

ORAL PROPHYLACTIC THERAPY IN HEMOPHILIA A AND B (FACTOR VIII AND IX DEFICIENCIES).
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The purpose of this ongoing nutritional study with massive doses of choline ultimately is to try and establish an alternative means of prophylactic therapy to recently proposed use of plasma fractions, plasma factor concentrates and earlier so-called "bleeding and clotting diets". Contrary to other prophylactic therapy studies, our premise is that if all other hemostatic and homeostatic factors are near normal through optimum nutrition, the over-all importance of Factor VIII and Factor IX deficiencies are less significant.

Case reports of patients with Hemophilia A and B are presented, which indicate a comparative decrease in the plasma fraction requirements for the treatment of the bleeding episodes in these patients during long term therapy with megavitamin doses of choline (a refinement of Boudreaux's "peanut factor"). One case (WFS) of severe Hemophilia A with less than 1 % Factor VIII, under therapy for over 10 years has had periods of remission not requiring plasma factor infusions for almost 2 years.

From our combined studies, we conclude that a long term nutritional prophylactic therapy program with emphasis on choline in Hemophilia A and B can result in a decrease in plasma needs, antibody formation and the occurrence of hepatitis.

HEMOPHILIA A AND VARIANT OF VON WILLEBRAND'S DISEASE IN ONE FAMILY.

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A 6 years old boy with the admitting diagnosis hemophilia A was transferred to our hospital because of persistent bleeding episodes inspite of adequate treatment with AHF. Laboratory data were as follow: F VIII coagulant activity 1.7 %, inconstantly prolonged bleeding time (hemorrhagometry), inconstantly reduced platelet adhesiveness, normal IVY-bleeding time, normal platelet aggregation with ADP and collagen, reduced Ristocetin-induced aggregation with PRP, reduced Ristocetin-cofactor activity, normal F VIII associated antigen. An inhibitor against F VIII could be excluded. The bleeding stopped and the pathological coagulation findings were corrected after substitution therapy with isoagglutininfree cryoprecipitate. Because of these findings we made the diagnosis of variant of von Willebrand's disease. His 8 years old brother had classical hemophilia A with a F VIII coagulant activity of 1.8 % and all other coagulation parameters mentioned above within normal limits. In the mother a carrier state could not be detected. In the mothers and fathers family bleeders are not known.

HEMOPHILIA IN NORWAY.

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Studies of all hemophilic families in Norway have resulted in the following distributions as to type and degree of factor defects.

	Number of families	Number of cases			Total
		severe (factor $\leq 1\%$)	moderate (2-5%)	light ($\geq 6\%$)	
Hemophilia A	137	119	33	79	231
Hemophilia B	36	19	46	11	76
Total	173	138	79	90	307

Of the hemophilia B families, 3 were of the type BM. In connection with multiple transfusions to patients with severe hemophilia A, 13 patients developed severe abnormal inhibitor to f.VIII, 3 of them died. In several other such transfused hemophilia A patients, weaker coagulation inhibitory activities have been demonstrated.