Correlation between the Electrolyte and Water Content of the Organs and Hypertension after Administration of Corticosteroids

It has been reported that, with renal hypertension, retention of sodium and loss of potassium occurred in the musculature[1-4] and that the same changes in electrolyte metabolism appear after application of hypertension-inducing corticosteroids[5-9]. Therefore these alterations in the inorganic metabolism were considered to be the direct cause of hypertension[10,11]. Tobian *et al.*[12] found an increase in sodium, potassium and water content of the aorta in all forms of experimental hypertension and concluded this to be the cause of hypertension.

Table 1

	Blood pressure		Striated muscle			Aorta*			Bladder*		
	Before treatment	After treatment	Na m.Eq.	K m.Eq.	H_2O (per cent)	Na m.Eq.	K m.Eq.	H_2O (per cent)	Na m.Eq.	K m.Eq.	H_2O (per cent)
Normal (10)	128 ± 3·2		23·45 ± 0·2	105·1 ± 0·85	76·0 ± 0·2	265·2 ± 0·8	149·2 ± 0·55	63·8 ± 0·6	310·9 ± 0·85	352·5 ± 0·7	78·4 ± 0·3
Normal + DOCA (7)	131 ± 3·5	135 ± 3·8	30·05 ± 0·75	88·0 ± 0·95	76·0 ± 0·25	299·3 ± 0·55	147·7 ± 0·65	66·5 ± 0·5	304·3 ± 0·9	354·5 ± 0·6	79·3 ± 0·2
Adrenalectomized + DOCA (7)	127 ± 3·2	168 ± 2·5	32·86 ± 0·5	87·4 ± 1·2	76·33 ± 0·3	313·3 ± 0·85	171·6 ± 0·75	67·7 ± 0·2	334·0 ± 1·2	358·0 ± 0·8	78·6 ± 0·3
Normal + CA (7)	130 ± 2·8	195 ± 4·2	17·90 ± 0·35	97·8 ± 0·5	75·3 ± 0·5	240·1 ± 0·8	171·5 ± 0·4	63·22 ± 0·2	254·0 ± 0·7	352·1 ± 0·6	78·7 ± 0·2
Adrenalectomized + CA (7)	127 ± 3·2	205 ± 4·0	17·1 ± 0·45	101·7 ± 0·6	75·1 ± 0·45	236·2 ± 0·95	180·5 ± 0·55	62·2 ± 0·25	225·2 ± 0·8	360·5 ± 0·7	77·4 ± 0·2
Normal + AA (5)	132 ± 3·5	130 ± 3·0	25·6 ± 0·4	98·6 ± 0·5	75·29 ± 0·3	294·0 ± 0·9	151·2 ± 0·65	67·8 ± 0·3	345·3 ± 0·9	352·7 ± 1·0	79·2 ± 0·3
Adrenalectomized + AA (5)	128 ± 3·1	122 ± 3·0	30·5 ± 0·4	91·5 ± 0·2	76·1 ± 0·3	318·2 ± 0·85	153·3 ± 0·4	68·5 ± 0·4	351·4 ± 0·95	349·1 ± 0·85	79·7 ± 0·2

* M.equiv./kgm. dry tissue.

DOCA, Deoxycorticosterone acetate; CA, cortisone acetate; AA, aldosterone acetate.

In connexion with other researches in the rat, we examined three corticosteroids which influence the electrolyte turnover and the blood pressure in different ways. We also studied their effect on sodium, potassium and water content in the aorta in order to detect which of the changes mentioned above in the electrolyte concentration is related to the blood pressure. The corticosteroids used were deoxycorticosterone acetate (5 mgm./100 gm. body-weight on the first day, then 2·5 mgm.), cortisone acetate (the same dosage) and aldosterone acetate (0·025 mgm./100 gm. body-weight). The treatment lasted 10 days. The daily sodium intake was 75–150 mgm. Sodium, potassium and water content was determined in serum, striated muscle, skin, aorta and in the urinary bladder as another organ containing smooth muscle. With the applied dosage of deoxycorticosterone acetate, the blood pressure remains unchanged in the normal rat (Table 1); as is known, the sodium content of striated muscle is increased and the potassium content is decreased. In the aorta, both sodium and water concentration were increased, whereas the potassium concentration was unchanged.

With adrenalectomized animals the same dosage already produces hypertension. In this case, the sodium as well as the potassium and water content of the aorta is increased; in the urinary bladder an increase in sodium only can be established. It should be emphasized that, by means of the double dosage, hypertension occurs even in normal rats without supplementary supply of sodium chloride.

Cortisone acetate causes hypertension in normal as well as in suprarenalectomized animals. The organs show a depletion of sodium and of potassium, the depletion of potassium being less important than after application of deoxycorticosterone acetate.

It is striking that although the animals survive, the loss of sodium is even more marked than with non-treated adrenalectomized animals. In the aortic wall also the sodium decreases; the potassium, on the contrary, increases strongly.

Aldosterone acetate provokes a retention of sodium in all the organs examined, including the aorta, where the potassium content is almost normal; however, blood-pressure remains unchanged. Thus the experiments show that the variations in sodium concentration of the organs in general or especially in the aortic wall are not conclusive for the production of high pressure.

The sodium content in the aorta can be high with normal blood pressure or even low with high blood pressure. On the contrary, the retention of potassium increases parallel with hypertension. It is assumed that the increase of potassium in the aorta wall causes a rise of the tension of the vascular muscle and therefore hypertension. In rats the diameter and length of the large vessels were measured by arteriography. The increase in tension to induce the blood-pressure determined was evaluated.

L. Laszt

Department of Physiology,
University of Fribourg,
Fribourg,
Switzerland.

1 Eichelberger, L., *J. Exp. Med.*, **77**, 205 (1943).
2 Laramore, D. C., and Grollman, A., *Amer. J. Physiol.*, **161**, 278 (1950).
3 Leonards, J. R., and Heisler, C. R., *Fed. Proc.*, **11**, 247 (1952).
4 Orbison, J. L., Christian, C. L., and Peters, E., *Arch. Path.*, **54**, 185 (1952).
5 Selye, H., Hall, C. E., and Rowley, E. M., *Canad. Med. Assoc. J.*, **49**, 88 (1943).
6 Thorn, G. W., Engel, L. L., and Eisenberg, H., *J. Exp. Med.*, **68**, 161 (1938).
7 Thorn, G. W., Engel, L. L., and Eisenberg, H., *Bull. Hopkins Hosp.*, **64**, 155 (1939).
8 Gaunt, R., Birnie, J. H., and Eversole, W. J., *Physiol. Rev.*, **29**, 281 (1949).
9 Overman, R. R., *Physiol. Rev.*, **31**, 285 (1951).
10 Braun-Menendez, E., and Covian, M. R., *Rev. Soc. arg. Biol.*, **24**, 31 (1948).
11 Braun-Menendez, E., Ciba Foundation Symposium on Hypertension, 238 (Churchill, London, 1954).
12 Tobian, L., and Binion, J. T., *J. Clin. Invest.*, **33**, 1407 (1954). Tobian, L., and Redleaf, P. D., *Amer. J. Physiol.*, **192**, 325 (1958); **189**, 451 (1957). Tobian, L., and Binion, J. T., *Circulation*, **5**, 754 (1952).