

Neues vom ESC

in Stockholm

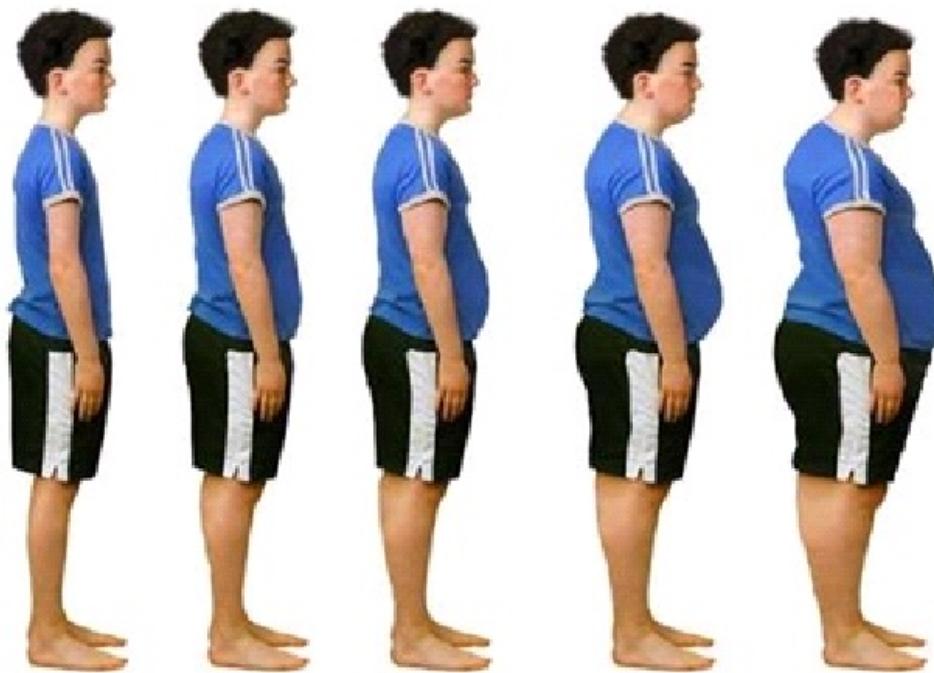
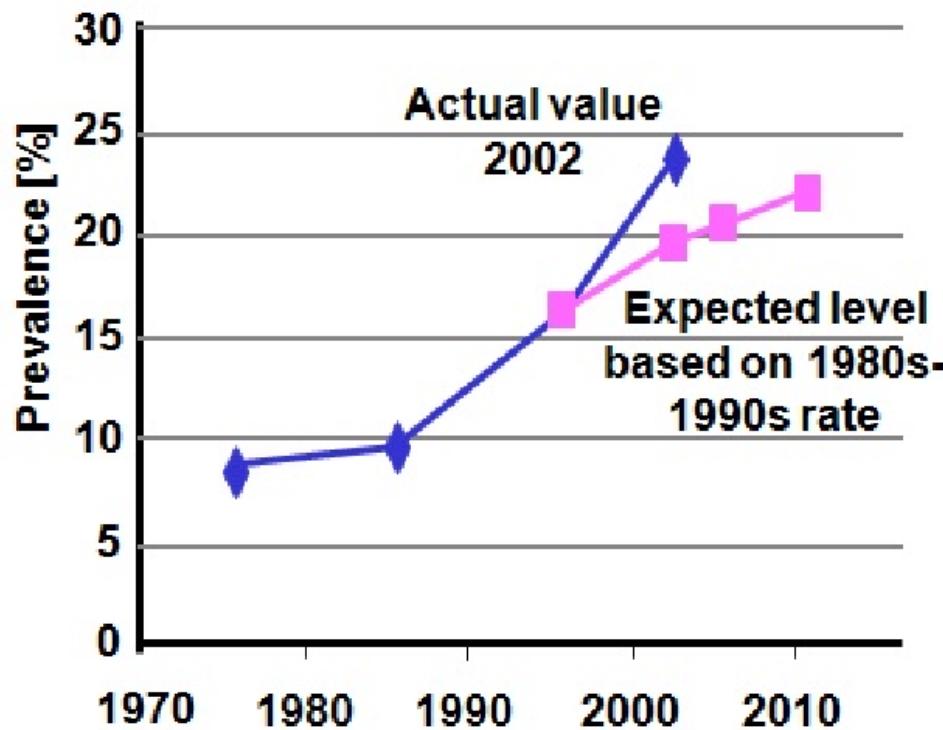
(28.09. - 01.09.2010)



Koronare
Herzerkrankung

C. Strunk-Müller

Prevalence of overweight among school children in Europe



Make them play



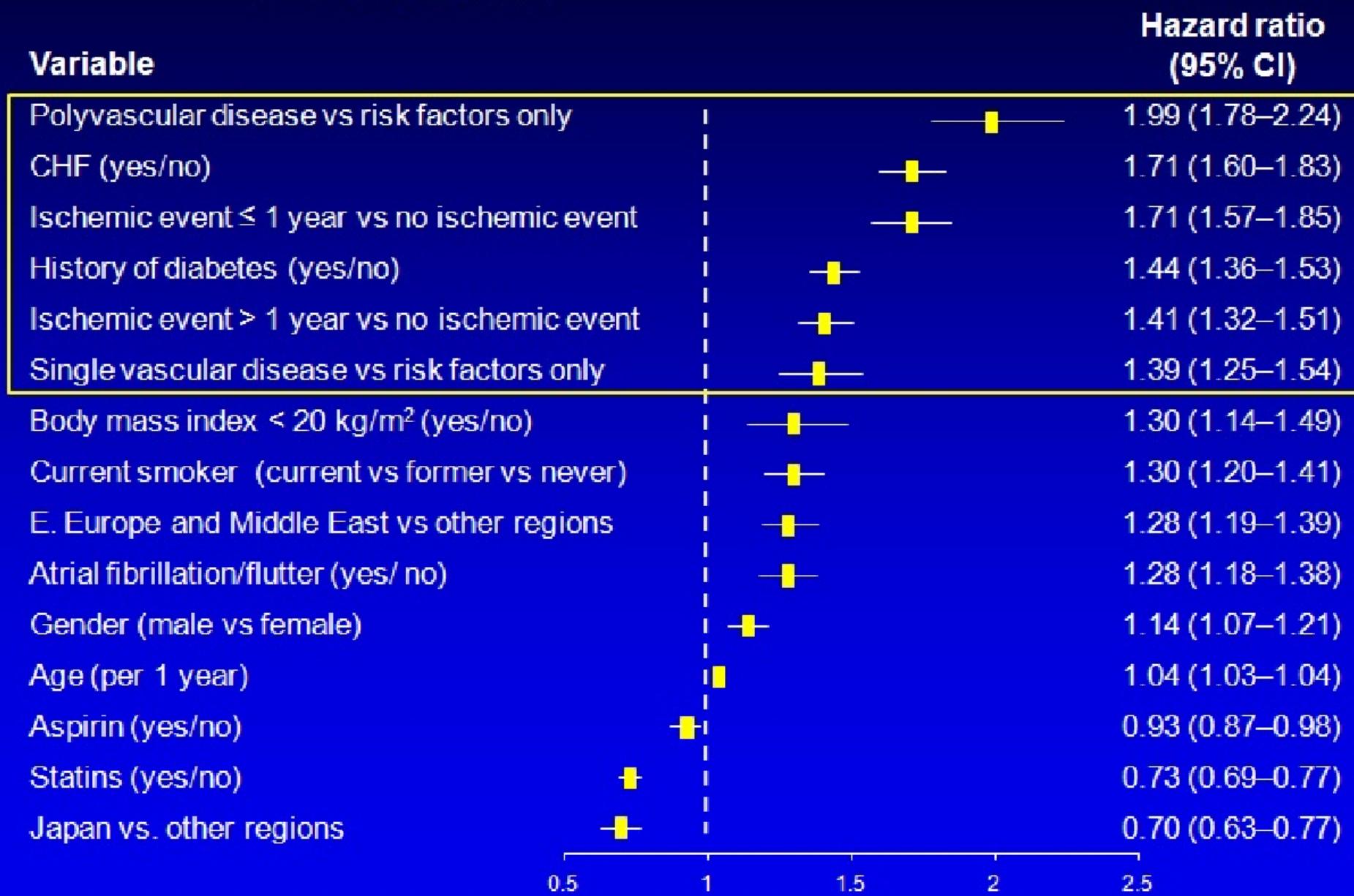
Eat less, change their mind

Intervention programmes

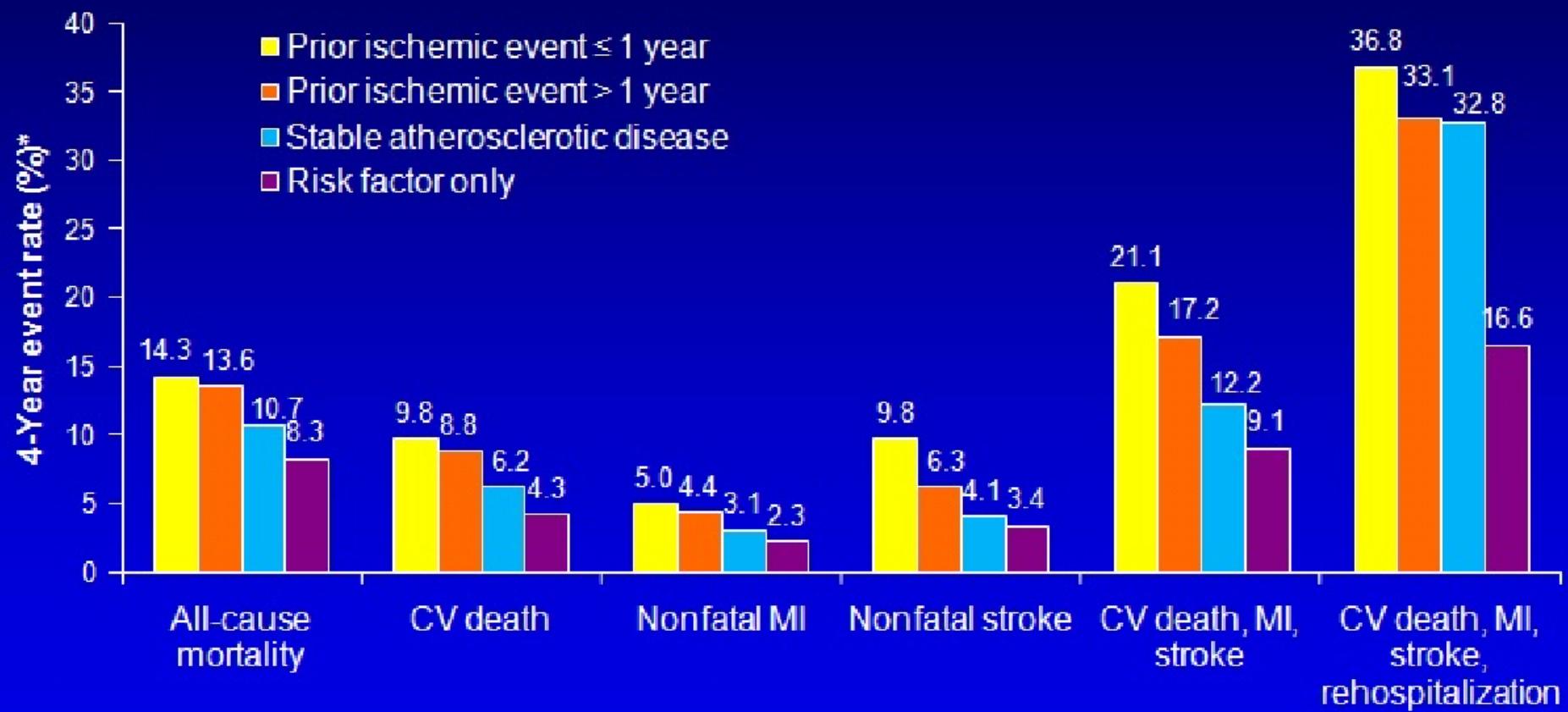
Treatment:	Physical activity Dietary habits Health education
Target group:	Family Children at risk Overweight and obese children Healthy children (Primary prevention)
Settings:	Kindergarten School / University Environment Sports clubs
Methods:	Objective parameters? Questionnaires Accelerometers Follow - Up



Multivariable predictors of CV events

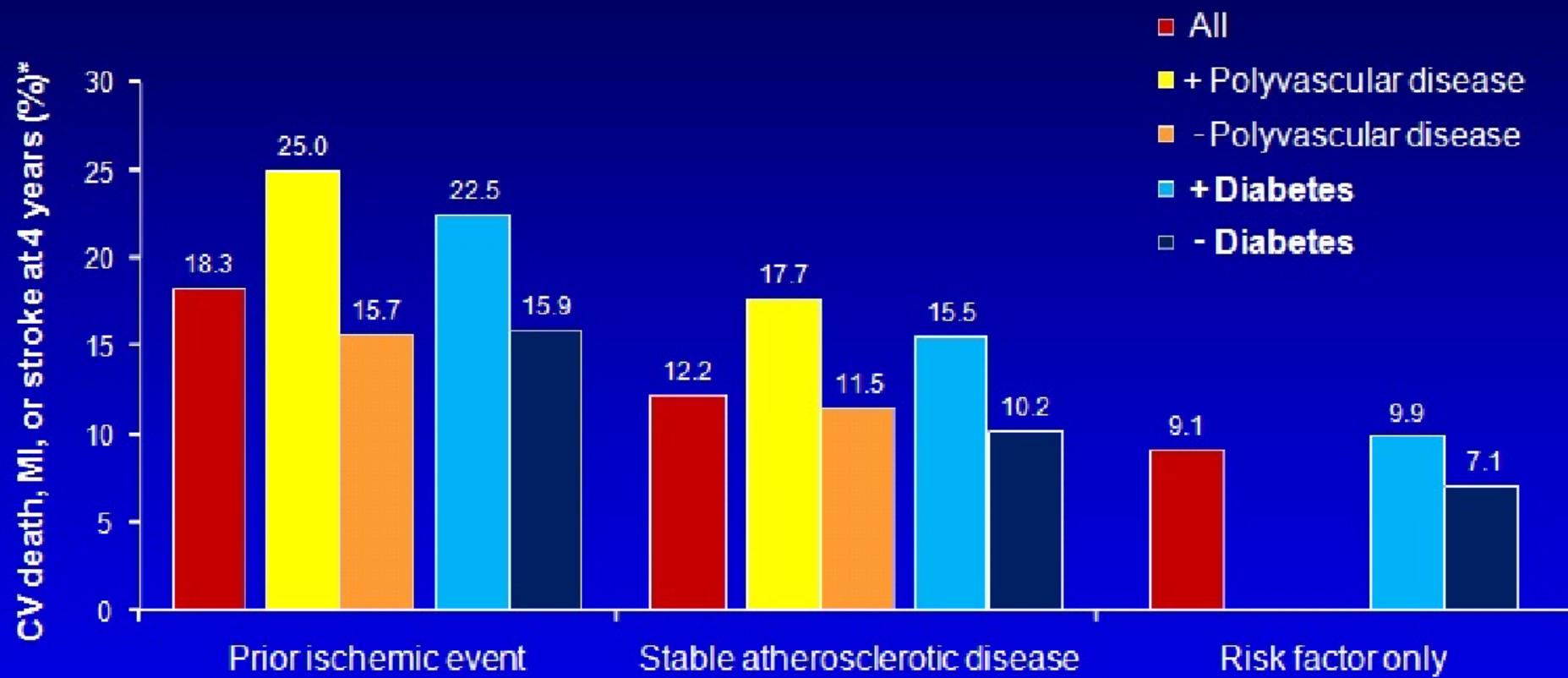


Comparative impact of ischemic event timing on 4-year event rates



*All event rates adjusted for age and gender.

Impact of polyvascular disease and diabetes on CV events at 4 years



*All event rates adjusted for age and gender.

Summary of key results

- There is a broad range of risk for stable atherothrombosis:

Patients with prior
ischemic events at
baseline



Patients with stable
atherosclerosis at
baseline



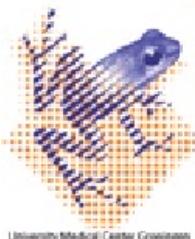
Patients with risk
factors only

- Polyvascular disease was the strongest independent predictor of future ischemic events
- In patients with prior ischemic events, an event \leq 1 year prior to enrollment was a stronger predictor for CV events (vs. $>$ 1 year)
- Diabetes increased the risk of CV events in all populations, but to a lesser extent than polyvascular disease or prior ischemic events at baseline

A Single Dose of Erythropoietin in ST-elevation Myocardial Infarction

Adriaan A. Voors, Anne M.S. Belonje, Felix Zijlstra, Hans L. Hillege, Stefan D. Anker, Riemer H.J.A. Slart, René A. Tio, Arnoud van 't Hof, J. Wouter Jukema, Hans Otto J. Peels, José P. S. Henriques, Jurriën M. ten Berg, Jeroen Vos, Wiek H. van Gilst, Dirk J. van Veldhuisen, on behalf of the HEBE III investigators

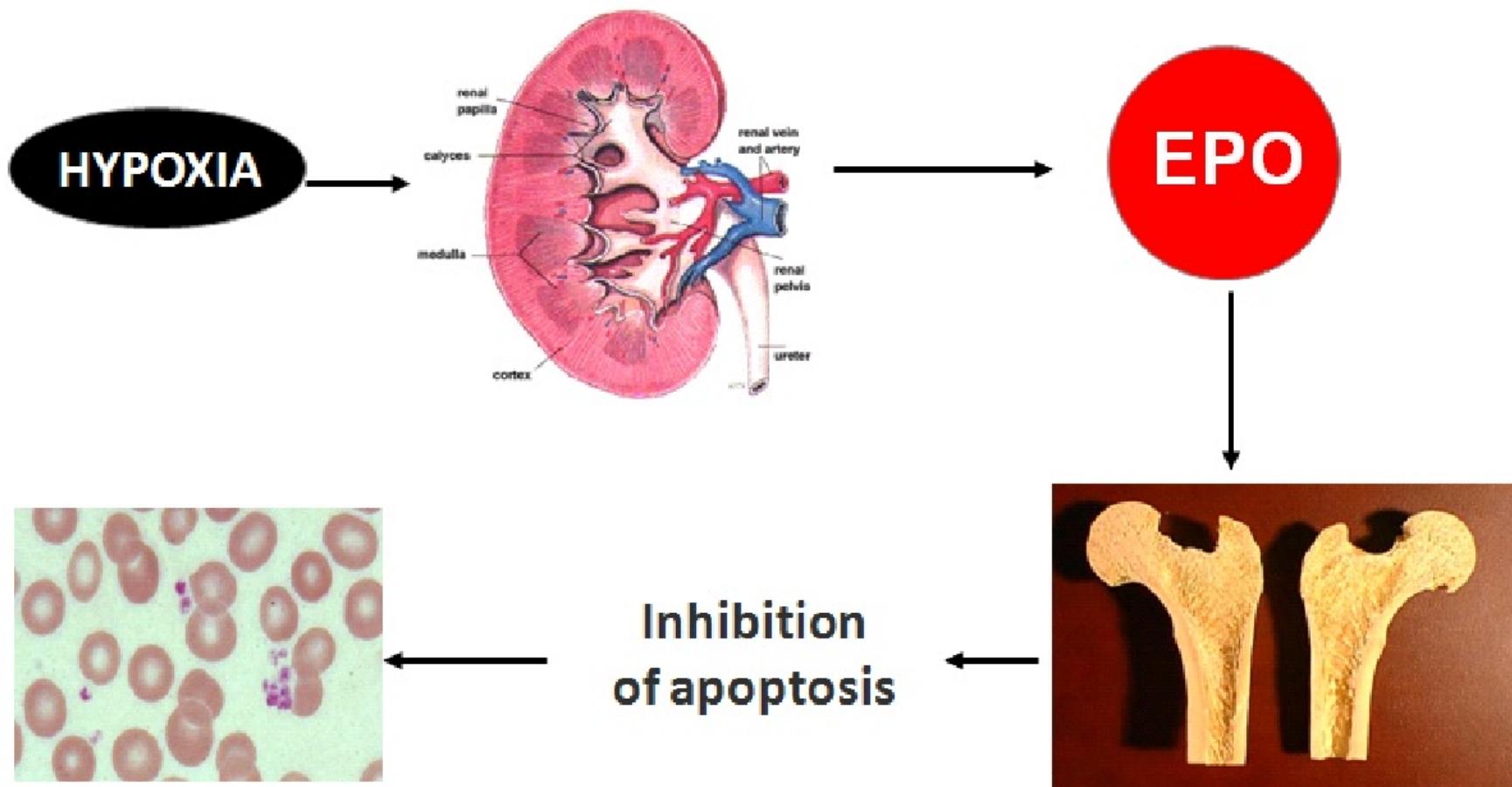
Department of Cardiology, University Medical Center Groningen, Groningen, The Netherlands; Applied Cachexia Research, Department of Cardiology, Charité, Campus Virchow-Klinikum, Berlin, Germany; Department of Nuclear Medicine and Molecular Imaging, University Medical Center Groningen, Groningen, The Netherlands; Isala Clinics, Zwolle, the Netherlands; Leiden University Medical Center, Leiden, the Netherlands; Medical Center Alkmaar, Alkmaar, the Netherlands; Academic Medical Center, Amsterdam, the Netherlands; St. Antonius Hospital, Nieuwegein, the Netherlands; Amphia Hospital, Breda, the Netherlands.



University Medical Center Groningen



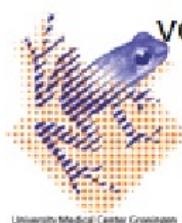
Classical Effect EPO: hematopoiesis



Experimental studies: cardioprotection with EPO

	Animal	Model	Outcome
Calvillo 2003	Rat	I/R: 30 min/7d	reduction of cardiomyocyte loss; improvement of LV function
Lipsic 2004	Rat	I/R: 40 min/1d	reduced infarct size
Parsa 2004	Rabbit	I/R: 30 min/3d	reduced infarct size ; improvement of LV function
Bullard 2005	Rat	I/R: 40 min/1d	reduced infarct size
Hirata 2005	Dog	I/R: 60 min/6h	reduced infarct size

	Animal	Model	Outcome
Parsa 2003	Rabbit	3d FU	improvement of LV function
Moon 2005	Rat	8w FU	reduced infarct size ; improvement of LV function
vd Meer 2005	Rat	9w FU	reduced infarct size improvement of LV function



University Medical Center Groningen

Lipsic et al. JACC 2006

Methods

- Prospective, multicenter, randomized open label trial with blinded endpoints (PROBE) in 529 first ST-elevation myocardial infarction patients
- Primary Endpoint: LVEF (radionuclide ventriculography) at 6 weeks after AMI
- Secondary Endpoints:
 - Enzymatic infarct size
 - Major Adverse Cardiovascular Events
- All endpoints were assessed in a blinded manner
- Major Adverse CV-events were adjudicated by an independent and blinded endpoint committee

Methods

- **Inclusion Criteria:**
 - Successful primary PCI (TIMI 2/3) for a first AMI
 - TIMI flow 0/1 before primary PCI on diagnostic coronary angiography
- **Key Exclusion Criteria:**
 - Anticipated additional revascularization within 6 weeks
 - Cardiogenic shock
 - End stage renal failure
 - Malignant hypertension
 - Previous treatment with RhEPO

Baseline characteristics

	Total cohort N=529	EPO N=263	Control N=266
Age (yrs) *	60.9 ± 11.1	60.8 ± 10.9	61.0 ± 11.3
% Male	77.7	75.7	79.7
Diabetes †	47 (9.1)	25 (9.9)	22 (8.4)
History of Hypertension †	174 (33.8)	84 (33.2)	90 (34.4)
Current smoker †	116 (22.7)	58 (23.2)	58 (22.2)
Hb at baseline (g/dL) *	14.2 ± 1.37	14.0 ± 1.35	14.3 ± 1.29
Ht at baseline (L/L) *	0.41 ± 0.04	0.40 ± 0.04	0.41 ± 0.03
Serum Creatinine (mg/dL) §	0.86 (0.75-1.0)	0.85 (0.74-1.0)	0.87 (0.76-1.01)
Systolic BP (mmHg) *	128.5 ± 24.1	127.2 ± 24.9	129.7 ± 23.3
Heart rate (beats/min) *	74.5 ± 15.8	74.9 ± 15.5	74.2 ± 16.0

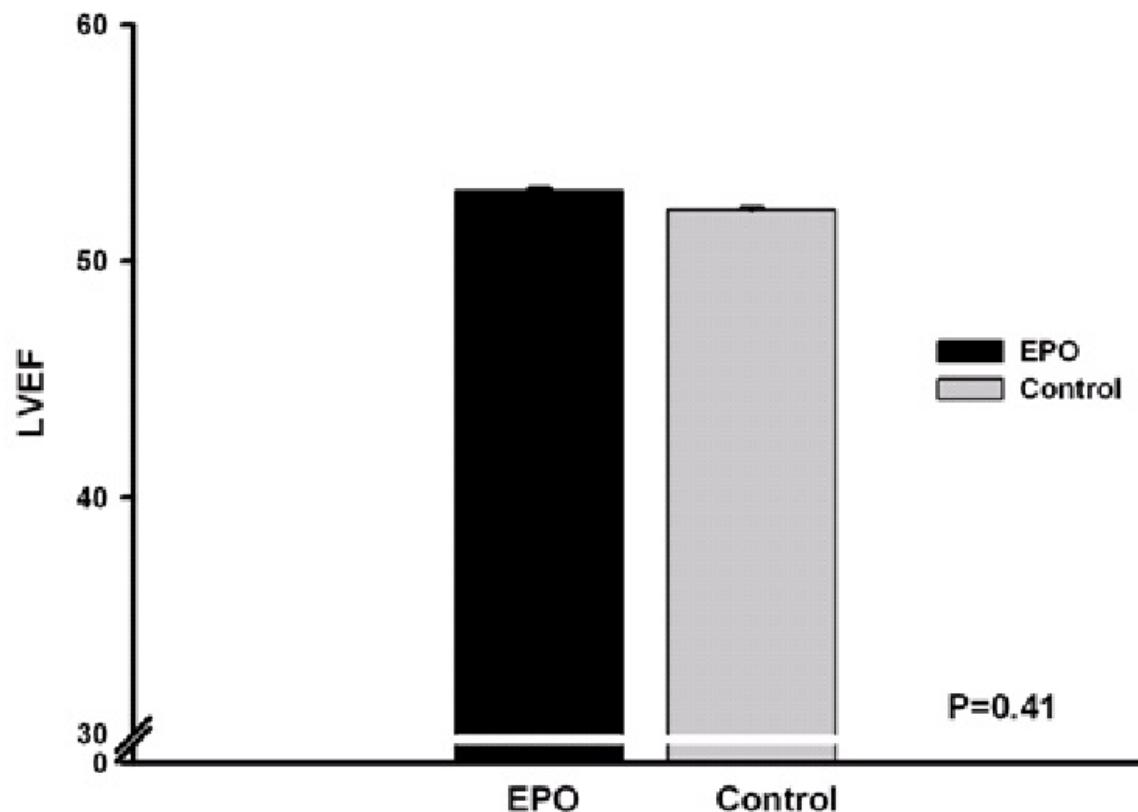
*mean \pm SD; †n (%); §median (IQR)

Baseline Characteristics

	Total cohort N=529	EPO N=263	Control N=266
Time symptoms to PCI (min) [§]	180 (120-270)	180 (126-288)	174 (120-251)
IIb/IIIa inhibitor [†]	411 (77.7)	199 (75.7)	212 (79.7)
Infarct Related Artery [†]			
LAD	209 (40.1)	105 (40.9)	104 (39.4)
RCA	226 (43.4)	112 (43.6)	114 (43.2)
RCx	85 (16.3)	40 (15.6)	45 (17.0)
Medication at follow up [†]			
Aspirin	468 (94.6)	226 (93.8)	242 (95.3)
Oral anticoagulants	33 (6.7)	16 (6.6)	17 (6.7)
Clopidogrel	425 (85.9)	209 (86.7)	216 (85.0)
Beta-blockers	460 (92.9)	225 (93.4)	235 (92.5)
ACE-inhibitors/ARB	385 (77.8)	182 (75.5)	203 (79.9)
Statins	478 (96.6)	235 (97.5)	243 (95.7)

*mean±SD; [†]n (%); [§]median (IQR)

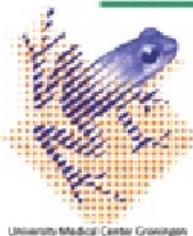
Primary Endpoint



Mean follow-up: 6.5 (± 2.0) weeks. Mean ($\pm SD$) LVEF was 0.53 (± 0.10) in the EPO group and 0.52 (± 0.11) in the control group ($p=0.41$)

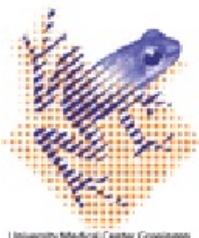
Secondary Endpoint: enzymatic infarct size

	EPO N=263	Control N=266	P-value
Peak CK (U/L)	1750 (895-2970)	1726 (967-3300)	0.293
AUC CK (U/L per 72h)	50,136 (28,212-76,664)	53,510 (33,973-90,486)	0.058
Peak CKMB (U/L)	214 (116-344)	219 (109-322)	0.955
AUC CKMB (U/L per 72h)	5622 (3487-8204)	5931 (3757-8801)	0.16
Peak Troponin T (μg/L)	4.30 (1.94-7.89)	5.90 (2.20-8.00)	0.564



Secondary Endpoint: Major Adverse Cardiovascular Events

	EPO N=263	Control N=266	P-value
All cardiovascular events	8	19	0.032
Cardiovascular death	1	2	0.569
Emergency re-PCI for In-stent thrombosis/reinfarction	2	7	0.288
Unstable angina	3	2	
Stroke	1	1	0.993
Heart failure	1	7	0.034

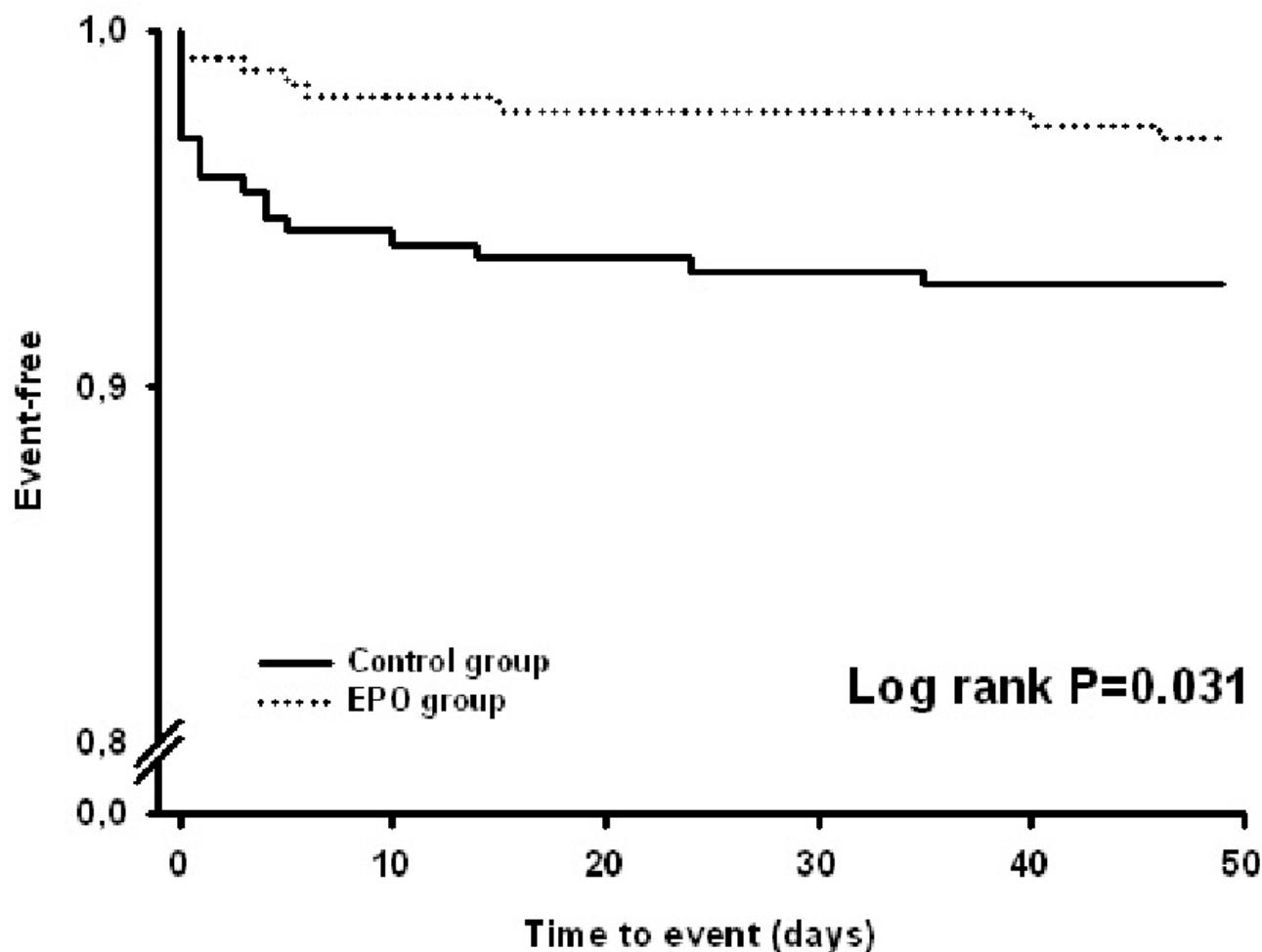


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Major Adverse Cardiovascular Events were defined as cardiovascular death, re-myocardial infarction, re-PCI, or coronary artery bypass graft, stroke, and clear symptoms of heart failure.



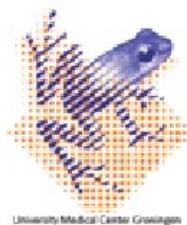
Time to first major adverse cardiovascular event



Major Adverse Cardiovascular Events were defined as cardiovascular death, re-AMI, re-PCI, or coronary artery bypass graft, stroke, and clear symptoms of heart failure.

Safety

- EPO was well tolerated
- no reports of malignant hypertension, seizure, or deep vein thrombosis.
- 49 Serious Adverse Events (SAE's) in 40 control patients, and 33 SAE's in 29 EPO patients.
- Changes in haemoglobin (n=201): mean drop in haemoglobin ($\pm SD$) 0.52 (± 1.09) g/dL in the EPO group and 0.55 (± 1.02) g/dL in the control group ($p=0.86$)
- No difference in the change in haematocrit ($p=0.73$), leucocyte count ($p=0.75$) or platelet count ($p=0.37$) between both groups



Underestimated and Under-recognised: The Late Consequences of Acute Coronary Syndrome (GRACE UK-Belgian Study).

Aims and Methods

- To define the long-term outcomes following ST Elevation and non-ST elevation ACS
- To determine whether the GRACE risk score predicts long-term risk of death, CV death and MI

GRACE ACS Risk Model

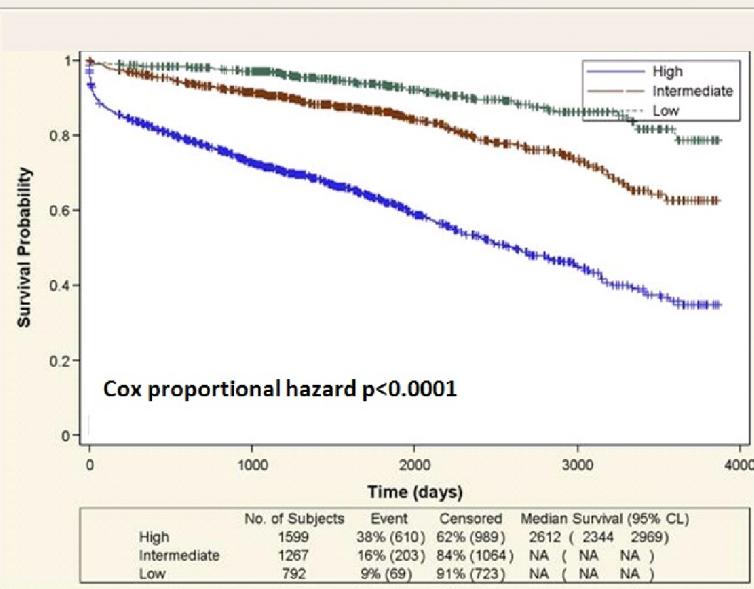
Global Registry of Acute Coronary Events

At Admission (in-hospital/to 6 months) At Discharge (to 6 months)

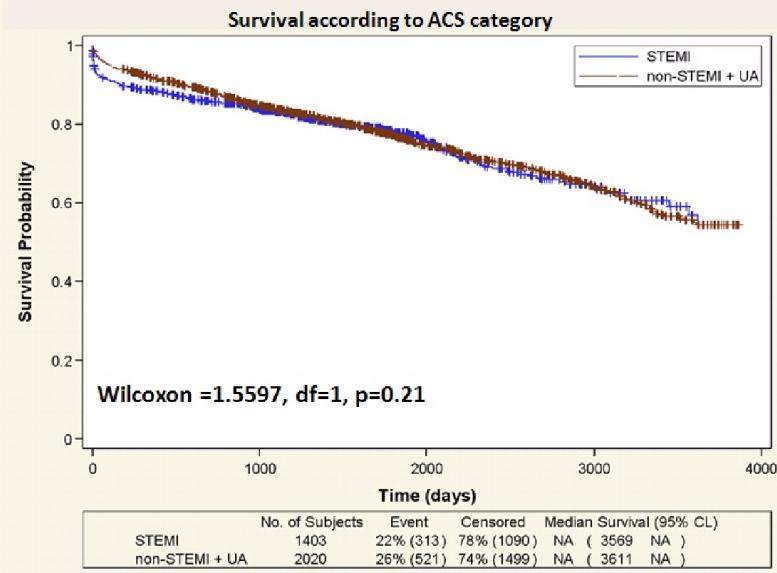
Age	Years	<input type="checkbox"/> Cardiac arrest at admission		
HR	bpm	<input type="checkbox"/> ST-segment deviation		
SBP	mmHg	<input type="checkbox"/> Elevated cardiac enzymes/markers		
Creat.	mg/dL	Probability of		
CHF	Killip Class	In-hospital	Death	Death or MI
		To 6 months	--	--
		SI Units	Reset	

Calculator | Instructions | GRACE Info | References | Disclaimer

Survival according to GRACE Score (score for death in-hospital)

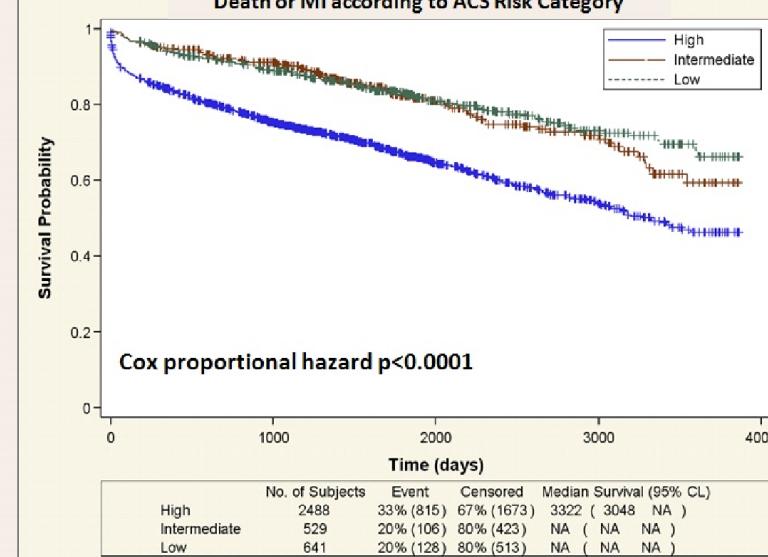


Survival according to ACS category

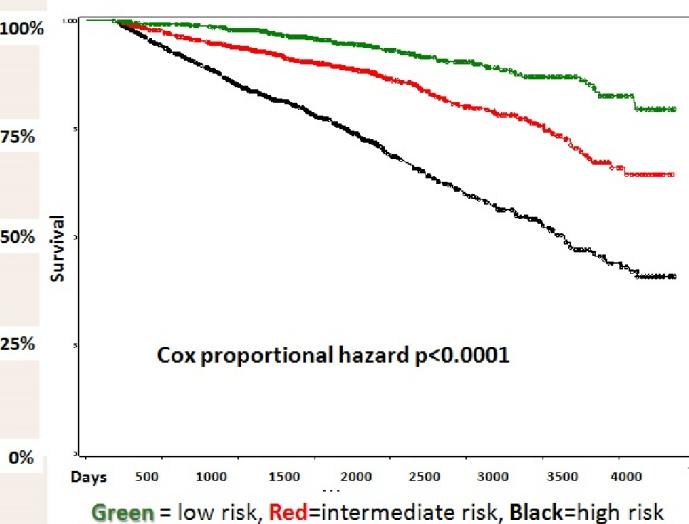


No evidence of a difference[HR 1.026, 95% CI 0.89-1.18]

Death or MI according to ACS Risk Category



Landmark Analysis: GRACE score and mortality after 6 months



Clinical Implications:

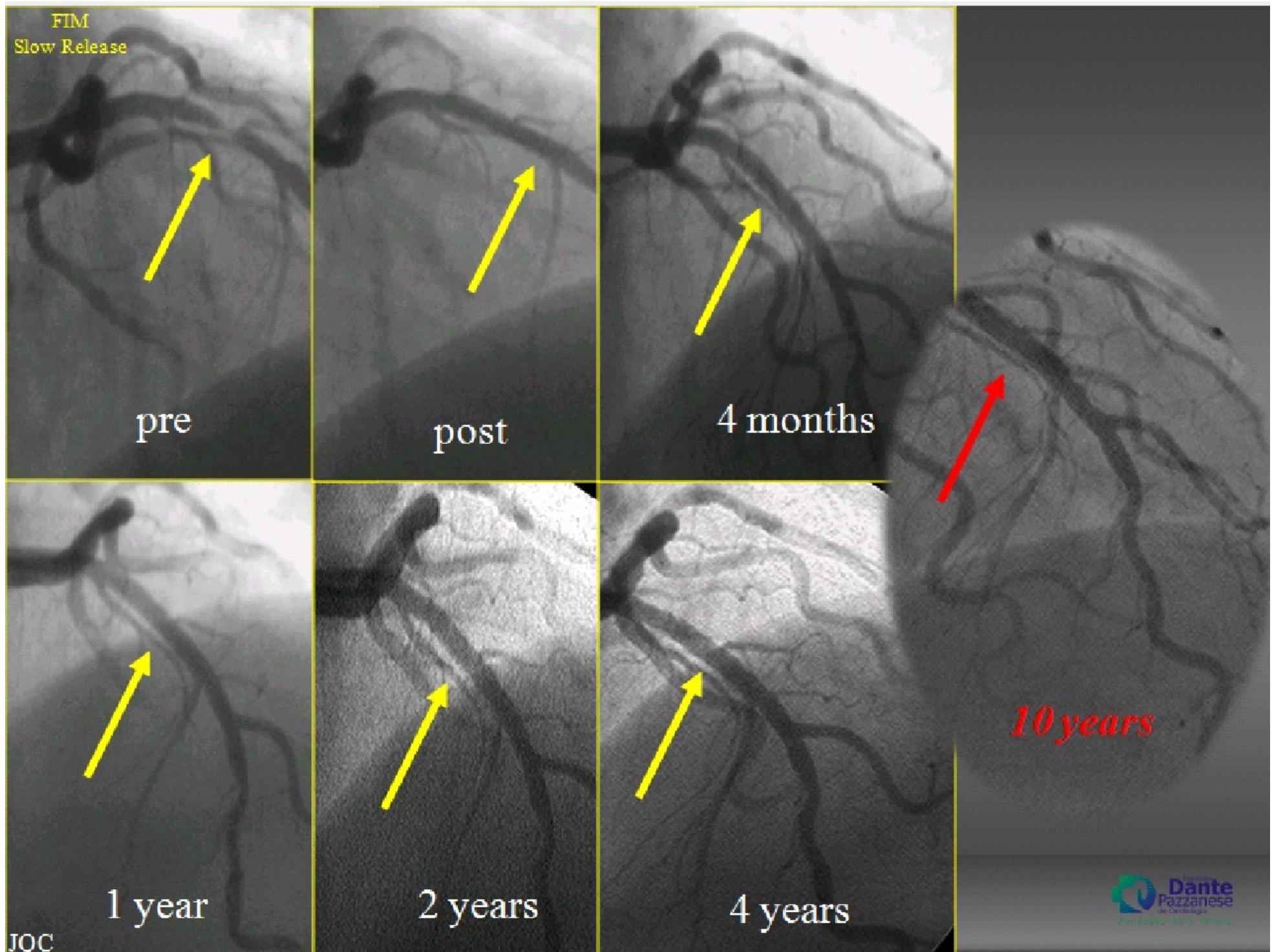
- The late complications of ACS are poorly recognised and often underestimated.
- This is despite substantial progress in acute treatment of ACS, and secondary prevention.
- Risk scores can accurately predict long-term outcome and identify those with most to gain from novel strategies and therapies

RAVEL-Study

(ESC, Stockholm, September 2001)

	Bare Metal Stent	Cypher-Stent
Patients	n=120	n=118
Results after 12 Months:		
Restenosis:	26,0%	0%





TUESDAY

ESC Congress News



EUROPEAN
SOCIETY OF
CARDIOLOGY®



WORLD HEART
FEDERATION®



World Congress of Cardiology 2006

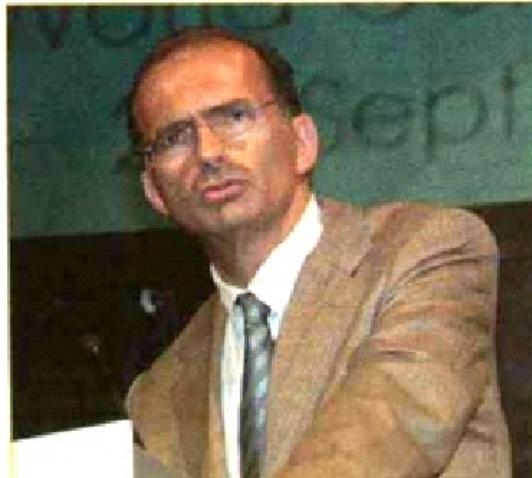
The unique meeting of the European Society of Cardiology Congress 2006 and the World Heart Federation's XVth World Congress of Cardiology



Do drug-eluting stents increase deaths?

TWO SEPARATE, independent meta-analyses, presented in Hot Line session I, suggest drug-eluting stents (DES) may increase death, Q-wave myocardial infarction (clinical surrogates of in-stent thrombosis) and cancer deaths, bringing the long-term safety of DES firmly into the spotlight. Discussant Salim Yusuf (McMaster University, Canada) hailed the data as one of the most important presentations to come out of this year's meeting.

"Six million people in the world have been implanted with DES, yet their long-term safety and efficacy is unknown," said Yusuf. "I've a feeling the data we're seeing today is only the tip of the iceberg. We need to encourage more



obtain this data from the manufacturer," said Nordmann. He speculated that the increase in cancer might be due to a rapid impairment of the immune system.

Yusuf widened the debate to include percutaneous coronary intervention (PCI). "The overuse of PCI is an insidious change in the culture of cardiology that needs to be reversed," he said. The use of PCI was established in MI, high-risk unstable angina and cardiogenic shock. However, its use in stable disease was a totally different question.

"There's no beneficial influence on mortality - PCI does nothing to prevent heart attack. All we are doing is providing short-term relief of chest pain - it's not re-stenosis that kills, but the

Stent thrombosis

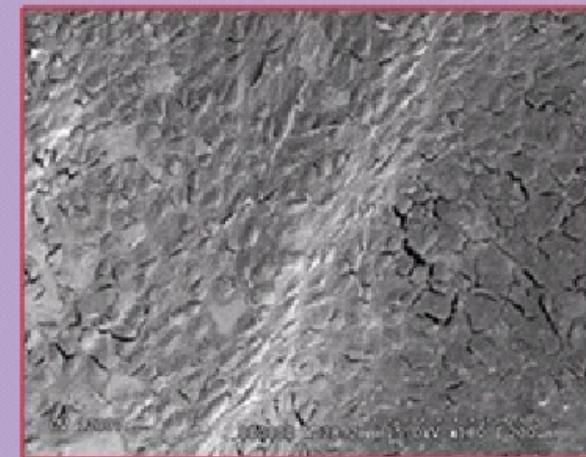
4 year F.U. of FIM patient



Pt # 4 (Fast release)

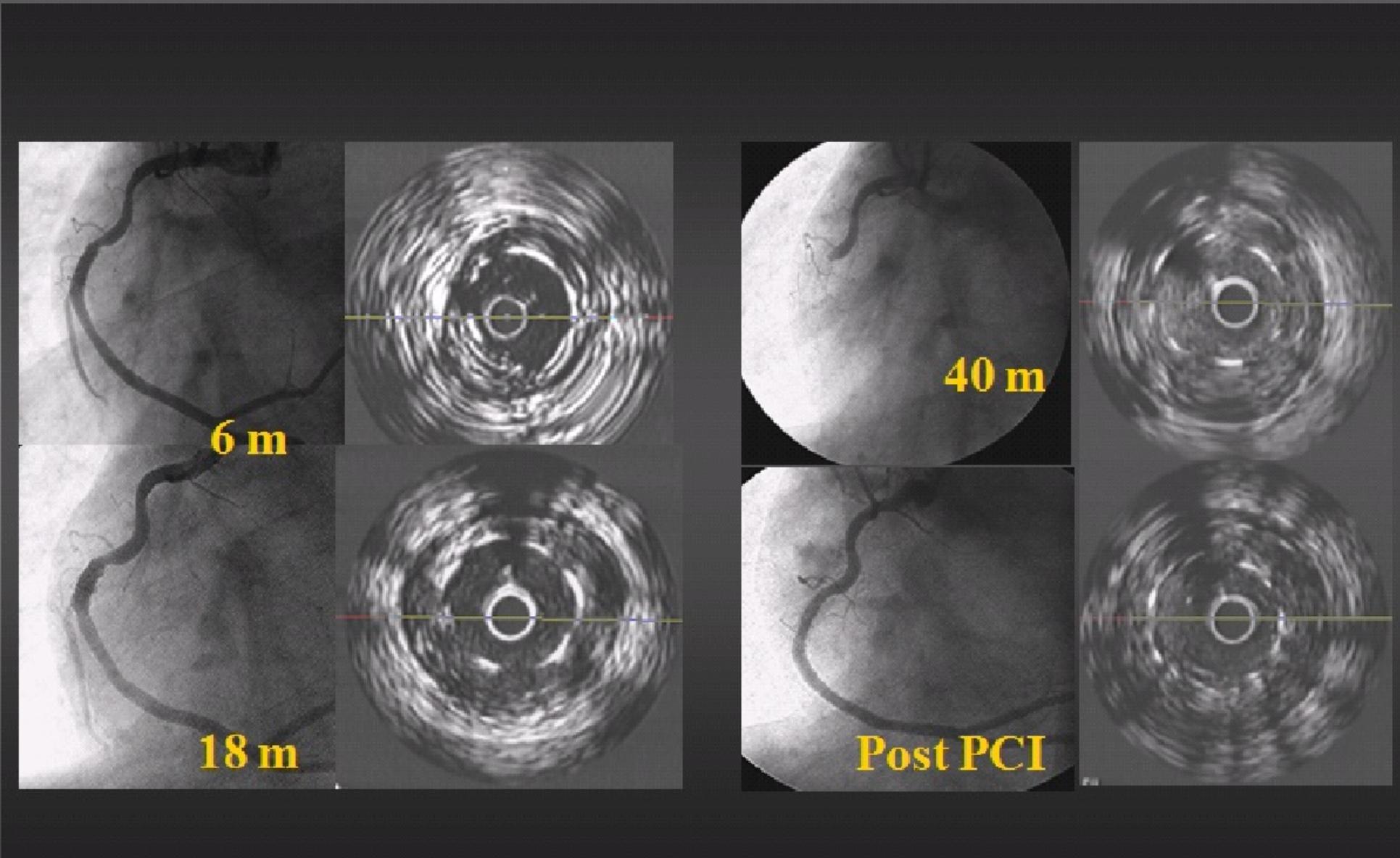
SEM showed > 95% endothelized
stent surface.

Uncovered stent strut (arrow).



Courtesy of R. Virmani

DES – Late incomplete apposition and stent thrombosis



Safety

- ❖ Late stent thrombosis is (was) an important limitation of first generation drug eluting stents
- ❖ Mechanism multifactorial :
 - Delayed/incomplete neointimal coverage
 - Hypersensitivity
 - Late malapposition
 - Abnormal vasomotion (?)
 - Premature discontinuation of antiplatelet τ
 - Resistance to thienopyridines
 - Stent fracture
- ❖ Prolonged dual antiplatelet τ is mandatory

Guidelines for Percutaneous Coronary Interventions

The Task Force for Percutaneous Coronary Interventions of the European Society of Cardiology



Authors/Task Force Members: Sigmund Silber, Chairperson* (Germany), Per Albertsson (Sweden), Francisco F. Avilés (Spain), Paolo G. Camici (UK), Antonio Colombo (Italy), Christian Hamm (Germany), Erik Jørgensen (Denmark), Jean Marco (France), Jan-Erik Nordrehaug (Norway), Witold Ruzyllo (Poland), Philip Urban (Switzerland), Gregg W. Stone (USA), William Wijns (Belgium)

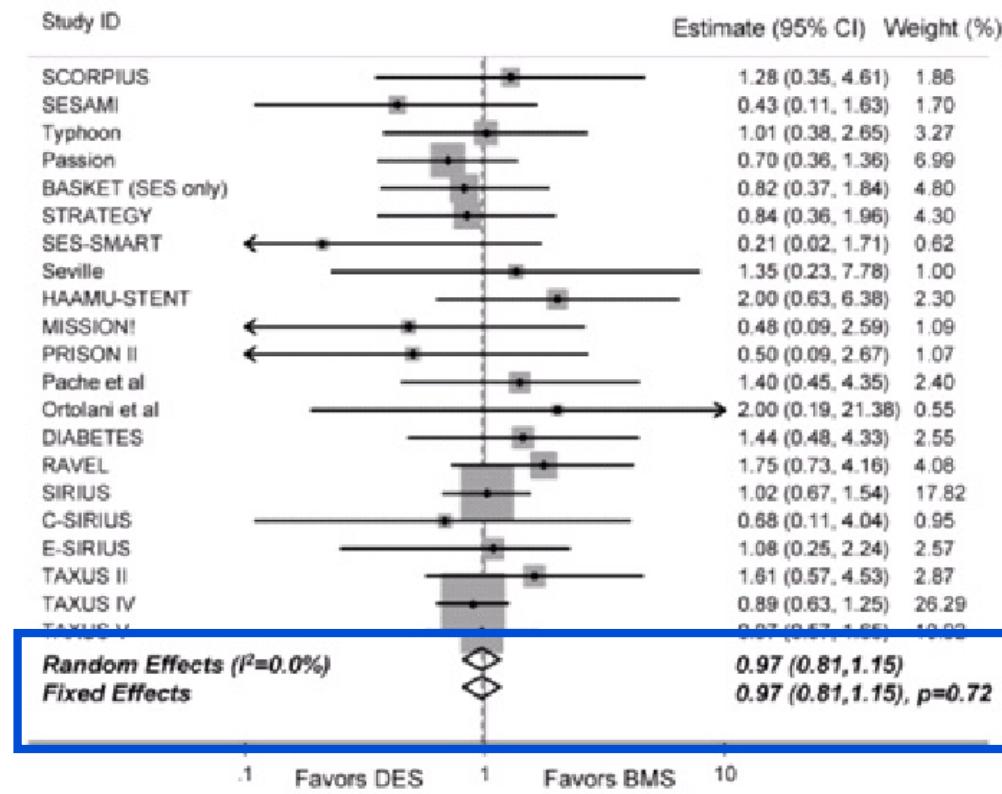
Table 8 Recommendations for clopidogrel as adjunctive medication for PCI

Indication	Initiation and duration	Classes of recommendations and levels of evidence	Randomized studies for levels A or B
Pre-treatment of planned PCI in stable CAD	Loading dose of 300 mg at least 6 h before PCI, ideally the day before	I C	—
Pre-treatment for primary PCI in STEMI or immediate PCI in NSTE-ACS or ad hoc PCI in stable CAD	Loading dose of 600 mg, immediately after first medical contact, if clinically justifiable	I C	—
After all bare metal stent procedures	3–4 weeks	I A	CLASSICS TOPPS Bad Krozingen
After vascular brachytherapy	12 months	I C	—
After drug-eluting stents	6–12 months	I C	—
After NSTE-ACS	Prolonged for 9–12 months	I B	CURE

Interventional Cardiology

Safety and Efficacy of Drug-Eluting and Bare Metal Stents Comprehensive Meta-Analysis of Randomized Trials and Observational Studies

Ajay J. Kirtane, MD, SM; Anuj Gupta, MD; Srinivas Iyengar, MD; Jeffrey W. Moses, MD;
Martin B. Leon, MD; Robert Applegate, MD; Bruce Brodie, MD; Edward Hannan, PhD;
Kishore Harjai, MD; Lisette Okkels Jensen, MD; Seung-Jung Park, MD, PhD; Raphael Perry, MD;
Michael Racz, PhD; Francesco Saia, MD, PhD; Jack V. Tu, MD, PhD; Ron Waksman, MD;
Alexandra J. Lansky, MD; Roxana Mehran, MD; Gregg W. Stone, MD



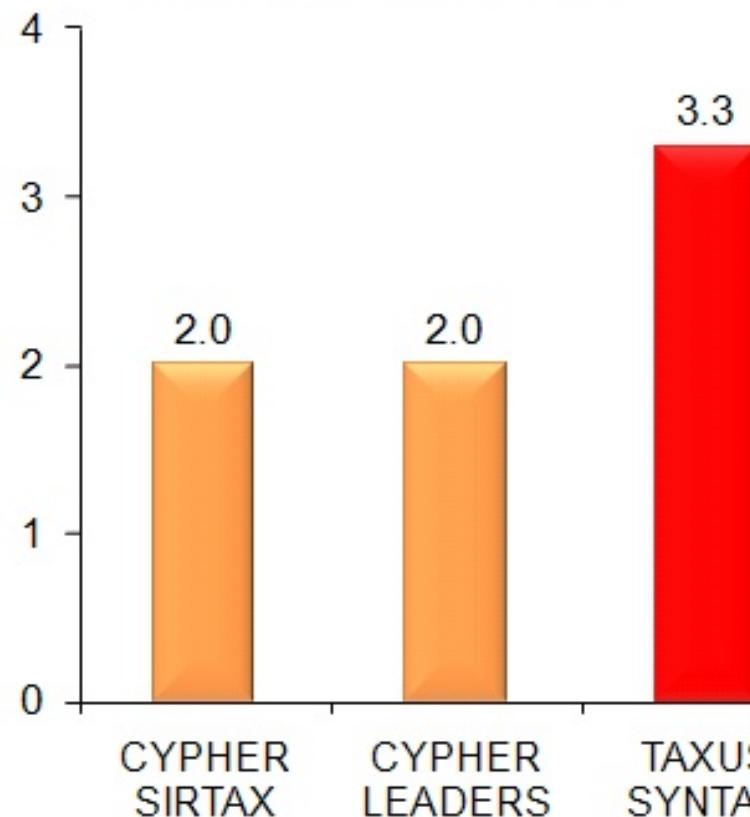
Mortality

(Circulation. 2009;119:3198-3206.)

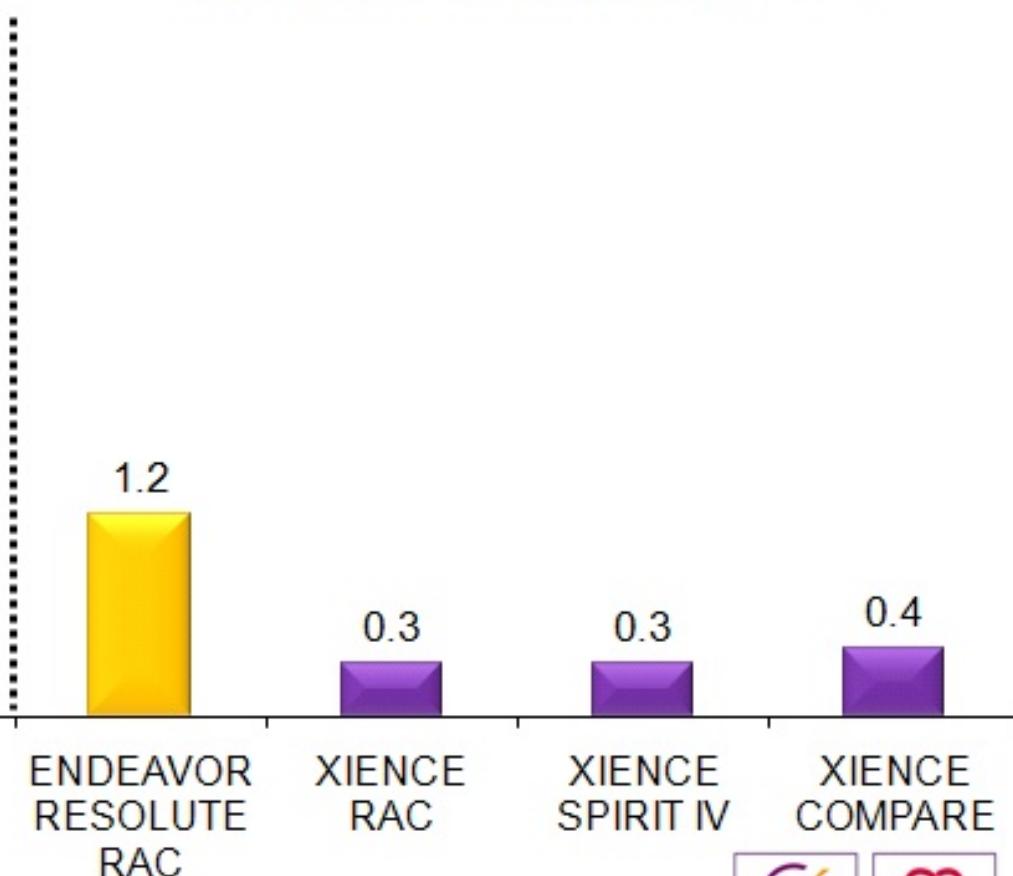
Stent Thrombosis in Perspective

Definite Stent Thrombosis in All Comer Trials @ 12 Months

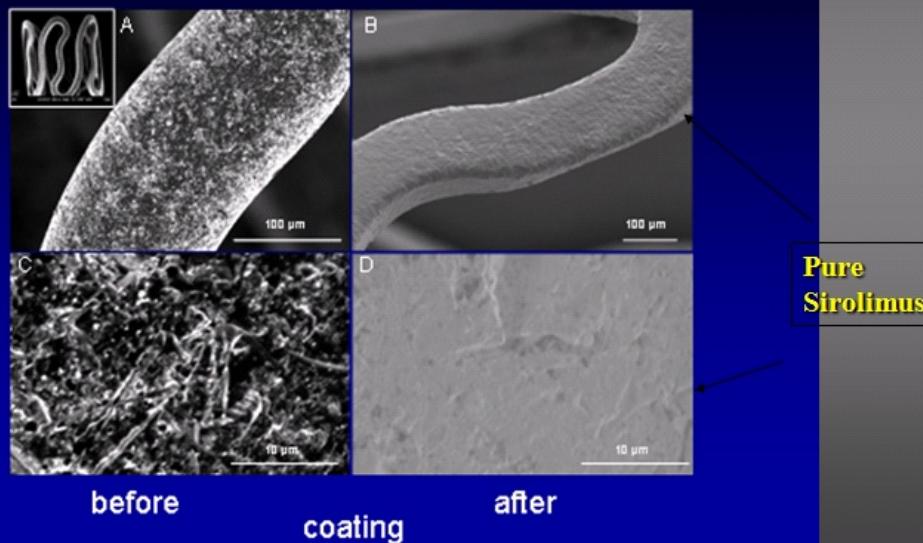
Early Generation DES



Newer Generation DES

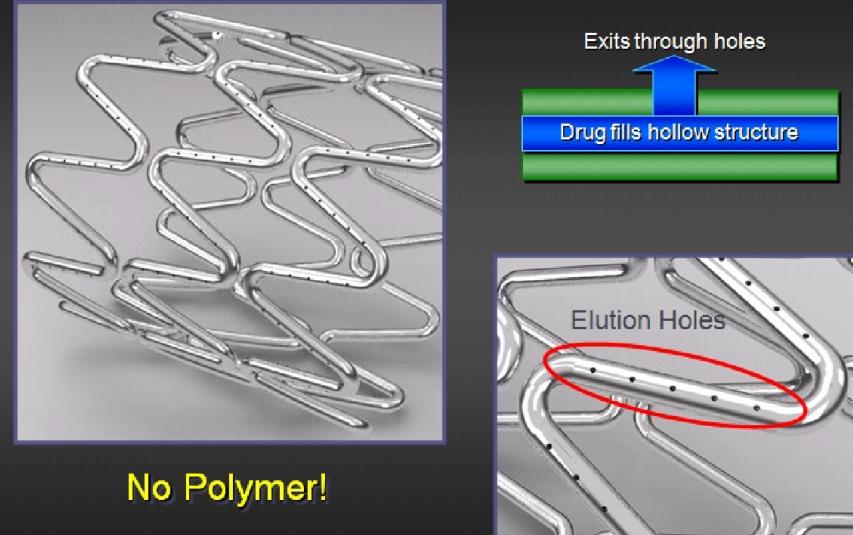


Unique microporous stent surface



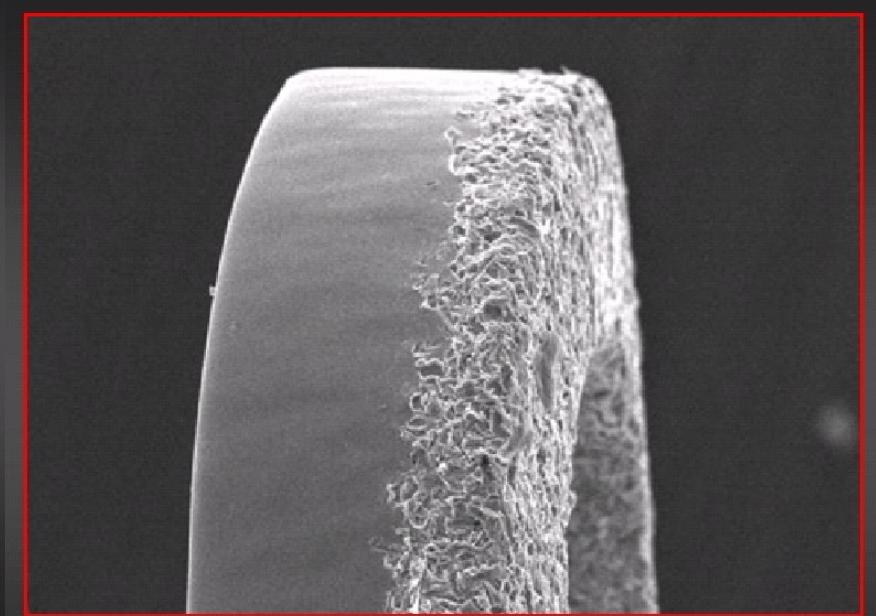
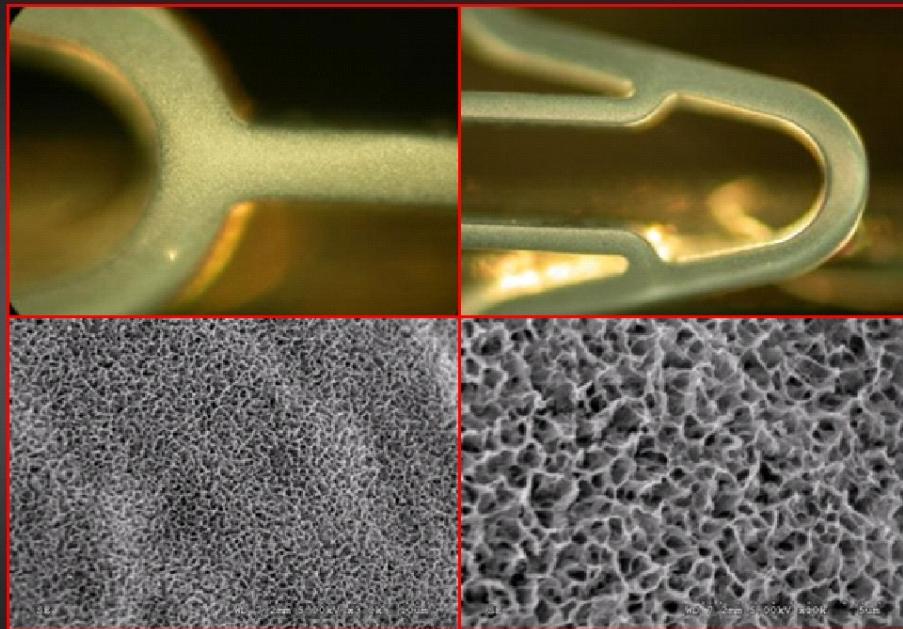
Drug Filled Stent (DFS) Concept (Medtronic)

Drug elution controlled by diffusion physics



3D MicroPorous Nanofilm HAp BioMatrix Freedom Stent

Micro-structured Surface



Bioresorbable Stents

Igaki-Tamai



PLA

BVS



PLA

REVA



Tyrosine-
Policarbonate

BTI



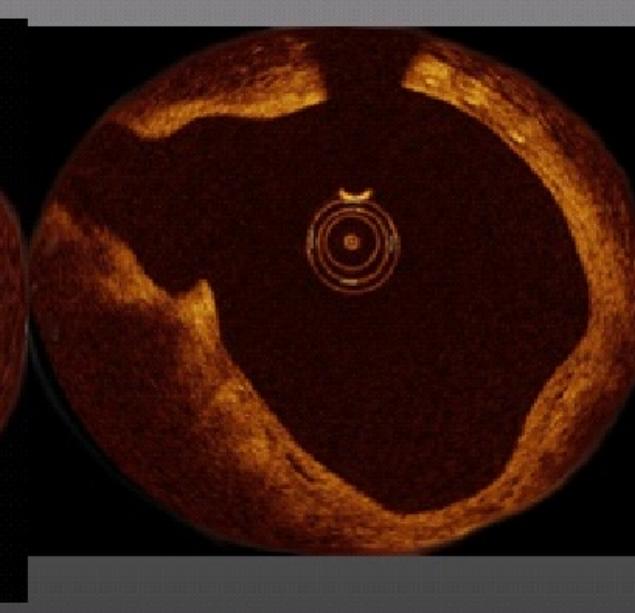
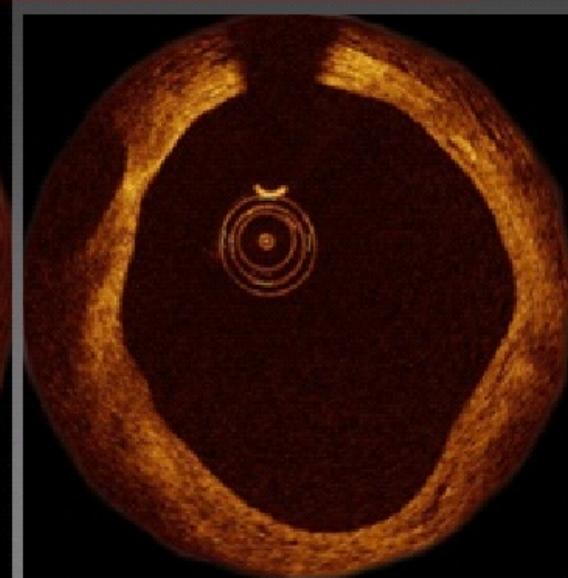
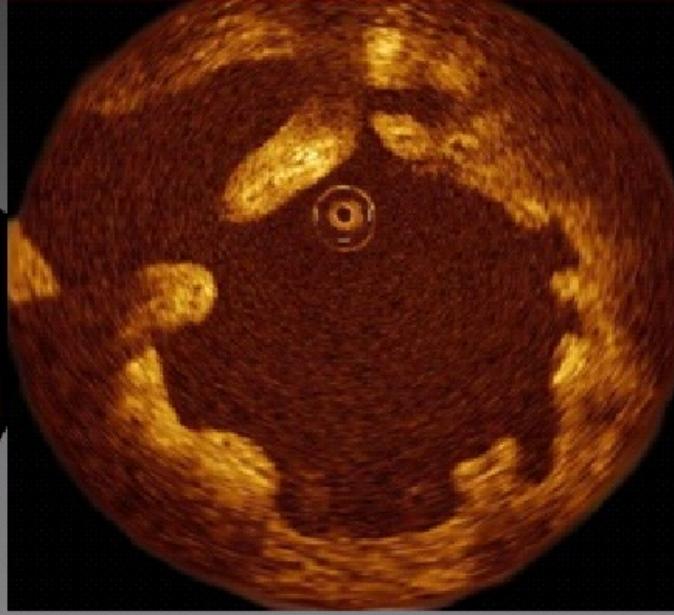
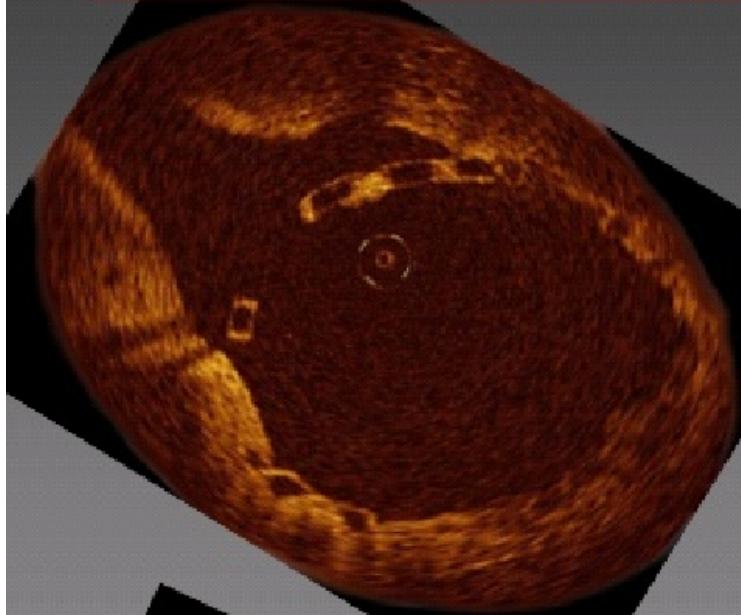
PAE-Salicylate

Biotronik



Magnesium

Bioresorption and vessel wall integration are real phenomena. (Lancet 2009)



Baseline

6 months

2 years

CURE ACTIVE

Effects of CYP2C19 genotypes on clopidogrel treatment in the CURE and ACTIVE trials

Guillaume Pare MD

Canada Research Chair in Genetic and Molecular Epidemiology



CURE ACTIVE

Results:

- No effect of CYP2C19 loss-of-function alleles on efficacy and safety in CURE and ACTIVE

CURE ACTIVE

CYP2C19 Alleles

3 allele classes

- "Wild type" (*1): 63%
- Loss-of-function (*2, *3): 13%
- Gain-of-function (*17): 24%

5 metabolizer phenotypes

- Poor: 2 loss-of-function alleles (2%)
- Intermediate: 1 loss-of-function and 1 wild type alleles (16%)
- Extensive: 2 wild type alleles (39%)
- Ultra: 1 or 2 gain-of-function alleles (37%)
- Unknown: 1 gain-of-function and 1 loss-of-function alleles (6%)

2 carrier status

- Loss-of-function carriers (1 or more *2, *3): 24%
- Gain-of-function carriers (1 or more *17): 41%

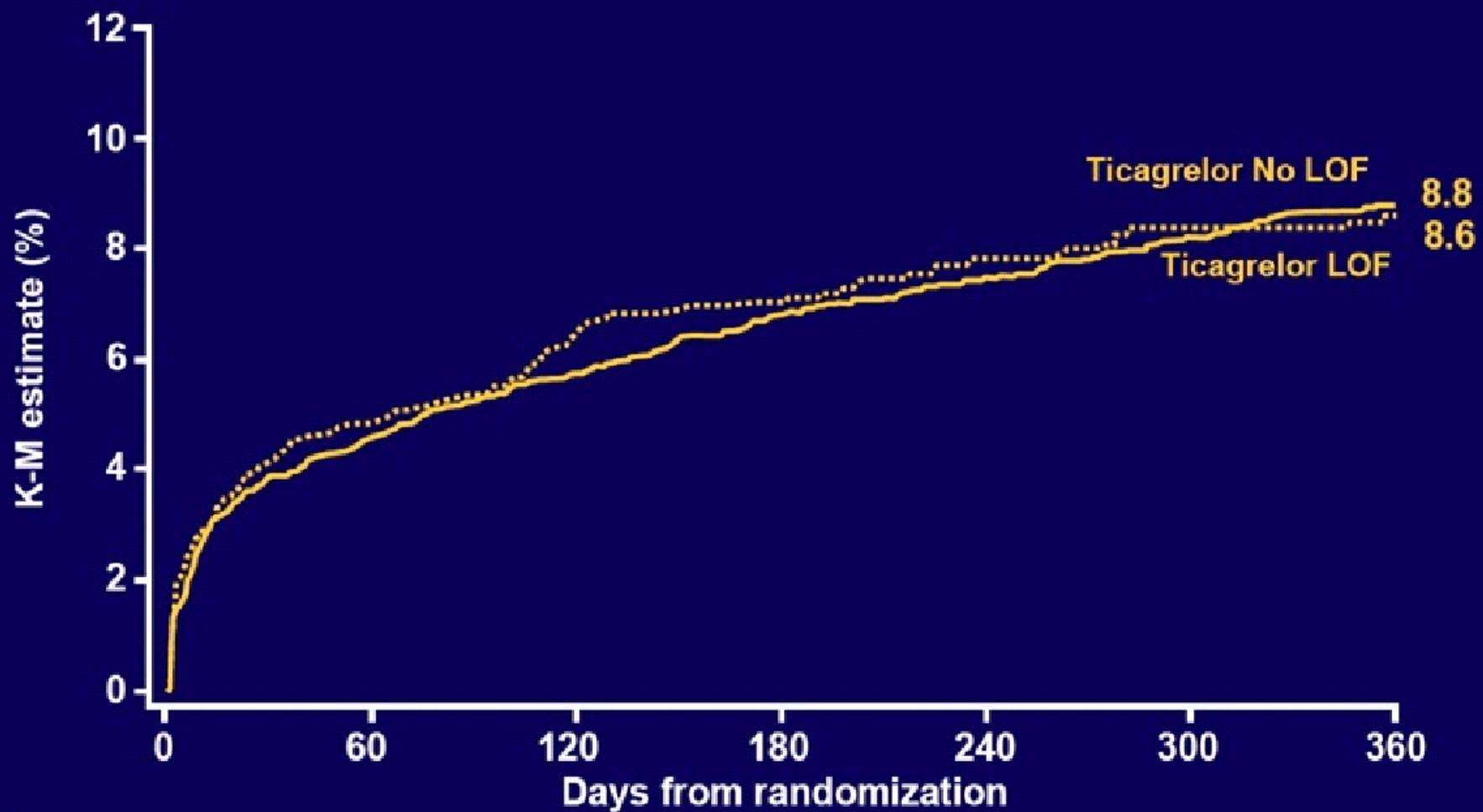


Impact of CYP2C19 and ABCB1 SNPs on outcomes with ticagrelor versus clopidogrel in acute coronary syndromes: a PLATO genetic substudy

Lars Wallentin, Stefan James, Robert F Storey,
Martin Armstrong, Bryan Barratt, Jay Horrow,
Steen Husted, Hugo Katus, Gabriel Steg, Richard Becker
for the PLATO investigators

Primary endpoint in the ticagrelor group
in relation to any CYP2C19 LOF allele (K-M estimate)

PLATO

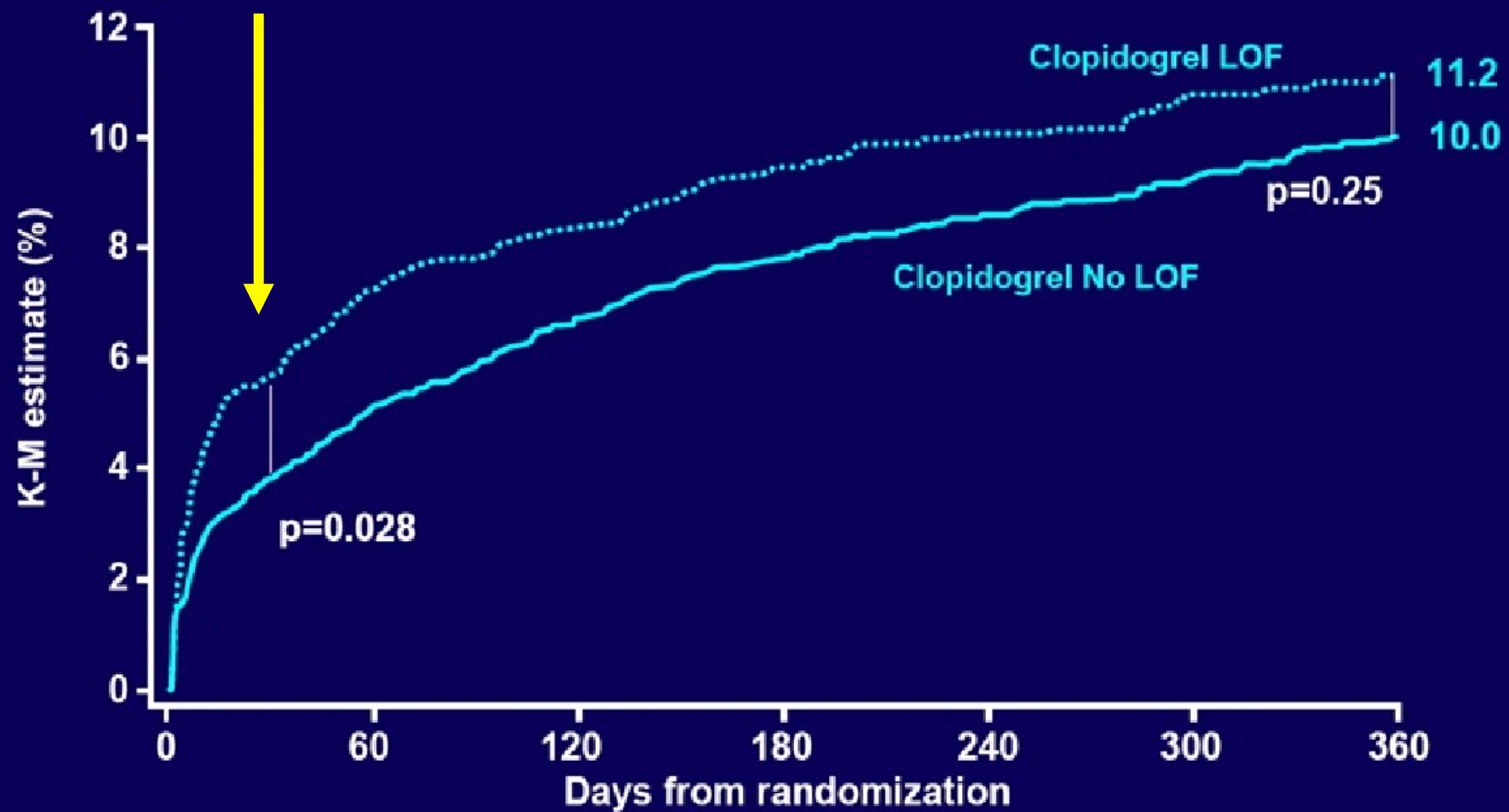


No. at risk

Ticagrelor LOF	1,384	1,305	1,274	1,250	1,053	834	683
Ticagrelor No LOF	3,554	3,352	3,301	3,222	2,718	2,127	1,761

Primary endpoint in the clopidogrel group in relation to any CYP2C19 LOF allele (K-M estimate)

PLATO



No. at risk

Clopidogrel LOF	1,388	1,275	1,259	1,226	1,027	801	658
Clopidogrel No LOF	3,516	3,321	3,256	3,186	2,691	2,123	1,757



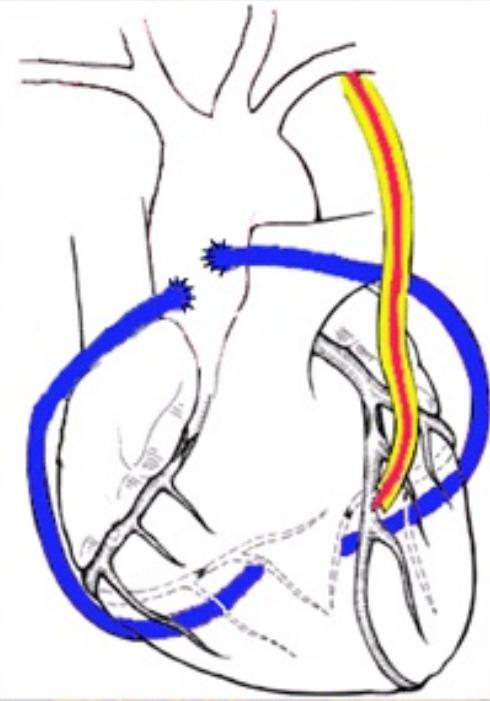
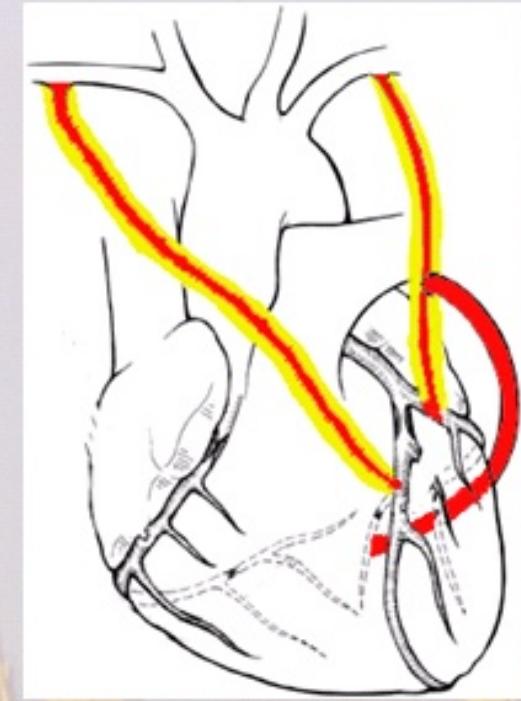
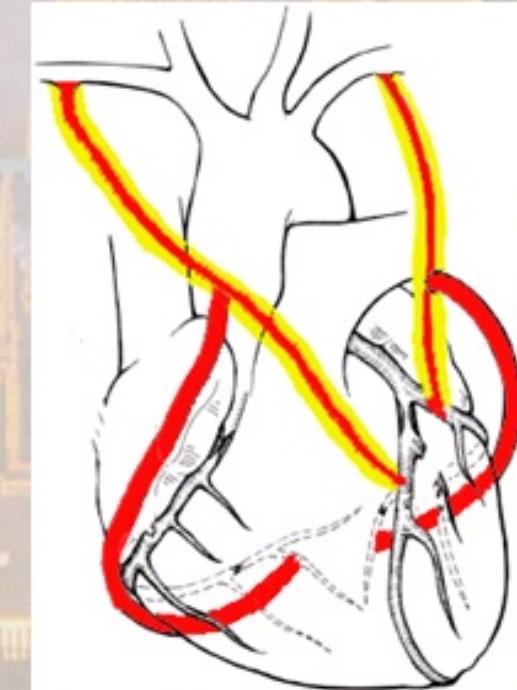
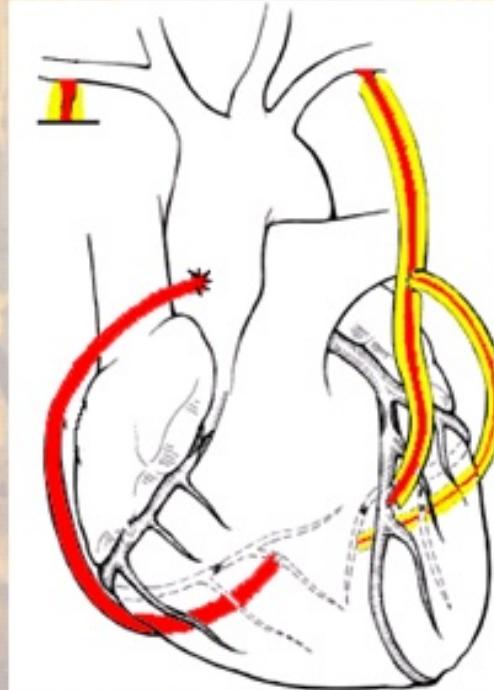
Randomized Trial to Compare Bilateral Versus Single Internal
Mammary Coronary Artery Bypass Grafting (CABG):
One Year Results of the Arterial Revascularisation Trial (ART)

DP Taggart, DG Altman, AM Gray, B Lees, F Nugara, LM Yu, H Campbell, M Flather, on
behalf of the ART Investigators

John Radcliffe Hospital Oxford, University of Oxford, Royal Brompton & Harefield NHS Foundation Trust London
and Imperial College London

ESC Hot Line 2010, Stockholm
On Line publication in EHJ



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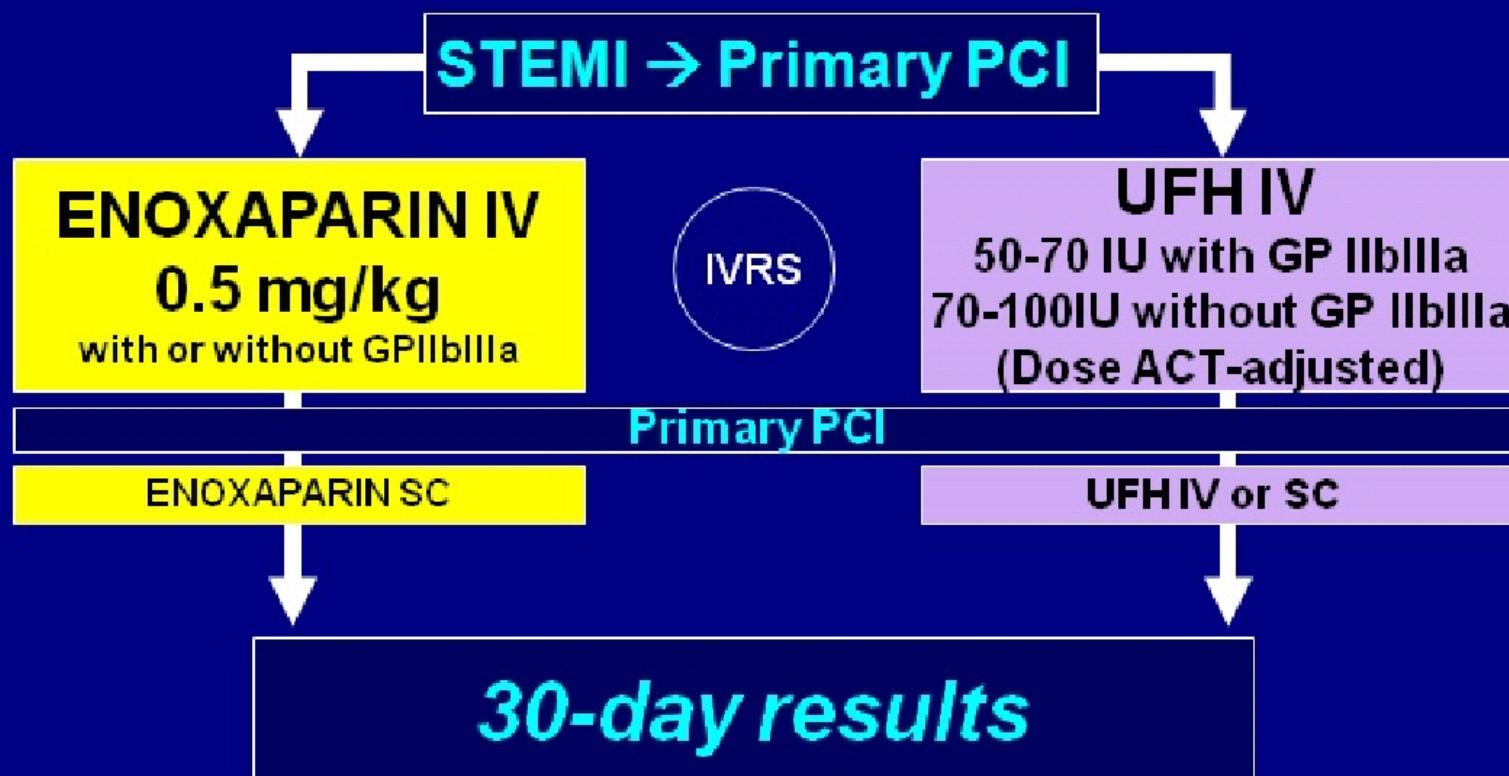
ART Surgery

		SIMA (n=1552)	BIMA (n=1542)	Δ
Off-Pump		40%	41.8%	
Grafts	1	0.7%	0.5%	
	2	17.7%	17.8%	
	3	48.5%	50.4%	
	4+	33.2%	31.3%	
Surgery length: mins	mean (SD)	199 (58)	222 (61)	23 mins
Ventilation length: mins	mean (SD)	863 (3293)	968 (3029)	105 mins
Duration ITU stay: hours	mean (SD)	38 (106)	41 (94)	3 hours
Duration of post-op stay: days	mean (SD)	7.5 (7.6)	8.0 (7.4)	0.5 days
Re-exploration for any cause		3.5%	4.3%	
Blood transfusion		12%	12%	
Intra Aortic Balloon Pump		3.7%	4.4%	
Renal support		4.4%	5.9%	



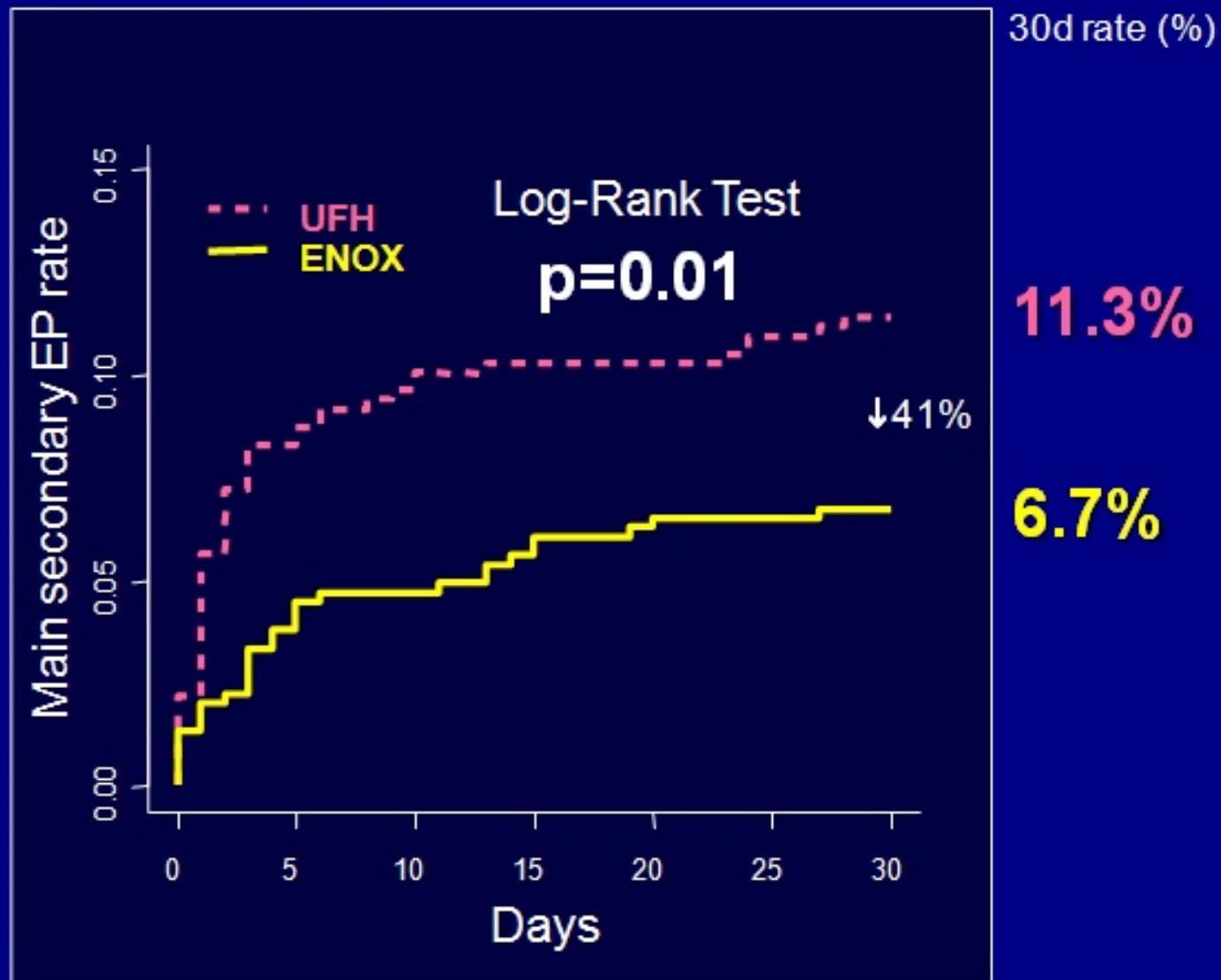
ATOLL Trial design

Randomization as *early* as possible (MICU +++)
Real life population (shock, cardiac arrest included)
No anticoagulation and no lytic before Rx
Similar antiplatelet therapy in both groups



Main Secondary Endpoint (ischemic)

Death, Recurrent MI/ACS or Urgent Revascularization



SYNTAX Trial Design

SYNTAX



62 EU Sites



23 US Sites

De novo 3VD and/or LM (isolated, +1,2,3 VD)

Limited Exclusion Criteria

Previous interventions, Acute MI with $CPK > 2x$, Concomitant cardiac surgery



Heart Team (Surgeon & Interventional Cardiologist)

Amenable for both treatment options

Amenable for only one treatment approach

Stratification:
LM and Diabetes

Randomized Arms
N=1800

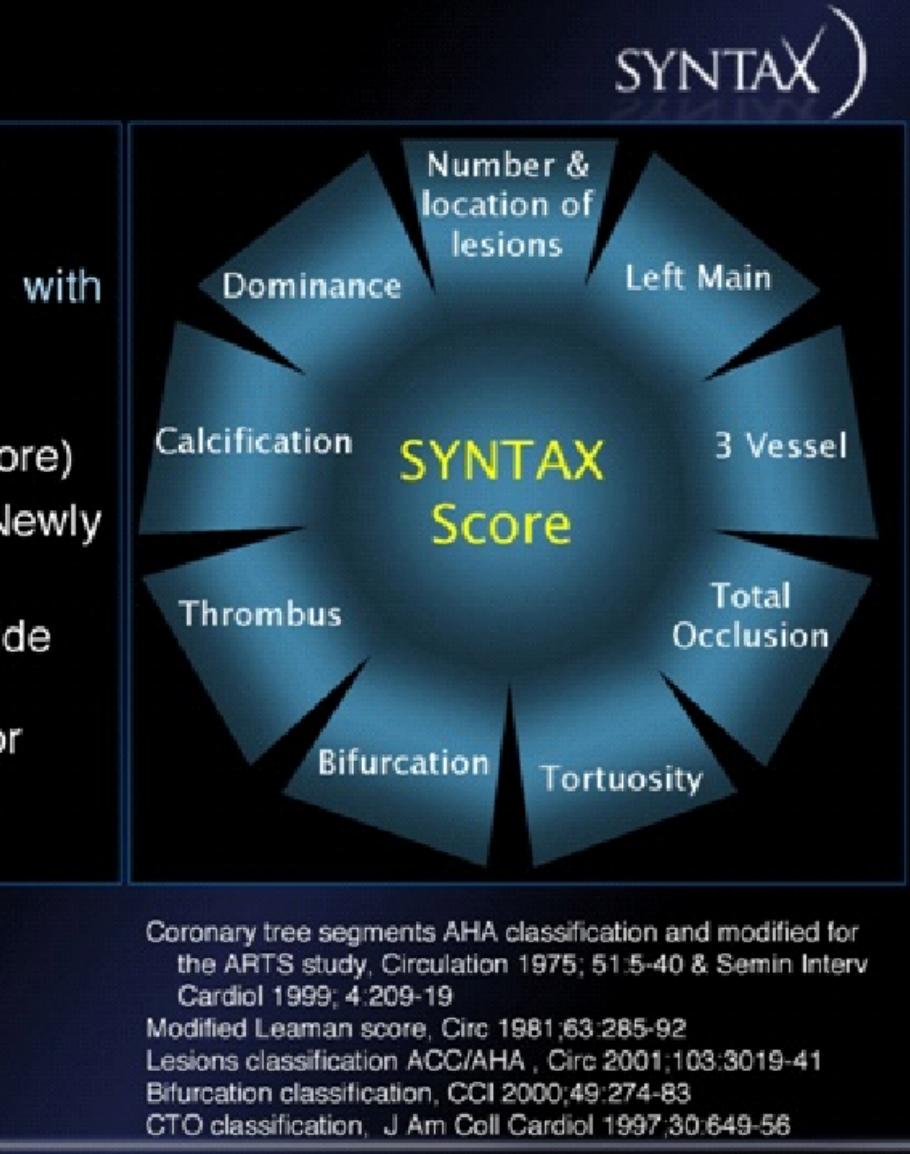
Two Registry Arms
N=1275

Patient Profiling

Local Heart team (surgeon & interventional cardiologist) assessed each patient with regards to:

- Patient's operative risk (euroSCORE & Parsonnet score)
- Coronary lesion complexity (Newly developed SYNTAX Score)
- Goal: SYNTAX Score to provide guidance on optimal revascularization strategies for patients with high risk lesions

Sianos et al, *EuroIntervention* 2005;1:219-27
Valgimigli et al, *Am J Cardiol* 2007;99:1072-81
Serruys et al, *EuroIntervention* 2007;3:450-9



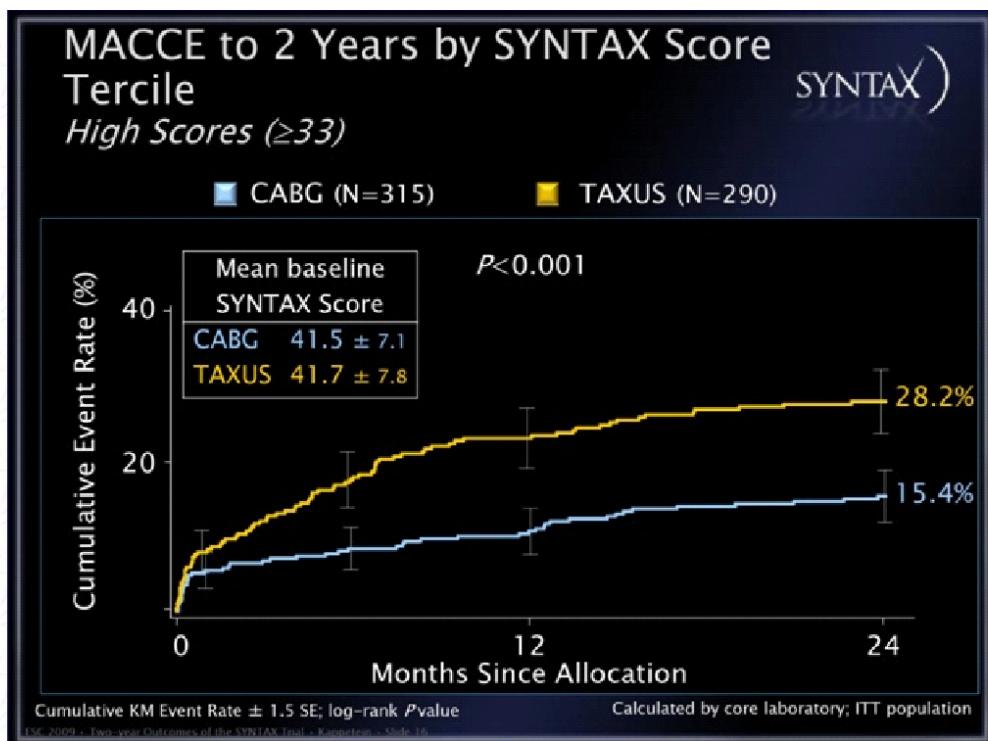
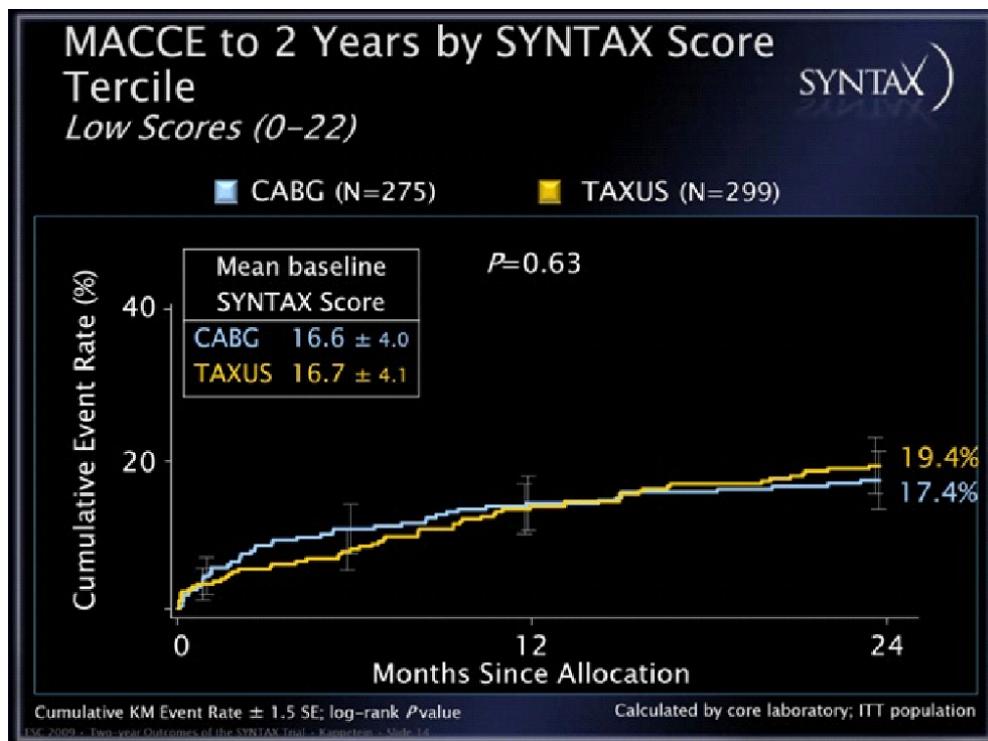
Coronary tree segments AHA classification and modified for the ARTS study, *Circulation* 1975; 51:5-40 & *Semin Interv Cardiol* 1999; 4:209-19
Modified Leaman score, *Circ* 1981;63:285-92
Lesions classification ACC/AHA, *Circ* 2001;103:3019-41
Bifurcation classification, CCI 2000;49:274-83
CTO classification, *J Am Coll Cardiol* 1997;30:649-56

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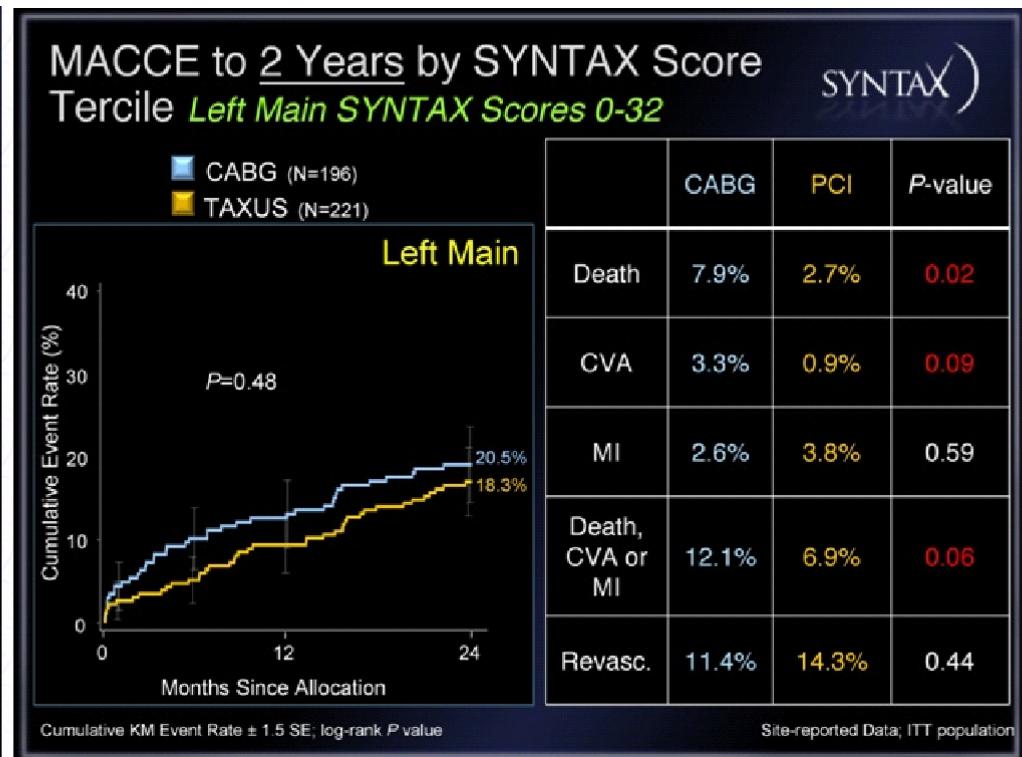
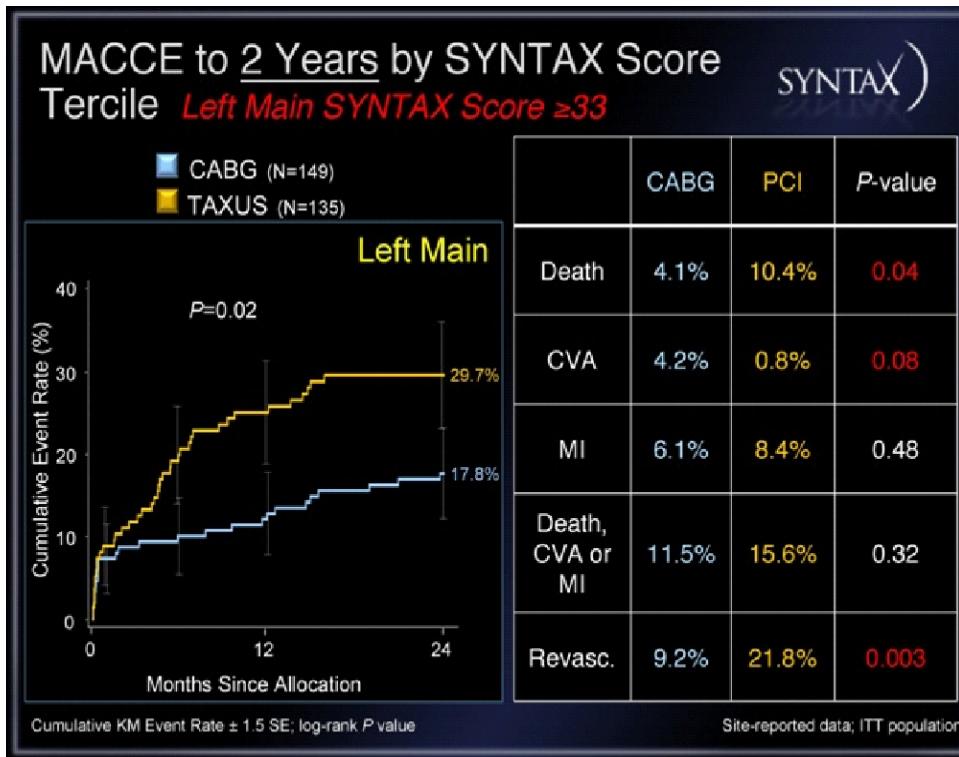
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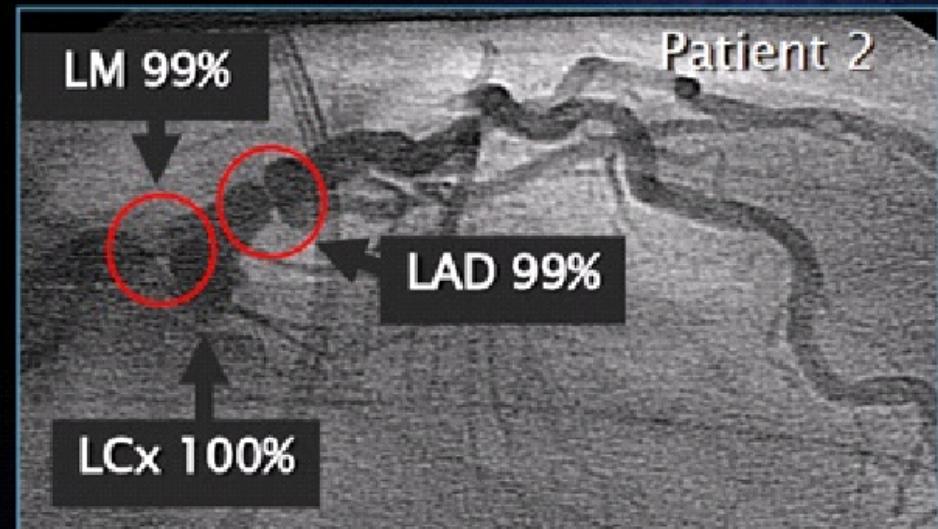
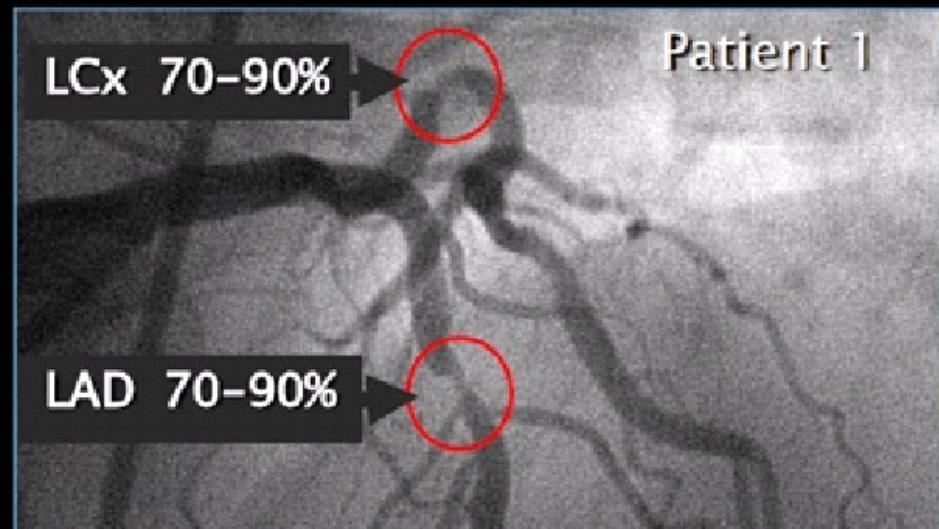
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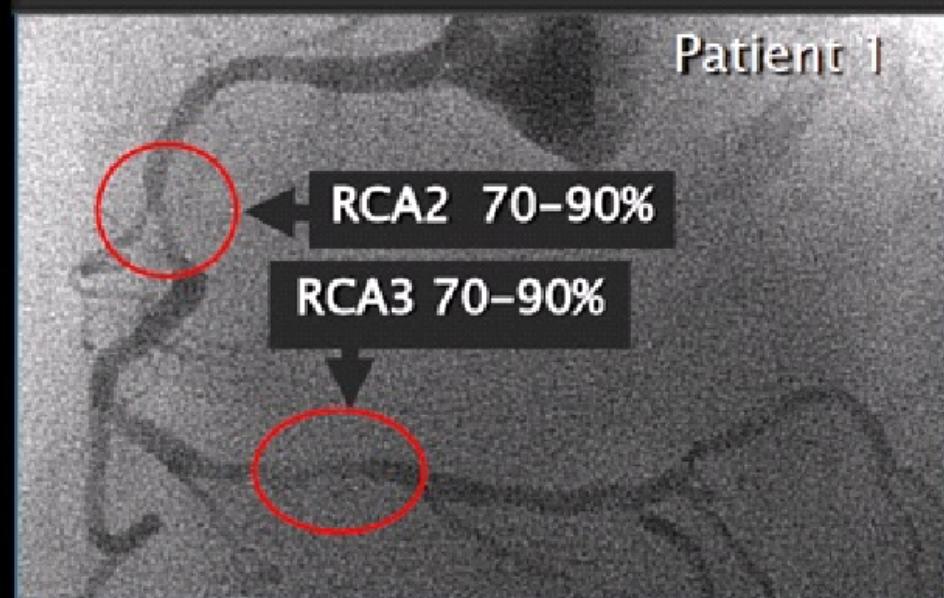
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There is '3-vessel disease' and '3-vessel disease'

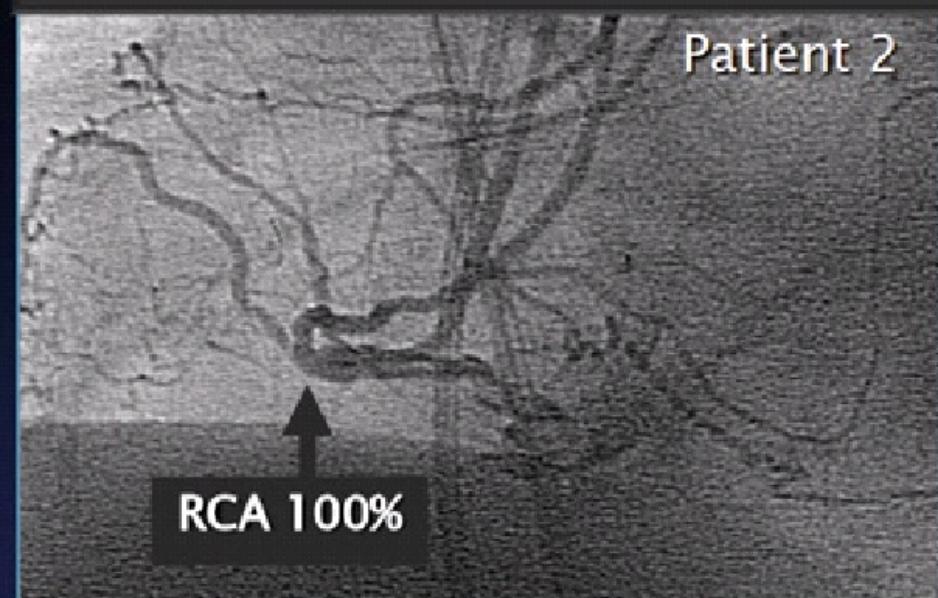
SYNTAX



SYNTAX SCORE 21



SYNTAX SCORE 52



Indications for CABG versus PCI in stable patients with lesions suitable for both procedures and low predicted surgical mortality

Subset of CAD by anatomy	Favours CABG	Favours PCI
1VD or 2VD - non-proximal LAD	IIb C	I C
1VD or 2VD - proximal LAD	IA	IIa B
3VD simple lesions, full functional revascularisation achievable with PCI, SYNTAX score ≤ 22	IA	IIa B
3VD complex lesions, incomplete revascularisation achievable with PCI, SYNTAX score > 22	IA	III A
Left main (isolated or 1VD, ostium/shaft)	IA	IIa B
Left main (isolated or 1VD, distal bifurcation)	IA	IIb B
Left main + 2VD or 3VD, SYNTAX score ≤ 32	IA	IIb B
Left main + 2VD or 3VD, SYNTAX score ≥ 33	IA	III B

Validated drug-eluting stents (DES) for clinical use

DES	Eluted Drug	Trials and references
Clinical primary endpoint reached		
BioMatrix Flex	Biolimus A9	LEADERS
Cypher	Sirolimus	SIRIUS
Endeavor	Zotarolimus	ENDEAVOR-II, -III and -IV
Resolute	Zotarolimus	RESOLUTE-AC
Taxus Liberté/ Element	Paclitaxel	TAXUS-IV and -V/ PERSEUS-WH
Xience V	Everolimus*	SPIRIT-III and -IV
Angiographic primary endpoint reached		
Nevo	Sirolimus	NEVO RES I
Nobori	Biolimus A9	NOBORI-I Phase-1 and -2
Yukon	Sirolimus	ISAR-Test

Recommended risk stratification scores to be used in candidates for PCI or CABG

Score	Validated outcomes	Class /Level	
		PCI	CABG
EuroSCORE	Short and long-term mortality	IIb B	I B
SYNTAX score	Quantify coronary artery disease complexity	IIa B	III B
Mayo Clinic Risk Score	MACE and procedural death	IIb C	III C
NCDR CathPCI	In-hospital mortality	IIb B	-
Parsonnet score	30-day mortality	-	III B
STS score	Operative mortality, stroke, renal failure, prolonged ventilation, deep sternal infection, re-operation, morbidity, length of stay < 6 or > 14 days	-	I B
ACEF score	Mortality in elective CABG	-	IIb C

ACEF score = [Age/Ejection fraction (%)] + 1 (if creatinine > 2 mg/dL).

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Alle Vorträge ab
Oktober abrufbar auf

www.theheart.de

Nächste Veranstaltungen:
Rhythmologisches Seminar:
Termin wird noch bekannt gegeben

Bielefelder Seminar über aktuelle
der Kardiologie

5.2.2011

Fragen in