Shock and metabolic response

Objectives:

- To understand physiology of sustaining blood pressure.
- To learn about the classifications of shock.
- To understand the consequences of the natural history of shock.
- To be able to diagnose and plan appropriate treatments for different types of shock. "Principles"

Resources:

- Davidson's.
- Slides
- Surgical recall.
- Raslan's notes.
- Linda

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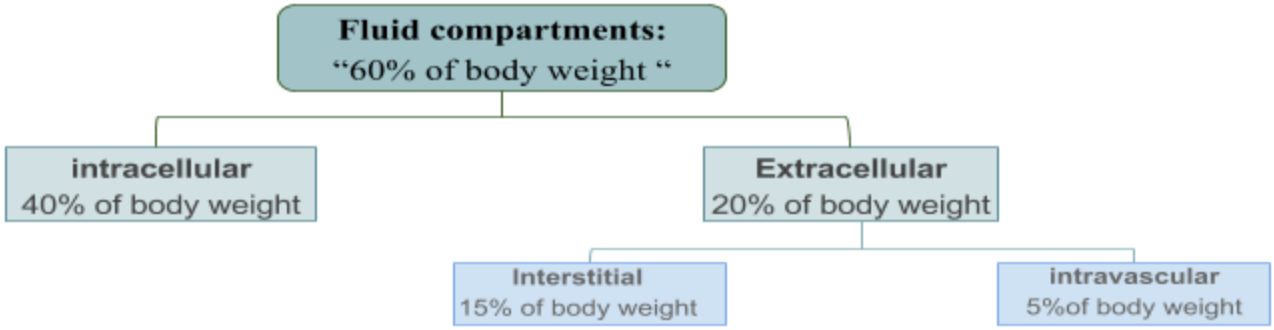
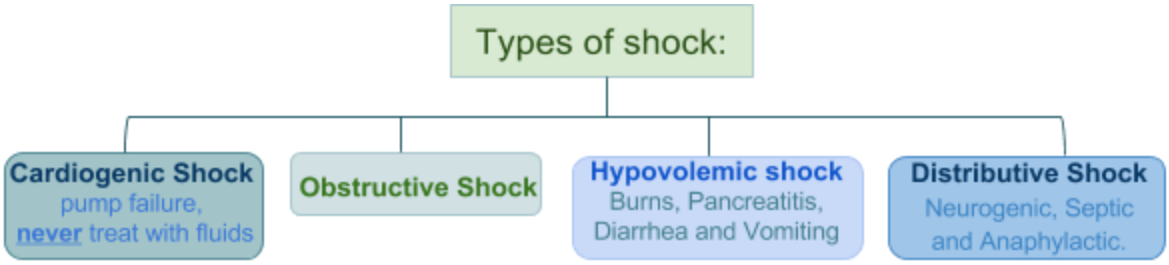
Once you stop learning you start dying.

In This link you will find a basic concepts and a detailed pathophysiology of shock:

Basic review:



- Average Cardiac Output = 5 LITERS:
  - CO (ml/min) = HR (beat/min) X SV (ml/beat)
    - (stroke volume depends on the end-diastolic volume and contractility).
- Shock: low perfusion that causes tissue hypoxia → cellular derangement.



Shock - causes, symptoms, diagnosis, treatment, pathology 10:13 minutes *highly recommended*

What is the normal reference BP?

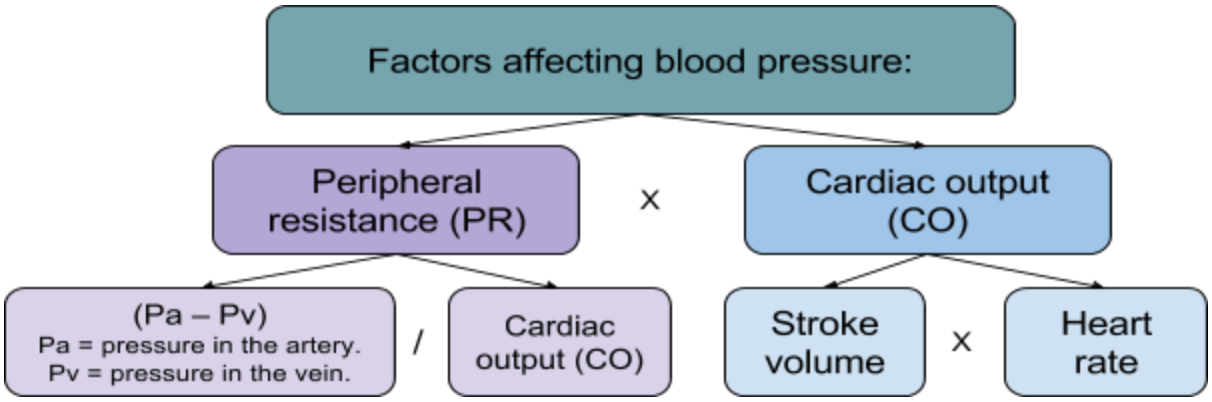
98% of people normal BP is 120/80, but 2% can have a systolic BP of 75-80 but they're asymptomatic and functioning perfectly well. These people do not require treatment, as **you don't treat numbers, you treat people.**

- An elder guy on 3 antihypertensive medications as he has a baseline DBP of 200, came to the ER with a DBP of 130. Is he hypotensive? Yes, his BP usually requires drugs to keep it normal but this time he is spontaneously almost within the normal range.
- بيجيكم في الامتحان - A runner/ athlete will normally have asymptomatic Bradycardia (e.g. 60 b/m), if she comes with an illness and a pulse rate of 80, this is not her norm and she is tachycardic.
- Tachycardia is the earliest response of illness (e.g. sepsis). However, this normal response might be blocked by beta-blockers or Cardiac block. " think about it before you diagnose"

CHANGES IN THESE ELEMENTS REGULATE BP AND PERFUSION:

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

Others: Kidneys, baroreceptors, hormones, peripheral resistance (vasoconstriction/ vasodilation)

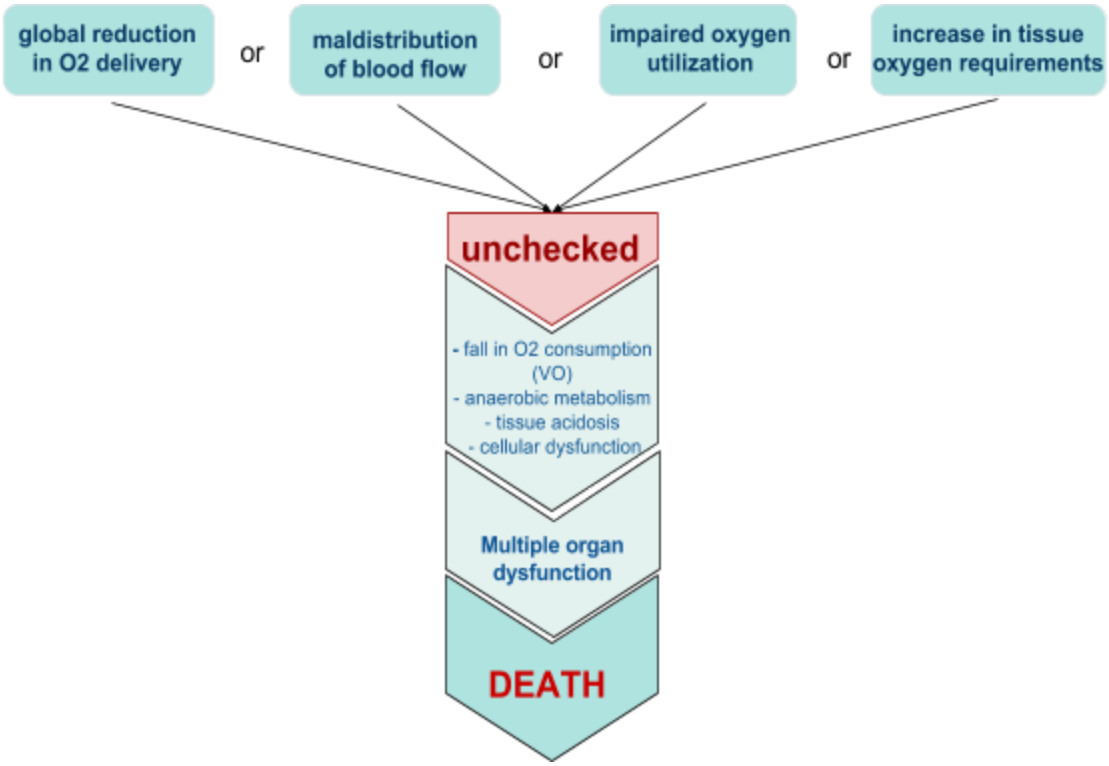


*__Don't worry about the formula__, but remember that any problem in the stroke volume your heart rate will increase to keep the CO constant, if CO increases vascular resistance will decrease to keep BP normal and compensated.

SHOCK

Shock is the state of altered tissue perfusion severe enough to induce derangements in normal cellular metabolic function. In short: shock is low perfusion that causes tissue hypoxia.
"Shock" is not synonymous with hypotension. "Hypoxia happens After loss of compensatory mechanisms."
It mostly affects veins since they maintain intravascular volume for longer time.

Pathophysiology:



Most shock (exception neurogenic) is associated with **increased sympathetic activity** and all share common pathophysiological features at the cellular level.

TYPES OF SHOCK:

- **MORE THAN ONE TYPE MAY BE PRESENT AT THE SAME TIME.**

Types of shock	Clinical cause	Primary mechanism
Hypovolemic	Volume loss	Exogenous blood, plasma, fluid or electrolyte loss
Cardiogenic	Pump failure	Myocardial infarction, cardiac arrhythmias, heart failure.
Distributive Shock that will result in vasodilation or leak > lead to the movement of the blood outside the vessel > decrease the end diastolic volume	Increased venous capacitance or arteriovenous shunting	Septic shock, spinal shock, autonomic blockade, drug overdose **"Neurogenic, anaphylactic, septic"**
Obstructive	Extra-cardiac obstruction of blood flow	Vena caval obstruction, cardiac tamponade, pulmonary embolism, aortic compression or dissection

Classification might be different in different books. E.g. distributive shock is a new term for septic, anaphylactic, neurogenic shock. But eventually it is the same disease.

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

- **Mental status changes:** due to decreased cerebral perfusion (last sign to occur) e.g. delirium, syncope, confusion, dizziness
- **Decreased urine output:** due to decreased renal perfusion. (most important and earliest predictor of shock).
- **Cold clammy cyanotic extremities:** due to decreased perfusion to the skin due to diverted blood flow.
- **EKG changes:**
  - May indicate myocardial ischemia.
  - May be primary event (cardiogenic shock) or due to decreased myocardial perfusion due to shock from other causes.

Effects of shock at the organ level:

- **Nervous system:**
  - Restlessness, confusion, stupor[1], coma
  - Encephalopathy and/or delirium common in sepsis

- **Renal:**
  - Renal hypoperfusion → activation of rennin–angiotensin system (to increase tubular reabsorption & decrease secretion).
  - **Oliguria** (< 0.5 ml/kg/h urine)[2] → anuria
  - **Acute renal failure** → ↑ urea, ↑ creatinine, ↑ K+ & metabolic acidosis

- **Respiratory:**
  - Tachypnoea
  - ↑ Ventilation/perfusion (V/Q) mismatch & ↑ shunt → hypoxia.

[1] it a fancy term describing " lethargy" used by Davidson.
[2] 400 mL daily in adults.

- **Pulmonary oedema** " Capillary leak associated with or caused by sepsis and infection", (common in cardiogenic shock)→ hypoxia.
- Acute lung injury and acute respiratory distress syndrome → hypoxia.

- **Cardiovascular:**
  - ↓ Diastolic pressure → ↓ coronary blood flow
  - ↓ Myocardial oxygen delivery → myocardial ischaemia → ↓ contractility & ↓ CO
  - Acidosis, electrolyte disturbances and hypoxia → predispose to arrhythmias
  - Widespread endothelial cell activation → microcirculatory dysfunction .

- **Gastrointestinal:**
  - **Splanchnic hypoperfusion** → breakdown "Failure" of gut mucosal barrier (sepsis, bleeding)
  - Stress ulceration
  - Translocation of bacteria/bacterial wall contents into blood stream → SIRS
  - Acute ischaemic hepatitis.
  - **Liver**: Liver failure, which is rare.

Someone is bleeding, will he have diarrhea? No. his body will absorb the fluid trying to increase intravascular volume.

Hemodynamic parameters that may indicate shock:

- **Heart rate:** Initial tachycardia (attempt to increase CO)
- **Rhythm:** Regular and tachycardic.
- **Blood pressure:** Low "it is often a late manifestation of circulatory failure or shock"
- **Cardiac output:** Usually low

◆ Remember:	**MAP = CO × SVR**

Hemodynamic response to shock:

Mechanisms for restoring cardiovascular homeostasis (BP):

1. Redistribution of blood flow: Attempt to preserve perfusion to vital organs "End organs"

- ❑ HYPOTENSION → NEUROENDOCRINE STIMULATION
- ➔ PROTECTION OF BLOOD FLOW TO: Heart, Brain & Adrenal/pituitary gland (because they are important for catecholamines)
- ➔ DECREASE IN BLOOD FLOW TO: Skin, Muscle & Splanchnic circulation

Moving extravascular fluid to maintain intravascular volume SV and CO (you don't need to know the neurohormonal mechanism).

E.g. 70 year old guy, hyperlipidemic hypertensive diabetic (IHD) got diabetic foot ulcer. Insult? Infection/Sepsis. Came to the ER with hypotension (60 systolic), tachycardia, oliguria, cold = shock. toxins prevent tight junctions and disturbed capillary bed, peripheral resistance couldn't compensate, blood moves in the extravascular space "edema".

His son (a healthy guy) came with ulcer, redness and hotness (signs of infection) but he didn't show up with a SBP of 60. Why? Compensation. His heart is able to pump more, increase HR and SV.

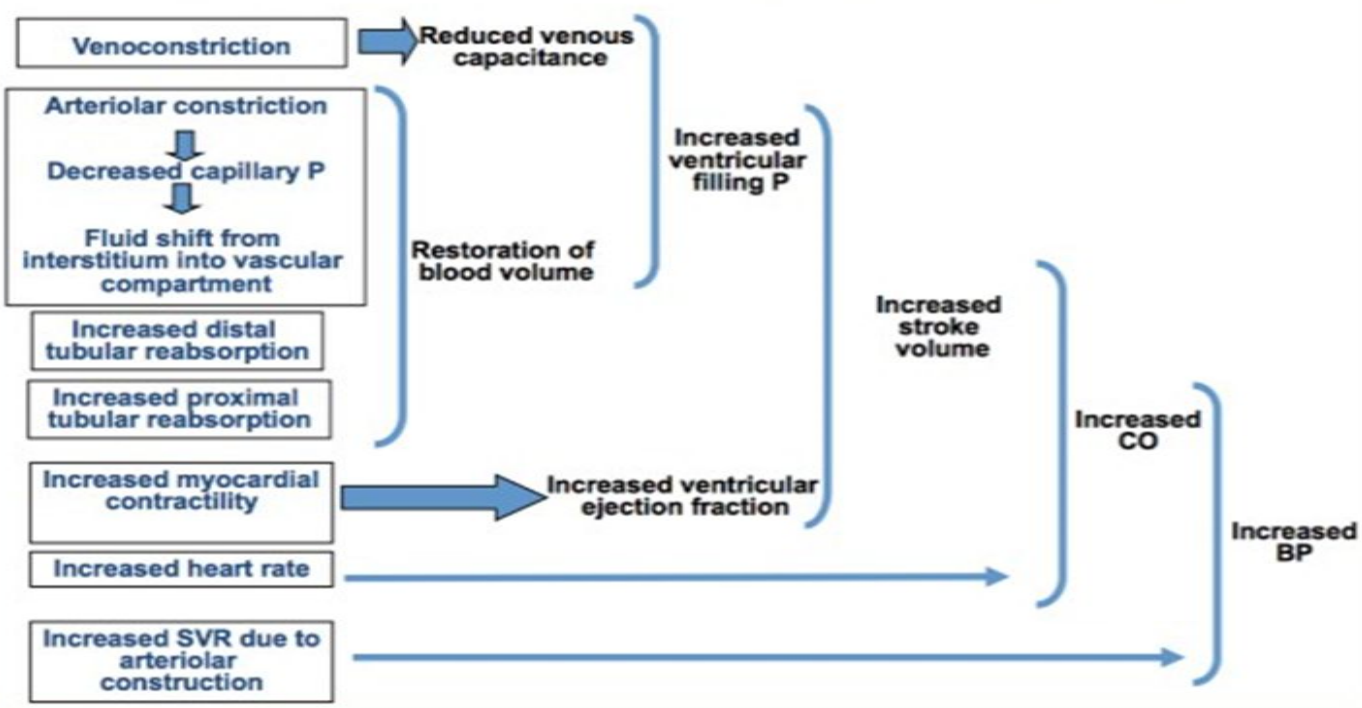
- ❑ Norepinephrine is released from the adrenal gland, it acts on alpha receptors which causes vasoconstriction. It's usually given in distributive shock.
  - ❑ Epinephrine ⇒ 50% α1 and 50% β2
  - ❑ Norepinephrine ⇒ 80% α1 and 20% β2
- ❑ Thus, NE has a more desirable effect in shocked patients

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.



- Vasoconstriction → reduced venous capacity → increase ventricular filling → increased stroke volume → increase BP
- Bleeding → ↑contractility → ↑ ejection fraction → ↑ CO → ↑BP

Recall :

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, T mental status and pulse pressure, Poor capillary refill, Poor urine output
What are the best indicators of tissue perfusion?
Urine output, mental status
What lab tests help assess tissue perfusion?
Lactic acid (elevated with inadequate tissue perfusion), base deficit, pH from ABG (acidosis associated with inadequate tissue perfusion)

1- HYPOVOLEMIC SHOCK:

Decrease in intravascular blood volume → decrease in cardiac output and tissue perfusion " commonest and most readily corrected cause of shock ".

Causes:

- Hemorrhage. e.g. **trauma, haematemesis, ruptured aortic aneurysm**, aortic dissection. "The commonest"
- Dehydration[3]: severe Vomiting/Diarrhea, diabetic ketoacidosis
- Fluid sequestration:
  - Intraluminal – bowel obstruction
  - Intraperitoneal – pancreatitis "third spacing[4]"
  - Interstitial - burns "due to loss of plasma " "evaporation due to loss of barrier"

Table 1.14 Causes of haemorrhagic hypovolaemic shock

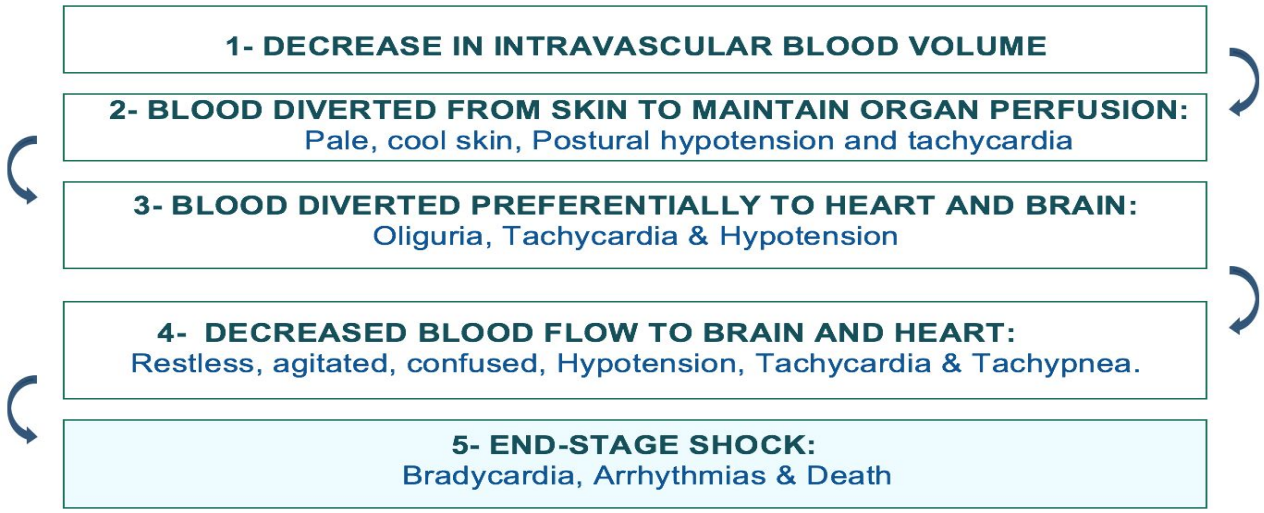
Gastrointestinal haemorrhage
- Oesophageal varices
- Oesophageal mucosal (Mallory–Weiss) tear
- Gastritis
- Gastric and duodenal ulceration
- Cancer
- Diverticula

Trauma

Ruptured aneurysm

Obstetric haemorrhage
- Ruptured ectopic pregnancy
- Placentia praevia
- Placental abruption
- Post-partum haemorrhage

Pulmonary haemorrhage
- Pulmonary embolus
- Cancer
- Cavitating lung lesions e.g. TB, aspergillosis
- Vasculitits



Treatment: replace volume + treat the underlying cause "e.g. someone is bleeding from his spleen should **remove** it surgically, diarrhea due to severe gastroenteritis should treat the gastroenteritis/ulcerative colitis/crohn's disease "

Hypovolaemic shock can be divided into four categories, depending on the amount of blood loss : (class I, II, III, IV).

- you need to memorize it as you'll be asked about it in exams…

	Blood loss (ml)	Pulse	Blood pressure	Symptoms:
Class I:	< 750 (<15%)	< 100 (15-30%)	Normal	minimal symptoms
Class II:	750-1500	>100(15-30%) Tachycardia	Decrease "Hypotension"	- tachypnea - decreased pulse pressure - pale, sweaty and cold peripheries. - anxious.
Class III:	1500-2000	>120 (30-40%) Tachycardia	Decrease "Hypotension"	- Classic symptoms of shock, - tachypnea - pallor - cold peripheries - oliguria ,- decreased conscious level **"confused"**
Class IV:	>2000	>140 (>40%) Tachycardia	Decrease "Hypotension, unobtainable diastolic"	- Immediate threat to life - pallor, - cold peripheries - anuria, unconscious (>50%) "Lethargic".

[3] loss of water and electrolytes

[4] **Third-spacing** occurs when too much fluid moves from the **intravascular space** (blood vessels) into the interstitial or "third" space-the nonfunctional area between cells. This can cause potentially serious problems such as edema, reduced cardiac output, and hypotension.

Recall :

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

How is the effectiveness of treatment evaluated:

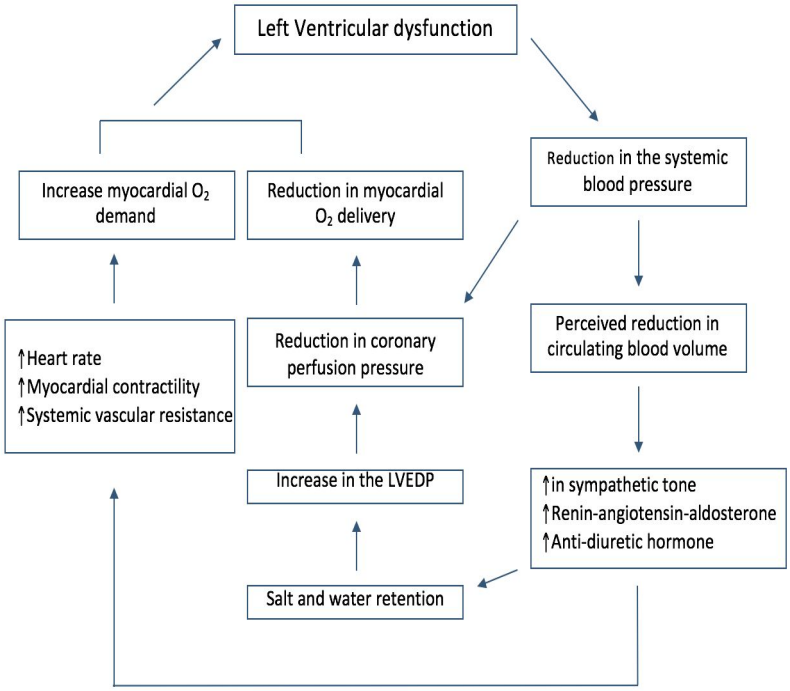
- **Bedside indicator?** Urine output, BP, heart rate, mental status, extremity warmth, capillary refill, body temperature
- **Labs?** pH, base deficit, and lactate level

What usually causes failure of resuscitation?
Persistent massive hemorrhage, requiring emergent surgical procedure
Why does decreased pulse pressure occur with early hypovolemic shock?
Pulse pressure (systolic–diastolic BP) decreases because of vasoconstriction, resulting in an elevated diastolic BP
What is the most common vital sign change associated with early hypovolemic shock?
Tachycardia
What type of patient does not mount a normal tachycardiac response to hypovolemic shock?
Patients on -blockers, spinal shock (loss of sympathetic tone), endurance athletes
Should vasopressors be used to treat hypovolemic shock?
No
Should patients with hypovolemic shock be put into the Trendelenburg position?
No

2- CARDIOGENIC SHOCK:

This occurs when the heart is unable to maintain a cardiac output sufficient to meet the metabolic requirements of the body

- Decreased ventricular function.
  - **Acute Myocardial Infarction**
  - **Pericardial tamponade**
  - **Tension pneumothorax**
- Ineffective cardiac contraction.
  - Primary arrhythmias



E.g. 80 years old guy hyperlipidemic hypertensive diabetic came with chest pain radiating to the left arm, his BP is 60 and pulse rate is 50 (he is on beta-blockers). He is cold clammy not producing urine. What type of shock? Cardiogenic.

How do you know if it is cardiogenic shock or not?

- Shortness of breath (SOB).
- Raised JVP.
- Lower limb edema.
- Basal crepitation.

CLINICAL FINDINGS:

Hypotension - Tachycardia - Tachypnea - Oliguria.

Etiology:

- Caused by the progressive loss of myocardium, Usually due to an acute (anterior) 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.
  a. Decreased myocardial blood flow.
  b. Decreased contractility=decreased stroke volume.
  c. Heart rate increases to maintain CO (CO=HR X SV).
  d. If HR cannot maintain CO, then compromised end-organ perfusion.
  e. Decreased myocardial perfusion leads to further decrease in contractility.

Treatment:

- Treat the underlying cause. E.g. beta agonists re-perfuse the heart in MI and restore its function. Thrombotic event? Antithrombolytics. Atherosclerosis? DM? HTN? Hyperlipidemia? Platelets aggregation? Aspirin. Digoxin to support the pump. You don't need to know what medication to give exactly but the point is to support the pump.
- 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 flow back to the 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**

19 years old involved in a RTA[5] with flail chest, hemodynamically stable (BP=120, pulse=110). All of a sudden developed difficulty to ventilate, drop in saturation, drop in BP to 60. No breath sound on the left side. Shock? Yse. cardiogenic? Yes, specifically obstructive. The heart can't expand due to increase pressure (air) around it (pneumothorax). The treatment is (MCQ):
A- give hem fluid? B- Cath lab? C- needle thoracostomy? Answer: C

Recall :

CARDIOGENIC SHOCK
What is the definition? Cardiac insufficiency; left ventricular failure (usually), resulting in inadequate tissue perfusion
What are the causes? MI, papillary muscle dys unction, 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 S2
- 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

What are the last resort support mechanisms?
Intra-aortic balloon pump (IABP), ventricular assist device (VAD)

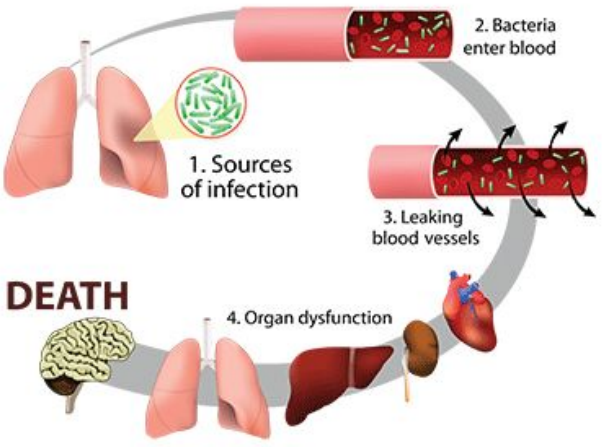
3- Distributive shock:

a great video

• SEPTIC SHOCK:

Severe infection and release of microbial products (release of vasoactive mediators).
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.
commonest sites of infection leading to sepsis are the lungs (50–70%), abdomen (20–25%), urinary tract (7–10%) and skin.
Infection triggers a **cytokine-mediated proinflammatory response** that results in HYPERDYNAMIC STATE (warm shock > early stage):

- Peripheral vasodilation, - Increased cardiac output
  "Fever, tachycardia, tachypnea, warm skin":
  1. Maintenance of intravascular volume **"Hyperdynamic shock"**
  2. Failure to maintain intravascular volume **" Hypodynamic shock"** (cold shock > late stage) "Cool skin, tachycardia, hypotension, oliguria"



[5] Road Traffic Accident.

Treatment: treat the underlying cause + **give antibiotics** and **replace volume**.

- In hyperdynamic sepsis, the peripheral arteriolar tone and BP are low but the cardiac output is often high; therefore the **vasoconstrictor noradrenaline** (norepinephrine) is appropriate to restore BP, usually at the price of some reduction in cardiac output.

A 70 year old guy, hyperlipidemic hypertensive diabetic (IHD) got diabetic foot ulcer. You gave hem fluid and antibiotics and debrided the ulcer. He had no signs of sepsis. Suddenly, he is in the ICU, he progressed and died! Why? SIRS. The damage was irreversible. Hypotension → ↓perfusion to kidneys → ↓GFR → oligouria.
when the inflammatory response become beyond what we able to control or the damage is irreversible

Systemic Inflammatory Response Syndrome (SIRS):

- The patients demonstrate a similar response as sepsis but without infective agents. It's just an inflammatory process.
  - The criteria: (**two or more** to call it SIRS)
    - Temperature >38 or < 36 (in sepsis it could be **hypo**thermia OR **hyper**thermia). maybe high or low
    - Heart rate >90 tachycardia
    - RR>20 or a pco2<34 mmHg (4.3kpa)
    - WBC > 12,000 or < 4,000 with more than 10% bands High

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.
Pulmonary edema, basement membrane of the alveoli will release mediators which move fluid from the ECF to the alveoli can't exchange gas, shifting of normal protein, hypoxemia. High mortality.

Recall :

What is septic shock?
Documented infection and hypotension
What is the specific etiology?
- Most common -- gram-negative septicemia
- Less common -- gram-positive septicemia, ungus

What actors 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?
- Initia -- vasodilation, resulting in warm skin and full pulses; normal urine output
- Delayed -- vasoconstriction and poor urine output; mental status changes; hypotension

What percentage of blood cultures is positive in patients with bacterial septic shock? Only about 50%!
What are the associated findings? Fever, hyperventilation, tachycardia
What are the associated lab findings?
- Early -- hyperglycemia/glycosuria, respiratory alkalosis, hemoconcentration, leukopenia
- Late -- leukocytosis, acidosis, elevated lactic acid (Note: Identifying organism is important to direct treatment/antibiotics)

What is the treatment?
1. Volume (IVF)
2. Antibiotics (empiric, then by cultures)
3. Drainage of infection
4. Pressors PRN
5. Zygris® PRN

● Neurogenic Shock:

It is a shock that result from a high spinal cord injury (e.g Cervical spine 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**. Loss of sympathetic tone will result in:
  - Arterial and venous dilatation causing **hypotension**.
  - 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 (cervical support)
3. Volume resuscitation
4. Rule out other causes of shock
5. High dose corticosteroids. (as anti-inflammatory to decrease edema)
6. In the non-trauma setting neurogenic shock is self-limiting.

Cont. 19 years old involved in a RTA. He is awake, you examine them and find out that he can't move his legs (loss of tone), tenderness at T5, his skin is normotonic.
Spinal cord injury → loss of epinephrine and norepinephrine → loss of tone → distributive shock.
"RTA cause either hypovolemic or neurogenic shock"

Treatment: Septic, anaphylactic, neurogenic shocks are all distributive (same pathophysiology), however they're treated differently.
septic: antibiotic
Anaphylactic: epinephrine, antihistamine, and corticosteroids, because it's immune response
Neurogenic: high dose of synthetic epinephrine and norepinephrine → vasoconstriction & treat the spinal injury.

Recall :

What is neurogenic shock? 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 or 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
What is the acronym or treatment options for anaphylactic shock? "**BASE**":

- **B**enadryl
- **A**minophylline
- **S**teroids
- **E**pinephrine

Diagnosing Shock state based on hemodynamic parameter:

Type	Central venous pressure	Cardiac output	SVR
Hypovolemic	↓	↓	↑
Cardiogenic	↑	↓	Normal or ↑
Septic	↓ or ↑	↑	↓
Traumatic	↓	↓ or ↑	↓ or ↑
Neurogenic	↓	↓	↓
Hypoadrenal	↓ or ↑	↓ or ↑	↓ or ↑

Principles of resuscitation :

- **Maintain ventilation:** ensure oxygen delivery

  Increased oxygen demand, Especially in: Sepsis, Hypovolemia & Trauma → Hyperventilation → Respiratory fatigue → Respiratory failure: Respiratory acidosis, lethargy-coma, hypoxia → Death
- **Enhance perfusion**
- **Treat underlying cause**

Major differences between types of shock:

	Hypovolemic	Cardiogenic	Septic	Neurogenic
HR	Tachycardia	Tachycardia	Tachycardia	Bradycardia
Skin	Cold and clammy	Cold and clammy	Warm	Cold and clammy
Treatment	Give fluid	Don't give fluid	Antibiotics	epinephrine and norepinephrine
	+ Treat the underlying cause			

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:

CIRCULATORY shock	SEPTIC shock
Critical reduction in tissue perfusion results in organ dysfunction and, if untreated, death	organ dysfunction due to dysregulated host response to infection
Usually accompanied by signs and symptoms: ·Oliguria. ·Mental status changes. ·Weak thread pulse. ·Cool clammy limbs.	· Hypotension · Vasodilation with warm limbs.

5. Treatment of shock is primarily focused on **restoring tissue perfusion** and **oxygen delivery** while eliminating the cause
    - Give fluids except cardiogenic shock
    - Give O2
    - Give pharmacological agents to restore perfusion (epinephrine vs norepinephrine)

Notes:

- splanchnic circulation and skin will vasoconstriction by neuroendocrine mechanism to push blood into vital organs when shock occurs.
- What cause neurogenic shock? Spinal injury (usually above C3) lose of sympathetic flow → loss of vasoconstriction → parasympathetic flow → shock
- How to restore volume? Kidney (decreased urine output) + movement of fluid from interstitial to vessels, decreased venous capacitance
- Only shock you DON'T give fluids is Cardiogenic shock → it will cause PULMONARY EDEMA. (Cardinal signs of HF: JVP + Pulmonary edema)
- low urine output patient → check first if he has low volume OR urinary tract obstruction/ kidney failure
- Case: pt w low uo → u give fluid → severe abdominal pain → obstruction?
- Septic shock: (vasodilation due to microbiological mediators)
- warm extremities → vasodilatation + tachycardia → blood flowing fast to extremities → warm extremities
- cold extremities → in late stage
- Hypotension + bradycardia + trauma = neurogenic shock → be careful in airway assessment you may increase the spinal injury