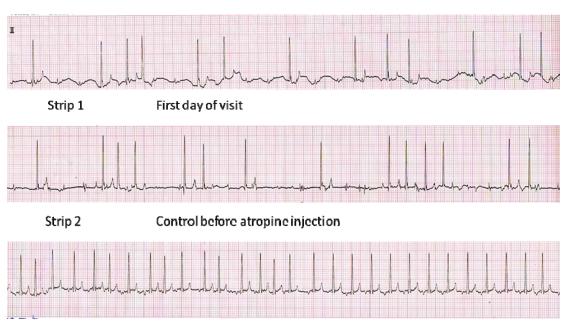
ECG Quiz

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Paper speed 25 mm/second



Strip 3 After atropine injection

These lead II ECG strips were recorded from an eleven years old, American Cocker Spaniel, weighing 10 kg that came to the Small Animal Hospital, Chulalongkorn University for health checkup after coughing for a month. From physical examination, he had soft systolic murmur grade II/VI. Dog had arrhythmia with the pulse deficit. Blood chemistries showed azotemia with BUN and creatinine concentrations of 56.1 and 1.9 mg/dl, respectively. The CBC was within the normal limit. The thoracic radiograph was performed and showed left heart enlargement with pulmonary edema. The echocardiography showed mild mitral regurgitation with FS of 40.61% and left ventricular inner diameter during diastole (LVIDd) was 37.2 mm. The LA/Ao was 1.5. The ECG was performed and the result of lead II was shown in strip 1. The systolic blood pressure was 138. A week later dog came back and ECG was repeated as shown in strip 2. By challenging with atropine at the dose of 0.04 mg/kg BW, ECG was recorded 15 minutes thereafter (tracing 3). The dog was sent home with angiotensin converting enzyme inhibitor and furosemide 1 mg/kg sid. Seven days after the medication was started, dog was presented at the hospital again with sign of muscular trembling and third eyelid protrusion for 1 hour but they disappeared when dog was presented at the hospital. No signs of exercise intolerance or respiratory distress were found.

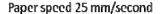
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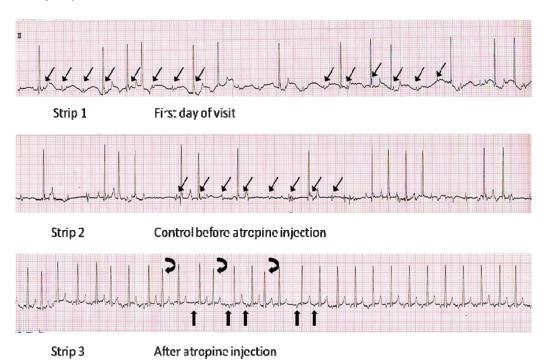
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Interpretation

Tracing 1, 2- Third degree atrio-ventricular block with supraventricular ectopic beats Tracing 3 - Supraventricular ectopic rhythm with multiple foci





Strip 1 and 2 showed the multiples P-waves (small arrows) in which some were non-conducted Pwaves (not followed by QRS complexes). Some Pwaves were closely couple to the QRS complexes. In both strip 1 and 2, the atrial rate was approximately 115 beats per minute and consistent with sinus arrhythmia. Thus the origin of P-waves was from sinus. The ventricular rate was 70 beats per minute. The PR interval could not be identified in this case and there was no association between the atrial and ventricular conduction. Therefore, complete AV dissociation was diagnosed. The ventricular escape beats, rather than wide and bizarre-shaped, looked similar to the normal QRS complexes suggesting the impulses may be originated from Purkinje cells in the Bundle of His. The narrow complex (complex 4 in strip 1) would be the one in which P-wave did not superimposition and it was emerged prematurely with impulse originating from somewhere high in the ventricular conducting system. The unassociation between atrial and ventricular rate without measurable PR interval is the landmark of third degree atrio-ventricular block. Strip 2, although had some noise, showed the rate of P-waves similar to strip 1. In strip 3, the heart rate was accelerated due to parasympatholytic action of atropine. The rate was increased up to 150 beats per minute. The ratio of atrial and ventricular rate was 1:1 with a constant PR interval suggesting both P and QRS waves originated from the same ectopic origin. It was noticed that the P-waves deflection in strip 3 appeared to be negative (straight big arrows). Therefore, the junctional tachycardia or supraventricular tachycardia may be possible. The premature complexes may be originated from multiple sites since other complexes with different shape occurred prematurely (curve arrows). The atrio-ventricular block in this case may be caused by other diseases rather than the organic disease of the heart. Overstimulation of vagus nerves was suspected due to heart rate acceleration after atropine administration. The clinical signs related to heart problem, although did not exist, should be monitored. No specific antiarrhythmic drug was given at this time. However, the underlying disease associated with vagal overactivity should be concerned.