Arrhythmia in a child with ventricular preexcitation: which is the diagnosis? Dr. Pedro Case

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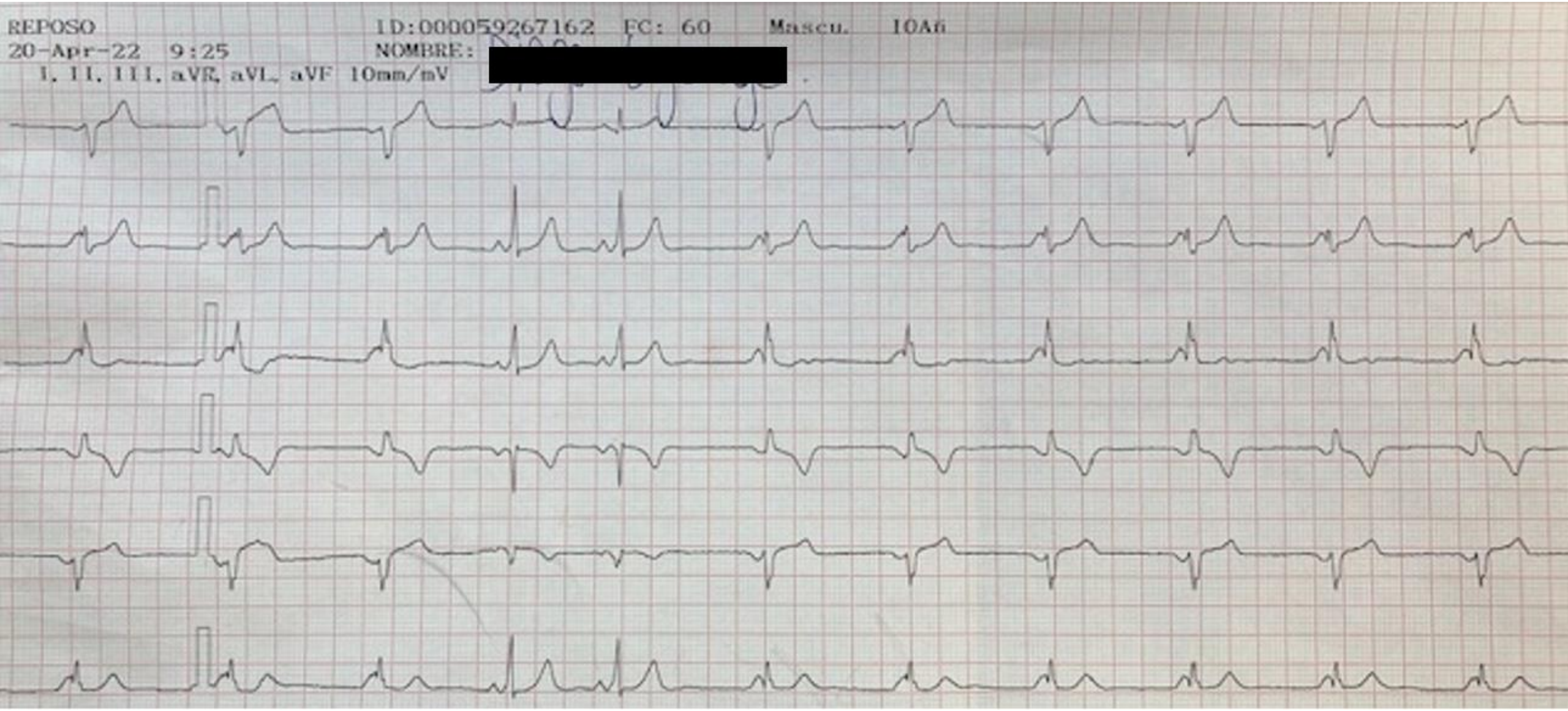
Asymptomatic child.

The RxTx shows a heart of normal size and position.

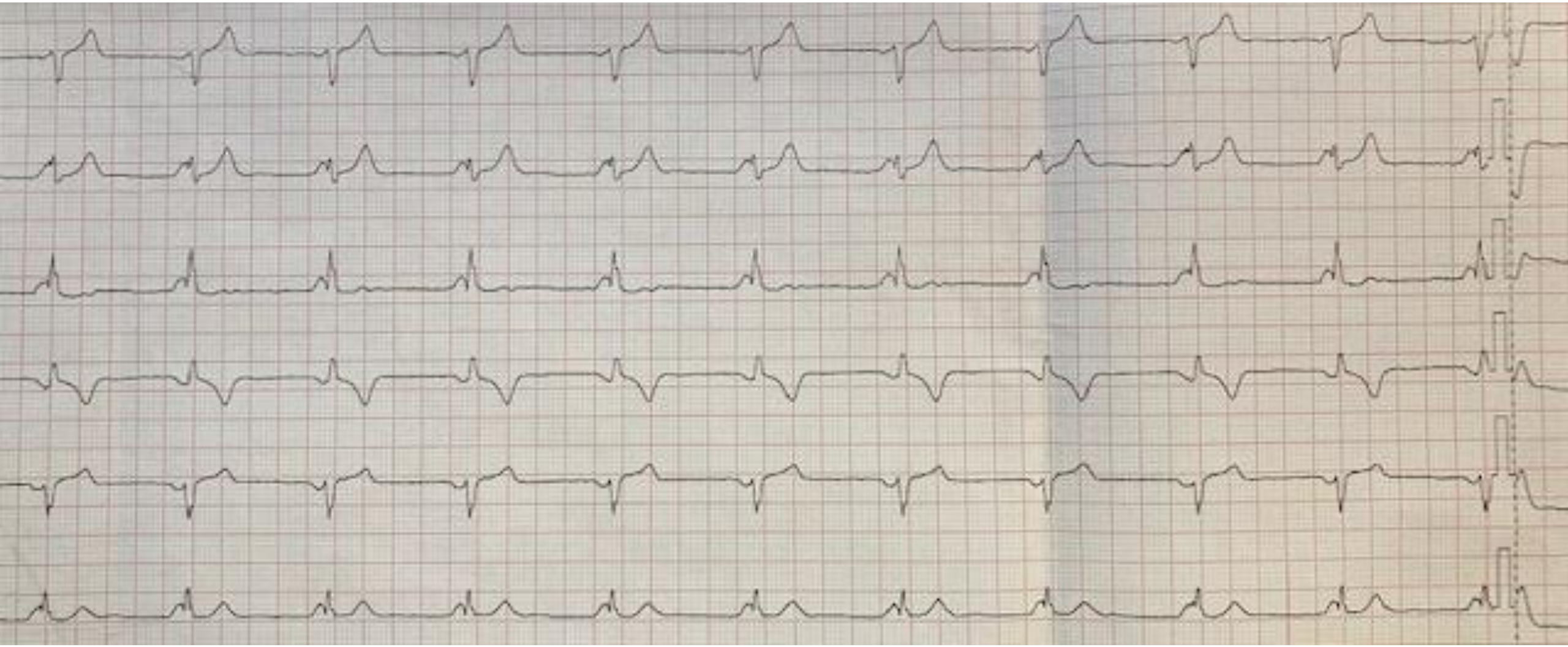
Echocardiogram shows normal cardiac structure and function.(Absence of structural heart disease)

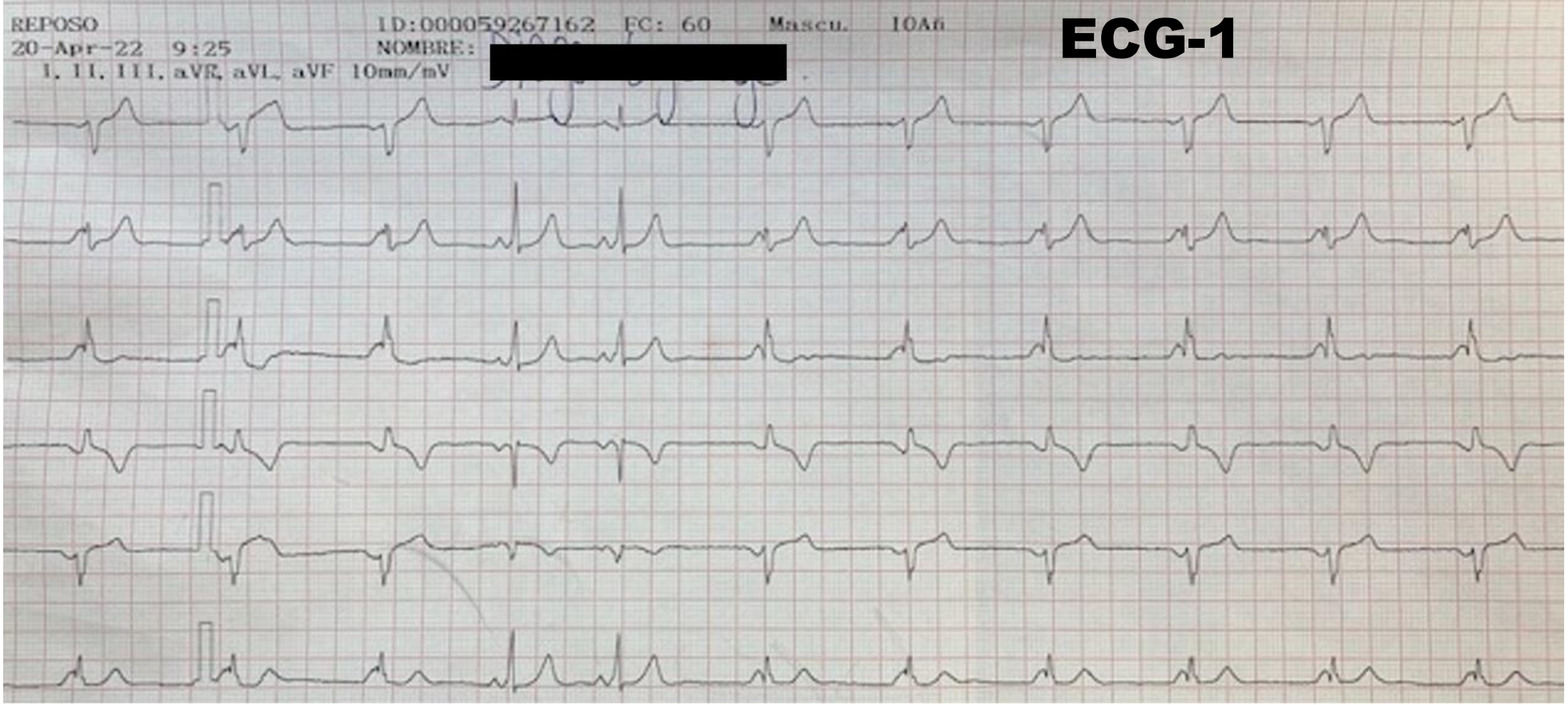
The diagnosis of WPW with no doubts. And the complexes of the first and two beats, except complexes 3 and 4, what do you think about the diagnosis of the first ECG?

ECG-1


REPOSO
20-Apr-22 9:25
I, II, III, aVR, aVL, aVF 10mm/mV
ID:000059267162 FC: 60 Mascu. 10A6
NOMBRE:

ECG-2





The first is labeled I, II, III, aVR, aVL, aVF, and the 2nd (not labeled) looks like the same 6 leads. Since I don't see precordial leads I can't presume this is a left sided accessory pathway (AP) . There is sinus arrhythmia that results in varying degrees of preexcitation. The 'degree' of preexcitation varies with the PP interval with less preexcitation as the heart rate picks up (beats 4-5 on the first ECG). Perhaps with slower heart rates (more vagal tone slowing AV node conduction and sinus rate) the P wave gets through the accessory pathway preferentially with more preexcitation. I think the PR gets shorter and preexcitation is greater at the slower heart rates, and I see this in both ECGs.

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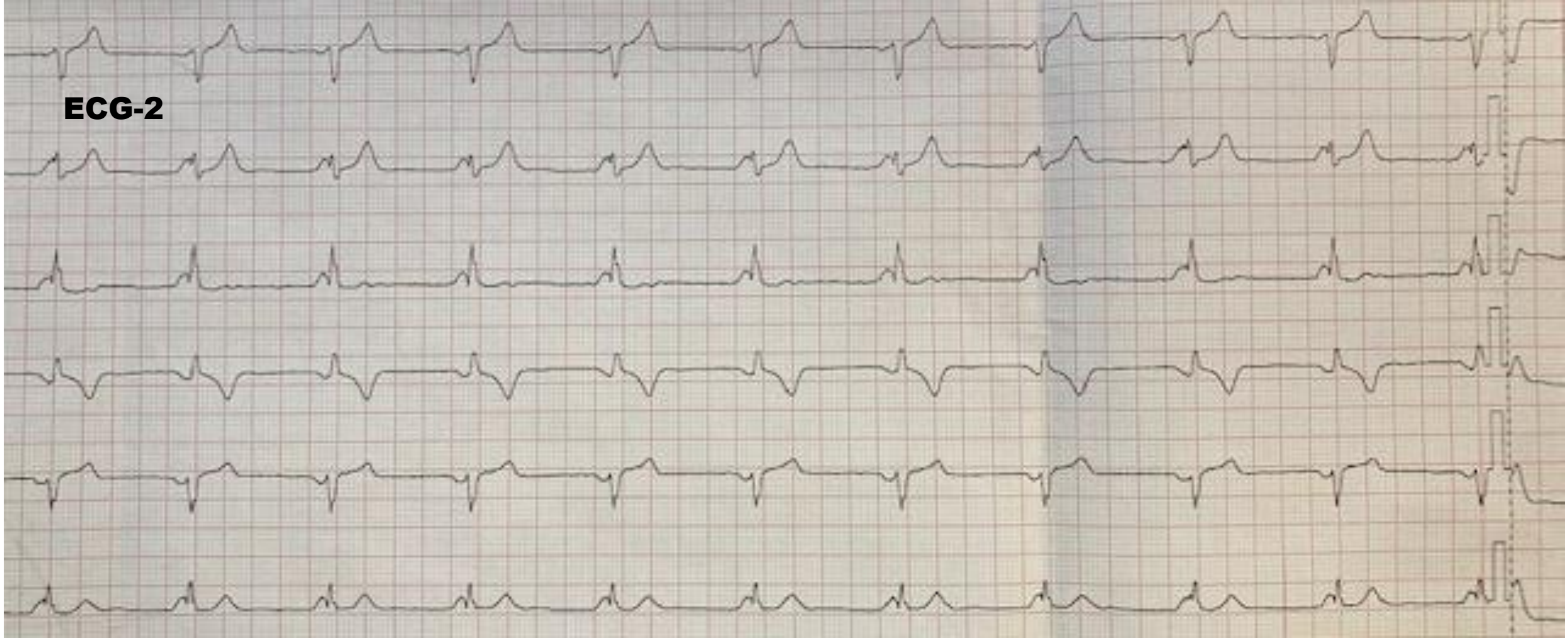
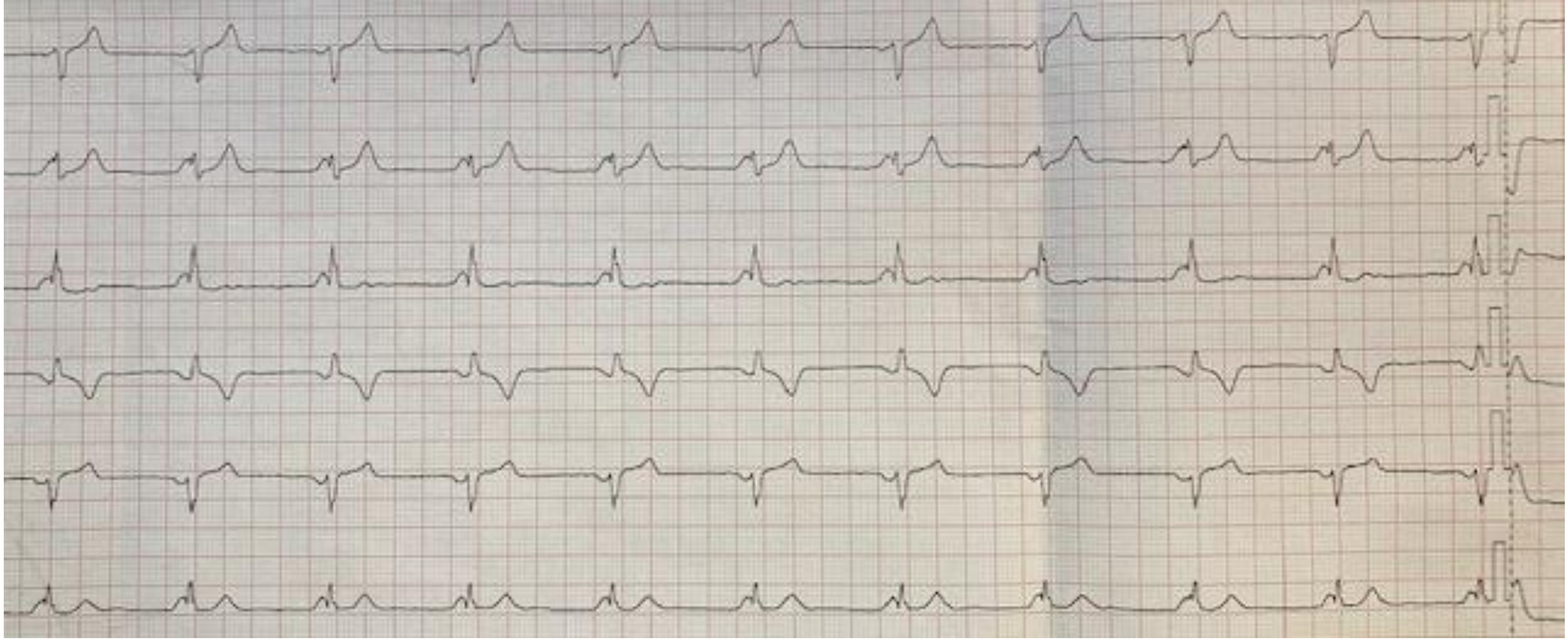
“Concertina” effect or Öhnell “accordion” phenomenon Öhnell or intermitent pre-excitation

Theoretical consideration: This is the progressive shortening of the PR interval and the concomitant prolongation of the QRS complex and vice-versa, observed in successive beats. It is due to a progressive greater or lesser percentage of ventricular activation by the AP, in comparison to the normal pathway, which is translated into a QRS complex with greater or lesser fusion degree. The phenomenon occurs by variations in the amount of ventricular muscle activated early through the parallel AP. The variations of vagal tone that increase or decrease the refractory period of the AV node, may manifest by the “concertina” effect, widening or shortening QRS complex duration successively. **Fusion beats:** QRS complex that is the result from two wave fronts: one preceding the anomalous AP in a parallel fashion, which activates the ventricles early (responsible for the δwave); and one preceding the normal pathway of the His node, more delayed. In others words, a fusion beat occurs when a supraventricular and a ventricular impulse coincide to produce a hybrid complex. The greater or lesser degree of fusion determines the greater or lesser duration of the QRS complex. The greater the component of the ventricular mass, activated through the AP, the greater the duration of the QRS complex.



Sinus rhythm with a very short PR interval (< 120 ms), wide QRS complexes with a slurred upstroke of QRS complexes: the delta wave, dominant R wave in V1 suggesting a left-sided Accessory pathway , sometimes referred to as "Type A" WPW, tall R waves and inverted T waves in V1-3 mimicking right ventricular hypertrophy (RVH). These changes are due to WPW and do not indicate underlying RVH, negative delta wave in aVL simulating the Q waves of lateral infarction — this is referred to as the "pseudo-infarction" pattern



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Take-Home Points (1)

- **Wolff-Parkinson-White syndrome refers to the electrocardiographic findings of WPW pattern and a tachyarrhythmia involving the accessory pathway.**
- **The degree of preexcitation in WPW pattern depends on the degree of conduction via the AP relative to the AV node, which depends on the autonomic tone affecting the AV node.**
- **An increase in parasympathetic tone can lead to a decrease in heart rate and slower AV nodal conduction, which can result in shorten**

1. **Anoop Muniyappa 1, Arvind Nishtala 1, Nora Goldschlager 2 3The Concertina Case of Ventricular Preexcitation JAMA Intern Med. 2017 Dec 1;177(12):1842-1844. doi: 10.1001/jamainternmed.2017.4857.**

A healthy man in his early 40s presented to the ED after a second syncopal episode in 2 weeks. Both episodes occurred without prodromal symptoms, tonic-clonic movements, tongue biting, or incontinence, and the patient felt normal afterward. He reported having rhinorrhea and sinus congestion, as well as cough, starting shortly before his first syncopal episode. He did not have a personal or family history of heart disease, syncope, presyncope, seizures, or SCD. Vital signs on admission were normal. Cardiac examination was notable fora normal jugular venous pressure and waveforms, a normal cardiac rhythm and rate, non displacement of a normal cardiac apical impulse, normal heart sounds and no murmurs, rubs, or gallops. The remainder of the physical examination was unremarkable. A 12-lead ECG was obtained (Figure 1). The ECG demonstrates sinus rhythm at a rate of 82 bpm, a short PR-interval of 128 milliseconds, broad QRS duration (132 ms), and δ wave measuring 60 ms, consistent with ventricular preexcitation (WPW] pattern. This wide QRS complex with a δ wave represents a fusion beat via the AP and the AV node. Given the concern for syncope due to a WPW-related tachyarrhythmia, the patient was admitted and scheduled for an EPS. Overnight, the on-call house officer was called by the telemetry technologist for widening QRS and possible new bundle-branch block. Another ECG was preformed (Figure 2). Questions: What is the difference between the 2 ECGs, and are these findings abnormal?

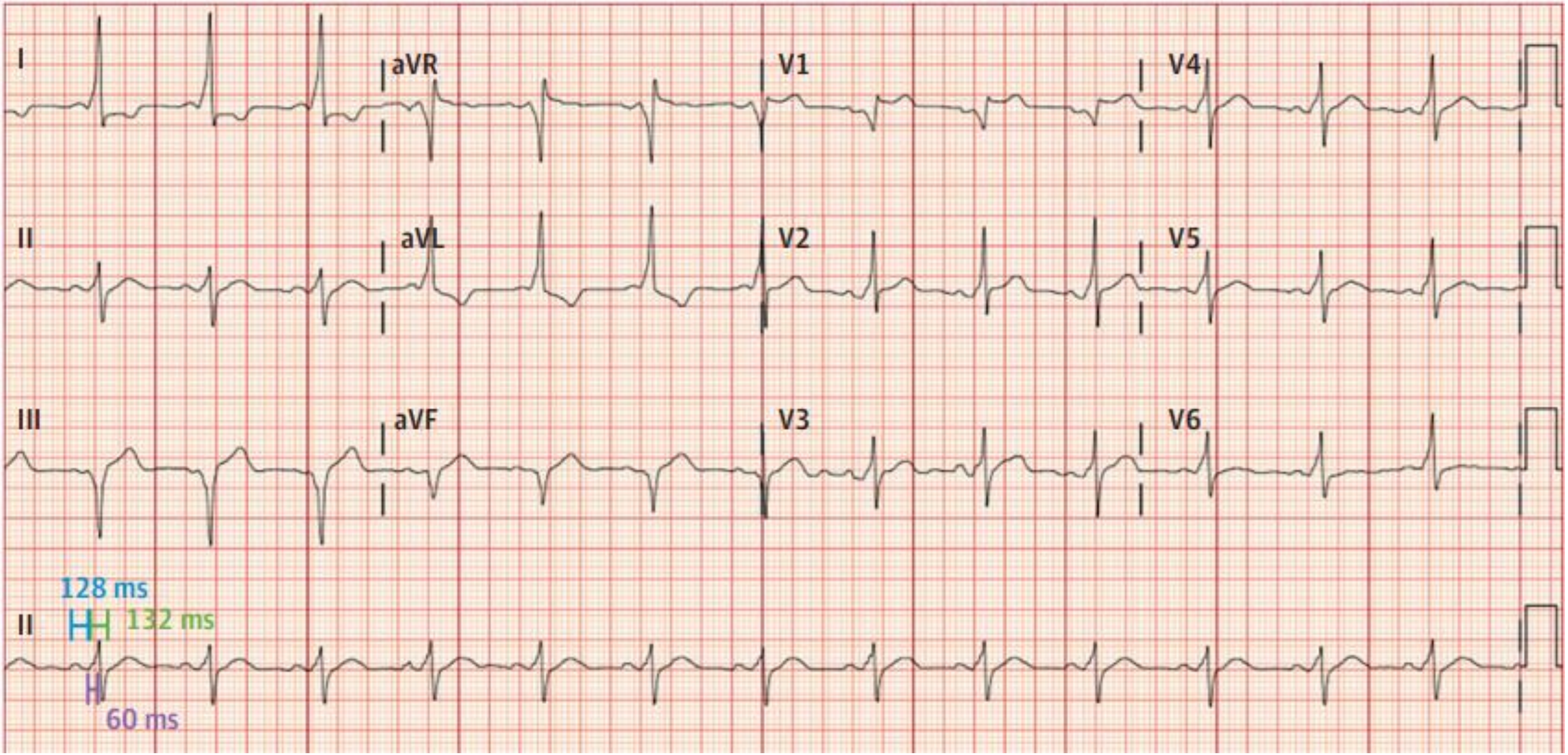


Figure 1 On this 12-lead ECG recorded on admission of a man in his 40s, the blue bar demarcates the PR interval (128 milliseconds), the green bar demarcates the QRS duration (132 milliseconds), and the purple bar demarcates the Δ wave (60 milliseconds).

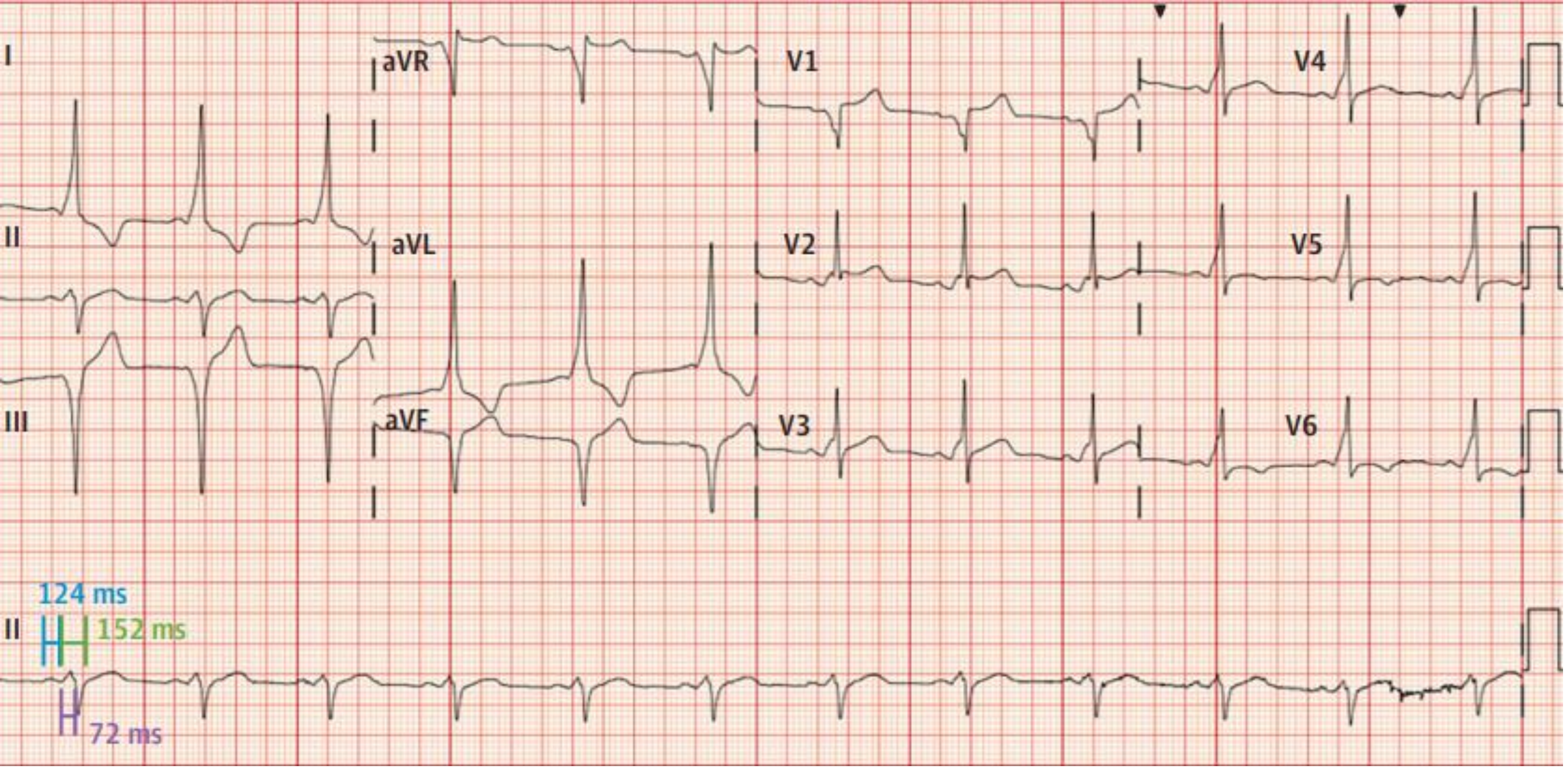


Figure 2 This 12-lead ECG was recorded overnight after a house officer was called for widening QRS, possible bundle-branch block. The blue bar demarcates the PR interval (124 ms), the green bar demarcates the total QRS duration (152 ms), and the purple bar demarcates the δ wave (80s).

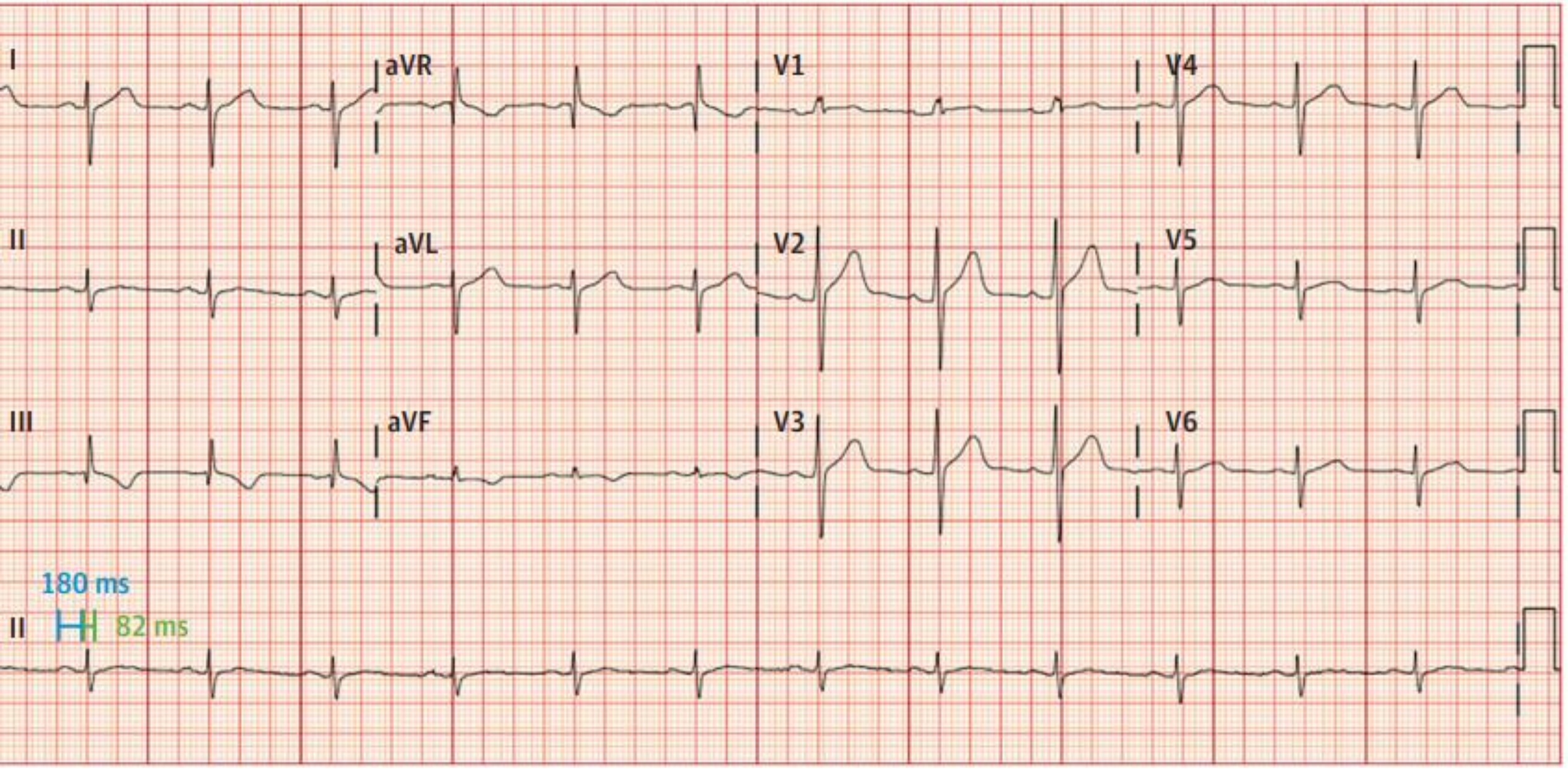
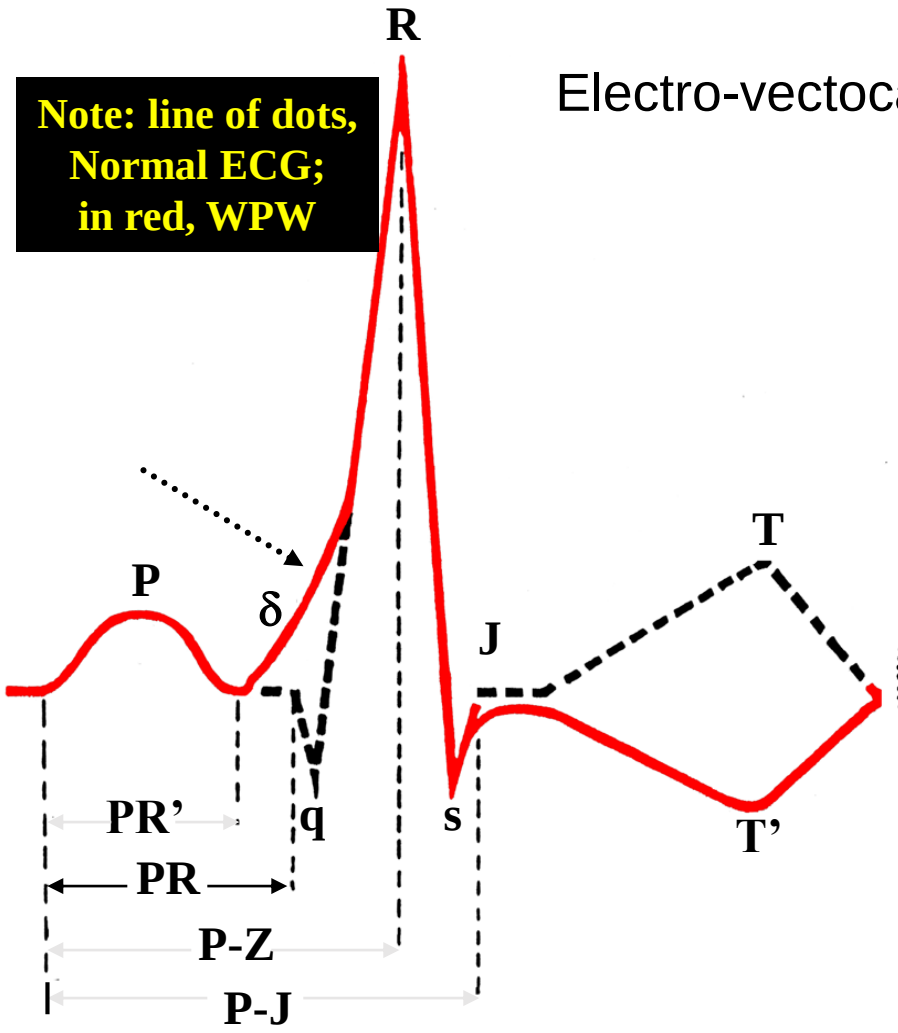


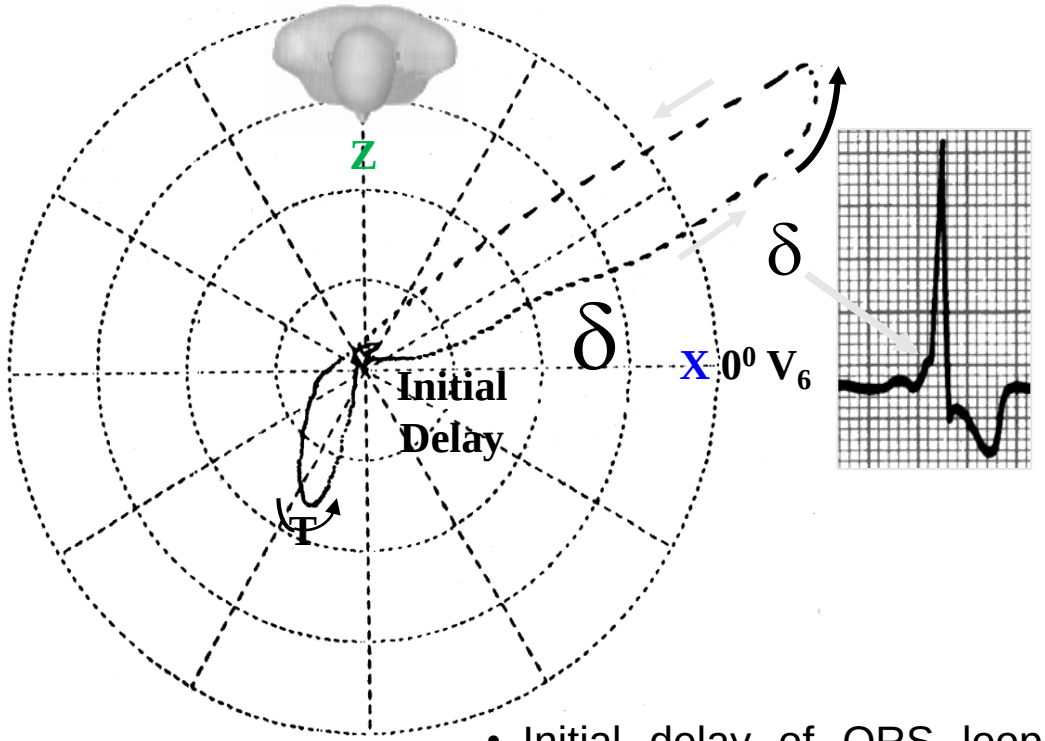
Figure 3 This 12-lead ECG was recorded following slow pathway modification and accessory pathway ablation. The blue bar demarcates the PR interval (180 ms) and the green bar demarcates the QRS duration (82 ms).

Clinical Course The patient had an EPS performed 4 days later that showed an antegrade-only conducting AP in themid-septum,with an effective refractory period of 330 ms and no evidence of inducible atrioventricular reentrant tachycardia. However, he was found to have inducible typical atrioventricular nodal reentrant tachycardia and underwent successful slow pathway modification and AP radiofrequency ablation. His postablation ECG (Figure 3) shows a normal, longer PR interval and a narrow QRS complex without evidence of preexcitation, demonstrating successful AP ablation. It also shows right axis deviation with right ventricular hypertrophy (or LPFB?). As the patient did not have a prior ECG for comparison, an outpatient workup for right ventricular hypertrophy was planned. Discussion: Preexcitation is the activation of the ventricular myocardium by an atrial impulse earlier than it would be activated if the impulse had been conducted via the AV node–His-Purkinje system.2 In WPW pattern, ventricular preexcitation occurs due to conduction of the atrial impulse down an aberrant atrioventricular connection that bypasses the AV node, known as an AP. This results in the classic ECG findings of WPW pattern—a short PR interval and wide QRS complex with a slurred initial deflection, known as a δ wave and followed by ST segment–T wave changes, generally directed opposite the major δ wave and QRS complex

WPW ECG/VCG correlation

Electro-vectocardiographic criteria for WPW type preexcitation.





- **PRi or PQ:** since the onset of P up to the onset of QRS. It represents the time the stimulus takes to go from the SA node until reaching the ventricles: 120 ms to 200 ms.
- **PZ:** distance between P wave onset until R apex: 150 to 230 ms.
- **PJ:** distance between P wave onset until j point: 180 to 260 ms.

- Initial delay of QRS loop: delta wave.
- T-loop opposite to QRS loop

Preexcitation by Accessory pathway Kent, classical or WPW type: ECG-VCG criteria

1) Short PRi interval: <120 ms in adults and 90 ms in children;
2) Wider QRS complex: ≥100 ms 70% of the cases. 30% < 100 ms;
3) A slurring and slow rise of the initial upstroke of the QRS complex (delta wave). In another words, thickening or notch at the onset of QRS complex: Delta δ wave, duration 30 ms to 60 ms and voltage of up to 5 mm, which corresponds to early depolarization by ventricular mass.
    - Unaltered P-J interval (normal): 180 to 260 ms (slide 18);
    - Unaltered P-Z interval (normal): 230 ms (150 to 230 ms);
    - Alterations secondary to ventricular repolarization (ST-T): depending on aberrant depolarization;
4) ST segment–T wave changes, generally directed opposite the major delta wave and QRS complex
5) Frequent association with tachyarrhythmias (40% to 80% of cases): if they are absent, “WPW pattern;” if they are present, WPW syndrome: PSVT either orthodromic (90%), antidromic (10%), AF (20%), atrial flutter, or ventricular fibrillation.
6) Characteristic initial delay of QRS loop in the three VCG planes (Delta loop).
7) Pseudo-infarction pattern can be seen in up to 70% of patients – due to negatively deflected δ waves in the inferior / anterior leads (“pseudo-Q waves”), or as a prominent R wave in V1-3 (mimicking lateral infarction (ancient dorsal).