Introduction to Clinical Physiology

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Lectures of Clinical Physiology are available on the internet:

https://aok.unideb.hu/en/clinfiz

Lectures

Semester II: 14 lectures

Circulation (9 lectures) Respiratory system (2 lectures) Nutrition (1 lecture) Nervous system (2 lectures)

(9th week: self control) (14th week: self control)

Seminars

Seminars are compulsory!

Semester II (week 1-14):

Electrocardiography (10th week: self control)

Respiratory functions Echocardiography Cardiac catheterisation

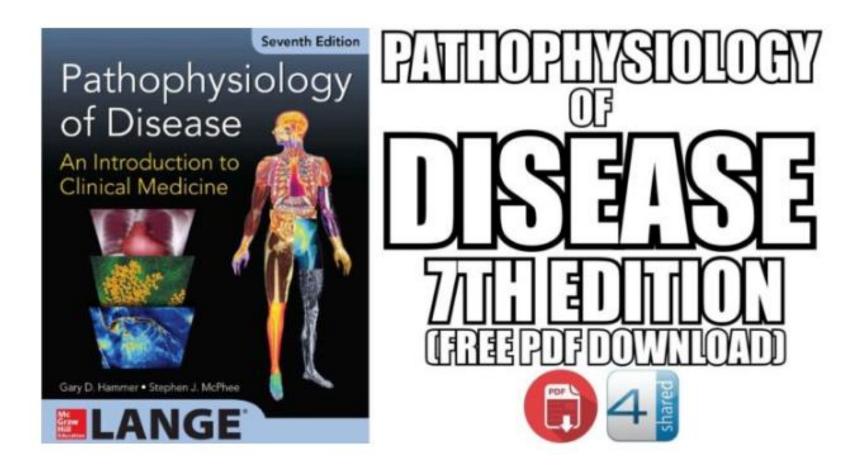
Exam at the end of semester II

"A" exam: written "B" exam: written "C" exam: oral

Exam results in 2024:

	final:	2nd self- control:
English program gen. med.:	2,57	fail: 20,3 %
Hungarian program gen. Med.:	3,00	fail: 12,1 %

Recommended textbooks

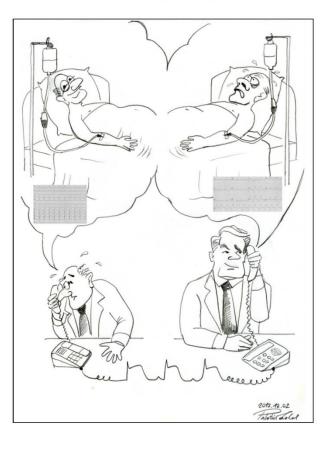






Written by: László Balogh M.D.

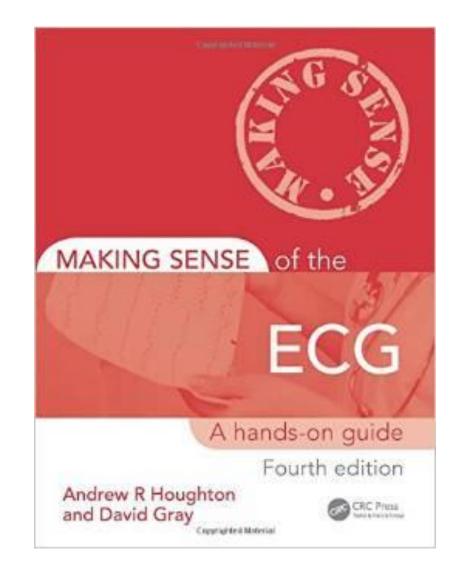
University of Debrecen Clinical Center Institute of Cardiology and Cardiac Surgery



https://aok.unideb.hu/en/educational-materialsclinical-physiology-medical-program

Making Sense of the ECG (4th edition, 2014)

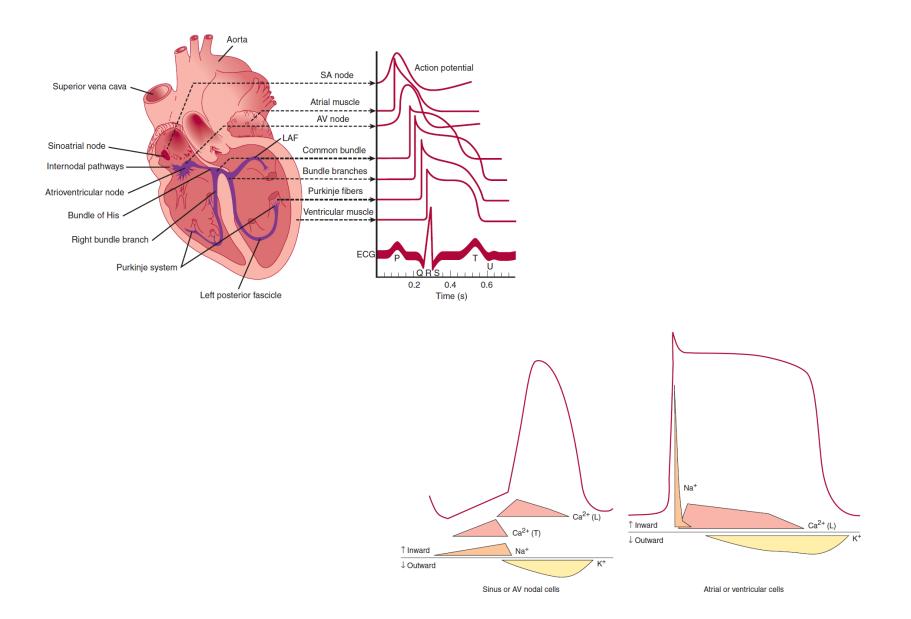
Date of publication: 2014 Publisher: CRC Press ISBN-13: 978-1444181821 ISBN-10: 1444181823



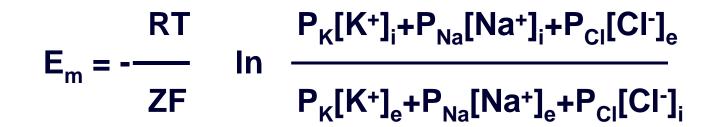
Cellular and molecular factors of pathologic cardiac excitability

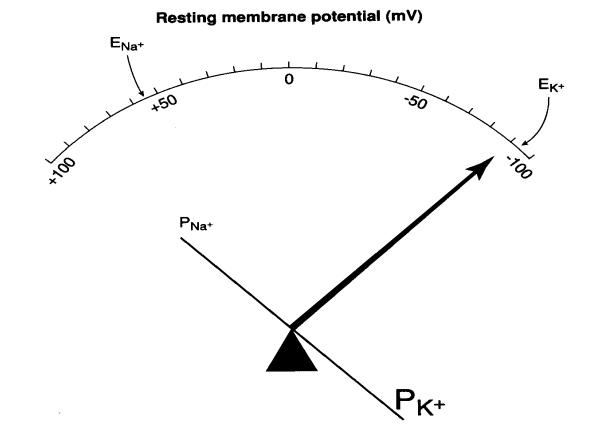
Dr. Zoltán Papp UD Faculty of Medicine Department of Cardiology Division of Clinical Physiology

Physiological background



K⁺ balance and membrane potential

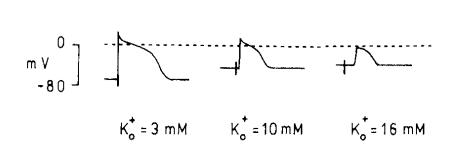




Hyperkalaemia and cardiac excitability

Depolarization

100 ms

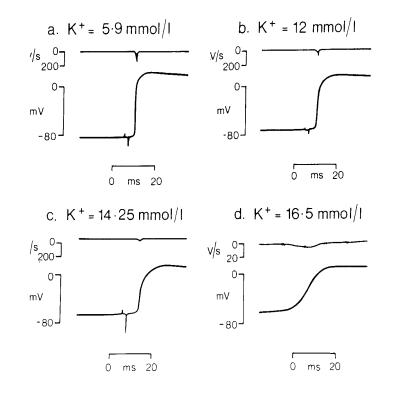


ECG alterations



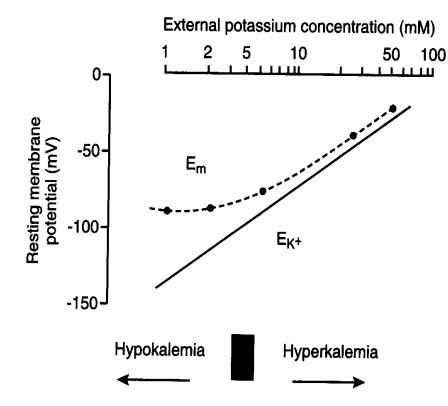
Normal level: 3.5 to 5.2 mmol/L

Na⁺ channel blockade



[K⁺]_e > 7.5 mM: cardiac arrest!

Hypokalaemia and cardiac excitability



[K⁺]_e < 2.7 mM:

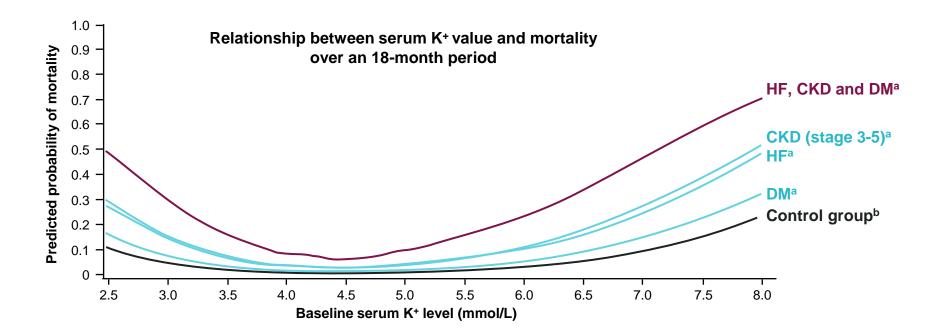
prolonged repolarization unstable resting potential ectopic activity[↑] ventricular ES ventricular tachycardia ventricular fibrillation

ECG alterations



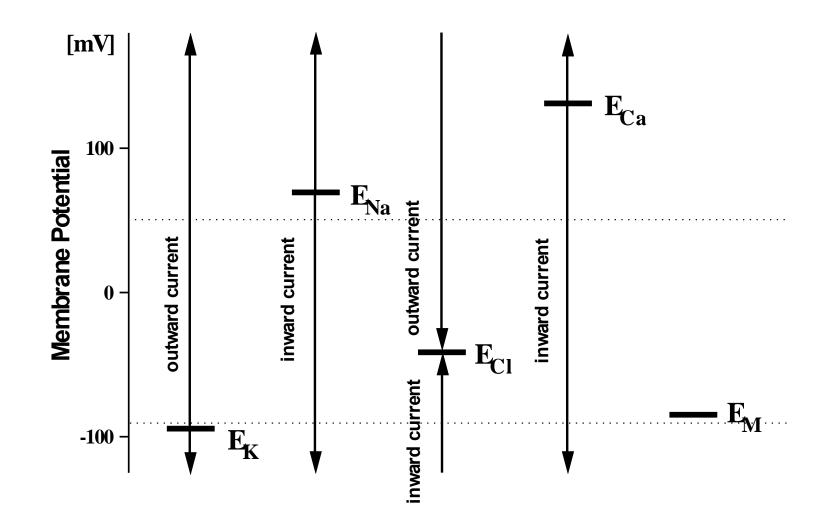
Hypo- and hyperkalemia associated mortality is higher with comorbidities

Analysis of electronic medical record data from multiple US integrated health delivery networks of 911,698 patients with ≥2 potassium measurements between 2007 and 2012

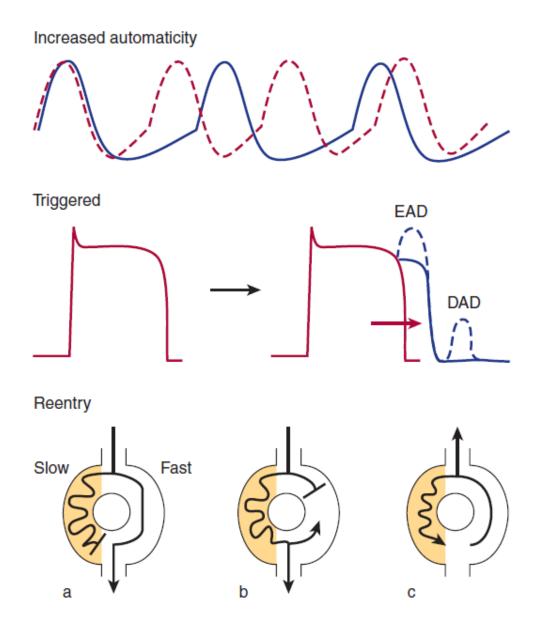


^aSignificant vs. control group; ^bControl group comprised of individuals without known HF, CKD, DM, CVD, or HTN. CKD = chronic kidney disease; CVD = cardiovascular disease; DM = diabetes mellitus; HF = heart failure; HTN = hypertension; US = United States. Collins AJ et al. *Am J Nephrol.* 2017;46:213–221.

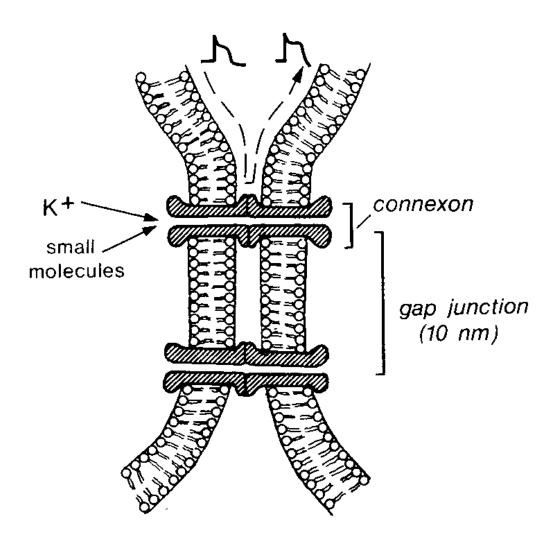
Ionic currents and membrane potential changes

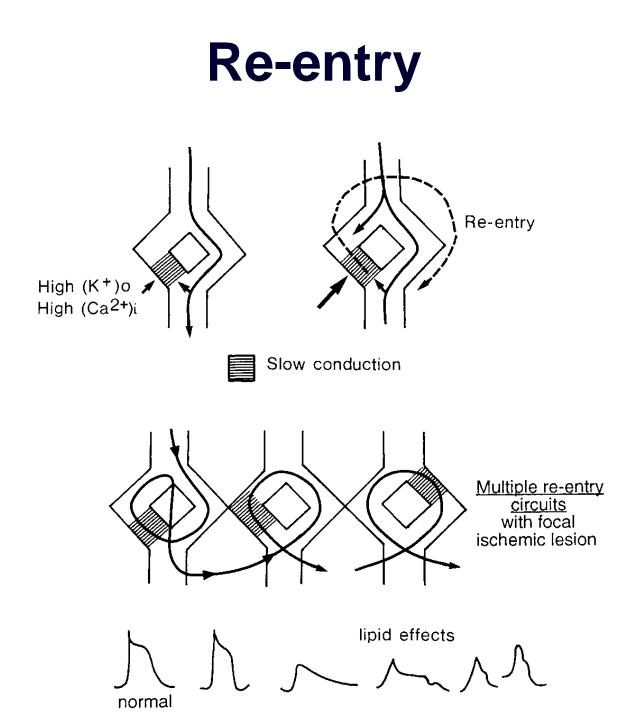


Basic arrhythmogenic mechanisms



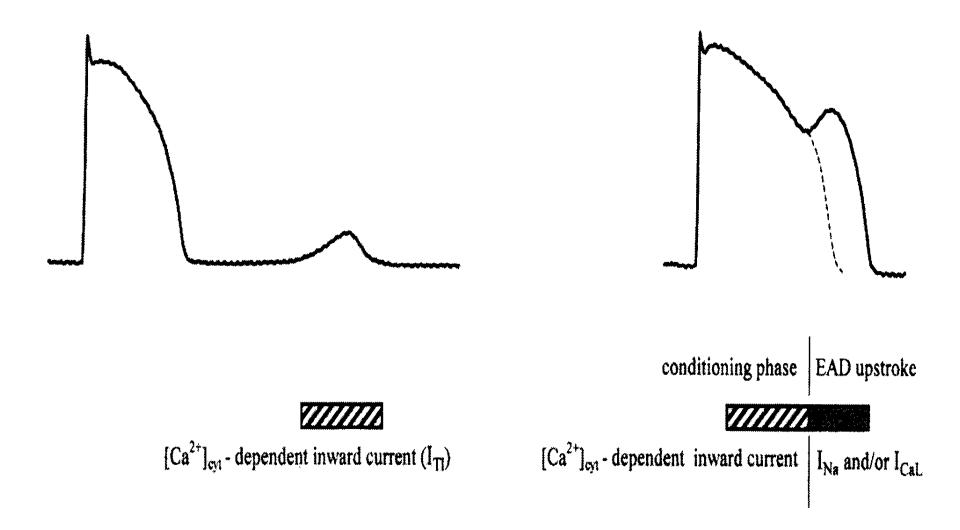
Cardiac gap junctions





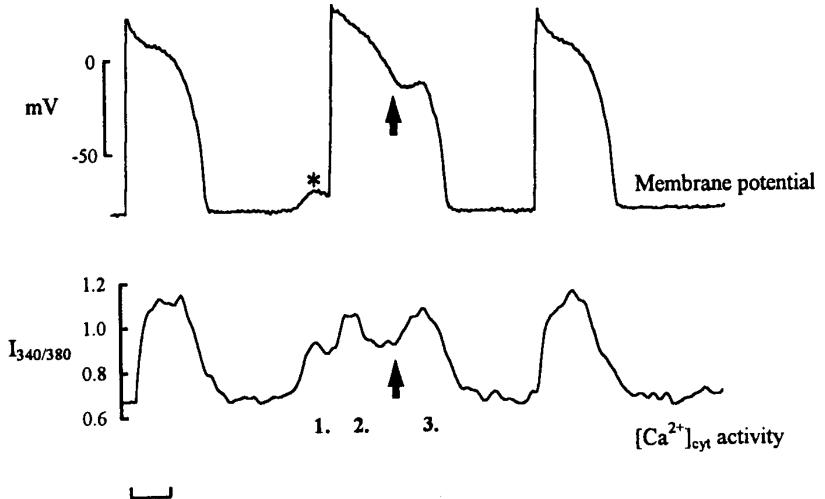
Afterdepolarizations (triggered activity)

P.G.A. Volders et al. / Cardiovascular Research 46 (2000) 376-392



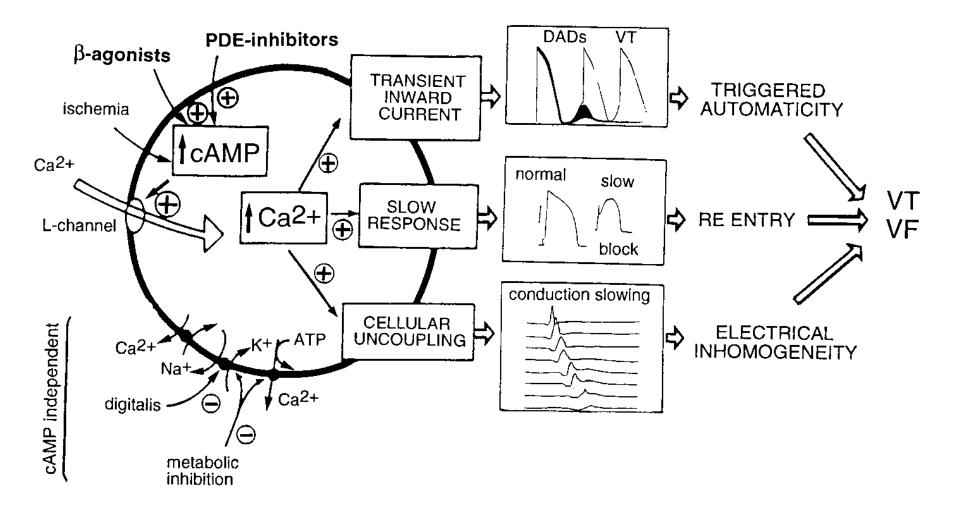
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Afterdepolarizations are triggered by spontaneous Ca²⁺ release from the SR

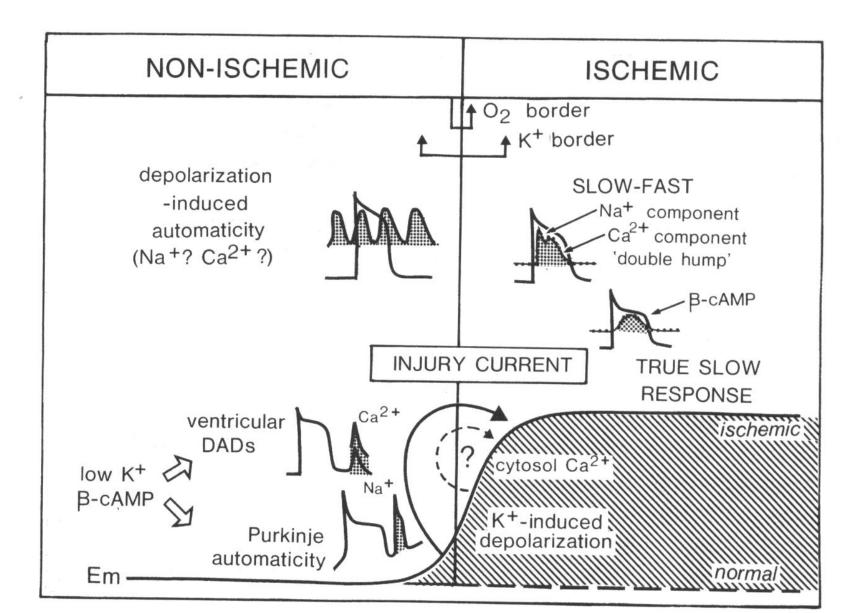


 $2\overline{00}$ ms

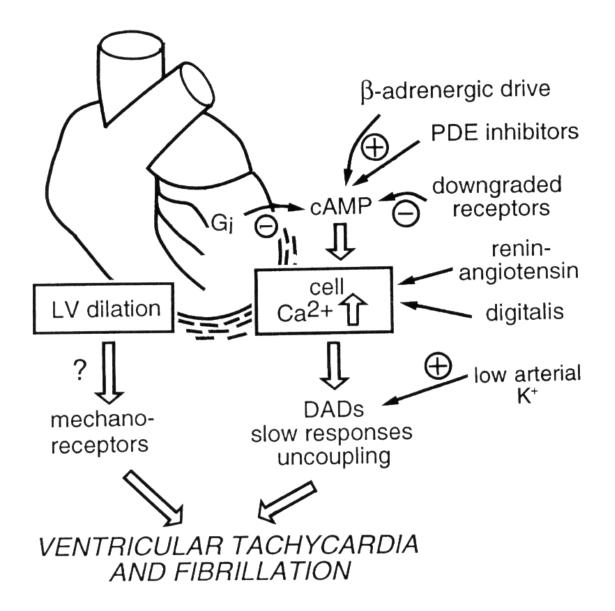
[Ca²⁺]_i –dependent arrhythmogenic mechanisms



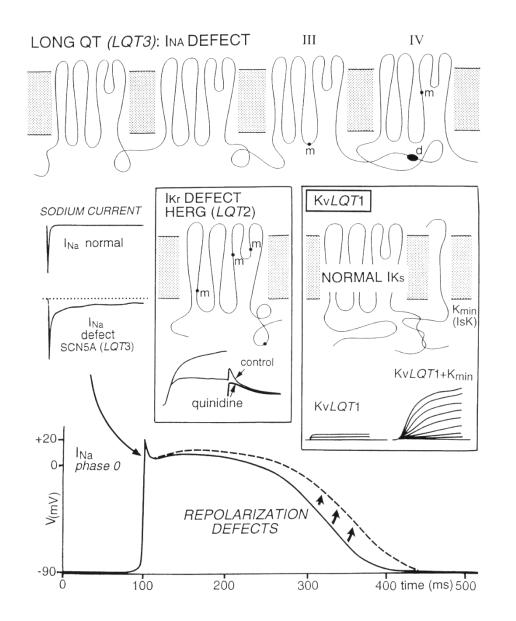
Myocardial infarction and arrhythmias



Chronic heart failure and arrhythmias



Inherited long QT syndromes



The beginning of evidence-based medicine



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ORIGINAL ARTICLE

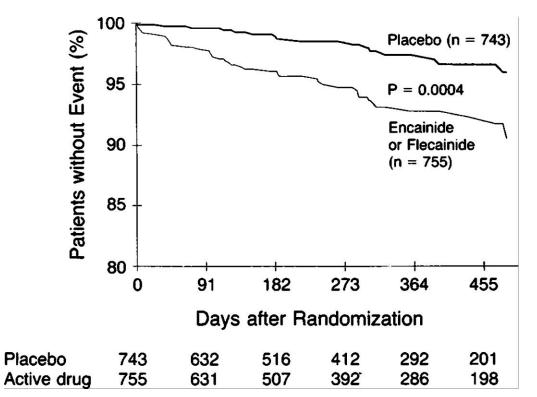
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Mortality and Morbidity in Patients Receiving Encainide, Flecainide, or Placebo — The Cardiac Arrhythmia Suppression Trial

Authors: Debra S. Echt, M.D., Philip R. Liebson, M.D., L. Brent Mitchell, M.D., Robert W. Peters, M.D., Dulce Obias-Manno, R.N., Allan H. Barker, M.D., Daniel Arensberg, M.D., **45**, and the CAST Investigators ***** Author Info & Affiliations

Published March 21, 1991 | N Engl J Med 1991;324:781-788 | DOI: 10.1056/NEJM199103213241201 VOL. 324 NO. 12 CAST = Cardiac Arrhythmia Suppression Trial

RCT = randomized clinical trial



Antiarrhyhtmic drugs

