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| **Class** |  | **Agent** | | **Mechanism** | **Clearance** | | | **Usual Half Life (t1/2) & Duration of Effect** | | | | | | | **Most Common Indications** | | | **Monitoring Methods** | | **Strategies for Rapid Reversal and/or Hemostatic Correction** | |
| Direct Thrombin Inhibitors | Parenteral | Argatroban (Argatroban) 5,6 | | Directly inhibits soluble & clot-bound thrombin by binding reversibly to its active site | Hepatic (bilirubin is the best indicator of clearance) | | | * t 1/2: 30 to 50 minutes * Duration: approx 2 hours after end of infusion | | | | | | | * Prophylaxis or treatment of thrombosis in patients with HIT * Patients with (or at risk for) HIT when undergoing PCI | | | * aPTT * ACT   TEG – Increase R | | * Not well defined * Differing experts have postulated roles for:   + PCCs   + Recombinant factor VIIa   + Fibrinogen (via plasma & cryoprecipitate transfusions)   + Antifibrinolytic drugs * Not well eliminated by dialysis | |
| Bivalirudin (Angiomax) 5,7 | | Directly inhibits soluble & clot-bound thrombin by binding transiently to its exosites & active sites | Proteolytic cleavage & unspecified renal mechanisms | | | * t 1/2: 30 minutes * Duration: 2 to 6 hours | | | | | | | * Used (always with aspirin) as an anticoagulant in patients:   + Undergoing PTCA   + Undergoing PCI (with provisional use for GPI)   + With (or at risk for) HIT | | | * aPTT * ACT   TEG – Increase R | | * Not well defined * Differing experts have postulated roles for:   + PCCs   + Recombinant factor VIIa   + Fibrinogen (via plasma & cryoprecipitate transfusions)   + Antifibrinolytic drugs   + Modified ultrafiltration & hemodialysis | |
| Lepirudin (Refludan) 5,8 | | Directly inhibits soluble & clot-bound thrombin by binding irreversibly to its exosites & active sites | Renal | | | * t 1/2:  1 to 2 hours | | | | | | | Adult patients with HIT & thromboembolic disease requiring parenteral antithrombotic therapy | | | * aPTT * ECT   TEG – Increase R | | (Same as for Argatroban) | |
| Oral | Dabigatran (Pradaxa) 9-12 | | Inhibits soluble & clot-bound thrombin as well as thrombin-induced platelet aggregation | Renal | | | * t 1/2:  12 to 17 hours * Duration: not well defined; coag tests down to 30% of max within 12 hours but increase risk of bleeding for 5 days | | | | | | | To reduce risk of stroke & thromboembolism in patients with non-valvular AF | | | * Not well defined (& usually not required) * Roles may exist for: * aPTT * TT * ECT   TEG – Increase R | | * Not well defined * Differing experts have postulated roles for:   + Diuresis & dialysis   + Recombinant factor VIIa   + PCCs (perhaps less useful) | |
|  |  | Notes:   * Other DTI to be aware of is:   + Hirudin, rHirudin (Lepirudin, Refludan) | | | | | | | | | | | | | | | | | | | |
| **Class** |  | **Agent** | | **Mechanism** | **Clearance** | | | **Usual Half Life (t1/2) & Duration of Effect** | | | | | | | **Most Common Indications** | | | **Monitoring Methods** | | **Strategies for Rapid Reversal and/or Hemostatic Correction** | |
| Anticoagulant Agents | | | | | | | | | | | | | | | | | | | | | |
| Unfractionated Heparin (UFH) | IV | Heparin 1,2 | * Enhances antithrombin-associated inhibition of factor Xa & thrombin * Also inhibits conversion of prothrombin to thrombin & prevents formation of stable fibrin clot by inhibiting activation of fibrin stabilizing factor | | | Removed by the reticuloendothelial system; some hepatic & renal clearance | | | * T 1/2: 1 to 2 hours (increased with higher doses); * Duration: Several hours | | | | | * Prophylaxis & treatment of:   + Venous thrombosis & its extensions   + PE   + Peripheral arterial embolism * AF with embolism * Diagnosis & treatment of DIC | | | | * Anti-factor Xa heparin assay, * aPTT, and/or * While blood activated clotting time (ACT)   TEG – Increase R &  Reverses with Heparinase | | * Protamine sulfate   Note: The transfusion of plasma has no role in drug reversal | |
| Low Molecular Weight Heparins | Subcutaneous | Dalteparin (Fragmin) 1,3 | * Enhances antithrombin-associated inhibition of factor Xa & thrombin * Drug’s effect on factor Xa exceeds effect on thrombin | | | Primarily renal | | | * T 1/2: 3 to 5 hours with normal renal function * Duration: 10 to 12 hours | | | | | * Prophylaxis of:   + Ischemic complications in unstable angina & non-Q-wave MI   + DVTs in selected patients * Extended treatment of symptomatic DVTs & PEs in cancer patients | | | | * Usually not required * When necessary, use anti-factor Xa heparin assay * The aPTT is not useful   TEG – Increase R &  Reverses with Heparinase | | * Protamine (though not as effective as with UFH)   Note: The transfusion of plasma has no role in drug reversal | |
| Subcutaneous | Enoxaparin (Lovenox) 1,4 | * Enhances antithrombin-associated inhibition of factor Xa & thrombin * Drug’s effect on factor Xa exceeds effect on thrombin | | | Primarily renal | | | * T 1/2: Up to 7 hours with normal renal function * Duration: approx. 12 hours | | | | | * Prophylaxis of:   + Ischemic complications in unstable angina & non-Q-wave MI   + DVTs in selected patients * Inpatient treatment of acute DVTs (with or without PE) * Outpatient treatment of acute DVTs (without PE) * Treatment of acute ST-segment elevation MI managed medically or with subsequent PCI | | | | * Usually not required * When necessary, use anti-factor Xa heparin assay * The aPTT is not useful   TEG – Increase R &  Reverses with Heparinase | | * Protamine (though not as effective as with UFH)   Note: The transfusion of plasma has no role in drug reversal | |
|  |  | Notes:   * Other LMWH’s to be aware of are   + Tinzaparin (Innohep, Logiparin)   + Reviparin (Clivarin)   + Nadroparin (Fraxiparin) | | | | | | | | | | | | | | | | | | | |
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| Factor Xa Inhibitors | Parenteral | Fondaparinux (Arixtra) 5, 13 | Selective, indirect inhibitor of the binding of factor Xa to antithrombin III | | | Renal | | | * t½: 17 to 21 hours with normal renal function * Duration: not well defined | | | | | * Prophylaxis of DVTs in patients undergoing selected surgical procedures * Treatment of DVT or acute PE when given in conjunction with warfarin | | | | * Usually not required * When needed, use anti-factor Xa (requires a different assay than used for heparin or LMWHs)   TEG – Increase R | | * Not well defined * Neither protamine sulfate nor plasma have proved useful * Differing experts have postulated roles for:   + Recombinant factor VIIa   + Antifibrinolytic agents | |
| Oral | Apixaban (not yet FDA approved) 14 | Direct inhibitor of factor Xa | | | Multiple routes; 56% unchanged in feces, 25% renal | | | * t½: 8 to 15 hours | | | | | Prevention of VTE in patients who have undergone elective total hip or total knee replacement | | | | Anti-Xa activity (requires a different assay than used for heparin or LMWHs)  TEG– Increase R | | Not well defined (further studies pending) | |
| Rivaroxaban (Xarelto) 10,15 | Direct inhibitor of factor Xa | | | Renal and hepatic | | | * t½: 5 to 9 hours * Duration at least 12 hours | | | | | * To reduce risk of stroke and systemic embolism in patients with non-valvular AF * Prophylaxis of DVT (and PE) in patients undergoing knee or hip replacement | | | | * PT better than INR * Note: INR results do not mean the same as when INR is used for warfarin   TEG – Increase R | | * Not well defined * Different experts have postulated roles for:   + Recombinant factor VIIa   + PCCs | |
| Coumarin | IV and PO | Warfarin (Coumadin) 1, 16, 17 | Blocks the synthesis of vitamin K dependent coagulations factors (ie factors II, VII, IX & X) and anticoagulant factors (eg proteins C & S) | | | Hepatic | | | * Effective t½ for R- and S- enantiomer s: 20 to 40 hours * Duration: Usually 2 to 5 days | | | | | * Prophylaxis and treatment of:   + Venous thrombosis & PE   + Thromboembolic complications associated with AF and/or cardiac valve replacement * Reduction in risk of death, recurrent MI and thromboembolic events (eg stroke or systemic embolization) after MI | | | | INR  TEG – Increase R | | * Vitamin K * FFP (some experts recommend combining with PCCs) * PCCs (see immediately above) * Rarely, recombinant factor VIIa | |
|  |  | Notes: |  | | |  | | |  | | | | |  | | | |  | |  | |
| **Class** |  | **Agent** | **Mechanism** | | | **Clearance** | | | **Usual Half Life (t1/2) & Duration of Effect** | | | | | **Most Common Indications** | | | | **Monitoring Methods** | | **Strategies for Rapid Reversal and/or Hemostatic Correction** | |
| Anti-Platelet Agents | | | | | | | | | | | | | | | | | | | | | |
| Salicylate | Oral | Aspirin | Irreversible inhibition of platelet cyclooxygenase, blocking the formation of thromboxane A2 from arachidonic acid and thereby reducing platelet function and aggregation | | | Hepatic and renal | | | * t½: 5 to 9 hours * Duration: until platelets replaced | | | | | * Reducing the risk of non-fatal MI, non-fatal stroke or vascular death among patients with established arterial disease * Multiple other indicators | | | | * Assess clinically for signs of microvascular bleeding * Potential roles for:   + Platelet function analyzer (eg PFA-100)   + Turbimetric optical detection (eg VerifyNow)   TEG – PLM AA | | | * Platelet transfusions can partially correct the defect * Desmopressin (DDAVP) * Rarely, recombinant factor VIIa |
| Glycoprotein (GP) IIb/IIIa Inhibitors | Parenteral | Abciximab (Reopro) | Monoclonal antibody fragment specific for the GP IIb/IIIa receptors on the platelet surface, blocking the final common pathway of platelet aggregation | | | Enzymatic cleavage | | | * t½: 30 minutes * Duration: Platelet function generally recovers within 48 hrs, but can be abnormal for over 7 days | | | | | * Use as an adjunct to PCI for prevention of cardiac ischemic complications in patients:   + Undergoing PCI   + With unstable angina not responding to conventional medical therapy with PCI is planned within 24 hours * Intended for use with aspirin and heparin | | | | * Assess clinically for signs of microvascular bleeding   TEG – PLM ADP & AA | | | * Platelet transfusions |
| Parenteral | Eptifibatide (Integrilin) | Inhibits GP IIb/IIIa receptos on the platelet surface, blocking the final common pathway of platelet aggregation | | | Renal | | | * t½: 2.5 hours * Duration: Platelet function recovers 2 to 4 hours after end of infusion if normal renal function | | | | | * Treatment of ACS (unstable angina/non-ST-segment elevation MI), including patients managed medically and those undergoing PCI * Treatment of patients undergoing PCI, including those undergoing intracoronary stenting * Generally used with aspirin and heparin | | | | * Assess clinically for signs of microvascular bleeding   TEG – PLM ADP & AA | | | * Platelet transfusions * There may also be a role for cryoprecipitate transfusions |
| Parenteral | Tirofiban (Aggrastat) | Inhibits GP IIb/IIIa receptors on the platelet surface, blocking the final common pathway of platelet aggregation | | | Renal | | | * t½: 1.5 to 3 hours * Duration: Platelet function recovers 4-8 hours after end of infusion w/ normal renal function | | | | | * Treatment of acute coronary syndrome, including patients who are to be managed medically and those undergoing PTCA or atherectomy * Use in combination with heparin | | | | * Assess clinically for signs of microvascular bleeding   TEG – PLM ADP & AA | | | * Platelet transfusions * There may also be a role for cryoprecipitate transfusions |
|  |  | Notes: |  | | |  | | |  | | | | |  | | | |  | | |  |
| **Class** |  | **Agent** | **Mechanism** | | | **Clearance** | | | **Usual Half Life (t1/2) & Duration of Effect** | | | | | **Most Common Indications** | | | | **Monitoring Methods** | | | **Strategies for Rapid Reversal and/or Hemostatic Correction** |
| Adenosine Phosphate (ADP) Induced Platelet Aggregation Inhibitors | PO | Clopidogrel (Plavix) | Binds to ADP P2Y12 receptors on platelets, preventing ADP from binding to & activating GP IIb/IIIa receptors | | | Hepatic and Renal | | | * t½: 6 hours * Duration: 5 to 7 days | | | | | * ACS for patients with:   + Non-ST segment elevation ACS including patients who are to be managed medically & those who are to be managed with coronary revascularization   + ST-elevation myocardial infarction (STEMI), who undergo primary PCI * Recent myocardial infarction (MI), recent stroke or established peripheral arterial disease | | | | * Assess clinical for signs of microvascular bleeding * Potential roles for:   + Platelet aggregometry   + Turbimetric optical detection (eg VerifyNow)   TEG – PLM ADP | | | * Platelet transfusions can partially correct the defect * Desmopressin may be helpful * Very rarely consider recombinant factor VIIa |
| PO | Prasugrel (Effient) | Binds to ADP P2Y12 receptors on platelets, preventing ADP from binding to & activating GP IIb/IIIa receptors | | | Renal and Hepatic | | | * t½: 17 to 21 hours * Duration: 5 to 9 days | | | | | * Reduction of thrombotic cardiovascular events (including stent thrombosis) in patients with ACS who are to be managed with PCI as follows:   + Patients with unstable angina or non-ST elevation myocardial infarction (NSTEMI)   + Patients with ST-elevation myocardial infarction (STEMI) when managed with either primary of delayed PCI | | | | * Assess clinically for signs of microvascular bleeding   TEG – PLM ADP | | | (same as for Clopidogrel) |
| PO | Ticagrelor (Brilinta) | Reversibly Interacts with the ADP P2Y12 receptors on platelets, preventing ADP from binding & activating GP IIb/IIIa receptors | | | Hepatic and Renal | | | * t½: 7 hours * Duration: approx. 5 days after multiple doses | | | | | * To reduce the rate of thrombotic cardiovascular events in patients with ACS (unstable angina, non-ST elevation myocardial infarction or ST-elevation myocardial infarction) * In patients treated with PCI, it also reduces the rate of stent thrombosis | | | | * Assess clinically for signs of microvascular bleeding   TEG – PLM ADP | | | (same as for Clopidogrel) |
|  | Notes:   * Competitive effect of statins with prasugrel and clopidogrel (excluding crestor) | | | | |  | | | | | |  | | |  |  | |  | |  |
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| ADP inhibitor- PO | Ticlopidine (Ticlid) | Binds to ADP P2Y12 receptors on platelets, preventing ADP from binding to & activating GP IIb/IIIa receptors | | | Hepatic | | | * t½: approx. 4 hours after repeated doses * Duration: 4 to 10 days | | | | | * To reduce risk of thrombotic stroke in patients who have experienced stroke precursors and in patients who have had a completed thrombotic stroke * As adjunctive therapy with aspirin to reduce incidence of subacute stent thrombosis in patients undergoing successful coronary stent implantation | | | | * Assess clinically for signs of microvascular bleeding   TEG – PLM ADP | | | (same as for Clopidogrel) |
|  |  | Dipyridamole (Persantine) | Dipyridamole inhibits the [phosphodiesterase](http://en.wikipedia.org/wiki/Phosphodiesterase) enzymes that normally break down [cAMP](http://en.wikipedia.org/wiki/Cyclic_adenosine_monophosphate) (increasing cellular cAMP levels and blocking the platelet response to [ADP](http://en.wikipedia.org/wiki/Adenosine_diphosphate)) and/or [cGMP](http://en.wikipedia.org/wiki/Cyclic_guanosine_monophosphate) (resulting in added benefit when given together with [nitric oxide](http://en.wikipedia.org/wiki/Nitric_oxide) [NO] or [statins](http://en.wikipedia.org/wiki/Statin)). blocks the thromboxane synthase as well as the thromboxane receptor.It inhibits the cellular reuptake of [adenosine](http://en.wikipedia.org/wiki/Adenosine) into [platelets](http://en.wikipedia.org/wiki/Platelets), [red blood cells](http://en.wikipedia.org/wiki/Red_blood_cell) and [endothelial cells](http://en.wikipedia.org/wiki/Endothelial_cell) leading to increased extracellular concentrations of adenosine. | | | Hepatic | | | * Alpha half-life (the initial decline following peak concentration) is approximately 40 minutes. The beta half-life (the terminal decline in plasma concentration) is approximately 10 hours. | | | | | * Post op valve replacement * VAD post op management | | | | TEG – PLM ADP | | | Platelet transfusions |
| Antiplatelet | PO | Dipyridamole + Aspirin (Aggrenox) | See above for asa and persantine | | | Hepatic | | | * See above | | | | | * Stroke management | | | | TEG – PLM ADP | | | Platelet transfusion |
|  |  | Cilostazol (Pletal) | Phosphodiesterase III inhibitor, widening vascular walls and lessening platelet ability to adhere | | | Hepatic | | | * 11-13 hrs | | | | | * PVD * Intermittent Claudication | | | | TEG – PLM ADP | | | Platelet transfusion |
|  |  | Notes: |  | | |  | | |  | | | | |  | | | |  | | |  |
| **Class** |  | **Agent** | | **Mechanism** | **Clearance** | | | **Usual Half Life (t1/2) & Duration of Effect** | | | | | | | **Most Common Indications** | | | **Monitoring Methods** | | **Strategies for Rapid Reversal and/or Hemostatic Correction** | |
| Thrombolytic Agents | | | | | | | | | | | | | | | | | | | | | |
| Tissue Plasminogen Activators | IV | Alteplase (Activase, Cathflo) | Tissue Plasminogen activator; initiatives fibrinolysis by binding to fibrin & converting plasminogen to plasmin | | | Hepatic and inactiviation by plasminogen activator inhibitor-1 (PAI-1) | | | * t½: 26 to 46 minutes * Duration: not defined | | | | | Patients with:   * Acute MI * PE * Acute Ischemic Stroke * Arterial Thrombosis Embolism (off label) * Central Venous Catheter Occlusion | | | | Fibrin split products | | | Discontinue agent |
| IV | Tenecteplase (TNKase) | A derivative of activase with longer activity, greater affinity for thrombin and less susceptibility to PAI-1 | | | Hepatic and PAI-1 | | | * t½: biphasic: 20-40 minutes and 115 minutes respectively * Duration: data lacking for humans | | | | | Patients with:   * Hemodialysis Catheter Occlusion * Acute MI | | | | Fibrin split products | | | Discontinue agent |
|  |  | Notes:   * Possible competitive interaction with ppi and/or statin therapy and pletal | | | | | | | |  |  |  | |  | | | |  | | |  |

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| **Class** | |  | **Agent** | | **Mechanism** | **Clearance** | | **Usual Half Life (t1/2) & Duration of Effect** | | | **Most Common Indications** | **Monitoring Methods** | **Strategies for Rapid Reversal and/or Hemostatic Correction** | |
| Anti-Fibrinolytics | | | | | | | | | | | | | | |
| Antifibrinolytic | IV | | Epsilon Aminocaproic Acid (Amicar) | A derivative and analogue of the [amino acid](http://en.wikipedia.org/wiki/Amino_acid) [lysine](http://en.wikipedia.org/wiki/Lysine), which makes it an effective [inhibitor](http://en.wikipedia.org/wiki/Enzyme_inhibitor) for [enzymes](http://en.wikipedia.org/wiki/Enzyme) that bind that particular residue. Such enzymes include [proteolytic](http://en.wikipedia.org/wiki/Proteolytic) enzymes like [plasmin](http://en.wikipedia.org/wiki/Plasmin), the enzyme responsible for [fibrinolysis](http://en.wikipedia.org/wiki/Fibrinolysis). | | | Renal | | 2 hours | * CABG * Fibrinolysis induced bleeding * tPA reversal | | TEG – Decreases LY30 if fibrinolysis present | | D/C agent |
| IV | | Tranexamic Acid (Cyklokapron, Transamin) | [Antifibrinolytic](http://en.wikipedia.org/wiki/Antifibrinolytic) that competitively inhibits the activation of plasminogen to [plasmin](http://en.wikipedia.org/wiki/Plasmin), by binding to specific sites of both plasminogen and plasmin, a molecule responsible for the degradation of [fibrin](http://en.wikipedia.org/wiki/Fibrin), a protein that forms the framework of blood clots. Tranexamic acid has roughly eight times the [antifibrinolytic](http://en.wikipedia.org/wiki/Antifibrinolytic) activity of an older analogue, ε-[aminocaproic acid](http://en.wikipedia.org/wiki/Aminocaproic_acid" \o "Aminocaproic acid). | | | Unknown | | 3.1 hours | * PO heavy Menses * IV reversal of fibrinolysis induced bleeding * Reveral of tPA | | TEG – Decreases LY30 if fibrinolysis present | | D/C agent |
|  | | Aprotinin (Trasylol)  NOT Approved in US | small protein bovine pancreatic trypsin inhibitor, or BPTI, which inhibits [trypsin](https://en.wikipedia.org/wiki/Trypsin) and related proteolytic [enzymes](https://en.wikipedia.org/wiki/Enzyme); Its action on [kallikrein](https://en.wikipedia.org/wiki/Kallikrein) leads to the inhibition of the formation of [factor XIIa](https://en.wikipedia.org/wiki/Factor_XII). As a result, both the intrinsic pathway of coagulation and fibrinolysis are inhibited. Its action on plasmin independently slows fibrinolysis.[[4]](https://en.wikipedia.org/wiki/Aprotinin#cite_note-Mannucci-4) | | | Unknown | | 150 min | * IV use during CABG for prevention of bleeding | | TEG – Decreases LY30 if fibrinolysis present | | D/C agent |
|  |  | | Notes: |  | | |  | |  |  | |  | |  |

**Abbreviations**:

* ACS – Acute Coronary Syndrome
* AF – Atrial Fibrillation
* DIC – Disseminated Intravascular Coagulation
* DVT – Deep Venous Thrombosis
* ECT – Ecarin Clotting Time
* FFP – Fresh Frozen Plasma
* HIT – Heparin-Induced Thrombocytopenia
* MI – Myocardial Infarction
* PCCs – Prothrombin Complex Concentrates
* PCI – Percutaneous Coronary Intervention
* PE – Pulmonary Embolism
* PTCA – Percutaneous Transluminal Coronary Angioplasty
* TT – Thrombin Time
* VTE – Venous Thromboembolic Event

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| Blood Product Information |
| Fresh Frozen Plasma (FFP):   * 1 unit having all factors and 800mg fibrinogen * TEG – Decrease R |
| Cryoprecipitate (cryo):   * 1500mg fibrinogen in 6 bags or ‘one pool’ - (small children typically get smaller dose of 4 units with 350mg fibrinogen in each unit) * Includes vWF, Factor VIII and Factor XIII * ??? different than our drug table??? This is what I pull from text book but lets see what Mike/Jeff say…it could be there are 2 types? * TEG – Decrease R & Increase alpha |
| Platelets:   * Up to 8 units of ‘pooled’ platelets from separate donors can go into one bag for transfusion – or apheresis platelets can be given which are single donor and equivalent of 4-6 units pooled * You will get 200-400ml of plasma with platelets with both |
| 3 Factor PCC:   * Factors II, IX, X * Prothrombin Complex Concentrate (human) is indicated for urgent reversal of acquired coagulation factor deficiency induces by vitamin K antagonist (VKA, eg Warafin) therapy in adult patients with acute major bleeding * TEG – Decrease R |
| 4 Factor PCC:   * Factors II, VII, IX, X * KCentra * Prothrombin Complex Concentrate (human) is indicated for urgent reversal of acquired coagulation factor deficiency induces by vitamin K antagonist (VKA, eg Warafin) therapy in adult patients with acute major bleeding * TEG – Decrease R |
| Factor VIIa (Novoseven):   * Coagulation Factor VIIa (recombinant) is indicated for use in the treatment of bleeding episodes in hemophilia A or B patients with inhibitors to Factor VIII or Factor IX * TEG – Decrease R |
| Fibrinogen Concentrate:   * **Haemocomplettan/RiaSTAP**HFCP for use in the US is made from US licensed human plasma and contains US licensed human albumin as a stabilizer. * HFCP is a sterile, preservative-free, lyophilized fibrinogen concentrate in a single-use 100 ml vial. The amount of HFCP is approximately 1 g of fibrinogen with the actual potency for each lot indicated on the vial label and carton. HFCP is reconstituted with 50 mL Sterile Water for Injection (~20 mg/mL) and is administered intravenously. Each vial contains 900 to 1300 mg fibrinogen, 400 to 700 mg human albumin, 375 to 660 mg L-arginine hydrochloride, 200 to 350 mg sodium chloride and 50 to 100 mg sodium citrate. Sodium hydroxide and hydrochloric acid may be added to adjust the pH. * HFCP is indicated for the treatment of congenital fibrinogen deficiency. The recommended initial dose is 70 mg per kg body weight with subsequent doses depending on target and measured fibrinogen levels. The infusion rate should not exceed 5 mL per minute (100 mg/minute). |

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| Botanicals | | | | |
| The following contain Coumarins (possible anticoagulant properties):   * Agrimony * Alfalfa * Angelica (Dong Quai) * Aniseed * Arnica * Asafoetida | * Boldo * Buchu * Celery * Chamomile * Dandelion * Fenugreek | * Horseradish * Licorice * Parsley * Capsicum * Tonka Beans * Wild Carrot | * Wild Lettuce * Bogbean * Horse Chesnut * Meadowsweet * Sweet Clover * Passion Flower | * Prickly Ash * Quassia * Red Clover * Sweet Woodruff * Nettle * Cassia |
| The following have anticoagulant properties:   * Bladder Wrack/Fucus (heparin-like) * Pau d’arco * Salvia Multiorrhizhae (Cinnabar root) * Danshen (ATIII-like activity) * Chondroitin (heparin-like) | | | | |
| The following contain Salicylate (possible antiplatelet effects):   * Agrimony * Aloe Gel * Aspen * Senega * Wintergreen | * Willow * Feverfew (also reduces release of serotonin & affects the protein kinase pathway) * Cassia * Clove | * Dandelion * Black Cohosh * Blackhaw * Bogbean * Ginger (↓ TxA2) * Ginkgo biloba (↓ PAF) | * Ginseng (↓ TF, thrombin & PAF & may interfere with Coumadin) * Licorice * Garlic (↓ TxA2) * German Sarsparilla | * Policosanol * Poplar * Tamarind * Meadowsweet * Onion |
| The following have fibrinolytic properties:   * Bromelains (from pineapple plants, proteolytic enzymes that are anti-inflammatory, thereby reducing platelet activation) * Capsicum * Ginseng * Onion * Inositol Nicotinate * Garlic (increases streptokinase activated plasminogen activator) | | | | |
| The following have coagulant properties:   * Mistletoe * Yarrow | | | | |
| The following antagonize Warfarin (contain Berberine):   * Mahonia Aquifolium (Oregon Grape) * Goldenseal * Berberis Vulagaris (barberry) * Agrimony (Vit K) * Alfalfa (Vit K) | | | | |
| The following may reduce Coumadin effect:   * St. John’s Wort (increase P450 2C9 induction, increasing Coumadin metabolism) * Coenzyme Q10 | | | | |
| The following have other effects on coagulation: Marijuana may also be added here as antiplatelet property.   * Anamu (hemostasis properties –triterpens) * Fish Oil (antiplatelet, omega-3 fatty acids reduce inflammation & may stabilize atherosclerotic plaque) * Vitamin E (antiplatelet) * Llanten (hemostatic | | | | |
| The following contain styptics (astringents/hemostasis to reduce bleeding): | * Cinnamon * Labrador tea * New Jersey tea | * Delesseria Sanguinea (algae) * Shepard’s purse | * Burnet * Stock’ |  |

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TEG – Drug Table (Feb 2014)