

with a history of active peptic ulcer or hiatus hernia, a recent cerebrovascular accident, and those who used oral anticoagulants, aspirin, or anti-aggregating drugs regularly were excluded from participation. At admission the patients were randomly allocated to one of three treatment groups: 1.5 gram of acetylsalicylic acid (ASA) in 3 divided doses, a combination of 300 mg dipyridamole (in 4 divided doses) + 1.5 gram of ASA, or phenprocoumon. All patients were confined to bed for 12 days. 135 Patients completed the trial. The 3 groups were well matched statistically with regard to sex, age, weight, and coronary prognostic index. The  $^{125}\text{I}$ -fibrinogen test was used for detection of D. V. T.

Results	No. of patients	D. V. T.	percent incidence
ASA	46	16	34.8
dipyridamole + ASA	44	5	11.4
phenprocoumon	45	6	13.3

The difference in frequency of D. V. T. between the ASA-group and the two other groups is highly significant ( $p < 0.01$ ). The combination dipyridamole + ASA was as effective as phenprocoumon in the prevention of D. V. T. after acute myocardial infarction.

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Heparinized plasma from certain dermatological patients mainly with psoriasis arthropathica demonstrated a precipitable fraction (HPF) when left at 4° C for 12 hours. The yield of isolated HPF-protein was 1.5–6.5 mg per ml of plasma, the clottability being of about 50–85 per cent. HPF consisted of three main components, fibrinogen, albumin and an unidentified protein (X-component). SDS polyacrylamide gel electrophoresis in the presence of 2-mercaptoethanol demonstrated the fibrinogen subunit chains to be of normal molecular weights. Quantitative N-terminal analysis demonstrated insignificant amounts of N-terminal glycine, indicating at most traces of soluble fibrin in the isolated HPF. Out of 21 patients with HPF 18 showed a negative ethanol gelation test in plasma, and 17 had no detectable FDP in serum.

The X-component was separated from albumin, subsequent to the removal of fibrinogen by heat precipitation (56° C–10 min), as a precipitating complex with concanavalin A and could be separated from this by a gel filtration on Sephadex G 150 after dissolution of the complex in 0.5 M  $\alpha$ -methyl-D-glucoside/0.3 M NaCl.

A possible interpretation is that HPF consists of a complex between essentially normal fibrinogen and the unidentified X-component with albumin as a main contaminant. The nature of the X-component and its quantitation in plasma in normal and pathological conditions are under study.

*S. Lopaciuk, R. P. McDonagh and Jan McDonagh* (Thrombosis Research Center, Department of Pathology, University of North Carolina, School of Medicine, Chapel Hill, North Carolina, 27514, U.S.A.): **Comparative Studies on Factor XIII in Plasma of Various Animal Species.** (472)

Factor XIII was assayed by the monodansylcadaverine incorporation method in platelet/thrombocyte-poor plasma of 14 species. In all plasmas activity was generated by thrombin. In horse, cat, goat, sheep, pig, rat, chicken, goose, and turkey values ranged from 55% to 120% of that found in human plasma. For rabbit, dog, guinea pig, and ox the values were 178%, 225%, 326%, and 471% respectively. Factor XIII activity was not correlated with fibrinogen concentration. The latter ranged from 1.37 to 4.44 mg/ml (mean:  $3.31 \pm 0.82$  mg/ml). Factor XIII of all species was neutralized to varying degrees by two human antibodies to factor XIII and, except for rabbit and the avian plasmas, by commercial rabbit antiserum to subunit A. No neutralization occurred with rabbit