



NOTES

BLOOD COMPONENTS & FUNCTION

BLOOD COMPONENTS

osms.it/blood-components

BLOOD COMPONENT SEPARATION

- Blood components separate by density in centrifuge
 - **Heaviest layer:** erythrocytes
 - **Middle layer:** buffy coat
 - **Lightest layer:** plasma

ERYTHROCYTES

- Comprise 45% (hematocrit) of total blood volume
- Carry O_2 to tissues; bring CO_2 to lungs
- **Biconcave** discs (depressed center)
 - Fit through vessels, ↑ **surface area** (for **gas exchange**)
- **No organelles**
 - ↑ space for hemoglobins

BUFFY COAT

- Comprises < 1% of total blood volume
- Contains platelets, leukocytes
- Platelets **clump together** → **seal damaged blood vessels**
- Leukocytes ward off pathogens, destroy cancer cells, neutralize toxins

PLASMA

- Comprises 55% of total blood volume
- **No cells:** 90% water + proteins, electrolytes, gases
- **Albumin:** maintains oncotic pressure, acts as transport protein
- **Globulins:** **antibodies**, transport proteins
- **Fibrinogen:** involved in clot formation (helps platelets attach)
- **Electrolytes:** include sodium, potassium, calcium, chloride, carbonate

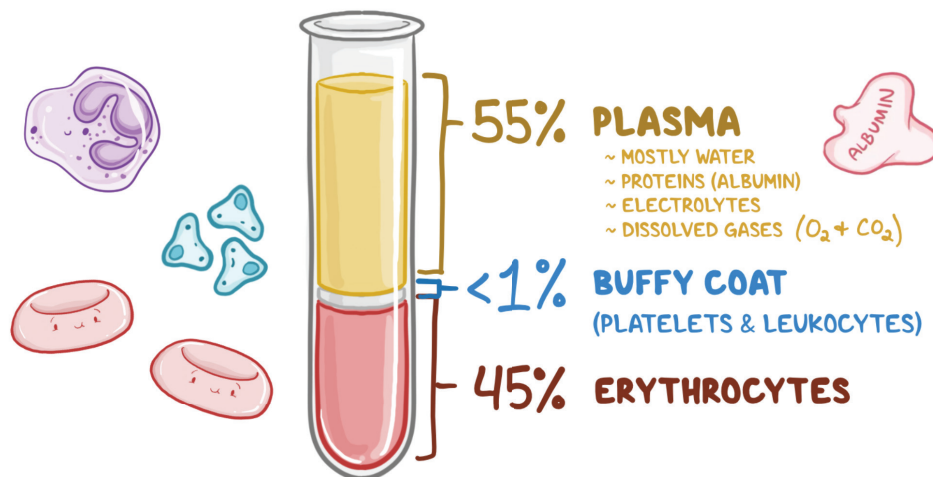


Figure 43.1 Blood components and their relative proportions.

PLATELET PLUG FORMATION (PRIMARY HEMOSTASIS)

osms.it/platelet-plug-formation-primary-hemostasis

- Hemostasis: blood-loss prevention
- First two hemostasis steps: platelets clump, form plug around injury site in five steps

PLATELET PLUG FORMATION STEPS

1. Endothelial injury

- Nerves, smooth muscle cells detect injury
- Trigger reflexive contraction of vessel (vascular spasm) → ↓ blood flow, loss
- Secretion of nitric oxide, prostaglandins stop; secretion of endothelin begins → further contraction

2. Exposure

- Damage to endothelial cells exposes collagen
- Damaged cells release Von Willebrand factor (binds to collagen)

3. Adhesion

- GPIIB surface proteins on platelets bind to Von Willebrand factor

4. Activation

- Platelet changes shape (forms arms to grab other platelets), releases more von Willebrand factor, serotonin, calcium, ADP, thromboxane A₂ (positive feedback loop)
- ADP, thromboxane A₂ result in GPIIB/IIIA expression

5. Aggregation

- GPIIB/IIIA binds to fibrinogen, links platelets → platelet plug

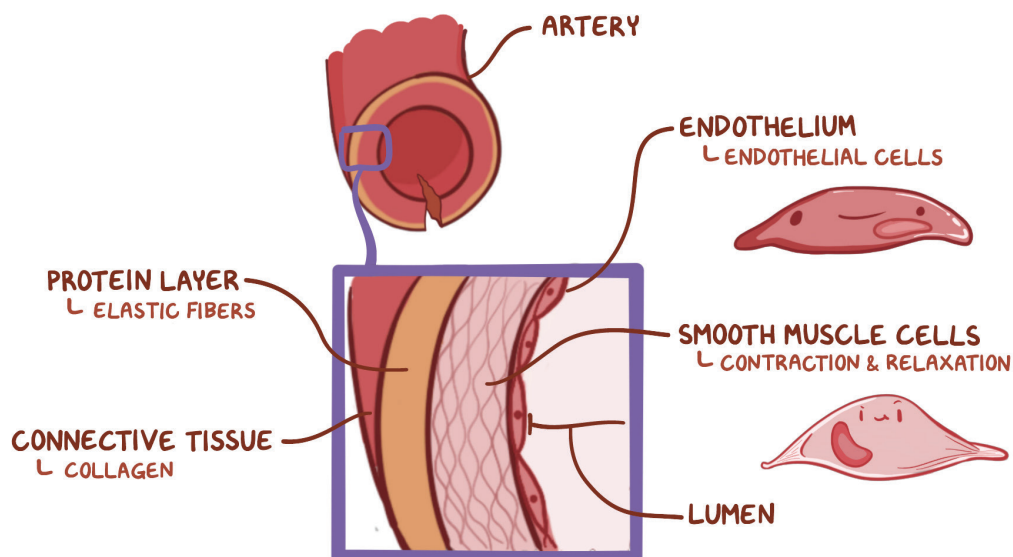
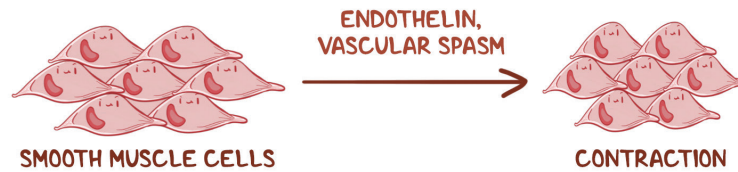


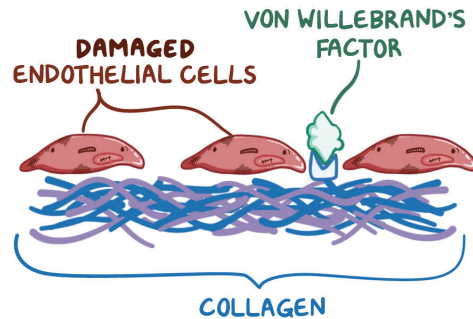
Figure 43.2 Layers of an arterial wall.

STEPS of PRIMARY HEMOSTASIS

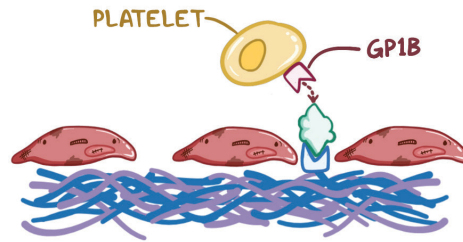
1) ENDOTHELIAL INJURY



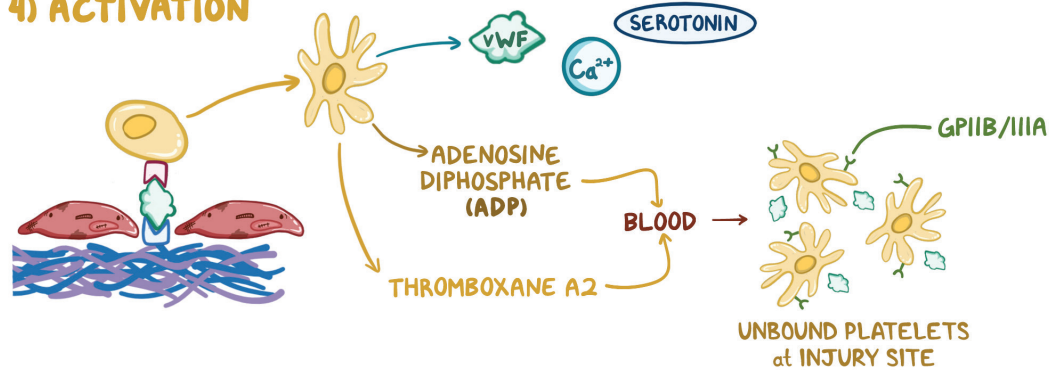
2) EXPOSURE



3) ADHESION



4) ACTIVATION



5) AGGREGATION

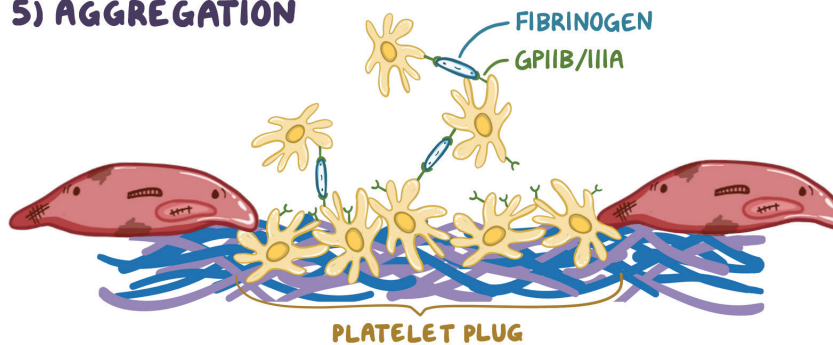


Figure 43.3 Platelet plug formation steps.

COAGULATION (SECONDARY HEMOSTASIS)

osms.it/coagulation-secondary-hemostasis

- Last two hemostasis steps: clotting factors activate fibrin, build fibrin mesh around platelet plug
- Begins with either extrinsic/intrinsic pathway; factor X activation → coagulation cascade (common pathway)

EXTRINSIC PATHWAY

1. Trauma damages blood vessel, exposes cells under endothelial layer
 - Tissue factor (factor III) embedded in membrane
2. Factor VII in blood binds to tissue factor, calcium → VIIa-TF complex

INTRINSIC PATHWAY

1. Circulating factor XII contacts negatively charged phosphates on platelets/ subendothelial collagen → factor XIIa
2. Factor XIIa cleaves factor XI → factor XIa
3. Factor XIa + calcium cleaves factor IX → factor IXa

4. Factor IXa + factor VIIIa (binds to Von Willebrand factor) + calcium → enter the common pathway

COMMON PATHWAY

1. Factor X is cleaved → factor Xa
2. Factor Xa cleaves factor V → factor Va
3. Factor Xa + factor Va + calcium → prothrombinase complex
 - Prothrombin (factor II) → thrombin (factor IIa)
4. Thrombin activates platelets, cofactors (V, VIII, IX); cleaves fibrinogen, stabilizing factor (→ factor XIIIa + calcium → cross-links in mesh)

COAGULATION TESTS

- Prothrombin time (PT): tests extrinsic pathway
- Activated partial thromboplastin time (aPTT): tests intrinsic pathway

ROLE OF VITAMIN K IN COAGULATION

osms.it/vitamin-k-in-coagulation

- Vitamin K regulates blood coagulation
 - Converts coagulation factors into mature forms
- 12 coagulation factors: (I–XIII, no factor VI); factors II, VII, IX, X require vitamin K
- Quinone reductase reduces vitamin K quinone (dietary form) into vitamin K hydroquinone
- Vitamin K hydroquinone donates electrons to γ -glutamyl carboxylase, converting

non-functional forms of II, VII, IX, X into functional forms

- Adds chemical group made of one carbon, two hydrogens, one oxygen to glutamic acid residues on proteins
- After carboxylation step, vitamin K (as vitamin K epoxide) is converted back into vitamin K quinone via epoxide reductase
- Coagulation factors appear in all coagulation pathways

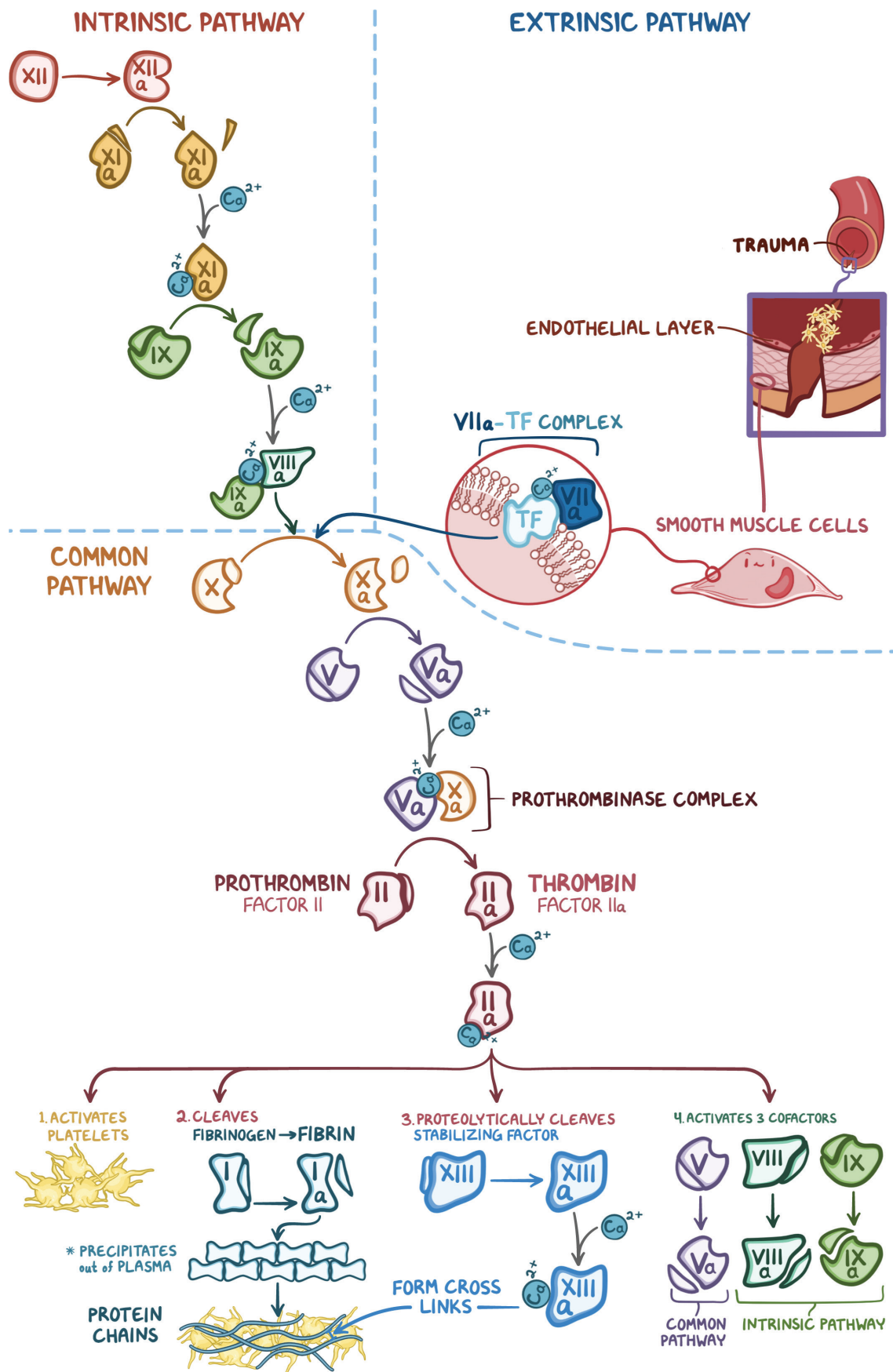


Figure 43.4 Coagulation steps, including the intrinsic, extrinsic, and common pathways.

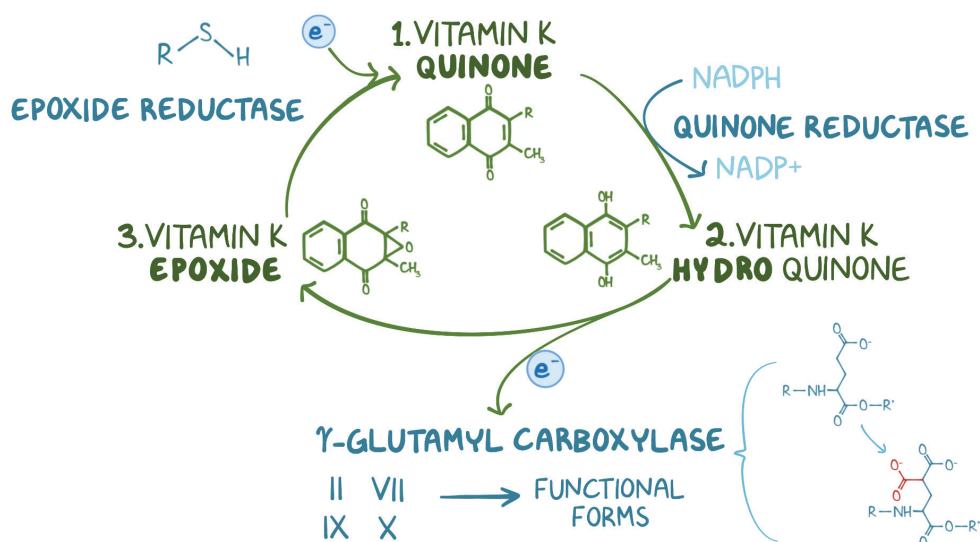


Figure 43.5 Vitamin K cycle. A single molecule of Vitamin K can be reused many times.

ANTICOAGULATION, CLOT RETRACTION & FIBRINOLYSIS

osms.it/clot-retraction-and-fibrinolysis

ANTICOAGULATION

- Occurs during primary, secondary hemostasis; regulates clot formation
- Prevents clots from growing too large → block blood flow, form emboli
- Regulation starts with thrombin (factor II)
 - Multiple pro-coagulative functions
 - Proteins C, S bind thrombomodulin-thrombin → cleaves, inactivates factors V, VIII
 - Antithrombin III binds thrombin/factor X → inactivates both (plus factors VII, IX, XI, XII with lower affinity)
- Other factors prevent platelets adhering during primary hemostasis
 - Nitric oxide, prostacyclin → ↓ thromboxane A₂

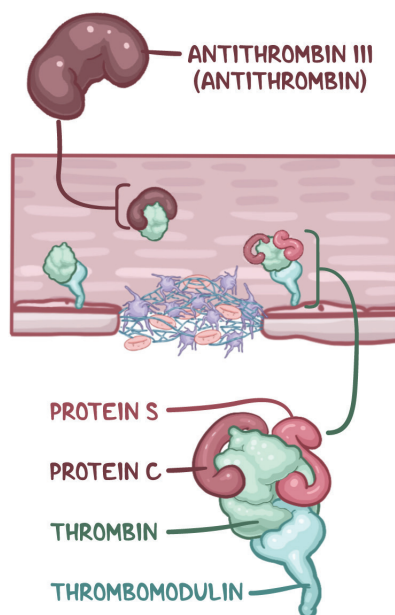


Figure 43.6 Proteins involved in anticoagulation. Thrombomodulin is found on the surface of intact epithelial cells lining blood vessels.

CLOT RETRACTION

- Occurs one hour after primary, secondary hemostasis
 - Contracts clot
- Platelets in clot express integrin $\alpha IIb\beta 3$ → binds to fibrin expressing actin, myosin → lamellipodia contract, fibrin mesh tightens closing wound

FIBRINOLYSIS

- Occurs two days after primary, secondary hemostasis; degrades clot
- Plasminogen → plasmin (via tissue plasminogen activator)
- Plasmin proteases fibrin → clot dissolves

BLOOD GROUPS & TRANSFUSIONS

osms.it/blood-groups-and-transfusions

BLOOD TRANSFUSIONS

- **Blood transfusion:** person receives blood/elements of blood (usually through intravenous infusion)
 - **Homologous transfusion:** anonymous donor
 - **Autologous transfusion:** self-donor (e.g. in planned surgery)
- Blood is mixed with calcium oxalate to prevent coagulation, refrigerated/frozen for storage

- Immune system produces antibodies against absent glycoproteins
- **Type AB:** no antibodies → universal recipients
- **Type O:** no antigens → universal donors

Rh system

- Determined by presence of Rh protein
 - Rh positive; Rh negative
- Rh+ can receive blood from either group
- Rh- can only receive Rh- blood

BLOOD TYPING

- Transfusion blood types not compatible → autoimmune reaction (hemolytic transfusion reaction)
- **Two classification systems** (based on presence/absence of proteins)
 - ABO system
 - Rh system

CROSS MATCHING

- Test to confirm donor's blood is safe for recipient
- Recipient serum is mixed with donor blood
 - **Agglutination reaction:** cannot receive

ABO system

- Determined by type of glycoproteins found on red blood cells (RBCs)
 - Type A; type B; type A & B; type O (neither)

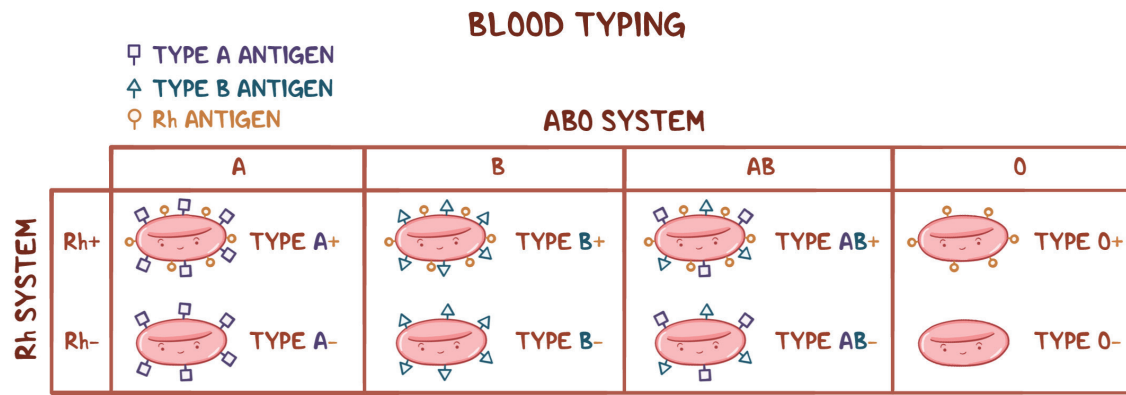


Figure 43.7 Blood types are reported as ABO group and Rh + or -. When both classification systems are combined, there are eight possible blood types: A+, A-, B+, B-, AB+, AB-, O+, O-.