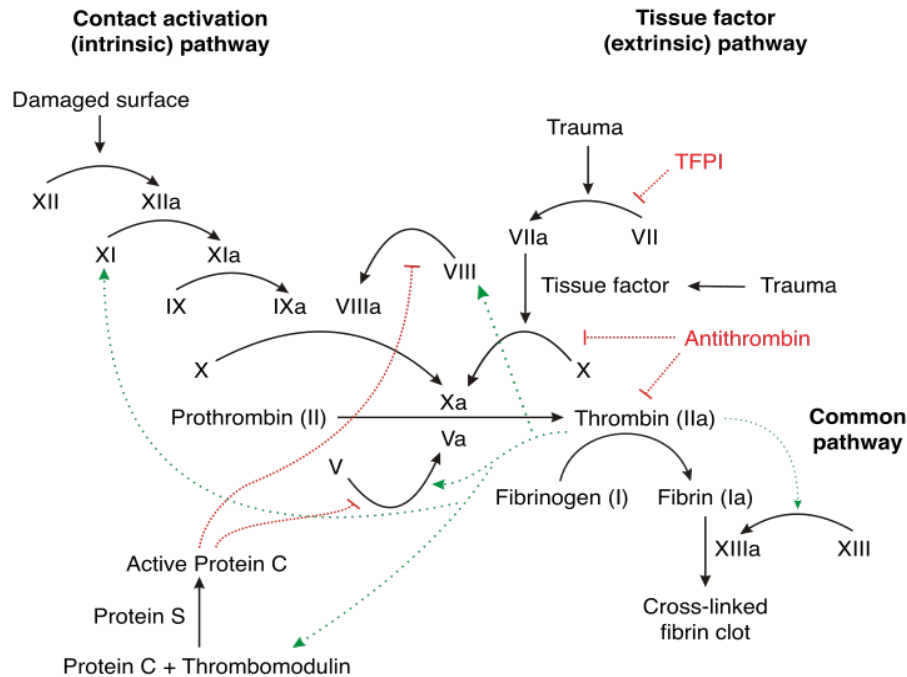


## Coagulation Cascade &amp; Bleeding

**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

## Coagulation Cascade & Bleeding

### **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 aspirin 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 fluids, 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 Leiden (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