

Krystal Kidder

Signalment

Species Canine

Breed Doberman pinscher

Sex Male Castrate

Age 4 years

Presenting Complaint

- Acute onset of non-productive, dry cough, lethargy, exercise intolerance, and pawing at chest

History

- Current on vaccinations and heartworm preventative
- No history of past medical problems
- Referred to KSU-VHC by primary veterinarian for a cardiac evaluation

Physical Examination

- Temperature: 103.1°F
- Pulse: 200
- Heart rate: ~ 200, irregular, irregular heart beat
- Respirations: 32
- Good body condition score
- Temperament: Bright, Alert, Responsive

Diagnostic Plan

- Complete blood count and serum chemistry
 - Lymphopenia, hyperglycemia, hypoproteinemia, hypoalbuminemia, hyponatremia
- Serum Total T4: within normal reference range
- Systolic blood pressure: 100 mmHg
- Echocardiogram
- Thorax Cardiac Radiographs

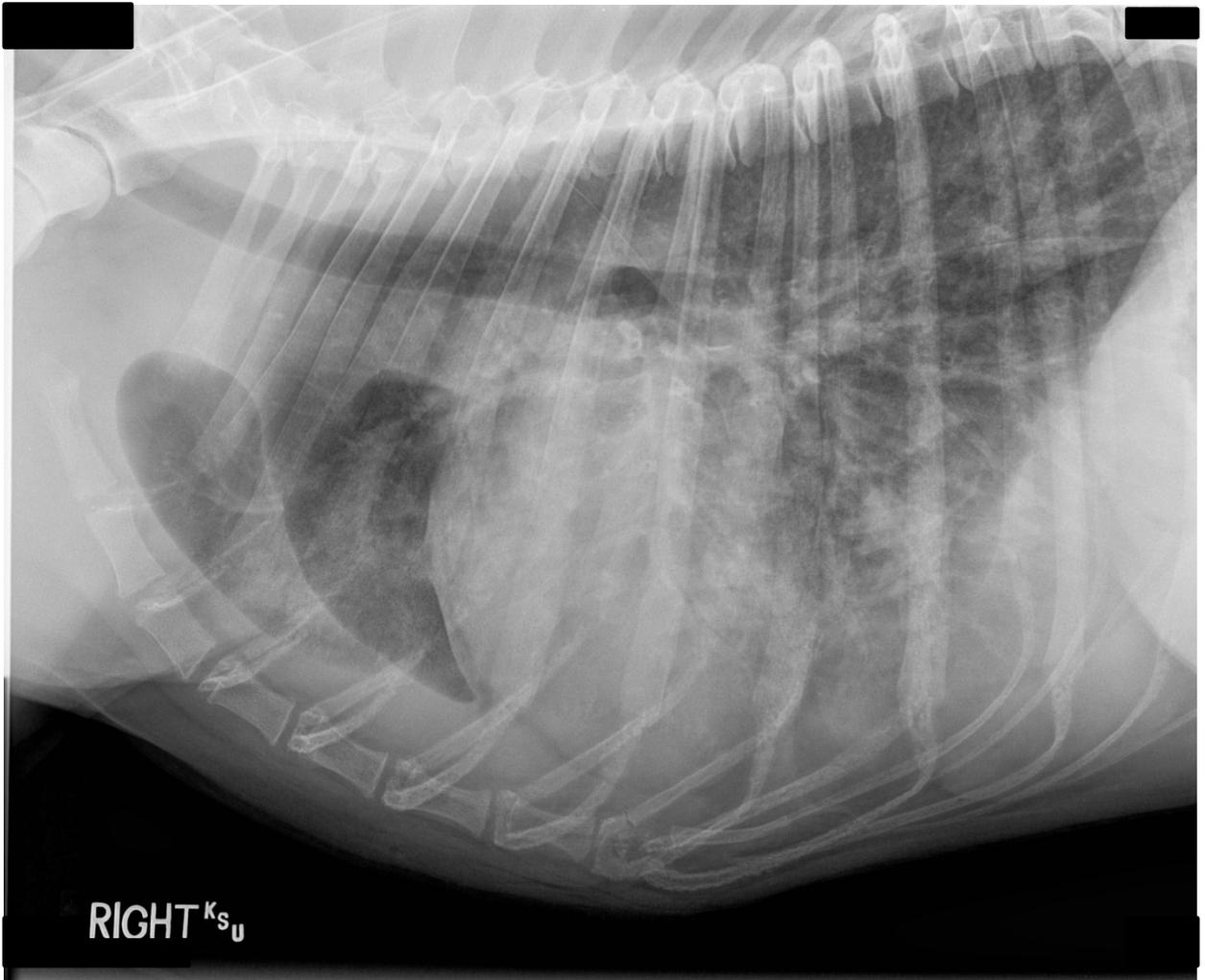


Figure 1. Right lateral view

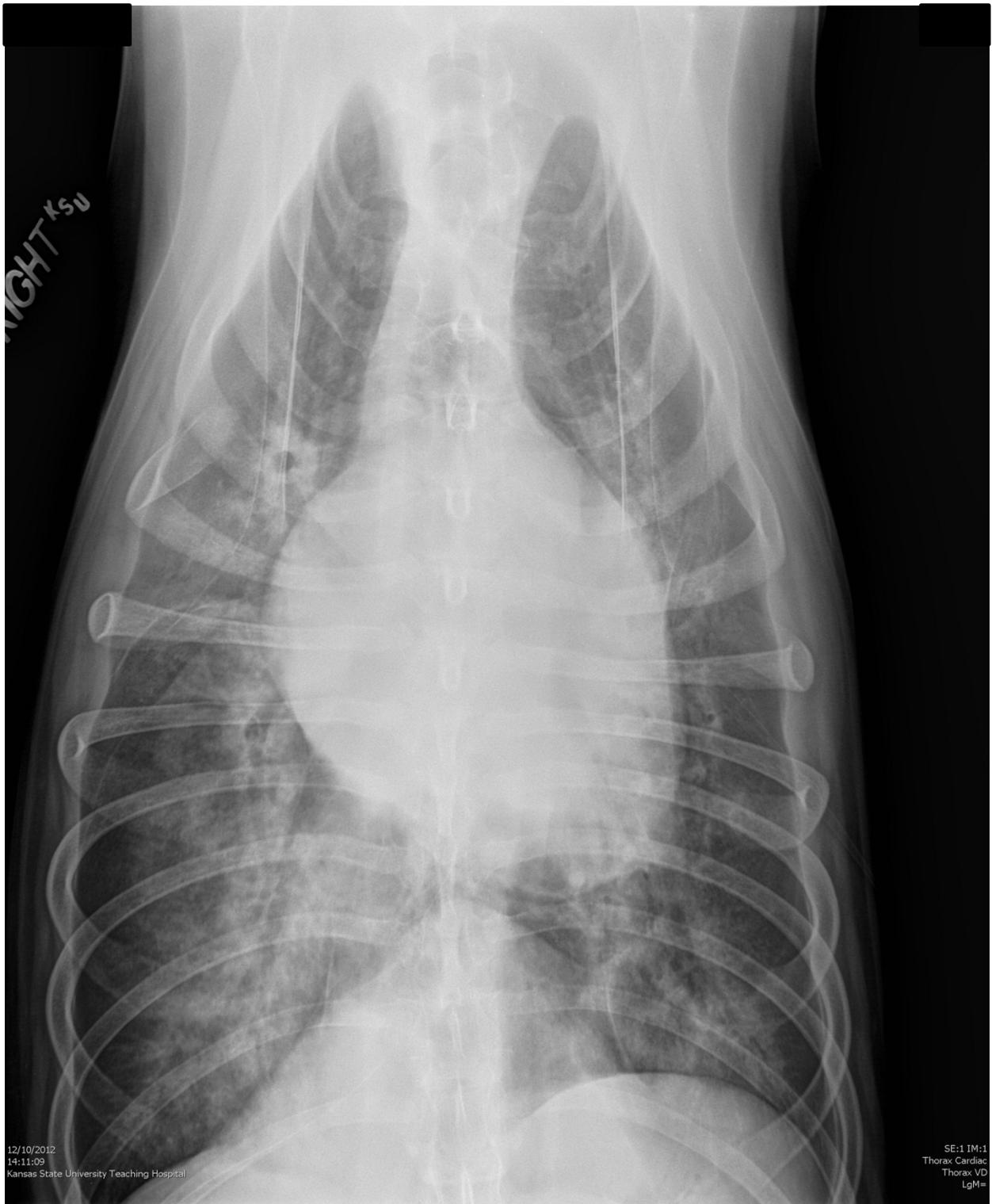


Figure 2. Ventrordorsal view



Figure 3. Dorsoventral view

Radiographic and Echocardiogram Interpretation:

Cardiac Thorax: There is generalized enlargement of the cardiac silhouette with a vertebral heart score of 11.25. Complete border effacement of the cardiac silhouette is present on the dorsoventral view, as well as, partial border effacement of the craniodorsal and caudal cardiac silhouette on the right lateral view. On the ventrodorsal view, the cranial mediastinum has increased soft tissue opacity with a more concave margin to the left than the right; normal in size and no shift present. The caudal lobar veins are enlarged on the dorsoventral view. There is a generalized increase in pulmonary opacity with perihilar and ventral patchy regions of an alveolar pattern and an unstructured interstitial pattern caudodorsally. An area of increased soft tissue/fluid opacity lies dorsal to the entire sternum on the right lateral view. There is a thin interlobar fissure line most prominent between the right cranial and right middle lung lobe at the level of the seventh rib on the ventrodorsal view.

Radiographic Impressions:

Generalized cardiomegaly, pulmonary edema, enlargement of pulmonary veins, and pleural effusion consistent with congestive heart failure. Primary differential dilated cardiomyopathy.

Echocardiogram:

Dilated cardiomyopathy, left ventricular end-diastolic dimension at upper reference, left ventricular end-systolic dimension far above upper reference, minimal mitral valve and tricuspid valve regurgitation, left ventricular percent fractional shortening (8.5%) worsened due to atrial fibrillation.

Case Follow-Up

- Medical management started with pimobendan, digoxin, diltiazem, furosemide, spironolactone, enalapril, as well as, a low salt diet and restricted exercise
- Further recheck examinations showed improvement of clinical signs of congestive heart failure
- Further diagnostics included
 - Renal panel: increased creatinine, hypophosphatemia, hyponatremia, hypochloremia
 - Digoxin level: 0.80 ng/mL (within reference range)
 - Post treatment thoracic radiographs showed improvement in pulmonary edema, resolving congestive heart failure, but heart still enlarged
- Patient was doing well at home

Post treatment radiographs

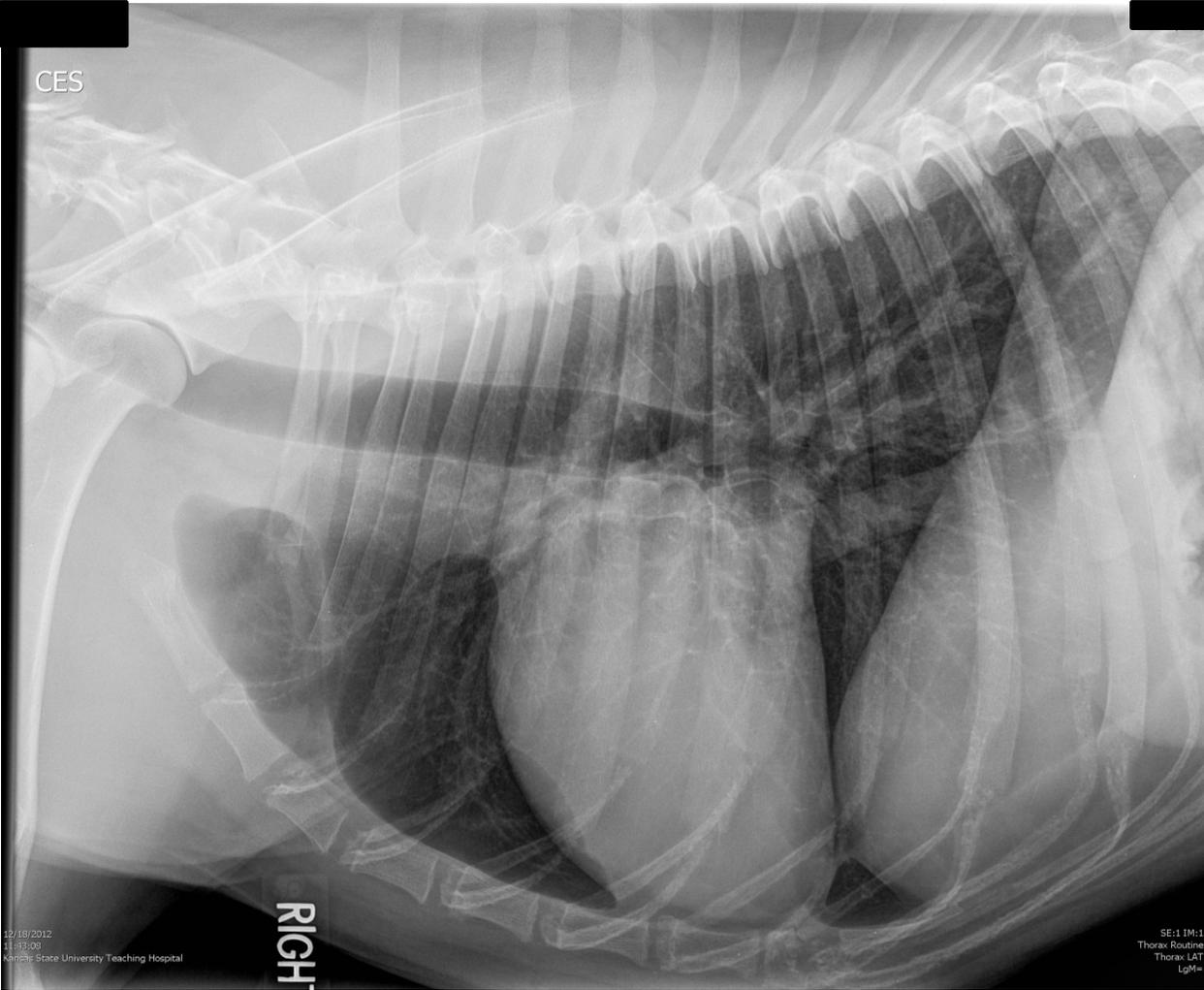


Figure 4. Right lateral view

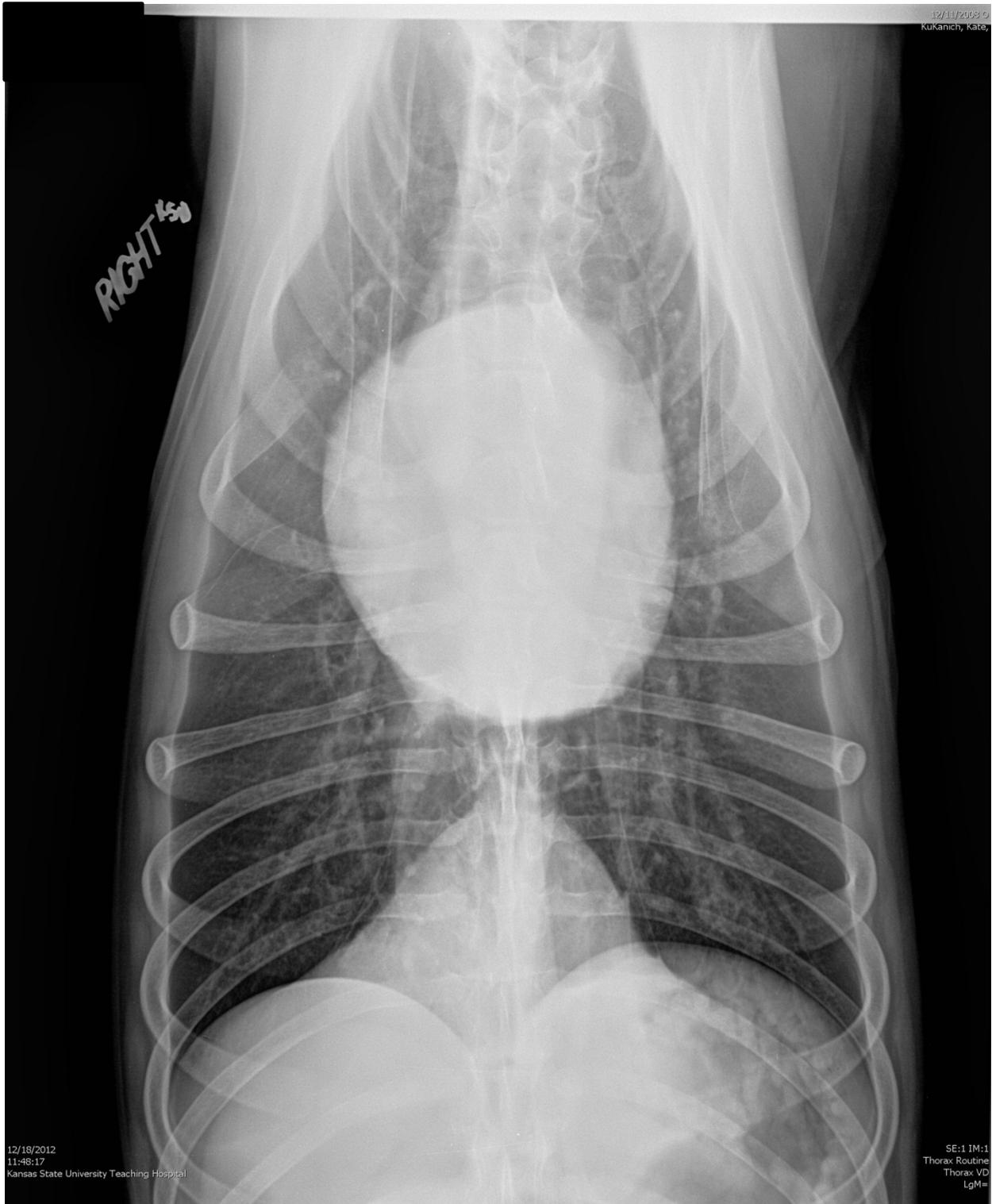


Figure 5. Ventrodorsal view

Post Treatment Radiographic Interpretation:

Cardiac Thorax: Compared to previous study dated 12/10/2012. The vertebral heart score is 10.5 (previously 11.25). There is visualization of the complete cardiac silhouette with minimal cranial cardiac waist and a straight dorsoventral caudal margin of the cardiac silhouette consistent with left atrial enlargement. There is resolution of the previously described interstitial and alveolar pulmonary opacity, cranial mediastinal, and retrosternal fluid opacity. There is persistent visualization of multiple thin pleural fissure lines. Pulmonary vasculature is unremarkable. On right lateral, there is a focal, triangular, irregular marginated, soft tissue opacity located dorsal to the second and third sternebra in the cranioventral portion of the cranial mediastinum.

Radiographic Impressions:

Left atrial enlargement most consistent with dilated cardiomyopathy. Resolution of pulmonary edema and pleural effusion. Appearance of cranial mediastinum may be due to mild sternal lymphadenopathy.

Final diagnosis: Dilated cardiomyopathy with secondary atrial fibrillation and improved congestive heart failure.

Pathophysiology of dilated cardiomyopathy:

Dilated cardiomyopathy is the most common cause of cardiac disability in middle-aged, large and giant purebred dogs; more common in males than females. This acquired heart disease is characterized by the progressive loss in cardiac contractility due to systolic dysfunction. Decrease in contractility leads to a decrease in cardiac output, systemic arterial blood pressure, stroke volume. This then leads to an increase in end-diastolic blood volume and pressure within the chambers causing the left atrium and ventricle or both ventricles to dilate. The body tries to compensate by increasing the sympathetic drive and renin-angiotensin-aldosterone system. Increasing the SNS and RAAS causes peripheral vasoconstriction and increase in myocardial contraction; increasing heart rate. Excessive stimulation of myocardium by the SNS stimulates ventricular arrhythmias and cardiomyocyte death. Also, excessive renin-angiotensin-aldosterone system activation causes vasoconstriction and retention of sodium and water, returning myocardial contractility to its previously reduced state. This is the prolonged subclinical phase, making prognosis poor to guarded. Clinical signs of DCM include: coughing, dyspnea, exercise intolerance, syncope, pleural effusion, ascites and sudden death. These signs can be secondary to severe ventricular arrhythmias, progressive systolic dysfunction, and left or right-sided congestive heart failure. Echocardiography is ideal for a definitive diagnosis of DCM.

Abnormal findings with the ECG are as follows: ventricle premature complexes, ventricular tachycardia, left atrial and ventricular enlargement and separation of valve leaflets (mitral insufficiency). Thoracic radiographs show mild to generalized cardiomegaly, pulmonary edema when heart failure is present and enlarged pulmonary veins. Blood work shows an increase in BUN, creatinine and alkaline phosphates with a decrease in sodium. Medical therapy is targeted to manage congestive heart failure and treat existing arrhythmias. Therapy will also aid in reducing the adverse effects of angiotensin II and other neurohormonal changes involved in the pathophysiology of DCM. Generally dogs with severe left-sided congestive heart failure have a worse prognosis than dogs with milder signs or signs of right-sided congestive heart failure at presentation. DCM is rare in cats which has a poor prognosis.