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Premature ventricular complexes

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ABSTRACT

Premature ventricular complexes (PVCs) are early ventricular contractions triggered by an ectopic focus from within the ventricle. They are common in the general population and may cause symptoms at any burden. They become clinically relevant in the context of symptoms or complications such as PVC-induced cardiomyopathy or, more rarely, PVC-triggered malignant ventricular arrhythmias. Investigating for underlying structural heart disease is essential in patients with clinically relevant PVCs as this will inform patient counselling and management options. Treatment is guided by symptoms or left ventricular dysfunction associated with PVCs. Management options include conservative management, medical therapy and catheter ablation. In this review, we discuss the definition of PVCs and how they may present, outline relevant investigations for these patients, and discuss management considerations and treatment options.

Introduction

Premature ventricular complex (PVC) describes the phenomenon of a propagating ventricular depolarisation originating from an ectopic focus within the ventricles, that is a ventricular contraction, seen as a QRS complex on surface ECG, that is not caused by transmission of an impulse from the atria. PVCs may be diagnosed from a surface ECG with an early and often wide (> 120 ms) QRS complex without a preceding P wave.

PVCs are common in the general population and may be encountered in a range of clinical settings from asymptomatic screenings to evaluating patients with established cardiovascular disease. The prevalence of PVCs correlates with the duration of monitoring. Over 24 h, one study found at least one PVC in 69% of participants,[1] whilst monitoring for 2 weeks increased this to 99.5%.[2] Some cohort studies have demonstrated an association with PVCs and major adverse cardiovascular events (MACE).[3,4] This may reflect a subclinical myocardial process and should prompt further evaluation for a pathological cardiac substrate in certain patients.

The underlying mechanism of PVCs differs among patients, though they share the same mechanisms that underpin all arrhythmias including reentry, automaticity and triggered activity. PVCs characteristically emerge from anatomic areas likely to have structural features that promote all three mechanisms and permit the propagation of the impulse when it arises, such as areas of myocardial fibrosis. An understanding of the mechanism is often not pursued, and identifying whether the PVC arises in a structurally normal or abnormal heart is of greater importance.

Diagnosis and investigations

Presentation with PVCs encompasses the whole spectrum, from incidental and asymptomatic to debilitating symptoms.[5] Symptom–rhythm correlation is essential as a determinant of management strategy. Classically, patients describe PVCs as an 'extra' or 'skipped' beat.[5] They may experience a sudden forceful beat, which represents a sinus beat with a greater left ventricular (LV) filling time resulting in stronger contraction, elicited by a PVC-induced compensatory pause. Other common symptoms include dyspnoea, fatigue and presyncope.

When evaluating patients with PVCs, it is crucial to assess for the presence of structural heart disease (SHD) as this has implications for management and prognosis. A detailed multigenerational family history is critical, including a history of cardiomyopathy, premature device therapy, and sudden cardiac or unexplained death under the age of 50. First-line investigations include a 12-lead ECG, an ambulatory ECG monitor and a transthoracic echocardiogram (TTE).[6] 12-lead ECGs may confirm the presence of PVCs, and can be important for estimating their site of origin (SOO). An ambulatory ECG assesses PVC burden and longer monitoring periods more accurately estimate overall burden due to daily variability.[7] 12-lead Holters may be useful for PVC localisation and give further information regarding the consistency of PVC

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morphology. Wearable heart rhythm monitors, including smart watches and mobile ECG monitors, may provide diagnostic single-lead recordings which can complement established monitoring methods, particularly with symptom–rhythm correlation.

TTE represents the first-line investigation to assess for SHD. An impaired LV ejection fraction (LVEF) may raise suspicion of a structural substrate for PVCs. Cardiovascular magnetic resonance (CMR) imaging provides detailed tissue characterisation, though due to regional variations in access, thresholds for its use vary. European guidelines advise CMR in all patients with suspicion of underlying cardiomyopathy, regardless of PVC burden and for PVC burdens > 10% with reduced LVEF not explained by underlying heart disease.[6] Burdens > 20% should also prompt CMR even in the absence of clinical suspicion. Exercise stress testing, though not routinely recommended in European guidelines, may be useful in identifying exercise-induced arrhythmias.[6] In one cohort of asymptomatic patients, frequent, multifocal or couplet PVCs during the recovery phase of exercise were associated with increased cardiovascular mortality.[7]

Prognosis

PVCs in SHD carry poorer prognoses and are associated with increased LV dysfunction and mortality.[8] In the absence of SHD, 'idiopathic' PVCs have also been associated with MACE in some population-based studies.[9] One study demonstrated increased incidence of atrial fibrillation and heart failure with PVC rates greater than 77/24 h or 0.8%.[3] Practically, such burdens should not trigger further investigations. Instead, evaluation for an underlying disease process should be pursued solely for clinically relevant PVCs, with features such as symptoms, concerning family history or burdens above 5%.

Attempts have been made to risk-stratify PVCs according to their morphologies and SOO. Features such as multifocal PVCs and non-outflow tract origins may have associations with higher rates of underlying myocardial disease.[4] There are numerous exceptions to these broad categorisations and therefore PVC morphology should not be considered in isolation when assessing overall risk.

In rare instances, PVCs can trigger malignant ventricular arrhythmias, acting as the initiating event in a subset of patients diagnosed with idiopathic ventricular fibrillation (VF).[10] The notion that a PVC falling on the T wave of the preceding beat ('R-on-T') precipitates VF originated in experimental work and proved neither common nor reliable.[11] Indeed, R-on-T ectopics are frequently seen in patients who never develop VF. Idiopathic VF is increasingly recognised to comprise several mechanistically distinct subforms,[12] often reflecting a previously unrecognised structural or electrical abnormality.[13] The coupling interval of the initiating PVC is variable and initiation is not

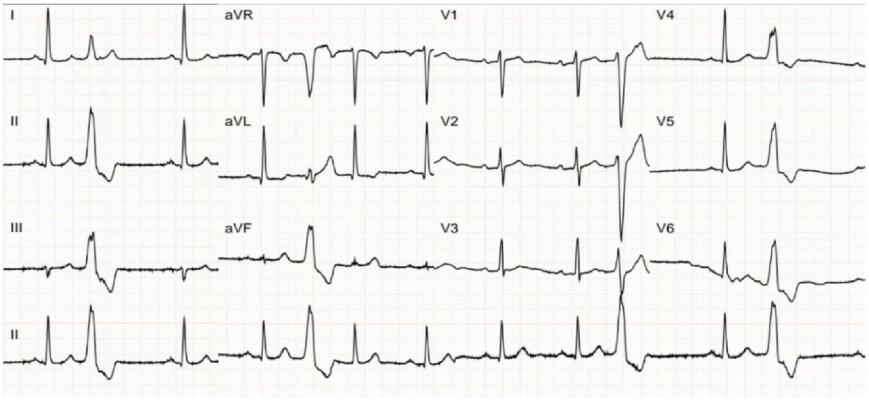

Fig. 1. A 12-lead ECG of multiple RVOT PVCs. The PVC has an inferior axis (positive in the inferior leads – II, III and aVF), propagating from the right to left (positive in lead I) with a left bundle branch block morphology. The RVOT is the most common SOO for idiopathic PVCs.
Source: Author's own image.

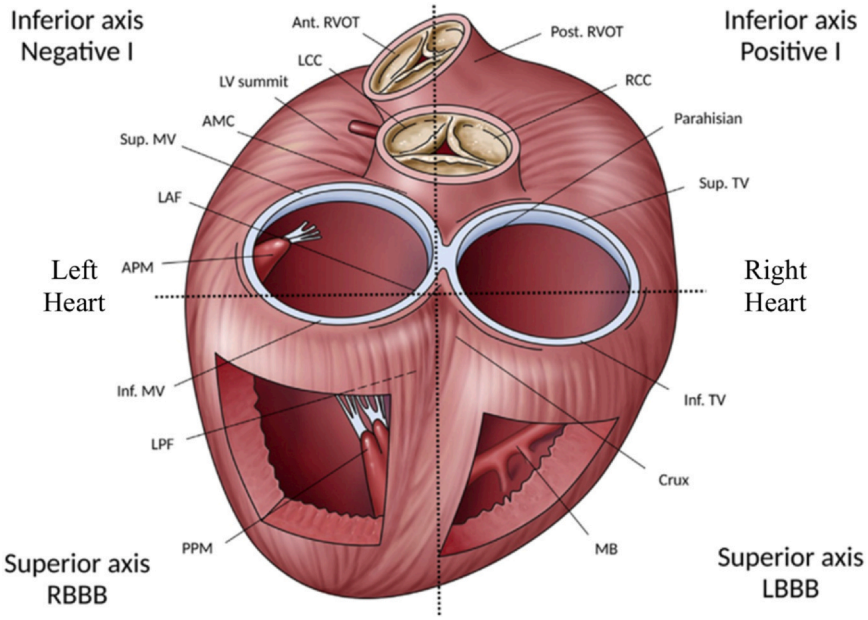

Fig. 2. Regionalisation of the SOO of a PVC into four quadrants. AMC, aortomitral continuity; APM, anterolateral papillary muscle; Inf., inferior; LAF, left anterior fascicle; LBBB, left bundle branch block; LCC, left coronary cusp; LPF, left posterior fascicle; LV, left ventricular; MB, moderator band; MV, mitral valve; PPM, posteromedial papillary muscle; RBBB, right bundle branch block; RCC, right coronary cusp; RVOT, right ventricular outflow tract; Sup, superior; TV, tricuspid valve.
Reproduced with permission from Enriques *et al.*[18]

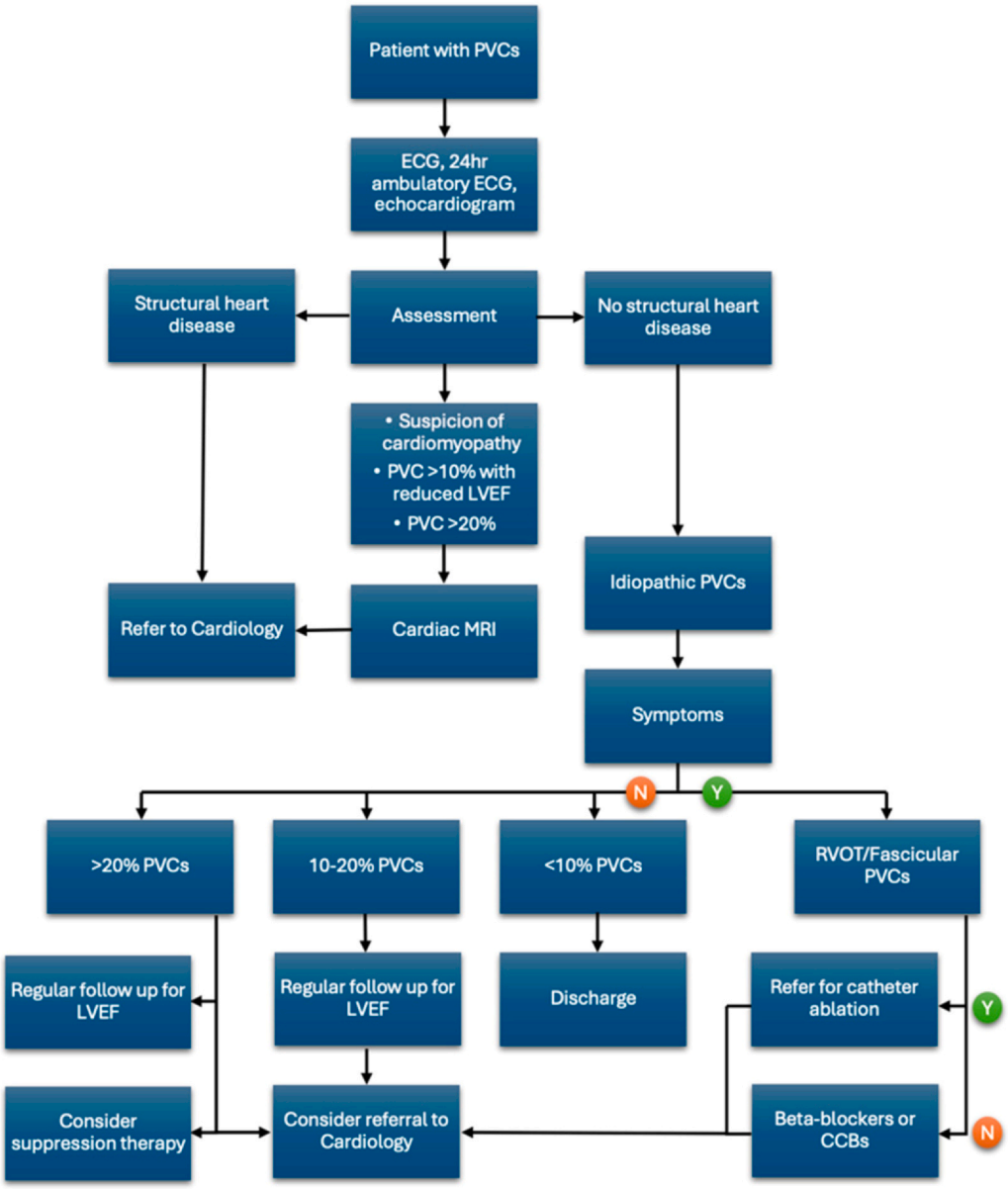

Fig. 3. Proposed treatment algorithm in patients with PVCs.

consistently pause-dependent.[10,14] Where the Purkinje system is involved, repetitive firing of a focus typically produces polymorphic VT that degenerates into VF due to wavebreak, which itself depends on dispersion of repolarisation and refractoriness. This is conceptually the same substrate that underlies the 'vulnerable period' invoked by the R-on-T hypothesis, but distinct from the idea that a single, appropriately timed ectopic can initiate VF in an otherwise normal heart. 'R-on-T' applies most clearly in defined substrates such as long-QT syndrome, where an early-afterdepolarisation-mediated beat captures on a prolonged, heterogeneously repolarising T wave. Thus, characterising PVC-triggered VF as arising primarily from short-coupled R-on-T PVCs oversimplifies a heterogeneous, substrate-dependent process.

High-burden idiopathic PVCs may cause LV dysfunction in 'PVC-induced cardiomyopathy.' This process is theorised to involve an element of ventricular dyssynchrony[15] impairing mechanical efficiency. Though there is no PVC threshold at which LV impairment is certain, there is a linear relationship. with most cases occurring at > 10% and higher risks with burdens > 20%.[16] Establishing whether PVCs are the primary cause for LV dysfunction, exacerbating an underlying cardiomyopathy, or an 'innocent bystander' is often challenging. Important considerations include the presence of a known cardiomyopathy, the presence of scar on CMR, and the time course of LV dysfunction and emergence of PVCs.

PVC localisation

Localising the origin of PVCs helps to guide management and may also have prognostic implications. Ventricular outflow tracts are the most common SOO of PVCs and are often amenable to CA.[17] Right ventricular outflow tract (RVOT) PVCs (Fig. 1) in particular are the most common SOO for idiopathic PVCs, but may also be an early manifestation of arrhythmogenic right ventricular cardiomyopathy (ARVC).[18] Numerous algorithms attempt to predict the PVC SOO from 12-lead ECGs, including one proposed by Enriquez and colleagues.[19] The frontal plane axis is first considered, indicating whether the PVC is coming from 'high to low' (positive in the inferior leads) or 'low to high' (negative in the inferior leads). Next, lead I polarity indicates whether propagation is 'right to left' (positive in lead I) or 'left to right' (negative in lead I), allowing the SOO to be projected from one of four quadrants (Fig. 2).

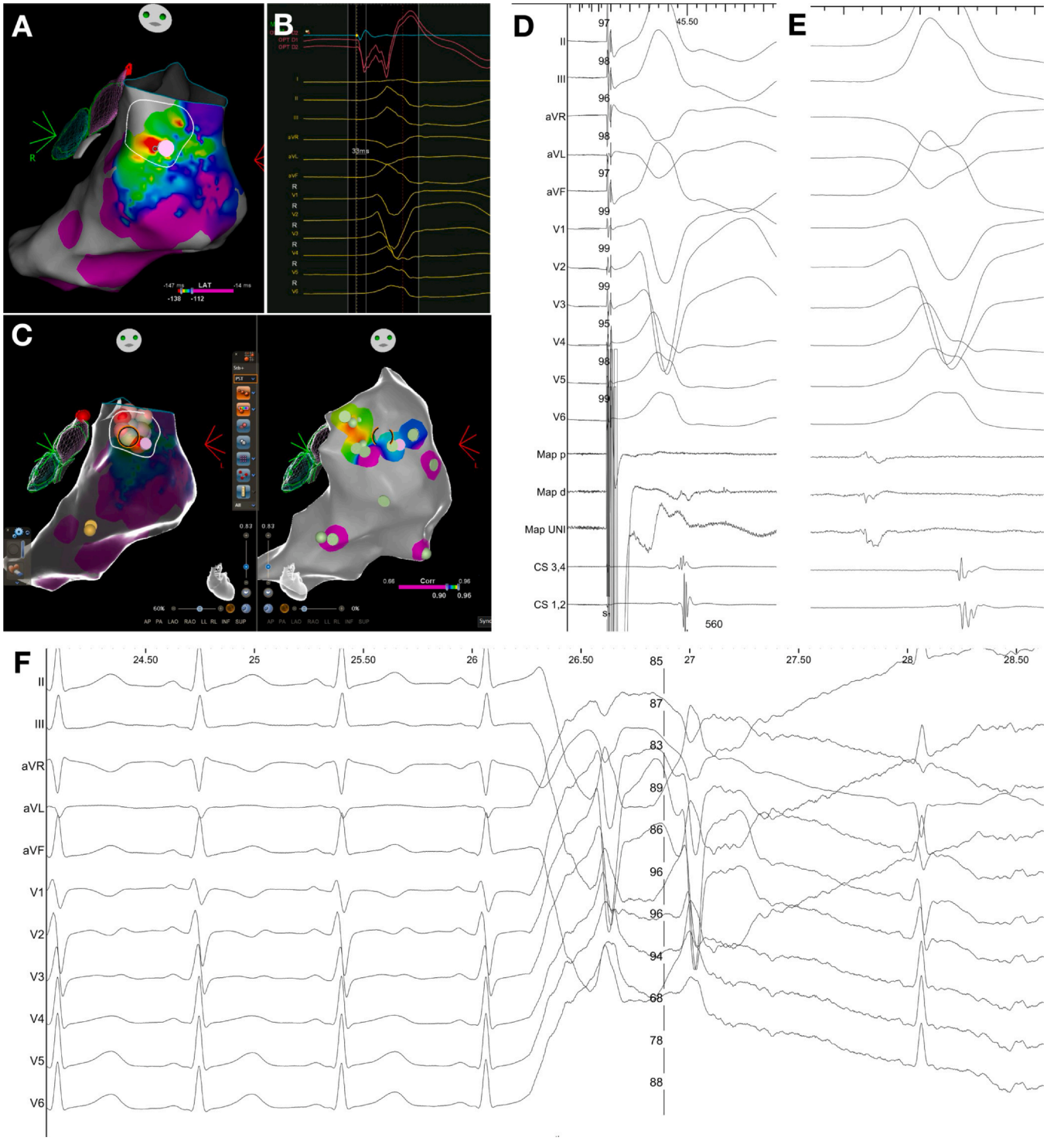

Fig. 4. Localising and ablating the PVC SOO. A – colour-coded activation map (CARTO™ system) of the right ventricle with the earliest activation point of a PVC activation indicated in red. B – Local unipolar electrograms at that same site denoted in red. Note the negative (Q) wave signifying an activation front moving away from that location, ie originating from that site. C – Pace mapping with the PASO system demonstrating correlation between activation and pace mapping. D and E – comparison of electrical signals from activation (D) and pace (E) mapping, demonstrating good correlation. Note the polarity discordance in signals between distal (Map d) and proximal (Map P) electrograms, indicating the SOO nearer to the distal tip of the catheter. F – Ablation at the PVC SOO which usually results in the flurry of PVCs – two in this case.

The QRS morphology in lead V1 (left or right bundle branch block (BBB) morphology) and other precordial leads further refines localisation prediction. The common assumption that a left BBB morphology PVC indicates a SOO in the right ventricle and vice versa has important exceptions in both structurally normal and abnormal hearts. As such, many discrepancies to existing prediction algorithms are encountered during invasive mapping of PVCs.

Management

PVC management is focused on relieving symptoms or improving PVC-mediated LV dysfunction. In asymptomatic patients with a low PVC burden and no SHD, reassurance alone is sufficient. With higher burdens (> 10%) and no LV dysfunction, regular interval assessment of LV

function is recommended. PVCs frequently spontaneously resolve without any intervention.[20] When burdens exceed 20%, suppression may be considered even in the absence of LV dysfunction or symptoms (Fig. 3).[6] PVCs associated with LV dysfunction should be suppressed regardless of symptoms, as this dysfunction typically recovers following suppression.

The most common medications used to suppress PVCs are beta-blockers and non-dihydropyridine calcium channel blockers. Both have been shown to be effective.[21] Other medications sometimes used include sotalol, flecainide and amiodarone, though these should only be initiated in specific cases under specialist cardiology care. The efficacy of anti-arrhythmic drugs is difficult to predict as they may also demonstrate pro-arrhythmic effects. Therefore, monitoring following their introduction is important.

Catheter ablation (CA) is another treatment option that has been shown to be more durable and effective. Current European guidelines recommend CA as first-line treatment for symptomatic PVCs arising from the RVOT or fascicles and for all PVC-induced cardiomyopathy with monomorphic PVCs.[6] CA importantly carries procedural risk. Overall complication rates are reported as 2–5.2%,[17] with PVC location being a significant factor.[22] RVOT PVCs are associated with lower complication rates with more frequent complications arising from non-outflow tract sites. Potential complications include vascular access issues, cardiac tamponade, AV block and stroke.

During PVC ablation, a number of strategies may be employed to localise the PVC SOO (Fig. 4). Ideally a process called activation mapping is performed, where the earliest electrical signal is identified preceding a PVC, which is then taken as the SOO of that PVC. This is visualised using computational clinical systems called electro-anatomic mapping (EAM) that records and displays anatomic and electrical data on a virtual model of the heart in real time. Other strategies include pace mapping, where a QRS generated by electrical stimulation ('pacing') at various points within the heart is compared to the PVC. Once identified, ablation therapy is delivered to destroy the tissue at the SOO. This may involve thermal energy (radiofrequency or cryo-ablation) or emerging novel ablation energies.

The success of CA is dependent on the SOO of the PVC as well as patient and procedural characteristics, most importantly the presence and frequency of PVCs on the day of the procedure. A large multicentre study has reported success rates of 85%,[22] with similar success rates of 86% from registry data.[17]

Conclusion

PVCs are a common finding in the general population among both asymptomatic and symptomatic patients. In patients with a significant burden, symptoms or a clinical suspicion of cardiomyopathy, evaluating for the presence of SHD is essential. This involves a 12-lead ECG, ambulatory ECG monitoring and TTE, with a low threshold for CMR. Advanced imaging may be helpful in differentiating early cardiomyopathies, identifying scar, and for all patients with LV dysfunction. In patients without SHD, treatment is guided by symptoms and the presence of PVC-mediated LV dysfunction. Management options include antiarrhythmic medications or CA. CA is the most effective strategy for the durable suppression of PVCs. Early referral to cardiac electrophysiology should be considered in patients with a significant PVC burden to facilitate an informed, patient-centred decision regarding ongoing management.

CRediT authorship contribution statement

Delia Calian: Visualization, Data curation. **Reuben Yap:** Writing – original draft. **John Whitaker:** Writing – review & editing, Supervision. **Aldo Rinaldi:** Supervision. **Matthew Wright:** Supervision.

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Declaration of competing interest

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