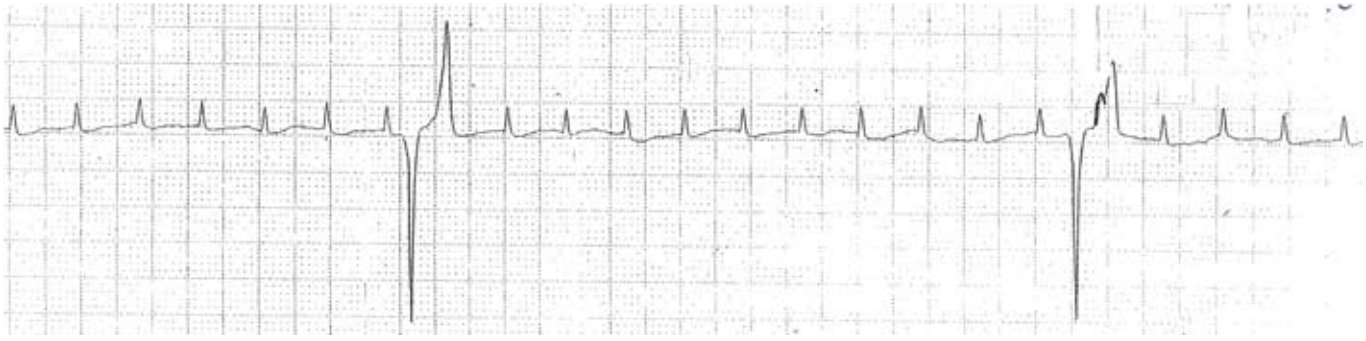
CARDIOLOGY

QRS IS NOT COMPLEX!

Ohad, D.
Cardiac Diagnostic Unit
The Koret School of Veterinary Medicine
The Robert H. Smith Faculty of Agriculture, Food and Environment
The Hebrew University of Jerusalem, Israel

Fig. 1:



HISTORY

The ECG strip in **Figure 1** was recorded from a 4 year old female spayed Boxer dog with a history of excitement-related and exertion-related syncope of which frequency has been progressive over the past several days. The strip was recorded in Lead-II, at a sweep speed of 25 mm/second and at a voltage calibration of 10 mm/mv.

What is your electrocardiographic differential diagnosis? What is your final diagnosis, and why?

Determine your diagnosis and turn the page.

ANSWER

Four basic kinds of deflections are seen on this strip:
- A 4 mm (0.4 mV) positive deflection, which is the most abundant morphology (labeled "A" in **Figure 2**).
- A 26 mm (2.6 mV) negative deflection, occurring twice (labeled "B" in **Figure 2**)
- A 16 mm (1.6 mV) positive deflection, occurring once (labeled "C" in **Figure 2**)
- A 10 mm (1.0 mV) positive deflection, occurring once (labeled "D" in **Figure 2**).

One of the most important and earliest steps in analyzing an ECG strip should be the determination of whether the rate is normal, too fast, or too slow. The strip is 190 mm long, and given its reported sweep speed its entire duration is 7.6 seconds. Throughout this period of time, 24 positive deflections have been recorded. For convenience, one can calculate the cycle rate based on the first 6 seconds only, which would be 150 mm (or 30 large squares from the left). Because, by definition, a single cardiac cycle is the interval between two consecutive and identical deflections, we should omit the first positive deflection from the left when calculating the cycle rate based on these data. We can then multiply the number of all other positive deflections recorded through the first 6 seconds, by 10. The calculated cycle rate would therefore be 17.5*10=**175 per minute**.

When considering the **differential diagnosis** list, there are **two theoretical possibilities** to interpret the positive deflection which is most abundant on this strip, labeled "A" in **Figure 2**. It can either be **1)** a **small R-wave** (representing ventricular depolarization), in which case the basic heart rate is 175/min, as in **sinus tachycardia**. Sinus tachycardia with highly regular low amplitude R-waves can, for example, reflect a condition such as cardiac tamponade. If sinus tachycardia is indeed the case, then the combination of the negative and larger positive deflection reflects an ectopic ventricular focus, potentially generating two ventricular premature complexes (**VPCs**) in which the negative deflection is a deep **S-wave** (ventricular depolarization) and the positive one is a **T-wave** (the end of which reflects the end of ventricular repolarization). If this is the correct interpretation, the lack of other visible deflections such as P or T-waves (especially in the presence of a tachycardia documented only by a single lead) does not necessarily mean there is no atrial depolarization or ventricular repolarization taking place. **2)** a **normal P-wave** (atrial depolarization), in which case the combined S-T complex would reflect an extremely **slow ventricular escape rhythm** occurring every 98 mm, or (at the reported paper speed) once in 3.8 seconds, which is a rate of only **16 ventricular cycles per minute**.

Note that the time-interval between each two consecutive small positive deflections (labeled "A" in **Figure 2**) is constant across the entire strip. Also note that the time-interval between the onset of the small positive deflection (labeled "A" in **Figure 2**) preceding the deep negative deflection (labeled "B" in **Figure 2**), and the onset of that same deep negative deflection itself, is not only different between the two consecutive occasions it can be recorded (shorter in the first relative to the second), but also different (shorter in both occasions) than the time-interval between each two consecutive small positive deflections.

This is not enough to determine which of the two differential diagnoses is correct, as it may fit them both: if the **sinus tachycardia** option is the correct one, then this shorter and inconsistent interval is simply compatible with the **prematurity of the two assumed VPCs** (which, by the way, do not necessarily have to occur at the exact same interval following the preceding sinus complex). If the **bradycardia** option is the correct one, on the other hand, then the short and inconsistent interval is highly compatible with a **third degree ("complete") atrio-ventricular (AV) block**, where there is absolutely no conduction through the AV-node. Consequently, there is no causative relationship (and therefore no constant interval) between any of the P-waves and the **escape** QRS-T complexes that happen to immediately follow them, whether the latter ones are supra-ventricular or ventricular in origin.

How, then, do we determine which of these two differential diagnoses is the correct one? Why is it even important that we do, anyway?

Ideally one should correlate these findings with the patient signalment, history, and physical examination findings. A severe bradycardia of 16 bpm would be more consistent with the provided history of progressively frequent syncope than would sinus tachycardia of 175 bpm. However, even in the absence of any history-related information, one can still make progress with this ECG interpretation and decide which of the two options is correct, based on the following:

Just following the second deep negative **S-wave**, and superimposed on the upstroke component of the following tall, positive ("D" in **Figure 2**) deflection which would be a **T-wave** for any and both of the differential diagnoses, is another small positive deflection which is very similar to all other small positive "A" deflections in **Figure 2**. If one carefully measures the constant interval between each pair of consecutive small positive deflections, one will notice this specific superimposed deflection occurs at the exact same interval from both small positive deflections flanking it. This would make it a likely true, regularly repetitive electrophysiological event, rather than a random artifact which was only recorded once.

Assuming this deflection is a **small R-wave** (as in **Differential Diagnosis #1**), the ventricular depolarization it reflects would necessarily have to occur before the ventricles reached the end of their absolute refractory period resulting

from the last S-wave (and still during the upstroke of the T-wave), which is electro-physiologically impossible.

On the other hand, assuming the small positive deflection is actually a **P-wave** (as in **Differential Diagnosis #2**), then the atrial depolarization it reflects is simultaneous to the ventricular repolarization but cannot interfere with it. These two events are anatomically and electro-physiologically separated from and "blind to" each other, as would be expected in a true **("complete") 3rd degree AV-block**. In this sense, it is indeed a graphical superimposition rather than a true interaction between two ("electrically" opposite) processes located at two different, and "electrically" separated regions of the heart.

Note that following the first (previously labeled "B") deep negative S-wave, too, there is actually a **P-wave** (labeled "C" in Figure 2) recorded simultaneous to the very peak of the **T-wave**, rendering it taller than the second ("D" in Figure 2) **T-wave** due to a similar graphical superimposition (**Figure 3**). Likewise, this specific superimposed **P-wave** occurs at the exact same P-to-P interval as that of both P-waves flanking it.

This interval, by the way, is both short (at a rate of 175 per minute) and highly regular. Both high rate and excessive regularity are manifestations of an extremely high sympathetic tone. This tone, in turn, is triggered by the very low cardiac output generated by the exceptionally slow (16/min) **ventricular escape rhythm**. At times of an increased metabolic demand, such as exertion or excitement, an extremely low cardiac output would especially compromise tissue perfusion-pressure, even that of highly preserved tissues such as the Reticular Formation of the brain stem, which helps maintain consciousness.

Between the two differential diagnoses, it should be clear now why this trace is compatible with **a 3rd AV-Block with a slow ventricular escape rhythm**, rather than with sinus tachycardia and occasional VPCs.

The reason why a correct diagnosis is crucial in a case like this is this: if one mistakes the ventricular complexes for VPCs and pharmacologically treats them as such, one will suppress this patient's last resort, and iatrogenically induce cardiac arrest followed by death. Rather, the ultimate approach should be pacemaker therapy which would not only improve quality of life by far, but will also prolong life expectancy by several (and potentially even many) years.

REFERENCES

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Fig. 2:

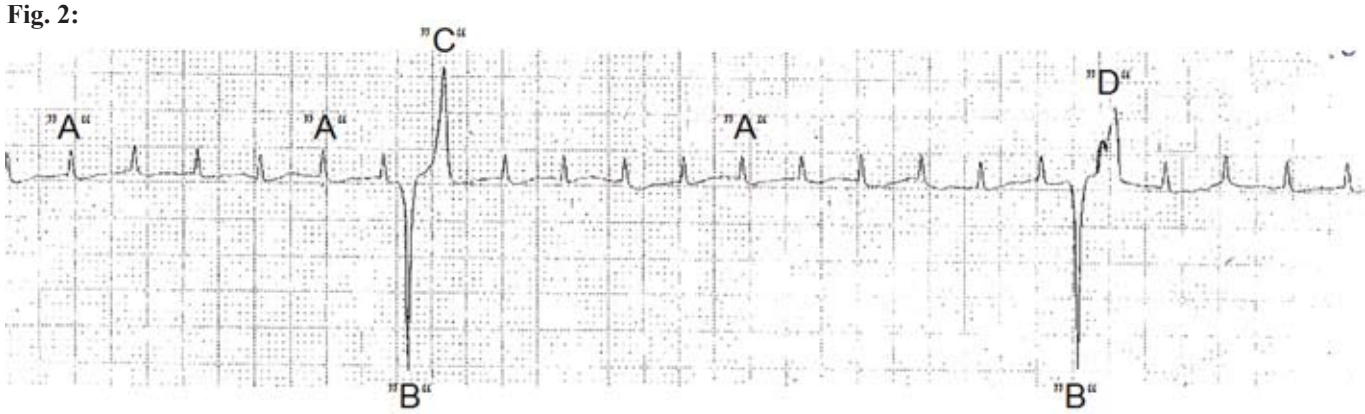

Fig. 3: