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# Anaesthetic management for balloon dilation of cor triatriatum dexter in a dog

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# **Abstract**

A three-month-old female Rottweiler puppy was referred for intravascular correction of a previously identified cor triatriatum dexter. Echocardiography confirmed the presence of a hyperechoic membrane that divided the right atrium into a cranial and caudal chamber. A foramen in this membrane allowed the blood to flow from the caudal to the cranial chamber. Balloon dilation of the defect under transthoracic echocardiographic guidance was scheduled for the following day. The dog was premedicated with 0.5 µg/kg sufentanil and 0.2 mg/kg midazolam administered intravenously. General anaesthesia was induced with 2 mg/kg propofol and maintained with inhaled isoflurane in oxygen; at the same time, a constant rate infusion of 0.5 µg/kg/h sufentanil was administered by means of an infusion pump. Uneventful ventricular and supraventricular tachyarrhythmias developed during the placement of catheters and balloon dilation. At the end of procedure, when the guide wire and balloon catheter were removed, normal sinus rhythm was observed. To the authors' knowledge, no previous reports have described the anaesthetic management of a balloon dilation procedure for cor triatriatum dexter in dogs.

Keywords: Anaesthesia, Arrhythmia, Balloon dilation, Canine, Cor triatriatum dexter, Puppy

# **Background**

Cor triatriatum dexter (CTD) is a rare congenital defect caused by the failure of the right sinus venosus valve to regress during embryogenesis [1]. The persistent membrane causes the division of the right atrium into a cranial and caudal chamber. Anatomic variations of CTD may be distinguished from the location of the inlet of the coronary sinus, which can enter either the cranial or the caudal chambers. The intra-atrial septum may be imperforate or perforate to varying degrees, resulting in hepatic congestion and ascites due to the mechanical obstruction of the blood flow through the caudal vena cava toward the right atrium [1]. Two techniques have been reported to treat CTD in dogs: surgical membrane resection [2–7] or percutaneous balloon dilation [8, 9].

To the authors' knowledge, no previous reports have described the anaesthetic management of a balloon dilation procedure for CTD in dogs. This report discusses the anaesthetic protocol used and the impact of balloon dilation on the cardiovascular system.

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## Case presentation

A three-month-old female Rottweiler puppy, weighing 11.6 kg, was referred to the cardiology service of the Veterinary Teaching Hospital of Perugia University, Italy for further assessment and treatment of CTD. When presented to the referring veterinarian, the dog had a history of gradual abdominal enlargement over the previous weeks. CDT was diagnosed by transthoracic echocardiography and medical treatment included furosemide(2 mg/kg orally twice daily), spironolactone (2 mg/kg orally once daily), and ramipril (0.25 mg/kg orally once daily). On clinical examination, the dog was bright and alert with a heart rate (HR, beats/min) of 136 and a respiratory rate (RR, breaths/min) of 36, but showed poor body condition (body condition score 3/9) and a markedly enlarged abdomen with a fluid wave. Abdominal ultrasound revealed severe ascites associated with evident hepatomegaly and hepatic venous congestion. Abdominocentesis revealed modified transudate ascitic fluid (specific gravity 1020, total protein 2.2 g/dL, poor cellularity). Cardiac auscultation revealed no heart murmur or arrhythmia.

Transthoracic echocardiography<sup>5</sup> showed a thin, hyperechoic membrane that divided the right atrium



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into a cranial and caudal chamber. The tricuspid valve, cranial vena cava inlet, and right auricle were included in the cranial chamber, whereas the caudal chamber included the caudal vena cava inlet and the coronary sinus. A 4.5 mm opening allowed the blood to flow from the caudal to the cranial chamber. A colour Doppler<sup>5</sup> examination showed turbulent flow from the caudal to the cranial chamber of the right atrium across the anomalous membrane, with a peak velocity of approximately 2.2 m/sec.

From these findings, CTD was confirmed and correction of the anomalous membrane by a balloon dilation technique was scheduled for the following day. Hematologic parameters had revealed alterations in both blood count and in biochemical analysis (Table 1).

Prior to the anaesthetic procedure, the two cephalic veins were aseptically catheterized with a 20 gauge cannula. Patient preoxygenation (flow-by: 5 L/min of pure oxygen) was allowed before and after premedication. Pre-anaesthesia included consecutive intravenous (IV) administration of 0.5  $\mu g/kg$  sufentanil and 0.2 mg/kgmidazolam. Five minutes after premedication, general anaesthesia was induced with 2 mg/kg propofol, administered as a single, progressive IV bolus. The dog's trachea was intubated with a 6.5 mm cuffed tube, which was connected to a non-rebreathing system (Bain-type). General anaesthesia was maintained with isoflurane diluted in oxygen (fresh gas flow: 3 L/min); concurrently, a constant rate infusion (CRI) of 0.5 µg/kg/h sufentanil<sup>9</sup> and 5 mL/kg/h of lactated Ringer's solution were administered IV by infusion pumps.

When an adequate depth of anaesthesia was achieved, the dog was positioned in right lateral recumbency for the cardiovascular corrective procedure. The patient was allowed to spontaneously breathe during the entire anaesthetic procedure and did not required artificial ventilation support.

**Table 1** Altered hematologic parameters in a dog undergoing balloon dilation of cor triatriatum dexter

Parameter	Value	Normal range
Hct (%)	28	37–55
Hb (g/dL)	9.84	12-18
RBC (M/μL)	4.68	5.50-8.50
MCV (f/L)	56.5	60-77
RDW (%)	8.6	12–16
TP (g/dL)	3.93	5.5-7.8
Alb (g/dL)	2.5	2.6-3.8
Urea (mg/dL)	49.7	10-45
Ast (U/L)	50	10-40

Hct hematocrit, Hb haemoglobin, RBC red blood cell, MCV mean cell volume, RDW high red blood cell distribution width, TP total protein, Alb albumine, Ast aspartate transaminase enzymes

A 24 gauge cannula was aseptically inserted into the metacarpal artery to measure direct blood pressure. HR, RR, electrocardiogram (ECG), invasive arterial blood pressure (systolic arterial pressure, diastolic arterial pressure, mean arterial pressure (MAP); mmHg), haemoglobin oxygen saturation (SpO<sub>2</sub>, %), end-tidal carbon dioxide partial pressure (EtCO<sub>2</sub>, mmHg), end-tidal isoflurane concentration (EtIso, %), and rectal temperature (T,°C) were continuously monitored and recorded every 5 min. During inflation and deflation of the balloon catheter (BC), the same parameters were recorded every 2 min. The transducer was zeroed at the level of the manubrium of the sternum.

The interventional procedure included percutaneous access from the right femoral vein by the Seldinger technique [10] and the subsequent positioning of two balloon valvuloplasty catheters<sup>20</sup> (first 12 mm and then 22 mm) across the intra-atrial membrane, under transthoracic echocardiography guidance<sup>5</sup>. Ten consecutive hand inflations with a 12 mm  $BC^{20}$  were performed. The dilation procedure was then repeated by using a 22 mm  $BC^{20}$ .

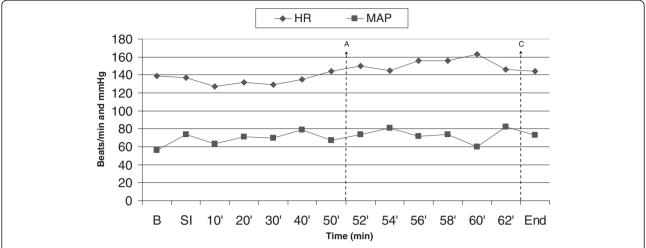
The HR from the beginning to the end of the anaesthesia ranged from 126 to 163 and the MAP from 62 to 82 (Fig. 1). Table 2 shows the values of RR, SpO<sub>2</sub>, EtCO<sub>2</sub>, EtIso, and T. During the interventional procedure, several arrhythmias were noted and recorded (ventricular and supraventricular tachyarrhythmias). Transient supraventricular extrasystoles were observed during the placement of the guide wire and BC<sup>20</sup>, but spontaneously resolved following the withdrawal of the dilation instrumentations from the atrial wall. During balloon inflations (22 mm BC20), ventricular and junctional beats (Fig. 2) and non-sustained supraventricular tachycardia (Fig. 3) occurred, with spontaneous conversion to sinus rhythm after the end of the dilation procedure. These arrhythmias did not show any haemodynamic impact on the arterial pressure.

Table 3 provides the total anaesthesia and cardiovascular procedure time, the total inflation-deflation time of the  $BC^{20}$ , the number of  $BC^{20}$  inflations, and the total dose and CRI time of sufentanil<sup>9</sup>.

The dog was extubated 9 min after the end of the anaesthesia and was able to stand after 14 min. The recovery was uneventful and the patient was hospitalized post-operatively for two days.

# **Conclusions**

Although a definitive conclusion cannot be made from a single case, the balanced anaesthesia used for the three-month-old puppy described in this report assured good patient haemodynamic stability for the safeness of this minimal invasive balloon dilation technique. The side effects that occurred, such as supraventricular extrasystoles,



**Fig. 1** Heart rate (HR, beats/min) and mean arterial pressure (MAP, mmHg) values of the dog undergoing balloon dilation for cor triatriatum dexter at Baseline (**b**, 15 min before the skin incision), at Skin incision (**si**) and every 10 minutes until the start of the balloon inflation-deflation (**a**), when the same parameters are reported every 2 minutes until the end of the procedure (**c**) (End, 15 min after the end of the cardiovascular procedure)

supraventricular tachycardia, and ventricular and junctional beats, did not have any haemodynamic impact and disappeared as soon as the wires were removed from the heart. Antiarrhythmic therapy was therefore not required.

During an interventional procedure, anaesthesia should ensure hypnosis, analgesia, and haemodynamic stability; titration should be easy and reliable in order to cope with rapidly changing loading conditions and intense, but short-lived, haemodynamic disturbances [11]. The anaesthetic drugs should not interfere with the electrophysiological processes in the cardiac conduction system and, above all, anaesthetic recovery should be fast and free of side effects [11]. Furthermore, puppies under anaesthesia are more likely to develop hypotension, hypothermia, hypoglycaemia, and hypoxia [12, 13]. For these reasons, management of the anaesthesia for a paediatric dog undergoing an interventional procedure is extremely critical. In addition to these fundamental principles, anaesthetists dealing with a patient with CTD must also follow the haemodynamic principles

**Table 2** Physiological parameters for a balloon dilation of cor triatriatum dexter in a dog under general anaesthesia

	Baseline	b-CBI C (range)	CBI (range)	End
RR (breaths/min)	26	22-30	23-27	12
SpO <sub>2</sub> (%)	95	92-100	94–95	98
EtCO <sub>2</sub> (mmHg)	38	37–41	39–45	37
Etlso (%)	1.2	1.1-1.2	1.1-1.2	1.3
T (°C)	36.5	35.9–36.5	35.6-35.9	35.2

Baseline 15 min before the skin incision, End 15 min after the end of the procedure, CB catheter balloon, b-CBI form Baseline to the beginning of CB inflation, CBI during CB inflation, RR respiratory rate, SpO<sub>2</sub> haemoglobin oxygen saturation, EtCO<sub>2</sub> end-tidal carbon dioxide partial pressure, EtIso end-tidal isoflurane concentration, T rectal temperature

used to manage patients with tricuspid stenosis: avoid tachycardia, avoid hypotension by maintaining euvolemia, and avoid increases in pulmonary vascular resistance [14].

The veterinary literature lacks information about the anaesthesia strategy, the interactions of drugs and the repercussions for the patient, and the possible cardiovascular impact of the drugs chosen in dogs affected by CTD who are undergoing balloon dilation of the defect. Some reports indicate only which anaesthetic drugs were used [8, 9, 15, 16]. In our case, the dog was sedated with a combination of a potent opioid (sufentanil) and midazolam in order to preserve the cardiovascular function of the patient [17, 18]. Premedication is a crucial phase of anaesthesia for a veterinary patient; it can calm the animal and decrease the amount of induction and maintenance drugs that are needed [19]. Mild sedation is desirable to prevent excitement during anaesthetic induction and to avoid excess sympathetic tone, which can cause cardiac arrhythmia or ventricular fibrillation [20, 21].

Opioids are the preferred agents in critically ill patients because of the minimal effect of these agents on cardiac output (CO), systemic blood pressure, and oxygen delivery. They also provide analgesic and sedative effects that can be reversed with naloxone, if necessary [22]. Generally speaking, however, at high dosages, they cause bradycardia mediated via a vagal mechanism [21]: a slowing of HR in puppies results in decreased CO due to the limited capacity of the heart to increase cardiac stroke volume [13]. These side effects must always be carefully weighed against their benefits, particularly in terms of sedation and analgesia. Atropine, an anticholinergic drug used to decrease respiratory tract secretions



**Fig. 2** Electrocardiographic findings in a dog with CTD during the 3<sup>d</sup> balloon inflation (22 mm balloon catheter). The trace reveals atrial (QRS complex similar to a sinus complex), ventricular (wide QRS complex followed by a large T wave) and junctional beats (QRS complex similar to a sinus complex and a negative P wave). The atrial beats are followed by ventricular beats, and junctional beats occurred when the balloon slipped distally against the tricuspid valve. Lead II tracing; Speed 25 mm/s; amplitude 10 mm/mV

and to treat bradyarrhythmias, was not included in our protocol because of its intrinsic arrhythmogenic potential [23, 24] and the probable tachyarrhythmias that the procedure itself could have caused [9, 15, 16].

Benzodiazepines such as midazolam and diazepam are unreliable anxiolytics when given alone, unless the animal has central nervous system depression; however, they are able to provide improved sedation when given in combination with opioids and dissociatives [22]. Furthermore, young puppies, unlike adult dogs, are more susceptible to the sedative effects of benzodiazepines.

Acepromazine, a phenothiazine derivate, is largely used as a sedative in dogs, but its tendency to cause mild hypotension by blocking  $\alpha$ -1 receptors and its long halflife made it unsuitable for use in our case. Alpha-2 agonists (e.g. dexmedetomidine, medetomidine) are excellent sedatives and anxiolytics, but significantly affect cardiovascular function. The main negative cardiovascular effects of these drugs include bradycardia with possible bradyarrhythmias (atrioventricular heart block and ventricular escape rhythm) and a dramatic reduction in CO caused by reduced central sympathetic tone. These molecules also cause an increase in systemic vascular resistance (peripheral action on α-2 receptors) and hypertension, which induces a reflex baroreceptor-mediated physiologic bradycardia and marked reduction in CO, further perpetuated by the central effects of sedation and reduced sympathetic tone [17, 25]. Healthy paediatric patients have limited stroke volume and cardiac reserve, and CO is dependent on HR [13]; therefore, this class of drugs, which causes bradycardia and a decrease in CO, was not considered for the anaesthetic management of this puppy. Furthermore, the cardiovascular procedure itself could cause arrhythmias, and so these drugs were excluded from the protocol.

Ideally, after the clinical effect of premedication, a slow transition to general anaesthesia should occur to allow time for the cardiovascular and nervous system to appropriately respond to and accommodate the medications [26]. Propofol is a hypnotic agent, which, because of its multiple routes of elimination and its smooth induction, especially for recovery, is most commonly used for paediatric patients to induce general anaesthesia [21, 27, 28]. Cardiovascular changes induced by propofol administration consist of a slight decrease in arterial blood pressures (systolic, mean, diastolic) without a compensatory increase in HR, and so the dose of propofol should always be carefully titrated against the needs and responses of individual patients, as their anaesthetic requirements vary considerably [28]. In our case, the dog required a low dose of propofol for the induction of general anaesthesia. Propofol binds strongly to serum protein (97-98 %); hence, in this specific puppy, hypoproteinaemia (hypoalbuminaemia and hypohaemoglobinemia) resulted in an increase of the unbound drug in the circulation (i.e. the active part of the drug), which may have been further increased by the strong protein binding of the other drugs used for premedication. Thus, we can assume that the poor clinical condition of the patient contributed to a lower than expected volume of distribution and, therefore, in a higher serum drug concentration. These possible contributing factors, added to the effects of premedication, probably justify the low dose of propofol used for induction of general anaesthesia.

All volatile inhalation anaesthetics cause dose-dependent and drug-specific changes in cardiovascular performance: decreased arterial blood pressure, stroke volume, and peripheral vascular resistance. The newer volatile anaesthetics tend to preserve CO at clinically useful concentrations [29, 30]. A major advantage of these newer agents (sevoflurane, desflurane) is their superior pharmacokinetic properties, including low solubility in blood and tissues [31], resulting in a faster



Fig. 3 Electrocardiographic findings in a dog with CTD during the last balloon inflation (22 mm balloon catheter). The trace shows a non-sustained supraventricular tachycardia (duration 6 s) with a rate of 250 beats/min. Lead II tracing; Speed 25 mm/s; amplitude 10 mm/mV

**Table 3** Anaesthetic protocol and details of cardiovascular procedure for a balloon dilation of cor triatriatum dexter in a dog

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Premedication	Sufentanil (0.5 μg/kg) + Midazolam (0.2 mg/kg)		
Induction	Propofol (2 mg/kg)		
Maintenance	Isoflurane in $O_2$ + Sufentanil CRI (0.5 $\mu$ g/kg/h)		
Total anaesthesia time	117 min		
Total cardiovascular procedure time	62 min		
Total time of the inflation-deflation of the BC	12 min		
Number of balloon inflations	20; 10 (12 mm BC) + 10 (22 mm BC)		
Total sufentanil CRI time	97 min		
Total dose of sufentanil	15.1 μg		

BC balloon catheter, CRI constant rate infusion

induction and recovery and improved control of the depth of anaesthesia [32]. However, recent veterinary literature lacks clear evidence of better clinical performance for sevoflurane and desflurane, compared with isoflurane, in non-critical dogs and cats. Furthermore, both isoflurane and sevoflurane are used in cardiac surgery in humans, resulting in a similar cardioprotective effect [33, 34].

Sufentanil decreases the minimum alveolar concentration (MAC) of volatile anaesthetics [35, 36]. In our case, isoflurane end tidal concentration was maintained mostly below 1 MAC (1 MAC = 1.28 vol.%), resulting in good haemodynamic stability (Table 2). Moreover, sufentanil and other potent opioids can have a cardioprotective and antiarrhythmic effect mediated by diverse intracellular signalling pathways and indirect effects [37, 38]. Indeed, this specific μ-opioid receptor agonist is largely used for cardiac surgery anaesthesia in children [39, 40]. On the other hand, the possible occurrence of bradycardia due to vagal stimulation is an undesirable event in a puppy because of the negative effect that bradycardia may have on CO. Little information has been reported in the literature about pain during minimally invasive vascular procedures. Portmans asserts that the inflation of the balloon (e.g. dilation of a stenotic valve) can cause pain, which is essentially acute and intense, but short-lived [11]. Thus, the reasons for choosing the potent opioid sufentanil for the management of anaesthesia include its halogensparing effects, its cardioprotective and analgesia effects during inflation of the balloon in the atrium, and the possibility of controlling undesirable bradycardia with the administration of atropine.

The puppy in this report, despite having hypoproteinaemia, an obstructed venous return, and an extremely tense abdomen, did not require any drugs to support cardiovascular function, because the physiological parameters were within normal ranges throughout the entire procedure. Hypoproteinaemia usually causes an increase in the active, unbound plasma concentration of drugs, and thus patients with this condition should be treated cautiously, as they are more prone to develop side effects from a relative overdose [41]. Hypoproteinaemia can also affect oncotic pressure, making the dog more prone to hypovolemia and hypotension [42].

The use of spontaneous ventilation in our patient was suggested for two main reasons: 1) Patients undergoing minimally invasive procedures usually do not require a surgical plane of anaesthesia and therefore most of the time do not easily adapt to the ventilator unless more anaesthetic drug or a neuromuscular blocker is administered; and 2) positive pressure ventilation may interfere with cardiovascular function, especially in haemodynamically compromised patients, whereas spontaneous ventilation may better preserve venous return because of negative intrathoracic pressure during inspiration [43]. In any case, positive pressure ventilation should always be available and ready to be used in case the patient's condition deteriorates.

During CTD dilation, atrial fibrillation, atrial flutter, and ventricular and supraventricular tachyarrhythmias have been reported in dogs [9, 15, 16]. One minute of cardiac arrest, successfully treated with internal cardiac massage and adrenaline injection, was described during the surgical treatment of CTD in a dog [7]. Although tachyarrhythmias appear to be most frequent, many types of arrhythmias may occur (atrioventricular block, tachy-bradyarrhythmias, cardiac arrest) [16, 44–46], and so the authors considered it more appropriate to administer the correct antiarrhythmic drug when needed, rather than administering it preventively.

In our case, supraventricular extrasystoles observed during the placement of the guide wire or balloon catheters were likely due to the contact of the catheter tip with the atrial wall. Supraventricular tachycardia occurred during balloon inflation as a consequence of atrial membrane and wall stretching. Ventricular and junctional beats were recorded when the balloon slipped distally to the membrane against the tricuspid valve during inflation. The fact that arrhythmias disappeared and baseline cardiovascular and respiratory parameters resumed at the end of the procedure suggests that these side effects were caused by the mechanical action of the catheters and guide wire on the cardiac structures (Table 3). Despite the numerous arrhythmias, the invasive blood pressure values did not show any major variations during balloon inflation (Fig. 1), probably because each individual dilation was brief (3-5 s) and because a 20- to 30-s pause was guaranteed between inflations in order to allow the blood to flow through the cardiac chambers.

In our experience, perfect interaction between cardiologist and anesthesiologist is the basis of patient management; on request by the anesthesiologist, displacement of the intracardiac catheter by even a few millimeters led to total interruption of the arrhythmias.

Although the arrhythmias recorded in our case report were benign and might be explained by the contact of the catheters and guide wire on the endocardial wall, it must be emphasized that the supraventricular and ventricular arrhythmias may place the patient at higher risk for development of malignant arrhythmias. Therefore, anti-arrhythmic drugs (e.g. lidocaine, esmolol, atropine) and a defibrillator must be available during the interventional procedure. Lidocaine IV via bolus or CRI has been used to provide perioperative antiarrhythmic effects and has been assessed as advantageous for dogs undergoing balloon valvuloplasty for pulmonic stenosis [47].

Our findings are in complete agreement with those described in the literature regarding the occurrence of arrhythmias and their spontaneous resolution following withdrawal of the catheters. The protocol described in this case report resulted in good management of anaesthesia and analgesia, as well as a fast, smooth, and uneventful recovery. Although in particular cases, it is highly recommended to tailor the anaesthetic protocol to the individual patient's clinical condition, the protocol described in this case can be considered a valid basic approach to be adapted as necessary; moreover, considerable attention should be given to arrhythmias that develop during the procedure to provide timely intervention.

# **Endnotes**

<sup>1</sup>MyLab 30 Vet, Esaote, Genova, Italy

<sup>2</sup>Diuren; Teknopharma S.p.A., Torino, Italy

<sup>3</sup>Prilactone; Ceva Vetem S.p.A., Monza e della Brianza, Italy

<sup>4</sup>Vasotop P; MSD Animal Health S.r.l., Latina, Italy

<sup>5</sup>MyLab<sup>™</sup>30 Vet Gold; Esaote, Genova, Italy

<sup>6</sup>Cell-Dyn 3700; Abbott, Roma, Italy

<sup>7</sup>Liasys; Assel S.r.l., Latina, Italy

<sup>8</sup>Delta ven<sup>®</sup> 1 G 22; Delta Med S.p.A., Mantova, Italy

<sup>9</sup>Sufentanil-hameln; Hospira Srl, Napoli, Italy

<sup>10</sup>Midazolam I.B.I.; Hospira, Lake Forest, IL, USA

<sup>11</sup>Proposure 1 %; Merial S.p.A., Milano, Italy

<sup>12</sup>Rüsch<sup>®</sup> 6.5 mm endotracheal cuffed tube; Teleflex Medical S.r.l., Monza e della Brianza, Italy

<sup>13</sup>Infant T-piece anaesthetic breathing system; Intersurgical S.p.A., Modena, Italy

<sup>14</sup>Excel 210; Ohmeda, Madison, WI, USA

<sup>15</sup>Isoflurane-Vet; Merial, Milano, Italy

<sup>16</sup>Ringer Lattato; S.A.L.F S.p.A., Bergamo, Italy

<sup>17</sup>SK500II sirynge pump; Foschi S.r.l., Roma, Italy

<sup>18</sup>Delta ven<sup>®</sup> 1 G 24; Delta Med SPA, Mantova, Italy

<sup>19</sup>Multiparameter Monitor HB100; Foschi, Roma, Italy

<sup>20</sup>Tyshak II<sup>®</sup> balloon valvuloplasty catheter; Infiniti Medical, LLC<sup>™</sup>, West Hollywood, CA, USA

<sup>21</sup>DPT-6000; Codan srl, Modena, Italy

<sup>22</sup>Rosen Wire; Infiniti Medical, LLC™, West Hollywood, CA, USA

#### **Abbreviations**

BC: Balloon catheter; CRI: Constant rate infusion; CTD: Cor triatriatum dexter; DAP: Diastolic arterial pressure; ECG: Electrocardiogram; EtCO<sub>2</sub>: End-tidal carbon dioxide partial pressure; EtIso: End-tidal isoflurane concentration; HR: Heart rate; MAP: Mean arterial pressure; RR: Respiratory rate; SAP: Systolic arterial pressure; SpO<sub>2</sub>; Haemoglobin oxygen saturation; T: Rectal temperature.

#### Competing interests

The authors declare they have no competing interest.

## Authors' contributions

DMV performed general anaesthesia of the dog and drafted the manuscript; SF contributed to the discussion of the anaesthetic management of the case and supervised and contributed to the drafting of the manuscript and its revisions; CD carried out the cardiovascular procedure and wrote the cardiological findings in the manuscript; BA carried out the anaesthesia and critically revised the manuscript for important intellectual content. All authors have read and accepted the final version of the manuscript.

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