

Compendium

Septic Peritonitis: Etiology, Pathophysiology, and Diagnosis

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Abstract: *Septic peritonitis is an inflammatory condition of the peritoneum that occurs secondary to microbial contamination. This clinically important condition has a wide variety of clinical courses as well as high morbidity and mortality due to secondary multiorgan dysfunction. This article reviews the etiology and pathophysiology of this condition and its diagnosis in small animals; a companion article addresses treatment and prognosis.*

Septic peritonitis is an inflammatory condition of the peritoneum originating from a combination of bacterial and chemical contaminants within the abdomen.¹ It is the most common form of peritonitis in dogs.¹ Septic peritonitis can have a wide variety of clinical courses and outcomes depending on the signalment of the patient, the source and degree of contamination, and the body's response to the contamination. This clinically important condition is associated with high morbidity, and mortality ranges from 20% to 68%.²⁻⁶ Successful treatment requires a basic understanding of pathophysiology, knowledge of useful diagnostic techniques, early diagnosis, appropriate medical therapy, and careful surgical planning.

Anatomy

The peritoneum is a serous membrane composed of a single layer of squamous cells of mesothelial origin. It has two components: a parietal portion that covers the abdominal wall, and a visceral portion that covers the abdominal organs. The surface area of the peritoneum is approximately 150% of the total skin surface area.¹

The peritoneal space may actively absorb or passively accumulate fluid across its surface depending on the balance between hydrostatic pressure and oncotic pressure in the abdomen and vasculature and the reflection coefficient ("leakiness") of the vasculature. The normal balance favors production of a small amount of free abdominal fluid (approximately 80 mL/kg/d). Most of this fluid follows a cranial path along the ventral body wall and drains out of the abdomen via the lymphatic system in <15 minutes.¹ Absorption of fluid from the peritoneal cavity is through terminal lymphatics called *lacunae*, which are located in the diaphragm.⁷ Lacunae communicate with the peritoneal cavity via the diaphragmatic stomata, which are channels formed by juxtaposed processes of mesothelial and lacunar endothelial cells.^{7,8} The absorption rate of

peritoneal effusion is determined by the intraperitoneal pressure and the volume and viscosity of effusion.⁷

The greater and lesser omenta contain aggregates of neutrophils, macrophages, and lymphocytes. These inflammatory cells are important components of the peritoneal defense mechanisms that isolate sources of infection and have angiogenic activity.

Classification and Etiology

Septic peritonitis in dogs and cats can be classified in a number of ways, but the two main schemes are (1) localized or diffuse and (2) primary, secondary, or tertiary. Localized septic peritonitis occurs when a small amount of contamination is well contained. The contamination usually originates from an intraabdominal organ secondary to surgery or an underlying disease process, such as gastrointestinal (GI) perforation due to a foreign body.

Key Points

- Septic peritonitis most commonly occurs secondary to a primary cause such as the loss of gastrointestinal integrity.
- Enteric organisms are the most common bacterial species isolated in patients with secondary septic peritonitis. In patients with primary septic peritonitis, gram-positive species are the most common pathogens.
- Abdominal ultrasonography is more accurate than radiography to detect the presence of free abdominal fluid.
- The presence of intracellular bacteria, organic debris, and/or toxic neutrophils on cytologic examination of the peritoneal fluid warrants surgical exploration.
- A peripheral blood glucose concentration >20 mg/dL higher than the abdominal fluid glucose concentration is suggestive of septic peritonitis.

Box 1. Causes of Secondary Septic Peritonitis in Dogs and Cats^{2,3,12,14}

- Leakage of gastrointestinal contents
 - Perforating foreign body
 - Perforating ulcers
 - Iatrogenic (e.g., dehiscence of intestinal surgical wound, perforation, feeding tube leakage)
 - Gastric rupture in gastric dilatation-volvulus
 - Ischemic intestinal injury
- Blunt abdominal trauma, penetrating abdominal wounds, bite wounds
- Urogenital
 - Ruptured pyometra
 - Ruptured prostatic abscess
 - Ruptured urinary tract with urinary tract infection
- Pancreatitis and pancreatic abscess
- Liver abscess or hepatitis, ruptured infected gallbladder
- Splenic abscess or splenitis, splenic torsion
- Mesenteric lymph node abscess
- Umbilical abscess
- Iatrogenic
 - Surgical peritoneal contamination
 - Peritoneal dialysis

Diffuse peritonitis arises from either a larger amount of contamination or a failure to control localized septic peritonitis.

Primary septic peritonitis is defined as a spontaneous infection of the peritoneal cavity with no identifiable intraperitoneal source of infection detected during surgery or necropsy. This type of peritonitis is more common in cats, with 14% of cats with septic peritonitis having primary septic peritonitis in one study.^{2,9,10} Primary septic peritonitis is usually monomicrobial, whereas secondary septic peritonitis is often polymicrobial.³ In one study,³ bacteria cultured from patients with primary peritonitis were gram positive in 80% of dogs and in 60% of cats. It is postulated that primary septic peritonitis may result from hematogenous or lymphogenous bacterial spread, transmural bacterial migration from the GI tract, or bacterial spread from the oviducts.¹⁰

Secondary septic peritonitis is a consequence of an underlying primary disease process and is the most common cause of septic peritonitis in dogs and cats.^{2,3} There are many possible causes of secondary septic peritonitis in animals (**BOX 1**); the most common are loss of integrity of the GI tract (53% to 75% of cases), foreign-body penetration, perforating ulcers (**FIGURE 1**), and surgical wound dehiscence.^{2,4-6,9} GI perforation has been reported with the use of various antiinflammatory drugs, and dogs can present as early as 48 hours after the initiation of treatment with one of these drugs.¹¹ The reported dehiscence rates following enterotomy range from 3% to 12%.¹² This rate may be greater for intestinal

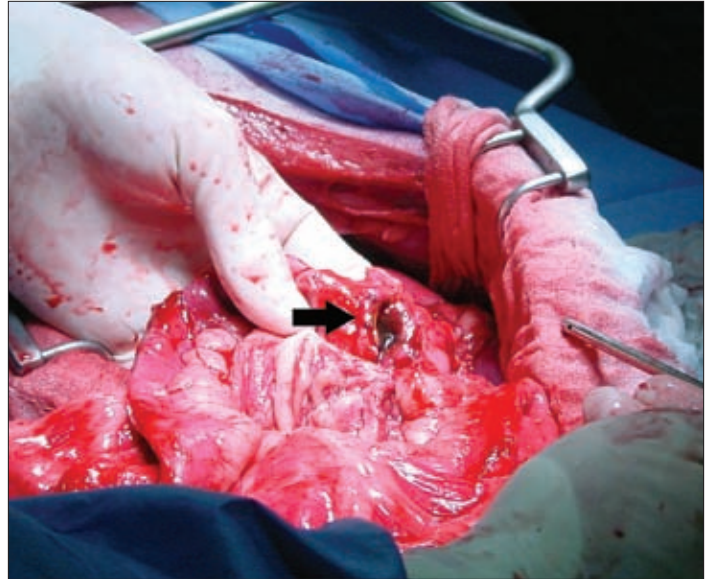


Figure 1. Septic peritonitis secondary to jejunal rupture from a perforating ulcer (arrow).

anastomosis, with 14% of dogs having leakage in a retrospective study.¹³ (None of the 25 cats in this study had leakage.) Dehiscence after intestinal surgery is reported to occur most commonly between 3 and 9 days after the surgery.^{12,13} Septic peritonitis can also occur after spillage of GI contents during surgery, perforation of the abdomen (e.g., foreign body, drainage device, traumatic perforation, bite wound), rupture of the urinary or reproductive tract, or rupture of an infected parenchymal organ (i.e., liver, pancreas, prostate, kidney). Septic peritonitis is associated with uroabdomen in dogs and cats only if the urine is already infected at the time of leakage.¹ The most common bacterial species isolated from patients with secondary septic peritonitis are enteric organisms such as *Escherichia coli*, *Bacteroides* spp, *Clostridium* spp, *Klebsiella* spp, and *Enterococcus* spp.^{2-4,9} *E. coli* may be associated with early mortality because of high circulating levels of endotoxins. It is also the most common bacterial isolate in patients with septic bile peritonitis.¹⁴

Tertiary septic peritonitis is persistent or recurrent peritonitis after appropriate treatment of primary or secondary peritonitis.^{10,15}

Pathophysiology

The clinical course depends on the cause and severity of the septic peritonitis. Localized, mild, or early cases of septic peritonitis are commonly associated with local manifestations. More severe cases of peritonitis are associated with local and systemic manifestations. Local manifestations involve the peritoneum, immune system, and digestive system, whereas systemic manifestations include dysfunction of the cardiovascular, urinary, respiratory, and endocrine systems.

Locally, septic peritonitis is associated with the release of vasoactive substances such as histamine, serotonin, cellular proteases, and microbial endotoxins, ultimately leading to increased capillary permeability and vasodilation. The presence of microorganisms and their endotoxins causes the greatest damage to the peritoneum.



Table 1. Findings Suggestive of SIRS in Small Animals^{6,a}

Clinical Parameter	Canine	Feline
Heart rate	>140 bpm	>240 or <130 bpm
Respiratory rate	>30 breaths/min	>40 breaths/min
Temperature	>102.5°F or <100°F (>39.2°C or <37.8°C)	>104.5°F or <100°F (>40.3°C or <37.8°C)
White blood cell count	>19,000/μL, <6000/μL, or >10% band	>18,000/μL, <5000/μL, or >10% band

SIRS = systemic inflammatory response syndrome

^aTwo or more of these criteria must be met to diagnose SIRS.

The resulting inflammation results in fibrin deposition around the site of injury and within the peritoneum,¹⁰ as well as the loss of isotonic fluid into the abdominal cavity (third spacing) with subsequent hemoconcentration.¹⁰ The abdominal fluid produced is initially clear (transudate), but within hours, it becomes turbid secondary to the influx of protein, macrophages, and neutrophils.^{1,10} Increased peritoneal fluid volume has a negative effect in experimental peritonitis, slowing bacterial clearance and increasing bacterial proliferation.¹ The omentum has the potential to migrate to the injury site to isolate the source of the contamination, as well as to increase the number of inflammatory cells, the absorption of bacteria and debris, and the tissue oxygen content.^{1,8}

Intestinal ileus can occur secondary to inflammation within the peritoneal cavity. It may lead to poor perfusion of the GI tract that could result in ischemia and bacterial translocation; however, ileus is also a protective mechanism, preventing bacterial spread throughout the abdomen.¹

A number of intraperitoneal substances (e.g., gastric mucin, bile salts, hemoglobin) are known adjuvants in peritonitis. These substances worsen the local or systemic inflammatory response.¹ Humoral opsonins, antibodies, and complement are activated with increased severity of the inflammatory response.^{8,10} Many cytokines are released, such as tumor necrosis factor, interleukins 1 and 6, prostaglandin E₂, and platelet aggregation factor.¹⁰ These lead to reduced cardiac output, arterial dilation, and reduced venous return, exacerbating the systemic hypotension resulting from endotoxin release and from the fluid shift into the abdominal cavity. Decreased renal perfusion reduces glomerular filtration rate, potentially leading to the accumulation of nitrogenous toxins, metabolic toxins, potassium, and hydrogen ions.¹⁶ Decreased oxygen delivery brings on anaerobic metabolism and lactic acid production. Reduced renal blood flow also limits the metabolic pathways for eliminating excess hydrogen ions.¹⁶ The compensatory respiratory pathway for hydrogen elimination may also be limited by visceral pain and guarding of the abdomen. The resulting acidosis is another significant problem.

Electrolyte changes such as hyperkalemia and hyponatremia are common.¹⁶ Hyperkalemia occurs secondary to cellular death, sodium-potassium ATP pump failure, metabolic/respiratory acidosis, and a decreased glomerular filtration rate. Hyperkalemia

Table 2. Diagnostic Findings Suggestive of Septic Peritonitis That Warrant Immediate Stabilization and Surgical Exploration^{23–25,a,b}

Diagnostic Tool	Diagnostic Findings
Abdominal radiography	<ul style="list-style-type: none"> • Pneumoperitoneum • Loss of serosal detail with underlying etiology
Abdominal ultrasonography	<ul style="list-style-type: none"> • Pneumoperitoneum • Underlying etiology
Abdominal effusion cytology	<ul style="list-style-type: none"> • Toxic and/or degenerate neutrophils with foreign debris • Intracellular bacteria
Biochemical testing	<ul style="list-style-type: none"> • Peripheral blood to abdomen fluid glucose difference >20 mg/dL • Abdominal fluid to blood difference in lactate value >2.0 mmol/L • A fluid lactate concentration >2.5 mmol/L in dogs

^aBonczynski JJ, Ludwig LL, Barton LJ, et al. Comparison of peritoneal fluid and peripheral blood pH, bicarbonate, glucose, and lactate concentration as a diagnostic tool for septic peritonitis in dogs and cats. *Vet Surg* 2003;32(2):161-166.

^bLevin GM, Bonczynski JJ, Ludwig LL, et al. Lactate as a diagnostic test for septic peritoneal effusions in dogs and cats. *J Am Anim Hosp Assoc* 2004;40(5):364-371.

depresses muscle contraction (including the myocardium and diaphragm) and limits neuroelectrical impulse conduction. Sodium is usually depleted in patients with diffuse septic peritonitis due to dilution in the circulating blood, fluid shift into the abdomen, and cellular sequestration resulting from sodium-potassium ATP pump failure. Hyponatremia eventually results in a failure to maintain intravascular volume and neuroelectrical impulses.

The presence of bacteria and their endotoxins and of inflammatory cells and their cytokines leads to endothelial damage and tissue factor expression that result in generalized activation of the coagulation cascade, causing thrombosis and fibrinolysis. This can initiate disseminated intravascular coagulation (DIC) in association with the loss of the antithrombin III protein into the abdomen.^{17,18} Microvascular and large-vessel thromboses lead to tissue hypoxia and, often, organ damage involving the myocardium, lung parenchyma, and GI tract.^{17,18} DIC may therefore compound the clinical picture of septic peritonitis.

The physiologic changes associated with septic peritonitis can eventually result in central arterial dilation, increased capillary permeability, decreased cardiac function, and multiple organ failure—the hallmarks of systemic inflammatory response syndrome (SIRS) and septic shock.⁸ Although the criteria for SIRS (**TABLE 1**) are neither sensitive nor specific for the diagnosis of septic peritonitis, septic peritonitis should be included in the differential diagnosis of animals presenting with SIRS that have historical and clinical findings supportive of abdominal disease. If the underlying problem continues, multiple organ dysfunction syndrome (MODS) may occur. Peritonitis is the leading cause of MODS in humans.¹ If this process is not controlled, the clinical outcome is usually poor.

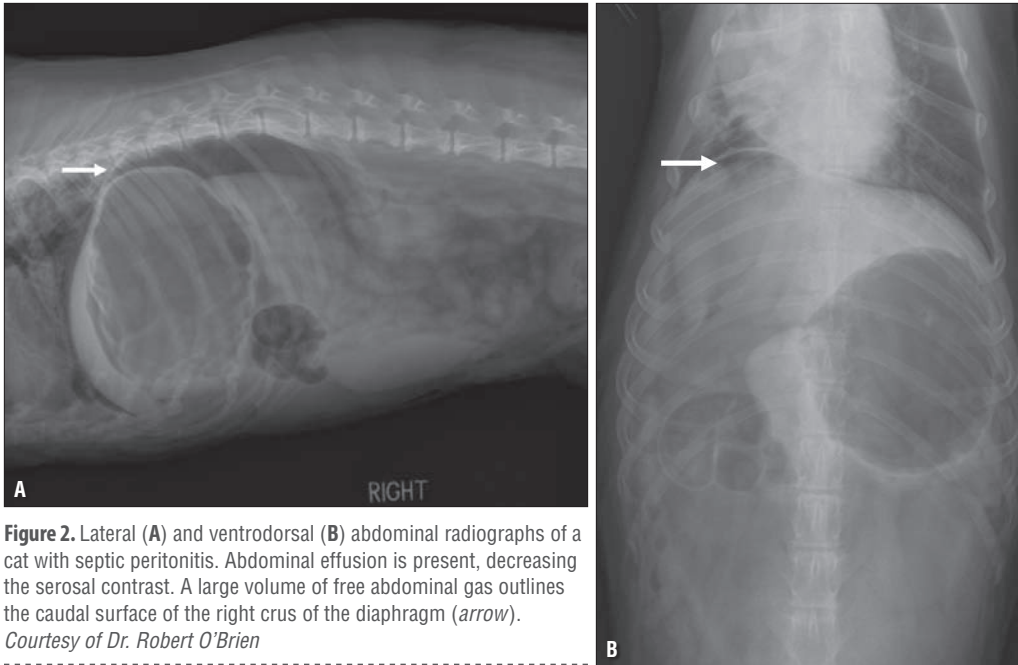


Figure 2. Lateral (A) and ventrodorsal (B) abdominal radiographs of a cat with septic peritonitis. Abdominal effusion is present, decreasing the serosal contrast. A large volume of free abdominal gas outlines the caudal surface of the right crus of the diaphragm (arrow).
Courtesy of Dr. Robert O'Brien

Diagnosis

Diagnostic findings that warrant immediate surgical exploration are listed in **TABLE 2**.

Initial Examination

Clinical signs in animals with septic peritonitis vary depending on the etiology, duration, and severity of the disease. In dogs, early stages of shock due to septic peritonitis are often characterized by vasomotor dysfunction, cytokine-induced peripheral vasodilation, tachycardia, hyperemic (brick red) mucous membranes with a rapid (<1 second) capillary refill time (CRT), bounding pulses, and hyperthermia. As shock progresses, abnormal physical findings increasingly result from decreased contractility and cardiac output.^{1,4,6,9} Specifically, these signs include pale mucous membranes with a prolonged CRT (>2 second), weak to absent peripheral pulses, hypothermia, tachypnea, tachycardia, dehydration, mental depression, and a serum lactate level >2.5 mmol/L; however, patients with septic shock do not regain normal vasomotor tone and therefore maintain inappropriately pink or red mucous membranes secondary to ongoing vasodilation. Increased heart rate is one of the earliest indicators that a dog may be hypoperfused, and its importance in detecting and treating shock should not be overlooked. Dogs with sepsis are more likely to have a higher temperature and heart rate than nonseptic dogs.¹⁹

In contrast to dogs, cats present with relative bradycardia (<140 beats/min) in 16% to 66% of cases.^{2,20} Patients may demonstrate abdominal pain (tucked-up abdomen or “prayer” position), but this feature seems to be less common in cats than in dogs (62% of cats in one study).² However, it should be noted that abdominal pain in patients with severe sepsis is not specific for abdominal disease. In fact, in one study on severe sepsis in cats,²⁰ only 50% of the patients with abdominal pain had abdominal disease.

Peritoneal effusion is a consistent finding but may be difficult to detect on physical examination when only a small amount of fluid is present. With increasing fluid volume, free peritoneal fluid may be detected by ballottement. At least 40 mL/kg of peritoneal fluid must be present to detect a fluid wave.²¹

The patient should be examined for any signs of external wounds (e.g., gunshot, bite, blunt trauma). A characteristic ring of subcutaneous hemorrhage around the umbilicus (Cullen sign) may appear in some patients due to direct extension of the infection to the subcutis if the mesodermal lining is incomplete at the level of the umbilicus.¹ In animals with postoperative peritonitis, sero-

sanguineous to purulent fluid may drip from the incision.

A minimum database should include a complete blood cell count, serum biochemical profile, blood gas analysis, electrolyte profile, serum lactate measurement, and urinalysis. A coagulation panel is recommended for early identification of patients with hemostatic changes and should include prothrombin time, partial thromboplastin time, and D-dimer levels. In one study, dogs with sepsis were more likely to have higher white blood cell (WBC) counts and band neutrophil counts than nonseptic dogs.¹⁹ In patients with suspected septic peritonitis, a peritoneal fluid analysis as described below should be part of the database after the initial examination.

Diagnostic Imaging

Abdominal radiography may demonstrate a decrease in the serosal contrast. This lack of serosal detail stems from fluid and inflammation within the peritoneal cavity. There must be at least 8.8 mL/kg of abdominal fluid for radiography to be consistently diagnostic for peritoneal effusion.²² A loss of serosal detail can also be seen in young dogs or in dogs with little abdominal fat in the absence of effusion. The etiology of the peritonitis may be evident from the radiographic study (e.g., mass, foreign body). Free gas, when present, is typically noted between the liver and diaphragm (**FIGURE 2A** and **FIGURE 2B**). In the absence of recent surgery (within 30 days), free gas may be present due to gas-forming bacteria, penetrating wounds, or perforated hollow viscera.²³ Signs of ileus may be present. Except for pneumoperitoneum, which warrants early surgical exploration, radiography is not very sensitive for the diagnosis of peritonitis.^{23,24}

Abdominal ultrasonography is useful in diagnosing peritonitis. It is more sensitive than radiography for detecting free peritoneal fluid.²² Ultrasonographic guidance allows aspiration of fluid even

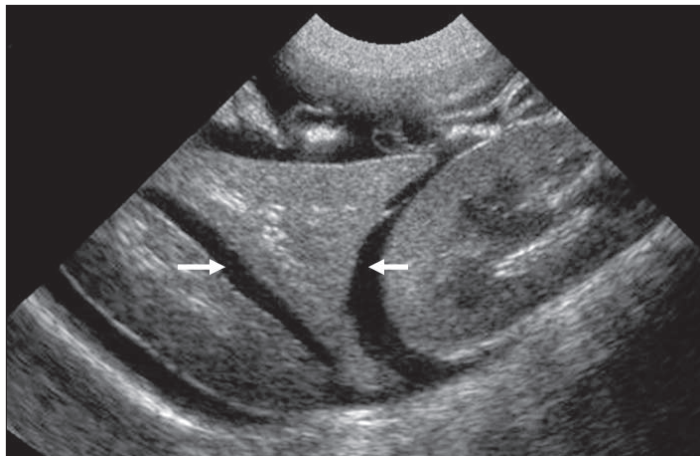


Figure 3. Abdominal sonogram showing free fluid (arrows) present around the liver lobes and the right kidney.

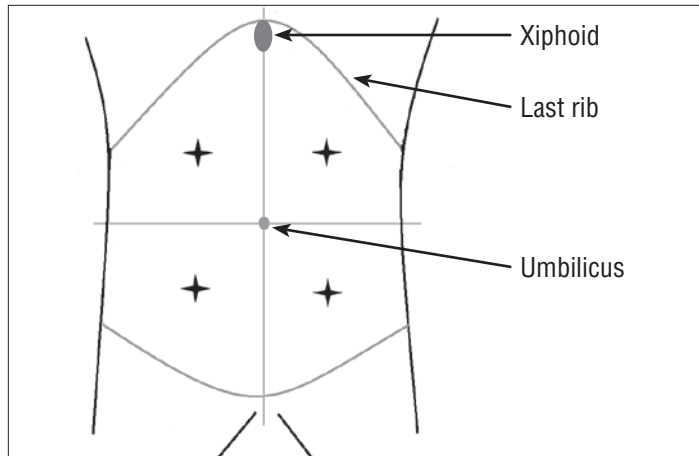


Figure 4. Diagram showing the sites of needle insertion (+) for standard four-quadrant paracentesis with the patient in dorsal recumbency.

from small pockets, facilitating early diagnosis (**FIGURE 3**). Detection of free abdominal fluid is more likely in the gravity-dependent lateral field or around the bladder and spleen if the patient is in dorsal recumbency.²² Ultrasonography may also reveal distended loops of bowel, free gas within the abdomen, and an underlying etiology such as a mass associated with the GI tract or a diseased parenchymal organ.

Peritoneal Fluid Analysis

Abdominocentesis is the single most useful diagnostic test for peritonitis. It can usually be performed on conscious animals. Aseptic technique is essential to prevent inoculation of bacteria into a noninfected abdomen. Fluid may be obtained with either a 20-gauge hypodermic needle or a butterfly catheter. A single tap can be performed on the ventral midline just caudal to the umbilicus. Results of a single abdominal paracentesis are positive for septic peritonitis in only 20% of animals with a peritoneal effusion volume of 3 mL/kg, but in 80% of animals with an effusion volume of 10 mL/kg.¹ A four-quadrant needle paracentesis improves the diagnostic yield. For this procedure, the abdomen is divided into four quadrants (cranial left and right, caudal left and right), and paracentesis is performed in each area (**FIGURE 4**).

If no fluid is obtained from either paracentesis method but peritonitis is suspected, a diagnostic peritoneal lavage can be performed. An over-the-needle catheter (usually 2.5 inch, 16 or 18 gauge) is inserted into the abdomen caudal and to the left of the umbilicus with the patient in lateral recumbency. Over-the-wire (Seldinger) catheters are available for veterinary patients and can also be used to perform diagnostic peritoneal lavage. Warm (98.6°F [37°C]) sterile saline solution is infused into the abdominal cavity at 20 mL/kg via an intravenous (IV) administration set. The animal is walked or carefully rolled from side to side to distribute and mix the fluid, and a sample is recovered for cytologic analysis. No attempt is made to remove all of the fluid. Potential complications include bladder or internal organ catheterization or rupture, so care must be taken to ensure the catheter is

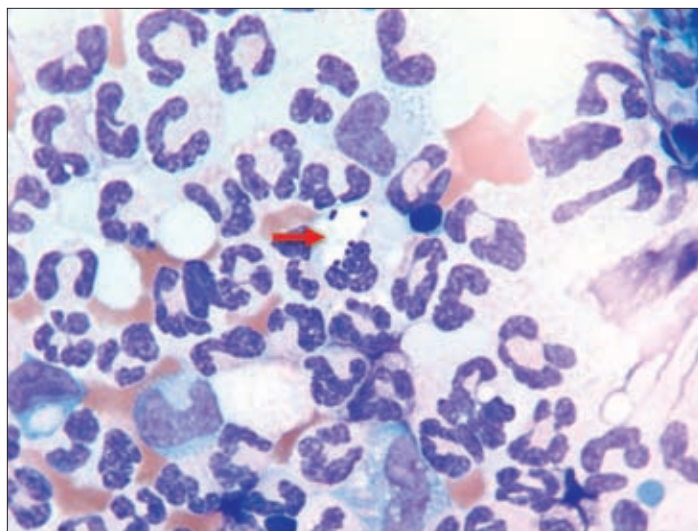


Figure 5. Photomicrograph of abdominal effusion sample from a dog with septic peritonitis. Intracellular bacteria are visible (arrow). Courtesy of Dr. Anne Barger

inserted into the peritoneal space and not within an organ. The urinary bladder can be emptied beforehand by voiding or urethral catheterization to prevent percutaneous insertion of the catheter into this organ.

The effusion sample is then analyzed and evaluated cytologically. Smears of peritoneal fluid can be stained and examined for neutrophils, organic debris, and bacteria.²⁵ The presence of toxic and/or degenerate neutrophils with intracellular bacteria, plant material, or bile crystals is an indication for surgical exploration. Bacteria may either be present within the cells (phagocytized more than 2 hours after the insult) or free within the fluid. Finding a mixed bacterial population and/or organic debris suggests a GI leak (**FIGURE 5**). If the sample is <0.5 mL or if diagnostic peritoneal lavage was performed, analysis is limited to WBC morphology and the presence or absence of bacteria and debris. Septic exudates contain at least 7000 and often >10,000 WBCs/ μ L (normal: <2500 cells/ μ L)



Table 3. Sensitivity and Specificity of Laboratory Results for the Diagnosis of Septic Peritonitis in Dogs and Cats

Study	Result	Sensitivity	Specificity
Bonczynski et al ^a	Abdominal effusion glucose concentration <55 mg/dL	57% (dogs)	100% (dogs)
Bonczynski et al ^a	Peripheral blood glucose concentration >20 mg/dL higher than the abdominal fluid glucose concentration	100% (dogs), 86% (cats)	100% (dogs and cats)
Levin et al ^b	Abdominal fluid lactate concentration >2.5 mmol/L	100% (dogs), 67% (cats)	91% (dogs), 67% (cats)
Bonczynski et al, ^a Levin et al ^b	>2.0 mmol/L difference in the lactate values of abdominal fluid and blood samples	63% to 100% (dogs)	100% (dogs)

^aBonczynski JJ, Ludwig LL, Barton LJ, et al. Comparison of peritoneal fluid and peripheral blood pH, bicarbonate, glucose, and lactate concentration as a diagnostic tool for septic peritonitis in dogs and cats. *Vet Surg* 2003;32(2):161-166.

^bLevin GM, Bonczynski JJ, Ludwig LL, et al. Lactate as a diagnostic test for septic peritoneal effusions in dogs and cats. *J Am Anim Hosp Assoc* 2004;40(5):364-371.

and a total protein level >3.0 g/dL (normal: <2.0 g/dL).²⁵ Cytologic analysis performed soon after gastric perforation may show an abundance of inflammatory cells and scant bacteria compared with lower intestinal rupture. Recent surgery will induce a mild, non-septic peritonitis with mature nontoxic neutrophils and <10,000 WBCs/ μ L and no evidence of organic debris or bacteria.²⁵ The WBC count in the peritoneal effusion of patients with primary septic peritonitis is lower than that in patients with secondary septic peritonitis.³ In one study on primary septic peritonitis, the median WBC count of the peritoneal effusion was approximately 7000 cells/ μ L in dogs and 3000 cells/ μ L in cats.³ The peritoneal effusion was classified as an exudate in 75% of the dogs and 55.5% of the cats.

Aerobic and anaerobic culture and sensitivity testing of peritoneal fluid are recommended. Specific biochemical testing (glucose, lactate, creatinine, or bilirubin levels) may also be useful. The sensitivity and specificity of some laboratory results for the diagnosis of septic peritonitis are summarized in **TABLE 3**. A peritoneal fluid creatinine concentration in excess of serum creatinine concentration is diagnostic for uroperitoneum in dogs and cats. A peritoneal fluid bilirubin concentration in excess of serum bilirubin concentration is diagnostic for bile peritonitis in dogs and cats.

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1. Normal abdominal fluid production in small animals is approximately _____ mL/kg/d.

- a. 20
- b. 40
- c. 80
- d. 160

2. The most common cause of secondary septic peritonitis in small animals is

- a. feline infectious peritonitis.
- b. loss of integrity of the GI tract.
- c. uroabdomen.
- d. gallbladder mucocele rupture.

3. Dehiscence after intestinal surgery most commonly occurs _____ after the surgery.

- a. during the first 12 hours
- b. during the first 3 days
- c. between 3 and 9 days
- d. between 5 and 15 days

4. One of the most common bacteria involved in septic peritonitis is

- a. *Staphylococcus aureus*.
- b. *Streptococcus intermedius*.
- c. *Pseudomonas aeruginosa*.
- d. *Escherichia coli*.

5. Local manifestations of septic peritonitis usually do not involve the

- a. immune system.
- b. urinary system.
- c. peritoneum.
- d. digestive system.

6. Early signs of shock in dogs include

- a. tachycardia, prolonged CRT, and weak pulses.
- b. tachycardia, rapid CRT, and bounding pulses.
- c. bradycardia, prolonged CRT, and hypothermia.
- d. bradycardia, rapid CRT, and weak pulses.

7. At least _____ of free peritoneal fluid is required to detect a fluid wave.

- a. 10 mL/kg
- b. 40 mL/kg
- c. 100 mL/kg
- d. 200 mL/kg

8. What is the minimum amount of peritoneal fluid needed to consistently diagnose peritoneal effusion from abdominal radiographs?

- a. 5.2 mL/kg
- b. 8.8 mL/kg
- c. 18.5 mL/kg
- d. 55 mL/kg

9. How much warm saline solution is infused into the abdomen to perform a diagnostic peritoneal lavage?

- a. 20 mL/kg
- b. 50 mL/kg
- c. 100 mL/kg
- d. 200 mL/kg

10. Which is characteristic of the composition of peritoneal fluid after abdominal surgery?

- a. a high WBC count and organic debris
- b. degenerate neutrophils
- c. a lactate concentration >5 mmol/L
- d. mature nontoxic neutrophils with no evidence of bacteria