



Part 1

Nursing the canine patient with negative pressure pulmonary oedema

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Lydia M. Barry, BSc (Hons), RVN, Cert VNECC
Paragon Referrals, Wakefield

✉ lydsbarry@hotmail.co.uk

Lydia graduated in 2017 from Edinburgh Napier University with a first-class BSc (Hons) Veterinary Nursing degree. She worked in private and charity practices before moving to Paragon Referrals in Wakefield. Lydia is the ward team leader, overseeing in-patient care and ICU. Her special interests include nursing neurological patients, emergency and critical care, and education.



Susannah Potheary, BVSc, MRCVS
Paragon Referrals, Wakefield

Susie graduated from the University of Liverpool in 2017. She worked in a busy small animal practice in the heart of Yorkshire for 2 years before moving to Paragon in 2020. Susie recently completed her rotating internship, where she developed a strong interest in anaesthesia and emergency and critical care, which she hopes to pursue in the future.

ABSTRACT Non-cardiogenic pulmonary oedema can occur in canine patients as a result of various events. Post-obstructive oedema can occur post choking and results in a rapid accumulation of fluid in the extravascular spaces of the lung. Arterial blood gas analysis and intense patient monitoring are vital to determine the need for further intervention. Treatment can include ventilation, oxygen therapy and medication.

Keywords pulmonary, oedema, canine

Introduction

Pulmonary oedema is the accumulation of fluid in the extravascular spaces of the lung (Bouyssou et al., 2017). It can be categorised into cardiogenic or non-cardiogenic, with the latter being less commonly encountered (Glaus, 2012). This article reports on the multidisciplinary team approach to caring for a puppy diagnosed with negative pressure pulmonary oedema, which developed after choking on a treat. This is a form of non-cardiogenic pulmonary oedema that occurs following obstruction of the upper airway.

The case report describes the pathophysiology of the disease, and discusses the effectiveness of nursing interventions based on current evidence. This includes the team approach to triage, patient monitoring and record keeping, ventilation and oxygenation status, and the medication prescribed to the patient.



Case presentation and investigations

Buddy, a 4-month-old border collie, was presented to the referring veterinary practice 24 hours after a choking event. The patient was eating a treat when he collapsed and began choking. Buddy dislodged the obstruction but deteriorated rapidly.

Prior to arrival at the clinic, the patient underwent conscious thoracic radiographs, along with blood analysis at the referring veterinary practice. There

were no significant changes on full biochemistry, and complete blood counts. Orthogonal thoracic radiographs revealed a diffuse bilateral alveolar to interstitial pattern. There was no obvious obstruction to the main airways and no cardiac abnormalities.

Patient assessment

The patient was triaged immediately on arrival by the veterinary nurses, while the clinician obtained a history and gained owner consent. The respiratory, cardiovascular, neurological and renal systems were assessed systematically during the primary survey.

Buddy was tachypnoeic with a respiratory rate between 60 and 80 breaths per minute, with an orthopnoeic posture. This was a concern as, in human medicine, tachypnoea is documented as a discrete and early sign of deterioration (Latimer-Jones, 2020). Additionally, the ribcage of paediatric patients is more compliant, resulting in less competent ventilation and increased respiratory effort (Louro et al., 2019). This could lead to worsened hypoxia and ventilatory fatigue (Louro et al., 2019).

On auscultation of the chest, crackles were identified in all lung fields. These sounds are helpful when considering differentials for diagnosis, as crackles could indicate pulmonary oedema, whereas wheezes are the result of airway narrowing, and absent sounds a result of pleural disease (Latimer-Jones, 2020).

The cardiovascular assessment identified tachycardia, weak and narrow peripheral pulses, cyanotic mucous membranes and an extended capillary refill time (CRT). A prolonged CRT is an indicator of poor peripheral perfusion, which was assumed to be because of peripheral vasoconstriction associated with hypovolaemic shock. Buddy was weak and unwilling to stand but had a normal mentation.

Differential diagnoses

The problems included tachypnoea, cyanotic mucous membranes, and a diffuse alveolar to interstitial pattern on radiographs. There were several differentials, including cardiogenic oedema, non-cardiogenic oedema, aspiration pneumonia or haemorrhage within the alveoli. In addition, systemic disease such as sepsis or acute pancreatitis may result in acute respiratory distress syndrome (ARDS), which can cause a marked non-cardiogenic pulmonary oedema (Glaus, 2012). Furthermore, infections such as leptospirosis are well documented to cause a protein-rich non-cardiogenic pulmonary oedema due to vasculitis (Glaus et al., 2010). Haemorrhage may be due to a multitude of conditions including trauma, coagulopathies, parasites such as *Angiostrongylus* and, potentially, neoplasia such as haemangiosarcoma (Powell, 2002).

Differentiating between these options is often done via a review of the clinical history, vaccination status and patient examination (Sumner & Rozanski, 2013). This patient had no audible murmur or dysrhythmia and no radiographic changes to indicate a cardiac cause (Tong & Gonzalez, 2020). In one paper, post-obstructive pulmonary oedema appeared to show a more asymmetrical unilateral presentation compared to neurogenic pulmonary oedema. However, the paper also states that if the condition is severe, then changes appear to be more diffuse and bilateral (Bouyssou et al., 2017).

In this case, the patient had an acute onset of signs after the owners witnessed an incidence of upper airway obstruction and a period of collapse immediately prior to the onset of clinical signs. In addition, younger animals are at a higher risk of hypoxia due to their smaller functional capacity and thus more at risk of developing pulmonary oedema (Louro et al., 2019). Therefore, a presumptive diagnosis was made of negative pressure pulmonary oedema. In retrospect, an electrocardiogram (ECG) and echocardiography could have been indicated, to rule out any cardiac disease (Agudelo & Schanilec, 2015).

Pathophysiology

Pulmonary oedema typically arises due to an increased intravascular hydrostatic pressure or a disturbed vascular permeability (Glaus, 2012). It can also be caused by an altered oncotic pressure gradient or lymphatic drainage (Glaus, 2012).

There are numerous events that may lead to the movement of fluid into the alveoli, including neurogenic oedema post brain-trauma or seizures, electrocution or re-expansion oedema following rapid removal of a pleural effusion or pneumothorax (Bouyssou et al., 2017). The described patient suffered negative pressure pulmonary oedema, secondary to airway obstruction (Louro et al., 2019). This can be caused by brachycephalic upper airway syndrome, laryngeal paralysis, tracheal collapse, endotracheal tube obstruction and strangulation.

During the obstruction, the patient struggled to breathe, resulting in an increased inspiratory effort. This can lead to mechanical ventilatory stress, which damages the pulmonary epithelium and endothelium (Glaus, 2012). The patient became hypoxic, leading to a sympathetic capillary vasoconstriction (Bouyssou et al., 2017). Additionally, the increased inspiratory effort likely resulted in a large negative transpulmonary and interpleural pressure gradient (Louro et al., 2019).

When negative pressure in the thoracic cavity occurs, it can lead to a decrease in venous blood flow to the heart, allowing blood to pool in the pulmonary vessels (Glaus, 2012). This increases the pulmonary intravascular volume which, when coupled with vasoconstriction,

results in a significant increase in hydrostatic pressure (Glaus, 2012).

There are limited reports in veterinary literature describing the treatment of such patients. The largest published research was a series of cases describing the radiographic appearance of 23 cases with pulmonary oedema (Boiyssou et al., 2017). Most recently, Louro et al. (2019) described the diagnosis and management of post-anaesthetic non-cardiogenic pulmonary oedema in a dog.

Arterial blood gas analysis

An arterial catheter was aseptically placed into the left dorsal pedal artery to allow for regular arterial blood gas analysis. A sample was taken on admission and then regularly throughout hospitalisation (**Figure 1**).

PATIENT'S BLOOD GAS ANALYSIS RESULTS

pH	– 7.4 (7.350–7.450)
PCO ₂	– 44.4 mmHg (35–38 mmHg)
pO ₂	– 56.5 mmHg (85–100 mmHg)
cHCO ₃	– 27.5 mmol/l (15–23 mmol/l)
BE (ecf)	– 2.7 mmol/l (–5–0.0 mmol/l)
cSO ₂	– 88.8% (90–100%)
Na ⁺	– 135 mmol/l (139–150 mmol/l)
K ⁺	– 3.4 mmol/l (3.4–4.9 mmol/l)
Ca ⁺⁺	– 1.19 mmol/l (1.12–1.40 mmol/l)
Cl ⁻	– 100 mmol/l (106–127 mmol/l)
TCO ₂	– 27.2 mmol/l (17–25 mmol/l)
Hct	– 33% (33–50%)
cHgb	– 11.1 g/dl (12–17 g/dl)
BE	– 2.3 mmol/l (–5–0.0 mmol/l)
Glu	– 7.6 mmol/l (3.3–6.4 mmol/l)
Lactate	– 1.33 mmol/l (0.6–2.9 mmol/l)
BUN	– 18 mmol/l (2.5–9.6 mmol/l)
Urea	– 6.5 mmol/l (3.6–9.3 mmol/l)
Crea	– 52 mmol/l (44–115 mmol/l)
BUN/Crea	– 31.0 mg/μg (0.2–400 mg/μg)
Urea/Crea	– 125.1 mmol/l (0.8–1615.4 mmol/l)

Figure 1. Patient's blood gas analysis results, including (in brackets) normal reference ranges for a canine arterial sample.

Arterial blood gas is considered the gold standard for monitoring oxygenation status (Farrell et al., 2019). The partial arterial pressure of oxygen (PaO₂) and carbon dioxide (PaCO₂) can indicate lung function.

The PaO₂ represents the oxygen saturation of haemoglobin in a sigmoid relationship according to

the oxygen dissociation curve (Waddell & King, 2018). Haemoglobin is expected to be fully saturated at a PaO₂ between 60 and 70 mmHg (Waddell & King, 2018). Animals with a normal lung function breathing room air typically have a PaO₂ five times greater than the inspired percentage concentration of oxygen (Angel & Seymour, 2015). Therefore, a patient breathing room air (~21% oxygen) should have a PaO₂ of 100 mmHg, whereas a patient breathing 100% oxygen when anaesthetised and intubated should have a PaO₂ of 500 mmHg (Angel & Seymour, 2015). A PaO₂ of less than 55 mmHg is immediately life threatening (Farrell et al., 2019).

Animals in severe respiratory distress are expected to have an increased respiratory rate and effort with hypoxaemia and hypocapnia with PaCO₂ values <35 mmHg. However, in this patient there was a marked hypoxaemia (PaO₂ 34.3 mmHg) and a relative hypercapnia (PaCO₂ 39.0 mmHg).

PaCO₂ is the primary indicator for the ventilatory function of a patient. Elevations in PaCO₂ typically develop due to hypoventilation as the patient is breathing inefficiently with a reduced alveolar minute ventilation (respiratory rate (RR) × tidal volume (TV)) (Hopper & Powell, 2013). It is important, however, to consider that in haemodynamically unstable patients, CO₂ will accumulate within the venous system due to low flow states and does not indicate an issue with ventilation (Hopper & Powell, 2013). Hypoventilation may be due to multiple issues, either neurological, metabolic or respiratory in origin, and can indicate respiratory fatigue.

In this patient, it was determined the relatively high PaCO₂ was a product of a reduced TV, as it has been shown that those with lung parenchymal disease have reduced compliance, which results in a lower TV (Rozanski, 2015). In addition, due to the poor compliance of the lungs, the work of breathing is greatly elevated and consequently respiratory fatigue may develop, resulting in orthopnoea and appearance of fatigue, like that of the patient (Hopper & Powell, 2013). To prevent this, mechanical ventilation was chosen as an intervention.

Further diagnostics

Diagnosis was based on history and clinical examination. Thoracic radiographs were taken at the referring veterinary practice and were not repeated on arrival. On reflection, thoracic ultrasound could have been used as it allows repeated assessment and is a non-invasive imaging method. Continuous ECG monitoring is necessary to monitor heart rate and the presence of dysrhythmias. However, in this case, the puppy was stressed and unwilling to allow ECG pads or clips to be placed.

Part 1 summary

There are numerous causes of non-cardiogenic pulmonary oedema, therefore it is vital to gain a detailed history to guide differential diagnoses. Having an understanding of the pathophysiology can help to guide diagnostic tests such as radiography and blood gas analysis, which will further allow specific treatments to be instigated. Often these patients present dyspnoeic and, to prevent further deterioration, the team must work efficiently and quickly to triage the patient. A whole-team approach proved useful in Buddy's case, where the veterinary nurses and interns assessed the patient while the veterinary clinician gained history and consent. The case study in Part 2 discusses the management of Buddy's airway, including ventilation, oxygen therapy and the medications prescribed.

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Case Study: Nursing the canine patient with negative pressure pulmonary oedema

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Lydia M. Barry, BSc (Hons), RVN,
Cert VNECC

Paragon Referrals, Wakefield

Susannah Pothecary, BVSc, MRCVS

Paragon Referrals, Wakefield



Initial treatment

The patient, a 4-month-old border collie named Buddy, was initially placed into a paediatric incubator with oxygen piped in. This allowed us to observe the patient while setting up materials and equipment for intravenous catheter placement

and blood sampling. This method was chosen over others, such as mask or flow-by, as the patient was easily stressed and did not tolerate handling. The aim of oxygen therapy was to correct hypoxaemia while providing supportive care to reduce stress. Oxygen should be delivered in the least stressful way, as movement and stress increase oxygen demand (Haskey, 2015).

However, the incubator resulted in an inappropriately warm environment and each time the door was opened the fractional inspired oxygen concentration (FiO_2) dropped. Following the analysis of the blood gases and the clinical presentation, it was determined the patient had signs consistent with respiratory fatigue and consequently failure. The decision was made to anaesthetise, intubate and ventilate the patient.

Ventilation

One of the major advantages of ventilating is the decreased effort of breathing by the patient and the ability to control tidal volume, respiratory rate, inspired oxygen and pressure controls (Rozanski, 2015). However, it is important to note that studies have shown patients with severe lung disease of non-infectious or cardiac causes that require ventilation tend to have a worse prognosis, with only 8% of these dogs managing to be weaned from ventilation, compared with 50% of dogs that required ventilation for aspiration pneumonia (Hopper & Powell, 2013).

Anaesthesia was induced with alfaxalone (Alfaxan, Jurox) to effect, and maintained on oxygen and isoflurane 1–2%.

Patients without neurological impairment require a light plane of anaesthesia to keep the patient comfortable while allowing sufficient pressures for adequate ventilation (Drellich, 2002). On review of this case, total intravenous anaesthesia (TIVA) would have been a better option as using an inhalant anaesthetic requires the damaged organ to maintain the anaesthesia (Drellich, 2002). In addition, it is important to consider the hypoxic pulmonary vasoconstriction (HPV) reflex, which results in the vascular smooth muscle of the pulmonary circulation constricting in response to regional hypoxia. This is an important mechanism to match regional ventilation and perfusion in the lungs, and all volatile agents, such as isoflurane, inhibit this beneficial mechanism (Tarry & Powell, 2017).

The patient was manually ventilated before changing to an anaesthetic ventilator (Wato Ex-35, Mindray) for more precise control of ventilation and use of several settings and measurements that can assist in the treatment of severe lung disease. These settings included pressure-controlled ventilation (PCV), volume-controlled ventilation (VCV), peak inspiratory pressure (PIP), positive end-expiratory pressure (PEEP), and synchronised intermittent mandatory ventilation (SIMV).

VCV results in a chosen tidal volume (TV) being delivered during inspiration compared to PCV, which delivers a breath until a pre-set PIP is reached (Hopper & Powell, 2013). PIP is the pressure generated in the anaesthetic breathing system that is required to push the gases into the lungs. In healthy lungs this should be relatively low, between 8 and 15 cmH_2O , but in diseased lungs, which can be less compliant, higher pressures may be required to drive a sufficient TV into the lungs (Drellich, 2002). PCV means that the volume delivered is limited to a specific pressure, which can reduce barotrauma to the lungs. However, when the lungs are less compliant, as they are when there is lung parenchymal disease, hypoventilation may occur as a smaller volume of fresh gas reaches the lungs and subsequent hypercapnia can develop (Rozanski, 2015; Drellich, 2002).

PEEP is the maintenance of a certain chosen pressure in the airway during expiration. This results in the lung being held in a semi-inflated state, preventing full exhalation, and stops any alveoli from collapsing (Hopper & Powell, 2013). PEEP can also prevent further injury to the damaged lung tissues as it reduces the shearing force that occurs when the alveoli snap open and closed (Drellich, 2002). It is important to remember, however, that cardiac filling relies on normal negative inspiratory pressures and PEEP can reduce cardiac output via reduced venous return in hypovolaemic patients (Barker, 2015).

The patient was initially placed on to VCV with a TV of 8–10 ml/kg and PEEP was increased to 5 cmH₂O. A lower TV was used because, in human patients with acute respiratory distress syndrome (ARDS), a lower TV is associated with a reduced mortality and a decrease in ventilator days (Brower, 2001). Although this information has not been shown in animals with ARDS, one small study confirmed lower TV are well tolerated in healthy dogs and it may be possible to use lower TV as a protective measure against ventilator-induced injury in patients with ARDS (Oura et al., 2012). Ideally PCV would be used to prevent any further trauma to the delicate alveoli. However, VCV was chosen due to the poor compliance of the lungs and risk of potentiating hypoventilation.

The first blood gas analysis of the patient indicated a marked hypoxaemia (PaO₂ 34.3 mmHg) and a relative hypercapnia (PaCO₂ 39.0 mmHg) for a patient with an increased respiratory rate. Immediately after induction, a second blood gas analysis indicated that, despite FiO₂ of 100%, the patient was still hypoxaemic (PaO₂ 98.8 mmHg) and severely hypercapnic (PaCO₂ 88.6 mmHg), confirming concerns that the patient was on the brink of respiratory failure. After the patient had been ventilated for 1 hour, a repeat arterial sample was taken. This showed improvements in oxygenation (FiO₂ 70%, PaO₂ 61 mmHg) and ventilation (PaCO₂ 54.5 mmHg). During this time, however, the patient continued to have an elevated respiratory rate due to the high ventilatory drive of hypoxaemia and, despite further sedatives being administered, operator control of ventilation was poor.

Following these blood gas results, the decision was made to try pressure-supported SIMV. This results in a set minimum number of breaths being delivered per minute, which are triggered when the patient spontaneously breathes (Drellich, 2002). The patient can breathe spontaneously between the given breaths when desired, as the operator only controls the minimum respiratory rate

and minute ventilation (Hopper & Powell, 2013). Pressure support can be used alongside SIMV by allowing the patient to trigger and create the breath, but ensures a pre-set pressure and thus a pre-set TV is met by supplementing the attempted breath (Drellich, 2002).

The patient continued to improve, with SpO₂ and end-tidal carbon dioxide (EtCO₂) values improving, and FiO₂ was weaned down to 60% while maintaining oxygen saturation. These values were monitored continually using a multi-parameter monitor unit, and recorded every 5 minutes by the RVN.

The patient was mechanically ventilated for 4 hours. Ideally the patient would have been slowly weaned off the ventilator when the PaO₂ and PaCO₂ were maintained within the normal ranges and ventilatory support could be incrementally decreased without a consequent deterioration in these values. Slowly decreasing the pressure support results in a controlled increase in effort of breathing and indicates that a patient can ventilate and oxygenate sufficiently without support (Drellich, 2002). Unfortunately, in this case, the patient unexpectedly awoke from anaesthesia so ventilation was stopped sooner than the team initially planned. In retrospect, a different maintenance anaesthetic should have been used as it has been shown that patients with more severely diseased lungs require a higher depth of anaesthesia to be comfortable, compared to those with neuromuscular or tracheostomy tubes (Drellich, 2002).

Ventilator care

While the patient was ventilated, nursing interventions were planned and implemented according to the practice's standard operating procedure. This can be split into seven sections: oral care; airway care; intravenous catheter care; eye care; urinary care; gastrointestinal care; and recumbency (**Table 1**).

Table 1. Summary of ventilator nursing care, outlining five of the seven sections.

Airway management	Oral care	Eye care	Urinary care
Every 4 hours: <ul style="list-style-type: none"> • Check cuff, deflate and move slightly before re-inflating • Suction, if required, using closed system to reduce contamination Every 24 hours: <ul style="list-style-type: none"> • Consider changing endotracheal tube 	Every 4 hours: <ul style="list-style-type: none"> • Clean and reposition SpO₂ probe • Inspect tongue for ranula formation • Inspect for ulceration • Clean and reposition mouth gag, if using • Cleanse oral cavity with chlorhexidine solution and suction oral cavity 	Every 2 hours: <ul style="list-style-type: none"> • Clean with sterile saline • Apply lubricant Every 24 hours: <ul style="list-style-type: none"> • Examine eye, stain with fluorescein for ulceration 	Every 4 hours: <ul style="list-style-type: none"> • Palpate and express, if short-term ventilation Place indwelling urinary catheter if ventilating for >24 hours
Recumbency			
Every 4 hours: <ul style="list-style-type: none"> • Passive range of motion exercises on limbs • Turn from lateral > sternal > lateral recumbency • If sternal must be maintained, turn hips lateral to lateral • Examine for decubital ulcers 			

The interventions were performed to prevent ventilator-associated pneumonia, corneal ulcer formation, the development of oral ulcers, glossal swelling and pooling of secretions, and regurgitation in the oral cavity (Donovan, 2017) – as well as preventing urine scalding and decubital ulcer formation (Donovan, 2017). The patient was maintained in a neutral horizontal position on a well-padded orthopaedic mattress, with limbs supported using blankets. If the patient were to have been anaesthetised longer, enteral feeding may have been required.

Oxygen therapy

Following recovery, oxygen supplementation was provided via nasal oxygen prongs, which the patient tolerated well initially. Nasal oxygen prongs are thought to deliver an FiO_2 of 0.4 (Waddel & King, 2018). The patient was positioned in sternal recumbency with the neck extended to improve comfort and aid lung expansion.



The oxygen was humidified using a bubble chamber filled with distilled water, to prevent desiccation of the airways. Waddel and King (2018) suggest that long-term oxygen therapy can damage the lungs, as the toxic metabolites of oxygen cause inflammation. This was prevented by minimising the FiO_2 used to maintain the patient at a stable level. The patient's respiratory rate, effort, heart rate and pulse rate, mucous membrane colour and capillary refill time were continually observed, while also monitoring arterial blood gases and utilising a pulse oximeter.

The pulse oximetry was used as it is a non-invasive method that is easy to use, cost effective and continual. Pulse oximetry measures the amount of oxygen attached to the haemoglobin (SpO_2). However, this value does not have a linear relationship with the PaO_2 (Waddel & King, 2018). Haemoglobin should normally be saturated with >95% oxygen. However, haemoglobin becomes almost fully saturated at a PaO_2 of 80 mmHg and, at this point, the curve plateaus and, despite a higher PaO_2 , the SpO_2 cannot be further increased. This curve may be altered in critically ill patients due to alterations in pH, temperature and PCO_2 (Farrell et al., 2019).

On reflection, further arterial blood gas analysis may have been beneficial in the recovery period. A study by Farrell et al (2019) found that pulse oximetry was not a clinically suitable surrogate for arterial blood gas analysis in dogs that are breathing room air, but was more useful in dogs undergoing mechanical ventilation. In both control groups, the sensitivity of SpO_2 to detect hypoxaemia was poor (Farrell et al., 2019). Waddel and King (2018) suggest that any condition where there is a diminished blood flow to tissues, such as hypotension or shock, can affect the accuracy of pulse oximetry.

The patient's SpO_2 overnight was between 93% and 95% when receiving oxygen via the nasal prongs. Numerous oxygen tests were performed where the patient was slowly weaned from the oxygen support to see if he could maintain his own oxygenation. Initially, the patient's SpO_2 depleted to 85% after 5 minutes without oxygen therapy. After 24 hours, the SpO_2 without oxygen supplementation gradually improved to 98%. At times, there were lower anomalous results that were assumed to be attributable to patient movement as the puppy was becoming very lively and playful.

Medications

Due to the excitable nature of the patient, sedative drugs were used during the treatment to reduce excitability and consequent respiratory distress. Butorphanol (Torbugesic, Zoetis UK Limited) at 0.1 mg/kg IV was used as required, alongside acepromazine (Acecare, Animalcare Limited) 0.01 mg/kg IV. The patient was maintained in a sedated manner for 24 hours to aid in the tolerance of the nasal prongs and to reduce stress. An important consideration for the use of these sedatives is that neither has a direct reversal agent so constant monitoring in an intensive care unit (ICU) is recommended (Tong & Gonzalez, 2020).

Diuretic use in non-cardiogenic oedema is controversial, with limited evidence for its use. During the stabilisation, while initiating ventilation, a single dose of furosemide (Dimazon, MSD Animal Health) at 5 mg/kg IV was administered. Studies suggest the use of diuretics can reduce the intravascular volume and modify Starling's equation in favour of intracapillary filtration and oedema resolution (Louis and Fernandes, 2002; Udeshi, Cantie

and Pierre, 2010). On reflection, this resulted in minimal improvement in the patient's ventilation parameters, despite an initial improvement in pulmonary auscultation. Therefore, due to the limited evidence of its use in non-cardiogenic oedema, the use of furosemide could have been avoided in this case.

Pulmonary oedema within bronchi results in increased turbulence, which can induce bronchoconstriction of the airways (Krodel et al., 2010). Bronchodilators such as terbutaline and salbutamol are both beta-agonists and can be considered for use in patients with lower airway disease (Tong and Gonzalez, 2020). In this patient, terbutaline was started at dose of 0.01 mg/kg subcutaneously BID for 4 days during hospitalisation. Salbutamol is another bronchodilator and is available as an aerosolised drug. The use of beta-agonists in pulmonary oedema is controversial, with previous studies indicating the use of aerosolised salbutamol. However, more recent studies have found beta-agonists to be detrimental (Smith et al., 2012). Due to the severity of the patient's clinical signs, a single dose was trialled but discontinued as the patient did not tolerate administration.

Prior to arrival at the clinic, the patient was administered a single dose of dexamethasone. Glucocorticoid use is also controversial and although there are studies showing dexamethasone use in humans is associated with a significant reduction in mortality, a recent meta-analysis of the data indicates further studies are required to confirm the safety of its use in ARDS patients (Zayed et al., 2020).

Nursing care

The patient was monitored closely in the ICU, which was always staffed by an RVN or veterinary surgeon. The patient received one-to-one care from an RVN or intern at all times. Kirby's Rule of 20 (**Figure 1**) was utilised to ensure that all the patient's requirements were addressed (Haskey, 2015). By considering all items, the team could assess the patient holistically while also implementing critical thinking skills (Waxman, 2020).

Due to the dynamic nature of the disease, close monitoring was vital to recognising changes that were indicative of deterioration and allow rapid intervention. Potential complications for the patient were identified and recorded, in order to tailor observations accurately. All treatments, procedures and observations were written accurately, to monitor ongoing trends.

Buddy was a demanding case that required a team effort with frequent reassessment. It was helpful to refer to the nursing process using the cyclical model of Evaluation > Assessment > Planning > Implementation (Haskey, 2015). This allowed the team to move through the stages continuously initially, then prolonging to every 30 minutes, then 60 minutes as the patient stabilised.

KIRBY'S RULE OF 20 CHECKLIST

1. Fluid balance
2. Albumin and oncotic pull
3. Electrolyte and acid-base
4. Mentation
5. Heart rate, rhythm and contractility
6. Blood pressure
7. Body temperature
8. Oxygenation and ventilation
9. Red blood cells and haemoglobin
10. Coagulation cascade
11. Renal function
12. Gastrointestinal motility and integrity
13. Nutrition
14. Glucose
15. Immune status and antibiotics
16. Wound healing and bandages
17. Drug doses and metabolism
18. Pain control
19. Nursing care
20. Tender loving care

Figure 1. Kirby's Rule of 20 checklist (Waxman, 2020).

Over the period of hospitalisation, serial oscillometric blood pressure monitoring helped to quickly identify hypertension or hypotension. Simultaneously, the cardiovascular system was further assessed by checking the heart rate and pulse quality, as critical illness often places significant demands on the cardiovascular system (Waxman, 2020). Hypotension can indicate circulatory compromise and is common in patients pre-arrest (Latimer-Jones, 2020). It is reflective of the cardiac output and the perfusion to tissues (Latimer-Jones, 2020). A mean blood pressure of >60 mmHg is essential for maintaining organ perfusion (Latimer-Jones, 2020). The patient's blood pressure remained within normal limits throughout hospitalisation. While this method of monitoring blood pressure gave indications of trends, using direct blood pressure measurements may have given more accurate results.

When recovered from anaesthesia, the patient's level of consciousness was assessed by observing demeanour and responsiveness to stimuli in the environment. The patient always remained alert.

Fluctuations in fluid balance can be responsible for weight alterations (Latimer-Jones, 2020). The patient

was weighed on admission and then every 6–8 hours afterwards, with no changes noticed. Temperature was also monitored frequently. It was essential to identify hyperthermia as this may cause excessive panting, which could influence monitoring. Latimer-Jones (2020) advises that a temperature of 40°C is a concern and anything over 42°C is a risk to life.

There was an extensive amount of information that needed to be passed between the team. A verbal cage-side handover occurred between shifts, which aimed to highlight the patient's problems and nursing considerations. Details of improvements, deteriorations, changes to the treatment plan, and diagnostics were discussed.

Discharge

The patient responded well to treatment. Similar to the case described by Louro et al. (2019), the patient was considerably brighter and more alert within 16 hours of the initial clinical signs. The respiratory rate stabilised at 30 breaths per minute, with no cough or stertor detected. The team made decisions together, utilising individual team-member skills and specialisms. The verbal handovers and written notes ensured that important information was passed between teams. In the future, written handovers for complex ICU patients may improve communication as there is a written record to refer to.

On reflection, the patient may have benefited from a longer period of ventilation under TIVA to maximise the ability to ventilate and oxygenate without support, and reduce the subsequent requirement of nasal oxygen therapy, leading to a potentially faster recovery.

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Reflective professional development notes. To access hyperlinks to the references, scan the QR code on page 3.

Part 1

Part 2