Azərbaycan Kardiologiya Cəmiyyəti

ESC European Society of Cardiology

VT epizodu olan AFaÜÇ xəstəsinin farmakolojik təqibi

Dr. Ceyhun Umudov

AFaÜÇ (HFrEF)

Type of HF		HFrEF	HFmrEF	HFpEF
CRITERIA	1	Symptoms ± Signs[a]	Symptoms ± Signs[a]	Symptoms ± Signs[a]
	2	LVEF ≤40%	LVEF 41–49%[b]	LVEF ≥50%
	3	–	–	Objective evidence of cardiac structural and/or functional abnormalities consistent with the presence of LV diastolic

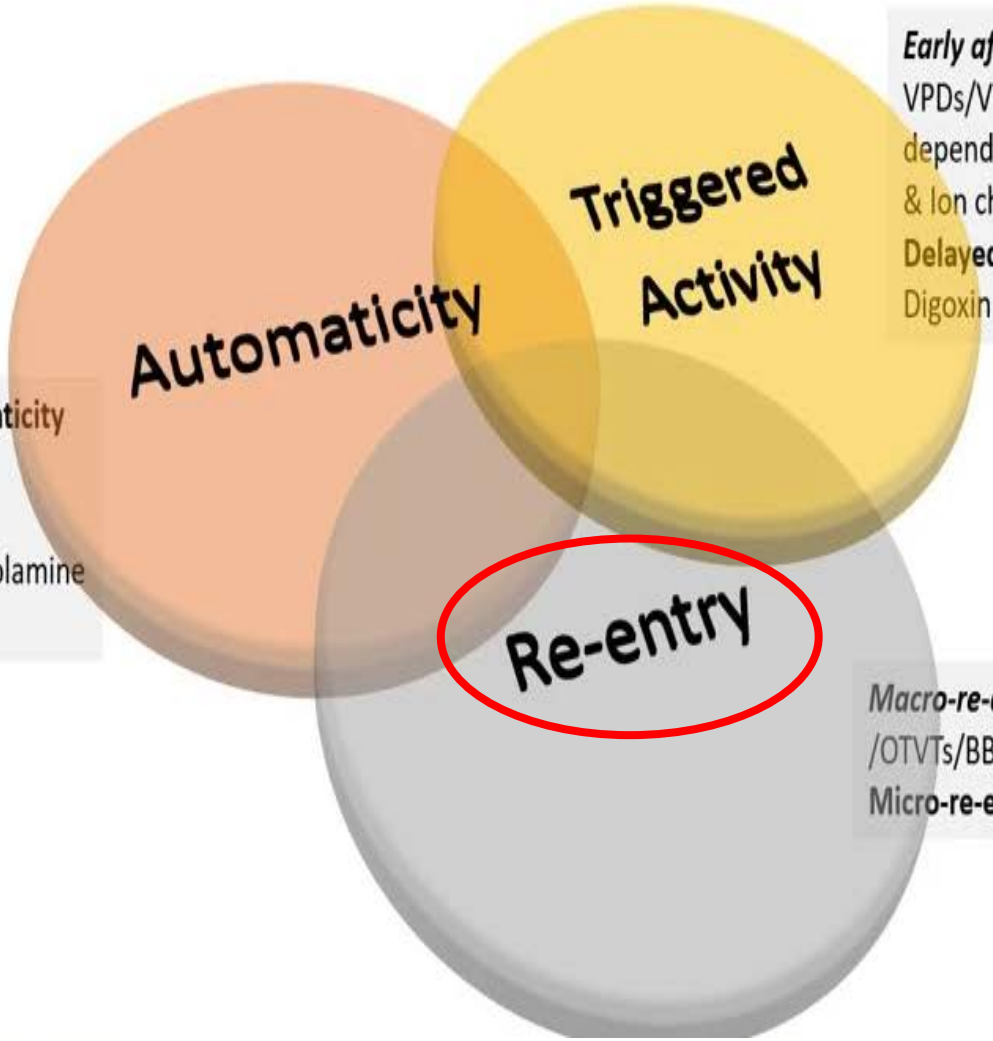
C 2021

Before disease-modifying therapies became available, the incidence of SCD in patients with HFrEF was higher than 20% per year [10], nevertheless with current pharmacologic and electric therapy, the incidence of SCD has decreased to about 3% per year [11].

Currently, SCD accounts for about 40% to 45% of all deaths in HFrEF patients, and the proportion of SCD is higher in patients with milder symptoms (New York Heart Association (NYHA) class II-III) [12], indeed two-thirds of patients with NYHA functional class II, experience SCD, compared with only one-third of those with NYHA functional class IV symptoms, who died preponderantly for advanced HF [13].

Reduced exercise tolerance Fatigue, tiredness, increased time to recover after exercise Ankle swelling	Laterally displaced apical impulse		Tachypnoea Cheyne-Stokes respiration Hepatomegaly Ascites Cold extremities Oliguria Narrow pulse pressure

VT patofiziologiyası:

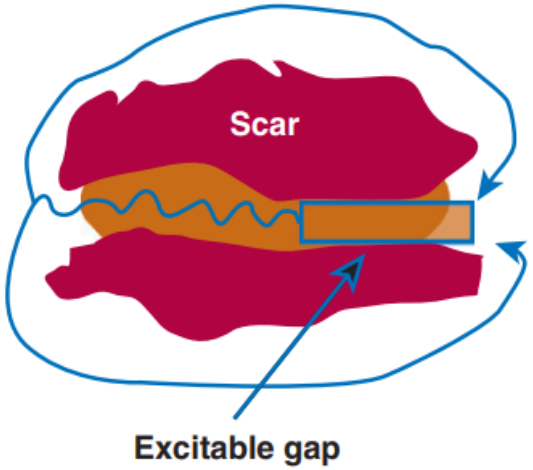


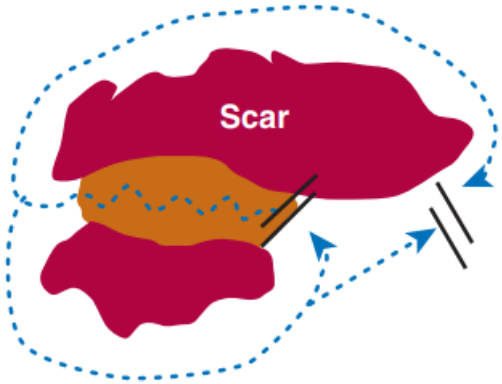
Early after depolarisation(EAD)*
VPDs/VT due to brady dependent/Hypoxia/Acidosis/hypokalemia/ & Ion channel defect
Delayed after depolarisation(DAD)
Digoxin related

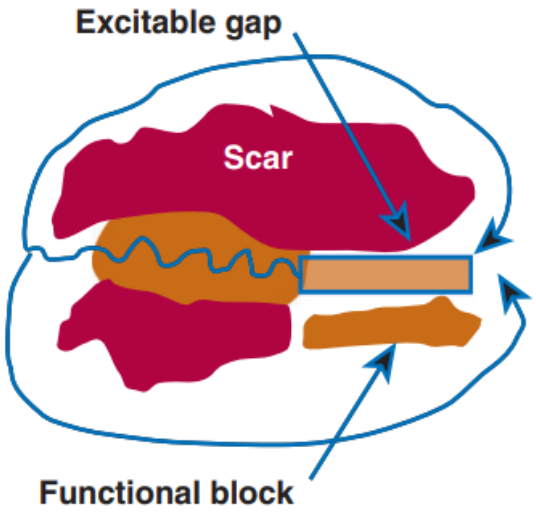
1.**Enhanced normal automaticity**
(Sinus tachycardia)
2.***Abnormal automaticity****
(Most Ischemic VT , Catecholamine sensitive)

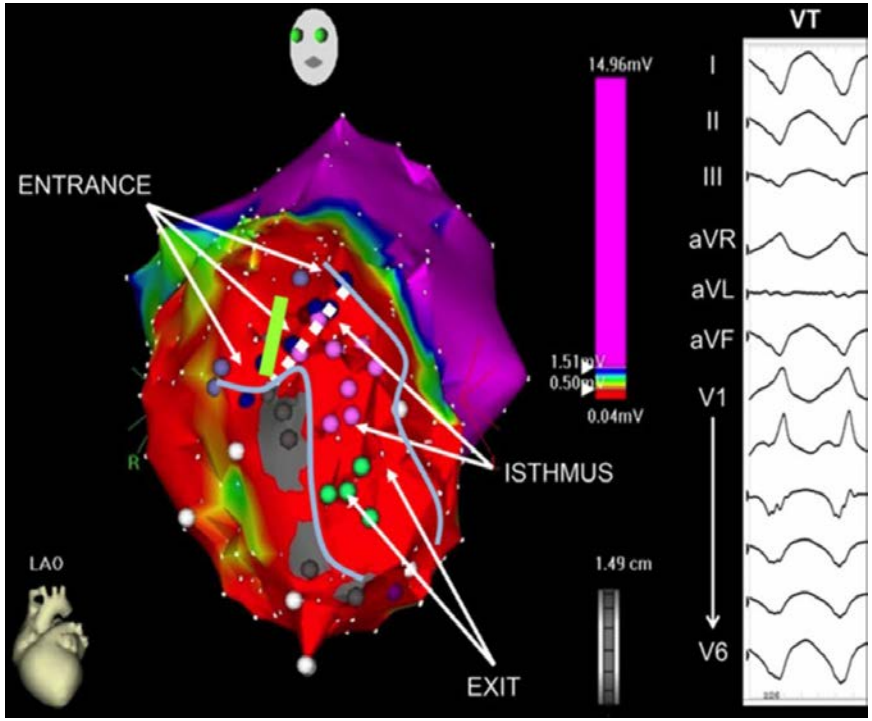
Macro-re-entry* (Commonest) Scar mediated VT /OTVTs/BBR/Fasc.VT/Atrial flutters)
Micro-re-entry (Ischemic /Brugada)

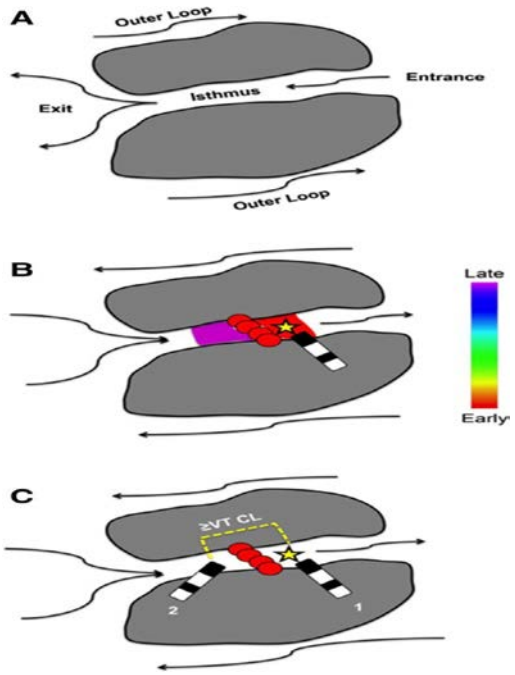
Re-entry:


Scar
Excitable gap

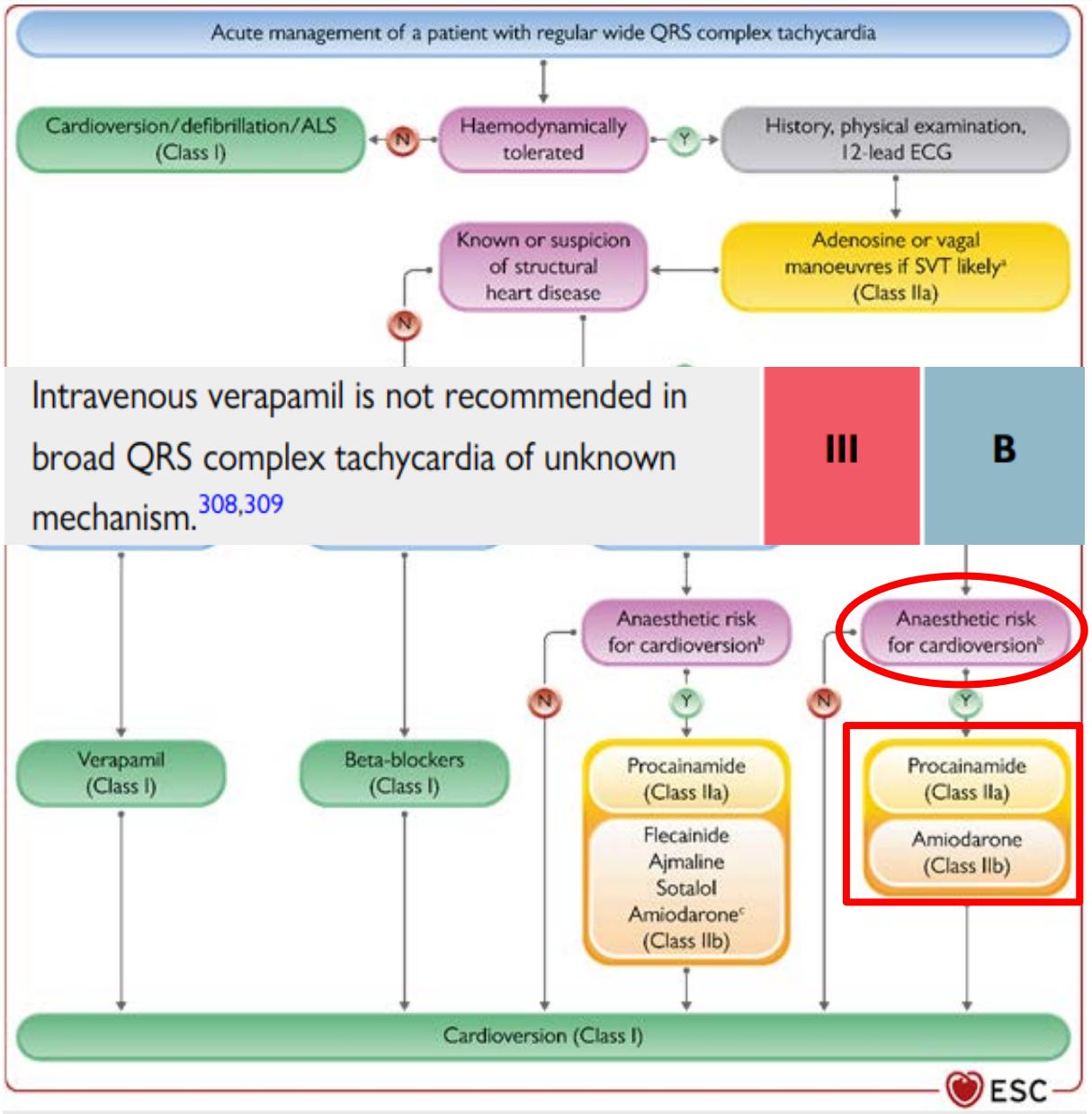

Scar


Excitable gap
Scar
Functional block


VT
14.96mV
1.51mV
0.50mV
0.04mV
ENTRANCE
ISTHMUS
EXIT
LA0
1.49 cm
I
II
III
aVR
aVL
aVF
V1
V6


A
Outer Loop
Entrance
Exit
Isthmus
Outer Loop
B
Late
Early
C
≥VT CL
2
1

VT kəskin dövr müalicəsi:



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European Heart Journal
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CLINICAL RESEARCH
Arrhythmia/electrophysiology

Randomized comparison of intravenous procainamide vs. intravenous amiodarone for the acute treatment of tolerated wide QRS tachycardia: the PROCAMIO study

Mercedes Ortiz[1†], Alfonso Martín[2], Fernando Arribas[3], Blanca Coll-Vinent[4], Carmen del Arco[5], Rafael Peinado[6] and Jesús Almendral[1*†], on Behalf of the PROCAMIO Study Investigators

In this randomized prospective study comparing intravenous procainamide and amiodarone for the treatment of the acute episode of sustained monomorphic well-tolerated wide QRS tachycardia (probably VT), procainamide therapy was associated with less major cardiac adverse events and a higher proportion of tachycardia termination within 40 min.

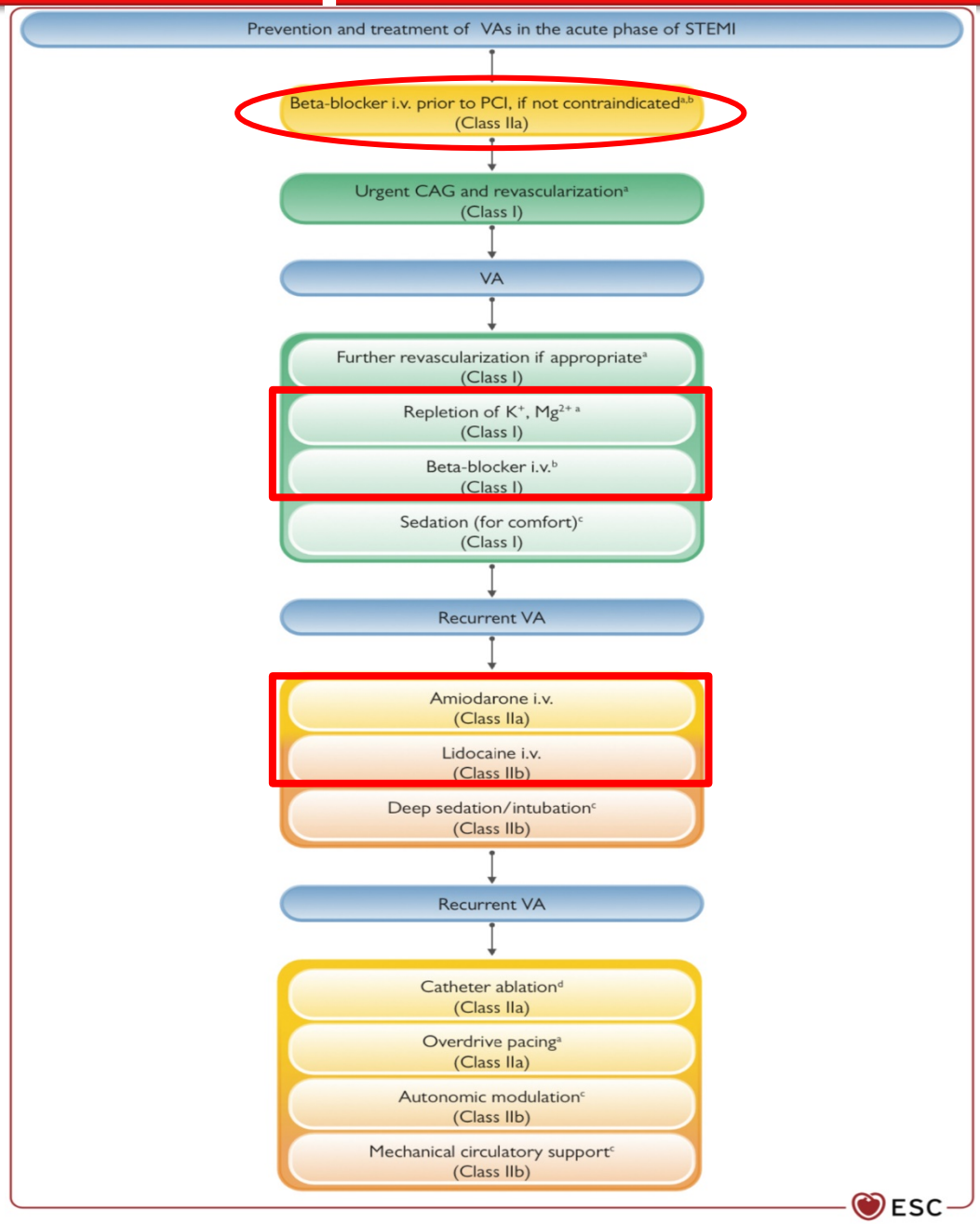
VT uzun dövr dərman müaliçəsi:

Cause	Examples of presentations	Specific investigations
CAD	Myocardial infarction Angina or "angina-equivalent" Arrhythmias	Invasive coronary angiography CT coronary angiography Imaging stress tests (echo, nuclear, CMR)
Hypertension	Heart failure with preserved systolic function Malignant hypertension/acute pulmonary oedema	24 h ambulatory BP Plasma metanephrines, renal artery imaging Renin and aldosterone
Valve disease	Primary valve disease e.g., aortic stenosis	Echo — transoesophageal/stress

In patients with heart failure with reduced ejection fraction (HfrEF), the 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure recommend angiotensin-converting enzyme inhibitor (ACE-I)/angiotensin receptor blocker (ARB)/angiotensin receptor neprilysin inhibitors (ARNIs), mineralo-corticoid receptor antagonists (MRAs), beta-blockers, and sodium–glucose co-transporter 2 (SGLT2) inhibitors to reduce mortality due to heart failure and SCD.

	Proteasome inhibitors RAF+MEK inhibitors	
Infiltrative	Amyloid Sarcoidosis Neoplastic	Serum electrophoresis and serum free light chains, Bence Jones protein, bone scintigraphy, CMR, CT-PET, EMB Serum ACE, CMR, FDG-PET, chest CT, EMB CMR, EMB
Storage disorders	Haemochromatosis Fabry disease Glycogen storage diseases	Iron studies, genetics, CMR (T2* imaging), EMB α-galactosidase A, genetics, CMR (T1 mapping)

KKS-da VT epizodunun müalicəsi:



European Heart Journal
Acute Cardiovascular Care
ESC European Society of Cardiology

Original scientific paper

vascular Care
2019

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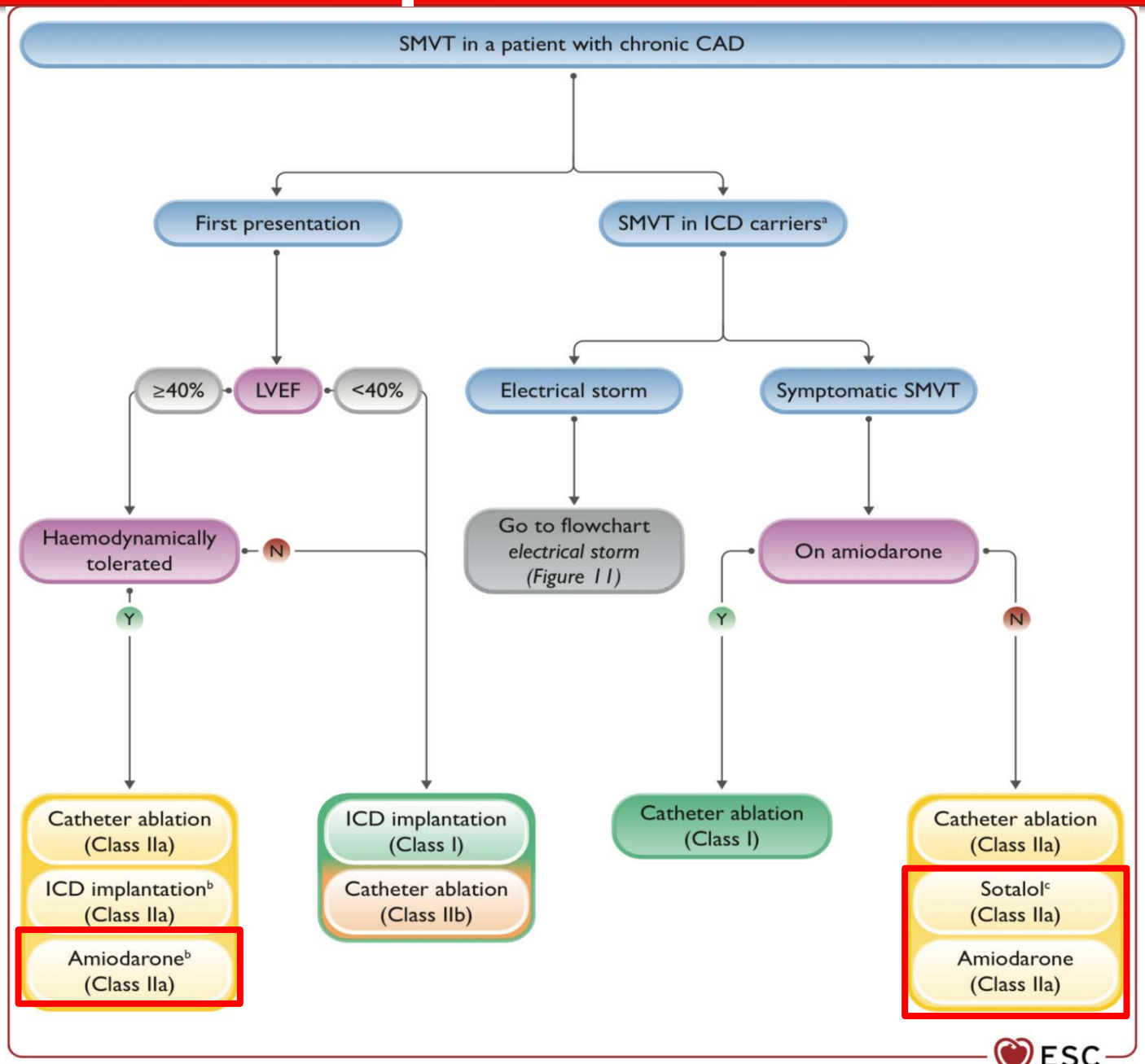
Intravenous beta-blocker treatment is indicated for patients with recurrent PVT/VF during STEMI unless contraindicated.[551,552]	I	B
Intravenous amiodarone treatment should be considered for patients with recurrent PVT/VF during the acute phase of ACS.[552,554,555]	IIa	C
Intravenous lidocaine may be considered for the treatment of recurrent PVT/VF not responding to beta-blockers or amiodarone, or if amiodarone is contraindicated during the acute phase of ACS.[554]	IIb	C

Conclusions: In a nonrestricted STEMI population, early intravenous metoprolol before PPCI was not associated with a reduction in infarct size. Metoprolol reduced the incidence of malignant arrhythmias in the acute phase and was not associated with an increase in adverse events.

Prophylactic treatment with AADs (other than beta-blockers) is not recommended in ACS.[322]	III	B

KAX-də VT epizodunun müalicəsi:



Comparison of β-Blockers, Amiodarone Plus β-Blockers, or Sotalol for Prevention of Shocks From Implantable Cardioverter Defibrillators

The OPTIC Study: A Randomized Trial

Results Shocks occurred in 41 patients (38.5%) assigned to β-blocker alone, 26 (24.3%) assigned to sotalol, and 12 (10.3%) assigned to amiodarone plus β-blocker. A reduc-
tio ker or
so 0.28-
0. com-
pa l (HR,
0. com-
pa study
dr 5.3%
for β-blocker alone. Adverse pulmonary and thyroid events and symptomatic brady-
cardia were more common among patients randomized to amiodarone.

The addition of oral amiodarone or beta-blocker replacement by sotalol should be considered in patients with CAD with recurrent, symptomatic SMVT, or ICD shocks for SMVT while on beta-blocker treatment.[318,581]

IIa | B

Conclusions Despite use of advanced ICD technology and treatment with a β-blocker, shocks occur commonly in the first year after ICD implant. Amiodarone plus β-blocker is effective for preventing these shocks and is more effective than sotalol but has an increased risk of drug-related adverse effects.

İdiopatik PVc/VT induce KMP:

Relationship between burden of premature ventricular complexes and left ventricular function

Timir S. Baman, MD,* Dave C. Lange, MD,* Karl J. Ilg, MD,* Sanjaya K. Gupta, MD,* Tzu-Yu Liu, MS,†
Craig Alguire, MD,* William Armstrong, MD, FACC,* Eric Good, DO, FACC,* Aman Chugh, MD, FACC,*
Krit Jongnarangsin, MD,* Frank Pelosi, Jr., MD,* Thomas Crawford, MD,* Matthew Ebinger, MD, DO,*

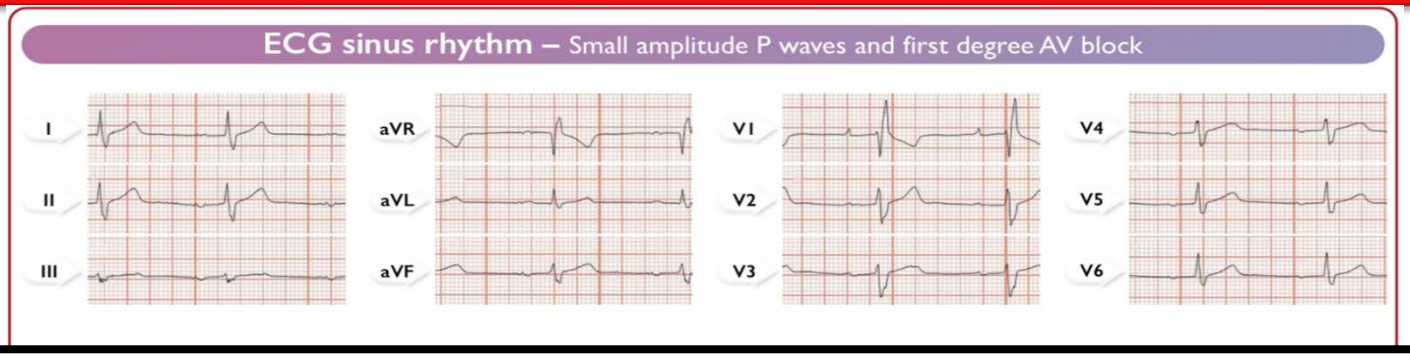
A PVC burden of at least 10% appears to be the minimal threshold for development of PVC-induced cardiomyopathy, and the risk increases further with a PVC burden >20%. In patients with a PVC burden <10%, other cardiomyopathy aetiologies should be suspected and further diagnostic work-up undertaken.

Aleksandr Voskoboinik, MBBS, PhD,*[1] Alexios Hadjis, MD, FHRS,*[1]
Christina Alhede, MD,* Sung Il Im, MD, PhD,*† Hansu Park, MD,†
Joshua Moss, MD, FHRS,* Gregory M. Marcus, MD, FHRS,* Henry Hsia, MD, FHRS,*
Byron Lee, MD,* Zian Tseng, MD,* Randall Lee, MD, PhD,* Melvin Scheinman, MD,*
Vasanth Vedantham, MD,* Eric Vittinghoff, PhD,* Kyoung-Min Park, MD, PhD,‡
Edward P. Gerstenfeld, MD, FHRS*

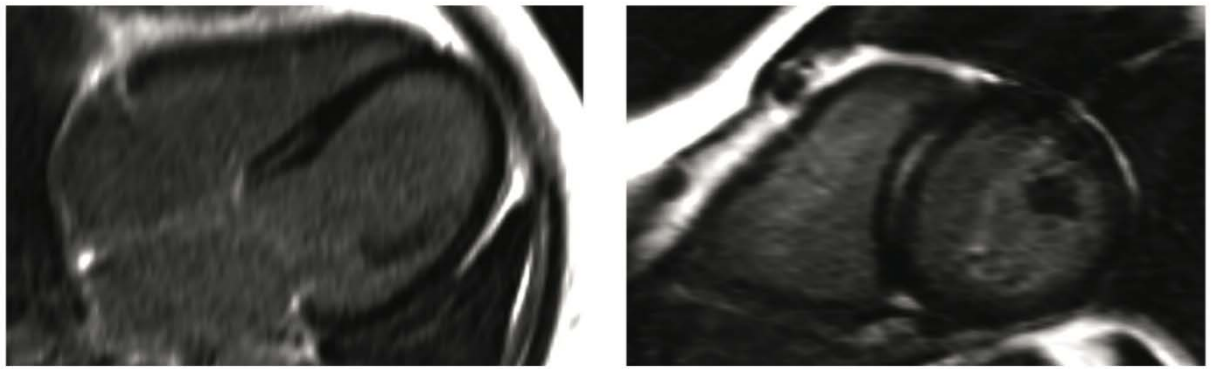
İdiopatik PVc/VT induce KMP:

	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

DKMP VT epizodunun müalicəsi :



Positive genetic testing for *LMNA* mutations has crucial clinical and prognostic implications. Mortality in patients with LMNA-CMP is estimated to be 40% at 5 years (Pasotti et al., 2008), whereas 45% suffered SCD or aborted SCD.



4-chamber

Short axis

ESC

Circulation
Volume 140, Issue 23, 3 December 2019; Pages e816-e817
https://doi.org/10.1161/CIRCULATIONAHA.119.042425

CORRESPONDENCE

Letter by Aimo et al Regarding Article, "Development and Validation of a New Risk Prediction Score for Life-Threatening Ventricular Tachyarrhythmias in

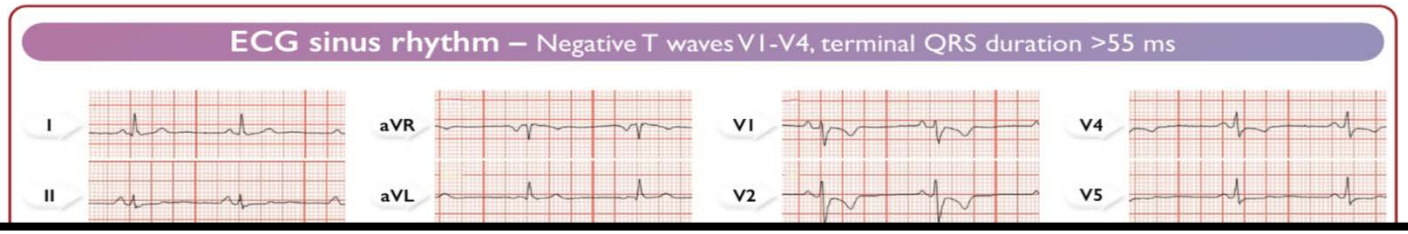
The addition of oral amiodarone or replacement of beta-blockers by sotalol should be considered in patients with DCM/HNDCM and an ICD who experience recurrent, symptomatic VA despite optimal device programming and beta-blocker treatment.[318]	IIa	B

Atrio-ventricular block	○ Absent ○ 1st degree ○ High degree	*Please select the highest degree. 1st degree AV block corresponds to ≥0.20 sec PR interval and high degree AV block to type II 2nd degree or 3rd degree (and not type I 2nd degree)*
Non-sustained ventricular tachycardia	○ Yes ○ No	*NSVT corresponds to ≥3 consecutive ventricular complexes at a rate ≥120 bpm on 24-h ambulatory electrocardiographic monitoring*
Left ventricular ejection fraction	____ %	*Left ventricular ejection fraction measurement derived from echocardiogram*

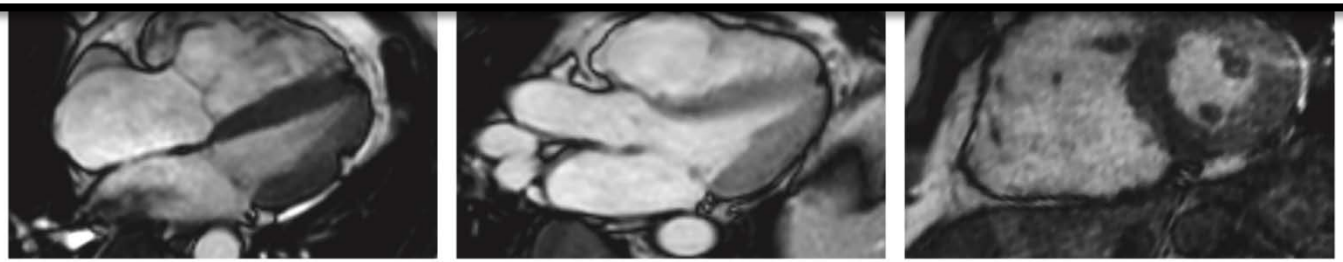
Risk of Life-Threatening Ventricular Tachyarrhythmias at 5 years

___ %

ARVD-də VT epizodunun müalicəsi:



Among definite/probable ARVC patients considered at high risk for VA, 23–48% will experience appropriate ICD intervention during a mean follow-up of 4.7 years. In 16–19% of cases, ICD intervention is triggered by fast VT ≥250 b.p.m. or VF, which is considered as surrogate for a life-threatening event. In a large cohort of 864 ARVC patients (38.8% with a prior VA), 43% had VT/VF during a median follow-up of 5.75 years, but only 10.8% a potentially life-threatening event. Thus, in 3 out of 4 ARVC patients, ICD therapy is appropriate but may not be considered acutely life-saving.



Beta-blocker therapy may be considered in all patients with a definite diagnosis of ARVC.	IIb	C

Data on AADs to prevent VT recurrence are limited to small observational studies and registries. In general, AAD therapy has limited efficacy. Although sotalol was effective to prevent inducibility of VT, it did not supress clinically relevant arrhythmias. Treatment with amiodarone or class 1 drugs was associated with a trend to lower VT recurrence as compared with sotalol. The addition of flecainide to beta-blockers/sotalol was beneficial in a small cohort.

In patients with ARVC and recurrent, symptomatic VT despite beta-blockers, AAD treatment should be considered.[709,710]	IIa	C

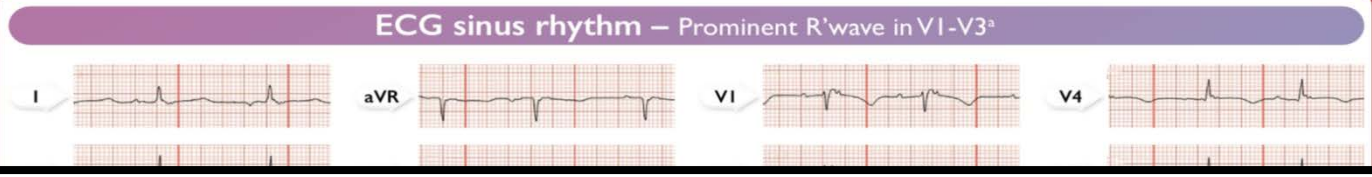
Miokarditdə VT epizodunun müalicəsi:

Sustained VAs may occur in acute myocarditis. In a large series of patients, in-hospital VF or CA was reported in 2.5% of cases. In

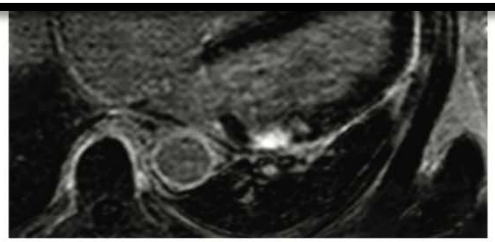
AADs should be considered (preferably amiodarone and beta-blockers) in patients with symptomatic non-sustained or sustained VAs during the acute phase of myocarditis.	**IIa**	**C**
In post-myocarditis patients with recurrent, symptomatic VT, AAD treatment should be considered.	**IIa**	**C**

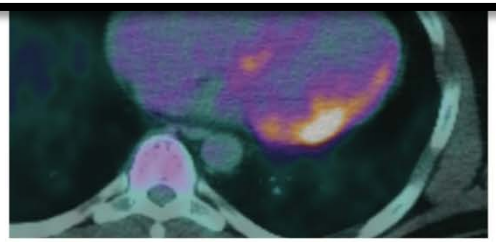
patients with sustained VAs during the acute phase of myocarditis (LVEF 53 ± 10%) had a high risk (45% at 3 years) of VT/VF recurrences during follow-up.

Kardiak Sarkoidozda VT epizodunun müalicəsi :



Unexpected SCD is an important characteristic and outcome of CS (*Take home figure*). It accounts for 14% of the presenting manifestations and as many as 80% of all fatalities in CS. Furthermore, nearly two-thirds of all deaths caused by CS occur suddenly in individuals with undiagnosed sarcoid granulomas in the heart. Of patients in whom CS causes symptoms during life, 85% can be expected to live beyond 5 years and 76% beyond 10 years from symptom onset. For patients receiving immunosuppressive and device therapy, the 5- and 10-year survival estimates are 93% and 87%, respectively.





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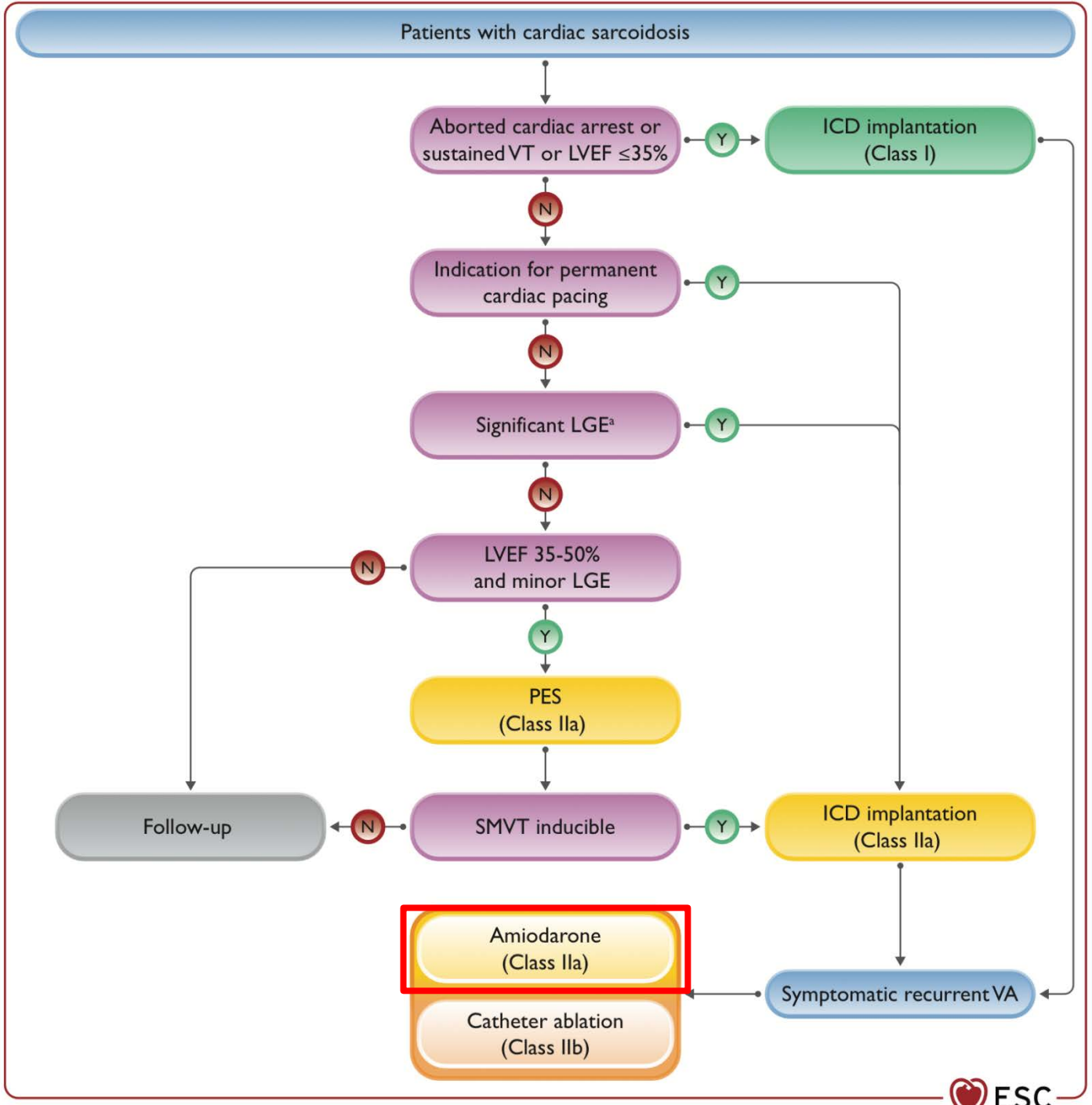

Patients with cardiac sarcoidosis
Aborted cardiac arrest or sustained VT or LVEF ≤35%
Y
ICD implantation (Class I)
N
Indication for permanent cardiac pacing
Y
N
Significant LGE[a]
Y
N
LVEF 35-50% and minor LGE
N
Y
PES (Class IIa)
Follow-up
N
SMVT inducible
Y
ICD implantation (Class IIa)
Amiodarone (Class IIa)
Catheter ablation (Class IIb)
Symptomatic recurrent VA
ESC

Effect of Corticosteroid Therapy on Ventricular Arrhythmias in Patients with Cardiac Sarcoidosis

Kenji Yodogawa, M.D.,* Yoshihiko Seino, M.D.,* Toshihiko Ohara, M.D.,† Hideo Takayama, M.D.,† Takao Katoh, M.D.,† and Kyoichi Mizuno, M.D.†

*From the *Division of Cardiology, Department of Internal Medicine, Nippon Medical School Chiba Hokusoh Hospital, Chiba, Japan; †Division of Cardiology, Hepatology, Geriatrics, and Integrated Medicine, Department Internal Medicine, Nippon Medical School, Tokyo, Japan*

Results: As a whole, there were no significant differences in the number of PVCs and in the prevalence of NSVT before and after steroid therapy. However, the less advanced LV dysfunction patients (EF ≥ 35%, n = 17) showed significant reduction in the number of PVCs (from 1820 ± 2969 to 742 ± 1425, P = 0.048) and in the prevalence of NSVT (from 41 to 6%, p = 0.039). Late potentials on SAECG were abolished in 3 patients. The less advanced LV dysfunction group showed a significantly higher prevalence of gallium-67 uptake compared with the advanced LV dysfunction group (EF < 35 %, n = 14). In the advanced LV dysfunction patients, there were no significant differences in these parameters.

Conclusions: Corticosteroid therapy may be effective for ventricular arrhythmias in the early stage, but less effective in the late stage.

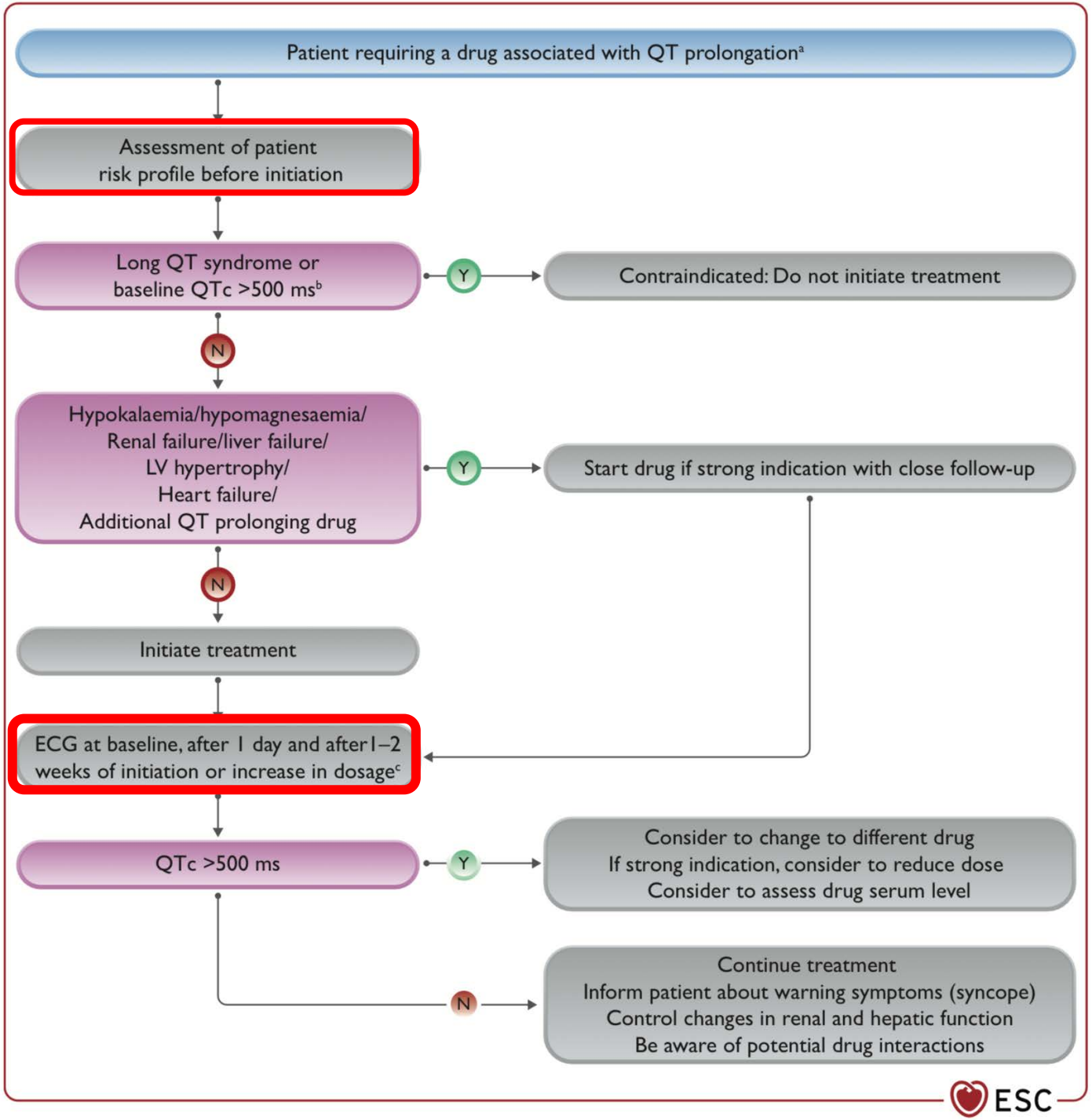
Anti-aritmik dərmanlar:

Anti-arrhythmic drug	Effects on ECG	Indications (specific indication)	Oral dose per day (i.v. dose)	Side effects	Contraindications, precautions, other considerations
Amiodarone	Decreases sinus node frequency, prolongs QT interval[a]	PVC, VT, VF	200–400 mg Loading dose: 600–1200 mg/ 24 h 8–10 days. (Loading dose: 5 mg/kg in 20 min–2 h, 2–3	*Cardiac:* Bradycardia, TdP (infrequent) *Extracardiac:* Photosensitivity, corneal deposits, hypothyroidism, hyperthyroidism,	*Precautions:* Sinus node dysfunction, severe AV conduction disturbances, hyperthyroidism *Other considerations:* Can be used in patients with

Until now,

no AAD except for beta-blockers has demonstrated reduction in all-cause mortality. Each drug has a significant potential for causing adverse events, including pro-arrhythmia.

				dry mouth	Discontinue if QRS widening >25% or bundle branch block
Sotalol	Decreases sinus node frequency, prolongs QT interval[a]	VT	160–640 mg (0.5–1.5 mg/kg in 10 min. If necessary, can be repeated after 6 h)	See beta-blockers, TdP[d] (>2% of patients, close monitoring of QT interval and CrCl)	*Contraindications:* Severe sinus node dysfunction, severe AV conduction disturbances, severe heart failure with reduced LVEF, significant LVH, CrCl <30 ml/min, coronary vasospasm, LQTS *Precautions:* Concomitant treatments associated with QT interval prolongation, hypokalaemia


Patient requiring a drug associated with QT prolongation[a]
Assessment of patient risk profile before initiation
Long QT syndrome or baseline QTc >500 ms[b]
Y
Contraindicated: Do not initiate treatment
N
Hypokalaemia/hypomagnesaemia/ Renal failure/liver failure/ LV hypertrophy/ Heart failure/ Additional QT prolonging drug
Y
Start drug if strong indication with close follow-up
N
Initiate treatment
ECG at baseline, after 1 day and after 1–2 weeks of initiation or increase in dosage[c]
QTc >500 ms
Y
Consider to change to different drug
If strong indication, consider to reduce dose
Consider to assess drug serum level
N
Continue treatment
Inform patient about warning symptoms (syncope)
Control changes in renal and hepatic function
Be aware of potential drug interactions
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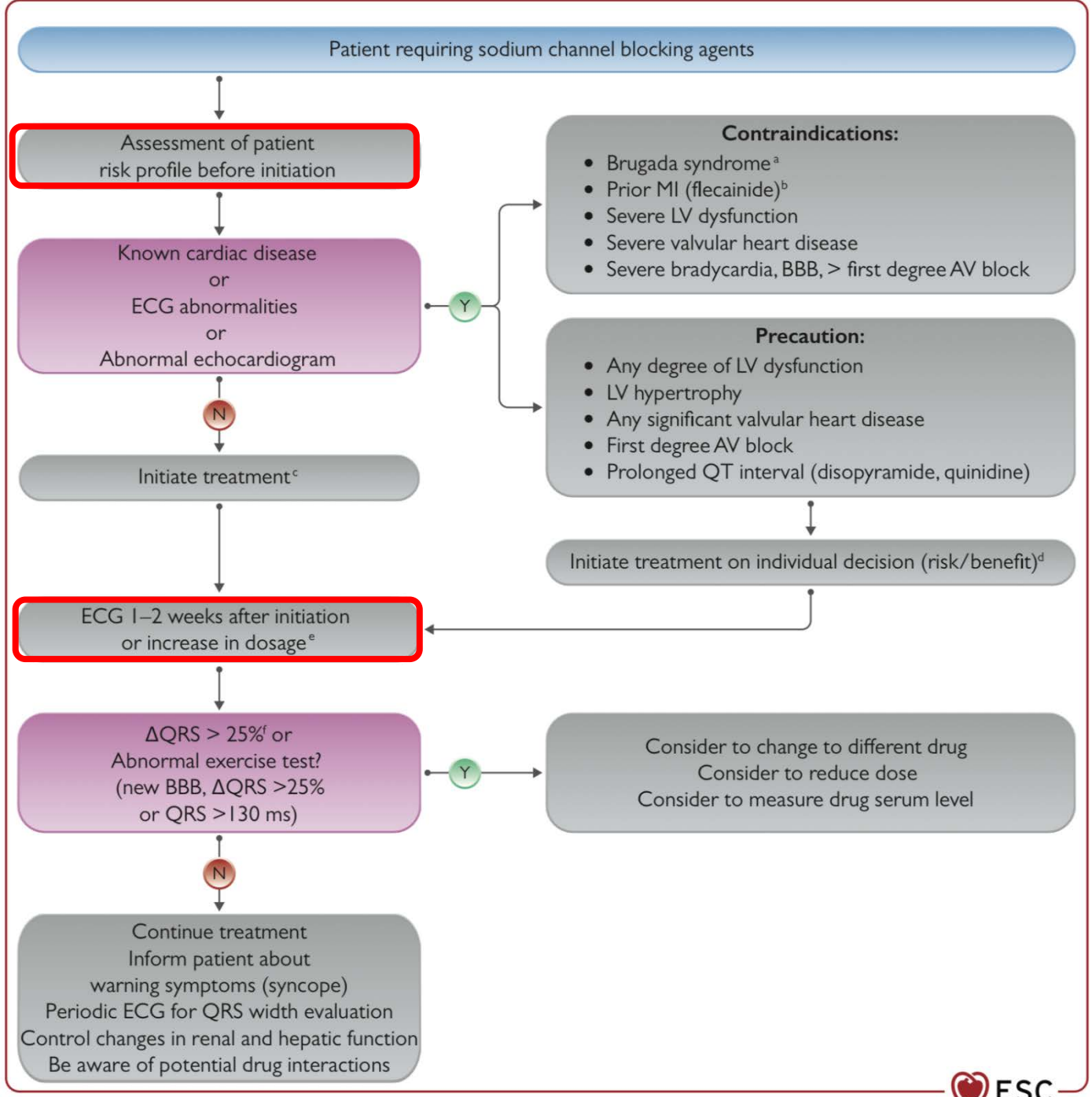
Patient requiring sodium channel blocking agents
Assessment of patient risk profile before initiation
Known cardiac disease or ECG abnormalities or Abnormal echocardiogram
Y
N
Contraindications:
Brugada syndrome[a]
Prior MI (flecainide)[b]
Severe LV dysfunction
Severe valvular heart disease
Severe bradycardia, BBB, > first degree AV block
Precaution:
Any degree of LV dysfunction
LV hypertrophy
Any significant valvular heart disease
First degree AV block
Prolonged QT interval (disopyramide, quinidine)
Initiate treatment on individual decision (risk/benefit)[d]
Initiate treatment[c]
ECG 1–2 weeks after initiation or increase in dosage[e]
ΔQRS > 25%[f] or Abnormal exercise test? (new BBB, ΔQRS >25% or QRS >130 ms)
Y
N
Consider to change to different drug
Consider to reduce dose
Consider to measure drug serum level
Continue treatment
Inform patient about warning symptoms (syncope)
Periodic ECG for QRS width evaluation
Control changes in renal and hepatic function
Be aware of potential drug interactions
ESC

Reference:

- A Companion to Braunwald Textbook of Cardiovascular Medicine-2018 (11th) Douglas P. Zipes MD, Peter Libby MD PhD, Robert O. Bonow MD MS, Douglas L. Mann MD
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- 2022 ESC Guidelines for the management ofpatients with ventricular arrhythmias and the prevention of sudden cardiac death
  2021 ESC Guidelines on cardiac pacing and cardiac resynchronization therapy
- Companion to Braunwald's Heart Disease-G. Michael Felker, Douglas Mann - Heart Failure-Elsevier (2019)


Rhythm of Life
Diqqətiniz üçün minnətdaram...