

peer reviewed

peer reviewed

Pyothorax in a cat managed by intrathoracic debridement and postoperative ventilatory support

Ronan S. Doyle, Christopher R. Bellenger, Luis Campoy and Hester McAllister

Department of Veterinary Surgery, Faculty of Veterinary Medicine, University College Dublin, Belfield, Dublin 4, Ireland.

A domestic-longhair cat presented due to lethargy, dyspnoea and hypersalivation. Radiographic examination revealed a bilateral pleural effusion, which was diagnosed as pyothorax based on cytological examination. Ultrasonographic examination revealed extensive loculations within the thoracic cavity. Exploratory sternotomy, under general anaesthesia, allowed the removal of approximately 100ml of purulent fluid and debridement of a partially walled-off abscess and necrotic material from the pleural cavity. Postoperative positive-pressure ventilation was required due to severe respiratory depression. Intensive postoperative care, including intensive continuous monitoring, thoracostomy tube drainage and lavage of the pleural cavity and oesophagostomy tube feeding, was performed. Complete resolution of clinical signs had occurred by 15 days postoperatively. Clinical or radiographic abnormalities were not detected at a follow-up examination one year after surgery.

Irish Veterinary Journal
Volume 58: 211-215, 2005

Introduction

Pyothorax is the accumulation of a septic inflammatory effusion within the pleural cavity and is a serious life-threatening condition in the cat (Jonas, 1982; Fooshee, 1988). Common clinical signs include inappetance or anorexia, lethargy, dyspnoea, weight loss, pyrexia, hypersalivation and muffled heart sounds (Waddell *et al.*, 2002; Demetriou *et al.*, 2002). The source of the bacterial contamination of the pleural cavity is unknown in most cases (Waddell *et al.*, 2002; Demetriou *et al.*, 2002); however, thoracic bite wounds are frequently implicated in the cat (Monnet, 2003). Diagnosis is usually straightforward, being based on clinical signs, thoracic radiography, thoracocentesis and cytology (Holmberg, 1979; Jonas, 1982; Fooshee, 1988; Waddell *et al.*, 2002; Demetriou *et al.*, 2002). This case report describes a cat with advanced pyothorax treated with aggressive medical and surgical management, which required the use of postoperative ventilatory support to achieve a successful result.

Case details

A two-year-old 2.7kg outdoor-indoor female neutered domestic-longhair cat from a multi-cat household presented to the University Veterinary Hospital, University College Dublin, with a history of lethargy, weakness and laboured breathing of 12 hours duration. The cat was reported to be inappetent and to have lost weight over the previous four to five days. Amoxycillin-clavulanate (Synulox; Pfizer, 50mg) had been administered orally 12 hours before presentation. There was no history of recent trauma. The cat had been in the owner's possession since three months of age and had been vaccinated annually against feline panleukopenia, feline calicivirus and feline herpesvirus.

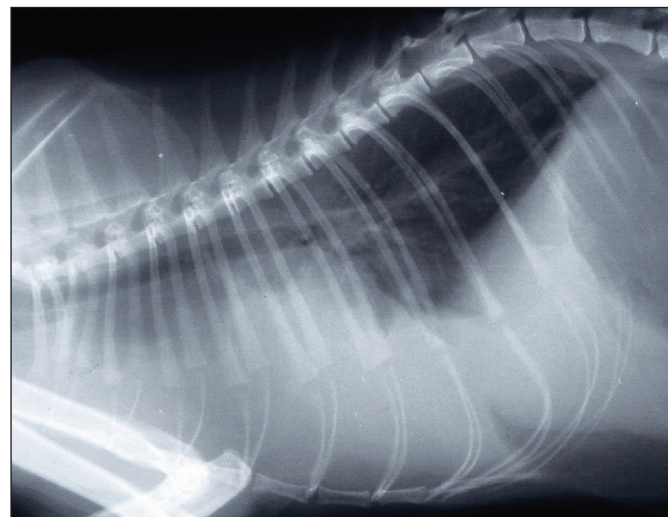


Figure 1: Right lateral thoracic radiograph pre-surgery.

Correspondence

Ronan Doyle

Department of Veterinary Surgery
Faculty of Veterinary Medicine
University College Dublin
Belfield, Dublin 4, Ireland
Tel: 01 716 6064
Fax: 01 716 6061
E-mail: ronan.doyle@ucd.ie

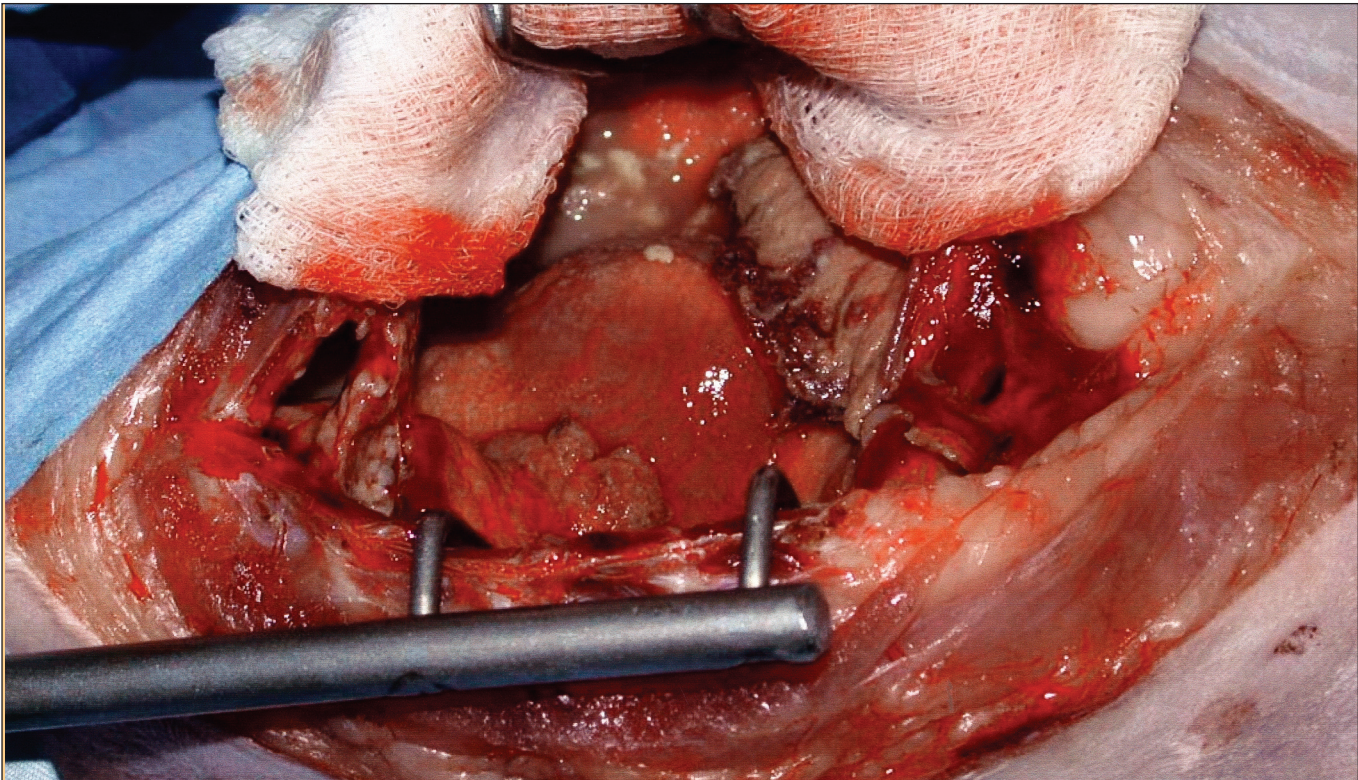


Figure 2: Intraoperative photograph of exploratory sternotomy of thoracic cavity.

On clinical examination the cat was dull, depressed and poorly responsive. It was estimated to be 10% dehydrated, with reasonable body condition. It was tachycardic with a weak peripheral pulse and cold extremities. The peripheral lymph nodes were normal. Hypersalivation, tachypnoea, and an inspiratory dyspnoea were observed. On thoracic auscultation lung sounds were decreased bilaterally. The rectal temperature was 38.3°C.

An intravenous catheter was placed in the left cephalic vein and lactated Ringer's solution (Compound Sodium Lactate; Ivex Pharmaceuticals) was administered at a rate of 40ml/kg/h. Thoracic radiographs (Figure 1, p211) were taken without sedation and revealed a fluid/soft tissue opacity in the ventral thorax obscuring the cardiac silhouette and ventral diaphragmatic margin. The lungs were separated from the thoracic wall at the costodiaphragmatic recess and right hemithorax, indicating the presence of a pleural effusion. Ultrasonographic examination of the thoracic cavity confirmed the presence of a pleural effusion. This was highly echogenic with large amounts of flocculent material oscillating within it. The thoracic cavity had a loculated appearance, particularly in the ventral caudal region.

Ultrasound-guided thoracocentesis was performed using a 5/8 inch 21-gauge needle at the costochondral junction of the right seventh intercostal space. Only seven millilitres of highly viscous purulent fluid was aspirated from the pleural cavity.

Cytological examination of the fluid revealed large amounts of necrotic debris with few intact inflammatory cells and massive numbers of mixed bacteria. Large numbers of Gram-positive filamentous organisms and Gram-negative organisms were present on Gram-stained smears. Subsequent bacteriological culture identified *Pasteurella multocida* and an *Actinomyces* spp. The cat was administered amoxicillin-clavulanate (Augmentin; GlaxoSmithKline: 25mg/kg) intravenously and enrofloxacin (Baytril; Bayer: 5mg/kg) subcutaneously. The *Pasteurella* and *Actinomyces* spp. were sensitive to both these antibiotics on subsequent antimicrobial sensitivity testing.

Haematological examination revealed a leukopenia [total white blood cell count $5.7 \times 10^9/L$; reference range 8 to $20 \times 10^9/L$], which was characterised by a severe mature neutropenia [$0.7 \times 10^9/L$; reference range 2.8 to $15 \times 10^9/L$], a moderate immature neutrophilia [$3.7 \times 10^9/L$; reference range 0 to $0.7 \times 10^9/L$] and a severe lymphopenia [$0.1 \times 10^9/L$; reference range 0.2 to $2 \times 10^9/L$]. Serum biochemical analysis revealed hypoproteinaemia [41.1g/L; reference range 59 to 68g/L] with hypoalbuminaemia [14.1g/L; reference range 23 to 35g/L]. There was a marked elevation in creatine kinase [423 U/L; reference range 10 to 70U/L], severe hyperchloraemia [118 mmol/L; reference range 99 to 110mmol/L] and severe hypoglycaemia [1.7mmol/L; reference range 3.3 to 5mmol/L]. The intravenous rate 40ml/kg/h of lactated Ringer's solution was continued and an 18-gauge central line (Leaderflex; Vygon)

peer reviewed

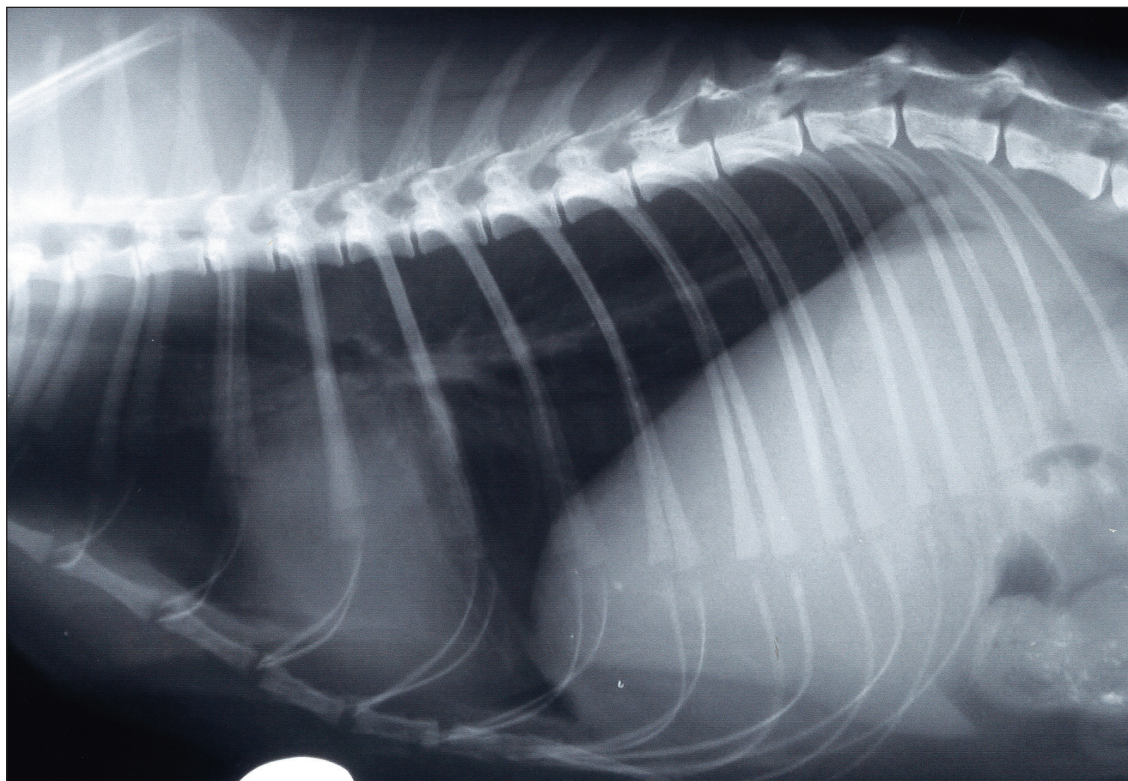


Figure 3: Right lateral thoracic radiograph 15 days post-surgery.

was placed under local anaesthesia in the right jugular vein to monitor central venous pressure and the response to fluid therapy. Pre-oxygenation via facemask was carried out prior to the administration of intravenous midazolam (Hypnovel; Roche) at 0.3mg/kg and ketamine (Vetalar; Pharmacia) 4.8mg/kg for premedication and induction of anaesthesia, respectively. After endotracheal intubation, ketamine at 30µg/kg/minute was administered on a continuous intravenous infusion supplemented with isoflurane (Forane; Abbott) at an end-tidal 0.2% in 100% oxygen. Systolic blood pressure was too low to be measured; therefore, intravenous infusions of dopamine hydrochloride (Dopamine Hydrochloride Solution BP; Antigen Pharmaceuticals: 5µg/kg/minute) and colloids (Gelofuscine; Braun: 20ml/kg) were commenced. Morphine sulphate (Morphine Sulphate Injection BP; Antigen Pharmaceuticals: 0.2mg/kg) was administered intravenously for intraoperative analgesia. Ventilation was maintained mechanically throughout anaesthesia and surgery. Forty minutes after induction of anaesthesia, venous blood gas analysis (i-Stat analyser; i-Stat Corporation) revealed a severe metabolic acidosis and moderate respiratory acidosis [pH 7.064, normal venous pH 7.376]. An intravenous bicarbonate infusion (8.4% Sodium Bicarbonate; Braun) was started (1mmol over 30 minutes and 3mmol over the next two hours) to improve the metabolic acidosis. In addition, tidal volume was increased to decrease end-tidal carbon dioxide and compensate for the excess of carbon dioxide

produced by the bicarbonate infusion.

An oesophagostomy tube was inserted on the left side of the neck. A ventral sternotomy was commenced caudal to the xiphisternum, extending cranially through two-thirds of the sternum. The muscles overlying the sternum were reflected using a combination of sharp incision, periosteal elevation and diathermy. The sternbrae were partly incised with a scalpel blade and the sternotomy was completed with scissors exposing both pleural spaces. Approximately 100ml of yellow-grey purulent material was aspirated from the pleural cavity. Necrotic mediastinal tissue was resected, including what appeared to be a walled-off abscess in the caudal central part of the thorax (Figure 2, p212). Complete debridement was not possible without lung trauma; therefore, necrotic adhesions to the visceral pleura were not removed. Copious lavage of the thoracic cavity with sterile saline was performed. Thoracic drains (Thoracic trocar and drain; Vygon: 14 Ch) were inserted into both pleural cavities, entering the thorax at the right and left eighth intercostal spaces and extending cranially on the thoracic floor. The sternotomy was closed with figure-of-eight sutures of 4 metric polypropylene (Prolene; Ethicon). The subcutaneous tissue and skin were closed routinely. A light dressing was placed around the thorax to secure the thoracic drains in place.

The cat was transferred to the intensive care unit for recovery from the surgical procedure. At this stage it was unconscious

with absence of bilateral palpebral or pupillary light reflexes. It remained intubated due to the lack of an airway reflex. Positive-pressure ventilation was necessary following the end of anaesthesia due to the increased work of breathing caused by restrictive pulmonary disease and drug-induced respiratory depression. Ventilatory support was continued for six hours on 100% oxygen until the cat could maintain adequate end-tidal carbon dioxide and oxygen saturation on spontaneous ventilation and supplemental 40% oxygen.

Continuous intensive monitoring was necessary in the postoperative period. For the first 24 hours after surgery, temperature, heart rate, respiratory rate and oxygen saturation were monitored hourly, and were then monitored every four hours for the following 72 hours. Urine output was assessed regularly and the cat was turned every four hours.

Blood pH progressively normalised over the following 24 hours. Lactated Ringer's solution with 2.5% glucose added was administered intravenously at a rate of 5ml/kg/h and the infusion of dopamine was maintained for 24 hours postoperatively to ensure adequate urine production. Effective analgesia was maintained with morphine sulphate (0.2mg/kg intramuscularly every six hours) and fentanyl (Durogesic; Janssen-Cilag, 2.5mg transdermal patch). Eighteen hours after the end of anaesthesia, the cat had regained bilateral palpebral and pupillary light reflexes. Its responsiveness gradually returned to normal over the following 48 hours.

The thoracic cavity was lavaged with 20ml/kg of warm sterile physiological saline solution four times daily for six days. Cytological analysis of the lavage fluid seven days post-surgery revealed a moderate purulent inflammatory exudate without evidence of bacteria. Thoracic lavage was continued once daily for a further two days and then the thoracic drains were removed. By this stage the cat was breathing normally and the thoracic lavage fluid had a clear physical appearance.

Oesphagostomy tube feeding was instituted once the cat could hold up its head. This was continued at four to six hour intervals until the seventh day post-surgery, at which stage the cat was spontaneously eating adequate quantities of food. The cat was maintained on amoxicillin-clavulanate (Synulox; Pfizer: 20mg/kg orally twice daily) and enrofloxacin (Baytril; Bayer: 5mg/kg orally once daily) for 21 days post-surgery.

Thoracic radiographs were taken 15 days post-surgery and showed a complete resolution of the pleural effusion. The lungs were fully inflated and the thoracic structures were clearly visible (Figure 3, p213). No clinical or radiographic abnormalities were detected at a follow-up examination one year after surgery.

Discussion

Possible causes of pyothorax in cats include thoracic trauma, extension from bacterial pneumonia, oesophageal perforation, migrating foreign bodies, haematogenous and iatrogenic from thoracocentesis or thoracotomy (Monnet, 2003; Waddell *et al.*, 2002). Affected cats were predominantly young cats and from multi-cat households (Waddell *et al.*, 2002). The cat in this report

displayed the characteristic signalment and presenting clinical signs, including hypersalivation, which has been associated with a lower survival rate (Waddell *et al.*, 2002).

Cytological examination of the pleural fluid confirmed the diagnosis of pyothorax. Bacteriological culture of *Pasteurella multocida* and an *Actinomyces* spp. is consistent with the literature on pyothorax in the cat (Walker *et al.*, 2000; Love *et al.*, 1982; Waddell *et al.*, 2002). Bilateral pleural effusion, as found in this case, is the most common radiographic finding in cats with pyothorax (Jonas, 1982; Waddell *et al.*, 2002; Demetriou *et al.*, 2002). Thoracic ultrasonography revealed extensive changes within the thoracic cavity indicative of loculation and this strongly aided the decision to perform surgery. Haematological examination in this case revealed a leucopenia with a neutropenia and a degenerative left shift. Waddell *et al.* (2002) found a correlation between these findings and a lower survival rate, and concluded that these cases had the more severe pyothorax.

Treatment for pyothorax can be medical and/or surgical, although the most appropriate treatment protocol has not been determined. Medical treatment involves administration of intravenous fluid, broad-spectrum antimicrobial therapy and thoracic drainage with or without thoracic lavage (Jonas, 1982; Tomlinson, 1980; Fooshee, 1988; Waddell *et al.*, 2002; Demetriou *et al.*, 2002; Monnet, 2003). Thoracic drainage is usually provided by placement of thoracostomy tubes. Thoracostomy tube placement has been associated with an improved survival rate (Waddell *et al.*, 2002). There is controversy regarding the necessity for thoracic lavage as a treatment for pyothorax (Waddell *et al.*, 2002). Recently Demetriou *et al.* (2002) reported faster recovery times with lavage than without lavage, although their criteria for the discontinuation of lavage are not clear.

Pyothorax in the cat is a serious life-threatening condition, with a reported survival rate of 43% to 92% for either medically or surgically treated cases (Jonas, 1982; Davies and Forrester, 1996; Waddell *et al.*, 2002; Demetriou *et al.*, 2002). In two reports, seven of 16 cats (Davies and Forrester, 1996) and 21 of 80 cats (Waddell *et al.*, 2002) presented dead or were euthanised within the first 24 hours of examination. Waddell *et al.* (2002) noted that the most severely affected cats may have been euthanised on presentation, resulting in a higher than expected survival rate in the treated cats.

Previously, surgical management has been recommended in cases where there had been no response to medical management (Jonas, 1982; Tomlinson, 1980; Fooshee, 1988; Monnet, 2003) and in cases with an intrathoracic mass or lung lobe abscess was diagnosed on thoracic ultrasonography (Waddell *et al.*, 2002; Demetriou *et al.*, 2002). The survival rate for surgically managed cats (five of five cats survived) was recently reported to be significantly higher than medically managed cats (34 of 54 cats survived) (Waddell *et al.*, 2002). The advantages of surgical management are thorough exploration and debridement of the pleural space. This must be weighed against the risk of a longer general anaesthetic in a compromised patient, an increased cost

peer reviewed

and length of hospital stay, and the pain associated with surgery (Waddell *et al.*, 2002).

In this case, the decision for surgical management was based upon the very low volume of viscous flocculent pleural fluid aspirated on thoracocentesis and the ultrasonographic appearance of extensive loculations within the caudal thoracic cavity. Due to the severity of the cat's clinical condition, it was felt that early aggressive surgical management was preferable to prolonged medical management. Exploratory sternotomy allowed debridement of a partially walled-off abscess and the removal of purulent and necrotic material from the pleural cavity.

The anaesthetic management of this case was complex and demanding due to severe restrictive pulmonary disease caused by the pyothorax, sepsis, metabolic and respiratory acidosis, low systolic blood pressure and the depressed mental state. Restrictive pulmonary disease is due to disorders involving the pleura and chest wall. It is characterised by decreased lung compliance so that lung volumes are typically reduced leading to increased work of breathing. Increased work of breathing may eventually lead to exhaustive respiratory failure.

Restrictive pulmonary disease along with pulmonary parenchymal disease (for example, pneumonia) may lead to persistent hypoxia, which may require positive-pressure ventilation (Pascoe, 1988; Moon and Concannon, 1992; King and Hendricks, 1994; Haskins, 2003). This is a labour-intensive procedure, requiring constant monitoring and care of the animal (King and Hendricks, 1994; Haskins, 2003). The recommended ventilation pattern should aim to maintain low tidal volumes (10ml/kg), a low peak proximal airway pressure (10 to 20cm H₂O) and a respiratory rate of about 15 times per minute (Haskins, 2003). Ventilation was essential in this case in the management of the severe acidosis and to ensure adequate oxygenation. Mechanical ventilation often requires sedation or anaesthesia (King and Hendricks, 1994; Haskins, 2003), but this was not necessary due to the depressed mental state of the cat in the post-operative period. Only short-term ventilation was required, until the acidosis was no longer life-threatening and oxygenation could be maintained by spontaneous ventilation and intranasal oxygen supplementation. Positive-pressure ventilation for pulmonary parenchymal disease in dogs and cats has been reported to have a survival rate of 20%; hypotension has been associated with a poorer prognosis (King and Hendricks, 1994).

Instituting early enteral nutrition in critically-ill patients is essential to manage and correct significant energy and protein deficits. Malnutrition is associated with higher complication rates and causes anaemia, hypoproteinaemia, delayed wound healing and decreased immune function (Mauldin and Davidson, 2003). Oesophagostomy tube placement and feeding is straightforward, well tolerated by the patient and associated with a low complication rate (Mauldin and Davidson, 2003).

In this case, the cat presented with clinical parameters associated with a poor outcome (Waddell *et al.*, 2002; King and Hendricks,

1994). However, intrathoracic debridement with postoperative ventilatory support and intensive supportive care led to a successful outcome.

References

- Davies, C. and Forrester, S.D. (1996). Pleural effusion in cats: 82 cases (1987 – 1995). *Journal of Small Animal Practice* **37**: 217-224.
- Demetriou, J.L., Foale, R.D., Ladlow, J., McGrotty, Y., Faulkner, J. and Kirby, B.M. (2002). Canine and feline pyothorax: a retrospective study of 50 cases in the UK and Ireland. *Journal of Small Animal Practice* **43**: 388-394.
- Fooshee, S.K. (1988). Managing the cat with septic pleural effusion. *Veterinary Medicine* **83**: 907- 913.
- Fuentes, V.L. (1998). Pleural and mediastinal disease. In: *Manual of Small Animal Cardiorespiratory Medicine and Surgery*. Edited by V.L. Fuentes and S. Swift. Cheltenham: British Small Animal Veterinary Association. pp235 - 244.
- Haskins, S.C. (2003). Positive-pressure ventilation. In: *Textbook of Small Animal Surgery, Volume II*. Third edition. Edited by D. Slatter. Philadelphia: Saunders. pp 2623-2629.
- Holmberg, D.L. (1979). Management of pyothorax. *Veterinary Clinics of North America: Small Animal Practice* **9**: 357-362.
- Jonas, L.D. (1983). Feline pyothorax: A retrospective study of twenty cases. *Journal of the American Animal Hospital Association* **19**: 865 –871.
- King, L.G. and Hendricks, J.C. (1994). Use of positive-pressure ventilation in dogs and cats: 41 cases (1990–1992). *Journal of the American Veterinary Medical Association* **204**: 1045 – 1052.
- Love, D.N., Jones, R.F., Bailey, M., Johnson, R.S. and Gamble, N. (1982). Isolation and characteristics of bacteria from pyothorax (empyema) in cats. *Veterinary Microbiology* **7**: 455 – 461.
- Mauldin, G.E. and Davidson, J.R. (2003). Nutritional support of hospitalised cats and dogs. In: *Textbook of Small Animal Surgery, Volume I*. Third edition. Edited by D. Slatter. Philadelphia: Saunders. pp87 - 111.
- Monnet, E. (2003). Pleura and pleural space. In: *Textbook of Small Animal Surgery, Volume I*. Third edition. Edited by D. Slatter. Philadelphia: Saunders. pp387-405.
- Moon, P.F. and Concannon, K.T. (1992). Mechanical ventilation. In: *Current Veterinary Therapy XI. Small Animal Practice*. Edited by R.W. Kirk and J.D. Bonagura. Philadelphia: Saunders. pp 98 - 104.
- Pascoe, P. (1988). Oxygen and ventilatory support for the critical patient. *Seminars in Veterinary Medicine and Surgery (Small Animal)* **3**: 202-209.
- Tomlinson, J. (1980). Review of pyothorax in the feline. *Feline Practice* **10**: 26-32.
- Waddell, L.S., Brady, C.A. and Drobatz, K.J. (2002). Risk factors, prognostic indicators, and outcome of pyothorax in cats: 80 cases (1986-1999). *Journal of the American Veterinary Medical Association* **221**: 819-824.
- Walker, A.L., Jang, S.S. and Hirsch, D.C. (2000). Bacteria associated with pyothorax of dogs and cats: 98 cases (1989 – 1998). *Journal of the American Veterinary Medical Association* **216**: 359 - 364.