Cardio Pulse 23

09, 10 & 11 ΙΟΥΝΙΟΥ
JUNE 2023
CRETA CONVENTION CENTRE
HERSONISSOS, CRETE

Υπάρχει χώρος στην σύγχρονη αρρυθμιολογία για φάρμακα;

Γιώργος Ανδρικόπουλος,
Α Καρδιολογική Κλινική/Ηλεκτροφυσιολογίας Βηματοδότησης
«Ερρίκος Ντυνάν» Hospital Center, Αθήνα

Presenter Disclosure Information

The presenter has received honoraria for participation in lectures and advisory boards from the following pharmaceutical and biotechnology companies:

- *Abbot*
- *AstraZeneca,*
- *Bard,*
- *Bayer Healthcare,*
- *Boehringer Ingelheim,*
- *Boston Scientific,*
- *Bristol-Myers Squibb,*
- *ELPEN,*
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- *Lilly,*
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- *MSD,*
- *Pfizer,*
- *Sanofi,*
- *Servier,*
- *Unifarma,*
- *Vianex.*

Pulse 2004

and Cardio Electrics

"The Genetics of Cardiac Arrhythmias"

George K. Andrikopoulos, MD, FESC
1st Cardiac dpt, "Evangelismos" Hospital, Athens, Greece

Effect of the COVID-19 pandemic on cardiac electrophysiological ablation procedures in Greece — Data from the Hellenic Society of Cardiology Ablation Registry

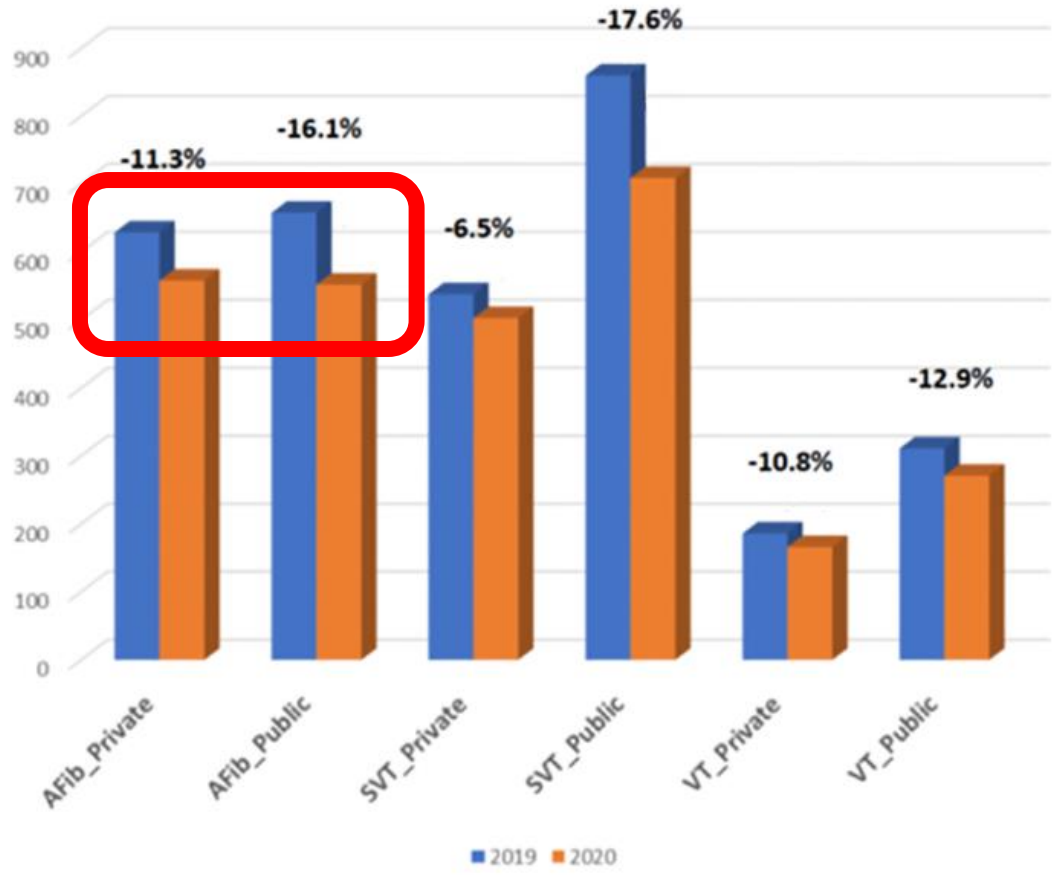


Figure 1. Caseloads of ablation procedures in private and public sector institutions in 2019 ("pre-COVID-19") and 2020 ("COVID-19"), as reported in the Hellenic Society of Cardiology Ablation Registry.

› Hellenic J Cardiol. 2022 Sep-Oct;67:76-78. doi: 10.1016/j.hjc.2022.07.002. Epub 2022 Jul 8.

Effect of the COVID-19 pandemic on cardiac electrophysiological ablation procedures in Greece - Data from the Hellenic Society of Cardiology Ablation Registry

Vassilios P Vassilikos [1], Georgios Giannopoulos [2], Nikolaos Fragakis [1], Antonis Billis [3], Michalis Efremidis [4], Konstantinos Letsas [4], Themistoklis Maounis [4], Anna Kostopoulou [4], Georgios Andrikopoulos [5], Sokratis Pastromas [5], Apostolos Katsivas [6], Charalambos Kossyvakis [7], Eleftherios Kallergis [8], Emmanouel Kanoupakis [8], Panagiotis Ioannidis [9], Stelios Tzeis [10], Dimosthenis Avramidis [10], Ioannis Papagiannis [10], Spyridon Deftereos [11], Eftychia Symeonidou [11], Dimitrios Tsiachris [12], George Theodorakis [13], Ioannis Rassias [13], Dimitrios Lysitsas [14], Eleni Chatzinikolaou [14], Nikolaos Mezilis [14], Stylianos Paraskevaidis [15], Spyros Kourouklis [16], Theodoros Apostolopoulos [16], Dimosthenis Katritsis [16], Sophia Chatzidou [17], Lilian Mantziari [18], Georgios Leventopoulos [19], Ioannis Chiladakis [19], George Kourgiannidis [20], George Stavropoulos [21], Sotirios Xydonas [22], Charilaos Ginos [23], Athanasios Kotsakis [24], Giannis Baltogiannis [25], Antonis S Manolis [26], Skevos Sideris [26], Konstantinos Gatzoulis [26]

Έλεγχος Ρυθμού ΟΧΙ Συχνότητας

ORIGINAL ARTICLE

Early Rhythm-Control Therapy in Patients with Atrial Fibrillation

P. Kirchhof, A.J. Camm, A. Goette, A. Brandes, L. Eckardt, A. Elvan, T. Fetsch, I.C. van Gelder, D. Haase, L.M. Haegeli, F. Hamann, H. Heidbüchel, G. Hindricks, J. Kautzner, K.-H. Kuck, L. Mont, G.A. Ng, J. Rekosz, N. Schoen, U. Schotten, A. Suling, J. Taggeselle, S. Themistoclakis, E. Vettorazzi, P. Vardas, K. Wegscheider, S. Willems, H.J.G.M. Crijns, and G. Breithardt, for the EAST-AFNET 4 Trial Investigators*

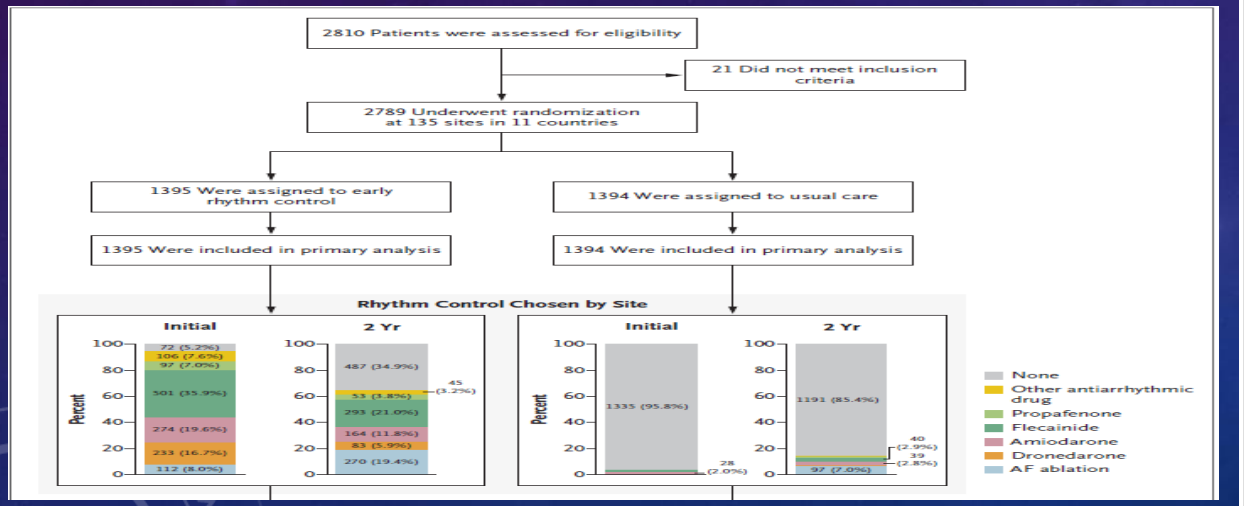
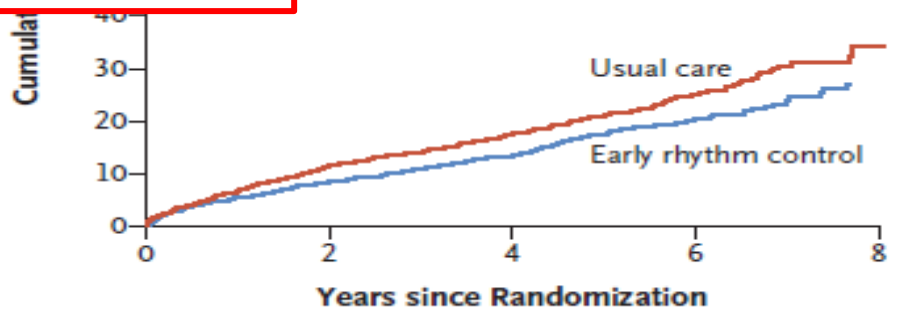


Table 1. Demographic and Clinical Characteristics of the Patients at Baseline.*

Characteristic	Early Rhythm Control (N=1395)	Usual Care (N=1394)
Age — yr	70.2±8.4	70.4±8.2
Female sex — no. (%)	645 (46.2)	648 (46.5)
Body-mass index†	29.2±5.4	29.3±5.4
Type of atrial fibrillation — no./total no. (%)		
First episode	528/1391 (38.0)	520/1394 (37.3)
Paroxysmal	501/1391 (36.0)	493/1394 (35.4)
Persistent	362/1391 (26.0)	381/1394 (27.3)
Sinus rhythm at baseline — no./total no. (%)	762/1389 (54.9)	743/1393 (53.3)
Median days since atrial fibrillation diagnosis (IQR)‡	36.0 (6.0–114.0)	36.0 (6.0–112.0)

Methods

The current analysis was prespecified in the statistical analysis plan and performed on the final, locked database of the EAST-AFNET 4 trial. Design and topline results of the main trial have been published.[8,10] In brief, the EAST-AFNET 4 trial is an international, investigator-initiated, parallel-group, open, blinded- outcome-assessment (PROBE) trial, which randomly assigned patients who had AF diagnosed ≤1 year before enrolment and cardiovascular conditions to receive either early rhythm control in all patients or usual care. Early rhythm control included treatment with antiarrhythmic drugs or AF ablation in all patients directly after randomization. Usual care included rhythm control therapy to improve AF-related symptoms.[8,10]



No. at Risk					
Usual care	1394	1169	888	405	34
Early rhythm control	1395	1193	913	404	26

Figure 2. Aalen–Johansen Cumulative-Incidence Curves for the First Primary Outcome.

The first primary outcome was a composite of death from cardiovascular causes, stroke, or hospitalization with worsening of heart failure or acute coronary syndrome.

Kirchof P., et al. NEJM August 19, 2020

ORIGINAL ARTICLE

Early Rhythm-Control Therapy in Patients with Atrial Fibrillation

P. Kirchhof, A.J. Camm, A. Goette, A. Brandes, L. Eckardt, A. Elvan, T. Fetsch, I.C. van Gelder, D. Haase, L.M. Haegeli, F. Hamann, H. Heidbüchel, G. Hindricks, J. Kautzner, K.-H. Kuck, L. Mont, G.A. Ng, J. Rekosz, N. Schoen, U. Schotten, A. Suling, J. Taggeselle, S. Themistoclakis, E. Vettorazzi, P. Vardas, K. Wegscheider, S. Willems, H.J.G.M. Crijns, and G. Breithardt, for the EAST-AFNET 4 Trial Investigators*

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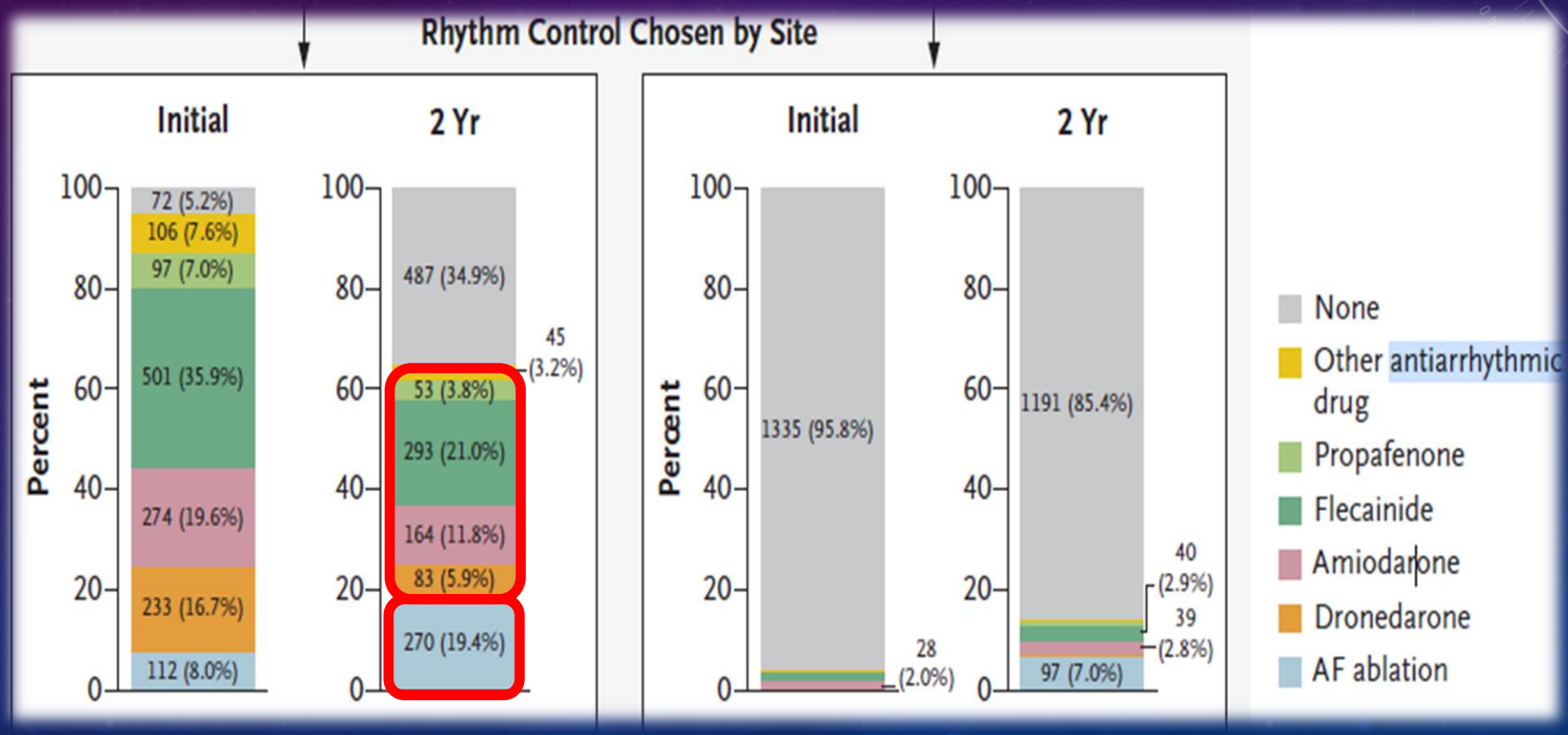


Kirchof P., et al. NEJM August 19, 2020

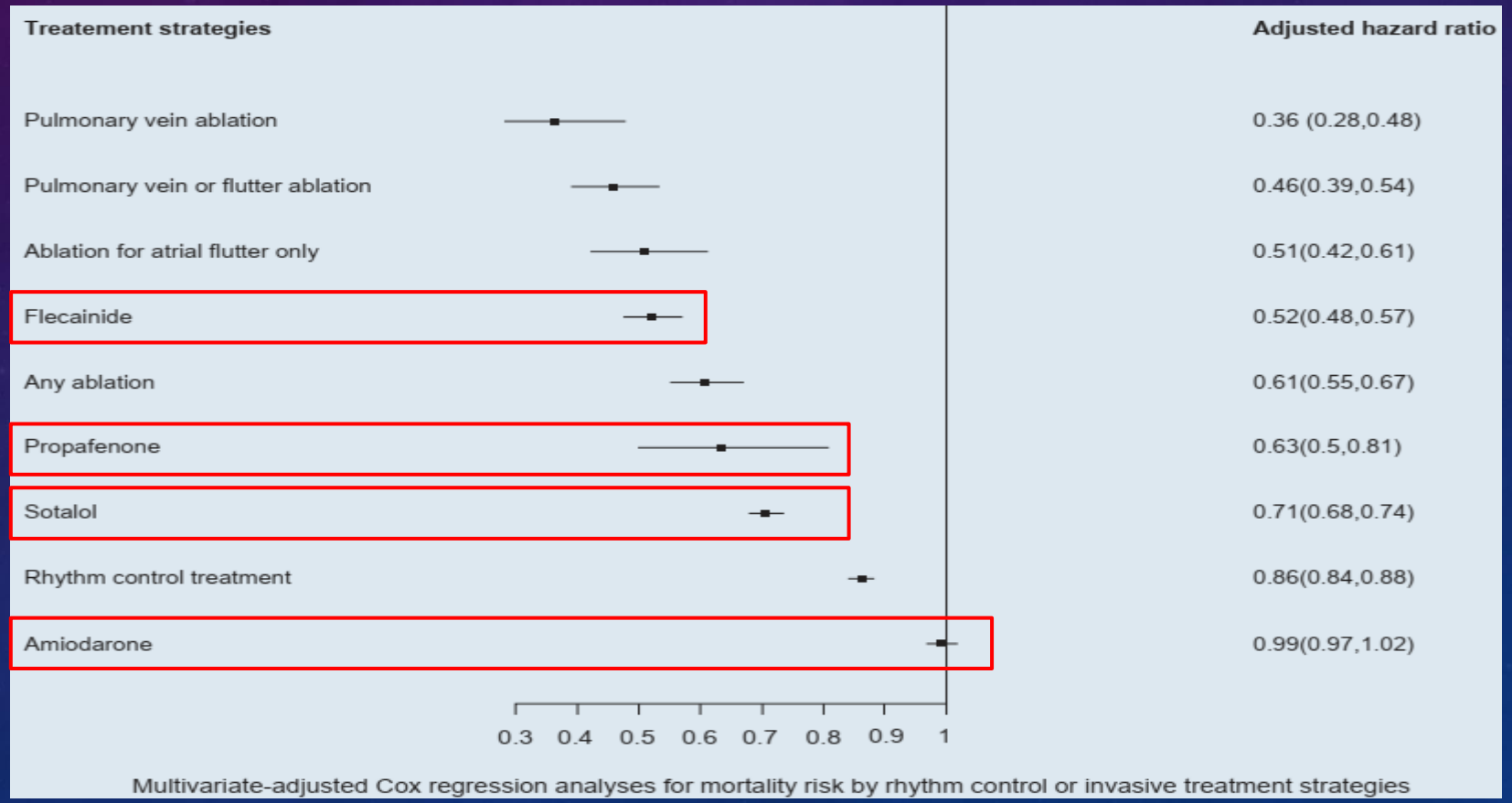
ESC European Society of Cardiology
Europace (2023) **25**, 351–359
https://doi.org/10.1093/europace/euac155

CLINICAL RESEARCH

Chung Sheng-Chia, et al. Europace (2023) 25, 351–359

Impact of anti-arrhythmic drugs and catheter ablation on the survival of patients with atrial fibrillation: a population study based on 199 433 new-onset atrial fibrillation patients in the UK

We identified 199 433 individuals (mean age at diagnosis 75.7 ± 12.7 years; 50.2% women) with new-onset AF diagnosis in nationwide electronic health records linking primary care consultation with hospital data and death registry data from 1998



Multivariate-adjusted Cox regression analyses for mortality risk by rhythm control or invasive treatment strategies

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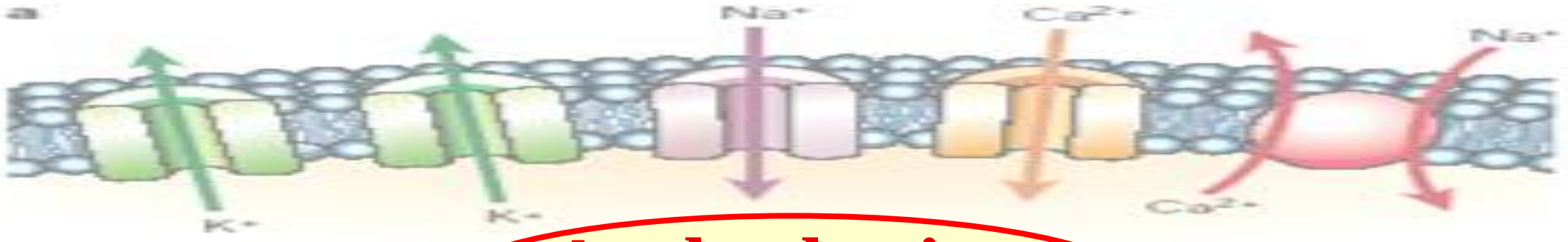
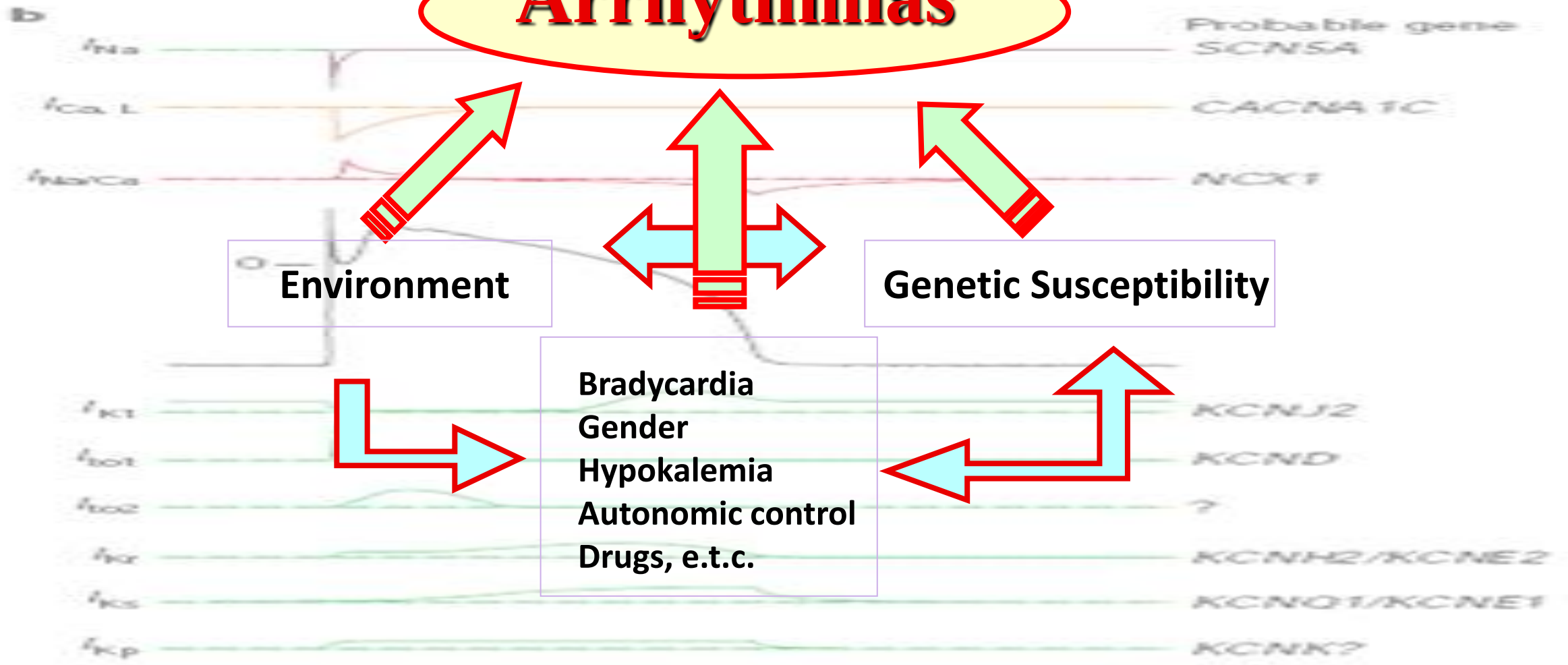
ΝΑΙ

Γιώργος Ανδρικόπουλος,
Α Καρδιολογική Κλινική/Ηλεκτροφυσιολογίας Βηματοδότησης
«Ερρίκος Ντυνάν» Hospital Center, Αθήνα



Arrhythmias



Environment

Genetic Susceptibility

Bradycardia
Gender
Hypokalemia
Autonomic control
Drugs, e.t.c.

VAUGHAN – WILLIAMS CLASSIFICATION

- Class I : sodium inhibitors
  - Ia : Quinidine, Disopyramide
  - Ib : Lidocaïne, Mexiletine
  - Ic : Flecaïnide, Propafenone, Cibenzoline
- Class II : beta-blockers
- Class III : potassium blockers : Amiodarone, Sotalol
- Class IV : calcium inhibitors : Verapamil, Diltiazem

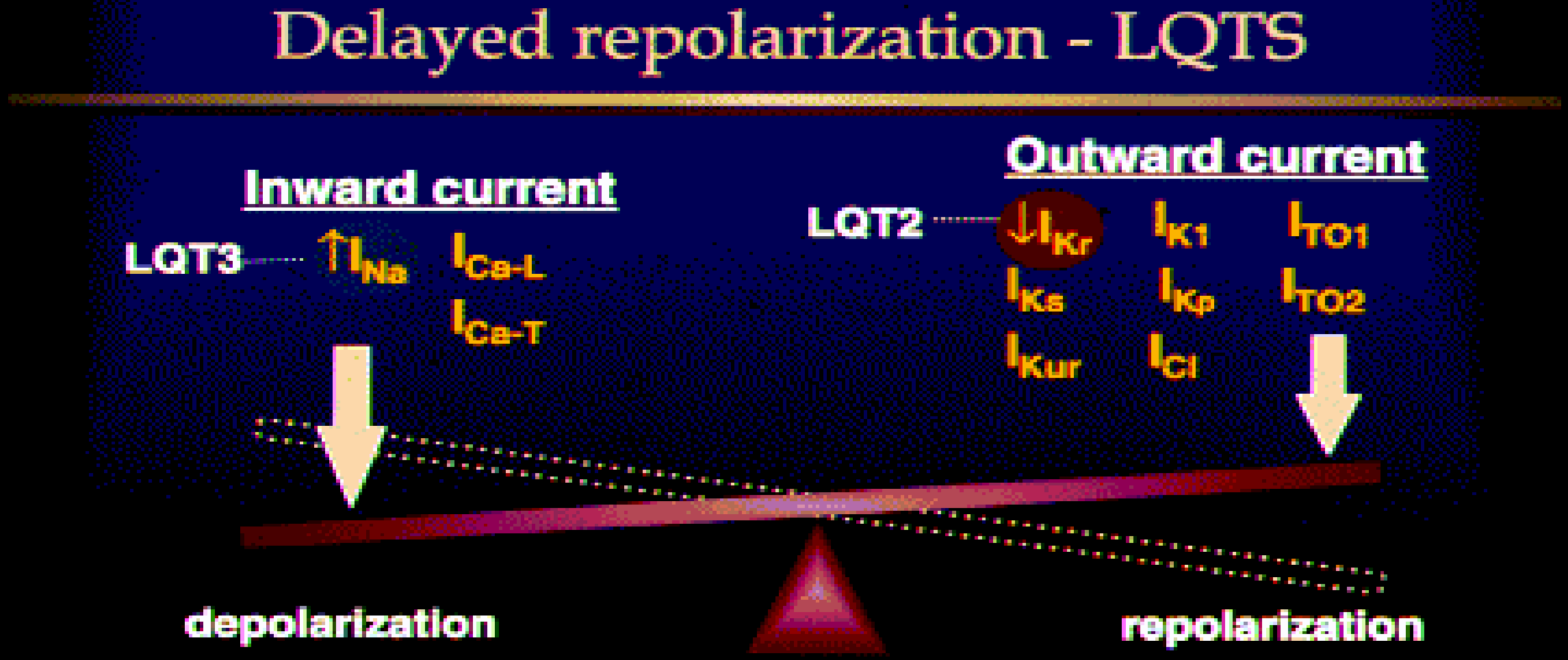
EVIDENCE FOR INCREASED MORTALITY IN PATIENTS TREATED WITH ANTIARRHYTHMIC DRUGS

Evidence for increased mortality in patients treated with antiarrhythmic drugs

Study	Population/ design	Results
CAST [1]	Randomized prospective comparison of placebo, flecainide and encainide in post-MI patients with PVCs.	Increased total and sudden death mortality with flecainide and encainide.
CAST II [2]	Randomized prospective comparison of placebo and moricizine, post-MI patients with PVCs.	Increased total and sudden death mortality with moricizine.
IMPACT [3]	Randomized prospective trial of mexiletine vs. placebo in post-MI patients with PVCs.	Increased mortality with mexiletine.
SWORD [4]	Randomized prospective comparison of placebo vs. D-sotalol in post MI patients with left ventricular dysfunction.	Increased total and sudden death mortality with D-sotalol.
Coplen et al. [5]	Meta-analysis of placebo-controlled studies of quinidine for AF.	Increased mortality with quinidine.
Flaker et al. [6]	Retrospective analysis of data from SPAF trial.	Excess mortality for AF patients with heart failure receiving antiarrhythmic drugs. No difference in absence of heart failure.
Nattel et al. [7]	Analysis of data from controlled trials of drug therapy of AF.	Increased mortality with quinidine, disopyramide, flecainide, and sotalol.
Moosvi et al. [8]	Retrospective analysis of empiric therapy for cardiac arrest patients.	Increased rate of recurrent cardiac arrest in patients receiving empiric quinidine or procainamide

Nattel S., et al. Cardiovascular Research 1998

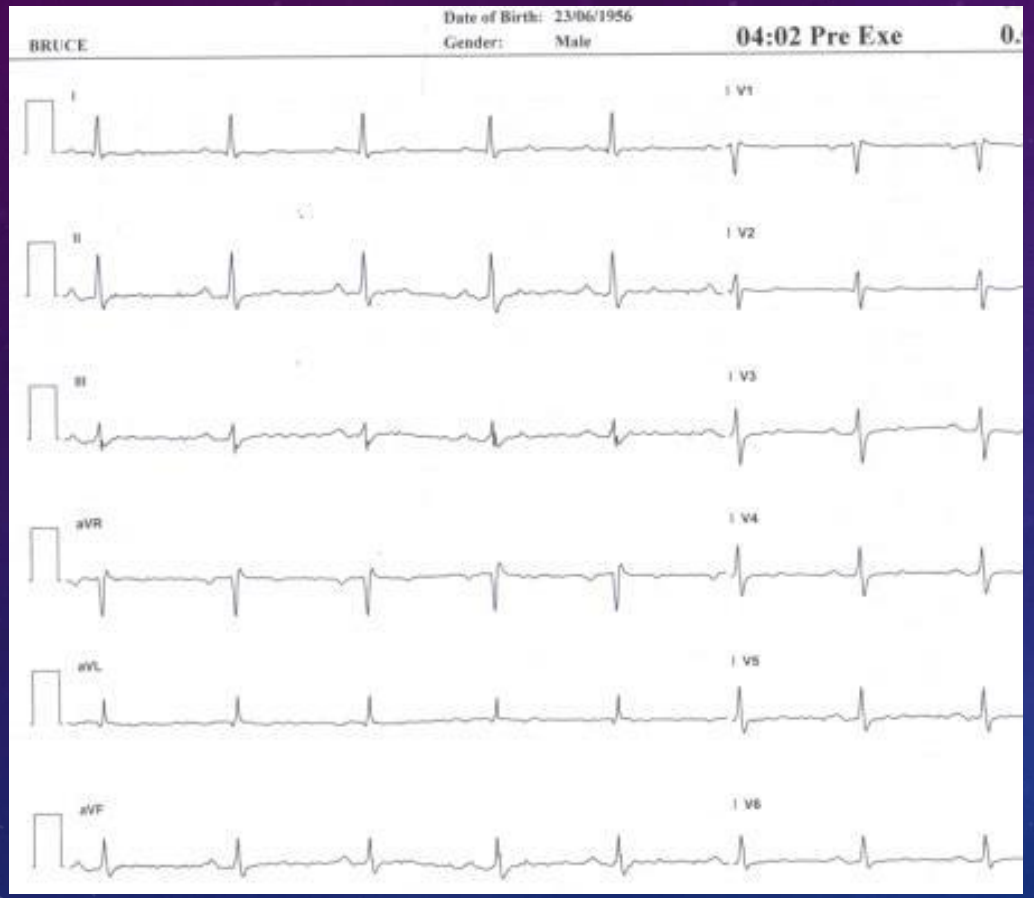
The concept of “Reduced Repolarization reserve”



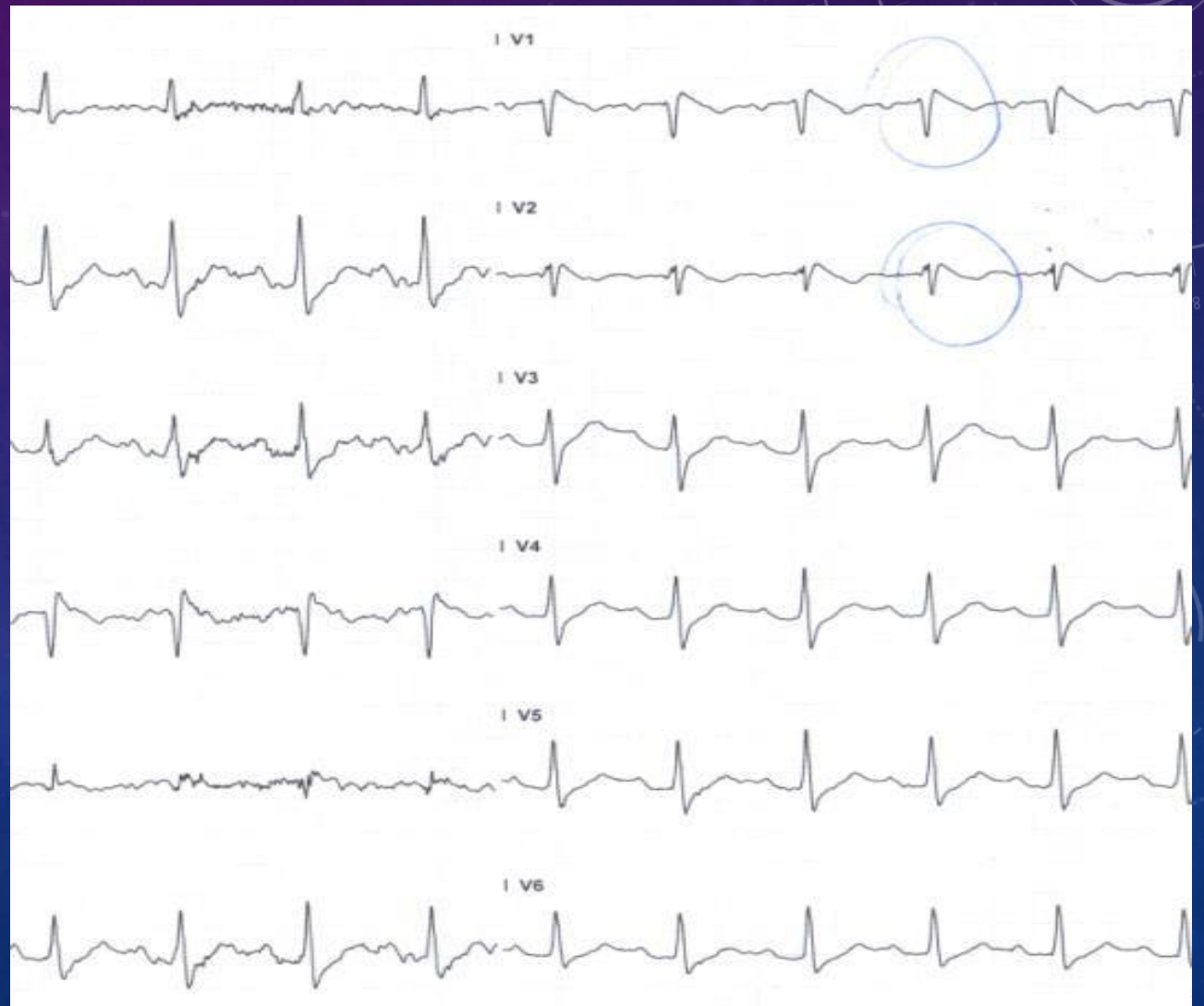
Even in susceptible substrate arrhythmias occur very rare and in an unpredictable fashion

Exercise-induced QRS prolongation in a 58-year old patient (physician) who was using propafenone to prevent AF

Before ET



Recovery (2 min)

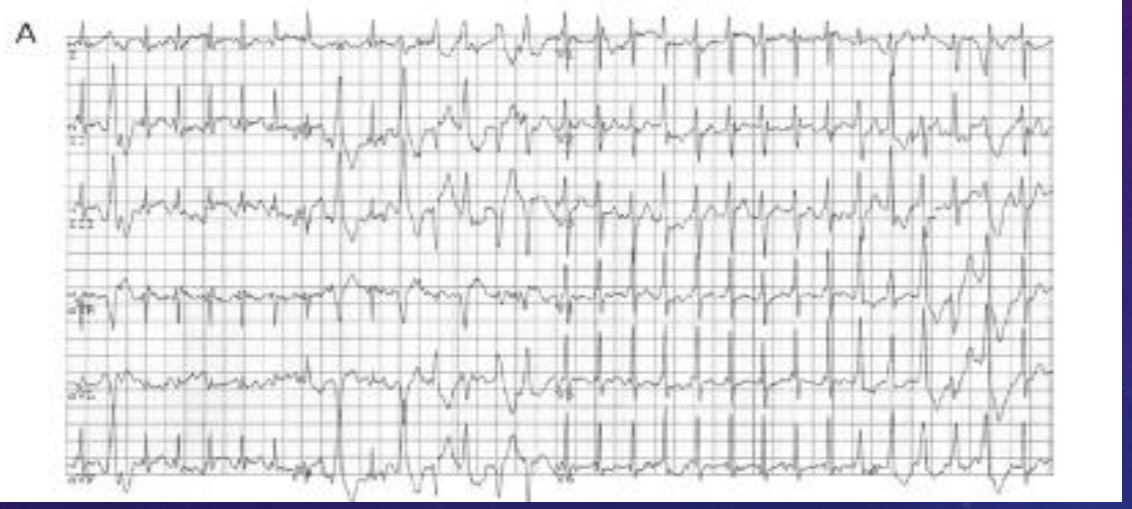


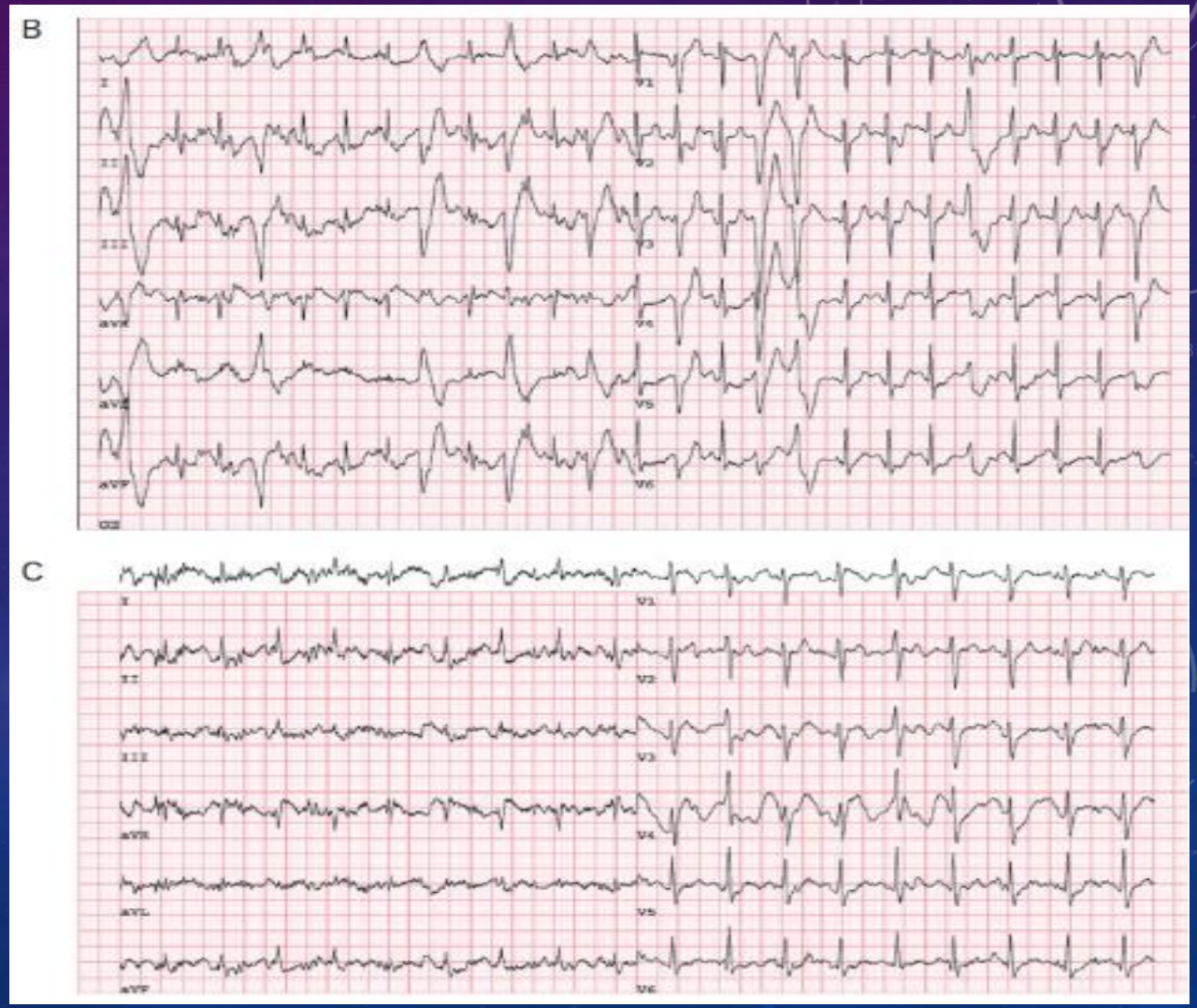
Αρχείο Γ.Ανδρικόπουλου

The Role of Flecainide in the Management of Catecholaminergic Polymorphic Ventricular Tachycardia

Krystien VV Lieve,[1] Arthur A Wilde,[1,2] Christian van der Werf[1]

Figure 1: ECGs at Maximum Heart Rate During Exercise Testing Before and After Drug Treatments in a Female Patient with Catecholaminergic Polymorphic Ventricular Tachycardia





A: At baseline before medication; polymorphic NSVT and VES were observed. B: After bisoprolol (5 mg/day); VES, bigeminy and a couplet. C: With metropolol 50 mg/day and flecainide 150 mg/day ventricular arrhythmias were completely suppressed. CPVT = catecholaminergic polymorphic ventricular tachycardia; NSVT = non-sustained ventricular tachycardia; VES = ventricular extrasystoles.

*Arrhythmia & Electrophysiology Review 2016;**5**(1):45–9*

Flecainide: Current status and perspectives in arrhythmia management

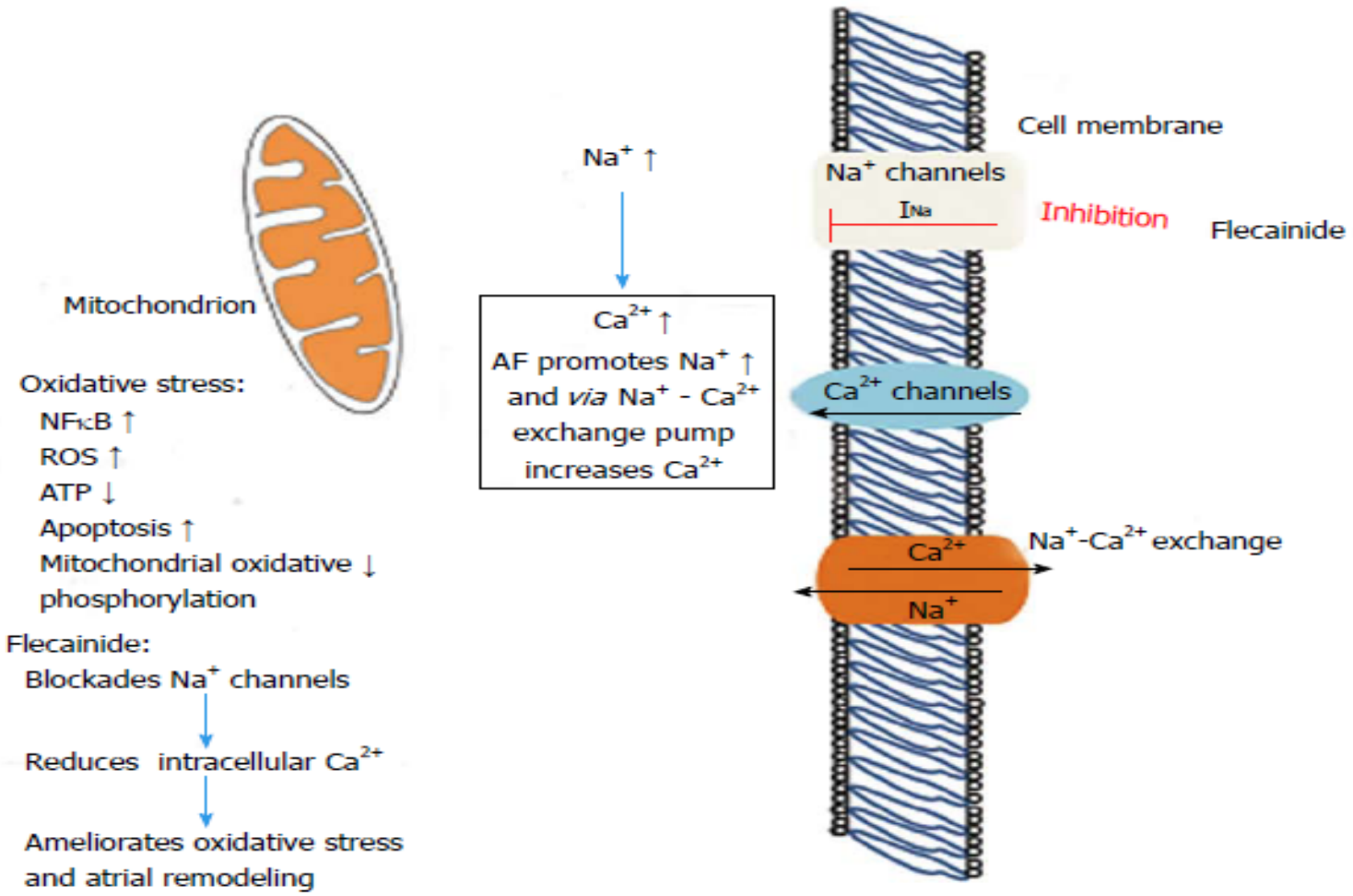
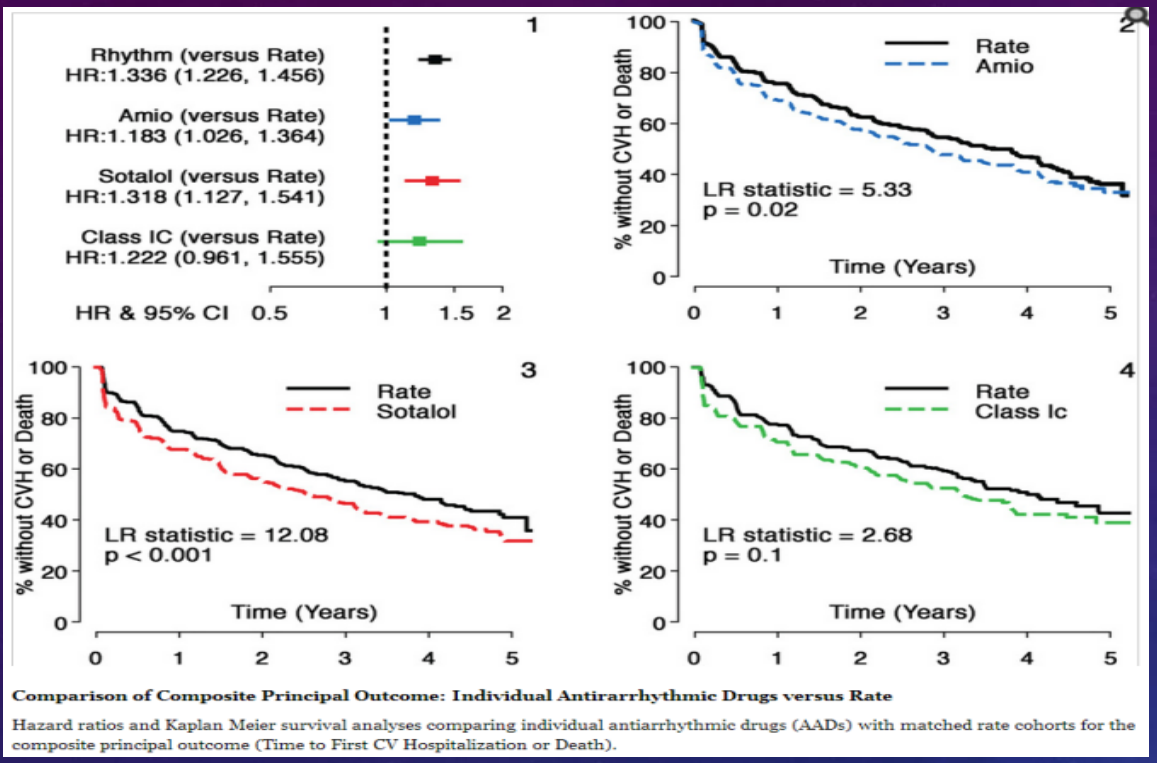


Figure 2 Mechanism of flecainide action during atrial fibrillation by inhibition of Na^+ channels which reduces intracellular Ca^{2+} accumulation and reduces oxidative stress and mitochondrial dysfunction. AF: Atrial fibrillation; INa: Fast inward Na^+ current; ROS: Reactive oxygen species; $NF_{\kappa}B$: Nuclear factor kappa β; ATP: Adenosine triphosphate.

Cardiovascular Outcomes in the AFFIRM Trial: An Assessment of Individual Antiarrhythmic Drug Therapies compared to Rate Control Using Propensity Score Matched Analyses



Comparison of Composite Principal Outcome: Individual Antirarrhythmic Drugs versus Rate

Hazard ratios and Kaplan Meier survival analyses comparing individual antiarrhythmic drugs (AADs) with matched rate cohorts for the composite principal outcome (Time to First CV Hospitalization or Death).

Results—729 amiodarone patients, 606 sotalol patients & 268 class 1C patients were matched. The composite outcome of mortality or CV hospitalizations (CVH) showed better outcomes with Rate compared to amiodarone (Hazard Ratio [HR] 1.18, 95% confidence intervals {CI}:1.03–1.36, p=0.02), sotalol (HR=1.32, CI: 1.13–1.54, p<0.001) and class 1C (HR=1.22, CI: 0.97–1.56, p=0.10). There was a non-significant increase in mortality with amiodarone (HR=1.20, CI: 0.94–1.53, p=0.15) with the risk of non-CV death, being significantly higher with amiodarone versus Rate. (HR=1.11, CI: 1.01–1.24, p=0.04). First CVH event rates at 3 years were 47% for amiodarone, 50% for sotalol and 44% for class 1C versus 40%, 40% and 36% respectively for Rate (amiodarone HR=1.20,CI:1.03–1.40,p=0.02, sotalol HR=1.364, CI:1.16–1.611, p<0.001, class 1C HR=1.24,CI:0.96–1.60,p=0.09). Time to CVH with intensive care unit stay (ICUH) or death was shorter with amiodarone (HR=1.22, CI: 1.02–1.46, p=0.03).

Potential benefit of rhythm control offset by antiarrhythmic drug toxicity

Conclusions—

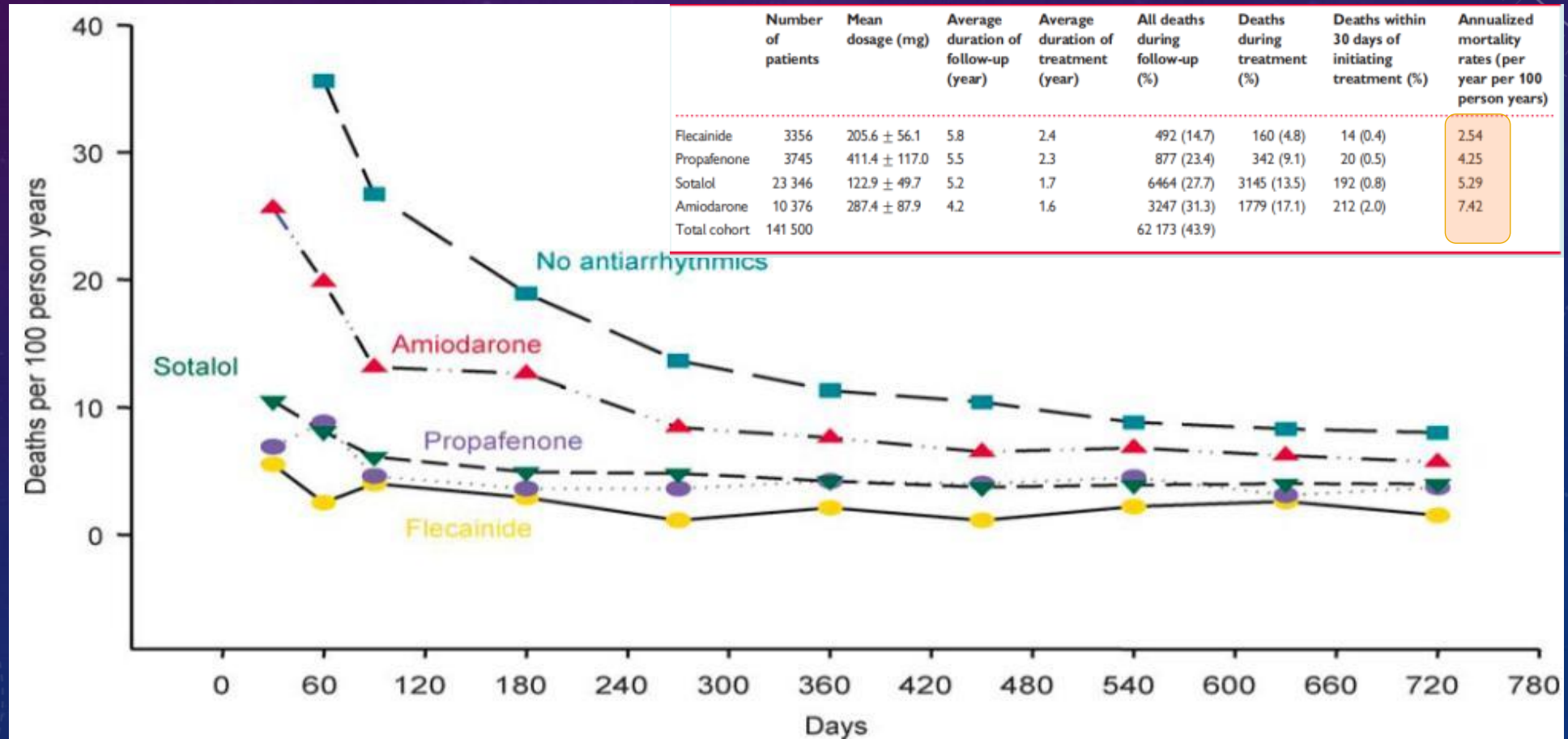
1. In AFFIRM, composite mortality and CVH outcomes differed for Rate and AADs due to differences in CVH; CVH event rates during follow-up were high for all cohorts, but they were higher for all groups on AADs.
2. Death, ICUH and non-CV death were more frequent with amiodarone.

Saksena S., et al. JACC 2011

EUROPEAN SOCIETY OF CARDIOLOGY®
Europace (2009) **11**, 886–891
doi:10.1093/europace/eup119

Antiarrhythmic therapy and risk of death in patients with atrial fibrillation: a nationwide study

	Number of patients	Mean dosage (mg)	Average duration of follow-up (year)	Average duration of treatment (year)	All deaths during follow-up (%)	Deaths during treatment (%)	Deaths within 30 days of initiating treatment (%)	Annualized mortality rates (per year per 100 person years)
Flecainide	3356	205.6 ± 56.1	5.8	2.4	492 (14.7)	160 (4.8)	14 (0.4)	2.54
Propafenone	3745	411.4 ± 117.0	5.5	2.3	877 (23.4)	342 (9.1)	20 (0.5)	4.25
Sotalol	23 346	122.9 ± 49.7	5.2	1.7	6464 (27.7)	3145 (13.5)	192 (0.8)	5.29
Amiodarone	10 376	287.4 ± 87.9	4.2	1.6	3247 (31.3)	1779 (17.1)	212 (2.0)	7.42
Total cohort	141 500				62 173 (43.9)			



Safety of Dronedarone in Routine Clinical Care

Methods All 174,995 patients with a diagnosis of AF during 2010 to 2012 were identified in the Swedish Patient Register. Of these, 4,856 patients had received dronedarone according to the Swedish Drug Register, and 170,139 patients who had not were used as a control population. Mean follow-up was 1.6 years, with a minimal follow-up of 6 months.

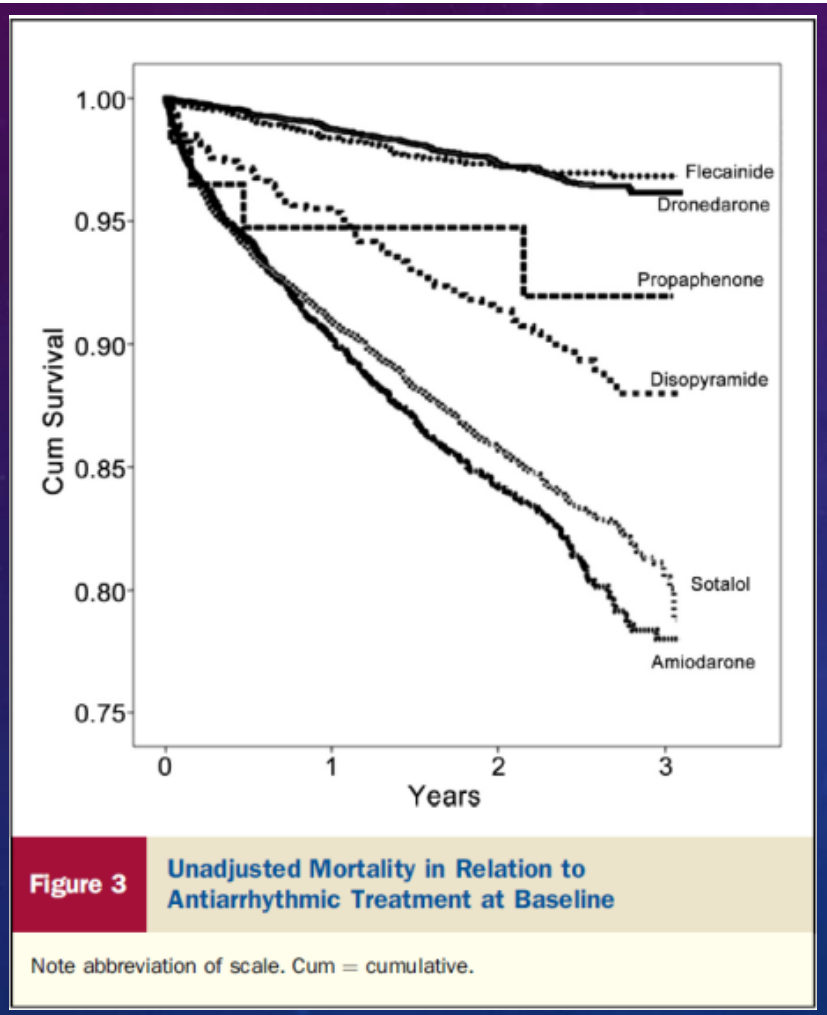


Figure 3 Unadjusted Mortality in Relation to Antiarrhythmic Treatment at Baseline

Note abbreviation of scale. Cum = cumulative.

Table 2 Hazard Ratios for Death of Any Cause With Dronedarone, With No Dronedarone as Reference

Adjustment	Intention to Treat (At Least 1 Purchase)	On Treatment (Drug ≥ 80% of Time at Risk)
Univariate	0.10 (0.08-0.12)	0.03 (0.02-0.06)
Age and sex	0.24 (0.20-0.29)	0.08 (0.04-0.13)
Clinical risk factors†	0.29 (0.25-0.35)	0.09 (0.06-0.16)
Clinical risk factors† and medication		
All ages	0.32 (0.27-0.38)	0.11 (0.06-0.19)
<65 yrs	0.37 (0.24-0.55)	0.21 (0.08-0.56)
65-74 yrs	0.39 (0.30-0.51)	0.12 (0.06-0.28)
≥75 yrs	0.31 (0.23-0.41)	0.08 (0.03-0.22)
Propensity score matched	0.41 (0.33-0.51)	0.18 (0.10-0.31)

Table 1 Clinical Characteristics of AF Patients With and Without Dronedarone

	Dronedarone		
	Yes (n = 4,856)	No (n = 170,139)	p Value
Age, yrs*	65.5 ± 9.9	75.7 ± 12.1	<0.0001
≥75 yrs	17.2	59.5	<0.0001
Sex*			<0.0001
Men	58.8	55.6	
Women	41.2	44.4	
$CHADS_2$ score	1.3 ± 1.1	2.3 ± 1.5	<0.0001
CHA_2DS_2-VASc score	2.5 ± 1.6	3.8 ± 1.9	<0.0001
HAS-BLED score	1.9 ± 1.1	2.6 ± 1.2	<0.0001
Heart failure*	16.7	32.5	<0.0001
Hypertension*	65.7	68.4	<0.0001
Diabetes mellitus*	11.3	19.5	<0.0001
Ischemic stroke	6.3	14.9	<0.0001
Thromboembolism (arterial)*	12.0	23.5	<0.0001
Myocardial infarction	11.3	19.0	<0.0001
Ischemic heart disease	19.3	27.6	<0.0001
Revascularization (PCI or CABG)	9.4	12.0	<0.0001
Vascular disease (as in CHA_2DS_2-VASc)*	14.1	24.3	<0.0001
Peripheral arterial disease	4.0	8.0	<0.0001
Valvular AF* (mitral stenosis or mechanical valve)	3.8	4.8	0.002
Other valvular disease*	8.9	11.4	<0.0001
Pacemaker or ICD*	9.2	9.4	0.73
Renal failure*	2.0	6.6	<0.0001
Liver disease*	0.8	1.4	0.001
Thyroid disease*	9.9	8.8	0.001
Chronic obstructive pulmonary disease*	4.6	8.3	<0.0001
Venous thromboembolism*	3.4	5.3	<0.0001
Any bleeding*	12.6	21.5	<0.0001
Intracranial bleeding	0.8	2.1	0.0001
Gastrointestinal bleeding	4.3	7.0	<0.0001
Other bleeding	8.0	10.4	<0.0001
Transfusion	1.7	7.6	<0.0001
Anemia*	4.0	10.7	<0.0001
Coagulation or platelet defect*	2.1	2.6	0.046
Cancer within 3 yrs*	13.8	19.0	<0.0001
Alcohol index*	1.5	3.2	<0.0001
Dementia*	0.1	4.7	<0.0001

Leif Frieberg. Safety of Dronedarone in Routine Clinical Care. J Am Coll Cardiol 2014;63:2376–84

Imperial College London

National Institute for Health Research

Royal Brompton & Harefield NHS Foundation Trust

The Changing Landscape of Oral Anti-arrhythmic Prescriptions for Atrial Fibrillation in England: 1998-2014

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[1]NIHR Cardiovascular Biomedical Research Unit, Royal Brompton Hospital, London, UK; [2]Royal Free Hospital, London, UK; [3]North West London Hospitals NHS Trust, London, UK; [4]The Heart Hospital, London, UK

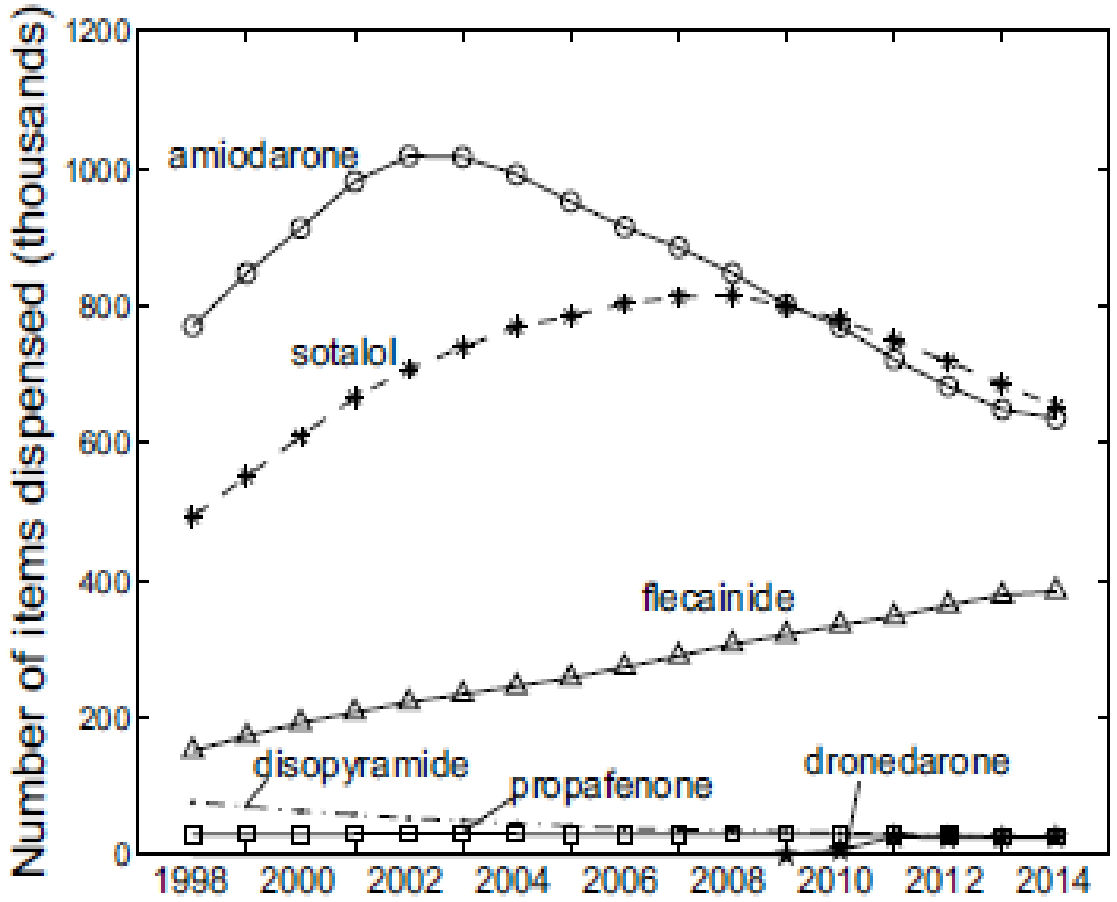
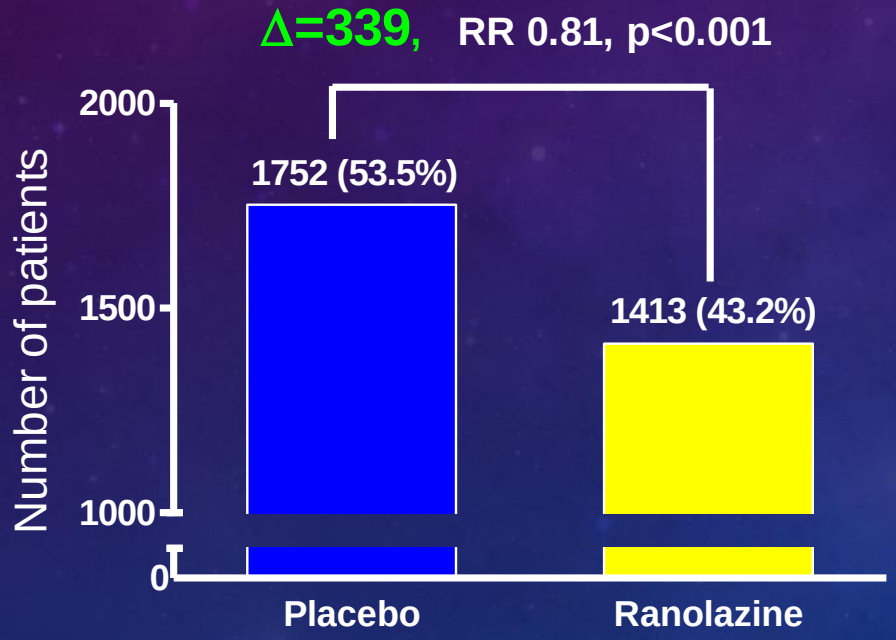


Figure 1: The trend in anti-arrhythmic drug dispensations in England 1998-2014

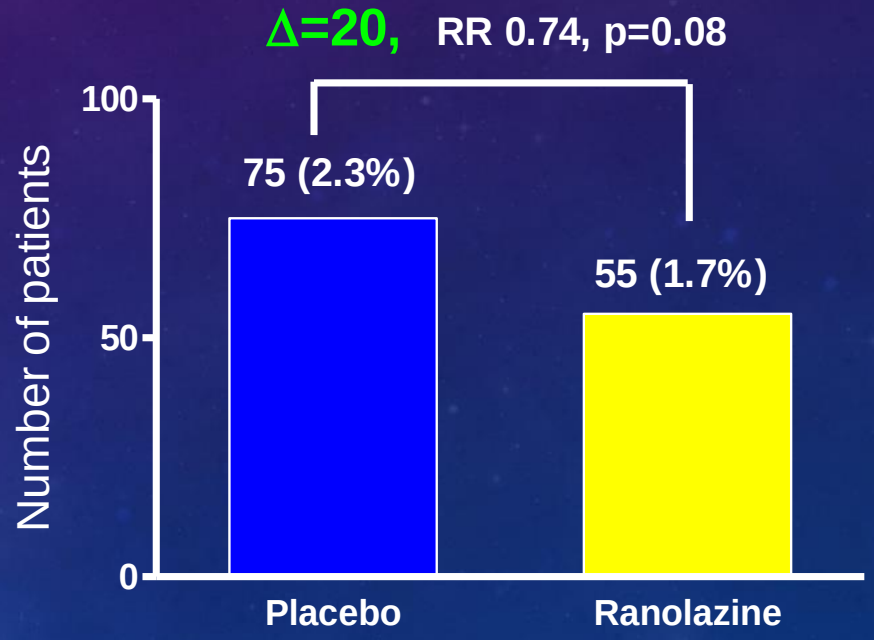
ESC 2015

SUPRAVENTRICULAR TACHYARRYTHMIAS* IN THE MERLIN - TIMI 36 TRIAL

A. Supraventricular Tachycardia



B. New-Onset Atrial Fibrillation



*Detected during 6 days of cECG monitoring

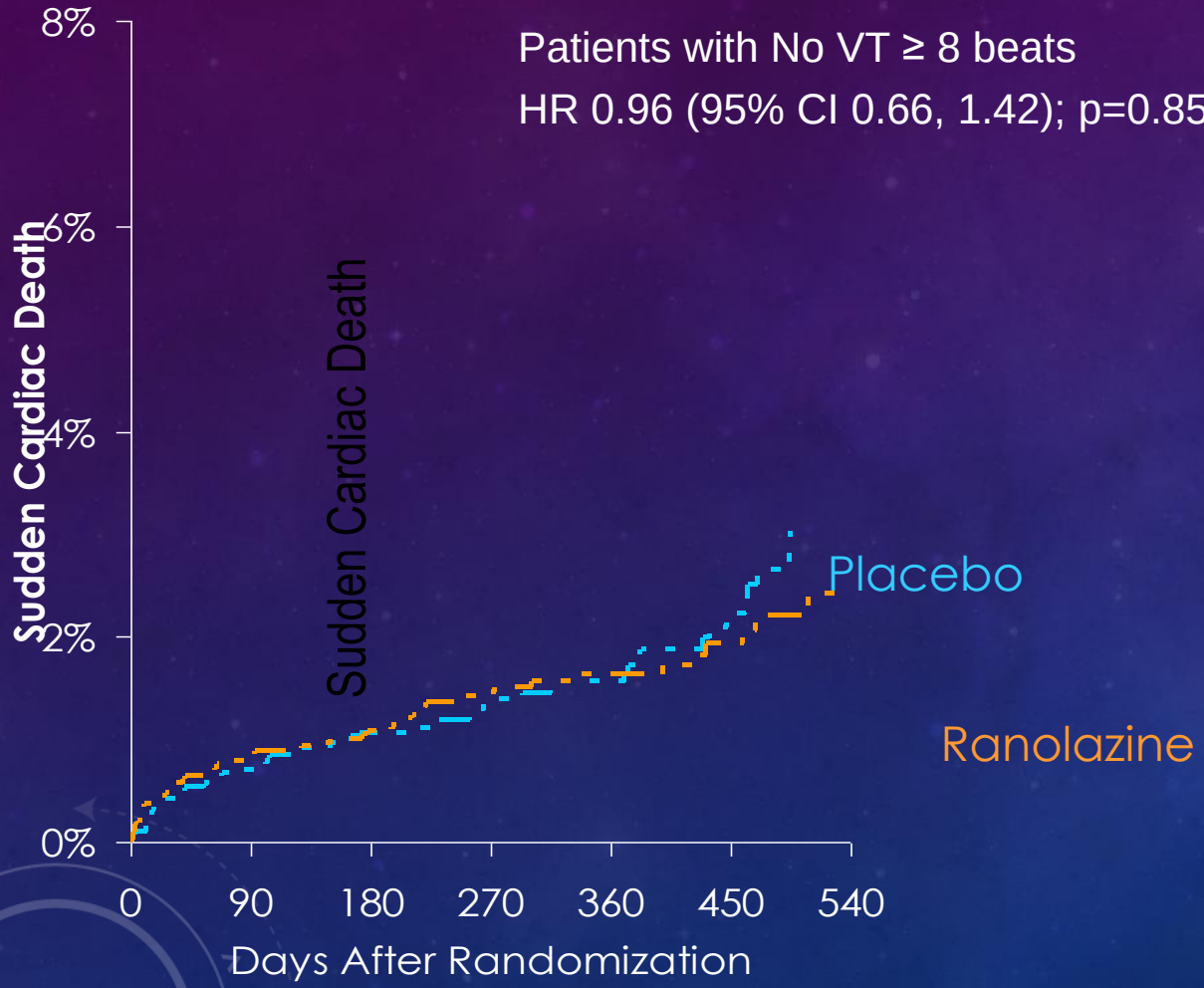
Scirica et al. Circulation. 116:1449-1457, 2007

RISK OF SUDDEN CARDIAC DEATH ASSOCIATED WITH VENTRICULAR TACHYCARDIA LASTING ≥ 8 BEATS

NO VT

Patients with No VT ≥ 8 beats

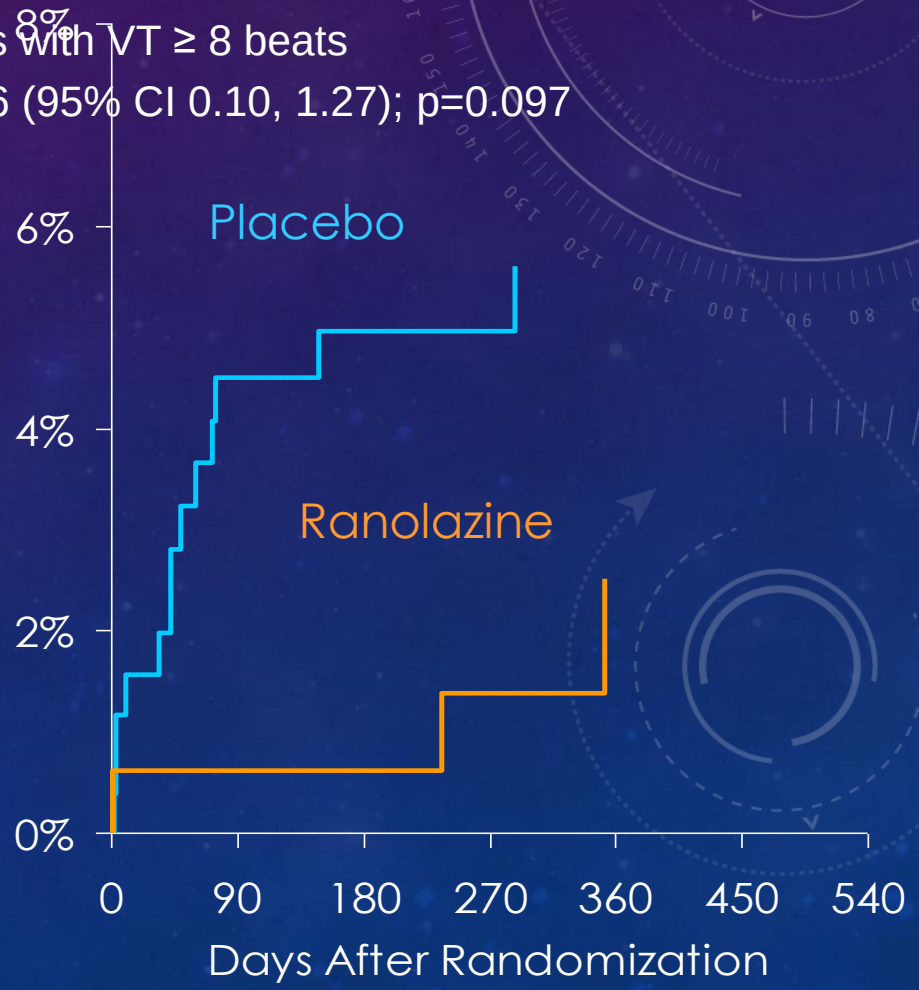
HR 0.96 (95% CI 0.66, 1.42); p=0.85



VT

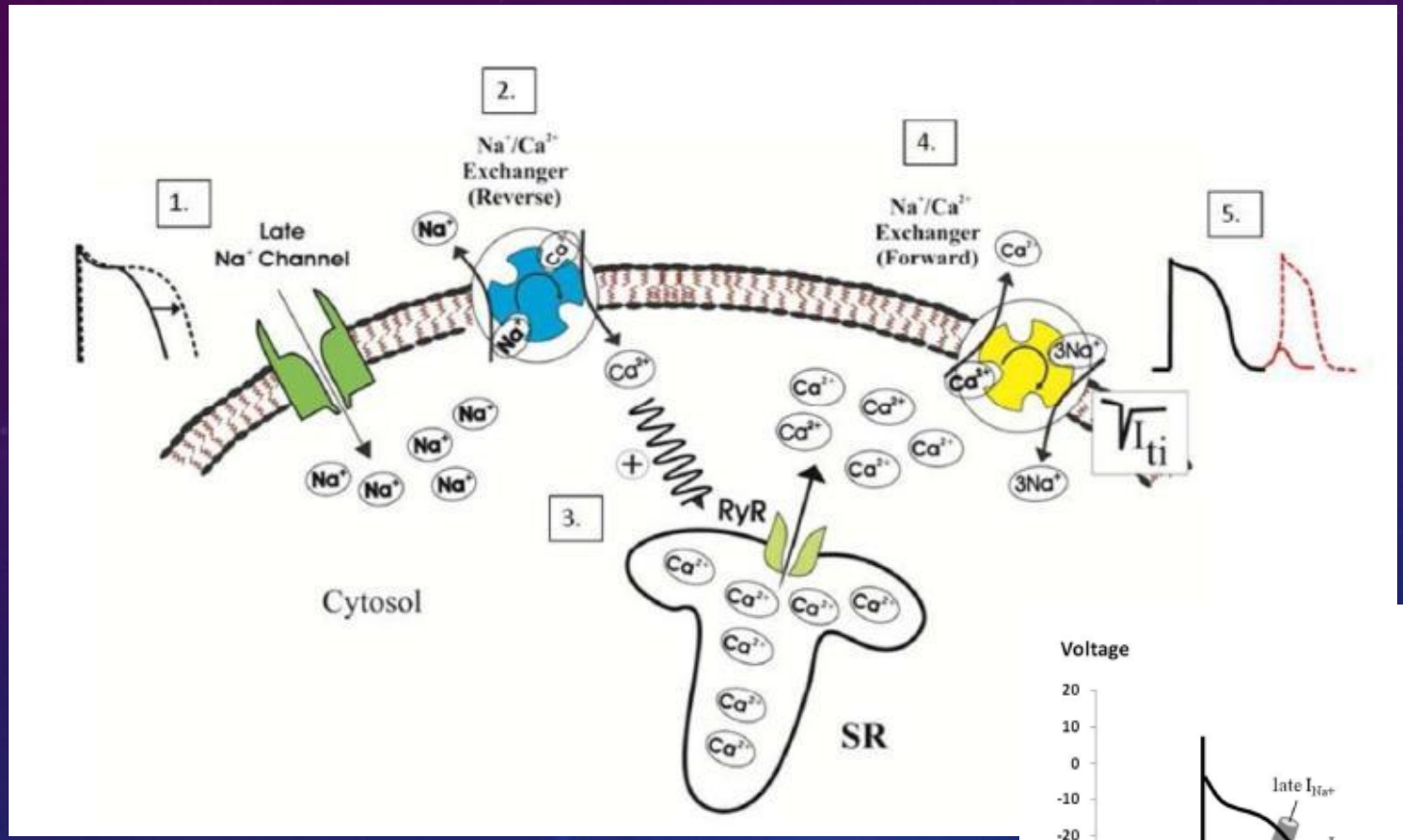
Patients with VT ≥ 8 beats

HR 0.36 (95% CI 0.10, 1.27); p=0.097



Wang W et al. *Circulation* 120(18): S661, 2562, 2009
Modified from Scirica B et al, Circulation 122:455, 2010

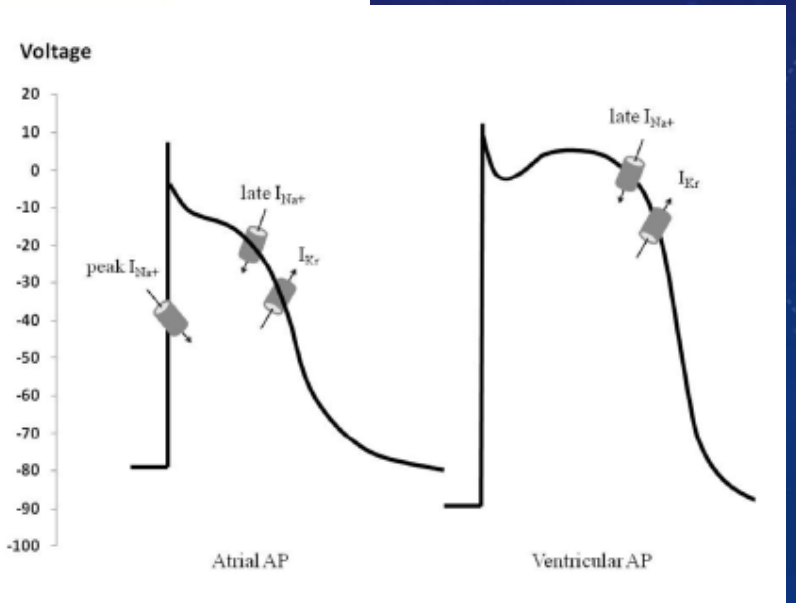
Types of ionic currents significantly inhibited by therapeutic concentrations of ranolazine in atrial and ventricular cells, and implication of late sodium channel activation in arrhythmogenesis



Ηλεκτρική Δυσλειτουργία
Αρρυθμίες

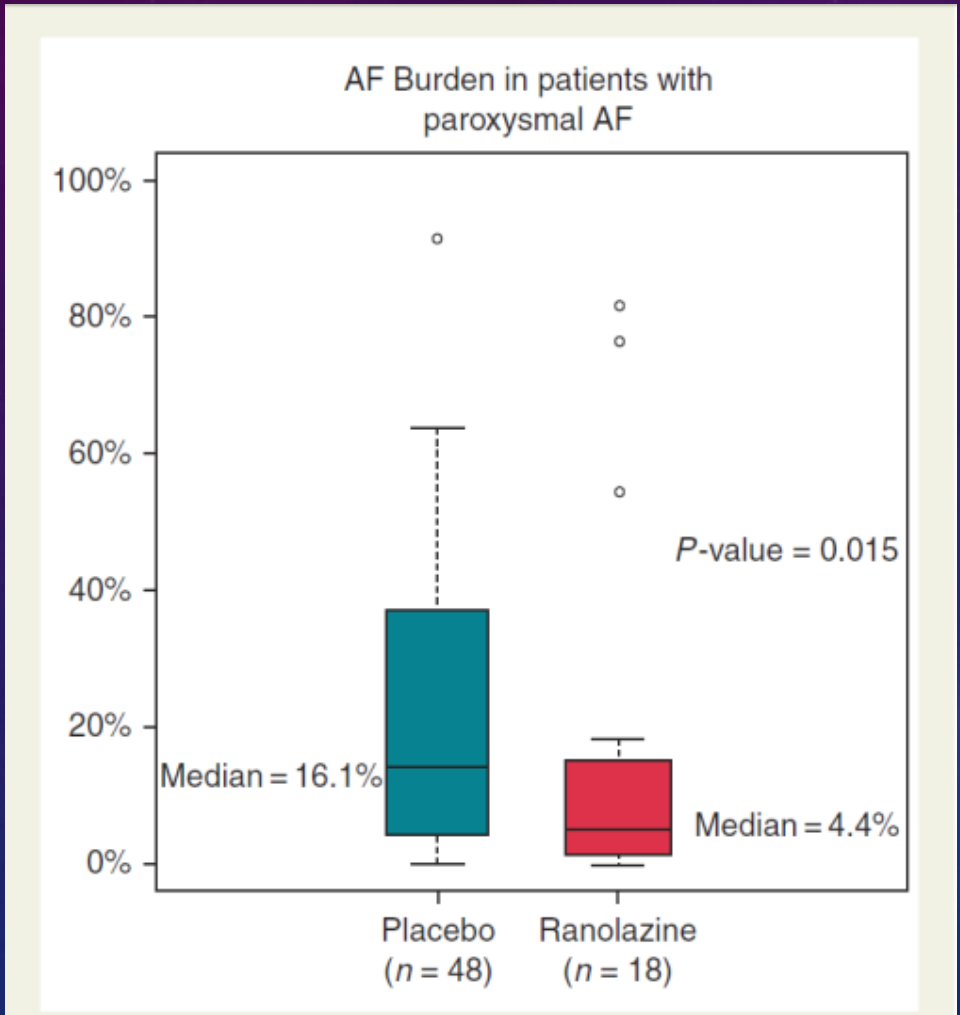
Μηχανική Δυσλειτουργία
↑ Διαστολική Τάση
↓ Συσταλτικότητα

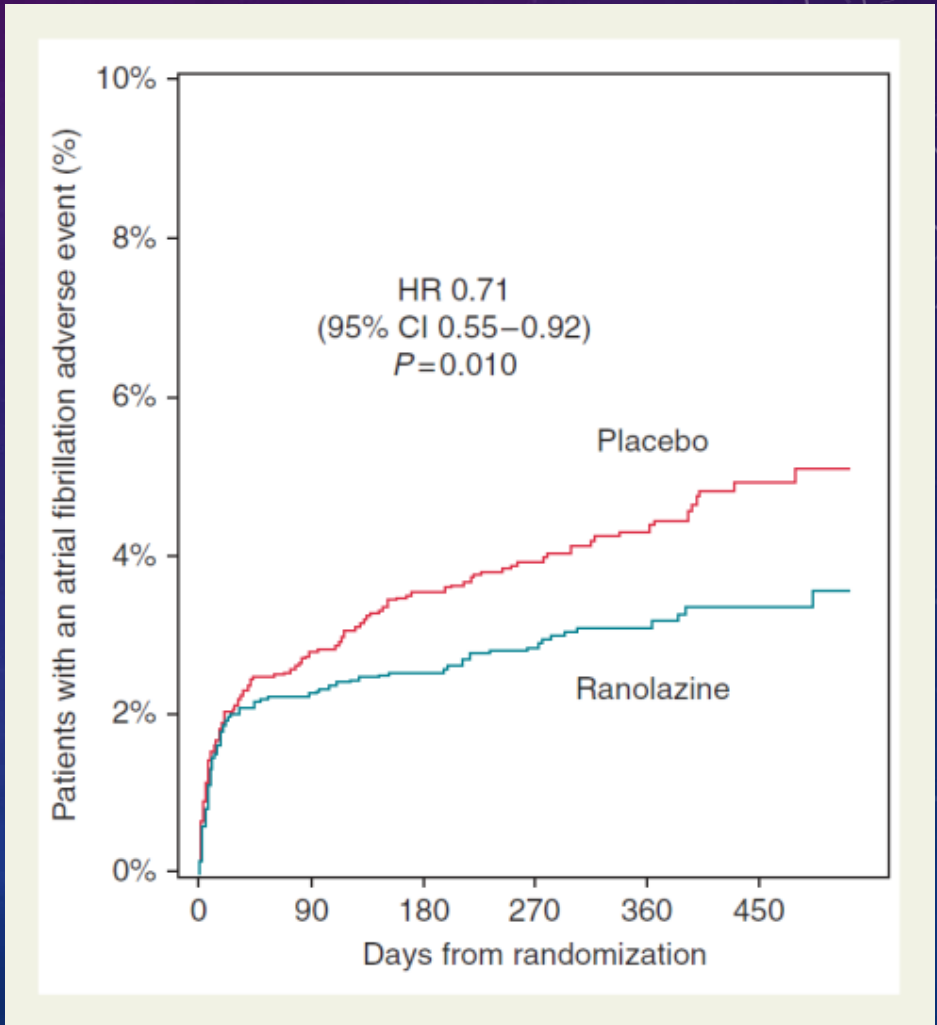
Προσφορά & Ζήτηση O_2
↑ Κατανάλωση ATP
↓ Σχηματισμός ATP



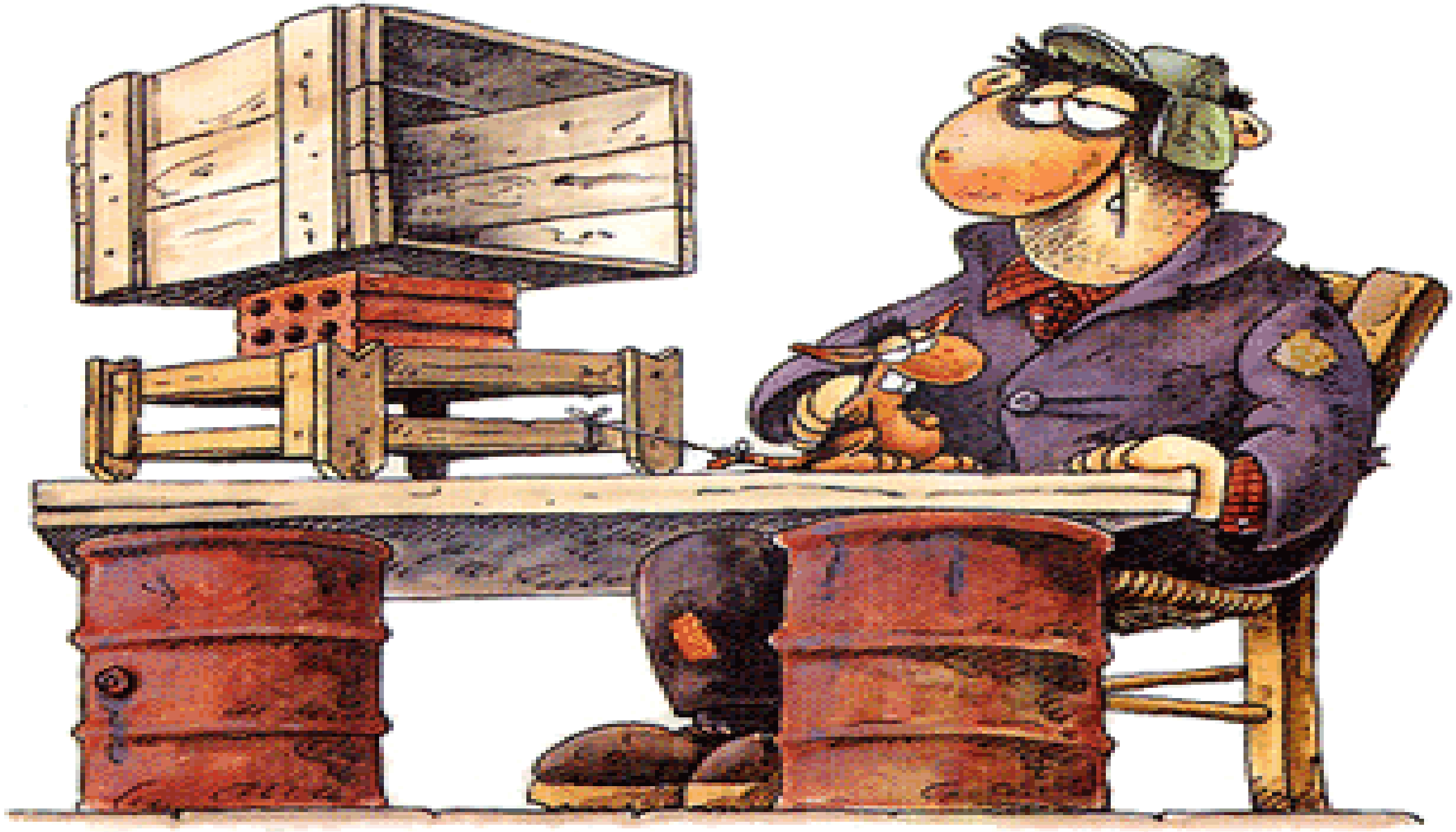
Tzeis S, Andrikopoulos G. Antiarrhythmic properties of ranolazine -from bench to bedside. Expert Opin Investig Drugs. 2012 Aug 23.

Effect of ranolazine on atrial fibrillation in patients with non-ST elevation acute coronary syndromes: observations from the MERLIN-TIMI 36 trial





Scirica B., et al. Europace 2014 Sep 10. pii: euu217. [Epub ahead of print]



New Antiarrhythmic Drugs and Formulations

- **Inhaled flecainide** – a new formulation
- **Etripamil** – intranasal administration
- **SK channel inhibitors** – several molecules
- **Sulcardine** – a multichannel blocker

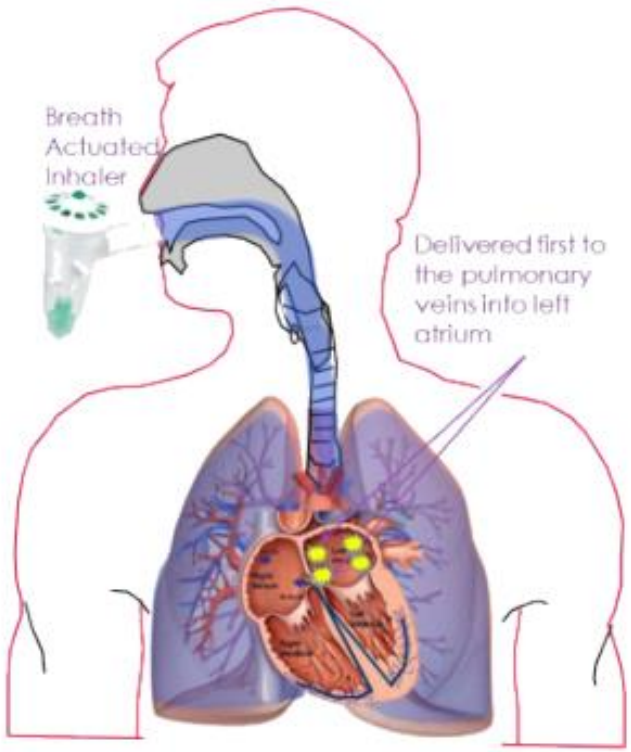
- **Bucindolol** – for the prevention of AF in heart failure
- **Doxapram** – a TASK 1 (K2P 3.1) inhibitor
- **Botulinum toxin** – for epicardial fat pad injection

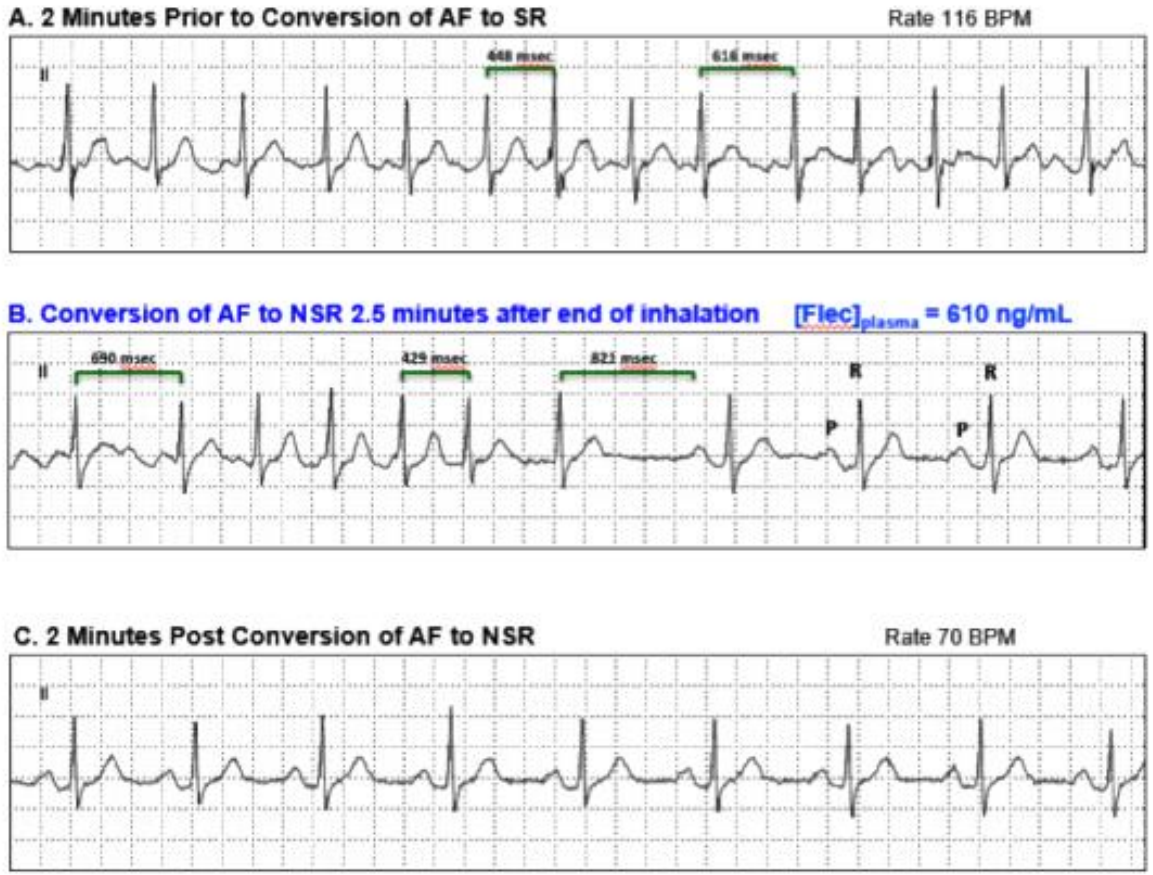
AJC St George's University of London

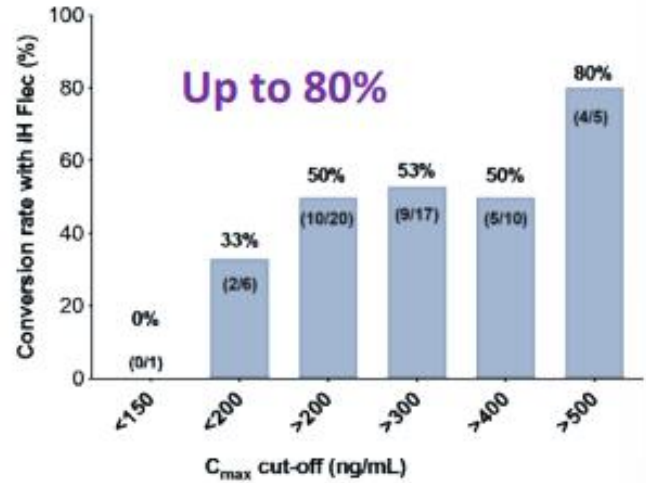
(As presented by john Camm at EHRA 2022)

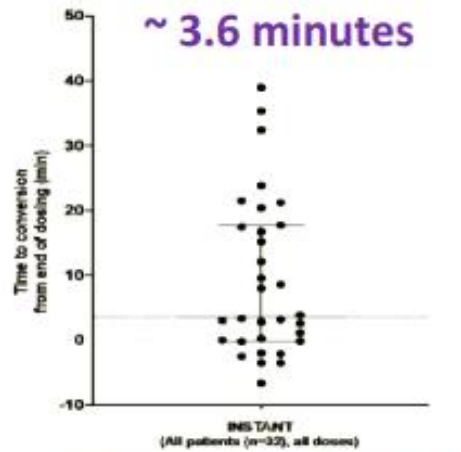
Inhaled Flecainide: INSTANT Study

InRhythm









Median time to conversion: 3.6 minutes after inhalation

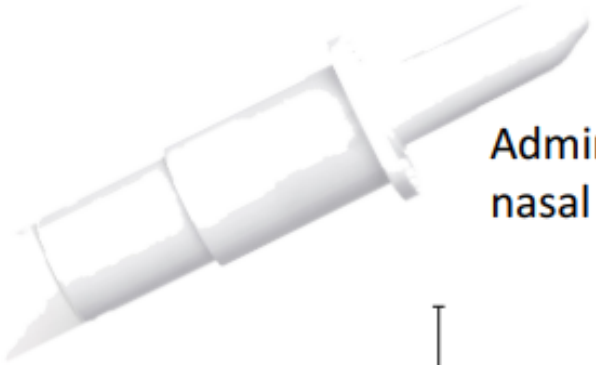
AJC St George's University of London

Crijns H, et al. American Heart Association Scientific Sessions 2020

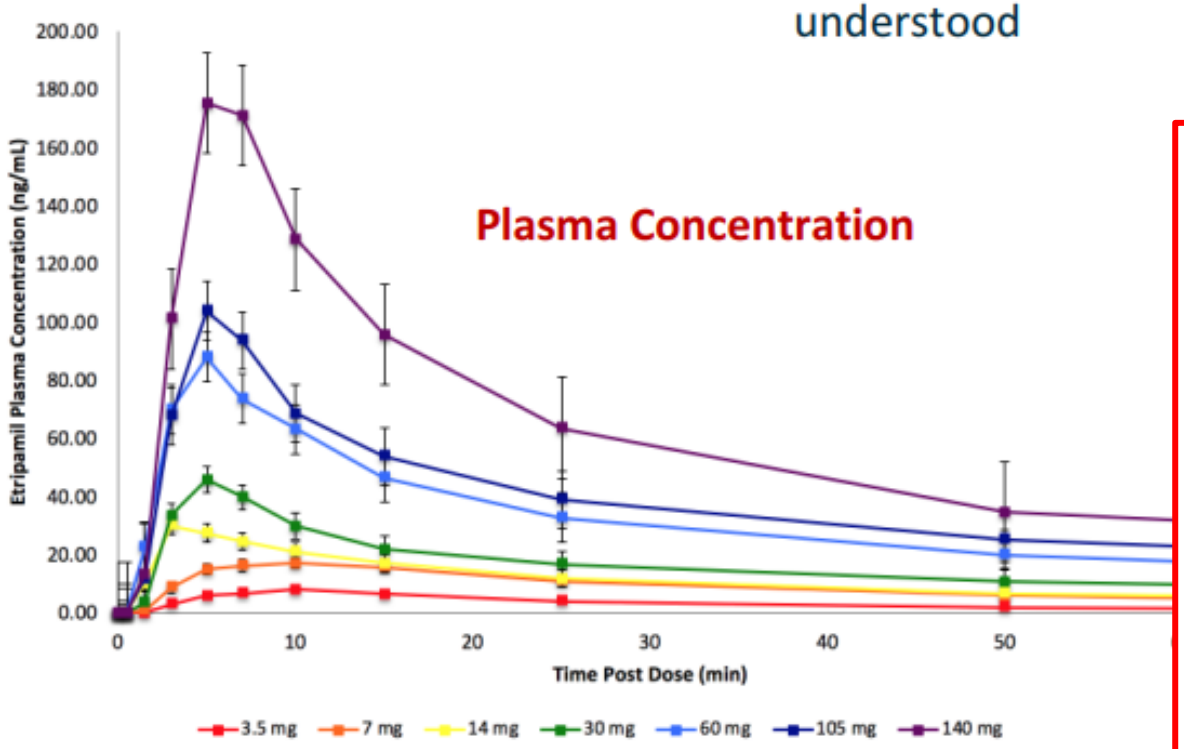
Etripamil: PK/PD

- Novel short-acting calcium channel antagonist
- Rapidly metabolized by blood esterases

- Known target: L-type calcium channels
- Mechanism of action on cardiac tissue very well understood



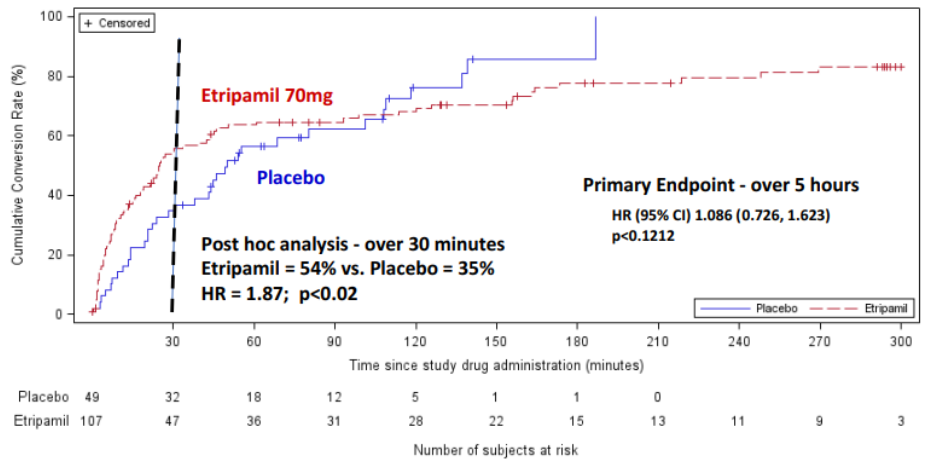
Administered by nasal insufflation



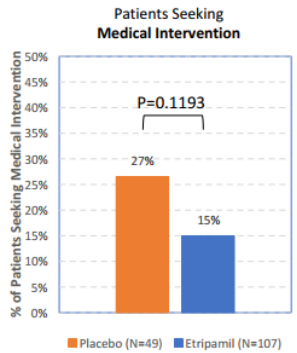
Data on file Milestone Pharmaceuticals

ETRIPAMIL NODE-301
Kaplan-Meier Plot of Conversion up to Hour 5

Pre-specified Primary Endpoint

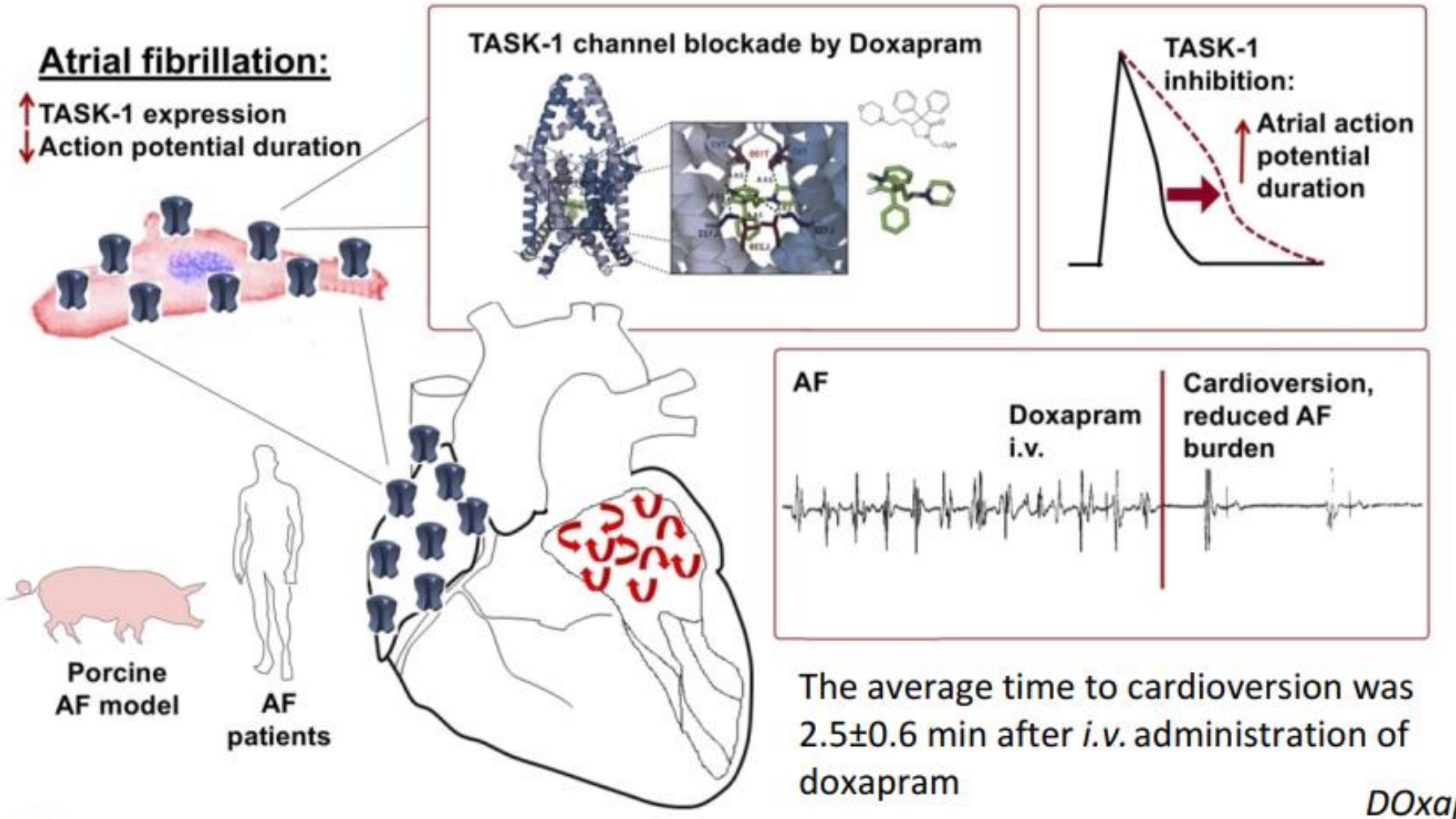


Key secondary Endpoint



Stambler B. et al. 2021 Oral Presentation

Doxapram – TASK-1 (K2P3.1) Inhibitor



The average time to cardioversion was 2.5±0.6 min after *i.v.* administration of doxapram

- Doxapram (a respiratory stimulant) is a TASK-1 inhibitor
- Preclinical pilot in porcine model successful for cardioversion of acute AF and rhythm control of paroxysmal and persistent AF
- DOCTOS Trial underway

DOxapram Conversion TO Sinus rhythm
EudraCT No: 2018-002979-17

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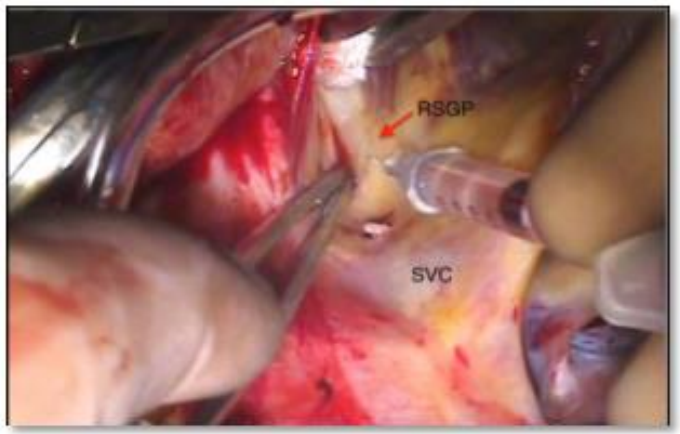
Wiedman F et al. Cardiovascular Research, cvab177, https://doi.org/10.1093/cvr/cvab177

Prevention of Post-Operative AF - Botulinum Toxin A

Botulinum toxin blocks the exocytotic release of acetylcholine stored in synaptic vesicles and thus interferes with cholinergic neurotransmission

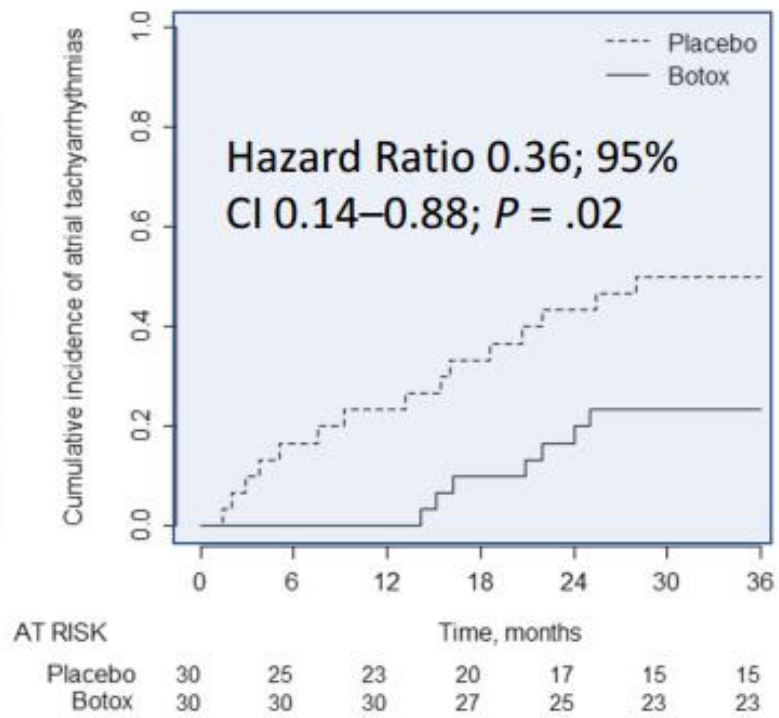
Prospective, randomized, double-blind study

NCT 01842529

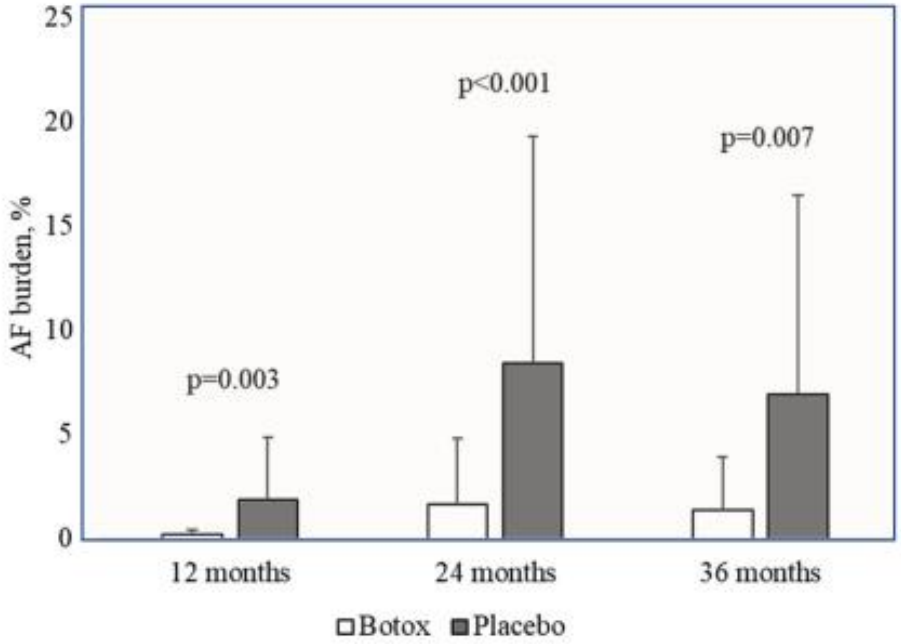


Injection of botulinum toxin into epicardial fat pads containing ganglionated plexuses

Incidence of AF



Burden of AF



NeuOtoxin for the PreVention of Post-Operative AF
NOVA trial RCT for POAF almost complete

Piccini JP, et al. Am Heart J. 2022

Romanov A et al. Heart Rhythm. 2019; 16:172-7

Effects of ANTIarrhythmics on Atrial High Rate Episodes and progression to clinical AF - ANTI-AHRE study

Stefan Simović, Jane Taleski, Željko Todorović and Bra
Department of Internal Medicine, Faculty of Medical Sciences, Univers
Clinic for Cardiology, University Clinical Center Kragujevac, Serbia

16/Apr/2023

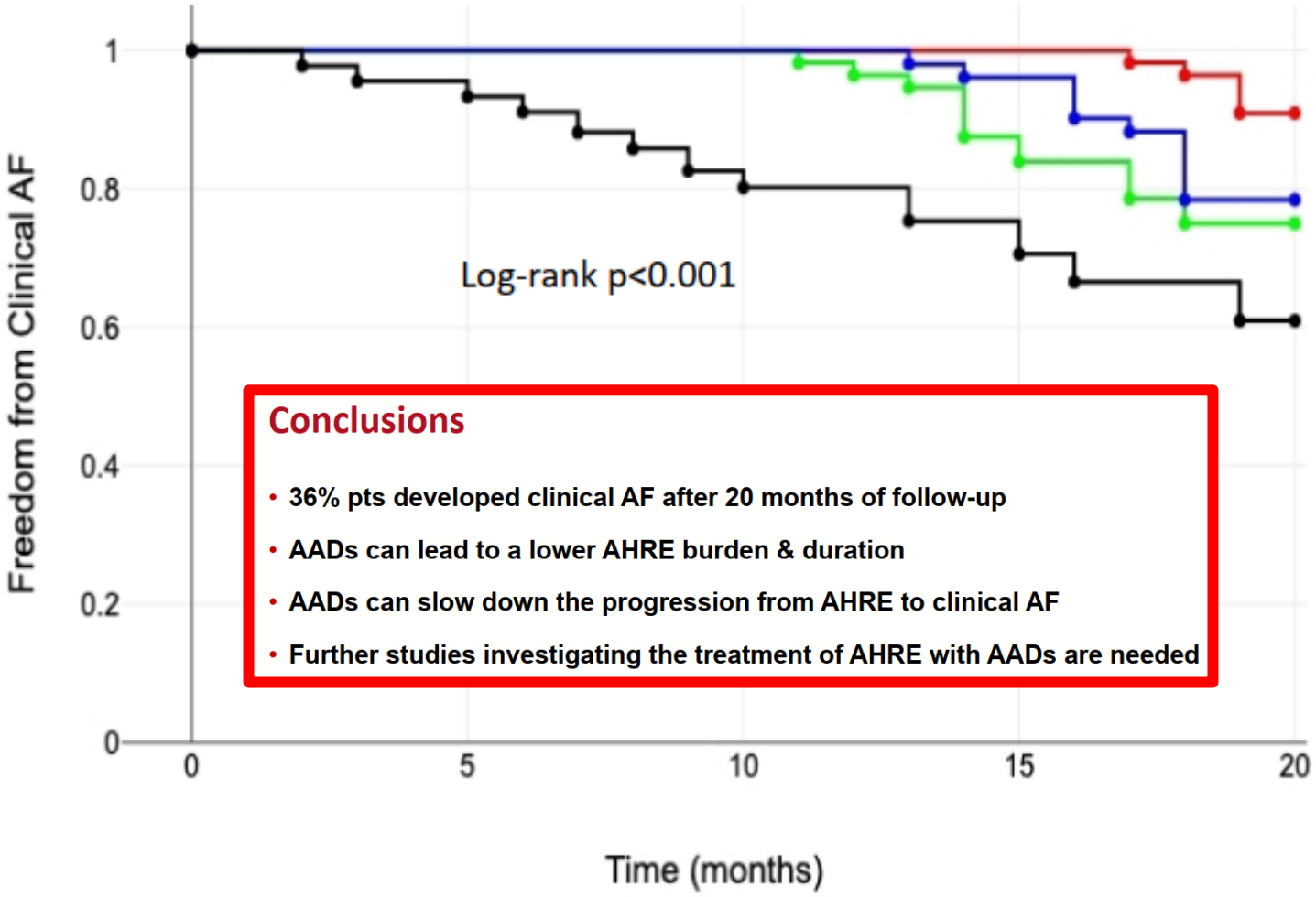
EHRA2023

Materials & Methods

- 307 pts with AHRE ≥ 24h detected by dual-chamber pacemakers
- Without a previous diagnosis of AF & treatment with AADs & BB
- Nominal settings for AHRE detection programmed to 200 bpm
- Randomized to the Intervention (n=169) and Control Group (n=138)
  - Intervention Group received AADs treatment:
    - Ic antiarrhythmics (n=54),
    - beta-blockers (n=58),
    - amiodarone (n=57).
- The primary endpoint was progression to clinical AF & the secondary endpoint was AHRE burden.

EHRA2023

Results



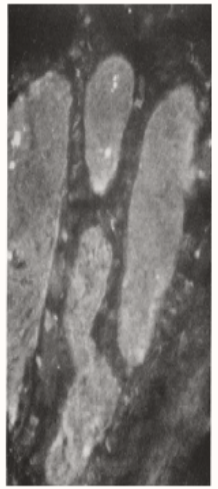
Conclusions

- **36% pts developed clinical AF after 20 months of follow-up**
- **AADs can lead to a lower AHRE burden & duration**
- **AADs can slow down the progression from AHRE to clinical AF**
- **Further studies investigating the treatment of AHRE with AADs are needed**

	HR (95% CI)	p-value
Ic antiarrhythmics	1.43 (0.86-4.02)	0.027
Beta blockers	1.79 (0.99-3.24)	0.055
Amiodarone	0.18 (0.07-0.45)	<0.001

	Ctrl	Ic	BB	Amio	p-value
Progression to clinical AF (pts)	50 (36.2%)	11 (20.4%)	15 (25.9%)	5 (8.8%)	<0.001
Average time of clinical AF dg (months)*	15.9	17.7	17.2	19	0.025

*for patients who developed clinical AF

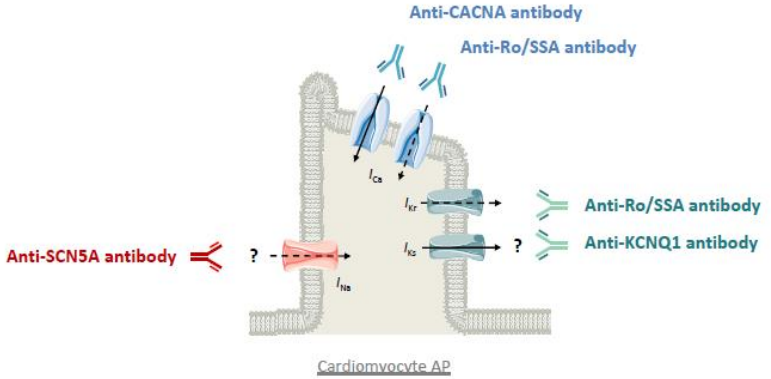


Clin. exp. Immunol. (1976) **23**, 1–8.

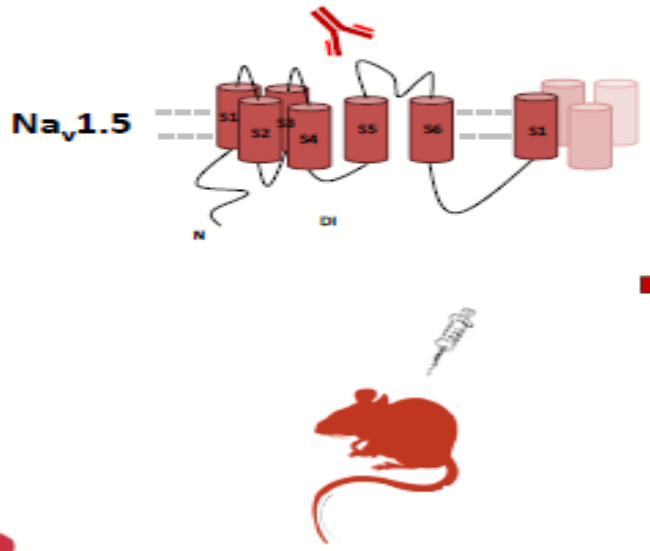
Autoantibodies to cardiac conducting tissue and their characterization by immunofluorescence

A. FAIRFAX & DEBORAH DONIACH *Department of Immunology, The Middlesex Hospital Medical School, London*

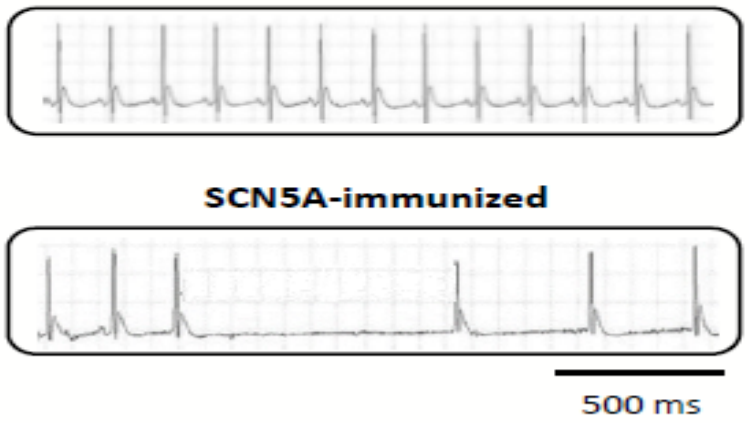
Antibodies targeting cardiac ion channels



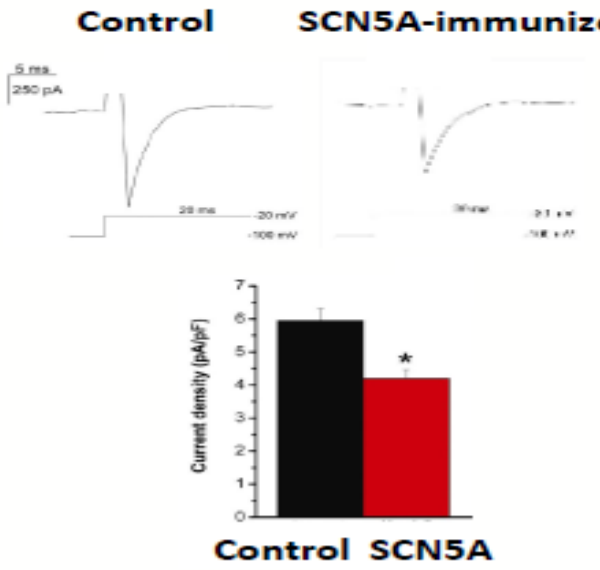
Anti-SCN5A antibody



ECG in rats

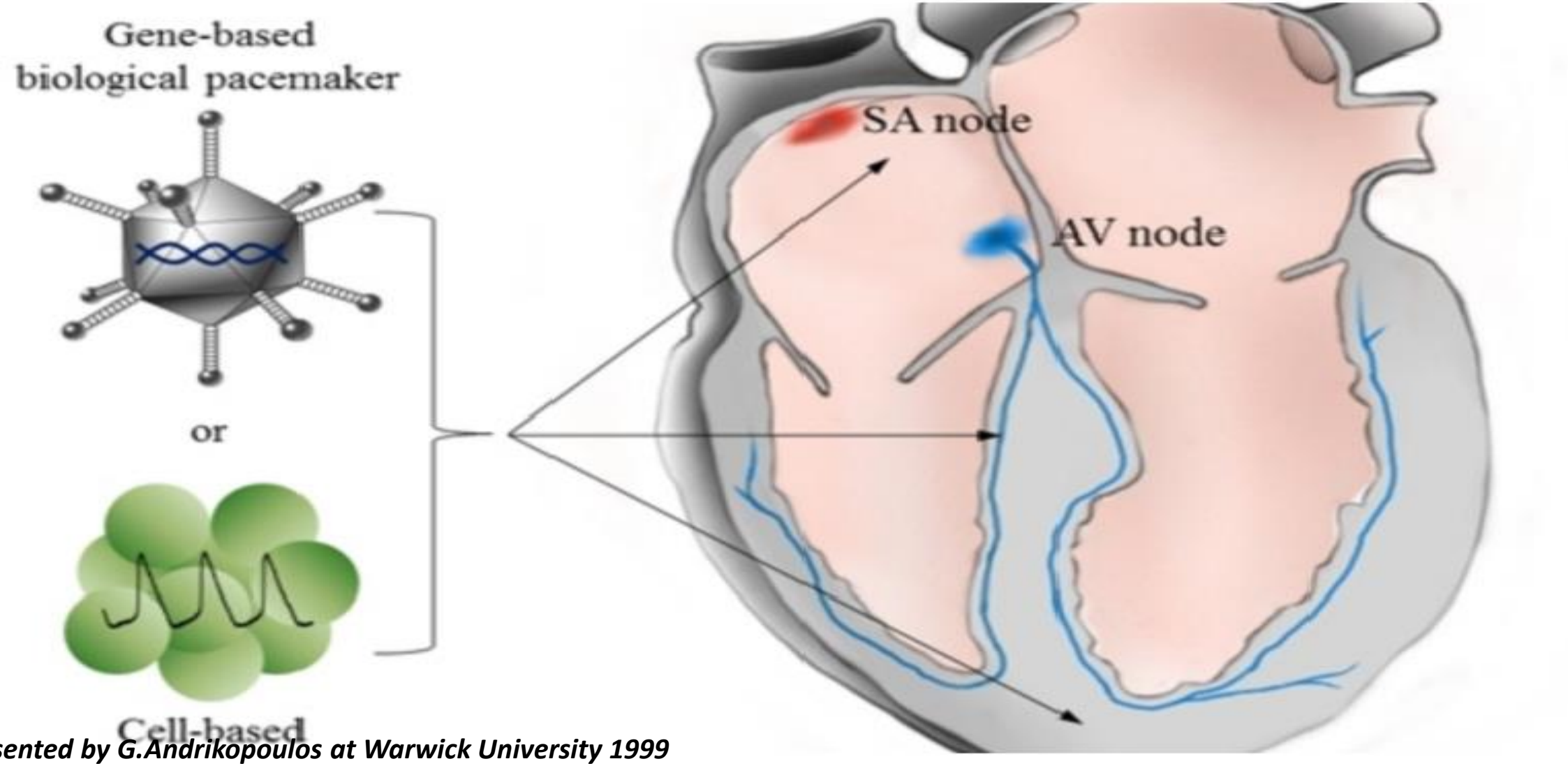


I_{Na} in rat cardiomyocytes with serum from



Korkmaz S et al. *J Am Coll Cardiol.* 2013

Biological Pace Makers

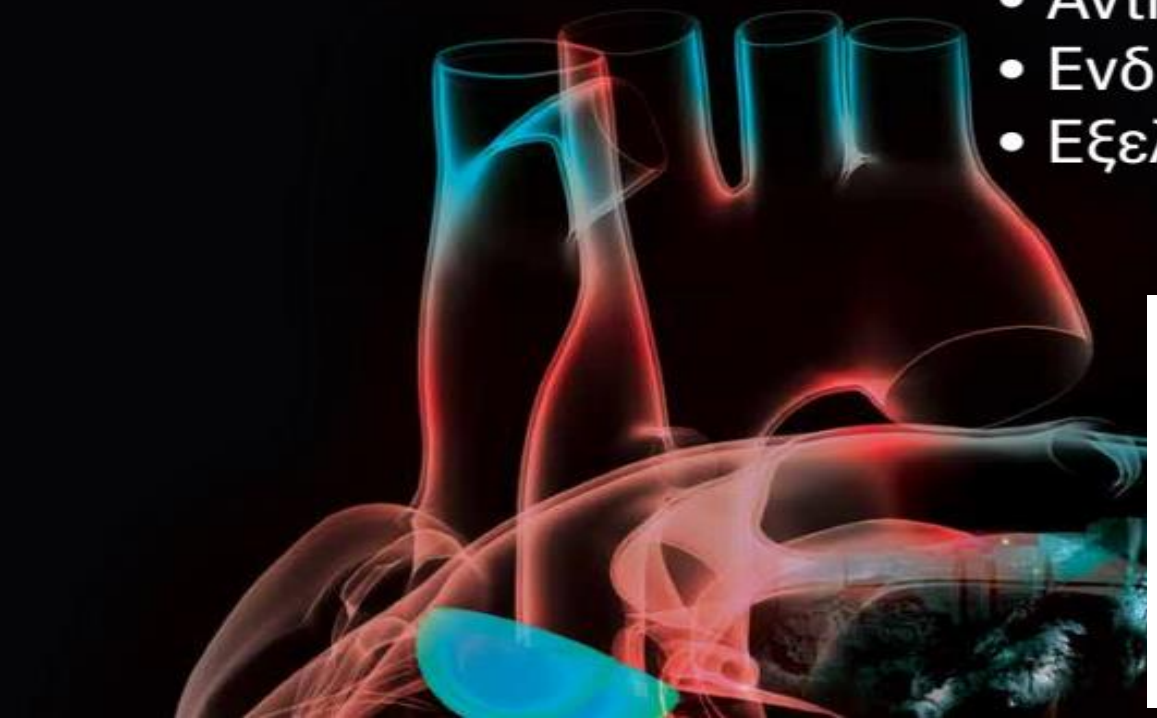


As presented by G.Andrikopoulos at Warwick University 1999

9ο WORKSHOP

Αρρυθμιών & Βηματοδότησης

- Ενδιαφέροντα ηλεκτροκαρδιογραφήματα
- Αντιπαραθέσεις
- Ενδιαφέροντα περιστατικά
- Εξελίξεις στην αντιμετώπιση των αρρυθμιών



SAVE THE DATE

9ο Workshop Αρρυθμιών & Βηματοδότησης

8 – 10 Δεκεμβρίου 2023 | Divani Caravel, Αθήνα