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Kamperis K[1], Hagstroem S[1], Rittig S[2], Radvanska E[1], Djurhuus J C[1]
1. Clinical Institute, University of Aarhus, 2. Dept. of Pediatrics, Aarhus University hospital, Skejby Sygehus

THE EFFECT OF SLEEP ON NOCTURNAL URINE OUTPUT

Hypothesis / aims of study

Aim of this study was to elucidate the impact of sleep on the quantity and quality of the nocturnal urine production in healthy individuals. Our hypothesis was that sleep deprivation is related to excess nocturnal urine production.

Study design, materials and methods

The study protocol was approved by the local Ethics Committee. Twenty healthy volunteers with no history of enuresis, incontinence or nocturia were investigated in the present study. The participants were admitted in the hospital for two 24-hour periods under standardized conditions regarding sodium (2 mmol/kg) and water (25 ml/kg). Normal activities were allowed during the day. Blood samples were drawn every 3 hours and urine was fractionally collected with 3-hour intervals during daytime and following spontaneous voidings at night. During one of the two experimental 24-hour periods subjects were deprived from sleep and the sequence was randomized. During these nights with sleep deprivation, participants were in a lying position in a dimly lit room and physical activities, food and fluid intake were not allowed. Smoking was not allowed throughout the entire experimental protocol. Determinations of electrolytes (Na^+, K^+, Ca^{2+}) creatinine, urea and osmolarity were made in plasma and urine. Blood pressure and heart rate were monitored every hour, using an ambulatory device. Arginine vasopressin (AVP) was measured in plasma by means of radioimmunoassay. Prostaglandin E2 (PGE2) was directly measured in urine using an enzyme immunoassay. 6-sulfatoxy-melatonin (MEL) was measured in urine using an ELISA assay. Clearances, excretions and fractional excretions were calculated for electrolytes, creatinine, urea, osmoles and solute free water. Comparisons were made between the nights with and without sleep deprivation. The circadian rhythm of AVP, PGE2 and MEL was evaluated at baseline and during sleep deprivation.

Results

No significant differences were found in the urinary production at daytime between the two experimental 24-h periods. Males excreted significantly higher amounts of urine on a 24-h basis. During night time and on the nights of sleep deprivation, both males and females produced markedly larger amounts of urine even though the effect was more pronounced for males (males from 1.05 ± 0.10 ml/h/kg to 1.82 ± 0.22 ml/h/kg, $p<0.001$, females from 0.98 ± 0.09 ml/h/kg to 1.41 ± 0.11 ml/h/kg). A similar effect was found for the urinary excretion of sodium (baseline: 0.06 ± 0.01 mmol/kg/h, sleep deprivation: 0.12 ± 0.01 mmol/kg/h), potassium and urine osmolality (baseline: 416 ± 142 mosm/kg, sleep deprivation: 366 ± 66 mosm/kg). No differences were seen in urinary calcium excretion between baseline night and the night with sleep deprivation.

The circadian rhythm in plasma AVP was not influenced by sleep deprivation. In accordance with this, solute free water reabsorption was not significantly different between baseline and during sleep deprivation (baseline 0.45 ± 0.08, sleep deprivation 0.47 ± 0.07 ml/min). We found a significant correlation between hemodynamics as these were assessed by blood pressure and heart rate and the degree of nocturnal polyuria following sleep deprivation.

Interpretation of results

Research into the field of incontinence has therefore during the past years taken sleep related physiological mechanisms into consideration. In the present study we report that acute sleep deprivation has a dramatic effect on the volume of nocturnal urine production in both genders although the effect is more pronounced in males. Natriuresis and kaliuresis were observed on nights with sleep deprivation and were related to differences in hemodynamics between nights with and without sleep deprivation. The circadian rhythms in AVP, PGE2 and melatonin all seem unaffected by sleep deprivation. Furthermore renal water handling was not influenced by sleep deprivation.

Concluding message

Sleep seems to be a major regulator of urine production at night and its deprivation leads to natriuresis, kaliuresis and the production of excess amounts of urine. Altered hemodynamics induced by the deprivation of sleep, seem partly responsible for these processes.