Blood

1. Functions

A) Transportation of nutrients & waste

B) Regulation of pH & body temperature

C) Protection from blood loss & foreign invasion

2. Characteristics

A) Liquid connective tissue

B) 8% of human body weight

C) 4-5 times more viscous than water

D) NaCl conc. of 0.85-0.9%

E) pH ranges from 7.35-7.45

F) Total blood volume varies

1) 5-6L for males

2) 4-5L for females

G) Components

1) Plasma (55%)

a) About 90-92% water

b) Contains nutrients & wastes

c) Contains plasma proteins

d) Serum

2) Formed elements (45%)

a) Erythrocytes

b) Leukocytes

c) Platelets (thrombocytes)

3. Red Blood Cells (erythrocytes)

A) Biconcave shape – provides:

1) Greater surface area

2) Flexibility

B) Anucleate

C) Contain hemoglobin (~280 million molecules/RBC)

1) Globin – structural protein composed of 4 polypeptide chains each containing a

heme group

2) Heme group – iron-containing functional portion of the hemoglobin

a) Fe++ – forms a reversible bond with O2

D) Normal RBC counts: (1 drop ~50mm3)

Males = 4.7–6.1 mil/mm3

Females = 4.2–5.4 mil/mm3

Children = 4.6 – 4.8 mil/mm3

E) New cells produced at rate of ~2 mil/sec, live ~120 days

1) Controlled by erythropoietin

F) Erythrocytic Disorders

1) Anemia

a) Aplastic anemia – faulty bone marrow

i) Often caused by radiation, drug use, bacterial toxins, some poisons, and some

antibiotics

b) Pernicious anemia – decreased B12 (necessary for RBC production)

c) Hemolytic anemia

d) Hemorrhagic anemia

e) Iron-deficiency anemia

i) Results in microcytes

f) Sickle-cell anemia

i) Genetic defect causes one globin chain to become rigid

g) Thalassemia

i) Genetic defect resulting in absent or faulty globin chain

2) Polycythemia

a) Primary polycythemia (polycythemia vera)

b) Secondary polycythemia

FYI: Blood-doping

G) Erythropoiesis – controlled by erythropoietin

1) Hemocytoblast

2) Proerythroblast

3) Erythroblast

4) Normoblast

5) Reticulocyte – enter circulation and carry O2; continue to lose organelles

6) Erythrocyte (RBC)

4. White Blood Cells (leukocytes)

A) ~5,000 - 10,000/mm3; mot live only a few days, but their lifespan ranges from a few

minutes to years

B) There are 5 types of WBC broken down into 2 categories

1) Granulocytes (granular WBC)

a) Neutrophils (polymorphonuclear leukocytes) (50-70%)

i) Numerous fine granules which stain pale lilac w/ a darker multi-lobed nucleus

ii) Phagocytes

b) Basophils (0.5-1%)

i) Purplish-black granules that often obscure a bilobed nucleus

ii) Intensify inflammation and allergic reactions

iii) Chemicals released include histamine, heparin, leukotrienes, and

prostaglandins

iv) Were once thought to develop into mast cells

(a) Have identical functions except mast cells are only found outside the

bloodstream

c) Eosinophils (2-4%)

i) Distinct red granules; bilobed nucleus

ii) Phagocytes, combat parasitic worms & release anti-inflammatory chemicals

2) Agranulocytes

a) Monocytes (3-8%)

i) Pale-blue cytoplasm w/ U or kidney shaped nucleus

ii) Develop into macrophages once in tissue

iii) Phagocytes

b) Lymphocytes (20-25%)

i) Large, dark-purple nucleus occupies most of the cell

ii) Produce antibodies and provide immunity

iii) 3 types

(a) B lymphocytes

(i) Attack bacteria & their toxins

(ii) Develop into plasma cells which release antibodies

(b) T lymphocytes

(i) Attack viruses, fungi, cancer cells, transplanted cells & some bacteria

(ii) Work w/ B cells to provide immunity

(c) Natural Killer (NK) cells

(i) Kill cells that have been bound by antibodies or cells that exhibit

abnormal traits

(ii) Use perforins & granzymes to destroy the cells

C) Leukocytic Disorders

1) Leukopenia – decreased WBC count

a) Often caused by gluccocorticoids and various cancer & HIV drugs

2) Leukocytosis – increased WBC count

a) Often caused by acute infections, inflammation, or hemorrhage

3) Infectious mononucleosis (mono)

a) Caused by Epstein-Barr virus

4) Leukemia

a) Group of cancerous conditions involving WBC

b) May be named for cells affected

i) Ex. myelocytic leukemia or lymphocytic leukemia

c) May be named for the WBC’s stage

i) Ex. acute leukemia or chronic leukemia

D) Leukopoiesis

1) Stimulated by interlukins (IL) & colony-stimulating factors (CSF)

2) Multiple pathways but all start w/ hemocytoblasts in red bone marrow

a) Monoblast

i) Promonocyte

ii) Monocyte

b) Lymphoblast

i) Prolymphocyte

ii) Lymphocyte

c) Myeloblast

i) Promyelocyte

ii) Myelocyte

(a) Eosinophilic, basophilic or neutrophilic

iii) Band cell

(a) Eosinophilic, basophilic or neutrophilic

iv) Eosinophil, basophil or neutrophil

5. Platelets

A) ~250,000 - 400,000/mm3, live 5-9 days

B) Stop blood loss – 3 mechanisms

1) Vascular spasm

a) Vasoconstriction of damaged vessel

b) Can last minutes – hours

2) Platelet plug formation

a) Triggered by von Willebrand factor (VWF)

i) Adhere to exposed collagen fibers

ii) Activates platelets

b) Platelet adhesion

i) Initial sticking of platelets to the wound site

ii) Activates more platelets

c) Platelet aggregation

i) The sticking of platelets to already present platelets

d) Platelet plug

i) A cluster of platelets that temporarily seals the break in a vessel wall

3) Coagulation (blood clotting)

a) Involves over 30 different chemicals including:

i) 13 different clotting factors

ii) Vitamin K

iii) Ca++

b) Prothrombin activator (enzyme) is formed following the coming together of

various clotting factors

c) Prothrombin activator converts prothrombin (plasma protein) to thrombin

(enzyme)

d) Thrombin converts fibrinogen (plasma protein) to fibrin (fibers of the clot)

e) Formed elements become trapped in fibrin

f) Serum filters out

i) Clot results

g) Clot retraction

h) Fibrinolysis

C) Clotting Disorders

1) Thrombus – a clot in a healthy vessel

2) Embolus – a thrombus that has broken free and entered circulation

3) Embolism – when an embolus becomes trapped in another vessel; can lead to death

if it occurs in the heart or lungs

4) Hemophilia – an inability of the blood to clot properly

a) Sex-linked disorder

5) Thrombocytopenia

a) Decreased platelet count

b) Caused by any condition that suppresses or destroys bone marrow

D) Thrombopiesis

1) Stimulated by thrombopoietin

a) Hemocytoblast

b) Megakaryoblast

c) Promegakaryocyte

d) Megakaryocyte

i) Ruptures as it enters circulation

e) Platelet

6. Blood Typing

A) Based on presence/absence of specific antigens

B) ABO Groups

1) Determined by the presence or absence of antigens A & B

a) Type A blood – has only antigen A

b) Type B blood – has only antigen B

c) Type AB blood – has antigens A & B

d) Type O blood – has neither antigen

2) Blood also contain antibodies against the antigen(s) the RBC don’t have

a) Type A blood – has B antibodies

b) Type B blood – has A antibodies

c) Type AB blood – has no antibodies

d) Type O blood – has A & B antibodies

D) Rh Group

1) Refers to the presence or absence of Rh antigens (there are at least 45 different

ones)

a) Rh+ has at least one Rh antigen

b) Rh- has no Rh antigens

2) Antibodies are not present against Rh antigen(s) unless conflicting blood has been

introduced

a) First exposure – no immune response (rejection)

b) Subsequent exposures will result in an immune response (rejection)

E) Transfusions

1) If the body sees a foreign antigen it will attack the RBC causing them to clump

together

2) Always try to match blood types

3) Some exceptions can be made

a) Type O is the universal donor

b) Type AB is the universal recipient

4) Complications

a) Clumping in small vessels

b) RBC are destroyed by immune system releasing hemoglobin

c) Flu-like symptoms are common

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| Blood  Type | Antigens Present | Antibodies Present | Can Receive Blood From: | Can Donate Blood To: |
| A | A | B | A & O | A & AB |
| B | B | A | B & O | B & AB |
| AB | A & B | none | A, B,  AB & O | AB only |
| O | none | A & B | O only | A, B,  AB & O |

5) Hemolytic Disease of the Newborn

a) Also called Erythroblastosis fetalis

b) Results when mother is Rh- and baby is Rh+

c) Upon delivery, Rh+ antigens from the baby are transferred to the mother’s

bloodstream which causes her to produce anti-Rh antibodies

d) If the mother becomes pregnant again with an Rh+ child, the antibodies cross the

placenta, enter the circulation of the fetus, and cause extensive fetal erythrocyte

damage

e) Can be prevented in most all cases with the use of RhoGam