ECG Quiz

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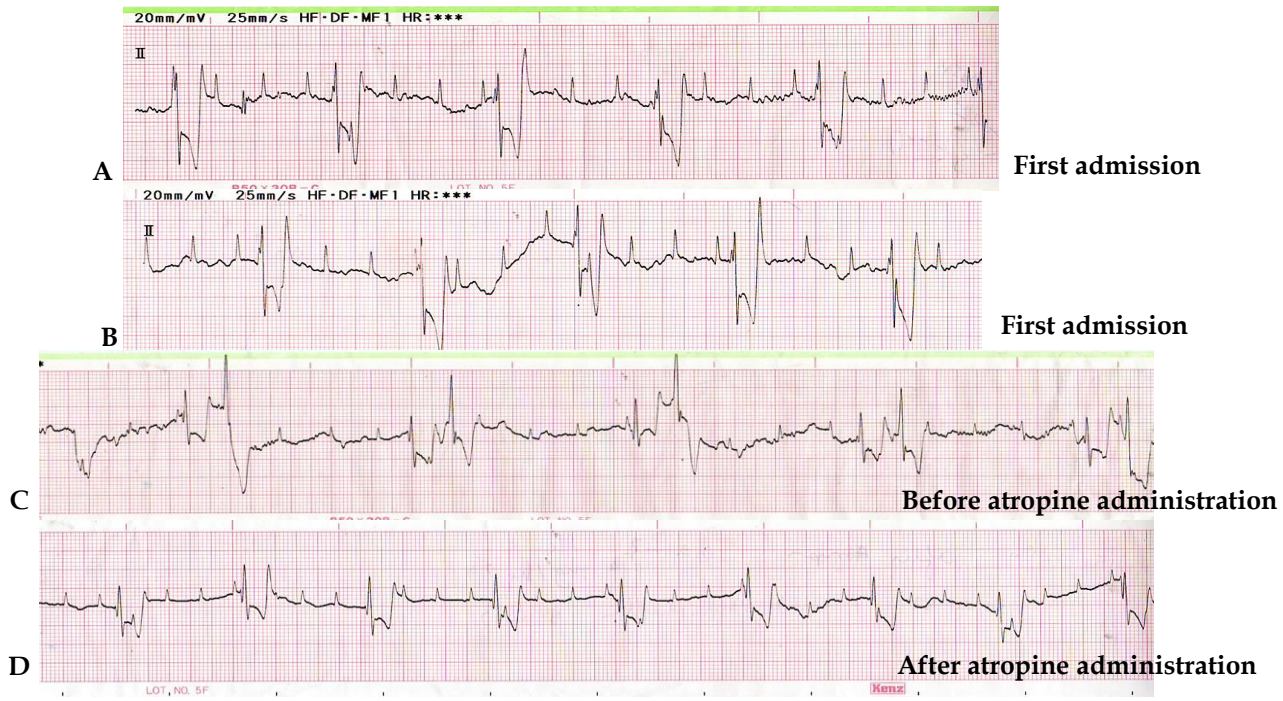

Figure 1 The ECG waveforms during admission (A and B), before atropine (C) and after atropine administration (D)

A five years old male Chihuahua weighing 4.8 kilograms was presented to a private hospital with symptoms of lethargy and exercise intolerance. The dog had periodic syncope and frequency of syncope was as high as once a day. During syncope, the dog was lied down and the owner tried to do the cardiac massage. The syncope lasted for approximately 30 seconds until the dog gained consciousness. From physical examination, the dog had low heart rate of approximately 40 beats/minute without murmur. The blood test was performed with PCV of 36% and high ALT (220 IU/L). The kidney function was within normal limit. The ECG was performed during the first admission which was shown in Figure 1A and 1B. Radiograph showed left ventricular enlargement with mild pulmonary distention. The lung and trachea were normal. Hepatomegaly was also noticed. The echocardiogram was performed and revealed mild left atrial enlargement. The LA/Ao was 1.4:1. Both mitral and tricuspid valves were normal. The atrial

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was contracted approximately 3 times more frequent than ventricle. Left ventricle was severely dilated. The thickening of interventricular septum and left ventricular free wall was also found. Fractional shortening was as low as 19%. The right atrium and ventricle were mildly enlarged with pulmonary distention. Aminophylline and positive inotrope, pimobendan were prescribed to this dog. The syncope was still appeared 2 times in a week. The dog came back one week after medication for remeasurement of ECG and performing atropine challenge test. The ECG before and 15 minnutes after 0.4 mg/kg of atropine given subcutaneously were shown in Figure 1C and 1D, respectively. The 24 hour holter measurement was performed the next day as shown in Figure 2 and 3. The dog was planned for cardiac pacemaker implantation which would be performed 5 days after holter measurement. Unfortunately, one day prior to implantation, the dog was excited while fighting with another dog at home and collapse to death.

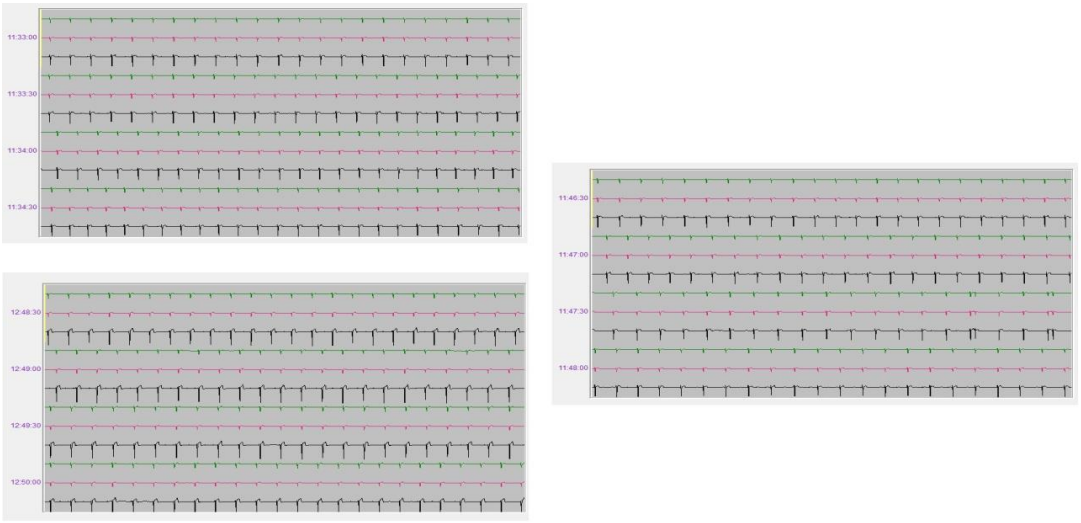

Figure 2 Continuous 24 hr ECG recording using holter device

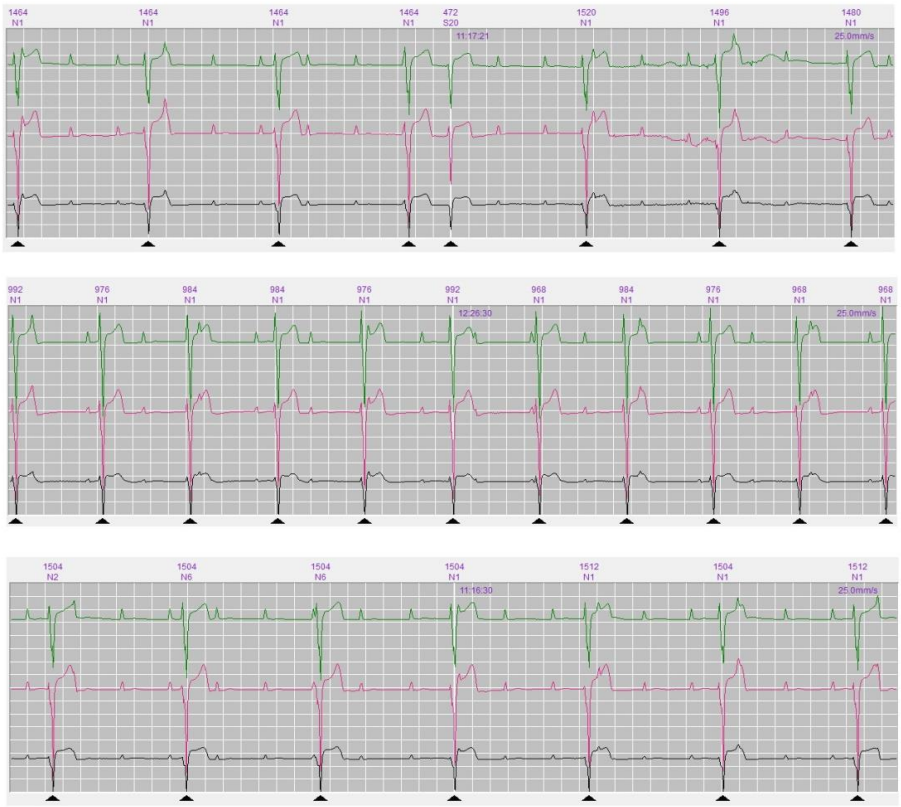

Figure 3 Magnification of the ECG waveforms from holter device

Please answer before turning to the next page.

Interpretation

Complete atrioventricular dissociation

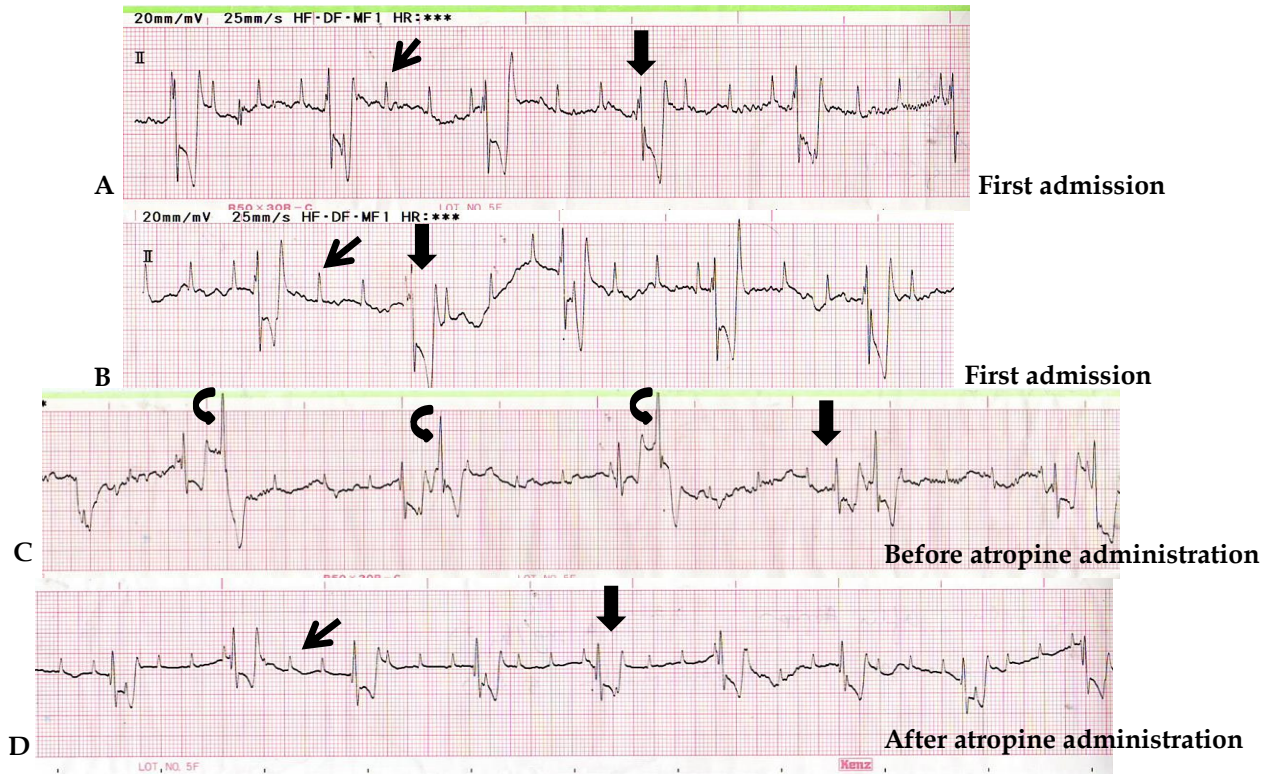

Figure 1

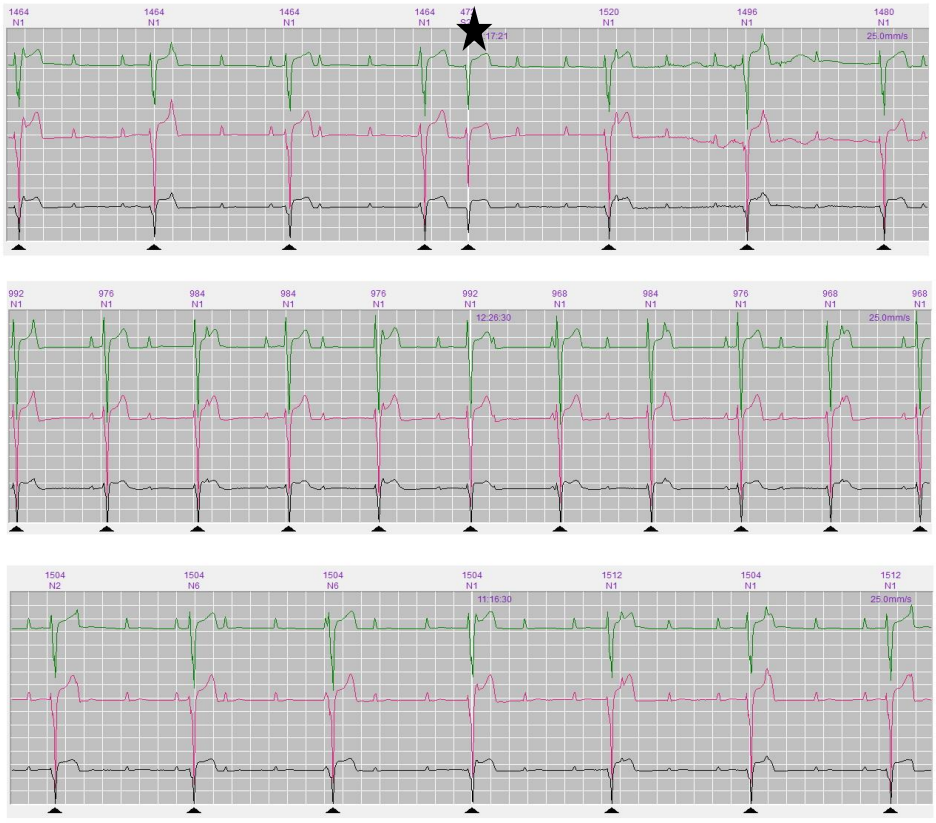

Figure 3

Tracing A and B showed the ECG wave formed with aberrant ventricular conduction (Big arrows). The rate of ventricular contraction was approximately 40 beats per minute. The ventricular impulse was abnormal which implied that the pacemaker was originated within the ventricle and/or atrioventricular node. The P waves (small straight arrows) had regular rate of 150 beats per minute in tracing A and B. The atrial contractions were corresponding to the impulses travelling from the SA node. The enlarged ventricular chamber in this case was due to the limitation of ventricular ejection causing an increase in ventricular preload. The ECG in tracing C after medicated with aminophylline and pimobendan showed the same results. The extrasystole from other ectopic beats were presented (curve arrows) although they had no effect on hemodynamic.

The atropine was challenged as shown in tracing D and the results showed higher atrial and ventricular rate of 200 and 50 beats per minute, respectively. The cardiac output was insufficient since the syncope was still persisted. The non-vagal mediated 3rd degree atrioventricular block suggested pathology of the node. Ectopic ventricular contraction cannot maintain the sufficient output.

The continuous ECG recording was shown in figure 2. The bizarre shape of QS wave with negative deflection confirmed the ectopic pacemaker was ventricular in origin. The ectopic ventricular complex from another ventricular site was also noted (star). The normal P-QRS-T complex was unseen. This situation confirmed that impulses from SA node was completely blocked at the level of AV node. Since the cardiac output was detriment, the pacemaker implantation should be put in immediately in this case. Unfortunately, temptation to put cardiac pacemaker was failed since the dog was sudden cardiac death due to limitation in heart rate acceleration when fighting.