3 animals received 10 mg (0.4 cc) of a saline suspension of hydrocortisone acetate (Merck) as well as 1.0 cc of a 1:4 sterile saline suspension of talc in a single intrathecal injection. On the 7th day another dose of 10 mg hydrocortisone was administered by the same route. In order to assess the effect of the hydrocortisone suspension on the meninges, 3 animals received 10 mg of hydrocortisone plus 1.0 cc saline, followed on the 7th day by another equal dose of hydrocortisone. Following the intrathecal administration of hydrocortisone both with and without talc, the animals showed striking convulsive behavior, extensor rigidity, and marked tachypnea for approximately 1 hour following the injection. There was complete recovery in every animal, and no residual neurological abnormalities were noted. No attempt was made to determine whether the hydrocortisone or the vehicle, including 0.9% benzyl alcohol, was responsible for the immediate clinical reaction. One of the animals injected with talc and hydrocortisone died on the 12th day; the cause of death was not ascertained.

The microscopic appearance of the meninges of the 2 surviving animals injected with talc and hydrocortisone showed some diminution of the meningeal reaction, although this effect was not as striking as with cortisone. The animals, however, did not show the emaciation and lethargy characterizing those treated with cortisone, and the weight loss averaged 240 g as compared to a mean weight loss of 625 g in the cortisone group. The gross and microscopic appeance of the meninges of those animals injected only with hydrocortisone was not abnormal.

Summary. Eighteen rabbits received intrathecal injections of 1.0 cc of talc suspension and were sacrificed 15 days later. Eight rabbits were untreated, serving as controls. These developed a meningeal reaction consisting of the appearance of histiocytes, macrophages, and giant cells. Ten animals treated with daily injections of 5 and 10 mg cortisone/kg showed a marked diminution of the meningeal response. Preliminary experiments with the use of hydrocortisone, injected intrathecally (10 mg) on the 1st and 7th days, also suggest diminution of the meningeal response to talc, though not as striking as with the use of intramuscular cortisone.

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Effects of Pressor and Depressor Agents on Pulmonary and Systemic Pressures of Normotensives and Hypertensives.* (20512)

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Hypertrophy of the right ventricle does not occur in uncomplicated essential hypertension (1). This well established observation indicates that pulmonary artery pressure is normal in hypertensive patients. Cournand (2) found normal right ventricular pressures in 5 hypertensives and used this as evidence that the pulmonary arterial system lacked vasomotor function comparable to that of the systemic arterioles. Normal pulmonary arterial pressures have been observed in a few hypertensives by others (3-6).

TABLE I. Effect of Epinephrine, Angiotonin, Nor-epinephrine, and Tetraethylammonium Bromide on Pulmonary and Brachial Pressures of Normotensive Patients.

Patient	Age	Sex	Pulmonary* S/D	Pulmonary* Mean	Brachial* S/D	Brachial* Mean	Pulse
			Control				
WJ	42	♂	29/16	22	100/70	85	100
JB	22	♂	12/0	6	120/60	90	94
PA	24	♂	41/25	27	100/60	80	60
EP	47	♀	20/10	15	110/65	87	60
MH	40	♀	43/6	20	120/70	95	80
JT	31	♂	13/4	8	120/80	100	100
LD	40	♂	16/5	10	100/60	80	80
CM	51	♂	25/7	13	94/60	77	80
JR	57	♂	15/5	8	104/58	81	80
EW	40	♀	14/4	8	100/70	80	80
ES	60	♂	17/7	12	110/60	85	80
VD	52	♂	20/5	14	90/70	85	80
AC	26	♂	12/1	5	120/80	100	110
LH	48	♂	5/0	1	80/50	65	70
AG	30	♀	10/0	3	120/84	102	120
HB	66	♂	9/1	4	80/60	70	60

Patient	Pulmonary S/D	Pulmonary Mean	Brachial S/D	Brachial Mean	Pulse
	Epinephrine				
WJ	40/22	30	130/ 70	100	120
JB	30/0	15	160/ 80	120	140
PA	45/30	40	180/110	145	120
EP	40/15	20	140/ 90	125	80
MH	77/25	50	140/ 80	110	100
JT	40/12	18	190/ 90	140	80
LD	20/7	14	120/ 50	85	100
CM	35/8	20	160/ 80	120	120
JR	18/7	10	140/ 70	105	100
EW	23/5	12	120/ 60	85	100
	Nor-epinephrine				
ES	34/18	25	200/100	150	60
VD	27/10	18	140/ 90	115	50
AC	30/10	18	200/120	160	60
LH	5/0	3	140/ 78	109	50
AG	15/0	6	190/100	145	120
HB	19/6	12	140/ 80	110	80

Patient	Pulmonary S/D	Pulmonary Mean	Brachial S/D	Brachial Mean	Pulse
	Angiotonin				
WJ	12/7	9	140/100	120	60
JB	24/9	14	158/100	129	80
PA					
EP	30/15	23	160/100	135	60
MH	67/18	34	140/110	125	80
JT	38/12	22	190/120	155	60
LD	25/10	16	160/90	125	60
CM	43/18	28	160/140	150	80
JR	20/5	12	160/90	125	80
EW	27/10	18	150/110	130	70
	Tetraethylammonium bromide				
ES	15/0	9	110/60	85	100
VD	3/0	0	74/60	67	120
AC	8/0	3	100/48	90	160
LH					
AG	1/0	0	100/48	74	160
HB					

Dosage: Epinephrine = 0.025 mg I.V.; angiotonin = 5-7 cc I.V.; nor-epinephrine = 0.025 mg I.V.; tetraethylammonium bromide = 300 mg I.V.
* mm Hg.

TABLE II. Effect of Epinephrine, Angiotonin, Nor-epinephrine, and Tetraethylammonium Bromide on Pulmonary and Brachial Pressures of Hypertensive Patients.

Patient	Age	Sex	Pulmonary* S/D	Pulmonary* Mean	Brachial* S/D	Brachial* Mean	Pulse
			Control				
QM	28	♀	12/2	5	138/100	129	84
NL	41	♂	12/2	4	194/110	152	80
ER	52	♂	10/0	4	160/100	130	100
PL	39	♀	18/6	10	190/120	155	100
NL	41	♂	12/2	4	194/110	152	80
EJ	37	♀	25/12	13	200/130	165	120
WW	55	♂	15/3	9	180/120	150	60

Patient	Pulmonary S/D	Pulmonary Mean	Brachial S/D	Brachial Mean	Pulse
	Epinephrine				
QM	30/5	17	180/120	150	120
NL					
	Nor-epinephrine				
ER	42/17	26	280/140	210	50
PL					
NL					
EJ					
WW					

Patient	Pulmonary S/D	Pulmonary Mean	Brachial S/D	Brachial Mean	Pulse
	Angiotonin				
QM	25/9	15	190/140	165	80
NL	50/25	38	220/150	185	100
	Tetraethylammonium bromide				
ER	4/0	1	140/100	120	140
PL	11/3	6	134/100	117	90
NL	3/0	0	160/120	140	120
EJ					
WW					

Dosage: Epinephrine = 0.025 mg I.V.; angiotonin = 5-7 cc I.V.; nor-epinephrine = 0.025 mg I.V.; tetraethylammonium bromide = 300 mg I.V.
* mm Hg.

It was the purpose of this study to measure the pressure in the pulmonary artery in normotensive and uncomplicated hypertensive patients. If the pulmonary artery pressure was the same in normotensives and hypertensives, it was then proposed to compare the responses of the brachial and pulmonary arterial pressures to pressor and depressor agents in these two groups. It was believed that this would yield information pertinent to an understanding of the pathogenesis of essential hypertension.

Pulmonary and brachial arterial pressures of 16 normotensive and 7 hypertensive subjects were studied. The responses of these pressures to angiotonin,* epinephrine, nor-epinephrine,* and tetraethylammonium bromide* were measured. Brachial pressure was obtained by the auscultatory method. The pulmonary artery was catheterized and the pressure was recorded by a Sanborn Electromanometer and Viso-Cardiette. Our usual sedation for this procedure was employed(7).

Results. The data are presented in Tables I and II. They show that the pulmonary artery presure in all normotensives was within the normal range established by Dexter(4) with the exception of patients P. A. and M. H. The elevated pulmonary artery pressure of P. A. is unexplained. Patient M. H. has chronic pulmonary heart disease secondary to pulmonary emboli. These 2 patients have been left in our series because their brachial artery pressures were normal.

The pulmonary artery pressure rose in normotensives whenever epinephrine, nor-epinephrine, or angiotonin caused an elevation of systemic arterial pressure except in W. J. This subject's pulmonary artery pressure fell from 22 to 9 mm Hg with angiotonin. Tetraethylammonium bromide caused a fall in pulmonary artery pressure in all normotensive individuals(8).

Table II shows that hypertensive subjects have normal pulmonary artery pressures. Angiotonin, epinephrine, and nor-epinephrine always produced an elevation of pulmonary arterial pressure in hypertensives if increased systemic pressures resulted from these agents. Tetraethylammonium bromide produced a fall in both pulmonary and systemic artery pressures in hypertensives.

Discussion. We have found that the pulmonary artery pressure in hypertensives is within normal limits (Table II). The response of systemic and pulmonary artery pressures to epinephrine, nor-epinephrine, angiotonin, and tetraethylammonium bromide is the same in hypertensives and normotensives. From this, we have concluded that the vasomotor mechanisms which control the pulmonary arterial and systemic arterial systems are similar if not identical. Inasmuch as the pulmonary artery pressure is normal in hypertensives and increases with epinephrine, nor-epinephrine, and angiotonin, it is improbable that these or any similar substances are responsible for the systemic blood pressure elevation of essential hypertension.

The effect of angiotonin is noteworthy because this substance has been considered so widely to be the cause of the elevated blood pressure in both experimental renal hypertension and essential hypertension. Inasmuch as pulmonary artery pressure is the same in subjects with essential hypertension and in individuals with normal arterial pressure and since angiotonin raises pulmonary and systemic arterial pressure of hypertensive subjects, it is concluded that angiotonin is not the cause of systemic artery hypertension in patients with essential hypertension.

Current opinions concerning whether the terminal pulmonary vascular branches contain constrictor components are conflicting(4,1,9, 10). The known anastomoses(11) of the pulmonary capillary bed with the bronchial arterial system also introduces factors which make difficult the precise interpretation of mechanisms involved in these effects. For these reasons further studies are in progress to elucidate the responsible mechanisms.

Summary and conclusions. The pulmonary and brachial artery pressures and their responses to pressor and depressor agents have

* Dr. Oscar H. Helmer of the Lilly Laboratory for Clinical Research supplied the *angiotonin* and *tetraethylammonium bromide* used in these experiments, and Dr. Kenneth M. Smoot of the Department of Medical Research, Winthrop Stearns Inc. supplied the *Levophed®*. We are grateful for the generous amounts of both these substances.

been studied in normotensive and hypertensive subjects. Pulmonary artery pressure is within the same range in both groups. The response of pulmonary artery pressure to pressor and depressor agents used is also the same in both groups. It is emphasized that the elevation of pulmonary artery pressure due to angiotonin, epinephrine, and nor-epinephrine is the same in hypertensive and normotensive subjects. It is concluded that none of these or any similarly acting substances plays a part in sustaining the blood pressure elevation in patients with chronic essential hypertension.

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Prevention of Dehydroascorbic Acid Diabetes by Atropine.* (20513)

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Dehydroascorbic acid, the reversibly oxidized form of ascorbic acid, produces permanent diabetes in rats(1) and rabbits(2) when multiple large doses are injected intravenously. Immediately following a single intravenous injection of dehydroascorbic acid, there is marked stimulation of the autonomic nervous system which is characterized by salivation, lacrimation, and a rise in blood pressure(3). Atropine administered before the injection of dehydroascorbic acid prevents these effects. Atropine also prevents the diabetogenic effect of dehydroascorbic acid. This paper is concerned with a consideration of this phenomenon.

Experimental. Male Sprague-Dawley rats weighing 108-175 g were injected intravenously with 0.2 g per kilo of dehydroascorbic acid followed in one hour by 0.7 g per kilo on each of 3 consecutive days. The rats receiving atropine sulfate were injected intravenously with 50 mg per kilo of the drug 20 minutes before each of the 0.7 g per kilo injections of dehydroascorbic acid. Those rats with blood sugars greater than 200 mg per 100 cc of blood were considered diabetic. At the end of 2 weeks 8 out of 13 control rats had diabetes, whereas only 1 out of 12 rats receiving atropine developed diabetes (Table I). The P value for this difference is 0.001 indicating that the effect of atropine is highly significant.

A similar experiment was performed on rats that had undergone splenectomy 8-10 days before the injection of dehydroascorbic acid and atropine. In this series (Table I) the atropine did not prevent diabetes.

Discussion. The fact that splenectomy reverses the effect of atropine in preventing dehydroascorbic acid diabetes suggests a peripheral locus for this action of atropine. It may be mediated by a substance produced in

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