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COMMENTARY

Portable Sleep Monitoring Systems: Broadening the Horizons

Commentary on Levendowski et al. The accuracy, night-to-night variability, and stability of frontopolar sleep electroencephalography biomarkers. *J Clin Sleep Med*. 2017;13(6):791–803.

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The validation study by Levendowski et al.[1] presented in this issue of the *Journal of Clinical Sleep Medicine* expands upon the rapidly evolving field of portable technologies for home sleep testing. A mobile, multichannel electroencephalography (EEG) device was tested against standard polysomnography and results demonstrated acceptable consistency between automated sleep staging and manual scoring.

In addition to generating the classic parameters of macrostructure of sleep, a novel feature of this unobtrusive and relative inexpensive system is the estimation of physiological metrics that have thus far been mostly confined to the research arena. These include quantification of EEG spectral power, sleep spindles, and autonomic activation during sleep. The functional and clinical significance of these measures is increasingly recognized in both healthy subjects and patient populations,[2-4] and may favor broader applications of mobile sleep recording systems, venturing beyond the traditional settings of sleep-disordered breathing (SDB) detection.

Provision of these elaborated metrics, together with accurate qualitative and quantitative sleep estimates obtainable from testing in the habitual sleep environment, make the system described by Levendowski et al.[1] potentially useful for assessment of insomnia. Although objective sleep evaluation is not part of the routine diagnostic apparatus for this disorder, EEG spectral measures could assist in discerning between insomnia phenotypes, as previously suggested.[5,6] Similarly, the autonomic activation index may conceivably be a helpful indicator of the physiological hyperarousal typical of insomnia.[7] If their diagnostic advantages are proven, such elaborated metrics could guide selection of therapeutic approaches, and changes could be tracked to monitor treatment effects to a much greater level of granularity than that yielded by sleep diaries, and even by actigraphy. It is worth noting than when the authors assessed the night-to-night variability of these sleep biomarkers in a clinical sample of patients with insomnia, they found them to be more stable across nights than conventional sleep indexes, which further advocates for their potential clinical utility.

A tangential but interesting and relevant finding reported by Levendowski et al.[1] refers to the significantly more deteriorated sleep recorded in patients with insomnia on pharmacological therapy for elevated blood pressure. Compared to insomnia patients not taking antihypertensive medications, those on treatment presented increased light N2 and reduced N3 sleep stages, along with decreased delta, theta, alpha, and sigma spectral power and impaired autonomic activation in rapid eye movement (REM) sleep. Although these observations may be confounded by the presence of multiple comorbidities in the population studied, and although the cross-sectional nature of the study precludes inference on causality, they nevertheless underscore an aspect too often overlooked in clinic, namely the potential iatrogenic effects of cardiovascular drugs on sleep.

In addition to the established link between SDB and high blood pressure,[8] accumulating evidence also identifies several other sleep disturbances such as insomnia, restless legs syndrome, periodic limb movements during sleep, and chronic sleep deficiency as risk factors for prevalent and incident hypertension.[9] However, hypertension in and of itself may contribute to sleep difficulties, which in turn would further raise blood pressure; hence, these conditions would exacerbate each other in a self-perpetuating cycle, resulting in amplification of cardiovascular risk.[10] In addition to direct mechanistic influences, an indirect, secondary pathway may involve the effect of pharmacotherapy for hypertension.

It has long been known that antihypertensive medications may impair nocturnal sleep, altering sleep duration and sleep architecture, and may also compromise daytime functioning by inducing hypersomnolence. Generally, complaints of sleep difficulties and/or sedation occur more frequently when the pharmacotherapy regimen includes beta-blockers and alpha-2 receptor agonists,[11,12] although occasional sleep symptoms have also been reported with administration of other blood pressure-lowering drugs such as angiotensin-converting enzyme inhibitors[13] and calcium channel blockers.[14] Distinct effects of each pharmacological agent depend on its individual properties (for a review, see Conroy and Brower[15]). Because of their ability to easily cross the blood-brain barrier and penetrate the central nervous system, lipophilic beta-blockers such as propranolol and metoprolol are more disruptive than hydrophilic agents such as atenolol, causing decreased total sleep duration and REM sleep, increased nocturnal awakenings, nightmares, and drowsiness.[16,17] Suppression of nocturnal melatonin synthesis by selective beta-1 adrenergic receptor

antagonists and central alpha-2 receptor agonists may also adversely affect sleep.[18,19]

With regard to patients with preexisting sleep disorders, concerns regarding depressant effects of antihypertensive drugs on breathing in patients with SDB have been largely ruled out, as a recent meta-analysis concluded that they may in fact ameliorate SDB severity by reducing respiratory disturbance index to a small yet significant extent.[20] Nevertheless, it is important to acknowledge that most of the literature on sleep-related complications of blood pressure-lowering medications has been derived from observational studies and from small, short-term randomized studies, which limits generalizability and definitive conclusions. Assessment of the long-term consequences of blood pressure-lowering regimens on both subjective and objective sleep patterns awaits investigation. Furthermore, given that age, sex, and ethnicity may affect vulnerability to both hypertension and sleep disturbances, as well as drug pharmacokinetics and pharmacodynamics, examination of their moderating role should be incorporated into the research agenda as well. For instance, sleep disruption and blunted blood pressure decreases caused by increased nocturia associated with diuretic intake may be more hazardous in the elderly,[21] because of their high-risk status.

To conclude, from a clinical perspective it is critical that both sleep physicians and hypertension specialists be cognizant of the likelihood of undesired sleep sequelae in patients treated with antihypertensive drugs. Close monitoring of development or worsening of sleep disturbances, and consequent treatment adjustments, may help mitigate further medical complications and improve overall quality of life.

CITATION

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