CARDIOLOGY
ECG OF THE MONTH

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CASE REPORT

A 10 year old spayed female, mixed brachycephalic dog, weighing 25 kg was presented for routine annual vaccination. The owners described a healthy energetic dog with a good appetite and normal bowel movements. On physical examination the heart rate was noted to be irregular and on femoral artery palpation an occasional pulse deficit was detected. The first pulse palpated after the pulse deficit appeared to be much stronger than others. The rest of the physical examination was unremarkable.

The concern generated by these findings led the clinician to carry out an ECG and to request a blood panel consisting of complete blood count, electrolytes and biochemistry. All clinical pathology results were within normal limits.

A 30-second-long Lead-II rhythm strip was recorded (Figure 1A). A short strip of all 6 leads at a paper speed of 50 mm/sec was also recorded (Figures 1B and 1C).

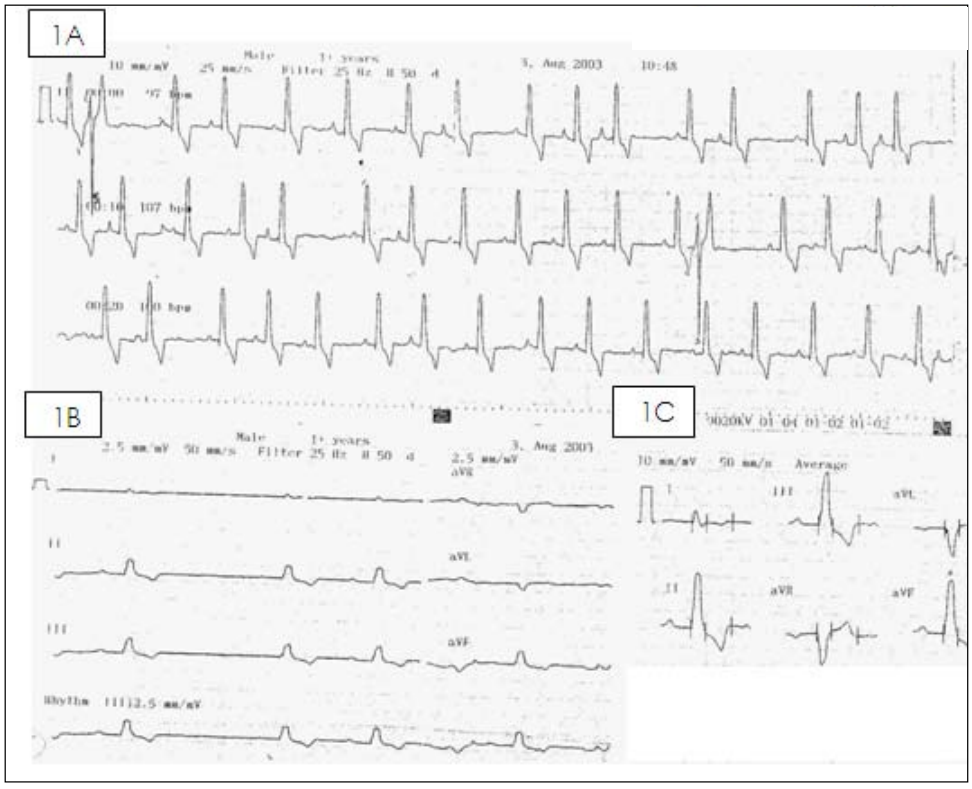
What is your diagnosis and why? What are the possible etiologies? How do you propose to manage this case?

LEGENDS FOR FIGURES

Figure 1: Electrocardiograms recorded from a 10 year old spayed female, mixed brachycephalic dog. **A**: a 30 second continuous recoding of Lead II divided into three continuous rows, each representing 10 seconds recording. Calibration is 10mm=1mV and paper speed is 25mm/sec. **B**: A short recording of six frontal plane leads, calibrated at 2.5mm=1mV (note the difference in calibration between Figure1A and 1B causing the amplitudes to appear different). Paper speed is 50mm/sec, which causes waves and complexes to appear wider than those in Figure 1A. **C**: This section represents an average of one beat per lead for each of the six leads. Calibration: 10mm=1mV, paper speed 50mm/sec. This calibration and paper speed is the most useful for measuring intervals such as P, PR (PQ), QRS, and QT.

Turn the following page to read the interpretation and diagnosis

Figure 1



What are the ECG findings? Should this patient be treated based on these finding, and if it should, what is the best treating strategy?

It is recommended to examine the ECG systematically in order to collect and then integrate all findings, prior to concluding a final diagnosis. The following features should be examined:

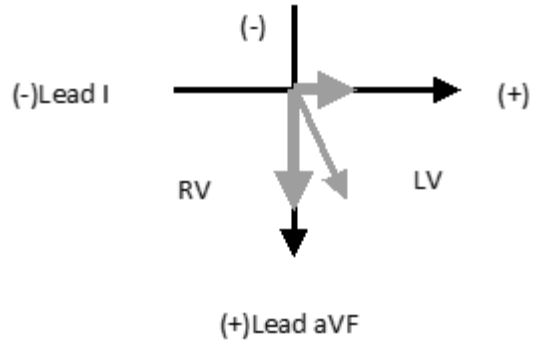
1. Is the ventricular rate normal? Determine whether Tachyarrhythmia or Bradyarrhythmia is present taking note of the paper speed.
2. Is the rhythm regular? If not, define the rhythm changes.
3. Try to find at least one "gold standard" (sinus) beat which can be used as a reference of comparison to other beats. Are all P-QRS-T complexes identical to this specific beat, in shape and character?
4. Are there any QRS complexes that do not have a P wave preceding them? Are there any P waves that are not followed by a QRS complex?
5. Is the P-R interval repeatable and constant?
6. Measure the width of P wave, P-R interval (from the beginning of P wave till the beginning of QRS), the width of QRS complex, the Q-T interval (from the beginning of QRS complex till the end of the T wave), and the amplitudes of P, R, and S waves. (See Table 1 for normal values).
7. Calculate the Mean Electrical Axis (**MEA**) using the algebraic sum of the predominant QRS deflections in any two leads. Using leads I and aVF may often be the most convenient, but nit necessarily the only pair for this purpose.
8. Are there any pre-mature complexes? Note their timing, width and morphology.

ANSWERS:

1. The calculated heart rate was 90 beats per minute (bpm).
2. The rhythm was irregular. The third row is the most useful to observe in this respect. The R-R interval appears to change in a predictive periodical manner which can be defined as "regularly-irregular". This finding is characteristic of Sinus Arrhythmia, indicating that the arrhythmia originates from the Sino-Atrial node (SA node) and is the result of regular and periodic changes in the autonomic input to that main pacemaker. Sinus arrhythmia confirms the presence of an arrhythmia but also establishes that the irregularity is not pathologic. The presence of sinus arrhythmia is incompatible with heart failure as it indicates a sufficiently high parasympathetic tone. On physical examination, one might have noticed a cyclic change of heart rate related to respiration, with an increase during inspiration and decrease during expiration. If so, that is also a normal finding characteristic, termed Respiratory Sinus Arrhythmia**.**
3. There were many "gold standard" P-QRS-T complexes (see the complex labeled "A" in Figure 2, as an example).
4. There is a QRS complex after each and every P wave. There is a P wave before every QRS complex, except for the complexes marked "B" and "C" in Figure 2
5. The P-R interval was constant, indicating a causative relationship between the P wave and the subsequent QRS complex. This finding confirms the presence of sinus rhythm.
6. Measurements of waves and intervals should be performed in Lead II. (Table 1)

Normal findings:

1. The amplitude of the P wave was seen to change in a cyclic way. This is considered a normal finding referred to as a "wandering pacemaker" which is commonly seen with sinus arrhythmia. Although P wave width was longer than the normal range (P>0.04sec) and might have indicated the presence left atrial enlargement, this is considered a non-specific finding and may be of little significance where there is no other supporting evidence such as radiographic or sonographic left atrial enlargement.
2. R wave amplitude was normal



3. Q-T interval was normal

Figure 2

Abnormal findings:

1. The P-R interval was longer than the normal range at 0.14 ms (see Table 1), indicating a first degree AV block. This conduction disturbance derives from a slow conduction of the AV-node. Possible etiologies of prolonged conduction across the AV-node may be associated with disorders that cause an increased parasympathetic tone. It may be an incidental finding or resultant to anti-arrhythmic drugs therapy, which was not administered to this patient). There are no immediate hemodynamic consequences for this condition and therefore there is no indication for treatment.
2. Prolongation of the QRS complex (0.09 sec). Possible etiologies: Severe hyperkalemia, ventricular escape beats due to severe bradycardia, an extremely sick myocardium (e.g. cardiomyopathy or sub-aortic stenosis with secondary myocardial fibrosis) (which are unlikely as dog seems clinically healthy and has a high vagal tone), ventricular premature complexes (VPC**)** or a bundle branch block (BBB).

Although there was a slight conduction disturbance through the AV-node, there was still a constant P-R interval with sinus rhythm at a normal rate of 90 bpm. All these findings rule out ventricular escape complexes or VPCs (as opposed to the 2 complexes labeled B and C; see below) and indicates that the cause for the prolonged QRS can only be derived from a ventricular conduction disturbance, i.e. a BBB.

In a normal heart, the activation of the ventricles takes place nearly simultaneously thanks to the fast conduction

through the Purkinje fibers. These fibers are located at the left and right branches of the bundle of His that runs through the sub-endocardium of the inter-ventricular septum. Each branch passes the electrical impulse arriving from the atria to the ventricles through the AV-node, to its corresponding single ventricle. With a (left or right) BBB, there is a lesion in one of the bundle branches that completely blocks the conduction down this branch to the ventricle. The result is a blockade of the "fast" conduction to the affected ventricle. The depolarization process in this ventricle, therefore, propagates very slowly through contracting myocytes that are not specialized in fast conduction. As a result, a slow "bypass" is formed in one ventricle while the other still does go through a normal "fast" propagation process that ends much earlier. The graphic result is a wider (slowly inscribed) QRS.

What kind of a Bundle Branch Block does this dog have? Right (R-) or Left (L-) BBB?

A left-BBB can often be recognized based on a supra-ventricular origin (i.e. having a P-wave in front of it, with a constant PR interval), a wide QRS, a left-sided ventricular MEA and a predominant R wave on Lead I. Often, a right BBB is similarly of a supra-ventricular origin with a wide QRS, a right ventricular MEA and a predominant S wave in Lead I.

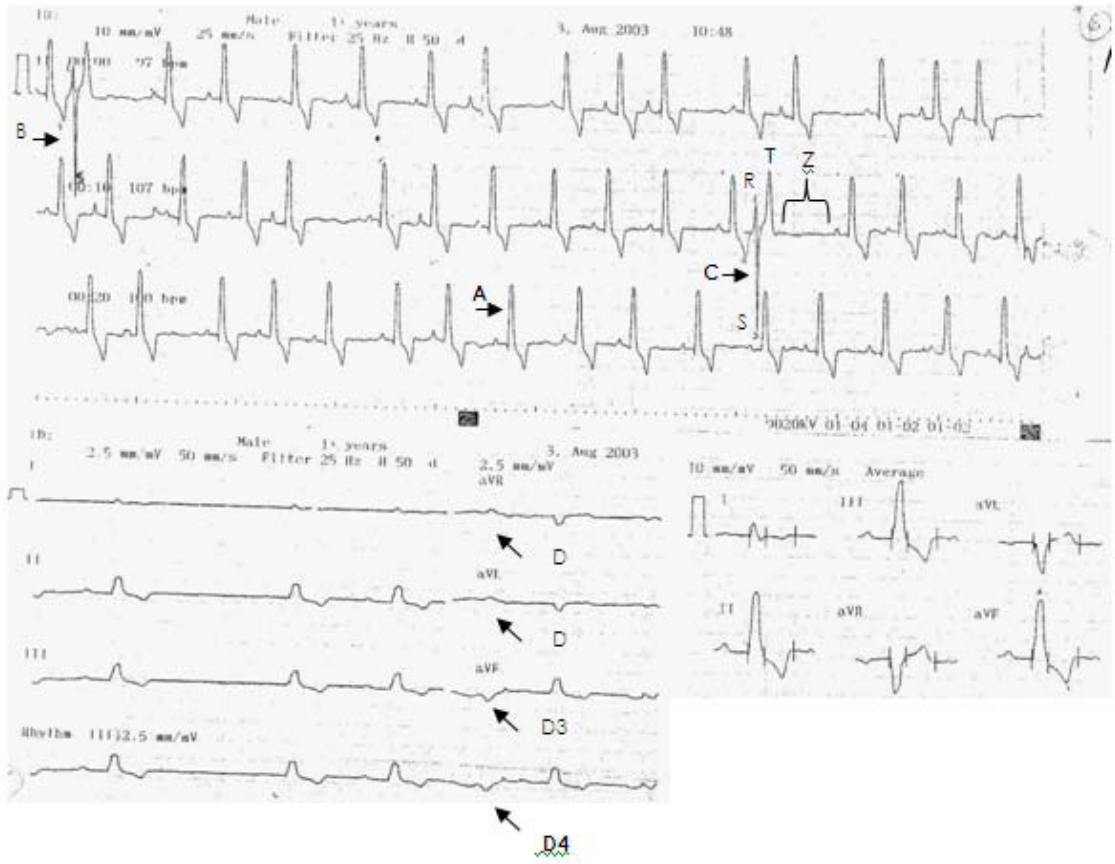
According to Lead I (at the lower right corner Labeled C in Figure 1), this dog had a left BBB. The normal "fast" propagation of the right ventricle ended much earlier than the left ventricular one. The graphic result was that the S wave (RV propagation) was "swallowed" and offset by the R wave (reflecting mostly the LV propagation), which is more dominant because by the time it completes its course there is no longer any negation coming from the faster-conducting right ventricle.

In a healthy myocardium, there is only little hemodynamic impact as a result of a BBB. Nonetheless, there may be some prognostic value to this diagnosis since a left BBB is typically expected to be less reversible than a right BBB. No treatment is indicated, nor is one possible (other than via invasive electrophysiological intervention which is not yet available to veterinary patients).

7) The mean electrical axis of the ventricles was calculated using the complexes illustrated in Figure 1C: the QRS complex in Lead I was mostly positive. In Lead aVF it was also positive. The MEA is aimed, therefore, towards the left ventricle (Figure 3). When combined with a sinus rhythm demonstrating wide QRS complexes, this finding is compatible with a left BBB.

Figure 3

Two pre-mature complexes were seen on the Lead II strip, labeled B and C in Figure 2. They were both identical to each other, but morphologically very different from the "gold standard". Their timing was early, and they begin at the very end of the preceding T wave with a direction opposite to the other complexes. The origin of a pre-mature complex that looks different form the "gold standard" is usually ventricular (e.g. a VPC) as opposed to an atrial pre-mature complex (APC) which looks identical (albeit early in timing) to the "gold standard". The VPCs' morphology is different, because there is an ectopic ventricular origin to this beat, that forces the ventricular depolarization front to propagate between myocytes in an abnormal course which does not utilize the



fast conducting Purkinje fiber network. This course does not allow for rapid conduction, hence is slower than usual and is inscribed as a wide QRS complex. Note that the T wave of this VPCs might be mistakenly identified as an R wave, appearing almost the same as the R wave of the preceding complex. Although its morphology is almost identical, there is an essential difference between the two: the R wave represents ventricular depolarization as opposed to the T wave that represents ventricular repolarization. This difference can be picked up by carefully paying attention to timing: in order to be sure a suspected wave is a T wave it is necessary to measure the Q-T interval, which is typically longer than a QRS complex. In this case, Q-T=0.22sec, which indicates that this is truly a T wave rather than an R wave.

Why do these VPCs seem so narrow? In fact, they are not narrow at all, as their width is above normal range at 0.09sec. Their appearing narrow is only an optical illusion because of a relative (rather than absolute) difference: while the VPCs are composed of 2 waves with a total duration of 0.09sec, the "gold standard" complex is composed of one single (R) wave of an identical duration, and therefore appears wider.

What's the location of the ectopic focus or foci? This question cannot be answered judging a single lead. In Lead II, the (negative) S wave in complexes B and C is dominant. Hence the depolarization front propagates away from the positive electrode of this particular lead, i.e. moving toward the right ventricle. Based on this one can only suspect that the origin may be left ventricular. Another VPC labeled "D" in Figure 2 is different in shape and direction from the other complexes. It has a dominant S wave in Leads II (D4) and aVF (D3), and a positive R wave in Leads aVR (D1) and aVL (D2). Because it is taller in aVR than it is in aVL, this indicates that the depolarization not only propagates towards the head, but also moves towards the right. It is now possible to confirm that Complexes B and C in Figure 2 are definitively left ventricular VPCs.

The hemodynamic consequence is only momentary. The pre-mature beat doesn't allow the heart to fill properly during the short preceding diastole, thus causing a transiently decreased stroke volume (SV) following this specific beat. This is the reason for the pulse deficit. The volume of the next beat will be above normal because there is a prolonged pause after the VPC (labeled "Z" in Figure 2), resulting in the diastole that immediately follows the VPC being longer and leading to a higher end-diastolic volume and an increased SV (according to the Frank-Starling law). This is felt as the single strong femoral pulse that follows the pulse deficit, as described earlier.

There is no treatment indicated for these two VPCs, as the dog is stable and asymptomatic, based on the history, physical examination, and background sinus arrhythmia attesting to a high vagal (parasympathetic) tone. There is probably no myocardial pathology and the VPCs will unlikely increase the risk of secondary complications. However, it would be advisable to consider an echocardiogram to confirm the condition of the heart, especially since there are two separate findings involving the left ventricle.

Summary of Findings:

1. Sinus arrhythmia with a wandering pacemaker, reflecting a high enough vagal tone.
2. A first degree AV Block, probably reflecting a conduction anomaly.
3. A Left BBB.
4. Left ventricular VPCs

The main finding of this case is the combination of two types of conduction disturbances in two different cardiac locations (one is supra-ventricular and the other is intra- ventricular). This is probably not coincidental and may be indicative of a rather diffuse conduction disturbance. Nonetheless, the conduction disturbances did not appear to have hemodynamic consequences at the time of ECG recording and the presence of a high vagal tone indicated there was no distress at that time. The VPCs occurred at a low frequency and therefore there appeared to be no need for treatment at that point in time. It is recommended, however, to follow up the case with a periodic ECG, to make sure the AV block does not degenerate to a higher degree. Also, in light of the findings echocardiography should be considered.

Table 1

Wave	Normal Reference Range	Amplitude (mV)	Width (sec)
P	**≤ 0.04 sec ≤ 0.4mV**	Changes along with the sinus arrhythmia up to a maximum of 0.3	**0.06**
R	**≥2.5mV (≥ 3.0 mV in Giant Breeds)**	**1.5**	
QRS	**≤ 0.06sec; (≤ 0.065 sec in Giant Breeds)**		**0.09**
P-R	**0.06-0.13 sec**		**0.14**
Q-T	**0.15-0.25 sec**		**0.22**