A 1-year-old castrated male German Shepherd Dog undergoing bite training was evaluated by the emergency service of the Massachusetts Veterinary Referral Hospital because of an acute onset of lethargy, tachypnea, and inappetence. On initial physical examination, the patient was tachypneic (respiratory rate, 86 breaths/min; reference range, 8 to 40 breaths/min). Heart sounds were muffled on auscultation of the right hemithorax, femoral pulse quality was weak bilaterally, and there was a palpable abdominal fluid wave. Systolic blood pressure measured with an oscillometric technique was 60 mm Hg (reference range, 90 to 160 mm Hg), and heart rate was 160 beats/min (reference range, 80 to 140 beats/min). A brief initial transthoracic ultrasound examination showed a large volume of pericardial effusion. Standard 3-view thoracic radiography revealed a markedly enlarged cardiac silhouette with granulomatous inflammation and fibroplasia. The patient also underwent exploratory laparotomy and umbilical herniorrhaphy during the same anesthetic episode and recovered from surgery without apparent complications. There were no further clinical signs of cardiac disease.

CLINICAL RELEVANCE
The patient described in the present report underwent successful subtotal pericardiectomy for treatment of a benign focal lesion causing recurrent pericardial effusion and cardiac tamponade. Prompt diagnosis and intervention may have contributed to the positive outcome in this case. (J Am Vet Med Assoc 2017;251:201–205)
nostic testing and with the patient’s condition stable, the dog was discharged from the hospital with no additional treatment recommendations at that time. A 7-day follow-up examination was scheduled.

The patient returned to our hospital for reevaluation 2 days after discharge because of restlessness, lethargy, and inappetence. Repeated echocardiography performed by the cardiology service revealed focal pericardial effusion adjacent to the right side of the cardiac apex, with mild compression of the adjacent right ventricle. Therapeutic pericardiotomy was again performed, and thoracotomy and pericardiectomy were scheduled for the following day. A CBC and serum biochemical analysis indicated slightly high alanine transaminase activity (140 U/L; reference range, 18 to 121 U/L), hyperphosphatemia (6.5 mg/dL; reference range, 2.5 to 6.1 mg/dL) consistent with a young growing dog, leukocytosis (21 × 10^3 cells/µL; reference range, 5 × 10^3 cells/µL to 17.5 × 10^3 cells/µL), neutrophilia (16 × 10^3 cells/µL; reference range, 3 × 10^3 cells/µL to 13 × 10^3 cells/µL), and monocytosis (2.5 × 10^3 cells/µL; reference range, 0.15 × 10^3 cells/µL to 1 × 10^3 cells/µL). Transabdominal ultrasonography revealed a bilobed umbilical hernia containing fat, with the caudal aspect appearing to be a full-thickness defect of the ventral abdominal wall.

An IV catheter was placed in a cephalic vein, the patient was sedated with butorphanol (0.2 mg/kg, IV) and midazolam (0.2 mg/kg) IV, and general anesthesia was induced with propofol (4 mg/kg [1.8 mg/lb]), IV.

**Figure 1**—Right lateral (A) and ventrodorsal (B) thoracic radiographic images of a 1-year-old castrated male German Shepherd Dog examined for an acute onset of lethargy, tachypnea, and inappetence. Note the markedly enlarged cardiac silhouette with a globoid shape suggestive of a diagnosis of pericardial effusion.

**Figure 2**—Right parasternal long axis (A) and left parasternal (B) transthoracic echocardiographic images of the patient in Figure 1. The patient is positioned in right lateral recumbency. Note the pericardial effusion and cardiac tamponade characterized by right ventricular compression (A). The effusion is localized to the right side of the cardiac apex (B). No masses are evident.
After endotracheal intubation, anesthesia was maintained with isoflurane in oxygen. Heart rate (by means of a lead II ECG), arterial oxygen saturation (by means of pulse oximetry), and respiratory rate were monitored throughout with a multiparameter monitor. A right fifth intercostal thoracotomy was performed to approach the pericardium. The pericardium was palpably thickened, and a 4-cm ventral incision was made. A mass was noted between the parietal and visceral layers of the pericardium. The mass (3 x 5 cm) grossly resembled a pale lobe of liver. A stalk of pink, firm, cord-like tissue extended from the mass to the apex of the pericardium, traversing the pericardiodiaphragmatic ligament and extending to the right crus of the diaphragm. After careful inspection, the mass and stalk were carefully excised with a combination of sharp and blunt dissection and a commercial blunt tip vessel sealer and divider. The mass was submitted for histologic examination. A subtotal pericardectomy was then performed. On completion, the patient was repositioned from left lateral to dorsal recumbency, and an exploratory laparotomy was performed to ensure that no diaphragmatic defect was present (none noted). The umbilical hernias were then repaired by means of routine techniques, and the dog recovered from anesthesia without apparent complications. The patient was discharged with amoxicillin-clavulanic acid (14 mg/kg [6.4 mg/lb], PO, q 12 h) and carprofen (2 mg/kg [0.9 mg/lb], PO, q 12 h) pending results of histologic examination. A follow-up echocardiographic evaluation was performed at the time of suture removal 2 weeks after surgery, and results were unremarkable. At the time of most recent follow-up 3 months after surgery, the owner noted that the dog continued to do well without any apparent problems.

Histologic examination of the pericardial mass indicated nodular adipose tissue with focal fat necrosis and associated granulomatous inflammation intermixed with granulation tissue. Viable adipose tissue abutted a wide band of reactive fibrovascular stroma intermixed with a moderate amount of fibrin. The histologic diagnosis was necrotic adipose tissue with granulomatous inflammation and fibroplasia. The clinical diagnosis was trauma to the pericardial adipose tissue; however, the pathologist commented that the spectrum of histologic changes shared some features with a strangulated lipoma.

**Discussion**

Pericardial effusion is the abnormal accumulation of fluid within the pericardial space and is the most common disease of the pericardium. The normal pericardium is composed of the outer fibrous pericardium and the parietal and visceral layers that comprise the inner serous pericardium. A small volume of serous fluid is normally present within the pericardium. Cardiac tamponade refers to a pathological state in which pericardial effusion causes an increase in intrapericardial pressure of sufficient magnitude to impair ventricular filling and reduce cardiac output. The most common causes of acquired pericardial effusion in older dogs are neoplasia and idiopathic pericarditis. Congenital pericardial defects include peritoneopericardial diaphragmatic hernia (also referred to as parietal pericardial diaphragmatic hernia), which occurs when the septum transversum improperly fuses with the pleuroperitoneal folds, and pericardial cysts, which result from entrapment of the omentum or the falciform ligament in the pericardium during embryonic development. Intrapericardial cysts in dogs and cats share histopathologic characteristics with those described in human patients and consist of a thick, fibrous tissue capsule surrounding a fluid-filled lumen. However, pericardial cysts, which can be congenital or acquired secondary to thoracic trauma, are reportedly rare.

Intrathoracic and pericardial lipomas have been reported in dogs. Lipomas are more common in older dogs but have been known to affect younger dogs. In human patients, primary cardiac lipomas are rare, reportedly accounting for 10% of all primary cardiac tumors and 14% of benign cardiac tumors. In dogs, only a small volume of adipose tissue is located around the coronary arteries, with none located in the pericardium. For the patient of the present report, it is possible that a lipoma or intra-abdominal fat became strangulated during embryonic development and formation of the pericardium; alternatively, the pericardial adipose mass may have developed secondary to trauma. At the time of follow-up, the owners noted that the dog had recently been undergoing bite training, which may have served as a source of repetitive thoracic trauma, thus causing pericardial effusion secondary to vascular obstruction of the adipose tissue.

Cardiac tamponade can develop as the result of direct compression from a cyst or as a result of pericardial effusion. The patient described in the present report was younger than most patients in a prior report on intrapericardial cysts in 6 dogs (ages, 6 months to 3 years). No single examination finding is pathognomonic for a diagnosis of pericardial effusion; rather, a combination of muffled heart sounds, jugular venous distention, poor pulse quality, and pulsus paradoxus should result in a high index of suspicion for this diagnosis. In patients with unrecognized, untreated progressive pericardial effusion, clinical signs will eventually progress to cardiogenic shock because of decreased cardiac output.

In the patient described in the present report, the pericardial effusion rapidly recurred after initial pericardiocentesis. Radiographic features of pericardial effusion characteristically include an enlarged cardiac silhouette with a globoid shape. However, thoracic radiography is neither sensitive nor specific for a diagnosis of cardiac tamponade secondary to pericardial effusion in dogs. Transthoracic echocardiography is the most sensitive and specific modality for the diagnosis of pericardial effusion and cardiac tamponade, allowing for estimation of pericardial fluid volume, measurement of pericardial thickness, and identification of concurrent right atrial or heart base...
masses. The patient of this report showed evidence of cardiac tamponade resulting from compression of the right ventricle because of intramural pericardial effusion on the initial echocardiogram. No distinct masses were evident. Cardiac magnetic resonance imaging is the diagnostic imaging modality of choice for evaluation for potential cardiac tumors in human patients. However, a recent study did not find a substantial benefit when compared with echocardiography for the differentiation of neoplastic and nonneoplastic pericardial effusion in dogs.

In patients with recurrent idiopathic pericardial effusion, pericardectomy is indicated and can be achieved by means of open thoracotomy (median sternotomy or intercostal approach) or thoracoscopy. The prognosis for patients with idiopathic and nonneoplastic effusions is generally good. Percutaneous balloon pericardiectomy has also been described as a palliative measure for management of recurrent pericardial effusion in human patients and dogs. A previous case report described an intrapericardial lipoma in a dog. A distinct intrapericardial mass was noted on transthoracic echocardiography. The inability to identify a distinct mass on transthoracic echocardiography for the dog of the present report may have been because of the location of the mass between the layers of the visceral and parietal pericardium. The location of the pericardial effusion was notably different also, with focal right-sided fluid predominating. A pericardectomy was recommended for treatment in the patient described here because of the focal hemorrhagic pericardial effusion and the lack of a distinct right atrial or heart base mass on echocardiography. Idiopathic pericardial effusion and a pericardial cyst were the working differential diagnoses prior to surgery. One previous report detailed the initial medical management and definitive surgical treatment of pericardial effusion secondary to a cystic pericardial lesion in a dog, presumed by the authors to be caused by a resolving abscess or hematoma. Possible etiologies included abnormal fetal development, fetal or other trauma during birth, or focal bacterial infection or hemorrhage with subsequent hematoma formation and compartmentalization. For the dog of the present report, the results of histologic examination of surgical tissue specimens did not support a diagnosis of a cyst or cystic lesion, although the mass was attached to the diaphragm. Nor was there evidence to suggest the mass was related to a parietal pericardial diaphragmatic hernia. It is possible the necrotic adipose tissue identified histologically represented a herniated portion of omentum or falciform fat despite the lack of a hernia noted during the exploratory laparotomy.

It was interesting to note the umbilical hernias present in the patient of this report, documenting an error in embryonic development. Burns et al identified concurrent congenital abnormalities in 16 of 28 (57%) dogs with parietal pericardial diaphragmatic hernias, including supraumbilical hernias, umbilical hernias, and cryptorchidism. We suggest that the normal gross intraoperative appearance of the diaphragm in our patient did not exclude the possibility of closure of a prior parietal pericardial diaphragmatic hernia. The presence of mitral valve regurgitation in the patient of this report, most commonly detected in small mixed-breed dogs, may further suggest congenital abnormalities in this patient. Abdominal ultrasonography and echocardiography may be recommended by clinicians as part of the diagnostic evaluation for those patients in which umbilical hernias are present, to further investigate the possibility of other congenital abnormalities.

Pericardial effusion is associated with a good prognosis with prompt diagnosis and appropriate surgical treatment. We are not aware of prior published cases of intramural focal pericardial effusion secondary to necrotic adipose tissue without evidence of a parietal pericardial diaphragmatic hernia, pericardial lipoma, or benign pericardial cyst in dogs. In the patient of the present report, the presence of a benign lesion and prompt surgical intervention contributed to a positive outcome.

References

Racing performance of Standardbred trotting horses undergoing surgery of the carpal flexor sheath and age- and sex-matched control horses

James L. Carmalt et al

OBJECTIVE
To determine factors affecting race speed in Swedish Standardbred horses undergoing surgery of the carpal flexor sheath (CFS), to investigate whether preoperative racing speed was associated with specific intraoperative findings and whether horses returned to racing, and to compare the performance of horses undergoing surgery of the CFS with that of age- and sex-matched control horses.

ANIMALS
149 Swedish Standardbred trotters undergoing surgery of the CFS and 274 age- and sex-matched control horses.

PROCEDURES
Medical records of CFS horses were examined. Racing data for CFS and control horses were retrieved from official online records. Generalizing estimating equations were used to examine overall and pre-surgery association of preoperative clinical and intraoperative findings with preoperative and postoperative speeds. Multivariable regression analysis was used to examine career earnings and number of career races. Kaplan-Meier survival analysis was used to compare career longevity between CFS and control horses.

RESULTS
CFS horses were significantly faster than control horses. The CFS horses that raced before surgery were slower as they approached the surgery date, but race speed increased after surgery. There were 124 of 137 (90.5%) CFS horses that raced after surgery. No intrathecal pathological findings were significantly associated with preoperative racing speed. Career longevity did not differ between CFS and control horses.

CONCLUSIONS AND CLINICAL RELEVANCE
Horses undergoing surgery of the CFS had a good prognosis to return to racing after surgery. Racing careers of horses undergoing surgery of the CFS were not significantly different from racing careers of control horses. (Am J Vet Res 2017;78:847–853)

From this month's AJVR

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