Pressure frequency characteristics of the pericardial space and thorax during subxiphoid access for epicardial ventricular tachycardia ablation

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BACKGROUND Nonsurgical subxiphoid pericardial access may be useful in ventricular tachycardia ablation and other electrophysiologic procedures but has a risk of right ventricular puncture.

OBJECTIVE The purpose of this study was to identify a signature pressure frequency that would help identify the pericardial space and guide access.

METHODS The study consisted of 20 patients (8 women and 12 men; mean age 59.1 ± 14.2 years; left ventricular ejection fraction 25.2% ± 12.2%; failed 1.8 ± 0.5 endocardial ablations; unresponsive to 2.0 ± 1.0 antiarrhythmic drugs; 6 ischemic cardiomyopathy, 12 nonischemic cardiomyopathy, 2 normal heart; 4 previous sternotomy) undergoing epicardial ventricular tachycardia ablation. After pericardial access was obtained, a 10Fr long sheath was used to record pressure inside the pericardium and pleural space. Pressures were analyzed using a fast Fourier transform to identify dominant frequencies in each chamber.

RESULTS Mean pressures in the pleural space and the pericardium were not different (7.7 ± 1.9 mmHg vs 7.8 ± 0.9 mmHg, respectively). However, the pericardial space in each patient demonstrated two frequency peaks that correlated with heart rate (1.16 ± 0.21 Hz) and respiratory rate (0.20 ± 0.01 Hz), whereas the pleural space in each patient had a single peak correlating with respiratory rate (0.20 ± 0.01 Hz).

CONCLUSION The pericardial space demonstrates a signature pressure frequency that is significantly different from the surrounding space. This difference may make minimally invasive subxiphoid pericardial access safer for nonsurgeons and may have important implications for electrophysiologic procedures.

KEYWORDS Catheter ablation; Epicardial ablation; Pressure frequency; Subxiphoid access; Ventricular tachycardia

ABBREVIATIONS ICE = intracardiac echocardiography; **LV** = left ventricle; **RV** = right ventricle; **VT** = ventricular tachycardia

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Introduction

Access to the pericardial space may allow for multiple innovative electrophysiologic procedures, including ventricular tachycardia (VT) ablation, atrial fibrillation ablation, left atrial appendage removal, and left ventricular (LV) pacing for cardiac resynchronization.[1–8] In particular, epicardial VT ablation may better target VT substrate compared with endocardial ablation alone. In a series of 231 patients undergoing endocardial VT, only 53% of patients were free of VT shocks at 6 months.[9] Although no series has compared the long-term success rates of endocardial to epicardial ablation, one reason for the failure of endocardial ablation is the significant portion of VT circuits that can be epicardial. For example, Sacher et al[10] found that 72% of patients with ischemic or nonischemic VT had at least some epicardial substrate.

Subxiphoid access allows for minimally invasive access to the pericardial space and the epicardium of the heart but is fraught with a 1% to 20% right ventricular (RV) perforation rate.[11–13] Although most of these inadvertent RV punctures do not require surgical repair or abortion of the ablation procedure, this risk can be a barrier to broader adoption of epicardial

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electrophysiologic techniques, particularly in centers that have not performed many epicardial procedures.

When accessing the subxiphoid space using a nonsurgical technique, the needle goes through the diaphragm, briefly into the thorax, and then into the pericardial space.[14] However, if the needle advances beyond the 1-mm-thick pericardial space, it can perforate the RV. One way to increase the safety of subxiphoid epicardial access would be to define a physiologic signature of the pericardial space that clearly differentiates it from the thorax. In transseptal access, for example, the pressure differences between the right and left atria and aorta are used to increase safety.

Because multiple studies have shown that thorax and pericardial space mean pressures are identical, pressure itself cannot be used to differentiate thorax from pericardial space.[15] Although noting the difference of pressures between the RV and pericardial space can help identify perforations, finding a signature of the pericardial space to inform clinicians when they are safely in the pericardium and when further needle advancement is dangerous would be more helpful. The pericardial space is actually bordered by two structures: the heart and the lung field. The inferior pericardial sac is also attached to the diaphragm. The heart typically moves at 60 to 90 bpm. The lung field and diaphragm moves at 12 cycles per minute in intubated patients (Figure 1). Thus, although the pressure is the same in the thorax and pericardial space, the pressure frequency signatures should be different. In particular, we expected that the thorax frequency would be 0.2 Hz (12 breaths per minute).

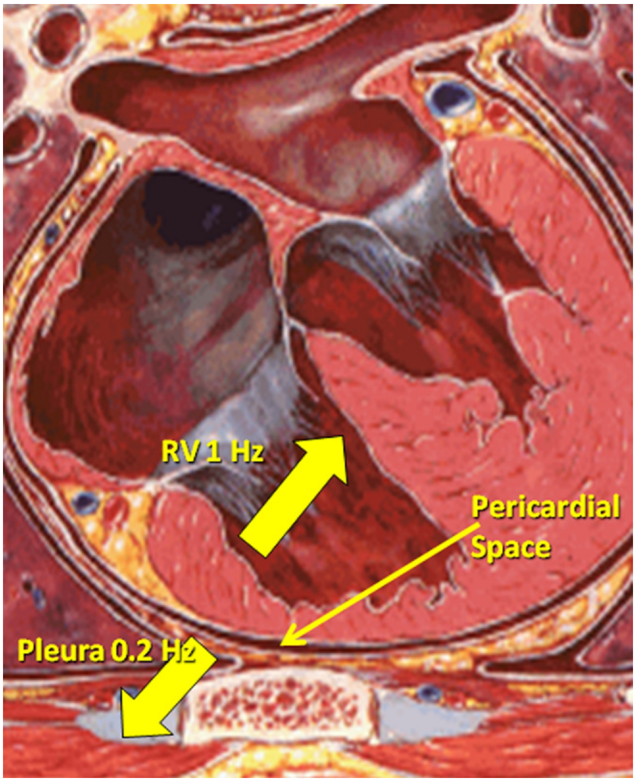

Figure 1 Graphic illustration suggesting that pericardial sac frequency is influenced by respiration and heart rate. RV = right ventricle.

Inside the heart, the frequency peak should be 0.8 to 2 Hz (50–120 bpm), whereas a double frequency peak would be seen in the pericardial space at both approximately 0.2 Hz and 1 Hz. Initial studies in three patients as well as computer simulations suggest this hypothesis is accurate.[16] Accordingly, we sought to determine if these signature pressure frequencies existed in 20 human patients undergoing subxiphoid epicardial access for VT ablation.

Methods

Patients

Twenty sequential patients underwent epicardial ablation for drug-refractory VT that had failed previous endocardial ablation (mean 1.8 $\pm$ 0.5, range 1–3). The 20 patients (12 male and 8 female) had a mean age of 59.1 $\pm$ 14.2 years and mean LV ejection fraction of 25.2% $\pm$ 12.2%. Four patients had undergone previous cardiac surgery. Twelve patients had nonischemic cardiomyopathy, 6 ischemic cardiomyopathy, and 2 idiopathic ventricular arrhythmia in the setting of preserved ejection fraction. All patients had a history of documented symptomatic VT. Patients had failed a mean of 2.0 $\pm$ 1.0 antiarrhythmic drugs, including amiodarone in 16 (80%). Eighteen patients had an implantable cardioverter-defibrillator and had received at least one appropriate shock (mean 11 $\pm$ 7 in the last 6 months). Eighteen patients were taking beta-adrenergic blockers, 14 angiotensin-converting enzyme inhibitors, and 15 HMG-CoA reductase inhibitors. Prior sternotomy was not an exclusion criterion, but patients who required a pericardial window for access were excluded from the study. Approval was obtained for all patients in accordance with the policy of the University of Virginia Institutional Review Board.

Intubation

All patients were intubated and mechanically ventilated at 12 breaths per minute using a Fabius GS anesthesia machine (ARYEO119) to achieve an end-tidal CO_2 of 35 to 45 mmHg. Tidal volumes and positive end-expiratory pressure were adjusted by the operator. Positive end-expiratory pressure was always set between 2 and 5 cmH_2O. Anesthesia was induced with propofol or etomidate as well as a short-acting muscle paralytic at the discretion of the attending anesthesiologist but was stopped 30 minutes prior to epicardial ablation. Deep general anesthesia was maintained with either propofol or volatile anesthetics (sevoflurane or desflurane). Fentanyl was administered at the discretion of the anesthesia care provider.

Access

The pericardial space was accessed using a 17-gauge epidural (Tuohy) needle as described by Sosa et al.[14] Intracardiac echocardiography (ICE) in the RV and fluoroscopy with contrast were used to guide access. An anterior approach was used initially in patients without prior sternotomy. In patients with a prior sternotomy or a failed anterior approach, a posterior approach was used.