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)

JOHN W. PATTERSON.

From the Department of Anatomy, School of Medicine, Western Reserve University, Cleveland, O.

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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TABLE I. Effect of Atropine and Splenectomy on the Development of Dehydroascorbic Acid Diabetes.

	Intact rats		Rats with splenectomy	
	Atropine	Control	Atropine	Control
No.	12	13	5	4
Wt (g)				
Range	108–165	120–175	127–162	135–170
Avg	126	150	147	153
% diabetic				
2 days	58	100	100	100
1 wk	8	84	80	75
2 "	8	61	80	75
Avg 2 wk blood sugar (mg/100 cc)				
Diabetic	260	430	400	390
Non-diabetic	122	140	125	190

the spleen or, since the pancreas and spleen are supplied by a common artery, it may be the result of a change in the relative amount of blood and diabetogenic agent flowing through each organ. The latter of these suggestions seems more probable. Changes in the blood supply to the pancreas are known to prevent alloxan diabetes. The injection of adrenalin(4) or the clamping of the renal pedicle(5) are effective. The production of diabetes with dehydroascorbic acid is similar to injecting alloxan and adrenaline simultaneously. Only a small amount of the diabetogenic agent reaches the islet tissue. Atropine is known to reverse the sympathomimetic effect of dehydroascorbic acid(3). If this reversal is greater in the spleen than in the pancreas, blood would be shunted through the spleen with a decrease in the amount of dehydroascorbic acid reaching the islets. It is claimed that the injection of adrenaline following the administration of a blocking agent causes a dilatation of the splenic vessels(6). If the injection of dehydroascorbic acid produces a similar effect in the atropinized rat the shunting of blood past the pancreas would be even greater.

Summary. The administration of atropine before the intravenous injection of dehydroascorbic acid prevents diabetes. This effect of atropine is not observed in splenectomized rats.

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