Salt and Hypertension — Controversy

R.H. Al-Badran
Military Hospital
Bahrain Defence Force

CONTROVERSY exists regarding the relationship between the intake of sodium and the development of hypertension. Watson[1] reported a weak but statistically significant positive correlation between systolic blood pressure and urinary excretion rate of sodium.

Similarly Cooper[2] reported a positive relationship between sodium excretion and hypertension in young patients aged 11 to 14 years, stressing that sodium restriction may be important in the prevention of systemic hypertension.

Waren[3] reported that salt restriction results in a modest decline in arterial pressure and increase in plasma renin.

Negroes over 40 years of age and first degree relatives of patients with essential hypertension have been shown to have a decreased Natriuretic capacity compared to normotensive subjects suggesting an inheritable intrinsic renal abnormality in sodium excretion.

Pickering[4] reviewed data concerning the role of sodium intake in the genesis of hypertension and concluded that only very high sodium intake, i.e. over 15gm/day has an impact on blood pressure.

Radcliff[5] suggested that salt excess may be related to hypertension only in genetically susceptible individuals who have a defect in the intrarenal production of dopamine. He points out that normal individuals double their renal dopamine production in response to oral high salt intake. This results in dilatation of renal blood vessels and increased sodium reflux into the renal tubules, which suggests a defect in dopamine production in hypertension. Abnormal cations have been demonstrated in the red cells of patients with essential hypertension.

Canessa [6] reported that red cells of hypertensive patients exchange Lithium for sodium, and by implication sodium for sodium.

Garay [7] reported a low ratio of sodium extrusion to potassium intake causing increased sodium in the red cells, and predicted that sodium transport in arteriolar muscle might be linked to vasoconstriction.

The Lancet[8] suggested that these abnormalities may serve as a marker for essential hypertension rather than play a role in its genesis.

The controversy continues and from a practical standpoint in the therapy of hypertension, until the situation becomes clearer, practising physicians are obliged to advise their hypertensive patients to reduce their salt dietary intake and/or to prescribe agents that reduce the body sodium load.

REFERENCES

1. Watson. Hypertension 2: 1-93, 80.
2. Cooper et al. Circ. 62: 79, 80.
3. Warren et al. Clin Cardial 3: 348,80.
4. Cardiovasc Rev and Rep 1 (1): 13, 80.
5. Cardiovasc Rev and Rep 2 (8): 785, 81.
6. Canessa et al. NEJM 302: 769, 80.
7. Garay et al. NEJMn302: 772, 80.
8. Lancet 2: 891, 82.