No. 2125

Quantitative Analysis of the Effect of Salt Loading on the Balance Between Pressure Diuresis and Renal Sympathetic Antidiuretic Function in Hypertensive Rats

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Summary

Sympathetic nerve activity (SNA) is involved in renal sodium and water excretion. An increase in renal SNA is generally considered to decrease urine output. However, when SNA increases primarily and the elevation of systemic arterial pressure (SAP) ensues, a pressure diuresis effect can antagonize the neurally-mediated anti-diuretic effect. Our previous studies indicate that urine volume increases under the conditions of primary acute sympathetic activation due to the pressure diuresis effect. Further, the slope of urine flow (UF) versus SAP was lower in spontaneously hypertensive rats (SHR) compared with normotensive Wistar-Kyoto rats. Unilateral renal denervation (RDN) was ineffective to increase the slope of UF versus SAP in SHR. The aim of the present study was to elucidate the effect of salt loading on the balance between the neurally-mediated anti-diuretic effect and the pressure diuresis effect. In a unilateral renal denervation model of SHR fed an 8% high-salt diet (SHR-HS), the SAP-UF relationship shifted upward compared with SHR fed with a normal salt diet. There was no significant difference in the SAP-UF relationship between the denervated and intact sides, suggesting a minor role of the renal SNA in increasing the urine output in the SHR-HS. The intravenous administration of telmisartan, an angiotensin II receptor blocker, did not significantly affect the SAP-UF relationship in the SHR-HS. Renin secretion might have been decreased under the high salt diet, and the anti-diuretic effect of angiotensin II was suppressed, resulting in an increase in urine volume. Renal denervation is being investigated as a device therapy for drug-resistant hypertension. Our results indicate that the peripheral effect of the renal sympathetic nerve on the urine output might be smaller than that of pressure diuresis. Further studies are warranted to elucidate the central effect of RDN through the renal afferent pathway.