Cardiogenic Shock

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Saint Luke's
CARDIOVASCULAR CONSULTANTS

Definition

Low-cardiac-output state resulting in life threatening end-organ hypoperfusion

Criteria:

1. Persistent hypotension (SBP <80 to 90 mm Hg or MAP 30 mm Hg lower than baseline)
2. Severe reduction in cardiac output
3. Adequate or elevated filling pressure

Diagnosis

Clinical Findings:

1. Cool extremities
2. Altered mental status
3. ↓ pulses/narrow pulse pressure
4. End-organ dysfunction: oliguria, shock liver, elevated lactate

Confirmatory testing:

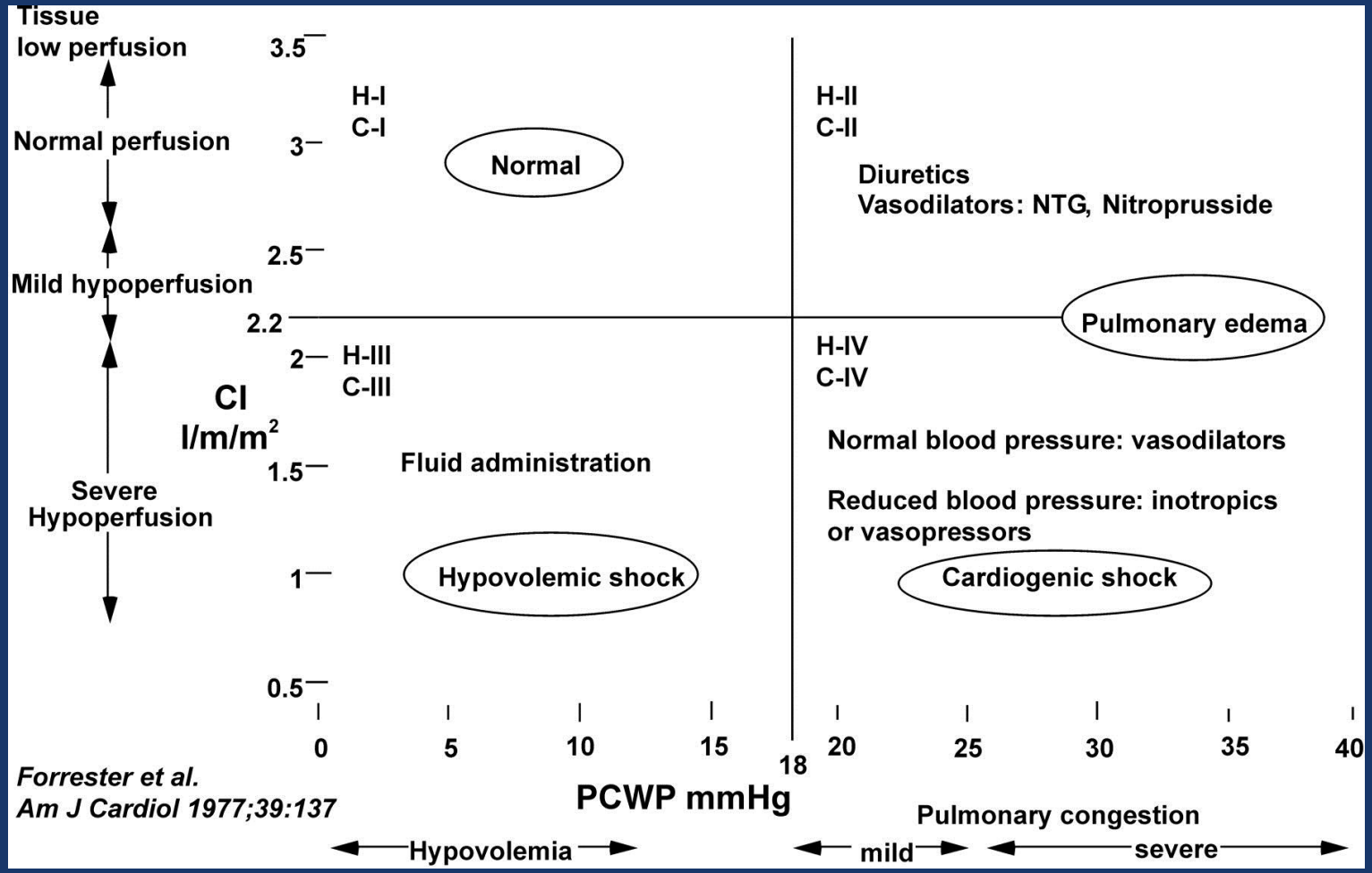
1. Pulmonary artery catheterization
2. Echocardiogram

Must Differentiate Types

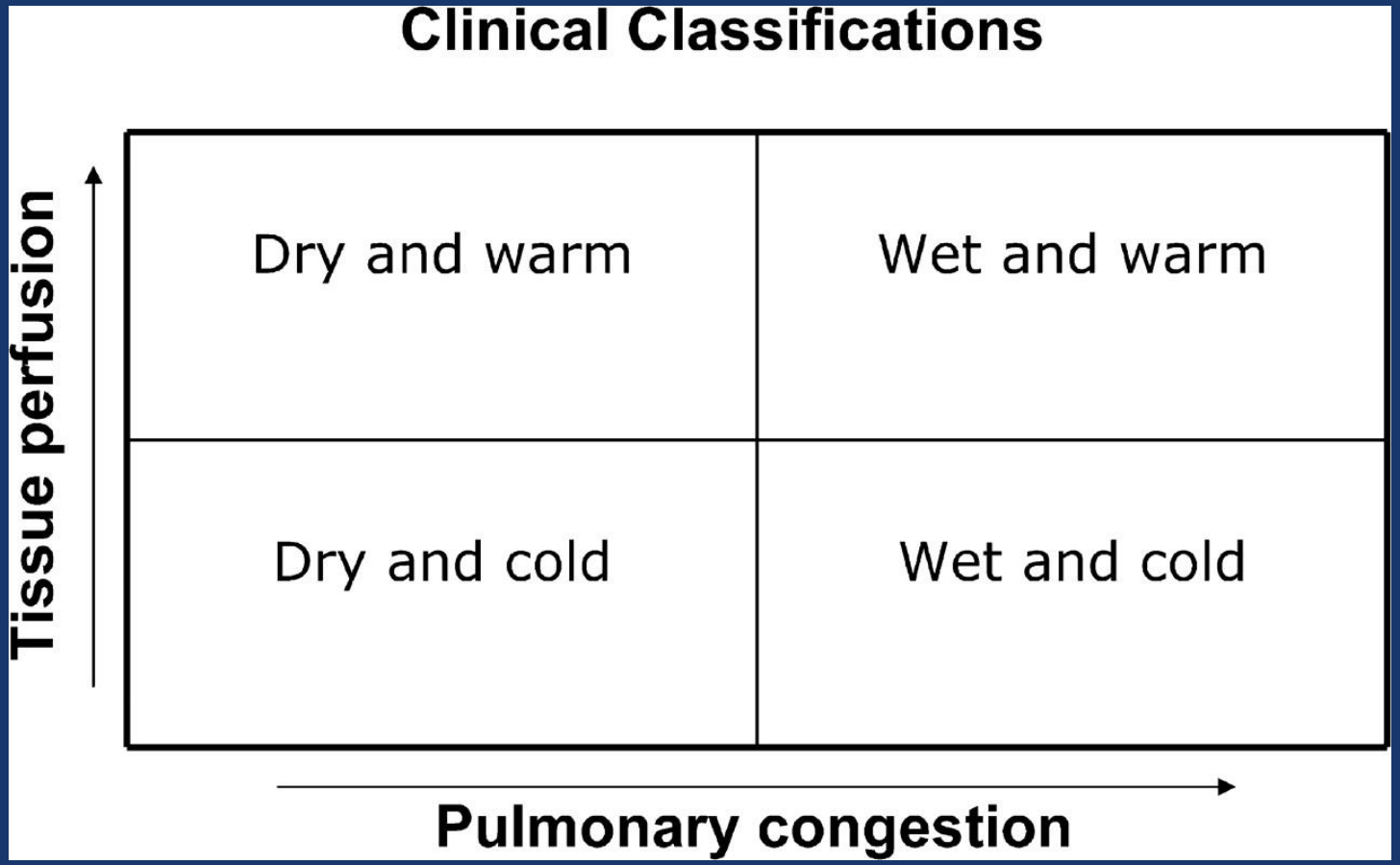
Type of shock	CO	SVR	PWP	CVP
Hypovolemic	↓	↑	↓	↓
Cardiogenic	↓	↑	↑	↑
Distributive	↑	↓↓	↓	↓

CO=Cardiac Output
SVR=Systemic vascular resistance
PWP=Pulmonary wedge pressure
CVP=Central venous pressure

Classification



Classification



Causes

Myocardial infarction with subsequent LV dysfunction

Most common cause

Mechanical Defect

1. **A**cute mitral regurgitation (papillary muscle rupture)
2. Ventricular wall rupture (free wall or septal defect)
3. Tamponade
4. Left ventricular outflow obstruction (HOCM, AS)
5. Left ventricular inflow obstruction (MS, atrial myxoma)

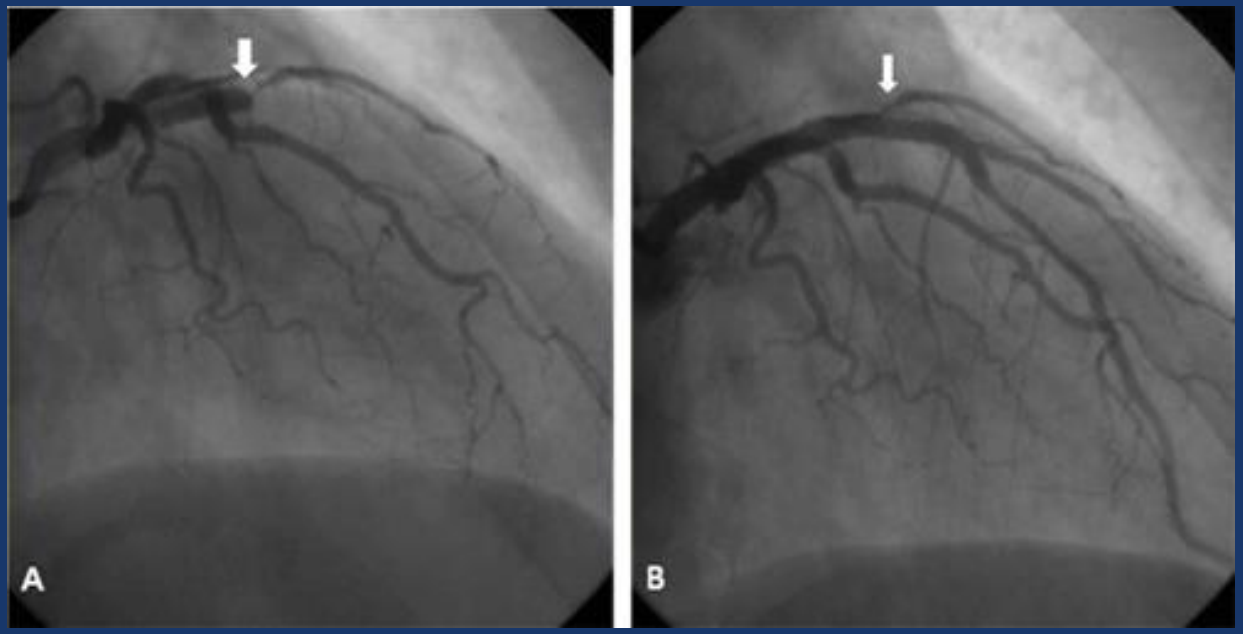
Causes – continued

Contractility Defect

1. Arrhythmias
2. Cardiomyopathy
3. Direct cardiac trauma
4. Sepsis
5. Medications
6. Myocarditis
7. Endocrine disorders
8. Pancreatitis
9. Pulmonary embolus

Myocardial Infarction

Most common cause of cardiogenic shock



Of the more than 1.2 million cases of MI that occur in the United States yearly, up to 8% will be complicated by cardiogenic shock

(5-8% of STEMI, 2.5% of NSTEMI)

Babaev, A; *JAMA* 2005 Jul 27;294(4):448-54
Fox KA; *Heart.*2007; 93: 177–182

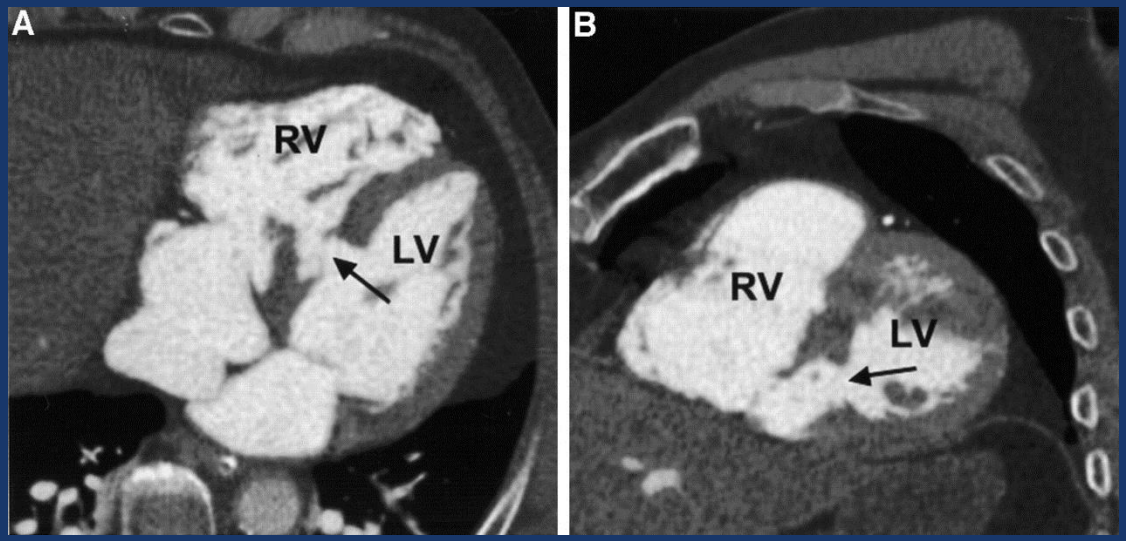
Mechanical Complications

Mechanical complications must be considered in all patients with shock in the setting of MI.

1. Ventricular septal rupture
2. Free wall rupture
3. Papillary muscle rupture

Ventricular Septal Rupture

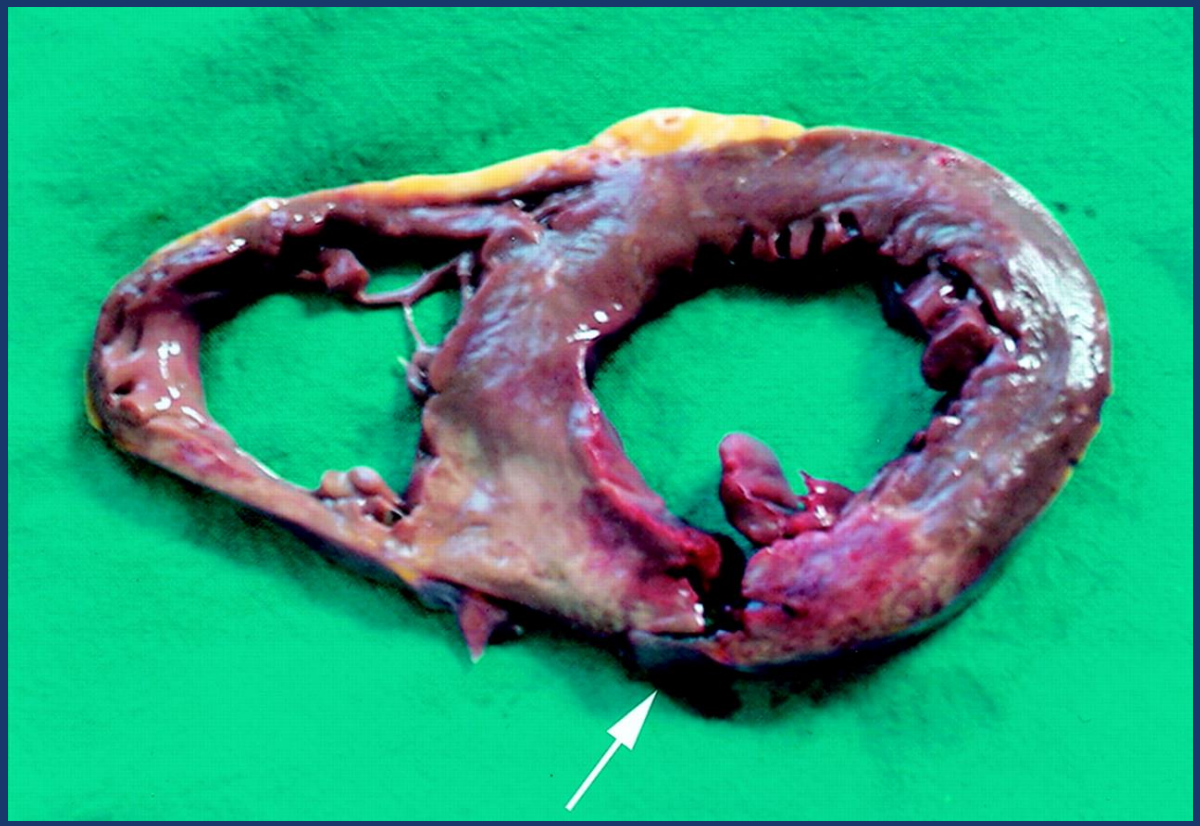
1. Occurs 2-8 days following MI
2. Loud systolic murmur, often with a thrill
3. 90% of patients die without surgery
4. Occurs more commonly in setting of first MI and/or delayed reperfusion



Circulation June 28, 2005, 25 e449-e450

Free Wall Rupture

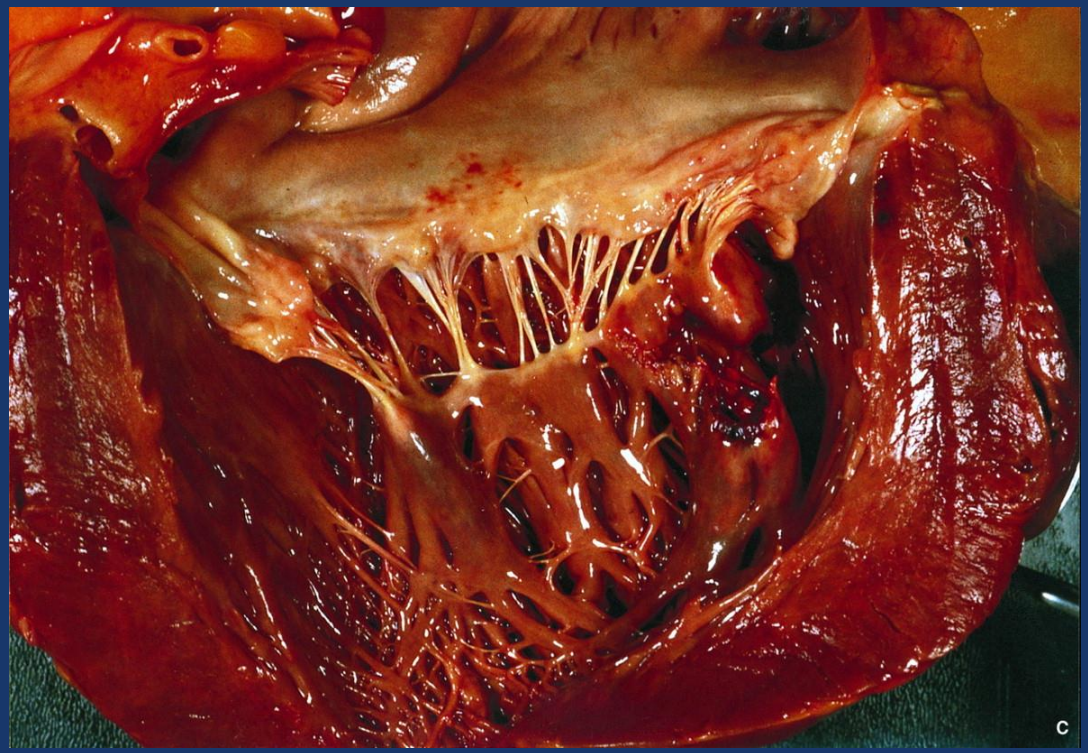
1. In-hospital mortality of > 60%
2. Risk factors first MI, advanced age, delayed presentation
3. JVD, pulsus pardoxus, diminished heart sounds
4. Emergent pericardiocentesis while transporting to operating room



Circulation 2003; 108: 498-499

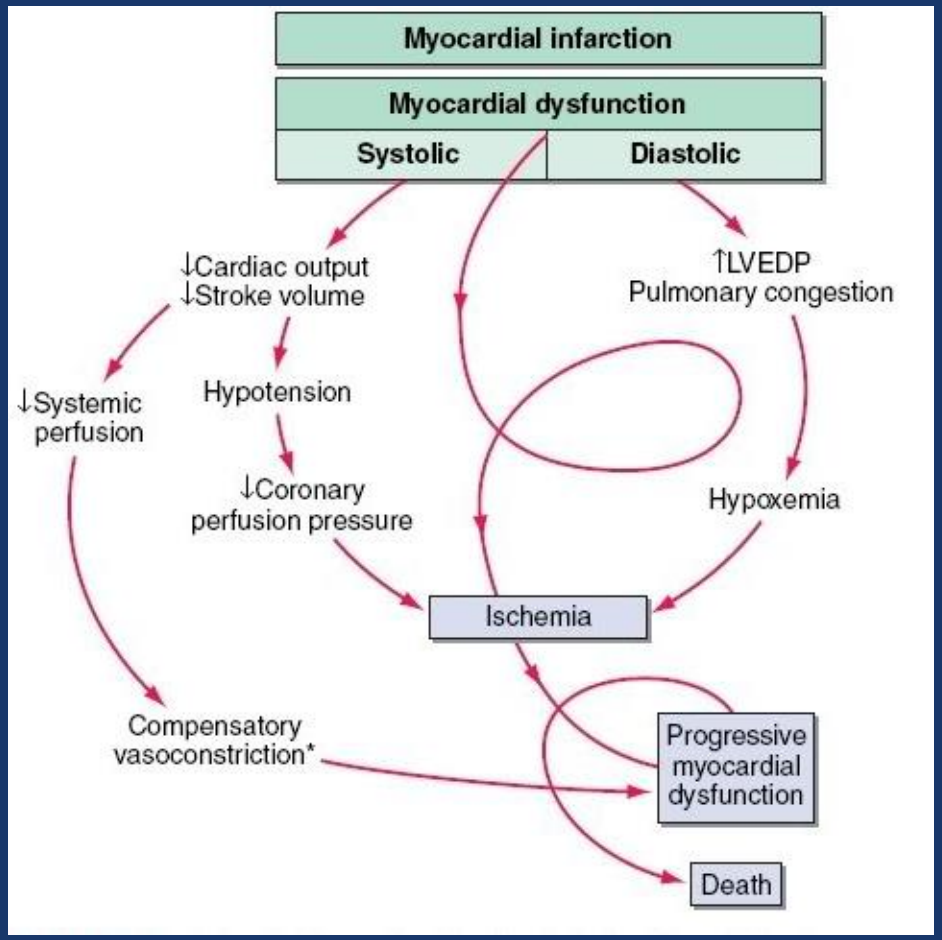
Papillary muscle rupture

1. Occurs 2-8 days following MI
2. Loud systolic murmur
3. More common with inferior MI (posteromedial papillary muscle supplied by PDA, anterolateral dual supplied by LAD & LCX)
4. IABP/vasodilators while awaiting surgery



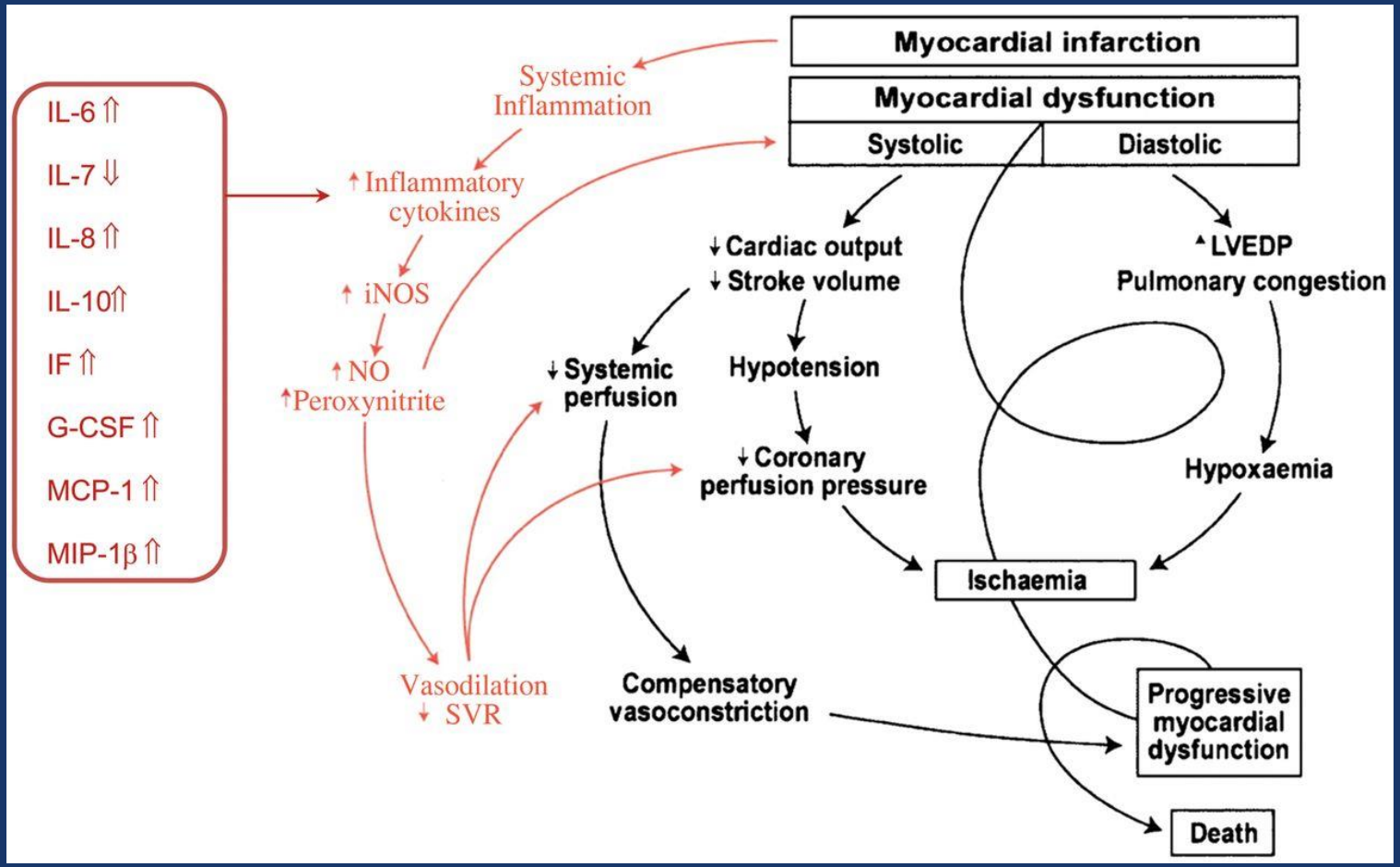
Tauke. Circulation 1997; 96: 698-699

Classic Pathophysiology



Hollenberg Ann Int Med 131:37, 1999

Contemporary Understanding



Werden. Eur Heart Jour (2014) 35, 156–167

CS Pathophysiology is Complex

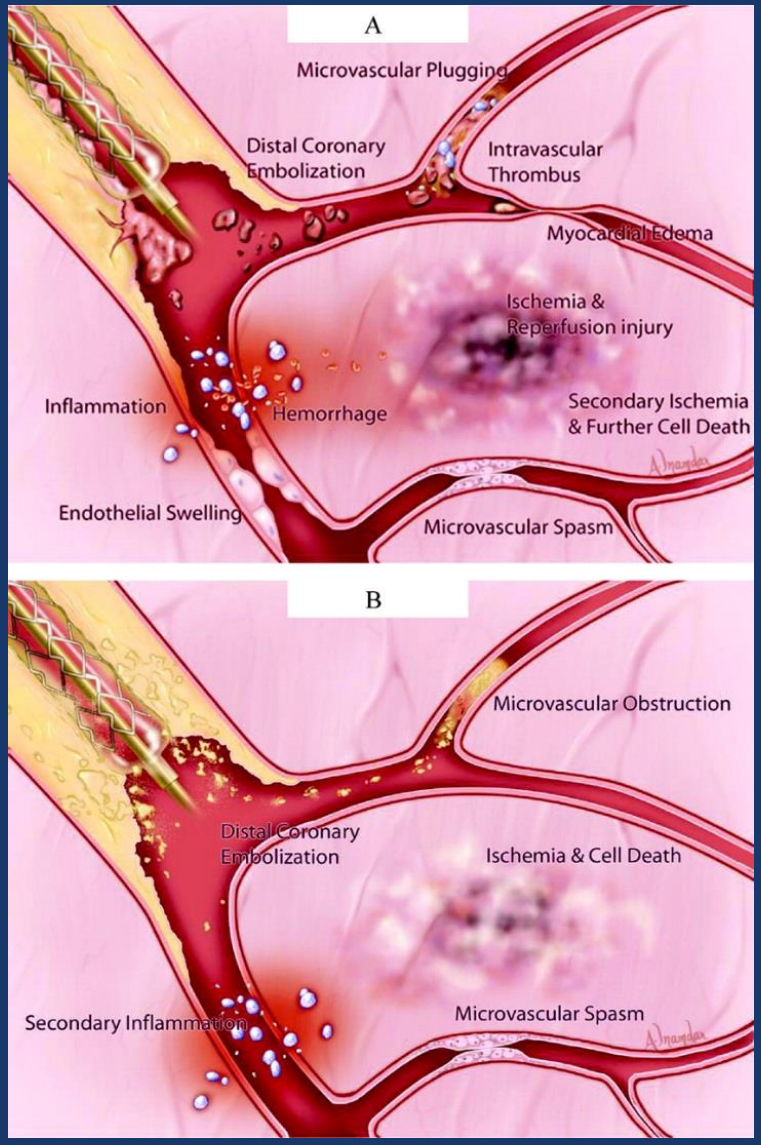
1. **The left ventricle simultaneously benefits and suffers from low afterload**
   - ↓ coronary flow
   - ↑ cardiac output with low afterload
2. **Hypoperfusion causes release of catecholamines**
   - ↑ contractility and peripheral blood flow
   - ↑ myocardial oxygen demand = ↑ afterload = myocardial dysfunction
   - ↑ arrhythmias
3. **Activation of neurohormonal cascade**
   - ↑ salt and water retention = ↑ improve perfusion & ↑ pulmonary edema

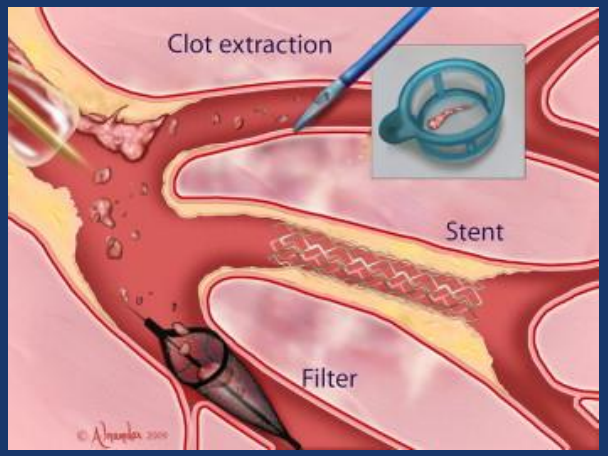
Reynolds. *Circulation* 2008; 117: 686-697

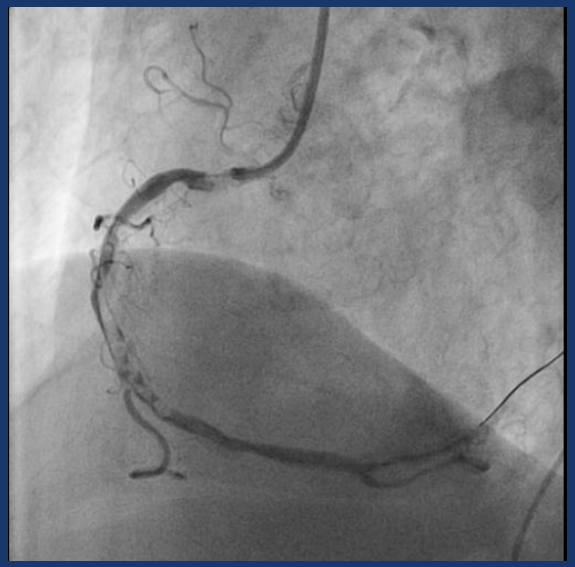
Causes of Persistent Shock

1. **No–reflow phenomenon**
2. **Reversible myocardial dysfunction: stunning of LV**
3. **Vasodilatory pathway of shock:**
   - ↑ levels of inflammatory cytokines
   - ↑activity of the inducible form of nitric oxide synthase (iNOS) in infarcted heart muscle
4. **Persistent ischemia in non-infarct related artery**
   - More than 2/3rd of patients with MI and CS have multivessel disease
5. **Bleeding**

CS due to No-Reflow

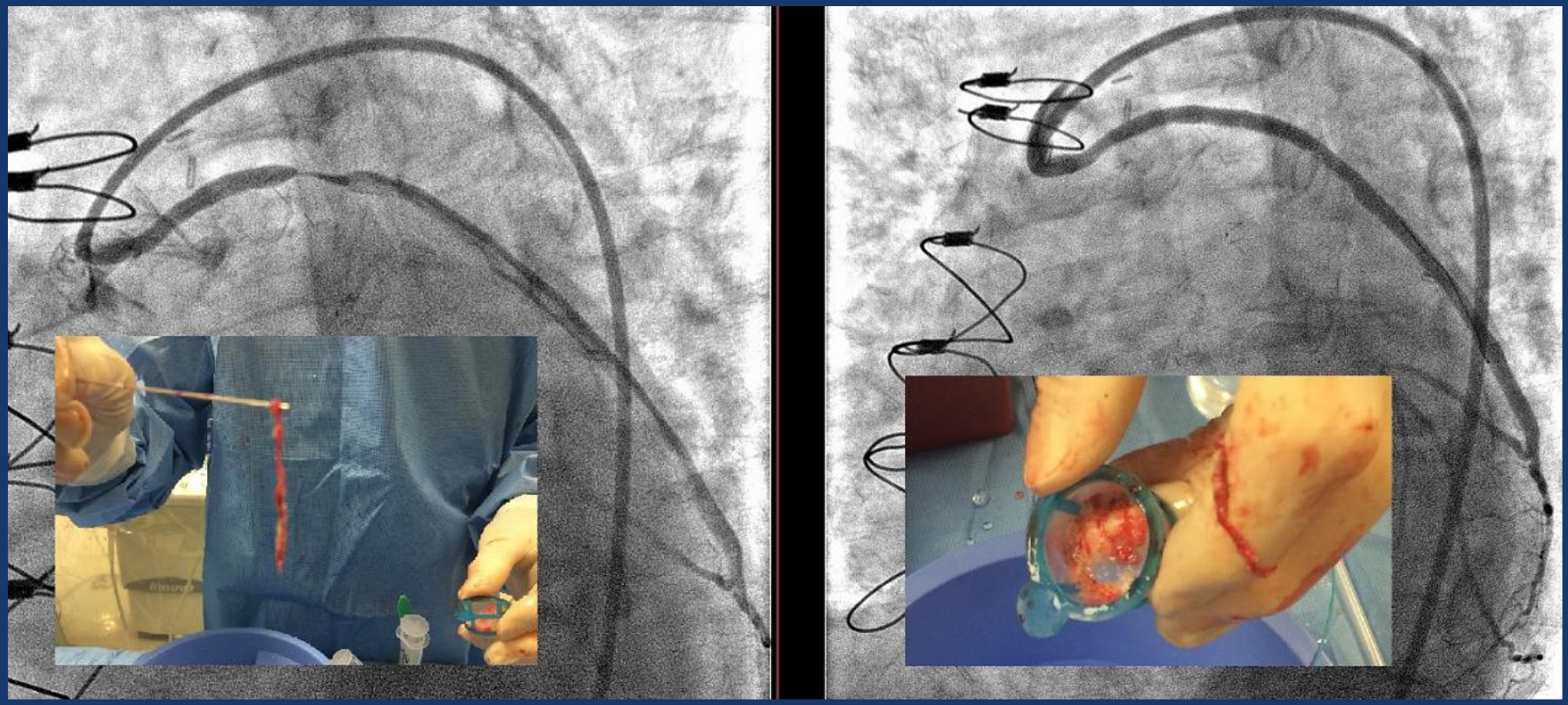




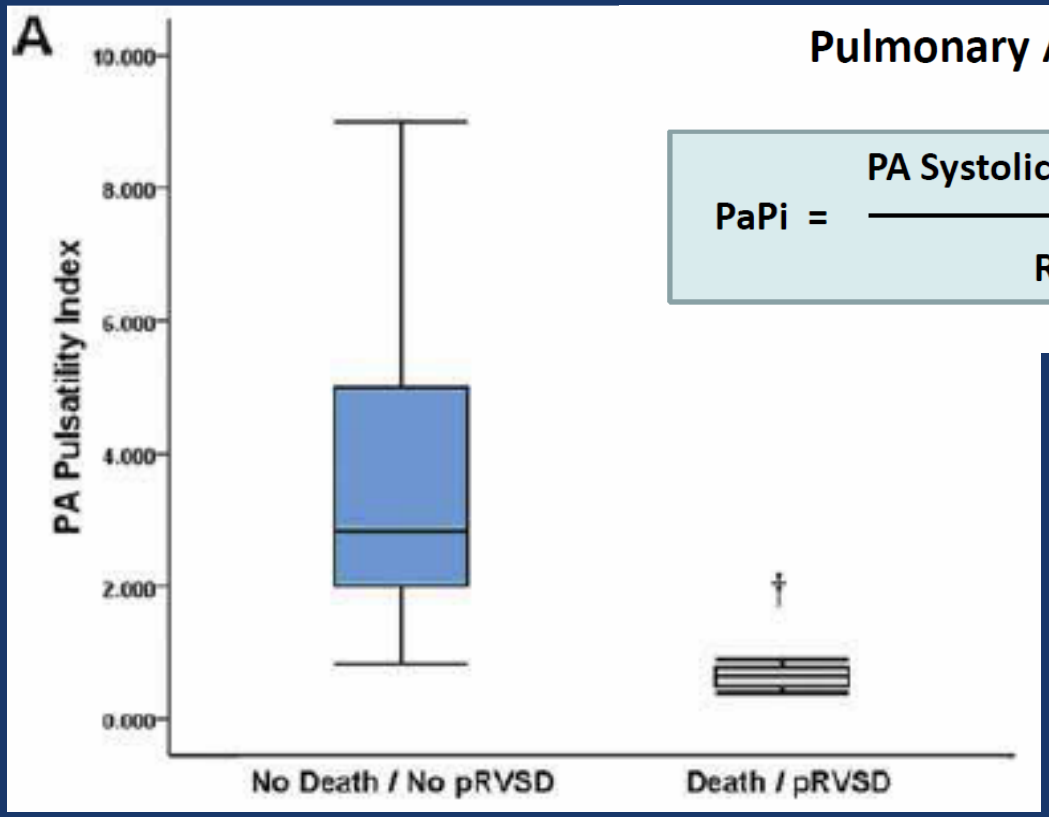


Jaffe. *Circulation* 117 2008:3152-3156.

CS due to No-Reflow



Right Ventricular Failure

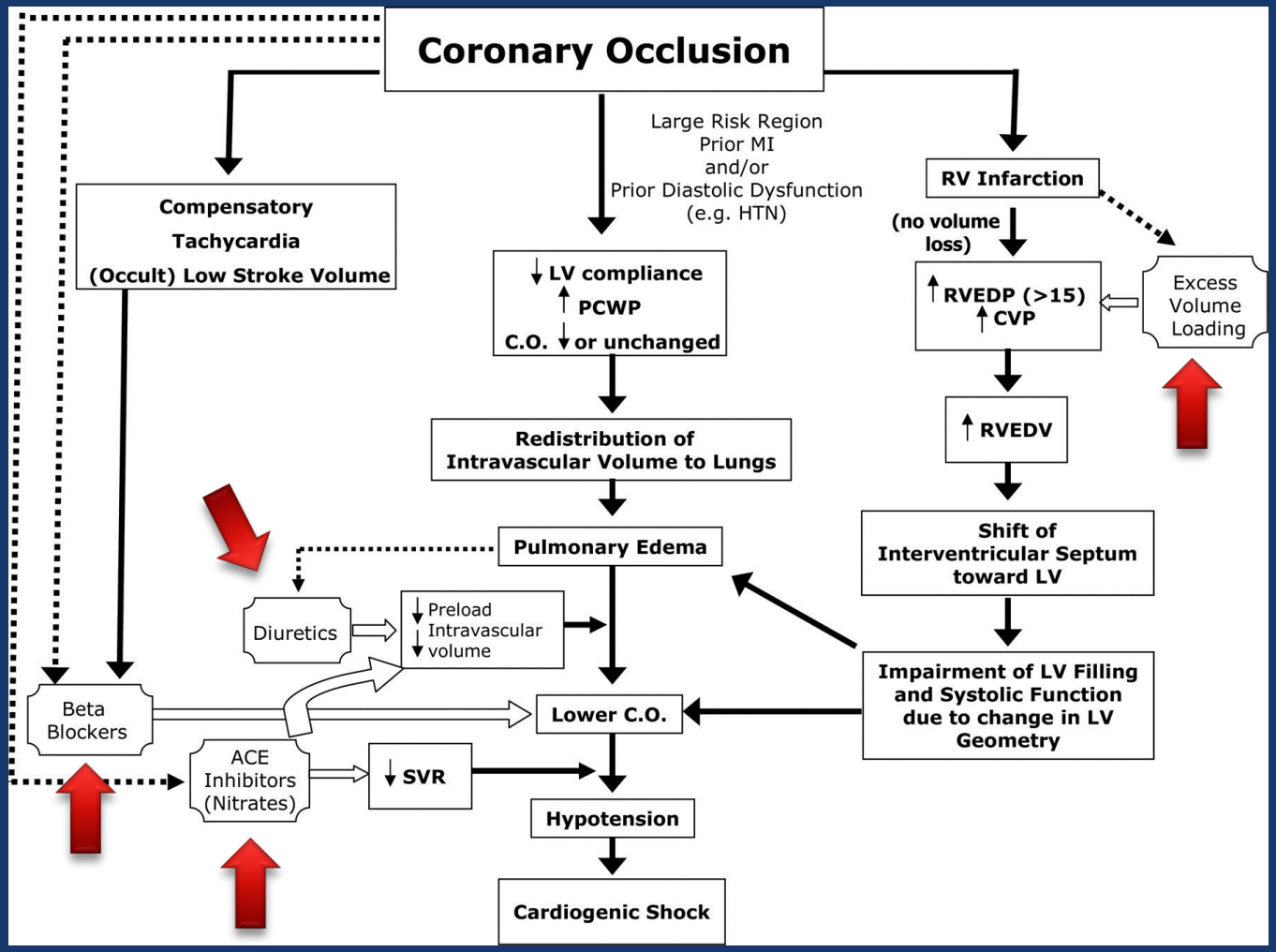


Pulmonary Artery Pulsatility Index

$$PaPi = \frac{\text{PA Systolic Pressure} - \text{PA Diastolic Pressure}}{\text{Right Atrial Pressure}}$$

Korabathina. Cath and Cardiovas Intervent 2012; 80:593-600

CS is Sometimes Iatrogenic

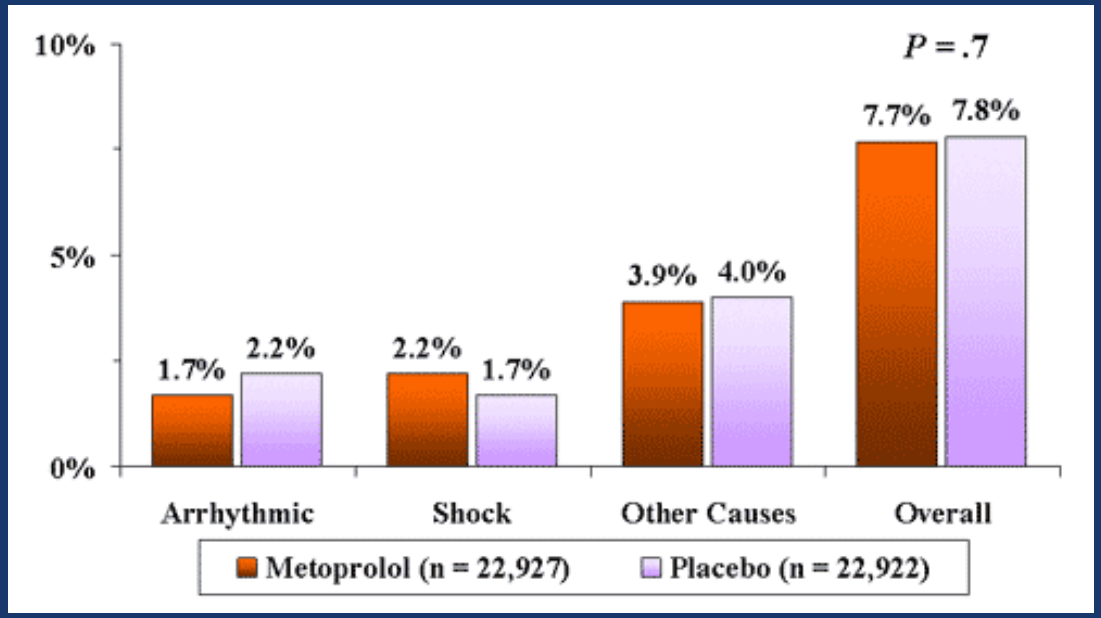


Reynolds. *Circulation* 2008; 117: 686-697

COMMIT Trial

ClOpidogrel and Metoprolol in Myocardial Infarction Trial

- **45,852 admitted to 1250 hospitals within 24 hours of AMI**
- **Randomly allocated to IV metoprolol then 200mg metoprolol versus placebo**

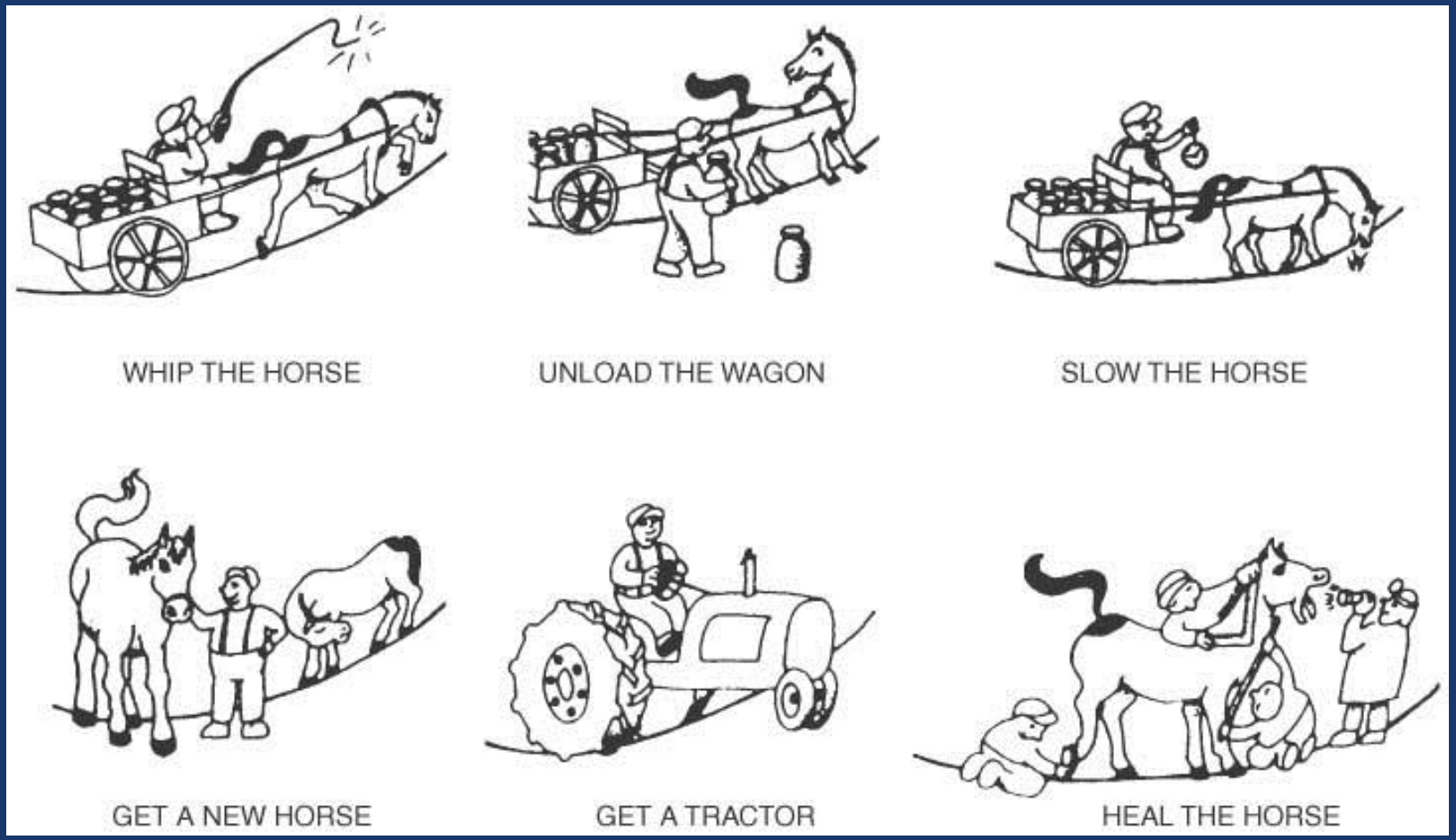


Lancet 366,9497:1622-1632

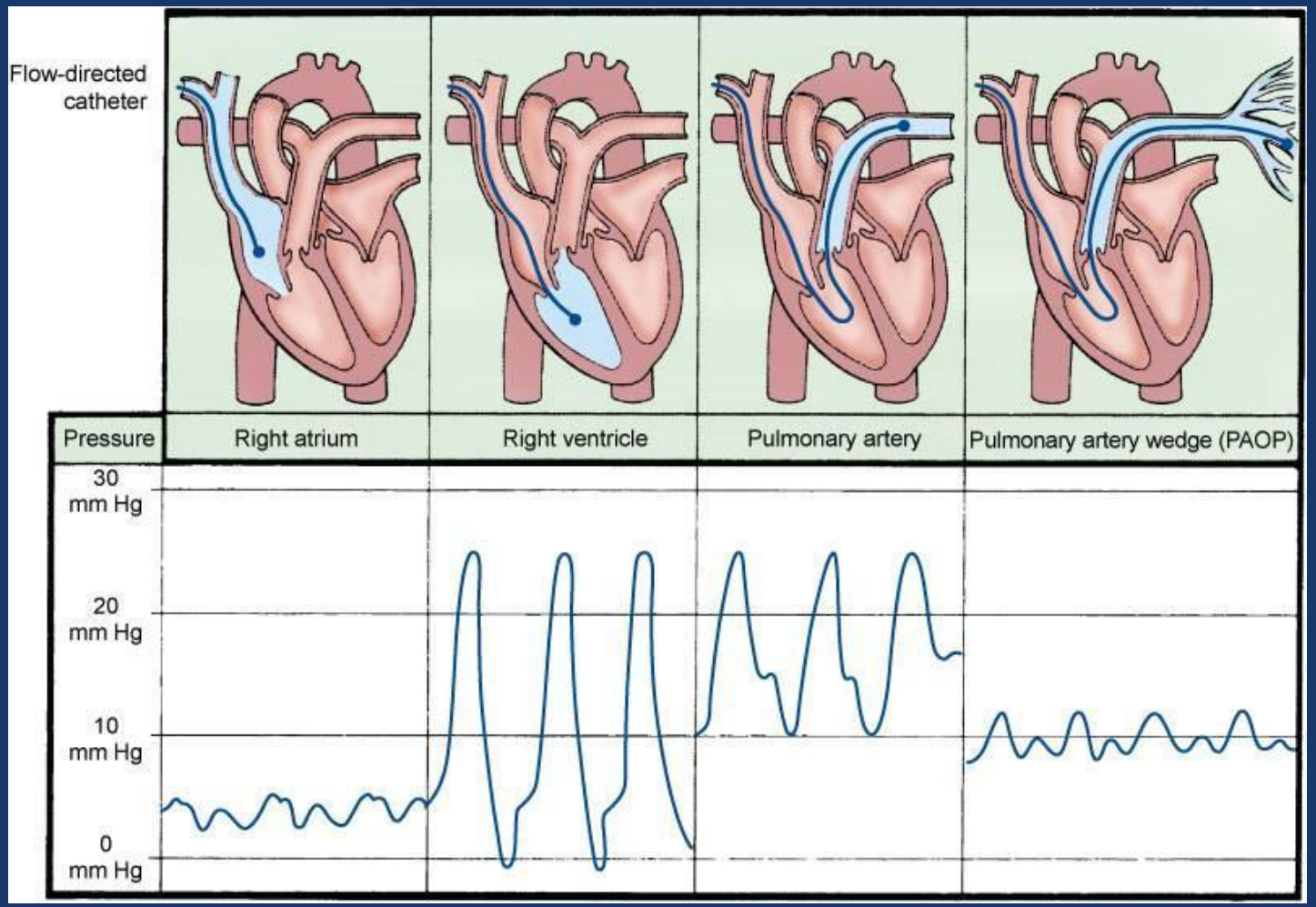
Management

1. Anti-thrombotics
2. Anti-platelets
3. Revascularization/Reperfusion
4. PA catheterization to guide therapy
5. Mechanical circulatory support

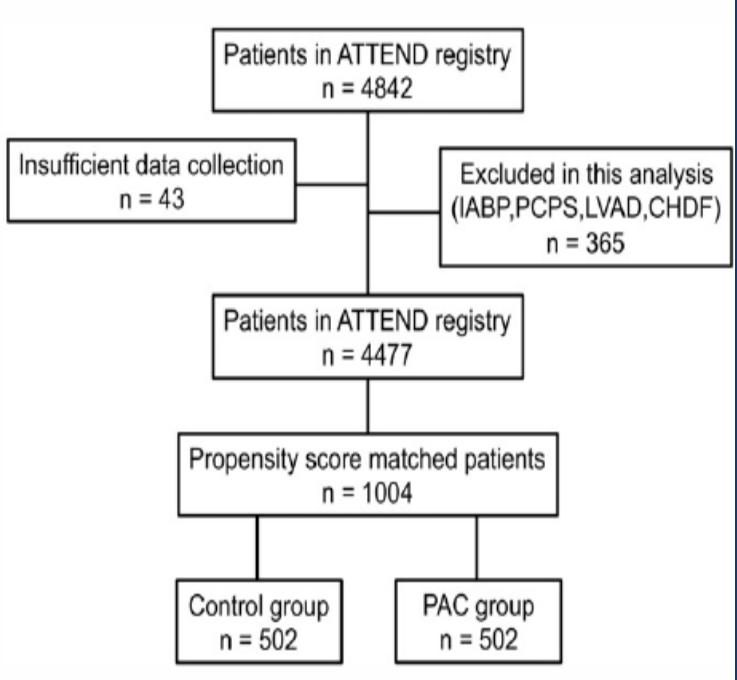
Management

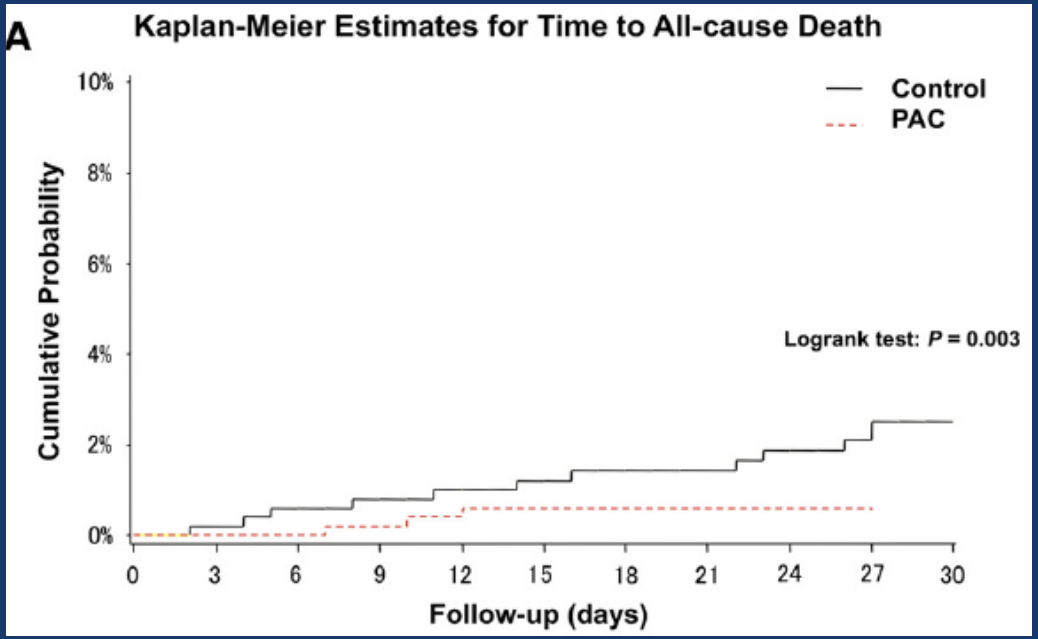

WHIP THE HORSE
UNLOAD THE WAGON
SLOW THE HORSE
GET A NEW HORSE
GET A TRACTOR
HEAL THE HORSE

Invasive Monitoring



Impact of PA catheter





Sotomi, et al. *Int Jour Card* 2016

60 yo with hx CAD c/b ICM w/ EF 35% p/w respiratory failure. SBP 90/50, HR 90. Intubated, oliguric, crackles at bases.

RA 15 mmHg
PA 37/23
PCWP 26 mmHg
C.I. 2.3
SVR 1853
SVO2 60

Medical therapy:
- Nitroprusside/afterload reduction
- IV diuresis

RA 23 mmHg
PA 28/18
PCWP 16 mmHg
C.I. 1.7
SVR 1020
SVO2 48

Consider
RV mechanical support

RA 10 mmHg
PA 37/23
PCWP 18 mmHg
C.I. 1.7
SVR 1200
SVO2 45

Consider
LV mechanical support

SHOCK Trial

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*

SHOCK Trial

Treatment	Revascularization (N=152)	Medical Therapy (N=150)
CPR, VT, or VF before randomization (%)*	32.7	23.9
Thrombolytic therapy (%)	49.3	63.3
Inotropes or vasopressors (%)	99.3	98.6
Intraaortic balloon counterpulsation (%)	86.2	86.0
Pulmonary-artery catheterization (%)	93.4	96.0
Left ventricular assist device (%)†	3.6	0.9
Heart transplantation (%)	2.0	0.7
Coronary angiography (%)	96.7	66.7
Angioplasty (%)	54.6	14.0
Stent placed‡	35.7	52.3
Platelet glycoprotein IIb/IIIa receptor antagonist§	41.7	25.0
Coronary-artery bypass grafting (%)	37.5	11.3
Angioplasty or coronary-artery bypass grafting (%)	86.8	25.3
Median time from randomization to revascularization (hr)¶	1.4 (0.6–2.8)	102.8 (79.0–162.0)

Hochman. *NEJM* 1999; 341:625-634

30-day Results

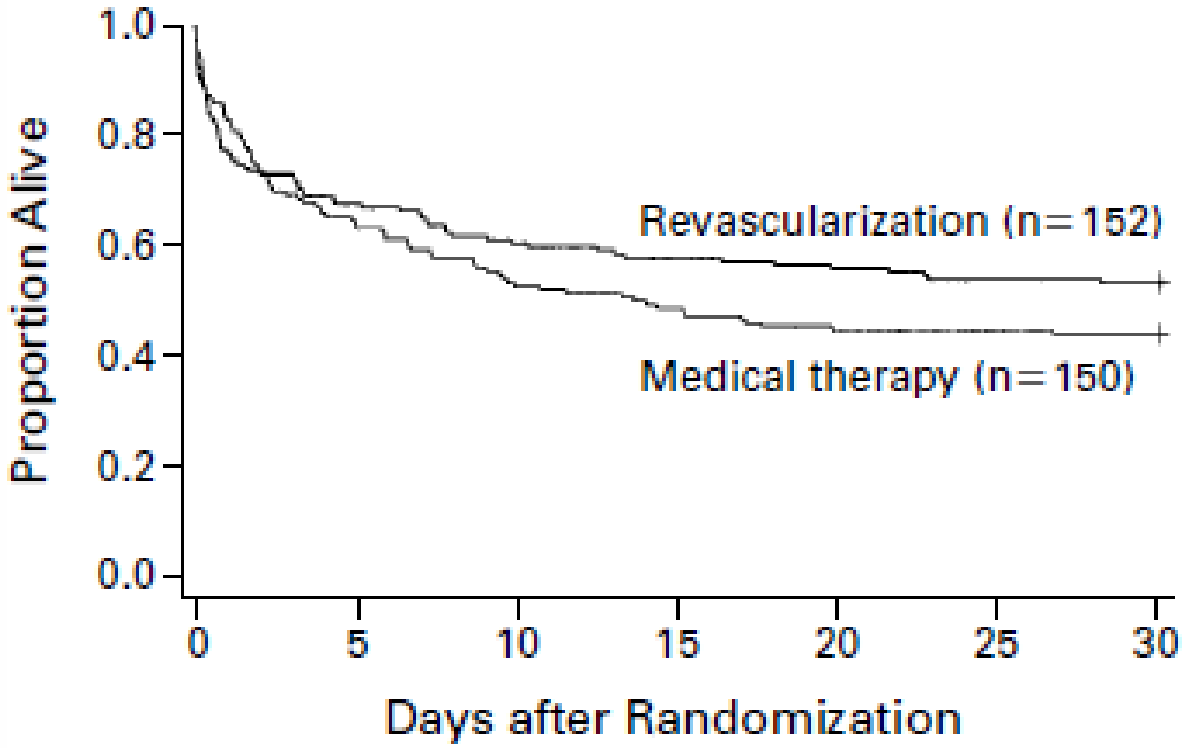
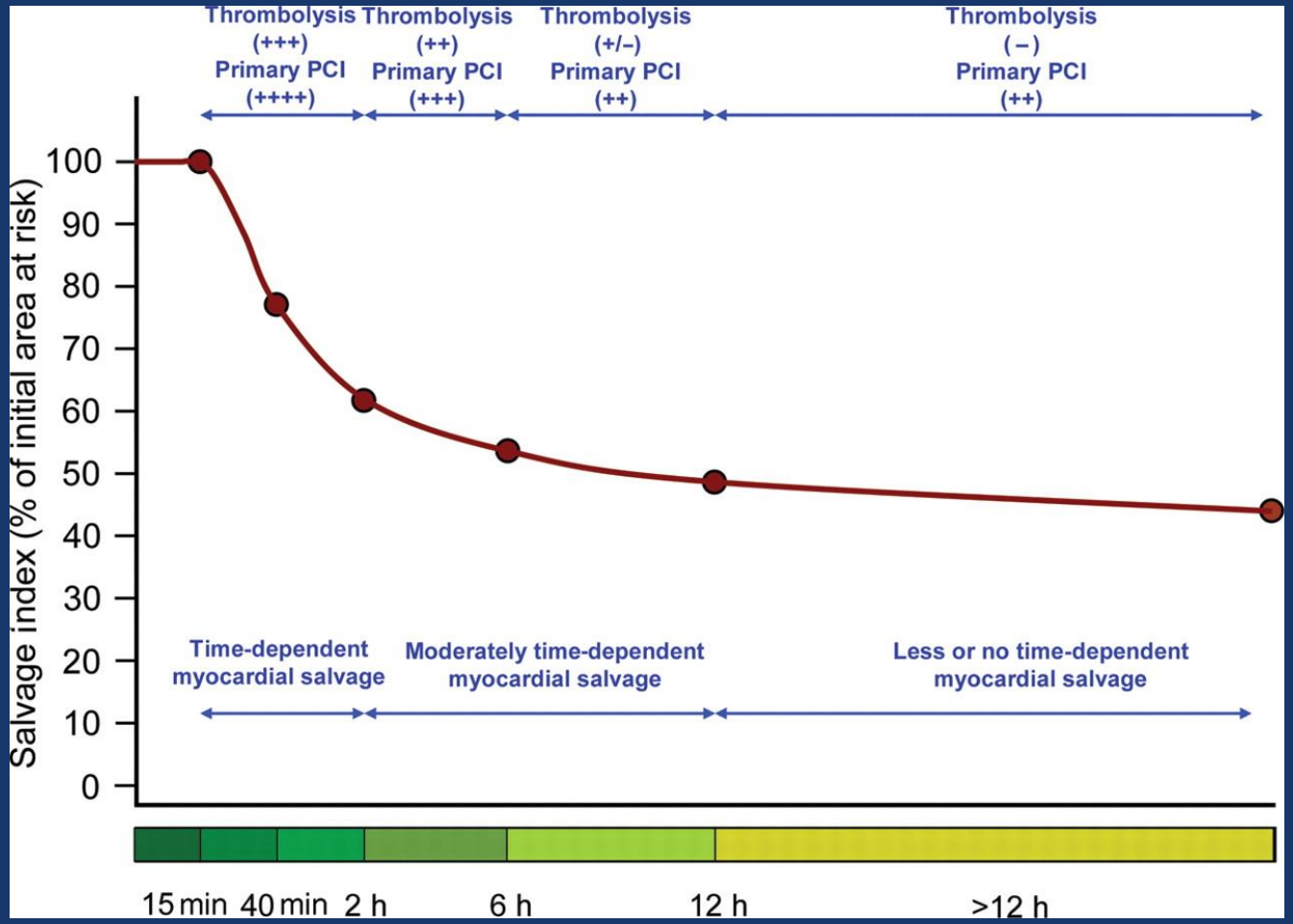


Figure 1. Overall 30-Day Survival in the Study.
The 30-day survival rate was 53.3 percent for patients assigned to revascularization and 44.0 percent for those assigned to medical therapy.

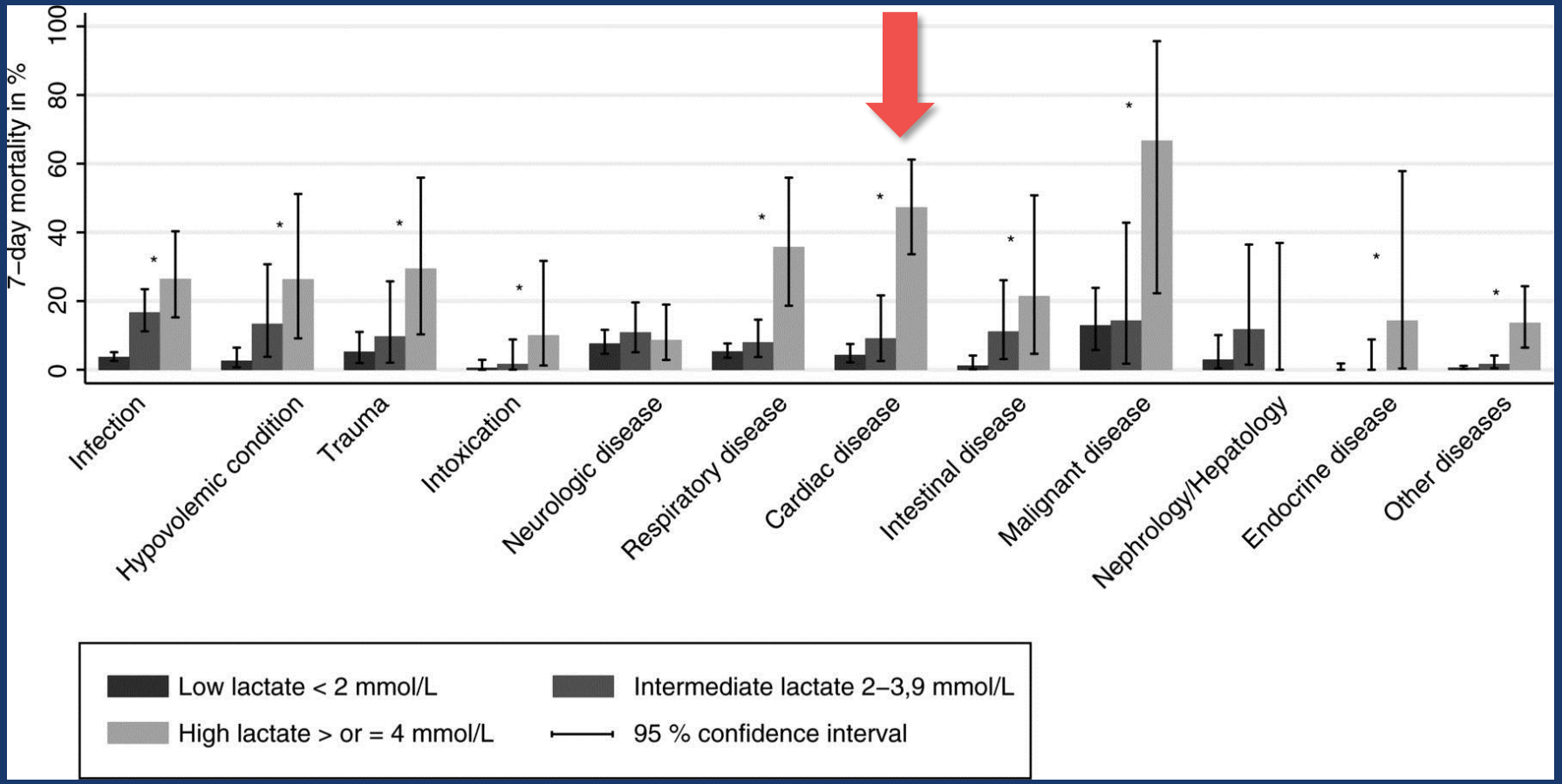
Hochman. *NEJM* 1999; 341:625-634

Time to reperfusion : Risk of Shock



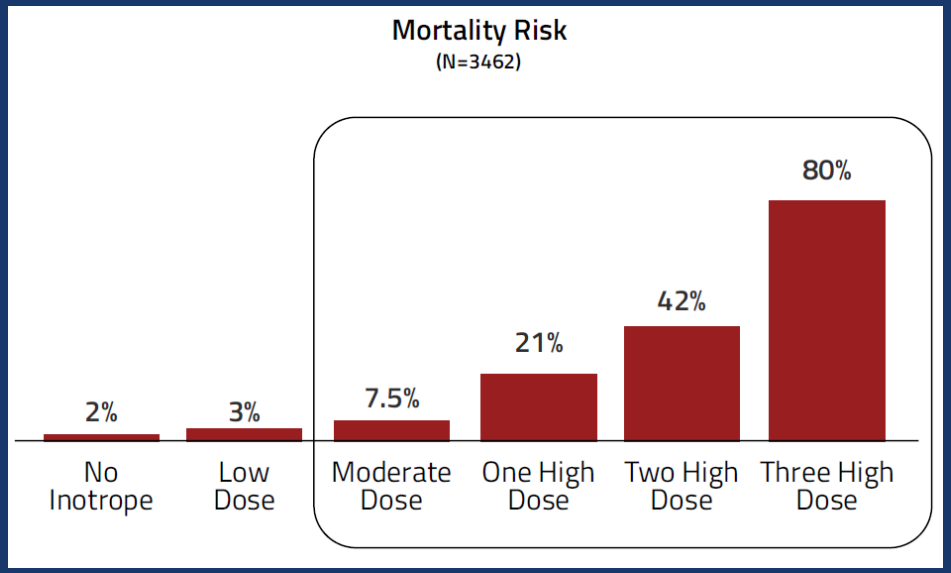
Schömig. EHJ 2006: 1900-1907

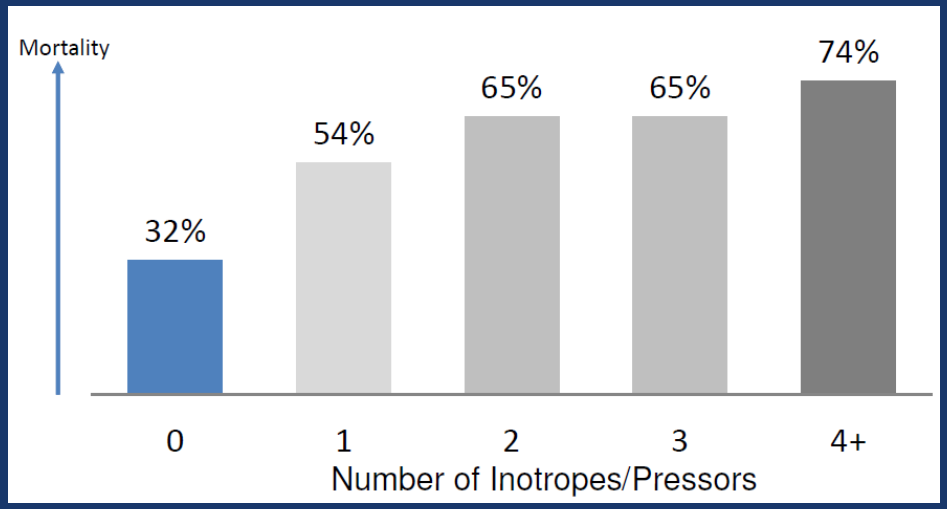
Increasing Lactate & Mortality



Pedersen M, et al. *Emerg Med J* 2015;**32:**678-684.

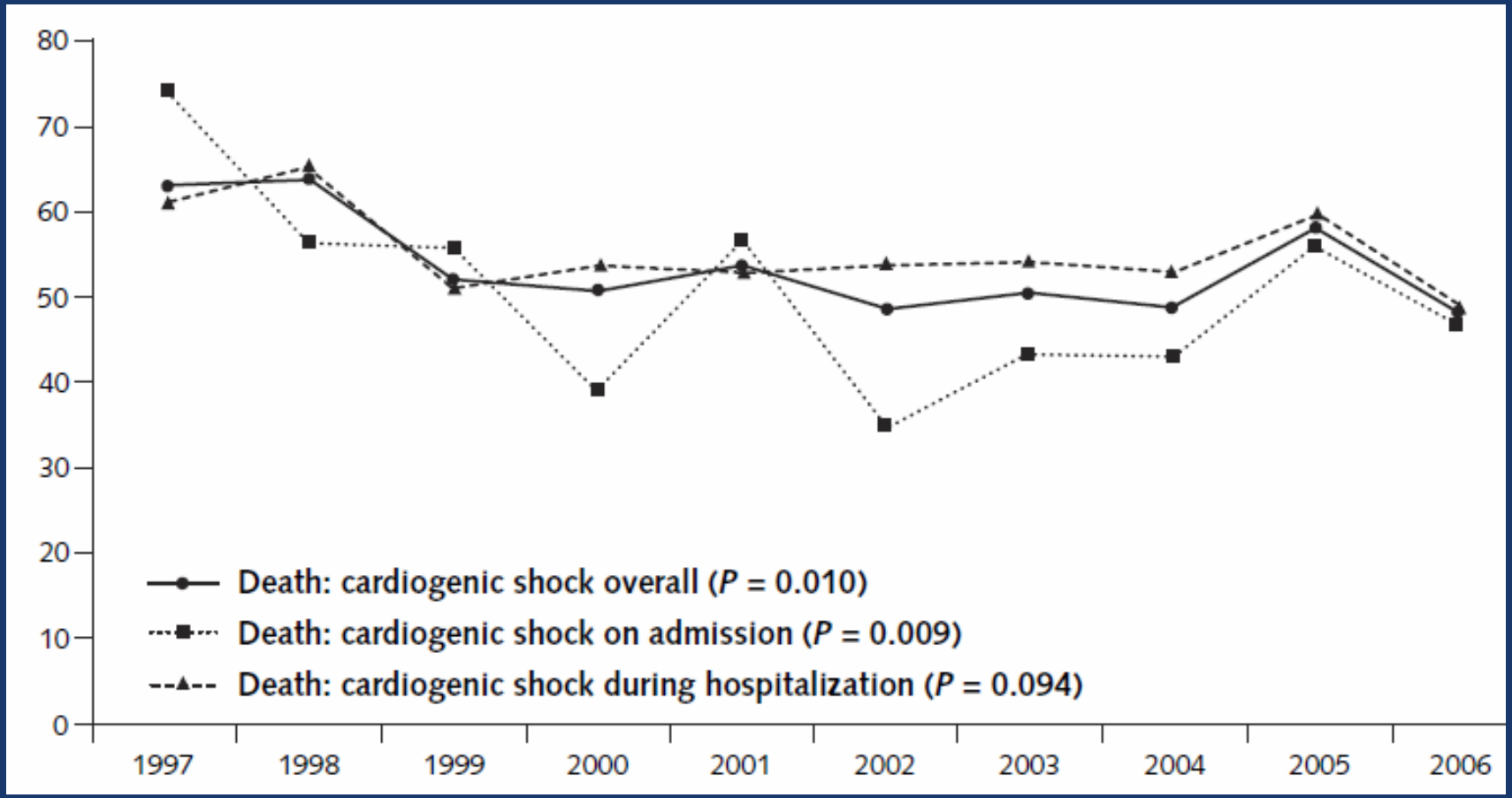
High Dose Pressors & Mortality





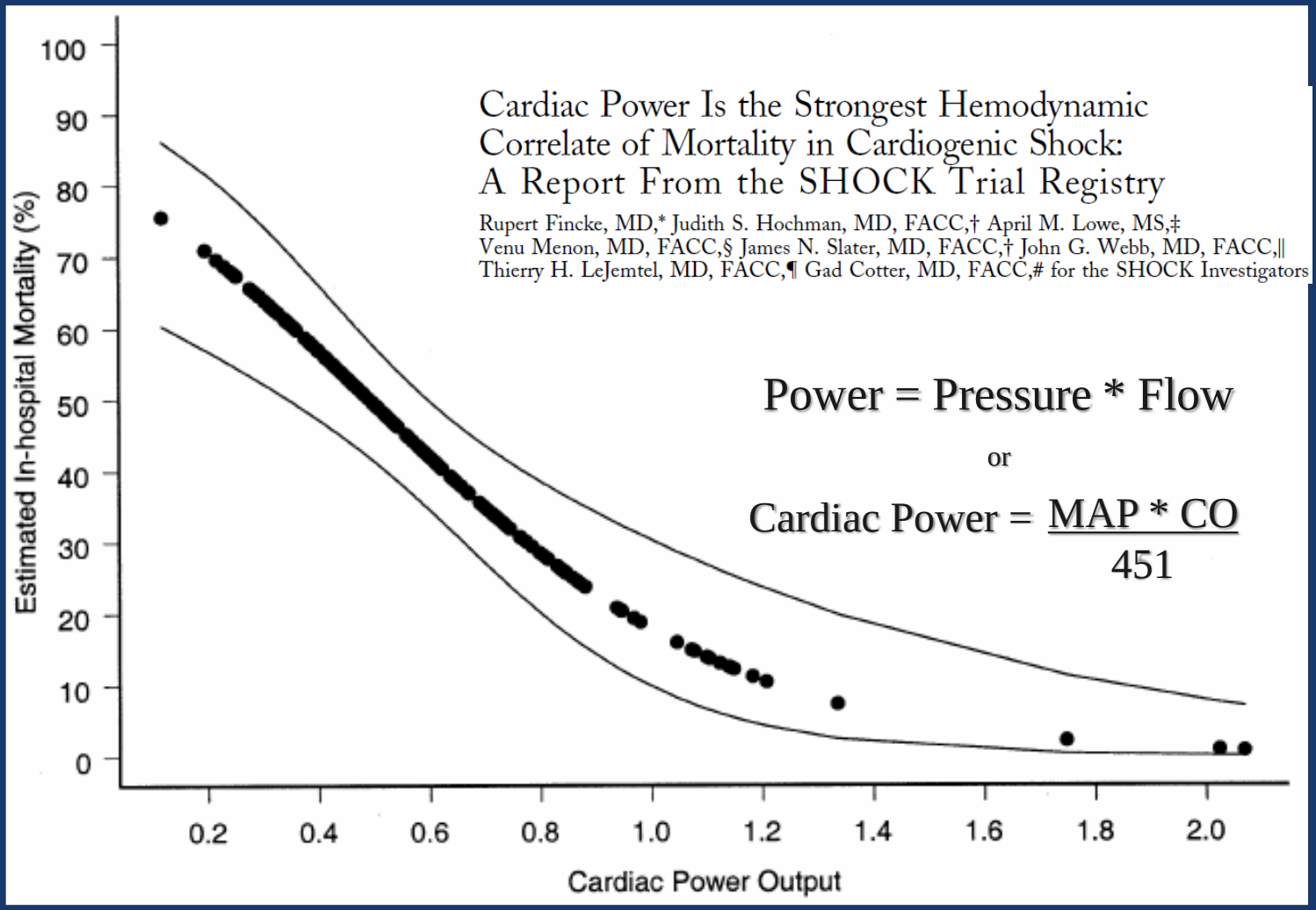
1. Samuel, et al. *J of Card Surg* 1999; 14(4):288-293
2. O'Neill, et al. *JACC Interventions* In press.

Survival in Cardiogenic Shock



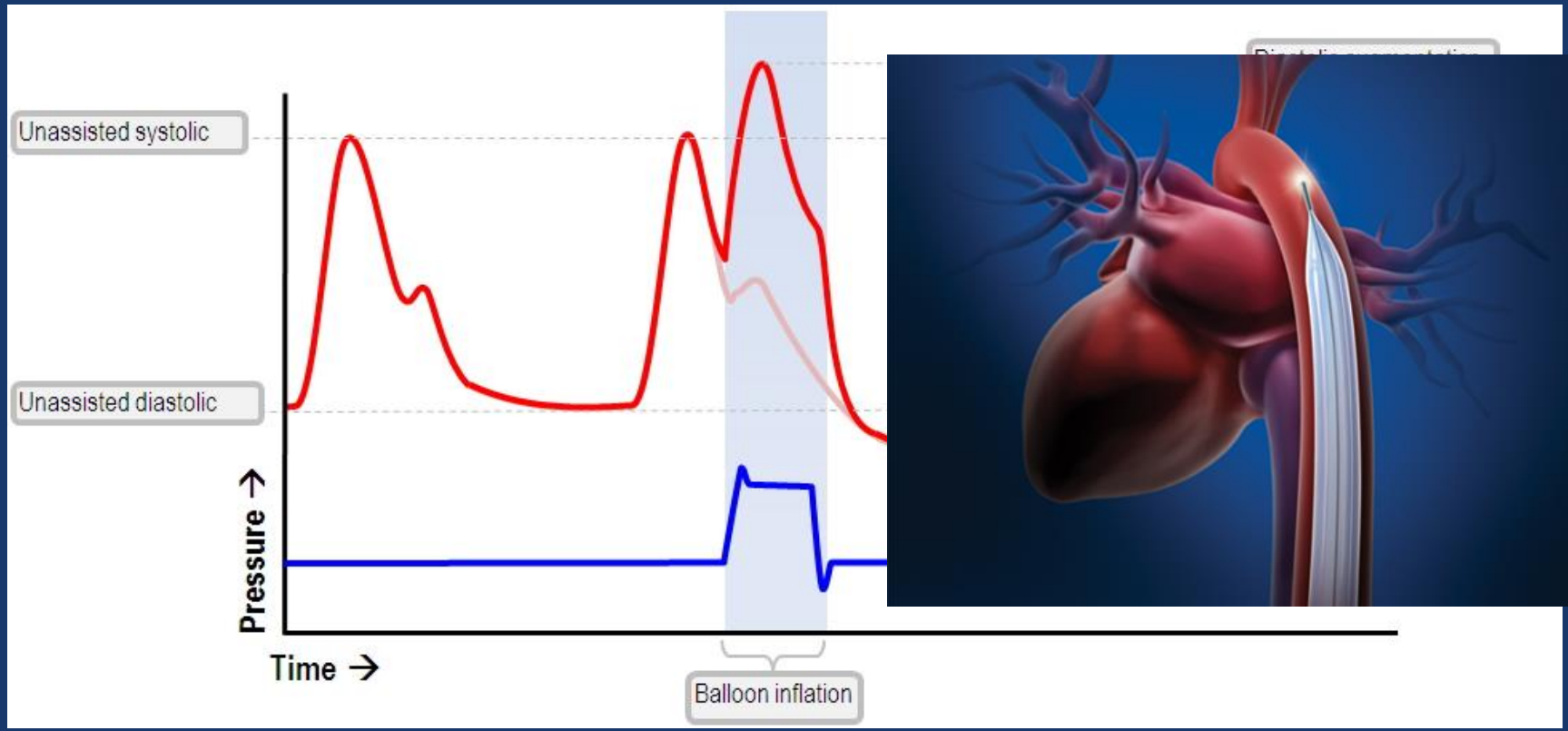
Jeger. *Ann Int Medicine* 2008;149(9):618-26

What Predicts Mortality?



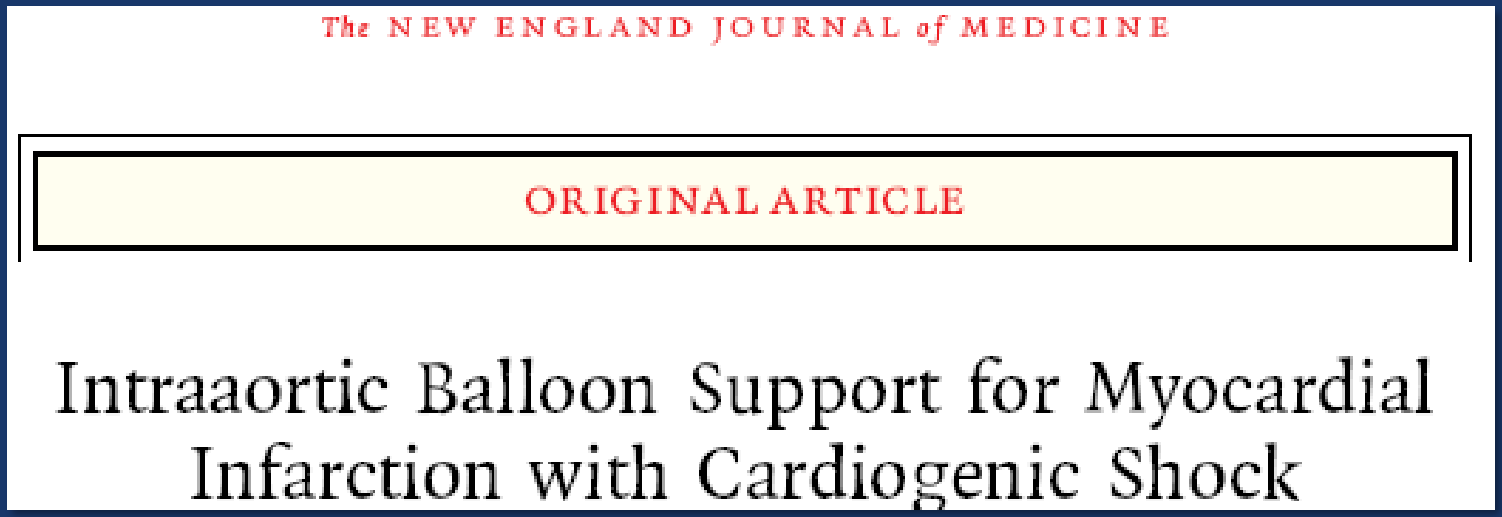
Fincke et al. *JACC* 2004

Intra-Aortic Balloon Pump



http://www.derangedphysiology.com/php/Shock-and-hemodynamic-support/IABP/Benefits-of-diastolic-augmentation.php

IABP-SHOCK II Trial



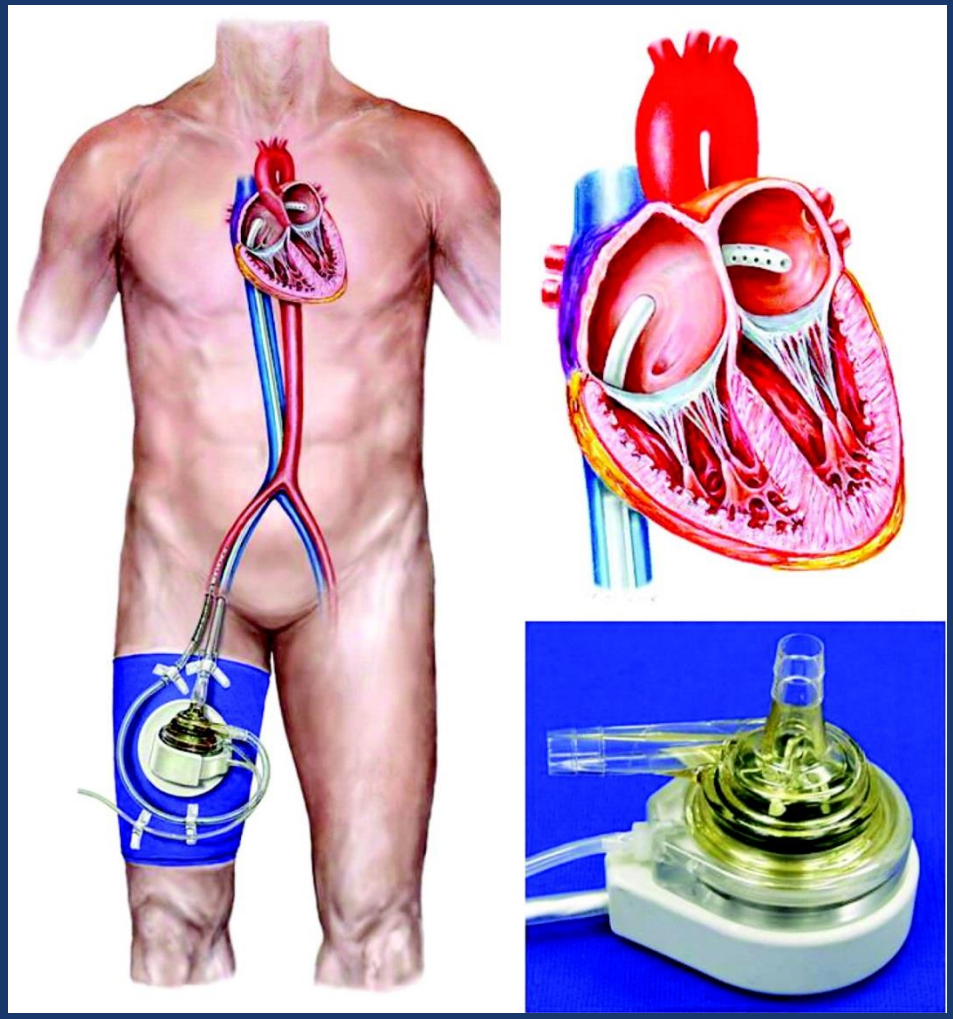
- IABP did not reduce 30-day mortality
- Post-hoc analysis showed no mortality benefit of IABP among patients with SBP < 80 mmHg

Thiele *N Engl J Med* 2012; 367:1287-1296

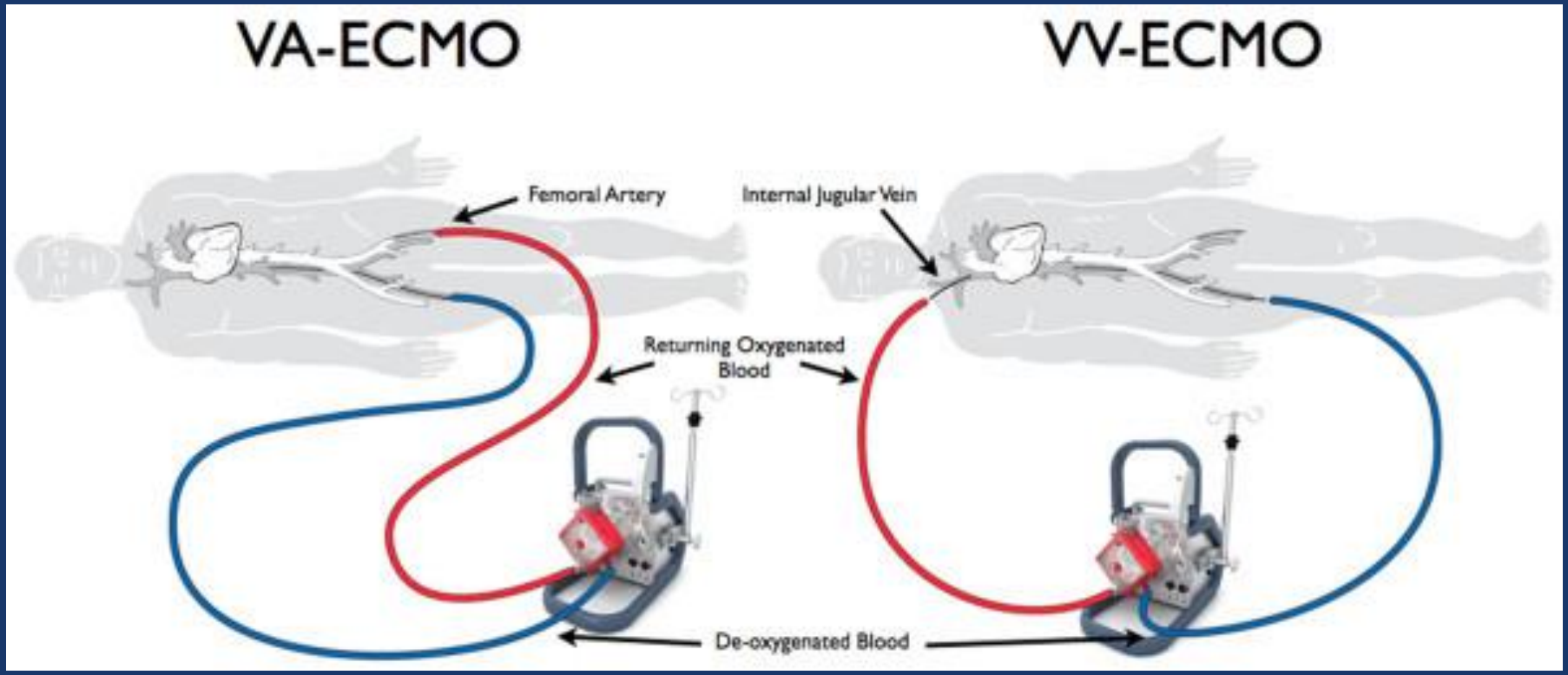
Percutaneous Ventricular Assist Devices

TandemHeart®

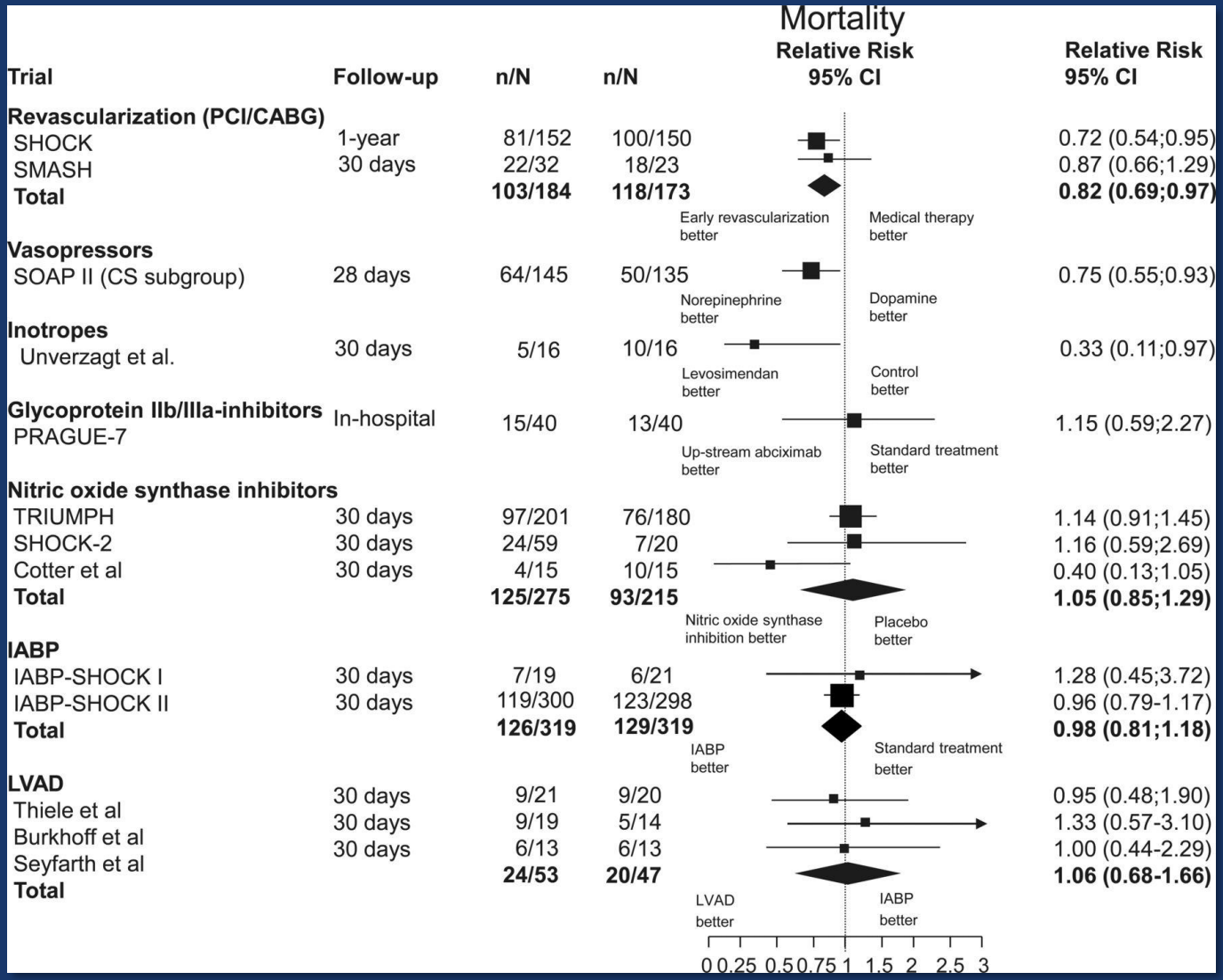
1. 21F inflow cannula in left atrium via femoral venous puncture
2. 17F arterial cannula
3. 4L/min



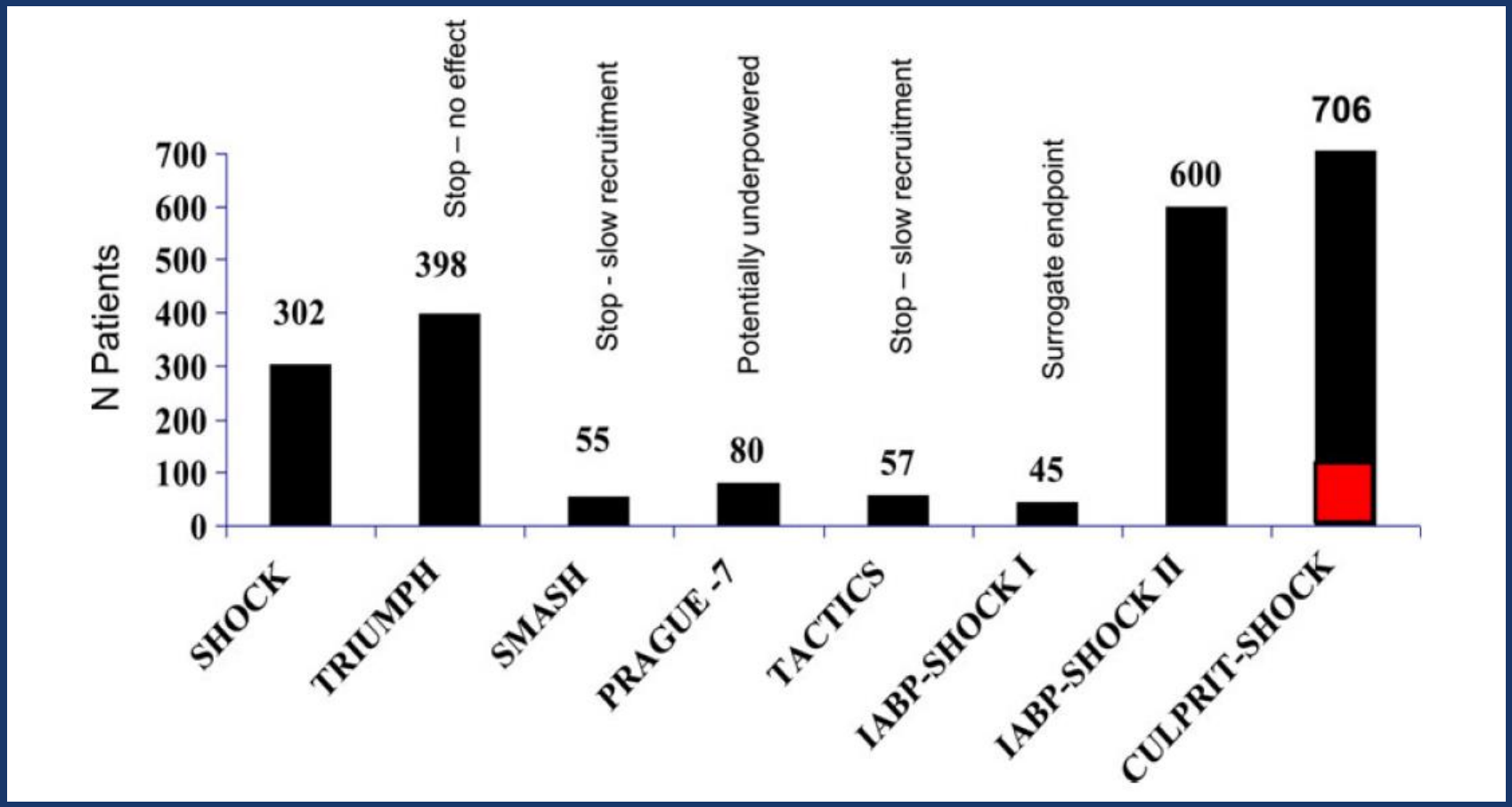
ExtraCorporeal Membrane Oxygenation



Summary of Treatment Studies

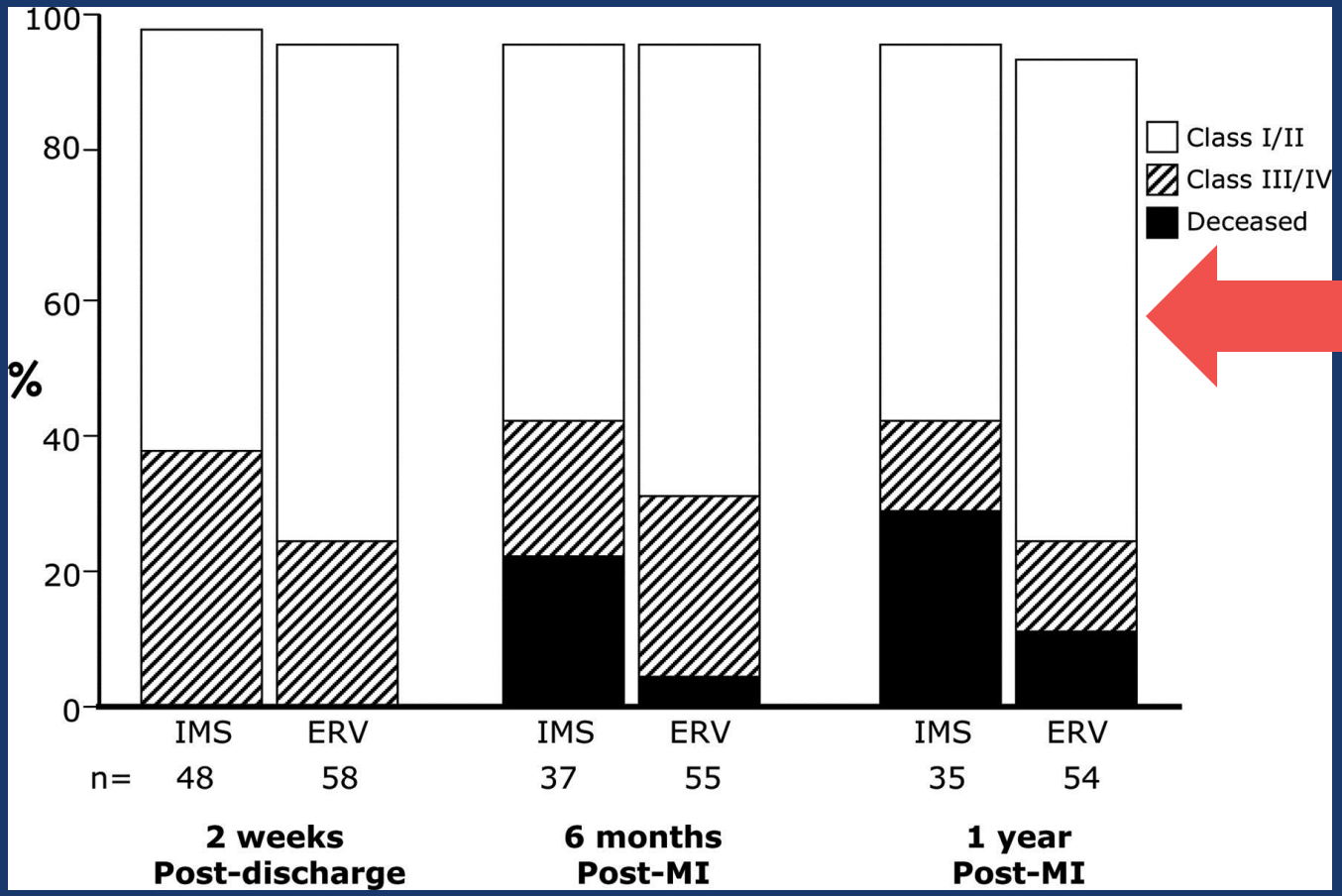


Summary of Treatment Studies



Feeling of Futility

Cardiogenic Shock Survivors



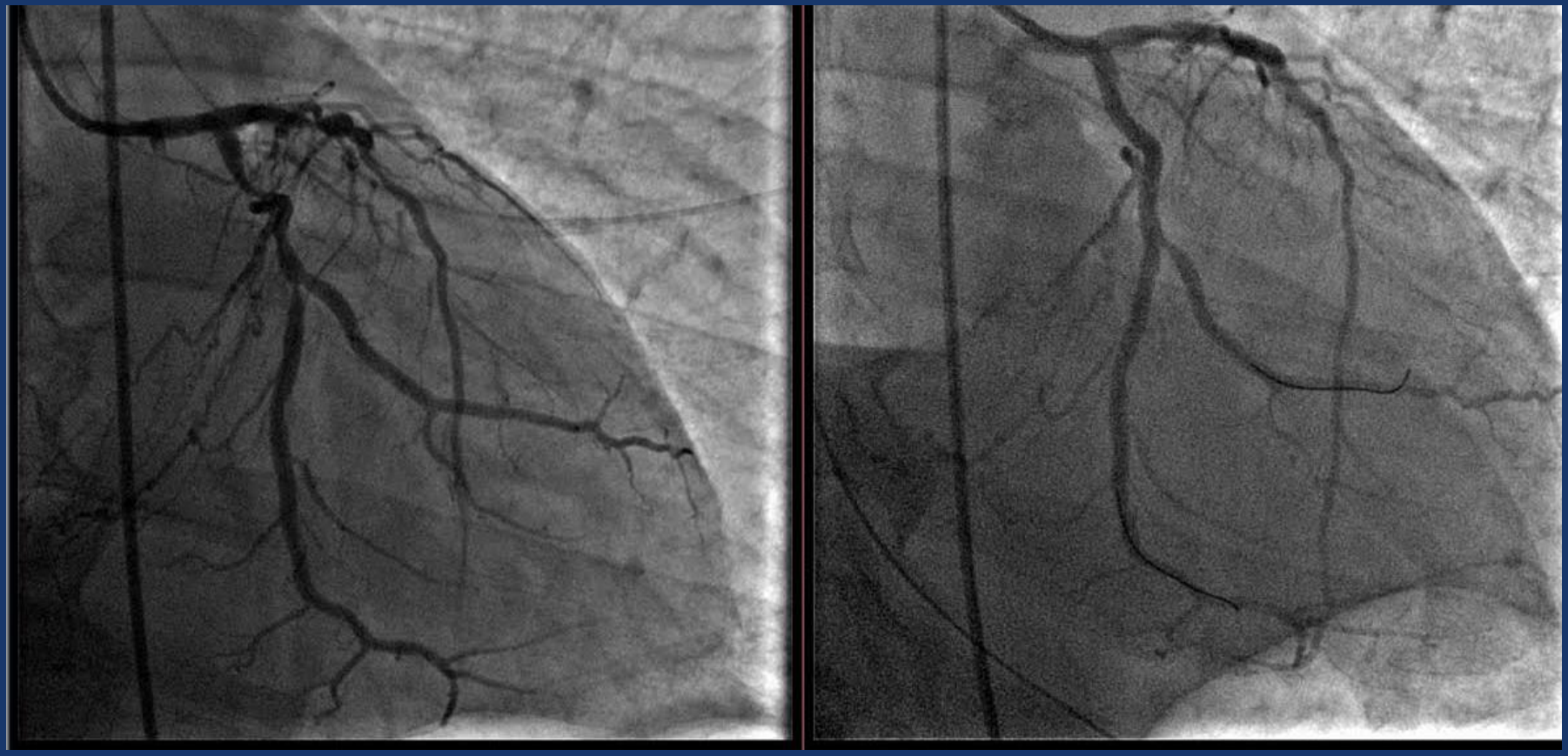
Sleeper. *J Am Coll Cardiol.* 2005; 46: 266–273

Cardiogenic Shock Survivors

Case history:

- 53 yo gentleman with no PMHx was found down by wife after searching for triathlons online
- Defibrillated in field by EMS
- Coded for 40 mins at ER
- Anterolateral STEMI on EKG – tx'd SLH
- Brought to cath lab with recurrent VT

Cardiogenic Shock Survivors



Cardiogenic Shock Survivors

Case history:

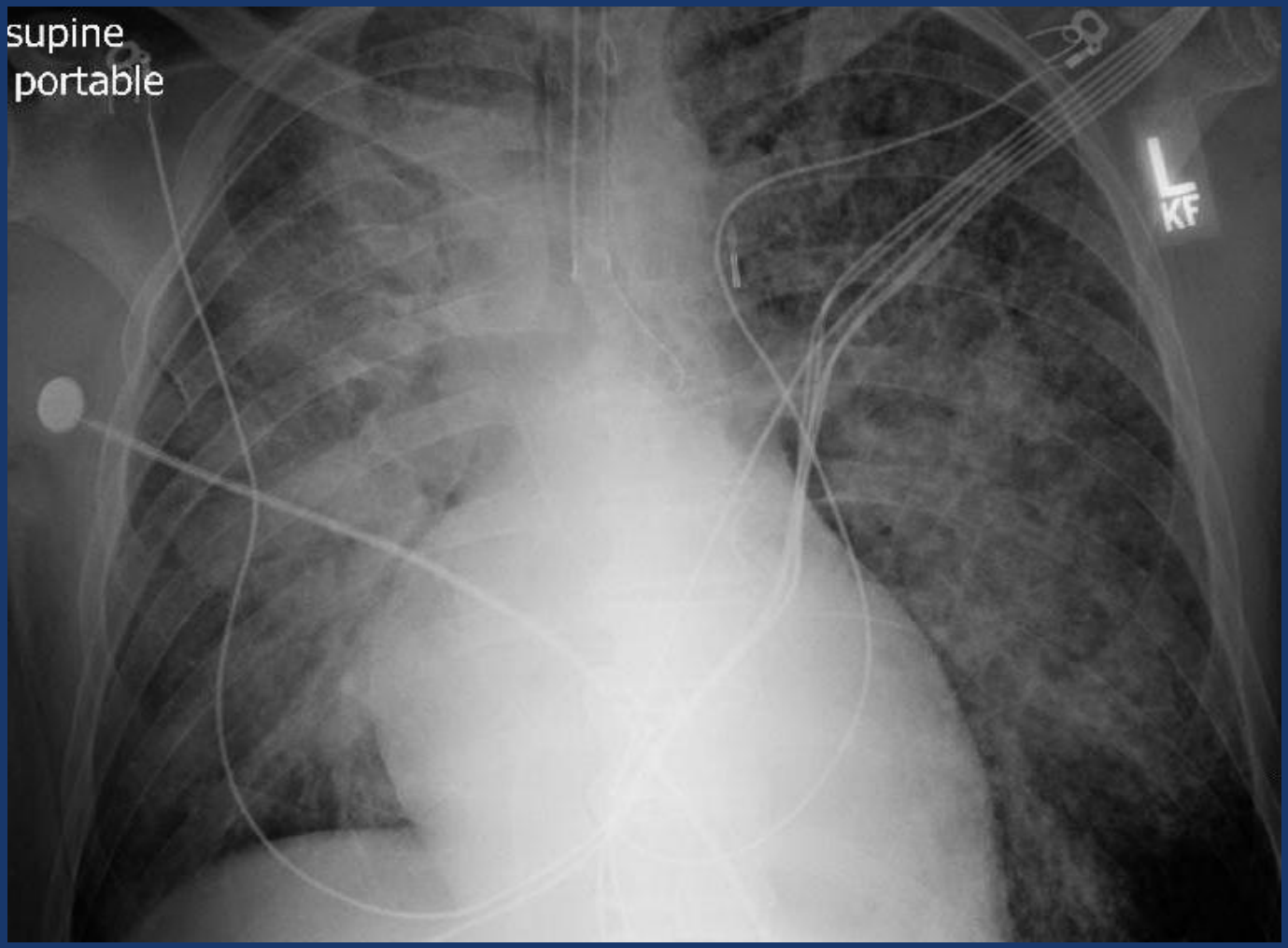
- **Shocked multiple times in lab**
- **Stents placed in LAD and LCx**
- **Glycoprotein IIb/IIIa given during procedure after fluoroscopy showed undigested ticagrelor pills in stomach**
- **Blood noted to be coming from ETT near conclusion of procedure**

Cardiogenic Shock Survivors

Case history:

- Severe hypoxia due to hemothorax – SpO2 in 30's
- Independent lung ventilation strategy pursued
- IR for bronchial artery embolization attempted
- Prolonged hospital course with acidosis, AKI, c.diff colitis, multiple transfusions, encephalopathy

Cardiogenic Shock Survivors

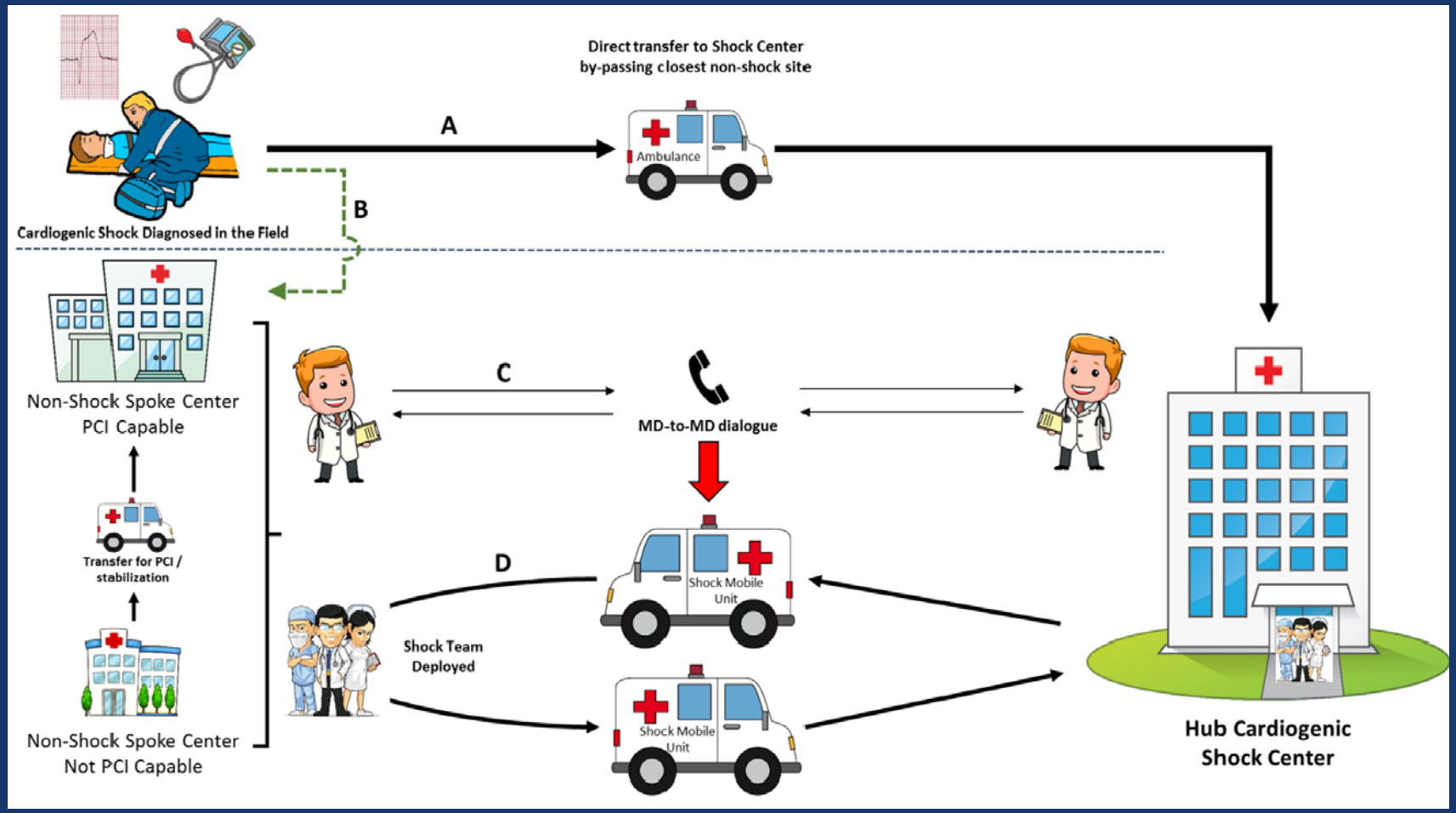


1 Year Later…

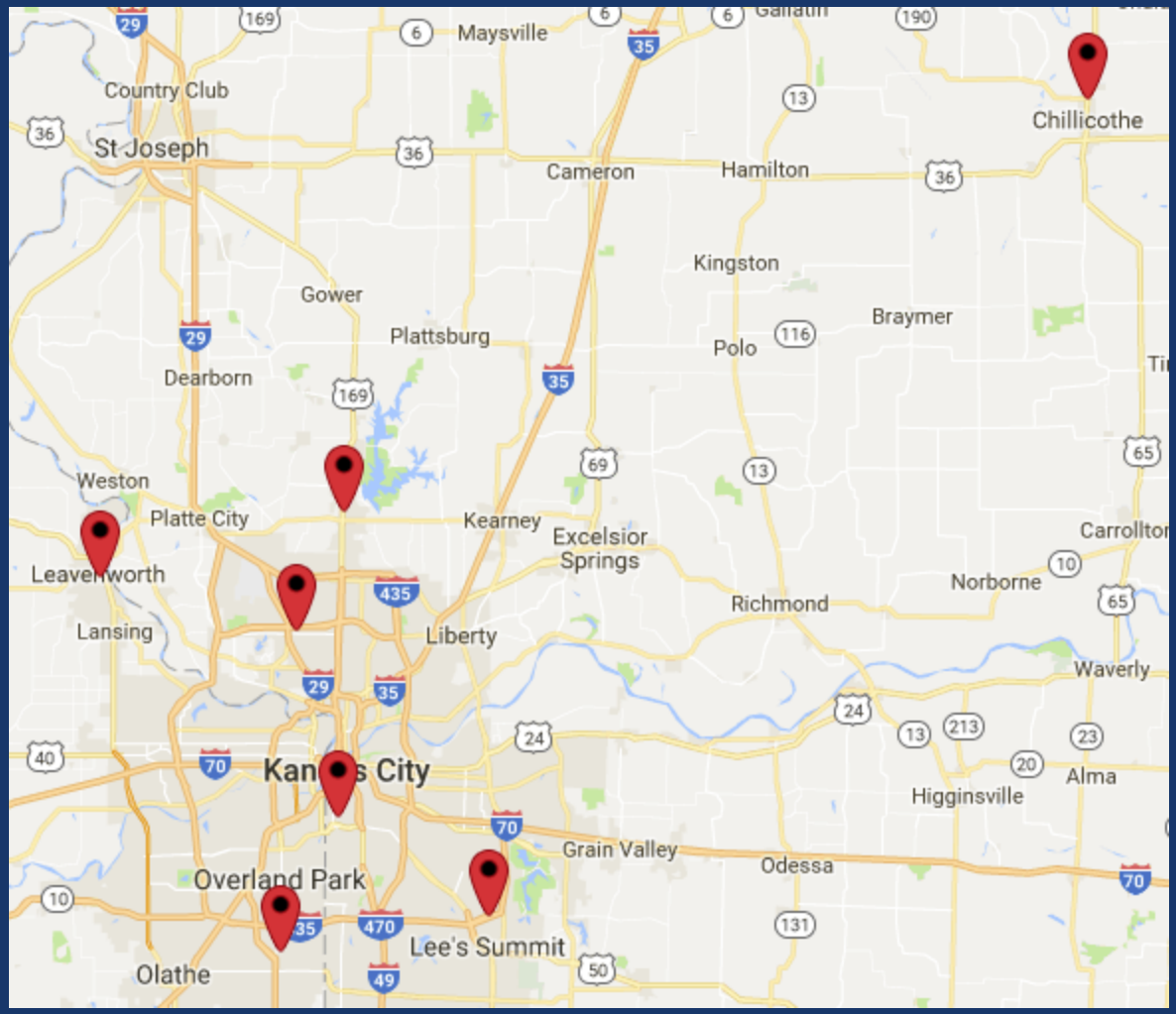
Shock Center

1. 24/7 PCI with available use of hemodynamic support
2. Available consultants
   - Advanced heart failure/transplant specialists
   - EP with expertise in complex VT ablation
   - Echocardiography 24/7
   - In-house intensivists
   - Neurology
   - Palliative care
   - CT surgery

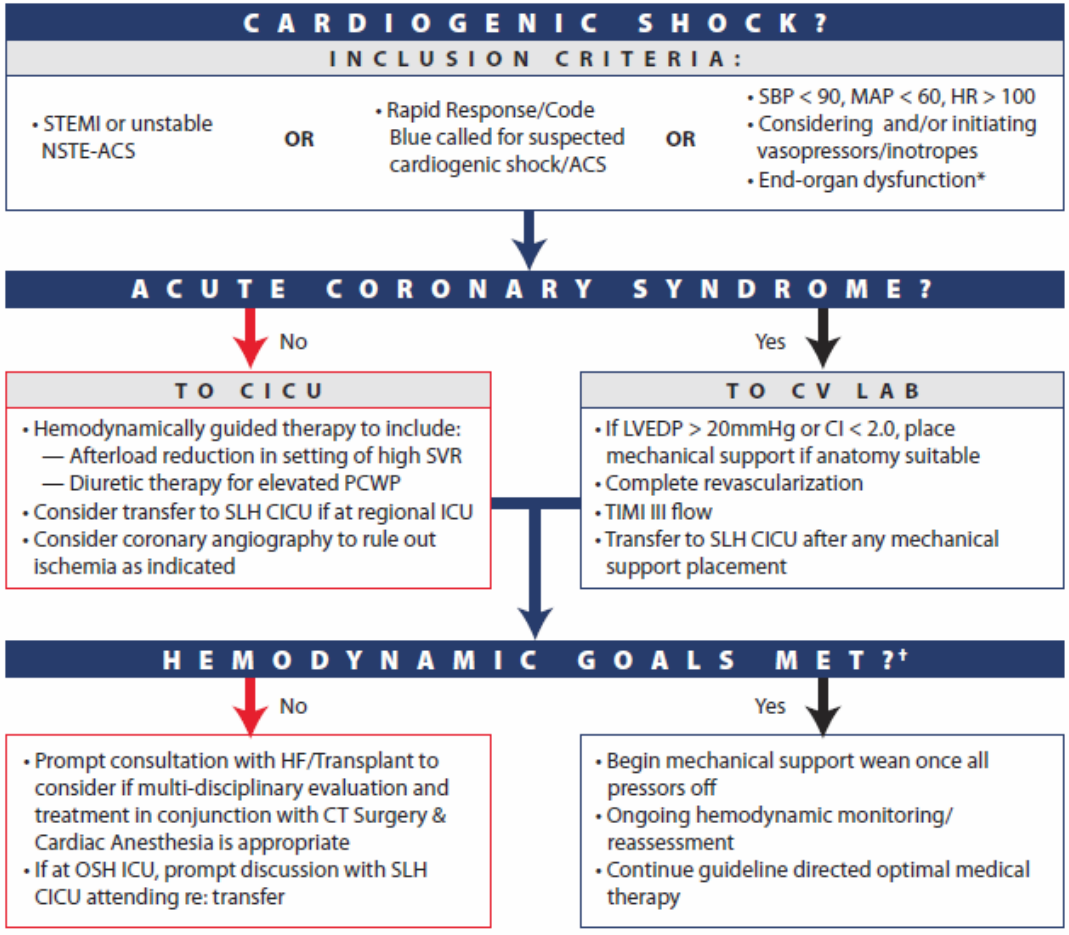
Regional System of Care



Saint Luke's Experience



Saint Luke's Mid America Heart Institute Cardiogenic Shock Protocol



*Signs of end-organ dysfunction may include the following, as acute/new findings:
1) lactate > 2
2) altered mental status
3) cold extremities/mottled skin
4) oliguria (< 30 mL/hr)
5) AST/ALT > 10x the ULN
6) troponin > 1

Best Practices
- Arterial line monitoring
- PA catheter placement
- Bedside echocardiogram
- Immediate CO/CI/SvO2/lactate upon arrival in CICU and q2 hours thereafter
- Prompt critical care consultation

†Hemodynamic Goals:
- CPO = (MAP*CO)/451 > 0.6 W
- PAPi = (sPAP-dPAP)/RA > 1.85
- Cardiac Index > 2
- SBP > 90 or MAP > 55
- SVO2 > 60
- Decreased lactate
- Able to wean off pressors
- No ventricular arrhythmias
- Improving end-organ dysfunction

Saint Luke's Mid America Heart Institute Cardiogenic Shock Protocol

CARDIOGENIC SHOCK?

INCLUSION CRITERIA:

- STEMI or unstable NSTE-ACS

OR

- Rapid Response/Code Blue called for suspected cardiogenic shock/ACS

OR

- SBP < 90, MAP < 60, HR > 100
- Considering and/or initiating vasopressors/inotropes
- End-organ dysfunction*

ACUTE CORONARY SYNDROME?

No

TO CICU

- Hemodynamically guided therapy to include:
  - — Afterload reduction in setting of high SVR
  - — Diuretic therapy for elevated PCWP
- Consider transfer to SLH CICU if at regional ICU
- Consider coronary angiography to rule out ischemia as indicated

Yes

TO CV LAB

- If LVEDP > 20mmHg or CI < 2.0, place mechanical support if anatomy suitable
- Complete revascularization
- TIMI III flow
- Transfer to SLH CICU after any mechanical support placement

HEMODYNAMIC GOALS MET?†

No

- Prompt consultation with HF/Transplant to consider if multi-disciplinary evaluation and treatment in conjunction with CT Surgery & Cardiac Anesthesia is appropriate
- If at OSH ICU, prompt discussion with SLH CICU attending re: transfer

Yes

- Begin mechanical support wean once all pressors off
- Ongoing hemodynamic monitoring/ reassessment
- Continue guideline directed optimal medical therapy

*Signs of end-organ dysfunction may include the following, as acute/new findings:

1) lactate > 2
2) altered mental status
3) cold extremities/mottled skin
4) oliguria (< 30 mL/hr)
5) AST/ALT > 10x the ULN
6) troponin > 1

Best Practices

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“A momentary pause in death”

“There is a golden hour between life and death. If you are critically injured you have less than 60 minutes to survive. You might not die right then; it may be 3 days or 2 weeks later but something has happened to your body that is irreparable.”

Hemodynamic Problem

Hemo-Metabolic Problem

Adapted from Kapur, N.
Quote from R Adams Cowley

Conclusions

1. Cardiogenic shock carries great risk of mortality, but great potential for recovery
2. Reversible causes must considered and ruled out
3. Pathophysiology is very complex with several potential iatrogenic causes.
4. Revascularization remains the cornerstone of therapy
5. Advanced circulatory support and transfer to tertiary referral center should be considered in patients not responsive to revascularization.

Thank You