

UNFORTUNATELY ONLY THE ABSTRACT IS FREELY ACCESSIBLE:

## Renal sodium handling in patients with normal pressure glaucoma

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Low BP (blood pressure) is a recognized risk factor for some patients with NPG (normal pressure glaucoma). We have shown previously that patients with orthostasis have impaired circadian renal handling of sodium, which may contribute to the low BP. Therefore the aim of the present study was to examine the renal handling of sodium, the circadian variations in BP and the neurohormonal response to an orthostatic test in a selected subpopulation of 18 patients with NPG with vasospastic and orthostatic symptoms, and in 24 healthy control subjects. The variations in BP and renal tubular sodium handling were evaluated using 24 h ambulatory BP recordings, 24 h urine collections and determination of endogenous lithium clearance as a marker of proximal sodium reabsorption. The neurohormonal and BP responses to changes in posture were also determined in a 30 min orthostatic test. This selected group of patients with NPG had lower 24 h ambulatory BPs ( $P<0.001$ ), and a more pronounced fall in BP when assuming an upright position ( $P<0.001$ ) compared with controls.  $FE_{Li}$ (fractional excretion of lithium) was higher in patients with NPG than controls during the day ( $36.6\pm21.8$  compared with  $20.4\pm8.7\%$  respectively;  $P<0.01$ ; values are means $\pm$ S.D.) as well as during the night ( $38.8\pm41.9$  compared with  $19.7\pm10.8\%$  respectively;  $P<0.02$ ), suggesting a reduced reabsorption of sodium in the proximal tubule. This was compensated for by an increased distal reabsorption of sodium in patients with NPG ( $P<0.01$ ). These data demonstrate that patients with vasospastic NPG have a high excretion of lithium, suggesting reduced sodium reabsorption in the proximal tubule, in spite of a low BP. The abnormal renal sodium handling might contribute to the maintenance of arterial hypotension and progression of the optic nerve damage in these patients.