Who we are
Majority owned by the NHS, but with the commercial freedom to invest in innovation, Viapath are on a mission to transform pathology services in the UK. We provide pathology services to the NHS, private hospitals and other organisations both across the country and internationally.

What we do
All our laboratories are either accredited or working towards accreditation by UKAS to ISO15189. To view our laboratory accreditation status please follow this link:
http://www.viapath.co.uk/about-viapath/quality-and-governance/accreditations

TEST OVERVIEW

Description
Adding ristocetin at a final concentration of 1.25 g/l to platelet rich plasma (PRP) of a patient with von Willebrand disease (VWD) almost invariably results in a reduced agglutination of the platelets compared to a normal PRP. The main exception is the hyper responsive von Willebrand factor (VWF) of type 2B VWD. Performing the test at reducing concentrations of ristocetin reveals diminishing and eventually absent agglutination in the normal PRP but agglutination in the patient sample remains. This is not diagnostic of type 2B VWD because the hyper responsive platelets of Pseudo VWD give the same results, so the normal and test platelets are then washed and resuspended in the other's platelet poor plasma before reacting with ristocetin again. The abnormality in type 2B VWD resides in the patient's plasma, so reacting their plasma with normal platelets will reproduce the hyper-response to ristocetin, whereas reacting their platelets with normal plasma will give normal results. Converse results are obtained in Pseudo VWD because the abnormality exists in the patient's platelets not plasma.

Clinical details
von Willebrand factor (VWF) is a large adhesive glycoprotein synthesised in endothelial cells and megakaryocytes. Unlike the activated coagulation factors of secondary haemostasis it is not an enzyme and its functions involve binding to cells and molecules. Upon vessel injury, VWF binds directly to exposed sub-endothelial collagen and remains anchored. Blood flow unravels anchored VWF to expose the binding site for the constitutively expressed platelet surface receptor glycoprotein Ib. VWF captures and tethers platelets arriving at the scene which promotes subsequent events of primary haemostasis towards formation of a platelet plug. VWF also serves as the plasma carrier of FVIII to protect it from proteolytic degradation and also to 'deliver' it to sites of injury and clot formation. von Willebrand disease (VWD) is the most common hereditary bleeding disorder and the deficiency can be quantitative, involving reduced levels of normally functioning VWF, or qualitative, involving dysfunctional molecules. Laboratory investigation of VWD encompasses a battery of assays that assess different aspects of the molecule which inform sub-classification and clinical management: Type 2B VWD involves a dysfunctional VWF with increased affinity for platelet glycoprotein Ib which leads to thrombocytopenia and loss of high molecular weight multimers. Pseudo VWD, also called platelet type VWD, involves platelet glycoprotein Ib with increased affinity for the patient's normal VWF.
ORDERING INFORMATION

Sample type and Volume required
Contact laboratory.

Turnaround time
Analysed immediately, report within 7 - 10 days

Contacts
Diagnostic Haemostasis and Thrombosis Department
020 7188 2797
St Thomas’ Hospital
North Wing - 4th and 5th Floors
Westminster Bridge Road
London SE1 7EH

Laboratory opening times
24/7

How can we help?

We have a number of partnering options to suit your needs, whether you require this specific test or a range of services, we are here to help. Contact one of our friendly Business Development Managers for more information, or visit our website.