CASE REPORT

Ventricular capture by anodal pacemaker stimulation

Eraldo Occhetta*, Miriam Bortnik, and Paolo Marino

Divisione Clinica di Cardiologia, Facoltà di Medicina e Chirurgia di Novara, Università degli Studi del Piemonte Orientale, Azienda Ospedaliera Maggiore della Carità, Corso Mazzini 18, 28100 Novara, Italy

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This report describes the case of an 86-year-old male with syncopal paroxysmal 2:1 atrioventricular block and a single chamber VVI pacemaker programmed to bipolar sensing and unipolar pacing. After recurrence of syncope, a complete loss of ventricular capture with regular ventricular sensing was observed on ECG; fluoroscopic examination suggested perforation of the right ventricle by the helix of the implanted screw-in lead. Reprogramming the pacemaker to bipolar pacing/sensing resulted in regular ventricular capture and sensing, suggesting effective anodal stimulation from the ring electrode permitting complete non-invasive palliation.

KEYWORDS
Anodal stimulation; Cardiac pacing; Atrioventricular block

Introduction

Permanent cardiac pacing is the standard treatment for a variety of symptomatic bradycardias. Modern pacemakers can often be programmed either to unipolar or bipolar pacing and/or sensing configurations, allowing the use of either unipolar or bipolar leads. In unipolar configuration, the electrode stimulating the cardiac chamber, typically the cathode (negative pole) of the pacing circuit, must be in direct contact with the myocardium, whereas the other pole completing the electric circuit, the anode, is provided by an electrode not in contact with heart muscle, typically the pacemaker can. In bipolar configuration, both the anode and the cathode are in contact with the heart. In transvenous bipolar leads, the most distal electrode usually serves as cathode, whereas the anode is realized as a more proximal ring electrode with larger surface area than the tip electrode, in order to reduce pacing impedance and avoid anodal stimulation (that has a higher diastolic capture threshold than cathodal stimulation using identical electrodes). The same description applies to sensing, which also requires the use of two electrodes, in order to record the voltage differences constituting the cardiac electrogram. Unipolar sensing typically also uses the pacemaker can as reference electrode while bipolar sensing, which is less affected by extracardiac signals such as myograms from muscles in the pocket or interference from electrical appliances, uses two electrodes in the heart.1

We present a case report in which ventricular capture was lost during unipolar pacing, because of right ventricle perforation, caused by the helix of an active fixation bipolar pacing lead. Regular ventricular stimulation was obtained by reprogramming the pacemaker to bipolar mode, suggesting effective anodal stimulation from the ring electrode of the implanted lead.

Case report

An 86-year-old man with severe Alzheimer’s disease was admitted to our institution for sudden loss of consciousness; on admission his surface ECG showed paroxysmal 2:1 atrioventricular block, right bundle branch block, left axis deviation in conducted complexes, and a ventricular rate of 40 bpm. A single chamber VVI pacemaker (Regency SC 2406 L, St Jude, Sylmar, CA, USA) was implanted and connected to a bipolar screw-in ventricular lead (Capsure Fix 4068, Medtronic Inc., Minneapolis, MN, USA); deep sedation was required during the procedure because of extreme agitation. During implantation, the ventricular threshold was 1.5 V, 2.4 mA at a pulse duration of 0.5 ms and R-wave amplitude was 14 mV. The patient’s post-implant hospitalization remained unremarkable and he was discharged with the pacemaker programmed to VVI 70 bpm, unipolar pacing, and bipolar sensing configuration, at an output voltage of 3.9 V (pulse duration 0.5 ms); unipolar pacing configuration was chosen to permit better visualization of the spike artefact. Two months later, the patient suffered a pre-syncopal episode and was found to have complete loss of ventricular capture on 12-lead ECG; proper sensing of ventricular spontaneous beat was also noticed. During pacemaker check, ineffective ventricular pacing was confirmed in unipolar configuration at maximum output voltage (Figure 1); when programming the system to the bipolar electrode configuration, regular ventricular capture was re-established.
Figure 1 12-lead surface ECG during unipolar pacing and bipolar sensing configuration with a pacing rate of 80 bpm shows correct sensing of the spontaneous ventricular events with complete loss of ventricular capture (output voltage, 6.9 V; pulse width, 1 ms).

Figure 2 ECG recorded after pacemaker reprogramming; bipolar pacing/sensing configuration allowed restoration of correct ventricular capture (pacing threshold 4.5 V, 0.5 ms).
capture was restored with an output voltage threshold of 4.5 V at a pulse duration of 0.5 ms (Figure 2). Lead impedance measured by the device telemetry was 429 and 484 Ω in the unipolar and bipolar configuration, respectively.

An antero-posterior chest X-ray was suggestive of perforation of the helix electrode through the right-ventricular apex (Figure 3).

The efficacy of ventricular capture during bipolar pacing configuration was interpreted as anodal stimulation from the right ventricular ring electrode.

The pacemaker was permanently programmed in bipolar pacing/sensing configuration with an output voltage of 6.9 V (pulse duration 1 ms) and no further signs of malfunction were observed on prolonged ECG monitoring; the described pacemaker reprogramming avoided lead repositioning, which, in this patient, with severe psychiatric disorder was considered highly problematic.

No further incidents of loss of ventricular capture have occurred over the following 12 months of follow-up.

Discussion

The phenomenon of anodal excitation of cardiac muscle has been described by Cranefield et al., in the late-1950s; later, also Dekker demonstrated that the myocardium could be excited by anodal stimuli. Although cathodal stimulation of the excitable tissues is explained by direct depolarization of excitable cell membranes closest to the electrode, anodal stimulation would result in hyperpolarization of these membranes; the ability to trigger an action potential is thus paradoxical. Prior studies have demonstrated that anodal stimulation results in a 'dog bone'-shaped zone of hyperpolarization under the stimulation electrodes, whereas the tissue lying in the convexity of the dog bone becomes depolarized and is referred to as 'virtual cathodes'. It is generally accepted that the excitation wavefront of anodal stimulation propagates from these virtual cathodes.

The absolute refractory period is typically shorter after anodal than cathodal stimulation. Consequently, if a stimulus falls in the vulnerable period of a spontaneous cycle, the risk of triggering a tachyarrhythmia is higher with anodal than with cathodal stimulation; furthermore, anodal electrostimulation is associated with higher pacing thresholds than cathodal; thus, anodal stimulation is generally rarely advised. Nevertheless, in our patient with spontaneous escape rhythm in which lead repositioning was considered highly problematic because of the concomitant severe psychiatric disease, this kind of stimulation allowed us to achieve complete non-invasive palliation of the clinical problem; no further complications have been observed during the following year of follow-up. Undoubtedly, in a healthy pacemaker-dependent patient, anodal stimulation ought not to be recommended and surgical revision of the system should be considered as first choice treatment.

References