

**Epicardial Ventricular Tachycardia Ablation by Percutaneous Pericardial Puncture****Running title:** Treatment of ventricular tachycardiaYuanfeng Zhou<sup>1,2</sup>, Lang Hong<sup>2</sup>, Linfeng Li<sup>2</sup>, Yun Qiu<sup>2</sup>, Menquan Hu<sup>1</sup><sup>1</sup>XinYu Iron and Steel Corporation Central Hospital, Xinyu 338001, China<sup>2</sup>The Second Department of Cardiology, People's Hospital of Jiangxi Province, Nanchang 330006, China\*E-mail: [honglangcn@126.com](mailto:honglangcn@126.com)

**Abstract:** This study aims to introduce epicardial ablation by percutaneous pericardial puncture in the treatment of ventricular tachycardia (VT). A female patient aged 45 years old with VT was enrolled in this study. Her electrocardiogram (ECG) showed a heart rate of 188 to 194 times/min with a wide QRS wave with upward dominant waves in leads I, II, III, aVF, V5, and V6. Results also showed evident incisures at the top of the R wave and a downward dominant wave in lead V1. Radiofrequency ablation by percutaneous pericardial puncture under the guidance of Carto mapping system was also performed. Conventional endocardial mapping detected the earliest activation on the right free ventricular wall. The local activation time (LAT) was 96 ms earlier than the QRS wave in lead V1 in the surface ECG, where the pacing of QRS wave from the large end is similar to the surface ECG at attacking time. Repeated discharge from the large end with cold saline (35 W to 40 W, 40 °C to 50 °C) could not cease VT. Percutaneous pericardial puncture was conducted under X-ray fluoroscopy. A 4 mm, magnetic-navigated, temperature-controlled large head was fed to the right epicardium through the pericardial cavity. LAT was 109 ms earlier than QRS wave in lead V1 in the surface ECG. After right coronary angiography, VT was ended by radiofrequency ablation under the condition of 40 W and 55 °C for 10 s, followed by consolidation discharge for 90 s. No recurrence of tachycardia was observed in the postoperative ECG after 18 months. Epicardial ablation by percutaneous pericardial puncture is an effective supplementary method for endocardial ablation.

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## 1. Introduction

The treatment of ventricular tachycardia (VT) remains a challenge because of the inaccessibility of the epicardial ventricular surface via an endocardial approach. An estimated 40% (Nagashima et al., 2011) of patients with haemodynamically unstable VT require epicardial ablation after an endocardial attempt, and 57% of those with previously failed ablations have an epicardial circuit (Schweikert et al., 2003; Sosa and Scanavacca, 2005). Traditional radiofrequency ablation therapy is performed intracardiacally because conventional endocardial mapping and radiofrequency ablation are ineffective for VT, which originates from the epicardium. A case of percutaneous pericardiocentesis ablation on a patient who suffers from epicardial VT (Tanner et al., 2005) was examined, and a favourable effect was achieved.

## 2. Materials and methods

### 2.1 Clinical data

The patient is a 45-year-old female who has suffered from repeated palpitation and chest distress for two years and a combined syncope twice. These symptoms emerged two years ago without evident predisposing causes. The initial diagnosis by the local hospital was VT, and antiarrhythmic drugs were given

as treatment, examples of which include Betaloc, Propafenone, and Amiodarone. The disease only aggravated from then on and was combined with amaurosis twice. The results of the electrocardiogram (ECG) showed the following: the patient has VT; heart rate was 188 to 194 times/min; the QRS wave was wide and malformed; leads I, II, III, aVF, V5, and V6 caused the main wave of the QRS to move upward while the V1 main wave was downward (Figure 1A); and an evident notch appeared on top of the R wave. By contrast, the heart echocardiography procedure showed the following results: weakened left ventricular diastolic function; left ventricular ejection fraction was 0.5; no evident abnormality in the X-ray chest film; and defects in hepatic and renal function, thyroid function, myocardial enzymes, myocardial enzymes, and other biochemical examinations. From these results, the admission diagnosis was VT. Given that drug therapy could not control repeated VT, and radiofrequency ablation was performed on 21 August 2007. This study was conducted in accordance with the declaration of Helsinki. This study was conducted with approval from the Ethics Committee of People's Hospital of Jiangxi Province. Written informed consent was obtained from all participants.

### 2.2 Endocardial mapping and ablation

Antiarrhythmic drugs were discontinued for more than five half lives before operation. Local anaesthesia was administered, and then the left subclavian vein was punctured to feed in 6F quadrupole coronary sinus electrode. Thereafter, the right femoral vein was punctured to feed in 3.5 mm cold saline perfusion ablation major end (Thermo-cool Navistar, Biosense Webster) to the right internal ventricle. Sliding collecting points along the right ventricular endocardium were modelled to construct the right ventricle 3D structure drawing. The major end was paced by S1S1 (180 to 280 times/min) to induce ventricular tachycardia while applying the Carto mapping system manufactured by Biosense Webster to activate mapping and pace mapping. The earliest activation point on the right ventricular free wall (Figure 1B) of the endocardium was ablated with 35 W to 40 W cold saline large end at 45 °C to 50 °C with a normal saline flow of 30 mL/min and a discharge gap of 2 mL/min.

### 2.3 Epicardial mapping and ablation

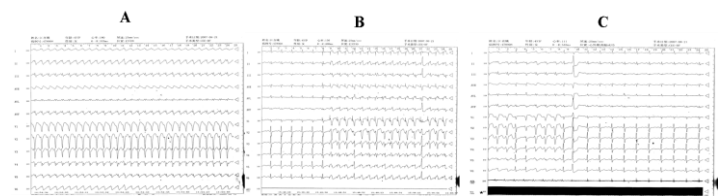
Pacing simulation was induced by applying a quadrupole right ventricle electrode to the right ventricle. Intravenous injection of 5 mg morphine and 0.1 mg fentanyl was administered. Pericardiocentesis was conducted using the percutaneous xiphoid process under X-ray. An 18 G puncture needle (length: 7 mm) was inserted through the left rib phrenic angle from the left upper side to the heart shadow. When the needle approached the heart profile, a bolus was injected with a small amount of contrast media to determine whether the needle entered the pericardial cavity. After confirming the success of pericardium cavity puncture, a j-shape steel wire and 9F sheath was inserted. This steel wire and sheath were fed to a 4 mm magnetic-navigated temperature-controlled big end via the pericardial cavity to the right ventricular epicardium. This procedure aims to establish the epicardium right ventricle 3D anatomical structure diagram and map the earliest active end on the right ventricular free wall of the epicardium. The left femoral artery was punctured using right coronary artery angiography procedure to observe the distance between the ablation large end and the coronary. Ablation was performed with 40 W at 55 °C discharge. The pacing of the right ventricular electrode was adjusted, and the simulation was repeated through S1, S1 and S1, S2 until VT ceased. ECG

### 3. Results

The map of the earliest active point on the right ventricular free wall showed that the local activation time (LAT) was 96 ms earlier than that of the surface ECG lead QRS (Figure 2A). The QRS waveform on the large-end point of pacing was similar to that in the attack time surface ECG. Repeated discharging ablation could not cease tachycardia. ECG

on attack time indicated that the QRS wave was wide and malformed, and its LAT reached up to 190 ms. The unipolar electrogram of the large-end catheter on the earliest active point in the endocardium showed that the initial part was forward and RS-shaped, which suggests that VT originated from the epicardium.

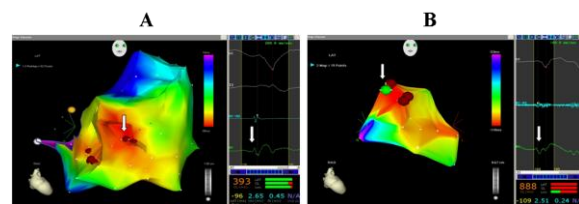
In conducting pericardiocentesis via percutaneous xiphoid process under X-ray, the earliest active point mapped on the right ventricular free wall of the epicardium was relative to that on the endocardium graph. The LAT was 109 ms earlier than the V1 lead QRS on the surface ECG. The local monopole electrogram exhibited a QS waveform (Figure 2B). Coronary angiography indicated a large distance between the large-end electrode and the right coronary artery (Figure 3). Operating ablation at 40 W and 55 °C gradually slowed down the frequency of VT to 140-145 times/min. Thereafter, VT was terminated for 10 s (Figure 1C), and discharge was consolidated for 90 s. The pacing of the right ventricle electrode for repeated simulation on S1, S1 and S1, S2 could not induce VT. No recurrence occurred post operation even after 18 months.



**Figure 1** A: Electrocardiography on attack time Ventricular tachycardia, heart rate was 188-194 times/minute, QRS wave was wide and malformative, I, II, III, aVF, V5, V6 Lead QRS main wave was upward, and there was obvious notch on the top of R wave. Meanwhile, the V1 lead main wave was downward.

B: Pacing the earliest activate point which was mapped on right ventricle free Wall endocardium, QRS waveform in pacing electrocardiogram was consistent with surface electrocardiogram on attack time.

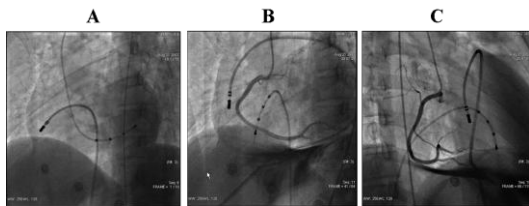
C: Epicardium successful ablation electrocardiogram



**Figure 2** A: Right ventricle endocardium three-dimensional anatomical structure diagram. The yellow point was his mark. Arrow pointed red mark was earliest activate point, LAT was 96ms earlier than

surface electrocardiogram V1 lead QRS. Repeatedly discharge could not cease tachycardia. Window M1 on the right was large end catheter distal unipolar electrogram (arrow pointed), initiation was forward wave, which indicated that ventricular tachycardia originated may be from epicardium.

B: Epicardium right ventricular 3 dimensional anatomical structure diagram. To map the earliest activate point (green spot where the arrow pointed) on epicardium right ventricle free wall where was related to endocardium graph. LAT was 109ms earlier than V1 lead QRS wave in surface electrocardiogram, and local monopole electrocardiogram was downward. Ablation by discharge for 20s, thus, ventricular tachycardia was ceased.



**Figure 3** A: Endocardium ablation image LAO45°; B: Epicardium ablation image LAO42°; C: Epicardium ablation RAO30°, right coronary angiography indicated that epicardium ablation zone was related to endocardium, which had certain distance to right coronary.

#### 4. Discussion

The radiofrequency ablation efficacy of VT is closely related to the occurrence mechanism, position, and combined underlying heart diseases. Epicardial VT usually occurs in cases of myocardial infarction (Yu et al., 2013), dilated cardiomyopathy (Soejima et al., 2004), and Chagas disease (Sosa et al., 1998, 2000). Some cases of left ventricular repetitive monomorphic tachycardia also originate from the epicardium (Arruda et al., 1996).

Some studies observed that the ablation injury range caused by a standard 4 mm large-end catheter has a diameter of  $7.8 \pm 2.8$  mm, short diameter of  $6.5 \pm 1.6$  mm, and depth of  $2.67 \pm 0.35$  mm (D'Avila et al., 2002). By contrast, the ablation injury range of the cold saline irrigated-tip large end has a diameter of 12.4 mm and an ablation depth of 8 mm (Nakagawa et al., 1998). The ventricular myocardial thickness of a normal adult is 2 mm to 5 mm, whereas the left ventricular myocardium thickness is 8 mm to 11 mm in the diastolic period and 14 mm to 16 mm (Nagueh et al., 2009) in the systolic period. Therefore, conventional endocardium ablation could not reach epicardial lesions in a thicker myocardium, so that induced ablation failure occurs. Moreover, interlaced muscle trabecular

on the ventricular endometrium surface would affect ablation efficacy. In this research, the patient lesions were on the right ventricular outer membrane. Theoretically, the ablation injury depth of the cold saline infusion by the large-end endometrium could reach the lesions, so that ablation failure was further considered. Previous reported cases on epicardium VT applied thoracoscopic ablation (Watanabe et al., 1998) and coronary vein ablation (Stellbrink et al., 1997). In a study by Campos et al. (2012), successful ablation was achieved in nine out of 16 patients (56%) targeting the LSV (five patients), adjacent LV endocardium (two patients), or both (two patients). Yamada et al. (2008) reported a case of VT with a possible myocardial fibre travelling from the origin in the aortic sinus cusp (ASC) to the epicardium of the ventricular outflow tract. This case may provide a clinical implication for the catheter ablation of VT originating from the ASC. Sosa et al. (1996) initially reported on radiofrequency ablation via the percutaneous xiphoid process, which had facilitated the development of new approaches to epicardium VT ablation. No similar clinical application has been reported in China. From this case, we have learned the following:

Repeated discharge could not cease tachycardia. ECG indicated that the epicardium VT QRS was wide and malformed, especially at the starting section of the QRS wave. The large end on the unipolar electrogram of the earliest active point in the endocardium was forward and RS-shaped, which indicates that VT originated from epicardium.

A suitable puncture needle should be sufficiently long with a short needle tip slope to reach the heart when pericardiocentesis is performed via the percutaneous xiphoid process. The pericardial cavity of normal human beings is usually slightly slurry (approximately 15 mL to 30 mL). Under the condition of non-pericardial effusion, pericardial cavity clearance is narrow. When the needle sticks to the wall layer, it would closely attach without a breakthrough feeling, so that myocardial and vascular injury could easily occur. To protect the myocardium and coronary artery, a needle with a short needle tip slope should be used.

Correct determination of puncture success. The tip of the needle should be carefully inserted close to the heart. Thereafter, a bolus was injected to administer---a small amount of contrast media to determine whether the needle tip entered the pericardial cavity. The contrast media would then appear fan-shaped, diffusing around the heart shadow. A J-shaped guide wire is fed through the puncture needle to reach the right margin of heart from the left along the heart shadow. After puncture success is confirmed, inlet sheath could be performed.

D'Avila et al. (2004) compared the depth and length-diameter of ablation injury on a normal

myocardial epicardium induced by a 4 mm temperature-controlled large end versus, a cold saline perfusion large end, and the results were  $3.7 \pm 1.3$  mm,  $11 \pm 2.7$  mm and  $6.7 \pm 1.7$  mm,  $13.7 \pm 3.5$  mm respectively. He believed that the fat pad is thick, making the cold saline perfusion large end more effective. However, the continuous inflow of normal saline from the cold saline perfusion large end could induce pericardial tamponade. In this research, a 4 mm temperature-controlled large end was applied. Large-end electrode indicated that ablation was effective at 40 W, 55 °C. Further studies should be conducted on whether the size, power, and temperature of the large end possess higher requirements.

Before epicardial ablation, the distance between the ablation section and coronary must be determined. The diameter of epicardial large-end ablation injury could reach  $11 \pm 2.7$  mm (Sosa et al., 1996). Thus, this study holds that when the distance is greater than the diameter, ablation can be safely performed.

Epicardial ablation stimulates the pericardium and causes severe pain, which would affect the operation procedure. In this research, the patient was administered with analgesics such as morphine, and fentanyl, but she still felt discomfort. Thus, administering general anaesthesia in this kind of operation should be considered to achieve a better effect

Pericardial effusion (Abhishek et al., 2011; Ma et al., 2012) may be caused by vascular injured by the ablation catheter during operation. Thus, a conventional echocardiography check should be conducted after operation. And if necessary, indwelled pericardial catheter drainage should be performed for 12 h to 24 h.

In conclusion, epicardial radiofrequency ablation through the percutaneous puncture pericardium is an effective complementary method that will enhance the success rate of VT treatment.

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