Rate-dependent Loss of Capture during Ventricular Pacing

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Abstract

A 63-year-old patient who had undergone atrial septal defect surgical repair received implantation of a single-chamber VVI pacemaker for long RR intervals during atrial fibrillation. One week later, an intermittent loss of capture and sensing failure was detected at a pacing rate of 70 beats/min. However, a successful capture was observed during rapid pacing. Consequently, the pacing rate was temporarily adjusted to 90 beats/min. At the 3-month follow-up, the pacemaker was shown to be functioning properly independent of the pacing rate. An echocardiogram showed that the increased pacing rates were accompanied by a reduction in the right ventricular outflow tract dimension. The pacemaker was then permanently programmed at a lower rate of 60 beats/min.

Key words: pacemaker, pacing rate, capture

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Introduction

Pacing and sensing functions, particularly the precise measurement of the pacing threshold, are crucial to assure a successful capture for permanent cardiac pacing. However, in some cases, the capture threshold may vary along with the pacing rate. In this study, we report a patient who suffered from chronic atrial fibrillation with long RR intervals in whom a rate-dependent loss of capture was elucidated within 3 months after pacemaker implantation.

Case Report

A 63-year-old man who underwent atrial septal defect surgical repair 6 months previously suffered from faintness and dizziness one month after surgery. The Holter monitor electrocardiogram showed chronic atrial fibrillation with 104 episodes of long RR interval (>1.5s), the longest of which was 5.92 s. The patient was referred to our hospital for pacemaker implantation. An echocardiogram revealed a right atrium enlargement and a widened coronary sinus complicated with persistent left superior vena cava. There was also evidence of severe pulmonary artery hypertension with moderate tricuspid regurgitation. The patient underwent implantation of a transvenous single-chamber pacemaker (Zephyr SR 5620, St. Jude Medical, St. Paul, USA) three days after admission. Due to the anatomical variation, the ventricular lead (1888TC-58) repeatedly strayed into the coronary sinus and swung during the course of implantation. By means of great efforts, the electrode was finally fixed at the right ventricular outflow tract (RVOT) septum. The intraoperative measurements revealed a capture threshold at 2.0 V with a 0.5 ms pulse duration, lead impedance at 780 Ω, and an R wave during spontaneous rhythm at 4.5 mV.

The patient complained of faintness at the one-week follow-up after implantation. A pacemaker interrogation showed a spontaneous R wave amplitude at 3.4-4.6 mV and lead impedance at 620 Ω, whereas intermittent pacing and sensing failure was recorded at a pacing rate of 70 beats/min. However, a successful capture was acquired at 90 beats/min with an output of 3.0 V/0.5 ms (Fig. 1). A chest X-ray was also performed and no sign of lead dislodgment was observed (Fig. 2). An echocardiogram showed no signs of lead perforation or effusion in the pericardial space. Considering the difficulty of the lead placement and fixation, the electrode was not repositioned since the patient was not dependent on the pacemaker. Therefore, the pacemaker was programmed to VVI 90 beats/min at an output of 4.0V (pulse duration: 1.0 ms) with an R wave sensitivity at 2.0 mV.

At the 3-month follow-up, the patient again underwent a
end-systolic diameter measured 2.7 cm, 2.3 cm, 2.3 cm, 2.1 cm, and 1.9 cm, respectively (Fig. 4). Therefore, the increased pacing rates were accompanied by a reduction in the RVOT dimension in the present case. The movement of the pacing lead did not result in any notable differences at various pacing rates.

**Discussion**

The main finding in the present case is a capture failure observed at the slower pacing rate of 70 beats/min but not during pacing at 90 beats/min at the initial follow-up, which is difficult to explain from an electrophysiological standpoint. In order to interpret this observed phenomenon, an echocardiogram was conducted. The potential role of a mechanical factor, or changes in the ventricular dimension affecting the contact between the stimulating electrode and endocardial surface, was explored in the present case. It was readily noted that the RVOT dimension became smaller at a higher rate, which is similar to the report by Katsumoto et al. (1), who found that a higher pacing rate decreased the atrial dimension and secondarily improved the electrode contact with the endocardial surface. Correspondingly, Chinushi et al. (2) reported a case in which pacing and a sensing failure was detected at a pacing rate of 60 beats/min but undetectable during rapid pacing. They hypothesized that at slower heart rates, the contact between the lead tip and the right ventricular myocardium was looser due to a larger ventricular volume, but did not present any echocardiographic data. In the present case, we verified this hypothesis through echocardiographic values. However, our findings did not suggest that such a phenomenon occurred in all cases. In the present case, severe pulmonary artery hypertension, moderate tricuspid regurgitation and myocardial injury due to atrial septal defect (ASD) and ASD surgery may be responsible. At the 3-month follow-up, this phenomenon disappeared when the electrode tip was tightly en
capsulated by fibrous tissues. Thus, pacing and sensing parameters should be measured during slower pacing rates, especially intraoperatively, in order to find the optimal pacing site. In the case of the above-mentioned phenomenon, lead repositioning is not imperative if the patient is not dependent on the pacemaker. The electrode tip may become securely fixed to the endocardium over time. Therefore, a close follow-up is essential and reoperation may sometimes be necessary if such rate-dependent threshold changes persist.

The authors state that they have no Conflict of Interest (COI).

Jingfeng Wang and Haiyan Chen contributed equally to this work.

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