

ABSTRACT

There is a need for data on cellular electrolytes and sodium pump activity in many models of hypertension because it has become evident that if sodium is involved in hypertension, it is at the cellular level that it is important. This study was designed to measure leucocyte sodium, potassium, and rate constants for sodium efflux, total and ouabain insensitive, in animal and human hypertension.

In Goldblatt-2-Kidney hypertension, leucocyte electrolytes and rate constants were undisturbed. In Goldblatt-1-Kidney hypertension, leucocyte sodium content was unchanged, potassium concentration was depressed, and the rate constant for total sodium efflux was lower. The passive efflux represented by the ouabain insensitive efflux, remained the same. In Goldblatt-1-Kidney rats drinking 1% saline leucocyte sodium content fell while rate constant for total sodium efflux was increased. Ouabain insensitive efflux was unchanged. In DOCA hypertension, leucocyte sodium content was marginally elevated while the rate constant for total sodium efflux was also

measured. Passive efflux was unchanged.

Leucocytes from patients with essential hypertension had a high sodium content and concentration, and normal potassium content. Potassium concentration was depressed because of a higher cell water. Total sodium efflux rate constant was unchanged as was ouabain insensitive efflux.

In pre-eclampsia, leucocyte sodium content and concentration were elevated while potassium content and concentration were depressed. The rate constant for total sodium efflux was lower while ouabain insensitive efflux was the same. These abnormalities disappeared six months after delivery.

These findings are consistent with a hypothesis that cellular sodium is important in the pathogenesis of hypertension, but that there are at least two schemata for its involvement in the various models.