SURGICAL PROBLEMS ON PATIENTS WITH COAGULATION DEFECTS. T. Mandalaki, B. Golematis, C. Loizou, P. Delikaris, E. Papalabros and Ch.Tsigris. 1st Surgical Department, Athens University and 2nd Blood Transfusion Centre, Athens, Greece.

Surgical experience over a period of three years referring to 40 cases of hemophiliacs, with special reference to rare surgical complications, is presented. Critical evaluation of the post-operative results suggests that two main points should be equally taken into consideration for a successful surgical treatment. a) Hematology-substitution treatment, b) Surgery-local hemostasis. Substitution therapy is adjusted according to the nature and severity of the defect. Full pre-operative correction of missing factor to normal levels. A level over 30% should be maintained post-operatively for at least 15 days, particularly in major abdominal surgery. The intensity of substitution treatment must be related to the kind of operation. Regarding Surgery meticulous local hemostasis using fine silk ligatures and atraumatic needle chrome cat-gut, for the gastrointestinal system, preferably with interrupted sutures. Electrocoagulation is avoided. Removal of skin sutures should be delayed, as well as peroral alimentation in gastrointestinal tract Surgery.

ACTIVATED PROTHROMBIN CONCENTRATES IN MANAGEMENT OF BLEEDING IN A CLASSICAL HEMOPHILIAC WITH INHIBITOR. J.M. Whaun and A. Kaegi. University of Calgary Faculty of Medicine, Calgary, Alberta, Canada.

Bleeding in hemophiliacs with circulating anticoagulants is still a serious management problem. We would like to report our efforts at achieving hemostasis in a 31 year old AHF deficient severe hemophiliac with inhibitor detected in Feb. 1972. He presented with right forearm flexor compartment bleed which did not respond to conservative management with Konyne (30 u/kg q3h) and necessitated a fasciotomy to relieve compression. No hemostasis occurred until 4 hrs. post-operatively when he received his first infusion of Auto-Factor IX*, 6 vials (60 u/kg). At this time he also received blood for his shocky state. With the institution of regular infusions of Auto-Factor IX alternating with Konyne, hemorrhage was controlled. A week later a split thickness skin graft (from R. thigh) was applied. With continued infusions of mainly Auto-Factor IX (60 u/kg q6h) the grafted and donor sites healed. The patient was subsequently discharged with full range of movement in all limbs. His inhibitor levels which ranged between 1 - 3 Bethesda units/ml shortly after admission over a period of 3 weeks rose to over 100 units/ml. Six months after the episode his inhibitor levels are still over 100 units/ml. Activated prothrombin concentrates are effective in hemostatic control of life-threatening bleeding in hemophiliac patients with inhibitors.

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THE MANAGEMENT OF TWO CASES OF "SPONTANEOUS" FACTOR VIII INHIBITORS WITH HIGH DOSE CYCLOPHOSPHAMIDE. E.D. Gomperts. Department of Haematology, School of Pathology and the South African Institute for Medical Research, Johannesburg, South Africa.

Two patients with excessive bleeding associated with "spontaneous" Factor VIII inhibitors were studied. The first, a White male aged 52 years, was treated with high dosage cyclophosphamide and steroids together with plasmapheresis. An antigenic stimulus via concentrate and fresh plasma was given together with the stat dose of cyclophosphamide (1.6gm) together with prednisolone 60mg/day. Repeat plasmopheresis was carried out on two subsequent occasions shortly thereafter. The inhibitor level dropped progressively from 6.6 u/ml to almost unrecordable levels. However, escape from control was associated with the onset of hepatitis. Further therapy with an identical form of treatment failed to subsequently modify the inhibitor level which rose progressively to very high levels. In the second case, a White female aged 79 years, plasmapheresis was not carried out (inhibitor level 2.0 u/ml). Cyclophosphamide and prednisone were given in doses of 20 mg/Kg and 60 mg/day respectively. A Factor VIII antigenic load was given 24 hours before the cyclosphosphamide. Two subsequent cyclophosphamide pulses of similar dosage were given at approximately 10 day intervals without an antigenic stimulus. The patient was then maintained on a small daily dose of cyclophosphamide (50 mg/day). The inhibitor level responded to this therapy resulting in disappearance of the inhibitor and a progressive rise in the Factor VIII to levels greater than normal (170%). Our experience with these two cases suggests that a more rational approach to immunotherapy in the second case resulted in a sustained satisfactory immunosuppressive response.