ECG Quiz

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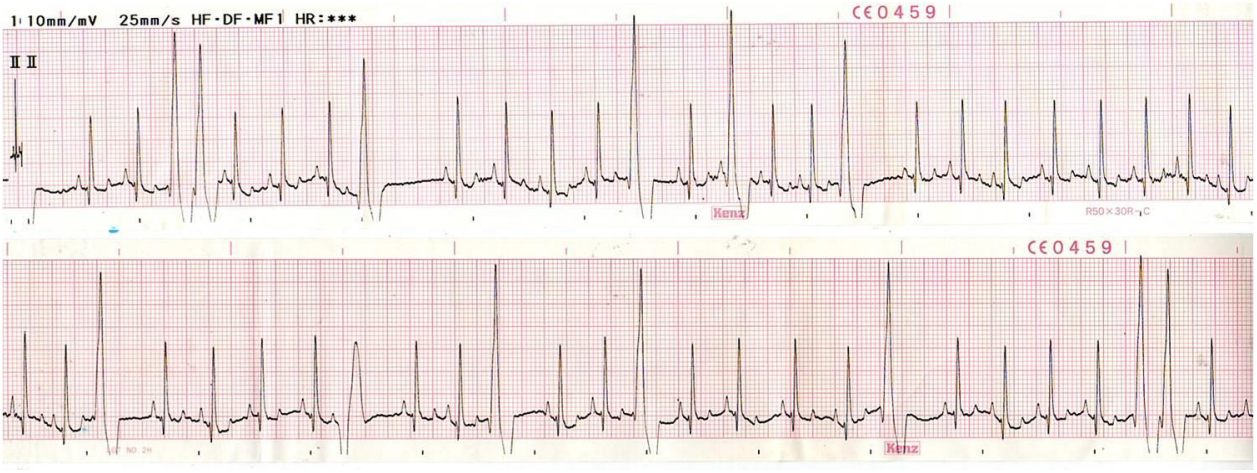

Figure 1 Surface ECG in a Boxer with sign of periodic syncope

A seven-year old spayed female Boxer weighing 29.6 kg was presented at the Small Animal Teaching Hospital, Chulalongkorn University with a chief complaint of periodic syncopes for the last few weeks. Dog also had chronic cystitis that had been treated with quinolone antibiotic. The owner noticed the enlarged left popliteal lymph node and brought the dog to the oncology clinic where the fine-needle biopsy was performed and revealed the tumor as epithelial cell in origin. Blood collection showed normal red blood cell and white blood cell counts with normal differential numbers of white blood cell types. The liver enzymes (alanine transaminase; ALT and alkaline phosphatase; ALP), kidney function parameters (blood urea nitrogen; BUN and plasma creatinine concentrations), plasma concentrations of protein and electrolytes including blood gas were within normal limits.

The electrocardiogram was performed and showed in Figure 1. Thoracic radiograph revealed mild left ventricular enlargement with vertebral heart score (VHS) = 11.4. There was moderate diffused interstitial pattern of lung. The spondylosis was found at T5-T6 and T9-L1. Echocardiography was performed and dog had slightly left ventricular dilation with thin wall during systole. The fractional shortening was 19%. The atrio-ventricular valves were normal without regurgitation with normal outflow tract. While she was in the hospital, the multiple syncopes were found. The continuous ECG recording using holter was performed and results were shown in Figures 2 and 3. One and a half hour after recording, the dog lost consciousness again and passed away with cardiac sudden death although cardiopulmonary resuscitation was performed.

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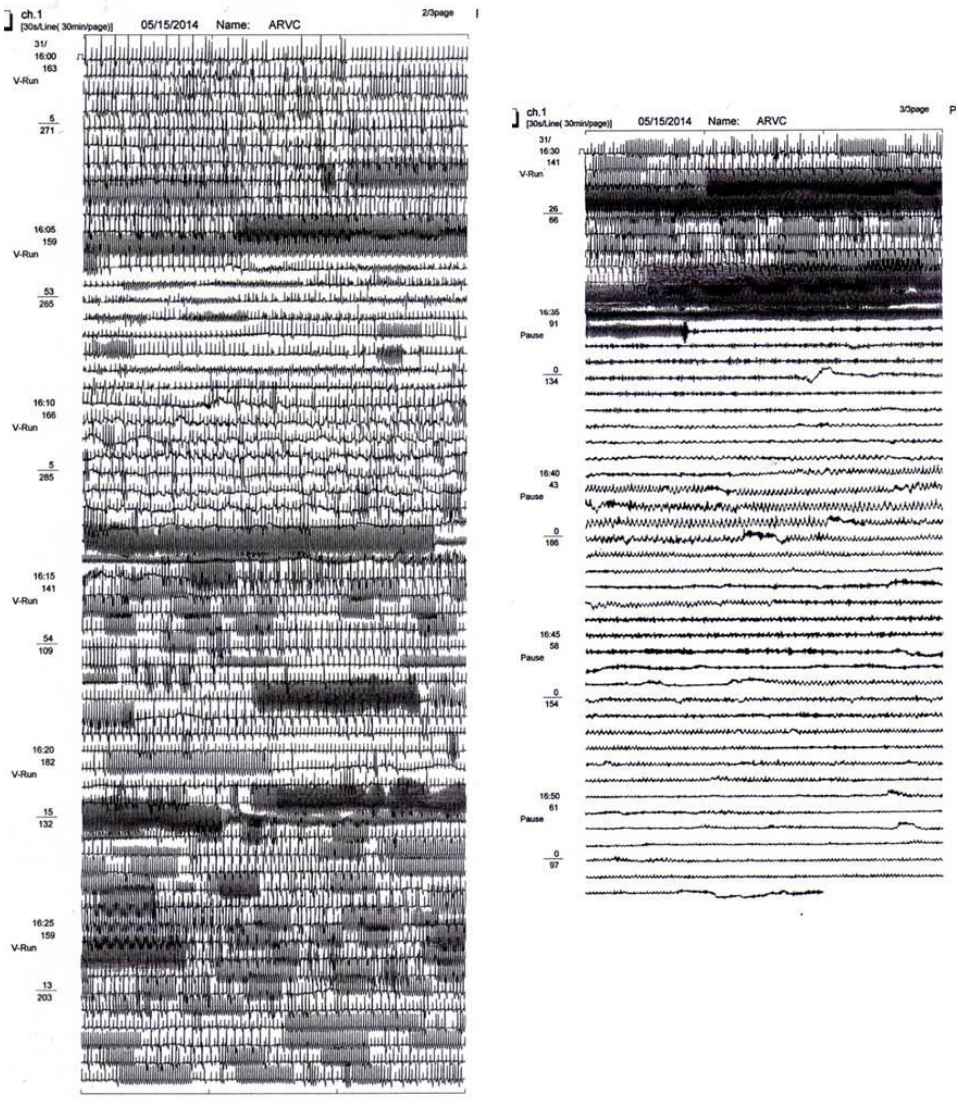

Figure 2 Holter recording 1 hour before death

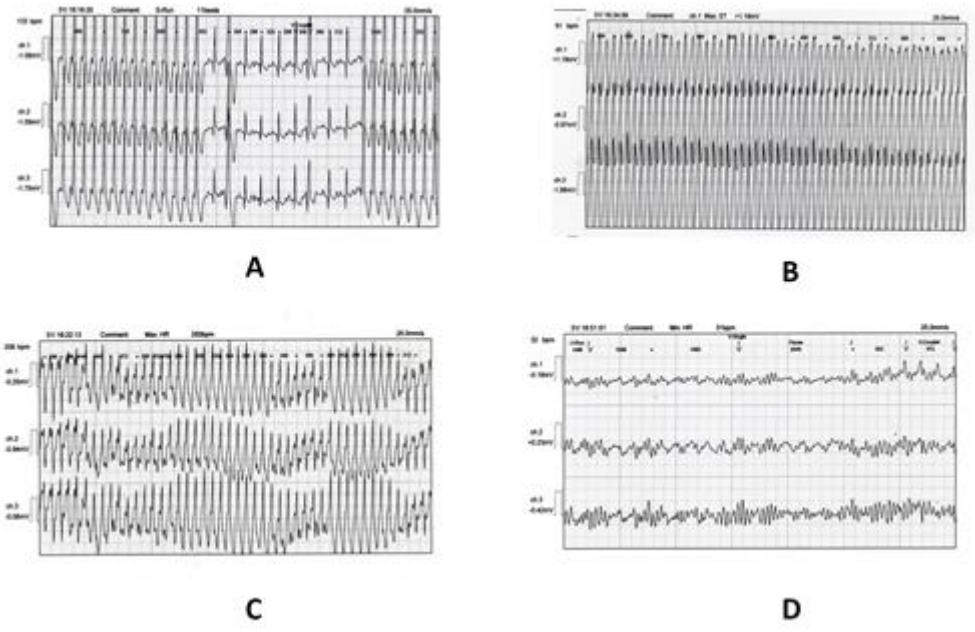

Figure 3 Examples of ECG from holter device

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Interpretation

Figure 1 Sinus arrhythmia with frequent ventricular premature complexes
Figure 2 ECG recording showed runs of ventricular tachycardia and Torsades de points
Figure 3 A Intermittent ventricular tachycardia
B The R on T ventricular tachycardia
C Ventricular arrhythmia with Torsades de points (TdP)
D Torsades de points with ventricular flutter

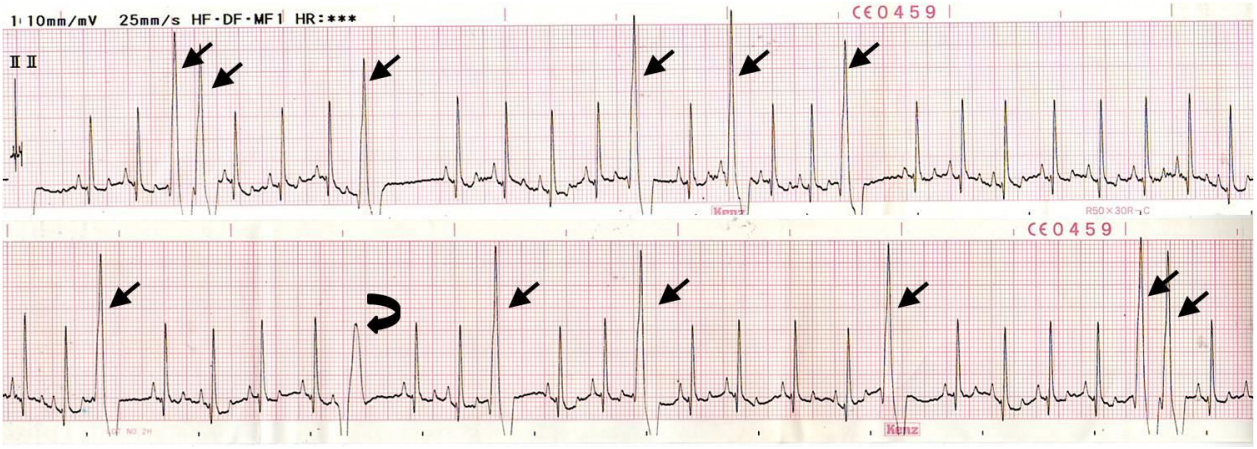

Figure 1

The heart rate was approximately 140 beats per minute. Most of the complexes were originated from the sinus node. However, the ventricular premature complexes (both straight and curve arrows) were found with high emerging rate. The ectopic rate was 30-35 beats per minute which was higher than >20% of normal beats. The VPCs had varying shapes as shown in straight and curve arrows which implied that the origin of impulses were different. Since the ectopic foci emerged from ventricle and did not travel through the specialized conducting system but rather through ordinary muscle with delay resulting in bizarre widened QRS complexes. Moreover, some VPCs were coupled with the next impulse (the beginning and the end of the strip) that can interrupt the ventricular filling during diastole. Some breeds have a high occurrence rate of ventricular arrhythmia such as German Shepherd and Boxer. The diagnosis of Boxer cardiomyopathy was more likely. Some call the disease as "Boxer arrhythmogenic right ventricular cardiomyopathy" which is characterized by the development of ventricular tachyarrhythmia resulting in syncope and sudden cardiac death. However, congestive heart failure is uncommon manifestations of the disease. The VPCs in lead II in this disease typically have an upright morphology (left bundle branch morphology) since the impulses arise in the right ventricle. It is a genetic disease inherited in an autosomal dominant pattern. The disease is an adult onset which is characterized into many forms such as the concealed form in which the dog is asymptomatic with VPCs, overt form in which the dog showed sign of fatigue with presences of ventricular tachyarrhythmias and syncope and less likely the form with myocardial systolic dysfunction. In this case, the dog had moderate systolic dysfunction with ECG of non-sustained ventricular arrhythmia when recording using surface ECG. However, the runs of ventricular tachycardia were noticed by continuous ECG recording using holter device as seen by hollow arrows in Figures 2 and 3A. The ventricular arrhythmias showed many forms including the R on T phenomenon in which R wave (ventricular depolarization) occurs during the relative refractory period at the end of the repolarization (latter half of the T wave) (Fig 3B). The R on T can initiate abnormal ECG waveforms called torsades de points (solid arrows in Figs 2, 3C and 3D). The torsades de points is characterized by polymorphic ventricular tachycardia with a twisting of the QRS complexes around the isoelectric baseline (peaks going up and down). It is hemodynamic unstable causing hypotension and syncope. If it persists for a period of time, the ventricular fibrillation will be encountered and becomes a cause of sudden death.

Although the anatomic changes of the heart muscle may not be remarkable, the presence of VPCs in Boxer breed make the veterinarian pay more attention on cardiac sudden death. During syncope, the ECG waveforms could be changed into the sustained ventricular tachycardia which may not be recognized without 24-hour ambulatory ECG monitoring using the holter device. The sudden cardiac death is a result of changes from ventricular tachycardia to ventricular fibrillation. Although the genetic test of Boxer cardiomyopathy is available, a limit testing is performed. The antiarrhythmic drugs can be used with or without response. The most frequent medications is sotalol alone or a combination of mexiletine and atenolol. The reduction in VPCs may be associated with reduction in syncope episodes but it is not clear whether it can prevent sudden cardiac death. The dog that does not respond to the medication may need the implantation of an implantable cardioconverter-defibrillator.

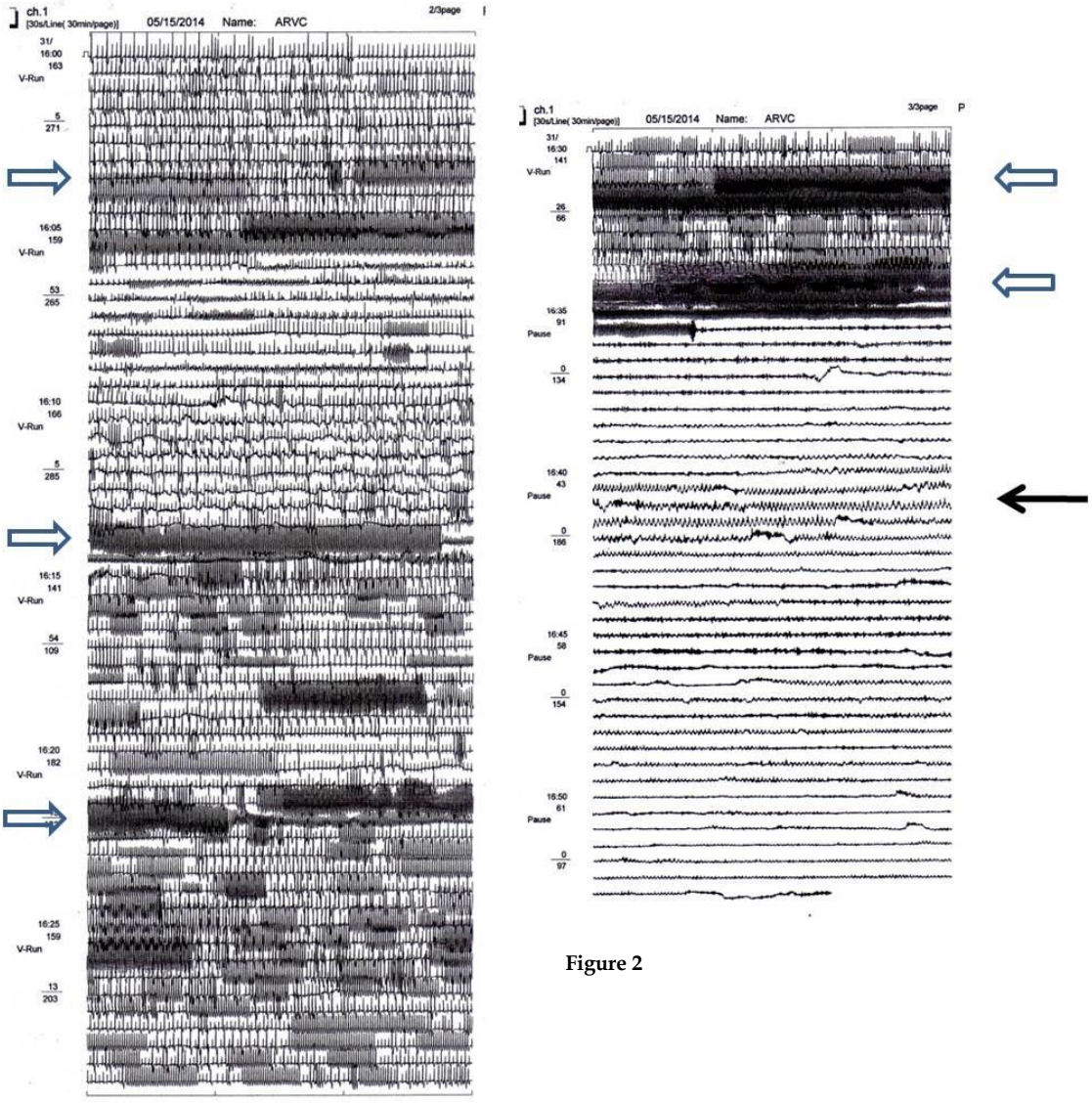

Figure 2

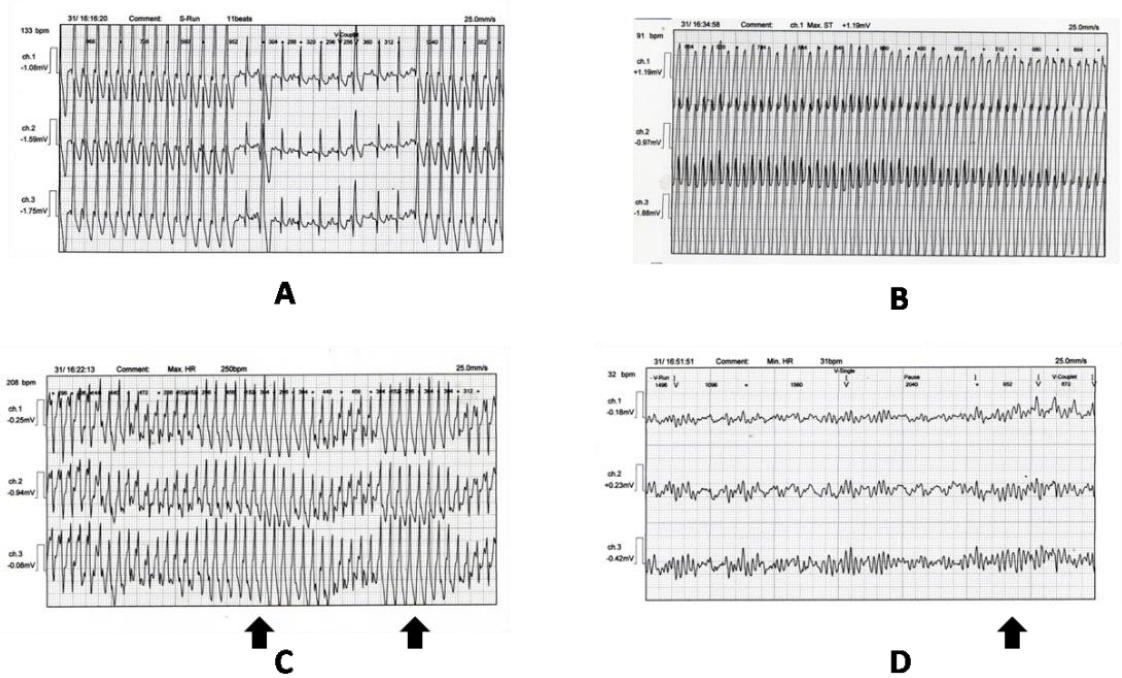

Figure 3