Project: Ghana Emergency Medicine Collaborative

Document Title: Achy Breaky Heart: Cardiogenic Shock, A Historical Perspective and Current Therapy Guidelines

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Objectives

- Fulfill a requirement for graduation
- Present a case that we can all learn from
- Discuss the various treatment options available for cardiogenic shock
- Discuss what we can do in the ED to potentially increase survivability

Case Presentation

- CC: Chest pain, Shortness of breath
- HPI: 44 y.o. M unknown PMH, chest pain and SOB for 2 days. Worsening dyspnea. Brought in by family. Difficult to obtain history secondary to DIB and language barrier.

Vitals

- HR: 167
- BP: 89/64
- RR: 37
- SaO2: 99% NRB
- Temp: NR

Physical Exam

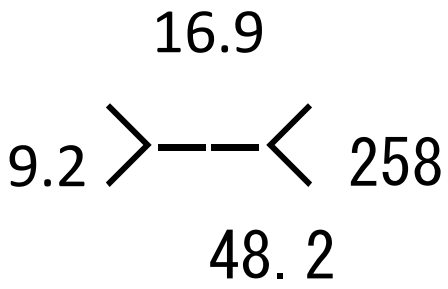
- General: Overweight gentleman, visibly short of breath, agitated, unable to sit still.
- Cardiovascular: Irregularly irregular. Tachycardic. No murmurs, rubs, or gallops appreciated. No JVD. Rapid but palpable radial pulses present.
- Pulmonary: Diffusely decreased air entry bilaterally with minimal wheezing noted.

Physical Exam

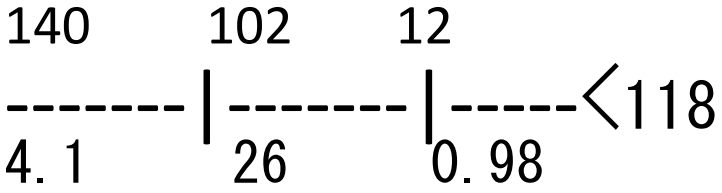
- Extremities: Warm, well-perfused. No evidence of lower extremity edema or swelling.
- Neurologic: Awake, alert, speaking to family members in 1-2 word sentences. Mostly nodding or shaking head to questions.

Lab work

CBC



Basic



Lab work

Myoglobin 163.7 ng/mL

Troponin 1.18 ng/mL

BNP 161 picogram/mL

D-dimer < 200 ng/mL

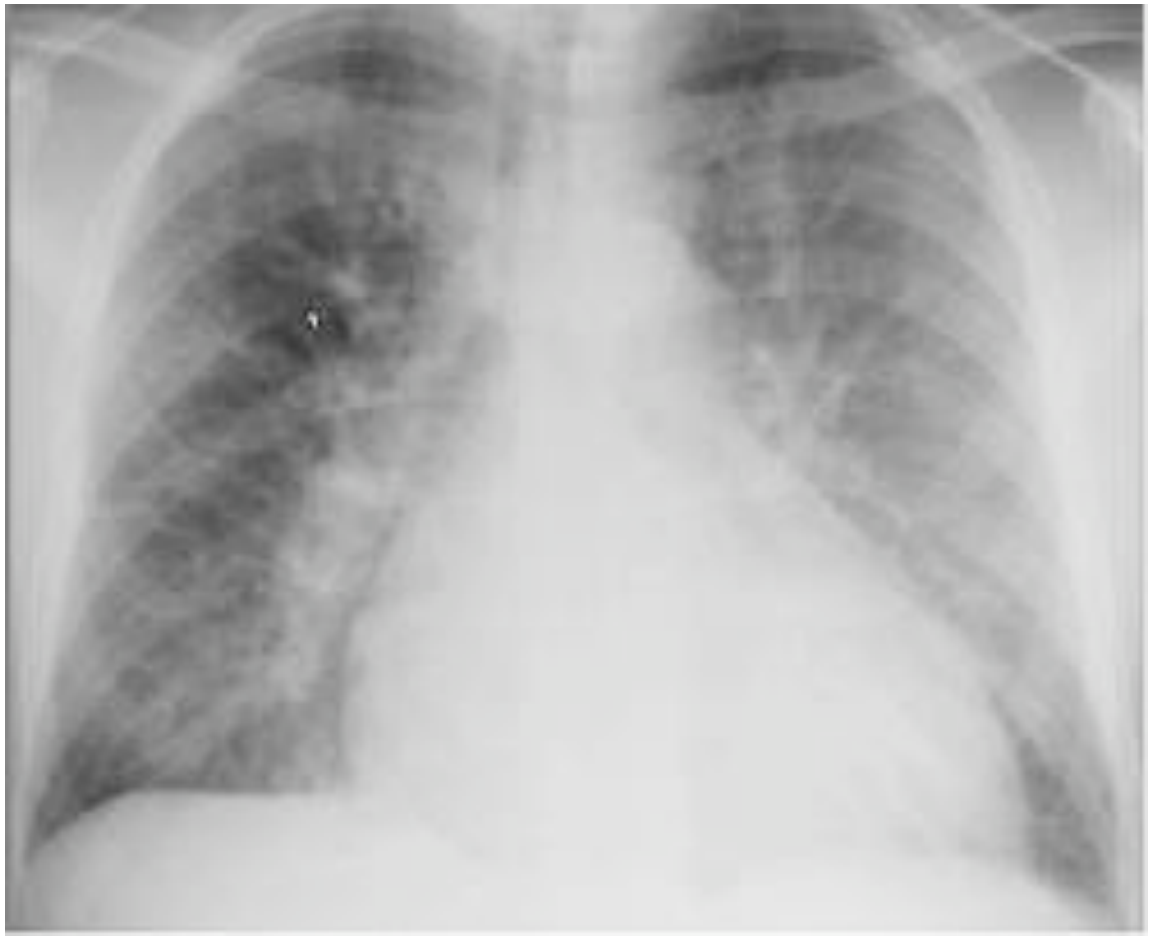
ABG: 7.31/42/304/21

Repeat: 7.21/51/168/20

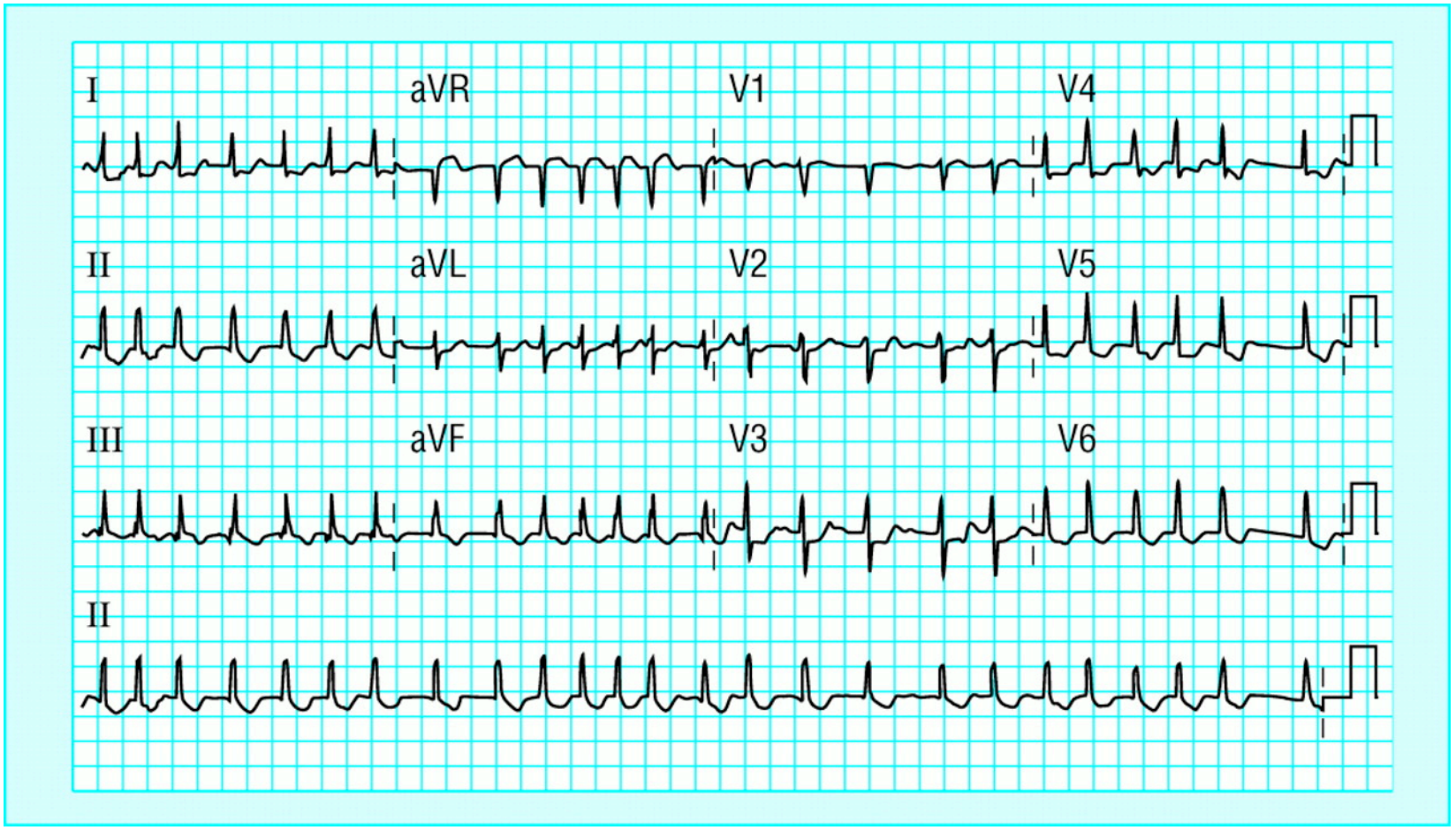
Therapies

- IV fluids
- Anti-arrhythmics
- Pressors
- BiPap
- Intubation
- Echocardiogram
- CT scan
- Cath lab

CXR



EKG



Phone a friend

a. Call your attending
b. Call the cardiologist
c. Call the cardiothoracic surgeon
d. Call your mother

Differential Diagnosis of Chest Pain and SOB

Differential Diagnoses (limited)

- MI
- Tension PTX
- Aortic dissection
- PE
- Cardiac tamponade
- Ruptured viscus
- Valvular abnormalities (mitral/aortic stenosis)

Some of the Many Causes of Cardiogenic Shock

- MI (most common)
- Aortic dissection
- PE
- Cardiac tamponade
- Ruptured viscus
- Hemorrhage
- Sepsis
- Cardiomyopathy (restrictive or dilated), myocarditis
- Medication overdose (beta/calcium-channel blockers)
- Cardiotoxic drugs (doxorubicin)
- Electrolyte abnormalities (calcium, phosphate)
- Valvular abnormalities (mitral/aortic stenosis)
- Papillary muscle or ventricular free wall rupture

A Lil' History

- 1700s: Shock first defined as a sequelae of severe trauma
- 1935, 1940: Harrison and Blalock classified types of shock
- 1950: Treatment of CS with O_2, phlebotomy, morphine. Also in favor was ethyl alcohol vapor, digitalis, quinidine
- 1960: Introduction of CCUs; improvement in mortality from arrhythmia, but not CS
- 1962: First IABP designed
- 1968: IABP placed by Dr. Kantrowitz in 5 patients with CS

Cardiogenic Shock

- 5-15% of ACS cases
- Small percentage with NSTEMI have CS (GUSTO II-B, PURSUIT trials)
- Loss of 40% of ventricular muscle mass
- Myocytes adjacent to infarct are susceptible to expanding ischemia

Risk Factors for Developing CS

- Older age
- Multivessel CAD
- Anterior MI location
- STEMI or LBBB
- HTN
- DM
- Prior MI
- Prior CHF

Diagnosing CS

- Clinically
  - SBP <90mmHg
  - HR >100 beats/min
  - RR >20 breaths/min ($Paco_2$<32 mm Hg)
  - Evidence of hypoperfusion

  - C.I <2.2L/min/m^2
  - LVEDP or PCWP >15mmHg

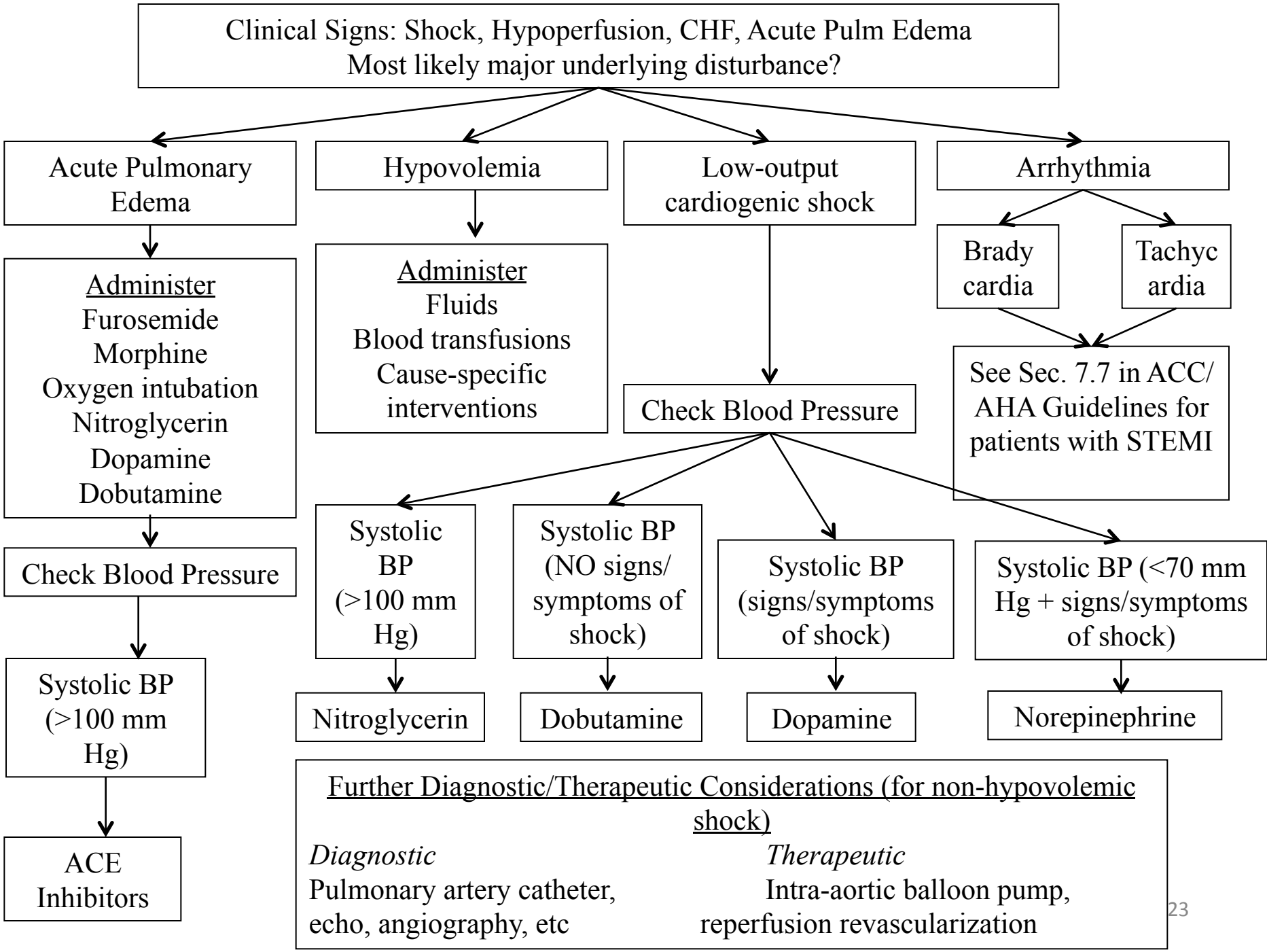
- Echocardiogram

Treatment for Cardiogenic Shock

- ABCs still take precedence
- 250-mL saline boluses over 5 to 10 minutes.
- Vasopressors or inotropic support
- Revascularization
- Consider IABP for refractory shock

Box 1. Conventional therapy for cardiogenic shock

Maximize volume (right atrial pressure 10 to 14 mm Hg, PCWP 18 to 20 mm Hg)
Maximize oxygenation (eg, ventilator)
Control rhythm (eg, pacemaker, cardioversion)
Correct electrolyte and acid–base imbalances
Sympathomimetic amines (eg, dobutamine, dopamine, norepinephrine, phenylephrine)
Phosphodiesterase inhibitors (eg, milrinone)
Vasodilators (eg, nitroglycerin, nitroprusside)
Diuretics (eg, furosemide)
Antiarrhythmics (eg, amiodarone)
Intra-aortic balloon counterpulsation


Clinical Signs: Shock, Hypoperfusion, CHF, Acute Pulm Edema
Most likely major underlying disturbance?
Acute Pulmonary Edema
Hypovolemia
Low-output cardiogenic shock
Arrhythmia
Brady cardia
Tachyc ardia
Administer
Furosemide
Morphine
Oxygen intubation
Nitroglycerin
Dopamine
Dobutamine
Administer
Fluids
Blood transfusions
Cause-specific interventions
See Sec. 7.7 in ACC/ AHA Guidelines for patients with STEMI
Check Blood Pressure
Check Blood Pressure
Systolic BP (>100 mm Hg)
Systolic BP (NO signs/ symptoms of shock)
Systolic BP (signs/symptoms of shock)
Systolic BP (<70 mm Hg + signs/symptoms of shock)
Systolic BP (>100 mm Hg)
Nitroglycerin
Dobutamine
Dopamine
Norepinephrine
ACE Inhibitors
Further Diagnostic/Therapeutic Considerations (for non-hypovolemic shock)
Diagnostic
Pulmonary artery catheter, echo, angiography, etc
Therapeutic
Intra-aortic balloon pump, reperfusion revascularization

Pharmacologic Treatment of Cardiogenic Shock

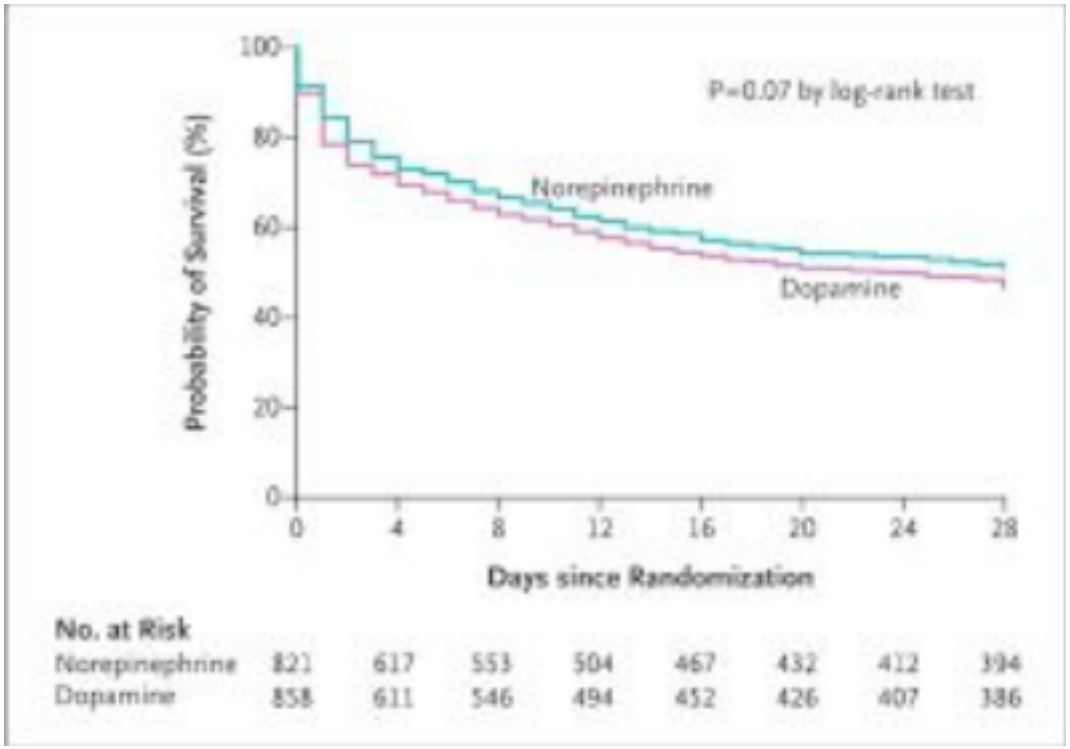
- SBP <70 mm Hg + shock
  →Norepinephrine
- SBP 70-100 mm Hg + shock
  →Dopamine
- SBP 70-100 mm Hg – shock
  →Dobutamine
- Refractory hypotension + shock
  →Amrinone or milrinone may improve cardiac output

The New England Journal of Medicine

- Multicenter, randomized, blinded study comparing Dopamine to Norepinephrine
- 1679 patients from 2003 – 2007
- Primary end point was rate of death at 28 days

The New England Journal of Medicine

- Multicenter, randomized, blinded study comparing Dopamine to Norepinephrine

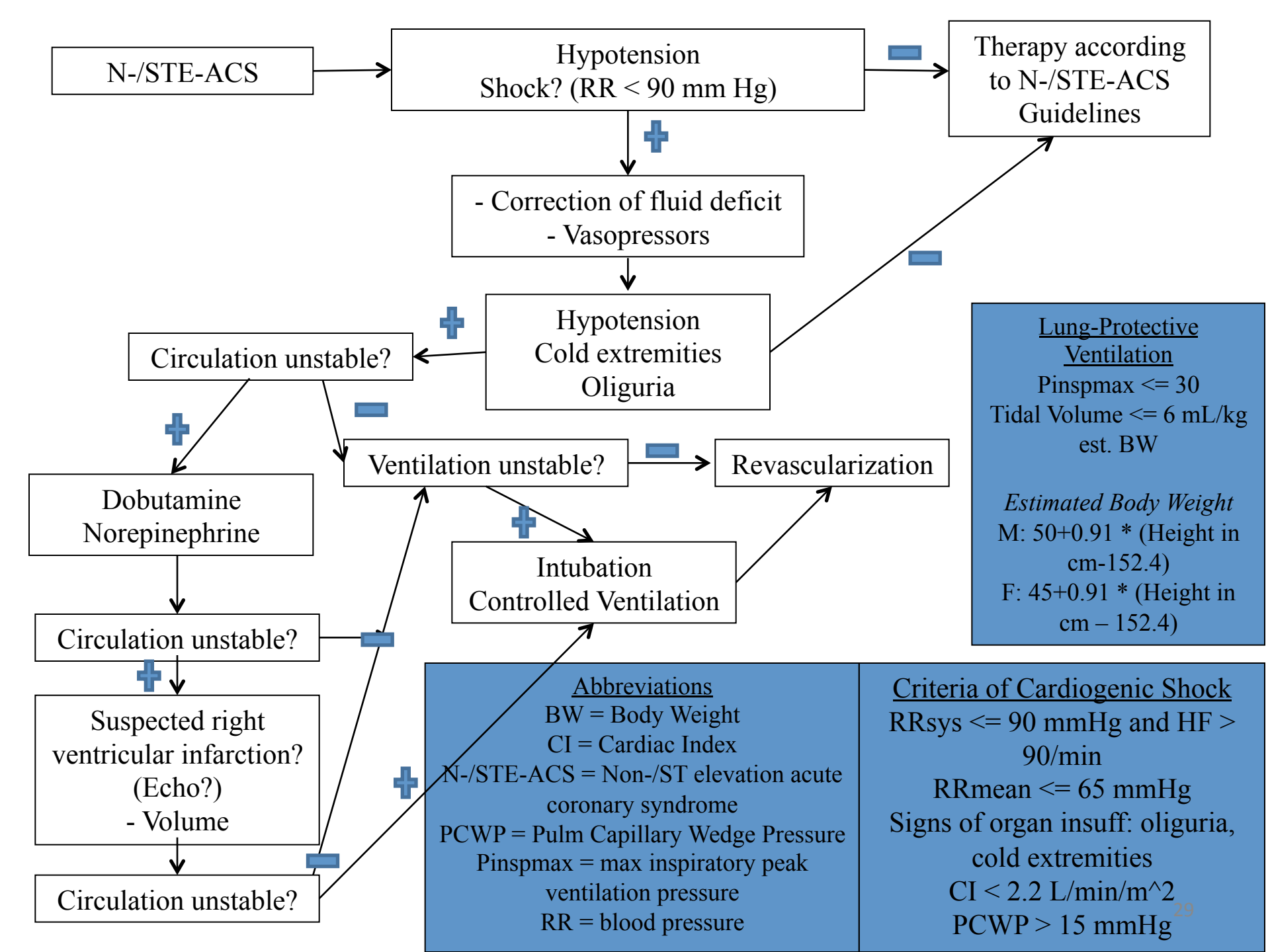


Levosimendan

- Novel inodilator; calcium-sensitizing agent
- Hemodynamic improvement
- The Survival of Patients with Acute Heart Failure In Need of Intravenous Inotropic Support (SURVIVE) trial.

Quick Review of ED Treatments

- Rapid assessment of history, PE, CXR
- Echo-Doppler to assess LV function, RV size, MVR, effusion, septal rupture
- Pressors/inotropes for hypotension
- ASA
- β-blockers and nitrates should be avoided in acute phase


N-/STE-ACS
Hypotension
Shock? (RR < 90 mm Hg)
Therapy according to N-/STE-ACS Guidelines
- Correction of fluid deficit
- Vasopressors
Hypotension
Cold extremities
Oliguria
Circulation unstable?
Ventilation unstable?
Revascularization
Dobutamine
Norepinephrine
Intubation
Controlled Ventilation
Circulation unstable?
Suspected right ventricular infarction? (Echo?)
- Volume
Circulation unstable?
Lung-Protective Ventilation
Pinspmax <= 30
Tidal Volume <= 6 mL/kg est. BW
Estimated Body Weight
M: 50+0.91 * (Height in cm-152.4)
F: 45+0.91 * (Height in cm – 152.4)
Abbreviations
BW = Body Weight
CI = Cardiac Index
N-/STE-ACS = Non-/ST elevation acute coronary syndrome
PCWP = Pulm Capillary Wedge Pressure
Pinspmax = max inspiratory peak ventilation pressure
RR = blood pressure
Criteria of Cardiogenic Shock
RRsys <= 90 mmHg and HF > 90/min
RRmean <= 65 mmHg
Signs of organ insuff: oliguria, cold extremities
CI < 2.2 L/min/m^2
PCWP > 15 mmHg

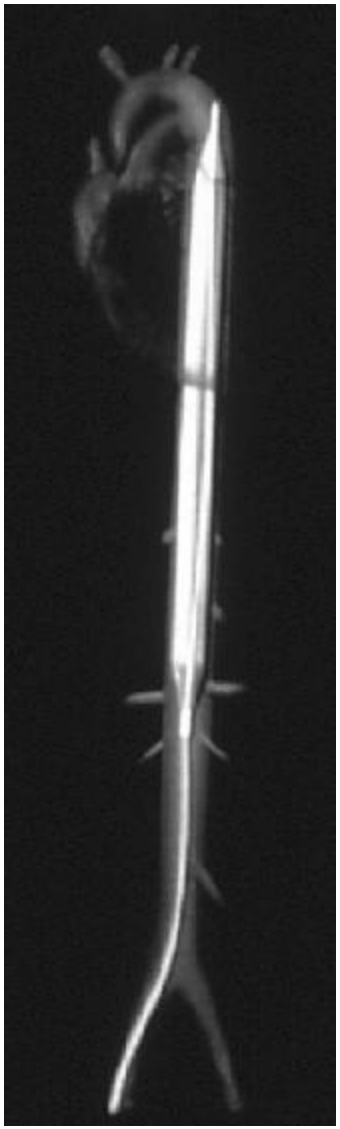
Therapies Beyond the ED

- IABP
- LVAD
- ECMO
- PCI
- CABG

Intra-Aortic Balloon Pump

- Increases coronary blood flow, decreases LV afterload and LV EDP without increasing O2 demand.
- Currently Class I recommendation for patients with low C.O. states, hypotension and CS not responding quickly to other measures.
- IABP-SHOCK II Trial

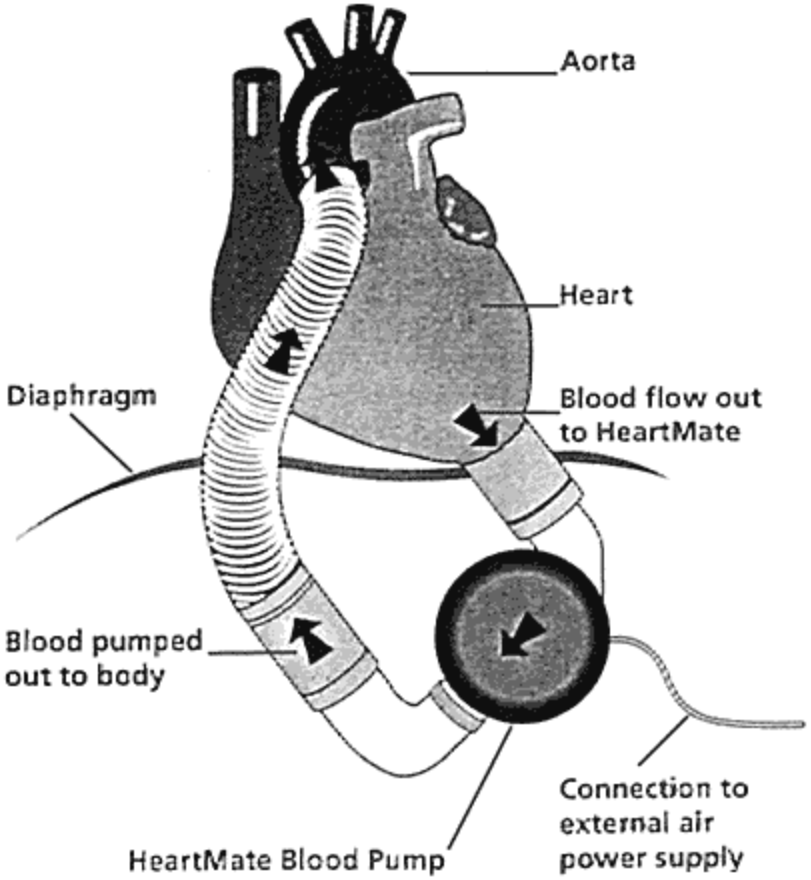
IABP



http://www.youtube.com/watch?v=o11fhdVOYWA&feature=player_detailpage

DSCP, Wikimedia Commons

Left Ventricular Assist Device



Steven M. Gordon, Centers for Disease Control, Wikimedia Commons

SHOCK Trial

- 1190 patients in SHOCK trial registry
- 60% mortality in CS
- Revascularization associated with decreased mortality

SHOCK Trial

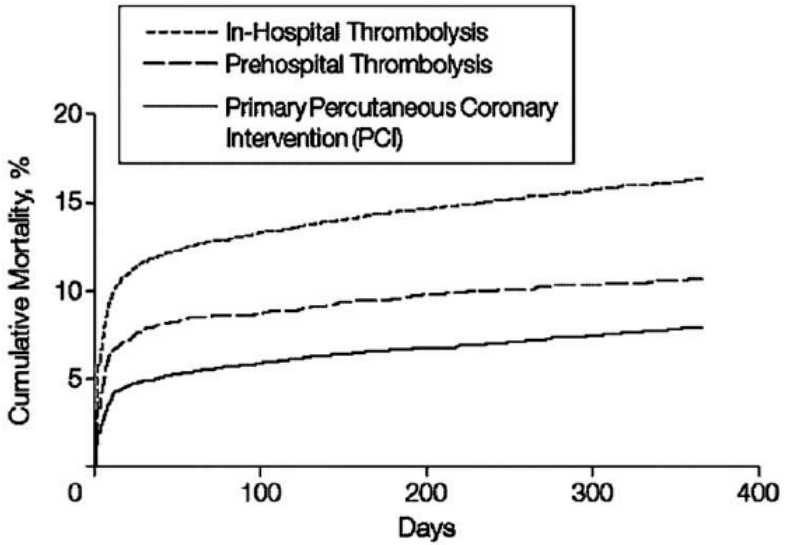
- Emergency revascularization neutralizes impact of CAD
- CABG performed in 39% of SHOCK trial patients; overall improved 1-year survival
- In presence of CS, LVEF, initial TIMI and culprit vessel were independent correlates of 1-year survival

GUSTO-1 Trial

- 41,021 from 15 countries
- Streptokinase vs. tPA
- tPA more efficacious than Streptokinase in preventing shock.
- However, if CS is already established, not as useful.

Fibrinolytics

- Fibrinolytic therapy not as effective in accomplishing reperfusion in STEMI with CS.
- Mortality benefit of IABP + thrombolytics is additive
- Still, IABP + thrombolytics worse than PCI or CABG



No. at Risk				
Thrombolysis				
In-Hospital	14260	12322	12100	11931
Prehospital	2736	2491	2460	2442
Primary PCI	6030	5661	5607	5555

Unadjusted mortality (Kaplan-Meier) first year after index admission for the 26205 patients with ST-segment elevation myocardial infarction receiving reperfusion therapy between 1999-2004.

Source Undetermined

Predictors of Death in CS (partial)

Table XII. Miscellaneous risk factors (without right heart catheterization)

Factor	Points
Killip class	
I	7
II	26
III	25
IV	0
Prior infarction	15
Altered sensorium	15
Cold, clammy skin	15
Oliguria	23
Ventricular-septal defect	38

Table X. Risk corresponding to total points (without right heart catheterization)

Points	Probability of 30-day mortality
103	10%
126	20%
141	30%
154	40%
165	50%
176	60%
189	70%
204	80%
227	90%

Source Undetermined

Failed therapies

- Tilarginine (NO synthase inhibitor) TRIUMPH trial, 2007 showed no survival benefit
- GIK (high-dose glucose, insulin, potassium)

Review Questions

Question #1

A 60y.o.m with PMH HLP presents to the ED with c/o 2 hours crushing substernal CP radiating to L arm, N/diaphoresis. BP 82/48 mmHg, HR 110 bpm, O2 95% 4L. Severe respiratory distress, cold clammy extremities, S3 gallop, bilateral crackles. EKG reveals STE in anterolateral leads and ST depression in inferior leads. Pt given ASA, nitroglycerin, heparin, IVF. Vasopressors started to maintain BP, but he remains hypotensive despite 2 pressors. Which of the following is the most appropriate next step in management until pt reaches cath lab?

- Add a phosphodiesterase inhibitor
- Initiate cardiac glycosides
- Insert an IABP
- More aggressive fluid resuscitation
- Sodium nitroprusside infusion

Review Questions

Question #1

A 60y.o.m with PMH HLP presents to the ED with c/o 2 hours crushing substernal CP radiating to L arm, N/diaphoresis. BP 82/48 mmHg, HR 110 bpm, O2 95% 4L. Severe respiratory distress, cold clammy extremities, S3 gallop, bilateral crackles. EKG reveals STE in anterolateral leads and ST depression in inferior leads. Pt given ASA, nitroglycerin, heparin, IVF. Vasopressors started to maintain BP, but he remains hypotensive despite 2 pressors. Which of the following is the most appropriate next step in management until pt reaches cath lab?

- Add a phosphodiesterase inhibitor
- Initiate cardiac glycosides
- **Insert an IABP**
- More aggressive fluid resuscitation
- Sodium nitroprusside infusion

Review Questions

- IABP is recommended for patients with MI when cardiogenic shock is not quickly reversed with pharmacologic therapy. Used as a stabilizing measure prior to angiography and prompt revascularization.
- Phosphodiesterase inhibitors 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.
- Digoxin can be used in shock to control HR but only if atrial arrhythmias exist.

Review Questions

Question #2

Which of the following steps has been shown to have a mortality benefit in patient with cardiogenic shock cause by MI?

- Addition of glycoprotein IIb/IIIa inhibitors
- B-adrenergic agonists
- Early cardiac cath followed by revascularization by PCI or surgical revascularization
- Initial medical stabilization with blood pressure control prior to catheterization
- Thrombolytic infusion

Review Questions

Question #2

Which of the following steps has been shown to have a mortality benefit in patient with cardiogenic shock cause by MI?

- Addition of glycoprotein IIb/IIIa inhibitors
- B-adrenergic agonists
- **Early cardiac cath followed by revascularization by PCI or surgical revascularization**
- Initial medical stabilization with blood pressure control prior to catheterization
- Thrombolytic infusion

Review Questions

- The SHOCK trial compared emergent revascularization for cardiogenic shock due to MI with initial medical stabilization and delayed revascularization. This showed a mortality benefit at 30 days that increased over time at 6 months an 1 year. The ACC/AHA recommend early revascularization for pts aged 75yrs or younger with STE or LBBB who develop shock within 36 hours of MI and suitable for revascularization that can be performed within 1 hours of shock.

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