CARDIOLOGY

AN ASYMPTOMATIC DOG WITH POTENTIALLY SEVERE HEART DISEASE. WHAT IS YOUR DIAGNOSIS?

Ohad D.
The Koret School of Veterinary Medicine
The Robert H. Smith Faculty of Agriculture, Food and Environment
The Hebrew University of Jerusalem
P.O.Box 12, Rehovot 76100

HISTORY

A 2 year old, totally asymptomatic, intact male Boxer dog was presented for an annual vaccination, after having received previous vaccinations another veterinary clinic. His appetite and activity levels were reportedly normal.

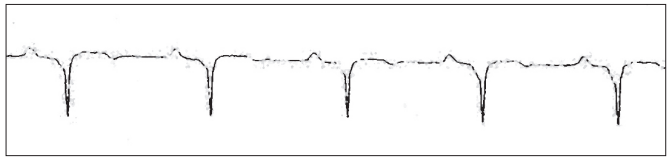
Physical examination revealed a perfectly normal looking, highly viable dog, of a normal body weight for dog's size and breed. Mucous membranes were moist and pink with a normal capillary refill time. Femoral arterial pulses were regular, strong and full, with a heart rate of 132/min.

The respiratory rate was 26/min and his rectal temperature is 38.7^{o}C. Respiratory sounds were normal ("broncho-vesicular") over all lung lobes and a Grade IV/VI systolic murmur was auscultated. The murmur was of the "diamond-shaped" type (also termed crescendo-decrescendo), indicating that it first gets louder and then becomes softer throughout each systolic event). The murmur was auscultated over the left hemi-thorax with the point of maximal intensity over the left heart-base.

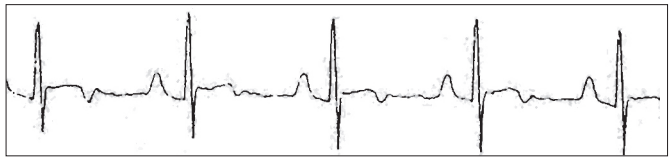
An electrocardiogram (ECG) tracing was recorded from this patient while in right lateral recumbency, under no chemical sedation, using a 10 mm/mV calibration and a sweep-speed of 50 mm/sec. How can the ECG tracing assist you in narrowing down this list, or even making a definitive diagnosis?

ECG tracing (10 mm/mV calibration and a sweep-speed of 50 mm/sec)

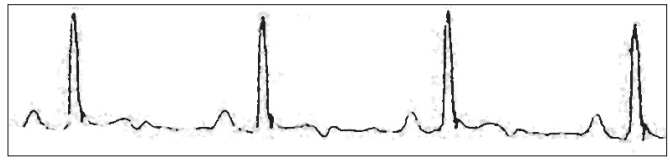
Lead I:



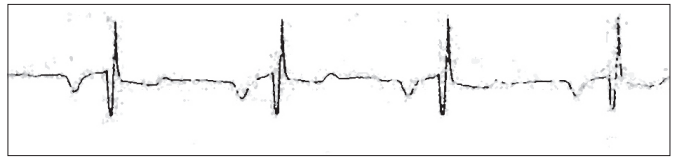
Lead II:



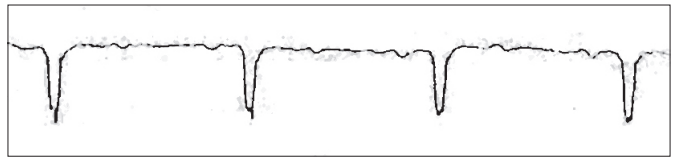
Lead III:



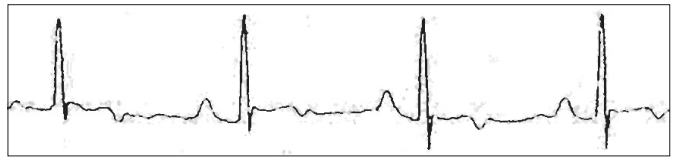
Lead aVR:



Lead aVL:



Lead aVF:



For the diagnosis and interpretation turn to Page 35

DIAGNOSIS AND ECG INTERPRETATION

The relatively young age and the patient's breed are both compatible with a congenital heart disease.

The murmur's description and location are both consistent with either a) subaortic stenosis, or c) pulmonic stenosis, because both of these disease entities involve an obstructive stricture at or near one of the two semi-lunar valves, both of which are located at the left heart base level. Any of these stenoses, if only severe enough, may make it necessary for the corresponding ventricle to contract very forcefully so as to overcome the increased resistance (afterload) to its emptying. Such a forceful contraction generates a high systolic pressure-gradient across the stricture, which translates into a high-velocity and a highly turbulent (rather than laminar) jet. This results in a loud systolic murmur. The murmur intensity increases during the early phase of systole when intra-ventricular pressure is building up very steeply, and then decreases during the later phase of that same systole, as ventricular contraction gradually ceases and an equilibrium is gradually generated between that ventricle and the major artery that is leaving it.

A 3rd differential diagnosis may be a "peri-membranotic" (i.e. located high at the inter-ventricular septum, near its membranous rather than its muscular component) ventricular septal defect (VSD). Its high-velocity turbulent systolic jet would be coming from the left ventricle across the cranio-dorsal aspect of the septum, into the right ventricular outflow tract, which is, too, located at the left heart base. However, another murmur is typically generated by this type of congenital heart defect and is best heard over the cranio-ventral right hemithorax rather than the left base. Because this is not included in the physical examination findings, a VSD is the least probable differential diagnosis in this specific case.

The ECG demonstrates regular sinus rhythm (there is a P-wave before each QRS-complex and a QRS-complex following each P-wave) with a constant PR-interval in each cardiac cycle, suggestive of a causative relationship between the P and the QRS. Measurable intervals in Lead-II (P, PR, QRS, and QT) and amplitudes (P and R) are within normal range for this lead. There is also a subtle, cyclic change in the P-wave amplitude throughout most leads, consistent with the so-called "wondering pacemaker" phenomenon, which is a perfectly normal finding and, in fact, has nothing to do with any "wondering" process of the sino-atrial node. Rather it is the result of cyclic changes in the angle between the positive electrodes in any of the bipolar ECG leads, and the physiologically changing location of the heart apex throughout the respiratory cycle.

The only true abnormality that can be noted has to do with QRS polarity in some of the leads, most noticeable in Lead-I and Lead-aVR: in the former, the QRS is totally negative while it should typically be positive, and in the latter it is more positive than negative, while it is often negative in other dogs. These two findings suggest that during the onset of the electrical ventricular systole, the intra-ventricular depolarization front propagates toward the negative electrode of Lead-I, which also "happens" to be one-and-the-same as the positive electrode of Lead-aVR (See Figures 1-3)[3]. This electrode is located over the front right limb, meaning that the depolarization front is traveling towards the right side of the heart, rather than towards the left ventricular apex, as it "should".

Theoretically, the reason for this abnormal direction can either be an intra-ventricular conduction aberrancy within the right ventricle (an unlikely option given the normal duration of the QRS-interval in all 6 leads) or a severe enough right-sided ventricular hypertrophy, which is most consistent with Differential Diagnosis #2, pulmonic stenosis. Despite the patient's lack of symptoms at this time, his disease is severe enough to have triggered measurable ECG findings, and is therefore likely to trigger some future symptoms such as exercise intolerance, syncope, and eventually right-sided congestive-heart-failure, with or without (potentially life-threatening) ventricular arrhythmia.

Note that sometimes the two front lead electrodes may be inadvertently switched by whoever connects them to the patient, resulting in a negative QRS in Lead-I. This is unlikely the case with this specific ECG tracing as the P-wave remained positive, which would not be likely, had the two electrodes been switched.

The distinction between subaortic stenosis and pulmonic stenosis is not merely academic, but rather a therapeutic, as well as a prognostic one: these two disease entities may differ in the level of life-threatening risks they carry, as subaortic stenosis also compromises myocardial coronary perfusion while pulmonic stenosis does not. Another difference in the risks involved includes the fact that canine patients with **sub-aortic stenosis** are typically much more predisposed to developing secondary infection of the valve cusps, leading to fatal infective endocarditis, than do patients with pulmonic stenosis.Also, while they can both be (at least temporarily) palliated pharmacologically using the same medications (ideally achieving beta-adrenergic-blockade to minimize the systolic pressure-gradient across the stenosis), only pulmonic stenosis can be practically addressed using a cardiac catheterization or even a surgical procedure, designed to achieve valvuloplasty in which the stricture is at least partially relieved.

Of importance is that the diagnosis of pulmonic stenosis could be reached in this patient solely based on cost-effective (not to call them "low-tech") means, including history, physical examination, and an ECG strip, with no dependency on more sophisticated technologies such as an echocardiogram. Any clinician with a good understanding of both cardiac physiology and the basics of electrocardiography should be able to reach such a diagnosis under these specific circumstances, even in a minimally equipped environment.

Lastly, another important lesson is to avoid only being familiar with Lead-II: if one only relies on a single ECG-lead, one is also highly likely to miss an opportunity to identify anything from a subtle technical problem such as an electrode switch between two limbs, to an important, quite common disease

such as pulmonic stenosis, which can be life-threatening on the one hand, but also amenable to low-risk and effective palliation on the other hand.

REFERENCES

1. Hill JD. Electrocardiographic diagnosis of right ventricular enlargement in dogs. J Electrocardiol 4:347, 1971.
2. Trautvetter E, Detweiler DK, Bohn FK, et al. Evolution of the electrocardiogram in young dogs with congenital heart disease leading to right ventricular hypertrophy. J Electrocardiol 14:275, 1981.
3. Modification from Sisson D. and Oysma M.

FIGURES

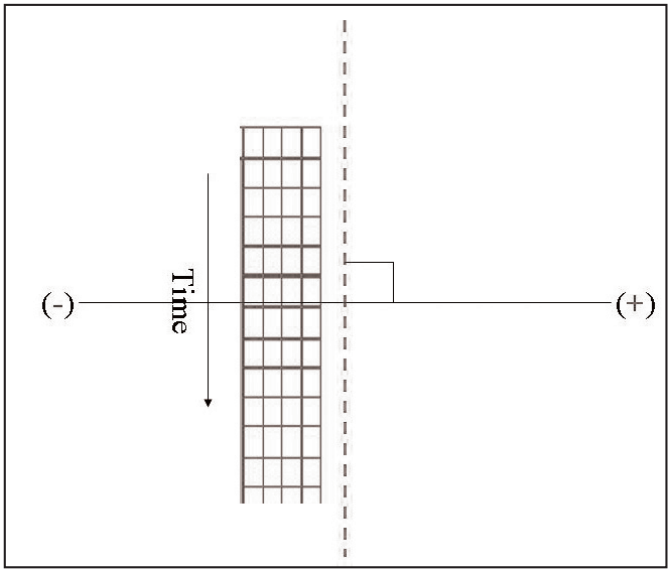


Figure 1:
The tracing on ECG paper represents electrophysiological events occurring through time, in a direction that is in right angles to the imaginary line connecting the two electrodes of each lead. Positive deflections represent propagation towards the positive electrode and negative deflections reflect propagation away from it.

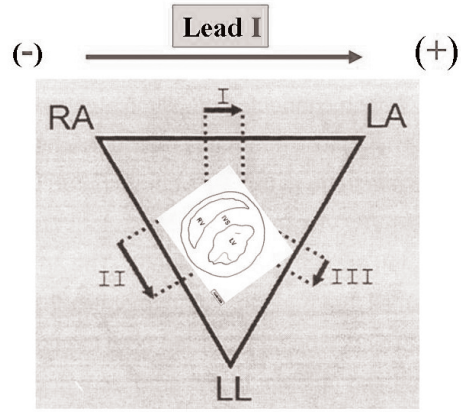


Figure 2:
Lead I configuration in the "frontal" plane, which is analogue to the ventro-dorsal plane traditionally used in radiography; RA, Right "Arm"; LA, Left "Arm"; L, Left Leg.

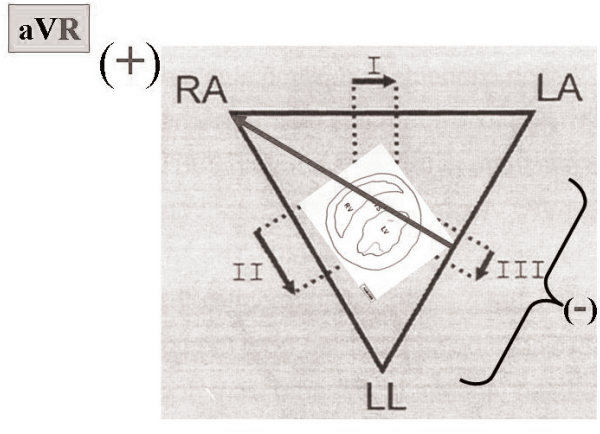


Figure 3:
Lead aVR (Augmented-Unipolar-Right) configuration in the "frontal" plane, which is analogue to the ventro-dorsal plane traditionally used in radiography. RA, Right "Arm"; LA, Left "Arm"; L, Left Leg.