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TRANSIENT PHRENIC NERVE STIMULATION IN A PATIENT WITH SINGLE CHAMBER PACEMAKER: CASE REPORT

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The article presents a clinical observation of a patient with episodes of phrenic nerve stimulation after inadvertent permanent ventricular pacing from the middle cardiac vein. The methods of early diagnosis of this complication and techniques for its prevention are described.

Key words: phrenic nerve stimulation; coronary sinus; middle cardiac vein; pacemaker; ventricular lead

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Diaphragmatic stimulation occurs in approximately 30% of pacemaker implantations but is predominantly observed with the implantation of cardiac resynchronization therapy (CRT) devices. This is attributed to the epicardial location of the left ventricular stimulation electrode and its proximity to the left diaphragmatic nerve [1]. Cases of unintentional positioning of the electrode for right ventricular (RV) stimulation in the coronary sinus (CS) branch, especially in the middle cardiac vein (MCV), which fluoroscopically resembles the typical position of the electrode in the RV apex, have also been described. Diaphragm stimulation in this case is also possible if the left diaphragmatic nerve is located in anatomic proximity to the MCV.

This complication usually does not lead to hemodynamic disturbances, but can cause a significant decrease in the patient's quality of life, causing recurrent episodes of pulsation in the left subcostal area (sometimes only at a certain body position) [3]. The use of various techniques and careful radiologic and electrocardiographic (ECG) monitoring during implantation can prevent positioning of the ventricular electrode in the CS and avoid the development of diaphragmatic stimulation. We present a clinical observation of a patient with episodic diaphragm stimulation for 16 years after inadvertent implantation of a RV electrode in the MCV.

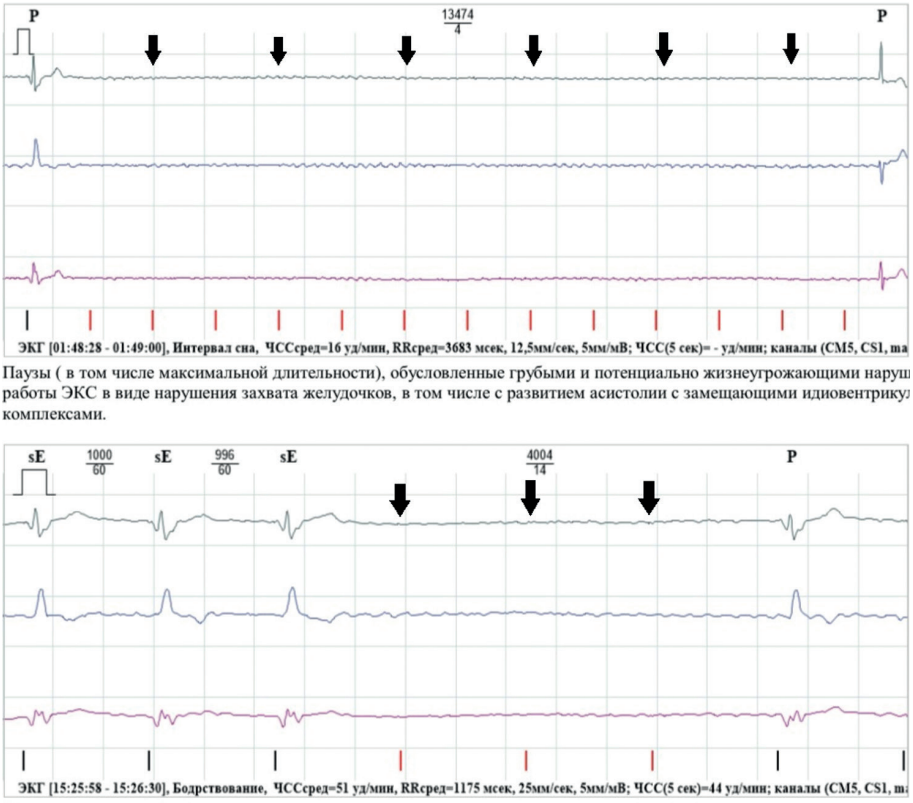

Fig. 1. Fragment of Holter monitoring with ineffective ventricular stimulation. Arrows indicate ineffective ventricular stimuli.

Clinical case

A 70-year-old man was admitted as an emergency to the intensive care ward of the cardiovascular surgery department with complaints of dizziness attacks unrelated to physical activity; he denied any loss of consciousness before admission to the hospital. The patient has a history of permanent atrial fibrillation, tachysystolic variant, since 2000. In 2001, he underwent atrioventricular junction ablation with implantation of a single-chamber pacemaker in VVI mode (ECS-300). The pacemaker (ECS-300) was replaced in 2004 due to «electronic circuit failure». In 2007, pacemaker malfunction was detected again (increase in the threshold of stimulation on the ventricular electrode), in connection with which the patient was urgently hospitalized, where pacemaker reprogramming with increase in the pulse amplitude was performed. However, the next day, « pacemaker failure» was documented, temporary pacemaker was established, and the pacemaker (model Sigma SSR303, Medtronic, USA) and ventricular electrode (model Polyrox PX-60-BP, Biotronik, Germany) were replaced. According to the patient's words, immediately after surgery he noted a pulsation in the left subcostal region, which he reported to the attending physician. The pulse amplitude was reduced without attempting electrode repositioning, after which the pulsation sensation became less frequent but persisted during further follow-up (mostly at night) until admission to our hospital. With these complaints, the patient repeatedly consulted neurologists, who established the diagnosis of asthenic neurosis.

In January 2016, the patient underwent pacemaker replacement due to critical battery exhaustion, and SENSIA SR pacemaker (Medtronic, USA) was implanted. Postoperative pacemaker programming showed no abnormalities in the function of the implanted electrode, documented stimulation threshold was <1 V. Due to the occurrence of episodes of vertigo in October 2023, the patient underwent Holter monitoring on an outpatient basis, which revealed episodes of ineffective ventricular stimulation with pauses up to 13.5 s (Fig. 1), in connection with which the patient was urgently hospitalized in our hospital.

When analyzing the ECG on admission, QRS morphology of the type of complete blockade of the right bundle branch of the Hiss with a QRS width of 200 ms was noteworthy (Fig. 2), which made it possible to suspect left ventricular stimulation. ICU pacemaker programming revealed an increase in stimulation threshold to 2.5 V, while the pulse amplitude in the pacemaker settings was also 2.5 V. Thus, episodes of ineffective ventricular stimulation were associated with a lack of a safe amplitude margin (a twofold margin is recommended for chronic threshold stimulation). The automatic stimulation threshold detection function has been programmed into monitor mode; when attempting to set automatic stimulation threshold tracking, after capture testing, enabling this function is not recommended by the device.

Increasing the pulse amplitude to a safe level of 5.0 V (2x), resulted in the appearance of diaphragm stimulation in the left subcostal region, which the patient had already experienced with some frequency since 2007. The prognostic life of the pacemaker battery after the newly programmed parameters was <2 months, so the decision was made to replace the pacemaker and ventricular electrode.

The operation was performed for emergency indications, given the patient's dependence on pacemaker and discomfort from constant stimulation of the diaphragm. Fluoroscopy in posterior-anterior (PA) and left lateral (LAO) projections revealed positioning of the ventricular electrode in the CS-MCV branch (Fig. 3). The electrode was mobilized, but retrieval by simple traction on the stylet was not possible. In the absence of systems for electrode extraction and the risk of CS damage, it was decided to refrain from further attempts to extract the electrode. A new electrode was implanted into the interventricular septum with satisfactory stimulation parameters (ventricular electrogram amplitude 16.1 mV, stimulation threshold 0.6 V). A new Reply SR pacemaker (Microport CRM-Sorin, China) has been connected.

The postoperative period proceeded without peculiarities. The sensation of pulsation in the left subcostal region has completely disappeared. On ECG performed at 1 day after surgery- effective ventricular stimulation with QRS morphology of the type of complete blockade of the left bundle branch of Hiss (Fig. 4). The patient was discharged on the 4th day after surgery in satisfactory condition.

DISCUSSION OF FINDINGS

In the presented clinical case, the patient developed persistent left subcostal pulsation in the early postoperative period after pacemaker replacement with implantation of a

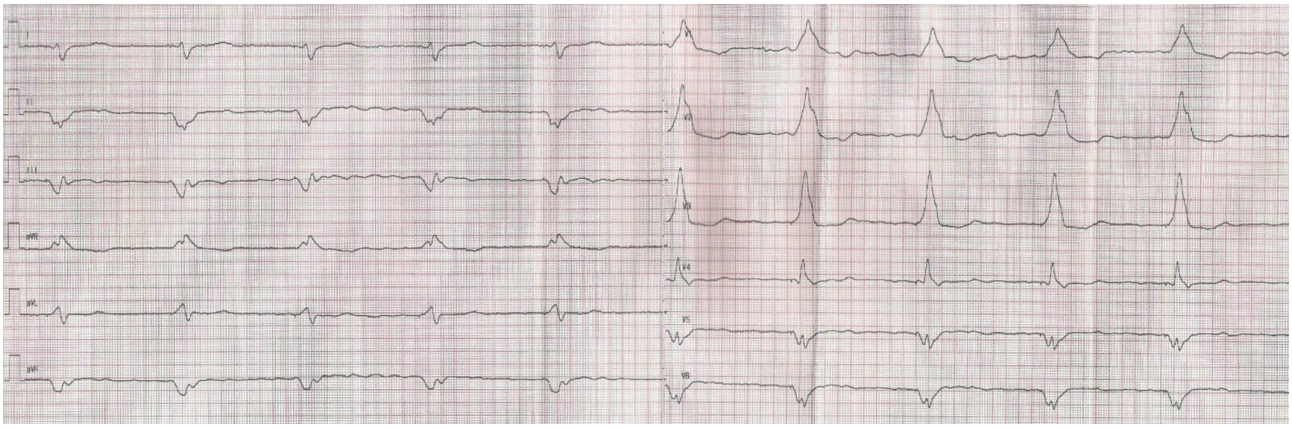

Fig. 2. Electrocardiogram of the patient on admission. Effective ventricular stimulation. QRS morphology is of the type of complete right bundle branch block.

new ventricular electrode. A tactic of decreasing the pulse amplitude was used to control this symptom, which, however, did not completely eliminate the stimulation of the diaphragm. The pulsation appeared predominantly at night (probably at a certain position of the patient's body), which eventually forced the patient to consult a neurologist. A diagnosis of asthenic neurosis was established, which obviously had a quite definite somatic cause. However, a detailed search for the causes of diaphragmatic stimulation was apparently not performed, because diaphragm pulsation was not reproduced during routine pacemaker programming (high current stimulation is not performed during programming in everyday practice). Electrode parameters (stimulation threshold, impedance) were also within normal limits.

Diaphragm stimulation after pacemaker implantation is not a life-threatening complication, but it can significantly reduce the patient's quality of life. This complication may occur after implantation of the electrode in the apex of the RV when, due to anatomical proximity of the RV apex to the left dome of the diaphragm and relatively small thickness of the RV wall, the diaphragm muscle is captured by the stimulus from the RV electrode, as well as in case of inadvertent positioning of the electrode in one of the branches of the CS. In the latter case, electrical stimulation of the left diaphragmatic nerve running along the left lateral surface of the pericardium with an impulse from an epicardially located ventricular electrode is possible. It is usually possible to recognize this complication intraoperatively with a high-amplitude (10 V) current stimulation maneuver and reposition the electrode to an alternative RV position (e.g., interventricular septum).

Unintentional electrode entry into the branches of the CS is possible due to anatomical reasons. The CS is located in the atrial-ventricular sulcus closer to the atrium and is a continuation of the great vein of the heart; its origin is considered to be the confluence of the oblique vein of the heart (Marshall's vein), the posterior, middle and small veins of the heart also flow into it. The MCV is located along the posterior interventricular sulcus and receives venous blood from both ventricles. In most cases, the mouth of the MCV is located near the mouth of the CS. During the implantation procedure, if certain rules described below are not followed, the ventricular electrode may inadvertently end up in the MCV, which may not be recognized in time because the anatomical course of the MCV resembles the position of the electrode in the apex of the right ventricle during fluoroscopy in the posterior-anterior (PA) projection (Fig. 5) [4, 5].

This complication can be avoided by following certain techniques:

- the ventricular electrode through the tricuspid valve using a «loop» by withdrawing the stylet from the distal part of the electrode or using a J-shaped stylet, usually included in the set with the electrode (this maneuver reduces the risk of the electrode getting into the orifice of the CS, which may be dilated for various reasons, and further into one of its branches;
- withdrawal of the ventricular electrode into the output tract of the RV, followed by its traction (in this case, the electrode «descends» to the apex of the RV, which confirms the free course of the electrode in the RV cavity);
- intraoperative use of different fluoroscopic projections (RAO, LAO) to confirm the electrode position;
- 12-lead ECG analysis (against ventricular stimulation) during or immediately after implantation to assess the morphology of the stimulated QRS (especially V1);
- in doubtful cases, echocardiography after pacemaker implantation, which allows to detect the absence of the electrode in the right ventricle and to suspect its incorrect positioning [6].

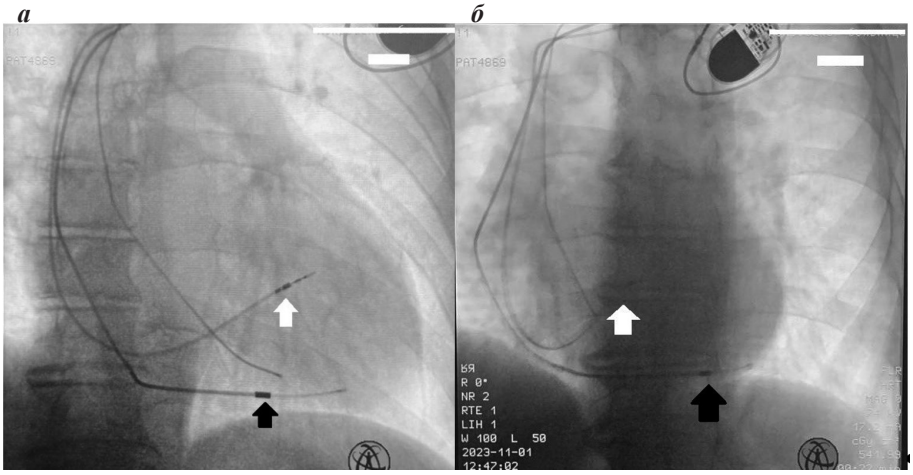

Fig. 3. Intraoperative fluoroscopy in posterior-anterior, PA (a) and left oblique projection, LAO (b). Black arrows indicate the electrode in the MCV. White arrows indicate the newly implanted electrode in the interventricular septum.

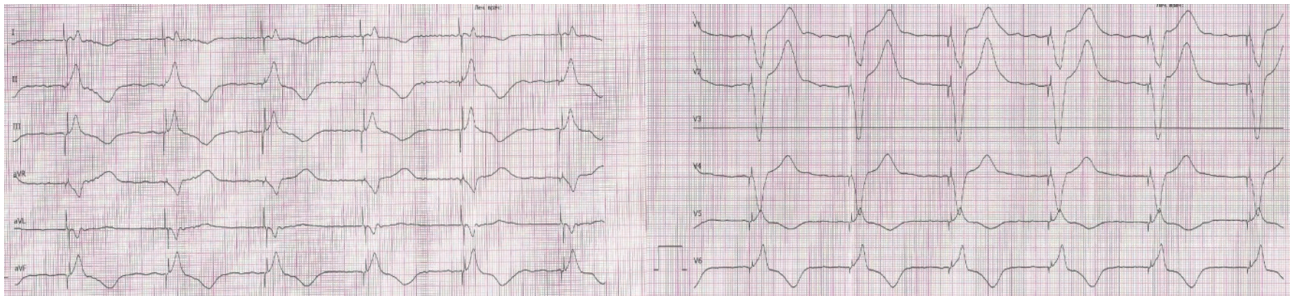

Fig. 4. Electrocardiogram of the patient at 1 day after surgery. Effective ventricular simulation. QRS morphology in lead V1 is of the type of left bundle branch block.

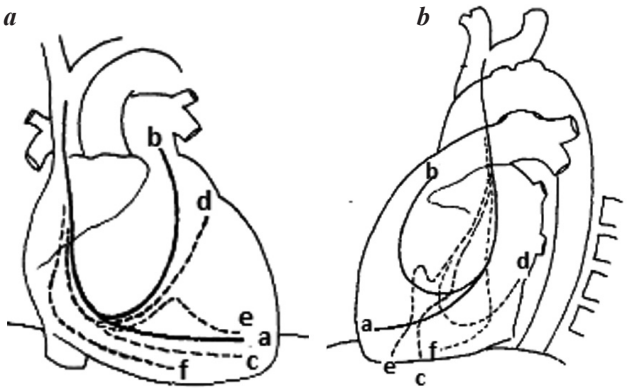

Fig. 5. Schematic location of electrodes in posterior-anterior (a) and lateral (b) fluoroscopic projections. The solid line indicates electrodes: a - in the apex of the right ventricle, b - in the pulmonary artery. Dotted lines indicate electrodes: c - in the middle vein of the heart, d - in the large vein of the heart, e - in the posterior vein of the heart, f - in the small vein of the heart [5].

In our clinical case, the cause of diaphragm stimulation was apparently stimulation of the diaphragmatic nerve by the electrode located in the MCV, although direct stimulation of the left dome of the diaphragm cannot be excluded. If we refer to the design of the implanted electrode with passive fixation (Polyrox PX-60-BP, Biotronik, Germany), it can be noted that the distance between the distal end of the electrode and its ring is increased and is 31 mm [7]. According to the manufacturer, this design improves sensitivity to ventricular potentials. However, stimulation was also carried out in bipolar mode, which in case of increased distance between bipolar contacts can lead to depolarization of a larger myocardial surface with possible capture of adjacent structures (left dome of the diaphragm). When such an electrode is in the MCV, as in our case, the risk of trapping the diaphragmatic nerve with a «wider» electric field is also increased.

The design features of this electrode include fractal coating of the distal end to reduce the stimulation threshold (the steroidal coating of the electrode is absent). However, an increase in the stimulation threshold did occur 16 years after implantation, which eventually led to the identification of the true cause of the diaphragm stimulation that had been bothering the patient for a long period of time and its successful elimination.

CONCLUSION

Diaphragmatic stimulation is not an uncommon complication after pacemaker implantation, which causes significant discomfort to the patient and impairs quality of life. One of its causes is inadvertent positioning of the electrode in one of the CS branches, most often MCV. Timely diagnosis of this complication allows early repositioning of the electrode and avoid deterioration of the patients' quality of life.

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