□ LETTERS TO THE EDITOR □

A Comment on Predicting Continuous Positive Airway Pressure from a Modified Split-Night Protocol in Moderate to Severe Obstructive Sleep Apnea-Hypopnea Syndrome**Key words:** AHI, sleep apnea, split night**(Inter Med 48: 1489, 2009)****(DOI: 10.2169/internalmedicine.48.2156)**

To the Editor I read with interest “Predicting Continuous Positive Airway pressure from A Modified Split-Night Protocol in Moderate to Severe Obstructive Sleep Apnea-hypnea Syndrome” (1). For those individuals with an Apnea-Hypopnea Index (AHI) from 5 events per hour to moderate OSAHS, whether or not the presented model helps is an issue to be clarified. Since the model is said to be helpful for those from moderate to severe OSAHS, it can also be argued that subjects with complaints of poor sleep and mild OSAHS require a diagnostic polysomnography (PSG) in order to explain their clinical symptoms (2-5).

BMI and neck circumference have been the variables that have most often been shown to predict OSAHS and therefore it is not surprising that BMI is used in the presented equation. Furthermore, since the variable of desaturation index (DI) is incorporated in the equation, it is worthwhile to mention the difference between 1999 and 2007 two different American Academy of Sleep Medicine (AASM) criteria. The difference between AASM’s 1999 and 2007 criteria in AHI follows (6, 7).

For example, the recommended hypopnea definition of 2007 has to meet the following criteria:

1) The nasal pressure signal excursions drop by $\geq 30\%$ of baseline.

2) The duration of this drop occurs for a period lasting 10 seconds.

3) There is a $\geq 4\%$ desaturation for pre-event baseline.

4) At least 90% of the event’s duration must meet the amplitude reduction of the criteria for hypopnea.

The aforementioned definition of AASM’s 2007 criteria is somehow different from that of AASM’s 1999 criteria. If the AASM 2007 criteria are used rather than the 1999 criteria, it would result in the deletion of some hypopnea cases in the classification. Such a consideration in the aforementioned difference is applicable to the model reported in that article.

Next, it was noticed that visceral fat accumulation of obese and overweight patients should be considered. The secondary increase of the negative intrathoracic pressure by respiratory efforts may play a role in the pathophysiology of sleep disordered breathing. The standard PSG used in most sleep studies does not include the measurement of esophageal pressure (Pes), which represents the intrathoracic pressure. Hence, the upper airway resistance syndrome is often overlooked because the severity of OSA has already been ‘evaluated’ by the AHI. It appears to be important to know whether or not the model introduced in that article can shed some light on the upper airway resistance syndrome.

Finally, the literature has shown that the total sleep time (TST) is not very different in OSA patients and normal subjects. Hence, the exclusion of TST from the equation reported is reasonable.

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