constitutes a previously unrecognized mechanism for the destruction of alloxan differing from those based on reduction as by glutathione(5), or condensation(5).

Conclusion. Borate greatly accelerates the conversion of alloxan to alloxanic acid. This observation provides an explanation for its protective effect against alloxan diabetes.

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Use of Desoxycorticosterone Acetate in Treatment of "Hyperheparinemia".* (18845)

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It has long been assumed that mast cells contain heparin in an active or inactive form(1). Recently, evidence has been presented(2) to show that, in response to certain hormonal products, chiefly of the stress-producing type, the morphologic pattern of the mast cells of mice and other animals are altered and, in the animals studied, the heparin content of the blood may increase. Of the hormones studied, only desoxycorticosterone acetate (DCA) was found to preserve the morphology intact and, in larger doses, prevented its alteration by subsequent administration of the stress-producing hormones(2).

Recently, an opportunity was afforded to study a case of idiopathic "hyperheparinemia"(3). Since this report, we have studied the effect of protamine sulphate and DCA on this patient's coagulation defect. The response to protamine therapy was excellent but, after a month, evidence of resistance to protamine appeared and he relapsed. Because of his subsequent failure to respond to protamine sulphate, it was decided to try the effect of large doses of DCA on this patient. The administration of 25 mg of DCA in oil daily by the intramuscular route resulted in a drop of the endogenous heparin from 23 γ per ml of plasma to 0 γ in 4 days (Table I).

During the course of observation, this patient, under control conditions, showed a clotting time (Lee-White) of 110-130 minutes, a prolonged one stage prothrombin time (Quick) of 75-90 seconds (normal 13.2 seconds), despite a normal prothrombin concentration by the modified 2-stage procedure of Ware and Seegers(4), and a prolonged recalcification time of oxalated plasma. Both the protamine titration test(5) and the protamine-heparin titration method(6) gave consistently abnormal values under control conditions. During therapy with DCA, the three other abnormal tests improved along with the drop in the protamine titration. A complete reversion to normal values occurred when the endogenous heparin had fallen to an insignificant amount in the plasma.

To rule out the possibility of a spontaneous remission, treatment was discontinued for 72 hours following which hyperheparinemia recurred. On the readministration of 90 mg of DCA in a period of 4 days, the heparin level of the blood again fell from 23 to 2 γ per ml of plasma. This observation suggests the possible use of this therapy in the hemorrhagic

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TABLE I.

Day of treatment	Clotting time (min)	1-stage prothrombin (sec)	Recalcification time of oxalated plasma (sec)	Endogenous heparin in plasma (γ/ml)
Before DCA	137	142	270	23
After 2 days	131			23
3 "	72			17
4 "	14	13.8	90	0

tendency following irradiation(5).

In this patient, the use of desoxycorticosterone acetate, in large doses, reduced the level of heparin or heparinoid substances in the blood. The mechanism of this response is unknown.

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Characteristic Electrophoretic Patterns in Hemophilia and Idiopathic Thrombocytopenia.* (18846)

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Observations on the changes in the composition of plasma proteins in the hemorrhagic diseases as revealed by electrophoresis are practically non-existent. Armstrong(1) failed to find any fundamental electrophoretic difference between normal and hemophilic plasma. No data are available in the literature on the electrophoretic patterns in idiopathic thrombocytopenia.

Routine electrophoretic analyses of plasma, by means of the technic described in a previous communication(2), have been conducted during the past year in a group of cases with various types of hemorrhagic manifestations. This group included 5 cases of hemophilia, 8 of idiopathic thrombocytopenic purpura, 6 of thrombocytopenia secondary to hypoplastic anemia or leukemia, 3 with the "anaphylactoid" type of vascular purpura, and 6 with vascular purpura. A characteristic anomaly of the electrophoretic patterns was observed in 17 of these 28 patients. The anomaly consisted in: (a) the appearance of an unusual peak in the group of the α-globulins, which we have designated as α_x (Fig. 1), with a motility ranging between 4.30 and 4.60 $\times$ 10^{-5} cm^2 $volt^{-1}$ sec^{-1} (in veronal-citrate buffer, pH 8.6, ionic strength 0.1); (b) complete disappearance of the usual α_2- and α_3-globulin peaks with mobility between 3.75 and 4.25 $\times$ 10^{-5} cm^2 $volt^{-1}$ sec^{-1}. These changes were more marked in the ascending than in the descending boundaries.

These anomalies were observed in all the cases of hemophilia and idiopathic thrombocytopenia which were studied; they were also found in 2 of the 6 cases of secondary thrombocytopenia, in 2 of 3 cases of the "anaphylactoid" type of vascular purpura. The characteristic anomaly was also observed in 2 cases of acquired hemolytic anemia, one of which

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