# Pulmonic stenosis balloon dilation valvuloplasty

**Nicole Van Israël DVM CertSAM CertVC DipECVIM-CA (Cardiology) MSc MRCVS**
ANIMAL CARDIOPULMONARY CONSULTANCY (ACAPULCO), NEARBY SPA, BELGIUM.

UK VET - VOLUME 10 No 2 MARCH 2005

CPD

## PRESENTING HISTORY

A 5-year old male entire Border Collie, rescued at the age of four, presented for the investigation of a murmur associated with collapsing episodes. The syncopal episode always happened on excitement. They became more frequent and lasted longer (average 2-5 min). In between the episodes the dog appeared completely normal.

## CLINICAL EXAMINATION

The dog seemed mildly overweight (26.8 kg; ideal weight 24 kg) but very bright, alert and responsive. His mucous membranes were pink and the capillary refill time was less than 2 seconds. He seemed fully hydrated. Mild distension with pulsation was visible in the lower third of the jugular vein. A precordial thrill was palpable over the left heart base. Abdominal palpation was unremarkable. All lymph nodes were within normal limits. All his extremities were nicely warm. His femoral pulses were slightly weak but without any deficits.

On auscultation a very regular heart rate was audible. The heart rate was 102 BPM and seemed to be sinus in origin. A grade 5/6 systolic murmur was audible over the left heart base. The lungs sounded unremarkable.

## PROBLEM LIST AND DIFFERENTIAL DIAGNOSIS

1. **Syncope:** syncope is a sudden and transient loss of consciousness resulting from cerebral malfunction (anoxia, hypoglycaemia). Syncopal episodes can be cardiogenic, non-cardiogenic (respiratory, metabolic, endocrine, iatrogenic, neurological) or undetermined in origin.

The most common cardiac reasons are:

***arrhythmias***

- bradyarrhythmias: sinus arrest, second and third degree AV-block
- tachyarrhythmias: supraventricular and ventricular tachycardia

***forward failure***

- outflow tract obstruction: aortic stenosis, pulmonic stenosis
- poor diastolic filling: pericardial tamponade, hypertrophic cardiomyopathy
- poor cardiac output: cardiomyopathy, secondary myocardial failure, severe mitral regurgitation
- right to left shunts: PDA, VSD, tetralogy of Fallot

***neurocardiogenic syncope***

2. **Grade 5/6 systolic left heart base murmur**

- aortic stenosis
- pulmonic stenosis

3. **Jugular distension**

This is a sign of right-sided heart failure or increased pleural pressure:

- pericardial tamponade
- tricuspid valve incompetence
- pulmonic stenosis
- dilated cardiomyopathy
- pulmonary hypertension
- right to left shunts

## DIAGNOSTIC WORK-UP

### ECG

A 12-lead ECG showed the presence of a sinus rhythm at 90 BPM. Deep S-waves in lead II, III, and aVF were highly suggestive of right ventricular hypertrophy (Fig. 1).

**Haematology** (Table 1)

Haematology was unremarkable.

**Biochemistry and electrolytes** (Table 2)

The biochemistry showed the presence of a mild prerenal azotaemia. All electrolytes were within normal limits.

**TABLE 1: Haematology**

| Haematology | Patient data | Normal |
|---|---|---|
| Haemoglobin | **18.3 g/dl**↑ | 12.0-18.0 |
| Red Cell Count | 7.230 x $10^{12}$/l | 5.5-8.5 |
| Packed Cell Volume | 50.6% | 39-55 |
| MCV | 70.0 fl | 60-77 |
| MCHC | **36.2 g/dl**↑ | 32-36 |
| White Cell Count | 8.9 x $10^{9}$/l | 6-15 |
| Neutrophils (mature) | 4.09 x $10^{9}$/l | 3.6-12.0 |
| Eosinophils | 0.80 x $10^{9}$/l | 0-1.0 |
| Lymphocytes | 3.382 x $10^{9}$/l | 0.7-4.8 |
| Monocytes | 0.534 x $10^{9}$/l | 0.0-1.5 |
| Platelets | 372 x $10^{9}$/l | 200-500 |

**TABLE 2: Biochemistry and electrolytes**

| Haematology | Patient data | Normal |
|---|---|---|
| Total Protein | 66.7 g/l | 58.0-73.0 |
| Albumin | 33.8 g/l | 26.0-35.0 |
| Globulin | 32.9 g/l | 18.0-37.0 |
| Bile Acids | 5.9 µmol/l | 0.0-7.0 |
| Cholesterol | 4.71 mmol/l | 3.8-7.0 |
| Creatinine | **153 µmol/l**↑ | 0.0-106.0 |
| Glucose | 3.8 mmol/l | 3.0-5.0 |
| Urea | **10.5 mmol/l**↑ | 1.7-7.4 |
| AP | **216 IU/L**↑↑ | 20.0-60.0 |
| ALT | **183 IU/L**↑↑ | 15.0-60.0 |
| Calcium | 2.75 mmol/l | 2.3-3.0 |
| Magnesium | 0.78 mmol/l | 0.69-1.18 |
| Inorganic phosphate | 1.14 mmol/l | 0.9-1.2 |
| T4 | 30.9 nmol/l | 15.0-48.0 |
| | | |
| Sodium | 147.0 mmol/l | 139.0-154.0 |
| Potassium | 5.1 mmol/l | 3.6-5.6 |
| Chloride | 115 mmol/l | 99.0-115 |

### Thoracic radiography

Thoracic radiographs demonstrated right ventricular enlargement (Figs. 2 and 3). The vertebral heart size (VHS) was 10.75 (normal 8-10.5).

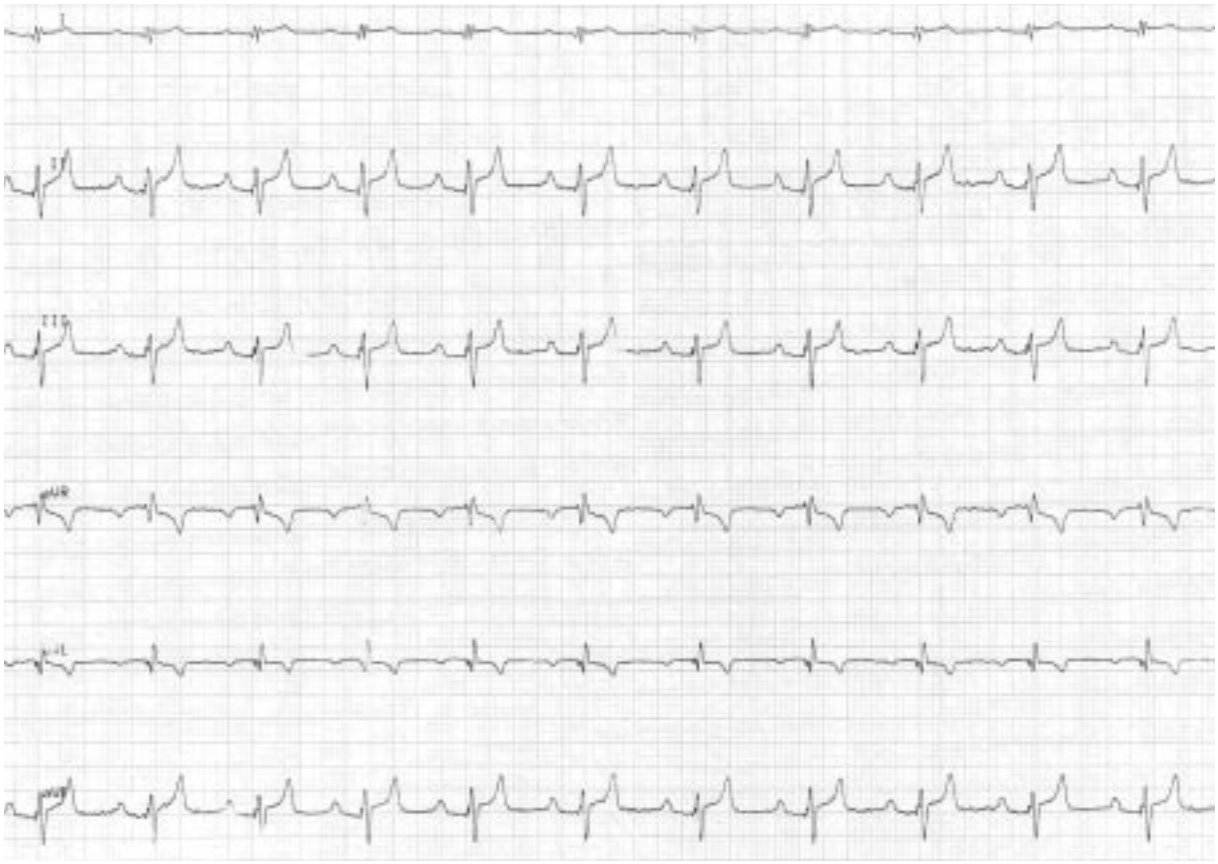

**Fig. 1:** *Electrocardiogram showing marked right axis deviation.*

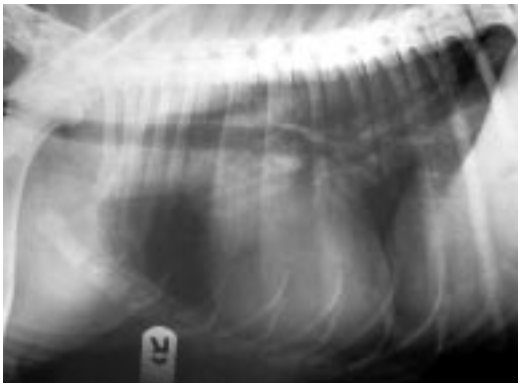

**Fig. 2:** *Lateral thoracic radiograph: rounded cranial border of the cardiac silhouette.*

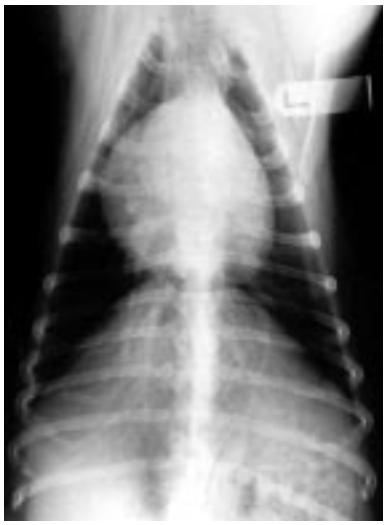

**Fig. 2:** *Dorsoventral thoracic radiograph: prominent right side of the heart.*

**Colour flow doppler echocardiography (CFDE)**

CFDE showed right ventricular wall hypertrophy (Fig. 4). The interventricular septum was flattened (Fig. 5) and the papillary muscles at the right side were thickened. There was sign of right ventricular diastolic dysfunction (E/A wave reversal). Left ventricular contractility was normal with a fractional shortening of 37%, but left ventricular internal diameter was decreased. There was no insufficiency of the mitral or tricuspid valves. The pulmonary artery annulus measured hypoplastic and the

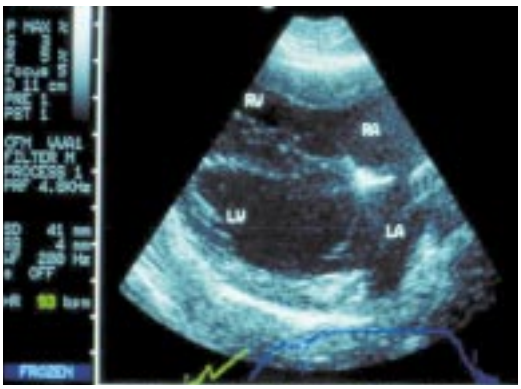


**Fig. 4:** *Right parasternal long-axis view: marked right ventricular hypertrophy.*

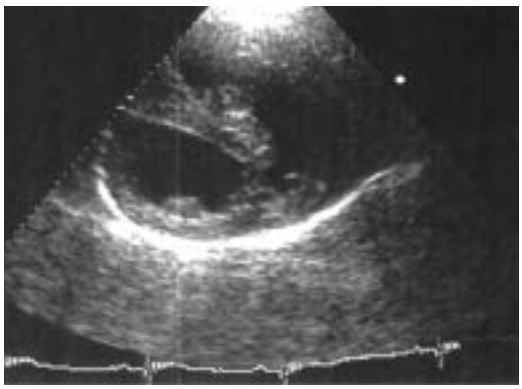

**Fig. 5:** *Right parasternal short-axis view: flattening of the interventricular septum indicating elevated right ventricular systolic pressure.*

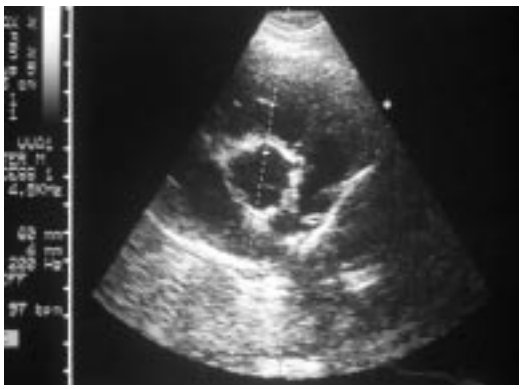

**Fig. 6:** *Right parasternal long-axis view showing thickened dysplastic pulmonic valves.*

pulmonic valves appeared thickened (valvular dysplasia) (Fig. 6) with an increased velocity across the valves (5.1 m/s) that represented a pressure gradient of 104 mm Hg. Pulmonic insufficiency was obvious. The velocity across the aortic valve was within normal limits (0.98 m/s).

## FINAL DIAGNOSIS

All these findings were consistent with severe valvular pulmonic stenosis (PS).

## TREATMENT

Initially atenolol (Tenormin® 25 mg q 12h PO; Zeneca) was prescribed to help decrease right ventricular hypertrophy and improve right sided filling. Unfortunately the collapsing episodes persisted and the dog was scheduled for balloon valvuloplasty of the pulmonic valve.

The dog was premedicated with diazepam (Diazepam® 0.1 mg/kg; Phoenix Pharmaceuticals) and morphine sulphate (0.25 mg/kg, Evans Medical Limited). Also prophylactic antibiotics were administered (Cephazolin, 20 mg/kg IV; Kefzol®, Lilly). General anaesthesia was induced by propofol IV (80 mg, Propofol®, Schering-Plough) and maintained with isoflurane (Isoflo®, Mallinckrodt) and oxygen. Under fluoroscopy the right ventricle was catheterised via the right jugular vein and

after localising the pulmonic valve by selective right ventricular angiography (Conray® 420, Mallinckrodt) a 23-mm balloon catheter was placed across the stenotic area and was inflated (Figs. 7 and 8). The whole procedure was monitored electrocardiographically. The jugular vein was ligated completely and the wound was closed routinely. The dog recovered well from anaesthesia. The ß-blockers were continued at the same dose rate.

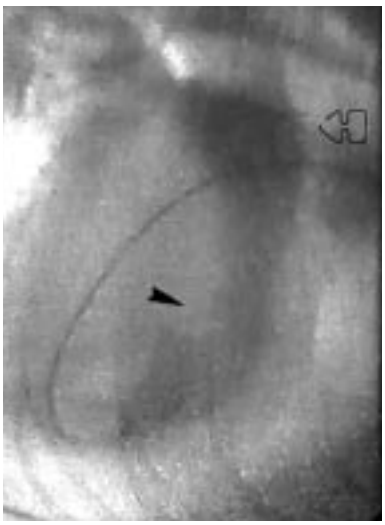

**Fig. 7:** *Fluoroscopic image of the right ventricular angiocardiogram (animal in left lateral recumbency): the contrast material is injected in the right ventricular outflow tract. The pulmonic valve is visualised (closed arrow) and the post stenotic dilatation is obvious (open arrow).*

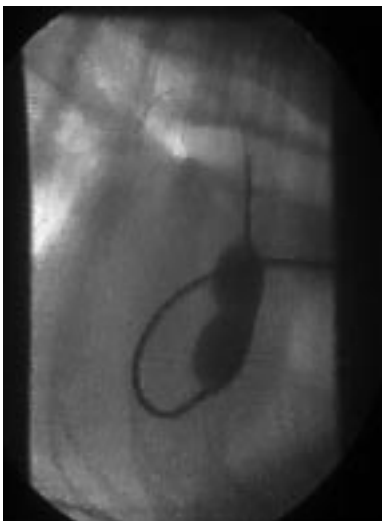

**Fig. 8:** *Fluoroscopic images of the valvuloplasty balloon partially inflated with a mixture of saline and angiographic dye. The balloon is placed across the stenotic pulmonary valve (indentation of the balloon).*

## OUTCOME AND PROGNOSIS

Ultrasonography three days post intervention showed a decrease in the velocity across the pulmonic valve (now 3.1 m/s = 38 mm Hg). This signified a decrease in pressure gradient by 60%. The dog stopped collapsing and regained normal exercise tolerance.

## DISCUSSION

Congenital obstruction of the right ventricular outflow tract is a common lesion in the dog. It can be valvular, subvalvular or supravalvular. Two types of valvular stenosis have been described: type A and B (Fig. 9). Malformation of the valve together with a hypoplastic pulmonary artery, as in this case, is reported to be the most common cause of pulmonic stenosis in dogs. Valvular pulmonic stenosis can also be caused by a single right coronary artery, but since the latter has only been observed in Bull breeds it was ignored in this case. Border Collies are not on the list of the predisposed breeds (Beagle, Bull breeds, Fox Terrier, Keeshond, Miniature Schnauzer).

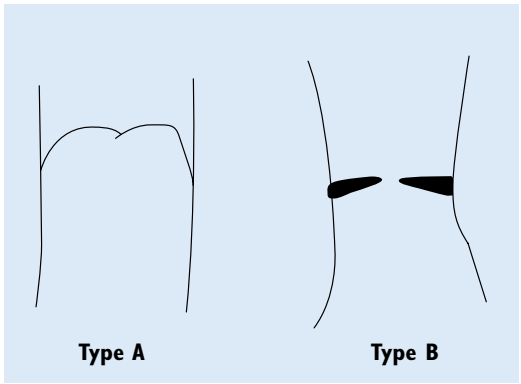


**Fig. 9:** *Types of valvular pulmonic stenosis in the dog. Type A (normal pulmonary artery diameter with parachute-like valve) and B (annular hypoplasia and a dysplastic valve).*

The clinical presentation of collapse here indicated the severity of the lesion. Most dogs with PS exhibit no clinical signs even with severe obstruction. Cardiac arrhythmias have been suggested as a cause of syncope, however this dog was in sinus rhythm at the time of examination. Paroxysmal arrhythmias may have occurred in this animal but it is more likely that collapse has resulted from an inadequate increase in cardiac output due to the outflow obstruction.

On physical examination the localisation and intensity of the murmur also indicated severe PS. The pulse in the jugular vein (canon a-waves) without concurrent jugular distension was a clinical indicator of severe PS without tricuspid regurgitation. The weak femoral pulses were secondary to decreased right ventricular output (and consequently decreased left ventricular output).

The electrocardiographic findings were typical for right ventricular hypertrophy. The presence of a normal size P-wave suggested the absence of right atrial enlargement, later confirmed on radiography and ultrasound.

Radiography was useful for differentiating pulmonic stenosis from aortic stenosis because both lesions will give

a systolic murmur at the heart base level. Despite the presence of obvious right ventricular enlargement no pulmonary artery bulge, a common finding in dogs with PS, was visible. Underperfusion of the lungs, also not seen in this case, is another common finding in animals with PS.

Echocardiography remains the primary method of diagnosing PS. On two-dimensional imaging the typical features were observed: right ventricular and papillary muscle hypertrophy, a flattened septum indicating elevated right ventricular systolic pressure and thickened pulmonary valves (dysplastic) accompanied by post-stenotic dilatation of the main pulmonary artery. Post-stenotic dilatation is not related to the severity of the stenosis but in this case echocardiography appeared to be more sensitive than plain radiography to recognise the dilatation. M-mode echocardiography showed the presence of paradoxical septal motion, an indicator of increased right ventricular diastolic pressure. The decreased left ventricular internal diameter was thought to be secondary to decreased right ventricular cardiac output and consequently decreased left ventricular filling. Once the stenosis had been identified continuous wave Doppler was used to determine the peak velocity across the valve. The modified Bernouilli equation ($\Delta P=4V^2$) was used to transform velocity into pressure gradient. In this case a pressure gradient of 104 mm Hg indicated severe PS. The significance of the pulmonary insufficiency was limited because mild to moderate (velocity less 2 m/s) regurgitation can be observed in completely normal animals. E/A-wave reversal indicated poor right-sided diastolic compliance secondary to the ventricular hypertrophy. Colour flow Doppler confirmed the absence of tricuspid regurgitation.

The first successful PS balloon dilation valvuloplasty was performed by Buchanan in 1980. Dilatation valvuloplasty was regarded as a palliative rather than a curative correction of PS in this case. Selective angiography was not used for diagnostic purpose but to help identifying the stenotic area to facilitate balloon placement.

Long-term follow-up in humans suggests excellent longevity and quality of life and similar short-term and mid-term results have been stated in veterinary medicine. Recently a long-term study in 40 dogs showed that balloon dilation valvuloplasty was successful and resulted in a sustained clinical improvement in 80% of previously symptomatic cases. However, a 7% mortality rate was present.

REFERENCES AND FURTHER READING

BUCHANAN, J.W. (1990) Pulmonic stenosis caused by single coronary artery in dogs: four cases (1965-1984) Journal of the American Medical Association, Vol 196 (1), Jan 1: 115-120.

BUSSADORI, C. M. *et al.* (2001) Balloon valvuloplasty in 30 dogs with pulmonic stenosis: effect of valve morphology and annular size in initial and 1-year outcome. Journal of Veterinary Internal Medicine 15, 553-558.

FINGLAND, B. R. *et al.* (1986) Pulmonic stenosis in the dog: 29 cases (1975-1984). Journal of the American Medical Association, Vol 189, No 2, July 15, 218-226.

KIENLE, R. (1998) Congenital pulmonic stenosis. In: Small Animal Cardiovascular Medicine. Eds Kittleson & Kienle, Mosby, Inc, St-Louis. Pp 248-259.

RISTIC, H. *et al* ( 2001) Congenital pulmonic stenosis: a retrospective study of 24 cases seen between 1990-1999. Journal of Veterinary Cardiology 3, 13-19.

STAFFORD JOHNSON, M. *et al.* (2004) Results of balloon valvuloplasty in 40 dogs with pulmonic stenosis. JSAP, 45,148-153.



## CPD CONTINUING PROFESSIONAL DEVELOPMENT SPONSORED BY PFIZER ANIMAL HEALTH

*These multiple choice questions are based on the above text. Readers are invited to answer the questions as part of the RCVS CPD remote learning program. Answers appear on page 99. In the editorial panel's view, the percentage scored, should reflect the appropriate proportion of the total time spent reading the article, which can then be recorded on the RCVS CPD recording form.*

**1. Which of the following is not on the differential list of syncope in the dog:**

a. Third degree AV-block
b. Pericardial tamponade
c. Neurocardiogenic syncope
d. Persistent foramen ovale
e. Dilated cardiomyopathy

**2. Which of the following is not on the differential list of jugular distension in the dog:**

a. Tricuspid valve incompetence
b. Pericardial tamponade
c. Right to left shunting PDA
d. Mitral valve endocardiosis
e. Dilated cardiomyopathy

**3. Which of the following statements regarding pulmonic stenosis in the dog is incorrect:**

a. Congenital obstruction of the right ventricular outflow tract is an uncommon lesion in the dog.
b. Pulmonic stenosis can be valvular, subvalvular or supravalvular.
c. Valvular pulmonic stenosis can be caused by a single right coronary artery.
d. The electrocardiographic findings often show right axis deviation.
e. Doppler echocardiography remains the primary method of diagnosing pulmonic stenosis.