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Received 11 December 2007; accepted 26 December 2007

Available online 7 March 2008

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doi:10.1016/j.sleep.2007.12.010

The importance of screening for obstructive sleep apnea before surgery

To the Editor:

Anesthesiologists are increasingly seeing more patients with obstructive sleep apnea (OSA) in their practice [1]. In a recent study, 24% of patients in a preoperative clinic were identified to be at high risk for OSA [2]. Perioperative complications are exacerbated by the increased use of patient controlled analgesia [3], and undiagnosed OSA patients may be at a higher risk for perioperative severe respiratory depression.

To illustrate this point, we presented a 66-year-old obese female (BMI-45.3 kg/m^2) with a past medical history of hypertension, asthma, smoking and transient ischemic attack undergoing an open reduction and internal fixation of her wrist under regional anesthesia. Postoperatively, a patient controlled analgesia pump with morphine was used for pain relief. Twelve hours after surgery, she suffered a respiratory arrest requiring resuscitation; narcan was given and she was transferred to ICU. Polysomnography on the second postoperative night showed severe obstructive sleep apnea (apnea hypopnea index = 81) with a mean SaO_2 of 87% and a minimum SaO_2 of 78% while sleeping in room air.

Patients need proper screening for OSA in the preoperative period to prevent postoperative complications. General anesthesia and narcotics can depress the ventilatory response of patients to respiratory obstruction and inhibit their normal arousal and awakening response to hypoxia and hypercapnia, resulting in central respiratory depression.

The American Society of Anesthesiologists recommends the consideration of regional anesthesia techniques to reduce or eliminate the requirements of systemic opioids in patients suspected to have OSA [4]. Regional anesthesia per se does not guarantee safety from the possibility of postoperative respiratory depression, as in this patient. It only delayed the occurrence of respiratory complications by a few hours and it could be potentially dangerous if the patient was discharged home on opioids. This case clearly indicates that there is a need for proper screening for OSA in the preoperative period, and postoperative analgesia must be used cautiously in OSA patients.

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Received 8 February 2008; received in revised form 12 February 2008; accepted 26 February 2008

Available online 20 May 2008

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1389-9457/$ - see front matter © 2009 Published by Elsevier B.V.
doi:10.1016/j.sleep.2008.02.009

Circadian variation of hypocretin-1 (orexin A) in restless legs syndrome

Concerning: Poceta JS, Parsons L, Engelland S, Kripke DF: Circadian rhythm of CSF monoamines and hypocretin-1 in restless legs syndrome and Parkinson's disease. Sleep Med 2008.

To the editor: Although the pathophysiology of Restless Legs Syndrome (RLS) is typically attributed to the dopaminergic system and/or brain iron metabolism, there is some evidence – at least in early onset RLS patients – that the hypocretinergic system might also be affected. Hypocretin-1 (Hrct-1, also named orexin A) was significantly increased in the cerebrospinal fluid (CSF) of early onset RLS patients in one study [1], and tended to be increased in a second one [2] when CSF samples were collected at night (10–11:00 p.m.) or in the evening (6:00 p.m.). In a third study that did not report on the proportion of early onset patients, CSF Hrct-1 in samples drawn at daytime (9:00 a.m.–7:00 p.m.) was normal [3]. In healthy subjects, CSF Hrct-1-levels showed a clear sinusoidal diurnal variation [4]. Together, these observations lead to speculate that the circadian variation of Hrct-1 may be dysregulated in early onset RLS, showing, in particular, elevated nocturnal levels of the peptide.

Therefore, we have read with great interest the article by Poceta et al. [5] who investigated the circadian rhythm of monoamines and Hrct-1 in three patients, two with early and one with late onset RLS, as well as three Parkinson's patients and three controls. Because of an apparent absence of group differences, Hcrt-1 data from all nine subjects were combined and subjected to a Rayleigh test that, in contrast to previous studies [4], did not yield evidence for the assumption of a 24-h Hcrt-1 CSF rhythm. However, we would like to point out that due to the very small number of subjects in each group, the statistical approach chosen by the authors is not appropriate to rule out differences between patients and controls. Therefore, a plausible explanation for the lack of an Hrct-1 circadian rhythm in the combined group is that by pooling the data, intergroup differences in CSF Hrct-1 rhythms and/or concentrations cancel each other out. As to date very little is known about the influence of the hypocretinergic system on RLS; every available information is important for the generation of novel hypotheses. Instead of presenting conclusions alone, it would, therefore, be of great value if the authors published the individual data or at least the mean, median, or range of CSF Hrct-1 concentrations of their subjects.

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Received 22 May 2008; accepted 30 May 2008

Available online 13 November 2008

doi:10.1016/j.sleep.2008.05.006