In the arms of Morpheus

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THE RECUPERATIVE POWERS of sleep have been alluded to from as early as 45 BC in Ovid’s Metamorphoses (3), from whence the title of this piece is derived. It was not until the 1900s, with the advent of the first electroencephalographic recording of sleep wave patterns and the discrimination of the different states of sleep, that the science of sleep physiology was born (1). Sleep was demonstrated to be vital to life in a study of total sleep deprivation in rats, in which all rats died within 2–3 wk (12). There is now strong evidence that sleep duration is associated with ill-health and, in particular, obesity (1). More recently, increasing evidence links short sleep duration as a predetermining risk factor for obesity, depression, and cardiovascular disease (1, 4, 9). An important question is the degree to which changes in sleep patterns negatively influence performance, health, and/or quality of life.

Normally, urine production is reduced at night, allowing undisturbed sleep. Mahler et al. (6), in a study presented in an issue of the American Journal of Physiology-Renal Physiology, examined the impact of acute sleep deprivation on urinary output and plasma levels of sodium-retaining hormones important in the regulation of renal function and arterial pressure, in young children (8–10 yr of age). Urine and blood samples were collected periodically over a 24-h period, during which the researchers endeavored to keep the children from sleeping. The results of this study clearly demonstrated that the control subjects that were allowed to sleep had a pronounced circadian rhythm in urinary diuresis and natriuresis, plasma vasopressin, renin, angiotensin II, aldosterone, osmolality, and arterial blood pressure. Acute sleep deprivation caused a loss of the normal circadian decline in urinary water and sodium output. This was associated with a reduced nocturnal dip in arterial pressure and plasma levels of the sodium-retaining hormones.

There were limitations associated with the current study. This study was likely difficult to perform due to the youth of the children, and full 24-h sleep deprivation was only achieved in 50% of subjects. However, this fault is more likely to reduce, rather than overemphasize, the impact of sleep deprivation. No significant change in glomerular filtration rate was observed; however, creatinine clearance was used to estimate glomerular filtration rate. Thus this study may have been underpowered to detect a difference in glomerular filtration rate.

The significance of the work of Mahler et al. (6) relates to two clinically significant problems. The first is enuresis, bed-wetting. The study suggests that the disturbance in sleep-associated rhythms in renal function may be related to enuresis, since the pattern of nocturnal polyuria in enuretic children is similar to that observed during acute sleep deprivation. Thus enuresis may be related to failure of sleep to cause a reflex reduction in arterial pressure and urine production. Taking this further, perhaps enuresis, associated with a reduced decline in nocturnal arterial pressure and renal function, may be a marker for future cardiovascular disease given the evidence that arterial pressure circadian rhythms are linked to cardiovascular disease (7).

The second important question that this study raises is whether chronic short sleep duration alters the regulation of arterial pressure and renal function by increasing sympathetic activity and thus contributes to the development of hypertension. Sleep influences the hypothalamic and cardiovascular control centers in the brain (8), and indeed there is evidence to support the hypothesis that sleep duration is a risk factor for cardiovascular health (7, 9). In adult rats, it has been demonstrated that sleep loss is associated with increased renal sympathetic nerve activity (10). A recent study has shown that alterations in sleep architecture can predict a future risk of hypertension in elderly men (4). Thus alterations in the sympathetic system in response to sleep loss may be an important pathway through which hypertension develops (9).

Moreover, early childhood may be a period of particular vulnerability to the effects of a reduced sleep period. In the postnatal period, tremendous changes in arterial pressure and renal function occur, which can be affected by the postnatal environment (5). This is a time of significant neural and renal plasticity (2), and changes in inputs (the amount of sleep) may permanently alter development in this critical period and program an increased risk of cardiovascular and renal disease. A study has recently demonstrated that rats subjected to the stress of maternal separation have altered sleep patterns in response to later episodes of stress as adults (13). Altering the nutritional environment during the postnatal period has been shown to alter arterial pressure and renal function (see Ref. 5). Short sleep duration has been shown to alter renal sympathetic activity and arterial pressure in adult rats (10), although this has yet to be demonstrated in children. Thus, these studies suggest that short sleep duration in children may alter the circadian control of arterial pressure and renal function and increase the risk of future cardiovascular and renal disease.

In conclusion, sleep deprivation is said to be a widespread and a growing problem in society, including in children (11). The impact of short sleep duration in the developing child may be greatly underestimated and warrants further investigation as it has largely been unexplored.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author.

REFERENCES


