ESC European Society of Cardiology

Europace (2025) **27**, euaf055
https://doi.org/10.1093/europace/euaf055

EHRA DOCUMENT

EHRA European Heart Rhythm Association

Epicardial ventricular arrhythmia ablation: a clinical consensus statement of the European Heart Rhythm Association of the European Society of Cardiology and the Heart Rhythm Society, the Asian Pacific Heart Rhythm Society, the Latin American Heart Rhythm Society, and the Canadian Heart Rhythm Society

Arash Arya (Chair)[1]*, Luigi Di Biase (Co-Chair)[2], Victor Bazán [3], Antonio Berruezo [4], Andrea d'Avila [5], Paolo Della Bella [6], Andres Enriquez [7,8], Mélèze Hocini [9], Josef Kautzner [10], Hui-Nam Pak [11], William G. Stevenson [12], Katja Zeppenfeld [13], and Alireza Sepehri Shamloo (Writing Group Coordinator)[14]

Document Reviewers: Christian Meyer (Review Coordinator)[15], Christian de Chillou[16,17], Thomas Deneke[18], Marta de Riva[19], Andreu Porta-Sanchez[20], John Sapp[21], Boris Schmidt[22], Kalyanam Shivkumar[23,24], Philipp Sommer[25], Kyoko Soejima[26], Gregory E. Supple[27], Arthur Wilde[28,29], and Giulio Zucchelli[30]

[1]Department of Cardiology, University Hospital Halle, Martin-Luther University Halle-Wittenberg, Ernst-Grube-Str. 40, 06120 Halle (Saale), Germany; [2]Cardiac Arrhythmia Center, Division of Cardiology at Montefiore-Health System, Albert Einstein College of Medicine, New York, USA; [3]Unidad de Arritmias, Servicio de Cardiología, Hospital Universitario Germans Trias i Pujol, Badalona, Barcelona, Spain; [4]Arrhythmia Department, Teknon Heart Institute, Teknon Medical Center, Barcelona, Spain; [5]Harvard-Thorndike Arrhythmia Institute and Division of Cardiovascular Diseases, Beth Israel Deaconess Medical Center and Harvard Medical School, Boston, MA, USA; [6]Department of Cardiac Arrhythmia and Electrophysiology, San Raffaele University-Hospital, Milan, Italy; [7]Section of Cardiac Electrophysiology, Hospital of the University of Pennsylvania, Philadelphia, PA, USA; [8]Division of Cardiology, Queen's University, Kingston, Ontario, Canada; [9]IHU LIRYC, Electrophysiology and Heart Modeling Institute, Cardiac Electrophysiology and Stimulation Department Fondation Bordeaux Université and Bordeaux University Hospital (CHU), Pessac-Bordeaux, France; [10]Department of Cardiology, Institute for Clinical and Experimental Medicine, Prague, Czech Republic; [11]Division of Cardiology, Department of Internal Medicine, Yonsei University College of Medicine and Yonsei University Health System, Seoul, Republic of Korea; [12]Department of Cardiology, Vanderbilt Heart and Vascular Institute, Nashville, TN, USA; [13]Department of Cardiology, Leiden University Medical Center, Leiden, The Netherlands; [14]Department of Cardiology, Deutsches Herzzentrum der Charité-Medical Heart Center of Charité, German Heart Institute Berlin, Berlin, Germany; [15]Division of Cardiology, Angiology, Intensive Care Medicine, EVK Düsseldorf, cNEP, cardiac Neuro- and Electrophysiology Research Consortium, Düsseldorf, Germany; [16]Department of Cardiology, CHRU-Nancy, Université de Lorraine, Nancy, France; [17]IADI, INSERM U1254, Université de Lorraine, Nancy, France; [18]Clinic for Arrhythmology, University Heart Center Nuremberg, University Hospital Nuremberg, South Campus, Nuremberg, Germany; [19]Department of Cardiology, Willem Einthoven Center of Arrhythmia Research and Management, Leiden University Medical Center,

* Corresponding author. Tel: 0049 345 557 4910. *E-mail address*: arash.arya@uk-halle.de

Developed in partnership with and endorsed by the European Heart Rhythm Association (EHRA), a branch of the European Society of Cardiology (ESC), the Heart Rhythm Society (HRS), the Asia Pacific Heart Rhythm Society (APHRS), the Latin American Heart Rhythm Society (LAHRS), and the Canadian Heart Rhythm Society (CHRS).

Leiden, The Netherlands; [20]Hospital Clínic de Barcelona, Universitat de Barcelona, Institut d'Investigacions Biomédiques August Pi i Sunyer (IDIBAPS), Barcelona, Catalonia, Spain; [21]QEII Health Sciences Centre, Dalhousie University, Halifax, Canada; [22]Cardionagiologisches Centrum Bethanien, Agaplesion Markus Krankenhaus, Frankfurt/Main, Germany; [23]Center for Interventional Programs, University of California, Los Angeles, CA, USA; [24]Cardiac Arrhythmia Center, UCLA Health System, Los Angeles, CA, USA; [25]Clinic of Electrophysiology, Heart- and Diabetes Center NRW, Ruhr University Bochum, Bad Oeynhausen, Germany; [26]Department of Cardiovascular Medicine, Kyorin University, Tokyo, Japan; [27]Cardiac Electrophysiology Section, University of Pennsylvania Perelman School of Medicine, Philadelphia, PA, USA; [28]Department of Cardiology, Amsterdam UMC Location University of Amsterdam, Meibergdreef 9, Amsterdam, The Netherlands; [29]Amsterdam Cardiovascular Sciences, Heart Failure and Arrhythmias, Amsterdam, The Netherlands; and [30]Second Division of Cardiology, Cardiothoracic and Vascular Department, Pisa University Hospital, Pisa, Italy

Received 5 March 2025; accepted after revision 10 March 2025; online publish-ahead-of-print 31 March 2025

Epicardial access during electrophysiology procedures offers valuable insights and therapeutic options for managing ventricular arrhythmias (VAs). The current clinical consensus statement on epicardial VA ablation aims to provide clinicians with a comprehensive understanding of this complex clinical scenario. It offers structured advice and a systematic approach to patient management. Specific sections are devoted to anatomical considerations, criteria for epicardial access and mapping evaluation, methods of epicardial access, management of complications, training, and institutional requirements for epicardial VA ablation. This consensus is a joint effort of collaborating cardiac electrophysiology societies, including the European Heart Rhythm Association, the Heart Rhythm Society, the Asia Pacific Heart Rhythm Society, the Latin American Heart Rhythm Society, and the Canadian Heart Rhythm Society.

Keywords Epicardial access • Ventricular arrhythmias • Ventricular fibrillation • Ventricular tachycardia • Cardiomyopathies • Catheter ablation • Electrophysiology procedures • Clinical consensus statement

Table of contents

Section 1: Introduction

Preamble

Epicardial access during electrophysiology (EP) procedures offers valuable insights and therapeutic options for managing ventricular arrhythmias (VAs), and is typically needed in 23–30% of patients undergoing VA ablation.[1,2] The need for epicardial VA ablation emerged from an increasing understanding that the critical sites of many VAs originate from the intramural myocardium and epicardium, making them less accessible for effective endocardial ablation.[3–6]

Epicardial VA ablation is most effectively performed in high-volume tertiary centres and is often combined with endocardial ablation to achieve optimal substrate modification and improve patient outcomes. Meticulous technique, careful patient selection, and vigilant monitoring are paramount to ensure procedural success and patient safety.

While epicardial ablation can improve the success of catheter ablation, the procedure is associated with an increased risk of complications, requires significantly greater resources and preparation than endocardial ablation alone, and is only considered in limited clinical scenarios and experienced referral centres.[7] Current indications for epicardial VA ablation include but are not limited to recurrent VA after previously failed endocardial ablation when electrocardiographic, imaging, or mapping findings suggest the presence of epicardial substrates. In selected patients, however, a first-line endocardial–epicardial approach may be appropriate.

This consensus is a joint effort of collaborating cardiac electrophysiology societies, including the European Heart Rhythm Association (EHRA), the Heart Rhythm Society (HRS), the Asia Pacific Heart Rhythm Society (APHRS), the Latin American Heart Rhythm Society (LAHRS), and the Canadian Heart Rhythm Society (CHRS).

Scope of the document

The current clinical consensus statement on epicardial VA ablation aims to guide clinicians in managing epicardial VA ablation across various scenarios. It offers structured advice and a systematic approach to patient management. Specific sections are devoted to anatomical considerations, criteria for epicardial access and mapping evaluation, methods of epicardial access, management of complications, training, and institutional requirements for epicardial VA ablation.

Organization of the writing committee

The EHRA, as the leading society, nominated the chair and the co-chair of the document based on their internationally recognized expertise in the field. The writing group was defined based on a list of representatives put forward by each organization. All members are internationally recognized experts. The members were qualified in order of preference if they did not meet any of the following: part-time employment

or salary from a related company, significant stock ownership, holding of a patent that generates significant revenues, and receipt of significant royalties for intellectual property related to the topic of the scientific paper. The entire group was comprised of 13 members, and they were appointed to section writing teams based on preference and expertise, aiming to cover specific content. All members provided disclosure statements to assess potential conflicts of interest. Details are available in supplementary material online.

Methods

After a comprehensive literature review and evaluation of existing evidence, a detailed survey comprising 112 questions was distributed to all members to capture common practices and preferences in the management of patients undergoing epicardial VA ablation. Considering the survey results, the writing group proposed an initial list of practical advice. This advice was refined through several face-to-face and web-based meetings, during which the proposed tables of advice were discussed and modified. A final list of clinical advice was then prepared for the voting process. During voting, each member had the option to agree (with or without modification), disagree, or abstain. Every proposed advice was included only if the voting results (excluding abstention) were at least 70% in support.

It should be emphasized that the current document is not intended as a guideline and aims to document the current expert consensus in the dedicated narrow field of VA epicardial ablation. Healthcare professionals should refer to the latest guidelines for the overall management of patients with VAs.[8–10] The classification of various categories of advice, along with their respective definitions, is outlined in *Table 1*. Additionally, the evidence supporting each category of advice has been organized based on the type, quality, and quantity of the respective sources (*Table 2*). Finally, the terms and abbreviations used throughout the consensus document are summarized in *Table 3*.

Document review and approval

A review committee conducted a peer review of the draft document. All peer reviewers were required to submit a declaration of interest. The final document was officially reviewed and endorsed by each partnering organization.

Brief history of epicardial access in electrophysiology

Inspired by the need to better treat VA in Chagas disease, Sosa *et al.*[11] introduced the subxiphoid percutaneous epicardial mapping and/or ablation in 1996. In the 1990s, d'Avila found an article on laser epicardial photocoagulation by Svenson suggesting that ventricular tachycardia (VT) was related to an epicardial circuit in some patients with post-myocardial infarction VT in whom the endocardial peeling technique had failed.[12] Since one mechanism of scar formation in Chagas disease is chronic distal ischaemia in the watershed regions, and VT typically originates from the inferolateral left ventricle (LV), a fax was immediately sent to Sosa suggesting that epicardial circuits would likely dominate in Chagas' VT, which would explain the poor outcomes of endocardial ablations at that time.

Sosa observed late potentials on ventricular electrograms recorded from the coronary sinus in patients with Chagas' disease. Soon, it became clear that alternative access to the pericardial space, other than surgical access, would be needed if the procedure was to be done in the EP Lab. In 1995, the Tuohy needle, a specially designed needle to prevent vascular and neural damage during epidural anaesthesia, was brought to the attention of Scanavacca, an experienced Brazilian electrophysiologist. Scanavacca thought that it would be worth trying a 'dry' epicardial puncture with the needle connected to a unipolar channel to identify an 'injury current' as soon the needle reached the epicardial surface. The first attempt was successful in a patient with several previous EP procedures. Sosa was amazed by what Scanavacca did. At that moment, it became clear that the epicardial space would become an integral part of cardiac ablation. Since then, more than 2000 papers have been published on various aspects of this approach, solidifying it as a critical step in enhancing the success of VA ablations.

Table 1 Classification and definition of different categories of advice

Definition	Categories of Advice
Evidence or general agreement that a given measure is clinically useful and appropriate	Advice TO DO
Evidence or general agreement that a given measure may be clinically useful and appropriate	May be appropriate TO DO
Evidence or general agreement that a given measure is not appropriate or harmful	Advice NOT TO DO
No advice can be given because of lack of data or inconsistency of data. The topic is important to be addressed	Areas of uncertainty

Table 2 Type and strength of supporting evidence

Type of supporting evidence	Strength of evidence	Icons
Published data	>1 high quality RCT Meta-analysis or high quality RCT	
	High quality RCT > 1 moderate quality RCT Meta-analysis or moderate quality RCT	
	High quality, large observational studies	
Expert opinion	Strong consensus > 90% of writing group supports advice	>90% agree
	Consensus > 70% of writing group supports advice	>70% agree

Table 3 Abbreviations

Term (abbreviation)	Definition
AF	Atrial fibrillation
APHRS	Asia Pacific Heart Rhythm Society
ARVC/D	Arrhythmogenic right ventricular cardiomyopathy/dysplasia
CHRS	Canadian Heart Rhythm Society
CT	Computed tomography
CF	Contact force
CMR	Cardiac magnetic resonance imaging
D5W	Dextrose 5% in Water
ECG	Electrocardiogram
EHRA	European Heart Rhythm Association
ESC	European Society of Cardiology
HRS	Heart Rhythm Society
ICD	Implantable cardiac defibrillator
ICE	Intracardiac echocardiography
ICM	Ischaemic cardiomyopathy
LAHRS	Latin American Heart Rhythm Society
LIMA	Left internal mammary arterial
LGE	Late gadolinium enhancement
LV	Left ventricle
NS	Normal saline
NICM	Non-ischaemic cardiomyopathy
RCT	randomized clinical trial
RF	Radiofrequency
RV	Right ventricle
VA	Ventricular arrhythmia
VT	Ventricular tachycardia

Section 2: Anatomy of the pericardium

General structure

The human pericardium is a double-layered fibro-serous membrane that surrounds the heart and the root of the great vessels. It consists of an outer fibrous envelope (the fibrous pericardium, *Figure 1A*) and an inner serous sac (the serous pericardium), which has a visceral layer adherent to the heart (the epicardium, *Figure 1B*) and a parietal layer lining the fibrous pericardium. The pericardial space is a virtual space between the visceral and parietal layers of the pericardium and typically contains 15–50 mL of serous fluid generated by the mesothelial lining. By separating the heart from adjacent structures (descending aorta, lungs, diaphragm, oesophagus, trachea), the pericardial space allows free cardiac motion within this sac.[13]

The pericardial reflections around the great vessels delineate two hilar orifices: the superior hilum (arterial pole) around the ascending aorta and pulmonary trunk and the posterior hilum (venous pole) around the pulmonary and caval veins (*Figure 2*).[14] Both hila are the sites of entrance and exit of extracardiac nerves and vessels to and from the subepicardial layer of the heart. Because the pericardial reflections are located posteriorly, the anterior, apical, and lateral surfaces of both ventricles are freely accessible within the pericardial space. The fibrous pericardium is attached inferiorly to the central tendon of the diaphragm and anteriorly to the posterior sternal surface by the superior and inferior sternopericardial ligaments, which help maintain the general position of the heart inside the thorax.

Pericardial sinuses and recesses

The reflection of the serous pericardium around the great vessels creates the pericardial sinuses and recesses (*Figure 3*—upper panel and Supplementary material online, *Figure S3.1*).[15]

- Transverse sinus: a transverse passage between the left and right sides of the pericardial cavity. It lies behind the aorta and the main pulmonary artery, bordered superiorly by the right pulmonary artery and inferiorly by the dome of the left atrium. The transverse sinus is separated from the oblique sinus by a pericardial reflection that extends between the left superior and right superior pulmonary veins and gives rise to several recesses that extend as diverticula between the major vessels (superior aortic, inferior aortic, right and left pulmonic recesses).
- Oblique sinus: a cul-de-sac located behind the left atrium, bounded on the right by the inferior vena cava and on the left by the pericardial reflection connecting the two left pulmonary veins.
- Superior aortic (or aortocaval) recess: the superior extension of the transverse sinus, located between the aorta and superior vena cava.
- Inferior aortic recess: located between the right lateral aspect of the ascending aorta and the right atrium.
- Right and left pulmonic recesses form the lateral extensions of the transverse sinus. The right pulmonic recess is inferior to the proximal right pulmonary artery, bounded by the reflection of serous pericardium extending from the right pulmonary artery to the superior vena cava. The left pulmonic recess is bounded superiorly by the left pulmonary artery, inferiorly by the left superior pulmonary vein, and medially by the ligament of Marshall.
- Postcaval recess extends along the superior vena cava as a lateral extension of the superior sinus, limited superiorly by the right pulmonary artery and inferiorly by the right superior pulmonary vein.
- Right and left pulmonary venous recesses are located between the superior and inferior pulmonary veins on each side, with significant variation in depth and width.

Anatomical relations

A thorough understanding of the anatomical relations of the pericardium within the mediastinum is crucial to minimizing the risks associated with epicardial access and ablation (*Figure 3*—lower panel and Supplementary material online, *Figure S3.2*).

- **Anterior relations**: Anteriorly, the pericardium is in contact with the anterior chest wall, pleura, and lungs, with approximately one-third of its surface located rightward and two-thirds leftward from the midline. A potentially life-threatening complication of epicardial access is the injury to the left internal mammary artery (LIMA) (see Section 4—*Figure 6* and Section 7). This artery originates from the left subclavian artery. It runs along the inner surface of the anterior chest wall, coursing between the trans vs. thoracis and intercostal muscles at the third intercostal space, where it can be found 10–20 mm lateral of the sternum. As it descends along the anterior chest wall, it gives rise to the anterior intercostal arteries that feed the intercostal muscles and overlying cutaneous tissues. As it approaches the 6th or 7th intercostal space, it bifurcates into the musculophrenic artery and the superior epigastric artery.
- **Inferior relations**: Through the diaphragm, the pericardium is related to the diaphragmatic surface of the liver (left lobe) and part of the gastric fundus. The Larrey's space or left sternocostal triangle is an anatomic area bordered by the sternum anteriorly, the diaphragm inferiorly, and the pericardium posteriorly, and it is the most common target for percutaneous epicardial access. This space is mainly avascular, except for the left superior epigastric artery, which runs along the left costal margin (see Section 7).

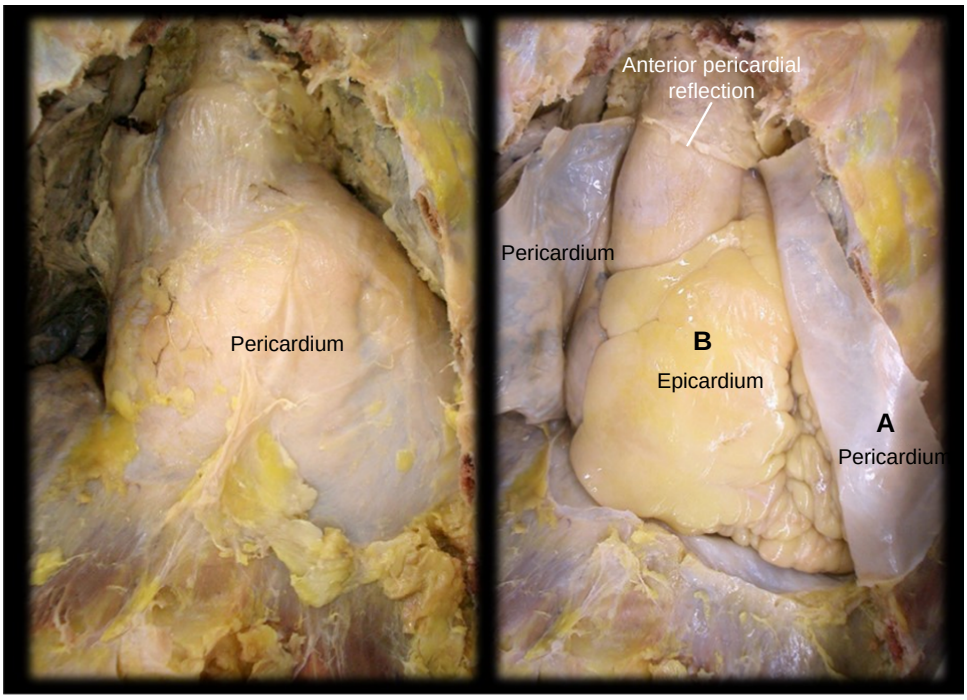

Figure 1 Anatomy of the human pericardium (anterior view)—fibrous pericardium (*A*) and serous pericardium with epicardium (*B*). Image adapted with permission from the Image Courtesy of the UCLA Cardiac Arrhythmia Center, Amara-Yad Project Collection.

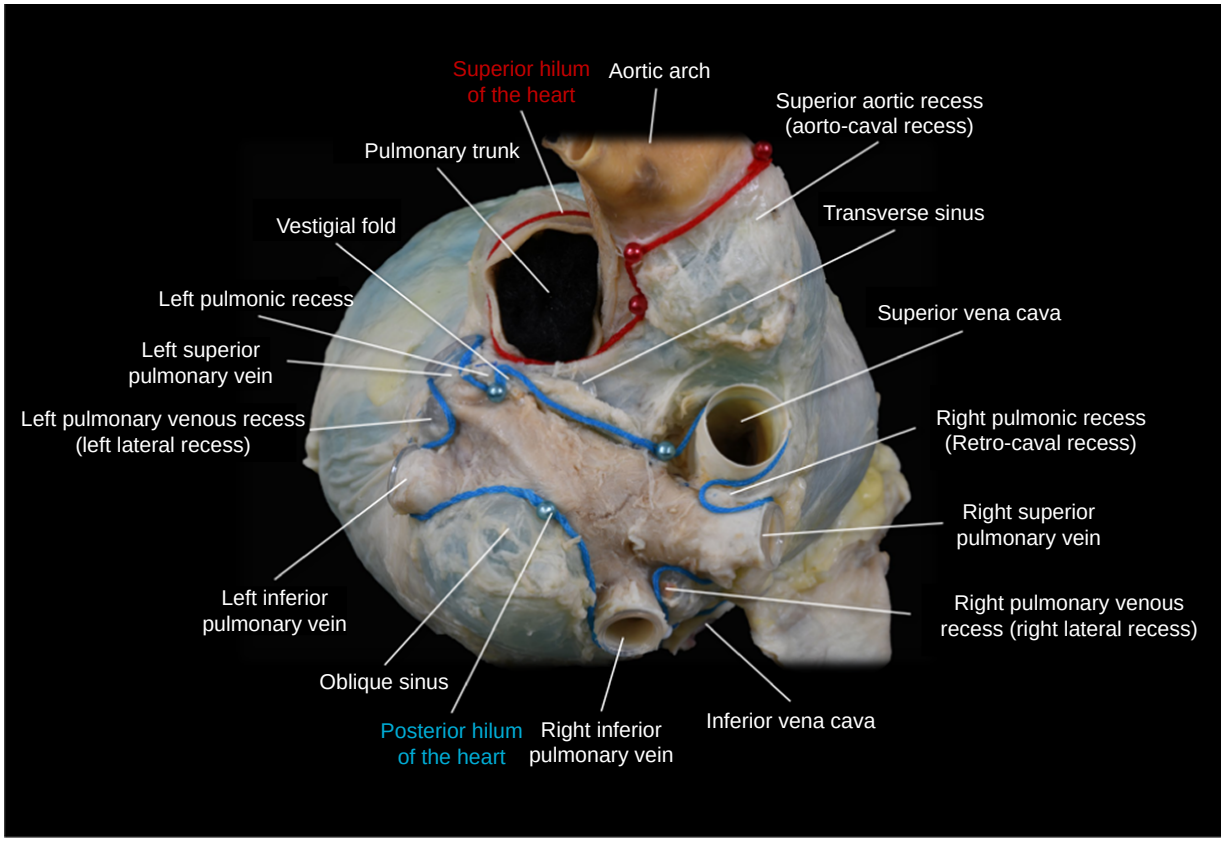

Figure 2 Pericardial reflections and hilar orifices around the great cardiac vessels (posterior view). Image adapted with permission from the Image Courtesy of the UCLA Cardiac Arrhythmia Center, Amara-Yad Project Collection.

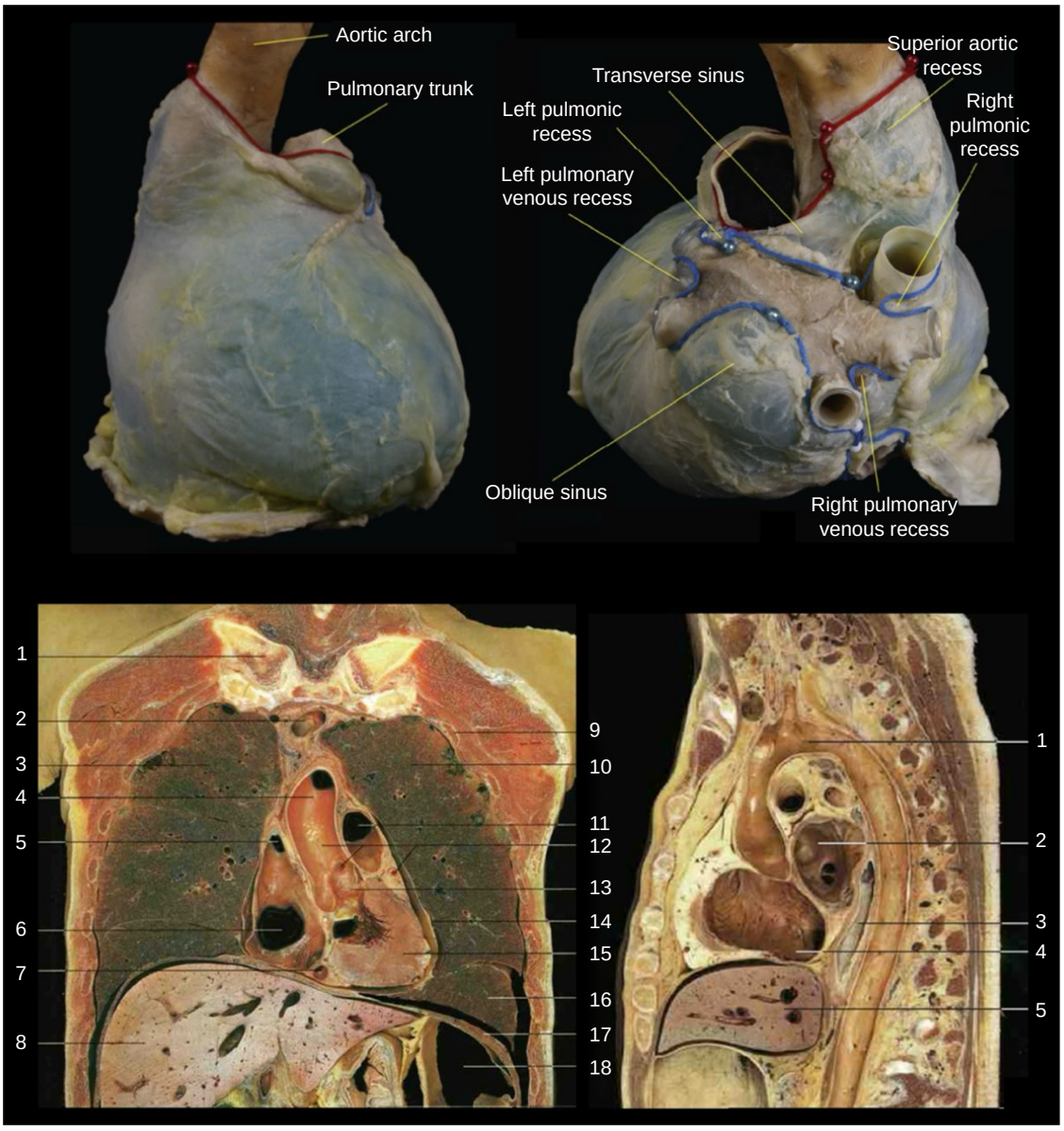

Figure 3 Upper panel: Anterior and posterosuperior view of the pericardium showing the superior or arterial hilum (red string) and the posterior or venous hilum (blue string), with the distribution of the main pericardial sinuses and recesses. Lower panel: Coronal and paramedian section of the thorax depicting the anatomical relations of the pericardium within the thorax. Left lower panel: 1. clavicle; 2. left brachiocephalic vein; 3. upper lobe of the right lung; 4. aortic arch; 5. superior vena cava; 6. right atrium; 7. coronary sinus; 8. liver; 9. second rib; 10. upper lobe of left lung; 11. pulmonary trunk; 12. ascending aorta and left coronary artery; 13. aortic valve; 14. pericardium; 15. left ventricle; 16. the lower lobe of the left lung; 17. diaphragm; 18. colon. Right lower panel: 1. aortic arch; 2. left atrium; 3. oesophagus; 4. right atrium; 5. liver. Image adapted with permission from the UCLA Cardiac Arrhythmia Centre, Amara-Yad Project Collection, and Wolters Kluwer.

- **Posterior relations**: Posteriorly, the pericardium is related to the organs of the posterior mediastinum, especially the oesophagus, which is adjacent and posterior to the oblique sinus and left atrium (see Supplementary material online, *Figure S3.3*).
- **Lateral relations**: Laterally, the pericardium is separated from the mediastinal pleura by a thin layer of loose connective tissue that contains the phrenic nerves and pericardiophrenic vessels. The left phrenic nerve runs over the left atrial appendage and then over the left ventricular free wall. The right phrenic nerve runs along the superior vena cava, close to the right superior pulmonary vein.

Vessels and nerves

The arterial supply of the pericardium is provided by a branch of the internal thoracic artery known as the pericardiophrenic artery, along with smaller contributions from the bronchial and oesophageal arteries, which are branches of the thoracic aorta. Venous drainage is through the pericardiophrenic veins, tributaries of the brachiocephalic veins, and varying tributaries of the azygos venous system. The fibrous pericardium and the parietal layer of the serous pericardium receive innervation from the phrenic nerves and vagal inputs from the oesophageal

plexus. The visceral layer lacks pain sensitivity. Pain associated with pericarditis only originates in the parietal layer and is transmitted by the phrenic nerve.

Section 3: Criteria to consider epicardial access and mapping

Epicardial VAs are defined as (i) focal VAs originating from the epicardial side or (ii) scar-related re-entrant VAs with an epicardial exit site. In these situations, epicardial ablation is necessary to terminate the VA and disrupt its circuit.

The candidates for epicardial VT ablation include patients with an arrhythmogenic substrate inaccessible from the endocardium and certain non-ischaemic cardiomyopathies (NICMs) that are known to predispose to the epicardial substrate. Patients with Chagas' disease, arrhythmogenic right ventricular cardiomyopathy/dysplasia (ARVC/D), or Brugada syndrome may also be candidates, given the propensity for epicardial involvement. Epicardial ablation is generally not useful when imaging, mapping, or electrocardiogram (ECG) suggests a septal origin.[16,17] Delayed transseptal activation time (>40 ms), disrupted transmural activation patterns, and long-duration (>95 ms) septal electrograms, fractionated/late/split electrograms observed during pacing adjacent to a mid-myocardial septal abnormality can be helpful in the identification of intramural septal substrates in NICM.[18]

Electrocardiographic identification of epicardial origin of ventricular arrhythmias

The ECG can help identify specific patterns suggestive of epicardial VT. However, while ECG criteria provide valuable insights, they are not infallible and should not be used as the sole determinant for selecting the epicardial ablation approach. They are notably less reliable in patients with extensive scars from prior myocardial infarction.[19]

Idiopathic ventricular arrhythmias

Idiopathic VA usually originates from the endocardial side. If endocardial mapping and ablation fail to identify an endocardial origin, epicardial catheter ablation may be necessary.[20]

Identifying epicardial left ventricular summit ventricular arrhythmia

A maximum deflection index of ≥0.55 could help to identify an epicardial left ventricular origin primarily located in the epicardial perivascular sites with a distance > 10 mm from the aortic sinus of Valsalva.[21] The left ventricular summit is a complex epicardial region.[22] LV summit VAs usually have an RBBB-like pattern. Some VAs from left ventricular summit have an LBBB-like pattern with an early transition in V2 or V3.[20] In these patients, a later than V2 transition or a precordial break pattern and a lack of an S wave in V5 and V6 suggest an epicardial site of origin.[23]

Rare epicardial VT origins: the crux of the heart

Rarely, epicardial idiopathic VA originates from the crux.[24] Epicardial VA from the crux shows superior axis, and a negative deltoid wave in the inferior leads, a prominent R wave in V2, an intrinsicoid deflection time in lead V2 ≥ 85 ms, and a precordial maximum deflection index ≥ 0.55. A shortest precordial RS duration ≥ 121 ms does not help differentiate this type of epicardial VA.[24]

Ventricular arrhythmias in non-ischaemic cardiomyopathies

Many forms of NICM are associated with dominant epicardial scarring. Consequently, many NICM-related VA circuits and exit sites are located at the right ventricle (RV) and LV epicardial regions.[25] Patients with left ventricular NICM can be classified according to their scar pattern and resulting arrhythmia as either anteroseptal (mainly intraseptal) or inferolateral.[25]

Epicardial VTs in ARVC/D generally originate from the basal inferior free wall and the infundibular anterior section of the RV, close to the interventricular groove. Although many of these VTs are epicardial, endocardial ablation can also be effective, especially in advanced stages of the disease characterized by significant dense epicardial scarring.[26] Certain premature ventricular contraction and VT-QRS morphology and interval ECG criteria suggest epicardial VT origin in patients with NICM.

Morphology criteria

ECG morphological criteria to pinpoint an epicardial VT origin rely on initial QRS polarity ('r' or 'q' wave) analysis in the ECG lead facing the VT's source. If ventricular depolarization starts at the epicardium, the vector moves away from the ECG lead, showing a Q wave or QS pattern (*Figure 4*). The presence of an initial Q wave in lead I suggests an epicardial VT origin at basal and apical superior LV sites; a Q wave in leads II, III, or aVF indicates the same at basal and apical inferior LV sites; and the absence of a Q wave in inferior leads points to an epicardial origin at basal superior sites.[27] A Q wave in all inferior leads indicates epicardial VTs originating from any lower section of the LV, while an aVR/aVL QRS-complex amplitude ratio of <1 points to epicardial VTs from the basal inferior LV. Additionally, a Q wave in lead V2 slightly suggests epicardial VTs at the LV apex.[28] The morphology criteria are less helpful in determining the epicardial origin of right ventricular VTs. Q or QS complexes in leads II, III, and aVF indicate an epicardial exit site for inferior and basal right ventricular VTs. A QS complex in leads I and V2 also suggests an epicardial origin in ARVC/D, stemming from the lower right ventricular outflow tract and free wall.[29]

Interval criteria

In NICM, VTs that originate from the epicardial side of the basal–lateral LV wall, ventricular activation begins at the epicardial (or subepicardial) layers and slowly progresses towards the endocardium, ultimately reaching and activating the subendocardial Purkinje fibres, which depolarize both ventricles. This initial slow depolarization resembles a delta wave.

For LV VTs, ECG criteria that suggest an epicardial origin include pseudodelta ≥ 34 ms, intrinsicoid deflection time ≥ 85 ms, shortest RS ≥ 121 ms, and maximum deflection index ≥ 0.55.[21,30] The overall duration of the QRS complex is not useful due to considerable overlap in QRS duration for both epicardial and endocardial types of VTs. However, a duration > 211 ms is suggestive of epicardial VT.[30]

However, applying interval criteria in NICM conditions must be approached with caution due to several factors. These include the pattern of transmural scarring, the affected LV region, the extent of LV remodelling or dilatation, the local wall thickness at the VT origin, and any existing baseline conduction system issues, all of which can influence the relevance of the pseudodelta wave and other interval criteria in NICM contexts.[28,31] In addition, interval criteria are invalid in right ventricular epicardial VAs.[31]

In conclusion, the effectiveness of ECG criteria for diagnosing LV epicardial VAs is reliable in the basal superior and lateral LV (diagnostic accuracy of 87% using computerized criteria). In these LV zones, morphological ECG criteria, particularly the presence of a Q wave in the lead I and a four-step ECG algorithm (*Figure 5*), reach a diagnostic precision comparable to that of more advanced computerized VT-QRS criteria.[32,33]

Indicators of an epicardial substrate during sinus rhythm

Analyzing the 12-lead sinus rhythm ECG features of patients *without* distal conduction system disease or ventricular pacing can help identify

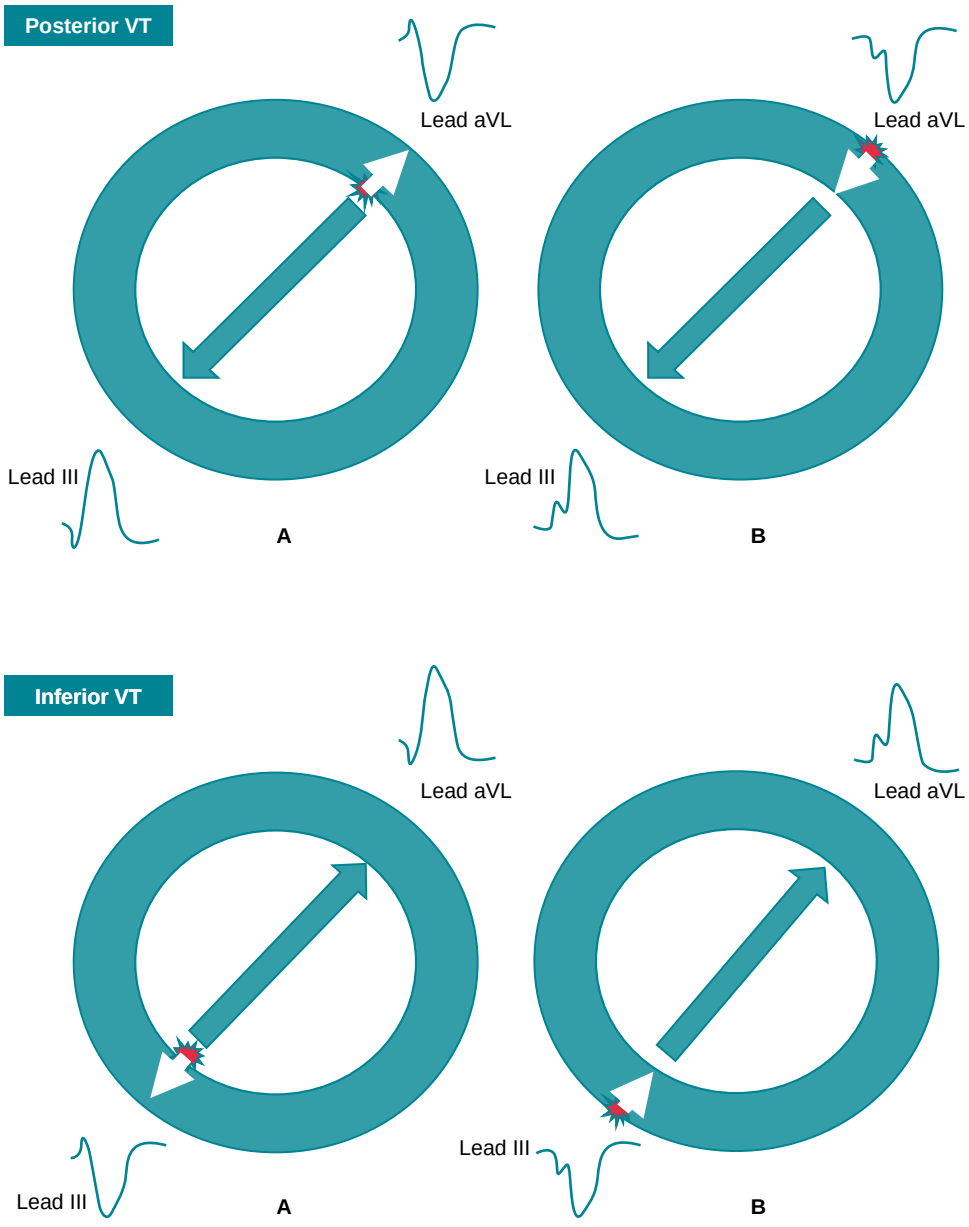

Figure 4 Basics for ventricular arrhythmia-ECG morphological criteria in patients with non-ischaemic cardiomyopathy. Depolarization and ECG morphology in posterior and inferior monomorphic ventricular arrhythmias in patients with non-ischaemic cardiomyopathies with endocardial (*A*) and epicardial (*B*) exit sites. Adapted with permission from Vallès *et al.*[27]

the presence of a left ventricular epicardial VT substrate in patients with NICM. Attention to these features and recognizing when epicardial mapping and ablation are necessary can significantly improve the effectiveness of pre-procedural planning.[34,35] *Table 4* summarizes these criteria.

In patients with NICM, the identification of those with a predominant inferolateral substrate was highly accurate, using three-step criteria including a PR interval duration of <170 ms (sensitivity 73%, specificity 100%), a QRS voltage of <0.6 mV in the inferior leads (sensitivity 50%, specificity 90%), and the presence of a q wave in the lateral leads (sensitivity 35%, specificity 100%). This stepwise approach achieved a sensitivity of 92% and a specificity of 90%.[36] On the other hand, the accurate prediction of a predominant anteroseptal substrate was achieved with these four criteria: presence of a paced ventricular rhythm (sensitivity 22%, specificity 95%), a QRS duration over 170 ms (sensitivity 50%, specificity 95%), a PR interval exceeding 230 ms (sensitivity 42%, specificity 95%), and an r wave in V3 ≤ 0.3 mV in the absence of complete bundle branch block (sensitivity 25%, specificity 100%). These parameters collectively resulted in a sensitivity of 92% and a specificity of 81%.[36]

Ventricular arrhythmias in ischaemic cardiomyopathy

Acknowledging the diverse and complex potential configurations of arrhythmogenic circuits in their relationship with the endocardial

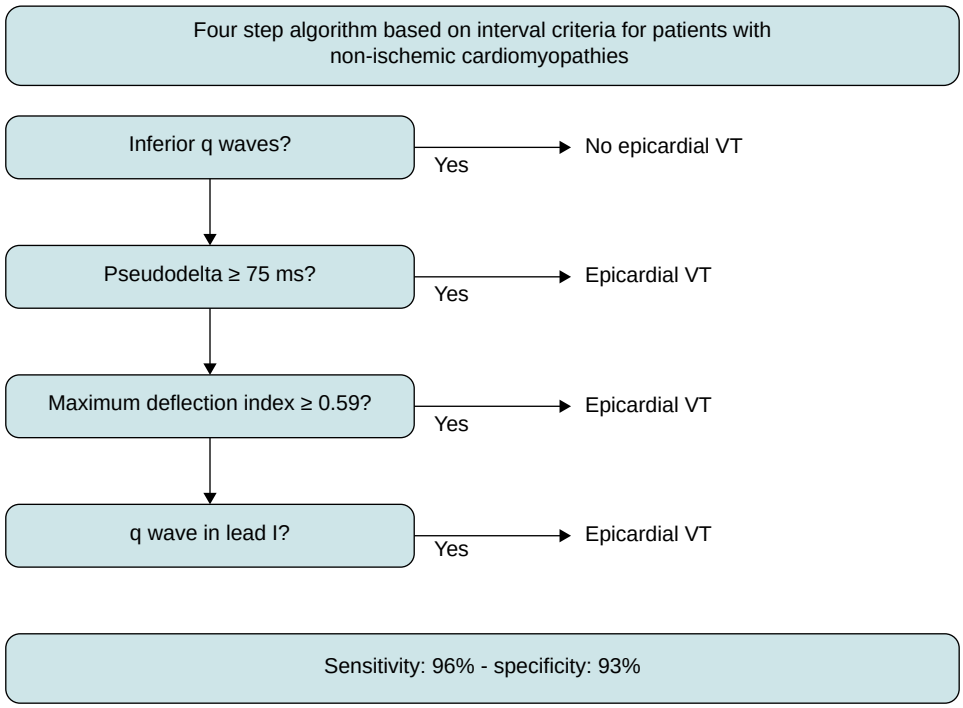

Figure 5 Four-step algorithm based on inferior Q-waves in inferior and lead I, pseudodelta, and maximum deflection index to identify left ventricular epicardial ventricular tachycardia (VT) in patients with non-ischaemic cardiomyopathy based on VT-QRS morphology. This algorithm applies to 'basal-superior/posterior' VTs in non-ischaemic cardiomyopathy. Adapted with permission from Vallès *et al.*[27]

Table 4 Indicators of an epicardial substrate during sinus rhythm

ECG Criteria	Sensitivity (%)	Specificity (%)
(1) QRS fragmentation in the lateral leads[a][34]	20	100
(2) Lack of inferior Q-waves[a] (if (1) negative)[34]	75	87
(3) S/R ratio in V6 ≥ 0.25[a] (if (1) and (2) negative)[34]	100	88
(4) R wave in V1 and S wave in V6 ≥ 0.15 mV[34]	86	88
(5) R wave in V1 ≥ 0.15 mV and S/R ratio in V6 ≥ 0.2[35]	57	88
(6) R wave in V1 ≥ 0.15 mV[35]	100	63
(7) S wave in V6 ≥ 0.15 mV[35]	86	50
(8) S/R ratio in V6 ≥ 0.2[35]	57	50

[a]Cumulative sensitivity 100% and specificity 77%.

Table 5 Unipolar voltage criteria based on conventional mapping catheter to identify epicardial substrate during endocardial mapping

Chamber	Unipolar threshold (mV)
Right ventricle	<5.5[a]
Left ventricle (non-ischaemic cardiomyopathy)	<8.3
Left ventricle (ischaemic cardiomyopathy)	<4.0–5.0

[a]In patients with arrhythmogenic right ventricular cardiomyopathy, a small study has shown that using a reduced unipolar voltage threshold of 3.3 mV along with micro-electrode mapping can most accurately identify epicardial scarring.[38]

and epicardial layers is essential. These circuits are not strictly confined to endocardial or epicardial locations; they exist along a continuum, including transmural and mid-myocardial distributions. Recognizing this spectrum enhances our understanding of the substrate and guides more effective therapeutic strategies. Therefore, the criteria that differentiate epicardial from the endocardial site of origin in patients with NICMs fail to identify epicardial origin in patients with ischaemic cardiomyopathy (ICM). In patients with monomorphic VA, ECG morphology with negative concordance and superior axis in patients with inferior scar predicted the need for epicardial VT ablation.[37]

Electrophysiological findings indicating epicardial ventricular arrhythmias

In addition to activation and entrainment mapping, endocardial unipolar voltage mapping is a sensitive method for detecting intramural or epicardial abnormalities, even effective in the presence of layered scarring (*Table 5*). This technique surpasses cardiac magnetic resonance imaging (CMR) and computed tomography (CT) in identifying the epicardial VT

substrate, offering real-time insights to pinpoint potential targets for epicardial ablation.[6,39,40]

Several factors affect the accuracy of unipolar voltage mapping:

- The accuracy of unipolar electrogram recordings depends on the proper electrode contact to the adjacent endocardium.
- Optimal voltage cut-offs for scar identification are catheter-specific. Smaller electrodes may require lower thresholds than the higher thresholds reported for larger electrode sizes.[38,41]
- Adjusting high-pass filter settings (ranging from ≤0.5–2 Hz to 30–100 Hz) impacts the capacity of unipolar recordings to detect low-frequency signals, typically indicative of distant pathologies, thereby affecting the effectiveness of endocardial unipolar mapping in identifying remote myocardial diseases.[42]
- Wall thickness correlates linearly with the unipolar voltage threshold for predicting epicardial scar tissue.[43]
- Unipolar mapping cannot distinguish between intramyocardial and epicardial scarring; therefore, its spatial resolution is inferior to that of late gadolinium enhancement (LGE)-CMR.

If epicardial ablation is not initially considered, detecting intramural/epicardial scarring in areas with normal endocardial bipolar readings (using <8.3 mV for LV and <5.5 mV for RV) should prompt consideration of accessing the epicardium for ablation to improve outcomes. Advanced stages of NICM often involve a transition to a less arrhythmogenic, fibrotic epicardial scar with endocardial arrhythmogenic zones.[31,38,42]

The conventional cut-off in patients with ICM overestimates the extent of epicardial scar. Lowering the voltage cut-off in LV to <4.0–5.0 mV provides an acceptable specificity and sensitivity in these patients for predicting epicardial scar.[44] A cut-off of unipolar voltage of <6.9 mV or <6.2 mV has been found to differentiate normal myocardium from transmural scar as detected by CMR or histopathological studies, respectively.[45,46]

Some writing group members use intracardiac echocardiography (ICE), in addition to mapping, cardiac CT, and CMR, to enhance visualization of ventricular scar tissue. This could potentially assist in determining whether an epicardial approach is necessary.

Section 4: Periprocedural management and access techniques

Advice	Strength of evidence
Advice TO DO	
Invasive arterial blood pressure monitoring is advised during epicardial VA ablation[7,47–49]	 >90% agree
Advice NOT TO DO	
Uninterrupted anticoagulation is not advised in patients undergoing epicardial VA ablation[50–53]	 >90% agree

Periprocedural anticoagulation

Pre-procedural

In patients taking vitamin K antagonists, oral anticoagulants are typically stopped before epicardial ablation to achieve an international normalized ratio of <1.5 at the time of the procedure. Direct oral anticoagulants should be withheld for at least 24 h before the procedure.

The need for heparin bridging is determined individually based on the presence and type of mechanical heart valves and other factors that increase thrombotic risk. Among the writing members, 50% preferred minimally interrupted anticoagulation (the last dose taken on the morning of the day before the procedure for once-daily regimens or missing one or two doses for twice-daily regimens), while 40% favoured interrupted anticoagulation (e.g. the last dose taken more than 24 h before the procedure) when performing epicardial VA ablation.

Intraprocedural

Systemic anticoagulation is not required in epicardial-only mapping and ablation during the procedure. However, in most VA ablation procedures, epicardial access is often paired with endocardial mapping and ablation. Systemic anticoagulation, usually with unfractionated heparin, is needed during endocardial mapping and ablation of left VAs. Detailed advice for intraprocedural systemic anticoagulation of LV ablation is described in the 2019 HRS/EHRA/APHRS/LAHRS expert consensus statement on catheter ablation of VAs.[7] In combined epicardial–endocardial VA ablation, the timing and approach of systemic anticoagulation are critical considerations. It is reasonable to confirm epicardial access before LV access and systemic anticoagulation in procedures where both epicardial and endocardial access are planned. This approach has the potential role of mitigating bleeding risk if complications related to obtaining epicardial access were to occur, most notably inadvertent RV puncture.

Eighty per cent of the writing group members routinely use imaging to exclude LV thrombus in all patients undergoing epicardial VA ablation, irrespective of presenting rhythm, history of atrial fibrillation (AF), and prior anticoagulation.[9,54,55] Therefore, we advise imaging for the exclusion of LV thrombus in all patients undergoing epicardial VA ablation.[9,55] Our survey showed that echocardiography and cardiac CT (each with around 37%) are the most common imaging modalities for excluding LV thrombus. However, transoesophageal echocardiography and CMR have also been used (12.5% for each). However, epicardial mapping and ablation can be done in the presence of an LV thrombus.

Unplanned epicardial access and post-procedure

If epicardial access is unplanned and becomes necessary after endocardial LV mapping and ablation, it must be performed following protamine reversal. Limited evidence exists regarding the safety of epicardial access attempts during full anticoagulation, with a few studies indicating safety.[50] Therefore, the writing group believes reversing heparin with protamine targeting an ACT < 200 s before epicardial access in patients fully anticoagulated with systemic heparin is reasonable.[50] No consensus could be reached on the appropriate time for resuming anticoagulation after epicardial sheath removal; however, resuming anticoagulation before 3–5 h is not advised.

Anaesthetic considerations

Obtaining epicardial access and ablation is a high-risk procedure. Therefore, the members (90%) routinely use invasive arterial blood pressure monitoring during epicardial VA ablation. Additionally, maintaining patient immobility with general anaesthesia or deep sedation assists the operator in successfully achieving access and substantially reduces access-related complications.

Deep sedation agents such as propofol and inhaled anaesthetics can suppress VAs and cause significant haemodynamic compromise, both of which are undesirable during VA ablation. Deep sedation using remifentanil with midazolam was reported to be feasible and safe; however, only in small studies.[56]

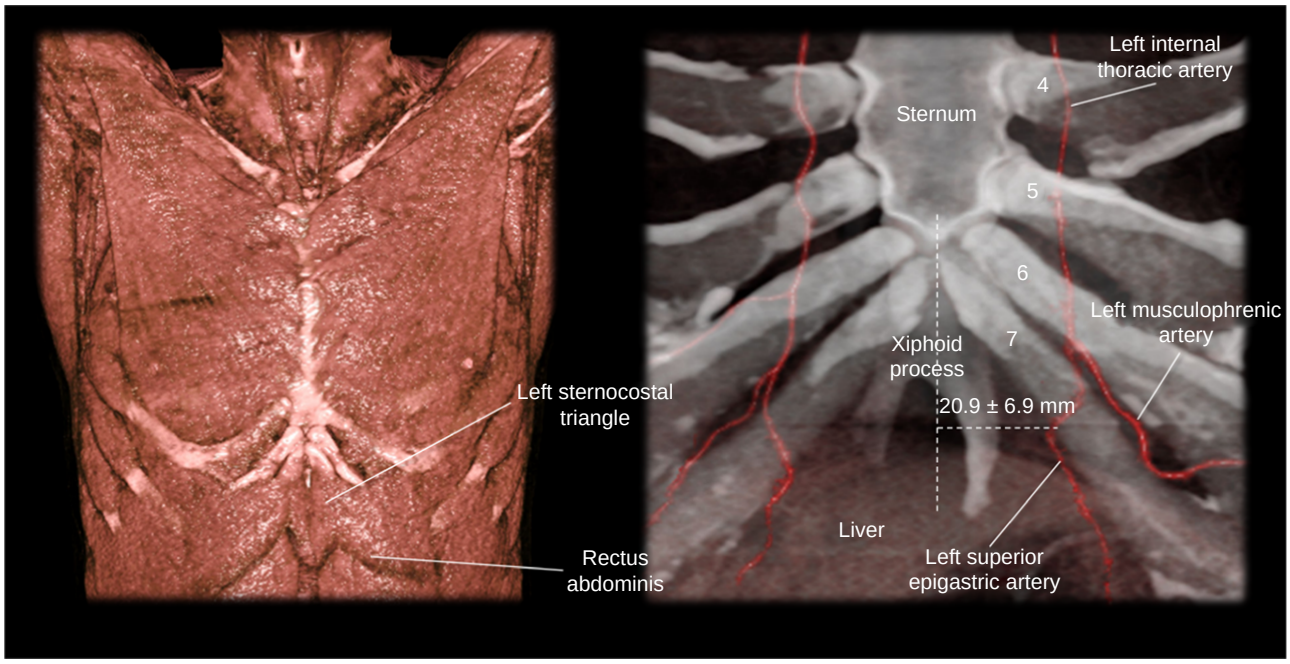

Figure 6 Major arteries located near the epicardial puncture site. Please note the vicinity of the left superior epigastric, left musculophrenic, and left internal thoracic (mammary) arteries to the puncture site. Image adapted with permission from the Image Courtesy of the UCLA Cardiac Arrhythmia Center, Amara-Yad Project Collection.

The decision to use general anaesthesia or conscious sedation should be carefully tailored to each patient, considering factors such as left ventricular function, haemodynamic stability during VA, the potential need for mechanical circulatory support, and the availability of specialized cardiac anaesthesia staff. Sixty per cent of the writing group supported the routine use of general anaesthesia for epicardial VA ablation. The remaining members preferred deep sedation over general anaesthesia, with 66% citing the limited availability of anaesthesiologists and 33% attributing their preference to concerns about prolonged procedural duration and an increased risk of noninducibility.

Percutaneous access techniques

Entry point

The dry pericardial puncture technique remains essentially unchanged since its original description.[11] To find the puncture site, the xiphoid process should first be palpated. The needle should be advanced under live fluoroscopy during an inspiratory breath hold or apnoea (if intubated). The entry site should be lateral to the xiphoid below the margin of the left-sided rib cage. The needle can be pointed towards the patient's left shoulder, and the stylet can be removed once under the skin. Some operators point the needle towards the chin to avoid the internal mammary artery; however, this may increase the risk of hepatic puncture. The optimal puncture site is represented by the triangle formed by the left border of the xiphoid process and the lower left rib (*Figure 6*).

Needle

Most centres utilize a Tuohy needle to facilitate access to the virtual space between the parietal and visceral pericardium while minimizing the risk of lacerating adjacent structures. Its spoon-shaped tip helps separate tissue planes along its trajectory with minimal trauma.

A 17- and/or 18-gauge, 120–180 mm long Tuohy needle is advanced, with its spoon-shaped tip towards the myocardium, under the skin and the rib cage towards the left shoulder, oriented according to the targeted ventricular surface (i.e. anterior, shallow < 45 vs. inferior, steep > 45°) (*Figure 7*—right). The needle-in-needle technique (micropuncture) has been described;[57,58] it uses a smaller 21-gauge, 15- and/or 20 cm long micropuncture needle for accessing the pericardium. The micropuncture needle is inserted through a standard 7 cm, 18-gauge access needle to provide stability. Once pericardial access is obtained using the micropuncture needle, a 0.018 inch long guidewire is advanced into the pericardium, which is subsequently exchanged for a standard 0.032 or 0.035 inch long guidewire to allow insertion of larger bore epicardial introducers.[57,59,60]

A multicentre observational study of 404 patients compared large-bore and micropuncture needles for epicardial access during VA ablation or Lariat procedures. While both showed similar rates of inadvertent myocardial puncture, the micropuncture needle group had significantly fewer complications, including large pericardial effusions and cardiac lacerations requiring surgery. The study suggested a shift towards routine use of micropuncture needles for epicardial access procedures.[57] The micropuncture technique is utilized by 40% of the writing group members.

Angle

During epicardial puncture, the angle between the needle and the skin will determine the ventricular surface being accessed (anterior vs. inferior). The needle angle can be adjusted based on the targeted region of the myocardium that the operator wishes to access. Most commonly, this is the medial third of the RV, an area typically free of major coronary vessels.

For an inferior puncture, the skin is punctured 2–3 cm below the rib cage margin at roughly 30–45° angles, which allows the needle to pass

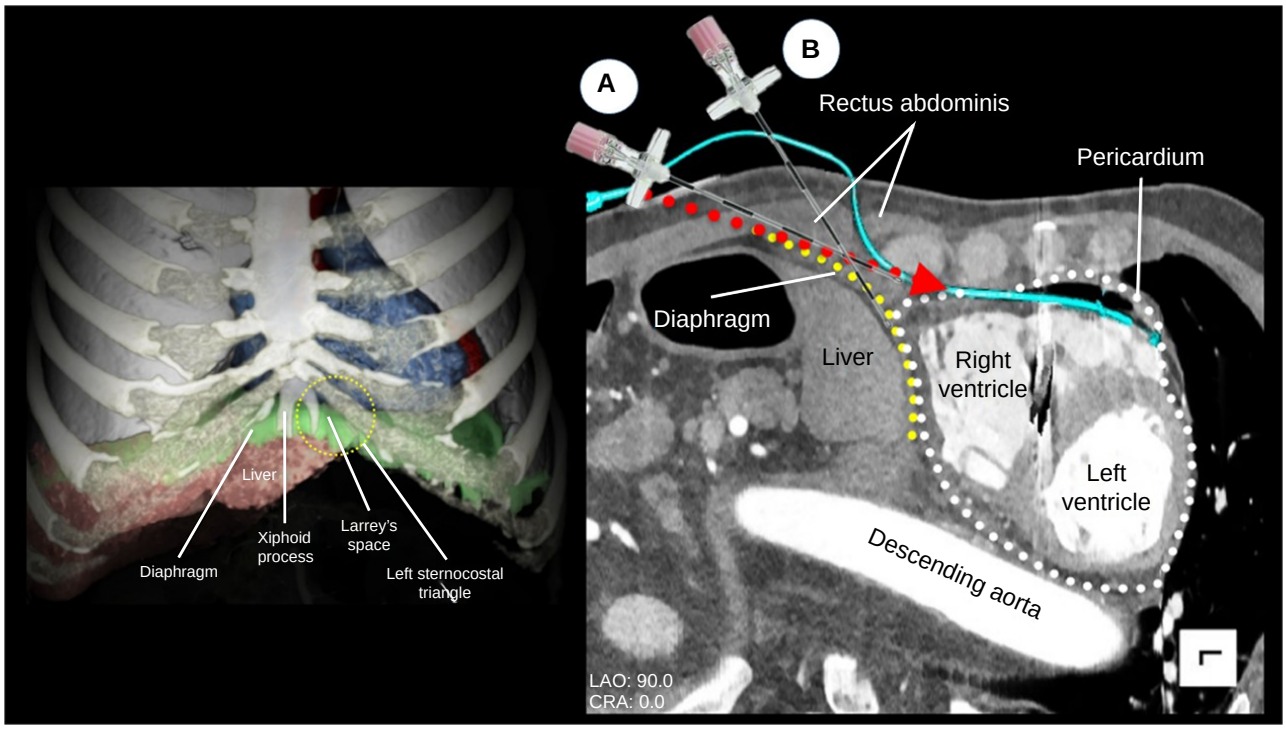

Figure 7 Left: Anatomic location of Larrey's space. Right: anterior (*A*, shallow < 45) vs. posterior (*B*, steep > 45) approach for subxiphoid puncture using Tuohy Needle. Be aware that using the posterior approach results in diaphragmatic puncture, potentially leading to intra-abdominal injuries. Image adapted with permission from the Image Courtesy of the UCLA Cardiac Arrhythmia Center, Amara-Yad Project Collection.

through the Larrey's space just superior to the diaphragm (*Figure 7*—left). An anterior puncture requires a shallow angle of 20–30° and requires skin entry 1–2 cm more caudally and a more medial trajectory to ensure the needle trajectory is not impeded by the underside of the sternum or passes too close to the internal mammary artery (*Figure 6*) (see also *Table 6*).

From skin to the heart

After the needle is inserted under the skin, the stylet should be removed, and the needle should be gradually advanced under fluoroscopic guidance towards the cardiac silhouette. Gentle aspiration should be performed continuously to monitor for inadvertent vascular punctures. Fluoroscopic guidance is typically used in an anteroposterior or right anterior oblique (RAO) projection for inferior punctures and in a left lateral projection for anterior punctures (*Figure 8*).

The Larrey's space is the target (*Figure 7*, left). This is a space bordered by the sternum anteriorly, the pericardium posteriorly, and the dome of the diaphragm inferiorly. The needle should always be advanced over the diaphragm because the latter is a highly vascularized structure. Fluoroscopic visualization of the needle in the left lateral projection can significantly aid with this. As stated, if the patient is intubated, the control of respiration through an inspirational breath-hold can further facilitate the puncture and is associated with a lower complication rate.[58]

Once the needle reaches the pericardium, cardiac pulsations can be felt. Initially, the pulsations may be intermittent and more prominent with inspiration. However, as the needle tip gently advances towards the pericardium, cardiac pulsations eventually become apparent with every heartbeat. The injection of a small amount of contrast medium will indicate the location of the needle tip (*Figure 8*). If contrast injection results in staining of the pericardial fat or the pericardium, the needle may be slowly advanced another 1–2 mm, and contrast injection should be repeated until the pericardium is accessed. Care should be taken to avoid injecting large amounts of undiluted contrast, which can considerably obscure the fluoroscopic image.

Epicardial pop

The puncture of the fibrous pericardium may be accompanied by a noticeable 'pop' and appreciated with the release of resistance on the needle. At this stage, injecting contrast medium will result in its diffuse dispersion throughout the pericardium and along the cardiac silhouette (*Figure 9A*). Visualization of contrast along the inferior pericardial border can be helpful because it validates the position of the needle inside this space (*Figure 9A*).

Some members of the writing group employ an alternative method in which the wire is introduced into the needle when approaching the heart. As the needle advances slowly, the wire can be used for probing. Once the pericardial cavity is accessed, the wire advances freely.

The needle tip can occasionally penetrate the RV, which can be easily recognized upon injection of contrast with rapid washout and/or by the presence of blood during syringe aspiration. In the event of an RV puncture, the needle can simply be withdrawn slightly while reinjecting a small amount of contrast until the pericardium is entered.

After the needle tip is positioned in the pericardium, a soft, long J-tip guidewire is inserted into the pericardium through the needle. The use of long guidewires is essential for demonstrating the crossing of multiple chambers, including transitions from the left to the right chambers (*Figure 9B*). The position of the guidewire must always be validated in

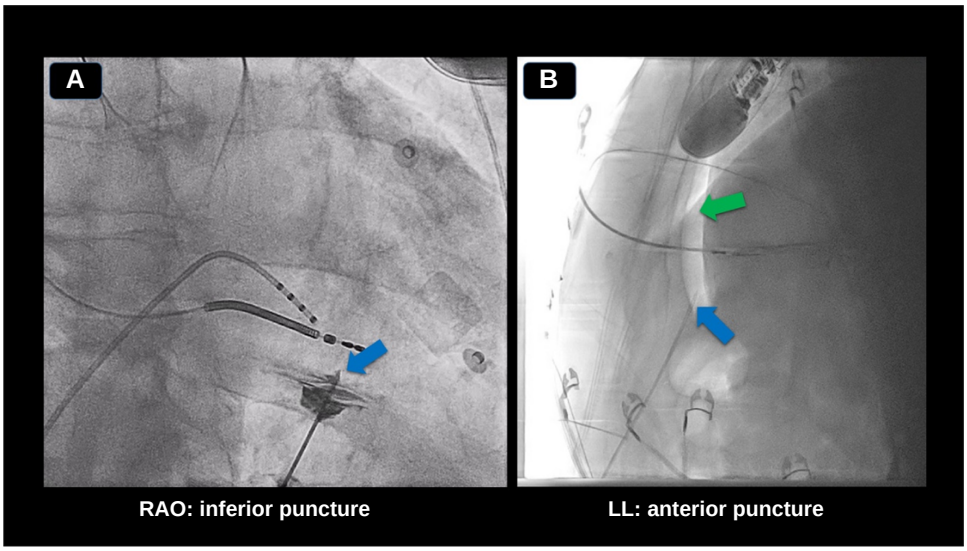

Figure 8 (*A*) Inferior subxiphoid puncture in right anterior oblique projection. (*B*) Anterior subxiphoid puncture in left lateral projection. Blue arrows show the entrance of the needle into the epicardial space. The green arrow indicates CO_2 insufflation (upper arrow - B).

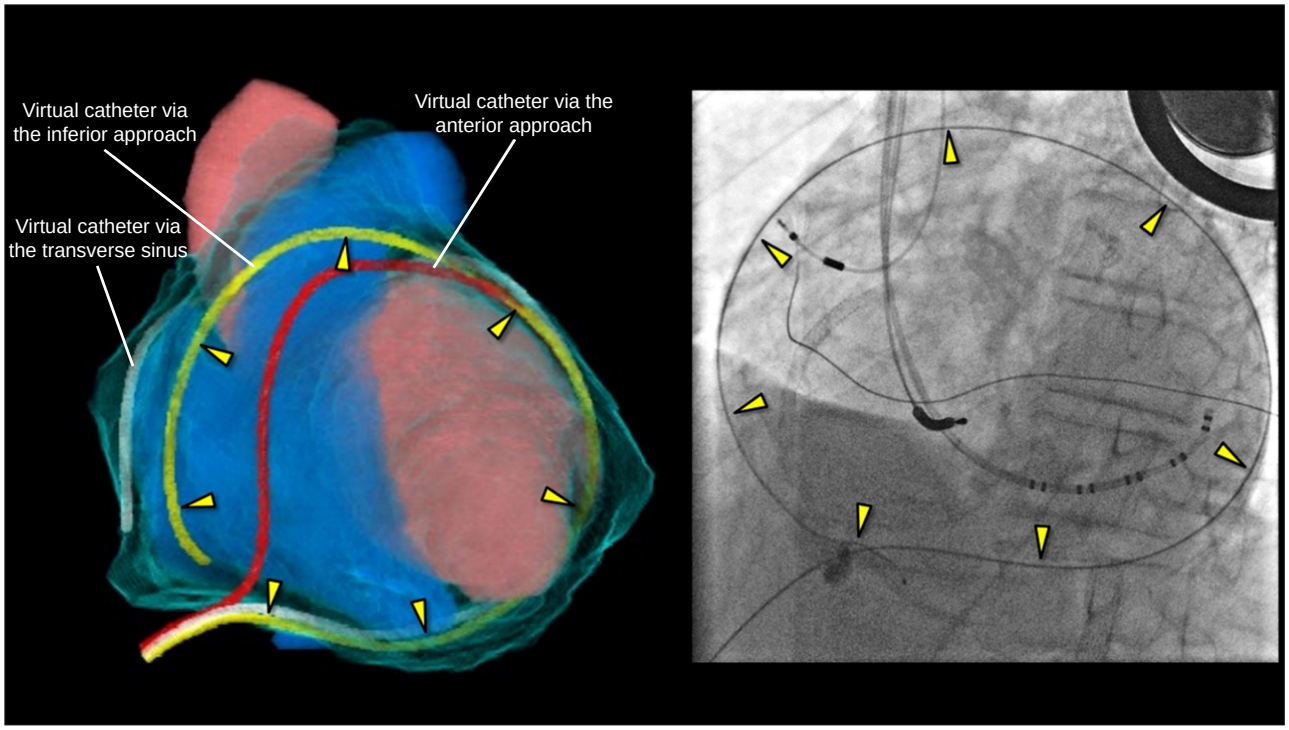

Figure 9 Virtual and fluoroscopic visualization of epicardial guidewire pathways is presented. The left panel: virtual guidewire trajectories through the anterior (red, the line without arrow) and inferior (yellow, the line with arrow) approaches on a 3D cardiac reconstruction. The arrowheads mark the location of the guidewire. The right panel: use of a long guidewire inside the pericardium (arrow heads) to demonstrate the crossing of multiple chambers encircling the heart. Image adapted with permission from the Image Courtesy of the UCLA Cardiac Arrhythmia Center, Amara-Yad Project Collection.

the RAO and left anterior oblique projections visualized following the left heart border and crossing unrestrictedly from left to right, anterior to the great vessels, to confirm the intrapericardial location (*Figure 9*).

The guidewire can be visualized on ICE passing in the pericardial space with the probe in the RV. Intracardiac echocardiography can also be used to monitor effusion after a puncture in real time.

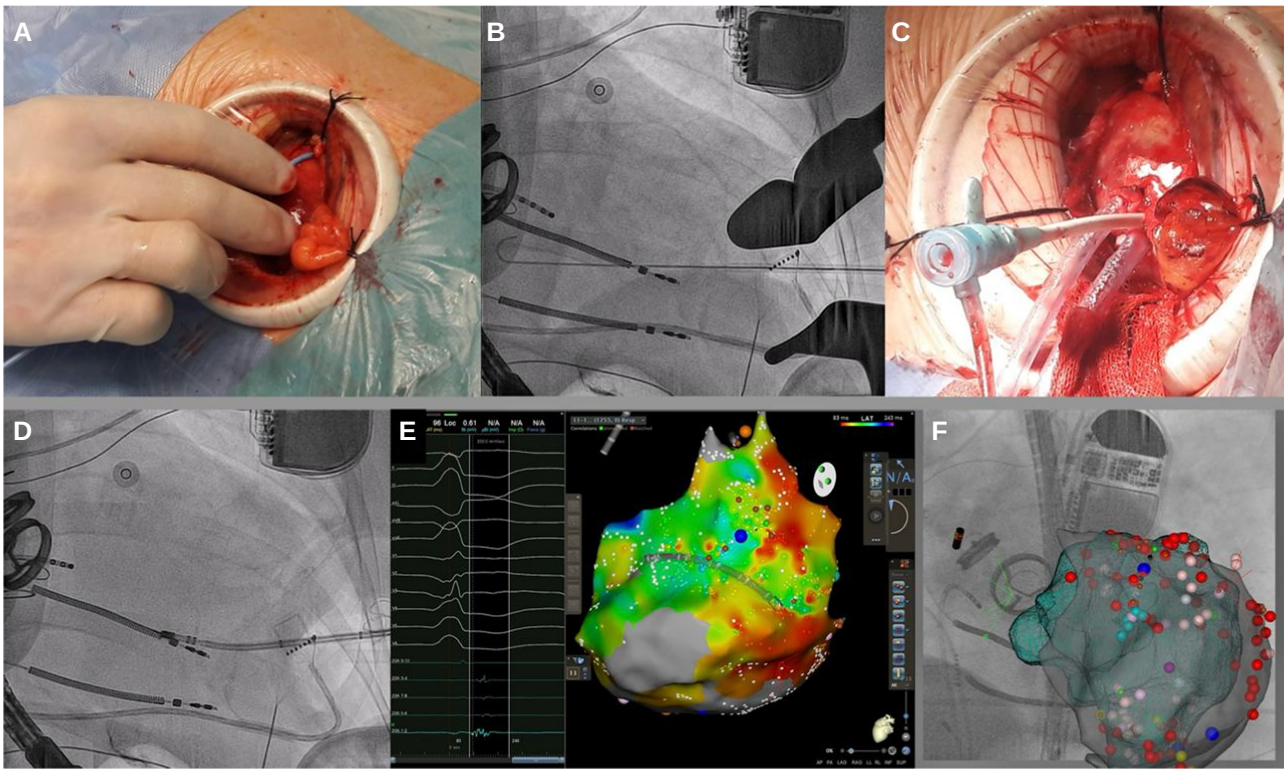

Figure 10 Surgical access for epicardial ablation. The surgical window approach for epicardial access involves creating a small subxiphoid incision to reach the pericardial space. (*A* and *B*) A limited incision is made just beneath the xiphoid process, and the subcutaneous tissues are dissected to reveal the pericardium. A small incision in the pericardium is performed under direct visualization to prevent injury to adjacent structures. (*C* and *D*) A flexible sheath is then advanced into the pericardial space, facilitating the insertion of mapping and ablation catheters. (*E* and *F*) 3D late potential mapping and 3D mapping integrated with fluoroscopy images.

Epicardial guidewire and sheaths

Once the position of the guidewire is confirmed in fluoroscopy, the epicardial introducer will advance into the pericardial space. A small skin nick at the puncture site is made, and after pre-dilatation with an 8–10 F dilator, a long sheath will be advanced. A Steerable Sheath (Abbott Vascular, Abbott Park, IL) is designed specifically for facile manipulation of intrapericardial catheters. Regardless of the type of sheath, the sheath and its introducer should never be left unprotected within the pericardial space due to the risk of myocardial and vascular lacerations. A guidewire, a blunt catheter (i.e. diagnostic mapping or ablation catheter), or a 5–7 Fr pigtail catheter must always be placed through the sheath into the pericardium until its final removal. Once an introducer has been placed inside the pericardium, the operator should monitor for pericardial bleeding by gently aspirating the sheath. Aspiration from the sheath should reveal only serous or serosanguinous fluid with a nontraumatic puncture.

Sheath removal and monitoring

At the end of the procedure, a pigtail catheter (5–7 Fr) can be positioned in the epicardial space, preferably posterior, after the removal of the catheter and steerable sheath. Any residual intrapericardial fluid should be aspirated (see Section 7). If no effusion is noted on ICE (or with conventional echocardiography) after sheath removal and vital signs are stable, the pigtail catheter may be removed and dressing applied.

Alternative epicardial access techniques

Carbon dioxide (CO_2) insufflation

The use of coronary venous exit for carbon dioxide (CO_2) insufflation in the pericardial space is becoming more common to aid subxiphoid puncture, as this method potentially enhances safety.[61,62] This technique may reduce the risks associated with traditional epicardial access practices, even in lower-volume centres.[63,64] Twenty per cent of the writing group members use CO_2 insufflation from the coronary sinus to enhance the safety of epicardial access.

Pericardial window as a surgical alternative

A pericardial window is a safe alternative to subxiphoid puncture, especially in patients with previous heart surgery. Romero *et al.*[65] recently presented a new surgical method using video-assisted thoracoscopy for safer subxiphoid epicardial access for epicardial access, especially for patients with severe pericardial adhesions and calcification (*Figure 10*).

Videoscope for percutaneous pericardial access

A novel videoscope for percutaneous pericardial access under direct visualization was also reported by Opfermann *et al.*[66] in animal studies. The device included a visualization channel where an embedded video camera allowed for direct visualization of subcutaneous tissue, surrounding vasculature, the parietal pericardium, adhesions, if any, and the ventricular wall during needle advancement through a separate working channel. No ventricular puncture was noted in their preclinical animal studies.

Other epicardial access methods

Liu *et al*. introduced an innovative puncture kit designed to improve the process of obtaining epicardial access. This kit aims to enhance precision and safety during subxiphoid puncture procedures, addressing common challenges faced with existing methods.[67]

Several studies have recently addressed the challenges of traditional approaches and demonstrated promising outcomes.[68] By pulling the parietal pericardium outward away from the RV, this device facilitates safer entry into the pericardial space, minimizing the risk of inadvertent puncture and associated complications. In a multicentre study involving 25 patients, successful epicardial access was achieved in all patients, guided by real-time pressure monitoring on the Tuohy needle. This method offers potentially safer epicardial access.[69] Further studies in larger patient populations are needed to evaluate these newer methods. Ronghui *et al*. also recently introduced needle tracking and needle electrogram analysis in 3D Electroanatomical mapping, which allows for safe epicardial access with no or minimal fluoroscopy.[70] Advancements in robotic technologies, such as the HeartLander epicardial robot, have the potential to increase precision and manoeuvrability during epicardial procedures.[71]

Key steps of the subxiphoid puncture

The subxiphoid puncture is a crucial step in accessing the pericardial space. *Table 6* summarizes the key steps for each approach, ensuring precision and safety throughout the procedure.

Section 5: Mapping techniques and selection of epicardial ablation targets

Advice	Strength of evidence
Advice TO DO	
Routine pre-procedural cardiac MRI to assess the presence, location, distribution, and extent of ventricular scars is advised in patients with non-ischaemic cardiomyopathy[55,72–80]	
May be appropriate TO DO	
Use of multielectrode mapping catheter may be useful to accelerate and enhance epicardial mapping[81]	

Table 6 Key steps of the subxiphoid puncture

Approach	Key steps	Details
Posterior	Check needle orientation	Ensure proper alignment.
	Contrast injection	Inject a small amount of contrast medium to identify the best puncture site and angulation relative to the cardiac silhouette.
	Needle orientation	After tenting, position the needle as parallel as possible to the heart profile.
	Advance the needle	Proceed slowly with a firm grip until a sudden loss of resistance is perceived. Confirm success with contrast layering and staining.
	Guidewire advancement	Insert a soft guidewire to follow the cardiac silhouette.
	Dilator positioning	Use a small dilator (4 Fr) and verify its position with contrast. Note this cannot rule out a "through and through" puncture.
	Guidewire re-insertion	Reinsert the guidewire as needed.
	Progressive dilatation	Gradually dilate the puncture tract with larger introducers to enable insertion of a firm guidewire and a larger deflectable sheath.
Anterior	Fluid accumulation	Recognize that fluid in supine-positioned, nonsurgical hearts typically accumulates anteriorly.
	Access advantage	The anterior approach provides access to the fibrous pericardium without crossing the diaphragm.
	Access site	Locate the puncture site 3–4 cm below the subxiphoid process and costal cage.
	Shallow needle angle	Advance the needle at a shallow angle (<30°) with gentle downward pressure to avoid the internal mammary artery.
	Needle trajectory	Use the left lateral view to visualize the needle's trajectory towards the anterior right ventricle.
	Needle orientation	Direct the needle towards the left clavicle or to the chin to prevent injury to the superior epigastric or left internal mammary artery.
	Contrast injection	Use contrast medium to visualize fibrous pericardium tenting, guiding the wire by tactile feedback and reduced resistance.
	Wire alignment	Ensure the wire aligns with and remains within the cardiac silhouette to avoid RV perforation.
	Progressive dilatation	Follow the same progressive dilatation steps as the posterior approach, ensuring safe sheath insertion and confirming no myocardial damage.

Identification of the ventricular arrhythmia substrate

Non-invasive mapping of the epicardial ventricular arrhythmia substrate by imaging techniques

Cardiac imaging has demonstrated its effectiveness in identifying the arrhythmogenic substrate that triggers and sustains VAs. Imaging also assists in assessing their endocardial accessibility, particularly by evaluating variations in wall thickness. It can be determined before the procedure through LGE-CMR, multidetector CT, or even during the procedure using ICE.[7,55,82]

In addition to choosing between the endocardial, epicardial, or combined approaches, CMR and multidetector CT imaging have the potential to facilitate mapping and probably improve outcomes by identifying the channels responsible for clinical or induced VT.[75,83–86] These images can be incorporated into the navigation system to guide the intervention (*Figure 11*). The use of ICE during the procedure also facilitates recognition of mid-myocardial or epicardial VA substrate.[88,89] While the usefulness of CMR is established in patients with NICM, its impact on the outcomes of catheter ablation in patients with ICM remains unclear.[55,89,90] Therefore, for patients with NICM undergoing catheter ablation of VAs, pre-procedural CMR is advisable to aid in procedural planning and potentially reduce VA recurrence.[7,55] Even in the majority of patients with implantable devices, LGE-CMR allows for identifying the scar location along the ventricular wall thickness, aiding in pinpointing the segment of origin and predicting the endocardial vs. epicardial successful ablation site for clinical VTs[74,91,92] (*Figure 11*).

Eighty per cent of the writing group members supported the routine use of pre-procedural CMR to evaluate the presence, location, distribution, and extent of ventricular scarring in patients with NICM. In contrast, 60% of the writing group opposed the routine use of pre-procedural MRI in patients with ICM. Forty per cent of the writing group members perform routine multidetector CT to measure fat thickness and/or coronary anatomy (for image integration) in epicardial VA ablation.

Catheter mapping of the epicardial ventricular arrhythmia substrate

Endocardial unipolar mapping

Endocardial unipolar mapping can be especially useful in the case of ARVC/D (see Section 3). A cut-off of 5.5 mV in the RV identifies the presence of epicardial substrate in patients with RV VTs and normal endocardial voltage.[93,94] A completely normal bipolar voltage in patients with ARVC/D can be found in about one-fourth of patients. A bipolar/unipolar low-voltage area ratio > 0.23 identifies patients with advanced-stage disease, where the most arrhythmogenic substrate is located endocardial, and the indication for an epicardial approach

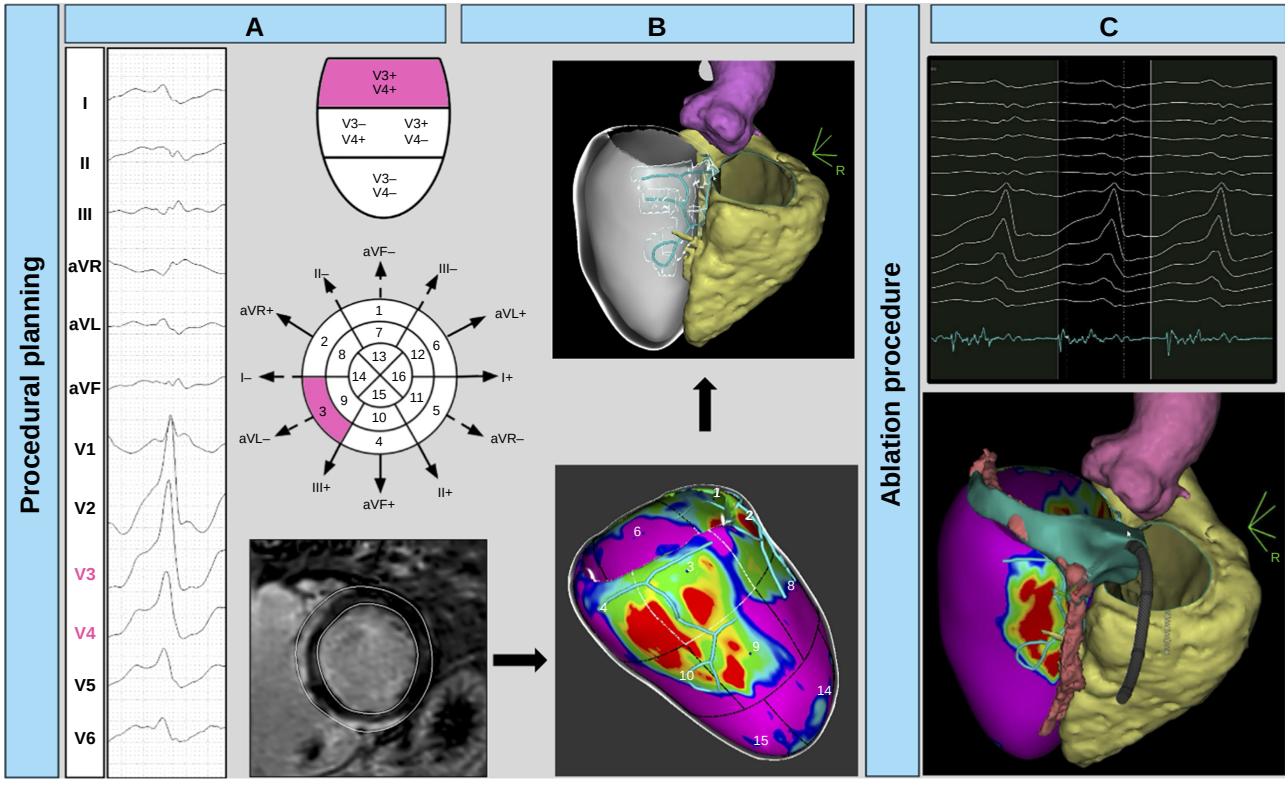

Figure 11 Planning and ablation of epicardial ventricular tachycardia (VT) in a patient with cardiac sarcoidosis. (*A*) ECG: The initial step in procedural planning involves the ECG, showing the presence of a large pseudodelta wave, and the QRS axis-based algorithm indicates an inferoseptal and basal exit of the VT.[87] The suspected segment of origin is highlighted in purple. (*B*) Cardiac imaging: Cardiac magnetic resonance imaging (CMR) confirms the presence of an inferoseptal scar. CMR Segmentation reveals a channel with an inferoseptal extension, potentially involved in the clinical VT. The CMR is integrated with a computed tomography scan to confirm the channel's anatomical location within the patient's specific anatomy. (*C*) Ablation procedure: Despite its clear epicardial origin, the VT was terminated after the first application, as the middle cardiac vein coincided with the VT isthmus trajectory. The lower panel illustrates the reconstruction of the coronary venous system and its integration with the electroanatomical map.

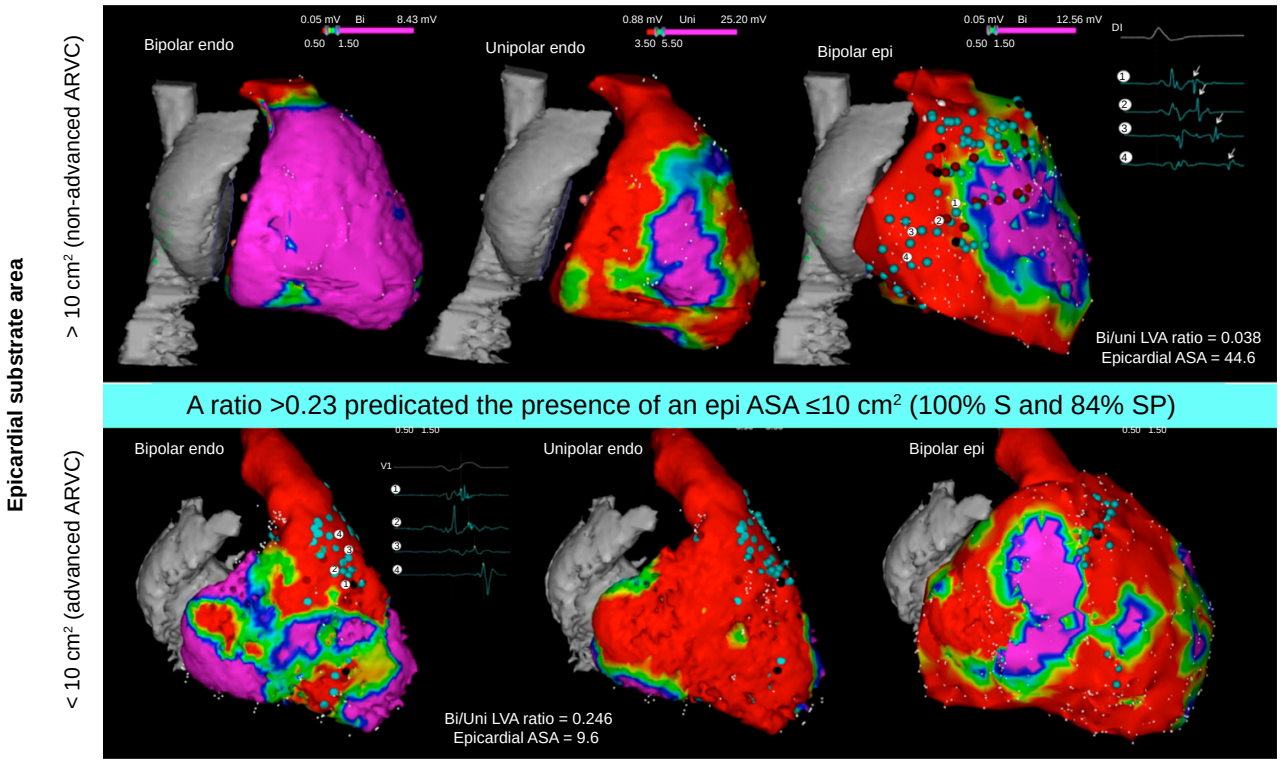

Figure 12 Epicardial puncture is not necessary in all patients with an epicardial substrate. Top panel: A patient with an early stage arrhythmogenic right ventricular cardiomyopathy (ARVC) is shown. The bipolar voltage map of the right ventricular endocardium (left) shows a small low-voltage area on the subtricuspid region. Unipolar voltage map of the right ventricular endocardium showing a significant area of low unipolar voltage (middle). Significant epicardial scar is observed in regions opposite those with low unipolar voltage. A large arrhythmogenic epicardial substrate area with late potential channels is shown (right). The ventricular arrhythmia (VA) was successfully ablated from the epicardium. Lower panel: A patient at a late stage of ARVC is shown. The bipolar voltage map of the right ventricle shows already a big area of low voltage on the endocardial surface (left). An extensive area of low unipolar voltage is observed (middle). The unipolar endocardial voltage map indicates the presence of epicardial involvement. The bipolar epicardial map shows a very large epicardial scar area. Bipolar vs. unipolar low-voltage area (LVA) ratios and epicardial arrhythmogenic substrate area (ASA) values are shown. The VAs were successfully ablated endocardially. The image was adapted with permission from Berruezo *et al.*[26]

should be assessed more cautiously (*Figure 12*).[26] The presence of intramyocardial scar may prevent rapid transmural activation, and the transmural activation delay in sinus rhythm (first endocardial to first epicardial activation, optimal cut-off 17 ms) can be used to identify intramyocardial VT substrates in patients with ARVC/D and predominantly haemodynamically non-tolerated VTs, although it requires epicardial access to be measured.[95]

Multielectrode mapping

Multielectrode and particularly omnipolar mapping accelerate and may enhance epicardial mapping.[96–101] Glashan *et al.*[96] demonstrated the efficacy of multielectrode unipolar voltage mapping and electrogram morphology in identifying post-infarct scar geometry, validated by histology, highlighting its potential to guide precise interventions. Anter *et al.*[98] introduced a novel multipolar electrogram configuration, which significantly enhances the accuracy of intracardiac signal detection, offering superior precision in identifying arrhythmic foci for targeted catheter ablation. Furthermore, Ehdaie *et al.* focused on the importance of multielectrode mapping in finding persistent late potentials after ablation of scar-related VT substrates, which could be a crucial step in preventing arrhythmia recurrence. Using 0.5–1.5 mV with conventional or multielectrode mapping is reasonable. However, further studies are needed to assess the optimal cut-offs for multielectrode mapping, as these cut-offs were originally defined by using conventional mapping catheters.[94,99]

Epicardial mapping

Epicardial mapping can be easily done in most cases once the epicardial space has been reached if there are no adhesions and obstacles. However, electrograms can be attenuated by inadequate catheter epicardial contact, particularly in the presence of pericardial effusion, coronary arteries, and fat. A bipolar voltage ≤ 1.81 mV and unipolar voltage ≤ 7.95 mV have been associated with epicardial scar in the absence of fat tissue ≥ 2.8 mm in thickness. Even if fat is present, prolonged electrogram duration and fractionated electrograms are indicative of scar (*Figure 13*).[39]

Identification of epicardial ablation targets

Mapping during sinus rhythm

Two primary scar patterns have been identified in patients with NICM; approximately half show a predominant anteroseptal scar distribution,

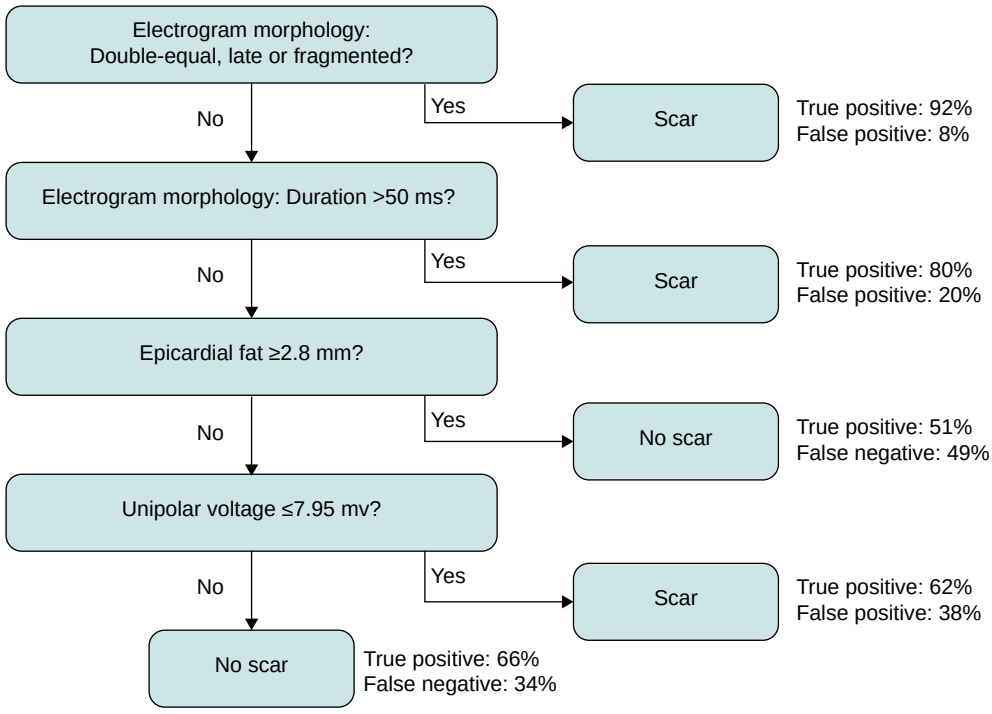

Figure 13 Algorithm for differentiation between scar and viable myocardium during epicardial mapping. Adapted with permission from Piers *et al.*[39]

while the other half exhibit a predominant inferolateral scar. Patients with anteroseptal scars typically have more extensive endocardial scarring, greater LV dysfunction, and a high prevalence of deep intraseptal substrates. Critical VT sites are often located in the left ventricular outflow tract or anteroseptal LV endocardium in up to 90% of these cases, often associated with higher recurrence rates following ablation. Epicardial mapping and ablation in these patients are typically not necessary unless there is scar tissue extending more laterally and towards the epicardium. Conversely, those with inferolateral scars usually present an epicardial substrate, with critical VT sites in the inferolateral epicardium in 63% of cases.[102]

Functional mapping during ventricular stimulation

In addition to substrate mapping, functional mapping can be used to identify lines of block and slow conduction zones. Substrate mapping improves target selection by narrowing the potential targets for pace-mapping in haemodynamically unstable VAs. Aside from applied cut-offs, the mapping-catheter type influences the substrate mapping.[103] The narrow, fluid-free space within the pericardium helps ensure optimal contact between linear catheters and the heart's epicardial surface.

Additionally, as the contact angle shifts from parallel to perpendicular, the bipolar signal amplitude decreases, and the electrogram starts to mirror a unipolar potential. In the empty pericardial space, both catheter electrodes maintain a contact angle close to 0°, resulting in a true bipolar electrogram with higher amplitude and fidelity, which is crucial for identifying near-field low-amplitude potentials.[103] The next step in sinus rhythm mapping involves functional mapping to identify slow conduction zones, complex potentials, and block lines.

Functional substrate mapping can detect areas of abnormal conduction and conduction block that occur in response to premature stimuli that are associated with re-entry circuits and may not be recognized based on sinus or paced rhythm alone.[104] Functional mapping includes but is not limited to, decrement-evoked potential mapping, sensed-evoked delayed potential mapping, and isochronal late activation mapping.[105–107]

Mapping during ventricular arrhythmia

With present substrate and functional mapping methods, mapping in VT is usually reserved for patients with slow, incessant VT during the procedure or who continue to have inducible clinical VT after a substrate-guided ablation. The same techniques applied to endocardial mapping can be applied to epicardial activation mapping.

Mapping consideration in special conditions

Arrhythmogenic right ventricular cardiomyopathy/dysplasia

Garcia *et al.*[108] initially demonstrated the feasibility and effectiveness of epicardial catheter ablation after previously undergoing unsuccessful endocardial VT ablation procedures. Based on current evidence, the use of an epicardial approach in ARVC/D patients undergoing VT substrate ablation can be tailored according to the substrate distribution pattern observed in the endocardial map.[109,110] Patients with a normal endocardial map or a limited endocardial low-voltage area should consider obtaining epicardial access. Conversely, a sequential strategy may be warranted for patients with significant endocardial involvement (e.g. a bipolar/unipolar low-voltage area ratio ≥ 0.23).[26] However, patients who are non-inducible after comprehensive endocardial ablation likely have a primarily endocardial arrhythmic substrate, showing minimal epicardial involvement, and might not benefit from a routine epicardial approach. Finally, first-line catheter ablation of monomorphic VTs in these patients reduced the composite primary

endpoint of VT recurrence, cardiovascular hospitalization, and death, which were driven by a reduction in implantable cardiac defibrillator (ICD) therapies.[111]

Hypertrophic cardiomyopathy

Endocardial and epicardial electroanatomic mapping and ablation represent an effective approach for treating VAs in selected patients with hypertrophic cardiomyopathy.[112] Substrate and functional mapping, along with activation and entrainment mapping on both the epicardial and endocardial surfaces, facilitates the identification of critical VT circuits. While endocardial-only ablation can successfully terminate VTs, often originating from an aneurysmal apex, the persistence of VT inducibility and prolonged VT termination time with reinduction may suggest the presence of an epicardial or intramyocardial substrate.[112]

Ischaemic cardiomyopathy

The epicardial approach is reasonable after a failed endocardial ablation, the presence of a large floating left ventricular thrombus, or an inaccessible LV due to double mechanical valves. A prior unsuccessful endocardial ablation alone, however, does not automatically justify performing an epicardial puncture, as the failure of the procedure may be due to incomplete endocardial mapping or insufficient ablation. Additionally, ablation can be successfully performed from the endocardium because of the presence of wall thinning. In a study of 93 patients with VT and ICM, definitive evidence of epicardial involvement in the VT circuit was found in only 13%, with many still successfully undergoing endocardial ablation.[113] Studies have found that an epicardial scar area $> 14\ cm^2$ on LGE MRI and/or wall-thinning < 3.59 mm is highly sensitive (1 and 0.91, respectively) and specific (1 and 0.93, respectively) indicators of epicardial arrhythmogenic substrates in post-MI patients.[114]

Brugada syndrome

Recent studies have improved our understanding of substrate abnormalities and ablation strategies in high-risk Brugada syndrome. Pappone *et al.*[115] identified novel electromechanical substrate abnormalities associated with arrhythmic risk, emphasizing the importance of thorough substrate mapping. Nademanee *et al.*[116] demonstrated that catheter ablation targeting the anterior epicardial region of the right ventricular outflow tract prevents ventricular fibrillation episodes, establishing ablation as an effective alternative to ICDs. The BRAVO registry further confirmed the long-term effectiveness of Brugada substrate ablation in reducing ventricular fibrillation recurrence.[117] Li *et al.*[118] showed that ablation significantly decreased ventricular fibrillation recurrence in high-risk Brugada syndrome patients who refused implantable cardioverter-defibrillator (ICD) implantation, providing an alternative for these patients. Santinelli *et al.*[119] reported that epicardial ablation combined with ICD implantation improved outcomes and reduced arrhythmic recurrence in high-risk Brugada syndrome. Collectively, these studies highlight substrate-guided ablation as a vital strategy for managing high-risk Brugada syndrome, tailored to individual patient risk and treatment preferences.[115–119]

Chagas heart disease

There are only a few reports of the results of Chagas VT ablation. An epicardial approach is usually necessary in >80% of patients with Chagas disease to achieve noninducibility at the end of the procedure. Performing epicardial ablation improves the outcomes of the catheter ablation in these patients.[120]

Section 6: Delivering lesions for the epicardial ablation of ventricular arrhythmias

Advice	Strength of evidence
Advice TO DO	
Achieving noninducibility and ablation of all late potentials are advised endpoints for epicardial VA ablation[121–124]	
Use of an irrigated-tip catheter is advised for epicardial ablation of VA[125–129]	
Setting 40–50 W as the upper power limit is advised when performing RF epicardial VA catheter ablation[130–132]	>90% agree

Epicardial ablation lesions can be created with radiofrequency (RF) ablation, cryoablation, and pulsed-field ablation. Ablation systems using ultrasound have also been introduced.[5,126,133]

Radiofrequency ablation

General considerations

During RF ablation, the current passing through tissue produces resistive heating, and this heat is transmitted to the tissue in the surrounding region. The region that reaches 50°C is irreversibly damaged and is surrounded by a region of reversible injury and oedema that will recover.[134] The amount of current that enters the myocardial target tissue is determined by multiple factors, including ablation time, the area of the ablation electrode in apposition with the tissue, the contact angle, catheter type, the contact force (CF), the power applied, and the current that is shunted away through the blood and surrounding tissue.[135] The size of the tissue area reaching 50°C expands as heat generated in the resistive heating zone conducts to the surrounding tissue. Depending on the catheter used, this effect may continue to increase over several minutes.[136,137]

Irrigated-tip catheter and epicardial lesions

Epicardial RF ablation is generally performed with externally irrigated catheter systems that maintain measured electrode temperature well below 70°C as high power is applied. External irrigation with unheparinized solution (5–17 mL/min) provides cooling of the epicardial surface, which reduces RF lesion width at the epicardial surface but not necessarily lesion depth.[138] In an *ex vivo* animal model, RF lesions at fixed power were larger with lower irrigated RF flow rates of 5–7 mL/min than at 10 mL/min with Thermocool STSF and Flexibility (Abbott) catheters.[138] In addition to a high-power setting (40–50 W), targeting baseline impedance $< 120\ \Omega$ (used by 20% of the writing group members) and use of a half-saline solution (used by 20% of the writing group members) can increase the lesion size during epicardial ablation of VAs.

Most of the writing group members (90%) employ 40–50 W as the upper power limit when performing RF epicardial VA catheter ablation. Among the writing group members, the torso (40%) or a combination of the torso and lower extremity, including the buttock (40%), was the most common area for placement of cutaneous skin patches for ablation of epicardial VAs, and only 20% use lower extremity including buttock only.

Non-irrigated catheter and epicardial lesions

RF ablation with a solid tip electrode, without irrigation, can create epicardial lesions.[126] The pericardial space is devoid of circulating blood to cool the electrode catheter such that there is likely less disparity between tissue and electrode temperature. For similar power and duration of RF, however, lesion size is greater with epicardial RF applications, compared to endocardial applications, likely due to less heat sinking from the circulating blood and the tendency for parallel orientation of the ablation catheter producing a larger tissue electrode interface compared to vertical orientation in the endocardium.[139,140] It is possible that higher temperature targets could be used in the epicardium, but this has not been explored.

Factors influencing epicardial lesions

Epicardial lesion formation during VA ablation is influenced by various factors, including energy delivery methods, contact force, ablation duration, and the choice of irrigation fluids, all of which impact the efficacy and safety of the procedure.

Power setting

In an *ex vivo* model, up to 70 W with an 8 mm electrode did not increase lesion size compared to externally irrigated RF ablation catheters.[136] A variety of approaches to adjusting power are in use and a single optimal approach has not been defined. During epicardial RF ablation, the impedance falls because of tissue heating in the same manner as during endocardial ablation.[126] In an animal model, RF lesion size correlated with the magnitude of the impedance fall. Some investigators titrate RF power to achieve an impedance fall of 10–15 Ω, or 10%.

Ablation duration

The duration at which lesion size plateaus is not defined. For epicardial scar-related arrhythmias, most investigators continue the RF application for at least 60 s. In an *ex vivo* model, lesion size increased beyond 3 min, and 5 min lesions were deeper than 3 min lesions.[136] Long-duration applications are sometimes used when the substrate is perceived to be located deep within the epicardium. Most of the writing group members use ≥60 s as a typical RF lesion duration for an epicardial VA ablation lesion (20% for 90 s, 40% for 60 s, 30% for <60 s).

Contact force

As in the endocardium, increasing contact force against the myocardium increases RF lesion size. Force-sensing catheters are potentially useful. Seventy-seven per cent of the writing members use CF catheters for epicardial VA ablation. Among the members, 22% target a minimum of 5–10 g, and 55% target a minimum of 10–20 g when using CF sensing catheters during epicardial VAs ablation; however, 22% only rely on the direction of the contact force vector. No consensus could be achieved for the appropriate upper CF limit to be implemented during epicardial VAs ablation with a CF sensing ablation catheter. However, most of the members use 30 g as an upper CF limit.

In the absence of force sensing, 22% of RF applications did not produce an identifiable lesion in an *in vivo* ovine model.[140] The presence of sharp electrograms and the ability to capture with pacing from the ablation catheter are also indications of contact. The writing group members, due to lack of clear evidence, did not reach a consensus regarding lesion size indices to guide energy delivery. Only half of the members believed that lesion size indicators (like ablation index, lesion size index, and force-time integral) are mandatory to guide energy delivery during epicardial VA ablation.

Selection of irrigation fluids

During ablation with external irrigation, the saline irrigant accumulates in the pericardial space and requires drainage, preferably continuously.[141] The presence of normal saline in the pericardial space reduces RF lesion diameter and depth by increasing current shunting away from the target tissue.[138] The use of half-normal saline irrigation can reduce current shunting away from the target area, increasing current delivery for a given power compared to normal saline irrigation and potentially increasing lesion size.[136] Lesion size is like that of lower osmolar fluid (Dextrose 5% in Water—D5W). Therefore, the use of half-normal saline could facilitate lesion creation in epicardium, but further study is needed.

Pacing and endpoints

Finally, routine pacing may be beneficial to assess the epicardial lesion creation.[142] In our survey, 60% of the writing group members routinely use pacing to assess lesion creation. The majority (80%) of writing group members defines noninducibility and ablation of all late potentials as required epicardial VA ablation endpoints.

Steam pops during epicardial irrigated radiofrequency ablation

Steam pops can occur during epicardial irrigated RF ablation and are more likely as power is increased.[126] In an animal model, steam pop produced an epicardial 'crater' without perforation to the endocardium. In an *ex vivo* model, steam pops were observed more frequently and earlier with RF applied over fat than over myocardium, which was consistent with the greater resistance and lower thermal conductivity of adipose tissue.[138] Irrigation with D5W was associated with more steam pops as compared to irrigation with normal saline or half-normal saline at the same power in an experimental model.[143] In human studies, steam pops have been uncommon, and we are unaware of any cardiac perforation related to steam pops in the epicardium.

Epicardial fat and irrigated radiofrequency ablation

Epicardial fat has lower electrical and thermal conductivity than muscle, limiting RF lesion creation.[126,144] Excessive intramural heating with steam pops is more likely to occur when heating over fat.[138] In human studies, effective RF lesions have been unlikely in regions where epicardial fat thickness is more than 6.1 mm on CT imaging.[145] Increasing CF (from 10 to 30 gm), long-duration applications, up to 5 min, and use of half-normal saline irrigation increased lesion size with RF applied over fat in an *ex vivo* model[136] (see Section 9).

In the presence of ≥2.8 mm of fat, unipolar or bipolar voltage does not discriminate between fat and scar.[39] Fat thickness of more than 2.8 mm was associated with bipolar electrogram amplitude of <1.5 mV, even in the absence of a scar.[146] Bipolar electrogram amplitude > 1.5 mV predicted the absence of fat. Fat does not alter electrogram duration, such that fractionated; long-duration (>50 ms bipolar) electrograms and late potentials are reliable indicators of fibrosis, although fat could still be present.[39,147]

Unipolar vs. bipolar epicardial radiofrequency ablation

Unipolar RF is usually applied between the catheter tip electrode and a dispersive cutaneous electrode. Bipolar ablation can be applied between ablation electrodes positioned on either side of the free wall of the ventricle, with one electrode on the epicardium. In an *ex vivo* animal model, bipolar RF produced deeper lesions than unipolar epicardial RF with normal saline irrigation, but not as deep as unipolar RF with half-normal saline.[136]

In an *in vivo* model, bipolar RF ablation (TactiCath™; Abbott, Chicago, IL, USA) at 30 W for 60 s, with normal saline irrigation at 30 mL/min, produced transmural lesions in 62% of applications in tissue with a mean thickness of 11.78 ± 5.12 mm and a 3.5% incidence of steam pops. This is compared to 7% transmural lesions achieved with sequential unipolar applications in thinner tissue (mean thickness 4.16 ± 0.66 mm) but with a higher steam pop rate of 10%.[148] This approach has also been used in humans, but data are limited.[149]

Other energy sources

Cryoablation has been studied in open-chest animal models, and there is substantial experience with epicardial cryoablation in surgical approaches to the epicardium.[150–152] The absence of circulating blood in the pericardial space facilitates freezing, and epicardial cryoablation produces a larger lesion than an endocardial application with the same catheter.[153,154] In the beating heart, endocardial circulating blood provides a heat sink that limits lesion depth.[155] Surgical epicardial cryoablation has been used to target epicardial VT and atrial arrhythmias and as part of a maze procedure.[133,152,156,157]

Electroporation has been studied and shown to be capable of creating large lesions that can exceed 1 cm in depth and potentially result in transmural lesions.[158–160] Recent studies show improved safety and energy delivery for epicardial ablation. These findings highlight its potential to potentially enhance VT ablation outcomes, warranting further studies.[161] Coronary spasm is a concern with epicardial ablation. Experimental studies suggest that laser and microwave energy appear more effective than RF ablation for creating lesions below the fat.[144] High-intensity focused ultrasound has been studied in experimental models and has the potential to create larger lesions than have been achieved with RF ablation.[162,163]

Section 7: Prevention and management of complications

Advice	Strength of evidence
Advice TO DO	
Availability of immediate in-house cardiac surgical backup is advised at all centres performing epicardial ventricular arrhythmia (VA) ablations[7,8,164,165]	 >90% agree
It is advised to perform routine echo-guided exclusion of pericardial effusion before the patient leaves the EP-lab in patients undergoing epicardial VA catheter ablation[166,167]	 >90% agree
Performing a phrenic nerve capture test and marking its course before epicardial VA ablation is advised[168]	
Measures to protect phrenic nerve including use of balloon, air, or fluid instillation in case of phrenic capture at a provisional ablation site are advised[169–172]	

Continued

Continued

Advice	Strength of evidence
Visualization of coronary arteries using selective coronary angiography is advised before performing epicardial VA ablation in areas with possible proximity to coronary arteries[145,173]	 >90% agree
In case of a high probability of coronary artery damage, performing 12-lead ECG for monitoring of ST-segment during the procedure is advised	 >90% agree
Selective coronary angiography after epicardial ablation of VAs in the presence of new and/or dynamic ST-segment changes is advised	 >90% agree
Performing abdominal sonography and if necessary, CT with contrast in patients with suspected intra-abdominal bleeding and/or injury is advised after epicardial VA ablation	 >90% agree
Advice NOT TO DO	
Routine abdominal sonography is not advised after performing epicardial VA ablation	 >90% agree
Routine chest X-ray assessment after epicardial VA ablation to exclude phrenic nerve damage is not advised	 >70% agree

Epicardial access and interventions can be associated with serious complications. The reported complication rate varies from 1% to 17.5%.[174,175] Some complications may occur immediately after the procedure, while others can be delayed. The most serious complications associated with the procedure are listed below, along with some suggestions about their prevention, diagnostics, and management. All centres performing epicardial VA ablation are encouraged to establish a registry for documenting potential complications and for transparent follow-up and monitoring.

Factors associated with procedural complications

Table 7 summarizes factors contributing to increased complications during epicardial access and ablation of VAs. Awareness of these situations is therefore required to assess whether to proceed with a percutaneous attempt or switch to a surgical approach, either via the subxiphoid or the lateral thoracotomy (pericardial window).[177,178]

General rules to prevent complications

Preventing complications in epicardial interventions requires adherence to established standards and meticulous planning to ensure patient safety and procedural success. The following principles should always be observed:

- Careful interdisciplinary preoperative assessment and procedure planning;
- Proper operator training and maintaining of high level of expertise;
- Adequate periprocedural patient monitoring;
- Appropriate periprocedural anticoagulation management to balance the risks of thrombotic and bleeding complications; and
- Availability of surgical and cardiac interventional backup in case of cardiac perforation with tamponade or ST elevation suggesting damage to coronary arteries.

Some small observational studies suggest that such procedures can be safely performed without in-house cardiac surgery in highly experienced centres.[179] However, all members of the writing group agreed that the availability of immediate in-house cardiac surgical backup is advised for all centres performing epicardial VA ablations.

Access specific complications

The inferior technique can potentially cause intra-abdominal bleeding through an inadvertent puncture of the diaphragm and sub-diaphragmatic structures (see *Figures 6* and *7*). The anterior approach is associated with an increased risk of puncturing the superior epigastric artery or LIMA. However, an inferior puncture approach is preferred in certain situations (e.g. a history of cardiac surgery and/or anterior sternotomy) (see Section 2).

A retrospective study evaluated the safety of anterior and inferior epicardial puncture techniques in 211 patients (average age 61.4 years, 84.8% male) undergoing 271 VA ablation procedures. Results showed that major complications occurred in 12.5% of procedures, with 8.5% directly related to the puncture. Anterior puncture was associated with a lower rate of complications (4.9%) compared to the inferior approach (10.1%). Additionally, damage to adjacent structures like the liver, colon, and gastric vessels occurred exclusively in the inferior access group.[59]

Table 7 Factors associated with increased rate of complications during epicardial access and ablation of ventricular arrhythmias

Patient-specific factors
• History of previous cardiac surgery or pericarditis
• Obesity
• Underlying coagulopathy
• Renal insufficiency
Anatomical variations
• Presence of pectus excavatum
• Proximity of the ablation site to the phrenic nerve and major coronary arteries
Procedure-related factors
• Prolonged procedure duration
• Extensive, long, and high-energy ablation
High PAINESD score[176]

Possible complications

Pericarditis

Pericarditis is the most common complication after epicardial VA ablation, occurring in up to 40% of patients.[180] In rare cases, constrictive pericarditis may also occur after epicardial procedures.[181] Clinical presentation, investigation, management, and prevention of pericarditis and its related symptoms are summarized in *Figure 14*. The risk of post-procedural pericarditis can be reduced by minimizing epicardial RF energy delivery as much as possible. Removal of the sheaths is advised at the end of the procedure to minimize irritation and inflammation, except in case of persistent bleeding (see Section 8—Access management). If there is no haemopericardium after the procedure, removing the pericardial catheter and sheaths immediately after intrapericardial steroid instillation is reasonable.

Management of pericarditis-related symptoms

There is a high incidence of pericarditic chest pain and ECG changes after epicardial VT mapping and ablation. Intrapericardial steroids significantly decrease pericarditic chest pain.[184,185] Intrapericardial liposomal bupivacaine is associated with significantly decreased numeric pain scores up to 48 h and a shorter length of hospital stay without an increase in the risk of adverse events.[183]

If pericarditis-related symptoms persist, non-steroidal anti-inflammatory drugs with or without colchicine can be administered

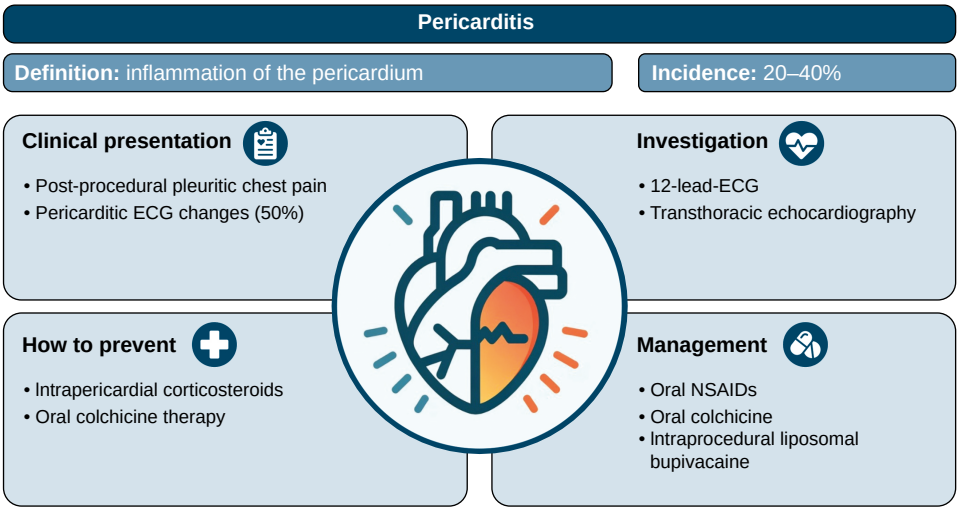

Figure 14 Clinical presentation, investigation, management, and prevention of pericarditis and pericarditis-related symptoms.[180,182,183] NSAID, non-steroidal anti-inflammatory drugs.

for pain control.[186] The use of colchicine for 7–10 days is associated with a significant reduction in post-procedural pericarditis and its associated pain and complications.[182] Non-steroidal anti-inflammatory drugs are given for 1–2 weeks and tapered off over the next week if the patient shows improvement/resolution of symptoms (see also Section 8).

Intrapericardial post-procedural liposomal bupivacaine and oral colchicine are other therapeutic options, which were found to be associated with significantly decreased numeric pain scores.[183] It was also associated with a significantly shorter length of hospital stay.[182] Despite these measures, some patients may continue to have severe chest pain and require opioids. In extreme cases, pericardiectomy due to recurrent drug-resistant chronic pericarditis after epicardial VT ablation may be used to alleviate the symptoms.[187]

Haemopericardium and tamponade

Haemopericardium and tamponade are life-threatening and are important complications associated with epicardial access and interventions. It usually occurs due to cardiac puncture or epicardial vessel damage by a puncture needle; however, it can also occur by a guidewire, catheter, or sheath, especially in patients with pericardial adhesion. Clinical presentation, investigation, management, and prevention of tamponade and haemopericardium are summarized in *Figure 15*.

Early haemopericardium

Most cases of haemopericardium that occur within 10 min of pericardial access are related to RV puncture or vascular damage and can be easily diagnosed and monitored with intracardiac or transthoracic echocardiogram[167,188–190] (see Supplementary material online, *Figure S15.1*). If coronary arterial bleeding is suspected, prompt diagnosis should be made with a coronary angiogram at the same time as pericardial drainage, and the amount of bleeding determines the use of a graft stent or surgical repair (*Figure 15*). Most writing members (90%) routinely perform echocardiography to exclude pericardial effusion before the patient leaves the EP laboratory.

Intraprocedural haemopericardium

Haemopericardium in the middle of the procedure is usually caused by slow bleeding after multiple punctures or vascular damage by catheter manipulation or steam pop.[167,188] Slow and continuous bleeding requiring surgical repair by the laceration of the middle cardiac vein or coronary vein has also been reported.[191]

Post-procedural haemopericardium

Post-procedural haemopericardium (<1%) is due to access track bleeding or, very rarely, double RV puncture.[167,188,190,192] The double RV puncture occurs more frequently with the anterior approach. Some operators suggest advancing a long wire through the pericardial sheath before its removal to permit sheath readvancement to seal the perforation sites and stabilize the patient until surgery. Another possible complication with similar consequences is a tear of the ventricular wall if the wire passes through the adhesions and the sheath is advanced over the wire. It may not bleed during the mapping and ablation, but bleeding starts when the sheath is removed.

RV pseudoaneurysm, discovered as chest pain one month after successful epicardial ablation, has also been reported.[191] Delayed haemopericardium may occur after the resumption of anticoagulation therapy, and Dressler syndrome (post-cardiac injury syndrome) may also occur after extensive epicardial ablation or long catheter indwelling time (<1%).[190]

Most procedure-related haemopericardium cases can be managed with catheter drainage. However, immediate blood transfusion, surgery, and extra-corporeal membrane oxygenator backup should always be available. Above all, the operator's calm, training, and experience, as well as ultrasound monitoring, are important.[193]

Left internal mammary arterial injury

While the anterior approach of percutaneous epicardial puncture reduces the risk of RV damage, it can be accompanied by LIMA damage.[194] The risk of LIMA damage is particularly high in very anterior

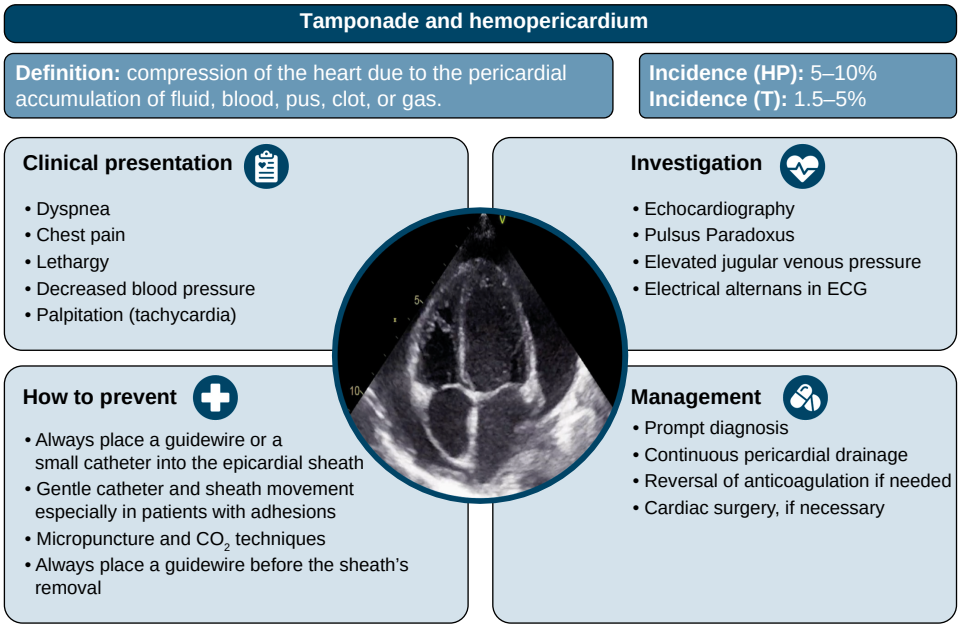

Figure 15 Clinical presentation, investigation, management, and prevention of tamponade and haemopericardium.[167,188–190] HP, haemopericardium; T, tamponade.

punctures (puncture needle angle < 20°) or higher-than-usual punctures. Left internal mammary artery injury can rarely occur also during inferior puncture (see Section 2).

Coronary artery injuries

Epicardial RF ablation in proximity to coronary arteries produces lesions ranging from thickening and damage to the intima and media with replacement of the media smooth muscle cells by the interstitial matrix to complete occlusion.[195–197] Changes clearly occur when RF is applied very close to the vessel.[197] RF ablation should be avoided within 5 mm of major epicardial arteries to minimize the risk of vascular injury.[7] Multiple factors likely determine the relation between the proximity of the ablation site and coronary damage, including the presence of adjacent fat and coronary blood flow that may reduce coronary arterial heating.[197]

To avoid coronary artery injuries, performing a coronary angiogram is appropriate to outline the location of major epicardial vessels relative to the region of interest before RF ablation. The 2019 HRS/EHRA/APHRS/LAHRS consensus statement on VT ablation recommends a distance of at least 5 mm away from the coronary vessel for ablation before ablation is considered.[7] Multiple angiographic views are recommended to assess the distance appropriately. Real-time integration of multidetector CT-derived coronary anatomy or angiogram can be used (*Figure 16*).[173] All the writing group members visualize the coronary arteries using selective coronary angiography or pre-procedural imaging before performing epicardial VA ablation in areas with possible proximity to coronary arteries. After the procedure, in case of a high probability of coronary artery damage, all members of the writing group perform a 12-lead ECG for ST-segment monitoring, and they perform selective coronary angiography after epicardial ablation of VAs in the presence of new and/or dynamic ST-segment changes.

Left phrenic nerve injury

Phrenic nerve damage with consequent diaphragmatic paralysis is a rare, well-recognized, but preventable serious complication of epicardial VA ablation[168,198] (*Figures 17* and *18*). The left phrenic nerve courses most frequently aside from the mid-ventricular wall (around 50% of cases), but more apical or basal courses are not infrequently encountered (see Section 2). Phrenic nerve limits the epicardial ablation in 7% of the patients.[169] Initial experience with pulsed-field ablation in the pericardial space suggests a favourable safety profile concerning phrenic nerve complications; however, further large-scale studies are needed to confirm these findings.[161,203]

Mapping of the phrenic nerve capture sites by high output low-rate pacing with the ablation catheter remains the only precise and reliable option to locate the phrenic nerve. Since many epicardial ablation procedures are performed under general anaesthesia, it is crucial to inform the anaesthetist to avoid using muscle relaxant agents during this manoeuvre. If residual muscle relaxant activity is present, administering an antagonist can help prevent false-negative responses.

Prevention of phrenic nerve injury

Several strategies have been reported to prevent phrenic nerve injury.

- Direct displacement of the pericardium, and consequently the phrenic nerve, from epicardial ablation sites can be accomplished by introducing and inflating peripheral angioplasty or valvuloplasty balloons.[170,204] This procedure requires double wiring of the epicardial puncture site and the subsequent introduction of two deflectable sheaths to steer the angioplasty device accurately.[169] For this specific purpose, the inferior puncture would probably allow better handling of the introducers, with a shorter and more direct course to the left posterior wall. The left anterior oblique projection is critical to confirm that the inflated (50% mixture of contrast and saline) balloon lies externally to the ablation

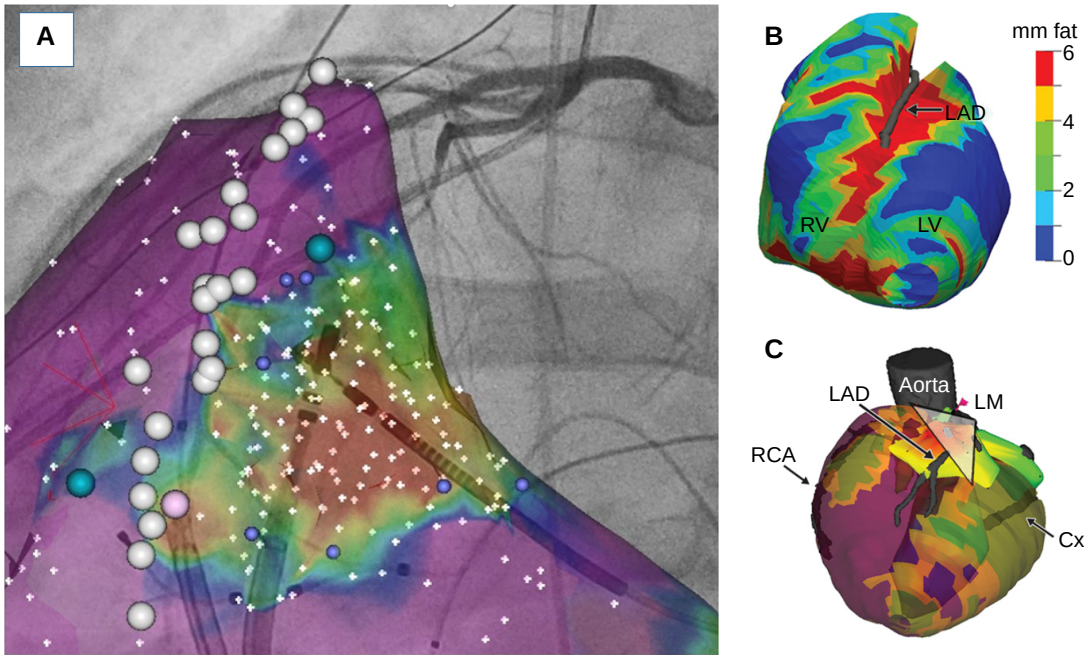

Figure 16 (*A*) Integration of coronary angiography in 3D mapping during the ablation procedure. The large white points delineate the course of the left phrenic nerve. (*B*) Three-dimensional epicardial surface mesh colour-coded for fat thickness. (*C*) Fusion between the electroanatomical mapping and epicardial three-dimensional surface reconstruction showing coronary arteries and colour-coded fat thickness, registered using the left main coronary artery (LM) landmark. The triangle demonstrates the predicted epicardial target site in the vicinity of the left anterior descending artery (LAD), covered by >4 mm of fat. Images *B* and *C* are adapted with permission from van Huls van Taxis *et al.*[145]

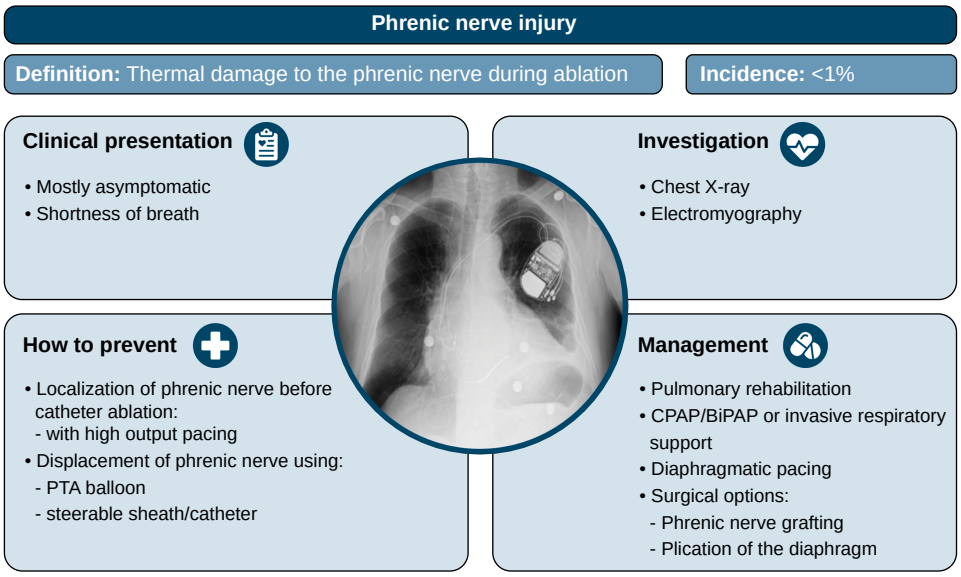

Figure 17 Clinical presentation, investigation, management, and prevention of phrenic nerve damage.[168,198–202] BiPAP, bilevel positive airway pressure; CPAP, continuous positive airway pressure; PTA, percutaneous transcatheter angioplasty.

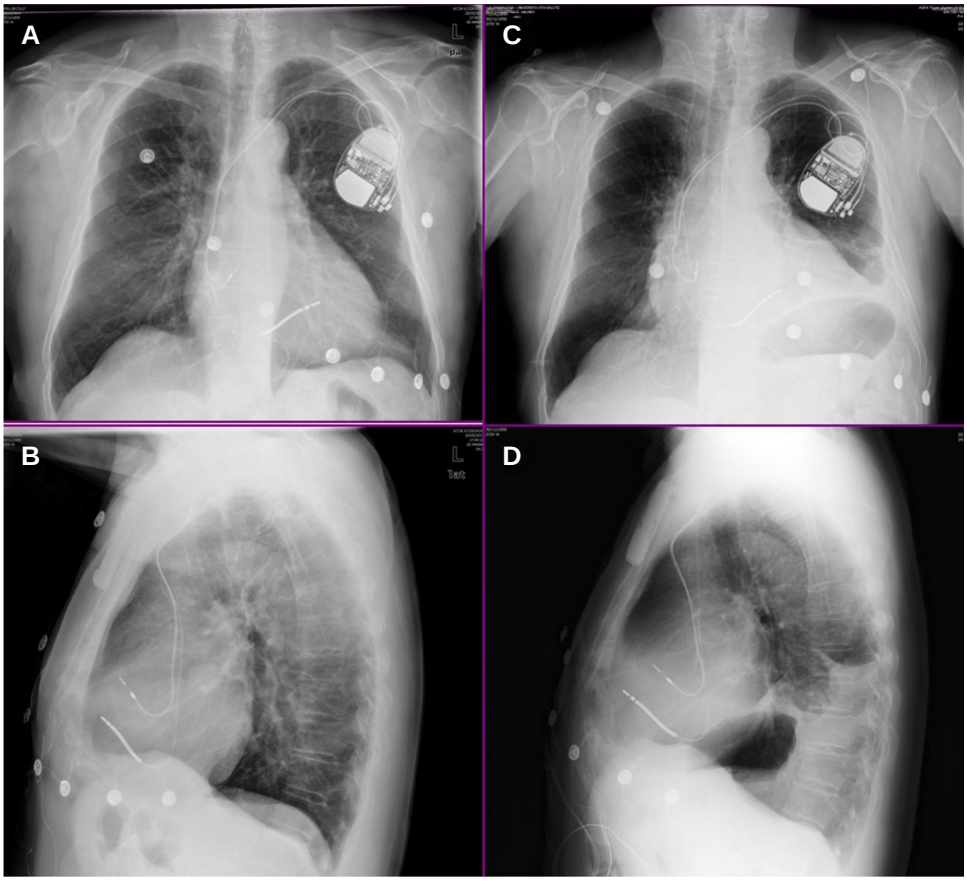

Figure 18 Phrenic nerve palsy after epicardial ablation for refractory ventricular tachycardia. (*A* and *B*) Anteroposterior and lateral chest X-rays before the procedure, showing the normal position of the left diaphragm dome. (*C* and *D*) Elevation of left hemidiaphragm, particularly in the posterior segment, after epicardial ablation. Note the relative elevation of the gastric bubble after the nerve palsy.

catheter tip to protect the pericardial membrane's displaced portion (see Supplementary material online, *Figure S17.1*). Loss of phrenic nerve capture after inflation should always be confirmed before proceeding with the ablation.
- CO_2 or fluid instillation is also beneficial for phrenic capture at a provisional ablation site.

All writing group members conduct a phrenic nerve capture test before epicardial VA ablation when ablating near the potential course of the phrenic nerve. The most common approach used by the writing group members to manage the phrenic nerve capture at a provisional ablation site was the balloon among 67% of members, whereas fluid instillation in the pericardium (22%) or air insufflation in the pericardium (11%) were the other approaches. Should the pericardial displacement strategy fail in preventing phrenic nerve capture at the selected ablation sites, the percutaneous epicardial ablation should be aborted, and a subsequent open chest (preferably via lateral thoracotomy) scheduled at another time to safely perform the procedure under visual guidance. All members of the writing group agree that routine chest X-ray to exclude phrenic nerve damage after epicardial VA ablation is not necessary.

Intra-abdominal bleeding and injuries

Intra-abdominal bleeding may also occur during the pericardial puncture, resulting in the development of a haemoperitoneum. However, its incidence is very rare (around 0.5%) in one case series[187] (see Supplementary material online, *Figure S17.2*). The bleeding can originate from a puncture of the liver or vessels beneath the diaphragm (see also Section 2). This complication may be detected after the procedure at different time periods and may require a blood transfusion or even surgical haemostasis. Bleeding within the liver may occur when the needle traverses the enlarged liver.[57] To minimize the risk of gastric injury during needle insertion, one could consider gastric decompression in patients exhibiting gastric dilatation (see Supplementary material online, *Figure S17.3*). This complication may present as abdominal pain with or without rebound tenderness. Abdominal sonography and, if necessary abdominal CT with contrast were the preferred approaches among the writing group members to exclude intra-abdominal bleeding and/or injury in case of abdominal pain after epicardial VA ablation. However, routine abdominal sonography is not deemed necessary after the procedure.

Oesophagus injury

Oesophageal damage after epicardial VT ablation is a rare complication. However, cases of pericardial oesophageal fistula and mediastinal oesophageal fistula that occurred after basal posterior wall near coronary sinus epicardial ablation have been reported.[205,206] Since the vagus nerve runs along the anterior aspect of the oesophagus towards the stomach, injury to this structure could lead to delayed gastric emptying[207] (see Supplementary material online, *Figure S17.4*).

Lungs and pleura injury

Due to the parietal pericardium's direct contact with the mediastinum, pleura, and lung, extensive epicardial RF energy delivery may result in collateral injuries.[126,187] Lung or pleural injury is an extremely rare complication.[187] Pleural effusion due to post-cardiac injury syndrome or procedure-related haemopericardium has been reported[187] (see Section 2).

Air in the pericardial space

Air may inadvertently be introduced into the pericardial space during catheter manipulation or catheter exchanges, and it can be easily detected on fluoroscopy. It does not usually have haemodynamic consequences; however, large amounts of air may increase the transthoracic defibrillation threshold and distort local ECGs and 3D electroanatomical mapping.

Atrial fibrillation

AF may complicate the post-ablation course of epicardial access. One study documented new-onset AF in 8 of 41 patients with epicardial ablation (19.5%).[208] All these patients had clinical symptoms of pericarditis and responded to short-term antiarrhythmic therapy or electrical cardioversion. At 18.0 ± 9.0 months of follow-up, no patient had a recurrence of AF, and all were without antiarrhythmic drugs.

The risk factors for AF included a lack of amiodarone after ablation, inadvertent right ventricular puncture, longer epicardial ablation times > 600 s, and significant pericardial pain on the day after the procedure. Current knowledge suggests that pericardial steroids and prophylactic antiarrhythmic drugs may decrease the risk of AF after epicardial ablation. Intrapericardial steroids can significantly reduce ECG changes due to pericarditis and AF after epicardial ablation.[184]

Section 8: Post-procedural management

Advice	Strength of evidence
Advice TO DO	
It is advised to perform first follow-up ICD interrogation within 4–8 weeks after epicardial VA catheter ablation, and then as routine or remote ICD-monitoring[209]	 >70% agree
Assessment of discontinuation of antiarrhythmic drugs after complete successful catheter ablation is advised	 >70% agree
May be appropriate TO DO	
Triamcinolone (2 mg/kg) or Methylprednisolone (250 mg) for intrapericardial injections after the procedure may be useful to avoid adhesions and chest pain[184,185]	
Routine administration of colchicine in patients undergoing epicardial VA catheter ablation to reduce the risk of post-procedural pericarditis and chest pain may be useful[182]	 >90% agree

Access management

At the end of the epicardial procedure, the first step is to manage the epicardial access space. We suggest checking for epicardial fluid with aspiration and echocardiography (transthoracic or intracardiac). After emptying the epicardial space, intrapericardial injection of triamcinolone or methylprednisolone to prevent epicardial inflammation and adhesions is reasonable, which can prevent subsequent epicardial access for future epicardial ablations.[185] However, around one-fourth of the writing members do not routinely use intrapericardial injections (see Section 7).

Early removal of the epicardial access, 30–60 min after the ablation procedure, can decrease post-procedural chest pain.[210] However, on

the other side, late tamponade after epicardial ablation is a known potentially lethal complication; therefore, keeping the epicardial drainage, if the aspirated epicardial fluid shows haemopericardium, for 6–12 h after the procedure and remove it, as soon as the drainage volume is <30 mL/h seems to be reasonable.[26,211]

Post-procedural care and monitoring

After completing the procedure, extubation, and removal of the sheaths, patients should be transferred to an intermediate care unit for 12–24 h. In case of haemodynamical stability, patients can be transferred to the ward with continuous telemetry until hospital discharge. Haemodynamic decompensation after access removal without pericardial bleeding suggests bleeding along the access tract into either the chest or the abdomen. In this case, a surgical consultation should be obtained immediately; CT imaging and an angiography of the internal mammary, if necessary, is advised[212] (see also Section 7).

Device interrogation

Device interrogation should be done before the patient leaves the EP Lab. The patient's chart should clearly reflect whether ICD antitachycardia therapies have been turned on after the procedure. Early recurrences, within 30 days, account for up to 50% of all recurrences within the first 12 months after VT ablation and are associated with increased mortality.[213–215] Hence, a structured follow-up, ideally using remote ICD monitoring, is essential for the early detection and management of recurrences following epicardial VA ablation. Almost all the writing members (90%) believe that the first follow-up ICD interrogation should be scheduled within 4–8 weeks after epicardial VA catheter ablation, and then routine follow-up or remote ICD monitoring is advised.

Anticoagulation management

Bleeding is the principal risk associated with pericardial access, primarily due to inadvertent cardiac puncture rather than anticoagulation management. Therefore, the main emphasis on enhancing safety should be the prevention of inadvertent cardiac puncture. Initiation or resumption of anticoagulation in 3–5 h after the procedure is reasonable. However, it is advised to rule out haemopericardium before restarting anticoagulation therapy. One study reviewed the anticoagulation strategy before and during pericardial access for 355 patients (average age 57 ± 14 years) undergoing VA mapping and ablation. Significant pericardial bleeding occurred in 46 patients (13%) irrespective of anticoagulation strategy ($P = 0.720$). Inadvertent cardiac puncture and left ventricular ejection fraction ≤ 35%, but not the anticoagulation strategy, were independently associated with pericardial bleeding.[52] Further studies are warranted to assess the safety of uninterrupted anticoagulation in epicardial ablation procedures.

Antiarrhythmic drug management

The decision to continue or discontinue antiarrhythmic drugs after ablation should be individualized and tailored to the specific clinical scenario, the effectiveness of the ablation, the presence of residual arrhythmogenic substrates, and the patient's overall risk profile for arrhythmia recurrence.[209,216]

Seventy per cent of the members of our writing group prefer to use short-term (3-month) antiarrhythmic drugs after catheter ablation to prevent early VAs recurrences following epicardial VA ablation. However, the writing group members emphasize the importance of carefully evaluating the discontinuation of antiarrhythmic drugs following successful catheter ablation. None of the members recommends long-term (more than 3 months) use of antiarrhythmic drugs for VA recurrence prevention.

Section 9: Specific scenarios

Advice	Strength of evidence
Advice TO DO	
It is advised to consider patient's body mass index when assessing risk of complications, outcome, and eligibility for epicardial VA ablation[217]	 >90% agree
A surgical epicardial window is advised after cardiac bypass surgery[177,218]	
May be appropriate TO DO	
A surgical epicardial window may be a useful alternative to standard subxiphoid puncture after non-bypass cardiac surgery OR in repeat procedures with severe adhesions[177,218–220]	

This section discusses how to overcome the more frequent challenges in epicardial VA procedures.

Obesity

Obesity can complicate epicardial access due to challenges in palpating anatomical landmarks, difficulties with fluoroscopic visualization, and the need to use steeper angles. Additionally, an anterior approach may be particularly challenging in patients with abdominal obesity. Epicardial access in patients with elevated body mass index can be obtained with similar success and complication rates as normal-weight individuals and is not a contraindication. However, half of the writing group members prefer to defer epicardial VA ablation in patients with morbid obesity (body mass index > 40) until significant weight loss is achieved unless there is an electrical storm, therapy refractory VT, or incessant VA.

Adhesions

Pericardial adhesions may harden or prevent access to the epicardial space. These adhesions are particularly common in patients with prior cardiac surgery. The obstacles to adequate epicardial mapping and ablation associated with pericardial adhesions may present three distinct challenges:[221]

(1) Accessing the epicardial space: When adhesions are present around the pericardial puncture site, tactile feedback may become distorted, along with insufficient spreading of the injected contrast medium.
(2) Restricted access to some potential regions of interest.
(3) Risk of complications: Blunt dissection of pericardial adhesions may increase the risk of significant bleeding in the pericardial space and possible tamponade, as inadequate drainage of the effusion can occur due to adhesion-related compartmentalization that makes pericardial blood inaccessible to percutaneous aspiration.

In cases of severe adhesions, minimally invasive surgical approaches, such as accessing the epicardial area through a subxiphoid window or a limited anterior thoracotomy, are generally advised. In some very experienced centres, dense adhesions after prior non-coronary cardiac surgery or pericarditis may be carefully bluntly dissected with a deflected ablation catheter and/or steerable sheath, allowing for sufficient

epicardial mapping in most patients.[220] In some other instances, a hybrid surgical-interventional approach may also be appropriate (*Figure 10*).[222]

Occasionally, pericardial adhesions are encountered in the absence of a prior history of cardiac surgery. This phenomenon may contribute to the roughly 2% incidence of unsuccessful percutaneous access to the pericardial space in patients undergoing VT ablation. The prevalence of pericardial adhesions was 8% in a retrospective series of 155 patients undergoing a first attempt of epicardial VT ablation, which was associated with underlying renal disease and encompassed poorer short-term procedural success.[223] Pre-procedural assessment of the extent and density of pericardial adhesions by means of CT scan, CMR, and/or 3D-echocardiography should be advisable prior to determining the mapping/ablation strategy of choice (either surgical or percutaneous) on an individual basis, especially in the setting of prior cardiac surgery.[224–226]

Repeat procedures

Pericardial adhesions seem to be the primary limitation for repeating an epicardial VA ablation procedure in the epicardial space. However, in 30 patients undergoing repeat mapping and ablation, access to the pericardial space was achieved in all patients a median of 110 days after the last ablation procedure. Significant adhesions were observed in seven (23%) of the cases, with six of these patients having received intrapericardial triamcinolone acetate during prior procedures. Even with pericardial adhesions present, complete epicardial mapping was successfully performed using blunt dissection, achieving noninducibility in 90% of patients, with no complications related to the pericardial access or disruption of the adhesions.[227] In a series by Tung and colleagues, successful repeat access into the pericardial space was achieved in 88% (14 of 16 cases) of 11 patients who had prior epicardial access 8 days to 3.5 years before the re-ablation procedure.[189]

Previous cardiac surgery

Patients with a history of prior cardiac surgery with sternotomy usually have compromised access to the anterior epicardial space due to post-surgical adhesions along the anterior surface of the heart. These patients would require an inferior epicardial puncture approach, with the inherent increased risk of intra-abdominal bleeding through an inadvertent puncture of the sub-diaphragmatic structures[228] (see Section 4).

The reported success rate of epicardial access in this setting is variable in the literature, ranging from 15% to 90%, although restriction of catheter movement was observed in all patients, and meticulous dissection of adhesions may be necessary.[229] Once the pericardium is accessed, contrast may be injected to confirm the location of the guidewire. In patients with diffuse and dense anterior epicardial adhesions, the contrast will pool along the inferior heart border as opposed to rapid dispersion around the cardiac silhouette. Typically, it is not possible to further advance the guidewire to encircle the cardiac silhouette, and for a highly experienced operator, it may be reasonable to first advance a smaller 5 F introducer over the guidewire to allow further injection of contrast medium to validate pericardial access before insertion of larger bore epicardial introducers and sheath. It has been postulated that epicardial adhesions are usually safely disrupted by using the elbow of the mapping/ablation catheter, with self-limiting and minor pericardial bleeding as the main complication.[220]

Epicardial fat

The areas of epicardial fat frequently follow the course of the main coronary vessels at the atrioventricular and interventricular grooves. In a series of 25 patients undergoing epicardial mapping for VT who had CT imaging, an average of 65 ± 16% of the epicardium was covered by fat, with 25 ± 15% covered by more than 4 mm of fat with the thickest regions over the basal and apical anterior RV and basal superior LV walls.[145]

Impact on ablation lesions

Epicardial fat both increases the distance between the ablation catheter and the actual VT 'origin' and impedes an adequate penetration of the RF energy due to the fat's low thermal and electrical conductivity.[126] The total amount of epicardial adipose tissue is directly associated with unsuccessful VT ablation.[230,231] Beyond its potential pro-arrhythmic role, the main explanation appears to be both the proximity of some VT origins to the great epicardial coronary arteries and an insufficient RF energy penetration to the VT origin from the epicardial surface through fat tissue.

Challenges in mapping and voltage interpretation

A >5 mm adipose layer attenuates the myocardial voltage, making the conventional 1.5 mV values for epicardial bipolar mapping potentially misleading.[232–235] It is suggested that the recognition of abnormal myocardium in this setting should not only lie on the absolute voltage value but also on the electrogram fragmentation and late and/or split potentials.[39,233,235] Impaired pacing capture, which limits the effectiveness of pace- and entrainment mapping, is another challenge posed by the interposition of a thick fat layer during epicardial mapping.

Emerging mapping and ablation strategies

Impedance mapping has emerged as a promising technique for differentiating normal and low-voltage areas (normal to low impedance) from epicardial fat (high impedance).[236,237] Epicardial fat patches can be effectively distinguished from the epicardium using a multidetector CT scan and integrated as a separate chamber into the mapped geometry. This facilitates the identification of fat interposition during VT mapping and ablation.[238]

Epicardial pulsed-field ablation creates uniform lesions and penetrates collagen and fat layers more effectively than RF ablation, though further research is needed to confirm its superiority.[239,240] Cryoablation is another possible alternative for VAs arising from the epicardial LV summit.[241]

Section 10: Training requirements

Successful and safe execution of epicardial VA ablation requires clinicians to have comprehensive training and to work in a well-equipped and adequately supported facility.[165]

Training requirements and competencies

Epicardial ablation for VA requires the expertise and coordination of various healthcare professionals (physician, anaesthesiologist, surgeon, imaging team), each playing a crucial role in the patient's care. This approach to team preparation includes:

- All practitioners, including cardiologists, electrophysiologists, anaesthesiologists, nursing staff, and technicians, understand the intricacies of epicardial VA ablation.
- Ongoing education and training opportunities in the field are provided.
- Access to advanced diagnostic and therapeutic technologies that are essential for the accurate mapping, effective ablation, and management of VAs is facilitated.
- Patient care plans that address pre-procedural preparations, intraoperative strategies, and post-operative care, including the management of potential complications, are provided.
- Patient preparation for epicardial ablation requires careful attention to clinical indicators that may signal an increased risk of adverse outcomes or complications, such as a history of prior cardiac surgery, obesity, pectus excavatum, or a high PAINESD score.[176]

Required technical knowledge

Epicardial ablation for VA requires a knowledge base underpinned by training that has been formally documented. This expertise encompasses a wide range of areas, including:

- Identifying candidates for the epicardial VA ablation procedure.
- Understanding the relevant anatomy is crucial for navigating the complexities of epicardial access and ensuring its effectiveness and success. One must also be familiar with the pros and cons of various technologies and methodologies employed in epicardial access and ablation.
- Awareness of the success rates associated with ablation across different patient conditions, providing realistic expectations for outcomes.
- Understanding best practices for post-operative care and patient follow-up is essential for optimizing recovery and long-term outcomes success.
- Strategies for preventing, detecting, and managing complications that may arise during or after the procedure.

Physicians should complete a structured training programme with their progress documented and validated by a senior mentor, while ongoing lifelong learning is strongly recommended. This training should provide them with the necessary skills to:

- Secure venous access, including the application of vascular ultrasound techniques.
- The use of 3D mapping systems for real-time guidance during the procedure.
- Accurately identify and ablate the areas of interest utilizing electrophysiological tracings and pacing manoeuvres.
- Understand the biophysics underlying various energy sources and make informed decisions about energy settings and applications.
- Effective haemostasis for various vascular accesses post-procedure.
- Promptly identify and manage a serious complication.
- Delineate coronary anatomy and phrenic nerve for epicardial ablation.

Completion of training

Determining the exact number of procedures required to achieve proficiency in epicardial ablation for VAs is difficult due to clinicians' varying learning speeds. However, it is advised that all trainees undergo a thorough training programme, after which their mentors should confirm their readiness to perform epicardial VA ablations independently.

The writing group members reached a consensus that the following number of procedures is the minimum that may provide sufficient experience for performing epicardial access, mapping, and ablation:

- Twenty-five cases involving the mapping and ablation of endocardial ventricular tachycardia circuits to develop a deep understanding of ventricular arrhythmia mechanisms and the nuances of intramural and epicardial substrate identification.
- Ten epicardial ablation procedures specifically targeting VAs to ensure exposure to a variety of scenarios and complexities inherent in ventricular ablation.

Institutional requirements

Healthcare institutions must meet specific staffing, equipment, and facilities requirements to effectively deliver epicardial VA ablation procedures.

Staffing requirements

- The availability of an on-site cardiothoracic surgeon is advocated as epicardial ablation and mapping carry the risk of ventricular laceration or perforation that can result in life-threatening tamponade and haemodynamic collapse, which usually requires emergent surgical intervention via sternotomy.[7,8,164,165]
- Availability of anaesthesiologists, cardiac interventionalists, and cardiac technicians for the application of haemodynamic support systems.

Equipment and facility requirements

- Fluoroscopic X-ray imaging for real-time visualization during the procedure.
- Advanced 3D mapping technologies to facilitate precise navigation and targeting of the arrhythmic substrate, which is critical for the success of epicardial ablation
- Equipment for pericardial drainage and reversal of anticoagulation
- Transthoracic (and, if available, intracardiac) echocardiography.
- Round-the-clock availability of imaging modalities such as abdominal ultrasound and/or CT.
- Haemodynamic support systems.

Section 11: Future directions

The future of epicardial access in EP is promising, driven by ongoing advancements in novel techniques and device technology. These advancements allow clinical electrophysiologists to enhance procedural success, reduce complications, improve patient outcomes, and make epicardial VA ablation more accessible. Continued research and collaboration within the field will be essential for realizing the full potential of epicardial access in EP and addressing patients' evolving needs.

Areas for future investigation

- RF with low ionic irrigation to assess safety, efficacy, and optimal RF parameters
- Optimal RF power and duration for creating lesions
- Methods to increase lesion depth and penetration
- Methods for assessing the characteristics, including size and depth of created lesions
- Identification and integration of epicardial fat in 3D mapping
- Improved methods for defining proximity to coronary arteries and protecting coronary arteries
- Improved methods for protecting the phrenic nerve
- New energy sources: cryoablation (including ultra-low temperature), pulsed-field ablation, and high-intensity focused ultrasound
- Optimal pre- and periprocedural anticoagulation management
- Role of on-site cardiac surgery in epicardial procedures with the advancement of safer access and ablation techniques.

Supplementary material

Supplementary material is available at *Europace* online.

Acknowledgements

The authors thank the EHRA Scientific Document Committee: Prof Katja Zeppenfeld, Prof. Jens Cosedis Nielsen, Prof. Luigi Di Biase, Prof. Isabel Deisenhofer, Prof. Kristina Hermann Haugaa, Dr Daniel Keene, Prof. Christian Meyer, Prof. Petr Peichl, Prof. Silvia Priori, Dr Alireza Sepehri Shamloo, Prof. Markus Stühlinger, Prof. Jacob Tfelt Hansen, and Prof. Arthur Wilde.

Conflict of interest: All members provided disclosure statements to assess potential conflicts of interest. Details are available in the Supplementary material.

References

1. Romero J, Cerrud-Rodriguez RC, Di Biase L, Diaz JC, Alviz I, Grupposo V *et al.* Combined endocardial–epicardial versus endocardial catheter ablation alone for ventricular tachycardia in structural heart disease: a systematic review and meta-analysis. *JACC Clin Electrophysiol* 2019;**5**:13–24.
2. Romero J, Patel K, Briceno D, Alviz I, Gabr M, Diaz JC *et al.* Endo-epicardial ablation vs endocardial ablation for the management of ventricular tachycardia in arrhythmogenic right ventricular cardiomyopathy: a systematic review and meta-analysis. *J Cardiovasc Electrophysiol* 2020;**31**:2022–31.

3. Natale A, Zeppenfeld K, Della Bella P, Liu X, Sabbag A, Santangeli P *et al.* Twenty-five years of catheter ablation of ventricular tachycardia: a look back and a look forward. *Europace* 2023;**25**:euad225.
4. Shirai Y, Liang JJ, Santangeli P, Arkles JS, Schaller RD, Supple GE *et al.* Comparison of the ventricular tachycardia circuit between patients with ischemic and nonischemic cardiomyopathies: detailed characterization by entrainment. *Circ Arrhythm Electrophysiol* 2019;**12**:e007249.
5. Aryana A, Tung R, d'Avila A. Percutaneous epicardial approach to catheter ablation of cardiac arrhythmias. *JACC Clin Electrophysiol* 2020;**6**:1–20.
6. Berruezo A, Penela D, Jáuregui B, de Asmundis C, Peretto G, Marrouche N *et al.* Twenty-five years of research in cardiac imaging in electrophysiology procedures for atrial and ventricular arrhythmias. *Europace* 2023;**25**:euad183.
7. Cronin EM, Bogun FM, Maury P, Peichl P, Chen M, Namboodiri N *et al.* 2019 HRS/EHRA/APHRS/LAHRS expert consensus statement on catheter ablation of ventricular arrhythmias. *Europace* 2019;**21**:1143–4.
8. Zeppenfeld K, Tfelt-Hansen J, de Riva M, Winkel BG, Behr ER, Blom NA *et al.* 2022 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death. *Eur Heart J* 2022;**43**:3997–4126.
9. Al-Khatib SM, Stevenson WG, Ackerman MJ, Bryant WJ, Callans DJ, Curtis AB *et al.* 2017 AHA/ACC/HRS guideline for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. *Heart Rhythm* 2018;**15**:e73–189.
10. Könemann H, Dagres N, Merino JL, Sticherling C, Zeppenfeld K, Tfelt-Hansen J *et al.* Spotlight on the 2022 ESC guideline management of ventricular arrhythmias and prevention of sudden cardiac death: 10 novel key aspects. *Europace* 2023;**25**:euad091.
11. Sosa E, Scanavacca M, d'Avila A, Pilleggi F. A new technique to perform epicardial mapping in the electrophysiology laboratory. *J Cardiovasc Electrophysiol* 1996;**7**:531–6.
12. Svenson RH, Littmann L, Gallagher JJ, Selle JG, Zimmern SH, Fedor JM *et al.* Termination of ventricular tachycardia with epicardial laser photocoagulation: a clinical comparison with patients undergoing successful endocardial photocoagulation alone. *J Am Coll Cardiol* 1990;**15**:163–70.
13. D'Avila A, Scanavacca M, Sosa E, Ruskin JN, Reddy VY. Pericardial anatomy for the interventional electrophysiologist. *J Cardiovasc Electrophysiol* 2003;**14**:422–30.
14. Mori S, Bradfield JS, Peacock WJ, Anderson RH, Shivkumar K. Living anatomy of the pericardial space: a guide for imaging and interventions. *JACC Clin Electrophysiol* 2021;**7**:1628–44.
15. Choe YH, Im JG, Park JH, Han MC, Kim CW. The anatomy of the pericardial space: a study in cadavers and patients. *Am J Roentgenol* 1987;**149**:693–7.
16. Sharma R, Kouranos V, Cooper LT, Metra M, Ristic A, Heidecker B *et al.* Management of cardiac sarcoidosis. *Eur Heart J* 2024;**45**:2697–726.
17. Compagnucci P, Dello Russo A, Gasperetti A, Schiavone M, Sehrawat O, Hasegawa K *et al.* Substrate characterization and outcomes of ventricular tachycardia ablation in amyloid cardiomyopathy: a multicenter study. *Circ Arrhythm Electrophysiol* 2024;**17**:e012788.
18. Betensky BP, Kapa S, Desjardins B, Garcia FC, Callans DJ, Dixit S *et al.* Characterization of trans-septal activation during septal pacing: criteria for identification of intramural ventricular tachycardia substrate in nonischemic cardiomyopathy. *Circ Arrhythm Electrophysiol* 2013;**6**:1123–30.
19. Martinek M, Stevenson WG, Inada K, Tokuda M, Tedrow UB. QRS characteristics fail to reliably identify ventricular tachycardias that require epicardial ablation in ischemic heart disease. *J Cardiovasc Electrophysiol* 2012;**23**:188–93.
20. Yamada T, McElderry HT, Doppalapudi H, Okada T, Murakami Y, Yoshida Y *et al.* Idiopathic ventricular arrhythmias originating from the left ventricular summit: anatomic concepts relevant to ablation. *Circ Arrhythm Electrophysiol* 2010;**3**:616–23.
21. Daniels DV, Lu YY, Morton JB, Santucci PA, Akar JG, Green A *et al.* Idiopathic epicardial left ventricular tachycardia originating remote from the sinus of Valsalva: electrophysiological characteristics, catheter ablation, and identification from the 12-lead electrocardiogram. *Circulation* 2006;**113**:1659–66.
22. Santangeli P, Marchlinski FE, Zado ES, Benhayon D, Hutchinson MD, Lin D *et al.* Percutaneous epicardial ablation of ventricular arrhythmias arising from the left ventricular summit: outcomes and electrocardiogram correlates of success. *Circ Arrhythm Electrophysiol* 2015;**8**:337–43.
23. Hayashi T, Santangeli P, Pathak RK, Muser D, Liang JJ, Castro SA *et al.* Outcomes of Catheter Ablation Of Idiopathic Outflow Tract Ventricular Arrhythmias With An R Wave Pattern Break In Lead V2: a distinct clinical entity. *J Cardiovasc Electrophysiol* 2017;**28**:504–14.
24. Doppalapudi H, Yamada T, Ramaswamy K, Ahn J, Kay GN. Idiopathic focal epicardial ventricular tachycardia originating from the crux of the heart. *Heart Rhythm* 2009;**6**:44–50.
25. Oloriz T, Silberbauer J, Maccabelli G, Mizuno H, Baratto F, Kirubakaran S *et al.* Catheter ablation of ventricular arrhythmia in nonischemic cardiomyopathy: anteroseptal versus inferolateral scar sub-types. *Circ Arrhythm Electrophysiol* 2014;**7**:414–23.
26. Berruezo A, Acosta J, Fernández-Armenta J, Pedrote A, Barrera A, Arana-Rueda E *et al.* Safety, long-term outcomes and predictors of recurrence after first-line combined endoepicardial ventricular tachycardia substrate ablation in arrhythmogenic cardiomyopathy. Impact of arrhythmic substrate distribution pattern. A prospective multicentre study. *Europace* 2017;**19**:607–16.
27. Vallès E, Bazan V, Marchlinski FE. ECG criteria to identify epicardial ventricular tachycardia in nonischemic cardiomyopathy. *Circ Arrhythm Electrophysiol* 2010;**3**:63–71.
28. Bazan V, Gerstenfeld EP, Garcia FC, Bala R, Rivas N, Dixit S *et al.* Site-specific twelve-lead ECG features to identify an epicardial origin for left ventricular tachycardia in the absence of myocardial infarction. *Heart Rhythm* 2007;**4**:1403–10.
29. Bazan V, Bala R, Garcia FC, Sussman JS, Gerstenfeld EP, Dixit S *et al.* Twelve-lead ECG features to identify ventricular tachycardia arising from the epicardial right ventricle. *Heart Rhythm* 2006;**3**:1132–9.
30. Berruezo A, Mont L, Nava S, Chueca E, Bartholomay E, Brugada J. Electrocardiographic recognition of the epicardial origin of ventricular tachycardias. *Circulation* 2004;**109**:1842–7.
31. d'Avila A, Aryana A, Reddy VY, Marchlinski FE. *Percutaneous Epicardial Interventions: A Guide for Cardiac Electrophysiologists*. Minneapolis, MN: Cardiotext Publishing; 2020.
32. Yokokawa M, Jung DY, Joseph KK, Hero AO 3rd, Morady F, Bogun F. Computerized analysis of the 12-lead electrocardiogram to identify epicardial ventricular tachycardia exit sites. *Heart Rhythm* 2014;**11**:1966–73.
33. Piers SR, Silva Mde R, Kapel GF, Trines SA, Schalij MJ, Zeppenfeld K. Endocardial or epicardial ventricular tachycardia in nonischemic cardiomyopathy? The role of 12-lead ECG criteria in clinical practice. *Heart Rhythm* 2014;**11**:1031–9.
34. Betensky BP, Deyell MW, Tzou WS, Zado ES, Marchlinski FE. Sinus rhythm electrocardiogram identification of basal–lateral ischemic versus nonischemic substrate in patients with ventricular tachycardia. *J Interv Card Electrophysiol* 2012;**35**:311–21; discussion 321.
35. Tzou WS, Zado ES, Lin D, Callans DJ, Dixit S, Cooper JM *et al.* Sinus rhythm ECG criteria associated with basal–lateral ventricular tachycardia substrate in patients with nonischemic cardiomyopathy. *J Cardiovasc Electrophysiol* 2011;**22**:1351–8.
36. Oloriz T, Wellens HJ, Santagostino G, Trevisi N, Silberbauer J, Peretto G *et al.* The value of the 12-lead electrocardiogram in localizing the scar in non-ischaemic cardiomyopathy. *Europace* 2016;**18**:1850–9.
37. Darma A, Bertagnolli L, Dinov B, Torri F, Dagres N, Bollmann A *et al.* A novel ECG finding in patients with epicardial infarct-associated ventricular tachycardia: a case series. *Herzschrittmacherther Elektrophysiol* 2022;**33**:217–23.
38. Chrispin J, Keramati AR, Assis FR, Misra S, Zghaib T, Berger RD *et al.* Correlation of right ventricular multielectrode endocardial unipolar mapping and epicardial scar. *Pacing Clin Electrophysiol* 2018;**41**:345–52.
39. Piers SR, van Huls van Taxis CF, Tao Q, van der Geest RJ, Askar SF, Siebelink HM *et al.* Epicardial substrate mapping for ventricular tachycardia ablation in patients with non-ischaemic cardiomyopathy: a new algorithm to differentiate between scar and viable myocardium developed by simultaneous integration of computed tomography and contrast-enhanced magnetic resonance imaging. *Eur Heart J* 2013;**34**:586–96.
40. Betensky BP, Dong W, D'Souza BA, Zado ES, Han Y, Marchlinski FE. Cardiac magnetic resonance imaging and electroanatomic voltage discordance in non-ischemic left ventricle ventricular tachycardia and premature ventricular depolarizations. *J Interv Card Electrophysiol* 2017;**49**:11–9.
41. Omara S, Glashan CA, Tofig BJ, Leenknegt L, Dierckx H, Panfilov AV *et al.* Multisize electrode field-of-view: validation by high resolution gadolinium-enhanced cardiac magnetic resonance. *JACC Clin Electrophysiol* 2024;**10**:637–50.
42. Stevenson WG, Soejima K. Recording techniques for clinical electrophysiology. *J Cardiovasc Electrophysiol* 2005;**16**:1017–22.
43. Glashan CA, Androulakis AFA, Tao Q, Glashan RN, Wisse LJ, Ebert M *et al.* Whole human heart histology to validate electroanatomical voltage mapping in patients with non-ischaemic cardiomyopathy and ventricular tachycardia. *Eur Heart J* 2018;**39**:2867–75.
44. Aldhoon B, Frankel DS, Hutchinson MD, Callans DJ, Epstein AE, Dixit S *et al.* Unipolar voltage abnormality is associated with greater left ventricular dysfunction in ischemic cardiomyopathy. *J Cardiovasc Electrophysiol* 2014;**25**:293–8.
45. Perin EC, Silva GV, Sarmento-Leite R, Sousa AL, Howell M, Muthupillai R *et al.* Assessing myocardial viability and infarct transmurality with left ventricular electromechanical mapping in patients with stable coronary artery disease: validation by delayed-enhancement magnetic resonance imaging. *Circulation* 2002;**106**:957–61.
46. Zheng Y, Fernandes MR, Silva GV, Cardoso CO, Canales J, Gahramenpour A *et al.* Histopathological validation of electromechanical mapping in assessing myocardial viability in a porcine model of chronic ischemia. *Exp Clin Cardiol* 2008;**13**:198–203.
47. Servatius H, Höfeler T, Hoffmann BA, Sultan A, Lüker J, Schäffer B *et al.* Propofol sedation administered by cardiologists for patients undergoing catheter ablation for ventricular tachycardia. *Europace* 2016;**18**:1245–51.
48. Miller MA, Dukkipati SR, Chinitz JS, Koruth JS, Mittnacht AJ, Napolitano C *et al.* Percutaneous hemodynamic support with Impella 2.5 during scar-related ventricular tachycardia ablation (PERMIT 1). *Circ Arrhythm Electrophysiol* 2013;**6**:151–9.
49. Miller MA, Dukkipati SR, Mittnacht AJ, Chinitz JS, Belliveau L, Koruth JS *et al.* Activation and entrainment mapping of hemodynamically unstable ventricular tachycardia using a percutaneous left ventricular assist device. *J Am Coll Cardiol* 2011;**58**:1363–71.

50. Nakamura T, Davogustto GE, Schaeffer B, Tanigawa S, Muthalaly RG, Kanagasundram A *et al.* Complications and anticoagulation strategies for percutaneous epicardial ablation procedures. *Circ Arrhythm Electrophysiol* 2018;**11**:e006714.
51. Alviz I, Tedrow U, Hincapie D, Miranda-Arboleda AF, Matos CD, Hoyos C *et al.* Efficacy and safety of epicardial access on uninterrupted anticoagulation using the SAFER epicardial approach. *JACC Clin Electrophysiol* 2024;**10**:1750–3.
52. Sawhney V, Breitenstein A, Ullah W, Finlay M, Sporton S, Earley MJ *et al.* Epicardial catheter ablation for ventricular tachycardia on uninterrupted warfarin: a safe approach for those with a strong indication for peri-procedural anticoagulation? *Int J Cardiol* 2016;**222**:57–61.
53. Page SP, Duncan ER, Thomas G, Ginks MR, Earley MJ, Sporton SC *et al.* Epicardial catheter ablation for ventricular tachycardia in heparinized patients. *Europace* 2013;**15**:284–9.
54. Bonnin T, Roumegou P, Sridi S, Mahida S, Bustin A, Duchateau J *et al.* Prevalence and risk factors of cardiac thrombus prior to ventricular tachycardia catheter ablation in structural heart disease. *Europace* 2023;**25**:487–95.
55. Deneke T, Kutyifa V, Hindricks G, Sommer P, Zeppenfeld K, Carbucicchio C *et al.* Pre- and post-procedural cardiac imaging (computed tomography and magnetic resonance imaging) in electrophysiology: a clinical consensus statement of the European Heart Rhythm Association and European Association of Cardiovascular Imaging of the European Society of Cardiology. *Europace* 2024;**26**:euae108.
56. Mandel JE, Hutchinson MD, Marchlinski FE. Remifentanil-midazolam sedation provides hemodynamic stability and comfort during epicardial ablation of ventricular tachycardia. *J Cardiovasc Electrophysiol* 2011;**22**:464–6.
57. Gunda S, Reddy M, Pillarisetti J, Atoui M, Badhwar N, Swarup V *et al.* Differences in complication rates between large bore needle and a long micropuncture needle during epicardial access: time to change clinical practice? *Circ Arrhythm Electrophysiol* 2015;**8**:890–5.
58. Romero JE, Diaz JC, Zei PC, Steiger NA, Koplan BA, Matos CD *et al.* Sustained apnea for epicardial access with right ventriculography: the SAFER epicardial approach. *JACC Clin Electrophysiol* 2023;**9**:1487–99.
59. Mathew S, Feickert S, Fink T, Rillig A, Reissmann B, Rottner L *et al.* Epicardial access for VT ablation: analysis of two different puncture techniques, incidence of adhesions and complication management. *Clin Res Cardiol* 2021;**110**:810–21.
60. Tonko JB, Lambiase PD. Current and novel percutaneous epicardial access techniques for electrophysiological interventions: a comparison of procedural success and safety. *J Cardiovasc Electrophysiol* 2023;**34**:2330–41.
61. Silberbauer J, Gomes J, O'Nunain S, Kirubakaran S, Hildick-Smith D, McCready J. Coronary vein exit and carbon dioxide insufflation to facilitate subxiphoid epicardial access for ventricular mapping and ablation: first experience. *JACC Clin Electrophysiol* 2017;**3**:514–21.
62. Gurin MI, Supple GE, Hyman MC, Callans DJ, Marchlinski FE, Markman TM. Simplified approach to CO(2) insufflation for epicardial access using distal anterior interventricular vein exit without venography. *Heart Rhythm* 2024;**21**:1042–4.
63. Julia J, Bokhari F, Uuetoa H, Derejko P, Traykov VB, Gwizdala A *et al.* A new era in epicardial access for the ablation of ventricular arrhythmias: the Epi-Co(2) registry. *JACC Clin Electrophysiol* 2021;**7**:85–96.
64. Zucchelli G, Parollo M, Di Cori A, Mazzocchetti L, Segreti L, Grifoni G *et al.* Feasibility of carbon dioxide insufflation and impact on epicardial approach utilization for ventricular tachycardia ablation in a midvolume referral center. *Heart Rhythm* 2024;**21**:1032–9.
65. Romero JE, Miranda-Arboleda AF, Hoyos C, Matos CD, Batnyam U, Sauer WH *et al.* Hybrid ventricular tachycardia ablation combining video-assisted thoracoscopy with subxiphoid epicardial access. *JACC Clin Electrophysiol* 2024;**10**:1773–80.
66. Opfermann JD, Contento JM, Mass PN, Krieger A, Berul CI, Kumthekar RN. A novel videoscope and tool kit for percutaneous pericardial access under direct visualization. *Biomed Eng Online* 2023;**22**:19.
67. Liu Y, Zhang Y, Jiang W, Qin M, Liu X. A novel puncture kit for facilitating epicardial access. *Heart Rhythm* 2024;**21**:2607–9.
68. Derejko P, Dzwonkowska D, Wróbel K, Kuśnierz J, Bardyszewski A, Menshes Z *et al.* Safety and efficacy of a novel blunt-tip concealed-needle epicardial access device: first-in-human feasibility study. *JACC Clin Electrophysiol* 2022;**8**:908–12.
69. Di Biase L, Burkhardt JD, Reddy V, Romero J, Neuzil P, Petru J *et al.* Initial international multicenter human experience with a novel epicardial access needle embedded with a real-time pressure/frequency monitoring to facilitate epicardial access: feasibility and safety. *Heart Rhythm* 2017;**14**:981–8.
70. Yu R, Liu N, You B, Wang H, Ruan Y, Wen S *et al.* Use of three-dimensional electroanatomic mapping for epicardial access: needle tracking, electrographic characteristics, and clinical application. *Europace* 2024;**26**:euae089.
71. Meglan DA, Cohen RJ, Riviere CN. Techniques for epicardial mapping and ablation with a miniature robotic walker. *Robot Surg* 2018;**4**:25–31.
72. Falasconi G, Penela D, Soto-Iglesias D, Francia P, Saglietto A, Alderete J *et al.* Cardiac magnetic resonance-aided epicardial ventricular tachycardia ablation in post-myocarditis patient. *J Interv Card Electrophysiol* 2024;**67**:249–51.
73. Xie S, Desjardins B, Kubala M, Liang J, Yang J, van der Geest RJ *et al.* Association of regional epicardial right ventricular electrogram voltage amplitude and late gadolinium enhancement distribution on cardiac magnetic resonance in patients with arrhythmogenic right ventricular cardiomyopathy: implications for ventricular tachycardia ablation. *Heart Rhythm* 2018;**15**:987–93.
74. Andreu D, Ortiz-Pérez JT, Boussy T, Fernández-Armenta J, de Caralt TM, Perea RJ *et al.* Usefulness of contrast-enhanced cardiac magnetic resonance in identifying the ventricular arrhythmia substrate and the approach needed for ablation. *Eur Heart J* 2014;**35**:1316–26.
75. Soto-Iglesias D, Penela D, Jáuregui B, Acosta J, Fernández-Armenta J, Linhart M *et al.* Cardiac magnetic resonance-guided ventricular tachycardia substrate ablation. *JACC Clin Electrophysiol* 2020;**6**:436–47.
76. Nishimura T, Patel HN, Wang S, Upadhyay GA, Smith HL, Ozcan C *et al.* Prognostic value of cardiac magnetic resonance septal late gadolinium enhancement patterns for periaortic ventricular tachycardia ablation: heterogeneity of the anteroseptal substrate in nonischemic cardiomyopathy. *Heart Rhythm* 2021;**18**:579–88.
77. Vázquez-Calvo S, Casanovas JM, Garre P, Ferró E, Sánchez-Somonte P, Quinto L *et al.* Evolution of deceleration zones during ventricular tachycardia ablation and relation with cardiac magnetic resonance. *JACC Clin Electrophysiol* 2023;**9**:779–89.
78. Siontis KC, Kim HM, Sharaf Dabbagh G, Latchamsetty R, Stojanovska J, Jongnarangsin K *et al.* Association of preprocedural cardiac magnetic resonance imaging with outcomes of ventricular tachycardia ablation in patients with idiopathic dilated cardiomyopathy. *Heart Rhythm* 2017;**14**:1487–93.
79. Torri F, Czimbalmos C, Bertagnolli L, Oebel S, Bollmann A, Paetsch I *et al.* Agreement between gadolinium-enhanced cardiac magnetic resonance and electro-anatomical maps in patients with non-ischaemic dilated cardiomyopathy and ventricular arrhythmias. *Europace* 2019;**21**:1392–9.
80. Kuo L, Liang JJ, Han Y, Frankel DS, Santangeli P, Callans DJ *et al.* Association of septal late gadolinium enhancement on cardiac magnetic resonance with ventricular tachycardia ablation targets in nonischemic cardiomyopathy. *J Cardiovasc Electrophysiol* 2020;**31**:3262–76.
81. Acosta J, Penela D, Andreu D, Cabrera M, Carlosena A, Vassanelli F *et al.* Multielectrode vs. point-by-point mapping for ventricular tachycardia substrate ablation: a randomized study. *Europace* 2018;**20**:512–9.
82. Blomström Lundqvist C, Auricchio A, Brugada J, Boriani G, Bremerich J, Cabrera JA *et al.* The use of imaging for electrophysiological and devices procedures: a report from the first European Heart Rhythm Association Policy Conference, jointly organized with the European Association of Cardiovascular Imaging (EACVI), the Council of Cardiovascular Imaging and the European Society of Cardiac Radiology. *Europace* 2013;**15**:927–36.
83. Takigawa M, Duchateau J, Sacher F, Martin R, Vlachos K, Kitamura T *et al.* Are wall thickness channels defined by computed tomography predictive of isthmuses of post-infarction ventricular tachycardia? *Heart Rhythm* 2019;**16**:1661–8.
84. John LA, Tomashitis B, Gowani Z, Levin D, Vo C, John I *et al.* inHEART models software—novel 3D cardiac modeling solution. *Expert Rev Med Devices* 2023;**20**:797–803.
85. Faga V, Dallaglio PD, Claver E, Rodriguez-García J, San Antonio R, Rodriguez M *et al.* Variations in threshold values for border zone and dense scar produce significant changes in scar parameters obtained by ADAS-3D. *Heart Rhythm* 2024;**22**:106–17.
86. Ramos-Prada A, Redondo-Rodríguez A, Roca-Luque I, Porta-Sánchez A, Ter Bekke RMA, Quintanilla JG *et al.* Novel systematic processing of cardiac magnetic resonance imaging identifies target regions associated with infarct-related ventricular tachycardia. *Europace* 2024;**26**:euae244.
87. Andreu D, Fernández-Armenta J, Acosta J, Penela D, Jáuregui B, Soto-Iglesias D *et al.* A QRS axis-based algorithm to identify the origin of scar-related ventricular tachycardia in the 17-segment American Heart Association model. *Heart Rhythm* 2018;**15**:1491–7.
88. Bala R, Ren JF, Hutchinson MD, Desjardins B, Tschabrunn C, Gerstenfeld EP *et al.* Assessing epicardial substrate using intracardiac echocardiography during VT ablation. *Circ Arrhythm Electrophysiol* 2011;**4**:667–73.
89. Chery G, Khoshknab M, Nazarian S. Imaging to facilitate ventricular tachycardia ablation: intracardiac echocardiography, computed tomography, magnetic resonance, and positron emission tomography. *JACC Clin Electrophysiol* 2024;**10**:2277–92.
90. Acosta J, Fernández-Armenta J, Penela D, Andreu D, Borras R, Vassanelli F *et al.* Infarct transmurality as a criterion for first-line endo-epicardial substrate-guided ventricular tachycardia ablation in ischemic cardiomyopathy. *Heart Rhythm* 2016;**13**:85–95.
91. Hilbert S, Weber A, Nehrke K, Börnert P, Schnackenburg B, Oebel S *et al.* Artefact-free late gadolinium enhancement imaging in patients with implanted cardiac devices using a modified broadband sequence: current strategies and results from a real-world patient cohort. *Europace* 2018;**20**:801–7.
92. Seewöster T, Löbe S, Hilbert S, Bollmann A, Sommer P, Lindemann F *et al.* Cardiovascular magnetic resonance imaging in patients with cardiac implantable electronic devices: best practice and real-world experience. *Europace* 2019;**21**:1220–8.
93. Polin GM, Haqqani H, Tzou W, Hutchinson MD, Garcia FC, Callans DJ *et al.* Endocardial unipolar voltage mapping to identify epicardial substrate in arrhythmogenic right ventricular cardiomyopathy/dysplasia. *Heart Rhythm* 2011;**8**:76–83.

94. Venlet J, Piers SRD, Kapel GFL, de Riva M, Pauli PFG, van der Geest RJ *et al.* Unipolar endocardial voltage mapping in the right ventricle: optimal cutoff values correcting for computed tomography-derived epicardial fat thickness and their clinical value for substrate delineation. *Circ Arrhythm Electrophysiol* 2017;**10**:e005175.
95. Venlet J, Piers SR, Hoogendoorn J, Androulakis AFA, de Riva M, van der Geest RJ *et al.* The transmural activation interval: a new mapping tool to identify ventricular tachycardia substrates in right ventricular cardiomyopathy. *Europace* 2023;**25**:478–86.
96. Glashan CA, Tofig BJ, Beukers H, Tao Q, Blom SA, Villadsen PR *et al.* Multielectrode unipolar voltage mapping and electrogram morphology to identify post-infarct scar geometry: validation by histology. *JACC Clin Electrophysiol* 2022;**8**:437–49.
97. Tan JL, Guandalini GS, Hyman MC, Arkles J, Santangeli P, Schaller RD *et al.* Substrate and arrhythmia characterization using the multi-electrode Optrell mapping catheter for ventricular arrhythmia ablation-a single-center experience. *J Interv Card Electrophysiol* 2024;**67**:559–69.
98. Anter E, Brem O, Greenbaum L, Bubar ZP, Younis A, Yavin H *et al.* Multipolar electrograms: a new configuration that increases the measurement accuracy of intracardiac signals. *JACC Clin Electrophysiol* 2024;**10**:1521–33.
99. Ehdaie A, Ramireddy A, Joshi S, Reyes KR, Aliyari A, Cuk N *et al.* Spatial analysis and characteristics of persistent late potentials after ablation of scar-related VT substrate: implications for late potential elimination as a procedural endpoint with high-resolution mapping. *Heart Rhythm* 2024;**22**:675–84.
100. Tonko JB, Ruipérez-Campillo S, Cabero-Vidal G, Cabrera-Borrego E, Roney C, Jiménez-Jáimez J *et al.* Vector field heterogeneity as a novel omnipolar mapping metric for functional substrate characterization in scar-related ventricular tachycardias. *Heart Rhythm* 2024. doi:https://doi.org/10.1016/j.hrthm.2024.10.066
101. Tonko JB, Lozano C, Moreno J, Chow A, Dhinoja M, Lambiase PD. Near-field detection and peak frequency metric for substrate and activation mapping of ventricular tachycardias in two- and three-dimensional circuits. *Europace* 2024;**26**:euae154.
102. Piers SR, Leong DP, van Huls van Taxis CF, Tayyebi M, Trines SA, Pijnappels DA *et al.* Outcome of ventricular tachycardia ablation in patients with nonischemic cardiomyopathy: the impact of noninducibility. *Circ Arrhythm Electrophysiol* 2013;**6**:513–21.
103. Ciaccio EJ, Coromilas J, Costeas CA, Wit AL. Sinus rhythm electrogram shape measurements are predictive of the origins and characteristics of multiple reentrant ventricular tachycardia morphologies. *J Cardiovasc Electrophysiol* 2004;**15**:1293–301.
104. Ciaccio EJ, Coromilas J, Ashikaga H, Cervantes DO, Wit AL, Peters NS *et al.* Model of unidirectional block formation leading to reentrant ventricular tachycardia in the infarct border zone of postinfarction canine hearts. *Comput Biol Med* 2015;**62**:254–63.
105. Jackson N, Gizurarson S, Viswanathan K, King B, Massé S, Kusha M *et al.* Decrement evoked potential mapping: basis of a mechanistic strategy for ventricular tachycardia ablation. *Circ Arrhythm Electrophysiol* 2015;**8**:1433–42.
106. de Riva M, Naruse Y, Ebert M, Androulakis AFA, Tao Q, Watanabe M *et al.* Targeting the hidden substrate unmasked by right ventricular extrastimulation improves ventricular tachycardia ablation outcome after myocardial infarction. *JACC Clin Electrophysiol* 2018;**4**:316–27.
107. Aziz Z, Shatz D, Raiman M, Upadhyay GA, Beaser AD, Besser SA *et al.* Targeted ablation of ventricular tachycardia guided by wavefront discontinuities during sinus rhythm: a new functional substrate mapping strategy. *Circulation* 2019;**140**:1383–97.
108. Garcia FC, Bazan V, Zado ES, Ren JF, Marchlinski FE. Epicardial substrate and outcome with epicardial ablation of ventricular tachycardia in arrhythmogenic right ventricular cardiomyopathy/dysplasia. *Circulation* 2009;**120**:366–75.
109. Chaumont C, Tschabrunn CM, Oraii A, Zado ES, Yogasundaram H, Petzl A *et al.* Long-term freedom from ventricular arrhythmias in ARVC with endocardial only ablation: predictors of success. *JACC Clin Electrophysiol* 2024;**10**:1551–61.
110. Shen L, Liu S, Zhang Z, Xiong Y, Lai Z, Hu F *et al.* Catheter ablation of ventricular tachycardia in patients with arrhythmogenic right ventricular cardiomyopathy and biventricular involvement. *Europace* 2024;**26**:euae059.
111. Tung R, Xue Y, Chen M, Jiang C, Shatz DY, Besser SA *et al.* First-line catheter ablation of monomorphic ventricular tachycardia in cardiomyopathy concurrent with defibrillator implantation: the PAUSE-SCD randomized trial. *Circulation* 2022;**145**:1839–49.
112. Dukkipati SR, d'Avila A, Soejima K, Bala R, Inada K, Singh S *et al.* Long-term outcomes of combined epicardial and endocardial ablation of monomorphic ventricular tachycardia related to hypertrophic cardiomyopathy. *Circ Arrhythm Electrophysiol* 2011;**4**:185–94.
113. Hayashi T, Liang JJ, Muser D, Shirai Y, Enriquez A, Garcia FC *et al.* Epicardial ventricular tachycardia in ischemic cardiomyopathy: prevalence, electrophysiological characteristics, and long-term ablation outcomes. *J Cardiovasc Electrophysiol* 2018;**29**:1530–9.
114. Soto-Iglesias D, Acosta J, Penela D, Fernández-Armenta J, Cabrera M, Martínez M *et al.* Image-based criteria to identify the presence of epicardial arrhythmogenic substrate in patients with transmural myocardial infarction. *Heart Rhythm* 2018;**15**:814–21.
115. Pappone C, Mecarocci V, Manguso F, Ciconte G, Vicedomini G, Sturla F *et al.* New electromechanical substrate abnormalities in high-risk patients with Brugada syndrome. *Heart Rhythm* 2020;**17**:637–45.
116. Nademanee K, Veerakul G, Chandanamattha P, Chaothawee L, Ariyachaipanich A, Jirasirirojanakorn K *et al.* Prevention of ventricular fibrillation episodes in Brugada syndrome by catheter ablation over the anterior right ventricular outflow tract epicardium. *Circulation* 2011;**123**:1270–9.
117. Nademanee K, Chung FP, Sacher F, Nogami A, Nakagawa H, Jiang C *et al.* Long-term outcomes of Brugada substrate ablation: a report from BRAVO (Brugada ablation of VF substrate ongoing multicenter registry). *Circulation* 2023;**147**:1568–78.
118. Li L, Ding L, Zhou L, Wu L, Zheng L, Zhang Z *et al.* Outcomes of catheter ablation in high-risk patients with Brugada syndrome refusing an implantable cardioverter defibrillator implantation. *Europace* 2023;**26**:euad318.
119. Santinelli V, Ciconte G, Manguso F, Anastasia L, Micaglio E, Calovic Z *et al.* High-risk Brugada syndrome: factors associated with arrhythmia recurrence and benefits of epicardial ablation in addition to implantable cardioverter defibrillator implantation. *Europace* 2023;**26**:euae019.
120. Henz BD, do Nascimento TA, Dietrich CO, Dalegrave C, Hernandes V, Mesas CE *et al.* Simultaneous epicardial and endocardial substrate mapping and radiofrequency catheter ablation as first-line treatment for ventricular tachycardia and frequent ICD shocks in chronic chagasic cardiomyopathy. *J Interv Card Electrophysiol* 2009;**26**:195–205.
121. Okubo K, Gigli L, Trevisi N, Foppoli L, Radinovic A, Bisceglia C *et al.* Long-term outcome after ventricular tachycardia ablation in nonischemic cardiomyopathy: late potential abolition and VT noninducibility. *Circ Arrhythm Electrophysiol* 2020;**13**:e008307.
122. Silberbauer J, Oloriz T, Maccabelli G, Tsiachris D, Baratto F, Vergara P *et al.* Noninducibility and late potential abolition: a novel combined prognostic procedural end point for catheter ablation of postinfarction ventricular tachycardia. *Circ Arrhythm Electrophysiol* 2014;**7**:424–35.
123. Tsiachris D, Silberbauer J, Maccabelli G, Oloriz T, Baratto F, Mizuno H *et al.* Electroanatomical voltage and morphology characteristics in postinfarction patients undergoing ventricular tachycardia ablation: pragmatic approach favoring late potentials abolition. *Circ Arrhythm Electrophysiol* 2015;**8**:863–73.
124. Hu J, Zeng S, Zhou Q, Zhu W, Xu Z, Yu J *et al.* Can ventricular tachycardia non-inducibility after ablation predict reduced ventricular tachycardia recurrence and mortality in patients with non-ischemic cardiomyopathy? A meta-analysis of twenty-four observational studies. *Int J Cardiol* 2016;**222**:689–95.
125. Jauregui-Abularach ME, Campos B, Betensky BP, Michele J, Gerstenfeld EP. Comparison of epicardial cryoablation and irrigated radiofrequency ablation in a Swine infarct model. *J Cardiovasc Electrophysiol* 2012;**23**:1016–23.
126. d'Avila A, Houghtaling C, Gutierrez P, Vragovic O, Ruskin JN, Josephson ME *et al.* Catheter ablation of ventricular epicardial tissue: a comparison of standard and cooled-tip radiofrequency energy. *Circulation* 2004;**109**:2363–9.
127. Tanner H, Hindricks G, Volkmer M, Furniss S, Kühlkamp V, Lacroix D *et al.* Catheter ablation of recurrent scar-related ventricular tachycardia using electroanatomical mapping and irrigated ablation technology: results of the prospective multicenter Euro-VT-study. *J Cardiovasc Electrophysiol* 2010;**21**:47–53.
128. Stevenson WG, Wilber DJ, Natale A, Jackman WM, Marchlinski FE, Talbert T *et al.* Irrigated radiofrequency catheter ablation guided by electroanatomic mapping for recurrent ventricular tachycardia after myocardial infarction: the multicenter thermocool ventricular tachycardia ablation trial. *Circulation* 2008;**118**:2773–82.
129. Marchlinski FE, Haffajee CI, Beshai JF, Dickfeld TL, Gonzalez MD, Hsia HH *et al.* Long-term success of irrigated radiofrequency catheter ablation of sustained ventricular tachycardia: post-approval THERMOCOOL VT trial. *J Am Coll Cardiol* 2016;**67**:674–83.
130. Baldinger SH, Kumar S, Barbhaiya CR, Mahida S, Epstein LM, Michaud GF *et al.* Epicardial radiofrequency ablation failure during ablation procedures for ventricular arrhythmias: reasons and implications for outcomes. *Circ Arrhythm Electrophysiol* 2015;**8**:1422–32.
131. Hasegawa K, Yoneda ZT, Powers EM, Tokutake K, Kurata M, Richardson TD *et al.* Safety of ventricular arrhythmia radiofrequency ablation with half-normal saline irrigation. *Europace* 2024;**26**:euae018.
132. Kobza R, Hilfiker G, Rissotto S, Mahida S, Grebmer C, Duru F *et al.* Performance and safety of temperature- and flow-controlled radiofrequency ablation for ventricular arrhythmia. *Europace* 2023;**26**:euad372.
133. Debenham R, Tzou WS. Epicardial ablation biophysics and novel radiofrequency energy delivery techniques. *Card Electrophysiol Clin* 2020;**12**:401–8.
134. Haines DE. Letter by Haines regarding article, "Direct measurement of the lethal isotherm for radiofrequency ablation of myocardial tissue". *Circ Arrhythm Electrophysiol* 2011;**4**:e67; author reply e68.
135. Wood MA, Goldberg SM, Parvez B, Pathak V, Holland K, Ellenbogen AL *et al.* Effect of electrode orientation on lesion sizes produced by irrigated radiofrequency ablation catheters. *J Cardiovasc Electrophysiol* 2009;**20**:1262–8.
136. Zipse MM, Edward JA, Zheng L, Tzou WS, Borne RT, Sauer WH *et al.* Impact of epicardial adipose tissue and catheter ablation strategy on biophysical parameters and ablation lesion characteristics. *J Cardiovasc Electrophysiol* 2020;**31**:1114–24.
137. Wong MC, Edwards G, Spence SJ, Kalman JM, Kumar S, Joseph SA *et al.* Characterization of catheter-tissue contact force during epicardial radiofrequency ablation in an ovine model. *Circ Arrhythm Electrophysiol* 2013;**6**:1222–8.

138. Aryana A, O'Neill PG, Pujara DK, Singh SK, Bowers MR, Allen SL *et al.* Impact of irrigation flow rate and intrapericardial fluid on cooled-tip epicardial radiofrequency ablation. *Heart Rhythm* 2016;**13**:1602–11.
139. Fenelon G, Pereira KP, de Paola AA. Epicardial radiofrequency ablation of ventricular myocardium: factors affecting lesion formation and damage to adjacent structures. *J Interv Card Electrophysiol* 2006;**15**:57–63.
140. Sacher F, Wright M, Derval N, Denis A, Ramoul K, Roten L *et al.* Endocardial versus epicardial ventricular radiofrequency ablation: utility of in vivo contact force assessment. *Circ Arrhythm Electrophysiol* 2013;**6**:144–50.
141. Powell B, Coons T, Lesiczka M, Markert C, Mehta R, Misra S. Dry suction water seal system for management of pericardial fluid during epicardial ablation. *J Cardiovasc Electrophysiol* 2023;**34**:1979–82.
142. Sapp JL, Soejima K, Cooper JM, Epstein LM, Stevenson WG. Ablation lesion size correlates with pacing threshold: a physiological basis for use of pacing to assess ablation lesions. *Pacing Clin Electrophysiol* 2004;**27**:933–7.
143. Bennett R, Campbell T, Byth K, Turnbull S, Kumar S. Catheter ablation using half-normal saline and dextrose irrigation in an ovine ventricular model. *JACC Clin Electrophysiol* 2021;**7**:1229–39.
144. Hong KN, Russo MJ, Liberman EA, Trzebucki A, Oz MC, Argenziano M *et al.* Effect of epicardial fat on ablation performance: a three-energy source comparison. *J Card Surg* 2007;**22**:521–4.
145. van Huls van Taxis CF, Wijnmaalen AP, Piers SR, van der Geest RJ, Schalij MJ, Zeppenfeld K. Real-time integration of MDCT-derived coronary anatomy and epicardial fat: impact on epicardial electroanatomic mapping and ablation for ventricular arrhythmias. *JACC Cardiovasc Imaging* 2013;**6**:42–52.
146. Desjardins B, Morady F, Bogun F. Effect of epicardial fat on electroanatomical mapping and epicardial catheter ablation. *J Am Coll Cardiol* 2010;**56**:1320–7.
147. Tung R, Nakahara S, Ramirez R, Lai C, Fishbein MC, Shivkumar K. Distinguishing epicardial fat from scar: analysis of electrograms using high-density electroanatomic mapping in a novel porcine infarct model. *Heart Rhythm* 2010;**7**:389–95.
148. Soucek F, Caluori G, Lehar F, Jez J, Pesl M, Wolf J *et al.* Bipolar ablation with contact force-sensing of swine ventricles shows improved acute lesion features compared to sequential unipolar ablation. *J Cardiovasc Electrophysiol* 2020;**31**:1128–36.
149. Derejko P, Kuśnierz J, Bardyszewski A, Dzwonkowska D, Polańska-Skrzypczyk M, Szumowski ŁJ *et al.* Bipolar endo-epicardial radiofrequency ablation of therapy-resistant ventricular arrhythmias: a brief case series. *JACC Clin Electrophysiol* 2023;**9**:733–7.
150. Bakker PF, Elbers HR, Vermeulen FE, Robles de Medina EO. Effects of cryothermia during cold cardioplegia on epicardial and intramural coronary arteries. *Ann Thorac Surg* 1993;**55**:127–30.
151. Hayase J, Fishbein G, Rerkpichaisuth V, Chung WH, Ajijola O, Shivkumar K *et al.* Linear epicardial cryoablation effects in a porcine model: lesion characteristics and vascular risk. *J Cardiovasc Electrophysiol* 2023;**34**:1878–84.
152. Kunkel M, Rothstein P, Sauer P, Zipse MM, Sandhu A, Tumolo AZ *et al.* Open surgical ablation of ventricular tachycardia: utility and feasibility of contemporary mapping and ablation tools. *Heart Rhythm O2* 2021;**2**:271–9.
153. Hashimoto K, Watanabe I, Okumura Y, Ohkubo K, Ashino S, Kofune M *et al.* Comparison of endocardial and epicardial lesion size following large-tip and extra-large-tip transcatheter cryoablation. *Circ J* 2009;**73**:1619–26.
154. Verma A, Essebag V, Neuzil P, Dyrda K, Balt J, Dinov B *et al.* Cryocure-VT: the safety and effectiveness of ultra-low-temperature cryoablation of monomorphic ventricular tachycardia in patients with ischaemic and non-ischaemic cardiomyopathies. *Europace* 2024;**26**:euae076.
155. Cox JL. Epicardial cryoablation and the heat-sink problem. *Ann Thorac Surg* 2022;**114**:1523.
156. Comas GM, Imren Y, Williams MR. An overview of energy sources in clinical use for the ablation of atrial fibrillation. *Semin Thorac Cardiovasc Surg* 2007;**19**:16–24.
157. Mulloy DP, Bhamidipati CM, Stone ML, Ailawadi G, Bergin JD, Mahapatra S *et al.* Cryoablation during left ventricular assist device implantation reduces postoperative ventricular tachyarrhythmias. *J Thorac Cardiovasc Surg* 2013;**145**:1207–13.
158. Neven K, van Driel V, van Wessel H, van Es R, Doevendans PA, Wittkampf F. Epicardial linear electroporation ablation and lesion size. *Heart Rhythm* 2014;**11**:1465–70.
159. Neven K, van Driel V, van Wessel H, van Es R, Doevendans PA, Wittkampf F. Myocardial lesion size after epicardial electroporation catheter ablation after subxiphoid puncture. *Circ Arrhythm Electrophysiol* 2014;**7**:728–33.
160. Solimene F, Compagnucci P, Tondo C, La Fazia VM, Schillaci V, Mohanty S *et al.* Direct epicardial validation of posterior wall electroporation in persistent atrial fibrillation. *JACC Clin Electrophysiol* 2024;**10**:1200–2.
161. Peichl P, Wichterle D, Schlosser F, Stojadinović P, Nejedlo V, Borišincová E *et al.* Mapping and ablation of ventricular tachycardia using dual-energy lattice-tip focal catheter: early feasibility and safety study. *Europace* 2024;**26**:euae275.
162. Engel DJ, Muratore R, Hirata K, Otsuka R, Fujikura K, Sugioka K *et al.* Myocardial lesion formation using high-intensity focused ultrasound. *J Am Soc Echocardiogr* 2006;**19**:932–7.
163. Nazer B, Salgaonkar V, Diederich CJ, Jones PD, Duggirala S, Tanaka Y *et al.* Epicardial catheter ablation using high-intensity ultrasound: validation in a swine model. *Circ Arrhythm Electrophysiol* 2015;**8**:1491–7.
164. Haines DE, Beheiry S, Akar JG, Baker JL, Beinborn D, Beshai JF *et al.* Heart Rythm Society expert consensus statement on electrophysiology laboratory standards: process, protocols, equipment, personnel, and safety. *Heart Rhythm* 2014;**11**:e9–51.
165. Trines SA, Moore P, Burri H, Gonçalves Nunes S, Massoullié G, Merino JL *et al.* 2024 updated European Heart Rhythm Association core curriculum for physicians and allied professionals: a statement of the European Heart Rhythm Association of the European Society of Cardiology: Syllabus, Objective Structured Assessment of Technical Skills, training assessment, and training centre/trainer/trainee requirements to guide the certification for cardiac implantable electronic devices and invasive cardiac electrophysiology. *Europace* 2024;**26**:euae243.
166. Yamada T, Kay GN. Recognition and prevention of complications during epicardial ablation. *Card Electrophysiol Clin* 2010;**2**:127–34.
167. Sacher F, Roberts-Thomson K, Maury P, Tedrow U, Nault I, Steven D *et al.* Epicardial ventricular tachycardia ablation: a multicenter safety study. *J Am Coll Cardiol* 2010;**55**:2366–72.
168. Fan R, Cano O, Ho SY, Bala R, Callans DJ, Dixit S *et al.* Characterization of the phrenic nerve course within the epicardial substrate of patients with nonischemic cardiomyopathy and ventricular tachycardia. *Heart Rhythm* 2009;**6**:59–64.
169. Okubo K, Trevisi N, Foppoli L, Bisceglia C, Baratto F, Gigli L *et al.* Phrenic nerve limitation during epicardial catheter ablation of ventricular tachycardia. *JACC Clin Electrophysiol* 2019;**5**:81–90.
170. Kumar S, Barbhaiya CR, Baldinger SH, Koplan BA, Maytin M, Epstein LM *et al.* Epicardial phrenic nerve displacement during catheter ablation of atrial and ventricular arrhythmias: procedural experience and outcomes. *Circ Arrhythm Electrophysiol* 2015;**8**:896–904.
171. Conti S, Bonomo V, Taormina A, Giordano U, Sgarito G. Phrenic nerve displacement by intrapericardial balloon inflation during epicardial ablation of ventricular tachycardia: four case reports. *World J Cardiol* 2020;**12**:55–66.
172. Di Biase L, Burkhardt JD, Pelargonio G, Dello Russo A, Casella M, Santarelli P *et al.* Prevention of phrenic nerve injury during epicardial ablation: comparison of methods for separating the phrenic nerve from the epicardial surface. *Heart Rhythm* 2009;**6**:957–61.
173. Yamashita S, Sacher F, Mahida S, Berte B, Lim HS, Komatsu Y *et al.* Role of high-resolution image integration to visualize left phrenic nerve and coronary arteries during epicardial ventricular tachycardia ablation. *Circ Arrhythm Electrophysiol* 2015;**8**:371–80.
174. Tarantino N, Della Rocca DG, Faggioni M, Zhang XD, Mohanty S, Anannab A *et al.* Epicardial ablation complications. *Card Electrophysiol Clin* 2020;**12**:409–18.
175. Darma A, Bertagnolli L, Weber A, Dinov B, Torri F, Lurz JA *et al.* Epicardial ablation of ventricular tachycardia in patients with structural heart disease: a single-centre experience over 12 years. *Europace* 2021;**23**:1980–8.
176. Darma A, Bertagnolli L, Dinov B, Torri F, Shamloo AS, Lurz JA *et al.* Predictors of long-term mortality after catheter ablation of ventricular tachycardia in a contemporary cohort of patients with structural heart disease. *Europace* 2020;**22**:1672–9.
177. Soejima K, Couper G, Cooper JM, Sapp JL, Epstein LM, Stevenson WG. Subxiphoid surgical approach for epicardial catheter-based mapping and ablation in patients with prior cardiac surgery or difficult pericardial access. *Circulation* 2004;**110**:1197–201.
178. Aryana A, d'Avila A. Epicardial approach for cardiac electrophysiology procedures. *J Cardiovasc Electrophysiol* 2020;**31**:345–59.
179. Chen S, Chun KRJ, Bordignon S, Tohoku S, Schmidt B. Epicardial mapping and ablation for ventricular arrhythmias in experienced center without onsite cardiac surgery. *Glob Cardiol Sci Pract* 2021;**2021**:e202103.
180. Raad M, Supple GE. Epicardial ventricular tachycardia ablation: patient selection, access, and ablation techniques and strategies to manage complications. *JACC Clin Electrophysiol* 2024;**10**:142–64.
181. Oesterle A, Singh A, Balkhy H, Husain AN, Moyer D, Tung R *et al.* Late presentation of constrictive pericarditis after limited epicardial ablation for inappropriate sinus tachycardia. *HeartRhythm Case Rep* 2016;**2**:441–5.
182. Gunda S, Reddy M, Nath J, Nagaraj H, Atoui M, Rasekh A *et al.* Impact of periprocedural colchicine on postprocedural management in patients undergoing a left atrial appendage ligation using LARIAT. *J Cardiovasc Electrophysiol* 2016;**27**:60–4.
183. Sharma SP, Turagam MK, Mohanty S, Di Biase L, Burkhardt D, Horton R *et al.* Epicardial interventions: impact of liposomal bupivacaine on postprocedural management (the EPI-LIBRE study). *Circ Arrhythm Electrophysiol* 2020;**13**:e007425.
184. Dyrda K, Piers SR, van Huls van Taxis CF, Schalij MJ, Zeppenfeld K. Influence of steroid therapy on the incidence of pericarditis and atrial fibrillation after percutaneous epicardial mapping and ablation for ventricular tachycardia. *Circ Arrhythm Electrophysiol* 2014;**7**:671–6.
185. d'Avila A, Neuzil P, Thiagalingam A, Gutierrez P, Aleong R, Ruskin JN *et al.* Experimental efficacy of pericardial instillation of anti-inflammatory agents during

percutaneous epicardial catheter ablation to prevent postprocedure pericarditis. *J Cardiovasc Electrophysiol* 2007;**18**:1178–83.
186. Cremer PC, Klein AL, Imazio M. Diagnosis, risk stratification, and treatment of pericarditis: a review. *JAMA* 2024;**332**:1090–100.
187. Killu AM, Friedman PA, Mulpuru SK, Munger TM, Packer DL, Asirvatham SJ. Atypical complications encountered with epicardial electrophysiological procedures. *Heart Rhythm* 2013;**10**:1613–21.
188. Della Bella P, Brugada J, Zeppenfeld K, Merino J, Neuzil P, Maury P *et al.* Epicardial ablation for ventricular tachycardia: a European multicenter study. *Circ Arrhythm Electrophysiol* 2011;**4**:653–9.
189. Tung R, Michowitz Y, Yu R, Mathuria N, Vaseghi M, Buch E *et al.* Epicardial ablation of ventricular tachycardia: an institutional experience of safety and efficacy. *Heart Rhythm* 2013;**10**:490–8.
190. Lin CY, Chung FP, Lin YJ, Chang SL, Lo LW, Hu YF *et al.* Safety and efficacy of epicardial ablation of ventricular tachyarrhythmias: experience from a tertiary referral center in Taiwan. *Acta Cardiol Sin* 2018;**34**:49–58.
191. Koruth JS, Aryana A, Dukkipati SR, Pak HN, Kim YH, Sosa EA *et al.* Unusual complications of percutaneous epicardial access and epicardial mapping and ablation of cardiac arrhythmias. *Circ Arrhythm Electrophysiol* 2011;**4**:882–8.
192. Liang JJ, Abou El Ela A, Bogun F. Double right ventricular puncture during percutaneous epicardial access: rare complication of epicardial ventricular tachycardia ablation. *JACC Clin Electrophysiol* 2022;**8**:707–11.
193. Rodin AE, Key JD. William Osler and Aequanimitas: an appraisal of his reactions to adversity. *J R Soc Med* 1994;**87**:758–63.
194. Khan M, Hendriks AA, Yap SC, Berger WR, de Ruiter GS, Szili-Torok T. Damage to the left internal mammary artery during anterior epicardial access for ventricular tachycardia ablation. *HeartRhythm Case Rep* 2018;**4**:534–7.
195. D'Avila A, Gutierrez P, Scanavacca M, Reddy V, Lustgarten DL, Sosa E *et al.* Effects of radiofrequency pulses delivered in the vicinity of the coronary arteries: implications for nonsurgical transthoracic epicardial catheter ablation to treat ventricular tachycardia. *Pacing Clin Electrophysiol* 2002;**25**:1488–95.
196. Roberts-Thomson KC, Steven D, Seiler J, Inada K, Koplan BA, Tedrow UB *et al.* Coronary artery injury due to catheter ablation in adults: presentations and outcomes. *Circulation* 2009;**120**:1465–73.
197. Viles-Gonzalez JF, de Castro Miranda R, Scanavacca M, Sosa E, d'Avila A. Acute and chronic effects of epicardial radiofrequency applications delivered on epicardial coronary arteries. *Circ Arrhythm Electrophysiol* 2011;**4**:526–31.
198. Sánchez-Quintana D, Cabrera JA, Climent V, Farré J, Weiglein A, Ho SY. How close are the phrenic nerves to cardiac structures? Implications for cardiac interventionalists. *J Cardiovasc Electrophysiol* 2005;**16**:309–13.
199. Joseph KR, Wong TS, Singh J, Orde S, Oborska Y, Mayorchak Y. Single port thoracoscopic diaphragm plication: a novel treatment approach to bilateral phrenic nerve palsy and diaphragm paralysis. *Int J Surg Case Rep* 2023;**108**:108387.
200. Garara B, Wood A, Marcus HJ, Tsang K, Wilson MH, Khan M. Intramuscular diaphragmatic stimulation for patients with traumatic high cervical injuries and ventilator dependent respiratory failure: a systematic review of safety and effectiveness. *Injury* 2016;**47**:539–44.
201. Dellon AL. Nerve grafting and end-to-side neurorrhaphies connecting phrenic nerve to the brachial plexus. *Plast Reconstr Surg* 1996;**98**:905.
202. El-Masri N, Saj F, Wehbe T, Nasrallah G, Ejbeh S. Management of phrenic nerve palsy following cardiac surgery. *J Card Surg* 2018;**33**:534–8.
203. Peichl P, Bulava A, Wichterle D, Schlosser F, Stojadinović P, Borišincová E *et al.* Efficacy and safety of focal pulsed-field ablation for ventricular arrhythmias: two-centre experience. *Europace* 2024;**26**:euae192.
204. Kumareswaran R, Marchlinski FE. Practical guide to ablation for epicardial ventricular tachycardia: when to get access, how to deal with anticoagulation and how to prevent complications. *Arrhythm Electrophysiol Rev* 2018;**7**:159–64.
205. Koruth JS, Chu EW, Bhardwaj R, Dukkipati S, Reddy VY. Esophageal damage during epicardial ventricular tachycardia ablation. *JACC Clin Electrophysiol* 2017;**3**:1470–1.
206. de Moura EG, Silva GL, de Moura ET, Pu LZ, de Castro VL, de Moura DT *et al.* Esophageal perforation after epicardial ablation: an endoscopic approach. *Endoscopy* 2015;**47 Suppl 1 UCTN**:E592–3.
207. Pisani CF, Hachul D, Sosa E, Scanavacca M. Gastric hypomotility following epicardial vagal denervation ablation to treat atrial fibrillation. *J Cardiovasc Electrophysiol* 2008; **19**:211–3.
208. Mahapatra S, LaPar DJ, Bhamidipati CM, McDaniel G, Kamath S, Bunch TJ *et al.* Incidence, risk factors, and consequences of new-onset atrial fibrillation following epicardial ablation for ventricular tachycardia. *Europace* 2011;**13**:548–54.
209. Di Biase L, Romero J, Du X, Mohanty S, Trivedi C, Della Rocca DG *et al.* Catheter ablation of ventricular tachycardia in ischemic cardiomyopathy: impact of concomitant amiodarone therapy on short- and long-term clinical outcomes. *Heart Rhythm* 2021; **18**:885–93.
210. Pedersen MEF, Leo M, Kalla M, Malhotra A, Stone M, Wong K *et al.* Management of tamponade complicating catheter ablation for atrial fibrillation: early removal of pericardial drains is safe and effective and reduces analgesic requirements and hospital stay compared to conventional delayed removal. *JACC Clin Electrophysiol* 2017;**3**: 367–73.
211. Killu AM, Wan SH, Munger TM, Hodge DO, Mulpuru S, Packer DL *et al.* Pericardial effusion following drain removal after percutaneous epicardial access for an electrophysiology procedure. *Pacing Clin Electrophysiol* 2015;**38**:383–90.
212. Richardson TD, Kanagasundram AN, Stevenson WG. How to perform an epicardial ventricular tachycardia ablation: a contemporary and practical approach. *Heart Rhythm* 2021;**18**:2009–13.
213. Muser D, Mendelson T, Fahed J, Liang JJ, Castro SA, Zado E *et al.* Impact of timing of recurrence following catheter ablation of scar-related ventricular tachycardia on subsequent mortality. *Pacing Clin Electrophysiol* 2017;**40**:1010–6.
214. Santangeli P, Frankel DS, Tung R, Vaseghi M, Sauer WH, Tzou WS *et al.* Early mortality after catheter ablation of ventricular tachycardia in patients with structural heart disease. *J Am Coll Cardiol* 2017;**69**:2105–15.
215. Lee JZ, Tan MC, Karikalan S, Deshmukh AJ, Srivathsan K, Shen WK *et al.* Causes of early mortality after ventricular tachycardia ablation in patients with reduced ejection fraction. *JACC Clin Electrophysiol* 2023;**9**:824–32.
216. Liang JJ, Yang W, Santangeli P, Schaller RD, Supple GE, Hutchinson MD *et al.* Amiodarone discontinuation or dose reduction following catheter ablation for ventricular tachycardia in structural heart disease. *JACC Clin Electrophysiol* 2017;**3**:503–11.
217. Wan SH, Killu AM, Hodge DO, Packer DL, Mulpuru S, Asirvatham SJ *et al.* Obesity does not increase complication rate of percutaneous epicardial access. *J Cardiovasc Electrophysiol* 2014;**25**:1174–9.
218. Michowitz Y, Mathuria N, Tung R, Esmailian F, Kwon M, Nakahara S *et al.* Hybrid procedures for epicardial catheter ablation of ventricular tachycardia: value of surgical access. *Heart Rhythm* 2010;**7**:1635–43.
219. Zhang PP, Heeger CH, Mathew S, Fink T, Reissmann B, Lemeš C *et al.* Left-lateral thoracotomy for catheter ablation of scar-related ventricular tachycardia in patients with inaccessible pericardial access. *Clin Res Cardiol* 2021;**110**:801–9.
220. Tschabrunn CM, Haqqani HM, Cooper JM, Dixit S, Garcia FC, Gerstenfeld EP *et al.* Percutaneous epicardial ventricular tachycardia ablation after noncoronary cardiac surgery or pericarditis. *Heart Rhythm* 2013;**10**:165–9.
221. Tung R, Shivkumar K. Epicardial ablation of ventricular tachycardia. *Methodist Debakey Cardiovasc J* 2015;**11**:129–34.
222. Ho CY, Al Sinan A, DeBoard Z, Swampillai J, Timmins K, Stiles MK. Hybrid surgical-catheter epicardial ablation of recurrent ventricular tachycardia in an arrhythmogenic cardiomyopathy patient with pericardial adhesions following COVID-19 infection. *HeartRhythm Case Rep* 2024;**10**:15–20.
223. Li A, Buch E, Boyle NG, Shivkumar K, Bradfield JS. Incidence and significance of adhesions encountered during epicardial mapping and ablation of ventricular tachycardia in patients with no history of prior cardiac surgery or pericarditis. *Heart Rhythm* 2018;**15**: 65–74.
224. Sanaka H, Haroun E, Arockiam AD, Dong T, Klein A, Wang TKM. Advances in the multimodality imaging and management of recurrent pericarditis: a contemporary review. *Curr Cardiol Rep* 2024;**26**:1359–75.
225. Cosyns B, Plein S, Nihoyanopoulos P, Smiseth O, Achenbach S, Andrade MJ *et al.* European Association of Cardiovascular Imaging (EACVI) position paper: multimodality imaging in pericardial disease. *Eur Heart J Cardiovasc Imaging* 2015;**16**:12–31.
226. Klein AL, Abbara S, Agler DA, Appleton CP, Asher CR, Hoit B *et al.* American Society of Echocardiography clinical recommendations for multimodality cardiovascular imaging of patients with pericardial disease: endorsed by the Society for Cardiovascular Magnetic Resonance and Society of Cardiovascular Computed Tomography. *J Am Soc Echocardiogr* 2013;**26**:965–1012.e15.
227. Tschabrunn CM, Haqqani HM, Zado ES, Marchlinski FE. Repeat percutaneous epicardial mapping and ablation of ventricular tachycardia: safety and outcome. *J Cardiovasc Electrophysiol* 2012;**23**:744–9.
228. Jincun G, Faguang Z, Weibin H, Yan W, Kang D, Tung R. Outside-in subepicardial dissection during percutaneous epicardial ventricular tachycardia ablation. *Circ Arrhythm Electrophysiol* 2016;**9**:e004499.
229. Killu AM, Ebrille E, Asirvatham SJ, Munger TM, McLeod CJ, Packer DL *et al.* Percutaneous epicardial access for mapping and ablation is feasible in patients with prior cardiac surgery, including coronary bypass surgery. *Circ Arrhythm Electrophysiol* 2015;**8**:94–101.
230. Wang Z, Li J, Chen J, Guo H, He H, Jiao S *et al.* Relationship between epicardial adipose tissue volume and recurrence after ablation in premature ventricular complexes. *Circ J* 2023;**88**:1047–54.
231. Sepehri Shamloo A, Schoene K, Stauber A, Darma A, Dagres N, Dinov B *et al.* Epicardial adipose tissue thickness as an independent predictor of ventricular tachycardia recurrence following ablation. *Heart Rhythm* 2019;**16**:1492–8.
232. Sasaki T, Mudd J, Steenbergen C, Zviman MM, Miller CF, Nazarian S. Impact of scar, viable myocardium, and epicardial fat on substrate identification of ventricular tachycardia in a case with nonischemic cardiomyopathy. *Pacing Clin Electrophysiol* 2012; **35**:e345–8.

233. Cano O, Hutchinson M, Lin D, Garcia F, Zado E, Bala R *et al.* Electroanatomic substrate and ablation outcome for suspected epicardial ventricular tachycardia in left ventricular nonischemic cardiomyopathy. *J Am Coll Cardiol* 2009;**54**:799–808.
234. Hutchinson MD, Gerstenfeld EP, Desjardins B, Bala R, Riley MP, Garcia FC *et al.* Endocardial unipolar voltage mapping to detect epicardial ventricular tachycardia substrate in patients with nonischemic left ventricular cardiomyopathy. *Circ Arrhythm Electrophysiol* 2011;**4**:49–55.
235. Bazan V, Frankel DS, Santangeli P, Garcia FC, Tschabrunn CM, Marchlinski FE. Three-dimensional myocardial scar characterization from the endocardium: usefulness of endocardial unipolar electroanatomic mapping. *J Cardiovasc Electrophysiol* 2019;**30**:427–37.
236. Jacobson JT, Hutchinson MD, Cooper JM, Woo YJ, Shandler RS, Callans DJ. Tissue-specific variability in human epicardial impedance. *J Cardiovasc Electrophysiol* 2011;**22**:436–9.
237. Aranyó J, Martínez-Falguera D, Bazan V, Fadeuilhe E, Teis A, Sarrias A *et al.* Biophysical tissue characterization of ventricular tachycardia substrate with local impedance mapping to predict critical sites. *JACC Clin Electrophysiol* 2023;**9**:765–75.
238. Sourwine M, Jeudy J, Miller B, Vunnam R, Imanli H, Mesubi O *et al.* Location, variations, and predictors of epicardial fat mapping using multidetector computed tomography to assist epicardial ventricular tachycardia ablation. *Pacing Clin Electrophysiol* 2017;**40**:1059–66.
239. Younis A, Zilberman I, Krywanczyk A, Higuchi K, Yavin HD, Sroubek J *et al.* Effect of pulsed-field and radiofrequency ablation on heterogeneous ventricular scar in a swine model of healed myocardial infarction. *Circ Arrhythm Electrophysiol* 2022;**15**:e011209.
240. Im SI, Higuchi S, Lee A, Stillson C, Buck E, Morrow B *et al.* Pulsed field ablation of left ventricular myocardium in a swine infarct model. *JACC Clin Electrophysiol* 2022;**8**:722–31.
241. Nitta T, Sakamoto SI, Murata H, Suzuki K, Yamada N, Iwasaki Y *et al.* Surgery for ventricular tachycardia originating from the left ventricular summit. *Eur J Cardiothorac Surg* 2023;**64**:ezad323.