REVIEW

Rhinology, 43, 242-250, 2005

Nasal obstruction and its impact on sleep-related breathing disorders*

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SUMMARY

Upper airway patency is essential during sleep in order to avoid sleep-related breathing disorders (SRBD). Nasal obstruction may have a negative impact on sleep quality and must be considered to be a co-factor in the pathophysiology of SRBD.
In this paper we will discuss the relation between nasal physiology at night and sleep quality and the possible mechanisms between nasal obstruction and obstructive sleep apnea–hypopnea syndrome (OSAS). We will review the effect of the relief of nasal obstruction (with nasal dilators, medication and/or surgery) on SRBD. Also an algorithm on the management of OSAS patients when nasal surgery is indicated will be proposed.

Key words: sleep-related breathing disorders, nasal obstruction, nasal dilators, nasal resistance, nasal surgery, nCPAP

INTRODUCTION

The different clinical aspects of sleep-related breathing disorders (SRBD) include primary snoring, upper airway resistance syndrome, obstructive sleep apnea–hypopnea syndrome (OSAS) and hypoventilation syndrome related to obesity.
The most studied form of SRBD is OSAS and its incidence is 2–4% in the general adult population [1]. In all circumstances, the nose may have a great impact on the severity of SRBD. Sensitized subjects during high allergen exposure will have experienced impaired nasal breathing and likewise everyone who has had a common cold will have experienced poor nasal breathing during the night. The consequences of daily nasal obstruction on sleep quality have been well demonstrated, resulting in day-to-day discomfort, frequent complaints of poor sleep quality and daytime fatigue. Risk factors for sleep-disordered breathing are numerous and include central obesity, male gender, smoking, alcohol consumption, upper airway obstruction and craniofacial abnormalities. Nasal obstruction must be considered to be a co-factor in the pathophysiology of SRBD but the relation between cause and effect remains a matter of debate [2].

THE NOSE AT NIGHT

Nasal resistance

Nasal airway resistance is responsible for approximately two thirds of the total airway resistance in wakefulness [3]. Unlike the oropharyngeal segment in the upper airway which is collapsible when muscular tone decreases during sleep, the nasal airway segment has a more rigid framework and is not collapsible. The nasal valve contributes most to total nasal resistance and can be thought of as a short resistor of a few millimetres in length [4]. Nasal resistance is mainly under the control of the sympathetic nervous system and dependent on the anatomical location of the nasal valve. Alar dilation muscles are synchronized with inspiration to prevent alar collapse but their role in nasal sleep resistance is limited.
The physiology of the nasal airway creates a dynamic situation where nasal resistance may vary under different circumstances and secondary to the nasal cycle. Nasal resistive reflexes which are anatomically mediated may change the nasal resistance [5]. Bilateral nasal decongestion may result from exercise and hypercapnia. On the other hand, hyperventilation with resulting hypocapnia is followed by an increase in nasal resistance.
The influence of posture is also well recognized in that nasal resistance increases in the recumbent position owing to a hydrostatic component. When a subject sleeps on the right side nasal resistance is higher in the right nasal cavity and vice-versa. This hydrostatic component contributes to the magnitude of the nasal resistive reflexes. For example, the amplitude of the nasal cycle is lower when standing up than when sleeping in the recumbent position [5]. This can explain the “paradoxical nasal

*Received for publication: August 3, 2004; accepted: May 1, 2005

obstruction" phenomenon. When a patient has a fixed unilateral obstruction, the nasal resistance is higher in the affected side than in the non-affected side. When the normal side undergoes the congested phase of the nasal cycle or when the patient is sleeping on the same side as his non-affected nostril, it results in bilateral occlusion and the patient will often attribute the problem to the normal side. Unilateral pressure on the shoulder or hip girdle area causes ipsilateral nasal congestion and contralateral decongestion. This phenomenon is called the corporo-nasal reflex. All these nasal reflexes play an important role in modulating upper airway patency during sleep.

Nasal breathing is for many reasons the preferential breathing route in wakefulness and in sleep even if resistance to airflow is high. Nasal airflow enters the nasal vestibule at the anterior nares with an air velocity of 2–3 m/s. The cross-sectional area at the anterior nares is about 90 mm^2. Where the nasal airflow crosses the nasal valve the cross-sectional area is decreased to 30 mm^2. Owing to the Venturi effect (as a given volume of fluid moves through a conduit of decreasing diameter, the velocity of the fluid will increase), the air velocity increases to 12–18 m/s at the nasal valve level. This contributes to a higher probability of upper airway collapse in the nasal valve area owing to the Bernouilli effect (as a fluid flows a negative pressure develops at the periphery of the flow and if the flow velocity increases so does the negative pressure). If the negative pressure increases at the interface between the airflow and the nasal valve compartment, the force to collapse increases. This is the case when compensatory forces from the alar and/or upper lateral cartilages are lacking (after surgical resection for instance). After passing the nasal valve, the airflow reaches the nasal cavity where the cross-sectional area is greater (130 mm^2) and air velocity decreases to 2–3 mm/s.

Nasal patency and SRBD are thus closely related. Although there is no strict linear relationship between the two, there is clear evidence that nasal patency must be maintained for patients with SRBD. Studies performed in normal subjects, in patients with SRBD or in patients with symptomatic nasal obstruction support the most favoured pathophysiological mechanism for the role of upper airway obstruction in the genesis of SRBD: the Starling resistor model. In this model, the pharynx which is the most collapsible part of the upper airway is seen as a collapsible resistor [6]. The maximal flow rate at the pharyngeal level is related to the pressure in the upstream segment (the nasal segment) and to the transmural pressure. Once the negative transmural pressure reaches a certain level (the critical closing pressure: P_{crit}) the calibre of the segment decreases and begins to either oscillate (snoring) or partly collapse (hypopnea) or totally collapse (apnea). This single model shows that the determinants of maximal flow rate through the pharyngeal airway are the upstream resistance (i.e. the nasal resistance), the transmural pressure, and the compliance of the pharyngeal wall. The Pcrit varies between the subjects (normal, snorers, and apneics) and between the sleep stages. The negative intraluminal pressure and the Starling resistor model are the mechanisms which explain upper airway obstruction during sleep and explain why nasal patency is so important.

Nasal patency and SRBD

Many studies have focused on the influence of nasal patency and its affect on sleep parameters and SRBD.

The effects of uni-(bi)lateral nasal obstruction, the effects of nasal anesthesia and the effects of nasal vasoconstriction have been studied in healthy subjects.

White and co-workers hypothesized that SRBD in patients with nasal obstruction are secondary to a loss of afferent nasal receptors which are responsible for ventilation control [7]. They examined the effects on sleep of blocking the nasal trigeminal sensory receptors using 4% lidocaine local anesthesia. Administration of lidocaine increased nasal and pharyngeal obstruction, disturbed sleep, increased the number of awakenings and apnea and thus sleep quality deteriorated. It was also demonstrated that upper airway anesthesia reduces the phasic activity of the most important upper airway dilating muscle during sleep, namely the genioglossus [8]. Nitric oxide is also considered to be an aerotransmitter between the nose and the lung and its role in maintaining upper airway patency using efferent pathways has been underlined [9]. Others investigators have looked at the role of experimental nasal occlusion on sleep quality in normal subjects. Using petroleum jelly and cotton to occlude the nostrils or adhesive tapes on nostrils and carrying out polysomnographic studies with or without the devices, it has been repeatedly shown that sleep quality worsened [10-14]. Complete nasal packing has also induced similar effects [15,16]. These studies performed on volunteers or on subjects with no SRBD but subjected to complete nasal obstruction with nasal packing suggest that nasal obstruction may trigger the induction of sleep disordered breathing in normal individuals and that nasal breathing increases ventilation efficiency by stimulating certain sensory trigeminal receptors in the nasal mucosa. However, the likelihood of developing SRBD after nasal resistance variation or nasal anaesthesia does not concern all the volunteers.

The relation between nasal patency and SRBD has also been studied in patients with SRBD and in patients with abnormal nasal patency. Miljeteig et al. studied 683 patients referred for snoring and/or apnea evaluation and assessed nasal resistance and sleep parameters [17]. They found no correlation between the unilateral or bilateral increase of nasal resistance and snoring and/or apnea and no direct relation between awake seated nasal resistance and sleep parameters. This was also confirmed by others studies [18-20].

Some others studies, however, suggested a relationship based on four different arguments.

Firstly, patients with a complaint of nasal obstruction and SRBD are at higher risk of developing OSAS [21,22].

The contribution of nasal resistance to OSAS is however weak;

2.3% of the variance in the apnea-hypopnea index (AHI) was explained by nasal resistance [21]. In a cohort study of patients referred for evaluation of SRBD, Liistro et al. found a correlation between the Mallampati score (MS; patients were asked to open the mouth wide open with voluntary protrusion of the tongue without phonation and were graded from 1 to 4) and simple nasal examination (patients were asked to gently block one nostril and to inspire through the unoccluded nostril, then nasal obstruction was reported if the examiner heard a noise suggesting nasal obstruction) and some sleep parameters. Body mass index, neck circumference and MS associated with nasal obstruction correlated with AHI [22]. Thus the presence of a high MS with concomitant nasal obstruction is associated with an increased risk of OSAS.

Secondly, patients with SRBD, both snorers and OSAS patients, switch more frequently from nasal to oronasal breathing during sleep if nasal obstruction is present. This may lead to an increase in respiratory effort and may result in alveolar hypoventilation [23,24].

Thirdly, cohort studies have demonstrated that patients with night-time symptoms of rhinitis or with nasal congestion are at higher risk of developing SRBD and of snoring [25,26].

Finally, total nasal resistance in a non-obese subgroup of patients measured in the supine position correlated with some sleep parameters. In this subgroup, Virkkula et al. demonstrated a positive relationship between total nasal resistance and the AHI ($r = 0.50$, $p < 0.05$) and oxygen desaturation ($r = 0.58$, $p < 0.05$)[27].

All together, these studies suggest a weak relation between nasal resistance and SRBD for the vast majority of the patients. It is also important to point out that this relationship may be more pronounced in a subgroup of patients if nasal resistance is measured in the supine position. Moreover, nasal obstruction appears to increase the risk of developing SRBD.

Several investigators have evaluated the relation between nasal obstruction secondary to sino-nasal disease and SRBD. Allergic rhinitis causes nasal obstruction and polysomnographic studies during increased allergen exposure have shown an increase in the AHI, in excessive daytime sleepiness, in snoring and in micro-arousal per hours of sleep though this increase in the AHI was not clinically relevant [26,28-31). In patients with allergic rhinitis treated with a nasal steroid, sleep disturbances and daytime fatigue tended to improve [32]. The exact role of allergic rhinitis, however, in poor sleep quality may also be secondary to medication such as antihistamines which are frequently prescribed in this disease and are known to induce somnolence [33]. The poor quality of sleep among patients with allergic rhinitis may be due to mechanisms other than nasal obstruction [33]. Inflammatory mediators secondary to allergic inflammation have a diurnal variation and peak during the early morning. This peak could explain the symptoms of allergic rhinitis on waking. This may result from a night-time decrease in glucocorticoid receptor binding affinity. It is also important to point out that sympathetic tone decreases at night producing a relative parasympathetic excess. Sleep quality may therefore decrease in patients with allergic rhinitis through different mechanisms: nasal obstruction, postural changes, clinical variations of inflammatory mediators or adverse effects of antihistamine therapy.

Anatomical obstruction owing to septal deviation or hypertrophy of the inferior turbinate may also predispose to SRBD. This is also true for patients with nasal polyposis [34-36].

Table 1. Effect of medical therapy on sleep-related breathing disorders.

Authors	Medication	Number of patients	Nasal disease	Sleep parameters
Craig, 1998	fluticasone	20	Perennial allergic rhinitis	No PSG Subjective sleep quality improvement
Hughues, 2003	budesonide	22	Perennial allergic rhinitis	No PSG Subjective sleep quality improvement and daytime fatigue
Ratner, 2003	fluticasone vs montelukast	705	Seasonal allergic rhinitis	No PSG Night-time symptoms score decrease FP > M
Craig, 2003	fluticasone	32	Perennial allergic rhinitis	No difference with objective data but well with subjective
Kiely, 2004	fluticasone	24	Snorers + Rhinitis	Slight decrease of AHI and related to NR.

PSG: polysomnographic data
FP: fluticasone propionate
M: montelukast
AHI: apnea–hypopnea index
NR: nasal resistance

Table 2. Effect of nasal dilators on sleep-related breathing disorders

Authors	Nasal dilators	Number of patients	Sleep parameters and remarks
Hoijer, 1992	Nozovent	10	Improvement
Hoffstein, 1993	Nozovent	15	No change except snoring in slow wave sleep
Liistro,1998	Breathe Right	10	No change
Todorova, 1998	Breathe Right	30	No change
Gosepath, 1999	Breathe Right	26	Reduced in 4/26
Bahamman, 1999	Breathe Right	18	No change
Schönhofer, 2000	Nozovent	21	No change
Pevernagie, 2000	Breathe Right	12	No change except snoring index in chronic rhinitis patients
Djupesland, 2001	Breathe Right	18	No change except slight reduction in AHI when MCA < 0.6 cm^2 (6/18)

AHI: apnea–hypopnea index

MCA: mean cross sectional area

TREATMENT OF NASAL OBSTRUCTION AND ITS IMPACT ON SRBD

If nasal obstruction and increased nasal resistance may promote SRBD, it seems logical to study the effects of a reduced resistance on sleep parameters. This has been done in a variety of ways with medication, nasal dilators and surgical procedures.

Medication

Medical relief of nasal obstruction has been studied in patients with allergic rhinitis and the effects analysed with questionnaires or polysomnographic data.

Subjective sleep quality improves in patients with allergic rhinitis after intranasal steroid (Table 1). This has been confirmed by two placebo-controlled studies performed with fluticasone propionate and budesonide in patients with perennial allergic rhinitis [32,37]. In seasonal allergic rhinitis, the night-time score for fluticasone propionate is better than for the antileukotriene montelukast in a large cohort of patients [38]. Finally, two recent studies have tried to demonstrate the objective modification of sleep parameters in patients with allergic rhinitis or in snorers with rhinitis. Both studies were performed with the topical application of fluticasone propionate. Craig et al., found no difference in the polysomnographic data but an improvement in subjective sleep parameters when treating patients with perennial allergy with fluticasone propionate [39]. On the other hand, Kiely et al., have demonstrated a more pronounced though slight decrease in the AHI in snorers with rhinitis treated with fluticasone propionate compared with placebo [40]. Nasal obstruction secondary to allergic inflammation has an impact on sleep quality and topical corticoid therapy seems to have a positive effect on subjective sleep quality and also on polysomnographic data in one study [32,37-40].

Nasal dilators

Nasal dilators are an attractive method of decreasing nasal resistance in the valve area and subsequently have a positive impact on snoring and/or apnea [41]. Measurements of nasal resistance in awake subjects with the device have shown a reduction in resistance, though not uniform, depending on the compliance of the nasal vestibule walls [42]. The dimension of the nasal valve is increased by approximately 30%. There are actually two devices commercially available as nasal dilators: Nozovent®, an internal device, and Breathe Right®, an external device. These devices have been studied in patients with polysomnographic measurements in nine studies (Table 2)[43-51].

The conclusions from these studies are that nasal dilators may reduce the subjective sensation of snoring but the objective measurements of snoring as well as sleep parameters such the AHI reveal that nasal dilators are ineffective for the vast majority of the SRBD patients. Only two studies have shown a positive effect on the AHI but in less than 30% of the patients [43,46]. The nasal dilators may be more effective in patients with SRDB with concomitant chronic rhinitis [50]. Djupesland et al. found that Breathe Right® was an effective treatment of snoring in a subgroup of patients with morning nasal obstruction and when acoustic rhinometry has revealed a minimal cross-sectional area <0.6 cm^2 [51]. Based on this information, nasal dilators are ineffective for the vast majority of apneic patients but may be recommended as a trial for non-apneic snorers. Nasal dilators have no side effects and are relatively inexpensive. Patients with nasal valve stenosis and/or chronic rhinitis are the best candidates.

Nasal and sinus surgery

Nasal and sinus surgery are often performed to reduce nasal obstruction and their effects on SRBD may also be expected. Various surgical procedures are described to increase nasal patency: septo(rhino)plasty, turbinoplasty, radiofrequency tissue volume reduction of the inferior turbinates, functional endoscopic endonasal surgery or correction of the nasal valve.

Table 3. Effects of nasal and sinus surgery on sleep-related breathing disorders.

Authors	Number of patients	AI before / after	AHI before / after	Results
Heimer, 1983	3			3 patients cured
Rubin, 1983	9	37.8/26.7		$p < 0.5$
Dayal, 1985	6		46.8/28.2	Not significant
Caldarelli, 1985	23	44.2/41.5		Not significant
Aubert, 1989	2	47.5/48.5		Not significant
Séries, 1992	20		39.8/36.8	Not significant
Séries, 1993	14	17.8/16		Not significant
Utley, 1997	4		11.9/27	Worse
Verse, 1998	2		14/57.7	Worse
Friedman, 2000	50 (only 22 OSAS)		31.6/39.5	Worse
Verse, 2002	26		31.6/28.9	Not significant 3 OSAS patients cured
Kim, 2004	21		39/29	$p = 0.0001$ 4 OSAS patients cured

OSAS: obstructive sleep apnea syndrome.

The primary goal of these procedures is related to the nasal obstruction itself, to the improvement of SRBD or both. Improvement of compliance with nasal continuous positive airway pressure therapy (nCPAP) by reducing nasal obstruction may also be an objective. Subjective improvement in sleep quality and reduction of snoring have been described in a study without polysomnographic data by correction of the nasal valve using a septorhinoplasty approach and spreader grafts insertion [52]. This was also confirmed in a study examining the effects of nasal surgery on snoring where 48/96 patients reported complete relief of snoring after nasal surgery [53].

There are objective data with pre-operative and postoperative polysomnographic studies regarding the effects of nasal surgery on SRBD in 12 studies (Table 3)[36,54-64). Although some of these studies have demonstrated a slight decrease in the AHI for some patients, the success rate (as defined by an AHI reduction by at least 50% and an AHI value < 20) is very low. Verse et al. reported a success rate of 15.8% (3/19 apneic patients) using these criteria [63]. Friedman et al. have also suggested that postoperative polysomnographic data may be worse for mild OSAS patients after nasal obstruction relief [62]. They explain this paradoxical effect of nasal surgery by the fact that nasal obstruction relief may allow the patients to sleep in deeper sleep stages. Therefore apnea and sleep fragmentation are increased because patients sleep more comfortably. Friedman et al. have also demonstrated that nCPAP pressure requirements are decreased after nasal surgery. The mean nCPAP titration level was 9.3 cm H_2O before surgery vs. 6.7 cm H_2O after surgery [62].

Nasal surgery, as with nasal dilators, may improve subjective snoring or daytime fatigue but cure of sleep apnea occurs only in approximately 15–20%. Results of nasal surgery in patients with sleep apnea/hypopnea are therefore barely predictable.

HOW TO MANAGE NASAL DISEASE AND SURGERY IN nCPAP PATIENTS?

Patients with moderate to severe OSAS are candidates for nCPAP [1]. This treatment remains the most effective for reducing snoring and morbidity related to apneic episodes [65]. This treatment, however, may have adverse effects of which nasal problems are the most frequently encountered [66]. It has been demonstrated that nCPAP therapy induces nasal mucosal inflammation and promotes nasal hyperreactivity [67]. More than half of patients using nCPAP therapy present with significant nasal complaints [65]. Rhinorrhea induced by nCPAP may be treated with anticholinergic medication (ipratropium bromide). Nasal congestion may appear after some days of nCPAP use and may be treated by topical nasal steroids or nasal vasoconstrictors for a few days. Saline nasal spray and heated humidification are commonly used to prevent problems related to nCPAP [68]. Corrective nasal surgery or turbinates volume reduction may help patients to tolerate nCPAP therapy [69].

Currently, there is no widely accepted consensus on the management of patients with OSAS undergoing nasal surgery. In general, such patients should undergo a preoperative polysomnographic study and OSAS should be treated in the preoperative period with nCPAP therapy. General anesthesia during surgery should be kept to a minimum length of time and appropriate sedatives, anesthetics and analgesics should be given (Figure 1). Patients with OSAS need to be closely monitored in the postoperative period. Although preferable, the authors do not consider a stay in intensive care mandatory. Minimum requirements, however, include nocturnal oxymetry with appropriate treatment in the event of oxygen desaturation.

A crucial question when performing nasal surgery in patients with OSAS is the use of postoperative nasal packing. Nasal packing is a standard procedure after nasal surgery and is supposed to decrease postoperative bleeding and the risk of synechiae. Nasal packing, however, has many adverse effects such as pain, discomfort, infection and obligatory mouth breathing. The complete obstruction of the nostril after surgery

in patients with OSAS may be associated with sleep interference as the so-called nasopulmonary reflex is abolished and blood gas tension may reveal a relative hypoxemia [70-72]. Other options, therefore, might be proposed in the postoperative period such as septal suturing without nasal packing, or the insertion of a nasal pack with an airway tube inside [73]. Application of nCPAP therapy may sometimes be difficult and oronasal masks are in some cases essential. An oronasal mask allows the application of the positive pressure through the mouth as long as the nose is blocked. There is no absolute consensus on the length of duration of the nasal packing nor on the duration of hospitalization but care should be taken to ensure that after removal of the packing patients will tolerate nCPAP well before being discharged (Figure 2). After nasal surgery, the apneic patient needs to undergo a second polysomnographic study to assess the benefit of the surgical procedure. This second polysomnography should be done some days after nCPAP therapy has been stopped. An easy but not totally accepted method is to carry out the assessment during a split night using the first part of the night for the SRBD diagnosis and the second part of the night to titrate the nCPAP level if the patient fulfills the criteria for this treatment.

CONCLUSION

Nasal obstruction may trigger sleep disorders in normal people and make worse SRBD in patients with apnea, hypopnea, upper airway resistance and/or primary snoring. Nasal examination and measurement of nasal resistance are mandatory in the management of patients with SRBD. Treatment is based on topical medication, nasal dilators or surgical procedures. Results are unpredictable in the vast majority of patients. Subjective analysis with questionnaires on snoring or daytime fatigue or excessive daytime sleepiness has revealed that the treatment of nasal disorders in SRBD patients may be beneficial. Objective data, however, with pre- and post-therapy polysomnographic studies are far less encouraging. The success rate of nasal surgery for instance appears to be less than 20% although normalization of nasal resistance is achieved in most of the cases. It should also be noted that nasal surgery may even worsen SRBD. Finally, rhinologic procedures may be a consideration for patients with poor compliance with nCPAP therapy. The nasal airway segment may also be considered when a multilevel strategy is proposed to the patient with SRDB.

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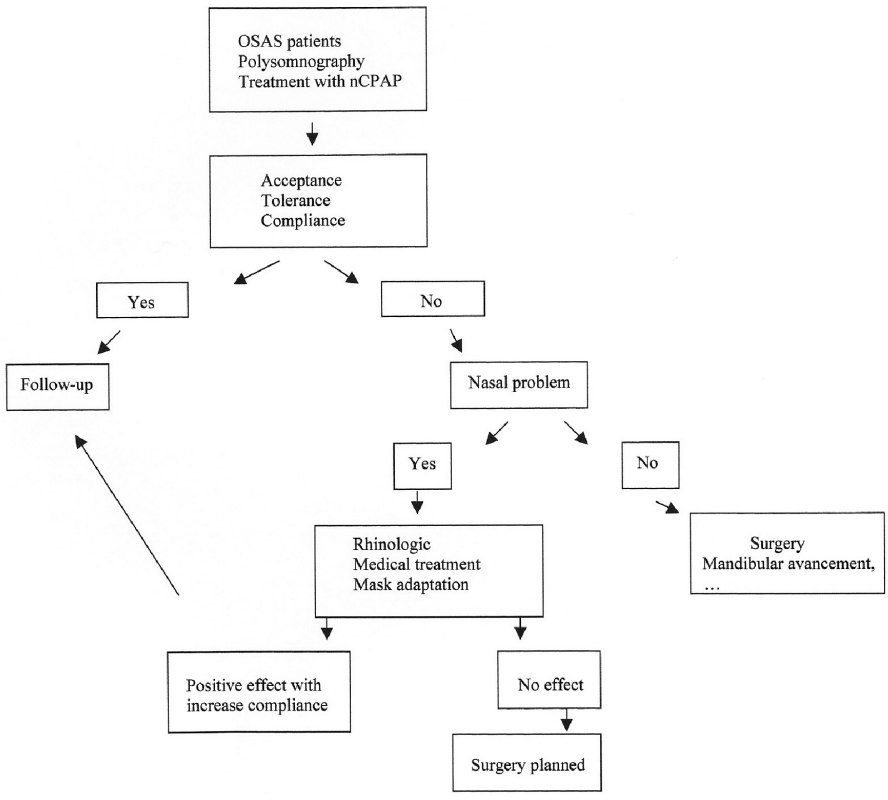

Figure 1. Management of nasal surgery in the apneic patient.

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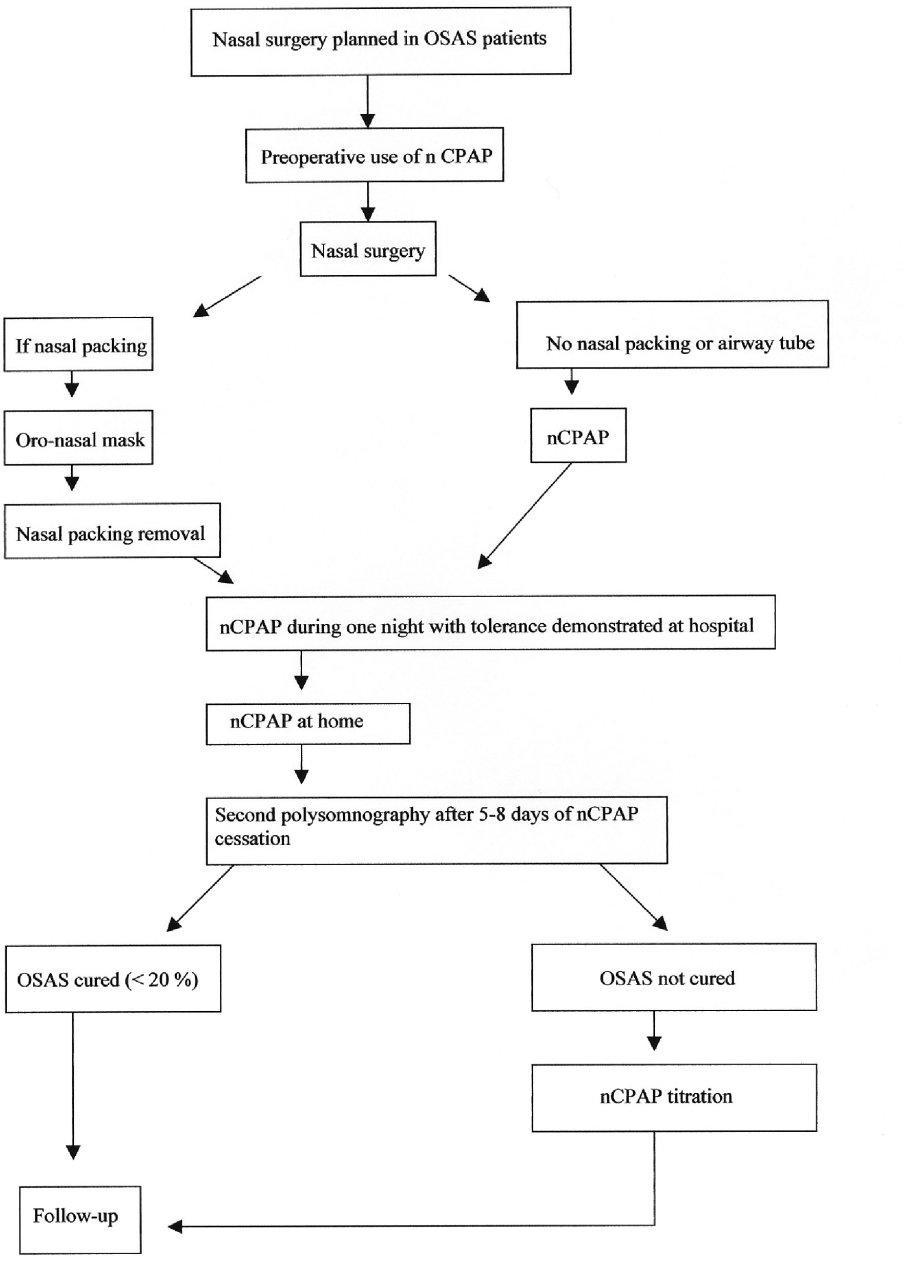

Figure 2. Management of nasal surgery in the apneic patient.

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