A Case of Transient Rise of Pacing Threshold during the Chronic Phase of Pacemaker Implantation

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We experienced a case of pacing failure with transient rise of the pacing threshold more than one year after implantation. Neither the generator nor lead system was found to be defective. During the antibiotics therapy to treat cholecystitis, which was found on admission, pacing failure was improved. The clinical course suggested that the infection was related to the improvement, although accurate mechanisms were unknown.

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Introduction

After pacemaker implantation, the pacing threshold usually shows a transient rise to a peak value at around two weeks, and lowering thereafter. In the following case, a transient rise in the pacing threshold with pacing failure was observed during the process of infectious disease, which was improved when the infection was treated.

Case Report

A sixty-nine year old female patient was hospitalized to the Neurology Department of our hospital because of brain infarction in July 2003. Her electrocardiogram (ECG) showed a sinus pause of 6.2 sec. An electrophysiological study (EPS) was performed and revealed sinus node dysfunction with normal atrio-ventricular (AV) conduction. On November 4th, 2003, implantation of atrial single chamber pacemaker (AAI, set rate: 70 bpm, generator: Philos SR, lead: Sy45 JBB) was performed. The pacemaker function has been checked at regular intervals. On the last check on April 19th, 2004, the threshold was a pulse amplitude of 0.7 V and a width of 0.4 msec (0.7 V/0.4 msec), with the output set as 3.0 V/0.4 msec. The patient was again hospitalized in October 2004, because of loss of appetite, nausea and vomiting accompanied by high CRP values and arrhythmias. Her ECG exhibited pacing spikes with intermittent atrial responses and AV junctional escaped beats (Figure 1). Blood examination revealed high values of Al-p (249 mg/dl), γ-GTP (126 U/l) and CRP (8.5 mg/dl), but otherwise normal values, including the blood electrolytes. Chest X ray film showed normal thoracic ratio and normal electrode site (Figure 2). Signs of myocardial ischemia were not found by 201Tl and 123I-BMIPP scintigraphy.

Pacemaker output had not changed since the prior check. No abnormal findings were found with the generator and lead system by telemetry. A threshold measurement was made using ordinary methods.
by gradually lowering the test pulses. During the threshold check, once a pulse of 7.5 V/0.4 msec captured the atrium, successive atrial captures were conducted until 0.8 V/0.4 msec (pacing threshold 424 nJ, lead impedance 604 Ω, cell voltage 2.79 V), although the intermittent pacing failure was observed with a pulse of 3.0 V/0.4 msec (pacing threshold 5960 nJ). When a further decrease in the stimuli strength caused capture loss, a pulse of 3.0 V/0.4 msec immediately given could not capture the atrium and was followed by loss of capture for 13 beats (9 sec) (Figure 3).

The infectious disease was diagnosed as acute cholecystitis based on blood chemistry data and abdominal ultrasonography. The patient was treated 5 days with SBT/CPZ 2 g/day since October 18th, 7 days with CMZ 2 g/day since October 26th along with 7 days with PSL 30 mg/day of steroid pulse therapy for the myocardial involvement. After the improvement of the infection, threshold measurement was performed again on November 2nd. This time, lowering of the test pulse to 0.3 V/1.5 msec (pacing threshold 216 nJ, lead impedance 623 Ω, cell voltage 2.77 V) caused failure of the response but increasing the pulse to 3.0 V/0.4 msec resulted in an immediate capture (Figure 4).

**Discussion**

The threshold rise after the pacemaker implantation has been known to occur within one month after the implantation in most cases. In the acute phase of implantation, transient rise in the threshold has been observed in many cases, followed by lowering of the threshold and a stable phase. In the chronic phase, factors for a threshold rise include dislocation of the leads, acidosis, alkalosis and administration of β-blockers or other antiarrhythmic agents, especially of Ic group. The lead dislocation is the most common cause, but usually in the acute phase. Other reports include the contribution of flecainide and acute cholestatic hepatitis due to propafenone administration. Flecainide acetate has been associated with 200–1000% increase in capture threshold. All the agents above are excluded in the present case.

One of the points of importance in the present case is the method of threshold measurement. When the test stimuli were gradually lowered in strength,
lower than threshold stimuli failed to capture. Interestingly, there was successive atrial capture at very low output during the threshold measurement in spite of pacing failure even at an adequately high output. The mechanisms of that phenomenon are unknown, but Igarashi5) described that successive paced beats with adequately higher suprathreshold stimuli might result in lowering the threshold even when the prior stimuli with the same current could not result in successive capture.

In the present case, the clinical course suggested the contribution of the inflammatory process. However, to our knowledge the relationship between the rise of the threshold and the systemic infection has not been reported. It is conceivable that local myocardial inflammation and edema may affect the threshold.

Conclusion

A case of pacing threshold of unknown cause was reported in the chronic stable phase of pacemaker implantation. The results of threshold measurement were interesting, with clinical implications.

References

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