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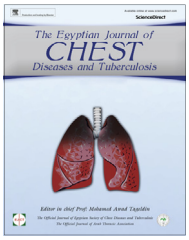

ORIGINAL ARTICLE

Severity of obstructive sleep apnea syndrome in relation to hypoglossal nerve dysfunction

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KEYWORDS

Hypoglossal nerve (HGN) dysfunction;
Obstructive sleep apnea syndrome (OSAS)

Abstract *Background:* Many researchers studied treatment of OSAS by hypoglossal nerve (HGN) stimulation with conflicting results, however few studies addressed the role of HGN dysfunction in the pathogenesis of OSAS.

Aim: Comparison of HGN conduction findings between patients with OSAS and healthy controls as well as correlation of abnormalities with the severity of OSAS.

Methods: 20 patients with mild and 16 with moderate-severe OSAS, and 12 healthy controls were included. All subjects had undergone sleep study (Apnea-hypopnea index, AHI; lowest oxygen saturation, LOS; Oxygen desaturation index, ODI) and HGN study (Latency and amplitude).

Results: Patients with OSAS had significantly higher values for latency and lower values for amplitude than controls ($p < 0.05$). There are non-significant differences between right (Rt) and left (Lt) HGN conduction findings in patients' groups ($p > 0.05$). There are significant differences between the two patients' groups regarding AHI, LOS and ODI ($p < 0.05$). In patients' groups there is significantly positive correlation for latency and negative correlation for amplitude with the severity of OSAS.

Conclusion: There is an evidence of HGN dysfunction even in patients with mild OSAS with significant correlation with the degree of severity. Bilateral assessment of nerve function is essential before treatment with HGN stimulation for proper selection of patients and side to be stimulated.

Introduction

The prevalence of obstructive sleep apnea syndrome (OSAS) is about 2–5% in adults [1]. Several studies have shown associations with systemic hypertension [2], cardiovascular morbidity and mortality [3] and traffic accidents [4] and more recently cancer progression and mortality [5,6].

The genioglossus (GG) muscle, one of the upper airway (UA) dilating muscles (by protruding the tongue) clearly plays

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an important role in physiological maintenance of UA patency and pathophysiology of sleep-disordered breathing conditions, including OSAS [7]. With the onset of sleep, GG muscle activity declines in both subjects with and without OSAS, but more so in subjects with OSAS. This suggests that compensatory reflex mechanisms are impaired during the transition period from wakefulness to sleep [8,9].

The GG muscle is innervated by the medial branch of the hypoglossal nerve and receives input from the central pattern generation and negative pressure receptors within the upper airway. Saboisky et al. confirmed the presence of neurogenic changes and they quantified the extent of neural remodeling in hypoglossal nerve branches [10].

Their study revealed that the genioglossus motor unit potential trains were longer in duration and larger in size index in patients with OSAS versus controls subjects. These changes were attributed to motor unit remolding from denervation, collateral sprouting, and re-innervation and were associated with the severity of hypoxemia during sleep.

Levy and colleagues suggested that pharyngeal neuropathy might be a consequence of sleep disordered breathing (SDB) nevertheless, it may also perpetuate OSAS [11].

Previous studies of electrical stimulation of the UA muscles gave conflicting results [12–15] and more recent studies [16,17] gave favorable results in patients intolerable of continuous positive airway pressure (CPAP).

Aim of the work

The study aimed to compare hypoglossal nerve conduction findings between patients with OSAS and healthy control subjects, and to correlate the conduction abnormalities with the degree of severity of OSAS.

Subjects and methods

The study comprised 12 healthy control subjects, with no history suggestive of OSAS with negative sleep study, and 36 patients with OSAS recruited from the outpatient clinics in Suez Canal University Hospital. All subjects gave informed consent to be involved in the study. One patient was withdrawn from the study because HGN conduction study was technically impossible as well as 5 controls withdrew because they could not tolerate spending whole night in the Sleep Lab. Ten patients (27.8%) were hypertensive and four (11.1%) had recently received a diagnosis of DM.

The following patients were excluded; patients with central or mixed sleep apneas, those with generalized neuro-muscular disorder, those with previous significant facial trauma, head and neck cancer (or received radiotherapy for head and neck cancer, in the past) and patients with implantable devices (e.g. pacemakers).

All subjects were subjected to

(1) Careful history concentrating on history suggestive of SDB, history of peripheral neuritis and diseases associated with OSAS e.g. cardiovascular disease, diabetes mellitus (DM).
(2) Comprehensive physical examination with emphasis on anthrompometric measures such as weight (wt), height (ht), body mass index (BMI), Neck circumference (NC), BP measurement, ENT, chest, cardiac and neurological examination.
(3) Overnight sleep study using SAM equipment, Inter care technologies, model 100, USA, which included flow sensor for detection of apnea and hypopnea, abdominal and, thoracic transducers for detection of respiratory efforts and pulse oximeter for recording arterial oxygen saturation ($SpaO_2$). The following parameters were recorded: apnea–hypopnea index (AHI) i.e., number of apneas and/or hypopneas per hour of sleep (AHI), more than 5/h is considered abnormal, lowest $SpaO_2$ (LOS) and oxygen desaturation index (ODI) i.e. the percentage of time of sleep in which $SpaO_2 < 90\%$.

OSAS was diagnosed based on clinical history consistent with OSAS, supported by overnight sleep study according to the criteria of the American Academy of Sleep Medicine (AASM) [18,19].

Grading of OSAS according to AASM (2005) [19] was done as follows

– Mild OSAS: AHI: 5–15/h.
– Moderate OSAS: AHI: 15–30/h.
– Severe OSAS AHI: >30/h.

(4) Hypoglossal nerve conduction studies (HGNCS) in terms of latency i.e. duration in milliseconds, and amplitude in millivolts, were done on right and left sides using Dantec key point 4 channels electro-myography. The patient was sitting and both recoding reference electrodes, were positioned 2 cm, on a tongue blade on the dorsal surface of the hemi-tongue, over intrinsic muscles of the tongue to study nerves of right and left sides. A ground electrode was placed on the cheek. Bipolar percutaneous stimulation was applied along the base of the mandible, with pressure applied to the stimulator. The intensity of the electrical stimulus was gradually increased until supra-maximal compound muscle action potential (CMAP) wave form was achieved. The latency and amplitude of "CMAPs" were measured from baseline to peak and the results were interpreted according to Redmond and DiBenedetto[20] (Normal latency: 2.04–2.66 ms, Normal amplitude: 3.45–5.43 mV).

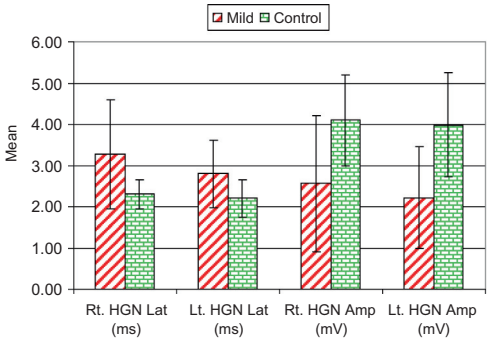

Figure 1 Comparison of nerve conduction findings between patients with mild OSAS and controls.