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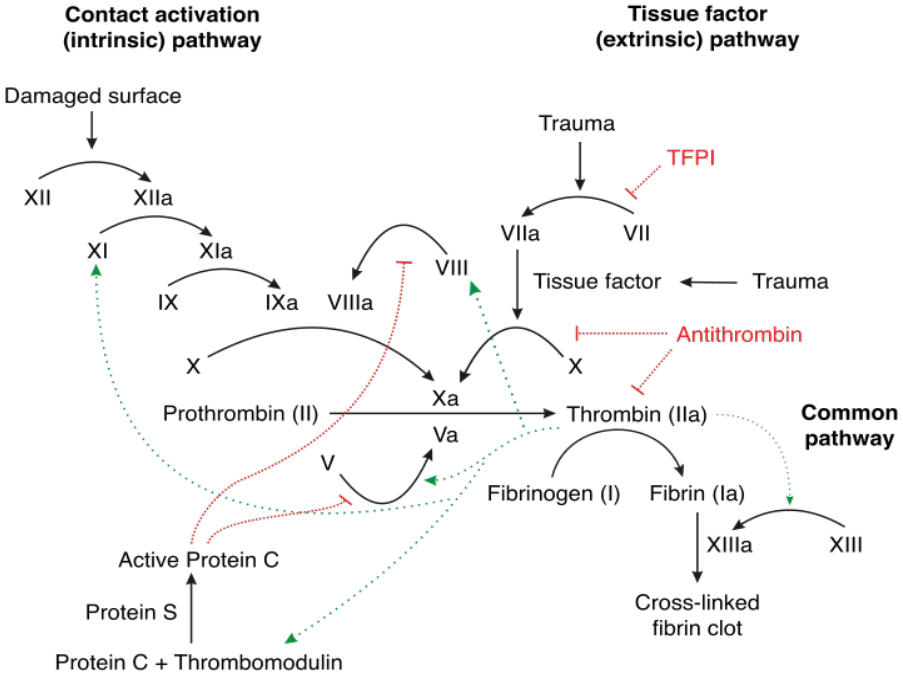
Coagulation Cascade & Bleeding

Coagulation Cascade

Coagulation cascade is a series of reactions that regulate prothrombic and antithrombic tendency
For prothrombic, goal is fibrinogen → fibrin, which requires thrombin in the common pathway



Common pathway – activated Factor X & Factor V → Thrombin → Fibrin formation
Extrinsic pathway – tissue trauma, Factor VII + Factor III (tissue factor) → common pathway
Intrinsic pathway – Activation of Factor VIII → common pathway. Smaller role in coagulation (but not insignificant)
Anticoagulant factors - TFPI, Antithrombin, Protein C, Protein S

Tests

PT/INR – Evaluates extrinsic and common pathways. Test with warfarin anticoag
APTT – Intrinsic and common pathways. Test with unfractionated heparin anticoag
Bleeding time – prolonged in thrombocytopenia, abnormal platelet function, von Willebrand's
Thrombin time – prolonged by low or abnormal fibrinogen, fibrin. Evaluate DIC, liver disease
Products of clot lysis – D dimer, fibrin degradation products. Evaluate DIC, DVT, PE

Therapeutic modification of coagulation

Warfarin – inhibits Vitamin K dependent factors (II, VII, IX, X). Prolongs PT & PTT.
Heparin – increases binding of antithrombin III to coag factors. Prolongs PTT and thrombin time. Reverse with protamine sulfate
LMW heparin can be given less often (subq daily vs continuous infusion), no need to monitor PTT, less risk of HIT
Aspirin – irreversible inhibition COX-1, preventing thromboxane formation in platelets
Plavix – inhibits ADP receptor on platelets
TPA, urokinase – activate plasmin, degrade formed clot. High levels of urokinase in prostate
If giving blood products, consider components of blood needed – coag factors, platelets, RBC, WBC
PRBC (incr Hct 3-4%/unit), platelet transfusions
FFP – correct coag factor deficiencies, no crossmatch required. ABO compatible if possible
Cryoprecipitate – concentrate of clotting factors

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Preexisting Defects of Hemostasis
Type A Hemophilia – abnormal factor VIII. Purified Factor VIII preparations available
Von Willebrand's – factor VIII, abnormal platelets. Can give desmopressin, promotes release of VWF

Acquired Defects of Hemostasis
Liver disease – decrease in synthesis of coagulation factors, prolonged PT, APTT
DIC – etiologies include tissue debris in bloodstream, excessive endothelial damage, hypotension and stasis, severe liver disease. Prolonged PT, PTT, thrombocytopenia, fibrin and fibrinogen split products, D dimer. Tx – platelets and cryoprecipitate to replace fibrinogen
Medical treatments:
Heparin, warfarin, NSAID, Lovenox
Thrombocytopenia and platelet function – aspirin, Plavix, decreased production (aplastic anemia), increased destruction (ITP, DIC), splenic pooling, uremia

Pre op assessment for bleeding

History – more useful than physical exam. Patient or family history of coagulation disorder, excessive bleeding or bruising with minor trauma, prior surgeries, dental work, nosebleeds
Physical exam – mild to moderate bleeding disorders do not usually have signs. Look for petechiae, ecchymoses, splenomegaly, hepatomegaly, hemarthroses
Prep – stop aspiring and Plavix 7-10 days before surgery. Get PT, PTT, platelet count as part of routine evaluation, but especially in patients with confirmed/possible bleeding disorder, or if blood loss expected

Intraoperative Bleeding

Bleeding in surgical field can be controlled with pressure, clamping of larger vessels, Gelfoam, Surgicel, Helistat, Surgiflo
Shock, hypothermia can exacerbate a coagulopathy
Excess transfusion (10+ units) with fluids dilutes clotting factors, transfused blood has few platelets
Treatment of transfusion reaction – can present with fever, back pain, hypotension, hemoglobinuria, bleeding, renal failure. Stop transfusion, manage hypotension with flulids, pressors, maintain renal function with diuretics, replace deficient clotting factors
Acute transfusion reaction can progress to DIC

Post op bleeding

50% post op bleeding due to poor hemostasis during surgery
Shock inducing a coagulopathy, decreased liver function after liver resection, DIC
Fibrinolysis – from administration of TPA, urokinase (prostate surgery)

Hypercoagulable states

Congenital conditions – factor V Leidin (prevents action of activated protein C), deficiencies of protein C and S.
Acquired – liver failure, high levels of procoag factors from trauma/stress, thrombocytosis, lupus
Virchow's triad – hypercoagulability, stasis, endothelial injury