tive colitis given insulin, 2 showed a rise in eosinophils (32%, 37%) and a third only a 12% drop. Five patients with milder ulcerative colitis showed a normal response.

Surgical Operations. Fifteen patients have been followed through the stress of major surgery. Following operation, 14 of these patients showed profound drops in their eosinophil counts, 6 falling to zero. In general the degree of fall varied directly with the magnitude of the operation. The peak of response occurred most commonly 3-8 hours after the beginning of the operation. The fifteenth patient, one with severe ulcerative colitis, showed a marked and progressive eosinophilia following an ileostomy.

Discussion. Epinephrine and insulin appear to exert similar effects on the circulating eosinophils of man, and our results suggest that this effect is mediated through the pituitary and adrenal cortex. The failure of response in a small number of cases of Addison's Disease and pituitary insufficiency leads us to anticipate that the eosinophil changes following epinephrine and insulin may be useful in the diagnosis of these conditions.

Browne and his associates[8] have measured the changes in excretion of glycogenic corticoids and other indices of adrenal function following major surgery, and have interpreted these changes as part of the process of adaptation to non-specific stress. The occurrence of eosinopenia following operations may be construed as further evidence of the role of the adrenal cortex in the "alarm reaction" in man.

In view of the postulated role of the adrenal in the genesis of "essential" hypertension, the exaggerated eosinophil response in patients with this disorder is being studied further. The absence of an eosinophil response in three patients with severe chronic ulcerative colitis suggests that adrenal insufficiency may occur in such patients. This possibility is being investigated.

Summary. Significant eosinopenia has been observed following injection of epinephrine and of insulin. This change was exaggerated in patients with essential hypertension, and diminished or absent in patients with adrenal or pituitary insufficiency. Marked eosinopenia has been found after surgical operations.

The authors are indebted to Dr. Peter Forsham for technical advice.

8 Browne, J. S. L., Conference on Bone and Wound Healing, Josiah Macy, Jr., Foundation, December 11-12, 1942, pp. 45-47.

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Electrophoretic Patterns After Dicumarol Medication.

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Dicumarol induces prothrombinopenia by inhibiting the production of prothrombin, presumably in the liver. At dosage levels used for therapeutic purposes, its action is reversible and can be counteracted by vitamin K.[1-3] In order to determine whether or not protein components other than prothrombin were measurably altered by the drug, comparisons by means of electrophoresis of human plasma before and after Dicumarol medication were made.

1 Campbell, H. A., Smith, W. K., Roberts, W. L., and Link, K. P., *J. Biol. Chem.*, 1941, **138**, 1.

2 Shapiro, S., Redish, M. H., and Campbell, H. A., PROC. SOC. EXP. BIOL. AND MED., 1943, **52**, **12.**

3 Cromer, H. E., Jr., and Barker, N. W., *Proc. Staff Meet. Mayo Clin.*, May, 1944, **19**, 217.

TABLE I.
Electrophoretic Data and Prothrombin Times on Plasma from Patients after Dicumarol.

Specimen	% concentration of					A/G	Prothrombin time
	Albumin	Globulins					Whole plasma, sec.
		α	β	Φ	γ		
Ma I	57.8	8.5	10.0	8.2	15.4	1.37	17.5
Ma II	59.3	8.2	11.9	6.7	13.9	1.45	39.5
Mo I	66.8	7.2	14.2	3.5	8.5	2.00	16.0
Mo II	66.2	7.6	13.9	3.8	8.3	1.97	40.6
Ox I	65.1	5.7	11.4	4.5	13.3	1.86	15.0
Ox II	64.4	4.5	12.2	4.5	14.4	1.81	38
Ow I	45.9	11.6	18.0	9.1	15.2	0.85	15.2
Ow II	42.8	12.0	17.3	9.1	18.8	0.76	23.0
Me I	49.7	8.6	21.0	6.9	13.8	0.99	15.2
Me II	49.7	6.6	21.8	9.1	12.8	0.98	37.6
Kr I	54.1	9.9	13.4	5.3	17.4	1.18	13.8
Kr II	56.2	9.2	12.5	4.5	17.6	1.28	23.5

Mo and Ox = Normal subjects.
I = Before medication (dicumarol).
II = 48 hr later.

Material. The plasmas of 6 adult persons, 3 of each sex, were studied. Four were patients being treated for intravascular thrombosis, and 2 were normal subjects. The doses of Dicumarol used in each case varied between 500 and 700 mg given in 2 doses about 12 hours apart, the first dose being usually about 100 mg greater than the second. Prothrombin estimations were made immediately prior to the administration of the initial dose and again about 48 hours later. Samples of blood for electrophoresis were obtained at the same time.

Estimations of prothrombin time were made by a method previously described.[4]

Since hemoglobin migrates electrophoretically with γ-globulin, hemolysis was carefully avoided. Plasmas from oxalated blood samples were diluted with 2 volumes of 0.02M sodium phosphate buffer of pH 7.4 which was 0.15M with respect to NaCl, ionic strength 0.2, and dialyzed in the cold against the same buffer solution for one day. Analyses were made in the Tiselius apparatus in the usual manner.

Results. Planimeter measurements and the calculated percentages of the several plasma components are recorded in Table I It is apparent from this table, that no consistent changes occurred in the electrophoretic patterns although there was marked Dicumarol-induced prothrombinopenia. This was true for samples of plasma taken both for 48 and 72 hours after Dicumarol administration. The quantity of prothrombin in plasma is too small to be demonstrated by electrophoresis, therefore, no evidence of its depression was expected in the patterns. Only one patient, Kr, exhibited a significant change in A/G ratio. Before medication, the value of this ratio was 1.18. It rose to 1.28 after 48 hours and dropped to 0.95 when estimated 24 hours later. The fall was a reflection of a change in all components. This patient had generalized arteriosclerosis, circulatory failure and peripheral venous thrombosis. It is possible that the disease processes were responsible for this change. We hesitate to ascribe it to the influence of the drug.

Summary. Dicumarol at therapeutic dosage levels failed to produce alterations in human plasma of sufficient magnitude to be detected by electrophoretic analysis.

[4] Shapiro, S., Sherwin, B., Redish, M., and Campbell, H. A., PROC. SOC. EXP. BIOL. AND MED., 1942, **50**, 85.