A 56-year-old man came to the Pacemaker Clinic for his regular pacemaker control. He had experienced a syncopal episode in the previous week, while he was carrying heavy weight. He felt nauseated after recovering.

He had had a previous diagnosis of non-obstructive hypertrophic cardiomyopathy and the last echocardiogram performed in 2003 showed: IVS: 1.9; LV post wall: 1.0; LVDD: 4.8; LVSD: 2.6; LA: 5.0; Ejection Fraction: 46%. Due to a previous syncopal episode, an electrophysiology study was carried out in 2003, which showed: AH (nodal conduction): 155 ms; HV (distal conduction): 60 ms in conducted beats; 2:1 infrahisian block; non-inducible VT (with a protocol of 3 extra-stimuli). A cardiac angiogram showed normal coronary arteries. The patient was treated with 50 mg of Metoprolol, twice a day and subsequently, a dual-chamber permanent pacemaker was implanted (Kappa 931, Medtronic).

At the time of the consultation, the physical examination was unremarkable. His 12-lead ECG showed atrial sensing with ventricular pacing. The device was interrogated, showing several ventricular high rate episodes. A careful analysis of the stored intracardiac electrograms showed ventricular tachycardia (VT) with 2:1 ventriculoatrial conduction. The following presentation discusses the alternative diagnosis and clinical management in an unusual diagnosis of VT in the presence of non-obstructive hypertrophic cardiomyopathy.

**Discussion**

This is an interesting case that assesses the diagnostic possibilities of the new pacemakers. The diagnostic ability of the current pacemakers allows intracardiac electrograms to be stored for further analysis. The perfect symptom-rhythm correlation (syncope during the ventricular high-rate episode) was paramount in the clinical decision-making.

The analysis of the intracardiac electrograms revealed initiation of the tachycardia with a PVC (Fig. 1, black arrow), followed by a rapid ventricular rhythm at 320 ms. The ventriculo-atrial conduction (VA) is 2:1, thus supraventricular arrhythmias were unlikely (Fig. 2). The ventricular channel (Fig. 2; arrows) showed atrial activation following ventricular activation every other beat.

Atrial flutter with 2:1 conduction and conducted atrial fibrillation were ruled out because the tachycardia started with a PVC and there were more ventricular beats than atrial beats. In addition, conducted atrial fibrillation is usually irregular and this tachycardia was regular. The fact that the patient was, most of the time, in atrial sensing-ventricular pacing (due to high-degree AV block) did not completely rule out conducted supraventricular rhythms; however, it made this possibility less likely to occur.

Pacemaker tachycardia could not be suspected because there was not a paced rhythm during tachycardia (a necessary requisite to suspect it).

The decision to upgrade the pacemaker to an ICD was based on a series of facts: 1) The presence of structural heart disease (non-obstructive hypertrophic cardiomyopathy); 2)
The clinical presentation (syncope) and 3) The documented sustained VT (through the analysis of the electrograms stored in the pacemaker).

In this clinical scenario, the presence of syncope alone increases the risk of sudden death by 5-fold. In addition, VT was detected through the analysis of the intracardiac electrograms (increasing the risk of sudden death by almost two-fold). An electrophysiology study may not add up more relevant information. The ICD implantation is preferred over antiarrhythmic drugs for high-risk patients (HCM, syncope, VT).

Conclusion
The current generation of pacemakers facilitates the diagnosis of complicated clinical situations. Proper interpretation of the stored intracardiac electrograms may help in the diagnosis of life-threatening ventricular arrhythmias. The identification of ventricular tachycardia in a patient with non-obstructive hypertrophic cardiomyopathy and syncope has helped in the decision-making of upgrading a pacemaker to an implantable cardioverter-defibrillator.

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References
