Part I. Clinical Applications

1. Mrs. Carlyle is pregnant for the first time. Her blood type is Rh negative, her husband is Rh positive, and their first child has been determined to be Rh positive. Ordinarily, the first such pregnancy causes no major problems, but the baby is born blue and cyanotic.

   A. What is this condition, a result of Rh incompatibility, called?
   Hemolytic disease of the newborn or erythroblastosis fetalis

   B. Why is the baby cyanotic?

   The RBCs have been destroyed by the mother’s antibodies; therefore, the baby’s blood is carrying insufficient oxygen

   C. Because this is Mrs. Carlyle’s first pregnancy, how can you account of the baby’s problems. Assume that there has been no fetal blood leakage to the mother.

   She must have received mismatched (Rh+) blood previously in a transfusion.

   D. Assume that the baby was born pink and healthy. What measures should be taken to prevent the previously described situation from happening in a second pregnancy and an Rh-positive baby?

   Give the mother RhoGAM to prevent her from becoming sensitized to the Rh+ antigen. RhoGAM is given to prevent the mother from producing antibodies towards the Rh factor protein. RhoGAM contains anti-Rh antibodies that remove fetal Rh positive antigens from the mother’s circulation before the mother’s immune system recognizes their presence and begins to produce Rh-positive antibodies. This protects the fetus of the next child in the event that this child has Rh-positive blood.

   E. Mrs. Carlyle’s sister has had two miscarriages before seeking medical help with her third pregnancy. Blood typing shows that she. Like her sister, is Rh negative; her husband is Rh positive. What course of treatment would be followed?

   Fetal progress will be followed in expectation of hemolytic disease of the newborn; intrauterine transfusions will be given if necessary, as well as complete blood transfusion to the newborn.

2. Cancer patients being treated with chemotherapy drugs designed to destroy rapidly dividing cells are monitored closely for changes in their RBC and WBC counts. Why?

   The stem cells for hematopoiesis in red bone marrow are a rapidly dividing cell population. Hence, they would be targeted (along with other rapidly dividing cells) by chemotherapeutic drugs.
3. Discuss the most significant danger experienced by athletes who engage in the illegal practice of blood doping.

Blood doping increases the number of RBCs and increases the viscosity of the blood, which increases the resistance to blood flow and makes the blood more difficult for the heart to pump. Increased viscosity, therefore, contributes to high blood pressure and increased risk of stroke.

4. Discuss four common characteristics of the many types of anemia.

1) reduced RBCs/hematocrit; reduced hemoglobin
2) fatigue (lack of oxygen needed for ATP production)
3) cold intolerance (lack of oxygen needed for heat production)
4) skin appears pale (low content of red-colored hemoglobin)

5. Describe the structural features of a mature red blood cell that help it function in gas transport.

a) It has the shape of a biconcave disc which helps make the cell flexible and increases its surface area for diffusion of gases
b) It lacks a nucleus and some organelles such as endoplasmic reticulum and thus has more space for gas transport.
c) The cytosol contains hemoglobin.
d) There are no mitochondria, and therefore the cell metabolizes anaerobically, and does not use up any of the carried oxygen.

6. Describe how aspirin and plavix can prevent clot formation.

Thromboxane A$_2$ (TxA$_2$) is a prostaglandin required for platelet aggregation and is a potent vasoconstrictor. The formation of thromboxane A2 is catalyzed by the cyclooxygenase-1 (COX-1) enzyme.

- Aspirin is a COX-1 and COX-2 inhibitor and thus reduces platelet aggregation by inhibiting thromboxane A$_2$ production.
- Since platelets are not compete cells, they cannot regenerate new enzymes and thus the COX-1 enzyme is inhibited for the life of the platelet (~10 days). Note: aspirin can significantly increase bleeding times.

Plavix inhibits platelet activation by blocking the receptors for ADP on the platelet plasma membrane. ADP is released by activated platelets to activate additional platelets.
Part III
1. anti-B
2. A, AB
3. A, O
4. B
5. B, AB
6. B, O
7. A, B
8. None
9. A, B, AB, O
10. Anti-A and Anti-B
11. A, B, AB, O
12. O
13. type 0
14. type AB
15. thicker; more
16. 38; 7.35 to 7.45; 0.9
17. 8; 5 to 6; 4 to 5
18. formed elements; plasma; 45; 55
19. 92; 8
20. Two differences:
   A. Leukocytes have a nucleus, mature erythrocytes do not
   B. Erythrocytes possess hemoglobin, Leukocytes do not

Part IV
1. Fractionated
2. Hypovolemic
3. Fibrin
4. Metalloproteins
5. Lipoproteins
6. Hematocrit
7. Hemoglobinuria
8. Hemolysis
9. Bilirubin
10. Thromboxane A2
11. Fibrinogen; Thrombin
12. Thrombocytopenia
13. Plasmin
14. Hematopoiesis
15. Hematocytoblasts
16. Hemoglobin level and Hematocrit
17. COX-1; COX-2; Thromboxane A2
18. Erythroblastosis fetalis
19. Prothrombinase or Factor X
20. Plavix
21. Tissue plasminogen activator; Urokinase; Streptokinase