Pacemaker-mediated tachycardia in an unconventional resynchronization device

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A 67-year-old male patient with heart failure and permanent atrial fibrillation underwent atrio-ventricular nodal ablation and a biventricular device implantation. Instead of a standard cardiac resynchronization therapy device, a DDD pacemaker was implanted with the left ventricular lead in the atrial channel. After the implant, a pacemaker-mediated tachycardia caused by T wave oversensing by the atrial channel was detected.
Pacemaker-mediated tachycardia (PMT) is an iatrogenic rhythm disorder which usually occurs in patients with dual-chamber pacemakers. Pacemaker-mediated tachycardia most commonly occurs due to a repetitive sensing of retrograde P waves in the atrial channel that trigger the atrio-ventricular interval (AVI) and repetitive ventricular stimulation. There are only a few reports on different types of PMT in cardiac resynchronization therapy (CRT) with defibrillator devices1–3 and here we present one similar case.

A 67-year-old male with dilated cardiomyopathy and permanent atrial fibrillation with severely reduced ejection fraction of the left ventricle (20%) was admitted to our department due to refractory heart failure. Despite maximal medical therapy, his ventricular rates were constantly high (>120 b.p.m.), which aggravated the concomitant heart failure. Since restoration of sinus rhythm was not a viable option, AV node ablation was performed and a pacemaker was implanted. To prevent dyssynchrony caused by permanent right ventricular (RV) stimulation, we decided to implant a DDDR (Biotronik Effecta DR) device with the left ventricular (LV) lead in the atrial channel (DDD pacemaker with the function of a CRT device). Biotronik Setrox S 60 lead was implanted in the RV apex, and Medtronic Attain Ability 4196 lead was implanted in the posterolateral branch of CS. The pacemaker was programmed in DDDR mode (90–130 b.p.m.), with paced AVI of 30 ms. All other parameters were standard ‘out-of-the-box’ settings for the mentioned device. After the implant, telemetry monitoring detected frequent paroxysms of wide QRS tachycardia with the rate of 130 b.p.m. It was a paced rhythm with a different morphology than the basic biventricular stimulation (Figure A).

Interrogation of the device revealed that the patient was pacemaker-dependent and the tachycardia cycle length was exactly at the upper tracking rate limit. By observing the intracardiac electrograms, we have detected intermittent sensing of the T waves in the atrial channel (Figure B). If the sensed event occurs after the post-ventricular atrial refractory period (PVARP), the AVI is triggered and right ventricle is paced. Therefore, during the tachycardia we can observe a different morphology of paced QRS complex. High ‘out-of-the-box’ sensing parameters in the atrial channel, combined with high amplitude T waves detected by the LV lead after PVARP, caused an endless-loop tachycardia. The problem was solved by decreasing the sensitivity in the atrial channel and by prolonging the PVARP. After the reprogramming, the tachycardia did not recur. Another simple solution for this problem would be using the DDI mode.

To reduce costs, for patients with permanent atrial fibrillation and an indication for cardiac resynchronization, we implant DDD pacemakers with the LV lead in the atrial channel, instead of costly CRT devices. Concurrently, AV nodal ablation is performed and the AVI is programmed to the minimum possible value to achieve optimal resynchronization.

This ‘off-label’ application of DDD devices can result with an unusual mechanism of PMT. Careful pacemaker programing in ‘off-label’ indications is warranted to avoid potential complications. In similar settings, we advise prolonging PVARP and lowering the atrial sensitivity promptly after the implant, or simply programming the pacemaker to DDIR mode.

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References