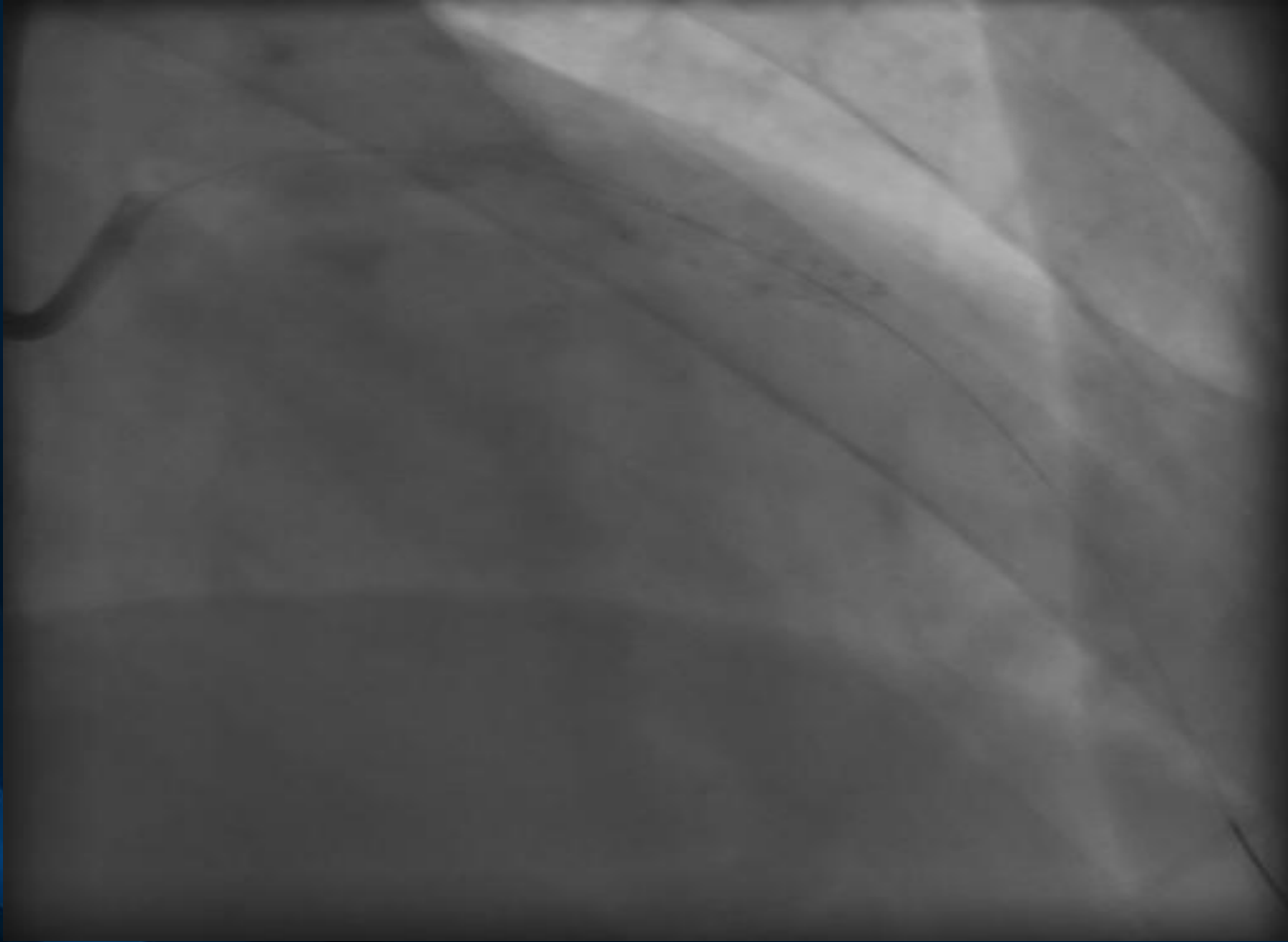


How can we manage “No Reflow” during Primary PCI ?

장기욱

순환기내과

서울성모병원, 가톨릭의대

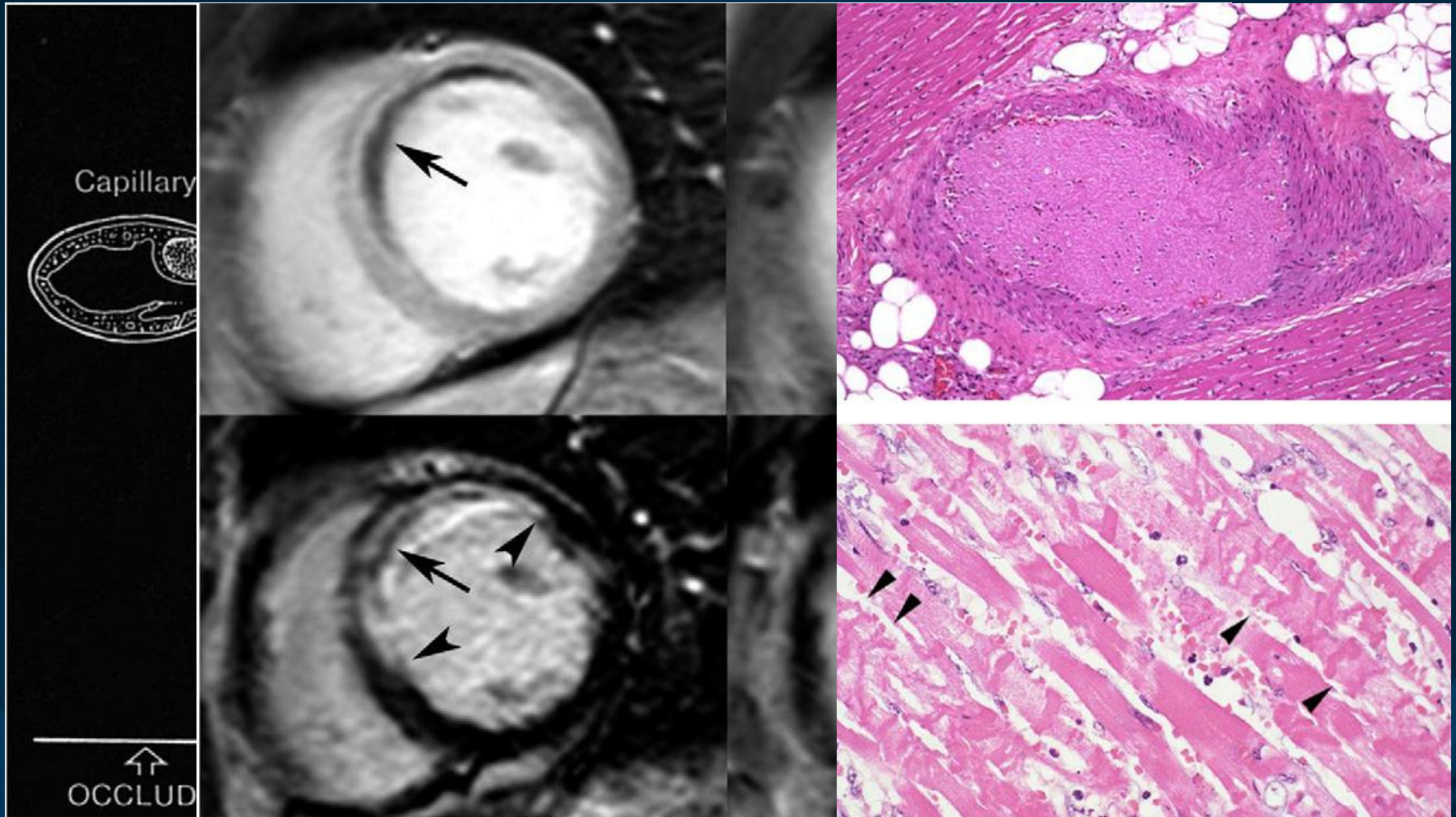


Treatment after no reflow occurs

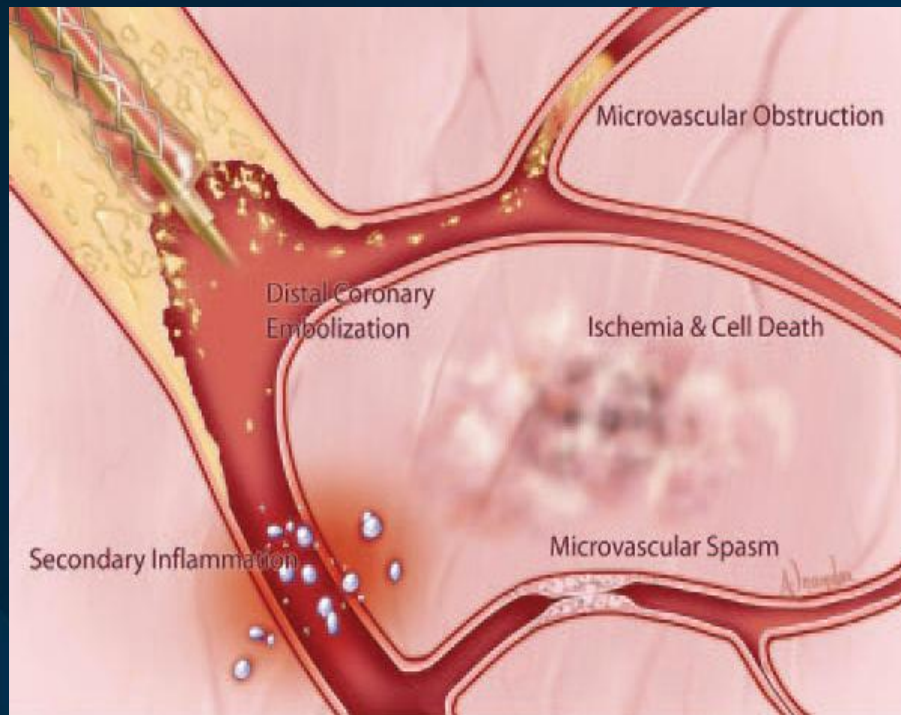
백약이 무효 (Unlikely to succeed)

Prevention is the treatment

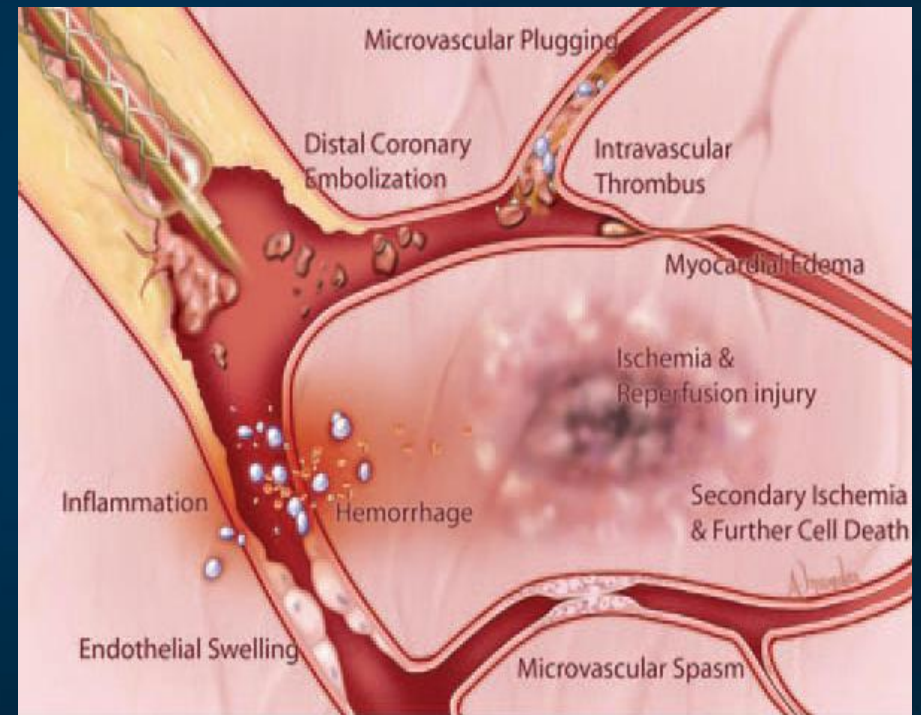
MVO (microvascular obstruction)



Mechanism of no reflow

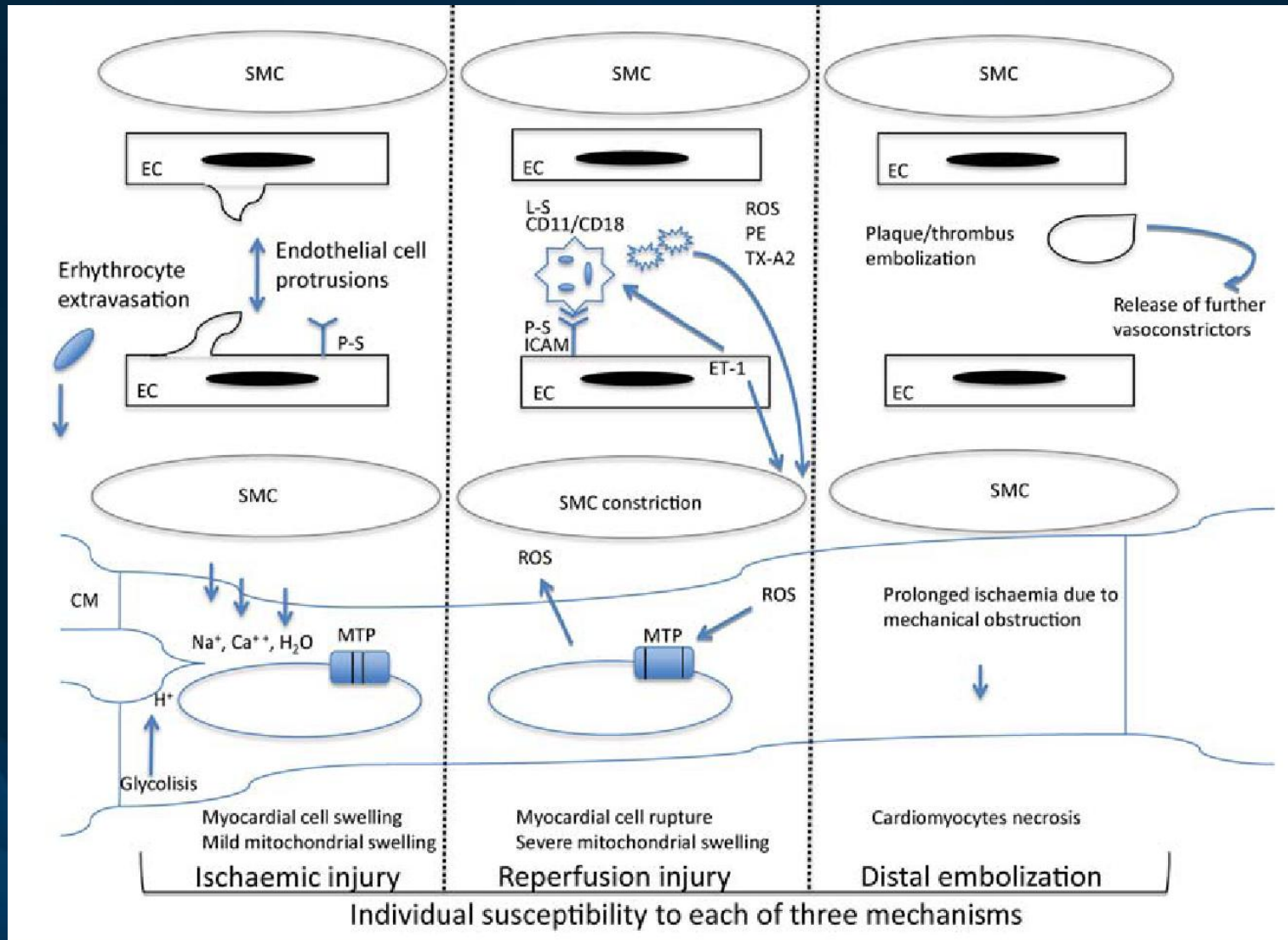


Interventional
no reflow (MVO)

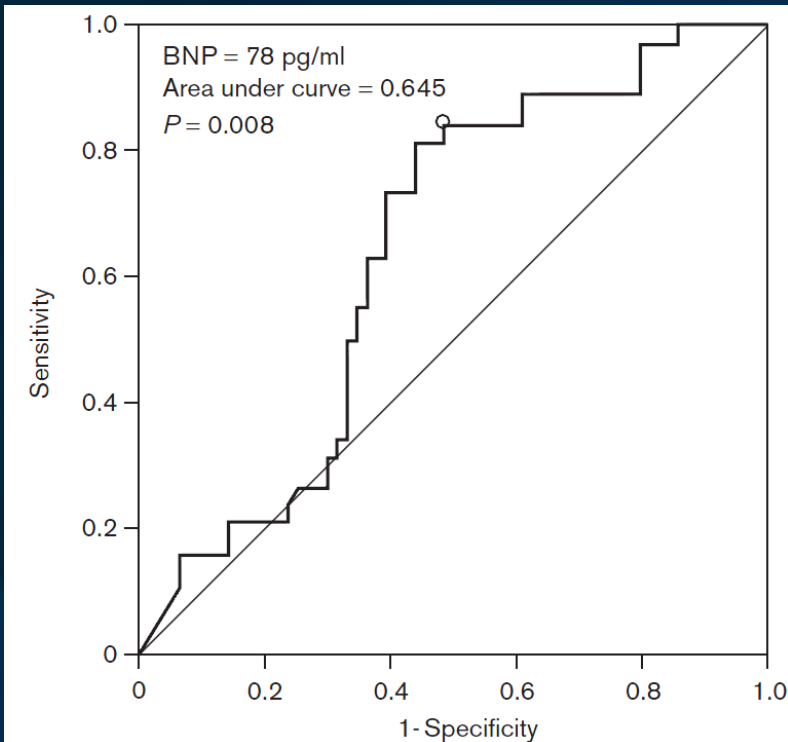


Reperfusion
no reflow (MVO)

Mechanism of no reflow



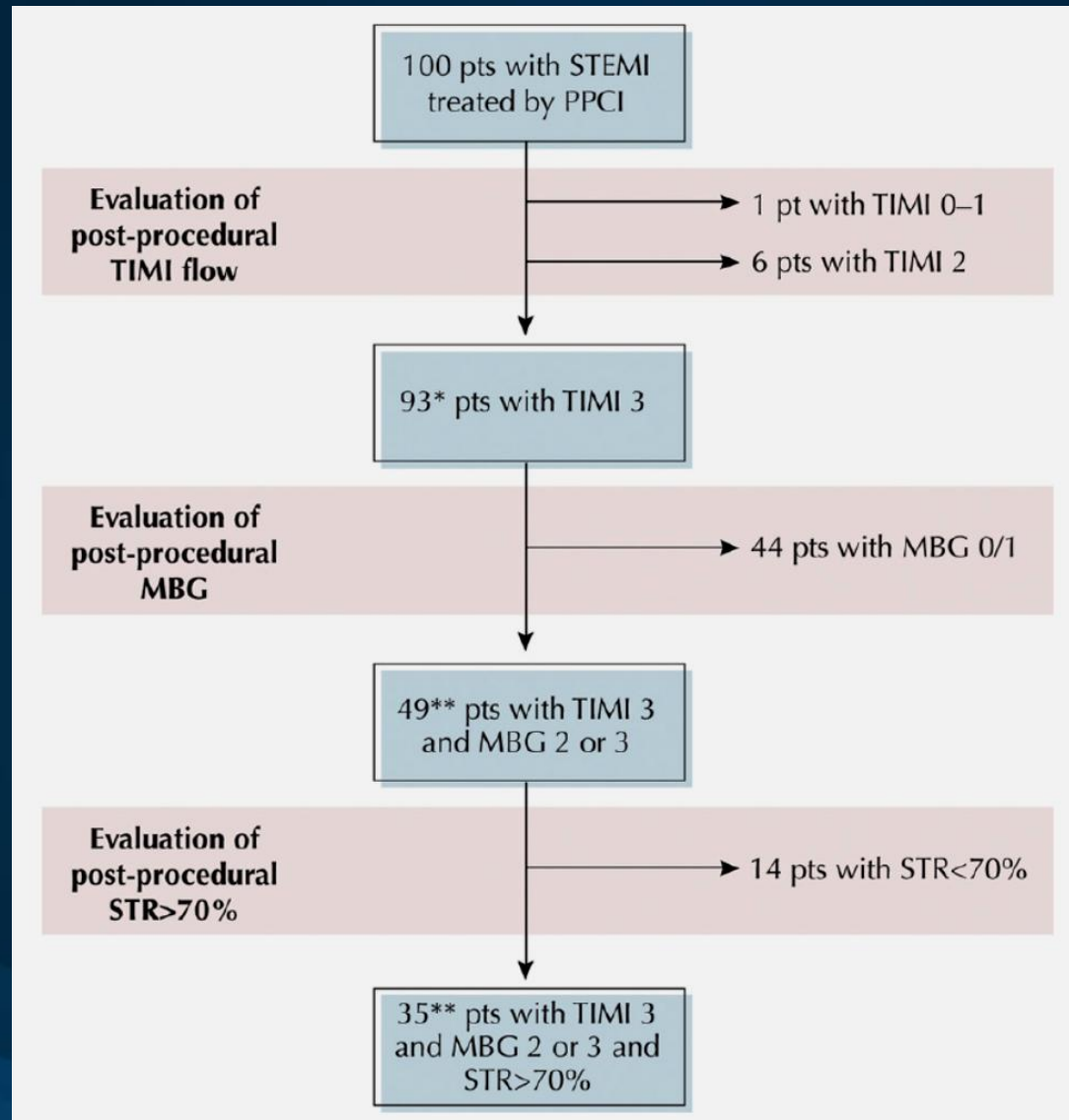
Extent of ischemic injury & no reflow



	BNP 80 pg/mL	High BNP (n=43)	Low BNP (n=59)	P
ST resolution				<0.01
≥70 %		21%	52.5%	
< 70%		79%	47.5%	
TIMI frame count		53.7±19.7	44.5±15.5	0.04
MBG				0.01
2, 3		67.5%	89.7%	
0, 1		32.5%	10.3%	

Seo SM, Chang K, Coron Artery Dis 2011;22:405

Prevalence of no reflow



Definition

- Epicardial coronary reperfusion without myocardial reperfusion
- Microvascular obstruction & resultant reduced myocardial flow after opening an occluded artery

Angiographic definition

- TIMI grade 0 to 2
- TIMI grade 3 with myocardial blush grade (MBG) 0-1

COREA-AMI registry

- COREA-AMI : Convergent Registry of cAtholic and chonnAm university for AMI
- Including STEMI and NSTEMI
- Data registered from January, 2004 to December 2009. in Catholic university and Chonnam university
- Total number of patients: 5694
- Median follow up: 25 months

Treatment modality of COREA-AMI registry

	No-reflow (n=492)	Reflow (n=2745)	p for trend
Treatment modality			0.001
Conventional PCI	350 (71.3)	1785 (65.2)	
GPI only	79 (16.1)	578 (21.1)	
Thrombus aspiration only	47 (9.6)	232 (8.5)	
TA + GPI	15 (3.1)	142 (5.2)	

Clinical outcome of no reflow

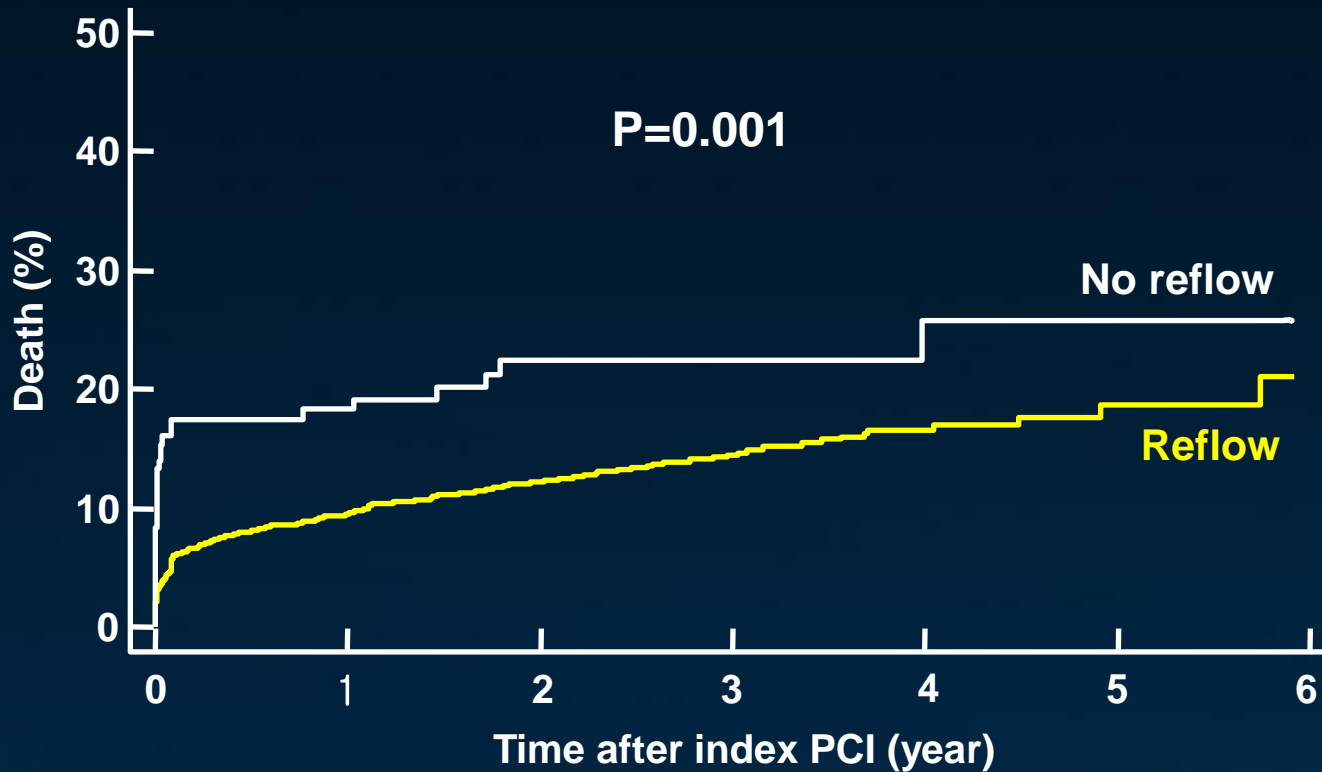
Clinical outcomes	Reflow (n=2745)	No reflow (n=492)	p Value
Death	303 (11.0)	82 (16.7)	<0.001
MI	32 (1.2)	9 (1.8)	0.226
TVR	195 (7.1)	32 (6.5)	0.631
Composite of Death, MI	320 (11.7)	88 (17.9)	<0.001

Hazard ratio of no reflow

Events	Unadjusted HR	95% CI	p Value	Adjusted HR*	95% CI	p Value
Death	1.492	1.166-1.908	0.001	1.478	1.045-2.090	0.027
MI	1.273	0.554-2.921	0.57	1.491	0.599-3.708	0.39
TVR	0.898	0.618-1.307	0.575	0.88	0.577-1.343	0.553
Composite of Death, MI	1.513	1.194-1.917	0.001	1.334	0.949-1.875	0.097

* Adjusted for age, sex, DM, hypertension, hyperlipidemia, current smoking, prior MI, hsCRP, Killip class, LV ejection fraction, culprit vessel, multivessel disease, treatment modality

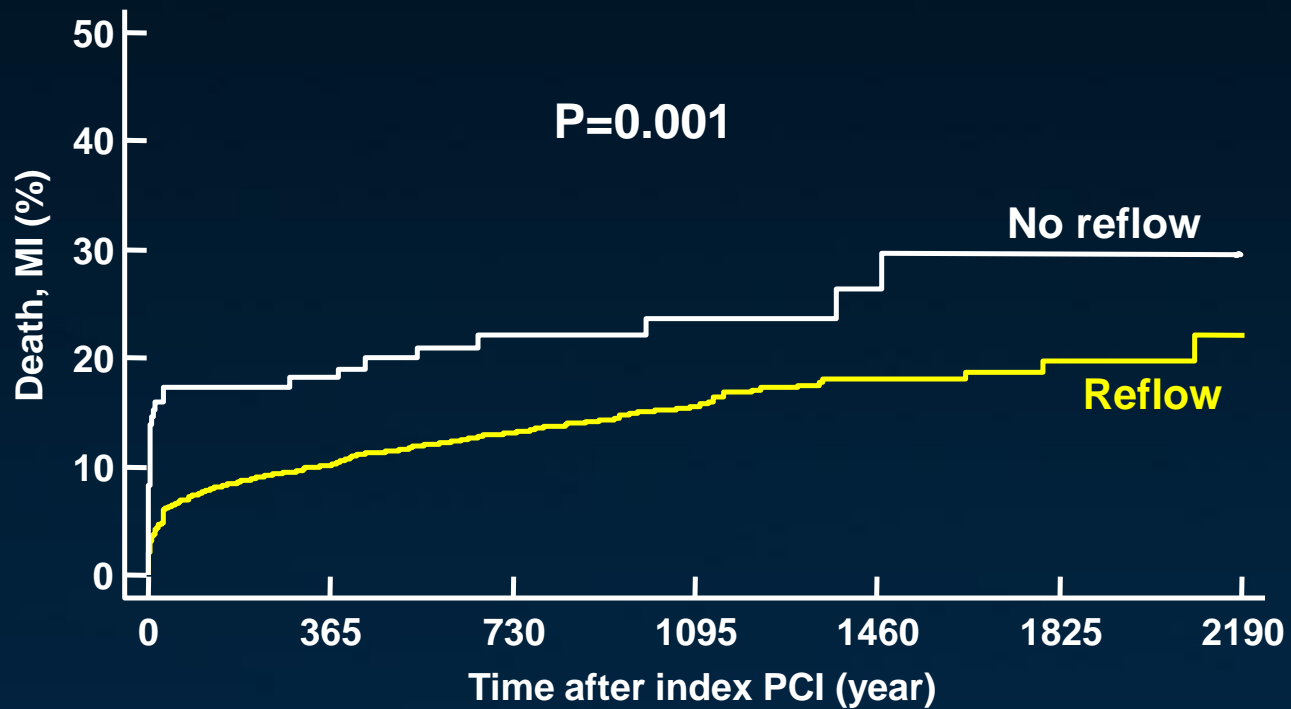
Kaplan-Meier curve : Death



Number at risk

No	2745	2049	971	450	214	71	15
Yes	492	397	257	179	93	35	1

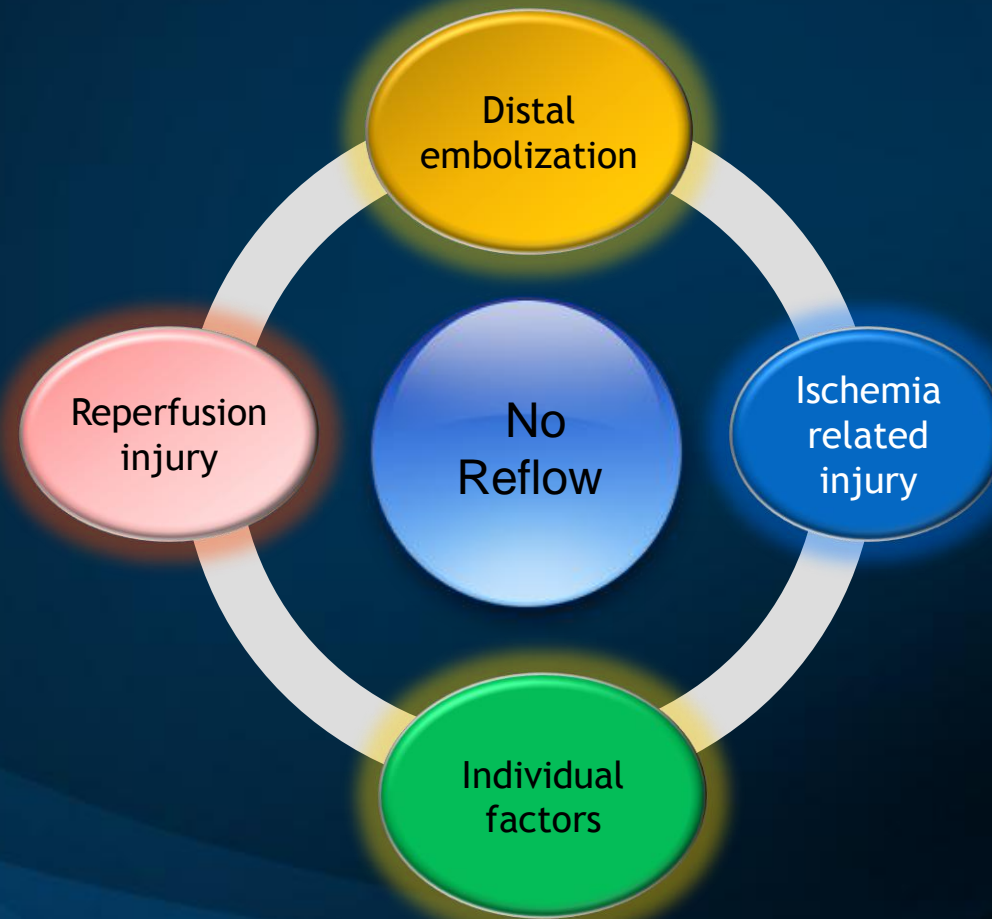
Kaplan-Meier curve : Death, MI



Number at risk

No	2745	2036	958	441	208	70	15
Yes	492	377	239	162	77	33	1

Prevention and treatment of no reflow (MVO)



Prevention of distal embolization

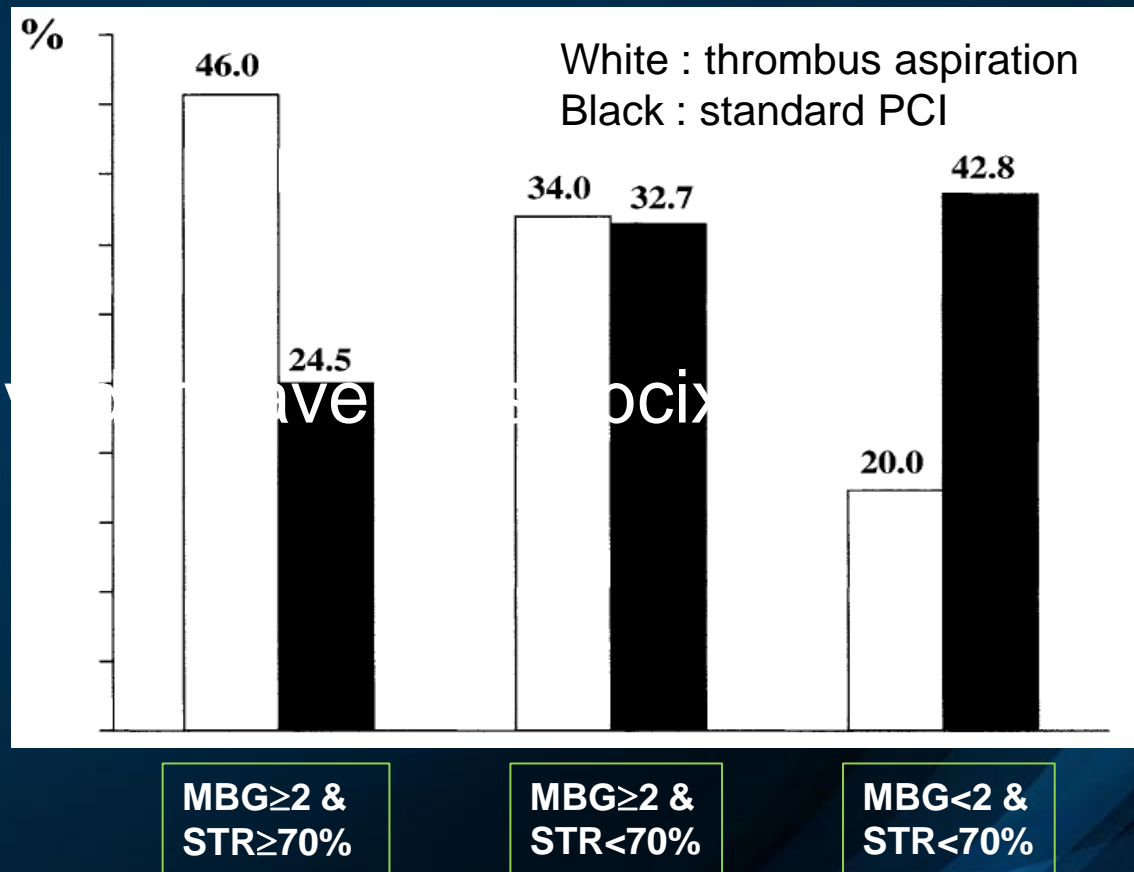
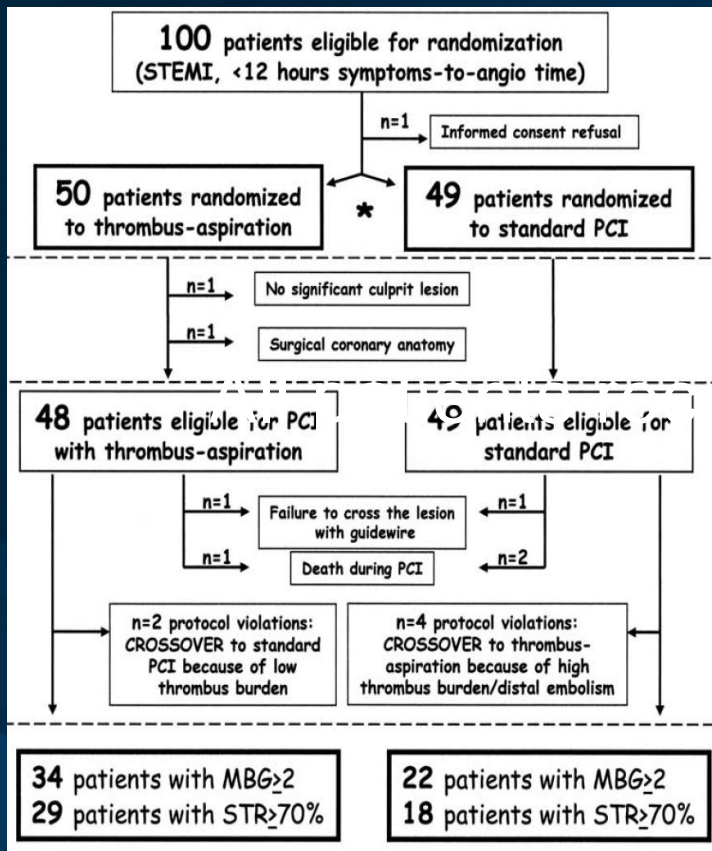
Manual thrombus aspiration

±

intravenous or intracoronary
abciximab

Manual thrombus aspiration

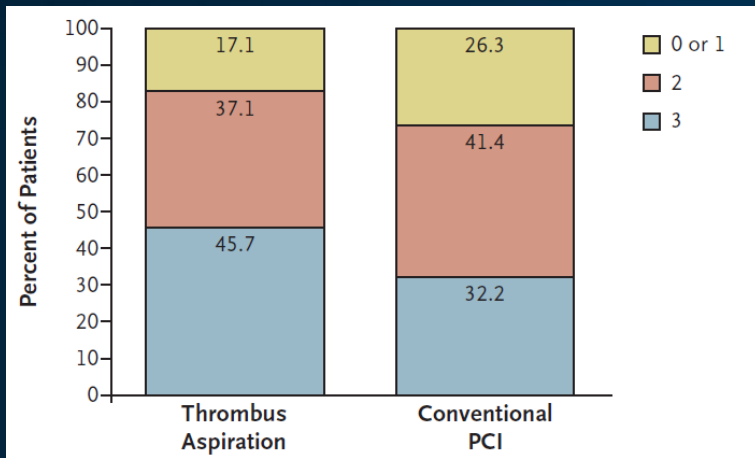
REMEDIA : 1ST RCT



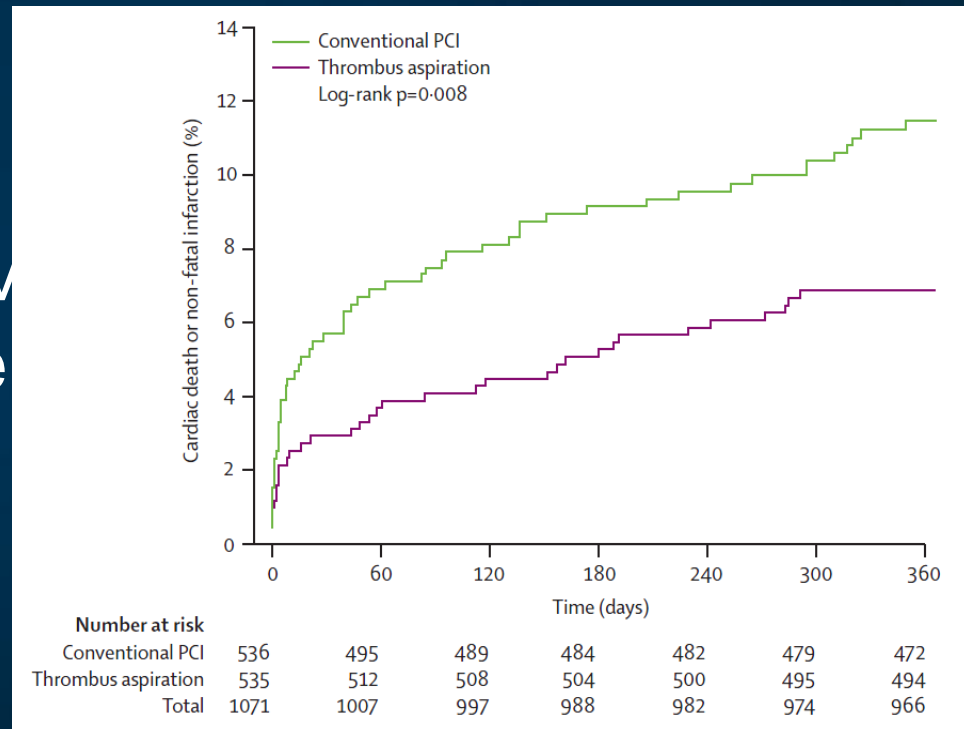
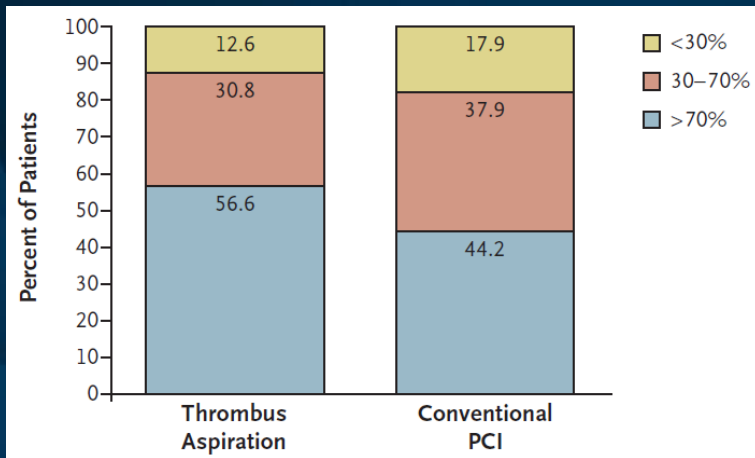
Manual thrombus aspiration

TAPAS : 1-YEAR CLINICAL OUTCOME

Myocardial Blush Grade



ST segment resolution



Svilaas T et al, N Engl J Med 2008;358:557

Vlaar PJ et al, Lancet 2008;371:1915

Manual thrombus aspiration in STEMI

1. Single center experience
2. Mixed results in infarct size reduction
3. Effect of abciximab?

2009 ACC/AH guideline on thrombus aspiration during PCI for STEMI

Class IIa

Aspiration thrombectomy is reasonable for patients undergoing primary PCI (evidence B)

Routine vs selective thrombus aspiration?

2009 ACC/AH guideline on glycoprotein IIb/IIIa receptor antagonists for STEMI

Class IIa

It is reasonable to start treatment with abciximab as early as possible before primary PCI in patients with STEMI.

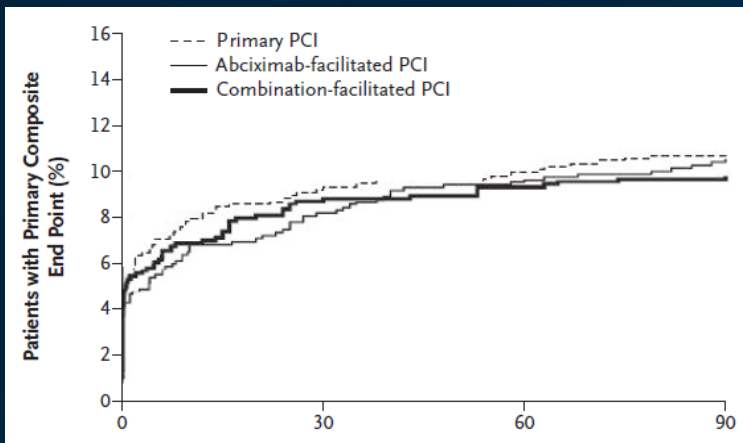


Class IIa

It is reasonable to start treatment with glycoprotein IIb/IIIa receptor antagonists at the time of primary PCI in selected patients with STEMI.

FINESSE (n=2452 STEMI)

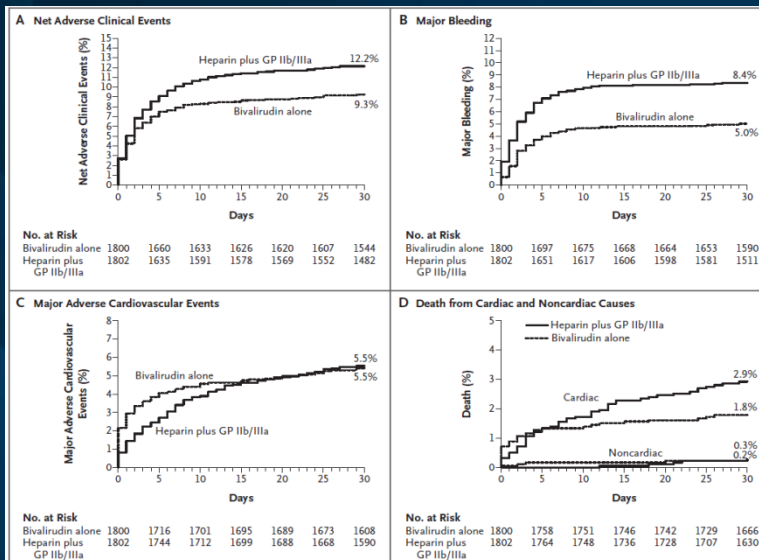
Pre-PCI abciximab



N Engl J Med 2008;358:2205

HORIZONS-AMI (n=3602 STEMI)

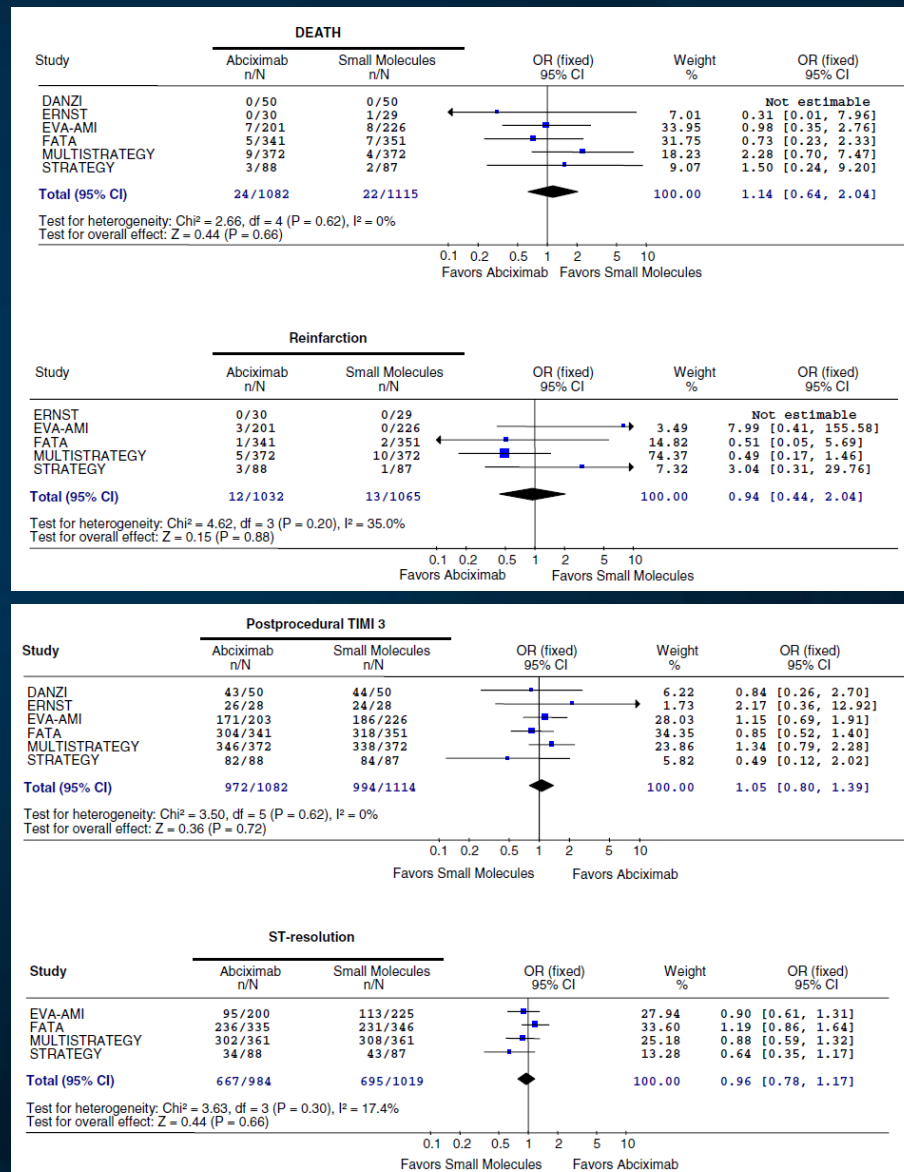
Bivalirudin vs Heparin+abciximab



N Engl J Med 2008;358:2218

Meta-analysis:

abciximab vs tirofiban or eptifibatide



Luca GD et al, J Am Coll Cardiol 2009;53:1668

Glycoprotein IIb/IIIa receptor antagonists in PCI for STEMI

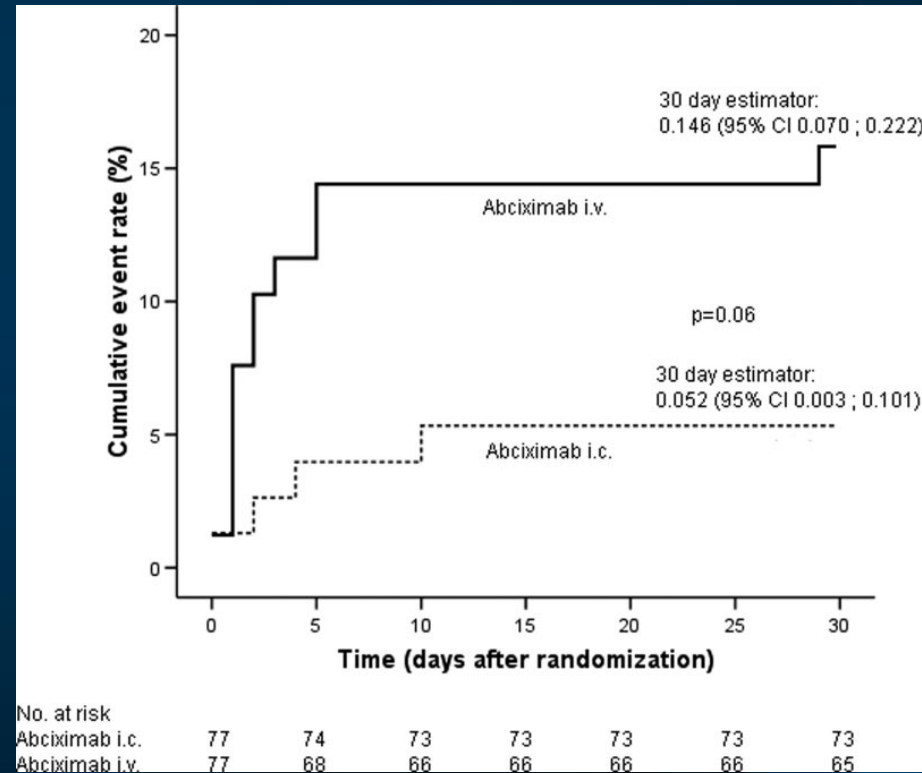
- In the setting of DAT with UFH or bivalirudin, the role of GP IIb/IIIa receptor antagonists should be reevaluated.

Esp. in the era of strong oral antiplatelet agents (prasugrel or ticagrelor)

- No routine therapy but in selected cases
 - Large thrombus burden
- Intracoronary bolus abciximab?

Intracoronary vs. intravenous abciximab?

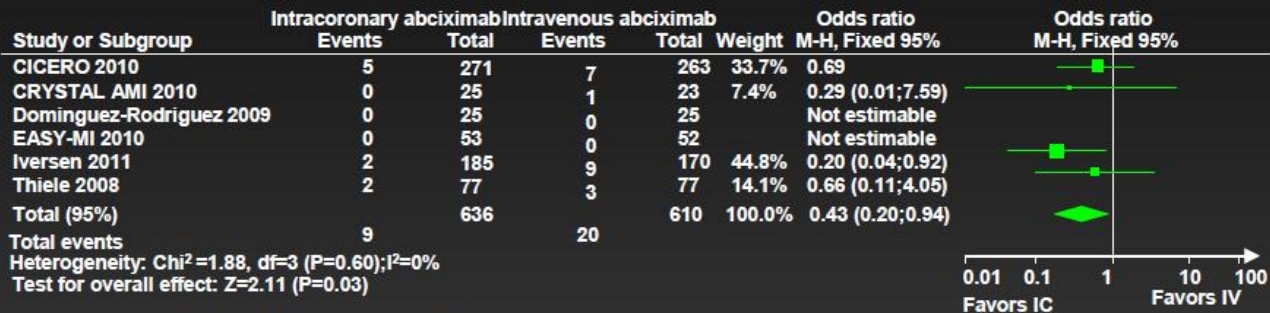
MRI	IC abciximab (n=67)	IV abciximab (n=71)	P
Infarct size	15.1%	23.4%	0.01
MVO, early	1.1%	3.4%	0.01
MVO, late	0.1%	1.1%	0.02



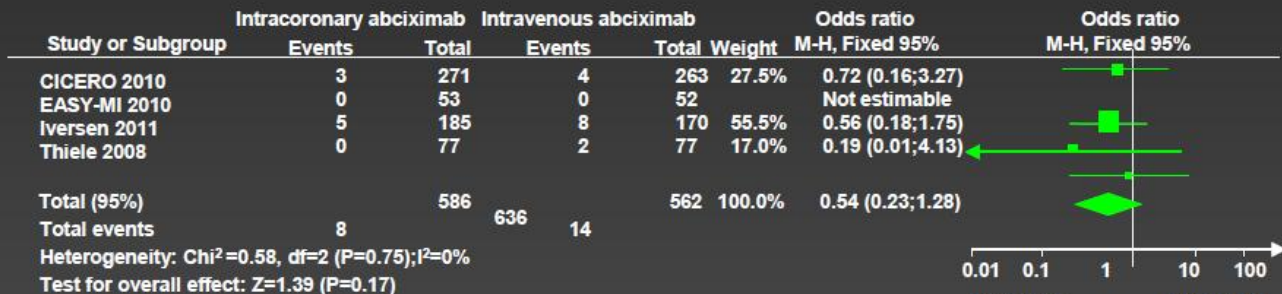
Thiele H et al, Circulation 2008;118:49

Intracoronary vs. intravenous abciximab?

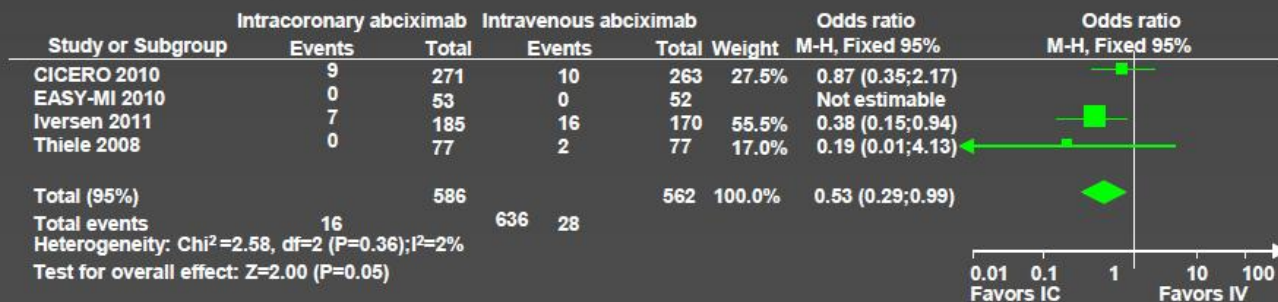
30-day Mortality



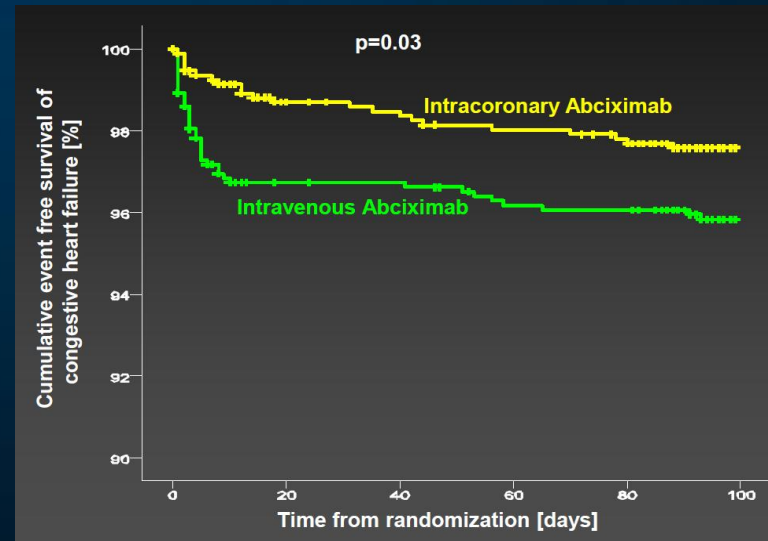
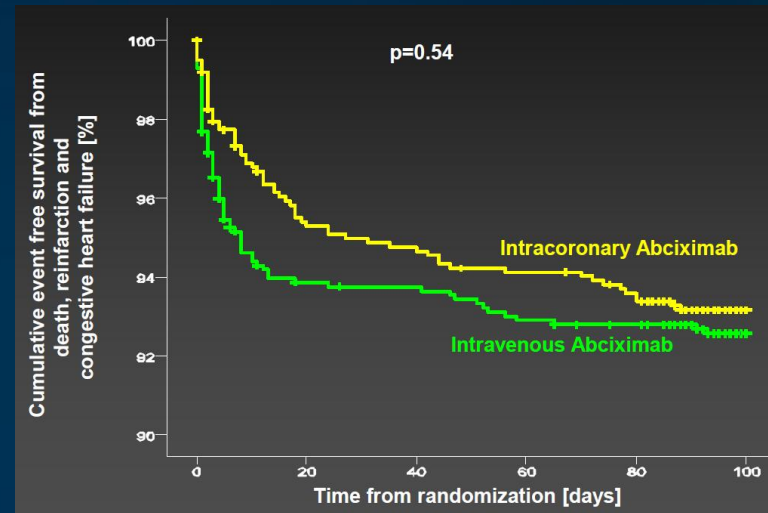
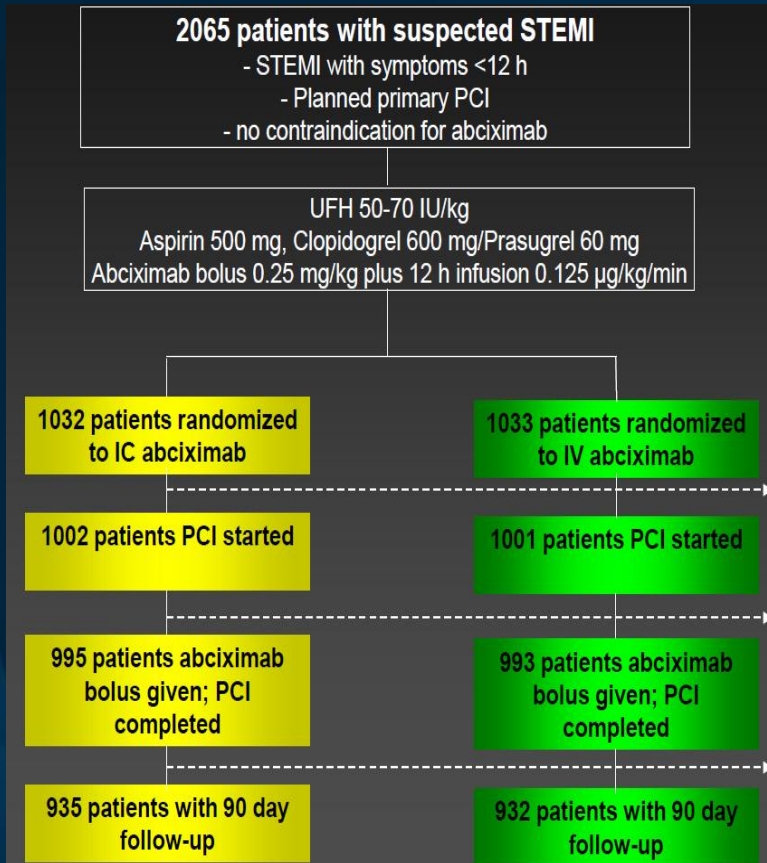
30-day Myocardial Infarction



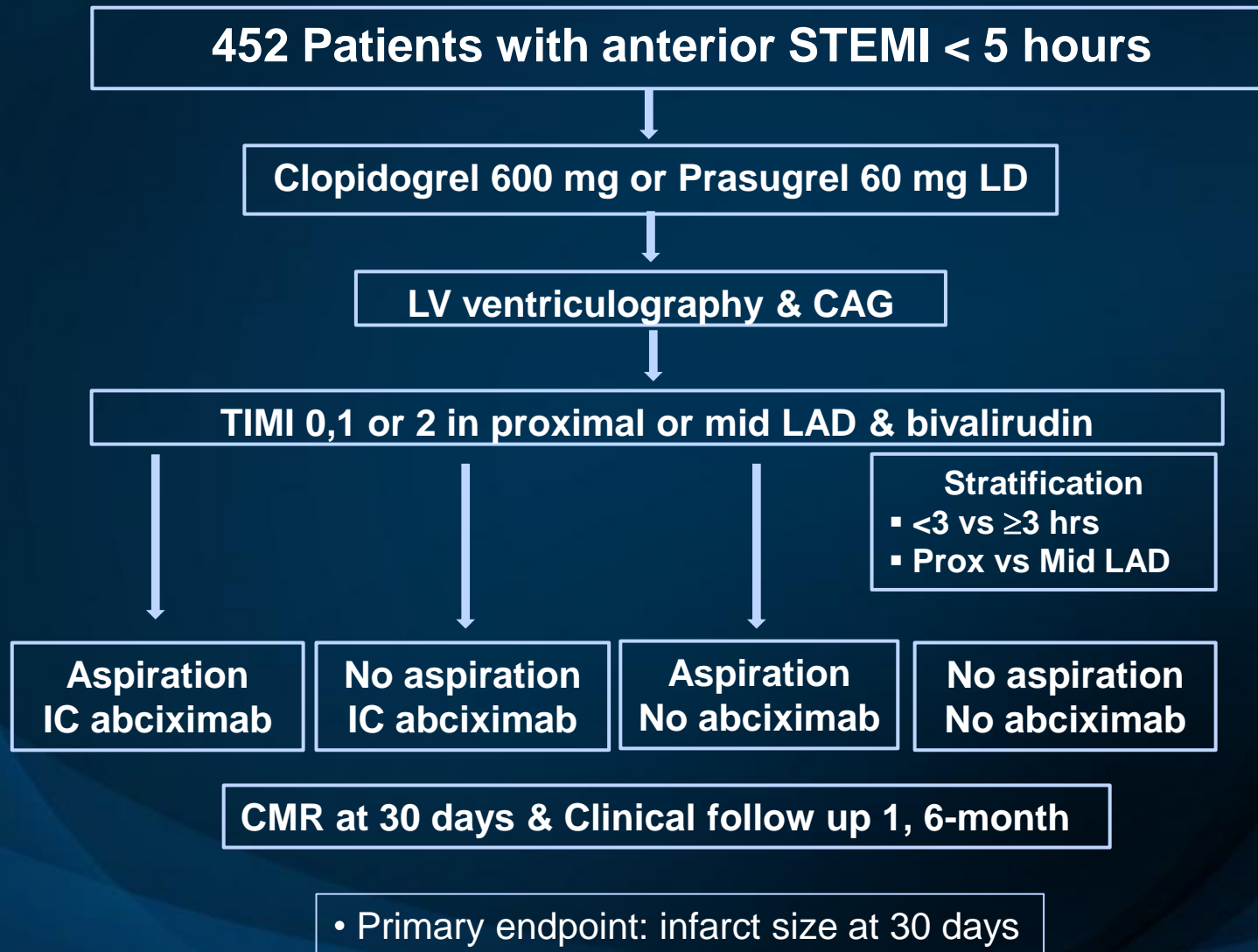
30-day Target Vessel Revascularization



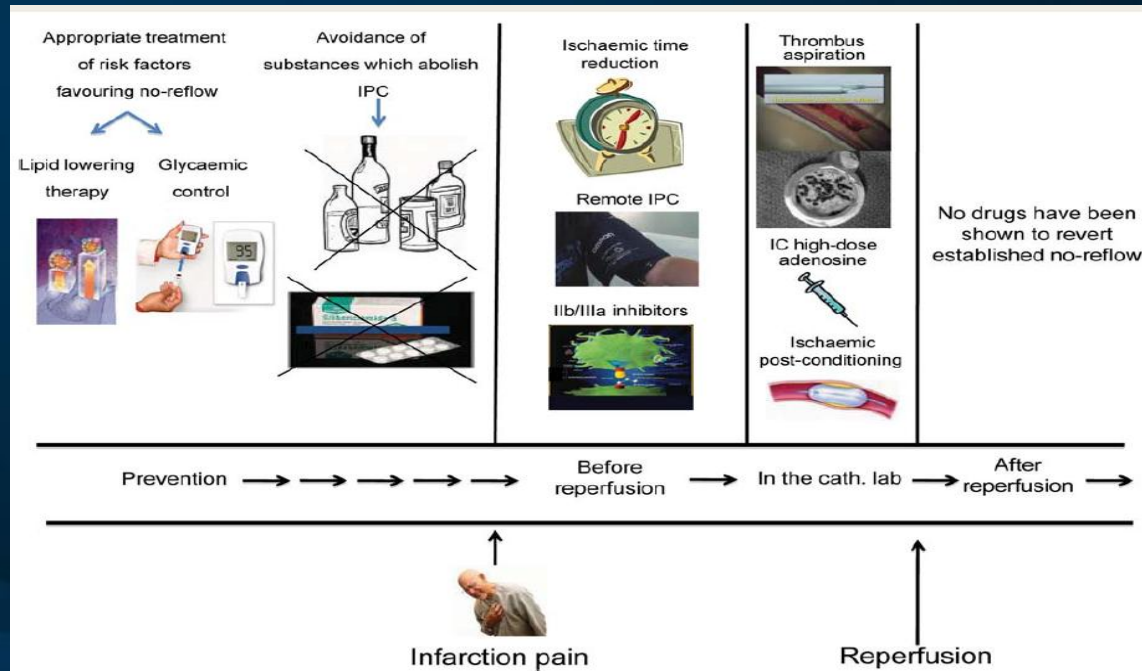
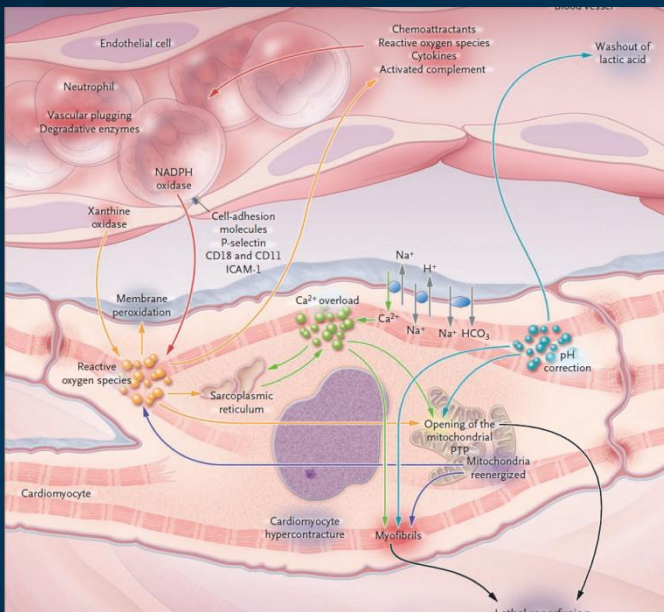
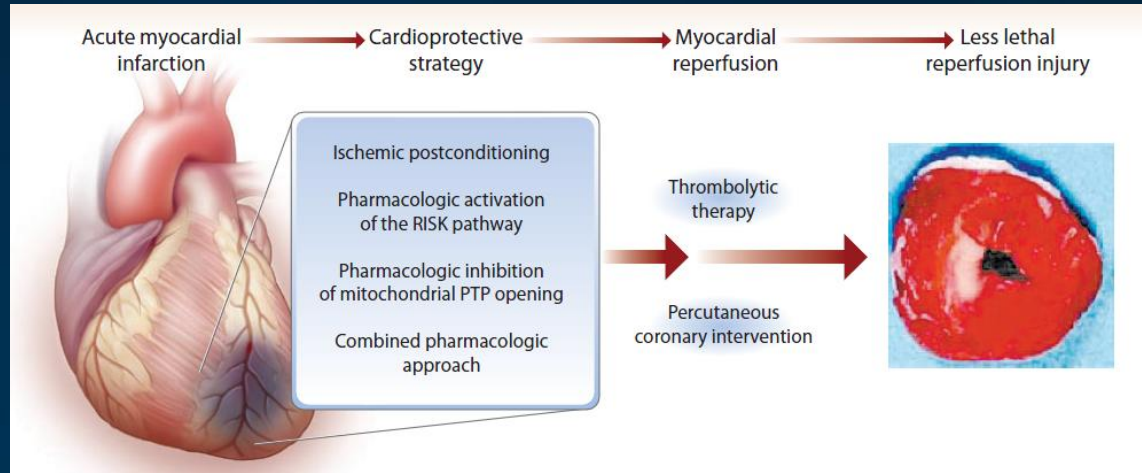
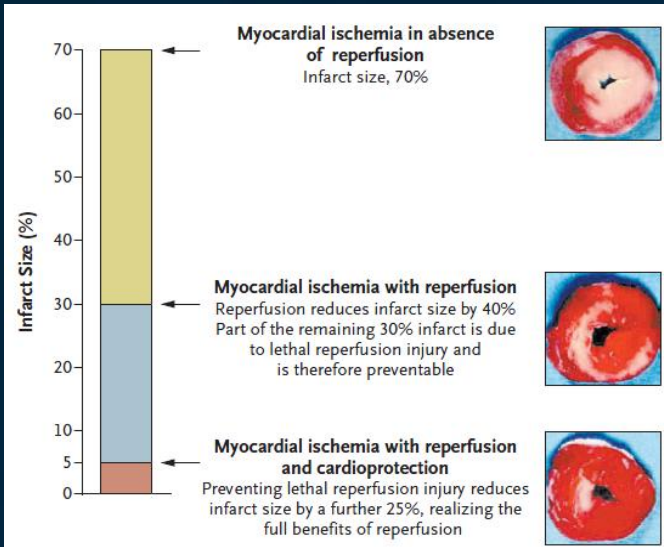
AIDA STEMI trial



INFUSE-AMI trial (Gregg W Stone)



Prevention of reperfusion injury



Take home messages

1. Prevention is the best treatment for no-reflow in PCI for STEMI.
2. Reduction in ischemic time is mandatory.
3. Thrombus aspiration should be the first procedure if possible. INFUSE-AMI may tell who is winner: routine vs. selective thrombus aspiration.
4. IC or IV abciximab should be given in selected patients.
5. Remote IPC, ischemic PC, or IC high-dose adenosine, abciximab should be reevaluated in the era of prasugrel or ticagrelor.
6. Novel therapy (drugs) against reperfusion injury becomes the remaining blue ocean in STEMI therapy.

**THANK YOU for
your attention!**