The Significance of Pulse Oximetry in the Diagnosis of Pacemaker Failure to Output

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In a routine monitor electrocardiogram few hours after implantation of a dual-chamber pacemaker, there was a suspicion of ventricular oversensing leading to failure to output. Pulse oximeter plethysmographic wave signal virtually excluded this suspicion. This case report demonstrates the clinical utility of initial pulse oximeter wave analysis in excluding pacemaker failure to output.

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CASE

A 67-year-old woman with hypertension, Type II diabetes mellitus, end-stage renal disease who is treated with hemodialysis and without known cardiac disease was admitted to internal medicine department after recurrent episodes of syncope and head trauma during the month prior to her admission. Subarachnoid hemorrhage was diagnosed by brain computerized tomography. The electrocardiogram (ECG) on admission showed junctional bradycardia of 40 bpm with QT 0.56 second and QTc of 0.46 second, without signs of acute ischemia. On 24-hour Holter ECG, many sinus pauses up to 10 seconds with episodes of junctional bradycardia up to 30 bpm were diagnosed. A diagnosis of symptomatic sinus node dysfunction was made; the patient was transferred to cardiology department and a dual-chamber (DDDR) pacemaker was implanted. The patient was monitored and several hours after implantation, while she was asymptomatic, we received many alerts from the monitor. Figure 1A raises a high suspicion of failure to pacing output. However, while monitoring the patient with pulse oximeter in order to check her blood oxygen saturation, we noticed normal plethysmographic wave signals indicating ventricular contraction, which helped to exclude failure to output. In the monitor recording, there is evidence of atrial pacemaker stimuli, which are not followed by ventricular pacemaker spikes or are followed

by small ventricular deflections. A subsequent 12-lead ECG exhibits consistent ventricular electrical activity and confirms that there is no pacemaker malfunction. (Fig. 1B)

Absence of a pacing output may be caused by oversensing or by output problems such as battery depletion or component failure. Oversensing of intrinsic or extrinsic signals make the device believe that a cardiac event has occurred and it inhibits pacemaker output. Possible causes of oversensing include myopotentials, electromagnetic interference, sensing the T wave, cross-talk (when the pacemaker senses and interprets the atrial pace as an intrinsic ventricular beat), lead fracture, lead insulation failure, and more relevant to this case report, lead dislodgement. Our patient had two-lead monitor ECG exhibiting atrial stimuli but no ventricular stimuli few hours after pacemaker implantation. A suspicion of ventricular oversensing leading to failure to output due to ventricular lead dislodgement was raised. The pulse oximeter plethysmographic wave signal virtually excluded the possibility of failure to output and 12-lead ECG confirmed this conclusion. There was no need for magnet application to identify the cause of oversensing, and there was no need for chest x-ray or any additional test to exclude lead dislodgement or other causes of failure to output. In conclusion, for every patient with a pacemaker and a suspected failure to output, we recommend initial simple pulse oximeter wave

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Figure 1. (A) Two-lead electrocardiogram (25 mm/s) recorded simultaneously with pulse oximeter plethysmography from a 67-year-old woman several hours after a dual-chamber (DDDR) pacemaker implantation. There is a normal plethysmographic wave signal after every visible atrial pacemaker stimuli. The letters added to the recording represent the following: a = atrial pacing; v = ventricular pacing; s = spontaneous ventricular complex. (B) Twelve-lead ECG confirmed that there is no loss of ventricular capture.

analysis followed by 12-lead ECG and magnet application if needed. Only if the diagnosis of fail-

ure to output is not excluded, additional tests are needed.

Figure 1. Continued.