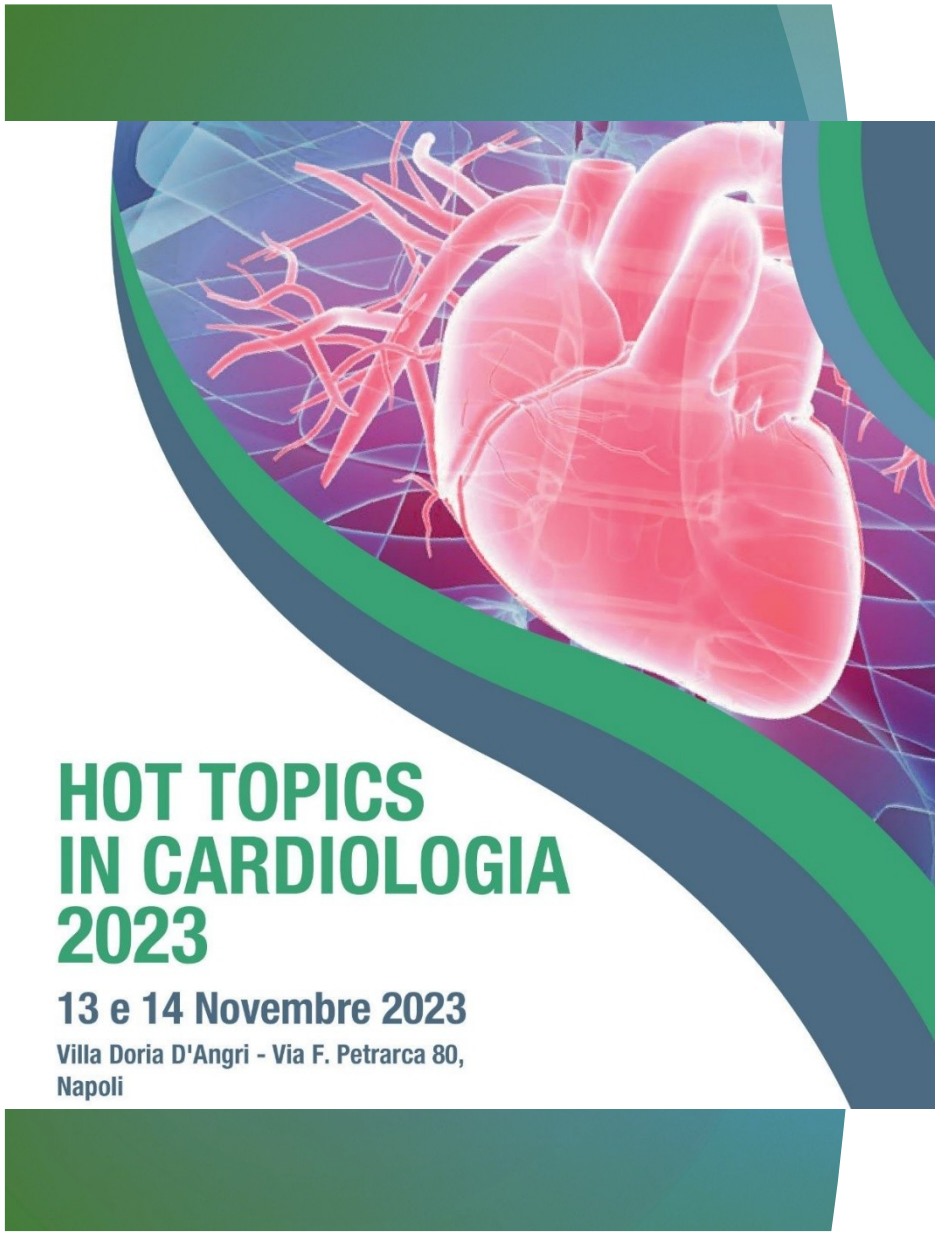

Tachicardia ventricolare: stato dell'arte

Giuseppe Stabile

CLINICA MEDITERRANEA, NAPOLI; CLINICA MONTEVERGINE, MERCOGLIANO (AV)
CLINICA SAN MICHELE, MADDALONI (CE);
CLINICA DEL SOLE, SALERNO;
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DOI: 10.1111/pace.14761

PACE WILEY

ORIGINAL ARTICLE

Trends in ventricular fibrillation/flutter mortality in US population, 1999 to 2019

Marco Zuin MD, FESC, FACC[1,2] | Gianluca Rigatelli MD, PhD, FACC, FSCAI[3] | Claudio Bilato MD, PhD[1]

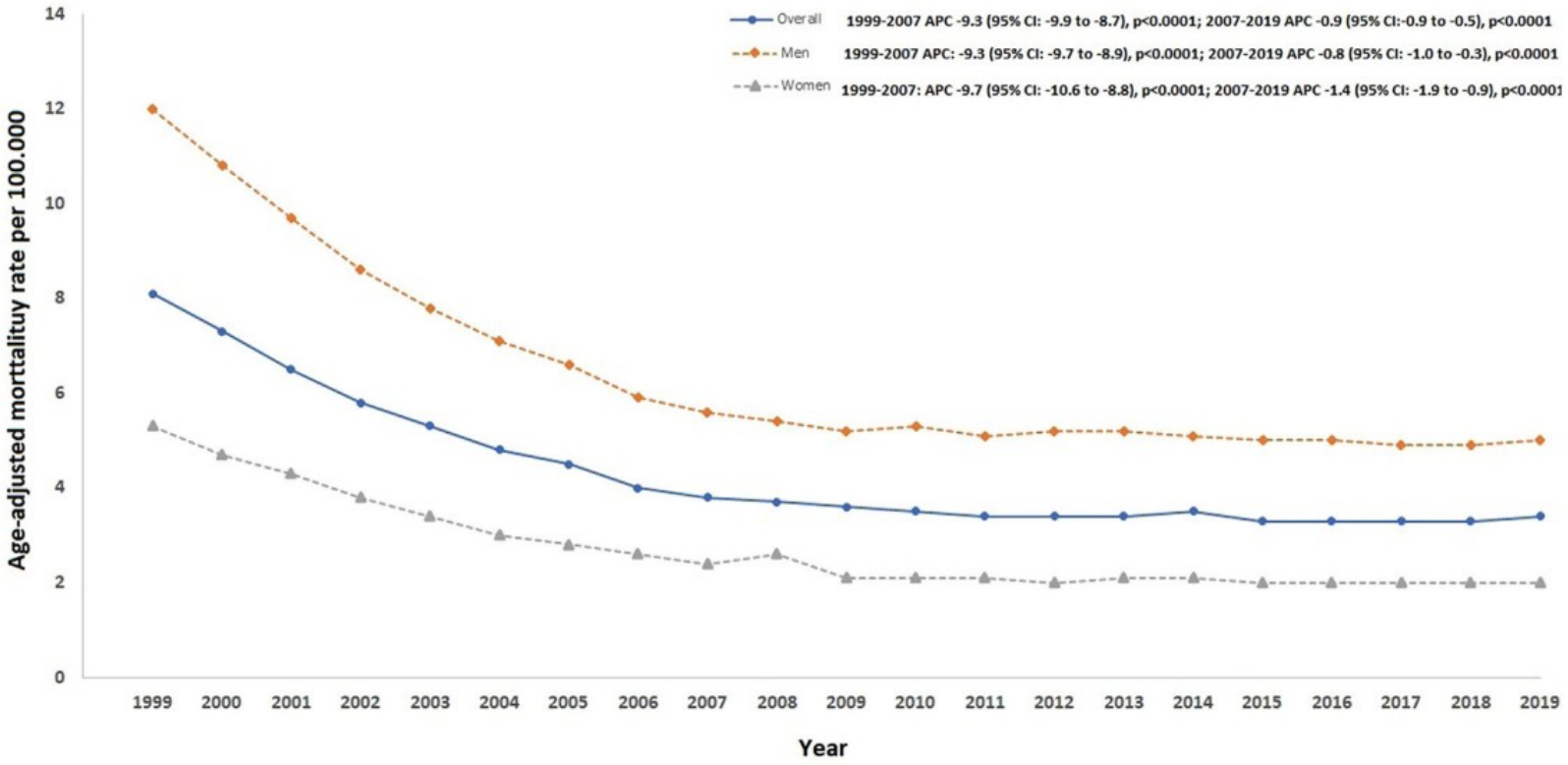

TABLE 1 Underlying causes of death in descendants for ventricular fibrillation/flutter in US 1999–2019.

Underlying causes of deaths	*n*	%
Atherosclerotic disease	61,492	25.3
Acute myocardial infarction	43,915	18.1
Cardiomyopathies (unspecified)	9,263	3.8
Ischemic cardiomyopathy	6,643	2.7
Diabetes mellitus	4,344	1.7

Abbreviation: VF/VFL, ventricular fibrillation/flutter.

Declining incidence of sudden cardiac death from 1990–2010 in a general middle-aged and elderly population: The Rotterdam Study

Maartje N. Niemeijer, MD,[*] Marten E. van den Berg, MD,[†] Maarten J.G. Leening, MD,[*‡] Albert Hofman, MD, PhD,[*] Oscar H. Franco, MD, PhD,[*] Jaap W. Deckers, MD, PhD,[‡] Jan Heeringa, MD, PhD,[*] Peter R. Rijnbeek, PhD,[†] Bruno H. Stricker, MMed, PhD,[*§¶] Mark Eijgelsheim, MD, PhD[*§]

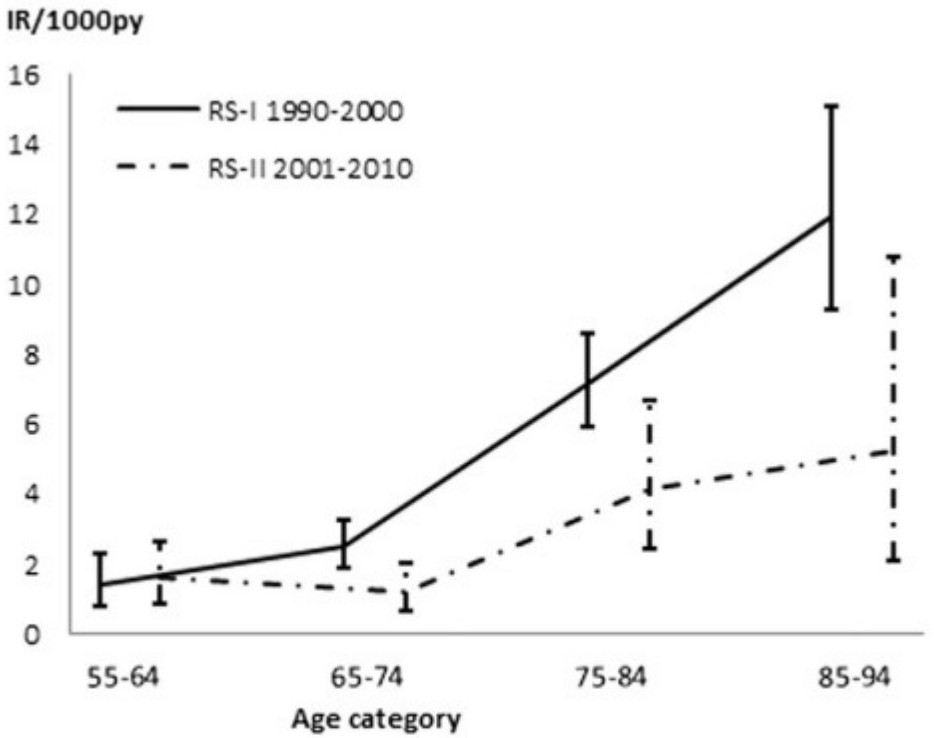

Figure 3 Incidence rate (IR) of sudden cardiac death per 1000 person-years (py) by calendar year for the RS-I (1990–2000) and RS-II (2001–2010) subcohorts.

Cause

Declino delle morti ischemiche (FV nella sindrome coronarica acuta).

L'implementazione della prevenzione primaria e secondaria.

Miglioramenti diagnostici e terapeutici

Table. Examples of Major Advances in the Prevention and Treatment of Coronary Heart Disease

Advance	Year or Period	Impact on the Prevention and Treatment of Coronary Heart Disease and Risk Factors
Framingham Heart Study identified smoking, high BP and high blood cholesterol as major cardiovascular risk factors	1960s	New targets for atherosclerotic coronary heart disease prevention and treatment
First coronary artery bypass surgery	1960	Surgical procedure to bypass clogged arteries
Surgeon General's Report on Smoking and Health	1964	Publicized dangers of cigarette smoking
Hypertension Detection and Follow-up Program	Early 1970s	Demonstrated benefit of treating even moderate hypertension
First percutaneous transvascular coronary angioplasty	1977	Successful restoration of perfusion in occluded coronary arteries via percutaneous catheter
Discovery of the low-density lipoprotein receptor	1970s	Michael Brown and Joseph Goldstein laid the groundwork for statins
LRC-CPPT trial (Lipid Research Clinics Coronary Primary Prevention)	1984	Established benefit of cholesterol lowering
National clinical practice guidelines for high BP and high blood cholesterol	1987	Established standards and targets for BP and cholesterol
Development of statins, angiotensin-converting enzyme-inhibitors and calcium channel blockers	1987–8	New powerful drugs for managing cholesterol and blood pressure
TIMI trial (Thrombolysis in Myocardial Infarction)	1987	Thrombolysis in acute myocardial infarction
First coronary stent	1988	Made angioplasty more durable
Scandinavian Simvastatin Survival Study (4S)	1994	First statin end point trial showed reduction in mortality. Many other statin trials followed
SHEP (Systolic Hypertension in the Elderly)	1996	Established the benefit of treating isolated systolic hypertension in elderly. Many other BP trials followed
SPRINT trial (Systolic Blood Pressure Intervention)	2015	Established the benefit of intensive BP control (to target systolic BP <120 mm Hg) in high-risk patients without diabetes

European Journal of Heart Failure (2014) **16**, 317–324
doi:10.1002/ejhf.16

Long-term prognostic impact of therapeutic strategies in patients with idiopathic dilated cardiomyopathy: changing mortality over the last 30 years

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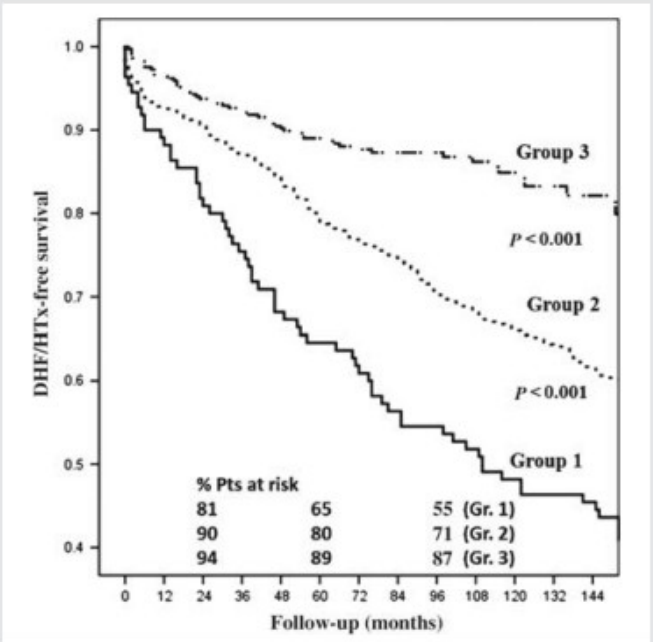


Figure 1 KM curves: survival free from all-cause mortality/heart transplant in IDCM patients according to the decade of enrolment.

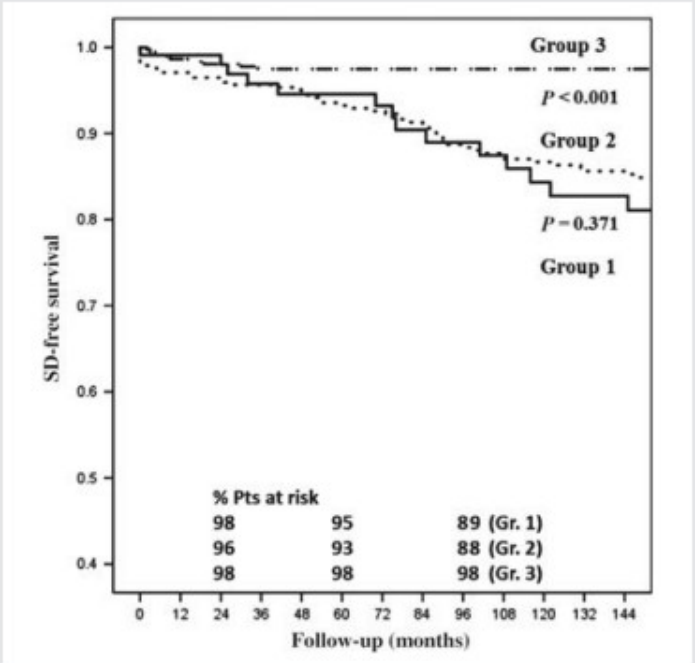


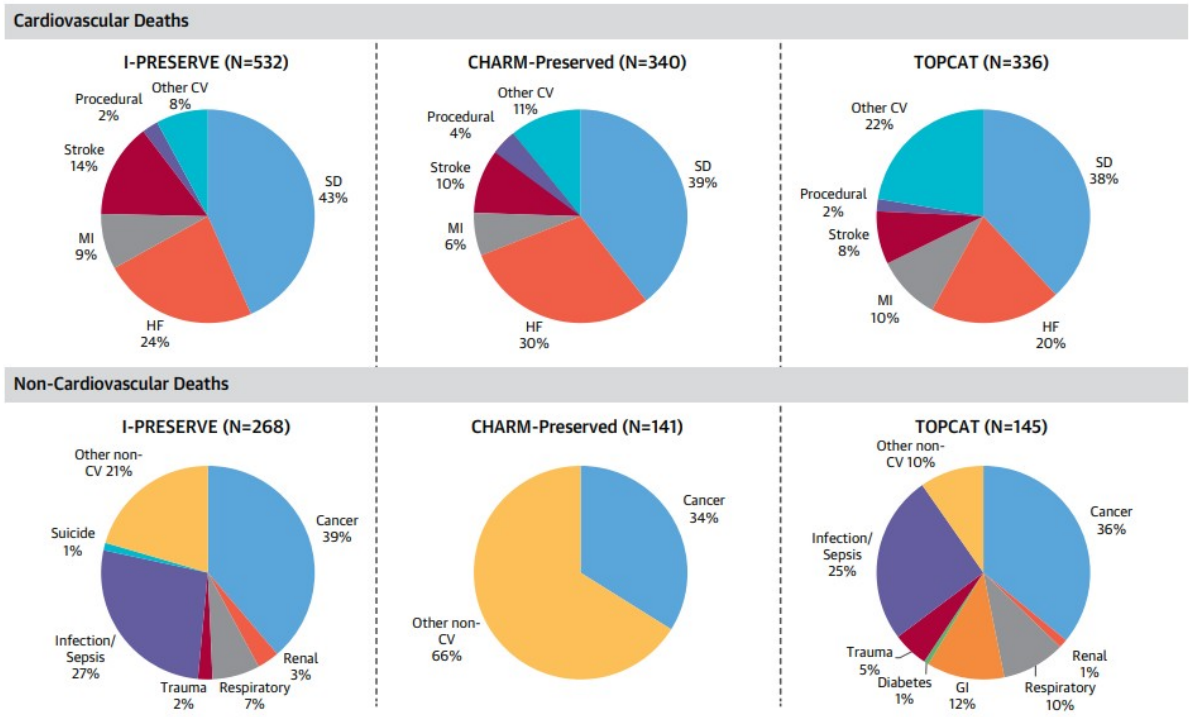
Figure 3 Cause-specific KM curves: survival free from sudden death in IDCM patients according the decade of enrolment.

Concerning SD, lower left ventricular ejection fraction emerged as a predictor, while ICD was the only therapy with a protective role (HR 0.08, CI 95% 0.01–0.61). Treatment with digitalis emerged as a predictor of both DHF/HTx and SD.

Mode of Death in Heart Failure With Preserved Ejection Fraction

Muthiah Vaduganathan, MD, MPH,[a] Ravi B. Patel, MD,[b] Alexander Michel, MD, MSc,[c] Sanjiv J. Shah, MD,[b] Michele Senni, MD,[d] Mihai Gheorghiade, MD,[e] Javed Butler, MD, MPH, MBA[f]

FIGURE 3 Cause-Specific Mortality in RCTs of HFpEF



Mode of death was stratified into CV **(top)** and non-CV deaths **(bottom)**. The N reported refers to the total number of CV or non-CV deaths in each individual trial. GI = gastrointestinal; HF = heart failure; MI = myocardial infarction; SD = sudden death; other abbreviations as in Figure 2.

Circulation

ORIGINAL RESEARCH ARTICLE

An International Multicenter Cohort Study on β-Blockers for the Treatment of Symptomatic Children With Catecholaminergic Polymorphic Ventricular Tachycardia

Puck J. Peltenburg, MD*; Dania Kallas, MS*; Johan M. Bos, MD, PhD*; Krystien V.V. Lieve, MD, MSc (EBP); Sonia Franciosi, PhD; Thomas M. Roston, MD, PhD; Isabelle Denjoy, MD; Katrina B. Sorensen, BA; Seiko Ohno, MD, PhD; Ferran Roses-Noguer, MD; Takeshi Aiba, MD; Alice Maltret, MD; Martin J. LaPage, MD, MS; Joseph Atallah, MD, CM, SM; John R. Giudicessi, MD, PhD; Sally-Ann B. Clur, MBBCh, MSc (Med), PhD; Nico A. Blom, MD, PhD; Michael Tanck, MSc, PhD; Fabrice Extramiana, MD, PhD; Koichi Kato, MD; Julien Barc, PhD; Martin Borggrefe, MD; Elijah R. Behr, MA, MBBS, MD; Georgia Sarquella-Brugada, MD, PhD; Jacob Tfelt-Hansen, MD, DMSc; Esther Zorio, MD, PhD; Heikki Swan, MD; Janneke A.E. Kammeraad, MD, PhD; Andrew D. Krahn, MD; Andrew Davis, MBBS, MD; Frederic Sacher, MD; Peter J. Schwartz, MD; Jason D. Roberts, MD, MAS; Jonathan R. Skinner, MD; Maarten P. van den Berg, MD, PhD; Prince J. Kannankeril, MD; MSCI, Fabrizio Drago, MD; Tomas Robyns, MD; Kristina Haugaa, MD, PhD; Terezia Tavacova, MD; Christopher Semsarian, MB, BS, MPH; Jan Till, MBBS, BSC, MD; Vincent Probst, MD; Ramon Brugada, MD; Wataru Shimizu, MD, PhD; Minoru Horie, MD, PhD; Antoine Leenhardt, MD*; Michael J. Ackerman, MD, PhD*; Shubhayan Sanatani MD*; Christian van der Werf, MD, PhD*; Arthur A.M. Wilde, MD, PhD*

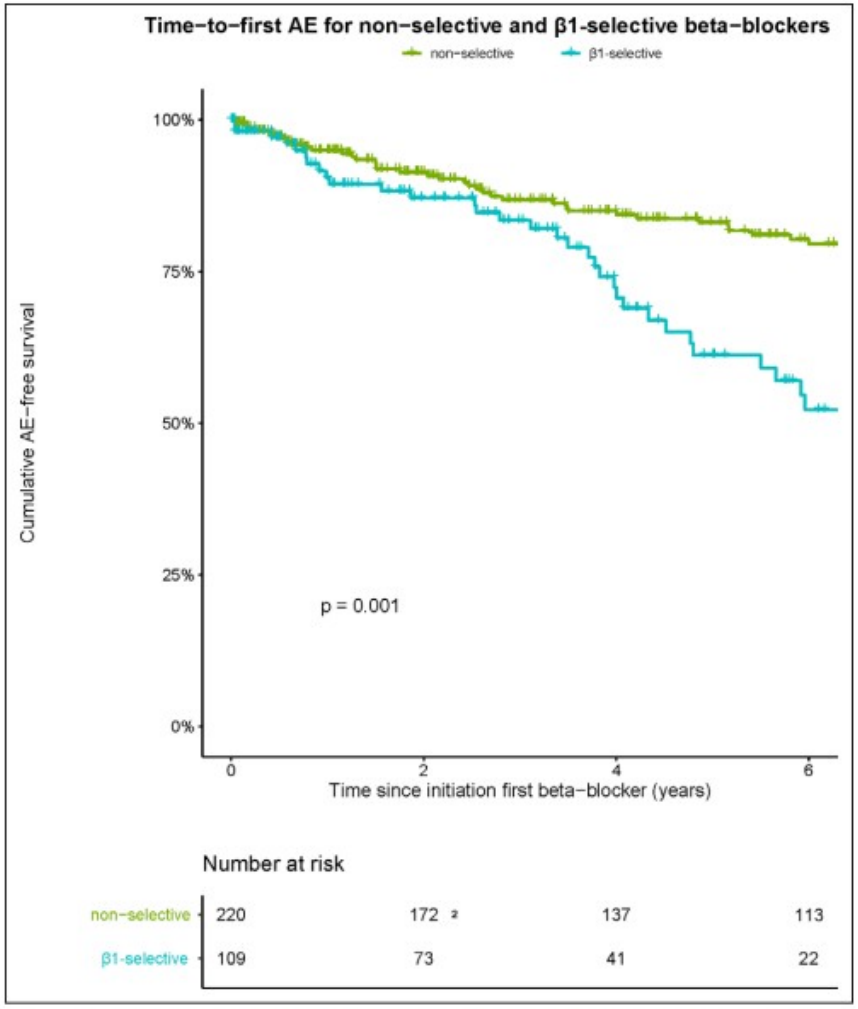


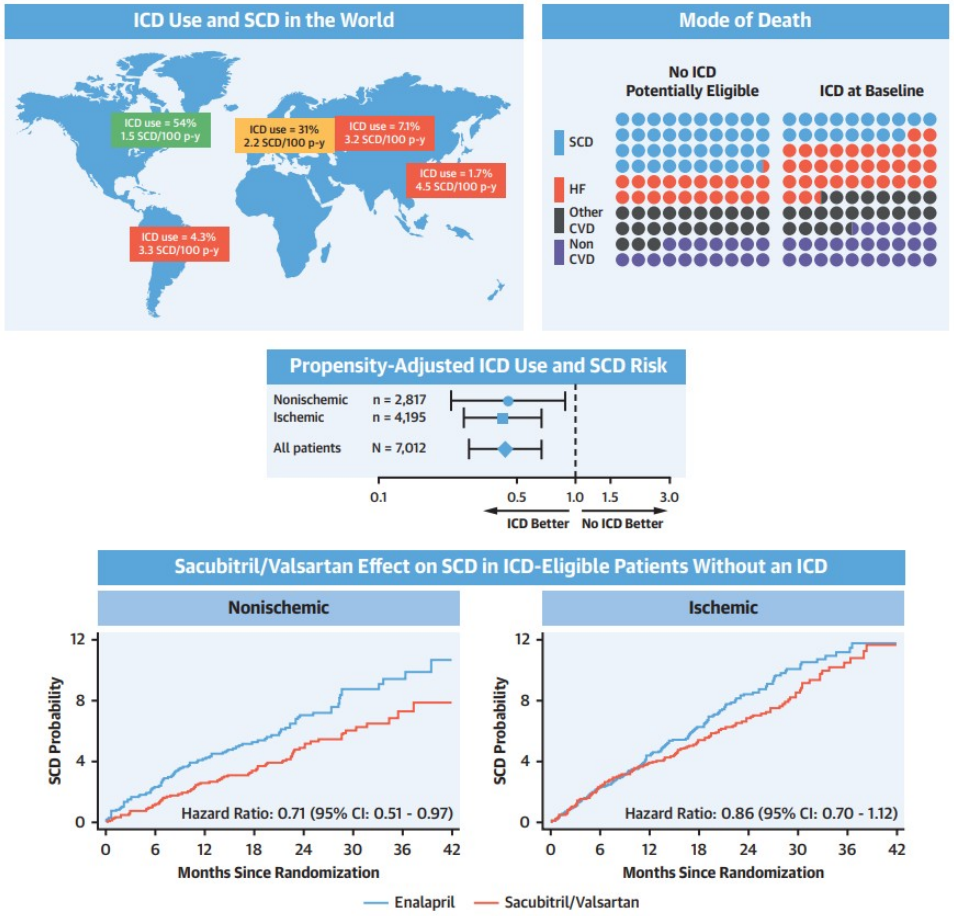
Figure 2. Kaplan-Meier curve showing the occurrence of AE in symptomatic children using nonselective versus β1-selective β-blockers.
AE indicates arrhythmic event.

Sacubitril/Valsartan and Sudden Cardiac Death According to Implantable Cardioverter-Defibrillator Use and Heart Failure Cause

A PARADIGM-HF Analysis

Luis E. Rohde, MD, ScD,[a,b] Neal A. Chatterjee, MD, MSc,[c] Muthiah Vaduganathan, MD, MPH,[a] Brian Claggett, PhD,[a] Milton Packer, MD,[d] Akshay S. Desai, MD,[a] Michael Zile, MD,[e] Jean Rouleau, MD,[f] Karl Swedberg, MD,[g] Martin Lefkowitz, MD,[h] Victor Shi, MD,[h] John J.V. McMurray, MD,[i] Scott D. Solomon, MD[a]

CENTRAL ILLUSTRATION SCD Risk, ICD Use, Mode of Death, and Sacubitril/Valsartan in the PARADIGM-HF Trial



Rohde, L.E. et al. J Am Coll Cardiol HF. 2020;8(10):844-55.

ICD use and SCD rates worldwide **(left upper panel)**, mode of death according to ICD use and eligibility **(right upper panel)**, propensity-adjusted association of ICD use with risk of SCD **(central panel)**, sacubitril-valsartan effect on SCD in patients with nonischemic cardiomyopathy **(lower left panel)** and in patients with ischemic cardiomyopathy **(lower right panel)**. ICD = implantable cardioverter-defibrillator; PARADIGM = Prospective Comparison of ARNI with an ACE-Inhibitor to Determine Impact on Global Mortality and Morbidity in Heart Failure; SCD = sudden cardiac death.

ESC European Society of Cardiology
Europace (2022) 24, 20–30
doi:10.1093/europace/euab177

CLINICAL RESEARCH
Sudden death and ICDs

Association between sodium–glucose cotransporter-2 inhibitors and risk of sudden cardiac death or ventricular arrhythmias: a meta-analysis of randomized controlled trials

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Aims Sudden cardiac death (SCD) and ventricular arrhythmias (VAs) are important causes of mortality in patients with type 2 diabetes mellitus (T2DM), heart failure (HF), or chronic kidney disease (CKD). We evaluated the effect of sodium–glucose cotransporter-2 (SGLT2) inhibitors on SCD and VAs in these patients.

Methods and results We performed a systematic review and meta-analysis of randomized controlled trials (RCTs) that enrolled patients with T2DM and/or HF and/or CKD comparing SGLT2i and placebo or active control. PubMed and ClinicalTrials.gov were systematically searched until November 2020. A total of 19 RCTs with 55,590 participants were included. Sudden cardiac death events were reported in 9 RCTs (48 patients receiving SGLT2i and 57 placebo subjects). There was no significant association between SGLT2i therapy and SCD [risk ratio (RR) 0.74, 95% confidence interval (CI) 0.50–1.08; $P = 0.12$]. Ventricular arrhythmias were reported in 17 RCTs (126 patients receiving SGLT2i and 134 controls). SGLT2i therapy was not associated with a lower risk of VAs (RR 0.84, 95% CI 0.66–1.06; $P = 0.14$). Besides the subgroup of low-dosage SGLT2i therapy that demonstrated decreased VAs compared to control (RR 0.45, 95% CI 0.25–0.82; $P = 0.009$), or to placebo (RR 0.46, 95% CI 0.25–0.85; $P = 0.01$), further subgroup analysis did not demonstrate any significant differences.

Conclusion SGLT2i therapy was not associated with an overall lower risk of SCD or VAs in patients with T2DM and/or HF and/or CKD. However, further research is needed since the number of SCD and VA events were relatively few leading to wide confidence intervals, and the point estimates suggested potential benefits.

Association of SGLT2 inhibitors with arrhythmias and sudden cardiac death in patients with type 2 diabetes or heart failure: A meta-analysis of 34 randomized controlled trials

Check for updates

Gilson C. Fernandes, MD,* Amanda Fernandes, MD,† Rhanderson Cardoso, MD,‡ Jorge Penalver, MD,* Leonardo Knijnik, MD,† Raul D. Mitrani, MD, FHRS,* Robert J. Myerburg, MD,* Jeffrey J. Goldberger, MD, MBA, FHRS*

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BACKGROUND Sodium-glucose cotransporter 2 inhibitors (SGLT2is) reduce hospitalizations and death from heart failure (HF), but their effect on arrhythmia expression has been poorly investigated.

OBJECTIVE The purpose of this study was to evaluate the association of SGLT2is with arrhythmias in patients with type 2 diabetes mellitus (T2DM) or HF.

METHODS We searched PubMed and ClinicalTrials.gov. Two independent investigators identified randomized double-blind trials that compared SGLT2is with placebo or active control for adults with T2DM or HF. Primary outcomes were incident atrial arrhythmias, ventricular arrhythmias (VAs), and sudden cardiac death (SCD).

RESULTS We included 34 randomized (25 placebo-controlled and 9 active-controlled) trials with 63,166 patients (35,883 SGLT2is vs 27,273 control: mean age 53–67 years; 63% male). Medications included canagliflozin, dapagliflozin, empagliflozin, or ertugliflozin. Except for 1 study of HF, all patients had T2DM. Follow-up ranged from 24 weeks to 5.7 years. The cumulative incidence of events was low: 3.6, 1.4, and 2.5 per 1000 patient-years for atrial arrhythmias, VAs and SCD, respectively. SGLT2i therapy was associated with a significant reduction in the risk of incident atrial arrhythmias (odds ratio 0.81; 95% confidence interval 0.69–0.95; $P = .008$) and the "SCD" component of the SCD outcome (odds ratio 0.72; 95% confidence interval 0.54–0.97; $P = .03$) compared with control. There was no significant difference in incident VA or the "cardiac arrest" SCD component between groups.

CONCLUSION SGLT2is are associated with significantly reduced risks of incident atrial arrhythmias and SCD in patients with T2DM. Prospective trials are warranted to confirm the antiarrhythmic effect of SGLT2is and whether this is a class or drug-specific effect.

KEYWORDS Sodium-glucose cotransporter 2 inhibitors; Atrial fibrillation; Ventricular arrhythmia; Sudden cardiac death; Meta-analysis

(Heart Rhythm 2021;18:1098–1105)

The NEW ENGLAND JOURNAL of MEDICINE

REVIEW ARTICLE

John A. Jarcho, M.D., *Editor*

Catheter Ablation of Ventricular Arrhythmias

Kalvanam Shivkumar. M.D.. Ph.D.

Table 2. Selected Clinical Trials of VT Ablation.*

Trial	Design	Study Population (Comparison)	No. of Patients	Months of Follow-up†	Outcome
SMASH VT,[49] 2007	RCT	Patients with ICM (ICD plus VT ablation vs. ICD alone)	128	22.5±5.5	Ablation superior (incidence of ICD therapy, 12% in ablation group vs. 33% in control group at 2 yr)
VTACH,[50] 2010	RCT	Patients with ICM (ICD plus VT ablation vs. ICD alone for stable VT)	107	22.5±9.0	Ablation superior (median time to recurrence of VT or VF, 18.6 mo in ablation group vs. 5.9 mo in control group)
VANISH,[48] 2016	RCT	Patients with ICM (VT ablation vs. escalation of antiarrhythmic-drug therapy for drug-refractory VT)	259	27.9±17.1	Ablation superior (primary composite end point of death, VT storm, or appropriate ICD shock, 59.1% in ablation group vs. 68.5% in control group)
Multicenter Thermocool VT Ablation Trial,[26] 2008	Observational	Patients with ICM	231	12	Catheter ablation of VT is a reasonable option for clinical management (freedom from recurrent VT, 53% at 6 mo)
IVTCC,[47] 2015	Retrospective	Patients with ICM or NICM	2061	12	Freedom from VT recurrence, 70% at 1 yr; transplantation-free survival, 90% for patients without recurrence vs. 71% for those with recurrence

* ICD denotes implantable cardioverter–defibrillator, ICM ischemic cardiomyopathy, IVTCC International VT Ablation Center Collaborative Group, NICM nonischemic cardiomyopathy, RCT randomized, controlled trial, SMASH-VT Substrate Mapping and Ablation in Sinus Rhythm to Halt Ventricular Tachycardia, VANISH Ventricular Tachycardia Ablation versus Escalated Antiarrhythmic Drug Therapy, VF ventricular fibrillation, and VTACH Ventricular Tachycardia Ablation in Coronary Heart Disease.
† Plus–minus values are means ±SD.

Catheter ablation has become well established in the clinical management of ventricular tachycardia. Ablation has a high rate of success for patients with PVCs and those with idiopathic ventricular tachycardia.

Among patients with structural heart disease, success rates are not as high, although technological advancements have resulted in improved outcomes. If ventricular tachycardia is not successfully controlled with catheter ablation or if it recurs despite ablation, the chance of survival is decreased.

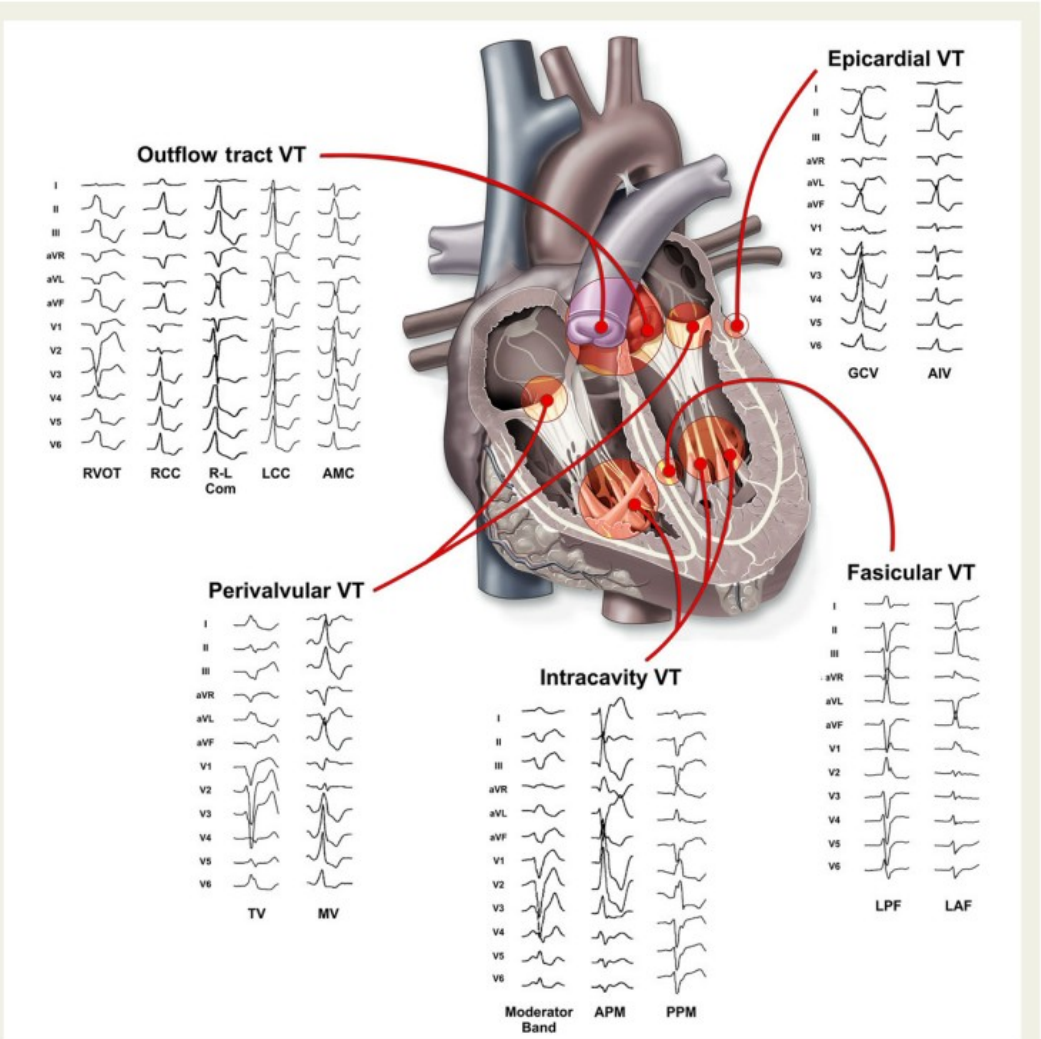
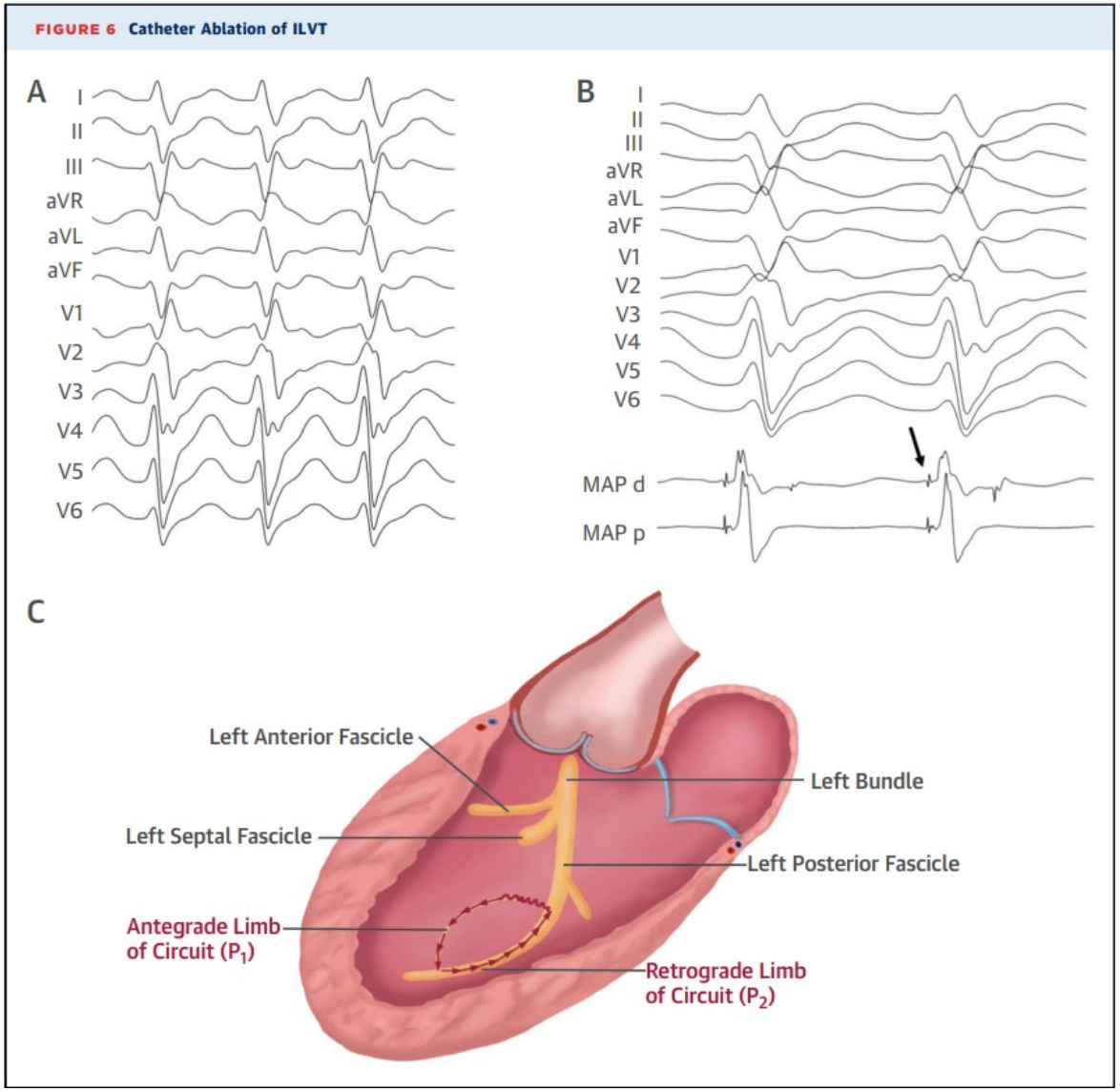


Figure 1 Twelve-lead electrocardiogram morphology of different sites of origin in idiopathic ventricular tachycardia (RVOT, right ventricular outflow tract; RCC, right coronary cusp; R–L com, right–left coronary cusp commissure; LCC, left coronary cusp; AMC, aortomitral continuity; TV, tricuspid annulus; MV, mitral annulus; APM, anterior PAP; PPM, posterior PAP; LPF, left posterior fascicle; LAF, left anterior fascicle; GCV, greater cardiac vein; AIV, anterior inter-ventricular vein).

FIGURE 6 Catheter Ablation of ILVT



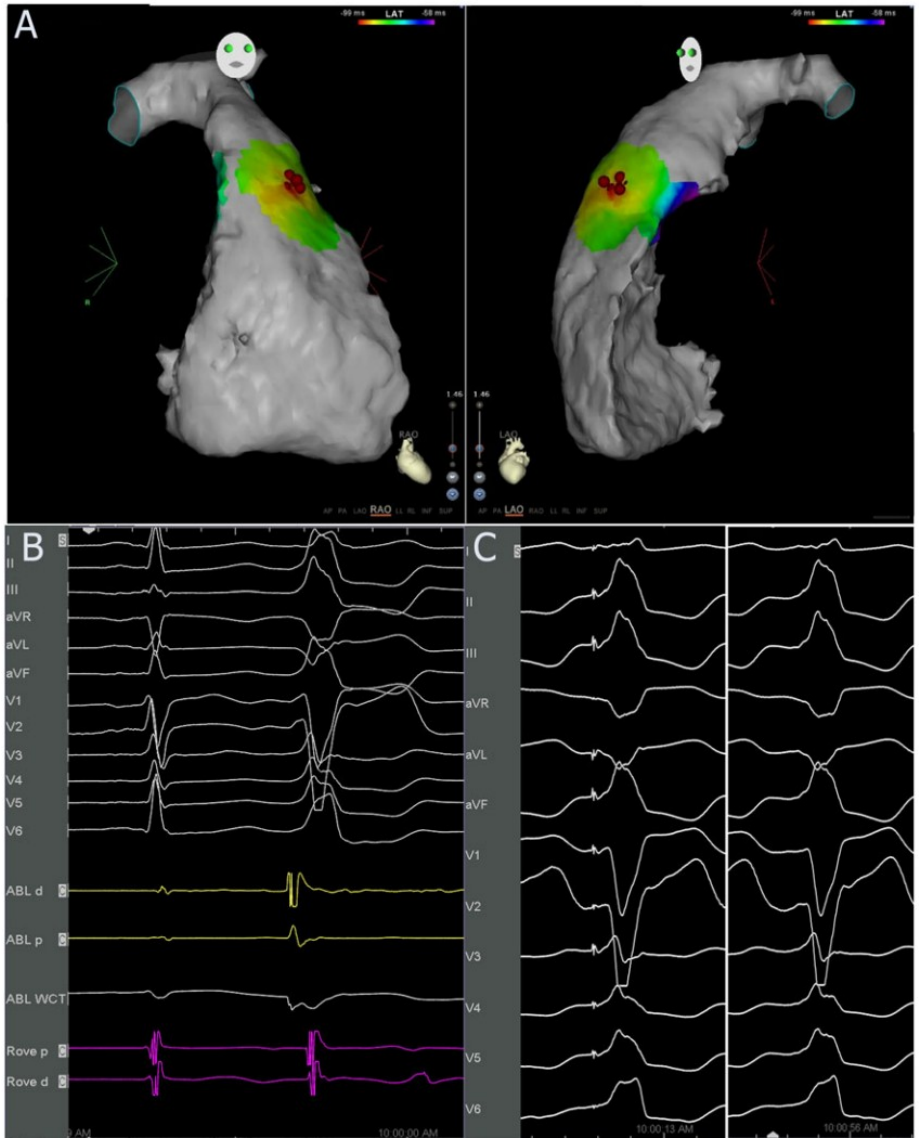

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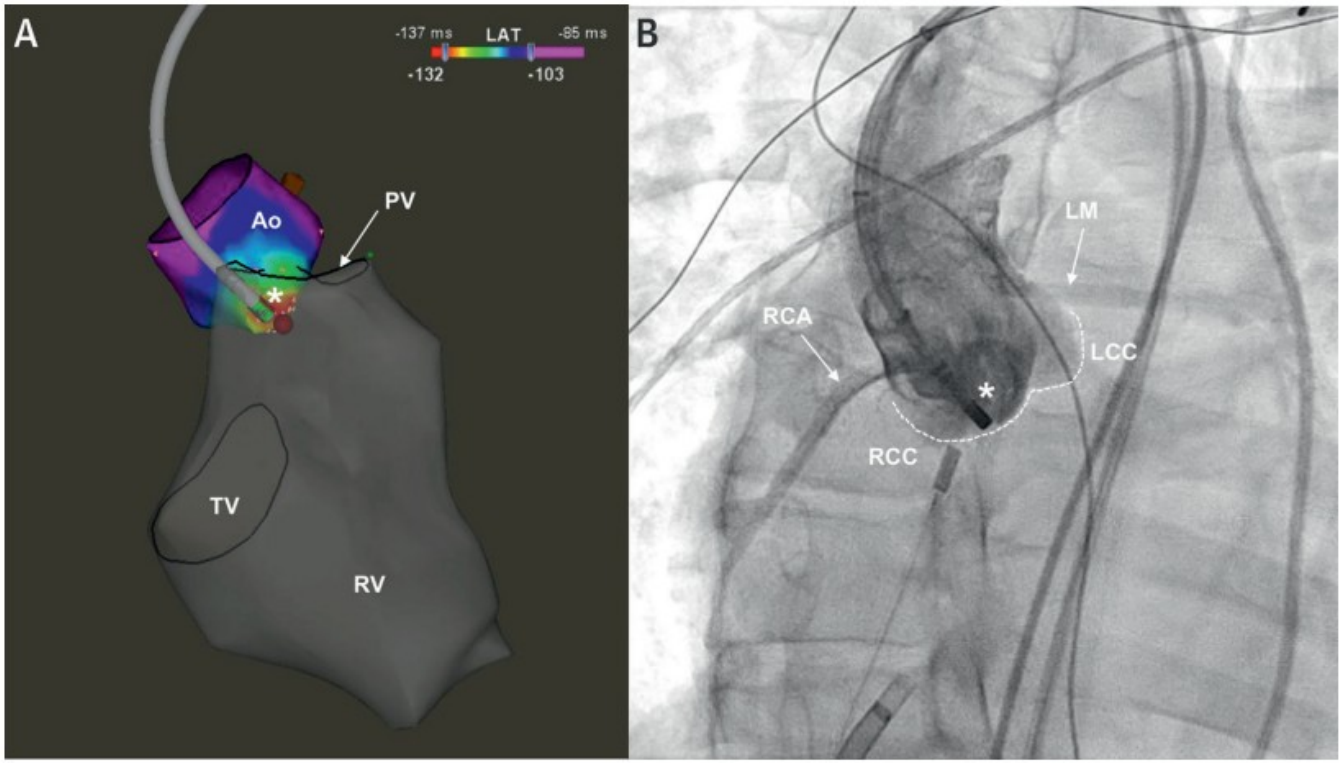
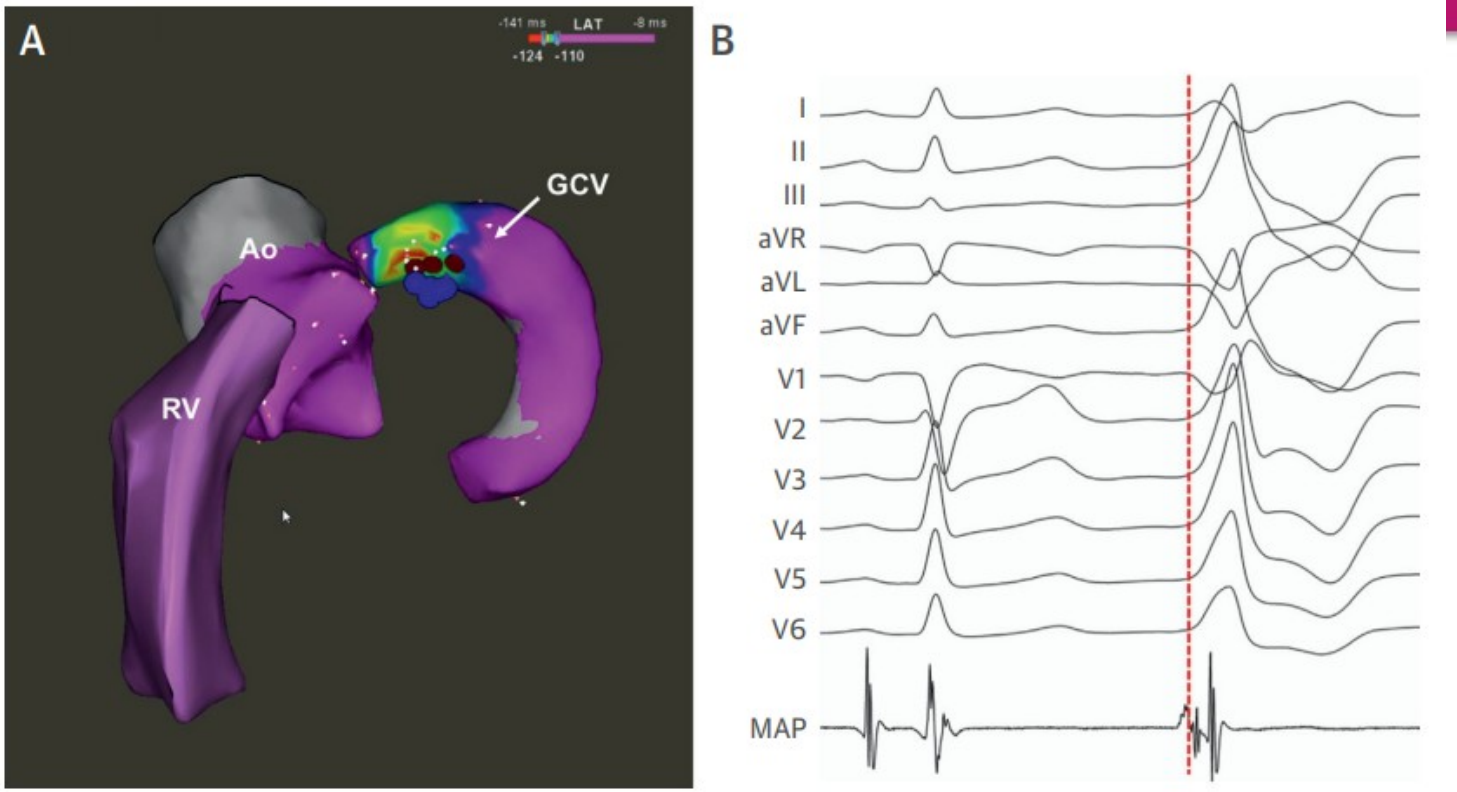
FIGURE 2 Catheter Ablation of Outflow Tract PVCs From the Aorta



(A) An activation map (anterior posterior view) of the Ao and RV during PVCs demonstrates the earliest site of activation **(red area)** to be in the RCC. The ablation catheter **(asterisk)** is positioned at the site of successful ablation **(red point)**. **(B)** An aortogram (anterior posterior view) was performed before ablation to delineate the locations of the RCA and LM relative to the putative ablation site. The ablation catheter **(asterisk)** can be seen in the RCC and is positioned at the site of earliest PVC activation. Ao = aorta; LCC = left coronary cusp; LM = left main artery; PV = pulmonary valve; PVC = premature ventricular contraction; RCA = right coronary artery; RCC = right coronary cusp; RV = right ventricle; TV = tricuspid valve.

FIGURE 3 Catheter Ablation of Outflow Tract PVCs

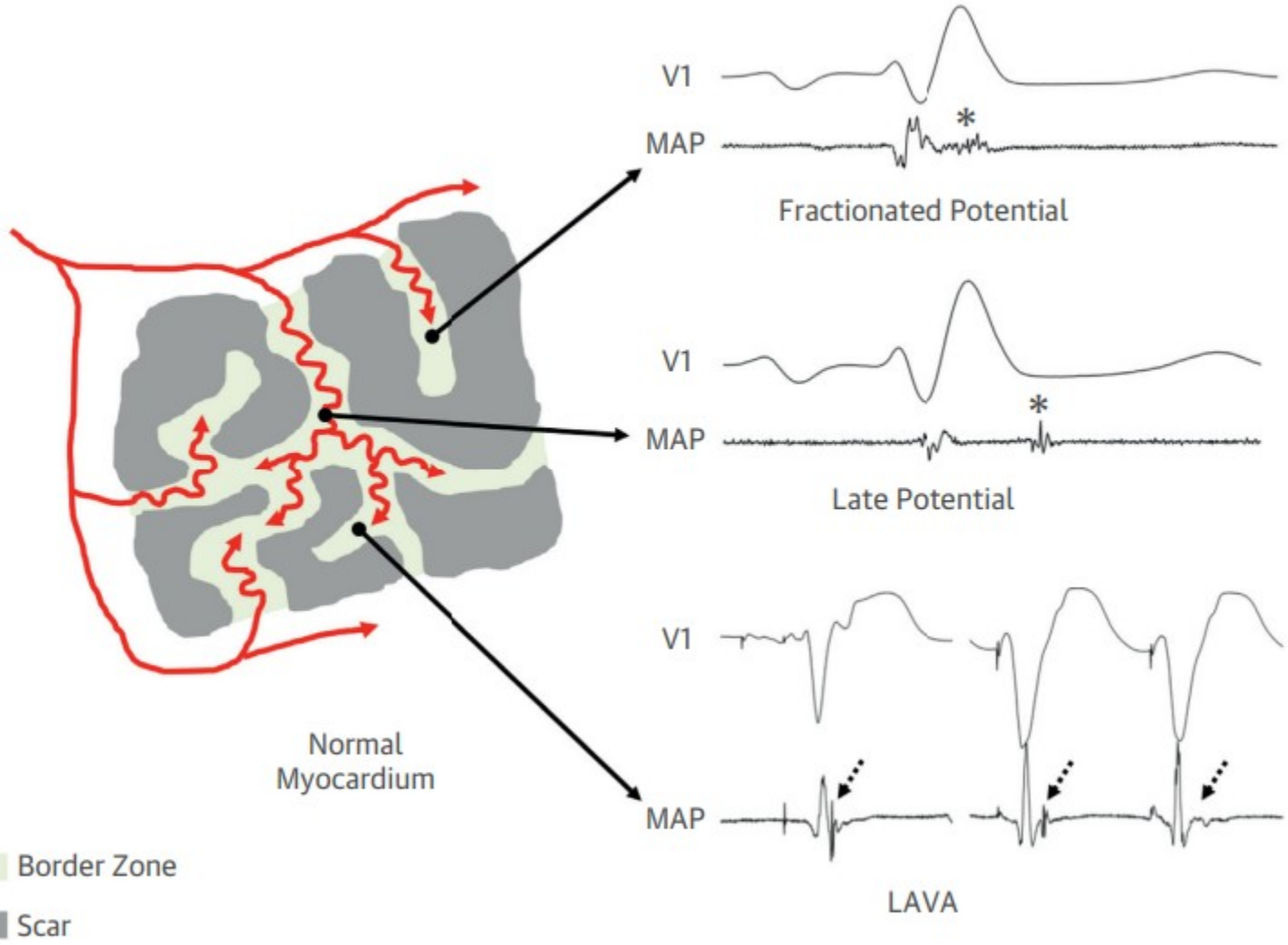


(A) Activation maps of the RV, Ao, GCV, and LV (not shown) were performed during PVCs. The area of earliest activation **(red)** was identified in the distal GCV, and ablation in this location **(red points)** only temporarily suppressed PVCs. Although activation was slightly later at the LV aortomitral continuity, prolonged ablation at this location **(blue points)** abolished the PVCs. Coronary angiography was performed before ablation in the distal GCV to exclude proximity to the coronary arteries. **(B)** The PVC morphology is of right bundle branch block morphology with an inferior axis (positive in leads II, III, and aVF) and is consistent with an outflow tract morphology. The qR pattern in lead V_1 is consistent with an aortomitral continuity location for the PVC. The MAP at the site of earliest activation in the distal GCV identified a local activation time that preceded the onset of the PVC **(orange line)** by 28 ms. MAP = mapping catheter; other abbreviations as in Figures 1 and 2.

Table 9 **Summary of the recommendations for the treatment of patients with frequent idiopathic premature ventricular complexes/ventricular tachycardia or premature ventricular complex-induced cardiomyopathy**

	Ablation	Beta-blocker	CCB	Flecainide	Amiodarone
RVOT/fascicular PVC/VT: Symptomatic, normal LV function	Class I	Class IIa	Class IIa	Class IIa	Class III
PVC/VT other than RVOT/fascicular: Symptomatic, normal LV function	Class IIa	Class I	Class I	Class IIa	Class III
RVOT/fascicular PVC/VT: LV dysfunction	Class I	Class IIa	Class III[a]	Class IIa[b]	Class IIa
PVC/VT other than RVOT/fascicular: LV dysfunction	Class I	Class IIa	Class III[a]	Class IIa[b]	Class IIa
PVC: Burden >20%, asymptomatic, normal LV function	Class IIb				Class III

FIGURE 1 Myocardial Scar and Substrate for Re-Entrant VT



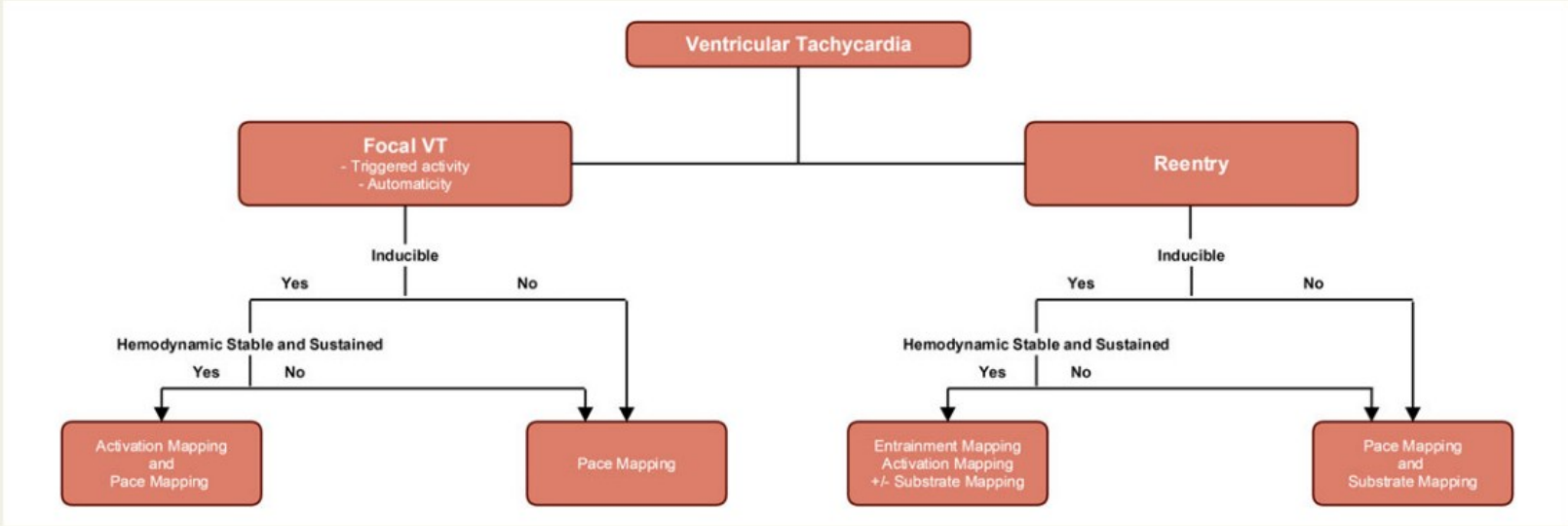
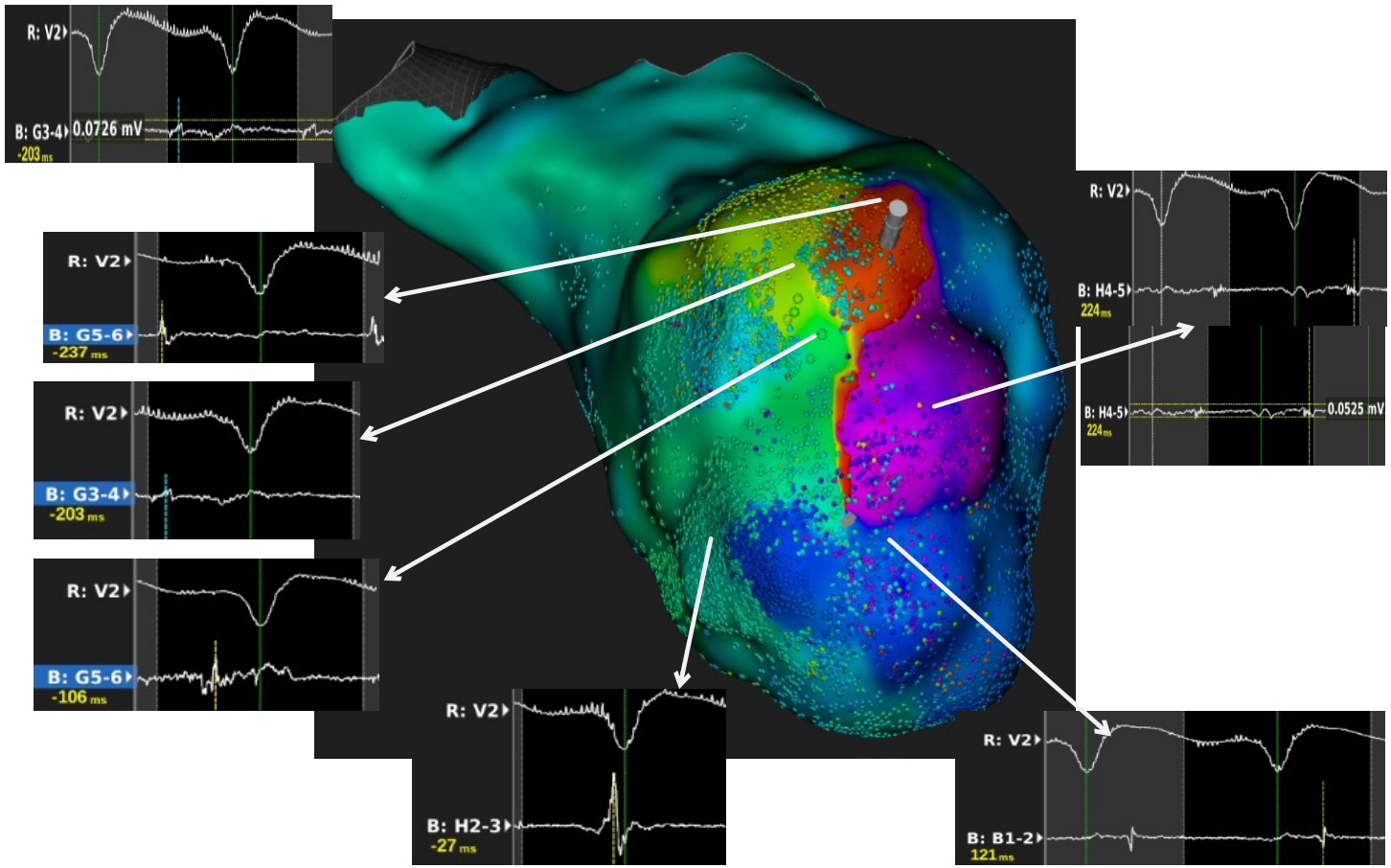
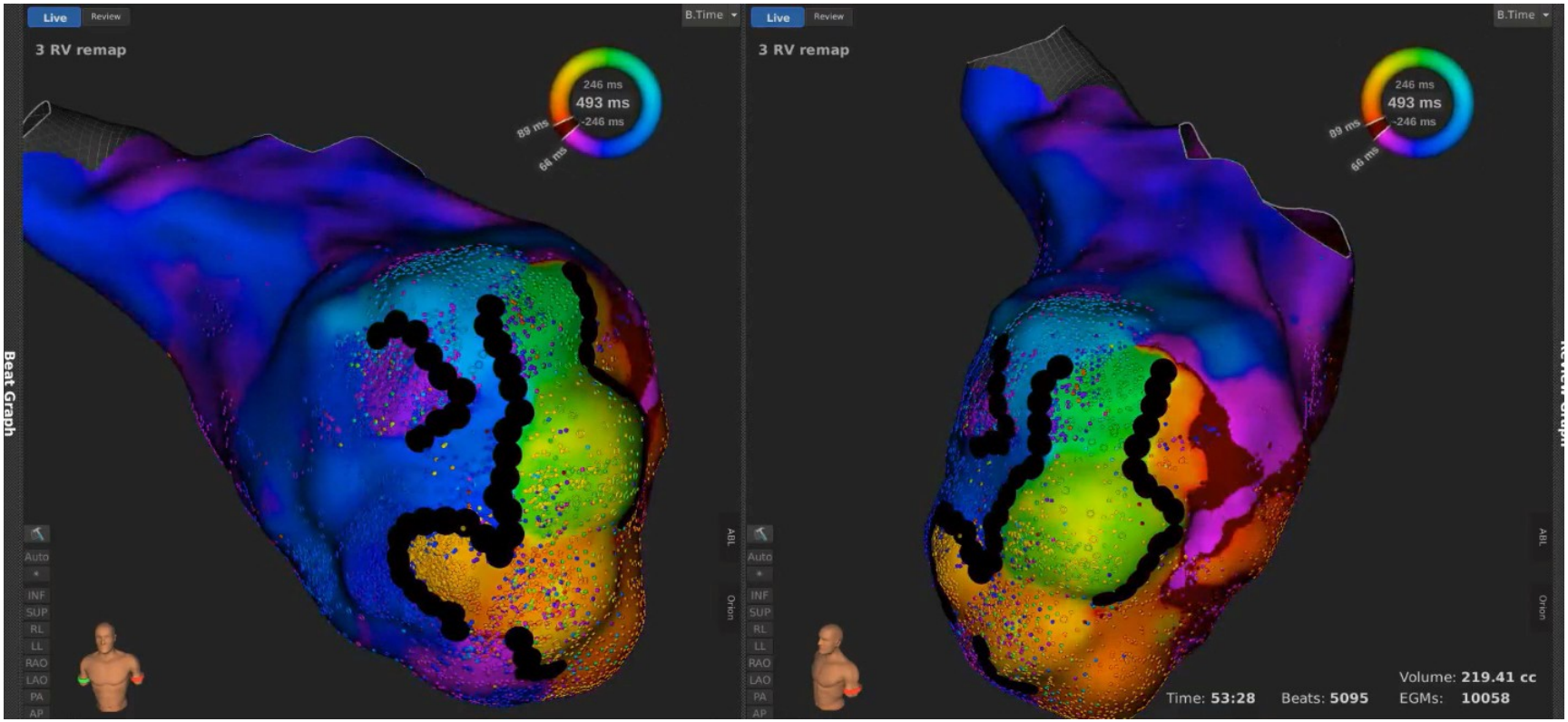


Figure 4 The algorithm shows mapping strategies based on haemodynamic stability during tachycardia, stability of VT reentry circuit (stable morphology and cycle length), and inducibility during procedure (VT = VT).

Potenziali in tachicardia



CIRCUITO IN TV CON ALTA RISOLUZIONE



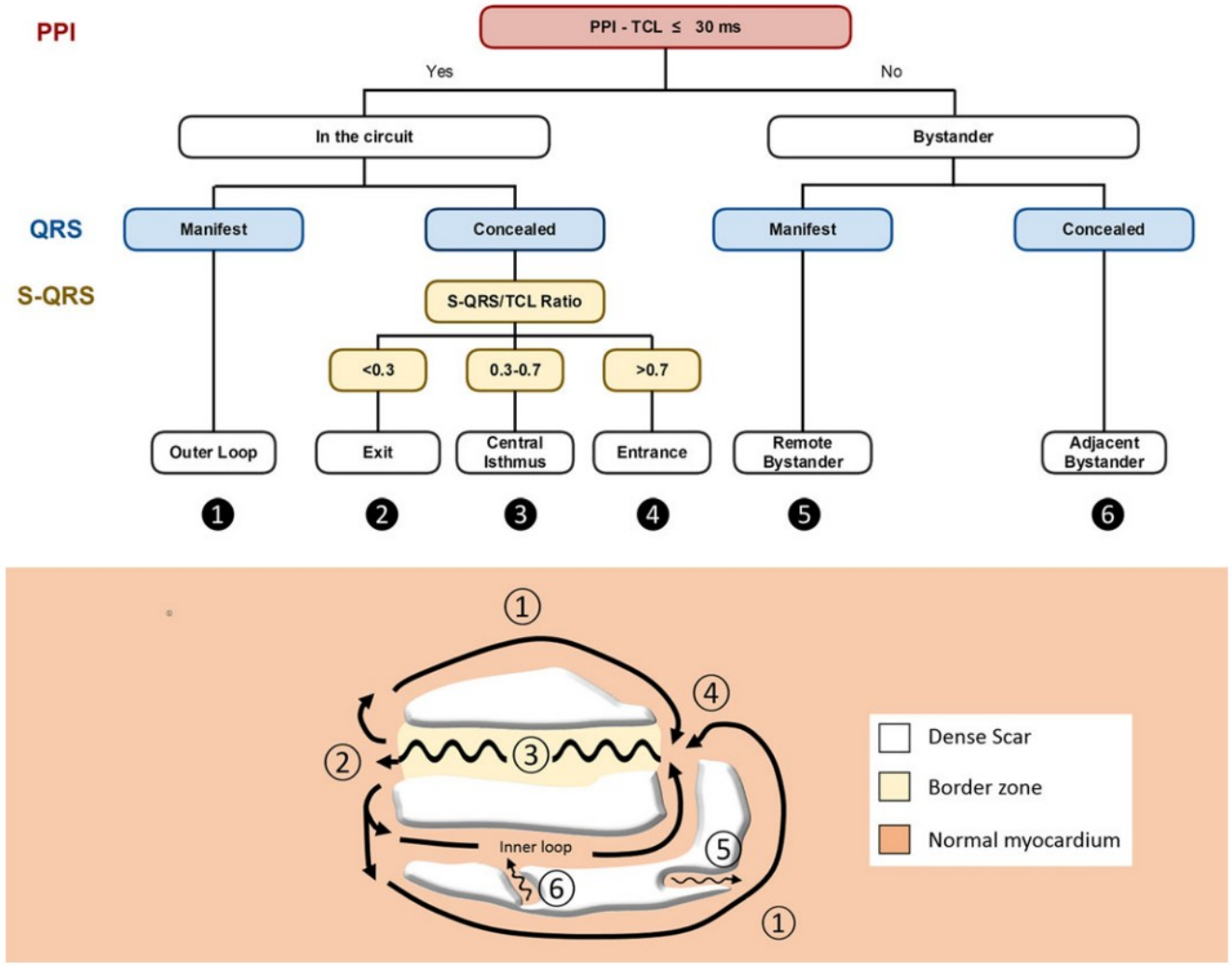


Figure 3 A schematic drawing of the components of the ventricular tachycardia circuit. The main components include entrance, central isthmus, and exit sites. Characteristics of entrainment mapping according to the site of pacing are summarized in the algorithm (PPI, post-pacing interval; TCL, tachycardia cycle length; S-QRS, stimulus to QRS interval).

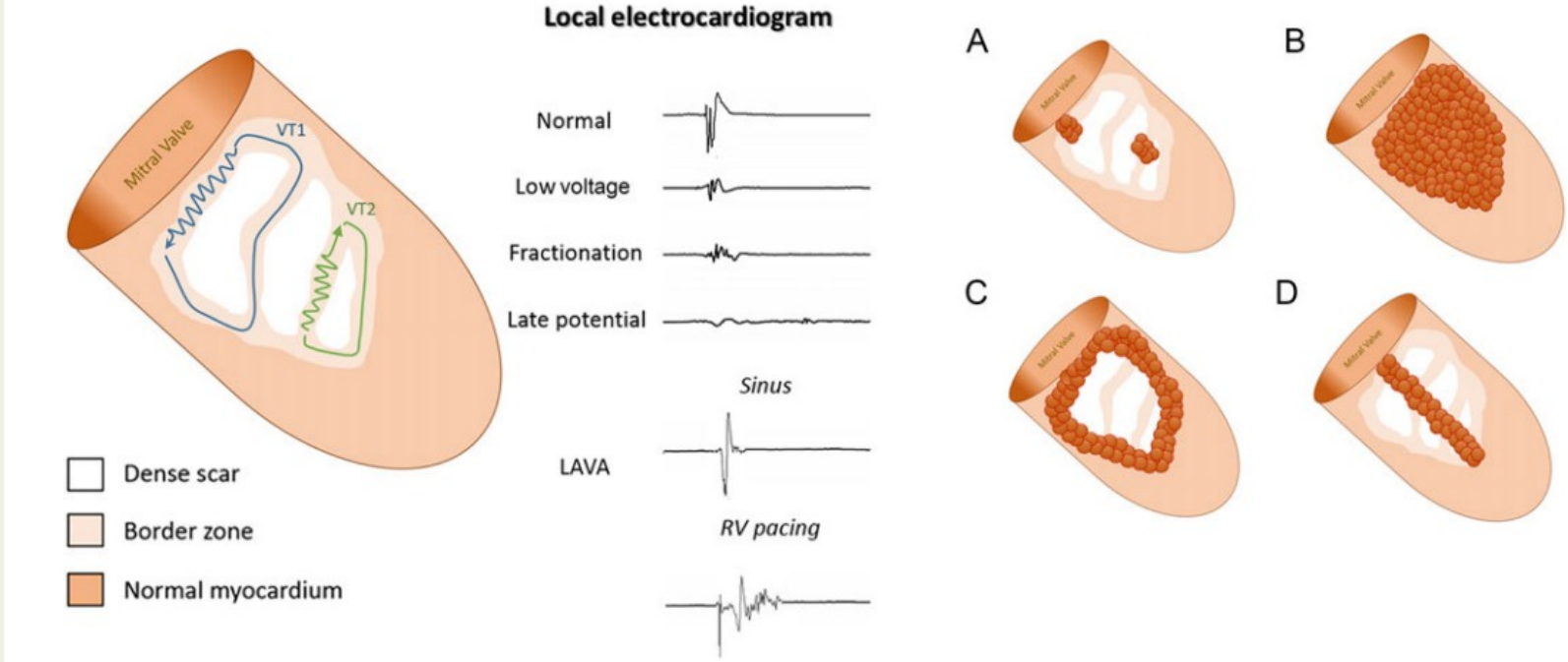
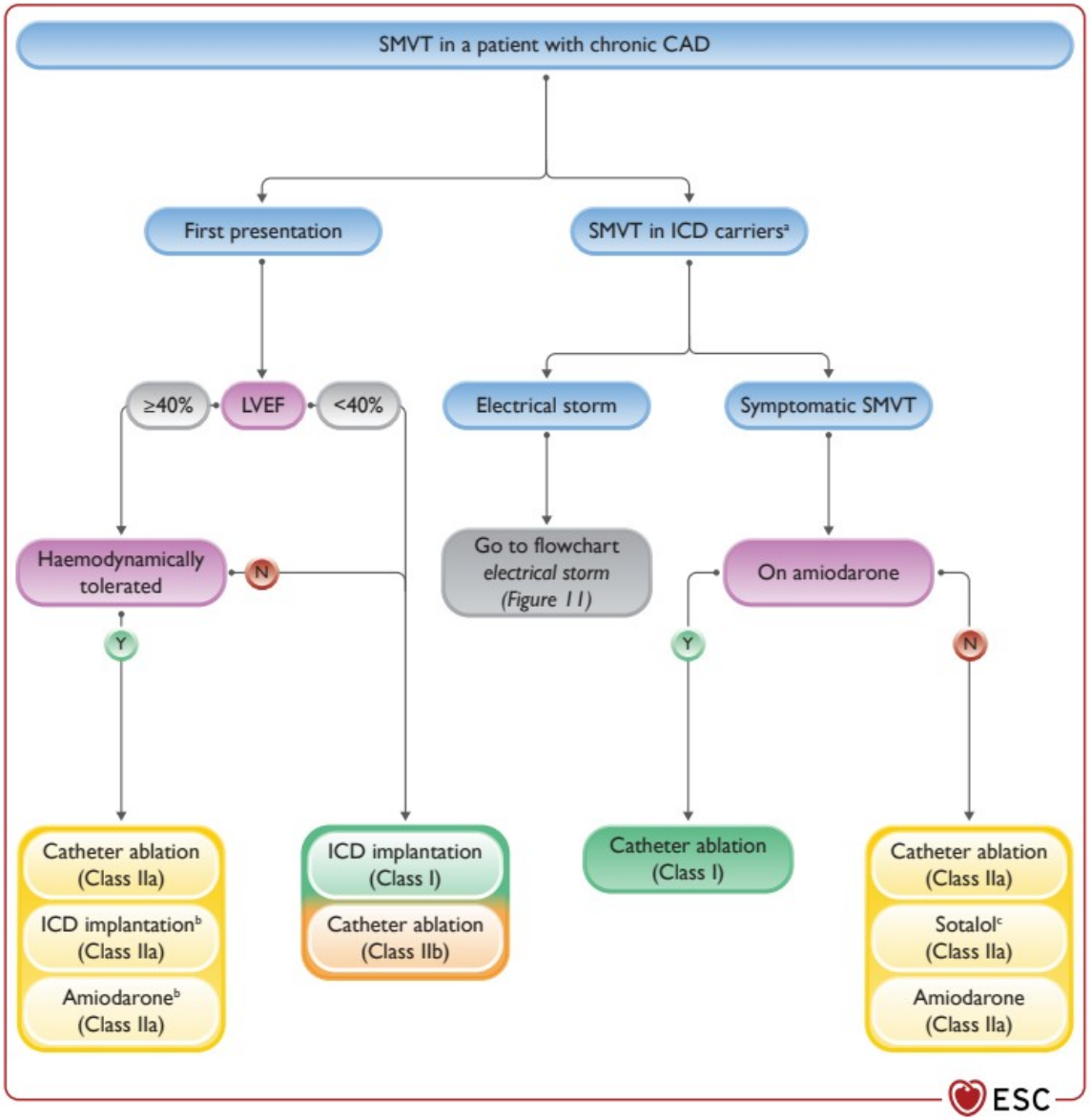


Figure 5 Different ablation strategies and abnormal electrograms are shown. These strategies can be used together. (*A*) Isthmus ablation. (*B*) Scar homogenization. (*C*) Ablation lines applied parallel to the scar border within the low-voltage area encompassing the exit region. (*D*) Ablation lines placed perpendicular to all defined isthmuses between islands of unexcitable segment or extend perpendicular to the scar border from the area of dense scar, across the border zone and connecting out to normal myocardium or anatomical barrier (e.g. mitral valve).


SMVT in a patient with chronic CAD
First presentation
SMVT in ICD carriers[a]
≥40%
LVEF
<40%
Electrical storm
Symptomatic SMVT
Haemodynamically tolerated
N
Y
Go to flowchart electrical storm (Figure 11)
On amiodarone
Y
N
Catheter ablation (Class IIa)
ICD implantation[b] (Class IIa)
Amiodarone[b] (Class IIa)
ICD implantation (Class I)
Catheter ablation (Class IIb)
Catheter ablation (Class I)
Catheter ablation (Class IIa)
Sotalol[c] (Class IIa)
Amiodarone (Class IIa)
ESC

Early Versus Late Referral for Catheter Ablation of Ventricular Tachycardia in Patients With Structural Heart Disease

A Systematic Review and Meta-Analysis of Clinical Outcomes

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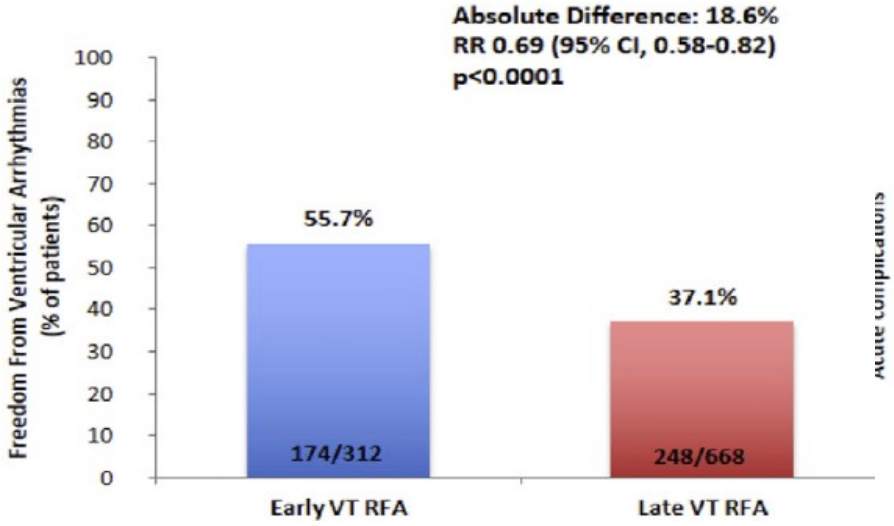
Circulation

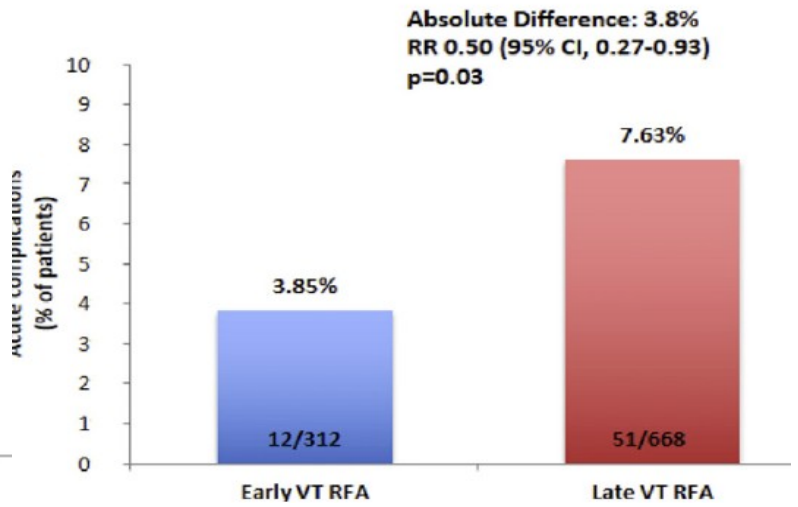
PERSPECTIVE

Arvind N. Kanagasundram, MD
Roy M. John, MBBS, PhD
William G. Stevenson MD

Ventricular Tachycardia Ablation in Patients With Implantable Cardioverter Defibrillators Should No Longer Be a Therapy of Last Resort

METAANALISI 980 PAZIENTI





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