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0345 BETA-THROMBOGLOBULIN IN PERIPHERAL ARTERIAL DISEASE

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Plasma beta-thromboglobulin concentration has been measured by radioimmunoassay using the kit from the Radiochemical Centre, Amersham, England. Results for 19 healthy volunteers had a mean of 33 ng/ml (range 13-60) with coefficients of variation within assay of 7%, within sample 14%, and within subject of 23%. Results for 50 patients with arterial disease had significantly higher mean of 51 ng/ml (range 10-127). The highest values were found in those with prosthetic (DACRON) grafts and previous endarterectomy. The raised values may be of significance as markers for anti-platelet drug therapy.

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0346 THE VALUE OF URINARY β-THROMBOGLOBULIN MEASUREMENTS IN CLINICAL SITUATIONS

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Plasma β-thromboglobulin (β-TG) measurements are subject to occasional false high values arising during sampling and processing. In the normal individual urinary β-TG is maintained at a constant low level (0.14 ± 0.09 ng. ml^{-1}), and elevations in this value reflect raised plasma concentrations. Plasma and urinary β-TG concentrations were measured in normal individuals, in 18 patients presenting with suspected deep venous thrombosis, and in 75 diabetic patients. Serial samples were also taken before and after 9 hip replacement operations. The results indicate that measurement of urinary β-TG concentration in patients may be a simpler and more reliable means of detecting platelet activation than assay of plasma samples. False positive results do not occur when urinary concentrations are measured, unless renal function is abnormal; grossly elevated values may even detect occult renal disease.

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0347 PLATELET FUNCTION IN ARTERIAL VASCULAR DISEASE.

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Plasma β-thromboglobulin (βTG) has been measured in 56 patients (mean age 63.6 years) with peripheral vascular disease (PVD) and 53 (Mean age 77.7) with recent and chronic cerebro-vascular disease (CVD). They were compared to 202 healthy subjects, 56 of whom were age and sex matched. In 33 of the PVD patients, platelet production time (PPT) heparin neutralising activity (HNA) and platelet aggregation (PA) induced by ADP, collagen epinephrine and thrombin were also measured. HNA was significantly shorter ($p \leq 0.0005$) and plasma βTG higher ($p \leq 0.0005$) in the PVD patients compared to the controls. The abnormal βTG was apparent (Wilcoxon test) only in 23 advanced PVD patients. In addition PPT was significantly shorter in 14 advanced PVD patients. The rate and extent of platelet aggregation in the PVD and plasma βTG in the CVD patients did not differ significantly from controls. There was no correlation between the four tests in the patients, suggesting that the tests were measuring various aspects of platelet function. These results indicate that in-vivo platelet consumption as well as platelet "release reaction" are enhanced in advanced PVD patients and may reflect a pre-thrombotic state.