Pathophysiology of circulatory shock

Dr Badri Paudel

Definition

- Inadequate perfusion (oxygen supply) of tissues, resulting in:
  - Organ dysfunction
  - Cellular and organ damage

And if not quickly corrected...

- Death

Characteristics of circulatory shock

Complex clinical syndrome encompassing a group of conditions with variable hemodynamic manifestations

Common denominator is **generalised inadequacy of blood flow** through the body; hypoperfusion **compromises the delivery of oxygen** and nutrients and the removal of metabolites; **tissue hypoxia** shifts metabolism to anaerobic pathways with **production of lactic acid**

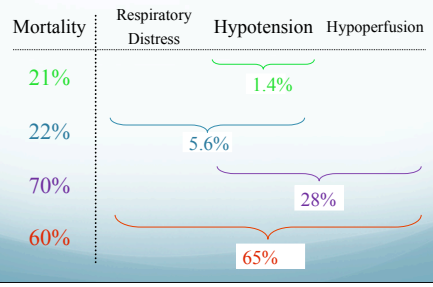
if shock is not corrected it leads to:

a) cell dysfunction
b) irreversible multiorgan insufficiency
d) death

Clinical features of shock

- drop of systolic blood pressure (BP < 90 mmHg) in hypertonic patients: decrease of 50 mmHg
- low cardiac output and tachycardia,pulse weak, thready-compensation of MAP by Baroreceptor
- Respiration:Shallow, irregular, labored
- vasoconstriction: skin and splanchnic areas
- oliguria (< 20 ml/hour)
- cold wet skin
- constriction of superficial veins
- marked muscle weakness
- usualy ↓ body temperature (except septic shock)
- disorientation
- metabolic acidosis

SHOCK Registry JACC Sept. 2000, Supp. A
Spectrum of Clinical Presentations

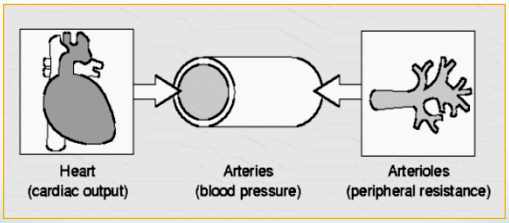


Emergency Treatment

- Evaluate vital signs
  - BP, Respiration Rate, Pulse Oximeter, Temp.
- Control bleeding
- Prevent loss of body heat

Blood pressure → Tissue perfusion

Cardiac output x **Vascular resistance**



MAP = CO X TPR
CO = SV X HR
SV = EDV - ESV

Factors determining tissue perfusion

A. cardial: cardiac output

B. vascular: changes in vascular resistance

regulation of vascular tone:
- tonic sympathetic activity
- systemic catecholamines
- myogenic response - constant tissue blood flow during changed perfusion pressure
- metabolic autoregulation - vasodilatory substances
- endothelial NO

C. humoral: renin, vasopresin, prostaglandins, kinins, atrial natriuretic factor

Factors determining microcirculation:
- adhesion of leukocytes and platelets on epithelial lesions
- intravascular coagulation
- constriction of precapillary and postcapillary vessels
- intense hypoxia → vasodilation of arteriols, venoconstriction continues → intravascular fluid loss
- ↑ capillary permeability → tissue edema

Causes of Shock: Classification (the real list)

- Low Output Circulatory Failure
  - Hypovolemic shock (too little volume)
  - Cardiogenic shock (pump failure)
  - Obstructive shock
  - Distributive shock: Venous pooling
- High Output Circulatory Failure
  - Distributive Shock: Sepsis, toxic shock, anaphylaxis:

Hypovolemic Shock

- CO reduced due to loss of intravascular VOLUME
  - Reduced venous return
- Causes
  - Most often, blood loss (hemorrhage)
  - Dehydration
  - Burns
  - Fluid lost into peritoneal cavity w/ pancreatitis

*MAP = CO * TPR*

Classic Criteria for Diagnosis of Cardiogenic Shock

1. **Systemic Hypotension**
   systolic arterial pressure < 80mmHg
2. **Persistent Hypotension** <90 mmHg
   at least 30 minutes
3. **Reduced Systolic Cardiac Function** <2.2 li/min.m2
   Cardiac index < 1.8 1 x m²/min
4. **Tissue Hypoperfusion**
   Oliguria, cold extremeties, confusion
5. **Increased Left Ventricular Filling** >15 mmHg
   Pulmonary capillary wedge pressure > 18mmHg

Cardiogenic Shock

Pathophysiology

Disorders that can result in the acute deterioration of cardiac function and can lead to cardiogenic shock include:

1) Myocardial infarction/Myocardial ischaemia

2) Sustained arrhythmia: heart block, ventricular tachycardia

3) obstructive and regurgitant lesions of intracardial blood flow mechanics

Autopsy studies have shown that cardiogenic shock is associated with loss of more than 40% of the left ventricular myocardial function

Mortality rate from cardiogenic shock is high (80%)

13

Causes Of Cardiogenic Shock:

1) Systolic Dysfunction

Myocardial contractility is abated

Systolic dysfunction mainly due to MI

Another cause for systolic dysfunction is severe myocarditis

2) Diastolic Dysfunction

Increased left ventricular diastolic chamber stiffness contributes to cardiogenic shock

Caused by ventricular hypertrophy

14

3) Valvular Dysfunction

Acute mitral valve obstruction by means of a left atrial thrombus results in decreased cardiac output

Aortic valve regurgitations reduce forward flow f blood and increase EDV and pressure ⇒ aggravate shock

4) Cardiac Arrhythmias

Bradycardia cause a reduction in cardiac output thereby aggravating shock

5) Greatly Increased Afterload/preload

Aortic or mitral stenosis

15

Symptoms

Clinical evidence of hypoperfusion (low cardiac output)

Low urine output

Cool extremities, ashen and cyanotic

Systemic hypotension ultimately develops (systolic pressure < 90 mm Hg and MAP decreases by 30 mm Hg)

Hypoperfusion propagates to tissues

Exertional dyspnea or dyspnea at rest, palpitations and generalized anxiety

16

Cardiovascular Mechanics Of Cardiogenic Shock

The left ventricle is able to eject less blood volume per beat

The stroke volume is decreased due to weakened ventricles

To compensate for the reduced stroke volume, venous return is increased

The EDV increases

However, this creates a left ventricular diastolic filling pressure

This results in backflow from the left ventricle to the lungs

This causes pulmonary oedema (this explains dyspnea)

17

Due to low cardiac output (because of low stroke volume), the tissues increase their oxygen extraction

This contributes to substantial arterial oxygen desaturation

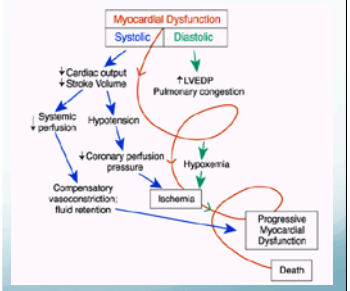
Other Effects

Myocardial ischemia is further exacerbated by compromised myocardial perfusion due to hypotension and tachycardia

18

Schematic

- LVEDP elevation
- Hypotension
- Decreased coronary perfusion
- Ischemia
- Further myocardial dysfunction
- Neurohormonal activation → Vasoconstriction
- Endorgan hypoperfusion

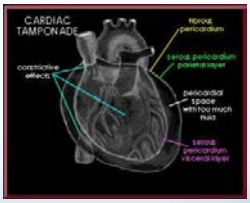


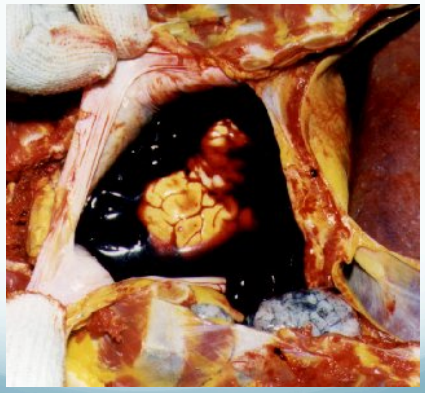
Obstructive Shock

- **factors extrinsic to cardiac valves and myocardium**
- CO reduced by vascular obstruction:
  - Obstruction of Venous return (vena cava syndrome – usually neoplasms)
  - Compression of the heart (pericardial tamponade*)
  - Outflow from heart (Massive pulmonary embolism, aortic dissection)

Pericardial Tamponade

- Life threatening condition caused by fluid (blood, effusion fluid) under pressure around the heart.
- Decreases CO by decreasing filling
- Causes include pericarditis and MI



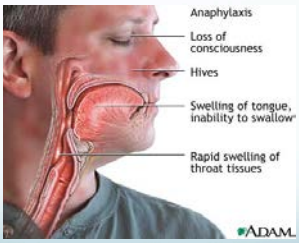


Distributive Shock

pathologic redistribution of intravascular fluid volume

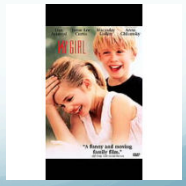
- *septicaemia*: endotoxic, secondary to specific infection
- *anaphylactic*
- Neurogenic-spinal cord trauma

Anaphylaxis



Anaphylactic Shock

- Histamine triggers vasodilation, increased capillary permeability
  - Can lead to low-output distributive shock



Aetiology of Septic Shock

- Usually severe infection, bacteremia
- Gram neg organisms
- May occur as aftermath of cardiogenic or hypovolaemic shock
- Unlike other types, often associated with other pathological complications eg ac respiratory failure, pulmonary edema, DIC

Septic shock

Typical causes: peritonitis, gangrenous infection, pyelonephritis

Special features:

1. high fever
2. marked vasodilatation (inflammation)
3. ↑ or normal CO: vazodilatation, ↑ metabolic rate
4. disseminated intravascular coagulation → clotting factors to be used up → hemorrhages occur into many tissue (GIT)

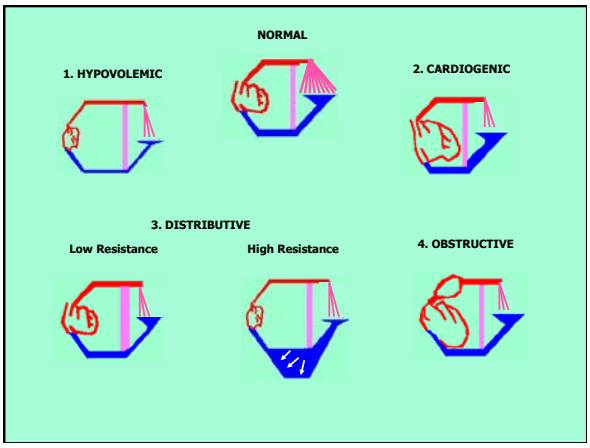
IL-1 and TNF: PGE_2, leukotrienes and NO
- vascular relaxation
- ↑ endothelial permeability (deficit of intravascular volume)
- ↓ myocardial contractility

Early stage: no signs of circulatory insufficiency

Progression of infection: circulatory disorders becomes

Bacterial toxins → **deterioration of circulation** → end-stage is not greatly different from the end-stage of hemorrhagic shock **(hypodynamic stage)**

Death: - hypotension
- multiorgan failure



Pathogenesis of circulatory shock

Usually results from **inadequate cardiac output (CO)**
Any factor reducing CO will likely lead to shock

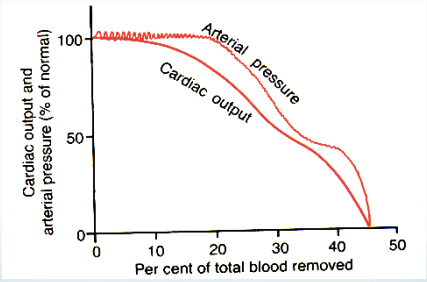
1. **Cardiac abnormalities**
   decreased ability of the heart to pump blood
   - myocardial infarction
   - toxic states of heart
   - severe heart valve dysfunction
   - arrhythmias
2. **Decreased venous return**
   - diminished blood volume
   - decreased vasomotor tone
   - obstruction to blood flow at some points in the circulation

Stages of shock

1. Nonprogressive stage (compensated)

Compensatory mechanisms **(negative feedback)** of the circulation can return CO and BP to normal levels

- **baroreceptor reflexes** → sympathetic stimulation → constrict arteriols in most parts of the body and venous reservoirs → **protection of coronary and cerebral blood flow**
- **angiotensin-aldosteron, ADH** → vasoconstriction, water and salt retention by the kidneys
- **absorption of fluid from Interstitial Fluid and GIT, increased thirst**



2. Progressive shock

- circulatory system themselves begin to deteriorate, without therapy shock becomes steadily worse until death

- **positive feedback mechanisms** are developed and can cause **vicious circle** of progressively decreasing CO

Cardiac depression - ↓ coronary blood flow, ↓ contractility

Vasomotor failure - ↓ cerebral blood flow

Release of toxins by ischemic tissues: histamine, serotonin, tissue enzymes

Intestines hypoperfusion → mucosal barrier disturbance → **endotoxin** formation and absorption → vasodilatation, cardiac depression

Vasodilation in precapillary bed

Generalised cellular deterioration: ↑ K^+ , ↓ ATP, release of hydrolases – first signs of multiorgan failure

Progressive Shock

- Compensatory mechanisms inadequate to compensate for loss of blood volume
- Cardiac circulation compromised → decreased heart function → decreased flow
- Positive feedback cycle: Shock worsens → less compensation → shock worsens...
  - Clotting in small vessels
  - Vessels dilate and permeability increases

3. Irreversible shock

- despite therapy circulatory system continues to deteriorate and **death ensues**
- marked hypoxic tissue damage
- endothelial dysfunction → adhesive molecules, neutrophils, macrophages → **inflammation**
- progressive acidosis
- microcirculation failure → plasma proteins leak to interstitium
- advanced disseminated intravascular coagulation

Irreversible Shock

- Cardiac and other tissue irreversibly damaged
- Characterized by:
  - Decreasing cardiac function
  - Progressive blood vessel dilation
  - Progressive increase in vessel permeability

Possible Mechanism in Development Irreversible Shock

Shock Stimulus
↓
Lysosomal Activation, Release Proteases
↓ ↑
Splitting of Plasma Proteins
↓
Vasoactive Peptides, Amines etc
↓
Hypotension, Fluid Loss
↓
Irreverisible SHOCK

Cell dysfunction

prolong tissue hypoperfusion → cell membrane lesion, lysosomal enzymes → **cell death**

mechanisms: hypoxia, inflammatory mediators, free radicals

Multiorgan failure

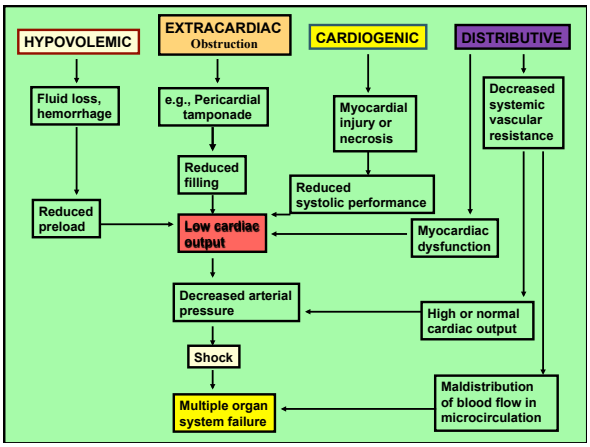
Kidney

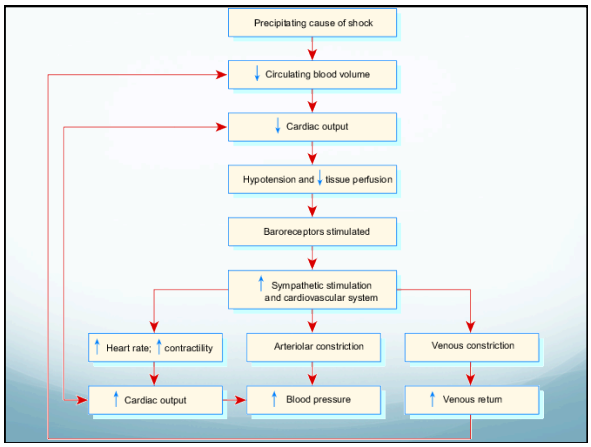
- ↓ blood flow (to 10%) → ↓ GF → **oliguria**
- ischemia → **acute tubular necrosis**
- countercurrent mechanism failure
- marked lesions → **acute renal failure**

Lungs

- disturbances of pneumocytes and endothelium
- accumulation of Tr, Neu in pulmonary circulation → release of proteases
- ↑ leukotriens and free radicals
- ↑ permeability - ↓ surfactant, edema and hemorrhagies

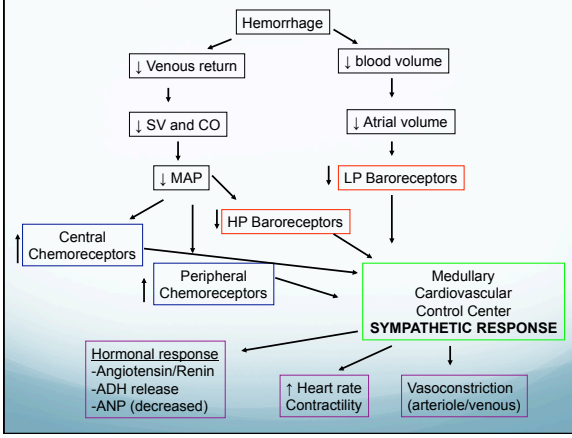
⇒ **respiratory insufficiency (ARDS)**



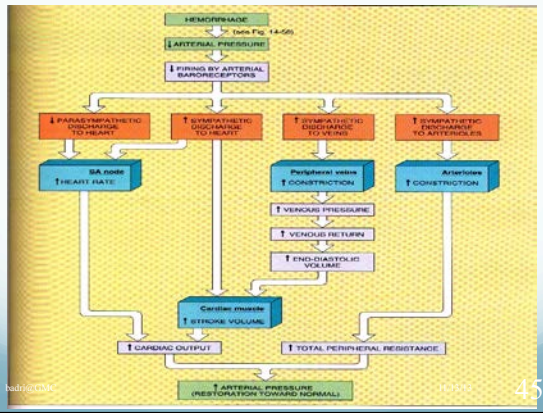


Additional Compensatory Mechanisms

- Renin-Angiotensin Mechanism
  - AII : vasoconstrictor
  - Aldosterone: Water conservation
- ADH: Water retention and thirst



REGULATION OVERVIEW



Treatment of Shock

- Resuscitation-A,B,C's
- Early & vigorous infusion fluids (crystalloids, colloids, plasma, blood)
- Monitoring- HR, BP, RR, UO, mental state, Temp, CVP (R ventricular preload), PCWP (LEDV)

Specific Treatment

Drugs

- Alpha-vasoconstrictors eg meteraminol, methoxamine, noradr- (disadv further restrict peripheral tissue perfusion, increase cardiac afterload)
- Inotropes eg adr, noradr, dobutamine, dopamine (if evidence myocardial involvement)

Intra-aortic Balloon Counterpulsation (IABC)

Prognosis Hypovolaemic Shock

Depends on

- Underlying cause
- Severity
- Duration
- Patient's age
- Pre-existing disease