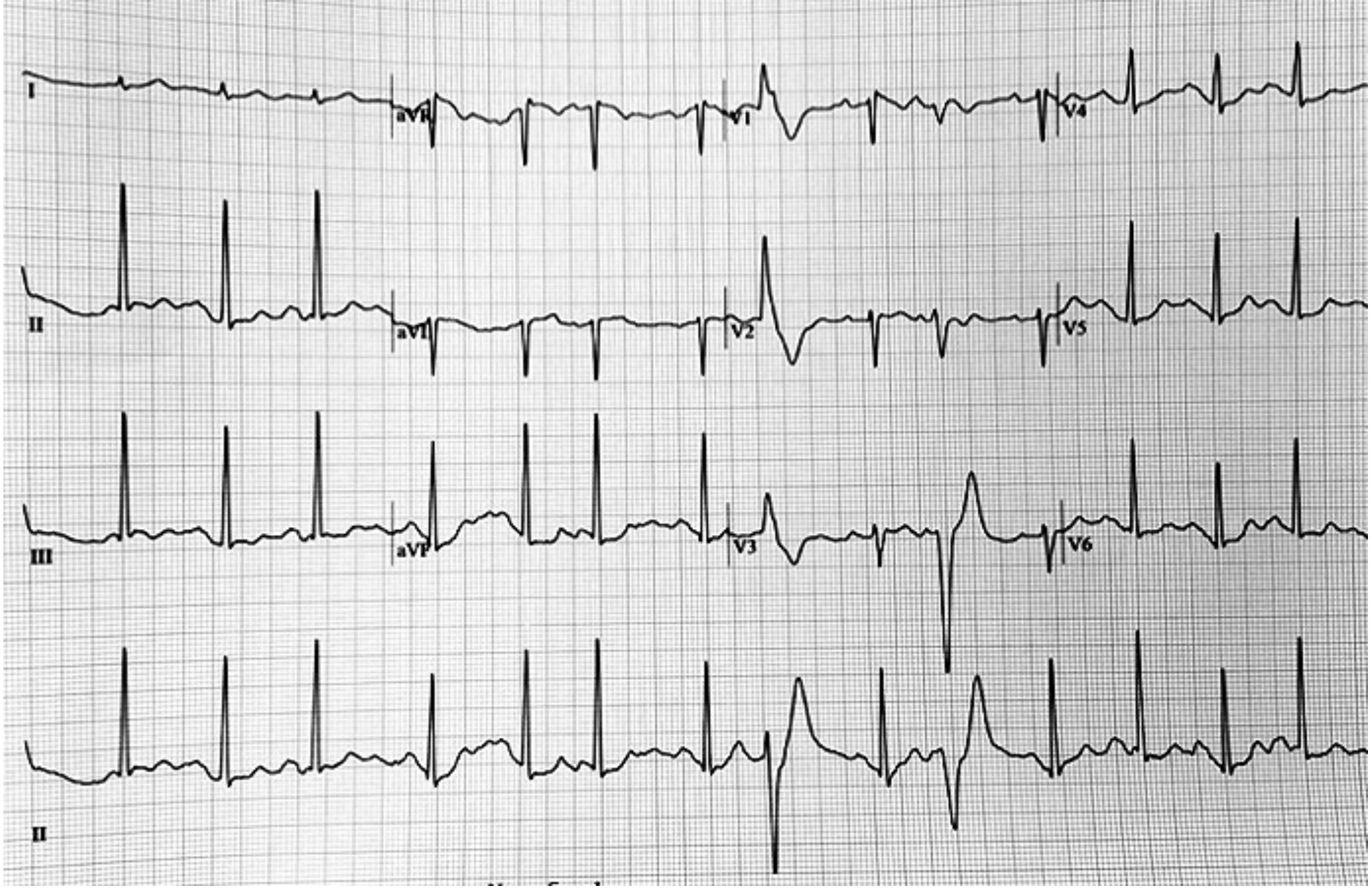
Senior athlete with exercise-induced tachyarrhythmia

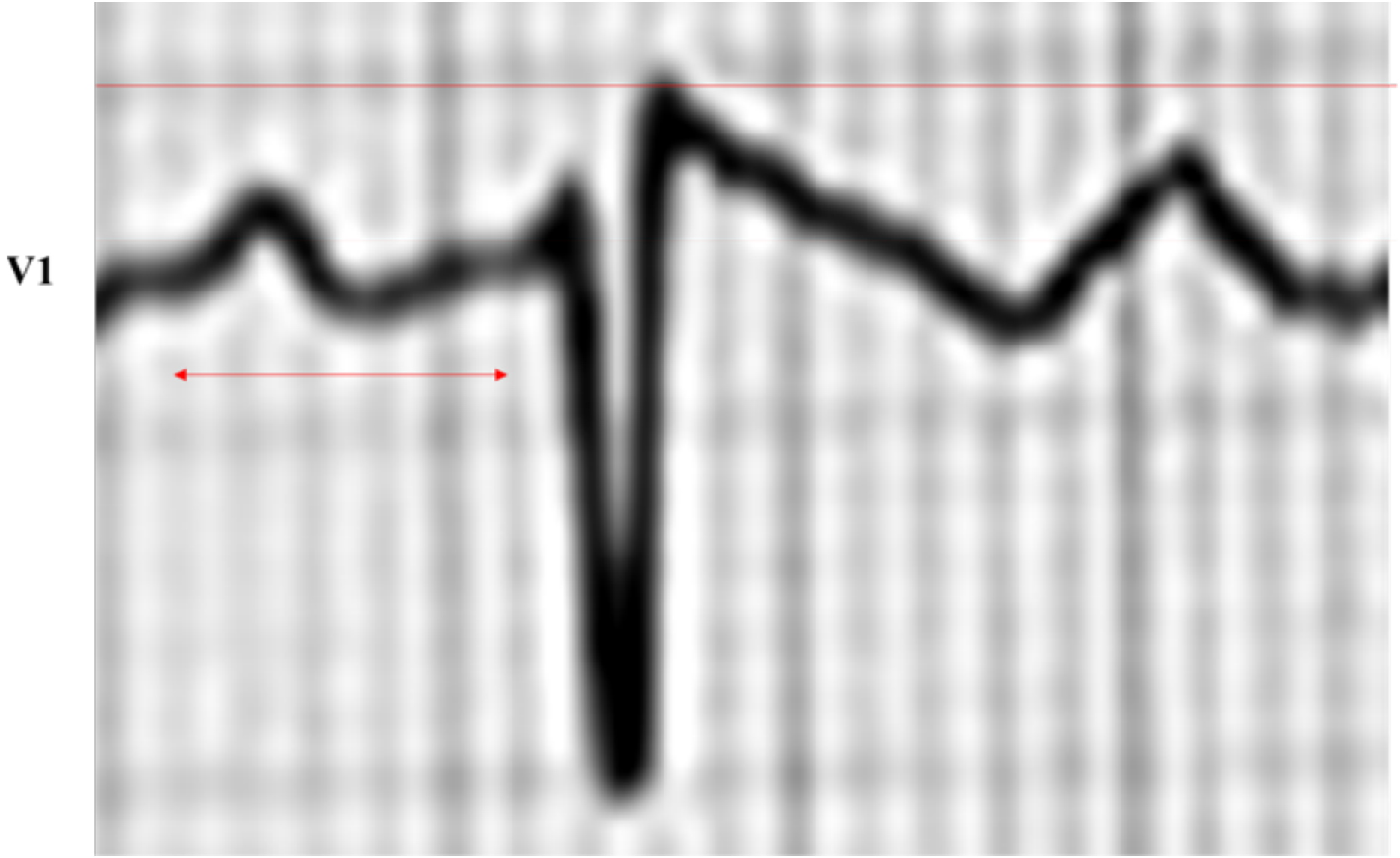
Dr. Andrés R. Pérez Riera

Male patient 65 yo. He carrier mitral valve prolapse (MVP) of anterior leaflet with mild mitral regurgitation. Sportsman o high-performance of mountain-bike mode. Asymptomatic and no history of sudden death in the family. During the test remained asymptomatic.

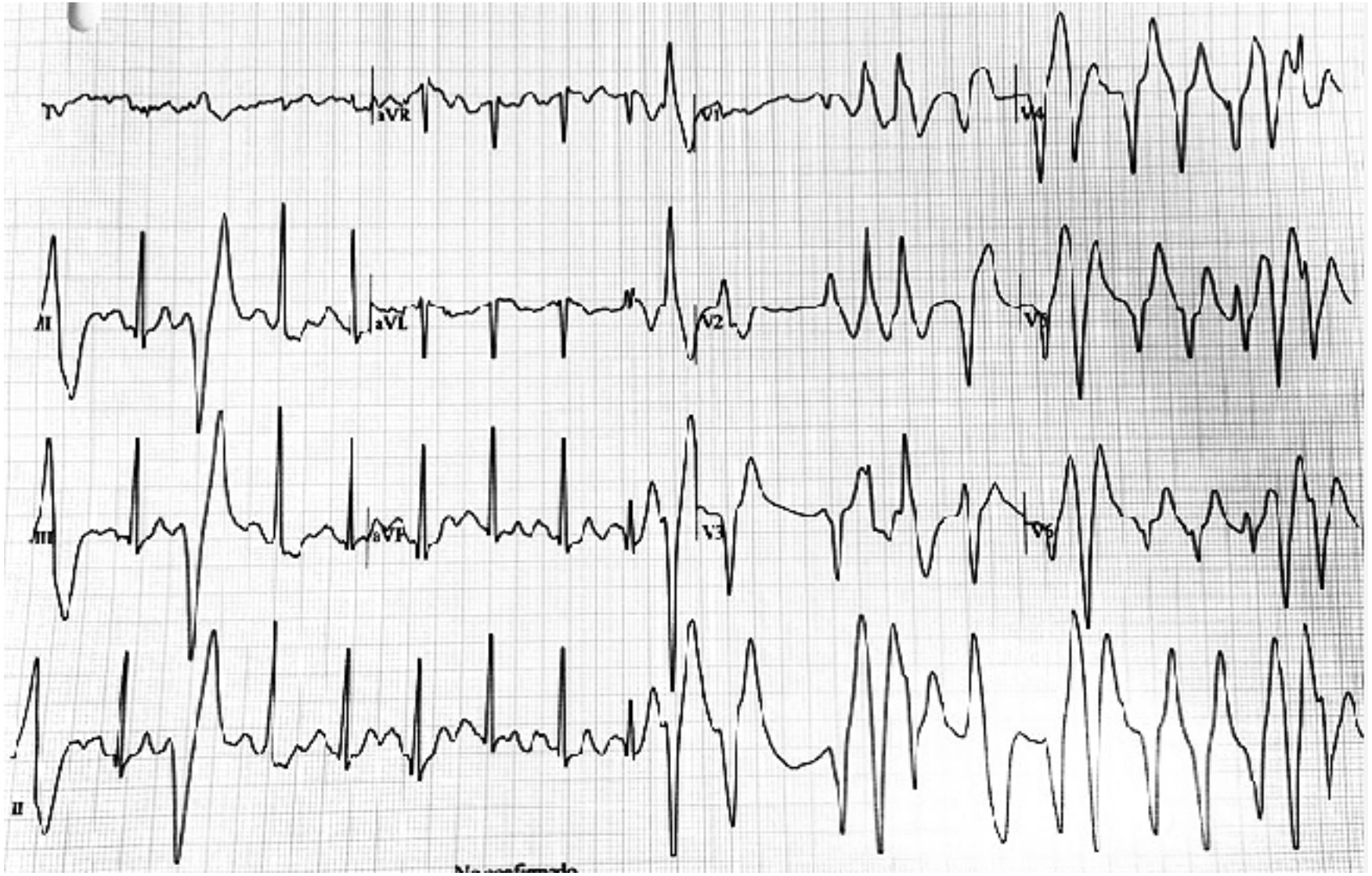
What is arrhythmia diagnosis ?
What is the proper conduct?



Inferolateral repolarization changes, polymorphic PVCs from left ventricle with R/T phenomenon

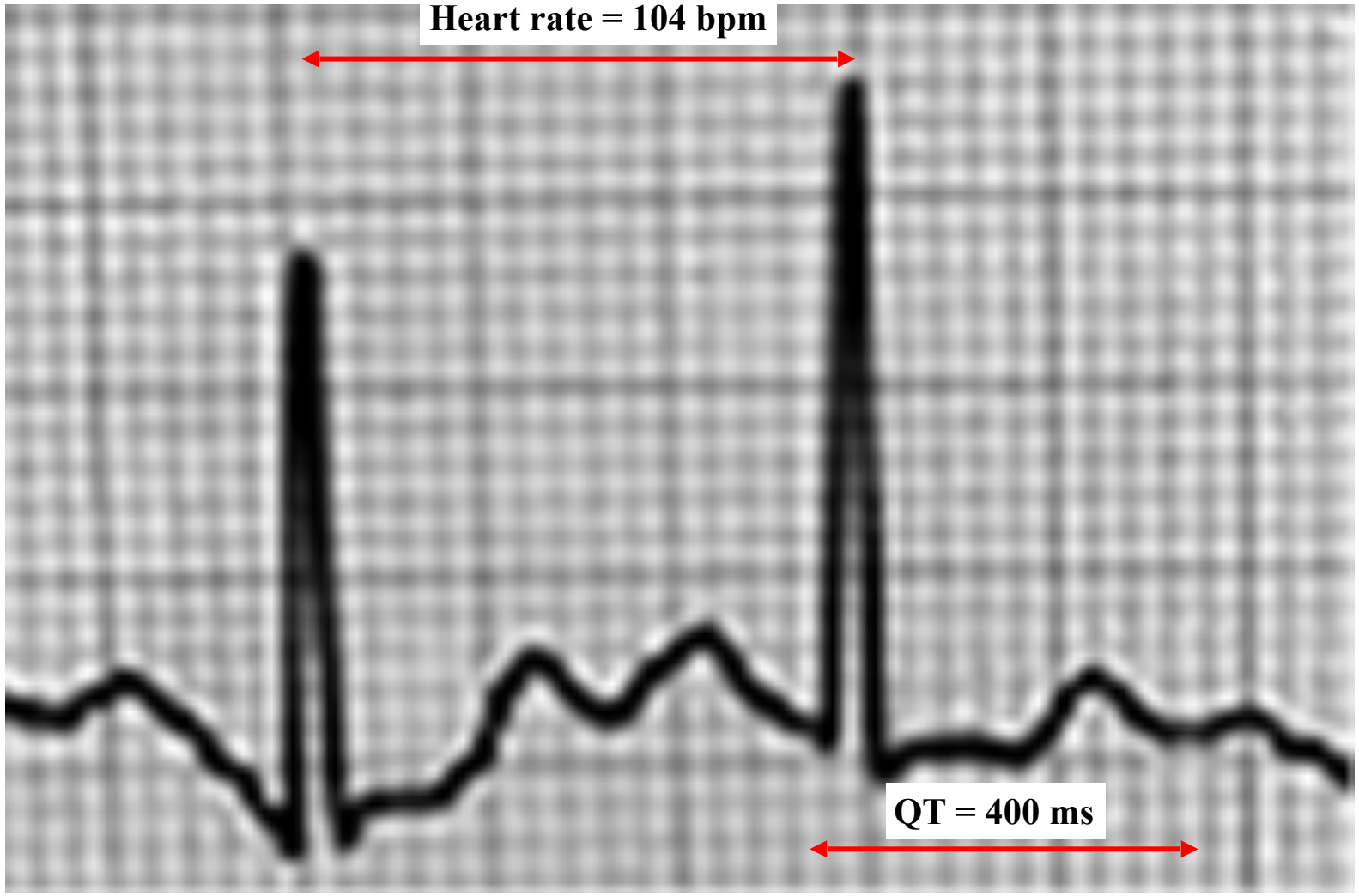


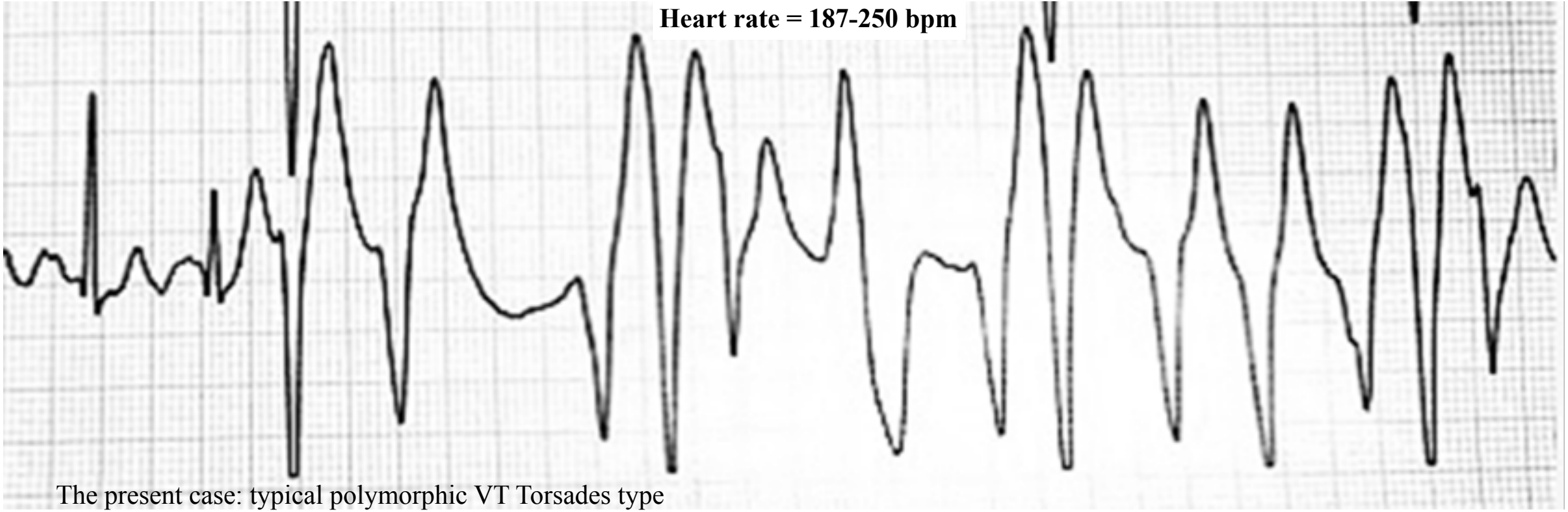
The presence of type 1 ECG Brugada pattern in V1 (J-point and ST segment elevation ≥ 2 mm followed by negative T-wave) points out that it is necessary to exclude the possibility of concealed forms of arrhythmogenic right ventricular cardiomyopathy/dysplasia (ARVC/D)? (**Corrado 1996; Corrrado 2001**).



Polymorphic PVCs with short couplet (R/T phenomenon) that degenerate into polymorphic ventricular tachycardia.

Heart rate = 104 bpm, QT = 400 ms. Normally, for heart rate = 100value of QT is 0.314 (lower limit 0.270, upper limit 0.358) for men. **Conclusion:** this patient has prolonged QT interval. Patients with MVP and sustained VT have higher age (the present case), more frequent syncopal attacks, longer QTc interval (present in this case).





The present case: typical polymorphic VT Torsades type

Polymorphic, polymorphous VT or atypical VT: Polymorphic or polymorphous VT (PVT) is recognized by a continuously changing QRS configuration form beat to beat, indicating a changing ventricular activation sequence and may precede development of VT. This may occur as part of the congenital long QT syndrome or acquired forms which are usually consequence of drug and /or electrolyte abnormalities (torsade de pointes: TdP) or other channelopathies, or may be because of reentry in a patient with structural heart disease. The "polymorphic" nature does not define an arrhythmia mechanism. In the TdP the coupling of the initial PVC is belatedly or telediastolic, the heart rate is high (from 200 to 250 bpm) and characteristically the axis of VT changes suddenly 180º. TdP: Rotation of the apex of QRS along the baseline. "swinging pattern" or "twisting appearance". Why is it not a short-coupled variant of torsade de pointes and normal QT interval, PVT verapamil sensitive or Leenhardt disease? Because we have LQT interval and the patient is a elderly man. It is a rare variant of polymorphic ventricular tachycardia (PVT), with unknown etiology. The entity is observed in young, healthy children and young adults (average: 34.6 years) and most probably covers several underlying electrophysiological abnormalities (**Coumel 1997**). The short-coupled variant of TdP is a polymorphic, polymorphous or multiform PVT with typical morphology of TdP: the QRS morphology shows alternating polarity in a modulating pattern, so that the complexes appear to be twisting around the baseline, observed in patients without organic heart disease, adverse drug effects, or electrolyte disturbances, which occurs

spontaneously and initiated by a very short-coupled coupling interval of the first premature ventricular complex (240 ms in average) in patients with normal QT interval. There are references in literature of electrical storm10 (ES) and intractable VF lifesaving with cardiopulmonary bypass or deep sedation followed by a combination therapy using verapamil and mexiletine. In these cases, the ECG pattern consisting of a prominent J wave in leads V3-V6 that disappears with the use of those drugs. The ES was evoked with autonomic receptor stimulation and a blockade test. The patient's frequent VF attacks were triggered by short-coupled premature ventricular contractions with RBBB morphology and left-axis deviation (**Durand-Dubief 2003**).

Mitral valve prolapse (MVP) is a common disorder that, in general, has a good prognosis. Rare occasions of sudden death have been reported in patients with MVP and it is presumed that the basis of sudden death is arrhythmic. Patients with MVP and malignant ventricular arrhythmias as rule are symptomatic: palpitations, presyncope, syncope or cardiac arrest, differently from the present case. A selected subset of patients with MVP, malignant ventricular arrhythmias, and mild mitral regurgitation are at risk of sudden death. Inferolateral repolarization changes, complex ventricular ectopy (**Vohra 1993**). Patients with sustained VT have higher age, more frequent syncopal attacks, longer QTc interval, more frequent negative T wave in inferolateral ECG leads, deeper ST deviations, lower oxygen consumption, more prominent left ventricular function impairment, more frequent polymorphic PVC's (more than 10/1000 ventricular complexes), paired PVC's and thicker anterior mitral leaflet than in patients with non-sustained VT. Non-invasive diagnostic methods could help to identify the patients with mitral valve prolapse at elevated risk for VT.

- Coumel P. Polymorphous ventricular tachyarrhythmias in the absence of structural heart disease. Pacing Clin Electrophysiol. 1997;20(8 Pt 2): 2065-7.
- Durand-Dubief A, Burri H, Chevalier P, Touboul P. Short-coupled variant of torsades de pointes with intractable ventricular fibrillation: lifesaving effect of cardiopulmonary bypass. J Cardiovasc Electrophysiol. 2003;14(3):329.
- Vohra J, Sathe S, Warren R, Tatoulis J, Hunt D. Malignant ventricular arrhythmias in patients with mitral valve prolapse and mild mitral regurgitation. Pacing Clin Electrophysiol. 1993;16(3 Pt 1):387-93.