REVIEW ARTICLE

Impact of Impaired Nasal Breathing on Sleep-Disordered Breathing

Thomas Verse, M.D.[1] and Wolfgang Pirsig, M.D.[2]

ABSTRACT

Objective: The role of the nose and its importance in the development and severity of sleep-disordered breathing (SDB) is still a matter of discussion. In the first part of this study, often-controversial data and theories about the nose are reviewed concerning its influence on the pathophysiology of SDB and to interpret certain clinical findings connected with impaired nasal breathing. In the second part, the effectiveness of some nonsurgical and surgical therapies is evaluated. Method: A worldwide literature research (Medline) was the basis for this review. Results: The study of the literature on nasal resistance and clinical findings about the effects of incomplete or complete nasal blockage, particularly in comparison of healthy persons and persons with SDB, allows the assumption of the existence of two different groups of responders: a larger group where the importance of the nose for SDB is negligible and a smaller group where the influence of the nose on SDB is crucial. The same seems to hold true for the responses to nonsurgical and surgical treatments with only a few surgical results available in the literature. While the success rate of nasal surgery for patients with obstructive sleep apnea, for instance, seems to be less than 20%, the normalization of nasal resistance often leads to a positive impact on the well-being and the sleep quality of these patients. However, because criteria to identify responders are lacking, the prediction of success of any treatment for the individual with SDB is not possible.

KEYWORDS: Rhinosurgery, treatment, nose, pathophysiology, sleep apnea, snoring

The Nose and Sleep-Disordered Breathing; Editors in Chief, Nikolaus C. Netzer, M.D., Kingman P. Strohl, M.D.; Guest Editor, Wolfgang Pirsig, M.D. *Sleep and Breathing*, volume 7, number 2, 2003. Address for correspondence and reprint requests: Thomas Verse, M.D., University ENT Clinic Mannheim, D-68135 Mannheim, Germany. E-mail: thomas.verse@hno.ma.uni-heidelberg.de. [1]University ENT Clinic Mannheim, Mannheim, Germany; [2]ENT Department, University Hospital Ulm, Ulm, Germany. 1520-9512,p;2003,07,02,063,076, ftx,en;sbr00231x.

Already in the ancient world there was evidence that impaired nasal breathing may lead to sleep-disordered breathing (SDB). Hippocrates, in *de morbis*, liber II, sect V, described snoring in addition to enlargement of the lateral nasal walls and croaky voice as symptoms of polyposis nasi. In 1581, Levinus[1] reported that mouth breathing in the supine position causes restless sleep. In 1892, Cline[2] published a first case of surgical relief of excessive daytime sleepiness following nasal surgery. In 1898, Wells[3] reported an increase of daytime vigilance in 8 of 40 patients after nasal surgery.

The purpose of this article is to summarize data and theories about the role of the nose in the pathophysiology of SDB, focusing on the nasal resistance and the interpretation of some clinical findings connected with impaired nasal breathing. A second section will discuss the effectiveness of some nonsurgical and surgical nasal treatments.

ROLE OF THE NOSE IN THE PATHOPHYSIOLOGY OF SDB

Nasal Resistance

The significance of impaired nasal breathing for the pathogenesis of SDB, in particular primary snoring and obstructive sleep apnea (OSA), has only been elucidated to a minor extent, although several studies indicate that there is a certain connection. A particularly attractive area of exploration concerns elevated nasal resistance, since it leads to an increase in inspiratory negative pressure in the unstable pharyngeal segments and in the thorax. If the inspiratory negative pressure falls below the critical collapse pressure in the pharynx, the pharynx collapses and an obstructive apnea occurs.

Nasal resistance is influenced by numerous factors such as environmental climate, physical activity, or body position. Rundcrantz[4] and Hasegawa and Saito[5] showed in healthy subjects that the nasal resistance is elevated when the person is in the supine position compared to the upright position. In patients with SDB, this effect could not be compensated even by administration of decongesting nasal drops.[6] De Vito and colleagues[7] analyzed the nasal resistance of 36 patients with OSA and retropalatal obstruction by performing anterior active rhinomanometry in the seated and supine positions. They found nasal obstructions in 44% of the patients but no significant correlation between the severity of OSA and the presence of nasal obstruction. In 9 patients the nasal resistance became pathologic in the supine position and in 7 patients the pathologic nasal resistance of the seated person increased in the supine position. Nasal resistance is lower in healthy people than in patients with OSA[8]; it is almost identical during sleep and in the awake state.[9,10] Nasal obstruction during pregnancy is very common during the second and third trimesters. It is thought to be caused by increased estrogen levels causing hyperemia and edema of the nasal mucosa with increased secretions.[11] Severe snoring or irregular breathing, however, are rarely observed in pregnancy.[12]

Healthy adults prefer nasal breathing both in the awake state and during sleep.[13] The nose, with its cartilaginous and bony framework and its mucosa, is designed to filter and condition the inspired air.[14] In this way the lower respiratory airways are protected against damaging exogenous influences. In the awake state, however, the nasal respiratory airway has a higher respiratory airway resistance than oral respiration. The nasal airway resistance accounts for about 50% of the total respiratory resistance to airflow in humans.[13,15] During wakefulness, respiratory effort is more than double with nasal breathing compared with breathing through the mouth.[16] During sleep, the pharyngeal resistance increases while the nasal resistance remains constant. Therefore, the nasal resistance during sleep is of less importance than during wakefulness.

The nasal mucosa is a dynamic organ controlled by the autonomic nervous system. The peri-

odic congestion and decongestion of the nose was termed the "nasal cycle" by Heetderks.[17] This nasal cycle exists in about 80% of the adult population. In patients with unilaterally fixed impairment of nasal breathing, the nasal cycle may contribute to a substantial increase of the total airway resistance.[18]

Yet the nasal mucosa also reacts independently of the nasal cycle. If the nasal mucosa is stimulated mechanically, respiration, heart rate, and the resistance in peripheral vessels may be influenced via trigeminal reflex pathways.[19]

In healthy subjects, lateral recumbency decreases the patency of the ipsilateral nasal passage and increases that of the contralateral nasal passage. This is not a hydrostatic effect but a reflex response elicited by asymmetric pressure on the body.[9,20] This reflex interrupts the nasal cycle.[21] Seen bilaterally, however, lateral recumbency does not lead to a significant alteration of the entire nasal cross-section when compared with the supine position.

Clinical Findings

EFFECT OF COMPLETE NASAL BLOCKAGE

Several working groups have investigated the effect of complete nasal blockage on breathing in the awake state and during sleep. In the awake state, a complete nasal obstruction by nasal packing led to significantly increased hemoglobin values, CO_2 partial pressure, and HCO_3. Moreover, a significant decrease in the O_2 partial pressure was observed, but there was no difference in the blood pH value.[22–25]

Several authors unanimously reported a more disturbed sleep and an increased number of arousals in patients with bilateral nasal packing.[26–29] In 12 patients with nasal packing after nasal septoplasty, Johannessen and associates[30] found a significantly longer period of time with peripheral oxygen saturation below 90% when compared with the values before and after nasal packing. Tanaka and Honda[31] recorded a lower pCO_2 during sleep in 6 healthy subjects with obstructed nose. The authors assume that a reduced CO_2 stimulus plays a role in the pathogenesis of sleep-related breathing disturbances.

Lavie and Rubin[32] supposed that a proneness to sleep apnea was inheritable. They investigated the effect of complete artificial nasal obstruction in 6 nonapneic patients, whose fathers suffered from severe OSA, and in 4 age-matched patients without a family history of snoring. The nasal blockage significantly increased the apnea index (AI) in the sons of the sleep apnea patients from 0.8 to 10.5 in comparison with the increase in the controls (AI from 1.12 to 2.7). The authors concluded that in at least some genetically-prone individuals, sleep apnea syndrome may be caused by increased upper airway resistance.

Also, the number of apneic and hypopneic episodes during sleep is significantly increased with obstructed nose.[26–29,33–36] There were more central, obstructive, and mixed apneic episodes. The results, however, had a substantial individual variation. A dramatic increase of respiratory episodes was only seen in 16 out of 45 cases (35.6%). From a pathophysiological point of view, there are apparently two groups of patients. For approximately one third of the examined persons the nasal airway seems to be more important for maintaining breathing during sleep. However, the small number of examined persons and the fact that mostly healthy subjects were included do not allow a more precise description of those patients who develop OSA caused by nasal obstruction.

EFFECT OF INCOMPLETE NASAL BLOCKAGE

As early as 1969 Masing and Horbaschk[37] observed uneasy sleep in 161 children after unilateral nasal blockage.

In 10 healthy subjects Lavie et al[28] observed an increased AI after unilateral nasal blockage from on average 1.4 to on average 3.1.

In patients with seasonal allergic rhinitis more apneas were described during the pollen season compared to reference months.[38] However, the AI

increase of 0.7 in the allergen-free period to 1.7 during pollen season is negligible. One case of mild OSA after guar gum dust exposure was described.[39] Patients with impaired nasal breathing caused by allergic rhinitis showed significantly more arousals in non-rapid eye movement sleep 1 + 2 when compared with people without nasal problems.[40] The authors conclude that an elevated nasal resistance leads to an increased number of arousals in light sleep stages.

STUDIES COMPARING PATIENTS WITH AND WITHOUT OSA

Rubinstein[41] compared 6 healthy volunteers and 8 OSA patients with respect to nasal inflammation. They found a significant increase of the percentage of neutrophiles, bradykinin, and VIP (vasoactive intestinal peptide) in the serums of the OSA patients.

Houser and associates[42] retrospectively compared the degree of nasal obstruction seen in allergic patients with (N = 10) and without (N = 40) mild OSA using acoustic rhinometry (AR) both in the predecongested and the postdecongested state. The mean congestion factors at the cross-sectional area at a distance of 2 cm into the nasal cavity on the AR graph were found to be significantly higher in the OSA group than in the non-OSA group ($p = 0.03$). Furthermore, the non-OSA patients noted a significant subjective improvement in nasal congestion after topical nasal decongestion, whereas the OSA patients did not ($p < 0.0001$ and $p = 0.064$, respectively). The authors conclude that the nasal obstruction associated with allergic rhinitis is associated with the presence of mild OSA.

Duchna and coworkers[43] investigated each of 28 patients with SDB, matched according to age, weight, and gender. In one group nasal breathing was normal (flow > 700 ccm/s at 150 Pa), in the other group nasal breathing was impaired (flow < 500 ccm/s at 150 Pa). There were significant differences regarding the subjective nightly dyspneic episodes (7 vs. 17) and the AI (9.6 vs. 20.4). No differences were found regarding daytime oxygen saturation, heart rate, hypopnea index, and pulmonary function test.

Anch et al[44] investigated the supraglottic respiratory resistance in healthy subjects and patients with OSA in sitting position and recumbency, with and without decongesting nasal drops. Sleep apneics exhibited greater respiratory resistance than healthy subjects, even after treatment with decongesting nasal drops. From their measurement results, the authors conclude that there is a nonmucosal difference between the groups that is responsible for the elevated impedance of the upper airway in patients with OSA.

Also, Blakley and Mahowald[45] described a significantly increased nasal resistance in 53 patients with OSA compared to 37 control persons. Yet an apnea-hypopnea index (AHI) dependence on nasal resistance was not found. The authors therefore believe that nasal resistance is not a main factor in the pathogenesis of OSA.

In a prospective study in 541 nonselected snorers, Lofaso and associates[8] performed posterior rhinomanometry in the awake state. Results showed that nasal obstruction is an independent risk factor for OSA and contributes 2.3% of the AHI variance, whereas other factors contribute as follows: overweight 4.6%; male gender 3%; age 1.3%; and cephalometric parameters MP-H 6%, SNB 0.9%.

Young and associates[46] investigated the role of chronic and acute nasal congestion in a population-based study. Data on nasal congestion and sleep problems were obtained by questionnaire (N = 4927) and by objective in-laboratory measurements (polysomnography, single nostril rhinometry in N = 911). Participants who often or almost always experienced night-time symptoms of rhinitis (5 or more nights a month) were significantly ($p < 0.0001$) more likely to report habitual snoring (3 to 7 nights a week), chronic excessive daytime sleepiness, or chronic nonrestorative sleep than were those who rarely or never had symptoms. Habitual snorers had significantly ($p < 0.02$) lower airflow than nonsnorers, although a linear relation between decreased airflow

and SDB severity did not exist. Participants who reported nasal congestion due to allergy were 1.8 times more likely to have moderate to severe SDB than were those without nasal congestion due to allergy. This analysis of a population-based sample showed that middle-aged men and women with nasal obstruction, particularly those with chronic night-time symptoms of rhinitis, are significantly more likely to be habitual snorers.

Another 46 patients (mean age 40 years, mean body mass index 26 kg m^{-2}) with severe nasal obstruction due to septal deviation were investigated by polygraphy and rhinomanometry.[47] Of these patients, 43% had breathing disorders at night and 67% also had heavily disturbing snoring. Sleep recordings revealed 12 cases with partial upper airway obstruction and one case with severe airway obstruction. The incidence of snoring and partial upper airway obstruction significantly exceeds the incidence of these disturbances in the population.

After all, there is some evidence in recent literature that nasal obstruction may act as a cofactor in the pathogenesis of at least mild OSA. This finding, however, is not undisputed in the literature. Several authors found in a total of 131 patients and 123 control persons no relationship at all between nasal obstruction and AHI.[48–50] Miljeteig and coworkers[51] allocated 683 snorers with or without suspected OSA into three groups according to the snorers' nasal resistance: normal or unilaterally or bilaterally increased. Also in this study no statistical differences were found with respect to the AI, the snoring sounds, and the number of primary snorers and patients with OSA.

The results cited are all derived from airway resistance measurements performed in the awake state. To sum it up, it may be stated only that an increased daytime nasal resistance is apparently not the main risk factor for OSA.

Miyazaki and colleagues[52] came to the same conclusion. These authors investigated 54 patients with OSA by polysomnography and multipressure transducer measurements during sleep. Only 7 of 54 (13%) showed decreased epipharyngeal pressure below −3 cm H_2O, indicating pathologically increased nasal resistance. The authors concluded that the nose is never the only factor that determines an OSA syndrome.

How Can Nasal Pathologies Lead to Respiratory Events During Sleep?

There are three possible ways that nasal pathology may lead to the occurrence or deterioration of SDB.

SWITCHING TO UNSTABLE ORAL BREATHING

The closed jaw and dental occlusion stabilize the upper respiratory airway.[15,53] The remaining nasal airway is seen as a physiological airway and is important to maintain the respiratory rhythm.[54] In this way, the nasal airway remains functional even in children with subtotal nasal obstruction.[55] Only with total nasal obstruction does switching to oral breathing occur. During sleep, however, oral breathing is unstable and (as opposed to the situation in the awake state) is associated with increased airway resistance.[27] Due to the altered mechanical situation oral breathing during sleep may well be associated with obstructive apneas.[15,54,56] However, such impaired nasal breathing is extremely rare compared to the incidence of OSA and is therefore not considered to be a main cause for OSA.

But central respiratory events were also increasingly reported with oral breathing during sleep. Tanaka and coworkers[31] suppose that after switching to oral breathing during sleep there is an elevated CO_2 expiration caused by an initially increased ventilatory stimulus. The authors explain the occurrence of central apneas with a secondarily reduced CO_2 ventilatory stimulus. The increase of central apneas suggests that the nose plays a role in regulating respiration rather than in maintaining the patency of the airway.

ELIMINATION OF NASAL REFLEXES

Already in 1870 Kratschmer[57] elicited in animal experiments a reflex cardiac and respiratory arrest by mechanical stimulation of the nasal mucosa. After cutting both trigeminal nerves it was possible to inhibit any other impact of natural or other stimulation of the nasal mucosa on respiration and circulation. A detailed description of nasal reflexes and their inhibition by local anesthetics was published by James and de Burgh Daly.[19]

The application of local anesthetics in the nose, however, leads per se to increased occurrence of both central and obstructive sleep apneas,[58–61] but the elevated obstructive apneas found were statistically not significant for the corresponding investigated groups. If the individual patients are assessed, the series of White and colleagues[58] makes clear that individuals may well react to local anesthetics with a distinctly increased number of apneas. In this series the AHI increased to pathologic levels in 3 of 10 patients, whereas it remained unchanged in 7 patients.

There is a sequential activation pattern of the muscles of the upper airway that depends on inspiration and expiration.[62] This pattern apparently plays a greater role during sleep than in the awake state. In this connection, nasal breathing seems to have a certain control function in the respiratory regulation. On the basis of currently available data, in a smaller number of patients the nasal reflex pathways are likely to play a central role in the pathogenesis of OSA.

INCREASED INSPIRATORY SUCTION

It is furthermore possible that a pathologically elevated nasal resistance must be balanced by increased inspiratory effort. A consequence thereof is an increased intrathoracic negative pressure that is transmitted to the collapsing segments of the upper airway between choanae and larynx and that may lead to inspiratory collapse of the upper airway.

A severe bilateral nasal obstruction only rarely occurs in adults in association with polyposis nasi or tumors. In this connection the occurrence of OSA was described.[63] In adults, partial nasal obstruction generally occurs but has only led to mild OSA.[28,39,51,64] It has not been possible to provoke severe OSA. For patients with seasonal allergic rhinitis more apneic episodes were reported during pollen season than during reference months[38,39]; the AI increase, however, was negligible. Nonetheless, a small number of patients with OSA and incomplete nasal obstruction can be healed by nasal treatment only. Average success rates are reported to be 9% after conservative treatment and 18% after operative treatment. However, only a few and often selected patients were included in these studies. More detailed information is given in the following paragraphs.

EFFECT OF NASAL TREATMENT

Nasal Decongestants

Kerr et al[65] treated 10 patients with decongesting nasal drops and placebo. While the nasal resistance improved highly significantly under xylometazoline treatment compared to placebo, the AHI remained unchanged. However, a significant reduction in sleep fragmentation was found. The authors believe that possibly a minor reduction of respiratory effort was achieved, which in turn caused a reduced number of arousals but did not lead to a reduced number of measurable respiratory events.

Nasal Corticosteroids

Brouillette and associates[66] investigated the effect of nasal fluticasone versus placebo in 25 children with OSA using a randomized, blinded, placebo-controlled study design. OSA was defined as an AHI greater than 1. Fluticasone was used by 13 children for 6 weeks, while 12 children took placebo. In the fluticasone group the AHI decreased from 10.7 to 5.8, while in the placebo group the AHI rose

from 10.1 to 13.1 (p = 0.04). The AHI decreased in 12 of 13 children in the verum group and in 6 of 12 children in the placebo group (p = 0.03). The respiratory movement/arousals index decreased as well in the group of children who used fluticasone (p = 0.5). The size of the adenoids remained constant in both groups.

Nasally applied topical corticosteroids were able to improve the subjective parameters of sleep quality, daytime sleepiness, and nasal breathing in 20 adults with allergic rhinitis.[67] Objective sleep studies were not included in this trial.

Nasal Dilators

Stenosis of the nasal valve and nasal vestibule may be dilated temporarily by using endonasal dilators (e.g., Nozovent) or extranasally by plasters in the area of the nasal valve (e.g., Breathe Right). Study results concerning the impact of nasal dilators on the severity of SDB are given in Table 1.[67a–67d] Long-term results on these dilators are not available. Nasal dilators are recommended to predict the outcome of surgeries on the nasal valve and vestibule.

Nasopharyngeal Tubes

In 1981, Afzelius and associates[68] presented two patients with severe OSA cured by self-intubation with a nasopharyngeal tube during sleep. Within the 6-month follow-up, no complications were found.

Nahmias and Karetzky[69] treated 44 patients with OSA with nasopharyngeal tubes. At 4 months' follow-up 44% of the patients still tolerated their tubes. The AI was reduced by 62.3%. Responder rates were quoted at 36.4%, which is higher than rhinosurgical success rates. The reason for this high responder rate might be the splinting of the nasopharynx, which is not affected by rhinosurgery.

Masters and colleagues[70] described the successful use of a modified nasopharyngeal tube that does not add airway space and resistance to relieve upper airway obstruction in nine infants with Pierre-Robin sequence, isolated micrognathia, Down syndrome, and idiopathic generalized hypotension. The well-tolerated tube allows simultaneous use of oxygen prongs. The tube was required for a median of 6 months in children with Pierre-Robin sequence (N = 6) and up to 15 months for the other infants. Apart from three infants who had regurgitation of food into the nasopharyngeal tubes in the initial period, no other complication occurred.

Surgical Treatment

To date there are no long-term results concerning the effectiveness of nasal surgery in the treatment of OSA. Present data are based on mostly noncontrolled and nonrandomized studies and do fulfill only the grade 1 and grade 2 criteria of evidence-based medicine. Some working groups provide subjective data regarding the impact of nasal surgery on simple snoring.

Table 1 Effect of Internal and External Nasal Dilators on the Severity of Obstructive Sleep Apnea

Author	Dilator	N	AHI Without	AHI With	p-value
Höijer et al[67a]	Internal	10	18 (AI)	6.4 (AI)	0.008
Metes et al[67b]	Internal	10	46	44	N.S.
Kerr et al[65]	Internal + ND	10	64.9	63.2	N.S.
Hoffstein et al[102]	Internal	15	35.4	33.9	N.S.
Schönhofer et al[67c]	External	30	38.1	40	N.S.
Gosepath et al[67d]	External	26	31.7	26.3	0.031

AHI, apnea hypopnea index; AI, apnea index; ND, nasal decongestants; N.S., not statistically significant.

When summarizing these very inhomogeneous data that usually lack polygraphic or polysomnographic investigation, it is worth noting that a reduction or disappearance of snoring is reported in a few studies. Woodhead and Allen[71] reported successful reduction of snoring in 20 patients (69%) and failure in 9 subjects (31%) 6 weeks after nasal surgery. Fairbanks[72] described 1 child who gained relief from snoring after only sinus surgery. Seven adults gained relief from snoring after septoplasty and turbinate surgery. Low[73] found snoring relief in 50% (N = 15) of his patients 4 to 12 months after septal surgery. On the other hand, Illum[74] reported that of the 50 patients who underwent septoplasty and conchal surgery, 58% were snoring preoperatively and 41.5% complained of snoring 5 years postoperatively. From these few examples (often cited) and similar publications it is impossible to estimate a percentage of success rate of nasal surgery in primary snorers.

Furthermore, there are a few case reports of cures of OSA after nasal surgery only.[75–77] However, already in 1977 Simmons and coworkers[78] reported cases with no significant AI reduction despite considerable subjective improvement of nasal breathing and sleep quality.

Up to the year 2000, only 9 studies on only nasal surgery for OSA were found which provide data on pre- and postoperative AI or AHI (Table 2). All together 102 patients from eight working groups[79–87] were included in the studies. The follow-up periods were predominantly short and lasted from 1 month[79] to 44 months.[80] A statistically significant improvement of the severity of OSA after nasal surgery alone was found in only one study.[79] This study includes 9 sleep apneics. Their AI decreased from 37.8 to 26.7. In four other studies with a total of 30 patients an increase in the severity of OSA was noticed postoperatively (Table 2) which was not statistically significant in all studies. Verse et al[86] recorded a noticeable worsening of OSA in two patients with polyposis nasi after paranasal sinus surgery. Despite the reconstitution of nasal breathing, the AHI rose from 14.0 before to 57.7 after surgery. Both patients developed excessive daytime sleepiness and required nasal continuous positive airway pressure (CPAP) therapy. Two similar cases after septorhinoplasty were reported by Dagan.[88]

Lavie and coworkers[89] reported on 14 patients with OSA who all underwent only septoplasty. OSA severity did not change after surgery, but 12 of their 14 subjects showed improved sleep quality in the polysomnography and reported less daytime fatigue.

Verse and associates[90] prospectively investigated the effect of nasal surgery in 26 patients with

Table 2 Effect of Nasal Surgery on the Severity of Obstructive Sleep Apnea

Author	N	Follow-up	AI pre	AI post	AHI pre	AHI post	p-value
Rubin et al[79]	9	1–6	37.8	26.7			< 0.05
Dayal, Phillipson[80]	6	4–44			46.8	28.2	N.S.
Caldarelli et al[81]	23	no data	44.2	41.5			N.S.
Aubert-Tulkens et al.[82]	2	2–3	47.5	48.5			—
Sériès et al[83]	20	2–3			39.8	36.8	N.S.
Sériès et al[84]	14	2–3	17.8	16			N.S.
Utley et al[85]	4	no data			11.9	27	—
Verse et al[86]	2	3–4	9.2	47.3	14	57.7	—
Friedman et al[87]	22	> 1.5			31.6	39.5	N.S.
Verse et al[90]	26	3–50			31.6	28.9	N.S.

AI, apnea index; AHI, apnea hypopnea index; N.S. not statistically significant.

SDB (7 patients with simple snoring, AHI < 10, and 19 patients with OSA, AHI > 10). Follow-up investigations were done 12.7 months postoperatively including a fully attended polysomnography in the sleep lab. The patients (1 female, 25 males) had a mean age of 52.5 and a mean body mass index of 29.16 kg m^{-2} that remained unchanged at the time of re-examination (29.2 kg m^{-2}). Nasal resistance (anterior rhinomanometry at 150 Pa) was significantly reduced after surgery ($p = 0.0089$). Daytime sleepiness was improved as well. The Epworth Sleepiness Scale (ESS) was scored at 11.9 before surgery and fell to 7.7 after nasal surgery ($p = 0.0004$). The arousal index decreased statistically significantly from 28.9 to 21.7 postoperatively ($p = 0.0336$). However, neither the AHI (31.6 vs. 28.9) nor the oxygen desaturation index showed statistically significant improvement after surgery. Despite a reduced nasal resistance the severity of OSA increased in four patients. Using the criteria set forth by Sher and associates[91] (reduction of AHI > 50% and to values < 20), only 3 of 19 patients with OSA (15.8%) were classified as cured after nasal surgery.

Other studies report cure rates of nasal surgery for OSA between 0%[82,86] and 33%.[80] In the literature, raw data for 76 patients with OSA are provided. Using Sher's criteria the cure rate of these patients is 17.5%. Unfortunately, we do not yet know any factors that can predict success. Low[73] did not find any influence of preoperative severity of nasal obstruction, preoperative intensity of snoring, preoperative collapsibility of the soft palate, or degree of reduction of nasal obstruction on the postoperative result. In 7 of 14 patients with mild OSA, Sériès et al[84] found that a radiocephalography revealed narrowing of the airway space behind the tongue and an increased distance from the mandible to the hyoid bone. Three months after nasal surgery a comparison of those with and without anatomical abnormalities showed that those with normal anatomy experienced a significant improvement in sleep and respiratory parameters (AHI, arousal index). In patients with pathological radiocephalographic findings, however, both indices remained unchanged after surgery. The authors conclude that the presence of craniomandibular abnormalities makes it unlikely that nasal surgery will improve mild OSA.

Busaba[92] retrospectively compared the safety of performing same-stage nasal and palatopharyngeal surgery (group 1; N = 63) with palatopharyngeal surgery at a stage separate from the nasal surgery (group 2; N = 28) for the treatment of moderate to severe OSA (mean AHI 36.5 versus 33.5). The two groups were fairly matched according to age, gender, comorbidities, and polysomnographic data. Uvulopalatopharyngoplasty (UPPP) with tonsillectomy was performed in 51% of group 1 and in 68% of group 2. The remaining patients underwent UPPP without tonsillectomy. After nasal surgery the noses were packed bilaterally. The packs were removed the next day before patient discharge. During these 23 guarded hours the patients were monitored with continuous pulse oximetry. Postoperative complications occurred in three patients (4.8%) of group 1 (pneumonia, tonsil bleed, septal hematoma) and in one patient (3.6%) in group 2 (tonsil bleed). The authors conclude that same-stage nasal and palatopharyngeal surgery for OSA is safe.

Antila et al[93] measured the volume of the nasal cavities and nasopharynx of 29 patients using acoustic rhinometry before and after UPPP with the laser technique. The static-change-sensitive bed was used for cardiorespiratory monitoring during sleep, but data were not reported. Subjectively, snoring had decreased in 97% of the patients and daytime somnolence in all cases. There was a tendency to higher postoperative values of midnasal volume in baseline and decongestion recordings, indicating that the conchal area is more patent after the velar operation. Using rhinomanometry, Welinder and associates[94] and Kawano and colleagues[95] found a significantly lower nasal resistance 18 and 3 to 6 months following UPPP, respectively.

Impact of Nasal Surgery on Nasal CPAP Treatment

There are only a few papers reporting on the effect of nasal surgery on the requested CPAP. Apart from some case reports,[96–98] Sériès et al[83] reported about seven patients who needed nasal operations before they were able to tolerate a nasal CPAP treatment.

In a first prospective trial, Friedman and associates[87] performed a septoplasty with partly bilateral reduction of the inferior nasal turbinates in 50 patients with different severities of OSA. All patients were re-examined 6 months after their surgical procedures. The mean body mass index remained constant (35.0 kg m^{-2} versus 35.7 kg m^{-2}). Forty-nine patients reported improved nasal breathing, 14 subjects (28%) snored less, and 3 patients (6%) no longer snored after surgery. Daytime activity was increased in 39 apneics (78%). A sample of 22 patients underwent a second polysomnography. Their AHI increased statistically not significantly from 31.6 to 39.5 postoperatively. However, the CPAP required to cure OSA fell from 9.3 mbar to 6.7 mbar. The reduction of CPAP was statistically significant in patients with severe OSA (N = 13). The authors conclude that nasal surgery should be included in a comprehensive treatment approach to OSA.

In the elderly patient with moderate to severe OSA (AHI > 30), the AHI may increase if nasal packing is used for epistaxis or after surgery. Even vital complications have been reported.[22,34] For this reason, Dorn and colleagues[99] investigated the use of oral CPAP application in a pilot study including five patients with severe OSA (mean AHI = 54.5) whose noses were packed after nasal surgery. Oral CPAP ventilation proved to be effective and safe. Apart from that the authors described a reduction of the requested nasal CPAP of 3.2 mbar as early as 6 weeks after surgery.

Biermann[100] retrospectively compared each of 35 severe sleep apneics with and without septoplasty and turbinoplasty. All patients were on nasal CPAP ventilation and a polysomnographic re-examination was performed at least once a year. In the group of patients who underwent a nasal operation the requested CPAP was 1.5 mbar lower ($p < 0.01$) and the mean duration of daily use was 0.8 hours longer ($p < 0.01$). Apart from that the author described a negative correlation between CPAP and the duration of its daily use.

All cited reports agree that the requested nasal CPAP can be statistically significantly reduced by nasal surgery. In some cases nasal surgery improves the patients' compliance regarding a necessary CPAP treatment.

Nasal surgery may reduce the sound intensity of snoring by 5 to 10 dB.[101] Nasal surgery improves nasal ventilation, sleep quality, and daytime vigilance and nasal surgery does reduce the required CPAP.

CONCLUSION

Complete or incomplete obstruction of the nasal airway during sleep generally leads to impaired sleep quality caused by increased sleep fragmentation with subsequent daytime fatigue. An increase of central, mixed, and obstructive respiratory events is also described.

In cases of complete obstruction the increased number of respiratory events in healthy subjects is to be assessed as being of minor importance. Statistical significance is not always achieved. However, if the individual case is assessed, in approximately one third of the subjects such a distinct increase of breathing events was provoked that a clinically measurable sleep-related breathing disturbance was found.

Incomplete nasal obstruction only leads to marginal increase of sleep-related breathing events that, according to current knowledge, have never caused severe OSA. Simultaneous occurrence of

central apneas suggests that the nose is important rather for respiratory regulation than for maintaining the patency of the upper airways.

Conversely, nonsurgical or operative reduction of nasal resistance significantly improves the well-being, daytime fatigue, and sleep quality of the persons concerned; moreover, the number of arousals can be reduced. The number of apneas and hypopneas hardly varies within the group of patients. Success rates for individual cases vary between 0% and 33%.

To recapitulate the rhinosurgical data of patients with SDB, the authors offer the following conclusions. The success rate of only nasal surgery for simple snoring is not available and the success rate for OSA seems to be less than 20%. The reasons for the low success rates have been articulated by Hoffstein et al[102]: "Neither the site of obstruction during apneas nor the site of generation of snoring is in the nose."

To sum up, patients may be allocated to two groups. With the majority of patients the normalization of nasal resistance leads to a positive impact on their well-being and sleep quality, but not on the severity of OSA. Even worsening of the condition has been described. In a smaller number of patients a partly distinct improvement, in some cases even healing, of an existing OSA can be achieved. Reliable criteria to identify responders have not yet been found. Therefore, the prediction of success of a rhinosurgical treatment for the individual with SDB is not possible.

REFERENCES

1. Levinus Lemnious. The touchstone of complexions. London 1581
2. Cline CL. The effects of intra-nasal obstruction on the general health. Med Surg Rep 1892;67:259–260
3. Wells WA. Some nervous and mental manifestations occurring in connection with nasal disease. Am J Med Sci 1898;116:677–692
4. Rundcrantz H. Postural variations of nasal patency. Acta Otolaryngol 1969;68:435–443
5. Hasegawa M, Saito Y. Postural variations in nasal resistance and symptomatology in allergic rhinitis. Acta Otolaryngol 1979;88:268–272
6. Desfonds P, Planès C, Fuhrman C, Foucher A, Raffestin B. Nasal resistance in snorers with or without sleep apnea: effect of posture and nasal ventilation with continuous positive airway pressure. Sleep 1998;21:625–632
7. De Vito A, Berrettini S, Carabelli A, et al. The importance of nasal resistance in obstructive sleep apnea syndrome: a study with positional rhinomanometry. Sleep Breath 2001;5:3–11
8. Lofaso F, Coste A, d'Ortho MP, et al. Nasal obstruction as a risk factor for sleep apnoea syndrome. Eur Respir J 2000;16:639–643
9. Hudgel DW, Robertson DW. Nasal resistance during wakefulness and sleep in normal man. Acta Otolaryngol 1984;98:130–135
10. Miljeteig H, Cole P, Haight SJ. Nasal resistance in recumbency and sleep. Rhinology 1995;33:82–83
11. Gluckman JL, Stegmeyer R. Nonallergic rhinitis. In: Paperella MM, ed. Otolaryngology. Philadelphia, PA: WB Saunders;1991:1892–1893
12. Feinsilver SH, Hertz G. Respiration during sleep in pregnancy. Clin Chest Med 1992;13:637–644
13. Proctor DF. The upper airways. I. Nasal physiology and defense of the lungs. Am Rev Respir Dis 1977;115:97–129
14. Scherer PW, Hahn II, Mozell MM. The biophysics of nasal airflow. Otolaryngol Clin N Amer 1989;22:265–278
15. Kuna ST, Remmers JE. Neural and anatomic factors related to upper airway occlusion during sleep. Med Clin North Am 1985;69:1221–1242
16. Butler J. The work of breathing through the nose. Clin Sci 1960;19:55–62
17. Heetderks DR. Observations on the reactions of normal nasal mucosa membrane. Am J Med Sci 1927;174:231–244
18. Olsen KD, Kern EB. Nasal influences on snoring and obstructive sleep apnea. Mayo Clin Proc 1990;65:1095–1105
19. James JE, de Burgh Daly M. Nasal reflexes. Proc Roy Soc Med 1969;62:1287–1293
20. Cole P, Haight JSJ. Posture and nasal patency. Am Rev Respir 1984;129:351–354
21. Cole P, Haight JSJ. Mechanisms of nasal obstruction during sleep. Laryngoscope 1984;94:1557–1559
22. Cassisi NJ, Biller HF, Ogura JH. Changes in arterial oxygen tension and pulmonary mechanics with the use of posterior packing in epistaxis: a preliminary report. Laryngoscope 1971;81:1261–1266
23. Cook TA, Komorn RM. Statistical analysis of the alterations of blood gases produced by nasal packing. Laryngoscope 1973;83:1802–1809
24. Cvetnic V, Cvetnic S, Grbac I. Temporary artificial obstruction of the nose and changes in gas exchange in the blood. Laryngoscope 1981;91:1001–1009

25. Hady MRA, Kodeira KZ, Nasef AH. The effect of nasal packing on arterial blood gases and acid-base balance and its clinical importance. J Laryngol Otol 1983;97:599–604
26. Zwillich CW, Pickett C, Hanson FN, Weil JV. Disturbed sleep and prolonged apnea during nasal obstruction in normal men. Am Rev Respir Dis 1981;124:158–160
27. Olsen KD, Kern EB, Westbrook PR. Sleep and breathing disturbance secondary to nasal obstruction. Otolaryngol Head Neck Surg 1981;89:804–810
28. Lavie P, Fischel N, Zomer J, Eliaschar I. The effects of partial and complete mechanical occlusion of the nasal passages on sleep structure and breathing in sleep. Acta Otolaryngol 1983;95:161–166
29. Taasan V, Wynne JW, Cassisi N, Block AJ. The effect of nasal packing on sleep-disordered breathing and nocturnal oxygen desaturation. Laryngoscope 1981;91:1163–1172
30. Johannessen N, Jensen PF, Kristensen S, Juul A. Nasal packing and nocturnal oxygen desaturation. Acta Otolaryngol Suppl 1992;492:6–8
31. Tanaka Y, Honda Y. Nasal obstruction as a cause of reduced PCO_2 and disordered breathing during sleep. J Appl Physiol 1989;67:970–972
32. Lavie P, Rubin AE. Effects of nasal occlusion on respiration in sleep. Evidence of inheritability of sleep apnea proneness. Acta Otolaryngol 1984;97:127–130
33. Suratt PM, Turner BL, Wilhoit SC. Effect of intranasal obstruction on breathing during sleep. Chest 1986;90: 324–329
34. Wetmore SJ, Scrima L, Hiller FC. Sleep apnea in epistaxis patients treated with nasal packs. Otolaryngol Head Neck Surg 1988;98:596–599
35. Houston D, Williams AJ, Finberg S, Santiago S, Wald R. Disturbed sleep and prolonged apnea during nasal obstruction in normal men [letter]. Am Rev Respir Dis 1982;125:484–485
36. Wilhoit SC, Suratt PM. Effect of nasal obstruction on upper airway muscle activation in normal subjects. Chest 1987;92:1053–1055
37. Masing H, Horbaschk G. The influence of the nose on the sleeping habits of infants. Int Rhinol 1969;7:41–45
38. McNicholas WT, Tarlo S, Cole P, et al. Obstructive apneas during sleep in patients with seasonal allergic rhinitis. Am Rev Respir Dis 1982;126:625–628
39. Leznoff A, Haight JS, Hoffstein V. Reversible obstructive sleep apnea caused by occupational exposure to guar gum dust. Am Rev Respir Dis 1986;133:935–936
40. Lavie P, Gertner R, Zomer J, Podoshin L. Breathing disorders in sleep associated with "microarousals" in patients with allergic rhinitis. Acta Otolaryngol 1981;92: 529–533
41. Rubinstein I. Nasal inflammation in patients with obstructive sleep apnea. Laryngoscope 1995;105:175–177
42. Houser SM, Mamikoglu B, Aquino BF, Moinuddin R, Corey JP. Acoustic rhinometry findings in patients with mild sleep apnea. Otolaryngol Head Neck Surg 2002; 126:475–480
43. Duchna HW, Rasche K, Orth M, Bauer T, Schultze-Werninghaus G. Anamnestic and polygraphic parameters in obstructive sleep apnea syndrome patients with reduced nasal respiration during the day in comparison with obstructive sleep apnea patients with normal nasal respiration [German]. Wien med Wschr 1996;146:348–349
44. Anch AM, Remmers JE, Bunce H III. Supraglottic airway resistance in normal subjects and patients with occlusive sleep apnea. J Appl Physiol 1982;53:1158–1163
45. Blakley BW, Mahowald MW. Nasal resistance and sleep apnea. Laryngoscope 1987;97:752–754
46. Young T, Finn L, Kim H. Nasal obstruction as a risk factor for sleep-disordered breathing. J Allergy Clin Immunol 1997;99(suppl 2):S757–S762
47. Silvoniemi P, Suonpää J, Sipilä J, Grénman R, Erkinjuntti M. Sleep disorders in patients with severe nasal obstruction due to septal deviation. Acta Otolaryngol Suppl 1997;529:199–201
48. Atkins M, Taskar V, Clayton N, Stone P, Woodcock A. Nasal resistance in obstructive sleep apnea. Chest 1994; 105:1133–1135
49. Fürst G, Riechelmann H, Mann WJ, Amedee RG. Measurement of pharyngeal pressure in patients using nasal CPAP. Am J Rhinol 1995;9:353–356
50. Schechter GL, Ware J, Perlstrom J, McBrayer RH. Nasal patency and the effectiveness of nasal continuous positive air pressure in obstructive sleep apnea. Otolaryngol Head Neck Surg 1998;118:643–647
51. Miljeteig H, Hoffstein V, Cole P. The effect of unilateral and bilateral nasal obstruction on snoring and sleep apnea. Laryngoscope 1992;102:1150–1152
52. Miyazaki S, Itasaka Y, Ishikawa K, Togawa K. Acoustic analysis of snoring and the site of airway obstruction in sleep-related breathing disorders. Acta Otolaryngol 1998; (suppl 537):S47–S51
53. Sauerland EK, Mitchell SP. Electromyographic activity of intrinsic and extrinsic muscles of the human tongue. Tex Rep Biol Med 1975;33:444–455
54. Gleeson K, Zwillich CW, Braier K, White DP. Breathing route during sleep. Am Rev Respir Dis 1986;134:115–120
55. Konno A, Togawa K, Hoshino T. The effect of nasal obstruction in infancy and early childhood upon ventilation. Laryngoscope 1980;90:699–707
56. Ohki M, Usui N, Kanazawa H, Hara I, Kawano K. Relationship between oral breathing and nasal obstruction in patients with obstructive sleep apnea. Acta Otolaryngol Suppl 1996;523:228–230
57. Kratschmer F. About reflexes of the nasal mucosa on respiration and circulation [German]. Sitzungsber Akad Wiss Wien 1870;62:147–170
58. White DP, Cadieux RJ, Lombard RM, et al. The effects of nasal anesthesia on breathing during sleep. Am Rev Respir Dis 1985;132:972–975
59. McNicholas WT, Coffey M, McDonnell T, O'Regan R, Fitzgerald MX. Upper airway obstruction during sleep in

normal subjects after selective topical oropharyngeal anesthesia. Am Rev Respir Dis 1987;135:1316–1319
60. Basner R, Ringler J, Garpestad E, et al. Upper airway anesthesia delays arousal from airway occlusion induced during human NREM sleep. J Appl Physiol 1992;73:642–648
61. Berry RB, Kouchi KG, Bower JL, Light RW. Effect of upper airway anesthesia on obstructive sleep apnea. Am J Respir Crit Care Med 1995;151:1857–1861
62. Strohl KP, Hensley MJ, Hallett M, Saunders NA, Ingram RH Jr. Activation of upper airway muscles before onset of inspiration in normal humans. J Appl Physiol 1980;49: 638–642
63. Rodgers G, Chan KH, Dahl RE. Antral choanal polyp presenting as obstructive sleep apnea syndrome. Arch Otolaryngol Head Neck Surg 1991;117:914–916
64. Duchna HW, Orth M, de Zeeuw J, et al. The impact of nasal obstruction on obstructive sleep apnea syndrome. Somnologie 2001;5:53–58
65. Kerr P, Millar T, Buckle P, Kryger M. The importance of nasal resistance in obstructive sleep apnea syndrome. J Otolaryngol 1992;21:189–195
66. Brouillette RT, Manoukian JJ, Ducharme FM, et al. Efficacy of fluticasone nasal spray for paediatric obstructive sleep apnea. J Pediatr 2001;138:838–844
67. Craig TJ, Teets S, Lehman EB, Chinchilli VM, Zwillich C. Nasal congestion secondary to allergic rhinitis as a cause of sleep disturbance and daytime fatigue and the response to topical nasal corticosteroids. J Allergy Clin Immunol 1998;101:633–637
67a. Höijer U, Ejnell H, Hedner J, Petruson B, Eng LB. The effects of nasal dilation on snoring and obstructive sleep apnea. Arch Otolaryngol Head Neck Surg 1992;118:281–284
67b. Metes A, Cole P, Hoffstein V, Miljeteig H. Nasal airway dilation and obstructed breathing in sleep. Laryngoscope 1992;102:1053–1055
67c. Schönhofer B, Wenzel M, Barchfeld T, et al. Value of intra- and extraoral devices in the treatment of obstructive sleep apnea and snoring [German]. Med Klin 1997; 92:167–174
67d. Gosepath J, Amedee RG, Romantschuck S, Mann WJ. Breathe Right nasal strips and the respiratory disturbance index in sleep related breathing disorders. Am J Rhinol 1999;13:385–389
68. Afzelius LE, Elmquist D, Hougaard K, et al. Sleep apnea syndrome—an alternative treatment to tracheotomy. Laryngoscope 1981;91:285–291
69. Nahmias JS, Karetzky MS. Treatment of the obstructive sleep apnea syndrome using a nasopharyngeal tube. Chest 1988;94:1142–1147
70. Masters IB, Chang AB, Harris M, O'Neil MC. Modified nasopharyngeal tube for upper airway obstruction. Arch Dis Child 1999;80:186–187
71. Woodhead CJ, Allen MB. Nasal surgery for snoring. Clin Otolaryngol 1994;19:41–44
72. Fairbanks DNF. Snoring: surgical vs nonsurgical management. Laryngoscope 1984;94:1188–1192
73. Low WK. Can snoring relief after nasal septal surgery be predicted? Clin Otolaryngol 1994;19:142–144
74. Illum P. Septoplasty and compensatory inferior turbinate hypertrophy: long-term results after randomized turbinoplasty. Eur Arch Otorhinolaryngol 1997;254(suppl 1): S89–S92
75. Heimer D, Scharf S, Liebermann A, et al. Sleep apnoea syndrome treated by repair of deviated nasal septum. Chest 1983;84:184–185
76. Simmons FB, Hill MW. Hypersomnia caused by upper airway obstruction. Ann Otol Rhinol Laryngol 1974;83: 670–673
77. Dreher A, de la Chaux R, Grevers G, Kastenbauer E. Influence of nasal obstruction on sleep-associated breathing disorders [German]. Laryngorhinootologie 1999;78:313–317
78. Simmons FB, Guilleminault C, Dement WC, et al. Surgical management of airway obstruction during sleep. Laryngoscope 1977;87:326–338
79. Rubin AHE, Eliaschar I, Joachim Z, Alroy G, Lavie P. Effects of nasal surgery and tonsillectomy on sleep apnea. Bull Eur Physiopathol Respir 1983;19:612–615
80. Dayal VS, Phillipson EA. Nasal surgery in the management of sleep apnea. Ann Otol Rhinol Laryngol 1985; 94:550–554
81. Caldarelli DD, Cartwright R, Lilie JK. Obstructive sleep apnea: variations in surgical management. Laryngoscope 1985;95:1070–1073
82. Aubert-Tulkens G, Hamoir M, van den Eeckhaut J, Rodenstein DO. Failure of tonsil and nose surgery in adults with long-standing severe sleep apnea syndrome. Arch Intern Med 1989;149:2118–2121
83. Sériès F, St Pierre S, Carrier G. Effects of surgical correction of nasal obstruction in the treatment of obstructive sleep apnea. Am Rev Respir Dis 1992;146(5 Pt 1):1261–1265
84. Sériès F, St Pierre S, Carrier G. Surgical correction of nasal obstruction in the treatment of mild sleep apnoea: importance of cephalometry in predicting outcome. Thorax 1993;48:360–363
85. Utley DS, Shin EJ, Clerk AA, Terris DJ. A cost-effective and rational surgical approach to patients with snoring, upper airway resistance syndrome, or obstructive sleep apnea syndrome. Laryngoscope 1997;107:726–734
86. Verse T, Pirsig W, Kroker BA. Obstructive sleep apnea and nasal polyps. [German] Laryngorhinootologie 1998; 77:150–152
87. Friedman M, Tanyeri H, Lim JW, et al. Effect of nasal breathing on obstructive sleep apnea. Otolaryngol Head Neck Surg 2000;122:71–74
88. Dagan Y. Rhinoplasty and sleep apnea syndrome. Somnologie 1997;1(suppl 2):S49
89. Lavie P, Zomer J, Eliaschar I, et al. Excessive daytime sleepiness and insomnia. Association with deviated nasal septum and nocturnal breathing disorders. Arch Otolaryngol 1982;108:373–377

90. Verse T, Maurer JT, Pirsig W. Effect of nasal surgery on sleep-related breathing disorders. Laryngoscope 2002; 112:64–68
91. Sher AE, Schechtman KB, Piccirillo JF. The efficiency of surgical modifications of the upper airway in adults with obstructive sleep apnea syndrome. Sleep 1996;19:156–177
92. Busaba NY. Same-stage nasal and palatopharyngeal surgery for obstructive sleep apnea: is it safe? Otolaryngol Head Neck Surg 2002;126:399–403
93. Antila J, Sipilä J, Tshushima Y, et al. The effect of laser-uvulopalatopharyngoplasty on the nasal and nasopharyngeal volume measured with acoustic rhinometry. Acta Otolaryngol Suppl 1997;529:202–205
94. Welinder R, Cardell LO, Uddman R, Malm L. Reduced nasal airway resistance following uvulopalatopharyngoplasty. Rhinology 1997;35:16–18
95. Kawano K, Usui N, Kanazawa H, Hara I. Changes in nasal and oral respiratory resistance before and after uvulopalatopharyngoplasty. Acta Otolaryngol Suppl 1996; 523:236–238
96. Mayer-Brix J, Mueller-Marschhausen U, Becker H, Peter JH. The incidence of pathological ENT findings in patients with clinically suspected obstructive sleep apnea syndrome. [German] HNO 1989;37:511–516
97. Williams AJ, Arand D, Yan-Go F, Clark G, Miller T. Adjuvant therapies for obstructive sleep apnea. Am Rev Respir Dis 1991;143:A590
98. Ripberger R, Pirsig W. Long-term compliance with nasal CPAP treatment for obstructive sleep apnoea: prospective study in 50 patients [German]. Laryngorhinootologie 1994;73:581–585
99. Dorn M, Pirsig W, Verse T. Management of patients with severe obstructive sleep apnea following rhinosurgical interventions. A pilot study [German]. HNO 2001;49:642–645
100. Biermann E. Nasal CPAP therapy in obstructive sleep apnea syndrome: does functional rhinosurgery improve compliance [German]? Somnologie 2001;5:59–64
101. Schäfer J. Spektralanalyse pathologischer schlafabhängiger Atemgeräusche der oberen Luftwege unter besonderer Berücksichtigung der Diagnostik und Therapie des obstruktiven Schlafapnoe-Syndroms und chronischer Rhonchopathien [thesis]. Ulm, Germany: University of Ulm; 1990
102. Hoffstein V, Mateika S, Metes A. Effect of nasal dilation on snoring and apneas during different stages of sleep. Sleep 1993;16:360–365