Cardiogenic Shock and Hemodynamics

Outline

- Overview of shock
  - Hemodynamic Parameters
  - PA catheter, complications
  - Differentiating Types of Shock
- Cardiogenic Shock
  - Etiologies
  - Pathophysiology
  - Clinical Findings
  - Treatment

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**

Hemodynamic Parameters

- Systemic Vascular Resistance (SVR)
- Cardiac Output (CO)
- Mixed Venous Oxygen Saturation (SvO2)
- Pulmonary Capillary Wedge Pressure (PCWP)
- Central Venous Pressure (CVP)

Normal Values

Right Atrial Pressure, CVP	Mean	0-6mmHg
Pulmonary Artery Pressure	Systolic End-diastolic mean	15-30mmHg 4-12mmHg 9-19mmHg
PCWP	Mean	4-12mmHg
Cardiac Output		4-8 L/min
Mixed Venous O2 Sat		>70%
SVR		800-1200

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 ↔	↑	↓	↑

PA Catheter Complications

- Path of PAC: central venous circulation → R heart→ pulmonary artery. The proximal port is in R atrium, distal port in pulm artery
- Arrhythmias
- RBBB
- PA rupture
- PAC related infection
- Pulmonary infarction

Cardiogenic Shock

- **Systemic hypoperfusion secondary to severe depression of cardiac output and sustained systolic arterial hypotension despite elevated filling pressures.**

Cardiogenic Shock

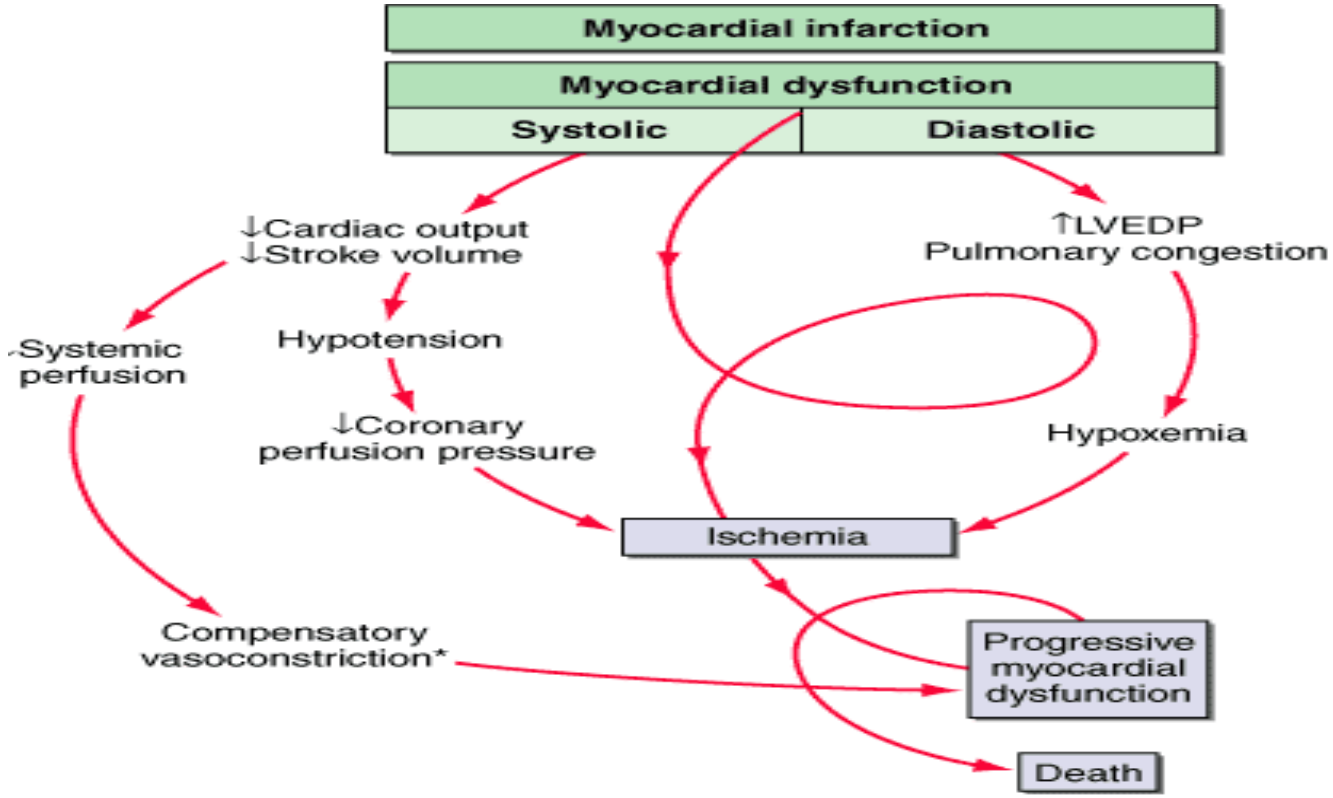
- Etiologies
- Pathophysiology
- Clinical/Hemodynamic Characteristics
- Treatment Options

Etiologies

- **Acute myocardial infarction/ischemia**
- **LV failure**
- **VSR (*Ventricular septal rupture)***
- **Papillary muscle/chordal rupture- severe MR**
- **Ventricular free wall rupture with subacute tamponade**

- **Other conditions complicating large MIs**
  - **Hemorrhage**
  - **Infection**
  - **Excess negative inotropic or vasodilator medications**
  - **Prior valvular heart disease**
  - **Hyperglycemia/ketoacidosis**
  - **Post-cardiac arrest**
  - **Post-cardiotomy**
  - **Refractory sustained tachyarrhythmias**
  - **Acute fulminant myocarditis**
  - **End-stage cardiomyopathyHypertrophic cardiomyopathy with severe outflow obstruction**
  - **Aortic dissection with aortic insufficiency or tamponade**
  - **Pulmonary embolu**
  - **Severe valvular heart disease - Critical aortic or mitral stenosis, Acute severe aortic or MR**

Pathophysiology



Clinical Findings

- **Physical Exam: elevated JVP, +S3, rales, oliguria, acute pulmonary edema**
- **Hemodynamics: dec CO, inc SVR, dec SvO2**
- **Initial evaluation: hemodynamics (PA catheter), echocardiography, angiography**

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.
- ***Vasopression*** – vasoconstriction

IABP is a temporizing measure

- Augments coronary blood flow in diastole
- Balloon collapse in systole creates a vacuum effect → decreases afterload
- Decrease myocardial oxygen demand

Indication for IABP

Class I - There is evidence and/or general agreement that an IABP should be used in patients with acute MI in the following settings
• Hypotension (systolic pressure less than 90 mmHg or ≥30 mmHg below the baseline mean arterial pressure) that does not respond to other interventions unless further support is limited by patient's wishes or contraindications or unsuitability for further invasive care.
• Low-output state
• Cardiogenic shock not quickly reversed with pharmacologic therapy as a stabilizing measure for angiography and prompt revascularization.
• Recurrent ischemic-type chest discomfort and hemodynamic instability, poor left ventricular function, or a large area of myocardium at risk. Such patients should be referred for urgent cardiac catheterization and, if appropriate, revascularization.
Class IIa - The weight of evidence or opinion is in favor of benefit from an IABP in patients with acute MI in the following setting
• Refractory polymorphic ventricular tachycardia in an attempt to diminish myocardial ischemia.
Class IIb - The evidence or opinion is less well established for an IABP in patients with acute MI in the following setting
• Refractory pulmonary congestion.

Contraindications to IABP

- Significant aortic regurgitation or significant arteriovenous shunting
- Abdominal aortic aneurysm or aortic dissection
- Uncontrolled sepsis
- Uncontrolled bleeding disorder
- Severe bilateral peripheral vascular disease
- Bilateral femoral popliteal bypass grafts for severe peripheral vascular disease.

Complications of IABP

- Cholesterol Embolization
- CVA
- Sepsis
- Balloon rupture
- Thrombocytopenia
- Hemolysis
- Groin Infection
- Peripheral Neuropathy

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)	

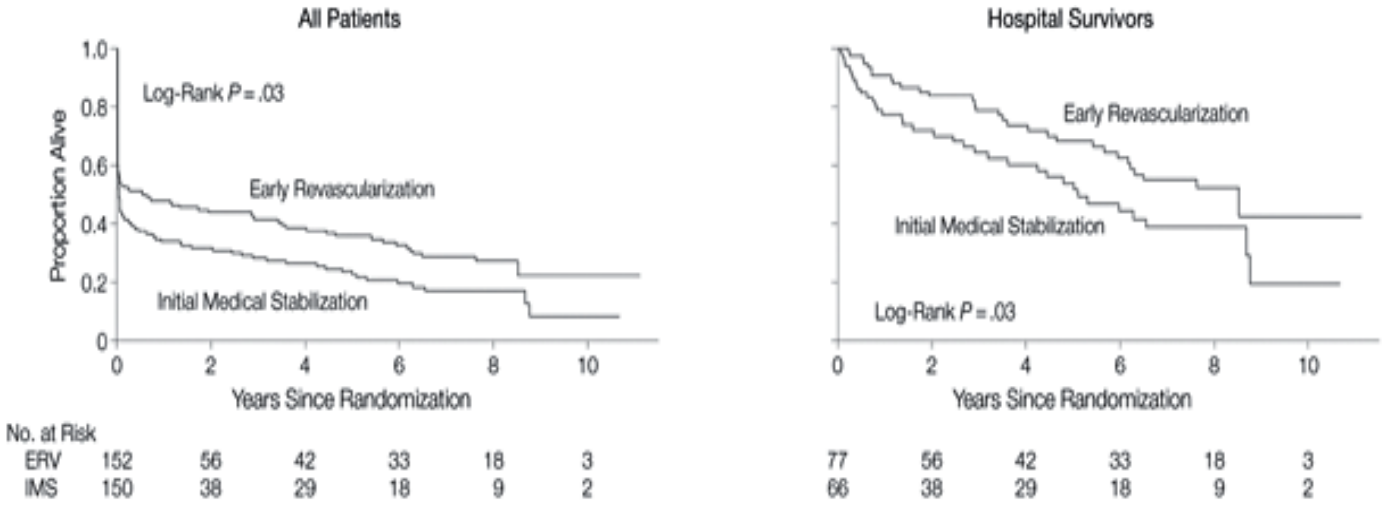
*CI denotes confidence interval.

†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

SHOCK – 6 years later

Kaplan-Meier Long-term Survival of All Patients and Those Discharged Alive Following Hospitalization



Hochman, J. S. et al. JAMA 2006;295:2511-2515.

JAMA

Question 1

1. **A 60-year-old man with a past medical history of hypercholesterolemia presents to the emergency department with 2 hours of crushing substernal chest pain radiating to his left arm, nausea, and diaphoresis. On examination, his blood pressure is 82/48 mm Hg, heart rate is 110 bpm, and oxygen saturation is 95% on 4 L of oxygen. He is in severe respiratory distress and has cold clammy extremities, an S_3 gallop, and bilateral crackles on auscultation. Electrocardiogram reveals ST elevation in the anterolateral leads and ST depression in the inferior leads. The patient is given aspirin, nitroglycerin, heparin, and intravenous fluids. Vasopressors are started to maintain blood pressure, but he remains hypotensive despite receiving 2 pressors. Which of the following is the most appropriate next step in management until the patient reaches the catheterization laboratory?**

   (A) Add a phosphodiesterase inhibitor
   (B) Initiate cardiac glycosides
   (C) Insert an intra-aortic balloon counterpulsation
   (D) More aggressive fluid resuscitation
   (E) Sodium nitroprusside infusion

Answer

(C) Insert an intra-aortic balloon counterpulsation. Intra-aortic balloon counterpulsation is recommended for patients with MI when cardiogenic shock is not quickly reversed with pharmacologic therapy.[1] It is also used as a stabilizing measure prior to angiography and prompt revascularization.[1] The synchronous deflation and inflation of the balloon during the cardiac cycle reduces afterload during systole and augments blood flow in coronary circulation during diastole, respectively.[2] In addition to their positive inotropic effect, phosphodiesterase inhibitors also have some vasodilatory properties and should not be used in patients with low mean arterial pressure. Nitroprusside also has a vasodilatory effect and should not be used in low cardiac output states. Aggressive fluid resuscitation may be limited by acute pulmonary edema from left ventricular dysfunction in patients with cardiogenic shock. Digoxin can be used in shock to control heart rate but only if atrial arrhythmias exist.

Question 2

2. Which of the following steps has been shown to have a mortality benefit in patients with cardiogenic shock caused by to myocardial infarction (MI)?

(A) Addition of glycoprotein IIb/IIIa inhibitors
(B) β-Adrenergic agonists
(C) Early cardiac catheterization followed by revascularization by percutaneous coronary intervention (PCI) or surgical revascularization
(D) Initial medical stabilization with blood pressure control prior to catheterization
(E) Thrombolytic infusion

Answer

(C) Early cardiac catheterization followed by revascularization by PCI or surgical revascularization. The SHOCK trial compared emergent revascularization for cardiogenic shock due to MI with initial medical stabilization and delayed revascularization.[3] The results of the study revealed a mortality benefit at 30 days that increased over time at 6 months and 1 year. The American College of Cardiology/American Heart Association guidelines recommend early revascularization (either PCI or coronary artery bypass grafting) for patients aged 75 years or younger with ST elevation or left bundle-branch block who develop shock within 36 hours of MI and who are suitable for revascularization that can be performed within 18 hours of shock.[1] Patients admitted to hospitals without facilities for revascularization should be immediately transferred to a tertiary care center with such facilities.

Question 3

A 65-year-old man with a history of type 2 diabetes presents to the emergency department with 4 hours of chest pain. Physical examination reveals a heart rate of 120 bpm and a systolic blood pressure of 62 mm Hg with a palpable pulse. Electrocardiogram reveals ST elevation in leads V_1 to V_4. The patient undergoes emergent cardiac catheterization followed by PCI. A pulmonary artery catheter is inserted for hemodynamic monitoring. Which of the following hemodynamic subsets satisfies the criteria for true cardiogenic shock in this patient?

(A) Pulmonary capillary wedge pressure (PCWP) < 18 mm Hg; cardiac index > 2.2
(B) PCWP > 18 mm Hg; cardiac index > 2.2
(C) PCWP < 18 mm Hg; cardiac index < 2.2
(D) PCWP > 18 mm Hg; cardiac index < 2.2

Answer

3. **(D) PCWP > 18 mm Hg; cardiac index < 2.2.** Patients with cardiogenic shock due to MI have low cardiac output due to left ventricular dysfunction, resulting in a low cardiac index. The left ventricle's inability to pump forward causes pooling of blood in the pulmonary circulation, resulting in high PCWP. Answer A is not consistent with shock, answer B represents congestive heart failure without shock, and answer C correlates with hypovolemic shock.[4]