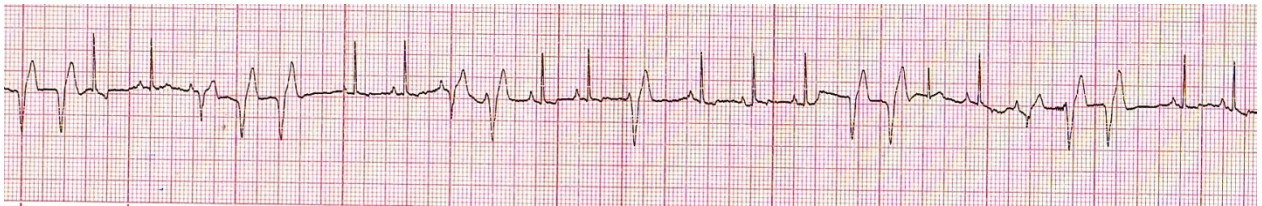


## ECG Quiz

Chollada Buranakarl<sup>1\*</sup> Winai Chansaisakorn<sup>2</sup>



Tracing I - 25 mm/sec



Tracing II - 25 mm/sec



Tracing III - 25 mm/sec



Tracing IV - 50 mm/sec

### History

A nine years old male Golden retriever weighing 32.5 kg came to the animal hospital, Chulalongkorn University with a history of walking difficulty and lameness for the last 3 months. After the weakness of both hind limbs started, it then progressed to the fore limbs. He also had difficulty in controlling urination. By physical examination, the dog had normal heart sound with arrhythmia. The tetraparesis was detected with muscular atrophy especially at the hamstring. The complete blood count, liver enzyme (ALT) and plasma creatinine concentration were normal. Wobbler syndrome with osteoarthritis was diagnosed. Thoracic radiograph

showed normal heart size and shape and mild diffused interstitial and bronchial pattern of lung.

The electrocardiography was performed as seen in the strip I and II. Echocardiography showed mild mitral valve regurgitation and mild systolic dysfunction. Blood pressure was measured with a systolic of 128 and diastolic of 86 mmHg using oscillometric technique. Dog was on doxycycline and prednisolone and came back 2 weeks later with clinical improvement and walked by himself. The ECG was repeated 2 and 4 weeks later and shown in tracing III and IV, respectively.

Please answer before turning to the next page.

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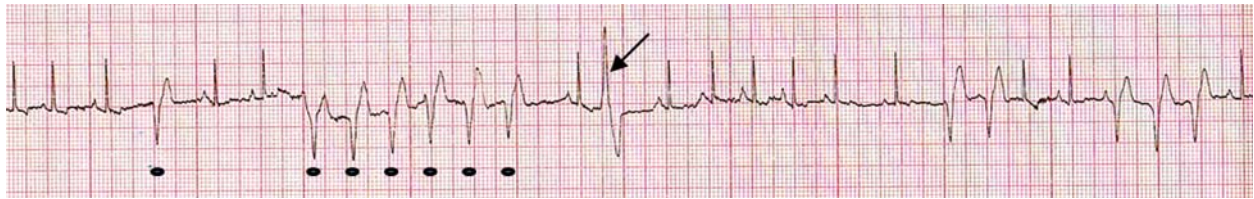
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### Interpretation

Tracing I, II- Accelerated ventricular rhythm with premature ventricular complex

Tracing III - Respiratory sinus arrhythmia

Tracing IV - Respiratory sinus arrhythmia



Tracing I



Tracing II

Accelerated ventricular rhythm is commonly seen in dogs with or without primary heart disease. It may be seen in dogs with trauma, gastric torsion, splenic tumor, etc. The heart rate in tracing I was approximately 140 beats/minute. The complexes with ventricular in origin are not a paroxysmal tachycardia although the transition from sinus to ventricular and back occurs abruptly but this term denotes an abrupt change in heart rate. There was a slowing of the sinus rhythm which may be due to respiratory sinus arrhythmia below the discharge rate of the VPCs, thereby removing the overdrive suppression by the faster sinus beats and allowing the ectopic focus to reach the threshold potential and initiate a ventricular response (solid dots). When the sinus rhythm accelerates and exceeds the rate of ventricular rhythm,

it again overdrives the ventricle and prevents the ectopic focus from reaching its threshold potential. The fusion beats (opened arrows) were seen in tracing II with a typical in which the P-wave have a shorter PR interval and the morphology of fusion beat is intermediate between the sinus and ectopic beats. In tracing I, one different VPC was noticed (arrow) which had bizarre shape and emerged very early. Therefore, multiple sites of ventricular ectopic foci were found. The ECG recorded at 2 and 4 weeks later showed normal sinus arrhythmia. These findings confirm that the electrical instability of the heart was not related to the organic disease of the heart but may be rather due to other factor including the disease associated with pain.