J.M.KRZESINSKI, A.GOFFARD, X.WARLING, G.L.RORIVE
Service Néphrologie, C.H.U. Sart-Tilman, LIEGE

IS A RENAL ABNORMALITY OF SODIUM EXCRETION THE MARKER OF GENETIC PREDISPOSITION TO HYPERTENSION ? ROLE OF A RENAL IMPAIRED DOPAMINE ELIMINATION.

Since Dahl's observation, a renal defect of sodium excretion is proposed as one of pathogenetic mechanism of hypertension (HTA).

Our study has tried to verify this concept in 20 young normotensives with (n = 12) and without (n = 8) familial predisposition to HTA allowing to test the genetic transmission of such potential renal abnormality of sodium balance.
Each people was submitted to 3 different Na diet (20, 170 and 340 mM NaCl) each for 1 week.

At each visit, blood pressure, vascular resistances, biological values were determined at rest (plasma renin activity, creatinin clearance, 24 h before the test catecholamines, aldosterone and ion urinary excretion). Then 1 liter of isotonic saline was perfused in 30 minutes with measures of blood pressure and 3 h urinary dopamine and Na excretion.

During the low and medium Na diets, but not during the high Na diet, the natriuresis and dopamine excretion were lower in the 3 h urine collection in patients with a family history of HTA ($p < 0.02$ and $p < 0.005$, respectively). No other clinical or biological difference was noted between the 2 groups.

Thus genetic hypertensive predisposition seems to be characterized by a lower Na excretion during acute Na loading in normal or depleted Na diet, linked to an impaired urinary dopamine excretion. These findings suggest that the defect responsible for the susceptibility to sodium intake is at the kidney level. Some dopamine agonists would be of great therapeutic value in treating such patients when blood pressure begins to rise.