

Oropharyngeal examination as a predictor of obstructive sleep apnea

Pilot study of gag reflex and palatal reflex

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ABSTRACT

Obstructive sleep apnea (OSA) has high prevalence and may cause serious comorbidities. The aim of this trial was to show if simple noninvasive methods such as gag reflex and palatal reflex are prospective multivariate assessments of predictor variables for OSA. **Method:** We evaluate gag reflex and palatal reflex, of fifty-five adult patients, and their subsequent overnight polysomnography. **Results:** Forty-one participants presented obstructive sleep apnea. The most relevant findings in our study were: [1] absence of gag reflex on patients with severe obstructive apnea ($p=0.001$); [2] absence of palatal reflex on moderate obstructive apnea patients ($p=0.02$). **Conclusion:** Gag reflex and palatal reflex, a simple noninvasive test regularly performed in a systematic neurological examination can disclose the impact of the local neurogenic injury associated to snoring and/or obstructive sleep apnea syndrome. **Key words:** obstructive sleep apnea, OSA, gag reflex, palatal reflex.

Avaliação orofaríngea como preditor da apneia obstrutiva do sono: estudo piloto dos reflexos nauseoso e palatal

RESUMO

A síndrome da apneia obstrutiva do sono (SAOS) possui alta prevalência e pode causar sérias comorbidades. O objetivo deste estudo foi mostrar se métodos não invasivos como os reflexos nauseoso e palatal podem ser avaliações prospectivas multivariadas preditoras para SAOS. **Método:** Avaliamos os reflexos palatal e nauseoso em 55 pacientes adultos, com exame polissonográfico subsequente. **Resultados:** 41 pacientes apresentaram SAOS. Os achados mais relevantes em nosso estudo foram: [1] ausência do reflexo nauseoso em pacientes com SAOS grave ($p=0,001$); [2] ausência do reflexo palatal em pacientes com SAOS moderada ($p=0,02$). **Conclusão:** Os reflexos nauseoso e palatal, um simples exame não invasivo, aplicado em uma avaliação neurológica rotineira, pode revelar o impacto de lesões neurogênicas locais associadas ao ronco e/ou a SAOS.

Palavras-Chave: apneia obstrutiva do sono, SAOS, reflexo palatal, reflexo nauseoso.

Obstructive sleep apnea (OSA) is a common disorder characterized by recurrent episodes of upper airway obstruction during sleep. The upper airway anatomy and neuromuscular functions play a crucial role in the pathophysiology of OSA¹⁻⁵.

Pathogenesis of airway obstruction in patients with OSA remains incompletely

understood⁶. The primary defect is probably an anatomically small or collapsible pharyngeal airway, in combination with a sleep-induced fall in upper airway muscle activity².

As mentioned in the study of Stuck and Maurer⁷, airway anatomic variables related to OSA have been incorporated into complex models involving detailed

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physical or radiographic measurements. Researchers and clinicians used different techniques to reveal potential differences in upper airway anatomy to better understand the pathophysiology of the disease but also to improve patient management and treatment.

There is an evidence for impairment of upper airway mucosal sensory function in the oropharynx of patients with OSA⁸⁻¹⁰. The pathophysiological mechanism is believed to be local neurogenic lesions in the oropharynx caused by the low-frequency vibration of habitual snoring in OSA^{11,12}.

Part of neurological examination is based on stimulus-response approach and the palate and pharynx should routinely be tested in the physical exam, but we assume a normal function of these structures when patients do not complain of dysphagia or choking, and we also rarely test gag reflex (GR) and palatal reflex (PR) in our daily clinics. The absence of GR and PR in the context of the neurological examination were not fully investigated in the scenario of obstructive sleep apnea.

The soft tissue trauma caused by vibration could develop secondary mechanical or inflammatory-related local neuropathy, what has been showed by studies using complex invasive techniques to assess the upper-airway sensation in OSA, such as endoscopic sensory tests, biopsies, and electrical stimulation⁷.

We hypothesize that patients with obstructive sleep apnea have gag and palatal reflexes impaired, hence the aim of this study is to verify if the well known neurological physical examination, including GR, and PR are predictive of pharynx impairment in patients with obstructive sleep apnea syndrome. We justify our study because patient's evaluation already include those approach, the procedures requires no special equipment or skills, are simple to learn, non-invasive, and evidently low costing.

METHOD

Participants

We enrolled 55 consecutive obstructive sleep apnea and control participants from 2009 to 2010 from the Neuro-Sono Sleep Center and São Paulo Hospital Sleep Laboratory, Department of Neurology, Federal University of São Paulo (UNIFESP). The study protocol was approved by the local ethics committee and all participants signed a consent form.

Patients

Forty-one consecutive patients referred to the Neuro-Sono Sleep Center, Department of Neurology, complaining of snoring and daytime sleepiness were examined clinically and had a standard polysomnography (PSG) done. There were 22 male and 19 female, age from 27 to 76 years (55.2±14.2), and body mass index (BMI)

from 18.5 to 46.6 Kg/m² (27.9±15.8). The characteristics of the forty-one patients are presented in Table 1.

Controls

The controlled group included 14 volunteers without snoring and/or sleep apnea, confirmed by polysomnography, randomly recruited from a pool of patients with normal PSG referred to our sleep center mostly for insomnia, parassomnia or periodic limb movements of sleep (PLMS). We matched them to the patients group according to the BMI, age, and gender. There were 7 male and 7 female, age from 17 to 78 years (42.6±14.0), and mean body mass index from 18.3 to 33.0 Kg/m² (24.2±5.3). The characteristics of the fourteen volunteers are presented in Table 1.

Sleep study

All patients had an overnight polysomnography including standard electroencephalographic leads, bilateral electrooculogram, chin and tibialis electromyograms, airflow via nasal pressure cannula and thermistor, thoracoabdominal movements via piezoelectric belt, body position via position sensor, and arterial oxygen-hemoglobin saturation via finger pulse oxymetry. All signals were acquired on a digital data-management system Neurotec model, EQSA-400, Itajuba, MG, Brazil. Studies were scored manually by trained, experienced clinical neurophysiologist blind for the purpose of this study. Sleep-wake state was defined according to standard criteria¹³. Obstructive sleep apnea episodes were defined as cessation of airflow lasting at least 10 seconds with persistent respiratory effort, and hypopneas as episodes of reduction in airflow or inspiratory flow limitation on the nasal cannula pressure signal lasting more than 10 seconds with an associated desaturation of at least 3% or arousal defined according to American Sleep Disorders Association criteria¹³. We considered mild OSAS when apnea-hypopnea index (AHI) was between 5 and 15, moderate when between 15 and 30, and severe when above 30/h¹⁴.

Table 1. Characteristics of participants.

N	Gender	Age	BMI	AHI (N%)
		(mean±SD)	(mean±SD)	
Group Control				
14	07♂ 07♀	42.6±14.0	24.2±5.3	Normal: 14 (100%)
Group Patients				
41	22♂ 19♀	55.2±14.2	27.9±15.8	Mild: 19 (46.4%) Moderate: 6 (14.6%) Severe: 16 (39%)

N: number of participants; SD: standard deviation; AHI: apnea-hypopnea index.

Table 2. Analysis of the absence of palatal reflex and gag reflex, comparing patients with mild, moderate, and severe OSAS to control group.

Group	Reflex	N (reflex absent)	p value (compared to control)
Control N=14	Palatal	1	-
AHI (N%) Normal: 14 (100%) Mild: 0 (0%) Moderate: 0 (0%) Severe: 0 (0%)	Gag	0 (zero)	-
Patients N=41	Palatal	Mild: 2 Moderate: 4 Severe: 5	n/s 0.02 n/s
AHI (N%) Normal: 0 (0%) Mild: 19 (46.4%) Moderate: 6 (14.6%) Severe: 16 (39%)	Gag	Mild: 1 Moderate: 1 Severe: 10	n/s n/s 0.001

N: number of participants; AHI: apnea-hipopnea index; n/s: not significant; OSA: obstructive sleep apnea syndrome.

Palatal reflex

The palatal reflex was obtained during the physical examination of each patient. For all patients, the assessment of scores was done or directly supervised by the same physician. The reflex was assessed by asking the patient to open his/her mouth as wide as possible, while protruding the tongue as far as possible. The patient was instructed to not emit sounds during the assessment. A wooden spatula was placed in contact with the mucosa of right or left anterior pillar of the soft palate stimulating the contraction of soft palate (palatoglossus muscle, Fig 1), in case of Mallampati IV, when necessary we depressed the tongue with another wooden spatula to better see the anterior pillar of the soft palate. Palatal reflex was considered present when the soft palate moves upward or backward. When the reflex was to be absent, the stimulus was repeated after one minute in the contralateral area, to confirm the absence of reflection or not.

Gag (nauseous) reflex

To assess the gag reflex we adopted the same condition above described and we touched the wooden spatula on the mucosa of the posterior wall of the pharynx evoking the gag reflex (Fig 2), in case of Mallampati IV, when necessary we depressed the tongue with another wooden spatula to better see the posterior wall of the pharynx. Gag reflex was considered present when the pharyngeal wall contracted associated or not to soft palate contraction. When the reflex was to be absent, the stimulus was repeated after one minute in the contralateral area, to confirm the absence of reflection or not.

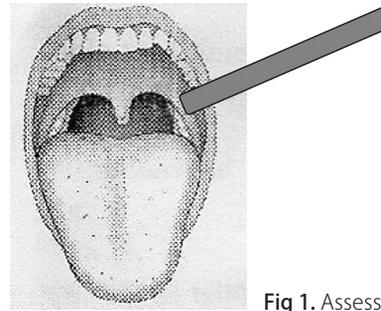


Fig 1. Assessment of palatal reflex.

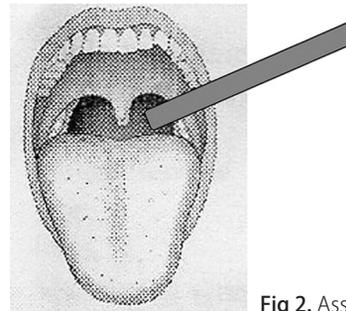


Fig 2. Assessment of nauseous reflex.

Statistical analysis

We used the Chi-square test and Fisher test to analyze the presence or absence of palatal reflex and gag reflex, comparing patients with mild, moderate, and severe OSAS to control group. We considered a p-value <0.05 for statistical significance.

RESULTS

Demographic data

We studied 55 individuals with suspected sleep disorders and who had been referred to the São Paulo Hospital Sleep Laboratory, 29 male and 26 female participants, with age from 17 to 78 years old, and body mass index from 18.3 to 46.6 Kg/m². Overall, forty-one of fifty-five individuals (74.5%) presented obstructive sleep apnea (19 mild, 06 moderate and 16 severe).

Palatal reflex

Overall the palatal reflex was absent on 12 participants (n=55; 21.8%). Palatal reflex was absent just in one patient of the control group (n=14; 7%); in 2 patients from mild group (n=19; 10.5%); in 4 patients from moderate group (n=6; 66.7%); and 5 patients from severe group (n=16; 31%). Compared to control group the absence of palatal reflex was significantly higher only in the moderate OSAS patients (66.7%; p=0.02) (Table 2).

Gag reflex

The gag reflex was absent on 12 participants (n=55; 21.8%) and none of the control group presented absence of gag reflex (n=14; 0%). The gag reflex was absent just

in one patient from mild group (n=19; 5.2%); one from moderate group (n=6; 16.6%); and ten patients from severe group (n=16; 62.5%). Compared to control group the absence of gag reflex was significantly higher only in severe OSAS patients (62.5%; p=0.001) (Table 2).

DISCUSSION

Upper airway (UA) tissues lesions on OSA patients are associated with a slowing of impulse conduction¹⁵, these sensory impairment is caused by axonal degeneration and segmental demyelization in afferent neurons. There is morphologic evidence to support the presence of a sensory neuropathy in the UA in obstructive sleep-disordered breathing¹⁶⁻¹⁸.

Several authors^{9-11,15,19} provided evidence for local neurogenic lesions in OSA. Most of these authors hypothesized that snoring was responsible for the histologic alterations reported.

These observations have led to the suggestion that the progression from mild snoring to heavy habitual snoring and then OSA may represent a progressive local neuropathy²⁰.

Overall we believe our findings are consistent with the UA sensory impairment developing as a consequence of snoring and while remaining partially reversible, representing a largely permanent injury, easily demonstrated by the physical examination of PR and GR, because our data showed that the absence of these reflex, as defined in the study of Davies and Kidd²¹, were more significant when associated to the severity of OSA.

Although, some inconsistent data such as PR, that showed a no significant result with severe OSA group, but with significant result with moderate OSA group, could be analyzed as a bias because of the small sample of participants, and the unknown illness time by the authors.

The UA sensory impairment could potentially represent a defect in the afferent limb of such protective reflexes¹⁰, in concordance with our findings.

The independent association between Gag reflex, Palatal reflex and the presence and severity of OSA suggests that these scoring systems will have practical value in clinical settings and in prospective studies of sleep-disordered breathing.

We remain cautious in asserting that the use of PR or GR as a simple diagnostic test may be sufficient to predict OSAS, as demonstrated in other tests (questionnaires for OSA, outpatient recording). But we hope that future studies with larger numbers of participants, become an opportunity to be a predictor test for OSAS, even as it would be an important tool, easy to perform, which could prioritize patients for polysomnography, and important consideration given the large backlog of patients awaiting assessment for OSA²¹⁻²⁵.

We concluded that gag and palatal reflex were altered on OSA patients, suggesting them as predictor factors as well. Patients with OSA gradually have neurogenic injuries caused by trauma of snore, contributing for the instability of upper airway, proved by complex tests, such as sensory measures by endoscopic methods, and biopsies. Our findings suggest that a simple noninvasive test like GR and PR physical examination, are associated to OSAS. We strongly encourage further trials to corroborate more evidence about PR and GR as predictor factors for OSA.

REFERENCES

1. Schwab RJ. Sleep apnea is an anatomic disorder. *Am J Respir Crit Care Med* 2003;168:270-271.
2. Strohl KP. Sleep apnea is not an anatomic disorder. *Am J Respir Crit Care Med* 2003;168:271-272.
3. Horner RL. Motor control of the paryngeal musculature and implications for the pathogenesis of obstructive sleep apnea. *Sleep* 1996;19:827-853.
4. Fogel RB, Malhotra A, White DP. Sleep 2: pathophysiology of obstructive sleep apnoea/hypopnoea syndrome. *Thorax* 2004;59:159-163.
5. Van Luterén E. Muscles of the pharynx: structural and contractile properties. *Ear Nose Throat J* 1993;72:27-33.
6. White DP. Sleep-related breathing disorder 2: pathophysiology of obstructive sleep apnoea. *Thorax* 1995;50:797-804.
7. Stuck BA, Maurer JT. Airway evaluation in obstructive sleep apnea. *Sleep Med Rev* 2008;12:411-436.
8. Nguyen ATD, Jobin V, Payne R, Beauregard J, Naor N, Kimoff J. Laryngeal and velopharyngeal sensory impairment in obstructive sleep apnea. *Sleep Disord Breathing* 2005;28:585-593.
9. Friberg D, Ansved T, Borg K, Carlsson-Nordlander B, Larsson H, Svanborg V. Histological indications of a progressive snorers disease in an upper-airway muscle. *Am J Respir Crit Care Med* 1998;157:586-593.
10. Kimoff RJ, Sforza E, Champagne V, Ofiara L, Gendron D. Upper airway sensation in snoring and obstructive sleep apnea. *Am J Respir Crit Care Med* 2001;164:250-255.
11. Friberg D, Gazelius B, Lindblad LE, Nordlander B. Habitual snorers and sleep apnoics have abnormal vascular reactions of the soft palatal mucosa on afferent nerve stimulation. *Laryngoscope* 1998;108:431-436.
12. Larsson H, Carlsson-Nordlander B, Lindblad LE, Norbeck O, Svanborg E. Temperature thresholds in the oropharynx of patients with obstructive sleep apnea syndrome. *Am Rev Respir Dis* 1992;146:1246-1249.
13. Rechtschaffen A, Kales A. A manual of standardized terminology, techniques and scoring system of sleep stages in human subjects. Los Angeles: Brain Information Service/Brain Research Institute, University of California, 1968.
14. American Academy of Sleep Medicine. International Classification of Sleep Disorders, Second Edition: Diagnostic and Coding Manual. Westchester, IL: American Academy of Sleep Medicine, 2005
15. MacKenzie RA, Skuse NF, Lethlean AF. A microelectrode study of peripheral neuropathy in man: part 2. Response to conditioning stimuli. *J Neurosci* 1977; 34:175-179.
16. Friberg D, Gazelius B, Hökfelt T, Nordlander B. Abnormal afferent nerve endings in the soft palatal mucosa of sleep apnoics and habitual snorers. *Regul Pept* 1997;71:29-36.
17. Woodson BT, Garancis JC, Toohill RJ. Histopathologic changes in snoring and obstructive sleep apnea syndrome. *Laryngoscope* 1991;101: 1318-1322.
18. Edström L, Larsson H, Larsson L. Neurogenic effects on the palatopharyngeal muscle in patients with obstructive sleep apnoea: a muscle biopsy study. *J Neurol Neurosurg Psychiatry* 1992;55:916-920.
19. Serriès F, Simoneau JA, St Pierre S, Marc I. Characteristics of the genioglossus and musculus uvulae in sleep apnea-hypopnea syndrome and in snorers. *Am J Respir Crit Care Med* 1996;153:1870-1874.
20. Friberg D. Heavy snorer's disease: a progressive local neuropathy. *Acta Otolaryngol (Stockh)* 1999;119:925-933.
21. Davies AE, Kidd D. Pharyngeal sensation and gag reflex in healthy subjects. *Lancet* 1995;346:345.
22. Rahaghi F, Basner RC. Delayed diagnosis of obstructive sleep apnea: don't ask, don't tell. *Sleep Breath* 1999;3:119-124.
23. Escourrou P, Luriau S, Rehel M, Nedelcoux H, Lanoe JL. Needs and costs of sleep monitoring. *Stud Health Technol Inform* 2000;78:69-85.
24. Flemons WW, Douglas NJ, Kuna ST, Rodenstein DO, Wheatley J. Access to diagnosis and treatment of patients with suspected sleep apnea. *Am J Respir Crit Care Med* 2004;169:668-672.
25. Pack AI. Sleep-disordered breathing: access is the issue. *Am J Respir Crit Care Med* 2004;169:666-667.