Intercostal muscle twitching: An unusual manifestation of extracardiac stimulation related to right ventricular outflow tract pacing

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The present case report describes a patient who underwent successful dual-chamber pacemaker implantation with active ventricular lead fixation at a high septal region in the right ventricular outflow tract. Unexpectedly, stimulation at a high output in the right ventricular outflow tract caused an unusual extracardiac stimulation, specifically, intercostal muscle twitching.

Key Words: *Complication; Extracardiac stimulation; Pacing; Right ventricular outflow tract*

The right ventricular outflow tract (RVOT) is now considered to be an alternative pacing site because of its reported potentially beneficial effects, which include improved left ventricular function, more synchronous ventricular activation and induction of fewer myocardial perfusion defects (1-4). In the present case report, a patient who underwent successful dual-chamber pacemaker implantation with ventricular lead fixation at a high septal region in the RVOT is described because of unusual extracardiac stimulation related to the lead.

CASE PRESENTATION

A 29-year-old man was admitted for presyncopal and syncopal episodes occurring for more than three years. His mother and brother had undergone pacemaker implantation at the ages of 45 and 30 years, respectively. Physical examination on admission revealed normal findings except for a regular bradycardia at 34 beats/min, which the patient tolerated well. A surface electrocardiogram taken on admission demonstrated a 2:1 atrioventricular (AV) block with a pattern of a right bundle branch block, left anterior fascicular block and a normal PR interval. Subsequently, the patient underwent permanent dual-chamber pacemaker implantation with a Kappa DR 901 (Medtronic Inc, USA) using an atrial active fixation lead (model 5076, Medtronic Inc, USA) and a ventricular active fixation lead (5076 CapSure Fix, Medtronic Inc, USA) that was placed at the high septal region of the RVOT (Figures 1 and 2). Paced QRS duration decreased from 160 ms baseline to 130 ms postprocedure, associated with positive complexes in leads II, III and aVF, and negative complexes in leads I and aVL. The right ventricular pacing threshold and impedance at implantation were 0.6 V at 0.5 ms and 680 ohms, respectively. The R wave sensing threshold and slew rate were 15.5 mV and 2.8 V/s, respectively. The atrial pacing threshold and

Une contraction musculaire intercostale : La manifestation inhabituelle d'une stimulation extracardiaque reliée à l'entraînement de la chambre de chasse du ventricule droit

Le présent rapport de cas décrit un patient qui s'est fait implanter un stimulateur double chambre avec l'installation de dérivations ventriculaires actives dans la région septale haute de l'entraînement de la chambre de chasse du ventricule droit. Contre toute attente, la stimulation à haute intensité de l'entraînement de la chambre de chasse du ventricule droit a provoqué une stimulation extracardiaque inhabituelle, une contraction musculaire intercostale.

Figure 1) Posteroanterior chest x-ray showing the active fixation lead position high in the septal right ventricular outflow tract site

impedance were 0.8 V at 0.5 ms and 1200 ohms, respectively. The P wave sensing threshold and slew rate were 6.8 mV and 0.8 V/s, respectively. During the implantation procedure, diaphragm stimulation was not observed on fluoroscopy during pacing outputs of up to 10 V. While the threshold tests were being performed, the patient described a mild twitching sensation in his chest. On

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Figure 2) Lateral view of the lead position in the right ventricular outflow tract

physical examination (inspection and palpation of the chest wall), it was obvious that a small area of intercostal muscle at the left sternal border around the fourth intercostal space was twitching. No pericardial effusion was observed on echocardiography and no clinical signs of perforation were detected. Twitching was more apparent when the heart was paced at a higher output (approximately 4 V to 5 V), and it diminished in intensity by decreasing the amplitude to 2.5 V. The patient also had an intrinsic rhythm at approximately 40 beats/min and 2:1 AV conduction that intermittently improved to 1:1. A long AV interval was programmed to periodically let the patient's own rhythm supervene. Because of acceptable pacing thresholds, optimal lead position and no intercostal muscle twitching at a decreased output (approximately 3 V), we elected to finish the procedure. During follow-up two months later, the patient was doing well, with no complaints, and transthoracic echocardiography did not show pericardial effusion. While in a sitting position, pacing at high outputs did not cause any intercostal muscle twitching.

DISCUSSION

Pacing in the RVOT is increasingly preferred over apical pacing because it provides synchronous activation, improves left ventricular

function and causes fewer myocardial perfusion defects (1-4). Extracardiac stimulation, such as diaphragmatic stimulation, is a well-known problem, especially when one places the lead in the apical position and stimulates at a high pacing output. However, extracardiac stimulation, such as in the intercostal muscles due to lead positioning in the RVOT, is very unusual and the present case is the second one reported in literature (according to a PubMed search). The first reported case (5) is very similar to the present case, with intercostal muscle twitching induced by RVOT pacing at a high pacing output. As it was stated by Oginosawa et al (5), perforation of the RVOT by the 1.8 mm helix of the lead screw, which serves as the electrode, could not be excluded because helix perforation is not invariably the cause of pericardial effusion, chest pain, pericardial friction rub or pacing failure. No sign of perforation was detected clinically or echocardiographically in the present case. During the procedure, twitching of the intercostal muscle was only minimally felt by the patient and only after he was informed of it happening. How the twitching occurred may be explained by the following mechanism. The stimulation site was very high in the septal region and was possibly very close to the thorax and intercostal muscles. Hence, stimulation at a high output could be responsible for intercostal muscle twitching that was in close proximity to the affixed lead position.

CONCLUSIONS

If one aims to fix the lead in the RVOT position, one must always be cautious and aware of potential problems such as the intercostal muscle twitching observed in the present case. Avoidance of this extracardiac stimulation could likely be achieved by fixating the lead low in the mid-septal area and performing a proper pacing threshold test commencing at higher outputs while observing and palpating the mid-sternal intercostal region.

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