Chapter 7

Beneficial effects of catheter ablation on left ventricular and right ventricular function in patients with frequent premature ventricular contractions and preserved ejection fraction

Adrianus P. Wijnmaalen, Victoria Delgado, Martin J. Schalij, Carine F.B. van Huls van Taxis, Eduard R. Holman, Jeroen J. Bax, Katja Zeppenfeld

* A.P. Wijnmaalen and V. Delgado contributed equally to this article and are shared first author.

Heart. 2010 Aug;96(16):1275-80.

Abstract

Objective: Improvement of left ventricular (LV) ejectionfraction (EF) after radiofrequency catheter ablation (RFCA) of frequent premature ventricular contractions (PVCs) has been reported. However, most patients with frequent PVCs have normal LVEF. In these patients subtle and early forms of PVC induced left and right ventricular (RV) impairment may not be detected by standard echocardiographic techniques. This study aimed to assess the effect of frequent PVCs on ventricular function in patients with preserved LVEF.

Methods: Forty-nine patients (30male, 49±16years) with recent onset (median 1.2years), frequent PVCs (burden 26±13%) and 25 healthy controls were studied. Thirty-four patients with PVCs underwent successful RFCA. Two-dimensional echocardiography was performed at baseline and follow-up. LV volumes and LVEF were calculated by Simpson's rule. Tricuspid annulus plane systolic excursion and fractional area change were calculated to assess RV function. Multidirectional LV strain (radial, circumferential, longitudinal) and RV free wall longitudinal strain were calculated by two-dimensional speckle tracking imaging. At baseline LVEF, volumes and RV dimensions were normal in patients and controls.

Results: Speckle tracking imaging demonstrated reduced LV and RV strain in PVC patients as compared to controls. At follow-up there were no changes in LVEF, LV volumes and RV dimensions and function in successfully treated by RFCA and untreated patients. However, radial, circumferential and longitudinal strain improved significantly in patients after RFCA but remained unchanged in untreated patients.

Conclusions: Frequent PVCs can induce subtle cardiac dysfunction detected by speckle tracking imaging analysis in patients without apparent cardiomyopathy. RFCA can successfully eliminate PVCs and improve cardiac function.

Introduction

Symptomatic ventricular ectopy is frequently encountered in clinical practice. The arrhythmia occurs commonly in patients without structural heart disease and is considered to be without prognostic significance.[1]

In some patients, frequent idiopathic premature ventricular contractions (PVCs) can cause moderate to severe left ventricular (LV) dysfunction.[2-4] Elimination of these PVCs by radiofrequency catheter ablation (RFCA) may result in improved LV ejection fraction (EF).[2-4] The majority of patients presenting with frequent symptomatic PVCs has normal LV volumes and LVEF. It has however been shown that impairment of LV function may evolve after several years of follow-up.[5] Small series demonstrated a reduction of LV dimension after successful RFCA in patients with normal LV function using conventional echocardiography assessment.[6, 7] This finding suggests that frequent PVCs may already have an early subclinical detrimental effect on LV function which may not be unmasked with conventional echocardiography. Whether frequent PVCs have an adverse effect on right ventricular (RV) function is unknown.

Recently, two-dimensional (2D) speckle tracking strain imaging has been introduced.[8, 9] This novel technique enables accurate detection of subtle abnormalities in ventricular function that are not revealed by conventional echocardiographic parameters such as LVEF and RV fractional area change.[10] In addition, 2D speckle tracking strain imaging allows angle-independent evaluation of multidirectional LV strain in radial, circumferential, and longitudinal directions and RV longitudinal strain.[11]

The purpose of this study was to assess the effect of recent-onset, frequent PVCs on RV and LV function in patients with preserved LVEF using 2D speckle tracking strain imaging. In addition, the effect of successful RFCA of frequent PVCs on LV and RV function was evaluated.

Methods

Study population and study protocol

Forty-nine consecutive patients with symptomatic, frequent PVCs (>5% PVC on 24h Holter monitoring) were included. All patients underwent extensive baseline evaluation to rule out structural heart disease including a clinical history with regard to the onset of symptoms, 12-lead electrocardiogram (ECG), exercise testing, 24-hour Holter monitoring and 2D transthoracic echocardiography.

The 12-lead ECG QRS duration, morphology and PVC coupling interval were evaluated. The PVC burden was calculated by dividing the number of PVCs by the total number of beats on Holter recording.

Echocardiographic reference values were obtained from a group of 25 individuals with structural normal hearts. The controls were frequency matched to patients by age, gender, body surface area and LV function. The control group included individuals who were referred for evaluation of chest pain and who had normal echocardiograms. Those individuals referred for echocardiographic evaluation of known valvular heart disease, murmur, syncope, arrhythmias, congestive heart failure or cardiac transplantation were excluded.

Radiofrequency catheter ablation

After informed consent catheter mapping and ablation was performed in the post-absorptive, non-sedated state. Antiarrhythmic drugs were discontinued for 5 half-lives, with the exception of amiodarone.

Mapping was facilitated by an electroanatomical mapping system (CARTO) using a transvenous or retrograde aortic approach. At the site of earliest activation based on the onset of bipolar electrograms with a local unipolar QS-pattern pace-mapping was performed to confirm a ≥11/12 lead QRS pace-match.

Radiofrequency energy was delivered at the site of earliest activation and best pace-map with the target temperature set at 60°C and maximum power output of 50W. After ablation programmed electrical stimulation was performed before and during the administration of isoproterenol (2-10μ/min) to confirm that PVCs were not inducible by adrenergic stimulation. Complete procedural success was defined as the absence of spontaneous or inducible PVC for at least 45 minutes after ablation.

Echocardiography

Two-dimensional echocardiography was performed using a commercially available system (Vivid-Seven, General Electric Vingmed, Horten, Norway) equipped with a 3.5-MHz transducer. Data acquisition was performed in the left lateral decubitus position at a depth of 16cm in the parasternal and apical views. Standard M-mode, grey-scale 2D and color-Doppler images, triggered to the QRS complex, were acquired and saved in cine-loop format for off-line analysis (EchoPAC 7.0.0., GE Medical Systems, Horten, Norway). All echocardiographic measurements were performed on sinus beats, avoiding the first postextrasystolic beat if feasible.

Left ventricular end-diastolic and end-systolic volumes were measured at the apical 2- and 4-chamber views by modified Simpson's rule and LVEF was derived.[12] RV dimensions were measured according to current guidelines.[12] From the apical 4-chamber view, the mid-cavity and basal RV diameters as well as RV longitudinal diameter were measured at end-diastole. From the parasternal short-axis images, the end-diastolic RV outflow tract diameter was measured proximal to the pulmonary valve. RV systolic function was evaluated by calculating the fractional area change and measuring the tricuspid annular plane systolic excursion (TAPSE) index from the apical 4-chamber view.[12]

Two-dimensional speckle tracking imaging analysis

Multidirectional analysis of LV strain (radial, circumferential and longitudinal directions) was performed using 2D speckle tracking imaging. Standard grey-scale 2D images were acquired at a high frame rate to assure adequate tracking of the speckles equally distributed within the myocardium. Myocardial strain can be calculated by measuring the change of the position of the speckles within a myocardial segment along the cardiac cycle.[13, 14]

Applied to the short-axis of the LV, radial strain assesses the thickening and thinning of the myocardial wall whereas circumferential strain assesses the shortening and lengthening of the myocardium along the curvature of the LV wall. The mid-ventricular short-axis of the LV is divided in 6 segments and the values of radial or circumferential strain are derived from the average of the 6 segmental peak systolic strain values (Figure 1A and B).

Applied to LV apical images (2-, 4-chamber and long-axis views), longitudinal strain assesses the shortening of the LV resulting from the excursion of the mitral annulus plane towards the LV apex. Each LV apical view is divided in 6 segments and the global longitudinal strain value is derived from the average of the 18 segmental peak systolic strain values (Figure 1C).

Myocardial deformation of the RV was assessed in the longitudinal direction.[15] From a modified apical 4-chamber view, focused on the RV, longitudinal strain of the free-wall was evaluated. The RV free-wall is divided in 3 segments and the global longitudinal strain value is obtained from the average of the 3 segmental peak systolic strain values (Figure 1D).

All echocardiographic analyses were performed by an independent observer blinded to the clinical history and the treatment. The inter-observer agreement for 2D speckle tracking strain measurements have been previously reported[16, 17], being 6.5±5.4 for radial strain, 2.3±2.4 for circumferential strain, 0.9±1.0 for longitudinal strain and 0.16±3.6% for RV longitudinal strain.

Statistical analysis

Continuous variables are expressed as mean±SD. Categorical variables are expressed as frequencies(%). Comparisons between controls and patients were performed by Student t-test for unpaired data. Patients treated with RFCA and patients who were not treated with RFCA were compared by using Mann-Whitney U-test for unpaired data. Changes in LV and RV dimensions and function at follow-up were compared with Student t-test and Wilcoxon Signed Ranks test for paired data, as appropriate. A p-value <0.05 was considered statistically significant. All statistical analyses were performed with SPSS software (version 16.0, SPSS Inc., Chicago, Illinois).

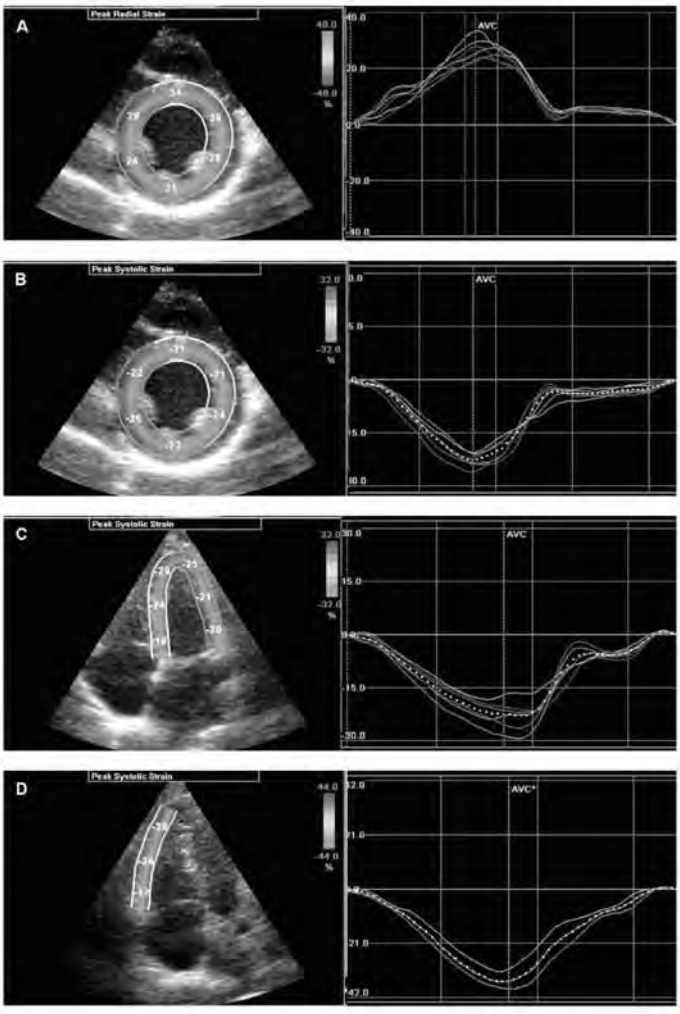

Figure 1. Multidirectional strain analysis with 2-dimensional speckle tracking imaging. In the LV, myocardial strain can be measured in the 3 directions with 2-dimensional speckle tracking imaging. Radial (panel A) and circumferential (panel B) strain are calculated at the mid-ventricular short axis view of the LV. The LV is divided in 6 segments and the segmental peak systolic strain values with the corresponding time-strain curves along the cardiac cycle are obtained. Longitudinal strain of the LV is calculated at the 4-chamber, 2-chamber and long-axis apical views (panel C). In each apical view, the LV is divided in 6 segments, obtaining the segmental peak systolic strain values and the corresponding time-strain curves. Longitudinal strain of the RV free wall (panel D) is measured at modified 4-chamber view, focused on the RV. The free wall is divided in 3 segments, providing the segmental peak systolic strain value and the time-strain curves along the cardiac cycle.

Results

Patient characteristics

Baseline characteristics of the 49 patients (30 men, mean age 49±16 years) are summarized in Table 1. The median duration of symptoms before the first evaluation was 1.2 years (interquartile range 0.4–3 years). In patients who underwent RFCA the 24-hour Holter monitoring was performed 3.0 months (interquartile range 1.0-5.6) prior to the RFCA procedure.

The burden of PVCs was 26±13% of total beats. The PVCs had one dominant morphology in all patients. The mean QRS duration of PVC was 175±24ms. The frontal plane axis was left inferior in 24(49%), left superior in 1(2%), right inferior in 23(47%) and right superior in 1(2%) patient. The average coupling interval was 498±115 ms. No evidence of ischemia was found on exercise testing in any of the patients.

The control group included 25 individuals with normal echocardiograms. By definition, controls were comparable to patients in terms of gender (14(56%) men, p=0.665), age (46±9 years, p=0.333), body surface area (1.8±0.1m^2, p=0.080) and LVEF (58±5%, p=0.201).

Table 1. Baseline characteristics

	n = 49
Age (years)	49 ± 16
Gender (M/F)	30 (61%) / 19 (39%)
Body surface area (m^2)	1.9 ± 0.2
PVC-burden (%)	26 ± 13
Cardiovascular risk factors	
Hypertension	10 (20%)
Hypercholesterolemia	10 (20%)
Family history CAD	13 (27%)
Smoking	7 (14%)
Medical treatment	
Beta-blockers	16 (33%)
ACEI	15 (31%)
Ca-antagonists	5 (10%)
Sotalol	11 (22%)
Amiodarone	1 (2%)
Class I antiarrhythmics	4 (8%)

ACEI = angiotensin-converting enzyme inhibitors, PVC = premature ventricular contraction, CAD = coronary artery disease

Mapping and Radiofrequency catheter ablation

Forty patients were scheduled for mapping and ablation; in nine no RFCA was attempted due to patients' preference. Thirty-four patients could successfully be ablated with a median of 3 RF applications (range 1–14). The PVCs originated from the RV in 24(71%) and the LV

Table 2. Baseline echocardiography and 2-dimensional speckle tracking strain analysis

	Controls (n = 25)	PVC-group (n = 49)	p-value
Heart rate (bpm)	68 ± 10	67 ± 10	0.588
LV end-diastolic volume (ml)	117 ± 29	123 ± 35	0.391
LV end-systolic volume (ml)	49 ± 13	55 ± 19	0.102
LV ejection fraction (%)	58 ± 5	56 ± 7	0.201
RV outflow tract diameter (mm)	29 ± 3	29 ± 4	0.973
RV basal diameter (mm)	31 ± 3	30 ± 4	0.439
RV mid-level diameter (mm)	30 ± 6	29 ± 5	0.421
RV longitudinal diameter (mm)	70 ± 10	71 ± 10	0.481
RV fractional area change (%)	41 ± 9	42 ± 8	0.628
TAPSE (mm)	2.4 ± 0.3	2.4 ± 0.5	0.867
LV Radial strain (%)	43.3 ± 12.7	30.5 ± 12.9	<0.0001
LV Circumferential strain (%)	-20.2 ± 3.1	-15.8 ± 4.0	<0.0001
LV Longitudinal strain (%)	-20.1 ± 1.0	-17.4 ± 3.0	<0.0001
RV Longitudinal strain (%)	-30.1 ± 4.4	-23.8 ± 6.7	<0.0001

LV = left ventricular; PVC = premature ventricular complex; RV = right ventricular; TAPSE = tricuspid annular plane systolic excursion.

in 10(29%) patients. The site of successful RF application was the RV outflow tract in 23, the pulmonary artery in 1, the LV outflow tract in 2, the mitral annulus in 1, the anterior cardiac vein in 3, the aortic cusps in 3 and the Purkinje system in 1 patient. In 2 of these patients 2 different morphologies could be successfully targeted in adjacent areas (RVOT in 1 and Aortic cusps in 1). In 5/6 patients with ablation failure the site of origin could not be determined by an endocardial approach including mapping within the cardiac veins (>1 PVC morphology in 2) and in 1 patient no ablation attempt was made due to the proximity of the His bundle. Procedure and fluoroscopy times were 150±60 min and 17±15 min respectively. In the successfully ablated patients the burden of PVCs was reduced to 0.2±0.8% confirming freedom of PVCs at repeated Holter monitoring 4.1 months (interquartile range 1.4-9.2 months) after RFCA. In contrast, in the 15 patients in whom no mapping and/or ablation attempt was made the PVC burden was stable at follow-up. No complications were observed.

Baseline echocardiography and 2D speckle tracking strain analysis

At baseline, all patients had a non-dilated LV with preserved LVEF (56±7%) and no differences were observed as compared to controls (Table 2). In addition, patients had comparable

RV dimensions and function to controls (fractional area change 42±8% and TAPSE index 2.4±0.5mm) (Table 2).

Multidirectional 2D speckle tracking assessment of the LV demonstrated that patients had significantly impaired LV strain in all the 3 directions (radial, circumferential and longitudinal). Furthermore, patients had lower values of RV longitudinal strain as compared to controls (Table 2). In 7 patients, 2D speckle tracking analysis was performed during sinusal post-extrasystolic beat. However, this had not significant impact on the values of multidirectional strain of the overall population.

Effects of PVC origin

To evaluate the impact of PVC origin on RV and LV function, patients with PVCs originating from the RV confirmed by successful RFCA (n=24) were compared to patients with LV origin (n=10). No differences were observed in LVEF, LV end systolic and diastolic function, RV fractional area change and TAPSE. Myocardial strain was decreased in all the directions to a similar extend in patients with LV and RV site of origin: LV circumferential strain (-16.3±4.0% vs. -15.6±3.3%, p=0.5), LV longitudinal strain (-18.3±3.1% vs. -17.1±2.3%, p=0.7), LV radial strain (30.1±13.8% vs. 33.1±15.6%, p=0.2) and RV longitudinal strain (-25.8±8.4% vs. -22.1±2.3%, p=0.07).

PVC burden and echocardiographic findings

To account for the effect of PVC burden the 30(61%) patients with a PVC burden of >20% (34±9.7%) were compared to 19(39%) patients with a PVC burden <20% (13.3±4.7%). Patients with a high PVC burden tended to have lower LVEF (55±8% vs. 58±6%, p=0.07) and larger LV end-diastolic volume (130±37ml vs. 113±31ml, p=0.13) and LV end-systolic volume (59±20ml vs. 49±17ml, p=0.09) as compared to patients with a moderate PVC burden, however these differences were not significant. Both groups showed comparable values of LV circumferential strain (-16.3±4.4% vs. -15.8±4.0%, p=0.9), radial strain (-31.9±14.2% vs. -32.1±16.0%, p=1.0), longitudinal strain (-17.1±3.5% vs. -17.9±2.2%, p=0.9) and RV longitudinal strain (-23.3±6.0% vs. -25.2±8.3, p=0.3).

Effect of RFCA on LV and RV function

After a median follow up of 13 months (interquartile range 5–22 months) a second echocardiogram was obtained in all patients. The follow-up, the 34 patients successfully treated with RFCA showed a significant decrease in LV end-systolic volume (from 56±21ml to 49±15ml, p=0.018). Changes in LV end-diastolic volumes and LVEF (from 57±8 ml to 59±5%, p=0.245) were not significant. However, with 2D speckle tracking imaging, a significant improvement of strain values was observed in all the 3 directions, with normalization of LV multidirectional myocardial strain. In addition, although there were no significant changes

in RV dimensions and function, RV longitudinal strain also improved at follow-up, with normalization of the longitudinal strain values in successfully treated patients. (Table 3)

In contrast, the group of 15 patients in whom RFCA could not successfully be performed or no RFCA attempt was made the PVC burden was stable and no changes in LV dimensions and function, assessed either by LVEF or multidirectional strain were found. Furthermore, no changes in RV dimensions, function and deformation properties were observed (Table 3).

Discussion

The major finding of the present study is that patients with frequent PVCs of recent onset and normal LV volumes, LVEF and RV dimensions show subtle LV and RV dysfunction. This cardiac impairment normalized after successful ablation of PVCs but remained unchanged in non-treated patients and in patients after unsuccessful RFCA. These findings suggest that recent onset, frequent PVCs can result in reversible biventricular dysfunction not detected by conventional echocardiographic parameters.

Effect of frequent PVCs on ventricular performance

Frequent PVCs are generally considered a benign condition when found in patients without structural heart disease. Small series have demonstrated a link between frequent PVCs and increased LV dimensions and impaired LVEF which improved or normalized after abolishing PVCs by pharmacological treatment or RFCA. However, the majority of patients presenting with frequent PVCs has a preserved LVEF.[1-5] This does not exclude a negative effect of PVCs on systolic function as subtle or early impairment may not be detected by conventional parameters.

Niwano prospectively followed 239 patients with frequent PVCs and only 13, the majority with a high PVC burden, developed a small but significant decline in LVEF after 4 years, suggesting that impaired LV function may develop only over long periods.[5] However, 42 patients with significant symptoms and/or LV dysfunction at baseline were excluded. These patients might represent a subgroup of patients in whom LV dysfunction occurs earlier.[5]

In the present study the median duration of symptoms was 13 months. Despite the short period, these patients had already decreased multidirectional strain as compared to controls, whereas LV volumes, RV dimensions and LV and RV function measured by conventional echocardiographic techniques were normal. This indicates that PVCs may induce early LV and RV dysfunction not detected by conventional 2D echocardiography measurements. The evaluation of multidirectional strain with 2D speckle tracking imaging enables the detection of functional abnormalities by exploring the mechanical properties of the myocardium.[10] 2D speckle tracking imaging may constitute a more sensitive tool to detect subtle ventricular dysfunction, in particular for RV function assessment since quantification of RV volumes

Table 3. Follow-up echocardiography study and 2-dimensional speckle tracking strain analysis

	PVC-RFCA (n = 34)			Non-RFCA PVC (n = 15)		
	Baseline	Follow-up	p-value	Baseline	Follow-up	p-value
Heart rate (bpm)	69 ± 11	68 ± 11	0.790	64 ± 8	68 ± 11	0.670
LV end-diastolic volume (ml)	127 ± 39	120 ± 34	0.077	116 ± 22	114 ± 23	0.551
LV end-systolic volume (ml)	56 ± 21	49 ± 15	0.018	52 ± 14	49 ± 12	0.073
LV ejection fraction (%)	57 ± 8	59 ± 5	0.245	55 ± 5	57 ± 8	0.244
RV outflow tract diameter (mm)	29 ± 4	30 ± 4	0.362	30 ± 4	31 ± 2	0.207
RV basal diameter (mm)	30 ± 4	29 ± 3	0.535	32 ± 4	31 ± 3	0.153
RV mid-level diameter (mm)	29 ± 6	28 ± 5	0.186	28 ± 4	28 ± 5	0.689
RV longitudinal diameter (mm)	73 ± 10	74 ± 10	0.632	68 ± 11	72 ± 11	0.091
RV fractional area change (%)	43 ± 9	45 ± 10	0.261	41 ± 5	40 ± 8	0.783
TAPSE (mm)	2.5 ± 0.5	2.4 ± 0.4	0.327	2.4 ± 0.3	2.2 ± 0.3	0.073
LV Radial strain (%)	31.1 ± 14.2	45.5 ± 16.3*	<0.0001	28.5 ± 9.7	32.0 ± 9.6	0.272
LV Circumferential strain (%)	-16.2 ± 3.9	-18.9 ± 4.2†	0.004	-15.2 ± 4.2	-15.4 ± 3.2	0.925
LV Longitudinal strain (%)	-17.8 ± 2.9	-19.6 ± 2.0*	0.007	-16.4 ± 3.2	-17.1 ± 2.5	0.638
RV Longitudinal strain (%)	-24.2 ± 7.4	-28.4 ± 6.0*	0.009	-22.8 ± 4.2	-23.2 ± 5.2	0.999

*p<0.01 vs. non-RFCA at follow-up; †p = 0.014 vs. non-RFCA at follow-up.
LV = left ventricular; PVC = premature ventricular complex; RFCA = radiofrequency catheter ablation; RV = right ventricular; TAPSE = tricuspid annular plane systolic excursion.

and function with conventional echocardiography remains problematic due to the complex geometry of the RV. However, the present results do not allow to define robust baseline cut-off values of multidirectional myocardial strain to identify and predict which patients will develop cardiomyopathy if RFCA is not applied. Additional studies including larger populations and longer follow-up are warranted to elucidate this aspect.

An inverse relationship between PVC burden and cardiac function has been described and PVC frequency has also been suggested as risk factor for PVC induced cardiomyopathy.[4, 6, 7]

In the current study, patients with a PVC burden of >20% were compared to patients with a PVC burden <20%. Although we did not observe significant differences in LVEF and volumes, there was a tendency to larger LV volumes in patients with a higher PVC burden, still within normal range. LV and RV strain were decreased to a similar extend in patients with a high and moderate PVC burden indicating that even a moderate burden of PVCs (mean 13.3%) can induce subclinical cardiac dysfunction. However, the proportion of patients with a moderate PVC burden was relatively small.

Previous studies reporting on a reversal of cardiomyopathy after RFCA only included patients with PVCs from the RV outflow tract.[3, 7] More recently it was demonstrated that a reversible reduced EF was equally prevalent among patients who had PVCs with a right or left bundle branch block configuration.[4, 18] This was confirmed by our study demonstrating that subtle and reversible LV and RV dysfunction can be induced by PVCs regardless of the site of origin.

RFCA and improvement of LV and RV strain

This study extends the finding of prior reports, that RFCA for PVCs can reverse cardiac functional abnormalities induced by PVCs.[3, 4, 6, 7] While Yarlagadda[3] and Bogun[4] observed reversal of mild to severe dilated cardiomyopathy after ablation of PVCs, Takemoto[7] and Sekiguchi[6] studied patients with normal LVEF detecting no functional abnormalities before RFCA, and demonstrating improvement of LV EF and diameters within the normal range during follow-up. The present study, however, shows that PVC induced LV and RV dysfunction detected at baseline by strain analysis in patients with normal ventricular dimensions and LVEF can be reversed after successful RFCA. In contrast, in the PVC RFCA -control group decreased strain at baseline did not change during short term follow-up. Additional studies with longer follow-up will be warranted to elucidate whether ventricular strain further impairs in patients with frequent PVC non-treated with RFCA.

Clinical implications

Two-dimensional speckle tracking strain imaging detects cardiac functional abnormalities in symptomatic patients with PVCs not detected by conventional echocardiographic measurements. This relatively novel imaging technique has been validated against sonomicrometry and tagged magnetic resonance imaging and several studies have demonstrated its accuracy to measure myocardial strain.[13, 14] By measuring the mechanical properties of the myocardium with this technique, subtle myocardial dysfunction can be detected. Indeed, despite preserved LV or RV functions as assessed with conventional echocardiography, 2D speckle tracking can demonstrate subtle myocardial dysfunction in different clinical conditions (i.e. aortic stenosis, diabetic cardiomyopathy, coronary artery disease).[16, 19, 20] In addition, subtle myocardial dysfunction as detected with 2D speckle tracking has important

prognostic implications.[21-23] As demonstrated in previous studies, LV longitudinal strain showed superior prognostic value than baseline clinical characteristics, LV ejection fraction or wall motion score index measured with conventional echocardiography.[23] Therefore, 2D speckle tracking may constitute a valuable imaging tool to improve the risk stratification of patients with different clinical conditions, including those that potentially can cause cardiomyopathy at long-term follow-up if not treated.

RFCA for PVCs is associated with a high success rate and few complications.[3, 4, 6, 7, 24] Beside its value in relieving patients symptoms, RFCA can play an important role in the management of patients with PVC induced ventricular dysfunction.

Since it has been suggested that advanced PVC induced ventricular dysfunction may lead to irreversible cardiomyopathy in individual patients, the early detection of PVC induced dysfunction might be of clinical relevance.[25] Although there seems to be an association between PVC frequency, duration and decline in LV function some patients with low PVC burden and/or short duration of symptoms have already deterioration of LV function. Therefore PVC burden and duration alone may not identify patients at risk.[2, 4, 5] Until additional risk factors are identified, 2D speckle tracking strain imaging may constitute a valuable test to identify patients that need to be re-assessed during long term follow-up. Whether

Conclusions

Frequent PVCs can induce subtle LV and RV dysfunction in patients without apparent cardiomyopathy. PVCs originating from the LV and RV have similar detrimental effects on ventricular function. RFCA can successfully eliminate PVCs and improve cardiac function. These changes can be evaluated by using 2D speckle tracking imaging.

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