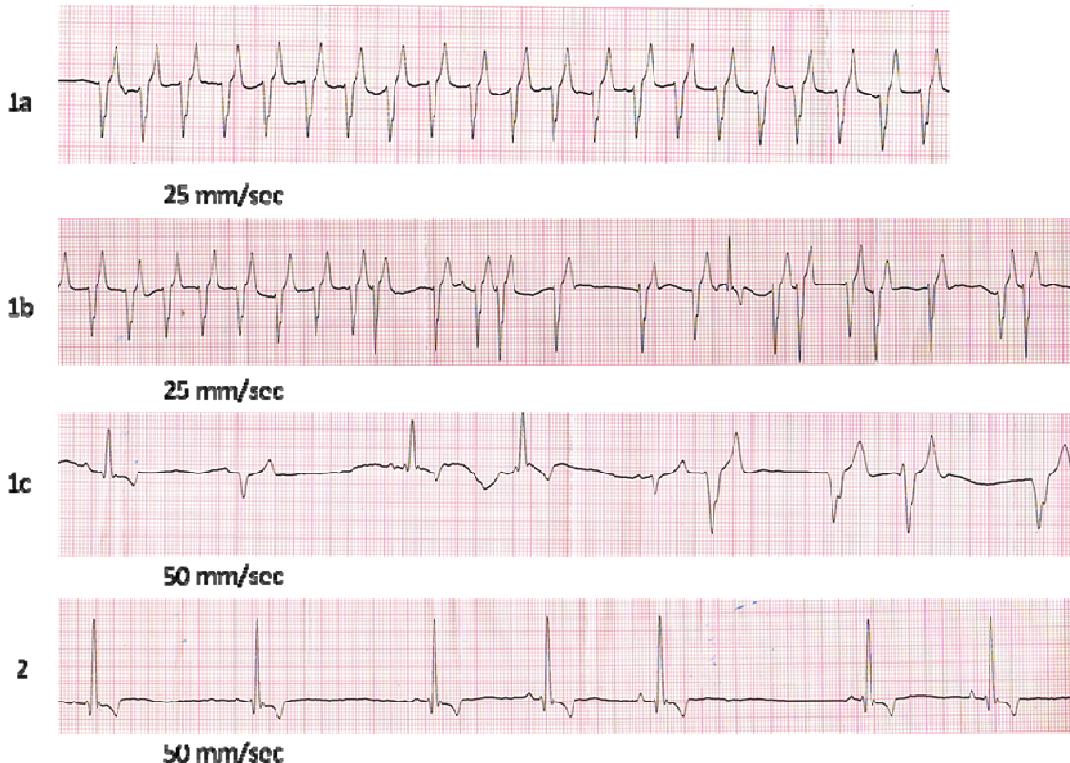


ECG Quiz

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History

A 5 years old male intact golden retriever weighing 28 kg was referred to the Small Animal Teaching Hospital, Chulalongkorn University for blood transfusion. Dog was diagnosed of ehrlichiosis infection and was azotemia with concentrations of plasma creatinine and blood urea nitrogen were 2.6 and 130 mg/dl, respectively. The packed cell volume was 7.9% while the white blood cell count was 16,600 cells/mm³. The concentrations of plasma total protein and blood glucose were 5.2 g/dl and 110 mg/dl, respectively. The blood gas results, plasma Na⁺ and plasma K⁺ were in the normal reference range. Urinalysis showed proteinuria with urinary protein creatinine ratio of 13.5. Blood was transfused to the dog and the PCV increased up to 25.8%. Twenty four hours later, the dog was oliguric and showed sign of

panting. The thoracic and abdominal radiographs were performed and the result of cardiomegaly with pleural effusion was presenting.

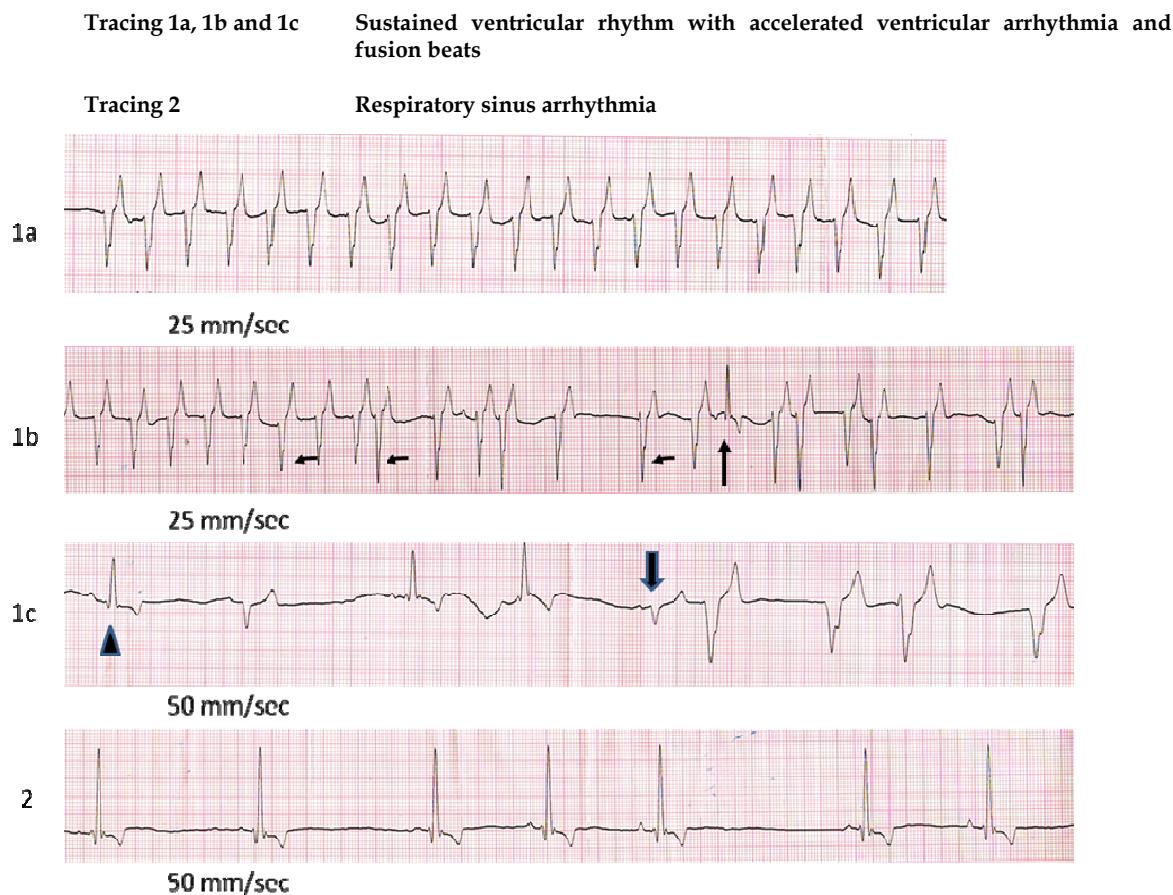
The ECG was recorded as shown in 3 consecutive runs (tracing 1a-c). The echocardiography showed mild right side heart enlargement without valvular disease. The fractional shortening was 47.4% and EPSS was 3.2 mm. The dog received doxycycline for treatment of blood parasite and some other medications (furosemide, ramipril, fish oil and vitamin E). The dog came back one week later for monitoring without any abnormal clinical symptoms. Blood pressure was measured with systolic and diastolic of 132/77. Recording of ECG was repeated as shown in tracing 2.

Please answer before turning to the next page.

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Interpretation



The tracing 1a, 1b and 1c were run consecutively on the first day of ECG recording after thoracic radiograph was taken. In tracing 1a, the ECG waveforms were regular in rhythm with bizarre shape of ventricular origin. The ventricular rate was approximately 150 beats per minute and regular. The P waves mostly were concealed and not readily emerged among the ventricular complexes. The ventricular ectopic rhythm ended followed by the slower rate in tracing 1b (120 beats/minute), thus, paroxysmal tachycardia may be existed. In tracing 1b, the different shapes of VPCs were noticed (curves arrows). There was one complex which looked similar to the QRS complex originated from supraventricular locus (straight arrow). However, the P-wave of this complex had negative deflection which may suggest that the origin may be from junction. The emergence of a supraventricular beat closely following the VPC suggesting the existent of accelerated VPCs. The ventricular complexes with different shapes may be due to the ectopic foci occurred from different locations in the ventricle. The presence of long pause in tracing 1b without sinus complex indicates that the rate of sinus was dramatically slow and may not be

competed with the ventricular rate. In tracing 1c, the 3 sinus impulses were appeared (triangle). When sinus rate slows down below the discharge rate of VPCs, thereby removing the overdrive suppression by the faster sinus beats and allowing the ectopic focus to reach threshold potential and initiate ventricular response. When the sinus rhythm accelerates and exceeds the rate of the accelerated ventricular rhythm, it again overdrives the ventricle and prevents the ectopic focus from reaching the threshold potential. There are several fusion beats throughout this recording which were clearly presented as two small complexes (big arrow). Typical of fusion beats, the P-waves have a shorter PR-interval and the morphology of the fusion beat is intermediate between the sinus and ectopic beats. In tracing 2, the ECG showed the respiratory sinus arrhythmia with the rate of approximately 70 beats/minute. No other abnormalities were found. In this case, ventricular arrhythmia with fusion beats found in the first recording may be other disease rather than the primary heart disease. The most likely abnormality is severe anemia which was resolved by blood transfusion along with normal ECG waveform.