

Potassium and Blood Pressure

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ABSTRACT

The relationships between serum potassium and urinary excretion of potassium and blood pressure were determined in an unmedicated adult population with a wide range of blood pressure (mean arterial blood pressure 100-130 mm Hg, n=71).

Inverse correlations between both serum potassium concentration and urinary excretion of potassium and standing (but not supine) mean blood pressure were seen ($r=-0.41$, $p<0.005$ and $r=-0.33$, $p<0.01$ respectively). These relationships persisted also when the influences of age, sex, obesity and kidney function were taken into account in a multiple regression analysis.

The present observation is in accordance with previous reports of an association between potassium metabolism and blood pressure.

INTRODUCTION

For several decades much attention has been paid to the relationship between salt and blood pressure. Also, as early as sixty years ago Addison proposed potassium to be of importance for blood pressure regulation (1). More recently, epidemiological studies have shown the potassium intake, serum potassium and urinary excretion of potassium all to be inversely related to blood pressure (8-10, 19, 21).

To further investigate this matter the relationships between blood pressure and serum potassium and urinary excretion of potassium were determined in a population with a wide range of blood pressure.

MATERIAL AND METHODS

The studied subjects were selected from a general health screening offered every third year to all adult inhabitants in Uppsala, Sweden. Subjects with a diastolic blood pressure (DBP) exceeding 85 mm Hg were re-investigated. If blood pressure still was

elevated on a repeated measurement they were offered a third examination at which also a serum sample was drawn for analysis of potassium and creatinine. Height and weight were recorded, blood pressure was measured and a 24 h urinary collection was performed.

Seventy-one subjects, without history of cardiovascular or antihypertensive treatment, were studied, 37 males and 34 females, mean age 61 ± 8.8 (SD). With the inclusion criteria mentioned above a wide range of blood pressure was obtained at the final investigation (mean blood pressure, MBP 100-130 mm Hg, systolic blood pressure, SBP 120-180 mm Hg, DBP 80-110 mm Hg, mean $159 \pm 13/95 \pm 4.8$ mm Hg).

Serum and urinary potassium was determined by flame photometry while creatinine was measured by a modified Jaffé method. Body mass index (BMI), was calculated by dividing body weight (kg) with the square height (m^2). Blood pressure was measured with a mercury sphyngomanometer in the supine position after 15 minutes of rest and thereafter in the standing position. SBP was read at the appearance of Korotkoff's sounds while DBP was defined as the total disappearance of the sounds. Mean blood pressure (MBP) was defined as $(SBP-DBP)/3 + DBP$.

The relation between single parameters was calculated using Pearson's correlation coefficient while the multiple regression analysis was applied when the relations between several independent variables were studied.

RESULTS

Both serum potassium and urinary excretion of potassium were significantly, inversely, correlated to standing, but not supine, blood pressure with the exception of the correlation between urinary potassium and DBP (Table and Fig 1 and 2). In the multiple regression analysis which included the potential confounding variables age, sex, BMI and serum creatinine both serum potassium and urinary excretion of potassium were significantly related to standing SBP while only serum potassium was significantly related to standing DBP.

DISCUSSION

In this study both serum potassium concentration and the urinary excretion of potassium were found to be related inversely to the standing blood pressure with the exception of the correlation between urinary potassium and DBP.

This finding is in accordance with previous epidemiological studies (3, 11, 12, 19-21) and focus the question how potassium could guard against the development of hypertension.

Relations between serum potassium, urinary excretion of potassium and blood pressure. Pearson's correlation coefficient given with two-tailed p-values in parenthesis. n=71

	<i>Supine</i>			<i>Standing</i>		
	SBP	DBP	MBP	SBP	DBP	MBP
Serum potassium	-0.11 (0.35)	-0.12 (0.30)	-0.14 (0.22)	-0.31 (0.01)	-0.38 (0.001)	-0.41 (0.004)
Urinary excretion of potassium	-0.22 (0.06)	-0.14 (0.24)	-0.07 (0.56)	-0.37 (0.002)	-0.17 (0.17)	-0.33 (0.006)

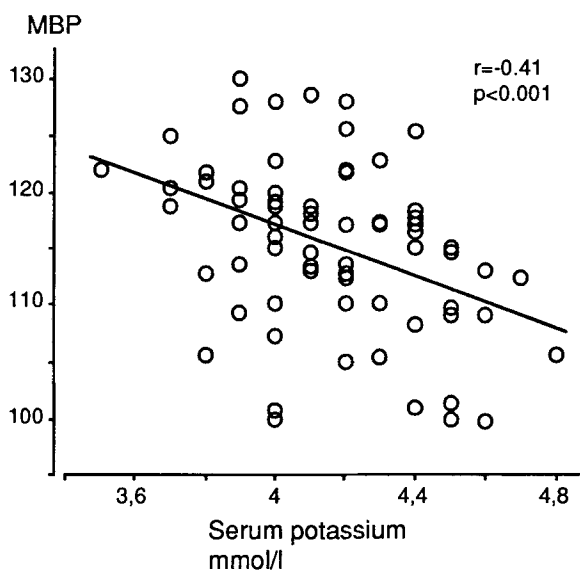


Figure 1 Relationship between serum potassium and standing mean blood pressure ($r=-0.41$, $p<0.004$).

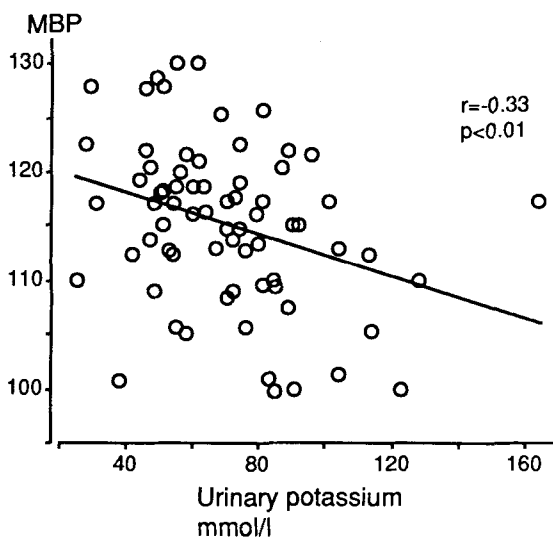


Figure 2

Relationship between urinary excretion of potassium and standing mean blood pressure ($r=-0.33$, $p<0.006$).

Potassium supplementation was found to reduce blood pressure in hypertensive subjects (14, 16). However, further studies demonstrated an antihypertensive effect of potassium only when the sodium intake was high (7, 17). Furthermore, the hypotensive action of potassium supplementation has been reported to be restricted to salt sensitive subjects (15, 18) or to subjects with low renin hypertension (7). In these studies the blood pressure lowering effect of potassium was associated with a natriuresis (7, 18) as well as with decreased pressor response to noradrenalin and angiotensin II (7) and raised levels of plasma prostaglandin E_2 (18). Potassium activates the Na-K-pump (6) which is known to be impaired especially in low renin hypertension (4). It thus seems as if several mechanisms could be involved in a hypotensive action of potassium.

In the present study, a relationship between potassium and blood pressure was seen only in the standing position. The change in posture from supine to standing involves an activation of the sympathetic nervous system, which is known to cause a rapid change in the relationship between extra- and intracellular potassium. A modest dose of adrenaline infusion in healthy volunteers caused a reduction in serum potassium by 10-20 % (13). The relationship between intracellular potassium and blood pressure has been found to be different at different levels of sympathetic activation (2). The inclusion criteria in the present study selected a sample showing a higher blood pressure than normally found in a general population with the same age-

and-sex-distribution. However, only a minor part of the population could be classified as hypertensive according to the WHO criteria. As subjects with a high blood pressure show raised levels of plasma catecholamines (5) an increased activity in the sympathetic nerve system in the present sample might explain the different relations between potassium and blood pressure in the standing and supine positions. Also the discrepancy towards other population studies in which significant relations between potassium and blood pressure were found in the supine position might be explained by the higher blood pressure found in the present study.

In conclusion, the present study demonstrates an inverse relationship between standing (but not supine) blood pressure and both serum and urinary potassium with the exception of the correlation between urinary potassium and DBP. However, the clinical significance of an increased potassium intake has to be further evaluated.

REFERENCES

1. Addison, W.L.T.: The use of sodium chloride, potassium chloride, sodium bromide, and potassium bromide in cases of arterial hypertension which are amenable to potassium chloride. *Can Med Assoc J* 18: 281-285, 1928.
2. Ambrosini, E., Costa, F.V., Borghi, C. & Boschi, S.: Intralymphocytic potassium content and blood pressure: their relationship at rest and during isometric exercise. *Klin Wochenschr* 63 (suppl III): 9-11, 1985.
3. Bulpitt, C.J. & Semmence, A.: Blood pressure and plasma sodium and potassium. *Clin Sci* 61: 852-857, 1981.
4. Burris, J.F., Pamnani, M.B., Hout, S.J., Jemionek, J.F., Freis, E.D. & Haddy, F.J.: Sodium-potassium pump activity in low renin essential hypertension. *Clin Res* 30: 733, 1982.
5. Campere, V., Myers, M.R. & De Quattro, V.: Plasma catecholamines and neurogenic hypertension. *N Engl J Med* 297: 53-60, 1977.
6. Glynn, I.M.: Sodium and potassium movements in human red cells. *J Physiol* 134: 278-310, 1956.
7. Iimura, O., Kijima, T., Kikuchi, K., Miyama, A., Ando, T., Nakao, T. & Takigami, Y.: Studies of the hypotensive effect of high potassium intake in patients with essential hypertension. *Clin Sci* 61: 77s-80s, 1981.
8. Khaw, K.T. & Rose, G.: Population study of blood pressure and associated factors in St Lucia, West Indies. *Int J Epidemiol* 11: 372-377, 1982.
9. Khaw, K.T.: Blood pressure and casual urine electrolytes in 93 London factory workers. *Clin Sci* 65: 1243-1245, 1983.
10. Khaw, K.T. & Barrett-Connor, E.: Dietary potassium and blood pressure in a population. *Am J Clin Nutrition* 39: 963-968, 1984.
11. Langford, H.G.: Dietary potassium and hypertension: epidemiologic data. *Ann Intern Med* 98: 770-772, 1983.
12. Lever, A.F., Beretta-Piccoli, C., Brown, J.J., Davies, J.J., Fraser, R. & Robertson, J.I.S.: Sodium and potassium in essential hypertension. *Br Med J* 283: 463-468, 1981.
13. Ljunghall, S., Joborn, H., Rastad, J. & Åkerström, G.: Plasma potassium and phosphate concentrations - influence by adrenaline infusion, beta-blockade and physical exercise. *Acta Med Scand* 221: 83-93, 1987.
14. MacGregor, G.A., Smith, S.J., Markandou, N.D., Banks, R.A. & Sagnella, G.A.: Moderate potassium supplementation in essential hypertension. *Lancet* ii: 567-570, 1982.

15. Morgan, T.O.: The effect of potassium and bicarbonate ions in the rise in blood pressure caused by sodium chloride. *Clin Sc* 63: 407s-409s, 1982.
16. Siani, A., Strazzullo, P., Russo, L., Gugliemi, S., Iacoviello, L., Aldo Ferrara, L. & Mancini, M.: Controlled trial of long term oral potassium supplements in patients with mild hypertension. *Br Med J* 294: 1453-1456, 1987.
17. Smith, S.J., Markandu, N.D., Sagnella, G.A. & MacGregor, G.A.: Moderate potassium chloride supplementation in essential hypertension: is it additive to moderate sodium restriction? *Br Med J* 290: 110-113, 1985.
18. Tabuchi, Y., Ogihara, T., Gotoh, S., Masuo, K., Hashizume, K. & Kumahara, Y.: Hypotensive mechanism of potassium supplementation in salt-loaded patients with essential hypertension. *J Clin Hypertens* 2: 145-152, 1985.
19. Ueshima, H., Tanigaki, M., Iida, M., Shimamoto, T., Konishi, M. & Komachi, Y.: Hypertension, salt, and potassium. *Lancet* i: 504, 1981.
20. Walker, W.G., Whelton, P.K., Saito, H., Patterson Russell, R. & Hermann, J.: Relation between blood pressure and renin, renin substrate, angiotensin II, aldosterone and urinary sodium and potassium in 574 ambulatory subjects. *Hypertension* 1: 287-291, 1979.
21. Yamori, T., Kihara, M., Nara, Y., Ohtaka, M., Horie, R., Tsunematsu, T. & Note, S.: Hypertension and diet: multiple regression analysis in a Japanese farming community. *Lancet* i: 204-206, 1981.

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