

## The Effects of Nasal Dilation on Snoring and Obstructive Sleep Apnea

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• The effects of nasal valve dilation on snoring and obstructed breathing were studied in 11 patients with habitual snoring and/or obstructive sleep apnea. The anterior part of the nose, the valve region, was dilated by means of a plastic device. Ten patients underwent polysomnographic investigation including pulse oximetry and measurement of snoring noise with and without the nasal dilator in a randomized manner. Snoring, nocturnal arousals, and daytime hypersomnolence were rated by the patient and partner on a questionnaire before and after a 10-day treatment period with the dilator. The nasal airflow, as assessed by rhinomanometry when awake in the sitting position, increased by 18% (range, 5.5% to 45%) when the nasal dilator was used. The frequency and severity of obstructed breathing decreased significantly with the nasal dilator. The apnea index with and without the nasal dilator was 6.4 (range, 1.3 to 15) and 18 (range, 1.8 to 60), respectively. The mean decrease of the apnea index was 47%. The overnight minimum arterial oxygen saturation (with and without the nasal dilator was 84% (range, 76% to 88%) and 78% (range, 68% to 89%), respectively. There was a substantial decrease in snoring noise (number of epochs with Leq values, equal energy level, above 55 or 60 dB) with the dilator in all patients who presented with snoring noise above these levels during the control night. No subjective effects on arousal frequency or daytime hypersomnolence were reported. Four of 11 patients were positive to continue using the nasal dilator.

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The exact pathogenic mechanism behind snoring and obstructive sleep apnea (OSA) remains to be clarified. However, several conditions that result in a pathologically narrow pharyngeal airway such as adenotonsillar enlargement<sup>1</sup> and pharyngeal tumors<sup>2</sup> may induce sleep-related impaired breathing and OSA. Similarly, impairment of nasal breathing may result in OSA.<sup>3,4</sup> Indeed,

a reduced nasal airflow during sleep in OSA may be of particular interest considering that the nasal valve region is the narrowest part of the normal upper airway. This region alone constitutes more than half the total nasal resistance.<sup>5,6</sup> Moreover, a decrease in upper airway sensory inflow, eg, as a result of decreased nasal airflow, may in itself be a vital cause of decreased upper airway dilator muscle activity, resulting in OSA.<sup>7</sup>

A nasal dilator that increases the cross-sectional area of the nasal valve region has been shown to improve nasal airflow in healthy volunteers significantly.<sup>8</sup> In addition, this device was shown to significantly reduce subjectively assessed snoring behavior in a group of 10 patients.<sup>9</sup>

This study was undertaken to objectively evaluate the effect of nasal valve region dilation on snoring and on the intensity of obstructed breathing during sleep in patients with habitual snoring and/or OSA.

### MATERIALS AND METHODS

Eleven consecutive patients (seven men and four women, with a mean age of 47 years [range, 32 to 65 years old]) were included in the study. All patients had been referred to the Department of Otorhinolaryngology, University of Göteborg (Sweden) due to a history of habitual snoring and/or witnessed apneas during sleep. The average body weight was 80.6 kg (range, 66.5 to 103.5 kg). No patient reported difficulty in breathing through the nose in the standing or supine position when awake. Tendency to collapse the nasal valve region during inspiration, known nasal allergy, or ongoing rhinitis were criteria for exclusion from the study. All patients except one had normal conditions in the nasal cavity and epipharynx on rhinoscopy and epipharyngoscopy. A minor deviation of the nasal septum, not influencing the nasal airflow, was found in the remaining case. The use of alcohol or hypnotics was not permitted during the study period. One patient who was unable to tolerate the nasal dilator during sleep was excluded from the study at an early stage, leaving 10 patients who completed the investigation. The patients included signed a consent form before the investigations were performed.

The nasal dilator used in this study was a plastic device consisting of two end tabs with a connecting bar (Nozovent) (Fig 1). The dilator is inserted into the nares and fitted to exert a continuous dilating force on the nasal valves by its elasticity.

The patients were instructed to use the nasal dilator for 10 nights at home. The investigation in the sleep laboratory involved two overnight recordings in randomized order with or without the nasal dilator applied.

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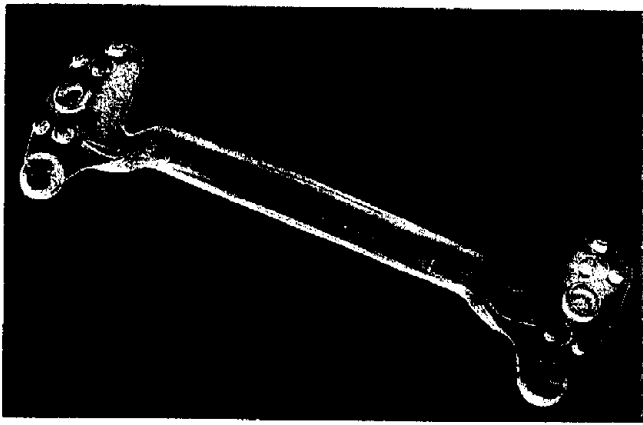


Fig 1.—The nasal dilator used in this study.

### Rhinomanometry

When awake the nasal airflow was measured in the sitting position, by active posterior rhinomanometry (Rhinoma 12 s) with and without the nasal dilator. The mean airflow (liter per second [L/s]) at a pressure drop of 150 Pa was calculated from 10 inspirations during tidal breathing (normal value,  $0.67 \pm 0.16$  L/s).<sup>10</sup>

### Polysomnography

All sleep investigations were conducted in the hospital sleep laboratory using standard polysomnographic techniques, including electroencephalography, electrooculography, and submental electromyography (Oxford Medilog System 9000, Oxford, England). Nasal and oral airflow were measured by thermistors and thoracic respiratory movements by changes in thoracic impedance. Arterial oxygen saturation ( $\text{SaO}_2$ ) and heart rate were continuously monitored by a pulse oximeter and a finger probe (Nellcor N-100). An apnea was defined as a cessation of airflow at the nose and mouth for more than 10 seconds and was classified as central if no paradoxical thoracic motion occurred during the event. Apneas with concomitant thoracic motion were defined as obstructive. The apnea index (AI) was calculated as the mean number of apneas, accompanied by arterial desaturations ( $\geq 4\%$ ), per hour of actual sleep time as assessed by the polysomnogram. All sleep investigations were performed between 11 PM and 6 AM.

### Measurement of Snoring Noise

The snoring noise was measured with a sound level dosimeter (Model 700 dosimeter, Larson and Davis Laboratories, Pleasant Grove, Utah) with the microphone placed 50 cm above the head of the patient. The dosimeter measured the equal energy level of the noise ( $\text{Leq}$ ) during epochs of 4 seconds, ie, the  $\text{Leq}$  values were measured 900 times every hour. The background noise in the sleep laboratory (the sound level in the room during the night without a sleeping patient) was found to be constant at a level of 36 dB. The number of epochs (4 seconds) with  $\text{Leq}$  values above 55 and 60 dB, respectively, was determined. Levels higher than 65 dB were discharged, since few epochs with  $\text{Leq}$  values higher than that were obtained. All dosimeter data were analyzed by a computer (IBM compatible). The average sound level was calculated from data collected between midnight and 5 PM only to reduce artifacts. Such artifacts may be produced by the patient during the process of falling asleep or waking up.

### Questionnaire

The effects and tolerability of the nasal dilator were rated by the patients in a questionnaire. The self-estimated number of arousals during sleep and the degree of daytime hypersomnolence were assessed on visual analogue scales (scale, 0 to 10, indicating: none, frequent, none, worst possible). A separate visual analogue scale was used by the patient's partner to score the effect of the nasal dilator on the patient's snoring noise.

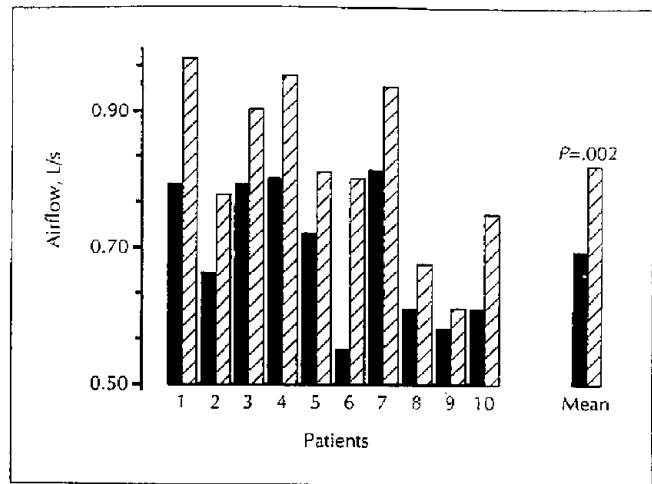


Fig 2.—Rhinomanometrically assessed nasal airflow at a pressure drop of 150 Pa (ordinate) in 10 patients, without (solid bars) and with (hatched bars) a nasal dilator. The individual values as well as means are shown.

### Statistics

A nonparametric permutation test for paired observations was used. *P* values of .05 or less in a two-tailed test were considered significant.

## RESULTS

### Rhinomanometry

All of the patients subjectively reported facilitated nose breathing with the nasal dilator. Nasal airflow increased in all of the patients ( $n=10$ ) with the nasal dilator from 0.70 L/s (range, 0.55 to 0.81 L/s) to 0.82 L/s (range, 0.61 to 0.98 L/s) (Fig 2). The average increase in airflow was 18% (range, 5.5% to 45%) ( $P=.002$ ).

### Polysomnography

Seven of the 10 patients investigated were found to have an AI of 5 or greater. With the nasal dilator, AI decreased in all but one patient (Fig 3). The two patients with the highest AI had the largest reduction of AI, 74% and 83%, respectively, with the nasal dilator. The mean AI with and without the nasal dilator was 6.4 (range, 1.3 to 15) and 18 (range, 1.8 to 60), respectively. Although AI increased slightly in one patient, the average decrease was 47% (range, 13% to 83%;  $P=.008$ ). Similarly, minimum overnight  $\text{SaO}_2$  increased in seven of the 10 patients with the nasal dilator (Fig 4). Again the most pronounced increase was seen in patients with the highest AI. Average minimum overnight  $\text{SaO}_2$  with and without the nasal dilator was 84% (range, 76% to 88%) and 78% (range, 68% to 89%), respectively ( $P=.03$ ). There was no significant correlation between the degree of improvement of nasal airflow in the sitting position when awake and AI or minimum  $\text{SaO}_2$  during sleep. No consistent changes in sleep architecture were seen with the use of the nasal dilator.

### Measurement of Snoring Noise

Four patients had less than two epochs with registered  $\text{Leq}$  values indicating a low degree of snoring noise (Table). In the remaining six patients there was a substantial decrease in snoring noise (number of epochs with  $\text{Leq}$  values above 55 or 60 dB) when the dilator was used. The reduction of the number of epochs with  $\text{Leq}$  values of more than 55 or 60 dB in the whole group was statistically

significant ( $P = .02$ ). There was no significant correlation between the recorded Leq values and either AI or minimum SaO<sub>2</sub> absolute values. Moreover, the change in noise level with the dilator did not relate to the change in AI or minimum SaO<sub>2</sub>.

### Questionnaire

No subjective effect on arousal frequency or daytime hypersomnolence was found. Four of eight sleeping partners (two patients had no partner) reported less disturbing snoring noise and four had no change during the trial period with the nasal dilator. Four of 11 patients wanted to continue using the nasal dilator.

### COMMENT

We have shown that mechanical dilation of the nasal airway during sleep by means of a nasal dilator decreased both the frequency and severity of obstructed breathing events in patients with OSA. The snoring noise was significantly reduced when the nose was dilated. The reduction of snoring noise, however, was not correlated to the reduction of AI or the increase of minimum overnight SaO<sub>2</sub>. This may be explained by the finding that the degree of snoring was not directly related to either the frequency or the severity of obstructive apneas.

Upper airway occlusion during inspiration and sleep may occur in patients with a narrow airway system due to, for example, adenotonsillar enlargement,<sup>1</sup> nasal septal deviation or polyps<sup>11</sup> as well as micrognathia,<sup>12</sup> and pharyngeal tumors.<sup>2</sup> Although OSA is most commonly seen in patients with no overt anatomic changes in the upper airway, these findings indicate that a decrease in upper airway airflow during sleep may be a potent generator of obstructed breathing. Specifically, these studies indicate that a decrease in nasal airflow may, in spite of the presence of undisturbed oral airflow, impair breathing during sleep.

The effects of nasal obstruction during sleep have been studied by several authors. During nasal obstruction, the time spent in deep sleep stages decreased in healthy volunteers, resulting in more time spent in stage I sleep and an increase in the number of arousals.<sup>3,4</sup> Nasal obstruction also resulted in more frequent events of obstructed breathing.<sup>3,4</sup> Indeed, although the mechanism is unclear, a decreased nasal airflow has been suggested to be an important determinant in the development of OSA.<sup>3</sup> Sensory inflow from the nasal airflow may function physiologically to overcome airway obstruction. Previous animal experiments<sup>13</sup> indicate that, in addition to chemoreceptor input to the central nervous system, upper airway sensory mechanisms are important contributors to the arousal response seen during nasal occlusion in both non-rapid eye movement and rapid eye movement sleep. A loss of this inflow may itself be a vital cause of failed upper airway dilator muscle activity and obstructive apnea.<sup>7</sup>

Our findings show a beneficial effect of the nasal dilation on nasal airflow in the sitting position when awake. This is in agreement with a previous study,<sup>8</sup> where nasal airflow was increased by 24% in a group of healthy volunteers. The magnitude of this increase in airflow is comparable with that induced by vasoconstricting nose drops. There was no correlation between the increase in nasal airflow in the awake state and the effect of nasal dilation on snoring behavior or apnea frequency. This is likely to

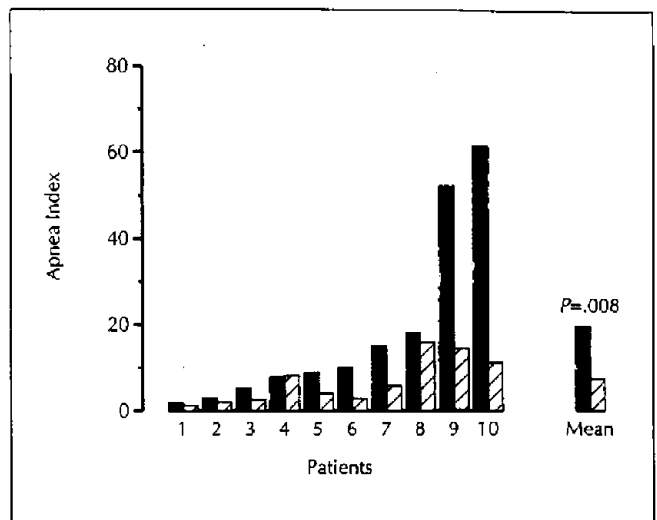


Fig 3.—Apnea index (mean number of apneas per hour of sleep) (ordinate) in 10 patients without (solid bars) and with (hatched bars) a nasal dilator. The individual values as well as means are shown.

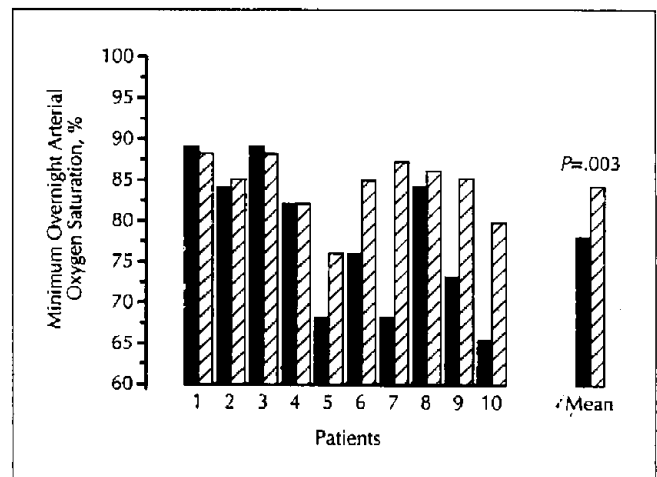


Fig 4.—Minimum overnight arterial oxygen saturation (ordinate) in 10 patients without (solid bars) and with (hatched bars) a nasal dilator. The individual values as well as means are shown.

Number of Epochs (4 Seconds) With Leq Values Above 55 and 60 dB; With and Without a Nasal Dilator\*

Patient	Leq > 55 dB		Leq > 60 dB	
	Without	With	Without	With
1	6	2	4	0
2	30	7	15	3
3	0	0	0	0
4	69	42	31	0
5	0	1	0	1
6	2	0	0	0
7	28	7	4	2
8	34	4	27	1
9	206	13	49	11
10	1	1	1	0

\*Leq indicates equal energy level of the noise.

be explained by the fact that rhinomanometry was performed in the sitting position in the awake state when airway geometry may have differed substantially from that in the supine position when sleeping.

The reduction in apnea frequency was closely correlated to the AI without the nasal dilator. This finding needs to be interpreted with some caution due to the relatively small number of patients studied. Still a substantial reduction in AI was found in all but one patient, indicating that the beneficial effect of nasal dilation may be generally found in patients with OSA independently of other factors of possible pathogenic importance such as body weight or upper airway airflow.

The subjective annoyance from snoring noise may not correlate to findings from objective acoustic measurements. A similar situation has been reported in investigations of traffic noise.<sup>14</sup> The complex pattern of road noise is usually expressed as the average noise level during a certain time period, eg, during one night (6 to 7 hours). Previous studies of annoyance from road traffic noise, however, have shown that the number of single noise peaks, eg, number of heavy vehicles is of major importance.<sup>14</sup> Thus, annoyance from snoring noise may be better reflected by the number of single snoring noise peaks. With the sound level meter available in this study, it was not possible to measure noise peaks during more than 2 hours. We therefore measured Leq during the shortest possible epochs collected from an adequate period of the night, ie, between midnight and 5 AM. The objective acoustic measurements used in this study correlated well with the subjective estimated degree of annoyance of the snoring noise. The beneficial effect of nasal dilation on snoring and obstructed breathing may be explained by an increase of the cross-sectional area of the nasal cavity and thereby a reduced nasal airway resistance. This may result in a decreased pharyngeal airway suction pressure during inspiration. As a consequence, the tendency to airway closure during inspiration is decreased in OSA patients.

Dilation of the nasal valve region significantly reduced AI, severity of oxygen desaturation, and snoring noise in

patients with habitual snoring and/or OSA. Although the findings in this study group indicate that nasal dilation may have a place in the treatment of habitual snoring and OSA, further studies are required to determine the long-term effects in a larger patient population.

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