

# Aneurysmal dilatation of the right auricle in two dogs

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- ▶ Right auricular aneurysmal dilatation should be included in differential diagnoses of masses detected radiographically in the cranial mediastinum.
- ▶ Herniation of the right auricle through a pericardial defect is a possible cause for such lesions in dogs.
- ▶ An absence of abnormal echocardiographic findings does not rule out the presence of a right auricular aneurysm.
- ▶ Angiographic evaluation may be indicated to confirm or rule out right auricular aneurysmal dilatation.

An 8-year-old male sexually intact Golden Retriever was evaluated at the Veterinary School of the University of Glasgow because of a history of collapse during exercise. Two months previously, the dog had fallen from a high wall onto concrete. On clinical examination, the dog was alert and in good body condition. The heart rate was 180 beats/min and the rhythm was irregularly irregular. The respiratory rate was 15 breaths/min, and there was no dyspnea at rest. The femoral pulses were strong, and there was a marked pulse deficit; mucous membranes were pink with prompt capillary refill. On thoracic auscultation, no abnormalities were detected. Thoracic radiography revealed an enlarged cardiac silhouette with a prominent bulge in the area of the right auricle (Figure 1). Echocardiography revealed atrial fibrillation, but cardiac function was within normal limits, and no valve regurgitation or stenosis was detected; there was marked right atrial dilatation that could not be delineated entirely because of lung interference. The dog was treated with digoxin<sup>a</sup> (0.25 mg, PO, q 12 h) to reduce the ventricular rate in response to atrial fibrillation and aspirin<sup>b</sup> (450 mg, PO, q 24 h) to reduce the risk of thrombus formation in the right atrium, although the efficacy of the latter treatment is unproven. To confirm the presence and extent of the bulge, nonselective venography via the right jugular vein and a contrast-medium-enhanced axial com-

puted tomographic scan were performed under anesthesia (Figure 2). Both evaluations revealed that the right auricle was markedly enlarged and had thin walls. To obtain a definitive diagnosis and pursue possible treatment options, an exploratory thoracotomy was performed. During this procedure, a defect in the pericardial sac (through which the right auricle and part of the right atrium had herniated) was identified. The epicardial surface of the herniated portion was roughened and granular. A subphrenic pericardectomy was performed. The dog recovered from anesthesia without complications and was discharged from the hospital 5 days later. The owners were instructed to continue the administration of digoxin and aspirin at the aforementioned dose. During a follow-

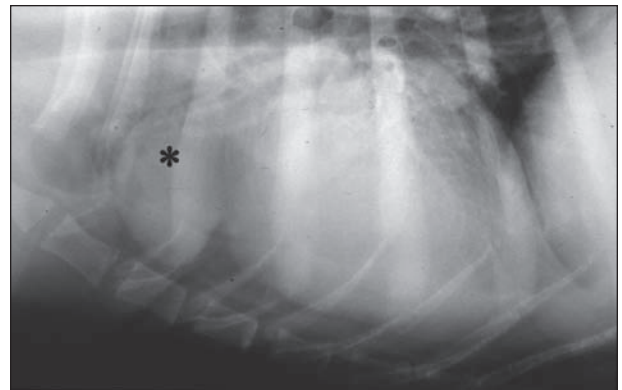


Figure 1—Lateral radiographic view of a portion of the thorax of an 8-year-old Golden Retriever with a history of collapse during exercise. Notice the ovoid soft tissue opacity (\*) that obliterates the cranial aspect of the cardiac silhouette, which represents the right auricle and parts of the atrium that had herniated through a defect in the pericardial sac.

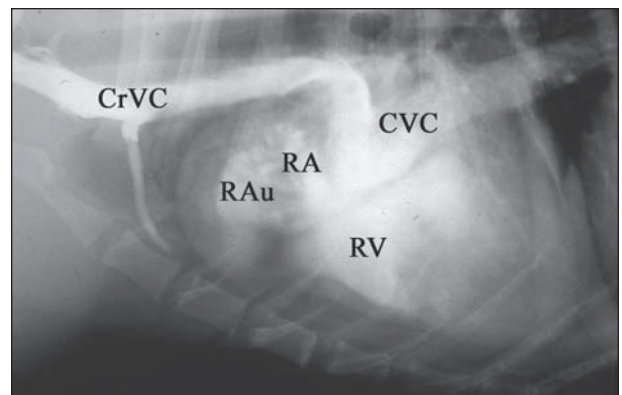


Figure 2—Lateral jugular venographic view of the same dog as in Figure 1. Radiopaque contrast medium can be seen in the cranial and caudal vena cavae and tributary veins, right atrium, and right ventricle and within the herniated right auricle. CrVC = Cranial vena cava. CVC = Caudal vena cava. RA = Right atrium. RAu = Right auricle. RV = Right ventricle.

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up examination 5 weeks after surgery, the dog had no clinical signs, although atrial fibrillation (heart rate of 80 to 100 beats/min at rest) was detected. Results of a digoxin assay indicated that the serum concentration (1.5 ng/mL at 7 hours after dosing) was within the therapeutic range (1 to 2.4 ng/mL). Thoracic radiography revealed that the bulge on the cranial cardiac border was less prominent than it had appeared previously. Conversion of atrial fibrillation to sinus rhythm with additional administration of propafenone<sup>c</sup> (225 mg, PO, q 8 h) was attempted over 2 weeks but failed and was discontinued after a follow-up examination 7 weeks after surgery revealed persistent atrial fibrillation.

Seventeen months after the initial evaluation, the dog was examined because of lethargy, weight loss, and vomiting. Findings on clinical examination included tachycardia and mild dehydration. Serum biochemical analyses revealed mild azotemia and hypoalbuminemia; results of a digoxin assay indicated that the serum digoxin concentration (3 ng/mL) was above the therapeutic range. Urinalysis revealed an abnormally high protein-to-creatinine ratio that was consistent with protein-losing nephropathy. Echocardiographically, cardiac function was within normal limits and there was an absence of valve regurgitation or stenosis. At this time, the provisional diagnosis was digoxin toxicosis, possibly secondary to impaired renal function. The dog was treated with IV fluid therapy, ramipril<sup>d</sup> (5 mg, PO, q 24 h), and reduced dosages of digoxin<sup>e</sup> (0.125 mg, PO, q 12 h) and aspirin<sup>b</sup> (75 mg, PO, q 24 h). The owners were given advice regarding feeding the dog a diet formulated to provide renal support but did not comply. For approximately 2 months, the dog was reasonably well despite increasing azotemia and persistent signs of protein-losing nephropathy; however, the dog had a poor appetite and developed signs consistent with gastrointestinal ulceration, which prompted the owners to have the dog euthanatized by their local veterinarian. No necropsy was performed.

A 12.5-year-old spayed female Lhasa Apso was referred to the University of Pennsylvania School of Veterinary Medicine for evaluation of a heart murmur and chronic cough. On clinical examination, the dog was bright, alert, and responsive. There was no history of trauma. The dog had a heart rate of 100 beats/min, normal femoral pulses and capillary refill time, and pink mucous membranes. On thoracic auscultation, a grade 3/6 pansystolic murmur with the point of maximal intensity over the right heart apex and a grade 2/6 holosystolic murmur with the point of maximal intensity over the left heart apex were detected. The dog's respiratory rate and effort were normal, and no adventitious respiratory sounds were auscultated. A cough could be elicited on tracheal palpation. Examination of an ECG tracing revealed sinus arrhythmia but no other abnormalities. Echocardiographically, systolic function and left heart wall measurements were within reference limits; there was mild thickening and regurgitation of the aortic, tricuspid, and mitral valves with prolapse of the anterior mitral valve leaflet. Compensated chronic valvular disease and mild tracheal collapse were diagnosed. No treatment was prescribed at this time, but the dog was scheduled for a follow-up examination 12 months later to monitor the progression of disease. During that examination of the now 13.5-year-

old dog, the cardiac disease was found to be unchanged and thoracic radiographs were obtained. A mass was seen in the cranial aspect of the thorax, obliterating the cranial cardiac silhouette. A repeat echocardiographic examination was performed that included imaging of the right heart during peripheral venous injection of 10 mL of physiologic saline (0.9% NaCl) solution<sup>f</sup> in which small air bubbles had been introduced via forceful fluid movement between 2 connected syringes (ie, agitated saline solution); the agitated saline solution was used as an intravascular contrast medium, but the procedure did not identify a right atrial dilatation or a mass in the cranial portion of the thorax. To confirm the presence and extent of the mass, nonselective jugular venography and contrast-medium-enhanced helical computed tomography were performed under general anesthesia (Figures 3 and 4). Both evaluations revealed a markedly dilated, thin-

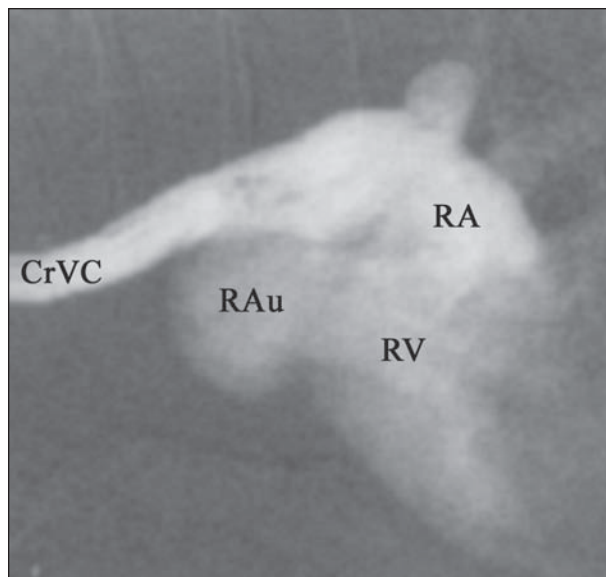


Figure 3—Digital subtraction fluoroscopic jugular venographic view of a 13.5-year-old Lhasa Apso with a heart murmur and chronic cough. From the CrVC, contrast medium has entered the RA, RV, and aneurysmal RAu, which is seen bulging cranially. See Figure 2 for key.

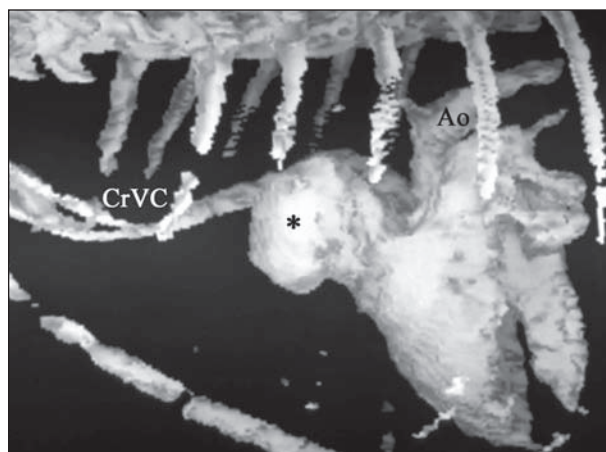


Figure 4—Three-dimensional reconstruction of a computed tomographic angiogram of the dog in Figure 3. Notice the contrast-enhanced lumina of the CrVC, heart, and aortic arch as well as parts of the axial skeleton. A large aneurysm of the RAu (\*) is visible. Ao = Aortic arch. See Figure 2 for remainder of key.

walled right auricle. The right auricular dilatation was considered an incidental finding, and because the cardiac disease had not progressed, no treatment was prescribed for either condition. At the age of 15.5 years, the dog had multiple seizures and died. No necropsy was performed.

In dogs, the most common abnormality of the pericardial sac is a peritoneopericardial diaphragmatic hernia with or without herniation of abdominal organs.<sup>1-5</sup> Complete absence of the pericardial sac is a very rare congenital abnormality in dogs.<sup>5-7</sup> The most extensive report<sup>8</sup> on partial defects of the parietal pericardium in dogs describes a series of 8 dogs with defects of various sizes and locations with or without cardiac herniation, which were incidental findings during necropsy procedures. The etiology of these defects is unknown, but those located ventral to the phrenic nerve are unlikely to be congenital.<sup>8</sup> Reports<sup>9-22</sup> of individual dogs with cardiac herniation through a defect or rupture of the pericardial sac with associated clinical signs have been published in German and earlier English scientific literature. In 13 of these reports that contained some clinical information, dogs had either sudden development of marked clinical abnormalities—such as dyspnea, collapse, and death<sup>9-13,15-18,22</sup> similar to findings in the first case described in the present report—or chronic deterioration of the function of the left side of the heart.<sup>19-21</sup> On the basis of those reports, clinical signs appear to be closely related to the location and degree of cardiac incarceration. A confirmed history of trauma was reported in 5 of the 14 single-case reports.<sup>9,13,15,19,22</sup> Analysis of signalment data in all published reports revealed that 14 dogs with pericardial defects or tears were male and 5 were female (the sex of 3 dogs was not reported); affected dogs had a wide age range (2 to 15.5 years) and were of various breeds.<sup>8-22</sup> In humans, congenital defects of the pericardium are approximately 3 times as common in men as in women,<sup>23</sup> and a similar sex distribution could be present in dogs. Alternatively, male dogs may be more susceptible to pericardial trauma. Among the 22 reported dogs with pericardial defects, 18 had cardiac herniation through the defect<sup>8-22</sup> and 4 did not.<sup>8</sup> The most commonly herniated structures were the cardiac apex with incarceration of parts of the left ventricle (9 dogs),<sup>8,11-14,16,18,19</sup> followed by the right auricle and parts of the right atrium (4 dogs),<sup>9,17,20,22</sup> the right ventricle with or without parts of the right atrium (4 dogs),<sup>5,8,10,21</sup> and the left auricle (1 dog).<sup>8</sup> In 5 dogs with incarceration of a part of the right side of the heart and reported radiographic signs, radiographic findings were similar to those noted in both dogs of this report.<sup>9,17,20-22</sup> Because a pericardial defect was confirmed in only 1 of the 2 dogs of this report, we chose the term aneurysm (which is defined as a localized vascular dilatation<sup>24</sup>) to describe the lesions in both dogs. Echocardiographic findings of cardiac herniation through pericardial defects have only been reported in 2 dogs.<sup>17,19</sup> In 1 of those dogs, left ventricular dilatation and poor ventricular myocardial contractility were thought to be related to cardiomyopathy, but necropsy revealed that the cardiac apex was incarcerated in a pericardial tear at the level of the circumflex coronary artery.<sup>19</sup> In the other dog, a calcified thrombus attached to the wall of

the herniated right auricle was diagnosed and confirmed during surgery.<sup>17</sup> The right paracostal short-axis and left parasternal long-axis echocardiographic views that were used in both dogs of this report are standardized views used to assess, among other structures, the right atrium and auricle. We attribute the failure to identify the dilated right auricle to the fact that it protruded far into the cranial mediastinum, where it was neighbored by well-aerated lung lobes. The difficulties in identifying the abnormal right auricle echocardiographically in both dogs of this report emphasize that a diagnosis of an aneurysmal right auricle should not be ruled out in dogs on the basis of echocardiographic findings alone. Angiography would be warranted in such patients. In the dogs of this report, nonselective angiographic (radiographic or fluoroscopic) or computed tomographic angiographic techniques successfully revealed the communication of the lesion with the right atrium. Among the 13 dogs with cardiac herniation for which some clinical information has been reported, treatment for the herniation was instigated in only 3.<sup>9,17,22</sup> A traumatic pericardial tear was surgically closed in 2 dogs<sup>9,22</sup>; 1 dog with ventricular tachyarrhythmia converted to a sinus rhythm immediately after closure of the pericardial suture, which suggests that the pericardial defect and herniation caused or contributed to the arrhythmia.<sup>9</sup> In another dog with atrial fibrillation and a right auricular thrombus, surgical amputation was performed to prevent pulmonary thromboembolism.<sup>17</sup> In the first dog of this report, it was assumed that the epicardial irritation caused by constriction of the pericardial defect around the margin of the herniated and dilated right auricle predisposed the heart to atrial fibrillation. Although a pericardectomy was performed to relieve the constriction, the dysrhythmia persisted possibly as a result of fibrosis, electrophysiologic remodeling of the atrial wall, and persistent right auricular dilatation.

As illustrated by the dogs of this report, cardiac herniation through a pericardial defect should be included in the differential diagnoses for dogs in which soft tissue opacities obliterating the cranial cardiac silhouette are detected radiographically. Nonselective angiography is most useful to confirm the diagnosis. In dogs with related clinical signs, a pericardectomy should be considered if the pericardial defect cannot be closed.

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- a. Lanoxin, GlaxoSmithKline, Uxbridge, UK.
  - b. Aspirin, Ashbourne Pharmaceuticals Ltd, Northampton, UK.
  - c. Arhythmol, Abbott Laboratories Ltd, Maidenhead, UK.
  - d. Vasotop, InterVetUK Ltd, Milton Keynes, UK.
  - e. 0.9% Sodium chloride injection, USP, Abbott Laboratories, Chicago, Ill.
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