What's Your Diagnosis?

Evaluation of Occasional, Sudden, Unprovoked, Episodes of Ataxia and Acute Collapse

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CLINICAL PRESENTATION

A 7-year-old, 35-kg, intact male Boxer dog presented to the cardiology service of the Koret School of Veterinary Medicine, the Hebrew University of Jerusalem, for evaluation of occasional, sudden, unprovoked, 10-45 second long episodes of ataxia and occasional acute collapse without loss of consciousness, of 4 years duration.

Upon presentation the dog was bright, alert and responsive. Physical examination revealed pink mucous membranes (MM) with a prompt capillary refill time (CRT). The respiratory rate was 32 breaths/minute and normal (bronchovesicular) lung sounds were auscultated over all lung lobes. An occasional irregular rhythm with a heart rate of approximately 140 beats per minute (bpm) was auscultated. A femoral arterial pulse deficit was palpated simultaneous to the abnormal rhythm.

A 20-second-long Lead-II ECG strip was recorded in a right lateral recumbence (Figure 1). An additional 30 second long Lead-II rhythm strip was also recorded during a collapse episode (Figure 2). During this episode, there was no loss of consciousness, the dog was quiet and responsive while lying in right lateral recumbence, MM were pale, CRT was prolonged, heart sounds were muffled and the femoral arterial pulse was not palpable.

What are the ECG findings, what is the ECG diagnosis, and should this patient be treated based on it?

ECG INTERPRETATION

The calculated heart rate is 162-168 bpm. The rhythm is irregular. There are many "gold standard" P-QRS-T complexes (the complex labeled "A" is of one example). Not all complexes
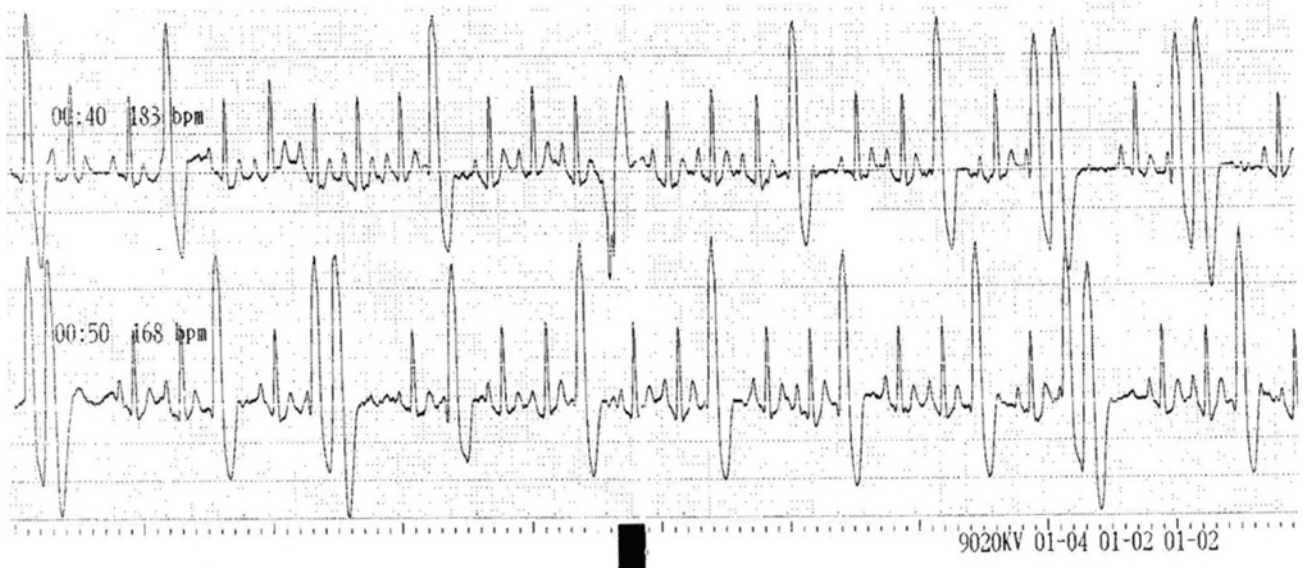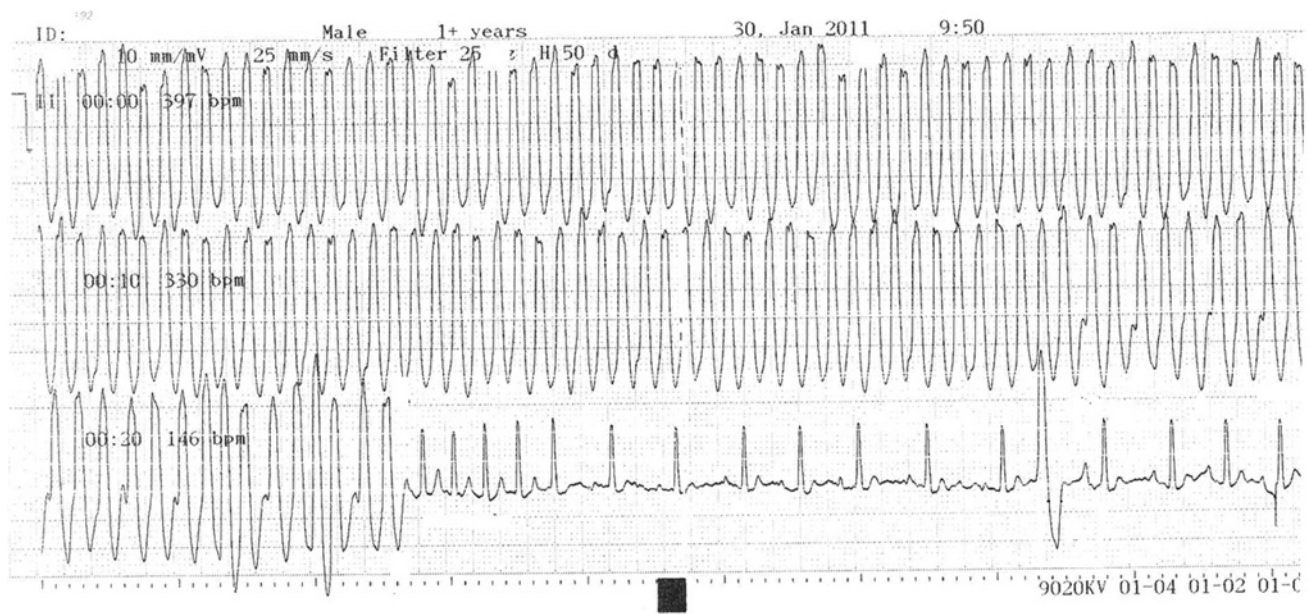

Figure 1

Figure 2

are identical to this specific beat in shape and character (e.g., complexes labeled "1-10").

In those "gold standard" complexes, there is a QRS complex after each and every P wave and a P wave precedes every QRS complex. The P-R interval in these complexes is constant, indicating a causative relationship between the P and the subsequent QRS complex. This finding, along with the rapid rate, confirms the presence of sinus tachycardia.

All measurable amplitudes and intervals are within normal range (WNR) for these complexes (P=0.2 mV & 40 msec, R=1.5mV, P-R=100 msec, QRS=60 msec, QT=200 msec). The Mean Electrical Axis (MEA) cannot be calculated using solely one Lead.

The complexes labeled "1-10" are all wide (QRS=80msec) and "bizarre" (i.e., have a different morphology when compared to the gold-standard complexes)
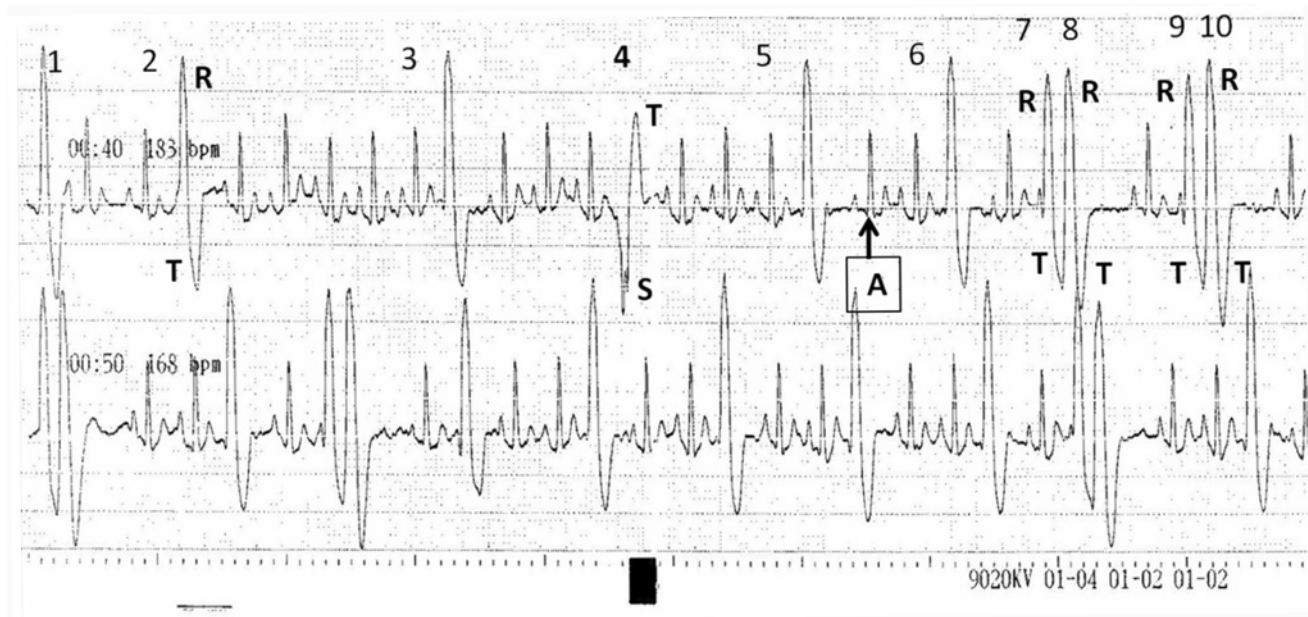

Figure 1 annotated: A 20 second long, continuous recording of Lead-II electrocardiogram divided into two continuous rows of 10 seconds each. Calibration is 10mm=1mV and paper speed is 25mm/sec.

and are also positive in Lead-II, with the exception of the complex labeled "4".

The complexes labeled "2, 3, 5, and 6" are all Ventricular Premature Complexes (VPC's). Since the MEA cannot be calculated using one single lead, one can only speculate that the origin of most VPC's is right-ventricular, since they reflect a depolarization process propagating away from the negative electrode and toward the positive electrode, which, when using Lead-II, means this front propagates towards the left ventricular apex. It is therefore likely (although not definitively proven) that this front originates in a focus localized at the right ventricle. The complexes labeled "7-10" are 2 pairs ("couplets") of tightly adjacent VPC's. Three additional such couplets are seen to follow in the next row of this rhythm strip. Finally, the negative complex labeled "4" is a VPC suspected to be of a left ventricular origin.

in any form may necessitate further diagnosis and sometimes also justify treatment.

Notice that the second R wave in each couplet comes just after (and sometimes before the completion of) the preceding T wave. This is called the "R-on-T phenomenon", a premature complex that might potentially initiate ventricular tachycardia or even ventricular flutter or fibrillation, especially in a patient with a sick (i.e. ischemic or fibrotic or metabolically deprived) myocardium.

Summary of Findings in Figure 1

1. Sinus Tachycardia.
2. Frequent VPC's (singles and couplets); suspected to be of right ventricular origin.
3. A single VPC suspected to be of left ventricular origin.
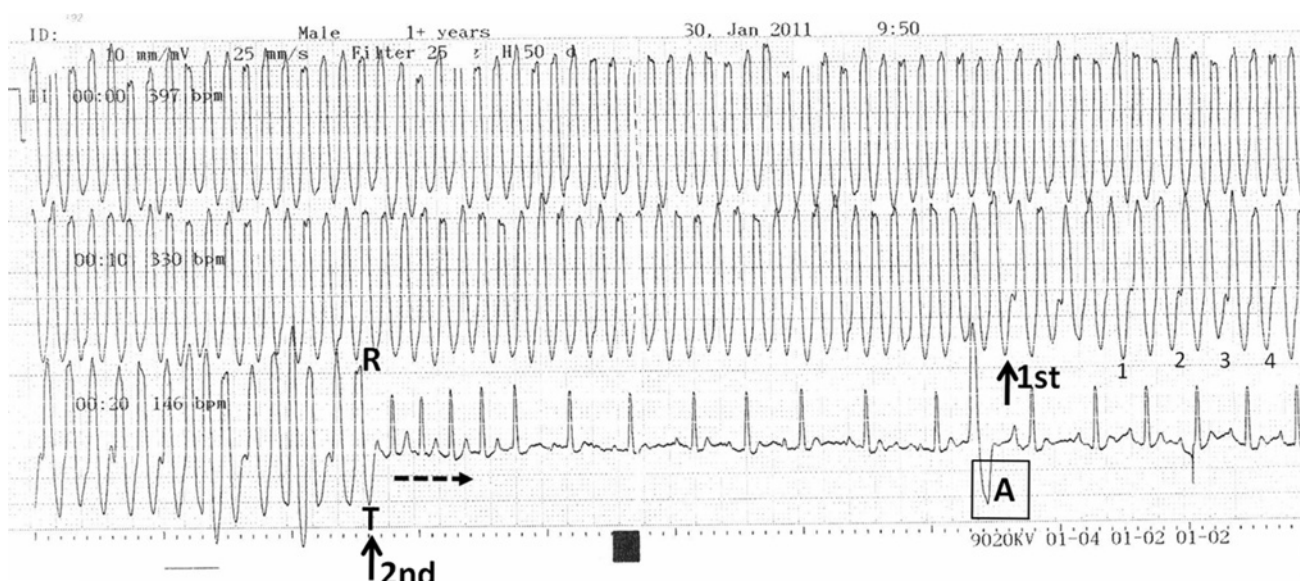4. "R-on-T" phenomenon.

Figure 2 annotated: A 30 second long, continuous recording of a Lead-II rhythm-strip divided into three continuous rows of 10 seconds each. Calibration is 10mm=1mV and paper speed is 25mm/sec.

Ventricular premature complexes are an example of an ectopic rhythm, originating from an area distal to the AV nodal (junctional) tissue. Their QRS complex may be abnormally wide and unusual ("bizarre") in shape. They can occur as singles, in pairs (couplets), triplets, or in runs or sequences or "spells" (four or more are called a "ventricular tachycardia"), or alternate regularly with sinus beats (ventricular bigeminy or ventricular trigeminy). Occasional VPC's may be an incidental finding in healthy animals. However, frequent VPC's

The calculated heart rate in the first two rows is 372-390 bpm. This rate is decreased to ~340 bpm during the last 2 seconds of the second row and the first second of the third row, and again to a 144 bpm during the second half of the third row. The horizontal arrow marks a change in rhythm through 5 atrial premature complexes (APC's) to the "gold standard" morphology, previously described as sinus rhythm. There is a single VPC (6 from last) during this sinus rhythm, labeled "A".

Up to the 1st arrow (17 second duration), one cannot distinguish QRS complexes from T waves. Within each pair of consecutive positive and negative deflections, each of these deflections seem wide (~80msec), the combination of which is suggestive of a ventricular origin. This tachyarrhythmia is likely sustained Ventricular Flutter (VFL), which is a variation of ventricular tachycardia (VT) but is considered to be a more "malignant" variation (i.e., carrying a higher risk of degenerating into ventricular fibrillation (VF)). This risk has to do with the fact that each and every complex during VFL actually reflects the "R-on-T" phenomenon. It is, therefore, characterized by an extremely high ventricular rate, typically with a regular rhythm. The ECG shows large sine wave-like complexes that oscillate in a regular pattern. P-waves are not present, and the QRS complex (reflecting ventricular depolarization) is indistinguishable from the T wave (reflecting ventricular repolarization).

During the time between the 1st and 2nd perpendicular arrows (5 second duration), there is a mixture of wide polymorphic complexes. Two of these (the 7th and 3rd ones preceding the second perpendicular arrow in the third row) are sine wave-like complexes consistent with VFL while in most other complexes the QRS and the T are, in fact, distinguishable, just because one of them (the negative deflection) is somewhat wider (longer in duration) than the other (the positive deflection). The longer likely represents ventricular repolarization while the shorter likely reflects ventricular depolarization. Their being distinguishable renders this short run of ~340 bpm consistent with non-sustained VT, rather than with VFL. Some of these VT complexes are labeled 1 through 4 under the second row.

Ventricular tachycardia occurs when three or more VPC's occur in a row, and is characterized by an elevated ventricular rate, often (but not always) with a typical regular rhythm. QRS complexes are usually aberrant in shape and P waves are not usually present, unless the overall heart rate is slow enough for them to be seen occasionally. T-waves may be distinguished from QRS complexes, and are typically "discordant" (i.e., opposite in direction relative to the most dominant deflection in the QRS complex).

What ventricle do you suspect the VFL/VT originate from?

One way to answer this question is to look at the very last complex of the VFL/VT run, just above the second perpendicular arrow. Here the last deflection seen has to be the T wave, since every electrical systole must end with ventricular repolarization. The T wave is negative in this specific Lead-II strip and the preceding deflection is therefore the QRS complex, which reflects ventricular depolarization. Since it is positive, a right ventricular focus can be suspected as its origin.

Another way to answer this question is to seek a single VPC (being single means its depolarization and repolarization are distinguishable, as, by definition, the former has to occur during its onset while the latter has to occur during its offset) of the same morphology as most other ventricular complexes on this strip, such as the many single VPC's in Figure 1, as well as the last VPC labeled "A". These single VPC's are suspected to be of a right ventricular origin because of their positive QRS complex in Lead -II. Lastly, the fact that the VT enables us to distinguish depolarization from repolarization would assist in such deduction as well, following the same reasoning. By way of deduction and comparison to the VFL morphology, one can also conclude in retrospect that in this patient VFL, too, probably originates in a right sided focus.

Summary of findings from Figure 2

1. Paroxysmal ventricular flutter (with an average rate of ~380bpm) of 20 second duration, suspected to be of a right ventricular origin.
2. Non-sustained ventricular tachycardia of 5 second duration immediately following the sustained VFL.
3. Five APC's following the offset of the VT run, just prior to sinus rhythm with a single VPC.

How do you propose to manage this case?

The first strip shows frequent VPCs, with an occasional "R-on-T Phenomenon" that is known to potentially carry a risk of degenerating to VFL followed by lethal ventricular fibrillation (VF), especially if myocardial disease is present. Such degeneration is indeed demonstrated in the second strip, recorded only minutes following the first strip. The clinical significance of VPC's depends on their frequency, duration, rate, and most of all, their hemodynamic consequences. This dog did demonstrate clinical signs concurrent with his recorded VFL, meaning it definitively did trigger hemodynamic "embarrassment". It should therefore receive a complete echocardiographic examination to seek concurrent myocardial or other cardiac disease. Another test to consider

is a 24-hour long, portable ambulatory ECG ("Holter monitor") or an "Event monitor" (a real time ECG monitor that is turned on by the owner when a clinically detectable episode occurs), depending on the typical frequency of such episodes. Such monitoring and recording may increase the chances of recording and identifying paroxysmal arrhythmias that might be causing clinically noticeable episodes.

"Luckily" (from a diagnostic point of view), this patient demonstrated a "live" episode of acute collapse with an ECG running simultaneously, revealing a sustained, rapid "malignant" VFL immediately followed by a somewhat slower (but still extremely rapid) non-sustained (i.e., of less than 30 seconds duration) VT.

The primary concern with such rapid VFL or VT is inadequate cardiac output due to the decreased venous return and therefore the decreased ventricular filling. Such decrease in cardiac output would immediately translate into systemic hypotension, as well as a diminished coronary (and therefore myocardial) perfusion, in addition to the decreased perfusion pressure to the rest of the body, including the kidneys and the brain, which (along with the myocardium) are the 3 organs that most depend on perfusion pressures. In a heart of which myocardium is sick (ischemic/fibrotic/inflamed/infiltrated) such decrease in myocardial perfusion at a time when its demand is maximal due to the rapid heart rate, metabolic and biochemical deterioration would immediately translate into electrophysiological deterioration, i.e. lethal VF.

For these reasons, when the VT is not only frequent but also rapid and sustained, and especially when symptomatic and when degenerating into VFL, it requires emergency therapy to prevent life-threatening hypotension and/or arrhythmogenic death.

One can start intervention with slow intravenous Lidocaine HCl at 2 mg/kg, which may be repeated up to a cumulative maximum of 8mg/kg over 10 minutes. If there is a response, begin a constant rate infusion (CRI) of 25 -80 micrograms/kg/min, *immediately* following an IV bolus of 2mg/kg. Potassium chloride can be added to the CRI bag to increase the likelihood of success, especially if hypokalemia is documented. Note that Lidocaine is also a negative inotropic agent and therefore has to be administered slowly and cautiously.

Chronic oral anti-arrhythmic drugs should also be initiated. One such possible agent is Sotalol (a class III and a beta-blocker), and others that may be considered include Procainamide, or a combination of Mexiletine and Atenolol.

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