SHOCK

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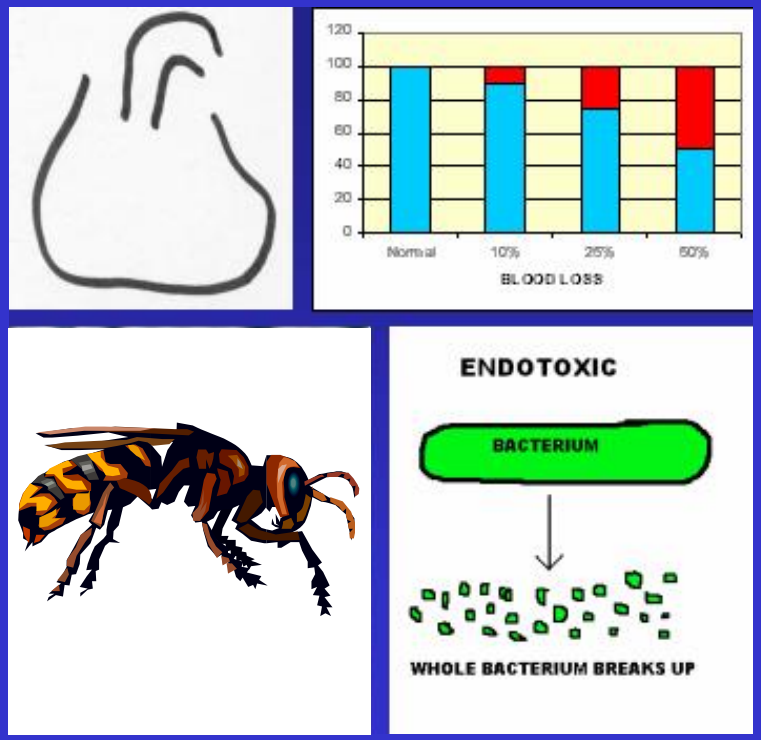
SHOCK

- Emotional/psychological
- Electrical
- Cardiovascular

This presentation concerns acute circulatory failure: cardiovascular shock.



What is Shock?

- is a systemic state of low tissue perfusion, which is inadequate for normal cellular respiration.
  - Tissue perfusion is dependent on SVR and CO
  - Imbalance between oxygen delivery and oxygen consumption which leads to:
    - cell death,
    - end organ damage,
    - multi-system organ failure,
    - and death

SHOCK

- The most common cause & most important cause of death in surgical pts.

- Death may occur:
  - Rapidly : due to profound state of shock
  - Delayed : due to organ ischemia or reperfusion injury

SHOCK

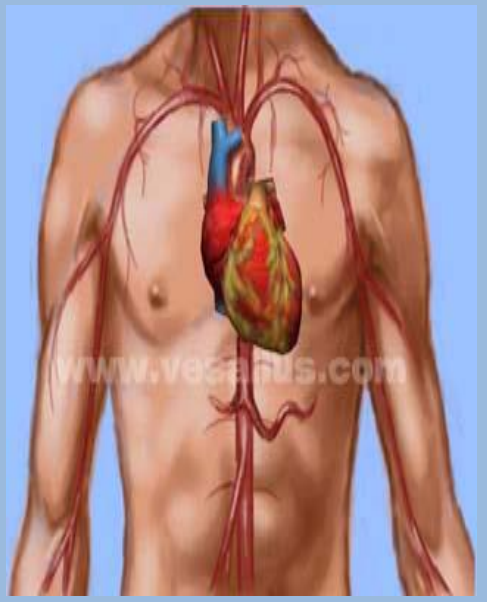
is a condition in which the cardiovascular system fails to perfuse tissues adequately

- 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
- 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 OF SHOCK

Levels of shock effects:

- cellular
- microvascular
- SYSTEMIC:
  - CVS
  - RESPIRATORY
  - RENAL
  - ENDOCRINE
- ISCHEAMIC-REPERFUSION SYNDROM

PATHOPHYSIOLOGY
CELLULAR LEVEL

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

↓

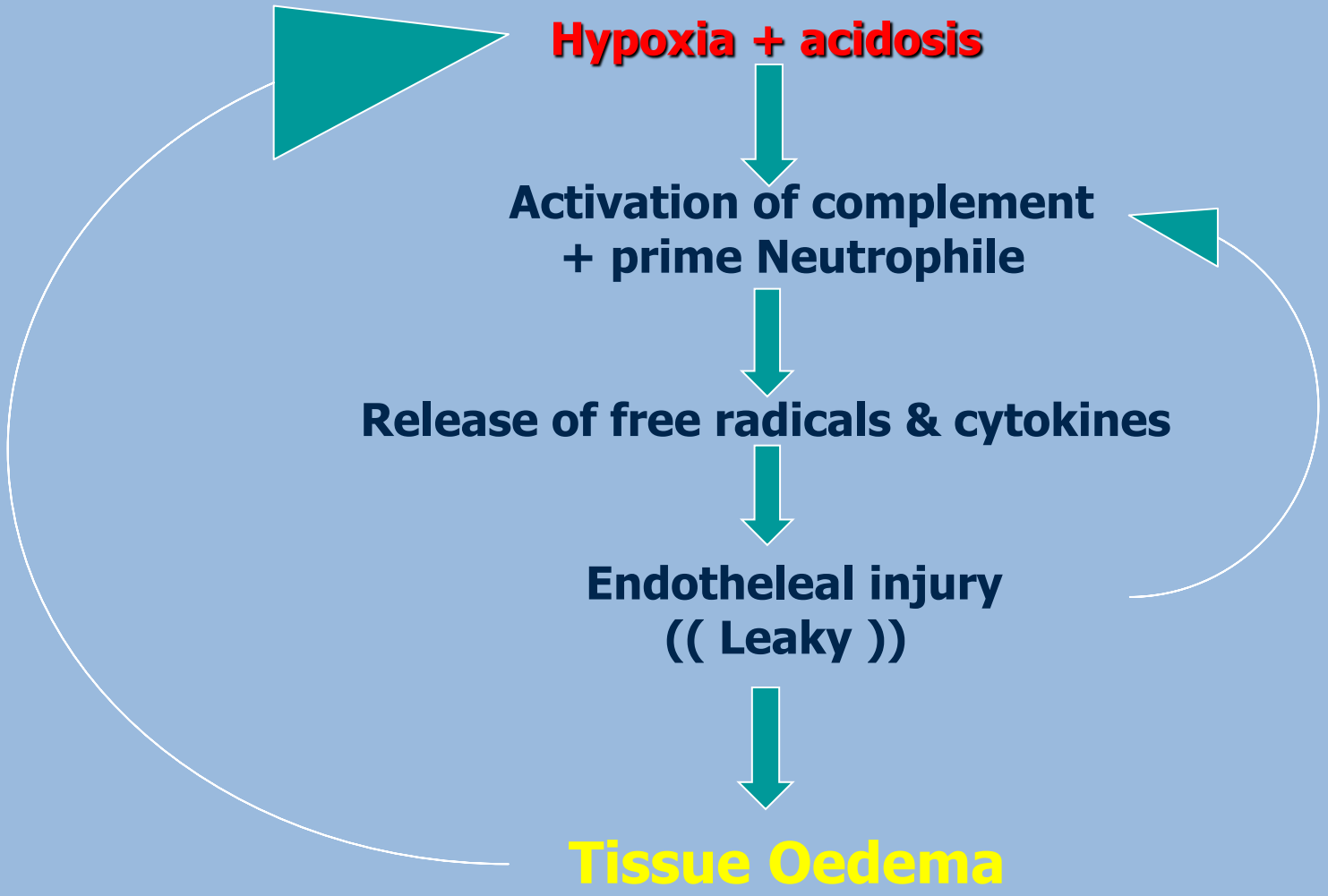
Na+/K+ pump impaired

↓

mitochondria damage

cell death

PATHOPHYSIOLOGY
MICROVASCULAR LEVEL



PATHOPHYSIOLOGY
SYSREMIC LEVEL

COMPENSATORY MECHANISMS:
Sympathetic Nervous System (SNS)-Adrenal Response

- SNS - Neurohormonal response Stimulated by baroreceptors

✓ Increased heart rate

✓ Increased contractility

✓ Vasoconstriction (Afterload)

✓ Increased Preload

COMPENSATORY MECHANISMS:
Sympathetic Nervous System (SNS)-Adrenal Response

- SNS - Hormonal: Renin-angiotension system

  Decrease renal perfusion →

  Releases renin → angiotension I

  angiotension II → potent vasoconstriction &

  releases aldosterone adrenal cortex →

  sodium & water retention (↑ intravascular volume)

COMPENSATORY MECHANISMS:
Sympathetic Nervous System (SNS)-Adrenal Response

- SNS - Hormonal: Antidiuretic Hormone

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

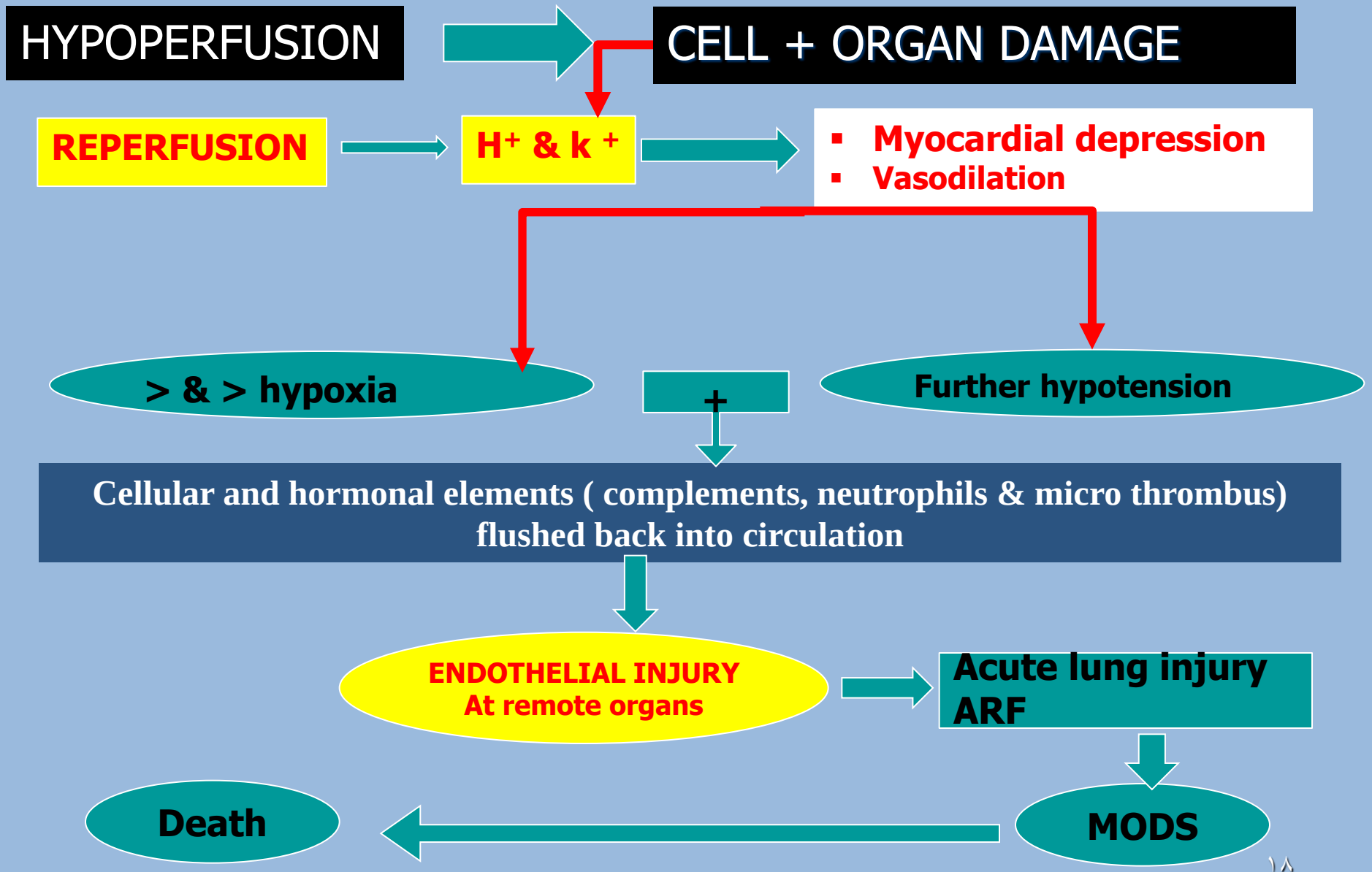
DEATH IS IMMINENT!!!!

Pathophysiology Systemic Level

Net results of cellular shock:

- £ decreased myocardial contractility
- £ systemic lactic acidosis
- £ decreased vascular tone
- £ decrease blood pressure, preload, and cardiac output

ISCHAEMIC – REPEFUSION SYNDROM



ISCHAEMIC – REPEFUSION SYNDROM

Only be attenuated by reducing the

- extent of tissue hypoperfusion
- duration of tissue hypoperfusion

Clinical Presentation: Generalized Shock

VITAL SIGNS

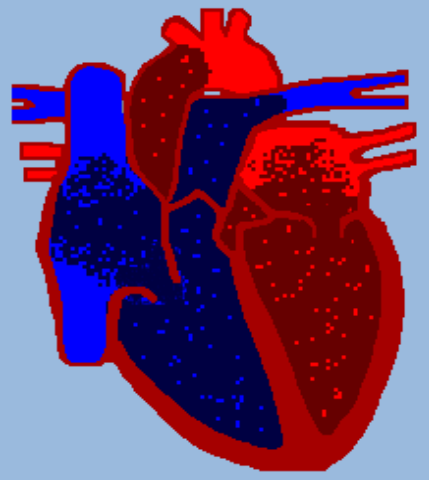
- **Hypotensive**: (may be WNL or ↑ due to compensatory mechanism) → < 90 mmHg
- **MAP** < 60 mmHg
- **Tachycardia**: Weak and Thready pulse
- **Tachypneic** : blow off CO2 → Respiratory alkalosis

Clinical Presentation: Generalized Shock

- **Mental status: (LOC)**
  restless, irritable, apprehensive
  unresponsive
- **Decreased urine output**
  < 30ml / hr

TYPES OF 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**

  → decrease tissue perfusion → general shock response

- **ETIOLOGY:**

  Internal or External fluid loss

  Most common causes:

  Hemmorhage

  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% - refactory 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!
- Control hemorrhage
- Restore circulating volume
- Optimize oxygen delivery
- Vasoconstrictor if BP still low after volume loading