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Case Report

A sea of troubles: upgrade of ICD to CRT-D in a patient with ischemic cardiomyopathy

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Abstract: This is a case describing a 54-year-old Chinese male with ischemic cardiomyopathy (ICM) coexisting with electrical and mechanical dyssynchrony and an implantable single-cavity cardioverter-defibrillator (ICD). The patient was admitted to our hospital for upgrade to cardiac resynchronization therapy defibrillator (CRT-D). Many difficulties were encountered during the process of ICD upgraded CRT-D, which including the left subclavian venous anomalous, the left brachiocephalic stenosis, coronary sinus (CS) Thebesian valve (TV), phrenic nerve stimulation, screw loosening, and mechanical dyssynchrony resulting from atrial tachycardia. Each of these difficulties may be encountered during an operation, but all difficulties are rare at the same time. Therefore, this case has clinical education value.

Keywords: Cardiac resynchronization therapy defibrillator, implantable cardioverter-defibrillator, ischemic cardiomyopathy, heart failure, pacing

Case report

A 54-years-old man had ICM with sustained ventricular tachycardia and had ICD insertion 5 years ago. He was admitted to our hospital because of drug refractory heart failure with electrical and mechanical dyssynchrony. As battery power was poor the system was scheduled for upgrading to CRT-D. On examination, the blood pressure was 96/74 mmHg, the respiration rate was 22/min, and the heart beat was 98/min. The lungs were clear and there is no murmur could be heard. Before implantation a 12-lead ECG showed sinus rhythm with atrial premature beat, a prolonged PR interval of 302 ms and wide QRS complexes of 158 ms. A transthoracic echocardiogram revealed enlargement of the left atrium and the left ventricular, coexisting with decreased contraction of left ventricular anterior wall with left ventricular ejection fraction of 33.6%. There was no difficulty to puncture the left axillary vein, but the 0.038 inch SJM® guide wire could not pass through the left subclavian vein (**Figure 1A**). Contrast venography was performed immediately, the results showed that the left subclavian vein was dilated, the left brachiocephalic vein was stenotic (**Figure 1B**, black arrows), and the left internal vein and the left external jugular vein were accompanied by reflux (**Figure 1B**, white arrows). In consideration of the success of homolateral operation ensured preservation of the venous capital, a 0.035 inch RADIFOCUS® guide wire was used again. After repeated efforts, two RADIFOCUS® guide wires eventually reached the inferior vena. Conventional CS angiography was performed, which showed CS and its branches. However, the 0.014-inch Runthrough® guide wire could not enter into the coronary sinus ostium (CSO) (**Figure 2A**). CS angiography were performed again, the result showed that the guide tube was prevented by TV. After the guide catheter was readjusted, the 0.014 inch Runthrough® guide wire referred to the distal of the CS (**Figure 2B**). The left ventricular electrode was initially inserted to the distal of the posterolateral branch. The pacing threshold was 0.6 V, but the phrenic nerve stimulation was obvious. Adjust the left ventricular electrode to the anterior interventricular venous branch. However, there was no ideal pacing site. The left ventricular electrode was re-

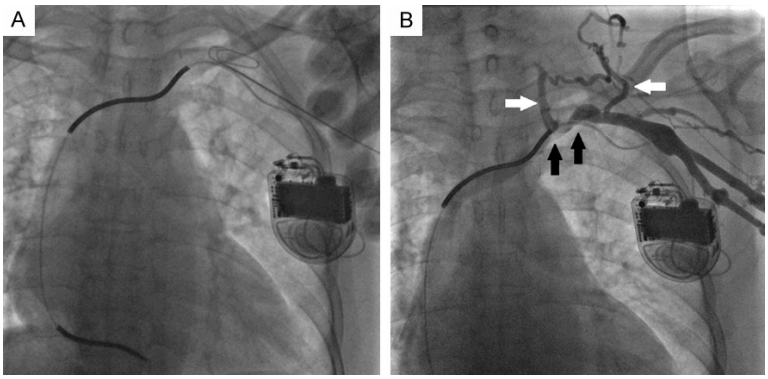

Figure 1. Stenosis in the left brachiocephalic vein and anomalous in the eft subclavian vein. A. A 0.038 inch SJM® guide wire couldn't through he left subclavian vein. B. Contrast venography demonstrated tenosis in the left brachiocephalic vein and anomalous in the left subclavian vein black arrows) and regurgitation in the left internal jugular vein and the left external jugular vein (white arrows).

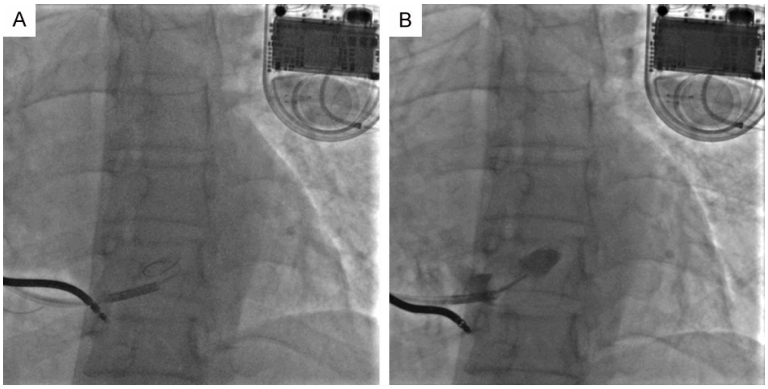

Figure 2. Presence of TV may prevent passage of a catheter from the right atrium to CS. A. The 0.014-inch Runthrough® guide wire could not enter into the distal of the CS. B. The coronary sinus angiography showed that the guide tube was prevented by the TV.

adjusted to the proximal side of the posterolateral branch vein, and no phrenic nerve stimulation was performed. The threshold was 0.75 V and the impedance was 980 Ω. A new pacing wire could be introduced without difficulty followed by Vitatron® ICQ09B in the right atrium. The atrial lead and the left ventricular pacing lead, together with the initial ICD lead were connected to a CRT-D device and placed in the original generator pocket. Before the pacemaker bag was to be sutured, the parameters were tested again. The results were all in the normal range except for the right ventricular electrode pacing impedance greater than 2000 ohms (**Figure 3A**). Careful examination revealed that the right ventricular electrode screw had become loose. Reconnect the right ventricular electrode to CRT-D and the right ventricular electrode impedance decreased to 1346 ohms (**Figure 3B**). Compared with the preoperative, the QRS duration decreased from 158 ms to 130 ms (**Figure 4**) and the left ventricular election fraction increased from 33.6% to 43.7% (**Figure 5**). The upgrade to CRT-D significantly reduced the left ventricular size and the improvement of heart failure symptoms was also observed. Five days later, the patient was discharged from hospital without symptoms. After successful implantation atrioventricular and interventricular delays were optimized guided by ECG. Device follow-up was performed 1 month after implantation. However the patient returned because of dyspnea and palpitation 50 days after the follow-up. Atrial tachycardia and loss of atrioventricular synchrony were found by ECG (**Figure 6A**). Because of the patient had first degree atrioventricular block, dislocation and/or fracture of right atrium electrode lead was suspected. However, no changes were observed in the pacing threshold, P-wave amplitudes and pacing impedance and the chest radiography excluded the lead dislodgment. With the treatment of amidarone, atrioventricular synchrony (**Figure 6B**) was recovered and the symptoms of heart failure were relieved.

Discussion

Cardiac resynchronization therapy (CRT) has become a well-established treatment for patients with severe left ventricular dysfunction who have persistent heart failure symptoms, despite optimal medical therapy [1]. Heart failure is a progressive condition and many patients initially implanted with an ICD potentially benefit from CRT. It is generally believed that upgrading to CRT carries a higher risk of

A

Device: **Egida CRT-D D394TRG**
Serial Number: **PXJ607956S**

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Device Information

Device	Medtronic	Egida CRT-D D394T...	PXJ607956S	Implanted: 22-Oct-2017

Device Status (Implanted: 22-Oct-2017)

Battery Voltage (RRT=2.63V)		3.19 V	22-Oct-2017
Last Full Charge		8.5 sec	22-Oct-2017
	Atrial	**RV**	**LV**
		SVC	
Pacing Impedance	437 ohms	2342 ohms	399 ohms
Defibrillation Impedance		RV=44 ohms	
		SVC=66 ohms	
In-Office Threshold		0.75 V @ 0.40 ms	0.75 V @ 0.40 ms
Programmed Amplitude/Pulse Width	2.00 V / 0.40 ms	2.50 V / 0.40 ms	3.50 V / 0.40 ms
Measured P/ R Wave	1.6 mV	8.9 mV	
Programmed Sensitivity	0.60 mV	0.30 mV	

B

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Battery Voltage (RRT=2.63V)		3.19 V	22-Oct-2017
Last Full Charge		8.5 sec	22-Oct-2017
	Atrial	**RV**	**LV**
		SVC	
Pacing Impedance	437 ohms	1346 ohms	399 ohms
Defibrillation Impedance		RV=44 ohms	
		SVC=66 ohms	
In-Office Threshold		0.75 V @ 0.40 ms	0.75 V @ 0.40 ms
Programmed Amplitude/Pulse Width	2.00 V / 0.40 ms	2.50 V / 0.40 ms	3.50 V / 0.40 ms
Measured P/ R Wave	1.6 mV	8.9 mV	
Programmed Sensitivity	0.60 mV	0.30 mV	

Figure 3. Increased impedance of the right ventricular pacing electrode resulted from the loosening of the screw. A. Before the pacemaker bag was to be sutured, the parameters were tested again. The results were all in the normal range except for the right ventricular electrode pacing impedance was 2342 ohms. B. Reconnection of the right ventricular electrode to CRT-D and the right ventricular electrode impedance decreased to 1346 ohms.

acute complications versus a de novo implant because of venous access issues, the risk of damage or extraction of old leads, the higher risk of infection, and the additional time that may be required. Failure of CRT may be due to a variety of factors, including inappropriate patient selection, inability to place a left ventricular pacing lead due to unfavorable coronary sinus, anatomical variation, or implantation of the CS lead at a suboptimal position. In addition, patients with ICM are less likely to benefit from CRT compared with their non-ischemic counterparts. Pacing at regions distant from dense infarct zones was not sufficient for guaranteeing optimal left ventricular mechanical response. Thrombosis can occur along cardiac implantation devices wires leading to stenosis and occlusions of the great veins [2]. ICD implantation is growing much more rapidly than pacemaker insertion, as the indications for ICD are expanding. ICD leads are bulkier and the potential for venous damage is expected to be

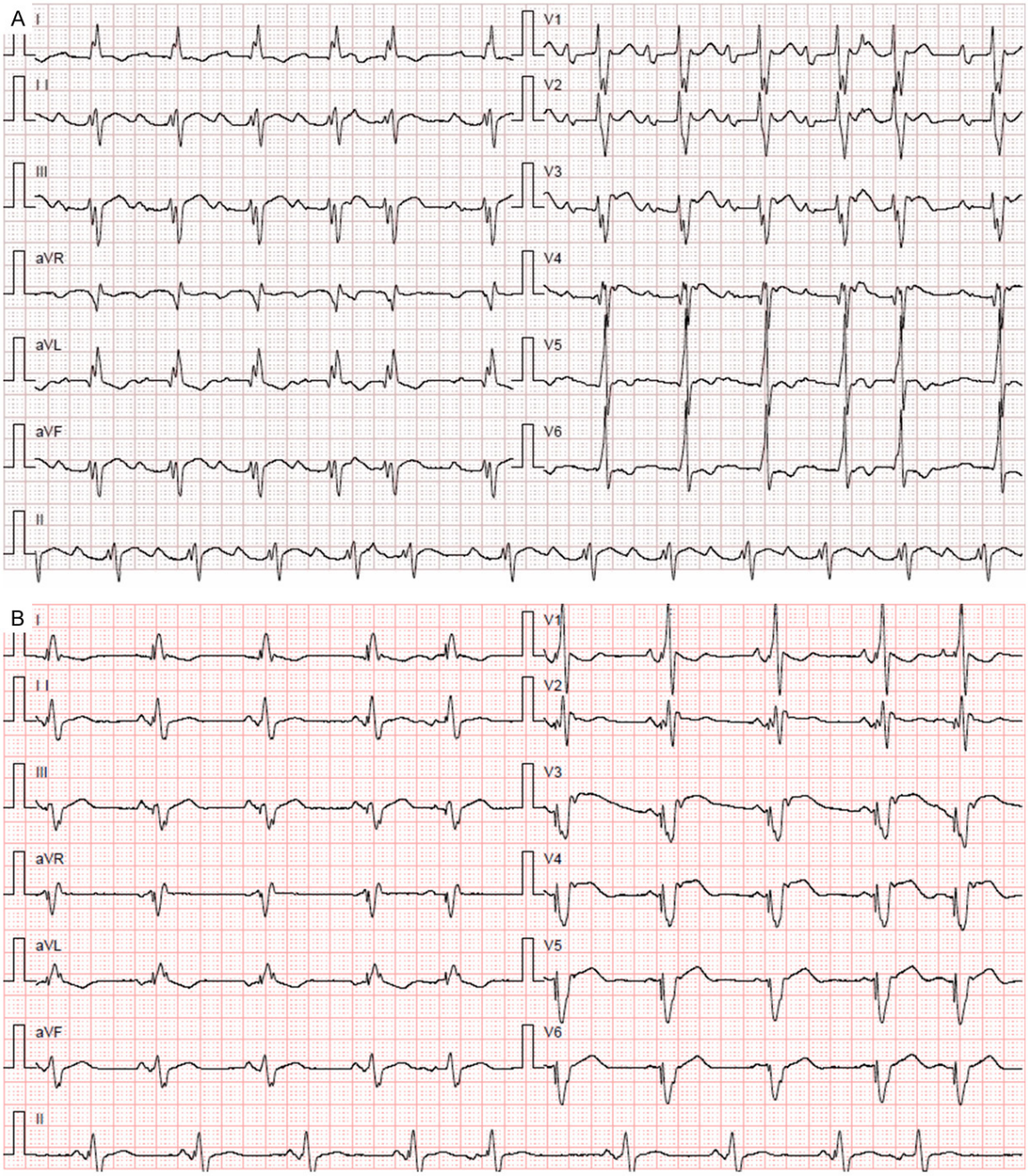

Figure 4. ECG changes after the patient had received the CRT-D implantation. A. On admission ECG showed sinus rhythm with atrial premature beat, a prolonged PR interval of 302 ms and wide QRS complexes of 158 ms. B. After CRTD implantation, the ECG showed sinus rhythm with atrial premature beat, biventricular pacing and QRS complexes of 130 ms.

higher. Venography is considered the gold standard for diagnosis venous obstruction and provides excellent characterization of venous anatomy. This case illustrates that definitive diagnosis and the location of venous obstruction can be determined by venography. The left ventricular pacing site is a principal determinant of acute CRT response in patients with ICM. The PATHCHF II study has demonstrated that pacing at left ventricular free wall sites consistently produces better short-term hemodynamic responses than does pacing at left ventricular anterior sites [3]. One of the key elements in procedural success and effectiveness

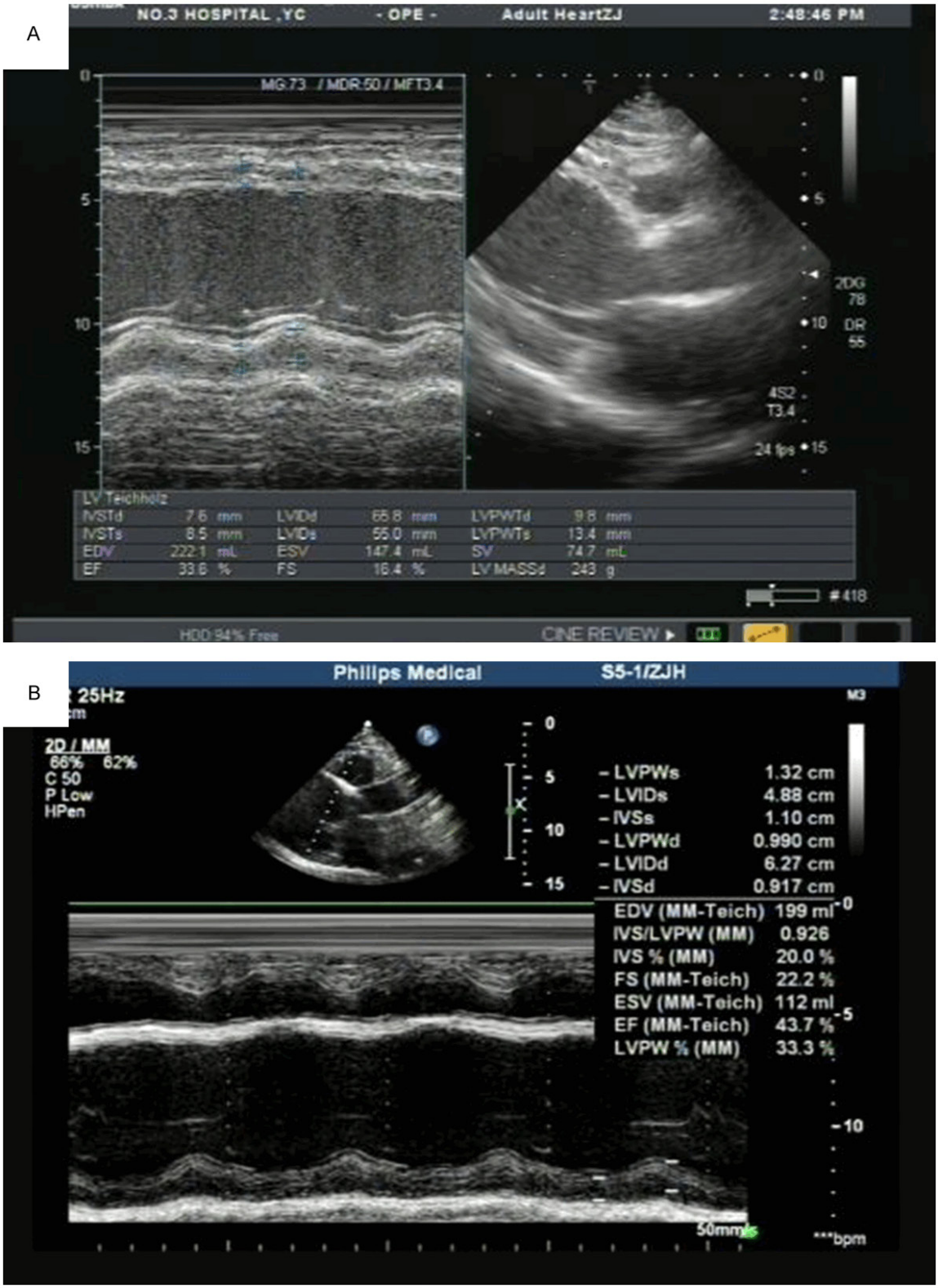

Figure 5. Transthoracic echocardiogram changes after the patient had received the CRT-D implantation. A. On admission transthoracic echocardiogram revealed enlargement of the left atrium and the left ventricular, coexisting with decreased contraction of left ventricular anterior wall with left ventricular ejection fraction of 33.6%. B. The left ventricular ejection fraction increased to 43.7% after the patient had received the CRT-D implantation.

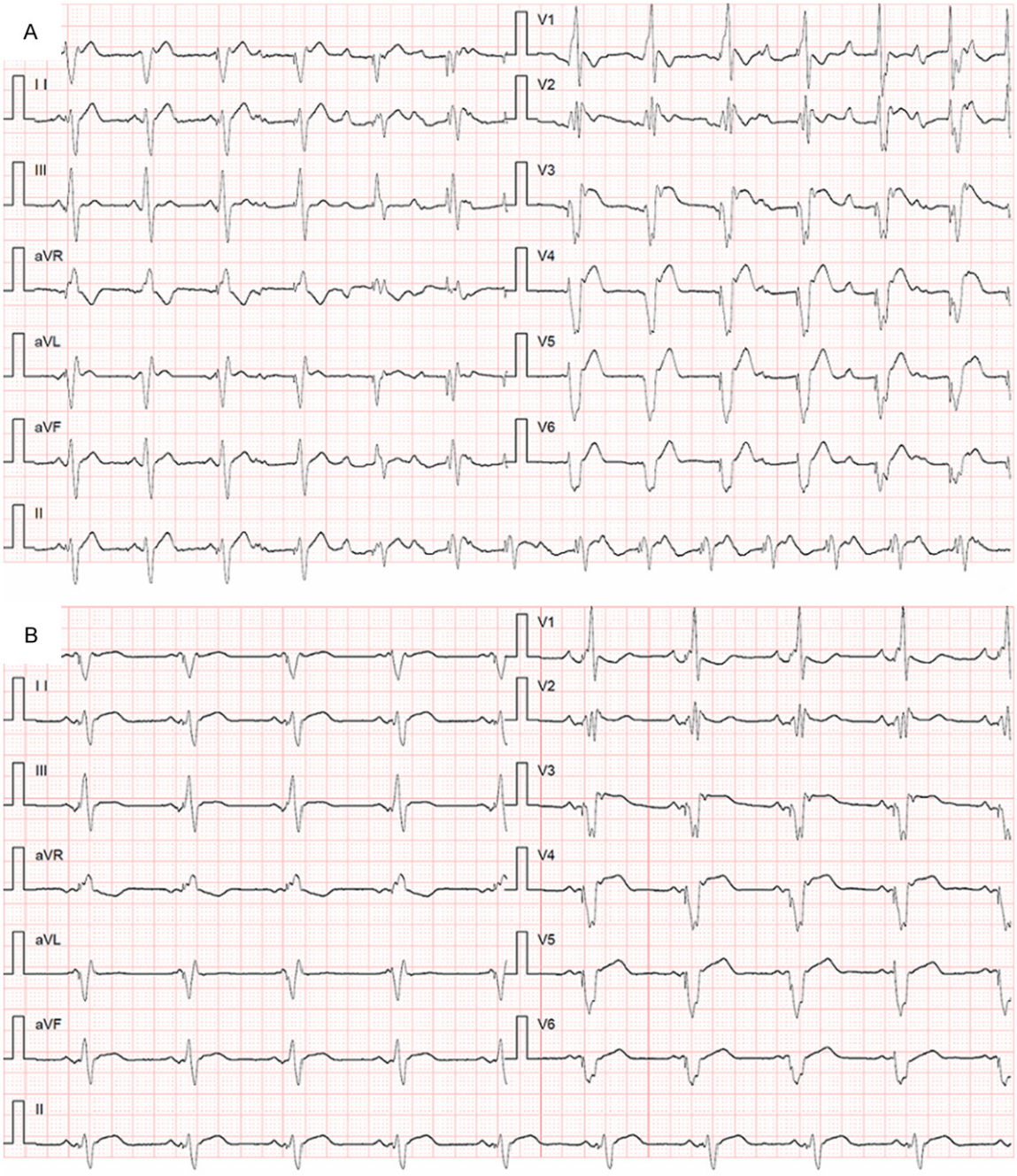

Figure 6. ECG changes when the patient had dyspnea and palpitation. A. When the patient had dyspnea and palpitation the ECG showed atrial tachycardia and loose of atrioventricular synchrony, biventricular pacing and QRS complexes of 132 ms. B. ECG showed atrial sense and biventricular pacing and QRS complexes of 128 ms after the patient had received the drug treatment.

is the ability to deliver the left ventricular lead into a stable position that is also effective electrically. Three elements of the CSO are related to successful cannulation of the CS: the size of the CSO, its entrance from the right atrium, and the presence of TV. The TV can be a significant obstacle to CS cannulation. The presence of a large TV that covers the entire CSO can in fact completely prevent the passage of a catheter from the right atrium into the CS [4].

Disclosure of conflict of interest

None.

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