vascular fibrin formation were studied: (1) patients with pre-eclampsia; (2) those having treatment with ancord (Arvin).

In pre-eclampsia, soluble fibrin formation was greatly increased in comparison with normal pregnant women. The isolated fibrin contained intact α, β and γ chains; on polyacrylamide gel electrophoresis, no crosslinking of γ chains could be found. A positive correlation was found, in fresh plasma samples, between the concentration of soluble fibrin as measured by affinity chromatography and the concentration of soluble fibrin–fibrinogen complexes as measured by agarose gel filtration. It appears that these soluble fibrin complexes consist of a mixture of fibrinogen and fibrin in a 1:1 ratio.

During ancord therapy, soluble fibrin was found in greatly increased amounts, and could be separated into two types by affinity chromatography. The greater part had a reduced α chain content, and could be eluted from the affinity column at 37°C. The smaller part had intact α chain and was not eluted at 37°C, suggesting that the extent and type of complex formation is dependent upon temperature.

The fact that complexes are partially degraded during ancord infusion suggests that they are formed by degradation of fibrin which is unstable due to the absence of crosslinking by factor XIII. In contrast, the fibrin complexes in pre-eclampsia may represent the early formation of fibrin which, if polymerization continues, may lead to intravascular fibrin deposition.

THE EFFECTS OF THERAPY AND SPLENECTOMY ON IMMUNITY IN MALIGNANT LYMPHOMA

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The immune status of eighty-five patients with malignant lymphoma (fifty-six with Hodgkin’s disease, twenty-nine with non-Hodgkin’s lymphoma) has been assessed at presentation and after radical radiotherapy or during intensive chemotherapy. Twenty-six of the patients with Hodgkin’s disease underwent diagnostic laparotomy with splenectomy; the effects of splenectomy on immunity were also studied. Only patients responding clinically to therapy have been included in all groups. Depression of skin test reactivity was seen mainly in patients having chemotherapy. We have recently reported out early observations on the effects of splenectomy on immunity in Hodgkin’s disease (Hancock, Bruce, Ward & Rich mond, 1976, British Medical Journal, 1, 313). Further studies confirm that cellular immunity after treatment was not influenced by splenectomy, whereas serum IgA and particularly IgM levels fell significantly with treatment in splenectomized patients; such changes in humoral immunity were not seen in non-splenectomized patients.

From this study it appears that irrespective of splenectomy some impairment of cellular immunity may occur with conventional treatment of malignant lymphoma, particularly combination chemotherapy. In splenectomized patients serum IgA and IgM may fall with treatment; this may be a predisposing factor in the development of systemic bacterial infection.

PLATELET ADHESION TO DAMAGED RABBIT AORTA AND THE EFFECT OF ACETYLSALICYLIC ACID AND SULPHINPYRAZONE

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(Introduced by G. P. McNicol)

Platelets are a major component of arterial thrombi and platelet–vessel wall interactions may be important in the pathogenesis of arterial thrombo-embolism. Many compounds inhibit platelet function in vitro, acetylsalicylic acid (ASA) being one of the most potent. However, it does not prolong platelet survival and there is no convincing evidence from clinical trials that it is effective in arterial thromboembolic disease in man. Sulphinpyrazone (SP) is a weak inhibitor of platelet aggregation and release, but does prolong platelet survival in man.

ASA and SP were examined for their effect on the adhesion reaction between isolated platelets and subendothelium exposed to balloon catheterization of rabbit aorta. Rabbit aorta was dissected to provide a leak-free tube, suspended in Tyrode’s solution at 37°C and perfused with washed rabbit platelets which had been labelled with sodium 51-chromate, and were suspended in Tyrode’s solution containing albumin and apyrase. After 10 min perfusion, radioactivity in the aortic sample was measured and platelet adhesion then calculated per mm² of exposed surface. Two samples were perfused simultaneously with control of drug-treated suspension. Adhesion was reduced to 40% of control by SP 250 μmol l⁻¹ (P<0.001) and to 55% of control by SP 25 μmol l⁻¹ (P<0.05). ASA at 1-2 mmol l⁻¹, which inhibits collagen-induced platelet aggregation, did not significantly reduce adhesion.

Results from a similar system had previously shown that ASA inhibited platelet adhesion to collagen-coated glass, and to scraped rabbit aorta averted on a rotating probe. When balloon-damaged aorta was tested in this system, ASA appeared to be effective. However, when the haematocrit in the system was raised to 40% by addition of washed rabbit red cells, the inhibitory effect of ASA was abolished, though the inhibitory effect of SP was not.