

Cardiac Magnetic Resonance in the Differentiation of Neoplastic and Nonneoplastic Pericardial Effusion

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Background: Cardiac magnetic resonance (CMR) is the imaging modality of choice for cardiac tumors in people. Although neoplastic pericardial effusion (PE) carries a poor prognosis, benign idiopathic pericardial effusion does not. Definitive diagnosis is critical for surgical intervention, but currently available diagnostic techniques such as echocardiography and pericardial fluid cytology often are inconclusive.

Hypothesis/Objective: Describe CMR findings associated with PE and determine whether CMR aids in differentiation of benign and neoplastic causes of PE.

Animals: Eight client-owned dogs with PE diagnosed by transthoracic echocardiography (TTE).

Methods: CMR was performed with a 1.5 T, including dark blood, steady-state free precession cine, pre- and postcontrast T1-weighted imaging, and delayed inversion recovery prepped imaging.

Results: CMR confirmed a cardiac mass and supported suspected tumor type in 4 dogs with suspected hemangiosarcoma. In 1 equivocal TTE case, CMR did not demonstrate a mass, but neoplasia was later diagnosed. In another equivocal case, CMR did not demonstrate a mass but showed findings consistent with a pericardiocentesis complication. In 1 dog without evidence of cardiac neoplasia, abdominal magnetic resonance imaging identified presumptive hepatic and splenic metastases. On reevaluation of the original CMR study, the 2 equivocal cases that were interpreted as tumor negative were reassessed as tumor positive.

Conclusions and Clinical Importance: CMR did not substantially improve diagnosis of cardiac tumors compared with TTE in these 8 cases, but it yielded useful descriptive information regarding extent, anatomic location, and potential tumor type and confirmed that CMR requires extensive additional training for tumor identification.

Key words: Anatomy; Cardiology; Cardiovascular; Diagnosis; Echocardiography oncology; Pathology.

Pericardial effusion (PE) is the accumulation of an excessive amount of fluid in the pericardial sac that may result in the clinical sign of cardiac tamponade. Patients with substantial PE with or without cardiac tamponade typically present with collapse, hypotension, muffled heart sounds, jugular distension or pulsation, and poor pulse quality. If fluid has accumulated gradually, the pericardium stretches and signs of right-sided congestive heart failure predominate.

The causes of PE include a range of neoplastic and nonneoplastic, eg, (inflammatory) causes. The most common neoplastic cause of PE is hemangiosarcoma (HSA), followed by chemodectoma (paraganglioma) and mesothelioma. Other neoplasms that have been reported in affected dogs include carcinomatosis, lymphosarcoma, ectopic thyroid carcinoma, myxoma, myxosarcoma, fibroma, fibrosarcoma, chondrosarcoma, osteosarcoma, rhabdomyosarcoma, and lipoma.^{1–39} The majority of these tumors have been associated with PE. PE also can

Abbreviations:

CMR	cardiac magnetic resonance
cTnI	cardiac troponin I levels
MRI	magnetic resonance imaging
PE	pericardial effusion
SSFP	steady-state free precession
TEE	transesophageal echocardiography
TTE	transthoracic echocardiography

occur with nonneoplastic disease processes. Nonneoplastic effusions can occur secondary to left atrial tears, benign idiopathic pericardial effusion (BIPE), intrapericardial cysts, chylopericardium, traumatic pericarditis, and various infectious causes that are rare.^{39–50} It can be difficult to diagnose the cause of PE unless a tumor is readily visible on 2D or 3D echocardiography, particularly if the echocardiogram is performed after pericardiocentesis.

Many tests have been evaluated as tools to aid in differentiating among the various causes of PE. The presence of PE results in shedding of reactive mesothelial cells which can be mistakenly identified as neoplastic cells. Therefore, cytologic evaluation of PE does not readily distinguish between neoplastic and nonneoplastic causes of PE unless the cause is infectious.⁵¹ In a study by Sisson et al⁵¹, 74% of neoplastic effusions were not detectable as neoplastic and 13% of nonneoplastic effusions were misdiagnosed as neoplastic. In people, the pH of the effusion aids in differentiation between neoplastic and nonneoplastic causes. Although results from 1 study suggested a similar correlation in dogs, this result has not been validated in later veterinary studies and there appears to be much overlap.^{52,53} Bauer and Moritz⁵⁴

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evaluated effusions (pericardial, pleural, and abdominal) with an automated hematology analyzer and found that carcinoma and mesothelial cells were classified as mononuclear blasts. Studies that evaluated cardiac troponin I (cTnI) and cardiac troponin T concentrations found that although there was some overlap, cTnI concentrations may be beneficial in differentiating benign from neoplastic effusions.^{55,56} One study demonstrated significantly higher lactate, hematocrit, and urea nitrogen concentrations in neoplastic PE, but the amount of overlap limited clinical utility.⁵⁷ Stepien et al⁵⁸ found that postbiopsy survival time and recurrence of pleural effusion was the best way to obtain an antemortem diagnosis of mesothelioma versus BIPE, noting that the cytologic analysis of biopsy samples often was inaccurate. Although these approaches to differentiating benign and neoplastic effusions are helpful, the diagnosis often remains equivocal and it is difficult for owners to make surgical decisions when the long-term prognosis is unclear.

Imaging of cardiac tumors has been greatly enhanced by improvements in transthoracic echocardiography (TTE), but imaging the heart base remains technically challenging. TTE is limited by other factors such as operator experience, restricted field of view, and animal body condition as well as demeanor. Transesophageal echocardiography (TEE) overcomes the limited acoustic window of TTE, but the equipment requires advanced training and can be cost prohibitive. Magnetic resonance imaging (MRI) has become available at most specialty veterinary hospitals and offers advanced imaging for many clinical problems involving multiple veterinary specialties. In human medicine, cardiac magnetic resonance (CMR) currently is the modality of choice to evaluate cardiac tumors.^{59–67} All echocardiographic modalities have been shown to be limited in their ability to provide tissue characterization.^{64–67} CMR offers multiplanar imaging without limitations on the available field of view. Additional advantages of CMR over TTE or TEE are improved resolution and soft tissue contrast, greater ability to characterize tissue and ability to demonstrate involvement of the mediastinum and lungs.^{60,64–67} The major contraindication to the use of CMR in dogs is that it requires anesthesia, advanced technical skills, and the potential for an allergic reaction to the contrast material exists, but the use of noniodinated agents minimizes that risk.

Materials and Methods

Inclusion Criteria

Eight client-owned dogs were enrolled prospectively in this study. Enrollment in the study required a diagnosis of PE on TTE made by a board-certified cardiologist or veterinarian actively enrolled in an approved cardiology residency program.

General Protocol

All procedures were approved by the institutional animal care and use committee. All dogs had TTE performed at the University of Pennsylvania Veterinary Medical Hospital with a Sonos 5,500

and a 2–4 MHz transducer.^a Each patient was premedicated with midazolam (0.3 mg/kg) and buprenorphine (10 µg/kg) IM, induced with 1.0 mg/kg etomidate, followed by intubation with an appropriately sized endotracheal tube, and maintained at a light plane of anesthesia with isoflurane. Normosol-R was administered IV at an appropriate surgical maintenance fluid rate throughout anesthesia.

CMR with ECG gating was performed with a Siemens Sonata 1.5 T magnet^b utilizing the following pulse sequences: axial, short axis, and 4-chamber ECG-triggered black blood T1-weighted imaging, multiplanar dynamic steady-state free precession (SSFP) ECG-gated cine gradient echo. Precontrast T1-weighted imaging was followed by hand injection of 0.2 mmol/kg gadolinium-DTPA. Immediate postcontrast T1-weighted imaging was performed, followed by delayed inversion recovery-prepped T1-weighted imaging. A single radiologist with subspecialty training in human medical CMR (HIL) performed and assessed all CMR acquisitions. This radiologist was not blinded to the echocardiographic finding of PE but was blinded to the findings of a mass.

Results

At echocardiography, HSA was suspected in 4 dogs, 2 cases were equivocal for a cardiac mass lesion, and 2 had no demonstrable cardiac mass. Two equivocal cases had additional CMR interpretations after considerable experience had been obtained. Cardiac MRI confirmed a cardiac mass and supported the tumor type suspected on echocardiography (based on contrast enhancement) in all 4 dogs with suspected HSA (Fig 1a,b). In 1 dog, HSA was confirmed by surgical biopsy (Fig 2) and in 3 other dogs, disease progression was consistent with this diagnosis (1 presented dead on arrival with recurrent PE).

In 1 equivocal case, a periaortic mass initially was interpreted as periaortic fat, but months later after reevaluation of the images, it was thought to represent a paraganglioma based on signal characteristics and enhancement pattern of the tissue (Fig 3a,b). In this same case, a subsequent thorascopic pericardial window procedure identified a small mass on the ventral aspect of the right atrium, a different location than suspected on echocardiography and CMR. Within a few weeks, the dog presented with a splenic mass, which was not present before the procedure, moderate to severe pleural fluid, and a clearly identifiable cavitated RA mass.

In another equivocal case, an LV thrombus or avulsed cranial papillary muscle was suspected on echocardiography. CMR failed to identify a tumor, but showed a LV thrombus with adjacent wall motion abnormality and delayed hyper-enhancement, thought to represent a complication of prior pericardiocentesis (Fig 4a,b). When initially evaluated by TTE, pericardiocentesis had been performed before the echocardiogram. A mild amount of PE was present as well as mild LA enlargement and an echogenic mass attached to the cranial papillary muscle. Fourteen weeks later, another pericardiocentesis was performed before TTE. No LV mass was noted and there was no indication of a mass attached to the papillary muscle. Cardiac function was normal but mild chronic valve disease was present. No mass was noted and no pericardial or pleural effusion was identified at the time of the examination. Sixteen weeks after

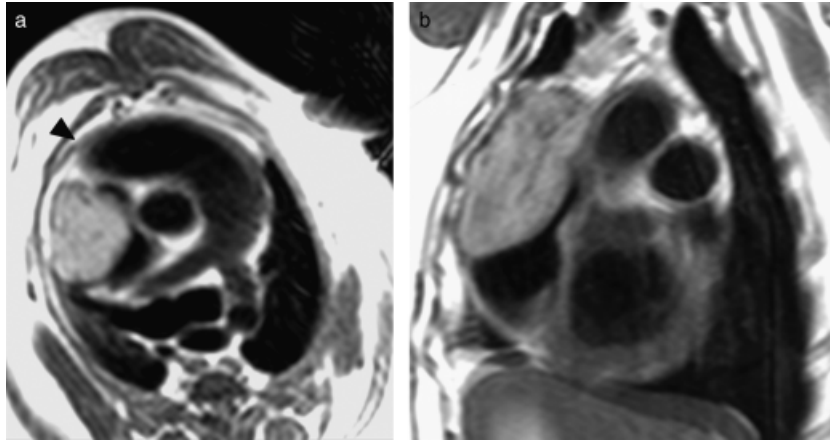


Fig 1. Suspect primary hemangiosarcoma in a dog (a and b). Dorsal dark-blood ECG-triggered T1-weighted spin-echo magnetic resonance images of the heart demonstrate a hyperintense mass located dorsal to and extending into the AV groove. There is a small amount of pericardial fluid present (arrowhead). These images are similar to short-axis transthoracic echocardiography views.

the initial evaluation, PE and a large mass adjacent to the right atrium with slightly hypoechoic echogenicity were identified, and the echocardiographer suspected fibrin. No LV mass was identified but mitral regurgitation and an abnormal anterior papillary muscle were observed. By 20 weeks after the initial evaluation, a 4 cm×6 cm mass was identified in the right atrial appendage arising from the right atrium. The mass was cavitated with areas of hypoechogenicity. No PE was observed and no LV mass was found. Finally, 32 weeks after initial presentation, the dog presented to the emergency service with respiratory difficulty, and large thoracic masses were found on radiography. The dog was discharged

and lost to follow-up. On earlier review, no mass had been observed on CMR, but on later review of the CMR images a mass was visualized.

In 1 dog without echocardiographic evidence of neoplasia, CMR revealed no cardiac tumor, but there were presumptive hepatic and splenic metastases on abdominal magnetic resonance imaging (AMR) (Fig 5).

Discussion

CMR has multiple advantages for the imaging of human patients with pericardial disease, cardiac neoplasia, or both. One main advantage is that CMR allows for the acquisition of images in any tomographic plane without limitations from body condition.⁶⁷ Another main advantage is that CMR has already proven to be effective in tissue characterization and is used clinically to distinguish masses such as thrombi, primary cardiac tumors, and metastases based on tissue characterization.⁶⁴⁻⁶⁷ In fact, today CMR is considered “the gold standard for the comprehensive imaging of pericardial disease and cardiac masses” as it provides superior tissue characterization relative to computed tomography (CT) and all modalities of echocardiography.⁶⁶ In this study, only 1 form of echocardiography (TTE) was chosen for comparison to CMR. Although TEE likely images the heart base better than TTE, it is not the most common form of echocardiography available in veterinary specialty hospitals. Also, MRI units can be used by multiple specialties in veterinary hospitals whereas TEE likely would be used in a limited capacity by 1 specialty.

CMR images of human angiosarcoma have a typical appearance that is heterogeneous on T1-weighted images with areas of low to high signal intensity reflecting tumor tissue, necrosis, and the presence of methemoglobin.⁶⁰ On T2-weighted imaging, angiosarcoma has a predominantly hyperintense appearance with marked surface enhancement, particularly after gadolinium injection.^{60,65,67} On SSFP imaging, the mass is hyperintense relative to the myocardium with areas of high signal

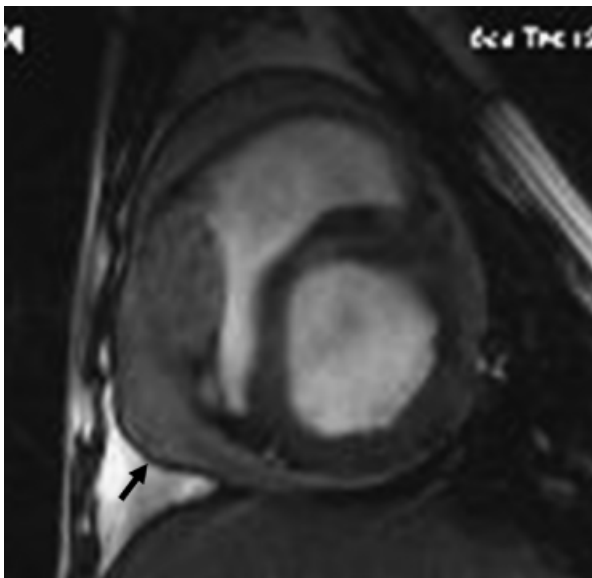


Fig 2. Primary hemangiosarcoma in a different dog. A dorsal ECG-gated steady-state free precession cine image demonstrates a hyperintense mass at the level of the AV groove. There is a small pericardial effusion (arrow). Hemangiosarcoma was confirmed in this patient by surgical biopsy.

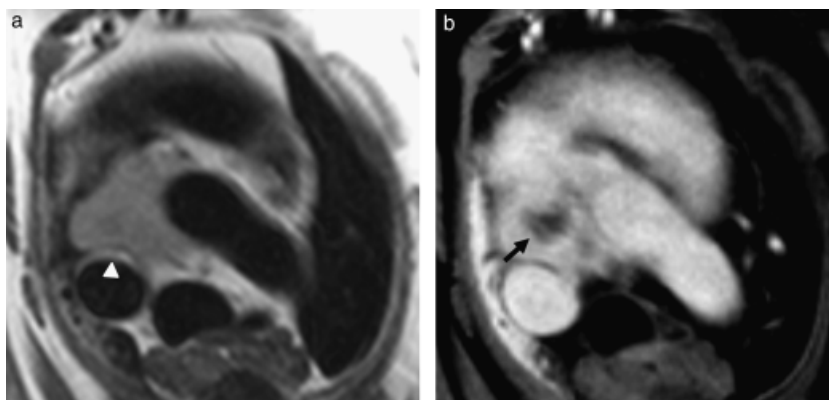


Fig 3. Suspected paraganglioma. (a) Oblique dorsal dark-blood ECG-triggered T1-weighted and (b) oblique dorsal postcontrast nongated T1-weighted imaging with fat saturation demonstrate a peri-aortic mass with preserved fat-plane between mass and the cranial vena cava (arrowhead) and central necrosis on postcontrast imaging (arrow).

intensity corresponding to hemorrhage and areas of low signal intensity corresponding to necrosis.^{60,65,66} Location of the tumor also helps differentiate tumor type because most other sarcomas have a left atrial predilection whereas angiosarcoma has a right atrial predilection.⁶⁶ In this study, suspected HSA cases had findings similar to angiosarcoma in people, and 1 surgical biopsy confirmed HSA, suggesting CMR may allow presumptive diagnosis of this tumor without necessitating biopsy.

One patient with equivocal TTE had a wall motion abnormality and suspected thrombus in the left ventricle on CMR. These findings were thought to be secondary to a complication from pericardiocentesis that was performed in the emergency room upon presentation the previous evening. In people, contrast-enhanced MRI, when compared with TTE or TEE, provided the highest sensitivity and specificity for identification of a LV thrombus and its differentiation from a neoplastic process.⁶⁸ Histopathologic confirmation that this abnormality was in fact a thrombus secondary to pericardiocentesis was not made. However, the location of

the defect was at the right 5th or 6th intercostal space where pericardiocentesis was performed in this patient. Also, the TTE progression was compatible with the diagnosis of a LV thrombus and this defect was not present on follow-up examinations.

Paragangliomas in humans demonstrate high signal intensity on T2-weighted sequences, and avid contrast enhancement with central necrosis.^{65,66} These findings also were seen in our case of suspected paraganglioma. Although CMR is the imaging modality of choice for tissue characterization in humans, we were not able to obtain cytologic confirmation in our canine patient and therefore cannot confirm that these findings truly are similar between human and canine patients. Furthermore this patient did not progress as would be expected in a patient with a paraganglioma. Another complicating factor is that TTE was performed by several different people, which potentially could increase the variability of the results.

Although this study evaluated CMR characterization of PE, HSA metastasis was found in the liver and spleen in a few cases. A previous study at the same institution

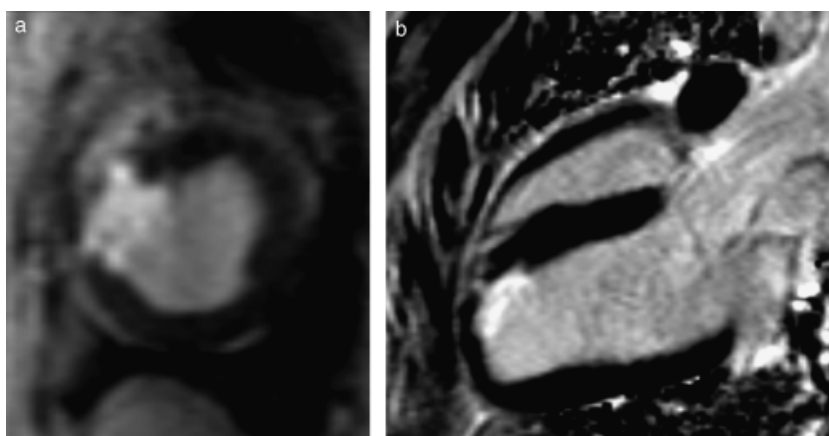


Fig 4. In this dog the echocardiogram was equivocal for a mass and the magnetic resonance imaging demonstrated no tumor; however, (a) dorsal and (b) transverse delayed postcontrast inversion recovery-prepped T1-weighted images demonstrate transmurular hyperenhancement, suggesting a potential complication of pericardiocentesis.

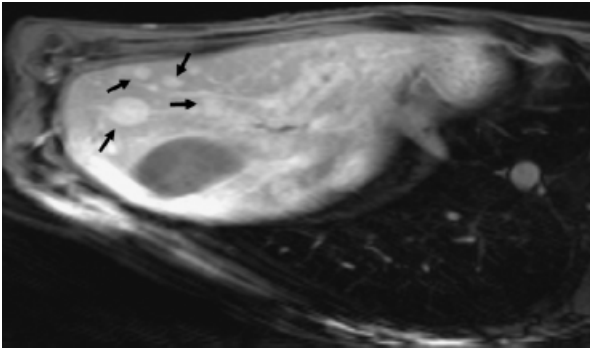


Fig 5. This dog had an equivocal echocardiogram for a mass and normal cardiac magnetic resonance imaging, but there was evidence of metastasis in the liver on postcontrast T1-weighted images (arrows).

suggested that MRI was a useful modality for abdominal imaging, and AMR accurately differentiated benign from malignant focal hepatic and splenic lesions with a sensitivity of 100% and a specificity of 90%.⁶⁹ Some patients may present with microscopic cardiac lesions that result in PE which is not readily seen on TTE or CMR but also may have splenic or hepatic lesions noted on AMR, suggesting that the PE is neoplastic in nature. Patients with HSA may have microscopic metastases and multiple organ imaging can be utilized to evaluate spread of neoplasia. We cannot assume that because AMR findings in the spleen and liver are similar to those observed in humans CMR findings also will be similar, but such a conclusion seems likely in the dog.

CMR offers improved resolution and soft tissue contrast when compared with echocardiography.^{60,65,66} In the hands of an experienced operator, CMR also allows for greater tissue characterization than does TTE or TEE (except for calcium deposition, which requires either surface radiography or CT).^{60,65–67} The greatest limiting factors with veterinary CMR are the training required for this advanced imaging modality and the fact that the technique requires general anesthesia. Experience also can be expected to influence interpretation as well. In our study, masses that were not originally identified were later identified after years of experience had been obtained. Even so, the suspicion that certain masses were consistent with HSA was strengthened after CMR. Also interesting was the fact that the most common location of masses thought to be HSA was the right AV groove. Although CMR in our study did not definitively differentiate between neoplastic and benign PE in the equivocal cases, it did prove beneficial in assessing tumor type, metastases, and complications associated with pericardiocentesis.

During this study, it became clear that ECG gating is very important for CMR acquisition and arrhythmias can result in artifacts. The main limitations of this study were the small sample size and lack of histopathologically confirmed diagnoses for most cases. However, despite these limitations, this study shows that CMR and AMR have the potential to yield clinically relevant information in many cases of canine PE.

Footnotes

^a Phillips Healthcare, Andover, MA

^b Siemens Medical Solutions, Malvern, PA

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References

1. Girard C, Helie P, Odin M. Intrapericardial neoplasia in dogs. *J Vet Diagn Invest* 1999;11:73–78.
2. Walter JH, Dolph R. Systemic, metastatic, Eu- and heterotopie tumours of the heart in necropsied dogs. *J Vet Med* 1996;43:31–45.
3. Ware WA, Hopper DL. Cardiac tumors in dogs: 1982–1995. *J Vet Intern Med* 1999;13:95–103.
4. Kirsch JA, Dhupa S, Cornell KK. Pericardial effusion associated with metastatic disease from an unknown primary tumor in a dog. *J Am Anim Hosp Assoc*. 2000;36:121–124.
5. Palumbo NE. Canine cardiac disease due to metastatic carcinoma. *J Am Vet Med Assoc* 1967;150:396–397.
6. Bright JM, Toal RL, Blackford LM. Right ventricular out-flow obstruction caused by primary cardiac neoplasia: Clinical features in two dogs. *JACVIM* 1990;4:12–16.
7. Darke PG, Gordon LR. Cardiac myxoma in a dog. *Vet Rec* 1974;95:565–567.
8. Briggs OM, Kirberger RM, Goldberg NB. Right atrial myxosarcoma in a dog. *S Afr Vet Assoc* 1997;68:144–146.
9. Lombard CW, Goldschmidt MH. Primary fibroma in the right atrium of a dog. *J Small Anim Pract* 1980;21:439–448.
10. Closa JM, Font A, Mascort J. Pericardial mesothelioma in a dog: Long-term survival after pericardiectomy in combination with chemotherapy. *J. Small Anim Pract* 1990;40:383–386.
11. McDonough SP, MacLachlan NJ, Tobias AH. Canine pericardial mesothelioma. *Vet Pathol* 1992;29:256–260.
12. Ogilvie GK, Brunkow CS, Daniel GB, Haschek WM. Malignant lymphoma with cardiac and bone involvement in a dog. *J Am Vet Med Assoc* 1989;194:793–796.
13. Southerland EM, Miller RT, Jones CL. Primary right atrial chondrosarcoma in a dog. *J Am Vet Med Assoc* 1993;203:1697–1698.
14. Schelling SH, Moses BL. Primary intracardiac osteosarcoma in a dog. *J Vet Diagn Invest* 1994;1:396–398.
15. Vicini DS, Didier PJ, Ogilvie GK. Cardiac fibrosarcoma in a dog. *J Am Vet Med Assoc* 1986;189:1486–1488.
16. Perez J, Perez-Rivero A, Montoya A, et al. Right-sided heart failure in a dog with primary cardiac rhabdomyosarcoma. *J Am Anim Hosp Assoc* 1998;34:208–211.
17. Krotje LJ, Ware WA, Niyo Y. Intracardiac rhabdomyosarcoma in a dog. *J Am Vet Med Assoc* 1990;197:368–371.
18. Gonin-Jmaa D, Paulsenand GP, Taboada J. Pericardial effusion in a dog with rhabdomyosarcoma in the right ventricular wall. *J of Small Anim Pract* 1996;1:193–196.
19. Kolm US, Kleiter M, Kosztolich A, et al. Benign intrapericardial lipoma in the dog. *J Vet Card* 2002;4:25–29.
20. Keene BW, Rush JE, Cooley AJ, Subramanian R. Primary left ventricular hemangiosarcoma diagnosed by endomyocardial biopsy in a dog. *J Am Vet Med Assoc* 1990;197:1501–1503.

21. Kleine LJ, Zook BC, Munson TO. Primary cardiac hemangiosarcomas in dogs. *J Am Vet Med Assoc* 1970;157:326–337.
22. Pearson GR, Head KW. Malignant haemangioendothelioma (angiosarcoma) in the dog. *J Small Anim Pract* 1976;1:737–745.
23. Baskerville A. Ruptured haemangiosarcoma of the right atrium in a dog. *Vet Rec* 1967;1:488–489.
24. Hilbe M, Hauser B, Zlinszky K, Ehrensperger F. Hemangiosarcoma with a metastasis of a malignant mixed mammary gland tumour in a dog. *J Am Vet Med Assoc* 2002;1:443–444.
25. Buchanan JW, Boggs LS, Dewan S, et al. Left atrial paraganglioma in a dog: Echocardiography, surgery, scintigraphy. *J Vet Intern Med* 1998;1:109–115.
26. Sanford SE, Hoover DH, Miller RB. Primary cardiac granular cell tumor in a dog. *Vet Pathol* 1984;1:489–494.
27. Cho K, Park N, Park I, et al. Metastatic intracavitary cardiac aortic body tumor in a dog. *J Vet Med Sci* 1998;60:1251–1253.
28. Balaguer L, Romano J, Nieto J, et al. Incidental finding of a chemodectoma in a dog: Differential diagnosis. *J Vet Diagn Invest* 1990;1:339–341.
29. Srebernik N, Appleby EC. Breed prevalence and sites of haemangioma and hemangiosarcoma in dogs. *Vet Rec* 1991;1:408–409.
30. Kock ND, Lane EP, Rowbotham F, et al. Concurrent systemic cryptococcosis and hemangiosarcoma in a dog. *J Comp Path* 1991;104:117–120.
31. Chastain CB, Riedesel DH, Graham DL. Ventricular septal hemangiosarcoma associated with right bundle branch block in a dog. *J Am Vet Med Assoc* 1974;165:177–179.
32. Brown NO, Patnaik AK, MacEwen EG. Canine hemangiosarcoma: Retrospective analysis of 104 cases. *J Am Vet Med Assoc* 1985;186:56–58.
33. Aronsohn M. Cardiac hemangiosarcoma in the dog: A review of 38 cases. *J Am Vet Med Assoc* 1985;187:922–926.
34. MacGregor JM, Faria ML, Moore AS, et al. Cardiac lymphoma and pericardial effusion in dogs: 12 cases (1994–2004). *J Am Vet Med Assoc* 2005;227:1449–1453.
35. Brisson BA, Reggeti F, Bienzle B. Portal site metastasis of invasive mesothelioma after diagnostic thoracoscopy in a dog. *J Am Vet Med Assoc* 2006;229:980–983.
36. Guglielmini C, Civitella C, Malatesta D, Palmieri C. Metastatic pericardial tumors in a dog with equivocal pericardial cytological findings. *J Am Anim Hosp Assoc* 2007;43:284–287.
37. Fewes D, Scase TJ, Battersby IA. Leiomyosarcoma of the pericardium, with epicardial metastases and peripheral eosinophilia in a dog. *J Comp Pathol* 2008;138:224–228.
38. Jackson J, Richter KP, Launer DP. Thorascopic partial pericardiectomy in 13 dogs. *J Vet Intern Med* 1999;1:529–533.
39. Kerstetter KK, Krahwinkel DJ, Millis DL, Hahn K. Pericardiectomy in dogs: 22 cases (1978–1994). *J Am Vet Med Assoc* 1997;211:736–740.
40. Aronsohn MG, Carpenter JL. Surgical treatment of idiopathic pericardial effusion in the dog: 25 cases (1978–1993). *J Am Anim Hosp Assoc* 1999;1:521–525.
41. Johnson MS, Martin M, Binns S, Day MJ. A retrospective study of clinical findings, treatment and outcome in 143 dogs with pericardial effusion. *J Small Anim Pract* 2004;45:546–552.
42. Mellanby RJ, Herrtage MH. Long-term survival of 23 dogs with pericardial effusions. *Vet Rec* 2005;156:568–571.
43. Heinritz CK, Gilson SD, Soderstrom MJ, et al. Subtotal pericardiectomy and epicardial excision for treatment of coccidiomycosis-induced effusive-constrictive pericarditis in dogs: 17 cases (1999–2003). *J Am Vet Med Assoc* 2005;227:435–440.
44. Boston SE, Moens NM, Martin DM. Idiopathic primary chylopericardium in a dog. *J Am Vet Med Assoc* 2006;229:1930–1933.
45. Day MJ, Martin MWS. Immunohistochemical characterization of the lesions of canine idiopathic pericarditis. *J Small Anim Prac* 2006;43:382–387.
46. Sadanaga KK, MacDonald MJ, Buchanan JW. Echocardiography and surgery in a dog with left atrial rupture and hemopericardium. *J Vet Intern Med* 1990;4:216–221.
47. Wright KN, DeNovo RC, Patton CS, et al. Effusive-constrictive pericardial disease secondary to osseous metaplasia of the pericardium in a dog. *J Am Vet Med Assoc* 1996;209:2091–2095.
48. Sisson D, Thomas WP, Reed J, et al. Intrapericardial cysts in the dog. *J Vet Intern Med* 1993;7:364–369.
49. Kolm US, Kosztolich A, Hoegler S, Kneissl S. Canine traumatic pericarditis by an esophageal foreign body. *J Vet Card* 2001;3:17–21.
50. Shubitz LF, Matz ME, Noon TH, et al. Constrictive pericarditis secondary to *Coccidioides immitis* infection in a dog. *J Am Vet Med Assoc* 2001;218:537–540.
51. Sisson D, Thomas WP, Ruehl WW, et al. Diagnostic value of pericardial fluid analysis in the dog. *J Am Vet Med Assoc* 1984;184:51–55.
52. Edwards NJ. The diagnostic value of pericardial fluid pH determination. *J Am Anim Hosp Assoc* 1996;32:63–67.
53. Fine DH, Tobias AH, Jacob KA. Use of pericardial fluid pH to distinguish between idiopathic and neoplastic effusions. *J Vet Intern Med* 2003;17:525–529.
54. Bauer N, Moritz A. Flow cytometric analysis of effusions in dogs and cats with the automated haematology analyser ADVIA 120. *Vet Rec* 2005;156:674–678.
55. Linde A, Summerfield NJ, Sleeper MM, et al. Pilot study on cardiac troponin I levels in dogs with pericardial effusion. *J Vet Cardiol* 2006;8:19–23.
56. Shaw SP, Rozanski EA, Rush JE. Cardiac troponins I and T in dogs with pericardial effusion. *J Vet Intern Med* 2004;18:322–324.
57. de Laforcade AM, Freeman LM, Rozanski EA, Rush JE. Biochemical analysis of pericardial fluid and whole blood in dogs with pericardial effusion. *J Vet Intern Med* 2005;19:833–836.
58. Stepien RL, Whitley NT, Dubielzig RR. Idiopathic or mesothelioma-related pericardial effusion: Clinical findings and survival in 17 dogs studied retrospectively. *J Small Anim Pract* 2000;41:342–347.
59. Kim EY, Choe YH, Sung K, et al. Multidetector CT, MR imaging of cardiac tumors. *Korean J Radiol* 2009;10:164–175.
60. Sparrow PJ, Kurian JB, Jones TR, Sivananthan MU. MR imaging of cardiac tumors. *Radiographics* 2005;25:1255–1276.
61. Funari M, Fujita N, Peck WW, Higgins CB. Cardiac tumors: Assessment with Gd-DTPA enhanced MR imaging. *J Comput Assist Tomogr* 1991;15:953–958.
62. Semelka RC, Shoenut JP, Wilson ME, et al. Cardiac masses: Signal intensity features on spin-echo, gradient-echo, gadolinium-enhanced spin-echo, and TurboFLASH images. *J Magn Reson Imaging* 1992;2:415–420.
63. Siripornpitak S, Higgins CB. MRI of primary and malignant cardiovascular tissue. *J Comput Assist Tomogr* 1997;21:462–466.
64. Gulati G, Sharma S, Kothari SS, et al. Comparison of echo and MRI in the imaging evaluation of intracardiac masses. *Cardiovas Interv Radiol* 2004;27:459–469.
65. Hoey ETD, Mankad K, Jones JB, et al. MRI and CT appearances of cardiac tumours in adults. *Clin Radiol* 2009;64:1214–1230.
66. Ang GB, Grizzard JD. Magnetic resonance imaging of pericardial disease and cardiac masses. *Magn Reson Imaging Clin N Am* 2007;15:579–607.

67. Hundley WG, Bluemke DA, Finn JP, et al. ACCF/ACR/AHA/NASCI/SCMR 2010 expert consensus document on cardiovascular magnetic resonance. *J Am Coll Cardiol* 2010;55:2614–2662.
68. Srichai MB, Junor C, Rodriguez LL, Stillman AE, et al. Clinical, imaging, and pathological characteristics of left ventricular thrombus: A comparison of contrast-enhanced magnetic resonance imaging, transthoracic echocardiography, and transesophageal echocardiography with surgical or pathological validation. *Am Heart J* 2006;152:75–84.
69. Clifford CA, Pretorius ES, Weisse C, et al. Magnetic resonance imaging of focal splenic and hepatic lesions in the dog. *J Vet Intern Med* 2004;18:330–338.