Patient care report for the stabilisation of a cat with a traumatic diaphragmatic rupture

Abstract

Diaphragmatic ruptures in cats are often seen following trauma and can cause severe respiratory distress. This article reflects on the author's experience of providing emergency nursing care to a 2-year-old, domestic short hair cat, with a traumatic diaphragmatic rupture. In order to provide effective nursing care it is essential for the veterinary nurse to understand the effects of shock in the feline patient. Creating a protocol for the management of the dyspnoeic trauma cat could facilitate a more efficient treatment plan, allowing for available monitoring equipment to be fully utilised. If advanced monitoring techniques are not available, a successful patient outcome is still achievable with basic nursing skills and a good underpinning knowledge of the emergency and critical patient.

Key words: diaphragmatic hernia, respiratory distress, shock, oxygen therapy, fluid therapy, cat

This patient care report highlights the initial veterinary nursing interventions provided to an emergency feline patient. The patient presented to the practice with shock and dyspnoea following a suspected traumatic incident.

Signalment

Species: Feline
Breed: Domestic short hair
Age: 2 years 3 months
Sex: Neutered male
Weight: 3.18 kg

Presenting problem

The patient presented with a history of acute dyspnoea and lethargy progressing to collapse. He was reported to be inappetant and it was unknown whether he had passed urine or faeces within the last 24 hours. The owner reported that he had not vomited overnight and that preceding this event he was a healthy cat. He had never been seen by a veterinarian and so had no known previous history.

Initial patient assessment
Respiratory

Visual assessment of the patient revealed tachypnoea (respiration rate (RR) of 44 breaths per minute

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(bpm)) and paradoxic respiration (when breathing movements are in reverse to normal, i.e. chest wall moves in on inspiration and out on expiration). He lay in sternal recumbency with abducted elbows and neck stretched outwards (orthopnoea). Auscultation revealed dull lung sounds and muffled heart sounds; his airways appeared clear from obstruction.

Cardiovascular

Heart rate (HR) was bradycardic at 120 beats per minute (btpm) with no notable arrhythmias. He had absent peripheral pulses and pale pink mucous membranes (MMs). The capillary refill time (CRT) was delayed at 2–3 seconds. He was severely hypothermic with a core temperature of 34.1°C. An initial systolic blood pressure (BP) reading was attempted using the Doppler technique (the only available method in that practice), but was unsuccessful, as the carpal pulse could not be determined.

Neurological

There were no signs of extracranial injuries, but he was obtunded and quiet. The patient's pupils were symmetrical and pupillary responses were normal.

Veterinary investigations

An intramuscular injection of a partial μ receptor agonist, buprenorphine (Vetergesic, Ceva Animal Health Ltd, Buckinghamshire, UK), 20 μg/kg was given. Alternatively a full μ opioid agonist such as morphine or methadone may have been used. These act at a number of different receptor systems and compared with partial μ receptor agonists provide more effective analgesia (Murrell, 2011). A study by Steagall et al (2006) found that administration of methadone and morphine provided a longer reduced sensitivity to pain compared with buprenorphine. The use of full μ opioid agonists are indicated in the management of moderate to severe pain and as methadone is the only full μ opioid agonist with veterinary marketing authorisation, this should be used over morphine (Murrell, 2011).

An initial blood sample was obtained to provide a survey of the patient's clinical status to tailor the treatment plan accordingly (Chapman, 2013; Tompkins, 2013). Biochemistry results revealed mildly elevated urea 14.9 mmol/litre (normal range 5.7–12.9

mmol/litre), high alanine transaminase 740 U/litre (normal range 12–130 U/litre) and high gamma-glutamyl transferase 4 U/litre (normal range 0–1 U/litre). Complete blood count revealed increases in haemoglobin 16.5 g/dl (normal range 9.0–15.1 g/dl) and neutrophilia 13.66 x 10⁹/litre (normal range 2.50–12.50 x 10⁹/litre); total protein and haematocrit were both normal.

A radiograph revealed a diaphragmatic rupture. No signs of a dilated stomach were detected (which would indicate the need for immediate surgical intervention (Gibson et al, 2005)), so it was decided that the patient should be stabilised for 24 hours prior to surgical repair.

Discussion

Diaphragmatic ruptures are a common cause of respiratory distress in cats (Cariou et al, 2009) and require rapid intervention (Rozanski and Chan, 2005; Jordan, 2011; Sharp and Rozanski, 2013; Sumner and Rozanski, 2013) such as oxygen therapy, shock interventions and pain management. Trauma is a common cause of diaphragmatic rupture and there are often concurrent problems such as shock (Schmiedt et al, 2003) that need to be addressed in order to ensure the most immediate threats to the patient's life are treated as priorities.

Nursing considerations and interventions

The patient displayed signs of pain on his hind quarters and his ability to mobilise, urinate and defecate was monitored. Also consideration was given to pain management, nutritional requirements, stress reduction and self care, which were analysed using the Orpet and Jeffery Ability Model (2007) (Orpet and Welsh, 2011). To discuss all of these nursing requirements is beyond the scope of this care report. Therefore, the author has focused this discussion on the nursing interventions required to address his immediate and life-threatening disorders.

Oxygen therapy

It is widely agreed that oxygen therapy should be provided immediately to the dyspnoeic patient (Mackay, 2001; Rozanski and Chan, 2005; Jordan, 2011; Sharp and Rozanski, 2013). In this incident a clinical examination took place before initiating oxygen therapy. Although this could have prolonged the patient's discomfort Sumner and Rozanski (2013) advise that a brief assessment before placement into an oxygen cage can exclude other causes of distress such as stress, cardiac and/or pulmonary disease. It could have been possible to achieve both the initial clinical examination and oxygen support for this patient if methods such as flow-by oxygen (achieving a mean fraction of inspired oxygen (FiO2) of 24–45% (Sumner and Rozanski, 2013)) had been provided. To further improve FiO2 from 24–45% to 35–55% a face mask could have been used (Sumner and Rozanski, 2013). This is not always tolerated well and can increase stress and therefore respiratory effort (Sharp and Rozanski, 2013). For this reason, providing flow-by oxygen may have been the better approach for this patient in order to minimise stress while oxygen therapy was provided. However, it should be noted that using flow-by oxygen is labour intensive as veterinary personnel need to be available to hold both the oxygen circuit and patient (Sharp and Rozanski, 2013). In addition, using flow-by oxygen is labour intensive as veterinary personnel need to be available to hold both the oxygen circuit and patient (Sharp and Rozanski, 2013).

A useful way to provide oxygen therapy is with an oxygen cage (Mackay, 2001; Mazzaferro, 2009). In the dyspnoeic cat this can be particularly useful as it does not require patient restraint (Mackay, 2001) and the cage can reach reasonably high concentrations of oxygen (up to 60% FiO2 (Sumner and Rozanski, 2013)) depending on the size of the cage, patient and flow rate (Richmond, 2010). This patient was placed into an oxygen cage after his initial assessment; on admission his RR was 48 bpm and after 30 minutes this had reduced to 38 bpm.

The oxygen cage (Buster® ICU cage, Kruuse, Marslev. DK) was a basic model and could not control all aspects of the environment inside the cage. Some complex models are able to control humidity, temperature and oxygen levels which can be modified from the outside of the cage. With the Buster® ICU cage the door had to be opened at regular intervals to gauge the temperature inside. This was to ensure the patient did not become hyperthermic as there was no temperature monitoring equipment in this particular Buster® ICU cage. This results in a rapid fall in the FiO2 (Sharp and Rozanski, 2013) which could have been detrimental to the patient. To compensate for this, the author suggests the use of a wall thermometer, attached to the inside of the cage. This could allow for the environmental temperature of the oxygen cage to be monitored visually, reducing the need to open the door unnecessarily.

Although the oxygen therapy improved the patient's RR, it did not resolve the dyspnoea. The lowest recorded RR for this patient was 36 bpm (normal ranges 10 to 20 bpm (Jordan, 2011)) and he still displayed some respiratory effort. This could be explained by the fact that a diaphragmatic hernia can be classified as a pleural space disease (Pachtinger, 2013), which may only benefit from therapeutic in-

tervention (Sumner and Rozanski, 2013) or surgical intervention (Gibson et al, 2005).

To assess general patient improvement the registered veterinary nurse (RVN) observed for changes to MM colour, a reduction in RR and improved demeanour. Blood gas analysis is the 'gold standard' method for assessing oxygenation and ventilation. Blood gas analysis or arterial blood gas (ABG) analysis provides a direct assessment of gas exchange, giving an overall view of pulmonary function (Miller, 2007). To assess the lung's ability to oxygenate blood the value of partial pressure of oxygen in arterial blood (PaO2) can be measured. However, this is meaningless without knowing the exact FiO2 (Moens and Coppens, 2007) (see *Table 1* for normal ranges). In order to assess ventilation, ABG remains the gold standard however, ventilation can be assessed by capnography, which may be advantageous as it is not invasive and allows for continuous monitoring (Pypendop, 2009). Capnography measures the arterial partial pressure of carbon dioxide (PCO2) although it is more accurate in intubated patients (Pypendop, 2009). Unfortunately capnography was not available in the author's practice. Therefore ventilation could not be accurately assessed.

As well as the lack of capnography, the author's practice does not currently have an ABG analyser. If these had been available they could have been used to determine how adequate the patient's pulmonary function was, facilitating the evaluation of his nursing care needs. When deciding on ABG analysis it should be considered that interpretation is complex. Furthermore, obtaining a sample may be distressing for the patient and therefore detrimental to their condition. The approach that patients should not be taken off oxygen therapy to obtain an assessment of oxygen levels (Sumner and Rozanski, 2013) should be applied.

Instead of ABG analysis the use of a pulse oximeter could have been considered, as this is non-invasive method of measuring the patient's oxygen saturation

(Pachtinger, 2013). However, pulse oximetery only provides a ratio of the percentage of haemoglobin saturated with oxygen (SpO2) using infared light (Moens and Coppens, 2007). The PaO2 measured in ABG displays the oxygen molecules dissolved in plasma, i.e. not bound to haemoglobin. Therefore, to determine how much oxygen is in the blood, combined measurements of PaO2, oxygen saturated arterial blood (SaO2) and haemoglobin content (CaO2) need to be assessed (Martin, 1999). Oxygen therapy was discontinued when the patient was considered stable (reduced RR, improved respiratory effort and improved pallor) and only auscultation and visual checks continued. A pulse oximeter could have been used for continued monitoring, giving a measurement of the patient's oxygenation. However, as patients with diaphragmatic ruptures are most likely to have issues with ventilation, measuring the PCO2 or partial pressure of carbon dioxide in the blood (PaCO2) may have been more beneficial (Barton, 2009).

Fluid therapy
The impairment to cellular oxygen delivery due to the failure of global tissue perfusion is described as hypoperfusion and is commonly referred to as shock (Boag and Hughes, 2005; Jordan, 2012). One of the most common forms of shock in the emergency patient is hypovolaemia (Boag and Hughes, 2005) and is characterised in most species by hypotension (decompensated hypovolaemia), tachycardia, pale MMs with a prolonged CRT, mild to absent peripheral pulses and altered mentation (Boag and Hughes, 2005; Goggs et al, 2008; Mazzaferro and Powell, 2013).

In comparison to dogs, cats have unique physiological responses to shock and display clinical effects associated with bradycardia, hypotension, and hypothermia, known as the 'shock triad' (Kirby, 2004). These findings are supported by Brady et al (2000) who found that in cats with distributive shock, over half presented with these three clinical abnormalities. The shock triad were present in this patient so bolus compound sodium lactate (Aquapharm 11 Hartmann's solution, Animalcare Ltd, York, Uk) 40 ml/kg (127.2 mls), over 30 minutes, was prescribed. This was to be continued until a minimum BP of 80 mmHg was recorded. During this time the RVN monitored for improvements in pulse strength, HR and mentation and systolic BP monitoring was repeated using the Doppler technique. The veterinary surgeon requested that the fluids were to be continued at 40 ml/kg, to achieve a minimum systolic BP of 80 mmHg, with the view to reassessing fluid administration rates in order to ensure a normal BP was reached. Waddel and Brown (2009) advise that in cats a normal systolic BP is between 120–170

Table 1. Normal values of blood gas analysis

Parameter	Normal value	Decrease	Increase
PaCO2	35–45 mmHg	Respiratory component: alkalosis	Respiratory component: acidosis
PaCO2	If oxygen levels in inspired gas (FiO2) = 21%: 80–100 mmHg (10.7–13.3kPa	Hypoxaemia: <60 mmHg (8kPa)	Due to increase FiO2 or increased atmospheric pressure
SaO2	95–100%	Hypoxaemia: <90%	
Moen and Coppens (2007)			

mmHg, with hypotension being defined as less than 80 mmHg. This supports the veterinary surgeon's goal of providing shock rate fluids to achieve a minimum systolic BP of 80 mmHg.

Venous access had to be achieved to provide fluid therapy, which is often the responsibility of the RVN (Goddard, 2010). The fur at the patient's right cephalic vein was clipped, and oxygen supplementation provided via flow-by during this time. There were no visual signs of the vein and it could not be palpated, so, the area at the right lateral saphenous vein was clipped instead. With successful visualisation of the saphenous vein, lignocaine 25 mg/g and prilocaine 25 mg/g cream (Emla, AstraZeneca UK Limited, Bedfordshire, UK) was applied and left for 15 minutes. A 22 g intravenous (IV) catheter was inserted by the RVN, using an aseptic technique and secured and dressed accordingly. The RVN calculated that 21.2 mls of Aquapharm 11 could be given every 6 minutes, in an attempt to provide the prescribed dose of 40 mls/kg/30 minutes. Although this only allowed the administration of 21.2 mls of fluid every 6 minutes and not the prescribed dose of 40 ml/kg over 30 minutes. Using an infusion pump or syringe driver may have been a more appropriate way to administer the recommended bolus of fluids (Aldridge, 2009).

Although the choice and rate of the fluids prescribed is the responsibility of the veterinary surgeon the RVN must understand the reasons for selected rates of infusion and the physiological effects of administration (Sheridan, 2009) to avoid associated complications such as over infusion or inadequate fluid resuscitation. It is generally the RVN's role to administer fluid therapy, and close observation of the patient while doing so is important. It should be appreciated that cats are at risk of volume overload; careful administration along with thoracic auscultation should be performed to monitor for signs of pulmonary and pleural fluid accumulation (Kirby, 2004), which would indicate signs of over infusion. Should these become apparent fluid therapy should be stopped, oxygen therapy initiated and the veterinary surgeon informed immediately, so that the patient can be assessed and administration of drugs (i.e. diuretics) can be initiated if indicated (Davis et al, 2013).

After the first 127.2 mls of Aquapharm 11 was administered, a systolic BP of 60 mmHg was recorded. After a further 18 minutes of fluid administration a systolic BP of 80 mmHg was achieved. At this stage the patient's other vital signs had also shown improvement. The RVN noted that peripheral pulses were stronger, the HR had increased to 168 btpm and MMs were pinker with a CRT of <2 seconds. The pa-

tient's temperature had also increased from 34.1°C to 37.9°C and he appeared more alert and responsive.

The method in which this patient was reperfused is supported by many (Kirby, 2004; Boag and Hughes, 2005; Goggs et al, 2008; Mensack, 2008; Pachtinger, 2013), but it could be argued that perfusion in this patient could have been monitored more accurately by measuring serum lactate concentrations (Karagiannis et al, 2006) or systemic oxygen parameters (Boag and Hughes, 2005). It is important to highlight that (as with this patient) these monitoring tools are not always available, regardless, the RVN's role of monitoring critical patients is crucial. They can detect physiological changes in the patient and facilitate the development of the care plan to avoid patient deterioration (Polton and Branscombe, 2008).

Hypothermia

In order to avoid peripheral vasodilation, caused by warming the patient while hypovolaemic, hypothermia was the last abnormality to be addressed. Severe hypothermia causes the hypothalamus to become less responsive and if the core temperature drops below 31°C, thermoregulation is unattainable (Kirby, 2004). To avoid the risk of diminished thermoregulation, patient warming was initiated using the Buster® ICU cage. This acted like a heated incubator by increasing the air temperature around the patient. It was decided against the use of blankets so that the patient could be observed easily. This approach was also adopted by Tompkins (2013) who opted out of the use of a forced air warming blanket so that patient observations could be continued without restrictions.

This method did help increase the patient's rectal temperature to 36°C. The patient was removed from the Buster® ICU cage when the environment inside was deemed to be too hot for the patient to reside in comfortably. This was judged by observing the patient's behaviour for signs of discomfort, e.g. restlessness, panting (Boag and Nichols, 2011) and by how warm the inside of the cage felt to the RVN. As previously stated, using a thermometer or thermostat mounted on the inside of the cage would have been a more reliable way to assess the temperature and could have reduced the risk of removing the patient from an oxygen enriched environment too early. When the patient was removed from the Buster® ICU cage, passive surface re-warming (with the use of bubble wrap and blankets) continued until a core temperature of 37.9°C was achieved. The use of heat pads was avoided to eliminate the risk of skin burns and to prevent peripheral vasodilation. As sensory nerve fibres in the skin are particularly sensitive to changes in local temperature (Sjaastad et al, 2003), peripheral vasodilation

Key Points

- Diaphragmatic ruptures are a common cause of respiratory distress in cats and require rapid intervention.
- In comparison to dogs, cats have uniquely different physiological responses to shock and display a triad of clinical effects identified as bradycardia, hypotension and hypothermia.
- When advanced monitoring techniques are not available, a successful patient outcome is still achievable with basic monitoring skills and good underpinning knowledge.

could cause a reduced response to neuronal feedback, thus decreasing the body's ability to maintain core internal temperature (Kirby, 2004).

Armstrong et al (2005) and Byers (2014) suggest that for moderate to severe hypothermic patients the use of active surface re-warming, such as forced air warming blankets, should be considered. These minimise heat loss by raising the temperature of the microenvironment of the skin, reducing the temperature gradient between the body and the environment (Armstrong et al, 2005). The author's practice does not currently have a forced air warming blanket, but this could have been useful instead of passive surface re-warming. With the use of bubble wrap and blankets it took approximately 4 hours for the patient to reach an optimal core body temperature, which may have been reduced with a forced air warming blanket.

Additional recommendations

In addition to the recommendations made in the previous section the author would suggest that a protocol for the management of dyspnoeic trauma patients is created. Although it is important to consider the individual needs of a patient, the availability of a guiding protocol could facilitate the development of a more efficient treatment plan. A study by Hancill (2013) found that introducing a 'care bundle' (a set of evidence-based interventions performed simultane-

ously) improved some individual quality indicators, i.e. patient checks, and although compliance in their use was low, the potential for these 'care bundles' to improve patient care was recognised. Once guiding protocols are established, they can be adopted by the RVNs to ensure that basic therapy, such as immediate oxygen supplementation or the use of available monitoring systems, is not overlooked.

Patient outcome

The care administered to this patient did have a positive outcome. The patient stabilised overnight and had surgery to repair the diaphragmatic rupture the following day. The patient made a full recovery and was discharged after a total of 3 days' hospitalisation.

Conclusion

Initial assessment of the dyspnoeic patient identifies relevant clinical issues and facilitates the creation of appropriate nursing interventions. In turn, this allows for the RVN to evaluate the care given and highlight any problems that have progressed or improved. Even when advanced monitoring techniques are not available, a successful patient outcome is still achievable with basic monitoring skills and a good underpinning knowledge of the pathophysiology of disease processes in the emergency and critical patient.   **VN**

Acknowledgements

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