



# *Hypertension and the kidney*

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There is a unique relation between the kidney and blood pressure (BP): on the one hand, renal dysfunction (and particularly renal disease) causes an increase in blood pressure, while on the other hand, high blood pressure accelerates loss of function of the diseased kidney.

Transplantation studies both in experimental animals and in humans, provided firm evidence that «blood pressure goes with the kidney», i.e. even a normotensive recipient of a kidney genetically programmed for hypertension will develop hypertension, while conversely hypertensive patients with renal failure receiving the kidney of a normotensive donor may develop normotension. Family studies showed higher blood pressure and more frequent hypertension in first degree relatives of patients with primary glomerulonephritis or diabetic nephropathy, both type 1 and type 2.

The notion that hypertension accelerates the loss of renal function has been proposed at the turn of the century, but definite evidence by observational and intervention studies has only been provided in the last two decades. The issue has been much con-

founded by the mistaken belief that damaged kidneys require higher blood pressure values in order to function properly (Erfordernishochdruck). The mechanism of blood pressure increases in renal disease comprise salt retention, inappropriate activity of the renin angiotensin system and (more recently identified) sympathetic overactivity as well as impaired endothelial cell mediated vasodilatation. There is ample evidence both in primary renal disease (AIPRI and REIN trials) and in nephropathy of type 1 (Lewis trial) and type 2 diabetes (IDNT, RENAAL) that pharmacological blockade of the RAS by ACEi or AT-1-RB has blood pressure-independent renoprotective effects. More recently it has also been shown that blockade of the sympathetic nerve system has blood pressure-independent effects on albuminuria and in animal experiments on progression.

Unresolved problems include particularly the importance of the circadian BP profile (higher renal risk in patients with attenuated nighttime decrease of BP?) and the relation between blood pressure variability on CV organ damage and progression of renal failure.