

# LEUKOCYTES

rvsd 23Feb2016

Martini's 5<sup>th</sup>: p 637-645, Martini's 6<sup>th</sup>: 666-672, 7<sup>th</sup>: 654-664, 8<sup>th</sup>: 666-673, 10<sup>th</sup>: 667-675

**WHITE BLOOD CELLS**, seen as buffy coat on top of packed hematocrit cells, usually 5000-10,000 /cmm

**Primary functions:** a) defend body against infection

b) remove debris

**Characteristics:**

a) amoeboid

b) **diapedesis**: ("across foot process") squeeze between endothelial cells in capillaries.

c) positive **chemotaxis**, migrate towards cell attractants: **cytokines, leukotrienes**

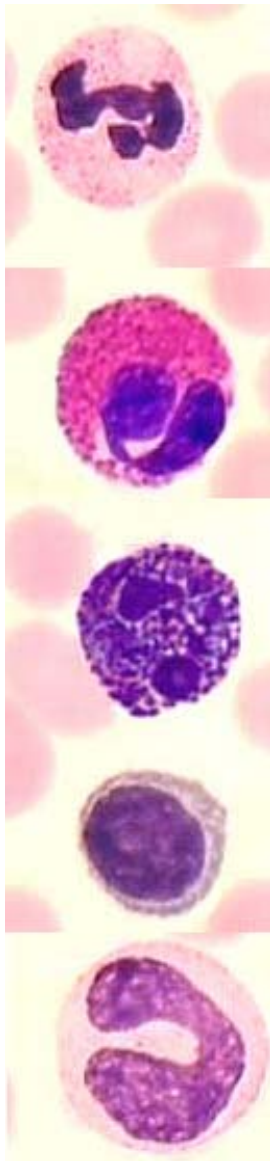
d) **phagocytic**

**TWO BROAD CLASSES** when stained with **Wright's stain: (p 670, 672)**

Wright's stain: **eosin** acid stain stains positive molecules red  
**methylene blue** basic stain stains negative molecules blue

**granulocytes** grains in cytoplasm, 90% of granulocytes are **polymorphonucleocytes**

**agranulocytes** no grains, non-lobed nucleus



**GRANULOCYTES:** (see TABLE: p 670, 672 for images, and lab handout for summary traits.)

- 1) **NEUTROPHILS:** pink&blue black 57-67%, **phagocytic**  
 (polymorphonuclear leukocytes= PMNs) increase with infection  
**lysozyme** in lysosomal granules, digests cell walls  
**H<sub>2</sub>O<sub>2</sub>** in "respiratory bursts" kills many pathogens  
**leukotrienes** attract other phagocytes when released
- 2) **EOSINOPHILS:** red speckled 1 - 3 % bilobed nucleus, **targets Ab labeled cells**  
 acidophils **phagocytoses Ag-Ab complex** (Ab marked cells etc)  
 Kills by exocytosis of NO and cytotoxic enzymes  
 increase in allergy & parasitic infection, leukemia
- 3) **BASOPHILS:** blue speckled 0.5 - 1% probably **like mast cells:**  
**histamine** (inflammation) & **heparin** (blood flows more freely)  
 "Alarm cells," accumulate in damaged tissue

**AGRANULOCYTES:** cytoplasm does not stain

- 1) **LYMPHOCYTES:** little cytoplasm 25 - 33% smaller cells, most numerous: produce antibodies, these increase during viral infections (nB: **mononucleosis**), other infections
- 2) **MONOCYTE:** large, "telephone receiver" 3 - 7% low in number: immature macrophage, incr in TB, protozoan infection, leukemia, mononucleosis. Aggressive phagocyte.

Origins of formed cells: 671

**LEUKEMIA:** can be of several types based on type of incr cell, often 500,000 WBC/cmm, resemble immature cells, nonfunctional: granulocytic lymphocytic monocytic

**Infectious mononucleosis:** (Epstein Barr virus) increase in monocytes ("benign disease");  
 signs and symptoms: incr temp enlarged lymph nodes, tender spleen, many lymphocytes fatigue sore throat

**PLATELETS:** thrombocytes 140,00-340,00/cmm:

Pinch off from **megakaryocytes** ("large nucleus cells"), 1/2 size of RBC. NOT cells, but rather pieces of cells.

Capable of ameoboid movement

Rich in ATP, collagen exposure causes them to clump together to form "platelet plug." Accelerated by thrombin.

Idiopathic thrombocytopenia purpura (ITP) ("clot-cell-poor")

**Petechiae** (pinhead size) hemorrhages are frequent in skin.

# HEMOSTASIS (INCLUDING BLOOD CLOTTING)

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Mareib, p. 592-597, Martini, pp. 667-676, Martini's 5<sup>th</sup>: 645-650, Martini's 6<sup>th</sup>: 672-677, 8<sup>th</sup>: 672-677, 10<sup>th</sup>: 674-680

**HEMOSTASIS:** prevent loss of blood from break in vascular wall. Three mechanisms:

1) **VASOCONSTRICTION (vascular phase of hemostasis)** (vascular spasm) is first response, due in part to **local trauma**. Endothelin released, stimulate vascular smooth muscle contraction. *Does not work well with clean cut.* (p 676)

2) **PLATELET PLUG:** (p 676)

Ruptured endothelium exposes **collagen**

collagen **causes platelets to swell, become spiked, sticky** (aggregation) to each other in exposed area,

Bound platelets release **thromboxane** (similar to prostaglandins synthesized by cyclooxygenase)

Platelets then react by releasing a battery of compounds (a process called degranulation):

**thromboxane** (a prostaglandin) stimulates further aggregation and vascular spasms (aspirin inhibits)

**serotonin** enhancing vascular spasm

**ADP** causes platelet aggregation

**Ca<sup>++</sup>** enhances clotting

Platelets join in positive feedback process, form a **platelet plug**

3) **COAGULATION:** (p 677) in outline, three stages. Extrinsic and intrinsic factors make **factor X**, first in **common pathway**:

A) **COMMON PATHWAY** (in blue on p 677)

1 **factor X** catalyzes formation of **thromboplastin (prothrombinase or prothrombin activator)**. Catalyzes:

2 **prothrombin converted to thrombin** Catalyzes:

3 **fibrinogen converted to fibrin** Fibrin is stabilized by fibrin stabilizing factor (XIII)

Balance of procoagulants (I through XIII, p 678) and anticoagulants prevents unwanted clotting:

**Most procoagulants are proteins formed in liver**

**ACTIVATION OF PROTHROMBINASE: TWO PATHWAYS LEAD TO COMMON PATHWAY**, both need Ca<sup>++</sup>

B) **EXTRINSIC** (factors released from *injured tissue*):

- 1) injured cells release tissue **thromboplastin** (Factor III also called tissue factor)
- 2) Ca<sup>++</sup> (factor IV) combines with thromboplastin, activates clotting factor VII
- 3) active tissue factor complex **converts factor X to prothrombinase**

C) **INTRINSIC** (factors found and activated in blood) :

- 1) aggregated platelets release platelet factor
- 2) Ca<sup>++</sup> (factor IV) combines, activates clotting factor VIII
- 3) acts on **VIII (hemophilic factor)** and IX, which **converts X to prothrombinase**

**ANTICOAGULANT, THERAPEUTIC AGENTS:**

Aspirin inhibits thromboxane production (As well as other prostaglandins)

Dicoumarol Vit K analog, prevents prothrombin synthesis (example: warfarin, rat poison)

Heparin inhibits thrombin

Ca<sup>++</sup> chelators prevent formation of clotting complex (Example: EDTA, citrate)

Streptokinase (a fibrinase) dissolves clots

**CONDITIONS:**

Thrombosis a clot is formed, exists in the vessels  
 embolus clot plugs vessel  
 infarct ("stuffed") lack of O<sub>2</sub> & nutrients causes death of heart tissue = flaccid heart muscle

