Diagnosis of thrombosis is grossly inadequate. 3. *Prophylaxis is:* (i) ineffective with aspirin and calf stimulation, either alone or in combination, or with low dosage heparin alone, (ii) significant with a combination of intraoperative electrical calf stimulation and perioperative low dosage heparin in that the incidence is markedly reduced and the onset delayed the longer the heparin is administered. 4. Low dosage heparin or aspirin does *not* increase the risk of haemorrhage.

C. A. M. de Swart, A. Nijmeijer, J. W. N. Akkerman and J. J. Sixma (Dept. of Haematology University Hospital, Utrecht, The Netherlands): **Disappearance Rate of the Anticoagulant Effect of Heparin. (344)**

The disappearance of the anticoagulant activity of a intravenously administered well-defined commercial heparin was followed in human and dogs utilizing a diluted activated partial thromboplastin time (Marder) and an anti-X^a-assay (Yin). The anticoagulant activity was followed after the injection of a large single dose. More accurate determination of the relation between heparin level and disappearance rate was achieved by continuous infusion with different heparin dosages. The anticoagulant effect was linearly related with dosage administered above a certain minimum threshold. This is in agreement with disappearance curves obtained after a single injection that can be described by the formula:

$$S = K_3\, e^{-K_1 t} - \frac{K_2}{K_1}$$

(in which S represents the heparin activity, K_1 and K_2 represent constants and K_3 is the integration constant).

References: Marder, V. J.: A simple technique for the measurement of plasma heparin concentration during anticoagulant therapy. Thromb. Diath. Haemorrh. *24*, 230–239, 1970.

Yin, E. T., Wessler, S.: Plasma heparin: A unique, practical, submicrogram sensitive assay. J. Lab. Clin. Med. *81*, 298–310, 1973.

W. Remde and K. Funke (Medizinische Klinik des Bezirkskrankenhauses 15 Potsdam, German Democratic Republic): **Anticoagulant Prophylaxis in Chronic Haemodialysis. (345)**

In 15 patients with chronic uraemic syndrome anticoagulant prophylaxis was carried out with chloride phenindione during three to twelve months. Indications were: repeated clotting of the Scribner shunt, unfavourable localisation of the shunt or difficulties during the implantation which let expect a shunt clotting.

In spite of the heparinisation during the haemodialysis we succeeded in maintaining the Quick level between 15 and 35 percent. In all cases the incidence of shunt clotting was considerably reduced. Severe bleeding complications did not occur.

I. B. Mink and J. E. Fitzpatrick (Roswell Park Memorial Institute, 666 Elm Street, Buffalo, New York, 14263, U.S.A.): **Heparin Therapy Monitoring Tests – Effects of Method and Patient Variables upon Accuracy. (346)**

Tests utilizing the recalcification time of whole blood or plasma (e.g. A. P. T. T.) to monitor heparin therapy of a patient with malignancy and an accompanying thrombotic disorder, are subject to unpredictable *in-vitro* changes which occur during the time interval between withdrawal of blood from the patient and eventual testing in the laboratory. Significant reduction or prolongation of clotting time occurs in as little as five minutes, necessitating the performance of the test (whole blood recalcification or alternately centrifrigation and plasma recalcification) as rapidly as possible after patient contact.

Correlations are presented between the direction and magnitude of these time-related changes and methodology variables (venipuncture stress, tube surface, decalcifying agent, storage temperature) as well as patient variables (heparin dosage, PF-4 activity, clotting factor lability, heparin tolerance). Repeated testing of the same blood samples may provide a pattern of change which reflects *in-vivo* heparin catabolism.