Chapter 10

Pancreatic disorders

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INTRODUCTION

Disorders of the digestive tract occur commonly in dogs and cats. The clinical signs involving diseases of the liver, pancreas, and bowel can show considerable overlap. This makes an accurate diagnosis a formidable challenge to the small animal practitioner.

Most experienced internists will attest that there is seldom anything pathognomonic about acute pancreatitis. Therefore, if a diagnosis is to be made, a thorough review of all the available clinical findings is necessary.

ACUTE PANCREATITIS

Definition/overview

Of the various clinical disorders treated by the small animal practitioner, there is probably none more difficult and frustrating to treat than acute pancreatitis. Despite the continuing acquisition of new knowledge regarding pancreatic physiology and pathophysiology, there is still no miracle treatment that can directly counteract the ravages of acute necrotizing pancreatitis. However, the optimal therapeutic outcome depends to a great extent on the clinician's working knowledge of the physiology, pathophysiology, clinical features, and medical and surgical treatments for this disorder in the dog and cat.

Etiology

Fortunately, the dog and cat are spared from many of the causes of pancreatitis that affect humans (**Table 10.1**). However, there are several general mechanisms that should be considered such as obstruction to the pancreatic duct, dietary factors, infectious agents, trauma, toxic drug reactions, metabolic abnormalities, and vascular alterations.

Pathophysiology

At the cellular level the calcium-dependent intra-acinar cell activation of pancreatic digestive zymogens, particularly proteases, is an early event in the initiation of acute pancreatitis. Activation of transcription factor NF-κB also occurs early in experimental pancreatitis. Early pathologic Ca^{2+} mobilization into acinar cells has a central role in the pathogenesis of acute pancreatitis. Another early acinar cell event is thought to be a decrease in compartmental pH. Other contributing factors to pathogenesis include neurally mediated inflammation and the production of large amounts of reactive oxygen species along with the simultaneous depletion of antioxidants.

The neutrophil migration that takes place is a result of the activation of trypsin, chymotrypsin, and oxygen radicals. The cytokines that are subsequently produced go on to cause the systemic inflammatory response syndrome. Other factors that augment the inflammatory response include altered pancreatic microcirculation, a shift from acinar cell apoptosis to necrosis, and the activation of other vasogenic pathways.

Prostaglandins are thought to have a key role in the pathogenesis of acute pancreatitis (**Figure 10.1**). Suspected mechanisms for these cyclo-oxygenase-2 effects include regulating heat shock protein 70 expression, inducible nitric oxide synthase activity, release of substance P,

Table 10.1 Some causes of acute pancreatitis in humans.

* Biliary tract disease
Ethanol abuse
Infectious agents (viral, bacterial, toxoplasmosis)
Peptic ulcer
Methanol
* Trauma, surgery
Scorpion bites (Trinidad)
* Vascular factors – ischemia, thrombosis
* Carcinoma of the pancreas
* Hyperlipoproteinemias (type I, IV, and V in humans)
* Hypotensive shock
* Hypercalcemia
* Ductal obstruction by tumors
* Drugs
* High-fat diet – dogs only
Hereditary pancreatitis, pancreas divisum

* Causes that have been implicated in dogs and cats

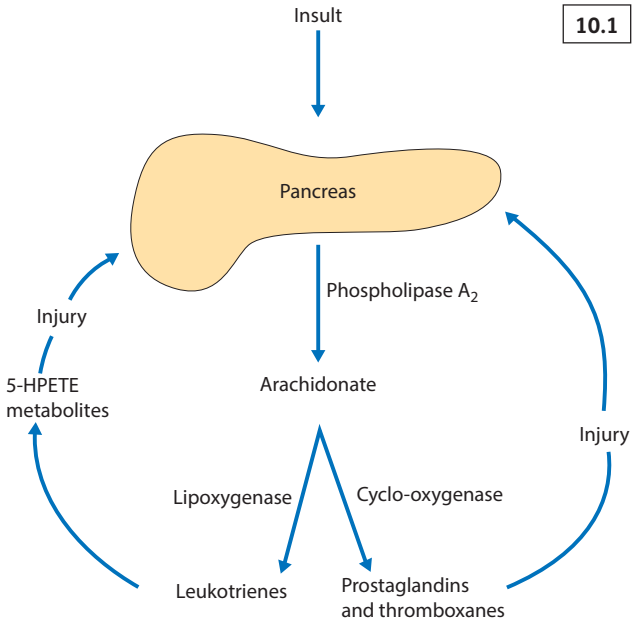


Figure 10.1 The role of prostaglandins in acute pancreatitis. Arachidonic acid metabolites are important mediators of inflammation in acute pancreatitis. 5-HPETE, 5-hydroperoxyeicosatetraenoic acid. (Source: Nager AB, Gorelick FS (2004) Acute pancreatitis. *Current Opinions in Gastroenterology* **20(5):**439–443.)

and neutrophil function. A number of factors that can cause the described disturbances of cellular metabolism will cause increased permeability of cellular lipoprotein membranes surrounding the lysosomal hydrolases in the acinar cells, with resultant inappropriate proenzyme activation and autodigestion. At first the antiproteases will attempt to counteract the released proteases, but eventually the activation of the pancreatic proteolytic enzyme cascade will prevail and cause clinical acute pancreatitis. The characteristics of activated pancreatic enzymes and their effects on the pancreas and other tissues are shown in **Table 10.2** and illustrated pathologically in **Figures 10.2–10.22**.

Marked hypotension can be observed in dogs and cats with acute pancreatitis, and it is probably the main contributing factor to their demise. Studies of the hemodynamic consequences of severe pancreatitis in humans demonstrate that the cardiac index is increased and the systemic vascular resistance is decreased; these findings are similar to those in patients with sepsis. The mechanisms responsible for these effects involve various inflammatory mediators including interleukin (IL)-1, IL-2, IL-6, IL-8, tumor necrosis factor (TNF), platelet activating factor, and interferon gamma. Circulating vasoactive

Table 10.2 Characteristics of the pancreatic enzymes.*

Enzyme	Substrate	Effects
Lipase	Triglyceride	Fat necrosis Hypocalcemia Cell membrane damage
Phospholipase A_2	Cell membranes Phosphatides	Lysophosphatide formation Membrane destruction Vascular leakage Acute respiratory distress syndrome
Trypsin	Other proenzymes Kallikreinogen Scleroproteins	Coagulation necrosis Vascular leakage, shock Proteolysis Coagulopathy Kinin release
Chymotrypsin/ carboxypeptidase	Scleroproteins	Coagulation necrosis Proteolysis Vascular leakage
Elastase	Scleroproteins Elastic/collagen fibers of blood vessels	Coagulation necrosis Elastocollagenolysis Proteolysis Vascular leakage Hemorrhage
Kallikrein	Kinins	Kinin release

Modified from Büchler MW, Uhl W, Malfertheiner P *et al*. (2004) (eds.) *Diseases of the Pancreas*. Karger, Basel.

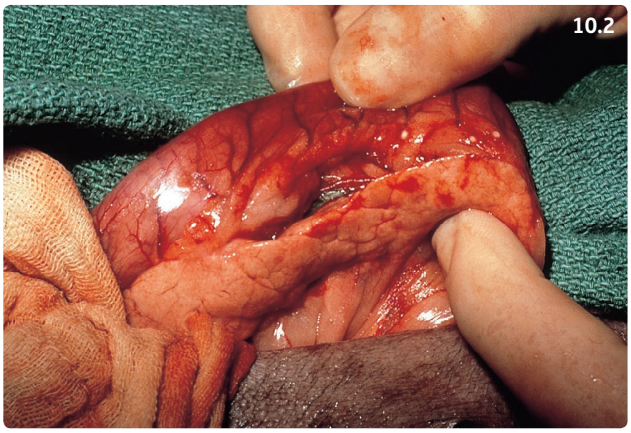


Figure 10.2 This 7-year-old male Poodle was taken to surgery after a 5–7-day period of medical treatment accompanied by persistent vomiting. Shown is edematous pancreatitis.

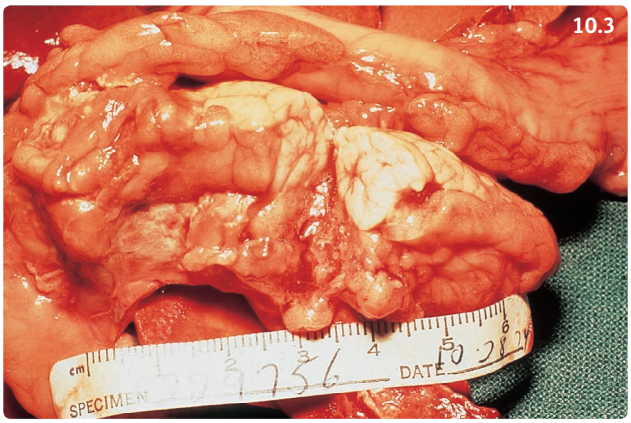


Figure 10.3 Postmortem specimen illustrating edematous pancreatitis and extensive peripancreatic fat necrosis. This patient was a 4-year-old female Yorkshire Terrier that also had diabetic ketoacidosis and renal failure.

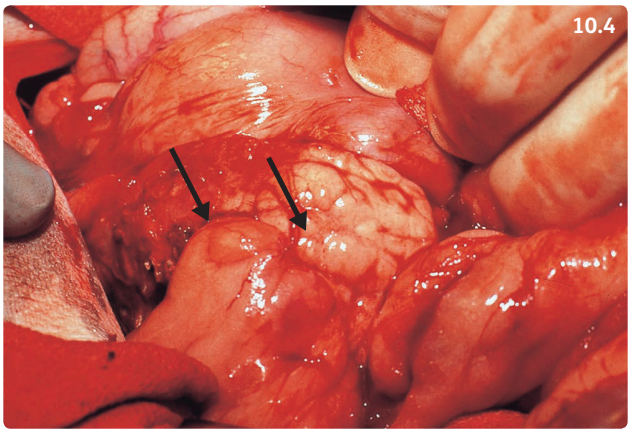


Figure 10.4 Surgical view of hemorrhagic necrotic pancreatitis in a 10-year-old male Wirehaired Fox Terrier. Note how one half of the pancreas is hemorrhagic and the other half is edematous (arrows).

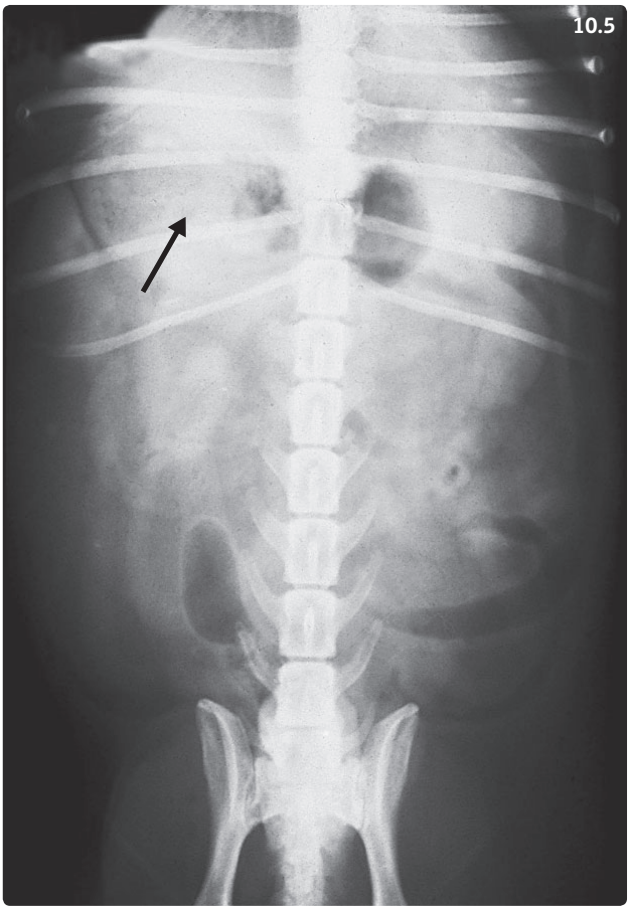


Figure 10.5 Radiograph, taken from the dog in Figure 10.4, typical of acute pancreatitis, showing increased fluid density in the right upper abdominal quadrant (arrow) and lateral displacement of the duodenum.

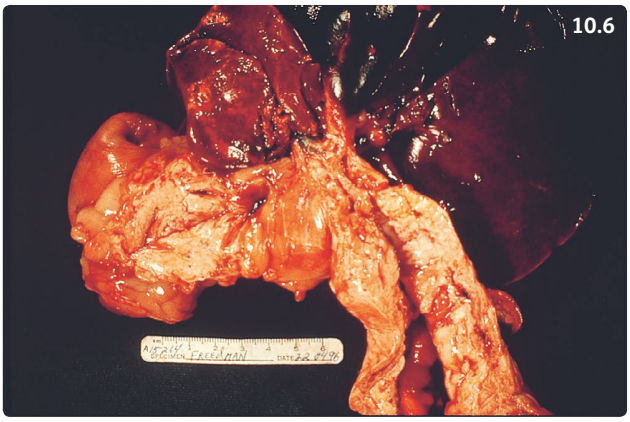


Figure 10.6 Over the ensuing year, the dog in Figures 10.4 and 10.5 had several episodes of relapsing pancreatitis that eventually terminated with renal shutdown. This postmortem specimen shows chronic pancreatic scarring along with recent necrotic changes.

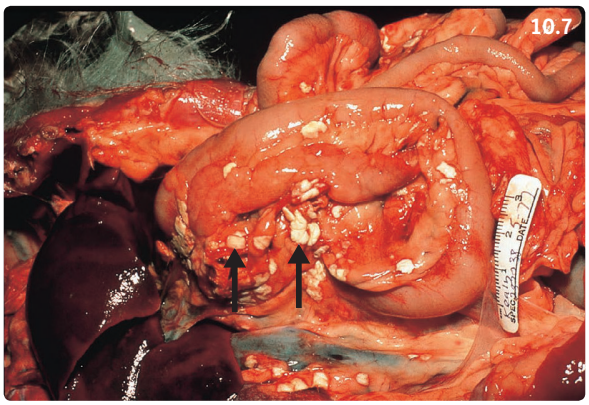


Figure 10.7 Postmortem view of the abdomen of a 13-year-old female Lhasa Apso showing diffuse edematous pancreatitis accompanied by diffuse calcium soap deposition (arrows) throughout the mesentery and omentum.

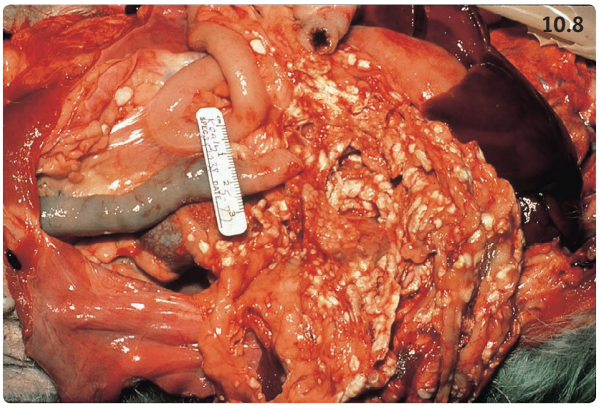


Figure 10.8 Another view of the specimen shown in Figure 10.7. This calcium soap formation is one of the accepted explanations for the lowered serum calcium level that can accompany acute pancreatitis.

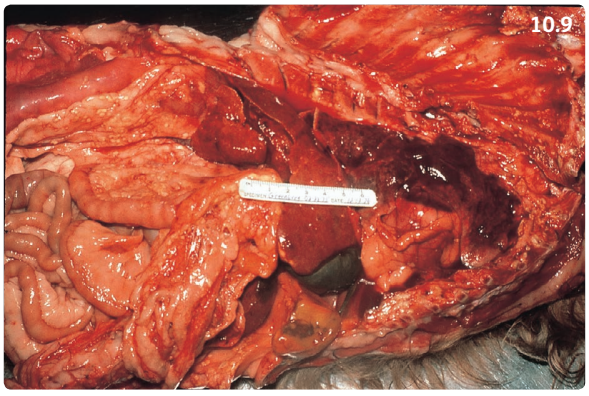


Figure 10.9 This 14-year-old male Dachshund had pleural effusion along with acute pancreatitis. Shown is the serosanguineous pleural effusion that is accompanied by calcium soap deposits on the pleural membranes underlying the thoracic viscera.

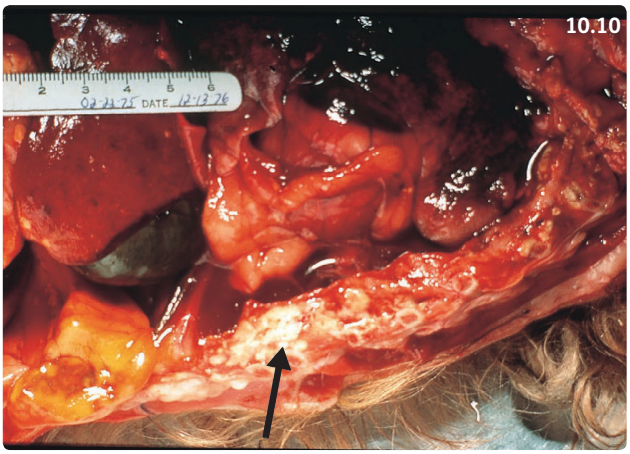


Figure 10.10 The same dog as in Figure 10.9, showing the intercostal muscles with calcium soap deposits (arrow).

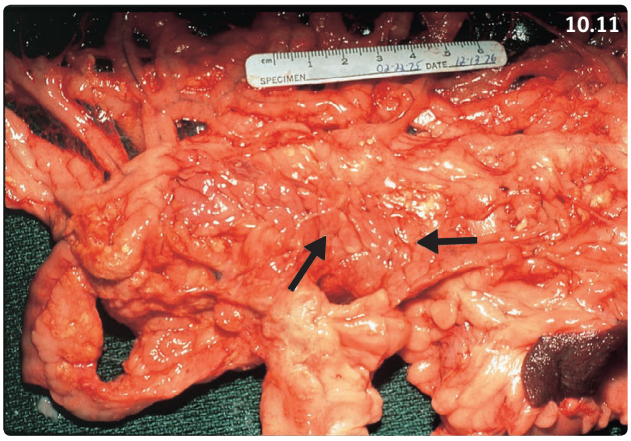


Figure 10.11 The dog in Figures 10.9 and 10.10 had edematous pancreatitis (arrows).

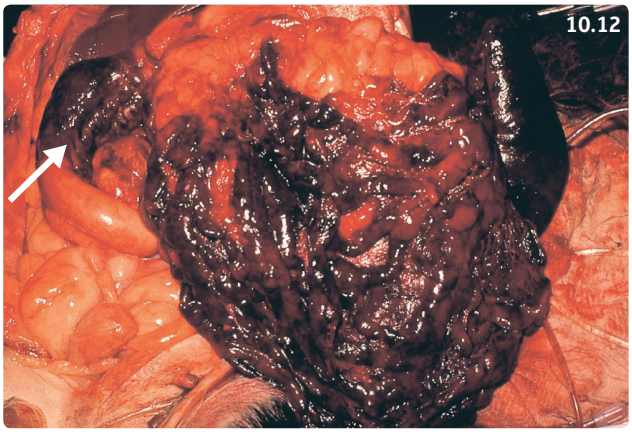


Figure 10.12 Postmortem findings of severe hemorrhagic pancreatitis complicated by pathologic coagulation causing infarction to the duodenum (arrow) and omentum.

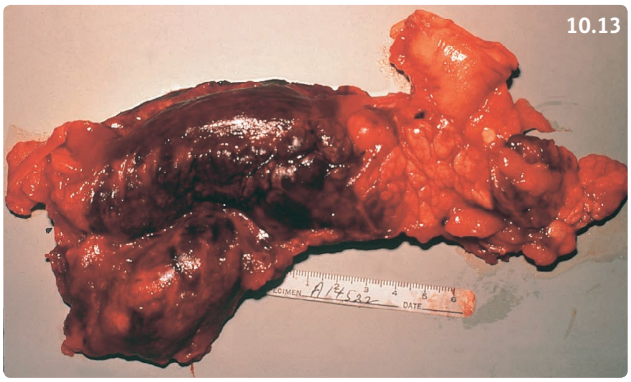


Figure 10.13 Marked pancreatic necrosis in the specimen shown in Figure 10.12. The peripancreatic lymph node is markedly enlarged from hemorrhagic necrosis.

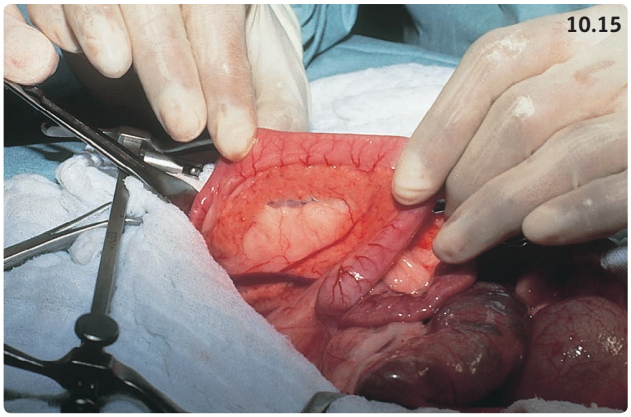


Figure 10.15 Surgical view of a 16-year-old cat with chronic active pancreatitis. This cat had been hospitalized 3 years earlier with acute pancreatitis, which responded well to conservative treatment.

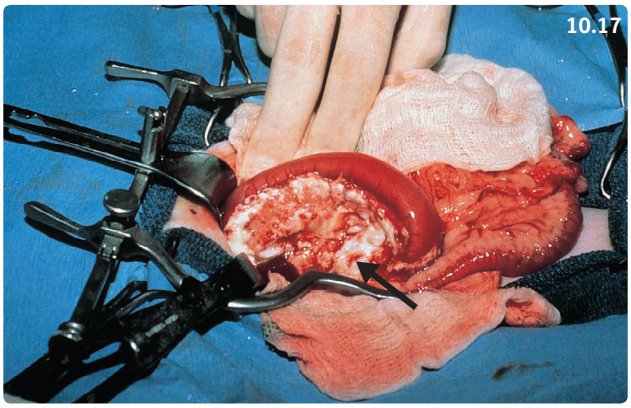


Figure 10.17 After 7 days of medical therapy, this 12-year-old female cat was taken to surgery because it showed no signs of improvement. Shown is hemorrhagic pancreatitis accompanied by extensive peripancreatitic and mesenteric calcium soap formation (arrow). Postoperatively, nutrition was provided through a jejunostomy tube that was inserted at the time of surgery. This cat survived after 3 weeks of illness.

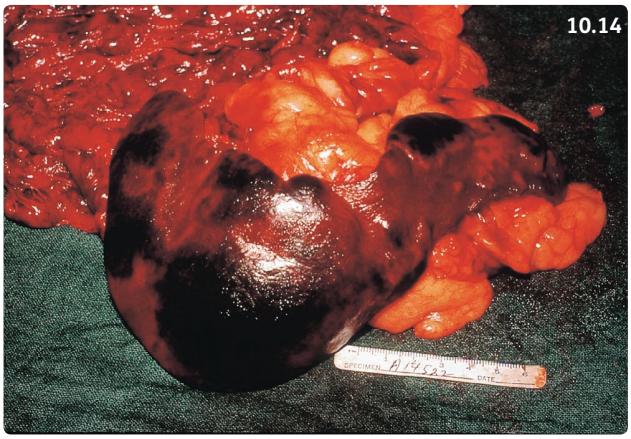


Figure 10.14 Multiple splenic infarcts in the 9-year-old dog in Figures 10.12 and 10.13. It was taken to surgery where it expired. Disseminated intravascular coagulation was the cause of the hypercoagulable state.

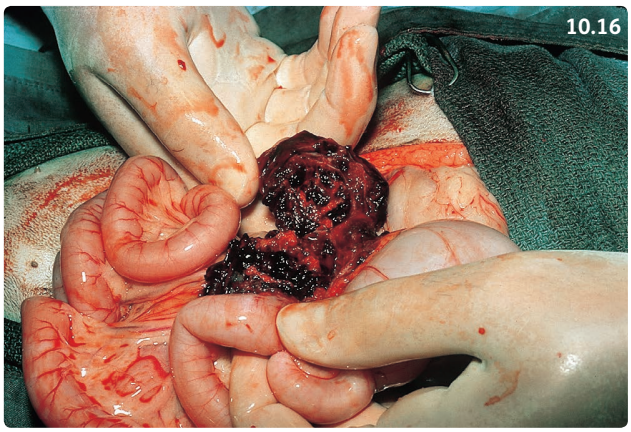


Figure 10.16 This 10-year-old cat was taken to surgery for the primary problems of vomiting, depression, and a hemorrhagic abdominal effusion. Shown is hemorrhagic pancreatitis. Copious surgical lavage and the insertion of abdominal drains until 2–3 days postoperatively would be the usual surgical measures for this type of pathology.

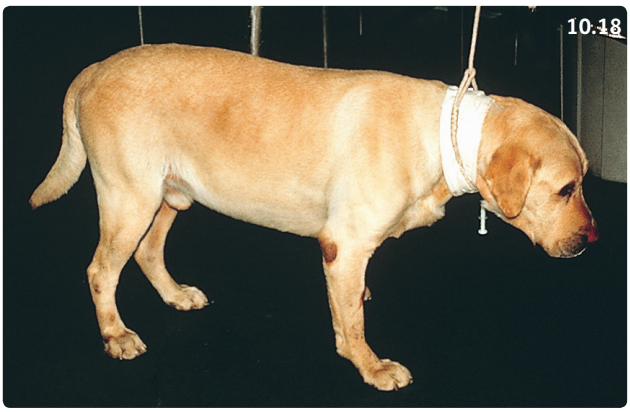


Figure 10.18 This 5-year-old male Labrador Retriever had vomiting, fever, depression, and abdominal pain.

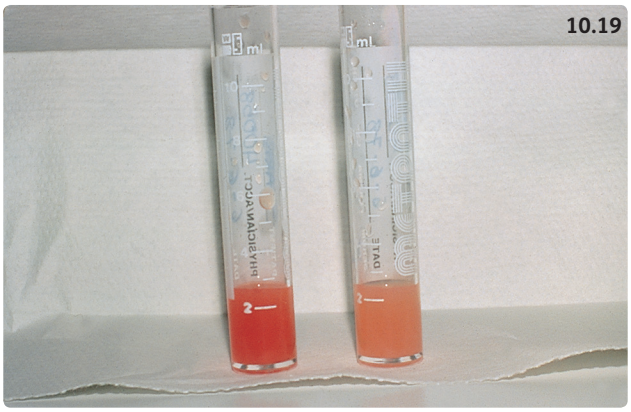


Figure 10.19 Blood samples taken from the dog shown in Figure 10.18 were markedly lipemic. The patient died despite intensive medical efforts.

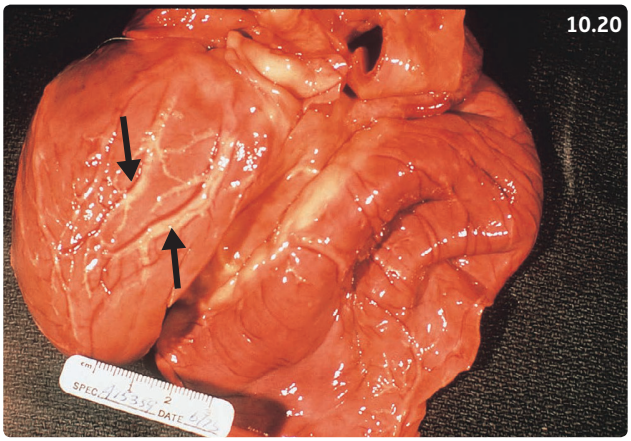


Figure 10.20 Postmortem examination of the dog in Figure 10.18 showed diffuse thyroid atrophy. The severe atherosclerotic vascular lesions shown here involved the coronary arteries (arrows).

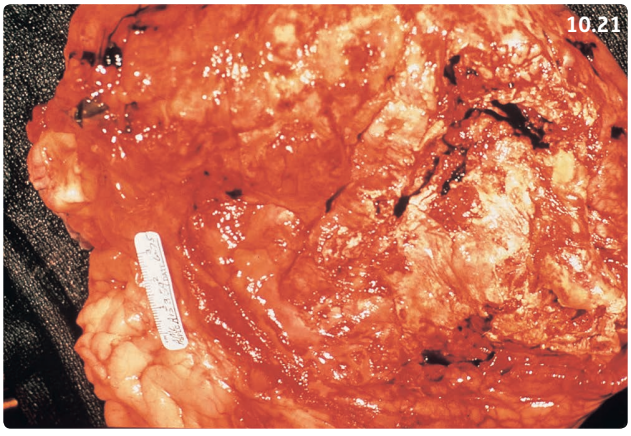
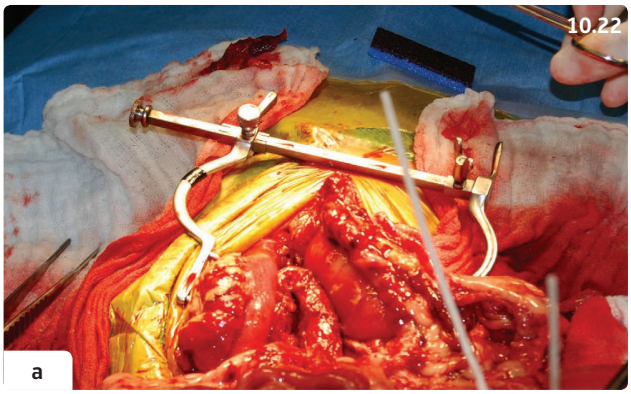
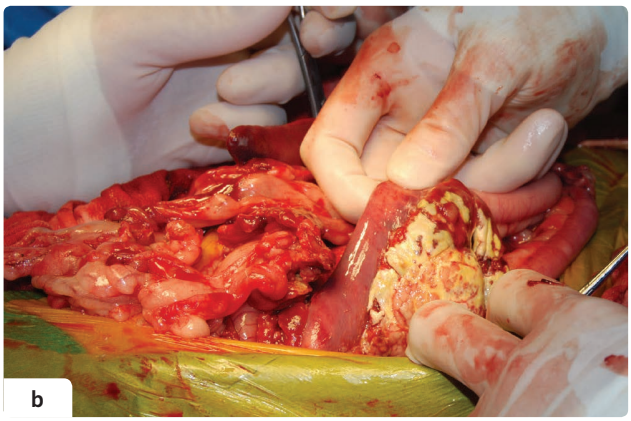


Figure 10.21 There was also extensive pancreatic phlegmon formation in the dog shown in Figure 10.18. The primary cause of this dog's pancreatitis was hypothyroidism, which caused marked hyperlipidemia; this, in turn, could have triggered the acute pancreatitis. Hyperlipidemia is a well-known common cause of acute pancreatitis in humans.





Figures 10.22a, b (a) Pancreatic phlegmon is illustrated in this surgical view showing extensive parapancreatic soft tissue adhesions. (b) The pancreas from the same dog showing extensive saponification.

com-pounds, such as bradykinin and myocardial depressant factor (in the dog), resulting from pancreatic necrosis, also contribute to the vasomotor instability. The low blood pressure may also be due to sequestration of fluid from the plasma space into the 'third spaces' of the peritoneal cavity and retroperitoneum. Experiments in the dog have shown that approximately 35% of the total plasma volume can be lost from the circulation 4 hours after the induction of acute pancreatitis. The local and systemic effects of acute pancreatitis are shown in **Figure 10.23**.

Clinical presentation

Most occurrences of acute pancreatitis in the dog involve middle aged, obese females; however, dogs with normal weight and male dogs can also be affected. The most common historical signs involve a sudden onset of vomiting, anorexia, and mental depression. Some occurrences reportedly follow the ingestion of a fatty meal, although this might not be a consistent finding. Initially, the vomitus might contain partially undigested food and this may be followed subsequently by vomitus consisting of bile and

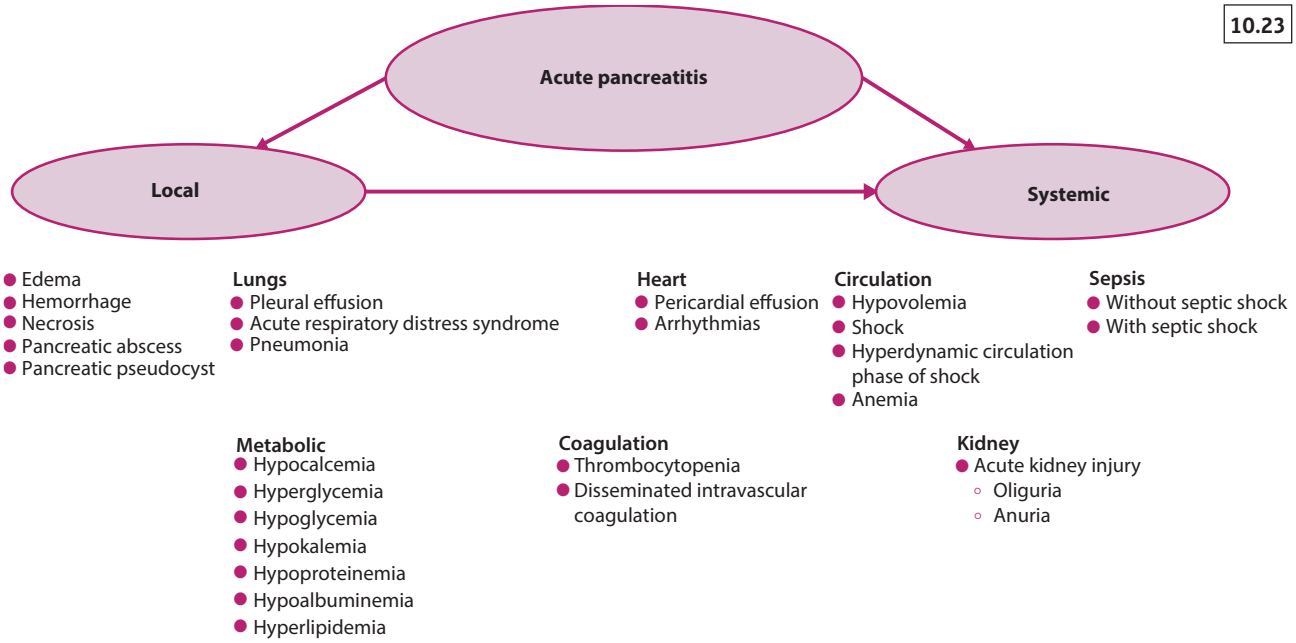
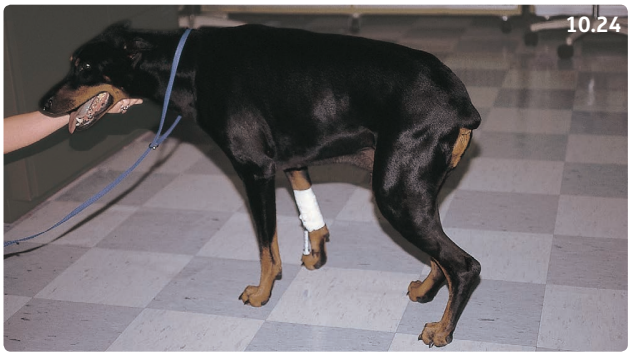


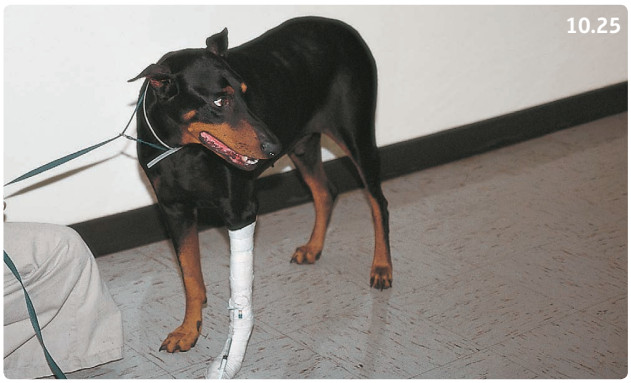
Figure 10.23 Pathophysiologic consequences of acute pancreatitis. The adverse effects of acute pancreatitis are far reaching, with involvement of several organ systems. In many cases this can lead to multiple organ dysfunction and the patient's demise. (Modified from Büchler MW, Uhl W, Malfertheiner P *et al*. (2004) *Diseases of the Pancreas*. Karger, Basel.)

watery mucus. After the initial vomiting, the dog might show regurgitant movements only because of nausea. Its attitude will vary from mild to marked depression, and its posture will either be normal, upright with abdominal tucking, or lateral recumbency depending on the degree of pain and hypovolemia (**Figures 10.24–10.28**). Diarrhea might occasionally occur, but scant or absent feces is more common due to the peritonitis-induced ileus.

The age of cats affected with acute pancreatitis can range from young to old, although in one study the majority of the cats were older than 8 years. Many have normal body weight. The clinical signs are similar to those in the dog, although vomiting might be occasionally absent and the clinical presentation much more subtle (**Figures 10.29, 10.30**).

The physical examination findings vary with the severity of the problem. Dogs and cats with mild pancreatitis might only show mild mental depression, normal vital signs, and equivocal palpable abdominal tenderness. Signs associated with the hemorrhagic necrotic form include: marked mental depression; fever; hypotension with accompanying tachypnea, tachycardia, and weak femoral pulse; a painful abdomen; and a moderate to marked degree of dehydration. Abdominal palpation in the cat can sometimes detect bowel adhesions (**Figure 10.31**). One retrospective study involving 40 cats reported the most common signs being severe lethargy, anorexia, and dehydration. Histopathology identified





Figures 10.24, 10.25 This 5-year-old male Dobermann displays an abnormal posture from abdominal pain caused by acute pancreatitis. After 3 days of treatment the dog was pain free and its posture was normal.

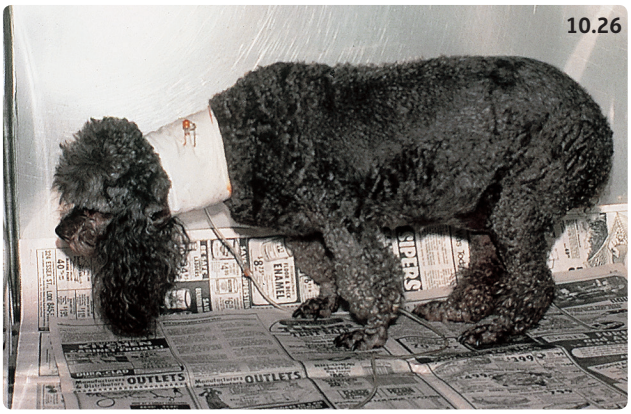


Figure 10.26 The clinical signs of acute pancreatitis can vary from mild to marked. This 13-year-old female Poodle had acute vomiting, mental depression, and marked abdominal pain peracutely immediately after dinner. Despite the use of intensive fluid therapy, the dog died from hemorrhagic necrotic pancreatitis.

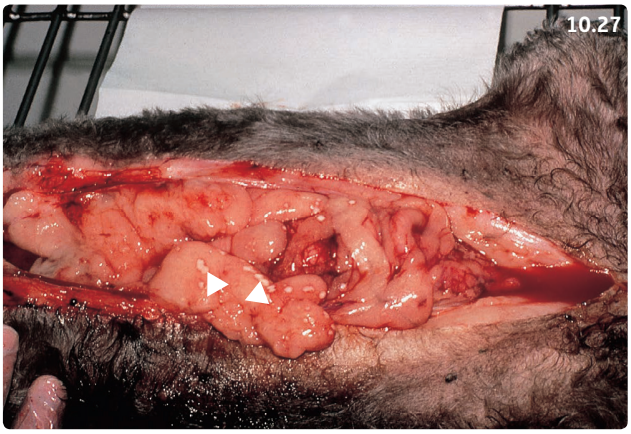


Figure 10.27 Postmortem examination of the dog in Figure 10.26. Shown here are calcium soap deposits (arrowheads) on the abdominal fat and pancreatic ascites.

acute necrotizing pancreatitis and acute pancreatic necrosis as the two main types.

Clinically detectable icterus will not occur initially with pancreatitis, but it might be evident by the third day and usually results from cholestasis; bile duct obstruction occurs rarely. Abdominal distension can result from paralytic ileus. A reddish-brown colored ascitic fluid (pancreatic ascites) can sometimes accumulate with hemorrhagic necrotic pancreatitis. A guarded to grave prognosis should always be given to pancreatitis patients that assume a lateral recumbent posture and have mental dullness, oliguria/anuria, and hypotension that is resistant to treatment. A more detailed and practical severity scoring system is provided in **Table 10.3**.

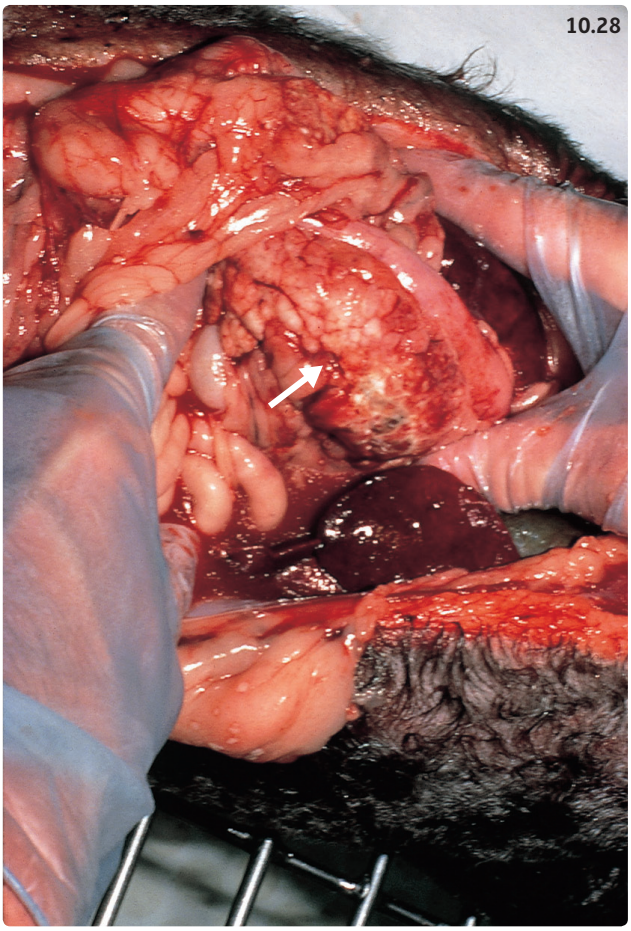


Figure 10.28 Severe hemorrhagic pancreatic necrosis (arrow) in the postmortem examination of the dog shown in Figure 10.26.

Figure 10.29 Pancreatitis in cats is typically accompanied by anorexia, mental depression, and marked inactivity. Although many cats will vomit, there are those who will not. Most cats with pancreatitis will show abdominal tenderness and varying degrees of radiographic pathology. A definitive diagnosis can only be obtained by visualizing the pancreas grossly with or without biopsy and histopathology.

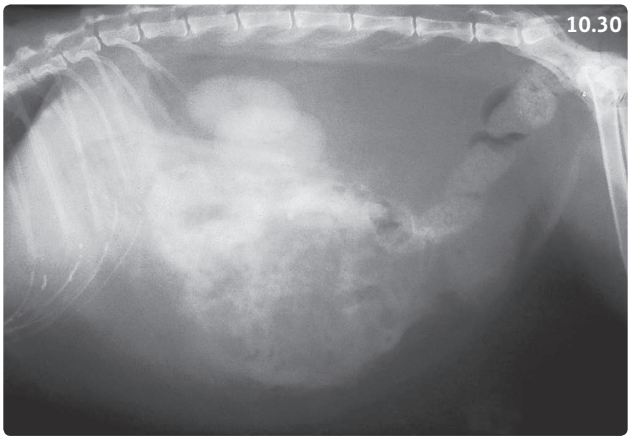


Figure 10.30 Lateral abdominal radiograph of the cat in Figure 10.29, which presented with acute pancreatitis, showing a diffuse abdominal effusion and a 'gathering effect' of the small bowel. These lesions were evident at surgery (see Figure 10.31). The abundant amount of retroperitoneal fat causes ventral depression of the descending colon.

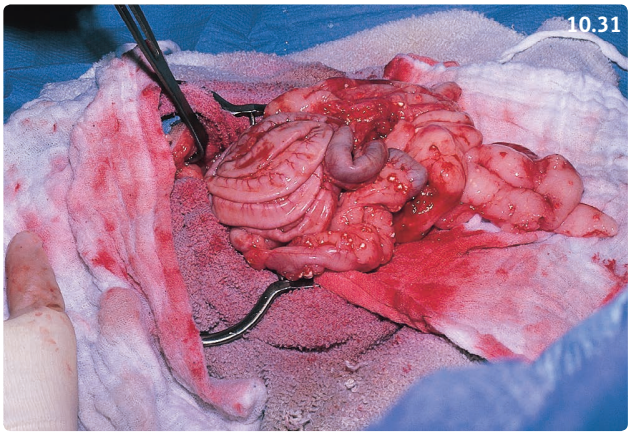


Figure 10.31 This surgical view of a 16-year-old female Domestic Shorthair cat shows small bowel adhesions, which were detectable on abdominal palpation. Also shown are diffuse calcium soap deposits on the parietal and visceral peritoneal surfaces. These deposits are pathognomonic of acute pancreatitis.

Table 10.3 Practical severity scoring in dogs.

Disease severity	Score*	Prognosis	Clinical presentation and typical therapy
Mild	0	Excellent	Often resolves spontaneously. Recovery is uncomplicated. Managed as an outpatient, if hydration status is good. Intravenous fluids can be given if necessary. Pancreatic rest (no food for 1–2 days if vomiting or nauseous) and/or pain control is usually all that is required. Maropitant will help the vomiting and nausea
Moderate	1	Good to fair	Usually dehydrated – the renal system is most often compromised (prerenal azotemia). Treatment involves the administration of crystalloids at twice the maintenance rate together with electrolytes. Nothing by mouth until vomiting stops, with analgesia and maropitant as appropriate. Recovery is usually uncomplicated provided adequate fluid therapy is given. If anorexia lasts for more than 2 days, consider additional nutritional support
	2	Fair to poor	Dehydrated, hypovolemic, often prerenal azotemia and degenerative left shift leukocytosis. Animals usually recover with intensive therapy, but may have to be euthanized for financial reasons. Intravenous crystalloids (initially administered as per treatment for shock) followed by colloids, with or without plasma, in many cases. Monitor urine output, renal function, and lung sounds. Control pain and consider special nutritional support. Monitor coagulation status carefully and intervene early with fresh frozen plasma and heparin, if necessary. May need referral if there is a poor response to initial therapy
Severe	3	Poor	Extensive therapy and life support is required with constant monitoring. Early referral is advised. Surgical intervention and peritoneal lavage may be necessary. Ventilatory support, central venous pressure monitoring, and high volume fluid therapy are usually needed. Jejunostomy feeding or total parenteral nutrition is often required. Most patients die and euthanasia may have to be considered. The pathology in these patients can include extensive pancreatic necrosis, pancreatic abscess, phlegmon formation, and macroscopic vascular compromise caused by regional infarction
	4	Grave	

Modified from Ruaux and Atwell (1998) and Ruaux (2000).
*The severity scoring system is based on the number of organ systems, apart from the pancreas, that show evidence of failure or compromise at initial presentation.

Table 10.4 Differential diagnosis of acute pancreatitis in the dog and cat.

Acute gastroenteritis
Intoxications
Blunt abdominal trauma
Gastrointestinal obstruction
Gastrointestinal perforation
Intestinal volvulus
Intestinal ischemia and infarction
Emphysematous cholecystitis
Ruptured organs (e.g. uterus, urinary bladder, gallbladder)
Acute kidney injury
Acute hepatopathy

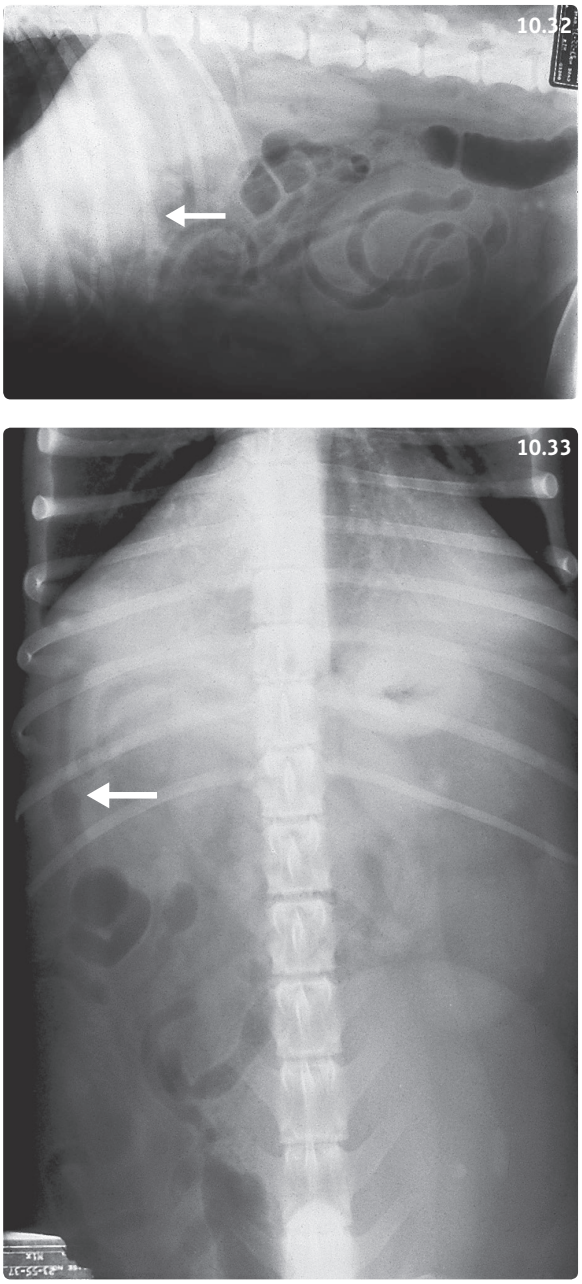
Differential diagnosis

The initial differential diagnosis of acute pancreatitis includes a variety of clinical disorders (**Table 10.4**). The list is extensive because the signs mimic any number of acute abdominal syndromes. Several are surgical emergencies that require rapid diagnosis and treatment.

Diagnosis

Imaging findings

The two most commonly used modalities are radiology and ultrasonography. Abdominal radiographs of dogs and cats with acute pancreatitis can show several abnormalities. In the mild edematous form the findings can range from normal to mild ileus involving the stomach and duodenum. The more severe forms cause the following changes as a result of the peritonitis: increased fluid density with loss of visceral detail in the anterior abdomen, right-sided lateral displacement of a gas-distended duodenum, and gastric distension (**Figures 10.32–10.34**). In the dog, visualization of the right kidney becomes apparent on the ventral dorsal projection of the abdominal radiograph (**Figures 10.35a, b, 10.36).** This finding has been demonstrated in many dogs in the author's experience and the results corroborated at surgery or at necropsy. The exact mechanism for this phenomenum has not been thoroughly explained, but local adhesions associated with the regional peritonitis might be major factors. In addition to these classic findings, acute pancreatitis can also cause radiographically demonstrable pleural effusion and pulmonary fluid accumulations, as shown in the pathologic illustrations (see **Figures 10.9, 10.10**). In the most advanced stages of this disease, the acute respiratory distress syndrome can occur as a result of the systemic inflammatory response syndrome. The outcome associated with this syndrome is usually ominous (**Figures 10.37a–c**). In cats, pleural and pericardial effusions have been described.



Figures 10.32, 10.33 Lateral and ventrodorsal abdominal radiographs of a dog illustrating several abnormalities indicative of acute pancreatitis. The lateral projection shows a loss of detail in the anterior mid abdomen and an increased fluid density in the anterior abdomen just caudal to the liver (arrow). The ventrodorsal view shows an increased right upper abdominal fluid density along with duodenal ileus and lateralization (arrow).

Ultrasonography is very useful in detecting pathology associated with pancreatitis in the dog and cat. While the normal pancreas is seldom visualized, the inflamed organ in the dog acquires an increased hypoechogenicity.

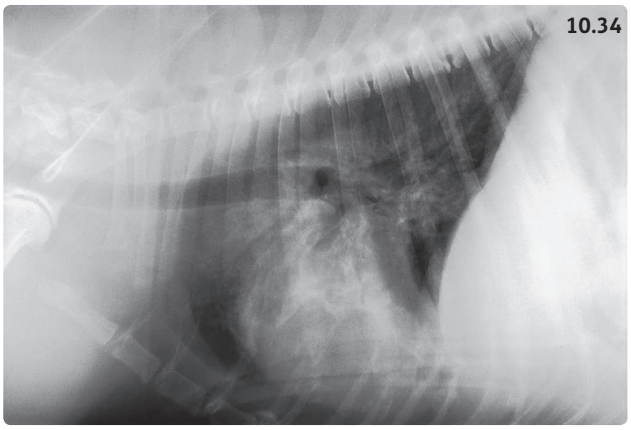


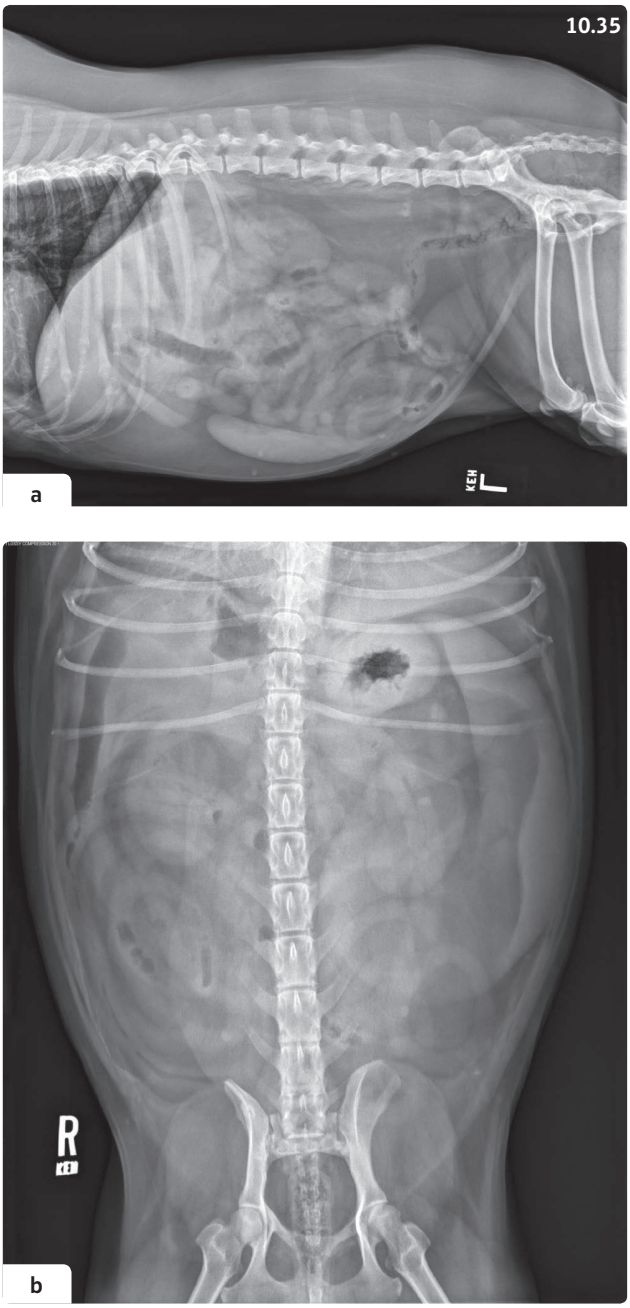
Figure 10.34 Lateral thoracic radiograph from an adult Labrador Retriever showing a pleural effusion as well as pulmonary infiltrate. The dog had hemorrhagic necrotic pancreatitis and secondary acute respiratory distress syndrome.

In the presence of peripancreatic fat inflammation, the perimeter of the pancreas becomes hyperechogenic. Additional findings in the dog include pancreatic enlargement and irregularity, peritoneal effusion, and evidence of extrahepatic biliary obstruction. Inflammation of the right pancreatic limb can cause the duodenum to appear thickened. One study describing the ultrasonographic findings of acute pancreatitis in cats included hypoechogenic pancreas, hyperechoic peripancreatic mesentery, peritoneal effusion, pancreatic enlargement, hyperechoic hepatomegaly, and mixed pancreatic hypo- and hyperechogenicity. Pancreatic anechoic pseudocyst and abscess formation and pancreatic ascites can also be detected with ultrasonography (**Figures 10.38, 10.39, 10.40a–d**). Overlying distended bowel loops are the major limitation to this imaging technique, but in most instances abdominal ultrasonography is a helpful diagnostic modality (**Figures 10.41–10.44**).

Clinicopathologic findings

The characteristic laboratory test abnormalities of acute pancreatitis in dogs and cats have been described. These are listed in **Table 10.5** and illustrated in **Figure 10.45**. In cats the serum biochemical profile is variable, ranging from normal to abnormalities involving renal, liver, glucose, protein, and electrolyte parameters. The hemogram often indicates an inflammatory response. One study of 46 cats with pancreatitis suggests that low plasma ionized calcium concentration is common and is associated with a guarded to grave prognosis, especially when ionized calcium concentrations are ≤1.0 mmol/l (4.0 mg/dl).

Choosing between amylase or lipase as a diagnostic test has been a subject of controversy for several years,



Figures 10.35a, b Lateral and ventrodorsal radiographs of a dog with acute pancreatitis showing a summation soft tissue fluid effect in the cranial abdomen on the lateral view. The ventrodorsal view clearly shows the right kidney.

because both enzyme levels can be normal or elevated from other conditions despite the presence of acute pancreatitis, thus causing them to lack both sensitivity and specificity. Increases in serum amylase levels to 3–5 times normal are strongly suspicious of acute pancreatitis in the appropriate clinical setting.

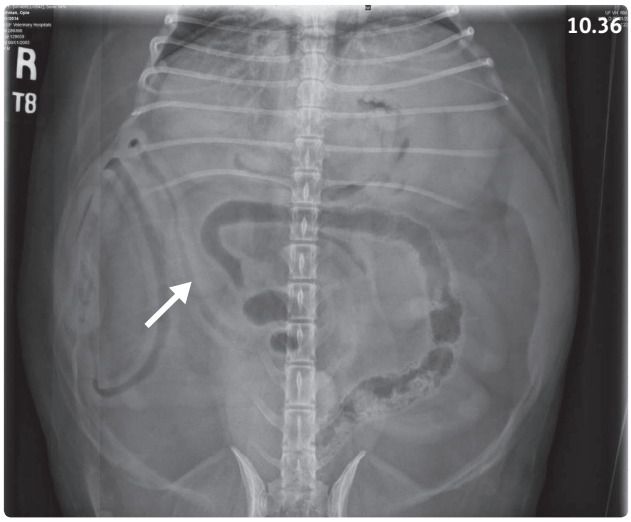


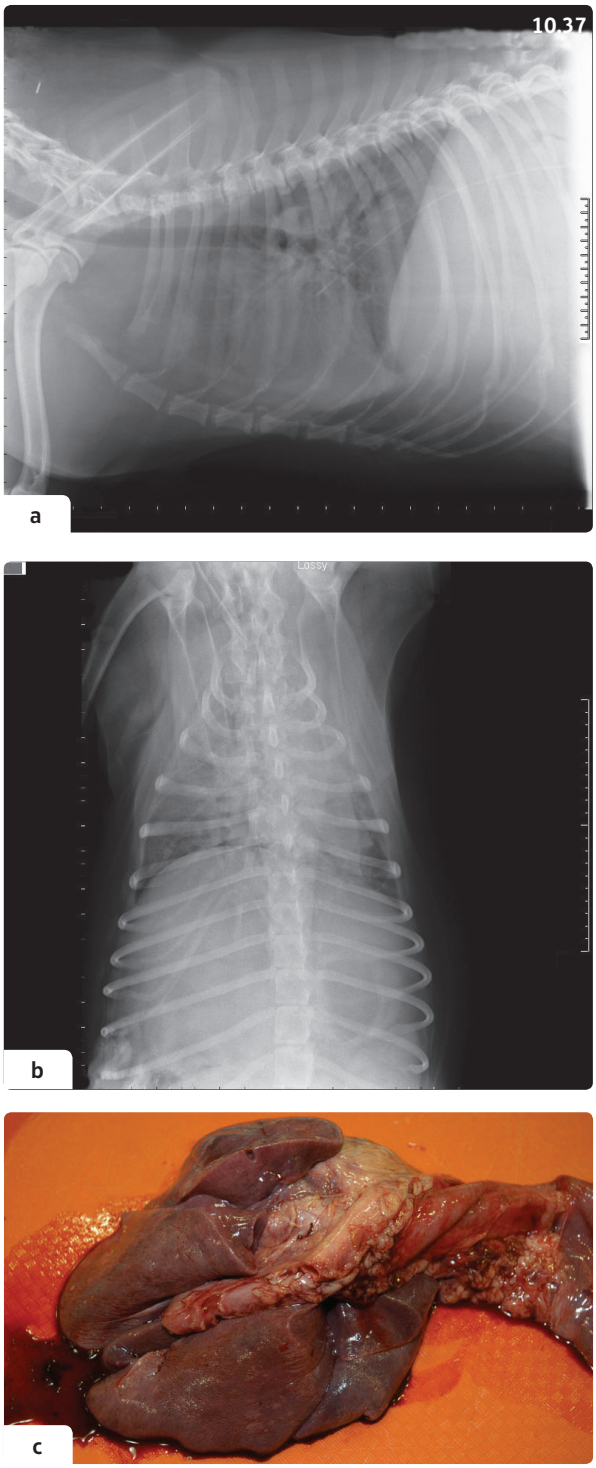
Figure 10.36 Ventrodorsal radiograph of another dog with acute pancreatitis showing a highly visible right kidney outline (arrow).

Serum lipase level has been reported to be more reliable than amylase for diagnosing acute pancreatitis in the dog. However, lipase as well as amylase may be elevated in patients with serious abdominal illnesses such as hepatopathies and renal and neoplastic disease. Serum amylase and lipase activities can be normal or even decreased despite the presence of serious pancreatic damage because of rapid renal clearance and diminished quantity after the initial elevation. The interpretation of serum amylase and lipase results should always be made solely within the context of the patient's other ongoing clinical findings.

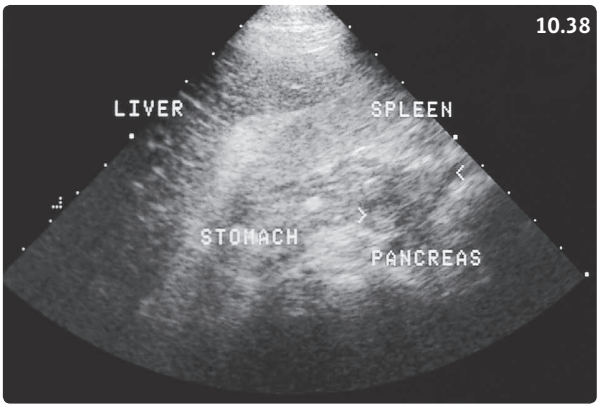
In feline pancreatitis the appearance of abnormally elevated serum amylase and lipase levels is typically variable and should never be the sole criteria for diagnosis. Normal levels of both enzymes commonly occur in the cat despite the presence of acute pancreatitis. A two-fold or more increase in serum amylase and lipase activity in the presence of normal renal function is suggestive of acute pancreatitis when used with other supportive clinical findings.

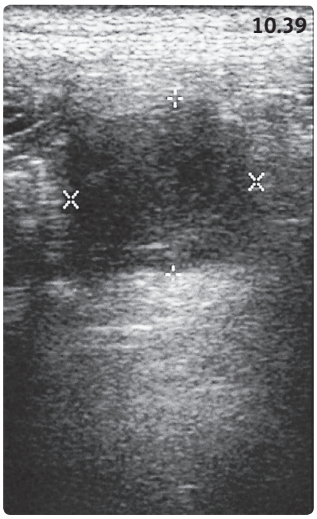
When the feline trypsin-like immunoreactivity (TLI) concentration test first became available, there was a false dependence on its supposed accuracy. It is no longer recommended as a diagnostic test for acute pancreatitis in cats.

In dogs the canine pancreatic lipase immunoreactivity (PLI) test is supposed to be fairly sensitive for diagnosing acute pancreatitis. The diagnostic cut-off value is 200 µg/l. This test can now be done in-house with a commercially available kit in addition to it being done in commercial laboratories. A recent (2012) multi-institutional study evaluating the canine pancreatic lipase immunoassay and the point of care (SNAP®) assay, showed that the tests



Figures 10.37a–c Thoracic radiographic images (a, b) from a dog that has severe acute pancreatitis with a pancreatic abscess. The dog acquired acute respiratory distress syndrome (ARDS) and was eventually euthanized. The radiographs, taken during the final days, show diffuse pulmonary infiltrate and some pleural effusion. The lungs as seen at necropsy (c) were consolidated and the histopathology features confirmed ARDS.





Figures 10.38, 10.39 Abdominal ultrasonograms from a 5-year-old male Irish Setter taken 2 weeks after a bout of acute pancreatitis. (10.38) General orientation of the dog's upper abdomen showing an anechoic area in the pancreas. (10.39) More detailed view of the pancreatic cavitation. The transabdominal fine needle aspiration yielded a dark colored fluid that was composed of sterile amorphous debris. All of the 'cystic' fluid was removed by aspiration and the dog recovered well.

have higher sensitivities and specificities than the serum amylase and lipase tests. However false-positive test results are still possible with both of these tests and their results should be interpreted along with other diagnostic modalities such as imaging.

There is also a feline PLI test that adds to the clinician's diagnostic acumen for acute pancreatitis in the cat. This feline pancreatic lipase test (fPLI) has been shown to have greater diagnostic accuracy than the feline TLI test. The same caution regarding interpretation of this test in the dog should be used when using this test in the cat. It is essential to realize that there is no one laboratory test that is 100% accurate in diagnosing acute pancreatitis in the dog and cat and that the newly available PLI tests are simply another diagnostic tool that can help lead to a diagnosis. The gold standard test for diagnosing acute pancreatitis in veterinary patients is gross observation of the pancreas with or without biopsy and histopathology.

Management

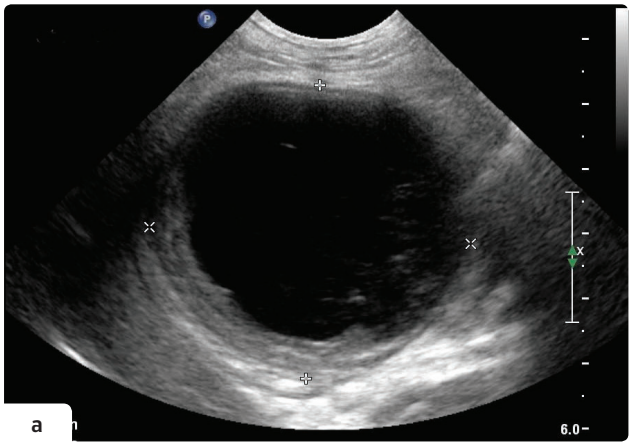
Medical therapy

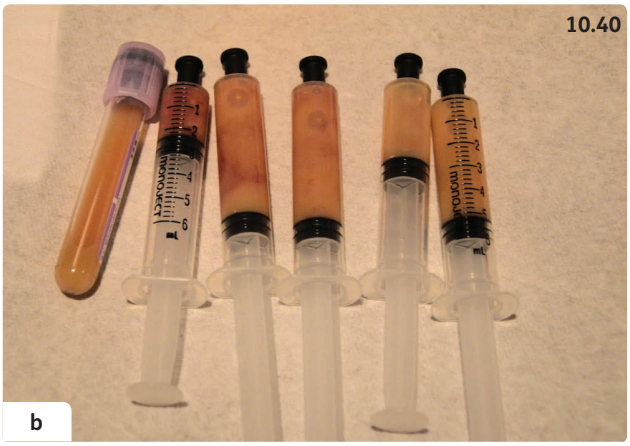
Therapy entails the restriction of oral intake (nothing by mouth) if the animal is vomiting profusely, while providing fluid and electrolyte needs by parenteral means. The steps listed in **Table 10.6** provide guidelines for treating the more severe form of pancreatitis.

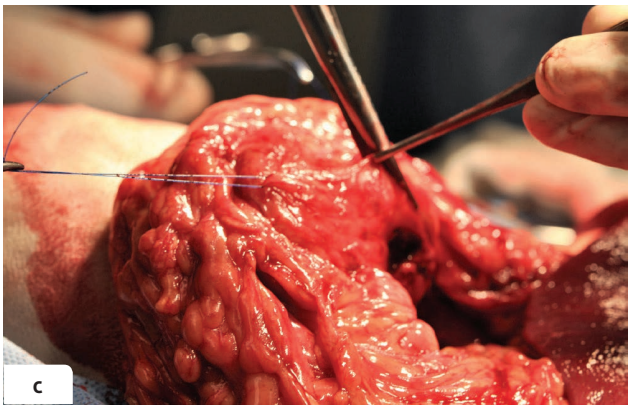
Patients who have suspicious yet mild historical and physical signs with unremarkable laboratory test results can often be treated conservatively with small feedings for 1–2 days, if they will eat voluntarily, and periodic offerings of water.

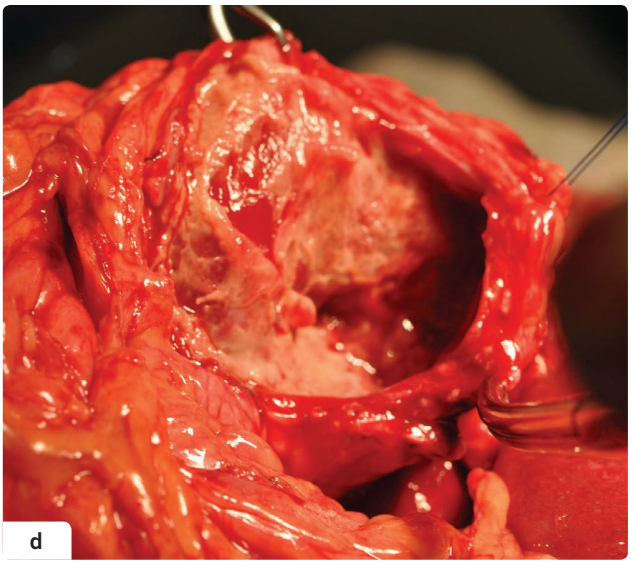
Esophageal, gastric, or jejunal feeding tubes can be placed early in the course of treatment and low-volume frequent feedings can be given through the tubes.

Initially, the most important component of treatment of severe pancreatitis is the provision of adequate parenteral fluids. Marked hypotension in the dog should be treated with rapid volume expansion with lactated or acetated Ringer's at an initial dosage of 20 ml/kg (for cats give 7–10 ml/kg) over the first 15 minutes of treatment and repeated at 15-minute intervals until the blood pressure is normalized. Normal saline (0.9%) is no longer a preferred fluid because of its tendency to cause hyperchloremic metabolic acidosis. Saline will be indicated if the patient is hyponatremic. Close patient monitoring is essential in order to prevent intravascular fluid overload. This can be done crudely by observing breathing quality and listening to lung sounds. After the vital signs are stabilized and urine output is noted as adequate, a maintenance fluid rate of 60–120 ml/kg can be given gradually over the remaining 20–22-hour period. The amount of maintenance fluid depends on the patient's vasomotor stability, the degree of abdominal effusion accumulation ('third spacing'), and the presence of ongoing losses through vomiting and diarrhea. The intravenous maintenance solution can consist of 2.5–5% dextrose in 0.45% saline solution supplemented with potassium chloride (3–5 mmol/kg/day [3–5 mEq/kg/day]) and soluble vitamin B complex. Any acid–base abnormalities should be recognized and treated appropriately. Severely hypoproteinemic animals (serum albumin <20 g/l [2.0 g/dl]) should receive fresh plasma (5–20 ml/kg per day IV). This will temporarily increase the plasma oncotic pressure









Figures 10.40a–d Images of a large pancreatic pseudocyst in a dog showing its ultrasonographic appearance, the cyst contents from a fine needle aspiration procedure, and the cyst at surgery (intact and after resection).

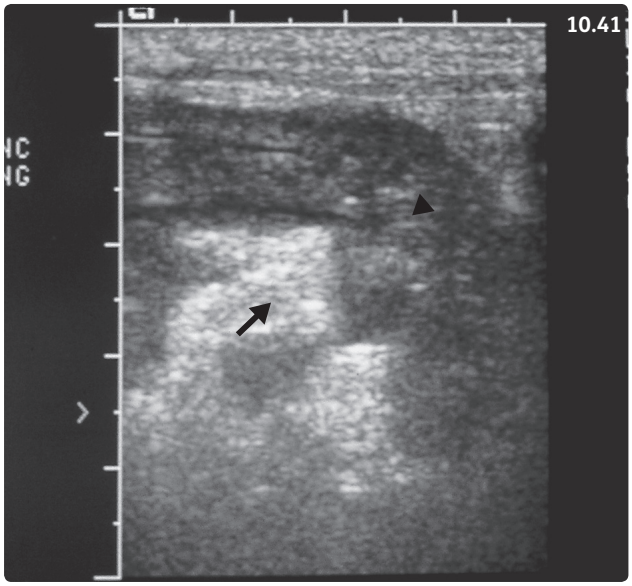
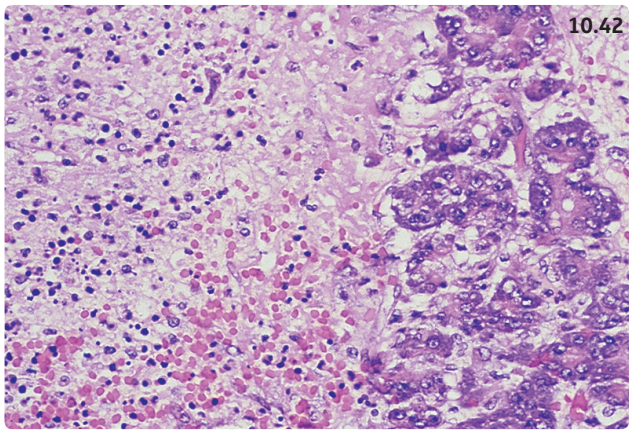
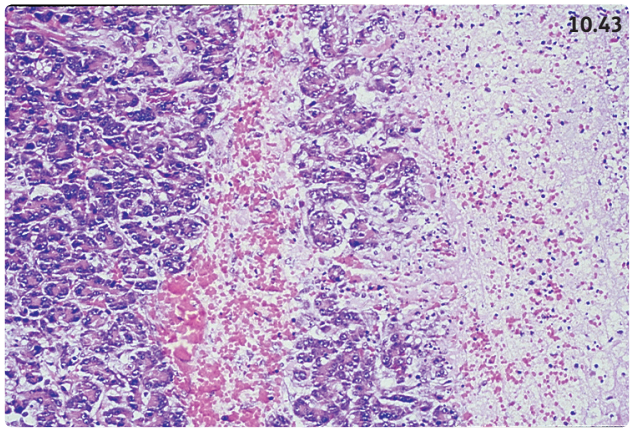


Figure 10.41 Abdominal ultrasound examination of a 7-year-old male English Cocker Spaniel showing lesions typical of acute pancreatitis, including hypoechogenicity of the pancreatic parenchyma (arrowhead) and prominent hyperechogenicity of the inflamed peripancreatic fat, which contained ample calcium soap deposits (arrow). This dog was also in an addisonian crisis. He became anuric and was subsequently euthanized. The postmortem findings included acute necrotizing pancreatitis (similar to that shown in Figure 10.27), bilateral adrenocortical atrophy, and thyroid atrophy. It was thought that the untreated hypothyroidism caused hyperlipidemia, which could have predisposed the dog to acute pancreatitis. The adrenal and thyroid conditions could have been associated with an autoimmune polyhypoendocrinopathy condition.





Figures 10.42, 10.43 Histopathology of the pancreas of the dog in Figure 10.36 showing glandular necrosis, hemorrhage, edema, and inflammatory infiltrate consisting of neutrophils and macrophages. (H&E) (Courtesy P. Ginn)

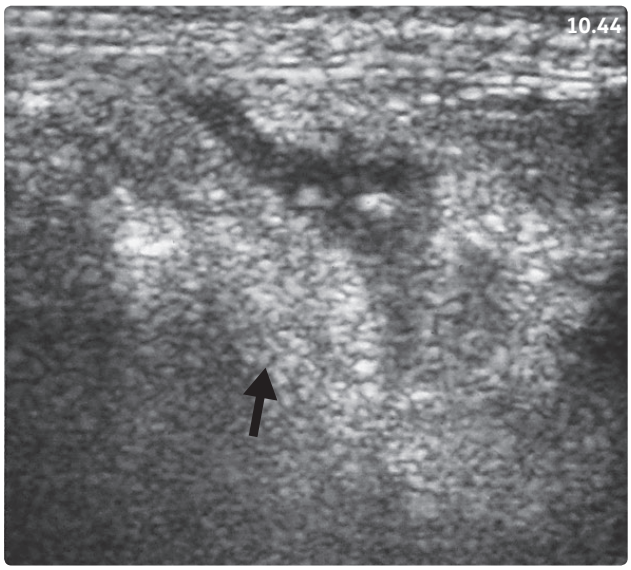


Figure 10.44 Cats can also have typical ultrasonographic signs of acute pancreatitis, as evidenced by the parenchymal hypoechogenicity and a 'rim' of hyperechogenicity (arrow). This 7-year-old male cat was also a ketoacidotic diabetic who gradually improved with 3 weeks of intensive medical treatment alone. He eventually became cured of both diseases.

and help prevent edema formation, pleural effusion, pulmonary edema, and renal failure. Note that the actual volume of plasma needed and the usual prohibitive cost per unit will usually limit the actual amounts administered. Plasma (10 ml/kg IV over a 3–6-hour period) is also a source of pancreatic protease inhibitors, but studies in humans have shown no clinical benefit from this antiprotease activity in transfused plasma. Other colloidal solutions, such as hydroxyethyl starch (20 ml/kg IV over 24 hours) or dextrans (10–20 ml/kg IV over 24 hours), were formerly used as plasma substitutes, but they are no longer recommended because of the potential complications they can cause in the patient (acute kidney injury and bleeding, respectively).

Urine output should be closely monitored after the volume of the patient's plasma space is adequately expanded. Oliguria or anuria should prompt a furosemide-induced diuresis after rehydration is completed, even though it will not reverse acute kidney injury. Potassium supplementation should be discontinued if hyperkalemia develops. The induction of osmotic diuresis should be avoided if the animal's plasma is already hyperosmotic (usually from hyperglycemia). During oliguria the maintenance parenteral fluid volumes should be equal to the volume of urine produced plus any insensible fluid losses (10–15 ml/kg/day). During anuria any unsuccessful forced IV fluid diuresis attempts can cause potentially fatal pulmonary edema.

Drugs used to treat gastrointestinal disorders (e.g. atropine and propantheline) were commonly used in the past to help stop vomiting. However, their adverse parasympatholytic side-effects often exceeded any benefits. The current recommendation is to administer maropitant (1.0 mg/kg SC), which functions as a neurokinin (NK1) receptor antagonist antiemetic. If vomiting persists, metoclopramide can be used without the parasympatholytic effects of the early-generation antiemetics. The recommended dosage is 0.2–0.4 mg/kg SC q6–8h or 1 mg/kg/q24h by continuous IV infusion. Ondansetron is a centrally acting antiemetic that can be given to dogs and cats at a dosage of 0.15 mg/kg IV over a 6–12-hour period and repeated every 6–12 hours.

Famotidine, an H2 blocker, and gastric acid inhibitors, such as the proton pump blockers

Table 10.5 Clinicopathologic abnormalities accompanying acute pancreatitis in the dog and cat.

Abnormality	Proposed mechanism(s)
Leukocytosis	Inflammation, stress, hemoconcentration, secondary infection
Leukopenia	Mobilization to inflamed site, bone marrow suppression from endotoxemia
Hemoconcentration	Dehydration, translocation of plasma into abdominal cavity
Hypoalbuminemia	Intestinal loss, translocation into the abdominal cavity and peripancreatic tissues
Thrombocytopenia	Consumption from thrombus formation
Anemia	Hemorrhagic abdominal effusion, iatrogenic crystalloid fluid infusion
Azotemia	Prerenal from dehydration, renal from hypovolemia or disseminated intravascular coagulation, idiopathic renal failure
Liver enzyme and bilirubin elevations	Focal hepatic necrosis, hepatic lipidosis, cholangitis, cholangiostasis
Hyperglycemia	Elevated 'stress' hormones (growth hormone, glucocorticoids, glucagon, epinephrine), hypoinsulinemia, destruction of islet β-cells
Hypoglycemia	Sepsis with impaired gluconeogenesis
Hypocalcemia	Calcium soap formation is most widely accepted mechanism
Hyperlipidemia	Might pre-exist as separate entity; can occur with pancreatitis, but exact mechanism unknown, possibly related to 'stress' hormone release
Hypernatremia	Dehydration
Hyponatremia	Vomiting, pseudohyponatremia from hyperlipidemia
Hypokalemia	Vomiting, failure to supplement parenteral fluids, osmotic diuresis from hyperglycemia
Hyperamylasemia and hyperlipasemia	Direct venous absorption of enzymes from the inflamed pancreas and absorption via transperitoneal lymphatics, and lymphatic drainage from the pancreas and peripancreatic tissues
Prolonged prothrombin and partial thromboplastin times, hypofibrinogenemia, thrombocytopenia	Disseminated intravascular coagulation

10.45

Glucose	48.6 mmol/l (884 mg/dl)
BUN	35.7 mmol/l (100 mg/dl)
Creatinine	424 µmol/l (4.8 mg/dl)
Cholesterol	7.8 mmol/l (300 mg/dl)
Total bilirubin	61.5 µmol/l (3.6 mg/dl)
Total protein	74 g/l (7.4 g/dl)
Albumin	26 g/l (2.6 g/dl)
AP	492 U/l
Calcium	2.2 mmol/l (8.8 mg/dl)
Phosphorus	1.5 mmol/l (4.7 mg/dl)
AST	110 U/l
ALT	46 U/l
Globulin	48 g/l (4.8 g/dl)
Chloride	103 mmol/l (103 mEq/l)
Sodium	130 mmol/l (130 mEq/l)
Potassium	4.4 mmol/l (4.4 mEq/l)
Total CO_2	12 mmol/l (12 mEq/l)
Amylase	3,000 U/l
Lipase	2.0 U/l
Osmolality	360 mOsmol/kg

Figure 10.45 Example of marked serum biochemical abnormalities taken from the dog in Figure 10.3. These results support the additional clinical diagnoses of hyperosmolar diabetic ketoacidosis and acute kidney injury. The serum amylase and lipase exceeded the upper limits of normal by two-fold, thus supporting the main clinical diagnosis of acute pancreatitis. The lowered serum calcium level is attributed to calcium soap formation. The elevated liver enzymes are likely due to hepatic lipidosis and cholangiostasis, while the hyponatremia could have been due to losses from impaired renal tubular sodium reabsorption, osmotic diuresis, and vomiting. Pseudohyponatremia could have also been present from hyperlipidemia or marked hyperglycemia. The low total CO_2 reflects metabolic acidosis, and the hyperosmolality is due to the marked hyperglycemia.

Table 10.6 Basic principles for treating severe pancreatitis in the dog.

Admit to intensive care and insert an indwelling intravenous catheter
Nothing by mouth if vomiting is profuse
Small amounts of oral nutrition can be given in the absence of severe vomiting
Pain relief
Antibiotics
Parenteral fluid replacement
Insulin (where appropriate)
Nutrition support eventually by total parenteral nutrition or enteral nutrition using jejunostomy tube if oral intake is not possible
Surgical débridement and drainage – if infected or not responsive to medical treatment

omeprazole (0.5–1.0 mg/kg q24h) and pantoprazole (0.7–1.0 mg/kg IV over 15 minutes q24h) have been recommended because of their effective anti-ulcer effects. Although there are theoretical justifications for their use, there are no well-controlled clinical trials that substantiate any proven benefit in the treatment of acute pancreatitis.

Antibiotics are usually reserved for moderately and severely ill patients. Such animals are prone to various complications including septicemia, urinary tract infection (especially when an indwelling urethral catheter is used), pneumonia, and pancreatic abscess formation. Since some of these infections are polymicrobial, broad-spectrum antimicrobial coverage for both aerobic and anaerobic bacteria is recommended. Bacterial infection of the pancreas can be due to the spread of bacteria from the portal lymph nodes, biliary tree, colon, or from other body sites. Ampicillin, quinolones, and first-generation cephalosporin antimicrobials can be used safely. Aminoglycoside antibiotics should be used with caution due to their potential nephrotoxicity in a clinical setting where renal function might already be impaired. The literature currently does not recommend antimicrobial use in the absence of a documented infection.

Glucocorticoids are not routinely used in acute pancreatitis. Although they might be helpful because of their anti-inflammatory activity, they can predispose the patient to infections that can be very detrimental.

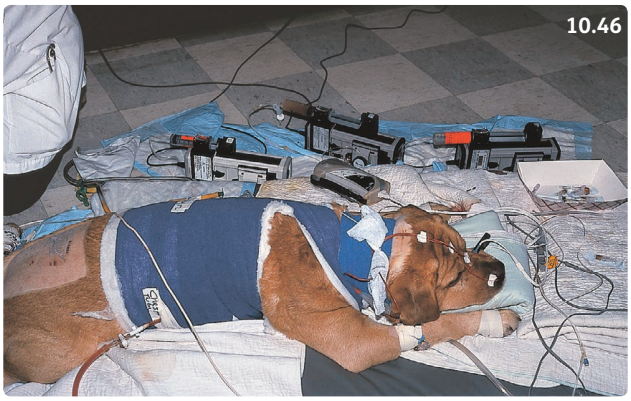
Providing adequate nourishment is essential. Although 5% dextrose solution provides the small amount of calories that might suffice for the first few days of treatment, it falls far short of providing the patient's caloric and protein needs over the 1–2-week period of inappetence caused by the animal's abdominal discomfort and persistent vomiting. Many patients will be able to resume the intake of liquids and then solids after the first 5–7 days following cessation of vomiting. A low-fat diet is recommended during this recovery period and for the long term thereafter. In cats, the introduction of food is also gradual, but without any apparent need for fat restriction.

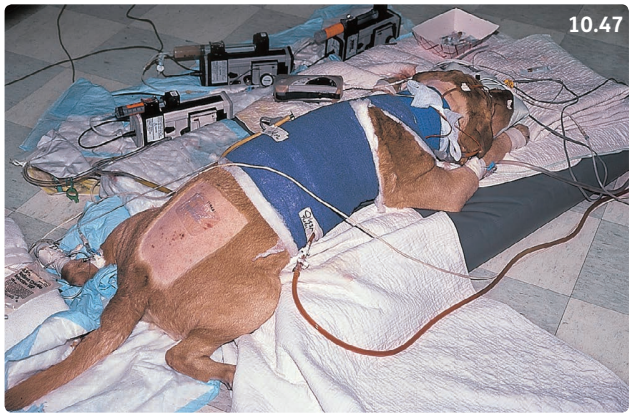
Intravenous parenteral nutrition or enteral tube feeding should be considered if the patient will not voluntarily eat. The former is somewhat controversial because there is evidence that intravenous infusion of amino acid and lipid solutions can stimulate pancreatic secretion in the dog. Problems associated with intravenous hyperalimentation include catheter-associated phlebitis and septicemia and plasma hyperosmolarity. Meticulous preparation is required. There is a need for multiple cannulae, and expense must be taken into consideration. Intravenous hyperalimentation is therefore usually reserved for tertiary medical facilities where the logistics are conducive to this particular treatment modality. Enteral tube feeding is an alternative way of nourishing an animal that has protracted acute pancreatitis. J-tube feeding is the preferred choice for extended nutritional support in postoperative patients because of its ease of maintenance and the advantages it provides to the small bowel mucosa.

Analgesic treatment using opioids should be reserved for animals that have severe and intractable pain. Phenothiazine drugs are contraindicated initially because they might worsen hypotension (see **Table 10.7** for specific drugs and dosages). Epidural analgesia can also be used for pain control (**Figures 10.46, 10.47**).

Table 10.7 Analgesia for the dog and cat with acute pancreatitis.

Species	Drug	Dose
Small dogs and cats	Fentanyl transdermal	25 µg/hour
Dogs 5–10 kg	Fentanyl transdermal	25 µg/hour
Dogs 10–20 kg	Fentanyl transdermal	50 µg/hour
Dogs 20–30 kg	Fentanyl transdermal	75 µg/hour
Dogs >30 kg	Fentanyl transdermal	100 µg/hour
Cats and dogs	Butorphanol	0.1–1.0 mg/kg IM, IV, or SC q1–3h
Cats and dogs	Buprenorphine	0.01–0.02 mg/kg IM, IV, or SC q6–12h





Figures 10.46, 10.47 This 5-year-old male Labrador Retriever was referred to surgery because of severe hemorrhagic pancreatitis. He required critical care, which included epidural analgesia, intensive patient monitoring, and the need for multiple intravenous and intra-arterial cannula sites. The dog died despite all the intensive care provided.

Insulin treatment is indicated when the blood glucose level exceeds 16.7 mmol/l (300 mg/dl). Regular crystalline zinc insulin (initial SC injection of 0.5 U/kg body weight) is preferred because of its short duration of action, especially if the hyperglycemia is transient. If the patient shows a continued need for insulin, it should be managed according to current protocol. Regular insulin should be given by slow constant IV infusion (0.05–0.1 U/kg/hour) if the patient is hypotensive, during which time subcutaneous absorption is impaired and undependable.

Surgical therapy

There are several major indications for surgery in patients with acute pancreatitis: to treat potentially correctable disease such as pancreatic pseudocyst or abscess; to eliminate diseases initiating pancreatic inflammation; to remove necrotic or infected foci during the septic phase of hemorrhagic necrotizing pancreatitis; and to correct complicating problems such as bile duct obstruction

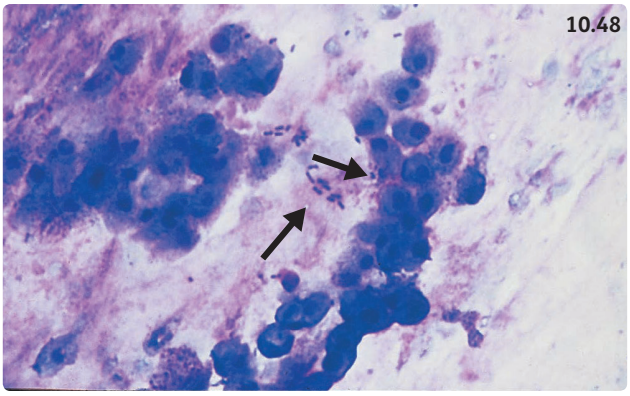


Figure 10.48 One of the indications for surgery in acute pancreatitis patients is the formation of pancreatic phlegmon and the onset of secondary bacterial infection. This cytology sample, from abdominal fluid in a dog with marked pancreatic necrosis with phlegmon and abscess formation, shows toxic neutrophils containing bacteria as well as free bacteria (arrows). Gram-negative bacteria are the most common organisms present on culture if infection occurs. (Courtesy D.J. Meyer)

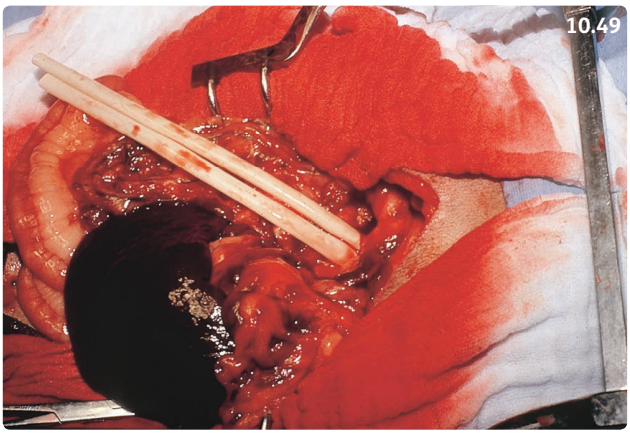


Figure 10.49 Surgery for pancreatitis in a 3-year-old female Schnauzer that was refractory to medical therapy alone. Shown here is peritonitis and an isolated mesenteric abscess containing drains that were surgically implanted and remained in place for 3 days postoperatively. This dog required two separate surgical procedures for débridement and drainage. She required intensive care for 5 weeks and went on to do well.

(**Figures 10.48–10.53**). Laparotomy also allows for the insertion of a J-tube and the commencement of enteral feeding. Although some might prefer earlier surgical intervention, the current recommendation in human medicine calls for delays of as long as 2 weeks in order to allow time for diffuse inflammation to subside and for any pancreatic abscess or pseudocyst to assume the form of a discrete mass.

Laparotomy and peritoneal lavage are recommended for moderately to severely sick animals that fail to

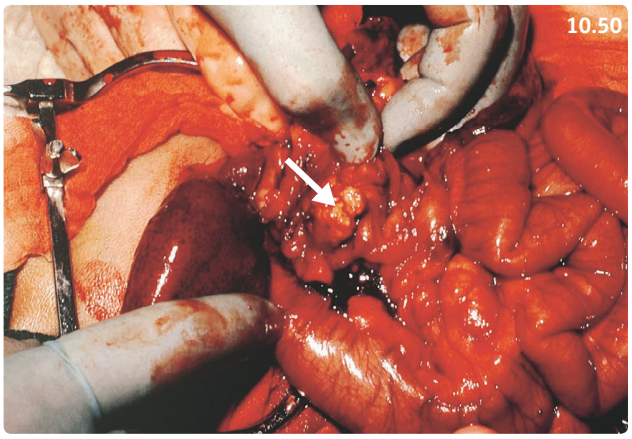


Figure 10.50 Surgery on a 10-year-old Yorkshire Terrier showing caseous debris (arrow) and peritonitis associated with acute hemorrhagic necrotic pancreatitis. Débridement and abdominal drains were used to treat this dog.

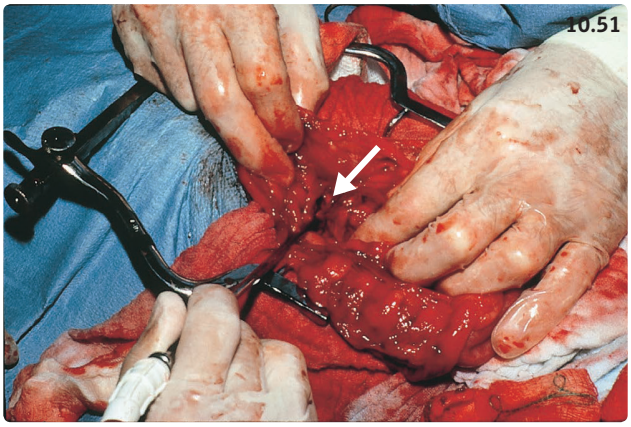


Figure 10.51 This 7-year-old male Wirehaired Fox Terrier had hemorrhagic pancreatitis that could barely be identified at surgery among the inflamed abdominal tissues. Shown is a large abscess cavity (arrow) that was located at the root of the mesentery.

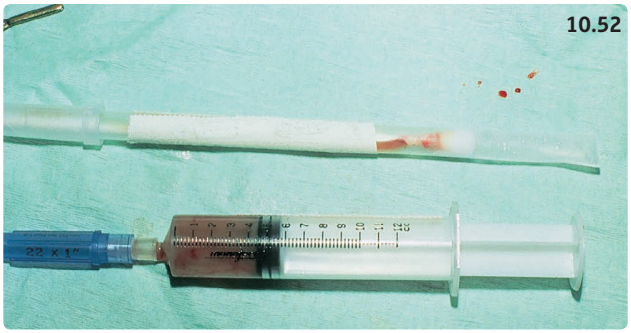


Figure 10.52 The cavity shown in Figure 10.51 contained a remarkable amount of pus from which *E. coli* was isolated.

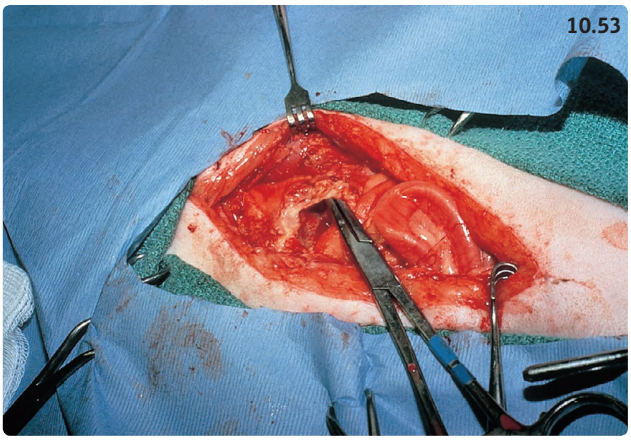


Figure 10.53 Acute pancreatitis in cats can cause similar pathology to that seen in dogs. Shown here is a phlegmon with cavitation that was found during an abdominal ultrasound examination preoperatively. Abdominal drains were inserted after the necrotic debris was removed. This 6-year-old male Siamese cat required two abdominal surgeries before recovery was attainable.

respond to medical treatment after the first 5–7 days or for those in the early stages of severe hemorrhagic necrotic pancreatitis. In many of these cases, the optimal time for surgery is left to the discretion of the clinician. Cases of infected pancreatitis should also be treated surgically as soon as possible, much in the same light as a septic abdomen would be treated. This condition is frequently associated with a guarded to grave prognosis. The diagnosis of infected pancreatitis is made by the cytologic demonstration of bacteria obtained from abdominal fluid or tissue via ultrasound-guided fine needle aspiration or through surgical exploration. Microbial isolation for identification and sensitivity should also be done, ideally prior to antibiotic administration. Lavage-induced hypoproteinemia and serum electrolyte deficiencies should be corrected with plasma and appropriately adjusted electrolyte solutions, respectively.

Complications and long-term management

Table 10.8 outlines the several complications that can occur during the acute or chronic (beyond 2 weeks) phase of pancreatitis. Long-term medical management for the dog includes a low-fat diet that is divided into two or three feedings per day. Diabetes mellitus and exocrine pancreatic insufficiency (EPI) should be treated according to established protocols. The diabetic condition can be either temporary or permanent and is the result of substantial beta-cell destruction (70–90%) as a result of the pancreatitis.

Table 10.8 Complications of acute or chronic pancreatitis.

Complication	Phase of disease
Diabetes mellitus	Acute or chronic
Pancreatic abscess and pseudocyst	Acute
Bowel infarction	Acute
Bowel obstruction	Acute
Bile duct obstruction and/or cholangiostasis	Acute
Renal failure	Acute
Septicemia	Acute
Consumption coagulopathy	Acute
Pulmonary edema (acute respiratory distress syndrome)	Acute
Pleural effusion	Acute
Relapsing pancreatitis	Chronic
Pancreatic exocrine insufficiency	Chronic

Note: 'Acute' refers to first 14 days; 'chronic' extends beyond 14 days.

EXOCRINE PANCREATIC INSUFFICIENCY

Definition/overview

EPI is a malnutrition disorder caused by a deficiency of pancreatic digestive enzymes. It is much more common in dogs than in cats.

Etiology

In the dog, where it is most commonly seen in German Shepherd Dogs and Rough-coated Collies, EPI is usually caused by an atrophy of the zymogen-containing acinar cells. The atrophy was once thought to be idiopathic, but recent studies show that an immune-mediated lymphocytic inflammatory process leads to the atrophy in the aforementioned breeds. The typical age of onset is between 1 and 5 years. EPI can also be caused by recurrent pancreatitis and the associated loss of acinar cells. It rarely occurs with pancreatic adenocarcinoma because the malignancy will often lead to the patient's demise long before it destroys the majority of the exocrine pancreatic cells. EPI occurs rarely in cats (**Figures 10.54–10.56**), where the signs are similar to those in the dog; chronic pancreatitis is thought to be the cause.

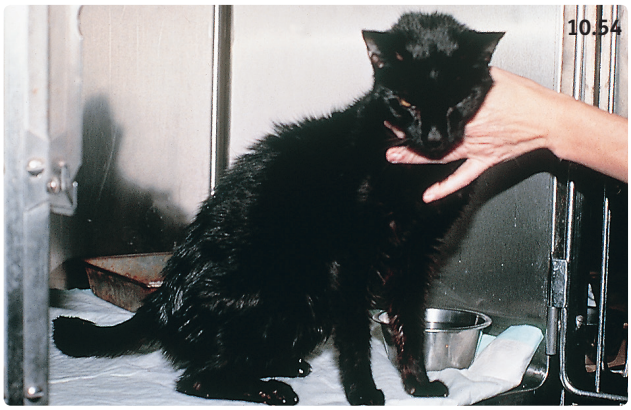


Figure 10.54 Exocrine pancreatic insufficiency is rare in the cat. This emaciated 13-year-old female Domestic Shorthair cat showed typical signs of weight loss and polyphagia in the absence of hyperthyroidism.

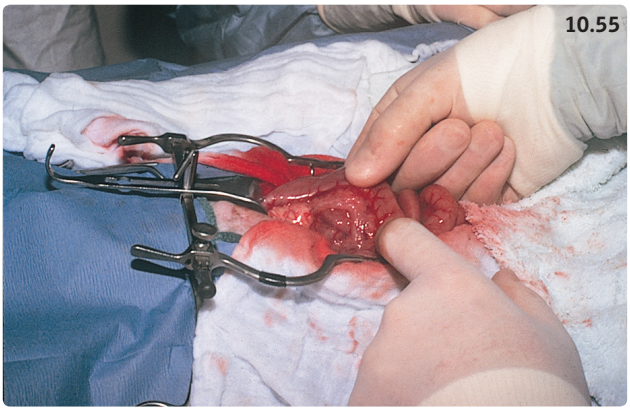


Figure 10.55 The fibrotic and atrophic pancreas of the cat in Figure 10.54 was evident at laparotomy.

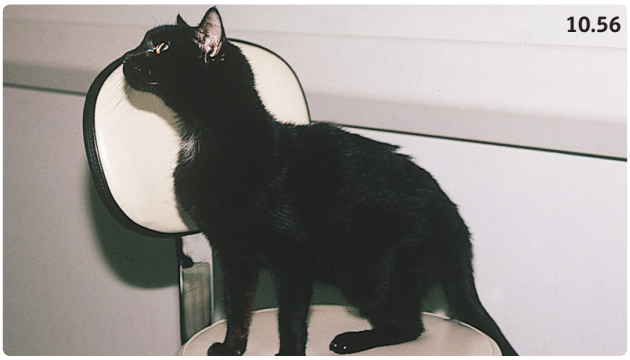


Figure 10.56 The cat in Figure 10.54 after treatment with pancreatic enzyme replacement.

Pathophysiology

A lack of digestive enzymes resulting in poor intraluminal digestion is the main cause of the malabsorption in this syndrome. This is furthered by changes in the small intestinal mucosa including abnormal intestinal enzyme

activity, impaired transport function, villous atrophy, and inflammatory cell infiltration. Bacterial overgrowth is a frequent complication of EPI and may contribute to some of the pathology.

Clinical presentation

The classic signs include polyphagia, weight loss, and hyperdefecation. Pica and coprophagia, flatulence, and borborygmus can also occur. The stool quality ranges from diarrhea to semi-formed, while the color varies from brown to yellow (**Figure 10.57**). Polydipsia and polyuria can be present if diabetes mellitus coexists.

EPI patients are typically thin and have dull dry haircoats reflecting their chronic malnourished condition (**Figures 10.58–10.60**). Examination of the stool and, sometimes, the perineum often shows a greasy texture caused by steatorrhea. The feces can give off a very foul odor. In cats, EPI must be differentiated from hyperthyroidism, and the physical examination should

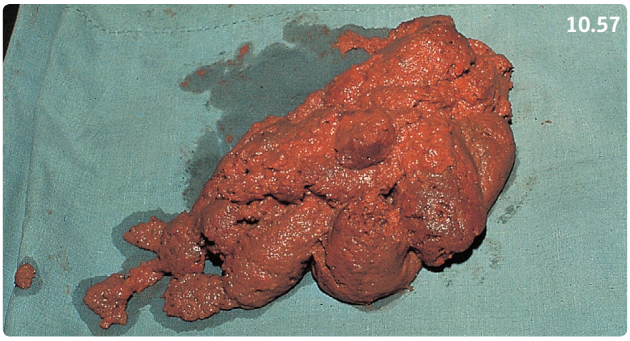
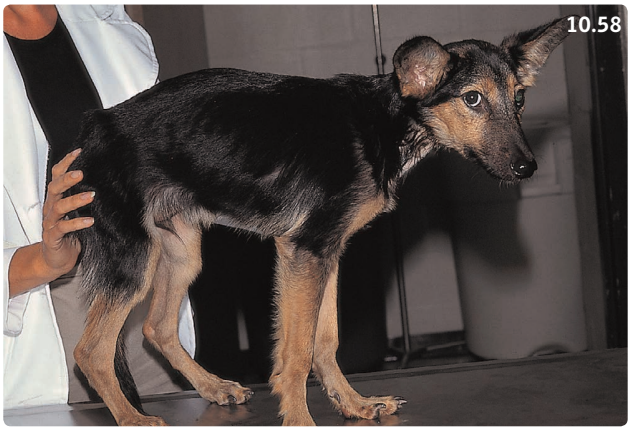
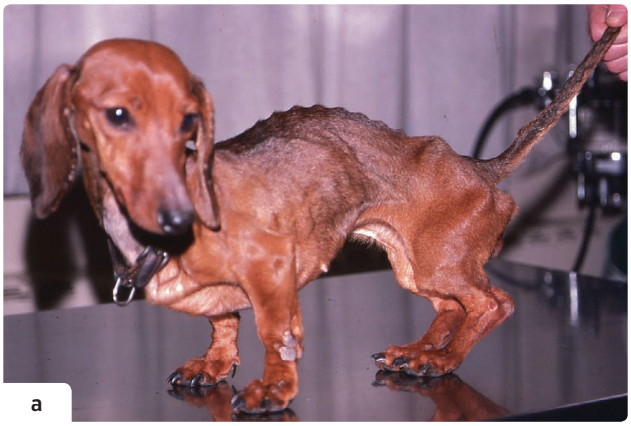


Figure 10.57 Fecal sample from a dog with exocrine pancreatic insufficiency and steatorrhea showing the typical yellow color and greasy texture.



Figures 10.58, 10.59 A 10-month-old male German Shepherd Dog showing its frail body and stunted growth (10.58) and its voracious appetite (10.59).



Figures 10.60a, b This young female Dachshund suffered severe emaciation from exocrine pancreatic insufficiency. The same dog is shown approximately 4 months after pancreatic enzyme replacement treatment.

include a thorough palpation of the neck in search of goiters, which will lend support to this diagnosis.

Differential diagnosis

Small intestinal malabsorption, pancreatic duct obstruction, primary small intestinal disease, parasitism, hyperthyroidism.

Diagnosis

The hemogram will be normal or show a mild normochromic normocytic anemia from the malnutrition. Serum chemistry levels are usually normal; however, hypoproteinemia can occur in some patients as a result of the faulty digestion and impaired assimilation of ingested proteins. Hyperglycemia can occur if most of the pancreatic beta cells have been destroyed by earlier pancreatitis.

Examination of feces for the presence of fat, carbohydrate, and trypsin activity (film digestion) is an empirical test and frequently yields inaccurate results (**Figures 10.61, 10.62**). However, a fecal proteolytic enzyme test has been found to be useful in cats. The serum TLI assay is a sensitive and accurate quantitative test for diagnosing EPI in the dog and cat. A value of <2.5 µg/l is diagnostic. In German Shepherd Dogs and Rough-coated Collies, serum TLI values ranging between 2.5 and 5 µg/l suggests subclinical EPI and partial atrophy.

Management

Once the diagnosis is made, treatment is simple, entailing the provision of a powdered ox- or pig-derived commercial pancreatic enzyme product. The powdered form is preferred over tablets. It should be thoroughly mixed in a moist, nutritionally balanced ration (one teaspoonful per 0.5 kg of food) at each feeding. Providing a balanced vitamin–mineral tablet will also be helpful. Some dogs with coexisting intestinal bacterial overgrowth will require antimicrobial treatment (tetracycline, 15 mg/kg q8h) in order to enhance the effectiveness of treatment. Gradually increased amounts of food should be provided until the patient's normal body weight is reached.

The outlook for the majority of these patients is excellent so long as they are maintained on their required amount of pancreatic enzyme therapy (**Figure 10.63**). In most cases, where regeneration of pancreatic exocrine tissue rarely occurs, treatment will be life-long (**Figures 10.64, 10.65**). Insulin-dependent diabetes mellitus might also require life-long insulin treatment if the EPI is the result of acute pancreatitis.

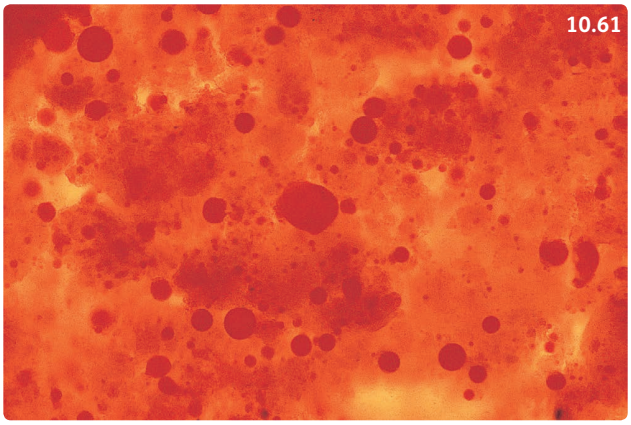


Figure 10.61 A microscopic view of steatorrheic stool stained with Oil Red O showing the large sized red-colored fat globules reflecting undigested fat (×100).

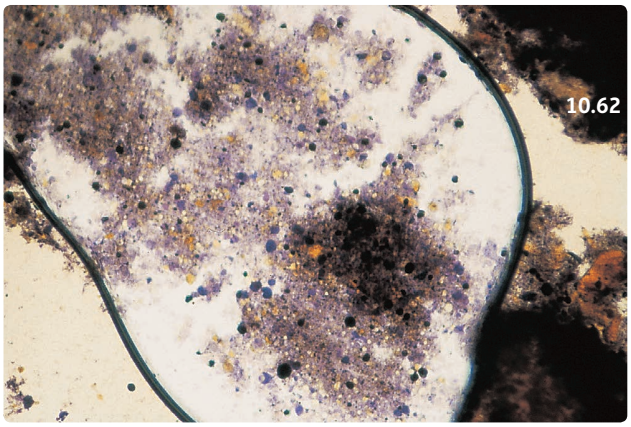


Figure 10.62 Lugol solution will stain starch a dark purple color, as shown on this microscopic view of a stool smear taken from a dog with exocrine pancreatic insufficiency (×100).

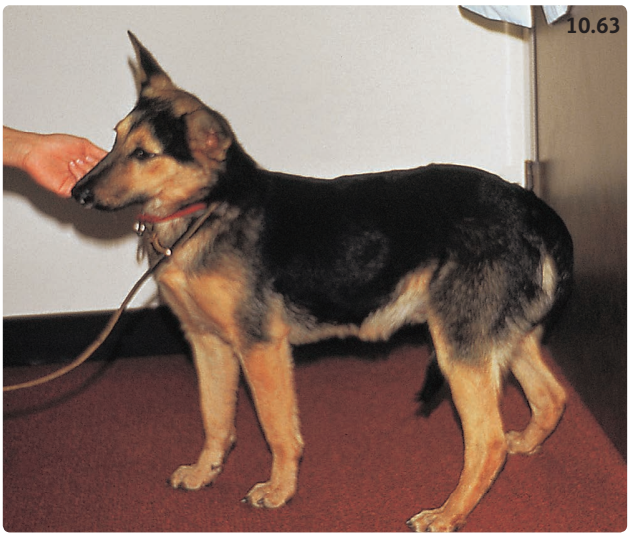
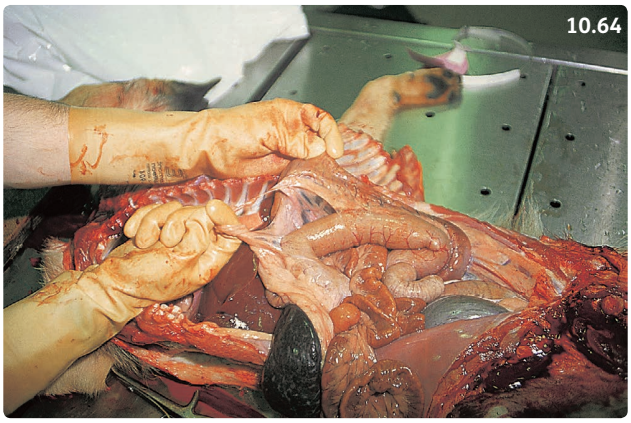
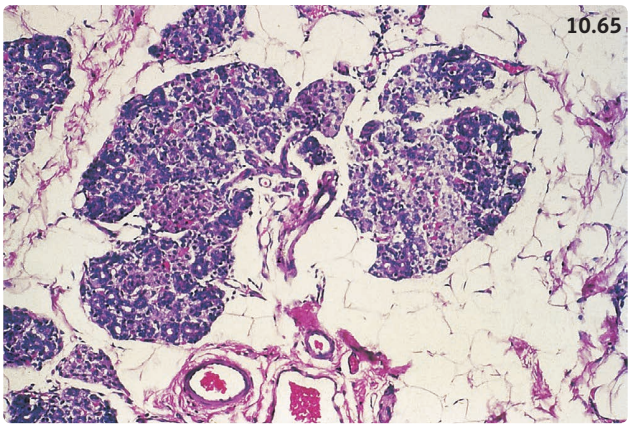


Figure 10.63 The dog in Figures 10.58 and 10.59 after 4 months of treatment with pancreatic enzyme replacement, showing marked improvement in stature.





Figures 10.64, 10.65 (10.64) A postmortem examination of a dog with exocrine pancreatic insufficiency showing marked pancreatic atrophy. (10.65) A microscopic view of the pancreas showing diminished numbers of acinar epithelial cells (H&E. ×40).

PANCREATIC TUMORS

Definition/overview

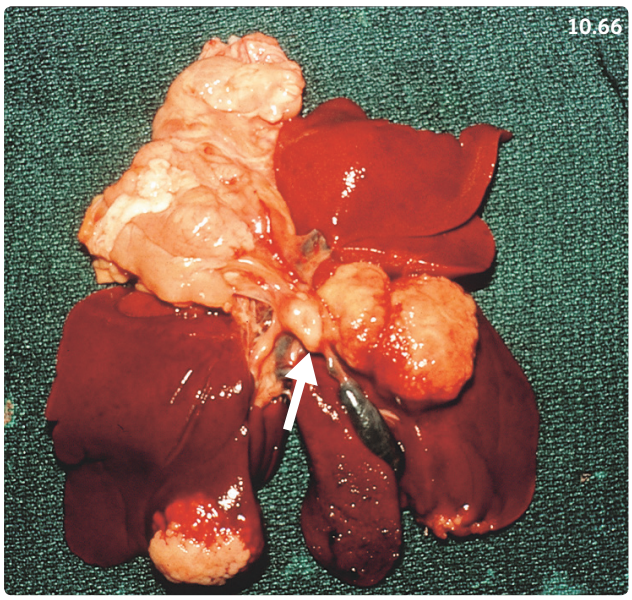
Exocrine cancer of the pancreas is rare in the dog and cat. The majority of the tumors are epithelial and most are adenocarcinomas of ductular or acinar origin. These tumors often have an aggressive behavior, with implantation on the peritoneum and metastasis to the liver common. The liver nodules can be small or quite large (**Figures 10.66–10.71**).

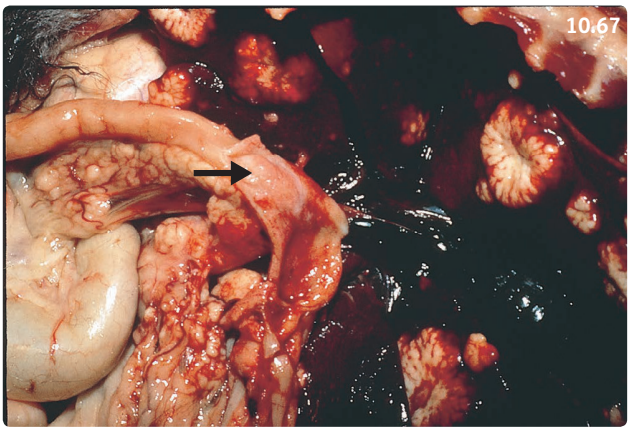
Etiology

There are no known causes of pancreatic adenocarcinoma in either the dog or the cat.

Pathophysiology

The exact stimulus for this particular tumor formation in the dog and cat is unknown.





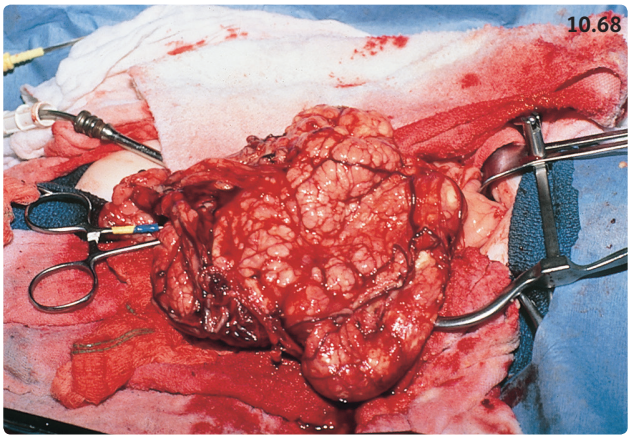
Figures 10.66, 10.67 Pancreatic adenocarcinoma in the cat usually involves the older age group. The usual signs include anorexia and weight loss. Icterus commonly occurs and is associated with obstructed bile flow. Shown here are postmortem findings on two aged cats illustrating rather large metastatic lesions involving the liver. One cat (10.66) had obstructed bile flow due to obstruction along the common bile duct (arrow), while the other cat's icterus was due to carcinoma involving the duodenal papilla (10.67) (arrow).

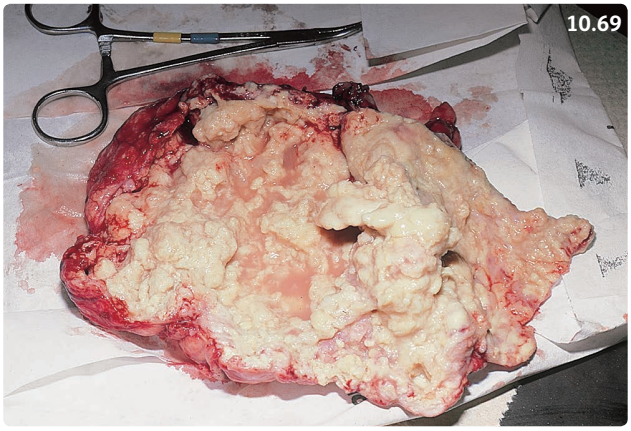
Clinical presentation

The most common clinical signs are weight loss and anorexia. Vomiting is sporadic. Ascites from peritoneal implants and icterus from common bile duct obstruction can also occur (**Figures 10.72, 10.73**).

Differential diagnosis

Chronic pancreatitis, any malignant tumor, chronic gastroenteric disease, chronic liver disease.





Figures 10.68, 10.69 This large pancreatic adenocarcinoma was found in a 12-year-old Siamese cat that was examined for the primary complaints of anorexia, weight loss, lethargy, and gradual abdominal enlargement. Physical examination revealed a large (>5 cm) anterior mid-abdominal mass. The only clinicopathologic abnormalities included a leukocytosis and a mildly elevated total bilirubin. Shown is the tumor at surgery (10.68) and its necrotic interior in the extirpated specimen (10.69).

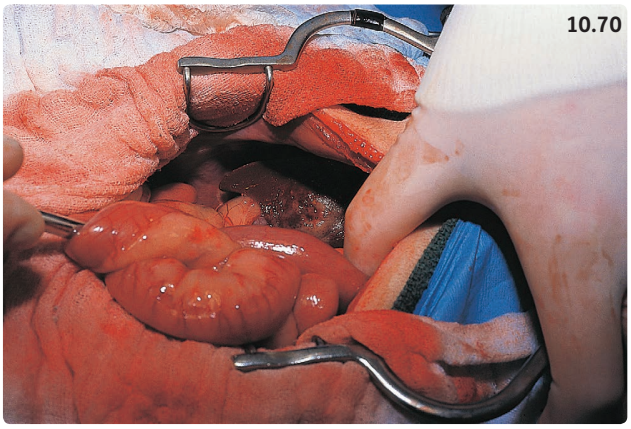


Figure 10.70 Surgical view of pancreatic adenocarcinoma in a 13-year-old male Poodle showing the metastatic lesions to the liver.

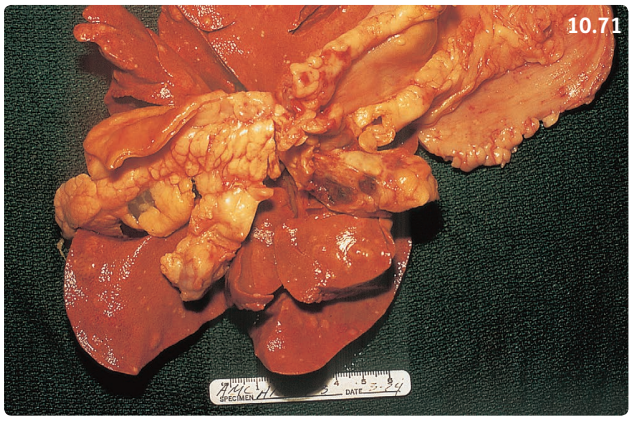
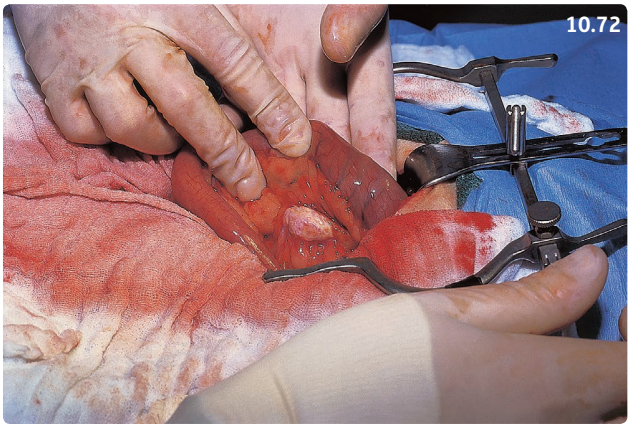
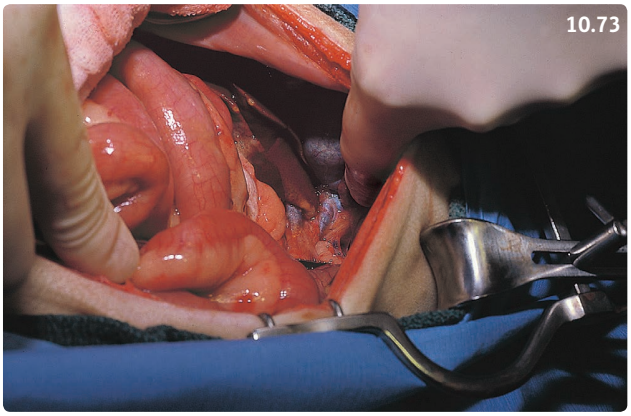


Figure 10.71 Postmortem view of a 15-year-old dog with pancreatic carcinoma with diffuse liver metastasis.





Figures 10.72, 10.73 Surgical views of a pancreatic adenocarcinoma in a 13-year-old male Poodle that has signs of anorexia and icterus. 10.72 shows the primary neoplasm and 10.73 shows metastatic lesions obstructing the bile duct. The pertinent serum chemistry abnormalities were: cholesterol 9.64 mmol/l (371 mg/dl), total bilirubin 117.9 mmol/l (6.9 mg/dl), ALP 28,240 U/l, AST 271 U/l, ALT 380 U/l. The serum amylase and lipase concentrations were normal at 1,332 and 0.6 U/l, respectively, as was the CBC.

Diagnosis

There are no specific diagnostic tests for pancreatic cancer. The hemogram and serum chemistry panel might reflect the effect of the disease, as seen with the cholangiostasis associated with metastasis involving the hepatobiliary tree. Pancreatic lipase assays will give varying results. One of the author's canine patients had markedly elevated levels of cPLI associated with lymphoma infiltrating the pancreas. Abdominal ultrasound can demonstrate abnormal pancreatic parenchyma and metastatic lesions involving the liver. The definitive diagnosis is obtained through a diagnostic laparotomy and histopathology on biopsied tissue specimens.

Management

Optimal treatment would be complete surgical extirpation of the tumor. However, a diagnosis is hardly ever made early enough before the malignancy has had a chance to spread. The prognosis is always grave.

RECOMMENDED FURTHER READING

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