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Veterinary TECHNICIAN

Case Report

Catastrophic Heart Failure

Kristen Antoon, CVT

At a routine veterinary visit, Jenny—a 20-lb, 3-month-old, female Gordon setter—was found to have a loud heart murmur suspected to be due to a patent ductus arteriosus (PDA). Jenny's breeder was advised to have the PDA surgically corrected, but this was not pursued promptly.

Triage

Seven weeks later, Jenny presented in severe respiratory distress to the emergency room at the Tufts Cummings School of Veterinary Medicine. She had been normal until a few days before, when she had started to occasionally cough.

On triage, Jenny was wagging her tail but was clearly unable to breathe without extreme effort. She was promptly brought to the emergency room and administered supplemental oxygen.

On abbreviated physical examination, Jenny had white mucous membranes, a V/VI continuous heart murmur heard all around the heart, bilateral crackles, evidence of pulmonary edema fluid coming from the mouth and nostrils, poor pulse quality, and a severe increase in respiratory rate and effort.

An intravenous catheter was placed immediately, and 4 mg/kg of furosemide, a potent loop diuretic, was given based on the clinician's evidence of pulmonary edema fluid coming from Jenny's nostrils and mouth and bilateral crackles. Diuretics are useful for treating heart failure because they remove intravascular and interstitial fluid, subsequently reducing intravascular blood volume and preload of the heart. As preload decreases, pulmonary edema is cleared, and heart function improves.

Treatment and Intervention

After 30 minutes of oxygen and furosemide therapy, Jenny's condition had not improved. She received 2 mg/kg of furosemide intravenously. Despite this second dose of furosemide, Jenny was still in severe respiratory distress. Nitroprusside therapy was initiated to further reduce preload and afterload of the heart. Nitroprusside is a balanced vasodilator, which means that it dilates the arteries and veins. In critical care, nitroprusside is always administered by constant-rate infusion (**BOX 1**). The line must never be flushed because flushing may cause severe hypotension due to profound vasodilation. Because of the potent vasodilatory effects of nitroprusside, it should not be administered by bolus. Ideally, blood pressure is monitored directly and continuously during nitroprusside therapy; however, because Jenny's condition was unstable, blood pressure could not be monitored continuously. An indirect blood pressure reading of 110 mm Hg was obtained by Doppler ultrasound at the start of the infusion, but Jenny's unstable condition prevented further blood pressure measurement. Nitroprusside was initiated at a rate of 1 µg/kg/min, which was increased according to Jenny's respiratory rate and pattern as well as clinical signs. While some cardiologists advocate blood pressure monitoring during nitroprusside infusion, others advocate limiting the patient manipulations required to noninvasively determine blood pressure. Because Jenny's respiratory rate and overall appearance were not improving, the administration rate of nitroprusside was increased in 15- to 30-minute periods until a rate of 7 µg/kg/min was reached. Jenny also received another 2 mg/kg of furosemide intramuscularly because the nitroprusside was being administered through the only IV catheter.

Despite oxygen therapy, nitroprusside infusion, and intermittent furosemide injections, Jenny continued to decline clinically.

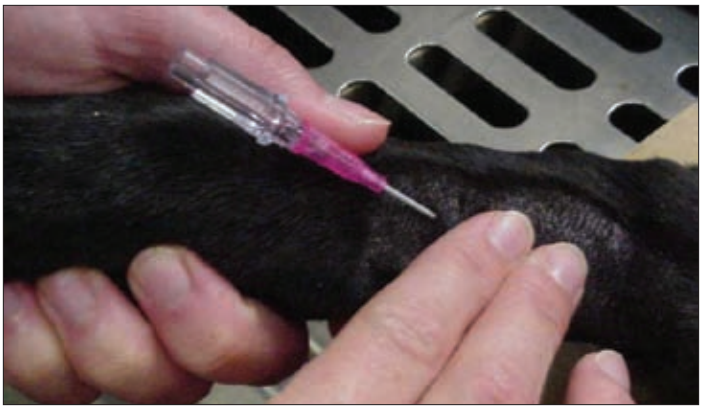

Figure 1. Placement of an arterial catheter. Palpation of the artery is required because it may not be directly visualized. If placement is successful, the blood will be bright red with a pulsatile flow.

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Box 1. Calculating a Constant-Rate Infusion

Need to know

- Body weight (kg)
- Drug dosage
- Drug concentration
- Fluid rate for administering drug
- Fluid volume in bag

Step 1

Determine the number of hours for the infusion. Divide the fluid volume in the bag by the administration rate. For example:

250-mL bag ÷ 10 mL/h = 250 ÷ 10 = 25 h

Some medications, such as nitroprusside, may be increased or decreased, so plan the hourly fluid rate accordingly. One of the major advantages of using constant-rate infusion is the ability to adjust the dose as needed. Pain medications may be increased if a patient is painful or decreased if a patient is excessively sedated but comfortable. When nitroprusside is given for vasodilation to treat volume overload, administration of a small hourly volume of nitroprusside is important. For example, if a patient is receiving nitroprusside at a rate of 1 µg/kg/min (1 mL/h), it is still safe to increase the rate by 500% to 5 µg/kg/min (5 mL/h).

Step 2

Determine how much medication to add to the bag. Multiply the body weight by the dosage, convert the result into the dose for the entire bag, and (if necessary) convert the result into milligrams and milliliters.

Example

- Body weight of dog: 10 kg
- Lidocaine[a] dosage: 50 µg/kg/min
- Lidocaine concentration: 20 mg/mL
- Fluid rate: 25 mL/h
- Fluid volume in bag: 500 mL

Step 1

500 mL ÷ 25 mL/h = 20 h
The fluid bag will last for 20 hours.

Step 2

- 10 kg × 50 µg/kg/min = 500 µg/min
- 500 µg/min × 60 min = 30,000 µg/h
- 30,000 µg/h × 20 h [how long the bag will last] = 600,000 µg/bag
- 600,000 µg ÷ 1000 = 600 mg (1 mg = 1000 µg)
- 600 mg ÷ 20 mg/mL = 30 mL of lidocaine

For drug volumes >10 mL/L, remove this amount of fluid from the bag. In this case, 30 mL of saline would be removed from the 500-mL bag, 30 mL of lidocaine would be added, and the infusion rate would be 25 mL/h. If only 2 mL of drug were being added, no fluid would need to be removed from the bag.

[a]Lidocaine is commonly used. Nitroprusside is used more often in critical care settings.

Because of concern that respiratory arrest was imminent, an arterial catheter was placed (**FIGURE 1**), and an arterial blood gas analysis was performed. The results were consistent with severe respiratory acidosis and compensatory metabolic alkalosis with profound hypoxemia (**TABLE 1**). Because of concern that cardiopulmonary arrest was imminent, Jenny was mechanically ventilated to relieve the work of breathing, provide positive end-expiratory pressure (PEEP; to help clear the pulmonary edema), improve carbon dioxide elimination, and improve oxygenation.

Table 1. Arterial Blood Gas Analysis of the Patient[a]

	Jenny	Normal Range
pH	7.136	7.36–7.44
P_{O_2} (mm Hg)	59.4	90–100
P_{CO_2} (mm Hg)	106.2	36–40
HCO_3^- (mmol/L)	36.2	20–24

[a]Interpretation is consistent with severe respiratory acidosis, with metabolic compensation and profound hypoxemia.

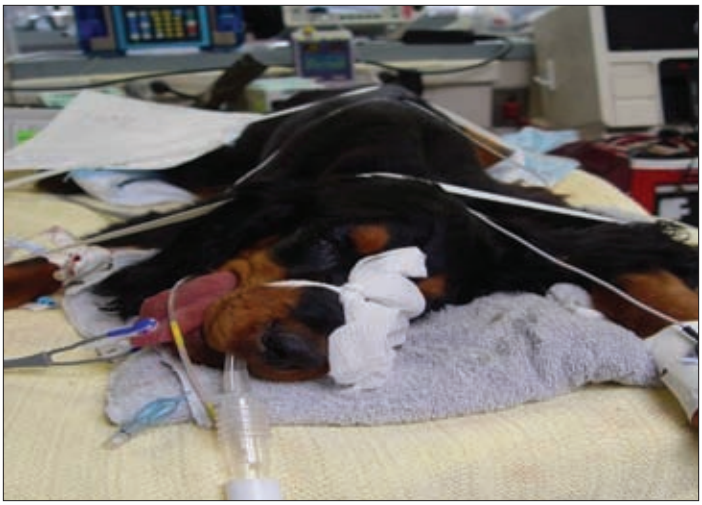

Figure 2. Jenny on the mechanical ventilator.

A Puritan Bennett 840 Ventilator System (Covidien, Boulder, CO), which is a human critical care ventilator, was set up in the intensive care unit (ICU). A table was set up with blankets, heat support, and endotracheal tubes for emergency intubation. Nitroprusside therapy was temporally discontinued. Jenny was brought to the ICU, where anesthesia was induced with diazepam (2.5 mg IV). She was intubated with a size 7 endotracheal tube, and the cuff was inflated. After intubation, pulmonary edema fluid poured from the endotracheal tube. The suction unit was readily available to clear the endotracheal tube using aseptic technique. Jenny was placed on the ventilator (**FIGURE 2**) with the following settings:

- Pressure control, with an inspired oxygen concentration of 100%
- Peak inspiratory pressure: 25 cm H_2O
- PEEP: 5 cm H_2O
- Respiratory rate: 25 breaths/min
- Inspiratory to expiratory proportion: 1:2

Another IV catheter was placed to initiate constant-rate infusions of fentanyl (10 µg/kg/h) and diazepam (10 µg/kg/h) for anesthesia

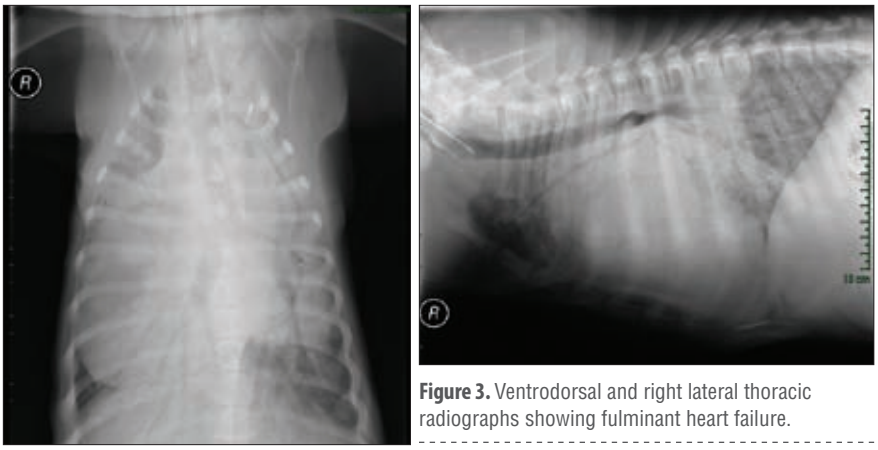

Figure 3. Ventrodorsal and right lateral thoracic radiographs showing fulminant heart failure.

to minimize the work of breathing and prevent patient–ventilator asynchrony ("fighting" or "bucking" the ventilator). Jenny was monitored by continuous electrocardiography, pulse oximetry, noninvasive blood pressure methods, and measurement of end-tidal carbon dioxide (CO_2). After several minutes of positive-pressure ventilation, the end-tidal CO_2 level was decreasing and oxygen saturation was increasing. Heart rate and rhythm remained stable, and the systolic blood pressure remained stable at 110 mm Hg. An hour after being placed on the ventilator, Jenny had improved significantly, so the ventilator settings were decreased to the following:

- Peak inspiratory pressure: 23 cm H_2O
- PEEP: 2.5 cm H_2O
- Fraction of inspired oxygen (FIO_2): 0.6 (60%)

Jenny's oxygenation level was 99%. Her end-tidal CO_2 level had greatly improved to 31 mm Hg (normal: 35 to 45 mm Hg) from a life-threatening level of 106 mm Hg. Chest radiographs (**FIGURE 3**) documented severe cardiomegaly with fulminant pulmonary edema.

Surgical Intervention

Bedside echocardiography confirmed the presence of marked left atrial enlargement and congestive heart failure due to volume overload resulting from a left-to-right shunting PDA. To survive, Jenny needed to have her PDA corrected. The fluoroscopy suite was set up, and Jenny was taken off the ventilator and manually ventilated during the procedure. Her vital signs were monitored closely during the transition from the ICU to the fluoroscopy suite.

Jenny was placed in left lateral recumbency, and her left inguinal region was clipped and prepared for catheter placement. A surgical cut-down was performed to identify the femoral artery. A cardiac catheter was passed up the femoral artery and through the aorta, and angiography was performed to visualize the PDA (**FIGURE 4**). Then a 10-mm Amplatzer Duct Occluder (AGA Medical Corporation, Plymouth, MN; **FIGURE 5**) was placed to occlude the abnormal blood flow.

Postoperative Care

After the successful procedure to correct the PDA, Jenny was weaned from the mechanical ventilator. An 8-Fr red rubber catheter was placed into her right nasal cavity to provide supplemental oxygen at a rate of 2 L/min (**FIGURE 6**). Jenny was successfully extubated and recovered in the ICU. She was monitored for arrhythmias and adequate respiratory function. After surgery, Jenny developed a fever (104°F [40°C]) and tachypnea (60 to 80 breaths/min). An antibiotic (imipenem [50 mg IV q8h]) was initiated, and blood cultures were submitted. Sepsis was suspected to have developed from ventilator-associated pneumonia or the cardiac procedure. Jenny was given a 250-mL bolus of lactated Ringer solution over a 30-minute period, and her IV fluid was supplemented with 20 mEq/L of potassium chloride because of anorexia. Throughout the day, Jenny developed various arrhythmias, including supraventricular tachycardia, accelerated idioventricular rhythm, and ventricular tachycardia. Pimobendan (5 mg PO q12h) and extended-release diltiazem (60 mg PO q12h) were initiated. Jenny was monitored for adverse reactions

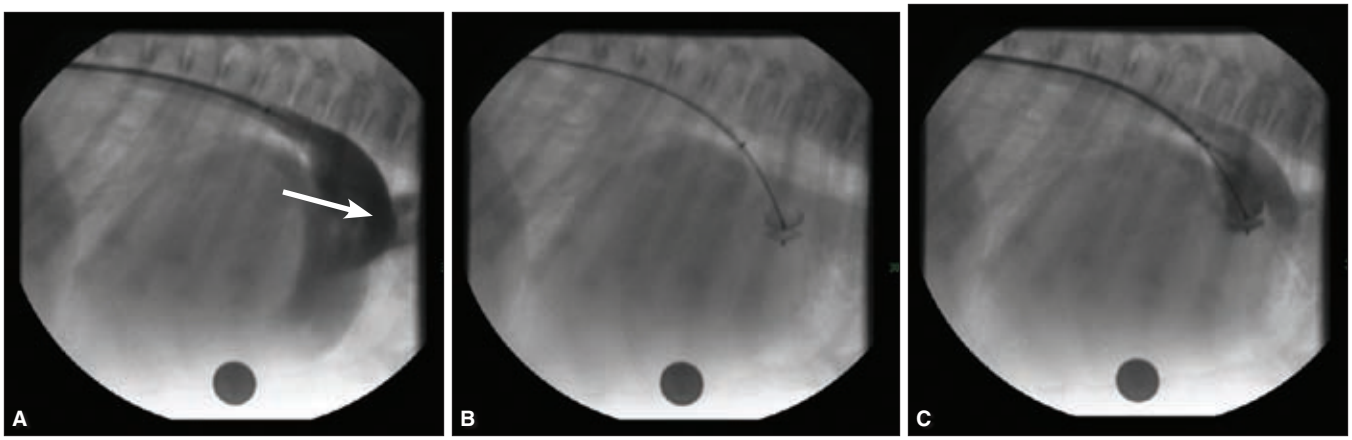

Figure 4. Angiograms of a PDA. (A US dime [the round density] is used for measurement purposes.) (**A**) The *white arrow* points to the PDA. Iodinated contrast material has been injected from the aorta, highlighting the path of the PDA. (**B**) Placement of the Amplatzer Duct Occluder. (**C**) Occlusion of blood flow at the PDA.

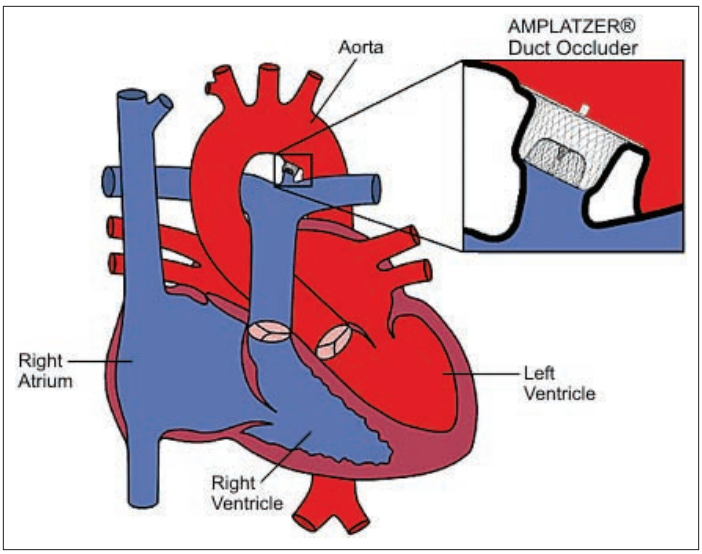

Figure 5. Illustration showing the position of an occluding device in a PDA. *Courtesy of AGA Medical Corporation, 2011. US Products: How Your Doctor Will Implant the Duct Occluder. Accessed April 2011 at http://www.amplatzer.com/products/pda_devices/implanting_duct_occluder/tabid/196/default.aspx; with permission.*

to diltiazem, including hypotension, bradycardia, and atrioventricular block.

Diltiazem is a calcium-channel blocker that is primarily used to control supraventricular arrhythmias, systemic hypertension, and hypertrophic cardiomyopathy. Diltiazem can also be used to control atrial flutter and other forms of tachycardia. The drug affects the heart tissues (the sinoatrial and atrioventricular nodes) rather than the blood vessels.

Pimobendan helps reduce the workload of the heart and make the heart pump more efficiently. The drug has been shown to increase survival and improve quality of life in patients with congestive heart failure secondary to mitral valve disease.

On the second day after surgery, the oxygen level was reduced to 1 L/min, and Jenny's arrhythmias were decreasing in severity, with mostly normal sinus rhythm. Laboratory testing revealed an onset of hypokalemia (2.85 mmol/L [normal: 3.8 to 4.5 mmol/L]) which was thought to be due to massive preprocedural diuresis. Potassium supplementation was administered at a rate of 0.4 mEq/kg/h. Jenny was recovering slowly and showed evidence of hypoxic brain injury. She had developed bilateral cortical blindness, ataxia, and an obtunded mental status. Her cardiovascular status was stable, and her respiration was normal. Over the next 24 hours, her mental status improved rapidly. She began to eat and drink. By 40 hours after surgery, she was a normal puppy and her vision had returned.

Follow-up

Three days after surgery, another echocardiogram was obtained. On auscultation, a II/VI left apical heart murmur was heard. The echocardiogram revealed no blood flow through the PDA. A fair amount of mitral regurgitation was present, and a mass-like structure, most likely a thrombus, was attached to the left atrium

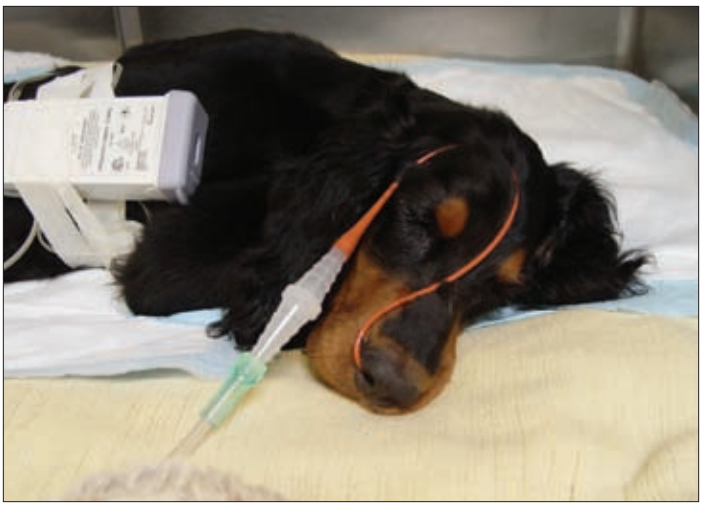

Figure 6. Jenny after surgery, with a unilateral nasal line for oxygen therapy.

wall. The left ventricle showed asymmetric function and a decrease in contractility. One of the papillary muscles and the left ventricular free wall were not moving as well as the rest of the left ventricle. Jenny's arrhythmias and wall motion abnormities were related to a myocardial infarction. It was recommended to initiate enalapril (5 mg PO q24h) and clopidogrel (75 mg PO q24h). Enalapril, an angiotensin-converting enzyme inhibitor, helps lower blood pressure and decrease the workload of the heart in patients with heart failure. Clopidogrel is an antiplatelet agent for preventing thrombi.

Jenny's breathing was improving, so nasal oxygen was discontinued. On the fourth day after surgery, Jenny was discharged from the hospital with various cardiac medications, including pimobendan, diltiazem, enalapril, and clopidogrel. Blood cultures were negative, and the fever had resolved within 24 hours after surgery. Antibiotics were continued for 5 days after Jenny was discharged.

Outcome

One month after Jenny's life-threatening event, Jenny had gained weight, her vision was still normal, and she was acting like a healthy puppy. An echocardiogram showed much-improved ventricle contractility and no thrombus. Jenny continues to do well.

Discussion

The ductus arteriosus is a normal anatomic structure that permits blood to bypass the lungs in utero; however, the ductus arteriosus should close shortly

Glossary

Arterial blood gas analysis — analysis of a blood sample collected directly from an artery via puncture or an indwelling arterial catheter

Positive end-expiratory pressure (PEEP) — a method of ventilation in which airway pressure is maintained above atmospheric pressure at the end of exhalation; PEEP optimizes the time for gas exchange throughout the respiratory cycle

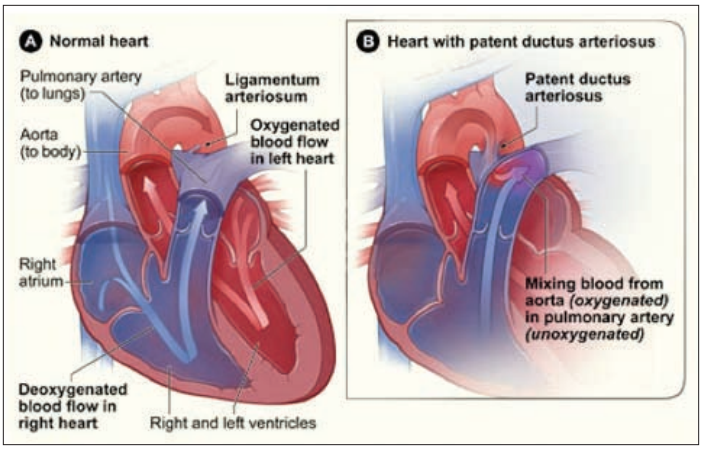

Figure 7. Normal heart structure compared with a heart with a PDA. *National Heart, Lung, and Blood Institute. Heart and Vascular Diseases: What Is Patent Ductus Arteriosus? Accessed April 2011 at www.nhlbi.nih.gov/health/dci/Diseases/pda/pda_what.html.*

after birth (**FIGURE 7**). When it does not close promptly after a neonate begins to breathe air, the neonate is considered to have a PDA, which often results in heart failure due to left-to-right shunting. This means that some of the blood that should go into arterial circulation is recirculated to the lungs via the PDA, resulting in a volume-overloaded heart, fluid retention, and subsequent heart failure. If left untreated, most dogs with a PDA die of progressive heart failure within 1 year. Treatment requires surgery: thoracotomy and ligation of the patent vessel or, more recently, an interventional procedure such as placement of an embolization coil(s) or an Amplatzer Duct Occluder, both of which mechanically occlude blood flow through the PDA and promote formation of an intravascular clot.

Almost all PDAs shunt left to right, meaning that the blood flows from the aorta through the pulmonary artery and then recirculates through the lungs. The blood flows left to right because aortic pressure is higher than pulmonary arterial pressure. Right-to-left shunting PDAs are rare and involve pulmonary hypertension, in which the pulmonary arterial pressure is higher than the aortic pressure. In both cases, the blood moves down the pressure gradient. Dogs with a right-to-left shunting PDA tend to present later in life and with syncope or seizures due to hyperviscosity resulting from hypoxemia, which triggers release of erythropoietin and subsequent development of a high hematocrit (typically 70% to 75%). Because of the associated altered viscosity of the blood, no murmur is heard.

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