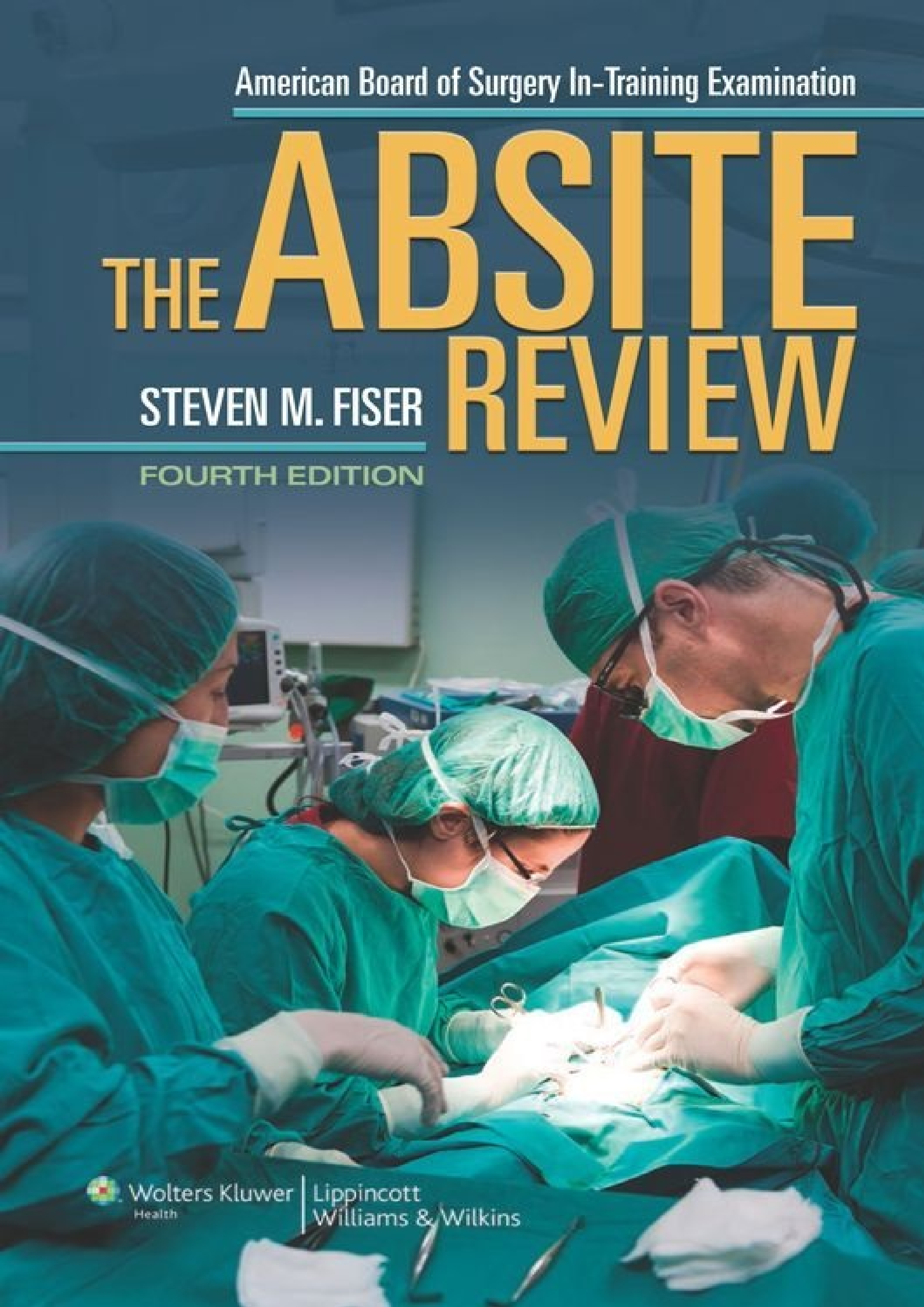
American Board of Surgery In-Training Examination

THE ABSITE REVIEW

STEVEN M. FISER

FOURTH EDITION



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The ABSITE Review

Fourth Edition

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PREFACE TO THE FIRST EDITION

Each year, thousands of general surgery residents across the country express anxiety over preparation for the American Board of Surgery In-Training Examination (ABSITE), an exam designed to test residents on their knowledge of the many topics related to general surgery.

This exam is important to the future career of general surgery residents for several reasons. Academic centers and private practices searching for new general surgeons use ABSITE scores as part of the evaluation process. Fellowships in fields such as surgical oncology, trauma, and cardiothoracic surgery use these scores when evaluating potential fellows. Residents with high ABSITE results are looked upon favorably by general surgery program directors, as high scorers enhance program reputation, helping garner applications from the best medical students interested in surgery.

General surgery programs also use the ABSITE scores, with consideration of feedback on clinical performance, when evaluating residents for promotion through residency. Clearly, this examination is important to general surgery residents.

Much of the anxiety over the ABSITE stems from the issue that there are no dedicated outline-format review manuals available to assist in preparation. *The ABSITE Review* was developed to serve as a quick and thorough study guide for the ABSITE, such that it could be used independently of other material and would cover nearly all topics found on the exam. The outline format makes it easy to hit the essential points on each topic quickly and succinctly, without having to wade through the extraneous material found in most textbooks. As opposed to question-and-answer reviews, the format also promotes rapid memorization.

Although specifically designed for general surgery residents taking the ABSITE, the information contained in *The ABSITE Review* is also especially useful for certain other groups:

- General surgery residents preparing for their written American Board of Surgery certification examination
- Surgical residents going into another specialty who want a broad perspective of general surgery and surgical subspecialties (and who may also be required to take the ABSITE)
- Practicing surgeons preparing for their American Board of Surgery recertification examination

PREFACE TO THE FOURTH EDITION

The 4th edition of The ABSITE Review is the most refined to date. It provides a very rapid review of the material found on the ABSITE while still providing sufficient explanations so the reader does not feel lost. Many of the tables and algorithms have been condensed and distilled down to relevant outlines, improving the efficiency of reading time. New sections have been added to reflect recent exams, including outlines on patient safety and surgical quality.

Again, I thank all of the residents who gave me feedback on the books or who I met at surgical meetings saying, “I used your books in residency and they were great.” I am glad I could help out.

Thank you again and good luck on the ABSITE.

CHAPTER 1. CELL BIOLOGY

CELL MEMBRANE

A **lipid bilayer** that contains protein channels, enzymes, and receptors

Cholesterol increases membrane fluidity

Cells are negative inside compared to outside; based on Na/K ATPase (3 Na^+ out/2 K^+ in)

The **Na^+ gradient** that is created is used for **co-transport** of glucose, proteins, and other molecules

Electrolyte Concentrations of Intracellular and Extracellular Fluid Compartments

	Extracellular Fluid (mEq/L)	Intracellular Fluid (mEq/L)
CATIONS		
Na^+	140	12
K^+	4	150
Ca^{2+}	5	10^{-7}
Mg^{2+}	2	7
ANIONS		
Cl^-	103	3
HCO_3^-	24	10
$SO_4 2^-$	1	–
HPO_4^{3-}	2	116
Protein	16	40
Organic anions	5	–

Adapted from Wait RB, et al. Fluids and electrolytes and acid-base balance. In: Greenfield LJ, et al., eds. *Surgery: Scientific Principles and Practice*. 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2001:245.

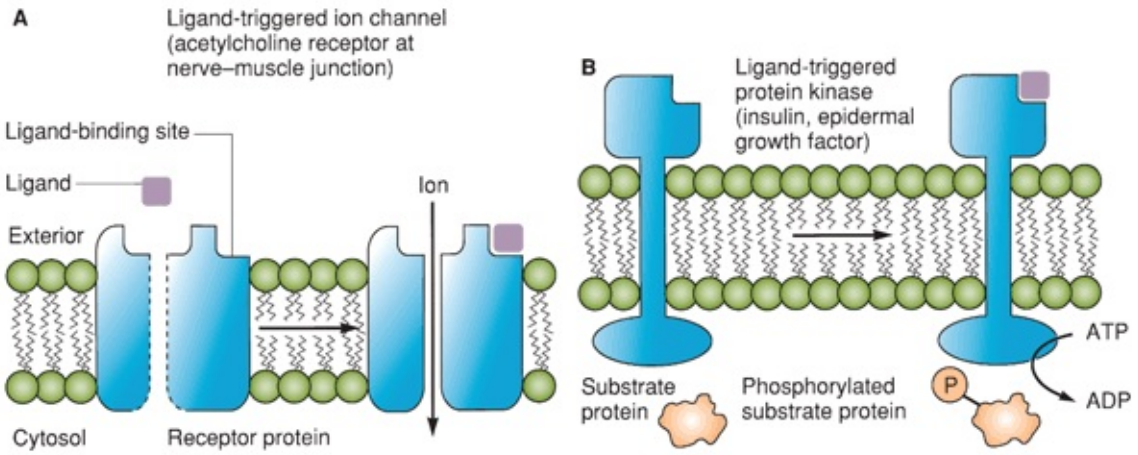
Desmosomes/hemidesmosomes – adhesion molecules (cell–cell and cell–extracellular matrix, respectively), which anchor cells

Tight junctions – cell–cell occluding junctions; form an impermeable barrier (eg epithelium)

Gap junctions – allow communication between cells (connexin subunits)

G proteins – intramembrane proteins; transduce signal from receptor to response enzyme

Ligand-triggered protein kinase – receptor and response enzyme are a single transmembrane protein



Types of cell surface receptors. *(A)* Ligand-activated ion channel; binding results in a conformational change, opening or activating the channel. *(B)* Ligand-activated protein kinase; binding activates the kinase domain, which phosphorylates substrate proteins. (*continued*)

ABO blood-type antigens – glycolipids on cell membrane
HLA-type antigens – glycoproteins (Gp) on cell membrane
Osmotic equilibrium – water will move from an area of low solute concentration to an area of high solute concentration and approach osmotic equilibrium

CELL CYCLE

G1, **S** (protein synthesis, chromosomal duplication), **G2**, **M** (mitosis, nucleus divides)
G1 most variable, determines cell cycle length
Growth factors affect cell during G1
Cells can also go to G0 (quiescent) from G1
Mitosis

- **Prophase** – centromere attachment, spindle formation, nucleus disappears
- **Metaphase** – chromosome alignment
- **Anaphase** – chromosomes pulled apart
- **Telophase** – separate nucleus reforms around each set of chromosomes

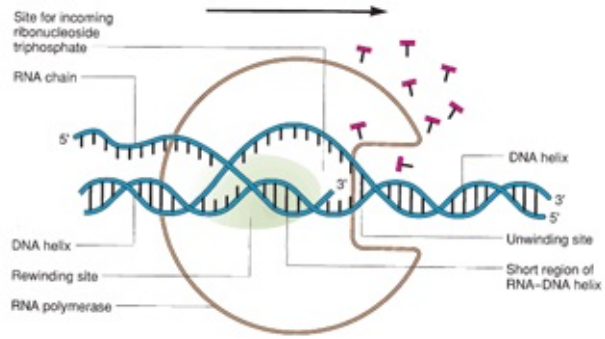
NUCLEUS, TRANSCRIPTION, AND TRANSLATION

Nucleus – double membrane, outer membrane continuous with rough endoplasmic reticulum
Nucleolus – inside the nucleus, no membrane, **ribosomes** are made here
Transcription – DNA strand is used as a template by **RNA polymerase** for synthesis of an mRNA strand
Transcription factors – bind DNA and help the transcription of genes

- **Steroid hormone** – binds receptor in cytoplasm, then enters nucleus and acts as transcription factor

- **Thyroid hormone** – binds receptor in nucleus, then acts as a transcription factor
- Other transcription factors – AP-1, NF-κB, STAT, NFAT

Initiation factors – bind RNA polymerase and initiate transcription



Transcription of DNA. RNA polymerase acts to unwind the DNA helix, catalyzes the formation of a transient RNA–DNA helix, and then releases the RNA as a single-strand copy while the DNA rewinds. In the process, the polymerase moves along the DNA from a start sequence to a stop sequence.

DNA polymerase chain reaction – uses oligonucleotides to amplify specific DNA sequences

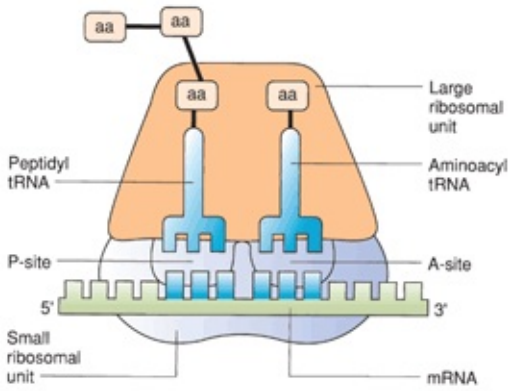
Purines – guanine, adenine

Pyrimidines – cytosine, thymidine (only in DNA), uracil (only in RNA)

- Guanine forms 3 hydrogen bonds with cytosine
- Adenine forms 2 hydrogen bonds with either thymidine or uracil

Translation – mRNA used as a template by **ribosomes** for the synthesis of **protein**

Ribosomes – have small and large subunits that read mRNA, then bind appropriate tRNAs that have amino acids, and eventually make proteins



Schematic view of the elongation phase of protein synthesis on a ribosome. As the ribosome moves along the mRNA, incoming aminoacyl–tRNA complexes bind to the A-site on the ribosome, after which a new peptide bond is formed with the nascent polypeptide chain previously attached to the peptide tRNA. The ribosome then moves, ejecting the now-empty tRNA and opening the A-site for the next aminoacyl–tRNA complex.

CELLULAR METABOLISM

Glycolysis – 1 glucose molecule generates 2 ATP and 2 pyruvate molecules

Mitochondria – 2 membranes, Krebs cycle on inner matrix, NADH/$FADH_2$ created

- **Krebs cycle** – the 2 pyruvate molecules (from the breakdown of 1 glucose) create NADH and $FADH_2$
- NADH and $FADH_2$ enter the electron transport chain to create ATP
- Overall, 1 molecule of glucose produces 36 ATP

Gluconeogenesis – mechanism by which **lactic acid** (Cori cycle) and **amino acids** are converted to glucose

- Used in times of starvation or stress (basically the glycolysis pathway in reverse)
- **Fat and lipids** are not available for gluconeogenesis because acetyl CoA (breakdown product of fat metabolism) cannot be converted back to pyruvate

Cori cycle – mechanism in which the **liver** converts **muscle lactate** into new **glucose**; pyruvate plays a key role in this process

OTHER CELL ORGANELLES, ENZYMES, AND STRUCTURAL COMPONENTS

Rough endoplasmic reticulum – synthesizes proteins that are exported (increased in pancreatic acinar cells)

Smooth endoplasmic reticulum – lipid/steroid synthesis, detoxifies drugs (increased in liver and adrenal cortex)

Golgi apparatus – modifies proteins with carbohydrates; proteins are then transported to the cellular membrane, secreted, or targeted to lysosomes

Lysosomes – have digestive enzymes that degrade engulfed particles and worn-out organelles

Phagosomes – engulfed large particles; these fuse with lysosomes

Endosomes – engulfed small particles; these fuse with lysosomes

Protein kinase C – activated by **calcium** and **diacylglycerol** (DAG)

- Phosphorylates other enzymes and proteins

Protein kinase A – activated by **cAMP**

- Phosphorylates other enzymes and proteins

Myosin – thick filaments, uses ATP to slide along actin to cause **muscle contraction**

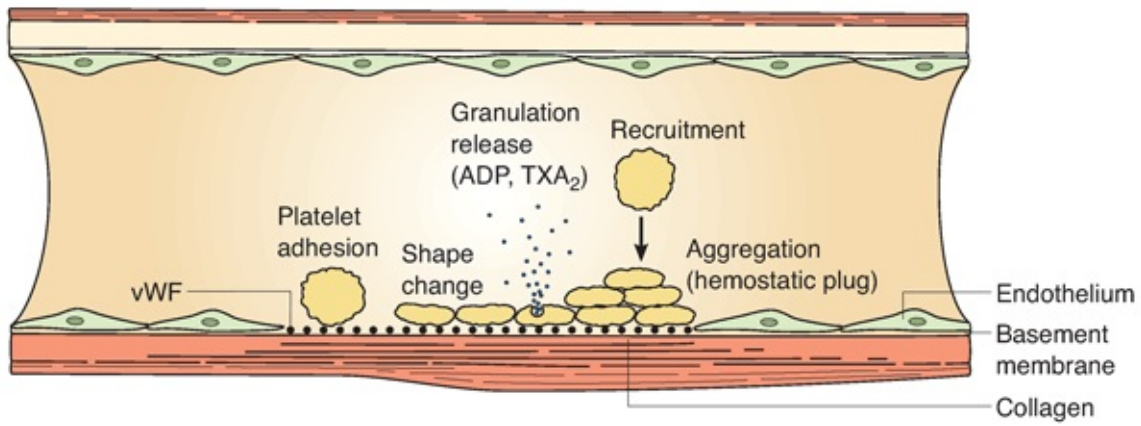
Actin – thin filaments, interact with myosin above

Intermediate filaments – keratin (hair/nails), desmin (muscle), vimentin (fibroblasts)

Microtubules – form specialized cellular structures such as cilia, neuronal axons, and mitotic spindles; also involved in the transport of organelles in the cell (form a latticework inside the cell)

- **Centriole** – a specialized microtubule involved in cell division (forms spindle fibers, which pull chromosome apart)

CHAPTER 2. HEMATOLOGY



Primary hemostasis is achieved initially with a platelet aggregation as illustrated. Note that platelet adhesion, shape change, granule release followed by recruitment, and the hemostatic plug at the area of subendothelial collagen and collagen exposure are the initial events for thrombus formation.

NORMAL COAGULATION

Three initial responses to vascular injury: vascular vasoconstriction, platelet adhesion, and thrombin generation

Intrinsic pathway: exposed **collagen** + **prekallikrein** + **HMW kininogen** + **factor XII**

↓

activate XI

↓

activate IX, then add VIII

↓

activate X, then add V

↓

convert **prothrombin** (factor II) to **thrombin**

↓

thrombin then converts **fibrinogen** to **fibrin**

Extrinsic pathway: **tissue factor** (injured cells) + **factor VII**

↓

activate X, then add V

↓

convert **prothrombin** to **thrombin**

↓

thrombin then converts **fibrinogen** to **fibrin**

Prothrombin complex (for intrinsic and extrinsic pathways)
- **X**, **V**, **Ca**, **platelet factor 3**, and **prothrombin**
- Forms on platelets
- Catalyzes the formation of thrombin

Factor X is the convergence point and is common for both paths
Tissue factor pathway inhibitor – inhibits factor X

Fibrin – links platelets together (binds GpIIb/IIIa molecules) to form platelet plug → hemostasis
XIII – helps crosslink fibrin

Thrombin
- **Key to coagulation**
- Converts **fibrinogen** to **fibrin** and fibrin split products
- Activates **factors V** and **VIII**
- Activates **platelets**

NORMAL ANTICOAGULATION

Antithrombin III (AT-III)
- **Key to anticoagulation**
- Binds and inhibits **thrombin**
- Inhibits **factors IX**, **X**, and **XI**
- Heparin activates **AT-III** (up to 1000× normal activity)

Protein C – vitamin K–dependent; degrades **factors V** and **VIII**; degrades **fibrinogen**
Protein S – vitamin K–dependent, protein C cofactor

Fibrinolysis
- **Tissue plasminogen activator** – released from endothelium and converts plasminogen to plasmin
- **Plasmin** – degrades **factors V** and **VIII**, **fibrinogen**, and **fibrin** → lose platelet plug
- **Alpha-2 antiplasmin** – natural inhibitor of plasmin, released from endothelium

Factor VII – shortest half-life
Factors V and **VIII** – labile factors, activity lost in stored blood, activity not lost in FFP
Factor VIII – only factor not synthesized in liver (synthesized in **endothelium**)

Vitamin K–dependent factors – II, VII, IX, and X; proteins C and S
Vitamin K – takes 6 hours to have effect
FFP – effect is immediate and lasts 6 hours
Factor II – prothrombin
Normal half-life – RBCs: 120 days; platelets: 7 days; PMNs: 1–2 days
Prostacyclin (PGI_2)

- From **endothelium**
- Decreases platelet aggregation and promotes vasodilation (antagonistic to TXA_2)

Thromboxane (TXA_2)

- From **platelets**
- Increases platelet aggregation and promotes vasoconstriction
- Triggers release of **calcium** in platelets → exposes **GpIIb/IIIa receptor** and causes platelet-to-platelet binding; platelet-to-collagen binding also occurs (**GpIb receptor**)

COAGULATION FACTORS

Cryoprecipitate – contains highest concentration of **vWF-VIII**; used in von Willebrand's disease and hemophilia A (factor VIII deficiency), also has high levels of fibrinogen
FFP (fresh frozen plasma) – has high levels of all coagulation factors, protein C, protein S, and AT-III
DDAVP and **conjugated estrogens** – cause release of **VIII** and **vWF** from endothelium

COAGULATION MEASUREMENTS

PT – measures II, V, VII, and X; fibrinogen; best for **liver synthetic function**
PTT – measures most factors ***except VII and XIII*** *(thus does not pick up factor VII deficiency);* also measures fibrinogen

- Want **PTT 60–90 sec** for routine anticoagulation

ACT = activated clotting time

- Want **ACT 150–200 sec** for routine anticoagulation, > **460 sec** for cardiopulmonary bypass

INR > 1.5 – relative contraindication to performing surgical procedures
INR > 1.3 – relative contraindication to central line placement, percutaneous needle biopsies, and eye surgery

BLEEDING DISORDERS

Incomplete hemostasis – most common cause of surgical bleeding
von Willebrand's disease

- ***Most common congenital bleeding disorder***

- Types I and II are autosomal dominant; type III is autosomal recessive
- **vWF** links **GpIb receptor** on **platelets** to **collagen**
- PT normal; PTT can be normal or abnormal
- Have long **bleeding time** (ristocetin test)
- **Type I** is **most common** (70% of cases) and often has only mild symptoms
- **Type III** causes the **most severe bleeding**
- **Type I** – reduced quantity of vWF
  - Tx: recombinant VIII:vWF, **DDAVP**, cryoprecipitate
- **Type II** – defect in vWF molecule itself, vWF does not work well
  - Tx: recombinant VIII:vWF, **cryoprecipitate**
- **Type III** – complete vWF deficiency (rare)
  - Tx: recombinant VIII:vWF; **cryoprecipitate**; *(DDAVP will not work)*

Hemophilia A (VIII deficiency)
- Sex-linked recessive
- Need levels 100% pre-op; keep at 80%–100% for 10–14 days after surgery
- **Prolonged PTT** and normal PT
- **Factor VIII** crosses **placenta** → newborns may not bleed at circumcision
- Hemophiliac **joint bleeding** – ***do <u>not</u> aspirate***
  - Tx: ice, keep joint mobile with range of motion exercises, **factor VIII** concentrate or **cryoprecipitate**
- Hemophiliac **epistaxis**, **intracerebral hemorrhage**, or **hematuria**
  - Tx: **recombinant factor VIII** or **cryoprecipitate**

Hemophilia B (IX deficiency) – Christmas disease
- Sex-linked recessive
- Need level 100% pre-op; keep at 30%–40% for 2–3 days after surgery
- **Prolonged PTT** and normal PT
- Tx: **recombinant factor IX** or **FFP**

Factor VII deficiency – **prolonged PT** and normal PTT, bleeding tendency. Tx: **recombinant factor VII** concentrate or **FFP**

Platelet disorders – cause bruising, epistaxis, mucosal bleeding, petechiae, purpura
- **Acquired thrombocytopenia** – can be caused by H_2 blockers, heparin
- **Glanzmann's thrombocytopenia** – <u>GpIIb/IIIa receptor deficiency</u> on platelets (cannot bind to each other)
  - Fibrin normally links the GpIIb/IIIa receptors together
  - Tx: **platelets**
- **Bernard Soulier** – <u>GpIb receptor deficiency</u> on platelets (cannot bind to collagen)
  - vWF normally links GpIb to collagen
  - Tx: **platelets**
- **Uremia** – inhibits platelet function
  - Tx: **hemodialysis** (1st), DDAVP, platelets

Heparin-induced thrombocytopenia (HIT)

- Thrombocytopenia due to **antiplatelet antibodies** (IgG PF4 antibody) results in platelet destruction
- Can also cause platelet aggregation and thrombosis (HIT**T**; **T** = thrombosis)
- Forms a **white clot**
- Can occur with low doses of heparin
- Low-molecular-weight heparin has a decreased risk of causing HIT
- Tx: ***stop heparin***; start **argatroban** (direct thrombin inhibitor) to anticoagulate

Disseminated intravascular coagulation (DIC)
- **Decreased platelets**, **low fibrinogen**, **high fibrin split products**, and **high D-dimer**
- Prolonged PT and prolonged PTT
- Often initiated by **tissue factor**
- Tx: need to treat the underlying cause (eg sepsis)

ASA – stop 7 days before surgery; patients will have prolonged bleeding time
- **Inhibits cyclooxygenase** in platelets and **decreases TXA_2**
- Platelets lack DNA, so they cannot resynthesize cyclooxygenase

Clopidogrel (Plavix) – stop 7 days before surgery; ADP receptor antagonist; Tx: platelets

Coumadin – stop 7 days before surgery, consider starting heparin while Coumadin wears off

Platelets – want them > 50,000 before surgery, > 20,000 after surgery

Prostate surgery – can release **urokinase**, activates plasminogen → thrombolysis
- Tx: **ε-aminocaproic acid** (Amicar)

H and P – best way to predict bleeding risk

Normal circumcision – does not rule out bleeding disorders; can still have clotting factors from mother

Abnormal bleeding with tooth extraction or tonsillectomy – picks up 99% patients with bleeding disorder

Epistaxis – common with vWF deficiency and platelet disorders

Menorrhagia – common with bleeding disorders

HYPERCOAGULABILITY DISORDERS

Present as venous or arterial thrombosis/emboli (eg DVT, PE, stroke)

Factor V Leiden mutation – 30% of spontaneous venous thromboses
- ***Most common congenital hypercoagulability disorder***
- Causes **resistance to activated protein C**; the defect is on **factor V**
- Tx: heparin, warfarin

Hyperhomocysteinemia - Tx: **folic acid** and **B_{12}**

Prothrombin gene defect G20210 A - Tx: heparin, warfarin

Protein C or S deficiency - Tx: heparin, warfarin

Antithrombin III deficiency