CARDIOGENIC SHOCK

BAHRAM SHAHRI

MD, INTERVENTIONAL CARDIOLOGIST

ASSISTANT PROFESSOR OF MUMS

WHAT IS SHOCK?

- Shock is the "physiologic state characterized by significant reduction of systemic tissue perfusion, resulting in decreased tissue oxygen delivery."
- Tissue perfusion is dependent on systemic vascular resistance (SVR) and cardiac output (COP).
  - Imbalance between oxygen delivery and oxygen consumption which leads to cell death, end organ damage, multi-system organ failure, and death

THREE TYPES OF SHOCK

- Cardiogenic
- Hypovolemic
- Distributive
  - Septic
  - Anaphylactic
  - Neurogenic
- Combined

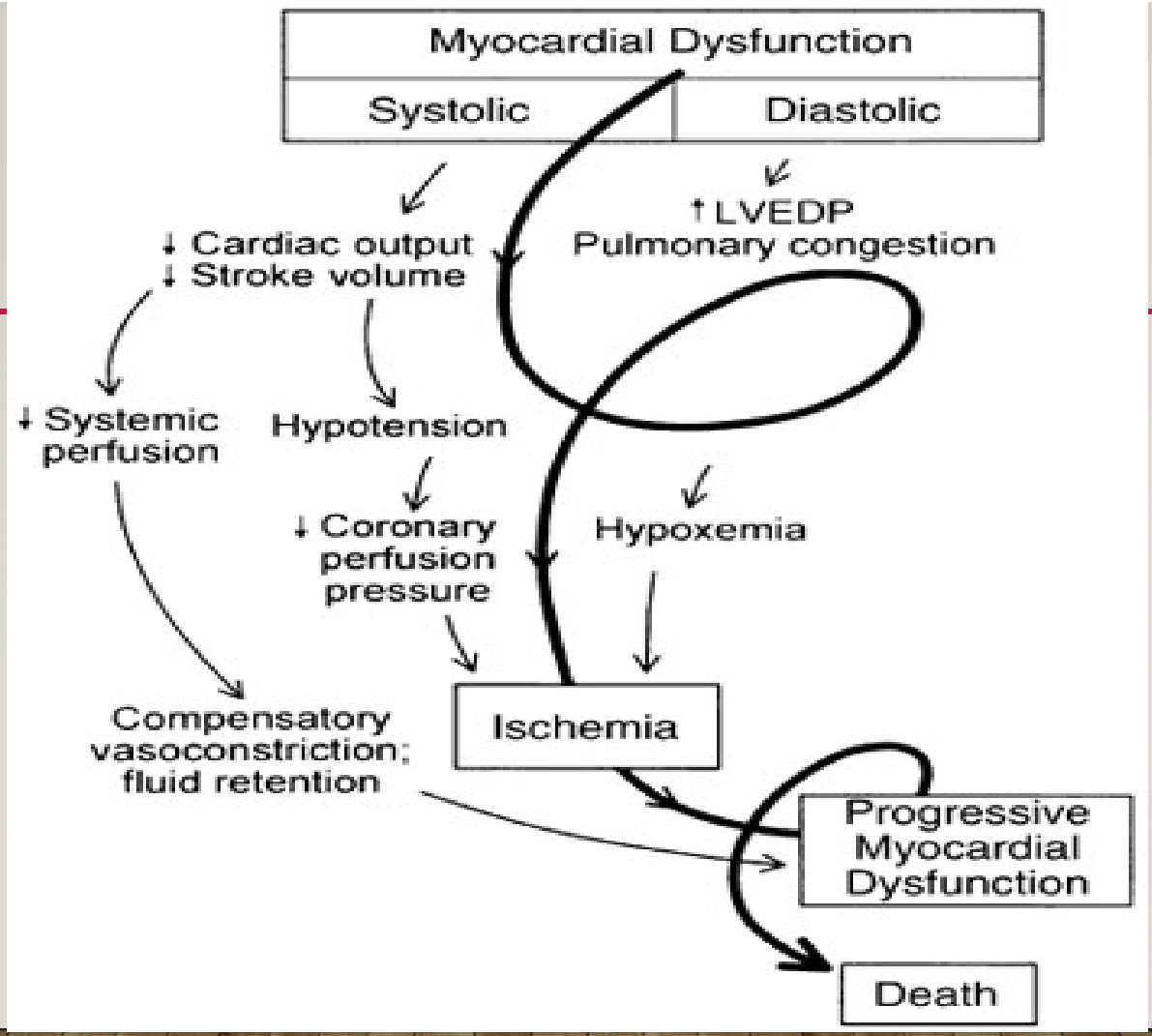
- Distributive Shock: variable cardiac output, decreased SVR
- Hypovolemic Shock: decreased effective circulating volume
- Obstructive Shock: circulatory failure caused by physical obstruction, e.g. cardiac tamponade or pulmonary embolism
- Cardiogenic Shock: decreased cardiac output, pump failure

CARDIOGENIC SHOCK

- Shock caused as a result of cardiac pump failure
  - Results in a decrease in COP
  - SVR is increased in an effort to compensate to maintain organ perfusion
- Inadequate tissue perfusion resulting from cardiac dysfunction
- Clinical definition: decreased CO and tissue hypoxia in the presence of adequate intravascular volume

DEFINITION OF CARDIOGENIC SHOCK

- SBP < 90 mm Hg for at least 30 minutes that is not responsive to fluid administration alone
- Secondary to cardiac dysfunction
- Associated with signs of hypoperfusion or a CI < 2.2 L/min/m2 and a PAWP > 15 mmg Hg


Myocardial Dysfunction
Systolic
Diastolic
↓ Cardiac output
↓ Stroke volume
↑ LVEDP
Pulmonary congestion
↓ Systemic perfusion
Hypotension
↓ Coronary perfusion pressure
Hypoxemia
Compensatory vasoconstriction; fluid retention
Ischemia
Progressive Myocardial Dysfunction
Death

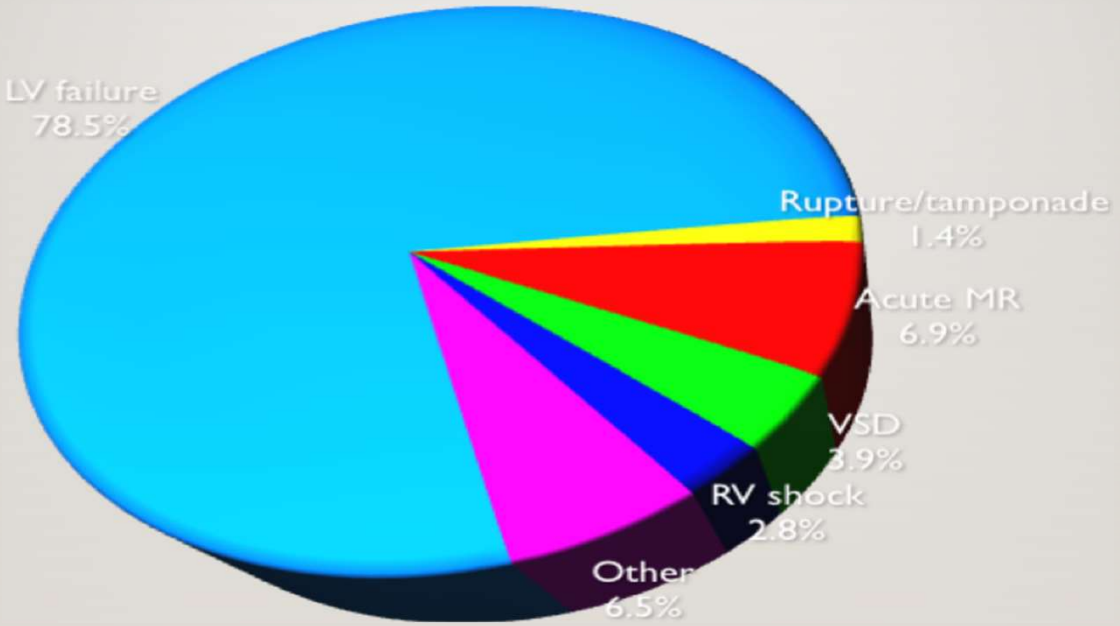
HOW TO IDENTIFY CARDIOGENIC SHOCK

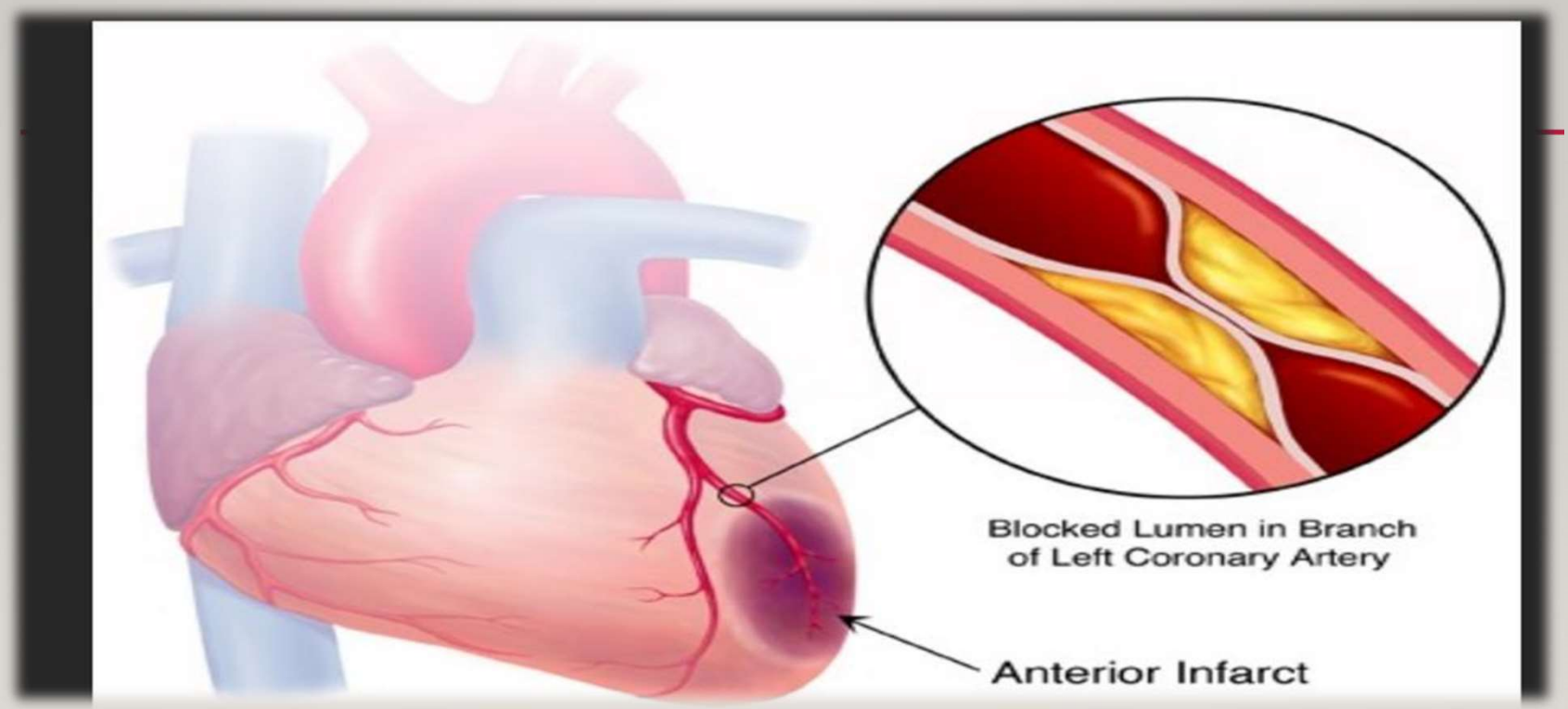
- History
- Physical Exam
- EKG
- Chest xray
- Echocardiogram
- Swan-Ganz Catheter

HISTORY: WHO GETS CARDIOGENIC SHOCK?

- Acute MI
  - Pump failure
  - Mechanical complications: VSD, Papillary septal rupture, free wall rupture and cardiac tamponade
  - Right ventricular infarction
- Other conditions
  - End-stage cardiomyopathy
  - Myocarditis
  - Myocardial contusion
  - Prolonged cardiopulmonary bypass
  - Septic shock with myocardial depression
  - Valvular disease: AS, AR, MS, MR

HISTORY: WHO GETS CARDIOGENIC SHOCK?




Blocked Lumen in Branch
of Left Coronary Artery
Anterior Infarct

PHYSICAL EXAM

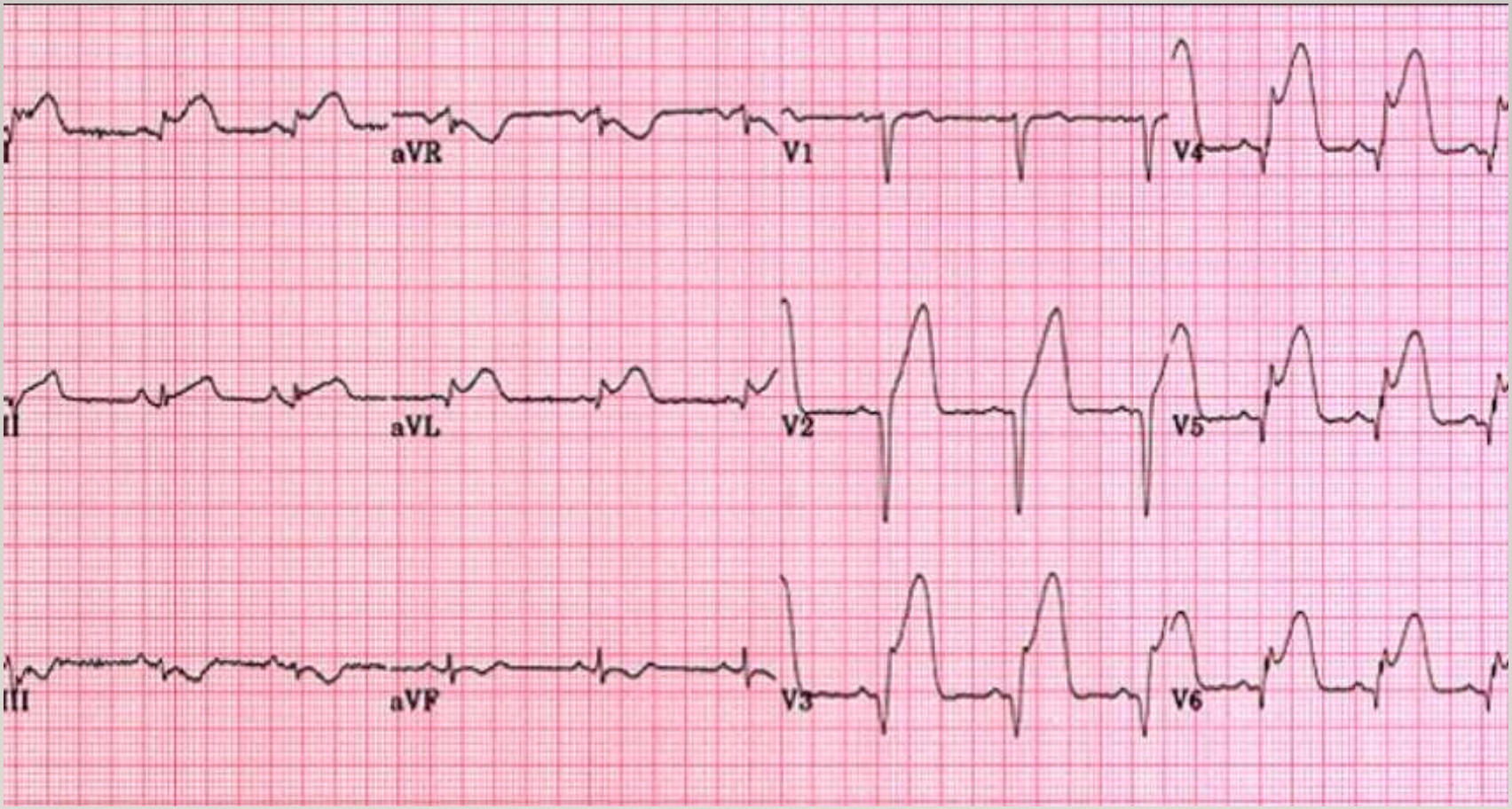
- ↓ CO — Cold extremities, distant pulses, acidosis,
- Pump Failure — Distended neck veins, S3, cold extremities
- ↓ Preload (CVP) — Flat or absent neck veins, tachycardia.
- ↑ Preload (CVP) — ↑ jugular vein distention, enlarged veins elsewhere
- ↑SVR — BP and mental state may be NORMAL.
  Findings: Cold extremities, distant pulses

Other valuable studies:

- Spot Echo exam of the heart: addresses tamponade, CHF, ischemia, hypovolemia
- O_2 saturation from CVP line or PICC line: provides indirect but meaningful estimates of the adequacy of DO_2, cardiac function.

EKG

- If STEMI, degree and severity of EKG should agree with severity of clinical condition
- If ST elevations in precordial leads -> likely anterior MI -> LV pump failure is likely cause
- If inferior STEMI -> need marked ST elevations with reciprocal ST depressions on EKG. Check RV leads. If no reciprocal changes or RV infarct, think mechanical problems such as papillary muscle rupture
- Normal EKG (especially with arrhythmias): think myocarditis


aVR
V1
V4
aVL
V2
V5
aVF
V3
V6

ECHOCARDIOGRAM

- Overall and regional systolic function
- Mechanical causes of shock
  - Papillary muscle rupture
  - Acute VSD
  - Free wall rupture
- Degree of mitral regurgitation
- Right ventricular infarction
- Other causes of shock (tamponade, PE, valvular stenosis)

THERAPY/TREATMENT

- ACC Guidelines
- Vasopressors and Inotropes
- Diuretics
- Cardiac Catheterization
- Intra-aortic balloon pumps (IABPs)
- Left Ventricular Assist Devices (LVADs)

INITIAL TREATMENT

- Position the patient
- Make certain that there is an adequate airway
- Maintain adequate oxygenation
- Draw blood for the tests
- Insert a Foley catheter into the urinary bladder to obtain accurate measurements of urinary output*
- Monitor the patient continuously
- Relieve pain
- Relieve agitation

THERAPY/TREATMENT: ACC GUIDELINES

- Early revascularization, either PCI or CABG, is recommended for patients with ST elevation or LBBB who develop shock within 36 hours of MI and who are suitable for revascularization that can be performed within 18 hours of shock unless further support is futile because of the patient's wishes or contraindications/unsuitability for further invasive care. *(Level of Evidence: A)*
- Fibrinolytic therapy should be administered to STEMI patients with cardiogenic shock who are unsuitable for further invasive care and do not have contraindications to fibrinolysis. *(Level of Evidence: B)*

VASOPRESSORS AND INOTROPES

- Goal: optimize perfusion while minimizing toxicity
- Invasive hemodynamic monitoring (arterial line, cardiac output monitoring) to guide therapy
- Low output syndrome without shock: start with an inotrope such as dobutamine
- Low output syndrome with shock: start with dopamine or norepinephrine

DRUG	RECEPTOR	DOSE	ACTION
Dobutamine	Beta-1 >> Beta-2	2 – 10 mcg/kg/min	Inotrope
Norepinephrine	Alpha-1 >Beta-1 >> Beta-2	0.5 - 20 mcg/min; Max 30 mcg/min.	Increases SVR, +/- impact of CO
Dopamine	Dopaminergic Dopa + Beta-1 Beta-1 Alpha-1 , some Beta-1	1-2 mcg/kg/min 2-5 mcg/kg/min 5-10 mcg/kg/min > 10 mcg/kg/min	Renal + mesenVD/VC Above, inotrope Inotrope Vasoconstriction, inotrope
Epinephrine	Alpha-1 = Alpha-2 Beta-1 = Beta-2	1-2 mcg/min 2-10 mcg/min >10 mcg/min	Beta → incr. HR/SV Beta > alpha Alpha → vasoconstriction
Phenylephrine	Alpha-1 > Alpha-2 >>>>> Beta	2 – 200 mcg/min	Increases SVR
Vasopressin		0.03 units/minute	Vasoconstriction

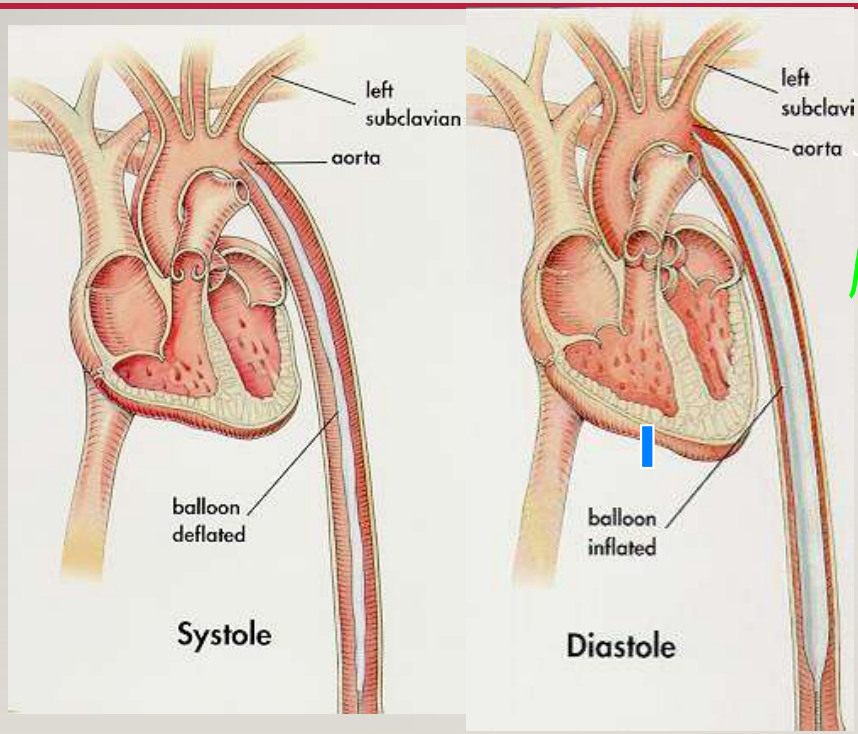
VASOPRESSORS AND INOTROPES

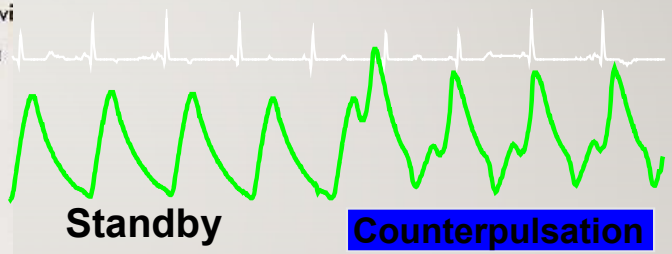
- Dobutamine: B1 and B2, inotropic but also causes peripheral vasodilation
  - Good for non-hypotensive cardiogenic shock
  - Start with 5 ug/kg/min, don't go higher than 20 ug/kg/min
- Dopamine: inotrope and vasopressor in hypotensive cardiogenic shock
  - Up to 3 ug/kg/min – vasodilation and increase blood flow to tissue beds, but no good evidence for "renal-dose dopamine"
  - Start at 5 ug/kg/min up to 15 ug/kg/min. Good inotropic and chronotropic effect at doses between 3 and 10 ug/kg/min (B1)
  - Mild peripheral vasoconstriction beyond 10 ug/kg/min (A1)

VASOPRESSORS AND INOTROPES

- Norepinephrine: primarily vasoconstrictor, mild inotrope
  - Increases SBP/DBP and pulse pressure
  - Increases coronary flow
  - Start 0.01 to 3 ug/kg/min
  - Good for severe shock with profound hypotension
- Epinephrine: B1/2 effects at low doses, A1 effects at higher doses
  - Increases coronary blood flow (increases time in diastole)
  - Prolonged exposure -> myocyte damage

INTRA-AORTIC BALLOON COUNTERPULSATION





CARDIAC CATHETERIZATION IN CARDIOGENIC SHOCK

- ACC Guidelines: emergent coronary revascularization is the standard of care for CS due to pump failure (acute MI and shock)
- Most often demonstrates multi-vessel disease:
  - Left main disease 23%
  - 3-vessel disease 64%
  - 2-vessel disease 22%
  - 1-vessel disease 14%

OUTCOMES IN CARDIOGENIC SHOCK

- In-hospital mortality rate: 50-60% for all age groups
- Mechanical complications: even higher rates of mortality
  - Ventricular septal rupture -> highest mortality (87% in SHOCK Registry)
- RV infarction: SHOCK – mortality unexpectedly high, similar to LV failure shock despite younger age, lower rate of anterior MI and higher prevalence of single vessel disease
- In hospital survival of diabetic patients in SHOCK was only marginally lower than non-diabetic patients

CARDIOGENIC SHOCK

XpertDox
Clinical, Education, Research

Life-threatening condition due to heart damage leading to poor pump function

Affects 1 in 10-20 patients with heart attack

Average time from onset of heart attack to cardiogenic shock is 7 hrs

Left ventricular failure accounts for about 79% cases

Treatment includes inotropic medicines, Ventricular Assist Devices & heart transplantation

Diagnosed by clinical exam, echocardiography & cardiac catheterization

Symptoms include palpitations, dizziness, shortness of breath & fainting attacks

Complications include liver damage, kidney damage, paralysis, heart attack & sudden death

Overall in-hospital mortality rate is 39%

For persons 75 years and older, the death rate is 55%