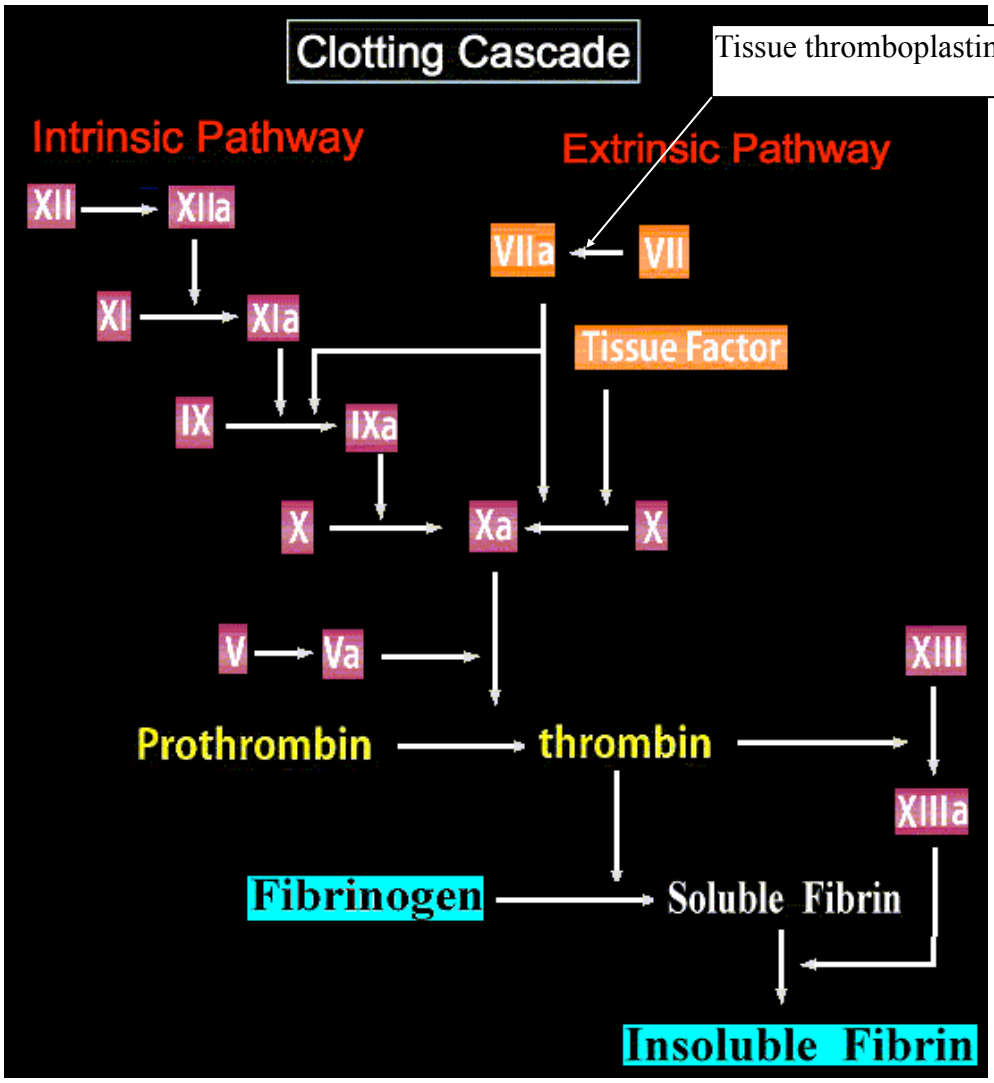
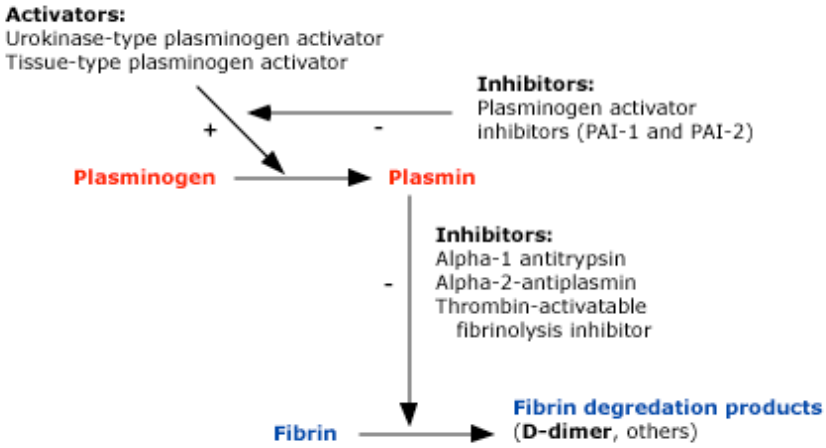
DIC – Disseminated Intravascular Coagulation



Balance exists between activation of the clotting and clot dissolution

The fibrinolytic pathway



Thrombin generation is normally restricted to the site of injury – the multiple natural anti-thrombotic pathways in the blood tightly regulate thrombin generation.

When these pathways are overwhelmed by production of thrombin, thrombin may circulate and lead to DIC.

The widespread deposition of fibrin results in tissue ischaemia and consumption of platelets, fibrinogen prothrombin and factors V and VIII, which in turn may cause bleeding – in acute DIC it is this picture which predominates.

The major initiating factors for DIC are:

- Extensive vascular injury exposing tissue thromboplastin
- Enhanced expression of tissue thromboplastin by monocytes in response to endotoxin and various cytokines

The major features of DIC are:

- Exposure of blood to procoagulants eg tissue thromboplastin and Ca procoagulant
- Formation of fibrin
- Fibrinolysis
- Depletion of clotting factors
- End-organ damage

Causes

- Sepsis
- Trauma and extensive surgery
- Malignancy
- Obstetric complications, eg
  - Amniotic fluid embolism
  - Abruptio placentae
- Miscellaneous
  - Acute haemolytic transfusion reactions

- Paroxysmal nocturnal haemoglobinuria
- Rattlesnake bites!
- Heat-stroke
- Rhabdomyolysis
- ARDS

Blood Tests

- Low platelets
- ↑ aPTT ↑ PT
- ↑ FDP / d-dimer

Prognosis

40 – 80% in patients with severe sepsis, trauma or burns

Treatment

Treat underlying cause!!

Others:

- **Platelet transfusion and FFP.** Pts with DIC bleed because of thrombocytopaenia and coagulation factor deficiencies. There is **no** evidence to support administration of platelets or clotting factors in patients who are **not** bleeding or at high risk of bleeding. However, treatment **is** justified in patients who have serious bleeding or are at high risk (eg after extensive surgery).
- **Heparin** – no evidence to support its use