management

Acute Pancreatitis. Despite numerous studies attempting to validate specific treatments, the key to the successful management of pancreatitis remains aggressive supportive therapy. In mild pancreatitis, this may involve only pain control and intravenous fluids, while in severe pancreatitis, patients may require intensive care unit (ICU) level of care, invasive monitoring, dialysis, and even mechanical ventilation.

All patients should receive adequate fluid resuscitation. Significant dehydration may occur as a result of third spacing of intravascular fluid due to the systemic inflammatory response and retroperitoneal sequestration of pancreatic secretions. Given the potential for significant fluid losses and shifts, a Foley catheter should be placed to monitor urine output in those patients requiring resuscitation. Urine output should be maintained at 0.5-1 cc/kg/hr. Patients should be resuscitated with normal saline or lactated Ringer's as needed to maintain systolic blood pressure (SBP) greater than 100 mmHg and heart rate (HR) less than 100. Five to six liters of fluid may be required initially. With hydration, patients are likely to develop electrolyte imbalances. In particular, sodium, potassium, magnesium, and calcium levels should be monitored regularly and repleted as necessary.

Pain control is fundamental in treating pancreatitis. Historically, meperidine was cited as the narcotic of choice because it was believed to cause less sphincter of Oddi spasm.[76] However, multiple literature reviews have failed to support this theory.[77,78] One study found no evidence that meperidine is superior to morphine in relieving pain and concluded that all narcotics increase biliary pressure and interfere with peristalsis of the sphincter of Oddi.[76] In fact, morphine may be of greater benefit because it provides longer pain relief with less risk of seizures.[77,78] Thus, any narcotic can be used safely for analgesia and should be titrated to patient comfort.

Traditionally, nasogastric suction had been an integral part of treatment because it was felt that eliminating gastric secretions and decreasing the acidity of secretions delivered to the duodenum would prevent stimulation of the pancreas. However, recent studies have shown that nasogastric suctioning does not improve pain relief, shorten hospital stay, or shorten time to onset of feeding.[79,80] It should be used only in patients with ileus or uncontrollable vomiting.

Initially, patients with mild pancreatitis should be kept on bowel rest or small amounts of clear liquids or ice chips, if tolerated, to decrease the stimulation of the pancreas during recovery. As the pain resolves, oral intake can be reinstituted slowly. However, patients with severe pancreatitis or those unable to take food orally for a prolonged period of time secondary to pain or vomiting will require supplemental nutrition. Multiple medical therapies have been studied for treatment of acute pancreatitis. H_2 antagonists, specifically cimetidine, gained popularity through their ability to decrease gastric acidity, which theoretically could prevent stimulation of the pancreas. However, studies have not shown their use to be of any benefit.[80,81] Other medications, including atropine, calcitonin, and somatostatin, also have failed to show a significant benefit in the treatment of pancreatitis.[82-84] More recently, the protease inhibitors gabexate mesilate and aprotinin were studied; however, neither medication has been shown to improve clinical outcomes. Lexipafant, a platelet activating factor antagonist, recently has been proposed as a potential therapy to attenuate the development of multi-organ failure. Results have been conflicting. None of the above medications consistently have been shown to improve morbidity and mortality in pancreatitis and their routine use is not recommended.

Peritoneal lavage has been employed with the thought of removing toxic pancreatic exudates, but studies have failed to support this theory. No studies have found improved mortality; therefore, peritoneal lavage is not recommended.

Superinfection is a major cause of mortality in pancreatitis. It is thought to occur secondary to translocation of bacteria from the gastrointestinal tract. The use of antibiotic prophylaxis in mild pancreatitis does not appear to improve mortality based on early controlled trials, although these studies evaluated ampicillin, which does not adequately penetrate pancreatic tissue.[85,86] Several meta-analyses have concluded that the early prophylactic use of antibiotics in acute severe necrotizing pancreatitis decreases the incidence of sepsis and mortality.[87,88] Other studies have shown that imipenem-cilastatin reduces the incidence of infection in severe pancreatitis, but does not significantly affect overall mortality.[89,90] Studies comparing various antibiotics have recommended the use of impenem over fluoroquinolones.[91,92] Currently, it is recommended that patients with acute necrotizing pancreatitis receive imipenem-cilastatin for 2-4 weeks, although any broad-spectrum antibiotic covering both aerobic and anaerobic bacteria should be of benefit given the lack of direct antibiotic comparisons in controlled studies.[87,92]

The use of selective decontamination of the gut to prevent bacterial translocation also has been studied with mixed results. One study found that oral administration of colistin sulfate, amphotericin, and norfloxacin significantly improved morbidity and mortality in patients with severe necrotizing pancreatitis as compared to controls.[93] Another study, however, found that giving oral polymyxin B, amikacin, and amphotericin B did not significantly improve survival.[94] The routine use of selective decontamination is not advocated.

The use of ERCP in gallstone-induced pancreatitis also has been studied with encouraging results. Several studies have

shown a decreased morbidity, including a reduction in the risk of biliary sepsis, in patients with both mild and severe gallstone pancreatitis who had gallstones removed urgently within 24-72 hours by ERCP with sphincterotomy.[95,96] Recently, it has been suggested that ERCP be performed only in patients with clear evidence of obstruction by increased bilirubin or signs of cholangitis because there may be a risk of causing infection in otherwise sterile pancreatitis.[92,97]

The use of early biliary surgery as a potential treatment of gallstone pancreatitis also has had controversial results. Some have found no difference between early and delayed surgery.[97] Others have found a higher mortality and morbidity in patients who had surgery within 48 hours vs. those who had surgery delayed after 48 hours, especially in patients with severe pancreatitis.[98,99] The timing of biliary surgery currently is decided based on patient stability and surgeon preference.

Patients with infected necrotic pancreatitis as documented by CT-guided percutaneous aspiration should undergo surgical debridement with drainage.[100-102,106] Patients with sterile necrotic pancreatitis initially should be managed medically, as studies have failed to show improvement in morbidity with surgical debridement, but debridement should be performed if symptoms persist after 4-6 weeks.[102,103]

Chronic Pancreatitis. The treatment of chronic pancreatitis includes pain control and avoidance of inciting factors such as alcohol. Patients with chronic pancreatitis often will need to be maintained on long-acting oral analgesics for a significant period of time and will have exacerbations that may require hospital admission for parenteral analgesia. Narcotic dependence often becomes an issue, and patients may benefit from participation in a pain clinic or other pain management program.

Patients with chronic pancreatitis and unremitting pain should be worked up for complications with a CT scan. ERCP is useful in some patients to assess for pancreatic strictures and obstructions. Dilation of strictures or stent placement can improve pain and reduce frequency of attacks. Surgical resection of the pancreas, such as the Whipple procedure, may be helpful in certain patients with uncontrollable pain by removing the damaged parenchyma and relieving duodenal and biliary obstruction. Drainage of pseudocysts can relieve pain, as well. Celiac plexus nerve block also has been attempted as a last resort, but side effects prevent the routine use of this therapy.[104] Despite numerous efforts, no therapy consistently has shown a benefit in chronic pancreatitis.

Complications

Almost 25% of patients with pancreatitis develop complications, with an overall mortality of about 5-10%.[4,102,105] Pancreatic infection accounts for 70-80% of deaths.[4] Complications occur both locally in the pancreas and systemically.

In the first week of hospitalization, systemic effects may include multisystem organ failure involving the cardiovascular, pulmonary, and renal systems. Cardiovascular collapse can occur secondary to the SIRS and from bleeding and exudation of plasma into the retroperitoneum. Myocardial infarction can occur from hypotension and increased demand on the heart from the acute inflammatory process. Respiratory complications range from mild atelectasis to pleural effusions to severe ARDS requiring mechanical ventilation. Renal complications include acute renal failure secondary to hypotension and subsequent acute tubular necrosis that may require dialysis until renal function improves. Gastrointestinal complications include bleeding secondary to stress ulcerations, gastric varices from splenic vein thrombosis, or rupture of pancreatic pseudoaneurysms.[4,111,116]

Locally, necrosis of the pancreas can occur as early as the first week of illness. There is damage to the pancreatic capillary parenchymal network, acinar cells, islet cells, and the pancreatic ductal system. Between 10% and 20% of patients with acute pancreatitis develop pancreatic necrosis, with a mortality of 15-20%.[2,104] Approximately 30-35% of patients with pancreatic necrosis develop infection, typically in the second or third week of illness.[104] It is difficult to distinguish sterile from infected necrosis, as both can present with similar clinical and laboratory findings (i.e., fever or leukocytosis). If the question arises, a guided percutaneous aspiration should be performed for Gram stain and culture.

Pancreatic pseudocysts are walled-off collections of pancreatic secretions without an epithelial lining that develop in 1-8% of patients with acute pancreatitis. They are responsible for 5-10% of all deaths in chronic pancreatitis.[4,104] Pseudocysts form after the third to fourth week of illness and last for more than four weeks. Up to 50% of pseudocysts resolve spontaneously, but when persistent, they can cause local effects such as pain, biliary or gastric obstruction, or they may erode into surrounding blood vessels, leading to hemorrhage. Pseudocysts also may rupture, leading to pancreatic ascites or the formation of pancreatic fistulas.[102,104,105]

Patients also can develop duodenal obstruction and biliary strictures. They will present with pain after eating, early satiety, nausea, vomiting, and abdominal distention.

Pancreatic fistulas to adjacent cavities, including bowel, skin, and biliary system, can develop if the pancreatic duct is disrupted. This complication should be suspected in patients who develop ascites, pleural effusions, or pericardial effusions. Diagnosis is confirmed by fistulograms for external fistulas or ERCP for internal fistulas. In most cases, surgery is required for definitive treatment.[4,104]

Abscesses also can develop near the pancreas when liquefied necrotic tissue is infected secondarily. They typically develop four or more weeks after the onset of symptoms and should be suspected in patients with persistent fevers, increasing white blood cell (WBC) count, and failure of improvement of symptoms. Diagnosis is made by CT scan with definitive treatment by percutaneous catheter drainage.[106]

Other rare complications include pancreatic encephalopathy, retinopathy, splenic rupture or hematoma, splenic vein thrombosis, pseudoaneurysms of adjacent arteries, and right kidney hydronephrosis and hydroureter.[4,104]

Prognosis

Various scoring systems integrating laboratory and clinical data have been developed to aid in predicting the severity of

Table 6. A Comparison of Ranson's and Glasgow Criteria

RANSON'S CRITERIA	GLASGOW (IMRIE) CRITERIA
At presentation:	***Over 48 hours:***
Age > 55 years	Age > 55 years*
WBC > 16,000/mm^3	WBC > 15,000/mm^3
Glucose > 200 mg/dL	Glucose > 180 mg/dL
LDH > 350 IU/L	LDH > 600 IU/L
AST > 250 IU/L**	AST > 200 IU/L**
	BUN > 96 mg/dL
	Ca < 8 g/dL
At 48 hours:	pO_2 < 60 mmHg
Hct decrease >10 mg/dL	Albumin < 3.2 g/dL
BUN increase > 5 mg/dL	
Ca < 8 mg/dL	
pO_2 < 60 mmHg	
Base deficit > 4 mEq/L	
Fluid sequestration > 6 L	

*1981 modification removed age as a criteria
**1984 modification replaced age and removed AST as a criteria
Key
WBC = white blood cell count; LDH = lactate dehydrogenase; AST = aspartate aminotransferase; BUN = blood urea nitrogen

pancreatitis and subsequent risk of mortality. While many different scoring systems have been developed and studied, the three most utilized systems are the Ranson, Glasgow, and Acute Physiology and Chronic Health Evaluation (APACHE) scores.

Ranson et al developed a set of 11 risk factors based on a retrospective analysis of patients with pancreatitis.[107] (*See Table 6.*) The five used on admission are: age older than 55 years, WBC count greater than 16,000/mm^3, glucose greater than 200 mg/dL, LDH greater than 350 IU/L, AST greater than 250 IU/L. These reflect the severity of the acute inflammatory process. The other six criteria, which are determined at 48 hours, include hematocrit decrease greater than 10, blood urea nitrogen (BUN) increase greater than 5 mg/dL, calcium less than 8 mg/dL, PO_2 less than 60 mmHg, base deficit greater than 4 mEq/L, and fluid sequestration greater than 6 L. These reflect the systemic effects of circulating enzymes on end organs.[102] Applying these criteria retrospectively, Ranson found that mortality was increased in those patients with a greater number of risk factors. Patients with fewer than three signs had a very small risk of death or complications. The risk markedly increased with more than three signs, with a mortality of greater than 50% when six or more signs were present.[30,102] However, one must take into consideration that these values were determined in an era prior to the aggressive resuscitation, antibiotics, and advanced critical care that is available presently. Thus, patients with greater than six Ranson criteria now routinely are surviving.[108]

The Glasgow or Imrie criteria were developed as a modification of the Ranson criteria by eliminating three of Ranson's criteria (fluid sequestration, base deficit, and hematocrit change) and adding albumin less than 3.2 as one of the criteria.[109] (*See Table 6.*) Imrie et al found that all patients in their study who died had three or more of the nine risk factors in the first 48 hours. A study in 1984 further modified the criteria to eight factors by eliminating AST, as the researchers found that it was not significant for predicting severe pancreatitis.[13]

The APACHE II criteria is a newer scoring system that is a simplified version of the APACHE criteria described by Knaus et al in 1981.[110,111] The score is based on a weighted index of 12 physiologic criteria that represent the degree of acute illness as well as a pre-admission health evaluation, which indicates the health status of the patient prior to the onset of acute illness.[102,110-112] The APACHE score has been shown to correlate with morbidity and mortality in patients with acute pancreatitis. One study found that no patients with a score greater than 20 survived.[113]

Contrast-enhanced CT also has been studied as a tool for establishing prognosis. Balthazar et al developed a CT severity index (*see Table 5*) based on a combination of peripancreatic inflammation, phlegmon, and degree of pancreatic necrosis (< 33%, 33-50%, > 50%). They found that patients with a high severity index (7-10 points) had a 92% morbidity and 17% mortality, while patients with a low CT severity index (0-2 points) had a 2% morbidity and 0% mortality.[73,102] Others have found that CT scan is useful for the evaluation of local complications, but that it is not a good predictor of systemic complications.[114]

Various serum markers also have been suggested for use as indicators of prognosis including elastase, carboxypeptidase-N, C-reactive protein, alpha-1 antitrypsin, alpha-2 macroglobulin, complement C3, and other markers of systemic inflammation.[115,116] While some of the serum markers appear promising, such as CRP and alpha-1 antitrypsin, further work needs to be done to confirm the reproducibility of previous findings. At this time, no single serum marker has been proven sufficient to predict prognosis.

Despite the many scoring systems and studies comparing these systems, the ideal system remains controversial. The APACHE II score, while more complicated to calculate, appears to be more useful because it can be calculated on admission to predict severity immediately and on a daily basis thereafter to monitor for improvement or further deterioration.[110,111,113,117] In addition, some have found it to be more sensitive and specific for determining risk of morbidity and mortality than other scoring systems.[113,114,117] Others, however, have not found it to be any more accurate.[108,118,119]

Critics of the Ranson criteria point out a lack of reproducibility in predicting severity in recent years. Its utility is questioned because of the biases present in the original study, including lack of confirmation of pancreatitis in all patients and absence of the final 11 criteria in all patients studied. A recent metaanalysis and comparison studies have supported these criticisms,[115,120] but others have found that the Ranson criteria remains a valid predictor in critically ill patients.[108,119]

None of the criteria described have been clearly proven to be better than the others. Utilizing one of the scoring systems is important to aid in predicting which patients may develop com-

plications and require ICU level of care, but should not be used alone in making an appropriate disposition. There is no set score in any scoring system above which a patient must be cared for in an ICU. Clinical judgment, aggressive treatment, and individual patient factors play an important role in determining outcome and disposition. These should be combined with the scoring system to determine disposition. Choosing the appropriate scale should be based on the physician's level of comfort with the scoring system, availability of CT scanning and various serum markers, and the rapidity with which laboratory results are available to the physician.

Disposition

Patients with acute pancreatitis secondary to a clear nonsurgical etiology who can hydrate orally and have minimal pain may be discharged home. They should be given narcotic analgesics, recommendations for a clear liquid diet, and instructions for follow-up in 24-48 hours. This represents only a minority of patients. Most patients with acute pancreatitis require admission for fluid hydration, parenteral analgesia, bowel rest, and monitoring for the development of complications. Patients with severe pancreatitis, as evidenced by hemodynamic instability or end organ dysfunction, should be admitted to an ICU for critical care monitoring and supportive treatment. A surgeon should be consulted for patients with necrotizing pancreatitis, pancreatic abscess, or gallstone pancreatitis. These patients may be candidates for a variey of interventions, including surgical debridement, percutaneous drainage, ERCP, or cholecystectomy.

Patients presenting with exacerbations of chronic pancreatitis may be discharged home if their pain is controlled with oral analgesics and they can tolerate oral intake. Decisions on appropriate analgesics should be made in conjunction with the patient's primary care physician. Outpatient follow-up should be arranged in 24-48 hours. Patients with severe pain, persistent vomiting, or evidence of dehydration should be admitted for intravenous analgesics and fluids.

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Physician CME Questions

181. What is the appropriate antibiotic for patients with severe necrotizing pancreatitis?
    A. Imipenem-cilastatin
    B. Ciprofloxacin
    C. Ampicillin
    D. Gentamicin
    E. Antibiotics are not indicated.

182. Which of the following medications has been shown in studies to reduce the incidence of infection in severe pancreatitis?

Emergency Medicine Reports
CME Objectives

To help physicians:

- quickly recognize or increase index of suspicion for specific conditions;
- understand the epidemiology, etiology, pathophysiology, and clinical features of the entity discussed;
- apply state-of-the-art diagnostic and therapeutic techniques (including the implications of pharmaceutical therapy discussed) to patients with the particular medical problems discussed;
- understand the differential diagnosis of the entity discussed;
- understand both likely and rare complications that may occur.

A. Octreotide
B. Cimetidine
C. Meperidine
D. Imipenem-cilastatin
E. Lexipafant

183. Which of the following statements is true regarding nasogastric suction in pancreatitis treatment?
A. It should be used to help improve pain relief.
B. Studies have shown that it may help to shorten a patient's hospital stay.
C. It should be used only in patients with ileus or uncontrollable vomiting.
D. It is used to help shorten time to onset of feeding.

184. Patients with acute pancreatitis can develop upper GI bleeding secondary to all of the following *except*:
A. stress ulceration.
B. gastric varices.
C. pancreatic pseudoaneurysms.
D. arteriovenous malformations.
E. splenic vein thrombosis.

185. At this time, no single serum marker has been proven sufficient to predict prognosis in pancreatitis.
A. True
B. False

186. Sterile and infected necrotizing pancreatitis can be differentiated by:
A. fever.
B. percutaneous aspiration.
C. degree of pain.
D. elevated white blood cell count.
E. hyperglycemia.

187. All of the following statements are true *except*:
A. The incidence of acute pancreatitis varies depending on the

CME Instructions

Physicians participate in this continuing medical education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to evaluate their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. *After completing this activity, you must complete the evaluation form that will be provided at the end of the semester and return it in the reply envelope provided to receive a certificate of completion.* When your evaluation is received, a certificate will be mailed to you.

Audio Conference Clarifies Final EMTALA Regulations

The final version of the recently proposed changes to the Emergency Medical Treatment and Labor Act (EMTALA) takes effect on Nov. 10.

To provide you with critical information on the updated regulations from the Centers for Medicare and Medicaid Services, Thomson American Health Consultants offers *New EMTALA Regulations: Are They Too Good to be True?*—an audio conference on Tuesday, Oct. 21, from 2:30-3:30 p.m., EST.

While the new rule is intended to ease restrictions for hospitals, it's only good news if you implement it correctly. You still could face violations, hefty fines, and misinterpretation.

* How do you provide emergency treatment during a national emergency?
* How does EMTALA apply to inpatients, including those admitted through the ED?
* What should be procedure regarding on-call lists?
* What's the new rule regarding hospital-owned ambulances?
* How are off-campus clinics affected?

Ensure that you and your staff are prepared with straightforward advice from a panel of EMTALA experts. The program will be presented by **James R. Hubler, MD, JD, FACEP, FAAEM, FCLM**, attending physician and clinical assistant professor of surgery, Department of Emergency Medicine, OSF Saint Francis Hospital and University of Illinois College of Medicine in Peoria, IL; and **Robert A. Bitterman, MD, JD, FACEP**, director of risk management and managed care, Department of Emergency Medicine, Carolinas Medical Center in Charlotte, NC.

Our expert advice will help you steer clear of potential pitfalls. At the end of this audio conference, participants will be able to:

* Compare previous EMTALA regulations with the final regulations;
* Identify several legal issues for a facility that might arise based on the final rule;
* Discuss at least three provisions of the final EMTALA regulation;
* Identify ways in which to initiate the required mandates of the final revisions.

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population being studied.

B. The structural changes associated with chronic pancreatitis are irreversible.
C. The cellular mechanisms behind acute pancreatitis have been elucidated.
D. Premature activation of pancreatic enzymes is the central event in the pathogenesis of acute pancreatitis.

188. Pancreatitis has clear association with all of the following conditions *except*:
A. alcohol abuse.
B. pregnancy.
C. mumps.
D. hypertriglyceridemia.
E. didanosine use.

189. No perfect test to diagnose pancreatitis exists.
A. True
B. False

190. Regarding radiographic tests in patients with acute pancreatitis, which of the following is true?
A. All patients should undergo plain abdominal radiographs in the ED.
B. All patients should undergo a CT scan of the abdomen while in the ED to evaluate the degree of pancreatitis and help determine prognosis.
C. All patients with acute pancreatitis should undergo abdominal ultrasonography early in their hospital stay to determine if a gallstone is the etiology of their pancreatitis.
D. All patients with acute pancreatitis should undergo ERCP to evaluate pancreatic and biliary ductal anatomy.

Answer Key

181. A	186. B
182. D	187. C
183. C	188. B
184. D	189. A
185. A	190. C

In Future Issues:

Foot and Ankle Injuries

Emergency Medicine Reports
The Practical Journal for Emergency Physicians

Pancreatitis

Causes of Acute Pancreatitis

OBSTRUCTION
- Gallstones (40%)
- Biliary sludge
- Ampullary or pancreatic tumors
- Foreign body or worm-obstructing papilla
- Choledochocele
- Hypertensive sphincter of Oddi

TOXINS
- Ethanol (40%)
- Drugs (2-5%) (*see Table 2 for full list*)
- Methanol
- Organophosphate insecticides
- Scorpion venom

TRAUMA
- Penetrating more common than blunt
- Iatrogenic injuries: ERCP (5%) or intraoperative

METABOLIC ABNORMALITIES
- Hypertriglyceridemia (1-2%)
- Hypercalcemia

INHERITED CONDITIONS
- Cystic fibrosis
- Genetic mutations

INFECTIOUS
- Viral (mumps, coxsackie B, Epstein-Barr, cytomegalovirus, rubella, hepatitis)
- Bacterial (mycoplasma, *M. tuberculosis*, *M. avium* complex, Leptospira, Legionella),
- Parasitic (ascaris, clonorchiasis, toxoplasma), fungal aspergillus)

VASCULAR ABNORMALITIES
- Hypoperfusion (intraoperative or due to hemorrhagic shock)
- Atherosclerotic emboli
- Vasculitis (SLE, PAN)

MISCELLANEOUS CONDITIONS
- Perforating peptic ulcer
- Hypothermia
- Reye's syndrome

IDIOPATHIC (10%)
- Many of the cases previously labeled idiopathic now are diagnosed using ERCP. The most frequent diagnosis is biliary sludge.

Note: For a full discussion with references, see reference 22. Percentage of cases is approximate, and is rare if not noted.

Key
ERCP = endoscopic retrograde cholangiopancreatography; SLE = systemic lupus erythematosus; PAN = polyarteritis nodosa

Drugs Strongly Associated with Acute Pancreatitis

CLASS	DRUG
ACE-inhibitors	Lisinopril
Anti-inflammatory drugs	Sulindac, salicylates
Antimicrobial agents	Metronidazole, stibogluconate, sulfonamides, tetracycline
Diuretics	Furosemide, thiazides
Drugs used for inflammatory bowel disease	Sulfasalazine, 5-ASA
HIV and AIDS therapy	Didanosine (ddI), pentamidine
HMG-CoA reductase inhibitors	Simvastatin
Immunosuppressive agents	L-asparaginase, azathioprine, 6-mercaptopurine
Neuropsychiatric agents	Valproic acid
Others	Calcium, estrogen, tamoxifen

Differential Diagnosis of Acute Pancreatitis

- Biliary colic or acute cholecystitis
- Renal disease (especially renal colic)
- Peptic ulcer disease
- Lung disease (pleurisy or pneumonia)
- Acute liver conditions
- Cardiac ischemia/inferior MI
- Closed loop bowel obstruction
- Dissecting aortic aneurysm
- Perforated hollow viscous
- Ectopic pregnancy
- Ischemic bowel
- Connective tissue disorders with vasculitis

MI = myocardial infarction

Causes of Increased Serum Amylase Activity

PANCREATIC DISEASES
- Acute pancreatitis
- Complications of pancreatitis
- Acute exacerbation of chronic pancreatitis
- Pancreatic tumors, cysts

OTHER INTRA-ABDOMINAL PROCESSES
- Acute cholecystitis
- Common bile duct obstruction
- Intestinal ischemia or infarction
- Intestinal obstruction or perforation
- Acute appendicitis
- Acute gynecologic conditions such as ruptured ectopic pregnancy and acute salpingitis

DISEASES OF SALIVARY GLANDS
- Mumps
- Effects of alcohol

TUMORS
- Ovarian cysts or papillary cystadenocarcinoma
- Lung carcinoma

RENAL INSUFFICIENCY

MACROAMYLASEMIA

MISCELLANEOUS
- Endoscopy
- Sphincter of Oddi stenosis or spasm
- Anorexia nervosa
- Head trauma with intracranial bleeding
- Diabetic ketoacidosis

CT Severity Index

GRADES OF INFLAMMATION	POINTS
A: Normal pancreas consistent with mild pancreatitis	0
B: Focal or diffuse enlargement of the gland, including contour irregularities and inhomogeneous attenuation but without peripancreatic inflammation	1
C: Grade B plus peripancreatic inflammation	2
D: Grade C plus associated single fluid collection	3
E: Grade C plus ≥ 2 peripancreatic fluid collections or gas in the pancreas or retroperitoneum	4
DEGREE OF NECROSIS	
None	0
< 33 %	2
33-50%	4
> 50%	6

Reprinted with permission from: Balthazar EJ, Robinson DL, Megibow AJ, et al. Acute pancreatitis: Value of CT in establishing prognosis. *Radiology* 1990;174:331-336.

Comparison of Ranson's and Glasgow Criteria

RANSON'S CRITERIA	GLASGOW (IMRIE) CRITERIA
At presentation:	***Over 48 hours:***
Age > 55 years	Age > 55 years*
WBC > 16,000/mm^3	WBC > 15,000/mm^3
Glucose > 200 mg/dL	Glucose > 180 mg/dL
LDH > 350 IU/L	LDH > 600 IU/L
AST > 250 IU/L**	AST > 200 IU/L**
	BUN > 96 mg/dL
	Ca < 8 g/dL
At 48 hours:	pO_2 < 60 mmHg
Hct decrease >10 mg/dL	Albumin < 3.2 g/dL
BUN increase > 5 mg/dL	
Ca < 8 mg/dL	
pO_2 < 60 mmHg	
Base deficit > 4 mEq/L	
Fluid sequestration > 6 L	

*1981 modification removed age as a criteria
**1984 modification replaced age and removed AST as a criteria
Key
WBC = white blood cell count; LDH = lactate dehydrogenase; AST = aspartate aminotransferase; BUN = blood urea nitrogen

Supplement to *Emergency Medicine Reports*, September 8, 2003: "Emergency Department Management of Pancreatitis." *Authors:* **Brian Clyne, MD,** Assistant Professor of Medicine, Division of Emergency Medicine, Brown University School of Medicine, Providence, RI; **Brian Patel, MD**, Department of Emergency Medicine, Rhode Island Hospital, Providence; and **Jeremiah Schuur, MD**, Department of Emergency Medicine, Rhode Island Hospital, Providence.

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Non-accidental Injury: Recognizing Child Abuse in the Pediatric Trauma Patient

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The recognition of non-accidental injury is critical for a pediatric trauma patient. In the year 2000, almost 3 million reports of child abuse were made to social service agencies.[1] More importantly, 1200 children died from neglect or abuse in that same year.[1] Forty-four percent of the fatalities were children younger than 1 year of age.[1] Not only are these statistics alarming, but they point out the need for emergency department (ED) and trauma physicians and nurses to recognize non-accidental injury and aggressively protect the children who seek our medical expertise and protection.

—The Editor

Child abuse and neglect are not uncommon, and clinicians who provide trauma care to children will encounter from several to many cases during a career. Child abuse is defined by federal legislation that provides minimum guidelines for states to incorporate into their criminal and civil statues. Subsequently, each state in the United States has a working legal definition of child abuse and neglect.[2-5]

There are four basic types of child maltreatment—physical abuse, sexual abuse, emotional abuse, and child neglect.[5] Child neglect is the most common form of child maltreatment investigated and is the failure to provide for a child's basic needs. This can include physical, emotional, medical, supervisional, and educational neglect. Each state has different definitions that help determine if a given situation meets the standard for neglect. Child Protective Services (CPS) is the branch of public social services that investigates reports of child abuse and neglect in which caretakers are involved as possible perpetrators. Law enforcement is involved in many of these cases and also has primary responsibility for investigating cases in which strangers and noncaretakers are the alleged perpetrators. CPS has to investigate any reported child maltreatment and then provide support services to the family if indicated.

Physical abuse and sexual abuse probably are the most common types of child maltreatment seen in ED settings. The physicians and nurses in these situations have the responsibility of recognizing clues to abuse or neglect and reporting them to appropriate agencies.

A medical provider must be very familiar with his or her state's reporting laws covering child maltreatment. All states list health care workers as mandated reporters of child maltreatment. To report a case to CPS, the provider need only have a suspicion that maltreatment or neglect has occurred, not proof.[2] CPS is

given the job of investigating any report when the agency believes the report meets a minimal standard of likelihood. All states mandate reporting without risk of liability if the report is made without malicious intent.[2] A medical provider can incur liability for failing to report a case of suspected child maltreatment. It is important to emphasize to parents (or other caretakers) that reporting is not an accusation, but is a legal requirement based on the assessment to that point.

Important factors to assess in possible abuse or neglect situations are the history, the child's developmental abilities, physical examination findings, and the presence of other risk factors for abuse and neglect. These risk factors include, but are not limited to, young maternal age (< 15 years), prior social service investigations, prior law enforcement involvement, substance abuse in the family, domestic violence, low socioeconomic status, and disability or prematurity of the child.[4,6,7] It also is important to remember that the absence of risk factors does not rule out abuse, and is not a reason to fail to report if suspect findings are present.

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The intended audience for this publication is emergency, family, osteopathic, and general practice physicians and nurses who have contact with trauma patients.

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The abused child presents a unique set of epidemiologic, physiologic, and social circumstances.[9,10] The clinical history often is difficult to obtain, and deliberately may be obfuscatory, false, or contradicted by physical findings or known patterns of injury. The presentation frequently is delayed, allowing many mechanisms of secondary injury to become well established. Abused children, especially infants, often present with nonspecific complaints and findings.[11-13] These children frequently present with respiratory distress or apnea, but vomiting, mental status changes (lethargy or irritability), poor feeding, or nonspecific behavioral changes also are common.[11,14] Seizures are reported in up to 70 % of patients.[11] Many abused children are repetitively traumatized; thus, the presenting incident may simply be the worst (or most recent) of several episodes.[15,16]

An unsuspecting physician may accept a false history of trauma and not consider the possibility of abuse. To identify abused children, child abuse needs to be included on the differential list of all pediatric injuries.[4] In most instances, child maltreatment quickly will be eliminated from the differential, but a small dose of suspicion is needed to identify abused children.

The diagnosis of child abuse is made like all others—a careful history, complete physical examination, and supporting laboratory and radiographic data lead to a working diagnosis. In some cases, the physical examination alone indicates abuse, but in most cases, the comparison of the history of trauma and the resulting injuries suggests the diagnosis. The following are general indicators of possible child abuse:[4]

- Infants and children with unexplained injuries;
- The history provided does not adequately explain the injuries;
- The history provided changes with time;
- The history provided does not correlate with the child's developmental abilities; or
- There is an unexplained delay in seeking medical care.

Clinically, child abuse may be obvious or subtle. A child with external signs of trauma, known as a "battered" child, should be treated the same as any trauma victim.[12,13,17] Abused children with less obvious presentations often have injuries falling into certain patterns. Retinal hemorrhages are common after head injury from child abuse, and need to be documented and taken into consideration with other clinical and radiographic findings.[11,18,19] Retinal hemorrhages are not all the same and should be described thoroughly, including amount, type, distribution, and side affected (bilateral, unilateral, or asymmetric).

Suspicion of nonaccidental trauma should prompt immediate investigation. Social work, CPS, and any appropriate social or legal authorities should be involved as soon as possible.

History and Physical Examination

The initial history often is the first clue to the detection of child abuse. Obtain a detailed history, including location, time, and mechanism of any injury described. The identification of significant injury unaccompanied by a history of trauma is cause for concern. If the history does not seem to explain the injury identi-

fied, the possibility of abuse should be considered. Parents who provide a false history of trauma to explain a child's abusive injuries most commonly offer simple household trauma as an explanation.[12] For example, infants or toddlers with abusive head injury often are said to have fallen off a bed or couch.

It is important to document the events from the time of the injury leading to the medical visit. Some parents, in an effort to explain inflicted injuries, provide various histories to explain an injury, or change the story once additional injures are identified. A comparison of histories obtained from various health care providers, pre-hospital personnel, emergency physicians, hospitalists, or intensivists may reveal inconsistencies. Also, compare histories obtained from adult caregivers present at the time of injury.

An unexplained delay in seeking medical care should be recorded and explored. In an effort to prevent detection of abuse by professionals, parents occasionally will keep their injured child at home, despite the obvious need for medical intervention.

The physical examination of the child must be complete. Examine all surfaces of the child's skin carefully, and document any injuries. In infants, subtle external injuries are often a clue to more serious internal injury, and should not be dismissed. Bruises, burns, and scars should be measured, and their size, shape, location, and color recorded. Photographs are an important adjunct to the recorded physical examination, but are not an appropriate substitute for accurate medical documentation.

Craniofacial, head, face, and neck injuries occur in more than half of the cases of child abuse.[3] Careful intraoral and perioral examination is necessary in all cases of suspected abuse. Some authorities believe that the oral cavity may be a central focus for physical abuse because of its significance in communication and nutrition.[3] The injuries most commonly are inflicted by blunt trauma with an instrument, eating utensils, hands, or fingers or by scalding liquids or caustic substances.[3,20] The abuse may result in contusions; lacerations of the tongue, buccal mucosa, palate, gingiva alveolar mucosa, or frenum; fractured, displaced, or avulsed teeth; facial bone and jaw fractures; burns; or other injuries. These injuries, including a lacerated frenum, also can result from unintentional trauma. Gags applied to the mouth may leave bruises, lichenification, or scarring at the corners of the mouth.

Laboratory and Radiographic Data

Abused children often have occult injuries, and some medical diseases may mimic abuse. Therefore, the laboratory and radiographic evaluation of the abused child is guided not only by the history and physical examination findings, but also by the above considerations. Necessary laboratory and/or radiographic testing varies by age, injury pattern, and severity. The following studies may be appropriate in the evaluation of an abused child.

Laboratory Studies. *Hematologic Evaluation.* A complete blood count (CBC) with platelet count, prothrombin time (PT), and partial prothrombin time (PTT) are indicated for children who present with bleeding or bruising. Elevations of PT and PTT may be the result of severe inflicted head injury.[21]

Liver Function Tests/Pancreatic Enzymes. Alanine aminotransferase (ALT), aspirate aminotransferase (AST), amylase, and/or lipase may be elevated with acute liver or pancreatic injury. Such injuries can be asymptomatic, and screening is recommended for injured infants and children in whom the abdominal examination may not be a sensitive indicator of injury.

Urinalysis. The urinalysis is used as a screen for renal or bladder trauma, and can detect myoglobinuria secondary to rhabdomyolysis from severe beatings.

Toxicology screens are indicated for infants and children with unexplained neurological symptoms.

Diagnostic Imaging. Imaging studies often are critical in the assessment of the infant and young child with evidence of physical injury, and they also may be the first indication of abuse in a child who is seen with an apparent natural illness.[4,22,23] When viewed in conjunction with clinical and laboratory studies, imaging findings commonly provide support for allegations of abuse. For severely abused infants, the imaging findings alone may form the basis for a diagnosis of inflicted injury.[22] The role of imaging in cases of suspected abuse is not only to identify the extent of physical injury when abuse has occurred, but to elucidate all imaging findings that may point to alternative diagnoses. Because most conventional imaging studies performed in these settings are noninvasive and entail minimal radiation risks, recommendations about imaging should focus on examinations that provide the highest diagnostic yield at acceptable cost.[22]

Roentgenographic Skeletal Survey. The skeletal survey is an important adjunct to the evaluation of abused infants and toddlers, and is indicated for all children younger than 2 years with any suspicious injury.[4] Guidelines for the appropriate imaging methods have recently been updated by the American Academy of Pediatrics.[22]

Although skeletal injuries rarely pose a threat to the life of the abused child, they often are the strongest radiologic indicators of abuse. In fact, in an infant, certain patterns of injury are sufficiently characteristic to permit a firm diagnosis of inflicted injury in the absence of clinical information.[22] This fact mandates that imaging surveys performed to identify skeletal injury be performed with at least the same level of technical excellence routinely used to evaluate accidental injuries. The "body gram" (a study that encompasses the entire infant or young child on one or two radiographic exposures) or abbreviated skeletal surveys have no role in the imaging of these subtle but highly specific bony abnormalities.[22]

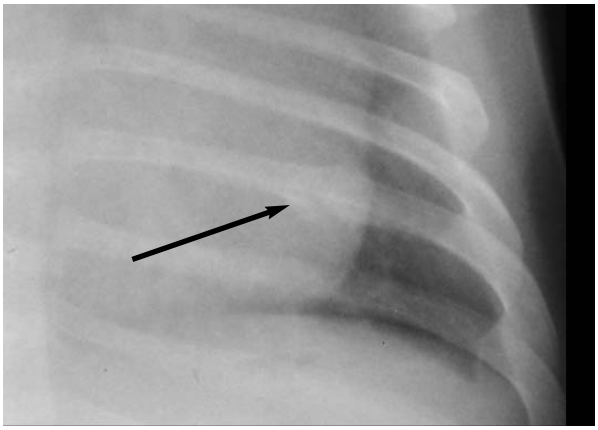
In general, the radiographic skeletal survey is the method of choice for global skeletal imaging in cases of suspected abuse. The standard skeletal survey imaging protocol that has been developed by the American College of Radiology is given in Table 1.[22,24] Anteroposterior and lateral views of the skull are mandatory even when cranial computed tomography (CT) has been performed because skull fractures coursing in the axial plane may be missed with axial CT. Skeletal injuries, especially those requiring orthopedic management, necessitate at least two radiographic projections. Oblique views of the thorax increase the yield for the detection of rib fractures. *(See Figure 1.)* Recent

Table 1. Standard Skeletal Survey

APPENDICULAR SKELETON	AXIAL SKELETON
Humeri (AP)	Thorax (AP and lateral)
Forearms (AP)	Pelvis (AP; including middle and lower lumbar spine)
Hands (Oblique PA)	Lumbar spine (lateral)
Femurs (AP)	Cervical spine (lateral)
Lower legs (AP)	Skull (frontal and lateral)
Feet (AP)	

AP= anteroposterior; PA=posteroanterior

Figure 1. Oblique View to Detect Rib Fracture



Shallow oblique radiograph demonstrating a subtle rib fracture in a victim of child abuse.

evidence suggests that a follow-up skeletal survey approximately two weeks after the initial study increases the diagnostic yield, and this procedure should be considered when abuse is strongly suspected.[24,25] The repeated study may permit more precise determination of the age of individual injuries. Lack of interval change may indicate that the initial radiographic finding is a normal anatomic variant or is related to a bone dysplasia.

Radionuclide Bone Scan. A bone scan identifies areas of increased bone turnover, and is a sensitive method for detecting rib fractures fewer than 7-10 days old, subtle diaphyseal fractures, and early periosteal elevation.[24] A bone scan is most commonly used as an adjunct to the skeletal survey when abuse is strongly suspected and the skeletal survey is normal. Conversely, the skeletal survey can be repeated 3-4 weeks after the initial survey to increase the detection of healing injuries.

Computed Tomography Scan. A CT scan is the method of choice for diagnosing acute intracranial, pulmonary, and solid abdominal organ abnormalities in children with serious injuries.[22-24]

Magnetic Resonance Imaging (MRI). MRI scans of the brain are more sensitive than CT scans in detecting certain traumatic injuries, including axonal shearing, cortical contusions, and brainstem injuries.[22-24]

Manifestations of Physical Abuse

Nonaccidental injuries may affect any organ system in the body, alone or in combination. The following are common manifestations of child abuse.

Head Trauma. Trauma is the most common cause of death in childhood, and inflicted head injury is the most common cause of traumatic death in infancy.[6,11,26,27] On average, among children hospitalized for blunt trauma, those injured by abuse sustain more severe injuries, use more medical services, and have worse survival and functional outcome than children with unintentional injuries.[16,26,28-30] This is despite a plethora of interventions developed over the last 30 years, including legislatively mandated reporting and the establishment in 1974 of the National Center on Child Abuse and Neglect as a mechanism to increase knowledge of the problem and identify steps to prevent it.[6]

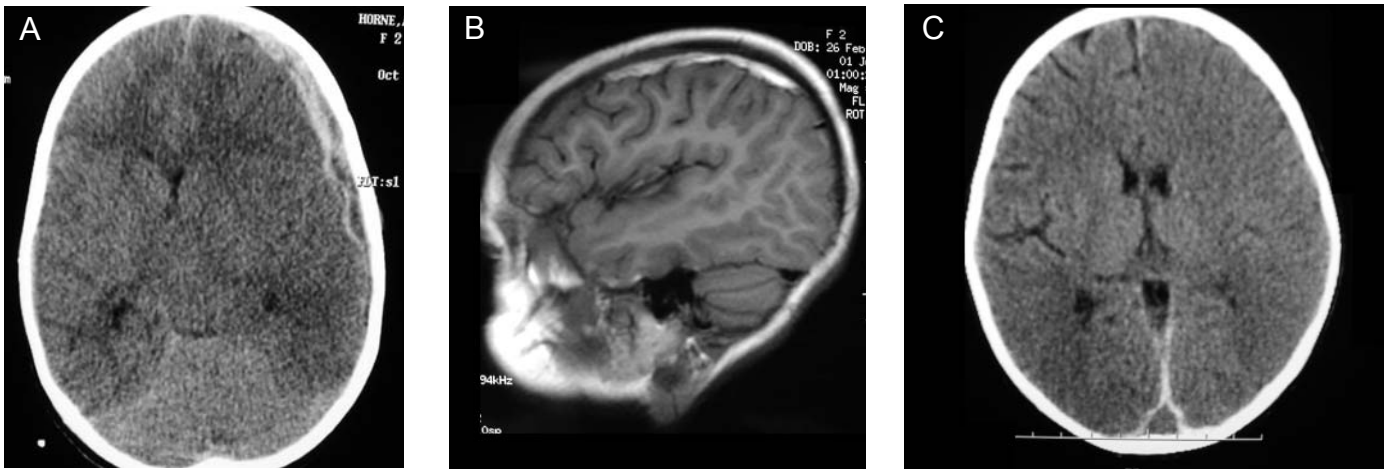
The major issue plaguing the description of abuse-related injuries to young children has been and continues to be accurate diagnosis.[26,31,32] The dire consequences of either false-positive or false-negative diagnosis intensifies the need to establish accurate diagnostic criteria.

Because of some unique features of the infant brain, the risk for severe intracranial injury is great.[12] With a large head-to-body ratio and relatively weak neck muscles, infants are more susceptible to the acceleration/deceleration forces of abusive head trauma. In addition, the immature brain, not fully myelinized, has only 25% of its adult weight at birth, and 75% by age 2 years.[33] There is more subarachnoid space, and therefore a greater vulnerability to shearing of blood vessels causing significant hemorrhage. There is a common misconception that infants will tolerate an acute insult better than their adult counterparts because the fontanel is open; however, it must be remembered that the brain is encased by the inelastic dura.[33] The infant has a shorter craniospinal axis than the adult and thus has less of a "potential space" to displace blood volume and cerebrospinal fluid; this can make the infant brain more susceptible to increased intracranial pressure and secondary brain injury.[33]

Head injuries in infants and toddlers can be difficult to diagnose because symptoms are often nonspecific.[12] Vomiting, fever, irritability, and lethargy are common symptoms of a variety of conditions seen in children, including head trauma. When caretakers do not give a history of injury and the victim is preverbal, an abusive head injury mistakenly can be diagnosed as a less-serious condition. When medical personnel fail to recognize that the child's symptoms are secondary to nonaccidental head injury, the child is frequently re-injured or has serious complications of the unrecognized, untreated head injury.[34]

The primary brain injury attributed to child abuse was originally thought to result from repetitive accelerative-decelerative forces of shaking, hence the term "shaken-baby syndrome."[11] However, biomechanical studies subsequently suggested that the inertial forces developed from shaking were insufficient to cause diffuse axonal injury (DAI), suspected to be the primary mechanism of neuronal injury after abuse.[11] Because impact with even a soft surface, such as a bed or crib, could theoretically cause DAI, the concept of the "shaken-impact syndrome" developed.[11] Recent reports have shown, however, that diffuse changes consis-

Figure 2. CT and MRI in Children with Subdural Hemorrhage and Ischemic Changes



A—Computed tomography (CT) from a 2-year old showing subdural hemorrhage, edema, and shift. **B**—T1-weighted magnetic resonance imaging (MRI) scan showing subdural hemorrhage in a nonaccidental trauma victim. **C**—Interhemispheric hematoma demonstrated on CT scan.

tent with hypoxic-ischemic damage are more common than DAI, which is found mostly in the brainstem, and that cervical spine injury is frequent.[9,35-37] This suggests that the inertial forces of shaking cause brainstem and spinal injuries, which lead to respiratory impairment and subsequent hypoxic injury.

Apnea induced by shaking or by shaking combined with impact plays a major role in the pathophysiology of nonaccidental head trauma and accounts for the poor outcome in this group of patients.[14,38] Trauma induced apnea causes cerebral hypoxia and possibly ischemia, which may be more fundamental to outcome than the mechanism of injury (shaken vs shaken with impact), subdural hemorrhage, subarachnoid hemorrhage, diffuse axonal injury, parenchymal shear, or brain contusion. In addition, the timing of the primary injury is closely linked to the onset of apnea.[37,39]

CT scanning is a mainstay of the diagnosis of nonaccidental head injury.[22,40] Subdural or subarachnoid hemorrhage almost always can be detected on CT scans, although the more subtle findings may be missed by less experienced observers.[40-44] The most frequent CT findings in nonaccidental traumatic brain injury are a combination of subdural convexity and interhemispheric hematomas.[16,26,45] The interhemispheric subdural hematoma is particularly characteristic of the shaking or shaking-impact mechanisms in which angular acceleration and deceleration forces are involved. *(See Figure 2.)*[46] The majority of subdural hemorrhages in children younger than 2 years are due to child abuse.[40,43] MRI is useful in detecting and characterizing small extraaxial hemorrhages in infants with equivocal CT findings.[46,47]

The neuroradiologic findings of abuse often are present within a few hours of the assault, particularly when the event results in hypoxia-ischemia.[14,37,46] In these cases, the CT may show findings of edema as early as 2 hours following the documented event.[46] The earliest CT sign is often a loss of cerebral gray-white matter differentiation in the cortical and subcortical regions, in deep basal ganglia and capsular regions, or in both. Hypoxic-ischemic brain injury may result from associated brainstem injury with apnea, from progressive edema and increased intracranial pressure, or from additional suffocation or strangulation.[46]

All infants and children with suspected intracranial injury must undergo either cranial CT or MRI. *(See Figure 2.)* Strategies should be directed toward the detection of all intracranial sequelae of abuse and neglect with a thorough characterization of the extent and age of the abnormalities. In the acute care setting, efforts are directed toward rapid detection of treatable conditions. Subsequent studies are designed to delineate more fully all abnormalities, determine the timing of the injuries, and monitor their evolution.[44,46]

Radiologists evaluating imaging studies of young children, even those in whom there is no suspicion of physical abuse, should routinely assess subtle signs of chronic brain damage in addition to acute findings.[16,41,44] Investigators have identified elevated rates of cortical atrophy, ventriculomegaly, and subdural hygroma only in children with inflicted traumatic brain injury.[16]

MRI is the best modality to assess fully intracranial injury, including extraaxial collections, intraparenchymal hemorrhages, contusions, shear injuries, and brain swelling, or edema.[43] Imaging should be performed with T_1 and T_2 weighting with proton-density or inversion-recovery sequences to differentiate cerebrospinal fluid collections from other water-containing lesions. Diffusion imaging is a new and valuable technique for the evaluation of cerebral ischemia and likely will have a role in the assessment of inflicted cerebral injury.[36,48] Abused infants may not demonstrate neurologic signs and symptoms, despite significant CNS injury. The MRI offers the highest sensitivity and specificity for diagnosing subacute and chronic injury and should be considered whenever typical skeletal injuries associated with shaking or impact are identified.

Table 2. Nonaccidental Trauma: Hints to Facilitate an Early Diagnosis

FRACTURES

- No single fracture is pathognomonic of child abuse.
- Certain patterns of fractures should increase the concern for abuse, including multiple fractures and fractures of different ages.
- Multiple, bilateral posterior rib fractures in infants are almost always the result of abuse.

BABIES WITH BRUISES

- With advancing age and motor skills, bruises occur more commonly.
- Bruises in mobile infants normally are located on the front of the body, typically on the shins and forehead.
- Bruises in infants younger than 9 months who are not yet beginning to ambulate should lead to consideration of abuse or illness.
- Bruises in toddlers that are located in atypical areas, such as the trunk, genitalia, neck, hands or buttocks, should be considered abuse.

SKULL FRACTURES: ACCIDENTAL VS. NON-ACCIDENTAL

- Inflicted
  - History does not correlate with mechanism of injury, OR no history
  - Associated with intracranial injury, especially subdural hematomas
  - Bilateral
  - Non-parietal
  - Comminuted/Stellate
  - Depressed
  - Wider than 1 mm
  - Associated with other injuries
  - Crossing suture lines
- Accidental
  - History consistent with injuries
  - Not associated with intracranial injury
  - Unilateral
  - Parietal
  - Linear
  - Nondepressed
  - Narrower than 1 mm
  - Involves only one body area
  - Does not cross suture lines

Many studies have demonstrated the usefulness of MRI in suspected acute nonaccidental head injury. It is particularly useful for the diagnosis of subdural hematomas in the subtemporal area, illustrating tearing of the bridging veins, delineating subdural hematomas of different ages, and demonstrating hemorrhages at the gray-white matter junction.[47] All these features are not well defined by CT.

Several selected points culled from an extensive literature review may prove useful in recognizing nonaccidental trauma in infants and children. *(See Table 2.)*[2,27]

There is an abundance of literature attesting to the fact that short falls (< 4 feet), such as from beds, couches or down stairs, do not cause serious injury in children, except the case of epidural hematoma, which can occur after short falls.[2,26,49-58] However, some of the most severe infant head injuries occur when an adult falls on the stairs while carrying an infant.[55,59,60] The relative severity of these injuries is probably the result of two different injury mechanisms: fall from a height and a fall down stairs.[60] These two mechanisms can increase the impact force.

Simple skull fractures are common in accidental falls, with complex fractures seen less frequently.[26] Subdural hematomas and subarachnoid hemorrhages seldom are seen and retinal hemorrhages virtually are never seen in short falls.[2]

Inflicted head injury in infants commonly presents as shaken baby syndrome. Altered level of consciousness, coma, seizure, listlessness/lethargy, irritability, apnea or respiratory difficulty, and poor feeding can be associated with shaken baby syndrome. Although not pathognomonic for child abuse, retinal hemorrhages occur in up to 75-85% of these cases and frequently correspond with subdural or subarachnoid bleeding.[13,18] Skeletal trauma, such as sternal fractures and posterior rib fractures, frequently are seen in shaken baby syndrome due to the placement of the perpetrator's hands. Acceleration/deceleration forces are created as the head whips forward, stopping as the chin strikes the chest, then backward until the occiput strikes the back, as well as when the baby is thrown on the sofa or bed. Shearing injuries to the intracranial bridging veins and incompletely myelinated cortical nerves are sustained. CT scanning is an excellent tool for detecting subdural and subarachnoid hemorrhages, especially when associated with retinal hemorrhages. Epidural hemorrhage in infants is less likely to be caused by shaking and more likely to be due to a blow or fall.[13,61,62] Epidural hemorrhages are rarely a result of abuse.

Finally, the evaluating physician also must be aware of certain conditions that are known to have clinical and imaging features that may mimic abuse.[2,12,63] These include accidental injury, certain coagulopathies, vascular diseases, infections, metabolic disorders, neoplastic diseases, certain therapies, and some congenital and dysplastic disorders. Infants who present with unexplained subdural and retinal hemorrhages most often are victims of child abuse. However, an autosomal recessive metabolic disorder, glutaric acidemia type 1, is a known cause of these findings in children.[63,64] Urine organic acid testing will show a characteristic large peak of glutaric acid and 3-hydroxy glutaric acid. The diagnosis can be confirmed through testing of enzyme activity in cultured fibroblasts and leukocytes or through genetic mutation analysis.

Abdominal Trauma. Severe abdominal injury is an uncommon, but well recognized, manifestation of abuse.[65] The history almost always is misleading, and rarely includes a history of abdominal trauma. Less severe injury is under-recognized and underreported, because symptoms are non-specific and external indications of abdominal trauma are often lacking.[23]

Most abusive abdominal injury is caused by blunt trauma, resulting in solid organ injury, perforation of hollow viscous, or shearing of mesenteric vessels.[4,65-68] Isolated solid organ injuries are most common with both accidental and inflicted injuries,

although hollow visceral injuries more commonly are associated with abuse.[4] Recent reports of abdominal trauma secondary to abuse reveal that liver and spleen injuries are most common, followed by duodenojejunal rupture, duodenal rupture, and pancreatic, vena cava, and renal trauma.[66,67] These injuries are thought to be due to compression of abdominal viscera against the vertebral column following a punch or a kick.[66] The small size of the child's abdomen predisposes them to multiple organ injury. Children with severe liver or mesenteric injury usually present with signs and symptoms of acute bleeding, including hemorrhagic shock.[66] Children with intestinal perforation typically present with signs of peritonitis. Their presentation for medical care can be delayed by hours or days, but an accurate history (often lacking) should demonstrate progressive abdominal symptoms.[66] Mortality is extremely high owing to delays in presentation and the magnitude of injuries.[68]

Abused children occasionally have asymptomatic abdominal injuries, which can be detected with evaluations of serum liver function tests (LFTs), amylase, and lipase.[66] Abdominal trauma is diagnosed by physical examination, screening LFTs, amylase, lipase, urinalysis, sonography and abdominal CT.[65-68]

Injuries involving the duodenum are a common finding in blunt trauma to the abdomen, and include duodenal hematomas or transection. Such injuries to the duodenum occur because the ligament of Treitz is a relatively fixed structure and allows compression of the duodenum against the vertebrae. *(See Figure 3.)*[65] It is not infrequently associated with injuries to the adjacent organs. Unfortunately, there are often delays in making the diagnosis because the retroperitoneum, in which part of the duodenum lies, offers some protection and this contributes to the morbidity and mortality of these injuries.

Life-threatening intraabdominal injuries can exist with few signs or symptoms. One research team describes a 2½ -year-old child who presented with relatively few symptoms, who at post mortem examination was found to have peritonitis secondary to a duodenal rupture which was considered to have occurred 2-3 days before death.[69]

Liver injuries also can be occult with no evidence of external injuries. One study found, in children suspected of nonaccidental injury who had no history or physical signs of abdominal trauma, evidence of occult liver lacerations on CT in 6% of cases.[70] Researchers also found that raised transaminases were associated with these cases and consider that this is a useful indicator of occult liver injury.[70]

Nonaccidental trauma as a cause of pancreatic injury is not uncommon. One group reported one-third of their series of 49 cases of pancreatitis in children as being associated with nonaccidental injury.[71] As in adults, pancreatitis in children is associated with considerable morbidity and chronic pancreatitis may develop.

Fractures. Fractures often are seen in physical abuse cases.[72] They also are a common accidental childhood injury. As in the preceding cases, the clinician must ask, "Is the injury consistent with the history and developmental stage?"

Abusive fractures are more common in younger children (< 5 years of age), often present without a history of trauma, and often are characterized by a delay in seeking medical care.[72] Any fracture can be the result of abuse.[3] It is the history, physical examination, and additional evaluation that are crucial in differentiating accidental from nonaccidental trauma. In children younger than 2 and selectively in children younger than 5 years of age, a skeletal survey is in order to look for other injuries, some of which may be occult.

A common type of history given in both abusive and unintentional injuries is a fall. Although a history of a fracture in a minor fall should be investigated, single unintentional fractures can occur from falls of fewer than two feet, and falls from fewer than four feet can result in injury to more than one bone.[73]

Radiographically, some fractures are more suspect for abusive injury than others.[24,72,74] Fractures that are highly specific for abusive injury in infants include the following: posterior rib fractures, scapular fractures, spinous process fractures, sternal fractures, and classic metaphyseal lesions. The classic metaphyseal lesions often are called bucket handle or corner fractures, and occur at the end of the long bones at the growth plate. Fractures with moderate specificity for abuse are complex skull fractures, digital fractures, vertebral body fractures, epiphyseal separations, fractures of different ages, and multiple fractures. Fractures common in childhood with low specificity for abuse are linear skull fractures, long bone fractures, and clavicular fractures. The appearance of subperiosteal new bone formation in infants also is common, but can be a normal variant. A pediatric radiologist may help with this evaluation when appropriate.

Linear parietal skull fractures are common whether unintentional or secondary to abuse.[72] However, skull fractures that are multiple, depressed, diastatic more than 3 mm, bilateral, or cross suture lines are more suggestive of intentional injury, especially coupled with a suspicious history.[24,72,73]

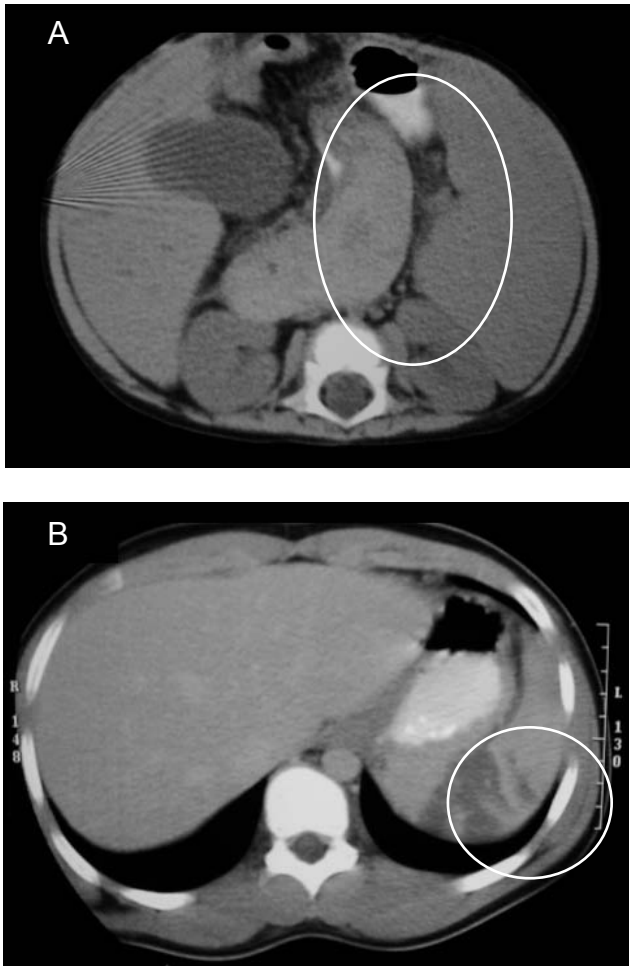
The humerus is the most commonly fractured bone in battered children.[72,75] Several authors have reported that the majority of humerus fractures in infants younger than 15 months are intentional.[72]

Rib fractures commonly are seen in abused children, with 90% of abuse-related fractures occurring in children younger than 2 years.[72,76,77] In infants, rib fractures are the most frequent fracture of abuse.[72,78] In general, rib fractures in children are much less common than in adults owing to their more compliant chest walls. Therefore, rib fractures, especially multiple fractures, are very suggestive for abuse in children younger than 2 years in the absence of major blunt trauma or prior bone pathology.[72]

Abusive fractures typically occur when the infant is manually grabbed around the thoracic cage and violently squeezed and shaken. This anteroposterior compressive force results most frequently in multiple, symmetrical, posterior rib fractures where mechanical stress is at its greatest. With increasing force, lateral then anterior fractures occur.[79] Of importance, rib fractures from cardiopulmonary resuscitation are rare in infants and young children.[72]

Diseases such as osteogenesis imperfecta, rickets, and osteomyelitis can mimic abusive injuries.[72] Most have other clini-

Figure 3. Abdominal Trauma



A—Duodenal hematoma. **B**—Splenic lacerations.

cal manifestations that distinguish them from abuse, and may be diagnosed by simple blood tests.[4]

Bruises. A bruise is an area of skin discoloration caused by the escape of blood from ruptured underlying blood vessels after an injury. It is the process of hemoglobin degradation and its expression through the "window" of the skin that determines the color of a bruise.[80] They are a common injury in children and often are accidental. However, bruises may be a sign of physical abuse.[80-85] To evaluate whether a bruise is abusive or accidental, it is important again to look at the history, the child's developmental level, the pattern of the bruising, and other findings on physical examination.

In general, bruises are very difficult to age by appearance alone, especially in children. A yellow color to the bruise indicates that it is probably more than 18 hours old. However, red, blue, purple, and brown can be seen at any time. The healing of bruises is affected by the area of skin involved, the depth of the bruise, the amount of blood in the bruise, and other factors.[80] Therefore, the dating and aging of bruises is inexact.[4]

The history of the bruise is important—both from the caretaker and the child, if obtainable. When was the first time the bruise was noted? What is the reported mechanism—fall, play, sports? Does the mechanism of injury match the bruise seen? Could the child do what is reported?

In the physical examination, it is important to look at all areas of skin and document any injuries. The area of the body affected is important because some areas are more suspect for abuse than others.[83-85] Some areas of the body such as the back, buttocks, neck, cheek, ear, thighs, genitalia, and hands are atypical areas of accidental bruises. Grab marks sometimes are seen on the upper arms. Some bruises may appear patterned. Patterns of concern for abusive injury are looped cord marks, hand prints, and other patterns that could be matched to an object such as a shoe, belt, or other implement.[2] Patterns of abusive bruising also may be determined by the anatomy of the injured body part rather than the shape of the injuring object. Two examples include patterned abusive bruises of the buttocks and the pinnae.[86,87]

Bite marks are lesions that may indicate abuse.[3] Bite marks should be suspected when ecchymoses, abrasions, or lacerations are found in elliptical or ovoid patterns. Bite marks may have a central area of ecchymoses. The normal distance between the maxillary canine teeth in adult humans is 2.5-4.0 cm, and the canine marks in a bite will be the most prominent on deep parts of the bite.[3] Bites produced by dogs and other carnivorous animals tend to tear flesh, whereas human bites compress flesh and can cause abrasions, contusions, and lacerations but rarely avulsions of tissue. If the intercanine distance is less than 2.5 cm, the bite may have been caused by a child. If the intercanine distance is 2.5-3.0 cm, the bite was probably produced by a child or small adult; if the distance is greater than 3.0 cm, the bite was probably by an adult.[3] The pattern, size, contour and colors of the bite mark should be evaluated by a forensic odontologist or forensic pediatrician or pathologist if an odontologist is not available. A photograph, taken at a right angle to the bite should be taken with an identification tag and scale marker in the photograph. Because each person has a characteristic bite pattern, a forensic odontologist may be able to match dental models (casts) of a suspected abuser's teeth with photographs of the site.

The differential diagnosis for bruises is long.[2,82] Mongolian spots often are confused with bruises. Mongolian spots do not fade or change over days to weeks. A repeat examination can make the diagnosis if there are questions. Minimal accidental trauma may result in dramatic bruises suggesting nonaccidental trauma in some medical problems such as idiopathic thrombocytopenic purpura, hemophilia, and vitamin K deficiency.[82,88] In settings of inappropriate bruising, it sometimes is appropriate to obtain a complete blood count and coagulation studies.

In some cultures, there are practices that can be confused with abusive bruising. These are cupping and coining.[82] These cultural practices are done with the intent of helping the child, and some report that it feels good.

The key question remains, "Is this injury consistent with the history provided by the caretaker, and is it consistent with the child's developmental abilities?" Several studies have looked at bruises in relation to development.[83,84] Based on their research,

Sugar, et al., reported, "Those who cannot cruise don't bruise."[83] They found that those children who are not yet cruising (walking while holding onto furniture) do not have significant bruises on their bodies. There may be one or two isolated bruises over bony prominences (e.g., forehead, knee), but most of these children do not have bruises when examined. Bruises on the face and neck necessitate a good history owing to the concern for physical abuse.

Burns. Burns occur when tissue is damaged by heat, chemicals, sunlight, electricity, or nuclear radiation.[89] The history related to how burns occurred and how that correlates with the burn pattern is more important than the depth of burns. But in investigating burns, records must include specific details about pattern, location, and degree. Burns can be classified into first-, second-, third-, and fourth-degree, based on the depth and severity of the burn.

First-degree burns are the most common and rarely require hospitalization. There is superficial tissue damage often characterized by painful erythema without blisters. These usually heal without scarring. An example would be the common sunburn. Other than raising issues of possible neglect, this severity of burn usually is not related to abuse.

Second-degree burns are considered partial-thickness burns. They are characterized by clear fluid-filled blisters that are very painful and sensitive to temperature and air. The lesions often blanch with pressure. They usually heal within 1-2 weeks and sometimes scar.

Third-degree burns involve the full thickness of the skin. The injury is characterized by the finding of charring or translucent white tissue with mottling. Over time, the overlying tissues may develop a leathery, dry appearance. There is minimal pain in the charred tissue because the nerve endings have been damaged. There often is marked edema, and the color can vary from white to gray to red to black or charred. These burns always scar and often require excision and grafting.

Fourth-degree burns involve not only all layers of the skin, but also subcutaneous fat and deeper structures. They usually have a charred appearance. A unique situation seen in child abuse with fourth-degree burns is when a body (a child) is placed in an operating microwave oven.[90] There are only a handful of case reports in the literature, all involving children. In such cases, the tissue injury often is worse than it appears because of the way that microwave ovens cook. This type of burn will show relative layered tissue sparing where the skin and underlying muscle are burned with relative sparing of the subcutaneous fat.[91] A child with this injury must be closely monitored for complications, preferably in a specialized burn unit.

Burns also can be classified as thermal, electrical, chemical, or radiation. The most commonly seen in abuse are thermal burns. Electrical burns can be seen in children when electrical cords are chewed (corners of the mouth) or outlets explored (fingertips). Chemical burns and radiation burns rarely are seen. Depending on severity and location of burns, some children do need to be transferred to the nearest burn facility.

In evaluating burns in children, there are several factors to consider. These include the history, physical examination, the child's developmental level, and the presentation of the injury to medical care.

When obtaining a history, the clinician first investigates the reported mechanism for the burn. It is important to note in the medical record the reported history and who reported it. In abuse cases, the history of the injury sometimes changes over time or with different witnesses. This can sometimes occur with accidental injuries, but such inconsistencies should raise a red flag for possible abuse.

The examiner also should look at the child's developmental level, both reported and observed. The clinician should remember to observe whether details provided by the caretaker are similar to those observed in interactions with the child. In evaluating burns, the examiner must ask himself or herself such questions as could that child have reached up and grabbed that cup of hot tea? Is the history compatible with the child's developmental level? CPS or law enforcement agencies usually can provide scene evaluations when necessary.

With possible child abuse, the physical examination is more than gathering information to assess the patient's injuries and provide care; it is a source of very valuable information about mechanisms of injury and possible abuse. What is the pattern of the burn injury—immersion, flow, or contact? Does this pattern match with the reported history? As an example, the stocking-and-glove distribution seen in immersion burns is fairly specific for nonaccidental injury.[4] These have a clear line of demarcation between the burned and unburned skin that looks like a sock line. There often are no splash marks. This is indicative of the limb being held forcefully in hot water. A donut distribution on the buttock area also can be seen with immersion burns. This is seen when the child is held in hot water in a tub (or sink). The donut appearance of central sparing and peripheral burns is related to the surface (usually of the tub) protecting the central skin, and the fluid in contact with the periphery resulting in second-degree burns. Skin in contact with other skin (e.g., between buttocks, behind knees) also is spared. By noting the burn pattern, the position the child was in at the time of the burn often can be recreated. As a protected area of the body, genital burns are uncommon accidental injuries. They often are seen in abusive situations, especially those involving toileting accidents.[4]

Burns may be inflicted by contact with hot solids, such as irons, radiators, stoves, or cigarettes.[4] Inflicted burns are characteristically symmetrical, deep, and leave a clear imprint of the hot instrument. Dermatologic and infectious disease can mimic abusive burns, including toxin-mediated staphylococcal and streptococcal infections, impetigo (which can be mistaken for cigarette burns), and phytophotodermatitis.[4,82]

How the child presented to medical care also is important. Did the child receive what appears to be a second-degree burn four days ago and is just now coming for treatment? Who is bringing the child for treatment? In abuse situations, there often is a delay in seeking appropriate medical care.

In completing the physical examination, the provider should be alert to other signs of trauma or neglect such as failure to

thrive or other injuries and scars. The physical examination should be fully documented with diagrams and pictures, if possible. This can help with longitudinally following healing of the burn and can assist greatly with recall in court if needed.

Conclusion

Neglect and abuse remain a difficult and emotionally charged topic. Occurring behind closed doors, it is unobserved and confessions are rare.[92] There are myriad presentations, and abuse and neglect may mimic other disease processes. While there is significant morbidity and mortality, the diagnosis and treatment are intertwined with legal issues of parental rights and family preservation.

Identifying children who are victims of abuse is a difficult and unpleasant part of pediatric health care. However, it also can be rewarding in that it can serve as the first step in saving or improving a child's life. Health care professionals must keep in mind that children are the victims of these horrible acts and that they are often silent victims. They must count on us to identify and rescue them. Don't forget to keep child abuse on the differential diagnosis of all childhood injuries.

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CME/CE Questions

1. Which of the following sites of injury is the least specific for child abuse?
   A. Scapular fractures
   B. Rib fractures
   C. Metaphyseal fractures
   D. Clavicular fracture

2. All of the following skull fractures more commonly are encountered in abusive rather than in accidental injury *except:*

CE/CME Instructions

Physicians and nurses participate in this continuing medical education/continuing education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to test their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. **After completing this activity, you must complete the evaluation form provided and return it in the reply envelope provided in order to receive a certificate of completion.** When your evaluation is received, a certificate will be mailed to you.

A. bilateral skull fractures.
B. fractures crossing suture lines.
C. isolated linear parietal skull fractures.
D. multiple skull fractures.

3. In a child with suspected physical abuse, the American College of Radiology recommends a full skeletal survey. This survey most appropriately is described by which of the following?
   A. A single frontal view of the entire child
   B. Anteroposterior and lateral views of the upper and lower extremities
   C. Multiple dedicated images of the axial and appendicular skeleton, including additional views as needed to document sites of injury
   D. Anteroposterior and lateral views of the skull plus a single anteroposterior view of the chest and abdomen

4. A 1-year-old girl has a history of not moving her arm for one day. The family denies any history of trauma. The child is acting normally and has no other evidence of acute injury. An x-ray reveals a midshaft humeral fracture. The next step in the work up of this child would be:
   A. to order a CT scan of the head to rule out a chronic subdural hematoma.
   B. to order liver enzymes to help rule out intra-abdominal injury.
   C. to obtain a skeletal survey to rule out other fractures.
   D. to apply a sling and swath and have the patient follow up with orthopedics.

5. Which one of the following statements concerning nonaccidental trauma is *not* true?
   A. Child abuse needs to be considered in all pediatric injuries.
   B. Subdural hematomas frequently are seen in children who fall out of bed.
   C. If the history does not seem to explain the identified injuries, the possibility of abuse should be considered.
   D. An unexplained delay in seeking medical care in an injured child is an indicator of nonaccidental trauma.

6. Which one of the following statements concerning nonaccidental head injury is *not* true?
   A. Inflicted head injury is the most common cause of traumatic death in infancy.
   B. Children with intentional injuries have worse functional outcome than children with unintentional injury.
   C. Infants will tolerate an acute brain injury better than adults because the fontanel is open.
   D. Head injuries in infants can be difficult to diagnose because the symptoms often are nonspecific.

7. When physicians fail to recognize that the child's symptoms are secondary to nonaccidental head injury, the child is frequently re-injured or has serious complications of the unrecognized, untreated head injury.
   A. True
   B. False

8. All of the following statements concerning imaging in nonaccidental pediatric head injury are true *except*:
   A. The most frequent presentation in nonaccidental trauma is a combination of subdural convexity and interhemispheric hematomas.
   B. MRI is useful in detecting small hemorrhages in infants with equivocal CT findings.
   C. The majority of subdural hematomas in children younger than 2 years are due to child abuse.
   D. Epidural hemorrhage is a common finding in shaken-infant syndrome.

9. Which one of the following statements regarding bruising in infants and children is *not* true?
   A. The dating and aging of bruises is precise.
   B. Some areas of the body such as the back, neck, and ears are atypical areas of accidental bruises.
   C. Mongolian spots often are confused with bruises.
   D. Children who are not yet walking do not have significant bruises on their bodies.

10. Severe abdominal injury is not a recognized manifestation of child abuse.
    A. True
    B. False

CME/CE Objectives

Upon completing this program, the participants will be able to:

a.) Quickly recognize or increase suspicion for non-accidental trauma in a pediatric patient;

b.) Be educated about rapid stabilization and management of a child who has sustained non-accidental injury;

c.) Understand the various diagnostic modalities available to recognize non-accidental trauma, and know the appropriate use of each modality;

d.) Integrate the ability to recognize suspicious injury patterns for non-accidental trauma into their clinical practice.

Answer Key:

1. D	6.C
2. C	7.A
3. C	8.D
4. C	9.A
5. B	10.B

In Future Issues: **ED Thoracotomy**