Cardiogenic Shock

Carlos Cafri, MD

SHOCK= Inadequate Tissue Perfusion

- **Mechanisms:**
  - **Inadequate oxygen delivery**
  - **Release of inflammatory mediators**
  - **Further microvascular changes, compromised blood flow and further cellular hypoperfusion**

- **Clinical Manifestations:**
  - **Multiple organ failure**
  - **Hypotension**

Differentiating Types of Shock

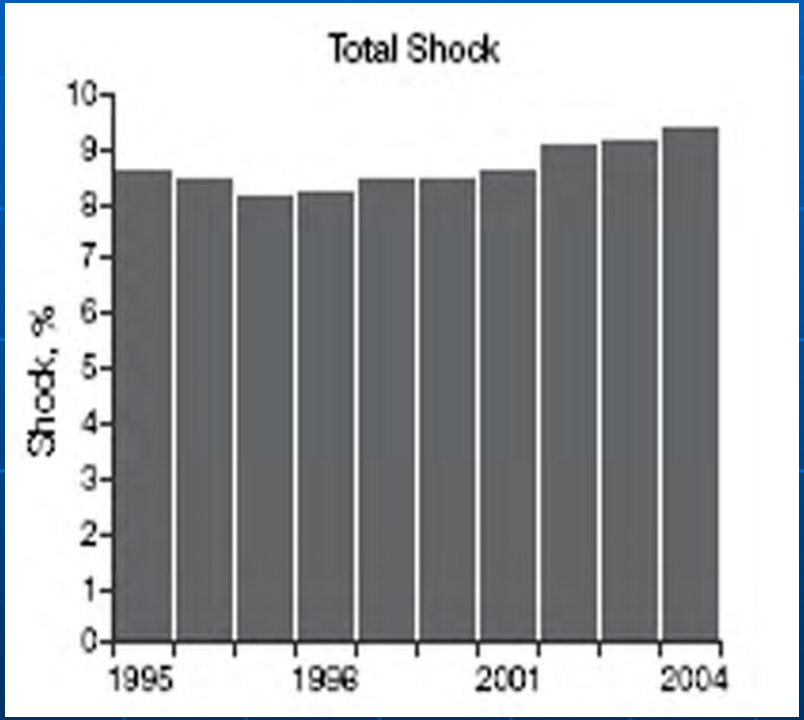
Physiologic variable	Preload	Pump function	Afterload	Tissue perfusion
Clinical measurement	Pulmonary capillary wedge pressure	Cardiac output	Systemic vascular resistance	Mixed venous oxygen saturation
Hypovolemic	↓	↓	↑	↓
Cardiogenic	↑	↓	↑	↓
Distributive	↓ or ↔	↑	↓	↑

Backgound

- Cardiogenic shock (CS) is a state of inadequate tissue perfusion due to cardiac dysfunction, and complicates 7-10% of cases of acute myocardial infarction
- Without treatment, cardiogenic shock is associated with a 70-80% mortality rate, and is the leading cause of death in patients hospitalized for an acute myocardial infarction
- Proper recognition and management of patients who develop cardiogenic shock will result in substantial improvements in early and late mortality

Frequency of CS Has Remained Steady Over Time

Frequency of Cardiogenic Shock



NRMI STEMI Registry[1]
N=25,311

NRMI Registry[1]

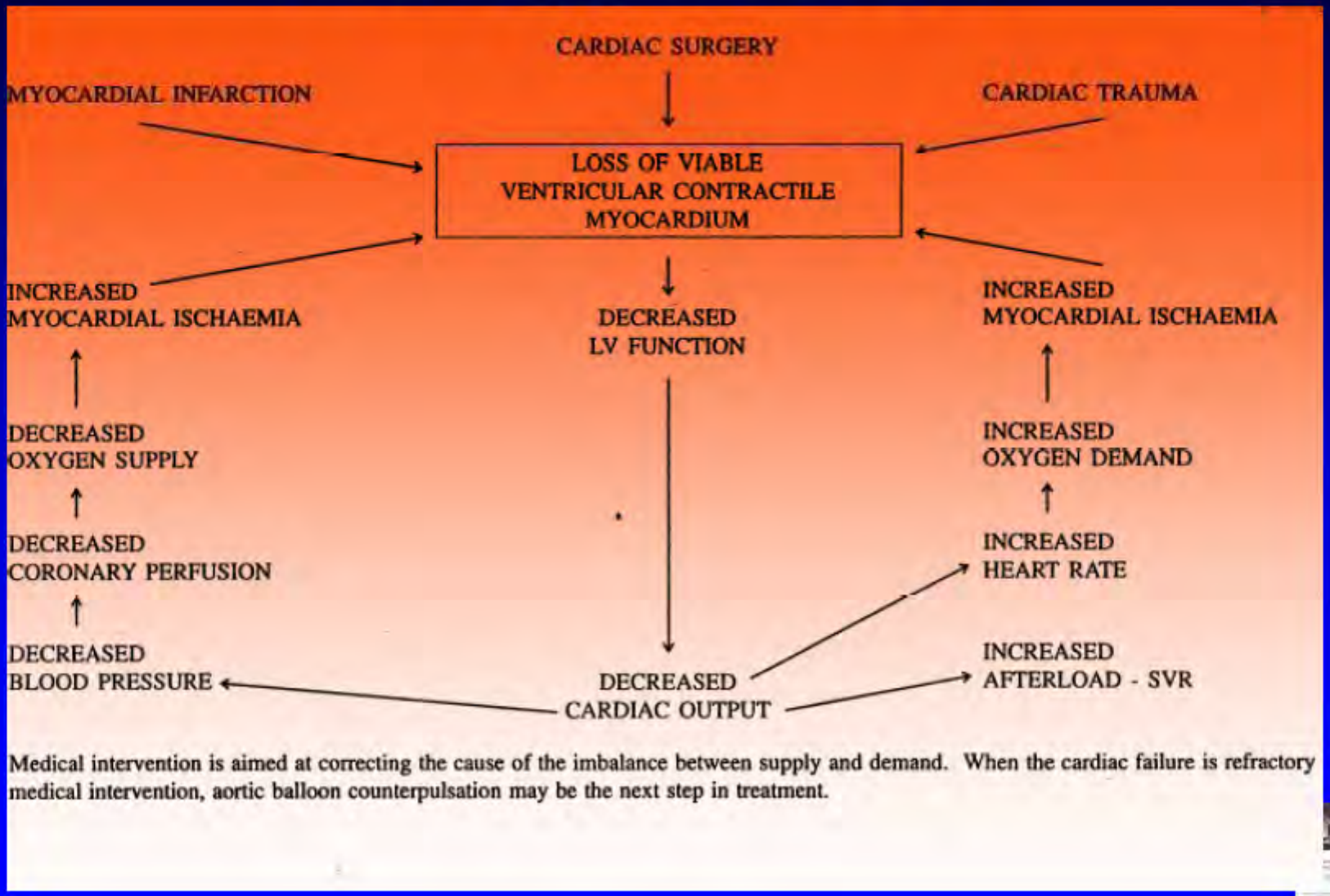
- Inclusion of 293,633 patients from Jan 1995-May 2004 with STEMI or new LBBB
- 775 US Hospitals with on-site PCI
- CS developed in 25,311 (8.6%) pts
- CS present on admission in 29%

Gusto-1[3]

- 1995 → 7.2%

[1]Babaev et al JAMA 2005 294:448
[2]Goldberg RJ NEJM 1991; 325:111
[3] Holmes DR JACC 1995 26:668

Cardiogenic Shock



Medical intervention is aimed at correcting the cause of the imbalance between supply and demand. When the cardiac failure is refractory medical intervention, aortic balloon counterpulsation may be the next step in treatment.

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Reversible Myocardial Dysfunction

- Myocardial stunning represents persistent myocardial dysfunction that occurs despite the restoration of normal flow. Develops as a result of alterations in calcium homeostasis, oxidative stress, and decreased myofilament responsiveness to calcium
- Hibernating myocaridum is a persistent state of myocardial dysfunction at rest because of severely reduced coronary flow. Develops as an adaptive response to hypoperfusion
- Both conditions may indicate recovery over time as reperfusion occurs

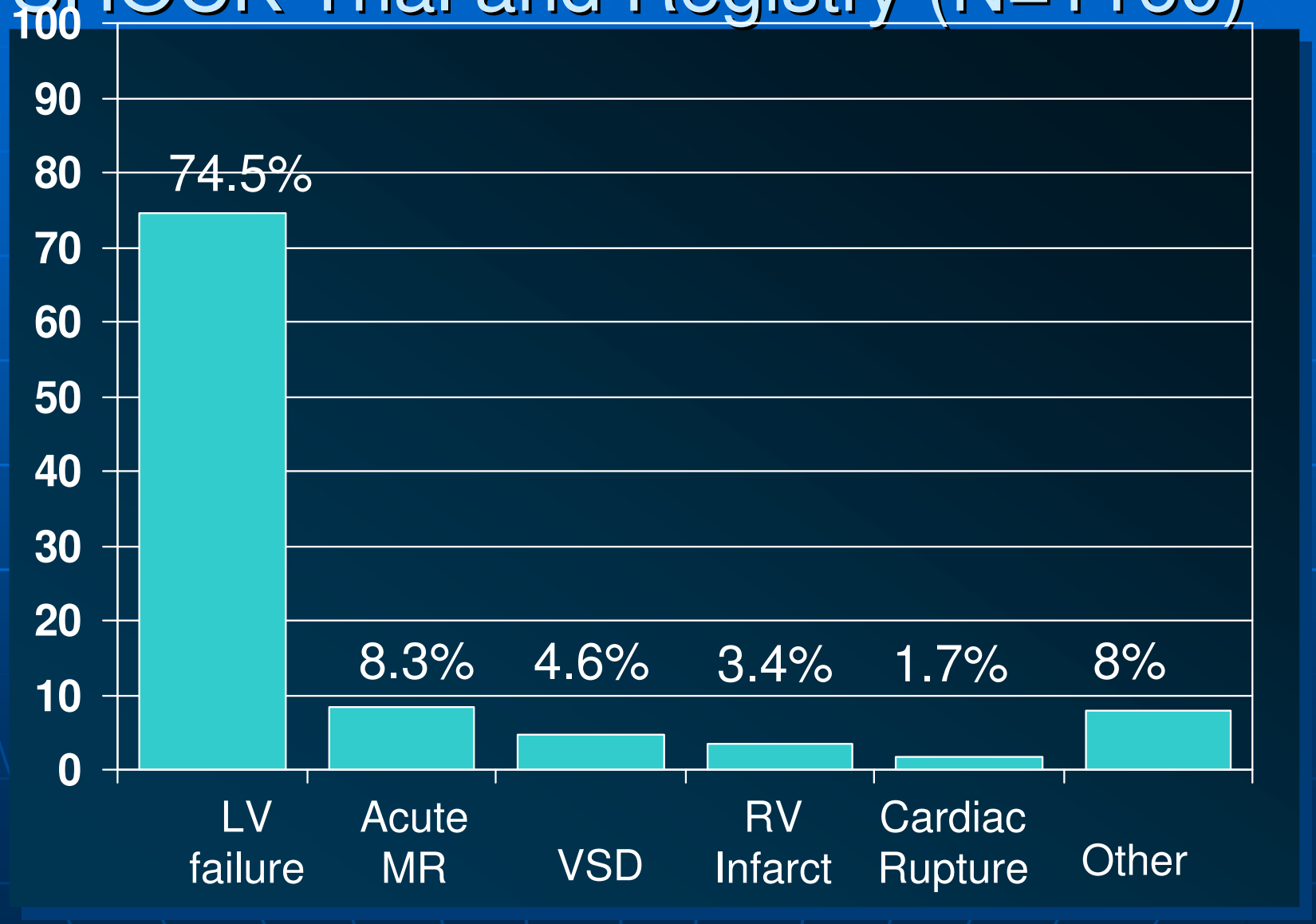
Hollenberg Ann Int Med 1999; 131:47-99

Etiology of Cardiogenic Shock

- Acute Myocardial Infarction (most common)
  - Pump Failure
    - Large infarction
    - Smaller infarctions with preexisting CHF
    - Infarction extension or expansion
  - Mechanical complications
    - Acute MR caused by papillary muscle dysfunction
    - Free wall rupture
    - Pericardial tamponade
- Other conditions
  - End-stage cardiomyopathy, myocarditis, prolonged cardiopulmonary bypass, aortic stenosis, mitral stenosis, left atrial mxyoma, acute aortic insufficiency

Causes of Cardiogenic Shock

SHOCK Trial and Registry (N=1160)



Hochman Circ 1995; 91:873-81

Shock onset after acute MI occurred within 24 h in 74% of the patients with predominant LV failure

Predictors of Early (< 24 h) Cardiogenic Shock

- Chest pain at shock onset
- ST-segment elevation in two or more leads
- Multiple infarct locations
- Inferior MI
- Left main disease
- Smoking

Predictors of Late (≥ 24 h) Cardiogenic Shock

- Recurrent ischemia,
- Q waves in ≥ 2 leads
- LAD culprit vessel

Webb JACC 2000; 36:1084

Clinical Observations from the SHOCK Trial

- The average LVEF is only moderately depressed (30%) with a wide range of EFs and LV sizes noted
  - While most patients were on IABP support and ionotropes, hemodynamic measurements demonstrated persistent hypotension, low CO, and high filling pressures despite a 30% LVEF
- The SVR was not markedly elevated in many cases, with the SVR ranging from 1350-1400 dynes-sec-cm^{-5} despite ionotropic support
  - Cardiac power = CI x MAP was the most powerful hemodynamic predictor of mortality
  - The ability to raise SVR may be an important compensatory mechanism to support BP
  - Endogenous/exogenous vasodilators inhibit this

Hochman Circulation 2003; 107:2998

Clinical Observations from the SHOCK Trial

- The classic notion that cardiogenic shock develops only when 40% of the myocardium is irreversibly damaged is inconsistent with:
  - 50% survival in PCI-treated patients
  - Improved LVEF in patients undergoing revascularization
  - NYHA Class I symptoms in 58% of patients after survival of the cardiogenic shock
- Resolution of the ischemia and neurohumeral-inflammatory mediates may result in resolution of the cardiogenic shock
- The range of LVEFs, LV size, and SVR in patients with cardiogenic shock indicate that the pathogenesis may be multifactorial.

Hochman Circulation 2003; 107:2998

Cardiogenic Shock: Diagnosis

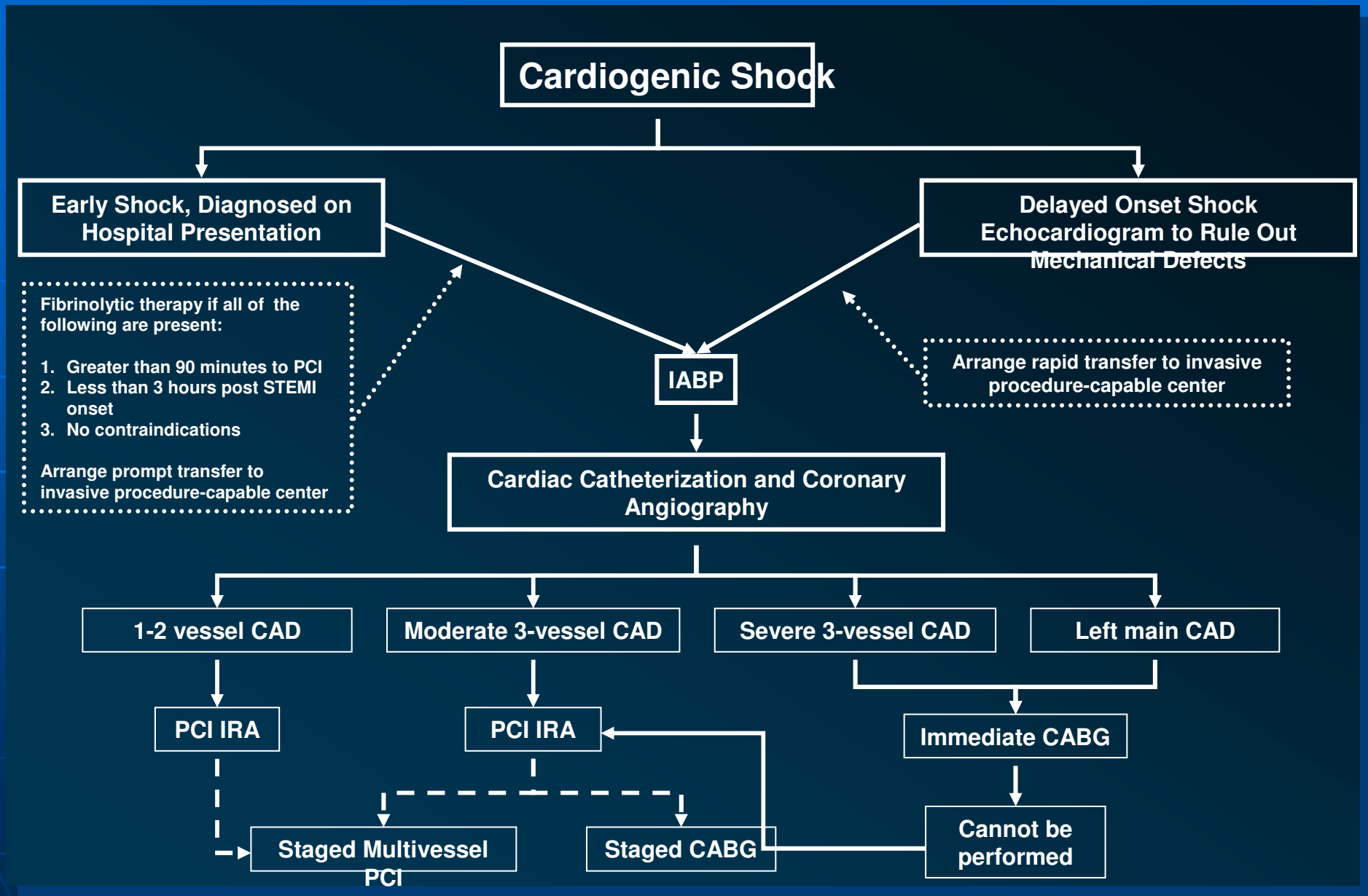
- Clinical definition[1] is a decreased cardiac output and evidence of tissue hypoperfusion in the presence of adequate filling pressures:
  - Marked and persistent (> 30 min) hypotension with a systolic BP < 90 mmHg
  - Reduction in the cardiac index (<2.2 L/min/M^2)
  - Normal or elevated PCWP (> 15 mmHg)
- Circulatory shock[2] is diagnosed by poor tissue perfusion, including oliguria, clouded sensorium, and cool mottled extremities

[2]Hollenberg Ann Int Med 1999; 131:47-99

Cardiogenic Shock

- Incidence
- Pathogenesis
- Diagnosis
- ✓ Treatment Options
  - Pharmacologic Treatment
  - PCI-CABG
    - The SHOCK Trial
  - Circulatory Support
- Prognosis
- ACC/AHA Guidelines
- Clinical Implications

PCI for Cardiogenic Shock



http:www.americanheart.org/stem

4 Potential Therapies

- Pressors
- Intra-aortic Balloon Pump (IABP)
- Fibrinolytics
- Revascularization: CABG/PCI

- Refractory shock: ventricular assist device, cardiac transplantation

Pressors do not change outcome

- ***Dopamine***
  - <2 renal vascular dilation
  - <2-10 +chronotropic/inotropic (beta effects)
  - >10 vasoconstriction (alpha effects)
- ***Dobutamine*** – positive inotrope, vasodilates, arrhythmogenic at higher doses
- ***Norepinephrine (Levophed):*** vasoconstriction, inotropic stimulant. Should only be used for refractory hypotension with dec SVR.

The SHOCK Trial (N=302)

Randomization from Apr 1993-Nov 1998

Emergency Revascularization
N = 152

- Angioplasty or CABG within 6 hours after randomization
- IABP recommended in all pts

Medical Therapy
N = 150

- IABP
- Thrombolytic Therapy
- Delayed Revascularization after 54 hours following randomization, if appropriate

- Primary Endpoint: Overall 30 day mortality
- Seconday Endpoints: 6 month and 1 year mortality

Hochman et al NEJM 1999;341:625

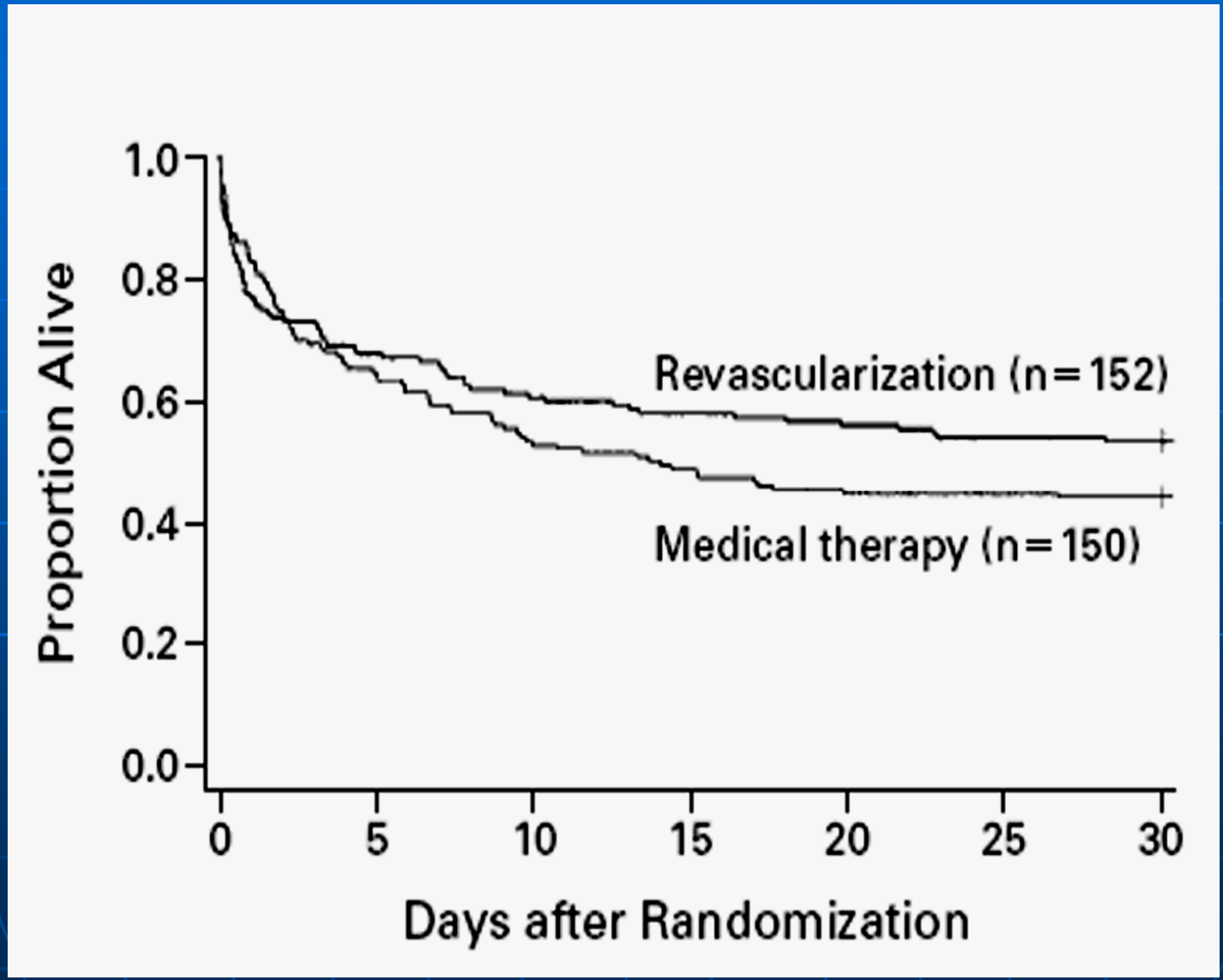
The Shock Trial:

Treatment

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 et al NEJM 1999;341:625

Shock Trial: 30 day mortality (1° Endpoint)



Hochman et al NEJM 1999;341:625

SHOCK trial

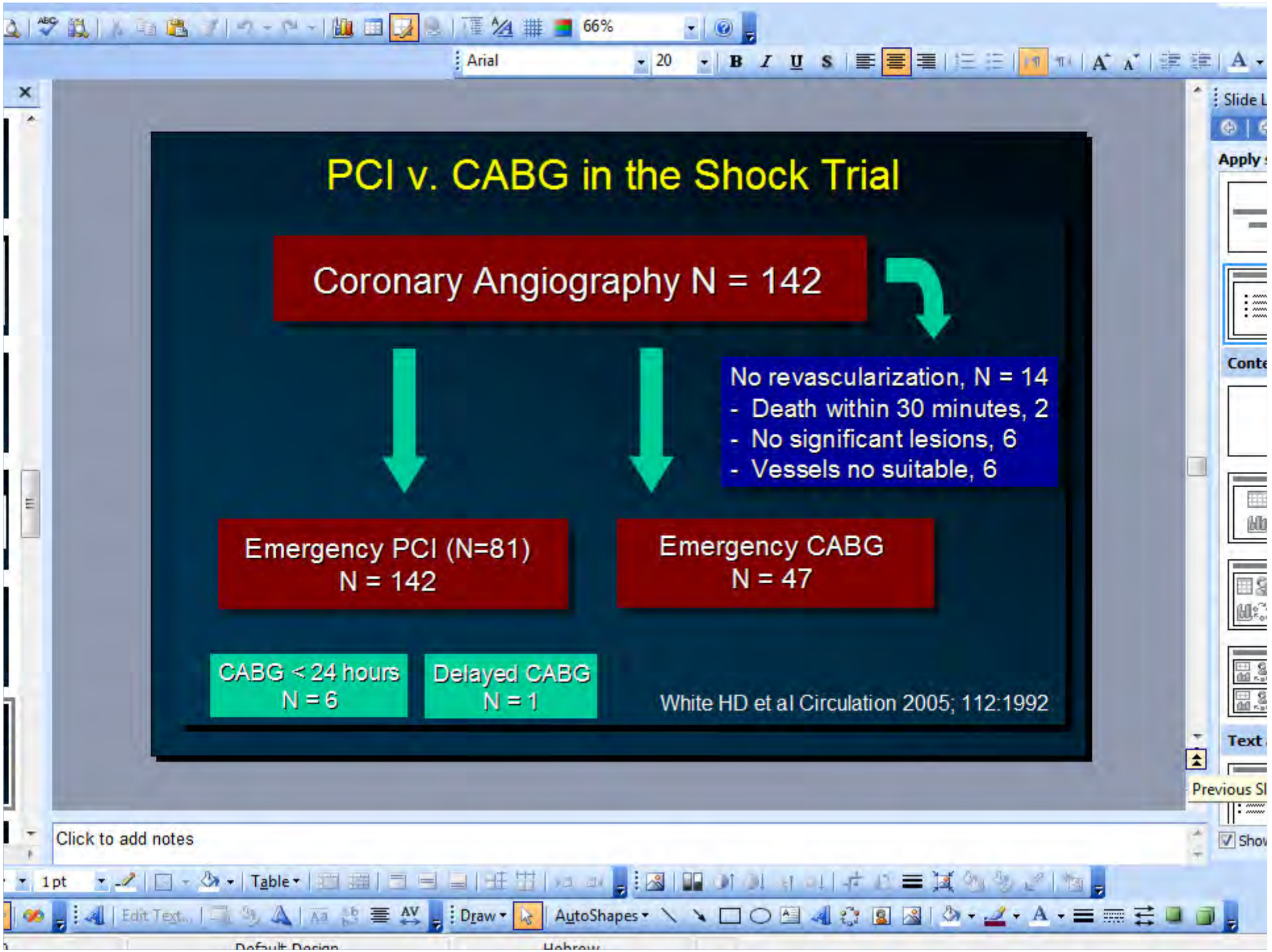
TABLE 4. MORTALITY AMONG STUDY PATIENTS.*

OUTCOME AND SUBGROUP	REVASCULARIZATION	MEDICAL THERAPY	DIFFERENCE BETWEEN GROUPS (95% CI)	RELATIVE RISK (95% CI)	P VALUE
	percent (number in subgroup)		percent		
30-day mortality					
Total	46.7 (152)	56.0 (150)	−9.3 (−20.5 to 1.9)	0.83 (0.67 to 1.04)	0.11
Age <75 yr	41.4 (128)	56.8 (118)	−15.4 (−27.8 to −3.0)	0.73 (0.56 to 0.95)	0.01†
Age ≥75 yr	75.0 (24)	53.1 (32)	+21.9 (−2.6 to 46.4)	1.41 (0.95 to 2.11)	
6-mo mortality‡					
Total	50.3 (151)	63.1 (149)	−12.8 (−23.2 to −0.9)	0.80 (0.65 to 0.98)	0.027
Age <75 yr	44.9 (127)	65.0 (117)	−20.1 (−31.6 to −7.1)	0.70 (0.56 to 0.89)	0.003†
Age ≥75 yr	79.2 (24)	56.3 (32)	+22.9 (0.7 to 46.6)	1.41 (0.97 to 2.03)	

*CI denotes confidence interval.

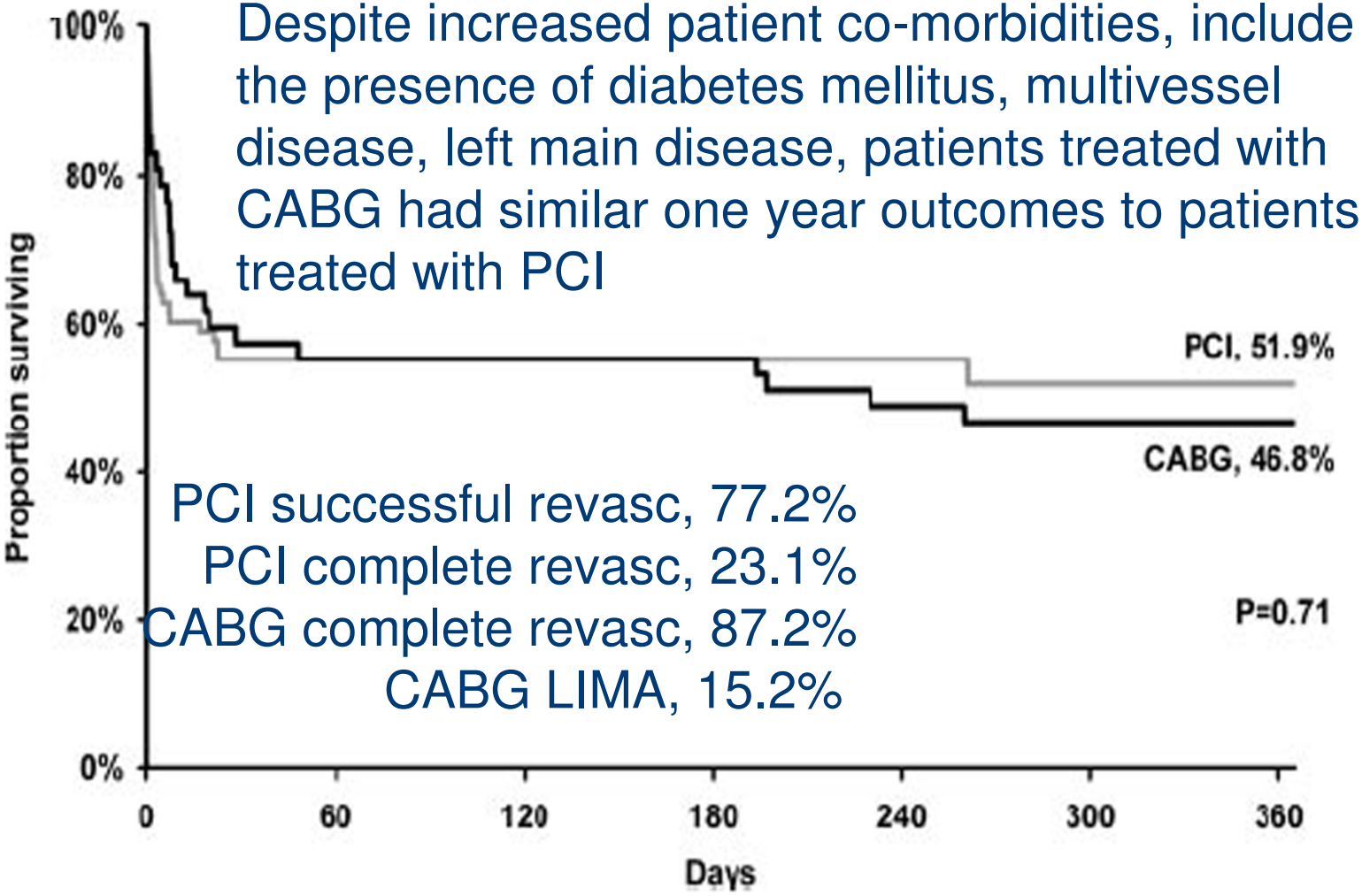
†Appropriate subgroup-analysis P values (for the interaction between treatment and the subgroup variable) are shown. Univariate P values for the comparison between treatments within subgroups were as follows: for 30-day mortality, P=0.02 for patients <75 years of age and P=0.16 for those ≥75 years of age; and for 6-month mortality, P=0.002 for patients <75 years of age and P=0.09 for those ≥75 years of age.

Hochman J et al. N Engl J Med 1999;341:625-634


PCI v. CABG in the Shock Trial
Coronary Angiography N = 142
No revascularization, N = 14
- Death within 30 minutes, 2
- No significant lesions, 6
- Vessels no suitable, 6
Emergency PCI (N=81)
N = 142
Emergency CABG
N = 47
CABG < 24 hours
N = 6
Delayed CABG
N = 1
White HD et al Circulation 2005; 112:1992
Click to add notes

PCI v. CABG in the Shock Trial

Despite increased patient co-morbidities, include the presence of diabetes mellitus, multivessel disease, left main disease, patients treated with CABG had similar one year outcomes to patients treated with PCI

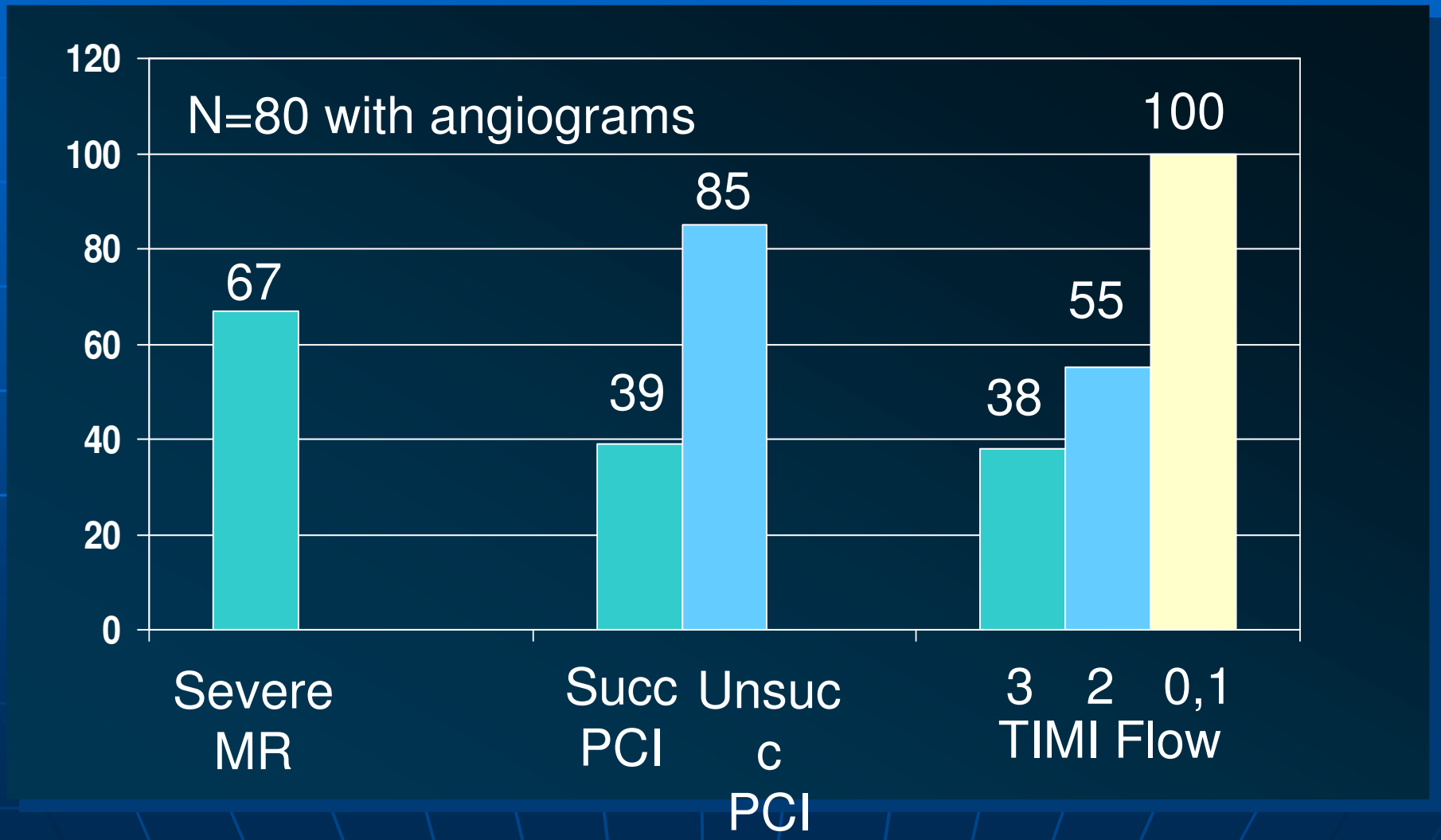


PCI successful revasc, 77.2%
PCI complete revasc, 23.1%
CABG complete revasc, 87.2%
CABG LIMA, 15.2%

White HD et al Circulation 2005; 112:199

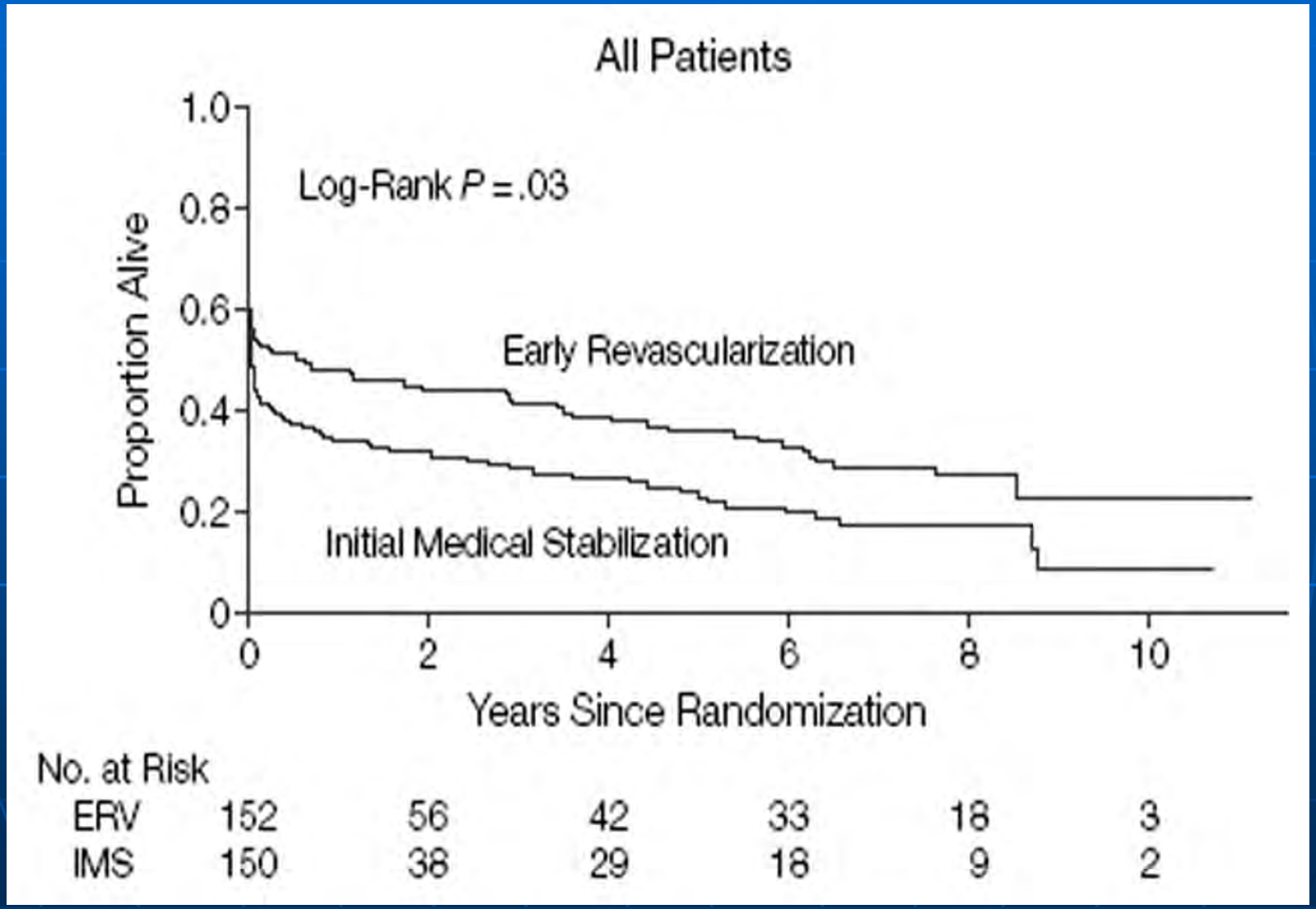
Shock Trial: Mortality Rates with PCI

Overall Mortality = 50%



Webb et al JACC 2003;42:138

6 Yr Outcome of SHOCK All Patients



Hochman et al JAMA 2006; 295:2511

ACC/AHA Guidelines for PCI in Patients with Cardiogenic Shock

Primary PCI is recommended for patients less than 75 years with ST elevation or LBBB or who develop shock within 36 hours of MI and are suitable for revascularization that can be performed within 18 hours of shock.

Primary PCI is reasonable for selected patients 75 years or older with ST elevation or LBBB or who develop shock within 36 hours of MI and are suitable for revascularization that can be performed within 18 hours of shock.

http:www.americanheart.org/stem

Management of Acute Ischemic Cardiogenic Shock

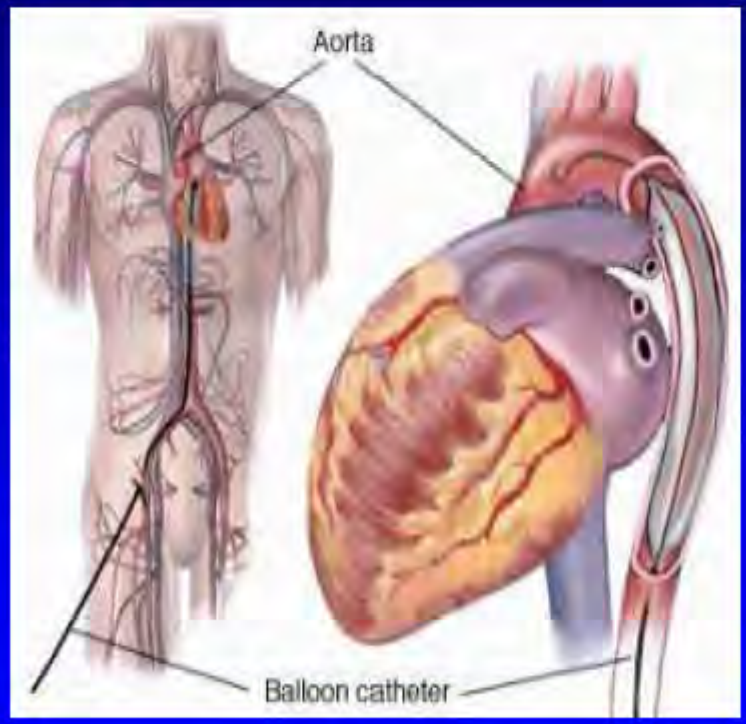
- Inotropic Support
- Circulatory Support
- Revascularisation !
- -> Target lesion + other relevant arteries

Topics for this talk

1. Balloon Counterpulsation
2. Results & Evidence
3. Guidelines
4. Assist Devices

Intra-Aortic Balloon Pump

- Inflatable 32-40 cc balloon
- Triggered to inflate with helium immediately after aortic valve closure
- Triggered to deflate with opening of the aortic valve



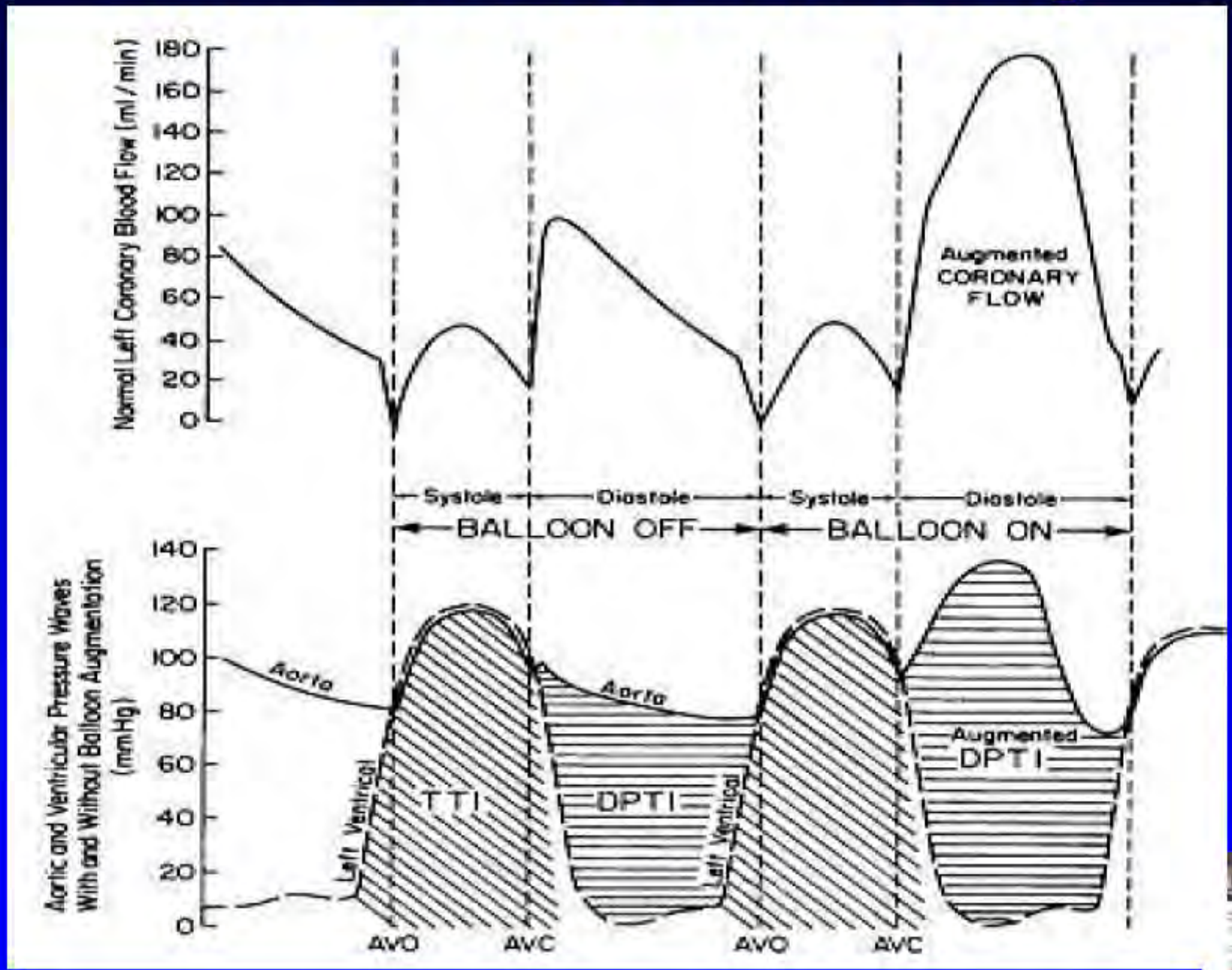
Insertion

- Sheathless or with sheath if scars/ fat
- Contralateral femoral artery
- Wire insertion
- Position balloon just distal to subclavian artery

Datascope Console

Understand the principles !

Intra-Aortic Balloon Pump



Intra-Aortic Balloon Pump

Decreases Afterload

Increases Diastolic Aortic Pressure

Increases Coronary Flow Velocity

Reduces Myocardial Oxygen Demand

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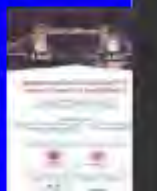
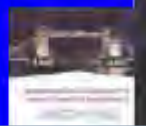
IABP Effects

- Mean pressure ↑
- Cardiac output ↑
- Cerebral perfusion ↑
- Renal perfusion ↑
- SVR ↓ -> peripheral perfusion ↑



Contraindications

- Severe Aortic Insufficiency
- Abdominal or Aortic Aneurysm
- Severe Aorto-Iliac Disease



Complications of IABP

- Vascular Complications
  - Limb ischemia
  - Dissection
  - Thrombus/Embolisation
- Infection

Circulatory Support

Balloon Counterpulsation

Results & Evidence

Guidelines

Assist Devices: Developments

Intra-Aortic Balloon Pump 'Current' Practice

Results from the Benchmark Registry

Ferguson et al. J Am Coll Cardiol 2001; 38:1456

Benchmark Registry: Indication

- Hemodynamic support during/after catheterisation 20.6%
- Cardiogenic shock 18.8%
- Weaning from CP bypass 16.1%
- Preoperative use in high risk pts 13%
- Refractory unstable angina 12.3%

Benchmark Registry: Complications

- Major: Limb ischemia, severe bleeding, balloon leak, death due to IABP 2.6%
- In –hospital mortality 21.2%
- Failed IABP insertion 2.3%
- Increased risk for major complications:
  - Women
  - Low BSA
  - Older patients
  - PVD

IABP Evidence

A prospective randomized evaluation of prophylactic intraaortic balloon counterpulsation in high risk patients with acute MI treated with primary angioplasty

Stone et al. J Am Coll Cardiol 1997

IABP in primary angioplasty

N:1100 Angio for MI

N: 908 randomised

N: 437 high risk

IABP 211 no IABP 226

Established 86% Crossover 13%

Stone et al. J Am Coll Cardiol

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IABP Evidence: SHOCK

Impact of thrombolysis, intra-aortic balloon pump counterpulsation, and their combination in cardiogenic shock complicating acute myocardial infarction

A report from the SHOCK trial registry

Sanborn et al. J Am Coll Cardiol 2000

SHOCK Result

IABP vs. no IABP mortality after adjustement for revascularisation p=0.313

Use of IABP with or without thrombolysis improves survival in pts with cardiogenic shock because of the higher rate of attempted revascularisation in the IABP group

IABP Trials

Elective versus provisional intraaortic balloon pumping in unprotected left main stenting.

Briguori C, Airoldi F, Chieffo A, Montorfano M, Carlino M, Sangiorgi GM, Morici N, Michev I, Iakovou I, Biondi-Zoccai G, Colombo A.

Am Heart J 2006; September

Provisional IABP for LMS

- N: 219 patients
- Preprocedural IABP: 69
- Conventional PCI: 150
- Severe hypotension & shock n:12 all in the conventional group

Circulatory Support

Balloon Counterpulsation

Results & Evidence

Guidelines

Assist Devices: Developments

ESC-GUIDELINES

Intra aortic balloon counterpulsation has become a standard component of treatment in patients with cardiogenic shock or severe acute left heart failure that

(i) do not respond rapidly to fluid administration, vasodilatatoin, and inotropic support

(ii) Is complicated by significant MR or rupture of the intraventricular septum, to obtain haemodynamic stabilisation for definitive diagnostic studies or treatment

(iii) Is accompanied by severe myocardial ischaemia in preparation for coronary angiography and revascularisation

AHA/ACC Guidelines

Recommendations for the use of IABP in the treatment of AMI

Class IIa

Signs of hemodynamic instability, poor LV , or persistent ischemia in patients with large areas of myocardium at risk

Class IIb
Following successful angioplasty to prevent reocclusion
Large areas at risk w/o active ischemia

Guidelines

"Emergency high risk PCI such as primary PCI for acute MI can usually be performed without IABP or CPS.

…

However, it should be noted that in patients with borderline hemodynamics, ongoing ischemia, or cardiogenic shock, insertion of an intra-aortic balloon just prior to coronary instrumentation has been associated with improved outcomes. Furthermore it is reasonable to obtain vascular access in the contralateral femoral artery prior to the procedure in patients in whom the risk of hemodynamic compromise is high…"

Summary IABP

Intra-Aortic Balloon Pump is an excellent tool for the management of hemodynamically unstable patients especially in the setting of acute MI

Indications

- Cardiogenic Shock
- Severe, refractory Ischemia
- Mechanical complications of MI
- Ischemia related intractable arrhythmias
- Support for high risk surgery
- Support for high risk PCI (?)