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SHOCK

Define shock.

Shock is:

- Not only low blood pressure (many normal patients have systolic BP less than 100 mmHg)
- Not only decreased peripheral perfusion (ischaemic limb has decreased peripheral perfusion of the lower limbs/not necessarily in shock state)
- Not only limited systemic oxygen delivery (severe anaemia also has decreased oxygen delivery)

Shock is a systemic state of low tissue perfusion (of the whole body) which is inadequate for normal cellular respiration. With insufficient delivery of oxygen and glucose, cells switch from aerobic to anaerobic metabolism. If perfusion is not restored in timely fashion, cell death occur.

1. **What are the different (5) types?**
   - Hypovolemic
   - Cardiogenic
   - Obstructive
   - Distributive
   - Endocrine

2. **What are the signs of shock?**
   - Pale, diaphoretic (heavy sweating), cool skin ⇒ clammy skin
   - Hypotension, tachycardia, tachypnea
   - Decrease mental status
   - Decreased pulse pressure
   - Poor capillary refill

3. **What clammy skin means?**
   - Clammy skin is a combination of **cold pale** (peripheral vasoconstriction) skin and **sweating**. Both due to sympathetic stimulation

4. **What are the best indicators of tissue perfusion?**
   - Urine output, mental status

5. **What lab tests help assess tissue perfusion?**
   - Lactic acid (elevated with inadequate tissue perfusion), base deficit, decrease pH (by ABG analysis, acidosis associated with inadequate tissue perfusion)

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HYPOVOLEMIC SHOCK

6. **What is the definition?**
    - Decreased intravascular volume
7. **What are the common causes?**
    - **Haemorrhagic**
        - Traumatic (e.g., RTA, stab wound, bullet injury, etc.…)
        - Non traumatic (e.g. abruptio placentae, ruptured ectopic pregnancy, ruptured AAA…)
    - **Non haemorrhagic :examples**
    - Burns
    - Bowel obstruction
    - Pancreatitis (excessive extravasation of intravascular fluid due to excessive vasodilatation causing third space loss)
    - Excessive fluid loss due to vomiting, diarrhoea, urinary loss as in diabetes, evaporation and third space loss.
8. **What is the third space? Give examples**
    - It is the space in which fluid achieves no functions
    - **Examples:-**
        - Ascetic fluid
        - Pleural effusion
        - Oedema (as in congestive heart failure)
        - Fluid accumulated in the bowel wall for any cause
9. **What are the signs of hypovolaemic shock?**
    - **Early**—Orthostatic hypotension, mild tachycardia, anxiety, diaphoresis (excessive sweating), vasoconstriction (decreased pulse pressure with increased diastolic pressure), and empty veins.
    - **Late**—Changed mental status, decreased BP, marked tachycardia
10. **What are the symptoms?**
    - Restlessness
    - Air hunger
    - Excessive thirst
    - Tinnitus and blurred vision are late features
11. **What are the specific changes in the vital organs in shock?**
    - kidney (acute tubular necrosis)
    - Brain (cerebral ischaemia)
    - Gut (non-occlusive infarction)
    - Adrenal (no normal reaction to stress)
    - Heart (cardiac failure)
    - Lungs (ARDS)

12. What non-occlusive infarction of the gut means?

- It means that the infarction is due to very low perfusion of the gut (not due to occlusion of the mesenteric vessels)

13. What are the 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

14. What is the treatment?

- Stop the bleeding (in traumatic cases)
- Volume: IVF (isotonic Ringer lactate) then blood products as needed (in haemorrhagic shock)
- Correction of fluid deficit in non-haemorrhagic cases
- Avoid excessive warmth because it cause vasodilatation and further decrease in the blood pressure

15. 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

16. What usually causes failure of resuscitation in haemorrhagic cases?

- Persistent massive hemorrhage requiring emergent surgical procedure for stopping the bleeding (surgery is part of resuscitation in some cases)

17. **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
18. **What is the most common vital sign change associated with early hypovolemic shock?**
    - Tachycardia

19. **What type of patient does not mount a normal tachycardia response to hypovolemic shock?**
    - Patients on β-blockers,
    - Spinal shock (loss of sympathetic tone),
    - Athletes and
    - Those with autonomic neuropathy as patients with DM.

20. **Should vasopressors be used to treat hypovolemic shock?**
    - No, concentrate on refilling the circulation i.e. giving blood or fluid or both.
    - Vasopressor used in types of shock in which there is loss of sympathetic tone as in spinal shock, or in shock states due to massive vasodilatation as in anaphylactic shock (i.e. cases of severe decrease in the peripheral vascular resistance).

21. **Should patients with hypovolemic shock be put into the Trendelenburg position? See below**
    - No
22. **What is Trendelenburg position**?

    A position of the body for medical examination or operation in which the patient is placed head down on a table inclined at about 45 degrees from the floor with the knees uppermost and the legs hanging over the end of the table.
23. **What is the reverse Trendelenburg position?**

    This is the exact opposite traditional Trendelenburg position and is also named the "anti-trendelenburg". The head is elevated higher 15 to 30 degrees in the air. The reverse trendelenburg position is used in thyroid surgery and exposing the upper abdominal region during surgery.

24. **What is the shock position?**

    The shock position is the position of a person who is lying flat on his/her back with the legs elevated approximately 8-12 inches. This is used when a patient is showing signs of shock. The shock position is also used for patients experiencing heat related emergencies and in those with vasovagal fainting.

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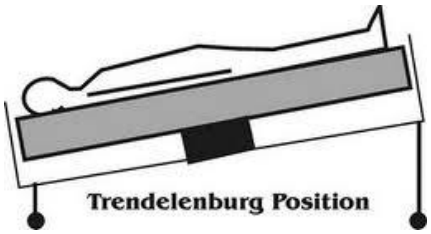

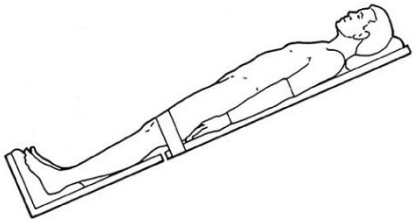

Trendelenburg position **Reverse Trendelenburg**

SEPTIC SHOCK

25. What is SIRS (systemic inflammatory response syndrome)?

- is a systemic inflammatory response characterized by the presence of two or more of the following:
  - Hyperthermia >38°C or hypothermia <36°C
  - Tachycardia >90 bpm
  - Tachypnoea >20 r.p.m. or PaCO2 <4.3 kPa
  - Neutrophilia >12 × 10^9 /L or neutropenia <4 × 10^9 /L.

26. What is sepsis syndrome?

- It is SIRS with documented infection

27. What is septic shock?

- Septic shock is sepsis with hypotension.

28. What is the specific etiology?

- Most common—gram-negative septicemia
- Less common—gram-positive septicemia, fungus

29. What factors increase the susceptibility to septic shock?

- Any mechanism that increases susceptibility to infection (e.g., trauma, immunosuppression, corticosteroids, hematologic disease, diabetes)

30. What complications are major risks in septic shock?

- Multiple organ failure, DIC, death

31. What happens in DIC

- Disseminated intravascular coagulation is a condition in which small blood clots develop throughout the bloodstream, blocking small blood vessels. The increased clotting depletes the platelets and clotting factors needed to control bleeding, causing excessive bleeding.

32. What are the signs/ symptoms of septic shock?

- **Initial—**vasodilation, resulting in warm skin and full pulses; normal urine output
- **Delayed—**vasoconstriction and poor urine output; mental status changes; hypotension

33. What percentage of blood cultures is positive in patients with bacterial septic shock?

- Only about 50%!

34. What are the associated findings?

- Fever, hyperventilation, tachycardia

35. What are the associated lab findings?

- **Early—**hyperglycemia/glycosuria, respiratory alkalosis (due to CO2 wash out resulting from tachypnoea), haemoconcentration, leukopenia
- **Late—**leukocytosis, metabolic acidosis due to elevated lactic acid

(*Note:* Identifying organism is important to direct treatment/antibiotics)

36. What is the treatment?

i. Volume (IVF)
ii. Antibiotics (empiric, then according to the culture results)
iii. Drainage of abscess
iv. Zygris® PRN- Activated protein C, shown to decrease mortality in septic shock and multiple organ failure

CARDIOGENIC SHOCK

37. What is the definition?

- Cardiac insufficiency; usually left ventricular failure, resulting in inadequate tissue perfusion; in other words the heart failed to pump enough blood to the circulation.

38. What are the causes?

- MI, papillary muscle dysfunction, massive cardiac contusion, cardiac tamponade, tension pneumothorax, cardiac valve failure and malignant arrhythmia as ventricular tachycardia.

Remember that cardiac tamponade, tension pneumothorax and pulmonary embolism can cause obstructive shock.

39. What are the signs/symptoms on exam?

- Dyspnea (usually orthopnea)
- Rales (crepitations, mainly basal), Pulsus alternans (increased pulse volume with greater filling following a weak pulse)
- Loud pulmonic component of S2
- Gallop rhythm

40. What are the associated vital signs/parameters?

- Hypotension, decreased cardiac output, elevated CVP/wedge pressure, decreased urine output (low renal blood flow), tachycardia (possibly)

41. Could cardiogenic shock associated with bradycardia?

- Yes, as in patient with MI developing heart block or in diabetic patient with severe autonomic neuropathy.

42. What are the signs on CXR?

- Pulmonary edema ,may be cardiomegaly

43. What is the treatment?

- Based on diagnosis/mechanism:
    - **Congestive heart failure:** diuretics and afterload reduction (e.g., ACE inhibitors), with or without pressors
    - **Left ventricular failure (MI):** pressors, afterload reduction
    - **Arrhythmias:** antiarrhythmic drugs

44. What are the last resort support mechanisms?

- Intra-aortic balloon pump (IABP), ventricular assist device (VAD)

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NEUROGENIC SHOCK and SPINAL SHOCK

Notes:

- Neurogenic shock could be part of spinal shock (loss of sympathetic tone)
- Neurogenic shock could be due to parasympathetic overstimulation
- In spinal shock there is characteristic loss of lower reflexes
- SCI (spinal cord injury) causes disruption of descendent pathways from central control centers in the brain to the spinal sympathetic neurons, originating into intermediolateral nuclei of T1–L2 spinal cord segments. Loss of supraspinal control over sympathetic nervous system results in reduced overall sympathetic activity below the level of injury and unopposed parasympathetic outflow through intact vagal nerve.

45. What is the definition?

- Inadequate tissue perfusion resulting from loss of sympathetic vasoconstrictive tone

46. What are the common causes?

- Spinal cord injury: (spinal shock)
- Total spinal anesthesia (spinal shock)
- Trauma to painful area as the epigastrium, the genitalia and the loins (neurogenic shock)

47. Does all patients with spinal anaesthesia develop spinal shock?

- No; it develop when the level of injecting the anaesthetic medication higher than L2 (especially thoracic and more specifically above T6)

48. What are the signs/ symptoms?

- **Hypotension** and **bradycardia**,
- Neurologic deficit as loss of reflexes (in spinal shock in which there is spinal cord injury or by high spinal anaesthesia)

49. Why is the heart rate and BP decreased?

- Because of loss of sympathetic tone (but hypovolemia [e.g., hemoperitoneum] must be ruled out in trauma cases)

50. Why bradycardia in spinal shock?

Because of the action of the unopposed parasympathetic (Vagus) which cause bradycardia

51. Does all patients with spinal shock develop bradycardia?

- No; only if the spinal cord injury involves areas above (Proximal) vertebrae T-6. Below T-6 the heart rate will not be affected, but blood will accumulate in the lower extremities causing warm, dry, and flushed skin.

52. What is the usual cause of pure neurogenic sock?

- **Severe pain**

53. In which mechanism severe pain cause neurogenic shock?

- Extreme pain causes neurogenic shock by overexciting the parasympathetic nervous system. This results in a significant decrease in heart rate (Bradycardia); which in turn leads to a dangerous drop in blood pressure.

54. What a pathognomonic finding in spinal shock in contrast to neurogenic shock?

- Neurologic deficits (loss of reflexes)

55. What MUST be ruled out in any patient where spinal shock is suspected?

- Hemorrhagic shock**!**

56. What is the treatment?

- **IV fluids** (vasopressors reserved for hypotension refractory to fluid resuscitation i.e concentrate on circulatory filling)

57. What percentage of patients with hypotension and spinal neurologic deficits have hypotension of purely neurogenic origin?

- About 67% (two thirds) of patients

58. What spinal shock usually means in the text books?

- Complete flaccid paralysis immediately following spinal cord injury; may or may not be associated with circulatory shock.
- What you conclude from this? The term shock in spinal shock do not necessarily fit the definition of shock (tissue Hypoperfusion) ‼
- Injuries above the level of T6 usually cause circulatory shock.

59. What is the lowest reflex available to the examiner in patient with spinal shock?

- **Bulbocavernous reflex:** checking for contraction of the anal sphincter upon compression of the glans penis or clitoris

60. What is the lowest level voluntary muscle?

- External anal sphincter

61. What are the classic findings associated with spinal shock?

- Hypotension
- Bradycardia or lack of compensatory tachycardia

62. What vasovagal shock means?

- It means fainting because the body overreacts to certain triggers as:-
    - Prolonged standing
    - Emotional stress
    - Pain
    - The sight of blood

63. What is the pathophysiology of vasovagal shock?

- Regardless of the trigger, the nucleus tractus solitarii of the brainstem is activated directly or indirectly by the triggering stimulus, resulting in simultaneous enhancement of parasympathetic nervous system (vagal) tone and withdrawal of sympathetic nervous system tone.

- This results in a spectrum of hemodynamic responses:

    i. On one end of the spectrum is the cardioinhibitory response, characterized by a drop in heart rate (negative chronotropic effect) and in contractility (negative inotropic effect) leading to a decrease in cardiac output that is significant enough to result in a loss of consciousness. It is thought that this response results primarily from enhancement in parasympathetic tone.

    ii. On the other end of the spectrum is the vasodepressor response, caused by a drop in blood pressure (to as low as 80/20) without much change in heart rate. This phenomenon occurs due to dilation of the blood vessels, probably as a result of withdrawal of sympathetic nervous system tone.

    iii. The majority of people with vasovagal syncope have a mixed response somewhere between these two ends of the spectrum.

Miscellaneous questions

64. What is the acronym for treatment options for anaphylactic shock?

“BASE”:

- **B**enadryl (diphenhydramine) is an antihistamine
- **A**minophylline
- **S**teroids
- **E**pinephrine (act as a vasoconstrictor and bronchodilator)

65. Is classifying shock of benefit in the treatment?

Yes and very yes!

- Hypovolemic shock mandates volume resuscitation.
- Cardiogenic shock mandates cardiac stimulation (pharmacologic and eventually mechanical).
- Peripheral vascular collapse shock mandates pharmacologic manipulation of the peripheral vascular tone (and direct attention to the cause of the vasodilation-typically sepsis).

66. What is the preferred access route for volume infusion?

Flow depends on catheter length and radius. Volume may be infused at twice the rate through a 5-cm (length), 14-gauge (diameter) peripheral catheter as through a 20-cm, 16-gauge central line. Assessment of central venous pressure (and left-sided filling pressure) is necessary if the patient fails to respond to initial volume resuscitation. The preferred peripheral veins are the antecubital veins (cephalic and basilic)

Notes:

- The flow of fluid through catheter is inversely related to the length and the square radius of the catheter (Poiseuille’s law)
- Poiseuille’s law states that the **flow (Q)** of fluid is related to a number of factors: the **viscosity (n)** of the fluid, the **pressure gradient** across the tubing **(P)**, and the **length (L)** and **diameter(r)** of the tubing.
- Central venous pressure reflect the right sided filling pressure

67. What is central venous pressure means?

- It is the blood pressure in the venae cava, near the right atrium of the heart. CVP reflects the amount of blood returning to the heart.
- CVP in response to infusions of intravenous fluid have been used to predict volume-responsiveness (i.e. whether more fluid will improve cardiac output)
- Central venous pressure is used to assess adequacy of blood volume. A long catheter is advanced via the jugular vein into the superior vena cava. The pressure is measured by venous manometers, fluid-filled tubing, and a ruler or an electrical pressure transducer. The zero reference level is the right atrium, which is approximated by the sternal manubrium. Central venous pressure is usually reported as cm H_2O (1 cm H_2O = 0.736 mmHg).

68. What is the normal value of central venous pressure?

- Normal central venous pressure ranges from 0 to 5 cm H2O. Pressures above 12 cm H2O might indicate hypervolemia or cardiac failure.

69. What are the factors that increase the central venous pressure?

Factors Increasing Central Venous Pressure	Primarily a change in compliance (C) or volume (V)
Decreased cardiac output	V
Increased blood volume	V
Venous constriction	C
Changing from standing to supine body posture	V
Arterial dilation	V
Forced expiration (e.g., Valsalva)	C
Muscle contraction (abdominal and limb)	V, C

70. Should one infuse crystalloid, colloid, or blood?

- If the goal is only to improve preload and to repair cardiac output and blood pressure, crystalloid solution should be sufficient.
- If the goal is to augment systemic oxygen delivery, red blood cells bind much more oxygen than plasma (i.e. give blood).
- Crystalloid should enhance flow, and blood should augment oxygen delivery.

71. What are the reasons that limit the use of colloids?

- First, cannot give more than one liter
- Second, it interferes with blood cross matching.

72. When cardiac preload is adequate but still the pumping heart weak, which inotropic agents are useful?

- Dobutamine, epinephrine, and norepinephrine are the chocolate, vanilla, and strawberry of the 32 flavors of cardiogenic drugs. These three drugs are all that the surgeon really needs.

73. Is dopamine the same as dobutamine?

- **No.** Dopamine stimulates renal dopaminergic receptors and may be useful in low doses (2-10 mcg/kg/min has β1 effect) to counteract the renal arteriolar vasoconstriction that accompanies shock. Dopamine at doses higher than 10 mcg/kg/min has alpha effect.

74. Dopamine or dobutamine?

- **The general role is as follow:**
  - If very low blood pressure despite well filled circulation, then dopamine is preferred (at doses higher than 10mcg/kg/min), getting benefit of the vasoconstrictor alpha effect of dopamine
  - If the blood pressure is not very low and the preload is corrected but still the heart is failed, then dobutamine is preferred, getting benefit of the β1 agonist effect which act as a cardiac inotrope.

75. Discuss the use of dobutamine, epinephrine, and norepinephrine.

- **Dobutamine: β1** agonist (cardiac inotrope) with mild-to-moderate **β2** effects (peripheral vasodilation).
- **Epinephrine:** combined **β-** and **α-**adrenergic agent, with the β effects predominating at lower doses and progressive vasoconstriction accompanying increased doses.
- **Norepinephrine:** combined **β-** and **α-**adrenergic agonist, with the alpha effects predominating at all doses.

76. When is an IABP (INTRA-AORTIC BALLOON PUMP) indicated?

- Mechanical circulatory support is indicated when the preload to both ventricles (CVP and PCWP) has been optimized and further cardiac stimulatory drugs are limited by frightening runs of premature ventricular contractions. Do not be afraid to resort to mechanical support.

77. What is IABP?

- INTRA-AORTIC BALLOON PUMP

78. What does an IABP do?

- Diastolic augmentation and systolic unloading.

79. What is diastolic augmentation?

A soft 40-mL balloon is inserted percutaneously through the common femoral artery into the descending thoracic aorta. The balloon is not occlusive (it should not touch the aortic walls). When it is inflated, it displaces 40 mL of blood and is exactly like acutely transfusing 40 mL of blood into the aorta, augmenting each left ventricular stroke volume by 40 mL. Balloon infusion is triggered off by the QRS complex from a surface ECG (any lead). The balloon always is inflated during diastole to increase diastolic blood pressure and augment coronary blood flow (CBF). Eighty percent of CBF occurs during diastole.

80. What is systolic unloading?

- Balloon deflation is an active (not a passive) process. Helium abruptly is sucked out of the balloon, leaving a 40-mL empty space in the aorta. The left ventricle can eject the first 40 mL of its stroke volume into this empty space-at dramatically reduced workload. An intra-aortic balloon increases coronary oxygen delivery (CBF) during diastole, while decreasing cardiac oxygen consumption just presystole.

81. KEY POINTS: INTRA-AORTIC BALLOON PUMP

- Indicated for cardiogenic shock refractory to pharmacologic manipulation.
- Triggered by QRS complex of surface ECG; inflates during diastole (T wave) and deflates on systole (R wave or at dicrotic notch on aortic pressure curve).
- 80% of coronary blood flow occurs during diastole.
- Mechanically results in diastolic augmentation and systolic unloading (afterload reduction).

82. Name the contraindications to IABP.

- **Aortic insufficiency:** diastolic augmentation distends and injures the left ventricle.
- **Atrial fibrillation:** balloon inflation and deflation cannot be appropriately timed.

83. Are clinical features of shock always typical?

Big no

A. Capillary refill: In distributive (septic) shock, the peripheries will be warm and capillary refill will be brisk despite profound shock.

B. Tachycardia not present in:

- Patients who are on β-blockers ,
- Patients who have implanted pacemakers (unable to mount a tachycardia).
- A pulse rate of 80 in a fit young adult who normally has a pulse rate of 50 is very abnormal.
- Furthermore, in some young patients with penetrating trauma, when there is haemorrhage but little tissue damage, there may be a paradoxical bradycardia rather than tachycardia accompanying the shocked state.

C. Blood pressure

- It is important to recognize that hypotension is one of the last signs of shock.
- Children and fit young adults are able to maintain blood pressure until the final stages of shock by dramatic increases in stroke volume and peripheral vasoconstriction. These patients can be in profound shock with a normal blood pressure.
- Elderly patients who are normally hypertensive may present with a ‘normal’ blood pressure for the general population but be hypovolaemic and hypotensive relative to their usual blood pressure.

84. What is distributive shock? give examples

Abnormal distribution of blood flow in the smallest blood vessels results in inadequate supply of blood to the body's tissues and organs. Inadequate organ perfusion is accompanied by vascular dilatation with hypotension, low systemic vascular resistance, inadequate after load and a resulting abnormally high cardiac output.

Causes:

- Sepsis (most common)
- Anaphylactic shock ,
- Spinal cord injury ,
- Neurogenic shock
- vasovagal shock and
- Psychogenic shock.

85. What part of the arterial system is responsible for the peripheral vascular resistance?

- The arterioles (not the major arteries nor the capillaries)

86. Why the arterioles is specifically responsible for peripheral vascular resistance?

- Because their walls are rich in smooth muscles and therefore much sympathetic innervation.

87. What is endocrine shock?

- A combination of hypovolemic, cardiogenic, and distributive shock.

Causes:

- **Hypothyroidism** causes shock state as a result of disordered vascular and cardiac responsiveness to circulating catecholamines. Cardiac output falls because of bradycardia in low inotropy.
- **Thyrotoxicosis** may cause a high output cardiac failure.
- **Adrenal insufficiency** cause shock as a result of hypovolemia and a poor response to circulating and exogenous catecholamine.

88. What is colloid?

A colloidal solution is one in which the majority of solute particles has a molecular weight greater than 30000. The term includes all plasma solutions, including human plasma protein fraction (HPPF), dextrans, gelatin (e.g. Haemaccel) and hydroxyethyl starch. Blood is not usually included in this term.

89. What is the cause of the signs found in shocked patient?

Sympathetic overstimulation and the purpose is to compensate the haemodynamic instability.

90. What is the meaning of compensated shock?

The body's cardiovascular and endocrine compensatory responses reduce flow to non-essential organs to preserve preload and flow to the lungs, brain and kidneys.

91. What is the meaning of decompensated shock?

- Further loss of circulating volume overloads the body's compensatory mechanisms and there is progressive renal, respiratory and cardiovascular decompensation.
- In general, loss of around 15% of the circulating blood volume is within normal compensatory mechanisms.
- Compensation exhausted when 30–40% of the circulating volume has been lost.

92. What are the first aids to shocked patient?

Ans. ABC

1) **A** ⟹ Ensure a patent airway
2) **B** ⟹ Adequate oxygenation and ventilation.
3) **C** ⟹ Cardiovascular resuscitation.

93. How you treat patient with shock?

- If there is initial doubt about the cause of shock, it is safer to assume the cause is hypovolaemia and begin with fluid resuscitation, followed by an assessment of the response.
- In all cases of shock, regardless the classification, hypovolaemia and inadequate preload must be addressed before other therapy is instituted.
- First-line therapy, therefore, is intravenous access and administration of intravenous fluids. Access should be through short, wide-bore cannulas that allow rapid infusion of fluids as necessary.
-

94. Why it is wrong to start with inotropic drugs?

For two reasons:

- Administration of inotropic or chronotropic agents to an empty heart will dramatically reduce diastolic filling and therefore coronary perfusion causing rapid and permanent depletion of oxygen to the myocardium
- Patients will enter the unresuscitable stage of shock as the myocardium becomes progressively more ischaemic and unresponsive to resuscitative attempts.

95. What type of fluid to be given to shocked patient?

- Crystalloid solutions (normal saline, Hartmann's solution, Ringer's lactate) are preferred over colloids (albumin or commercially available products because they are cheap and available).
- Most importantly, the oxygen-carrying capacity of crystalloids and colloids is zero. If blood is being lost, the ideal replacement fluid is blood, although crystalloid therapy may be required while awaiting blood products.
- Hypotonic solutions (e.g. dextrose) are poor volume expanders and should not be used in the treatment of shock unless the deficit is free water loss (e.g. diabetes insipidus) or patients are sodium overloaded (e.g. cirrhosis).

96. How you give the fluid for shocked patient?

- Give 250–500 ml of fluid over 5–10 min and the cardiovascular responses in terms of heart rate, blood pressure and central venous pressure (CVP) are observed. Then the patients can be divided into ‘responders’, ‘transient responders’ and ‘non-responders’.

Responders: show an improvement in their cardiovascular status, which is sustained. These patients are not actively losing fluid but require filling to a normal volume status.
Transient responders: show an improvement but then revert to their previous state over the next 10–20 min. These patients either have moderate on-going fluid losses (either overt haemorrhage or further fluid shifts reducing intravascular volume).
Non-responders: are severely volume depleted and are likely to have major on-going loss of intravascular volume, usually through persistent uncontrolled haemorrhage.

97. Treatment of different types of shock in brief

Hypovolaemic shock

- Lie patient flat; high flow O_2; lift legs to auto-transfuse.
- Give and repeat fluid infusion 500mL IV rapidly: you should see rise in BP. If no rapid improvement in BP look for other causes.
- Take blood and send for FBC, U & E, clotting, and cross-match.
- Take arterial blood gas: estimate pH as well as blood gases.

Anaphylactic shock

- ***Causes:*** drug allergy, blood product reaction.
- ***Clinical features:*** history of sudden onset after administration of drug. Stridor or bronchospasm, angioedema, urticaria, pruritus, rash.
- ***Treatment***
  - Sit patient up; give high flow O2; call anaesthetist if stridor.
  - If IV access present: give 1mL of 1:10 000 adrenaline bolus; flush; then 100mg hydrocortisone bolus; flush; then 10mg chlorpheniramine IV.
  - Repeat again in 5-10 min if no improvement.
  - If no IV access present: give 1mL 1:1000 adrenaline IM. Then secure IV access.
  - If wheezy give 5mL nebulized salbutamol.
  - IV fluid

Septic shock

- ***Cause.*** Overwhelming sepsis.
- ***Clinical features.*** May be the same as hypovolaemic shock or, if established, with circulatory collapse. Earlier in the evolution the patient may look septic pyrexial, flushed, bounding pulses.
- ***Treatment***

    - As for hypovolaemic shock.
    - Take blood cultures; then give IV antibiotics.

Cardiogenic shock

- ***Rapidly reverse the reversible causes:*** cardiac tamponade (trauma, post-cardiac surgery), arrhythmias, and tension pneumothorax.

Treatment

According to the cause:

- ***For heart failure*** give high flow O_2, 2.5mg morphine IV (anxiolytic, venodilator, analgesic, anti-arrhythmic) put the patient on cardiac monitors; request 12-lead ECG, treat arrhythmias.
- ***For*** **myocardial ischaemia** give 0.1mg GTN, 300mg aspirin.
- **Tension pneumothorax and cardiac tamponade** need surgical intervention.
- ***Additional measures include***

    - Consider central venous and peripheral arterial monitoring.
    - Send blood for arterial blood gases, FBC, U & E, clotting, troponin.
    - Catheterize the patient.
    - Request CXR look for pulmonary oedema.
    - Treat fluid overload with diuretics: frusemide 40mg IV.
    - Consider transthoracic echo to exclude pericardial effusion and valvular lesions, and to assess LV function.
    - Consider urgent coronary artery catheterization

98. What are the parameters to be monitored in shocked patient?

Minimum

- Electrocardiogram
- Pulse oximetry O2 saturation and pulse rate)
- Blood pressure and pulse rate
- Urine output

Additional modalities

- Central venous pressure
- Invasive blood pressure monitoring as PCWP
- Cardiac output
- Base deficit and serum lactate

99. What is the meaning of preload and afterload?

Preload ⟹ preheart ⟹ decrease filling of the right heart (or the left heart)

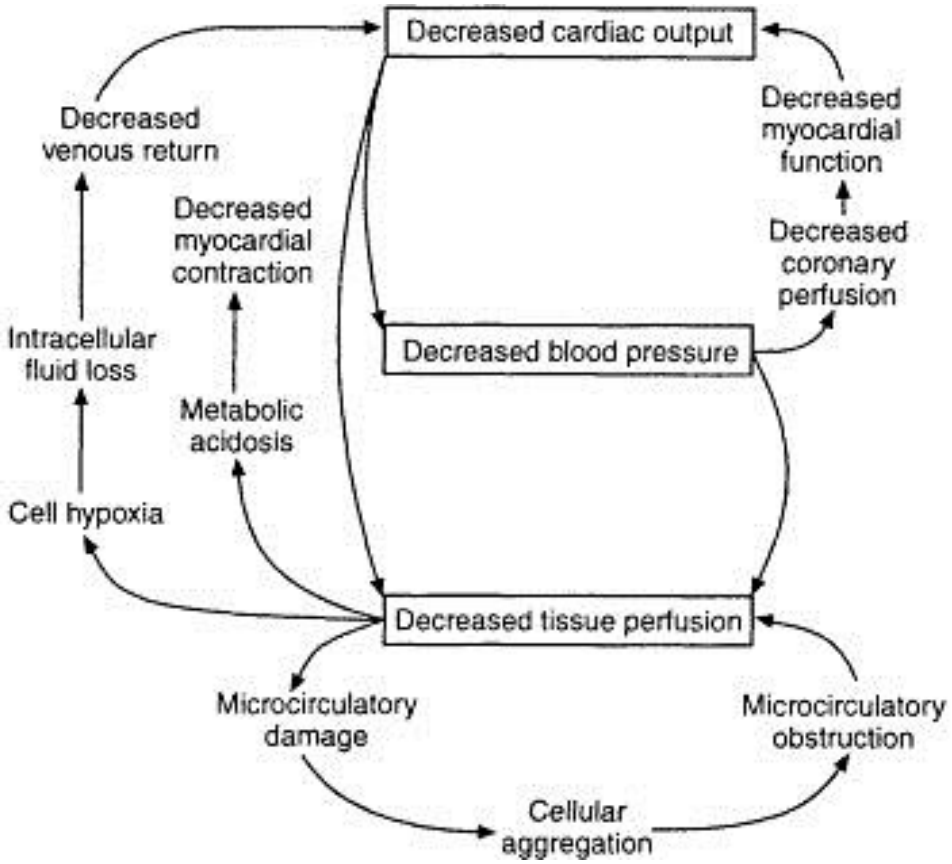
Afterload ⟹ After heart ⟹ decreased peripheral resistance ⟹ High output heart failure

100. How different types of shock affect the preload and afterload?

- Hypovolaemic shock ⟹ decrease preload
- Distributive shock ⟹ decrease afterload
- Right heart failure ⟹ decrease preload (for the left heart)
- Obstructive shock ⟹ decrease preload (decrease filling of the right heart (or the left heart)
- Endocrine shock is a combination of hypovolaemic, distributive and cardiogenic shock, so according to the endocrine problem, it could decrease the preload or the afterload.

101. Can shock state enter a vicious cycle?

- Yes; look at the figure below



- Vicious circles in shock. Initiation of shock can occur at any point, but the endpoint is often the same.

102. Discuss the pathophysiology of haemorrhage and shock

1. At the circulation and organ level

Low cardiac output (poor tissue perfusion)

Vasoconstriction (sympathetic stimulation) occurs in an attempt to maintain perfusion pressures to the vital organs, such as the brain, liver and kidneys, as well as the heart muscle itself

Venoconstriction pushes more blood into the dynamic circulation

Tachycardia helps to maintain a falling cardiac output

The minute ventilation rises 1.5—2 times and the respiratory rate 2—3 times maintaining oxygenation (except in cardiogenic shock with pulmonary oedema).

The renal blood flow is reduced with consequent reduction in glomerular filtration and urine output.

The renin—angiotensin mechanism is activated with further vasoconstriction and aldosterone release, causing salt and water retention.

Release of antidiuretic hormone (ADH) decreases the volume and increases the concentration of urine. However, in early sepsis the patient, although hypovolaemic, may produce inappropriately large amounts of dilute urine

As cardiac output falls, the hypotension and tachycardia cause poor perfusion of the coronary arteries, and this, in conjunction with hypoxia, metabolic acidosis and the release of specific cardiac depressants (endotoxaemia or pancreatitis), causes yet further cardiac depression and pump failure.

2. At the cellular level

Low cardiac output (poor tissue perfusion)

The cells become starved of oxygen, (metabolism shift from aerobic to anaerobic) and anaerobic metabolism leads to lactic acidosis.

Eventually, the cell membranes cannot pump sodium out of the cells; sodium enters the cells and potassium leaks out. Thus, the serum potassium is elevated.

Calcium, however, leaks into the cells lowering the serum calcium. Furthermore, the intracellular lysosomes break down and release powerful enzymes causing further damage — 'the sick cell syndrome'.

The platelets are activated in shock owing to the stagnation of blood in the capillaries.

Blood sludging with red cell aggregation may progress to the formation of small clots and, indeed, to DIC.

Several coagulation factors are consumed (platelets, fibrinogen, Factor V, Factor VIII, prothrombin), and troublesome bleeding may occur from needle puncture sites, wound edges and mucosal surfaces.

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103. Discuss the pathophysiology septic (endotoxic) shock

1. **Hyperdynamic (warm) septic shock.** This occurs in serious Gram-negative infections (strangulated intestine, peritonitis, leaking oesophageal or intestinal anastomoses, or suppurative biliary conditions.

At first, the patient has abnormal or increased cardiac output with tachycardia and a warm, dry skin,

But

The blood is shunted past the tissue cells, which become damaged by anaerobic metabolism (lactic acidosis).

The capillary membranes start to leak and endotoxin is absorbed into the blood-stream, leading to a generalised systemic inflammatory state.

The immediate and ready treatment of the cause, including the drainage of pus, is vital to the recovery of the patient at this stage (in strangulated hernia ‘the danger is in the delay, not in the operation’).

2. **Hypovolaemic hypodynamic (cold) septic shock.** This follows if severe sepsis or endotoxaemia is allowed to persist.

Generalised capillary leakage and other fluid losses lead to severe hypovolaemia with reduced cardiac output, tachycardia and vasoconstriction.

The systemic infection induces cardiac depression, pulmonary hypertension, pulmonary oedema and hypoxia

Reduction of cardiac output still further.

The patient becomes cold, clammy, drowsy and tachypnoeic,

But

Still can be converted to hyperdynamic (warm) shock by the administration of several litres of plasma or other colloidal solution.
(The similar use of crystalloid solutions (in large amount) may give rise to systemic and pulmonary oedema because of the larger volumes necessary.

Remember not to send shocked patient to the radiology unit because the patient may die in the way