# **CLINICAL RESEARCH**

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Received: 2016.01.11 Accepted: 2016.01.21 Published: 2016.02.15		The Effect of Nasal Sept Treatment on Objective Parameters		
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Material/M Con MeSH Ke	<ul> <li>no studies in the English literature have investigated parameters. In this study, we aimed to investigate parameters in those parameters after treatment of N</li> <li>Material/Methods: Nineteen patients diagnosed with NSP were included dure polysomnographies (PSG) after insertion of silic</li> <li>Results: Both median AHI [5.30 (14.40) vs. 2.40 (14.50)] and creased after correction of the perforation. There wa a perforation size &gt;66 mm² [10.10 (34.15) vs. 1.60 (2</li> <li>Conclusions: We conclude that NSP did not cause any deterioration</li> </ul>		d in the study. All patients had baseline and post-proce- one septal button for closure of NSP. d median supine AHI [10.00 (42.10) vs. 6.60 (37.00)] de- s a large reduction in median supine AHI in patients with 8.30)]. fon in objective sleep parameters as determined by PSG, increase in supine AHI. Correction of NSP did not affect ment.	
Full-1	text PDF:	http://www.medscimonit.com/abstract/index/idArt/		
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MEDICAL SCIENCE MONITOR

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Nasal septal perforation (NSP) is a rare disorder characterized by composite loss of mucosa and bone or cartilage that compose the nasal septum [1]. Septal surgery, trauma, inflammatory diseases, and use of topical nasal sprays have been implicated in the etiology of NSP (2, 3). Although most of the patients with NSP are asymptomatic, complaints may occur, depending on the size and location of the perforation. Patients with posterior septal perforations are usually asymptomatic. Anteriorly located small perforations may cause a whistling sound while breathing, and patients with larger anterior perforations complain of dry and blocked nose, crusting, foreign body sensation, and bleeding. Nasal septal perforations may also alter nasal airflow patterns and physiology [2].

Although a number of studies have evaluated the effect of nasal septal deviation and its surgical treatment on sleepdisordered breathing (SDB), there is little data on sleep parameters and SDB in a cohort of patients with nasal septal deviation [3,4]. Similarly, to the best of our knowledge, no studies in the English literature have investigated the effect of NSP and its treatment on polysomnographic parameters.

Therefore, in this study we aimed to investigate polysomnographic sleep and respiratory parameters in patients with NSP as well as changes in those parameters after treatment of NSP. To the best of our knowledge, this is the first such report to be published.

## **Material and Methods**

We conducted a clinical study at the Otorhinolaryngology Department of Ankara Numune Training and Research Hospital. Approval of the local Ethics Committee was obtained before starting the study. All investigations were performed in accordance with the Declaration of Helsinki on biomedical studies involving human subjects, and informed consent was obtained from all participants before the study began.

The participants were selected from patients who presented with NSP and were admitted to the otorhinolaryngology clinic. Prior to the onset of the sleep study, the data were registered for each participant individually, including age, sex, height and weight (to calculate body mass index [BMI]), medical history of comorbid diseases, previous surgeries, and complaints caused by NSP.

Exclusion criteria included nasal problems other than septal perforation (e.g., nasal polyps, chronic rhinosinusitis, allergic rhinitis, and septal deviation), other sleep disorders (e.g., insomnia, hypersomnia, and sleep-related movement disorders), and neurologic and psychiatric disorders.

First, all patients had a detailed otorhinolaryngologic examination, and the location of NSP was determined by diagnostic nasal endoscopy. The size of the NSP was determined via paranasal sinus computerized tomography. The septal perforation width and length were measured in millimeters and septal perforation area was calculated as width × length.

After detailed medical history-taking and physical examination, participants who were found to be appropriate to include in this study completed the Epworth Sleepiness Scale (ESS) questionnaire and underwent a full-night polysomnography (PSG) study.

#### Epworth Sleepiness Scale (ESS)

The ESS is an 8-item questionnaire designed to capture an individual's propensity to fall asleep during commonly encountered situations, on a scale ranging from 0 to 3 [5]. The scores for the 8 questions are added together to obtain a single total score that can range from 0 to 24. In adults, an ESS score  $\geq$ 10 indicates increased daytime sleepiness [5].

#### Polysomnographic evaluation

We used the Alice 5 PSG (Alice<sup>®</sup> 5 Diagnostic Sleep System, Philips Respironics, Philips Healthcare, the Netherlands) device to record baseline and post-procedure PSG. All PSGs were performed during at least 7 hours of spontaneous sleep, under the supervision of an experienced sleep technician in the Sleep Center of Ankara Numune Education and Research Hospital. Audio and video recordings were also obtained. The channels included in PSG were 4-channel electroencephalography, 3-channel electromyography (mentalis, and right and left tibialis anterior muscles), 2-channel electrooculography (right and left eyes), 2-channel electrocardiography, nasal airflow, thoracic and abdominal respiratory movements, pulse oximetry, and body position.

Sleep and respiratory parameters were evaluated according to the American Academy of Sleep Medicine (AASM) criteria [6], consisting of non-rapid eye movement (NREM) stages N1, N2, N3, and REM stage during sleep. Stage 1 (N1) sleep represents a transition from wakefulness to sleep. Stage 2 (N2) represents established NREM sleep. Stage 2 is identified by the presence of K-complexes or sleep spindles (or both). Stage 3 (N3) represents the deepest stages of sleep and is also described as slow-wave sleep. Rapid eye movement (REM) sleep is commonly described as paradoxical sleep and is characterized by a highly activated brain within a paralyzed body. REM sleep is identified by the combination of 3 conditions: (1) the

	Baseline Mean ±SD	Post-procedure Mean ±SD	Р
Sleep efficiency(% TIB)	86.89±7.68	85.17±11.23	0.364
N1 (% TST)	4.90±3.67	4.30±2.02	0.555
N2 (% TST)	55.55±8.23	53.44±10.95	0.468
N3 (% TST)	24.79±8.84	25.81±11.82	0.690
REM (% TST)	15.4±5.2	16.4±4.2	0.398
Median AHI-TST (IQR)	5.30 (14.40)	2.40 (14.50)	0.387
Median AHI-REM (IQR)	4.50 (17.10)	6.20 (20.60)	0.494
Median AHI-NREM (IQR)	4.30 (12.60)	2.40 (10.80)	0.828
Median supine AHI (IQR)	10.00 (42.10)	6.60 (37.00)	0.777
Mean PO2 (%)	95.10±0.99	95.31±1.16	0.297
Minimum PO2 (%)	87.17±5.50	87.17±6.56	1.000

Table 1. Comparison of baseline and post- procedure polysomnographic findings.

TIB – total time in bed; TST – total sleep time; AHI – apnea hypopnea index; REM – rapid eye movement sleep; NREM – non-rapid eye movement sleep; IQR – interquartile range; PO2 – partial oxygen pressure.

EEG returns to a relatively low-voltage, mixed-frequency pattern within cessation of sleep spindles, K-complexes, and highamplitude slow waves; (2) the chin EMG falls to the lowest level of the recording; and (3) the EOG channels demonstrate the presence of rapid eye movements [6]. PSG records were scored according to the guidelines American Academy of Sleep Medicine Manual for Scoring of Sleep and Associated Events, version 2.0 (2014) [7,8].

# Treatment of septal perforation and postoperative evaluation

Following acquisition of baseline PSG data, a silicone septal button was placed under local anesthesia to cover the perforation, and complete closure of the perforation was confirmed by endoscopic nasal examination.

A post-procedure PSG was obtained at least 7 days after the insertion of the septal button.

PSG parameters, including sleep efficiency, durations of stages 1, 2, and 3 and REM, total apnea hypopnea index (AHI), REM AHI, non-REM AHI, and mean and lowest PO2 saturation were compared between the baseline and post-procedure recordings.

These parameters were also compared in relation to perforation size. Baseline and post-procedure parameters related to sleep were also compared after dividing the patients into 2 groups, those with AHI <5 and those with AHI  $\geq$ 5.

#### Statistical analysis

Statistical analysis was performed using SPSS version 11.5 for Windows (SPSS, Inc., Chicago, IL). To compare baseline and post-procedure PSG data, the paired samples *t* test was used for the variables with a normal distribution, and Wilcoxon matched pair test was used for variables that were not normally distributed. The independent samples *t* test was used for the variables with normal distribution in relation with the perforation size, and the Mann-Whitney U test was used for the variables that were not normally distributed. Normally distributed variables are presented as mean  $\pm$ SD, whereas variables that were not normally distributed are shown as median [interquartile range (IQR)]. The statistical significance was set at P=0.05.

#### Results

Nineteen patients diagnosed with NSP between September 2013 and April 2014 were included in the study. Our study included 12 (63%) males and 7 (37%) females. The mean age of the patients was  $42.26\pm13.20$  years (range, 23-65 years). Among the total of 19 patients, 15 (79%) patients had septoplasty, 2 (11%) had use of nasal sprays, 1 (5%) had nasal trauma, and 1 (5%) patient did not have any clear etiologic factors in the history. The most common complaint was dry nose and crusting (n=19, 100%), followed by nasal blockage (n=13, 68%), whistling during respiration (n=5, 26%), and daytime sleepiness (n=2, 10%). Mean duration of complaints was 4.3 years (range, 21.44–34.53), and the mean ESS score was 4. When

baseline PSG parameters were analyzed, it was seen that sleep efficiency and percentages of N1, N2, and N3 were within normal limits; however, there was a small decrease in percentage of REM sleep (15.4±5.2,% total sleep time) (Table 1). REM sleep accounts for about 20-25% of the sleep time in normal adults (9) and in our cases, percentage of REM sleep was less than in normal adults. Baseline AHI was <5 in 9 patients, 5-14 in 5 patients, and ≥15 in 5 patients. Median baseline AHI was 5.30 (14.40), and there was only a small deviation from the normal limits. On the other hand, median supine AHI was 10.00 (42.10), and there was a tendency for supine position-dependent sleep apnea in patients with NSP. Mean PO<sub>2</sub> saturation was 95.10±0.99%, and it was in the normal limits. Median AHI [5.30 (14.40) vs. 2.40 (14.50)], and median supine AHI [10.00 (42.10) vs. 6.60 (37.00)] decreased after correction of the perforation, but the differences were not statistically significant. Other studied baseline PSG parameters did not change significantly after treatment of NSP (Table 1).

The median NSP size was 66.00 (86.00) mm2 (range, 5–402 mm<sup>2</sup>). Ten (53%) patients had perforations  $\leq$ 66 mm<sup>2</sup>, and 9 (47%) patients had perforations >66 mm<sup>2</sup>. We divided the patients into 2 groups: ones with a perforation size  $\leq$ 66 mm<sup>2</sup>, and the ones with a perforation > 66 mm<sup>2</sup>. None of the studied parameters showed significant differences between the groups (p>0.05 for all). However, the median baseline supine AHI was 10.10 (34.15) in the group with a perforation >66 mm<sup>2</sup>, and it was 8.55 (51.43) in the group with a perforation size  $\leq$ 66 mm<sup>2</sup>. Although median supine AHI decreased to 1.60 (28.30) after closure of the perforation in the group with a perforation >66 mm<sup>2</sup>, the difference was not statistically significant (p=0.130). There were no significant changes in the 2 groups for other baseline and post- procedure parameters studied (p>0.05 for all) (Table 2).

The patients were divided into 2 groups according to their AHI: ones with AHI <5, and ones with AHI  $\geq$ 5. None of the parameters studied in the 2 groups showed significant changes after closure of the perforation (Table 3).

### Discussion

Sleep quality can be an important symptom of many sleep and medical disorders [9]. Factors relating to anxiety and stress are the most important concomitants of sleep complaints in the general population [10]. The nose provides the greatest resistance to airflow. Nasal airway resistance during sleep constitutes <sup>2</sup>/<sub>3</sub> of total airway resistance. The nasal airway is quite rigid during sleep, and, in contrast to the oropharyngeal segment, it does not collapse. Nasal septal deviation and a small nasal cavity volume may affect nasal airflow [1]. A number of studies investigated the presence of nasal septal deviation and

the impact of its surgical correction on sleep quality and respiratory parameters in patients with obstructive sleep apnea, showing that nasal surgery alone was not effective in restoring a normal sleep architecture or in treatment of obstructive sleep apnea [11–14]. However, Sufioğlu et al. reported that nasal surgery might lead to a reduction in continuous positive airway pressure (CPAP) levels [11]. On the other hand, sleep disorders in patients with nasal septal deviation were investigated in only a few studies [4]. Silvoniemi et al. showed oxygen desaturations during sleep in patients with nasal septal deviation [4].

Crusting due to NSP and impaired nasal airflow may lead to nasal blockage. Nasal blockage decreases sleep quality and exacerbates SDB [15]. Increased airway resistance, unstable mouth breathing, and nasal reflexes may cause SDB or exacerbate those that already exist [16].

The first study published on nasal airflow in NSP was conducted by Grützenmacher et al. [15], and reported that there was not a correlation between NSP size and abnormal aerodynamics. Air temperature in patients with NSP was significantly lower than the controls with normal noses, due to air mixing in NSP. The airflow passing through the perforation was turbulent and fast, and airflow speed was higher towards the rear end of the perforation [15]. In addition, blood flow was greater at the edges of the perforation. Steady turbulent flow occurring at the sagittal plane caused prolonged and extensive mucosal contact, leading to mucosal irritation and dryness. Mucosal dryness and damage at the increased wall tension caused bleeding and crusting [15]. NSP may cause nasal obstruction and lead to sleep problems, similar to nasal septal deviation. Our study is the first one in the literature that studied polysomnographic sleep parameters in patients with NSP.

The results of this study indicate that REM sleep duration was shorter in patients with NSP, and it did not increase after treatment [15.4 $\pm$ 5.2 vs. 16.4 $\pm$ 4.2]. REM sleep accounts for about 20–25% of the sleep time in normal adults (9) and in our cases, percentage of REM sleep was less than in normal adults. In our study, in 5 patients AHI values were  $\geq$ 15. Therefore, in these patients, obstructive sleep apnea may cause lower REM percentages. In addition, both median AHI [5.30 (14.40) vs. 2.40 (14.50)], and median supine AHI [10.00 (42.10) vs. 6.60 (37.00)] decreased after correction of the perforation. There was a great reduction in median supine AHI in patients with a perforation size >66 mm<sup>2</sup> [10.10 (34.15) vs. 1.60 (28.30)].

Our study has some limitations. First, our sample size was small. A larger sample size would increase the statistical power of the study. Second, we analyzed only objective parameters, and not the subjective ones after closure of the perforation. Third, we treated NSP with a prosthesis (silicone nasal septal button), and not with surgery.

	Parameter	Perforation area ≤66 mm² Mean ±SD	Perforation area >66 mm² Mean ±SD	Р*
Sleep efficiency (% TIB)	Baseline	87.66±8.06	86.04±7.61	0.660
	PP	86.09±11.71	84.14±11.29	0.718
	P**	0.581	0.491	0.492
	Baseline	5.47±4.75	4.27±2.03	
	PP	4.04±1.96	4.59±2.17	0.570
N1 (% TST)	P**	0.422	0.745	
	Baseline	52.22 <u>+</u> 7.94	59.24±7.24	0.061
	PP	51.57±12.37	55.52±9.40	0.448
N2 (% TST)	P**	0.885	0.345	
	Baseline	28.47±8.41	20.71±7.77	0.053
	PP	29.13±12.49	22.11±10.46	0.205
N3 (% TST)	P**	0.876	0.641	
	Baseline	15.0 <u>+</u> 4.4	15.8±6.3	0.759
	PP	15.2±3.3	17.7±4.9	0.214
REM (% TST)	P**	0.898	0.326	
	Baseline	4.75 (23.00)	2.90 (12.15)	0.566
	PP	9.00 (34.95)	4.90 (7.55)	0.270
Median AHI-REM (IQR)	P**	0.646	0.678	
	Baseline	6.70 (16.50)	3.80 (14.35)	0.307
	PP	2.40 (15.27)	1.30 (18.85)	0.567
Median AHI-NREM (IQR)	P**	0.286	0.594	
	Baseline	6.20 (18.08)	5.30(14.90)	0.462
	PP	4.35 (18.30)	1.60 (16.10)	0.205
Median AHI TST (IQR)	P**	0.646	0.374	
	Baseline	8.55 (51.43)	10.10 (34.15)	0.652
	PP	7.35 (66.45)	1.60 (28.30)	0.130
Median supine AHI (IQR)	P**	0.959	0.674	
	Baseline	94.90±1.10	95.33±0.87	0.357
	РР	95.30±1.25	95.33±1.12	0.952
Mean PO2 (%)	P**	0.223	1.000	
	Baseline	86.56±5.41	87.78±5.85	0.652
Minimum PO2 (%)	PP	87.20±6.63	87.56±6.62	0.908
		0.838	0.899	

**Table 2.** Analysis of polysomnographic parameters when the patients are divided into two groups with relation to the size of the nasalseptal perforation ( $\leq 66 \text{ mm}^2$  and  $> 66 \text{ mm}^2$ ).

PP – post-procedure; TIB – total time in bed; TST – total sleep time; AHI – apnea hypopnea index; REM – rapid eye movement sleep; NREM – non-rapid eye movement sleep; IQR – interquartile range; PO2 – partial oxygen pressure. P\* – values compared with regard to area of septal perforation; P\*\* – comparison of baseline and post- procedure sleep parameters.

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	Parameter	AHI <5 Mean ±SD	AHI ≥5 Mean ±SD	Р*
Sleep efficiency (% TIB)	Baseline	85.29±8.03	88.34±7.46	0.402
	PP	84.90±11.28	85.41±11.79	0.924
	P**	0.861	0.355	
	Baseline	3.58±1.43	6.09±4.67	0.141
N1 (% TST)	PP	4.42±2.13	4.19±2.02	0.811
	P**	0.331	0.290	
	Baseline	58.66±9.10	52.75±6.61	0.121
N2 (% TST)	PP	50.78±10.49	55.84±11.34	0.328
	P**	0.069	0.414	
	Baseline	22.94±9.27	26.46±8.56	0.402
N3 (% TST)	PP	28.79±12.66	23.12±10.95	0.310
	P**	0.134	0.304	
	Baseline	14.4±4.3	16.2±6.1	0.485
REM (% TST)	PP	15.9±4.3	16.8±4.4	0.653
	P**	0.501	0.65	
	Baseline	1.40 (3.30)	16.20 (25.13)	0.004
Median AHI-REM(IQR)	PP	3.20 (7.90)	8.75 (37.05)	0.028
	P**	0.066	0.646	
	Baseline	0.90 (2.15)	11.60 (23.28)	0.001
Median AHI-NREM(IQR)	РР	0.80(1.35)	10.70 (20.00)	0.001
	P**	0.674	0.878	
	Baseline	0.80 (0.90)	34.15 (45.05)	0.001
Median supine AHITST(IQR)	РР	2.70 (3.85)	28.75 (63.00)	0.010
	P**	0.401	0.959	

**Table 3.** Analysis of polysomnographic parameters when the patients are divided into two groups with relation to their apnea hypopnea index (< 5 and ≥5).

 $PP - post-procedure; TIB - total time in bed; TST - total sleep time; AHI - apnea hypopnea index; REM - rapid eye movement sleep; NREM - non-rapid eye movement sleep; IQR - interquartile range; PO2 - partial oxygen pressure. P* - values compared with regard to AHI <5 and <math>\geq$ 5; P \*\* - Comparison of baseline and post- procedure sleep parameters.

# Conclusions

Our preliminary results indicated that NSP did not cause any deterioration in objective sleep parameters as determined by PSG, other than a decrease in REM sleep duration and an increase in supine AHI. Correction of NSP did not affect REM duration, but supine AHI decreased after treatment. Further studies performed on a larger patient cohort may shed light on the effects of NSP and its treatment on sleep.

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*Conflict of interest:* All authors certify that they have no affiliations with or involvement in any organization or entity with any financial interest (such as honoraria; educational grants; participation in speakers' bureaus; membership, employment, consultancies, stock ownership, or other equity interest; and

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*Ethics approval:* All procedures performed in studies involving human participants were in accordance with the ethics standards of

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the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethics standards.

*Informed consent:* Informed consent was obtained from all individual participants included in the study.

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