A 53-year-old man died suddenly while playing soccer. Two years before, a dual chamber pacemaker including a rate adaptive sensor (DDDR) was implanted to alleviate sick sinus syndrome with symptomatic sinus bradycardia. Autopsy failed to disclose a morphological cause of sudden death. The pacemaker and its leads were correctly placed. Pacemaker interrogation 3 months prior to death revealed normal battery voltage as well as normal thresholds, impedances, and intrinsic amplitudes. The stored intracardiac cardiogram (Figure 1) identified pacemaker-induced ventricular tachycardia (VT) as the reason for sudden death. After normal pacemaker activity, an atrial sensing during the post-ventricular atrial refractory period (PVARP; arrow 1) elicited an ineffective atrial stimulation (AP-SR). The following native ventricular beat (arrow 2) could not be detected by the device because it occurred during the cross-chamber blanking period (40 ms). Consequently, after the atrioventricular delay was complete, a ventricular pacing artifact was delivered (arrow 3), inducing fatal VT.

The underlying event for the VT seems to be the presence of ectopic ventricular complexes and functional undersensing of atrial and ventricular signals. The functional undersensing of the atrial signal is caused by the programmed dynamic PVARP of 350 ms. Functional undersensing of the ventricular signal is caused by the ventricular blanking time. To avoid pacemaker-induced VT, AAI-pacemakers should be preferred in sick sinus node syndrome. Pacemakers should include a ‘ventricular safety pacing’ function delivering a ventricular stimulus very early (110 ms) after a sensed ventricular signal in the first portion of the AV-interval, thus avoiding both cross-talk and pacemaker-induced VT. Furthermore, the ventricular blanking period should be programmed to be as short as possible to minimize the risk of functional ventricular undersensing.
Figure 1 Stored intracardiac cardiogram showing pacemaker-induced ventricular tachycardia. After normal pacemaker activity, an atrial sensing during the post-ventricular atrial refractory period (PVARP; arrow 1) elicited an ineffective atrial stimulation (AP-SR). The following native ventricular beat (arrow 2) could not be detected by the device because it occurred during the cross-chamber blanking period (40 ms). Consequently, after the atrioventricular delay was complete, a ventricular pacing artifact was delivered (arrow 3), inducing fatal ventricular tachycardia (VT).