HEMATOLOGY · COAGULATION · PHARM

Platelet Aggregation *vs* Clot Formation Pathways

A side-by-side breakdown of **primary hemostasis** (platelet plug) and **secondary hemostasis** (coagulation cascade) — including the drugs that block each step and the NCLEX traps built around them.

DIANE'S NGN NCLEX 2026 VISUAL STUDY LIBRARY · HEMATOLOGY MODULE

Source note: This topic was not present in your uploaded reference notes. Content drawn from standard NGN NCLEX 2026 references — Saunders Comprehensive Review (2026), ATI Pharmacology Made Easy, Lippincott Illustrated Reviews: Pharmacology, and current CDC anticoagulation guidance.

1 Definition & Overview

WHAT HEMOSTASIS IS — AND THE TWO PATHWAYS THAT ACHIEVE IT

Hemostasis is the body's process of stopping bleeding after vascular injury. It happens in **two sequential but overlapping pathways**:

PRIMARY HEMOSTASIS

Platelet Aggregation

- Forms an **unstable platelet plug** within seconds-to-minutes
- "White thrombus" — platelets + von Willebrand factor (vWF)
- Quick patch for small vessel injuries
- Blocked by **antiplatelets** (aspirin, clopidogrel)

SECONDARY HEMOSTASIS

Clot Formation

- Coagulation cascade produces **fibrin mesh** over minutes-to-hours
- "Red thrombus" — fibrin + trapped RBCs stabilizes the plug
- Required for larger vessels & durable hemostasis
- Blocked by **anticoagulants** (heparin, warfarin, DOACs)

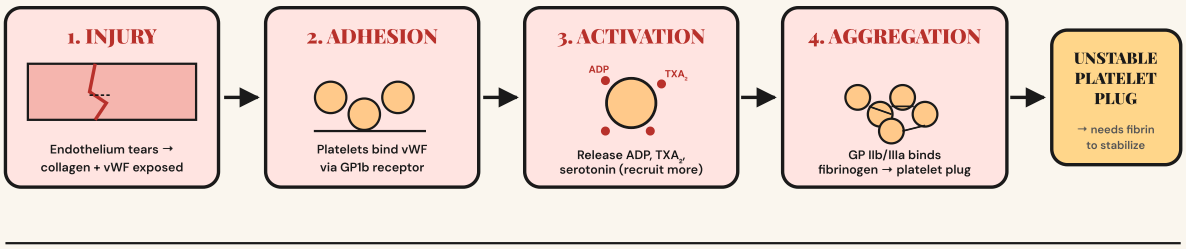
The two pathways work together: the platelet plug is the bandage; fibrin is the stitching.

2 Pathophysiology — Step by Step

VISUAL MECHANISM OF BOTH PATHWAYS

Primary Hemostasis · Platelet Plug

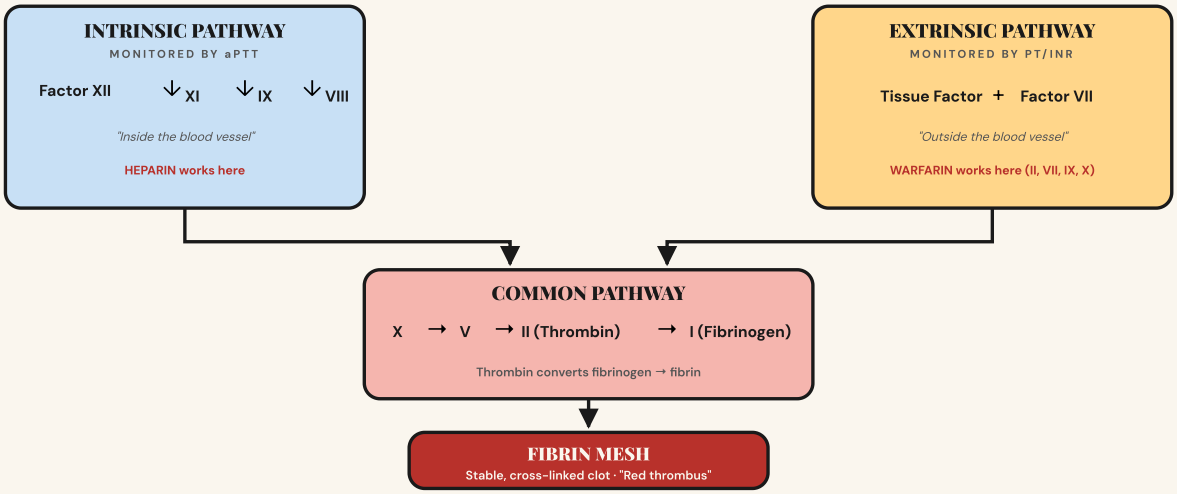
VASCULAR INJURY → ADHESION → ACTIVATION → AGGREGATION



Primary hemostasis is FAST — first responder to vessel injury

Secondary Hemostasis · Coagulation Cascade

INTRINSIC + EXTRINSIC → COMMON PATHWAY → FIBRIN



BIG-PICTURE TAKEAWAY

Platelets form the **fast plug**. **Fibrin** is the **durable cement**. If either system fails, the patient bleeds — but the type of bleeding looks different, and that's how NCLEX writes questions.

3 Signs & Symptoms of Failure

PLATELET PROBLEM VS COAGULATION FACTOR PROBLEM

PLATELET PATHWAY FAILURE

Surface / Mucosal Bleeding

- **Petechiae** — pinpoint red dots (classic platelet sign)
- **Purpura** & easy **bruising**
- **Epistaxis** (nosebleeds)
- **Gingival bleeding** (gums)
- Heavy **menorrhagia**
- Prolonged bleeding from small cuts
- *Bleeding starts IMMEDIATELY*

Think: thrombocytopenia, ITP, aspirin toxicity, vWD

COAGULATION PATHWAY FAILURE

Deep / Delayed Bleeding

- **Hemarthrosis** — joint bleeds (hallmark of hemophilia)
- Deep muscle **hematomas**
- **Retroperitoneal** & intracranial bleeds
- Hematuria, GI bleeding (melena, hematemesis)
- Bleeding from surgical sites hours later
- *Bleeding is DELAYED (hours after injury)*

Think: hemophilia, warfarin/heparin excess, liver failure, DIC

RED FLAG BLEEDING — NOTIFY HCP IMMEDIATELY

- **Sudden severe headache, confusion, or unequal pupils** → intracranial hemorrhage
- **Frank red blood per rectum, melena, hematemesis** → GI bleed (often massive)
- **Flank pain + hypotension + falling H&H** → retroperitoneal bleed
- **Platelets < 20,000/mm³** → spontaneous bleeding risk
- **INR > 5 (or > 4 with bleeding)** → reverse warfarin urgently
- **Platelet drop > 50% from baseline on heparin** → suspect HIT, stop heparin

4 Priority Nursing Interventions

ABCS · SAFETY · PRIORITIZATION

- **A — Airway:** Assess for oropharyngeal bleeding or hematoma compromising the airway; have suction ready.
- **B — Breathing:** Monitor for hemoptysis or pulmonary hemorrhage; auscultate breath sounds; pulse ox.
- **C — Circulation:** Monitor vital signs (watch for **tachycardia + hypotension** = hidden bleed), trend H&H, assess capillary refill and peripheral perfusion.
- **Assess** ALL sites of potential bleeding: skin (bruising, petechiae), mucous membranes (gums, nose), urine (hematuria), stool (melena, occult), emesis, IV/surgical sites.
- **Monitor labs:** PT/INR (warfarin), aPTT (heparin), platelet count, H&H, fibrinogen, D-dimer.
- **Implement bleeding precautions:** soft-bristle toothbrush, electric razor, no IM injections when possible, hold firm pressure ≥10 minutes after venipuncture, no rectal temps/suppositories, fall prevention.
- **Hold** antiplatelets/anticoagulants if active bleeding — notify HCP before next dose.
- **Type and crossmatch** if active bleeding or surgical risk; have blood products available.
- **Administer reversal agents** per order: **protamine sulfate** (heparin), **vitamin K / FFP / PCC** (warfarin), **idarucizumab** (dabigatran).
- **Avoid:** aspirin, NSAIDs, herbal supplements (ginkgo, garlic, ginger, ginseng) — all increase bleeding.

5 Pharmacology — Blocking Each Step

ANTIPLATELETS · ANTICOAGULANTS · THROMBOLYTICS

Antiplatelets

TARGETS PRIMARY HEMOSTASIS

DRUG	MECHANISM	KEY CONSIDERATIONS
Aspirin (ASA)	Irreversibly inhibits **COX-1** → ↓ thromboxane A_2 → ↓ aggregation	Effect lasts **7–10 days** (lifespan of platelet); hold 5–7 days pre-op; watch for tinnitus (salicylism)
Clopidogrel (Plavix)	Blocks **ADP** P2Y12 receptor on platelets	Used post-stent/MI; hold 5 days pre-op; watch for TTP (rare)
Ticagrelor (Brilinta), Prasugrel	Newer ADP blockers	Used in ACS; ticagrelor causes **dyspnea** (~15%)
Abciximab (ReoPro), Eptifibatide (Integrilin)	Block **GP IIb/IIIa** receptor — final step of aggregation	IV only, used in cath lab; **high bleeding risk**; monitor platelets & ACT

Anticoagulants

TARGETS SECONDARY HEMOSTASIS

DRUG	MECHANISM / PATHWAY	MONITOR	ANTIDOTE
Heparin (UFH) IV/SQ	↑ Antithrombin III → inactivates thrombin (IIa) & Xa · *Intrinsic*	**aPTT** therapeutic 1.5–2.5× control (~46–70 sec)	**Protamine sulfate**
Enoxaparin (Lovenox) SQ — LMWH	Selective Factor **Xa** inhibition	Anti-Xa level (no routine aPTT)	Protamine (partial reversal)
Warfarin (Coumadin) PO	Vitamin K antagonist → ↓ factors **II, VII, IX, X** · *Extrinsic*	**PT / INR** therapeutic INR 2–3 (2.5–3.5 mech valve)	**Vitamin K**, FFP, PCC
Rivaroxaban (Xarelto), Apixaban (Eliquis) — DOACs	Direct **Factor Xa** inhibitors	No routine labs	**Andexanet alfa**
Dabigatran (Pradaxa) — DOAC	Direct **thrombin (IIa)** inhibitor	No routine labs	**Idarucizumab (Praxbind)**

Thrombolytics ("Clot Busters")

DISSOLVES EXISTING CLOT

DRUG	MECHANISM	CRITICAL NURSING CONSIDERATIONS
Alteplase (tPA)	Converts plasminogen → plasmin → lyses fibrin	**Strict time windows:** Ischemic stroke ≤ 3 hrs (up to 4.5); STEMI ≤ 12 hrs; PE per protocol. **Major bleeding** is the chief risk. Screen for absolute contraindications: recent surgery (<14 d), active bleeding, hx of hemorrhagic stroke, BP > 185/110, recent head trauma.

6 NCLEX Test Traps

WHERE STUDENTS LOSE POINTS

✗ "Aspirin is an anticoagulant."

✓ Aspirin is an antiplatelet — it blocks aggregation, not the coagulation cascade.

✗ "Monitor PT/INR for heparin."

✓ PT/INR is for warfarin. Heparin → aPTT. (Think: PTT = "Play with Heparin")

✗ "Give vitamin K to reverse heparin."

✓ Vitamin K reverses warfarin. Heparin is reversed with protamine sulfate.

✗ "Stop heparin if platelets drop a little."

✓ A drop of > 50% from baseline = HIT — stop heparin immediately and never give it again.

✗ "Therapeutic INR is the same for everyone."

✓ Most conditions: 2–3. Mechanical valve: 2.5–3.5. Above 5 = bleed risk.

✗ "Petechiae = coagulation factor problem."

✓ Petechiae are a platelet sign. Joint bleeds = factor problem.

✗ "tPA can be given any time after symptom onset."

✓ Strict windows — ischemic stroke ≤ 3 (up to 4.5) hours. Outside the window = harm.

✗ "Hold pressure for 1–2 minutes after a stick on a patient on warfarin."

✓ Hold firm pressure for at least 10 minutes — longer if arterial.

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Patient & Family Education

HOME SAFETY ON ANTIPLATELETS / ANTICOAGULANTS

- Use a **soft-bristle toothbrush** and an **electric razor** — no straight razors, no flossing if gums bleed easily.
- **Wear a medical alert bracelet** identifying your blood thinner.
- **Fall prevention**: remove throw rugs, use night lights, non-slip footwear, install grab bars.
- Report immediately: **black tarry stools, red/pink urine, coffee-ground emesis, severe headache, confusion, unexplained bruising, prolonged nosebleed**.
- Tell **every** provider (including dentists) that you're on a blood thinner — *before* any procedure.
- **Warfarin patients:** keep **vitamin K intake CONSISTENT** day-to-day (don't suddenly increase or decrease leafy greens — kale, spinach, broccoli, Brussels sprouts). It's about consistency, not avoidance.
- Avoid OTC **NSAIDs, aspirin**, and herbal supplements (*ginkgo, garlic, ginger, ginseng, St. John's wort*) unless approved by HCP.
- Limit **alcohol** — potentiates warfarin and increases GI bleed risk.
- Keep all **follow-up lab appointments** (INR for warfarin); never skip doses or double up if missed.
- **Pregnancy:** warfarin is teratogenic — use heparin/LMWH instead; notify HCP if planning pregnancy.

8 Memory Boosters

PATTERN RECOGNITION FOR THE TEST

1972

Warfarin inhibits vitamin K–dependent factors **II, VII, IX, X** — read it as "**1972**." Easy to recall under pressure.

PT Boats Sail in the Sea

PT goes with the "**sea**" in Coumadin (warfarin). **PTT** goes with **Heparin**. Or simply: *Pl*a*T*elets, *PT* for warfarin pills.

Platelets Plug, Fibrin Fixes

Primary hemostasis = the **temporary plug**. Secondary = the **fibrin fix** that stabilizes it. Two pathways, one goal.

BLEEDS

Bruising · **L**ethargy · **E**pistaxis · **E**cchymosis · **D**ark stools · **S**welling (joint) — assessment checklist for any patient on a blood thinner.

Protamine ↔ Heparin

Both start with letters near the beginning of the alphabet — pair them. **Vitamin K ↔ Warfarin** (both have a "K"-sounding letter). Mismatched antidotes are a classic NCLEX distractor.

"Petechiae = Platelet"

Both start with **P**. If you see pinpoint dots, the platelet pathway is the problem — not the coagulation cascade.

9 NCJMM Clinical Judgment Scenario

NEXT-GEN NCLEX · 6-STEP MODEL

CASE SCENARIO

Patient: 68-year-old female with history of atrial fibrillation, on warfarin 5 mg daily for 3 years. Presents to the ED with sudden severe headache, slurred speech, and confusion that began 45 minutes ago. Husband reports she started a 10-day course of *trimethoprim-sulfamethoxazole (Bactrim)* 5 days ago for a UTI.

Vitals: BP 168/94 · HR 102 · RR 22 · SpO_2 96% · GCS 13

Labs: INR **8.4** (baseline 2.4) · Hgb 11.2 · Platelets 198K · aPTT 32 sec

1 Recognize Cues

STEP 1

Critical cues: **INR 8.4 (supratherapeutic),** new neurological deficits (slurred speech, confusion, severe headache, GCS 13), **elevated BP**, **recent Bactrim initiation** (potent warfarin potentiator — displaces warfarin from albumin and inhibits CYP2C9), age > 65.

2 Analyze Cues

STEP 2

The combination of **focal neurologic deficits + severe headache + INR 8.4 + warfarin–Bactrim interaction** points strongly to **intracranial hemorrhage**. The supratherapeutic INR alone places her at imminent risk of catastrophic bleeding anywhere in the body. Bactrim is a classic NCLEX-tested warfarin interaction.

3 Prioritize Hypotheses

STEP 3

#1 Intracranial hemorrhage (life-threatening, time-critical) → **#2** Severe over-anticoagulation from drug interaction → **#3** Acute ischemic stroke (less likely given INR and clinical pattern, but must be ruled out before any reversal decision in some protocols).

4 Generate Solutions

STEP 4

Hold warfarin immediately · STAT non-contrast **CT head** · Reverse anticoagulation with **IV vitamin K + 4-factor PCC (or FFP)** per protocol · Continuous neuro checks · BP control (avoid aggressive drops) · NPO · IV access × 2 large bore · Type & crossmatch · Neurosurgery consult.

5 Take Action

STEP 5

Hold warfarin · Administer **vitamin K 10 mg IV** per order · Prepare **4-factor PCC** · Transport STAT to CT · Neuro checks q15 min · Maintain head of bed at 30° · Monitor for seizure · Insert second large-bore IV ·

Strict bleeding precautions · Notify family.

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Evaluate Outcomes

STEP 6

Effective: INR trending toward normal (≤ 1.5 within hours of PCC), GCS stable or improving, no new neurologic deficits, BP controlled, no expansion on repeat imaging. **Ineffective / escalate:** GCS declining, new lateralizing signs, pupillary changes, INR remaining elevated → repeat reversal agents, neurosurgical intervention.