Balloon valvuloplasty for palliative treatment of tricuspid stenosis with right-to-left atrial-level shunting in a dog

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- Tricuspid stenosis is a rare condition in dogs that may result from tricuspid dysplasia.
- In dogs with tricuspid stenosis, high right atrial pressures may cause right-to-left atrial-level shunting and chronic cyanosis. Secondary polycythemia is one of the most important clinical sequelae of chronic cyanosis and results in hyperviscosity syndrome.
- There is limited information available concerning the treatment and prognosis of dogs with tricuspid stenosis. Balloon valvuloplasty offers a means to palliate the clinical signs without having to perform thoracic surgery.

A 3.75-year-old castrated male Chesapeake Bay Retriever that weighed 24.5 kg (53.9 lb) was referred for evaluation of exercise intolerance, tachypnea, and cyanosis of 2 months' duration. In the 48 hours prior to referral, the owner had perceived an increase in the dog's water consumption and urine output; hematuria and a decrease in appetite were observed coincident with development of these signs. The Hct of blood samples collected 2 days and approximately 2 months prior to referral were 66 and 63%, respectively; the total protein concentration of both samples was < 6 g/dl. Two days prior to referral, the serum creatinine concentration was slightly high (1.88 mg/dl; reference range, 0.50 to 1.80 g/dl). A urinalysis had been performed on a sample obtained by catheterization; the urine specific gravity was 1.015, the pH was 6.5, the protein concentration was > 200 mg/dl, and moderate concentrations of blood and bilirubin were evident. *Escherichia coli* was cultured from this sample, and appropriate antibiotic treatment had been initiated. Thoracic radiographs were available for review. The cardiac silhouette was enlarged, with right-sided emphasis, and pulmonary hypoperfusion was evident (**Fig 1**).

On initial examination at the referral hospital, rectal temperature was normal, and pulse rate was 150 beats/min. The dog was panting, and there was generalized cyanosis of the mucous membranes. The first and second heart sounds were clear, and no murmurs were heard; a third transient sound, which could not be further defined, was also heard on cardiac auscultation. The PCV was 75%, with a total protein concentration of 6.8 g/dl.

Electrocardiography revealed sinus rhythm with

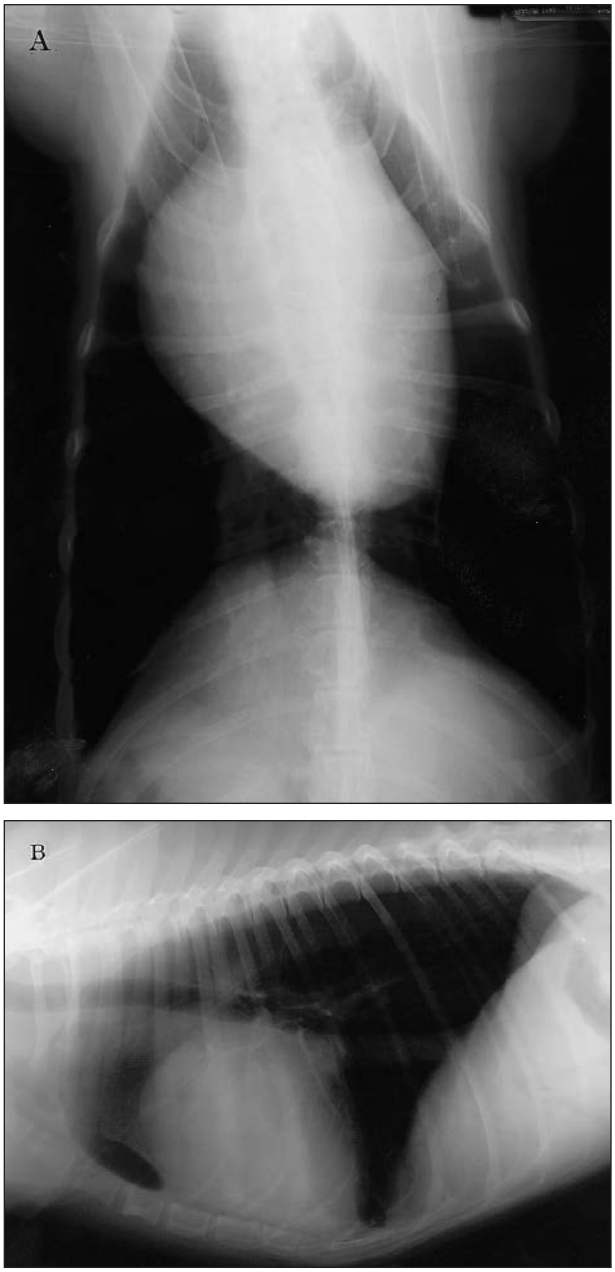

Figure 1—Ventrodorsal (A) and lateral (B) radiographic projections of the thorax of a Chesapeake Bay Retriever with severe tricuspid stenosis. The cardiac silhouette is large, and the right atrium is prominent. Pulmonary hypoperfusion is evident.

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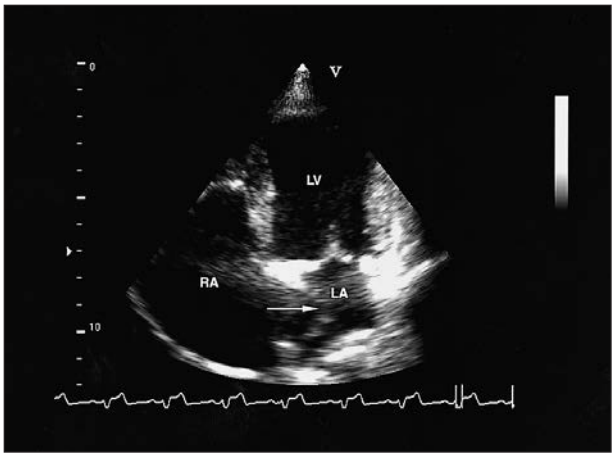

Figure 2—Left caudal (apical) parasternal location, 4-chamber echocardiographic view of the heart of the dog in Figure 1. Notice the right atrial enlargement and leftward bowing of the interatrial septum (arrow). RA = Right atrium. LA = Left atrium. LV = Left ventricle.

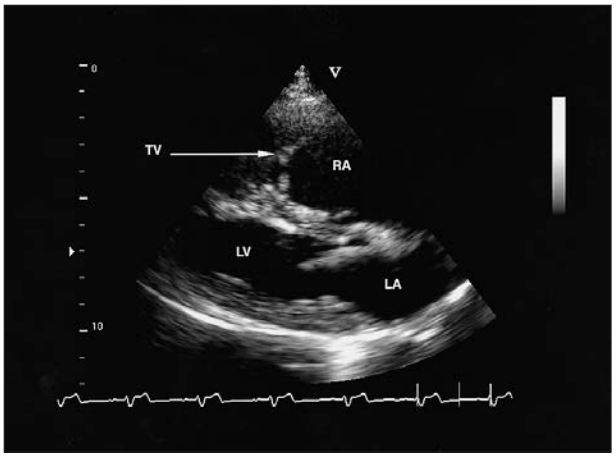

Figure 3—Right parasternal location, long-axis 4-chamber echocardiographic view of the heart of the dog in Figure 1. The image was obtained during diastole. The tricuspid valve (TV) leaflets (arrow) appear adhered to each other and poorly opened at a time when the normal mitral valve leaflets are widely separated. The chordal attachments of the tricuspid valve are abnormal, and the point of tricuspid leaflet apposition is apically displaced.

an intraventricular conduction delay compatible with right bundle branch block; duration of the QRS exceeded 80 milliseconds, the mean electrical axis was directed rightward, and there were S waves in leads I, II, III, and aVF. The P waves were abnormally broad and tall, with a duration of 60 milliseconds and an amplitude of 0.5 mV. Heart rate was 68 beats/min.

Two-dimensional echocardiography revealed moderate enlargement of the right atrium. The tricuspid valve apparatus was structurally abnormal, as was the motion of the tricuspid leaflets. The right ventricular papillary muscles were thick, and the chordae tendineae were abnormally short. Some segments of the valve leaflets appeared to insert directly onto the papillary muscles. The valve leaflets were thicker than normal, and their motion appeared restricted. In some planes, diastolic doming of the tricuspid valve leaflets was evident. The point of leaflet apposition was apically displaced. Leftward bowing of the atrial septum and basilar aspect of the interventricular septum was also observed (**Fig 2 and 3**). Because the anatomic relationships between the right atrium, tricuspid valve, and right ventricle were distorted, it was difficult to evaluate right ventricular size; however, in some planes, the right ventricle appeared to be mildly enlarged relative to the size of the left ventricle. M-mode electrocardiography was also performed. The diastolic left ventricular dimension (34.6 mm) was smaller than expected on the basis of the dog's body weight (predicted dimension based on a linear regression equation derived from values for healthy dogs[1]; 39.9 mm). However, left ventricular systolic performance was normal (left ventricular fractional shortening, 31.6%). Motion of the tricuspid valve and basilar aspect of the interventricular septum was abnormal. The normal caudal movement of the parietal leaflet toward the septum did not follow the early diastolic excursion; instead, the motion of the leaflet described a plateau during diastole. Because the basilar aspect of the interventricular septum was effectively incorporated into the right atrium, the motion of this structure was also abnormal. During diastole, the septum moved toward the left ventricular caudal wall in a paradoxical fashion that, presumably, reflected high diastolic pressures proximal to the tricuspid orifice (**Fig 4**).

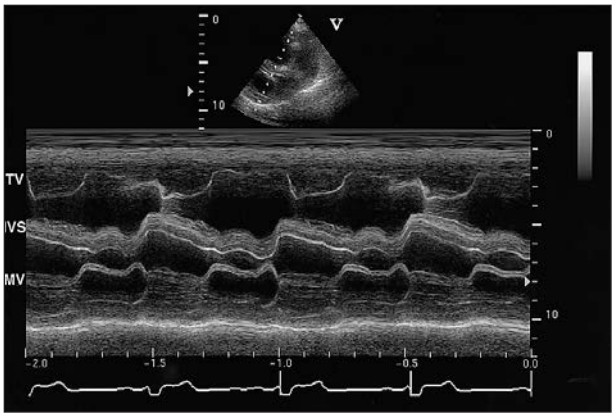

Figure 4—M-mode echocardiogram of the dog in Figure 1. The motion of the parietal tricuspid valve leaflet (TV) and interventricular septum (IVS) is abnormal. The basilar aspect of the IVS was effectively incorporated into the right atrium; paradoxical motion of the IVS toward the caudal wall of the left ventricle during diastole was likely a result of high diastolic pressure in the right atrium. During early diastole and during atrial contraction, right atrial pressure exceeded left ventricular pressure, causing the septum to move towards the caudal left ventricular wall. In systole, left ventricular pressure exceeded pressure within the right atrium, the left ventricle assumed a more normal configuration, and the interventricular septum moved away from the caudal left ventricular wall. The parietal tricuspid valve leaflet described a plateau, reflecting persistence of the right atrioventricular pressure gradient during diastole. MV = Mitral valve.

Color-flow Doppler echocardiography revealed evidence of mild tricuspid valve regurgitation and diastolic flow disturbance at the tricuspid orifice. Spectral Doppler echocardiographic assessment demonstrated tricuspid stenosis; peak transtricuspid velocity (2.15 m/s; reference range, < 1 m/s) followed atrial contraction and was abnormally high. Deceleration of early diastolic ventricular filling was prolonged. The mean and maximal diastolic pressure gradients across the tri-

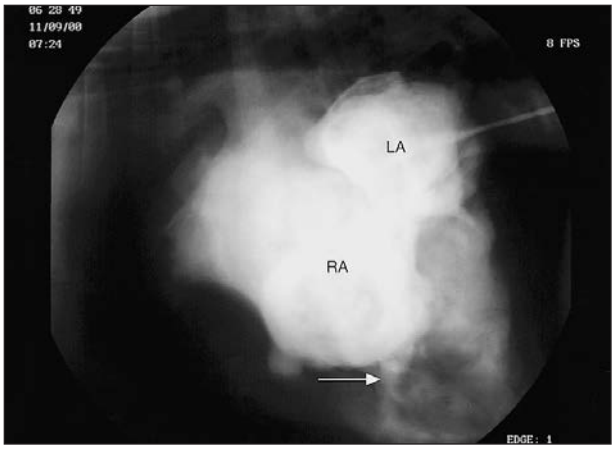

Figure 5—Digital angiogram obtained following injection of contrast material into the right atrium of the dog in Figure 1. The right atrium (RA) and left atrium (LA) are both opacified because of an interatrial communication. The stenotic jet (arrow) is visible, as is a portion of the catheter in the caudal vena cava.

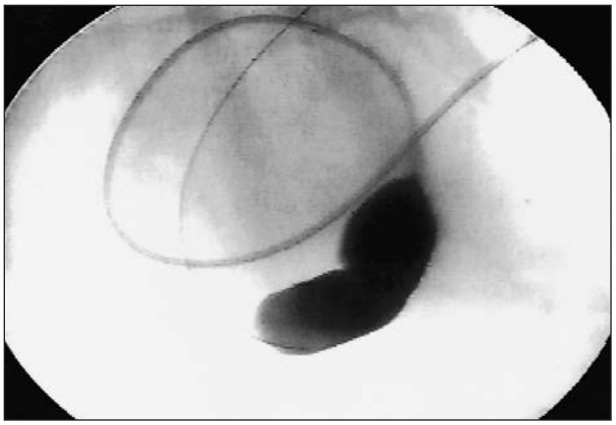

Figure 6—Fluoroscopic image obtained during balloon valvuloplasty of the stenotic tricuspid valve in the dog in Figure 1. The indentation or "waist" disappeared following complete inflation of the balloon.

cuspid valve were calculated to be 5.42 mm Hg (reference range, 0 to 1.5 mm Hg) and 18.65 mm Hg (reference range, < 4 mm Hg), respectively. Contrast echocardiography was performed by injecting agitated saline (0.9% NaCl) solution into a cephalic vein. A large right-to-left atrial-level shunt was evident. On the basis of the echocardiographic findings, a diagnosis of cyanotic heart disease characterized by tricuspid stenosis and atrial-level shunting was made. Tricuspid balloon valvuloplasty was recommended and scheduled for 2 weeks later. The dog was discharged to the owner's care, and therapeutic phlebotomy was performed by the referring veterinarian about 24 hours later.

Two weeks later, the dog was returned for balloon valvuloplasty. The Hct was 63% at this time. The dog was sedated with diazepam and oxymorphone, and anesthesia was induced with diazepam and etomidate. An endotracheal tube was placed, and anesthesia was maintained with isoflurane. A 5-F vessel sheath-introducer system was placed percutaneously into the right femoral vein, and a 5-F MPA catheter was advanced to the right atrium under fluoroscopic guidance. Right atrial pressures were as high as 16 mm Hg during atrial contraction. Attempts to advance the catheter into the right ventricle were unsuccessful.

In an attempt to clarify the anatomy of the tricuspid apparatus, 25 ml of iodinated contrast medium was infused into the right atrium through a 5-F Berman catheter, using a pressure injector. This study confirmed the echocardiographic findings of tricuspid stenosis with severe right atrial enlargement and atrial-level shunting (**Fig 5**). The 5-F sheath-introducer was then exchanged over a guidewire for a 9-F system. The tip of a 7-F flow-directed monitoring catheter was placed in the right ventricle. Right ventricular pressure was 18 mm Hg during systole and 0 mm Hg during diastole, confirming the transtricuspid pressure gradient. A 0.035-in 260-cm-long exchange wire was advanced into the right ventricle. However, placement of the wire was not sufficiently secure to allow exchange of catheters. At this time, the flow-directed catheter was advanced into the pulmonary artery; the exchange wire was used to stiffen the catheter and aid in catheter manipulation. The tip of the exchange wire was left in place in the pulmonary artery, and the catheter and sheath system were removed. An attempt was made to advance a 14 F balloon dilation catheter that carried a 4-cm-long balloon with an **outer diameter (OD)** of 30 mm over the wire and into the caudal vena cava. However, this proved impossible, despite the use of the dilator component of a 14-F vessel sheath to enlarge the site of vessel entry. An 11-F balloon dilation catheter that carried a 5-cm-long balloon with an OD of 25 mm was advanced to the level of the tricuspid valve and inflated 4 times (**Fig 6**).

Following balloon inflation, there was a decrease in right atrial pressure (**Fig 7**) that was accompanied by a marked increase in oxygen-hemoglobin saturation and arterial oxygen tension. The arterial oxygen tension was 49.2 mm Hg prior to balloon inflation and 456.2 mm Hg after valvuloplasty; the dog was breathing 100% oxygen when arterial blood samples were obtained. Angiography was performed, and magnitude of the atrial-level shunting was markedly decreased. Hemostasis was effected through direct pressure, and anesthetic recovery of the dog was routine.

The day following the procedure, the dog had a grade-II/VI systolic plateau-shaped murmur with the point of maximal intensity at the right cardiac apex. Results of 2-dimensional and M-mode echocardiography were similar to results obtained prior to balloon valvuloplasty, except that there was increased mobility of the tricuspid valve leaflets and a suggestion that a right ventricular papillary muscle or chorda tendineae had been ruptured. Moderate tricuspid regurgitation was now evident during Doppler echocardiography. During spectral Doppler echocardiography, peak transtricuspid velocity (1.13 m/s) during diastole was lower than previous measurements, and deceleration time of early diastolic right ventricular filling was decreased (**Fig 8**). Mean and maximal diastolic pressure gradients across the tricuspid valve were 2.10 and 5.12 mm Hg, respectively. Contrast echocardiography with agitated saline solution revealed a subjective decrease in right-to-left shunting, compared with

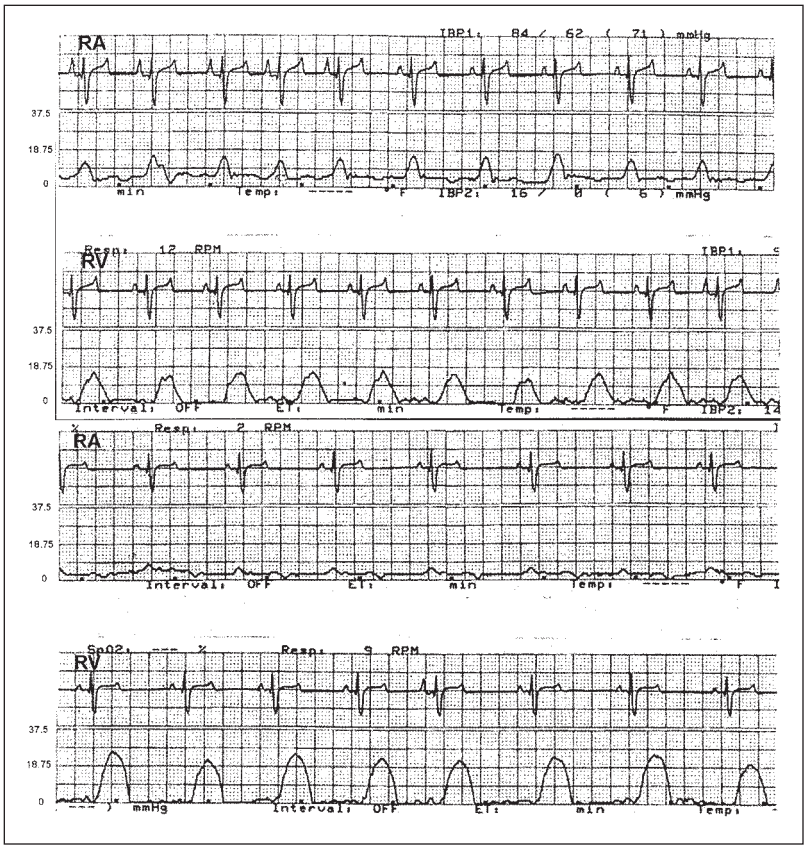

Figure 7—Right atrial and right ventricular pressure recordings obtained from the dog in Figure 1 before (top 2 panels) and after (bottom 2 panels) tricuspid balloon valvuloplasty. Following the procedure, the right atrial pressure decreased, as did the atrioventricular pressure gradient. The right ventricular systolic pressure increased following the procedure, probably as a result of improved ventricular filling. RA = Right atrium. RV = Right ventricle. Paper speed = 25 mm/s; 1 cm = 18.75 mm Hg. Recordings obtained before valvuloplasty were only approximately contemporaneous; recordings obtained after valvuloplasty were recorded during withdrawal of the catheter from the right ventricle to the right atrium.

results of contrast echocardiography performed before balloon valvuloplasty. At this time, heart rate was 45 beats/min. Electrocardiographically, the rhythm was sinus bradycardia with pauses terminated by ventricular escape complexes. The bradycardia was believed to have resulted from administration of oxymorphone; the dog had received three 1-mg doses of oxymorphone SC at 6-hour intervals following balloon valvuloplasty.

The dog was discharged to its owner and was returned to the veterinary teaching hospital 30 days later for reevaluation. The owner reported marked improvement in exercise tolerance, reduction in tachypnea, and increased activity. The PCV was 53%. The peak transtricuspid velocity during diastole (1.75 m/s) was increased, compared with velocity 24 hours after valvuloplasty, but was still lower than velocity before balloon valvuloplasty. Mean and maximal diastolic pressure gradients across the tricuspid valve were 5.24 and 12.42 mm Hg, respectively. Heart rate was 98 beats/min. Contrast echocardiography revealed only a small amount of right-to-left atrial-level shunting. The improvement in exercise tolerance was, in the perception of the owner, sustained; the Hct was 49% 2, 4, and 6 months after balloon valvuloplasty. Approximately 1 year after the procedure, the dog was observed recumbent shortly after running out of the owner's field of view. The dog recovered promptly, and 2 days later, the Hct was 55%, and the total protein concentration was 6.37 g/dl. Three weeks later, the Hct was 50%, and the total protein concentration was 6.0 g/dl. At the time of writing, echocardiographic reevaluation had not been performed; however, the owner believed the dog to be doing well, and additional episodes had not been observed.

Tricuspid stenosis (TS) is rare in dogs, and only a few cases[2-5] have been reported in the literature. To the authors' knowledge, TS with secondary central cyanosis in a dog has not been reported previously. Tricuspid stenosis may result from tricuspid dysplasia, a congenital valvular malformation that usually results in tricuspid regurgitation.[6] Acquired TS in dogs has not been reported, and this fact combined with the echocardiographic appearance of the tricuspid apparatus in the dog described in the present report led us to believe that the lesion was congenital. With TS, right atrial pressure increases commensurate with severity of the obstruction, and a pressure gradient is established across the valve. The pressure gradient persists throughout diastole but is augmented during atrial contraction. Tricuspid stenosis results in pressure overload of the right atrium and, potentially, atrial dilatation, high venous pressures, and right-sided congestive heart failure.

Cyanotic heart defects are those that result in shunting of deoxygenated blood into the systemic circulation. High right atrial, pulmonary artery, or right ventricular pressures are generally a prerequisite for development of right-to-left shunting. In dogs, congenital cyanotic heart disease is most commonly associated with severe pulmonic stenosis or high pulmonary vascular resistance.[7] In the dog described in the present report, TS resulted in high right atrial pressures, which caused the right-to-left atrial-level shunting. Secondary polycythemia is one of the most important clinical sequelae of chronic cyanosis and results in hyperviscosity syndrome.[7]

In the dog of this report, severe TS was associated with right-to-left atrial-level shunting. Although this may have resulted from a combination of tricuspid valve dysplasia and a congenital atrial septal defect, it seems more likely that the primary defect was tricuspid valve dysplasia causing high right atrial pressures, which, in turn, resulted in patency of the foramen ovale. The foramen ovale is the fetal interatrial communication that allows preferential shunting of the caudal venous return to the systemic circulation. In the immediate postnatal period, left atrial pressures increase, exceeding those in the right and forcing the

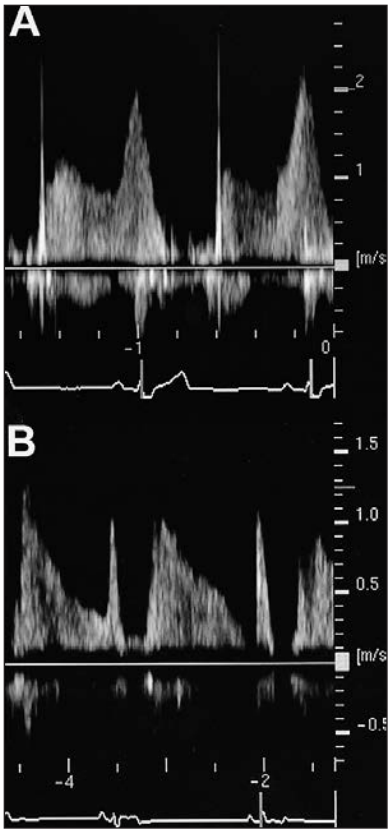

Figure 8—Spectral Doppler echocardiographic recordings of the tricuspid valve of the dog in Figure 1 before (A) and after (B) tricuspid balloon valvuloplasty. Prior to the procedure, peak diastolic velocity was in excess of 2 m/s, and deceleration of early diastolic ventricular filling was markedly prolonged. Following the procedure, peak diastolic velocity was reduced, and the deceleration of early diastolic ventricular filling was more rapid. Velocity scale and sweep-speed of the 2 recordings were different. Both recordings were pulsed Doppler echocardiographic recordings, but the recording obtained before valvuloplasty was a high pulse repetition frequency recording.

septum primum, which forms the valve of the foramen ovale, against the septum secundum.[8] This results in functional closure of the foramen ovale that is followed in most individuals by fusion of septal components and mechanical closure. High right atrial pressures can lead to progressive enlargement of the foramen ovale.[8]

Cyanosis resulting from right-to-left atrial-level shunting was the clinical sign first recognized by the owner of the dog described in the present report. We hypothesize that clinical signs became evident to the owner after gradual enlargement of the foramen ovale resulted in clinically consequential right-to-left shunting. The owner reported that the dog's water consumption had increased prior to referral. Water consumption was not quantified; however, polycythemia is a potential explanation for polydipsia.[9]

Atrioventricular valve stenosis can result in a diastolic murmur, although available evidence suggests that the diastolic rumble of atrioventricular valve stenosis is seldom heard in affected veterinary patients.[3,10] Thus, the absence of distinct abnormalities during thoracic auscultation of the dog of the present report is not surprising. Additionally, hyperviscosity associated with polycythemia has rheologic effects that limit the development of turbulence, and this may have served to mask a soft murmur. The transient heart sound heard during the initial examination was not characterized phonocardiographically but possibly was an opening snap or split first heart sound, both of which have been identified in human patients with TS.[11]

There is limited information available concerning the treatment and prognosis of dogs with TS. Surgical treatment of congenital malformations of the tricuspid valve has been attempted, but results have been poor.[12] Balloon valvuloplasty offers an alternative means to palliate the clinical signs without having to perform thoracic surgery[3] and has been attempted in dogs as well as in people. Treatment for most forms of cyanotic congenital heart disease in dogs is typically palliative and often limited to periodic phlebotomy and exercise restriction.

Balloon dilation of left and right ventricular outflow tract obstruction has been reported in the veterinary literature.[13-15] However, only a single report[3] of tricuspid balloon valvuloplasty in a dog has been published. Tricuspid balloon valvuloplasty in people has been reported in the medical literature, but most publications[16-21] consist of case reports or small case series, and few describe treatment of congenital tricuspid stenosis. Consequently, little information is available regarding technical aspects of tricuspid balloon valvuloplasty. Initially, a balloon with an OD of 30 mm was chosen on the basis of a suggestion that balloon diameter should approximate that of the tricuspid annulus.[21] It proved impossible to advance a catheter of this size to the tricuspid orifice in the dog described in the present report; however, it is likely that this large-diameter balloon could have been used had a 0.038-in exchange guidewire been available.

In this dog, balloon valvuloplasty resulted in an acute reduction in the right atrial pressure and transvalvular pressure gradient. However, during a reevaluation 30 days after the procedure, the velocities and pressure gradients were higher than those recorded after surgery, although lower than those recorded prior to valvuloplasty. We suspect that the increase in velocity was attributable, at least in part, to the fact that the heart rate during the more recent study was much greater than that recorded during the postoperative evaluation. Increases in heart rate result primarily from a reduction in the diastolic interval, and when atrioventricular valve stenosis is present, ventricular filling is highly dependent on the duration of diastole. With TS, therefore, an increase in heart rate would further limit ventricular filling, resulting in diminished transtricuspid flow and an increase in the transvalvular pressure gradient.[22] In addition, the pressure gradient is a flow-dependent variable and increases in proportion to cardiac output, which accompanies increases in heart rate. It is also possible that the increase in pressure gradient could have resulted from restenosis related to fibrosis. Further, heart rate recorded during echocardiographic evaluations prior to valvuloplasty was greater than that recorded during the evaluation 24 hours after surgery, and this may explain some of the apparent hemodynamic improvement. Regardless, 1 month following the procedure, clinical status had not deteriorated, and contrast echocardiography continued to demonstrate only a small amount of right-to-left atrial-level shunting.

The owner of this dog reported an excellent response following balloon valvuloplasty. There was a marked improvement in exercise tolerance and a

reduction in respiratory rate and effort. Although there was an episode that probably represented exercise-induced collapse about a year following the procedure, the dog was, in general, more active after the intervention. Follow-up Hct measurements suggest that the reduction in resting right atrial pressure observed coincident with balloon valvuloplasty was sustained.

Our results and those of Brown and Thomas[3] suggest that balloon valvuloplasty is an appropriate treatment for severe TS in dogs. In the dog described in the present report, the procedure resulted in palliation of clinical signs, including those associated with central cyanosis and secondary polycythemia resulting from an atrial-level shunt.

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