Journal of Sleep Disorders & Therapy

Hirata and Satoh, J Sleep Disord Ther 2015, 4:4
http://dx.doi.org/10.4172/2167-0277.1000207

Research Article Open Access

Pilot Study of a Nasal Airway Stent for the Treatment on Obstructive Sleep Apnea

Yumi Hirata[1*] **and Makoto Satoh**[2,3*]

[1]*Division of Sleep Medicine, Graduate School of Comprehensive Human Sciences, University of Tsukuba, Japan*

[2]*International Institute for Integrative Sleep Medicine, University of Tsukuba, Japan*

[3]*Ibaraki Prefectural Center for Sleep Medicine and Sciences, Japan*

***Corresponding author:** Makoto Satoh and Yumi Hirata, International Institute for Integrative Sleep Medicine, University of Tsukuba, 1-1-1 Tennodai, Tsukuba, Ibaraki 305-8575, Japan, Tel: +81 29 853 5643, fax: +81 29 853 5643; E-mail: yumi.h.n.19811013@gmail.com, satoh@md.tsukuba.ac.jp

Received date: May 26, 2015, **Accepted date:** Jun 27 2015, **Published date:** Jul 05, 2015

Abstract

Study background: Obstructive sleep apnea (OSA) is a common disease characterized by repetitive upper airway obstruction during sleep. OSA is associated with an increased risk of cardiovascular morbidity. Continuous positive airway pressure (CPAP) has been established as a standard therapy for OSA, but it is not always tolerated by OSA patients.

Objective: In a pilot study, we evaluated the therapeutic effects of the nasal airway stent (NAS), a new nasopharyngeal device placed in the nasopharynx, on OSA and snoring.

Methods: Six subjects with OSA were enrolled. The apnea-hypopnea index, oxygen desaturation index, lowest oxygen saturation, and snore index were analyzed before and during treatment with the NAS.

Results: All subjects tolerated the NAS, with good compliance. Treatment with the NAS ameliorated apnea and hypopnea events and improved oxygen saturation in subjects with mild to moderate OSA. Snoring in all subjects was improved by the NAS.

Conclusions: The NAS stent is a readymade device that could be an alternative therapy for patients with mild to moderate OSA who cannot tolerate CPAP or for subjects with snoring.

Keywords: Nasal airway stent; Obstructive sleep apnea; Sleep medicine; Snoring

Abbreviations:

AHI: Apnea Hypopnea Index; BMI: Body Mass Index; CPAP: Continuous Positive Airway Pressure; HI: Hypopnea Index; NAS: Nasal Airway Stent; ODI: Oxygen Desaturation Index; OSA: Obstructive Sleep Apnea; s-NAS: Single NAS; SpO_2: Oxygen Saturation With Pulse Oximetry

Introduction

Obstructive sleep apnea (OSA) is a common public health problem that affects as much as 4% of the adult population [1,2]. Because OSA is associated with recurrent hypoxia and apnea during sleep, patients are exposed to decreased oxygen saturation, leading to the potential development and exacerbation of not only cardiovascular and cerebrovascular, but also metabolic diseases [3-6]. Several therapies have been developed to treat OSA, including surgery and continuous positive airway pressure (CPAP) [7]. CPAP has been established as a standard therapy for patients with OSA [7-9]. However, although it is effective, low patient tolerance remains an obstacle to be overcome [10,11]. Moreover, patients with OSA may have difficulties with both poor portability of CPAP machines (which can be large and heavy) and lack of availability of electrical power supply during travel. Alternative therapeutic devices that could replace CPAP for short or long periods are therefore required. Some previous studies have reported oral or nasopharyngeal appliances as non-surgical alternatives to CPAP for OSA patients who do not tolerate CPAP treatment [12-18]. Although the concept of nasopharyngeal tube insertion to maintain airflow through the upper airway during sleep was proposed in the 1980s and has been reviewed before [7,12,18], such devices are not currently in common use. The nasal trumpet has been used successfully to decrease airway obstruction in the short term, such as immediately after surgery for OSA [18]. However, the stiffness, length, and consequent discomfort of nasal trumpets mean that they need to be improved for long-term tolerability [18].

We developed a nasal airway stent (NAS), which is a ready-made, non-invasive, and tolerated treatment device for subjects with OSA or snoring. The NAS is specifically designed as a nasopharyngeal stent that maintains the patency of the nasal airway through to the nasopharynx and retropalatal oropharynx during sleep. Although we have developed three kinds of NAS—single NAS (s-NAS), double NAS, and expandable NAS [19] these devices have not yet been manufactured commercially. Here, we performed a pilot study to assess the therapeutic efficacy of the s-NAS on OSA and snoring in subjects with varying degrees of severity of OSA. We verified that the

use of the s-NAS is feasible as a supportive therapy for mild to moderate OSA and snoring.

Materials and Methods

Subjects

The subjects were six male subjects (age, 42.1 ± 8.9 years; body mass index (BMI), 26.6 ± 2.1) who had been previously diagnosed as having OSA by polysomnography. All subjects provided written informed consent after receiving a full explanation of the procedures involved. This study was approved by the University of Tsukuba institutional review committee and was performed in accordance with the recommendations in the Helsinki Declaration.

The s-NAS

The s-NAS is specially designed as a nasopharyngeal stent that maintains the patency of the nasal airway through to the nasopharynx and retropalatal oropharynx by mechanically preventing the upper airway obstruction by the movement and vibration of soft palate during sleep. Unlike currently available nasopharyngeal appliances, the s-NAS is constructed of resilient silicone rubber for the purpose of maintaining the patency of the upper airway by insertion into the nasal cavity. The device consists of a tube-shaped shaft (4.4 mm internal diameter) encapsulated by a water-soluble gel as a lubricant, and a proximal nose clip to fix the device in the appropriate position for optimal treatment efficacy (Figure 1a). The shaft of s-NAS is designed to maintain not to collapse by a pressure of more than 20 cm H_2O in an airflow of 0.1 to 0.51 L/sec—approximately the maximum speed of inspiration under normal breathing conditions. s-NAS devices with a variety of lengths (120 to 145 mm) were manufactured to enable appropriate treatment efficacy for all subjects (Figure 1b) [12]. The distal end of the s-NAS is inserted via the nostril into the nasal cavity until it reaches the retropalatal oropharynx (Figure 1c). Each device comes singly wrapped in a sterile bag. All manufacturing processes were kindly performed by Seven Dreamers Laboratories, Inc., Tokyo, Japan.

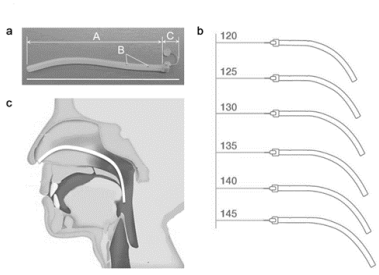

Figure 1: Diagrams of the single nasal airway stent (s-NAS). (a) Overview. A, tube; B, air holes; C, nose clip. White scale bar, 120 to 145 mm. (b) Schematic diagram of s-NAS devices of various lengths. (c) Schematic diagram of nasopharyngeal insertion of the s-NAS. The device is inserted via a nostril into the nasal cavity until it reaches the retropalatal oropharynx.

Study design

Nocturnal respiratory events in all subjects, including oxygen saturation, snoring, and apnea and hypopnea events, were recorded by portable monitoring during treatment with the s-NAS. Physicians determined the appropriate length of the s-NAS for each subject and then instructed and trained all subjects on how to insert the s-NAS into the nasal cavity by themselves to maintain patency of the upper airway. Respiratory events in subjects using the s-NAS were recorded no more than 1 month after the first recording without the s-NAS.

Measurements

Each subject wore a 4-channel portable device (SAS-2100 for airflow, snoring, oxygen desaturation, and heart rate; Nihon Kohden, Tokyo, Japan); this device is categorized as a Level 4 device by the American Academy of Sleep Medicine [20]. Nasal flow, oxygen saturation with pulse oximetry (SpO_2), and snoring were recorded for at least 4 h at night by portable monitoring with the SAS-2100. The apnea-hypopnea index (AHI) and 3% oxygen desaturation index (ODI) were analyzed manually with the QP-021W software (Nihon Kohden). On the basis of the respiratory event scoring of the American Academy of Sleep Medicine [21], apnea was defined as a reduction in amplitude of airflow by more than 90% from the pre-event baseline for no less than 10 sec. Hypopnea was defined as a reduction in amplitude of the airflow by more than 30% from the pre-event baseline for no less than 10 sec, in association with a drop of 3% or more in oxygen saturation. The AHI was calculated as the total number of apnea events per hour (apnea index; AI) plus hypopnea events per hour (hypopnea index; HI) in nocturnal recording periods. The 3% ODI was defined as the total number of 3% desaturations of oxyhemoglobin divided by the total number of nocturnal recording hours; it was represented as the number of events per hour. Snores were analyzed automatically by using the QP-021W software. The snore index was calculated as the number of snores per hour during nocturnal recording periods.

Results

The s-NAS is specifically designed as a nasopharyngeal stent that maintains the patency of the nasal airway through to the nasopharynx and retropalatal oropharynx during sleep (Figure 1). Although nasopharyngeal appliances are currently available, unlike those appliances, the s-NAS is constructed of resilient silicone rubber that enables the upper airway to maintain the patency. The silicone-made shaft of the device is designed to have a resistant to collapse by a pressure of the maximum speed of inspiration under normal breathing conditions. Furthermore, as compared with many of conventional nasopharyngeal appliances, another important feature of the s-NAS is a readymade device that patients readily insert it into the nasal cavity by themselves, leading to the better compliance for patients with OSA and subjects with snoring than currently available nasopharyngeal appliances.

Six male subjects with varying severities of OSA were enrolled in the study. To assess the therapeutic effects of the s-NAS on mild ($5 \leq AHI < 15$), moderate ($15 \leq AHI < 30$), and severe ($30 \leq AHI$) OSA, and on snoring, we evaluated AHI, 3% ODI, the lowest SpO_2, and the snore index before and during treatment with the s-NAS by portable monitoring (Table 1).

Subject		Length of s-NAS (mm)	Sex	Age	BMI (kg/m2)	AHI (/hr)	3% ODI	Lowest SpO2	Snore index
A	Pretreatment		Male	39	24.7	12.7	10.6	88	0.7
	Treatment	135				4.6	5.0	93	0.4
B	Pretreatment		Male	37	25.9	13.1	11.0	73	9.8
	Treatment	130				12.0	11.5	79	0.3
C	Pretreatment		Male	34	26.9	18.5	17.0	85	104.7
	Treatment	130				9.0	9.0	89	24.7
D	Pretreatment		Male	39	24.1	19.8	16.3	82	25.5
	Treatment	130				7.4	6.9	92	5.8
E	Pretreatment		Male	45	29.6	22.4	21.8	70	129.4
	Treatment	130				10.1	10.5	79	93.3
F	Pretreatment		Male	59	28.4	30.4	30.4	77	368.2
	Treatment	130				19.1	17.1	88	18.4

Table 1: Summary of subjects enrolled in the study. (Age, BMI, AHI, 3% ODI, lowest SpO_2, and snore index before and during treatment with the single nasal airway stent (s-NAS) were recorded overnight by portable monitoring with an SAS-2100 monitor and were analyzed with QP-021W software. Respiratory events in subjects using the s-NAS were recorded no more than 1 month after the first recording without the s-NAS).

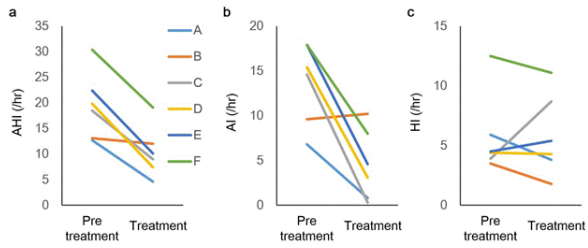

Figure 2: Therapeutic effect of the single nasal airway stent (s-NAS) on apnea-hypopnea index, (AHI), apnea index (AI), and hypopnea index (HI). (a) AHI, (b) AI, and (c) HI before treatment and during treatment with the s-NAS are shown. Subjects A and B had mild OSA, subjects C, D, and E had moderate OSA, and subject F had severe OSA (Table 1).

All subjects tolerated the use of the s-NAS, and compliance of all subjects with the device for a month was confirmed. Treatment with the s-NAS obviously ameliorated mild and moderate OSA, as confirmed by the decrease in 3% ODI, AHI, and AI and the improvement in the lowest SpO_2 (Table 1, Figure 2a and 2b); the device had limited therapeutic impact on HI (Figure 2c). However, the s-NAS had no effect on hypoxic events in one subject with tonsillar hypertrophy (subject B), even though this subject had only mild OSA.

The therapeutic impact of the s-NAS was also limited in subjects with severe OSA, although there was some improvement in their apneic events. Remarkably, s-NAS treatment had a marked therapeutic impact on snoring in all subjects, regardless of the severity of OSA (Table 1). Throughout the clinical trial, no adverse effects, such as nosebleed, pain, discomfort, infection, or allergic responses, were reported following insertion of the s-NAS.

Discussion

In this pilot study we examined the effectiveness of the s-NAS for treating OSA, as determined by the AHI and oxygen desaturation, as well as snoring. We verified the therapeutic efficacy of the device in mild to moderate OSA and in snoring, with good compliance for a month. The s-NAS could be useful as not only a ready-made tool for subjects with OSA or snoring, or both, when traveling, but also as an alternative for OSA patients who cannot tolerate CPAP. Unlike oral appliances the device is immediately available [16]. The s-NAS is designed to maintain airflow through the nasopharynx and retropalatal oropharynx by mechanically preventing the upper airway obstruction by the movement and vibration of soft palate during sleep, but it does not ameliorate the hypopharyngeal airway obstruction theoretically. Thus, the device has a therapeutic effect on the movement and vibration of soft palate during sleep anatomically. Although we did not classified our patients in accordance to the Fujita classification, all subjects who showed ameliorated OSA and snoring by using the s-NAS would be classified in Fujita type I [22]. As we expected, on the basis of features of the s-NAS (length, structure, and design), the device was not effective in one subject with mild OSA and tonsillar hypertrophy and it was unable to normalize the AHI in severe OSA; these findings were consistent with those in previous studies of nasal and oral appliances [12,14,17,18]. Intriguingly, the device improved the number of apnea events, rather than the number of hypopnea events, in subjects with varying degrees of severity of OSA (Figure 2). The precise mechanism by which the device was especially effective in treating apnea events remains undetermined. The s-NAS

may ameliorate episodes of apnea by shifting a total obstruction to a partial obstruction, whereas hypopnea events could be curable, resulting in a decrease in the total AHI. Further randomized clinical trials with a larger number of subjects are required to precisely validate the feasibility of the s-NAS in patients with OSA.

A further structural modifications, such as the production of an expandable NAS with an attachment for expandable parts on its distal end [19], could improve the device's efficacy in treating both apnea and hypopnea events, as well as snoring. The NAS should be beneficial as a tool for OSA therapy in combination with CPAP, oral appliances, surgical approaches, or weight loss programs.

Acknowledgments

We thank Dr. Tsukasa Nabekura (University of California, San Francisco, U.S.A.) for critical comments on the manuscript. We also thank the staff of the Division of Sleep Medicine at the Tsukuba University Hospital for their technical help. This research was supported by grants from the Ministry of Health, Labour and Welfare of Japan.

Conflict of Interest

Seven Dreamers Laboratories, Inc. was not involved in any part of the study apart from the manufacture and provision of the s-NAS.

References

1. Young T, Palta M, Dempsey J, Skatrud J, Weber S, et al. (1993) The Occurrence of Sleep-Disordered Breathing Among Middle-Aged Adults. N Engl J Med 328: 1230-1235.
2. Strollo PJ Jr, Rogers RM (1996) Obstructive sleep apnea. N Engl J Med 334: 99-104.
3. Peppard PE, Young T, Palta M, Skatrud J (2000) Prospective study of the association between sleep-disordered breathing and hypertension. N Engl J Med 342: 1378-1384.
4. Somers VK, White DP, Amin R, Abraham WT, Costa F, et al. (2008) Sleep Apnea And Cardiovascular Disease: An American Heart Association/American College of Cardiology Foundation Scientific Statement from the American Heart Association Council for High Blood Pressure Research Professional Education Committee, Council on Clinical Cardiology, Stroke Council, and Council on Cardiovascular Nursing. J Am Coll Cardiol 52: 686-717.
5. Somers VK, White DP, Amin R, Abraham WT, Costa F, et al (2008) Sleep apnea and cardiovascular disease: an American Heart Association/ american College Of Cardiology Foundation Scientific Statement from the American Heart Association Council for High Blood Pressure Research Professional Education Committee, Council on Clinical Cardiology, Stroke Council, and Council On Cardiovascular Nursing. In collaboration with the National Heart, Lung, and Blood Institute National Center on Sleep Disorders Research (National Institutes of Health). Circulation 118: 1080-1111.
6. Vgontzas AN, Papanicolaou DA, Bixler EO, Hopper K, Lotsikas A, et al. (2000) Sleep apnea and daytime sleepiness and fatigue: relation to visceral obesity, insulin resistance, and hypercytokinemia. J Clin Endocrinol Metab 85: 1151-1158.
7. Liistro G, Aubert G, Rodenstein DO (1995) Management of sleep apnoea syndrome. Eur Respir J 8: 1751-1755.
8. Sullivan CE, Issa FG, Berthon-Jones M, Eves L (1981) Reversal of obstructive sleep apnoea by continuous positive airway pressure applied through the nares. Lancet 1: 862-865.
9. McDaid C, Durée KH, Griffin SC, Weatherly HL, Stradling JR, et al. (2009) A systematic review of continuous positive airway pressure for obstructive sleep apnoea-hypopnoea syndrome. Sleep Med Rev 13: 427-436.
10. Kohler M, Smith D, Tippett V, Stradling JR (2010) Predictors of long-term compliance with continuous positive airway pressure. Thorax 65: 829-832.
11. Weaver TE, Grunstein RR (2008) Adherence to continuous positive airway pressure therapy: the challenge to effective treatment. Proc Am Thorac Soc 5: 173-178.
12. Nahmias JS, Karetzky MS (1988) Treatment of the obstructive sleep apnea syndrome using a nasopharyngeal tube. Chest 94: 1142-1147.
13. Colrain IM, Brooks S, Black J (2008) A pilot evaluation of a nasal expiratory resistance device for the treatment of obstructive sleep apnea. J Clin Sleep Med 4: 426-433.
14. Rossi VA, Winter B, Rahman NM, Yu LM, Fallon J, et al. (2013) The effects of Provent on moderate to severe obstructive sleep apnoea during continuous positive airway pressure therapy withdrawal: a randomised controlled trial. Thorax 68: 854-859.
15. Tsuiki S, Isono S, Minamino O, Maeda K, Kobayashi M, et al. (2012) Tongue position controller as an alternative treatment for obstructive sleep apnea. Sleep Breath 16: 957-960.
16. Lazard DS, Blumen M, Lévy P, Chauvin P, Fragny D, et al. (2009) The tongue-retaining device: efficacy and side effects in obstructive sleep apnea syndrome. J Clin Sleep Med 5: 431-438.
17. Barthlen GM, Brown LK, Wiland MR, Sadeh JS, Patwari J, et al. (2000) Comparison of three oral appliances for treatment of severe obstructive sleep apnea syndrome. Sleep Med 1: 299-305.
18. Kumar AR, Guilleminault C, Certal V, Li D, Capasso R, et al. (2015) Nasopharyngeal airway stenting devices for obstructive sleep apnoea: a systematic review and meta-analysis. J Laryngol Otol 129: 2-10.
19. Satoh M (2012) Effect Of Nasal Airway Stent (NAS) On Obstructive Sleep Apnea. Proceedings of the American Thoracic Society International Conference published in American Journal of Respiratory and Critical Care Medicine.
20. Chesson AL Jr, Berry RB, Pack A; American Academy of Sleep Medicine; American Thoracic Society, et al. (2003) Practice parameters for the use of portable monitoring devices in the investigation of suspected obstructive sleep apnea in adults. Sleep 26: 907-913.
21. Berry RB, Brooks R, Gamaldo CE, Harding SM, Lloyd RM, Marcus CL and Vaughn BV for the American Academy of Sleep Medicine (2013) The AASM Manual for the Scoring of Sleep and Associated Events: Rules, Terminology and Technical Specifications, Version 2.0.2. www.aasmnet.org, American Academy of Sleep Medicine, Darien, Illinois.
22. Fairbanks DN, Fujita S, Ikematsu T (1987) Snoring and Obstructive Sleep Apnea. Raven Press, New York, New York.