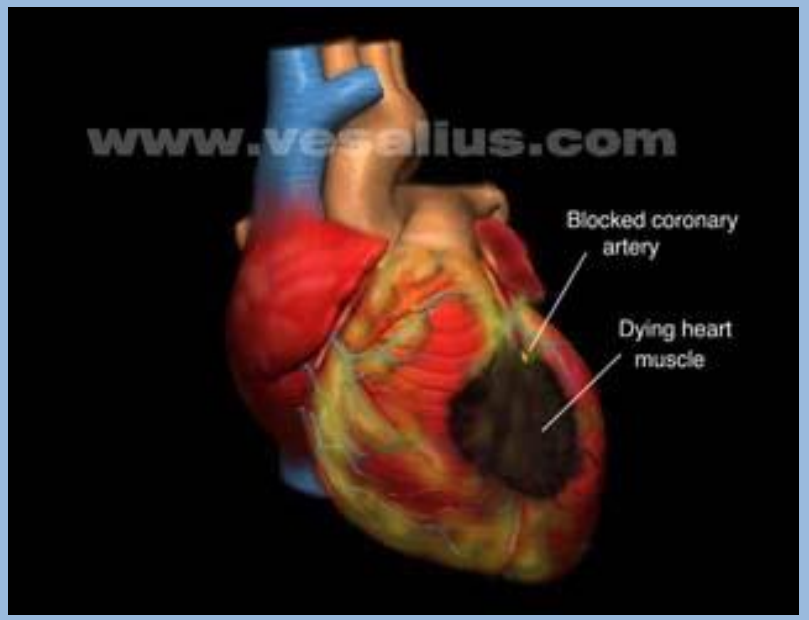
SHOCK

By
Assist. Prof. Ziad H. Abd
Specialist Urologist
Department of Surgery
Anbar Medical College

CAROIOGENIC SHOCK

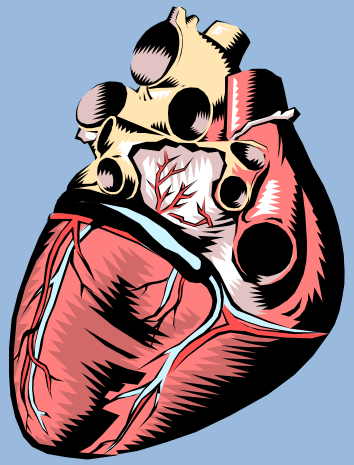
- The impaired ability of the heart to pump blood
- Pump failure of the right or left ventricle
- Most common cause is LV MI (Anterior)
- Occurs when > 40% of ventricular mass damage
- Mortality rate of 80 % or MORE



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 !!!!**

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

MANAGEMENT

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)

MANAGEMENT

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

Management Cardiogenic Shock

OPTIMIZING PUMP FUNCTION:

- **Pulmonary artery monitoring is a necessity !!**
- Aggressive airway management: Mechanical Ventilation
- Judicious fluid management
- Vasoactive agents
  - **Dobutamine**
  - **Dopamine**

Management Cardiogenic Shock

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**

Pathophysiology Anaphylactic Shock

- 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, resp. distress
- Circulatory collapse
  - tachycardia, vasodilation, hypotension

Management Anaphylactic Shock

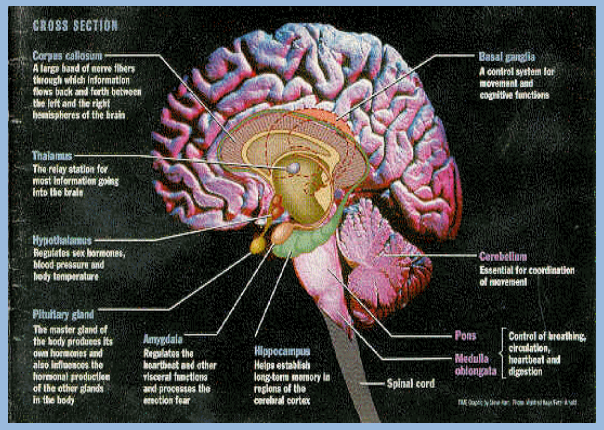
- **Early Recognition, treat aggressively**
- AIRWAY SUPPORT
- IV EPINEPHRINE (open airways)
- Antihistamines
- Corticosteroids
- IMMEDIATE WITHDRAWAL OF ANTIGEN IF POSSIBLE
- PREVENTION

Management of Anaphylactic Shock

- Judicious crystalloid administration
- Vasopressors to maintain organ perfusion
- Positive inotropes
- Patient education

NEUROGENIC SHOCK

- 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**

MANAGEMENT OF NEUROGENIC SHOCK

- **OBSERVE FOR BRADYCARDIA-MAJOR DYSRHYTHMIA**
- **Observe for DVT- venous pooling in extremities make patients high-risk>>P.E.**
- **Use prevention modalities [anticoagulation]**

...

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 associate with organ failure or hypotention**

SEPTIC SHOCK

SEPSIS + Hypotension despite adequate fluid resuscitation

- Hypotension
- SBP < 90
- or > 40 reduction from baseline

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-ve (most common) or gram +ve bacteria,** fungi, or viruses

  - Cell walls of organisms contain Endotoxins
  - Endotoxins release inflammatory mediators (SIR) 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

Gram Positive Organisms ⬇ Gram Negative Organisms

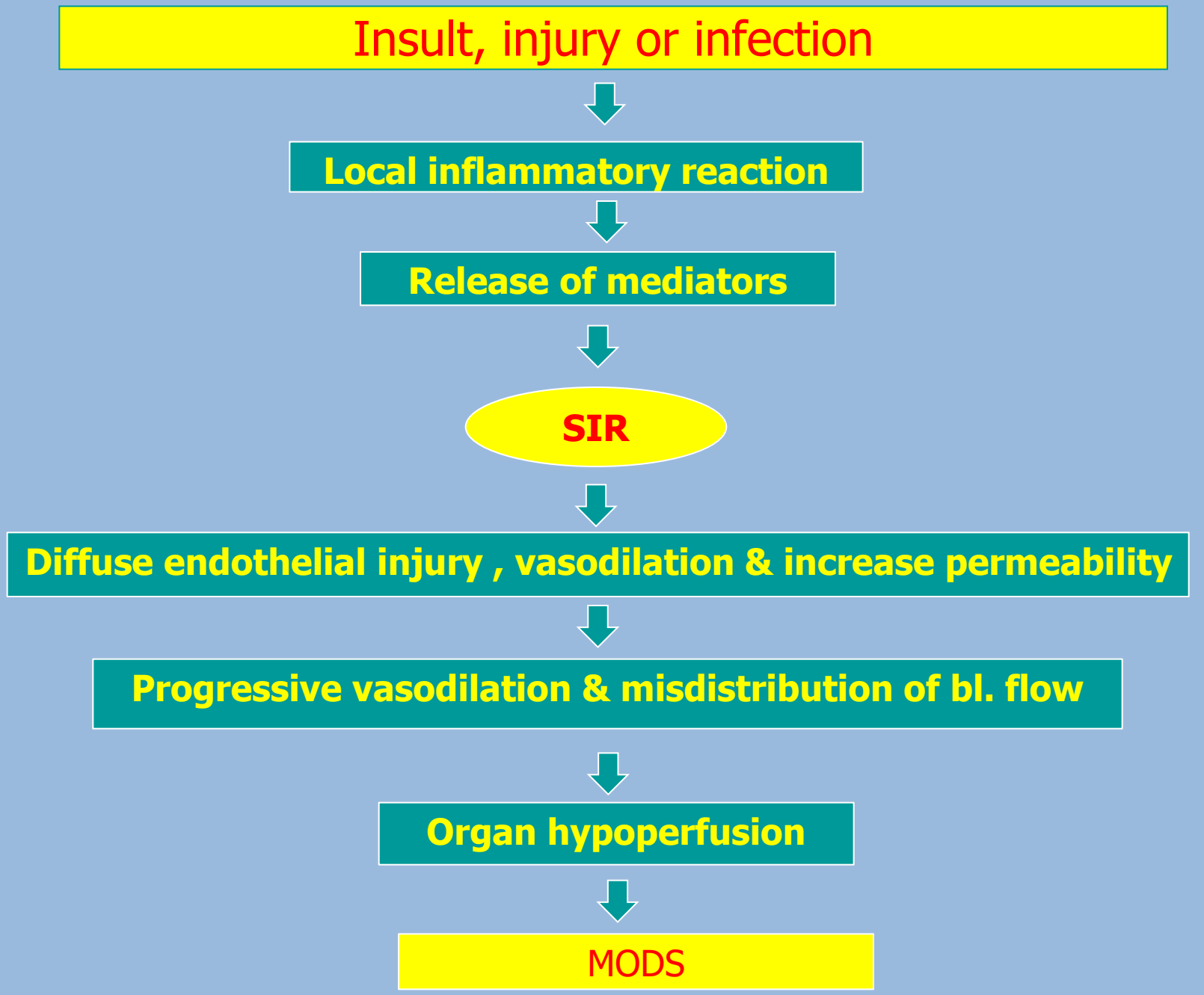
Mediator Release

⬇

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
SIR
Diffuse endothelial injury , vasodilation & increase permeability
Progressive vasodilation & misdistribution of bl. flow
Organ hypoperfusion
MODS

Identifying Acute Organ Dysfunction as a Marker of Severe Sepsis

Altered Consciousness
Confusion
Psychosis

Tachypnea
PaO_2 < 70 mm Hg
SaO_2 < 90%
$PaO_2/FiO_2 \leq 300$

Jaundice
↑ Enzymes
↓ Albumin
↑ PT

Tachycardia
Hypotension
Altered CVP
Altered PAOP

Oliguria
Anuria
↑ Creatinine

↓ Platelets
↑ PT/APTT
↓ Protein C
↑ D-dimer

1. Balk RA. *Crit Care Clin*. 2000;16:337-352.

Clinical Presentation Septic Shock

- **TWO PHASES:**
  - **"Warm" shock - early phase**
    - **hyperdynamic response, VASODILATION**
    - **COMPENSATION STATE**
  - **"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**

Clinical manifestation

- Late hypodynamic state ---- decompansation:

- **Vasoconistriction**
- **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**