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PREDICTION OF POST-OPERATIVE DVT BY SALINE DILUTION. D Chayen, SD Blair, CN McCollum, RM Greenhalgh. Department of Surgery, Charing Cross & Westminster Medical School, London, UK.

Clinically, it is difficult to predict deep vein thrombosis (DVT), but the in vitro saline dilution test using the Thrombo-elastograph (TEG) is reported to identify the risk for individual patients [1]. The Biobridge Impedance Clotting Time (ICT) is more sensitive and reproducible than the TEG [2], and we therefore studied 33 patients undergoing elective laparotomy to see if pre-operative saline dilution tests using both the TEG and ICT predicted post-operative DVTs. Post-operatively, both legs were scanned daily for 7 days using ^{125}I Fibrinogen to detect DVTs.

The mean age of the patients was 65.7±2.4 years and 17 had malignant disease. In this clinically high risk group, 24 developed a DVT.

	TEG High Risk	TEG Low Risk	ICT High Risk	ICT Low Risk
DVT	9	15	20	4
No DVT	1	8	3	6
Correct Prediction	90%	39%	87%	60%

Fifty-one percent were predicted correctly by TEG. The ICT was significantly better as a predictor with 79% of all patients correctly predicted ($p<0.01$).

The saline dilution test using the ICT is a significant improvement on the TEG, and may enable us to tailor DVT prophylaxis policy to each patient's specific requirements.

1. Heather BP, Jennings SA, Greenhalgh RM. The saline dilution test - a preoperative predictor of DVT. Br J Surg 1980; 67: 63-65

2. Blair SD, Menashi S, Samson D, Greenhalgh RM. Can the hypercoagulability of surgery be measured? Br J Surg 1986; 73: 500.

1404

ALTERATIONS IN THE COAGULATION AND FIBRINOLYTIC SYSTEMS AS PREDISPOSING FACTORS IN THE DEVELOPMENT OF DEEP VENOUS THROMBOSIS (DVT). L. Tengborn and A. Wallmark. Department for Coagulation Disorders and Department of Internal Medicine, University of Lund, Malmö General Hospital, Malmö, Sweden.

From Jan. 1982 to Jan. 1987, 1213 patients were investigated either because of thrombotic episode(s) or thrombosis heredity. Diagnosis of DVT was confirmed by phlebography in 567 cases. Patients were first examined at least three months after an acute episode.

Methods. Antithrombin (AT) and plasminogen were assayed, using chromogenic substrates S-2238 and S-2251, respectively, followed if values were low by immunochemical assessment. Furthermore, fibrinogen, thrombin and reptilase times, APTT, P&P were assessed. Only in the latter study period was protein C activity determined (129 cases). Fibrinolysis was assayed on fibrin plates after 20 min venous occlusion of the arms. In 1984 only, plasminogen activator inhibitor of endothelial cell type (PAI 1) was measured in most DVT patients (n=75).

Results. DVT first occurred by the age of 45 in 337 patients, of whom five had AT deficiency (four classic and one abnormal), one had low and another abnormal plasminogen), one had abnormal fibrinogen, seven had lupus anticoagulants (LA), and 72 (21%) had decreased lysis on fibrin plates. Defective fibrinolysis was re-investigated in 42 patients; at check-up 13 were found to have normalised. Of the 75 patients from 1984, fibrinolysis on fibrin plates was normal in 50 cases, of which PAI 1 was normal in 44 and increased in six; of the remaining 25 patients from 1984, fibrin plate activity was decreased, PAI 1 was normal in five cases and increased in 20.

DVT first occurred after the age of 45 in 230 patients, of whom none had pathological AT or protein C, five had LA, and one had abnormal plasminogen; of the 51 (22%) patients found to have defective fibrinolysis, 29 were re-investigated at check-up and 14 of them found to have normalised.

Conclusion. Alterations in coagulation inhibitors are rare in patients with DVT. A more frequent finding, although intra-individual fluctuations occur, is defective vessel wall fibrinolysis.

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PROTEIN C, FIBRINOGEN KINETICS AND PLATELET SURVIVAL IN PATIENTS WITH RECURRENT VENOUS THROMBOSIS. M.J. Crow (1), P.A. Learoyd (2), R. Warrington (2), S.M. Rajah (1). N.H.R.F. Cardiac Research Unit, Leeds Regional Cardio-thoracic Centre, Killingbeck Hospital, Leeds, U.K. (1) and Leeds Regional Blood Transfusion Centre, Leeds, U.K. (2).

Recurrent venous thrombosis though uncommon leads to post phlebitic limb syndrome. The pathogenesis is not readily definable in all cases. We have examined the platelet, coagulation and fibrinolytic systems of a small group of patients with venographically proven recurrent thromboses. Currently 14 patients have been studied. Besides routine investigations all patients had the following: 111 In labelled platelet survival, 125 I fibrinogen kinetics, Protein C (functional and antigenic), a full fibrinolytic screen, antithrombin 111 and plasma B-Tg. With the exception of isotopic studies all investigations were performed on two occasions. No patient was suffering a thrombotic event at the time of inclusion into the study. Protein C levels, fibrinolytic screens and antithrombin 111 levels were within normal limits for all patients, while plasma B-Tg was raised. Platelet and fibrinogen kinetic data is shown in the table (mean+/-SEM):

Platelet survival	days	6.77+/-0.29
Platelet turnover	$\times 10^9$/l/day	41.85+/-4.11
Fibrinogen T1/2	days	4.17+/-0.33
FCR		0.249+/-0.021

Mean fibrinogen kinetics were normal with only 2 patients displaying increased turnover. Mean platelet survival was reduced and turnover raised, although a further 2 patients had normal values. These results indicate that this group of patients requires extensive investigation including isotopic studies. The shortened platelet survival time found appears to indicate platelet activation and consequently a possible therapeutic role for antiplatelet drugs combined with anticoagulants.

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SERIC ANGIOTENSIN CONVERTING ENZYME : AN ENDOTHELIAL CELL MARKER (application to thrombo-embolic pathology). **L. Drouet, (*) B. Baudin, (*) (**) F. Ch. Baumann (**), J.P. Caen (*). Département d'Angio-Hématologie - INSERM U 150 - Hôpital Lariboisière - Hôpital Saint-Louis, Paris (*) Laboratoire de Biochimie A (Dr J. Giboudeau) - Hôpital Saint-Antoine, Paris (**).**

As a blood marker of endothelium, we investigated the seric activity of Angiotensin Converting Enzyme (ACE) at rest and after stimulation either by local venostasis or dDAVP infusion. dDAVP did not induce any significant change in ACE contrarily to venostasis. Searching for an endothelial abnormality implicated in the genesis of Deep Vein Thrombosis (DVT) we applied the local venostasis test to patients affected by recurrent DVT. Patients, divided in 3 groups (group I : documented history of recurrent DVT, group II : only one DVT or recurrent superficial venous thrombosis, group III : history of arterial thrombo-embolism), and controls were screened for ACE as well as for plasmatic fibrinolytic activity and von Willebrand factor (vWF) level. Two types of abnormalities of seric ACE activity were found : low basal level in group I and low response to venostasis in groups I and III : group II did not differ from controls. This suggests an endothelial lesion participating to the etiology of some recurrent DVT and supports the measurement of seric ACE to discriminate some patients at high risk of DVT. Measures of fibrinolytic and ACE activities are not redundant since the two types of ACE abnormalities were not individually encountered in the same patients and were independent from abnormalities of the fibrinolytic system.