COMPANION OR PET ANIMALS

Cardiovascular complications during anaesthesia for surgical correction of an aberrant right subclavian artery in a dog

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SUMMARY

A six-month-old neutered male German shepherd dog with aberrant right subclavian artery (ARSA) was premedicated with methadone, medetomidine and then fentanyl; anaesthesia was induced with propofol and maintained with infusions of medetomidine and fentanyl and isoflurane in oxygen. Hartmann's solution and noradrenaline were administered for cardiovascular support. Arterial pressure was measured using Doppler technique in both metacarpal arteries and invasive technique in both dorsal pedal arteries. During ARSA occlusion test, severe hypotension (30 mmHg) in the right forelimb, bradycardia (45 bpm) and advanced atrioventricular blocks (AVB) were observed. The occlusion test was interrupted; atropine was administered; cardiovascular parameters, except AVB, were normalised; medetomidine and noradrenaline were interrupted. Other two attempts of ARSA occlusion generated similar cardiovascular side effects. The surgical correction was precluded and the animal was recovered from anaesthesia. This case indicates that ARSA occlusion can be followed by severe arrhythmias and focus on the management of these alterations.

BACKGROUND

The aberrant right subclavian artery (ARSA) is a congenital vascular ring anomaly, resulting from a failure of the seventh intersegmental artery on reaching the fourth aortic arch before it separates from the dorsal aorta.[1] In humans, it rarely causes symptoms although representing one of the most common anatomic variations of the aortic arch branching, occurring in 0.5–2 per cent of the population.[2] In dogs, the incidence is thought to be rare since there are only two clinical cases of dogs with ARSA described in the literature.[3 4] However, there are reports of at least 21 anatomical findings of an ARSA observed during postmortem dissections of dogs,[5–12] suggesting that the anomaly may be undetected due to the absence of clinical signs. Despite consisting of a vascular anatomical variation, dysphagia and regurgitation are described as clinical signs since the ARSA usually courses dorsal to the aorta and entraps the oesophagus.[1] The surgical correction is indicated when the quality of life is deteriorated by dysphagia, regurgitation and pneumonia.[3 4] During surgery, digital occlusion tests of the ARSA are performed to check the patency of the blood flow by the collateral circulation to the right forelimb.[3] If the collateral circulation is not adequately recruited, the limb will not have a proper blood flow and may need to be amputated. In this case, the owners have to be consulted in order to choose between the permanent ARSA occlusion with limb amputation and preclusion of ARSA closure with continuing dysphagia, regurgitation and pneumonia. Other limitations may be present but the lack of surgical cases described in the literature impedes anticipation of complications and adequate preparation of an anaesthetic protocol. This case report describes the management of cardiovascular alterations that developed during anaesthesia following the attempted surgical correction of an ARSA in a dog.

CASE PRESENTATION

A six-month-old neutered male German shepherd dog of 23 kg bodyweight with clinical history of cough, dysphagia, regurgitation and pneumonia was referred to our institution for further evaluation. Clinical examination revealed a slightly elevated rectal temperature (39.1°C) and lung auscultation revealed stridors more remarkable over the right ventral side of the chest. The dog showed cough and regurgitation only after drinking a few sips of water.

INVESTIGATIONS

The endoscopic examination revealed a ring-shaped extraluminal narrowing of the oesophagus at the level of the heart. The stricture represented no impairment to the introduction of the 1.5 cm diameter endoscope into the oesophagus. Chest radiographs showed an alveolar pattern within the ventral-cranial lobes indicating bronchopneumonia. The oesophagram revealed an oesophageal indentation at the level of T1-T2 with a mild dilation cranial to the focal stricture observed following introduction of liquids.

An angiography CT scan of the thorax was performed to confirm the suspected diagnosis of a vascular ring anomaly. The exam showed the presence of an ARSA arising from the aortic arch (instead of the brachiocephalic trunk), next to the left subclavian artery, to the left of the oesophagus, and extending over the dorsal part of the oesophagus towards left, causing a partial constriction to it.

The diagnosis of an ARSA causing an oesophageal narrowing was confirmed and surgical correction

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was proposed to the owners of the dog. They were advised that an occlusion test of the blood flow to the right forelimb using a Doppler probe would have to be performed during surgery to ensure adequate activation of the collateral circulation before permanent ARSA closure. In the case of inadequate blood flow to the limb, the owners decided to refrain from surgical correction of the ARSA. With the owners' written consent, the thoracotomy was scheduled for 15 days later. During these days, the dog was prescribed the association of amoxicillin and acid clavulanic 20 mg/kg every 12 hours orally (Synulox, Zoetis).

DIFFERENTIAL DIAGNOSIS

Possible differential diagnosis for oesophageal obstruction included megaoesophagus, oesophageal tumour, foreign body, diverticulum and gastro-oesophageal intussusception.

TREATMENT

The dog was fasted eight hours prior to anaesthesia with free access to water. The animal was very agitated and was premedicated with methadone 0.2 mg/kg (Comfortan, Eurovet Animal Health) and medetomidine 10 µg/kg (Sedator, Eurovet Animal Health) intramuscularly. After 20 minutes, a 20 G catheter (Henry Schein) was fixed in the left cephalic vein and medetomidine 1 µg/kg (Sedator, Eurovet Animal Health) and fentanyl 3 µg/kg (Fentanyl, Janssen-Cilag) were administered intravenously over 10 minutes using syringe driver (Volumetric Infusion Pump, Caesarea Industrial Park Israel) followed by a constant rate infusion (CRI) of 1 and 5 µg/kg/hour, respectively, that were maintained thereafter. Anaesthesia was induced with propofol 1 mg/kg (Diprivan, AstraZeneca) 35 minutes after premedication. Orotracheal intubation (13.3 mm outer diameter) was performed in sternal recumbency and anaesthesia was maintained using isoflurane in oxygen/air mixture maintaining the fraction of expired isoflurane (FE_{ISO}; IsoFlo, Zoetis) at 0.91–1.30 per cent and the fraction of inspired oxygen (FIO_2) at 0.85 by the use of a rebreathing circuit. The association of amoxicillin and acid clavulan ic 20 mg/kg (Augmentin, GlaxoSmithKline) was administered intravenously and was continued every 90 minutes until the end of surgery.

The animal was positioned in left lateral recumbency for surgical preparation of the thorax. The animal was manually ventilated with a respiratory rate (RR) of 14–26 breaths/minute, which maintained the end-tidal carbon dioxide ($ETCO_2$) concentration between 31 and 43 mmHg (Datex Capnomac Ultima, General Electric) and oxyhaemoglobin saturation measured by pulse oximetry (SpO_2) of 99–100 per cent (Model 64S, Hewlett Packard). A Hartmann's solution[j] was delivered at 5 ml/kg/hour using an infusion pump (Scil Infuvet, Animal Care) throughout. Two 20 G catheters (Henry Schein) were fixed in the right and left dorsal pedal arteries, respectively, for invasive blood pressure (IBP) measurement (Model 64S, Hewlett Packard) and collecting blood samples for blood gas analysis (Cobas b 123, Roche). The first blood gas analysis performed 20 minutes after induction revealed no alterations (Table 1). A Doppler probe (Model 811-B, Parks Medical Electronics) was applied over the metacarpal artery (cuff applied proximal to the carpus) of both forelimbs for measuring the systolic arterial pressure (SAP). The heart rate (HR) and II-derivate electrocardiogram (Model 64S, Hewlett Packard) were monitored using patches attached to the forelimbs and left hindlimb.

Mechanical ventilation (Neptune, Medec) was started in the surgical theatre using a pressure-controlled ventilation mode and an initial peak inspiratory pressure of 10 cmH_2O and RR of 16 breaths/minute that were adjusted to maintain $ETCO_2$ between 35 and 45 mmHg throughout. The HR, IBP, SpO_2, FE_{ISO}, FIO_2 and oesophageal temperature were assessed using a multiparameter monitor (Datex Ohmeda S/5, General Electric). Body temperature was maintained by use of a heat and moisture exchanger, a heating mat and active warm air device (Bair Hugger, Mistral Air). The mean arterial pressure (MAP) was 55 and 54 mmHg in the right and left pedal dorsal arteries, respectively, which was treated with FE_{ISO} decrease from 1.6 to 1.1 per cent, bolus of 10 ml/kg/10 minutes of Hartmann's solution (HTM, B Braun) and CRI of noradrenaline (Levophed, Hospira) (started at 0.5 µg/kg/minute for five minutes followed by 0.3 µg/kg/minute).Intercostal nerve blocks (second to fifth right ribs) were performed by infiltration of 2 mg/kg of 0.5 per cent levobupivacaine (Chirocaine, AbbVie) in total.

Thoracotomy in the right third intercostal space started at 75 minutes after induction of anaesthesia, when a second blood gas analysis was performed (Table 1). An occlusion test of the subclavian artery performed manually by the surgeons was started at 105 minutes after induction of anaesthesia (Fig 1). At this moment, a suspected high-grade second-degree atrioventricular block (AVB) with possible intermittent third-degree AVB was observed and the HR decreased from 65 to 45 bpm. The blood flow in the right forelimb was less audible and SAP changed from 100 to 40 mmHg in the right metacarpal artery, from 140 to 100 mmHg in the left metacarpal artery, from 113 to 110 mmHg in the right pedal dorsal artery (MAP from 66 to 65 mmHg) and from 111 to 122 mmHg in the left pedal dorsal artery (MAP from 67 to 71 mmHg) (Fig 1 and Table 1). Atropine 10 µg/kg (Atropine, Laboratory STEROP) was administered intravenously, which increased HR to 107 bpm, and increased the SAP in all limbs. The arrhythmia consisted now of a second-degree AVB and medetomidine was stopped immediately, followed by interruption of noradrenaline after two minutes. These manoeuvres allowed for normalisation of HR and SAP increased to 195 mmHg in the right forelimb, but cardiac rhythm was still impaired.

In the second occlusion test, the blood flow to the right forelimb was almost not audible, with hypotension only in this limb (SAP of 30 mmHg) (Fig 1 and Table 1), and persistence of suspected intermittent second-degree AVB. The arterial pressure in the right forelimb was again normalised by releasing the blood flow of the ARSA, but cardiac arrhythmia remained.

The third occlusion test followed the same alterations previously observed, that is, great decrease in SAP with much less audible blood flow in the right forelimb and cardiac arrhythmia. The occlusion tests were performed at intervals of approximately 10 minutes. Given the risk of ischaemia and necrosis of right forelimb, the permanent ARSA closure was precluded. A third blood gas analysis was performed (Table 1) and the thoracotomy was closed routinely. Before closure, a thoracic drain was placed and levobupivacaine (1 mg/kg diluted into 10 ml of saline) was administered locally through the drain and continued every four hours in the first 24 hours postoperatively.

Isoflurane was terminated, a fourth blood gas analysis was performed (Table 1), and the animal was allowed to recover from anaesthesia with oxygen supplementation through the endotracheal tube. Extubation was performed after recovering repetitive deglutition reflex, which occurred at 12 minutes after terminating isoflurane. After extubation, rectal temperature was 38.8°C, HR was 112 bpm, SpO_2 was 91 per cent and the animal was panting. Oxygen supplementation was continued through mask, and a single dose of acepromazine 5 µg/kg

TABLE 1: Cardiorespiratory parameters and blood gas analysis of an anaesthetised dog undergoing thoracotomy for correction of an aberrant right subclavian artery

	Minutes from induction of anaesthesia				Minutes after extubation	
	20	**75**	**150**	**210**	**5**	**120**
FE_{ISO} (%)	0.95	1.1	1.2	1.1	–	–
SAP left forelimb (mmHg)	130	118	138	120	–	–
SAP left hindlimb (mmHg)	98	50	112	111	–	–
MAP left hindlimb (mmHg)	63	36	72	65	–	–
MAP right hindlimb (mmHg)	50	40	72	65	–	103
DAP left hindlimb (mmHg)	50	30	59	49	–	–
DAP right hindlimb (mmHg)	30	51	58	49	–	82
RR (bpm)	15*	16*	13*	46	–	100
SpO_2 (%)	98	96	98	97	–	96
$ETCO_2$ (mmHg)	35	52	40	31	–	–
Ventilation mode	Sp	PCV	PCV	Sp	Sp	Sp
V_T (ml)	–	290	305	167	–	–
PIP (cmH_2O)	–	8	16	1	–	–
Temperature (°C)	39.1	38.5	38.8	38.8	38.8	38.8
pH	7.298	7.231	7.264	7.171	7.311	7.342
FIO_2	1.0	0.86	0.85	0.98	0.21	0.21
PaO_2 (mmHg)	504.7	492.6	346.7	234.8	80.7	83.9
SaO_2 (%)	100	100	100	100	98	99.5
$PaCO_2$ (mmHg)	43.7	56.5	52.6	69.7	44.2	42.1
HCO_3^- (mmol/l)	20.9	23.2	23.3	24.9	21.8	22.3
BE (mmol/l)	–5.14	–4.61	–3.83	–4.54	–4.18	–2.94
Hct (%)	29.6	27	27.1	28	30	28.7
Na^+ (mmol/l)	141.8	143.1	140.5	143.9	141.2	142.3
K^+ (mmol/l)	4.4	4.65	4.88	4.31	4.21	3.45
Ca^+ (mmol/l)	1.442	1.378	1.296	1.304	1.218	1.364
Cl^- (mmol/l)	115.2	114.9	114.3	113.7	114.1	114.5

'75 minutes' corresponded to the beginning of surgery; '150 minutes' corresponded to the end of the occlusion tests; '210 minutes' corresponded to the end of surgery.

BE, arterial base excess; Ca^+, arterial calcium ion concentration; Cl^-, arterial chloride ion concentration; DAP, diastolic arterial pressure; $ETCO_2$, end-tidal carbon dioxide; FE_{ISO}, fraction of expired isoflurane; FIO_2, fraction of inspired oxygen; HCO_3^-, arterial bicarbonate concentration; Hct, haematocrit; K^+, arterial potassium ion concentration; MAP, mean arterial pressure; Na^+, arterial sodium ion concentration; $PaCO_2$, partial pressure of carbon dioxide in the arterial blood; PaO_2, partial pressure of oxygen in the arterial blood; PCV, pressure-controlled ventilation; PIP, peak inspiratory pressure; RR, respiratory rate; SaO_2, arterial oxygen saturation; SAP, systolic arterial pressure; SpO_2, pulse oximetry; V_T, tidal volume.

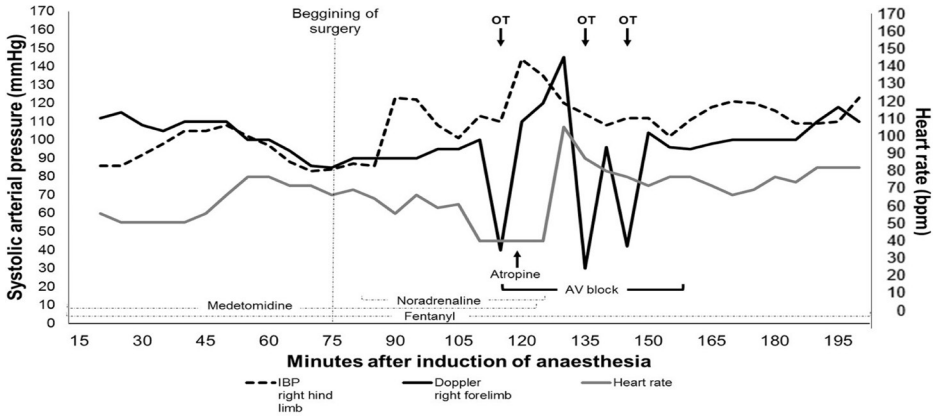

FIG 1 Systolic arterial pressure (SAP) measured non-invasively using the Doppler technique over the metacarpal artery on the right forelimb and measured invasively using an arterial catheter into the pedal dorsal artery in the right hindlimb. Aberrant right subclavian artery occlusion tests (OT) were followed by the development of atrioventricular (AV) block and decrease in SAP only in the right metacarpal artery. Treatment consisted of interrupting the infusion of medetomidine and noradrenaline and administration of atropine. IBP, invasive blood pressure.

intravenously (Placivet, Kela) and carprofen 4 mg/kg intravenously (Rimadyl, Zoetis) was administered. A CRI of fentanyl 3 µg/kg/hour (Fentanyl, Janssen-Cilag) was continued during the first 12 hours after surgery, then replaced by methadone 0.2 mg/kg every four hours intravenously (Comfortan, Eurovet Animal Health) for 12 hours. Additionally, in the first 24 hours postoperatively, the dog was treated with Hartmann's solution at 4 ml/kg/hour and the association of amoxicillin and clavulanate potassium (Augmentin, GlaxoSmithKline) at 20 mg/kg intravenously every eight hours. No cardiac arrhythmias, pain and alterations in breathing, SpO_2 and blood gas analysis (at 5 and 120 minutes after extubation; Table 1) were observed postoperatively. The thoracic drain was removed 24 hours after surgery as no significant amount of air or fluid was observed.

OUTCOME AND FOLLOW-UP

The dog was discharged two days later and was prescribed tramadol 2 mg/kg (Contramal, Grunenthal) every eight hours orally for seven days and the association of amoxicillin and acid clavulanic 20 mg/kg every 12 hours orally (Synulox, Zoetis) for 10 days. The owners were advised to feed the dog with more solid food placed on a higher level. They never showed up for the follow-up that was scheduled for 10 days after discharge. No response was obtained from the owners and referral veterinarian when contacted several times by phone and email.

DISCUSSION

The subclavian artery is the main artery providing blood flow to all the other arteries of the forelimb (ie, axillary, brachial, median).[1] In children, the ARSA is a common anomaly and the surgical ligation of the ARSA is often successful, with no loss of function, likely due to compensatory circulation via anastomosis of vascular branches.[7] In dogs, the ARSA is a rare congenital disorder with an apparent low incidence of clinical signs.[6] From the two dogs with clinical signs described in the literature, one was managed with conservative treatment[4] and the other was submitted to surgical correction.[3] In both reports, the outcome was good and no cardiovascular complications were observed. It is not known what is the main trigger for activation of collateral circulation and the low number of clinical cases makes it difficult to predict the potential cardiovascular alterations that can develop during surgery and, therefore, to prepare an appropriate anaesthetic protocol. The present case report describes a dog with ARSA in which surgical correction was not successful. The ARSA occlusion test was not followed by the establishment of an adequate blood flow to the forelimb and probably initiated the development of cardiovascular complications. The present findings should be reported since the literature lacks such information.

The deterioration of heart conduction could be attributed to an early afterdepolarisation-induced triggered activity, which are oscillations on the membrane potential that occur during or immediately after a preceding action potential (trigger). A new action potential is generated when they reach the threshold potential, which triggers a new response, resulting in a self-sustaining triggered activity. The early afterdepolarisation could have been induced by different mechanisms, such as slow HR (eg, bradycardia, complete heart block), mechanical stretch by surgical manipulation, acidosis, ion imbalances and hypoxia.[13] The most likely causes are slow HR and mechanical stretch, since acidosis and ion imbalances were not observed on blood gas analysis.

Medetomidine and fentanyl have the potential to decrease HR and cause arrhythmias that could have been potentiated by the occlusion test. The bradyarrhythmia induced by medetomidine is first a vagal response to a baroreceptor reflex and then is a prolonged reduction in sympathetic nervous system tone, via an effect mediated at presynaptic alpha-2 adrenoceptors located in the CNS.[14] Medetomidine, as well as fentanyl, decreases the outflow of noradrenaline in the CNS, dampening the central sympathetic tone which, in turn, can decrease HR and induce first and second-degree AVB.[14] The arrhythmia could also be associated with noradrenaline which has a beta-adrenoceptor agonist effect that predisposes to the generation of cardiac arrhythmias.[15] Usually, all these drugs are well tolerated; however, more severe arrhythmias were potentially initiated by the ARSA occlusion test. Atropine was effective to increase HR, indicating bradycardia was caused by an increased vagal tone. In addition, atropine increases firing of the sinoatrial node and conduction through the atrioventricular node, which explains its efficacy for treating the arrhythmia in the present case.[16] Other potentially preferential agents for use during an AVB, such as isoproterenol, could have been used if atropine had not been effective, which could have been the case if the breakdown in signal transmission was in the bundle of His/Purkinje fibres.

Hypoxaemia and hypotension can lead to myocardial hypoxia which can result in heart conduction disorders. Despite some increase in alveolar-arterial oxygen pressure gradient [$P(A\text{-}a)O_2$] from 50 to 377 mmHg, calculated using the standard formula for alveolar oxygen pressure, $PAO_2 = FIO_2 \times (P_{ATM} - P_{H2O}) - (PaCO_2/0.8)$ (P_{ATM}: atmospheric pressure of 760 mmHg; P_{H2O}: vapour pressure of 47 mmHg; $PaCO_2$: partial pressure of carbon dioxide in the arterial blood; '0.8' as the respiratory quotient),[17] which was attributed to bronchopneumonia, the dog had no low values for partial pressure of oxygen in the arterial blood (PaO_2). In addition, hypotension lasted a few minutes and was successfully treated with noradrenaline. The decrease in arterial pressure could have occurred through an effect of medetomidine on alpha-2 adrenoceptors in the CNS that is observed after an initial increase of approximately 10 minutes in arterial pressure.[18] However, at the dose used, medetomidine was described to have analgesic and isoflurane-sparing effects[19] with minimal and transient haemodynamic changes.[20] Medetomidine and fentanyl alone can decrease the isoflurane minimum alveolar concentration (MAC) in dogs by 17 per cent during ovariohysterectomy[19] and by 54–66 per cent during mastectomy,[21] respectively, and despite differences in surgical stimulation, it was decreased by 8–27 per cent, considering that the MAC of dogs is 1.27 per cent.[22]

Some differences on blood pressure between limbs were observed and might be attributed to the different techniques used for measuring arterial pressure, that is, invasive and non-invasive, and interference of measuring blood pressure in an uppermost or undermost limb with an animal positioned in lateral recumbency. However, overall the values were in accordance to indicate low and normal blood pressure and it is unlikely that they were not reliable. During the occlusion tests, the great decrease in the blood flow sound and in arterial pressure was only observed in the right forelimb.

In conclusion, it is not possible to predict the surgical correction of an ARSA prior to surgery, and monitoring blood flow and pressure from the metacarpal artery as well as from another artery during surgery is mandatory. The ARSA occlusion test can induce severe arrhythmias and, therefore, the use of drugs that can potentially cause bradycardia and arrhythmias, such

as alpha-2 adrenoceptor agonists and noradrenaline, should be used with caution.

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REFERENCES

1 Kau T, Sinzig M, Gasser J, *et al*. Aortic development and anomalies. *Semin Intervent Radiol* 2007;24:141–52.
2 Thompson JL, Burkhart HM. Translocation of an aberrant right subclavian artery with resolution of dysphagia lusoria. *Ann Thorac Surg* 2016;102:e65–e67.
3 Yoon HY, Jeong SW. Surgical correction of an aberrant right subclavian artery in a dog. *Can Vet J* 2011;52:1115–8.
4 Miller R, Wilson C, Wray J, *et al*. Adult-onset regurgitation in a dog with an aberrant right subclavian artery: a CT angiographic study. *Vet Rec Case Rep* 2015;3:e000138.
5 Paiva OM. [Two cases of right subclavian artery as the last collateral of the aortic arch in the dog]. *Rev Fac Med Vet Univ Sao Paulo* 1948;3:203–22.
6 Vitums A. Anomalous origin of the right subclavian and common carotid arteries in the dog. *Cornell Vet* 1962;52:5–15.
7 DeKleer VS. An anomalous origin of the right subclavian artery in the dog. *Br Vet J* 1971;127:76–82.
8 Tsukise A, Sugawa Y, Okano M. Two anomalous cases of the right subclavian artery arising directly from the aortic arch in dogs. *Nihon Juigaku Zasshi* 1972;34:11–15.
9 Gerrity LW, Martin JE. Anomalous right subclavian artery in a dog. *Southwestern Veterinarian* 1980;33:76–7.
10 Bezuidenhout AJ. Anomalous origins of the right subclavian and common carotid arteries in the dog. *J S Afr Vet Assoc* 1989;60:215–8.
11 Griffiths D. Three cases of aberrant right subclavian artery in the dog. *Acta Vet Scand* 1989;30:355–7.
12 Culau P, Oliveira JCD, Reckziegel SH, *et al*. Origem ectópica da artéria subclávia direita e do tronco bicarotídeo no cão. *Ciência Rural* 2004;34:1615–8.
13 Gaztañaga L, Marchlinski FE, Betensky BP. Mechanisms of cardiac arrhythmias. *Rev Esp Cardiol* 2012;65:174–85.
14 Murrell JC. Pre-anaesthetic medication and sedation. In: Duke-Novakovski T, de Vries M, Seymour C, eds. *BSAVA manual of canine and feine anaesthesia and analgesia*. Gloucester: British Small Animal Veterinary Association, 2016:170–89.
15 Wengrowski AM, Wang X, Tapa S, *et al*. Optogenetic release of norepinephrine from cardiac sympathetic neurons alters mechanical and electrical function. *Cardiovasc Res* 2015;105:143–50.
16 Trappe HJ, Hans-Joachim T. Concept of the five 'A's for treating emergency arrhythmias. *J Emerg Trauma Shock* 2010;3:129–36.
17 Fantoni DT, Ida KK, Lopes TF, *et al*. A comparison of the cardiopulmonary effects of pressure controlled ventilation and volume controlled ventilation in healthy anesthetized dogs. *J Vet Emerg Crit Care* 2016;26:524–30.
18 Murrell JC, Hellebrekers LJ. Medetomidine and dexmedetomidine: a review of cardiovascular effects and antinociceptive properties in the dog. *Vet Anaesth Analg* 2005;32:117–27.
19 Rioja E, Gianotti G, Valverde A. Clinical use of a low-dose medetomidine infusion in healthy dogs undergoing ovariohysterectomy. *Can Vet J* 2013;54:864–8.
20 Kaartinen J, Pang D, Moreau M, *et al*. Hemodynamic effects of an intravenous infusion of medetomidine at six different dose regimens in isoflurane-anesthetized dogs. *Vet Ther* 2010;11:E1–16.
21 Barletta M, Quandt J, Hofmeister E. Determination of minimum alveolar concentration of isoflurane in dogs and cats using the up-and-down method. A preliminary study. *Res Vet Sci* 2016;106:81–3.
22 Steagall PV, Teixeira Neto FJ, Minto BW, *et al*. Evaluation of the isoflurane-sparing effects of lidocaine and fentanyl during surgery in dogs. *J Am Vet Med Assoc* 2006;229:522–7.

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