ministered by mouth instead of being incorporated in the basal diet. The same substantial difference in the plasma cholesterol levels was encountered during this regimen. The control animal, no. 38, had an average value of 257, nos. 36† and 37†, 381 and 444 mg % respectively. As might be expected, the average plasma level of no. 38 was lowered somewhat because of a reduction in his intake of cholesterol.

Discussion. The greater hypercholesterolemia induced in pyridoxine deficient monkeys by the feeding of cholesterol is of interest. Whether this results from enhanced absorption or a decline in the ability of the vitamin B6 deficient monkey to handle cholesterol is not known. It is possible that cholesterol may be absorbed more efficiently in the pyridoxine-deprived animal than it is in the control. However, this appears to be contrary to what we have observed in the case of glucose. Evidence obtained in this laboratory with the latter indicates that glucose absorption is much more rapid in the animal supplied with pyridoxine. The fact that glucose absorption is decreased in the pyridoxine deficient animal does not prove that cholesterol absorption will also be retarded since they are 2 entirely unrelated chemical compounds. On the other hand, Gubler *et al.*(7) have obtained evidence which they consider indicative of increased iron absorption in the vitamin B6 deficient rat. The results with the animals in Group I do, nevertheless, show that considerable absorption of cholesterol took place in spite of the fact that the diet was low in fat, containing only 2% corn oil.

It is perhaps pertinent to note that the basal diet fed the animals is essentially cholesterol free. In spite of this, animals which have been maintained on this diet over long periods of time showed 'normal' plasma cholesterol levels indicating that cholesterol synthesis is a normal mechanism in the rhesus monkey.

While our studies are not yet completed, to date, we have not found arteriosclerotic lesions in cholesterol fed monkeys which had received adequate supplements of pyridoxine, and the arteriosclerotic lesions of pyridoxine deficiency do not appear to have been enhanced by the administration of cholesterol.

Summary. *Ad libitum* feeding of a low or moderate fat diet containing 1% cholesterol to rhesus monkeys resulted in a greater hypercholesterolemia in the pyridoxine deficient monkey than it did in the control monkey. This occurred despite the fact that the cholesterol intake of the control animal was 2-4 times greater than that of the deficient animals.

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† Deficient.

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Cortisone and Blood Pressure.* (18566)

GEORGE A. PERERA.

From the Columbia University College of Physicians and Surgeons, the Presbyterian Hospital, and the Edward Daniels Faulkner Arthritis Clinic, New York.

It is now generally accepted that the favorable effects of cortisone and the adrenocorticotropic hormone on the manifestations of many disorders are related to the production of some measure of hyperadrenocorticalism (1). The similarities of Cushing's syndrome and the alterations which ensue after cortisone therapy are noteworthy(2). With the ex-

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TABLE I. Effect of Daily Dose of 100 mg Cortisone on Blood Pressure in Subjects Without and With Renal Disease.

Diagnosis	Avg "resting" B.P. For 10 days before cortisone	Avg "resting" B.P. 10th to 20th day of treatment
	A	
Normal	136/ 83	138/ 84
Rheumatoid arthritis	126/ 85	125/ 85
" "	115/ 74	115/ 77
Normal	113/ 64	116/ 62
Rheumatoid arthritis	110/ 70	110/ 70
Eczema	104/ 76	106/ 75
Rheumatoid arthritis	100/ 66	98/ 70
" "	96/ 60	94/ 61
	B	
Malignant hypertension	170/105	181/115
" "	190/118	215/130
Hypertension with nephrosclerosis	155/103	170/118
Hypertension with nephrosclerosis	160/101	186/120
Chronic glomerulonephritis	185/128	210/160
Nephrotic syndrome	110/ 70	136/ 90
Renal amyloidosis	116/ 80	140/108
Disseminated lupus	122/ 65	138/ 76
" "	136/ 90	150/101
Diabetes with nephrosis	118/ 82	138/ 96

ception of data indicating a depressor effect in uncomplicated hypertensive vascular disease and a rise in blood pressure in Addisonian patients(3), there is little information concerning the production of hypertension by cortisone, a conspicuous feature in Cushing's syndrome. In fact, modification of arterial tension by this steroid has usually been regarded as negligible(4).

The widespread use of cortisone in many clinical conditions prompts a review of this problem in both short and long-term studies.

Results. Table IA summarizes observations on "resting" blood pressure(3) in 8 normotensive patients with repeatedly negative urinalyses who received parenteral cortisone† for short periods while in the hospital. No significant changes in arterial tension were evident in this group.

An additional 25 normotensive ambulatory patients, again with no evidence of renal disease, were treated for rheumatoid and other arthritic disorders for periods of from one to 7 months. Some of these received 50 to 75 mg of cortisone by mouth 2 or 3 times daily, others obtained satisfactory clinical responses by intramuscular injections given from one to 4 times per week with weekly dosages varying from 300 to 600 mg. The blood pressure, recorded casually at approximately weekly intervals, showed no significant alterations beyond those which might be attributed to disappearance of fever or increased physical activity, and remained within normal limits (below 140/90) in all but one subject. This patient, taking 25 mg of cortisone orally 3 times per day, developed some edema and transient hypertension up to 160/100 after one month of therapy, but thereafter the majority of readings were normal although occasional diastolic values of 90 to 95 mm of mercury appeared during the third and fourth months.

Table IB summarizes observations in 10 patients with albuminuria in varying disease states in whom nitrogen retention was not a consistent feature. A rise in "resting" blood pressure was noted in every instance irrespective of the presence or absence of antecedent hypertension. No subject in this group developed congestive failure and, judging from weight changes, fluid balance and hematocrit studies, the occasional signs of mild salt and water retention were comparable to the series of patients without albuminuria.

Discussion. The results would indicate that cortisone exerts little influence on the arterial tension of normotensive patients without renal disease treated for several months with the dosages customarily employed. It remains possible, however, that larger doses or therapy continued for longer periods of time, may yet be associated with hypertension similar to that seen in Cushing's syndrome.

On the other hand, rises in both systolic and diastolic pressures were evident within a

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† Purchased from Merck & Co., Rahway, N. J., through funds supplied by the U.S.P.H.S.

few weeks in the group with renal involvement. Whether kidney damage results in greater retention or chemical modification of administered cortisone, or whether the response of the diseased kidney to the steroid is altered, cannot be determined from the present study. It is of interest that hypertension associated with desoxycorticosterone can be achieved more readily in nephritic rats (5). It is recommended that cortisone be used with greater care in patients with renal disease, particularly if antecedent hypertension is evident.

Conclusions. Modification of arterial tension by cortisone, in the doses generally employed, has been noted but rarely in short- and long-term observations of subjects without renal disease. A rise in blood pressure has been the rule among patients with renal damage of varying types irrespective of the presence or absence of antecedent hypertension.

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Specificities of Hyaluronidases Formed by Several Groups of Streptococci.* (18567)

HERBERT A. WENNER, DAVID M. GIBSON,† AND RUTH JAQUES.

From the Departments of Pediatrics and Pathology, and the Hixon Memorial Laboratory, University of Kansas School of Medicine, Kansas City, Kan.

In a previous study(1) enzyme-inhibitors appearing in human serums during streptococcal infections could not be accounted for on the assumption that Group A enzymes were type-specific. It appeared that heterotypic Group A strains of streptococci gave rise *de novo* to analogous enzyme-inhibitors. Since then, some experimental observations have been made in regard to the relative specificities of hyaluronidases produced by Groups A, B, C and G strains of streptococci. These studies form the basis for the present report.

Materials and methods. Streptococcal hyaluronidases. One hundred and fifteen strains of streptococci‡ were studied for hyaluronidase activity. Each strain was grown in buffered Todd-Hewitt broth for a period of 15 to 18 hours. The enzyme titer was determined by the mucin clot prevention test of McClean (1,3). Streptococcal hyaluronidases of satisfactory titer (*e.g.* >1:16) were stored in sealed glass ampoules at −70°C. The number of strains in each group studied and the number producing enough enzyme for working purposes appear in Table I.

Enzyme-inhibitor. Hyaluronidases of high activity (titer of 1:128 or more) were derived from 10 strains of streptococci.§ Bacteria-free filtrates were titered and aliquots of ac-

TABLE I. Hyaluronidase Activity of Streptococci.

Group	No. of strains	Enzyme activity			No. of enzymo producers	% producing enzyme
		<16*	16-128	128-1024		
A	35	27	7	1	8	23
B	7	5	2	0	2	29
C	28	15	10	3	13	46
D	16	16	0	0	0	0
G	28	10	13	5	18	64
K	1	1	0	0	0	0

* MCP test; reciprocal of enzyme dilution.

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† Trainee, National Cancer Institute.

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