

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

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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 (${\rm FiO_2}$) 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 cmH₃O, 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 (PaO, 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 (FiO₃ 70%, PaO, 61 mmHg) and ventilation (PaCO, 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 preset pressure and thus a pre-set TV is met by supplementing the attempted breath (Drellich, 2002).

The patient continued to improve, with SpO_2 and endtidal 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.

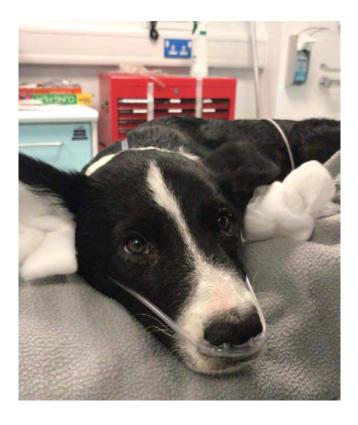
Airway management	Oral care	Eye care	Urinary care	
 Every 4 hours: Check cuff, deflate and move slightly before re-inflating Suction, if required, using closed system to reduce contamination Every 24 hours: Consider changing endotracheal tube 	 Every 4 hours: Clean and reposition SpO₂ probe Inspect tongue 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: Clean with sterile saline Apply lubricant Every 24 hours: Examine eye, stain with fluorescein for ulceration 	 Every 4 hours: Palpate and express, if short-term ventilation Place indwelling urinary catheter if ventilating for >24 hours 	
Recumbency				
Every 4 hours:				

- 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 ${\rm FiO_2}$ 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 FiO₂ 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 (${\rm SpO}_2$). However, this value does not have a linear relationship with the ${\rm PaO}_2$ (Waddel & King, 2018). Haemoglobin should normally be saturated with >95% oxygen. However, haemoglobin becomes almost fully saturated at a ${\rm PaO}_2$ of 80 mmHg and, at this point, the curve plateaus and, despite a higher ${\rm PaO}_2$, the ${\rm SpO}_2$ cannot be further increased. This curve may be altered in critically ill patients due to alterations in pH, temperature and PCO₂ (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 SpO₂ 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 SpO₂ 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 SpO₂ depleted to 85% after 5 minutes without oxygen therapy. After 24 hours, the SpO₂ 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.

REFERENCES

- Barker, M. (2015) Intermittent positive pressure ventilation: basic principles. Veterinary Nursing Journal. 30 (2), 42–46.
- Brower, R.G. (2001) Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. New England Journal of Medicine. 342 (18), 1301–1308.
- Donovan, S. (2017) Nursing care for the mechanically ventilated patient. Veterinary Nursing Journal. 32 (9), 257–259.
- Drellich, S. (2002) Principles of mechanical ventilation. Veterinary Clinics of North America: Small Animal Practice. 32 (5), 1087–1100.
- Farrell, K., Hopper, K., Cagle, L. & Epstein, S. (2019) Evaluation of pulse oximetry as a surrogate for PaO2 in awake dogs breathing room air and anesthetized dogs on mechanical ventilation. Journal of Veterinary Emergency and Critical Care. 29 (6), 622–629.
- Haskey, E. (2015) Nursing the critical patient: Part 1. Veterinary Nursing Journal. 30 (1), 16–21.
- Hopper, K. & Powell, L. (2013) Basics of mechanical ventilation for dogs and cats. Veterinary Clinics of North America: Small Animal Practice. 43 (4), 955–969
- Krodel, D.J., Bittner, E.A., Abdulnour, R., et al. (2010) Case scenario: Acute postoperative negative pressure pulmonary edema. Anesthesiology. 113 (1), 200–207.
- Latimer-Jones, K. (2020) How to detect the subtle changes of early deterioration. The Veterinary Nurse. 11 (7), 325–330.
- Louis P.J. & Fernandes, R. (2002) Negative pressure pulmonary edema. Oral Surgery, Oral Medicine, Oral Patholology, Oral Radiology, and Endodontics. 93 (1), 4–6.
- Louro, L.F., Raszplewicz, J., Hodgkiss-Geere, H. & Pappa, E. (2019) Postobstructive negative pressure pulmonary oedema in a dog. Veterinary Record Case Reports. 7 (3), e000892.
- Oura, T., Rozanski, E.A., Buckley, G. & Bedenice, D. (2012) Low tidal volume ventilation in healthy dogs. Journal of Veterinary Emergency and Critical Care. 22 (3), 368–371.
- Rozanski, E. (2015) Oxygenation and ventilation. Veterinary Clinics of North America: Small Animal Practice. 45 (5), 931–940.
- Smith, F.G., Perkins, G.D., Gates, S., et al. (2012) Effect of intravenous -2 agonist treatment on clinical outcomes in acute respiratory distress syndrome (BALTI-2): a multi-centre, randomised controlled trial. The Lancet. 379 (9812), 229–235.
- Sumner, C. & Rozanski, E. (2013) 'Management of respiratory emergencies in small animals', Veterinary Clinics of North America Small Animal Practice. Elsevier Inc. 43 (4), 799–815. DOI: 10.1016/j.cvsm.2013.03.005.
- Tarry, D. & Powell, M. (2017) 'Hypoxic pulmonary vasoconstriction', BJA Education. 17 (6), 208–213. DOI: 10.1093/bjaed/mkw076.
- Tong, C.W. & Gonzalez, A.L. (2020) Respiratory emergencies. Veterinary Clinics of North America: Small Animal Practice. 50 (6), 1237–1259.
- Udeshi, A., Cantie, S.M. & Pierre, E. (2010) Postobstructive pulmonary edema. Journal of Critical Care. 25(3), 538.e1–538.e5.
- Waddell, L.S. & King, L.G. (2018) General approach to respiratory diseases. In: King, L..G. & Boag, A. (eds.) BSAVA Manual of Canine and Feline Emergency and Critical Care, 3rd edition Gloucester British Small Animal Veterinary Association, 93–122.
- Waxman, C. (2020) Kirby's rule of 20: the veterinary nurse's critical patient checklist part 1. The Veterinary Nurse. 11 (6), 270–275.
- Zayed, Y., Barbarawi, M., Ismail, E. (2020) Use of glucocorticoids in patients with acute respiratory distress syndrome: a meta-analysis and trial sequential analysis. Journal of Intensive Care, 8, 43.

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