Role of renal sympathetic nervous system in the control of renal potassium handling

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ABSTRACT

Background: It is well established that renal sympathetic nerves are primarily involved in renal sodium and water regulation. However, the relationship between renal sympathetic nerve activity (RSNA) and renal potassium handling is not extensively known. The present study was performed to investigate the role of the renal sympathetic nervous system in the regulation of tubular potassium reabsorption and secretion.

Methods: Male Sprague Dawley (SD) rats (each group, n=6) were fasted overnight, anaesthetized with pentobarbital sodium (60 mg/kg intraperitoneal), denervated by application of phenol to the left renal artery and maintained on an intravenous infusion of saline for 2 hours. During this period, 4 urine and plasma samples were collected at 20-minute intervals to study kidney function parameters.

Results: In denervated rats, there were significantly higher (all p<0.05 vs. innervated control) urine flow rate (UFR), glomerular filtration rate (GFR), absolute sodium excretion (U Na⁺), fractional sodium excretion (FE Na⁺), absolute potassium excretion (U K⁺), fractional potassium excretion (FE K⁺) and urinary sodium to urinary potassium ratio (U Na⁺/U K⁺). No appreciable differences were seen in the mean arterial pressure (MAP) and plasma sodium (P Na⁺) between denervated and innervated SD rats. However, plasma potassium (P K⁺) levels were significantly lower (p<0.05) in denervated rats as compared with innervated counterparts.

Conclusions: There is a possible involvement of renal nerves in the regulation of renal potassium handling. This effect is largely attributable to a direct action of renal sympathetic nerves on the renal tubular segments.

Key words: Kidney function, Renal artery, Renal denervation, Renal sympathetic nerve, Sprague Dawley rat

INTRODUCTION

It is well acknowledged that regulation of fluid and electrolytes in the body and, therefore, mean arterial blood pressure homeostasis are major imperative functions of the kidney. One mechanism by which the kidney is believed to control fluid homeostasis is by the action of the renal sympathetic nerves (1). The renal sympathetic nerves enter the hilum of the kidney along with the renal artery and the renal vein and thereafter are distributed along the renal arterial segments in the renal cortex and outer medulla (1-5). In rats, adrenergic sympathetic nerve endings are found on the cells of afferent and effenter arterioles and renal tubules, whereby perturbations in the degree of renal sympathetic nerve activity (RSNA) modulate renal secretion from juxtaglomerular cells and sodium reabsorption from renal tubular cells (5, 6). Many lines of evidence have indicated that removal of renal sympathetic nerve influence by denervation contributes to a marked increase in sodium and water excretion in several mammalian species (7, 8). These effects are principally due to a strong depression of salt and water reabsorption by the proximal convoluted tubule (PCT), with partial compensation by an elevation in the absolute rate of reabsorption in the loop of Henle, distal convoluted tubule (DCT), and collecting duct (7, 5-11).

While adrenergic control of renal sodium handling is well described in both experimental and clinical settings, a considerable paucity of information still exists on the role of the adrenergic mechanisms in the regulation of renal potassium reabsorption and secretion.

Advances in our knowledge of the renal mechanisms of potassium excretion have depended on the application of a wide variety of techniques such as renal clearance, micropuncture, microperfusion and electrophysiological studies.