Cardiogenic shock - Diagnosis & Management

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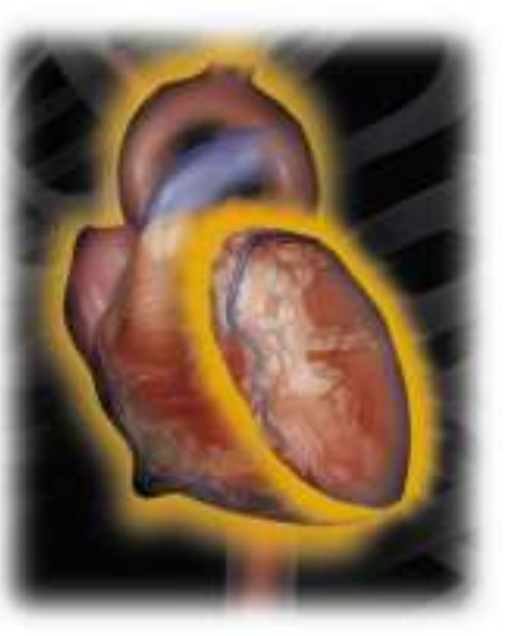
DM Cardiology Std



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Introduction

- Cardiogenic shock (CS) occurs in 5% to 8% of patients hospitalized with ST-elevation myocardial infarction (STEMI).
- Recent research has suggested that the peripheral vasculature and neurohormonal and cytokine systems play a role in the pathogenesis and persistence of CS.
- Early revascularization for CS improves survival substantially.
- New mechanical approaches to treatment are available, and clinical trials are feasible even in this high-risk population.
- Most importantly, hospital survivors have an excellent chance for long-term survival with good quality of life.

J Am Coll Cardiol. 1994;23:1630 –1637.

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Aims of my seminar

This review will outline –

- Definition of cardiogenic shock
- The causes of cardiogenic shock
- Pathophysiology of cardiogenic shock
- Diagnosis of cardiogenic shock
- Treatment of CS with a focus on CS complicating myocardial infarction (MI.)

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Definition

- CS is a state of end-organ hypoperfusion due to cardiac failure.
- The definition of CS includes hemodynamic parameters:

➢ Persistent hypotension (systolic blood pressure < 80 to 90 mm Hg or mean arterial pressure 30 mm Hg lower than baseline) with

➢ Severe reduction in cardiac index (<1.8 L · min1 · m2 without support or <2.0 to 2.2 L · min1 · m2 with support) and

➢ Adequate or elevated filling pressure (eg, left ventricular [LV] end-diastolic pressure >18 mm Hg or right ventricular [RV] end-diastolic pressure >10 to 15 mm Hg).

J Am Coll Cardiol. 1994;23:1630–1637.

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Causes of cardiogenic shock - LV failure

- Systolic dysfunction

  - CAD : acute MI or ischemia(most common cause)

  - other conditions : severe myocarditis, end stage cardiomyopathy (including valvular cause), myocardial contusion

  Prolonged cardiopulmonary bypass, global hypoxemia, myocardial depression(betablocker, calcium blocker, antiarrhythmic drug), respiratory acidosis,

  Metabolic derangement eg.acidosis, hypoPO4, hypo Ca

Tachycardia related cardiomyopathy

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Heart. 2007;93:177–182

Causes of cardiogenic shock -LV failure

- Diastolic dysfunction

  - CAD

  - ventricular hypertrophy,
- Restrictive cardiomyopathy
- Consequent of prolonged hypovolemia or septic shock
- External compression by pericardial tamponade

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Heart. 2007;93:177–182

Causes of cardiogenic shock -LV failure

- Greatly increased afterload
  - AS
  - HOCM: dynamic ventricular outflow obstruction
  - Coarctation of aorta
  - Malignant HT

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Heart. 2007;93:177–182

Causes of cardiogenic shock -LV failure

- Valvular or structural abnormality

  - MS

  - endocarditis

  - MR, AR

  - obstruction due to atrial myxoma or thrombus

  - MI complication : papillary muscle dysfunction or ruptured with severe MR(1%),

    : ruptured LV free wall

    (0.8-6.2%)

    : ventricular septal rupture(1-3%)

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Heart. 2007;93:177–182

Causes of cardiogenic shock -LV failure

- Arrhythmia
  - VT/VF, bradycardia can cause shock
  - sinus tachycardia or atrial tachycardia can aggravate shock

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Heart. 2007;93:177–182

Causes of cardiogenic shock -RV failure

- Greatly increased after load
  - pulmonary emboli
  - pulmonary vascular disease eg. PAH, venoocclusive disease
  - hypoxic pulmonary vasoconstriction
  - PEEP
  - high alveolar pressure
  - ARDS
  - pulmonary fibrosis
  - sleep disorder breathing
  - COPD
- RV infarction : 50% of inferior MI(10-15% with hemodynamic problem)

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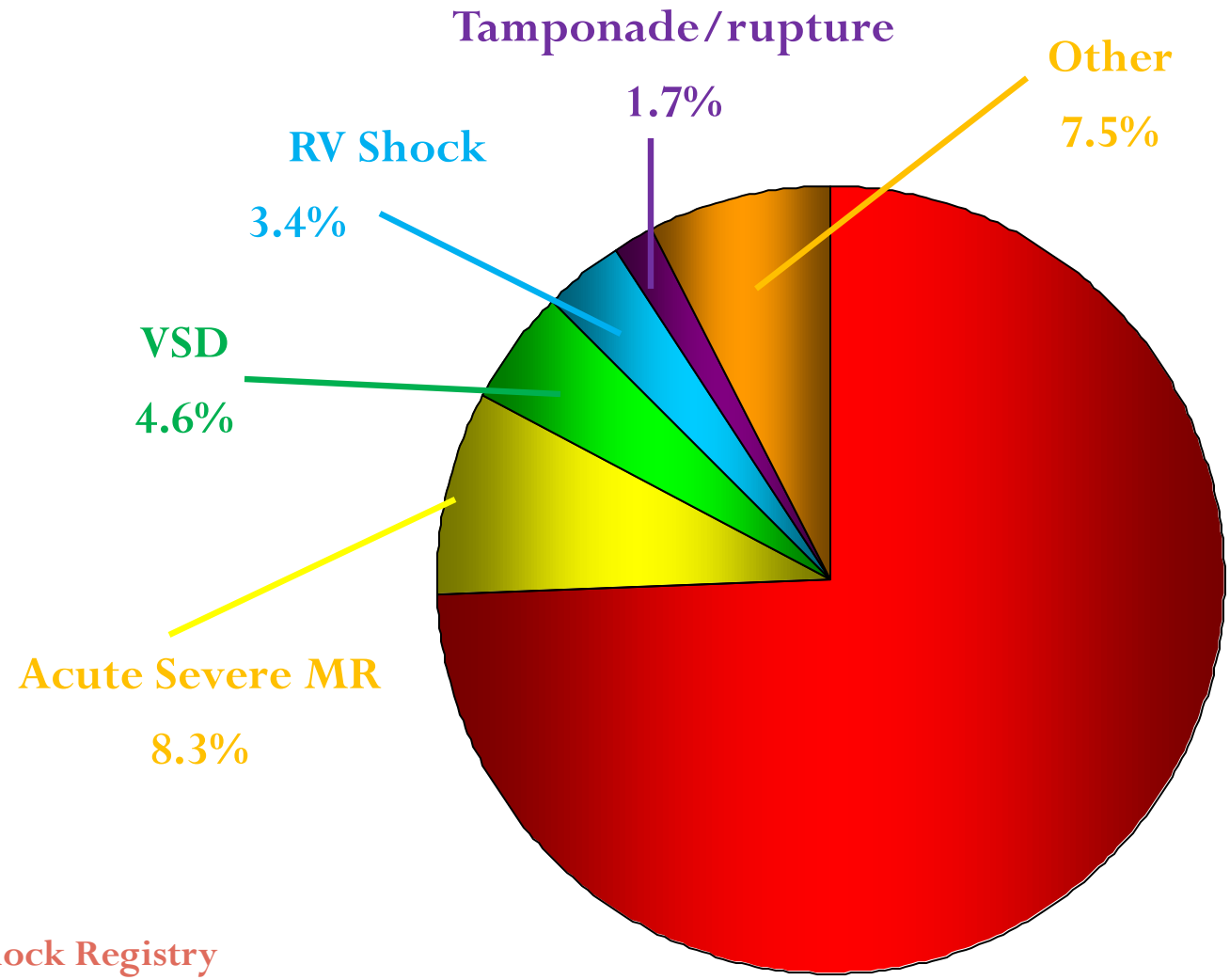
Heart. 2007;93:177–182

Causes of Cardiogenic Shock



Shock Registry

JACC 2000 35:1063

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INCIDENCE

- After decades of remarkable stability in the incidence of CS, it appears that the incidence is on the decline in parallel with increasing rates of use of primary PCI for acute MI.
- CS continues to complicate approximately 5% to 8% of STEMI and 2.5% of non-STEMI cases.
- The routine use of troponin to define non-STEMI will result in a drop in this percentage as more MIs are detected but will not alter the total number of cases of CS.

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Hasdai et al. JACC 2000;36:687

- Mortality of such patients approximately 80% or higher
- Very few patients develop shock immediately after AMI
- About half of the patients develop shock within 24h

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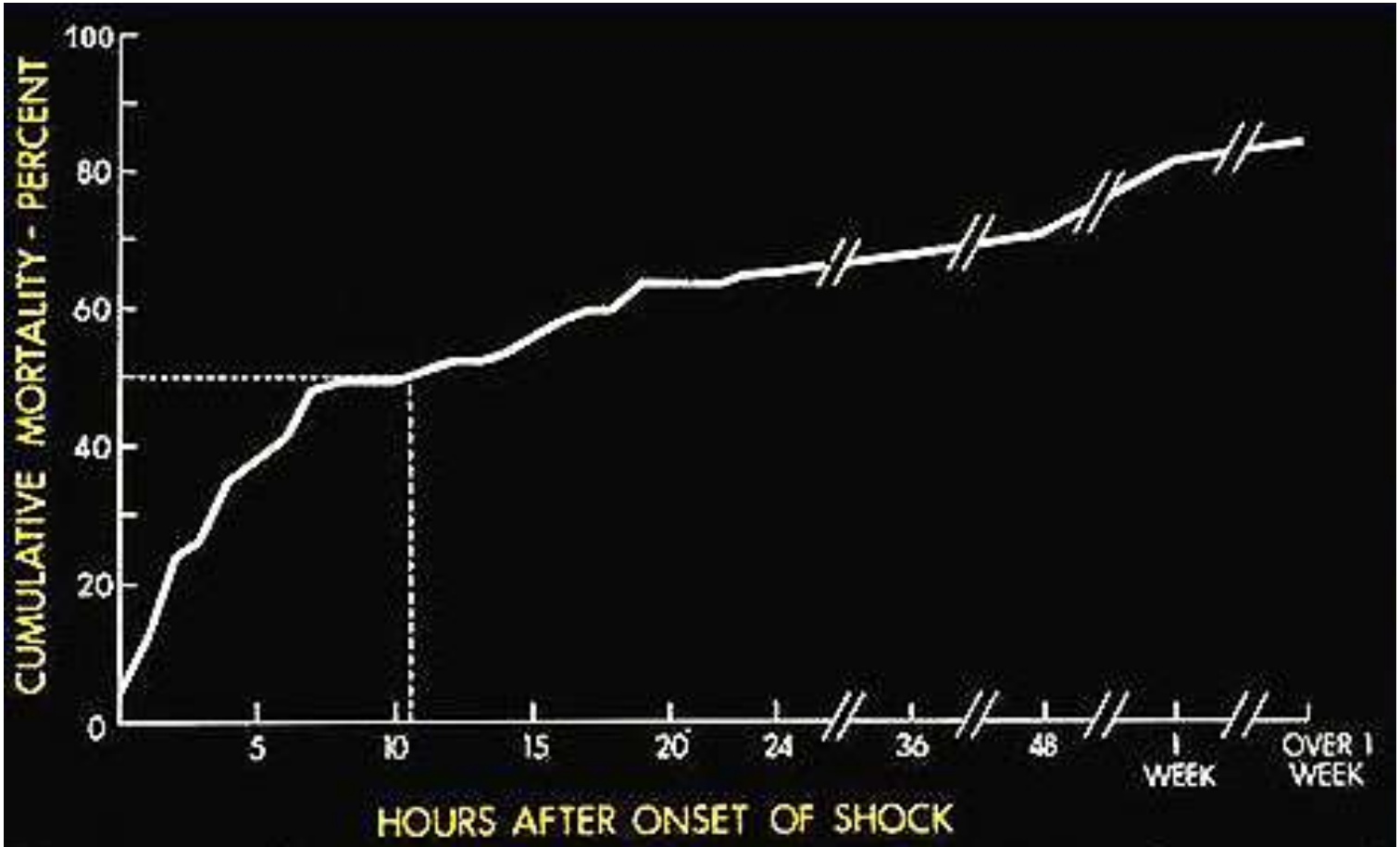

Figure 7. Cumulative mortality from the time of onset of shock. Half the group are dead within 10.2 hr (thin dashed line). Overall mortality is 86 percent.

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Hasdai et al. JACC 2000;36:687

Factors that increase risk for cardiogenic shock in STEMI

- Age > 70 years
- SBP < 120 mmHg
- Sinus tachycardia rate > 110/min.
- Bradycardia rate < 60/min.
- Increased time from onset of STEMI
- Anterior MI
- H/O Hypertension, diabetes mellitus
- Multi vessel coronary artery disease
- Prior MI or angina
- Prior diagnosis of heart failure, STEMI
- Left bundle branch block.

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JAMA. 2005;294:448–454.

Pathophysiology of Shock

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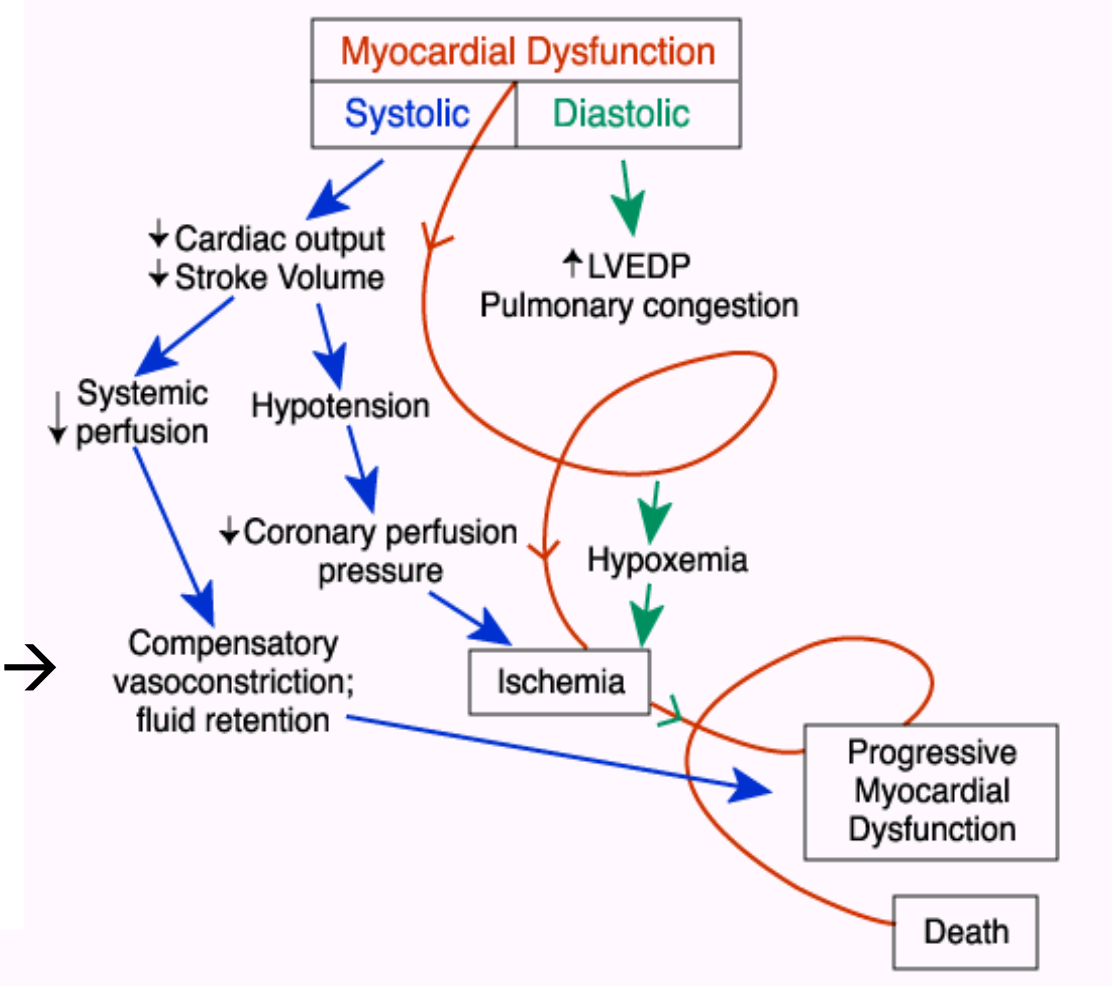
Schematic

- LVEDP elevation
- Hypotension
- Decreased coronary
- perfusion
- Ischemia
- Further myocardial
- dysfunction
- Neurohormonal activation →
- Vasoconstriction
- End organ hypoperfusion



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Pathophysiology of Shock

- Effect of Hypotension
  - Flow in normal coronary:
    - Regulated by microvascular resistance
    - Coronary flow may be preserved at AO pressures as low as 50 mm Hg
  - In coronary vessel with critical stenosis:
    - Vasodilator reserve of microvascular bed is exhausted
    - Decrease in AO pressure => Coronary hypoperfusion

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J Am Coll Cardiol. 2006;47(suppl A):111A.

Pathophysiology of Shock

Effect of Hypotension (continued…)

Normal heart extracts 65% of the O2 present in the blood

➔

Little room for augmentation of O2 extraction

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Pathophysiology of Shock

Hypotension + ↑LVEDP and critical stenosis → Myocardial Hypoperfusion → LV dysfunction → Systemic lactic acidosis → Impairment of non-ischemic myocardium → worsening hypotension.

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Right Ventricle

- RV dysfunction may cause or contribute to CS.
- Predominant RV shock represents only 5% of cases of CS complicating MI.
- RV failure may limit LV filling via a decrease in CO, ventricular interdependence, or both.
- Treatment of patients with RV dysfunction and shock has traditionally focused on ensuring adequate right-sided filling pressures to maintain CO and adequate LV preload; however, patients with CS due to RV dysfunction have very high RV end-diastolic pressure, often >20 mm Hg.

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J Am Coll Cardiol. 2003;41:1273–1279

Peripheral Vasculature, Neurohormones, and Inflammation

- Hypoperfusion of the extremities and vital organs is a hallmark of CS.
- The decrease in CO caused by MI and sustained by ongoing ischemia triggers release of catecholamines, which constrict peripheral arterioles to maintain perfusion of vital organs.
- Vasopressin and angiotensin II levels increase in the setting of MI and shock, which may further impair myocardial function.
- Activation of the neurohormonal cascade promotes salt and water retention; this may improve perfusion but exacerbates pulmonary edema

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Arch Intern Med. 2005;165:1643–1650.

CS May Be an Iatrogenic Illness

- Approximately three fourths of patients with CS complicating MI develop shock after hospital presentation.
- In some, medication use contributes to the development of shock.
- Several different classes of medications used to treat MI have been associated with shock, including Beta-blockers, angiotensin- converting enzyme inhibitors, and morphine.
- Although early use of each of these medications is associated with only a small excess risk of CS, the large number of patients treated with these therapies translates into a substantial potential number of events.

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Circulation. 2003;107:2998 –3002

- When high-dose diuretics are administered, plasma volume declines further.
- A trial of a low diuretic dose coupled with low-dose nitrates and positional measures to decrease preload (eg, seated position with legs down) should be attempted in patients with MI and pulmonary edema to avoid precipitating shock.
- Excess volume loading in patients with RV infarction may also cause or contribute to shock.

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Circulation. 2003;107:2998 –3002

Multivariable Mortality Predictors

- Increasing age and female gender
- Lower left ventricular ejection fraction
- Chronic renal insufficiency
- Initial and Final TIMI Flow grade
- Lower systolic blood pressure
- Diabetes mellitus
- Prior MI
- Increasing time from symptom onset to PCI
- Total Occlusion of the LAD
- Mitral regurgitation
- Multivessel PCI

1 Webb et al JACC 2003;42:1380
2 Sutton Heart 2005;91:339
3 Tedesco AHJ 2003:146; 472
4 Zeymer et al EHJ 2004;25:322
5 Tedesco JV Mayo Clin Proc 2003; 78:561
6 Sanborn JACC 2003:42; 1373
7 Klein et al AJC 2005; 96:35

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Diagnosis of cardiogenic shock

- History
- Physical examination
- ECG
- Cardiac biomarker
- CXR
- Echocardiography
- Pulmonary artery catheterization

  : Swan-Ganz catheter

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Diagnosis:physical examination

Sign of hypoperfusion

- Cyanosis, cooled skin, mottle extremities
- Elevated JVP and pulmonary crackles
- usually(but not always) presented
- Peripheral edema may be presented
- Heart sound : usually distant
- : S3, S4 may be presented
- Pulse pressure may be low, usually tachycardia
- Parasternal thrill : ventricular septal ruptured
- MR murmur may be limited to early systole
- HOCM : systolic murmur- louder upon valsalva and promp standing

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Diagnosis :ECG

- Acute coronary syndrome : STEMI, nonSTE ACS, RV infarction
- Cardiomyopathy
- Arrhythmia

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Diagnosis

CXR : pulmonary congestion, bilateral pleural effusion, cardiomegaly
: pericardial effusion
: widened mediastinum
: pneumothorax, pneumomediastinum

Echocardiogram
: LV function
: complication of MI eg. Acute MR, ventricular septal ruptured,free wall ruptured, cardiac tamponade
RV infarct : RV dilation and asynergy, abnormal inter ventricular and interatrial septal motion, Rt to Lt shunting through a patent PFO

Swan-Ganz catheter

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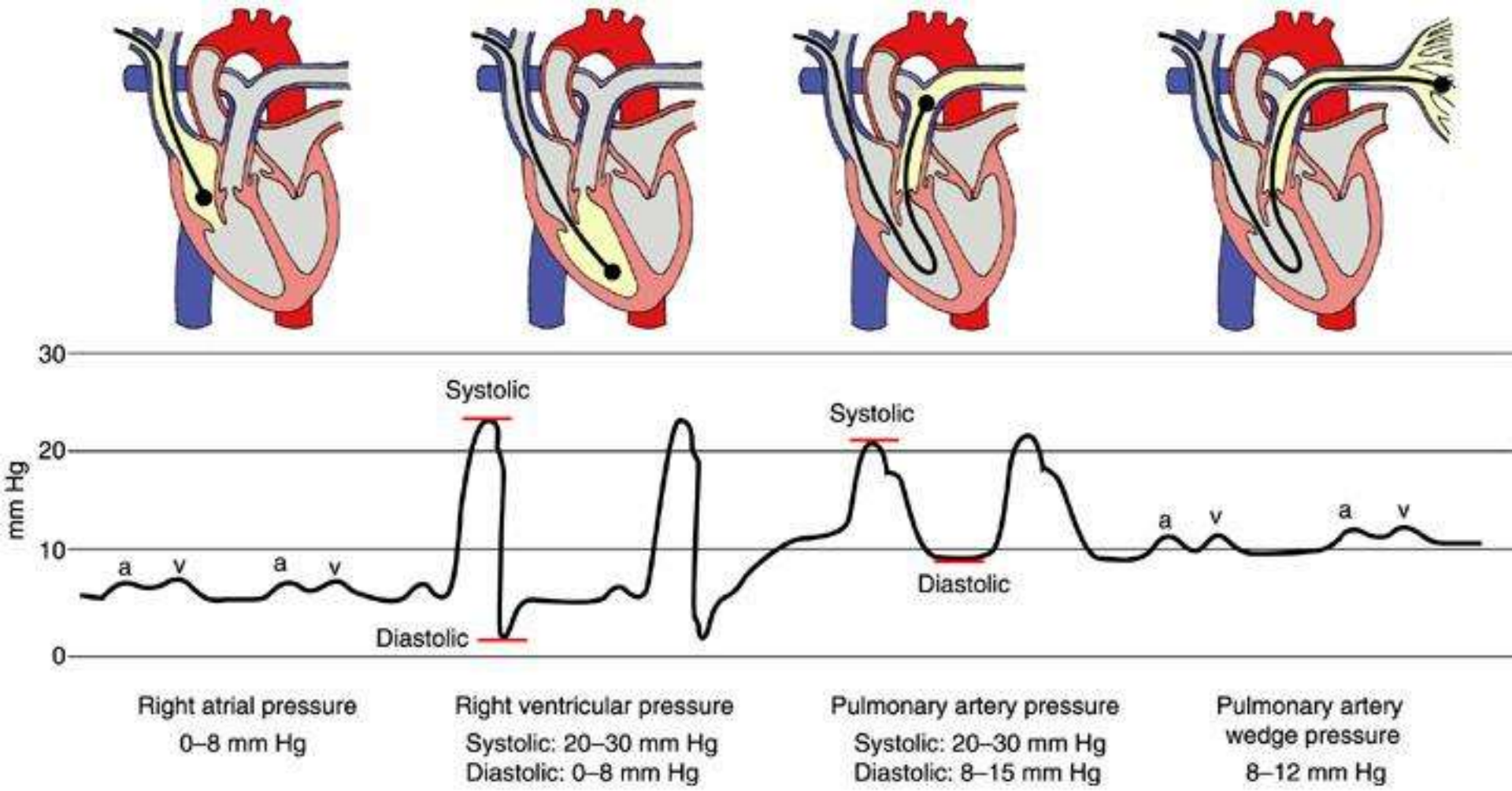

Right atrial pressure	Right ventricular pressure	Pulmonary artery pressure	Pulmonary artery wedge pressure
0–8 mm Hg	Systolic: 20–30 mm Hg Diastolic: 0–8 mm Hg	Systolic: 20–30 mm Hg Diastolic: 8–15 mm Hg	8–12 mm Hg

Normal values and wave configurations produced by the pulmonary artery catheter.

Copyright © 2005 Lippincott Williams & Wilkins. Instructor's Resource CD-ROM to Accompany Critical Care Nursing: A Holistic Approach, eighth edition.

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Pulmonary arterial catheterization: Swan-Ganz catheter

- Exclude other cause of shock
- Diagnosis of cardiogenic shock

1-PCWP > 15, CI < 2.2

2-Larged V wave on PCWP, very high PCWP = severe MR

3-Step up in oxygen sat.from RA to RV

large V- wave on RA pressure

= ventricular septal ruptured

4-High right sided filling pressure in the absense of elevated PCWP(RA > 10 and > 80% of PCWP) (accompanied with ECG criteria) = RV infarct

5-Classic sign of cardiac tamponade (equalization of diastolic pressure among the cardiac chambers) = free wall ruptured

(not always present)

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Cardiogenic shock in Acute MI

- Cardiogenic shock is the most common cause of death in patients with acute myocardial infarction (AMI)
- The incidence within the community over a 23-year period (1975-1997) was found to be 7.1%
- In the, Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Arteries (GUSTO-1) trial , the incidence of cardiogenic shock was likewise 7.2%, and consistent with other studies .
- In the GUSTO trial, 11% of patients had shock on presentation while 89% of patients subsequently developed shock

The GUSTO-I Investigators. Global utilization of streptokinase and tissue plasminogen activator for occluded coronary arteries.
J Am Coll Cardiol 1995; 26: 668-74.

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- Despite emerging innovative treatments, in-hospital mortality in patients with cardiogenic shock continues to be as high as 70-80%
- Cardiogenic shock seems to occur with a greater frequency amongst patients with ST-segment elevation myocardial infarction (STEMI). It was observed that shock developed in 7.5% of patients with STEMI and in 2.5% of patients with non-ST-segment elevation myocardial infarction (NSTEMI)

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SHOCK trial registry. J Am Coll Cardiol 2000; 36: 1091-96.

Causes of shock in AMI

- Acute Myocardial Infarction
- Left ventricular dysfunction
- Acute mitral regurgitation
- Ventricular septal rupture
- Right ventricular shock
- Cardiac Tamponade
- Cardiac Rupture

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Predisposing Factors for Cardiogenic Shock

- Age
- Sysolic blood pressure
- Heart rate
- Killip class
- Diabetes
- Anterior infarction
- Previous infarction
- Peripheral vascular disease
- Reduced ejection fraction
- Large infarctions
- Cardiac power

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Ventricular Septal Rupture

- Incidence 1-2%
- Timing 2-5 d p MI
- Murmur 90%
- Thrill common
- Echo shunt
- PA cath O2 step up > 9%

- IABP
- Inotropic Support
- Surgical Timing is controversial, but usually < 48 h

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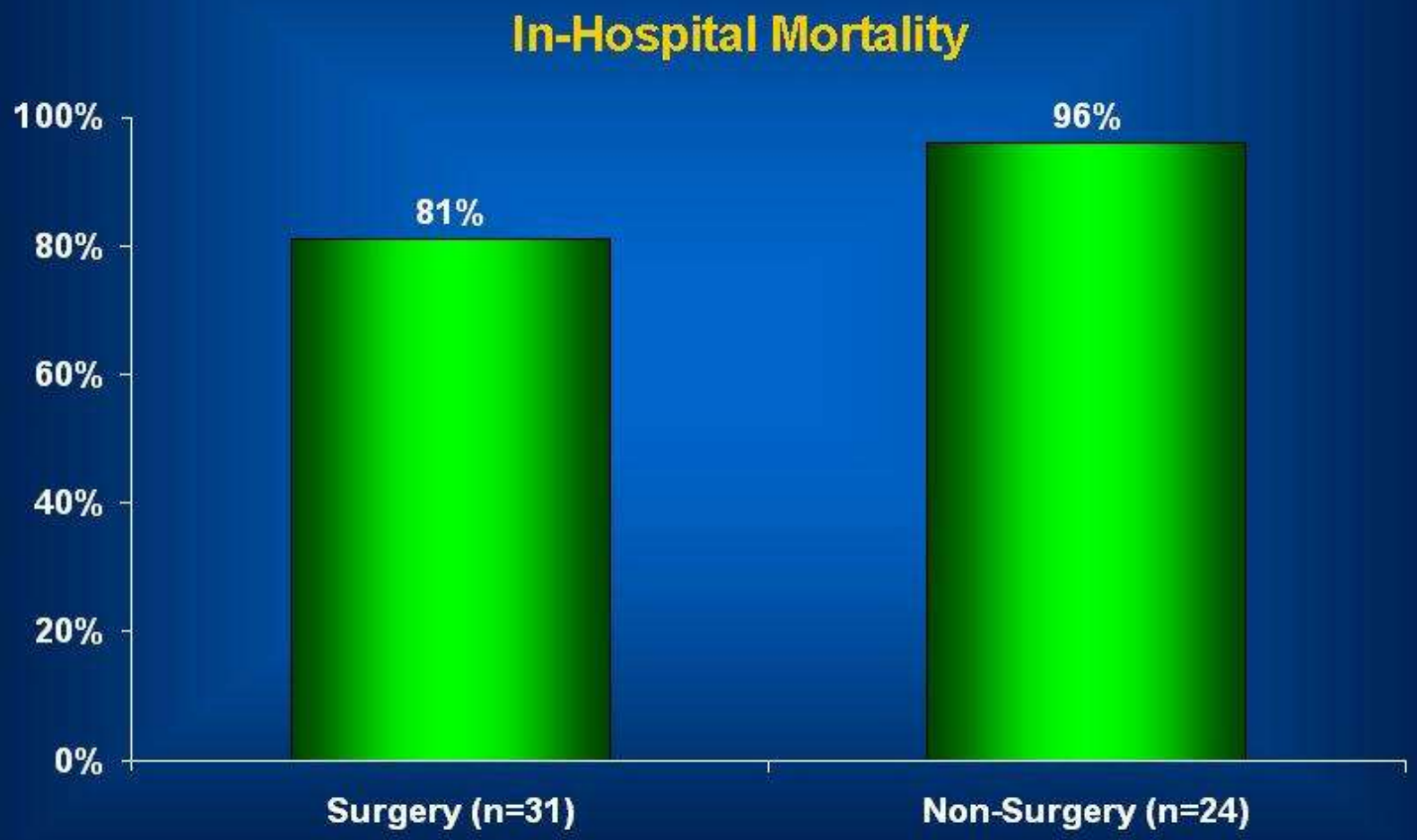
SHOCK Registry: Ventricular Septal Rupture



Menon, JACC 36: 1110, 2000

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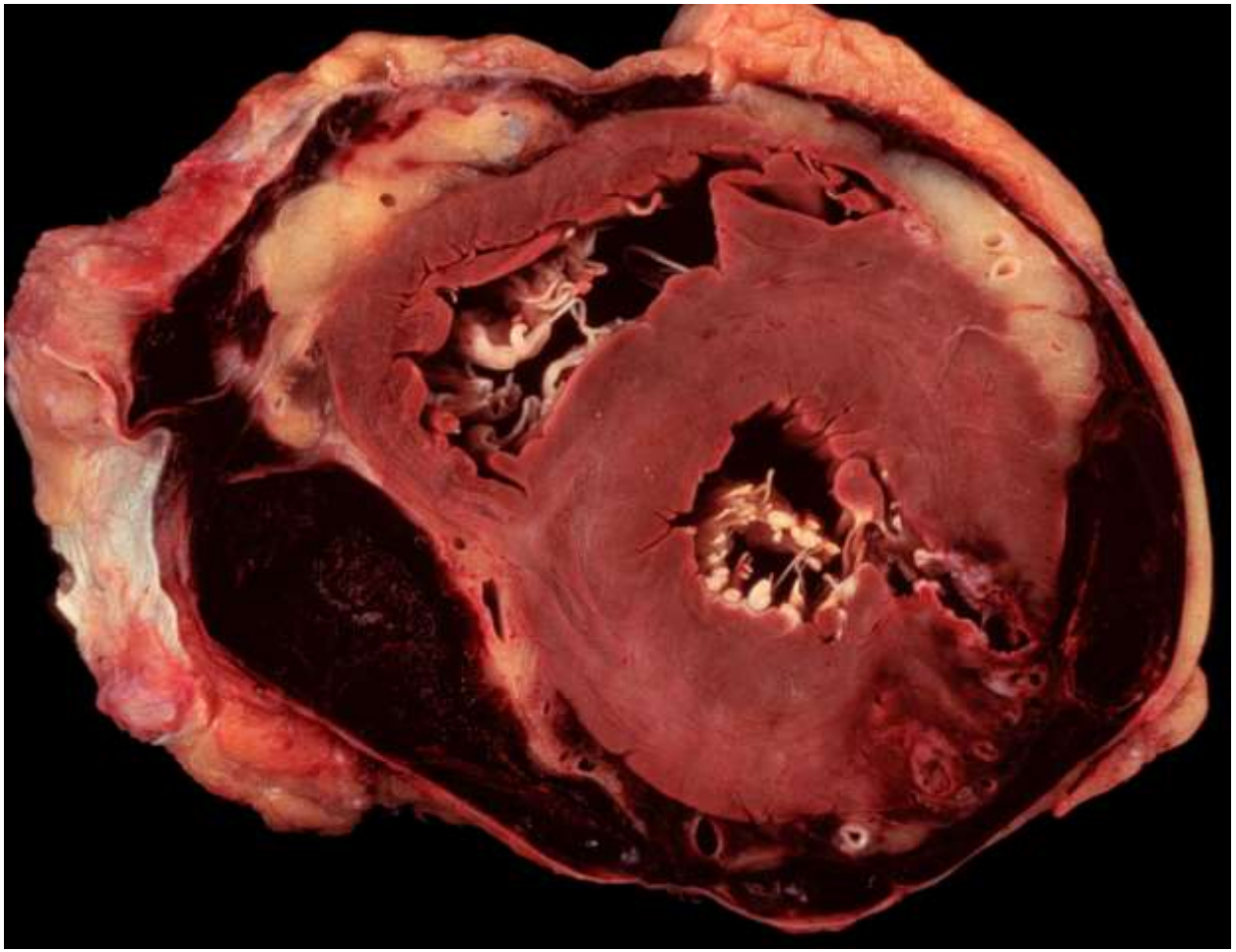
Free wall rupture



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Free Wall Rupture

- Incidence: 1-6%
- Occurs during first week after MI
- Classic Patient: Elderly, Female,Hypertensive
- Early thrombolysis reduces incidence but Late increases risk
- Echo: pericardial effusion,
- PA cath: equal diastolic pressure
- Treat with pericardiocentesis and early surgical repair

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J Am Coll Cardiol. 2000;36:1063–1070.

Acute Mitral Regurgitation

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Management of Acute MR

- Incidence: 1-2%
- Echo for Differential Diagnosis:

– Free-wall rupture

– VSD

– Infarct Extension

- PA Catheter: large v wave
- Afterload Reduction
- IABP
- Inotropic Therapy
- Early Surgical Intervention

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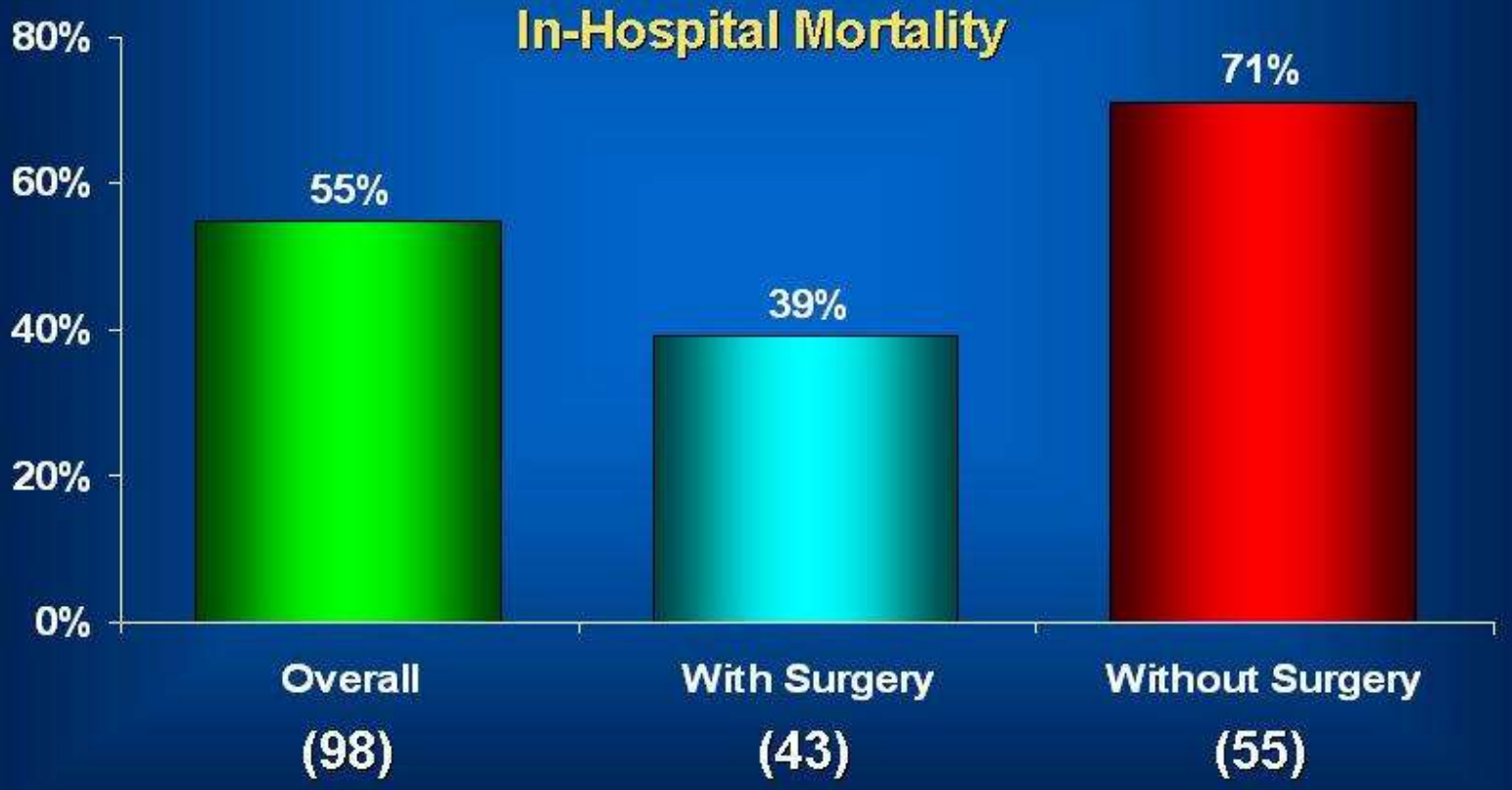
J Am Coll Cardiol. 2000;36:1063–1070.

SHOCK Registry: Acute Severe MR



Thompson, JACC 36: 1104, 2000

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Right Ventricular Infarction: Diagnosis

- **Clinical findings:**

Shock with clear lungs,

Elevated JVP

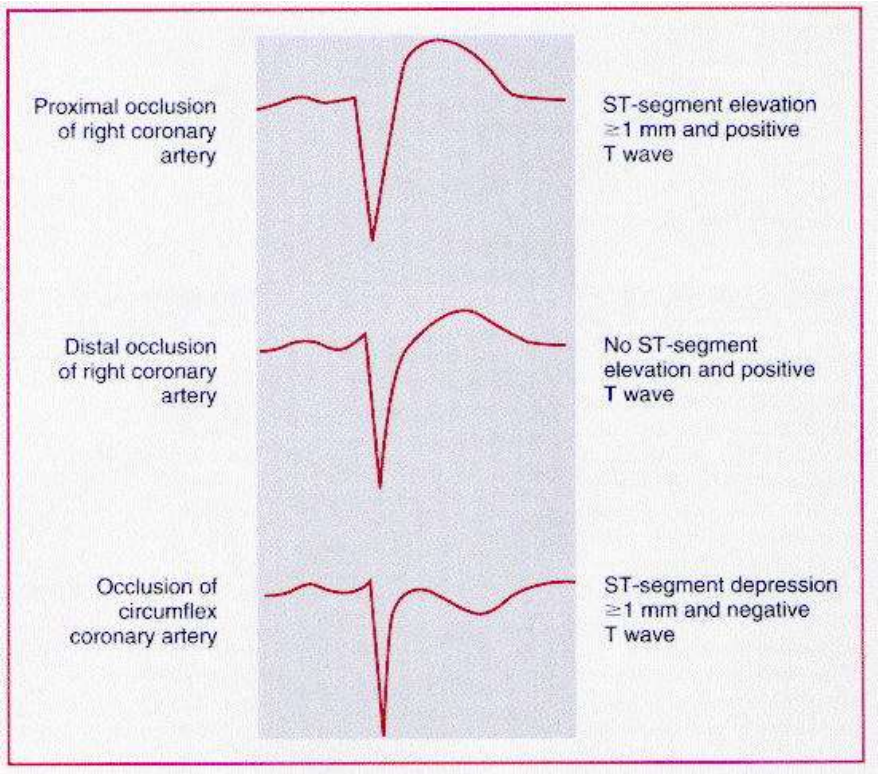
Kussmaul sign

- **ECG:**

ST elevation in R sided leads

- **Echo:**

Depressed RV function



V4R

Modified from Wellens. N Engl J Med 1999;340:381.

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Management of RV Infarction

- Cardiogenic Shock secondary to RV Infarct has better prognosis than LV Pump Failure
- IV Fluid Administration
- IABP
- Dobutamine
- Maintain A-V Synchrony
- Mortality with Successful Reperfusion = 2%
- Unsuccessful Reperfusion = 58%

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Outcomes of Cardiogenic Shock

The SHOCK registry

- **Similar mortality** in the two groups
  - 62.5% in non-ST elevation
  - 60.4% with ST elevation

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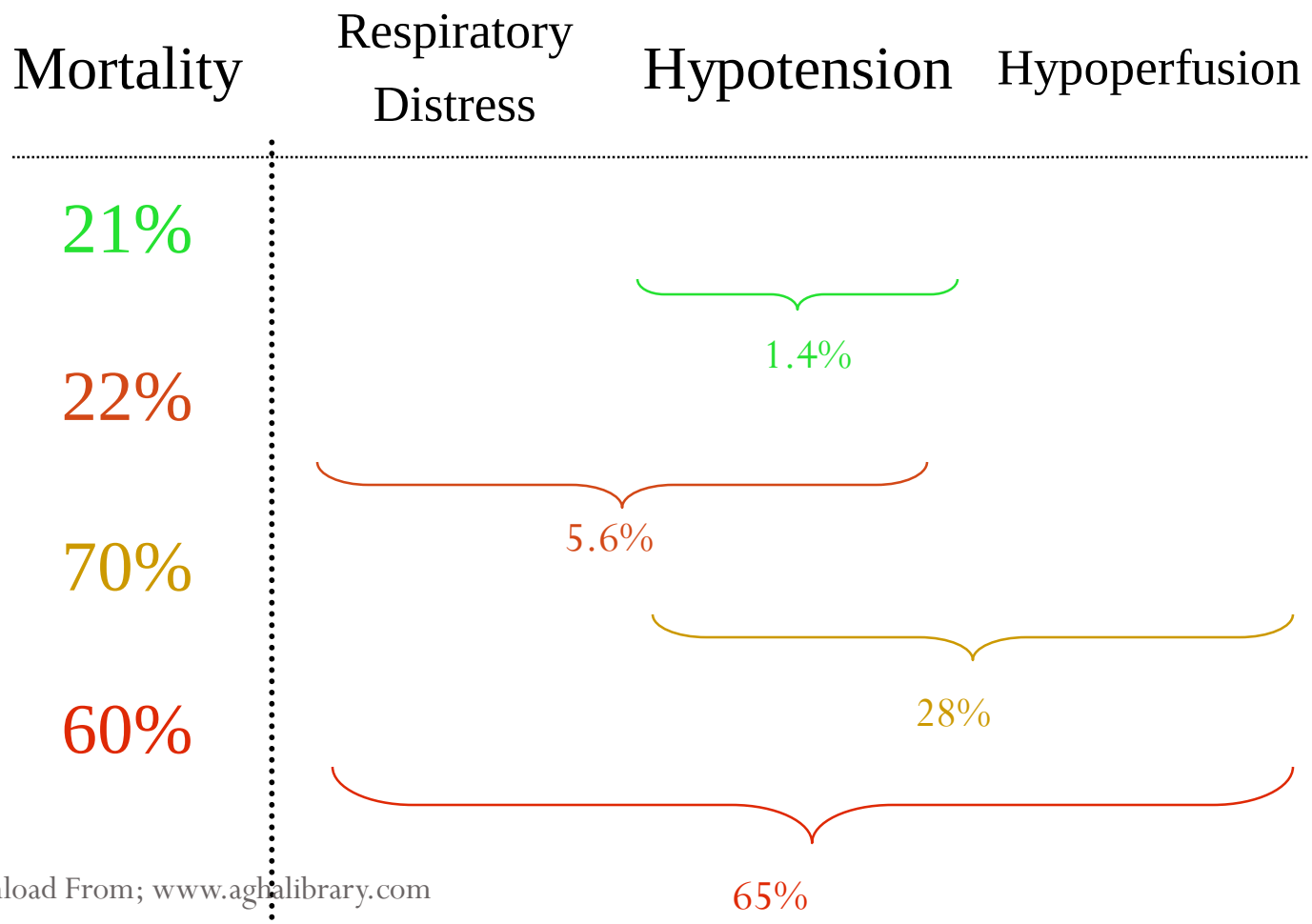
SHOCK Registry JACC Sept. 2000, Supp. A

Spectrum of Clinical Presentations



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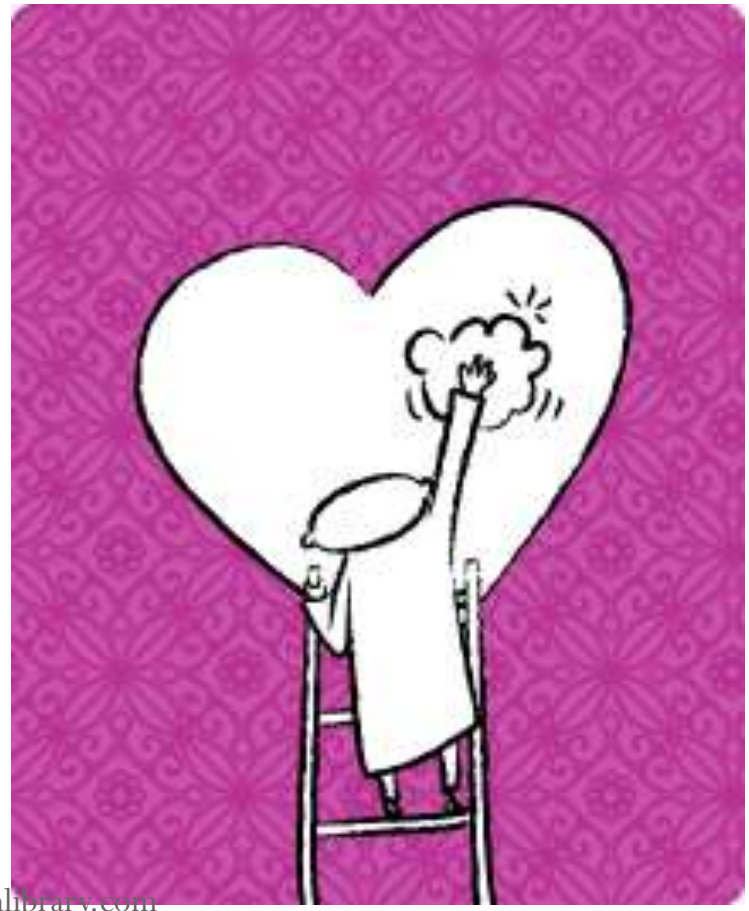
Treatment



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Supportive treatment

- Inotropic agent : dopamine, dobutamine, milrinone, amrinone
- Vasopressors : norepinephrine ,epinephrine, dopamine
- IABP
- Oxygen therapy and mechanical ventilator
- Arrhythmia treatment
- Magnesium, potassium, acidosis
- Volume replacement for RV infarct (0.5 – 1 L) or HOCM
- Ventricular assist device (VAD)
- Relief of pain and anxiety : morphine (or fentanyl if SBP is compromised)

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Dopamine

- Precursors of norepinephrine and epinephrine

. < 5 mcg/kg/min.: vasodilation of renal, mesenteric and coronary bed

. 5-10 mcg/kg/min.: beta-1: increase contractility and HR

.>= 10 mcg/kg/min.: alpha: arterial vasoconstriction and increase BP

.BP increasing : primarily due to inotropic effect

.undesirable effect : tachycardia, increase ,pulmonary shunt, decrease splanchnic perfusion, increase PCWP

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Norepinephrine

- Potent alpha with minimal beta adrnergic agonist

. Can increase BP successfully in patients remain hypotensive with dopamine

0.2-1.5 mcg/kg/min.

. As high as 3.3 mcg/kg/min. for sepsis

(because of alpha receptor down regulation)

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Epinephrine

- Increase MAP : increase CI, SV, SVR, HR
- Increase oxygen delivery and oxygen consumption

. Decrease splanchnic blood flow

. Increase systemic and regional lactate concentration

. Recommend only in patient who are unresponsive to traditional agent

. Undesirable effect : increase lactate conc., increase myocardial ischemia and arrhythmia,decrease splanchnic flow

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Dobutamine

- Sympathomimetic agent
- Beta-1, some beta-2, minimal alpha receptor activity

. Significant positive inotropic with mild chronotropic effect

. Mild peripheral vasodilation(decrease afterload)

. Significant increase CO

. Could increase infarct size of MI(increase oxygen consumption)

. Should be avoided in moderate to severe hypotension(SBP < 80 mmHg) because of peripheral vasodilation

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Phosphodiesterase inhibitor (PDIs): currently amrinone and milrinone

- Inotropic agent with peripheral vasodilation properties(decrease after load)

. Decrease PVR(decrease preload)

. Long half life

. May require concomittant vasopressors

. Unlike catecholamine inotrope: not dependent on adreno receptor activity- less likely to develop tolerance

. Less likely than catecholamine to cause adverse effect associated with adrenergic activity(eg.increase myocardial oxygen demand, myocardial ischemia, tachycardia)

. Incidence of tachyarrhythmia: PDIs> dobutamine

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Inotropes and Vasopressors

ACC/AHA Guidelines

SBP <70:-

Norepinephrine (0.5-30 μg/min)

Switch to Dopamine (5-15 μg/kg/min) once SBP ≥80

SBP 70-100

Dopamine (5-15 μg/kg/min)

Add dobutamine (2-20 μg/kg/min) once SBP ≥90

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Levosimendon

- Levosimendan, a myofilament Ca2+ sensitizer with inotropic effects, increases myocardial performance without substantial changes in oxygen consumption and with neutral effects on heart rhythm.
- Levosimendan has vasodilatory effects that are achieved by stimulation of adenosine triphosphate-dependent potassium channels.
- This action may be of specific interest in the setting of myocardial ischemia.
- The use of levosimendan is contraindicated in patients with: moderate-to-severe renal impairment, severe hepatic impairment, severe ventricular filling or outflow obstruction, severe hypotension and tachycardia, and/or history of torsades de pointes
- Dose 12ugm/kg f/b 0.2 ugm/kg/min

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Anesthesiology. 2006 Mar;104(3):556-69

Treatment –cont.....

- Antithrombotic therapy with aspirin and heparin should be given as routinely recommended for MI.
- Clopidogrel may be deferred until after emergency angiography, because on the basis of angiographic findings, coronary artery bypass grafting (CABG) may be performed immediately.
- Clopidogrel is indicated in all patients who undergo PCI, and on the basis of extrapolation of data from MI patients who were not in shock, it should also be useful in patients with shock as well.
- Negative inotropes and vasodilators (including nitroglycerin) should be avoided.
- Arterial oxygenation and near-normal pH should be maintained to minimize ischemia.

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Mechanical Support: IABP

- Intra-aortic balloon counterpulsation has long been the mainstay of mechanical therapy for CS.
- Use of an IABP improves coronary and peripheral perfusion via diastolic balloon inflation and augments LV performance via systolic balloon deflation with an acute decrease in afterload.
- Accurate timing of inflation and deflation provides optimal support.
- Not every patient has a hemodynamic response to IABP; response predicts better outcome.

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INDICATIONS

A) . Cardiogenic shock-(ACC/AHA Class IB recommendation)

- Bridge to revascularization-

  early placement of IABP - ↓ mortality.

  (Anderson, JACC 1997)

- Bridge to tertiary centre-

  ↑ 1 yr.survival rate (67%v/s 32%)

- Sec. to ischemic myocarditis

  (Kovack JACC,1997)

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The use of intra-aortic balloon counterpulsation in patients with cardiogenic shock complicating acute myocardial infarction: Data from the National Registry of Myocardial Infarction 2☆☆☆★

Abstract

Background Cardiogenic shock complicating acute myocardial infarction (AMI) remains the leading cause of death in patients hospitalized with AMI. Although several studies have demonstrated the importance of establishing and maintaining a patent infarct-related artery, it remains unclear as to whether intra-aortic balloon counterpulsation (IABP) provides incremental benefit to reperfusion therapy. The purpose of this study was to determine whether IABP use is associated with lower in-hospital mortality rates in patients with AMI complicated by cardiogenic shock in a large AMI registry. **Methods** We evaluated patients participating in the National Registry of Myocardial Infarction 2 who had cardiogenic shock at initial examination or in whom cardiogenic shock developed during hospitalization (n = 23,180). **Results** The mean age of patients in the study was 72 years, 54% were men, and the majority were white. The overall mortality rate in all patients who had cardiogenic shock or in whom cardiogenic shock developed was 70%. IABP was used in 7268 (31%) patients. IABP use was associated with a significant reduction in mortality rates in patients who received thrombolytic therapy (67% vs 49%) but was not associated with any benefit in patients treated with primary angioplasty (45% vs 47%). In a multivariate model, the use of IABP in conjunction with thrombolytic therapy decreased the odds of death by 18% (odds ratio, 0.82; 95% confidence interval, 0.72 to 0.93). **Conclusions** Patients with AMI complicated by cardiogenic shock may have substantial benefit from IABP when used in combination with thrombolytic therapy. (Am Heart J 2001;141:933-9.)

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Use of Intraaortic Balloon Counterpulsation in Patients Presenting With Cardiogenic Shock: Observations From the GUSTO-I Study

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Durham, North Carolina; Rochester, Minnesota; Brussels, Belgium; Ann Arbor and Farmington Hills, Maryland; and Cleveland, Ohio

Objectives. We sought to examine the use, complications and outcomes with early intraaortic balloon counterpulsation (IABP) in patients presenting with cardiogenic shock complicating acute myocardial infarction and treated with thrombolytic therapy.

Background. The use of IABP in patients with cardiogenic shock is widely accepted; however, there is a paucity of information on the use of this technique in patients with cardiogenic shock who are treated with thrombolytic therapy.

Methods. Patients who presented within 6 h of chest pain onset were randomized to one of four thrombolytic regimens. Cardiogenic shock was not an exclusion criterion, and data for these patients were prospectively collected. Patients presenting with shock were classified into early IABP (insertion within one calendar day of enrollment) or no IABP (insertion on or after day 2 or never).

Results. There were 68 (22%) IABP placements in 310 patients presenting with shock. Early IABP use occurred in 62 patients (20%) and none in 248 (80%). Most IABP use occurred in the United States (59 of 68 IABP placements) involving 32% of U.S. patients presenting with shock. Despite more adverse events in the early IABP group and more episodes of moderate bleeding, this cohort showed a trend toward lower 30-day and 1-year mortality rates.

Conclusions. IABP appears to be underutilized in patients presenting with cardiogenic shock, both within and outside the United States. Early IABP institution is associated with an increased risk of bleeding and adverse events but a trend toward lower 30-day and 1-year all-cause mortality.

(J Am Coll Cardiol 1997;30:708–15)
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Despite more adverse events in the early IABP group and more episodes of moderate bleeding, this cohort showed a trend toward lower 30-day and 1-year mortality rates.

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J Am Coll Cardiol 1997;30:708 –15

Circulation
JOURNAL OF THE AMERICAN HEART ASSOCIATION

Use of aortic counterpulsation to improve sustained coronary artery patency during acute myocardial infarction. Results of a randomized trial. The Randomized IABP Study Group.

E M Ohman, B S George, C J White, M J Kern, P A Gurbel, R J Freedman, C Lundergan, J R Hartmann, J D Talley and M J Frey

Circulation. 1994;90:792-799
doi: 10.1161/01.CIR.90.2.792

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Background Aortic counterpulsation has been observed to reduce the rate of reocclusion of the infarct-related artery after patency has been restored during acute myocardial infarction in observational studies. To evaluate the benefit-to-risk ratio of aortic counterpulsation during the early phase of myocardial infarction, a multicenter randomized clinical trial was performed.

Methods and Results Patients who had patency restored during acute cardiac catheterization within the first 24 hours of onset of myocardial infarction were randomly assigned to aortic counterpulsation for 48 hours versus standard care. Intravenous heparin was used similarly in both groups and was continued for a median (25th, 75th percentile) of 5 (2,7) days. A total of 182 patients were enrolled; 96 were assigned to aortic counterpulsation and 86 to standard care. Repeat cardiac catheterization was performed at a median of 5 (4,6) days after randomization in 89% of patients assigned to aortic counterpulsation and in 90% of control patients. Patients randomized to aortic counterpulsation had similar rates of severe bleeding complications (2% versus 1%), number of units of blood transfused (mean, 1.3±2.6 versus 0.9±1.8 units), and vascular repair or thrombectomy (5% versus 2%) compared with patients treated in a conventional manner. Patients randomized to aortic counterpulsation had significantly less reocclusion of the infarct-related artery during follow-up compared with control patients (8% versus 21%, $P<.03$). In addition, there was a significantly lower event rate in patients assigned to aortic counterpulsation in terms of a composite clinical end point (death, stroke, reinfarction, need for emergency revascularization with angioplasty or bypass surgery, or recurrent ischemia): 13% versus 24%, $P<.04$.

Conclusions This randomized trial showed that careful use of prophylactic aortic counterpulsation can prevent reocclusion of the infarct-related artery and improve overall clinical outcome in patients undergoing acute cardiac catheterization during myocardial infarction. (***Circulation.*** **1994;90:792-799.**)

Key Words • aorta • myocardial infarction • aortic counterpulsation • angioplasty

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JACC Vol. 29, No. 7
June 1997:1459–67

A Prospective, Randomized Evaluation of Prophylactic Intraaortic Balloon Counterpulsation in High Risk Patients With Acute Myocardial Infarction Treated With Primary Angioplasty

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Objectives. A large, international, multicenter, prospective, randomized trial was performed to determine the role of prophylactic intraaortic balloon pump (IABP) counterpulsation after primary percutaneous transluminal coronary angioplasty (PTCA) in acute myocardial infarction (AMI).

Background. Previous studies have suggested that routine IABP use after primary PTCA reduces infarct-related artery reocclusion, augments myocardial recovery and improves clinical outcomes.

Methods. Cardiac catheterization was performed in 1,100 patients within 12 h of onset of AMI at 34 clinical centers. Clinical and angiographic variables were used to stratify patients undergoing primary PTCA into high and low risk groups. High risk patients were then randomized to 36 to 48 h of IABP (n = 211) or traditional care (n = 226). The study had 80% power to detect a reduction in the primary end point from 30% to 20%.

Results. There was no significant difference in the predefined primary combined end point of death, reinfarction, infarct-related artery reocclusion, stroke or new-onset heart failure or sustained hypotension in patients treated with an IABP versus those treated conservatively (28.9% vs. 29.2%, p = 0.95). The IABP strategy conferred modest benefits in reduction of recurrent ischemia (13.3% vs. 19.6%, p = 0.08) and subsequent unscheduled repeat catheterization (7.6% vs. 13.3%, p = 0.05) but did not reduce the rate of infarct-related artery reocclusion (6.7% vs. 5.5%, p = 0.64), reinfarction (6.2% vs. 8.0%, p = 0.46) or mortality (4.3% vs. 3.1%) and was associated with a higher incidence of stroke (2.4% vs. 0%, p = 0.03). IABP use did not result in enhanced myocardial recovery as assessed by paired admission to predischarge and 6-week rest and exercise left ventricular ejection fraction.

Conclusions. In contrast to previous studies, a prophylactic IABP strategy after primary PTCA in hemodynamically stable high risk patients with AMI does not decrease the rates of infarct-related artery reocclusion or reinfarction, promote myocardial recovery or improve overall clinical outcome.

(J Am Coll Cardiol 1997;29:1459–67)

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European Heart Journal (2009) **30**, 459–468
doi:10.1093/eurheartj/ehn602

CLINICAL RESEARCH
Coronary heart disease

A systematic review and meta-analysis of intra-aortic balloon pump therapy in ST-elevation myocardial infarction: should we change the guidelines?

Krischan D. Sjauw, Annemarie E. Engström, Marije M. Vis, René J. van der Schaaf, Jan Baan Jr, Karel T. Koch, Robbert J. de Winter, Jan J. Piek, Jan G.P. Tijssen, and José P.S. Henriques*

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Aims Intra-aortic balloon counterpulsation (IABP) in ST-segment elevation myocardial infarction (STEMI) with cardiogenic shock is strongly recommended (class IB) in the current guidelines. We performed meta-analyses to evaluate the evidence for IABP in STEMI with and without cardiogenic shock.

Methods and results Medical literature databases were scrutinized to identify randomized trials comparing IABP with no IABP in STEMI. In absence of randomized trials, cohort studies of IABP in STEMI with cardiogenic shock were identified. Two separate meta-analyses were performed respectively. The first meta-analysis included seven randomized trials ($n = 1009$) of STEMI. IABP showed neither a 30-day survival benefit nor improved left ventricular ejection fraction, while being associated with significantly higher stroke and bleeding rates. The second meta-analysis included nine cohorts of STEMI patients with cardiogenic shock ($n = 10529$). In patients treated with thrombolysis, IABP was associated with an 18% [95% confidence interval (CI), 16–20%; $P < 0.0001$] decrease in 30 day mortality, albeit with significantly higher revascularization rates compared to patients without support. Contrariwise, in patients treated with primary percutaneous coronary intervention, IABP was associated with a 6% (95% CI, 3–10%; $P < 0.0008$) increase in 30 day mortality.

Conclusion The pooled randomized data do not support IABP in patients with high-risk STEMI. The meta-analysis of cohort studies in the setting of STEMI complicated by cardiogenic shock supported IABP therapy adjunctive to thrombolysis. In contrast, the observational data did not support IABP therapy adjunctive to primary PCI. All available observational data concerning IABP therapy in the setting of cardiogenic shock is importantly hampered by bias and confounding. There is insufficient evidence endorsing the current guideline recommendation for the use of IABP therapy in the setting of STEMI complicated by cardiogenic shock. Our meta-analyses challenge the current guideline recommendations.

European Heart Journal (2009) **30**, 459–468

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The NEW ENGLAND JOURNAL of MEDICINE

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Intraaortic Balloon Support for Myocardial Infarction with Cardiogenic Shock

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ABSTRACT

BACKGROUND

In current international guidelines, intraaortic balloon counterpulsation is considered to be a class I treatment for cardiogenic shock complicating acute myocardial infarction. However, evidence is based mainly on registry data, and there is a paucity of randomized clinical trials.

METHODS

In this randomized, prospective, open-label, multicenter trial, we randomly assigned 600 patients with cardiogenic shock complicating acute myocardial infarction to intraaortic balloon counterpulsation (IABP group, 301 patients) or no intraaortic balloon counterpulsation (control group, 299 patients). All patients were expected to undergo early revascularization (by means of percutaneous coronary intervention or bypass surgery) and to receive the best available medical therapy. The primary efficacy end point was 30-day all-cause mortality. Safety assessments included major bleeding, peripheral ischemic complications, sepsis, and stroke.

RESULTS

A total of 300 patients in the IABP group and 298 in the control group were included in the analysis of the primary end point. At 30 days, 119 patients in the IABP group (39.7%) and 123 patients in the control group (41.3%) had died (relative risk with IABP, 0.96; 95% confidence interval, 0.79 to 1.17; P=0.69). There were no significant differences in secondary end points or in process-of-care measures, including the time to hemodynamic stabilization, the length of stay in the intensive care unit, serum lactate levels, the dose and duration of catecholamine therapy, and renal function. The IABP group and the control group did not differ significantly with respect to the rates of major bleeding (3.3% and 4.4%, respectively; P=0.51), peripheral ischemic complications (4.3% and 3.4%, P=0.53), sepsis (15.7% and 20.5%, P=0.15), and stroke (0.7% and 1.7%, P=0.28).

CONCLUSIONS

The use of intraaortic balloon counterpulsation did not significantly reduce 30-day mortality in patients with cardiogenic shock complicating acute myocardial infarction for whom an early revascularization strategy was planned. (Funded by the German Research Foundation and others; IABP-SHOCK II ClinicalTrials.gov number, NCT00491036.)

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Reperfusion

- The survival benefit of early revascularization in CS, reported in several observational studies, was shown convincingly in the randomized SHOCK trial, which found a 13% absolute increase in 1-year survival in patients assigned to early revascularization.
- This corresponds to a number needed to treat of 8 patients to save 1 life.
- The benefit was similar in the incomplete, randomized Swiss Multicenter Study of Angioplasty for Shock.

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JAMA.
2006;295:2511–2515.

- Numerous registry studies have confirmed the survival advantage of early revascularization, whether percutaneous or surgical, in the young and the elderly.
- Thrombolytic therapy is less effective but is indicated when PCI is impossible or if a delay has occurred in transport for PCI and when MI and CS onset were within 3 hours.

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Timing of PCI

- As in MI without shock, earlier revascularization is better in CS.
- Presentation 0 to 6 hours after symptom onset was associated with the lowest mortality among CS patients undergoing primary PCI in the Arbeitsgemeinschaft Leitende Kardiologische Krankenhausarzte (ALKK) registry, in which door-to-angiography times were 90 minutes in approximately three fourths of patients.
- In the SHOCK trial, there appeared to be increasing long-term mortality as time to revascularization increased from 0 to 8 hours.
- However, there is a survival benefit as long as 48 hours after MI and 18 hours after shock onset

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JAMA.
2006;295:2511–2515.

Stenting and Glycoprotein IIb/IIIa Inhibition

- Stenting and glycoprotein IIb/IIIa inhibitors were independently associated with improved outcomes in patients undergoing PCI for CS in multiple registries, including the large ACC-National Cardiovascular Data Registry.
- A trend toward benefit of abciximab was noted in a small subset of STEMI patients with CS undergoing PCI in a randomized trial.

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JAMA.
2006;295:2511–2515.

Revascularization Approach: Surgery or PCI

- Revascularization in the SHOCK trial could be percutaneous or surgical.
- Thirty-seven percent of patients assigned to the early revascularization strategy underwent CABG at a median of 2.7 hours after randomization.
- Despite a higher prevalence of triple-vessel or left main disease and diabetes mellitus in patients who underwent CABG compared with PCI, survival and quality of life were similar.
- The rate of emergency CABG in CS is much lower in the community (<10%).

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JAMA.
2006;295:2511–2515.

- Survival in patients with CS who have CABG may improve further with advancing surgical techniques.
- In CS patients undergoing surgery, a trend toward better survival with beating heart techniques was present despite the use of slightly lower numbers of grafts.
- This latter point could be of particular importance when one considers the potential for long-term survival in CS patients

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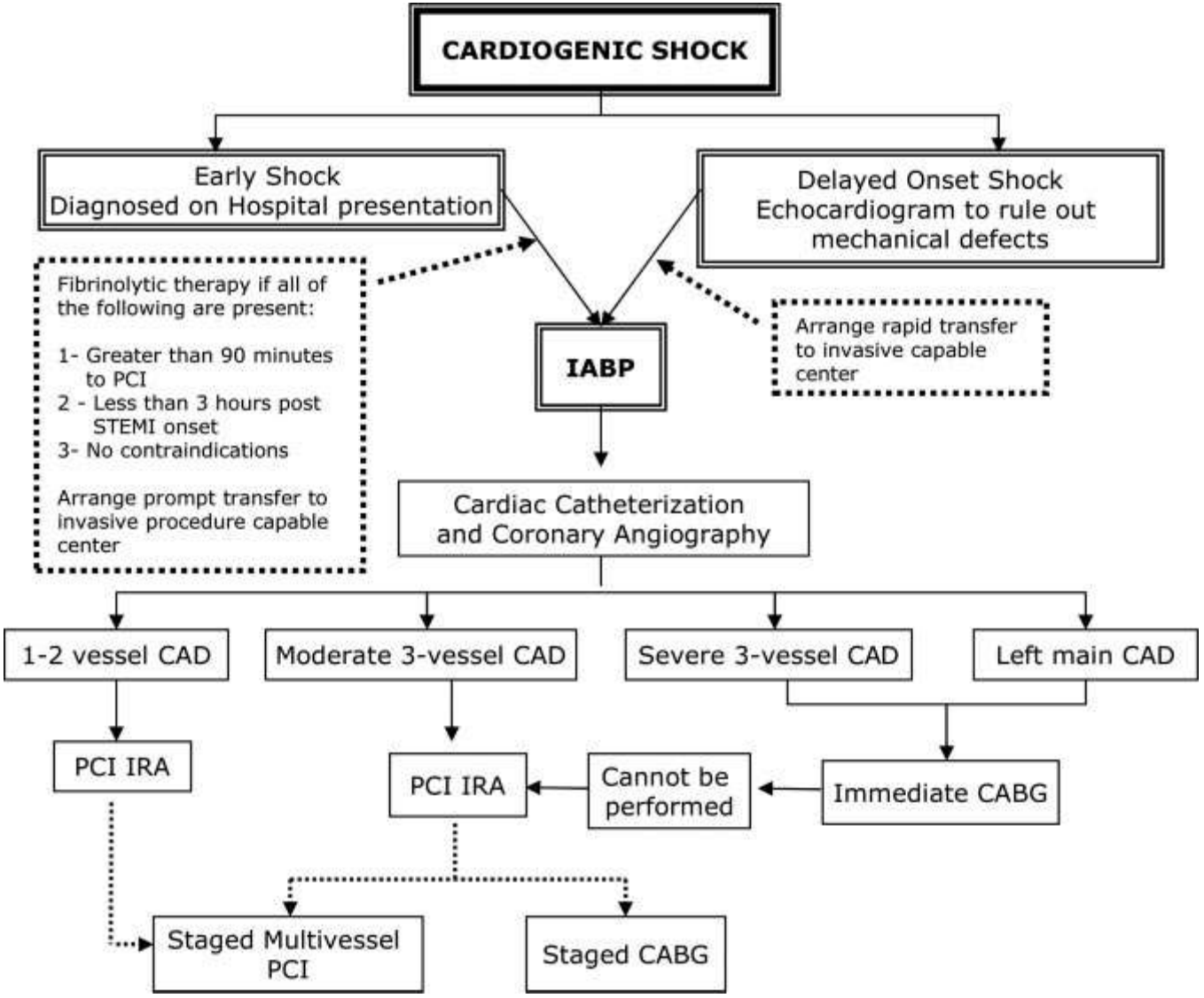
Eur Heart J. 1999;20:1030 –1038



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Circulation. 2004;110:588–636

Thrombolytic therapy

- The outcome of cardiogenic shock is closely linked to the patency of the culprit coronary arteries
- Thrombolytic therapy has decreased the occurrence of shock among patients with persistent STEMI.
- The GUSTO-I : t-PA is more efficacious than streptokinase in preventing shock.

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Thrombolysis in cardiogenic shock

- Results have been disappointing
- Cause : ? limited efficacy of lytics in the setting of low perfusion pressure.

- GISSI-I Study

  Mortality of thrombolysis(streptokinase) group = 69.9%

  Mortality of. control group = 70.1%

David Hasdai et al,Lancet 2000;356:753

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Risk Stratification and Targeting the Population for Revascularization

- Mortality due to CS is not as high as many clinicians may believe and is 50% in the modern era, far lower than historic figures of 80% to 90%.
- Mortality can range from10% to 80% depending on demographic, clinical, and hemodynamic factors.
- These factors include age, clinical signs of peripheral hypoperfusion, anoxic brain damage, LVEF, and stroke work.
- Female sex does not appear to be an independent predictor of poor outcome.

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Total Circulatory Support: LV Assist Devices and Extracorporeal Life Support

- Temporary mechanical circulatory support with LV assist devices (LVADs) is theoretically appealing to interrupt the vicious spiral of ischemia, hypotension, and myocardial dysfunction, allowing for recovery of stunned and hibernating myocardium and reversal of neurohormonal derangements.
- Device-related complications and irreversible organ failure remain major limitations.

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- VAD with active circulatory support reverse hemodynamic and metabolic parameters in cardiogenic shock more effectively than with standard IABP treatment alone.

- The use of newer percutaneous devices is still in its infancy.

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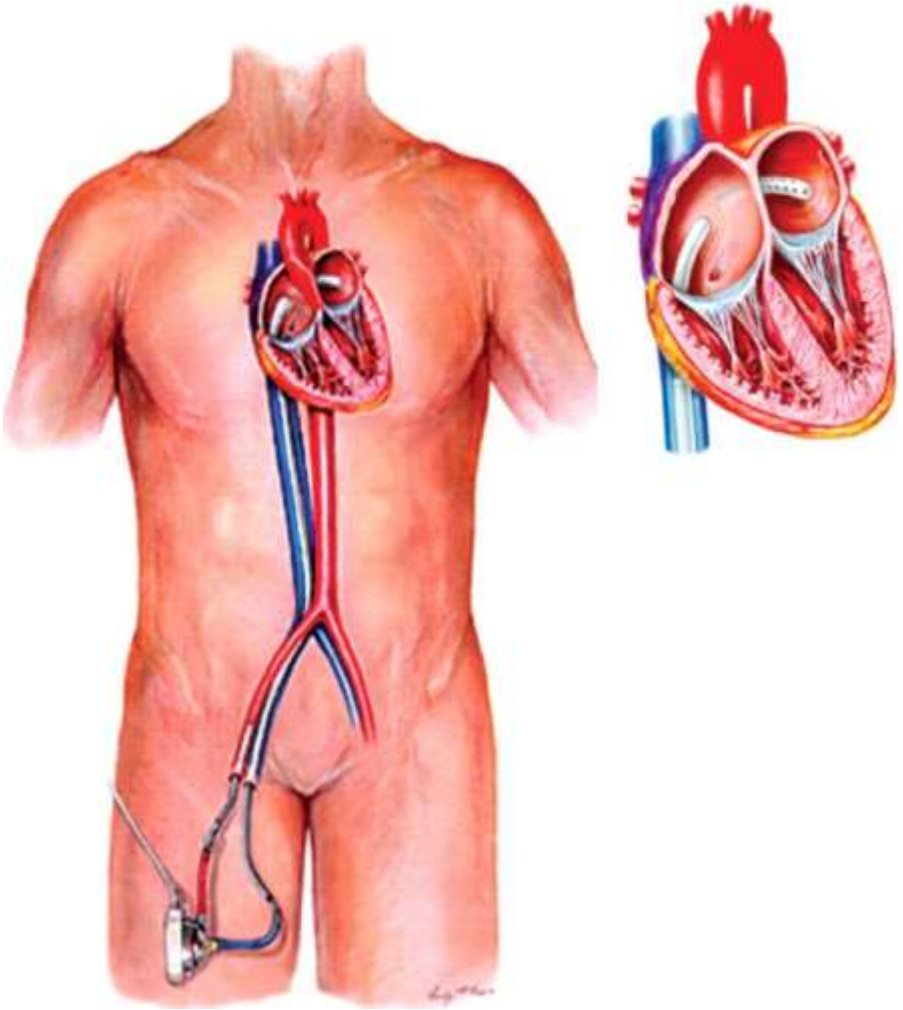
Tandem Heart LVAD-Extracorporeal devices

- Left atrial-to-femoral arterial LVAD
- Low speed centrifugal continuous flow pump
- 21F venous transeptal cannula
- 17F arterial cannula
- Maximum flow 4L/minute



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- The TandemHeart (Cardiac Assist, Inc, Pittsburgh, Pa) removes blood from the left atrium using a cannula placed through the femoral vein and into the left atrium via transseptal puncture.
- Blood is then returned to a systemic artery, usually the femoral, with retrograde perfusion of the abdominal and thoracic aorta.

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European Heart Journal (2005) 26, 1276–1283
doi:10.1093/eurheartj/ehi161

EUROPEAN SOCIETY OF CARDIOLOGY®

Clinical research

Randomized comparison of intra-aortic balloon support with a percutaneous left ventricular assist device in patients with revascularized acute myocardial infarction complicated by cardiogenic shock

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Aims Mortality in cardiogenic shock (CS) following acute myocardial infarction (AMI) remains unacceptably high despite percutaneous coronary intervention (PCI) of the infarcted artery and use of intra-aortic balloon pump (IABP) counterpulsation. A newly developed percutaneous left ventricular assist device (VAD) (Tandem Heart™, Cardiac Assist, Pittsburgh, PA, USA) with active circulatory support might have positive haemodynamic effects and decrease mortality.

Methods and results Patients in CS after AMI, with intended PCI of the infarcted artery, were randomized to either IABP ($n = 20$) or percutaneous VAD support ($n = 21$). The primary outcome measure cardiac power index, as well as other haemodynamic and metabolic variables, could be improved more effectively by VAD support from 0.22 [interquartile range (IQR) 0.19–0.30] to 0.37 W/m^2 (IQR 0.30–0.47, $P < 0.001$) when compared with IABP from 0.22 (IQR 0.18–0.30) to 0.28 W/m^2 (IQR 0.24–0.36, $P = 0.02$; $P = 0.004$ for intergroup comparison). However, complications like severe bleeding ($n = 19$ vs. $n = 8$, $P = 0.002$) or limb ischaemia ($n = 7$ vs. $n = 0$, $P = 0.009$) were encountered more frequently after VAD support, whereas 30 day mortality was similar (IABP 45% vs. VAD 43%, log-rank, $P = 0.86$).

Conclusion Haemodynamic and metabolic parameters can be reversed more effectively by VAD than by standard treatment with IABP. However, more complications were encountered by the highly invasive procedure and by the extracorporeal support.

European Heart Journal (2005) 26, 1276–1283

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Intracorporeal devices

IMPELLA LP 2.5 & LP 5.0

- Helical propellar-axial flow
- Minimally –invasive,percutaneous catheter LVADs.
- Insertion similar to IABP but device rests across the aortic valve,with the tip in the LV cavity.
- Full anticoagulation needed.
- FDA approved for high risk PCI,post PCI,cardiogenic shock,myocarditis &bridge to decision.

TandemHeart and Impella are currently available percutaneous LVAD for hemodynamic support in cath lab.

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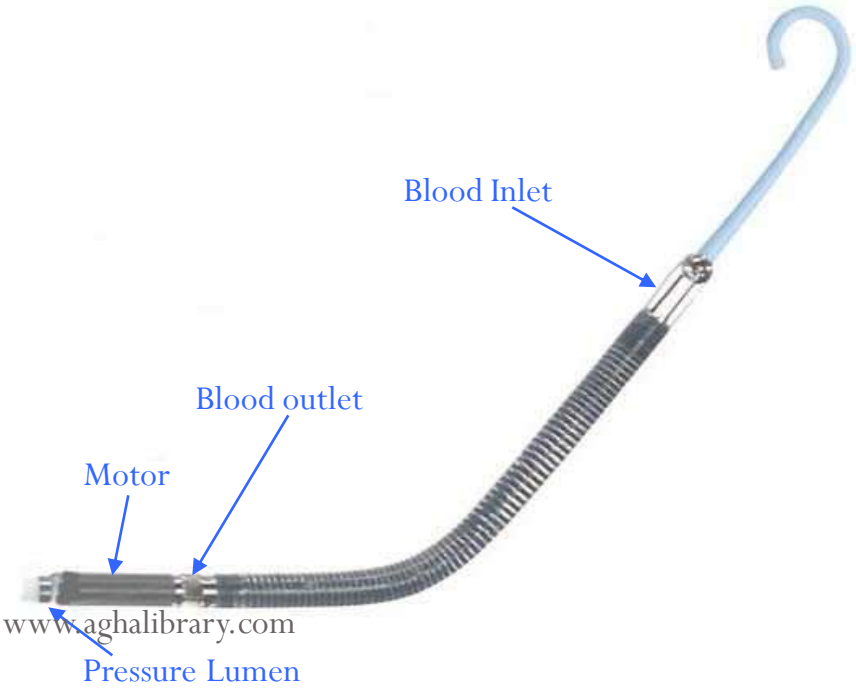
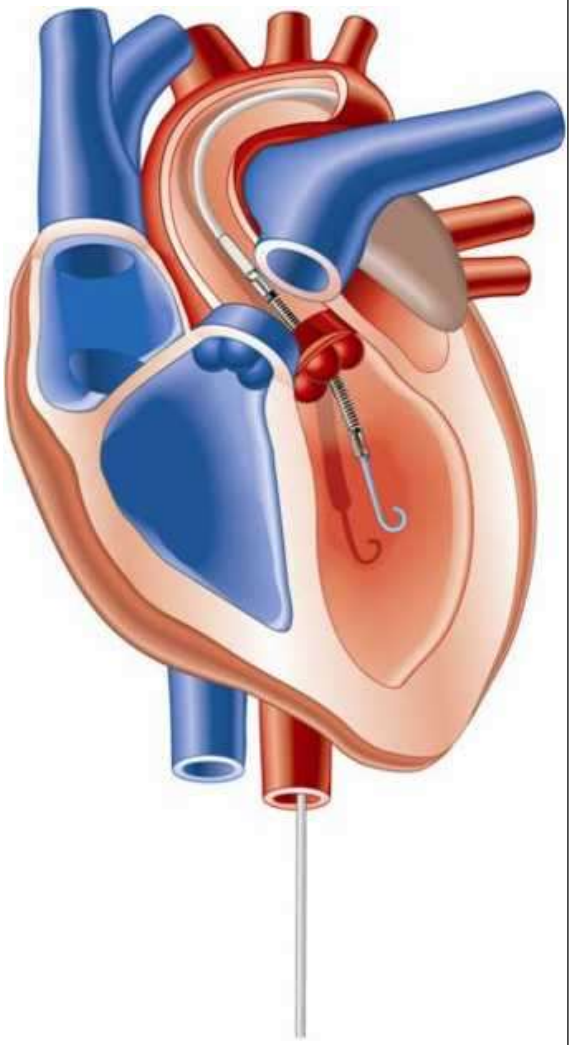
Impella

- Axial flow pump
- Much simpler to use
- Increases cardiac output & unloads LV
- LP 2.5
  - 12 F percutaneous approach; Maximum 2.5 L flow
- LP 5.0
  - 21 F surgical cutdown; Maximum 5L flow





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WORKS IN PROGRESS

A Randomized Clinical Trial to Evaluate the Safety and Efficacy of a Percutaneous Left Ventricular Assist Device Versus Intra-Aortic Balloon Pumping for Treatment of Cardiogenic Shock Caused by Myocardial Infarction

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Munich, Germany

J Am Coll Cardiol 2008;52:1584–8

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Objectives The aim of this study was to test whether the left ventricular assist device (LVAD) Impella LP2.5 (Abiomed Europe GmbH, Aachen, Germany) provides superior hemodynamic support compared with the intra-aortic balloon pump (IABP).

Background Cardiogenic shock caused by left ventricular failure is associated with high mortality in patients with acute myocardial infarction (AMI). An LVAD may help to bridge patients to recovery from left ventricular failure.

Methods In a prospective, randomized study, 26 patients with cardiogenic shock were studied. The primary end point was the change of the cardiac index (CI) from baseline to 30 min after implantation. Secondary end points included lactic acidosis, hemolysis, and mortality after 30 days.

Results In 25 patients the allocated device (n = 13 IABP, n = 12 Impella LP2.5) could be safely placed. One patient died before implantation. The CI after 30 min of support was significantly increased in patients with the Impella LP2.5 compared with patients with IABP (Impella: $\Delta CI = 0.49 \pm 0.46$ l/min/m^2; IABP: $\Delta CI = 0.11 \pm 0.31$ l/min/m^2; $p = 0.02$). Overall 30-day mortality was 46% in both groups.

Conclusions In patients presenting with cardiogenic shock caused by AMI, the use of a percutaneously placed LVAD (Impella LP 2.5) is feasible and safe, and provides superior hemodynamic support compared with standard treatment using an intra-aortic balloon pump. (Efficacy Study of LV Assist Device to Treat Patients With Cardiogenic Shock [ISAR-SHOCK]; NCT00417378) (J Am Coll Cardiol 2008;52:1584–8)

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J Am Coll Cardiol 2008;52:1584–8

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Impella outcome data

- ISAR-SHOCK
  - 26 patient RCT Impella vs IABP
  - ↑ Cardiac Index, ↑ MAP (by 10mmHg) vs IABP
  - Complications ≤ IABP
  - No difference in mortality

- PROTECT-II
  - 654 patients RCT IABP vs Impella in high-risk PCI
  - Stopped after n= 305 due to futility
  - Primary EP composite of 10 MAEs
  - Incidence 38% Impella vs 43% IABP

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IMPELLA LP 2.5

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Management of Special Conditions

- The treatment of certain conditions that lead to CS is marked by important differences from management of CS due to LV failure.
- The recognition of LV outflow obstruction is critical in patients with hypotension, because diuretics and inotropic agents exacerbate obstruction.
- Treatment of CS with hypertrophic obstructive cardiomyopathy includes volume resuscitation and beta-blockade.

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- Outflow obstruction may also be seen in some cases of tako-tsubo cardiomyopathy when extensive akinesis/ dyskinesis of apical zones occurs with hyperkinesis of remaining myocardium.
- Therapy is guided by echocardiography and clinical response.
- IABP may provide circulatory support.
- Beta-blockade is often not indicated in this circumstance because it exacerbates LV dysfunction.

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Long-Term Survival and Quality of Life

- Long-term survival data from the SHOCK trial were reported recently.
- Remarkably, the 3- and 6-year survival rates in the early revascularization group were 41.4% and 32.8% with persistence of treatment benefit.
- These rates are similar to or better than 30-day survival rates reported in studies in which patients did not routinely receive invasive therapy and similar to 5-year survival rates for many forms of cancer.

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JAMA.
2006;295:2511–2515.

- At least as important as long-term survival is quality of life in survivors.
- Again, this is far better than many clinicians would suspect.
- Already at 2 weeks after discharge, 75.9% of patients assigned to revascularization and 62.5% of patients assigned to medical stabilization in the SHOCK trial were in New York Heart Association functional class I to II .
- 60 Among patients who were in functional class III to IV at 2 weeks, 55% of survivors improved to class I to II by 1 year.

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2013 ACCF/AHA Guideline for the Management of ST-Elevation Myocardial Infarction

Developed in Collaboration with American College of Emergency Physicians and Society for Cardiovascular Angiography and Interventions

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Primary PCI in STEMI

Primary PCI should be performed in patients with STEMI and ischemic symptoms of less than 12 hours' duration.

Primary PCI should be performed in patients with STEMI and ischemic symptoms of less than 12 hours' duration who have contraindications to fibrinolytic therapy, irrespective of the time delay from FMC.

Primary PCI should be performed in patients with STEMI and cardiogenic shock or acute severe HF, irrespective of time delay from MI onset.

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Indications for Coronary Angiography in Patients Who Were Managed With Fibrinolytic Therapy or Who Did Not Receive Reperfusion Therapy

	COR	LOE
Cardiogenic shock or acute severe HF that develops after initial presentation	I	B
Intermediate- or high-risk findings on predischarge noninvasive ischemia testing	I	B
Spontaneous or easily provoked myocardial ischemia	I	C
Failed reperfusion or reocclusion after fibrinolytic therapy	IIa	B
Stable* patients after successful fibrinolysis, before discharge and ideally between 3 and 24 h	IIa	B

*Although individual circumstances will vary, clinical stability is defined by the absence of low output, hypotension, persistent tachycardia, apparent shock, high-grade ventricular or symptomatic supraventricular tachyarrhythmias, and spontaneous recurrent ischemia.

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Indications for PCI of an Infarct Artery in Patients Who Were Managed With Fibrinolytic Therapy or Who Did Not Receive Reperfusion Therapy

	COR	LOE
Cardiogenic shock or acute severe HF	I	B
Intermediate- or high-risk findings on predischarge noninvasive ischemia testing	I	C
Spontaneous or easily provoked myocardial ischemia	I	C
Patients with evidence of failed reperfusion or reocclusion after fibrinolytic therapy (as soon as possible)	IIa	B
Stable* patients after successful fibrinolysis, ideally between 3 and 24 h	IIa	B
Stable* patients >24 h after successful fibrinolysis	IIb	B
Delayed PCI of a totally occluded infarct artery >24 h after STEMI in stable patients	III: No Benefit	B

*Although individual circumstances will vary, clinical stability is defined by the absence of low output, hypotension, persistent tachycardia, apparent shock, high-grade ventricular or symptomatic supraventricular tachyarrhythmias, and spontaneous recurrent ischemia.

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CABG in Patients With STEMI

I IIa IIb III

Urgent CABG is indicated in patients with STEMI and coronary anatomy not amenable to PCI who have ongoing or recurrent ischemia, cardiogenic shock, severe HF, or other high-risk features.

I IIa IIb III

CABG is recommended in patients with STEMI at time of operative repair of mechanical defects.

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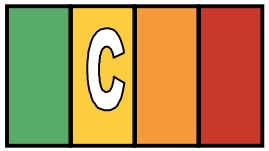
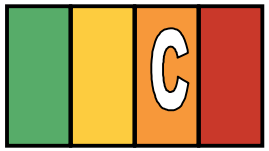
CABG in Patients With STEMI

I IIa IIb III



The use of mechanical circulatory support is reasonable in patients with STEMI who are hemodynamically unstable and require urgent CABG.

I IIa IIb III



Emergency CABG within 6 hours of symptom onset may be considered in patients with STEMI who do not have cardiogenic shock and are not candidates for PCI or fibrinolytic therapy.

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Complications After STEMI

Treatment of Cardiogenic Shock

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Treatment of Cardiogenic Shock

I IIa IIb III

Emergency revascularization with either PCI or CABG is recommended in suitable patients with cardiogenic shock due to pump failure after STEMI irrespective of the time delay from MI onset.

I IIa IIb III

In the absence of contraindications, fibrinolytic therapy should be administered to patients with STEMI and cardiogenic shock who are unsuitable candidates for either PCI or CABG.

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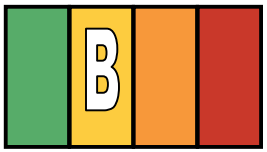
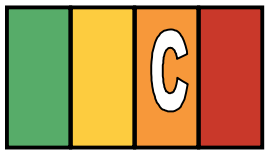
Treatment of Cardiogenic Shock

I IIa IIb III



The use of intra-aortic balloon pump counterpulsation can be useful for patients with cardiogenic shock after STEMI who do not quickly stabilize with pharmacological.

I IIa IIb III



Alternative LV assist devices for circulatory support may be considered in patients with refractory cardiogenic shock.

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PCI in Specific Clinical Situations: Cardiogenic Shock

PCI is recommended for patients with acute MI who develop cardiogenic shock and are suitable candidates.

A hemodynamic support device is recommended for patients with cardiogenic shock after STEMI who do not quickly stabilize with pharmacologic therapy.

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ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation

European Heart Journal (2012) 33, 2569–2619
doi:10.1093/eurheartj/ehs215

www.escardio.org/guidelines

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Primary PCI

Recommendations	Class	Level
Indications for primary PCI		
Primary PCI is the recommended reperfusion therapy over fibrinolysis if performed by an experienced team within 120 min of FMC.	I	A
Primary PCI is indicated for patients with severe acute heart failure or cardiogenic shock, unless the expected PCI related delay is excessive and the patient presents early after symptom onset.	I	B

FMC = first medical contacts; PCI = percutaneous coronary intervention.

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Procedural aspects of primary PCI

Recommendations	Class	Level
Procedural aspects of primary PCI		
Stenting is recommended (over balloon angioplasty alone) for primary PCI.	I	A
Primary PCI should be limited to the culprit vessel with the exception of cardiogenic shock and persistent ischaemia after PCI of the supposed culprit lesion.	IIa	B
If performed by an experienced radial operator, radial access should be preferred over femoral access.	IIa	B
If the patient has no contraindications to prolonged DAPT (indication for oral anticoagulation, or estimated high long-term bleeding risk) and is likely to be compliant, DES should be preferred over BMS.	IIa	A
Routine thrombus aspiration should be considered.	IIa	B
Routine use of distal protection devices is not recommended.	III	C
Routine use of IABP (in patients without shock) is not recommended.	III	A

BMS = bare-metal stent; DAPT = dual antiplatelet therapy; DES = drug-eluting stent; IABP = intra-aortic balloon pump; PCI = percutaneous coronary intervention.

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European Heart Journal 2012 - doi:10.1093/eurheartj/ehs215

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Treatment of cardiogenic shock (Killip class IV)

Recommendations	Class	Level
Oxygen/mechanical respiratory support is indicated according to blood gasses.	I	C
Urgent echocardiography/Doppler must be performed to detect mechanical complications, assess systolic function and loading conditions.	I	C
High-risk patients must be transferred early to tertiary centres.	I	C
Emergency revascularization with either PCI or CABG in suitable patients must be considered.	I	B
Fibrinolysis should be considered if revascularization is unavailable.	IIa	C
Intra-aortic balloon pumping may be considered.	IIb	B
LV assist devices may be considered for circulatory support in patients in refractory shock.	IIb	C
Haemodynamic assessment with balloon floating catheter may be considered.	IIb	B
Inotropic/vasopressor agents should be considered:		
• Dopamine;	IIa	C
• Dobutamine;	IIa	C
• Norepinephrine (preferred over dopamine when blood pressure is low).	IIb	B

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VIEWPOINT

Post-Myocardial Infarction Cardiogenic Shock Is a Systemic Illness in Need of Systemic Treatment

Is Therapeutic Hypothermia One Possibility?

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Early observations of cardiogenic shock as a systemic clinical syndrome were first described in 1942. Today, cardiogenic shock remains the leading cause of death among patients hospitalized for myocardial infarction (MI). Mortality rates in post-MI cardiogenic shock approach 50% despite rapid revascularization, optimal medical care, and use of mechanical support. New therapeutic strategies with global systemic effects may offer advances in treatment and outcome in post-MI cardiogenic shock. Therapeutic hypothermia for post-MI cardiogenic shock has multiple potentially beneficial physiologic effects, including the potential to improve post-ischemic cardiac function and hemodynamics, decrease myocardial damage, and reduce end-organ injury from prolonged hypoperfusion. Available data in animal models of post-MI cardiogenic shock and ischemia/reperfusion injury and small case series of human patients with cardiogenic shock suggest its promise as a potential therapeutic strategy for cardiogenic shock in the post-MI setting. We hypothesize that systemic therapeutic hypothermia could decrease morbidity and mortality in post-MI patients with cardiogenic shock and warrants study a new treatment that could be widely available at hospitals worldwide. (J Am Coll Cardiol 2012;59:644–7)

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My ideal approach to a STEMI Cardiogenic Shock

- Emergency angiography and revascularisation: Primary PCI preferably
- On-table echo to rule out mechanical defects
- Stabilise the patient in the lab before revascularisation
  - IABP
  - Pressors if required (Norepinephrine/dopamine)
  - Anaesthetic support
- Consider calling the surgeon for true surgical disease
- PCI of culprit artery. Other vessels if shock persists
- Use abciximab for PCI
- Consider percutaneous LVAD if shock persists with IABP + multi-vessel revascularisation

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Conclusions

- CS is a treatable illness with a reasonable chance for full recovery.
- The CS literature has traditionally focused on the very high mortality associated with this diagnosis.
- It is important to recognize that although patients with CS are at very high risk for early death, great potential exists for salvage.
- Recent evidences challenges the notion that patients with CS are a “lost cause.”

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