

Young syncopal man and decision about permanent cardiac pacing

Case Report

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Abstract: A 20-year old man experienced recurrent syncope, that suggested a partially neutrally-mediated mechanism, but in some cases were without a prodrome. The tilt test was negative. The 12-lead ECG and electrophysiological study showed first-degree AV block. Syncope in bradyarrhythmia was suspected and an implantable loop recorder was indicated. In the first month after implantation the patient experienced one syncopal episode. In the stored ECG, AV junctional rhythm was detected. The patient received a pacemaker and symptoms improved.

Keywords: *Syncope, Implantable loop recorder, Pacemaker*

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1. Case report

In the general population and in patients of younger age the most common cause of syncope is neurocardiogenic. The diagnosis can be determined with great accuracy from a careful history, although the mechanism of syncope remains unexplained in 20% of episodes [1]. The causes of syncope are age-dependent: in young patients neurocardiogenic, psychiatric and primary arrhythmic (e.g. LQTS, Wolff-Parkinson-White syndrome) disturbances are primarily responsible, whereas in elderly patients syncope usually results from heart and cardiopulmonary diseases (e.g. aortic stenosis, pulmonary embolus) or arrhythmias, or neurocardiogenic and neurologic problems [2].

We report the case of a 20-year-old man with repetitive syncope and a negative head-up tilt test, with proximal first-degree atrio-ventricular (AV) block seen on ECG and during electrophysiological study, in whom episodes of AV junctional rhythm in implantable loop recording were documented.

The 20-year old man had experienced 2 episodes of syncope and 5 episodes of pre-syncope during the past 16 months and was referred to our department.

Some of the experienced episodes suggested neurally-mediated triggers but other incidents were sudden without a prodrome. A family history was negative for syncope or sudden death. The initial evaluation including clinical history, physical examination, baseline ECG and orthostatic blood pressure measurements was performed. No history of infection or myocarditis existed. The patient did not take medications. The laboratory examinations which could detect inflammatory and thrombotic causes of syncope were within the normal range: White Blood Count $7.6 \cdot 10^9/\text{mm}^3$, C-reactive protein 200 $\mu\text{g/l}$, ASL-O 17 IU/ml, fibrinogen 2.1 g/l, latex D-dimer 0.2 $\mu\text{g/ml}$. The proteinogram and arterial blood gases were unremarkable.

The findings in baseline ECG were normal sinus rhythm of 48 bpm, PR interval 234 ms and normal QRS complex with duration 80 ms. A 24 h Holter ECG recording confirmed the transient first degree AV block. A head-up tilt test under the Italian protocol (angle of 60°, 20 min passive tilting and nitroglycerin challenge) was performed twice and was negative both times. Transthoracic echocardiogram showed no abnormality. An electrophysiological study was performed. The patient gave written informed consent. Tests of sinus and atrioventricular node function were within normal

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Figure 1. Implantable loop recording during syncopal episode. A - activation point of ILR activator.

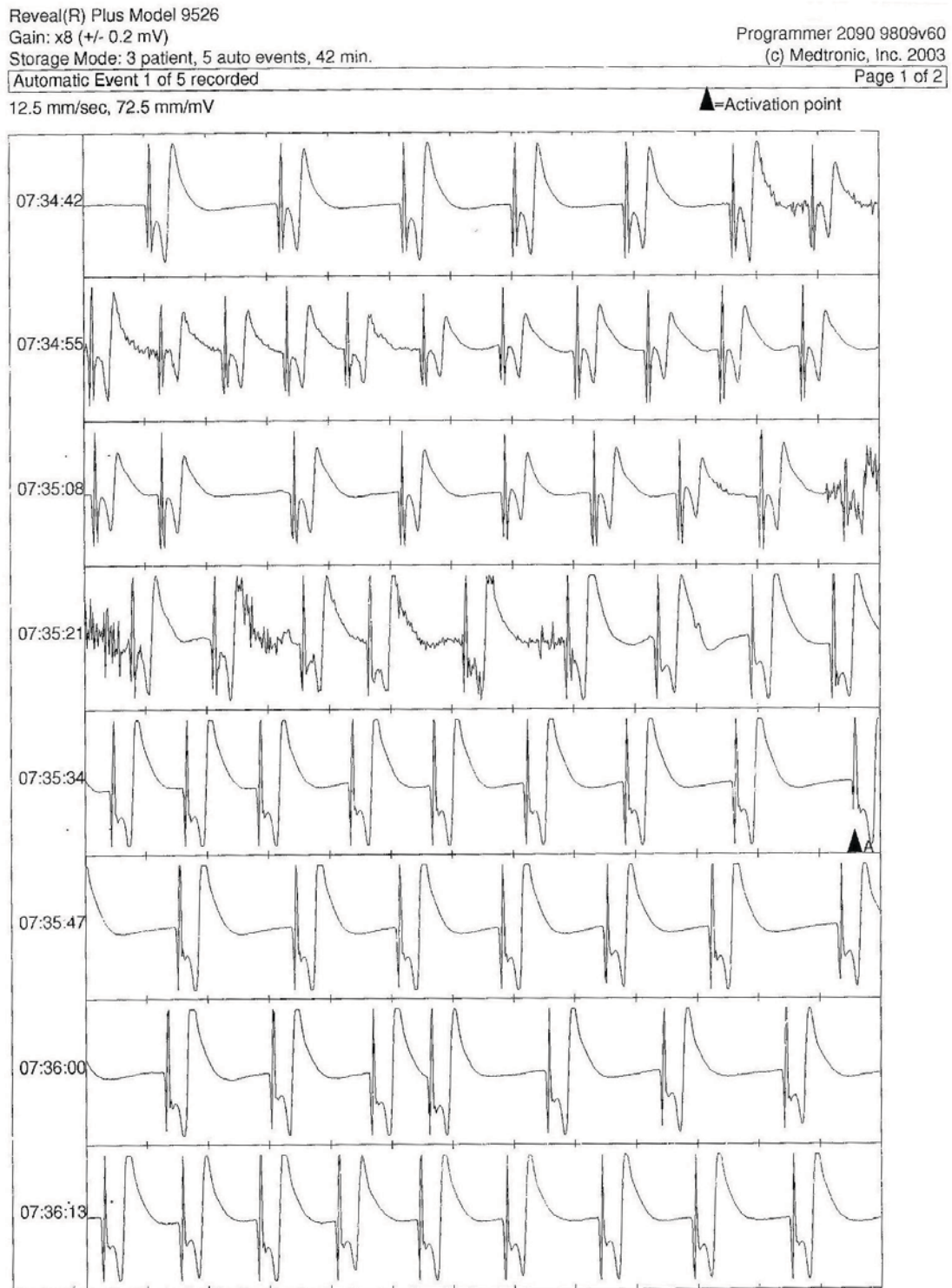
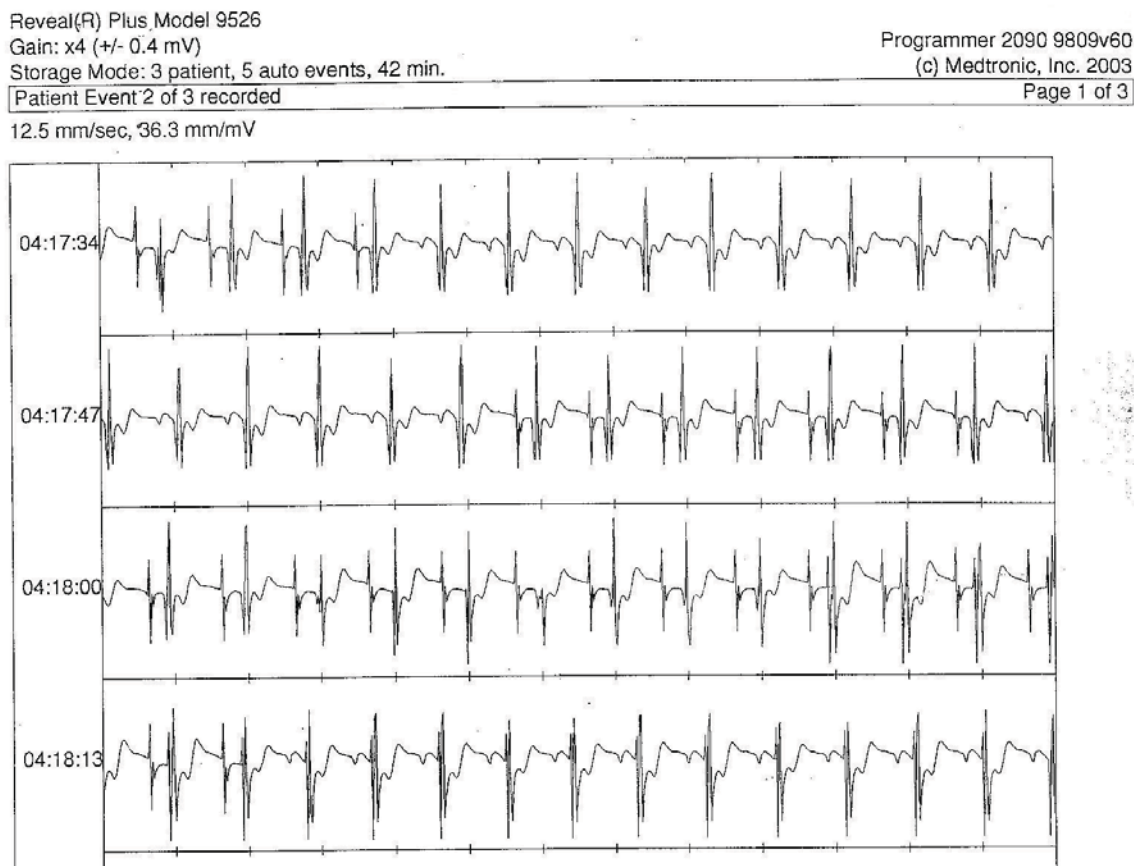


Figure 2. Implantable loop recording during syncopal episode after cardiac pacemaker implantation.

ranges. AH conduction during incremental atrial pacing showed AH prolongation (140 ms) with an AV nodal Wenckebach conduction pattern at 120 bpm. Rest HV interval was 30 ms. No manifestation of ventricular pre-excitation was observed during atrial stimulation manoeuvres. Ventricular pacing was not performed. Proximal first-degree AV block was diagnosed.

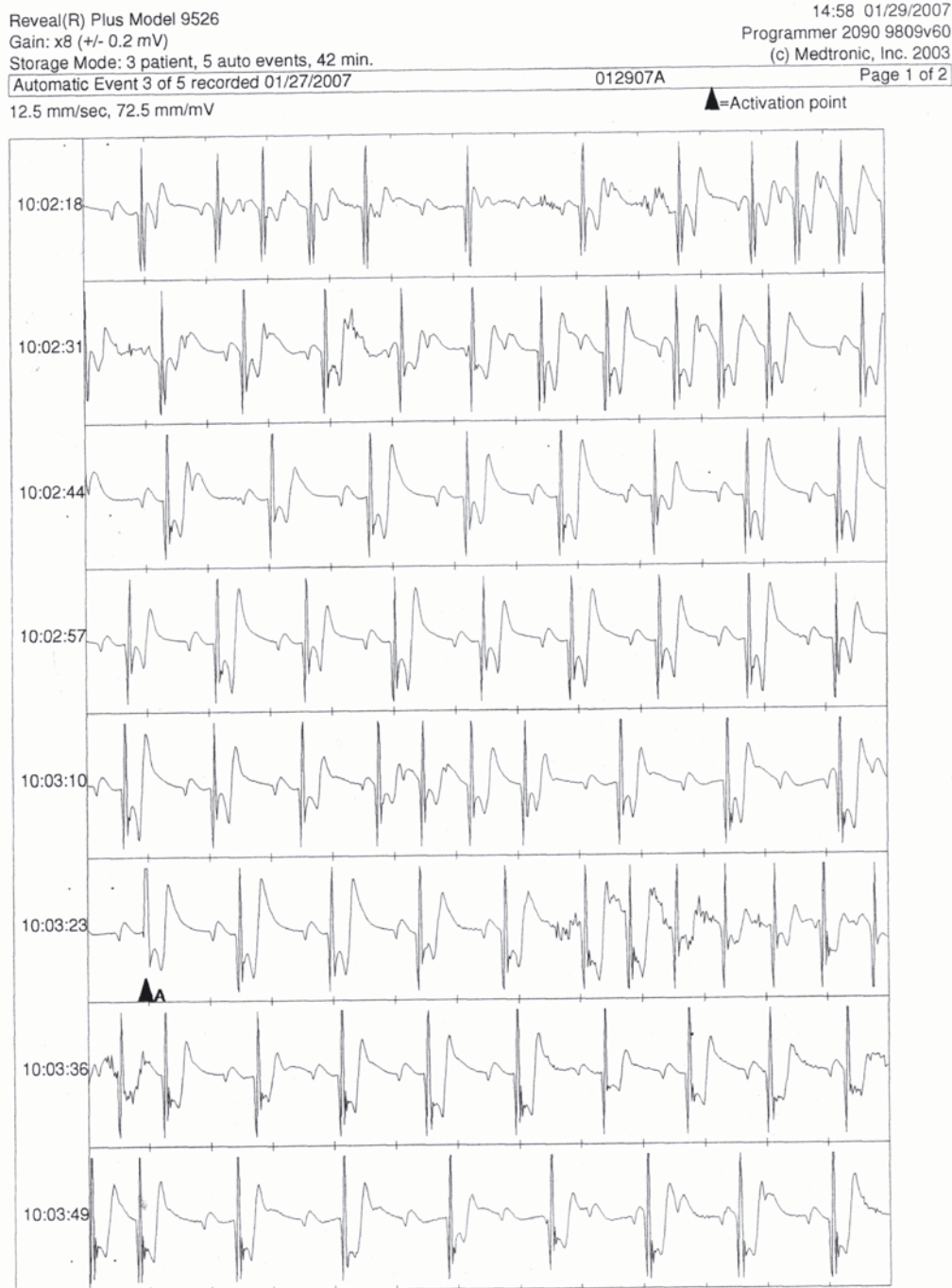
Under the suspicion of syncope in bradyarrhythmia an implantable loop recorder (ILR) was implanted subcutaneously (Reveal 9526, Medtronic Inc., Minneapolis, MN, USA). In the first month after implantation the patient experienced two days of weakness with one syncopal episode that activated the device. In the stored ECG (Figure 1), AV junctional rhythm was detected at a QRS rate of 32 bpm. The patient received a pacemaker (Biotronik, Berlin Germany) with DDDR+hysteresis mode. During six months of follow-up, syncope occurred twice with similar clinical manifestations to those experienced prior to cardiac pacing. In both episodes the ILR was activated and recorded sinus rhythm or correct cardiac pacing (Figure 2). The incidents were associated with typical prodromal symptoms of vasovagal syncope and a blood

pressure drop to 80/50 mmHg was measured by the patient's family. Isometric counter-pressure manoeuvres and volume expansion by salt supplements were indicated (1). In a further year of follow-up the patient's symptoms improved (no syncope, 2 episodes of pre-syncope). During routine pacing control symptomatic second and third degree atrioventricular block was accidentally triggered (Figure 3).

2. Discussion

In young patients a decision about permanent cardiac pacing is always difficult since over the patient's lifetime many reimplantations will be required, increasing the chance of complications. The situation may become even more complex when the conduction disturbances are intermittent and syncope is infrequent. The conventional 24 h Holter ECG in this situation is of limited value since the likelihood of symptom-ECG correlation is very low – no higher than 2 % (1). In such a case as this one when the interval between recurrences of syncope is measured in months or years, ILR is required [2]. The

Figure 3. Implantable loop recording during pre-syncopal episode at the time of cardiac pacing control. A - activation point of ILR activator.



use of ILR for one year in patients with unexplained syncope yielded diagnostic information in more than 90% of patients [3]. This approach is cost-effective [4] and more likely to identify the mechanism of syncope than conventional ECG monitoring (Holter, event Holter) or electrophysiological tests [5].

In this patient, an invasive electrophysiological procedure was performed since ECG features

(asymptomatic sinus bradycardia) suggested an arrhythmic syncope but did not identify the brady-mechanism of syncope. In patients without heart disease electrophysiological testing has a low yield in diagnosis of syncope [1] and low sensitivity for the detection of bradyarrhythmias [6].

Tilt table testing was used to establish whether a neurocardiogenic mechanism of syncope existed.

Although the pretest probability of a neurocardiogenic finding was high, the tilt test was negative twice. Negative tilt testing does not exclude vasovagal syncope, and it could be possible that in the patient only a vasovagal mechanism was causative. However, the recorded atrioventricular block during the cardiac pacing control and earlier 2 days of weakness was diagnostic for arrhythmia. As it turned out, syncope in this patient resulted from two different mechanisms (neurocardiogenic and arrhythmic), with predominance of bradyarrhythmia. After the pacemaker implantation the second mechanism was revealed – vasovagal syncope with blood pressure drop.

Recommendations for cardiac pacing in vasovagal syncope belong to evidence level C, class II [7]. This should not be surprising, since pacing can be expected to correct bradycardia but not prevent hypotension, which is the dominant mechanism leading to syncope in

vasovagal syndrome. The gold standard for diagnosing an arrhythmic cause of syncope is documenting rhythm disturbances on ECG at the time when the patient experiences symptoms. The decision to implant a pacemaker in this patient was based on the presence of symptoms that were directly related to the bradycardia caused by the AV junctional rhythm documented in ILR. In conclusion, given the previous reports regarding the potential usefulness of ILR as a diagnostic approach in syncopal subjects, we applied it in this patient in whom the origin of syncope was unclear after an initial diagnostic workup. Our observations in this patient indicate that ILR is a valuable tool for identifying the mechanism of syncope. It can also decrease the time to pacemaker implantation, and ultimately shorten the duration of arrhythmia symptoms experienced by the patient.

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