Shock
Pathophysiology, Classification, and Approach to Management
Cardiogenic shock - a major component of the mortality associated with cardiovascular disease (the #1 cause of U.S. deaths)

Hypovolemic shock - the major contributor to early mortality from trauma (the #1 cause of death in those < 45 years of age)

Septic shock - the most common cause of death in American ICUs (the 13th leading cause of death overall in US)
Shock: Definitions

Kumar and Parrillo (1995) - “The state in which profound and widespread reduction of effective tissue perfusion leads first to reversible, and then if prolonged, to irreversible cellular injury.”
Shock: Classification

- **Hypovolemic shock** - due to decreased circulating blood volume in relation to the total vascular capacity and characterized by a reduction of diastolic filling pressures.

- **Cardiogenic shock** - due to cardiac pump failure related to loss of myocardial contractility/functional myocardium or structural/mechanical failure of the cardiac anatomy and characterized by elevations of diastolic filling pressures and volumes.

- **Extra-cardiac obstructive shock** - due to obstruction to flow in the cardiovascular circuit and characterized by either impairment of diastolic filling or excessive afterload.

- **Distributive shock** - caused by loss of vasomotor control resulting in arteriolar/venular dilatation and characterized (after fluid resuscitation) by increased cardiac output and decreased SVR.
Classification of Circulatory Shock

HYPOVOLEMIC

◥ Hemorrhagic
  ● Trauma
  ● Gastrointestinal
  ● Retroperitoneal

◥ Fluid depletion (nonhemorrhagic)
  ● External fluid loss
    - Dehydration
    - Vomiting
    - Diarrhea
    - Polyuria
  ● Interstitial fluid redistribution
    - Thermal injury
    - Trauma
    - Anaphylaxis

◥ Increased vascular capacitance (venodilatation)
  ● Sepsis
  ● Anaphylaxis
  ● Toxins/drugs

Kumar and Parrillo, 2001
Classification of Circulatory Shock

CARDOGENIC

Myopathic
- Myocardial infarction (hibernating myocardium)
- Left ventricle
- Right ventricle
- Blunt Cardiac Injury (trauma)
- Myocarditis
- Cardiomyopathy
- Post-ischemic myocardial stunning
- Septic myocardial depression

Pharmacologic
- Anthracycline cardiotoxicity
- Calcium channel blockers

Mechanical
- Valvular failure (stenotic or regurgitant)
- Hypertropic cardiomyopathy
- Ventricular septal defect

Arrhythmic
- Bradycardia
- Tachycardia

Kumar and Parrillo, 2001
Classification of Circulatory Shock

EXTRACARDIAC OBSTRUCTIVE

✈ Impaired diastolic filling (decreased ventricular preload)
  • Direct venous obstruction (vena cava)
    - intrathoracic obstructive tumors
  • Increased intrathoracic pressure
    - Tension pneumothorax
    - Mechanical ventilation (with excessive pressure or volume depletion)
    - Asthma
  • Decreased cardiac compliance
    - Constrictive pericarditis
    - Cardiac tamponade

✈ Impaired systolic contraction (increased ventricular afterload)
  • Right ventricle
    - Pulmonary embolus (massive)
    - Acute pulmonary hypertension
  • Left ventricle
    - Saddle embolus
    - Aortic dissection

Kumar and Parrillo, 2001
Classification of Circulatory Shock

**DISTRIBUTIVE**
- Septic (bacterial, fungal, viral, rickettsial)
- Toxic shock syndrome
- Anaphylactic, anaphylactoid
- Neurogenic (spinal shock)
- Endocrinologic
  - Adrenal crisis
  - Thyroid storm
- Toxic (e.g., nitroprusside, bretylium)

Kumar and Parrillo, 2001
## Shock Hemodynamics

<table>
<thead>
<tr>
<th></th>
<th>CO</th>
<th>SVR</th>
<th>PAOP</th>
<th>EDV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypovolemic</td>
<td>↓</td>
<td>↑</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Cardiogenic</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Obstructive</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Obstructive afterload</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Obstructive preload</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Distributive</td>
<td>↓</td>
<td>↑</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Distributive pre-resusc</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Distributive post-resusc</td>
<td>↑</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
</tr>
</tbody>
</table>
Classification of Shock

Hypovolemic Cardiogenic
- (e.g., hemorrhage)
- ↓ Preload
- ↓ Diastolic filling
- ↓ Systolic and diastolic function

Cardiogenic
- (e.g., Myocardial infarction)
- ↓ Myocardial damage
- ↓ Diastolic filling
- ↓ Systolic and diastolic function

Extracardiac Obstructive
- (e.g., tension pneumothorax or pericardial tamponade)
- ↓ Diastolic filling
- ↓ Systolic function

Obstructive
- (e.g., massive pulmonary embolus)
- ↓ Ventricular afterload
- ↓ Preload
- ↓ Diastolic filling

Distributive
- (e.g., septic)
- ↓ Myocardial depression
- ↓ Systolic and diastolic function
- ↓ SVR (↑ CO)
- ↓ MAP
- ↓ Shock
- ↓ MODS
- Maldistribution of flow

CO = cardiac output; SVR = systemic vascular resistance; MAP = mean arterial blood pressure; MODS = multiple organ dysfunction syndrome.

Kumar and Parrillo, 2001
Hypovolemic Shock

Degree of volume loss → response

- 10% well tolerated (tachycardia)
- 20 - 25% failure of compensatory mechanisms (hypotension, orthostasis, decreased CO)
- > 40% loss associated with overt shock (marked hypotension, decreased CO, lactic acidemia)
# Clinical Correlates of Hemorrhage

<table>
<thead>
<tr>
<th>Class</th>
<th>Class I</th>
<th>Class II</th>
<th>Class III</th>
<th>Class IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood loss (mL)</td>
<td>&gt; 750</td>
<td>750 - 1500</td>
<td>1500 - 2000</td>
<td>&gt; 2000</td>
</tr>
<tr>
<td>Blood loss (% total)</td>
<td>&gt; 15%</td>
<td>15 - 30%</td>
<td>30 - 40%</td>
<td>&gt; 40%</td>
</tr>
<tr>
<td>Pulse rate</td>
<td>&lt; 100</td>
<td>&gt; 100</td>
<td>&gt; 120</td>
<td>&gt; 140</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>Normal</td>
<td>Normal</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Pulse pressure</td>
<td>Normal or ↑</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Orthostasis</td>
<td>Absent</td>
<td>Minimal</td>
<td>Marked</td>
<td>Marked</td>
</tr>
<tr>
<td>Capillary refill</td>
<td>Normal</td>
<td>Delayed</td>
<td>Delayed</td>
<td>Delayed</td>
</tr>
<tr>
<td>Resp rate</td>
<td>14 - 20</td>
<td>20 - 30</td>
<td>30 - 40</td>
<td>&gt; 34</td>
</tr>
<tr>
<td>UO (mL/hr)</td>
<td>&gt; 30</td>
<td>20 - 30</td>
<td>5 - 15</td>
<td>&lt; 5</td>
</tr>
<tr>
<td>CNS mental status</td>
<td>Slight anxiety</td>
<td>Mild anxiety</td>
<td>Anxious/confused</td>
<td>Confused/lethargic</td>
</tr>
<tr>
<td>CI (L/min)</td>
<td>↓ 0-10%</td>
<td>↓ 20-50%</td>
<td>↓ 50-75%</td>
<td>↓ &gt;75%</td>
</tr>
</tbody>
</table>

*American College of Surgeons, 1989*
Hypovolemic Shock

Rate of volume loss and pre-existing cardiac reserve response:

- Acute 1L blood loss results in mild to moderate hypotension with decreased CVP and PWP
- Same loss over longer period may be tolerated without hypotension due to increased fluid retention, increased RBC 2,3 DPG, tachycardia, and increased myocardial contractility
- Same slow loss in patient with diminished cardiac reserve may cause hypotension or shock.
Cardiogenic Shock

#1 cause of in-hospital mortality from Q-wave MI

Requires at least 40% loss of functional myocardium (single MI or cumulative damage) - stunned, nonfunctional, but viable myocardium may contribute to post-MI cardiogenic shock

Usually involves left main or left anterior descending obstruction

Historically, incidence of cardiogenic shock post-Q wave MI has run 8 - 20% with mortality 70 - 90% (? reduced incidence with thrombolytics 4 - 7%)
Mortality substantially better for cardiogenic shock due to surgically remediable lesions:

- **aortic valve failure** (endocarditis, occasionally prosthetic valve failure or aortic dissection)
- **papillary muscle rupture** (infarct, post-blunt chest trauma, endocarditis, prosthetic valve failure)
  - ischemic form seen 3 - 7 days post-LAD territory infarct (often preceded by new MR murmur)
  - v wave of > 10 mm often seen in PWP trace
- **VSD** (post-infarct, rarely traumatic)
  - post-infarct seen 3 - 7 days post-LAD occlusion
  - 5 - 10% oxygen saturation step-up
Cardiogenic Shock

- RV infarction with cardiogenic shock seen in only 10 - 20% largest inferior wall MIs
- Isolated RV infarcts rare - almost all have some degree of LV involvement
- DX includes cardiac tamponade, restrictive cardiomyopathy, constrictive pericarditis, and PE - Kussmaul’s sign, pulsus paradoxus, filling pressure equalization may be seen in all
- Rx fluids and inotropes rather than pressors
- Good prognosis relative to LV infarct + shock
Obstructive Shock

Rate of development of obstruction to blood flow response:
- acute, massive PE involving 2 or more lobar arteries and 50% pulmonary bed can cause shock (sPAP max 50 mm Hg) but chronic PE can cause > 75% obstruction without shock (sPAP 100 + mm Hg)
- acute cardiac tamponade can occur with 150 mL fluid, but over 2L can be well tolerated if slow accumulation

Similar variability based on presence of pre-existing cardiopulmonary disease
Distributive Shock

- Defining feature: loss of peripheral resistance
- Dominantly septic shock, anaphylactic and neurogenic shock less common
- Clinical form of shock with greatest contribution of other shock elements - i.e., hypovolemia, cardiac failure
Distributive Shock

- **Anaphylactic shock**: immediate hypersensitivity reaction mediated by the interaction of IgE on mast cells and basophils with the appropriate antigen resulting in mediator cascade.

- **Anaphylactoid reactions** involve similar release of mediators via non-immunologic mechanisms.

- Primary mediators include histamine, serotonin, eosinophil chemotactic factor, and proteolytic enzymes.

- Secondary mediators include PAF, bradykinin, prostaglandins, and leukotrienes.
Distributive Shock

Anaphylactic shock
- insect envenomations
- antibiotics (beta-lactams, vancomycin, sulfonamides)
- heterologous serum (anti-toxin, anti-sera)
- blood transfusion
- immunoglobulins (esp IgA deficient)
- Egg-based vaccines
- latex

Anaphylactoid shock
- ionic contrast media
- protamine
- opiates
- polysaccharide volume expanders (dextran, hydroxyethyl starch)
- muscle relaxants
- anesthetics
**Hypodynamic Shock: Perfusion**

- Extrinsic regulatory mechanisms dominate in most vascular beds except brain and heart.
- Blood flow to other organs decreased via sympathetic vasoconstrictive effects.
- Post-resuscitation, perfusion abnormalities may persist for days (decreased perfusion of brain, kidneys, liver, splanchnic organs) with potential persistent ischemia.
- ? irreversible hypodynamic shock.
Hyperdynamic Shock: Perfusion

- Organ blood flow disturbed at higher pressures suggesting a primary microvascular regulatory defect.
- Cerebral perfusion decreased by 33% while coronary vascular resistance is significantly increased in septic shock - i.e., coronary and cerebral autoregulatory mechanisms are relatively intact in sepsis.
- All other vascular beds exhibit similarly decreased vascular resistance suggesting active vasodilatory process and failure of extrinsic control mechanisms.
- Microvascular studies also show aberrant distribution of perfusion within tissues and organs.
Determinants of Effective Tissue Perfusion

Cardiovascular Performance
  Cardiac Function
  Venous Return

Vascular Performance

Microvascular Function

Oxygen Unloading and Diffusion

Cellular Energy Metabolism
Cardiac Performance

- Preload
- Left ventricular size
- Myocardial fiber shortening
- Stroke volume
- Heart rate
- Peripheral resistance
- Cardiac output
- Arterial pressure

Factors affecting cardiac performance:
- Preload
- Contractility
- Afterload
Organ Blood Flow in Shock

Dependent on maintenance of blood pressure within an acceptable range

For humans, good overall auto-regulation of blood flow between 60 - 100 mm Hg

However, experimental data in animals shows brain and heart have wider ranges while skeletal muscle has a significantly narrow auto-regulatory range.
Vascular Failure: Potential Causes

1) Tissue acidosis
2) Catecholamine depletion and resistance
3) Endogenous vasoactive substances
4) Decreased central sympathetic tone
5) Pathophysiologic nitric oxide generation
Microvasculature in Shock

- Vessels of 100 to 150 um diameter
- Precapillary vs. postcapillary sphincters
- Intrinsic control (autoregulation)
  - stretch receptors
  - chemoreceptors (CO$_2$, H+)
- Extrinsic control via autonomic nervous system
Determinants of Effective Tissue Perfusion (cont)

- Oxygen unloading and diffusion
  - Oxyhemoglobin affinity
    - RBC 2, 3 DPG
    - Blood pH
    - Temperature

- Cellular Function
  - Cellular energy generation/substrate utilization
    - Citric acid (Krebs) cycle
      - Oxidative phosphorylation
      - Other energy metabolism pathways

RBC = Red blood cells  DPG = Diphosphoglycerate
Mechanisms of Cellular Injury in Shock

1) Cellular ischemia
2) Free radical reperfusion injury
3) Inflammatory mediators (local and circulating)
Physiologic Oxygen Supply Dependency

Oxygen Consumption

Oxygen Delivery

Lactic Acidosis

Critical Delivery Threshold

Pathologic Oxygen Supply Dependency

Cellular Ischemia in Shock

Evidence

- Oxygen supply-dependent oxygen consumption
- Washout of organic acids (from ischemic tissues) in patients with sepsis and MODS after vasodilator Rx
- Elevated ATP degradation products with decreased acetoacetate/hydroxybutyrate ratio (suggestive of altered hepatic mitochondrial redox potential)
Cardiovascular and Metabolic Compensatory Responses to Shock

- Maintain mean circulatory pressure (venous pressure)
  - **Volume**
    - Fluid redistribution to vascular space
      - From interstitium (Starling effect)
      - From intracellular space (osmotic)
    - Decrease renal losses
      - ↓ Glomerular filtration rate (GFR)
      - ↑ Aldosterone
      - ↑ Vasopressin
  - **Pressure**
    - Decreased venous capacitance
      - ↑ Sympathetic activity
      - ↑ Circulating (adrenal) epinephrine
      - ↑ Angiotensin
      - ↑ Vasopressin

Kumar and Parrillo, 2001
Cardiovascular and Metabolic Compensatory Responses to Shock

- Maximize cardiac performance
  - Increase contractility
    - Sympathetic stimulation
    - Adrenal stimulation

- Redistribute perfusion
  - Extrinsic regulation of systemic arterial tone
  - Dominant auto-regulation of vital organs (heart, brain)

- Optimize oxygen unloading
  - ↑ RBC 2,3 DPG
  - Tissue acidosis
  - Pyrexia
  - ↓ Tissue Po2

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# Organ System Dysfunction in Shock

<table>
<thead>
<tr>
<th>ORGAN SYSTEM</th>
<th>MANIFESTATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>CNS</td>
<td>Encephalopathy (ischemic or septic)</td>
</tr>
<tr>
<td></td>
<td>Cortical necrosis</td>
</tr>
<tr>
<td>Heart</td>
<td>Tachycardia, bradycardia</td>
</tr>
<tr>
<td></td>
<td>Supraventricular tachycardia</td>
</tr>
<tr>
<td></td>
<td>Ventricular ectopy</td>
</tr>
<tr>
<td></td>
<td>Myocardial ischemia</td>
</tr>
<tr>
<td></td>
<td>Myocardial depression</td>
</tr>
<tr>
<td>Pulmonary</td>
<td>Acute respiratory failure</td>
</tr>
<tr>
<td></td>
<td>Adult respiratory distress syndrome (ARDS)</td>
</tr>
<tr>
<td>Kidney</td>
<td>Prerenal failure</td>
</tr>
<tr>
<td></td>
<td>Acute tubular necrosis</td>
</tr>
<tr>
<td>GI</td>
<td>Ileus</td>
</tr>
<tr>
<td></td>
<td>Erosive gastritis</td>
</tr>
<tr>
<td></td>
<td>Pancreatitis</td>
</tr>
<tr>
<td></td>
<td>Acalculous cholecystitis</td>
</tr>
<tr>
<td></td>
<td>Colonic submucosal hemorrhage</td>
</tr>
<tr>
<td></td>
<td>Transluminal translocation of bacteria/endotoxin</td>
</tr>
</tbody>
</table>

*Kumar and Parrillo, 2001*
## Organ System Dysfunction in Shock

<table>
<thead>
<tr>
<th>Organ System</th>
<th>Manifestations</th>
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<tbody>
<tr>
<td>Liver</td>
<td>Ischemic hepatitis</td>
</tr>
<tr>
<td></td>
<td>&quot;Shock&quot; liver</td>
</tr>
<tr>
<td></td>
<td>Intrahepatic cholestasis</td>
</tr>
<tr>
<td>Hematologic</td>
<td>Disseminated intravascular coagulation</td>
</tr>
<tr>
<td></td>
<td>Dilutional thrombocytopenia</td>
</tr>
<tr>
<td>Metabolic</td>
<td>Hyperglycemia</td>
</tr>
<tr>
<td></td>
<td>Glycogenolysis</td>
</tr>
<tr>
<td></td>
<td>Gluconeogenesis</td>
</tr>
<tr>
<td></td>
<td>Hypoglycemia (late)</td>
</tr>
<tr>
<td></td>
<td>Hypertriglyceridemia</td>
</tr>
<tr>
<td>Immune System</td>
<td>Gut barrier function depression</td>
</tr>
<tr>
<td></td>
<td>Cellular immune depression</td>
</tr>
<tr>
<td></td>
<td>Humoral immune depression</td>
</tr>
</tbody>
</table>

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Society of Critical Care Medicine
Diagnosis and Evaluation

Clinical Signs

- Primary diagnosis - tachycardia, tachypnea, oliguria, encephalopathy (confusion), peripheral hypoperfusion (mottled, poor capillary refill vs. hyperemic and warm), hypotension

- Differential DX:
  - JVP - hypovolemic vs. cardiogenic
  - Left S3, S4, new murmurs - cardiogenic
  - Right heart failure - PE, tamponade
  - Pulsus paradoxus, Kussmaul’s sign - tamponade
  - Fever, rigors, infection focus - septic
Diagnosis and Evaluation

Laboratory

- Hgb, WBC, platelets
- PT/PTT
- Electrolytes, arterial blood gases
- BUN, Cr
- Ca, Mg
- Serum lactate, SVO2
- ECG
Diagnosis and Evaluation

Invasive Monitoring

- Arterial pressure catheter
- CVP monitoring
- Pulmonary artery catheter (+/- RVEF, oximetry)
- SVO2 / ScVO2
- DO and VO
A Clinical Approach to Shock Diagnosis and Management

Initial Diagnostic Steps

- CXR
- Abdominal views*
- CT scan abdomen or chest*
- Echocardiogram*
- Pulmonary perfusion scan*
A Clinical Approach to Shock Diagnosis and Management

Initial Therapeutic Steps

- Admit to intensive care unit (ICU)
- Venous access (1 or 2 wide-bore catheters)
- Central venous catheter
- Arterial catheter
- EKG monitoring
- Pulse oximetry
- Hemodynamic support (MAP < 60 mmHg)
  - Fluid challenge
  - Vasopressors for severe shock unresponsive to fluids
A Clinical Approach to Shock Diagnosis and Management

Diagnosis Remains Undefined or Hemodynamic Status Requires Repeated Fluid Challenges of Vasopressors

- **Pulmonary Artery Catheterization**
  - Cardiac output
  - Oxygen delivery
  - Filling pressures

- **Echocardiography**
  - Pericardial fluid
  - Cardiac function
  - Valve or shunt abnormalities
**Diagnosis of Shock Etiology Using Pulmonary Artery Catheterization**

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Pulmonary Artery Occlusion Pressure</th>
<th>Cardiac Output</th>
<th>Miscellaneous Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cardiogenic Shock</strong></td>
<td>↑↑</td>
<td>↓↓</td>
<td>Usually occurs with evidence of extensive myocardial infarction (40% of LV infarcted), severe cardiomyopathy, or myocarditis.</td>
</tr>
<tr>
<td>Cardiogenic shock due to myocardial dysfunction</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiogenic shock due to a mechanical defect</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute VSD</td>
<td>↑</td>
<td>LVCO ↓↓</td>
<td>Predominant shunt is left to right, pulmonary blood flow is greater than systemic blood flow: oxygen &quot;step-up&quot; occurs at RV level.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>and RVCO &gt; LVCO</td>
<td></td>
</tr>
</tbody>
</table>

*Kumar and Parrillo, 2001*
### Diagnosis of Shock Etiology Using Pulmonary Artery Catheterization (cont)

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Pulmonary Artery Occlusion Pressure</th>
<th>Cardiac Output</th>
<th>Miscellaneous Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cardiogenic Shock</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute mitral regurgitation</td>
<td>↑↑</td>
<td>Forward CO ↓↓</td>
<td>V waves in pulmonary artery occlusion pressure tracing.</td>
</tr>
<tr>
<td>Right ventricular infarction</td>
<td>Normal or ↓</td>
<td>↓↓</td>
<td>Elevated RA and RV filling pressures with low or normal pulmonary artery occlusion pressures.</td>
</tr>
<tr>
<td><strong>Extracardiac obstructive forms of shock</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pericardial tamponade</td>
<td>↑</td>
<td>↓ or ↓↓</td>
<td>RA mean, RV end-diastolic, pulmonary artery occlusion mean pressures are elevated and within 5</td>
</tr>
</tbody>
</table>
# Diagnosis of Shock Etiology Using Pulmonary Artery Catheterization (cont)

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Pulmonary Artery Occlusion Pressure</th>
<th>Cardiac Output</th>
<th>Miscellaneous Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Extracardiac obstructive forms of shock</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Massive pulmonary embolism</td>
<td>Normal or ↓</td>
<td>↓↓</td>
<td>Usual finding is elevated right-sided pressures.</td>
</tr>
<tr>
<td><strong>Hypovolemic shock</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Distributive forms of shock</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Septic shock</td>
<td>↓ or normal</td>
<td>↑ or normal, rarely ↓</td>
<td></td>
</tr>
<tr>
<td>Anaphylactic shock</td>
<td>↓ or normal</td>
<td>↑ or normal</td>
<td>Pre-resuscitation cardiac output is decreased</td>
</tr>
</tbody>
</table>

*Kumar and Parrillo, 2001*
A Clinical Approach to Shock Diagnosis and Management

Immediate Goals in Shock

**Hemodynamic support**
- MAP > 60 mmHg
- PAOP = 12 - 18 mmHg
- Cardiac Index > 2.2 L/min/m²

**Maintain oxygen delivery**
- Hemoglobin > 10 g/dL
- Arterial saturation > 92%
- Supplemental oxygen and mechanical ventilation

**Reversal of oxygen dysfunction**
- Decreasing lactate (< 2.2 mM/L)
- Maintain urine output
- Reverse encephalopathy
- Improving renal, liver function tests

MAP = mean arterial pressure; PAOP = pulmonary artery occlusion pressure.
Hypovolemic Shock

- Rapid replacement of blood, colloid, or crystalloid
- Identify source of blood or fluid loss:
  - OR
  - Endoscopy/colonoscopy
  - Angiography
  - CT/MRI scan
  - Other
A Clinical Approach to Shock Diagnosis and Management

Cardiogenic Shock

LV infarction
- Intra-aortic balloon pump (IABP)
- Cardiac angiography
- Revascularization
  - angioplasty
  - coronary bypass

RV infarction
- Fluid and inotropes with PA catheter monitoring

Mechanical abnormality
- Echocardiography
- Cardiac cath
- Corrective surgery
A Clinical Approach to Shock Diagnosis and Management

Extra-cardiac Obstructive Shock

- **Pericardial tamponade**
  - pericardiocentesis
  - surgical drainage (if needed)

- **Pulmonary embolism**
  - heparin
  - ventilation/perfusion lung scan
  - pulmonary angiography
  - consider:
    - thrombolytic therapy
    - embolectomy at surgery
Septic shock

- Identify site of infection and drain, if possible
- Antimicrobial agents (key rules)
- ICU monitoring and support with fluids, vasopressors, and inotropic agents
- EGDT (Rivers, 2001)
- Surviving Sepsis Guidelines (CCM, 2007)
- Goals:
  - SV02 > 70%
  - improving organ function
  - decreasing lactate levels
Fluid Therapy

Crystalloids
- Lactated Ringer’s solution
- Normal saline

Colloids
- Hetastarch
- Albumin

Packed red blood cells

Infuse to physiologic endpoints
Correct hypotension first (golden hour)
Decrease heart rate
Correct hypoperfusion abnormalities
Monitor for deterioration of oxygenation
Therapy: Resuscitation Fluids

- Crystalloid vs. colloid
- Optimal PWP 10 - 12 vs. 15 - 18 mm Hg
- 20 mL/kg fluid challenge in hypovolemic or septic shock with re-challenges of 5 - 10 mL/kg
- 100 - 200 mL challenges in cardiogenic shock
## Fluid Therapy

<table>
<thead>
<tr>
<th>Agent</th>
<th>Dose</th>
<th>Cardiac</th>
<th>Peripheral Vascular</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Heart Rate</td>
<td>Contractility</td>
<td>Vasoconstriction</td>
</tr>
<tr>
<td>Dopamine</td>
<td>1 - 4 (m/k)/min</td>
<td>1+</td>
<td>1+</td>
</tr>
<tr>
<td></td>
<td>4 - 20 (mg/kg)/min</td>
<td>2+</td>
<td>2 - 3+</td>
</tr>
<tr>
<td>Norepi</td>
<td>2 - 20 mg/min</td>
<td>1+</td>
<td>2+</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>2 - 15 (mg/kg)/min</td>
<td>1 - 2+</td>
<td>3 - 4+</td>
</tr>
<tr>
<td>Isoproterenol</td>
<td>1 - 5 mg/min</td>
<td>4+</td>
<td>4+</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>1 - 20 mg/min</td>
<td>4+</td>
<td>4+</td>
</tr>
<tr>
<td>Phenylephrine</td>
<td>20 - 200 mg/min</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Vasopressin</td>
<td>0.01 - 0.04 u/min</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Milrinone</td>
<td>37.5 - 75 mg/kg bolus; then 0.375 - 0.75 ug/kg/min</td>
<td>1+</td>
<td>3+</td>
</tr>
</tbody>
</table>

Kumar and Parrillo, 2001