ECG Quiz

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A 14.5-year-old neutered female Bichon Frise dog weighting 3.3 kilograms with a chief complaint of sudden syncope was presented to the Emergency Unit of the Small Animal Teaching Hospital, Chulalongkorn University. From the history taking, the dog was diagnosed with the chronic kidney disease (CKD) stage 3 according to the International Renal Interest Society (IRIS) staging system over a past year. After CKD was diagnosed, the dog had regular visits to nephrology unit for treatment and monitoring. The dog periodically showed signs of uremia, anemia, anorexia, hypertension (approximately 190 mmHg of systolic blood pressure) and depression. The ultrasonography revealed bilateral chronic nephropathy and mild hepatopathy with bile retention. The vertebral heart size was within normal limit and no murmur heart sound was detected. Before syncope episode, the owner reported that the dog was weakness without anorexia, syncope or coughing. On the day of syncope episode, the dog was still responsive to the environment. The physical examination showed normal hydration status. The murmur heart sound with heart rate (HR) of 116 beats per min (bpm) and strong femoral pulse were noticed. The pale pink mucous membrane was found with systolic blood pressure of 120 mmHg. The blood works indicated moderate anemia (packed cell volume; PCV = 17%), leukocytosis with neutrophilia (total white count of 21,520 cell/µl), and hyperglycemia (blood glucose 163 mg/dl). Few hours later, the dog underwent cardiopulmonary resuscitation. The treatments were including intravenous fluid administration with bicarbonate, furosemide, dexamethasone, and Amoxicillin-clavulanic acid. The endotracheal intubation was also performed for oxygen supplementation. Arterial blood gas analysis post resuscitation indicated metabolic acidosis (pH 7.335), while potassium was 4.53 mmol/L. Eight hours later, the dog had bradycardia (HR 47 bpm) and hypotension (systolic blood pressure 81 mmHg). The oliguria (urine output 0.8 ml/kg/hr was also developed. Atropine and adrenaline infusion were initiated and HR was increased up to 78 bpm. However, 1 hr later, the severe bradycardia (HR 20 bpm) was encountered without response to repeated dose of atropine and dopamine infusion. The severe bradycardia was still persistence with severe metabolic acidosis (pH 6.927) and hyperkalemia (6.37 mmol/L) even after dobutamine administration. Unfortunately, the dog died a few hours later.

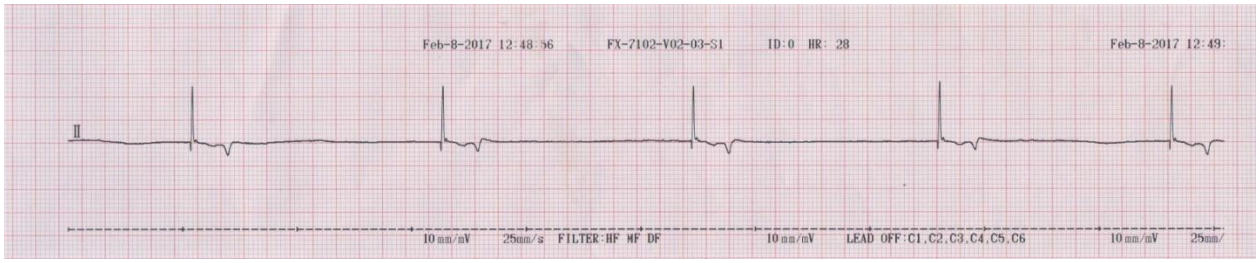

Figure 1 ECG tracing recorded in emergency room after admission

Please answer before turning to the next page.

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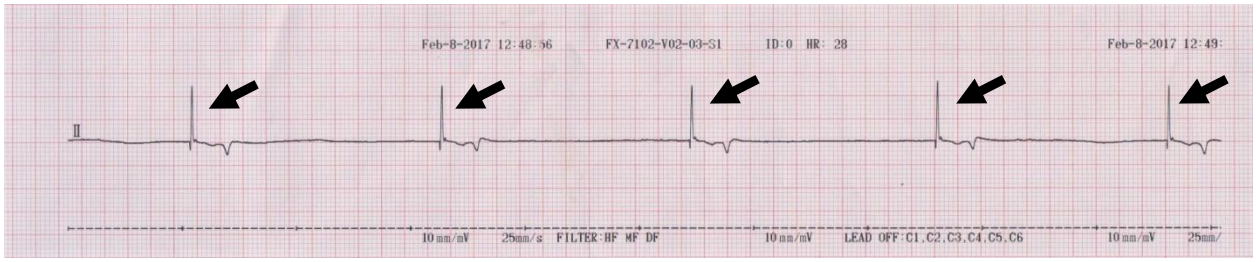
Interpretation

Sinus arrest with slow junctional rhythm



The ECG was recorded while the dog was unconcious in the emergency unit of the hospital. The heart rate is very low approximately 30 bpm. Please notice normal QRS complexes (arrows) which suggest that the impuses could propagate and travelling along the Bundle of His throughout the ventricles. However, the absence of preceding p-wave on every beats along with the slow heart rate implied that the sinus node does not discharge impulse or sino atrial (SA) arrest was encountered. The pacemaker location may be at the atrioventricular (AV) node or at the junction with an abnormally low rate. The rate was so low that the cardiac output may be compromised with hypotension. The prolonged QT interval (0.36 sec) may be due to the slow heart rate.

Another consideration that have to be clarified is whether the hyperkalemia was presence in this dog as found in hyperadrenocortism or in dog with renal failure. However, plasma potassium and sodium were within normal limit prior to bradycardia. Therefore, atrial standstill with absence of p-wave caused by severe hyperkalemia was unlikely.

Please notice that other escape beats especially ventricular escape beats could not be seen. Since the dog clinical signs was normal 2 days before presenting in the hospital and no other cardiac signs were previously noticed, thus, the cause of severe bradycardia may be due to other factors such as the effects of toxin or other chemical toxicity that suppressed both SA and AV nodes.

The overactivity of vagus may be one factor responsible of bradycardia. Giving first dose of atropine showed the improvement of heart rate which was escalated from 47 to 78. However, the heart rate was maintained only for 1 hr and the bradycardia with the rate of 20-30 bpm recurred. The second dose of atropine could not reverse the bradycardia. The result suggests that parasympathetic alone may not be solely responsible for this bradycardia. The dopamine was given by continuous infusion to maintain blood pressure and the dobutamine was also administrated to increase heart rate. However, the heart rate was remained very low without p-wave. The dog did not gain conciousness and died 4 hr later with low output and cardiogenic shock.