

Case Report



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Received 23 October 2018; received in revised form 2 May 2019; accepted 7 May 2019

KEYWORDS Canal; Endocardial cushion; Congenital **Abstract** A 9-month-old kitten with increased resting respiratory rate and exercise intolerance was diagnosed with a congenital partial atrioventricular septal defect causing pulmonary over circulation and presumed pulmonary hypertension based on echocardiogram. Invasive pressure measurements and contrast angiography confirmed this diagnosis. The cat underwent pulmonary artery banding under general anesthesia. Findings of echocardiogram 10 days postoperatively suggested reduced left-to-right shunt volume. Echocardiographic findings were static 4 months postoperatively.

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A 9-month-old 4.6-kg male castrated cat was presented on referral to Friendship Cardiology Specialists for increased sleeping respiratory rate. The cat was initially presented to his primary veterinarian soon after adoption for increased resting respiratory rate (measured at 50 breaths per minute by owners) but no murmur, arrhythmia, or gallop were ausculted. Owners were instructed to monitor him and return if there were any concerns. Five months later, the cat represented to his primary veterinarian for decreased exercise tolerance and persistent tachypnea. At that time, a new heart murmur was ausculted. Serum NT-Pro B-type natriuretic peptide concentration was elevated (170 pmol/L, reference 0-100 pmol/L), and a cardiology consultation was recommended.

 $[\]stackrel{\star}{\rightarrow}$ A unique aspect of the Journal of Veterinary Cardiology is the emphasis of additional web-based images permitting the detailing of procedures and diagnostics. These images can be viewed (by those readers with subscription access) by going to http://www.sciencedirect.com/science/journal/17602734. The issue to be viewed is clicked and the available PDF and image downloading is available via the Summary Plus link. The supplementary material for a given article appears at the end of the page. Downloading the videos may take several minutes. Readers will require at least Quicktime 7 (available free at http://www.apple.com/quicktime/download/) to enjoy the content. Another means to view the material is to go to http:// www.doi.org and enter the doi number unique to this paper which is indicated at the end of the manuscript.

https://doi.org/10.1016/j.jvc.2019.05.003

Abbreviations		
ASD	atrial septal defect	
AV	atrioventricular	
AVSD	Datrioventricular septal defect	
IV	intravenous	
MPA	main pulmonary artery	
PH	pulmonary hypertension	
VSD	ventricular septal defect	

On presentation, the cat had a grade II/VI sternal systolic murmur, heart rate of 180 beats per minute, and strong femoral pulses. Respiratory rate was 52 breaths per minute, and lung sounds were harsh without crackles or wheezes. Jugular veins did not appear overtly distended. Echocardiogram revealed a large (~ 7 mm) primum atrial septal defect (ASD) with continuous left-toright interatrial shunting with peak velocity 1.1 m/ s (peak pressure gradient 4.8 mmHg). There was loss of normal apical positioning of the right atrioventricular (AV) valve, a cleft left AV valve with bridging leaflets that appeared to insert directly on the septal crest, and mild systolic right and left AV valve regurgitations. Velocities of regurgitation were difficult to accurately assess, as the jets were eccentrically directed across one another. There was ventricular septal flattening and occasional septal bounce in diastole. The right atrium and ventricle were subjectively dilated, and right ventricular free wall thickness appeared increased. Pulmonary outflow velocity was greater than aortic outflow velocity (1.4 m/s and 0.7 m/s, respectively); the pulmonary valve appeared normal, and the main pulmonary artery (MPA) and branches were dilated (pulmonary valve annulus 1.2 cm, aortic valve annulus 1.1 cm). Pulmonary to systemic flow ratio (Qp/Qs) calculated using velocity time integrals of pulse wave Doppler and area of outflow tracts was 3.64, although AV valve regurgitation could have affected this assessment. The cat was diagnosed with a partial atrioventricular septal defect (AVSD) and suspected pulmonary hypertension (PH) due to pulmonary over circulation. The murmur was presumed due to AV valve regurgitations or mildly increased pulmonary outflow velocity secondary to the ASD (relative pulmonary stenosis). The cat was prescribed 5 mg of sildenafil (1.1 mg/kg) twice daily and 6.25 mg of spironolactone (1.36 mg/kg) once daily. Owners reported improved exercise tolerance and subjectively improved respiratory effort one week later, but the cat's resting respiratory rate remained elevated, and owners were keen to explore surgical options. Given the lack of locally available cardiopulmonary bypass and the risks and challenges involved with definitive patch repair in cats, palliative pulmonary artery banding was elected with aims to reduce pulmonary over circulation and mitigate further pulmonary vascular remodeling and irreversible PH.

The cat presented for surgery one month later. Examination findings were unchanged. Systemic blood pressure was normal (139 mmHg systolic). A complete blood count and chemistry panel showed no abnormalities. The cat became fractious and further preoperative diagnostics were postponed to be performed under general anesthesia. The cat received 100 mg of gabapentin (21.7 mg/kg) and 37.5 mg of trazodone (8.1 mg/kg) orally, then 10 mg of alfaxalone (2.2 mg/kg), 0.4 mg of hydromorphone (0.09 mg/kg), and 15 mg of ketamine (3.3 mg/kg) subcutaneously before intravenous (IV) catheter placement. An 18-gauge IV catheter was placed in each medial saphenous vein. The patient was induced with 10 mg of alfaxalone (2.2 mg/kg) IV, intubated with a 5F endotracheal tube, and maintained on isoflurane and oxygen and IV lactated ringer solution. Pulse oximetry after induction revealed oxygen saturation of 98%. Three-view thoracic radiographs were obtained (Fig. 1) and revealed cardiomegaly with dilated pulmonary vasculature consistent with pulmonary over circulation and hepatomegaly suspected because of venous congestion. Twelvelead electrocardiogram was obtained as per Kraus' modified Wilson's precordial placement as previously described [1] and revealed a sinus rhythm with mildly prolonged QRS duration and an axis deviation of -80° which could be due to extreme right axis deviation (right heart enlargement) or concurrent partial left and right bundle branch blocks (Fig. 2). Echocardiography was repeated. Color flow Doppler showed subjectively reduced degree of AV valve regurgitation and end-diastolic left-to-right interatrial flow. Otherwise, findings were static as described previously (videos 1, 2, and 3). Agitated contrast injected in the left medial saphenous vein did not reveal right-to-left shunt, and a negative contrast effect was noted from left-to-right shunt. The cat was placed in right lateral recumbency in the operating room, and the left lateral thorax and neck were shaved and prepared for aseptic surgery. The cat then became hypotensive (65 mmHg systolic) and subsequently desaturated (oxygen saturation 80%) and became bradycardic (heart rate 60bpm). Isoflurane was discontinued, 25ug of glycopyrrolate (5.4ug/kg) IV was administered, and a dopamine infusion was initiated at 23ug/minute (5ug/kg/

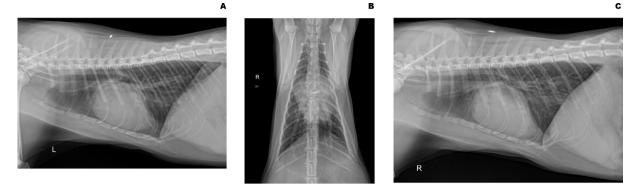


Fig. 1 Left lateral (A), dorsoventral (B), and right lateral (C) thoracic radiographs obtained under general anesthesia preoperatively in a cat with an atrioventricular septal defect and pulmonary hypertension. The cardiac silhouette is markedly enlarged, and the pulmonary arteries and veins are enlarged consistent with pulmonary over circulation. The pulmonary parenchyma is normal. There is hepatomegaly.

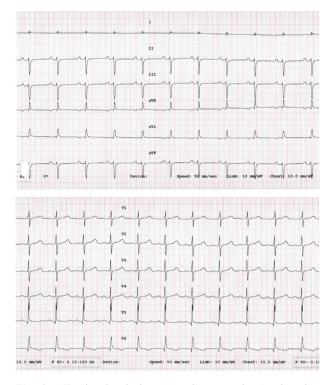


Fig. 2 Twelve-lead electrocardiogram obtained under general anesthesia preoperatively in a cat with an atrioventricular septal defect and pulmonary hypertension. There is a sinus rhythm with mean heart rate 140 bpm. The mean electrical axis is -80° , and QRS duration is mildly increased (0.05s) which could be due to extreme right axis deviation (right heart enlargement) or concurrent partial left and right bundle branch blocks.

min). Vital parameters returned to baseline. Infusions of fentanyl at 23ug/hour (5ug/kg/hour) and alfaxalone at 23ug/hour (5ug/kg/hour) were initiated, and isoflurane was not restarted. A left lateral 4th intercostal thoracotomy was performed

in a standard fashion. Hemostasis was maintained with unipolar cautery. The pericardium was incised ventral to the phrenic nerve through a 5-cm incision. The dorsal and ventral pericardia were loosely sutured to the incision to improve exposure to the heart base, and the PA was dissected just distal to the pulmonary valve. Sterile umbilical tape was passed around the MPA in preparation for banding (video 4). A jugular cutdown was performed. A 5F 5.5-cm introducer sheath^a was placed using an 18-gauge peripheral IV catheter and .035-inch guidewire. A 5F 50-cm Berman Angiographic Balloon catheter^b was advanced through the introducer sheath into the right ventricle. The catheter was connected to a pressure transducer on the surgical table at the level of the heart. Right ventricular systolic pressure was measured at 56 mmHg; then the catheter was retracted, and peak right atrial pressure was measured at 8 mmHg. The cat again became hypotensive and desaturated, and further attempts to perform invasive pressure measurements were aborted. Dopamine was increased to 35ug/minute (7.6ug/kg/min). Five milliliters of iodinated contrast^c was injected in the cranial cava through the angiographic catheter (video 5) and passed fairly equally into both left and right atria and then left and right ventricles. Retrograde contrast was seen in the azygous vein, cranial cava, and caudal cava. The pulmonary arteries appeared dilated. The umbilical tape was tied with a single throw, reducing the diameter of the MPA

^a Check-Flo Performer Introducer; Cook Incorporated, Bloomington, IN.

^b Arrow Berman Angiographic Balloon Catheter; Teleflex Incorporated, Morrisville, NC.

^c Omnipaque 300mgI/mL; GE Healthcare, Princeton, NJ.

by approximately 40%. To prevent the single throw on the umbilical tape from loosening, a circumferential 0 silk suture was tied around the umbilical tape. This silk suture applied compression to the single umbilical tape throw and increased friction on the umbilical tape to prevent it from loosening. The cat remained hypotensive and became bradycardic and received 0.09 mg of atropine (0.02 mg/kg) IV and 0.05 mg of epinephrine (0.01 mg/kg) IV. The balloon of the angiographic catheter was deflated in case it was contributing to hypotension because of impaired preload. The suture on the umbilical tape was loosened. Several minutes later, the cat's vital parameters had improved, and the suture around the MPA was retightened to reduce the diameter to approximately 40% (video 6). The pulmonary arteries appeared less dilated during systole, presumably because of reduction in pulmonary flow. Vital parameters remained stable with oxygen saturation of 98%. Five-milliliters of contrast was injected through the angiographic catheter into the cranial vena cava (video 7). Contrast passed into the right atrium and ventricle, and negative contrast effect was noted within the right atrium. The pulmonary arteries appeared less dilated. Repeat invasive pressure measurements were planned, but the cat's systolic blood pressure again began to decrease, and all further diagnostics and interventions were aborted. The thorax was flushed with saline, and the pericardium was closed with 4-0 polydioxanone suture (PDS) in a cruciate pattern. A 14-gauge MILA thoracostomy tube was placed through the caudal rib spaces and secured with 2-0 nylon. The thoracotomy was closed in a standard fashion. The introducer sheath was replaced with a triple lumen catheter. The cranial jugular was ligated, and the distal stay suture was tied loosely over the catheter and vessel to help secure the position. The cat recovered slowly but uneventfully and received one dose of 37ug of naloxone (8ug/kg) IV. Overnight, continuous electrocardiogram showed a sinus tachycardia (200-220 bpm) without ectopy. The following morning, the patient had mild tachypnea (50 breaths per minute) and tachycardia (210 beats per minute), and pulse oximetry revealed oxygen saturation of 90% on room air. Dopamine and fentanyl infusions were weaned and discontinued, and his chest tube and triple lumen catheter were removed. He was discharged three days postoperatively with 100 mg of gabapentin (21.7 mg/kg) three times daily, 37.5 mg of trazodone (8.1 mg/kg) twice daily, 50 mg of

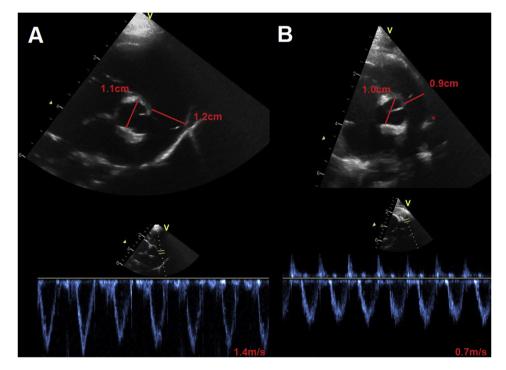


Fig. 3 Right parasternal short axis base echocardiographic images at initial visit (A) and 10 days after pulmonary band placement for partial atrioventricular septal defect (B). Initial images show a dilated main pulmonary artery with increased right ventricular outflow tract velocity consistent with the left-to-right shunt. Recheck images show reduction in pulmonary valve annulus diameter with normal outflow tract velocity supporting reduced left-to-right shunt. The asterisk denotes the location of the band.

cefpodoxime (10.8 mg/kg) once daily, and continued sildenafil and spironolactone as previously prescribed. He was kept cage rested. At recheck 10 days postoperatively, the owners reported normal respiratory effort and respiratory rates between 40 and 50 breaths per minute. On examination, the cat's systolic murmur was louder (grade III/VI) and his respiratory rate was 40 breaths per minute. Systemic blood pressure remained stable. Pulse oximetry was not possible due to poor signal quality. The cat was sedated for additional diagnostics (1.8 mg of butorphanol (0.39 mg/kg), 10 mg of alfaxalone (2.2 mg/kg), and 7 mg of ketamine (1.5 mg/kg) SQ). A 6-lead electrocardiogram showed no changes. Recheck thoracic radiographs showed static cardiomegaly and pulmonary vascular dilation. Recheck echocardiogram revealed a supravalvular pulmonary stenosis (minimum diameter 0.5 cm) consistent with the pulmonary band. There was aliasing of color flow Doppler in this region, and continuous wave interrogation revealed a peak velocity of 2.6 m/s consistent with a pressure gradient of 27 mmHg, with the step-up occurring at the level of the band (right ventricular outflow tract velocity 0.7 m/s, MPA just before the band 0.9 m/s) (video 8). The pulmonary artery annulus diameter was reduced in size (0.9 cm), and the right ventricle appeared less dilated (Fig. 3). Interatrial flow remained left to right with slightly reduced peak flow velocity compared with initial examination (0.85 m/s; pressure gradient 2.9 mmHg) and was no longer completely continuous; the enddiastolic component of flow was reduced compared with initial examination. Pulmonary to systemic flow ratio was recalculated as described previously (acknowledging similar limitations) and was markedly improved (1.16). The cat was continued on spironolactone and sildenafil as previously prescribed. He presented for another recheck 4 months postoperatively. He was doing well at home with no changes in his condition. Echocardiogram was repeated, and findings were static. The cat was sedated as previously described for thoracic radiographs, and static cardiomegaly and pulmonary vascular distension were present. In addition, there was a focal small patchy interstitial pattern in the left caudal lung field. Differincluded entials atelectasis or pulmonary thromboembolism. While the cat had no respiratory signs of pulmonary thromboembolism or echocardiographic evidence of spontaneous echocontrast or emboli, given the right heart dilation which could predispose to embolus formation, he was started on 18.75 mg of clopidogrel (4.1 mg/kg)

by mouth once daily. Spironolactone and sildenafil were continued as previously prescribed.

Discussion

Atrioventricular septal defects, also known as canal or endocardial cushion defects, are uncommon in cats (2-10% of cats with congenital cardiac disease) [2,3]. The AV septum is a small portion of the membranous septum that separates the lumen of the left ventricle from the right atrium and is formed by the fusion of the endocardial cushions with the septum primum [4,5]. Partial AVSDs involve either a primum ASD or an inlet VSD, whereas complete AVSDs result in concurrent ASD and VSD [2,6]. Disruption of the AV septum results in a bridging AV valve with attachments to the muscular interventricular septum and eliminates the normal apical displacement of the right AV valve [6]. Flow across a large ASD is dictated by the relative compliances of the ventricles; the right ventricle is more compliant than the thicker left ventricle, and thus, interatrial flow is left to right throughout most of the cardiac cycle [4]. Pulmonary over circulation induces protective pulmonary vasoconstriction, PH, and potentially clinically signs such as dyspnea or syncope [7]. The increased respiratory rate of the cat of the present report was suspected due to PH, which was supported by echocardiogram, thoracic radiographs, and invasive pressure measurements. The severity of PH may have been underestimated due to anesthetic drugs and oxygen. Phosphodiesterase inhibitors such as sildenafil may be beneficial in reducing pulmonary arterial pressure and exercise capacity initially [8]. However, chronic pulmonary over circulation results in endothelial damage and a cascade of signaling pathways that may alter extracellular matrix induce proliferation of smooth muscle cells and promote connective tissue synthesis, resulting in fibrosis and plexiform lesions and potentially irreversible PH [7]. Reversible PH has been reported in a cat with a patent ductus arteriosus treated with oxygen and sildenafil [9]. and plexogenic pulmonary arteriopathy has been reported in a cat with a non-restrictive VSD that died suddenly at 8 years of age [10]. In a previous study, 5-year survival rate for cats with AVSDs was 53%, half of all cats were symptomatic, and causes of death included congestive heart failure (CHF) or sudden death, perhaps because of ventricular arrhythmias or severe PH [2]. Given the young age, clinical signs of PH with subjectively positive clinical response to sildenafil, hepatomegaly

suggesting early right heart decompensation, and possibility of mitigating irreversible pulmonary vascular damage, the decision was made to pursue palliative surgery for the cat of the present case. Human patients with AVSD typically undergo surgical closure and cleft AV valve repair within the first months of life, and palliative banding is performed before definitive repair for symptomatic infants with complete AVSD [5]. Cardiopulmonary bypass for definitive repair of AVSDs with a patch graft has been reported in dogs [11-13], and there is a single case report of AVSD repair in a kitten that was suspected to have PH and died 5 h following surgery [14]. Pulmonary banding has been reported as palliative treatment for VSD in cats [15,16]. Pulmonary banding is also performed in human pediatric patients with complex congenital disease as staged palliation before definitive repair [5]. Pulmonary banding for VSD increases right ventricular afterload and increased right ventricular systolic pressure, thereby reducing interventricular pressure gradient and left-to-right systolic shunting of blood. Potential benefit of a pulmonary band for an atrial-level shunt is less intuitive with a more indirect result; increased afterload shifts the right ventricular pressure volume loop rightward causing an increase in right ventricular diastolic pressure. This increase in diastolic pressure may reduce left-to-right interatrial flow due to impaired right ventricular compliance. To the authors' knowledge, pulmonary banding has not previously been reported for partial AVSD in a human or veterinary patient. For treatment of VSD, recommendations for the reduction in MPA diameter range from 30 to 60% [14,15,17], forming the basis for the aimed 40% reduction by visual estimation in the present case. Recheck echocardiogram suggested this goal had been achieved based on the minimal diameter measured at the band. Several guidelines for banding have been put forth to help ensure adequate palliation without negative consequences; the risks of excessive constriction of the MPA are shunt reversal or right ventricular failure and congestion [17]. A distal pulmonary artery catheter can be used to determine that pulmonary pressures have improved, although with permanent vascular remodeling, pressures will not normalize [17]. A decrease in pulmonary artery oxygen saturation by 5-10% may signal improved left-to-right shunting, and transesophageal echocardiography can be helpful in documenting undesirable shunt reversal [17]. The cat of this report showed only left-toright shunting of blood with agitated saline study before surgery as expected. Similarly, color flow Doppler echocardiogram showed only left-to-right ASD flow: however, the end-diastolic component of interatrial flow was reduced under anesthesia as compared with initial awake examination. In addition, and unexpectedly, the initial angiogram showed a more balanced interatrial shunt. These findings were suspected because of vasodilation hypotension under anesthesia causing and increased left ventricular compliance, thereby reducing left-to-right interatrial flow. On reviewing the anesthetic record, the cat's body temperature was low throughout the procedure (initial 90.6 °F and in recovery 84.9 °F as measured by esophageal temperature probe). Hypothermia, inhaled anesthetic gases, or the presence of the inflated angiographic balloon impeding preload could have contributed to the cat's hypotension. After banding, repeat angiogram indicated no significant right-to-left interatrial shunting. However, the dynamic systemic blood pressure in the present case and lack of a comparative initial angiographic study under conditions of normotension made intraoperative assessment of efficacy of surgical treatment via angiography or invasive pressure measurements challenging. Intracardiac oximetry was not performed, and transesophageal echocardiography was not available. At follow-up examination, the cat had a louder murmur secondary to the iatrogenic supravalvular pulmonary stenosis. Recheck echocardiographic findings of reduced interatrial flow velocity, decreased right ventricular dilation, decreased right ventricular outflow tract velocity, and reduced pulmonary to systemic flow ratio supported reduced left-to-right shunting. Thoracic radiographs showed persistently dilated pulmonary vasculature; this may represent permanent vascular remodeling but will be reassessed at follow-up visits. Repeat invasive pressure measurements were not performed, and it is unknown whether the band helped reduce PH. In addition, while no shunt reversal was documented four months following surgery, the patient may be at lifelong risk for this occurring. This report illustrates successful pulmonary band placement for AVSD in a cat, but long-term outcome is not yet known. Anesthetic considerations for pulmonary artery banding include maintaining systemic vasoconstriction and normothermia, and ensuring diagnostic catheters do not impede preload to prevent desaturation and aid in comparative invasive pressure assessment and angiography.

Conflict of Interest Statement

The authors do not have any conflicts of interest to disclose.

Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jvc.2019.05.003.

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Video table			
Video 1	Right parasternal long-axis transthoracic echocardiogram performed under anesthesia in a cat with partial atrioventricular septal defect.	There is a large primum atrial septal defect. There are bridging atrioventricular valve leaflets and loss of normal apical displacement of the right atrioventricular valve. The right atrium is dilated with bowing of the atrial septum toward the left atrium. The right ventricle is dilated, and the right ventricular free wall thickness appears increased. There is ventricular septal flattening. The right pulmonary artery is dilated.	
Video 2	Right parasternal short-axis transthoracic echocardiogram performed under anesthesia in a cat with partial atrioventricular septal defect.	There is a cleft left atrioventricular valve. The right ventricle is dilated.	
Video 3	Right parasternal long-axis transthoracic echocardiogram with color flow Doppler performed under anesthesia in a cat with partial atrioventricular septal defect.	There is low-velocity left-to-right flow across a large primum atrial septal defect. Pulmonary venous flow appears prominent.	
Video 4	Surgical approach to pulmonary band placement in a cat with partial atrioventricular septal defect.	Umbilical tape is in place around a dilated main pulmonary artery. Note: in this video, ventral is down and dorsal is up.	
Video 5	Initial angiogram of a cat with partial atrioventricular septal defect.	There is a 5F Berman angiographic balloon catheter in the cranial vena cava with the balloon inflated. Contrast is seen retrograde in the azygous vein and cranial cava which may be due to the inflated balloon or increased right heart pressure or a combination. Contrast fills the right atrium and left atrium fairly equally before passing into the left and right ventricles and is seen retrograde filling the caudal vena cava. Contrast highlights a dilated main pulmonary artery and branches.	
Video 6	Pulmonary band in place in a cat with partial atrioventricular septal defect.	The umbilical tape has been sutured in place, reducing the main pulmonary artery diameter by approximately 40%. Note: in this video, dorsal is down and ventral is up.	
Video 7	septal defect after pulmonary band placement.	There is a 5F Berman angiographic balloon catheter in the cranial vena cava with the balloon partially inflated. Contrast is seen retrograde in the cranial cava but not the caudal cava or azygous vein. Contrast enters the right atrium, and there is negative contrast effect from left-to-right interatrial flow. The main pulmonary artery and branches show less contrast enhancement compared with initial angiogram which could be due to the pulmonary band, contrast dilution from left-to-right shunt, or a combination.	
Video 8	Right parasternal short-axis echocardiogram of a cat 10 days after pulmonary band placement for partial atrioventricular septal defect.	The pulmonary valve annulus is reduced in size compared with initial echo. There is aliasing of color flow Doppler in the main pulmonary artery consistent with supravalvular stenosis due to the pulmonary artery band.	

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