Shock and haemorrhage

- **Definition:** Shock is a physiologic state characterized by systemic reduction in tissue perfusion, resulting in decreased tissue oxygen delivery.

According to Bailey and Love:

Classification of shock:

- Hypovolaemic shock
- Cardiogenic shock
- Obstructive shock
- Distributive shock
- Endocrine shock

- **Cardiogenic** : Shock caused as a result of cardiac pump failure

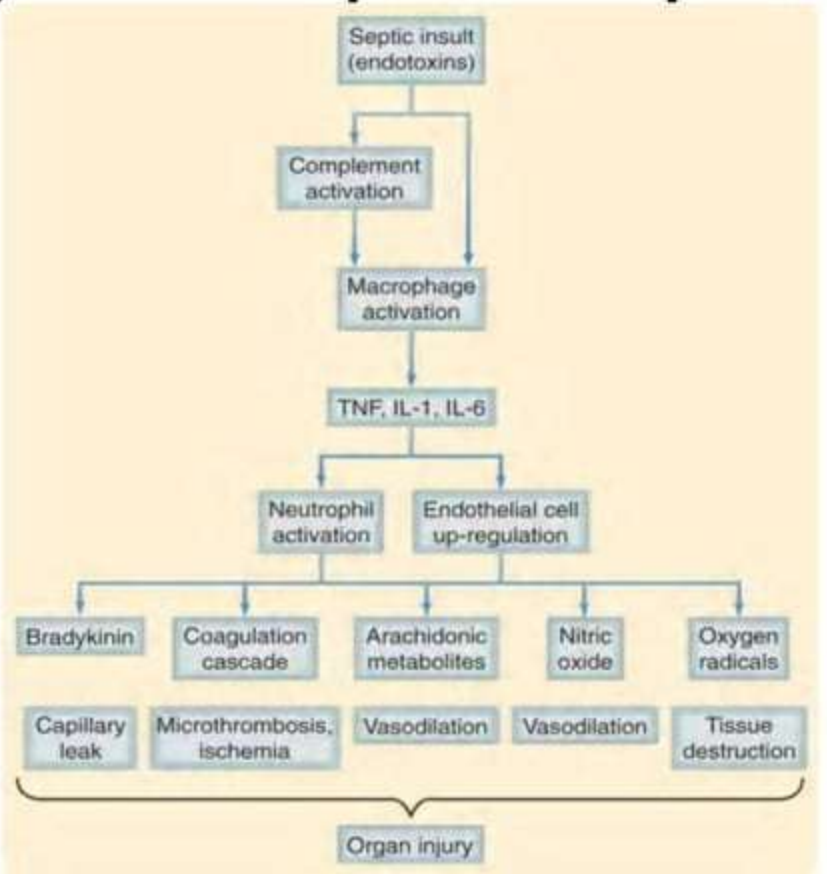
Causes:

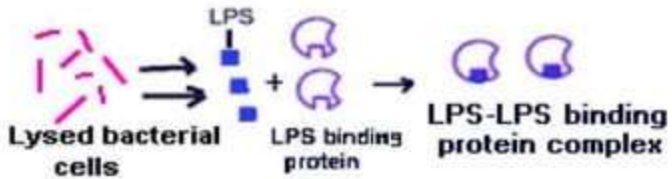
- Myocardial Infarction
- Arrythmias (Atrial fibrillation, ventricular tachycardias, bradycardias, etc)
- Mechanical abnormalities (valvular defects)
- Extracardiac abnormalities (PE, pulm HTN, tension pneumothorax)

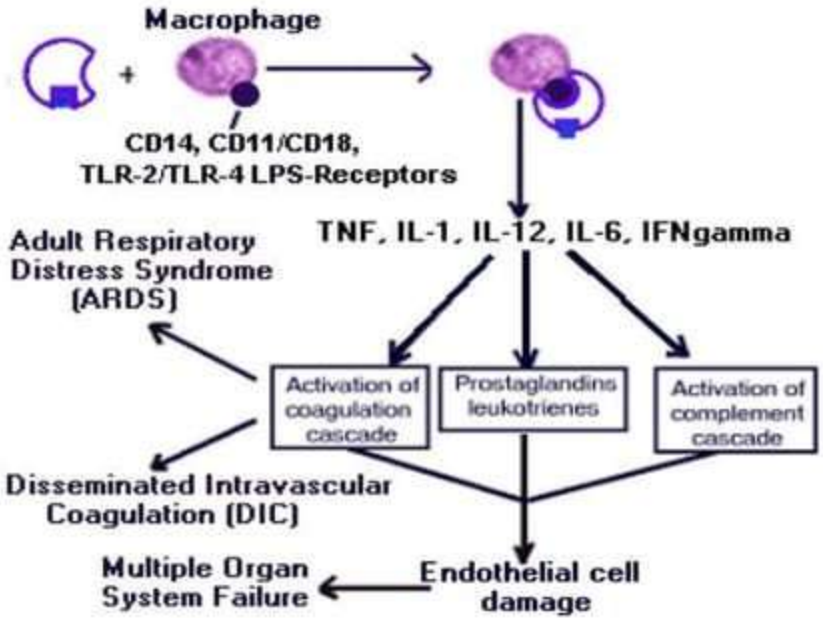
- **Hypovolemic**: Shock caused by decreased preload due to intravascular volume loss. Causes:
- Hemorrhagic – trauma, GI bleed, hemorrhagic pancreatitis, fractures
- Fluid loss induced – Diarrhea, vomiting, burns

- **Distributive**: Shock as a result of severely diminished systemic vascular resistance.
  - Septic: secondary to an overwhelming infection
  - Anaphylactic: secondary to an overwhelming infection
  - Neurogenic: secondary to a sudden loss of the autonomic nervous system function.

Pathogenesis of sepsis and septic shock




LPS
Lysed bacterial cells
LPS binding protein
LPS-LPS binding protein complex


Macrophage
CD14, CD11/CD18, TLR-2/TLR-4 LPS-Receptors
TNF, IL-1, IL-12, IL-6, IFNgamma
Adult Respiratory Distress Syndrome (ARDS)
Activation of coagulation cascade
Prostaglandins leukotrienes
Activation of complement cascade
Disseminated Intravascular Coagulation (DIC)
Multiple Organ System Failure
Endothelial cell damage

Pathophysiology of septic shock:

General Clinical Signs:

- Flu-like symptoms – fever, chills – general malaise, irritability, lethargy
- Tachycardia and hypotension
- Hyperventilation
- Site of infection may or may not be evident

Cardiovascular:

- Systemic vasodilation and hypotension (Psys < 90 mmHg)
- Tachycardia (>100 beats/min)
- Increased cardiac output (hyperdynamic), although contractility is depressed; hypodynamic in late shock
- Ventricular dilation; decreased ejection fraction
- Loss of sympathetic responsiveness
- Hypovolemia due to vascular leakage; central venous pressure may be decreased or increased depending upon fluid resuscitation
- Compromised nutrient blood flow to organs; decreased organ oxygen extraction

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Pulmonary and renal:

- Hyperventilation with respiratory alkalosis
- Pulmonary hypertension and edema
- Hypoxemia (arterial $pO_2 < 50$ mmHg)
- Reduced pulmonary compliance; increased work
- Respiratory muscle failure
- Renal hypoperfusion; oliguria
- Acute tubular necrosis and renal failure

Other

- Disseminated intravascular coagulation (DIC)
- Blood dyscrasias:
  - Leukopenia
  - Thrombocytopenia
  - Polycythemia
- Central and peripheral nervous dysfunction
- Increased lactate occurs early

Management

Diagnosis:

- Before the initiation of antimicrobial therapy, at least two blood cultures should be obtained
- At least one drawn percutaneously.
- At least one drawn through each vascular access device if inserted longer than 48 hours
- Other cultures such as urine, cerebrospinal fluid, wounds, respiratory secretions or other body fluids should be obtained as the clinical situation dictates
- Other diagnostic studies such as imaging and sampling should be performed promptly to determine the source and causative organism of the infection may be limited by patient stability.

Treatment primarily consists of:

- Volume resuscitation
- Early antibiotic administration
- Early goal directed therapy
- Rapid source identification and control.
- Support of major organ dysfunction.
- Sequestration of lipopolysaccharides.

- Antibiotics (early administration)
- Hemodynamic support – (fluid resuscitation)
- Restore tissue perfusion
- Normalize cellular metabolism
- Tight glycemic control
- Vasopressor agents Dopamine, norepinephrine, dobutamine

- Source control – Surgical debridement of infected, devitalized tissue – Catheter replacement
- Supplemental oxygen (treatment of acute respiratory distress syndrome,ARDS)
- Nutritional support
- Anti-inflammatory agents :
  - Cortocosteroids
  - Ibuprofen,
  - Prostaglandin E1
  - Pentoxifylline
- Oxygen Scavengers :N-acetylcysteine , selenium
- Drugs modifying coagulation – Anti-thrombin III

- Drugs enhancing host defenses
  - Intravenous immunoglobulin (IVIG)
  - Interferon-gamma
  - GM-CSF
  - Immunonutrition
- Other drugs
- Growth hormone, antibiotics, fresh frozen plasma, anesthetic sedative and analgesic agents, catecholamines

- Hemofiltration, plasma filtration, plasma exchange
- Experimental therapies
    - Anti-endotoxin therapies – IVIG, BPI protein
    - IL-1Ra
    - Anti-TNF-alpha, soluble TNFR
    - PLA2 inhibitors, PAF inhibitors
    - iNOS inhibitors
    - Anti-coagulants (APC)

MODS

- Multiple organ dysfunction syndrome (MODS), previously known asmultiple organ
- failure (MOF) or multisystem organ failure (MSOF), is altered organ function in an acutely ill patient requiring medicalintervention to achieve homeostasis. Multiple organ failure is defined as two or more failed organ systems.

- **Cause:** The condition usually results from infection, injury (accident, surgery), hypoperfusion and hypermetabolism. The primary cause triggers an uncontrolled inflammatory response.
- Sepsis is the most common cause in operative and non-operative patients. Sepsis may result in septic shock. In the absence of infection, a sepsis-like disorder is termed as systemic inflammatory response syndrome (SIRS).
- Both SIRS and sepsis could ultimately progress to multiple organ dysfunction syndrome.
- However, in one-third of the patients no primary focus can be found Multiple organ dysfunction syndrome is well established as the final stage of a continuum: SIRS + infection leads to sepsis can turn to severe sepsis can lead to Multiple organ dysfunction syndrome.

Effects of organ failure:

- Lung- Acute respiratory distress syndrome
- Kidney- Acute renal insufficiency
- Liver- Acute liver insufficiency
- Clotting- Coagulopathy
- Cardiac - Cardiovascular failure.

SIRS

Criteria for Four Categories of the Systemic Inflammatory Response Syndrome

Systemic Inflammatory Response Syndrome (SIRS)

- **Two or more of the following:**
- Temperature (core) >38°C or <36°C
- Heart rate >90 beats/min
- Respiratory rate of >20 breaths/min for patients spontaneously ventilating or a $Paco_2$ <32 mm Hg
- White blood cell count >12,000 cells/mm^3 or <4000 cells/mm^3 or >10% immature (band) cells in the peripheral blood smear

- **Sepsis : Same criteria as for SIRS but with a clearly established focus of infection**
- **Severe Sepsis: Sepsis associated with organ dysfunction and hypoperfusion.**

Indicators of hypoperfusion:

- Systolic blood pressure <90 mm Hg
- >40 mm Hg fall from normal systolic blood pressure
- Lacticacidemia
- Oliguria
- Acute mental status changes

Septic Shock

- **Patients with severe sepsis who**
- Are not responsive to intravenous fluid infusion for resuscitation.
- Require inotropic or vasopressor agents to maintain systolic blood pressure

HAEMORRHAGE

- **Types of haemorrgage:**
- Recognition of types of haemorrhage:
- Arterial/ venous/capillary:
- Arterial haemorrhage: Arterial haemorrhage is recognised as bright red blood, spurting as a jet which rises and falls in time with the pulse. In protracted bleeding, and when quantities of intravenous fluids other than blood are given, it can become watery in appearance.
- Venous haemorrhage: Venous haemorrhage is a darker red, a steady and copious flow. The colour darkens still further from excessive oxygen desaturation when blood loss is severe, or in respiratory depression or obstruction. Blood loss is particularly rapid when large veins are opened, e.g. common femoral or jugular. Venous bleeding can be under increased pressure as in asphyxia, or from ruptured varicose veins
- Capillary haemorrhage: Capillary haemorrhage is bright red, often rapid, ooze. If continuing for many hours, blood loss can become serious, as in haemophilia.

Primary/reactionary/secondary:

- Primary haemorrhage: Primary haemorrhage occurs at the time of injury or operation. Reactionary haemorrhage
- Reactionary haemorrhage: may follow primary haemorrhage within 24 hours (usually 4—6 hours) and is mainly due to rolling ('slipping') of a ligature, dislodgement of a clot or cessation of reflex vasospasm.
- Secondary haemorrhage: Secondary haemorrhage occurs after 7—14 days, and is due to infection and sloughing of part of the wall of an artery. Predisposing factors are pressure of a drainage tube, a fragment of bone, a ligature in an infected area or cancer. It is also a complication of arterial surgery and amputations.

External/internal:

- External haemorrhage: External haemorrhage is visible, revealed haemorrhage.
- Internal haemorrhage: Internal haemorrhage is invisible, concealed haemorrhage. Internal bleeding may be concealed as in ruptured spleen or liver, fractured femur, ruptured ectopic gestation or in cerebral haemorrhage. Concealed haemorrhage may become revealed as in haematemesis or melaena from a bleeding peptic ulcer, as in haematuria from a ruptured kidney, or via the vagina in accidental uterine haemorrhage of pregnancy.

Classes of haemorrhage:

	CLASS 1	CLASS 2	CLASS 3	CLASS 4
Blood loss(ml)	Up to 750	750-1500	1500-2000	>2000
Blood loss(%	Up to 15%	15%-30%	30%-40%	>40%
Pulse rate	<100	100-120	120-140	>140
Blood pressure	Normal	Normal	Decreased	Decreased
Pulse pressure (mmHg)	Normal or increased	Decreased	Decreased	Decreased
Respiratory rate	14-20	20-30	30-40	>35
Urine output (ml/hr)	>30	20-30	5-15	Negligible
CNS/mental status	Slightly anxious	Mildly anxious	Anxious, confused	Confused, lethargic
Fluid replacement	Crystalloid	Crystalloid	Crystalloid and blood	Crystalloid and blood

- **Methods of determining acute blood loss:**
- Assessment and management of blood loss must be related to the pre-existing circulating blood volume, which can be derived from the patient's weight:
- • Infant 80—85 ml/kg;
- • Adult 65—75 ml/kg.
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- **Measuring blood loss:**
- • **Blood clot:** The size of a clenched fist is roughly equal to 500 ml.
- • **Swelling in closed fractures:** Moderate swelling in closed fracture of the tibia equals 500—1500 ml blood loss. Moderate swelling in a fractured shaft of femur equals 500—2000 ml blood loss.

- **Swab weighing.** In the operating theatre, blood loss can be measured by weighing the swabs after use and subtracting the dry weight. The resulting total obtained (1 g = 1 ml) is added to the volume of blood collected in the suction or drainage bottles. In extensive wounds and operations, the blood loss is grossly underestimated, due to evaporation of water from the swabs before weighing each batch.Prompt transfer of discarded swabs into polythene bags reduces this source of error. Blood, plasma and water are also lost from the vascular system because of evaporation from open wounds, into the tissues, sweating and expired water via the lungs. Indeed, for operations such as radical mastectomy or partial gastrectomy it may be necessary to multiply the swab weighing total by a factor of 1.5. For prolonged surgery via larger wounds, as in abdominothoracic or abdominoperineal operations, the total measured may need to be multiplied by 2.

- **Haemoglobin level**
- This is estimated in g/100 ml (g/dl), normal values being 12—16 g/100 ml (12—16 g/dl). There is no immediate change in haemorrhage, but after some hours the level falls by influx of interstitial fluid info the vascular compartment in order to restore the blood volume.
- **Measurement of central venous pressure**
- **Continuous tissue oxygen tension measurement**

Managing internal bleeding:

- ABC's
- High concentration oxygen
- Assist ventilations
- Control external bleeding
- Stabilize fractures
- RICE – resuscitation, investigations, clinical examination, evaluation
- Transport rapidly to appropriate facility.

Control of external bleeding:

- Pressure Dressing: Use bandage to secure dressing in place
- Tourniquets:
- Final resort when all else fails
- Used for amputations
- 3-4" wide (blood pressure cuffs)
- Write "TK" and time of application on forehead of patient
- Notify other personnel
- Once applied, DO NOT REMOVE

Treatment of haemorrgagic shock:

- The primary treatment of hemorrhagic shock is to control the source of bleeding as soon as possible and to replace fluid.
- In controlled hemorrhagic shock (CHS), where the source of bleeding has been occluded, fluid replacement is aimed toward normalization of hemodynamic parameters.
- In uncontrolled hemorrhagic shock (UCHS), in which the bleeding has temporarily stopped because of hypotension, vasoconstriction, and clot formation, fluid treatment is aimed at restoration of radial pulse or restoration of sensorium or obtaining a blood pressure of 80 mm Hg by aliquots of 250 mL of lactated Ringer's solution (hypotensive resuscitation).

- Crystalloid is the first fluid of choice for resuscitation. Immediately administer 2 L of isotonic sodium chloride solution or lactated Ringer's solution in response to shock from blood loss.
- Fluid administration should continue until the patient's hemodynamics become stabilized. Because crystalloids quickly leak from the vascular space, each liter of fluid expands the blood volume by 20-30%; therefore, 3 L of fluid need to be administered to raise the intravascular volume by 1 L.

- Alternatively, colloids restore volume in a 1:1 ratio. Currently available colloids include human albumin, hydroxy-ethyl starch products (mixed in either 0.9% isotonic sodium chloride solution or lactated Ringer's solution), or hypertonic saline-dextran combinations. The sole product that is avoided routinely in large-volume (>1500 mL/d) restoration is the hydroxy-ethyl starch product mixed in 0.9% isotonic sodium chloride solution because it has been associated with the induction of coagulopathy.

- In patients with hemorrhagic shock, hypertonic saline has the theoretical benefit of increasing intravascular volume with only small amounts of fluid. The combination of dextran and hypertonic saline may be beneficial in situations where infusion of large volumes of fluid may be harmful, such as in elderly persons with impaired cardiac activity.
- PRBCs should be transfused if the patient remains unstable after 2000 mL of crystalloid resuscitation. For acute situations, O-negative noncrossmatched blood should be administered. Administer 2 U rapidly, and note the response. For patients with active bleeding, several units of blood may be necessary.

- There are recognized risks associated with the transfusion of large quantities of PRBCs. As a result, other modalities are being investigated. One such modality is hemoglobin-based oxygen carriers (HBOC).

- If at all possible, blood and crystalloid infusions should be delivered through a fluid warmer.
- A blood sample for type and cross should be drawn, preferably before blood transfusions are begun.
- Start type-specific blood when available.
- Patients who require large amounts of transfusion inevitably will become coagulopathic.
- FFP generally is infused when the patient shows signs of coagulopathy, usually after 6-8 U of PRBCs.
- Platelets become depleted with large blood transfusions.
- Platelet transfusion is also recommended when a coagulopathy develops.