432 Surgery Team

11 Shock and Metabolic Response to Surgery

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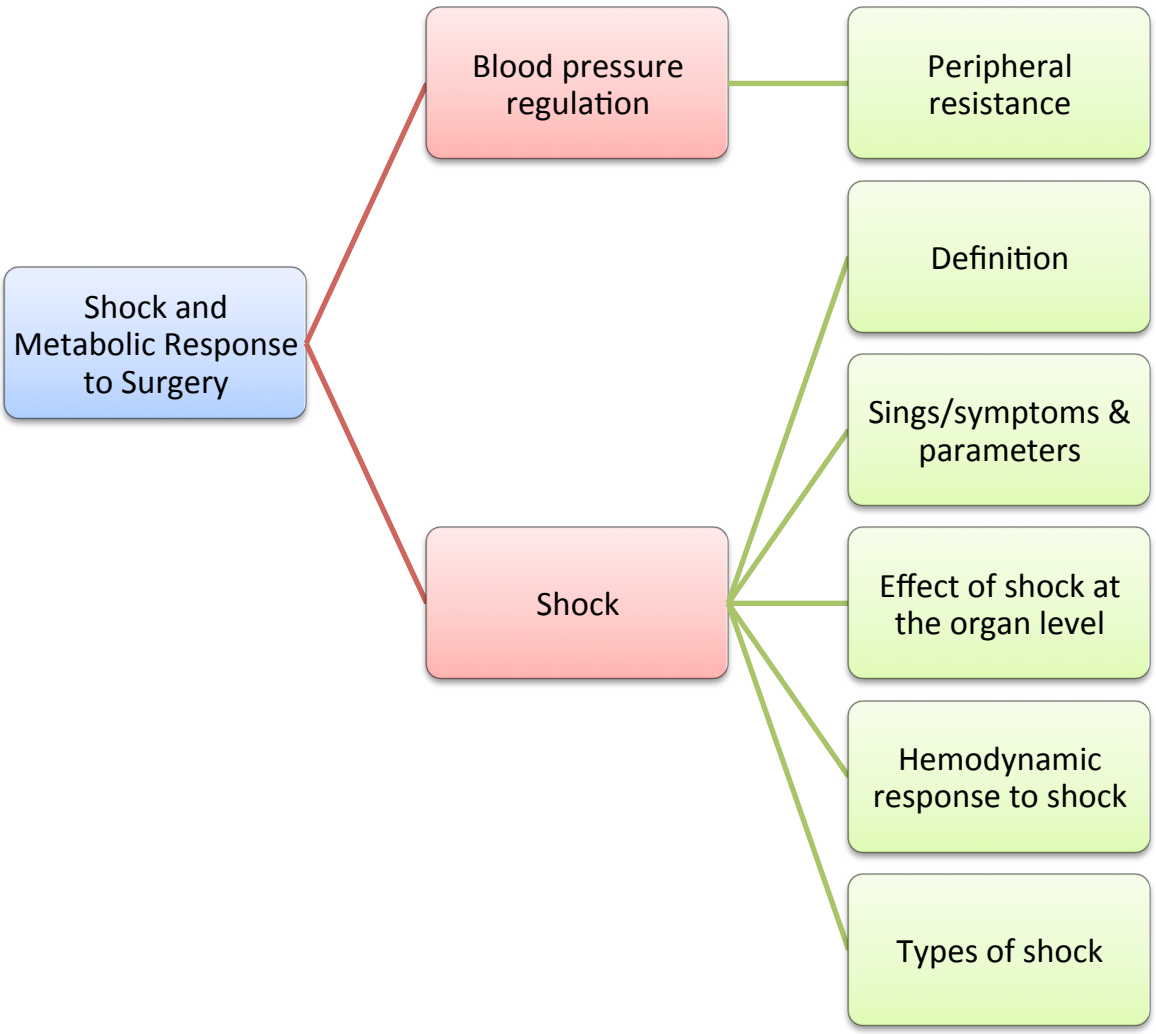
COLOR GUIDE: • Females' Notes • Males' Notes • Important • Additional

Objectives

1. To understand physiology of sustaining blood pressure.
2. To learn about the classifications of shock.
3. To understand the consequences of the natural history of shock.
4. To be able to diagnose and plan appropriate treatments for different types of shock.



Blood Pressure Regulation

Changes in many elements regulate blood pressure (BP) and perfusion:

1. Intravascular volume.
2. Heart.
3. Arteriolar bed.
4. Capillary exchange network.
5. Venules.
6. Venous capacitance circuit.
7. Large vessel patency.

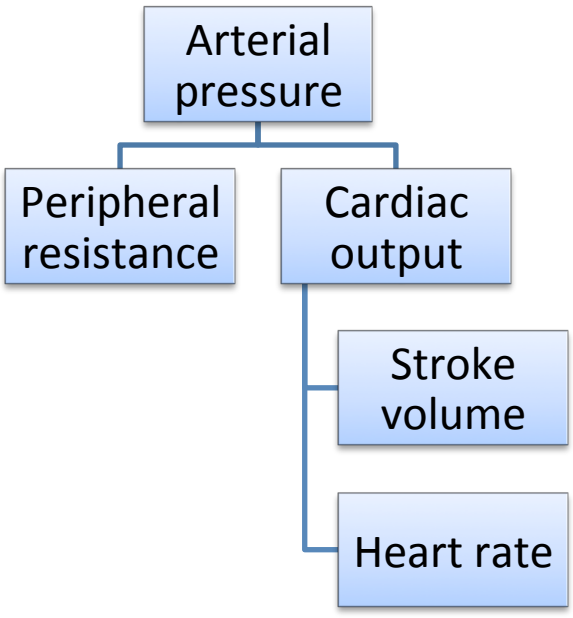
Peripheral resistance (PR):

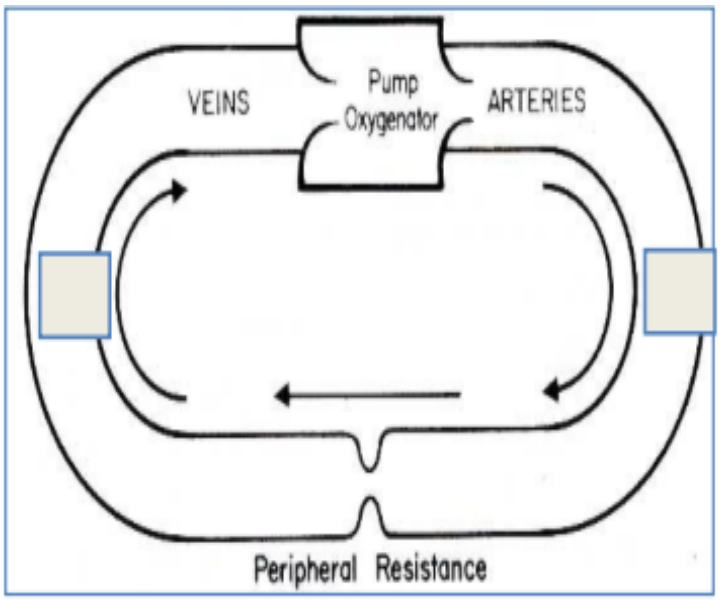
• Decreased peripheral resistance: decreased arterial blood pressure (MAP = CO X PR).

• Increased peripheral resistance:

- Decreased venous return →
- Decreased EDV →
- Decreased SV →
- Decreased CO (CO = HR X SV) →
- Decreased arterial blood pressure (MAP=CO X PR).

Heart Rate X Stroke Volume (↓intravascular volume, ↓EDV) = Cardiac Output.
Cardiac Output X Peripheral Resistance = Arterial Pressure.





Peripheral Resistance:
PR = (Pa – Pv) / CO
** Pa = pressure in the artery.*
** Pv = pressure in the vein.*

Abbreviations:
MAP = mean arterial pressure.
CO = cardiac output.
PR = peripheral resistance.
EDV = end diastolic volume.
HR = heart rate.
SV = stroke volume.
SVR = systemic vascular resistance.

Effects of those elements on BP and perfusion:

1. Intravascular volume:

- Alters mean blood pressure.
  - Decrease in intravascular volume=decreased BP.
- Alters venous return (VR) to the heart.
  - Decrease in intravascular volume =
  - Decreased venous return=
  - Decreased end diastolic volume.
  - CO = HR x SV
  - CO X SVR = MAP

venous return is mainly affected by: Vasodilation & pump faliure

How can intravascular volume be lost?

Examples:

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

2. Heart (cardiac function): it maintains BP in our system & arteries.

Cardiac output is the result of:

- Heart rate (HR).
- Contractility.
- Loading conditions.

Examples of changes that can alter cardiac output:

- Heart rate (bradycardia or tachycardia).
- Contractility (MI or cardiomyopathy) i.e. pump failure.
- Load (histamine release: vasodilation).

CO = HR x SV

HR:

- Bradycardia.

SV:

- MI (pump failure.

- Vasodilation (decreased end diastolic volume).

3. Arteriolar bed (resistance circuit).

- **Decreases** in arteriolar tone produce:
  - Hypotension.
  - Decreased perfusion to vital organs.
- Increases in tone will prevent optimal cardiac performance (increased afterload = decreased contractility).

Further explanation:

The heart delivers blood to all organs by the same mean arterial pressure. Because of that, the width of the arterioles is what determines blood flow to each organ. Arterioles dilate and contract to alter their vascular radius depending on each organs requirement. Arteriolar tone can be modulated by complex substances and mechanisms *but the most are:*

Vasoconstrictors:

1- Sympathetic tone.
2- Vasopressin.
3- Endothelin.

Vasodilators:

1- Decreased organ perfusion
2- Nitric oxide (NO)
3- Any decrease in inherits vasoconstriction regularly provided by the myogenic activity and sympathetic stimulation.

What determines the BP in the arterioles?

- Increase in the permeability and the oncotic pressure.

- Oncotic pressure will increase due to the presence of proteins in the blood vessel which lead to fluid shift from extravascular to intravascular space, leading to volume expansion.

- This physiological process can be disturbed in case of sepsis, trauma or systemic inflammatory response (SIR).

What is the oncotic pressure?

- It's the osmotic pressure, which result from the process of proteins in the blood vessel.

Blood Pressure Regulation (Cont.)

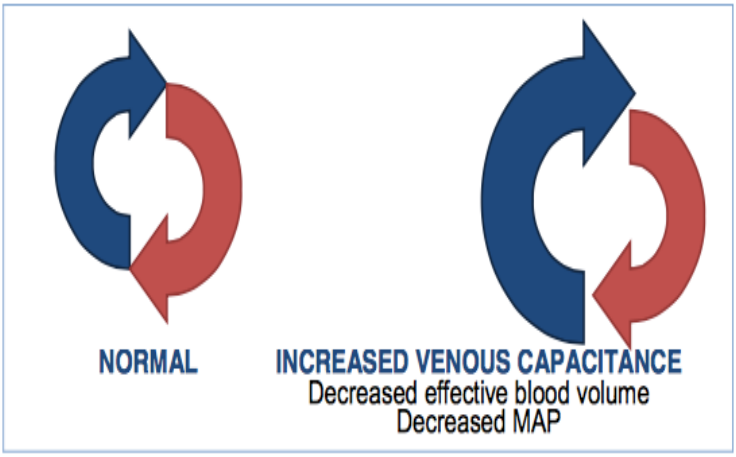
4. Capillary exchange network:

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

Capillary leak syndrome:
1- Vasodilation.
2- A-V shunting.
3- Maldistribution of flow.
4- Increased capillary permeability + interstitial edema.
5- Decreased oxygen extraction.
6- Primary defect of oxygen utilization at cellular level.

5. Venous capacitance circuit:

- Portion of the circulatory system contains 80% of the intravascular volume (the volume is 80% but it's ineffective volume because we want it to return back to the heart).
- Decrease in effective circulating blood volume and MAP caused by:
  - Decreases in venous tone (vasodilation).
  - Increases in venous vascular capacitance.

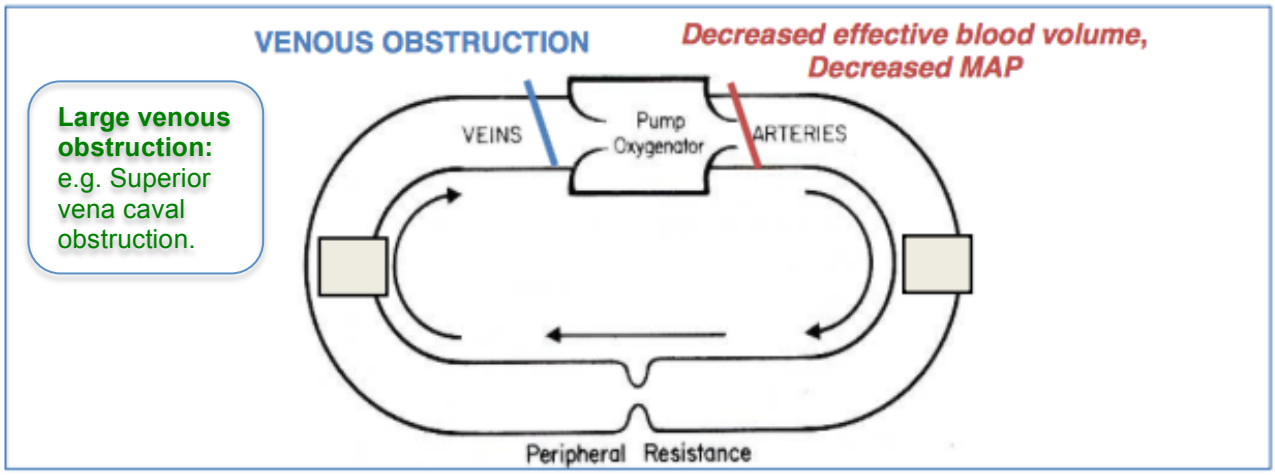


6. Large vessel patency:

- 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.

Large venous obstruction: e.g. Superior vena caval obstruction.



Shock:
It mostly affects veins since veins maintain intravascular volume for longer time.

Shock

Definition: It is the state of altered tissue perfusion severe enough to induce derangements in normal cellular metabolic function (becomes anaerobic metabolism).

- In short: low perfusion that causes tissue hypoxia.

Types of shock: more than one type may be present.

Type of Shock	Clinical Causes	Primary mechanism
1. Hypovolemic	Volume loss	Exogenous blood, plasma, fluid or electrolyte loss
2. Cardiogenic	Pump failure	Myocardial infarction, cardiac arrhythmias, heart failure
3. Distributive *"shock that will result in vasodilatation > vasodilatation or leak > lead to the movement of the blood outside the vessel > decrease the end diastolic volume."*	↑ venous capacitance or arteriovenous shunting It's vasodilation problem leading to decrease in PR & VR.	Septic shock, spinal shock, autonomic blockade, drug overdose ***"Neuorogenic, anaphylactic, septic"***
4. Obstructive	Extra-cardiac obstruction of blood flow	Vena caval obstruction, cardiac tamponade, pulmonary embolism, aortic compression or dissection

Signs/symptoms:

Clinical signs and symptoms of shock relate to decreased organ perfusion:

- **Mental status changes:** decreased cerebral perfusion, e.g. delirium, syncope.
- **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.

Hemodynamic parameters that may indicate shock:

- **Heart rate:** Initial tachycardia (attempt to increase CO).
- **Rhythm:** Regular and tachycardic.
- **Blood pressure:** Low.
- **Cardiac output:** Usually low.

* VC = vasoconstriction.

Effects of shock at the organ level:

- **Kidney**: Oliguric renal failure (to increase tubular reabsorption & decrease secretion).
- **Lung**: Capillary leak associated with or caused by sepsis and infection.
- **GI tract**: Failure of intestinal barrier (sepsis, bleeding); VC of splanchnic circulation; hypoperfusion.
- **Liver**: Liver failure, which is a rare cause.

Hemodynamic response to shock:

Mechanisms for restoring cardiovascular homeostasis:

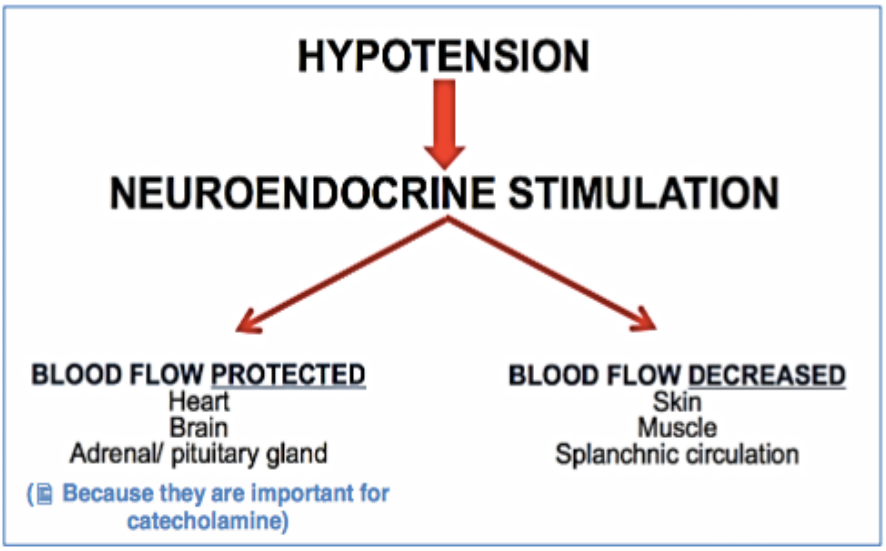
1. **Redistribution of blood flow**: Attempt to preserve perfusion to vital organs.
2. **Augmentation of cardiac output**:
   - Increased heart rate.
   - Increased peripheral resistance.
3. **Restoration of intravascular volume**.

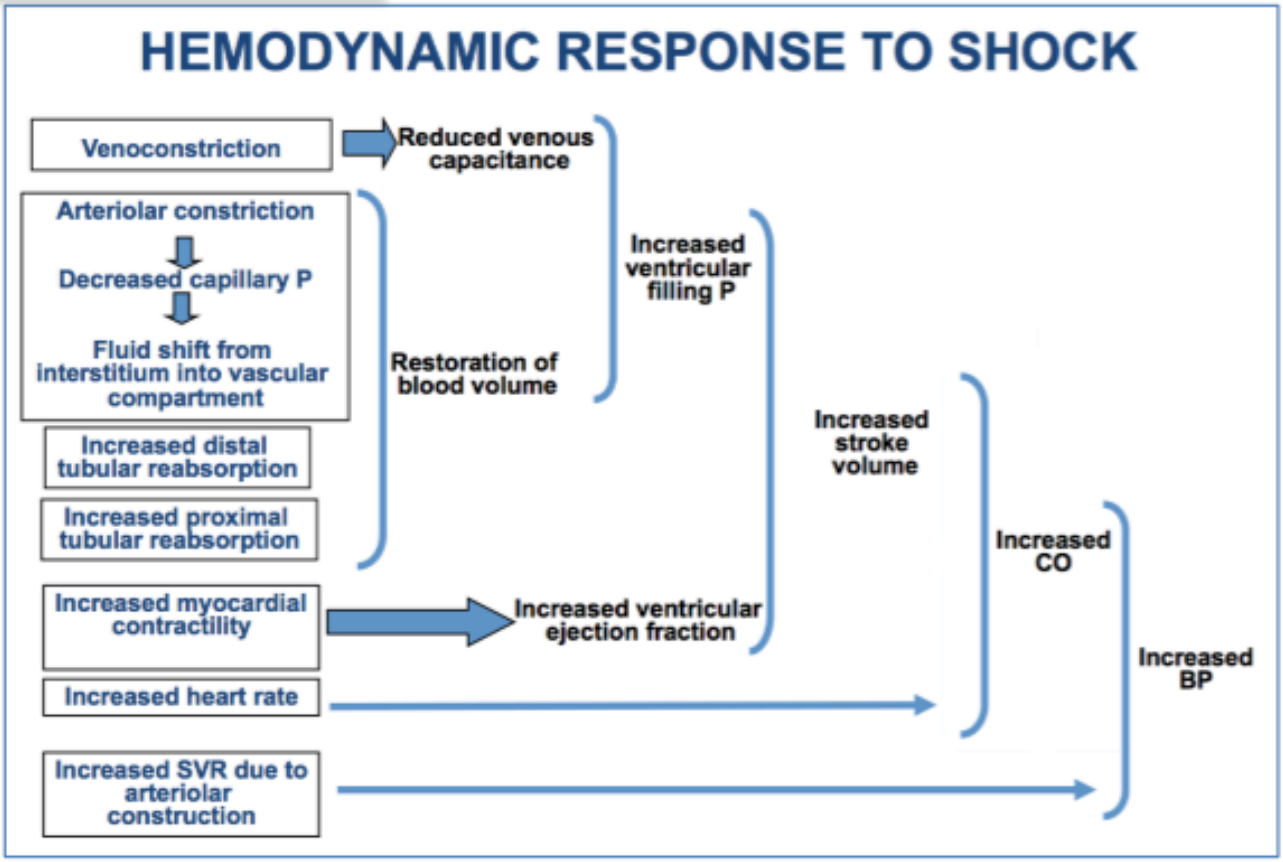
The organ that will contribute in responding to shock is the kidney, how?
- The kidneys are part of the solution not the problem when the body responds to shock.
- It will retain salt that will maintain intravascular volume.

Redistribution of blood flow:

*- **Norepinephrine** is released from the adrenal gland → it acts on alpha-receptors which causes vasoconstriction.*
- It's usually given in distributive shock.

Epinephrine —> 50% a1 and 50% b2
Norepinephrine —> 80% a1 and 20% b2
Thus, NE has a more desirable effect in shocked patients

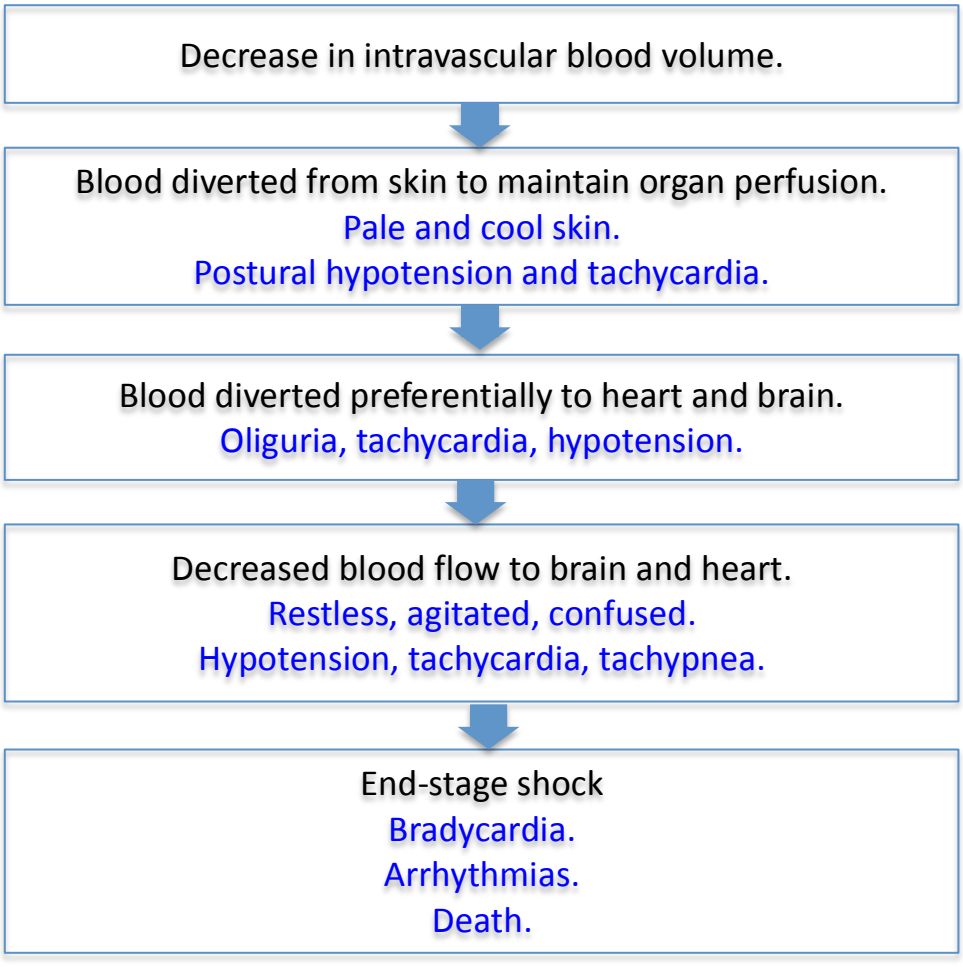




Types of shock:

1- Hypovolemic shock:

- Decrease in intravascular blood volume → decrease in cardiac output and tissue perfusion.
- **Causes:**
    a. Hemorrhage. e.g. trauma, haematemesis, ruptured aortic aneurysm
    b. Vomiting. → Dehydration
    c. Diarrhea. ↗
    d. Fluid sequestration:
        - Intraluminal – bowel obstruction.
        - Intraperitoneal – pancreatitis.
        - Interstitial – burns.
- **To treat it:** replace volume + treat the underlying cause.



NOTES FROM EXTERNAL RESOURCES

	Notes
Principles & Practice of Surgery, 6th edition, Davidson's	This is probably the commonest and most readily corrected cause of shock encountered in surgical practice and results from a reduction in intravascular volume secondary to **loss of blood** (e.g. trauma), **plasma** (e.g. burns) or **water and electrolytes** (e.g. vomiting, diarrhea, diabetic ketoacidosis).

IMPORTANT NOTES FROM EXTERNAL RESOURCES

	Notes
Churchill's Pocketbook of surgery	-Hypovolaemic shock can be divided into four categories, depending on the amount of blood loss : (class I, II, III, IV). **-The symptoms and signs relate to the amount of blood lost:** I. minimal symptoms II. Tachycardia >100, tachypnoea, decreased pulse pressure, pale, sweaty, cold peripheries. III. Classic symptoms of shock – tachycardia >120, hypotension, tachypnoea, pallor, cold peripheries, decreased conscious level, oliguria. IV. Immediate threat to life – tachycardia >140, hypotension (unobtainable diastolic), pallor, cold peripheries, unconscious (>50%), anuria.

Types of shock:

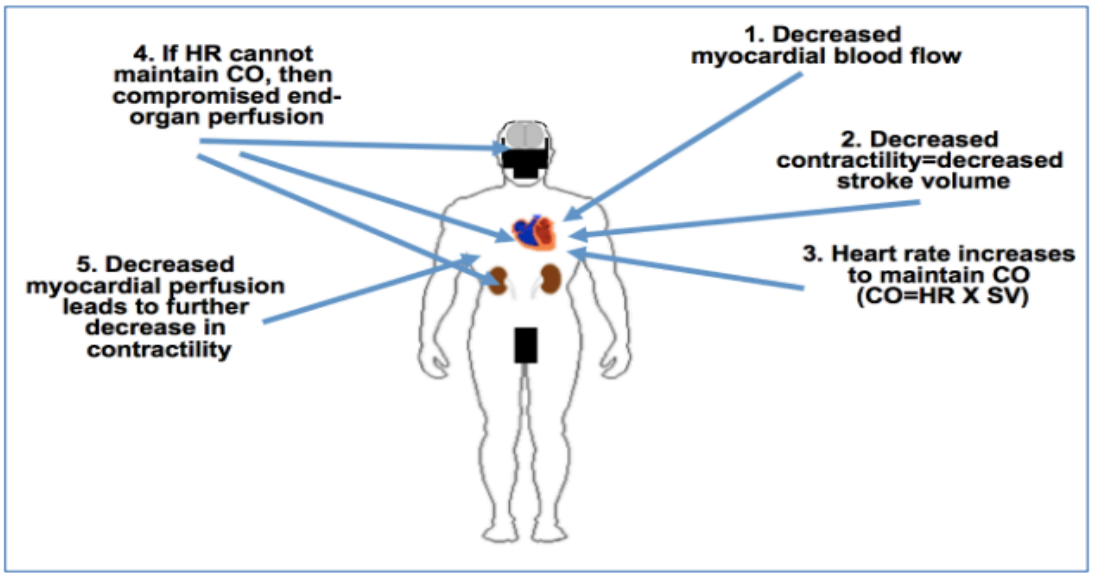
2- Cardiogenic shock:

- 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.

Decreased cardiac function:

- Decreased ventricular function:
  a) Myocardial infarction.
  b) Pericaridal tamponade.
  c) Tension pneumothorax.
- Ineffective cardiac contraction:
  a) Primary arrhythmias.

How do you know if it is cardiogenic shock or not?
1. Shortness of breath (SOB).
2. Raised JVP.
3. Lower limb edema.
4. Basal crepitation.



Types of shock:

2-Cardiogenic shock (Cont.):

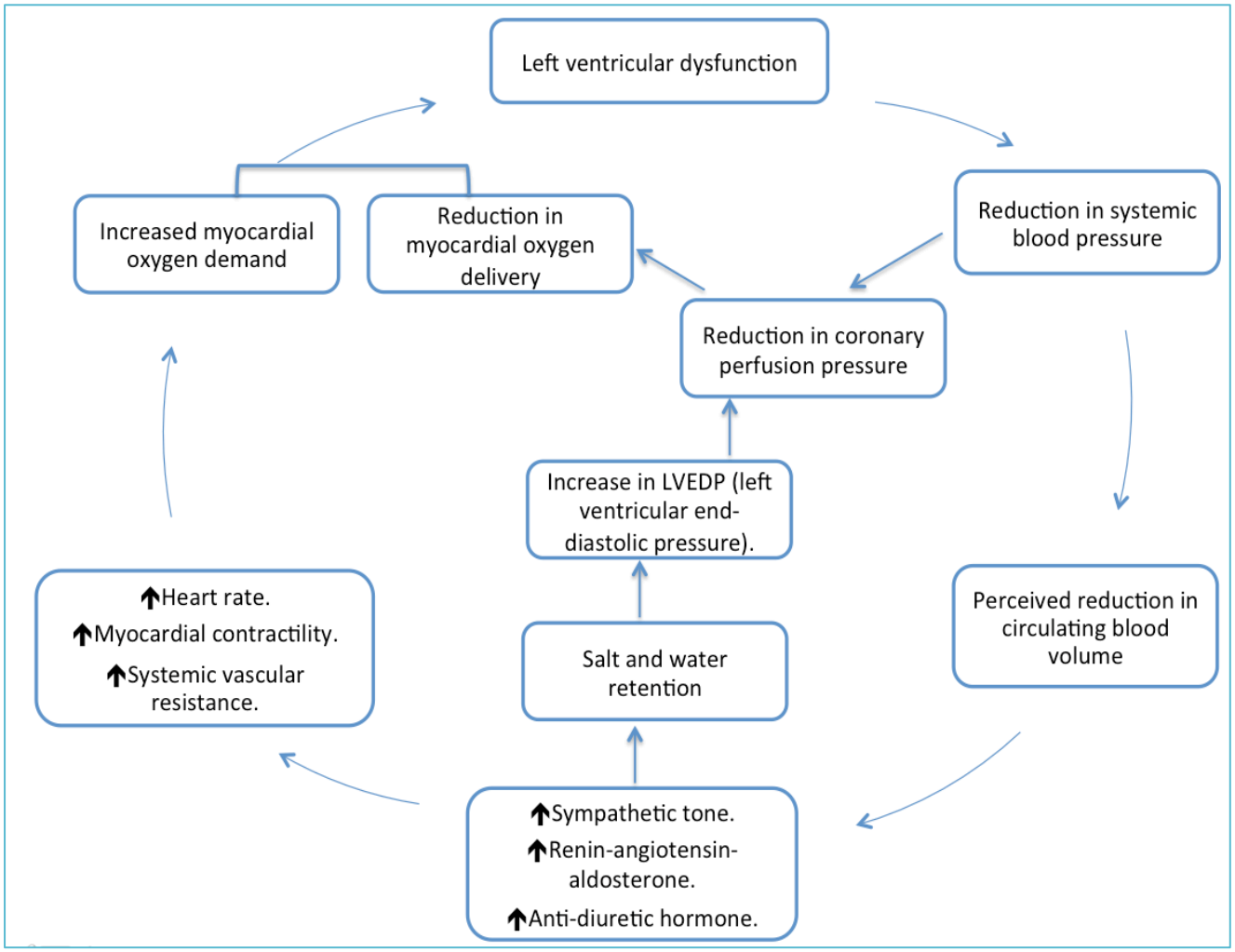
Clinical findings:

1. Hypotension.
2. Tachycardia.
3. Tachypnea.
4. Oliguria.

• **To treat it:**

- Treat the underlying cause.
- In cardiogenic shock the volume is not the problem.
- It's the only shock that you DON'T give fluid because the patient might develop pulmonary edema (because the ventricle is not functioning, all volume will go to the right ventricle then to lung).
- It is very important to differentiate between cardiogenic shock and other types, because cardiogenic shock is the only type in which patient is !!never!! given more volume

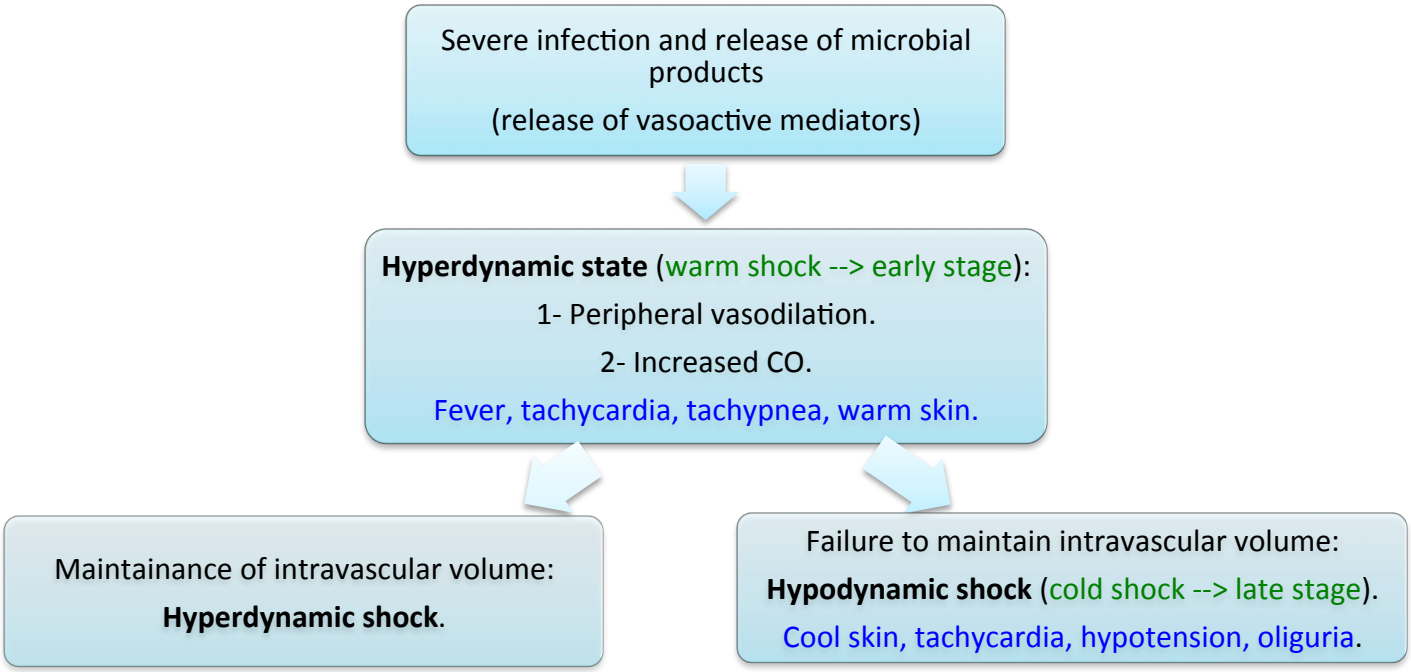
Events in cardiogenic shock:



Types of shock:

3- Distributive shock:

A. Septic shock:



- **To treat it:** replace volume + give antibiotics.

In septic shock, initially extremities are warm (warm shock)

IMPORTANT NOTES FROM EXTERNAL RESOURCES

	Notes
Principles & Practice of Surgery, 6th edition, Davidson's	- Sepsis usually arises from a localized infection, with Gram-ve (38%) and increasingly Gram+ve (52%) bacteria being the most frequently identified pathogens. - Cardiac output typically increases to compensate for the peripheral vasodilation.

IMPORTANT NOTES FROM EXTERNAL RESOURCES

	Notes
Churchill's Pocketbook of surgery	-Septic shock is part of the systemic inflammatory response syndrome (SIRS). Sepsis is defined as SIRS with a confirmed source of infection. Septic shock is defined as hypotension and hypoperfusion despite adequate fluid resuscitation. Septic shock is uncommon in trauma unless there has been a delay in presentation. Septic shock is due to the release of a number of pro-inflammatory mediators and as a result of bacterial endotoxins. It is usually due to Gram -ve organisms such as E.coli, pseudomonas..etc. - **Pathophysiology:** peripheral vasodilation –> high vascular permeability (3rd space loss) –> peripheral arteriovenous shunting –> myocardial depression due to toxic effect on heart –> uncoupling of oxidative phosphorylation and anaerobic respiration leading to severe metabolic acidosis. **-Complication:** sepsis and septic shock can progress to MODS (multi-organ dysfunction syndrome) and MOFS (multi-organ failure syndrome). With continued illness, organ dysfunction progress to organ filure.

Systemic Inflammatory Response Syndrome (SIRS):

Definition:	The patients demonstrate a similar response as sepsis but WITHOUT INFECTIVE AGENTS. It's just an inflammatory process.
The criteria are 2 or more to call it SIRS.	
1) Temperature:	>38 or < 36 (in sepsis it could be hypothermia OR hyperthermia!).
2) Heart rate:	>90
3) RR (respiratory rate):	> 20 or a pCO2 < 34 mmHg (4.3 kpa).
4) WBC:	> 12,000 or < 4,000 with more than 10% bands.

What happens to the lung in systemic inflammatory response (SIR)?
- Adult respiratory distress syndrome (ARDS).
What is ARDS?
- It is a systemic release of inflammatory mediators, causing inflammation, hypoxemia and frequently multiple organ failure (the end result of untreated shock).
- It may accompany many conditions, but most importantly: sepsis, pancreatitis, and severe traumatic injury.

Types of shock:

3- Distributive shock (Cont.):

B. Neurogenic shock:

• It is a shock that result from a high spinal cord injury (e.g. cervical spine traumatic injury). The injury is at level T2 or above. Neurogenic shock is usually due to spinal cord injury above T2, mostly C3

• This will result in loss of sympathetic tone (unopposed parasympathetic tone).

• Loss of sympathetic tone will result in:

1- Arterial and venous dilatation causing **hypotension**.
2- **Bradycardia** as a result of unopposed vagal tone.

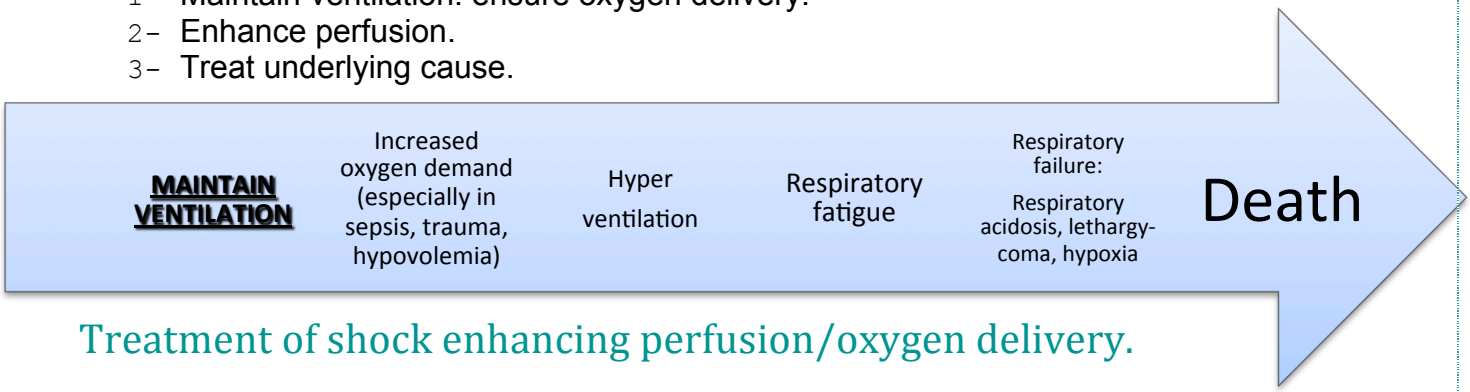
• The typical feature (unique finding) is hypotension with bradycardia (non-neurogenic patient usually have **tachycardia** as a result of shock).

• **Management of neurogenic shock**:

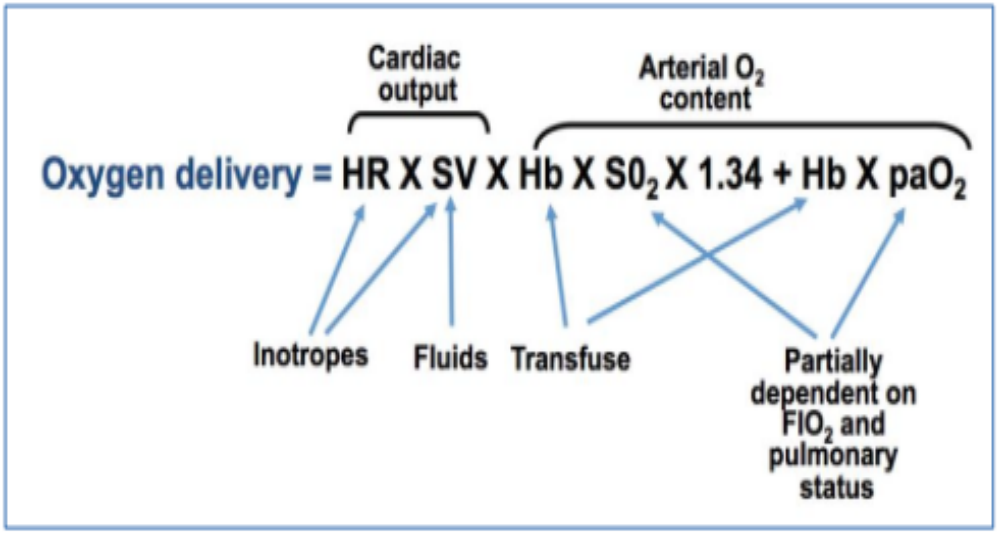
1. Assessment of airway.
2. Stabilization of the entire spine.
3. Volume resuscitation.
4. Rule out (R/O) other causes of shock.
5. High dose corticosteroids.
6. In the non-trauma setting neurogenic shock is self-limiting.

Principles of resuscitation:

1- Maintain ventilation: ensure oxygen delivery.
2- Enhance perfusion.
3- Treat underlying cause.



Treatment of shock enhancing perfusion/oxygen delivery.



SUMMARY

1. Shock is an altered state of tissue perfusion severe enough to induce derangements in normal cellular function.
2. Neuroendocrine, hemodynamic and metabolic changes work together to restore perfusion.
3. Shock has many causes and often may be diagnosed using simple clinical indicators.
4. Generic classification of shock:
   a. **CIRCULATORY shock**:
      - Critical reduction in tissue perfusion results in organ dysfunction and, if untreated, death.
      - Usually accompanied by signs and symptoms:
        - Oliguria.
        - Mental status changes.
        - Weak thread pulse.
        - Cool clammy limbs.
   b. **SEPTIC shock**:
      - Hypotension.
      - Vasodilation with warm limbs.
5. Treatment of shock is primarily focused on restoring tissue perfusion and oxygen delivery while eliminating the cause.

Diagnosing shock state based on hemodynamic parameters

Type	Central venous pressure	Cardiac output	Systemic vascular resistance (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

IMPORTANT NOTES FROM EXTERNAL RESOURCES

	Notes
Principles & Practice of Surgery, 6th edition, Davidson's + Churchill's Pocketbook of surgery	- Anaphylactic shock: this is a severe systemic hypersensitivity reaction (Type I) following exposure to an agent triggering the release of vasoactive mediators (histamine, kinins). - Anaphylaxis may be immunologically mediated (the reaction mediated by IgE) or non-immunologically mediated. - Clinical features: vasodilation, intravascular volume redistribution, capillary leak and a reduction in CO. - Symptoms & signs: Generalized urticarial, wheezing, laryngeal odema, hypotension, loss of consciousness. - Common causes: drugs (e.g. beta-lactam antibiotics), colloid solutions (e.g. gelatin containing solutions, dextrans), radiological contrast media, food (e.g. peanuts, shellfish), hymenptera stings and latex.

Cases:

Case 1: Circulatory Shock

10 y/o female fell off bike riding down a hill. Initially well but 4 hrs later complained of abdominal pain and left shoulder pain.

- On examination:
  - Vital signs (VS): BP 90/60, P 120 (tachycardic), RR 30 (tachypneic), T 100.1, O2 sat 95% (low)
  - General (GEN): pale, anxious
  - Lung: clear to auscultation
  - Precordium (COR): tachycardic with murmur best heard at base
  - Abdomen (ABD): diffuse tenderness without peritonitis or mass
- Labs: Hb 7.5 (low)
- Hemodynamics:

Central venous pressure	Decreased
Cardiac output	Decreased
Systemic vascular resistance	Decreased

- Abdominal CT: splenic laceration with free peritoneal fluid
- Patient is in **respiratory failure**:

- **Treatment** of respiratory failure:
  1) Primary resuscitation
  2) Oxygen
  3) Mechanical ventilation if necessary

Case 2: Septic Shock

15 y/o male with a 4 day history of abdominal pain, N/V and anorexia

- On examination:
  - VS: BP 70/60 (low), P 130 (high), RR 28 (high), T102.4, O2 sat 99%
  - GEN: moderate distress from abdominal pain
  - COR: tachycardic
  - ABD: diffuse tenderness w peritonitis
- Labs:
  - WBC 19,600 (high), 90% segments
  - Hb 14.2
- Hemodynamics:

Cardiac output	Increased
Systemic vascular resistance	Decreased

- **Dx**: perforated appendicitis

Case 3: Neurogenic Shock

17 y/o male, diving into water

- On examination:
  - VS: BP 90/60 (low), P 110 (high), RR 24 (high)
  - Paralysis below C5
- Hemodynamics:

Central venous pressure	Decreased
Cardiac output	Decreased
Systemic vascular resistance	Decreased

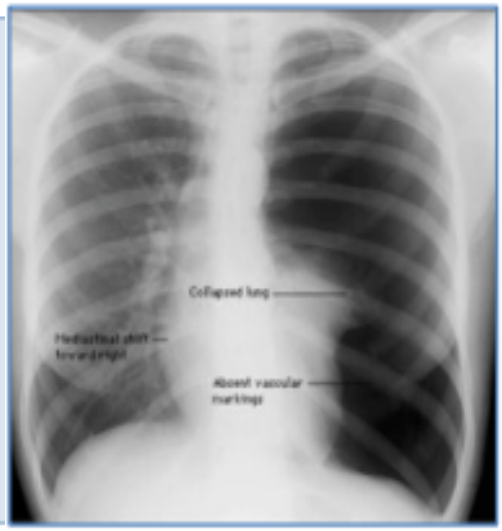
- Cervical X-ray: C5 fracture

Case 4: Cardiogenic Shock

17 y/o male, training for track team

- On examination:
  - VS: BP 70/50 (low), P 140 (high), RR 35 (high), O_2 sat 88%
  - Absent breath sounds in left lung field, distended neck veins
- Dx: tension pneumothorax
- Hemodynamics:

Central venous pressure	Increased
Cardiac output	Decreased
Systemic vascular resistance	Normal

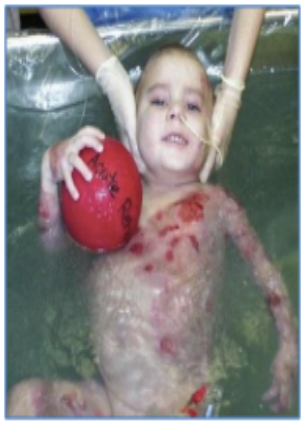


Case 5: Capillary Leak Syndrome

3 y/o male, clothes ignited from roaster

- On examination:
  - VS: BP 60/60 (low), P 170 (high), RR 35 (high), T102.4, O2 sat 89%
  - GEN: moderate distress
  - LUNG: tachypneic, clear to auscultation
  - COR: tachycardic, regular
  - SKIN: 60% TBSA partial and full thickness burn
- Hemodynamics:

Cardiac output	Decreased
Systemic vascular resistance	Increased



- **Dx**: 60% of total body surface area (TBSA) burn; hypovolemic shock (loss of fluid into interstitium, called "third spacing")
- **Rx**: MAINTAIN VENTILATION

Questions from surgical recall book

What is the definition of shock?	Inadequate tissue perfusion
What are the different types (5)?	Hypovolemic Septic Cardiogenic Neurogenic Anaphylactic
What are the signs of shock?	Pale, diaphoretic, cool skin Hypotension, tachycardia, tachypnea ↓ mental status and pulse pressure Poor capillary refill Poor urine output
What are the best indicators of tissue perfusion?	Urine output, mental status

HYPOVOLEMIC SHOCK

What is the definition?	Decreased intravascular volume
What are the common causes?	Hemorrhage Burns Bowel obstruction Crush injury Pancreatitis
What are the signs?	**Early**—Orthostatic hypotension, mild tachycardia, anxiety, diaphoresis, vasoconstriction (decreased pulse pressure with increased diastolic pressure) **Late**—Changed mental status, decreased BP, marked tachycardia
What are the signs/ symptoms with:	
Class I hemorrhage (<15% or 750 cc blood loss)?	Mild anxiety, normal vital signs
Class II hemorrhage (15%–30% or 750–1500 cc blood loss)?	Normal systolic BP with decreased pulse pressure, tachycardia, tachypnea, anxiety
Class III hemorrhage (30%–40% or 1500–2000 cc blood loss)?	Tachycardia (heart rate >120), tachypnea (respiratory rate >30), **decreased systolic BP**, decreased pulse pressure, confusion

Class IV hemorrhage (>40% or >2000 cc blood loss)?	Decreased systolic BP, tachycardia (heart rate >140), tachypnea (respiratory rate >35), decreased pulse pressure, confused and lethargic, no urine output
What is the treatment?	1. **Stop the bleeding** 2. **Volume:** IVF (isotonic LR) then blood products as needed

SEPTIC SHOCK

What is the definition?	Documented infection and hypotension
What is the specific etiology?	Most common—gram-negative septicemia Less common—gram-positive septicemia, fungus
What factors increase the susceptibility to septic shock?	Any mechanism that increases susceptibility to infection (e.g., trauma, immunosuppression, corticosteroids, hematologic disease, diabetes)
What complications are major risks in septic shock?	Multiple organ failure, DIC, **death**
What are the signs/ symptoms?	Initial—vasodilation, resulting in warm skin and full pulses; normal urine output Delayed—vasoconstriction and poor urine output; mental status changes; hypotension
What are the associated findings?	Fever, hyperventilation, tachycardia

CARDIOGENIC SHOCK

What is the definition?	Cardiac insufficiency; left ventricular failure (usually), resulting in inadequate tissue perfusion
What are the causes?	MI, papillary muscle dysfunction, massive cardiac contusion, cardiac tamponade, tension pneumothorax, cardiac valve failure
What are the signs/symptoms on exam?	Dyspnea Rales Pulsus alternans (increased pulse with greater filling following a weak pulse) Loud pulmonic component of S_2 Gallop rhythm
What are the associated vital signs/parameters?	Hypotension, decreased cardiac output, elevated CVP/wedge pressure, decreased urine output (low renal blood flow), tachycardia (possibly)
What are the signs on CXR?	Pulmonary edema
What is the treatment?	Based on diagnosis/mechanism: 1. CHF: diuretics and afterload reduction (e.g., ACE inhibitors), with or without pressors 2. Left ventricular failure (MI): pressors, afterload reduction

NEUROGENIC SHOCK

What is the definition?	Inadequate tissue perfusion from loss of sympathetic vasoconstrictive tone
What are the common causes?	Spinal cord injury: Complete transection of spinal cord Partial cord injury with spinal shock Spinal anesthesia

What are the signs/ symptoms?	**Hypotension** and **bradycardia**, neurologic deficit
Why are heart rate and BP decreased?	Loss of sympathetic tone (but hypovolemia [e.g., hemoperitoneum] must be ruled out)
What are the associated findings?	Neurologic deficits suggesting cord injury
What MUST be ruled out in any patient where spinal shock is suspected?	Hemorrhagic shock!
What is the treatment?	**IV fluids** (vasopressors reserved for hypotension refractory to fluid resuscitation)
What percentage of patients with hypotension and spinal neurologic deficits have hypotension of purely neurogenic origin?	About 67% (two thirds) of patients
What is spinal shock?	Complete flaccid paralysis immediately following spinal cord injury; may or may not be associated with circulatory shock

What are the classic findings associated with spinal cord shock?	Hypotension Bradycardia or lack of compensatory tachycardia

MCQ,S

1) Which one of these parameters will appear first and can be diagnosed for shock?

a. Hypotension.
b. Bradycardia.
c. Decreased tissue perfusion.
d. Tachycardia.

2) The most sensitive tissue to ischemia is:

a. Muscle.
b. Nerve.
c. Skin.
d. Adipose tissue.
e. Bone.

3) Which one of the following doesn't cause hypovolemic shock?

a. Hemorrhage
b. Trauma.
c. Surgery.
d. Myocardial infarction (MI).
e. Burns.

4) A 25 y/o driver sustained a car accident presented to the ER with flaccid paralysis, bradycardia, and hypotension. The most likely Dx:

a. Neurogenic shock.
b. Cardiogenic shock.
c. Hypovolemic shock.
d. None of the above.

5) The commonest cause of the previous case is:

a. Massive external bleeding.
b. Ischemic heart disease.
c. Injury to the high thoracic spine.
d. Internal bleeding.

Answers:

1st Questions: d

2nd Questions: b

3rd Questions: d

4th Questions: a

5th Questions: c