Introducción

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The kidney plays a crucial role in the maintenance of blood pressure as proposed by Guyton and established in elegant transplantation experiments by Rettig. He showed that hypertension (and normotension) go with the kidney. This is also true in humans as suggested by the observations of Curtis in renal graft recipients. In view of the crucial role of the kidney in blood pressure regulation it is not surprising that hypertension is common in renal disease. Nevertheless, the relation between renal disease and hypertension is not as simple as thought in the past. Studies in families of proposi-ti with diabetic nephropathy and with non-diabetic renal disease showed that, «essential» hypertension was more frequent in first degree relatives, indicating that genetic predisposition to hypertension in somehow linked to the risk of, and potentially also the rate progression of, primary renal disease. Obviously, hypertension- associated genes are candidate genes for renal risk, but the evidence for specific genes is not yet in. Elegant crossing experiments of Jacobson showed that in one rat strain of progressive glomerulosclerosis (fawn hooded rat) blood pressure-independent gene loci code for the risk of glomerulosclerosis. Whether this is part of the explanation of the notable familial aggregation of endstage renal failure in humans remains to be seen.

Blood pressure starts to rise very early in primary renal disease, even before GFR is reduced, presumably because GFR is still maintained within the normal range by an elevation of SNGFR even when renal parenchyma has been reduced. In the past 2 paradigms were proposed to explain hypertension in renal disease, i.e. (a) the tendency to retain sodium and (b) inappropriate activation of the renin angiotensin system. More recently, both experimental and clinical studies showed an important role of the activation of the sympathetic nerve system on blood pressure in renal disease, primarily through stimulatory afferent signals emanating from the diseased kidney. These findings provide a clear rationale for selection of antihypertensive drugs.

One particularly important point is the issue which blood pressure is optimal for renal protection in individuals with renal disease. It has become obvious that blood pressure values much lower than normotension according to WHO and JNC VI are optimal in diabetic and non-diabetic renal disease. The NKF recommendation is target blood pressure of 125/75 mmHg, although such low levels are very difficult to achieve and are associated with considerable side effects.

There is no doubt that effective antihypertensive treatment is one of most powerful instruments for the nephrologist to interfere with progression of renal disease.