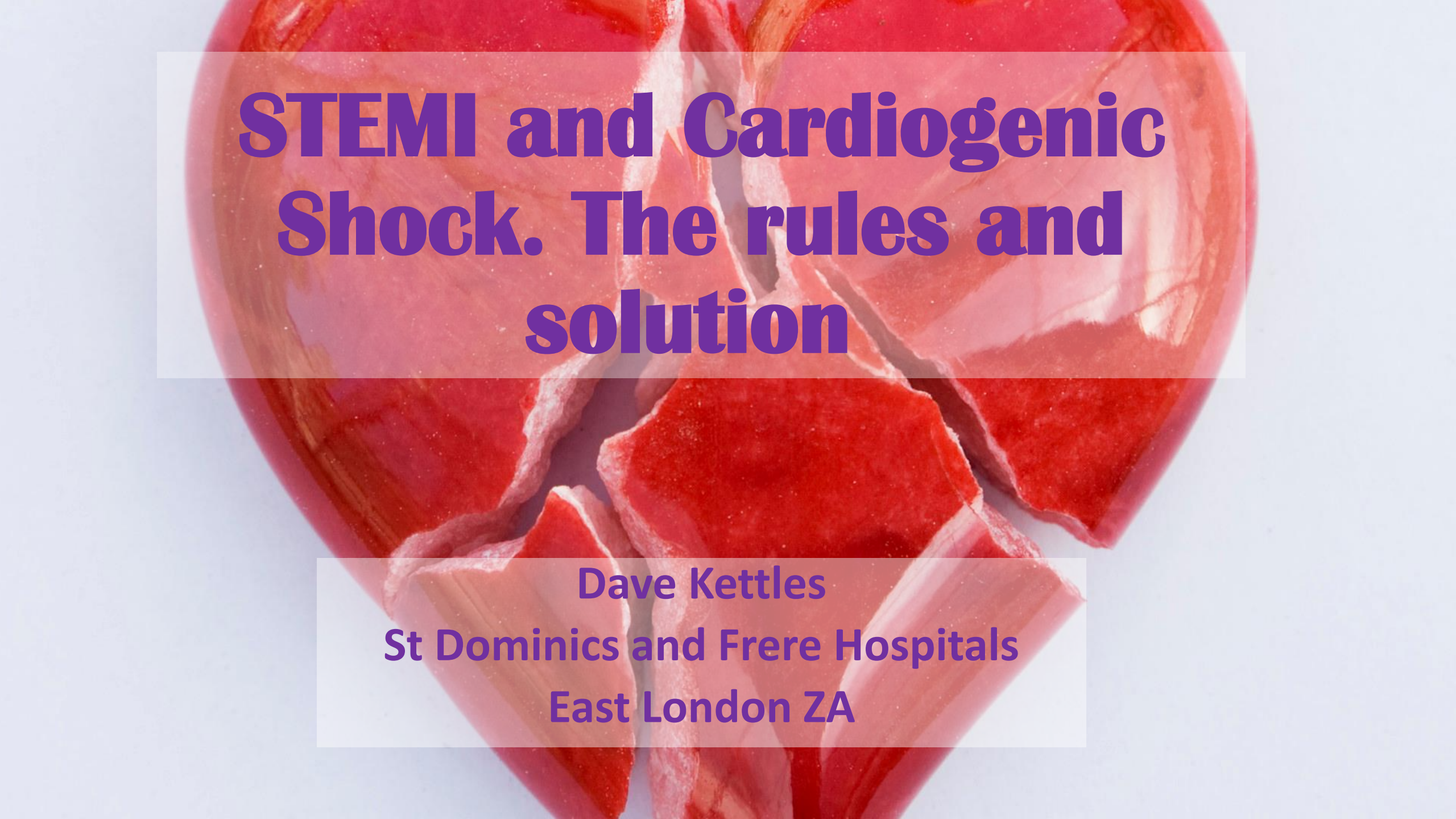

STEMI and Cardiogenic Shock. The rules and solution

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Definitions:

Shock is a life threatening, but initially reversible state of cellular and tissue hypoxia

- Prolonged shock: end organ damage, multiorgan failure and death

Cardiogenic shock: pump failure

STEMI: ischaemia, myocyte necrosis leads to pump failure

‘Clinical condition of inadequate tissue perfusion due to the inability of the heart to pump an adequate amount of blood’

- Low BP, Low CI, with normal or elevated filling pressures
- Result: hypoperfusion syndrome, pulmonary congestion, systemic venous congestion

STEMI Cardiogenic shock

5-9% of STEMI, lower in reperfusion era

Hospital fatality rates remain 40-50% (35% has been reported)

British heart attack study:

- 21 210 patients treated in 8 centres 2005-2015
- 1890 pats with CS: (8.9%)
- Increasing mortality over the course of the study, 45-70%

Case fatality rates are declining:

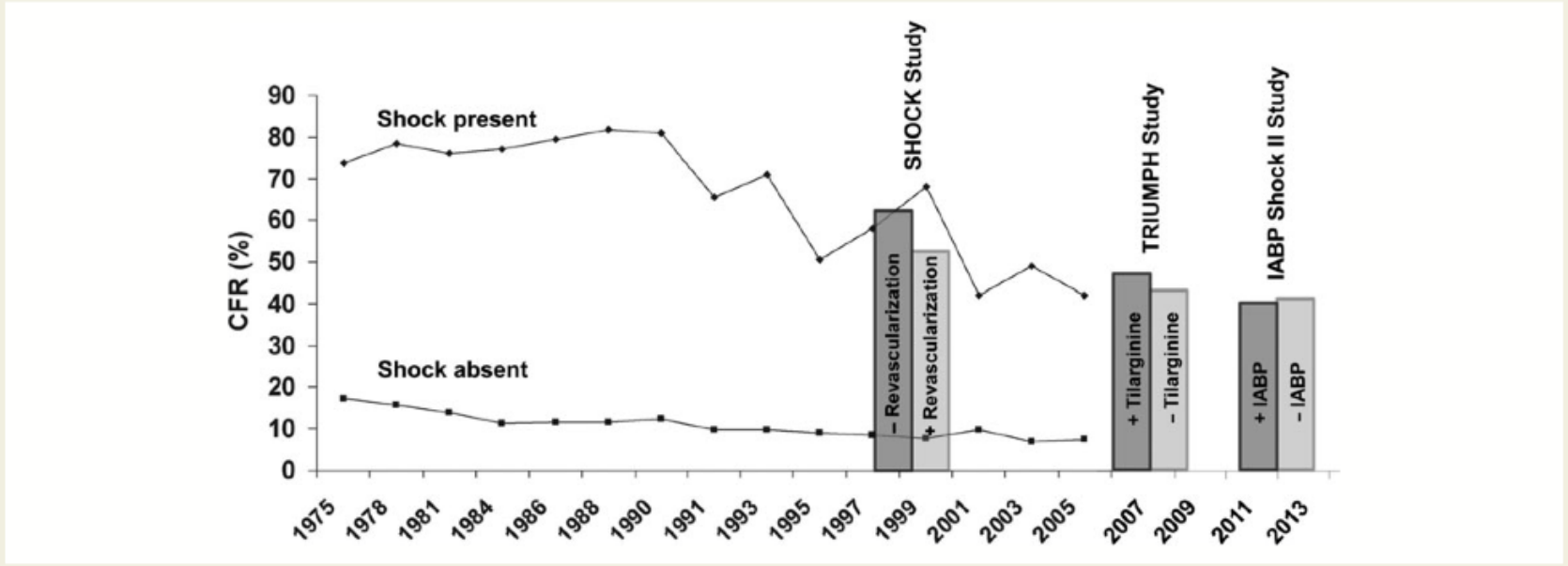


Figure 1 Time trends in hospital case fatality rates (CFR) in patients with acute myocardial infarction ± cardiogenic shock in the Worcester (MA, USA) metropolitan area. Despite the survival improvement resulting from more widespread use of acute interventional reperfusion strategies, overall cardiogenic shock mortality rates remain high at over 40%. The additional columns (from left to right) represent mortality rates from (*A*) the SHOCK study[2] (dark grey column: IMS, Initial Medical Stabilisation Group; light grey column: ERV, Early Revascularisation Group); (*B*) the TRIUMPH Study[52] [all patients with ERV; with (dark grey column) or without (light grey column) the nitric oxide synthase (NOS) inhibitor Tilarginine]; and (*C*) the IABP SHOCK II Trial[4] [all patients with ERV; with (dark grey column) or without (light grey column) intraaortic balloon counterpulsation (IABP)]. Modified from Goldberg *et al.*[80]

Werdan, Gielan, et al. E H J (2014) 35, 156–167

Mechanisms

Acute myocardial dysfunction: anteroseptal STEMI (40% LV mass infarcted) commonest

Previous MI scar, or Ischaemic from Multivessel disease can contribute

Acute severe RV failure contribution in 5%

Consider Mechanical complications

STEMI cardiogenic shock

Low CO combined with high SVR results in poor tissue perfusion

(may have normal SVR with inflammatory response)

Vicious circle of reduced coronary perfusion, further ischaemia, worsening pump failure, worsening hypoperfusion, ultimately death

Cardiogenic shock is the leading cause of death in STEMI patients

Diagnosis of CS

Mostly clinical diagnosis: appropriate MI history, hypotension (often SBP less than 90mmHg), hypoperfusion, pulmonary congestion

Appropriate ECG

Echo: Structure, regional wall motion, filling pressures, contractility and complications: SHOCK trial: **mean EF 31%**

mandatory, but can be difficult, eg miss flail MR (TEE?)

Angiographic evaluation for all, immediate,

Consider Left ventriculogram

Consider PA balloon tipped catheter – SBP, CI, PCWP, SVR, RA all help!

Diagnosis

Main differentials

- HCM, with eg vasodilatation
- Stress CMO, Takutsubo (possible dynamic LVOT obstruction)
- Acute myopericarditis
- Massive PE
- Acute valvular lesions
- Aortic dissection with infarction

Timing?

GUSTO-1 and SHOCK: 50-75% develop shock within 24 hours

Gradual development, progressively lowering blood pressure common

Late cardiogenic shock, >24 hours

- Reinfarction/reocclusion
- Mechanical complications
- Iatrogenic

Can we predict who will develop shock?

age >70 years

prior stroke/transient ischaemic attack

cardiac arrest upon admission

anterior STEMI

first medical contact-to-pPCI delay >90 min

Killip class

heart rate >90/min

A combination of systolic blood pressure <125mmHg and pulse pressure <45 mmHg

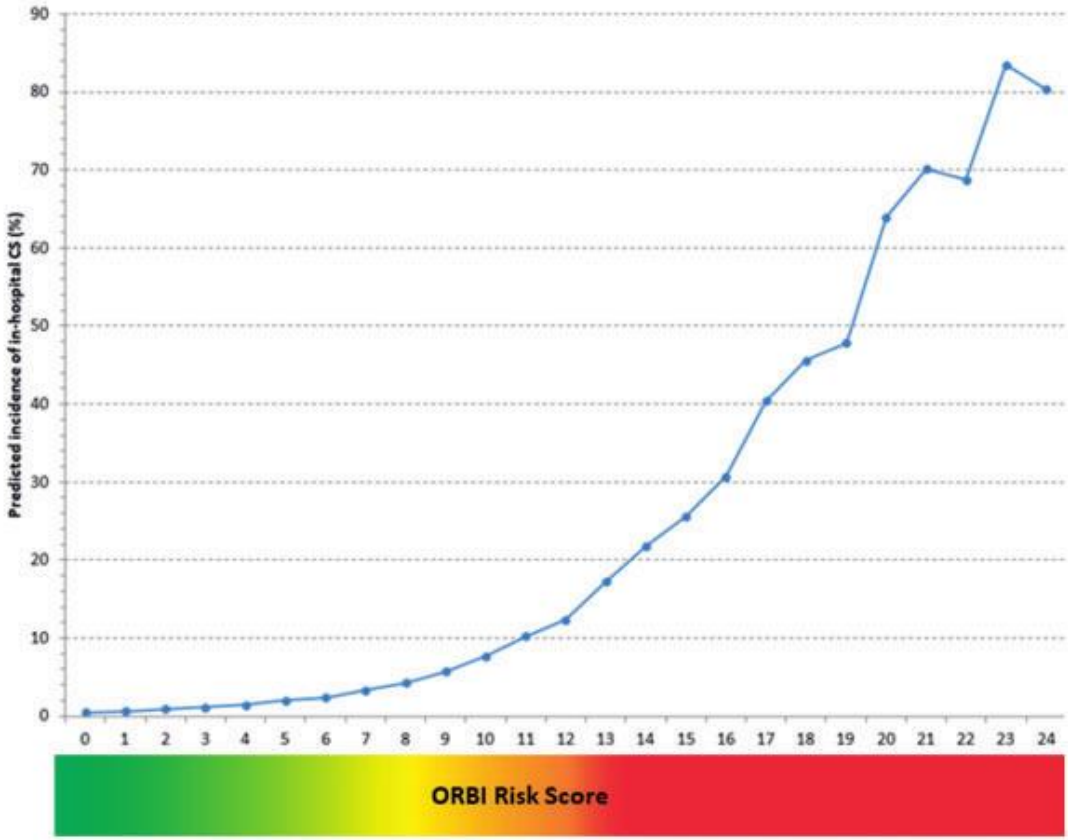
glycaemia >10 mmol/L

Culprit lesion of the left main coronary artery

post-pPCI thrombolysis in myocardial infarction flow grade <3

Auffret V, Cottin Y et al, European Heart Journal (2018) 39, 2090–2102

Variable	Points
Age > 70 years old	2
Previous stroke / TIA	2
Presentation as cardiac arrest	3
Anterior myocardial infarction	1
First medical contact-to-pPCI delay > 90 min	2
Killip class II on admission	2
Killip class III on admission	6
Heart rate > 90/min on admission	3
SBP < 125 mmHg and PP < 45 mmHg on admission	4
Glycaemia > 10 mmol/l on admission	3
Culprit lesion of the left main	5
Post-pPCI TIMI flow < 3	5



Risk categories

Category	Score	Observed incidence of CS
Low	0-7	1.3
Low-to-intermediate	8-10	6.6
Intermediate-to-high	11-12	11.7
High	≥ 13	31.8

Auffret V, Cottin Y et al, European Heart Journal (2018) 39, 2090–2102

Can we predict who will develop **late** shock?

2 high volume tertiary centres admitted 2247 patients with suspected STEMI

225 (10%) developed CS

- 56% on admission, 16% in the cath lab, 28% late
- CS vs non CS: 30 day mortality of 47% vs 3.1% mortality
- Age, previous stroke, time from symptom onset to intervention, Anterior STEMI, heart rate/SBP ratio, comatose state after resuscitation all predicted **late cardiogenic shock**

Obling, Frydlandt et al, EHJ: Acute cardiovascular care, 7(1) 2018.

Can we predict mortality?

From GUSTO-1 data base

- Age
- Previous MI
- Clinical parameters of hypoperfusion, including oliguria

- Low EF
- High lactate
- Coronary anatomy: LMS or SVG lesions
- Echo: EF<28 and mod-severe MR predict mortality (1 yr, 24 % and 31%)
- Symptom onset to reperfusion time: HUGE impact.

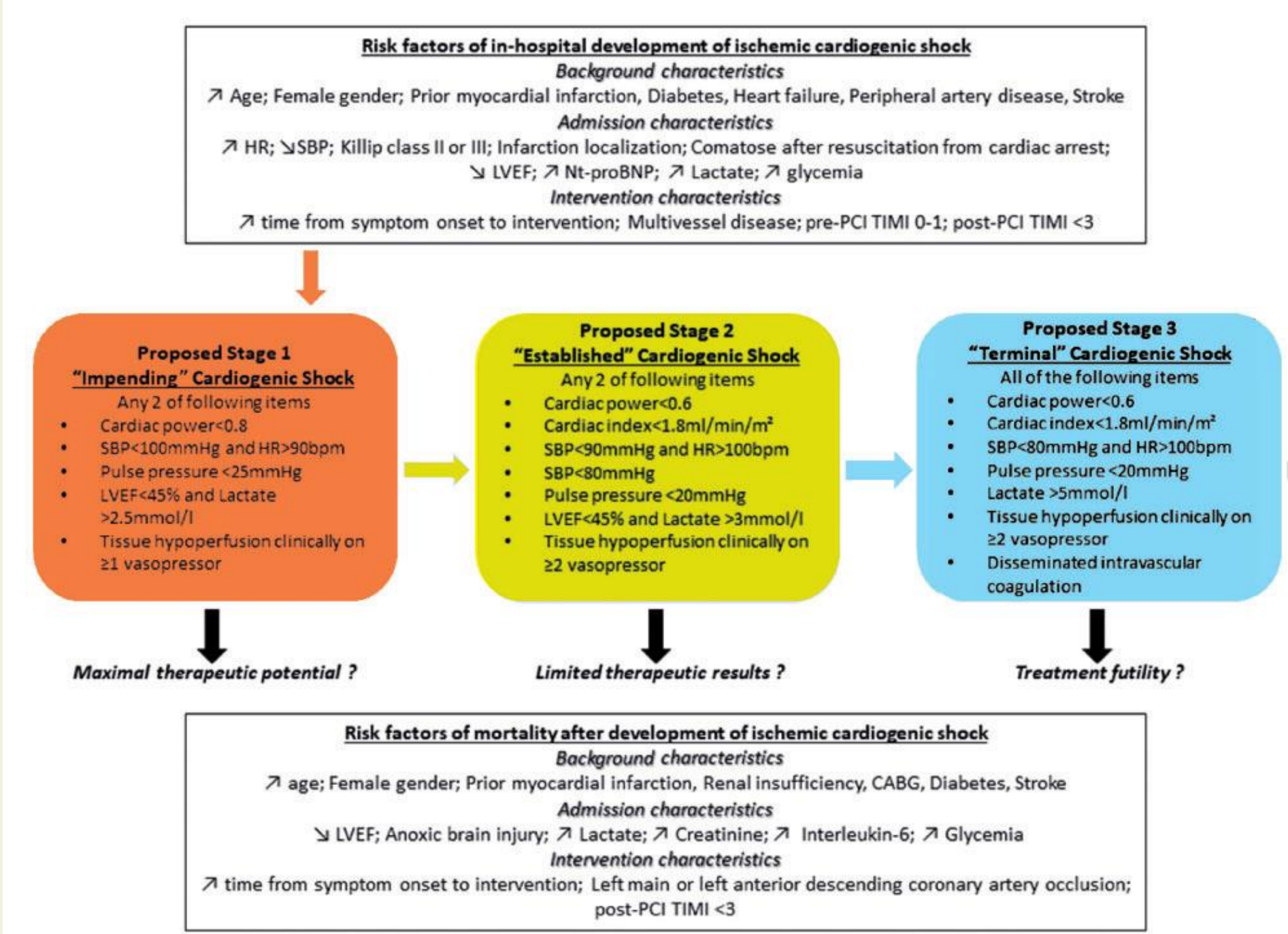
Can we predict mortality?

Early successful revascularisation predicts better outcomes

Altered mental status is a key pointer to adverse prognosis

- 215 adults with cardiogenic shock
- Lower pH, higher lactate, older age, lower SBP correlated with altered mental status
- Higher mortality at 90 days, 51% vs 22%

Kataja , Tarvasmaki, et al. EHJ: Acute Cardiovascular Care, 7(1)2018.



Gangas G, Guedeney P, European Heart Journal (2018) 39, 2103–2105

Routine treatment approaches:

Pharmacotherapy:

- Avoid clopidogrel until angio
- Avoid routine admin of drugs that lower blood pressure
- Consider IVI route for heparin in hypoperfused patients
- Consider GP 2b3a inhibitors: some mortality benefit demonstrated
- Be very careful with beta blockers in ‘pre-shock’
  - COMMIT: early beta blockers increased risk of CS if patients were
    - 70+
    - BP <120
    - HR >110
    - Killip > 1
- Similar caution with antiarrhythmics and CCB’s

Consider A –line and SGC: both are generally useful.

Routine treatment approaches:

- Judicious fluid administration
  - Consider hypovolaemia, but pulmonary congestion is an ever present risk
  - Haemodynamic monitoring helpful
  - Higher fluid volumes may be needed
    - RV infarction
    - Inferior MI with hypotension and vasodilatation
- Assisted ventilation as needed
  - Pulmonary congestion, airway protection, raise pH, assist in IABP wean.
- Manage glucose if above 10mmol, and avoid hypoglycaemia
  - Digami 1, 2, Hi 5 etc: no consensus on best strategy
  - Usually short acting insulin infusion, only as needed, and avoid glucose containing IV fluids

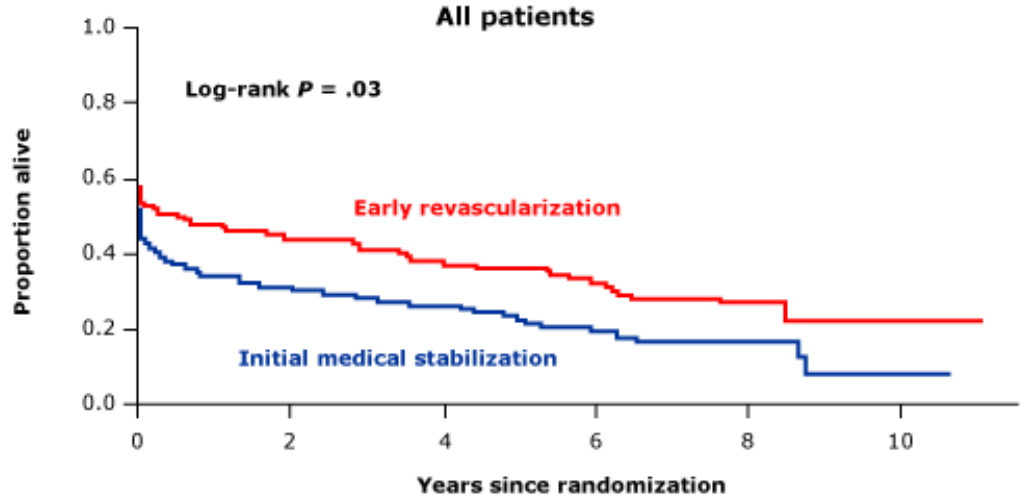
Reperfusion therapy

The New England Journal of Medicine

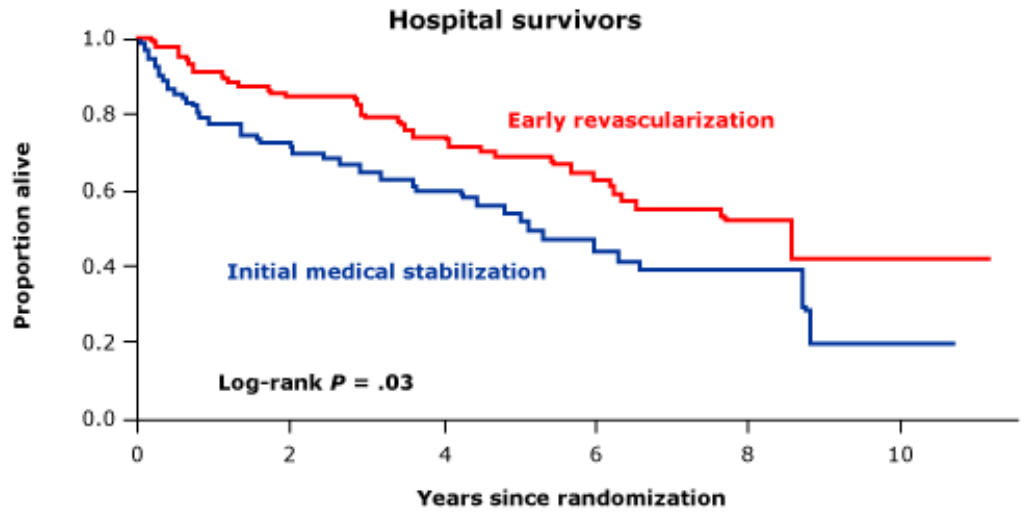
VOLUME 341 AUGUST 26, 1999 NUMBER 9

EARLY REVASCULARIZATION IN ACUTE MYOCARDIAL INFARCTION COMPLICATED BY CARDIOGENIC SHOCK

JUDITH S. HOCHMAN, M.D., LYNN A. SLEEPER, SC.D., JOHN G. WEBB, M.D., TIMOTHY A. SANBORN, M.D., HARVEY D. WHITE, D.SC., J. DAVID TALLEY, M.D., CHRISTOPHER E. BULLER, M.D., ALICE K. JACOBS, M.D., JAMES N. SLATER, M.D., JACQUES COL, M.D., SONJA M. MCKINLAY, PH.D., AND THIERRY H. LEJEMTEL, M.D., FOR THE SHOCK INVESTIGATORS*



Number at risk						
ERV	152	56	42	33	18	3
IMS	150	38	29	18	9	2



Number at risk						
ERV	77	56	42	33	18	3
IMS	66	38	29	18	9	2

Reperfusion therapy

An open artery conveys prognostic benefit

- Irrespective of how achieved
- Early is best, some benefit at least to 24 hours
- Fibrinolysis: if PCI delayed or unavailable
  - Small mortality benefit in CS shock in FTTCG 1994 meta-analysis (54 vs 61% mortality at one month)
  - SHOCK post hoc analysis: 1 year mortality reduced 60 vs 78% in medical therapy arm

Immediate PCI on the culprit lesion of the infarct related artery

- SHOCK registry, and ALKK registry: in hospital mortality is directly related to degree of reperfusion obtained at PPCI
  - Eg: 33/50/86 or 37/66/78 percentage in hospital mortality for TIMI gr 1, 2, 3 respectively

Reperfusion: CABG?

SHOCK trial: 18% LMS disease, and 53% triple vessel disease

370 patients in 22 studies in patients with STEMI and CS

- 36% in hospital mortality when CABG performed at same hospitalisation (?selection bias)
- 41% in SHOCK trial patients randomised to surgery within 6 hours
- NRMI (US data): CABG for STEMI with cardiogenic shock stable around 3%
- SHOCK trial: surgical patients had more extensive disease but similar mortality rate PCI patients at 30 days and one year

PCI or CABG: consider anatomy, local skills, availability

Sometimes PCI then CABG!

What about the non-culprit lesion in PCI patients?

PCI of non-culprit lesion is recommended in STEMI **without** shock

Only the timing is still debated

The NEW ENGLAND JOURNAL of MEDICINE

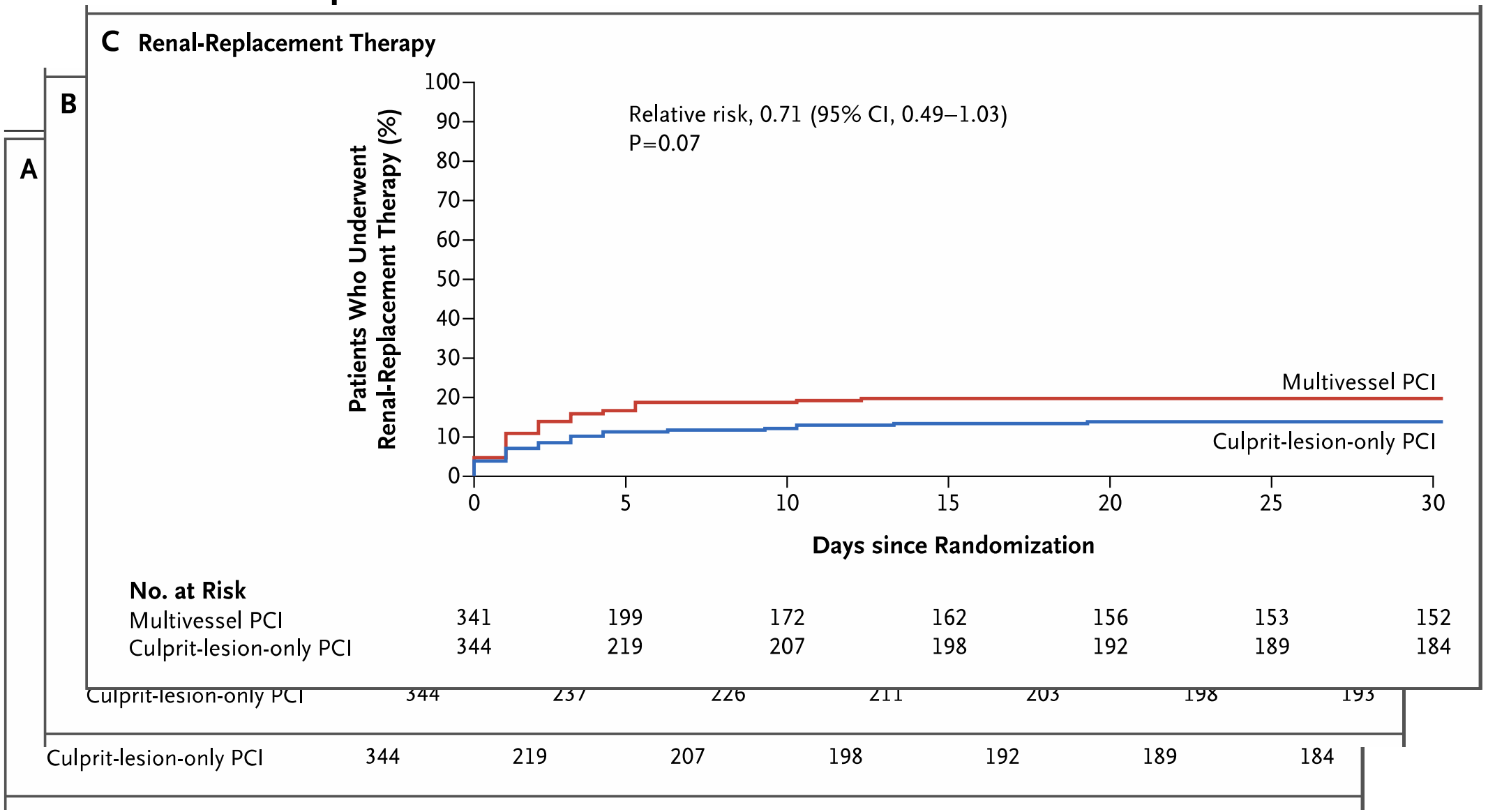
ORIGINAL ARTICLE

PCI Strategies in Patients with Acute Myocardial Infarction and Cardiogenic Shock

H. Thiele, I. Akin, M. Sandri, G. Fuernau, S. de Waha, R. Meyer-Saraei, P. Nordbeck, T. Geisler, U. Landmesser, C. Skurk, A. Fach, H. Lapp, J.J. Piek, M. Noc, T. Goslar, S.B. Felix, L.S. Maier, J. Stepinska, K. Oldroyd, P. Serpytis, G. Montalescot, O. Barthelemy, K. Huber, S. Windecker, S. Savonitto, P. Torremante, C. Vrints, S. Schneider, S. Desch, and U. Zeymer, for the CULPRIT-SHOCK Investigators*

Thiele, Akin et al, N Engl J Med 2017; 377:2419-2432

Non-culprit PCI?



Meta-analysis: culprit IRA only vs MV-PCI

10 cohort studies: 6051 patients, 20% MV-PCI

Short term mortality (in hospital): 28.8% vs 37.5%

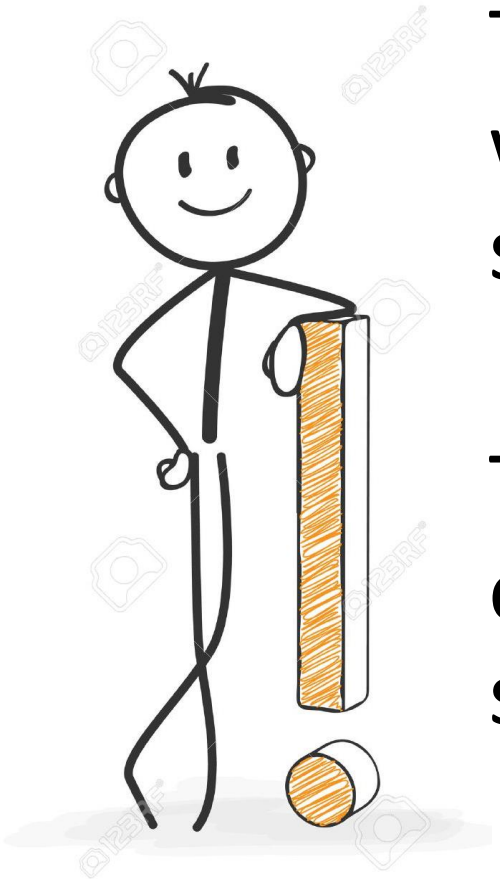
At 30 days, all-cause death, occurred less often in the culprit-lesion-only group (relative risk, 0.84; 95% CI 0.72-0.98)

Expected higher repeat revasc or rehospitalization in culprit only group

Selection bias may play a role in this result

De Waha, Jobs et al. EHJ: acute cardiovascular care. 7(1) Feb 2018

There is a solution?



Treat as many STEMI patients as possible with a timeous, effective reperfusion strategy

This ‘simple’ intervention will save the lives of the vast majority of STEMI cardiogenic shock patients