

## ***Chapter 4: Haemodynamic disorders, shock***

1. Regarding platelets (2006)

- (a) They are the main source of thrombin
- (b) they number  $150-300 \times 10^3$  per microlitre
- (c) They contain a nucleus
- (d) They are biconcave discs
- (e) They contain  $\alpha$  and  $\beta$  granules

2. Non-inflammatory oedema

- (a) has a high protein content
- (b) is caused by low levels of aldosterone
- (c) has a SG  $>1.012$
- (d) is associated with high ANP
- (e) is caused by raised plasma oncotic pressure

3. Vascular hyperaemia

- (a) is caused by inflammatory mediators
- (b) results in cyanosis
- (c) results in oedema
- (d) results in brown induration
- (e) causes haemosiderin laden macrophages

4. Active hyperaemia is associated with (2006)

- (a) cyanosis
- (b) dilation of arteries and arterioles
- (c) release of a tourniquet on a peripheral limb
- (d) inflammatory mediators
- (e) the blood flow to all parts of the body except the brain

5. In the triple response, reactive hyperaemia is due to

- (a) exercise
- (b) blushing
- (c) arteriolar dilation
- (d) inflammatory mediators
- (e) is still present after sympathectomy

6. Which of the following is involved in the initiation of the clotting cascade, complement and kinin systems? (2004)

- (a) Hageman factor (XII)
- (b) factor VII
- (c) antigen-antibody complex
- (d) Tissue factor
- (e) Platelet activating factor

7. Activated Factor XII (factor XIIa, Hageman factor) *(from old paper 2004)*
- (a) activates clotting, the kinin cascade and fibrinolysis
  - (b) activates the coagulation cascade via the extrinsic pathway
  - (c) converts low molecular weight kininogen (LMWK) to high molecular weight kininogen
  - (d) converts factor VIII to active factor VIII (factor VIIIa)
  - (e) along with kallikrein, converts plasminogen to plasmin
8. Post mortem features of clot include: (2004, 2006)
- (a) adherence to the vascular wall
  - (b) absence of red cells in the supernatant
  - (c) lines of Zahn
  - (d) firmer texture and have more fibrin than venous clot
  - (e) none of the above features
9. Blood put in a glass tube
- (a) normally clots in 6 to 10 minutes
  - (b) clots because of the extrinsic pathway
  - (c) and then slowly tipped backwards and forwards gives a clinically useful clotting time
  - (d) clots almost instantaneously
  - (e) never clots
10. The process of blood coagulation involves
- (a) prothrombin activator converting fibrinogen to fibrin
  - (b)  $\alpha$ -2 macroglobulin
  - (c) the action of antithrombin III to promote clotting
  - (d) the action of plasmin on fibrin
  - (e) The removal of peptides from each fibrinogen molecule
11. White infarcts (2006)
- (a) may be transiently red
  - (b) occur in the intestine
  - (c) result from venous occlusion
  - (d) are always septic
  - (e) characteristically has lines of Zahn
12. White infarcts occur in
- (a) kidney
  - (b) the small intestine
  - (c) lung
  - (d) sigmoid colon
  - (e) oesophagus
13. Which of the following tissues/organs is most at risk of a venous infarct
- (a) appendix
  - (b) stomach
  - (c) ovary
  - (d) brain
  - (e) lung

14. With regard to embolism
- (a) arterial emboli most often lodge in the viscera
  - (b) pulmonary emboli are rarely multiple
  - (c) amniotic fluid emboli are associated with the highest mortality
  - (d) all emboli consist of either a gas or a solid intravascular mass
  - (e) most pulmonary emboli produce signs of respiratory compromise or distress
15. Fat embolism syndrome is associated with
- (a) symptoms within 24 hours after fracture of a long bone
  - (b) severe skeletal injuries in 30% of such patients
  - (c) thrombocytosis, and anaemia
  - (d) mortality in up to 10% of patients
  - (e) petechial rash, which is non-thrombocytopenic
16. Regarding air embolism, what is the least amount required to produce symptoms
- (a) 10mL
  - (b) 20mL
  - (c) 100mL
  - (d) 1000mL
  - (e) 1mL
17. Regarding air embolism
- (a) they do not leave the heart as the air is incompressible
  - (b) 200mL is the lethal dose
  - (c) Air may coalesce to form frothy obstructive masses
  - (d) It generally does not cause focal ischaemia
  - (e) It requires decompression as treatment
18. Amniotic fluid embolism (*subject in old MCQ paper*)
- (a) is seen in 0.5% of all deliveries
  - (b) is associated with a 20% to 40% mortality rate
  - (c) causes profound haemorrhage, due to the haemophilic properties of amniotic fluid
  - (d) is associated with disseminated intravascular coagulation in 20% of affected patients
  - (e) is due to rupture of uterine arteries
19. What best describes the pathophysiology underlying shock
- (a) Widespread tissue hypoxia as a result of decreased effective blood volume
  - (b) lactic acid production
  - (c) low cardiac output
  - (d) decreased blood volume
  - (e) Cellular hypoxia resulting from impaired tissue perfusion
20. Shock results in
- (a) decreases capillary hydrostatic pressure
  - (b) decrease in lipopolysaccharide levels
  - (c) Baroreceptor inhibition
  - (d) paradoxical inhibition of the sensation of thirst by the actions of ADH
  - (e) maximal sympathetic outflow after thirty minutes of profound hypotension

21. In acute cardiogenic shock there is
- (a) a decrease in right atrial pressure
  - (b) an increase in mean arterial pressure
  - (c) a decrease in capillary hydrostatic pressure
  - (d) oedema
  - (e) a survival rate of 50%
22. Irreversible shock is characterised by
- (a) widespread peripheral vasoconstriction
  - (b) increased intracellular adenosine
  - (c) increased adenosine levels in the blood
  - (d) distinct differences in renal function when compared with compensated shock
  - (e) none of the above
23. Septic shock may cause all of the following except
- (a) myocardial depression
  - (b) vasoconstriction
  - (c) DIC
  - (d) ARF
  - (e) ARDS
24. Disseminated intravascular coagulation is associated with
- (a) thrombocytosis
  - (b) a bleeding diathesis presentation in a patient with malignancy
  - (c) pregnancy in 50% of cases
  - (d) a haemolytic anaemia due to splenic activation
  - (e) bacterial exotoxins in the majority of patients with septic shock
25. Bradykinin is (2006)
- (a) produced by the actions of kallikrein on a precursor molecule
  - (b) is factor XII
  - (c)
  - (d)
  - (e)

## Answers

- Regarding platelets (p 126-128, 419 Guyton 10<sup>th</sup>, p650)
  - Thrombin is cleaved from a circulating precursor molecule thrombinogen by activated factor X and V, **and is produced by the liver**
  - they number  $150-300 \times 10^3 \mu\text{L}$**
  - They contain **no nucleus, and do not reproduce**
  - They are **round or oval discs 1-4  $\mu\text{m}$  in diameter**
  - They contain  **$\alpha$  granules (P-selectin, fibronectin, fibrinogen factors V & VIII and platelet factor IV which binds heparin), and  $\delta$  granules (ADP, ATP, Ca<sup>2+</sup>, histamine, 5-HT, and adrenaline)**
- Non-inflammatory oedema p8, 120-121
  - inflammatory oedema** has a high protein content because of leaky capillaries SG >1.020
  - is caused by **high levels of aldosterone**, due to decreased perfusion of the kidneys in situations such as CHF (secondary aldosteronism).
  - is **protein poor and has a SG <1.012**
  - is associated with high ANP (cardiac hypertrophy is associated with re-expression of ANP)**
  - is caused by **increased hydrostatic pressure (venous outflow obstruction, CHF) or reduced plasma osmotic pressure (excessive protein loss, decreased synthesis)**.  
Raised plasma oncotic pressure would draw fluid from the tissues
- “Vascular” hyperaemia is not mentioned in the texts, active and reactive hyperaemia is. (p122, Ganong 21<sup>st</sup> p629, Guyton 10<sup>th</sup> p178)
  - is caused by inflammatory mediators as it is part of the inflammatory response, and because of exercise**
  - results in cyanosis (**congestion**)
  - results in oedema (**not seen in exercise, seen in congestion**)
  - results in brown induration (**seen in the oedema of CHF, congestion**)
  - \*causes haemosiderin laden macrophages (**seen in the oedema of CHF, congestion**)
- Active hyperaemia is associated with
  - cyanosis (**reactive**)
  - dilation of arteries and arterioles**
  - \*release of a tourniquet on a peripheral limb (**reactive**)
  - \*inflammatory mediators (**reactive**)
  - \*the blood flow to all parts of the body **including the brain, which has increased blood flow in times of increased mental activity**
- In the triple response (reaction, wheal and flare), reactive hyperaemia is (Ganong 21<sup>st</sup> p629, Guyton 10<sup>th</sup> p178), unlikely to be a question as x3 response is not mentioned in recommended texts.
  - due to exercise (active)
  - due to blushing (active)
  - due to arteriolar dilation**
  - ?due to inflammatory mediators (substance P)
  - is still present after sympathectomy, not present after local anaesthetic, ?retrograde nerve signal.**

6. Which of the following is involved in the initiation of the clotting cascade, complement and kinin systems? (2004) (p67, 1278)
- (a) Hageman factor (XII): activates prekallikrein, kallikrein activates plasminogen to plasmin, which can then change C3 to C3a. XII activates the intrinsic clotting pathway.**
- (b) factor VII (*extrinsic pathway*)
- (c) antigen-antibody complex *alternative pathway of complement only*
- \* (d) Tissue factor (*extrinsic pathway*)
- \* (e) Platelet activating factor (*stimulates broncho/vasodilation*)
7. Activated Factor XII (factor XIIa, Hageman factor) (*from old paper 2004*) p67 fig. 2-15, p128
- (a) activates clotting, the kinin cascade and fibrinolysis**
- (b) activates the coagulation cascade via the *intrinsic* pathway
- (c) *high molecular weight kininogen (HMWK), activated platelets, basement membrane and collagen activate factor XII to XIIa*
- (d) converts *factor XI* to *active factor XI* (factor XIa)
- (e) *XIIa converts prekallikrein to kallikrein*. Kallikrein converts plasminogen to plasmin.
8. Post mortem features of clot include: (*Previous old paper 2004*) p133
- (a) adherence to the vascular wall is *a feature of living clots*
- (b) absence of red cells in the supernatant, and a chicken fat appearance**
- (c) lines of Zahn: *arterial thrombi*
- (d) firmer texture and have more fibrin = *venous clot*
- (e) none of the above features
9. Blood put in a glass tube (p429 Guyton 10<sup>th</sup>)
- (a) normally clots in 6 to 10 minutes, but varies with test method, so is not clinically useful**
- (b) clots because of the intrinsic pathway (no tissue trauma ∴ no tissue factor)
- (c) and then slowly tipped backwards and forwards gives a clotting time *but time varies with test method, so is not clinically useful*
- \* (d) *normally clots in 6 to 10 minutes*
- \* (e) *normally clots in 6 to 10 minutes*
10. The process of blood coagulation involves (p128, 130, p422, 424 Guyton 10<sup>th</sup>)
- (a) prothrombin activator converting *prothrombin to thrombin*
- (b) α-2 macroglobulin (*immunoglobulin- not a function*)
- (c) the action of antithrombin III *is to inhibit the activity of thrombin, and other serine proteases, factors IX-XII. It is activated by binding heparin like molecules on the endothelial surface*
- (d) the action of plasmin on fibrin, *acts to digest fibrin, break down factor V, VIII, and Hageman factor (XII) and therefore is anticoagulant*
- (e) The removal of [four low molecular weight] peptides from each fibrinogen molecule, to produce a fibrin monomer**

11. The colour of an infarct depends on whether or not there is bleeding. White (anaemic) infarcts are arterial occlusions in solid organs. They occur in the heart, spleen and kidney (end organs) p138

- (a) **may be transiently red, but the solid organ does not allow further bleeding**
- (b) occur in the intestine (*usually a red infarct*)
- (c) result from *arterial* occlusion
- (d) are *not necessarily septic in nature, they can be "bland"*
- (e) *arterial thrombi characteristically has lines of Zahn.*

White infarcts do not occur in the liver, as there is a dual blood supply

12. White infarcts occur with arterial occlusions in solid organs with end arterial circulations such as p138

- (a) **kidney, spleen and heart.**

Red infarcts occur with venous occlusions (ovarian torsion, loose tissue, which allows blood to pool in the infarcted area (lung) and areas with dual circulation (lung, liver)

- (b) the small intestine
- (c) lung
- (d) sigmoid colon
- (e) oesophagus

13. Which of the following tissues/organs is most at risk of a (red) venous infarct (p138)

- (a) appendix (*?small intestine, dual circulation stated as the reason for red infarct*)
- (b) stomach (*stomach infarct must be v rare*)
- (c) **ovary: torsion as the example given, but risk factors not stated**
- (d) brain
- (e) lung: *reason stated as dual circulation rather than obstruction of venous outflow*

14. With regard to embolism pp135-137

- (a) arterial emboli most often lodge in the *legs (75%), viscera (20%), brain (10%), arms (5%).*
- (b) pulmonary emboli are *usually multiple*
- (c) **amniotic fluid emboli are associated with the highest mortality 20-40%, (80% in old Robbins) 1:50,000 deliveries**
- (d) all emboli consist of either a gas or a solid intravascular mass, *or fluid*
- (e) most pulmonary emboli *are silent (60-80%), as they are small*

15. Fat embolism syndrome is associated with p136 (*from old paper*)

- (a) symptoms *1 to 3 days* after fracture of a long bone, including pulmonary insufficiency and cerebral irritability
- (b) *10% of patients* with severe skeletal injuries
- (c) *thrombocytopenia*, and anaemia, presumably due to platelets adhering to the numerous fat globules and being taken out of the circulation. Anaemia may be due to erythrocyte aggregation and haemolysis
- (d) **mortality of up to 10%**
- (e) petechial rash, which *is thrombocytopenic*

16. Regarding air embolism, what is the least amount of air required to produce symptoms? p137

- (a) 10mL
- (b) 20mL
- (c) 100mL, generally in excess of 100cc, a/c to the book**
- (d) 1000mL
- (e) 1mL

17. Regarding air embolism (p137) *answer not in examples.*

- (a) ***they can leave the heart and cause areas of ischaemia in peripheral and central tissues including the lungs***
- (b) ***>100mL causes symptoms***
- \* (c) Air may coalesce to form frothy obstructive masses**
- \* (d) It *can cause focal ischaemia* in a number of tissues including the brain and heart**
- \* (e) It requires *compression* as treatment**

18. Amniotic fluid embolism p137

- (a) is seen in ***1:50,000 of deliveries, important cause of maternal death***
- (b) is associated with a 20% to 40% mortality rate** (only answer in old example was 80% mortality)
- (c) causes ***DIC, due to the thrombogenic properties*** of amniotic fluid
- (d) is associated with disseminated intravascular coagulation in ***50% of affected patients***
- (e) is due to rupture of uterine veins, or a tear in the placental membranes, which allows amniotic leak. Classic findings are squamous cells in the pulmonary microcirculation, hair, or fat, or mucin from the GI tract of the baby.

19. What ***best*** describes the pathophysiology underlying shock (p139-142, Guyton 10<sup>th</sup> p253-261) not a great question, no text actually states the answer like this & a/c to the examiners, the text has to state the answer as it reads

- (a) Widespread tissue hypoxia as a result of reduction *in cardiac output*, or decreased or effective blood volume**
- (b) lactic acid production (***is a result of hypoperfusion***)
- (c) low cardiac output (***& or decrease effective circulating blood volume***)
- (d) ***decreased blood volume & or low cardiac output***
- (e) [impaired tissue perfusion and cellular hypoxia resulting from impaired tissue perfusion [regardless of the underlying pathology]**
- (x) infection
- (y) cardiac failure

20. Shock results in (p139-142, Guyton 10<sup>th</sup> p253-261)

- (a) decreased capillary hydrostatic pressure (but not actually stated)**
- \* (b) lipopolysaccharide is the *bacterial endotoxin that can initiate shock***
- \* (c) Baroreceptor *activation***
- \* (d) the sensation of *thirst by the actions of ADH***
- \* (e) maximal sympathetic outflow after *thirty seconds* of profound hypotension**

21. In acute cardiogenic shock there is (p139-142, Guyton 10<sup>th</sup> p239, 253-261)
- (a) **a decrease in right atrial pressure, with *decreased venous return*** (fig 24-3 Guyton)
  - (b) mean arterial pressure (*it can initially be normal or low*)
  - (c) ?a decrease in capillary hydrostatic pressure
  - (d) ***acute heart failure does not cause immediate development of oedema***
  - \* (e) a survival ***rate of <15%***
22. Irreversible shock is characterised by (p141, Guyton 10<sup>th</sup> p258, Ganong 21<sup>st</sup> p642)
- (a) widespread peripheral vasodilation (***severe ischaemia affects the brain, and vasomotor tone is lost***)
  - (b) ***adenosine is leached*** into the blood
  - (c) **increased adenosine levels in the blood, after all the ATP has been degraded to ADP and then AMP, it is converted to uric acid (Guyton)**
  - (d) these stages are ***indistinct***, and the patient can pass imperceptibly into refractory shock
  - (e) none of the above
23. Septic shock may cause all of the following ***except*** p140
- (a) [diminished]myocardial[activity] depression
  - (b) ***systemic vasodilation***
  - (c) DIC
  - (d) ARF
  - (e) ARDS
24. Disseminated intravascular coagulation is associated with p657-8
- (a) ***thrombosis and consumptive coagulopathy with consumption of platelets, which can result in a serious bleeding disorder***
  - (b) a bleeding diathesis presentation in a patient with ***obstetric complications or major trauma, whereas chronic DIC which occurs in cancer patients tends to present as complications of thrombosis***
  - \* (c) ***pregnancy in 50% of cases***
  - \* (d) a haemolytic anaemia due to ***microangiopathy. Fibrin deposits narrow the microvasculature***
  - \* (e) bacterial ***endotoxins*** in the majority of patients with septic shock