Shock: Introduction

- Cellular Injury
- Multiorgans Dysfunction
- Irreversible Processing
- Unbalance Supply vs Demand
- Perfusion and Oxygenation
- Pathophysiologic Response
Shock: Introduction

- Early Diagnosis
- Appropriate Management
- First: Empiric Management
- Identification for Etiologies
- Equilibrium Supply vs Demand
- Restoration Perfusion Oxygenation
Shock: Definition

- Inadequate Organs Perfusion
- Inadequate Tissue Perfusion
- Maintain Normal Tissue and Cellular Function
Shock: General Findings

- Tachycardia
- Cutaneous Vasoconstriction
- Low Cardiac Output
- Hypotension
- Etiologies Type Specific
Pathophysiologic Response

- Normal Tissue: **Aerobic Metabolism**
- Supply vs Demand
- Tissue Hypoperfusion: Dis-Equilibrium
- Pathophysiologic Response
Physiologic Response

- Degree of Shock
- Duration of Shock
- Etiology

- Common Pathways
Start: Endpoint Response

- Impulse Stimulation
- Restoration Perfusion of Vital Organs (Heart, Brain)
- Compensated Phase
- Decompensated Phase
- Irreversible Phase
RAT HEMORRHAGIC SHOCK MODEL
24-hour Survival Following Resuscitation

MEAN ARTERIAL PRESSURE

Compensation Endpoint

% Shed Blood Return

0% 10% 20% 30% 40% 50% 100%

Compensated Decompensated Irreversible

A Transition to Acute Irreversible Shock
B Transition to Subacute Lethal Shock
Pathophysiologic Response

- Neuro-Endocrine Response
  1. Cardiovascular response
  2. Hormonal response
  3. Cellular response

- Inflammatory and Immune Response
Neuro-Endocrine Response

- Hemorrhage

- Autonomic Control of:
  1. Peripheral Vascular Tone
  2. Cardiac Contractility
  3. Hormonal Response
  4. Local Microcirculatory System
Neuro-Endocrine Response

- Hemorrhage
- Loss of Circulating Volume
- **Depend on:** Volume Rate
- Afferent Impulse
- Intrinsic Adaptive Response (CNS)
Afferent Impulse

- Loss of Circulating Volume
- Hypovolemia
  - Baroreceptor and Volume receptor
  - Normal Volume Status = Inhibit
  - Increase Sympathetic Output
  - Peripheral Vasoconstriction
Cardiovascular Response

- Hemorrhage
- Reduced Venous Return
- Reduced Stroke Volume (SV)
- Decrease Cardiac Output (CO)

\[ \text{CO} = \text{SV} \times \text{HR} \]
Cardiovascular Response

- **Stroke Volume (SV)**
  1. Preload
  2. Myocardial Contractility
  3. Afterload
Stroke Volume (SV)

- **Preload**
  1. Venous Capacitance
  2. Volume Status
  3. Venous Systemic Pressure
  4. Right Atrium Pressure
Stroke Volume (SV)

- Myocardial Contractility
  1. Myocardial Muscle Fiber Length
  2. Quality of Contraction
  3. Starling’s Law
Stroke Volume (SV)

- **Afterload**
  1. Peripheral Vascular Resistance
  2. Vasoconstriction Response
  3. Myocardial Work-Load
  4. Myocardial Oxygen Demand
When Hemorrhage Occurred!

- Decrease CO and Venous return
- **Beta-1-Adrenergic Receptor**
  - HR Contractility
- **Alpha-1-Adrenergic Receptor**
  - Peripheral Vasoconstriction
- Autoregulatory Mechanism
Hormonal Response

- Autonomic Nervous System (ANS)
- HPA-axis
- Corticotropin-Releasing Hormone
- ACTH
- Adrenal Cortex: Cortisol
Hormone: Cortisol

- Gluconeogenesis
  : Insulin resistance
- Hepatic Gluconeogenesis
  : Lipolysis
Hormonal Response

- Hemorrhage with Shock
- Reduced Renal Blood Flow
- Renin  Angiotensinogen
- ACE  Angiotensin II
- Potent Vasoconstrictor
- Aldosterone  ADH
Hormonal Response

- **Aldosterone**
  - Reabsorb Sodium and Water
  - Excrete Potassium and H-ion

- **Antidiuretic Hormone (ADH)**
  - Increase Water Permeability
  - Potent Vasoconstrictor
  - Intestinal Mucosa Barrier Dysfunction
Cellular Response

Magnitude of Shock

1. Compensated Response
2. Cellular Dysfunction
3. Cell Death
Cellular Response

- Oxydative Phosphorylation
- Aerobic Respiration
- Inadequate Oxygen Delivery
- Stop or Delay ATP Production
- Dysoxia
Shock → Cell
↓
Membrane Altered
(\(H^+\) accumulates)

Na\(^+\) enters, K\(^+\) leaves

\((Na^+ + K^+)^{ATPase}\) Activated

ATP used

Mitochondria Stimulated

Na\(^+\) in Cell and Mitochondria

ATP

Cyclic AMP

Cell Swells
Mitochondria Swell
Endoplasmic Reticulum Swells

Alternations in effects of Insulin
Glucagon?
Catecholamines?
Corticosteroids?

Toxic Factors Released

Cell Destroyed

Lysosomes Leak

Further Deterioration

Less ATP Produced by Glycolytic and Oxidative Reactions
ATP Levels Low

Metabolic Capability ↓
Cellular Response

- Dysoxia
- Anaerobic Respiration
- Cellular Glycolysis
- Pyruvate
- Lactate  Inorganic Phosphate
- Intracellular Metabolic Acidosis
Under Hypoxic Metabolism!

- Hypovolemic Hemorrhage
- Decrease ATP Production
- Cell Membrane Function
- Accumulation Intracellular Na+
- Cell Swelling
- Leakage to Extracellular K+
NORMAL (P.D.-90 mV)

mEq/L
Na⁺ = 9.9
K⁺ = 173
Cl⁻ = 3.9

HEMORRHAGIC SHOCK (P.D.-60 mV)

mEq/L
Na⁺ = 18.4
K⁺ = 162
Cl⁻ = 11.1

Neutral Na⁺ - K⁺ Exchange Pump
Electrogenic Na⁺ Pump
Relative Na⁺ Permeability

ECW
ICW
Cell Membrane
NORMAL

Vascular Tree → Arteriole → Sphincter → Capillary Bed

Interstitial Fluid ↔

Cell Fluid

HEMORRHAGIC SHOCK

Vascular Tree → Arteriole → Sphincter → Capillary Bed

Interstitial Fluid ↔

Cell Fluid
Cellular Response

- Cell Death: *Apoptosis*
- Lymphocyte  Hepatocyte  etc
- Intestinal Epithelial Cell
- **Translocation of Bacteria**
- Endotoxin
- Portal Circulation
Cellular Response

- Tissue and Cellular Hypoxia
- Anaerobic Glycolysis
- Systemic Metabolic Acidosis
- Oxy-Hemoglobin Dissociation Curve
- Erythrocyte 2,3-Diphosphoglycerate (2,3 – DPG)
FiO\textsubscript{2}

Ventilation

Perfusion
- Systemic circulation
- Pulmonary circulation

Diffusion

PaO\textsubscript{2}

Hb

Oxyhemoglobin Dissociation
HbO₂ → O₂ → Tissue, cells

%O₂ Hgb Saturation

Pao₂ (mm Hg)

↑ pH
↓ Temp.
↓ Paco₂
↓ 2,3-dpg

↑ pH
↑ Temp.
↑ Paco₂
↑ 2,3-dpg

Left Shift
Right Shift
Inflammatory Response

- Hemorrhage
- Multisystem Trauma
- Proinflammatory Mediators
- Host-Inflammatory Response
- Anti-Inflammatory Mediators
Inflammatory Response

- Prolonged Inflammatory Process
  1. Multi-Organs Dysfunction (MODS)
  2. Adult Respiratory Distress Syndrome (ARDS)
  3. Post-Traumatic Immunosuppression
Proinflammatory Mediators

- Interleukin (IL)
- IL-1  IL-2  IL-6  IL-8
- Interferon (IFN)
- Tumor Necrosis Factor (TNF)
- Platlet Activating Factor (PAF)
Complement Cascade

- Shock Injury Infection
- C3a  C4a  C5a
- Vascular Permeability
- Histamine Release
- Neutrophil Adherence
- ARDS  MODS
Shock: Etiology

- Hypovolemic or Hemorrhagic
- Cardiogenic
- Septic or Vasodilatory
- Neurogenic
- Extracardiac Obstructive
- Traumatic
Hypovolemic Hemorrhagic Shock

- Acute Circulating Volume Loss
- Baroreceptor  Chemoreceptor
- Sympathetic Activity
- Vasoconstriction
- Epinephrine  Norepinephrine
- Renin-Angiotensin Cascade
**Measurements:**
- Arterial Pressure
- A-V O₂ difference
- Arterial Blood
  - pO₂
  - pH
  - pCO₂
  - Lactate
  - Hct.
- Peripheral resistance

**Cardiac Index**

**Observations:**
- Mental status depressed
- Veins flat
- Ventilation rate increased
- Pulse weak rapid thready
- Color pale
- Skin cool clammy
Hypovolemic Shock: Etiology

- Hemorrhage
  - Trauma
  - Non-Trauma

- Non-Hemorrhage
  - External Fluid Loss
  - Interstitial Fluid Redistribution
Hemorrhage

- Acute Circulating Volume Loss
- Blood Volume = 7% BW
- BW=70 kg  Blood Volume= 5 L
- Clinical presentation depend on magnitude and rate of circulating volume loss
Classification of Shock

- Class *I* (Up to 15% BL Vol)
- Class *II* (15% to 30% BL Vol)
- Class *III* (30% to 40% BL Vol)
- Class *IV* (More than 40% BL Vol)
Class I Hemorrhage

- Little Symptom
- Mild Tachycardia
- Not Change In: BP  PP  RR

- *Not Need For Fluid Bolus Rx*
Class II Hemorrhage

- Volume Loss 750-1500 ml
- Tachycardia (HR>100) Tachypnea
- Normal BP Decrease of PP
- Little Change of Urine Output
- Mental Status Anxiety

Crystalloid +/- Blood Transfusion
Class III Hemorrhage

- Volume Loss ~ 2000 ml
- HR > 120/min Tachypnea
- Decrease in BP PP Mental Status
- Decrease in Urine Output
- *Crystalloid + Blood Transfusion*
Class IV Hemorrhage

- More than 40% of BL Vol
- Life Threatening Condition
- HR > 140/min Tachypnea
- Very low BP Narrow PP
- Negligible Urine Output Lethargy

Blood Tx + Surgical Intervention
Three-For-One-Rule (3:1)

- From Class III Hemorrhage
- Initial Fluid Resuscitation
- Interstitial vs ECF
- BW = 70 kg In Class III
- Volume Loss = 1.47 L
- Fluid Rx = 3 x 1.47 (4.4 L)
Failed Initial Fluid Rx

- Ongoing Blood Loss?
- Associated Cardiogenic Shock?
- Cardiac Tamponade?
- Tension Pneumothorax?
- Associated Neurogenic Shock?
- Previously Drugs Using?
Cardiogenic Shock

- Circulatory Pump Failure
- Decrease in CO  SV  MAP
- Tissue Hypoxia
- Normal Intravascular Volume
Measurements:

- Cardiac Index
- Arterial Pressure
- A-V O₂ difference (widened)
- Arterial Blood pO₂
- Arterial Blood pH
- Arterial Blood pCO₂
- Arterial Blood Lactate
- Arterial Blood Hct
- Urine Output
- Peripheral resistance (usually normal)

Observations:

- Mental status
  - normal or depressed
- Veins
  - full or not flat
- Ventilation
  - rate (normal or ↑)
- Pulse
  - normal or weak
  - Rate-normal or ↑
- Color
  - normal or pale
- Skin
  - normal or cool
Cardiogenic Shock: Diagnosis

- SBP < 90 mmHg at least 30 min
- Reduced Cardiac Index: < 2.2 L/min/m²
- Elevated Pulmonary Capillary Wedge Pressure (PCWP) > 15 mmHg
Cardiogenic Shock

- **Mortality Rate**: 50%-80%
- **Acute Myocardial Infarction (MI)**
- **More than 40% of LV Wall**
- **Multi-Vessels Coronary Artery Disease**
- **Myocardial Demand vs Supply**
Cardiogenic Shock: Diagnosis

- Exclude Other Possible Causes
- Cardiac Rhythm? Dysrhythmias?
- Electrocardiogram (EKG)
- Echocardiography
- Chest Radiography
- Cardiac Enzyme
Cardiogenic Shock: Etiology

- Myocardium
  - Trauma
  - Non-Trauma
- Valvular
- Intracardiac Obstruction
- Pericardium
Septic Shock

- Vasodilatory Shock
- Failure of Vasoconstriction
- Invasive Infection
- Immune Mediators Response
  - Isoform Nitric Oxide Synthase (iNOS)
  - NO: Potent Vasodilator
Septic Shock: Diagnosis

- **Severe Sepsis**
  - Fever
  - Leukocytosis
  - Tachycardia

- **Tissue Hypoperfusion**

- **Systemic Hypotension**
Neurogenic Shock

- Loss of Vasomotor Tone
- Decrease Vasoconstrict Impulse
- Decrease: Venous return  CO
- Spinal cord Injury
- Vertebral Fracture
- Obstruction Sympathetic Activity
Neurogenic Shock: Diagnosis

- Decrease BP with Bradycardia
- Warm Extremities
- Motor and Sensory Deficit
- Radiographic Vertebral Fx
Obstructive Shock

- Extra-Cardiac
- Mechanical Obstruction
- Venous Return
- Cardiac Output
Obstructive Shock

- Pericardial Tamponade
- Tension Pneumothorax
- Massive Pulmonary Emboli
- Constrictive Pericarditis
Traumatic Shock

- Systemic Response
- Soft Tissue Injury  Long-Bone Fx
- Blood Loss
- Proinflammatory Mediators
- Cellular Response
- Risk to ARDS
A classification of shock showing interrelationships among the different forms of shock. CO = cardiac output; MAP = mean arterial pressure; MODS = multiple organ dysfunction syndrome; SVR = systemic vascular resistance.
Shock: Management Aim

- Turn to Adequate **Organs**
- **Perfusion** and **Tissue**
- **Oxygenation**
Initial Diagnostic Steps

- **History**  **Physical Examination**
- Hb  CBC  Platlet  PT  PTT
- Arterial Blood Gas (ABG)
- Electrolyte  Mg  Ca
- Bun  Cr  Lactate  Glucose
- Chest Radiography  ECG
Immediate Goal Rx

- Hemodynamically Support
- Maintain Oxygen Delivery
- Reversal of Organs Dysfunction
Management in Hypovolemic Shock

- Securement Airway and Oxygenation
- Control Source of Bleeding
- Adequate Fluid Resuscitation
- Search for Others Sources
Management in Hypovolemic Shock

- Ongoing Bleeding?
- *Surgical Intervention Standby*
- *Short Shock Time*
- Consider Vasopressor Drugs
- *Hypothermia*
- Coagulopathy Acidosis
Management in Cardiogenic Shock

- Adequate Airway and Oxygenation
- Exclude Others Causes of Shock
- Equilibrium Volume Input/Output
- Adequate Myocardial O2 Delivery
- Correct Electrolyte Imbalance
Management in Cardiogenic Shock

- Correct Dysrhythmias
- *Inotropic Drugs*  *Contractility*
- *Consultation Cardiologist*
- Intra Aortic Balloon Pump (IABP)
- Coronary Angiography (CAG)
- PTCA  CABG
IABP

Balloon increases blood flow to the heart and relieves some of the workload by inflating when the heart relaxes and deflating just before the heart contracts.

This perspective shows the aorta as it extends down behind the heart.
Management in Septic Shock

- Securement Airway and Oxygenation
- Appropriate Fluid Resuscitation
- Empiric to Limit Spectrum ATB
- Adequate Drainage and Debridement
- SVO2, Lactate level, Blood sugar
- New Trial Treatment
Management in Neurogenic Shock

- Adequate Airway and Oxygenation
- Adequate Fluid Resuscitation
- Adequate Intravascular Volume
- Vasoconstrictor Agents
- Stabilization Vertebral Fx
Management in Obstructive Shock

- Adequate Airway and Oxygenation
- Appropriate Fluid Resuscitation
- **Drainage and Definite Rx**
  - Tension Pneumothorax
  - Cardiac Tamponade
Initial Fluid Resuscitation in Hemorrhagic Shock

- Isotonic Electrolyte Solution: Intravascular volume expansion
- Ringer Lactate Solution
- Normal Saline: Hyperchloremic acidosis
Initial Fluid Resuscitation in Hemorrhagic Shock

- In adult: Initial 1-2 Liters
- In pediatric: Initial 20 ml/kg

Initial Evaluation

Adequate organs and tissue perfusion
What Type of Fluid Should be Used For Initial Resuscitation?

- **Deficit Contents**
  1. Red Blood Cell Mass
  2. Plasma Volume
  3. Extracellular Fluid (ECF)
Favourable Fluid in Hemorrhagic Shock

- Warm Isotonic Electrolyte Solution

Controversy in

1. Colloid Fluid Solution
2. Albumin
After Initial Fluid Therapy

- Hemodynamic Stable
- Hemodynamic Normal
Response to Initial Fluid Therapy

- Rapid Response
- Transient Response
- Minimal or No Response
Rapid Response

- Loss Circulating Volume $< 20$
- Maintainance or Reduced Fluid
- *Not Need Bolus Fluid*
- *No Blood Transfusion*
- *Surgical Consultation*
Transient Response

- Loss Circulating Volume 20-40%
- Sequence to Deterioration
- Ongoing Blood Loss
- Inadequate Fluid Resuscitation
- Further Fluid Rx +/- Blood Tx
- Surgical Intervention
Minimal or No Response

- Even Adequate Fluid Resuscitation
- Even Blood Transfusion
- Exanguination Hemorrhage
- Cardiac Tamponade

*Emergent Surgical Intervention*
Evaluation After Initial Fluid Therapy

- **General Consideration**
- **Urine Output**
- **Acid-Base Balance**
Blood Replacement

- In Hemorrhagic shock; Most important factor: Patient Response

1. PRC  Whole Blood
2. Typing
3. Autotransfusion
4. Coagulopathy
PRC Whole Blood

- Resuscitation fluid
- Separate Component Therapy: PRC Platlet FFP
- Volume Depletion: Crystalloid Solution
Typing

- Fully Crossmatch (1 HR)
  - Rapid response and stabilization

- Type-Specific Blood (10-15 min)
  - Compatible ABO and Rh blood
  - Transient response

- Type O PRC  Rh negative
  - Exanguination hemorrhage
Autotransfusion

- Shed Blood
- Sterile Instrument
- Anticoagulant
- Retransfusion

Hemothorax
Coagulopathy

■ Massive Blood Transfusion: Dilutional of Platlet and Clotting Factors
■ Hypothermia: Platlet Aggregation
■ Clinical Evaluation
■ Coagulation Profiles: PT PTT Platlet count
Special Intrinsic Factors

- **Blood Pressure**  Cardiac Output
  \[ V = I \times R \]

- **Aging**
  1. Decrease catecholamine response
  2. Decrease cardiac compliance
  3. Atherosclerotic vascular disease
  4. Decrease pulmonary compliance
Special Intrinsic Factors

- Aging

  5. Narrow Therapeutic range especially in fluid resuscitation
  6. Decrease Physiologic reserve
  7. Rationale Morbidity  Mortality
Special Intrinsic Factors

- **Athletes**
  1. Increase **Blood volume** 15%-20%
  2. Increase **Cardiac output** 6 times
  3. Increase **Stroke volume** 50%

- **Pregnancy**
  1. Maternal Hypervolemia
  2. Fetal Hypoperfusion
Special Extrinsic Factors

- Medications
  1. Beta-adrenergic receptor blocker
  2. Calcium channel blockers
  3. Chronic diuretic
  4. Insulin overdose
  5. NSAID
Special Extrinsic Factors

- Hypothermia
  1. Inducible for Coagulopathy
  2. Alcohol: Vasodilatation

Monitor Core Temperature

Prevention is the best
Special Extrinsic Factors

- Pacemaker
- Cardiac Output = SV X HR

Unresponse for loss circulating volume
Shock: Conclusion

- Definition  Highly Suspicious
- *Early Diagnosis*
- Pathophysiologic Response
- *Empiric Management*
- *Search for Etiologies*
- *Organs and Tissue Perfusion*