Obstructive sleep apnea

General characteristics in hypertensive patients,
positional sensitivity,
and upper airway sensory neuropathy

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Abstract

Introduction: Obstructive sleep apnea (OSA) is a highly prevalent disorder, especially in populations with cardiovascular disease. Unfortunately most cases with OSA remain undiagnosed. The ability to identify OSA is important for both the individual and the society, as it is a treatable risk factor for cardiovascular disease and also associated with impaired quality of life. This could be particularly important in populations with cardiovascular disease where the most beneficiary treatment effects could be expected. However, the diagnostic process in OSA may be affected by positional dependency (a majority of OSA patients have more breathing interruptions in supine sleep compared to other sleeping positions). Based on the assumption that individuals have different proportions of supine and non-supine sleep on different nights, positional dependency may be a potential confounder in both diagnosis, classification of OSA severity and evaluation of treatment efficacy. Another aspect of OSA is that the pathogenesis is not fully understood. Data indicate that OSA might be a progressive disease, and many patients report years of snoring before witnessed apneas and symptoms occur. One hypothesis on the pathogenesis of OSA is that long-standing, snoring-induced vibrations cause neurogenic lesions in upper airway tissues, progressively damaging the reflex circuits responsible for keeping the upper airway open during sleep.

Aims: To describe the occurrence of undiagnosed OSA and to identify determinants of moderate/severe OSA in patients with hypertension (*study I*). To describe the prevalence of position dependent OSA (POSA) and its relation to OSA severity classification (*study II*). To compare two methods for quantitative testing of cold sensory function (as a sign of neuropathy) in the upper airway with special focus on test-retest repeatability (*study III*). To evaluate signs of upper airway sensory neuropathy, by cold sensory testing, in non-snorers, snorers, and snoring OSA subjects with special reference to AHI and duration of snoring history (*study IV*).

Methods: In *study I* 411 consecutive patients with hypertension from four primary care health centers in Sweden were evaluated for OSA as measured by the apnea hypopnea index (AHI) through polygraphic (PG) recordings. Different predictors for moderate/severe OSA were evaluated. In *study II* the PG recordings of 265 subjects were specially assessed for POSA and the relation between severity classification based on POSA and traditional OSA severity classification. In s*tudy III* 40 non-snoring subjects were tested for cold detection thresholds at the soft palate and the lip at two separate occasions with two different methods (MLE/MLI). Bland-Altman analysis was used to compare test-retest repeatability. In *study IV* cold sensory testing of the soft palate and lip was used to evaluate signs of upper airway sensory neuropathy in both non-snorers; snorers, and subjects with OSA (groups were formed based on AHI and snoring history, n=90).

Results and Conclusions: Undiagnosed OSA is common in Swedish primary care patients with hypertension, and male gender, BMI>30 kg/m^2, and a clinical history of snoring and witnessed apneas are predictors of moderate/severe OSA. POSA is common both in subjects that by traditional classification had OSA as well as those without OSA. The severity of OSA, if based on total AHI, could be dependent on supine time in a substantial amount of subjects. Cold sensory testing is easily performed in the oropharynx, with acceptable test–retest repeatability. MLI is considerably faster to perform and have a slightly better repeatability than MLE. Therefore MLI should be the used method for cold thermal testing at the soft palate. Both self-reported snoring years and OSA severity are correlated to the degree of cold sensory impairment in the upper airway. Our results strengthen the hypothesis that snoring vibrations may cause a neuropathy in the upper airway, which may contribute to the progression and development of OSA.