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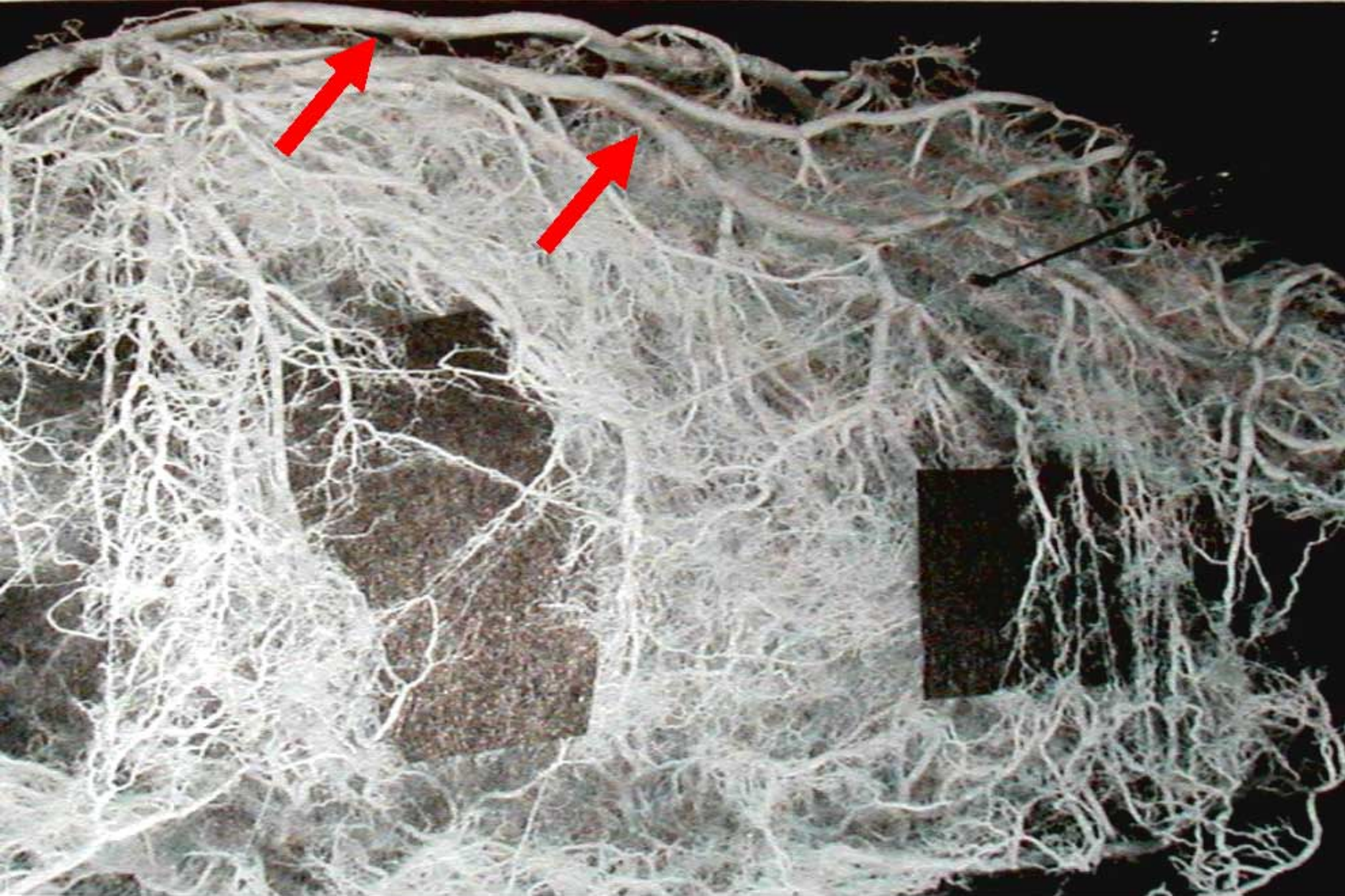
Coronary microvascular dysfunction

Filippo Crea
Institute of Cardiology
Catholic University of the Sacred Heart
Rome, Italy



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REVIEW ARTICLE

MEDICAL PROGRESS

Coronary Microvascular Dysfunction

Paolo G. Camici, M.D., and Filippo Crea, M.D.

Pathophysiology of CMD

Alterations	Conditions
Structural	
Luminal obstruction	STEMI, PCI
Vascular wall infiltration	Fabry's disease
Vascular remodeling and rarefaction	LVH (HCM, Hypertension, Aortic stenosis)
Functional	
Endothelial dysfunction	Risk factors, MVA, SA, NSTE-ACS
SMC dysfunction	Takotsubo syndrome
Autonomic dysfunction	STEMI, MVA
Extravascular	
Extramural compression	LVH (Aortic stenosis, Hypertension, HCM)
Diastolic perfusion time	LVH (Aortic stenosis, Hypertension, HCM)

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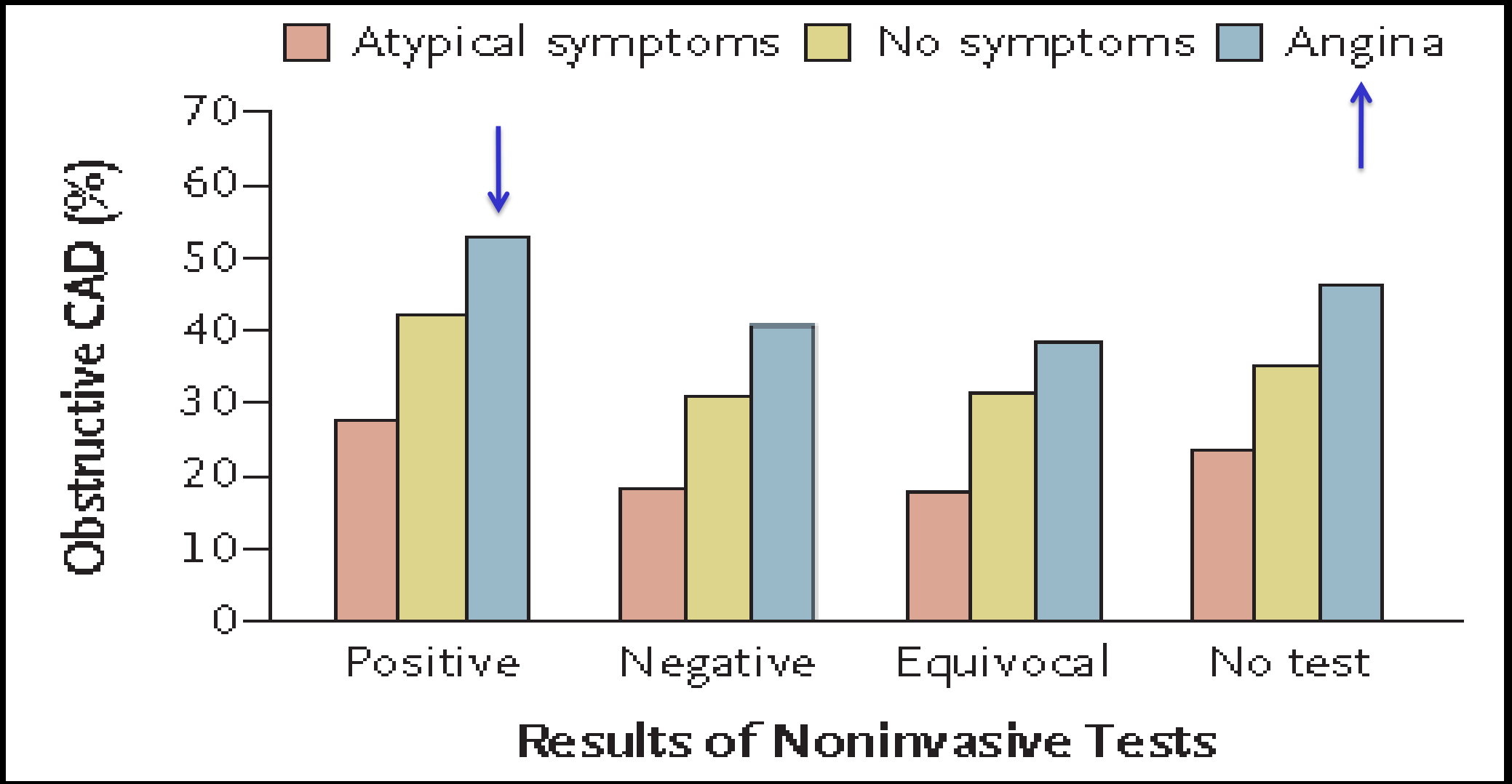
Stable MVA

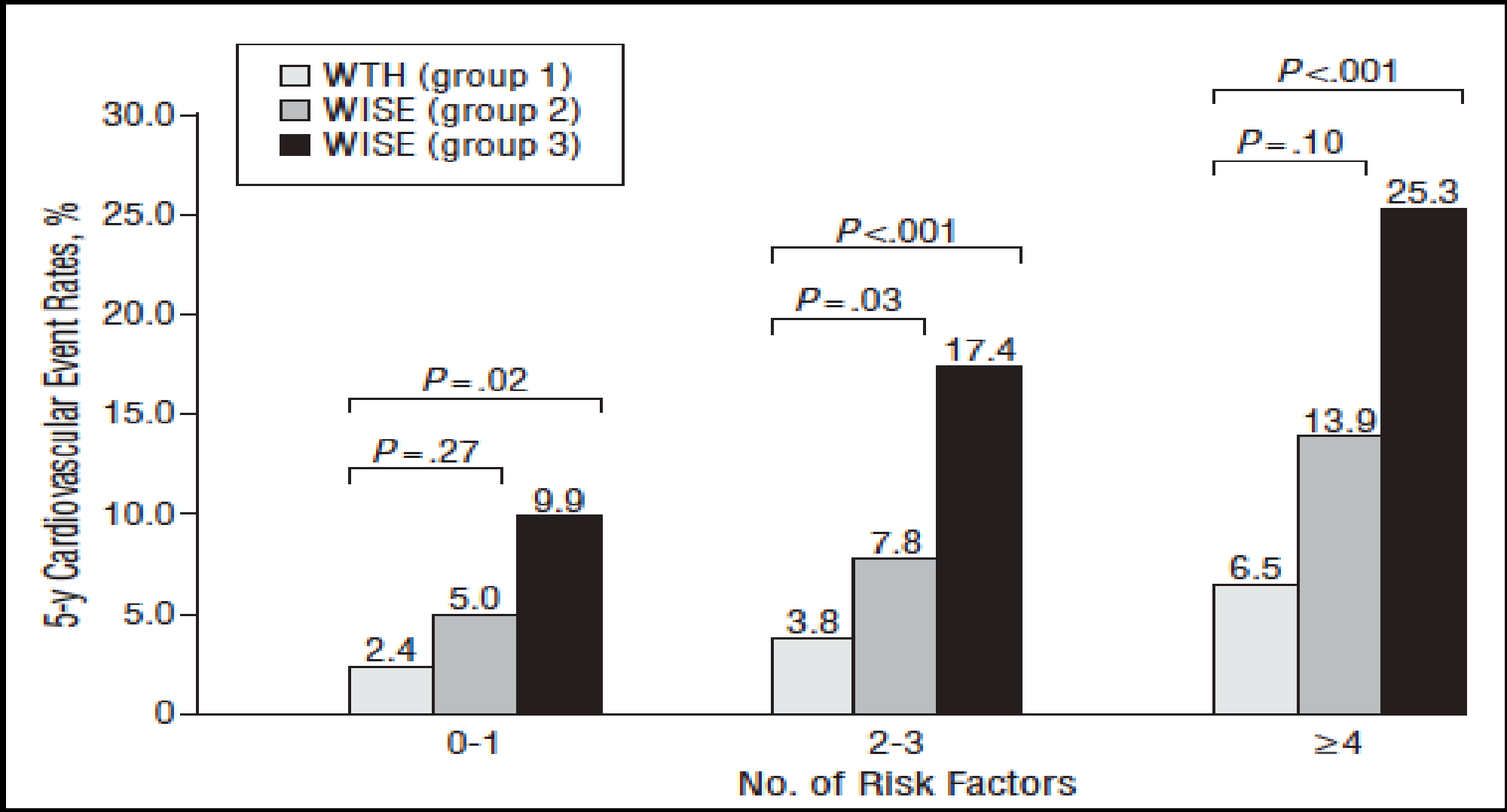
- Angina
- Evidence of stress-induced myocardial ischemia
- Normal coronary arteries
- No coronary spasm

Prevalence of obstructive CAD in relation to symptoms and non invasive testing in patients undergoing selctive coronary angiography (n=398,978)



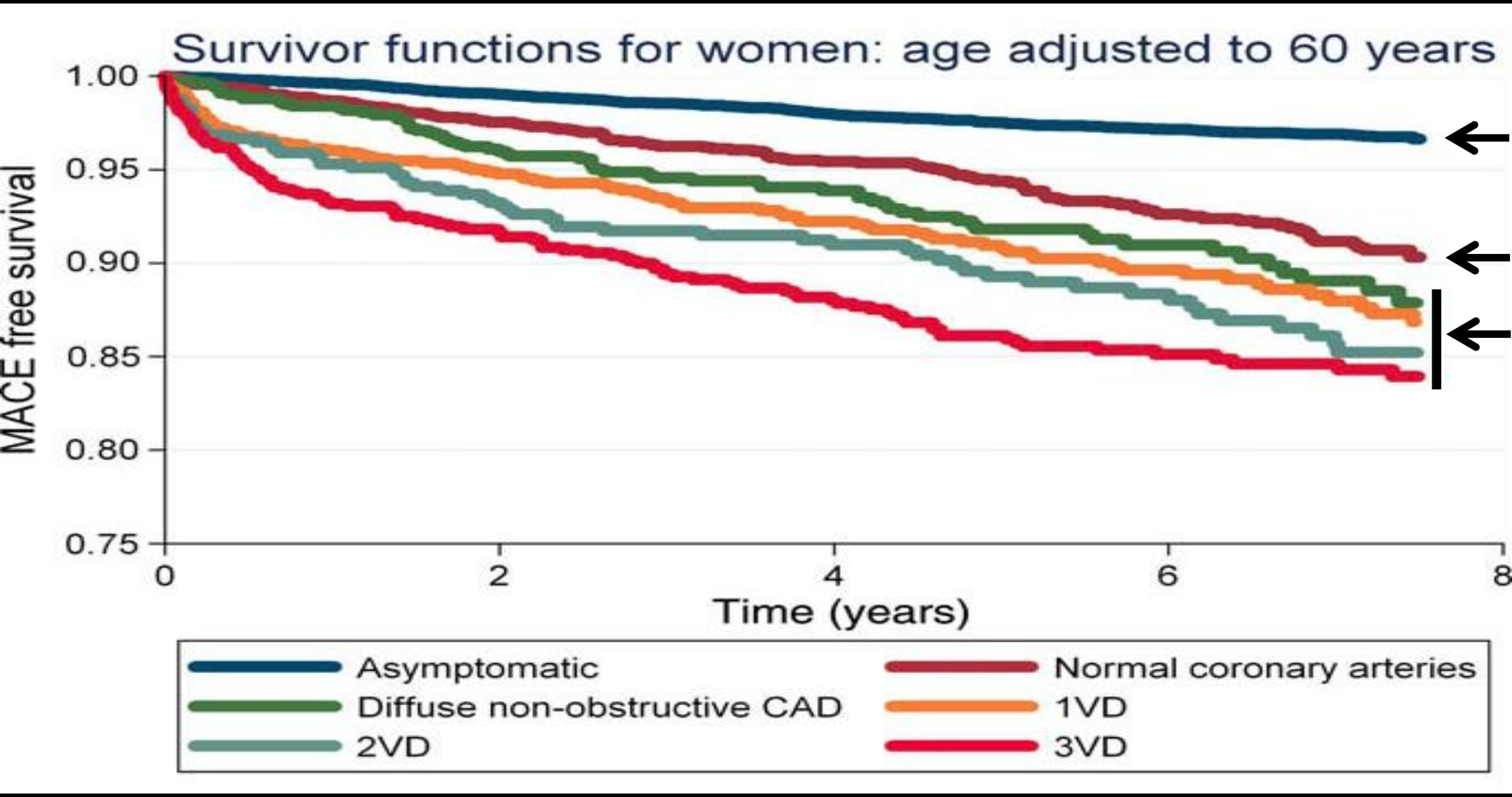
(Patel et al, NEJM 2010)

Stable MVA in women is associated with higher risk of MACEs (n=1,540)



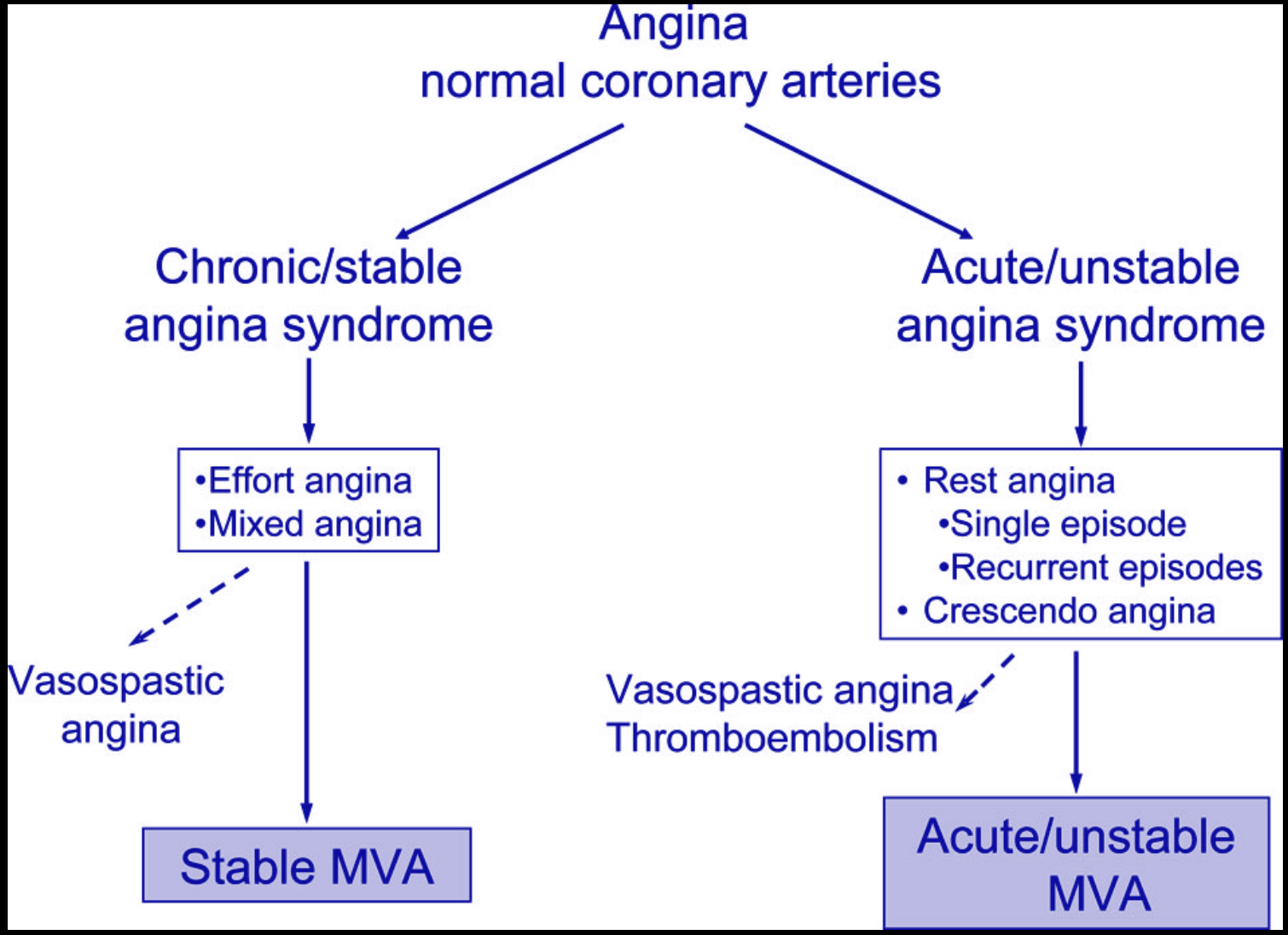
(Gulati et al, Arch Int Med 2009)

SA in women with NCA is associated with higher risk of MACEs (n=4,711)



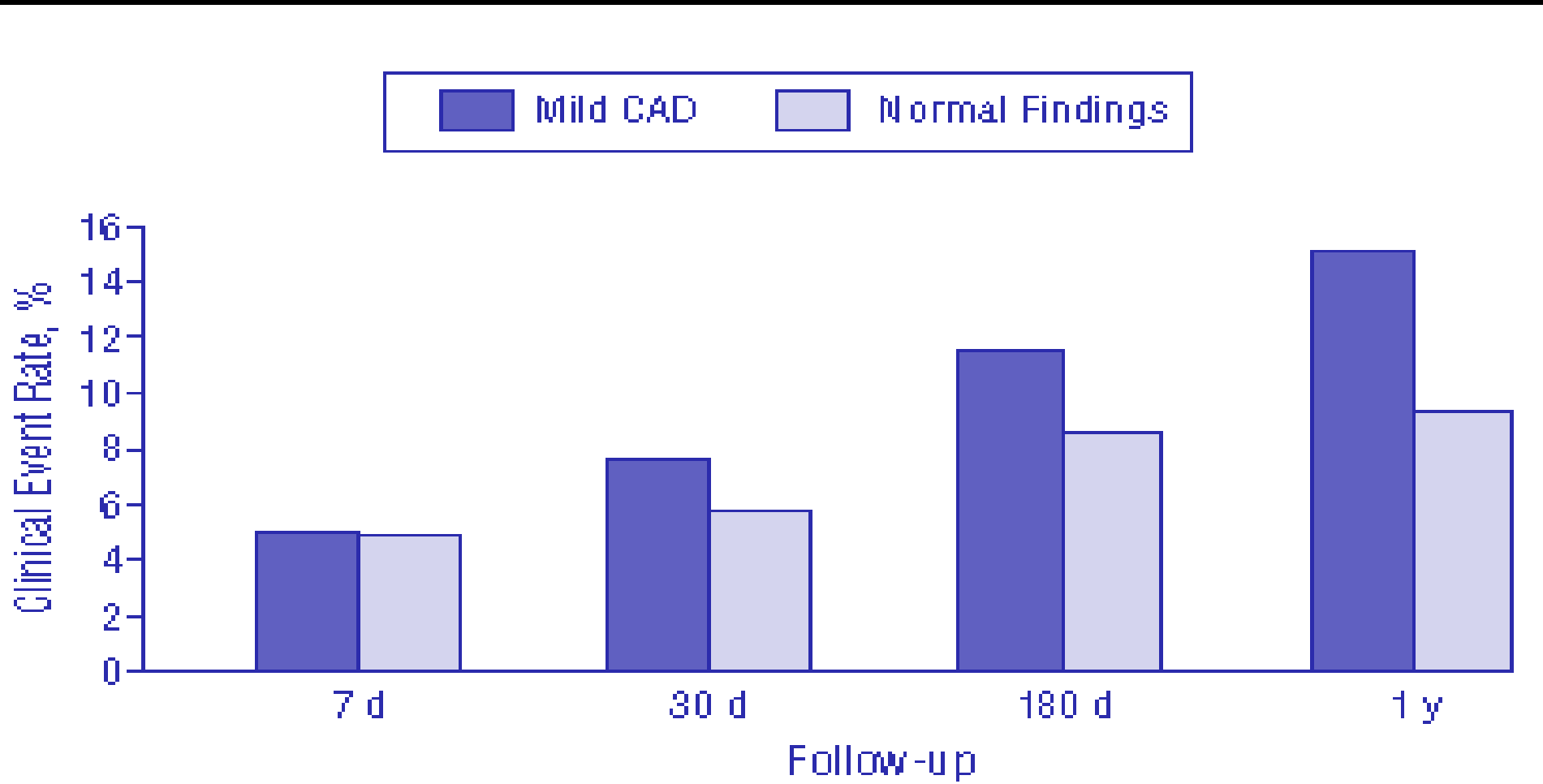
(Prescott et al, EHJ 2011)

Primary Coronary Microvascular Dysfunction



(Lanza and Crea, Circulation 2010)

Outcome of patients with NSTE-ACS and normal coronary arteries or mild CAD enrolled in TIMI 11B, TIMI 16 and TIMI 22 (9.1% di 7656 pazienti)



(Bugiardini et al, Arch Intern Med 2006)

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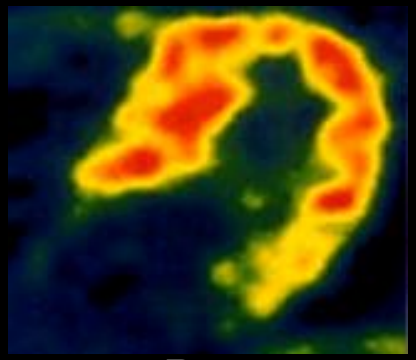
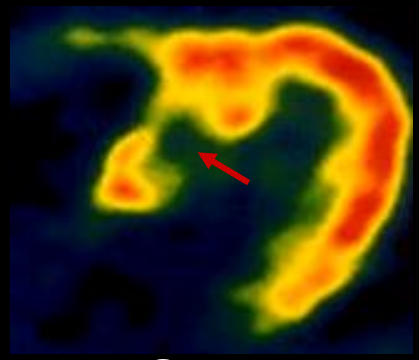
Microvascular dysfunction and prognosis in HCM

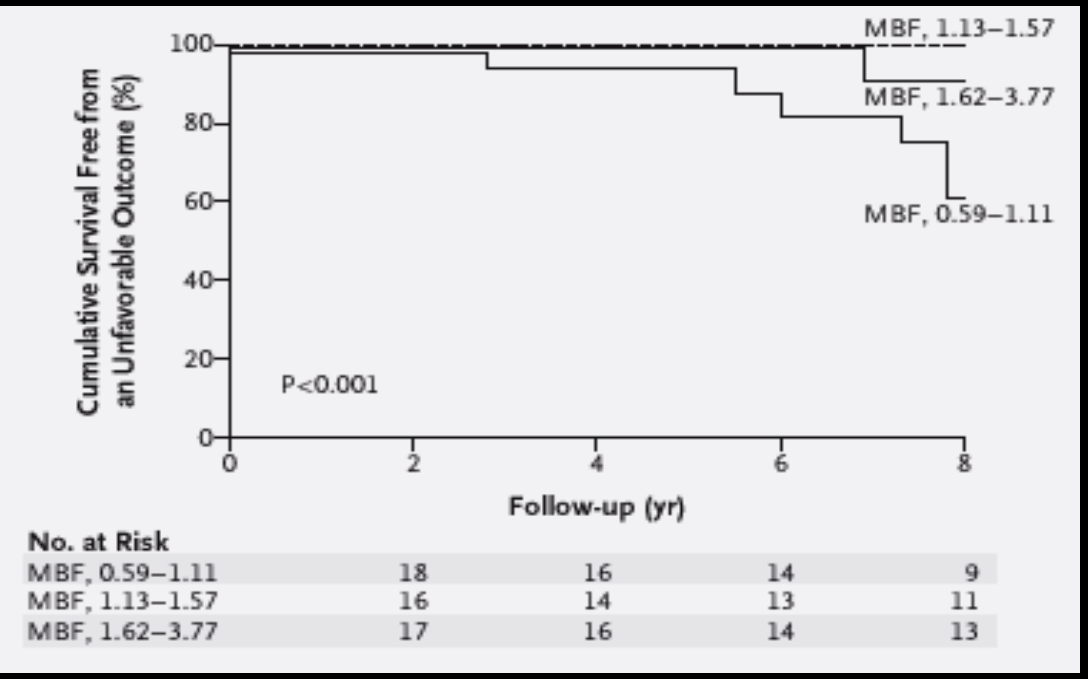
PET myocardial blood flow



Rest

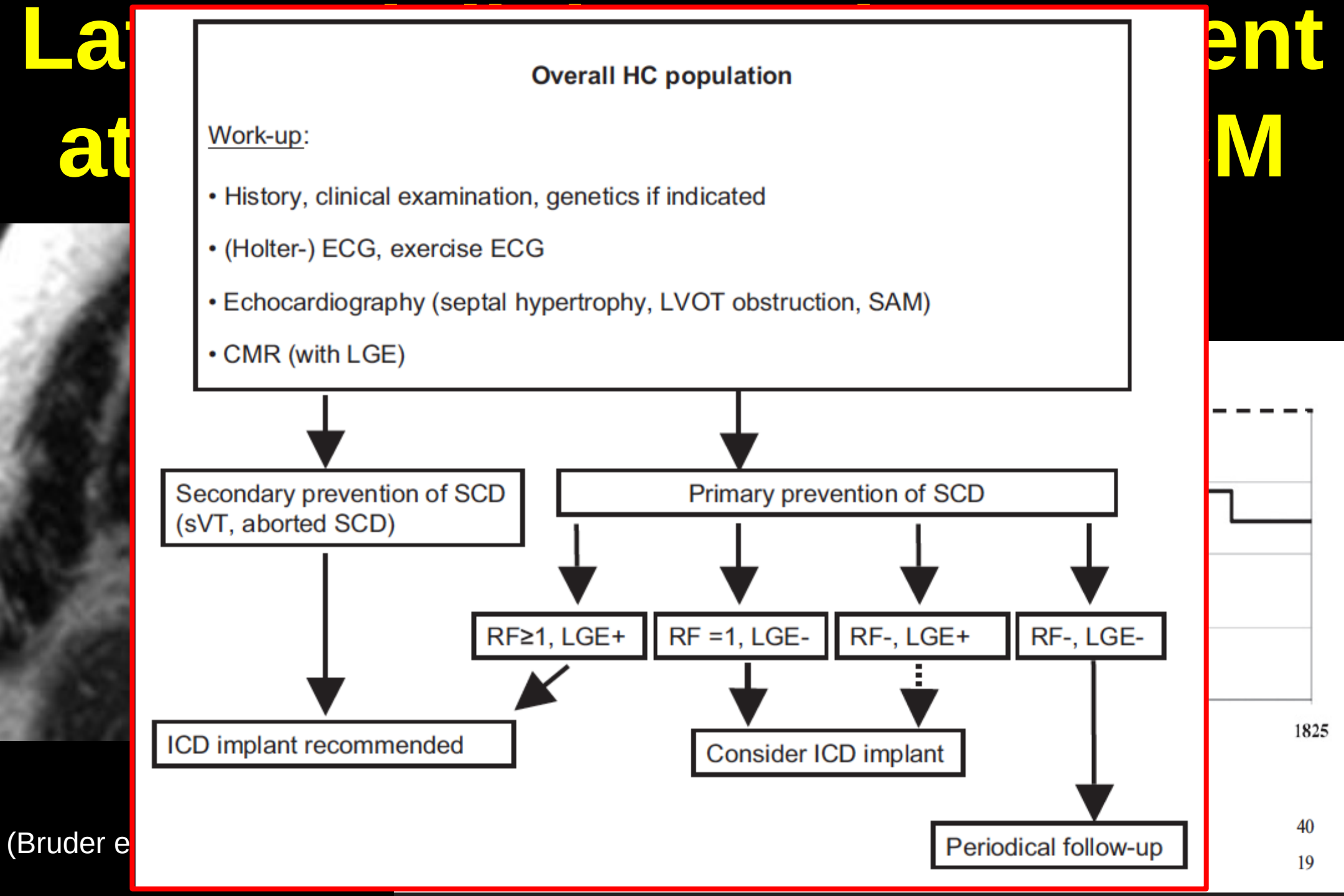


Stress



No. at Risk				
MBF, 0.59–1.11	18	16	14	9
MBF, 1.13–1.57	16	14	13	11
MBF, 1.62–3.77	17	16	14	13

(Cecchi et al, NEJM 2003)


Overall HC population
Work-up:
• History, clinical examination, genetics if indicated
• (Holter-) ECG, exercise ECG
• Echocardiography (septal hypertrophy, LVOT obstruction, SAM)
• CMR (with LGE)
Secondary prevention of SCD (sVT, aborted SCD)
Primary prevention of SCD
RF≥1, LGE+
RF =1, LGE-
RF-, LGE+
RF-, LGE-
ICD implant recommended
Consider ICD implant
Periodical follow-up

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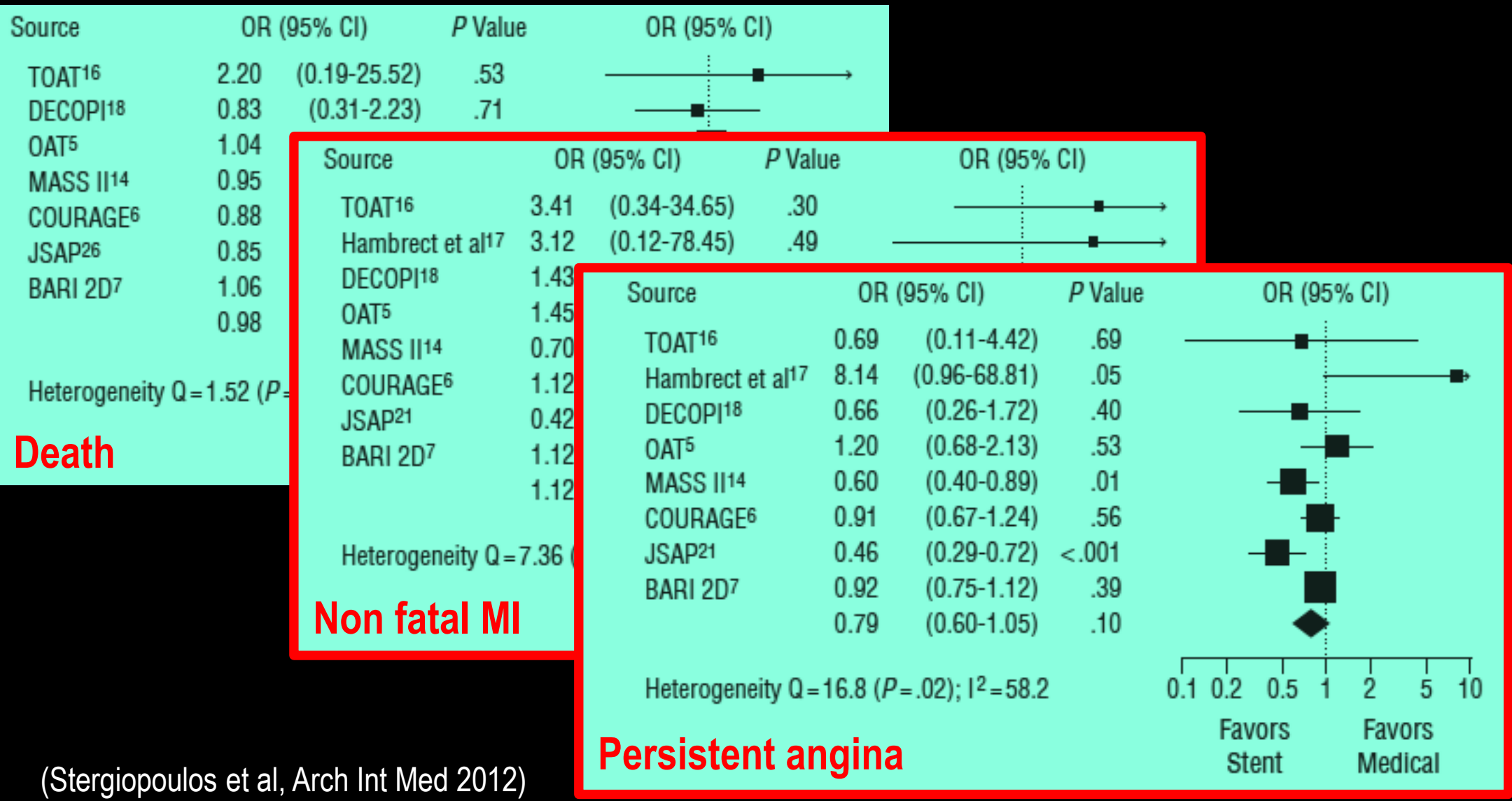
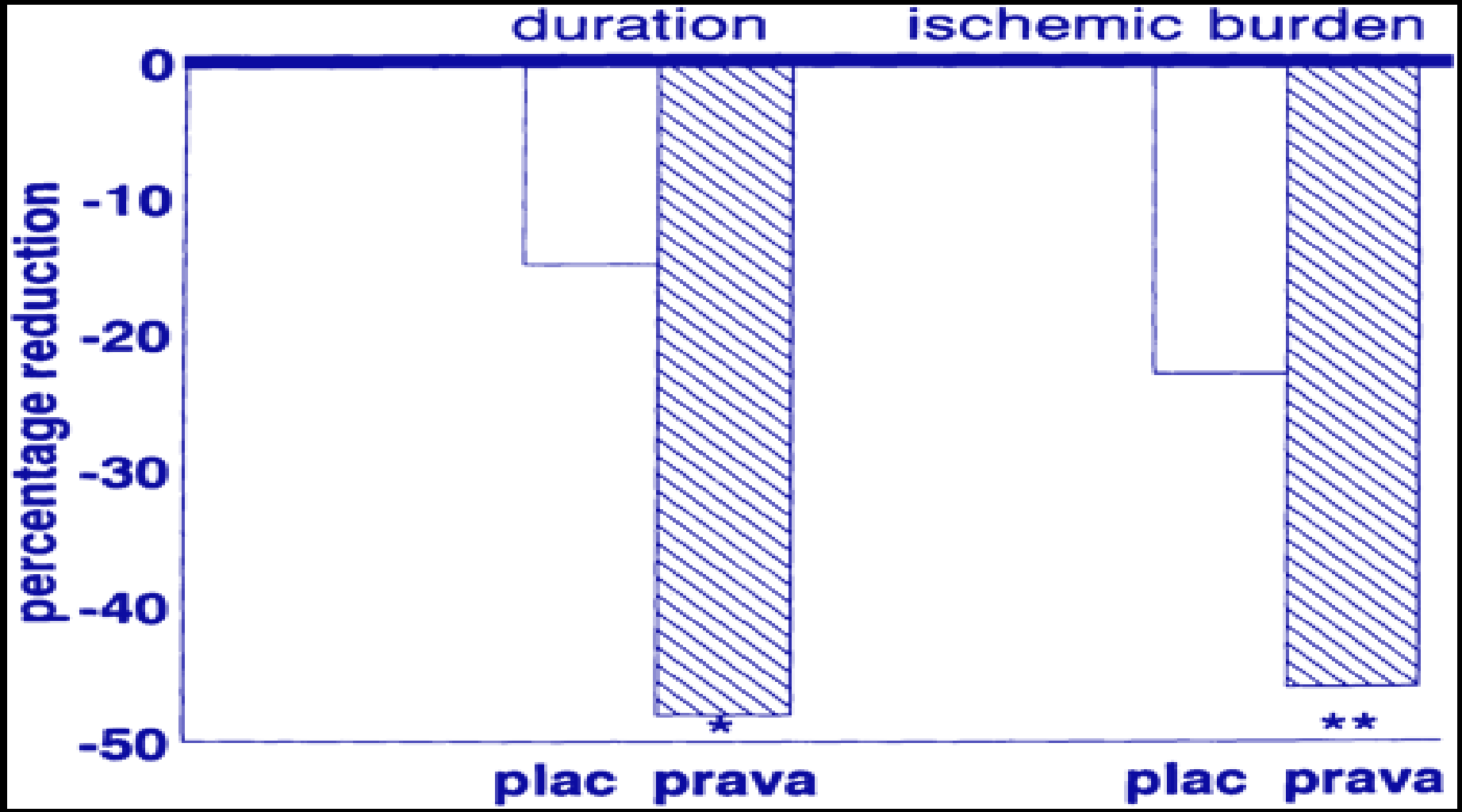
OMT vs OMT + stenting in patients with SA (n=7229)

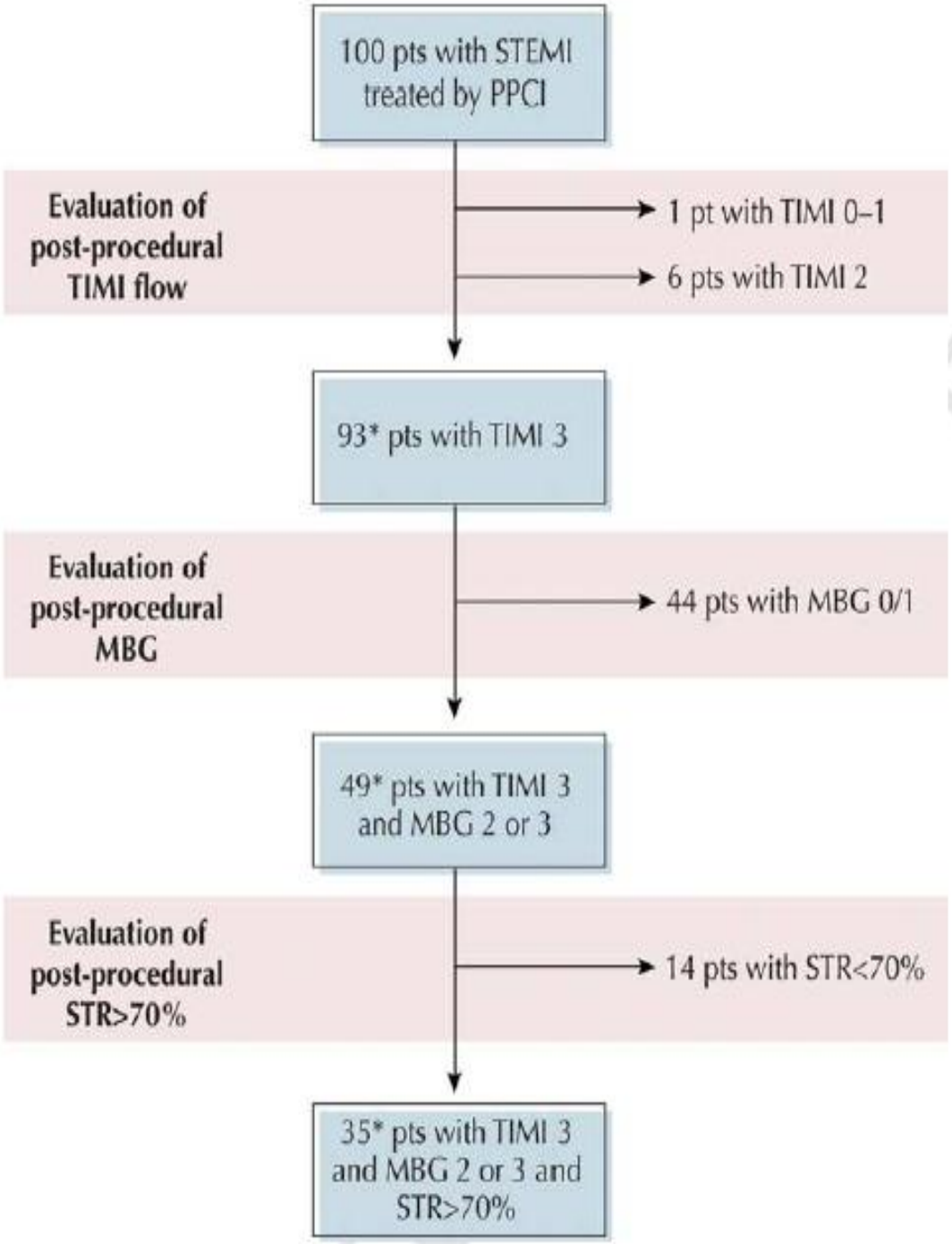


(Stergiopoulos et al, Arch Int Med 2012)

Reduction of transient myocardial ischemia with Pravastatin in stable angina (n=768)



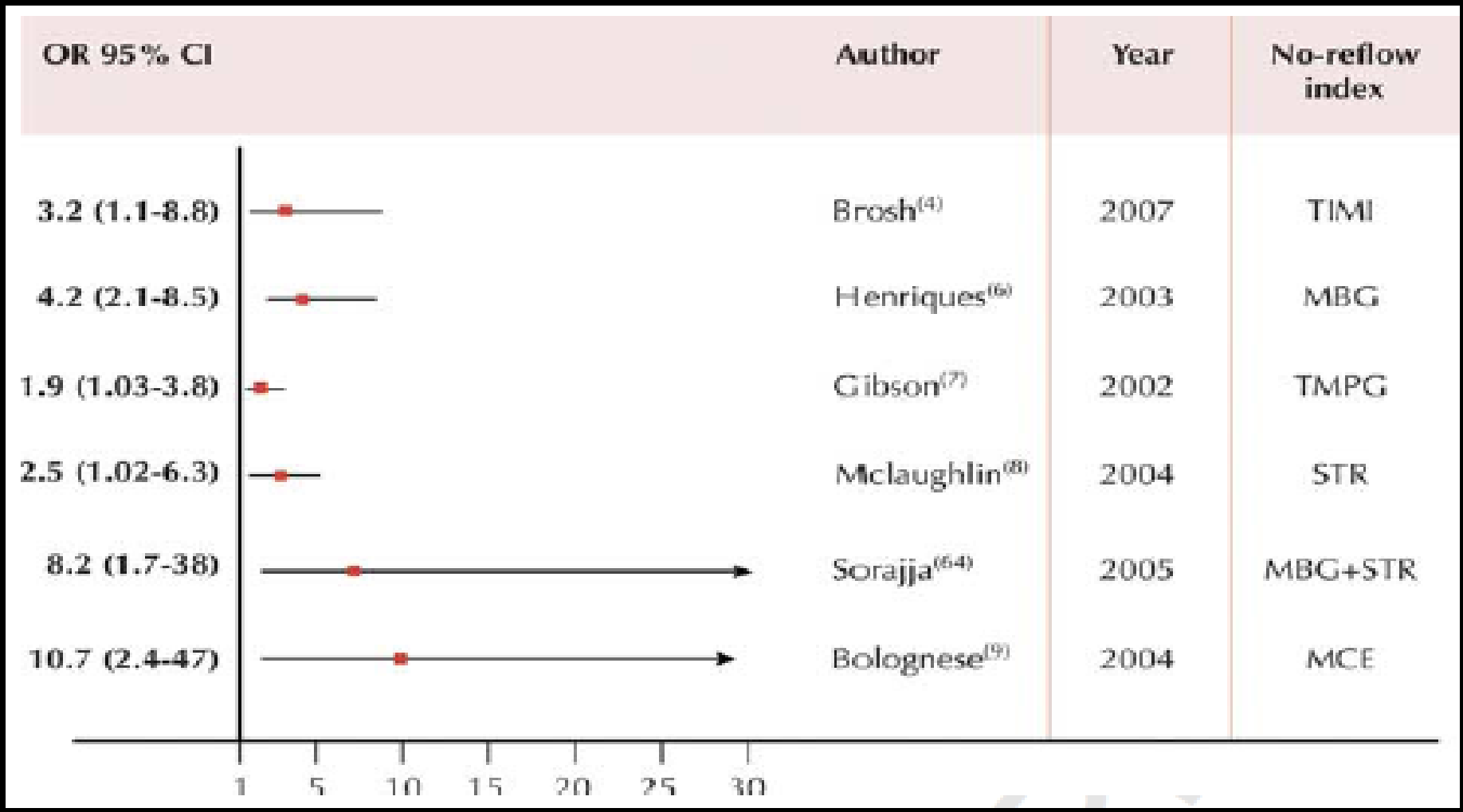
(REGRESS, Circulation 1996)



The illusion of reperfusion after primary PCI

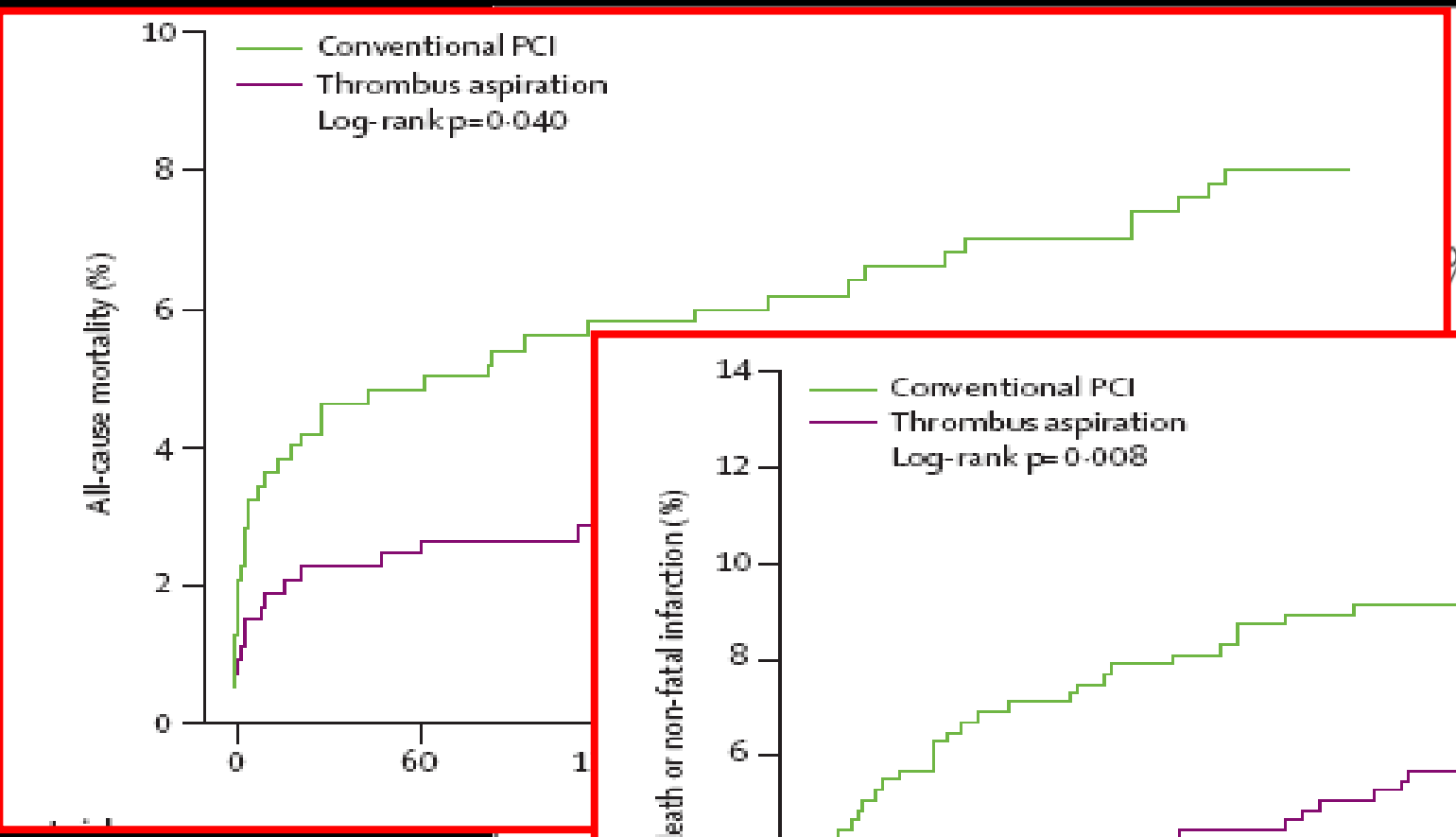
(Niccoli et al. JACC 2009)

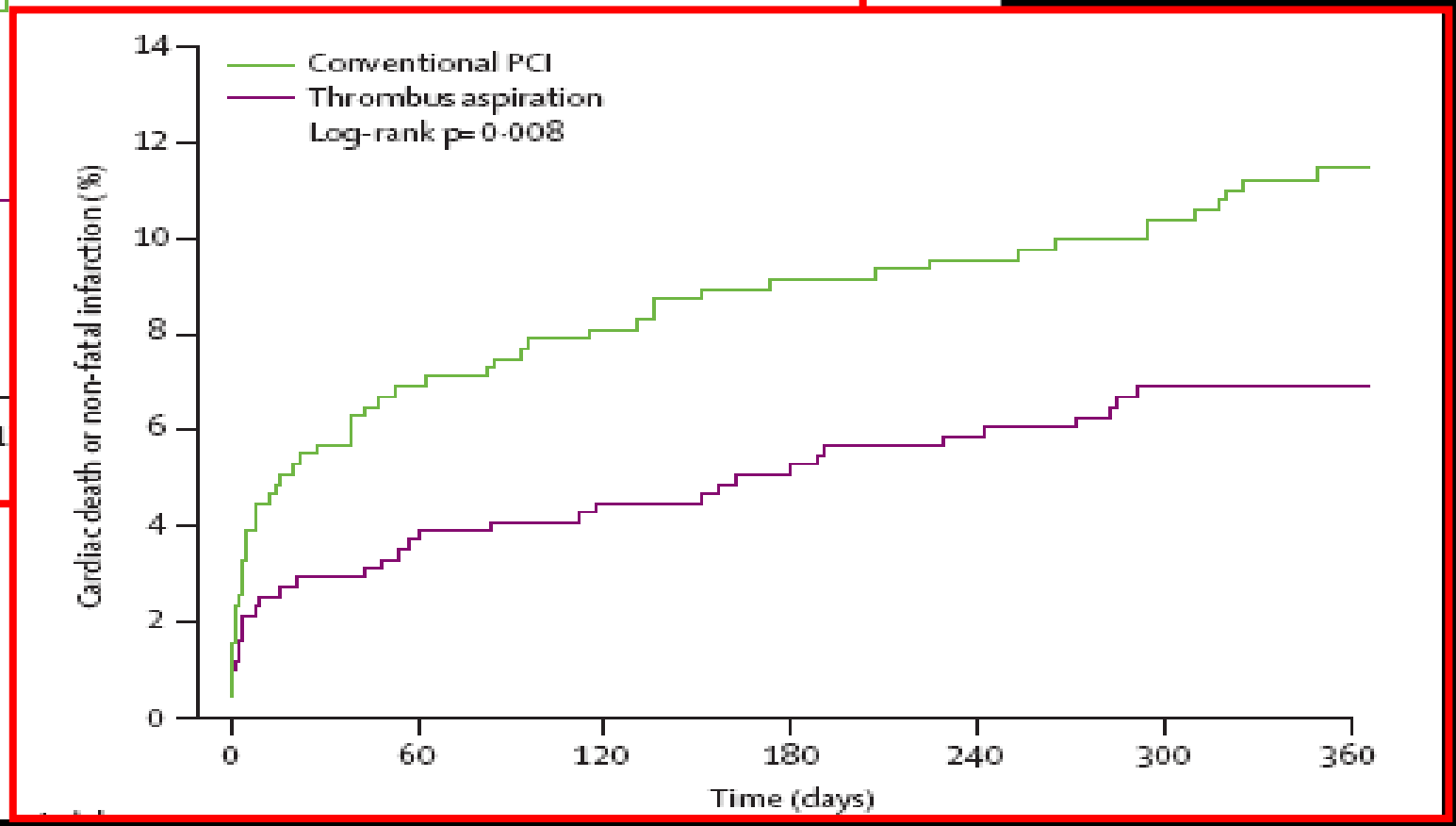
Prognostic Value of MO According to Angiographic, ECG, and Echo-contrastographic Indexes



(Niccoli et al. JACC 2009)

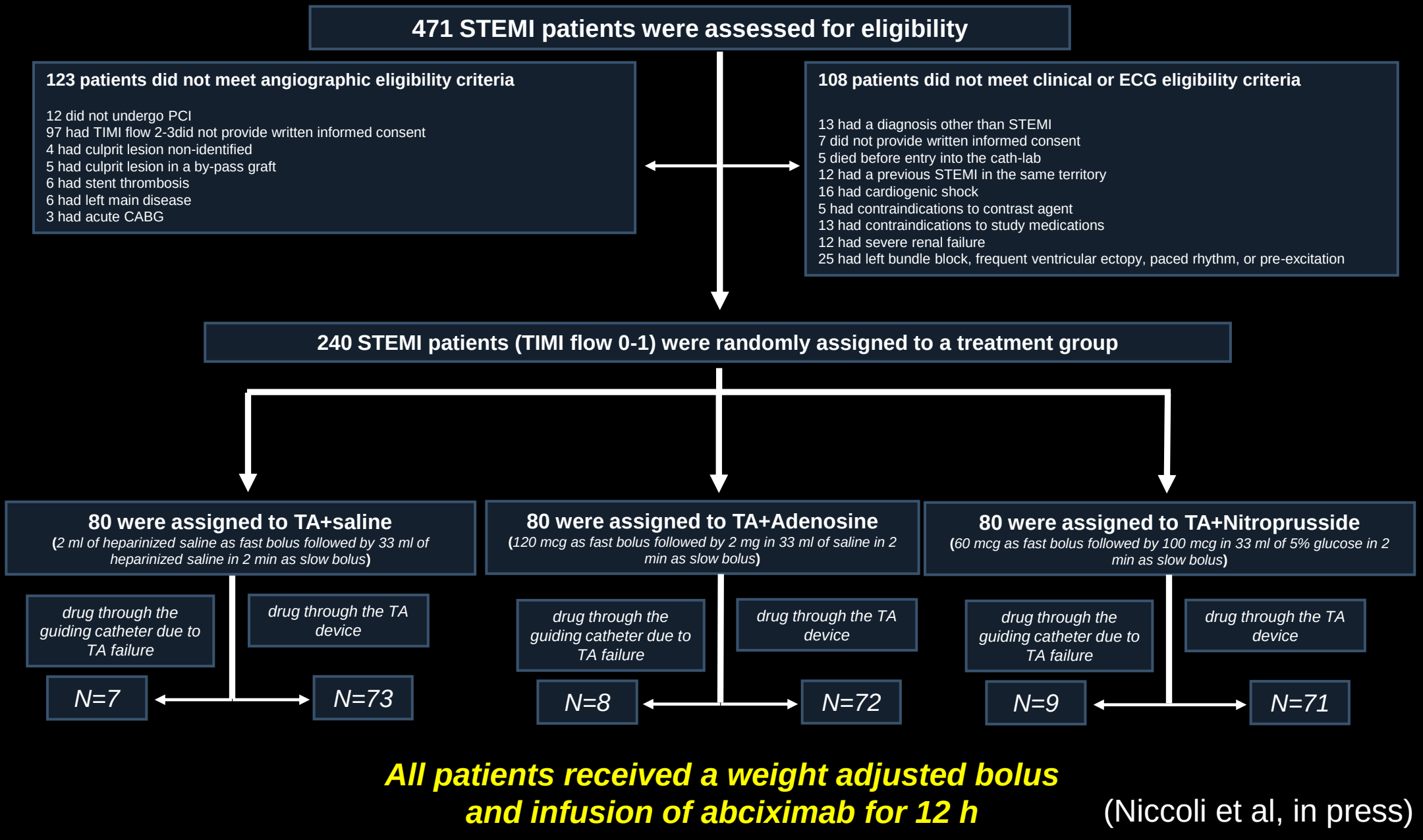
TAPAS trial (n=1060)





(Vlaar et al, Lancet 2007)

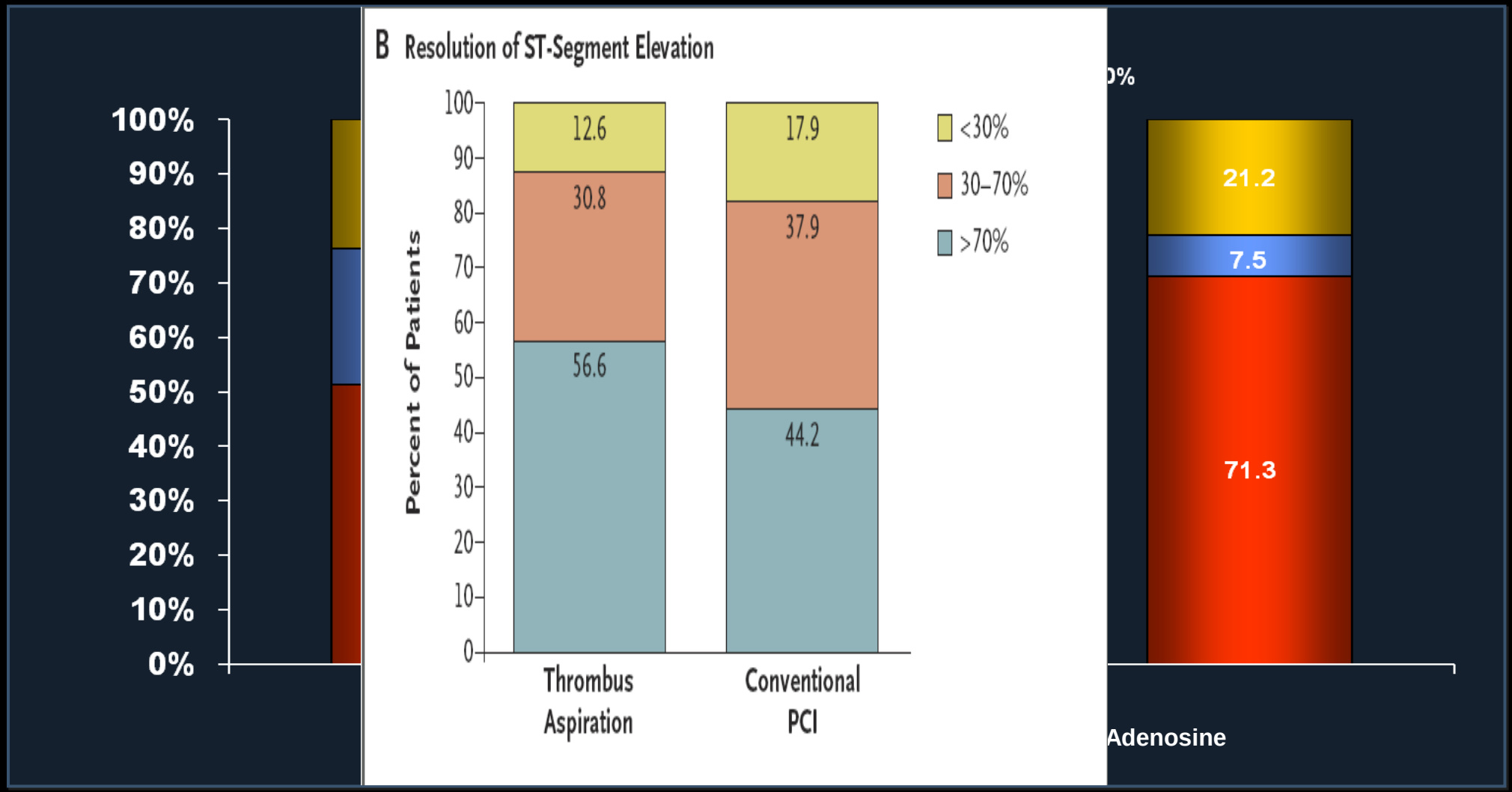
REOPEN-AMI



All patients received a weight adjusted bolus and infusion of abciximab for 12 h

(Niccoli et al, in press)

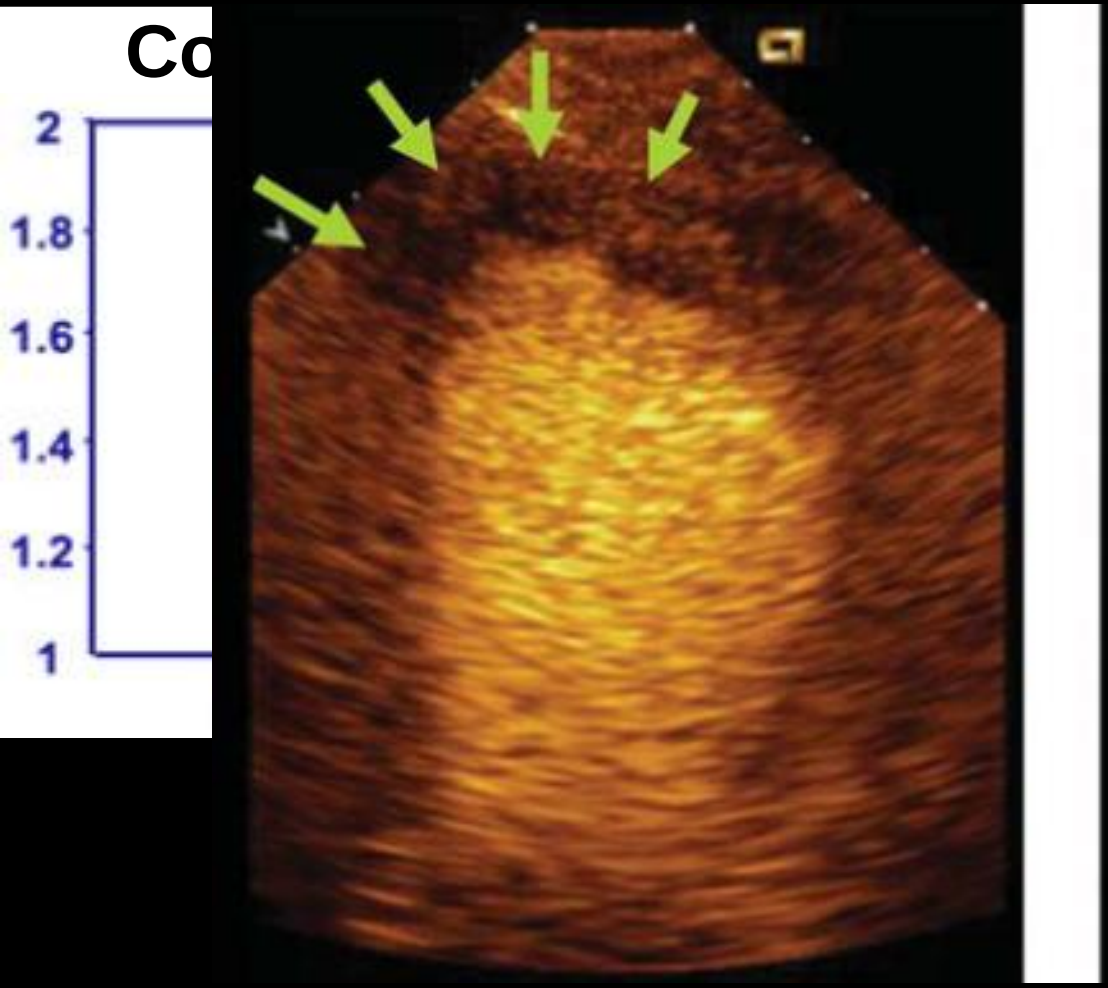
ST-segment resolution



Adenosine vs Saline p = 0.009
Nitroprusside vs Saline p = 0.75

(Niccoli et al, in press)

Coronary microvascular dysfunction in Tako-tsubo syndrome



(Galiuto et al, EHJ 2010)

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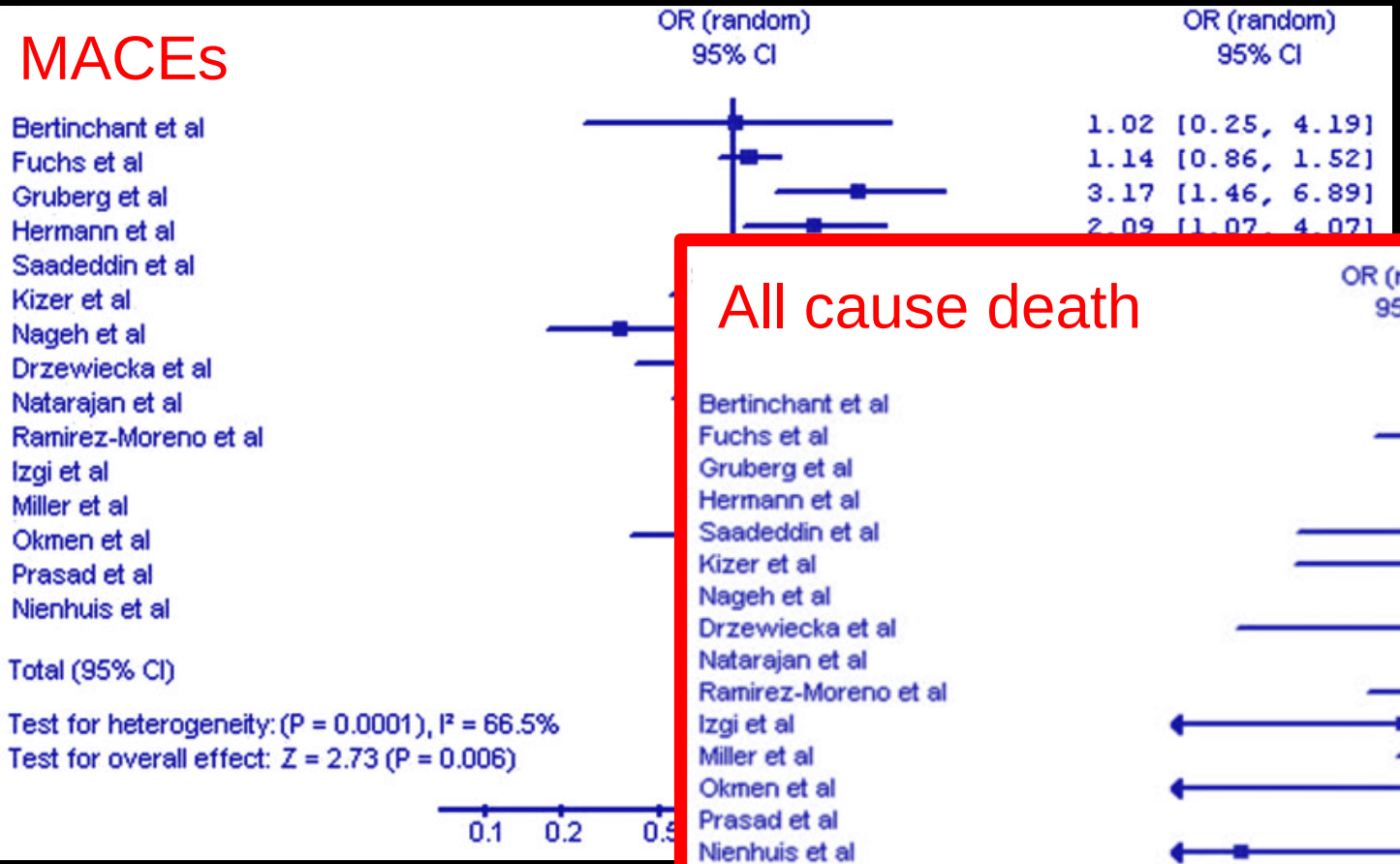
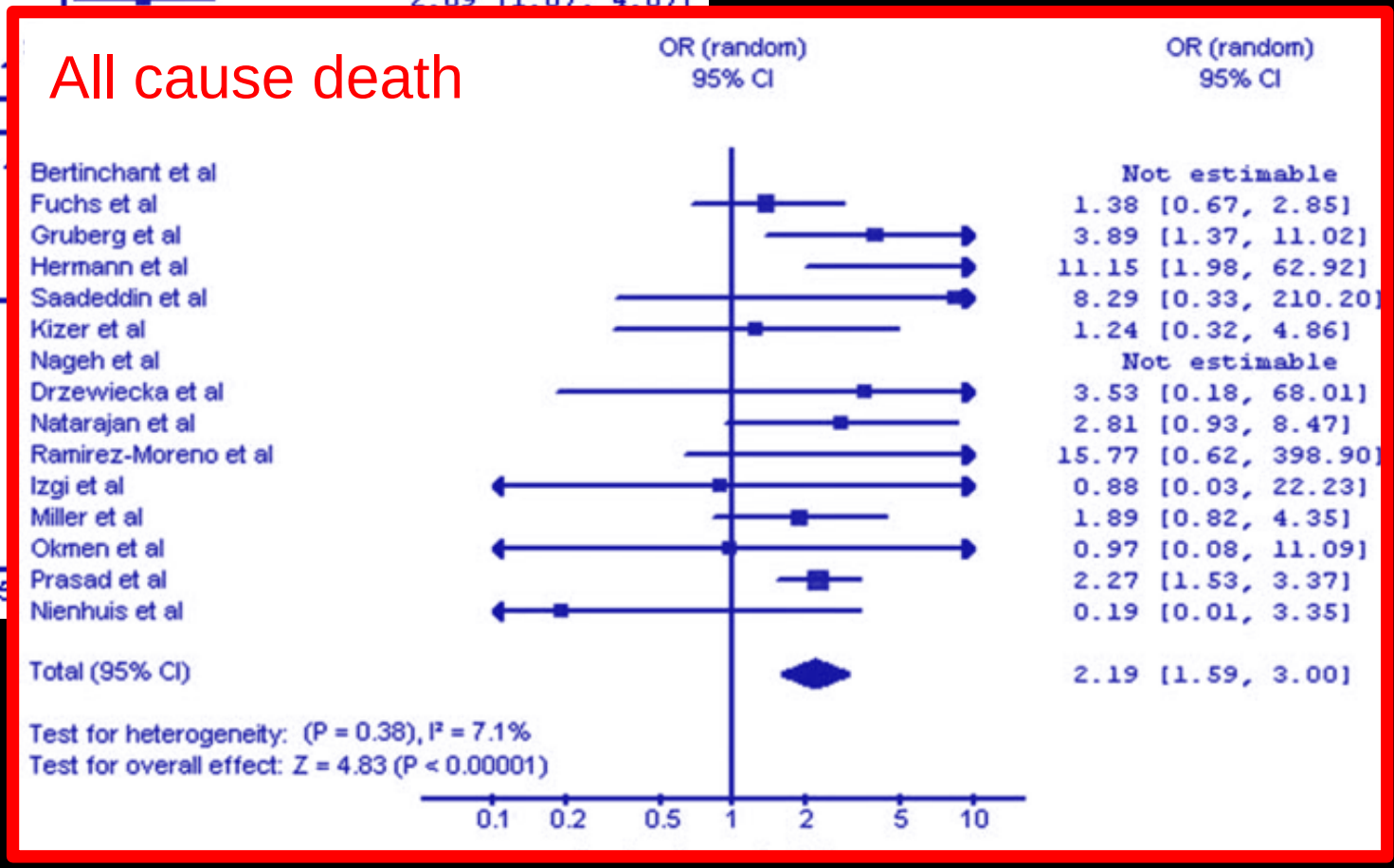
MI after PCI: a meta-analysis of troponin elevation (29%) (n=7578)





(Testa et al, QJM 2009)

Conclusions

- CMD is frequent in a large number of cardiovascular diseases
- The pathohysiolgy is complex as CMD can be caused by structural, functional and extravascular alterations
- In some cases CMD is simply a marker of disease, in other cases is a useful prognostic marker, in other cases it is an important therapeutic target