No. 1921

To Identify the Role of Renal Sympathetic Nerves in Sodium Diuresis by Comparing the Baroreflex-Mediated Urine Volume Control between Intact and Denervated Kidneys

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Summary

Although renal denervation (RDN) is explored as a new therapy for drug-resistant hypertension, its effects show large inter-individual variability. To identify possible responders beforehand, we need to understand mechanisms behind the hypotensive effect of RDN. During hypotensive stress such as acute bleeding, a reduction of systemic arterial pressure (SAP) causes a reflex increase in sympathetic nerve activity (SNA). The reduction of SAP reduces urine output via a pressure diuresis mechanism. The increase in SNA also reduces urine output via direct renal control. However, an increase in SNA is a cause of some type of hypertension, in which case the effects of SNA and SAP on urine output could be antagonistic. We examined if an increase in SNA reduces urine output when SAP increases following changes in SNA, by using a baroreceptor isolation procedure in anesthetized rats with unilateral RDN. In our experimental setting, SAP increased with SNA. The relationship between SAP and urine output showed a positive slope in the intact side, which suggests that the pressure diuresis mechanism was stronger than the urine output reduction via the direct renal control by SNA. Nevertheless, the slope was significantly greater in the denervated than the intact side, which suggests that the renal SNA counteracted the pressure diuresis mechanism to a certain extent. Renal function estimated by creatinine clearance and fractional sodium excretion did not differ significantly between the intact and denervated sides. The RDN increased urine flow at the operating-point, which suggests that urine output could be maintained at a lower SAP pressure after RDN. The results may explain a previous finding that bilateral RDN caused hypotension but did not affect the water balance or the sodium balance in conscious rats. Further studies are required to elucidate the relative effect of the renal neural control versus the pressure diuresis in hypertension.