SHOCK

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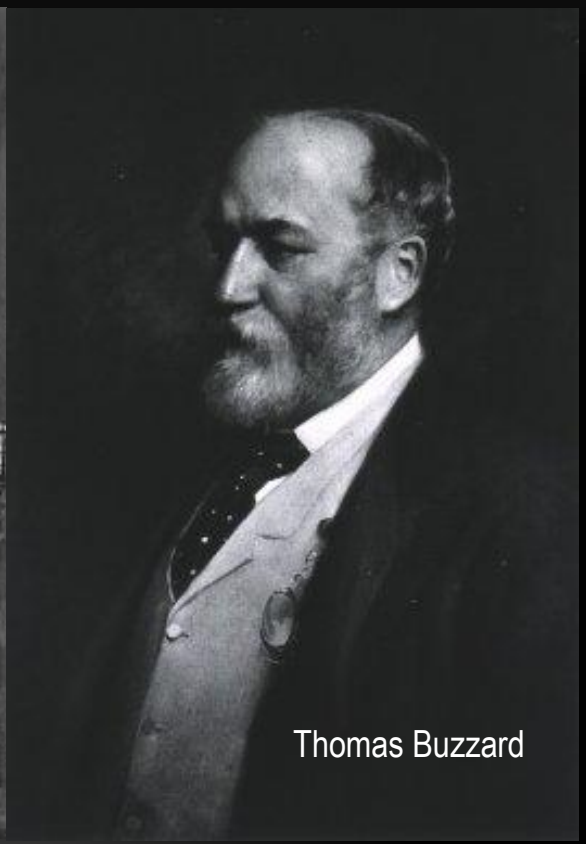
WHAT IS SHOCK



WHAT IS SHOCK

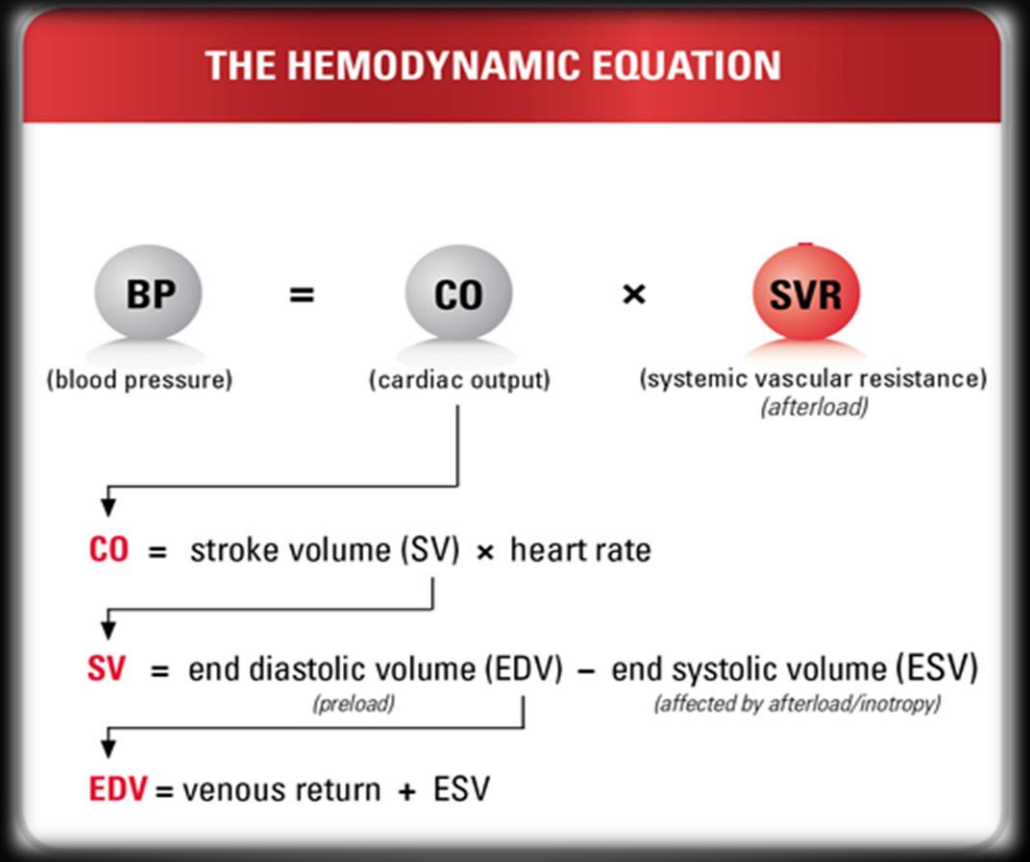
- THE BEGINNINGS OF UNDERSTANDING: THE LATE 19TH CENTURY
- THE AGE OF REASON: 1890—1925
- THE MODERN ERA: BLALOCK'S EPIPHANY
- POSTMODERNISM: CELLULAR PHYSIOLOGY OF SHOCK 1945—1965
- REACHING THE NEW MILLENNIUM: 1990--PRESENT

WHAT IS SHOCK

- A physiologic state characterized by
  - Inadequate tissue perfusion
  - imbalance between the delivery of and requirements for oxygen and substrate

- Clinically manifested by
  - Hemodynamic disturbances
  - Organ dysfunction

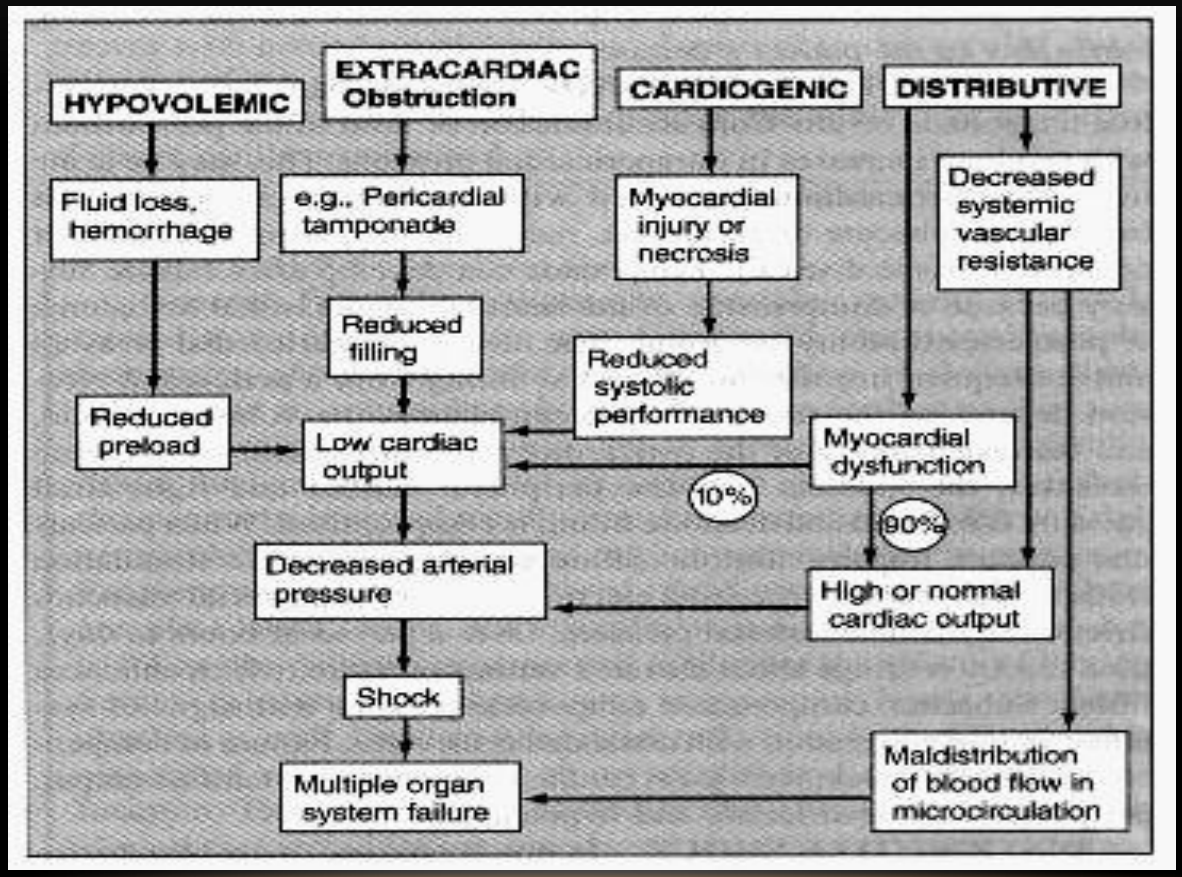
CLASSES OF SHOCK



CLASSES OF SHOCK

- Cargiogenic Shock
- Hypovolemic Shock
- Distributive Shock
- Obstructive Shock

CLASSES OF SHOCK



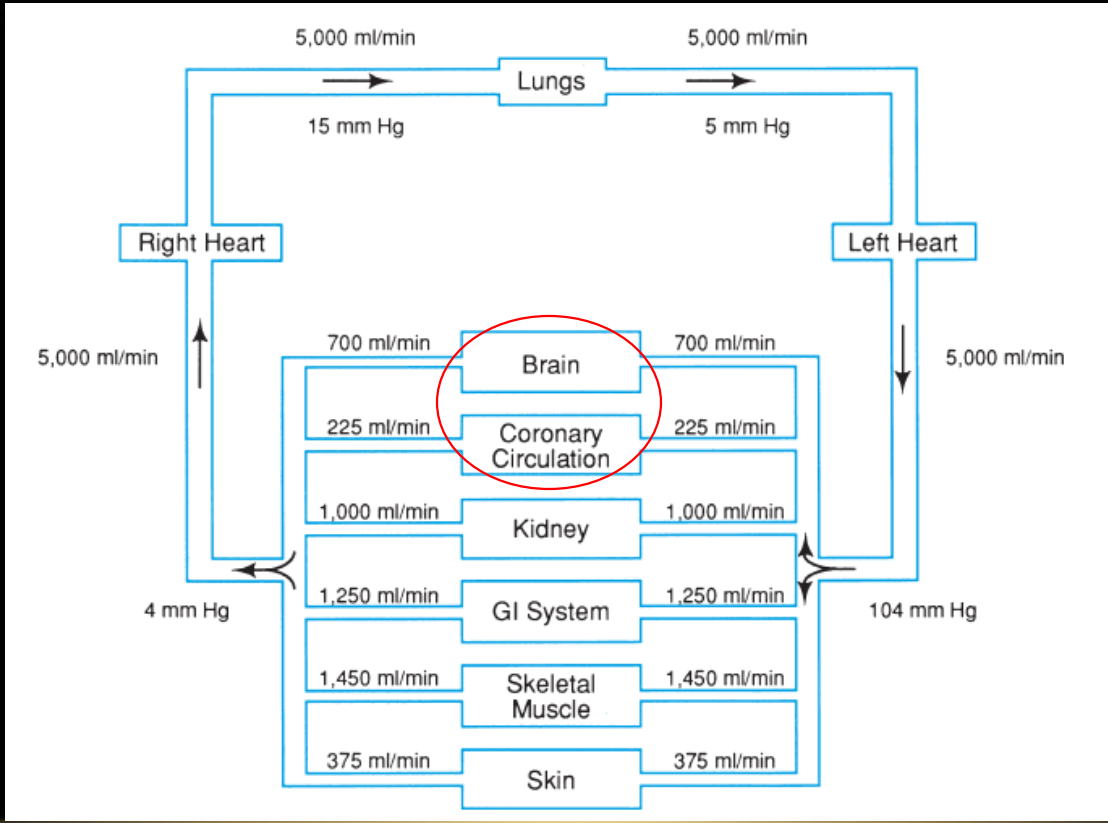
PATHOPHYSIOLOGY

Compensatory mechanism

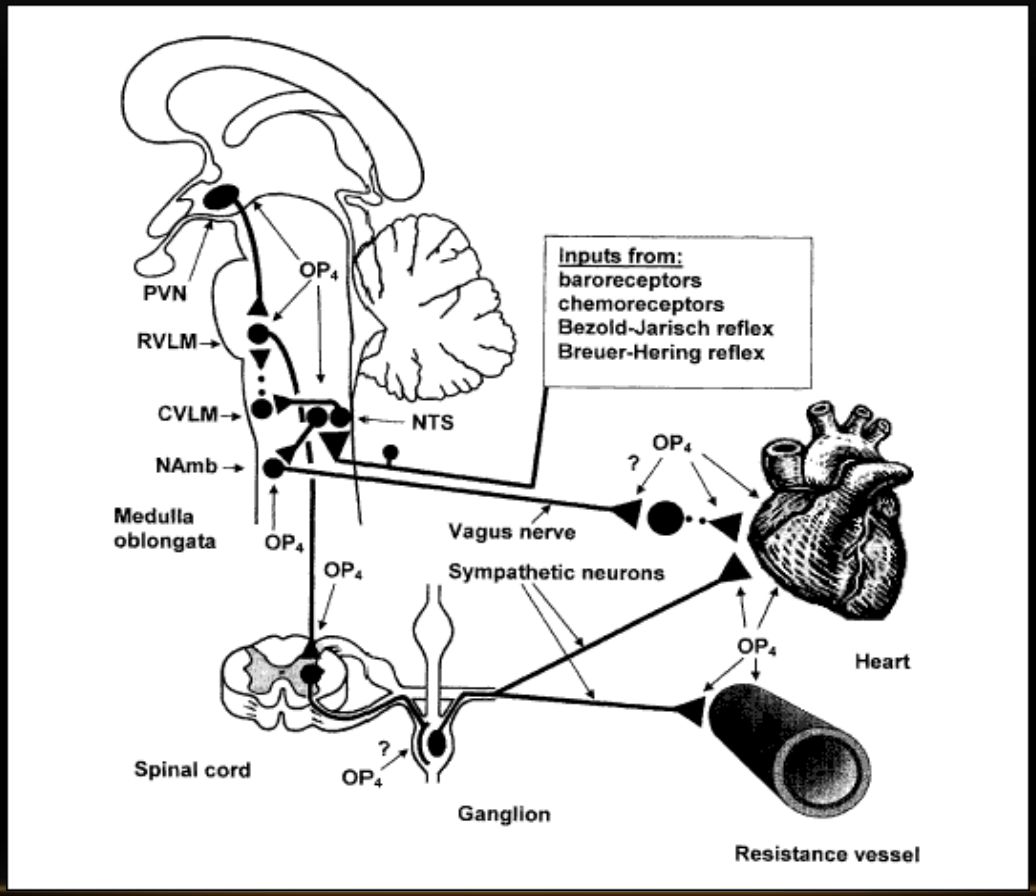
Cellular changes

Organ responses

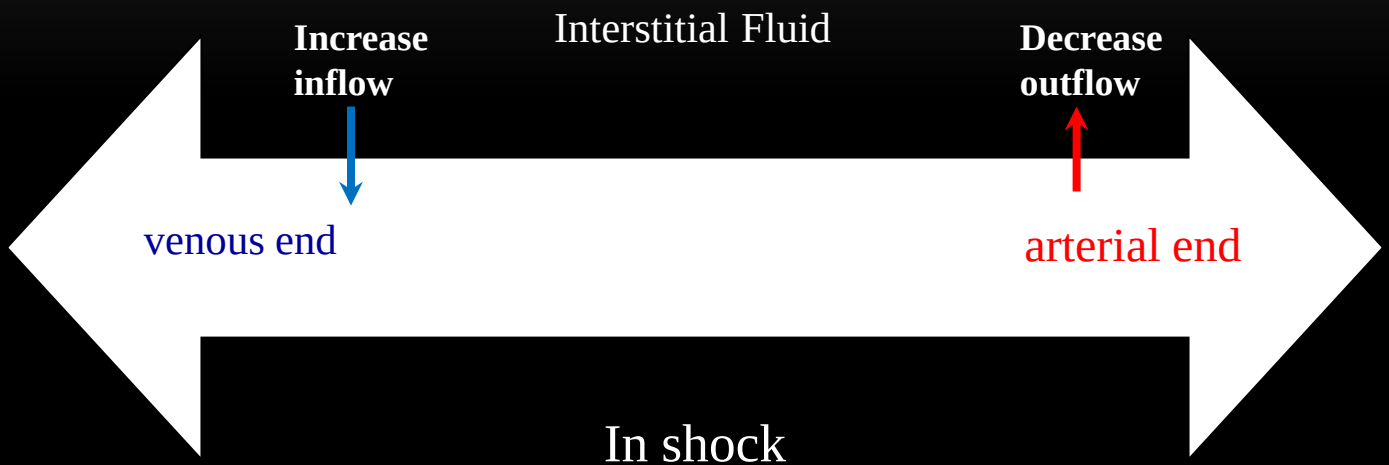
COMPENSATORY MECHANISM



COMPENSATORY MECHANISM



COMPENSATORY MECHANISM



Fluid shift system

CELLULAR CHANGES

- Cell membrane ion pump dysfunction
- Leakage of intracellular contents into the extracellular space
- Intracellular pH dysregulation

RESULTANT SYSTEMIC CHANGES

- Initial signs of end organ dysfunction
  - Tachycardia
  - Tachypnea
  - Metabolic acidosis
  - Oliguria
  - Cool and clammy skin

RESULTANT SYSTEMIC CHANGES

- End Organ Dysfunction
  - Progressive irreversible dysfunction
  - Oliguria or anuria
  - Progressive acidosis and decreased CO
  - Agitation, obtundation, and coma
  - Patient death

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Kim Jong-il dead

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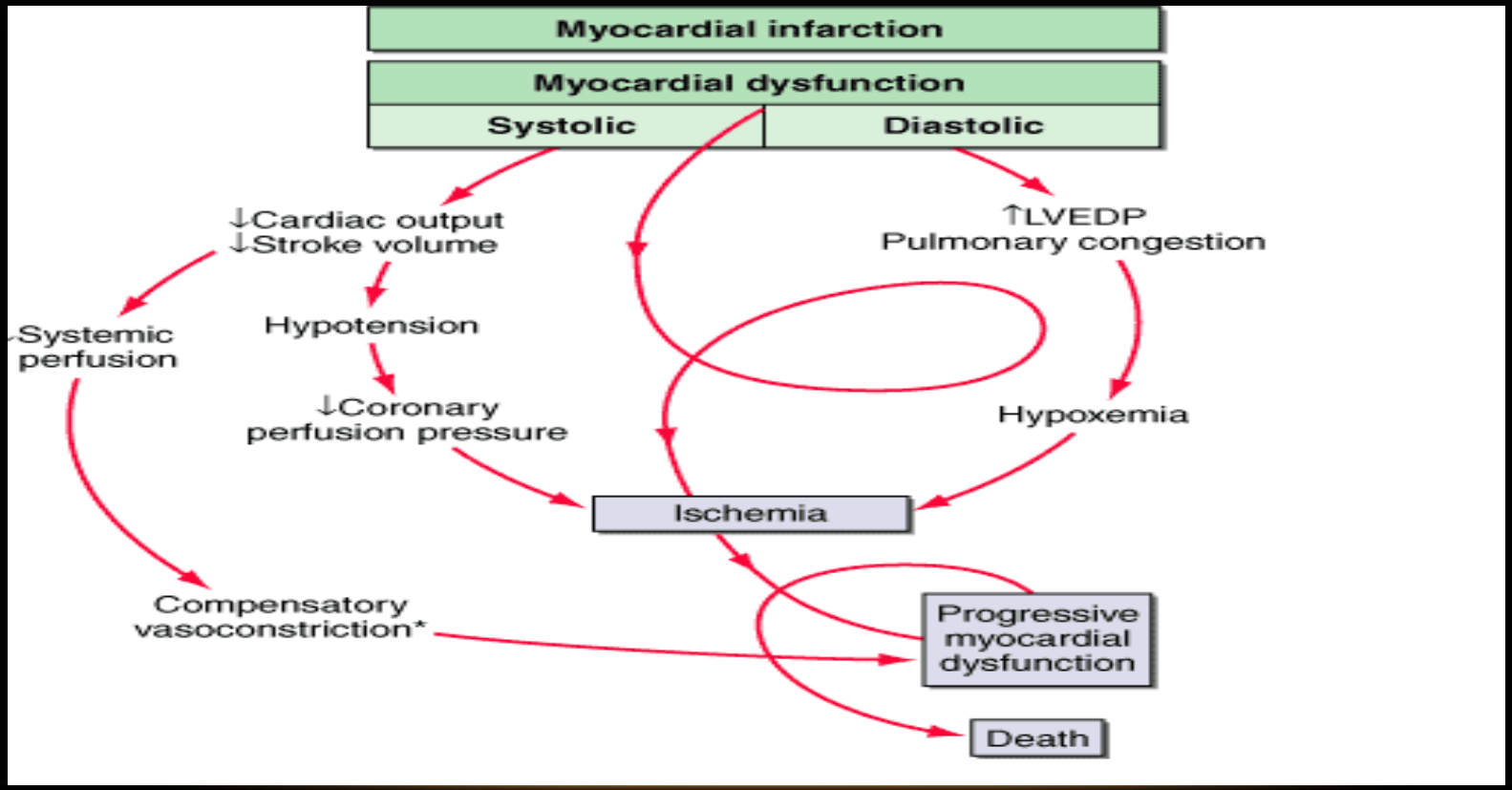
CARDIOGENIC SHOCK

- systemic hypoperfusion secondary to severe depression of cardiac output
- sustained systolic arterial hypotension despite elevated filling pressures

CARDIOGENIC SHOCK



CARDIOGENIC SHOCK

- Physical Examination:

dilated JV, +S3, rales, oliguria, acute pulmonary edema

- Hemodynamics:

decreased CO, increased SVR, decreased SvO2

CARDIOGENIC SHOCK

- Pressors
- Intra-aortic Balloon Pump (IABP)
- Revascularization: Fibrinolytics/PCI/CABG
- Refractory shock: ventricular assist device, cardiac transplantation

PRESSORS

- Dopamine
  - <2 renal vascular dilation
  - <2-10 +chronotropic/inotropic (beta effects)
  - >10 vasoconstriction (alpha effects)
- Dobutamine
  - positive inotrope
  - Vasodilates
  - arrhythmogenic
- Norepinephrine (Levophed):
  - Vasoconstriction
  - inotropic stimulant.
- Vasopression – vasoconstriction
  - Vasoconstriction

HYPOVOLEMIC SHOCK

- Results from decreased preload
- Etiologic classes
  - Hemorrhage - e.g. trauma, GI bleed, ruptured aneurysm
  - Fluid loss - e.g. diarrhea, vomiting, burns, third spacing, iatrogenic

HYPOVOLEMIC SHOCK

- Physical Examination:

flat JV, cool, cyanotic extremities, clear lungs, oliguria, evidence of bleeding

- Hemodynamics:

decreased CO, increased SVR, decreased SvO_2

HYPOVOLEMIC SHOCK

Parameter	I	II	III	IV
Blood loss (ml)	<750	750–1500	1500–2000	>2000
Blood loss (%)	<15%	15–30%	30–40%	>40%
Pulse rate (beats/min)	<100	>100	>120	>140
Blood pressure	Normal	Decreased	Decreased	Decreased
Respiratory rate (bpm)	14–20	20–30	30–40	>35
Urine output (ml/hour)	>30	20–30	5–15	Negligible
CNS symptoms	Normal	Anxious	Confused	Lethargic

Crit Care. 2004; 8(5): 373–381.

DISTRIBUTIVE SHOCK

- Results from a severe decrease in SVR
  - Vasodilation reduces afterload
  - May be associated with increased CO

DISTRIBUTIVE SHOCK

- Sepsis
- Neurogenic / spinal
- Systemic inflammation – pancreatitis
- Toxic shock syndrome
- Anaphylaxis and anaphylactoid reactions
- Toxin reactions – drugs, transfusions
- Addisonian crisis
- Myxedema coma

SEPTIC SHOCK

- Physical Examination:

  Early– warm wet/ vasodilation, often adequate urine output, febrile, tachypneic.

  Late – vasoconstriction, hypotension, cool, clammy skin , oliguria,altered mental status.

- Monitor/findings:

  Early—hyperglycemia, respiratory alkylosis, hemoconcentration, WBC typically normal or low

  Late – Leukocytosis, lactic acidosis

  Very Late– Disseminated Intravascular ,Coagulation & Multi-Organ System Failure

- Hemodynamics:

  decreased CO, decreased SVR, decreased SvO_2

SEPTIC SHOCK

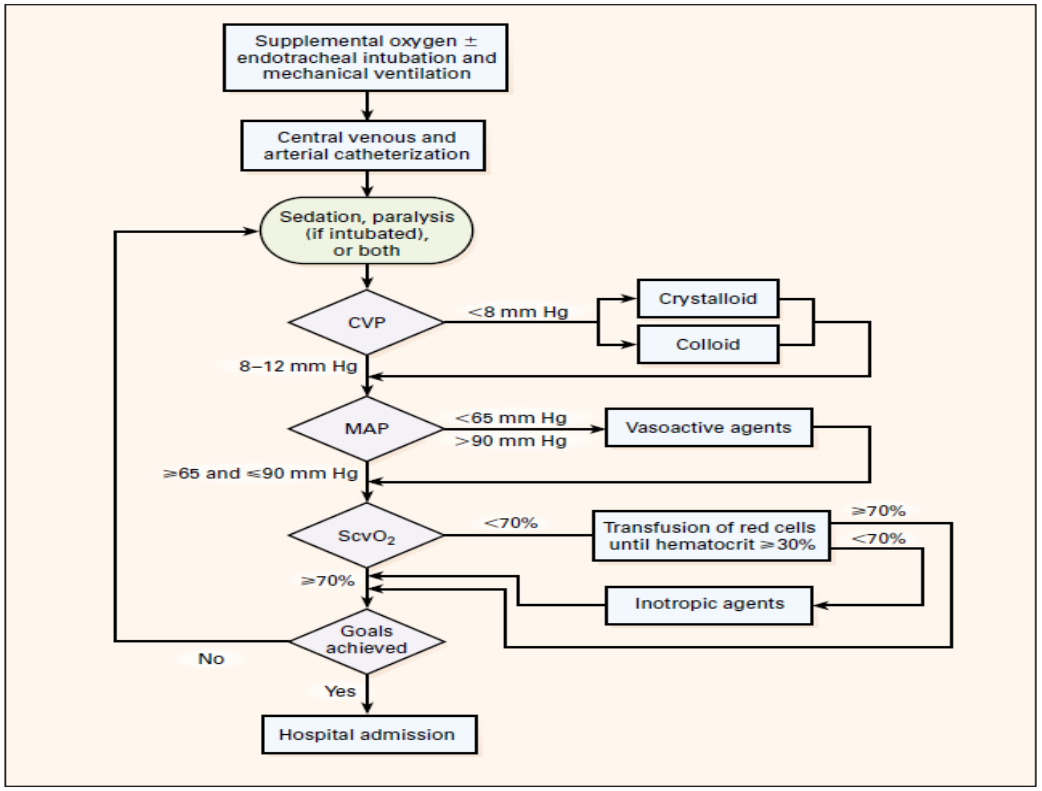


Figure 2. Protocol for Early Goal-Directed Therapy.
CVP denotes central venous pressure, MAP mean arterial pressure, and $ScvO_2$ central venous oxygen saturation.


IBM
02mph

OBSTRUCTIVE SHOCK

- Pulmonary Embolism
- Cardiac Tamponade
- Tension Pneumothorax
- Presentation will be according to underlying disease process.

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 ↔	↑	↓	↑

MANAGEMENT

- Manage the emergency
- Determine the underlying cause
- Definitive management or support

MANAGE THE EMERGENCY

- How long do you have to manage this?
- Suggests that many things must be done at once
- Draw in ancillary staff for support!
- What must be done?

MANAGE THE EMERGENCY

- One person runs the code!
- Control airway and breathing
- Maximize oxygen delivery
- Place lines, tubes, and monitors
- Get and run IVF on a pressure bag
- Get and run blood (if appropriate)
- Get and hang pressors
- Call your senior

EVALUATION

- Done in parallel with treatment!
- distinguish type of shock
- Full laboratory evaluation
- Basic studies – CXR, EKG
- Basic monitoring

DETERMINE THE CAUSE

- Often obvious based on history
- Trauma most often hypovolemic
- Postoperative most often hypovolemic
- Debilitated hospitalized patients most often septic
- Must evaluate all patients for risk factors for MI

DEFINITIVE MANAGEMENT

- Hypovolemic – Fluid resuscitate and control ongoing loss
- Cardiogenic - Restore blood pressure and prevent ongoing cardiac death
- Distributive – Fluid resuscitate, pressors for maintenance, immediate control for infection, steroids for adrenocortical insufficiency