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ECG Quiz

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History

A nine year old intact male Miniature Pinscher weighing 4.8 kilogram was presented to veterinarian with signs of severe coughing and exercise intolerance. He was diagnosed of heart disease and previously described the angiotensin converting enzyme inhibitor and diuretic for a period of 6 months. Neither signs of syncope nor seizure was found. Physical examination revealed the dog was depressed and lethargy. The electrocardiography was performed and shown in Figure 1. The blood test showed normal complete blood count with normal kidney and liver blood profiles. The blood parasite was also negative. The thoracic radiograph revealed the whole heart enlargement (VHS = 12.5) with pulmonary vessels distention. The lung field was normal and no evidence of perihilar lung edema. Echocardiography was performed and results showed moderate insufficiency and thickening of both tricuspid and mitral valves. The right side heart was enlarged while the left atrium was dilated The left atrium to aortic root ratio (LA/Ao) was 1.79. The averaged mean pulmonary pressure was higher than 25 mmHg. The sand calculi were also found in the urinary bladder. The dog was treated with positive inotrope (pimobendan), angiotensin converting enzyme inhibitor and furosemide. The sildenafil was also prescribed. The bronchodilator was given in case of severe coughing due to bronchial stenosis. After, medication, the dog was more alert and was able to maintain normal activity at home. No sign of coughing or syncope was found. The blood test every 6 months showed normal concentrations of blood urea nitrogen, plasma creatinine as well as normal complete blood count. The electrocardiography at 1 month and 6 month thereafter showed the same pattern as on the first recording. Unfortunately, the dog died one and a half year later with unknown cause of acute kidney injury although previous blood test was still within normal limit 1 week before passing away.

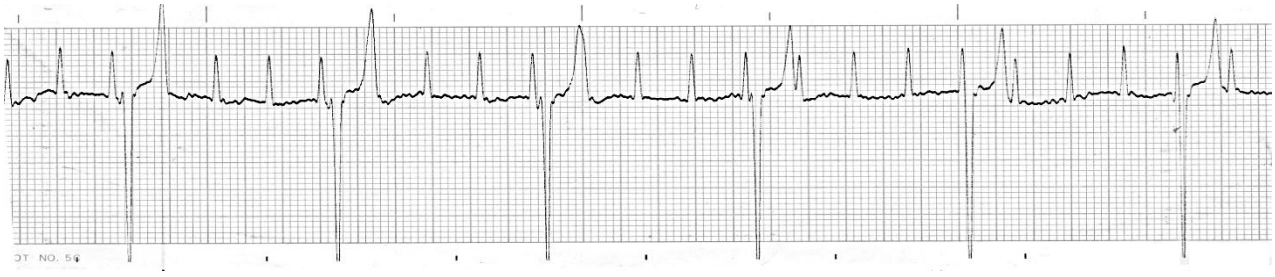

Figure 1 The ECG that was recorded in a Miniature Pinscher came with clinical sign of severe coughing.

Please answer before turning to the next page.

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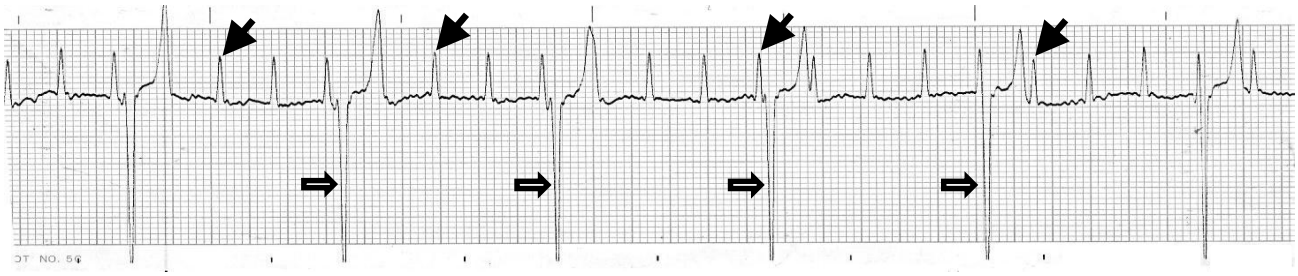
Interpretation

Figure 1 Complete Atrioventricular (AV) Block



In Figure 1, there are 2 types of complexes. The first one is the positive deflection (solid big arrows) and the second one was a big negative deflection (open small arrow). The rate of a small positive deflection is approximately 200 beats per minute while the large negative one is 55 beats per minute. The positive waves are the P-wave that originated from sinus node. The large bizarre negative complexes are the impulses that originated from the ventricular tissues with a lower rate. Neither normal QRS complex nor PR interval could be seen in this tracing which suggests that the propagating impulses originated from sinus node which spread throughout both atria cannot pass through AV node at any circumstance. Therefore, the complete AV dissociation or complete AV block was diagnosed.

The clinical signs in this dog were resemble the dog with chronic congestive heart failure because the myxomatous mitral valve disease and tricuspid regurgitation were found. The enlarged left atrium and right ventricle also support the mechanical heart problem.

In the present case, the ventricular rate was higher than 50 that can maintain sufficient blood supply to organs especially brain. Therefore, no seizure or syncope was seen. The cause of third degree AV block was unknown but the lesion such as fibrosis at the AV conducting tissue may be suspected. While repeated ECG for a year showed no change in electrocardiographic pattern suggests that the pathology of conduction pathway could not be corrected by medication. In this case, modification of cardiac autonomic nervous system may also not be helpful. The bronchodilator which preserves sympathomimetic action could not correct the blockage. However, the clinical signs became better after treatment with medication because of treating the congestive heart failure due to valvular problems. The phosphodiesterase-5 (PDE5) inhibitor, sildenafil, was also prescribed in this case to reduce the pulmonary hypertension since more cyclic GMP will be available for the blood vessels inside the lungs. If congestive heart failure is progress overtime, sympathetic activation may not be enough to maintain cardiac output with slow ventricular rate.