Systemic coagulopathy similar if not identical to DIC which accompanies meningococcaemia
- extensively also in other severe TBI
- icd9 study- stopped for feasibility
- unacceptably high vital bleed in infants.

Hallmark time ischaemia + necrosis
- microvascular mononuclear

Severely low Ptc
- completely normal
- PAI-1 / plasminogen activator inhibitor
- highest levels in meningococcaemia
- APC + PAI-1 form complex inactivates inhibitor
- (inhibitor)
- likely why Ptc in meningococcaemia
- many trials is Ptc / APC

DIC studies in baboons (arc)
- TIC not activated ? Ptc suppressed not effective, adult
- TIC not comparable
- APC - anticoagulant
- ATIII study - no benefit from ATIII

Small series - benefit from ATIII
- no benefit TPA.

DIC studies - don't show T monoclonal pathogen
- pH reaction 7.2 c.f. not
- ucgo to recheck pti - /bag/10kg 98-124
- if fibrinogen < 0.75 g/L (units)
- recheck fibrinogen post Apc
- Aprot C - case by case basis
- recombinant VIIa - corrects bleeding from aggression
- "In monocled TBI even"
Coagulation

Ig A1 protein enhances coagulation (prothrombin).
- Impairs phagocytosis
- Inhibits lysozyme

LPS -> SIRS
- induces Schwartzman
- endothelial damage + h. sym.

Complement deficiencies (esp. C5-C8)
- Properdin def. → UMN disease
- Consider if the meningococcal
  B is present.

GP B meningococci
- Sialic acid on surface mimics other
  surface adhesion molecules.
- Resistant to serum mediated/pannus
  killing.