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Letter to the Editor

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In their summary of changes in renal function with age, Porush and Faubert[1] do not mention one of the most troublesome and least understood: the reversal of the circadian rhythm of water and electrolyte excretion. Young adults normally excrete the greater part of their 24-hour urine volume during the day and have a low night output.[2] This rhythm has been shown to weaken by the age of 50 years[3,4] and to be lost or apparently reversed by the eighth decade.[3–5] Increased urine production is probably the principal cause of the need to get up at least once at night experienced by practically all elderly people. Together with bladder hyperreflexia,[6] it is also an important cause of nocturnal incontinence.

The cause of the normal circadian rhythm of excretion of water and electrolytes is unknown. Stanbury and Thomson[7] suggested that it might be due to metabolic changes in tubule function associated with sleep and wakefulness. The increased output of potassium in the morning appears to be linked to the corticosteroid cycle,[8] but the accompanying high excretion rates of sodium and water are paradoxical. Electrolyte output is largely unaffected by the glomerular filtration rate[7,8] or by raised arginine vasopressin during sleep which increases tubular reabsorption of water but not of sodium or potassium.[2,7]

Unlike the integrated reversal of the diurnal rhythm which occurs in people working night shifts,[2] the pattern in old age is more variable[5] and more dependent on posture.[9] Potassium is largely unaffected.[5,9] It is therefore probably not due to a brain-mediated shift in the phasing of the rhythm as has been suggested,[5] but is more likely to result from renal tubular changes causing its deterioration and loss. This permits variations in salt and water output caused by renin-angiotensin-aldosterone activity due to posture, which are normally modified by the diurnal rhythm,[10] to become dominant. During the day, the relative inability to excrete a waterload in the upright position[9] causes fluid retention, whilst at night there is a high output of sodium and water due to low aldosterone levels[11] and volume expansion with increased plasma natriuretic peptide concentrations[12] resulting from resorption of fluid from the legs when lying flat in bed.

The nocturesis is particularly severe in chair-bound patients, including elderly patients in hospital who are prevented from resting on their beds during the day. Patients who stop going to bed and spend 24 hours sitting in chairs inevitably develop chronic leg oedema and ulceration due to sodium and water overload, exacerbated by venous and lymphatic stasis.

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