Robbins Review Questions - Chapter 4

1. Accumulation of fluid in the body tissue is known as _____ (edema/effusion) while fluid accumulation in the body cavities is known as _____ (edema/effusion).
2. Exudate is fluid which is ____(high/low) in protein.
3. Conditions leading to the inadequate synthesis of or increased loss of albumin leads to _____ (increased/reduced) oncotic pressure.
4. Edema in parts of the body with loose connective tissue (e.g. eyelids - periorbital edema) occurs when what happens?
    a. Heart failure
    b. Renal dysfunction
    c. Lymphatic dysfunction
    d. Liver dysfunction
5. Lymphedema can be due to
    a. Fibrosis
    b. Invasive tumors
    c. Infectious agents
    d. All of the following
6. Hyperemia is a/an _____ (active/passive) process due to augmented blood inflow from arteriolar dilation while congestion is a/an _____ (active/passive) process from the reduced outflow from a tissue.
7. Tissues that appear blue-red (cyanotic) are due to _____ (hyperemia/congestion).
8. Chronic pulmonary congestion, often caused by congestive heart failure, displays heart failure cells which are also known as
    a. Kupffer cells
    b. Hemosiderin laden macrophages
    c. Histiocytes
    d. Necrotic erythrocytes
9. Nutmeg liver is indicative of what general condition?
    a. Chronic passive hepatic congestion
    b. Chronic active hepatic hyperemia
    c. An abundance of hemosiderin-laden macrophages
    d. Acute hepatic congestion
10. Disruption of the endothelium exposes subendothelial _____ which promotes platelet adherence and activation.
    a. Gp11b
    b. Gp111b
    c. Thrombin
    d. Von Willebrand factor
11. Deposition of fibrin occurs in _____ (primary/secondary) hemostasis.
12. What 2 things must occur (in order) before fibrin deposition can begin?
    a. Clot stabilization and arteriolar vasoconstriction
    b. Secondary hemostasis and arteriolar vasoconstriction
    c. Arteriolar vasoconstriction and primary hemostasis
    d. Activation of thrombin and clot resorption

13. Once exposed following an injury to the endothelium, tissue factor activates
    a. Fibrin
    b. Platelets
    c. Tissue factor 7
    d. Tissue plasminogen activator
14. Fibrinogen is converted to fibrin by
    a. Von Willebrand factor
    b. Tissue factor 7
    c. Thrombin
    d. Tissue plasminogen activator
15. The inability of fibrin to bind to platelets due to a deficiency or extremely low levels of glycoprotein binding sites (GpIIb/IIIa) is caused by what coagulative disorder?
    a. Von Willebrand disease
    b. Glanzmann's thrombasthenia
    c. Bernard-Soulier syndrome
    d. None of the above
16. Bernard-Soulier syndrome is a cause of ineffective hemostasis due to/because
    a. Von Willebrand factor is no longer present on the subendothelium for platelets to bind to
    b. Platelets lack the requisite binding sites to "stick" to the subendothelial von Willebrand factor
    c. Activated platelets are unable to adhere to one another
    d. Fibrin is unable to bind to aggregating platelets
17. Partial Thromboplastin Time (PTT) is used as a time marker for the _____ (intrinsic/extrinsic) pathway.
18. Thrombin is the most important coagulation factor due to its many roles in hemostasis, some of which are
    a. Conversion of fibrinogen into fibrin
    b. Platelet activation
    c. Pro-inflammatory effects
    d. Anticoagulant effects
    e. All of the above
19. Within the fibrinolytic cascade, fibrinolysis is largely accomplished through the enzymatic activity of _____.
    a. Thrombin
    b. Plasmin
    c. Tissue factor 7
    d. None of the above
20. Once activated, _____ is/are released into the bloodstream which is a clinically useful marker of the state of fibrinolysis.
    a. Protein C
    b. D dimers
    c. Plasminogen
    d. Thromboxane A2
21. A factor 5 mutation, also known as a Leiden mutation, leads to a _____ (hypercoagulable/hypocoagulable) state.

22. Lines of Zahn are laminations recognizable in what?
    a. Scar tissue
    b. Thrombi
    c. Serous inflammation
    d. Necrotic vasculature
23. Arterial thrombi tend to grow _____ (in the direction of blood flow/retrograde) while venous thrombi tend to grow _____ (in the direction of blood flow/retrograde).
24. Mural thrombi occur within
    a. Deep leg veins
    b. Superficial leg veins
    c. Pulmonary arteries
    d. Heart chambers
25. Thrombi attached to heart valves are known as _____.
26. _____ (Superficial vein/deep vein) thrombi within the lower extremities are more prone to embolization.
27. The Virchow triad of thrombus formation includes all of the following except
    a. Endothelial injury
    b. Laminar blood flow
    c. Hypercoagulability
    d. Abnormal blood flow
28. _____ is an area of ischemic necrosis caused by occlusion of either the arterial supply or the venous drainage.
    a. Congestion
    b. Infarct
    c. Embolism
    d. Caisson disease
29. _____ (Red/White) infarcts occur in tissues with venous occlusions; in loose, spongy tissues (e.g., lung) where blood can collect in the infarcted zone; in tissues with dual circulations (e.g., lung and small intestine) that allow blood to flow from an unobstructed parallel supply into a necrotic zone; in tissues previously congested by sluggish venous outflow; and when flow is reestablished to a site of previous arterial occlusion and necrosis (e.g., following angioplasty of an arterial obstruction).
30. The dominant histologic characteristic of infarction throughout the body, except for the CNS, is
    a. Fibrinoid necrosis
    b. Liquefactive necrosis
    c. Gangrenous necrosis
    d. Ischemic coagulative necrosis
31. Histologic evidence of necrosis following infarction is evident after
    a. 0-4 hours
    b. 4-12 hours
    c. 12-24 hours
    d. 24-36 hours
32. Tissues generally resistant to tissue damage following vessel occlusion include all of the following except

a. Lung
b. Liver
c. Hand
d. Spleen

33. Infarcts are more likely a result of _____ (arterial/venous) occlusion.
34. _____ is a state in which diminished cardiac output or reduced effective circulating blood volume impairs tissue perfusion and leads to cellular hypoxia.
    a. Thrombosis
    b. Shock
    c. Infarction
    d. None of the above
35. Which of the following changes are not seen in shock?
    a. Adrenals show cortical cell lipid depletion due to the conversion of the relatively inactive vacuolated cells to metabolically active cells that utilize stored lipids for the synthesis of steroid
    b. Kidneys exhibit acute tubular necrosis
    c. Lungs are seldom affected in pure hypovolemic shock, because they are somewhat resistant to hypoxic injury. However, when the shock is caused by sepsis or trauma, diffuse alveolar damage results
    d. Compensatory protective mechanisms lead to progressive CNS hyperperfusion, indicated by Cushing's triad (Cheyne-Stokes breathing, elevated blood pressure and bradycardia)

1. Edema. Effusion (pg 113)
2. High (pg 113)
3. Reduced (pg 114)
4. B (pg 115)
5. D (pg 114)
6. Active. Passive (pg 116)
7. Congestion (pg 116)
8. B (pg 116)
9. A (pg 116)
10. D (pg 116)
11. Secondary (pg 116)
12. C (pg 116)
13. C (pg 116)
14. Thrombin (pg 116)
15. B (pg 118)
16. C (pg 117)
17. Intrinsic (pg 119)
18. E (pg 119-120)
19. B (pg 120)
20. B (pg 120)
21. Hypercoagulable (pg 123)
22. B (pg 125)
23. Retrograde. In the direction of blood flow (pg 125)
24. D (pg 125)
25. Vegetations (pg 125)
26. Deep vein (pg 126)
27. B (pg 126-127)
28. B (pg 129)
29. Red (pg 129-130)
30. D (pg 130)
31. B (pg 130)
32. D (pg 130)
33. Arterial (pg 129)
34. B (pg 131)
35. D (pg 134)