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CASE REPORT

Companion or pet animals

Anaesthetic and perioperative management of a dog with biventricular congestive heart failure and advanced second-degree atrioventricular block

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Abstract

A 7-year-old male neutered Cocker Spaniel presented with a 6-hour history of severe tachypnoea. Cardiac evaluation revealed advanced second-degree atrioventricular block (2AVB) and biventricular congestive heart failure (CHF). Due to concerns of impending respiratory arrest, mechanical ventilation (MV) and emergency transvenous pacemaker implantation were planned. General anaesthesia was achieved using methadone premedication, midazolam and etomidate induction and isoflurane in oxygen maintenance. On intubation, large volumes of serosanguinous fluid poured from the airway with haemoglobin oxygen saturation (SPO_2) reading 10%. Suctioning of the airway and initiation of MV improved SPO_2 to 85%. Temporary cardiac pacing was achieved with transthoracic pacing pads. Following uneventful pacemaker implantation, the dog continued on MV for 12 h with gradual resolution of pulmonary oedema assessed using serial thoracic ultrasound. The dog was successfully discharged 24 h later. This report highlights anaesthetic considerations and management of a critically ill bradycardic dog, with biventricular CHF.

KEYWORDS
anaesthesia, cardiology, critical care, heart failure, ventilation

BACKGROUND

Permanent pacemaker implantation has become common practice in veterinary medicine for the treatment of symptomatic bradyarrhythmias, including advanced second-degree atrioventricular block (2AVB), third degree AVB (3AVB), atrial standstill and sick sinus syndrome.[1–3] In dogs, a transvenous approach to pacemaker lead placement is often preferred. In this approach the pacing lead is inserted into the right ventricle via the right jugular vein. An epicardial approach to pacing lead placement via thoracotomy or laparotomy is an alternative, but less desirable option for pacemaker lead placement.[4]

AVB describes impaired electrical impulse conduction through the AV node. Using surface electrocardiography (ECG), the AVB can be further classified into three categories: first degree, second degree and third degree (complete) AVB. First degree AVB (1AVB) describes an ECG rhythm in which the *p* wave is always followed by a QRS complex but with a prolonged P-R interval, representing the delay in electrical conduction through the AV node. Second degree AVB describes an ECG rhythm in which *p* waves are sometimes not followed by a QRS complex, representing intermittent stopping of conduction by the AV node. Second degree AVB can be further classified as Mobitz type I, II, 2:1 and advanced 2AVB, depending on the variation in the P-R interval and conduction ratio (number of *p* waves to QRS complexes). Lastly, 3AVB describes an ECG rhythm in which *p* waves and QRS complexes are occurring unrelated to one another (known as AV dissociation) and represents complete cessation of electrical conduction through the AV node. The resultant ventricular escape rhythm which occurs during advanced 2AVB or 3AVB prevents cardiac arrest and in many instances prevents a loss of consciousness. However, the slow escape rhythm (bradyarrhythmia) can have a negative impact on cardiac output (CO) and blood pressure (BP) resulting in clinical signs including depressed mentation, lethargy, and congestive heart failure (CHF). Concomitant cardiac disease may result in a more severe presentation.[5]

This case report describes the successful management of a dog with severe biventricular CHF and advanced 2AVB, with a focus on anaesthetic considerations and the challenges encountered throughout treatment. Three main anaesthetic considerations are discussed, in relation to both advanced 2AVB and CHF: 1) *Hypoxaemia and hypoventilation*: oxygen supplementation should be provided immediately for patients

showing signs of respiratory distress. A high fraction of inspired oxygen (FiO_2) (>95%) and mechanical ventilation (MV) should be provided during anaesthesia, to maintain normoxaemia; 2) C*ardiovascular instability*: Stress should be minimised where possible to reduce the risk of adverse cardiovascular consequences.[6] Electrolyte abnormalities should be identified and treated prior to anaesthesia. Anaesthetic protocol is important to prevent additional reductions in myocardial conduction. A decrease in conduction may further reduce heart rate (HR), and hence decrease CO and BP. 3) *Risk of post-operative complications*: Oxygen supplementation should be continued into the recovery period until the patient can maintain adequate oxygenation when breathing room air. Post-operative MV may be necessary in patients with severe pulmonary oedema.

Further to this, we focus in detail on the use of MV both intra- and post-operatively to maximise ventilatory efficiency, in a patient with CHF and associated pulmonary oedema.

LEARNING POINTS/TAKE-HOME MESSAGES

- Careful consideration of anaesthetic drug choice is very important in high risk patients; this report discusses one appropriate protocol for use in a patient undergoing anaesthesia with cardiac failure secondary to advanced atrioventricular block.
- Patients with pulmonary oedema present a unique set of considerations for anaesthesia.
- Oxygen supplementation, furosemide administration, rapid airway control, airway clearance and mechanical ventilation are of paramount importance in the anaesthetic success of cases with pulmonary oedema.
- While hypoxia is a highly likely complication under general anaesthesia, mechanical ventilation can be used to somewhat mitigate this issue.
- Short-term, post-operative mechanical ventilation was a useful tool allowing time for resolution of cardiogenic pulmonary oedema in the dog.

CASE PRESENTATION

A 7-year-old male neutered Cocker Spaniel, weighing 20.5 kg, body condition score 5 out of 9, presented with a 6-h history of tachypnoea. The dog also had a 3-day history of lethargy, inappetence and intermittent vomiting. No history of cardiac disease was reported. On presentation, the patient was obtunded, non-ambulatory and tachypnoeic with a severe increase in respiratory effort. Physical examination revealed pale mucus membranes with a prolonged capillary refill time. Cardiac auscultation revealed no murmur, an irregular cardiac rhythm and an HR of 28 beats per minute (bpm) with strong, synchronous femoral and metatarsal pulses. Thoracic auscultation revealed harsh lung sounds bilaterally. Abdominal distension was noted with a positive fluid ballottement. No further abnormalities were detected on physical examination.

INVESTIGATIONS

ECG revealed advanced 2AVB (a conduction ratio of greater than 2:1 i.e., two or more *p* waves are not conducted in sequence) with an atrial rate of 158 bpm and ventricular escape rate of 27 bpm (Figure 1). An emergency point of care ultrasound was performed (Z5 with 35C20EA convex probe, Mindray Bio-Medical Electronics Co. Shenzhen, China) revealing diffuse B-lines, suggestive of pulmonary oedema, marked peritoneal effusion and dilation of all four cardiac chambers. Left atrial to aortic ratio (LA:Ao) was 2.3 (normal < 1.7).[7] Venous blood gas, electrolyte and metabolite analysis (ABL800 FLEX, Radiometer Limited, Crawley, England) revealed the following changes: hypercarbia (venous partial pressure of carbon dioxide: 57.4 mm Hg, reference range: 37–47 mm Hg), hypoxaemia (venous partial pressure of oxygen: 34.7 mm Hg, reference range: 45—65 mm Hg), increased creatinine (creatinine 225 umol/L, reference range: 50–140 umol/L), mild hyperkalaemia (potassium 4.8 mmol/L, reference range: 3.6–4.6 mmol/L) and hyperlactataemia (lactate 5.9 mmol/L, reference range: 0.6–2.5 mmol/L). Blood pH was not provided at this time due to machine error. Due to the instability of the patient, further diagnostic tests (including an atropine response test) were not performed at this stage.

DIFFERENTIAL DIAGNOSIS

Treatment

Soon after presentation, flow-by oxygenation was initiated, and an 18-gauge intravenous catheter (Jelco; Smith's Medical, UK) was placed in the right lateral saphenous vein. The patient was administered a 2 mg/kg furosemide (Dimazon; MSD Animal Health, UK) bolus intravenously (IV) and placed in an oxygen kennel, set to provide an FiO_2 of 60%. This was immediately followed with a furosemide continuous rate infusion at 0.5 mg/kg/h IV. Despite initial therapy with oxygen and furosemide, the patient remained hypercarbic and hypoxaemic on venous blood gas analysis. Due to the severity of CHF and the concern for patient exhaustion leading to respiratory arrest, emergency general anaesthesia for MV and

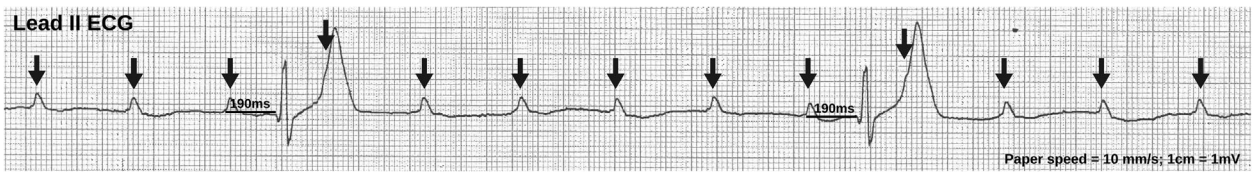

FIGURE 1 Lead II ECG displaying advanced second-degree atrioventricular block. The *p* waves are annotated (arrow). The ventricular escape rate was 27 beats per minute. Note some hidden *p* waves in the T wave. The P-R interval is fixed at 190 ms, which differentiates this rhythm from third degree atrioventricular block

FIGURE 2 Transthoracic pacing pads (Quick-Combo RTS Pediatric Pacing/Defibrillation/ECG Electrodes Radiotransparent System, Physio-Control, Inc., Redmond, USA) were placed across the heart in contact with the skin of the dog and taped around the thorax

pacemaker implantation were performed, with MV continued post-operatively.

The dog was assigned an American Society of Anesthesiologists score of 4(E).[8] Prior to anaesthesia, transthoracic pacing pads (Quick-Combo RTS Pediatric Pacing/Defibrillation/ECG Electrodes Radiotransparent System, Physio-Control, Inc., Redmond, USA) were placed across the patient's thorax and secured using elastoplast (Figure 2). The patient was pre-medicated with 0.2 mg/kg methadone (Comfortan: Dechra Veterinary Health, UK) IV, 5 minutes prior to anaesthetic induction. Induction of anaesthesia was performed using 0.2 mg/kg midazolam (midazolam, Hameln Pharmaceuticals Ltd., UK) IV and 2 mg/kg etomidate (Hypnomidate, Piramal Critical Care, UK) IV. The patient was intubated with an 8 mm polyvinylchloride, cuffed endotracheal tube (ETT). The ETT cuff was inflated with a 5 ml syringe, with inflation pressure estimated by palpation of the pilot balloon. The patient was immediately connected to an anaesthetic machine with built in electronic ventilator (Wato EX-30, Mindray Bio-Medical Electronics Co. Shenzhen, China) via a circle breathing circuit. Anaesthesia was maintained with sevoflurane (initial vaporiser setting 2.5%) delivered in greater than 95% oxygen. To provide neuromuscular block and hence prevent muscle contraction during external pacing, 0.2 mg/kg atracurium (atracurium besilate, Hameln Pharmaceuticals Ltd., UK) was given slowly IV. No further atracurium was required. Transthoracic temporary pacing was initiated at 80 bpm, via the pre-placed pacing pads, using an external defibrillator device (BeneHeart D3, Mindray Bio-Medical Electronics Co. Shenzhen, China). On securing the ETT to the patient and lowering the head, large volumes of serosanguinous fluid poured from the airway, filling the ETT, spirometry loop, side-stream capnography line, capnograph water trap (Figure 3) (Datex S5; GE Healthcare, Amersham, UK) and breathing circuit. A pulse oximetry probe (Datex S5; GE Healthcare, Amersham, UK) had been placed on the patient's tongue revealing a haemoglobin oxygen saturation (SPO_2) of 10% (although pulse oximetry is inaccurate outside the 80–95% range[9]) with an appropriate plethysmography trace. The patient's airway was immediately suctioned and pressure controlled MV initiated with the following settings: peak inspiratory pressure (PIP) of 22 cmH_2O achieving a tidal volume of between 160 and 240 ml; positive end expiratory pressure (PEEP) of 7 mm Hg; inspiratory:expiratory ratio of 1:2.5; respiratory rate (RR) of 26 breaths per minute (brpm). RR was increased to 30 brpm after 1 hour of MV for the remainder of anaesthesia (due to hypoventilation), in an attempt to maintain end-tidal carbon dioxide ($ETCO_2$) between 35 and 45 mm Hg. Haemoglobin oxygen saturation rose to 84% within 15 min and remained between 83% and 95% for the remainder of the anaesthetic. Unfortunately, it was not possible to take blood gas samples during anaesthesia to confirm that the SPO_2 was accurate.

Anaesthetic monitoring included ECG, HR, SPO_2, oscillometric non-invasive BP, capnography, spirometry (Datex S5; GE Healthcare, Amersham, UK) and Doppler BP (Parks Medical Electronics, Inc., USA). Both the audio from the Doppler BP machine and plethysmograph waveform were used to monitor the paced ('capture') beats, as the loss of capture would result in loss of pulse, and hence a loss of both the Doppler sound and plethysmograph waveform. Monitoring of neuromuscular blockade was not utilised in this case for two reasons: firstly, the emergency nature of the case prevented set up of monitoring prior to surgery; and secondly, the patient was to remain under anaesthesia for MV post-operatively, allowing time for adequate metabolism of the atracurium administered. Intravenous fluid therapy was omitted for this case, due to the already present fluid overload. Continuous intravenous infusions of 0.5 mg/kg/h furosemide IV and 0.2 μg/kg/min fentanyl (Fentadon, Dechra Veterinary Products, UK) were initiated. A loading dose of 2 μg/kg fentanyl was administered IV on initiation of the fentanyl infusion. The sevoflurane vaporiser setting was reduced to achieve an end-tidal anaesthetic concentration of 1.6%, which remained constant thereafter throughout the anaesthetic.

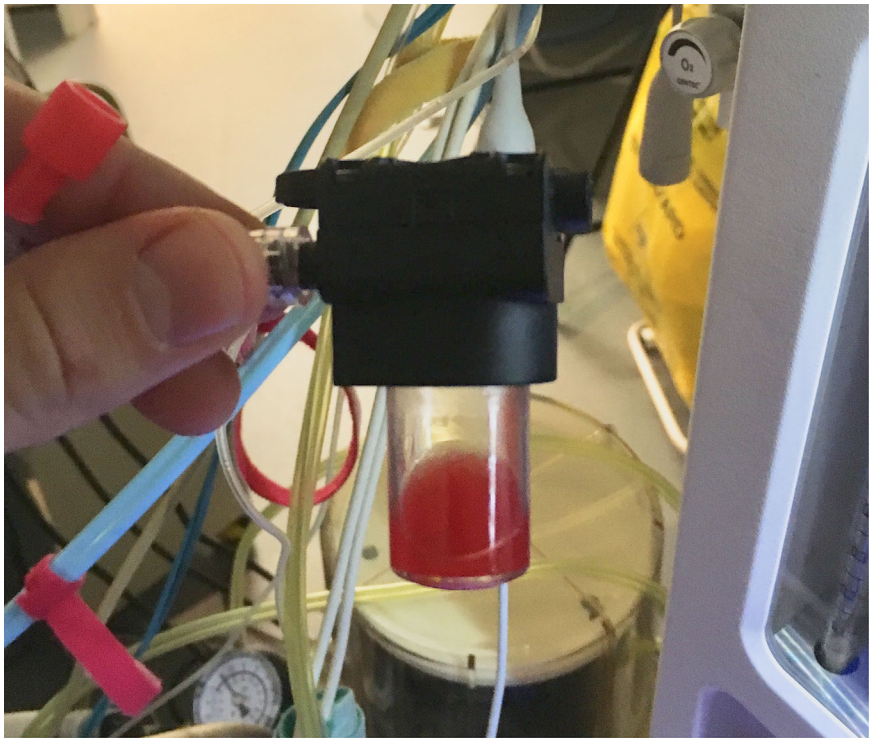

FIGURE 3 Serosanguinous pulmonary oedema fluid collected in the water trap of the S5 Datex multiparameter monitor (GE Healthcare, Amersham, UK). This fluid was accidentally sampled from the spirometry connector (D-lite) via the capnography line and trapped here to protect the capnography module of the monitor from damage

A permanent pacemaker lead was implanted in the right ventricle, using fluoroscopic guidance. Following a successful pacemaker implantation, the transthoracic pacing was turned off, and the dog was paced using the Reply 200SR pulse generator (MicroPort CRM, Clamart, France). The SPO_2 remained between 83% and 95%, with HR remaining between 90 and 100 bpm depending on pacing rate, and normotension maintained throughout the anaesthetic period.

Following the procedure, the patient was transferred to the intensive care unit (ICU) for ongoing MV. Continuous infusions of 0.1 mg/kg/min propofol (Lipurovet, Virbac Ltd., UK), 0.2 mg/kg/h midazolam and 3 μg/kg/h fentanyl were administered IV to facilitate MV. Furosemide was discontinued as the cause of CHF was thought to be primarily related to the advanced 2AVB. MV was achieved using an electronic ICU ventilator (SV300 Ventilator, Mindray Bio-Medical Electronics Co. Shenzhen, China). Pressure controlled, synchronised intermittent-mandatory ventilation (SIMV) was initiated with the following settings: FiO_2 60%, PIP of 13 cmH_20 delivering a tidal volume of approximately 200 ml, PEEP of 5 cmH_2O, a minimum ventilation frequency (fSIMV) of 20 brpm and a pressure trigger of 2 cmH_20.

Over the subsequent 12 hours, PIP was gradually reduced to 6 cmH_20, PEEP to 2 cmH_2O and fSIMV to 4 brpm, although the patient's RR was 24 brpm prior to stopping ventilation. Resolution of pulmonary oedema was observed using 2 hourly thoracic ultrasound examination, as well as SPO_2 monitoring using pulse oximetry. Haemoglobin oxygen saturation was at least 95% during SIMV, dropping to 89% temporarily following reduction in fSIMV. The FiO_2 was gradually decreased from 60% to 25%, and the patient was weaned off from the ventilator. On recovery the patient was kept in an oxygen kennel providing an FiO_2 of 60%. The SPO_2 remained above 95%, and the patient was removed from the oxygen kennel as soon as he was ambulatory. RR and effort remained normal. The increased serum creatinine noted prior to surgery had resolved by this time.

OUTCOME AND FOLLOW-UP

The patient was successfully discharged 24 hours after the discontinuation of MV. A follow up cardiology appointment at 4 weeks following discharge showed that the dog was back to his normal self. Clinical examination was unremarkable. Brief echocardiography revealed a significant reduction in cardiac size (LA:Ao 1.7) and appropriate positioning of the pacing lead with no evidence of intracardiac thrombus. Pacemaker interrogation showed improved AV nodal function, and the dog was pacing only 49% of the time. The dog was in sinus rhythm throughout the repeat cardiac assessment. At the time of writing this case report (20 months post-pacemaker implantation), the dog is clinically well and on no medication. The resolution of CHF and a significant reduction in left and right cardiac dimensions at recheck, would suggest that the bradycardia was the main cause of CHF in this case.

DISCUSSION

We describe the successful management of a dog with severe biventricular CHF and advanced 2AVB, with focus on anaesthetic considerations and the challenges encountered throughout the peri-anaesthetic period. We also describe the use of MV post-operatively in this patient to facilitate resolution of pulmonary oedema.

The case presented here occurred outside normal business hours. In a study by Ward et al transvenous pacemakers placed outside normal business hours in patients with advanced AVB were associated with an increased risk of complications such as lead dislodgement or infection.[10] Brodbelt showed that induction of anaesthesia between 5 PM and 8 AM carries an 11.8 times increased risk of mortality in dogs compared to induction of anaesthesia between 8 AM and 5 PM.[11] Careful patient management is therefore of utmost importance to optimise clinical outcome.

Three main anaesthetic considerations were considered in this case: 1) *Hypoxaemia and hypoventilation*: Oxygen supplementation should be provided immediately for patients showing signs of respiratory distress. In a case of CHF and pulmonary oedema, a high FiO_2 (>95%) and MV should be provided during anaesthesia to maintain normoxaemia. Hypoxaemia is likely to worsen due to the adverse cardiorespiratory effects of anaesthetic drugs administered to the patient. Following presentation and initial stabilisation, the dog was placed in an oxygen kennel to prevent the stress associated with mask oxygenation; 2) *Cardiovascular instability*: Stress should be minimised where possible to reduce the risk of adverse cardiovascular consequences such as an increase in sympathetic nervous system activity, resulting in arrhythmias and an increase in anaesthetic drug consumption.[6] Methadone was administered IV as a premedicant to provide mild sedation and hence facilitate handling prior to induction of anaesthesia. Handling was kept to a minimum, and a calm, quiet environment was maintained where possible. Electrolyte abnormalities should be identified and treated prior to anaesthesia. Alterations in potassium and calcium could cause further rhythm disturbances. In this case, venous blood analysis showed no significant electrolyte abnormalities requiring immediate treatment. Anaesthetic protocol is important to prevent additional reductions in myocardial conduction. A decrease in conduction may further reduce HR, and hence decrease CO and BP. This is discussed further below. 3) *Risk of post-operative complications*: Oxygen supplementation should be continued into the recovery period until the patient can maintain acceptable oxygenation (assessed using SPO_2 or arterial partial pressure of oxygen [PaO_2] with arterial blood gas analysis) when breathing room air. Post-operative MV may be required in patients with severe pulmonary oedema, to maintain oxygenation and to allow time for the oedema to resolve.

A retrospective study by Sanchis-Mora et al reported the most commonly used anaesthetic protocols and complications of pacemaker implantation, in 57 dogs over a 10-year period. Premedication agents included pethidine (41/57) methadone (8/57), morphine (2/57) and buprenorphine (1/57). Induction agents included etomidate (47/57) with or without a co-induction agent such as a benzodiazepine, alfaxalone (6/57) or propofol (3/57). Common complications included intraoperative hypothermia (33.3%), bradycardia (12.3%), intraoperative ventricular premature complexes (8.8%), intraoperative hypotension (8.8%), intraoperative hypertension (8.8%) and pulmonary oedema (1.8%), with hypothermia and hypotension the most common complications directly associated with anaesthesia.[12]

For premedication the opioid drug methadone was chosen for its reversible sedation and analgesia, while having little effect on myocardial contractility. Higher doses should be avoided to minimise their effect of increasing vagal tone and hence significantly reducing HR and CO.[13] For induction, etomidate and midazolam were used in combination. Etomidate is often reserved for induction of patients with severe cardiac compromise. In healthy patients, HR, stroke volume, CO, mean arterial pressure and cardiac index are maintained following a single induction dose of etomidate.[14,15] Furthermore, baroreceptor reflexes and sympathetic responses are also maintained after administration of etomidate.[16] This is not true for other commonly available induction agents, and so etomidate was thought to be an appropriate induction agent in this case. Also, arterial pressure is maintained during etomidate anaesthesia induction, in the presence of induced left ventricular dysfunction. However, an increase in afterload can further compromise myocardial performance in patients with left ventricular systolic dysfunction.[17] Etomidate has also been associated with adrenocortical suppression. However, cortisol levels have been shown to remain higher than baseline values, and any suppression has been shown to be transient (2–6 hours) following a single 2 mg/kg etomidate IV bolus. Hence, etomidate is considered a safe induction agent in dogs.[18] Benzodiazepines such as midazolam may decrease the dose of induction agent required[19] and help to prevent the myoclonus seen if etomidate were administered alone. However, benzodiazepines may cause excitation when administered as a premedicant.[20] For this reason, co-administration with an induction agent is preferable. In the reported case, intravenous injection of 2 mg/kg etomidate and 0.2 mg/kg midazolam produced an adequate plane of anaesthesia for endotracheal intubation, connection to the breathing system, subsequent suctioning of the airway and then reconnection to the breathing system, as described above.

Induction of anaesthesia in patients with 2AVB has also been reported using ketamine, propofol and alfaxalone.[20] Ketamine is a dissociative anaesthetic that usually causes an increase in sympathetic activity and hence an increase in HR. In patients with a maximally stimulated sympathetic nervous system however, ketamine may result in significant myocardial depression.[21] In a study comparing alfaxalone to etomidate for anaesthesia induction in dogs, alfaxalone induction resulted in a significant decrease in arterial BP, and significant increases in HR and cardiac index compared to etomidate. Arterial pressure remained within a clinically acceptable range, likely due to the increase in HR that occurs as a compensation for the decrease in systemic vascular resistance, as seen by Rodriguez et al.[14] In this case, the patient's advanced 2AVB would prevent the increase in HR, and hence a decrease in cardiac index would be more likely than the increase seen in healthy dogs. Propofol has been shown to produce a decrease in systolic and mean arterial pressure,[15] as well as a dose-dependent decrease in systolic function, while etomidate did not affect regional myocardial function.[22]

In cases of advanced 2AVB with biventricular CHF, the use of alpha-2 adrenergic receptor agonists and acepromazine are not recommended, due to their significant cardiovascular effects.[23] While etomidate may be the most logical choice, it may not be as widely available as propofol or alfaxalone. Use of propofol or alfaxalone should be carefully considered for the reasons stated above, and their use with a co-induction agent such as midazolam, should be strongly considered to reduce the dose of induction agent required.[19]

Maintenance of anaesthesia was achieved with sevoflurane in greater than 95% oxygen for this case. Fluorinated anaesthetics have a number of cardio-respiratory effects. Sevoflurane for example has been shown to increase HR from baseline at 1, 1.5 and 2 times the minimum alveolar concentration (MAC). As with isoflurane, sevoflurane has also been shown to cause a dose dependent decrease in systemic vascular resistance. In healthy dogs, despite a decrease in stroke index, cardiac index remains unchanged due to the

significant increase in HR.[24] In a patient with AVB as described in this case report, HR is very unlikely to increase secondary to sevoflurane administration, and so cardiac index will undoubtedly decrease. For this reason, transthoracic pacing was initiated immediately following induction of anaesthesia in the present case. A study assessing AV conduction times found that both isoflurane and sevoflurane have less effect on the cardiac conduction system compared to other fluorinated anaesthetics, which may be important for cardiac rhythm stability under anaesthesia.[25] While this may not be relevant for cases of high grade 2AVB or 3AVB, this may be relevant when anaesthetising patients with 1AVB or non-advanced 2AVB. Utilising a fentanyl infusion in this case allowed for a reduction in the sevoflurane MAC required to maintain an appropriate anaesthetic depth, due to its central narcotic effect, subsequently reducing the adverse cardiovascular effects of volatile anaesthesia.

While treatment of cardiogenic pulmonary oedema requires diuresis, MV may be an additional effective therapeutic option in dogs with severe CHF.[26] In humans, non-invasive positive pressure ventilation such as continuous positive airway pressure (CPAP) is recommended for patients with pulmonary oedema resulting in hypoxia, despite medical therapy.[27] It has been suggested since the 1970s that rapid correction of hypoxaemia and acidosis, combined with a reduction in the work-of-breathing and sympathetic stimulation, may increase patient survival.[28] Furthermore, provision of a positive intrathoracic pressure may reduce left ventricular preload and subsequently improve CHF.[29,30] Since the patient described in this report required anaesthesia for pacemaker implantation, pressure controlled MV was utilised as a treatment option. An attempt was made to apply lung protective ventilation strategies. Applying a PEEP of 7 cmH_2O allowed for a reduction in average driving pressure, while achieving a tidal volume of around 10 ml/kg. While this tidal volume is greater than lung protective recommendations in humans (4–8 ml/kg),[31] studies applying these guidelines to dogs have shown that higher tidal volumes are required to achieve acceptable blood gas values in dogs compared to humans.[32] Driving pressure remained lower than the maximum plateau pressure recommended in human lung-protective ventilation strategies (<30 cmH_2O).[31] As described above, the patient in this report required post-operative ventilation due to the presence of pulmonary oedema. While CPAP is recommended as the first line for ventilatory support of humans with cardiogenic pulmonary oedema,[27] this technique is challenging to adapt to animals. CPAP devices have been tested[33] and applied in canine patients for various pathologies, including canine cardiogenic pulmonary oedema.[34] However, due to patient compliance problems and a lack of experience with these devices in UK veterinary practice, this technique is not in widespread use. As the patient reported here was already anaesthetised and intubated, the decision was made to continue pressure-controlled MV post-operatively, despite the availability of CPAP equipment at our institution.

In the ICU, SIMV was applied to this patient. Synchronous intermittent MV is a commonly used MV mode for weaning patients from the ventilator; however evidence for its use in dogs is scarce. It consists of a combination of mandatory and assisted breaths. Positive pressure breaths can be triggered by the patient, when respiratory effort exceeds the trigger value (a set pressure or flow generated by the patient, which triggers an assisted breath). By gradually reducing the number of mandatory breaths over time, the patient can be weaned from the ventilator.[35] Synchronous intermittent MV also allows for weaning by reduction of the peak pressure provided by the ventilator. A number of studies in paediatric patients have identified an improvement in patient-ventilator synchrony when using SIMV, compared to conventional pressure-controlled ventilation strategies.[36] In one study, an improvement in oxygenation was noted for paediatric patients receiving SIMV.[37] Studies comparing ventilatory strategies are lacking in veterinary medicine. While a specific ventilation strategy cannot be recommended, the benefit of simple pressure-controlled ventilation is likely to be adequate for management of cases of pulmonary oedema in dogs. This is highlighted further by Bruniges and Rigotti, who successfully managed a case of pulmonary oedema as an adverse reaction to naloxone administration, using pressure controlled MV.[38]

In this patient, MV was ceased 12 hours after the end of surgery, despite a significantly elevated RR. At this time the dog was breathing with minimal effort, achieving a tidal volume of 9 ml/kg and maintaining a normal $ETCO_2$. At 2 hours prior to stopping ventilation, arterial oxygenation was 149 mm Hg (FiO_2 60%), improving to 280 mm Hg (FiO_2 60%) at time of ventilation cessation. This would equate to a PaO_2/FiO_2 ratio of 248 (mild acute respiratory distress syndrome) increasing to 467 (normal PaO_2/FiO_2 ratio ~400-500 mm Hg).[39] In this case report ultrasound assisted with the identification of pulmonary oedema.[40] Lung ultrasound could also be a useful tool for monitoring of pulmonary oedema in dogs with left-sided CHF.[41] In this case, serial lung ultrasound showed a reduction in the number of B-lines (pulmonary oedema) over time; however a degree of pulmonary oedema was likely still present at the time of weaning this patient from the ventilator. It was believed that in waking the patient and removing the cardio-respiratory effects of the maintenance anaesthesia infusions (fentanyl, midazolam and propofol), further improvements in lung function (i.e., an improvement in functional residual capacity and pulmonary compliance) would be seen. For many animals with pulmonary disease, the anaesthetic recovery period can be difficult. Whether dogs undergo auto-recruitment of atelectic lung regions on recovery from anaesthesia is currently unknown. During recovery, dogs are observed to take 'sigh' breaths. These sigh breaths have been shown in humans to increase functional residual capacity and pulmonary compliance,[42] likely through the opening of collapsed alveoli.[43] While auto-recruitment is not reported in dogs, this phenomenon has been reported in horses.[44] The addition of breath holding is something that the authors believe may also be important in the auto-recruitment of lungs in the dog during the immediate post-anaesthetic period; however this is yet to be studied as it has been in horses.[44]

At the time of recheck, 4 weeks following pacemaker implantation, pacemaker interrogation showed improved AV nodal function, and pacing was occurring only 49% of the time. Regression of AVB has been noted in dogs 1 month following pacemaker implantation. In a study by Santilli et al, 13% of dogs had AVB regression, of which 67% returned to sinus rhythm. Transient AVB in humans occurs as a result of acute myocarditis; however the prevalence of

myocarditis-related AVB in dogs is unknown. Temporary pacing was considered for this case but not chosen as it is impossible to say if the AV node would recover and how long this would take.[45]

In conclusion, we describe the successful management of a critically ill dog with severe biventricular CHF and advanced 2AVB. We highlight the importance of careful anaesthetic planning and the use of MV to achieve a good outcome.

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