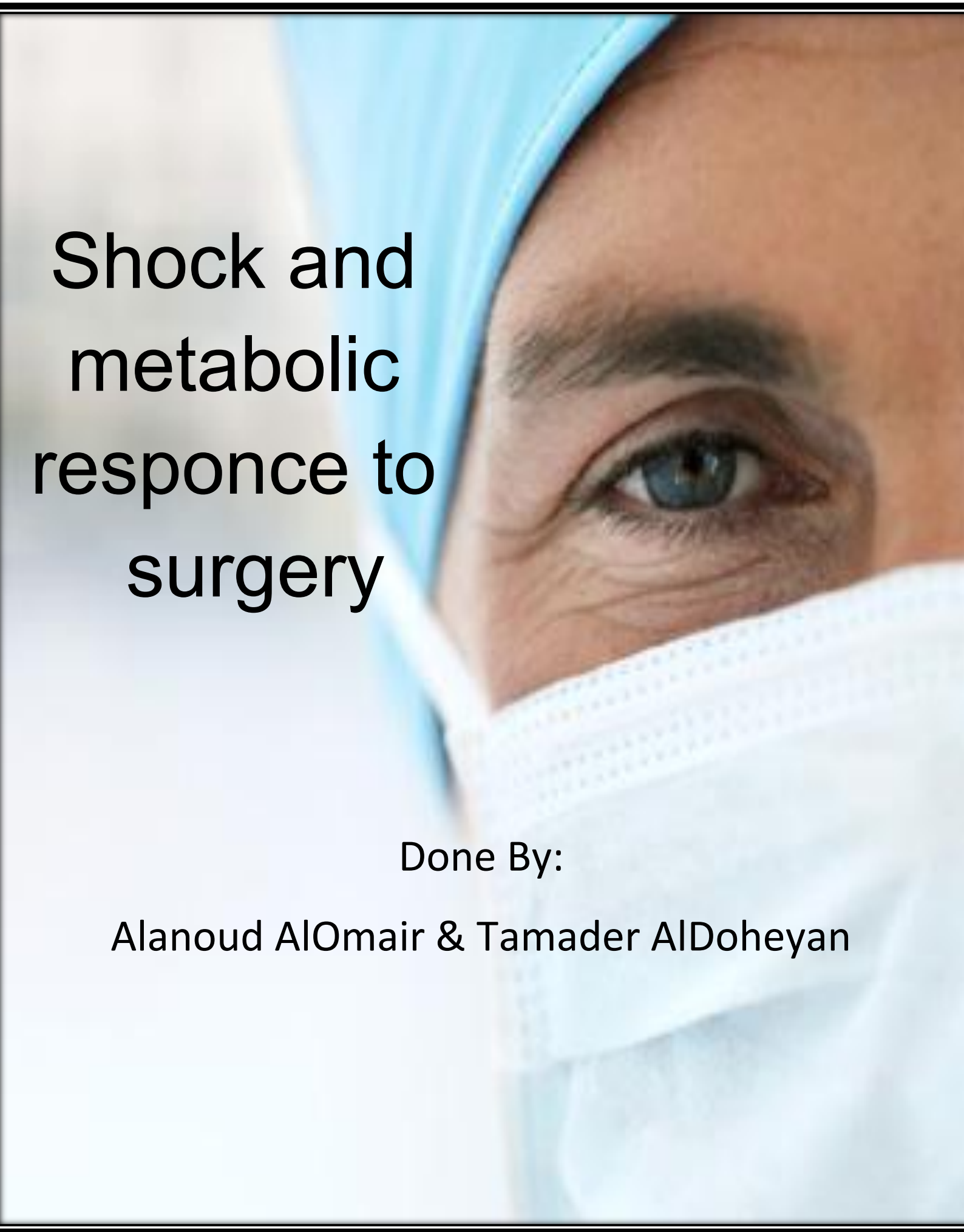
430 Surgery Team



Shock and metabolic responce to surgery

Done By:

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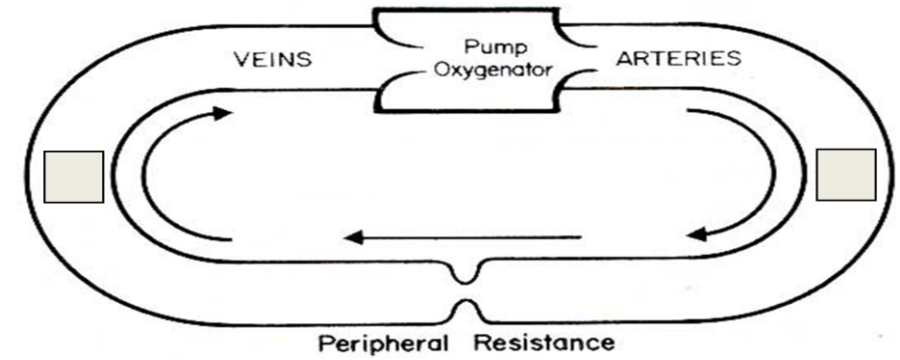
Changes in all of these elements regulate BP and perfusion:

- Intravascular volume
- Heart
- Arteriolar bed
- Capillary exchange network
- Venules
- Venous capacitance circuit
- Large vessel patency



- Decreased peripheral resistance

  Decreased arterial blood pressure (MAP = CO X PR)

- Increased peripheral resistance
  - *Decreased venous return*
  - *Decreased EDV*
  - *Decreased SV*
  - *Decreased CO (CO = HR X SV)*
  - *Decreased arterial blood pressure (MAP=CO X PR)*

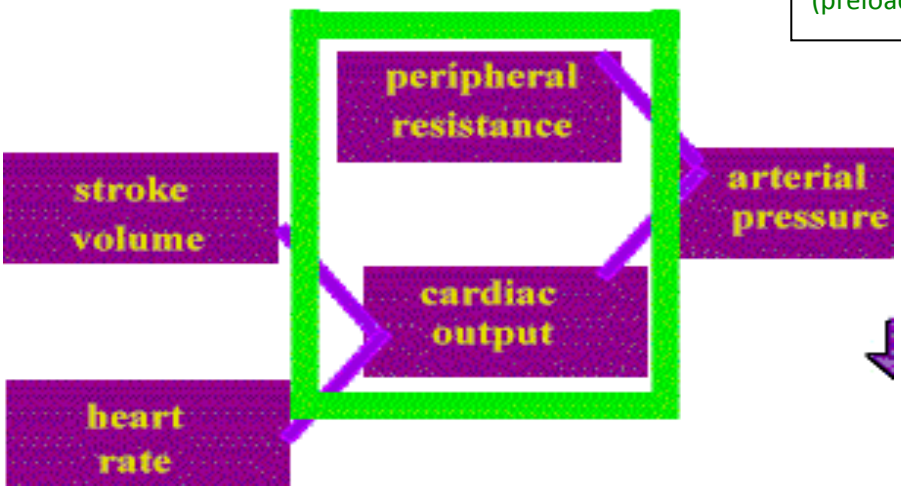
PR: The opposition of blood as it flows through blood vessels, caused by friction between the blood and the walls of blood

The ↓ in BP results in RVP ni↑

Heart Rate X Stroke Volume = Cardiac Output

Cardiac Output X Peripheral Resistance = Arterial Pressure

Stroke volume depends mainly on contractility and end diastolic pressure (preload)



HOW DOES *INTRAVASCULAR VOLUME* AFFECT BP AND PERFUSION?

- Alters mean blood pressure

  Decrease in intravascular volume=decreased BP

- Alters venous return to the heart

Decrease in intravascular volume ➔ Decreased venous return ➔Decreased end diastolic volume

- CO = HR x SV
- MAP= CO X SVR

SVR = systemic vascular resistance or TPR= total peripheral resistance

- How can intravascular volume be lost?

  Examples:

  - Bleeding
  - Failure to rehydrate
  - Loss of third space fluids (sweating)

*HOW DOES **CARDIAC FUNCTION** ALTER BP AND PERFUSION?*

Cardiac output is the result of:

- Heart rate
- Contractility
- Loading conditions

Examples of changes that can alter cardiac output

- Heart rate (bradycardia or tachycardia)
- Contractility (MI or cardiomyopathy)
- Load (histamine release: vasodilation)

HOW DOES THE *RESISTANCE CIRCUIT: ARTERIOLAR BED* AFFECTS BP AND PERFUSION?

Decrease in arteriolar tone produces:

- Hypotension
- Decreased perfusion to vital organs

Increases in tone will prevent optimal cardiac performance (increased afterload=decreased contractility)

HOW DOES THE *CAPILLARY EXCHANGE NETWORK* AFFECT BP AND PERFUSION?

- Largest area of the vascular tree
- Site of exchange of nutrients, electrolytes and fluids

These 2 points are characters of THE CAPILLARY EXCHANGE NETWORK

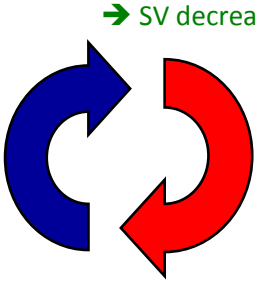
- Alterations in microvascular integrity (e.g., capillary leak syndrome) result in loss of intravascular volume
- Blockage of or shunting away from small vessels leads to decreased tissue perfusion

HOW DOES THE VENOUS CAPACITANCE CIRCUIT AFFECT BP AND PERFUSION?

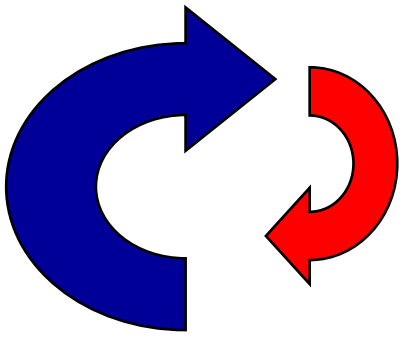
It is the portion of the circulatory system contains 80% of the intravascular volume

Decrease in effective circulating blood volume and MAP caused by:

- Decreases in venous tone
- Increases in venous vascular capacitance➔ venous return decreases ➔ EDV decreases ➔ SV decreases



NORMAL



INCREASED VENOUS CAPACITANCE
Decreased effective blood volume
Decreased MAP

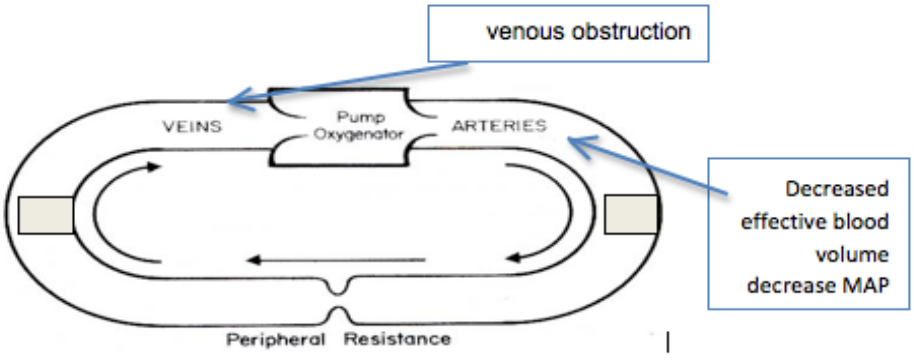
HOW DOES LARGE VESSEL PATENCY AFFECT BP AND PERFUSION?

Obstruction of the systemic or pulmonic circuit will decrease ventricular ejection and systemic perfusion

Venous obstruction will decrease venous return

Examples of obstructive shock:

- Massive pulmonary embolism
- Venous occlusion



DEFINITION OF SHOCK

State of altered tissue perfusion severe enough to induce derangements in normal cellular metabolic function. The definition implies that the normal balance between perfusion and cellular needs becomes disrupted, leading to pathophysiologic changes.

TYPES OF SHOCK*

Type of shock	Clinical causes	Primary mechanism
Hypovolemic	Volume loss	Exogenous blood, plasma, fluid or electrolyte loss
Cardiogenic	Pump failure	Myocardial infarction, cardiac arrhythmias, heart failure
Distributive, occurs due to vasodilation or leakage of fluids outside the vessels, (septic, neurogenic and anaphylactic)	Increased venous capacitance or arteriovenous shunting	Septic shock, spinal shock, autonomic blockade, drug overdose
Obstructive	Extra-cardiac obstruction of blood flow	Vena caval obstruction (such as lung tumor), cardiac tamponade, pulmonary embolism, aortic compression or dissection

***MORE THAN ONE TYPE MAY BE PRESENT**

The clinical signs and symptoms of shock relate to decreased organ perfusion

- Mental status changes: decreased cerebral perfusion
- Decreased urine output: decreased renal perfusion
- Cold clammy extremities: Decreased perfusion to the skin due to diverted blood flow
- EKG changes:

1. May indicate myocardial ischemia
2. May be primary event (cardiogenic shock) or due to decreased myocardial perfusion due to shock from other causes

At first ➔ tachycardia and oliguria

Unconsciousness and hypotension occur later because the body is preserving blood flow to the brain and heart (the most imp vital organs).

HEMODYNAMIC PARAMETERS THAT MAY INDICATE SHOCK

- Heart rate: Initial tachycardia (attempt to increase CO), heart is trying to compensate
- Rhythm: Regular and tachycardic
- Blood pressure: Low ➔BP usually decreases later
- Cardiac output: Usually low

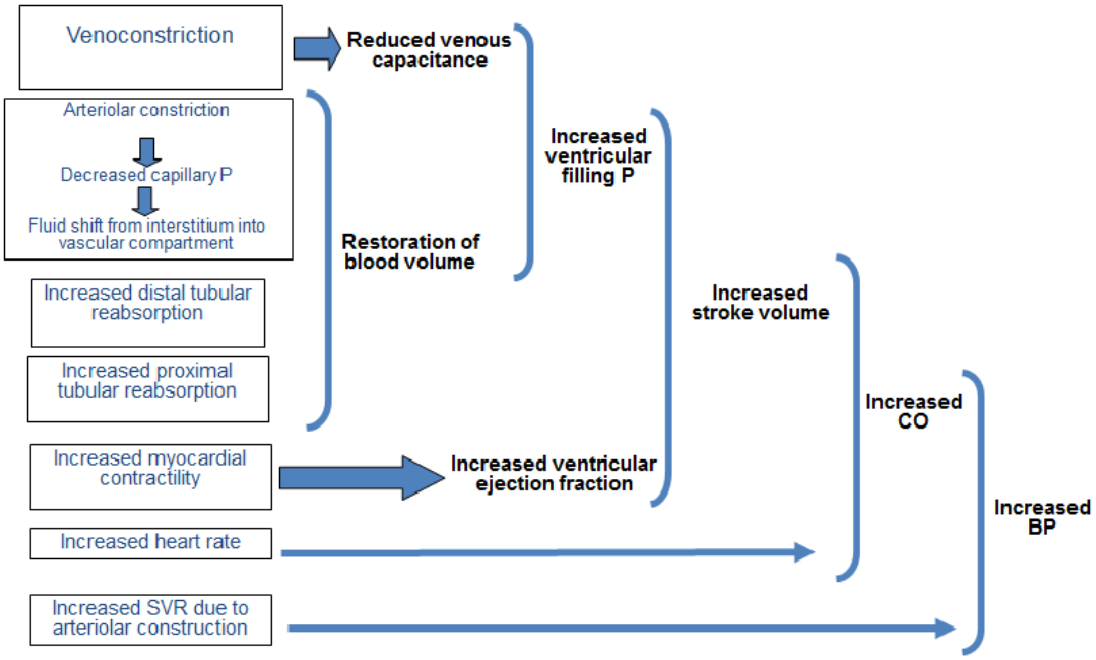
EFFECTS OF SHOCK AT THE ORGAN LEVEL

Multi-organ failure may happen.

- Kidney➔Oliguric renal failure
- Liver➔Liver failure
- GI tract➔ Failure of intestinal barrier (sepsis, bleeding)
- Lung➔Capillary leak associated with or caused by sepsis and infection (ARDS = adult respiratory distress syndrome)

HEMODYNAMIC RESPONSE TO SHOCK

Mechanisms for restoring cardiovascular homeostasis



- Redistribution of blood flow

Attempt to preserve perfusion to vital organs

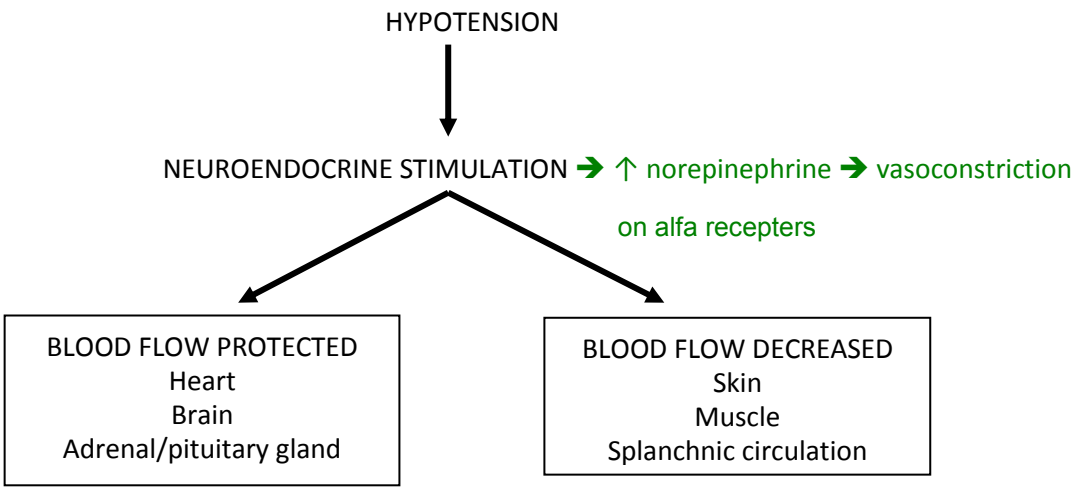
- Augmentation of cardiac output

Increased heart rate

Increased peripheral resistance, by arterioconstriction

- Restoration of intravascular volume, from interstitial space to intravascular

HEMODYNAMIC RESPONSE TO SHOCK (***REDISTRIBUTION OF BLOOD FLOW***)



CARDIOGENIC SHOCK

DECREASED CARDIAC FUNCTION

Decreased ventricular function

Myocardial infarction

Pericaridaltamponade

Tension pneumothorax

Ineffective cardiac contraction

Primary arrhythmias

CLINICAL FINDINGS
Hypotension
Tachycardia
Tachypnea
Oliguria

Caused by the progressive loss of myocardium

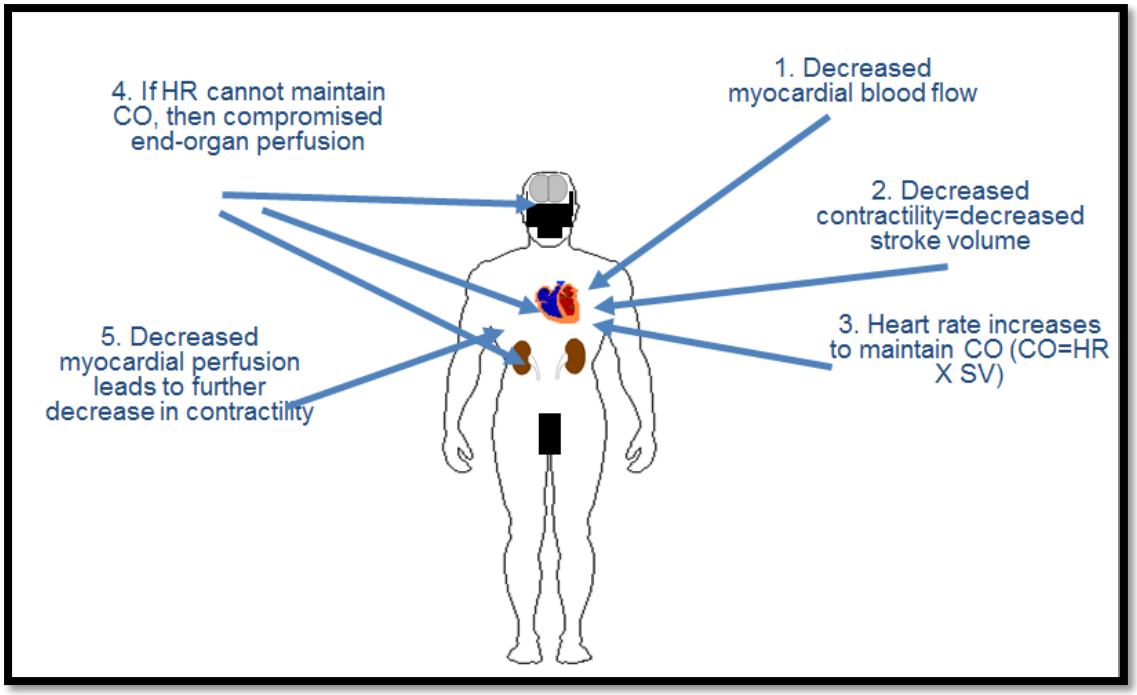
Usually due to an acute myocardial infarction

When the total amount of myocardium affected reaches a critical point, myocardial function begins to deteriorate

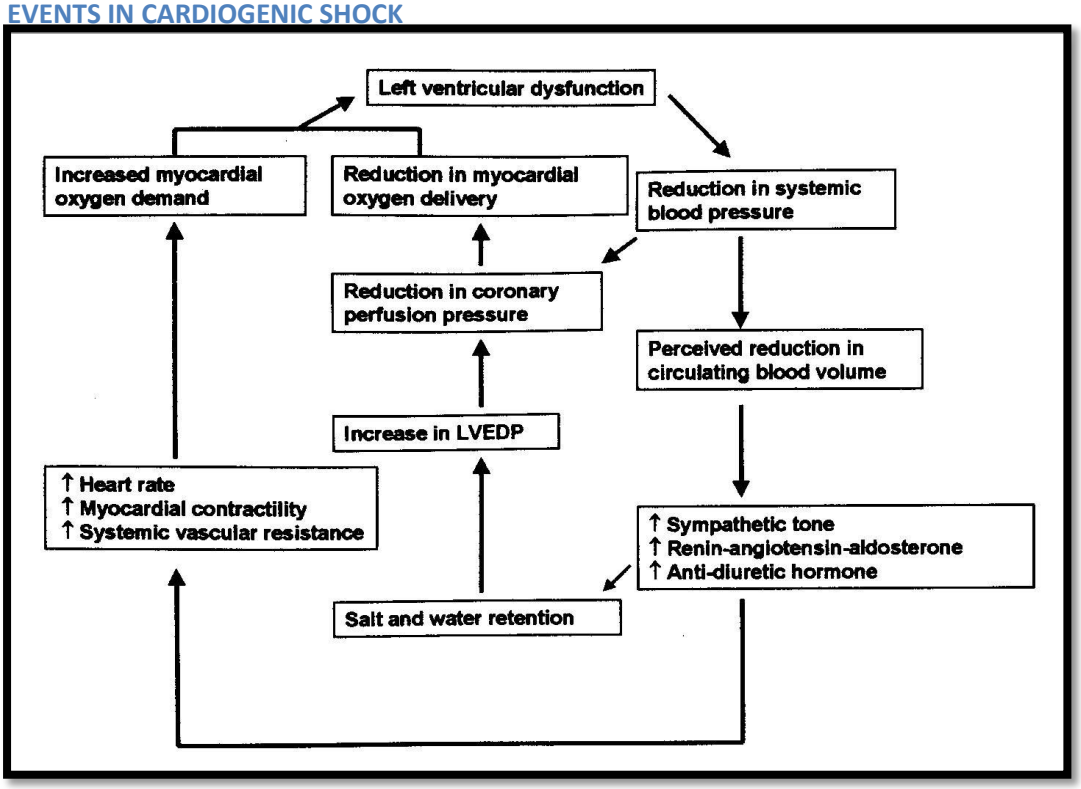
While stroke volume decreases, the heart rate increases in an effort to maintain cardiac output (CO = SV x HR)

But increased HR is limited and CO falls to levels that are inadequate to support end-organ function

Coronary perfusion decreases and this in turn causes progressive myocardial ischemia with progression of myocardial injury

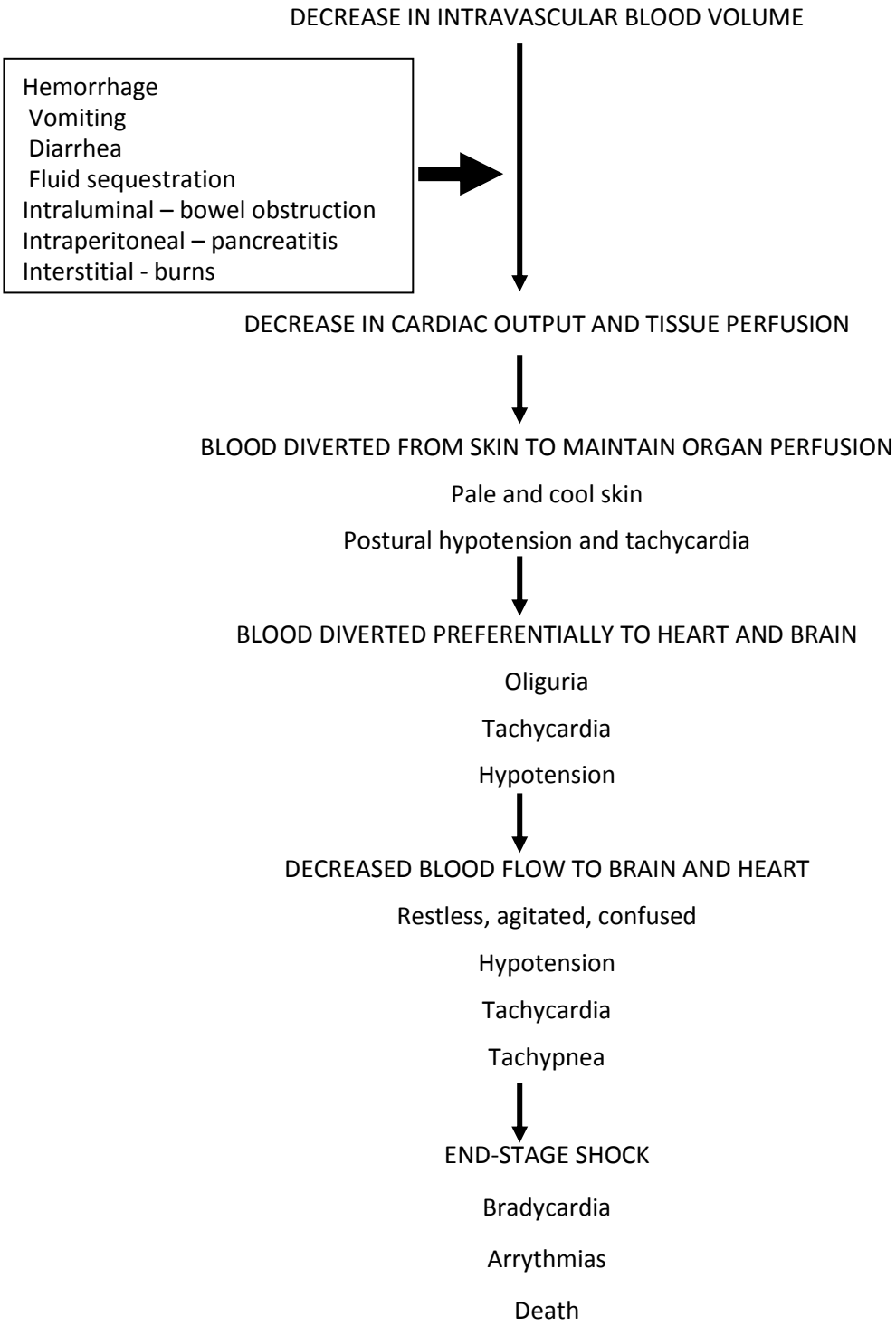


EVENTS IN CARDIOGENIC SHOCK

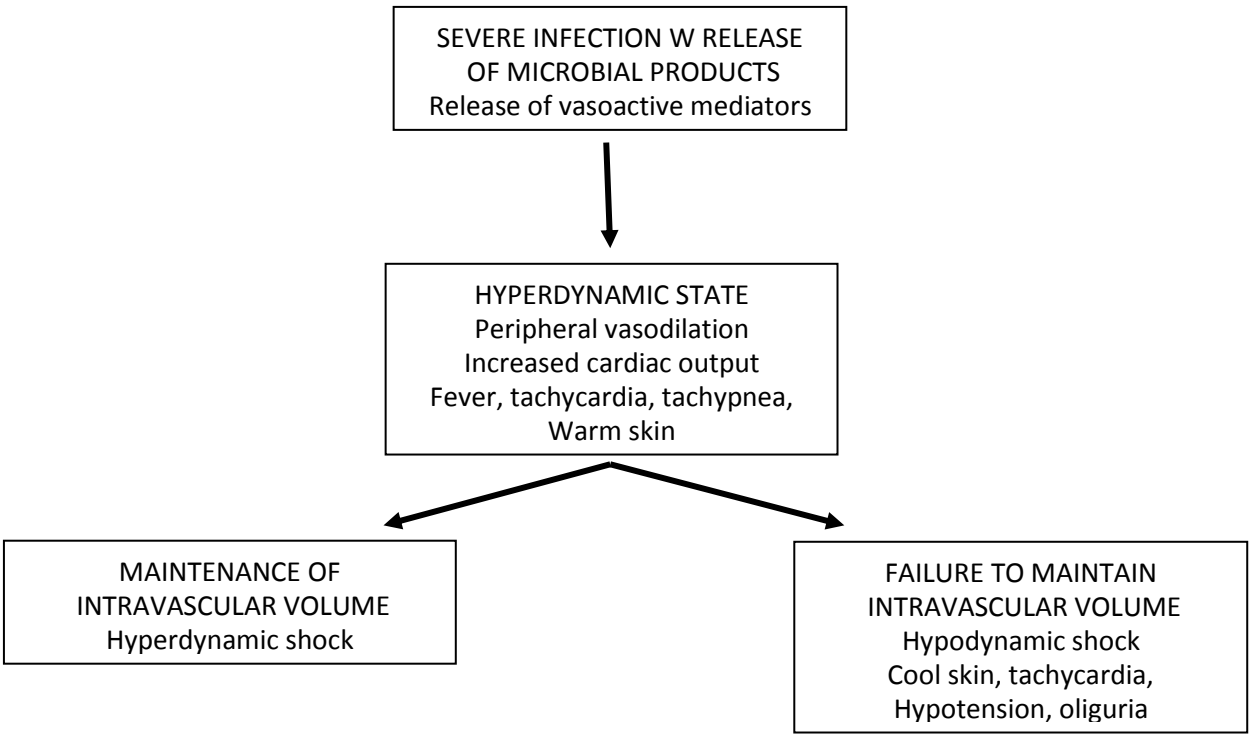


To differentiate cardiogenic shock from others: Jugular venous pressure rises + pulmonary edema

HYPOVOLEMIC SHOCK



SEPTIC SHOCK



Initially vasodilation occurs ➔ SV ➔ HR ➔ hyperdynamic state ➔ warm extremities (warm phase) ➔ no more blood is coming easily ➔ severe vasoconstriction ➔ cold extremities (cold phase)

DIAGNOSING SHOCK STATE BASED ON HEMODYNAMIC PARAMETERS

Type	Central venous pressure	Cardiac output	SVR
Hypovolemic	Decreased	Decreased	Increased
Cardiogenic	Increased	Decreased	Normal or increased
Septic	Decreased or increased	Increased	Decreased
Traumatic	Decreased	Decreased or increased	Decreased or increased
Neurogenic	Decreased	Decreased	Decreased
Hypoadrenal	Decreased or increased	Decreased or increased	Decreased or increased

Systemic Inflammatory Response Syndrome (SIRS)

- The patients demonstrate a similar response as sepsis but without infective agents.
- The criteria are: (two or more to call it SIRS)
  1. Temperature >38 or < 36
  2. Heart rate >90
  3. RR > 20 or a pco2 < 34 mmHg (4.3 kpa)
  4. WBC > 12,000 0r < 4,000 with more than 10% bands

NEUROGENIC SHOCK

- It is a shock that results from a high spinal cord injury (e.g. Cervical spine injury), at the level of C3 or above, where the autonomic supply of the heart is coming from.
- This will result in loss of sympathetic tone
- Loss of sympathetic tone will result in:
  1. Arterial and venous dilatation causing hypotension. Venous capacitance increases.
  2. Bradycardia as a result of unopposed vagal tone.
- The typical feature is hypotension with bradycardia

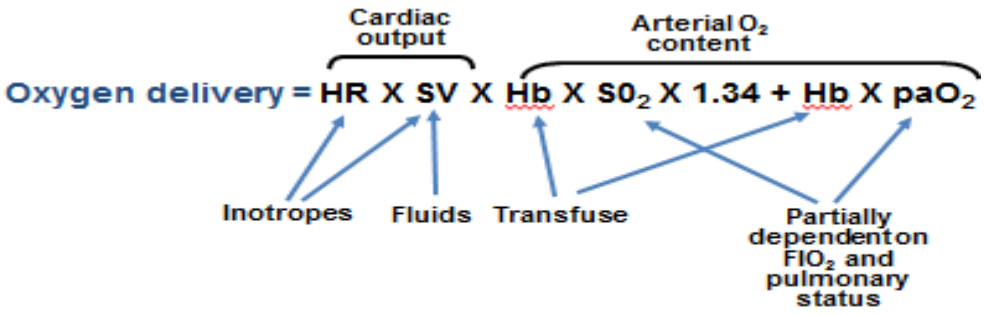
Management of neurogenic shock

- Assessment of airway
- Stabilization of the entire spine, only in trauma
- Volume resuscitation
- R/O other causes of shock
- High dose corticosteroids.

PRINCIPLES OF RESUSCITATION

- Maintain ventilation: ensure oxygen delivery
- Enhance perfusion
- Treat underlying cause

TREATMENT OF SHOCK*: ENHANCING PERFUSION/OXYGEN DELIVERY*



We use ABC principle in managing pt with shock; stands for ensure adequate Airway and Breathing and restore Circulation.

All types of shock are treated with volume replacement, except for cadiogenic shock, treatment depends on etiology.

SUMMARY

- Shock is an altered state of tissue perfusion severe enough to induce derangements in normal cellular function
- Neuroendocrine, hemodynamic and metabolic changes work together to restore perfusion
- Shock has many causes and often may be diagnosed using simple clinical indicators
- Treatment of shock is primarily focused on restoring tissue perfusion and oxygen delivery while eliminating the cause