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PREOPERATIVE IDENTIFICATION OF PATIENTS AT HIGH RISK OF DEEP VENOUS THROMBOSIS DESPITE PROPHYLAXIS IN TOTAL HIP REPLACEMENT.
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Preoperative prediction of postoperative venous thrombosis was investigated in 111 patients undergoing total hip replacement prophylactically treated with aspirin (1 g/d) or a combination of heparin (5000 IU) plus dihydroergotamine (0.5 mg) twice a day during 7 days. The following preoperative parameters were determined: age, sex, overweight percentage, previous thromboembolism, varicose veins, heart disease, malignancy, platelet count, platelet-crit, mean platelet volume, circulating platelet aggregates, platelet factor 4, ß-thromboglobulin, fibrinogen, Factor Xa, VIII:C, AT III, fibrin monomers, FDP, euglobulin lysis time, α_2-antiplasmin, tissue-type plasminogen activator (t-PA) and its inhibitor (PAI). Postoperatively deep vein thrombosis (DVT) developed in 16 patients was detected by ascending venography. Stepwise logistic discriminant analysis was used to identify factors which predicted DVT. Three such factors, FDP, PAI and t-PA, were significantly associated with DVT and used to construct a predictive index. The predictive index, I = 2.09 + 0.46 (FDP) + 1.39 (PAI) - 0.24 (t-PA), was 100% sensitive and 95% specific in the prediction of DVT. This index could allow for identification of those patients in whom routine prophylaxis would be sufficient and for selecting those in whom more effective prophylactic regimens would be necessary.

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TOTAL HIP REPLACEMENT AND DEEP VEIN THROMBOSIS - RELATIONSHIP TO THE FIBRINOLYTIC SYSTEM.
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Reduced fibrinolytic activity increases the risk of recurrent thromboembolism and it has been suggested that it also plays a role in postoperative thrombosis.

Material and methods. Fibrinolytic parameters were analysed in 29 patients submitted to total hip replacement. Dextran 70 was given as thrombosis prophylaxis. Blood samples were taken preoperatively, one day and one week postoperatively. Venous occlusion test was done in all patients. 125 I-fibrinogen test was used for deep vein thrombosis (DVT) screening. Positive test was confirmed with phlebography.Fibrinolytic activity was measured on fibrin plates. Tissue plasminogen activator (t-PA) and its specific inhibitor (PAI) were analysed with photometric and immunological methods.

Results. The first postoperative day t-PA activity decreased and PAI increased significantly. One week after operation only PAI showed significant difference from preoperative values. 10 of the patients developed DVT. The PAI level significantly higher in DVT patients preoperatively. This difference in PAI level was significant compared with non-DVT patients also one day and one week postoperatively.

		Preop.		1 Day Postop.		1 Week Postop.	
		DVT	No DVT	DVT	No DVT	DVT	No DVT
t-PA activity IU/ml	Mean	0.54	0.67	0.28	0.24	0.45	0.47
	SD	0.32	0.34	0.22	0.20	0.27	0.31
t-PA inhibitor U/ml	Mean	7.6	4.0	20.7	12.1	16.0	5.2
	SD	4.3	4.1	8.6	8.0	11.9	4.1

Conclusion. The recently discovered t-PA inhibitor (PAI) seems to be correlated to postoperative thrombosis in total hip surgery.

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ENDOGENOUS VASOPRESSIN RELEASE CONTRIBUTES TO THE THROMBOTIC RISK OF HIP SURGERY.
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Vasopressin (aVP) infusions in man that simulate physiological concentrations in plasma produce increases in both factor VIII and plasminogen activator activity (PAA) and we have found evidence that aVP release contributes to the activation of coagulation during abdominal surgery. The aim of this study was to investigate whether aVP has a similar role in the regulation of haemostasis during hip surgery. Venous blood samples were taken for FVIII:C, FVIII RiCof, vWF:Ag, fibrinopeptide A (FPA), ECLT, platelet aggregation in whole blood and aVP from separate venepuncture sites in 7 patients undergoing elective hip surgery. Samples were taken pre-operatively, post induction of anaesthesia, at skin incision, during division of the femoral neck, reaming of the acetabulum, cementing of the prosthesis and on the first post-operative day. FVIII:C increased during the operation from a geometric mean of 70% pre-operatively to 109% ($p < 0.05$) post-operatively. vWF:Ag and FVIII RiCof rose in a similar manner. PAA (10^6/ECLT^2) rose significantly from 12 units pre-operatively to a peak value of 167 units ($p < 0.001$) at prosthesis cementing, and post-operatively fell to subnormal levels. FPA concentrations followed a similar pattern rising from 13 pmol/ml to 58 pmol/ml ($p < 0.02$) at prosthesis cementing, and falling to 9 pmol/ml post-operatively. Plasma aVP rose from 0.5 pg/ml pre-operatively to 40 pg/ml ($p < 0.01$) at division of the femoral neck, and returned to 0.5 pg/ml post-operatively. There were no changes in platelet aggregation in whole blood using a single dose of 1.5 µM ADP. These results are similar to those we have observed during abdominal surgery. They confirm that during surgery, thrombin generation occurs with increased PAA, both of which are preceded by a rise in aVP. This is consistent with the hypothesis that aVP is an important mediator of changes in haemostatic function occurring during surgery, and we are now investigating the relationship between intra-operative changes in haemostatic function and risk of post-operative DVT.

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VENODILATION AND DEVELOPMENT OF DEEP VEIN THROMBOSIS IN TOTAL HIP AND KNEE REPLACEMENT PATIENTS.
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Postoperative deep vein thromboisis (DVT) is a frequent complication following total hip (THR) or knee (TKR) replacement but no test has been devised to identify specific patients who will develop DVT. Because conventional prophylaxis does not significantly reduce the incidence of DVT, monitoring is widely used to detect evolving thrombosis. More intense anticoagulation (adjusted dose heparin, two step warfarin) may be effective but requires laboratory tests and carries increased risk of bleeding. It would be an economic and medical advantage to restrict prophylaxis and monitoring to patients who will develop DVT. Based on observations in a canine model of THR, we developed and tested a method that shows promise of being able to identify, intraoperatively, patients who will develop DVT.

In the canine model we found characteristic venous lesions (gaping tears through endothelium and basement membrane, localized to confluences, selectively infiltrated with platelets and leukocytes). Incidence of lesions correlated with intraoperative venodilation, measured by a modified ultrasound scanner. Lesions might serve as sites for initiation and anchorage of thrombi. Diagnostic ultrasound was used to monitor cephalic vein diameter in 25 THR patients and 12 TKR patients. In THR patients, 1 of 9 patients with venodilation of 6-16% developed DVT. At 21-57% venodilation 12 of 12 THR patients developed thrombi. In the intermediate range of venodilation (19%, 20%), 2 of 4 patients developed DVT. In 12 TKR patients, 10 had venodilation of 0-16% and none developed DVT in the non-operated leg. In patients with 22% and 55% venodilation, one did and one did not develop DVT in the non-operated leg (expected from equal distribution between legs in THR patients). DVT in the operated leg did not correlate with venodilation. We suggest that in THR patients substances released at the operative site circulate briefly, causing venous dilation. In TKR patients the tourniquet prevented substances from being circulated, reducing venodilation and DVT in the non-operated leg. Proximity of surgical wound to calf veins and tourniquet pressure may have contributed to DVT in the operated leg.