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EHRA DOCUMENT

EHRA European Heart Rhythm Association

# Workup and management of rhythm disorders in myocarditis and inflammatory cardiomyopathy: a clinical consensus statement of the European Heart Rhythm Association and the Heart Failure Association of the ESC, the ESC Working Group on Myocardial & Pericardial Diseases, the European Association of Preventive Cardiology of the ESC, the Heart Rhythm Society, the Asian Pacific Heart Rhythm Society, and the Latin American Heart Rhythm Society

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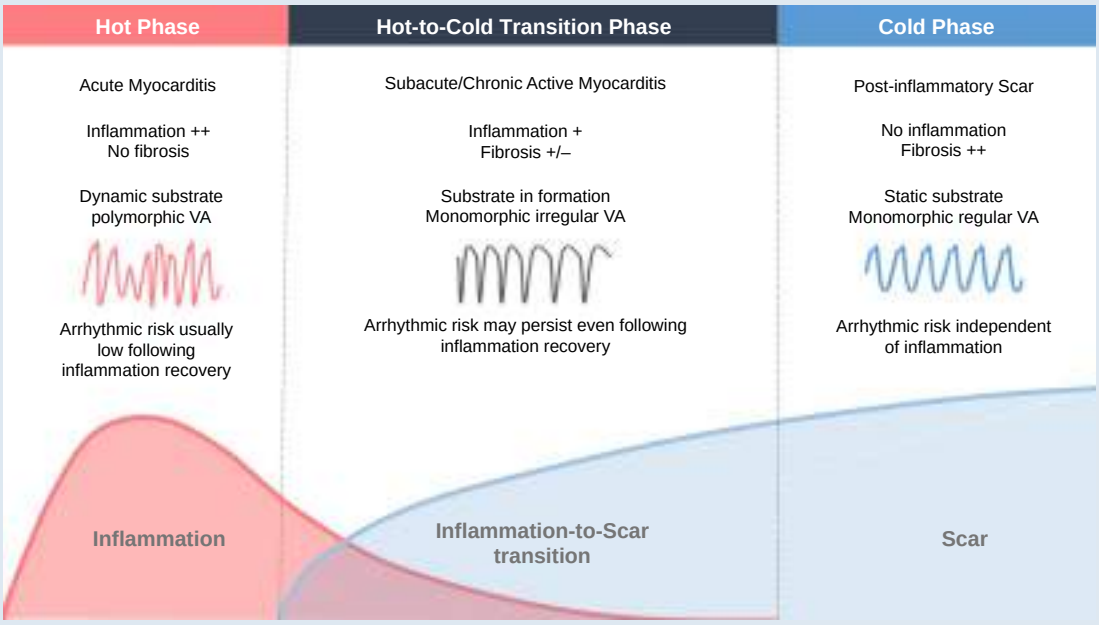
Myocarditis is an important and frequently underrecognized cause of atrioventricular conduction disease and ventricular arrhythmias and may also be associated with atrial arrhythmias and sinus node dysfunction. Arrhythmic risk evolves over time as a result of the interplay between myocardial inflammation, fibrosis, and genetic predisposition. Despite its clinical relevance, guidance for the diagnosis, treatment, and follow-up of arrhythmias in myocarditis has remained limited. This consensus statement provides a structured framework for the management of arrhythmias in myocarditis and inflammatory cardiomyopathy (Infl-CMP), integrating disease phase and genetic background into clinical decision-making. It offers practical guidance across the disease spectrum, including diagnostic evaluation, therapeutic strategies, and longitudinal follow-up. The document was jointly developed by the European Heart Rhythm Association of the European Society of Cardiology (ESC) in collaboration with the Heart Failure Association of the ESC, the ESC Working Group on Myocardial & Pericardial Diseases, and the European Association of Preventive Cardiology of the ESC, together with the Heart Rhythm Society, the Asia Pacific Heart Rhythm Society, and the Latin American Heart Rhythm Society. Consensus advice is organized using a phase-aware framework distinguishing hot, hot-to-cold, and cold phases, with clinical consensus statements graded according to opinion-, observational-, or randomized trial-based evidence and supported by formal author voting. Arrhythmia management is stratified by disease phase. During active inflammation, antiarrhythmic therapy is combined with aetiology-targeted treatment or immunosuppression when indicated. In later stages, substrate-based strategies include pharmacological therapy, device implantation, or ablation. Emphasis is placed on hot-to-cold transition as an arrhythmogenic window and on risk stratification.

## Graphical Abstract




**Keywords** Myocarditis • Inflammatory cardiomyopathy • Arrhythmias • Rhythm disorders • Sudden cardiac death • Heart failure • Hot • Cold • Hot-to-cold

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# Introduction

## Arrhythmic myocarditis: a clinical interface between inflammation and electrophysiology

Arrhythmic myocarditis represents a critical overlap between myocardial inflammation and electrical instability. The condition frequently manifests with ventricular arrhythmias (VA), conduction disturbances, or electrical storm—even in the absence of overt heart failure or left ventricular (LV) dysfunction.[1–3] Supraventricular tachyarrhythmias may occur in the whole spectrum of inflammatory myopericardial syndromes (IMPS).[1] Inflammatory and fibrotic substrates evolve over time, and the clinical trajectory can shift rapidly from acute, reversible inflammation to chronic, scar-driven arrhythmogenicity. While the 2025 European Society of Cardiology (ESC) guidelines[1] classify myocarditis as acute, subacute or chronic based on clinical criteria (duration of symptoms), arrhythmic risk often evolves through stages that do not perfectly match with the time of symptom onset. We therefore introduce the phases 'hot', 'hot-to-cold', and 'cold', corresponding to active inflammation, transition to early scar formation, and stable post-inflammatory scar. This model complements the ESC framework and provides the additional granularity required for arrhythmic risk assessment and management.

### We hereby define the phases (or stages) of arrhythmic myocarditis as follows:

- **Hot phase**: Characterized by active myocardial inflammation with electrically unstable myocardium, typically presenting during acute myocarditis with polymorphic/pleomorphic ventricular arrhythmias, new-onset conduction disturbances, supraventricular tachyarrhythmias, or electrical storm, often irrespective of left ventricular function.

*Example: a patient presenting with ventricular fibrillation and newly diagnosed acute myocarditis with myocardial oedema and necrosis on imaging/histology, without evidence of tissue scarring.*

- **Hot-to-cold transition**: a distinct electrically high-risk window occurring after the acute inflammatory phase, in which inflammatory activity progressively declines while early fibrotic remodelling promotes the development of re-entrant arrhythmogenic substrates. During this stage, ventricular arrhythmias increasingly reflect organized re-entry mechanisms despite apparent normalization of biomarkers and partial recovery on cardiac imaging.

*Example: a patient presenting with monomorphic ventricular tachycardia and persistent myocardial inflammation on imaging/histology, associated with evolving tissue scarring.*

- **Cold phase**: Defined by chronic post-inflammatory scar-related arrhythmogenicity, in which ventricular tachycardia and conduction disease are predominantly sustained by stable fibrotic substrate rather than active inflammation.

*Example: a patient presenting with monomorphic ventricular tachycardia late after healed myocarditis, with isolated tissue scarring on imaging/histology and no residual inflammation.*

The hot phase is marked by active inflammation, polymorphic/pleomorphic VA, new-onset bradyarrhythmias and supraventricular tachyarrhythmias.[4,5] During the cold phase tachyarrhythmias and conduction disease emerge from residual myocardial scarring.[4,5] In the hot-to-cold transition phase re-entrant VA may manifest despite progressive normalization of imaging and biomarkers.[6,7] Acknowledging this phase is essential for improving risk stratification and guiding treatment.[6,8] Therapeutic decisions—whether anti-inflammatory, antiarrhythmic, interventional, or device-based—must therefore be tailored to the underlying phase and rhythm phenotype.[8] *Figure 1* shows the phases of arrhythmic myocarditis.

Not all patients are at equal risk. Genetic predisposition plays a pivotal role in shaping both the inflammatory response and the arrhythmic profile.[9–11] Variants in genes such as *LMNA*, *DSP*, or *FLNC* may predispose to early VA, atrioventricular (AV) block, or rapid fibrotic remodelling—even in the setting of preserved ejection fraction or mild inflammatory triggers.[12,13] In particular, the concept of *hot-phase genetic cardiomyopathy* (HPGC) has emerged as a unifying framework for genetically primed myocarditis-like presentations with disproportionate electrical instability.[10,14] We also acknowledge the ESC 2025 terminology for the IMPS, distinguishing myopericarditis (pericarditis-predominant with limited myocardial injury and preserved systolic function) from perimyocarditis (myocarditis-predominant with pericardial involvement).[1] Since arrhythmic risk is driven by myocardial substrate (scar burden/topology, VA), our phase model (hot, cold, transition phase) applies mainly to perimyocarditis.

In this clinical consensus statement, we provide a structured, phase-specific, and rhythm-guided framework for managing arrhythmic myocarditis and inflammatory cardiomyopathy (Infl-CMP). By integrating electrophysiology, imaging, immunology, and genetics, this document aims to support decision-making in this complex clinical field. Arrhythmic myocarditis is not a fixed entity—it is a moving target. Clinical vigilance, interdisciplinary collaboration, and phase-adapted action are essential for improving patient outcomes.[1]

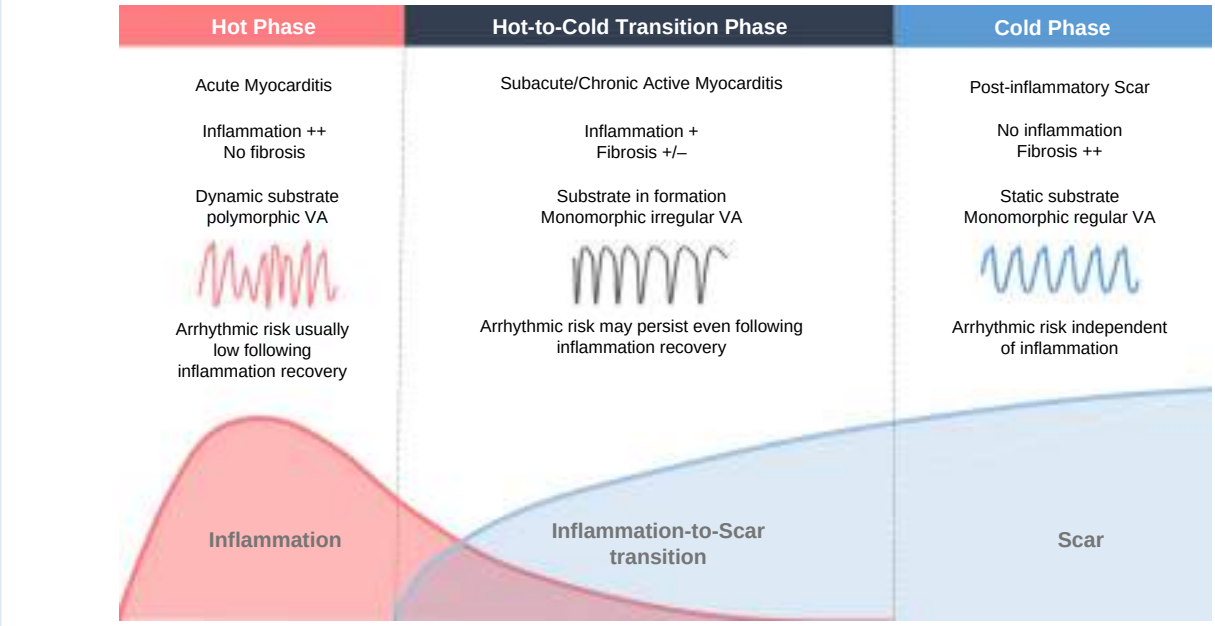


**Figure 1** Phases of arrhythmic myocarditis. Phases (or stages) or arrhythmic myocarditis are shown. VA, ventricular arrhythmias.

# Methods

This clinical consensus statement is not intended as a guideline but as a support for clinical management. The definitions of the category of advice and associated areas of uncertainty are based on updated systematic review of the literature, as detailed in Supplementary material online, *Table S0.1*. The type of supporting evidence and strength of evidence are listed in Supplementary material online, *Table S0.2*. Throughout, ESC 2025 Guidelines recommendations are cited as context,[1] while our statements are issued as consensus advice with expert opinion (OPN), observational study (OBS), and randomized clinical trials (RCT) grading as detailed in Supplementary material online, *Table S0.2*.

Each advice statement underwent independent voting by all co-authors, regardless of evidence level. In detail, ≥70% agreement was required for inclusion; ≥90% defined strong consensus. We report our grading (RCT/OBS/OPN) and voting percentage for each statement; where helpful, we add the ESC Guidelines recommendation as contextual information.

# Chapter 1: risk stratification and patient profiling

**Who is at risk?**

Risk stratification in myocarditis and Infl-CMP requires a nuanced, phase-oriented understanding of the interaction between inflammatory activity, structural remodelling, and arrhythmic phenotype. Both patient-specific and disease-specific factors—genetic background, inflammation severity, substrate distribution—contribute to arrhythmic risk, which evolves over time. This chapter summarizes definitions, aetiologies, clinical phases, and risk enhancers that shape the arrhythmic trajectory in myocarditis, with supporting tables and structured references.

## Concepts and definitions: inflammation meets electrophysiology

According to World Health Organization/ESC consensus, myocarditis is diagnosed histologically by myocardial inflammatory infiltrates with non-ischaemic myocyte necrosis,[1,3,15,16] and immunohistologically by ≥14 leukocytes/mm$^2$ including ≥7 CD3+ T cells.[1,3,17–20] Refined diagnostic criteria for lymphocytic myocarditis with a scoring system (so-called Seaport criteria) have been recently published,[21] and may help to rule out differential diagnoses. Infl-CMP describes chronic myocarditis in association with cardiac dysfunction and ventricular remodelling with clinical phenotype of hypokinetic, either dilated or non-dilated cardiomyopathy with/without arrhythmogenic substrate.[1] These entities form a dynamic continuum ranging from self-limiting inflammation to fibrotic remodelling and heart failure. EMB defines both myocarditis stage and histotype (*Figure 2*).[3,22] The 2025 ESC guidelines on Myocarditis and Pericarditis highlight the value of cardiac magnetic resonance (CMR) as the first-choice gold standard diagnostic test in uncomplicated scenarios.[1,23] Instead, in patients haemodynamically unstable, as well as in those with other arrhythmia-related limitations to CMR (implanted cardiac devices, fast/irregular heartbeat), EMB is still strongly advised.[1–3,24] In addition to troponin and natriuretic peptides, C-reactive protein (CRP) is useful to monitor inflammatory activity in suspected IMPS.[1,25] The combination of EMB, multimodality imaging and biomarkers may help to appropriately stage arrhythmic myocarditis. *Figure 3* shows relevant diagnostic tests and the main applications of EMB and CMR.

In turn, identification of the hot-to-cold phase requires patient-tailored, guideline-directed multi-parameter assessment: (i) EMB (when indicated): partial regression of inflammation with early fibrosis; (ii) CMR: residual or near-normal T2 values with persistent or modifying late gadolinium enhancement (LGE); (iii) Rhythm monitoring: persistence of VA despite clinical improvement; and (iv) Clinical/biomarkers: improving symptoms with declining troponin/CRP. This phase is frequently subacute/chronic by ESC Guidelines criteria,[1] yet carries distinct arrhythmic implications that may influence timing of ablation or device therapy.

A relapsing pattern may be associated with genetically primed disease, often linked to variants in *DSP* and other desmosomal

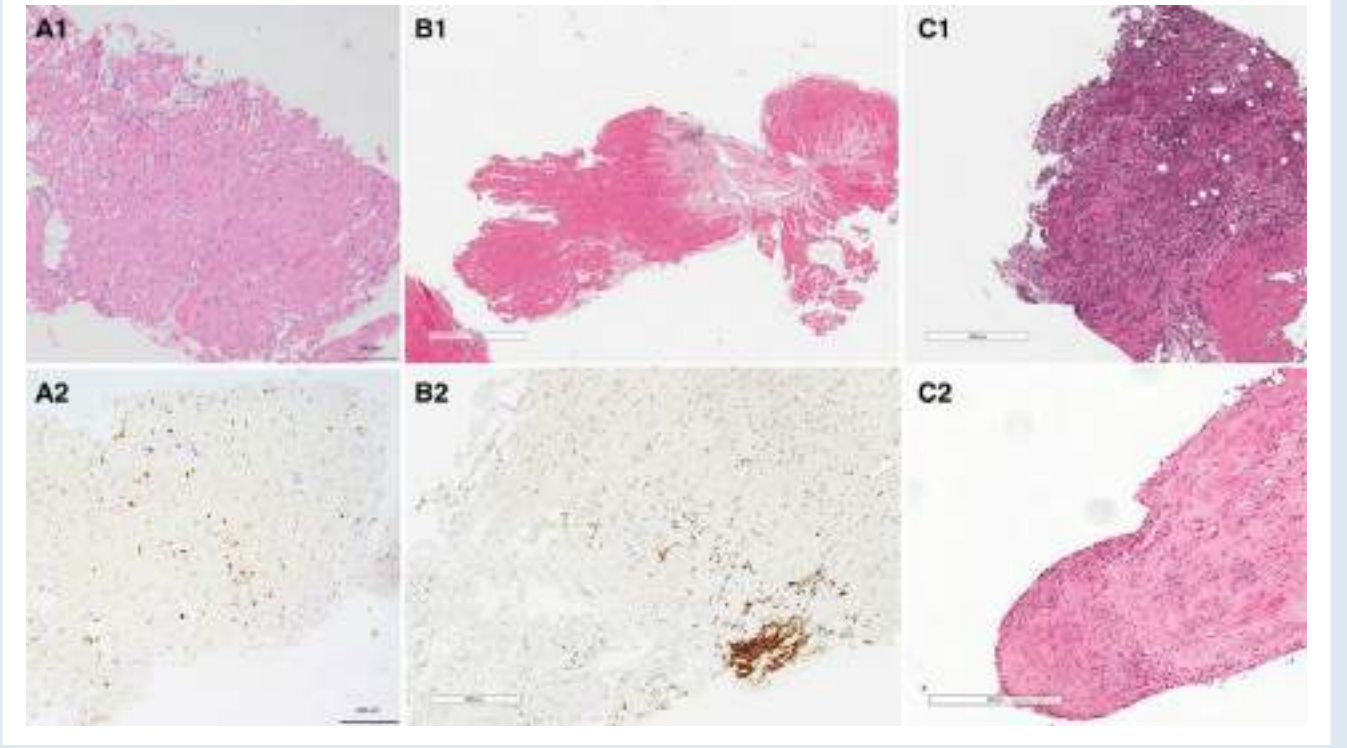


**Figure 2** EMB-defined stages and histotypes of arrhythmic myocarditis. EMB-defined stages and histotypes of arrhythmic myocarditis. (*A*) Lymphocytic myocarditis, hot phase (acute): inflammatory infiltrates, oedema, non-ischaemic necrosis (A1, HE), CD3+ T lymphocytes > 7/mm$^2$ and >15/high power field (mild myocarditis according to 2025 Seaport criteria) (A2, IHC). (*B*) Lymphocytic myocarditis, hot-to-cold phase (chronically active): inflammatory infiltrates, replacement fibrosis (B1, HE), >2 clusters of CD3+ T lymphocytes (moderate myocarditis according to 2025 Seaport criteria) (B2, IHC). (*C*) Special histotypes with strong arrhythmic associations, namely: giant cell myocarditis (C1, HE) and cardiac sarcoidosis with noncaseating granulomas (C2, HE). EMB, endomyocardial biopsy; HE, haematoxylin eosin; IHC, immunohistochemistry.

genes.[9–11,26–30] These entities, grouped under the umbrella of HPGC,[1,14] challenge classical distinctions and require dedicated stratification strategies. Supplementary material online, *Table S1.1* summarizes working definitions of arrhythmic myocarditis and overlap conditions, including subtypes with recurring inflammation, scar formation, or genetic triggers. The temporal and mechanistic evolution of inflammation is further illustrated in Supplementary material online, *Table S1.2*, which aligns phases with clinical and electrophysiological implications.

## Aetiological spectrum and arrhythmia patterns

The arrhythmogenic potential of myocarditis varies by aetiology (see Supplementary material online, *Table S1.3*). Viral pathogens—such as parvovirus B19, human herpesvirus 6 (HHV6), and adenovirus—may drive both acute VA and delayed scar-related ventricular tachycardia (VT).[1,31] In some cases (e.g. HHV6), latency and reactivation may contribute to relapses.[2]

Bacterial and parasitic infections add specific patterns: Lyme myocarditis causes mostly reversible AV block;[32,33] Trypanosoma cruzi (Chagas disease) leads to sustained VT and conduction disease due to diffuse fibrosis.[34] Tuberculosis is more frequently associated with pericardial diseases but may also cause isolated myocarditis.[1] A range of tropical infections (e.g. Dengue, malaria, leptospirosis, rickettsioses, West Nile virus) may present with arrhythmic complications in the febrile or subacute phase.[35] These are summarized in Supplementary material online, *Table S1.4*, highlighting the importance of travel history in unexplained arrhythmias.

Immune-mediated forms (e.g. sarcoidosis, giant cell, eosinophilic myocarditis) may affect the conduction system or provoke sustained VA.[4,36] Giant cell myocarditis (GCM) in particular shows aggressive course and demands early immunosuppression.[37,38] Arrhythmias frequently complicate myocarditis even in the context of systemic rheumatologic diseases.[39] Drug-induced or immune-mediated myocarditis (e.g. immune checkpoint inhibitors) increasingly contributes to malignant arrhythmias.[40]

Finally, pathogenic/likely pathogenic variants in cardiomyopathy-associated genes, including but not limited to desmoplakin (*DSP*), filamin C (*FLNC*), and lamin A/C, (*LMNA*) encoding genes, may predispose to electrical instability, even under mild inflammatory triggers.[10,29] Inflammatory events may unmask these genetic substrates, initiating a progressive course. Supplementary material online, *Table S1.5* consolidates risk-enhancing features and clinical red flags[41] that require early recognition.

## Mechanisms and substrates of arrhythmia in myocarditis

Arrhythmias in myocarditis range from polymorphic VT in the hot phase to re-entrant scar-related VT in the cold stage,

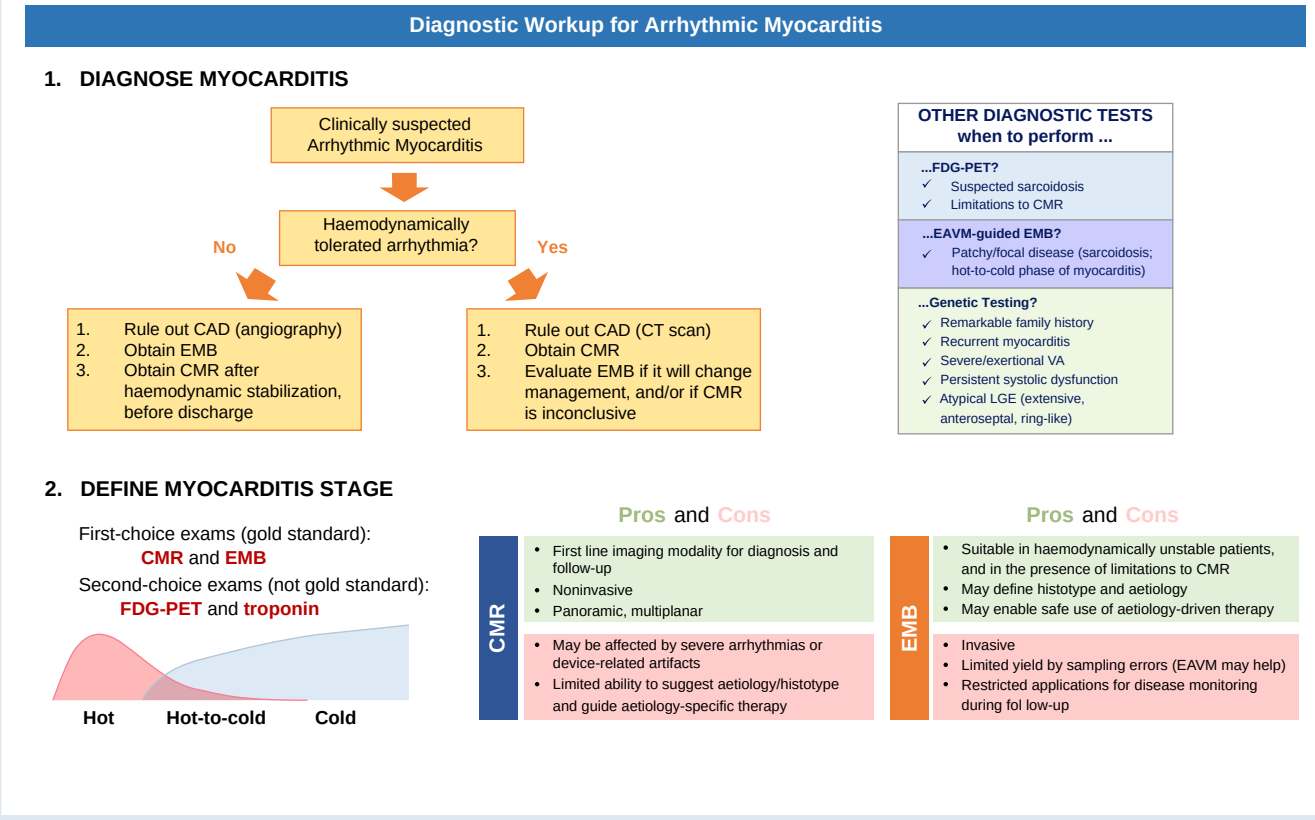


**Figure 3** Diagnostic workup for arrhythmic myocarditis. Diagnostic workup for arrhythmic myocarditis is shown, along with comparison between gold standard techniques (CMR and EMB). CMR and EMB provide complementary information; neither modality alone is sufficient to fully characterize arrhythmic myocarditis. CAD, coronary artery disease; CMR, cardiac magnetic resonance; CT, computed tomography; EAVM, electroanatomic voltage map; EMB, endomyocardial biopsy; FDG-PET, 18F-fluorodeoxyglucose positron emission tomography; ICD, implantable cardioverter defibrillator; LGE, late gadolinium enhancement; VA, ventricular arrhythmias.

causing 3–6% of the sudden cardiac death (SCD) in the young.[4,42,43] Sustained monomorphic VTs have been described even in the hot-to-cold transition phase,[6,8] when early fibrotic remodelling and arrhythmogenic re-entry substrates may already form. High-degree AV block may indicate specific aetiologies involving the interventricular septum (e.g. sarcoidosis, Chagas disease, GCM, Lyme disease, HPGC related to *LMNA* or other genes with septal involvement),[44] whereas non-sustained VT (NSVT) and frequent premature ventricular complexes (PVCs) reflect substrate irritability.[6] In this setting, inflammation of the conduction tissue may play a proarrhythmic role.[45] Brugada-like ECG patterns may transiently emerge during fever or epicardial inflammation, revealing latent channelopathies.[46,47] Inflammatory cytokines such as IL-1β and TNF-α induce proarrhythmic changes, including QT prolongation, repolarization instability, and increased susceptibility to early afterdepolarizations.[7,48] In turn, the functional impairment of intercalated disks may favour myocyte uncoupling and arrhythmias.[49] The complex interplay between inflammatory cytokines and electrical instability advocate for increased vigilance regarding arrhythmia detection and early stratification.[4] An overview of arrhythmia mechanisms is provided in Supplementary material online, *Table S1.6*.

These rhythm types, their phase associations, and underlying mechanisms are detailed in Supplementary material online, *Tables S1.2* and *S1.6*, providing a substrate-focused taxonomy of arrhythmia types. Supplementary material online, *Tables S1.2* and *S1.3* together form the functional backbone of clinical risk interpretation across the inflammatory spectrum.

## Summary

Arrhythmic risk in myocarditis is multifactorial, time-dependent, and frequently dynamic. Accurate risk profiling requires integration of clinical phase (hot vs. hot-to-cold vs. cold), imaging and biopsy data, arrhythmia type, and genetic background. Supplementary material online, *Table S1.7* summarizes the key features of hot vs. hot-to-cold vs. cold arrhythmic myocarditis. As reported in ESC Guidelines, NSVT, extensive LGE on CMR, unexplained syncope, positivity of electrophysiological testing, and reduced systolic function (LVEF <50%) are listed as the risk factors for SCD.[1] Additional factors may include non-lymphocytic histotypes[50] and distinct cardiomyopathy-associated genotypes.[28] Importantly, monomorphic VA complicating the cold and hot-to-cold phases of myocarditis are at higher risk of recurrences as compared to polymorphic VA occurring in the hot phase.[6] Multidisciplinary approach and phase-adapted surveillance and individualized therapy are essential to prevent arrhythmic progression and sudden cardiac death.[1,51]

**Table of Advice 1** Concepts, definitions, and phase labelling

| Advice | Phase | Category of advice | Strength of evidence | Vote (%) | References |
|---|---|---|---|---|---|
| **Label arrhythmic myocarditis at every patient contact with phase (hot; hot-to-cold; cold).** | All | ✓ | 👥 | 85 | • Peretto *et al.* 2020[4]<br>• Peretto, *et al.* 2020[5]<br>• Peretto *et al.* 2025[8] |
| **Define myocarditis stage by CMR and, when appropriate, EMB.** | All | ✓ | 🧩 | 85 | • Ferreira *et al.* 2018[23]<br>• Schulz-Menger *et al.* 2025[1]<br>• Caforio *et al.* 2013[3] |
| **Identify the hot-to-cold transition phase by CMR (persistent/evolving LGE and residual T2-imaging) and/or EMB (fibrosis and persistent inflammation), and treat it as an evolving and proarrhythmic window.** | Hot-to-Cold | ✓ | 👥 | 85 | • Schulz-Menger *et al.* 2025[1]<br>Rav-Acha *et al.*[6] |
| **Refer to multidisciplinary teams when uncertainty exists in diagnosis or treatment decisions.** | All | ✓ | 🧩 | 100 | • Peretto *et al.* 2020[51] |

CMR, cardiac magnetic resonance; EMB, endomyocardial biopsy; ESC, European Society of Cardiology; LGE, late gadolinium enhancement; OBS, observational study-based evidence; OPN, expert opinion-based evidence.

# Chapter 2: substrate-oriented diagnostics in myocarditis and inflammatory cardiomyopathy

**How to detect and define the substrate?**

This chapter outlines how to detect and define the arrhythmogenic substrate in myocarditis and inflammatory cardiomyopathy. The diagnostic tools presented here support the structured algorithms discussed in Chapter 3. Supplementary material online, *Tables S2.1* and *S2.3* provide a concise overview of diagnostic red flags, immune-genetic markers, and substrate-related arrhythmogenic findings. *Figure 4* shows representative examples of imaging findings in arrhythmic myocarditis.

## Diagnostic rationale

Accurate identification of the arrhythmogenic substrate is central to managing rhythm disorders in myocarditis. Diagnostic strategies must distinguish inflammation-driven arrhythmias in the hot phase from scar- or genetically mediated mechanisms in the cold phase. This requires a multiparametric approach combining biomarkers, ECG, imaging, rhythm monitoring, and—when appropriate—EMB or genetic testing. In IMPS, early separation of myopericarditis from perimyocarditis matters because risk pathways diverge. Myopericarditis follows a pericarditis-led pathway; myocardial 'flags'—meaningful scar or VA—prompt closer rhythm surveillance.[1]

## ECG and rhythm monitoring

Standard ECG remains a key initial tool and may reveal conduction delays, QRS fragmentation, or repolarization abnormalities.[52] Distinct ECG features, rather than VA types (e.g. sustained VT, NSVT, PVCs) are suggestive for different stages of myocarditis. In the hot phase, polymorphic/pleomorphic VA or electrical instability predominate, while monomorphic VT with fixed QRS morphology suggests scar-related re-entry in the cold phase.[4,5] However, clinical onset with sustained monomorphic VT may be observed also in the hot-to-cold transition phase.[6,53] In this setting, analysis of VT cycle stability may help differentiate transition (high variability) vs. post-inflammatory stage (low variability).[4,5] To enable appropriate characterization of VA, 12-lead recording is always advised.[4,54] Supplementary material online, *Table S2.1* summarizes key arrhythmia types, ECG clues, and substrate correlations. Holter and extended rhythm monitoring are essential for risk stratification, especially in patients with unexplained symptoms, persistent LGE, or reduced left ventricular ejection fraction (LVEF).[55] Implantable loop recorders (ILRs) allow sensitive detection of arrhythmias, and may help monitoring selected patients who do not meet the criteria for implantable cardioverter defibrillator (ICD).[56] The suggested criteria for candidacy to ILR are listed in Table of Advice 2. For device carriers, high-sensitivity programming and remote monitoring help quantify arrhythmia burden over time.

## Echocardiography

Echocardiography is valuable for initial and serial assessment.[57,58] In acute myocarditis, it may reveal global systolic dysfunction or wall motion abnormalities, oedema-related pseudohypertrophy, or pericardial effusion.[3] In chronic stages, findings include wall thinning with regional akinesia or

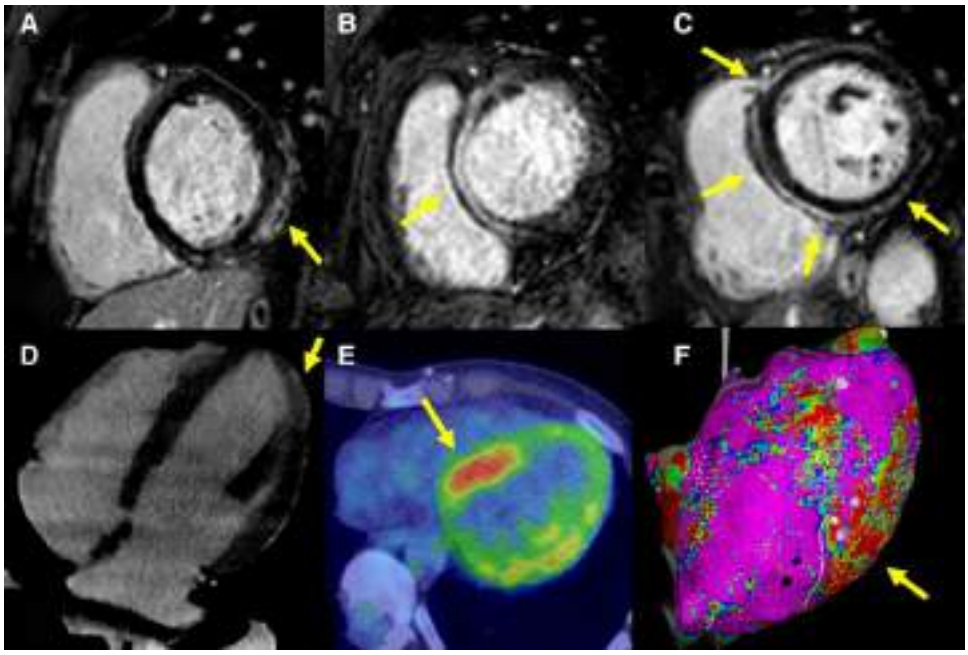


**Figure 4** Imaging findings in arrhythmic myocarditis. Imaging findings are shown for patients with arrhythmic myocarditis. (*A*–*C*). CMR showing nonischaemic LGE in a patient with: (*A*) autoimmune myocarditis (inferolateral distribution, arrow); (*B*) *FLNC* gene variant (septal distribution, arrow); (*C*) *DSP* gene variant (ring-like distribution, arrows). (*D*) CT scan showing nonischaemic late iodine enhancement (lateral apical distribution, arrow) in a patient with acute myocarditis and normal coronary arteries. (*E*) FDG-PET scan consistent with hot-phase cardiomyopathy in a patient with *LMNA* gene variant (septal distribution, arrow). (*F*) Endocardial bipolar EAVM (left anterior oblique view; cut-off values 0.5 mV for dense scar—red; 1.5 mV for normal voltage—purple) showing low-voltage areas (dominant inferolateral distribution, arrow) in a patient with chronic myocarditis (cold phase). CMR, cardiac magnetic resonance; *DSP*, desmoplakin gene; EAVM, electroanatomic voltage mapping; FDG-PET, 18F-fluorodeoxyglucose positron emission tomography; *FLNC*, filamin C gene; LGE, late gadolinium enhancement; *LMNA*, lamin A/C gene.

dyskinesia. Global longitudinal strain enhances detection of systolic dysfunction even in patients with preserved LVEF.[1]

## Cardiac magnetic resonance

CMR with contrast remains the gold standard for non-invasive tissue characterization.[1,2] As deemed by the updated Lake Louise criteria,[23] T2-weighted imaging and T'-mapping detect oedema in the hot phase. As a marker of myocardial injury, LGE may revert after the acute phase of myocarditis[58]; as opposed, striae of LGE persisting in the chronic stage commonly indicate fibrosis,[1,4] and are linked to arrhythmia recurrences following presentation with sustained VT.[59] Recent data[6] suggest that the diagnostic consistency of CMR in arrhythmic myocarditis is higher as compared to prior studies.[58] Moreover, striae of LGE displaying either septal/anteroseptal or ring-like patterns (see Supplementary material online, *Table S2.1*) are linked to sustained VA and worse outcomes in most[13,60] but not all studies.[6] Extensive LGE (although this definition lacks a universally accepted quantitative cut-off) provides prognostic insight and may prompt ICD consideration in borderline cases, especially during the cold phase of myocarditis.[61] Finally, delayed-enhanced CMR plays a growing role in planning and guiding electrophysiological procedures.[62]

## PET and cardiac CT

$^{18}$F-fluorodeoxyglucose positron emission tomography (FDG-PET) is particularly helpful in suspected sarcoidosis or GCM.[1] Special patient preparation lacking carbohydrates and using a high fat diet with prolonged fasting is needed. It detects metabolically active lesions, even in preserved myocardium.[63] Anteroseptal FDG uptake correlates with arrhythmic risk.[64] In selected cases, gallium-67 scintigraphy plays as an alternative technique to monitor myocardial inflammation.[65] Coronary CT angiography helps to exclude significant coronary artery disease;[3] delayed iodine enhancement CT may visualize fibrosis when CMR is contraindicated or altered by device-related artifacts.[66] Both FDG-PET and CT scan provide relevant information during the pre-procedural workup for patients undergoing VA catheter ablation.[62]

## Electroanatomic mapping as a diagnostic test

Electroanatomic mapping allows identification of arrhythmogenic scar substrates, especially in the cold phase of myocarditis.[4] In detail, electroanatomical voltage mapping (EAVM) identifies low-voltage areas that correspond to fibrotic substrate. Bipolar mapping detects dense scar (<0.5 mV), while unipolar mapping captures deeper or epicardial involvement (<8.3 mV LV, <5.5 mV RV) with higher sensitivity.[67–70] In most patients with myocarditis, substrate abnormalities are located in inferolateral LV wall (either epicardial or endocardial unipolar mapping), whereas septal localization is more common in sarcoidosis, GCM, and HPGC as *LMNA* or *FLNC*.[65,71–73]

In the cold phase, EAVM guides ablation strategies;[70] in the hot phase, it may help distinguishing transient oedema from

fixed scar.[74] EAVM provides accurate tissue characterization in patients with limitations to CMR;[75] combined with imaging, it improves diagnostic yield and supports targeted EMB.[73,76,77] Importantly, since low-voltage areas correlate with LGE rather than oedema,[74] the use of EAVM as a diagnostic tool—and for guiding EMB sampling—, is particularly advised in the chronic/hot-to-cold transition rather than in acute/hot phase.[76,78] As shown in *Figure 5*, EAVM-guided sampling in experienced centres can raise diagnostic yield in focal/patchy IMPS as sarcoidosis,[73] where differentiation from arrhythmogenic right ventricular cardiomyopathy (ARVC) may be an issue.[79] To enable a more sensitive targeting of non-ischaemic scars in chronic myocarditis, future applications may include the use of percutaneous epicardial biopsy, a technique already proven feasible in pericardial diseases.[80,81] See Supplementary material online, *Table S2.1* for key substrate-related red flags.

## Endomyocardial biopsy

EMB is the invasive gold standard for diagnosing myocarditis.[1] It enables direct confirmation of myocardial inflammation, assessment of disease stage, and identification of infectious or autoimmune aetiology. Also, it allows differentiation of distinct histotypes even in cases when CMR is not conclusive or unrevealing.[82,83] However, in routine clinical practice, EMB may be constrained by local availability, expertise, and centre-specific logistics. EMB should be considered in accordance with the 2025 ESC Guidelines,[1] particularly in patients with new-onset heart failure, life-threatening VA, high-grade AV block, or suspected non-lymphocytic histotypes. In arrhythmia-predominant presentations, EMB may have higher diagnostic and therapeutic yield,[4] and can help refine individualized clinical strategies, for example, by (i) identifying aetiology and guiding immunosuppressive therapy (e.g. giant cell, sarcoidosis, virus-negative autoimmune forms)[1,3]; (ii) clarifying recurrent or atypical disease patterns, including HPGC[1,10,28]; (iii) distinguishing transient oedema from fixed scar when planning ICD, immunosuppression, or ablation[4,8]; and iv) confirming and staging myocarditis in patients in whom CMR cannot be reliably performed.[4,64]

Despite this, in carefully selected cases diagnostic certainty may also be achieved non-invasively through integration of clinical context, imaging, and serology—for example, positive Borrelia serology in Lyme myocarditis.[1]

## Genetic and autoimmune substrates

Some patients with myocarditis and arrhythmias harbour pathogenic variants in genes, such as *DSP*, *FLNC*, *LMNA*.[9–11,26–28] Other common genes are listed in Supplementary material online, *Table S2.2*. While evidence is still preliminary, susceptible genotypes may favour recurrent inflammation, chronic scar formation, and arrhythmias.[10,12,28] The molecular pathways linking genotypes, inflammation, and arrhythmias are still under investigation.[10] As reported in ESC Guidelines, genetic testing is now recommended in multiple conditions, not limiting to patients with familial cardiomyopathy (see Table of Advice 2).[1] Also, the identification of distinct genotypes may significantly contribute to arrhythmic risk stratification.[12]

Immune-genetic background, including human leukocyte antigen,[84,85] circulating autoantibodies,[86,87] intercalated disk abnormalities,[49] and microbiota,[88] may contribute to disease onset and/or outcome.

Supplementary material online, *Table S2.2* and *S2.3* summarizes key genetic and immune-related risk markers relevant to arrhythmogenic progression.

## Multidisciplinary and specialized care

Effective management of arrhythmic myocarditis requires multidisciplinary expertise. ESC-endorsed teams integrating cardiology, imaging, electrophysiology, pathology, infectious diseases, immunology, and genetics provide the infrastructure for accurate diagnosis and precision therapy.[1,12,51] The diagnostic tools in Supplementary material online, *Tables S2.1* and *S2.3* should be integrated into individualized clinical pathways.

# Summary

Identifying the arrhythmogenic substrate in myocarditis is critical for personalized risk stratification and treatment. While EMB confirms the diagnosis and aetiology, complementary tools, such as CMR, rhythm monitoring, EAVM, and genetic testing provide additional insight.[1,89] Structured interpretation of these parameters—summarized in Supplementary material online, *Tables S2.1* through *S2.3*—enables a phase-adapted and evidence-informed diagnostic strategy. As derived by multimodality diagnostic workup, the red flags suggesting specific aetiologies in arrhythmic myocarditis are summarized in Supplementary material online, *Table S2.4*. Early and repeated multimodal evaluation is recommended, especially in high-risk or genetically predisposed patients. This approach should be standard practice in specialized centres managing arrhythmia-prone myocarditis patients.[1] The structured evaluation of inflammation, scar, rhythm, and genetics not only supports diagnostic certainty, but directly informs therapeutic decision-making presented in Chapter 3.

# Chapter 3: pharmacological management of arrhythmias in myocarditis

**How to treat pharmacologically**

This chapter focuses on pharmacological rhythm management in patients with myocarditis and Infl-CMP. Based on the disease stage (hot/hot-to-cold/cold) therapeutic decisions must be guided by arrhythmia type, ventricular function, and genetic background. Procedural and device-based strategies are discussed in subsequent chapters; here, the emphasis lies on tailored pharmacological therapy. *Figure 6* shows clinical decision making based on clinical VT features. In patients with fulminant myocarditis, diagnostic workup and management of acute heart failure, including mechanical circulatory support, should align with the ESC Guidelines.[1]

## Antiarrhythmic therapy in the hot phase

In the hot phase of myocarditis, rhythm control drugs that compromise haemodynamics should be used with caution in decompensated patients.[90] Beta-blockers—especially short-acting intravenous agents like esmolol or landiolol—are first line for VA, provided there is no acute decompensation.[25,91] In electrical storm, non-selective agents such as propranolol may offer additional sympathetic modulation.[91] Amiodarone is the preferred agent for refractory VA in patients with impaired LV function.[1,4] Sotalol may be an alternative in patients with reduced LVEF.[69,92] Lidocaine may be used as short-term bridging therapy in unstable arrhythmias or during flares.[93] Class IC agents (e.g. flecainide or propafenone) are contraindicated in acute heart failure, and should generally be avoided in the setting of acute myocarditis due to potential proarrhythmia.[25,94] However, their use in

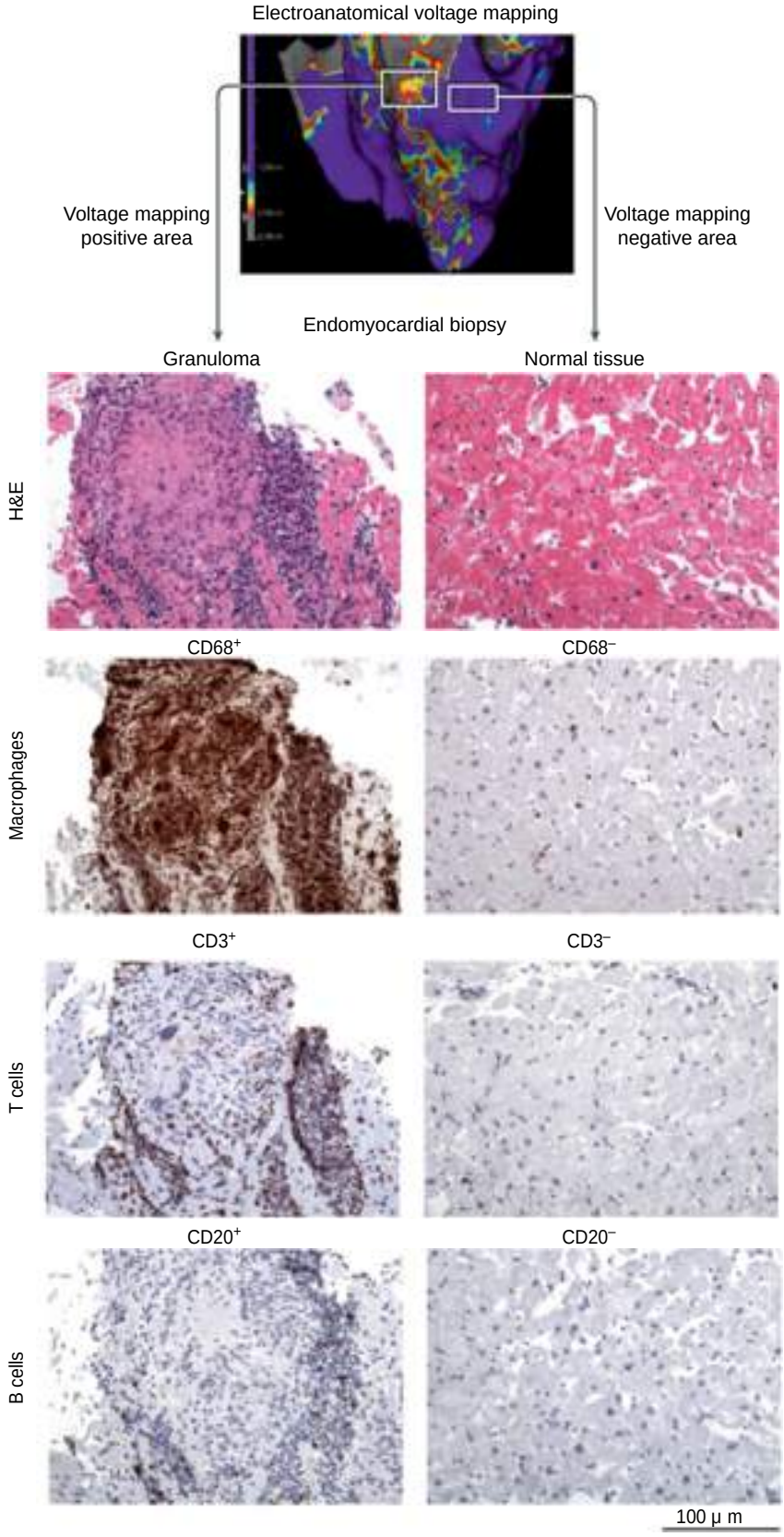


**Figure 5** EAVM-guided EMB. Usefulness of EAVM in guiding EMB in a patient with cardiac sarcoidosis and patchy distribution of substrate abnormalities. Low-voltage areas (endocardial bipolar map, left anterior oblique view; cut-off values 0.5 mV for dense scar—red; 1.5 mV for normal voltage—purple) reveal inflammation and noncaseating granulomas, whereas normal voltage points to healthy tissue. Permissions for Figure obtained by Tschöpe *et al.* 2021.[2] EAVM, electroanatomic voltage mapping; EMB, endomyocardial biopsy.

**Table of Advice 2** Diagnostic work-up

| Advice | Phase | Category of advice | Strength of evidence | Vote (%) | References |
|---|---|---|---|---|---|
| Use CMR with contrast at baseline (T2 and T1, including LGE topology and burden) and schedule repeat CMR at ~ 6 months. | Hot, Hot-to-cold |  |  | 95 | • Schulz-Menger *et al.* 2025[1] |
| Obtain continuous ECG telemetry (possibly 12-lead monitoring) in all patients with myocarditis admitted to the hospital, irrespectively of their clinical presentation. | All |  |  | 85 | • Schulz-Menger *et al.* 2025[1]<br>• Peretto *et al.* 2020[5]<br>• Peretto *et al.* 2021[55] |
| Consider ILR in patients with no indication to ICD/WCD, particularly in the following scenarios:<br>• sporadic/rare symptoms;<br>• asymptomatic arrhythmias;<br>• isolated risk features as NSVT, LVEF <50% at discharge, atypical LGE (extensive, septal/anteroseptal, ring-like), high-risk genotypes. | All |  |  | 85 | • Peretto *et al.* 2021[55] |
| Obtain EMB when results will change management (aetiology definition, immunosuppressive therapy, device/ablation planning) and/or when CMR is inconclusive. | All |  |  | 100 | • Schulz-Menger *et al.* 2025[1]<br>• Caforio *et al.* 2013[3]<br>• Halushka *et al.* 2025[21]<br>• Peretto *et al.* 2020[8] |
| Consider EAVM-guided EMB in patients with patchy disease (i.e. sarcoidosis) or focal myocarditis. | All |  |  | 90 | • Tschoepe *et al.* 2021[2]<br>• Casella *et al.* 2020[76] |
| Obtain EAVM to characterize arrhythmic substrate (LVA topology/burden) in patients with myocarditis requiring invasive EP evaluation. | All |  |  | 95 | • Casella *et al.* 2021[67]<br>• Peretto *et al.* 2020[69] |
| Consider FDG-PET in patients with suspected sarcoidosis; or with suspected myocarditis and limitations to CMR (i.e. implantable device carriers), or with inconclusive results from CMR and EMB. | Hot, Hot-to-cold |  |  | 100 | • Peretto *et al.* 2022[64] |
| Perform genetic testing in patients with:<br>• family history of myocarditis, cardiomyopathy or sudden death<br>• recurrent myocarditis;<br>• severe/exertional arrhythmias;<br>• persistent systolic dysfunction;<br>• atypical LGE (extensive, septal/anteroseptal, ring-like). | All |  |  | 95 | • Schulz-Menger *et al.* 2025[1]<br>• Lota *et al.* 2022[9]<br>• Ammirati *et al.* 2022[28]<br>• Peretto *et al.* 2023[26]<br>• Caforio *et al.* 2025[89] |

CMR, cardiac magnetic resonance; EAVM, electroanatomic voltage map; ECG, electrocardiogram; EMB, endomyocardial biopsy; EP, electrophysiological; ESC, European Society of Cardiology; FDG-PET, 18F-fluorodeoxyglucose positron emission tomography; ICD, implantable cardioverter defibrillator; ILR, implantable loop recorder; LGE, late gadolinium enhancement; LVA, low-voltage areas; LVEF, left ventricular ejection fraction; NSVT, non-sustained ventricular tachycardia; OBS, observational study-based evidence; OPN, expert opinion-based evidence; PV/LPVs, pathogenic/likely-pathogenic variants; RCT, randomized clinical trial-based evidence; VA, ventricular arrhythmias; WCD, wearable cardioverter defibrillator.

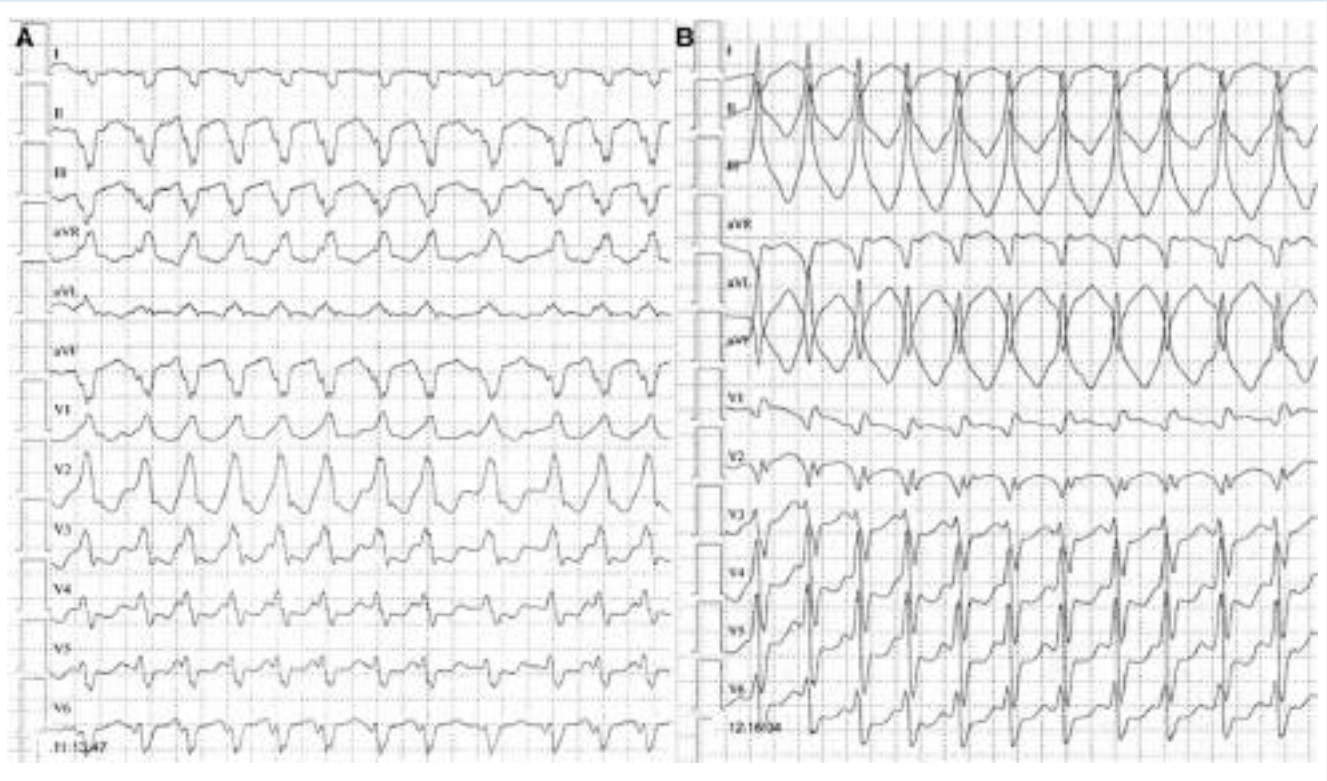


**Figure 6** ECG-guided treatment in arrhythmic myocarditis. ECG features of sustained monomorphic VTs are shown in two patients with myocarditis. (*A*) Right bundle branch block, superior right axis (negative in lead DI) morphology suggests inferolateral LV VT exit, as commonly observed in the cold phase of myocarditis; variability in cycle length suggests hot-to-cold transition phase in a patient with acute presentation (VT) but evolving inflammatory process: immunomodulatory treatment is suggested to target VT before considering ablation ('cool down, then ablate' strategy). (*B*) Regular VT with qR in V1 and RS V4-V6 and inferior axis, suggesting cold-phase and septal origin in a patient with history of cardiac sarcoidosis: immunomodulatory therapy may not help, and referral to an experienced centre for VT ablation is advised. In all cases, ECG provides only partial information. Treatment strategies are driven by the clinical context and gold standard diagnostic techniques for myocarditis (cardiac magnetic resonance, endomyocardial biopsy), LV, left ventricular; VT, ventricular tachycardia.

chronic myocarditis with preserved or mildly reduced LVEF has been proven safe and effective in controlling NSVT/PVC burden.[95] The management of VA in acute heart failure should follow updated expert consensus documents.[77,91,94] For a complete overview of drug profiles, see Supplementary material online, *Table S3.1*. For the use of immunomodulatory therapy during hot/hot-to-cold phases, please see Chapter 3.3.

## Rhythm and substrate control in the cold phase

In the chronic, post-inflammatory stage, monomorphic VTs arising from scar regions become predominant. Beta-blockers remain foundational.[1] Sotalol may be used cautiously in patients with preserved or non-severely reduced LVEF, provided QTc is monitored closely.[5,77] In selected cases—such as post-inflammatory myocarditis with normal or near-normal function and in the absence of extensive LGE—Class IC agents may be considered.[5,77,94]

Importantly, heart failure pharmacotherapy complements arrhythmic management by stabilizing myocardial substrate.[1,25] As recommended by the ESC Guidelines on Heart Failure,[25] early initiation of renin–angiotensin–aldosterone system inhibition, angiotensin receptor–neprilysin inhibitors, mineralocorticoid receptor antagonists, and sodium-glucose cotransporter 2 inhibitors may improve outcomes even in the underinvestigated field of myocarditis/Infl-CMP.[1] See Supplementary material online, *Table S3.2* for phase-adapted heart failure strategies. Monitoring results should be revisited after 3–6 months[1] to reassess the need for device therapy or further procedural interventions.

## Immunomodulatory therapy

To date, the use of immunomodulatory therapy is reserved to patients with EMB-proven diagnosis of myocarditis following a demanding clinical presentation, which often involves heart failure and arrhythmias.[1] In autoimmune, virus-negative myocarditis, immunosuppression is essential to mitigate inflammatory substrate and reduce arrhythmia burden.[3] Steroids associated with azathioprine or mycophenolate mofetil form the standard first-line approach (usually minimum 6 for up to 12–24 months);[96,97] in GCM, triple immunosuppressive therapy is indicated. Immunosuppressive therapy may indirectly stabilize the arrhythmogenic substrate during the hot/hot-to-cold phases, and contribute to lower VA burden in the long term.[98–100] The use of anakinra and other biologicals in arrhythmic myocarditis

is still investigational.[26,99,101] Therapeutic agents and indications are summarized in Supplementary material online, *Table S3.3*. Indications should follow existing myocarditis and Infl-CMP guidelines.[1] Electrical storm may require i.v. immunosuppressive therapy in fulminant myocarditis,[93] provided ongoing infections are excluded. Consistent with ESC 2025 guidelines,[1] immunosuppression in virus-negative disease is biopsy-guided and managed by dedicated multidisciplinary teams.[51] Pericarditis-directed therapy in IMPS include non-steroidal anti-inflammatory drugs and colchicine according to the current ESC recommendations.[1]

### Special clinical scenarios

In pregnancy, arrhythmia treatment must account for foetal safety.[1] Among beta-blockers, propranolol and labetalol are safe; however, to improve the control of arrhythmias the cautious use of other agents (e.g. metoprolol, sotalol) is allowed.[94,102,103] Amiodarone should be avoided during the first trimester.[91,98] In life-threatening arrhythmias, lidocaine or procainamide may be used.[94] Multidisciplinary counselling is advised, especially in cases mimicking peripartum cardiomyopathy,[104] to guide both therapy and genetic evaluation.[12] Genetic predisposition, Chagas cardiomyopathy or ICI-associated myocarditis require tailored strategies.[1] In Chagas disease, ablation is frequently needed because of drug-refractory VA.[33] In ICI myocarditis, drug withdrawal is part of the treatment and should be discussed within a multidisciplinary team.[1] In HPGC preliminary data support the use of immunosuppressive therapy[26,105]; however, treatment type and duration are still to be defined. When diagnostic resources or aetiology-specific treatments are unavailable, referral to specialized tertiary centres is strongly advised.[1]

## Summary

Pharmacological therapy in myocarditis must be stratified by disease phase and arrhythmia mechanism. Antiarrhythmic drugs, heart failure agents, and immunomodulatory therapies work synergistically to control rhythm, modulate substrate, and stabilize long-term risk. Supplementary material online, *Tables S3.1* through *S3.3* provide a structured overview for phase-appropriate therapy selection. Device and procedural strategies are addressed in detail in the following chapters.

# Chapter 4: provocative tests in arrhythmic myocarditis

**When should we go invasive**

The decision to perform a provocative test in arrhythmic myocarditis depends on disease phase, symptom severity, and diagnostic uncertainty. This chapter discusses indications for conventional and cardiopulmonary exercise stress test, and invasive electrophysiological studies. Supplementary material online, *Table S4.1* summarizes procedural indications by clinical context.

### Functional testing

Functional testing supports longitudinal risk stratification in clinically stable patients. Cardiopulmonary exercise test (CPET) may reveal reduced functional reserve or delayed oxygen kinetics, indicating subclinical inflammation or early heart failure. In clinically stable patients, exercise stress testing (EST) may be useful ≥3–6 months after active inflammation to detect exertional arrhythmias or electrical instability.[106] EST has a relevant role in allowing safe readmission to sport in athletes.[1,107] See Supplementary material online, *Table S4.1* for test modalities, indicative findings, and clinical relevance.

### Programmed ventricular stimulation

Invasive programmed ventricular stimulation (PVS) may have a role in stratifying arrhythmic risk of myocarditis and guiding treatment decisions.[1] Indeed, PVS can identify individuals at higher risk of life-threatening VA, particularly when indication to ICD for primary prevention is uncertain. The evidence in support of PVS is stronger in cardiac sarcoidosis[108] than in unspecific myocarditis. Per expert consensus, PVS may find application in selected patients with myocarditis, as in those with either intermediate risk of SCD, unexplained syncope, or indication to pacemakers in the presence of concurrent risk factors for VA. Overall, the technique was shown to be more reliable when used in the cold stage of myocarditis.[104] Instead, the use of early PVS during active myocarditis is discouraged.[1,109] Finally, either invasive or non-invasive PVS can be used in candidates to ablation to capture the morphology of the clinical VT by 12-lead ECG recording.[68]

### Other provocative testing

Provocative pharmacologic testing—such as ajmaline, flecainide, or procainamide challenge—may unmask latent Brugada syndrome or conduction defects in ambiguous cases.[46,77,110] Tilt-table or autonomic stress testing may clarify syncope mechanism and help avoid unnecessary device therapy in patients with vagal prodromes.[77] However, results should be interpreted with caution, as arrhythmias remain a major cause of syncope in patients with myocarditis.[1,4] In general, these tests are reserved for selected patients and should only be performed when the result will directly inform management decisions. Such findings may inform decisions on ICD indication, ablation candidacy, or activity counselling.

## Summary

Non-invasive functional tests such as CPET and EST are informative in clinically stable patients late after the acute phase of myocarditis (cold phase). Invasive PVS may be useful to guide arrhythmic risk prediction and ablation strategy, especially in patients with cold phase myocarditis. Comprehensive risk stratification should integrate imaging, histological findings, genetic information, and clinical parameters.[1]

# Chapter 5: device strategies in arrhythmic myocarditis

**When is a device indicated**

Device therapy in myocarditis and Infl-CMP must be personalized, based on disease phase, arrhythmia type, LVEF, genetic predisposition, and the extent of myocardial scar. The challenge lies in balancing early protection against sudden cardiac death with the risks of overtreatment in a potentially reversible condition. This chapter provides advice on ICD, cardiac resynchronization therapy (CRT), pacemaker, wearable cardioverter-defibrillator (WCD), and conduction system pacing (CSP), integrating substrate, rhythm burden, and recovery trajectory. Indications by disease phase and substrate type are summarized

**Table of Advice 3** Immunomodulation and antiarrhythmic drug therapy

| Advice | Phase | Category of advice | Strength of evidence | Vote (%) | References |
|---|---|---|---|---|---|
| In patients with non-infectious myocarditis, initiate EMB-guided immunosuppression in demanding clinical presentations (mainly heart failure and arrhythmias) | Hot, hot-to-cold |  |  | 100 | • De Luca *et al.* 2020[96]<br>• Frustaci *et al.* 2009[97]<br>• Caforio *et al.* 2024[98]<br>• Peretto *et al.* 2020[99]<br>• Lakkireddy *et al.* 2019[100] |
| In EMB-proven non-infectious myocarditis, favour steroid-sparing agents (AZA/MMF; MTX in sarcoidosis), ensure regular multidisciplinary surveillance, and tailor treatment duration (6–24 months) to clinical endpoints. | Hot, hot-to-cold |  |  | 100 | • Caforio *et al.* 201[33]<br>• Caforio *et al.* 2024[98]<br>• Peretto *et al.* 2020[99] |
| Manage non-lymphocytic myocarditis (eosinophilic, giant cell, sarcoidosis) according to updated guidelines. Consult an infectious disease specialist for viral/bacterial/protozoal myocarditis. Withdraw immune checkpoint inhibitor (ICI) or other toxic agents upon multidisciplinary risk/benefit evaluation, and start specific therapy if needed. | Hot, hot-to-cold |  |  | 100 | • Schulz-Menger *et al.* 202[51]<br>• Caforio *et al.* 2024[98] |
| Manage acute-phase electrical storms according to the updated protocols, and consider using i.v. immunosuppressive agents after excluding infections. | Hot |  |  | 95 | • Lenarczyk *et al.* 2024[93] |
| Use beta-blockers in patients with any stage of myocarditis to treat or prevent arrhythmias, unless contraindicated. | All |  |  | 90 | • McDonagh *et al.* 2021[25]<br>• Merino *et al.* 2025[94] |
| In patients with arrhythmic myocarditis and limitations/unsatisfactory response to betablockers, use:<br>• amiodarone (or sotalol outside the acute phase and in the absence of decompensated heart failure) if LVEF is < 40%;<br>• class IC agents (i.e. flecainide, propafenone) if LVEF is ≥40% (in selected cases, outside the acute phase and in the absence of decompensated heart failure). | All |  |  | 100 | • McDonagh *et al.* 2021[25]<br>• Zeppenfeld *et al.*[74]<br>• Connolly *et al.* 2006[92]<br>• Merino *et al.* 2025[94]<br>• Peretto *et al.* 2026[95] |
| Use renin–angiotensin–aldosterone system inhibitors, angiotensin receptor–neprilysin inhibitors (ARNIs), mineralocorticoid receptor antagonists, and sodium-glucose cotransporter 2 inhibitors, according to the recommendations of the ESC Guidelines on Heart Failure. | All |  |  | 100 | • McDonagh *et al.* 2021[25]<br>• Merino *et al.* 2025[94] |

AZA, azathioprine; EMB, endomyocardial biopsy; ESC, European Society of Cardiology; LVEF, left ventricular ejection fraction; MMF, mofetil mycophenolate; MTX, methotrexate; OBS, observational study-based evidence; OPN, expert opinion-based evidence; RCT, randomized clinical trial-based evidence; VA, ventricular arrhythmias.

in Supplementary material online, *Table S5.1*. We anchor device decisions to the ESC categories of acute and non-active myocarditis,[1] and refine these with our phase model by recognizing a hot-to-cold transition, during which arrhythmic risk may remain elevated despite decreasing T2 signal. Arrhythmic risk stratification should align with the phase-aware myocarditis pathway in patients with perimyocarditis or other arrhythmic risk features (see below). Instead, in myopericarditis with preserved LVEF and no VA, WCD/ICD is generally not indicated.[1]

## Risk stratification for device therapy

Conventional device indications based solely on LVEF are insufficient in myocarditis. In line with ESC Heart Failure Guidelines 2021,[25] arrhythmic risk stratification should integrate clinical features with CMR-based scar assessment, particularly the extent and distribution of LGE. Septal/anteroseptal and ring-like patterns, or extensive LGE, are associated with higher arrhythmic risk and adverse outcomes.[6,13,53,67] When combined with high-risk genotypes (e.g. *LMNA*, *DSP*, *FLNC*), these findings inform early ICD consideration even in patients with preserved or only mildly reduced LVEF.[6,12,13] Careful consideration for ICD is needed also for patients with non-lymphocytic histotypes of myocarditis, in particular cardiac sarcoidosis and GCM.[1,50] Conversely, transient VA, QT prolongation or reversible AV block in acute myocarditis may be cytokine-mediated,[7,48] and not require permanent device therapy.[33] Finally, the hot-to-cold transition phase represents a key arrhythmogenic window; close arrhythmia monitoring by Holter ECGs or ILRs may help detecting scar-related VA[55]; additional factors, as worsening systolic function/failure to normalize LVEF,[6,111,112] expanding LGE,[41,113] high-risk scar patterns[13,60] or malignant genotypes,[13,28] may act as risk modifiers calling for an early ICD implant (Table of Advice 5). Supplementary material online, *Table S5.1* outlines key clinical scenarios.

## Choosing the right device

The choice of device must reflect clinical trajectory, pacing needs, and arrhythmic profile. Transvenous ICDs are generally preferred when pacing is needed or anti-tachycardia pacing (ATP) is desirable.[77] In younger patients or those with vascular access issues, a subcutaneous (S-)ICD offers effective protection against sudden cardiac death without the risks associated with transvenous leads.[114] In this setting, extravascular (EV-)ICD may offer a valuable alternative in patients with unsuitable anatomy for S-ICD and suffering from tolerated monomorphic VTs potentially responsive to ATP.[115] Where permanent pacing/CRT becomes necessary, we align with the ESC/EHRA 2025 CSP consensus.[116] In detail, CRT should be considered in patients with LVEF $\leq$35%, QRS duration $\geq$150 ms, and left bundle branch block (LBBB) morphology, particularly in chronic stage.[24] We acknowledge CSP in patients (i) who meet CRT criteria but are non-responders or in whom conventional CRT is unfeasible[117]; (ii) with indication to a permanent pacemaker, when a high ventricular pacing burden (>20%) is anticipated,[116] especially in the presence of a low septal scar burden[118]; and iii) after AV node ablation (very uncommon in myocarditis), where LBB area pacing (LBBAP) is commonly preferred over conventional pacing.[116] These CSP choices sit alongside our myocarditis pathway and do not change phase-aware device timing. Device selection strategies are summarized in Supplementary material online, *Table S5.2* and *Figure 7*.

## Temporary protection and device deferral

In the acute setting, according to the ESC 2025 Guidelines on Myocarditis and Pericarditis, ICD may be appropriate for the secondary prevention of SCD;[1] we therefore favour WCD bridging during hot phase or early convalescence[54,119,120] and defer permanent ICD to the cold stage when indicated. ICD implantation during the hot or hot-to-cold phases is advised only in selected cases,[1] including monomorphic VTs and additional risk features presented in Table of Advice 5 (Consensus grading: OBS/OPN, Task Force agreement 95%).[6,31,53,55,67,109,121–123] ILRs may support long-term risk stratification in uncertain cases.[55] Importantly, transient AV block—such as in Lyme myocarditis or toxic myocarditis—usually resolves with appropriate treatment, and temporary pacing (preferentially transvenous with active fixation) may suffice.[32,77] In all patients with acute myocarditis and no compelling indication to ICD, re-evaluation at 3 to 6 months should guide definitive therapy.[1] The same considerations apply to primary prevention, where ICD may be avoided during the hot phase (and reassessment during follow-up is advised to test the response of LVEF, inflammatory substrate and arrhythmic burden to optimal cardiac therapy or immunosuppression.[1,97] A deferred implantation strategy may help preventing delayed wound healing in immunosuppression receivers. Finally, WCD may be also an option for intermediate-risk patients who are waiting for the result of genetic test. Instead, other conditions may call for early ICD implant (see Table of Advice 5), as occurs in patients with cardiac sarcoidosis or GCM even when presentation is AV block.[1,77,124]

## Genetic substrates and ICD timing

Pathogenic/likely-pathogenic variants in cardiomyopathy-associated high-risk genes are linked to conduction disease, myocardial fibrosis, and early VA.[12] In such cases, ICD implantation for primary prevention is reasonable even with preserved or moderately reduced LVEF in the presence of additional risk factors. For gene-directed arrhythmic risk stratification, both ESC Guidelines and specific risk scores are available.[125–127] While genotype is not a stand-alone indication to ICD, particular attention should be paid to patients with familial sudden cardiac death, NSVT, or atypical LGE patterns on CMR.[12,13] Remote monitoring—either through ILR or device-based telemetry—supports early detection of dynamic arrhythmic risk in this population. Genetic counselling is advised for patients and potentially affected family members.[12]

## Programming, follow-up, and reassessment

Device programming should aim to minimize inappropriate therapies, prioritize ATP, and allow individualized detection zones.[128,129] Remote monitoring enables early recognition of arrhythmic patterns, alerts for sustained VT, and documentation of asymptomatic/subclinical events.[128] This is especially valuable in patients with evolving substrate (e.g. WCD carriers during the hot-to-cold transition phase) or risk features persisting beyond the acute phase (e.g. carriers of pathogenic variants in cardiomyopathy-associated genes). Routine device follow-up should include arrhythmia burden analysis and periodic reassessment of indication.[1] In those with recovery of LVEF, regression/clearance of LGE and low VA burden, downgrading or explantation may be considered after careful evaluation of risks and benefit (expert consensus). These aspects are addressed in more detail in Chapter 7.

**Table of Advice 4** Provocative testing

| Advice | Phase | Category of advice | Strength of evidence | Vote (%) | References |
|---|---|---|---|---|---|
| Perform symptom-limited exercise testing (± CPET) before return-to-play to exclude exertion-triggered VA. | Hot-to-cold, cold | ✓ | | 90 | • Schulz-Menger *et al.* 2025[1]<br>• Peretto *et al.* 2022[106] |
| Use PVS in cold phase for risk refinement or ablation planning. | Cold | ✓ | | 80 | • Cronin *et al.* 2019[68]<br>• Zeppenfeld *et al.* 2022[77]<br>• Peretto *et al.* 2020[109] |
| Avoid EST and PVS during the acute phase of myocarditis. | Hot | ✗ | | 95 | • Schulz-Menger *et al.* 2025[1]<br>• Peretto *et al.* 2020[109] |

CPET, cardiopulmonary exercise test; EST, exercise stress test; OBS, observational study-based evidence; OPN, expert opinion-based evidence; PVS, programmed ventricular stimulation; VA, ventricular arrhythmias.

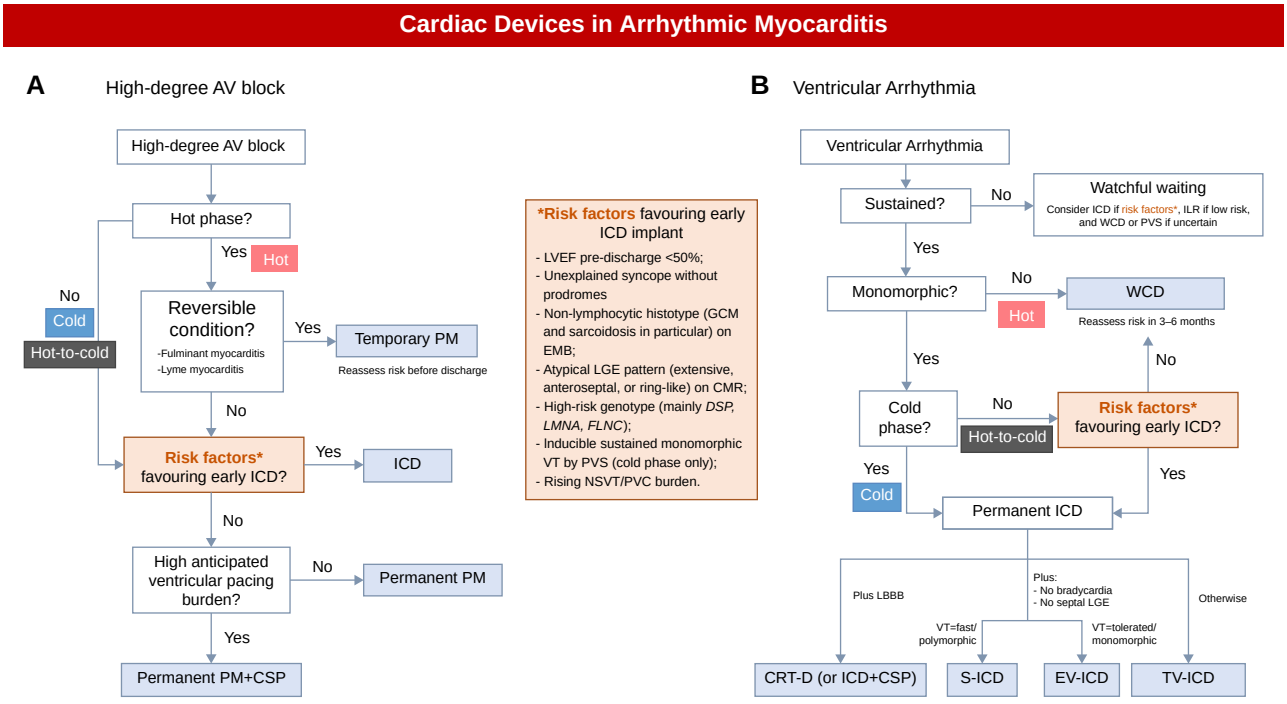


**Figure 7** Cardiac devices in arrhythmic myocarditis. Algorithms to guide the choice of cardiac devices in arrhythmic myocarditis are shown, for high-degree AV block (panel *A*) and VA (panel *B*) scenarios. Where relevant, myocarditis phases (hot/hot-to-cold/cold) are shown. AV, atrioventricular; CMR, cardiac magnetic resonance; CRT, cardiac resynchronization therapy; CSP, conduction system pacing; *DSP*, desmoplakin gene; EMB, endomyocardial biopsy; *FLNC*, filamin C gene; GCM, giant cell myocarditis; ICD, implantable cardioverter defibrillator (EV–, extravascular; S–, subcutaneous; TV–, transvenous); LBBB, left bundle branch block; LGE, late gadolinium enhancement; *LMNA*, lamin A/C gene; LVEF, left ventricular ejection fraction; NSVT, nonsustained ventricular tachycardia; PM, pacemaker; PVS, programmed ventricular stimulation; VA, ventricular arrhythmias; VT, ventricular tachycardia; WCD, wearable cardioverter defibrillator.

## Summary

Device therapy in myocarditis and Infl-CMP must be guided by disease phase, arrhythmic risk profile, structural recovery, and underlying aetiology.[1] ICDs are essential for secondary prevention and should also be considered for primary prevention in patients with high-risk genotypes, even with preserved LVEF. The recognition of a 'hot-to-cold' transition phase provides a critical framework underscoring the need for extended surveillance and individualized ICD strategies in patients with sustained monomorphic VT and/or reduced LVEF at discharge. CRT is indicated in patients with LBBB and LV dysfunction, while CSP offers a viable alternative in selected non-responders with a high anticipated ventricular pacing burden.[111] WCD, ILR, and remote monitoring allow for individualized risk adaptation, particularly in early or evolving disease. Proper device selection, thoughtful programming, and structured follow-up are key to optimizing outcomes and avoiding overtreatment. Multidisciplinary collaboration—including heart failure specialists, electrophysiologists, geneticists, and imaging experts—is crucial to tailor decisions over time.[1] Supplementary material online, *Tables S5.1* and *S5.2* support structured, phase-specific decision-making within an integrated, rhythm-focused care model.[4]

# Chapter 6: electrophysiological mapping and ablation in arrhythmic myocarditis

**How to control rhythm invasively**

Catheter ablation is a cold/non-acute phase intervention targeting scar-related arrhythmias; it is not advised during active inflammation. The 'cool down, then ablate' principle applies in intermediate cases, such as during the hot-to-cold transition phase.[6,8,69] Indications to catheter ablation of cardiac arrhythmias in myocarditis are in turn phase-sensitive, and more commonly apply to myocarditis without associated pericarditis.[1] As a general rule, ablation does not replace the role of ICD in preventing sudden arrhythmic death. This chapter reviews the roles of advanced mapping, ablation techniques, and supportive strategies in managing arrhythmic myocarditis across disease phases, with a focus on timing, substrate specificity, and safety. Supplementary material online, *Table S6.1* summarizes procedural indications by clinical context. *Figure 8* puts ablation in the right clinical frame, whereas *Figure 9* shows representative findings during VT ablation.

## Electroanatomical mapping: applications and techniques

Consistently with the deep or subepicardial localization of the scar, most patients with myocarditis require an epicardial/endoepicardial approach for VT ablation.[69,130–132] In most cases, the optimal access is guided by both scar localization and 12-lead VT morphology. As described in Chapter 4, bipolar and unipolar EAVM allows identification of arrhythmogenic scar substrates.[70,133,134] Mapping during sinus rhythm or ventricular pacing allows annotation of late potentials (LPs) and local abnormal ventricular activities (LAVAs), which serve as surrogate markers for re-entry circuits and primary ablation targets.[68] (see Supplementary material online, *Table S6.1*). Advanced tools such as high-density mapping, multipolar catheters, steerable sheaths, and contact-force sensing improve procedure accuracy. Epicardial access requires dedicated expertise.[68,69] Innovative mapping methods such as ECGi and signal complexity analysis are under evaluation.[135]

## Catheter ablation of ventricular arrhythmias

Catheter ablation is primarily indicated in the cold phase, when inflammation has resolved and VTs are scar-driven.[6,69] Inducible and tolerated VTs can be mapped and terminated by energy delivery at critical isthmus to exit sites.[136] Substrate-based ablation targeting LPs and LAVAs is preferred, especially when VT is non-inducible.[68] CMR, and in selected cases contrast-enhanced CT, may help identify potential slow conducting channels and guide ablation.[137,138] AI-supported mapping and advanced signal processing tools may help identifying suitable targets for ablation.[135] At present, procedural endpoints include non-inducibility and elimination of abnormal potentials.[68,69] Supplementary material online, *Table S6.2* provides a structured overview of mapping and ablation tools in the context of arrhythmic myocarditis.

Ablation during the hot phase is discouraged except in electrical storm refractory to medical therapy and supportive care.[68] Outcomes after VT ablation post-myocarditis are generally favourable,[139] except for cases with active myocardial inflammation or septal involvement (e.g. sarcoidosis, HPGC).[69,140] Disease-phase suitability and procedural timing are outlined in Supplementary material online, *Table S6.3*.

## VT ablation in specific myocarditis subtypes

In patients with sustained monomorphic VT and underlying Infl-CMP, catheter ablation is a reasonable option in the cold phase, when pharmacological therapy fails or when ICD shocks occur. ESC and HRS consensus documents endorse substrate-guided ablation in the setting of electrical storm, recurrent ICD therapies, or intolerable antiarrhythmic side effects.[1,68,77] In lymphocytic myocarditis, ablation is rarely needed unless arrhythmias persist post-inflammation. Sarcoidosis and GCM often affect the septum and cause re-entrant VT, making ablation necessary.[140,141] In Chagas disease, diffuse epicardial scar mandates a combined endoepicardial approach.[142] In HPGC (e.g. *DSP*), ablation may need to target extensive/biventricular substrates[143] and should be delayed until inflammation subsides. These distinctions are reflected in Supplementary material online, *Table S6.3*.

## Supportive strategies in ablation context

In patients with electrical storm or unstable VT/VF, external overdrive pacing and shocks, autonomic modulation (e.g. stellate ganglion block) and sedation may stabilize rhythm.[91] As a rescue strategy, ablation finds limited applications in patients with refractory electrical storms or fulminant myocarditis. In this setting, mechanical circulatory support (e.g. veno-arterial extracorporeal membrane oxygenator -VA-ECMO-, Impella) may enable safe mapping and ablation of life-threatening VA.[141,144] These high-risk procedures should be performed in experienced centres.[1,77] See Supplementary material online, *Table S6.2* for procedural endpoints and supportive considerations.

## Ablation of supraventricular arrhythmias

Supraventricular arrhythmias (e.g. atrial fibrillation, atypical flutter, atrial tachycardia) may complicate myocarditis.[145] Ablation should only be considered in the cold phase.[146–148] In systemic

**Table of Advice 5** Device therapy

| Advice | Phase | Category of advice | Strength of evidence | Vote (%) | References |
|---|---|---|---|---|---|
| Device indication | | | | | |
| In acute myocarditis, use temporary pacing (preferentially transvenous) to treat high-degree AV block. | Hot |  |  | 100 | • Schulz-Menger *et al.* 2025[1] |
| In acute myocarditis, prefer WCD (life vest) bridging and reassess indication to ICD after 3–6 months. | Hot, Hot-to-Cold |  |  | 95 | • Schulz-Menger *et al.* 2025[1] |
| In cold phase myocarditis, implant ICD in patients with sustained monomorphic VT, especially if haemodynamically not-tolerated (secondary prevention). | Cold |  |  | 100 | • Schulz-Menger *et al.* 2025[1] |
| In cold phase myocarditis, implant ICD in patients with LVEF<35% in spite of optimal treatment for 3 months (primary prevention). | Cold |  |  | 100 | • Arbelo *et al.* 2023[12] |
| In uncertain cases, evaluate the following risk factors favouring early ICD implant over WCD, especially if presenting in combination.<br>Secondary prevention<br>• Sustained monomorphic VT.<br>Both primary and secondary prevention<br>• LVEF pre-discharge<50%;<br>• Unexplained syncope without prodromes<br>• Non-lymphocytic histotype (GCM and sarcoidosis in particular);<br>• Atypical and quantitatively significant LGE pattern (extensive, septal/anteroseptal, or ring-like);<br>• High-risk genotype (mainly *DSP*, *LMNA*, *FLNC*);<br>• Inducible sustained monomorphic VT by PVS (cold phase only);<br>• Rising NSVT/PVC burden. | All |  |  | 95 | • Schulz-Menger *et al.* 2025[1]<br>• Rav-Acha *et al.* 2024[6]<br>• Peretto *et al.* 2025[13]<br>• Ammirati *et al.* 2019[50]<br>• Gentile *et al.* 2021[53]<br>• Rosier *et al.* 2020[122] |
| Special aetiologies | | | | | |
| In Lyme myocarditis, avoid a permanent pacemaker unless high-degree AV block is persistent (despite antibiotic treatment for 2–3 weeks) or late-onset (>3–6 months). | Hot, Hot-to-Cold |  |  | 100 | • Schulz-Menger *et al.* 202[51] |

*Continued*

**Table of Advice 5** ***Continued***

| Advice | Phase | Category of advice | Strength of evidence | Vote (%) | References |
|---|---|---|---|---|---|
| In cardiac sarcoidosis, GCM, or HPGC presenting with high-degree AV block, favour ICD over pacemaker implant. | Hot, Hot-to-Cold |  |  | 90 | • Schulz-Menger *et al.* 2025[1]<br>• Arbelo *et al.* 2023[12] |
| In HPGC, estimate genotype (mainly *LMNA*, *DSP*, *FLNC*) as a risk modifier, not as a stand-alone indication to ICD. | All |  |  | 100 | • Arbelo *et al.* 2023[12]<br>• Peretto *et al.* 2025[13] |
| Device choice | | | | | |
| Favour transvenous over subcutaneous ICD (S-ICD) in the following scenarios:<br>• need for pacing/symptomatic bradycardia;<br>• risk of AV block (GCM, sarcoidosis, high-risk genotype as *LMNA*, septal LGE);<br>• non-fast, tolerated VT, responsive to ATP (case-by-case decision; here, extravascular ICD is an alternative). | Hot-to-cold, cold |  |  | 100 | • Markman *et al.* 2025[115]<br>• Wilkoff *et al.* 2016[127] |
| When transvenous ICD is indicated, prefer dual-chamber devices in the following scenarios:<br>• bradycardia (to avoid unnecessary ventricular pacing and/or preserve AV synchrony);<br>• supraventricular tachyarrhythmias (to improve VT/VF discrimination);<br>• high-burden PVCs (to favour atrial overdrive pacing in selected refractory cases). | Hot-to-cold, cold |  |  | 95 | • Wilkoff *et al.* 2016[128]<br>• Fadahunsi *et al.* 2015[129] |
| Favour PM implant, only after carefully excluding the risk factors favouring early ICD (see above). | Hot-to-cold, cold |  |  | 100 | • Wilkoff *et al.* 2016[128] |
| Align with ESC Guidelines on Heart Failure for indications to CRT after the acute phase. | Hot-to-cold, cold |  |  | 100 | • McDonagh *et al.* 2021[25] |
| Use CSP in patients with an anticipated ventricular pacing burden >20%, as well as in cases who cannot benefit from CRT. | Hot-to-cold, cold |  |  | 85 | • Glikson *et al.* 2025[116] |

AV, atrioventricular; ATP, anti-tachycardia pacing; CSP, conduction system pacing; CRT, cardiac resynchronization therapy; *DSP*, desmoplakin gene; ESC, European Society of Cardiology; *FLNC*, filamin C gene; GCM, giant cell myocarditis; HPGC, hot-phase genetic cardiomyopathy; ICD, implantable cardioverter defibrillator; LGE, late gadolinium enhancement; *LMNA*, lamin A/C gene; LV, left ventricular; LVEF, left ventricular ejection fraction; OBS, observational study-based evidence; OPN, expert opinion-based evidence; PM, pacemaker; PVC, premature ventricular complex; PV/LPVs, pathogenic/likely-pathogenic variants; PVS, programmed ventricular stimulation; VA, ventricular arrhythmias; VF, ventricular fibrillation; VT, ventricular tachycardia; WCD, wearable cardioverter defibrillator.

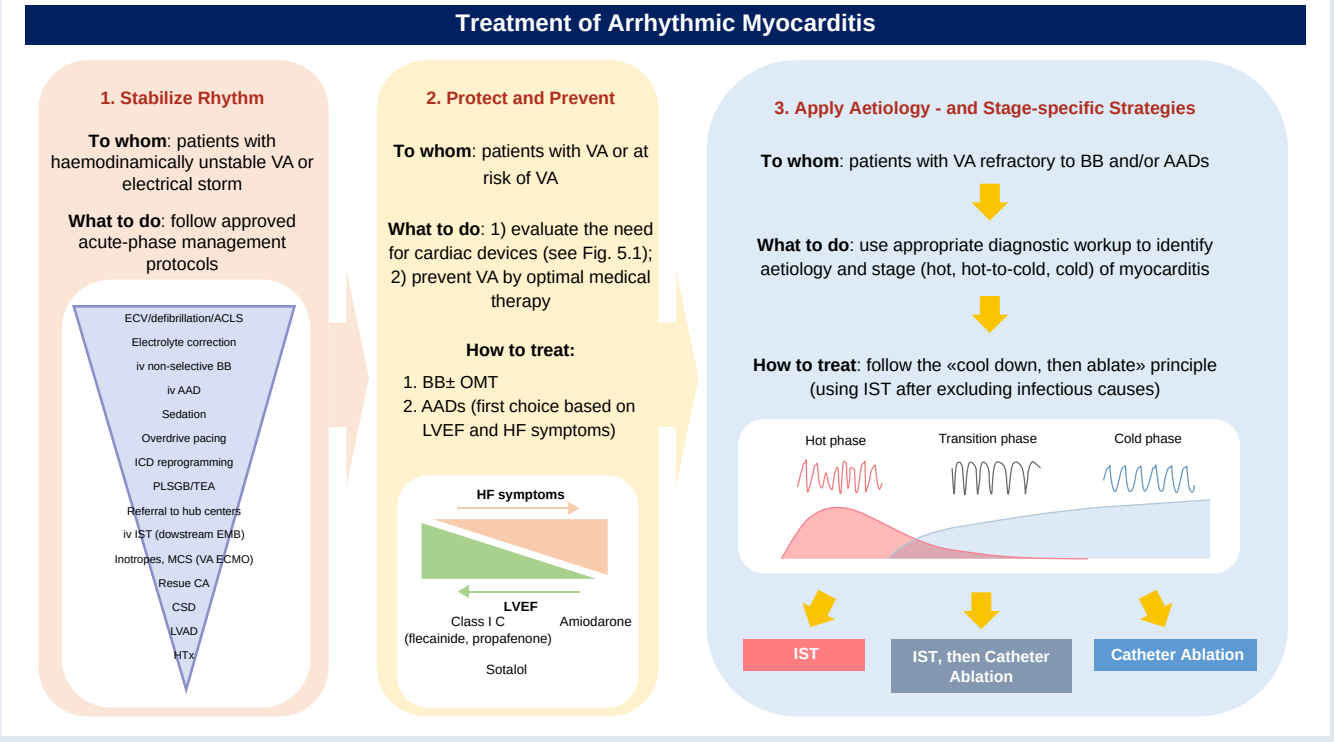


**Figure 8** Treatment of arrhythmic myocarditis. Treatment principles are shown for arrhythmic myocarditis, ranging from rhythm stabilization to cardiologic and aetiology-/phase-specific treatment. AADs, antiarrhythmic drugs; ACLS, advanced cardiovascular life support; BB, betablockers; CSD, cardia sympathetic denervation; ECV, electrical cardioversion; HF, heart failure; HTx, heart transplant; ICD, implantable cardioverter defibrillator; IST, immunosuppressive therapy; iv, intravenous; LVAD, left ventricular assist device; LVEF, left ventricular ejection fraction; MCS, mechanical circulatory support; OMT, optimal medical therapy (guideline-directed); PLSGB, percutaneous left stellate ganglion blockade; TEA, thoracic epidural anaesthesia; VA, ventricular arrhythmias; VA-ECMO, venoarterial extracorporeal membrane oxygenator.

inflammatory diseases (e.g. sarcoidosis), coordination with inflammation control is advised.[149]

If AV node ablation is part of rate control, the 'cool down, then ablate' principle still applies; post-ablation pacing can follow the CSP consensus (LBBAP commonly preferred; His bundle pacing feasible with attention to thresholds and a possible back-up lead).[116] Even in this setting, it is advised to carefully evaluate the risk factors favouring the choice of ICDs over pacemakers (Table of Advice 5).

## Summary

Invasive strategies in arrhythmic myocarditis must be precisely timed and substrate-adapted. Catheter ablation is strongly advised in patients with scar-related VT during the cold phase, where outcomes are favourable and substrate clearly defined.[8,69] Here, EAVM serves both interventional and diagnostic purposes, especially when imaging is inconclusive or EMB targeting is needed. Disease-specific patterns—as seen in sarcoidosis, Chagas cardiomyopathy, or *DSP*-associated inflammation—require tailored approaches. Life-threatening arrhythmias in the hot phase demand stabilization prior to ablation. Invasive procedures should be performed in experienced centres under multidisciplinary guidance. The ESC 2025 guidelines are non-prescriptive for ablation in active disease[1]; our 'cool down, then ablate' approach is consensus-based and safety-oriented. See Supplementary material online, *Tables S6.1–S6.3* and Table of Advice 6 for detailed advices to support the clinical decision-making.

## Chapter 7: surveillance—follow-up and long-term monitoring

**How closely and how long to monitor effectively therapy**

Long-term rhythm surveillance in myocarditis requires a structured, risk-adapted strategy that reflects the dynamic course of inflammation, scar evolution, and arrhythmic progression. This chapter provides phase-specific advices for follow-up intervals, remote monitoring, pharmacologic therapy reassessment, and device downgrading. Supplementary material online, *Tables S7.1–S7.4* summarize key indicators and interventions.

### Structured follow-up and rhythm monitoring

According to the ESC Guidelines,[1,6,12,25,77] longitudinal follow-up should include repeat assessment of rhythm status, mechanical function, and fibrosis burden. Follow-up intervals typically include 3, 6, and 12 months (see Supplementary material online, *Table S7.1*), depending on disease activity and clinical course.[1] In patients with recent myocarditis, restriction from

**Table of Advice 6** Catheter ablation and EP procedural strategy

| Advice | Phase | Category of evidence | Strength of evidence | Vote (%) | References |
|---|---|---|---|---|---|
| Patient selection and Timing | | | | | |
| Follow the 'cool down, then ablate' principle, and postpone VT ablation to the cold phase of myocarditis to target monomorphic VTs. | Cold |  |  | 100 | • Peretto *et al.* 2025[8]<br>• Peretto *et al.* 2020[69] |
| Avoid VT ablation in acute/fulminant myocarditis, unless it is needed as a rescue procedure for refractory electrical storms. | Hot |  |  | 100 | • Peretto *et al.* 2025[8]<br>• Lenarczyk *et al.* 2024[93] |
| Prefer ablation in patients with occurrence of VT during the cold phase, despite optimal medical treatment including betablockers and/or antiarrhythmics. | Cold |  |  | 100 | • Peretto *et al.* 2025[8]<br>• Peretto *et al.* 2020[69] |
| The use of VT ablation in replacement for ICD is not advised as a standard practice. | Cold |  |  | 100 | • Peretto *et al.* 2025[8]<br>• Zeppenfeld *et al.* 2022[77] |
| Prefer PVC ablation in patients with monomorphic high-burden ectopy causing symptoms or PVC-induced cardiomyopathy, when medical treatment is not effective, not tolerated, or not desired. | Cold |  |  | 100 | • Cronin *et al.* 2019[68] |
| Avoid ablation in patients with polymorphic PVCs during active myocarditis, and prefer immunosuppression as part of medical treatment instead. | Hot, hot-to-cold |  |  | 100 | • Lakkireddy *et al.* 2019<span dataode-nwrap= 100 |
| Prefer ablation of atrial fibrillation in patients with symptomatic and drug-refractory arrhythmia during the chronic stage of myocarditis. | Cold |  |  | 100 | • Weng *et al.* 2020[149] |
| Avoid ablation in patients with acute-phase atrial fibrillation, and prefer immunosuppression as part of medical treatment instead. | Hot, hot-to-cold |  |  | 95 | • Peretto *et al.* 2025[8]<br>• Peretto *et al.* 2020[99] |
| Techniques for VT ablation | | | | | |
| Use endo-epicardial approach in patients with myocarditis, especially when VT morphology and/or imaging identifies an epicardial or deep substrate. | Cold |  |  | 95 | • Peretto *et al.* 2020[69] |

*Continued*

**Table of Advice 6** ***Continued***

| Advice | Phase | Category of evidence | Strength of evidence | Vote (%) | References |
|---|---|---|---|---|---|
| Use high-density mapping catheters to characterize border-zone circuits typical of inflammatory scars. | Cold |  |  | 85 | • Cronin *et al.* 2019[68] |
| Define ablation endpoints as both: 1) non-inducibility of clinical VTs; and 2) substrate homogenization by abolition of LPs/LAVAs. | Cold |  |  | 90 | • Cronin *et al.* 2019[68] |
| Perform VT ablation at experienced centres with available resources, including EP expertise and mechanical circulatory support (i.e. VA-ECMO) for unstable arrhythmias. | All |  |  | 100 | • Zeppenfeld *et al.* 2022[77]<br>• Lenarczyk *et al.* 2024[93] |

ICD, implantable cardioverter defibrillator; LAVA, local abnormal ventricular activity; LP, late potential; OBS, observational study-based evidence; OPN, expert opinion-based evidence; PVCs, premature ventricular complexes; VA, ventricular arrhythmias; VA-ECMO, venoarterial extracorporeal membrane oxygenator; VF, ventricular fibrillation; VT, ventricular tachycardia.

competitive or high-intensity sports is generally advised for 3 to 6 months,[1] with individualized reassessment based on imaging and clinical stability. Patients with prior sustained VA,[53,108] reduced LVEF at presentation[150,151] or discharge,[6] persistent atypical LGE (e.g. extensive, septal/anteroseptal, ring-like) on CMR,[13,60,67,113] or high-risk genotypes (e.g. *LMNA*, *DSP*, *FLNC*)[12,13,28] require intensive follow-up.

As recommended by the ESC Guidelines on Myocarditis and Pericarditis,[1] reassessment during follow-up should include cardiac biomarkers, ECG, echocardiogram, rhythm monitoring, functional tests, and restaging of myocarditis by gold standard techniques. Remission of myocarditis is suggested by symptom relief, normalization of cardiac biomarkers, absence of residual LV dilation or dysfunction, and clearance of arrhythmias.[1] Phase-adapted surveillance of rhythm disorders through serial ECGs, Holter or ILR monitoring is central. Polymorphic VAs may reflect residual inflammation, while monomorphic VT suggests either fixed substrate or hot-to-cold transition phase.[4–6] ILR carriers benefit from remote telemetry, with alerts for bradycardia <40 bpm, pauses >6 s, or asymptomatic AF >30 s.[152] CMR follow-up within 6 months evaluates the resolution or persistence of LGE and T2 abnormalities (hot-to-cold transition phase), which informs both arrhythmic risk and the safety of re-engaging in physical activity.[107] In the presence of ICDs or other limitations to CMR, FDG-PET scan and biomarker reassessment may help in disease monitoring.[64] EMB may be repeated in selected cases to guide critical therapeutic choices as immunosuppression.[89]

## Monitoring antiarrhythmic therapy

Antiarrhythmics use requires structured safety surveillance. Supplementary material online, *Table S7.2* outlines specific monitoring for agents such as amiodarone, sotalol, flecainide, and mexiletine.[1,94] Indications should be re-evaluated regularly, particularly in the setting of changing LVEF, arrhythmia morphology, or suspected drug toxicity. Antiarrhythmics tapering may be considered in the long term in patients with resolved inflammation, low-risk substrate, and absence of residual VA, but only after shared decision-making and re-evaluation via rhythm and imaging surveillance.

## Activity guidance, return to sport, and pregnancy

Physical activity is strongly discouraged during the hot and hot-to-cold transition phases.[1,107] Clearance requires: resolution of symptoms, troponin and inflammatory markers, normalization or near-complete LVEF recovery, absence of arrhythmias on extended monitoring, and remission of myocarditis with or possibly without residuals.[1] This phase-aware clearance pathway complements the 2025 ESC Guidelines on Myocarditis and Pericarditis emphasis on structured follow-up and individualized return to exercise by adding arrhythmia-specific gates (no symptoms, normalized troponin and natriuretic peptides, normal LVEF, no VT/NSVT on Holter and exercise testing, T2/FDG quiescence, stable/non-progressive LGE).[1] Persistent LGE alone is not an absolute contraindication, but mandates individualized discussion; atypical LGE (extensive, septal/anteroseptal, or ring-like), EAVM-confirmed low-voltage areas and lack of VA suppression with exercise argue against competitive sport until stability is confirmed on serial CMR and continuous rhythm monitoring by ILR in selected cases.[13,55,153] Documenting the absence of high-risk genotype is not a current requirement, but may help the decision making when HPGC is suspected.[41] The 2025 ESC Guidelines on Myocarditis and Pericarditis suggest a personalized approach for activity guidance, with athletic training and competitive sport generally withheld for 1–6 months.[1] In myopericarditis (pericarditis-predominant, preserved LVEF, minimal myocardial LGE, no VA), return to play is primarily

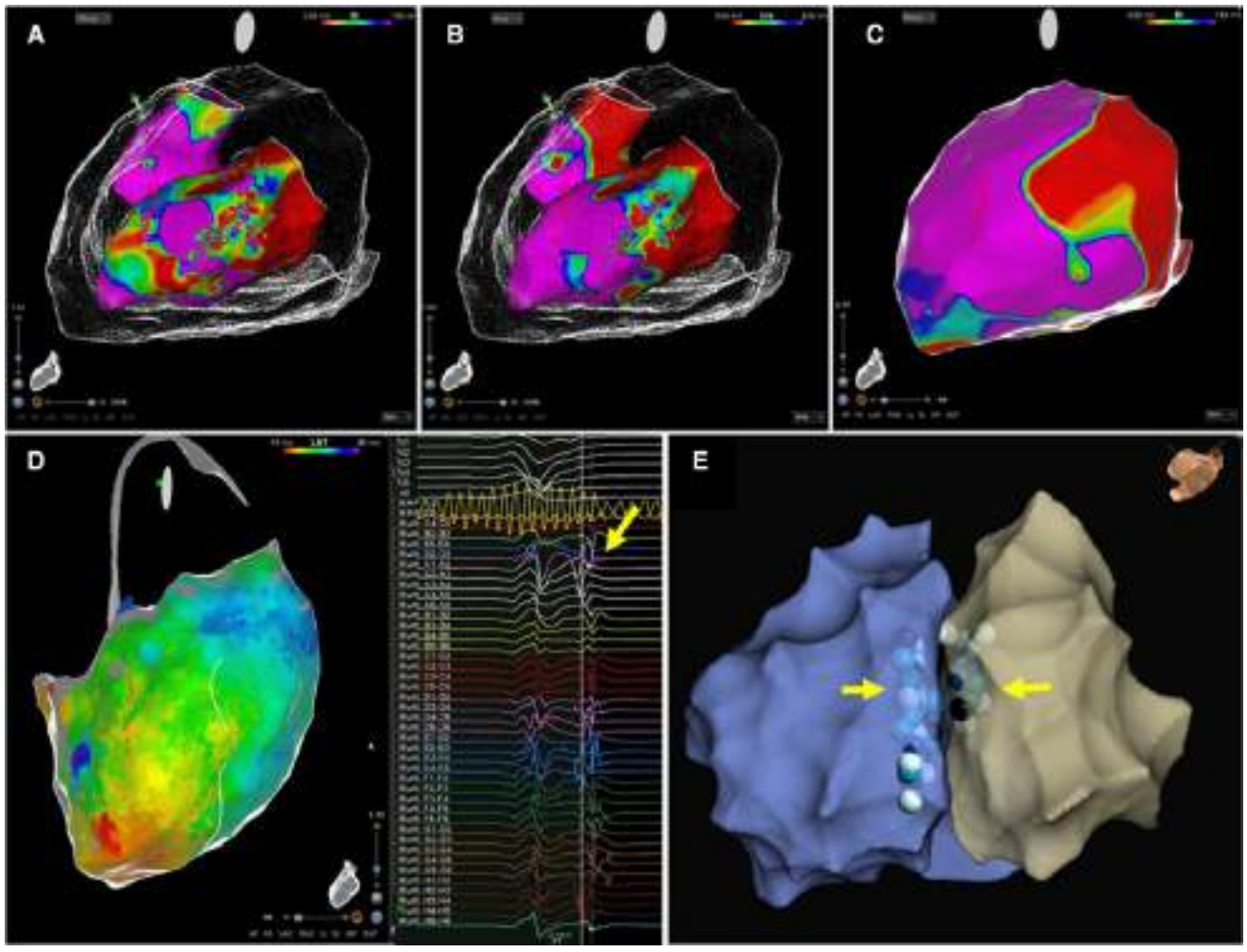


**Figure 9** Findings during VT ablation in cold-phase myocarditis. Findings of VT ablation are shown for patients with myocarditis. A-C. EAVM in a patient with a history of myocarditis and extensive inferolateral LV scar. Dense scar (red) and borderzone (rainbow) are compared as recorded by endocardial bipolar (*A*), endocardial unipolar (*B*) and epicardial bipolar map (*C*). (*D*) Late potentials (arrow) in a patient with chronic myocarditis (cold phase) and inferolateral scar. (*E*) Ablation lesions (arrows) at both sides of interventricular septum in a patient with giant cell myocarditis and multiple septal VTs refractory to triple immunosuppression. EAVM, electroanatomic voltage mapping; VT, ventricular tachycardia.

pericarditis-led and may be shorter than arrhythmic myocarditis, where a more conservative strategy is generally advised. Women in childbearing age should receive appropriate counselling regarding allowed drugs, risks, and delivery mode.[102] All patients should be educated on warning symptoms and be offered fast-track reassessment for symptom relapse.

## Long-term management: device and pharmacotherapy reassessment

Beyond the acute phase, patients may enter a phase of clinical stability—raising questions about therapy duration and device permanence. In patients, arrhythmia-free for ≥5 years without LGE or high-risk genotype, downgrade/explantation may be considered as an expert consensus strategy beyond guidelines and following multidisciplinary assessment (see Supplementary material online, *Table S7.4*). Medical therapy may be cautiously tapered in recovered patients without fibrosis or genetic predisposition. However, ongoing treatment remains the default in individuals with persistent LGE, incomplete LVEF recovery, or underlying HPGC. Patients receiving immunosuppressive therapy should undergo close multidisciplinary surveillance to review medications, evaluate toxicity, and ensure protection from infections.[3,96] Treatment duration may be longer in patients with systemic rheumatologic diseases or HPGC.[26,105]

# Summary

A structured follow-up strategy—integrating rhythm surveillance, imaging, and functional assessment—is essential to ensure patient safety, prevent relapse, and guide lifestyle changes. Supplementary material online, *Tables S7.1–S7.4* offer practical frameworks for clinical follow-up, drug monitoring, return-to-activity decisions, and long-term device strategies. Intensive rhythm surveillance is essential in patients with

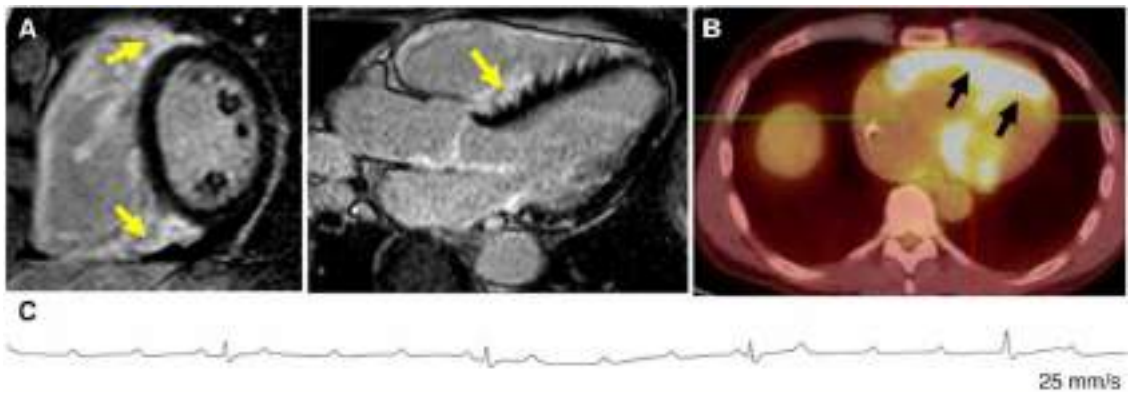


**Figure 10** CMR (*A*) demonstrating septal and right ventricular late gadolinium enhancement (LGE); FDG-PET (*B*) showing extensive myocardial inflammation (arrows) and mediastinal/hilar lymph node uptake, consistent with cardiac sarcoidosis presenting with high-degree AV block (*C*).

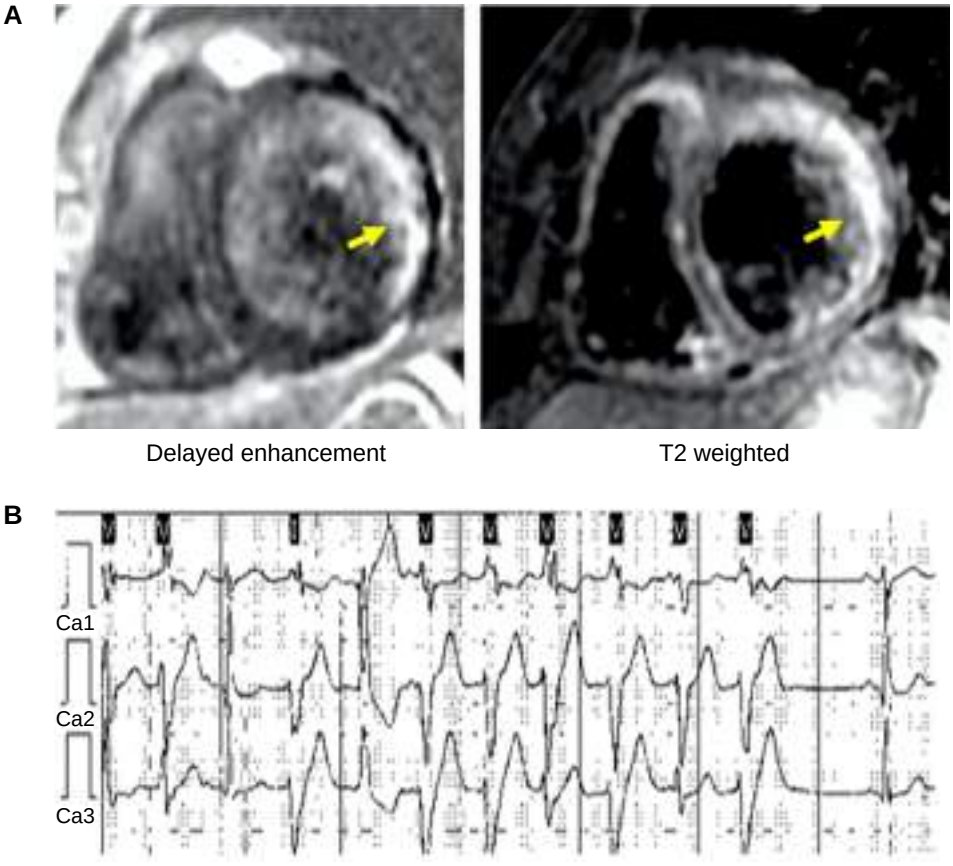


**Figure 11** CMR (*A*) revealing extensive lateral wall LGE and myocardial oedema on T2-weighted imaging; acute inflammation identified as the underlying substrate for polymorphic non-sustained ventricular tachycardia (*B*).

persistent inflammation, fibrosis, or high-risk genotypes. The possibility of downgrading or explanting devices should be weighed carefully, guided by arrhythmic silence, imaging stability, and genetic context. Reassessment of therapy duration—both pharmacologic and device-based—should be embedded in a personalized, multidisciplinary care model.

**Table of Advice 7** Return-to-play, lifestyle, and follow-up

| Advice | Phase | Category of evidence | Strength of evidence | Vote (%) | References |
|---|---|---|---|---|---|
| Plan patient multidisciplinary follow-up by in-person visits and/or remote telemedicine consultations, according to the ESC schedule (exams and timing). | All |  |  | 100 | • Schulz-Menger *et al.* 2025[1]<br>• Peretto *et al.* 20202[51] |
| Evaluate arrhythmia burden and trends during follow-up using 12-lead ECG Holter, or ILR depending on phase and symptoms. | All |  |  | 100 | • Peretto *et al.* 2021[55] |
| Repeat CMR (or FDG-PET if CMR is limited) by 6 months to confirm transition to cold phase. | Hot-to-Cold |  |  | 95 | • Schulz-Menger *et al.* 2025[1]<br>• Peretto *et al.* 2022[64] |
| **Favour return-to-play with an individualized approach, upon verification of all safety gates:**<br>• symptoms remission;<br>• normalized biomarkers;<br>• stable/normalized LVEF;<br>• no documented VT/NSVT by EST and rhythm monitoring;<br>• absence of inflammation (CMR T2/FDG);<br>• stable/non-progressive LGE. | Hot-to-Cold, Cold |  |  | 95 | • Schulz-Menger *et al.* 2025[1]<br>• Pelliccia *et al.* 2019[107] |
| Periodically reassess sports/occupation eligibility and ICD indication as substrate stabilizes. | Cold |  |  | 100 | • Pelliccia *et al.* 2019[107] |
| Educate on warning symptoms (syncope, palpitation, chest pain recurrence, exercise intolerance) and favour rapid access pathway for evaluation. | All |  |  | 100 | |

CMR, cardiac magnetic resonance; CPET, cardiopulmonary exercise test; ECG, electrocardiogram; ESC, European Society of Cardiology; EST, exercise stress test; FDG-PET, 18F-fluorodeoxyglucose positron emission tomography; ICD, implantable cardioverter defibrillator; OBS, observational study-based evidence; OPN, expert opinion-based evidence.

Tailored return-to-activity guidance and periodic rhythm re-evaluation help prevent recurrence and support functional recovery. Multidisciplinary coordination between heart failure, electrophysiology, imaging, and immunology teams is critical for optimizing long-term outcomes.[1,51]

# Chapter 8: application of clinical case scenarios

**How does it work in real life?**

This chapter presents eight clinical vignettes that illustrate how rhythm disturbances in myocarditis and inflammatory cardiomyopathy are managed in real world setting. These vignettes focus on arrhythmia-predominant myocarditis where the ESC Guidelines[1] necessarily provide a framework but non-prescriptive steps. The clinical cases are meant to illustrate individualized, phase-adapted diagnostic and treatment strategies, intentionally spanning complex scenarios where expert advice may help in clinical decisions. For challenging clinical questions (Q), the degree of concordance among experts is shown in ad-hoc consensus footers.

## Case 8.1: High-degree AV block in a young patient: unmasking inflammatory cardiomyopathy

A 43-year-old man presented with fatigue and abdominal discomfort. ECG revealed a complete AV block (third-degree). Troponin T was mildly elevated; CMR showed oedema and subepicardial LGE in the septum and RV (see *Figure 10*) (Q.8.1.1). FDG-PET confirmed inflammation. EMB revealed multinucleated giant cells, consistent with GCM. An ICD was implanted (Q8.1.2), and immunosuppression initiated (Q8.1.3) with corticosteroids (prednisone 1 mg/kg/day, oral, then tapered),

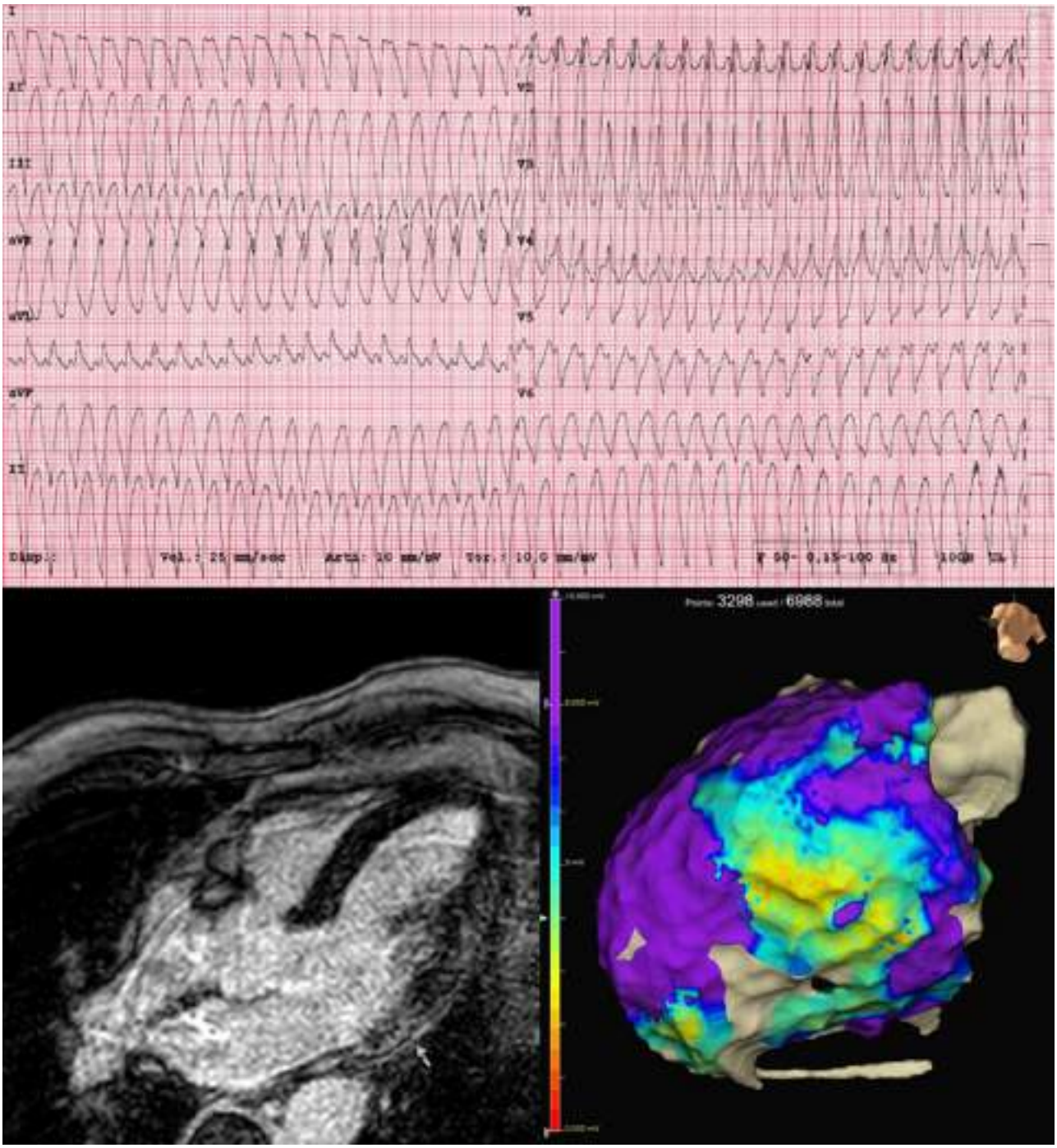

**Figure 12** Monomorphic VT documented on 12-lead ECG (*A*). CMR without oedema but with subepicardial LGE (*B*), indicating a chronic scar-related re-entrant substrate. High-density epicardial voltage mapping of the lateral left ventricle (*C*) showing extensive low-voltage areas (<1.5 mV); Genetic testing was planned.

mycophenolate mofetil (2.5 g day, oral), and cyclosporine (300 mg day, oral) for 6 months.

**Consensus footer**:

| | | |
|---|---|---|
| Q8.1.1 | Next exam after CMR? | EMB (47%), PET (41%), genetic test (12%) |
| Q8.1.2 | Device type? | PM (6%), TV-ICD (75%), WCD (19%) |
| Q8.1.3 | IST indicated? | Yes (100%), No (0%) |

**Rationale:** This case illustrates the typical presentation of acute myocarditis with high-grade AV block. Inflammatory infiltration of the conduction system is frequently observed in GCM and sarcoidosis (see Chapters 1.2 and 1.3). The indication for permanent pacing or ICD therapy should be guided by the underlying disease subtype and associated arrhythmic risk (see Supplementary material online, *Table S5.1*). In GCM, even transient AV block may herald malignant arrhythmias and frequently requires permanent ICD implantation.

**Key message:** AV block in younger patients may reflect sarcoidosis or GCM. CMR, PET, and EMB are essential; early ICD and tailored immunosuppression are often required.

## Case 8.2: Atrial fibrillation in systemic rheumatologic disease with chronic myocarditis

A 38-year-old woman with systemic lupus erythematosus (SLE) developed frequent atrial fibrillation episodes. EMB had previously confirmed lupus myocarditis, active stage. Succesful rhythm control was initially obtained by corticosteroids, methotrexate, and metoprolol. A few years later, atrial fibrillation recurred. CMR showed no gross oedema or dense LGE (Q.8.2.1). Catheter ablation was performed (Q8.2.2); voltage mapping showed preserved atrial substrate. In light of the persistence of sporadic palpitation an ILR was implanted (Q8.2.3); direct oral anticoagulant initiated.

**Consensus footer**:

| | | |
|---|---|---|
| Q8.2.1 | EMB needed as well? | Yes (41%), No (59%) |
| Q8.2.2 | Would have started antiarrhythmic drugs before ablation? | Yes (76%), No (24%) |
| Q8.2.3 | Follow-up by Holter ECG instead? | Yes (53%), No (48%) |

**Rationale:** Autoimmune myocarditis often affects the atria and may lead to atrial fibrillation or flutter, especially in systemic inflammatory diseases such as SLE (see Chapters 1.2 and 1.3). Rhythm monitoring and recurrence risk should guide decisions on ILR implantation or ablation strategies (see Supplementary material online, *Table S6.3*). Structured follow-up with device-based monitoring is advised in patients with persistent inflammation or recurrent SVA (see Supplementary material online, *Table S7.3*).

**Key message:** Autoimmune-mediated atrial fibrillation may require ablation despite immunosuppressive therapy. ILRs support rhythm monitoring; therapy must address both systemic and cardiac inflammation.

## Case 8.3: Polymorphic ventricular tachycardia in active myocarditis

A 28-year-old man presented with syncope and polymorphic VT (see *Figure 11*). Coronary arteries were normal. CMR showed oedema and inferolateral LGE. EMB confirmed acute, virus-negative lymphocytic myocarditis (Q8.3.1). He received corticosteroids (prednisone 1 mg/kg/day tapered down over 3 months) and mofetil mycophenolate (titrated up to 3 g day for 12 months), and a WCD was prescribed (Q8.3.2). At 6 months, CMR showed LGE without residual abnormality in T2-weighted sequences, systolic function was normal in both ventricles, and both exercise stress test and Holter ECG showed no arrhythmias. Genetic testing was negative. WCD was removed (Q8.3.3). No proven or suspected VA recurrences were observed in the long term.

**Consensus footer**:

| | | |
|---|---|---|
| Q8.3.1 | Would have performed EMB to enable safe use of IST? | Yes (71%), No would have started betablocker/ antiarrhythmic therapy without EMB (29%) |
| Q8.3.2 | Agree on choosing WCD? | Yes (94%), No, would have chosen ICD instead (6%) |
| Q8.3.3 | Safe to remove WCD at month 6? | Yes (65%), No, would reassess risk by repeating EMB (6%), No, would reassess risk by PVS (29%) |

**Rationale:** Polymorphic VT or electrical storm in acute myocarditis typically reflects active inflammation and substrate instability (see Chapters 1.3 and 3.1). In virus-negative cases, immunosuppressive therapy should be initiated alongside rhythm control (see Supplementary material online, *Table S3.1* and *S3.3*). Ablation is not advised in the hot phase unless life-threatening instability persists despite optimized medical therapy (see Supplementary material online, *Table S6.1*). Temporary protection with a WCD or mechanical support may be needed (see Supplementary material online, *Table S5.1*).

**Key message:** Inflammatory polymorphic VT requires early immunosuppression. WCDs mitigate transient SCD risk in reversible phases.

## Case 8.4: Scar-related ventricular tachycardia in healed myocarditis

A 66-year-old man with prior EMB-proven lymphocytic myocarditis presented with monomorphic VT (see *Figure 12*) despite ongoing treatment with metoprolol 200 mg/day and

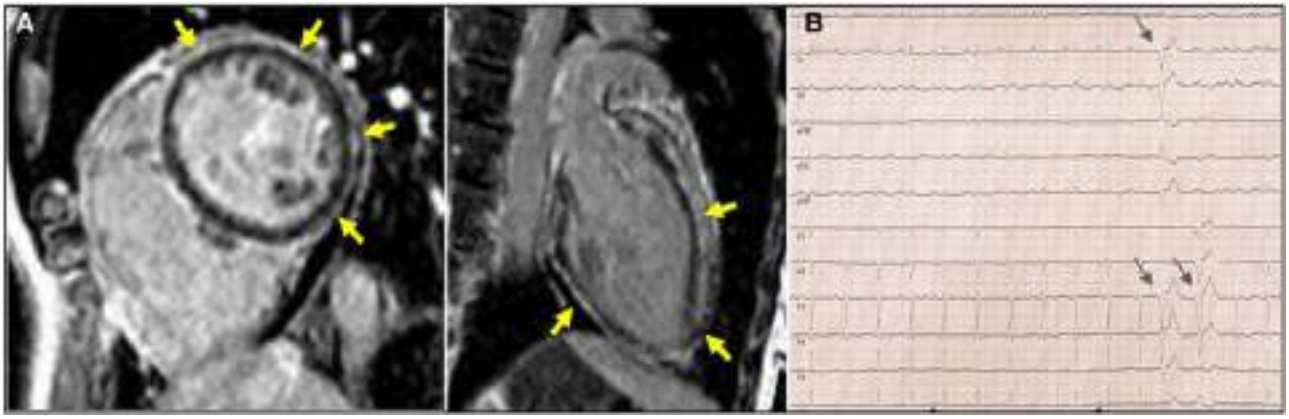


**Figure 13** CMR (*A*) demonstrates diffuse subepicardial LGE in a ring-like pattern involving the lateral left ventricular wall and basal right ventricle, accompanied by myocardial oedema. The corresponding ECG (*B*) shows fragmented QRS complexes, frequent premature ventricular complexes (PVCs), and T-wave inversions—typical features of an arrhythmogenic inflammatory phenotype associated with desmoplakin (*DSP*) cardiomyopathy in the hot phase.

amiodarone 200 mg/day. CMR showed dense subepicardial scar without oedema; FDG-PET was negative. Mapping confirmed a stable re-entry circuit. Ablation and secondary prevention ICD were performed (Q8.4.1–8.4.3).

**Consensus footer**:

| | | |
|---|---|---|
| Q8.4.1 | Right timing for VT ablation? | Yes (88%), No, would have repeated EMB to start immunosuppression instead (12%) |
| Q8.4.2 | Agree with ablation first, ICD next? | Yes (82%), No, would have implanted ICD first (18%) |
| Q8.4.3 | Would have suggested ICD even in the event of a prior successful VT ablation? | Yes (88%), No (12%) |

**Rationale:** This patient with recurrent sustained VT and inferolateral scar represents a typical post-inflammatory phenotype with fixed substrate (see Chapters 1.1 and 1.3). Imaging (LGE) and electroanatomical mapping (LVA) guide ablation strategy and ICD indication (see Supplementary material online, *Tables S5.1* and *S6.1* and ). In stable chronic myocarditis, ICD therapy and targeted ablation can prevent recurrence and improve quality of life. Extensive LVA suggest chronic cardiomyopathic process rather than focal post-inflammatory scarring, and called for ICD implant independently of VT ablation results.

**Key message:** Cold-phase scar-related VT requires ablation and ICD. Inactive myocarditis does not warrant immunosuppressive therapy.

## Case 8.5: Recurrent arrhythmic myocarditis and hot-phase genetic cardiomyopathy

A 32-year-old woman presented with recurrent episodes of chest pain, palpitations, and exertional dyspnoea. Family history was unremarkable. ECG revealed fragmented QRS complexes with low voltages, frequent PVCs, and T-wave inversions. Echocardiogram showed LVEF 50%. CMR revealed subepicardial, ring-like LGE involving the lateral LV and RV basal segments (burden 15%), alongside myocardial oedema (see *Figure 13*). EMB confirmed virus-negative inflammatory cardiomyopathy (CD3+ T lymphocytes > 7/mm$^2$). Genetic testing identified a pathogenic *DSP* variant (Q8.5.1).

During telemetry, frequent non-sustained VT, and frequent PVC (14% burden) were documented. An ICD was implanted for primary prevention of sudden cardiac death (Q8.5.2). Immunosuppressive therapy (Q8.5.3) included prednisone 1 mg/kg plus azathioprine 2 mg/kg per os. Sotalol, a class III antiarrhythmic with beta-blocking effects, was initiated (80 mg tid, oral) to suppress ventricular ectopy and reduce arrhythmic burden, given the preserved LVEF and absence of contraindications. Given the autosomal dominant inheritance pattern of *DSP*-related disease, cascade family screening was initiated, including cardiogenetic counselling, ECG, echocardiography, and CMR in first-degree relatives.

**Consensus footer**:

| | | |
|---|---|---|
| Q8.5.1 | Would have considered genetic testing even if family history was unremarkable and EMB concluded for myocarditis? | Yes, because of LGE pattern on CMR (82%), No, would have managed just as virus-negative myocarditis (18%) |

*Continued*

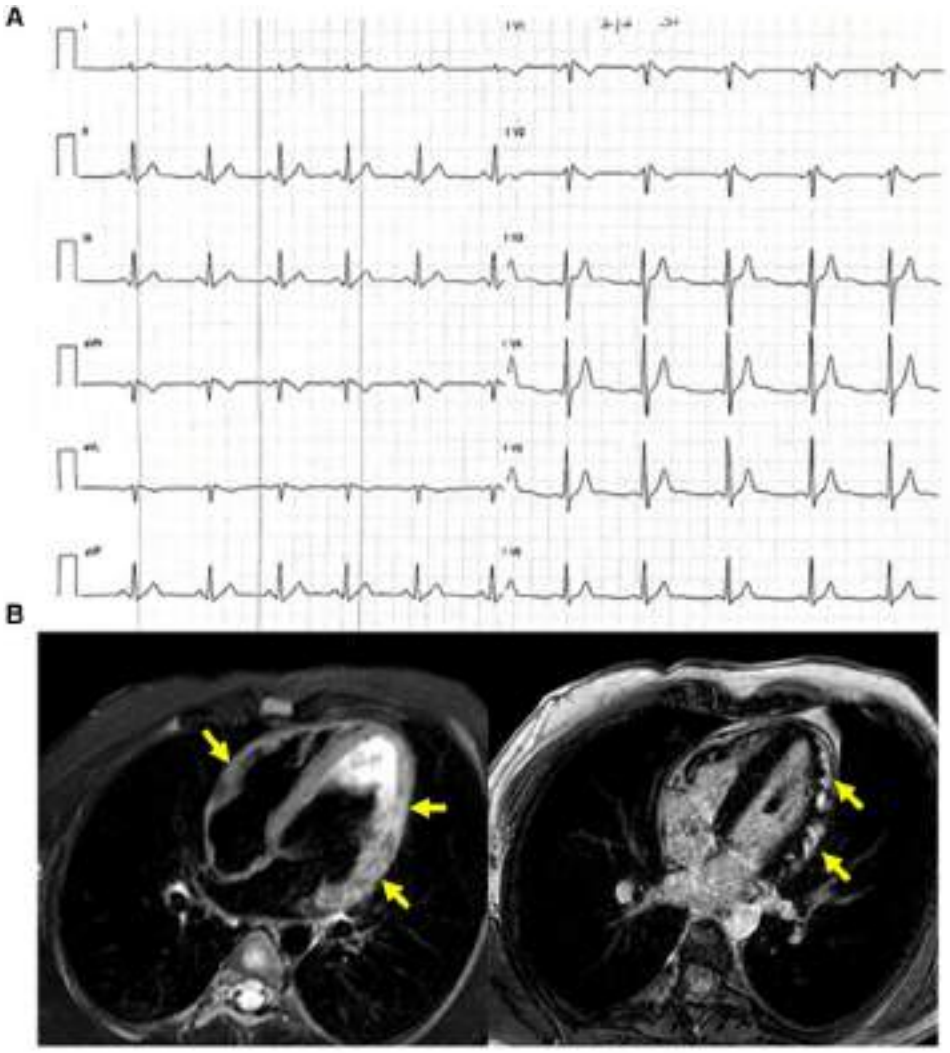


**Figure 14** Electrocardiogram (*A*) showing fever-induced type 1 Brugada pattern; CMR (*B*) revealing diffuse myocardial oedema of both ventricles and non-ischaemic LGE in lateral LV wall—findings consistent with transient inflammatory modulation of RV conduction.

***Continued***

| | | |
|---|---|---|
| Q8.5.2 | Would have preferred S-ICD instead of transvenous device? | Yes (41%), No (59%) |
| Q8.5.3 | Would have started immunosuppression despite diagnosis of genetic cardiomyopathy? | Yes, because of EMB findings (58%), No, would have limited to cardiological therapy (42%) |

**Rationale:** As detailed in Chapters 1.1 and 1.2, recurrent myocarditis with ring-like or biventricular LGE is highly suggestive of *DSP*-related HPGC. *DSP* variants often mimic ARVC on imaging, but up to one-quarter present with atypical or left-dominant patterns—including global ring-like enhancement and subepicardial fibrosis—which may mask the underlying genetic aetiology unless specifically considered. This case reflects a high arrhythmic risk profile with troponin elevation, frequent PVCs, fragmented QRS, and subepicardial scar (see Supplementary material online, *Table S1.3*). Beta-blockers and amiodarone remain first-line pharmacotherapy (see Supplementary material online, *Table S3.1*); sotalol is a cold-phase alternative in patients with preserved function and low QT risk, to avoid long-term toxicity of amiodarone.[93] Given the documented NSVT and the *DSP* genotype, early ICD implantation was justified (see Supplementary material online, *Table S5.1*), and lack of risk features for bradyarrhythmias support the choice of S-ICD. Long-term rhythm surveillance and genetic counselling are essential and should follow the risk-adapted strategy outlined in Chapter 7 (see Supplementary material online, *Table S7.3*).

**Key message:** Recurrent myocarditis with ring-like or biventricular LGE may indicate *DSP*-related hot-phase cardiomyopathy—even in the absence of typical ARVC features. Recognizing these atypical patterns is critical for early risk stratification, ICD implantation, family screening, and individualized therapy.

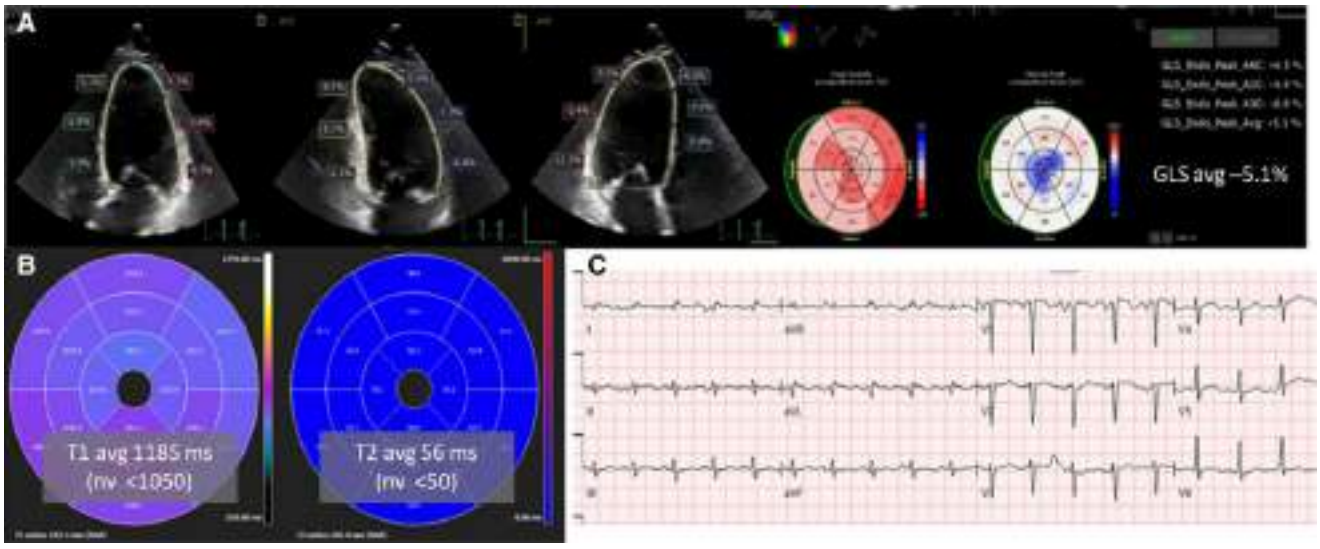


**Figure 15** Transthoracic echocardiography (*A*) with tissue Doppler imaging (TDI) demonstrating severely reduced left ventricular systolic function and elevated filling pressures (E/e′ ratio); CMR (*B*) showing T1 and T2 mapping abnormalities consistent with myocarditis; corresponding ECG (*C*) with sinus tachycardia. Findings support a diagnosis of inflammatory postpartum cardiomyopathy in the differential with peripartum cardiomyopathy.

## Case 8.6: Paediatric myocarditis with Brugada-like ECG pattern

A 14-year-old boy presented with fever and Brugada-like ECG (see *Figure 14*). He had no family history of sudden death or cardiomyopathy. Biventricular function was normal. The patient had no history of syncope or palpitation. No ventricular arrhythmias occurred. CMR showed diffuse oedema and LV lateral LGE; elevated native T1 and T2 mapping supported the diagnosis of myocarditis (Q8.6.1). The ECG normalized after defervescence. Genetic testing for *SCN5A* was negative (Q8.6.2). He was discharged with indication to reassessment though Holter ECG and in 3 months (Q8.6.3). At 6 months, CMR showed no oedema and no residual LGE. The patient was readmitted to sport activity.

**Consensus footer**:

| | | |
|---|---|---|
| Q8.6.1 | Would have performed EMB here? | Yes, to enable safe use of immunosuppression (29%), No, given the absence of treatment endpoints (71%) |
| Q8.6.2 | Would have performed genetic testing? | Yes (35%), Yes, but not limited to *SCN5A* (47%), No (18%) |
| Q8.6.3 | Agree with rhythm monitoring strategy (Holter ECG)? | Yes (76%), No, would have chosen WCD (12%), No, would have chosen ILR (12%) |

**Rationale:** As discussed in Chapter 1.2, febrile illnesses can transiently unmask Brugada-like ECG patterns due to inflammatory modulation of cardiac sodium channels. This phenomenon is particularly relevant in paediatric myocarditis, where *SCN5A* mutations may coexist or be mimicked by inflammation. Chapter 1.3 supports a non-invasive, watchful follow-up strategy in children without sustained ventricular arrhythmias, preserved cardiac function, and negative genetic screening. Serial ECGs and CMR (see Supplementary material online, *Table S2.1* and *S7.1*) are critical to confirm recovery. ICD implantation is generally not indicated in the absence of arrhythmic events, structural abnormalities, or genetic predisposition.

**Key message:** Febrile Brugada-like patterns may reflect transient inflammation. Genetic testing and follow-up help avoid unnecessary ICD in children.

## Case 8.7: Arrhythmic myocarditis in the postpartum period

A 36-year-old woman presented 10 days postpartum with heart failure symptoms. Echocardiogram showed severe biventricular dysfunction (LVEF 18%) (see *Figure 15*). CMR showed no LGE but elevated T1/T2 values (Q8.7.1). EMB revealed lymphocytic inflammatory infiltrates, no fibrosis and no necrosis (borderline myocarditis). Peripartum cardiomyopathy (PPCM) was considered in overlap with myocarditis. Genetic testing excluded cardiomyopathy-associated gene variants. She received heart failure therapy and WCD monitoring (Q8.7.2). At 6 months, LVEF normalized and WCD was withdrawn (Q8.7.3).

**Consensus footer:**

| | | |
|---|---|---|
| Q8.7.1 | Given CMR results agree on performing EMB and genetic testing? | No, findings were already consistent with PPCM (12%), Yes, only EMB (12%), Yes, only genetic test (23%), Yes, both (53%) |
| Q8.7.2 | Agree on bridging with WCD for 6 months? | Yes (82%), No, would have implanted ICD (6%), No would have follow-up non-invasively by Holter ECG (12%) |
| Q8.7.3 | What would have proposed in the event of failure to normalize LVEF at month 6? | EMB-driven immunosuppression and further reassessment (35%), primary prevention ICD (65%) |

**Rationale:** Postpartum myocarditis may mimic PPCM, but advanced tissue characterization (see Chapter 1.3 and Supplementary material online, *Table S3.4*) support differentiation. As outlined in Chapter 3.1 and 3.3, immunosuppression is not routinely indicated in PPCM but may benefit biopsy-proven Infl-CMP. Chapter 5.3 and Supplementary material online, *Table S5.1* support WCD use as a bridge in patients with severely reduced LVEF and elevated arrhythmic risk during the hot phase. Serial reassessment (see Supplementary material online, *Table S7.1*) ensures safe decision-making regarding defibrillator therapy, especially in reversible cases.

**Key message:** Postpartum myocarditis may mimic PPCM. Tissue mapping and WCDs support diagnosis and risk management.

## Case 8.8: Arrhythmic myocarditis in a competitive athlete

A 29-year-old athlete had syncope after recent SARS-CoV-2 infection. Troponin was mildly elevated, but coronary CT scan was normal. Echocardiogram showed mild biventricular enlargement with normal function, overall consistent with athlete's heart. CMR showed multiple LGE striae (septal and inferolateral, non-ischaemic pattern) and mild diffuse oedema. After 6 months, oedema resolved, LGE persisted. Genetic testing was negative (Q.8.8.1). ILR showed multiple asymptomatic NSVT and the patient refused betablockers (see *Figure 16*); PVS induced sustained VT (Q8.8.2). He received ICD. Return to competitive sport was not advised (Q8.8.3).

**Consensus footer:**

| | | |
|---|---|---|
| Q8.8.1 | At this point (persistent multifocal LGE, history of syncope, normal function, no documented VA, no high-risk genotype) would have chosen ILR? | Yes (69%), No, just Holter ECG by 6 months (0%), No, WCD for at least 3 months (12%), No, ICD implant right now (19%) |
| Q8.8.2 | With multiple but asymptomatic NSVT in cold-phase would have performed PVS? | Yes (69%), No, would have implanted ICD independently of PVS (6%), No would have continue to follow-up (25%) |
| Q8.8.3 | Agree on prohibiting competitive sport? | Yes in any case (56%), No with ICD and yes without (44%), No in any case (0%) |

**Rationale:** As discussed in Chapters 1.2 and 1.3, athletes with myocarditis are at elevated arrhythmic risk, particularly when LGE persists beyond resolution of oedema. In this case, extensive LGE with septal involvement warranted advanced risk stratification with genetic testing, high-sensitivity detection of NSVT by ILR, and programmed ventricular stimulation (see Supplementary material online, *Table S4.1* and *Table S5.1*). The induction of sustained VT justified ICD implantation for primary prevention. Chapter 7.3 and Supplementary material online, *Table S7.2* emphasize that return to competitive sports is contraindicated in the presence of persistent scar, symptomatic arrhythmias, or device therapy due to the risk of sudden cardiac death.

**Key message:** In athletes with prior myocarditis, persistent/extensive/septal LGE, NSVT, and inducible VT indicate high arrhythmic risk. ICD implantation is required, and return to competitive sports is contraindicated due to the risk of sudden cardiac death.

# Closing remarks

Across the clinical cases, the voting results revealed a generally high level of agreement among experts. However, some variation in case-specific responses reflected the inherent complexity of translating theoretical or guideline-based recommendations into individualized patient management. This diversity of expert opinion underscores the need for nuanced decision-making in real-world myocarditis care.

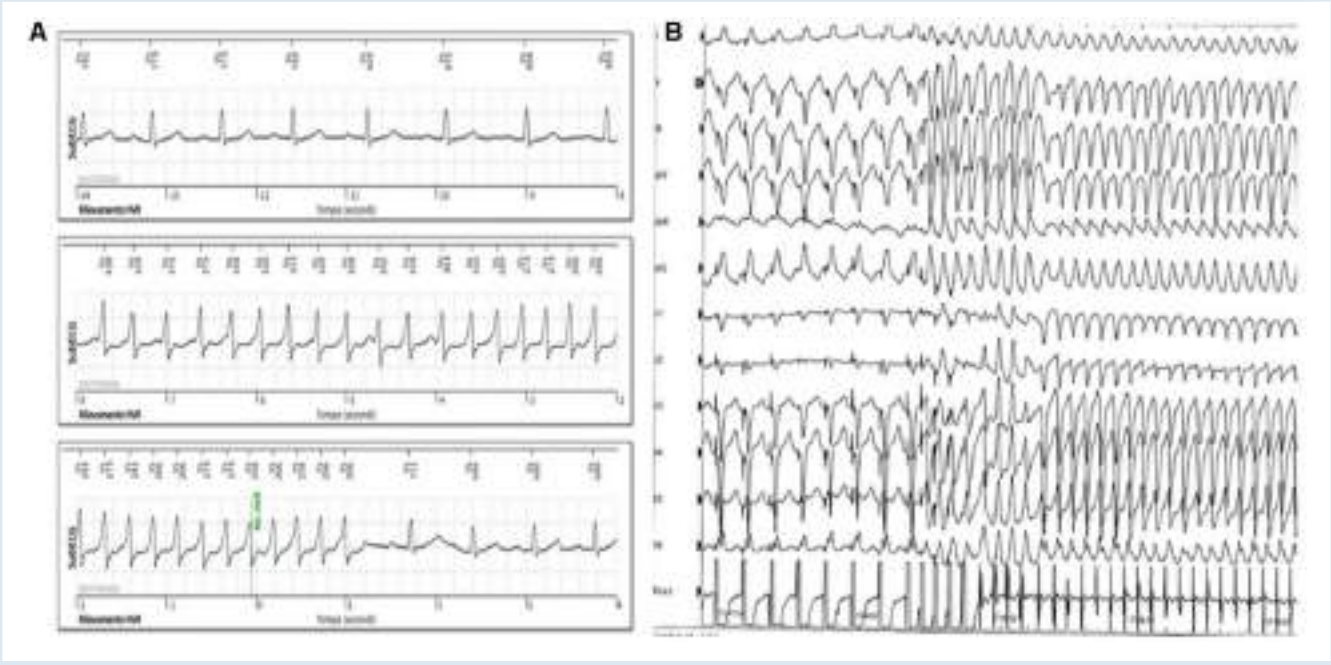


**Figure 16** Spontaneous NSVT detected by ILR (*A*). Programmed ventricular stimulation (*B*) induced a sustained monomorphic VT with LBBB morphology (atypical for classic inferolateral myocarditis)—supporting chronic cardiomyopathic substrate in the athletic heart.

# Chapter 9: perspectives, limitations, and conclusive remarks

**Where do we go next**

Rhythm disorders in myocarditis and Infl-CMP remain a dynamic and incompletely understood field. This chapter outlines unmet needs, emerging research directions, and future strategies for improving diagnostics, risk stratification, and individualized therapy. These proposals are hypothesis-generating and ESC-concordant,[1] designed to deepen areas where the guideline is concise.

## Hot-to-cold transition: a new frontier in arrhythmic risk stratification

The recognition of a distinct hot-to-cold transition phase[6,8] provides a novel and actionable framework for stratifying arrhythmic risk beyond the ESC acute/non-active dichotomy.[1] Observational data already support the clinical relevance of this phase, particularly in patients discharged with sustained monomorphic VT or reduced LVEF[6,53]; vulnerability appears substrate-driven (fibrosis/scar) and is not fully mitigated by the resolution of inflammation. In practice, this translates into considering early ICD implant in high-risk cases (Table of Advice 5). In the remaining patients, WCD bridging and deferral of definitive ICD decisions to the cold/non-acute stage is advised—consistent with the ESC Guidelines on Myocarditis and Pericarditis in the setting of acute myocarditis[1]—and reflects our view that permanent devices are safer and more informative once the substrate has stabilized. Prospective work should now refine the temporal boundaries of the transition, develop biomarkers of substrate evolution (including LGE topology and burden), and test intervention timing (duration of WCD, thresholds for ICD, and the role of PVS and ablation after cool-down) in phase-stratified cohorts.

## Arrhythmic risk stratification and the role of genetics

The integration of genetic profiling into risk stratification is essential. HPGC—particularly variants in *DSP*, *LMNA*, *FLNC*, other genes listed in Supplementary material online, *Table S2.2*—frequently manifest with myocarditis-like flares and VA. In keeping with the 2025 ESC Guidelines on Myocarditis and Pericarditis framework,[1] genetic evaluation is advised when clinical, imaging, or family cues raise suspicion (e.g. recurrent myocarditis, family history of cardiomyopathy/sudden death, exertion-triggered VA, or atypical scar patterns).[9–11,26–30] In clinical decision-making, genotype functions as a risk modifier: it lowers thresholds for intensified surveillance through the hot-to-cold phase, may justify earlier inherited cardiomyopathy referral and family screening, and can influence indication and timing of interventions. Importantly, genotype is not a stand-alone indication for ICD outside established frameworks. Based on available preliminary evidence,[26,105] this Task Force prompts the need for further investigation of immunosuppressive therapy in HPGC through dedicated prospective registries and clinical trials. Future studies should disentangle isolated myocarditis from genetically primed inflammatory expression and quantify genotype–substrate interactions across disease phases to refine phase-specific management.

## Artificial intelligence and predictive modelling

In parallel with classical risk models, academic initiatives are developing AI tools such as ARHITHMOS, RACE-MYO, and

PROTECT-VT (ARHITHMOS (Academic Risk-based Health Intelligence Tool for Myocarditis and Sudden death) is an AI-driven risk modelling platform developed within a European academic consortium (coordinated by Utrecht Medical Center). RACE-MYO (Risk Assessment in Cardiomyopathy and Myocarditis) and PROTECT-VT (Prediction and Outcome Tools for Early Cardiac Threat—Ventricular Tachycardia) are research networks based in Germany and Israel, respectively, focusing on integrating imaging, ECG, and biomarker data to stratify arrhythmogenic risk in inflammatory cardiomyopathies.), that integrate ECG features, cardiac imaging (including LGE extent and topology), serum biomarkers, and genomics into individualized risk trajectories. These tools remain investigational and lack large-scale external validation; clinical use is still exploratory. Priorities include open-access validation, transparent calibration and drift monitoring, standardized inputs (e.g. ECG formats, LGE quantification), and integration into audited decision-support with human oversight. Of particular relevance is reliable detection of phase transitions—notably entry into the hot-to-cold stage—together with early warning for VT/VF recurrence and prediction of therapy response. Consistent with the 2025 ESC Guidelines on Myocarditis and Pericarditis,[1] which emphasize established diagnostic and follow-up pathways and do not issue specific recommendations for AI-based risk prediction, such tools should enter routine care only after rigorous prospective evaluation

## Novel immunomodulatory therapies

Within the ESC 2025 Guidelines on Myocarditis and Pericarditis context[1]—biopsy-guided immunosuppression in virus-negative disease and no arrhythmia-directed recommendations—there remains a critical unmet need to determine whether targeted suppression of active myocardial inflammation can terminate malignant ventricular arrhythmias even with preserved LVEF, and prevent substrate maturation during the hot/hot-to-cold phases. In arrhythmia-predominant phenotypes, immunosuppression should be selective and management-changing (e.g. biopsy-proven autoimmune disease, relapsing inflammatory VT), and undertaken under multidisciplinary oversight in experienced centres.[39,96,99] Priority questions are who should be treated (virus-negative patients with objectively or strongly suspected activity by CMR T2/FDG and/or recent histology), when (early, phase-aware initiation with WCD bridging and ICD deferral unless secondary prevention is mandatory), and what to use (short steroid pulse with structured taper vs. standard care; addition of steroid-sparing agents such as azathioprine/mycophenolate—or methotrexate in sarcoidosis; biologics targeting IL-1/JAK strictly investigational). In acute relapsing VT/VT-storm, where immediate EMB/CMR may be impractical, a time-limited, closely monitored trial in expert centres—after exclusion of infection and with pre-specified arrhythmic and imaging endpoints—may be justified. The current evidence for arrhythmia reduction is largely observational and requires prospective confirmation; hence prospective, phase-stratified studies are needed to define efficacy, safety, and the role of specific drug classes in arrhythmogenic myocarditis.

## Arrhythmia-specific clinical trials and registries

A critical gap is the paucity of trials with arrhythmia-focused endpoints. Future studies should mandate rhythm outcomes (VT/VF recurrence, appropriate device therapies, sudden cardiac death), use adjudicated core endpoints, and stratify by phase (hot vs. hot-to-cold vs. cold) to reflect the evolving substrate. Phase-stratified registries should also capture myocarditis cases requiring pacing to compare CSP vs. RVP/BiV-CRT in inflammatory conduction disease (patient-centred outcomes, LV function, VA burden). Harmonized definitions and multicentre registries linked to biobanked genotype, imaging (including LGE topology/burden), and histology will enable phenotype-specific analyses and robust meta-analyses across centres. This strategy is consistent with the 2025 ESC Guidelines' emphasis on standardized pathways and collaborative data infrastructures,[1] while adding the phase-aware, arrhythmia-specific detail not prescriptively defined in the guideline text.

## Inflammation resolution and risk persistence

Long-term arrhythmic risk may persist despite apparent resolution of inflammation. This is especially true in patients with persistent LGE, non-recovery of LVEF, or genetic predisposition.[1,12] Prospective, phase-stratified studies should explicitly evaluate IMPS, testing whether myopericarditis without myocardial 'flags' can safely follow shorter, pericarditis-led surveillance, while perimyocarditis adheres to myocarditis pathways. ILRs, periodic imaging, and advanced rhythm analytics (e.g. frequency-domain analysis, AI telemetry) should guide follow-up in high-risk individuals. A re-evaluation of ICD indications and sport eligibility over time is also warranted.

## Refinement of classification systems

Current classifications do not fully capture the breadth of arrhythmic myocarditis nor its overlap with inflammatory cardiomyopathies. A phase-aware framework that maps the ESC acute/non-acute scheme onto hot/hot-to-cold/cold would sharpen clinical decision-making and improve trial eligibility in arrhythmia-predominant phenotypes across IMPS (myopericarditis to perimyocarditis). Future frameworks should distinguish:

- Purely inflammatory forms vs. genetically primed inflammation
- Arrhythmia-predominant vs. heart failure–dominant phenotypes
- Scar-mediated vs. inflammation-triggered arrhythmias

Such granularity is essential for rational therapy selection and meaningful study enrolment.

# Conclusion

This consensus sits alongside and operationalizes the 2025 ESC Myocarditis Guidelines. Where the guidelines provide clear direction, we align and cite ESC recommendations as context; where arrhythmia-specific gaps persist—particularly around risk evolution and substrate dynamics—we offer expert consensus advice, transparently graded (OPN/OBS/RCT) and supported by author voting (see Tables of Advice). To make guideline principles actionable in rhythm care, we overlay a phase-aware lens—hot, hot-to-cold, cold—on the ESC acute/non-acute categorization, providing the additional granularity needed for device timing, return-to-play, ablation strategy, rhythm surveillance, and selective EMB decisions.

Looking ahead, a translational agenda is required to convert this framework into definitive practice. Priorities include validating the hot-to-cold phase (temporal boundaries, LGE topology/burden, biomarkers of substrate evolution); testing WCD-bridging vs. earlier ICD, the timing and yield of PVS in non-acute disease, and the 'cool-down, then ablate' strategy; evaluating whether targeted, biopsy/imaging-guided immunosuppression can reduce the burden of malignant VA in virus-negative patients with documented activity (including

'hot-phase' HPGC cohorts); and externally validating phase-aware AI that detects transitions and forecasts VT/VF using standardized inputs and audited decision support. Multicentre, phase-stratified registries and trials with adjudicated arrhythmia endpoints—linked to biobanked imaging, histology, and genotype—should underpin this work. We envisage periodic updates of this position as far as new evidence emerges.

## Supplementary material

Supplementary material is available at *Europace* online.


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## Data availability

No new data were created or analyzed in this study. Data sharing is not applicable to this article.

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