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Journal search and commentary

Article reviewed: Drug-induced arterial pressure elevation is associated with arousal NREM from sleep in normal volunteers ☆

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Category

Sleep-disordered breathing

Objective

To test the hypothesis that elevation of systemic arterial pressure associated with infusion of phenylephrine precipitates arousal from non-rapid eye movement (NREM) sleep.

Study design

Blinded, placebo-controlled.

Study population

Data obtained from five normal subjects.

Methods

Data was obtained from five normal subjects who underwent polysomnographic (PSG) monitoring. Recorded variables included the electroencephalogram (EEG), electrooculogram and electromyogram. Continuous recording of arterial blood pressure was performed using a digital photoplethysmograph (Finapres®, Ohmeda). While subjects were in Stage 2 nREM sleep, infusions of phenylephrine in incremental doses of 50 μg were administered until an increase of mean arterial pressure (MAP) of 20 mmHg or a maximal dose of 250 μg was delivered. Subsequently, during Stage 2 sleep, ten boluses of phenylephrine and ten boluses of volume matched saline were administered in random order and the impact on arousal determined. Arousals were defined by published American Sleep Disorders Association (ASDA) criteria, occurring within 90 s of the bolus delivery.

Results

Phenylephrine administration increased MAP by 24 ± 7 mmHg (mean ± SD) and decreased heart rate by 16 ± 5 beats/min. For the study group of five subjects, 58% of phenylephrine infusions were associated with arousal, compared with 12% of saline infusions ($P = 0.007$).

Conclusion

The authors concluded that drug-induced hypertension precipitates arousals in normal subjects.

☆ Kesler B, Anand A, Launois SH, Weiss JW. Drug-induced arterial pressure elevation is associated with arousal NREM from sleep in normal volunteers. J Appl Physiol 1999:87:897–901.

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Although they did not express the opinion that blood pressure (BP) elevation during apnea contributes to post-apneic arousals, they speculated that the rise in BP during arousal heightens the intensity of arousal.

Comment

It is generally accepted that the hypersomnolence associated with obstructive sleep apnea/hypopnea (OSA/H) is related to frequent arousals from sleep in conjunction with apnea/hypopnea termination. Nonetheless, the degree to which sleep fragmentation translates into daytime sleepiness varies across patients. It therefore behooves clinicians and scientists to obtain a better understanding of arousal physiology. Although much new information has been obtained over the last decade in this regard, it is evident that much remains to be learned. The study by Kesler et al. highlights the complex interaction between cortical and sub-cortical arousal mechanisms and inputs from other sources. It has long been recognized that arousal from sleep precipitates acute BP elevation. The data of Kesler et al. suggests that the opposite is also true, reflecting a positive feedback system.

Even if acute intra-apneic hypertension does not precipitate arousal in OSA/H patients, a positive feedback system of variable potency across patients may modulate the post-apneic arousal intensity producing differences in daytime sleepiness across OSA/H patients despite similar apnea + hypopnea indices.