Primary Bacterial Septic Peritonitis in Cats: 13 Cases

The purpose of this paper is to describe the signalment, clinical signs, laboratory results, culture results, and response to treatment for primary septic peritonitis in cats. This is a retrospective study of 12 client-owned animals. Medical records were reviewed for clinical findings, laboratory results, microbial culture results, radiographic findings, diagnosis, treatment, and outcome. The overall mortality rate for this group of cats was 31%, consistent with previous reports of septic peritonitis in cats. All cats that were both bradycardic and hypothermic on presentation did not survive. Other clinicopathological findings were consistent with previously reported cases of septic peritonitis in cats. Results suggest that clinicopathological findings and outcomes in cats with primary septic peritonitis are similar to those in cats with septic peritonitis from a determined cause. A specific mechanism of inoculation has yet to be determined, but an oral source of bacteria is suggested for cats with primary bacterial septic peritonitis. **J Am Anim Hosp Assoc 2009;45:268-276.**

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Introduction

Primary septic peritonitis is defined as a septic peritonitis without an evident intraabdominal focus of infection.[1] Primary septic peritonitis is a rare condition in animals. Typically, animals that have a primary cause for septic peritonitis have a more chronic illness than animals that have a secondary cause for septic peritonitis.[2] In cats, the most common cause of primary septic peritonitis is infection with feline coronavirus, causing feline infectious peritonitis.[2] Less is known about primary bacterial septic peritonitis in cats. Previous studies have mentioned cases of primary bacterial septic peritonitis, but none have looked at these cases individually.[3,4]

The purposes of this paper are to describe a group of cats in which no apparent cause for septic peritonitis was identified and to determine if there are any significant characteristics that are different for this group of cats compared to previously reported cats with septic peritonitis from an intraabdominal cause of infection.

Materials and Methods

Medical records for cats diagnosed with primary septic peritonitis between 1999 and 2006 at Veterinary Surgical Associates were reviewed. Cats diagnosed with feline coronavirus infection or feline infectious peritonitis using histopathology were excluded.

The medical records for each cat were reviewed, and information was obtained regarding signalment, indoor-outdoor status, physical examination abnormalities, serum biochemical profile findings, radiological findings, abdominal fluid cytological findings, microbial culture results, treatments administered, and outcome. In all cases, a diagnosis of septic peritonitis was based on results of abdominal fluid cytology and culture and/or gross surgical findings. All surgeries were performed by a diplomate of the American College of Veterinary Surgeons. In all cases, the surgeon involved found no evidence of an intraabdominal source of infection.

Results

Twelve cats with primary septic peritonitis met the inclusion criteria for this study. Because one cat had two episodes of primary septic peritonitis 7 months apart, there were 13 episodes in 12 cats. Signalment information is presented in Table 1. Eight (67%) were castrated males, and four (33%) were spayed females. Eight (67%) were domestic shorthair, three (25%) were domestic longhair, and one (8%) was a Burmese.

Physiological data is presented in Table 1. In four (31%) of the cases, the cat had a heart rate <140 beats per minute. The respiratory rate was >40 breaths per minute in nine (69%) cases. In five (38%) of the cases, the cat had a temperature >102.5˚F, and in three (23%) cases, the cat had a temperature <98˚F. In four (31%) of the 13 cases, the cat had abdominal pain on physical examination. In eight (62%) cases, the cat had palpable abdominal fluid. One (8%) cat had a grade III/VI systolic murmur. No other abnormal physical examination findings were recorded.

Results of complete blood count (CBC) and biochemical findings are listed in Table 2. A CBC or quantitative blood count was available for nine of the cats out of 13 cases. Band neutrophils were reported in four of the cats, and the median band neutrophil count was 3614 cells/μL (range 258 to 13,692 cells/μL). In four of the cases, toxic white blood cell changes were seen, designated as moderate in three cats and mild in one. Anemia was identified in six of the cats in the 13 cases. In all cats with anemia, the anemia was characterized as normocytic, normochromic, and nonregenerative.

Serum albumin was measured in nine of the cats out of 13 cases, and hypoalbuminemia was identified in all nine. Blood glucose concentration was measured in 10 cats out of 13 cases; hyperglycemia was identified in one cat, and hypoglycemia was identified in one cat. Serum calcium concentration was measured in nine of the cats out of 13 cases, and hypocalcemia was identified in four cats. Total bilirubin concentration was measured in nine cats out of 13 cases, and hyperbilirubinemia was identified in four cats. No elevations were seen in either serum alanine transaminase (ALT) or serum alkaline phosphatase (ALKP) in the cats in which they were measured. Blood urea nitrogen was elevated in two cats out of nine cases in which it was measured, and serum creatinine was normal in all nine cats in which it was measured. Hyperphosphatemia was identified in one cat out of the nine cases in which it was measured. Serum potassium, chloride, and sodium were evaluated in eight cats out of 13 cases. Hyperkalemia was identified in three cats, and hypokalemia was identified in one cat. Serum chloride concentration was normal in all cats in which it was measured. Hyponatremia was identified in three cats.

Abdominal radiographs were obtained in seven out of 13 cases. Diffuse loss of serosal detail, consistent with abdominal effusion, was identified in every radiograph. No other significant abnormalities were identified. Thoracic radiographs were performed in four out of 13 cases, and no abnormalities were identified in any cat. Abdominal ultrasound was performed in two cats, and abdominal effusion (n=2), thickened mesentery (n=1), and a bunched intestinal tract (n=1) were the identified abnormalities.

Peritoneal effusion was evaluated in 11 cats out of 13 cases. Degenerate neutrophils and a mixed population of intracellular bacteria were identified in every cat. Culture and susceptibility test results were available for nine cats out of 13 cases. No culture results were available for the cats that did not survive. Organisms identified are presented in Table 3. Polymicrobial infection was identified in eight (89%) cats. In the only cat of a pure culture, a *Bacteroides* spp. was isolated.

All cats had exploratory celiotomy after stabilization and diagnosis of probable septic peritonitis. Surgical findings in all cats were consistent with diffuse peritonitis. The peritoneal fluid in all cats was foul smelling and yellow to tan in color, with yellow particulate matter. A partial omentectomy was performed in three (23%) cats. Biopsies were obtained in seven (54%) cats out of 13 cases. Various organs were biopsied, including the liver (n=4), omentum (n=4), mesenteric lymph nodes (n=3), stomach and jejunum (n=2), pancreas (n=1), and body wall (n=1). All biopsy samples contained inflammatory changes consistent with peritonitis. Cats were either closed primarily or closed over a drain, or they were treated as an open abdomen for a period of time. These data are presented in Table 1. One of the cats treated with open peritoneal drainage was treated by closure over an intraperitoneal closed-suction drain at the time of the second surgical procedure.

Median time of hospitalization was 4.5 days (range 2 to 8 days). Eight of 12 cats survived their episodes of septic peritonitis. One of these eight cats had recurrence 7 months after the first episode and again survived. Survival information is presented in Table 1. One cat survived 5 days postoperatively, then acutely suffered cardiopulmonary arrest. Three cats died within 24 hours after surgery. None of the cats were euthanized.

In three (23%) cats out of 13 cases, dopamine was administered to address hypotension, and two of these cats survived. Two (15%) cats required a blood transfusion, and one of the two survived. Two (15%) cats required fresh-frozen plasma, and both cats survived. Seven (54%) cats had a gastrostomy feeding tube placed at the time of surgery, and one (7.7%) had an esophagostomy feeding tube placed. The cat with the esophagostomy tube arrested and died 1 day postoperatively. Three of the cats with gastrostomy tubes died. One (7.7%) cat that did not have a feeding tube placed required partial parenteral nutrition and survived.

All cats received routine postoperative care including intravenous fluids, analgesics, and antimicrobials. Intravenous fluid composition, rate, and total volume infused were determined by clinician discretion and need of the cat. Analgesics selected were either buprenorphine or fentanyl as a constant-rate infusion based on clinician discretion. A variety of antimicrobials were used in the treatment of these cats. Antimicrobial selection is presented in Table 3. Polyantimicrobial therapy was used in all cases.

Table 1

Comparisons of Physical Examination Findings, Culture Results, Surgical Closure Method, and Outcomes in 13 Cases of Primary Septic Peritonitis in Cats Negative for Feline Infectious Peritonitis

Case No.	Signalment*	Heart Rate	Respiratory Rate	Body Temperature	Indoor/Outdoor Status	Surgical Closure	Outcome
1	8-y-old, NM DSH	132	42	101.4	Indoor/Outdoor	Closed	Deceased
2	11-y-old, NM DSH	152	40	103.0	Indoor/Outdoor	Open initially and closed 2 d later	Survived
3	5-y-old, NM DSH	160	40	103.3	Indoor	Open initially and closed 3 d later over drain	Survived
4	13-y-old, SF DSH	100	40	94.7	Indoor	Open	Deceased
5	5-y-old, SF DSH	188	60	101.0	Indoor/Outdoor	Closed	Survived
6	9-y-old, NM Burmese	160	56	102.4	Indoor/Outdoor	Closed	Survived
7	12-y-old, SF DSH	200	32	102.5	Indoor	Closed	Survived
8	8-y-old, NM DLH	200	50	104.0	Indoor/Outdoor	Closed	Survived
9	8-y-old, NM DSH	100	68	92.0	Indoor/Outdoor	Open	Deceased
10	9-y-old, NM DLH	100	36	95.0	Indoor/Outdoor	Closed	Deceased
11	10-y-old, NM DLH	180	36	101.9	Outdoor	Closed	Survived
12	3-y-old, SF DSH	162	42	104.7	Indoor/Outdoor	Open initially and closed 3 d later	Survived
12†	3-y-old, SF DSH	180	36	102.9	Indoor/Outdoor	Closed over drain	Survived

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Table 1 (cont'd)

Comparisons of Physical Examination Findings, Culture Results, Surgical Closure Method, and Outcomes in 13 Cases of Primary Septic Peritonitis in Cats Negative for Feline Infectious Peritonitis

Case No.	Signalment*	Heart Rate	Respiratory Rate	Body Temperature	Indoor/Outdoor Status	Surgical Closure	Outcome
Mean	8.4 y	153	44	100.8			
Median	8.5 y	160	40	102.4			
Standard deviation	2.97 y	34.6	10.0	4.04			

* NM=neutered male; DSH=domestic shorthair; SF=spayed female; DLH=domestic longhair
† These are the results of the second episode for case no. 12.

Table 2

Results of Complete Blood Cell Count and Serum Biochemical Profiles for Cats With Primary Septic Peritonitis

Case No.	WBC* (thous/L)	PCV†	Albumin (g/dL)	Glucose (mg/dL)	Calcium (mg/dL)	Bilirubin (mg/dL)	ALT‡ (U/L)	ALKP§ (U/L)	BUN\ (mg/dL)	Creatinine (mg/dL)	Phosphorus (mg/dL)	Potassium (mmol/L)	Sodium (mmol/L)	Chloride (mmol/L)
1		25 (30-45)	1.57 (2.6-3.9)	55 (76-145)		1.46 (0-0.5)						2.69 (3.5-5.8)	136 (150-165)	109 (112-129)
2	20.0 (5.0-18.9)	15 (30-45)	2.14 (2.6-3.9)	155.7 (76-145)	7.87 (7.8-11.3)	1.63 (0-0.5)	<10 (12-130)	19 (14-111)	21.3 (16-36)	0.97 (0.8-2.4)	6.24 (3.1-7.5)	5.02 (3.5-5.8)	155.5 (150-165)	121.2 (112-129)
3	45.8 (3.5-16.8)	22 (30-45)	2.1 (2.5-3.9)	74 (64-170)	7.3 (8.2-10.8)	0.1 (0.1-0.4)	21 (10-100)	2 (1-100)	34 (14-36)	0.9 (0.6-2.4)	6.7 (2.4-8.2)	6.1 (3.4-5.6)	145 (145-158)	112 (104-128)
4	32.7 (5.0-18.9)	23 (30-45)	1.79 (2.6-3.9)	99.9 (76-145)	8.0 (7.8-11.3)	0.17 (0-0.9)	<10 (12-130)	24 (14-111)	24.6 (16-36)	0.75 (0.8-2.4)	4.74 (3.1-7.5)			
5														
6	26.6 (5.0-18.9)	31 (30-45)	2.27 (2.6-3.9)	100.1 (76-145)	7.78 (7.8-11.3)	1.09 (0-0.9)	24 (12-130)	19 (14-111)	59.3 (16-36)	0.99 (0.8-2.4)	5.8 (3.1-7.5)	7.97 (3.5-5.8)	155.8 (150-165)	122.5 (112-129)
7	15.8 (3.5-16.0)	26 (30-45)	2.7 (2.5-3.9)	125 (64-170)	8.8 (8.2-10.8)	0.1 (0.1-0.4)	7 (10-100)	10 (6-102)	20 (14-36)	1.3 (0.6-2.4)	4.8 (2.4-8.2)	4.4 (3.4-5.6)	151 (145-158)	116 (104-128)
8	72.3 (5.0-18.9)		2.55 (2.6-3.9)			0.74 (0.1-0.4)								
9														
10														
11	43.2 (4.2-15.6)	30 (30-45)	2.1 (2.3-3.9)	97 (70-150)	8.0 (8.2-11.8)	0.1 (0-0.4)	8 (28-100)	10 (0-62)	31 (15-34)	0.9 (0.8-2.3)	5.2 (3.0-7.0)	6.7 (3.9-5.3)	144 (147-156)	114 (111-125)
12	8.6 (5.5-19.5)	27 (30-45)	1.72 (2.2-3.9)	91 (70-150)	8.9 (7.5-10.8)	0.8 (0-0.4)			32 (15-34)	0.9 (0.8-2.3)	4.8 (3.0-7.0)	4.1 (3.7-5.9)	145 (150-165)	116 (115-126)
12 second episode	26.5 (4.2-15.6)	33 (30-45)	2.1 (2.3-3.3)	89 (70-150)	9.3 (8.2-11.8)	0.2 (0-0.4)	7 (28-76)	6 (0-62)	47 (15-34)	1.1 (0.8-2.3)	7.1 (3.0-7.0)	4.2 (3.9-5.3)	141 (147-156)	111 (111-125)

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Table 2 (cont'd)

Results of Complete Blood Cell Count and Serum Biochemical Profiles for Cats With Primary Septic Peritonitis

Case No.	WBC* (thous/L)	PCV†	Albumin (g/dL)	Glucose (mg/dL)	Calcium (mg/dL)	Bilirubin (mg/dL)	ALT‡ (U/L)	ALKP§ (U/L)	BUN\ (mg/dL)	Creatinine (mg/dL)	Phosphorus (mg/dL)	Potassium (mmol/L)	Sodium (mmol/L)	Chloride (mmol/L)
Mean	32.38	25.7	2.1	98.5	8.24	0.64	13.4*	12.86	33.65	0.98	5.67	5.15	146.66	115.2
Median	26.6	26	2.1	97	8.0	0.47	8*	10	31.5	0.94	5.5	4.71	145	115
Standard deviation	18.11	5.14	0.33	27.12	0.63	0.63	8.39*	7.38	12.58	0.17	0.87	1.58	6.51	4.45

Note: Normal values are listed in parentheses.
* WBC=white blood cell count
† PCV=packed cell volume
‡ ALT=alanine transaminase
§ ALKP=alkaline phosphatase
\ BUN=blood urea nitrogen
¶ Values reported are excluding the two values listed as <10, since no specific numerical value was recorded.

Table 3

Antibiotic Therapy Administered to Cats With Primary Septic Peritonitis

Case No.	Culture Results	Initial Antibiotic Selection	Antibiotics Sent Home
1	Unknown	Enrofloxacin, Ampicillin	
2	*Actinomyces*, *Bacteroides*	Enrofloxacin, Ampicillin	Amoxicillin/Clavulanic acid, Enrofloxacin
3	*Bacteroides*	Enrofloxacin, Ampicillin	Amoxicillin/Clavulanic acid, Enrofloxacin
4	Unknown	Enrofloxacin, Ampicillin	
5	*Bacteroides*, *Fusobacterium*	Ciprofloxacin, Amoxicillin; then added Chloramphenicol; then switched to Penicillin G and Trimethoprim Sulfa	Clindamycin, Trimethoprim Sulfa
6	*Bacteroides*, *Fusobacterium*	Enrofloxcin, Ampicillin	Clindamycin, Trimethoprim Sulfa
7	*Bacteroides*, *Fusobacterium*	Ampicillin; then switched to Timentin, Enrofloxacin	Amoxicillin/Clavulanic acid, Trimethoprim Sulfa
8	*Fusobacterium*, *Peptostreptococcus*	Enrofloxacin, Ampicillin	Amoxicillin/Clavulanic acid, Enrofloxacin
9	Unknown	Enrofloxacin, Cefazolin, Metronidazole; then switched to Timentin	
10	Unknown	Enrofloxacin, Cefazolin	
11	*Bacteroides*, *Fusobacterium*	Enrofloxacin, Cefazolin	Amoxicillin/Clavulanic acid
12	*Actinomyces*, *Bacteroides*	Enrofloxacin, Ampicillin	Amoxicillin/Clavulanic acid
12 second episode	*Morganella morganii*, *Fusobacterium*, *Peptostreptococcus*	Enrofloxacin, Ampicillin	Amoxicillin/Clavulanic acid

The cats were discharged with antimicrobial therapy for at least 1 month. Three (23%) cats were discharged with only amoxicillin and clavulanic acid for antimicrobial therapy, and the rest received polyantimicrobial therapy.

No apparent differences were seen in the serum biochemical values between cats that did and did not survive. Two (28.5%) of the seven cats that were treated with a closed abdomen and two (40%) of the five cats that were treated with open peritoneal drainage died. All cats that were hypothermic (<98˚F) and had a heart rate of <140 beats per minute on presentation did not survive.

Discussion

The results of this study indicate that the clinical abnormalities in cats with primary septic peritonitis include tachypnea (respiratory rate >44 breaths per minute), bradycardia (<140 beats per minute), abdominal effusion, hypoalbuminemia, and anemia. Thirty to forty percent of cases had a fever (temperature >102.5˚F), abdominal pain, a band neutrophilia, and hyperbilirubinemia. Total calcium concentration did trend with albumin levels in these cats. Cats with hypoalbuminemia are thought to possibly have hypocalcemia resulting from a decrease in the protein-bound fraction of calcium;[5]

however, a dependable formula to adjust for this phenomenon in cats has not been identified.[6] These findings are consistent with previous studies regarding severe sepsis in cats.[2,7] Ionized calcium values may be a more accurate representation of the available calcium stores for cats, but they were not evaluated in any of these cats.

All of the bacterial isolates were either nonspore-forming obligate anaerobes or facultative gram-negative anaerobes, and they are common in the alimentary tract.[8] Nonspore-forming obligate anaerobes comprise 33% of all positive cultures from pyonecrotic material in animals.[8] Disease caused by these bacteria is usually a result of extension of normal gastrointestinal flora into a compromised site or direct inoculation into tissue.[8] The ability to establish an obligate anaerobic infection is based on the creation of anaerobic conditions by trauma, vascular compromise, or concurrent infection with facultative anaerobes.[8] A pure culture was found in one case in this study, despite seeing a mixed population of bacteria cytologically. This cat was given systemic antibiotics prior to culture sampling, which may have altered the results.

All of the bacteria cultured in this study, except *Morganella morganii*, are common isolates of the feline gingival margin.[9] The two most common isolates were *Bacteroides* and *Fusobacterium*, consistent with results of anaerobic bacterial cultures obtained from pyothorax and subcutaneous abscesses in cats.[10-12] This supports the fact that these infections are potentially the result of spread from the oral cavity either through direct penetration or a hemotogenous route. Importantly, these bacteria are commonly isolated from the oral cavities of normal cats and not from cats with gingival disease.[9,11] Severe dental disease was not identified in any of the cats in this study and does not appear to be a contributing factor. Another possible mechanism of infection is a migrating foxtail because of the high prevalence in this geographical area; however, most infections from migrating plant awns culture positive for *Streptococcus* and *Staphylococcus* species, and plant material was not identified in any case of this study.[13]

Initial empirical antibiotic selections were made in these cases to provide broad-spectrum coverage prior to receipt of culture and sensitivity results. A large proportion of cats were initially treated with enrofloxacin, a drug that does not have a significant anaerobic spectrum, in combination with various other antibiotics to broaden coverage. Given the fact that all effusions cultured ultimately yielded only anaerobes, it would be prudent to ensure that appropriate anaerobic antibiotics are initiated as soon as possible in cats suspected to have primary septic peritonitis. The addition of antibiotics that are efficacious against aerobic organisms may not be necessary.

Three cats had a partial omentectomy performed at the time of surgery. Surgical reports did not detail why omentectomy was performed in these cases; however, the assumption is that it was done to address suspected omental necrosis based on gross appearance, or it was for culture and biopsy samples.

In the present study, 69% of the cases with surgical and medical treatment of primary septic peritonitis survived; this percentage is consistent with the published survival rates in cats with septic peritonitis.[3,4] The decision regarding the method of closure was surgeon dependent, and the operative reports did not indicate reasons a particular method was chosen. Two (28.5%) out of seven of the cats that were treated with primary closure did not survive, and two (40%) out of five of the cats treated with open peritoneal drainage did not survive. Previous reports indicate no apparent difference in survival rates between cats undergoing lavage and primary closure, closure over a drain, or open peritoneal drainage.[3,14,15] Because of the low number of cases in our study, significant differences in survival rates based on method of closure could not be established. However, a trend was seen toward increased survival for cats that underwent primary closure versus open peritoneal drainage. Only one case was originally closed over an intraperitoneal drain, and one case was closed over an intraperitoneal drain several days later, making it difficult to comment on the efficacy of that method because of the small number of cases.

In this study, the combination of hypothermia and bradycardia on presentation appeared to be a negative prognostic indicator. Previous studies regarding sepsis in cats do not support this finding.[4] This may be a result of the relatively small number of cases in this study, or it may be a unique finding with primary septic peritonitis. Hypothermia has been associated with a worse outcome in both humans and experimental animal models of sepsis.[16] Bradycardia has been previously reported as a unique response to the hypodynamic phase of sepsis in cats.[3,4] A report links hypothermia to the development of mild bradycardia in humans.[17] Several mechanisms have been proposed for this bradycardia, including alteration in beta adrenergic receptor function, a response to a humoral factor(s) produced by the invading organism or host, and simultaneous baroreceptor stimulation of vagal and sympathetic fibers.[3,4,18,19] An association between elevated ALT activity and a higher mortality rate was made in a previous study; however, the difference in median ALT activities between survivors and nonsurvivors was not clinically relevant.[4] Increased ALT activity was not a negative prognostic indicator in this study, and serum ALT was not elevated in any of the cats in which it was measured.

Several difficulties were encountered because of the retrospective nature of this study. None of the cats that died had culture or histopathology results, and the diagnosis of primary bacterial septic peritonitis could not be confirmed. These cats were included because of their similarities with survivors in regard to surgical findings and the presence of bacteria in their abdominal effusion when it was cytologically evaluated. Also, determining if these cats had feline infectious peritonitis was not possible. Histopathology results for seven of the nine survivors were not consistent with feline infectious peritonitis. The other two surviving cats were negative for feline coronavirus infection on serological testing.

Statistical analysis was not performed in this study because of the small sample size.

Based on our results, cats with primary bacterial septic peritonitis have a fair to good prognosis for survival if they are treated appropriately prior to signs of hemodynamic shock (i.e., hypothermia and bradycardia). Treatment should include exploratory celiotomy with copious peritoneal lavage, nutritional support as indicated, and appropriate antimicrobial therapy based on aerobic and anaerobic culture and sensitivity results. Empirical antibiotic therapy with a good anaerobic spectrum should be started prior to the receipt of final culture results if primary septic peritonitis is strongly suspected. A definitive recommendation regarding abdominal cavity closure cannot be made based on our results, because the population size was small. However, an argument could be made to recommend primary closure following lavage because of theoretically decreased case morbidity when compared to management with open peritoneal drainage or closure over an intraperitoneal drain.[17] Also, in this study a trend toward a higher survival rate was seen in the cats treated with primary closure.

Conclusion

Results suggest that clinicopathological findings and outcomes in cats with primary septic peritonitis are similar to those in cats with septic peritonitis from a determined cause. The combination of bradycardia and hypothermia appears to be a negative prognostic indicator for this group of cats. A specific mechanism of inoculation has yet to be determined, but an oral source of anaerobic bacteria is suggested for cats with primary bacterial septic peritonitis.

References

1. Bjorling DE, Crowe DT. Peritonitis. In: Sherding RG, ed. The Cat: Disease and Clinical Management. Philadelphia: WB Saunders Co., 1994:1403-1506.
2. Kahn CM. Peritonitis. In: Kahn CM, ed. The Merck Veterinary Manual. Whitehouse Station, NJ: Merck & Co., Inc., 2005:537-543.
3. Brady CA, Otto CM, Van Winkle TJ, *et al*. Severe sepsis in cats: 29 cases (1986-1998). J Am Vet Med Assoc 2000;217:531-535.
4. Costello MF, Drobatz KJ, Aronson LR, *et al*. Underlying cause, pathophysiologic abnormalities, and response to treatment in cats with septic peritonitis: 51 cases (1990-2001). J Am Vet Med Assoc 2004;225:897-902.
5. Peterson ME, Randolph JF, Mooney CT. Endocrine diseases. In: Sherding RG, ed. The Cat: Disease and Clinical Management. Philadelphia: WB Saunders Co., 1994:1403-1506.
6. Flaunders JA, Scarlett JM, Blue JT, *et al*. Adjustment of total serum calcium concentration for binding albumin and protein in cats: 291 cases (1986-1987). J Am Vet Med Assoc 1989;194:1609-1611.
7. Wright KN, Gompf RE, DeNovo RC. Peritoneal effusion in cats: 65 cases (1981-1997). J Am Vet Med Assoc 1999;214:375-381.
8. Hirsh DC. Non-spore-forming obligate anaerobes of the alimentary tract. In: Hirsh DC, Zee YC, eds. Veterinary Microbiology. Malden, MA: Blackwell Science, Inc., 1999:83-85.
9. Love DN, Vekselstein R, Collings S. The obligate and facultatively anaerobic bacterial flora of the normal feline gingival margin. Vet Micro 1990;22:267-275.
10. Barrs VR, Allan GS, Beatty JA, *et al*. Feline pyothorax: a retrospective study of 27 cases in Australia. J Fel Med and Surg 2005;7:211-222.
11. Love DN, Johnson JL, Moore LVH. *Bacteroides* species from the oral cavity and oral-associated diseases of cats. Vet Micro 1989;19:275-281.
12. Walker AL, Jang SS, Hirsh DC. Bacteria associated with pyothorax of dogs and cats: 98 cases (1989-1998). J Am Vet Med Assoc 2000;216:359-363.
13. Brennan KE, Ihrke PG. Grass awn migration in dogs and cats: a retrospective study of 182 cases. J Am Vet Med Assoc 1983;182:1201-1204.
14. Mueller MG, Ludwig LL, Barton LJ. Use of closed-suction drains to treat generalized peritonitis in dogs and cats: 40 cases (1997-1999). J Am Vet Med Assoc 2001;219:789-794.
15. Staatz AJ, Monnet E, Seim HB. Open peritoneal drainage versus primary closure for the treatment of septic peritonitis in dogs and cats: 42 cases (1993-1999). Vet Surg 2002;31:174-180.
16. Remick DG, Xioa H. Hypothermia and sepsis. Front Biosci 2006;11:1006-1013.
17. Drake CE, Flowers NC. ECG changes in hypothermia from sepsis and unrelated to exposure. Chest 1980;77:685-686.
18. Buhimschi C, Weiner CP. Endotoxemia causing fetal bradycardia during urosepsis. Obstet Gynecol 2001;97:818-820.
19. Tobias JD, Bozeman PM, Stokes DC. Postsepsis bradycardia in children with leukemia. Crit Care Med 1991;19:1172-1176.