Antithrombotic Literature Updates

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Anticoagulation FORUM
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VTE in Pregnancy: Pathophysiology, Diagnostic Algorithms, and Therapeutic Approaches

Background: Pregnancy increases VTE risk, including deep vein thrombosis (DVT) and pulmonary embolism (PE), 4-to 5-fold through all three components of Virchow's triad. VTE is a leading cause of maternal morbidity and mortality during pregnancy and postpartum. This narrative review outlines treatment considerations specific to VTE in pregnancy.

Risk stratification and prophylaxis: Screen women for personal/family history of prior VTE and thrombophilia before conception or early in pregnancy and evaluate for consideration of VTE prophylaxis. See this Antithrombotic Clinical Guidance on prophylaxis strategies.

Diagnosis: VTE diagnosis in pregnancy is challenging, as VTE symptoms overlap with normal pregnancy physiology. Use pregnancy-specific tools, including the LEFt rule (for DVT) and the pregnancy-adapted YEARS algorithm (for PE), combined with selective D-dimer testing, and stepwise imaging (compression US for DVT; CTPA for PE).

Treatment: **Low molecular weight heparin (LMWH)** is preferred, as it does not cross the placenta, has no teratogenic effects, and is minimally excreted in breast milk. **Unfractionated heparin (UFH)** via anti-Xa monitoring (preferably) is reserved for severe renal impairment, weight >150 kg, or hemodynamically unstable PE requiring rapid reversal. **VKAs & DOACs** are contraindicated due to teratogenicity. **Inferior Vena Cava (IVC) filters** are reserved for high-risk patients, and *suprarenal placement* is preferred in pregnancy to avoid compression by the gravid uterus. See this Antithrombotic Clinical Guidance on VTE treatment in pregnancy.

Peripartum Management: Discontinue therapeutic LMWH ≥24 hours before planned delivery/neuraxial anesthesia. Discontinue UFH 4-6 hours before delivery.

Postpartum Management: Continue anticoagulation for 6 weeks to 6 months, with consideration to transition from LMWH to warfarin (target INR 2.0–3.0) with ≥ 4-5 days of LMWH bridge.

Angiology. 2026 May 2: 33197261443810.

Discontinuation of Oral Anticoagulation After Successful Atrial Fibrillation Ablation

Background: Current atrial fibrillation (AF) guidelines recommend oral anticoagulation (OAC) for at least 2-3 months (ESC; AHA/ACC/HRS) after catheter ablation (CA) and long-term therapy based on CHA_2DS_2VASc stroke risk rather than rhythm status. Asymptomatic recurrence of AF creates uncertainty regarding OAC discontinuation.

Evidence: Recent trials such as OCEAN (n=1,284) and ALONE-AF (n=840), suggest OAC discontinuation in low-risk patients ($CHA_2DS_2VASc \leq 2$) who maintain ≥12 months of sinus rhythm, with similar thromboembolic and fewer major bleeding events. A 2026 meta-analysis (271,808 patients) found lower bleed risk (OR 0.35, P<0.01) without increased thromboembolism; but higher thromboembolic risk when discontinuing OAC in patients with $CHA_2DS_2VASc > 2$.

Rapid Takeaways:

- Continue OAC in higher-risk patients (e.g., CHA_2DS_2-VASc >2, left atrial enlargement, reduced ejection fraction, atrial fibrosis, systemic inflammation, or prior stroke).
- Silent AF is common; intermittent monitoring and personal devices may miss recurrences, making continuous rhythm monitoring important when considering OAC cessation.
- OAC remains recommended during the first 2-3 months after ablation because of procedure-related thrombotic risk.
- Existing data have limited generalizability, as trials primarily enrolled low-risk patients and excluded newer technologies, such as pulsed-field ablation.
- Emerging approaches, including implantable monitor-guided and episode-triggered ("pill-in-the-pocket") anticoagulation, remain investigational (REACT-AF, RESPOND-AF).

Med Sci Monit. 2026 May 8:32:e952757

Critical Review of International Clinical Practice Guidelines Recommendations for Prevention, Diagnosis, and Management of Central Venous Catheter Thrombosis

Section 2: Diagnostic Approach

Clinical Scenario	Recommendation / Action
POD 1-4 thrombocytopenia	Do not use 4T score (4Ts)
POD 0-4 + new thrombosis + UFH 5-10 days pre-op	Order HIT immunoassay (no 4Ts). Early HIT.
POD 5-30 + biphasic PLT fall ≥30% or PLT <100K with ≥30% fall on/after POD5, or new thrombosis	Calculate 4Ts to assess HIT probability.
4Ts ≤3 (Low probability)	Continue heparin; do not test or treat for HIT.
4Ts ≥4 (intermediate/high probability)	Stop heparin and send immunoassay.
Intermediate 4T (4-5) + no thrombosis	Reasonable to defer non-heparin AC until assay results.
Negative immunoassay (-)	HIT ruled out; no further testing unless with compelling clinical or laboratory features.
Positive immunoassay (+)	Obtain functional assay (e.g., SRA).
Weak ELISA (i.e., OD 0.4-0.99) + no thrombosis	Reasonable to await functional assay before starting non-heparin AC.
High 4Ts, ELISA ≥1.0, positive rapid assay	Start therapeutic non-heparin AC and run functional assay.

Section 4: Management of Patients with HIT Before Cardiac Surgery

Clinical Scenario	Recommendation / Action
History of HIT; non-urgent surgery	Obtain immunoassay to determine antibody (Ab) persistence.
Confirmed HIT; elective surgery	Delay surgery until functional assay negative.
Urgent/emergent surgery + positive functional assay	Use bivalirudin for CPB or UFH with IVIg/TPE pre-CPB.
History HIT + functional assay negative	Heparin acceptable intraoperatively, even if immunoassay remains positive.

AC = Anticoagulation, CPB = Cardiopulmonary Bypass, ELISA = Enzyme-linked Immunosorbent Assay, HIT = Heparin Induced-Thrombocytopenia, IVIg = intravenous immunoglobulin, POD = Post-Operative Day, SRA = Serotonin Release Assay, TPE = Therapeutic Plasma Exchange, UFH = Unfractionated Heparin

See full guideline for further discussion of epidemiology, treatment, and ECMO/temporary mechanical circulatory support considerations and HIT.

J Thorac Cardiovasc Surg. 2026 May 4:S0022-5223(26)00889-5.

How to Monitor and Manage Unfractionated Heparin in Practice (Part 1): Guidance from the SSC of the ISTH

Pre-Analytical Issues

- Sample handling: Centrifuge blood within 1 hour of collection, and test within 10 minutes of centrifugation (optimal) or up to 1 hour (acceptable). Standard 3.2% (109 mM) buffered trisodium citrate tubes are suggested.

Analytical Issues

- Assay selection: Anti-Xa assays (without dextran sulfate and without added antithrombin) are preferred over activated partial thromboplastin time (aPTT) for UFH monitoring due to less biologic and lab interference. If aPTT is used, each institution should establish a local heparin therapeutic range. Obtain a baseline aPTT before UFH initiation; if prolonged above the local normal range, use anti-Xa monitoring. Avoid routinely running aPTT and anti-Xa in parallel, as there is no guidance on managing discordant results.

Clinical Issues

- Therapeutic target: An anti-Xa level of **0.30-0.70 U/mL** for acute venous (VTE) or arterial thromboembolism, and mechanical valve (MV) bridging is suggested. For high-risk MV patients (e.g. mitral position, 1st-generation valves), target the upper end of this range. Robust outcome-based validation remains limited.
- Dosing initiation: Use a **weight-based nomogram**, including an initial bolus, to achieve therapeutic levels within 24 hours, as failure to reach the therapeutic threshold early is associated with increased recurrent VTE risk. No single nomogram is endorsed over another due to insufficient clinical event data.
- Heparin resistance: The clinical benefit of antithrombin administration in acquired deficiency has not been demonstrated and may increase bleeding risk.

J Thromb Haemost. 2026 May 20: S1538-7836(26)00336-3

Efficacy and Safety of DOACs Versus Traditional Anticoagulants in Cirrhotic Patients With VTE and Atrial Fibrillation: A Systematic Review and Meta analysis

Background: Anticoagulation in cirrhosis has traditionally relied on vitamin K antagonists (VKAs) and low molecular weight heparins (LMWH). Patients with advanced liver disease were excluded from major direct oral anticoagulant (DOAC) trials, evidence is largely derived from DOAC observational studies.

Study Design: Systematic review and meta-analysis of studies comparing the safety and efficacy of DOACs with VKAs/LMWHs in adults with cirrhosis treated for VTE or AF.

Results: Twenty-six studies (11,258 patients; 23 retrospective cohort analysis, 2 prospective cohort, 1 randomized controlled trial) were included for analysis, most having moderate risk of bias. Apixaban and rivaroxaban were the most used DOACs; warfarin accounted for 91.2% of comparator therapy. Most patients had Child-Pugh A and B cirrhosis.

- Lower thrombotic events with DOACs compared to VKAs: (14 studies) RR 0.72 (95% CI 0.58–0.89); greatest benefit observed in VTE / portal vein thrombosis (PVT) (RR 0.32).
- Lower major bleeding with DOACs compared to VKAs: (19 studies) OR 0.63 (95% CI 0.47–0.85); strongest effect in AF (OR 0.53).
- Minor bleeding was numerically lower but not significant: (9 studies) RR 0.85.
- Apixaban demonstrated the lowest major bleeding risk based on a subgroup analysis (RR 0.65).

Rapid Takeaway: In predominantly compensated cirrhosis, DOACs were associated with lower thrombotic and major bleeding events compared to VKAs. However, evidence remains largely observational, and caution is warranted in decompensated cirrhosis. ***Check Out this Archived Webinar:*** *Anticoagulation for AF, VTE and PVT in Cirrhosis*

Scand J Gastroenterol. 2026 May 13:1-17.

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Authors: Hannah Marks, PharmD & Youssef Bessada, PharmD

Editorial Team: Candace Bryant, PharmD; Cecilia Gollaher, PharmD; Greg Hadlock, PharmD; Yamel Herrera, PharmD; Carlee O'Connor, DNP; Bishoy Ragheb, PharmD; Kelly Rudd, PharmD