and device are from different manufacturers, due to differences in signal processing. This was particularly notable in Biotronik devices connected to integrated bipolar leads from other manufacturers. In this case, both lead and device were made by Medtronic.

In the reported case, the abnormality was only apparent during lead impedance measurement in DDI mode, and during DDD or AAI-DDD pacing lead impedance was measured normally with no oversensing (Figure 1, middle/lower panels). Review of electrograms and programmed parameters confirmed that the apparent abnormalities were normal device function, explained by the algorithms determining refractory periods during DDI pacing and delivery of stimuli for lead impedance testing.

Lead impedance is measured by delivery of a sub-threshold stimulus, triggered by an atrial-sensed (AS) or atrial-paced (AP) event; the stimulus then falls within a refractory period and is not sensed. If no AS or AP is detected within 4 s of initiation of impedance testing, the stimulus is delivered asynchronously, and will be sensed if not within a refractory period. In our patient, the device was programmed DDI, with the post-ventricular atrial refractory period (PVARP) set to Auto. Post-ventricular atrial refractory period is then calculated using the formula:

\[
\text{PVARP (ms)} = \text{Escape interval (ms)} - \text{programmed AV delay (ms)} - 350 \text{ ms}
\]

The parameters from our patient give the value:

\[
\text{PVARP (ms)} = 1500 \text{ ms} - 180 \text{ ms} - 350 \text{ ms} = 970 \text{ ms}
\]

The long-derived PVARP causes the intrinsic P-wave to fall within this period and be labelled as an atrial refractory event (AR; Figure 1A, top panel). The impedance test therefore times out, and the sub-threshold stimulus is delivered asynchronously. This is sensed in both channels and labelled as a ventricular event (VS). In view of the AR–VS pattern seen during this configuration, the manufacturers do not recommend the use of Auto-PVARP in DDI mode, and PVARP should be set manually to an appropriate value.

In the DDD or AAI-DDD mode, Auto-PVARP is calculated to maintain a 2:1 block rate of 30 bpm above current atrial rate. In our patient, the atrial rate was ~100 bpm, therefore:

\[
\text{PVARP (ms)} = \text{atrial escape interval at 130 min} - \text{AV delay} = 461 \text{ ms} - 180 \text{ ms} = 281 \text{ ms}
\]

The P-wave is therefore sensed normally, and the sub-threshold stimulus triggered to fall within the refractory period.

**Summary**

DDI mode with a low base rate is commonly used to minimize RV pacing in ICDs. When used with Auto-PVARP, the algorithm may lead to an AR–VS marker pattern and asynchronous stimuli for impedance testing. It is important to be aware of the potential for this to occur, and to understand the associated algorithms, in order that device malfunction is not suspected incorrectly.

**Conflict of interest:** none declared.

**References**


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**CASE REPORT**

doi:10.1093/europace/euq412
Online publish-ahead-of-print 24 November 2010

**Temporary external implantable cardioverter defibrillator in the pacemaker-dependent ventricular tachycardia patient**

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A patient with ischaemic cardiomyopathy underwent implantable cardioverter defibrillator (ICD) extraction for a severe pocket infection and sepsis. During 5 weeks of critical medical care after device extraction, heart block and recurrent monomorphic ventricular tachycardia (VT) were managed with an ‘externalized’ active fixation pacemaker lead and a resterilized ICD generator. This case demonstrates how a permanent pacing lead and an external ICD generator can provide reliable temporary pacing and automatic anti-tachycardia pacing for recurrent VT until a new device can be implanted and more permanent VT treatment options are feasible.

**Case**

When an implanted pacemaker or implantable cardioverter defibrillator (ICD) becomes infected, the entire system, including the pulse generator and all leads, must be removed for infection resolution. The timing of permanent device re-implantation is dependent upon

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infection severity, the effectiveness of antimicrobial treatment, and the documentation of sterile blood cultures. In a pacemaker-dependent patient, temporary pacing is needed until the new device system can be implanted. An active fixation permanent pacemaker lead can be percutaneously implanted via the internal jugular or subclavian vein and attached to a resterilized, external permanent pace-maker for temporary right ventricular (RV) pacing.1 This technique is associated with fewer adverse events than other temporary pacing modalities and is cost-effective.2,3 In ICD patients with recurrent ventricular tachycardia (VT) after device extraction, tachyarrhythmias also need to be managed.

A 74-year-old man with ischaemic cardiomyopathy was transferred to our institution with a purulent pocket infection and severe sepsis with Staphylococcus aureus, 3 weeks after an ICD generator change. His dual-chamber ICD was implanted 9 years prior for VT, and he developed complete heart block in the interim, leaving him pacemaker-dependent. His left ventricular ejection fraction was 30%, and, since the recent ICD replacement, he had 81 episodes of asymptomatic monomorphic VT (cycle length 370–430 ms), despite chronic treatment with amiodarone. Each episode was successfully terminated with a single anti-tachycardia pacing (ATP) burst, and no shocks were delivered.

On presentation, he was ill-appearing, with persistent methicillin-sensitive S. aureus bacteraemia and fevers to 103°F, despite treatment with intravenous nafcillin. The patient underwent device removal, extensive debridement of the grossly purulent left pectoral device pocket, and laser extraction of his chronic atrial and ventricular leads. A femoral quadripolar catheter was used for temporary pacing during the procedure. The right internal jugular vein site was then steriley prepped, an active fixation permanent pacemaker lead was percutaneously implanted in the RV apex, and the lead was connected to an external resterilized ICD generator (Figure 1). The ventricular pacing threshold was 0.4 V at 0.5 ms. The device was programmed for bradycardia pacing at VVI 80 p.p.m., and a VT zone of 140–250 b.p.m. was set, with six burst ATP therapies only.

The patient remained intubated after the extraction procedure because of S. aureus pneumonia, and a tracheostomy was necessary after 2 weeks because of ongoing dependence on mechanical ventilation. He required ongoing pressor support due to sepsis-induced hypotension in the setting of severe cardiomyopathy, and his critical clinical status precluded a VT ablation procedure. Over a 5-week period, the temporary device system provided reliable pacing and multiple successful ATP therapies for recurrent monomorphic VT, and no external shocks were needed. The patient recovered from his infection, and a new permanent right-sided biventricular ICD was implanted in the right pectoral region.

**Figure 1** (Top, left) Chest radiograph of temporary pacing system. An active fixation permanent pacemaker lead is affixed to the RV apex (black arrow) and is connected to an external ICD (black asterisk), which is secured to the neck with an adhesive dressing. (Top, right) Photograph of external temporary pacing system at the time of removal. The pacemaker lead is sutured to the skin via its suture sleeve at the right internal jugular vein access site (white arrow) and is connected to a resterilized ICD (white asterisk). (Bottom) Sample telemetry strip from a VT event after extraction of the infected ICD system. Ventricular tachycardia is successfully detected and treated by the temporary pacing/external ICD system with a single 10-pulse burst of ATP. VVI pacing at 80 p.p.m. is seen after VT termination, with heart block and dissociated P-waves noted.
Conflict of interest: J.M.C., S.D., and R.J.V. have modest conflicts of interest to report with Medtronic, Boston Scientific, St Jude, and Biotronik in the form of modest honoraria for educational programmes and training support for the institution’s fellowship programme.

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