

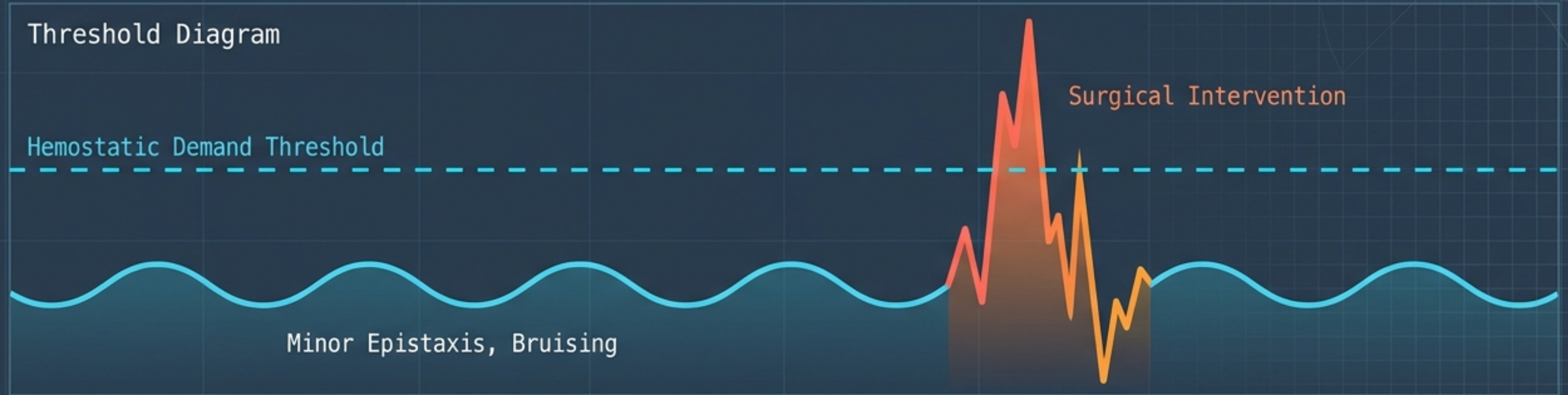
A Perioperative Blueprint for Surgery and Procedures in VWD

Surgery is where uncertainty becomes practical.
And consequential.



The Core Premise: The Hemostatic Stress Test

Surgery does not create VWD; it exposes the limits of compensatory hemostasis. The body must suddenly maintain clot stability across exposed microvasculature, sustained tissue disruption, local fibrinolysis, and mechanical stress.



1

Type 1 VWD

is not a perioperative plan.

2

Low VWF

is not a perioperative plan.

3

Responds to desmopressin

is not always a perioperative plan.

The goal is not to react to bleeding. The goal is planned hemostasis under stress.

The Planning Equation: Challenge vs. Reserve

The Procedural Challenge (The Stressor)

1. Procedure type & anatomic site
2. Mucosal involvement & fibrinolytic environment
3. Compressibility of the bleeding site
4. Duration of tissue healing & rehabilitation



The Patient Reserve (The Biology)

1. VWD subtype (qualitative vs. quantitative)
2. Baseline VWF activity, VWF antigen, and FVIII levels
3. Bleeding history and prior surgical challenges
4. Documented desmopressin (DDAVP) response

Procedural planning begins with the procedure, not the laboratory value.
The plan must perfectly balance the challenge against the reserve.

Deconstructing the Challenge: Tissue Biology Matters



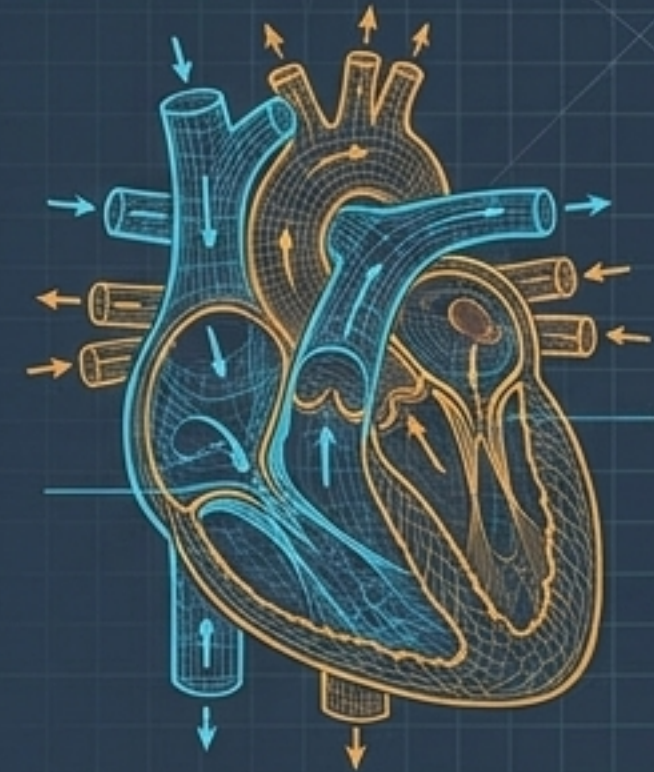
Mucosal Tissue (Oral, Nasal, Gynecologic)

-  Environment: Wet, mobile, exposed.
-  Mechanism of Failure: Highly fibrinolytic. Clots form but dissolve or dislodge prematurely.
-  Clinical Need: Clot protection (Antifibrinolytics) is as critical as clot formation.



Orthopedic Tissue (Joints, Deep Muscle)

-  Environment: Deep tissue, mechanical load.
-  Mechanism of Failure: Repetitive mechanical stress during postoperative movement and rehabilitation.
-  Clinical Need: Extended hemostatic coverage tailored to rehab milestones.



Cardiac/Vascular Tissue

-  Environment: High shear stress, extracorporeal circuits.
-  Mechanism of Failure: High shear alters VWF biology, causing loss of high-molecular-weight multimers (acquired VWD-like state).
-  Clinical Need: Sustained, specialized VWF/FVIII monitoring.

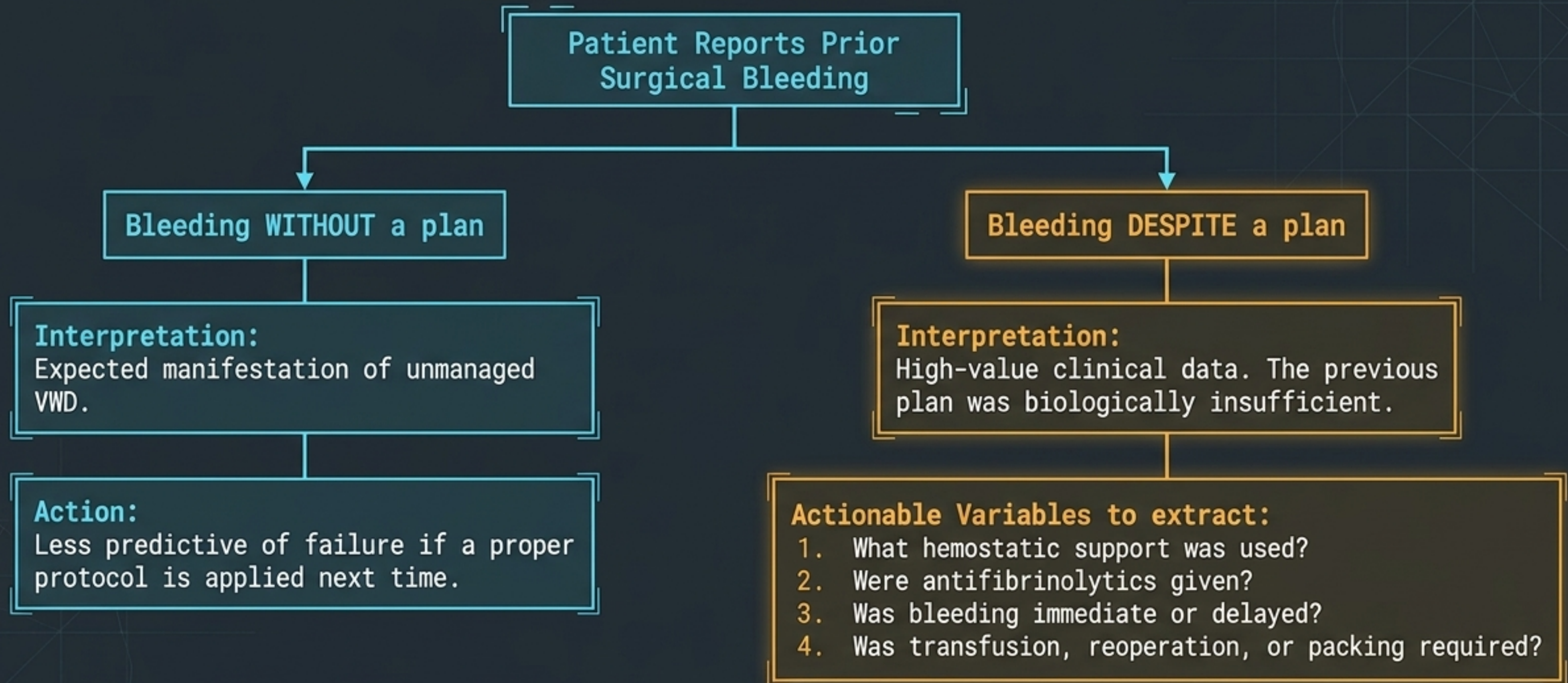
Re-evaluating 'Major vs. Minor' Surgery

Traditional View (Generic Labels)	Expert View (Mechanistic Variables)
Minor Outpatient, small incisions (e.g., simple dental extraction, superficial biopsy).	☐ Is the site inherently mucosal or highly fibrinolytic? ☐ Is the bleeding source mechanically compressible? ☐ Is delayed postoperative bleeding common for this tissue? ☐ Can local hemostatic measures be effectively applied?
Major Large body cavities, joint interventions, life-threatening sites.	

The Dental Exception

A simple extraction may be 'minor.' An impacted third-molar extraction carries higher bleeding risk and demands a 'major' protocol. 'Minor' does not automatically mean 'low risk.'

Decoding Patient Bleeding History

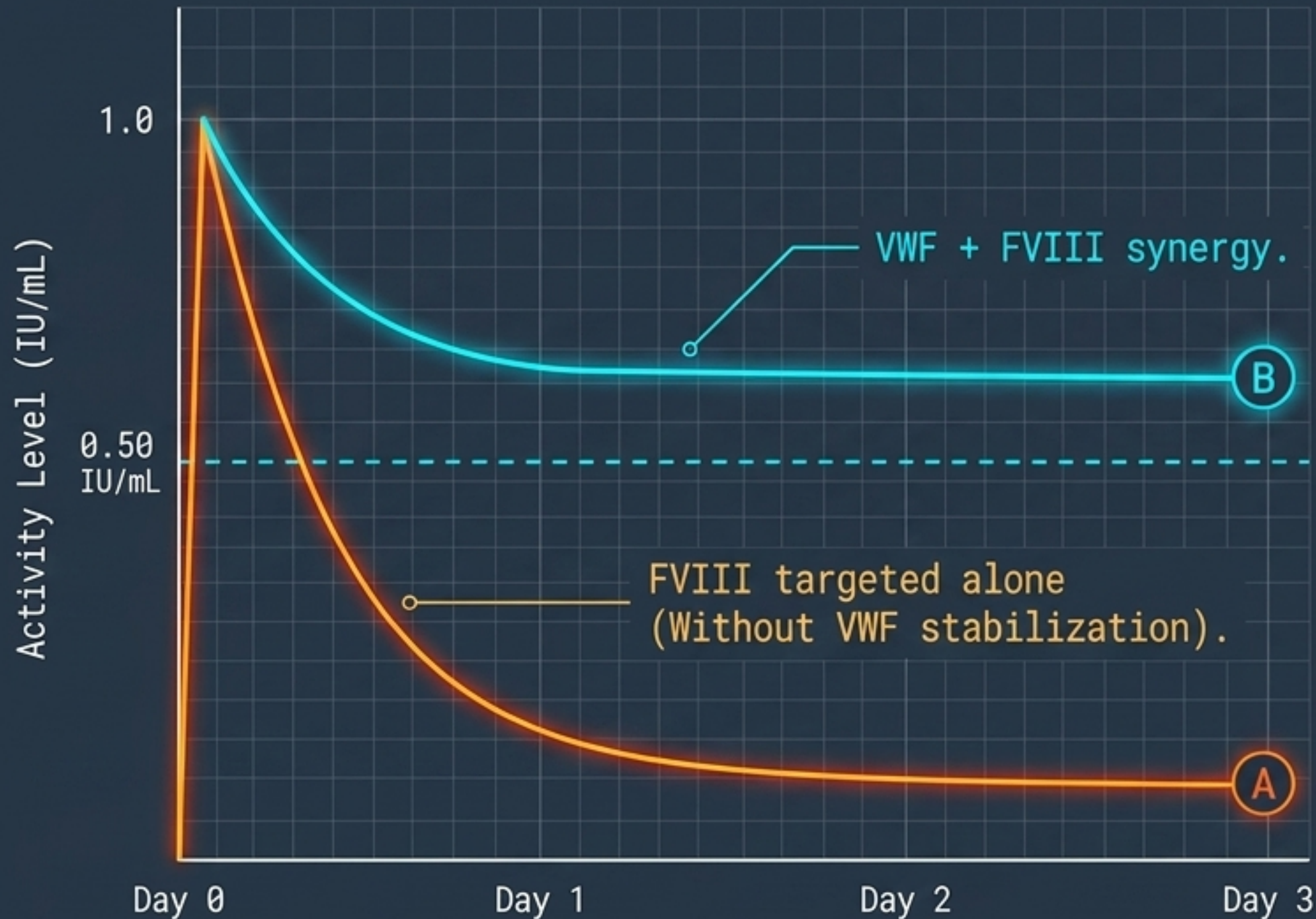


No bleeding after a minor procedure does not guarantee safety for major surgery.
The question is not "Did the patient bleed?" but "What does the event teach us about the next challenge?"

The Therapeutic Toolkit: Systemic Interventions

Desmopressin (DDAVP)	VWF Replacement	Tranexamic Acid (TXA)
Mechanism: Releases endogenous VWF/FVIII stores.	Mechanism: Exogenous restoration of VWF (and FVIII).	Mechanism: Antifibrinolytic; prevents clot dissolution.
Ideal Fit: Short/limited procedures in known, durable responders.	Ideal Fit: Type 3, Type 2 variants, major surgery, severe baseline deficiency, DDAVP non-responders.	Ideal Fit: Crucial for mucosal procedures (dental, ENT, GYN).
Contraindications: Ineffective in Type 3 (no reserve). Generally contraindicated in Type 2B (exacerbates thrombocytopenia). Not for major, critical-site surgery.	Clinical Note: Must match VWF and FVIII kinetics to the operative timeline.	Contraindications: Use cautiously/avoid in gross hematuria or upper urinary tract bleeding (risk of clot obstruction).

The Mechanistic Target: Why FVIII Alone Fails



FVIII alone does not correct the primary VWF defect.

VWF serves two distinct, critical roles:

1. Mediates primary platelet adhesion to the vessel wall.
2. Acts as the biological stabilizer for endogenous FVIII.

Patients with Type 2 or Type 3 VWD will not achieve adequate hemostasis if only FVIII is maintained.

Guideline Rule: For major surgery, maintain both VWF and FVIII activity at ≥ 0.50 IU/mL for at least 3 days.

The Major Surgery Protocol: Sustained Coverage

Pre-Op	Intra-Op	Early Post-Op (Days 1-3)	Late Post-Op (Days 4+)
1.50 IU/mL			
Safe Zone	Dashboard Metrics: Minimum Target: VWF and FVIII activity ≥ 0.50 IU/mL. Minimum Duration: At least 3 days (individualized based on rehab and tissue healing).		
0.50 IU/mL			

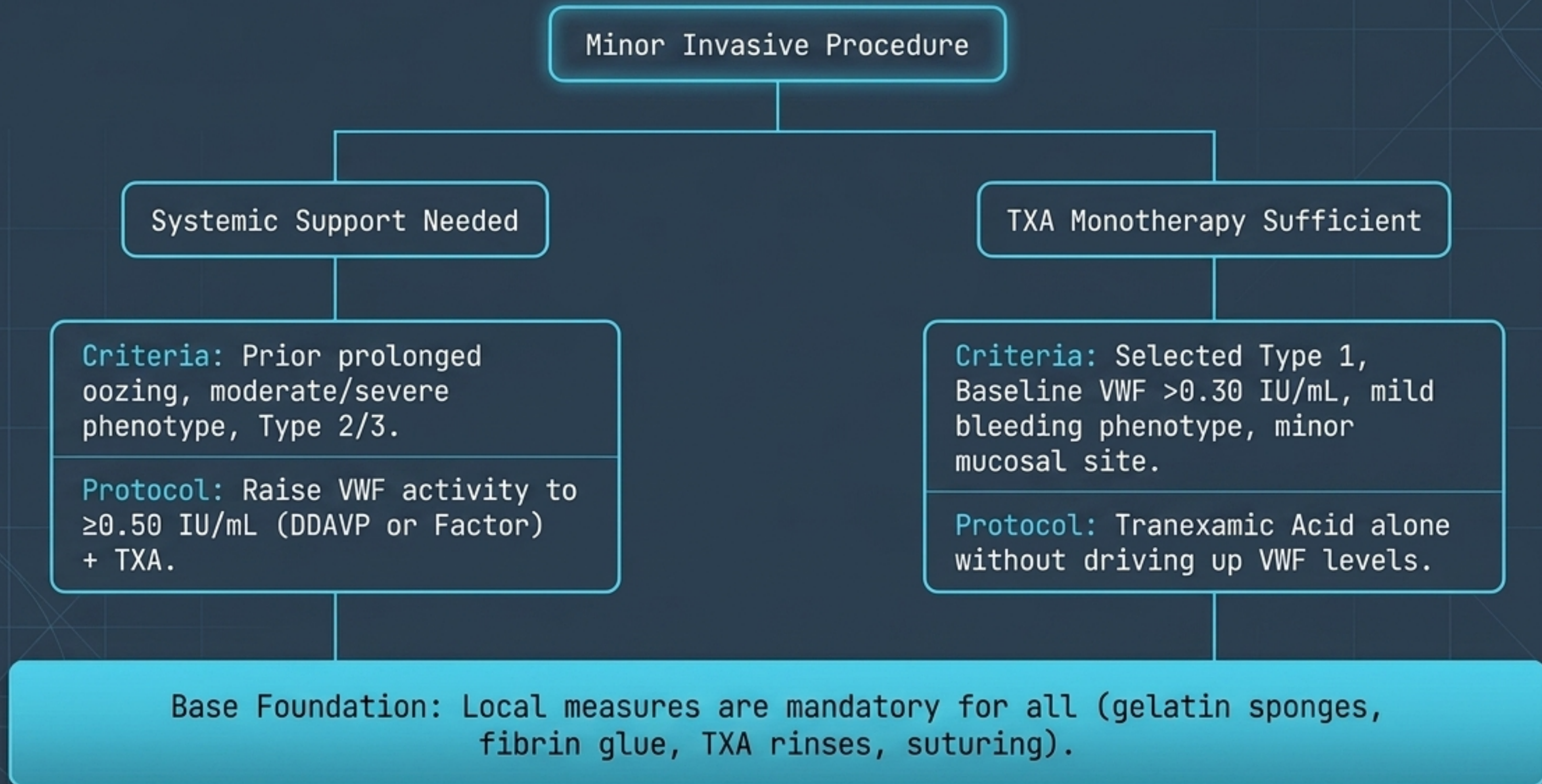
Warning Panel (Thrombosis Risk)

- VWD patients bleed, but aggressive perioperative treatment creates a new pro-thrombotic physiology.
- Risk Modifiers: Older age, immobility, orthopedic/cancer surgery, cardiovascular disease.

The Overcorrection Limit

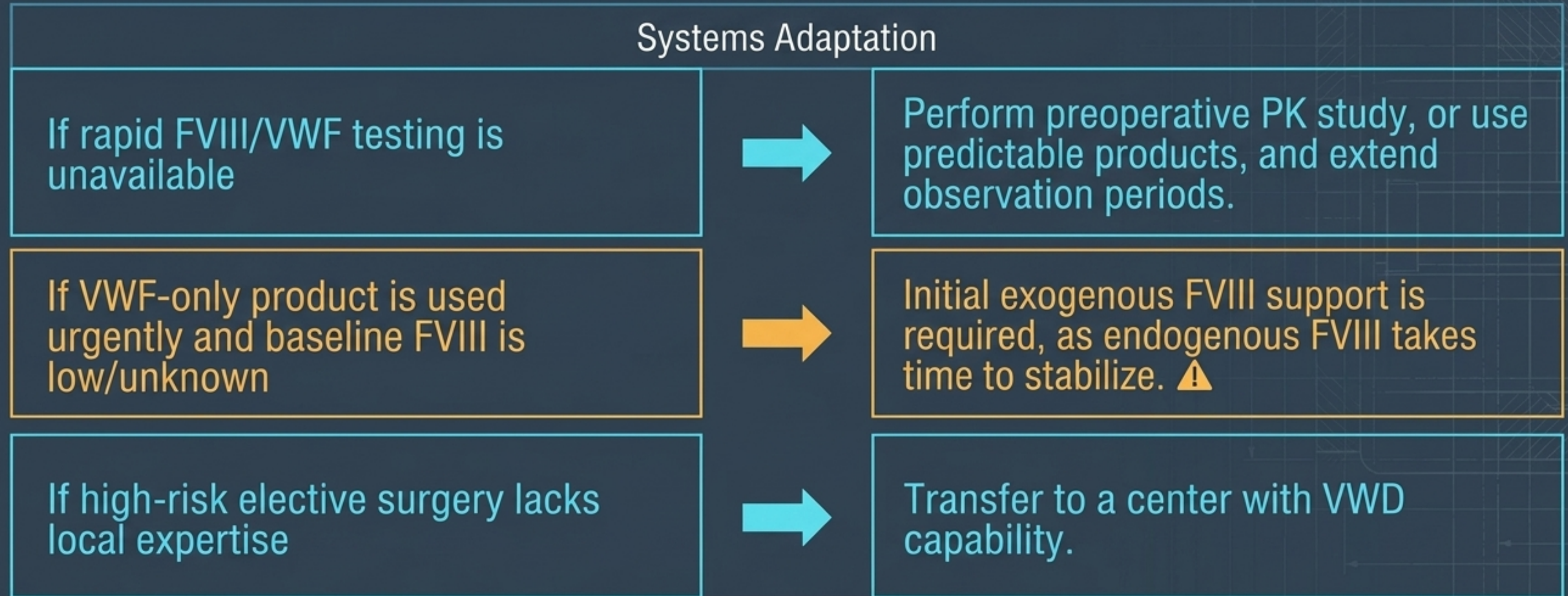
Avoid combining extended VWF/FVIII levels >1.50 IU/mL with extended TXA use in high-risk patients. Calibrate hemostasis to avoid both extremes.

The Minor Surgery Protocol: Proportional Coverage



Adapting to Uncertainty (Emergencies & Limited Monitoring)

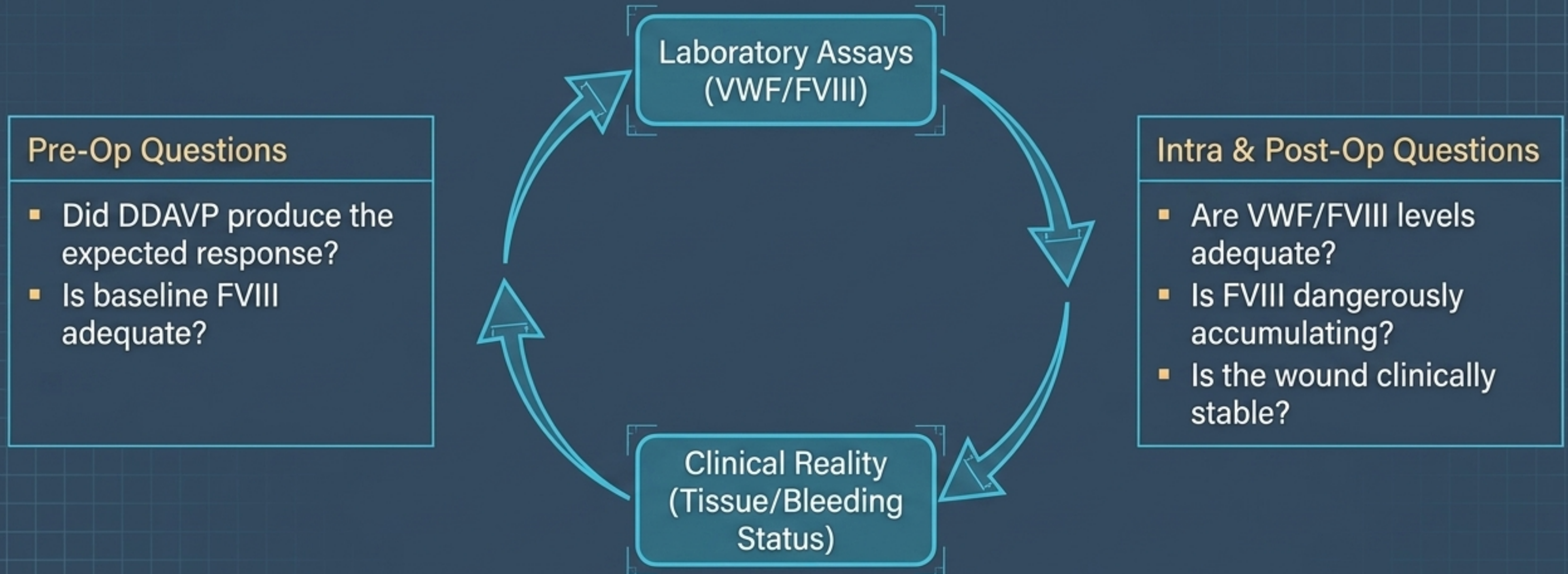
Elective procedures allow for perfect diagnostic precision. Emergency procedures prioritize immediate hemostatic coverage. Do not wait for perfect classification if urgent coverage is required.



Rule of Thumb: A plan that cannot be monitored must be more conservative when the stakes are high.

The Monitoring Loop: Knowing the Plan is Working

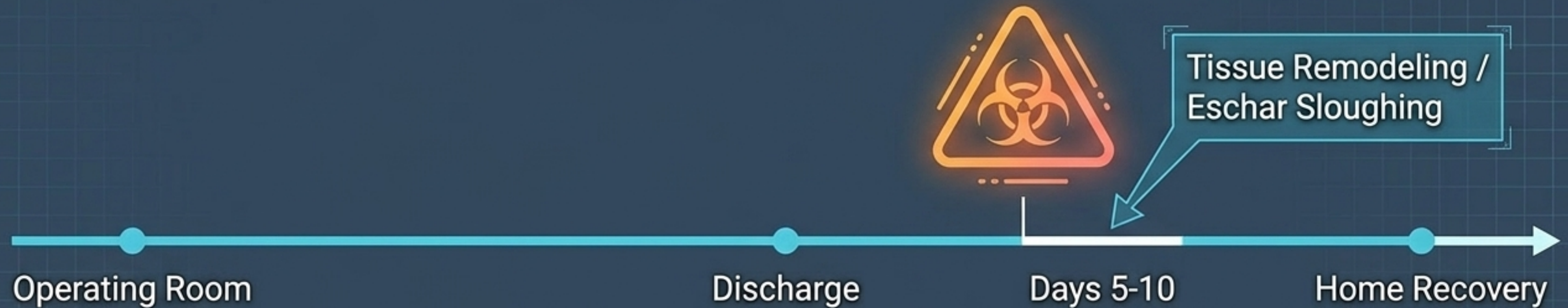
Core Principle: Monitoring must answer a specific clinical decision. Monitoring without a linked decision has limited value.



System Note: Monitoring is a systems issue, not just a lab issue. A safe protocol requires the institutional ability to verify if the plan is holding under stress.

The Discharge Problem: Surviving the Recovery

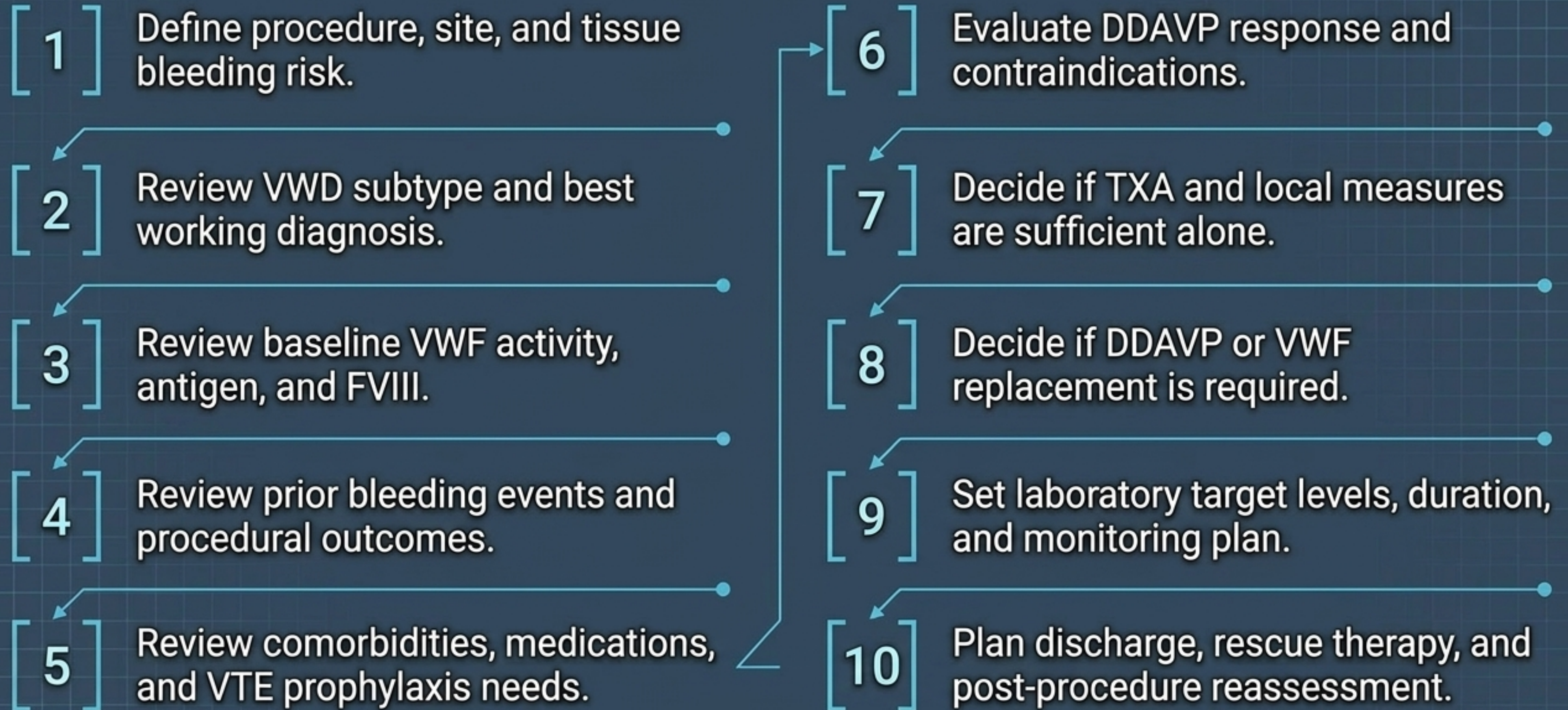
Delayed bleeding is an inherent part of procedural risk in VWD, not a random complication—especially in oral, nasal, tonsillar, and GYN procedures. A perioperative plan must survive the discharge summary.



The VWD Discharge Checklist

Exact duration of home antifibrinolytic therapy.	Clear definitions of expected vs. abnormal bleeding.
Specific rescue instructions (Who to call, where to go, product needed).	Confirmation that the local ED has the treatment plan.

Synthesis: The 10-Step Practical Planning Sequence



Application: The Pediatric Orthopedic Case

The Reserve - Patient Profile

Age/Gender: 10-year-old boy.

Diagnosis: Type 3 VWD.

Lab Baselines: Undetectable VWF,
FVIII 9 IU/dL.

Challenge: First major orthopedic surgery (deep tissue, major dissection, prolonged rehab).



The Execution – Tactical Breakdown

Why DDAVP fails here: Type 3 indicates zero endogenous reserve to release.

The Early Need: Requires **exogenous VWF plus FVIII** coverage **immediately (FVIII is deeply deficient)**.

The Long Game: Orthopedic rehab extends the hemostatic vulnerability window. **Target >0.50 IU/mL** must be maintained into the mechanical stress phase, closely monitored to avoid thrombotic overcorrection.

Final Takeaway: Peri-procedural VWD care is not a formula. It is planned hemostasis under stress.