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Abstract

AIMS: Patients with isolated left ventricular non-compaction (IVNC) are at high risk for developing ventricular tachyarrhythmias. However, no analysis of invasive electrophysiological (EP) findings in these patients has yet been performed. METHODS AND RESULTS: We performed a retrospective analysis of EP findings in 24 patients with IVNC. Ventricular tachyarrhythmias were inducible in nine patients; of these, two patients had sustained monomorphic ventricular tachycardia (VT) and two patients had ventricular fibrillation. No specific electrocardiographic or echocardiographic finding was predictive of VT inducibility. Three of the 9 patients with inducible VT experienced ventricular tachyarrhythmias during the follow-up of 61.4+/-50 months, whereas no tachyarrhythmias or sudden deaths were noted in 12 patients without inducible VT during the follow-up of 30+/-19 months (3 patients in the latter group were lost to follow-up). Supraventricular tachyarrhythmias were inducible in seven patients. CONCLUSION: Our present study provides the first comprehensive analysis of EP findings in patients with IVNC. Ventricular and supraventricular arrhythmias can readily be induced in these patients, whereas the inducibility of a sustained monomorphic VT is relatively low. Further studies including long-term follow-up are required to investigate the role of EP testing for arrhythmic risk stratification in these patients.

Electrophysiologic Findings in Patients with Isolated Left Ventricular Noncompaction

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Abstract

Background – Patients with isolated left ventricular noncompaction (IVNC) are at high risk for developing ventricular tachyarrhythmias. However, no analysis of invasive electrophysiologic (EP) findings in these patients has yet been performed.

Methods and Results – We performed a retrospective analysis of EP findings in 24 patients with IVNC. Ventricular tachyarrhythmias were inducible in 9 patients; of these, 2 patients had sustained monomorphic ventricular tachycardia (VT) and 2 patients had ventricular fibrillation. No specific electrocardiographic or echocardiographic finding was predictive of VT inducibility. Three of the 9 patients with inducible VT experienced ventricular tachyarrhythmias during the follow-up of 61.4 ± 50 months, while no tachyarrhythmias or sudden deaths were noted in 12 patients without inducible VT during the follow-up of 30 ± 19 months (3 patients in the latter group were lost to follow-up). Supraventricular tachyarrhythmias were inducible in 7 patients.

Conclusions – Our present study provides the first comprehensive analysis of EP findings in patients with IVNC. Ventricular and supraventricular arrhythmias can readily be induced in these patients, while the inducibility of a sustained monomorphic VT is relatively low. Further studies including long-term follow-up are required to investigate the role of EP testing for arrhythmic risk stratification in these patients.

Condensed Abstract

We performed the first comprehensive analysis of electrophysiologic findings in 24 patients with isolated left ventricular noncompaction. Importantly, we show that ventricular and supraventricular arrhythmias can readily be induced during EP testing, while the inducibility of a sustained monomorphic ventricular tachycardia is relatively low.

Keywords: Noncompaction, Cardiomyopathy, Electrophysiology, Ventricular Tachycardia, Implantable Cardioverter Defibrillator

Introduction

Isolated left ventricular noncompaction (IVNC) is a primary genetic cardiomyopathy,[1] which is morphologically characterized by a two-layered structure of the myocardium consisting of a compacted, thin epicardial layer and a non-compacted, severely thickened endocardial layer, which by definition occur in the absence of other coexisting congenital lesions.[2, 3] The clinical spectrum of presentation of these patients is highly variable, ranging from asymptomatic, coincidental discovery of the disease to severe heart failure.[3, 4]

IVNC is a rare disorder: In a large series of patients referred to a tertiary care echocardiography laboratory, the prevalence of IVNC was 0.014%;[5] in a single center heart failure clinic, IVNC was the underlying cause of heart failure in 2.7% and heart transplantation in 2% of patients.[6] However, both benign as well as life-threatening arrhythmias such as ventricular tachycardia (VT) and ventricular fibrillation (VF) have been reported in patients with IVNC.[4, 7] For the prevention of cardiac sudden death, implantation of an implantable cardioverter-defibrillator (ICD) may be considered; indeed, we recently reported on 12 patients with IVNC who received an ICD either for primary or secondary prevention.[8] However, as IVNC is a rare disorder, it is unclear at present under which circumstances such arrhythmias are most likely to occur. Invasive electrophysiologic (EP) studies are frequently used to assess the propensity for developing malignant ventricular tachyarrhythmias. To date, however, EP findings in patients with IVNC have not been comprehensively analyzed. The purpose of the present retrospective analysis, therefore, was to characterize the electrophysiological properties in our relatively large cohort of patients with IVNC.

Methods

Study population

Between January 1998 and November 2008, an EP study was performed in 20 patients with IVNC at the University Hospital Zurich, and 4 patients at the Cantonal Hospital Lucerne. Seven of these cases, who subsequently underwent ICD implantation, were previously reported.[8]

Echocardiographic criteria for the diagnosis of IVNC were the same as previously published and remained unchanged during the entire study period:[3-5] 1) The absence of coexisting cardiac anomalies (other than 2–4); 2) Two-layered structure of the myocardium with a thick, non-compacted endocardial layer consisting of a trabecular meshwork with deep endocardial spaces and

a much thinner, compacted epicardial layer, and a maximum end-systolic ratio of the non-compacted endocardial layer to the compacted myocardium of ≥2, measured at end-systole; 3) Predominant segmental location of the abnormality; and 4) Colour Doppler-echocardiographic evidence that deep intertrabecular recesses are perfused with blood from the left ventricular cavity. Of note, the diagnosis of "right ventricular non-compaction" is not attempted anymore at our institutions, as a differentiation between normal variants of the usually highly trabeculated right ventricle and pathological forms may be difficult if not impossible.[3, 5, 9] Furthermore, patients not fulfilling criteria of *isolated* non-compaction cardiomyopathy (i.e., IVNC in the presence of other congenital heart diseases) were also excluded from the analysis. All patients underwent a complete echocardiographic examination at first presentation; mean time between echocardiography and EP study was 2.1 ± 2.9 months. ECGs at the time of the EP study were independently analyzed by two readers (JS and FD).

Electrophysiologic study

EP studies were performed according to a standard protocol using 6F diagnostic electrophysiology catheters (Bard Inc., Lowell, MA) on a Bard EP lab with a Micropace EPS 320 Cardiac Stimulator System (Micropace, Tustin CA). All antiarrhythmic drugs were discontinued 5 half-lives before the electrophysiologic study. Programmed ventricular stimulation protocol included 3 drive-cycle lengths and 3 ventricular extrastimuli while pacing from 2 right ventricular sites (apex and outflow tract). Ventricular stimulation was performed until refractoriness or until a minimum coupling interval of 180 ms. In patients in whom VT was not inducible at baseline, isoproterenol (Hospira Enterprises, Hoofddorp, The Netherlands) was administrated intravenously (up to 4 μg/min), followed by application of up to three extrastimuli as well as burst pacing (until a minimum of 250ms) if deemed clinically indicated by the operator.

Sustained VT was defined as tachycardia of ventricular origin lasting longer than 30 seconds or resulting in hemodynamic compromise; non-sustained VT was defined as a tachycardia of ventricular origin of more than 3 beats but less than 30 seconds, and not resulting in hemodynamic compromise. Programmed stimulation in the atrium was performed using an electrode positioned in the high right atrium. Sinus node recovery time (SNRT) was measured after overdrive pacing in the high right atrium for 30 seconds; corrected SNRT was calculated by subtracting the individual patient's basic cycle length (BCL) from the measured SNRT.

Statistics

Comparison of categorical variables was performed by Fisher's exact test. Continuous variables were analyzed by two-sided Student's t-test (for normally distributed variables) or Mann-Whitney U test (for non-normally distributed variables) and are presented as mean ± standard deviation. A p-value < 0.05 was considered significant. Statistical analysis was performed using GraphPad Prism 4 for Windows (GraphPad Software Inc, La Jolla, CA).

Results

Baseline parameters and indication for EP study

Baseline characteristics and indications for the invasive EP study are summarized in table 1. Most frequently, EP testing was performed for the evaluation of syncope, documented VT, symptomatic ventricular extrasystoles (VES), or for arrhythmic risk stratification. Since no guidelines or recommendations with respect to EP testing in IVNC exist, the decision to perform an EP study was made on an individual basis and according to the treating physician's judgment.

An entirely normal EP study was found in 11 patients (46%). While these patients were significantly younger (25 ± 9.8 vs. 46.3 ± 12.8 years, $p<0.001$), no other clinical, electrocardiographic or echocardiographic parameters were predictive of a normal EP study.

Rhythm at EP study and atrioventricular conduction

The underlying rhythm at the time of the EP study as well as atrioventricular conduction properties are summarized in table 2. Most patients presented with sinus rhythm; 2 patients had ventricular preexcitation via an accessory pathway. High degree AV block was present in 2 patients; one of these patients had a pacemaker in place with a slow escape rhythm showing a His potential before each QRS complex (indicating location of the AV block proximal to the bundle of His), while the other had underlying atrial fibrillation with a junctional escape rhythm. One patient had evidence of dual AV nodal physiology with a reproducible jump in AH conduction, but without inducible AV nodal reentry tachycardia (AVNRT). The HV interval was slightly prolonged in one patient (58 ms), while one patient had a rather short AV block cycle length of 225 ms. There was no evidence of sinus node disease in any of the patients examined.

Inducibility of ventricular tachyarrhythmias

Type and origin of VT, ease of inducibility, concomitant ECG, echocardiographic and clinical findings (evidence of coronary artery disease, Holter ECG findings, signs of heart failure, heart failure or antiarrhythmic medication at the time of EP study, if available) are summarized in table 3. Ventricular tachyarrhythmias were inducible in 9 patients (38%). Two patients had sustained monomorphic VT (Fig. 1), 2 patients had sustained polymorphic VT or VF, whereas the remaining 5 patients had non-sustained polymorphic VT. One of the latter patients (patient 6, Tab. 3) had a monomorphic sustained VT originating from the right ventricular outflow tract (RVOT) at initial presentation. No specific electrocardiographic or echocardiographic finding was predictive of VT inducibility during EP testing. The prevalence of significant coronary artery disease was low, both in patients with (Tab. 3) and without (Tab. 4) inducible VT.

Since no guidelines or recommendations regarding ICD implantation in patients with IVNC exist, this decision was left to the discretion of the treating physician. In our cohort, 7 of the 9 patients who had inducible VT or VF underwent ICD implantation following the EP study. In contrast, non-sustained VT in the EP study of 2 patients was judged to be nonspecific, and therefore these patients did not have an ICD implanted. 3 of the 9 patients with an inducible VT showed evidence of ventricular tachyarrhythmias during the mean follow-up period of 61 ± 50 months, all of which were adequately treated with either anti-tachycardia pacing or shock delivery.

Clinical parameters, resting ECG and echocardiographic findings of the 15 patients (63%) without inducible ventricular arrhythmias are summarized in table 4. Three of the 15 patients without inducible ventricular tachyarrhythmias were lost to follow-up; mean follow-up of the remaining 12 patients was 30 ± 19 months. During this period, no symptomatic tachyarrhythmias were reported.

Inducibility of supraventricular tachyarrhythmias and therapeutic interventions

Supraventricular tachyarrhythmias were inducible in 7 patients (Tab. 2). In 2 patients, an orthodromic AV reentry tachycardia (AVRT) was inducible; one of these patients (in whom AVRT was easily induced) subsequently underwent successful ablation of a left anterolateral accessory pathway, while an intervention was not performed in the other, asymptomatic patient (in whom AVRT was only inducible under isoproterenol infusion). One further patient had evidence of a right anterior accessory pathway (without inducible AVRT during the EP study), which was subsequently ablated. AVNRT was induced in one patient under isoproterenol infusion, who subsequently underwent successful slow pathway modification. In one patient who presented with typical atrial flutter, the tricuspid isthmus was ablated successfully. Of the two patients with

inducible atrial fibrillation, one was induced by singular atrial stimulation while the other occurred under isoproterenol infusion.

Discussion

This is the first study to report the electrophysiological characteristics in a large cohort of patients with IVNC. This rare disease is known to be associated with life threatening ventricular tachyarrhythmias possibly due to the noncompacted myocardium serving as the arrhythmic substrate. Furthermore, impaired flow reserve in structurally compacted myocardial segments with resultant (intermittent) ischemia may play an important role.[10] Indeed, previous studies have reported ventricular arrhythmias in 47%, and sudden cardiac death in as many as 18% of (mostly) adult patients with IVNC.[5, 8, 9] In our cohort, however, a sustained monomorphic VT was only rarely induced, and only two patients had inducible polymorphic VT/VF. Moreover, infusion of isoproterenol did not facilitate the induction of sustained monomorphic tachycardia. Non-sustained polymorphic ventricular tachycardia was observed more commonly, which in general is believed to be a nonspecific finding, especially under isoproterenol infusion and/or application of three extrastimuli or burst pacing. However, we deliberately chose this protocol in view of the high propensity of patients with IVNC to develop malignant ventricular arrhythmias and sudden death. Indeed, two such patients (patient 4 and 5, Tab. 4) subsequently demonstrated ventricular arrhythmias on follow-up, which were adequately treated by their ICDs. No specific clinical, electrocardiographic or echocardiographic finding was predictive of VT inducibility or of a normal EP study (except for the younger age of patients in the latter group). It is of note, however, that none of the patients with an EF more than 50% developed spontaneous VT during follow-up.

One patient (patient 1, Tab. 3) with an inducible VT demonstrated a severely reduced systolic left ventricular function, which in itself is known to increase the propensity for developing ventricular tachyarrhythmias. Based on current evidence, patients with a severely reduced left ventricular EF have an ICD implanted empirically and do not undergo EP testing for risk stratification. However, several of our patients (including patient 1, Tab. 3) underwent EP testing before the advent of landmark studies such as the SCD-HeFT trial. Since IVNC is a rare disorder, it is unclear whether currently available guidelines are also applicable to this patient population. Nevertheless, prophylactic ICD implantation without prior EP study nowadays appears to be reasonable in patients with IVNC who fulfill the SCD-HeFT criteria.

In our study, six patients did not have any clinical signs or predictors of ventricular tachyarrhythmias prior to the EP study, and underwent EP testing for risk stratification in view of the increased risk of ventricular arrhythmias in this patient population. A sustained monomorphic VT could be induced in one of these patients (patient 2, Tab. 4), while the others had normal EP studies. In contrast, one patient had a sustained monomorphic VT originating from the RVOT at presentation, which was not reproducible during EP testing. We cannot exclude the possibility of a non-reentrant mechanism for the induction of the monomorphic tachycardia in this particular patient. Furthermore, it cannot be excluded that RVOT tachycardia was unrelated to the presence of IVNC in this patient.

No ventricular arrhythmias or sudden cardiac deaths were recorded in the clinical follow-up of 12 patients in whom no VT was inducible on EP testing. Two of these patients also had an ICD implanted, which showed no arrhythmic events during follow-up. These data indicate that a negative EP study may identify a subset of patients with IVNC at low risk of developing malignant tachyarrhythmias. However, follow-up duration in this subgroup was considerably shorter (i.e., 30 months) as compared to that of patients with inducible VT (i.e., 61 months). Indeed, a VT occurred in one patient from the latter group 8 years after EP testing, indicating that malignant arrhythmic events may occur outside the 30 months follow-up period for patients without inducible VT. Furthermore, this observation raises the possibility that progression of the disease (and hence change of the arrhythmogenic substrate) may be an important factor contributing to the propensity of developing malignant VT, especially in the long term. Hence, further studies are warranted to determine the role of EP testing in IVNC, especially with respect to the prospective value of a negative EP study in these patients.

Intraventricular conduction delay as well as first-degree AV block are common in patients with IVNC.[3-5] Interstitial fibrosis and subendocardial fibroelastosis, which are frequently found on endomyocardial biopsies in these patients, may be the underlying pathoanatomic correlate.[11] In addition, sinus bradycardia was frequently observed in children with IVNC.[12] In our patient cohort, abnormal AV conduction was discovered in several patients. Interestingly, in both patients with complete heart block, the conduction block was at the supra-His level.

Wolf-Parkinson-White (WPW) syndrome is equally a common finding in the pediatric population with IVNC and present in up to 17% of these children,[13] while it is rarely found in adults.[3, 4] Accessory AV pathways are most likely the result of persisting AV muscular continuity having failed to regress during embryogenesis, which appears pathophysiologically similar to the failure of regression of the non-compacted myocardium in IVNC.[11] However, the prevalence of WPW in the general population is approximately 0.15-0.2%, and the coexistence of WPW and

IVNC may have simply occurred by chance. In our cohort, a total of 7 patients (29%) with IVNC had at least one supraventricular tachycardia, which included atrial fibrillation and atrial tachycardia. It remains to be determined if this developmental disease of the myocardium also plays an important role in atrial arrhythmogenesis.

Limitations: The goal of our present study was to comprehensively analyze invasive EP findings in patients with IVNC. Although our cohort of patients with this rare disorder undergoing EP testing is the largest reported so far, the study is limited by several factors. The unequal follow-up duration of patients without (30 months) and with inducible VT (61 months) makes a recurrent VT more likely in the latter group simply due to the longer length of follow up. Further limitations include a low absolute number of cases as well as a possible selection bias, since an EP study was only performed in patients in whom it was deemed clinically indicated by the treating physician. In addition, three patients without inducible VT were lost to follow-up.

Conclusion

Ventricular as well as supraventricular arrhythmias can readily be induced during EP testing in patients with IVNC, while the inducibility of a sustained monomorphic VT is relatively low. Our data further indicate that in selected high-risk patients, EP testing may be of limited value, and ICD implantation may be considered in patients with IVNC clinically judged to be at high risk for ventricular tachyarrhythmias. These include patients with a prior history of sustained VT or VF, or patients fulfilling well-established primary prevention criteria for ICD implantation. Additional studies including (prospective) long-term follow-up are required to investigate the role of EP testing for arrhythmic risk stratification, especially in patients without inducible VT.

Conflict of Interest Disclosure

None of the authors report a conflict of interest.

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Legends to Figures

Figure 1:

12-lead ECG demonstrating a sustained monomorphic tachycardia with right bundle branch block morphology and superior axis, indicating a left infero-apical origin. The tachycardia was induced by programmed electrical stimulation at the right ventricular apex with two extra stimuli. Overdrive pacing (see bottom V1 rhythm trace) terminated the tachycardia.

Figure 1:

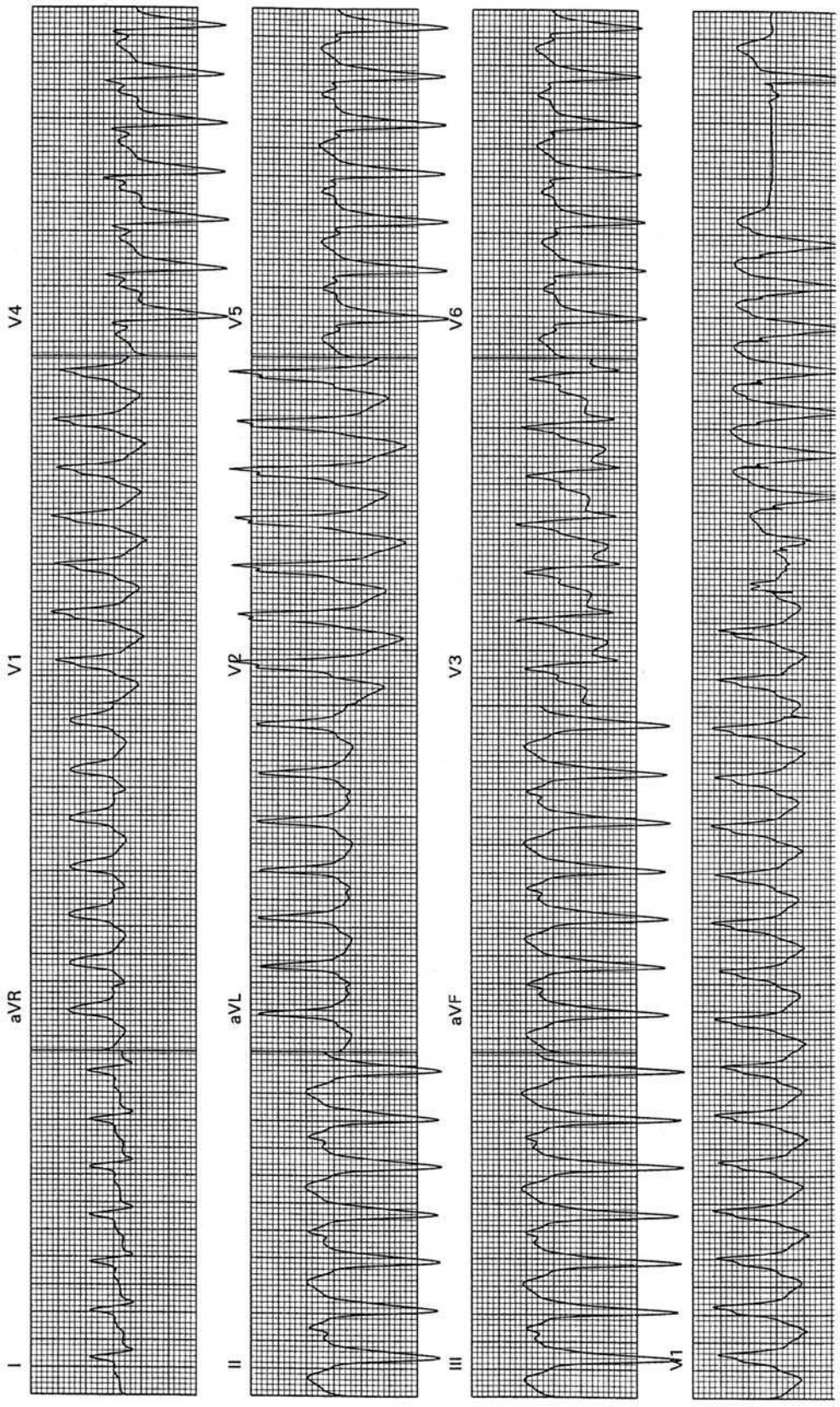

Table 1:

Baseline characteristics and indications for electrophysiologic testing

Characteristic	
Men - no. (%)	18 (75)
Age at diagnosis (yrs) - Mean (SD, range)	38.2 (±15, 16 - 63)
Indication for EPS - no. (%)	
Risk stratification	6 (25)
Syncope / presyncope	5 (21)
Symptomatic VES	5 (21)
Documented non-sustained VT	2 (8)
Documented sustained VT	2 (8)
Suspected WPW	3 (13)
Symptomatic atrial flutter	1 (4)

Table 2:

Spontaneous rhythm at EP study, inducibility of supraventricular tachyarrhythmias and therapeutic interventions

Characteristic	
Spontaneous rhythm at time of EPS - no. (%)	
Sinus rhythm	22 (92)
with preexcitation	2 (8)
3rd degree AV block, atrial fibrillation, junctional escape rhythm	1 (4)
3rd degree AV block, VAD paced, junctional escape rhythm	1 (4)
AV conduction - no. (%)	
Normal AV node physiology	19 (79)
3rd degree AV block	2 (8)
Dual AV node physiology	1 (4)
Prolonged AV conduction	1 (4)
Supernormal AV conduction	1 (4)
Supraventricular tachycardia inducible	7 (29)
AVRT (orthodromic)	2 (8)
Atrial fibrillation	2 (8)
Atrial flutter	1 (4)
AVNRT	1 (4)
Non-specific atrial tachycardia	1 (4)
Interventions	
Accessory pathway ablation (WPW)	2 (8%)
Isthmus ablation (atrial flutter)	1 (4%)
Slow-pathway modification (AVNRT)	1 (4%)

Table 3: Overview of patients with inducible ventricular arrhythmias

ACE-I = Angiotensin converting enzyme inhibitor; AF = Atrial fibrillation; ARB = Angiotensin receptor blocker. AVB = Atrioventricular block; CAD = Coronary artery disease; ES = extrastimuli; G = gender; H/o = History of; inf = inferior; lat = lateral; NYHA = New York Heart Association Class; LBBB = left bundle branch block; LVEDD = Left ventricular enddiastolic diameter; LVEF = Left ventricular ejection fraction; LVH = left ventricular hypertrophy; Mo = Months; NC = noncompacted; nsVT = Non-sustained VT; post = posterior; RV = right ventricular; VES = ventricular extrasystoles; VF = ventricular fibrillation; VT = ventricular tachycardia. *This patient underwent EP testing over 10 years ago; in the available report, the ease of inducibility is not specified, and original recordings are no longer available.

Patient			**EPS**			**ICD**	**Follow up**		**Resting ECG**	**Echo**		
#	Age / G	H/o syncope, VT, VF	Type	Ease of inducibility	Local.	impl.	Mo.	Arrhythmias	Abnormalities	NC segments	LVEF / LVEDD	Other
1	62 f	Sustained VT on ECG	Sustained, monomorphic VT	2 ES	RV Apex	Yes	102	VF after 3 years and several times afterwards	LVH with pathologic repolarisation	Posterobasal; Midventricular lat., inf./septal;	17% / 4.5cm/m^2	NYHA II (ARB) CAD (70-80% RCA)
2	44 m	no	Sustained, monomorphic VT	3 ES	RV Apex	Yes	14.6	None	LBBB and LVH with secondary repolarisation abnormalities	Basal post.; midventricular lat., post; apex	39% / 3.5cm/m^2	No VT on Holter No CAD Amiodarone, ACE-I, BB
3	63 m	nsVT on Holter ECG (7 beats)	Polymorphic VT, degenerating into VF	2 ES	RVOT	Yes	9.9	None	AF + AVB III°; junctional escape rhythm	Midventricular ant., septal, post.; lateral wall; apex	45% / 3.2cm/m^2	No CAD
4	20 m	no	Polymorphic VT & VF	2 ES	RV Apex	Yes	121	VT after 8 years	QTc 504 ms	Infero-posterior wall; apex	50% / 2.9cm/m^2	No VT on Holter No CAD
5	52 m	Syncope	Non-sustained, polymorphic VT (10s); symptomatic	3 ES	RV Apex	Yes	38.6	VT after 11 months	T-wave inversion V5, V6, I, avL	Midventricular lat., inf.; apex	38% / 2.8cm/m^2	No VT on Holter No CAD
6	49 f	Sustained VT on ECG	Non-sustained polymorphic VT (7 beats)	3 ES (under iso-proterenol)	RV Apex	Yes	29.5	None	QTc 540ms, several polymorphic VES	Isolated apical	50% / 2.9cm/m^2	No CAD

7	31 f	no	Non-sustained polymorphic VT (5s)	Not specified*	RV Apex	Yes	116	None	Non-specific intraventricular conduction delay	Midventricular lat., inf.; apex	49% / 3.5cm/m^2	—
8	32 m	Pre-syncope	Non-sustained polymorphic VT (7s)	3 ES	RV Apex	No	111	None	Normal ECG	Isolated apical	50% / 2.9cm/m^2	No VT on R-test
9	18 m	Syncope	Non-sustained monomorphic VT (5s)	Burst pacing (under iso-proterenol)	RVOT	No	6	None	U-wave	Inferior and inferolateral wall	65% / 2.5cm/m^2	—

Table 4: Overview of patients without inducible ventricular arrhythmias

ACE-I = Angiotensin converting enzyme inhibitor; AF = Atrial fibrillation; ARB = Angiotensin receptor blocker. AVB = Atrioventricular block; BB = Beta blocker; CAD = Coronary artery disease; ES = extrastimuli; G = gender; H/o = History of; inf = inferior; lat = lateral; ltf = lost to follow-up; NYHA = New York Heart Association Class; LBBB = left bundle branch block; LVEDD = Left ventricular enddiastolic diameter; LVEF = Left ventricular ejection fraction; LVH = left ventricular hypertrophy; Mo = Months; NC = noncompacted; nsVT = Non-sustained VT; post = posterior; RV = right ventricular; RVH = RV hypertrophy; VES = ventricular extrasystoles; VF = ventricular fibrillation; VT = ventricular tachycardia.

Patient				**Follow up**		**Resting ECG**	**Echo**		**Other**
#	Age / G	H/o syncope, VT, VF	ICD	Mo.	Arrhythmias / Sudden death	Abnormalities	NC segments	LVEF / LVEDD	
1	44 m	Syncope (AVB III)	No	11.8	No	Normal ECG; AVB III° on rhythm strip	Basal lat.; midventricular ant/sept, lat., post.; apex	59% / 2.64cm/m^2	—
2	47 m	No	No	17.3	No	ST segment depression V5,V6,II,III,avF	Midventricular ant./lat., lat., inf; apex	42% / 2.92cm/m^2	NYAH II (BB, ACE-I). No CAD
3	25 f	No	No	14.3	No	ST segment depression V5,V6, II,III,avF	Isolated apical	57% / 3.09cm/m^2	NYHA I-II; Holter: no VT
4	51 m	No	Yes	33.2	No	PQ 120ms. Normal ECG	Basal lateral; midventricular lateral; apex	44% / 2.49cm/m^2	NYHA II
5	58 f	No	No	28.9	No	Normal ECG	Basal lat., post; midventricular lat., post.; apex	38% / 3.12cm/m^2	NYHA II. No CAD
6	18 m	No	No	11.5	No	QRS 110 ms. Voltage signs of LVH	Midventricular sept., lat., inf.; apex	63% / 2.42cm/m^2	No VT on Holter

7	45 f	No	No	27.8	No	Normal ECG	Midventricular lat.; apex	60% / 2.99cm/m^2	No VT on Holter
8	20 m	Syncope	No	35.1	No	Right axis deviation. LVH. Early repolarisation II,III,avF	Isolated apical	66% / 2.42cm/m^2	No VT on R-test
9	44 m	No	No	ltf	--	T-wave inversion V3,V4,V5,V6	Basal ant./sept.; Midventricular ant./sept.; apex	52% / 2.62cm/m^2	No CAD
10	31 m	No	No	ltf	--	Sinus bradycardia 45 / min. Discrete ST segment elevation II,III,avF	Isolated apical	40% / 3.23cm/m^2	No VT on Holter
11	30 m	Short nsVT on exercise stress test	No	47.8	No	Normal ECG	Midventricular inf/lat., inf.; apex	56% / 2.72cm/m^2	—
12	49 f	No	No	39.1	No	Ventricular bigeminus; non-specific repolarisation abnormalities	Isolated apical	50% / 2.69cm/m^2	No VT on Holter
13	48 m	No	Yes	77	No	AVB III°, junctional escape rhythm (48 / min)	Midventricular lat., inf.	17% / 3.89cm/m^2	NYAH II. No CAD
14	20 m	No	No	ltf	--	RVH. Non-specific repolarisation abnormalities	Midventricular lat., inf., post.; apex	74% / 3.06cm/m^2	—
15	16 m	No	No	15.3	No	QRS 120 ms. LVH. ST segment depression V4,V5,V6,II,avF,I,avL. Preexcitation (WPW)	Isolated apical	56% / 2.5cm/m^2	—