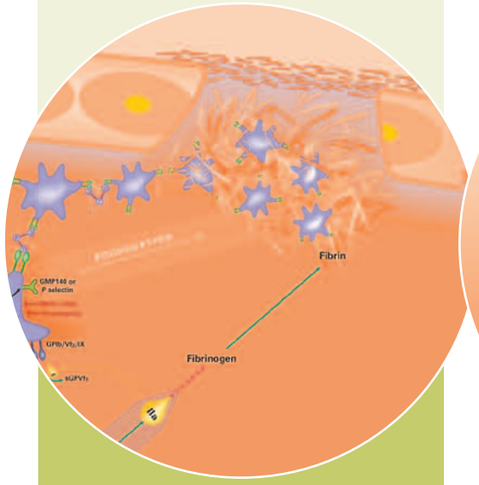
Innovation dedicated to Haemostasis

Primary Haemostasis

The primary Haemostasis corresponds to the reactions occurring after vascular damage and leads to the formation of a stable platelet clot. This is the first stage of the Haemostasis. To be effective, primary Haemostasis requires the optimal function of Von Willebrand Factor and platelets.

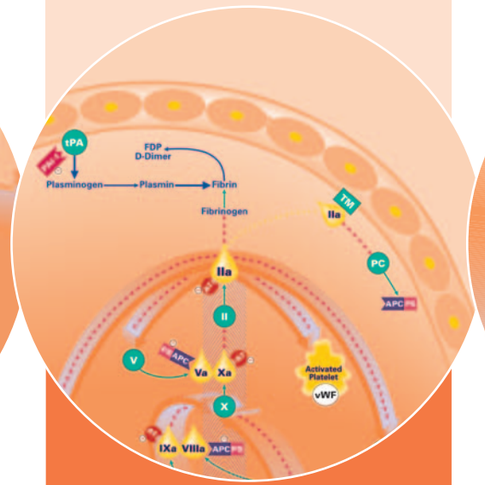


The parameters:

- Von Willebrand Factor
- Fibrinogen
- Platelet Factor 4
- ß-Thromboglobulin
- Soluble Glycoprotein V (sGPV)
- Platelet Glycoproteins by Flow Cytometry
- Anti-platelet antibodies by Flow Cytometry
- Thrombin Generation

Haemostasis activation

Following platelet activation and plasmatic coagulation, new molecules appear circulating in the plasma and the platelet membrane proteins are modified. An increase of these markers can reveal a prothrombotic state.

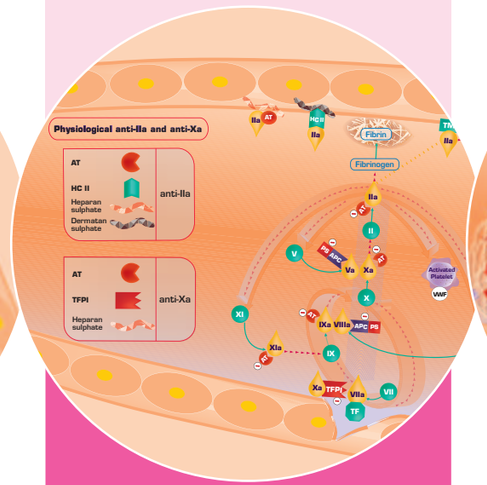


The parameters:

- D-Dimer
- Coagulation factors
- Von Willebrand Factor
- Fibrin Monomers
- Platelet Factor 4
- ß-Thromboglobulin
- Soluble Glycoprotein V (sGPV)
- Thrombomodulin
- Soluble Endothelial Protein C Receptor (sEPCR)
- Platelet Glycoproteins by Flow Cytometry
- Microparticules by Flow Cytometry
- Activated Factor VII - Antithrombin complex
- Thrombin Generation

Thrombosis

The onset of plasma coagulation is an «explosive» event that triggers the generation of thrombin. Various control pathways involving a number of different inhibitors regulate thrombin generation and ensure that homoeostasis is maintained. Anomalies regarding these inhibitors are the chief cause of venous and/or arterial thrombosis. However, thrombosis may also result from the presence of antiphospholipid antibodies.

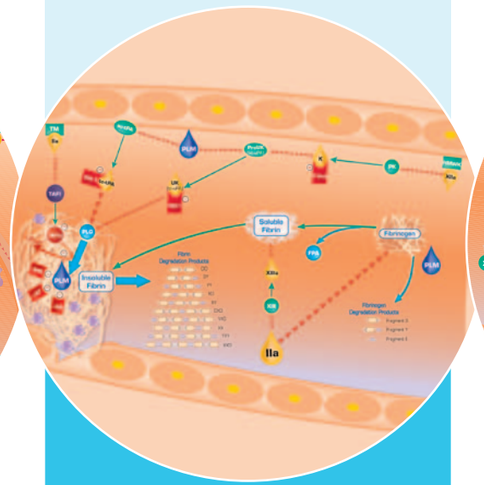


The parameters:

- Antithrombin
- Protein C
- Activated Protein C Resistance
- Protein S
- C4b-BP
- Protein Z
- Heparin Cofactor II (HCII)
- Inhibitors of the Extrinsic Pathway (TFPI)
- Soluble Endothelial Protein C Receptor (sEPCR)
- Lupus Anticoagulants
- Antiphospholipid Antibodies
- Thrombin Generation

Fibrinolysis

Fibrinolysis is the enzymatic process which, along with vascular repair, leads to the destruction of the clot to restore normal blood circulation. An imbalance of the stability in anti-fibrinolytic factors results in a Haemostasis disorder.

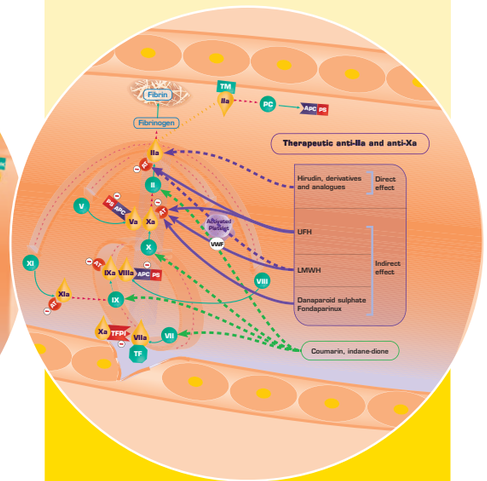


The parameters:

- D-Dimer
- Fibrinogen Degradation Products (FDP)
- Soluble Fibrin Monomer Complexes
- Fibrin Monomers
- Plasminogen
- tPA (Tissue Plasminogen Activator)
- Antiplasmin
- Plasminogen Activator Inhibitor (PAI)
- Thrombin Activatable Fibrinolysis Inhibitor (TAFI)

Therapeutic monitoring

Haemostasis disorders can be regulated by a broad panel of anti-thrombotic or anti-haemorrhagic treatments. Many assays are available to measure the activity of these molecules.



The parameters:

- INR for monitoring OAT
- Anti-Xa activity for monitoring directs (xarelto) and indirects (heparins, fondaparinux...) anti-Xa
- Anti-IIa activity for Direct Thrombin Inhibitors determination (dabigatran)
- Monitoring of P2Y12 ADP receptor antagonists
- Monitoring of GpIIb/IIIa antagonists by Flow Cytometry
- Coagulant Activity Monitoring for Activated Factor VII
- Clotting assay for monitoring Factors VIII and IX
- Anti-heparin antibodies/PF4 detection
- Thrombin Generation

Stago
At the Heart of Haemostasis

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