TABLE I.

	No. of Animals	Blood Sugar Fast	½ hr.	1 hr.	2 hr.	3 hr.	4 hr.	Liver Glycogen % at 8 hr.	Alanine % absorbed
Normal ♂, Fed Alanine	7	89	113	111	108	108	109	.70	99.04
" " " H_2O	3							.13	
Adrenalectomized ♂, Fed Alanine	7	85	88	98	93	93	86	.29	97.41
" " " H_2O	3							.16	
Normal ♀, Fed Alanine	7							.79	100.00
" " " H_2O	3							.11	
Adrenalectomized ♀, Fed Alanine	7							.24	99.21
" " " H_2O	3							.08	

store glycogen, as reported by Deuel and Butts[3] after glucose administration, does not appear in this experiment. Further work is being continued to determine the course of alanine metabolism in adrenalectomized animals.

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Effect of Transfusion of Blood From Dogs with Experimental Renal Hypertension into Normal Dogs.*

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Ischemia of the dog's kidney, produced by constriction of the renal arteries, results in a chronic hypertension (Goldblatt, Lynch, Hanzal and Summerville[1]). Destruction of the nerves to the kidney does not prevent the development of this hypertension (Page,[2] Collins[3]). Hence it may be supposed that we are dealing with a chemical mechanism. A number of workers (Prinzmetal and Friedman[4]; Harrison, Blalock and Mason[5]; Govaerts and Dicker[6]) have

3 Deuel, H. J., Jr., Gulick, M., Grunewald, C., and Cutler, C. H., *J. Biol. Chem.*, 1934, **104**, 519.

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1 Goldblatt, H., Lynch, J., Hanzal, R. F., and Summerville, W. W., *J. Exp. Med.*, 1934, **59**, 347.

2 Page, I. H., *Am. J. Physiol.*, 1935, **112**, 166.

3 Collins, D. A., *Am. J. Physiol.*, 1936, **116**, 616.

4 Prinzmetal, M., and Friedman, B., PROC. SOC. EXP. BIOL. AND MED., 1936, **35**, 122.

5 Harrison, T. R., Blalock, A., and Mason, M. F., PROC. SOC. EXP. BIOL. AND MED., 1936, **35**, 38.

6 Govaerts, P., and Dicker, E., *Compt. rend. Soc. de biol.*, 1936, **122**, 809.

reported that extracts from the kidneys of dogs with such experimental renal hypertension exert a greater pressor effect than similar extracts from normal kidneys. This finding leads one to believe that the hypertension is due to the liberation of pressor substances into the blood stream from the ischemic kidney. The theory would be greatly supported if increased amounts of pressor substance could be demonstrated in the blood. Dicker[7] in fact reports that the blood of such dogs contains a hypertensive substance, while that of normal dogs does not. However, Page[8] could not find increased pressor substances. Alcohol extracts of the plasma of such dogs, when injected into cats, showed no more pressor activity than similar extracts made from normal plasma. We have attempted in this investigation to demonstrate increased pressor substances in the blood by massive transfusions from large dogs with renal hypertension into small normal dogs. The results have been entirely negative.

The hypertension in the donor dogs was produced by constriction of the renal arteries by a method described previously (Collins[5]). The systolic blood pressures of these animals varied from 180 to 230 mm. Hg. These dogs were large (20-30 kg.), and small dogs (4.5-9 kg.) were chosen as recipients in order that large amounts of blood, in relation to the size of the animal, could be injected. For example in one experiment the recipient dog received an amount of blood equal to 20% of his weight. As the blood was injected, an equivalent amount was withdrawn from the animal through a cannula in the femoral artery, thus avoiding the effects of plethora. The bloods of the 2 animals were always previously checked for incompatibility by a cross agglutination test. This precaution is wise, as Ottenberg, Kaliski and Friedman[9] and also McEnery, Ivy and Pechous[10] have shown. The arterial blood pressure of the recipient dogs was recorded by means of a mercury manometer, connected to a carotid artery. Morphine-ether anesthesia was employed in all except one case, since it does not lower the blood pressure of dogs with experimental renal hypertension. Table I illustrates this fact.

The transfusions were accomplished in two ways. In the first method no anticoagulant was used. The blood was taken from the femoral artery of the donor either with a needle and syringe or by

[7] Dicker, E., *Compt. rend. Soc. de biol.*, 1936, **122**, 476.

[8] Page, I. H., PROC. SOC. EXP. BIOL. AND MED., 1936, **35**, 112.

[9] Ottenberg, R., Kaliski, D. J., and Friedman, S. S., *J. Med. Research*, 1913, **28**, 141.

[10] McEnery, E. T., Ivy, A. C., and Pechous, C. E., *Am. J. Physiol.*, 1924, **68**, 133.

TABLE I.

Time, min.	Arterial blood pressure, mm. Hg.	Time, min.	Arterial blood pressure, mm. Hg.	Time, min.	Arterial blood pressure mm. Hg.
		60	210/108	100	204/104
5	192/118	65	212/108	110	212/114
10	188/106	70	208/106	120	206/118
		Ether anesthesia			
15	186/98	75	196/96	130	198/110
20	190/100	80	188/96	135	204/108
25	196/104	85	190/102		
30	192/98	90	196/100		
Morphine sulphate subcutaneously— 0.01 gm. per kg.		95	198/108		
55	198/96				

cannulation and collection of the blood in a 50 cc. paraffined vessel, and then injected immediately into the recipient dog by means of a cannula in the external jugular vein. This method was necessarily slow, since only 50 cc. of blood could be transferred at one time. In the other method, which involved the use of heparin, the transfer was made in one operation. In this case the blood from the hypertensive dog was obtained from the cannulated renal vein. It is reasonable to suppose that, if the kidney is liberating a pressor substance, this material is present in greater quantities in renal vein blood than in blood drawn elsewhere. Also the kidneys of the recipient dog were tied off just previous to the experiment through lumbar incisions. After the blood had been collected and heparinized, it was immediately injected into the recipient dog through a cannula in the external jugular vein.

Six successful experiments have been performed, and in no case was there any evidence to show that the blood of the dogs with renal hypertension contained more pressor substance than normal blood. The blood pressures of the recipient dogs were not significantly altered by the transfusions of blood which they received from the hypertensive animals.

Summary. The transfusion of blood from dogs with hypertension caused by constriction of the renal arteries into normal dogs does not cause an elevation of blood pressure. In some cases relatively enormous amounts of blood were transferred—as much as 20% of the weight of the recipient. Even when this blood was taken from the renal vein and when the kidneys of the recipient were previously tied off, there was no elevation of the recipient's blood pressure. The method employed, then, gives no evidence for an increased amount of pressor substances in the blood of dogs with hypertension resulting from constriction of the renal arteries.