No. 2029

Quantitative Analysis of the Effect of Renal Denervation on Baroreflex-Mediated Pressure Diuresis and Natriuresis in Hypertensive Rats

Toru Kawada[1], Takuya Nishikawa[1], Hiromi Yamamoto[2]

[1]Department of Cardiovascular Dynamics, National Cerebral and Cardiovascular Center,
[2]Department of Cardiology, Kurashiki Central Hospital, Ohara HealthCare Foundation

Summary

Catheter renal denervation (RDN) is explored as a new device therapy for drug-resistant hypertension, but its antihypertensive effect shows large inter-individual differences. To select proper candidates, we need to clarify the mechanisms underlying the antihypertensive effect of RDN. Increased renal sympathetic nerve activity (SNA) is considered to reduce urine output. The reduction of urine output by increased renal SNA may be beneficial to maintain circulation against blood pressure drops resulting from massive bleeding etc. However, when SNA increases primarily, such as during psychological stress, the antidiuretic effect from the increased SNA and the pressure diuresis effect from the increased arterial pressure can act antagonistically. The purpose of this study was to elucidate the effect of RDN on the pressure-urine output relationship in a unilateral RDN model of spontaneously hypertensive rats (SHR) using a baroreflex open-loop analysis. The SNA and systemic arterial pressure were changed in the same direction via baroreceptor pressure inputs between 60 and 220 mmHg, and urine output was compared between the intact and denervated sides. The results indicated that the arterial pressure and urine output were positively correlated on the intact side. Hence, the pressure diuresis is more potent than the antidiuretic effect via the renal sympathetic nerve. On the other hand, the slope of the pressure-urine output relationship in SHR was less than 1/3 of that in normotensive Wistar-Kyoto rats. The RDN did not significantly increase the slope of the pressure-urine output relationship, suggesting that the ongoing renal SNA was unlikely the culprit for the impaired urine output function in SHR. Further research is warranted regarding the involvement of the renal sympathetic nerve in the arterial pressure elevation during high salt loading in SHR.