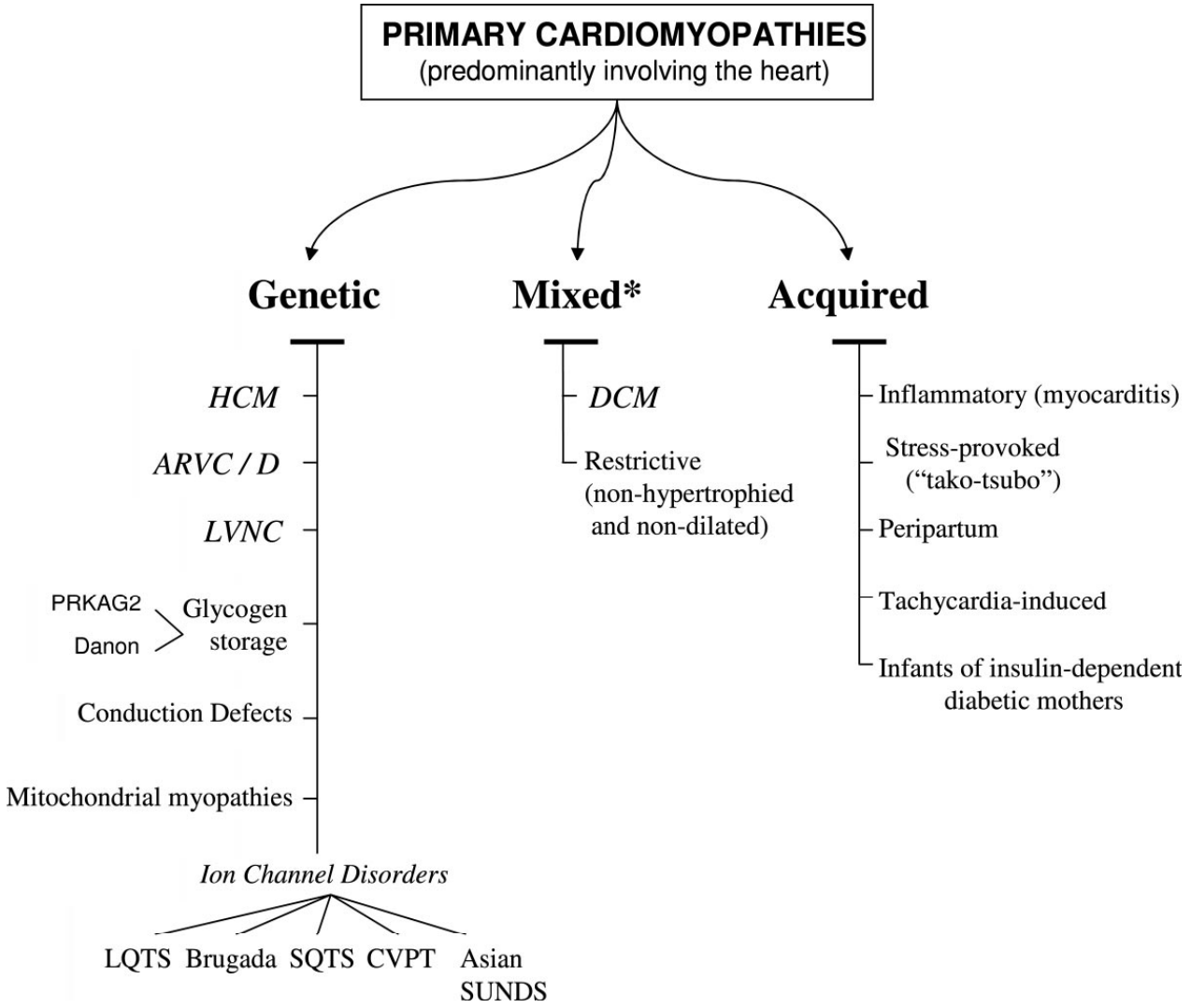
2010 춘계 순환기

심근증에서의 심실빈맥

울산의대

서울 아산병원
남 기병

Cardiomyopathy



Circulation. 2006;113(14):1807-16

Dilated cardiomyopathy

The 5-y mortality for DCM has been recently estimated at 20% with SCD accounting for approximately 30% (8-51%) of deaths.

Mechanism of SCD

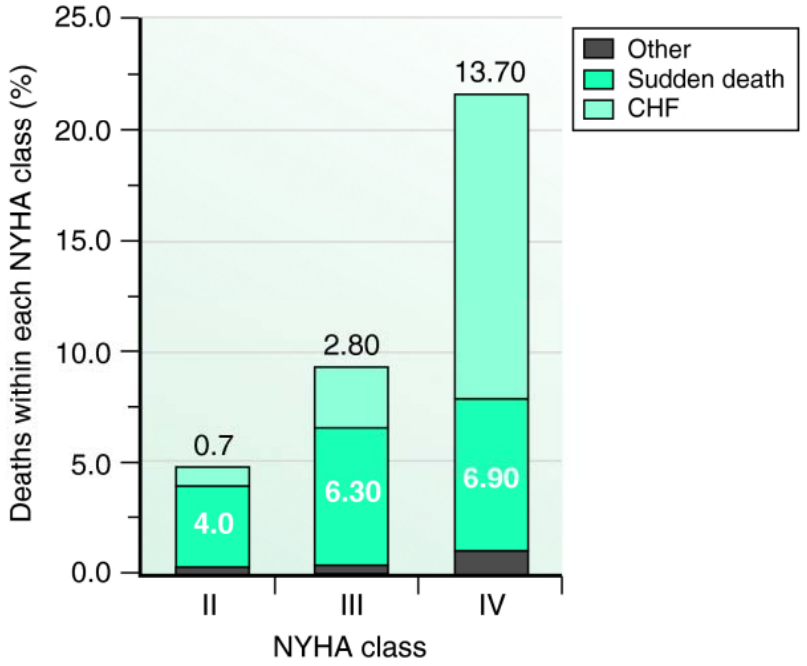
VT and/or VF

bradycardia,

pulmonary embolus,

EMD

* In stage I, II, ventricular tachyarrhythmias could play a major part in SCD. In stage III, IV, ventricular arrhythmias indicate only the degree of ventricular dysfunction, and SCD may follow bradycardia, EMD.



Circulation. 2006;114:e385-e484

Dilated cardiomyopathy

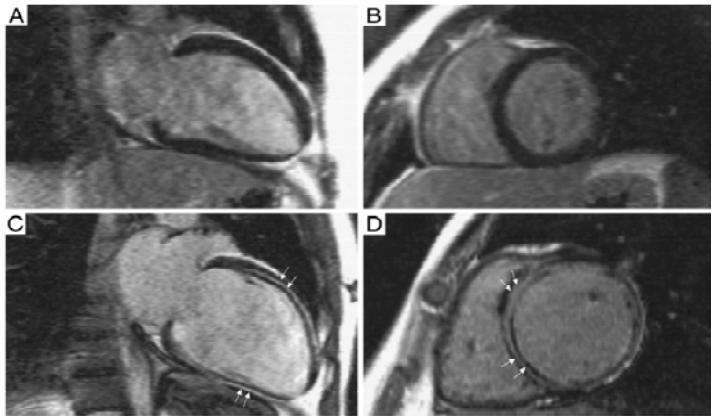
NSVT on HM: 42-60%
EPS, SAECG, EF, wall stress, LV mass, less well correlate

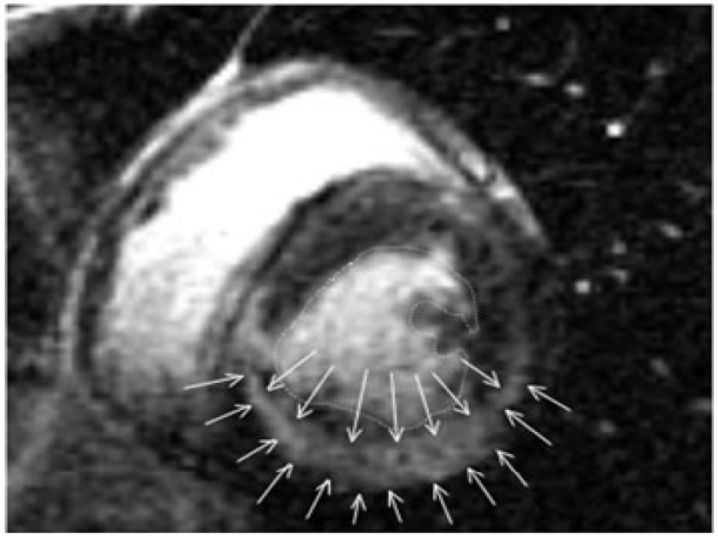
Degree of fibrosis, ass w spontaneous VT
Grossly visible scar in 14% of pts.
Histologically evident fibrosis in 57% of pts.
LV is more affected by fibrosis and myofibrillar destruction.

Fibrosis predominate in perivalvular areas in pts with SM-VT.
Reduced success rate of endocardial catheter ablation,
ID-CM vs. I-CM: epicardial origin of VT

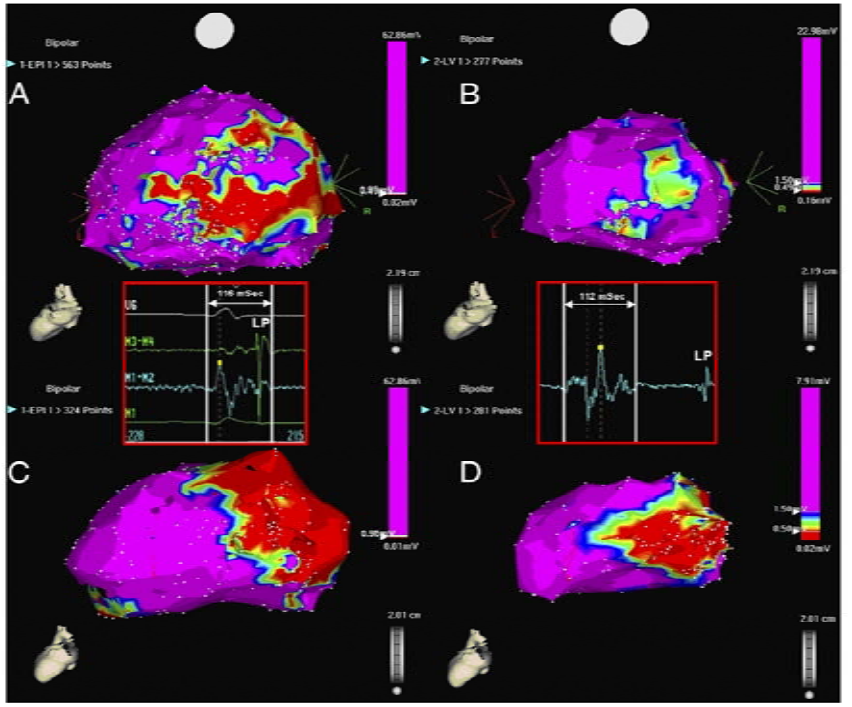
Catheter Ablation of Cardiac Arrhythmias; p352
Wilber D, Packer D, Stevenson W (eds)

Epicardial or midmyocardial scar in D-CM





Wide (>80mm), split, delayed potentials



J Am Coll Cardiol 2009;54:799–808

J Cardiovasc Electrophysiol, Vol. pp. 1-4

J Am Coll Cardiol 2006;48:1977– 85

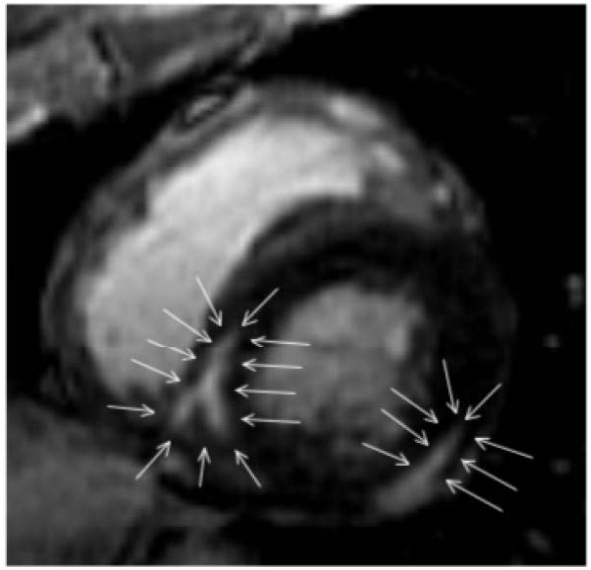
Dilated cardiomyopathy

Delayed-Enhanced MR Imaging in Non-Ischemic Cardiomyopathy
: Utility For Identifying the Ventricular Arrhythmia Substrate

In 14/29 cardiac MRIs, there was evidence of DE in the MRIs.
The distribution of scar: predominantly endocardial (n=7),
midmyocardial (n=5) or epicardial (n=2).

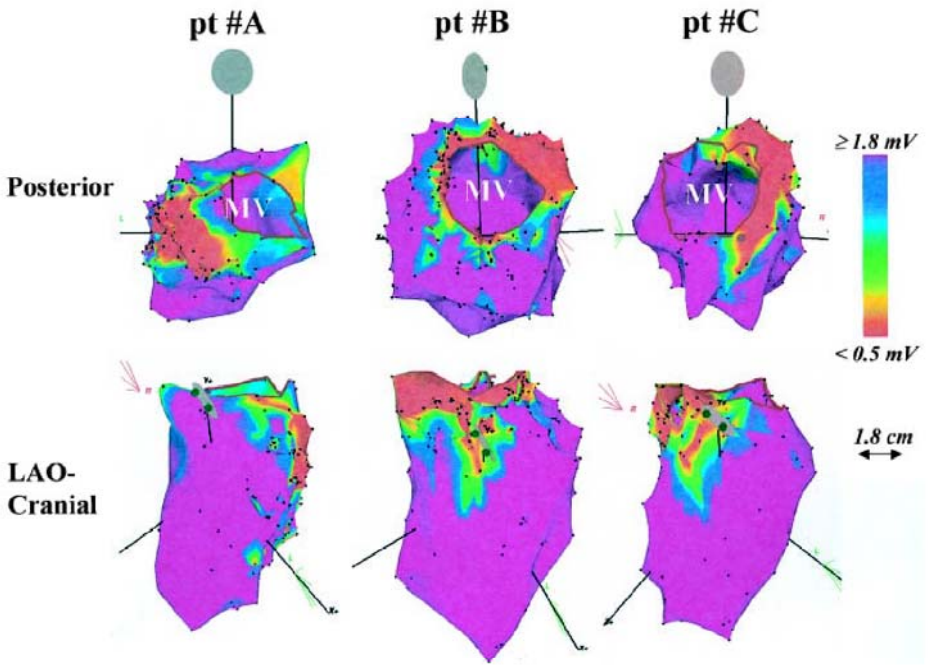
In 2/14 patients with DE in the LV epicardium,
an epicardial mapping procedure eliminated VT.

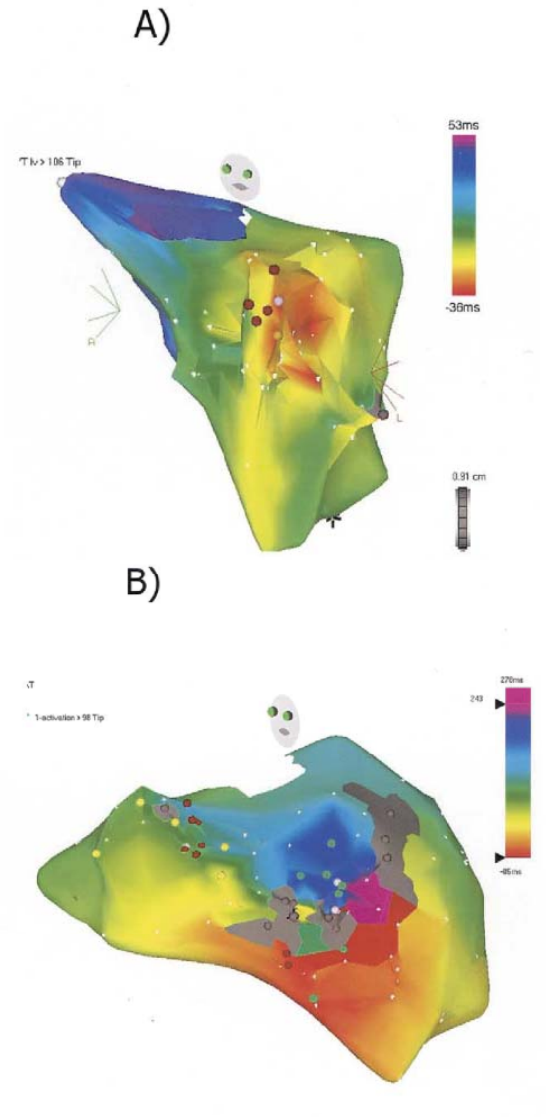
In 5 /14 patients with intramural DE, RF ablation
failed to eliminate the targeted VA in all patients.



F. Bogun, F. Morady JACC 2009;53:1138-45

Scar reentry

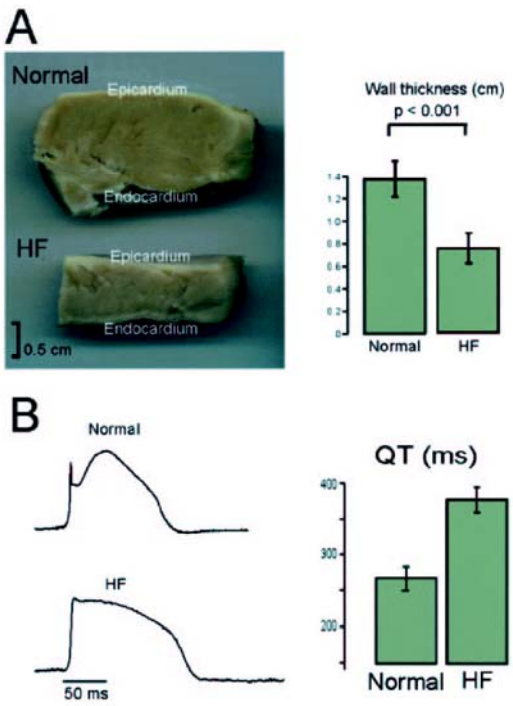


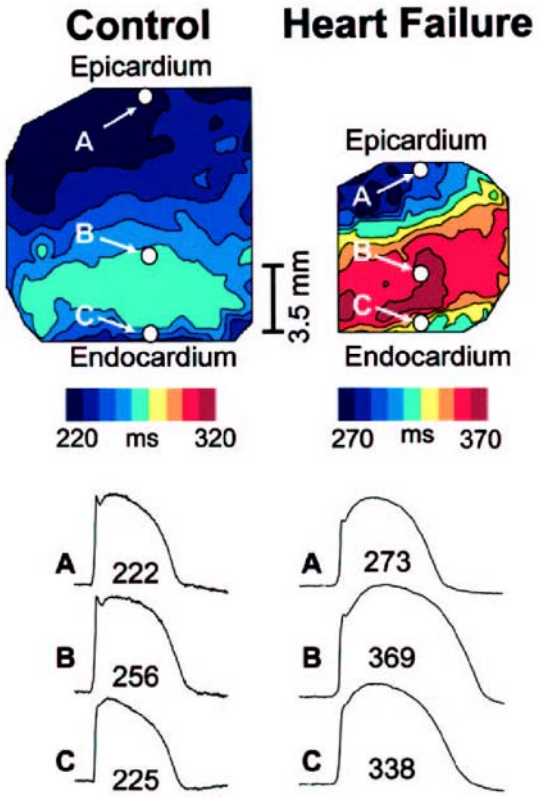


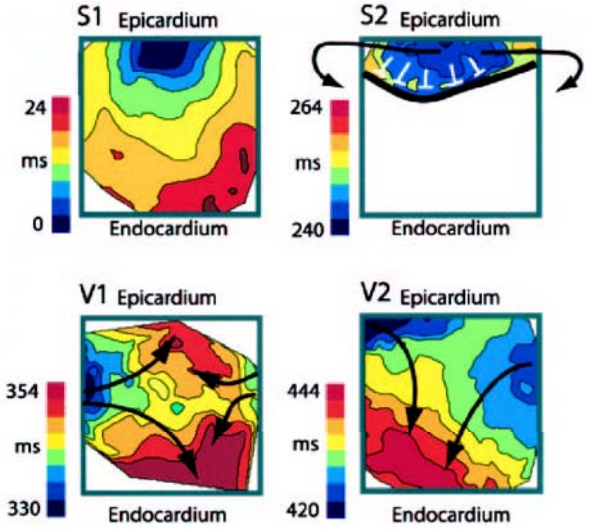
Francis E. Marchlinski *Circulation*. 2003;108:704-710

Kyoko Soejima J Am Coll Cardiol 2004;43:1834–42

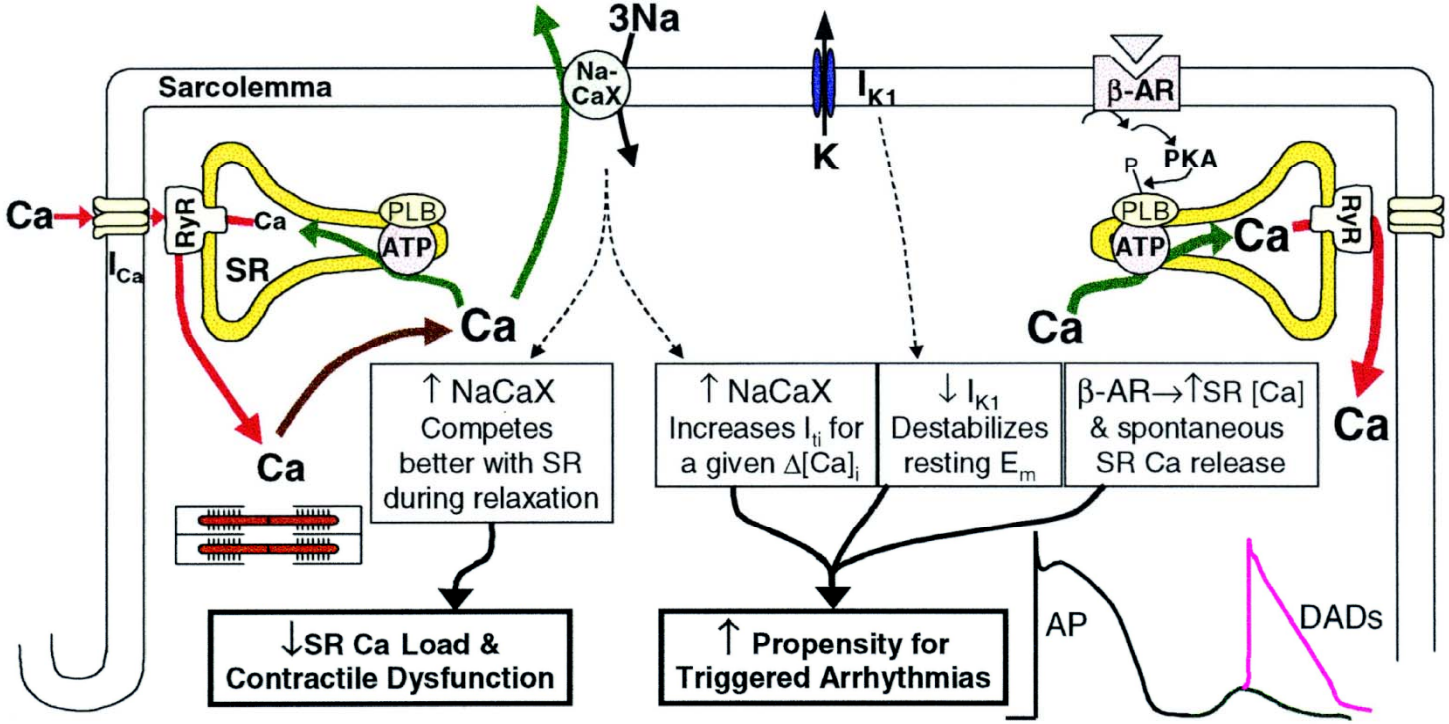
Functional reentry





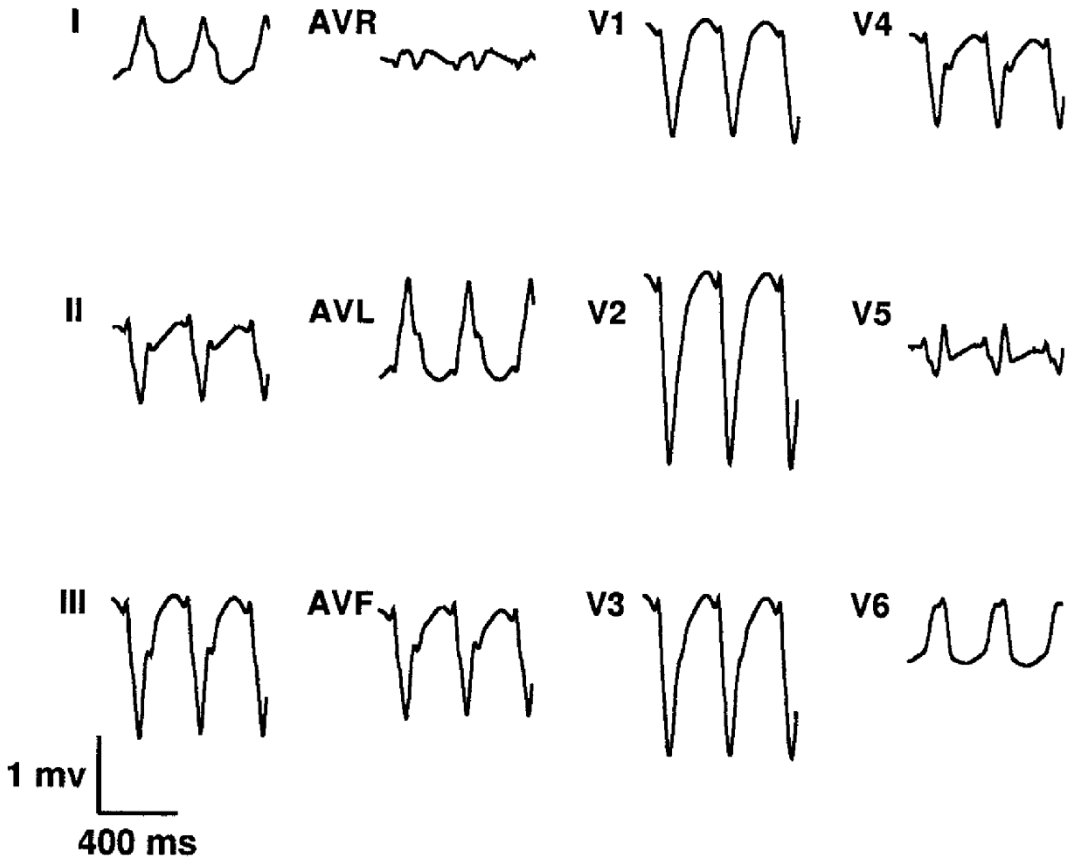


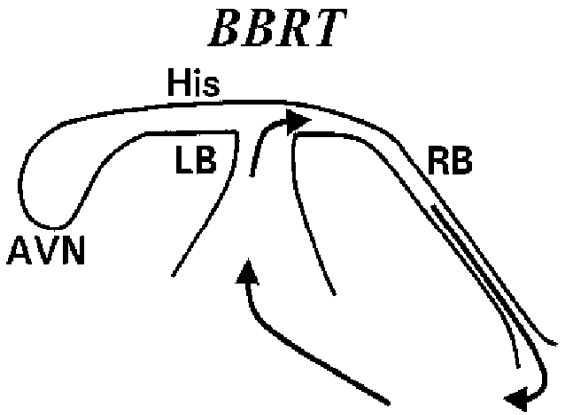
Focal mechanism



Steven M. Pogwizd, Circ Res. 2001;88:1159-1167

Bundle-Branch Reentry VT





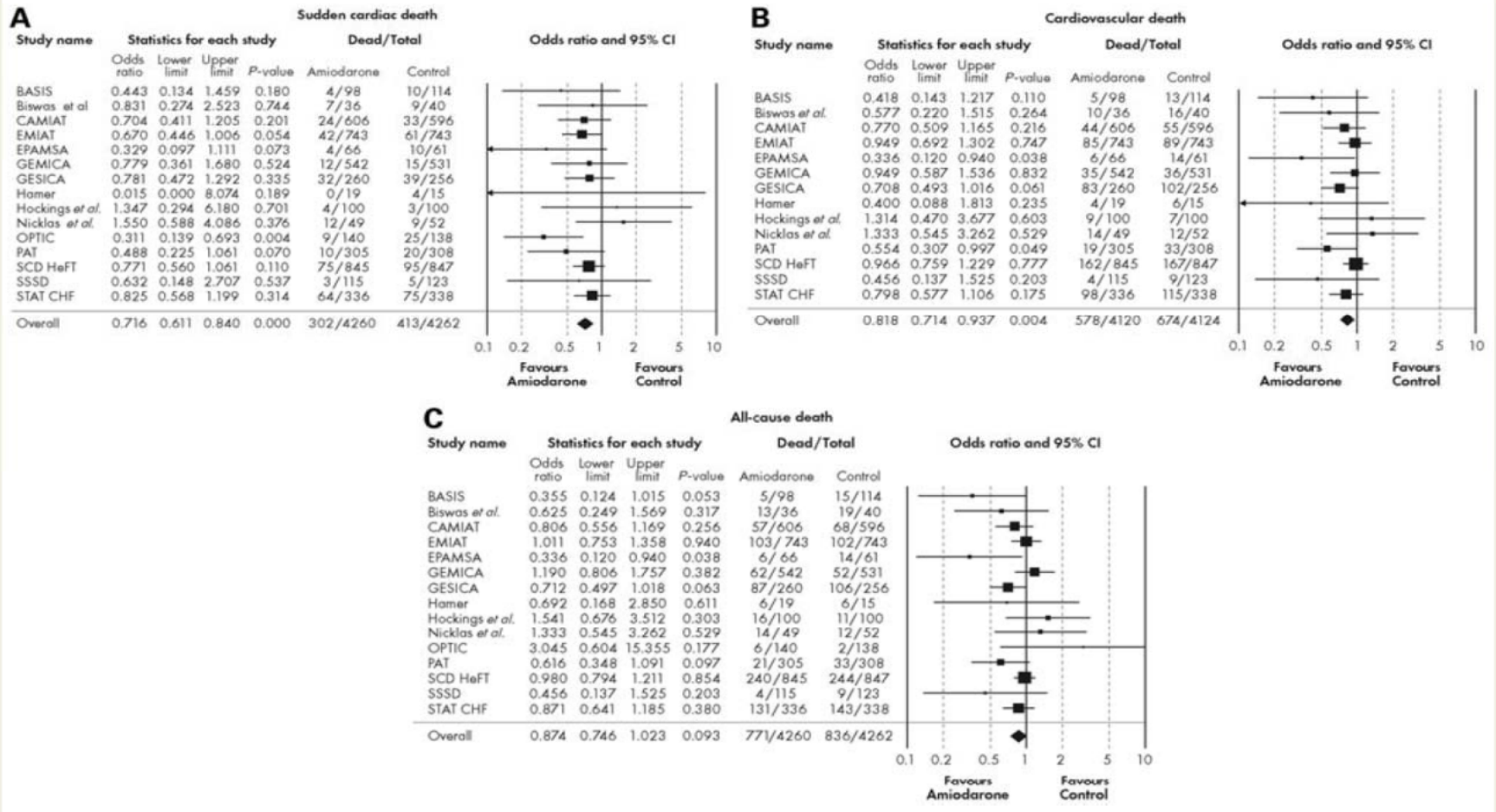
VT in nonischemic CM

A total of 28 patients (regular, EP-mapped)

Focal VT: 5
BBR-VT: 2
Myocardial reentry: 22 (12, endo 7, epi)
Functional reentry?

Kyoko Soejima J Am Coll Cardiol 2004;43:1834–42

Role of Antiarrhythmic Therapy: amiodarone



Adverse reaction	Amiodarone (%)	Control (%)	Random effects odds ratio (95% CI)	*P*-value	Number needed to harm (95% CI)	*P*-value for heterogeneity
Pulmonary toxicity	82/2787 (2.9)	41/2777 (1.5)	1.97 (1.27–3.04)	0.002	69 (45–144)	0.383
Thyroid toxicity	134/3732 (3.6)	15/3724 (0.4)	5.68 (2.94–10.98)	<0.001	32 (26–39)	0.254
Hepatic toxicity	35/1889 (1.85)	16/2276 (0.7)	2.10 (1.15–3.82)	0.015	87 (54–222)	0.767
Brady-arrhythmia	90/3245 (2.8)	45/2938 (1.5)	1.78 (1.16–2.72)	0.008	81 (51–191)	0.368

European Heart Journal (2009) 30, 1245–1253

Hypertrophic Cardiomyopathy : overview

Prevalence: 1/500

In 50-60% of patients, molecular genetic investigation reveals a pathogenic mutation in one of the sarcomeric protein genes.

* Ned Tijdschr Geneeskd. 2009;154(4):A698.

Approx 60-70 % of all pt die suddenly (Otto Hess JACC 2003)

SCD annual incidence : up to 6 % in tertiary referral center,
1% in regional population (WM, 1999, JACC)

* This relatively low incidence creates a challenge for risk stratification because the false positive values for any stratifier may overwhelm the true positive values.

Risk stratification : difficult due to relatively low prevalence and striking heterogeneity in clinical expression /outcome

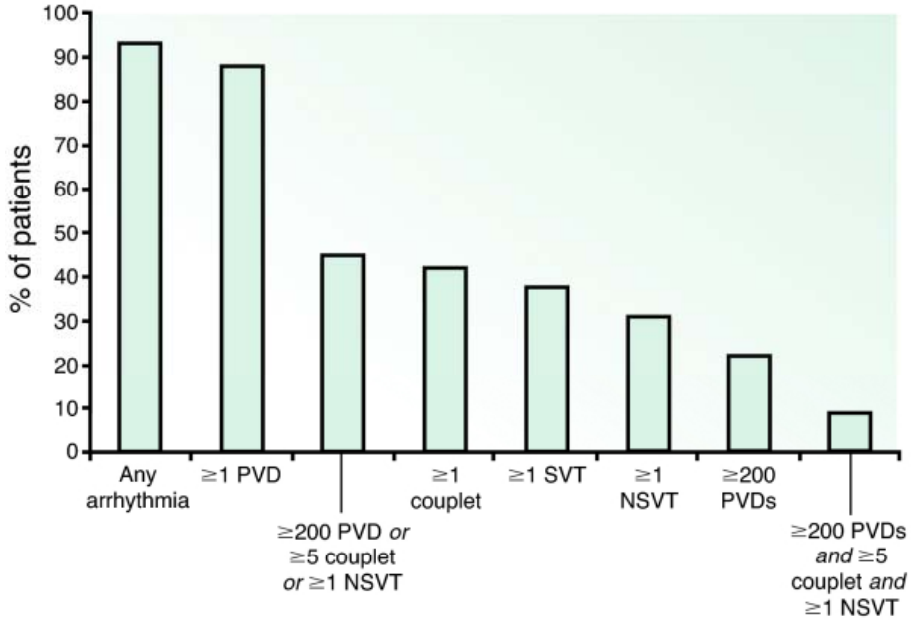
(BM, Heart 2003)

Circulation. 2006;114:e385-e484

Hypertrophic Cardiomyopathy : VT

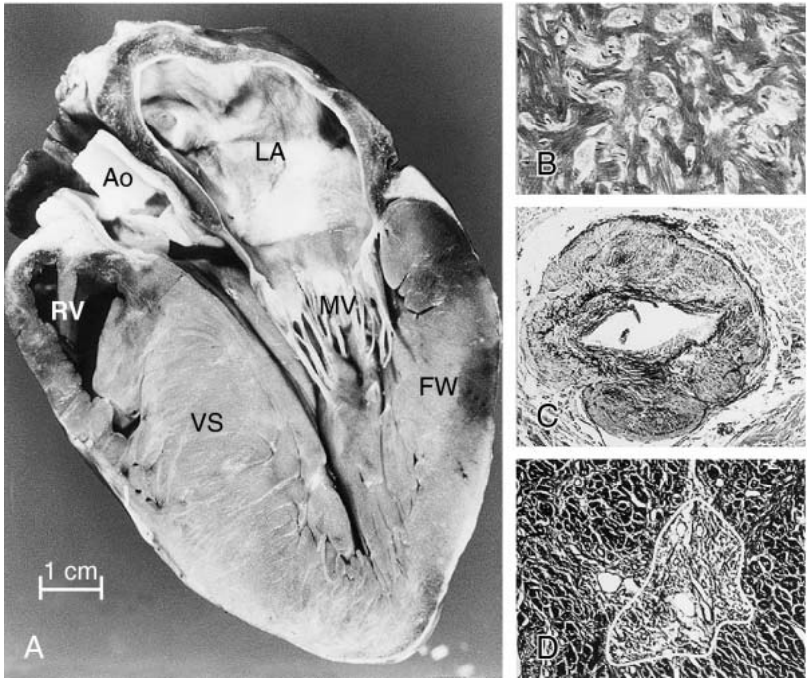
Short bursts of NSVT (usually 3 to 6 beats): markers of SCD, 8%/yr SCD
Sustained monomorphic VT: a high-risk marker

High negative predictive value (i.e., 95%)
Relatively low positive predictive value (i.e., aproximately 20%)
The absence of NSVT on HM: no- or low-risk clinical profile



Prevalence of ventricular and supraventricular arrhythmias on 24-hour ambulatory (Holter)

Hypertrophic Cardiomyopathy : VT



Myocardial substrate

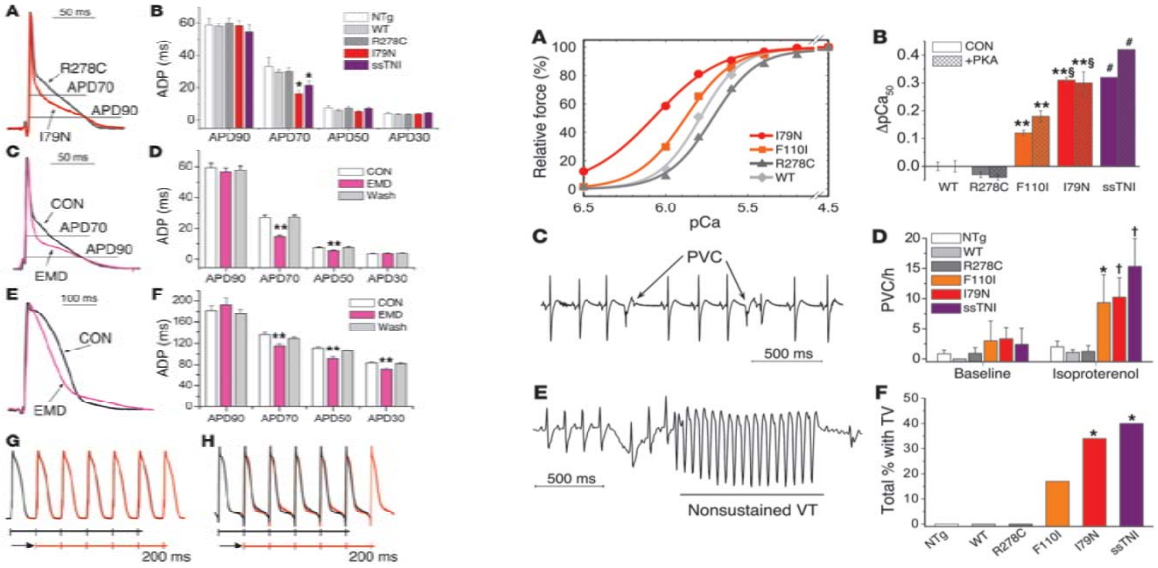
disorganized LV myocardial architecture
“bursts” of myocardial ischemia (narrowed intramural arterioles),
myocyte necrosis and repair in the form of replacement fibrosis and scarring

A variety of triggers

intrinsic (abrupt increase in outflow obstruction)
extrinsic, environmental factors such as intense physical exertion

Myofilament Ca^{2+} sensitization causes susceptibility to cardiac arrhythmia in mice

Franz Baudenbacher,[1] Tilmann Schober,[2] Jose Renato Pinto,[3] Veniamin Y. Sidorov,[1]
Fredrick Hilliard,[1,2] R. John Solaro,[4] James D. Potter,[3] and Björn C. Knollmann[2]



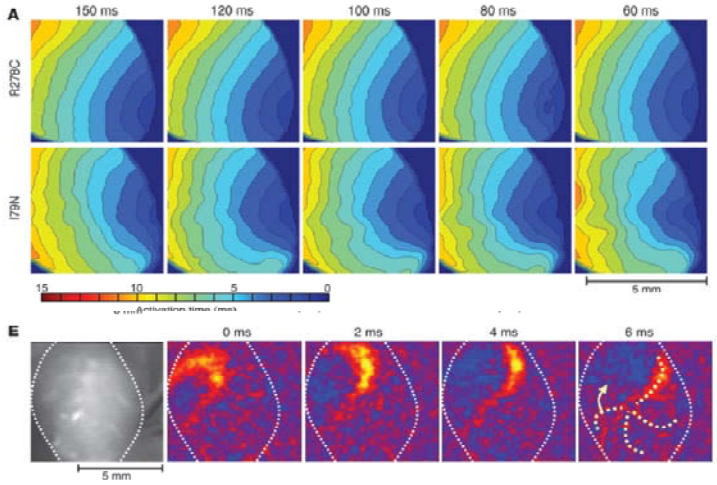


Figure 3

increased myofilament Ca2+ sensitivity
decreased Ca2+ transients w a slower decay rate
AP shortening and triangulation
incomplete recovery of Na+ channel
APD alternans, spatial dispersion of ventricular activation

Knollmann et al. J Clin Invest

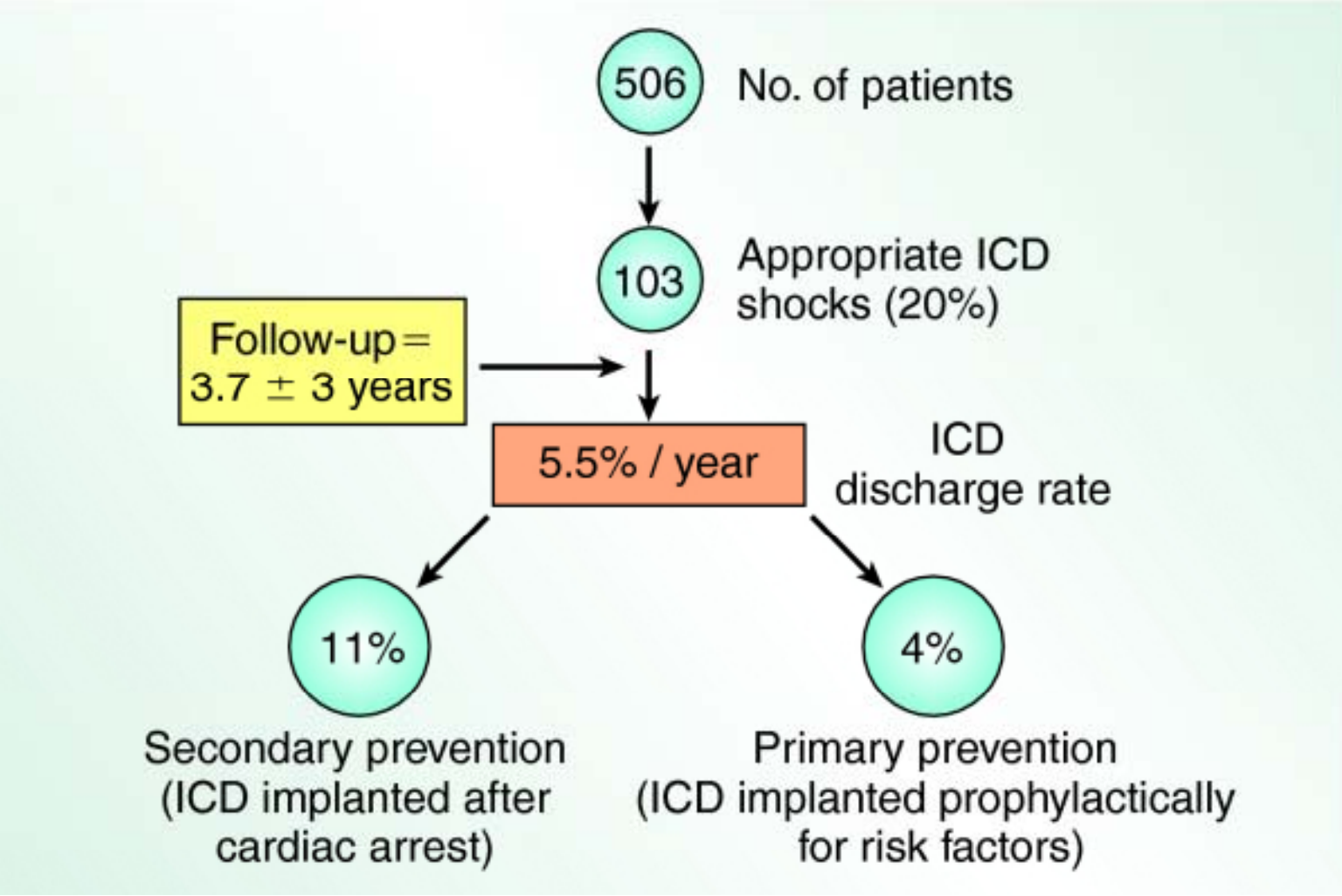
Hypertrophic Cardiomyopathy : risk stratification

Major Risk Factors	Possible in Individual Patients
Cardiac arrest (VF)	AF
Spontaneous sustained VT	Myocardial ischemia
Family history of premature SCD	LV outflow obstruction
Unexplained syncope	High-risk mutation
LV thickness >= 30 mm	Intense (competitive) physical exertion
Abnormal exercise BP	

* The degree of outflow obstruction has been shown to predict cardiovascular death but not SCD.

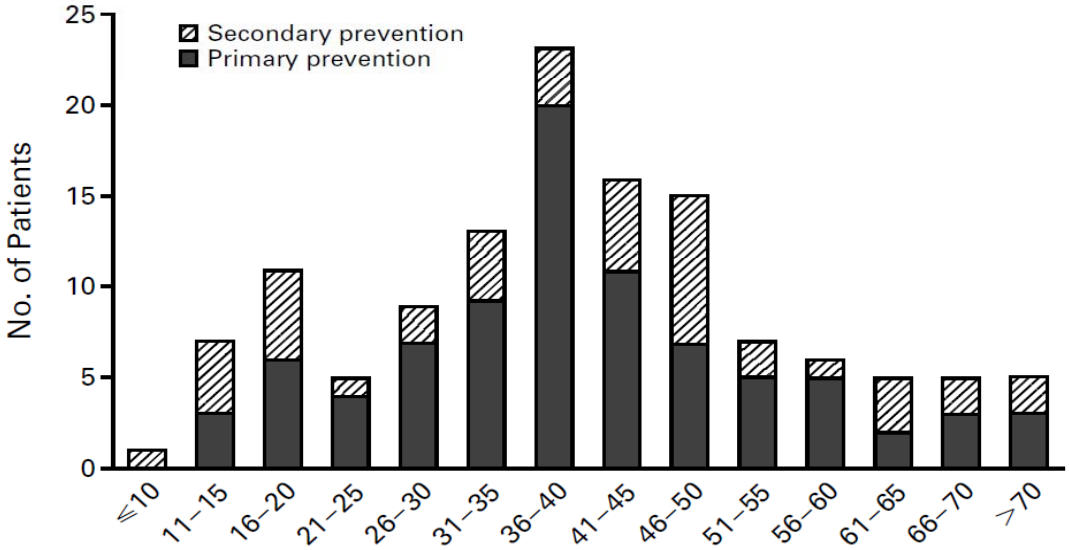
Circulation. 2006;114:e385-e484

Outcome for high-risk patients with HCM and ICDs

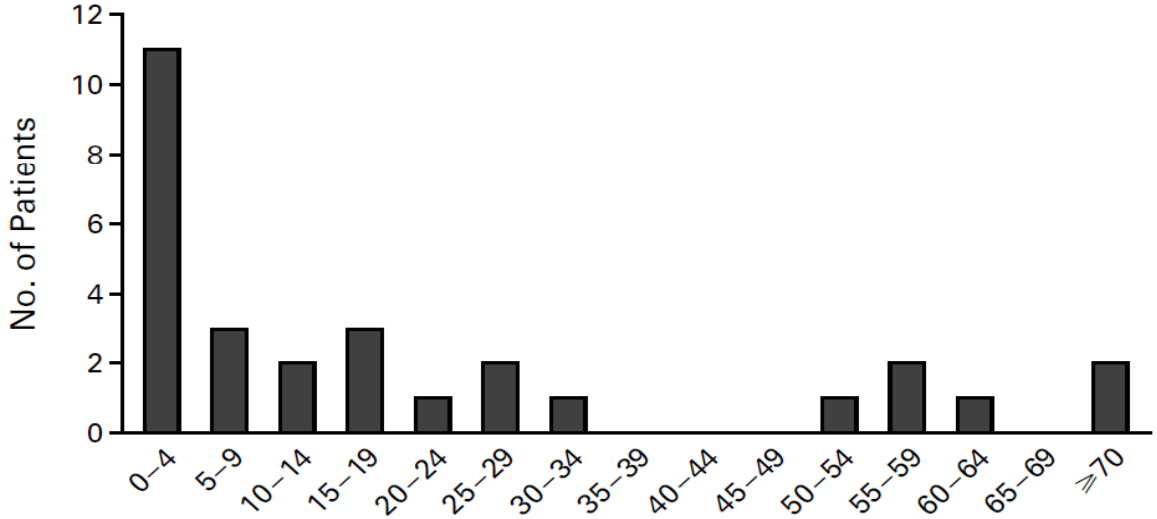


Unpredictable timing of lethal tachyarrhythmias and SCD in HCM

Age at the time of implantation(yr)



Interval btw implantation and discharge(months)



(N Engl J Med 2000;342:365-73.)

Arrhythmogenic Right Ventricular Dysplasia

A type of cardiomyopathy

Fatty infiltration, thinning and dilatation of RV

Fibrous, adipose replacementof the RV muscle

Sporadic, familial (30-50%), genetic

Male predominates

VT with LBBB and inferior axis, RV dysfn

Resting ECG :

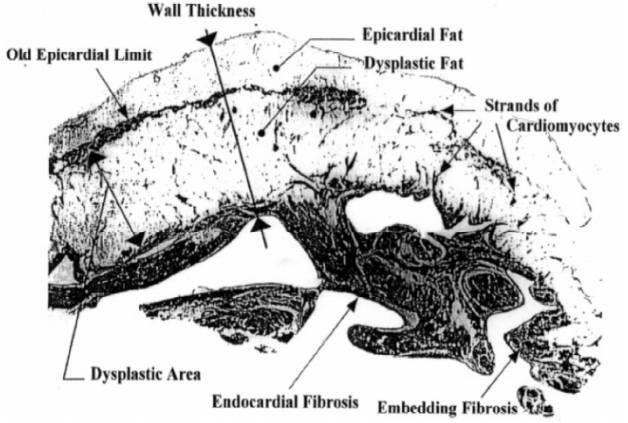
in(complete) RBBB, Epsilon wave(30-55%),

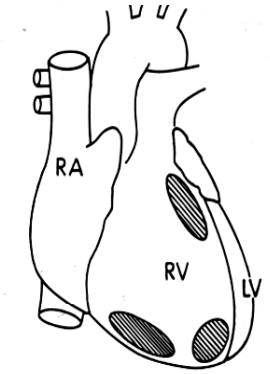
T wave inversion in ant precordial leads

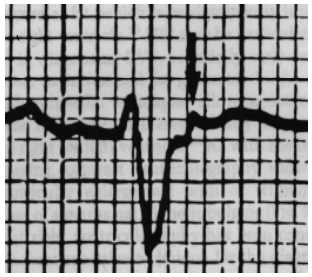
Triangle of dysplasia

Usually abnormal in Echo, MRI, RV graphy, CT

Abnormal SAECG (50-80%)







Triangle of dysplasia:
apex, infundibulum, basal inferior wall

Arrhythmogenic Right Ventricular Dysplasia: SCD

The annual incidence of SCD has varied, ranging from 0.08% to 9%.
(In one Italian series, up to 25% of SCD in athletes was related to ARVC.)
SCD is frequently the first manifestation of the disease.
SCD occurs relatively frequently during exercise or during stress, but SCD with no apparent provocation is not uncommon.

Although SCD usually occurs in individuals with grossly visible RV abnormalities, it can occur in those with only microscopic abnormalities and no obvious RV enlargement.

RV dilation, precordial repolarization abnormalities, and LV involvement have been associated with risk of sudden death.
Certain genetic types may be associated with higher risk of SCD.

DDx from benign RVOT VT

Circulation. 2006;114:e385-e484

Natural History and Risk Stratification of Arrhythmogenic Right Ventricular Dysplasia/Cardiomyopathy

Jean-Sébastien Hulot, MD; Xavier Jouven, MD, PhD; Jean-Philippe Empana, MD; Robert Frank, MD; Guy Fontaine, MD, PhD

Background—Management of patients with arrhythmogenic right ventricular dysplasia/cardiomyopathy (ARVD/C) is complicated by the incomplete information on the natural history of the disease and by the lack of risk stratification for cardiovascular death. The aim of the study was the identification of risk factors related to long-term prognosis.

Methods and Results—Data were collected from 130 patients (100 men; age at onset of symptoms, 31.8±14.4 years) from a tertiary center between 1977 and 2000 who fulfilled the international standardized diagnostic criteria for ARVD/C. Risk factors for cardiovascular death were determined by a logistic regression model. After a mean follow-up of 8.1±7.8 years, 24 deaths were recorded, with a mean age at death of 54±19 years (annual mortality rate, 2.3%). There were 21 deaths with a cardiovascular origin (progressive heart failure for 14 patients and sudden death for the remaining 7 patients). All patients who died had a history of ventricular tachycardia. Multivariate analysis showed that after adjustment for sex, history of syncope, chest pain, inaugural ventricular tachycardia, recurrence of ventricular tachycardia, and QRS dispersion, clinical signs of right ventricular failure and left ventricular dysfunction both remained independently associated with cardiovascular mortality. The combined presence of one of these risk factors and ventricular tachycardia identifies high-risk subjects for cardiovascular mortality, whereas patients without ventricular tachycardia displayed the best prognosis.

Conclusions—The information on the natural history of patients with ARVD allowed us to identify risks factors for cardiovascular mortality. An analysis of a large international registry is needed to refine these results. **(*Circulation*. 2004;110:1879-1884.)**

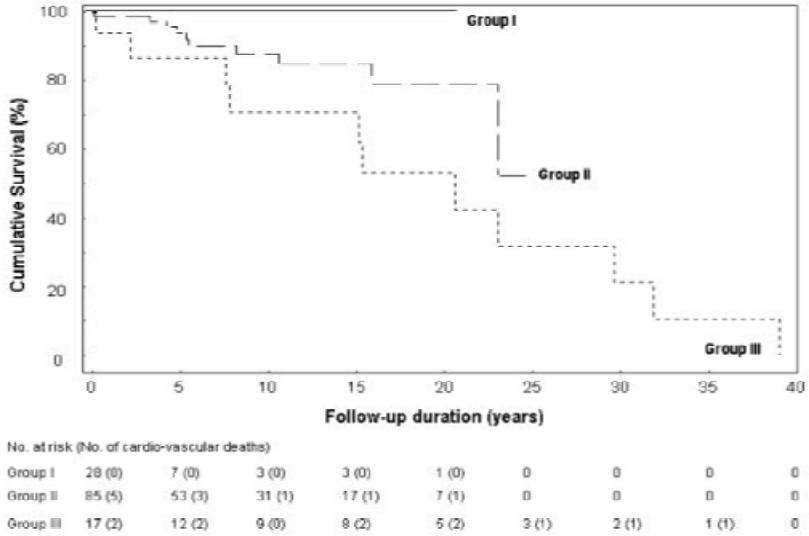
Key Words: cardiomyopathy ■ death, sudden ■ heart failure ■ risk factors

* There were no deaths among the 10 patients who had an ICD.

TABLE 2. Univariate and Multivariate Analysis of Factors Predicting Cardiovascular Death

	Univariate Analysis			Multivariate Analysis*		
	OR	95% CI	P	OR	95% CI	*P*
Syncope	3.51	1.34–9.17	0.01	...	...	...
Atypical chest pain	3.06	1.16–8.02	0.02	...	...	...
Clinical signs of right ventricular failure	10.99	2.40–50.73	0.002	13.70	2.58–71.42	0.002
Inaugural ventricular tachycardia	3.04	1.16–7.93	0.02	...	...	...
Recurrence of ventricular tachycardia	2.69	1.03–7.05	0.04	...	...	...
QRS dispersion >40 ms	2.30	0.89–5.95	0.08	...	...	...
Left ventricular dysfunction	10.64	3.02–37.03	0.002	10.87	2.84–41.7	0.0005

*Stepwise logistic regression adjusted for syncope, chest pain, clinical signs of right ventricular failure, inaugural ventricular tachycardia, recurrence of ventricular tachycardia, QRS dispersion, and left ventricular dysfunction.



All patients who died of a CV cause had at least 1 episode of LBBB form VT. There were no deaths among the 28 patients in whom no VT was documented.

group 1: patients without VT (n28)
group 2: patients with VT but no RV failure or LV dysfunction (n85)
group 3: patients with VT and RV failure and/or LV dysfunction (n17)

Annual mortality rate
0% in group 1, 1.4% in group 2, 4.7% in group 3

(*Circulation*. 2004;110:1879-1884.)

Arrhythmogenic Right Ventricular Dysplasia
A United States Experience

Darshan Dalal, MD, MPH; Khurram Nasir, MD, MPH; Chandra Bomma, MD; Kalpana Prakasa, MD; Harikrishna Tandri, MD; Jonathan Piccini, MD; Ariel Roguin, MD; Crystal Tichnell, MGC; Cynthia James, PhD, ScM; Stuart D. Russell, MD; Daniel P. Judge, MD; Theodore Abraham, MD; Philip J. Spevak, MD; David A. Bluemke, MD, PhD; Hugh Calkins, MD

Background—Arrhythmogenic right ventricular dysplasia (ARVD) is an inherited cardiomyopathy characterized by right ventricular dysfunction and ventricular arrhythmias. The purpose of our study was to describe the presentation, clinical features, survival, and natural history of ARVD in a large cohort of patients from the United States.

Methods and Results—The patient population included 100 ARVD patients (51 male; median age at presentation, 26 [interquartile range {IQR}, 18 to 38; range, 2 to 70] years). A familial pattern was observed in 32 patients. The most common presenting symptoms were palpitations, syncope, and sudden cardiac death (SCD) in 27%, 26%, and 23% of patients, respectively. Among those who were diagnosed while living (n=69), the median time between first presentation and diagnosis was 1 (range, 0 to 37) year. During a median follow-up of 6 (IQR, 2 to 13; range, 0 to 37) years, implantable cardioverter/defibrillators (ICD) were implanted in 47 patients, 29 of whom received an appropriate ICD discharge, including 3 patients who received the ICD for primary prevention. At follow-up, 66 patients were alive, of whom 44 had an ICD in place, 5 developed signs of heart failure, 2 had a heart transplant, and 18 were on drug therapy. Thirty-four patients died either at presentation (n=23: 21 SCD, 2 noncardiac deaths) or during follow-up (n=11: 10 SCD, 1 of biventricular heart failure), of whom only 3 were diagnosed while living and 1 had an ICD implanted. On Kaplan-Meier analysis, the median survival in the entire population was 60 years.

Conclusions—ARVD patients present between the second and fifth decades of life either with symptoms of palpitations and syncope associated with ventricular tachycardia or with SCD. Diagnosis is often delayed. Once diagnosed and treated with an ICD, mortality is low. There is a wide variation in presentation and course of ARVD patients, which can likely be explained by the genetic heterogeneity of the disease. **(*Circulation*. 2005;112:3823-3832.)**

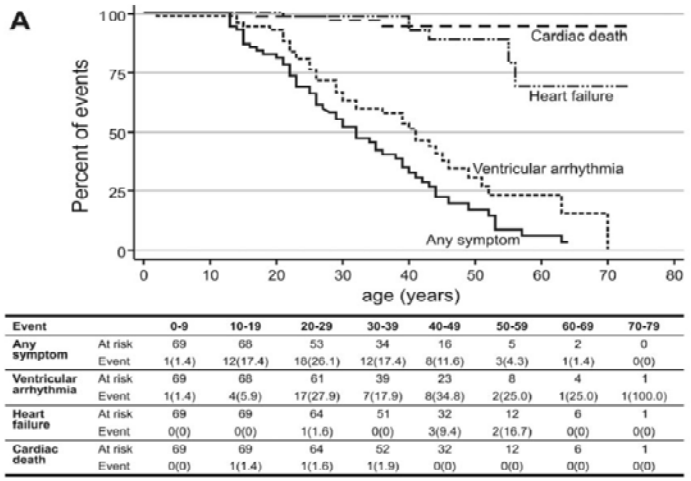
Key Words: arrhythmia ■ cardiomyopathy ■ death, sudden ■ heart failure ■ tachycardia

ARVD: A United States Experience

TABLE 2. Clinical Data and International Task Force Criteria in the 69 ARVD Patients Who Were Diagnosed While Living

Clinical Characteristic	n=69
Imaging and cine imaging studies	
Severe dilatation and reduction of RVEF with no LV impairment*	21 (30)
Localized ventricular aneurysms*	7 (10)
Severe segmental dilatation of the RV*	7 (10)
Mild dilatation and reduction of RVEF with no LV impairment†	36 (67)
Mild segmental dilatation of RV†	8 (12)
Regional RV hypokinesis†	37 (54)
ECG abnormalities	
Prolongation of QRS in leads V_1 through V_3*	40 (58)
Presence of epsilon waves*	20 (29)
Inverted T waves in leads V_1 through V_3 or beyond†	56 (81)
S-wave upstroke ≥55 ms in leads V_1 through V_3	53/58 (91)
SAECG abnormalities	
Late potentials on SAECG†	40/59 (67)
Arrhythmias	
LBBB type VT documented†	51/66 (77)
Frequent ventricular extrasystoles†	35/52 (67)
Family history	
Family history confirmed by biopsy or autopsy*	12 (17)
SCD in family at <35 years of age†	4 (6)
Family members diagnosed by the present criteria†	6 (9)
Histopathological study of biopsy/autopsy specimen	
Infiltration of RV by fat with presence of surviving strands or cardiomyocytes (n=25)*	12/29 (41)

Data are represented as frequency (%). EF indicates ejection fraction; LV, left ventricle; and LBBB, left bundle branch block.
*Major criteria; †minor criteria.



Event		0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79
Any symptom	At risk	69	68	53	34	16	5	2	0
	Event	1(1.4)	12(17.4)	18(26.1)	12(17.4)	8(11.6)	3(4.3)	1(1.4)	0(0)
Ventricular arrhythmia	At risk	69	68	61	39	23	8	4	1
	Event	1(1.4)	4(5.9)	17(27.9)	7(17.9)	8(34.8)	2(25.0)	1(25.0)	1(100.0)
Heart failure	At risk	69	69	64	51	32	12	6	1
	Event	0(0)	0(0)	1(1.6)	0(0)	3(9.4)	2(16.7)	0(0)	0(0)
Cardiac death	At risk	69	69	64	52	32	12	6	1
	Event	0(0)	1(1.4)	1(1.6)	1(1.9)	0(0)	0(0)	0(0)	0(0)

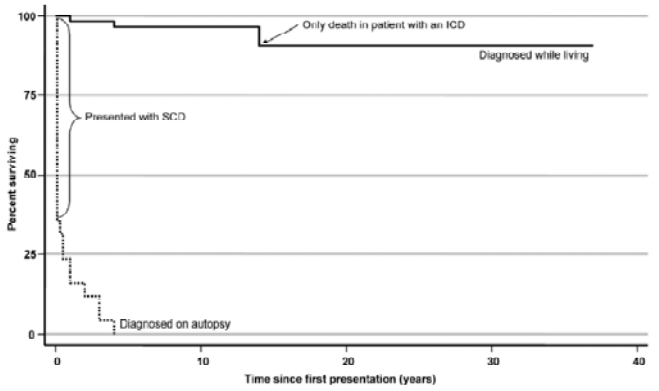


Figure 3. Kaplan-Meier survival analysis demonstrating proportion of patients surviving free from cardiac causes of death since first clinical presentation.

(*Circulation*. 2005;112:3823-3832.)

Comprehensive Desmosome Mutation Analysis in North Americans With Arrhythmogenic Right Ventricular Dysplasia/Cardiomyopathy

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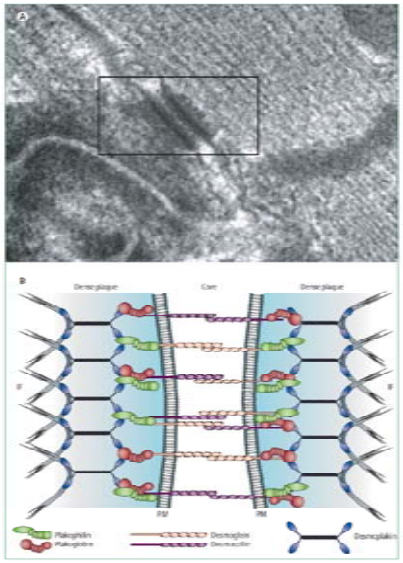
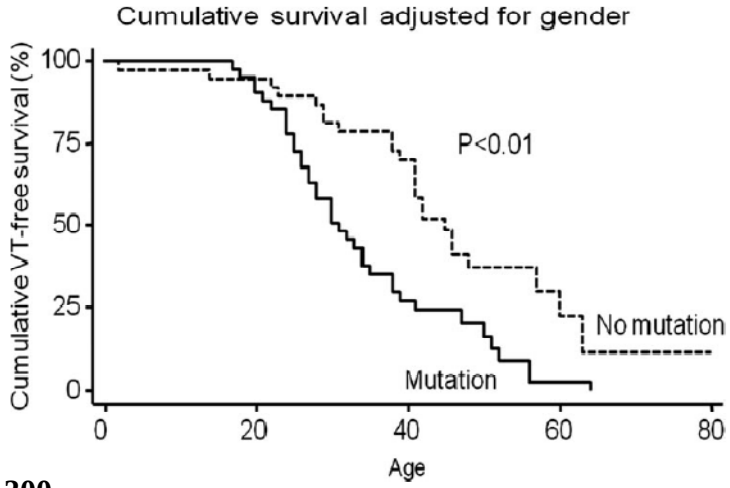
Background—Arrhythmogenic right ventricular dysplasia/cardiomyopathy (ARVD/C) is an inherited disorder typically caused by mutations in components of the cardiac desmosome. The prevalence and significance of desmosome mutations among patients with ARVD/C in North America have not been described previously. We report comprehensive desmosome genetic analysis for 100 North Americans with clinically confirmed or suspected ARVD/C.

Methods and Results—In 82 individuals with ARVD/C and 18 people with suspected ARVD/C, DNA sequence analysis was performed on *PKP2, DSG2, DSP, DSC2,* and *JUP*. In those with ARVD/C, 52% harbored a desmosome mutation. A majority of these mutations occurred in *PKP2*. Notably, 3 of the individuals studied have a mutation in more than 1 gene. Patients with a desmosome mutation were more likely to have experienced ventricular tachycardia (73% versus 44%), and they presented at a younger age (33 versus 41 years) compared with those without a desmosome mutation. Men with ARVD/C were more likely than women to carry a desmosome mutation (63% versus 38%). A mutation was identified in 5 of 18 patients (28%) with suspected ARVD. In this smaller subgroup, there were no significant phenotypic differences identified between individuals with a desmosome mutation compared with those without a mutation.

Conclusions—Our study shows that in 52% of North Americans with ARVD/C a mutation in one of the cardiac desmosome genes can be identified. Compared with those without a desmosome gene mutation, individuals with a desmosome gene mutation had earlier-onset ARVD/C and were more likely to have ventricular tachycardia. **(*Circ Cardiovasc Genet*. 2009;2:428-435.)**





***Lancet* 2009; 373: 1289–1300**

Short- and Long-Term Success of Substrate-Based Mapping and Ablation of Ventricular Tachycardia in Arrhythmogenic Right Ventricular Dysplasia

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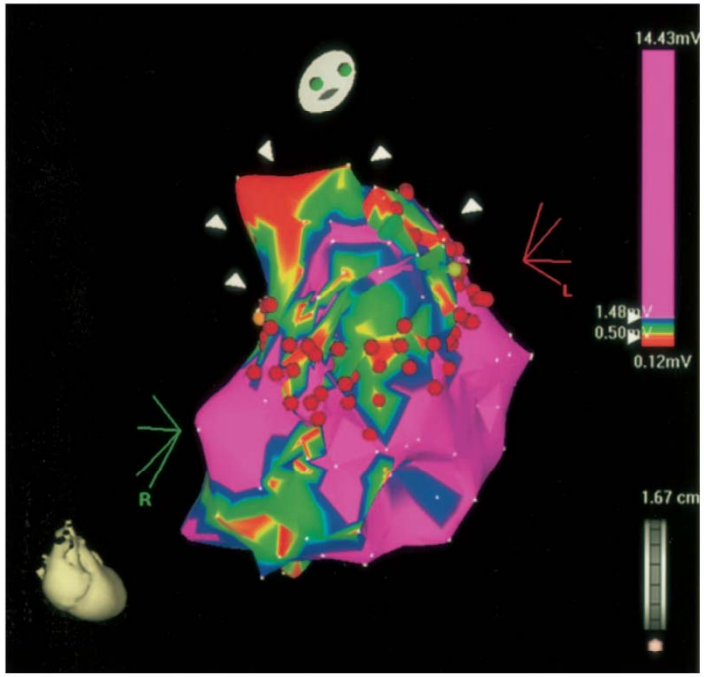
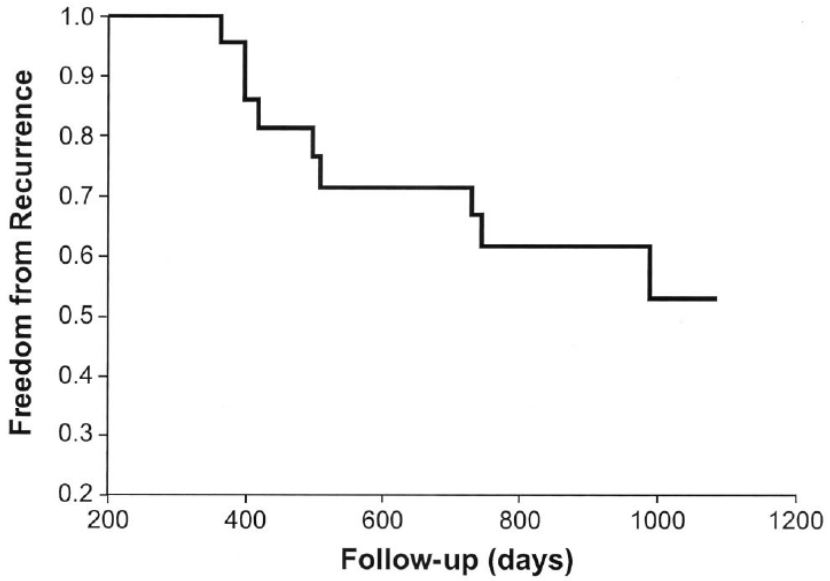
Background—Multiple morphologies, hemodynamic instability, or noninducibility may limit ventricular tachycardia (VT) ablation in patients with arrhythmogenic right ventricular dysplasia (ARVD). Substrate-based mapping and ablation may overcome these limitations. We report the results and success of substrate-based VT ablation in ARVD.

Methods and Results—Twenty-two patients with ARVD were studied. Traditional mapping for VT was limited because of multiple/changing VT morphologies (n=14), nonsustained VT (n=10), or hemodynamic intolerance (n=5). Sinus rhythm CARTO mapping was performed to define areas of "scar" (<0.5 mV) and "abnormal" myocardium (0.5 to 1.5 mV). Ablation was performed in "abnormal" regions, targeting sites with good pace maps compared with the induced VT(s). Linear lesions were created in these areas to (1) connect the scar/abnormal region to a valve continuity or other scar or (2) encircle the scar/abnormal region. Eighteen patients had implanted cardioverter defibrillators, 15 had implanted cardioverter defibrillator therapies, and 7 had sustained VT (6 with syncope). VTs (3±2 per patient) were induced (cycle length, 339±94 ms), and scar was identified in all patients. Scar areas were related to the tricuspid annulus, proximal right ventricular outflow tract, and anterior/inferior-apical walls. Lesions connected abnormal regions to the annulus (n=12) or other scars (n=4) and/or encircled abnormal regions (n=13). Per patient, a mean of 38±22 radiofrequency lesions was applied. Short-term success was achieved in 18 patients (82%). VT recurred in 23%, 27%, and 47% of patients after 1, 2, and 3 years' follow-up, respectively.

Conclusions—Substrate-based ablation of VT in ARVD can achieve a good short-term success rate. However, recurrences become increasingly common during long-term follow-up. (***Circulation*. 2005;111:3209-3216.**)

Circulation. 2005;111:3209-3216





Circulation. 2005;111:3209-3216

Arrhythmia/Electrophysiology

Epicardial Substrate and Outcome With Epicardial Ablation of Ventricular Tachycardia in Arrhythmogenic Right Ventricular Cardiomyopathy/Dysplasia

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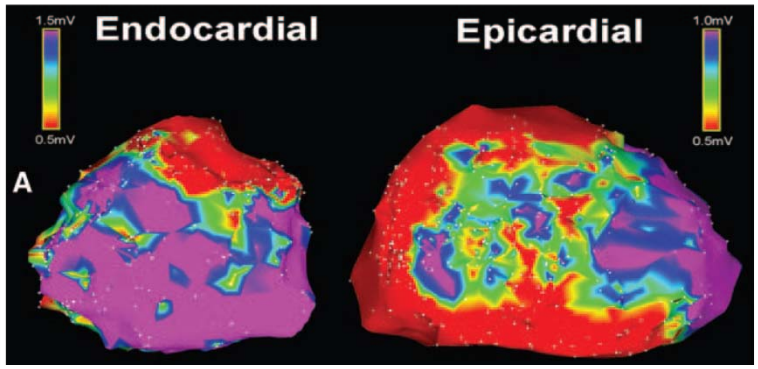
Jian-Fang Ren, MD; Francis E. Marchlinski, MD

Background—Efficacy of endocardial ventricular tachycardia (VT) ablation in arrhythmogenic right ventricular cardiomyopathy/dysplasia may be limited by epicardial VT, right ventricular thickening, or both. We sought to characterize the endocardial versus epicardial substrate, measure right ventricular free wall thickness, and determine epicardial ablation efficacy in patients with right ventricular cardiomyopathy/dysplasia.

Methods and Results—Thirteen consecutive patients (3 female; aged 43±15 years; range, 17 to 70 years) undergoing endocardial and epicardial sinus rhythm voltage mapping and epicardial VT ablation after failed endocardial VT ablation were included. In each patient, the low bipolar voltage area (<1.0 mV for epicardium and <1.5 mV for endocardium) was more extensive on the epicardium (95±47 versus 38±32 cm^2; $P<0.001$) and was uniformly marked by multicomponent and late electrograms. The basal right ventricular thickness assessed by electroanatomic map was >10 mm in 6 of 13 patients compared with 5 to 10 mm in 4 reference patients without structural disease. Twenty-seven VTs were targeted on the epicardium with the use of activation, entrainment, or pace mapping with focal/linear ablation and targeting of late potentials. Epicardial VTs were targeted opposite normal endocardium in 10 patients (77%) and/or opposite ineffective endocardial ablation sites in 11 patients (85%). During 18±13 months, 10 of the 13 patients (77%) had no VT, with 2 patients having only a single VT at 2 and 38 months, respectively.

Conclusions—Patients with right ventricular cardiomyopathy/dysplasia and VT after endocardial ablation have a more extensive epicardial area of electrogram abnormalities and frequently have basal right ventricular wall thickening. Epicardial substrate and VT mapping identifies targets, and ablation results in VT control. (***Circulation*. 2009;120:366-375.**)

Key Words: ablation ■ cardiomyopathy ■ epicardium ■ arrhythmogenic right ventricular cardiomyopathy-dysplasia ■ ventricular tachycardia



***Circulation*. 2009;120:366-375**

Perivalvular Fibrosis and Monomorphic Ventricular Tachycardia: A Unifying Hypothesis in Nonischemic Cardiomyopathy

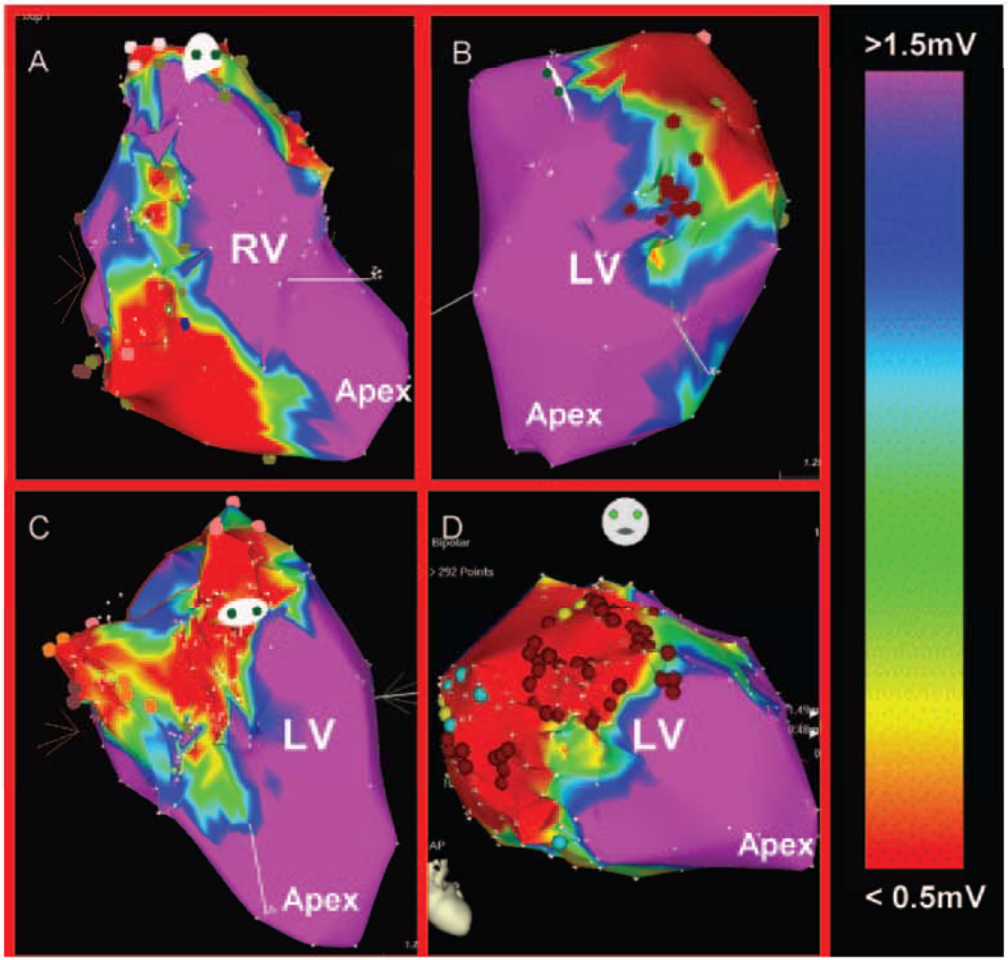


Figure. Bipolar endocardial voltage maps in patients with a variety of nonischemic cardiomyopathies and VT. All maps show a common substrate of perivalvular low voltage (<1.5 mV) consistent with fibrosis as the substrate for scar-based reentry originating from the basal or perivalvular regions of the RV and/or LV, suggesting a link in the pathogenesis among the disorders. A, Right anterior oblique view of the RV bipolar voltage map from a patient with RV cardiomyopathy/dysplasia with low voltage surrounding the peritricuspid RV free wall and extending inferiorly from the pulmonic valve. B, Coronal view of LV bipolar voltage map in a patient with aortic valve replacement 6 years before VT presentation with an area of low voltage in front of the aortic and mitral valves. C, Modified coronal view of a voltage map from a patient with idiopathic nonischemic cardiomyopathy with more extensive involvement that includes regions surrounding the superior mitral and aortic valves and the top of the basal septum. D, Anterior-posterior view of the LV showing an extensive area of low voltage extending from the basal perivalvular region and involving the basal anterior free wall and septum in a patient with a history of biopsy-proven giant cell myocarditis.

Circulation. **2007;116:1998-2001**

요약

1. 다양한 질환의 복합체 **(RV/LV, acquired, ion-channel disease)**
2. 유전이 중요한 원인
3. 심실빈맥은 예후와 밀접한 관련
4. 치료는 제세동기, 재동기화
5. 약물치료는 아직 확실한 자료 없음, 전극도자 절제술은 빈맥의 빈도를 줄일 수 있음