

# Septic Peritonitis

William T. N. Culp, VMD, DACVS  
University of California-Davis

David E. Holt, BVSc, DACVS  
University of Pennsylvania

**Abstract:** Bacterial septic peritonitis is a serious condition that requires immediate treatment. The pathogenesis is complex, and the list of diagnostic differentials is extensive. The keys to successful treatment are early recognition of the condition and elimination of the causative organism. Multiple options for draining the peritoneal cavity exist, and further studies are necessary to establish specific, evidence-based guidelines. The prognosis is generally guarded in dogs and cats. Much depends on whether the patient develops concurrent sepsis, systemic inflammatory response syndrome, or multiple organ dysfunction syndrome.

Septic peritonitis is a life-threatening condition that occurs secondary to many intraabdominal diseases in dogs and cats. This article reviews bacterial peritonitis and sepsis and describes treatment options and prognosis.

## Anatomy and Intrinsic Defense Systems of the Peritoneal Cavity

### Mesothelium

The peritoneal cavity is lined with a serous layer of mesothelial cells.<sup>1-3</sup> After peritoneal injury, regeneration begins with an influx of round cells that eventually transform into mesothelial cells. This regeneration can occur as rapidly as 4 hours after injury.<sup>2</sup>

### Intraperitoneal Fluid

A small amount of fluid is normally found within the peritoneal cavity. This fluid constantly lubricates organs to prevent friction. Normal human peritoneal fluid has been shown to have antimicrobial activity against gram-positive and gram-negative bacteria.<sup>4</sup>

Fluid injected into the abdomen is dispersed throughout the peritoneal cavity within 15 minutes to 2 hours.<sup>5</sup> It is likely that peritoneal contaminants are disseminated by a similar movement of peritoneal fluid.<sup>2,6</sup> Fluid moves toward the diaphragm, where it is absorbed by diaphragmatic lymphatics.<sup>6-8</sup> Bacteria in contaminated peritoneal fluid penetrate the diaphragm through 8- to 12- $\mu$ m gaps (stomata) between the peritoneal mesothelial cells and enter specialized lymphatic collecting vessels (lacunae).<sup>7</sup> The bacteria travel from these vessels to the mediastinal lymph node and eventually to the thoracic duct system and bloodstream.<sup>8,9</sup>

### Neutrophils, Macrophages, and Mast Cells

Normally, the peritoneal cavity contains a diverse array of cells capable of reacting to antigens.<sup>10,11</sup> When a peritoneal injury occurs, vasoactive substances (e.g., histamine) are released. Histamine from degranulating resident peritoneal mast cells stimulates vasodilation and exudation of fluid containing complement and opsonins that are capable of coating bacteria, promoting phagocytosis and encouraging an influx of neutrophils and macrophages into the peritoneal cavity.<sup>7,10,11</sup> Peritoneal mast cells, neutrophils, macrophages, and lymphocytes interact to promote cytokine expression, chemoattraction, and phagocyte recruitment. Phagocytes kill bacteria by generating reactive oxygen species, acidifying vacuoles containing phagocytosed bacteria, and releasing hydrolytic lysosomal enzymes.<sup>11</sup>

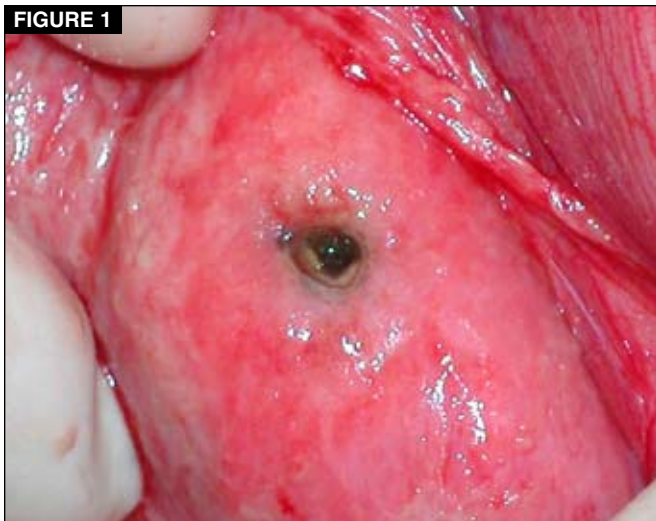
### Complement System

Complement system proteins control inflammation and assist in bacterial phagocytosis.<sup>10</sup> The complement system can be activated by the presence of antigen-antibody complexes, endotoxin, damaged cells, and other components of bacterial cell walls.<sup>12</sup> Activated forms of complement components 3 and 5 are formed and act as anaphylotoxins. The secondary effects of these complement proteins are vasodilation, chemotaxis of neutrophils, degranulation of mast cells and basophils, clearance of immune complexes, and cytolysis.<sup>7,10,11</sup>

### Fibrin and Abscesses

The fluid exuded into the inflamed peritoneal cavity contains plasma proteins, including fibrinogen and thrombo-

FIGURE 1

**Perforated gastric ulcer.**

plastin.<sup>7</sup> Tissue thromboplastin converts prothrombin to thrombin, which activates fibrinogen. Fibrin is generated, polymerizes, and accumulates—in part because fibrinolysis is down-regulated.<sup>13</sup> Fibrin walls off bacteria, preventing phagocytosis by neutrophils and macrophages and leading to abscess formation.<sup>6,7,11</sup> However, fibrin also prevents systemic bacterial spread. In one study performed in mice, prevention of adhesion formation resulted in earlier systemic spread of bacteria and higher mortality.<sup>14</sup>

## Etiology

### Primary Peritonitis

In human medicine, peritonitis is generally categorized as primary, secondary, or tertiary. Primary peritonitis occurs spontaneously, meaning that no obvious intraperitoneal cause of bacterial contamination is found.<sup>8,15,16</sup> This process is more common in people than in companion animals and is generally associated with the presence of ascites secondary to alcoholic cirrhosis.<sup>15,16</sup> The presence of bacteria in the peritoneal cavity in primary peritonitis is attributed to spread via a hematogenous or lymphogenous route or to bacterial translocation through an intact intestinal wall.<sup>15</sup> Feline infectious peritonitis (FIP) is the most common cause of primary peritonitis in companion animals.<sup>8,17</sup> FIP occurs in two major forms: dry (granulomatous) or wet (effusive).<sup>18</sup> The wet form is characterized by abdominal effusion and peritonitis. The pathogenesis of this process is complex and still not fully elucidated.<sup>18</sup> A retrospective study evaluating peritoneal effusion in 65 cats found only four cases of effusive FIP.<sup>19</sup>

### Secondary Peritonitis

Secondary peritonitis is the most common form of peritonitis encountered in companion animals and results from leakage of bacteria-containing fluid into the peritoneal cav-

ity.<sup>16</sup> Any major abdominal organ may act as the septic nidus for peritonitis.

### Gastrointestinal

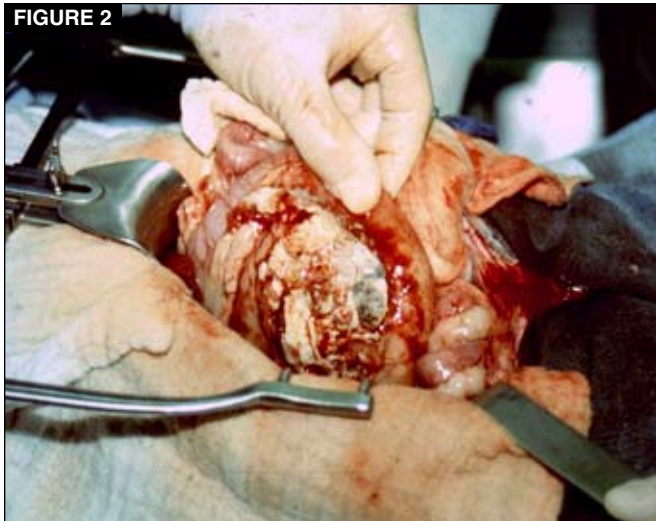
Leakage of contents from the gastrointestinal (GI) tract is the most common cause of septic peritonitis in dogs and cats. GI diseases account for 38% to 75% of cases of secondary septic peritonitis,<sup>20–25</sup> and GI ulceration accounts for 24% to 35% of GI-associated peritonitis cases<sup>21,23,25</sup> (FIGURE 1). Conditions that predispose dogs to GI ulceration include underlying hepatic disease (33%) and administration of NSAIDs and corticosteroids.<sup>26</sup> Concurrent administration of NSAIDs and corticosteroids or administration of NSAIDs at a higher-than-approved dosage may increase the risk of GI ulceration.<sup>27</sup> Many different NSAIDs<sup>28–30</sup> have been linked to ulcer-associated peritonitis in dogs. Carprofen was a potential cause of a duodenal ulcer in a feline case report.<sup>31</sup> Corticosteroid administration has been associated with colonic perforation and peritonitis in dogs with intervertebral disk disease.<sup>32</sup>

Septic peritonitis can also occur after GI resection and anastomosis.<sup>33,34</sup> In one study of 121 dogs that underwent this procedure, the overall dehiscence rate was 15.7%, and dehiscence was often fatal (73.7% mortality).<sup>33</sup> Dogs that required resection and anastomosis for foreign-body entrapment or traumatic injury were significantly more likely to have anastomotic dehiscence. Risk factors associated with anastomotic leakage in another retrospective study included preoperative peritonitis, hypoalbuminemia, and the presence of an intestinal foreign body.<sup>34</sup> The leakage rate for dogs in that study was 14%. None of the 25 cats in the study developed leakage.<sup>34</sup>

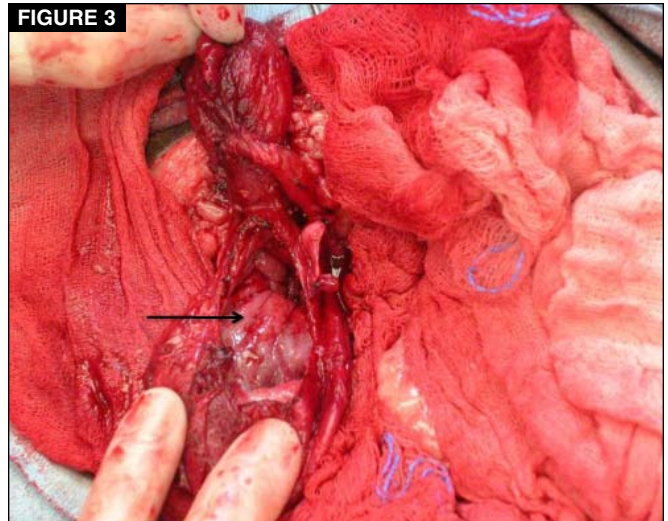
Primary GI neoplasia and non-GI neoplasia (gastrinoma, mast cell tumors)<sup>26</sup> can cause GI perforation. Neoplasia is a common cause of GI leakage in cats, accounting for 25% of septic peritonitis cases in one study.<sup>35</sup> Adenocarcinoma and lymphosarcoma were the most common neoplasms reported.<sup>35</sup>

Other documented causes of GI leakage include gastric necrosis secondary to gastric dilatation–volvulus (GDV), gastropexy site leakage, megacolon perforation, postoperative GI biopsy site dehiscence, uremic gastropathy, stress-induced GI lesions, and traumatic wounds.<sup>8,17,26,34–36</sup>

Bite wounds to the abdomen should be explored immediately to evaluate for viscus rupture that may result in the leakage of bacteria into the peritoneal cavity.<sup>37,38</sup> Bacteria from the attacking animal's mouth can contaminate the peritoneal cavity even in the absence of GI perforation. Gunshot injuries can cause extensive damage to the stomach and intestines. Five dogs in one series of 84 gunshot wounds had peritoneal penetration.<sup>39</sup> All five dogs had multiple, severe intestinal injuries, emphasizing the need for early surgical intervention in such cases.<sup>39</sup>



**FIGURE 2**  
Severe pancreatitis with abscessation.



**FIGURE 3**  
Ruptured prostatic abscess (arrow).

### Biliary Tract

Bile leakage often results in septic peritonitis.<sup>40,41</sup> Bile leakage can occur from trauma, gallbladder infarction, necrotizing cholecystitis, cholelithiasis, and iatrogenic biliary damage (e.g., intraoperative manipulation, gallbladder expression, gallbladder aspiration).<sup>41–43</sup> A retrospective study evaluating 17 dogs and two cats with biliary tract leakage found septic effusion in 10 cases (seven due to necrotizing cholecystitis and three due to trauma).<sup>41</sup> Although not compared statistically, animals in that study with septic bile peritonitis seemed to have a lower survival rate than those with aseptic peritonitis.<sup>41</sup> In another study evaluating extrahepatic biliary surgery in dogs, septic bile peritonitis was significantly associated with mortality.<sup>44</sup>

### Pancreatic/Splenic/Hepatic

Abscessation of the pancreas, liver, or spleen is rare in dogs and cats. Pancreatic abscesses (**FIGURE 2**) are usually sequelae to the development of necrotizing pancreatitis, but they can also occur when a pancreatic pseudocyst becomes infected.<sup>45</sup> Hepatic abscesses have been documented more often than pancreatic or splenic abscesses but are still a very uncommon finding in dogs and cats.<sup>46–49</sup> The development of septic peritonitis secondary to hepatic abscess rupture was only documented in one case among several retrospective studies.<sup>47–49</sup> Septic peritonitis has also been documented secondary to liver lobe torsion in one dog<sup>50</sup> and liver lobe necrosis in one cat.<sup>35</sup>

Splenic abscesses accounted for 0.3% of all splenic disease in dogs in one large retrospective study<sup>51</sup> and 1.7% of all splenic disease in cats in another retrospective study.<sup>52</sup> However, these studies did not mention whether the abscesses were septic or nonseptic, and the feline retrospective study included splenic abscessation in a general category of splenitis.<sup>52</sup>

### Urogenital

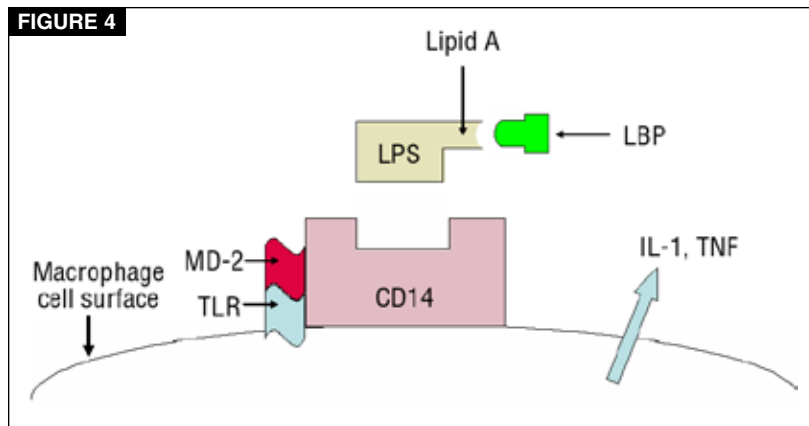
Rupture of a pyometra is a potential but uncommon cause of septic peritonitis. This complication occurs either before surgery or during surgical manipulation of the friable uterus. *Escherichia coli* is the bacteria isolated in most cases of pyometra.<sup>53</sup> Although not routinely evaluated in clinical practice, a lower blood endotoxin concentration is associated with a better prognosis in dogs with pyometra.<sup>54</sup> A retrospective study of pyometra in 183 cats found that seven cats (3.8%) developed uterine rupture. Four of these cats died as a result of septic peritonitis.<sup>55</sup>

Prostatic abscessation can be life-threatening, and rupture of prostatic abscesses (**FIGURE 3**) resulting in septic peritonitis has been reported to occur relatively frequently in dogs (17 of 92 cases; 18%).<sup>56</sup> Seven of these 17 dogs died within 1 week due to sepsis.

Septic peritonitis secondary to infection from the urinary system is rare.<sup>57</sup> Documentation of renal abscessation is limited to a few case reports and a large retrospective study of 61 dogs that included only three cases.<sup>58</sup> Septic peritonitis may also be associated with trauma to the urinary tract and subsequent urine leakage.<sup>58</sup> One retrospective study evaluating uroperitoneum in cats found three positive bacteriologic cultures among the five samples submitted from the peritoneal cavity.<sup>59</sup> *Enterococcus* spp were the most commonly isolated organisms in that study.<sup>59</sup> Four cases of septic peritonitis secondary to urinary tract infection were reported in another retrospective study.<sup>35</sup> Sources of infected urine included a ruptured urinary bladder (n = 2) and leakage from a diseased bladder (n = 1); the source was unknown in a cat with pyelonephritis.<sup>35</sup>

### Tertiary Peritonitis

Tertiary (recurrent) peritonitis has not been documented in dogs and cats. In humans, *tertiary peritonitis* refers to a



**This figure demonstrates the process by which endotoxin (LPS) binds to LPS-binding protein (LBP), accelerating further binding between LPS and CD14 on macrophages. Binding is further promoted by interactions between CD14, TLR, and MD-2. The result of these interactions is the release of cytokines (IL-1, TNF) from the macrophage. (CD14 = cluster differentiation antigen 14, IL-1 = interleukin 1, LPS = lipopolysaccharide, MD-2 = extracellular adaptor protein, TLR = Toll-like receptor, TNF = tumor necrosis factor)**

recurrent infection of the peritoneal cavity after an episode of primary or secondary peritonitis.<sup>60</sup> This syndrome tends to occur after optimal surgical treatment and manifests as systemic sepsis without an evident septic focus.<sup>61,62</sup> Organisms cultured from the peritoneal cavity in cases of tertiary peritonitis tend to come from the GI tract or to be lesser pathogenic organisms such as *Candida* spp or *Staphylococcus epidermidis*.<sup>15,62</sup> Bacterial translocation is thought to play a role in these cases because humans with severe illness are more likely to have intestinal hypoperfusion, altered gut flora, and inadequate gut barrier function.<sup>62</sup> Recent studies of tertiary peritonitis have found a possible association with endocrine dysfunction and immune paralysis.<sup>62</sup>

## Pathophysiology

### Bacteria

The presence of a bacterial contaminant within the peritoneal cavity induces the cascade of events associated with septic peritonitis. In animals, peritonitis is most commonly associated with gram-negative bacteria.<sup>20,21,23,36</sup> The effects of gram-negative bacteria are primarily related to endotoxin (lipopolysaccharide [LPS]). Endotoxin is composed of three parts: lipid A and two groupings of polysaccharides, the R-core and the O-polysaccharide.<sup>7,63</sup> Most toxic effects are attributed to the lipid A segment<sup>64</sup> (FIGURE 4).

Endotoxin binds to LPS-binding protein, thereby accelerating the binding of LPS to the cluster differentiation (CD) 14 antigen gene receptor on macrophages.<sup>7,14,63</sup> A specific interaction between the CD14 receptors, Toll-like receptors (TLRs, which initiate cellular activation), and the lymphocyte antigen MD-2 (another cell surface molecule) also promotes binding.<sup>64–66</sup> This binding stimulates macrophages to release multiple cytokines<sup>67,68</sup> (FIGURE 4). TLR-4 is specifi-

cally important to this cascade, facilitating the intracellular signaling that leads to cytokine release after interacting with MD-2.<sup>65,68</sup>

The process by which gram-positive bacteria induce their effects is less well understood. Gram-positive bacteria contain many substances—including peptidoglycan, teichoic acid, and polysaccharide-hyaluronic acid capsules—that can act as proinflammatory agents.<sup>65,69</sup> Peptidoglycan released by bacteria has demonstrated proinflammatory activities that are mediated via the cell surface molecule CD14. Teichoic acid also seems to use CD14 receptors to induce proinflammatory actions, such as the production of cytokines, including interleukin 1 (IL-1), tumor necrosis factor (TNF), and nitric oxide (NO). Gram-positive capsules are capable of inducing resistance to phagocytosis.<sup>69</sup> Gram-positive bacteria can also produce superantigenic toxins that can lead to T-lymphocyte activation and cytokine release.<sup>65,69</sup>

### Cytokines

The major cytokines in the inflammatory process are IL-1 and TNF- $\alpha$ .<sup>67,70</sup> Both gram-positive and gram-negative bacteria can cause the release of these cytokines.<sup>69,70</sup> IL-1 and TNF- $\alpha$  initiate many events, including activation of the arachidonic acid cascade, production of acute-phase proteins in the liver, and induction of fever and intravascular coagulation.<sup>7,11,61</sup> These cytokines also signal peritoneal mesothelial cells to release interleukin 8 (IL-8),<sup>11</sup> which acts as a chemoattractant for polymorphonuclear leukocytes (PMNs).

Platelet-activating factor (PAF), high mobility group box-1 (HMGB1), and macrophage migration inhibitory factor (MIF) are cytokines or cytokine-like substances that are released from macrophages secondary to TNF- $\alpha$  activation.<sup>61,67</sup> PAF may be produced from the same membrane phospholipid as leukotrienes during inflammation.<sup>71</sup> Leukotrienes and PAF increase vascular permeability and PMN activation.<sup>71</sup> LPS has been shown to increase levels of HMGB1. Patients with increased levels of HMGB1 during sepsis have higher mortality.<sup>67</sup> In mice, MIF has been shown to act as a proinflammatory agent and can alter the antiinflammatory effects of glucocorticoids.<sup>67</sup>

### Hypovolemia/Hypoproteinemia

The combined effects of the inflammatory mediators (especially cytokines) increase vascular permeability and vasodilation.<sup>72</sup> Increased vascular permeability results in fluid and albumin influx into the peritoneal cavity. The resulting decreased albumin concentration in the intravascular space and decreased albumin synthesis in the liver during times of stress<sup>73</sup> allow more fluid to extravasate as oncotic pressure decreases.

As more fluid enters the peritoneal cavity, hypovolemia worsens. This results in decreased venous return to the heart with subsequent decreased cardiac output and poor tissue perfusion. Cardiac compromise may be worsened by the release of a myocardial depressant factor.

### Arachidonic Acid Cascade

The activation of the arachidonic acid cascade by endotoxin and bacteria-induced cytokine release results in the production of leukotrienes, prostacyclins, and prostaglandins.<sup>7,11,12,61</sup> These substances recruit leukocytes such as PMNs and eosinophils to the site of inflammation and can result in vasodilation.<sup>7,61,71</sup>

### Coagulation Abnormalities

Many studies have documented coagulation abnormalities that accompany sepsis.<sup>67,74–76</sup> Both animals and humans have been shown to develop a procoagulant state.<sup>67,75,76</sup> Endotoxin initiates coagulation by stimulating expression of tissue factor,<sup>67,75</sup> which in turn activates the intrinsic and extrinsic coagulation pathways to produce fibrin. Fibrinolysis is impaired by high levels of plasminogen activator inhibitor type 1 and down-regulation of tissue factor pathway inhibitor (TFPI), protein C, protein S, and antithrombin III.<sup>13,67,76</sup>

The role of sepsis in TFPI system dysfunction is not fully understood, but some authors have suggested that TFPI may not be able to deactivate the increased levels of tissue factor associated with sepsis.<sup>13</sup> In addition, the structure of TFPI may be altered by the endothelial injury induced by inflammatory cytokines.<sup>13</sup> Higher amounts of protein C are consumed, contributing to a decrease in levels. The anticoagulant activity of protein C is also decreased by sepsis-induced increases in levels of protein C inhibitor.<sup>13</sup> Antithrombin III levels fall as liver production decreases and peripheral consumption and inactivation by activated neutrophils increase.<sup>13,77</sup> The imbalance associated with a highly procoagulant state and decreased fibrinolytic activity results in clinical disseminated intravascular coagulation (DIC) and the formation of microvascular thromboses, further compromising tissue perfusion.<sup>13,77</sup>

### Adjuvant Substances

Intraperitoneal mucin, bile, foreign bodies, hemoglobin, and blood can worsen septic peritonitis. Gastric mucin decreases the phagocytic activity of intraperitoneal macrophages by exerting an anticomplement effect.<sup>7,8</sup> Bile salts destroy peritoneal mesothelial cells, inhibit PMN function,<sup>11</sup> and lower surface tension, leading to altered cell adhesion and destruction of red blood cells.<sup>8</sup> Foreign material in the peritoneal cavity may induce bacterial translocation.<sup>11</sup> Hemoglobin acts as an adjuvant substance by interfering with chemotaxis and phagocytic activities.<sup>7</sup> Blood can be an excellent growth medium for bacteria,<sup>11</sup> and the presence of intraperitoneal

## Key Facts

- Septic peritonitis may not be immediately recognizable based on physical examination.
- Peritoneal fluid analysis is essential to diagnosing and treating septic peritonitis.
- Severe sequelae such as systemic inflammatory response syndrome or multiple organ dysfunction syndrome may still occur after the nidus of infection has been removed.

fluid has also been shown to lead to increased bacterial numbers and decreased bacterial clearance.<sup>8</sup>

### Nitric Oxide

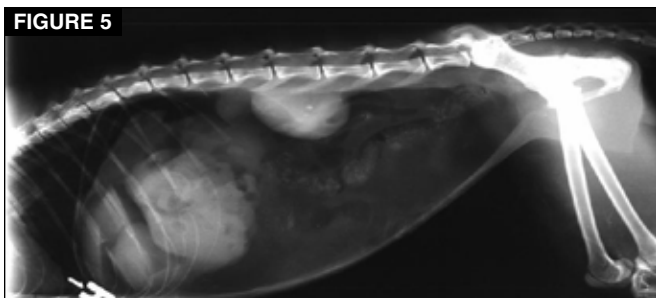
Endotoxin, interferon  $\gamma$  (IFN- $\gamma$ ), lipoteichoic acid, and peptidoglycan stimulate macrophage production of NO.<sup>65,69</sup> In addition, IL-1 and TNF can initiate transcription and translation of NO synthase.<sup>69</sup> NO causes vasodilation, decreasing systemic vascular resistance and resulting in hypotension.<sup>70</sup> NO can also interact with reactive oxygen molecules (e.g., superoxide) to generate a series of cytotoxic nitrogen radicals, including peroxynitrite.<sup>65</sup>

All of these processes culminate in the initiation of systemic inflammatory response syndrome (SIRS) and sepsis. Antiinflammatory cytokines are produced to attenuate this massive reaction but can be overwhelmed. Eventually, the effects of vascular compromise, poor tissue perfusion, thromboembolism, and infarction manifest as multiple organ dysfunction syndrome (MODS), including acute respiratory distress syndrome, pulmonary thromboembolism, pleural effusion, renal hypoperfusion and infarction, GI hypoperfusion and perforation, vomiting, diarrhea, malaise, and neurologic infarction.

### Clinical Signs

Dogs and cats with septic peritonitis generally present with vague signs. Many have a history of lethargy, depression, vomiting, diarrhea, or collapse. These signs are nonspecific, and unless the history includes a penetrating peritoneal wound or obvious trauma, the index of suspicion for peritonitis may not be high. Clinical signs of vomiting and diarrhea may be associated with a primary GI problem (e.g., foreign body) or may occur due to the developing peritonitis. Icterus may be encountered in animals with severe pancreatitis or primary biliary disease. Hematuria, pollakiuria, polyuria, and polydipsia are common indications of urogenital diseases such as urinary tract infection or pyometra. Abdominal discomfort and distention are often noticed on abdominal palpation.

Many animals with septic peritonitis present in shock



**FIGURE 5**  
**Pneumoperitoneum in a cat secondary to GI perforation due to lymphoma.**

due to hypovolemia from intraperitoneal fluid loss and the effects of SIRS. Animals may present in one of three broad stages of shock: compensatory, early decompensatory, and terminal decompensatory. The clinical signs depend on when the animal is evaluated.<sup>78</sup> In the compensatory stage of shock, animals are generally hypermetabolic with tachycardia, tachypnea, and hypertension and may have injected mucous membranes with a rapid capillary refill time. In early decompensatory shock, tachycardia continues, but blood pressure decreases. In addition, the animal may have depressed mentation, poor pulse quality, pale mucous membranes, and a prolonged capillary refill time. Tachycardia is not as common in cats as in dogs during this stage. When terminal decompensatory shock is reached, bradycardia, hypotension, pale or cyanotic membranes, weak or absent pulses, and severe mental depression develop. Renal failure and pulmonary edema may also be encountered.<sup>78,79</sup>

## Diagnostics

### Clinicopathologic Findings

Anemia and leukocytosis are common abnormalities noted on complete blood cell count in animals with septic peritonitis. Band neutrophils are often present, and if the infection is long-standing, the animal may be leukopenic. Coagulation profiles may be normal in the early stages of septic peritonitis, but prothrombin time (PT) and activated partial thromboplastin time (aPTT) often become prolonged. In animals with DIC, thrombocytopenia and higher levels of fibrin degradation products and D-dimer may be encountered. A hypercoagulable state can be detected by thromboelastography.<sup>80</sup>

Serum biochemistry findings vary widely depending on the underlying cause and stage of the peritonitis. Extravasation of protein-rich fluid rapidly leads to hypoalbuminemia. Increases in blood urea nitrogen and creatinine values may be prerenal (i.e., from decreased kidney perfusion) or associated with primary renal disease such as pyelonephritis or (rarely) renal abscessation. Uroperitoneum may also result in azotemia or alterations in electrolytes. Liver enzyme levels increase with many primary hepatic and biliary diseases, such as biliary mucocele, gallbladder infar-

tion, and cholelithiasis.<sup>43</sup> Liver enzyme values may also rise in animals without primary liver disease and may be related to alterations in hepatic perfusion.<sup>35</sup>

Animals with poor perfusion or sepsis may have hyperlactatemia.<sup>81</sup> The plasma lactate level has been shown to be a prognostic indicator in dogs with GDV,<sup>82</sup> but its usefulness as a prognostic indicator in septic peritonitis is less clear. Septic patients may demonstrate hyperglycemia or hypoglycemia.<sup>83</sup> Proposed mechanisms for hypoglycemia include endotoxin-induced hepatic glycogen depletion, increased peripheral glucose use, and impaired gluconeogenesis.<sup>83</sup>

### Radiography and Ultrasonography

Survey radiography is generally a first-line diagnostic tool when evaluating a dog or cat with suspected peritonitis. Loss of abdominal serosal detail, ileus, and pneumoperitoneum are radiographic signs suggestive of intraperitoneal disease. Loss of serosal detail can be caused by free peritoneal fluid or a mass effect. Ileus can be seen as a result of bowel obstruction secondary to neoplasia or foreign body ingestion but may also be induced by peritonitis.<sup>84</sup> Pneumoperitoneum that occurs without any history of recent surgery or penetrating wounds is most commonly of GI origin<sup>85</sup> and warrants immediate surgical exploration (**FIGURE 5**). Radiography can also be used to diagnose specific diseases, including GDV, GI foreign bodies,<sup>86</sup> pyometra,<sup>53</sup> cholelithiasis, biliary emphysema,<sup>43</sup> and organomegaly secondary to intraabdominal abscessation (hepatic, splenic, renal, prostatic)<sup>46,47,49</sup> or neoplasia.<sup>86</sup>

Ultrasonography can generally localize a tumor or mass to a specific organ and guide sampling of free peritoneal fluid. Specific findings on ultrasonography relative to the many disease processes that can lead to septic peritonitis are beyond the scope of this article.

### Peritoneal Fluid Analysis

#### Sample Collection

Several techniques can be used to obtain a sample of fluid from the peritoneal cavity and have been detailed elsewhere.<sup>87-91</sup> All techniques involve clipping the hair in the area that will be tapped, preparing the abdomen with sterile technique, and sterile introduction of a needle or an over-the-needle catheter. Ideally, this procedure should be performed with ultrasonographic guidance. Although definitive studies are lacking, clinical experience with ultrasound-guided fluid aspiration suggests that this technique has a much higher yield than blind peritoneal tapping and decreases the need for diagnostic peritoneal lavage (DPL). DPL is likewise superior to blind needle paracentesis.<sup>92</sup> A study comparing needle paracentesis, catheter paracentesis, and DPL found an accuracy rate of less than 50% for needle paracentesis compared with 94.6% for DPL. DPL was 100% accurate in dogs with septic peritonitis.<sup>92</sup>

Complications are uncommon with these techniques. Needle paracentesis is generally considered safe, but there is some disagreement about whether a syringe should be attached to the needle.<sup>92</sup> Some authors believe that the lack of syringe attachment allows for the introduction of air, causing pneumoperitoneum that confounds diagnostics.<sup>91</sup> Others think that suction with a syringe may increase the risk of obstruction with omentum or abdominal viscera and impede sample collection.<sup>91</sup> Overall complication rates for the placement of peritoneal dialysis catheters and DPL have been reported at 4.7%; for significant complications (e.g., splenic laceration, urinary bladder puncture), the rate is 1.6%.<sup>91</sup>

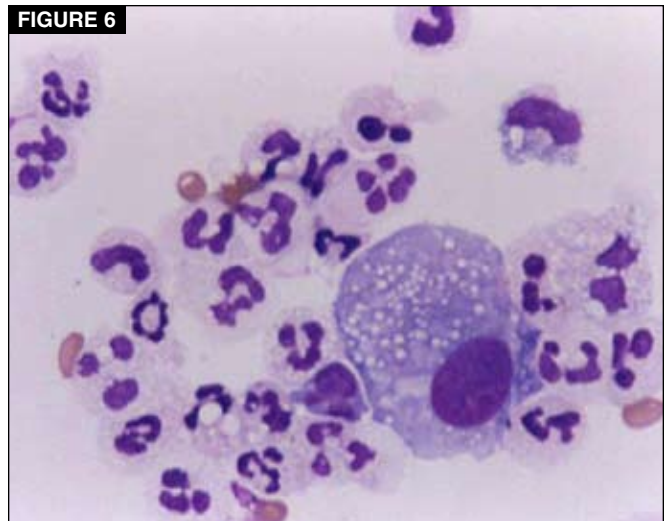
### Cytology

Normal cell counts for the peritoneal cavity are  $<3000 \times 10^6/L$  in small animals.<sup>89</sup> One study<sup>93</sup> evaluated the use of DPL preoperatively and postoperatively to assess cell count and intraabdominal enzyme content. The surgeries performed in that study included GI resection and anastomosis, cystotomy, gastrotomy, pyloromyotomy, and repair of a diaphragmatic laceration. Macrophages and segmented neutrophils were the most common cells found in the peritoneal fluid, and preoperative nucleated cell counts ranged from 71 to 697 cells/ $\mu L$ . White blood cell and nucleated cell counts were significantly increased in postoperative fluid compared with preoperative fluid.<sup>93</sup>

Septic effusions generally contain increased numbers of neutrophils and macrophages, and intracellular or extracellular bacteria may be present (FIGURE 6). Peritoneal fluid samples are specifically evaluated for degenerate neutrophils. Bacterial toxins cause changes in the cell membrane permeability of neutrophils, leading to cell and/or nuclear swelling, nuclear fragmentation, and eosinophilic nuclear chromatin.<sup>88,90</sup> The lack of degenerate neutrophils does not rule out septic peritonitis,<sup>88</sup> and it is important to differentiate degenerate neutrophils from aging and toxic neutrophils.<sup>94</sup> The presence of intracellular bacteria is diagnostic for septic peritonitis.<sup>90</sup>

Reagent strips that test for the presence of leukocytes in peritoneal fluid have been used to diagnose primary peritonitis in humans. A PMN count of  $\geq 250$  cells/ $\mu L$  in ascitic fluid is considered the “gold standard” for diagnosis of primary peritonitis, and in humans, the sensitivity and specificity of some of these strips have been reported as high as 88.2% and 99.6%, respectively.<sup>95</sup> The use of these strips to diagnose peritonitis in small animals has not been evaluated.

Results of using total peritoneal fluid leukocyte counts and cytology to diagnose septic peritonitis have been variable. Peritoneal leukocyte counts ranged from 5,400/ $\mu L$  to 43,100/ $\mu L$  in an experimental peritonitis model.<sup>93</sup> One study found an 86% sensitivity and a 100% specificity for peritonitis in dogs and 100% sensitivity and specificity in cats when



**FIGURE 6**  
Peritoneal fluid cytology shows (1) predominantly neutrophils that are mildly to moderately degenerate in morphology and (2) occasional mononuclear cells. A large mesothelial cell is present in the center of the image.

the peritoneal fluid total nucleated cell count was  $>13,000$  cells/ $\mu L$ .<sup>96</sup> Cytology was diagnostic for septic peritoneal effusion in 86% of the cases in the latter study.<sup>96</sup> Other reports have found cytology to be accurate in diagnosing septic peritonitis 57%,<sup>25</sup> 87%,<sup>26</sup> and 100%<sup>96</sup> of the time.

### pH

Peritoneal fluid pH as an indicator of septic peritonitis has been evaluated in dogs and cats. In one study,<sup>96</sup> the pH of the fluid in septic effusions in cats was significantly lower than the pH of the fluid in nonseptic effusions. This study did not find the same to be true in dogs. Comparison of blood pH to peritoneal fluid pH was not found to be useful for dogs or cats.<sup>96</sup>

### Glucose

A study has found that comparing blood glucose levels to peritoneal glucose levels is diagnostic for peritonitis in 100% of dogs and 92% of cats.<sup>96</sup> When the blood glucose concentration exceeded the peritoneal fluid glucose concentration by  $>20$  mg/dL, the sensitivity and specificity were 100% in dogs and 86% and 100%, respectively, in cats.<sup>96</sup>

### Lactate

Peritoneal fluid lactate concentration has been evaluated in two studies. In the first,<sup>96</sup> a blood-to-peritoneal fluid lactate concentration of less than  $-2.0$  mmol/L was found to be 100% sensitive and specific for the diagnosis of a septic peritoneal effusion in dogs but did not reach statistical significance in cats. The second study<sup>97</sup> reported an accuracy of 95% in the diagnosis of septic peritonitis in dogs when the peritoneal fluid lactate concentration was  $>2.5$  mmol/L. When using a blood-to-peritoneal fluid lactate concentration difference of

## BOX 1

**Medical Management Checklist for the Treatment of Septic Peritonitis<sup>a</sup>****Fluid therapy**

- Crystalloids
  - Plasmalyte A
  - Lactated Ringer's solution
  - Normosol-R
- Colloids
  - Hetastarch
  - Dextrans

**Blood products**

- Fresh whole blood
- Stored whole blood
- Packed red blood cells
- Plasma
- Albumin

**Analgesia**

- Intravenous bolus
  - Buprenorphine: 0.005–0.02 mg/kg IV, IM, SC q4–6h
- Constant-rate infusion
  - Fentanyl: 1–5 µg/kg/h
  - Lidocaine: 30–50 µg/kg/min
  - Ketamine: 0.1 mg/kg/h

**Gastrointestinal protectants**

- Antiemetics
  - Metoclopramide: 0.1–0.5 mg/kg PO, SC q8h or 1–2 mg/kg/d IV CRI
  - Dolasetron: 0.6–1 mg/kg/d IV, PO
  - Chlorpromazine: 0.2–0.5 mg/kg IM, SC q6–8h
- Antisecretory
  - Famotidine: 0.5–1 mg/kg IV, IM, PO, SC q12–24h
  - Omeprazole: 0.5–1 mg/kg PO q24h
- Cytoprotective
  - Sucralfate: 0.5–1 g PO q8h (dogs)  
0.25–0.5 g PO q8–12h (cats)
  - Misoprostol: 1–5 µg/kg PO q6–8h

**Antibiotics (empiric)**

- Gram-positive coverage
  - Ampicillin: 22 mg/kg IV q8h
  - Clindamycin: 10 mg/kg IV, PO q12h
- Gram-negative coverage
  - Enrofloxacin: 10–15 mg/kg IV, PO (dogs)  
5 mg/kg IV, PO (cats)
  - Cefotaxime: 25–50 mg/kg IV, IM, SC q8h
- Anaerobic coverage
  - Metronidazole: 10 mg/kg IV, PO q12h

<sup>a</sup>All dosages listed are for dogs and cats unless otherwise indicated.

less than –1.5 mmol/L, the accuracy was 90% for the diagnosis of septic peritoneal effusion in dogs. Lactate concentration differences were diagnostically inaccurate in cats.<sup>97</sup>

**Culture**

Gram-negative bacteria predominate in septic peritonitis. *E. coli* is the most commonly isolated organism, being found in 57% to 74% of cultures from the peritoneal cavity. Other common gram-negative bacteria include *Klebsiella* and *Bacteroides* spp.<sup>20,21,25,36</sup> Gram-positive bacteria that have been isolated include *Streptococcus*, *Staphylococcus*, *Enterococcus*, and *Clostridia* spp.<sup>20,21,23,36</sup> Commonly, multiple species of bacteria are isolated from a septic peritoneum. In one report, multiple organisms were isolated in 15 of 19 (79%) cases.<sup>36</sup>

In dogs with cholelithiasis, bile cultures are often positive for aerobic or anaerobic bacteria. One study<sup>40</sup> found a positive aerobic culture in 14 of 20 dogs and a positive anaerobic culture in eight of 18 dogs. Although cholelithiasis with secondary biliary tract rupture is uncommon in dogs, it should be considered in the differential diagnosis when a dog has septic peritonitis.

In 23 cases of necrotizing cholecystitis treated surgically, bacteria were found in 13 of 16 (81%) of gallbladder mucosa samples submitted for culture. The most common bacterial isolate was *E. coli* (69%). Nine of the 23 dogs died in the perioperative period; the deaths were attributed to sepsis, peritonitis, or debilitation.<sup>98</sup> *E. coli* has also been isolated in 59% to 96% of cases of pyometra<sup>52</sup> and approximately 85% of cases of prostatic abscessation.<sup>55</sup>

**Medical Management****Fluids**

Fluid resuscitation is essential to the management of dogs and cats with septic peritonitis. The goal of resuscitation is to restore and maintain tissue perfusion. The loss of large volumes of fluid and protein from the vasculature into the peritoneal cavity, combined with vasodilation secondary to systemic inflammation, can result in shock. **BOX 1** provides a checklist for the treatment of septic peritonitis.

Both crystalloids and colloids are often necessary for adequate resuscitation. Intravenous (IV) crystalloid solutions are the initial mainstay of intravascular volume replacement. Ongoing resuscitation with crystalloids alone may lead to an increased capillary hydrostatic pressure and a decreased plasma oncotic pressure. This decreased oncotic pressure from dilutional and sepsis-associated hypoalbuminemia, combined with the increased vascular permeability seen in sepsis, can result in significant fluid extravasation from the vasculature into the interstitial tissue spaces.

Synthetic colloids such as hetastarch and dextrans decrease the fluid volume requirement for resuscitation. Natural colloids such as fresh whole blood, stored whole blood, and plasma can also be given but do not provide



as significant an increase in colloid osmotic pressure (COP) as do synthetic colloids. Synthetic and natural colloids are often administered simultaneously because natural colloids can provide other important substances such as coagulation factors and albumin.

Several factors should be considered when administering crystalloids and colloids. In a patient with shock, boluses of both fluid types may be necessary. A shock bolus of crystalloids in dogs (90 mL/kg) and cats (55 mL/kg) should be given over 10 to 15 minutes.<sup>8</sup> The initial bolus of hetastarch in shock patients is 10 to 20 mL/kg given as quickly as possible.<sup>99</sup> Further boluses of these fluids may be required depending on the patient's status. If further colloidal support is necessary, synthetic colloids can be continued as a constant-rate infusion (CRI) at a dose of 1 mL/kg/h.<sup>99</sup> Higher doses may be necessary if the COP remains low. When administering CRIs of crystalloids and colloids simultaneously, the amount of crystalloid can be decreased by as much as 40% to 60%.<sup>99</sup>

Fluid resuscitation goals include maintaining the mean arterial blood pressure above 80 mm Hg and the heart rate between 80 and 120 bpm in dogs and 180 and 200 bpm in cats. Other parameters to maintain include central venous pressure (6 to 8 cm H<sub>2</sub>O) and urine output (>1 mL/kg/h). New human guidelines focus on arterial lactate levels and mixed venous oxygen saturation as more sensitive and specific indicators of tissue perfusion.<sup>100</sup> When administering synthetic colloids, a COP of 17 mm Hg should be maintained while avoiding volume overload.<sup>99,101</sup>

### Blood Products

Blood products can be required in animals with septic peritonitis for a variety of reasons. Animals with bleeding GI ulcers, peritonitis secondary to trauma, GDV, or avulsion of the short gastric blood vessels may need transfusions of whole blood or packed red blood cells. The decision to transfuse an anemic patient with whole blood or packed red blood cells depends on several factors. In dogs and cats, transfusions should often be given if the packed cell volume (PCV) drops below 20%.<sup>102</sup> Other factors to consider include a massive loss of blood volume (>30%), ongoing hemorrhage, inadequate response to fluid resuscitation, or severe clinical signs (e.g., collapse). Transfusing 10 mL/kg of packed red blood cells or 20 mL/kg of whole blood can increase the PCV by 10%.<sup>102</sup>

Whole blood, packed red blood cells, and plasma can be used for colloidal support, but these products can raise the oncotic pressure by only a limited amount.<sup>101</sup> This increase is often transient because of the ongoing loss of albumin into the peritoneal cavity. However, fresh frozen plasma transfusions are often used clinically to treat coagulation abnormalities associated with peritonitis and sepsis. Coagulation abnormalities should be suspected in animals

with biliary obstruction and rupture because fat-soluble vitamins, including vitamin K, cannot be absorbed without bile emulsification of fat. Coagulation abnormalities are also commonly associated with nonbiliary peritonitis. In one study, septic dogs were found to have significantly longer PT and aPTT and higher levels of fibrin degradation products and D-dimers than control dogs.<sup>74</sup> When the PT and aPTT are prolonged, plasma should be given until these values are within normal limits.

An animal receiving a transfusion of blood products should be watched carefully. It is especially important to monitor pulse, temperature, and respiratory rate during the first 30 minutes of the transfusion. If a patient appears to be experiencing a transfusion reaction, the transfusion should be stopped immediately.<sup>103</sup>

### Antibiotics

If septic peritonitis is suspected, antibiotic use is mandatory. Ideally, the antibiotic regimen should be based on the results of culture and susceptibility testing. However, in most cases, empirical therapy is instituted while awaiting these test results. Dosing should be based on an understanding of the pharmacodynamic properties that predict antimicrobial efficacy.

Antibiotic pharmacology is reviewed extensively elsewhere.<sup>104</sup> In general,  $\beta$ -lactam drugs (e.g., penicillins, cephalosporins) are classified as time-dependent, meaning that their bactericidal effect corresponds to the amount of time their plasma and tissue concentrations remain above the minimum inhibitory concentration for the pathogen. Therefore,  $\beta$ -lactams should be administered frequently to enhance efficacy. Conversely, fluoroquinolones and aminoglycosides are concentration-dependent drugs with maximum effect when a high peak plasma concentration is attained, even if only for a short period of time. Therefore, the entire dose of these drugs is appropriately administered once daily. General recommendations for broad-spectrum empiric antibiotic treatment in cases of septic peritonitis include either a penicillin or cephalosporin for gram-positive coverage combined with either a fluoroquinolone or an aminoglycoside for gram-negative coverage until culture and susceptibility data become available.

Anaerobes are a large component of normal GI flora, especially in the colon. Not all anaerobes are susceptible to penicillins, and many are resistant to cephalosporins.<sup>105</sup> The addition of an antibiotic with documented or enhanced antianaerobic activity (e.g., metronidazole) is often justified and should be considered empirically while awaiting culture results.

### Analgesics

Abdominal pain has been extensively described elsewhere<sup>86</sup> and may be either visceral (dull) or somatic (piercing).

Analgesia is an important aspect of presurgical and post-surgical management. Opioids are a first-line choice for the treatment of pain in patients with septic peritonitis. Some opioids, such as morphine and hydromorphone, can induce GI ileus and vomiting and cause dose-dependent respiratory depression. These drugs should be avoided in patients for whom further GI upset is contraindicated. Buprenorphine tends to have fewer GI effects and may be a better option for these patients. The usual dose of buprenorphine in dogs and cats is between 0.005 and 0.02 mg/kg. CRIs of analgesics may also be required. Common medications that are used in analgesic CRIs include fentanyl, lidocaine, and ketamine.

### Gastrointestinal Protectants

Most dogs and cats with septic peritonitis benefit from GI protectants. As these animals often experience vomiting, antiemetics such as dopamine antagonists, serotonin antagonists, and phenothiazines may be indicated. Metoclopramide, a dopamine antagonist, has some prokinetic activity and is contraindicated in cases of GI obstruction because increases in peristalsis may lead to further “bunching” of intestine. Phenothiazines have been associated with sedation and hypotension.<sup>106</sup>

Drugs that decrease gastric acid secretion may also be used. Commonly employed H<sub>2</sub>-receptor antagonists include famotidine and cimetidine. Proton pump inhibitors (e.g., omeprazole) are considered better alternatives to H<sub>2</sub>-receptor antagonists in patients with gastric ulcers.<sup>106</sup>

Sucralfate and misoprostol are commonly used in cases of GI ulceration. Sucralfate binds with ulcers and coats the mucosa to prevent acid damage. Misoprostol has both anti-secretory and cytoprotective properties, directly inducing GI ulcer healing.

### Other Medical Treatments

#### Corticosteroids

The use of corticosteroids in the treatment of septic peritonitis remains controversial.<sup>107</sup> Steroids were previously advocated in the treatment of septic shock, based on membrane stabilization and decreased vascular permeability.<sup>1</sup> Steroids also exert antiinflammatory effects by modulating cytokines (including IL-1 and TNF) and stimulating production of anti-inflammatory factors such as IL-10 and IL-1 receptor antagonist.<sup>108</sup> However, a lack of efficacy in the treatment of sepsis and septic shock has been reported,<sup>107</sup> and their routine use has fallen out of favor.

Recently, much attention has been paid in human medicine to the early treatment of adrenal insufficiency in septic patients.<sup>108,109</sup> In humans, sepsis and SIRS are the most common causes of acute adrenal insufficiency,<sup>108</sup> decreasing production of the glucocorticoids that help to modulate the inflammatory response in sepsis.<sup>109</sup>

It may be beneficial to obtain a baseline cortisol level in

dogs and cats with septic peritonitis. In humans, physiologic doses of hydrocortisone and fludrocortisone in a group of septic patients with inadequate adrenocorticotropic hormone stimulation resulted in a significantly reduced risk of death at 28 days.<sup>110</sup> The incidence and role of adrenal insufficiency in septic peritonitis is under investigation in companion animals.

The dose of corticosteroids in septic patients varies based on the purpose of administration. High doses of corticosteroids (hydrocortisone, 150 mg/kg, and dexamethasone, 4 to 10 mg/kg)<sup>111</sup> have been used in the treatment of shock, whereas much lower “stress” doses (hydrocortisone, 1 mg/kg,<sup>112</sup> and dexamethasone, 0.5 to 1 mg/kg) are used to treat relative adrenal insufficiency.

#### Insulin

As with humans, septic companion animals often develop insulin resistance.<sup>113,114</sup> A study<sup>114</sup> evaluating intensive insulin therapy in 1548 critically ill human patients found a decreased mortality in those whose glucose levels were maintained with insulin at  $\leq 110$  mg/dL. Patients with a septic focus and secondary MODS had the greatest reduction in mortality.<sup>114</sup>

#### Protein C

Protein C levels can be decreased in people<sup>74</sup> and dogs<sup>75</sup> with sepsis. A multicenter study<sup>74</sup> evaluating the administration of protein C to humans with septic conditions found some promising results. In that study, 840 patients received a placebo and 850 patients received activated protein C. The patients receiving the protein C had a 19.4% reduction in the relative risk of death and a 6.1% reduction in the absolute risk of death compared with those in the control group. However, the incidence of bleeding was higher in the group that received activated protein C.<sup>74</sup>

#### Fluconazole

The antifungal azoles have demonstrated antiinflammatory properties by interfering with leukocyte function.<sup>115</sup> It has been suggested that fluconazole may decrease mortality in patients with bacterial sepsis.<sup>115</sup> Fluconazole caused no side effects (e.g., hepatotoxicosis) in a group of dogs with bacterial peritonitis, but the number of subjects was too small to determine whether fluconazole therapy produced any improvement in outcome.<sup>116</sup>

### Surgical Management

Despite the induction of numerous inflammatory cascades in septic peritonitis, the crucial therapeutic intervention is surgical removal of the nidus of infection, once the animal is stabilized. After the patient is anesthetized, the hair over the abdomen should be generously clipped and the area prepared with sterile technique and draped. The surgical approach involves a large ventral midline incision often

extending from just caudal to the xiphoid cartilage to just cranial to the pubis.

Obvious contamination of the peritoneal cavity should be removed immediately and thorough abdominal exploration performed. When the inciting organ is identified, it should be isolated from the rest of the peritoneal cavity with laparotomy sponges.

Leakage from the GI tract is the most common cause of bacterial peritonitis. Perforated gastric ulcers can generally be treated by partial gastrectomy. Duodenal ulcer resection may be performed by a local resection of diseased tissue, Y-U antral advancement flap pyloroplasty, or biliary rerouting, depending on the location and size of the ulcer. It is essential that sutured bowel edges be healthy and demonstrate sufficient blood supply. If a previous GI anastomosis has dehisced, the entire area must be removed and another anastomosis performed. The anastomosis site is reinforced by omental wrapping or serosal patching.<sup>117</sup> Serosal patching has been used to treat defects in the small intestine, colon, and urinary bladder.<sup>118</sup> This technique involves suturing loops of jejunum over an area of questionable viability.

As pancreatic abscesses are uncommon and little studied, results of various treatment methods should be interpreted with caution. Omentalization of the abscess cavity and abdominal closure resulted in better survival (five of eight patients) than treatment by open peritoneal drainage (one of four patients) in one study.<sup>119</sup>

Omentalization has also largely replaced Penrose drainage or partial prostatectomy for the treatment of prostatic abscessation.<sup>120</sup> Long-term resolution of disease (>12 months) was achieved in 19 of 20 dogs treated with omentalization. One dog had a ruptured prostatic abscess before surgery but was successfully treated by combining prostatic omentalization with open abdominal drainage.<sup>120</sup>

Treatment of abscesses in other abdominal organs generally involves removal of the organ in question. Ovariohysterectomy is recommended in cases of pyometra; care should be taken to prevent leakage from the uterus during its removal. Liver lobe resection, nephrectomy, and splenectomy are recommended for abscessation of the liver, kidneys, and spleen, respectively. One study evaluating percutaneous drainage and alcoholization of hepatic abscesses in five dogs and one cat obtained favorable results in all six animals.<sup>121</sup>

Uroperitoneum presents a specific and urgent challenge because of potential life-threatening hyperkalemia. Every effort should be made to reestablish normal renal function and urine flow. In cases of bladder rupture, an indwelling urinary catheter and a peritoneal dialysis catheter are placed to drain urine from the bladder and peritoneal cavity, respectively. Serum potassium and calcium levels and acid-base status should be normalized, if possible, before the animal is anesthetized for laparotomy.

Diagnostic positive-contrast imaging of the urinary tract can generally locate the site of urine leakage before surgery. Unilateral ureteral ruptures are treated by primary repair or unilateral ureteronephrectomy, provided the contralateral kidney is functioning normally. Bladder ruptures are located and sutured with 3-0 or 4-0 polydioxanone suture in a simple interrupted pattern. Incomplete urethral tears may be managed with an indwelling urethral catheter for several days to weeks; complete urethral transections often require surgical repair.

Copious peritoneal lavage is an appropriate part of the standard of care for animals with septic peritonitis. The peritoneal cavity is viewed as a large, contaminated wound, and lavage with a large volume of warm, balanced electrolyte solution is used to remove bacteria and proinflammatory cytokines, GI contents, hemoglobin, mucus, and bile. However, peritoneal lavage is not entirely benign and may impair normal peritoneal defense mechanisms. Bacteria adhere to the peritoneal surfaces and may not be removed by low-pressure lavage. Lavage may disseminate bacteria, remove opsins and complement proteins necessary for phagocytes, prevent phagocytes from gaining access to bacteria, and damage peritoneal mesothelial cells.<sup>122</sup> Detailed, controlled clinical trials studying the efficacy of lavage in companion animals with septic peritonitis are lacking. The following recommendations are based on experimental and human clinical studies:

- A large volume of lavage fluid should be used (e.g., 500 mL to 1 L in a cat, several liters in large dogs).
- All of the lavage fluid must be aspirated from the peritoneal cavity.
- The addition of antibiotics to the lavage fluid has not proved beneficial, provided the animal is receiving appropriate doses of antibiotics parenterally.
- The addition of chlorhexidine to the lavage fluid has not proved beneficial. Experimentally, 0.05% chlorhexidine reduced bacterial numbers in residual lavage fluid and improved survival in a mouse peritonitis model,<sup>123</sup> but it is potentially toxic to normal cells.
- Povidone iodine should never be added to lavage fluid, as it has been associated with high mortality and sclerosing, encapsulating peritonitis.<sup>122</sup>

Early postoperative enteral nutrition appears to be beneficial in experimental peritonitis, but a distinct survival benefit has not been demonstrated. Potential benefits include increased anastomotic strength and more rapid healing, positive nitrogen balance, and decreased bacterial translocation through a compromised GI mucosal barrier.

In a rat peritonitis model, early postoperative enteral feeding resulted in a significantly higher nitrogen level, higher anastomotic bursting strength, and lower TNF- $\alpha$  lev-

els. TNF- $\alpha$  has the ability to decrease the synthesis of collagen and up-regulate collagenase activity, contributing to potential anastomotic breakdown and leakage.<sup>124</sup>

In another study,<sup>125</sup> mortality in human patients receiving early postoperative enteral nutrition via a jejunostomy tube was similar to that in a control group (19.1% and 18.2%, respectively). However, the patients in the study group had a positive nitrogen balance by the third postoperative day, whereas those in the control group had a negative nitrogen balance until the end of the study. Furthermore, the patients receiving early enteral nutrition had significantly fewer septic complications compared with the control group.

In one veterinary study,<sup>36</sup> 14 of 23 animals with peritonitis received nutritional support with either total parenteral nutrition or a feeding tube. Nine of these 14 patients survived. Although nutritional support was not associated with outcome in this study, this may have been due in part to small group numbers.

### Peritoneal Drainage

Once the underlying disease process has been treated, the peritoneal cavity lavaged, and the fluid aspirated, the surgeon must decide whether postoperative peritoneal drainage is required and, if so, whether the abdomen should be left open. There are no clear-cut, objective guidelines for this decision. Prevailing opinion views the peritoneal cavity in the same manner as any other contaminated wound: if substantial contamination remains despite the surgeon's best efforts, drainage options include open peritoneal drainage and the placement of a closed suction or sump drain.<sup>126,127</sup>

Advantages of open peritoneal drainage include effective removal of excess amounts of peritoneal fluid, easier access for reexploration of the peritoneal cavity, and the creation of an environment unfavorable to anaerobic bacteria.<sup>127</sup> Complications associated with open peritoneal drainage include evisceration of abdominal organs, formation of intestinal fistulae, visceral adhesion to the peritoneal bandage, nosocomial infection of the peritoneum, excessive fluid loss, and hypoproteinemia with secondary peripheral edema.<sup>21,22,128</sup> In humans, vacuum-assisted suction is commonly employed when open peritoneal drainage is used to prevent abdominal compartment syndrome.<sup>129</sup>

Closed-suction drainage is being used in patients with septic peritonitis with increasing frequency. A clinical study found it to be effective for treating generalized peritonitis, reporting no clinically significant complications.<sup>23</sup> Potential concerns with any intraabdominal drain include ineffective drainage secondary to omental or serosal walling off of the drain and nosocomial infection.

Sump-Penrose drainage has been compared with open drainage in normal dogs,<sup>130</sup> yielding no significant differences in blood chemistry, peritoneal fluid analysis, or necropsy findings. Nosocomial infections were cultured in

three cases (two dogs with sump-Penrose drainage and one with open peritoneal drainage).<sup>130</sup>

Several studies have reported results of primary closure without the use of abdominal drainage,<sup>24,25</sup> open drainage compared with primary closure,<sup>25,128</sup> and open peritoneal drainage.<sup>20-22,25</sup> It is difficult to compare results between studies because of the heterogeneity of the patient populations, the lack of a single standard for patient care, and variability in the surgeons' preference. None of the three treatment options (primary closure, open peritoneal drainage, or closed suction drainage) seems to have an obvious survival benefit based on the available clinical information in companion animals. The lack of statistically significant survival differences in studies that compare peritoneal drainage methods may be associated with small sample sizes but may also reflect the possibility that other factors (e.g., severity of sepsis, preoperative and postoperative management) are more important in determining survival.

### Prognosis

Mortality in 50 dogs with septic peritonitis was reported to be 68%, regardless of drainage technique.<sup>131</sup> A study of 51 cats with septic peritonitis<sup>35</sup> found a mortality of 30% in 23 cats that were treated by surgical exploration. Treatment was not pursued in the other 28 cats either because the cats died or were euthanized before treatment could be attempted, or because the extent of disease found during exploratory surgery was too great.<sup>35</sup>

Prognostic indicators in dogs and cats with septic peritonitis have been evaluated in two studies.<sup>22,36</sup> In one study,<sup>22</sup> survivors had significantly lower preoperative values for alanine aminotransferase and  $\gamma$ -glutamyl transferase than nonsurvivors. In the other study,<sup>36</sup> animals that experienced hypotension that was not correctable with treatment by IV fluids and those that developed respiratory dysfunction or DIC had a worse prognosis.

### Conclusion

The clinical syndrome of septic peritonitis presents a therapeutic challenge. Although prognoses vary, the potential for sepsis and its sequelae makes timely diagnosis and treatment essential. More study is needed to identify treatment modalities that can be directed at specific targets involved in the inflammatory cascade, but advances in human medicine are promising.

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3 CE  
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1. In dogs and cats, the bacteria that cause septic peritonitis most commonly originate from the
  - a. uterus.
  - b. bladder.
  - c. stomach/intestines.
  - d. liver.
  - e. spleen.
2. Which of the following does not directly contribute to the intrinsic defense system of the peritoneal cavity?
  - a. complement
  - b. neutrophils
  - c. macrophages
  - d. mast cells
  - e. fibrinogen
3. What substance is the primary contributor to the pathophysiology induced by gram-negative bacteria?
  - a. endotoxin
  - b. teichoic acid
  - c. peptidoglycan
  - d. polysaccharide/hyaluronic acid capsule
  - e. slime layer
4. The presence of \_\_\_\_\_ in a peritoneal effusion is diagnostic for septic peritonitis.
  - a. red blood cells
  - b. toxic neutrophils
  - c. mesothelial cells
  - d. macrophages
  - e. intracellular bacteria
5. When evaluated in a peritoneal effusion, \_\_\_\_\_ has not been found to be useful in the diagnosis of septic peritonitis in dogs.
  - a. glucose
  - b. lactate
  - c. total nucleated cell count
  - d. pH
  - e. cytology
6. The organism most commonly cultured from peritoneal effusions in cases of septic peritonitis is
  - a. *Streptococcus pyogenes*.
  - b. *Clostridium perfringens*.
  - c. *Escherichia coli*.
  - d. *Enterococcus faecalis*.
  - e. *Staphylococcus aureus*.
7. Which is not a benefit of corticosteroids in patients with septic peritonitis?
  - a. decreased vascular permeability
  - b. decreased negative effects of inflammatory cytokines such as IL-1 and TNF
  - c. promotion of production of antiinflammatory factors such as IL-10
  - d. treatment of adrenal insufficiency
  - e. promotion of wound healing
8. The options for surgical management of septic peritonitis do not include
  - a. omentalization.
  - b. serosal patching.
  - c. copious lavage with a balanced electrolyte solution.
  - d. removal of the nidus of infection.
  - e. lavage with iodine.
9. \_\_\_\_\_ is not a disadvantage of open peritoneal drainage.
  - a. Evisceration of abdominal organs
  - b. Nosocomial infection
  - c. Hypoproteinemia
  - d. Increased likelihood of anaerobic infection
  - e. Formation of intestinal fistulae
10. \_\_\_\_\_ has been associated with a negative outcome in cases of septic peritonitis.
  - a. Respiratory dysfunction
  - b. Open peritoneal drainage
  - c. Anemia
  - d. Hypertension
  - e. Peritoneal fluid lactate concentration >2 mmol/L