Which Is The Appropriate Arrhythmia Burden To Offer RF Ablation For RVOT Tachycardias?

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Abstract

Premature ventricular complexes (PVCs) and ventricular tachycardia (VT) in patients with structurally normal hearts originate from the right ventricular outflow tract (RVOT) in the majority of cases. In the last few decades catheter ablation of these arrhythmias has been proven to be effective. RVOT VT/PVCs may cause disabling symptoms or arrhythmia induced cardiomyopathy. However, the PVC burden at which catheter ablation should be recommended is still controversial. What adds to the controversy is why some patients with only a low number of PVCs can be highly symptomatic and may even develop arrhythmia induced cardiomyopathy, whilst others may have a higher PVC/VT burden and remain asymptomatic and do not develop cardiomyopathy for a long period of time. Therefore, although catheter ablation of RVOT PVCs has high success and low complication rates, the time point of when ablation should be recommended is currently still under debate. This review discusses the treatment strategies and prognosis for RVOT tachycardias and focuses on the question of which arrhythmia burden is appropriate to offer RF ablation.

Introduction

The incidence of PVCs or non-sustained VTs (NSVTs) in subjects with apparently healthy hearts varies according to observational studies and is predominantly dependent on the sampling technique. PVCs were seen in 7.8% of the participants during evaluation of 12-lead ECGs in a large healthy military population, with a much lower incidence seen in younger patients below the age of 20 (4.6%) compared to those older than 50 years of age (21.7%). In another study published by Hinkle et al., the incidence of asymptomatic ventricular arrhythmias was 62% in a mixed population of healthy individuals and patients with known heart disease. Monomorphic PVCs are usually associated with a favourable prognosis, whereas polymorphic PVCs or NSVTs may indicate an increased risk of cardiovascular morbidity and mortality. In addition, the occurrence of PVCs or NSVTs during the exercise or recovery phases may be a predictor for poorer prognosis even when no structural heart disease is present. Furthermore, the prognostic implication of PVCs may vary depending on the patient’s age, baseline heart disease, left ventricular (LV) function and co-morbidities. In general however, patients with structurally normal hearts and aged less than 30 are not at increased cardiovascular risk, whereas PVCs may be associated with an increased risk in patients older than 30 years.

Generally, PVCs originate predominantly from the right or left outflow tract in young patients. In these patients with structurally normal hearts and right ventricular outflow tract (RVOT) PVC/VTs, MRI studies have shown variable results ranging from completely normal hearts to subtle structural wall abnormalities of the RVOT. PVCs originating from the RVOT have an inferior axis with left bundle branch block (LBBB) morphology, and a late R/S transition at V4 in the precordial leads. A QRS duration <140ms is suggestive of a PVC with a ‘septal’ origin, whereas a QRS duration >140ms favours a ‘free wall’ origin, particularly when notches are seen in the downstroke of the QRS of the inferior leads. In addition, Hoffmayer, et al. demonstrated that ventricular arrhythmias due to arrhythmogenic right ventricular cardiomyopathy (ARVC) have a significantly longer mean QRS duration in lead I, more often exhibit a precordial R/S transition at V4 in the precordial leads. A QRS duration <140ms is suggestive of a PVC with a ‘septal’ origin, whereas a QRS duration >140ms favours a ‘free wall’ origin, particularly when notches are seen in the downstroke of the QRS of the inferior leads. (Figure 1).

Due to the close anatomical relationship between the RVOT and left ventricular outflow tract (LVOT), the differences in the ECG characteristics can be subtle (Figure 2). Large R-wave amplitude and duration in V1-V2, and early transition in the precordial leads suggest a PVC/VT origin from the LVOT. In addition, Hoffman, et al. demonstrated that ventricular arrhythmias due to arrhythmogenic right ventricular cardiomyopathy (ARVC) have a significantly longer mean QRS duration in lead I, more often exhibit a precordial R/S transition at lead V6, and more often have notching in at least one ECG lead. In fact, a R/S transition at lead V6 was the most specific criterion for ARVC (100% specificity), and a QRS duration ≥120ms in lead I was the most sensitive (88% sensitivity).

Disclosures:
None.

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Typical ECG morphology of PVCs originating from the right ventricular outflow tract (RVOT).

Figure 1: Typical ECG morphology of PVCs originating from the right ventricular outflow tract (RVOT).

Clinical Presentation Of RVOT PVC/VTs

The clinical presentation of patients with RVOT PVC/VTs varies and usually occurs as follows:

1) Most patients report various symptoms such as dyspnea, reduced exercise capacity, palpitations, a racing heart rate, dizziness, pre-syncope and syncope.

2) Other associated symptoms include coughing, dysphagia and claudication. The development of tachycardia-induced cardiomyopathy with LV dilatation can worsen the clinical symptoms of heart failure and reduced exercise capacity.

3) Quality of life (QOL) is often reduced in patients with RVOT PVC/VTs. Radiofrequency (RF) ablation of PVCs and VTs in patients with structurally normal hearts has been shown to result in a significantly improved QOL in 6 out of 8 SF-36 domains.

4) Recurrent syncope may occur in patients with RVOT PVC/VT. In these patients and particularly those with short PVC coupling intervals or fast VT cycle lengths (CL), a so-called “malignant variant” of RVOT tachycardia has to be considered, which has a worse prognosis.

Cardiomyopathy Associated With RVOT PVC/NSVTs

General Aspects Of PVC/NSVT-Induced Cardiomyopathy

Cardiomyopathy induced by atrial tachyarrhythmias has been well described. The suggested underlying pathophysiology is consistent with animal models of tachycardia-induced cardiomyopathy. Furthermore, cardiomyopathy secondary to VT in patients with structurally normal hearts was initially described in the early 1990s, and the first reported case of a successfully reversed PVC-induced cardiomyopathy after catheter ablation was published in 2000 by Chugh et al. Although the mechanism for tachycardia-induced cardiomyopathy secondary to atrial tachyarrhythmias with fast ventricular activation is well understood, the mechanisms for PVC-induced cardiomyopathy are less clear. Besides LV dyssynchrony due to LBBB during PVCs, other causes such as increased oxygen consumption have been implicated. Furthermore, the so-called apical-to-basal "squeezing effect" in systole during physiological activation of the LV is disrupted during RVOT PVCs with LBBB morphology, which may further impair LV systolic output.

What Is The Arrhythmia Burden That Leads To Cardiomyopathy?

The initial case reports of patients with PVC-induced cardiomyopathy described recovered LV systolic function after successful catheter ablation in patients with a high monomorphic PVC burden arising from the RVOT (generally >20000 PVCs/24h). However, to date the important question of how many PVCs are required to cause PVC-induced cardiomyopathy is not yet completely answered. Whereas in one study from Carbeillera Pol et al., the PVC burden was not associated with the development of cardiomyopathy (PVC burden >10% was used as inclusion criteria in this study), several other studies showed correlation between PVC frequency and cardiomyopathy.

In most series, a higher PVC burden was associated with a higher incidence of impaired LV systolic function. Del Carpio Munoz et al. demonstrated that patients with impaired LV function had an increased PVC burden (29.3±14.6% vs 16.7±13.7%, P=0.004). Baman et al. evaluated the impact of PVC burden on reduced LV systolic function in 57 patients with PVCs predominantly originating from the RVOT. The initial mean LV systolic function in this patient population was 35%, which improved by at least 15% or normalized after successful RF ablation, defined as suppression of the PVC burden >80%. The PVC burden was significantly higher in patients with suppressed LV systolic function compared to those without LV dysfunction (33% vs 13%). A PVC burden cut-off of >24% best separated patients likely to develop impaired LV function from those less likely to develop LV dysfunction (sensitivity 79%, specificity 78%, area under curve 0.89). The authors also stated that a PVC burden cut-off of 16% would result in a higher sensitivity of 90% for PVC-induced cardiomyopathy, but the specificity would decrease to 58%. Importantly, patients with preserved LV function but a dilated LV were associated with an intermediate PVC burden of 22%, which was significantly lower compared to patients with LV systolic dysfunction. In these patients, a dilated LV may mask LV function, and may not accurately reflect left ventricular function. The authors suggested that a PVC burden cut-off of >24% best separated patients likely to develop impaired LV function from those with normal LV systolic function and dimensions. Another interesting finding was that the development of cardiomyopathy was independent of the site of PVC origin. Thus, patients with an epicardial or left-sided PVC origin also developed impaired LV function. This finding was in line with reports from other investigators who described cardiomyopathy induced by monomorphic PVCs originating from other locations such as the LV or from above the pulmonary valve that resolved after successful catheter ablation.

In another study from Baman et al., successful RF ablation could be achieved in 82% of patients with PVC/VTs. In these patients,
RF ablation not only improved LV systolic function, it also reduced LV diameters (end-systolic and end-diastolic diameters) after a follow-up period of 6 months. Notably, cardiomyopathy was also observed in one patient with a PVC burden of only 10%. This is in line with results from several other studies and case reports. In a study published by Yarlagadda et al., even a PVC burden of only 5500 per day resulted in cardiomyopathy in one patient. Shanmugam et al. reported resolution of significant LV systolic impairment after successful RF ablation in a patient with <5000 PVCs per day (i.e. a PVC burden of 4%/24h). In contrast, Takemoto et al. reported a patient with a PVC burden <10% who did not have improvement of LV dysfunction after successful ablation. This suggests that PVCs in some patients may be associated with the early stages of DCM or other cardiac diseases. Kanei et al. evaluated the prevalence of cardiomyopathy in patients with PVCs as assessed by 24-h holter monitoring categorized as follows: low PVC burden of <1000, intermediate PVC burden of 100-10000 and high PVC burden of >10000. The prevalence of reduced LV function was associated with the number of PVCs (4%, 11% and 32%, respectively) in this retrospective study.

Another entity called interpolated PVCs might be associated with PVC-induced cardiomyopathy. Olgun et al., evaluated 21 patients with PVC-induced cardiomyopathy for the presence of interpolated PVCs. Although overall a higher PVC burden was seen in patients with PVC-induced cardiomyopathy, interpolated PVCs were found to be an independent predictor for cardiomyopathy. The mechanism for this phenomenon is not clear, but it is possible that a higher heart rate in sinus rhythm may lead to a higher likelihood for PVC occurrence. Therefore in these patients, the PVC burden itself may not be a reliable criterion for deciding whether ablation should be performed or not.

Specific Aspects Of PVC/NSVT-Induced Cardiomyopathy

In a consecutive series of 294 patients with idiopathic PVCs evaluated by Yokokawa et al., the QRS duration found to be significantly longer in patients who had reversible cardiomyopathy after RF ablation. Importantly, a QRS-duration of >150ms identified patients with reversible cardiomyopathy compared with those without reversible cardiomyopathy with a sensitivity of 80% and specificity of 52%. Although PVCs with an epicardial origin had the longest QRS duration, an epicardial origin itself was independently associated with a reversible cardiomyopathy. Another study published 2011 demonstrated that patients with PVC-induced cardiomyopathy had broader QRS complexes than those without cardiomyopathy. A reasonable explanation for this phenomenon could be that a wider QRS duration leads to a higher degree of dyssynchrony in the heart. Furthermore, symptom duration in patients with PVCs may also be associated with the occurrence of PVC-induced cardiomyopathy. Carbeillera Pol et al. found that patients with PVCs with a broader QRS complex (>150ms) are at higher risk of developing PVC-induced cardiomyopathy after RF ablation. Furthermore, a non-right-sided origin was also associated with irreversible cardiomyopathy. Deyell et al. found that a longer QRS duration was associated with a lower likelihood of recovery from PVC-induced cardiomyopathy. The hypothesis was that a combination of both fibrosis and myofiber disarray may play a role in the prolongation of PVC QRSs.

The incidence of PVC-induced cardiomyopathy is higher in older patients and the pathophysiology is less well understood. It is often more difficult to determine whether PVCs are a result of reduced LV function or the cause. It is well described that PVCs/NSVTs are a common finding on 24-hour holter recordings in patients with ischemic or dilated cardiomyopathy. The indication for primary ICD implantation in these patients to reduce the arrhythmogenic risk for SCD is dependent on LV function and not arrhythmia burden. The increased risk for SCD is present even if no arrhythmias have been documented.

When Does LV Dysfunction Resolve?

In patients with PVC-induced cardiomyopathy, an improvement of systolic function can usually be observed post-ablation after a short follow-up period of approximately 3 months. When the LV dysfunction completely resolves is less clear and may vary, as demonstrated by Yokokawa et al., who evaluated the time course of LV function recovery in 87 patients with PVC-induced cardiomyopathy after successful RF ablation, defined as a reduction of >80% of the initial PVC burden (Yokokawa et al HR 2013). The majority (68%) of patients experienced LV function recovery within 4 months post-ablation, whilst in the remaining patients LV recovery was seen after a mean of 12±9 months. Importantly, one patient had LV function normalization only after 45 months. In the patients with delayed improvement of LV function, QRS width was significantly longer. Furthermore, an epicardial origin was an independent predictor for delayed LV recovery from PVC-induced cardiomyopathy. Also, a right-sided PVC origin was associated with faster recovery of LV function. Therefore, it is recommended that final evaluation of LV function recovery after successful PVC ablation should only be performed after a minimum of one year. Although animal studies demonstrated no structural changes such as fibrosis, delayed remodelling or prolongation of Ca2+ channel function has been hypothesized to cause slower LV function recovery in some patients.

Medical Therapy For The Treatment Of PVC/NSVTs

Data regarding medical therapy for the treatment of idiopathic PVCs or VTs is sparse. Duffe et al. described four patients where PVC-induced cardiomyopathy was successfully treated with beta-blockers or amiodarone. According to the current guidelines for the management of symptomatic PVCs, beta-blockers are the drug of choice. However, the efficacy of beta-blocker therapy (namely...
### Table 1: Overview of relevant studies about RVOT ablation.

<table>
<thead>
<tr>
<th>Author Author and year of publication</th>
<th>Patients with reduced LV function, n (%)</th>
<th>PVC burden before ablation</th>
<th>PVC burden after ablation</th>
<th>Success rate n (%)</th>
<th>Systolic ejection fraction at baseline</th>
<th>Systolic ejection fraction after ablation</th>
<th>Patients with normalized systolic LV function, n (%)</th>
<th>LVEDD before ablation (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chugh et al. 2000</td>
<td>25.000-56.000 PVCs/24h</td>
<td>1.100-1.800 PVCs/24h</td>
<td>1/1 (100)</td>
<td>43%</td>
<td>58%</td>
<td>1 (100)</td>
<td>65</td>
<td></td>
</tr>
<tr>
<td>Redfearn et al. 2003</td>
<td>31.000 PVCs/24h</td>
<td>1 PVC/24h</td>
<td>1/1 (100)</td>
<td>Mildly-moderately reduced</td>
<td>54%</td>
<td>1 (100)</td>
<td>59</td>
<td></td>
</tr>
<tr>
<td>Shiraiishi et al. 2002</td>
<td>50.000 PVCs/24h</td>
<td>1.100-1.800 PVCs/24h</td>
<td>1/1 (100)</td>
<td>34%</td>
<td>normalized</td>
<td>1 (100)</td>
<td>65</td>
<td></td>
</tr>
<tr>
<td>Efremidis et al. 2008</td>
<td>&gt;50.000 PVCs/24h</td>
<td>No PVCs/24h</td>
<td>1/1 (100)</td>
<td>40-45%</td>
<td>55%</td>
<td>1 (100)</td>
<td>n.a.</td>
<td></td>
</tr>
<tr>
<td>Deyell et al. 2012</td>
<td>(Inclusion Criterion PVC burden ≥ 10%/24h)</td>
<td>34.202 ±15.493 PVCs/24h in patients with reversible LV function</td>
<td>n.a.</td>
<td>37/44 (84%; 4 lost to FU)</td>
<td>38.2% in Pts with reversible LV function</td>
<td>Median improvement of LV function 17.5% (IQR 10–40) for patients with reversible LV dysfunction</td>
<td>24/48 (50)</td>
<td>56±0.7</td>
</tr>
<tr>
<td>Ezatt et al. 2007</td>
<td>30000 PVCs/24h (30%/24h)</td>
<td>2.022 PVCs/24h</td>
<td>1/1 (100)</td>
<td>40%</td>
<td>62%</td>
<td>1 (100)</td>
<td>65</td>
<td></td>
</tr>
<tr>
<td>Olgun et al. 2011</td>
<td>21/51 (41.2)</td>
<td>PVC burden 30±10%/24h</td>
<td>n.a.</td>
<td>42/51 (82)</td>
<td>37±10%</td>
<td>55±9%</td>
<td>21/21 (100)</td>
<td>n.a.</td>
</tr>
<tr>
<td>Takemoto et al. 2005</td>
<td>14/40 (35)</td>
<td>PVC burden &gt;20%/24h (mean 34±3%)</td>
<td>PVC burden 1.3 ±0.9</td>
<td>37/40 (92.5)</td>
<td>66±2%</td>
<td>72±2%</td>
<td>13/14 (93)</td>
<td>54±1</td>
</tr>
<tr>
<td>Yarlagadda et al. 2005</td>
<td>8/27 (29.6)</td>
<td>1754±11479 PVCs/24h</td>
<td>50±722</td>
<td>23/27 (85)</td>
<td>39±6%</td>
<td>62±6%</td>
<td>7/8 (87.5)</td>
<td>n.a.</td>
</tr>
<tr>
<td>Yokokawa et al. 2013</td>
<td>87/264 (33)</td>
<td>PVC burden 26 ±11%/24h</td>
<td>2% ± 4%/24h</td>
<td>75/87 (86.2)</td>
<td>39±10%</td>
<td>59±4%</td>
<td>51/ 75 (68)</td>
<td>56±6</td>
</tr>
<tr>
<td>Baman et al. 2010</td>
<td>57/174 (32.8)</td>
<td>PVC burden 33 ±14%/24h</td>
<td>1.9% ±4.4%/24h</td>
<td>146/174 (84)</td>
<td>35±9%</td>
<td>54±10%</td>
<td>46/57 (81)</td>
<td>57±6</td>
</tr>
</tbody>
</table>

Atenolol and metoprolol is generally modest, with a reduction of PVC burden between 10-25%,72, 73, 74 The efficacy of calcium channel antagonists such as verapamil varies in several reports, with reasonable efficacy in patients with idiopathic VT,70, 75, 76 but it is less effective in patients with only PVCs, most likely as a result of different underlying mechanisms.74 Following these, Class I antiarrhythmic drugs such as propafenone or flecainide are recommended. Although treatment of symptomatic patients with PVCs in structurally normal hearts with these antiarrhythmic drugs may be reasonable,71 they are contraindicated in patients with cardiomyopathy due to their proarrhythmic effects.77 In these patients, beta-blockers and amiodarone are the only antiarrhythmic drugs available. However, due to the frequent and significant side effects associated with amiodarone,78 it should only be administered in patients refusing catheter ablation or after failed catheter ablation. Since catheter ablation of RVOT PVC/VTs is a highly effective therapy with a very low complication rate, this should be the treatment of choice.

A recent study by Zhong et al. further highlights the superiority of RF ablation compared with antiarrhythmic drug therapy for the treatment of idiopathic PVCs. In this study, RF ablation reduced the PVC burden by 93%, which was significantly higher compared to treatment with Class I and III antiarrhythmic drugs.79 How To Differentiate Cardiomyopathy Associated With Structural Heart Disease From PVC-Induced Cardiomyopathy?

At times it may not be clear if reduced LV function is due to the early stages of dilated cardiomyopathy or undetected ischemic heart disease, myocarditis or as a result of frequent PVCs in patients without structural heart disease. We propose the following recommendations for the initial evaluation of such patients:

Firstly, patients with idiopathic PVCs are usually young, with a higher incidence seen in women.71 Echocardiographic findings generally show reduced LV function with global hypokinesia, but without local wall motion abnormalities and with or without enlarged LV diameters but preserved LV wall thickness.53 Coronary angiography rules out coronary heart disease in these patients.53, 59 As described above, although a low PVC burden could also cause cardiomyopathy, the PVC burden is usually high (>24% or >20000 PVCs/24hrs), with a predominant monomorphic PVC morphology (literature) most often emanating from the RVOT.53 Polymorphic PVCs are usually a sign of a more complex tachyarrhythmia or extensive myocardial damage, and are more often seen in patients with structural heart disease.80 Whenever the cause of the cardiomyopathy remains unclear, one should consider antiarrhythmic drug therapy for 3-6 months with the aim for PVC suppression to see if an improvement of LV systolic function occurs. The antiarrhythmic drug of choice is amiodarone as most others are contraindicated,71 and short-term treatment with this drug is reasonable in selected patients.

**Success Rate And Complications Of RF Ablation**

In general, the success rate of catheter ablation of arrhythmias originating from the RVOT is reported to be high.81-83 In most studies, the acute success rate is reported to be >80%.71 After initial successful ablation the recurrence rate is low, generally not exceeding 5% even after long-term follow-up.21, 22, 71, 84-86 The acute success rate of catheter ablation is usually lower in patients who have only infrequent PVCs or non-inducibility of the clinical arrhythmia during the procedure. The pathophysiologic basis for idiopathic PVCs, NSVTs and ventricular tachycardias vary, but are considered to be mostly related to triggered activity due to intracellular calcium overload mediated by cyclic-AMP. Therefore, an isoproterenol infusion may be required to induce these arrhythmias during an electrophysiology study.87-89 Difficulties in inducing the clinical arrhythmia even after isoproterenol administration may decrease the...
success rate of catheter ablation in these patients due to the lack of a clear endpoint. When there is non-inducibility of the clinical VT, ablation of monomorphic PVCs with the same morphology as the documented arrhythmia to eliminate the arrhythmia trigger may be a reasonable strategy. In addition to activation mapping, pace-mapping may be performed. Non-contact mapping can be considered in difficult cases to identify the PVC origin even in patients with rare PVCs. As deep sedation is considered by some investigators to reduce the spontaneous PVC burden, reducing the amount of sedation and analgesia can often reduce suppression of PVCs and increase their frequency.

In general, catheter ablation of idiopathic PVCs originating from either the RVOT or left-sided structures is usually considered to be safe. Zhong et al. recently published a procedure-related complication rate of 5.6% (12 patients). The use of intracardiac echocardiography may help to better define the anatomy in complex cases during ablation of RVOT arrhythmias and has been previously shown to be helpful, particularly during ablation of tachycardias from the LVOT.

Although RF energy is still the gold standard for ablation within the RVOT, with high efficacy and safety, cryoablation is a reasonable alternative that allows for almost pain-free ablation.

What Is The Arrhythmia Burden To Offer RF Ablation?

RF Ablation To Treat Symptomatic Patients

Catheter ablation in symptomatic patients with PVC/VTs has been described for many decades and is highly effective. Catheter ablation in symptomatic patients with PVC/VTs is superior to antiarrhythmic drug treatment and is safe. Therefore we recommend that ablation may be offered to all patients regardless of the PVC burden. In patients with asymptomatic PVC/VTs and is superior to antiarrhythmic drug treatment.

RF Ablation In Asymptomatic Patients With Preserved Systolic Ejection Fraction

As described above, RVOT PVCs may result in reduced LV systolic function or even PVC-induced cardiomyopathy. Superior to antiarrhythmic drug therapy, catheter ablation is the treatment of choice for PVC-induced cardiomyopathy. The cutoff is still not yet defined, however two large studies have demonstrated that a cutoff value of 20% to 24% PVCs per 24 hours is associated with an increased risk of developing reduced LV function and cardiomyopathy. Therefore, in patients with such a high volume of PVCs, prophylactic catheter ablation may be proposed even when patients are asymptomatic. Furthermore as described above, even a PVC burden of less than 5000 PVCs per 24 hours can result in impaired LV systolic function. Therefore, also in this last condition, close monitoring of these patients is recommended to screen for early forms of impairment of LV function. In addition, a complete cardiac work-up should be performed to exclude any underlying early-stage structural cardiomyopathy that may require a different therapeutic strategy.

RF Ablation In Asymptomatic Patients With Reduced Systolic Ejection Fraction

Whenever reduced LV systolic function is observed in a patient with frequent monomorphic PVCs, catheter ablation should be offered to prevent worsening of LV systolic function or development of heart failure symptoms.

Because LV function impairment has also been described despite a lower PVC burden, catheter ablation should also be offered. Therefore, catheter ablation should be offered to all patients with reduced LV systolic function to prevent further progression of LV dysfunction and to normalize cardiac function. Given that LV function recovery may take up to one year or longer, RF ablation should be recommended without delay in these patients.

Special Considerations For Particular Conditions

The prognosis in patients with RVOT PVC/VTs is usually favourable. However special attention should be given to patients fulfilling the criteria of potentially malignant variants of RVOT arrhythmias. These include symptoms of recurrent syncpe, PVCs with very short coupling intervals, VTs with short cycle lengths, or documented polymorphic RVOT PVCs/VTs or “benign” RVOT PVCs in the presence of channelopathy or structural heart disease. Whenever one of the above-mentioned criteria is present, further treatment should be modified with an early recommendation for catheter ablation independent of the arrhythmia burden or LV function, associated with the evaluation of the need for further treatment based on the underlying cardiopathy.

Conclusion

Catheter ablation is an excellent option for the treatment of RVOT PVC/VTs and is superior to antiarrhythmic drug treatment. High success rates of >80% are achieved with catheter ablation, even during long-term follow-up and significant complications are rare. Therefore we recommend that ablation may be offered to all patients with symptomatic RVOT PVC/VT refractory to antiarrhythmic drug treatment or when antiarrhythmic drugs are not desired by the patient regardless of the PVC burden. In patients with asymptomatic RVOT PVCs and preserved LVEF, catheter ablation should be considered if the arrhythmia burden is >20-24% to prevent the development of tachycardia-induced cardiomyopathy. In patients with reduced LVEF where a possible etio-pathogenetic link between the PVCs and impaired LVEF is suspected, catheter ablation should be considered unless contraindicated.

Special attention should be given to patients with syncope, very short RVOT-PVC coupling intervals, fast RVOT-VT or polymorphic PVC/VTs. In these cases, evaluation should focus on the exclusion/confirmation of the presence of an underlying structural heart disease with subsequent treatment directed at the management of potential life-threatening arrhythmias.

References


