

ECG Quiz

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History

The eleven years old female spayed Saint Bernard weighting 55 kg. was presented at the emergency room, Small Animal Teaching Hospital, Chulalongkorn University with a history of exercise intolerance and severe panting after walking for a short distance on July 20. The dog had been checked up regularly every 6 months. She had gastric dilatation-volvulus in 2007 and was successfully corrected. The spleen was still intact. The cardiac arrhythmia such as accelerated idioventricular rhythm, paroxysm of both supraventricular and ventricular in origins and ventricular tachycardia were presented 1 to 2 weeks after the operation. The dog was treated with antiarrhythmic drug and a beta-adrenergic blocker.

From 2009 to 2010, she had history of skin mass which was diagnosed as mast cell tumors and also had mammary gland tumors which were also removed by surgery. In January 2012, the ovariohysterectomy was performed because of pyometra. Five years earlier, multiple small nodules were found in the spleen. Another symptom that was arisen and treated periodically was chronic cystitis. By performing echocardiography on 2010, the dog had dilated heart with fractional shortening (FS) of

18% and dilated cardiomyopathy was diagnosed. In April 2012, ECG was recorded (Fig 1a) while echocardiographic FS decreased to 15%. The holter was attached and recorded; periodic ventricular premature complexes (PVCs) were found while the arrhythmia was classified as grade II. One month after positive inotrope, pimobendan and the angiotensin converting enzyme inhibitor had been prescribed, the FS increased up to 20%.

After admission in the ER, blood chemistries and CBC were performed and the results were within normal ranges. The abdominal ultrasonogram revealed unremarkable changes of splenic nodules except a slightly larger two hypochoic nodules with diameter of 2.8 x 3.6 cm and 2 x 3 cm. The ECG was recorded as shown in figure 1b. The holter was re-performed; multiple (>15) VPCs were emerged in duplets throughout 24 hour period while grade III arrhythmia was classified (figure 2). The amiodarone, class III antiarrhythmic drug was prescribed. The ECG was repeated 4 days later (Fig 1c and 1d) with more PVCs. Amiodarone was still continued and ECG was recorded 17 days after administration as shown in figure 1e.

Please answer before turning to the next page.

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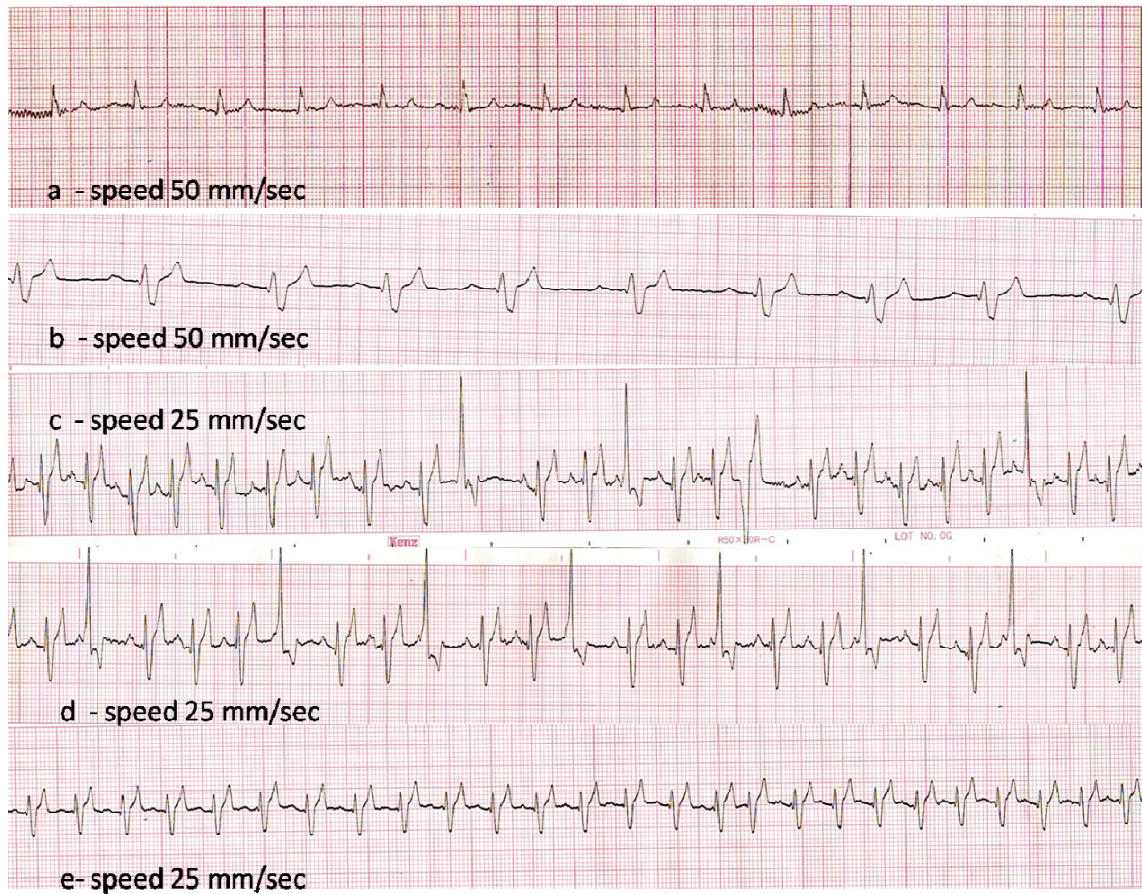


Figure 1 Electrocardiographic recording during 2012 on April (a), 20 July (b), 27 July (c and d) and 14 August (e).

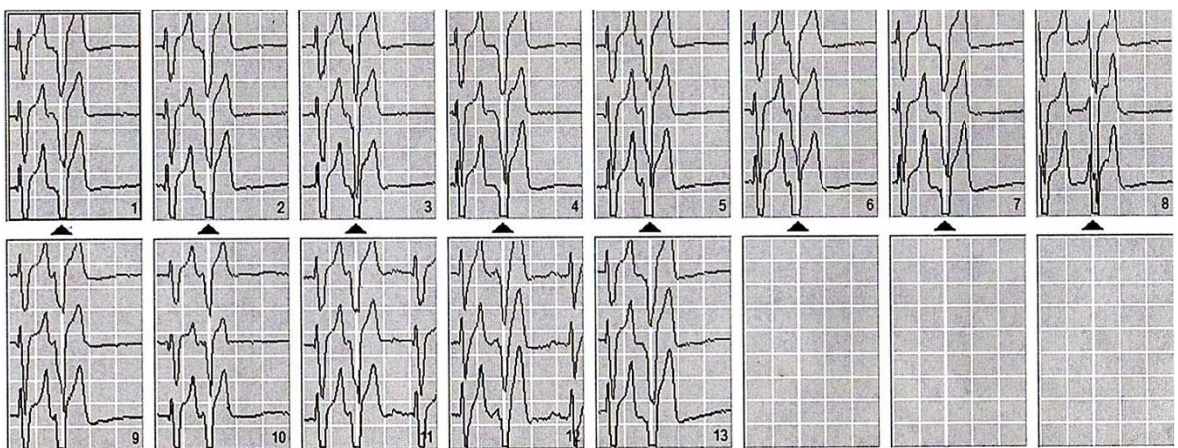
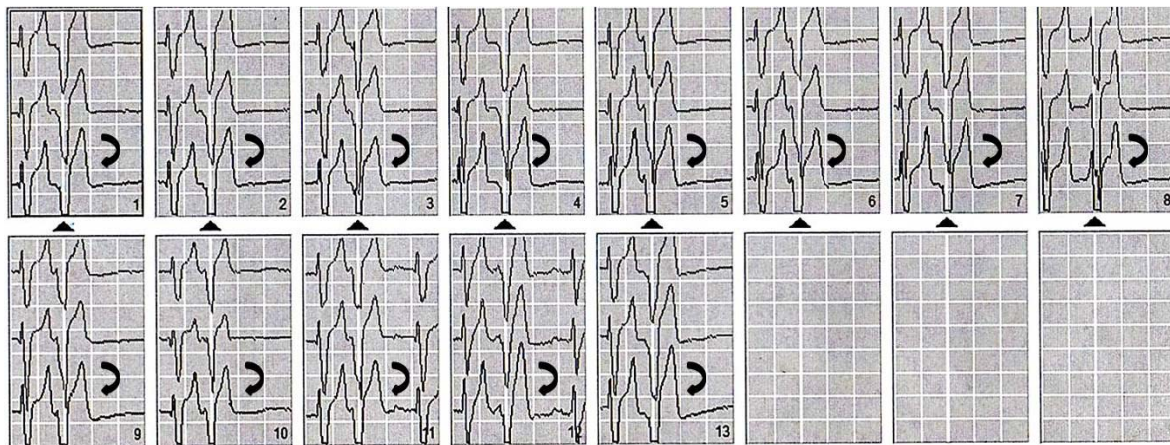
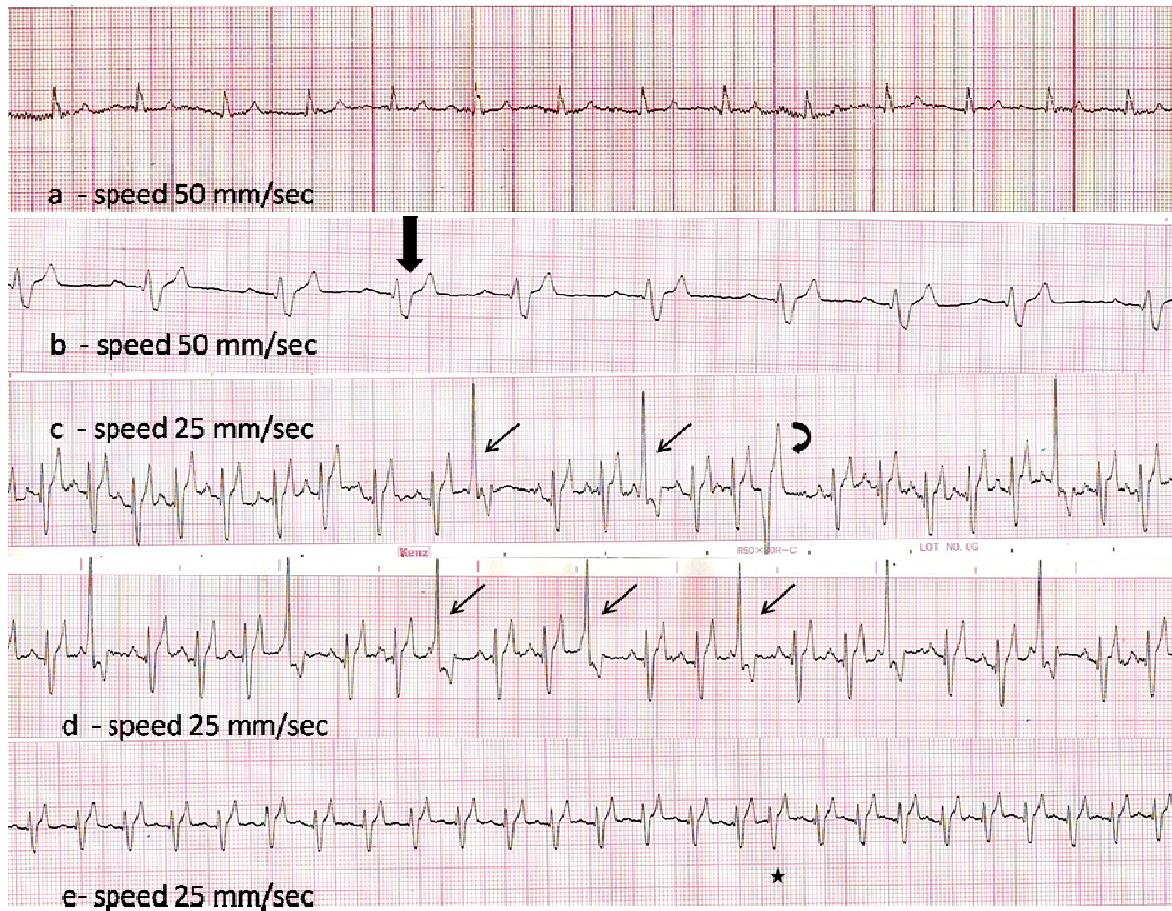


Figure 2 Hotler results on 19 July, 2012.

Interpretation

- Figure 1a – Normal sinus rhythm
 Figure 1b – Aberrant right ventricular conduction or right bundle branch block (RBBB)
 Figure 1c – RBBB with junctional complexes and premature ventricular complex
 Figure 1d – RBBB with bigeminy
 Figure 1e – RBBB
 Holter data – Sinus complexes with VPCs

From Figure 1a which was recorded in April, 2012, the dog ECG became normal with sinus origin. The rate was approximately 150 beats per minute

which was higher than normal for this breed. No clinical signs were complained by the owner. However, in the middle of July, dog had periodic

panting and ECG showed the complexes with deep S wave and wide QRS complexes (straight big arrow in figure 1b) with the rate of 100 beats per minute. This bizarre shape may be confused with the complexes from ventricular origin. However, all complexes had P-waves. The aberrant conduction especially on the right side of the heart existed. This type of ECG corresponds to the right bundle branch block (RBBB). Although the surface ECG could not catch the ventricular ectopic complexes, the 24 hour ECG recording using Holter monitoring showed the multiple ventricular premature complexes which occurred immediately right after the sinus beats (curve arrow in figure 2). The durations between VPCs and preceding sinus complexes were not identical. Therefore, they were not the ventricular bigeminy. The VPCs appeared very early closing to the repolarization of the preceding sinus complexes suggesting that the R on T could be arisen. When the surface ECG was reperformed a week later after the dog already received amiodarone for 4 days, the VPC with the same pattern was presented (curve arrow in figure 1c). Moreover, the normal shape QRS complexes which were coupled with preceding sinus complexes were seen (thin straight arrows in figure 1c and 1d). Since the shape of the premature complexes was resembled to the normal QRS shape, the impulse

may be originated from nearby the atrioventricular node. The junctional rhythm was presented with the rate of approximately 40 beats per minute (figure 1d). Since the duration between sinus complexes with aberrant right ventricular conduction and the junctional complexes were constant, therefore, the junctional complexes rhythm may appear due to reentry resulting in a bigeminy pattern. Reports of VPCs and accelerated idioventricular rhythm were demonstrated in dogs with splenic mass or GDV. This pattern even existed in this dog 1 to 2 weeks after correction of GDV in year 2007. Additionally, the VPCs could be found in a case of myocardial disease such as DCM. Increased occurrence of VPCs may also indicate the severity of DCM. After 17 days of amiodarone which is a class III antiarrhythmic drug, the ectopic foci from junctional and ventricular origins were not presented except one on supraventricular premature complex (star in figure 1e). The aberrant conduction of right ventricular pathway was still persisted with a high heart rate. The holter should be re-evaluated to see whether tachycardia developed when the dog stays at home without hospital stress. Other cardiac drugs that act on reducing heart rate without suppressing myocardial contractility may be needed.