the γ-globulin content of the effusion was >32%.[11] Unfortunately, a negative DIF test on the effusion cannot be used to exclude the possibility of FIP; the NPV of this test is lower than that observed for certain other tests used alone or together (lymphopenia hyperglobulinemia, anti-FCoV titers).[12]

For the effusive form of FIP, protein analysis, cytology, and the DIF test for FCoV performed on the effusion could improve the probability of correct diagnosis of the disease.

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Sources and manufacturers

a. Abbott Laboratories, Irving, TX.
b. Abbott Laboratories, Abbott Park, IL.
c. Cytospin 2, Shandon Scientific, Runcorn, Cheshire, UK.
d. VMRD, Pullman, WA.

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J Vet Diagn Invest 11:361–365 (1999)

Cor triatriatum dexter in an English Bulldog puppy: case report and literature review

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A 3.5-month-old male English Bulldog was presented to a referral veterinary practice with a primary complaint of abdominal distention. The dog was previously presented to the same practice with a complaint of cyanosis when nursing at 3 weeks of age. Clinical examination at that time did not reveal any defects of the hard or soft palate and the puppy was bright, alert, and responsive. However, there was dorsoventral compression of the thorax and the rear legs were splayed. Abdominal distention, coughing, and respiratory distress developed at 2 months of age, and the puppy was reevaluated. Abdominocentesis was performed, and 220 ml of clear red fluid was removed but was not evaluated. Ascites developed again, and abdominocentesis was performed at 3.5 months of age. Laboratory evaluation of the fluid yielded a hemorrhagic modified transudate with a protein concentration of 2.6 g/dl. There were 2,500 nucleated cells mm^3 with 52% monocytes, 25% lymphocytes, 15% neutrophils, and 8% eosinophils. There were 150,000 red blood cells mm^3. Erythrophagocytic macrophages and rare mesothelial cells were observed. A blood sample was obtained. Serum chemistry values included a total protein value of 4.8 g/dl, alkaline phophatase of 81 units/liter, alanine aminotransferase of 117 units/liter, phosphorus of 8.7 mg/dl, potassium of 6.3 meq/liter, albumin/globulin ratio of 2.0, and

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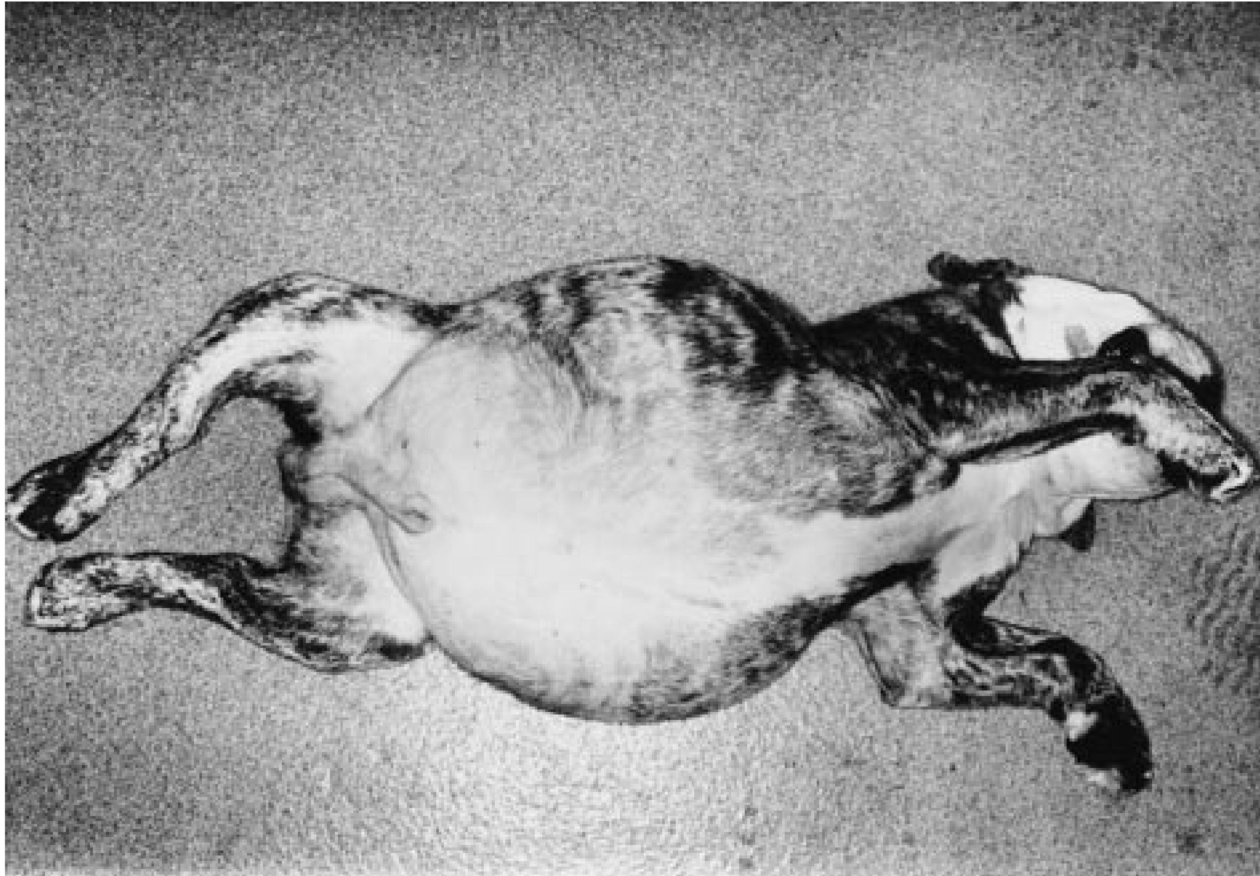

Figure 1. Radiograph. Ventral view of puppy with cor triatriatum dexter. Note marked abdominal distention due to ascites.

globulin of 1.6 g/dl. The hematocrit was 30.0%, and the reticulocyte count was 6.2%. The white blood cell count was 13,800 cells/mm^3, with 55% neutrophils, 0% bands, 37% lymphocytes, 1% monocytes, and 7% eosinophils. Radiographs were taken. Euthanasia was performed, and the dog was presented along with the radiographs to the necropsy service of the Virginia-Maryland Regional College of Veterinary Medicine for complete necropsy.

The heart was poorly visualized on the lateral radographic view because of superimposition of the front limbs. There was no significant left heart enlargement, and the area of the caudal vena cava was not well visualized. The heart was better visualized on the thoracic dorsoventral view, and there was a prominent bulge in the area of the caudal vena cava. No significant pulmonary infiltrate was noted. The abdomen was markedly distended with ascites. A source of the ascites was not identified.

At necropsy, the puppy had a markedly distended abdomen (Fig. 1), the abdominal wall was thin, and there was a ballotable fluid wave. The abdomen contained >2 liters of clear red fluid. The liver was enlarged and represented approximately 7.3% of the body weight. Hepatic margins were rounded, and cut surfaces bulged, were wet, and exuded blood. Hepatic veins and the caudal vena cava were distended. Evaluation of the heart revealed a bipartate right atrium composed of cranial and caudal chambers separated by a thin white septum. The caudal and cranial chambers communicated through a 3-mm-diameter orifice located in the center of the septum (Fig. 2). The caudal vena cava entered the caudal chamber caudally, and the septum was positioned cranially. The cranial chamber contained the openings of the right azygous vein, cranial vena cava, and coronary sinus, and it communicated with the right ventricle via a normal right atrioventricular orifice and valve. The fossa ovalis was immediately cranial to the septum and dorsal to the coronary sinus. The remainder of the heart was unremarkable.

Histologically, the wall of the caudal chamber of the right atrium was composed of normal cardiac muscle and did not differ from the musculature of the cranial chamber. The septum was composed of endothelium-lined fibroelastic connective tissue that was continuous with the endocardium of both the cranial and caudal chambers. Hepatic sinusoids were markedly dilated and filled with blood, consistent with passive hepatic congestion. Other tissues were histologically normal.

In humans, cor triatriatum and cor triatriatum dexter are terms that designate anomalous partitioning of the left and right atria, respectively.[9] Cor triatriatum results from failure of the common pulmonary vein to incorporate into the left atrium and corresponds to partitioning of the left atrium into a proximal or accessory and distal chambers. The proximal chamber receives venous blood from the lungs via the pulmonary veins, and the distal chamber includes the foramen ovale and left atrial appendage. The distal chamber communicates with the left ventricle via the left atrioventricular

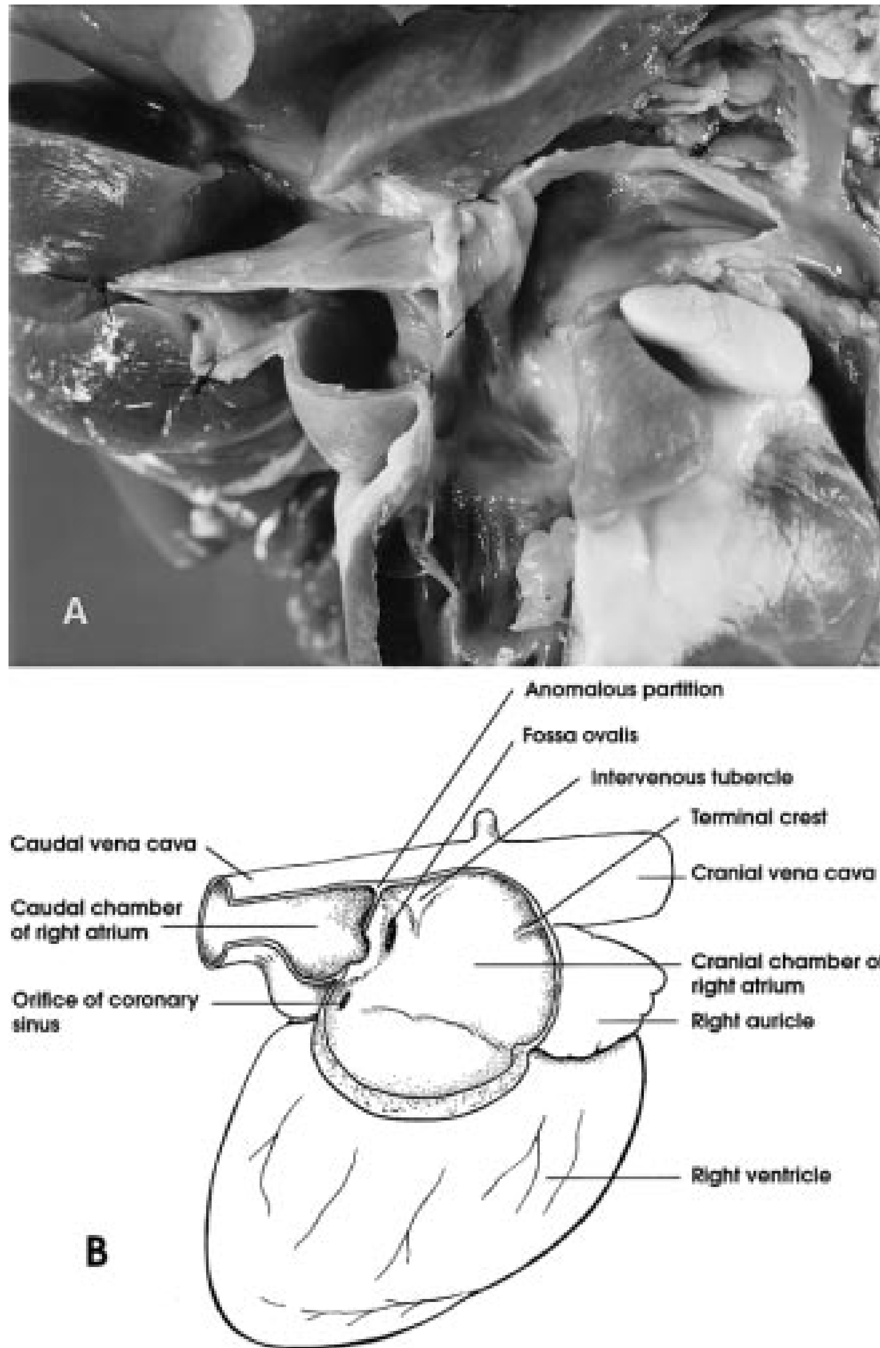

Figure 2. Photograph (**A**) and line drawing (**B**) of the formalin-fixed cardiac specimen from a puppy with cor triatriatum dexter. The specimen has been dissected in cut-away view to demonstrate anomalous partitioning of the right atrium by a septum or diaphragm; small arrows indicate the axial edge of the septum orifice. The abaxial portion of the septum has been cut away.

or mitral valve. This anomaly is rare and has a prevalence of approximately 0.1% of cases of congenital heart disease.[9]

Cor triatriatum dexter is a very rare malformation where the right atrium is partitioned into cranial and caudal chambers by a diaphragm or septum.[9] Previous reports have suggested that the abnormal partitioning results from persistent valve remnants of the sinus venosus.[2,6,11,12,14] Typically, the caudal vena cava and fossa ovalis are present in the caudal chamber and the coronary sinus and right atrioventricular valve are in the cranial chamber.[9] However, several anatomic variations have been reported:[11] 1) a septum-like membrane extending across the right atrium laterally from the caudal vena cava to below the coronary sinus and from the crista terminalis to the medial atrial wall, 2) a membrane obstructing the openings of the cranial and caudal venae cavae (the coronary sinus empties into the cranial right atrial chamber), 3) the membrane covering only the caudal vena cava, 4) the ostium of the coronary sinus being excluded from the true

right atrium, 5) the caudal vena cava and coronary sinus being excluded from the true right atrium, and 6) a fibrous sac resembling a windsock being formed and stretched forward by the blood current.[11]

In dogs, cor triatriatum dexter has been reported 8 times previously, in a total 14 dogs,[6,7,11–13] and anatomic variations occur. The caudal chamber in 5 of these 14 dogs contained the entrances of the caudal vena cava and coronary sinus.[7,11–13] In another dog, the caudal vena cava entered the caudal chamber, and the coronary sinus entered the cranial chamber.[8] In other dogs, the location of the coronary sinus was not characterized.[1,4–6] Anatomic variations of cor triatriatum in the dog have included division of the right atrium by both perforate[1,4,6–8,12] or imperforate[4,5,11–13] septa. Previous reports document partitioning of the right atrium by an incomplete septum with the entrances of the caudal vena cava and coronary sinuses located in the caudal and cranial chambers, respectively, the caudal vena cava[6,7,12] and coronary sinus[7,12] entrances located in the caudal chamber, or complete separation of the cranial and caudal chambers by a complete intratrial septum, again with the caudal vena cava and coronary sinus located in the caudal chamber.[11–13] In dogs with imperforate septa, anomalous collateral vessels usually connected the caudal vena cava to the azygous or vertebral venous circulation.[4,5,12] In previous cases, the fossa ovalis has been located in the caudal chamber of the right atrium.[7,11,13] In the present case, the entrance of the caudal vena cava was located in the caudal chamber of the right atrium and there was an incomplete intraatrial septum at the level of the intervenous tubercle. The fossa ovalis and the entrance of the coronary sinus were located in the cranial chamber. One other report has documented anatomic features similar to those in this dog.[8]

In normal development of the embryonic heart, the sinus venosus serves as a receiving chamber for blood entering the primitive atrium from the right and left common cardinal veins and the developing caudal vena cava (Fig. 3A).[10] The left and right sinus valves delimit the opening, the sinuatrial orifice, between the sinus venosus and the primitive atrium (Fig. 3B). With continued development of the heart and great vessels, including interatrial partitioning by the septa primum and secundum, the wall of the right sinus venosus becomes incorporated into the sinus venarum cavarum.[10] This smooth portion of the definitive right atrium receives blood entering directly from the venae cavae. The cranial part of the right and left valves of the sinus venosus normally fuse with the septum spurium and give rise to the terminal crest, the ridge of atrial tissue that separates the sinus venarum cavarum from the rough right auricle.[2,10,14] The left valve becomes absorbed into the septum secundum of the developing interatrial septum (Fig. 3C).[10] Portions of the right sinus valve may variably persist as the valves of the caudal vena cava and coronary sinus, formerly the eustachian and thebesian valves, respectively.[2,10,14]

The likely source of partitioning of the right atrium in this case is persistence of most of the caudal part of the right valve of the sinus venosus. The aberrant caudal chamber of the right atrium, formerly the caudal portion of the sinus venosus, was not absorbed into the wall of the sinus venarum cavarum of the definitive right atrium. This explanation would account for the positioning of the intervenous tubercle, fossa ovalis, and orifice of the coronary sinus in the cranial chamber of the right atrium, cranial to the anomalous septum. It follows that, if the caudal-most portion of the right sinus valve had also persisted, the opening of the coronary sinus would have been located in the caudal chamber as reported in previous cases.

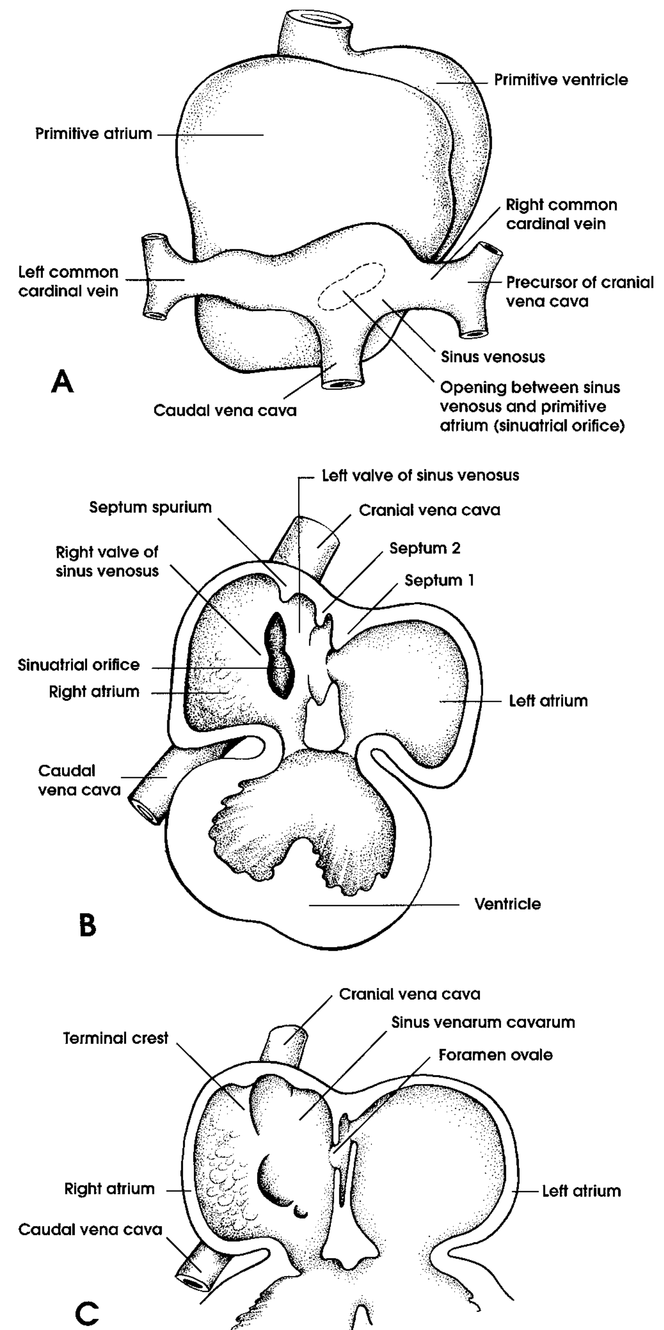

Figure 3. Normal embryonic development of the heart. **A**. External view of the dorsal aspect of the embryonic heart with the sinus venosus superimposed over the primitive atrium. **B**. Open view of the developing embryonic heart showing communication between the sinus venosis and right atrium through an orifice delimited by the right and left valves of the sinus venosus. **C**. Developing atria following absorption of the sinus venosus.

Functional consequences of cor triatriatum dexter in dogs is dependent on presence or absence and the size of the opening in the septum between the two compartments.[7] The main historical finding in this case and most previously reported cases is prolonged abdominal distention due to ascites, ranging from 6 weeks to 2 years in duration.[4–8,11–13] Ascites was not the main clinical feature in 7-year-old German short-haired pointer that presented with exercise intolerance and episodic weakness of over >3 years' duration[12] or in a 6-year-old German shepherd dog cross that presented with a 24-hour history of lethargy.[1] Age at diagnosis ranged from 8 weeks to 7 years, and breeds included chow chow,[3,4,7] cocker spaniel,[8,12] English bulldog,[4] German shepherd dog cross,[1] German short-haired pointer,[12] golden retriever,[13] greyhound,[4] rottweiler,[5,6] and mixed breed.[4,11] Another common finding in previous cases is hepatic congestion and/or hepatomegaly.[6,7,13] Venous engorgement and distention were noted in the abdominal veins and in the body wall in previous cases.[7,11–13] Where ascitic fluid was evaluated, there were high-protein (3.4–5.8 g/dl) modified transudates with specific gravities ranging from 1.015 to 1.028.[4,6,7,12,13]

Cor triatriatum dexter is an extremely uncommon congenital heart defect in dogs. This is the ninth report of canine cor triatriatum dexter, and it represents a third unique anatomic variant, which is characterized by partitioning of the right atrium by a perforate diaphragm or septum with the coronary sinus and fossa ovalis located in the cranial chamber. Cor triatriatum dexter represents a diagnostic challenge at necropsy, requiring careful examination of the heart. This defect should be considered as a differential diagnosis in young dogs with signs of abdominal distention and ascites.

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Fatal canine parvovirus type-1 infection in pups from Italy

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Canine parvovirus type 1 (CPV-1), also known as minute virus of canines (MVC), is an autonomous parvovirus of dogs that was isolated in 1967 from normal canine feces.[1] Antigenic and genomic properties of MVC are distinct from those of canine parvovirus type 2 (CPV-2), which emerged in 1978 in the canine population and is responsible for worldwide outbreaks of severe hemorrhagic gastroenteritis in dogs.[2,3]

The natural pathogenicity of MVC for dogs is undetermined; however, the virus has been isolated from the feces of normal dogs[1] and of dogs with mild diarrhea[4] and from the small intestine and lungs of young pups with mild to fatal enteritis.[6–9] Experimental studies have shown that CPV-1 may cause mild to severe pneumonitis and enteritis in neonatal pups and embryo resorptions or fetal deaths in pregnant bitches infected between gestational days 25 and 35.[5,6] In this report, we describe a natural outbreak in Puglia (Italy) of fatal CPV-1 infection in 35-day-old pups with pulmonary and cardiac disease.

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