Unusual adverse consequence of reverse ventricular remodelling following cardiac resynchronization therapy

Pow-Li Chia1*, Rajesh N. Subbiah1,2, Dennis Kuchar1, and Bruce Walker1

1Department of Cardiology, St Vincent’s Hospital Sydney, 390 Victoria Street, Darlinghurst, NSW 2010, Australia; and 2University of New South Wales, Sydney, NSW 2052, Australia

* Corresponding author. Tel: +65 97341738; fax: +6563526682, Email: powlichia@gmail.com

Cardiac resynchronization therapy has been shown to produce reverse ventricular remodelling in patients with severe heart failure. We report an unusual case of T-wave oversensing, most likely as a consequence of reverse ventricular remodelling resulting in change of the implantable cardioverter-defibrillator lead redundancy.

Case presentation
A 28-year-old male underwent implantation of a biventricular implantable cardioverter defibrillator (ICD) (Medtronic Concerto II CRT-D, Medtronic Inc., Minneapolis, MN) for non-ischaemic dilated cardiomyopathy with a left ventricular ejection fraction (LVEF) of 20%. An active fixation ICD lead (Medtronic Sprint Quattro Secure 6944) was positioned at the right ventricular (RV) apex, the atrial lead (Medtronic 5076) at the antero-superior right atrium, and the left ventricular (LV) lead (Medtronic 4194) in the lateral marginal branch of the coronary sinus. Pacing thresholds and impedances were normal in all leads. P-wave amplitude was 1.1 mV, and R-wave amplitude was 17 mV in the RV and 20 mV in the LV. Device programming parameters were as follows: DDDR mode, 50–150 b.p.m.; paced atrioventricular (AV) delay 130 ms, sensed AV delay 100 ms; LV offset 210 ms; ventricular and atrial sensitivity at 0.3 mV. At implant, defibrillation threshold was 25 J and sensing was 100% with RV sensitivity at 1.2 mV.

Follow-up device interrogations revealed stable lead parameters. The patient’s functional capacity improved to New York Heart Association Class I and LVEF increased to 40%. At 12 months of follow-up, intermittent T-wave oversensing was noted only after paced QRS complexes with resultant double-counting detection (VS–VS) following a biventricular paced event (see Figure 1). Right ventricular R-wave amplitude was 20 mV. The rate threshold criteria for ventricular tachycardia or fibrillation were not met and there was no inappropriate device therapy.

Serum electrolyte levels were normal. A chest X-ray revealed significant reduction in cardiac size compared to Day 1 post-implant (see Figure 1). Reverse ventricular remodelling was accompanied by an increase in ICD lead redundancy, which may have changed the orientation of the lead to the endocardium resulting in T-wave oversensing.

The device was reprogrammed to LV pacing only with upper tracking rate and upper sensor rate of 125 b.p.m. Ventricular sensitivity was altered from 0.30 to 0.45 mV, ventricular blank post-ventricular pacing increased from 200 to 240 ms, and ventricular sense response maximum rate decreased from 130 to 125 b.p.m. Following these changes, T-wave oversensing was no longer observed even at faster rates and continuous ventricular pacing was restored.

Discussion
Causes of T-wave oversensing include reduction of R-wave amplitude and relative or dynamic increase in the T-wave amplitude,1 progressive cardiomyopathy,1 electrolyte abnormalities,1 and myocardial ischaemia.2 In our patient, reverse ventricular remodelling with cardiac resynchronization therapy (CRT) resulted in reduced cardiac size and greater ICD lead redundancy. The change in ICD lead orientation likely led to T-wave oversensing and subsequent loss of biventricular pacing. Cardiac resynchronization therapy has also been shown to partially restore abnormal Ca2+ homeostasis induced by dysynchronous cardiac contraction in heart failure patients.3 Through such ion channel remodelling, CRT may alter cardiac repolarization with consequent T-wave oversensing.

Conflict of interest: none declared.
Device troubleshooting: cross connection of ventricular leads in a patient with decreased right ventricular electrical activity

Daisuke Kutsuzawa, Takanori Arimoto*, Tetsuro Shishido, Shintaro Sasaki, Tadateru Iwayama, Daisuke Ishigaki, Hiroki Takahashi, Takehiko Miyashita, Takuya Miyamoto, Joji Nitobe, Tetsu Watanabe, and Isao Kubota

Department of Cardiology, Pulmonology, and Nephrology, Yamagata University School of Medicine, 2-2-2 Iida-nishi, Yamagata 990-9585, Japan

* Corresponding author. Tel: +81 23 628 5302; fax: +81 23 628 5305, Email: t-arimoto@med.id.yamagata-u.ac.jp

A patient with cardiac sarcoidosis showed low R-wave amplitude in the entire right ventricle (RV). To troubleshoot this, a left ventricular (LV) lead was implanted in the coronary vein. The pace/sense terminal of the defibrillation lead was connected to LV:IS-1 of

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