REVIEW

The role of the nose in the pathogenesis of obstructive sleep apnoea and snoring

M. Kohler*, K.E. Bloch#,¶ and J.R. Stradling*

ABSTRACT: Data from observational studies suggest that nasal obstruction contributes to the pathogenesis of snoring and obstructive sleep apnoea (OSA). To define more accurately the relationship between snoring, OSA and nasal obstruction, the current authors have summarised the literature on epidemiological and physiological studies, and performed a systematic review of randomised controlled trials in which the effects of treating nasal obstruction on snoring and OSA were investigated.

Searches of bibliographical databases revealed nine trials with randomised controlled design. External nasal dilators were used in five studies, topically applied steroids in one, nasal decongestants in two, and surgical treatment in one study.

Data from studies using nasal dilators, intranasal steroids and decongestants to relieve nasal congestion showed beneficial effects on sleep architecture, but only minor improvement of OSA symptoms or severity. Snoring seemed to be reduced by nasal dilators. Nasal surgery also had minimal impact on OSA symptoms.

In conclusion, chronic nasal obstruction seems to play a minor role in the pathogenesis of obstructive sleep apnoea, and seems to be of some relevance in the origin of snoring. The impact of treating nasal obstruction in patients with snoring and obstructive sleep apnoea on long-term outcome remains to be defined through randomised controlled trials of medical and surgical therapies.

KEYWORDS: Nasal obstruction, nose, obstructive sleep apnoea, sleep-disordered breathing, snoring

Obstructive sleep apnoea syndrome (OSAS) is a common disorder affecting 2–4% of males and 1–2% of females in middle age [1, 2]. It is caused by periodic reduction or cessation of airflow during sleep resulting from pharyngeal narrowing or collapse. Risk factors for OSAS in adults may include obesity, male sex, craniofacial dysmorphia, hypothyroidism and nasal obstruction [1, 3–5]. The prevalence of snoring is high; 25–50% of middle-aged males are estimated to snore regularly [3, 6]. Male sex, obesity, alcohol, sedatives, smoking and nasal obstruction are generally believed to be causative factors in the pathogenesis of snoring [3, 7–10].

Data from early physiological studies suggest that upper airway narrowing and consequent snoring and apnoea can be induced by subatmospheric nasal pressure [11], indicating the upper airway may resemble a Starling resistor with a collapsible segment in the oropharynx [12]. If the pharynx indeed behaves as a true Starling resistor then it may be destabilised during sleep by the decreased pharyngeal luminal pressure associated with inspiratory nasal obstruction so that vibrations (snoring) and a partial collapse occur. In theory, inspiratory nasal obstruction would not provoke continuing collapse of the pharynx. This is because once there is no flow, upstream resistance becomes irrelevant, intra-pharyngeal pressure returns to atmospheric, and the pharynx would re-open, a cycle generating snoring in theory but not apnoeas. However, it is unlikely that the pharynx does behave as a perfect Starling resistor, partly due to hysteresis and partly due to surface tension forces from mucus, which will tend to hold the pharynx closed once collapsed. Various observational and cross-sectional studies have documented a relationship between chronic nasal obstruction, snoring and obstructive sleep apnoea (OSA) [4, 5, 13, 14].

Therefore, it seems reasonable to assume that nasal obstruction plays a role in the pathogenesis of snoring and, to some extent, OSA, and improved nasal patency might thus alleviate sleep-disordered breathing (SDB).

Recently, different interventional trials with randomised controlled design have been published on the topic and have added important new data. Therefore, the current authors have reviewed the literature to clarify the role of the nose in the pathogenesis of OSA and snoring. Findings from physiological, observational and cross-sectional studies concerning the relationship between the nose, snoring and OSA will be discussed before systematically reviewing randomised controlled interventional trials. Finally, a suggestion for future research in this field is given and the authors approach to patients with chronic nasal obstruction presenting in a sleep clinic is briefly described.

BASIC CONSIDERATIONS

The nose usually accounts for ~50–60% of the total airway resistance, and the anterior part of the nose, including the nasal valve, contributes most to total nasal resistance (NR) in a normal nasal airway [15]. The lumen of the nasal valve is modulated by the alar, procerus and compressor naris muscles, as well as by erectile mucosa. Other sites of nasal airflow resistance are the nasal vestibule and nasal turbinates. NR is predominantly influenced by mucosal swelling of the turbinates, which fill a large proportion of the nasal cavity. During exercise, sympathetic nasal mucosal vasoconstriction in the capacitance vessels can decrease NR markedly, similar to the effects of sympathomimetic medication, such as xylometazoline [16].

In up to 80% of healthy individuals, mucosal congestion undergoes a cyclical reciprocal change across the two sides of the nose every 0.5–10 h, known as the nasal cycle [17, 18]. This cycle can be interrupted by a reflex mediated by pressure on the side of the chest wall [19–21], consequently in the decubitus position, thus the upper nostril becomes clearer and the lower more congested. Recent publications have documented the existence of the nasal cycle during sleep, but the physiological role remains largely unknown [22]. NR is highly variable both over time [23] and during sleep [16], but its interaction with sleep architecture and SDB has not been investigated rigorously because convenient and unobtrusive measurement techniques have been lacking until recently [16].

In addition to the mechanism described above, nasal congestion increases on lying down due to a hydrostatic capillary pressure rise. These physiological effects may have important implications in the pathogenesis of OSA and snoring.

CLINICAL CONSIDERATIONS

Nasal obstruction may result from rhinitis and anatomic abnormalities. Primary anatomical abnormalities are septal deviation, nasal polyps and hypertrophied turbinates, among others. The most common rhinitis syndrome causing nasal obstruction is the allergic form, but a variety of nonallergic rhinitis types are recognised in clinical practice (table 1) [24]. Vasomotor rhinitis, in which no specific aetiology can be found, represents almost two-thirds of all cases of nonallergic rhinitis [25]. Data from large epidemiological studies suggest

that the prevalence of allergic rhinitis compared with nonallergic rhinitis is 3:1 [26, 27].

Allergic rhinitis affects 9–42% of the population [24], and peak prevalence is found in early adulthood. It is caused by inhalation of airborne allergens resulting in production of immunoglobulin (Ig)E antibodies, which bind to IgE receptors on mast cells in the nasal mucosa and to basophils in the blood. As a consequence, mast cells release chemical mediators and cytokines that lead to inflammation of the nasal mucosa resulting in nasal congestion, sneezing, nasal pruritus and rhinorrhoea [28].

Data from epidemiological and physiological studies suggest that nasal congestion is a risk factor for snoring and OSA. Oral breathing during sleep has been shown to increase pharyngeal resistance and the propensity to OSA [29]. SURATT et al. [30] induced obstructive apnoeas in eight normal males by occluding the nose with petrolatum gauze. LAVIE et al. [31] investigated the influence of partial and complete obstruction of the nose in 10 normal subjects and found a significant increase in the number of apnoeas during sleep. ZWILLICH et al. [32] reported a loss of deep sleep stages and a two-fold increase in sleep arousals in 10 male subjects during an upper respiratory infection associated with nasal obstruction.

Based on the theory that nasal receptors are sensitive to airflow, and may have a reflex effect on ventilation and muscle tone in the upper airways, McNICHOLAS et al. [33] found that ventilation was greater during obligate nasal breathing than during mouth breathing in normal subjects. These findings are supported in another study, which showed that resting breathing frequency and minute ventilation were also greater with nose rather than with mouth breathing in awake normal males [34].

Data from an electromyographic study in healthy males by BASNER et al. [35] showed that upper airway tone is lower with oral breathing than nasal breathing, suggesting that upper airway dilating muscle activity may be modulated by receptors in the nasal mucosa sensitive to airflow or pressure. However, two other studies found that the breathing route did not influence electromyographic activity of genioglossus muscles

TABLE 1	Causes of chronic rhinitis
Allergic rhinitis	
Seasonal, perennial, occupational allergic rhinitis	
Nonallergic rhinitis	
Perennial rhinitis with nonallergic triggers	
Idiopathic rhinitis (basophilic nasal disease, NARES, vasomotor rhinitis)	
Atrophic rhinitis	
Chronic sinusitis	
Drug induced	
Metabolic conditions (acromegaly, oestrogen related, hypothyroidism)	
Structural causes (e.g. nasal polyps, septal deviation, adenoid hyperplasia)	
Neurogenic (e.g. nociceptive, irritant)	
Vasculitis/autoimmune and granulomatous diseases	

NARES: nonallergic rhinitis with eosinophils syndrome.

in normal subjects, but other pharyngeal dilators were not studied [36, 37].

There is evidence from various observational and cross-sectional studies that objectively measured increases in NR [4, 5, 38] and allergic rhinitis [13, 39–41] are associated with OSA. LOFASO et al. [5] performed posterior rhinomanometry in 528 patients, and found higher NR in patients with OSA than in patients without OSA. In a large population-based study, YOUNG et al. [13] identified chronic nasal congestion as a risk factor for OSA. Participants who reported nasal congestion due to allergic rhinitis were 1.8 times more likely to suffer from moderate-to-severe SDB than those without nasal congestion. However, there was no such association in participants with chronic night-time symptoms of rhinitis without allergy, and nasal airflow measured by rhinometry was not correlated with sleep apnoea.

Recent publications have suggested that daytime sleepiness in patients with allergic rhinitis is caused by disrupted sleep due to nasal congestion [42–45]. There is evidence from three randomised controlled trials that intranasal steroids improve subjective daytime sleepiness in patients with chronic allergic rhinitis [42–44]. Although these studies did not specifically include patients with OSA, and no data are given on snoring, the subjective improvement in daytime sleepiness provides an indirect suggestion that nasal congestion due to allergic rhinitis may lead to sleep fragmentation from OSA or snoring.

Snoring has been associated with chronic nasal congestion in multiple cross-sectional studies. Data from the Wisconsin sleep cohort study [14] showed that chronic rhinitis is an independent risk factor for snoring. VIRKKULA et al. [46] found that high NR was related to snoring in male subjects who were referred to an ear, nose and throat (ENT) hospital for evaluation of suspected SDB. NR was found to be a determinant of the frequency of snoring in a cross-sectional study including 361 snorers [47].

The potential effects of surgical treatment of chronic nasal obstruction on snoring and OSA have been investigated in various nonrandomised, uncontrolled trials [48–53]. In an early study, DAYAL and PHILLIPSON [51] investigated the effect of nasal valve correction on snoring and OSA in six patients, and found significant improvement in snoring and apnoea/hypopnoea index (AHI) post-operatively. FAIRBANKS [52] found post-operative elimination of snoring in 77% of patients who underwent operative correction of nasal septum and turbinate deformity. SÉRIÈS and co-workers [49, 50] found that NR decreased (at 2–3 months) after surgical intervention to the nose (septoplasty, turbinectomy and polypectomy), which was associated with a decrease of AHI to <5 in patients with normal cephalometrics, but not in patients with abnormal cephalometrics. In the study by FRIEDMAN et al. [53], which included 50 patients with nasal airway obstruction and OSA, nasal surgery did not improve respiratory disturbance index and only 34% of the patients noticed an improvement in snoring. In the same study, continuous positive airway pressure (CPAP) pressure levels required to correct OSA decreased significantly after nasal surgery. However, in a recently published observational, uncontrolled, prospective study including 40 snoring males, operative treatment of nasal

obstruction decreased NR but did not improve snoring, nocturnal breathing or sleep architecture [48].

Adenotonsillar hypertrophy is a frequent cause of OSA in children and may be associated with impaired nasal patency [54–56]. Adenotonsillectomy has been shown to improve oxygen dip rate during sleep, sleep disturbance and multiple daytime symptoms compatible with sleep disturbance in a partially controlled trial including 61 snoring children [57]. MITCHELL and KELLY [58] found that children with severe OSA who undergo adenotonsillectomy show a significant improvement in respiratory disturbance index and quality of life after surgery, but OSA did not fully resolve in the majority of these children. Similarly, a recently published prospective survey of children with OSA submitted to adenotonsillectomy showed that almost 50% of the children still had abnormal polysomnography after surgery [59]. It has to be mentioned that there is an absence of randomised controlled trials investigating the efficacy of adenotonsillectomy in the treatment of OSA in children [60].

SEARCH AND REVIEW METHODS FOR IDENTIFICATION OF RANDOMISED CONTROLLED STUDIES

Trials concerned with nasal conditions and SDB were identified by searches of bibliographic databases including EMBASE, PubMed, and hand-searching of respiratory journals. All abstracts found in the databases were assessed by two authors (M. Kohler and J.R. Stradling) to determine potential relevance for full review. Subsequently the full text of studies with randomised controlled (single- or double-blind) design including adults of either sex was reviewed. No restriction was placed upon the kind of intervention to the nose or the duration of the study.

Surgical intervention

As nasal obstruction in patients with OSA or snoring may result from septal deviation, nasal polyps and hypertrophied turbinates among various other abnormalities, surgical intervention (e.g. temperature-controlled radiofrequency tissue ablation and septoplasty) seems to be a potential therapeutic option. Currently there is no randomised controlled study investigating the effects of surgical nasal intervention either on OSA severity and symptoms, or on objective measures of snoring. However, POWELL et al. [61] randomly assigned 22 CPAP-treated patients with turbinate hypertrophy in a pilot study to either temperature-controlled radiofrequency reduction of turbinate hypertrophy (n=17) or placebo control (n=5) to assess the feasibility of a randomised controlled trial in this setting. Unfortunately, there is no explanation given for the different size of the two groups. The primary outcome was a change in the blinded examiners' findings of nasal obstruction on a 10-cm visual analogue scale. Secondary outcomes included unblinded examiner assessments of nasal obstruction on a visual analogue scale, objective and subjective adherence to CPAP, Epworth sleepiness scale and the 36-item Short-Form Health Survey. The treatment effect on nasal obstruction assessed with the visual analogue scale by the blinded examiner was -0.9 cm (95% confidence interval (CI) -3.4–0.3; nonsignificant), but was much larger by the unblinded examiner (-3.0 cm, 95% CI -4.9– -1.1), proving a subjective element to the examiners' estimation of visible nasal obstruction. From the

other outcomes, only self-reported CPAP adherence improved significantly. It has to be mentioned that the study by POWELL *et al.* [61] was designed as a pilot study and was therefore underpowered to assess the value of radiofrequency tissue ablation treatment to the nose in patients with nasal obstruction and OSA.

Given the lack of further randomised controlled trials, the beneficial effect of surgical interventions to the nose on snoring and OSA needs to be further defined.

Nasal dilators

The nasal airway is narrowest at the nasal valve region, which usually contributes most to total NR in a normal nasal airway. Assuming the nose has a major role in the pathogenesis of SDB, any treatment that increases nasal patency in this region may improve snoring or OSA. There are two commercially available products that may effectively dilate the nasal airway in the region of the nasal valves. One is the Breathe Right® (CNS Inc., Bloomington, MN, USA), an elastic plastic nasal strip, which is applied externally and pulls the nares open. The other product is Nozovent® (Prevancure AB, Västra Frölunda, Sweden), which consists of an elastic plastic bar with two tabs at each end that are fitted inside the nostrils and dilate the nasal valves by pushing outwards.

Five studies were identified with adequate study design, three using Breathe Right® and two using Nozovent®; a summary of each is given in table 2 [62–66]. BAHAMMAM *et al.* [62] found that patients with upper airway resistance syndrome spent a lower percentage of time during the sleep study in desaturation ($>2\%$ below mean awake arterial oxygen saturation (S_{a,O_2})) using Breathe Right® ($9.1\pm1.3\%$ on treatment *versus* 12.2 ± 2.2 % on placebo), and treatment reduced stage 1 sleep. In contrast, no change in sleep architecture or oxygen desaturation index was found using Breathe Right® treatment in the two other studies [63, 64]. Subjective improvement of snoring was only reported in the study by PEVERNAGIE *et al.* [63]. In none of these three studies did Breathe Right® treatment improve OSA severity or symptoms. It can be concluded that improving nasal patency by external nasal dilators has some beneficial effects on subjective snoring, and possibly on sleep architecture and desaturation time, but does not decrease the frequency of apnoeas or improve daytime sleepiness. Meta-analysis was not possible because of a considerable variability in outcome variables between these studies.

In a nonblinded study, HÖIJER *et al.* [66] increased nasal airflow by using Nozovent® in patients with mostly mild OSA; mean apnoea index (AI) decreased from 18 (range 1.8–60) without the nasal dilator, to 6.4 (1.3–15) with the nasal dilator. Thus, the mean decrease of the AI was 47% (13–83%; p=0.008). The average minimum overnight S_{a,O_2} with and without the nasal dilator was 84% (76–88%) and 78% (68–89%; p=0.03), respectively. There are no data reported on sleep architecture and, interestingly, sleepiness did not improve with nasal dilator therapy despite the apparent effect on AI. HÖIJER *et al.* [66] also found a significant decrease in objective snoring (number of episodes with level of the noise >55 or >60 dB) when the dilator was used.

SCHÖNHOFER *et al.* [65] used Nozovent® in order to improve nasal patency during CPAP titration nights in patients with proven OSA. With Nozovent®, the median CPAP pressure was reduced from 0.84 to 0.78 kPa, but no difference was found in respiratory disturbance index, AI or arterial oxygen saturation measured by pulse oximetry. Nasal patency was not measured either objectively or subjectively in the study by SCHÖNHOFER *et al.* [65].

TABLE 2	Randomised controlled trials of nasal dilator treatment in sleep-disordered breathing			
First author [Ref.]	**Number and characteristics of patients**	**Study design, intervention**	**Outcomes**	**Comments**
BAHAMMAM [62]	18, snorers with UARS, mean AHI 8.9, no information on nasal complaints	Crossover, Breathe Right® *versus* placebo strips	Improved desaturation time and sleep architecture, no difference in AHI, arousal index, MSLT	Nasal dilation resulted in an increase of nasal cross-sectional area. No data on snoring
PEVERNAGIE [63]	12, snorers, mean AHI 6, chronic rhinitis and nasal obstruction	Crossover, Breathe Right® *versus* placebo strips	Decrease in snoring events, no difference in AHI, arousal index and sleep architecture	Nasal dilation resulted in a nearly significant reduction of NR
DJUPESLAND [64]	18, heavy snorers, median AHI 9.3, nocturnal nasal obstruction	Crossover, Breathe Right® *versus* placebo strips	No difference in ODI, snoring time and sleep architecture. Increase in AHI with active dilator	Nasal dilation resulted in an increase in total nasal cross-sectional area and volume
SCHÖNHOFER [65]	38, OSA patients undergoing CPAP titration, AI 17.1, no information on nasal complaints	Crossover, Nozovent® *versus* no intervention	Decrease of CPAP pressure, no difference in AI, S_{p,O_2}	Nasal dilation was not controlled by objective or subjective measurement
HÖIJER [66]	10, mainly mild OSA, mean AI 18, no nasal complaints	Crossover, Nozovent® *versus* no intervention	Decrease in snoring events, AI, and minimal S_{p,O_2}. No change in daytime hypersomnolence	Nasal dilation resulted in an increase in nasal airflow

UARS: upper airway resistance syndrome; AHI: apnoea/hypopnoea index; MSLT: multiple sleep latency testing; NR: nasal resistance; ODI: oxygen desaturation index; OSA: obstructive sleep apnoea; CPAP: continuous positive airway pressure; AI: apnoea index; S_{p,O_2}: arterial oxygen saturation measured by pulse oximetry.

Taken together, there is evidence from these five studies that nasal dilators improve nasal patency, which results in a reduction of snoring events, but they seem to have an only minor effect on OSA severity and symptoms.

Topical drugs

Excessive mucosal swelling of the nasal turbinates, which is the predominant cause of high NR in patients with chronic rhinitis, can be addressed by topical application of sympatho-mimetic vasoconstrictors (*e.g.* xylometazoline) or topical nasal steroids. Only one study was identified with appropriate design evaluating the effect of topical nasal steroids on sleepiness, snoring and OSA in adults [67]. In the only publication available using objective outcomes from sleep studies, KIELY *et al.* [67] investigated the effect of 4 weeks of intranasal fluticasone treatment on 23 patients: 13 apnoeic snorers with mild-to-moderate OSA (mean AHI 26.5) and 10 nonapnoeic snorers. They reported an apparently significantly lower AHI (median -6.5, 95% CI -29.5–1.8) in the 13 patients with OSA after intranasal corticosteroid treatment *versus* placebo, although their reported CIs crossed 0. Nasal steroid treatment did not improve subjective sleep quality, sleep architecture or oxygen saturation derivatives in either OSA patients or in the nonapnoeic snorers. In the same study [67], fluticasone did not improve objectively measured and sub-jectively bed-partner-reported snoring, despite topical steroids significantly lowering NR. The data suggest that nasal obstruction due to allergic rhinitis contributes to the severity of OSA, and treatment with topical steroids might be of some small benefit in patients with mild-to-moderate OSA.

Two studies were identified investigating the effect of nasal decongestants on SDB [68, 69]; in one of them the decongestant was combined with a nasal dilator [68] and, in the other, with intranasally applied tubes [69], respectively. In both studies, sleep architecture improved with therapy to some extent, but a positive effect on AHI (average change -12, 95% CI -3- -22) was found only in the trial performed by MCLEAN *et al.* [68]. Subjective daytime sleepiness did not improve in either of the two studies. Data on snoring were not available from these studies. Further details on these studies investigating the effects of topical nasal drug therapy on SDB are given in table 3.

It must be noted that in the reviewed studies, NR has been determined by single assessments in the evening or morning only, and may not reflect the physiological conditions during an entire night's sleep.

Data from these studies suggest that pharmacologically induced improvement of nasal patency in patients with OSA and chronic nasal obstruction has some beneficial effects on the frequency of apnoeas and on sleep architecture. Data on objectively assessed snoring and sleepiness are mainly lacking. Since the observed reduction in OSA severity was only modest, it might not be in a range of clinical significance.

THE AUTHORS' CLINICAL APPROACH

Since the available evidence on the value of treating chronic nasal congestion in patients with OSA and snoring is limited, the current authors' clinical approach is outlined briefly in the following paragraph. Nasal congestion is a common complaint in patients with OSA and snoring, and can interfere with, or result from, CPAP usage; therefore, the history behind this symptom should be taken systematically. This might include questions concerning symptoms of allergic rhinitis, nonallergic rhinitis and other forms of chronic nasal obstruction, previous injuries or surgery, and nasal discharge, as well as the ability to breathe through the nose in the sitting and supine positions.

Inspection of the shape of the nose, checking the position of the nasal septum, and anterior rhinoscopy are sufficient to assess most anatomical causes of nasal obstruction. Objective mea-surement of NR by anterior or posterior rhinomanometry [70] on the awake patient, or during sleep with recently developed unobtrusive techniques [16, 71, 72], can be useful in selected patients, *e.g.* to monitor pharmacological treatment efficacy or to assess the outcome of surgical interventions.

In patients with allergic rhinitis, topical intranasal steroids (*e.g.* fluticasone and beclomethasone) should be prescribed as first-line therapy in addition to avoidance of allergens, and medication should be continued for $\geqslant$2–4 weeks before

TABLE 3	Randomised controlled trials of topical nasal drug treatment in sleep-disordered breathing			
First author [Ref.]	Number and characteristics of patients	Study design, intervention	Outcomes	Comments
KIELY [67]	23, 10 snorers and 13 OSA patients, median AHI of OSA patients 26.5, chronic allergic rhinitis	Crossover, double-blind, intranasal fluticasone *versus* placebo (saline)	AHI and subjective NR decreased No difference in sleep architecture, snoring and S_{p,O_2}	Fluticasone decreased NR
MCLEAN [68]	10, moderate-severe OSA, chronic nasal obstruction	Crossover, single-blind, intranasal oxymetazoline and Breathe Right® *versus* placebo strips and sodium chloride	AHI and sleep architecture improved, and mouth breathing decreased No change in subjective sleepiness	Oxymetazoline and Breathe Right® reduced NR No data on snoring
KERR [69]	10, moderate-to severe OSA, six out of 10 with chronic nasal obstruction	Crossover, single-blind, intranasal oxymetazalone and vestibular stents *versus* placebo (saline)	Arousal index (and sleep architecture in patients with nasal obstruction) improved No change of AHI and S_{p,O_2} No change in subjective sleepiness	Oxymetazalone and vestibular stents reduced NR No data on snoring

OSA: obstructive sleep apnoea; AHI: apnoea/hypopnoea index; NR: nasal resistance; S_{p,O_2}: arterial oxygen saturation measured by pulse oximetry.

assessing treatment efficacy [73]. An oral antihistamine or oral decongestant can be effective in treating symptoms of allergic rhinitis, but these medications may be associated with significant side-effects. Topical decongestant nasal sprays (*e.g.* oxymetazoline) can also reduce nasal congestion, but treatment duration is limited, as therapy lasting >5 days can lead to rebound nasal congestion [74].

In the case of nonallergic rhinitis, treatment of the underlying disease and avoidance of irritants is the mainstay of therapy. Patients with nonallergic rhinitis might also benefit from treatment with intranasal steroids or a locally applied antihistamine (*e.g.* azelastine), and if rhinorrhoea is a prominent symptom, ipratropium nasal spray is appropriate [73–76].

It must also be mentioned that about one-quarter of all patients with rhinitis have a mixed allergic–nonallergic variant [77], and symptoms of nonallergic rhinitis might be indistinguishable from those occurring in allergic rhinitis [24]. Therefore, from the current authors' point of view, a trial of intranasal steroids can be performed in all patients with characteristic symptoms of chronic rhinitis without specific IgE or skin testing. Whether these patients benefit from long-term topical steroid treatment, and whether this approach is cost-effective, remains to be proven.

Patients with chronic nasal obstruction often struggle to tolerate nasal CPAP. Humidification of inhaled air, correcting potential leakage of the nasal mask and a trial of a full-face mask are initial steps to dealing with this problem. Patients who still cannot tolerate CPAP, or have obvious nasal polyps, severe turbinate hypertrophy or a distinctive abnormality of the nasal anatomy should be referred to an ENT specialist for a surgical opinion.

PRIORITIES FOR FUTURE RESEARCH
Although some progress has been made in recent years in exploring the relationship between snoring, OSA and chronic nasal congestion, substantial areas of uncertainty remain. Many of the studies investigating this topic lack an appropriate sample size, carefully defined patient populations, objectively measured snoring variables and suitable techniques for the assessment of objective NR. Therefore, it is not surprising that conflicting findings have been reported in some of the recently published studies. From the current authors' point of view, objective measurement of snoring, indices of SDB and NR are essential outcome variables in a randomised controlled trial investigating the role of the nose, since there is poor agreement between subjective assessment with questionnaires or visual analogue scales and objectively measured data during a sleep study [22, 78].

Possible strategies for future research could include large-scale randomised, controlled studies in carefully defined patient populations with OSA or snoring, and chronic nasal congestion, allowing identification of patients who benefit from either surgery or pharmacological treatment of the nose in the long term. In addition to the end-points of objectively measured snoring, indices of SDB and daytime sleepiness, such randomised controlled studies should perhaps include measurements of nocturnal NR due to the considerable variability of NR over time.

CONCLUSIONS
Data from epidemiological and physiological studies suggest that nasal congestion contributes to the pathogenesis of snoring and obstructive sleep apnoea. Randomised controlled trials that used nasal dilators, topically applied steroids and nasal decongestants to reduce nasal resistance are not sufficiently robust and showed only minor improvement of obstructive sleep apnoea symptoms and severity. Snoring was positively influenced by nasal dilators. Architecture and quality of sleep could be improved by treating nasal congestion, but clinical relevance remains to be proven. Although nasal surgery may be helpful in patients who are unable to tolerate continuous positive airway pressure because of nasal obstruction, this has never been proven in randomised, controlled trials. As there are no data from adequately designed and sized trials investigating the effect of nasal surgery on obstructive sleep apnoea or snoring, surgical intervention should be used within the context of a randomised controlled trial, or reserved for highly selected cases. Taken together, the current evidence suggests that the nose may not play a significant role in the pathogenesis of obstructive sleep apnoea, but it seems to be of some relevance in the origin of snoring, as might have been predicted based on the Starling resistor model of the pharynx discussed earlier.

REFERENCES
1 Stradling JR. Obstructive sleep apnoea: definitions, epidemiology, and natural history. *Thorax* 1995; 50: 683–689.
2 Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S. The occurrence of sleep-disordered breathing among middle-aged adults. *N Engl J Med* 1993; 328: 1230–1235.
3 Stradling JR, Crosby JH. Predictors and prevalence of obstructive sleep apnoea and snoring in 1001 middle-aged men. *Thorax* 1991; 46: 85–90.
4 Liistro G, Rombaux P, Belge C, Dury M, Aubert G, Rodenstein DO. High Mallampati score and nasal obstruction are associated risk factors for obstructive sleep apnoea. *Eur Respir J* 2003; 21: 248–252.
5 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.
6 Lugaresi E, Cirignotta F, Coccagna G, Piana C. Some epidemiological data on snoring and cardiocirculatory disturbances. *Sleep* 1980; 3: 221–224.
7 Counter P, Wilson JA. The management of simple snoring. *Sleep Med Rev* 2004; 8: 433–441.
8 Lim PV, Curry AR. The role of history, Epworth Sleepiness Scale score and body mass index in identifying non-apnoeic snorers. *Clin Otolaryngol Allied Sci* 2000; 25: 244–248.
9 Gavriely N, Jensen O. Theory and measurement of snores. *J Appl Physiol* 1993; 74: 2828–2837.
10 Schwartz AR, Smith PL, Wise RA, Bankman I, Permutt S. Effect of positive nasal pressure on upper airway pressure–flow relationships. *J Appl Physiol* 1989; 66: 1626–1634.
11 Schwartz AR, Smith PL, Wise RA, Gold AR, Permutt S. Induction of upper airway occlusion in sleeping individuals with subatmospheric nasal pressure. *J Appl Physiol* 1988; 64: 535–542.

12 Smith PL, Wise RA, Gold AR, Schwartz AR, Permutt S. Upper airway pressure–flow relationships in obstructive sleep apnea. *J Appl Physiol* 1988; 64: 789–795.

13 Young T, Finn L, Kim H. Nasal obstruction as a risk factor for sleep-disordered breathing. The University of Wisconsin Sleep and Respiratory Research Group. *J Allergy Clin Immunol* 1997; 99: S757–S762.

14 Young T, Finn L, Palta M. Chronic nasal congestion at night is a risk factor for snoring in a population-based cohort study. *Arch Intern Med* 2001; 161: 1514–1519.

15 Ferris B, Mead J, Opie L. Partitioning of respiratory flow resistance in man. *J Appl Physiol* 1964; 19: 653–658.

16 Kohler M, Thurnheer R, Bloch KE. Side-selective, unobtrusive monitoring of nasal airflow and conductance. *J Appl Physiol* 2006; 101: 1760–1765.

17 Hasegawa M, Kern EB. Variations in nasal resistance in man: a rhinomanometric study of the nasal cycle in 50 human subjects. *Rhinology* 1978; 16: 19–29.

18 Lang C, Grutzenmacher S, Mlynski B, Plontke S, Mlynski G. Investigating the nasal cycle using endoscopy, rhinoresistometry, and acoustic rhinometry. *Laryngoscope* 2003; 113: 284–289.

19 Widdicombe JG. The physiology of the nose. *Clin Chest Med* 1986; 7: 159–170.

20 Haight JJ, Cole P. Reciprocating nasal airflow resistances. *Acta Otolaryngol* 1984; 97: 93–98.

21 Wilde AD, Jones AS. The nasal response to axillary pressure. *Clin Otolaryngol Allied Sci* 1996; 21: 442–444.

22 Kohler M, Clarenbach CF, Thurnheer R, Bloch KE. Perception of nocturnal nasal obstruction in chronic rhinitis. *Eur Respir J* 2005; 26: Suppl. 49, 565.

23 Cole P. Stability of nasal airflow resistance. *Clin Otolaryngol Allied Sci* 1989; 14: 177–182.

24 Settipane RA, Charnock DR. Epidemiology of rhinitis: allergic and nonallergic. *Clin Allergy Immunol* 2007; 19: 23–34.

25 Settipane GA, Klein DE. Non allergic rhinitis: demography of eosinophils in nasal smear, blood total eosinophil counts and IgE levels. *N Engl Reg Allergy Proc* 1985; 6: 363–366.

26 Leynaert B, Bousquet J, Neukirch C, Liard R, Neukirch F. Perennial rhinitis: an independent risk factor for asthma in nonatopic subjects. Results from the European Community Respiratory Health Survey. *J Allergy Clin Immunol* 1999; 104: 301–304.

27 Engberg RN. Perennial nonallergic rhinitis: a retrospective review. *Ann Allergy Asthma Immunol* 1989; 63: 513–516.

28 Skone B. Allergic rhinitis: definition, epidemiology, pathophysiology, detection and diagnosis. *J Allergy Clin Immunol* 2001; 108: 52–58.

29 Fitzpatrick MF, McLean H, Urton AM, Tan A, O'Donnell D, Driver HS. Effect of nasal or oral breathing route on upper airway resistance during sleep. *Eur Respir J* 2003; 22: 827–832.

30 Suratt PM, Turner BL, Wilhoit SC. Effect of intranasal obstruction on breathing during sleep. *Chest* 1986; 90: 324–329.

31 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.

32 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.

33 McNicholas WT, Coffey M, Boyle T. Effects of nasal airflow on breathing during sleep in normal humans. *Am Rev Respir Dis* 1993; 147: 620–623.

34 Douglas NJ, White DP, Weil JV, Zwillich CW. Effect of breathing route on ventilation and ventilatory drive. *Respir Physiol* 1983; 51: 209–218.

35 Basner RC, Simon PM, Schwartzstein RM, Weinberger SE, Weiss JW. Breathing route influences upper airway muscle activity in awake normal adults. *J Appl Physiol* 1989; 66: 1766–1771.

36 Shi YX, Seto-Poon M, Wheatley JR. Breathing route dependence of upper airway muscle activity during hyperpnea. *J Appl Physiol* 1998; 84: 1701–1706.

37 Williams JS, Janssen PL, Fuller DD, Fregosi RF. Influence of posture and breathing route on neural drive to upper airway dilator muscles during exercise. *J Appl Physiol* 2000; 89: 590–598.

38 Morris LG, Burschtin O, Lebowitz RA, Jacobs JB, Lee KC. Nasal obstruction and sleep-disordered breathing: a study using acoustic rhinometry. *Am J Rhinol* 2005; 19: 33–39.

39 Leger D, Annesi-Maesano I, Carat F, *et al.* Allergic rhinitis and its consequences on quality of sleep: an unexplored area. *Arch Intern Med* 2006; 18: 1744–1748.

40 Canova CR, Downs SH, Koblauch A, Andersson M, Tamm M, Leuppi JD. Increased prevalence of perennial allergic rhinitis in patients with obstructive sleep apnea. *Respiration* 2004; 71: 138–143.

41 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.

42 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.

43 Craig TJ, Mende C, Hughes K, Kakumanu S, Lehman EB, Chinchilli V. The effect of topical nasal fluticasone on objective sleep testing and the symptoms of rhinitis, sleep, and daytime somnolence in perennial allergic rhinitis. *Allergy Asthma Proc* 2003; 24: 53–58.

44 Hughes K, Glass C, Ripchinski M, *et al.* Efficacy of the topical nasal steroid budesonide on improving sleep and daytime somnolence in patients with perennial allergic rhinitis. *Allergy* 2003; 58: 380–385.

45 Craig TJ, Hanks CD, Fisher LH. How do topical nasal corticosteroids improve sleep and daytime somnolence in allergic rhinitis. *J Allergy Clin Immunol* 2005; 116: 1264–1266.

46 Virkkula P, Bachour A, Hytonen M, Malmberg H, Salmi T, Maasilta P. Patient- and bed partner-reported symptoms, smoking, and nasal resistance in sleep-disordered breathing. *Chest* 2005; 128: 2176–2182.

47 Metes A, Ohki M, Cole P, Haight JS, Hoffstein V. Snoring, apnea and nasal resistance in men and women. *J Otolaryngol* 1991; 20: 57–61.

48 Virkkula P, Bachour A, Hytonen M, *et al.* Snoring is not relieved by nasal surgery despite improvement in nasal resistance. *Chest* 2006; 129: 81–87.

49 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: 1261–1265.

50 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.

51 Dayal V, Phillipson EA. Nasal surgery in the management of sleep apnea. *Ann Oto Rhinol Laryngol* 1985; 94: 550–554.

52 Fairbanks DN. Effect of nasal surgery on snoring. *South Med J* 1985; 78: 268–270.

53 Friedman M, Tanyeri H, Lim JW, Landsberg R, Vaidyanathan K, Caldarelli D. Effect of improved nasal breathing on obstructive sleep apnea. *Otolaryngol Head Neck Surg* 2000; 122: 71–74.

54 Guilleminault C, Korobkin R, Winkle R. A review of 50 children with obstructive sleep apnea syndrome. *Lung* 1981; 159: 275–287.

55 Swift AC. Upper airway obstruction, sleep disturbances and adenotonsillectomy in children. *J Laryngol Otol* 1988; 102: 419–422.

56 Brouillette RT, Fernbach SK, Hunt CE. Obstructive sleep apnea in infants and children. *J Pediatr* 1982; 100: 31–39.

57 Stradling JR, Thomas G, Warley ARH, Williams P, Freeland A. Effect of adenotonsillectomy on nocturnal hypoxaemia, sleep disturbance, and symptoms in snoring children. *Lancet* 1990; 335: 249–253.

58 Mitchell RB, Kelly J. Outcome of adenotonsillectomy for severe obstructive sleep apnea in children. *Int J Pediatr Otorhinolaryngol* 2004; 68: 1375–1379.

59 Guilleminault C, Huang YS, Glamann C, Li K, Chan A. Adenotonsillectomy and obstructive sleep apnea in children: a prospective survey. *Otolaryngol Head Neck Surg* 2007; 136: 169–175.

60 Lim J, McKean M. Adenotonsillectomy for obstructive sleep apnoea in children. *Cochrane Database Syst Rev* 2003; 1: CD003136.

61 Powell NB, Zonato AI, Weaver EM, *et al.* Radiofrequency treatment of turbinate hypertrophy in subjects using continuous positive airway pressure: a randomized, double-blind, placebo-controlled clinical pilot trial. *Laryngoscope* 2001; 111: 1783–1790.

62 Bahammam AS, Tate R, Manfreda J, Kryger MH. Upper airway resistance syndrome: effect of nasal dilation, sleep stage, and sleep position. *Sleep* 1999; 22: 592–598.

63 Pevernagie D, Hamans E, Van Cauwenberge P, Pauwels R. External nasal dilation reduces snoring in chronic rhinitis patients: a randomized controlled trial. *Eur Respir J* 2000; 15: 996–1000.

64 Djupesland PG, Skatvedt O, Borgersen AK. Dichotomous physiological effects of nocturnal external nasal dilation in heavy snorers: the answer to a rhinologic controversy? *Am J Rhinol* 2001; 15: 95–103.

65 Schönhofer B, Kerl J, Suchi S, Köhler D, Franklin KA. Effect of nasal valve dilation on effective CPAP level in obstructive sleep apnea. *Respir Med* 2003; 97: 1001–1005.

66 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.

67 Kiely JL, Nolan P, McNicholas WT. Intranasal corticosteroid therapy for obstructive sleep apnoea in patients with co-existing rhinitis. *Thorax* 2004; 59: 50–55.

68 McLean HA, Urton AM, Driver HS, *et al.* Effect of treating severe nasal obstruction on the severity of obstructive sleep apnoea. *Eur Respir J* 2005; 25: 521–527.

69 Kerr P, Millar T, Buckle P, Kryger M. The importance of nasal resistance in obstructive sleep apnea syndrome. *J Otolaryngol* 1992; 21: 189–195.

70 Cole P, Ayiomanimitis A, Ohki M. Anterior and posterior rhinomanometry. *Rhinology* 1989; 27: 257–262.

71 Kohler M, Thurnheer R, Bloch KE. Non-invasive, side-selective nasal airflow monitoring. *Physiol Meas* 2005; 26: 69–82.

72 Thurnheer R, Bloch KE. Monitoring nasal conductance by bilateral nasal cannula pressure transducers. *Physiol Meas* 2004; 25: 577–584.

73 Nassef M, Shapiro G, Casale TB. Identifying and managing rhinitis and its subtypes: allergic and nonallergic components – a consensus report and materials from the Respiratory and Allergic Disease Foundation. *Curr Med Res Opin* 2006; 22: 2541–2548.

74 Dykewicz MS. Clinical approach to diagnosis and treatment of nonallergic rhinitis. *Clin Allergy Immunol* 2007; 19: 335–350.

75 Sjogren I, Jonsson L, Koling A, Jansson C, Osterman K, Hakansson B. The effect of ipratropium bromide on nasal hypersecretion induced by methacholine in patients with vasomotor rhinitis. A double-blind, cross-over, placebo-controlled and randomized dose-response study. *Acta Otolaryngol* 1988; 106: 453–459.

76 Bronsky EA, Druce H, Findlay SR, *et al.* A clinical trial of ipratropium bromide nasal spray in patients with perennial nonallergic rhinitis. *J Allergy Clin Immunol* 1995; 95: 1117–1122.

77 Settipane RA. Demographics and epidemiology of allergic and nonallergic rhinitis. *Allergy Asthma Proc* 2001; 22: 185–189.

78 Stradling JR, Barbour C, Glennon J, Langford BA, Crosby JH. Prevalence of sleepiness and its relation to autonomic evidence of arousals and increased inspiratory effort in a community based population of men and women. *J Sleep Res* 2000; 9: 381–388.