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Hybrid technique for ventricular septal defect closure in a dog using an Amplatzer® Duct Occluder II☆

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Abstract A left-to-right shunting muscular ventricular septal defect (VSD) was diagnosed in a 4-month-old, female, 1.8 kg Bichon Frise — poodle mix dog. Echocardiographic evidence of cardiac remodeling, calculated pulmonary blood flow (Qp) to systemic blood flow (Qs) ratio of 2.8, and radiographic evidence of pulmonary edema supported the diagnosis of a hemodynamically important VSD. Using a combination of surgery and interventional catheter-based techniques to approach the VSD through the right ventricle, the VSD was occluded with an Amplatzer® Duct Occluder (ADO) II device. The ADO II is a low profile, flexible device originally

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developed for patent ductus arteriosus closure in humans that has been used to close muscular and perimembranous VSD in children. This report describes the hybrid procedure and imaging that was essential for successful occlusion of the VSD in this dog.

Abbreviations

ADO II	Amplatzer® Duct Occluder II
LA_{SAX}:Ao	left atrium short-axis to aorta ratio
LVIDd	left ventricular internal dimension in diastole
LVIDs	left ventricular internal dimension in systole
TEE	transesophageal echocardiography
VSD	ventricular septal defect

A 4-month-old, female, 1.8 kg, Bichon Frise − poodle mix dog was referred to the Texas A&M University Veterinary Medical Teaching Hospital for evaluation of a heart murmur noted at an initial puppy examination. The owners reported an inconsistent history of possible mild tachypnea with exercise. A grade VI/VI systolic right midheart murmur was present. The remainder of the physical examination was unremarkable. Thoracic radiographs were obtained and documented enlargement of the main pulmonary artery and left atrium and a vertebral heart size (VHS) of 12.2 (normal $<$ 10.7).[1] The pulmonary vessels were generally enlarged and consistent with overcirculation while mild cardiogenic edema was noted in the right caudal lung lobe. Transthoracic echocardiographic findings included an enlarged left ventricular internal dimension in diastole (LVIDd 2.6 cm; 95% prediction interval, 1.56−2.27) and high end of the normal range dimension in systole (LVIDs 1.5 cm; 95% prediction interval, 0.88−1.57) based on M-mode measurements.[2] A central jet of mitral regurgitation was documented, and the left atrium short-axis to aorta ratio (LA_{SAx}:Ao) revealed left atrial enlargement (LA_{SAx}:Ao 2.15, normal $<$ 1.6).[3] A muscular ventricular septal defect (VSD) measuring 4.4 mm in diameter was visualized in an outlet position. Left-to-right shunting was documented with color Doppler and an agitated saline contrast study (Fig. 1, Video 1). The septum measured 3.3 mm thick at the ventral aspect of the defect and 4.5 mm thick at the dorsal aspect of the defect. The distance from the dorsal aspect to the aortic annulus was 8.1 mm. Spectral Doppler peak jet velocity across the VSD was 3.3 m/s corresponding to an estimated pressure gradient between the ventricles of 43.6 mmHg. Aortic outflow (1.4 m/s) and pulmonary outflow (1.2 m/s) peak systolic velocities were normal. The calculated pulmonary blood flow (Qp) to systemic blood flow (Qs) ratio was 2.8 and indicative of a hemodynamically important shunt.[4] Systolic blood pressure obtained via Doppler on the left forelimb was 128 mmHg. Laboratory work including complete blood count and biochemistries were within normal limits.

The calculated shunt ratio, presence of cardiac remodeling, and radiographic evidence of pulmonary edema supported the diagnosis of a hemodynamically important VSD. The small size of the dog precluded open-heart surgery or strictly catheter-based closure. The dog was discharged with enalapril (0.35 mg/kg PO q 12 h) and furosemide (1.11 mg/kg PO q 12 h) and was scheduled for blood work evaluation within 10 days while corrective techniques were considered. One month after initial presentation, the dog presented for VSD closure through a combination of thoracic surgery and catheter-based closure (hybrid procedure). History, physical examination, and thoracic radiographs were minimally changed from the initial evaluation except for an increase in body weight (2.5 kg), reduction in VHS to 11.2, and radiographic resolution of pulmonary edema. CBC and biochemistries (to match the earlier use of biochemistries instead of serum chemistries) were within normal limits. The dog received methadone (0.2 mg/kg SC) approximately 30 min prior to anesthetic induction with midazolam (0.2 mg/kg IV) and etomidate (0.8 mg/kg IV). Anesthesia was maintained with sevoflurane in oxygen (vaporizer setting ranging from 0 to 2.0%) and a fentanyl (8 μg/kg/h IV) and midazolam (0.48 mg/kg/h IV) constant rate infusion. Just prior to the skin incision, a bolus of lidocaine (2 mg/kg IV) was administered followed by a constant rate infusion (75 μg/kg/min IV) in anticipation of ventricular arrhythmias associated with the procedure. During the procedure, 2 additional boluses of lidocaine (2 mg/kg IV) were administered along with procainamide (20 mg/kg IV slowly over 20 min) to control arrhythmias. Additional

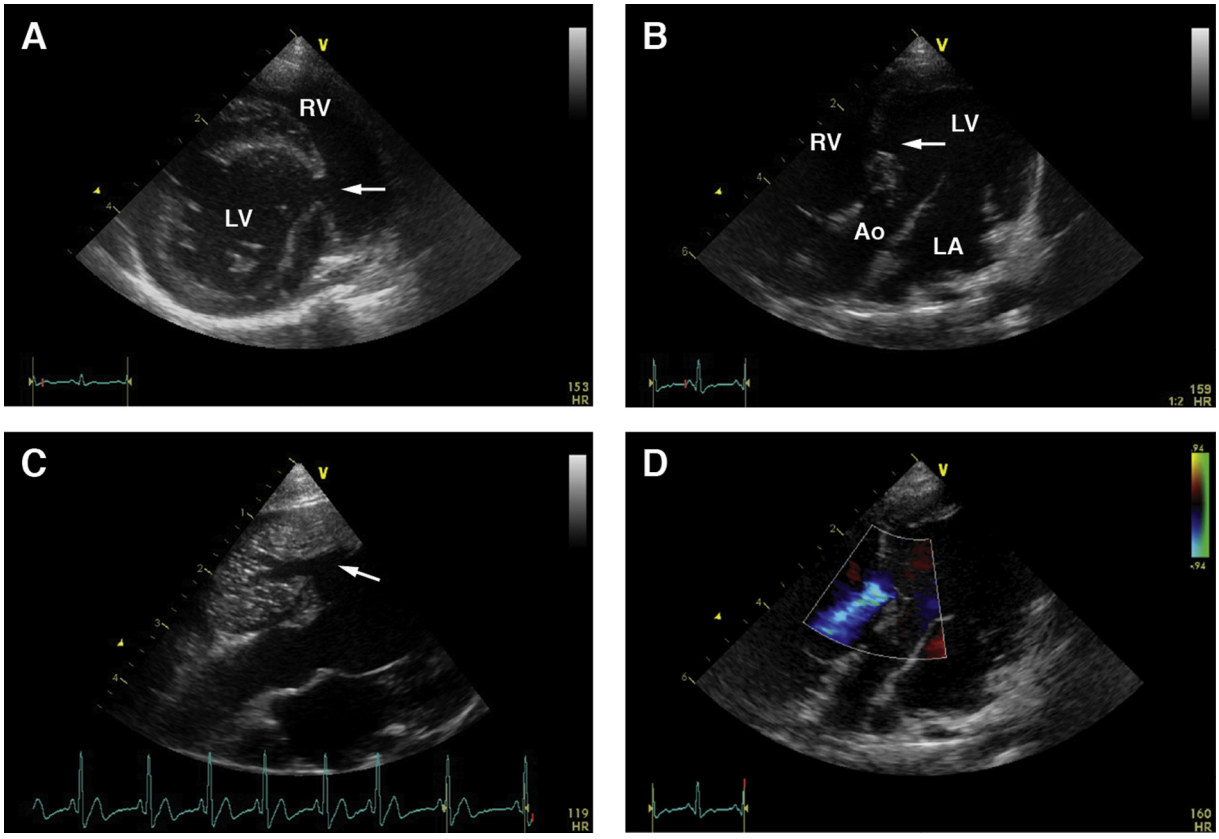

Fig. 1 Transthoracic echocardiographic images obtained from a right parasternal short-axis view at the level of the mitral valve (A) and a modified left parasternal view (B) in a dog with a VSD (arrow). Following injection of agitated saline in the cephalic vein, microbubbles can be seen within the right ventricle and the negative contrast caused by the left-to-right shunting across the VSD (C). Color Doppler demonstrating left-to-right shunting across the VSD (D). Ao = aorta, LA = left atrium, LV = left ventricle, RV = right ventricle.

medications included dobutamine (1.0—5.0 μg/kg/min IV) utilized intermittently to maintain systemic blood pressure and antibiotic therapy with cefazolin (20 mg/kg IV). Once the dog was anesthetized, transesophageal echocardiography (TEE) was utilized to measure the VSD and to provide intraoperative monitoring of device placement (Fig. 2). The dog was positioned in dorsal recumbency and a 7 cm skin incision was made from the caudal prominence of the xiphoid to the third sternebrae. Electrosurgical dissection was used to expose the caudal four sternebrae while the xiphoid process was removed, and the sternum was opened on midline. Following sternal retraction, a ventral midline pericardial incision was made that curved over the right ventricular outflow tract and a short distance distal to the pulmonary annulus. Using TEE guidance, the surgeon palpated the right ventricular free wall to identify a location to approach the VSD perpendicular to the septum. Suture was placed in a diamond pattern with 3 mm × 3 mm pledgets[c] in the right ventricular free wall to create a purse-string. An 18 gauge IV catheter[d] was used to puncture the right ventricle through the purse-string and an 0.035″ guide-wire[e] was passed through the catheter. Various manipulations of the wire using TEE guidance were unsuccessful in crossing the VSD. Epicardial ultrasound was then used to locate an area that would improve the approach across the VSD. The original purse-string was tied off and an additional purse-string was placed in the direction of the right ventricular outflow tract approximately 1 cm toward the pulmonary annulus and 1 cm toward the paraconal interventricular groove. The guide-wire was introduced using an IV catheter as previously described and was advanced across the VSD into the left ventricular lumen. In preparation for device placement, a 6 Fr vascular introducer[f] was passed over the guide-wire into the right ventricle

[c] Teflon pledget, DeRoyal Industries, Inc. Powell, TN, USA.

[d] Jelco I.V. Catheter Radiopaque, Smiths Medical International LTD, Rosendale, Lancashire, UK.

[e] Glidewire, Terumo Medical Corporation, Somerset, NJ, USA.

[f] 6 Fr Cath Lab Introducer Kit, Infiniti Medical, Malibu, CA, USA.

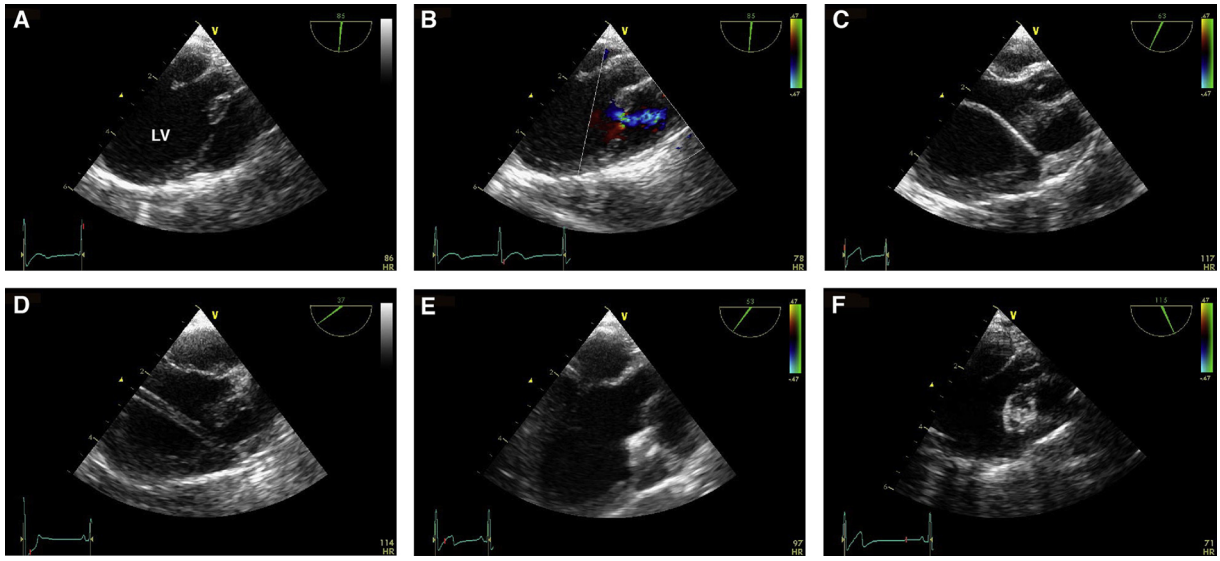

Fig. 2 Transesophageal echocardiographic images obtained intraoperatively demonstrating the VSD (A) with left-to-right shunting on color Doppler (B). A guide-wire can be visualized passing through the right ventricular free wall and crossing the septum into the left ventricular lumen (C). A vascular introducer was advanced over the wire into the left ventricular lumen (D). The first retention disc of the ADO II device has been deployed and is in contact with the left ventricular endocardial surface of the interventricular septum (E). Both retention discs have been deployed (F). LV = left ventricle.

through the VSD and into the left ventricular lumen (Fig. 2) being careful not to puncture the left ventricular free wall. Device size selection was based on pre-operative transthoracic echocardiographic measurements of the VSD that were confirmed with intraoperative TEE. An Amplatzer® Duct Occluder II (ADO II) device[g] (Fig. A, available in the online supplemental materials) was selected because the 4 mm waist diameter would approximate the measured diameter of the VSD (4.4 mm), the 6 mm device length would accommodate the septum, and the 10 mm diameter retention discs would not impinge on the aortic or tricuspid valve. The device was prepared by soaking it in saline, screwing it onto a delivery cable, and loading it into the introducer for delivery. With TEE guidance, the first retention disc was deployed in the lumen of the left ventricle and retracted until it was in contact with the left ventricular endocardial surface of the interventricular septum. The introducer was retracted into the right ventricle, and with continued gentle traction on the delivery cable, the waist and second retention disc were deployed. The second retention disc could be visualized on the epicardial surface which was attributed to the increased flexibility of the device allowing the disc to be retracted more than expected. The second disc was captured within the introducer and re-deployed in the proper position against the interventricular septum (Fig. 2). After device deployment, there was no transseptal flow seen on Color Doppler TEE. The device was released following confirmation of proper placement and verification that the aortic and tricuspid valve leaflets were not entrapped by the device. The introducer was removed, and the purse-string suture was tightened and tied. The pericardium was left open, and the chest was closed in a routine manner. Angiography was performed through a right jugular venous catheter to confirm device position and assess for residual shunting (Fig. B, available in the online supplemental materials).

Recovery was uneventful although the dog did develop hyperthermia (105 °F) but this resolved within 15 min after removal of the warming blanket. The following day, repeat thoracic radiographs (Fig. 3) and echocardiography (Fig. 4, Video 1) confirmed appropriate device location, no residual shunting, minimal changes in heart size (LVIDd 2.2 cm, LVIDs 1.6 cm, VHS 11.1), and normal outflow tract velocities. Multiple pleural fissure lines were noted on thoracic radiographs consistent with mild pleural effusion and considered normal following the thoracotomy. The dog received tramadol (2.0 mg/kg PO q 8—12 h) for pain management. Approximately 3 h post-operatively, clopidogrel (7.5 mg/kg PO q 24 h) and

[g] Amplatzer® Duct Occluder II, 9-PDA2-04-06, AGA Medical Corporation, Golden Valley, MN, USA.

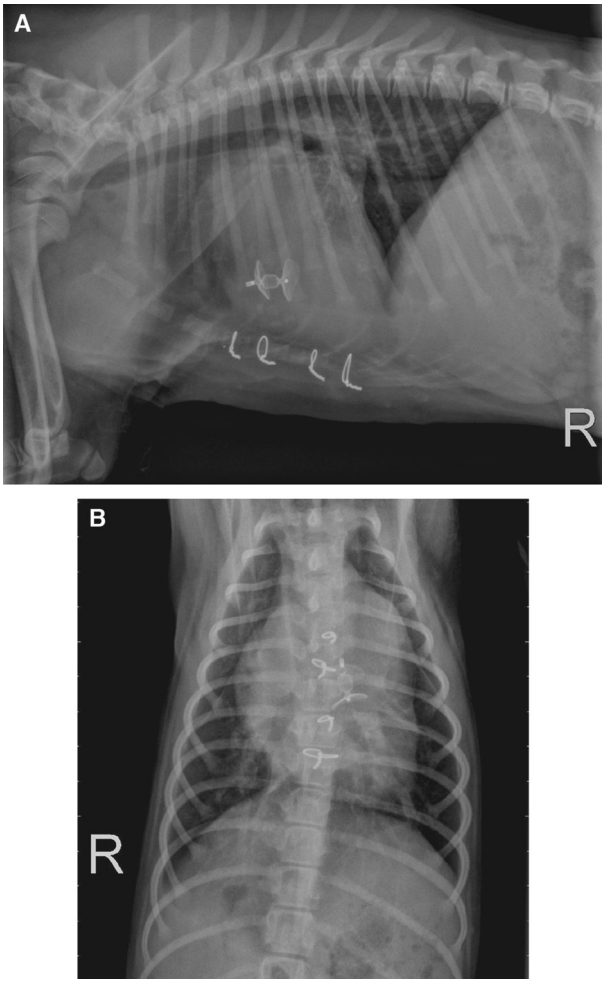

Fig. 3 Lateral (A) and dorsoventral (B) thoracic radiographs following ADO II device placement in a VSD in a dog.

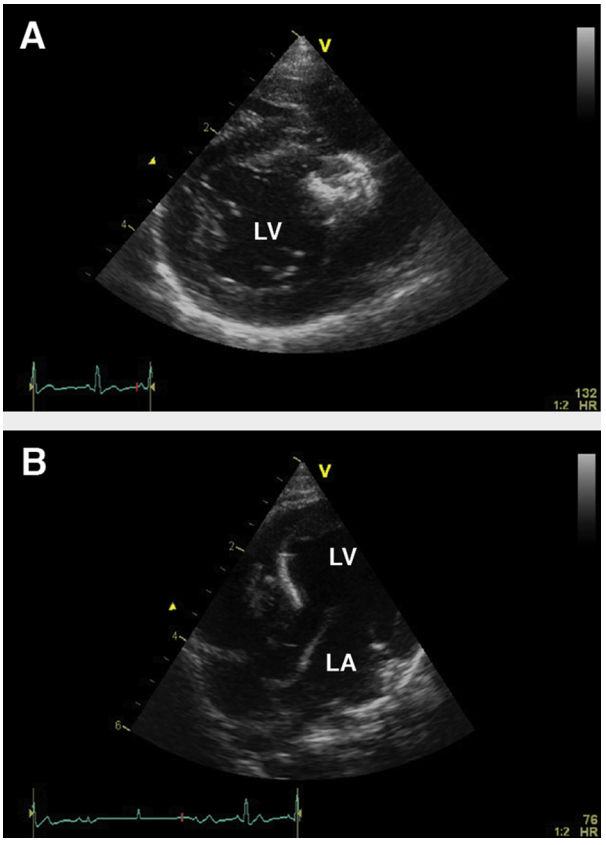

Fig. 4 Transthoracic echocardiographic images of the ADO II device from the right parasternal short-axis (A) and left parasternal long axis (B) views. LA = left atrium, LV = left ventricle.

aspirin (0.4 mg/kg PO q 24 h) were administered once the dog was fully awake to prevent thrombus formation on the device. Enalapril and furosemide were discontinued following the procedure. The owners were instructed to restrict activity for 4 weeks.

The dog was re-evaluated 17 days after device placement and was clinically doing well with a normal physical exam. Thoracic radiographs and echocardiography documented reverse remodeling (LVIDd 2.4 cm, LVIDs 1.5 cm, VHS 10.3) and proper placement of the device. A small soft tissue mass on the posterior wall of the left atrium, which was noted prior to surgery, appeared larger and more hyperechoic than on the previous exam (Fig. C, available in the online supplemental materials). Diagnostic tests were performed to assess for possible thrombotic complications. Complete blood count and biochemistries were within normal limits and the urine specific gravity was 1.012 g/dL (reference range, 1.015—1.045) without proteinuria. A coagulation panel was performed and documented normal prothrombin time, slightly prolonged partial thromboplastin time (10.9 s, reference range, 7.1—10.0 s), increased D-dimers (616 ng/mL, reference range, 116.2—371.5 ng/mL), and normal antithrombin activity (138%, reference range, >114%). Abdominal ultrasound findings included irregular hyperechoic regions in the cortices of both kidneys and a mildly increased resistive index in both kidneys suggestive of bilateral renal infarcts. As a precaution, the dog was admitted to the hospital and administered dalteparin (300 IU, SC, q 12 h) for 3 days. The dog was discharged with instructions for strict exercise restriction and to continue dalteparin, clopidogrel and aspirin until the next recheck evaluation.

The dog presented 11 days later and the owners reported no abnormalities while the physical examination remained normal. Abdominal ultrasonography documented an abnormal shape to the left kidney with a loss of the ventral cortical margin and multiple small hyperechoic regions

associated with the right renal cortex while the resistive index for both kidneys was within the normal range. The wall of the left atrium appeared thickened on echocardiography. The dog was discharged and was to continue the clopidogrel and aspirin for 6 months.

Four months later, the dog continued to do very well, and the physical examination and biochemistry panel remained normal. The coagulation panel, echocardiographic, and abdominal ultrasound findings were unchanged. Clopidogrel was discontinued, and aspirin was continued for 2 additional months. At the time of this writing, the dog continues to do well 11 months after the procedure.

Discussion

Ventricular septal defects in dogs are typically located in the upper portion of the septum in a perimembranous location and less often in the muscular septum.[5] The ventricular septum can be divided into membranous and muscular components, with the term perimembranous referring to defects within both components.[6] The VSD in the dog of this report was located in an outlet position and although it was fairly high in the septum, it was characterized as muscular and not perimembranous based on continuous muscular borders.[7] A muscular VSD in an outlet position is rare in humans.[7] Most notably, the VSD in the dog of this report was located well below the aortic valve with enough distance to prevent damage or impingement by a device. Transcatheter occlusion with embolization coils or an Amplatzer® muscular VSD occluder have been reported in a few dogs with hemodynamically important defects.[8–11] Ventricular septal defect closure is recommended in humans with a Qp:Qs greater than 2.0 and evidence of volume overload.[12] Transcatheter device closure of a VSD provides a less invasive approach when compared to surgery. Factors that complicate successful device closure include VSD size and location related to important adjacent structures, patient size, and device characteristics. Small patient size is associated with an increased risk of procedural complications during transcatheter closure of a VSD in humans,[13] and the ideal patient size for delivering standard VSD devices is greater than 8 kg.[14] A hybrid procedure combines surgical access to the defect and catheterization techniques to deliver the device.[15,16] It is a treatment option for children weighing less than 5 kg when vascular access and small patient size preclude device delivery and does not require cardiopulmonary bypass.[17] The small size of the dog in this report prevented either surgery or a catheter-based procedure alone as treatment options which lead the authors to consider a hybrid procedure. The authors have previously published experience using similar hybrid techniques for atrial septal defect (ASD) closure in dogs.[18,19] As with the ASD procedure, fluoroscopy alone is inadequate to optimally monitor catheter placement and device delivery thus a combination of TEE and epicardial imaging were essential to the success of the procedure. A sternotomy was performed in this dog for multiple reasons including surgeon preference so that the left ventricle was easily accessible if there was penetration or damage to the left ventricular posterior wall and to provide adequate exposure for epicardial echocardiography. A median sternotomy is a preferred approach for the hybrid procedure to close muscular VSDs in humans.[15,20]

The ADO II is a self-expanding device made of nitinol mesh with two symmetrical retention discs separated by a central waist. The delivery system for the ADO II is 4—5 Fr and is smaller than the 6—9 Fr system required for the Amplatzer® muscular VSD occluder. The ADO II was developed for patent ductus arteriosus closure in humans and has been used to close muscular and perimembranous VSDs in children.[21–24] The increased flexibility of the ADO II device and lengthier separation of the discs (waist) creates space for the interventricular septum. The range of device sizes is narrow limiting device use to VSDs smaller than 5.5 mm in diameter.[21] The flexibility of the device allows it to elongate and stretch across the septum with tension applied on the delivery cable. Deployment of the second retention disc epicardially has been reported in children, and occurred in this dog.[17] Deploying the device very slowly with minimal traction and allowing it to take shape within the right ventricle may prevent this from occuring.[17,21] Reported complications associated with device closure of VSDs include device embolization, wire or catheter perforation, and conduction abnormalities.[13–15]

The dog had evidence of thromboembolic complications without associated clinical signs. A thrombus was never documented associated with the ADO II device, however evidence of thromboembolism was observed during abdominal ultrasound. The ridge of tissue in the left atrium was present at initial examination. It appeared more prominent and hyperechoic at some point after discharge following device placement and the first recheck evaluation, which might have served as a substrate for thrombus formation. In humans, a

crescent shaped ridge of tissue in the wall of the left atrium that separates the left auricle from the left superior pulmonary vein is a normal variant known as the coumadin or warfarin ridge because it can be interpreted as a thrombus.[25] It is standard practice to administer antithrombotic medications at the time of deployment of an intracardiac device in humans although there is very little published in regards to hybrid procedures and strategies differ based on the type of procedure in part because antiplatelet therapy can complicate bleeding during surgery.[17,23,26] In children, aspirin therapy is recommended for 6 months following device placement.[13,17] Oral medications were initiated once the dog in this report was fully awake which was approximately 3 h after surgery. The clopidogrel dose for this dog was based on a loading dose and was higher than the reported maintenance dose of 2 mg/kg/day which was a function of tablet size.[h] Thrombus formation associated with an Amplatzer® atrial septal occluder has reported in one dog[19] despite aspirin therapy and this has lead to the authors' using a combination of clopidogrel and aspirin for 6 months after transcatheter ASD closure. Clinically, the dog in this report continues to do well 11 months following VSD closure and is monitored routinely. This report documents successful closure of a VSD in a small dog using an ADO II device with a hybrid technique.

Conflict of interest

No conflict of interest declared by any author.

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Supplementary data

Supplementary data associated to this article can be found at http://dx.doi.org/10.1016/j.jvc.2013.06.003.

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Video 1	Transthoracic and transesophageal echocardiographic images of a dog with a muscular VSD before and after hybrid closure with an Amplatzer® Duct Occluder (ADO) II device.

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