Myocardial infarction: Treatment in a hospital with or without catheterisation laboratory. Treatment of non-ST-elevation myocardial infarction.

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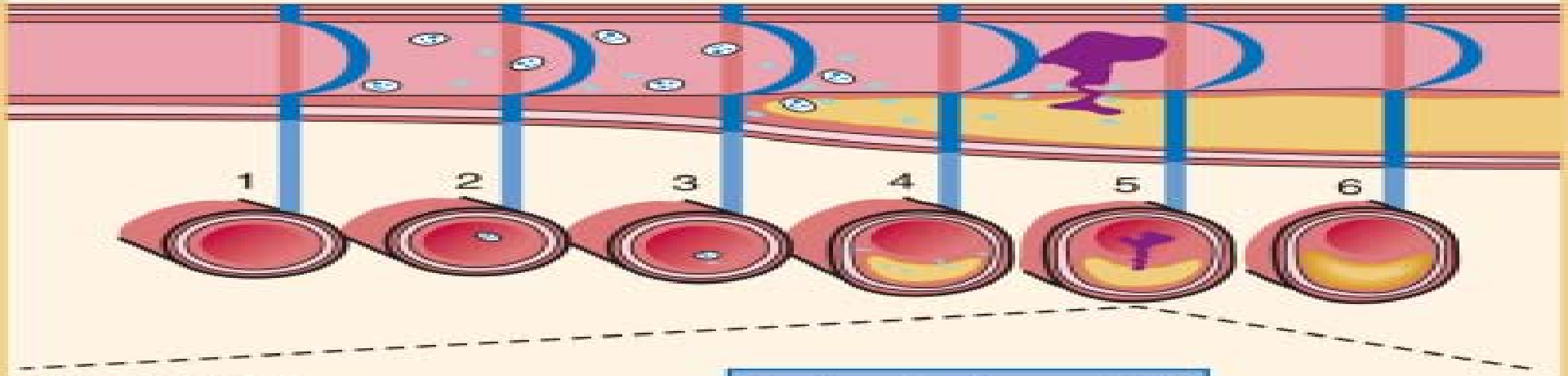
Common Causes of Acute Chest Pain

System	*Syndrome*
Cardiac	Angina
	Rest or unstable angina
	Acute myocardial infarction
	Pericarditis
Vascular	Aortic dissection
	Pulmonary embolism
	Pulmonary hypertension
Pulmonary	Pleuritis and/or pneumonia
	Tracheobronchitis
	Spontaneous pneumothorax
Gastrointestinal	Esophageal reflux
	Peptic ulcer
	Gallbladder disease
	Pancreatitis
Musculoskeletal	Costochondritis
	Cervical disc disease
	Trauma or strain
Infectious	Herpes zoster
Psychological	Panic disorder

ACUTE CORONARY SYNDROMES
SPECTRUM OF CLINICAL PRESENTATION



Presentation

Working Dx

ECG

Cardiac biomarker

Final Dx

Ischemic discomfort

Acute coronary syndrome

No ST elevation

Unstable angina

NSTEMI

ST elevation

Myocardial infarction

NQMI

QwMI

UA/NSTEMI

- UA/NSTEMI comprises a heterogeneous group of patients.

- In this group, evidence of myocardial necrosis on the basis of elevated cardiac serum markers, such as creatine kinase isoenzyme (CK-MB) and/or troponin T/I, leads to the diagnosis of NSTEMI.

Epidemiology

- NSTE-ACS is more frequent than STEMI.
- NSTE-ACS events continue over days or weeks, STEMI events occur before or shortly after presentation.
- Mortality of STEMI and NSTE-ACS after 6 months comparable (13%vs12%).
- Death rates increase two fold at 4 years NSTEMI>STEMI.

Pathophysiology

- The pathophysiology of UA/NSTEMI involves a broad timeline with three phases rather than an isolated ischemic event.

- In UA/NSTEMI the pathophysiology may actually begin several decades before the acute clinical event, and then may span more than 20 years afterward.

- The acute event, which usually involves thrombus formation at the site of a ruptured or eroded atherosclerotic plaque is currently referred to as “atherothrombosis” a term that is replacing “atherosclerosis”.

Causes

Common Cause

- Thrombus or thromboembolism, usually arising on disrupted or eroded plaque with dynamic obstruction (spasm) of epicardial and/or microvascular vessels, and coronary arterial inflammation.

Non-atherosclerotic aetiology

- Non–plaque-associated coronary thromboembolism, coronary artery dissection, arteritis, trauma, cocaine abuse, congenital anomalies and complications of cardiac catheterisation.

Diagnostic Tools

- History
- Physical examination
- ECG
- Biochemical markers of myocardial necrosis
- Laboratory tests
- Non invasive Testing
- Invasive Testing

Risk Stratification

- Plays a central role in the evaluation and management.
- Specific subgroups of patients are identified as being at higher risk of adverse outcome.
- Higher risk subgroups appear to derive greater benefit from aggressive antithrombotic and/or interventional therapies.
- Contributes to patient triage.

Approach to Risk Stratification

- Diagnosis and risk stratification should be based on a combination of:

  History

  Symptoms

  ECG

  Biomarkers

  Risk score results

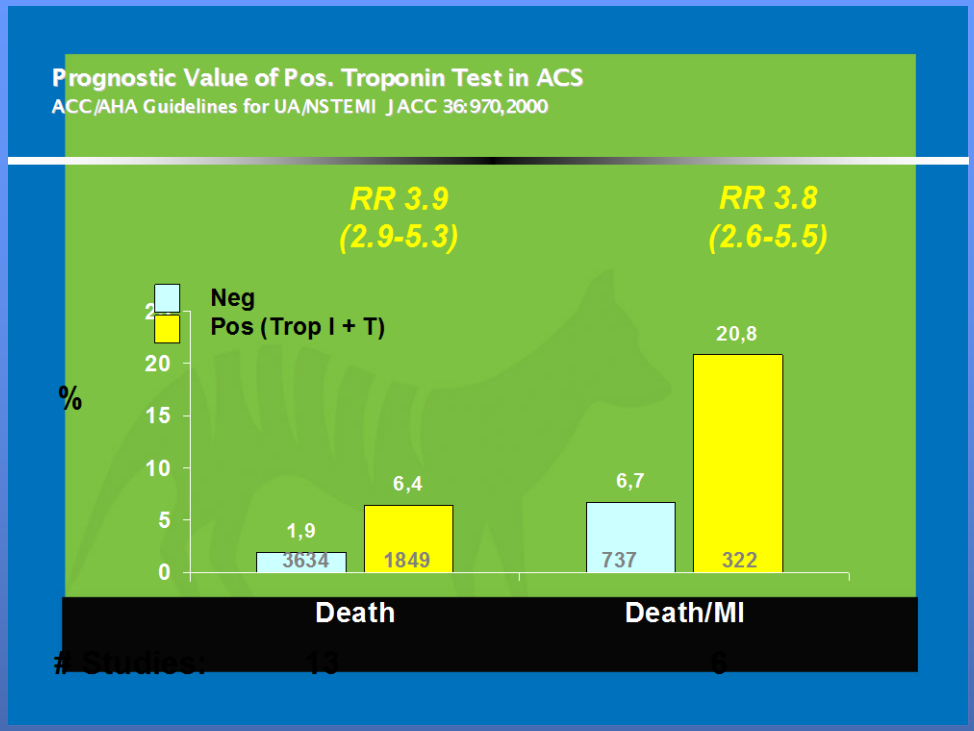
- *Individual risk stratification is a dynamic process updated as the clinical situation evolves.*

Risk assessment by cardiac markers of necrosis

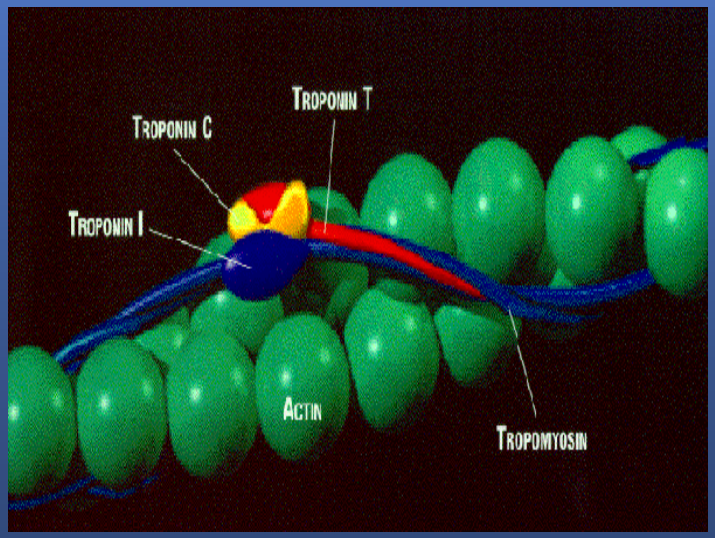
- NSTEMI pts have a worse long-term prognosis than UA pts.
- There is a linear relation between the level of trop T or I and subsequent risk of death.

  N Engl J Med 335: 1342-1349, 1996
- Several other studies observed a higher risk of MI (or recurrent MI) with lower degrees of troponin elevation.
- Overall rate of death or MI is equally high with low or higher troponin values.

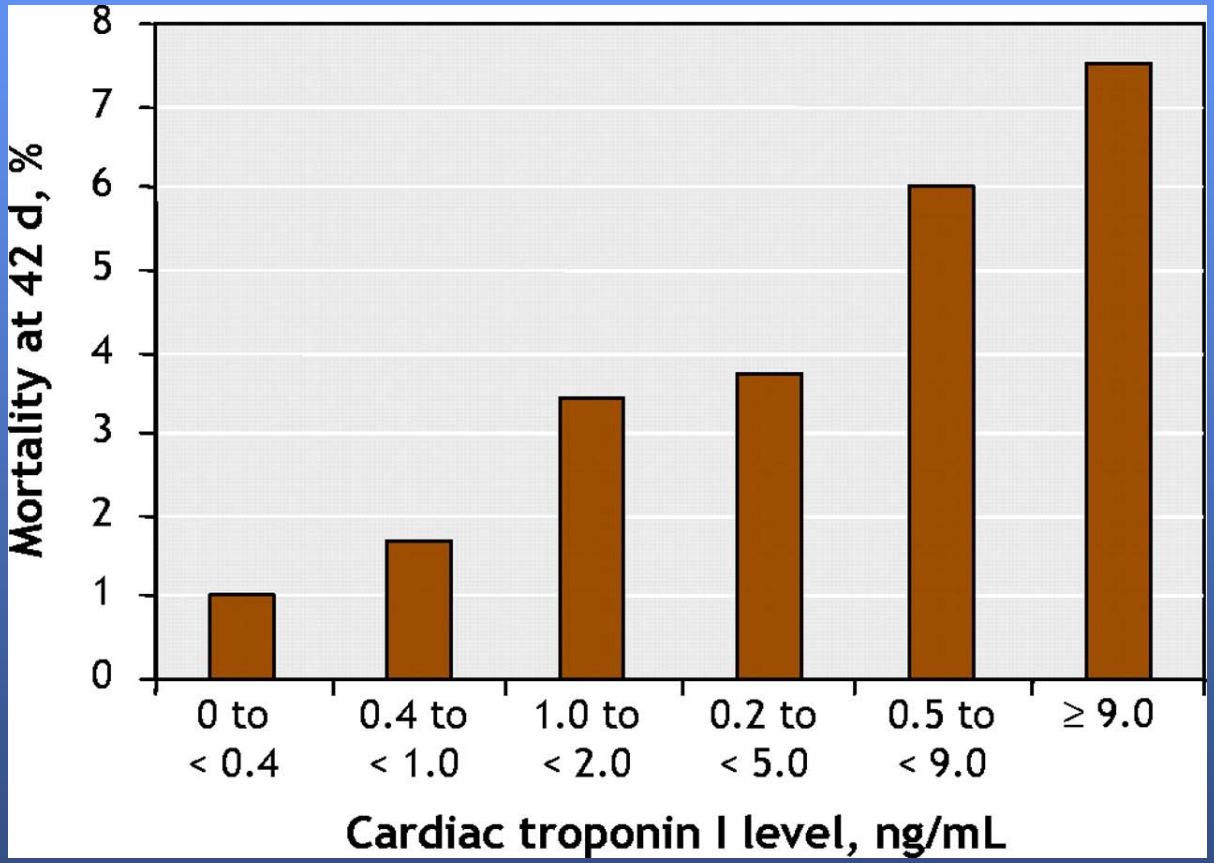
  Morrow DA, et al; JAMA 286: 2405-2412, 2001



Troponin is the preferred biomarker for diagnosis of MI. cTnT or cTnI > 99th %ile on any determination

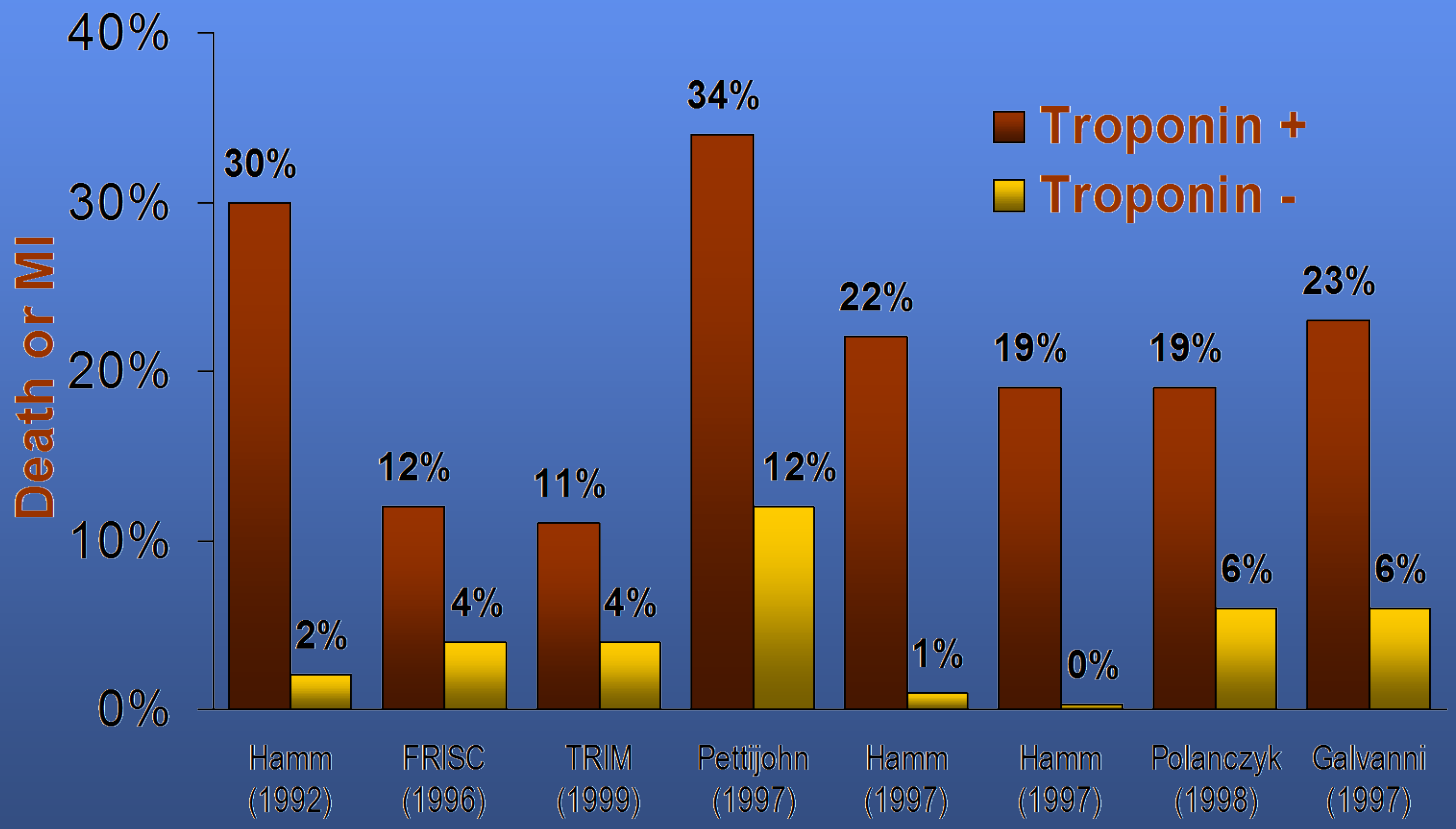


Mortality rates according to level of cardiac troponin I at baseline



N Engl J Med 1996;335:1342-9.

Troponin as a Marker of Increased Risk in ACS



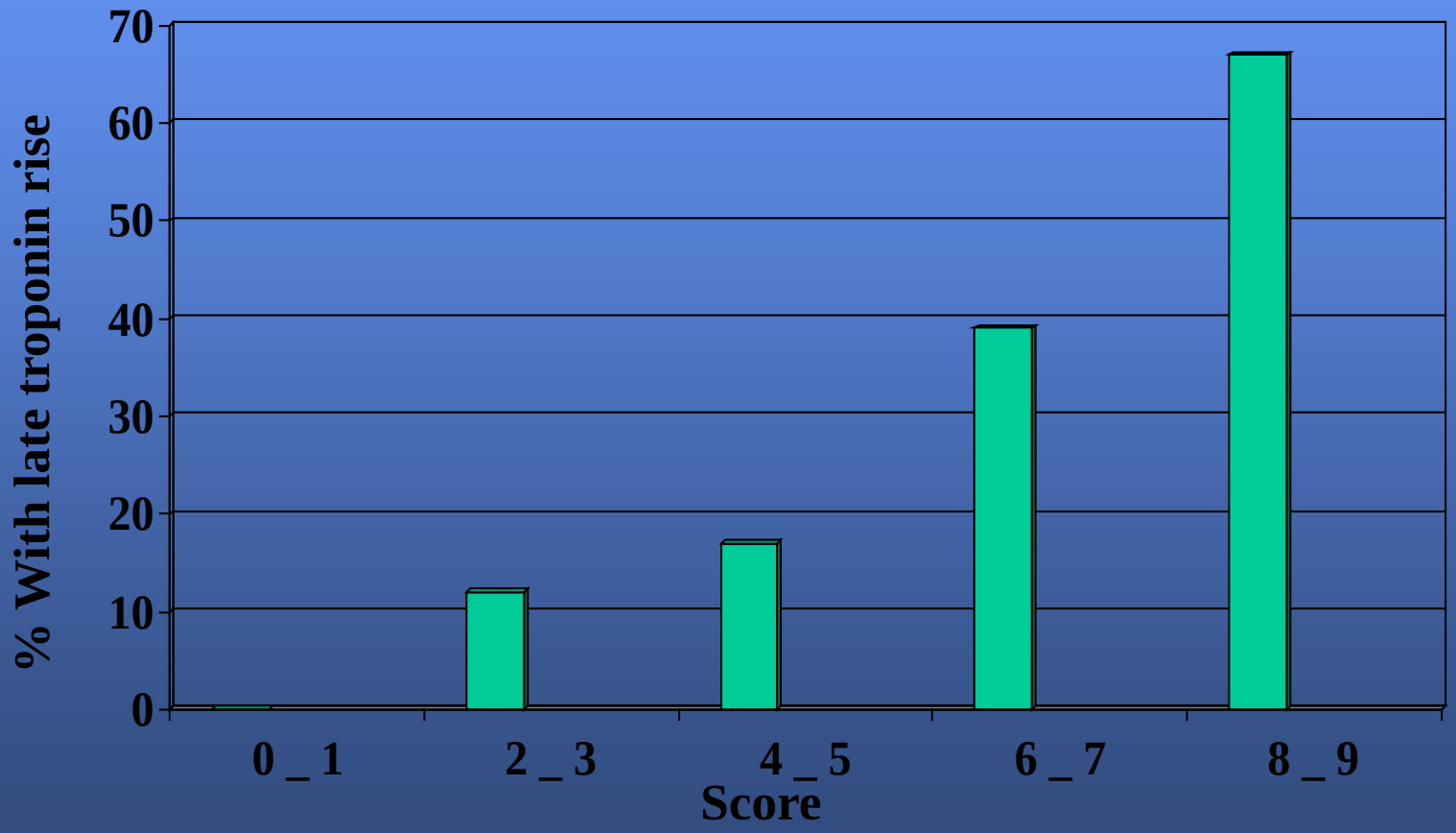
Non-coronary conditions with troponin elevations

- Severe congestive heart failure: acute and chronic
- Aortic dissection, aortic valve disease, or hypertrophic cardiomyopathy
- Cardiac contusion, ablation, pacing, cardioversion, or endomyocardial biopsy
- Inflammatory diseases, e.g. myocarditis, or myocardial extension of endocarditis/pericarditis
- Hypertensive crisis
- Tachy- or bradyarrhythmias
- Pulmonary embolism, severe pulmonary hypertension
- Hypothyroidism
- Apical ballooning syndrome
- Chronic or acute renal dysfunction
- Acute neurological disease, including stroke, or subarachnoid haemorrhage
- Infiltrative diseases, e.g. amyloidosis, haemochromatosis, sarcoidosis, scleroderma
- Drug toxicity, e.g. adriamycin, 5-fluorouracil, herceptin, snake venoms
- Burns, if affecting >30% of body surface area
- Rhabdomyolysis
- Critically ill patients, especially with respiratory failure, or sepsis

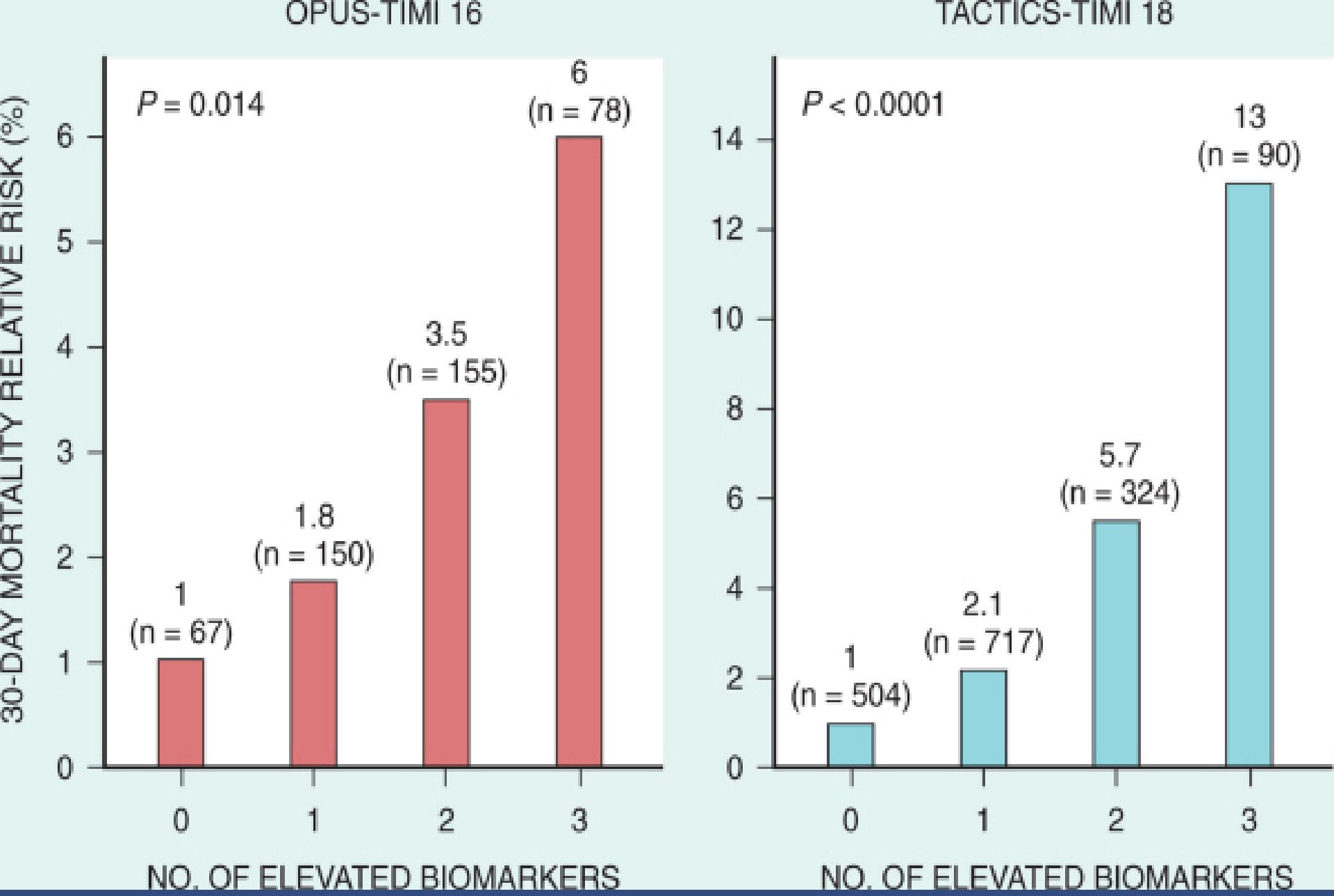
Predictors of late (12h) troponin level rise in initially troponin-negative patients TIMI-IIIB

Predictor	Score
ST- segment deviation	2
Presentation <8 hr from symptom onset	2
No prior PCI	2
No prior beta-blokade	1
Unheralded angina	1
History of MI	1

(N=200)	Score
ST- segment deviation	2
Presentation <8 hr from symptom onset	2
No prior PCI	2
No prior beta-blokade	1
Unheralded angina	1
History of MI	1



The TIMI-III B score to identify pts who become troponin positive later during hospital admission



A multimarker strategy to predict mortality, Circulation. 105: 1760-63, 2002

Predictors of long-term death or MI to be considered in risk stratification

- Clinical indicators: age, heart rate, blood pressure, Killip class, diabetes, previous MI/CAD.
- ECG markers: ST-segment depression.
- Laboratory markers: troponins, GFR/CrCl/cystatin C, BNP/NT-proBNP, hsCRP.
- Imaging findings: low EF, main stem lesion, 3VD.
- Risk score results.

Risk scores

- **TIMI risk score**
- **GRACE risk model**
- FRISC II risk score
- PERSUIT risk score

TIMI Risk Score Variables

- Age ≥ 65 years
- At least 3 risk factors for CAD

Diabetes

Cigarette smoking

HTN (BP 140/90 mm Hg or on antihypertensive medication)

Low HDL cholesterol (< 40 mg/dL)

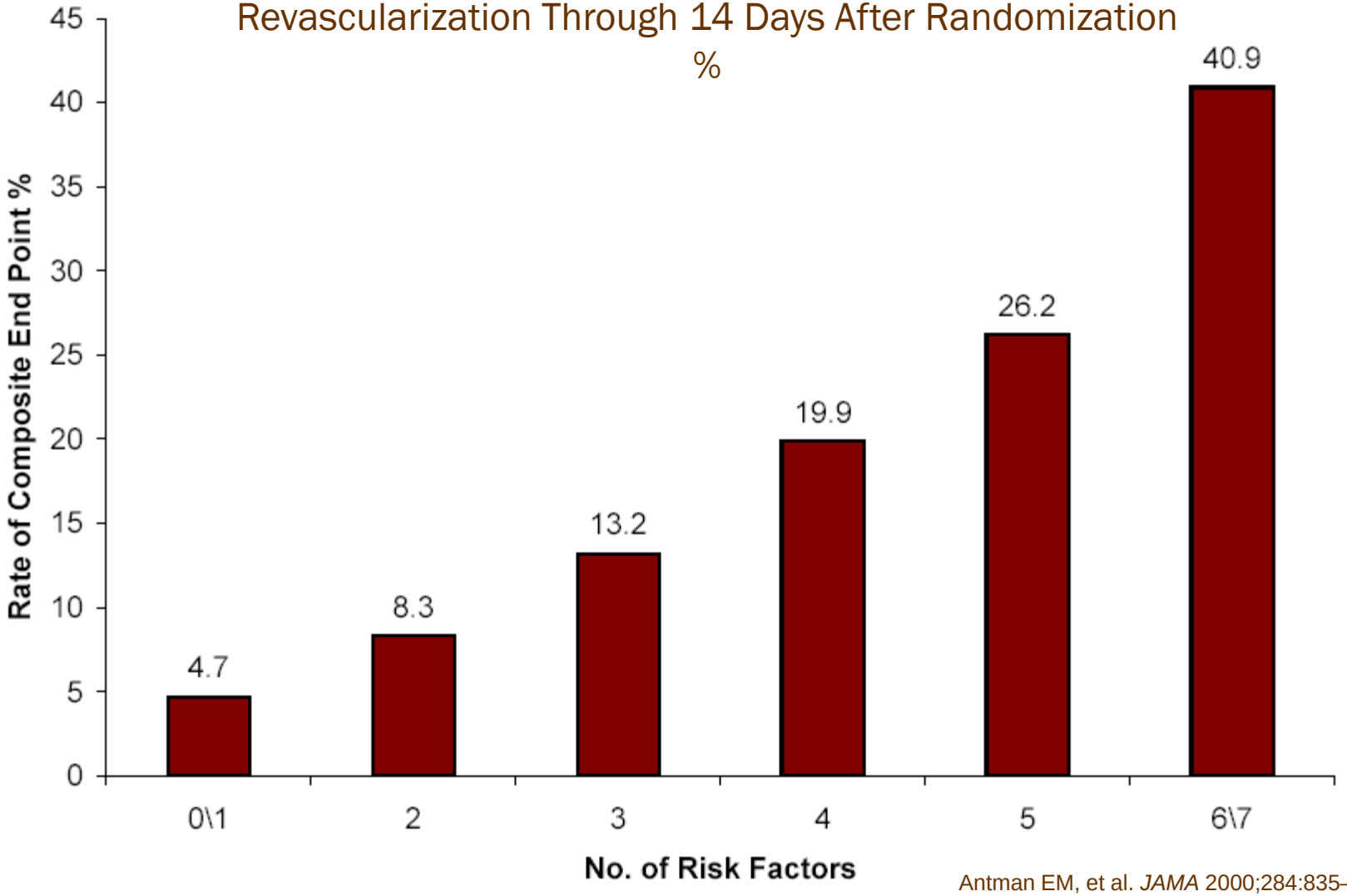
Family history of premature CAD (CAD in male first-degree relative 55 or younger, CAD in female first-degree relative 65 or younger)

Age (men 45 years; women 55 years)

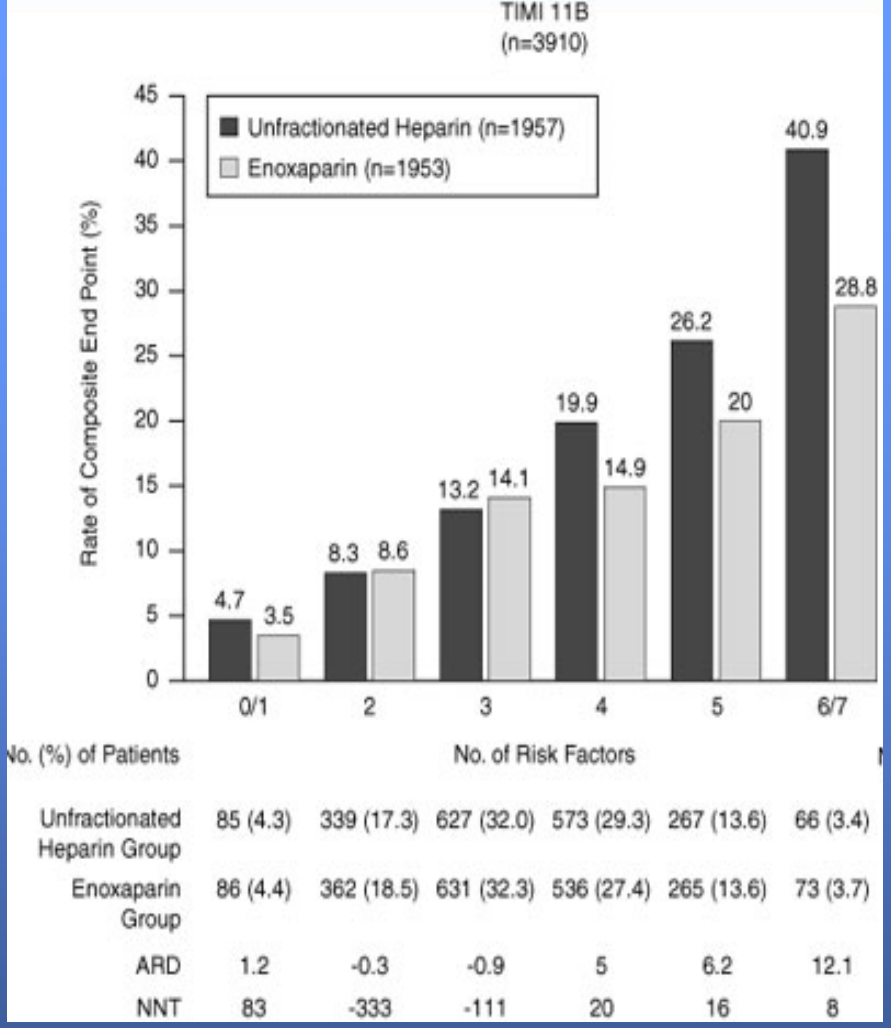
- Prior coronary stenosis of ≥ 50%
- ST-segment deviation on ECG presentation
- At least 2 anginal events in prior 24 hours
- Use of aspirin in prior 7 days
- Elevated serum cardiac biomarkers

Antman EM, et al. *JAMA* 2000;284:835–42.

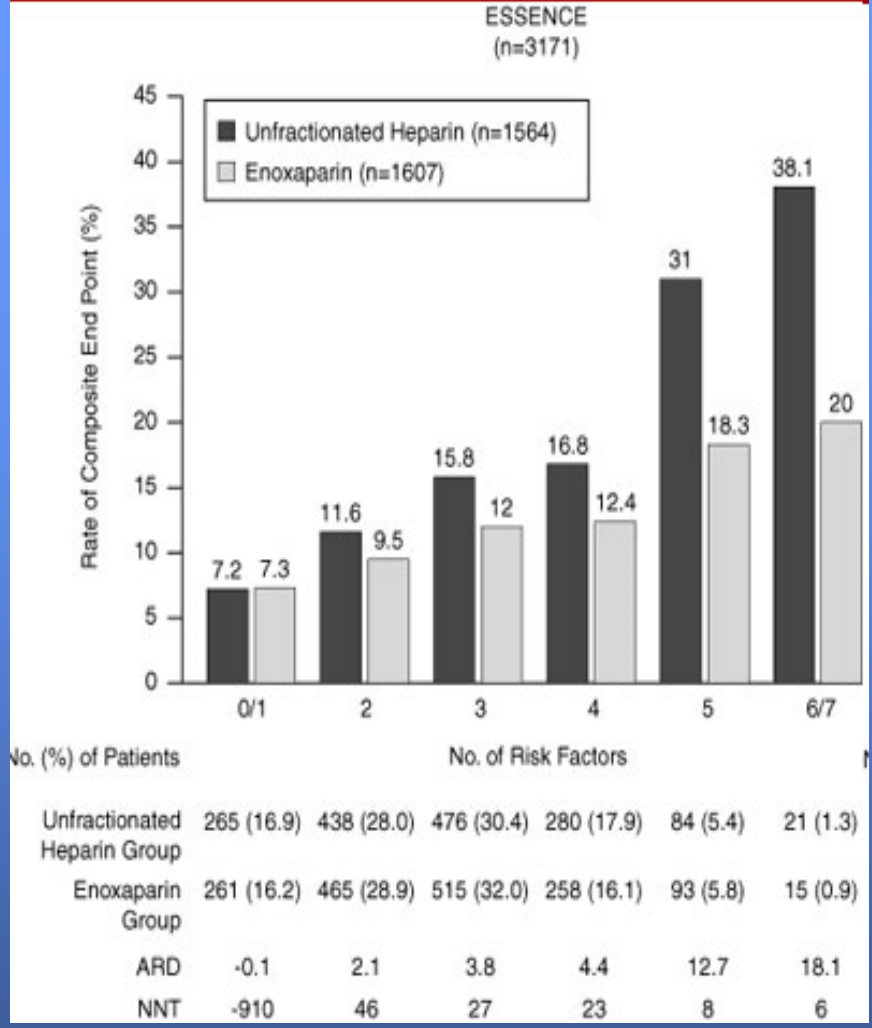
TIMI Risk Score, All-Cause Mortality, New or Recurrent MI, or Severe Recurrent Ischemia Requiring Urgent Revascularization Through 14 Days After Randomization



Antman EM, et al. *JAMA* 2000;284:835–42.

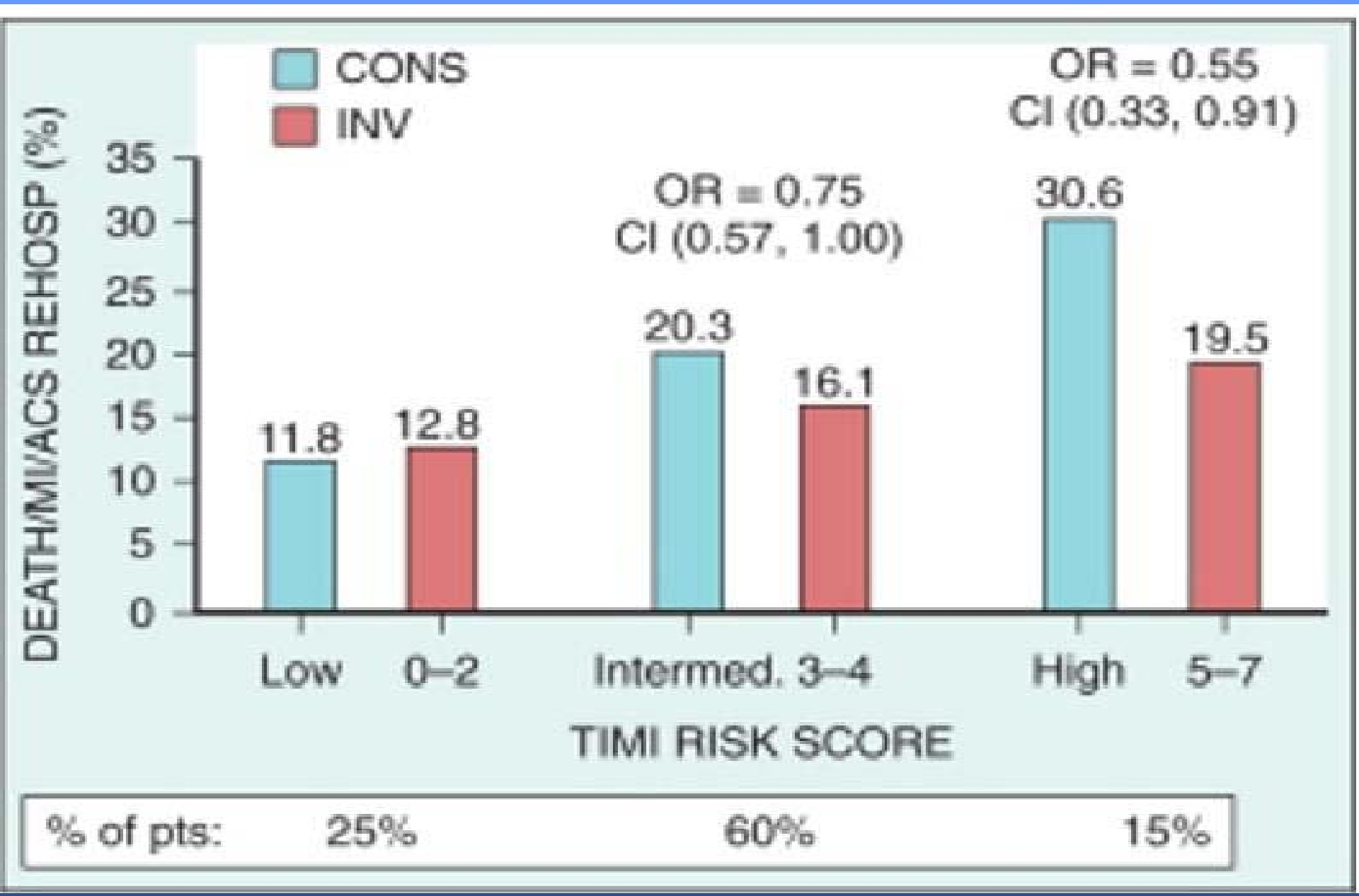


No. (%) of Patients						
Unfractionated Heparin Group	85 (4.3)	339 (17.3)	627 (32.0)	573 (29.3)	267 (13.6)	66 (3.4)
Enoxaparin Group	86 (4.4)	362 (18.5)	631 (32.3)	536 (27.4)	265 (13.6)	73 (3.7)
ARD	1.2	-0.3	-0.9	5	6.2	12.1
NNT	83	-333	-111	20	16	8



No. (%) of Patients						
Unfractionated Heparin Group	265 (16.9)	438 (28.0)	476 (30.4)	280 (17.9)	84 (5.4)	21 (1.3)
Enoxaparin Group	261 (16.2)	465 (28.9)	515 (32.0)	258 (16.1)	93 (5.8)	15 (0.9)
ARD	-0.1	2.1	3.8	4.4	12.7	18.1
NNT	-910	46	27	23	8	6

Validation of TIMI risk score and assessment of treatment effect according to score in the TIMI 11B and ESSENCE



Use of the TIMI Risk Score for UA/STEMI to predict the benefit of an early invasive strategy, N Engl J Med 344:1879, 2001

Killip Class	Points	SBP, mm Hg	Points	Heart Rate, Beats/min	Points	Age, y	Points	Creatinine Level, mg/dL	Points
I	0	≤80	58	≤50	0	≤30	0	0-0.39	1
II	20	80-99	53	50-69	3	30-39	8	0.40-0.79	4
III	39	100-119	43	70-89	9	40-49	25	0.80-1.19	7
IV	59	120-139	34	90-109	15	50-59	41	1.20-1.59	10
		140-159	24	110-149	24	60-69	58	1.60-1.99	13
		160-199	10	150-199	38	70-79	75	2.00-3.99	21
		≥200	0	≥200	46	80-89	91	>4.0	28
						≥90	100		

Other Risk Factors	Points
Cardiac Arrest at Admission	39
ST-Segment Deviation	28
Elevated Cardiac Enzyme Levels	14

2. Sum Points for All Predictive Factors:

Killip Class	+	SBP	+	Heart Rate	+	Age	+	Creatinine Level	+	Cardiac Arrest at Admission	+	ST-Segment Deviation	+	Elevated Cardiac Enzyme Levels	=	Total Points								

3. Look Up Risk Corresponding to Total Points:

Total Points	≤60	70	80	90	100	110	120	130	140	150	160	170	180	190	200	210	220	230	240	≥250
Probability of In-Hospital Death, %	≤0.2	0.3	0.4	0.6	0.8	1.1	1.6	2.1	2.9	3.9	5.4	7.3	9.8	13	18	23	29	36	44	≥52

For example, a patient has Killip class II, SBP of 100 mm Hg, heart rate of 100 beats/min, is 65 years of age, has serum creatinine level of 1 mg/dL, did not have a cardiac arrest at admission but did have ST-segment deviation and elevated enzyme levels.

His score would be: 20 + 53 + 15 + 58 + 7 + 0 + 28 + 14 = 196

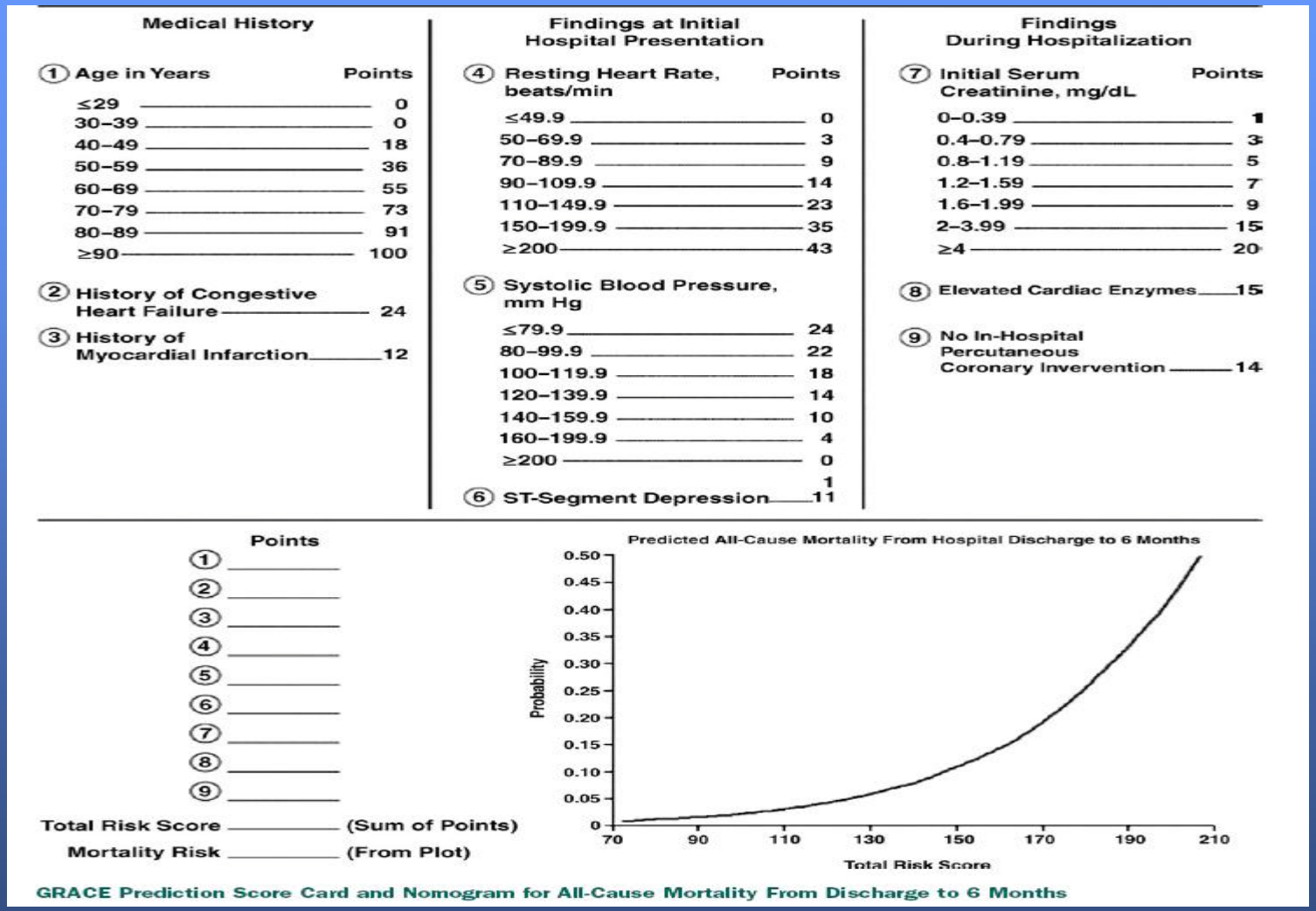
This person would have about a 16% risk of having an in-hospital death.

Similarly, a patient with Killip class I, SBP of 80 mm Hg, heart rate of 60 beats/min, is 55 years of age, has serum creatinine level of 0.4, and no risk factors would have the following score:

0 + 58 + 3 + 41 + 1 = 103, which gives approximately a 0.9% risk of having an in-hospital death.

Global registry Of Acute Coronary Events Risk Model nomogram.

GRACE Prediction Score Card and Nomogram for All-Cause Mortality From Discharge to 6 Months



Anderson, J. L. et al. J Am Coll Cardiol 2007;50:e1-e157

Mortality in hospital and at 6 months in low-, intermediate-, and high-risk categories in registry populations according to the GRACE risk score

Risk category (tertiles)	GRACE risk score	In-hospital deaths (%)
Low	≤108	<1
Intermediate	109–140	1–3
High	>140	>3

Risk category (tertiles)	GRACE risk score	Post-discharge to 6 months deaths (%)
Low	≤88	<3
Intermediate	89–118	3–8
High	>118	>8

Risk assessment by cardiac markers

- CK-MB and troponins
- C-Reactive Protein
- White Blood Cell Count
- Myeloperoxidase
- B-type Natriuretic Peptide
- Creatinine
- Glucose

ACC/AHA High Risk Indicators for Non-ST-Segment Elevation Acute Coronary Syndrome

- Recurrent angina/ischemia at rest or with low-level activities despite intensive anti-ischemic therapy
- Elevated TnT or TnI
- New or presumably new ST-segment depression
- Recurrent angina/ischemia with CHF symptoms, an S_3 gallop, pulmonary edema, worsening rales, or new or worsening MR
- High-risk findings on noninvasive stress testing
- Depressed LV systolic function (e.g., EF <0.40 on noninvasive study)
- Hemodynamic instability
- Sustained ventricular tachycardia
- PCI within 6 months
- Prior CABG

European Society of Cardiology Guidelines for Risk

High-Risk Indicators	Low-Risk Indicators
Elevated troponin levels	Normal troponin levels
Recurrent ischaemia	No recurrent ischaemia
ST-segment depression	No release of CK-MB
Early unstable angina after MI	Presence of negative or flat T waves
Diabetes mellitus	Normal ECG
Heamodynamic instability	
Major arrhythmias: VF/VT	

Treatment Strategies and Intervention

- Cardiac catheterisation and revascularisation.
- Conservative strategy with initial medical management with catheterisation and revascularisation only for recurrent ischemia.

Medical Therapy-General Measures

- Intensive care unit (high risk)- monitored bed (low or intermediate risk).
- Bed rest: ambulation after 12-24h stability and following revascularisation.
- Supplemental O2: cyanosis, extensive rales and/or when SO2 <92%.
- Relief of chest pain.
- Nitrates
- Beta-Blockers
- Calcium channel blockers

Medical Therapy-Antithrombotic Therapy

- ASA
- Clopidogrel
- GP IIb/IIIa inhibitors

Medical Therapy-Anticoagulants

- Heparin (UFH)
- Low-Molecular-Weight Heparin (LMWH)
- Fondaparinux
- Direct thrombin inhibitors
- Fibrinolytic therapy is not indicated for UA/STEMI. Prothrombotic effect can lead a patent culprit artery to total occlusion.

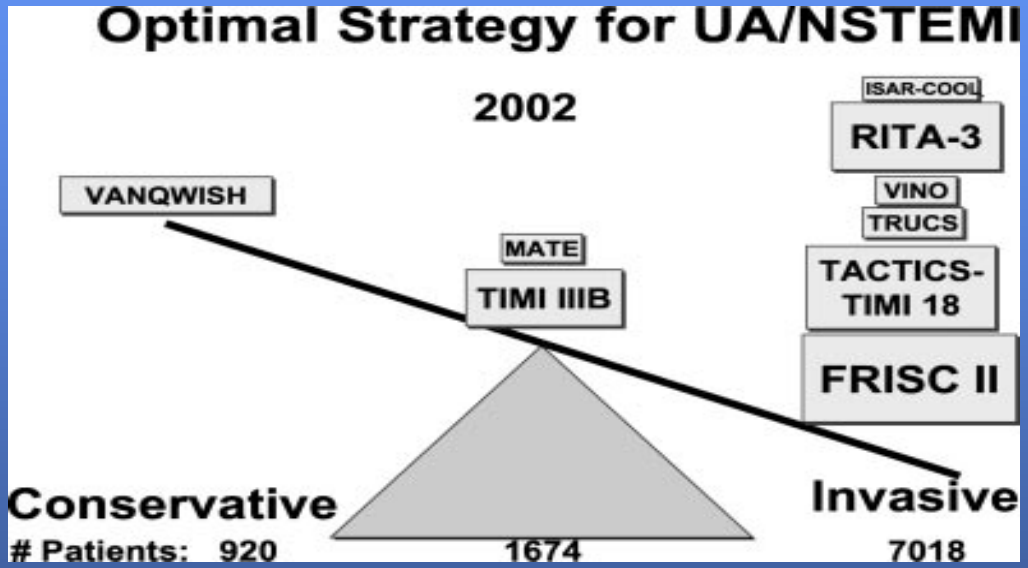
Risk Stratification and Benefit of IIb/IIIa inhibitors

- Greater benefit in high risk pts.
- Diabetics have a greater mortality reduction than non-diabetics.
- Troponin positive pts (high-risk) have the greatest benefit with or without revascularization.
- Benefit is confirmed even in the background of clopidogrel pretreatment.

Other Therapies

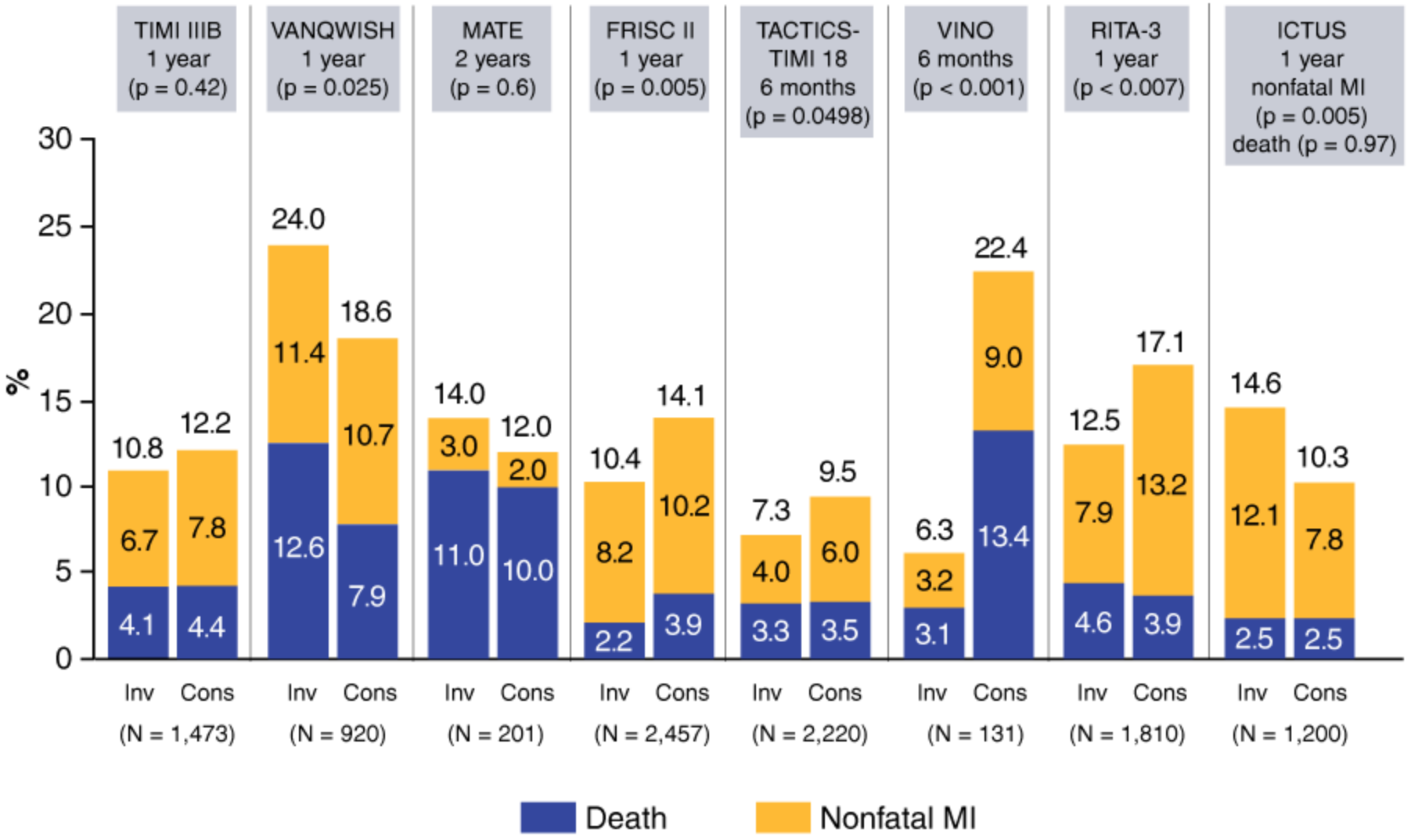
- ACE-Inhibitors
- Aldosterone-Receptor-Blockers
- Lipid-lowering therapy
- IABP

The “weight of the evidence” showing benefit of an invasive versus conservative strategy in patients with UA/NSTEMI

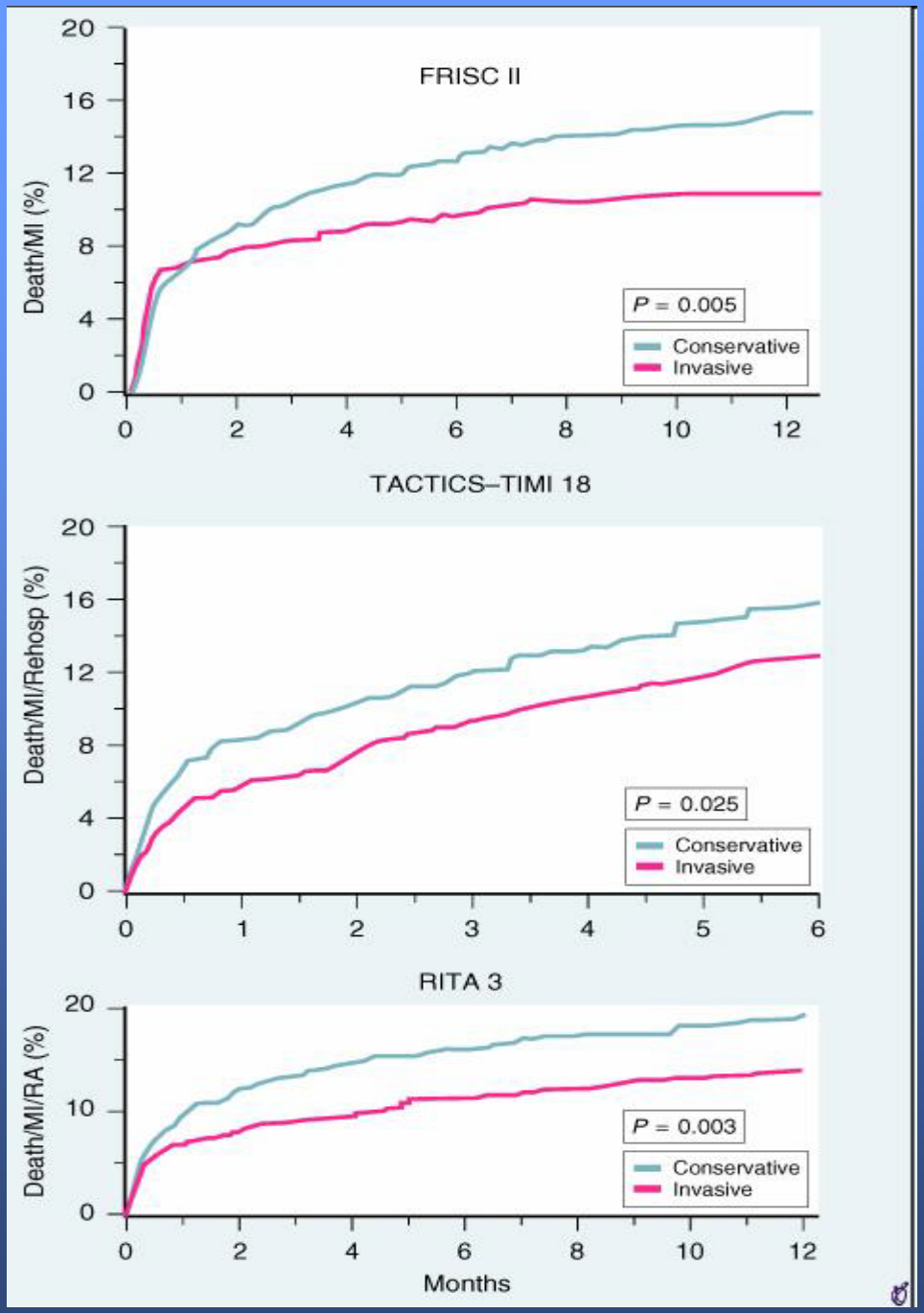


Cannon and Turpie Circulation 2003; 107 (21): 2640

Invasive vs. Conservative Strategies in UA/NSTEMI



UA = unstable angina; NSTEMI = Non-ST-elevation myocardial infarction

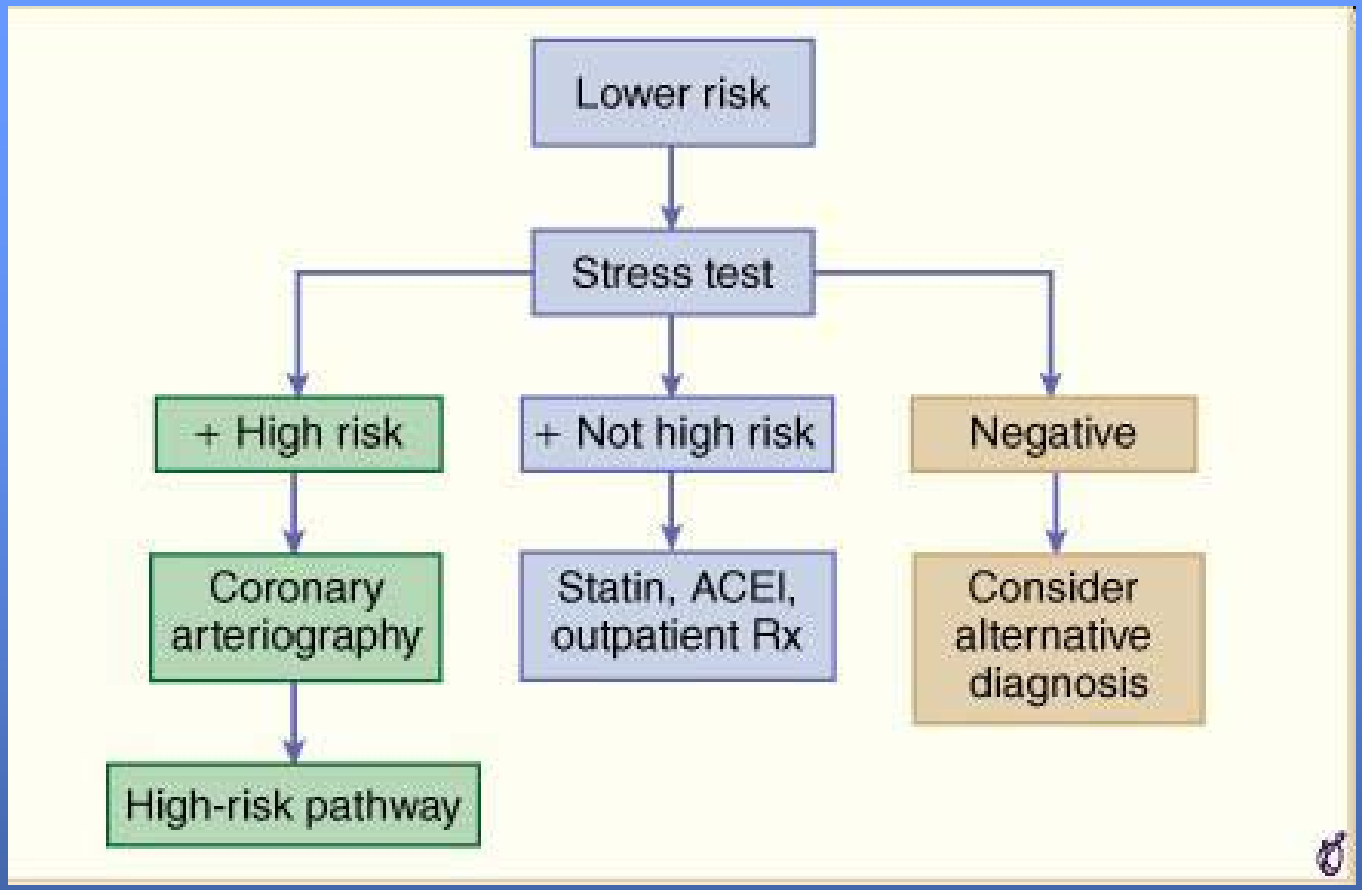


Kaplan-Meier event curves of three trials comparing invasive versus conservative strategies

FRISC II, Lancet 2000: 356; 9

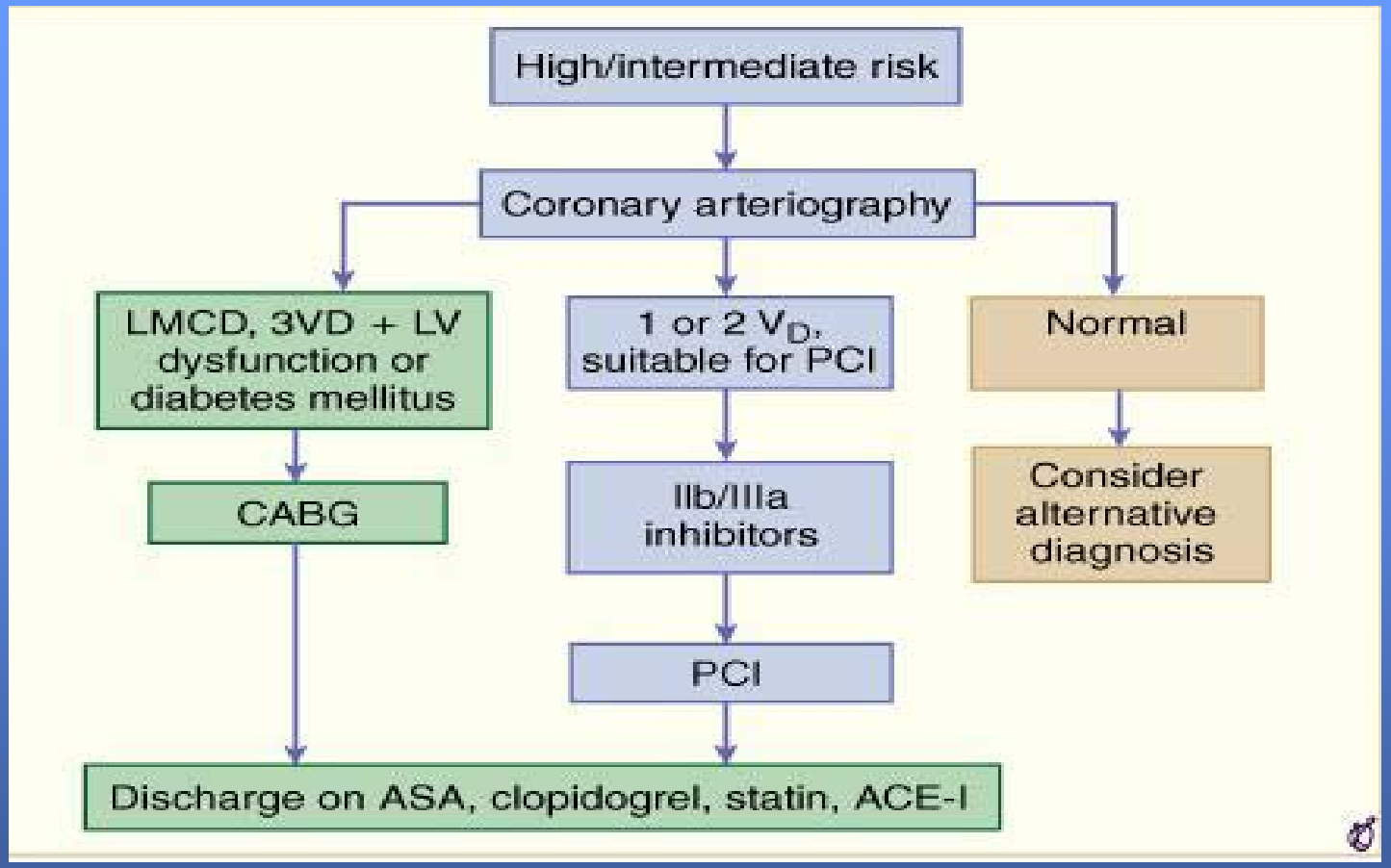
TACTIS-TIMI 18, N Engl J Med 2001: 344;1879

RITA-3, Lancet 2002: 360:743



Management of lower risk patients with unstable angina or non-ST elevation myocardial infarction

Circulation 2003:108; III-28



Management of high- and medium-risk patients with unstable angina or non-ST elevation myocardial infarction

Circulation 2003:108; III-28

Coronary Angiographic Findings

TACTICS-TIMI 18 invasive arm

34 % significant obstruction (>50 percent luminal diameter stenosis) of three vessels

28 % two vessel disease

26 % single vessel disease

13 %t no coronary stenosis >50 %.

5 -10 % left main stem stenosis >50 %.

Registries of unselected UA/NSTEMI patients have reported similar findings. Women and non-whites with UA/NSTEMI have less extensive coronary disease than their counterparts, whereas patients with NSTEMI have more extensive disease than those who present with unstable angina.

Fragmin and Fast Revascularisation during Instability in Coronary artery disease (FRISC II) Investigators.

- Patients within 48 h UA/NSTEMI
- Early invasive vs conservative - dalteparin vs placebo
- 3048 patients → dalteparin for 5–7 d → 2457 continued dalteparin/placebo & received either invasive or conservative strategy
- Medication: ASA, β-blockers unless contraindicated

✓ No ↓ death/MI at 3 months by dalteparin

✓ **↓ Death/MI at 6 mo, 1 y and 5 y for invasive strategy**

Wallentin L, et al. *Lancet* 2000;356:9–16 (1-year results). Lagerqvist B, et al. *J Am Coll Cardiol* 2001;38:41–8 (women vs men). Lagerqvist B, et al. *Lancet* 2006;368:998–1004 (5-yr follow-up).

Treat Angina with Aggrastat and Determine Cost of Therapy with an Invasive or Conservative Strategy (TACTICS-TIMI-18)

- 2,220 patients within 24 h UA/NSTEMI
- Medication: ASA, heparin and tirofiban
- Early invasive treatment (4-48h) or conservative treatment (coronary angiography and PCI only if objective recurrent ischemia present)

✓ **↓ Death, MI, and rehospitalisation for an ACS at 6 months for invasive strategy**

Cannon CP, et al. *N Engl J Med* 2001;344:1879–87.

Third Randomized Intervention Treatment of Angina (RITA-3)

- 1,810 moderate-risk ACS patients
- Early invasive or conservative (ischemia-driven) strategy

✓ ↓ Death, MI, & refractory angina for invasive strategy

✓ ↓ in refractory angina

✓ ↓ Death/MI at 5 y for early invasive arm

Fox KA, et al. *Lancet* 2002;360:743–51. Fox KA, et al. *Lancet* 2005;366:914–20 (5-y results).

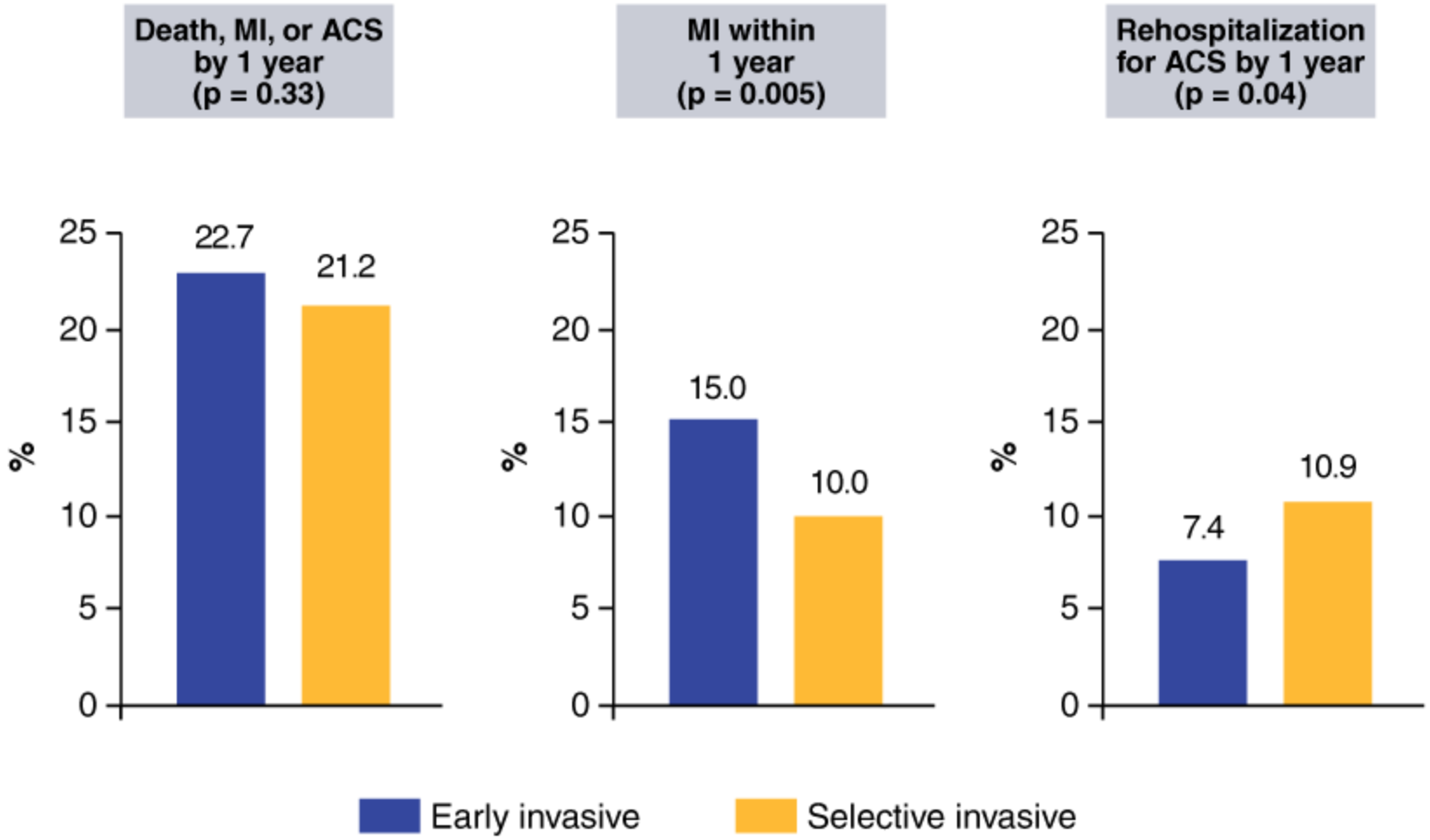
Invasive versus Conservative Treatment in Unstable coronary Syndromes (ICTUS)

- 1,200 high-risk ACS patients.
- Routine invasive vs selective invasive strategy.
- Medication: ASA, clopidogrel, enoxaparin, and lipid-lowering agents; abciximab for revascularisation patients.

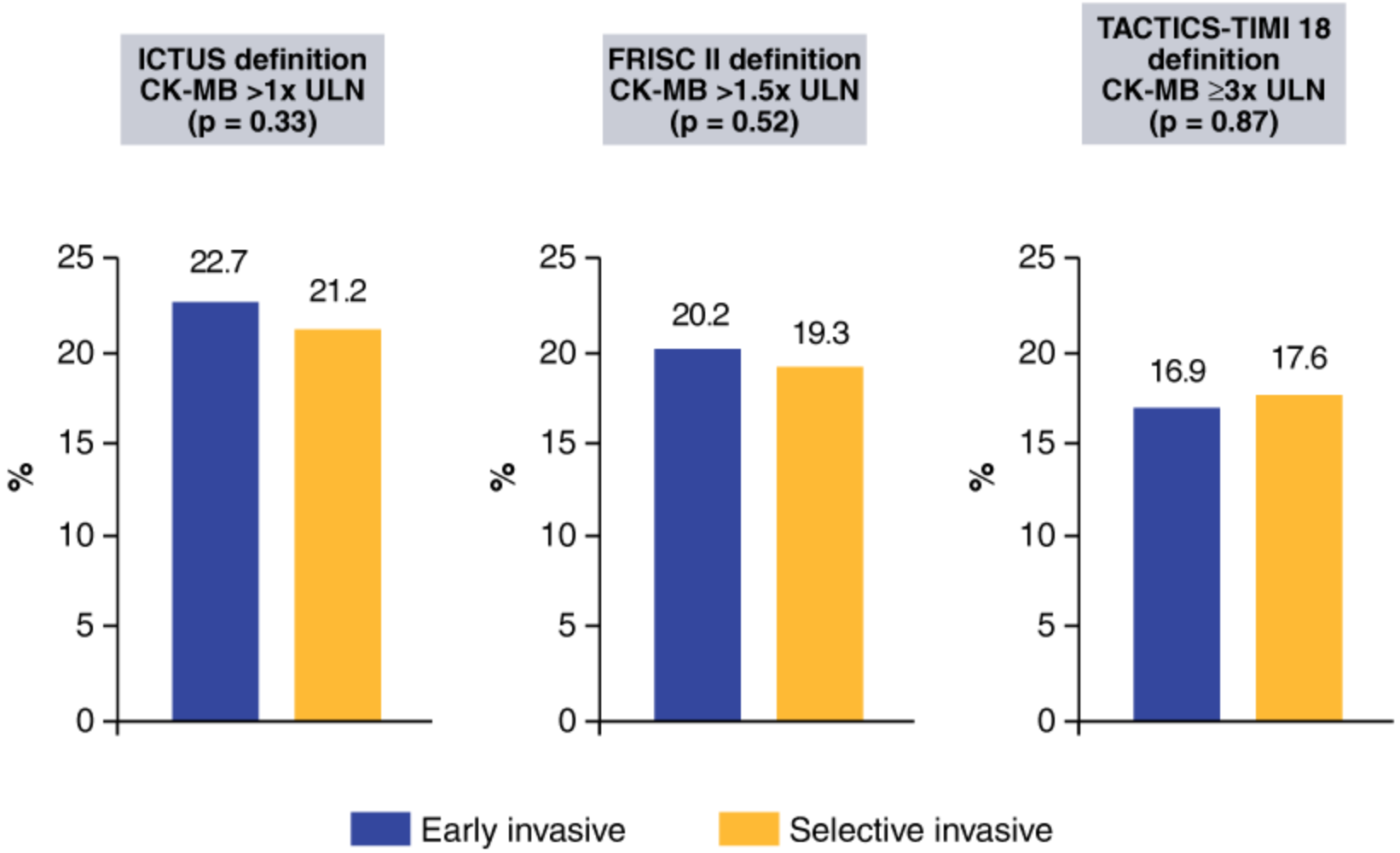
✓No ↓ death, MI, and ischemic rehospitalisation 1y and longer-term follow-up by routine invasive strategy.

de Winter RJ, et al. *N Engl J Med* 2005;353:1095–104.

ICTUS: Early Invasive vs. Selectively Invasive Management of ACS



Primary Composite Endpoint of ICTUS Using Different Definitions of Myocardial Infarction



ULN = upper limit of normal.

Timing of Intervention

- Few studies have shown superiority of **very early** intervention vs. deferred intervention.

  ISAR-COOL (small sample size) JAMA 2003;290:1593

- Many trials, registries and meta-analysis have shown early hazard with **early intervention vs. deferred** intervention.

  GRACE and CRUSADE Registry Heart 2007;93:177 and Arch Intern Med 2006;166:2027

  Mehta Meta-Analysis JAMA 2005;293:2908

  ICTUS study NEJM 2005;353:1095

- Timing of intervention recommended on the basis of risk stratification.

Intracoronary Stenting with Antithrombotic Regimen Cooling-off Study (ISAR-COOL)

- 410 patients within 24 h moderate-high risk UA/NSTEMI.
- Very early angiography (median time 2.4 h) + revascularisation or delayed invasive/“cooling off” (median time 86 h) strategy.
- Medication: ASA, heparin, clopidogrel (600mg) and tirofiban.

✓↓ Death/MI at 30 d for early angiography group.

Neumann FJ, et al. *JAMA* 2003;290:1593–9

Outcomes of the ISAR-COOL study during 30d

Event	No. (%)		RR (95% CI)	*P* Value
	Prolonged Antithrombotic Pretreatment (n = 207)	Early Intervention (n = 203)		
Death and nonfatal MI	24 (11.6)	12 (5.9)	1.96 (1.01-3.82)	.04
Death	3 (1.4)	0		.25
Nonfatal MI	21 (10.1)	12 (5.9)	1.72 (0.87-3.40)	.12
Q-wave	7 (3.4)	4 (2.0)	1.72 (0.51-5.77)	.54
Non–Q-wave	14 (6.8)	8 (3.9)	1.72 (0.74-4.00)	.21
Major bleeding event	8 (3.9)	6 (3.0)	1.31 (0.46-3.70)	.61
Nadir platelet count $<20 \times 10^3/\mu L$	2 (1.0)	1 (0.5)	1.96 (0.18-21.5)	>.99

Abbreviations: CI, confidence interval; MI, myocardial infarction; RR, relative risk.

Neumann FJ, et al. *JAMA* 2003;290:1593–9

Value of First Day Angiography/Angioplasty In Evolving Non-ST Segment Elevation Myocardial Infarction: An Open Multicenter Randomized Trial The VINO Study

- 131 patients, NSTEMI.
- 1st day angiography/angioplasty vs. early conservative therapy.

- ✓ Death/Reinfraction within 6mo 6.2% vs. 22.3%.
- ✓ 6month-mortality 3.1% vs. 13.4%.

Spacek et al. Eur Heart J 23 (3): 230.

CRUSADE Registry (Can Rapid Risk Stratification of UA Suppress Adverse Outcomes with Early Implementation of the ACC/AHA Guidelines?)

- Retrospectively classified pts:
  - very early catheterisation (23,4h)
  - later (46,3h)
- No difference in hospital death and MI.
- No exclusion of important risk reduction for early catheterisation within 12h of presentation.

Timing of Intervention in patients with NSTEMI-ACS in the CRUSADE Registry

In Hospital Events	46,3h	23,4h	p value
Death (%)	4,4	4,1	0,23
Recurrent MI (%)	2,9	3,0	0,36
Death/MI (%)	6,6	6,6	0,86

CRUSADE Quality Improvement Initiative

- Investigation of the use of early invasive management (within 48h) in high-risk. NSTEMI pts (positive early markers and/or ischemic ECG changes).

- The risk for death and MI was lower for pts who underwent early invasive management.

Outcomes of the CRUSADE Trial: In-Hospital Death or Myocardial Infraction

Outcome	No EIM	EIM	Adj. Odds Ratio
Mortality (%)	6,2	2,0	0,63
Post-admission MI (%)	3,7	3,1	0,95
Death/MI (%)	8,9	4,7	0,79

EIM: Early invasive management

ELISA Pilot Study (Early or Late Intervention in Unstable Angina)

- 220pts with NSTE-ACS were randomised to:
  - early angio without tirofiban pretreatment.
  - late angio after pretreatment with tirofiban.

✓ Delayed angio with pretreatment with tirofiban was associated with a smaller enzymatic infract size.

✓ There was no difference in clinical outcome at 30 days.

- Limitation: unknown whether the beneficial effect of the Late strategy is due to the delay itself or due to tirofiban pre- treatment.

Recommendations for PCI in Patients With UA/NSTEMI

Early PCI is reasonable for patients with:

- no serious comorbidity.
- coronary lesions amenable to PCI.
- any of the high-risk features.
- with 1- or 2-vessel CAD with or without significant proximal LAD stenosis but with a large area of viable myocardium.
- multivessel coronary disease with suitable coronary anatomy, with normal LV function, and without diabetes mellitus.

Recommendations for invasive evaluation and PCI

- **Urgent** (<2h) when refractory or recurrent angina associated with dynamic ST-deviation (>2 mm), heart failure, life threatening arrhythmias or hemodynamic instability present.

- **Early** (<72h) followed by PCI or CABG in pts with intermediate to high-risk features.

- **Routine invasive evaluation** of pts without intermediate to high-risk features is not recommended, but non-invasive assessment of inducible ischemia is advised.

Percutaneous coronary intervention PCI

- Risk of bleeding complications should be balanced against the severity of ischemia and the patients risk profile.
- Choice of access site depends on operator expertise and local preference.
- Non-pharmacological strategies to reduce access site bleeding complications include the use of closure devices and the radial approach.
- Femoral approach is preferred in haemodynamically compromised pts topermit the use of IABP.

When is PCI not recommended ?

- Non-significant coronary disease (<50%), [plaque sealing].
- Currently no outcome data support routine PCI in non-culprit coronary obstructions, [plaque sealing].
- Pts who have indication for CABG.

Stent implantation

- Helps to reduce the threat of abrupt closure and restenosis.
- The safety and efficacy of DES has not been tested prospectively.
- Subgroup analysis of randomised trials with DES's show equal effectiveness in reducing restenosis in NSTEMI's compared to BMS's.
- Consideration should be given to the type of stent implantation when a temporary withdrawal of dual antiplatelet therapy is required due to scheduled non-cardiac surgery (BMS recommended).

Special groups I

- Elderly patients (>75 years)

  - should be considered for routine early invasive strategy.
- - Treatment decisions for elderly, tailored according to life expectancy, comorbitities, patient wishes. Inherently raised risk of procedure-related complications.

- Women

  - should be considered according to the same principles as men.

- Diabetics

  - tight glycemic control to achieve normoglycaemia as soon as possible in the acute phase.

  - early invasive strategy.

  - GP IIb/IIIa inhibitors consist part of the initial medical management which should be continued through the completion of the PCI.

Special groups II

- Chronic kidney disease

  - CKD with CrCl<60ml/min are high risk for further ischemic events and therefore should be submitted to invasive evaluation and revascularisation whenever possible

  - CrCl and/or GFR should be measured for every hospitalised patient

  - Patients with CKD should receive the same first line treatment as any other patient, in the absence of contraindication.

  - Special measures should be taken for anticoagulants and contrast induced nephropathy.

- Anaemia

  - It should be taken into consideration when assessing initial risk.

  - Low baseline haemoglobin is an independent marker of risk of ischemic and bleeding events at 30 days.

  - Measures should be taken to avoid worsening of anaemia by bleeding.

  - Transfusion should be considered only in case of compromised. haemodynamic status.

Conclusions

- NSTEMI and UA consist a heterogenous group of manifestations of CAD.
- CAD process begins long before the acute event and continuous for years after the acute event.
- The optimal management of UA and NSTEMI is still evolving. Further studies are needed.

Thank You!