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COMPARISON OF A MODIFIED BLEEDING TIME, "HAEMOSTASIS TIME" (HT), WITH THE DUKE AND IVY/TEMPLATE BLEEDING TIMES IN HAEMOPHILIA AND VON WILLEBRAND'S DISEASE.

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The occlusion time of a Butterfly 25 short cannula inserted into the cubital vein was assessed as a new bleeding time modification. The method excludes the influences of vessel wall and tissue factors since primary haemostasis takes place on the standard surface of the cannula. Avoiding scar formation, the technique can be repeated deliberately. It could be shown that HT correlated better than the Duke and Ivy/template bleeding times with reduced platelet counts and reflected the effects of aspirin and unfractionated heparin in the same way (Janzarik et al., Blut 1986, 52:345).
HT was applied simultaneously with the Duke and Ivy/template or Simplate bleeding times in haemophilia and von Willebrand's disease (vWD). According to most of the other authors, the skin bleeding times were normal in patients with haemophilia. However, HT was prolonged in 9/16 patients irrespective of the factor VIII:C/IX:C values. The duration of HT correlated significantly to the onset of bleeding symptoms and to the degree of spontaneous bleeding (e.g., haemarthroses). In 31 patients with clinically mild or moderate vWD, the Duke bleeding time was prolonged in 3/31 patients, Simplate II in 13/31 patients and HT in 14/31 patients. With the two latter methods, corresponding results were observed in 20 cases, different results in 11 cases. HT and both the other methods correlated significantly to f. VIII:RCo and VIII:C but not to VIII:vWF.
We conclude that the repeatable HT method is an interesting diagnostic parameter in haemophilia and vWD. With the Simplate and HT methods simultaneously performed, the influence of vessel wall factors on primary haemostasis may be differentiated.

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EVALUATION AND STANDARDISATION OF THE "IN VITRO" BLEEDING TIME TECHNIQUE.

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Recently a model of primary hemostasis has been introduced (Kratzer & Born, Haemostasis 15:357;1985). This in vitro method detects very sensitively pathological platelet functions (Kratzer, Bellucci & Caen, Haemostasis 15:363;1985). In order to use this technique routinely in the hospital the instrument now called "Thrombostat 4000" has been completely computerized and standardized by Kratzer and von der Goltz, VDG (8221 Seeon, West Germany).
An artificial vessel (Aperture: cellulose acetate coated with collagen typ I, 2.2 ug/ mm2) with a single 150 um hole; capillary: teflon, 200 um, length 20 mm) was perfused with anticoagulated whole blood (Na-citrate 1 to 10) using a pressure of 40 mbar. To determine the reproducibility of the new technique, in vitro bleeding volume (V), time (T) and flow at the beginning of the experiment (IF), depending on blood viscosity, was measured in a healthy control person at different days (mean +- standard deviation, n= number of experiments).

Date	V(ul)	T(sec)	IF(ul/min)	n
21.08.86	178+-10	103+-4	148+-1	3
17.10.86	181+-12	109+-5	152+-2	7
17.11.86	177+-14	107+-10	148+-5	8
18.11.86	165+-10	97 +-12	152+-9	7
20.12.86	190+-5	-	149+-5	6
05.01.87	155+-14	105+-4	134+-10	7
08.01.87	193+-17	114+-10	143+-6	12
12.01.87	176+-21	107+-28	136+-4	10
16.01.87	180+-13	104+-8	149+-3	5
Mean:	177+-11	105+-5	145+-6	65

The mean of the intraassay variance (%) was: V (7.1), T(6.8) and IF(3.3). It was possible to measure the mean from day to day with the following variances (%): V(6.2), T(4.7) and IF(4.1). The low variance, which approaches enzymatic determinations is astonishing because platelet cell functions depend on a complex interaction of several thousands of enzymes.
The excellent assistance of P. Gossweiler is greatly aknowledged

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PILOT STUDY WITH AN "IN VITRO BLEEDING TIME" ("IN VITRO BT") TO MONITOR THE USE OF A STABLE PROSTACYCLIN (PGI_2) ANALOGUE IN HAEMODIALYSIS (HD).

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Administering PGI_2 instead of heparin (H) during HD enables the risk of bleeding to be reduced. A serious disadvantage of this method of inhibiting coagulation by inhibiting thrombocyte function is however that, in contrast to anticoagulation with H alone, there exists no quickly measurable monitoring parameter. Recently Kratzer and Born (Haemostasis 1985; 15: 357-62) presented a new method for measuring the primary haemostatis: the "in vitro BT". Our pilot study was to determine whether this "in vitro BT" is concentration-dependently prolonged by administering a stable PGI_2 analogue during HD. In 5 HD's the stable PGI_2 analogue CG 4203 (dose gradually raised starting 45 min before HD to 25 ng/kg b.w./min during HD; Gruenenthal GmbH, FRG) were given. Before, during and after the HD the "in vitro BT" (Thrombostat 4000, VDG, FRG) was measured under standardised conditions 15 min after taking 1 ml of citrate blood from the arterial branch of the extracorporeal system. The "in vitro BT" was concentration-dependently prolonged by CG 4203. The time interval between taking and measuring the blood sample affect the measured value. If the blood sample is taken directly before the dialyser, the "in vitro BT" is greater than if the sample is taken directly after the dialyser; from which it can be assumed that the dialyser activates the platelets. 1 h after ending HD and discontinuing the CG 4203, the "in vitro BT" is shorter than it was before starting HD; perhaps this is due to platelet activation during HD in the extracorporeal system and to removal of uraemic toxins. Furthermore,the "in vitro BT" was measured in a healthy volunteer 20 times consecutively. The coefficient of variability was 9.3 %. Further studies should be performed to see whether adjusting the "in vitro BT" to a given range allows the dosing of PGI_2 or a stable PGI_2 analogue to be so controlled as to prevent occlusion in the extracorporeal system.

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DO PLATELET INHIBITORS INCREASE OPERATIVE BLOOD LOSS?

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Although platelet inhibitory therapy improves arterial graft patency, surgeons are anxious that preoperative administration may increase operative bleeding. We investigated the effect of platelet inhibitors on blood loss during femoral artery replacement in dogs.

Thirty greyhounds were randomised to receive placebo, a thromboxane antagonist GR32191 25mg (Glaxo Group Research) or aspirin 150mg (ASA) plus dipyridamole 50mg (DPM) twice daily starting 48 hours prior to implanting a 6cm length of 6mm PTFE in the femoral artery using standardised incision, mobilisation and anastomosis with 6.0 prolene. All bleeding was collected in swabs which were then thoroughly washed in 2L heparinised saline. The erythrocytes were haemolysed by adding potassium cyanide and the haemoglobin concentration measured in a Coulter Haemoglobinometer (Coulter Electronics). Blood loss was calculated by comparison to 1:500 dilution of the same animal's venous blood. The bleeding time of the arterial anastomosis was also recorded.

The mean (± sem) blood loss was similar in all three groups tending to be slightly less with GR32191 and ASA + DPM at 135±25 and 115±21 mls respectively, compared to 152±29 mls on placebo (NS). Anastomosis bleeding time appeared to be prolonged at 390±31 secs by the thromboxane antagonist compared to 291±40 with placebo and 224±36 with ASA and DPM, but this difference did not achieve statistical significance. There was a significant correlation (r=0.53) between blood loss and anastomotic bleeding time ($p<0.001$).

This method of measuring blood loss is easily applicable to patients and does not demonstrate any important tendency to increased bleeding with preoperative platelet inhibitors.