Shock

Objectives

- Define Shock.
- List the types and clinical features of shock.
- Define the terminology distributive and obstructive shock.
- Discuss the pathophysiology of shock (Macrocirculation, Microcirculation, Cellular function).
- Discuss the systemic effects of shock.
- Discuss the general principles of management (airway, breathing and circulation).
- Discuss the specific treatment of each type of shock.

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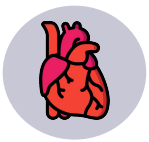
Summary File

Editing File

Shock:

What is shock?

- Shock is syndrome that result in death if it is not treated , and it is final step before the body give up. It is easy to detect the shock in the last stage however our job is to detect it in the early stage before coming very profound because as shock get worse reverse from shock will be harder.
- Inadequate oxygen delivery to meet metabolic demand. (There might be good oxygen supply but the metabolic demand increased or normal metabolic demand but oxygen supply decreased)
- Results in global tissue hypoperfusion and metabolic acidosis.[1]
- To be more precise the hallmark of shock is tissue hypoperfusion then metabolic acidosis develops.
- Shock can occur with normal blood pressure, and hypotension can occur without shock.[2] initially we can detect some signs ex. an increase in the lactate level caused by metabolic acidosis however some patients might die due to shock while having normal lactate levels.
- Oxygen delivery is the function of the circulatory system. This system[3] is basically:



Pump (heart[4])

Pipes (vessels)

- It regulates the system by shunting blood from less O2 demanding tissues to the more O2 demanding tissue.
- It maintains normal vascular tone by **vasoconstriction** to allow enough pressure for blood flow.

Solution (blood)

- Needs to function at adequate pressure (depends on the individual) , volume and O_2 carrying capacity[5].

Understanding shock:

- Inadequate systemic oxygen delivery activates autonomic responses (first thing to be activated) to maintain systemic oxygen delivery. Our bodies response to any lack of O2 delivery rapidly by activating the three parts above no matter what's the cause of hypoxia. During clinical assessment you'd see all three parts are activated except the site of defect because it's the most likely cause of shock. Ex: in septic shock you'll see tachycardia, urine output is decreased to conserve water but the skin is warm, flushed because the defect is in the vessels which causes vasodilation due to cytokines & bacterial toxins…etc . Another ex: A cardiogenic shock resulted from MI: you'd see all the parts are activated except the heart, so you'll not see tachycardia.
- When body sense low O_2 it tries to alarm us be activating the sympathetic system to make us feel anxious and to increase the heart rate, sometimes this stimulation is enough and sometimes damage might happen.
- Body mechanisms to compensate (important):

Sympathetic nervous system

- NE, epinephrine, dopamine, and cortisol release causes vasoconstriction to shunt the blood from non-vital organs eg. skin , muscle , and kidney to vital organs eg. heart and brain, the heart muscle and brain they are the most vital organs so there is no vasoconstriction, increase HR (chronotropy), and increase cardiac contractility (inotropy) (cardiac output) to stimulate the pump and increase HR and CO

Renin-angiotensin axis

- Water and sodium conversation and vasoconstriction. (by angiotensin II)
- Increase in blood volume and blood pressure.

- Not any patient will go into shock especially if the patient is young. As they have 5L of blood , vascular tone , a heart pumping 5L at rest, and the capacity to compensate if the demand increases (ex. if 7L or 12L is needed instead of 5L the healthy heart can do it and shock doesn't develop) , but if the patient is elderly or middle aged with comorbidities such as DM , HTN or IHD and the demands increased their heart can't compensate that way they go into shock. So the problem with compensation is in the elderly because even if these mechanisms happened it won't compensate same as younger patients and that is the reason why heart problems are seen more commonly in elderly.
- Hemoglobin isn't one of the mechanisms for compensation in case of shock because it requires time. This mechanism is helpful in case of smokers since it is a chronic case but bleeding is considered an acute case.

1. Hypoperfusion and ischemia will shift the cells to anaerobic glycolysis and the production of lactate causing acidosis.
2. Shock is more than hypotension, don't restrict yourself on the BP, you should think about molecular changes.
3. Failure in any of these can result in a different type of shock.
4. The heart is designed to pump about 5L of blood per minute. In young healthy adults it can pump more, and in some athletes it can pump up to 13L. Thus in abnormal situations in healthy young adults the heart is able to pump more to compensate and the situation won't progress to shock. However, in an elderly patient with underlying conditions and a maximum capacity of 5L AT REST the heart won't be able to meet the demand when there's an increased effort or when they get an infection or bleeding and their condition will progress to shock.
5. In any given moment, about 30% of the vessels are closed (shunted blood is part of the normal circulatory system) e.g. sitting and relaxing after having a heavy meal, will cause shunting of the blood from muscle to your splanchnic circulation to optimize digestion and absorption; that is whywe can't eat during exercises .

Shock:

Understanding Shock:

Cellular responses to decreased systemic oxygen delivery

- ATP depletion[1] ⟶ Ion pump dysfunction
- Cellular edema[2]
- Hydrolysis of cellular membranes and cellular death

Regardless to the essential role of O2 in many functions like muscle & cardiac contractility … etc , when coming to the basic level every cell needs O2 (ATP) to maintain the electrolyte balance inside and outside the membrane, for its integrity and to stay alive, that's why hypoxia is killer. NA , K and H pump need enough ATP to maintain electrolytes balance.

The body tries to maintain vital organs: cerebral and cardiac perfusion[3]

- Vasoconstriction of splanchnic, musculoskeletal, and renal blood flow.

As shock progresses, vasoconstriction, blood shunting away from other organs to the most vital organs (brain & heart) but in advanced cases they get hypoxic.

Global cellular reliance on anaerobic glycolysis and increased lactate production[4]

High lactate level is a bad sign but not necessarily! Sometimes the patient is too sick to switch to anaerobic and they die from shock with normal lactate levels, and some others cannot generate lactate, so low lactate doesn't mean that the patient isn't in shock, and the significance of it is that when the lactate is high and it gets normalized after the management.

Systemic metabolic lactic acidosis [5] (The hallmark of shock)

Aerobic metabolism $\xrightarrow{\text{Hypoxia}}$ Anaerobic metabolism ⟶ Lactic acid ⟶ ↓PH lactic acidosis ⟶ Homodynamic + Metabolic disturbance ⟶ Reversible cell injury ⟶ Irreversible cell injury ⟶ Death

Multi-organ Dysfunction Syndrome (MODS):

- Progression of physiological effects as shock ensues[6]:

01	02	03	04
Cardiac depression	Respiratory distress	Renal failure	DIC[7]

- Result is end organ failure.
- As a physician you should be able to detect shock signs before reaching this stage, because in septic shock with:
  - 1 organ failure the risk of mortality is 30%
  - 2 organs failure the risk of mortality is 50%
  - 3 organs failure the risk of mortality is 90%

1. Due to oxygen insufficiency which will shift the cells to anaerobic glycolysis (Aerobic glycolysis produces 36 + 2 ATP while anaerobic glycolysis produces only 2 ATP).
2. Normally, Na/K pump will remove excess Na+ from the cells, but with ion pump dysfunction(Anaerobic metabolism بسبب ال ATPقل لان بسبب ما) Na+ influx will occur and it will withdraw water with it, which will cause swelling and edema.
3. The most important vital organs. If vasoconstriction is intact, there will be vasoconstriction everywhere except in the cardiac and the cerebral vessels.
4. Under normal (aerobics) conditions glycolysis converts glucose to pyruvate which is converted to (acetyl-CoA) and enters the Krebs cycle to generate (NADH) and (FADH2), which enter the ETC to produce ATP. In hypoxaemia aerobic glycolysis is blocked, leading to pyruvate accumulation, which will be converted to lactate enabling the limited production of ATP by anaerobic glycolysis generating only 2 moles of ATP.
5. Accumulation of lactic acid reduces intracellular pH together with ATP depletion (failure of vital ATP dependent cell functions) results in disruption of protein synthesis, damage to lysosomal and mitochondrial membranes, and ultimately cell necrosis.
6. As the shock progresses it gets harder to differentiate between the different types of shock as they will start to overlap.
7. When a cell dies it releases mediators and tissue factors and activates the coagulation cascade which will ultimately lead to disseminated intravascular coagulation.

Types of shock:

Low cardiac output states (Hypovolemic & Cardiogenic):[1]

The hallmark of these types is an intact vascular resistance (closure of vessels), thus you see cold & dry skin, delay capillary refill...

1 **Hypovolemic** Most common

2 **Cardiogenic**

3 Neurogenic

4 Vasogenic

1 Hypovolemic (↓solution)

- Patient most commonly will have history of trauma, the bleeding either internally or externally.
- (Blood pump is working with no blood) most common and most readily corrected cause of shock encountered in surgical practice results from a reduction in intravascular volume secondary to loss of:
  - Blood e.g. bleeding (the most common cause of acute hypovolemic shock in surgical practice)
  - Plasma (e.g. burns).
  - Water and electrolytes (dehydration[2]) (e.g. vomiting, diarrhoea , diabetic ketoacidosis).
- In dehydration, there's a low cardiac output state. However, the heart is working just fine. There is less blood volume to carry oxygen, thus the mediators (such as NE and hormones) will force the heart to increase tropic volume and inotropy. This increase is up to a certain point then a shock might happen.
- Clinical features: heart is functional well and strong **tachycardia** (to compensate) + increased inotropy + high vascular resistance as result of compensatory mechanism so the patients will come with delayed capillary refill + **cool temp** + **dry skin**, muscle will be deprived of blood and dysfunctional.
- In hypovolemic shock (especially in haemorrhage) the skin at the beginning will be clammy (wet and cold), when body loses more volume it will try to conserve fluid so there will be no sweating (dry).

2 Cardiogenic (↓pump)

- Impaired inflow e.g. tamponade (compression of the heart by an accumulation of fluid in pericardial sac), constrictive pericarditis and tension pneumothorax
- Primary pump dysfunction e.g.HF, ACS leading to MI,Myocarditis
- Impaired outflow e.g. massive PE
- In cardiogenic shock, there's adequate blood volume but the heart itself is abnormal. We will notice blunted heart response to stimulatory mediators such as NE and epinephrine.
- The patient might be tachycardic (or even bradycardic) but with ECG we find that stroke volume is low, some sick hearts can increase the rate but cannot increase the volume some patients will enter bradycardia in later stages since the heart is too sick to compensate with tachycardia.
- Clinical features: tachycardia, vasoconstriction, cold clammy skin, systemic vascular resistance will be high.
- Same signs of hypovolemic (vasoconstriction, cold clammy skin…) but the heart function is impaired as in MI.
- In cardiogenic shock:
  - The main problem related to heart function (decrease amount of O2 > hypoxia > activation of sympathetic system > irregular tachycardia or bradycardia) .
  - High vascular resistance as result of compensatory mechanism so the patients will come with (cold clammy skin .
  - Blood volume is normal.

1. Using ultrasound probe can confirm whether the heart is functioning (in hypovolemic) or not (in cardiogenic).
2. Dehydration is less common, but it complicates other types of shock e.g. patient with infection (septic shock) and severe vomiting → dehydration (caused by the vomiting) → hypovolemic shock along with the septic shock.
3. The skin is the 1st organ in which blood is shunted from unless it's sepsis/anaphylaxis and the vasodilation is the issue so the blood is more to skin.
4. First organ to lose blood: 1st: skin 2nd: GI (splanchnic circulation) —> bowel edema + ischemia 3rd: muscle 4th: Renal blood flow——> decrease urine output, kidney dysfunction

Types of shock:

Low peripheral resistance states,↑pipes (Neurogenic and Vasogenic):

1 Hypovolemic

2 Cardiogenic

3 **Neurogenic**
Cerebral or spinal cord injury

4 **Vasogenic**
AKA (distributive shock)

3 Neurogenic

- Loss of sympathetic tone (this will cause bradycardia and vasodilation)
  - Complete loss of sympathetic tone that normally maintains some vasoconstriction.
  - **Loss of sympathetic tone** also affects the heart, so the heart won't be able to compensate the vasodilation with tachycardia.
  - This typically occurs following injury to the thoracic or cervical spinal cord.
  - A temporary drug-induced form can also occur in 'high' spinal anaesthesia.
- Important: since both a neurogenic shock and a hemorrhagic shock can result from trauma always make sure your patient is not bleeding, treat as hemorrhagic shock after which take a step back and determine whether the patient is tachycardic or not? Bradycardia and warm skin indicate a neurogenic shock.
- Neurogenic shock is very rare and it doesn't happen to anyone, you should have a specific injury such as spinal cord injury or severe traumatic nerve injury, usually it happens after a trauma but what also happens with trauma? BLEEDING (it's almost always bleeding) so when you treat a patient in shock caused by trauma you should resuscitate and search for the bleeding and if you couldn't find a bleeding and the patient is bradycardic and his skin is warm then you can think about neurogenic. **Any shock after trauma we assume it's Bleeding because it's the most common cause of shock after trauma and it's more dangerous and it needs blood restore unlike the neurogenic which is rare and can be controlled with vasopressors.**
- Clinical features: **Bradycardia** and **vasodilation** are very characteristic of the neurogenic shock

4 Vasogenic

- **In all types of shock, the pulse pressure is (low-narrow), but in vasogenic (distributive) shock the pulse pressure is (high-wide).**
- The hallmark of this type is impaired vascular tone, due to anaphylactic infectious reactions that cause vasodilators releasing, so you see warm skin, capillary refill is intact, sweating.
- The blood volume is normal, heart is functioning normally but the **vessels are leaky and dilated** (warm and flushed skin) , thus, the heart needs to push through all of that. Young healthy individuals can compensate with **tachycardia**, but if the heart isn't able to compensate (as in an elderly patient) then shock might happen.
- Septic[1]
  - Bacteria (gram + bacteria is the most common cause) > toxins > cell damage > release of cell mediators > uncontrolled vasodilation and leakage.
  - Patient is febrile (having the symptoms of fever) and it takes time to develop.
- Anaphylactic
  - Allergic reaction resulting in the the release of histamine which causes vasodilation and shortness of breath.
  - Clinical features: low peripheral vascular resistance (warm skin and sweating), heart is intact and try to compensate (tachycardia).
- **You can diagnose and differentiate between the anaphylactic and septic shock based on patient history and clinical features:**
  - Patient with anaphylactic shock will come with urticaria and more respiratory symptoms (very bad wheezing and bronchospasm)
  - Anaphylaxis most commonly affect young patients, that's why they rarely progress to shock because they have good response to the compensatory mechanisms. However, anaphylaxis might develop into shock if left without intervention.

1. As septic shock progresses cardiac ventricular dysfunction impairs the compensatory increase in cardiac output. As a result, peripheral perfusion falls and the clinical signs may become indistinguishable from those associated with the low cardiac output state.

Types of shock:

You have to know how to diagnose shock based on hemodynamic parameters.

Shock type	Hypovolemic	Cardiogenic	Obstructive[4]	Distributive[2]	Neurogenic	Dissociative[5]
Example	-Hemorrhage -Dehydration	-Myocarditis -Dysrhythmia	-Tamponade -Tension pneumothorax (Once a tension pneumothorax occurs air can continue to accumulate within the pleural space but cannot escape. This continued accumulation of air increases pressure and ultimately obstructs venous blood return to the heart)	-Sepsis -Anaphylaxis	-Spinal cord injury -Traumatic brain injury	-Carbon monoxide -Cyanide
HR	↑	↑[1]	↑	↑	↓	↑
BP[6]	↓	↓	↓	↓	↓	Normal or ↑
CO (cardiac output)	↓	↓	↓	↓ Or ↑[3]	↓	↑
Capillary Refill	Delayed	Delayed	Delayed	Flash or delayed	Flash or normal	Normal
Extremity Temperature	Cool	Cool	Cool	Warm or cool	Warm (due to vasodilation)	Normal
SVR (systemic vascular resistance)	High	High	High	Low or high	Low	Low to normal
Treatment	- Stop bleeding -Fluid resuscitation	-Inotropes -Caution with fluids -ECMO Kissing ventricles (reduced ejection fraction) one of the features that can be seen in Echo to diagnose cardiogenic shock.	-Pericardiocentesis. (is a procedure done to remove fluid that has built up in the sac around the heart (pericardium). -Chest tube (A plastic tube that is used to drain fluid or air from the chest).	-Antibiotics -Fluids -Epinephrine	-Fluid resuscitation -Vasopressors	-Antidotes (drugs that negate the effect of a poison or toxin) -Hyperbaric therapy

In summary : Pathogenesis of shock : Decrease amount of oxygen will shift cell from aerobic glycolysis to anaerobic glycolysis , cell will start to produce markers and acid > The body will sense the hypoxia > will turn on the all corrector mechanisms > therefore failure of one of the correcting mechanism will help you to detect the cause of shock . For example 1) Patient with flushed skin , high temperature and he was still in shock , most likely the shock related to vessels they loss of vascular tone so the cause could be anaphylaxis, sepsis , or neurogenic shock 2) Patient with cold skin indicate an intact or exaggerated vascular tone result of shunted away of blood so the cause not related to vessels the cause could be related to the heart cardiogenic shock , or hypovolemic shock.

-Corrector mechanismes : V.C , increase heart function , conserve the water and sodium.

Classes of Hypovolemic Shock:

- Shock is a spectrum, not zero or one.
- There are four classes of hypovolemic shock depending on the blood loss.
- The more blood loss the more/severe symptoms.
- We shouldn't rely on the patient's blood pressure because hypotension is late finding.
- To detect shock we look at the end organ perfusion, most importantly the brain, it's very sensitive to oxygen delivery. Secondly, the kidney through the urine output (the less blood flow the less urine).

This table isn't important clinically. However, it's important academically and you might get asked about the numbers in this table

#439 Dr : It is good know the values for USMLE exam but personally I don't ask about it

	I	II	III[7]	IV[7]
Blood loss (mL)	Up to 750	750–1500	1500–2000	> 2000
Blood loss (% blood volume)	Up to 15	15–30	30–40	> 40
Pulse rate (per minute)	< 100	100–120	120–140	> 140
Blood pressure	Normal	Normal	Decreased	Decreased
Pulse pressure (mm Hg)	Normal or increased	Decreased	Decreased	Decreased
Respiratory rate (per minute)[8]	14–20	20–30	30–40	> 35
Urine output (mL/hour)	> 30	20–30	5–15	Negligible
Central nervous system/ mental status[9]	Slightly anxious	Mildly anxious	Anxious, confused	Confused, lethargic

1. Initially tachycardia followed by bradycardia if not treated.
2. Initially may appear as hypovolemic shock that's why we hydrate the patients first until symptoms of septic shock (like warm skin) start to appear. Big examples seen in patients with acute abdomen (gastroenteritis) this is not seen in patients with sepsis of the chest or anywhere else (if patient came with typical symptoms of shock complaining of abdominal pain (gastroenteritis) + culture was positive for microorganism think about hypovolemic shock caused by a dehydration FIRST. Anywhere else = septic shock.
3. In distributive shock (also called vasogenic shock, including septic and anaphylactic shock) cardiac output may initially increase, then decrease in cases where the heart is fatigued or when the heart itself is a part of the problem e.g. if the heart is the source of the infection that caused sepsis.
4. Obstructive shock is a type of cardiogenic shock where the problem is outside the heart obstructing it. eg: tension pneumothorax, pulmonary embolism, and cardiac tamponade.
5. In dissociative shock, everything is normal. However, there's no oxygen delivery due to abnormal hemoglobin.
6. BP could be normal in the early stage of shock.
7. Note that BP starts to decrease in advanced classes (III, IV).
8. We check for reduced O2 supply in end organs such as kidney and brain by ordering their respective function tests.
9. Mental status deteriorates very early because the brain is very sensitive to O2.

Pathophysiology of shock:

Pathophysiology of Shock (macro-micro circulation):

- There is often significant overlap between the causes of shock, ex: patients with septic shock are frequently also hypovolemic. All share common pathophysiological features at the cellular level. Whilst differences can be detected at the level of the **macrocirculation**:

Hypovolemic

Fall in intravascular volume results in a fall in cardiac output > tachycardia and increased myocardial contractility to preserve cardiac output whilst vasoconstriction acts to maintain arterial blood pressure.

Cardiogenic

Unlike hypovolemic shock, circulating volume is typically normal or increased secondary to increased circulating AT-II and aldosterone levels, If associated with left ventricular failure, there may be pulmonary oedema.

Neurogenic

Loss of cardiac accelerator fibers, anhydrosis (inability to sweat normally because the sweat gland innervated by the sympathetic nervous system so it will damage here) as a result of loss of sweat gland innervation (patient presents with warm dry skin).

Septic

Discussed in page 5 footnote 2

- In **microcirculation** level arteriolar vasoconstriction, seen in early hypovolemic and cardiogenic shock, helps to maintain a satisfactory MAP, and the resulting fall in the capillary hydrostatic pressure encourages the transfer of fluid from the interstitial space into the vascular compartment
- If shock remains uncorrected:

1. Complications of shock override compensatory vasoconstriction leading to precapillary vasodilation.
2. Pooling of blood within the capillary bed and endothelial cell damage.
3. Capillary permeability increases with the loss of fluid into the interstitial space and increase in blood viscosity
4. Further compromises flow through the microcirculation, predisposing to platelet aggregation and the formation of microthrombi.

- In sepsis, endothelial and inflammatory cell activation results in widespread activation of coagulation.
- Microthrombi include capillary blood flow, and the consumption of platelets and coagulation factors leads to thrombocytopenia, coagulopathy and DIC.

Pathophysiology of shock:

Pathophysiology of Shock (cellular function):

- We've discussed previously the shifting of aerobic glycolysis to anaerobic glycolysis in page 3.
- Under normal conditions, the tissues globally extract about 25% of the oxygen delivered to them, with the normal oxygen saturation of mixed venous blood[1]. As oxygen delivery falls, cells extract about 50%.
- Further reductions in oxygen delivery lead to a critical reduction in oxygen consumption and anaerobic metabolism, a state described as dysoxia.
- Sepsis is associated with significant mitochondrial dysfunction and marked inhibition of oxidative phosphorylation. The phrase 'cytopathic shock' has been used to describe this condition.

The effect of shock on individual organ system:

	Effect
CVS	• Ischaemia in the watershed areas of the endocardium. This impairs myocardial contractility. • Acid–base and electrolyte abnormalities, combined with local tissue hypoxia, increase myocardial excitability and predispose to both atrial and ventricular dysrhythmias. • In sepsis inflammatory mediators depress myocardial contractility and ventricular function, increase endothelial permeability (resulting in intravascular volume depletion) and DIC.
Nervous system	• Due to the increased sympathetic activity, patients may appear inappropriately anxious, there is increasing restlessness, progressing to confusion, stupor and coma. • In septic shock, the clinical picture may be complicated by the presence of an underlying (septic) encephalopathy (brain dysfunction mediated by septic inflammatory response) and/or delirium.
Respiratory system	• Tachypnoea driven by pain, pyrexia (fever), local lung pathology, pulmonary oedema, metabolic acidosis or system cytokines is one of the earliest features of shock. • The increased minute volume typically results in reduced arterial PCO2 and a respiratory alkalosis (compensating for the metabolic acidosis). • Sepsis (injured the lung because of the circulatory inflammatory mediators) and hypovolemic shock are both recognised causes of acute respiratory distress syndrome.
Renal system	• Reduced renal blood flow results in the production of low-volume, high-osmolality and low sodium content urine. • Hypoxia leads to acute tubular necrosis. • With a fall in glomerular filtration, blood urea and creatinine rise; hyperkalemia and metabolic acidosis are also usually present. • Renal failure occurs in about 30–50% of patients with septic shock.
GIT	• Marked reduction in splanchnic blood flow. In the stomach predispose to stress ulceration and haemorrhage. • In the intestine movement of bacteria and/or bacterial endotoxin from the gut lumen to the portal vein and hence systemic circulation may occur. This is thought to be an important pathophysiological mechanism in the development of SIRS (systemic inflammatory response syndrome) and multiple organ failure in shock.
Hepatobiliary system	• Ischaemic hepatic injury is frequently seen following hypovolaemic or cardiogenic shock system. • Increases in prothrombin time and/or hypoglycaemia are markers of more severe injury. • Significant ischaemic hepatitis is more frequent in patients with underlying cardiac disease and a degree of hepatic venous congestion.

1. MVO (mixed venous oxygen saturation): the amount of O_2 bound to hemoglobins who aren't extracted from tissues and going back to the heart, will be decreased in hypovolemic and cardiogenic shock, and will be normal or increased in distributive (vasogenic) shock.

Treatment of shock:

- Goal is to restore perfusion[1] and reverse the cause (both are priority)
- As with most clinical emergencies, treatment and diagnosis should occur simultaneously with the immediate and management following an Airway, breathing, circulation (ABC) approach.
- Method: Depends on type of shock, detecting the type of shock is a priority also.
- Each component Injury of the CVS will result in a different type of shock which requires a different type of management. So if a patient is bleeding (hypovolemic shock) giving him inotropes won't manage the situation, you should restore the blood. And if a patient is in cardiogenic shock, he should receive inotropes, giving him blood won't do anything.

General principles of management (airway, breathing and circulation):

Airway and breathing

- Hypoxaemia (O_2 saturation < 90%) must be prevented and, if present, rapidly corrected by maintaining a clear airway (e.g head tilt, chin lift) and administering high flow oxygen (e.g. 10-15 liters/min).
- The adequacy of the therapy can be estimated continuously using pulse oximetry, but frequent arterial blood gas analysis allows more accurate assessment of oxygenation (PaO2), ventilation (PaCO2) and indirect measures of tissue perfusion (pH, base excess, HCO3 and lactate levels.
- In patients with severe hypoxaemia, cardiovascular instability, depressed conscious level or exhaustion . Intubation and ventilatory support may be required.

Circulation

- Initial resuscitation should be targeted at arresting haemorrhage.
- Providing fluid (crystalloid or colloid) to restore intravascular volume and optimize cardiac preload, It is common practice to use blood to maintain a haemoglobin concentration > 10 g/dl (with haematocrit around 0.3) during the initial resuscitation of shock if there is evidence of inadequate oxygen delivery, such as a raised lactate concentration or low central venous saturations.

Endpoints of resuscitation in shock management:

01 Normal vital signs (Can be misleading)

For instance, an elderly patient with a usual BP of 170/100 then suddenly his BP drops to 100/60 (which is considered normotensive in healthy individuals). However, in his case it's considered hypotension and the patient is still in shock (because this is half of his usual BP).

02 Normal serum lactate levels)

1. Lactate can be misleading because in some situations of shock, cells are too sick for anaerobic metabolism, so we won't find elevation of lactate.
2. If the lactate was high then you resuscitated the patient successfully, and the levels of lactate returned back to normal this is called "lactate clearance" and it's a reassuring sign.

03 Evidence of adequate tissue perfusion (the best)

- Normal mental status
- ★ Normal urine output (best marker) remember, kidney may get damaged during Shock.
- Normal liver function

1. With fluid, inotropes and vasopressors.
2. ★ If blood pressure remains low and/or signs of inadequate tissue oxygen delivery persist, then inotropes and/or vasopressors may be required. Adrenaline, which has both vasopressor and inotropic effects, is a useful first line drug in the emergency treatment of shock. Dopamine can be used

Treatment of shock:

Hypovolemic shock treatment:

- **ABCs**
  (Airway, Breathing, Circulation) For any sick patient
- **Control any bleeding**
- **Establish 2 large bore IVs[1] or central line**
- **Colloid solution** Contain protein particles that exert oncotic pressure and cause fluid to remain in the intravascular.
- **Crystalloids[2]**
  Normal saline or Lactate Ringers.
  For resuscitation and blood is way better than fluids
- **PRBCs[3]** (Packed RBCs)
  O negative or cross matched
  (Are red blood cells that have been separated for blood transfusion)
- **Arrange definitive treatment**

Evaluation of hypovolemic shock:

- CBC & Electrolytes
- ABG (arterial blood gas/Lactate)
- Kidney function: BUN (blood urea nitrogen/Creatinine)
- Coagulopathy: Coagulation studies
- Types and cross match

As indicated:

- Trauma patient: CXR, Pelvic X-Ray
- Stable patient to detect bleeding source: CT scan
- Incase of hematemesis: GI endoscopy
- Incase of hemoptysis: Bronchoscopy
- Incase of history of AAA: Vascular radiology

IV Resuscitation:

- 2 large bore IV is usually the best option for resuscitation. Why? High flow rate achieved in a short period of time. Large bore IV means using 14 or 16 gauge needles to administer fluid quickly. For instance, with two 14G IV 2L of fluid is administered in 4 minutes.
- The forearms or antecubital fossa are the most accessible peripheral sites, but the nature and location of the injuries may require alternative sites, such as the femoral or external jugular vein.
- Central venous cannulation is difficult and potentially hazardous in shocked hypovolemic patients.

You don't have to memorize the numbers but you have to understand the concept

Flow Rates in IV/IO Access

Gauge	Approximate Flow Rate to Gravity (mL/min)	Time to Infuse 1L (min)
14G	250	4
16G	150	7
Cordis	130	8
18G	100	10
15G Humeral IO	80	13
16G Distal Port Triple Lumen	70	15
15G Tibial IO	70	15
20G	60	17
22G	35	29
18G Prox Port Triple Lumen	30	34

1. 2 large bores are used for hypovolemic shock, blood is used instead incase of hemorrhagic shock.
2. A types of IV fluids containing water and electrolytes.
3. Preferred in hemorrhagic shock, because it prevents coagulopathy.

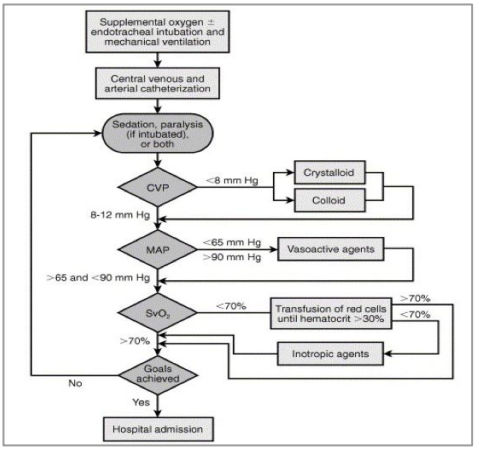
Treatment of shock:

Septic shock treatment:

- 2 large bore IVs, fluid resus[5]. If a patient with adequate blood volume like 5L and he got into a septic shock the blood vessels will be dilated so then he will need 7L of blood that's why we give fluids (note that the heart here is working efficiently), so septic shock can initially resemble hypovolemic shock, you give fluids the patient gets better. We say to the patients with pneumonia or other infections to take fluids to avoid septic shock.
- Supplemental oxygen.
- ★ Broad spectrum IV antibiotics, based on suspected source, as soon as possible.
- Goal directed therapy.

Treatment algorithm:

- Treatment Algorithm: (this is one way to manage sepsis. However, this has changed now)
- What we actually do if we suspect a patient with sepsis:
  1. Blood Culture.
  2. Broad spectrum antibiotic (very important, in every one hour delay of antibiotic administration you increase the mortality chance by 10%) .
  3. Fluid in case of hypovolemia.
  4. If the patient is still hypotensive we give vasopressors.
  5. In the meantime look for the source of the sepsis and control it.



Anaphylactic shock treatment:

Diagnosis

- Clinical Diagnosis [1]
  Defined by airway compromise[2], hypotension, or involvement of cutaneous[3], respiratory, or GI systems
- Look for exposure to drug, food, or insect.
- Labs have no role.

Treatment

- ABC's Airway is a major step
  Angioedema and respiratory compromise require immediate intubation
- IV, cardiac monitor, pulse oximetry.
- IVFs, oxygen.
- **First line:** Epinephrine[4]
- **Second line:**
  - Corticosteroids:
    Hydrocortisone 200 mg IV
  - H1 and H2 blockers:
    Chlorphenamine 10–20 mg slow IV

1. No test will help you, in fact it's just a waste of time.
2. Hallmark of anaphylaxis. (due to laryngeal or pharyngeal edema)
3. E.g rash and urticaria.
4. To achieve severe vasoconstriction and bronchodilation.
5. 1-2L only, not as much as hypovolemic shock.

Treatment of shock:

Cardiogenic shock treatment:

- The commonest cause of cardiogenic shock is acute (anterior) myocardial infarction.
- As with other forms of shock, the management of cardiogenic shock is based upon the identification and treatment of reversible causes and supportive management to maintain adequate tissue oxygen delivery.
- Routine investigations to identify the cause of cardiogenic shock include:
  - 12-lead ECGs.
  - Troponin
  - Creatinine kinase-MB (CK-MB) levels.
  - CXR.
  - Transthoracic[1]

- **General supportive treatment include**:
  1- Administration of high concentrations of inspired oxygenation.
  2- Intra-aortic balloon pump (IABP) (is a mechanical device that increases myocardial oxygen perfusion and Indirectly increases CO through afterload reduction) as an adjunct and as a last resort supportive treatment.
  3- Correction of hypovolaemia and optimization of intravascular volume (preload) through cautious fluid resuscitation (Except for HF patients).

1

Acute heart failure

Usually normovolaemic or relatively.

Hypovolaemic as a result of intravascular fluid loss into the lungs and the development of pulmonary oedema.

2

Chronic heart failure

Usually hypervolemic as a result of long-standing activation of the renin-angiotensin system and salt and water retention.

3

Acute myocardial infarction

IV opiates titrated cautiously to control pain, reduce anxiety and reduce myocardial oxygen demand afterload by causing peripheral vasodilation.

4

Emergency cardiogenic shock

The most appropriate choice of vasoactive drug in cardiogenic shock is one that has both ionotropic and vasodilating properties such as β-agonist dobutamine. Alternative inodilating agents include: calcium sensitizer levosimendan and the PDE inhibitor milrinone

Neurogenic shock treatment:

- IV fluid resuscitation (Only add vasopressors if hypotension refractory to IV fluids).

1. Echocardiogram may provide useful information on (systolic and diastolic) ventricular function and exclude potentially treatable causes of cardiogenic shock such as cardiac tamponade, valvular insufficiency and massive pulmonary embolus

Cases:

What type of shock is this?

Q: 68 yo M with hx of HTN and DM risk factors for intra abdominal bleeding presents to the ER with abrupt onset of diffuse abdominal pain[1] indicates possible bleeding into the abdomen with radiation to his low back. The patient is hypotensive shock. tachycardic, afebrile cannot be anaphylactic or septic with cool but dry skin. Vasoconstriction is functional.

A.Hypovolemic shock
(there is evidence of blood shunt)

Q: A 34 female presents to the ER after dining at a restaurant where shortly after eating the first few bites of her meal[2], became anxious, diaphoretic, began wheezing, noted diffuse pruritic rash, nausea, and a sensation of her “throat closing off”. She is currently hypotensive, tachycardic and ill appearing.

A. Anaphylactic shock
(History is the most important) Note that in all shock types you won’t find bronchospasm unless if it was an anaphylactic shock)

Q: A 73 year old lady with a history of ischemic heart disease, HTN, DM II presents to the ED with altered mental status. She is febrile[3] to 39.4, hypotensive with a widened pulse pressure[4], tachycardic, with warm extremities and decreased urine output.

A. Septic shock

1. Sudden Pain and radiate to the back? GI bleeding caused by ruptured Aneurysm, pancreatitis takes 3 days
2. Anaphylactic within minutes, sepsis within days.
3. Cardiogenic shock patients will not have fever.
4. Wide pulse pressure: high systolic (heart is working fine) + low diastolic (Vasodilation) > Sepsis.

Summary:

	Hypovolemic	Cardiogenic	Neurogenic	Septic	Anaphylactic
Clinical picture	-Hypotension -Tachycardia -Cold,clammy skin	-Hypotension -Tachycardia -Cold, clammy skin	-Hypotension -Bradycardia -Warm, dry skin	-Hypotension -Tachycardia -Warm, dry skin -Febrile	-Hypotension -Tachycardia -Warm, dry skin
Cause/s	Most common: Hemorrhagic: GLOATS(GI hemorrhage, Lung hemorrhage, Obstetric hemorrhage, Aneurysms, Trauma, Major surgical blood loss) Nonhemorrhagic: -Dehydration -Plasms(burns)	Most common: -MI -Cardiac tamponade -Tension pneumothorax -Arrhythmias -Massive PE -Valve dysfunction	Most common: -Spinal cord injury (Thoracic.cervical) -Traumatic brain injury	-Localized infection (mostly from gram +ve bacteria)	-Exposure to allergic agents
Investigation/Diagnosis	-Clinical diagnosis -Blood tests	-ECG -Chest X-ray -Troponin/CK-MB	-	-Clinical diagnosis -Culture	-Clinical diagnosis
1st step management	Arrest the hemorrhage if found	Treat the underlying cause	IV fluid resuscitation	IV broad spectrum antibiotics	-Maintain airway and give 100% O_2 -IV/IM Epinephrine
Further management	1- IV fluid 2- Blood Transfusion 3- Vasopressors	-	Vasopressors in IVF refractory cases	1- IV fluid 2- Vasopressors	1-Chlorpheniramine 2- Hydrocortisone

438's Quiz

Q1:A 76-year-old male is brought to the hospital with persistent diarrhoea and vomiting for the past 4 days. He has been unable to keep his food down and feels very tired. On examination he is very dehydrated. His pulse is 128/min and his BP is 88/52 mmHg

A) Hemorrhagic hypovolemic shock
B) Non-hemorrhagic hypovolemic shock
C) Cardiogenic shock

Q2:An 86-year-old male has been complaining of increasing lower abdominal pain for the past week. On examination he looks very unwell with warm peripheries. He has signs of generalised peritonitis. His pulse is 130/min and his BP 84/50 mmHg.

A) Neurogenic shock
B) Septic shock
C) Hypovolemic shock

Q3: Which of the following statements regarding hypovolaemic shock are true?

A) The venous pressure is low.
B) The vascular resistance is low.
C) The mixed venous saturation is high

Q4: A 50-year-old male who's previously diagnosed with post-traumatic amnesia and vitally stable complaining of pain in all 4 extremities,which type of shock might've develop in his case?

A) Septic shock
B) Hypovolemic shock
C) Neurogenic shock

Q5: A 19-year-old male is brought to the hospital after sustaining an abdominal injury while playing rugby. He is complaining of left upper abdominal pain and has some bruising over the same area. His pulse is 140/min and his BP is 100/82 mmHg.

A) Hemorrhagic hypovolemic shock
B) Non-hemorrhagic hypovolemic shock
C) Septic shock

Q6: A 24-year-old man presents to the ED with 3 stab wounds to the abdomen. He was intubated in the field for airway protection. Blood pressure is 70/30 mm Hg and pulse 140/min. On examination, 3 penetrating wounds covered by abdominal pressure pads are noted. What is the best next step in management?

A) IV fluids
B) Abdominal X-ray
C) Start broad spectrum antibiotics immediately.

Answers

Q1	B	Q4	C
Q2	B	Q5	A
Q3	A	Q6	A

439's Quiz

Q1: A 22-year-old man sustains severe blunt trauma to the back. He notes that he cannot move his lower extremities. He is hypotensive and bradycardic. Which of the following is the best initial management of the patient?

A) Administration of phenylephrine
B) Administration of dopamine
C) Intravenous fluid bolus

Q2: 15-year-old s/p trampoline injury with tetraplegia (a.k.a. "quadriplegia") with hypotension. No other injuries on CT scans. What type of shock is this?

A) Neurogenic shock.
B) Cardiogenic Shock.
C) Hypovolemic shock.

Q3: A 35-year-old man is admitted after severing his arm on industrial machinery. His airway is patent and there is no identifiable hindrance to breathing. His pulse is 110 beats/min, blood pressure is 130/105 mmHg and respiratory rate is 25 breaths/min. Which stage of shock is this patient in?

A) Class I
B) Class II
C) Class III

Q4: A 7-year-old boy with nut allergy develops stridor and collapses after eating a snack. He requires airway and breathing support. His BP is 60/38 mmHg. What type of shock is this?

A) Neurogenic shock.
B) Hypovolemic shock.
C) Anaphylactic shock.

Q5: Which of the following statements regarding cardiogenic shock is true?

A) Vascular resistance is low.
B) Cardiac output is low.
C) Venous resistance is low.

Q6: Which of the following regarding blood pressure in shock are false?

A) Elderly patients who are normally hypertensive may present with a 'normal' blood pressure.
B) Children and fit young adults are able to maintain blood pressure until the final stages of shock.
C) Hypotension is one of the first signs of shock.

Answers

Q1	C	Q4	C
Q2	A	Q5	B
Q3	B	Q6	C

Extra Questions

Good Luck!

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Feedback