SHOCK PATHOPHYSIOLOGY
Shock is a condition in which the cardiovascular system fails to perfuse tissues adequately.

- An impaired cardiac pump, circulatory system, and/or volume can lead to compromised blood flow to tissues.
Shock

Inadequate tissue perfusion can result in:

- generalized cellular hypoxia (starvation)
- widespread impairment of cellular metabolism
- Tissue damage → organ failure
- death
Pathophysiology of shock

- Impaired tissue perfusion occurs when an imbalance develops between cellular oxygen supply and cellular oxygen demand.

- All Types of shock eventually result in impaired tissue perfusion & the development of acute circulatory failure or shock syndrome.
PATHOPHYSIOLOGY

- Cells switch from aerobic to anaerobic metabolism
- Lactic acid production
- Cell function ceases & swells
- Membrane becomes more permeable
- Electrolytes & fluids seep in & out of cell
- Na+/K+ pump impaired
- Mitochondria damage cell death
COMPENSATORY MECHANISMS:

Sympathetic Nervous System (SNS)-Adrenal Response:

SNS - Neurohormonal response Stimulated by baroreceptors

- Increased heart rate
- Increased contractility
- Vasoconstriction (Afterload)
- Increased Preload
Sympathetic Nervous System (SNS)-Adrenal Response:
SNS - Hormonal: Antidiuretic Hormone

- Decrease renal perfusion
- Releases renin $\rightarrow$ angiotension I $\rightarrow$ angiotension II $\rightarrow$ potent vasoconstriction & releases aldosterone adrenal cortex $\rightarrow$ sodium & water retention ($\uparrow$ intravascular volume)
COMPENSATORY MECHANISMS:

Sympathetic Nervous System (SNS)-Adrenal Response:

SNS - Hormonal: Renin-angiotension system

- Osmoreceptors in hypothalamus stimulated ➔
- ADH released by Posterior pituitary gland ➔
- Vasopressor effect to increase BP
- Acts on renal tubules to retain water
COMPENSATORY MECHANISMS:

Sympathetic Nervous System (SNS)-Adrenal Response:

SNS - Hormonal: Adrenal Cortex

- Anterior pituitary releases adrenocorticotropic hormone (ACTH)
- Stimulates adrenal Cx to release glucorticoids
- Blood sugar increases to meet increased metabolic needs
Failure of Compensatory Response

- Decreased **blood flow** to the tissues causes cellular hypoxia
- Anaerobic metabolism begins
- Cell swelling, mitochondrial disruption, and eventual cell death

If Low Perfusion States persists:

**IRREVERSIBLE ➔ DEATH IMMINENT!!**
Stages of Shock

- **INITIAL STAGE** - tissues are under perfused, decreased CO, increased anaerobic metabolism, lactic acid is building

- **COMPENSATORY STAGE** - Reversible. SNS activated by low CO, attempting to compensate for the decrease tissue perfusion.

- **PROGRESSIVE STAGE** - Failing compensatory mechanisms: profound vasoconstriction from the SNS ISCHEMIA ➔ Lactic acid production is high ➔ metabolic acidosis

- **IRREVERSIBLE OR REFRACTORY STAGE** - Cellular necrosis and Multiple Organ Dysfunction Syndrome may occur

  DEATH IS IMMINENT!!!!
Net results of cellular shock:

- decreased myocardial contractility
- systemic lactic acidosis
- decreased vascular tone
- decrease blood pressure, preload, and cardiac output
Clinical Presentation: Generalized Shock

- **Vital signs**
  - **Hypotensive:** (may be WNL or ↑ due to compensatory mechanism) ➔ < 90 mmHg
  - **MAP** < 60 mmHg
  - **Tachycardia:** Weak and Thready pulse
  - **Tachypneic:** blow off CO2 ➔ Respiratory alkalosis
Clinical Presentation: Generalized Shock

- **Mental status: (LOC)**
  - restless, irritable, apprehensive
  - unresponsive

- **Decreased Urine output**
Shock Syndromes

- **Hypovolemic Shock**
  - blood **VOLUME** problem

- **Cardiogenic Shock**
  - blood **PUMP** problem

- **Distributive Shock**
  - [septic; anaphylactic; neurogenic]
  - blood **VESSEL** problem
HYPOVOLEMIC SHOCK

- Loss of circulating volume “Empty tank” ➔ decrease tissue perfusion ➔ general shock response

- ETIOLOGY:
  - Internal or External fluid loss
  - Intracellular and extracellular compartments

- Most common causes:
  - Hemorrhage
  - Dehydration
External loss of fluid

- **Fluid loss: Dehydration**
  Nausea & vomiting, diarrhea, massive diuresis, extensive burns

- **Blood loss:**
  trauma: blunt and penetrating
  BLOOD YOU SEE
  BLOOD YOU DON’T SEE
Internal fluid loss

- Loss of Intravascular integrity
- Increased capillary membrane permeability
- Decreased Colloidal Osmotic Pressure
  (third spacing)
Pathophysiology of Hypovolemic Shock

- Decreased intravascular volume leads to....
- Decreased venous return (Preload, RAP) leads to...
- Decreased ventricular filling (Preload, PAWP) leads to....
- Decreased stroke volume (HR, Preload, & Afterload) leads to ....
- Decreased CO leads to...(Compensatory mechanisms)

- Inadequate tissue perfusion!!!!
Assessment & Management

S/S vary depending on severity of fluid loss:

- **15% [750ml]** - compensatory mechanism maintains CO
- **15-30% [750-1500ml]** - Hypoxemia, decreased BP & UOP
- **30-40% [1500-2000ml]** - Impaired compensation & profound shock along with severe acidosis
- **40-50%** - refractory stage: loss of volume = death
Clinical Presentation Hypovolemic Shock

- Tachycardia and tachypnea
- Weak, thready pulses
- Hypotension
- Skin cool & clammy
- Mental status changes
- Decreased urine output: dark & concentrated
Initial Management Hypovolemic Shock

**Management goal:**
*Restore circulating volume, tissue perfusion & correct cause:*

- Early Recognition- Do not relay on BP! (30% fluid loss)
- Control hemorrhage
- Restore circulating volume
- Optimize oxygen delivery
- Vasoconstrictor if BP still low after volume loading
CARIOIIOGENIC SHOCK

- The impaired ability of the heart to pump blood
- Pump failure of the right or left ventricle
- Most common cause is LV MI (Anterior)
- Occurs when > 40% of ventricular mass damage
- Mortality rate of 80 % or MORE
Cardiogenic Shock: Etiologies

Mechanical: complications of MI:
- Papillary Muscle Rupture
- Ventricular aneurysm
- Ventricular septal rupture

Other causes:
- Cardiomyopathies
- Tamponade
- Tension pneumothorax
- Arrhythmias
- Valve disease
Cardiogenic Shock: Pathophysiology

- Impaired pumping ability of LV leads to...
  - Decreased stroke volume leads to.....
  - Decreased CO leads to .....
  - Decreased BP leads to.....
  - Compensatory mechanism which may lead to
  - Decreased tissue perfusion !!!!
Cardiogenic Shock: Pathophysiology

- Impaired pumping ability of LV leads to...
  - Inadequate systolic emptying leads to ...
  - Left ventricular filling pressures (preload) leads to...
  - Left atrial pressures leads to ....
  - Pulmonary capillary pressure leads to ...
  - Pulmonary interstitial & intraalveolar edema !!!!
Clinical Presentation Cardiogenic Shock

- Similar catecholamine compensation changes in generalized shock & hypovolemic shock

- May not show typical tachycardic response: if pt on Beta blockers, in heart block, or if bradycardic in response to nodal tissue ischemia

- Mean arterial pressure below 70 mmHg compromises coronary perfusion ($MAP = SBP + (2) DBP/3$)
Clinical Presentation Cardiogenic Shock

- Pericardial tamponade
  - muffled heart tones, elevated neck veins

- Tension pneumothorax
  - JVD, tracheal deviation, decreased or absent unilateral breath sounds, and chest hyperresonance on affected side
CLINICAL ASSESSMENT

- Pulmonary & Peripheral Edema
- JVD
- ↓ CO
- Hypotension
- Tachypnea,
- Crackles
- ↓ PaO2
- ↓ UOP
- ↓ LOC
Goal of management:

- Treat Reversible Causes
- Protect ischemic myocardium
- Improve tissue perfusion
- Early assessment & treatment!!!
- Optimizing pump by:
  - Increasing myocardial O2 delivery
  - Maximizing CO
  - Decreasing LV workload (Afterload)
Limiting/reducing myocardial damage during Myocardial Infarction:

- Increased pumping action & decrease workload of the heart
  - Inotropic agents
  - Vasoactive drugs
  - Intra-aortic balloon pump
  - Cautious administration of fluids
  - Transplantation

- Consider thrombolytics, angioplasty in specific cases
OPTIMIZING PUMP FUNCTION:

- Pulmonary artery monitoring is a necessity!!
- Aggressive airway management: Mechanical Ventilation
- Judicious fluid management
- Vasoactive agents
  - Dobutamine
  - Dopamine
OPTIMIZING PUMP FUNCTION (CONT.):

- Morphine as needed (Decreases preload, anxiety)
- Cautious use of diuretics in CHF
- Vasodilators as needed for afterload reduction
- Short acting beta blocker, for refractory tachycardia
DISTRIBUTIVE SHOCK

- Inadequate perfusion of tissues through maldistribution of blood flow
- Intravascular volume is maldistributed because of alterations in blood vessels
- Cardiac pump & blood volume are normal but blood is not reaching the tissues
Vasogenic/Distributive Shock

Etiologies

- Septic Shock (Most Common)
- Anaphylactic Shock
- Neurogenic Shock
Anaphylactic Shock

- A type of distributive shock that results from widespread systemic allergic reaction to an antigen
- This hypersensitive reaction is LIFE THREATENING
Antigen exposure
- body stimulated to produce IgE antibodies specific to antigen
  - drugs, bites, contrast, blood, foods, vaccines

Reexposure to antigen
- IgE binds to mast cells and basophils
- Anaphylactic response
Anaphylactic Response

- Vasodilatation
- Increased vascular permeability
- Bronchoconstriction
- Increased mucus production
- Increased inflammatory mediators recruitment to sites of antigen interaction
Clinical Presentation Anaphylactic Shock

- Almost immediate response to inciting antigen
- Cutaneous manifestations
  - urticaria, erythema, pruritis, angioedema
- Respiratory compromise
  - stridor, wheezing, bronchorrhea, resp. distress
- Circulatory collapse
  - tachycardia, vasodilation, hypotension
Management Anaphylactic Shock

- Early Recognition, treat aggressively
  - AIRWAY SUPPORT
  - IV EPINEPHRINE (open airways)
  - Antihistamines
  - Corticosteroids
  - IMMEDIATE WITHDRAWAL OF ANTIGEN IF POSSIBLE
  - PREVENTION
Management Anaphylactic Shock

- Judicious crystalloid administration
- Vasopressors to maintain organ perfusion
- Positive inotropes
- Patient education
A type of distributive shock that results from the loss or suppression of sympathetic tone.

Causes massive vasodilatation in the venous vasculature, ↓ venous return to heart, ↓ cardiac output.

Most common etiology: Spinal cord injury above T6

**Neurogenic is the rarest form of shock!**
Pathophysiology of Neurogenic Shock

- Disruption of sympathetic nervous system
- Loss of sympathetic tone
- Venous and arterial vasodilation
- Decreased venous return
- Decreased stroke volume
- Decreased cardiac output
- Decreased cellular oxygen supply
- Impaired tissue perfusion
- Impaired cellular metabolism
Assessment, Diagnosis and Management of Neurogenic Shock

PATIENT ASSESSMENT

- Hypotension
- Bradycardia
- Hypothermia
- Warm, dry skin
- ↓ CO
- Flaccid paralysis below level of the spinal lesion

MEDICAL MANAGEMENT

- Goals of Therapy are to treat or remove the cause & prevent cardiovascular instability, & promote optimal tissue perfusion
MANAGEMENT OF NEUROGENIC SHOCK

- Hypovolemia- RX with careful fluid replacement for BP<90mmHg, UO<30cc/hr Changes in LOC
- Observe closely for fluid overload
- Vasopressors may be needed
- Hypothermia- warming: avoid large swings in pts body temperature
- Treat Hypoxia
- Maintain ventilatory support
Observe for Bradycardia-major dysrhythmia

- Observe for DVT- venous pooling in extremities make patients high-risk>>P.E.
- Use prevention modalities [TEDS, anticoagulation]
Management Neurogenic Shock

- Alpha agonist to augment tone if perfusion still inadequate
  - dopamine (> 10 mcg/kg per min)
  - ephedrine (12.5-25 mg IV every 3-4 hour)
- Treat bradycardia with atropine 0.5-1 mg doses to maximum 3 mg
  - may need transcutaneous or transvenous pacing temporarily
SEPSIS

- **Systemic Inflammatory Response (SIRS) to INFECTION manifested by**: two or > of following:
  - Temp > 38 or < 36 centigrade
  - HR > 90
  - RR > 20 or PaCO2 < 32
  - WBC > 12,000/cu mm or < 4,000 > 10% Bands (immature wbc)

- **Sepsis syndrome**: SIRS with confirmed infectious process associated with organ failure or hypotension
SEPTIC SHOCK

SEPSIS WITH:

- Hypotension (SBP < 90 or > 40 reduction from baseline) despite adequate fluid resuscitation
Risk Factors Associated with Septic Shock

- Age
- Malnutrition
- General debilitation
- Use of invasive catheters
- Traumatic wounds
- Drug Therapy
Pathophysiology of Septic shock

- Initiated by gram-negative (most common) or gram positive bacteria, fungi, or viruses
  - Cell walls of organisms contain Endotoxins
  - Endotoxins release inflammatory mediators (systemic inflammatory response) causes......
  - Vasodilation & increase capillary permeability leads to
  - Shock due to alteration in peripheral circulation & massive dilation
Pathophysiology of Septic Shock

IMMUNE / INFLAMMATORY RESPONSE

Microorganisms enter body

↓

Mediator Release

↓

Activation of Complement, kallikrein / kinin / coagulation & fibrinolytic factors platelets, neutrophils & macrophages >> damage to endothelial cells.

ORGAN DYSFUNCTION
Insult, injury, or infection

Local inflammatory reaction

Release of mediators (see Table 61-19)

Systemic inflammatory response

Diffuse endothelial injury, vasodilatation, and increased capillary permeability

Progressive vasodilatation and maldistribution of blood flow

Organ hypoperfusion

Multiple organ dysfunction syndrome
Identifying Acute Organ Dysfunction as a Marker of Severe Sepsis

- Altered Consciousness
  - Confusion
  - Psychosis

- Tachypnea
  - PaO₂ < 70 mm Hg
  - SaO₂ < 90%
  - PaO₂/FiO₂ ≤ 300

- Jaundice
  - Enzymes
  - Albumin
  - PT

- Tachycardia
  - Hypotension
  - Altered CVP
  - Altered PAOP

- Oliguria
  - Anuria
  - Creatinine

- Platelets
  - PT/PTT
  - Protein C
  - D-dimer


Xigris
drotrecogin alpha (activated)
Two phases:

1. “Warm” shock - early phase
   - hyperdynamic response, VASODILATION
2. “Cold” shock - late phase
   - hypodynamic response
   - DECOMPENSATED STATE
Clinical Manifestations

EARLY... HYPERDYNAMIC STATE ... COMPENSATION

- Pink, warm, flushed skin
- Increased Heart Rate
- Tachypnea
- Massive vasodilation
- Increased CO
- Crackles
Late hypodynamic state ... decompensation:

- Vasoconstriction
- Skin is pale & cold
- Tachycardia
- Decrease BP
- Change LOC
- Decrease UOP
- Decrease CO
- Metabolic & respiratory acidosis with hypoxemia
MANAGEMENT

- Prevention !!!
- Find and kill the source of the infection
- Fluid Resuscitation
- Vasoconstrictors
- Inotropic drugs
- Maximize O2 delivery Support
- Nutritional Support
- Comfort & Emotional support
Sequelae of Septic Shock

The effects of the bacteria’s endotoxins can continue even after the bacteria is dead!!!
In summary, Treatment of Shock

- Always don’t forget your ABC
- Identify the patient at high risk for shock
- Control or eliminate the cause
- Implement measures to enhance tissue perfusion
- Correct acid base imbalance
- Treat cardiac dysrhythmias
- early intervention & always remember the prevention
- Prompt recognition and Rx make the difference outcome