

## Effect of Nasal Dilation on Snoring and Apneas During Different Stages of Sleep

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**Summary:** This study was designed to test the hypothesis that nasal dilation reduces snoring. To achieve this we performed nocturnal polysomnography, including measurement of snoring, in 15 patients without nasal pathology before and after insertion of a nasal dilator (NOZOVENT®). Snoring was quantified for each sleep stage by recording the number of snores per minute of sleep, number of snores per minute of snoring time and nocturnal sound intensities (maximum, average and minimum). We found that nasal dilation had no effect on the number of apneas, hypopneas or oxygen saturation. Snoring parameters were unaffected by NOZOVENT during stages I, II and REM sleep, but were all significantly reduced during slow wave sleep. We conclude that dilation of the anterior nares in patients without nasal pathology has a relatively weak effect on snoring, and routine use of nasal dilating appliances is not recommended for treatment of snoring. **Key words:** Snoring—Sleep architecture—NOZOVENT®.

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The importance of nasal breathing has been emphasized for many years (1,2). It is widely accepted that the inability to breathe through the nose may lead to snoring and sleep apnea. In recognition of the adverse social and health consequences of snoring and sleep apnea (3-6), there has been a great deal of interest in designing appliances that can dilate nasal passages, thus eliminating snoring and perhaps sleep apnea. However, their efficacy has not been established. Even if these appliances are shown to reduce snoring, it must be ascertained that this beneficial effect is not due to alteration in sleep architecture caused by the appliance. Consequently, the purpose of this study was to evaluate the effect of one such nasal dilator—NOZOVENT® (7) on snoring while controlling for sleep architecture.

### METHODS

We prospectively studied 15 patients referred to our sleep disorders clinic because of snoring and suspicion of sleep apnea. The patients did not have nasal pathology, as confirmed by an examination carried out by an otorhinolaryngologist. All patients verbally agreed to use NOZOVENT (Fig. 1) during the sleep study.

All patients had nocturnal polysomnography in-

cluding measurements of snoring. This was monitored using a microphone attached to a sound level meter (model SL 120, Pacer Industries, Toronto, Canada). The output of the sound level meter was displayed on the Grass polygraph recorder (model 78 E, Grass Instruments, Quincy, MA) and simultaneously sampled by an A/D converter at a rate of 5 Hz. The sound recording was calibrated using a sound chamber over the range of sound intensities from 40 to 100 dB. The microphone was positioned on the patient's forehead, just above the level of the nasion. With this set-up, normal breathing registered at <50 dB. Spikes in sound intensity >50 dB were always perceived as snores by the sleep technologist and were easily identified as such on the polygraph recording. A typical plot of sound intensity vs. sleep time generated by the computer from the sampled points is shown in Fig. 2. The software also generates a histogram of sound intensity and provides a summary of statistics, which includes the minimum, maximum and average sound intensity over the sampling period, as well as the number of snores per hour of sleep.

Polysomnograms were staged in 30-second epochs, according to the standard criteria. Apneas and hypopneas were identified and the number of these events per hour of sleep was reported as the apnea/hypopnea index (AHI). Snoring was quantified using 1-minute epochs for each stage of sleep. The following parameters were employed to describe snoring: snoring time

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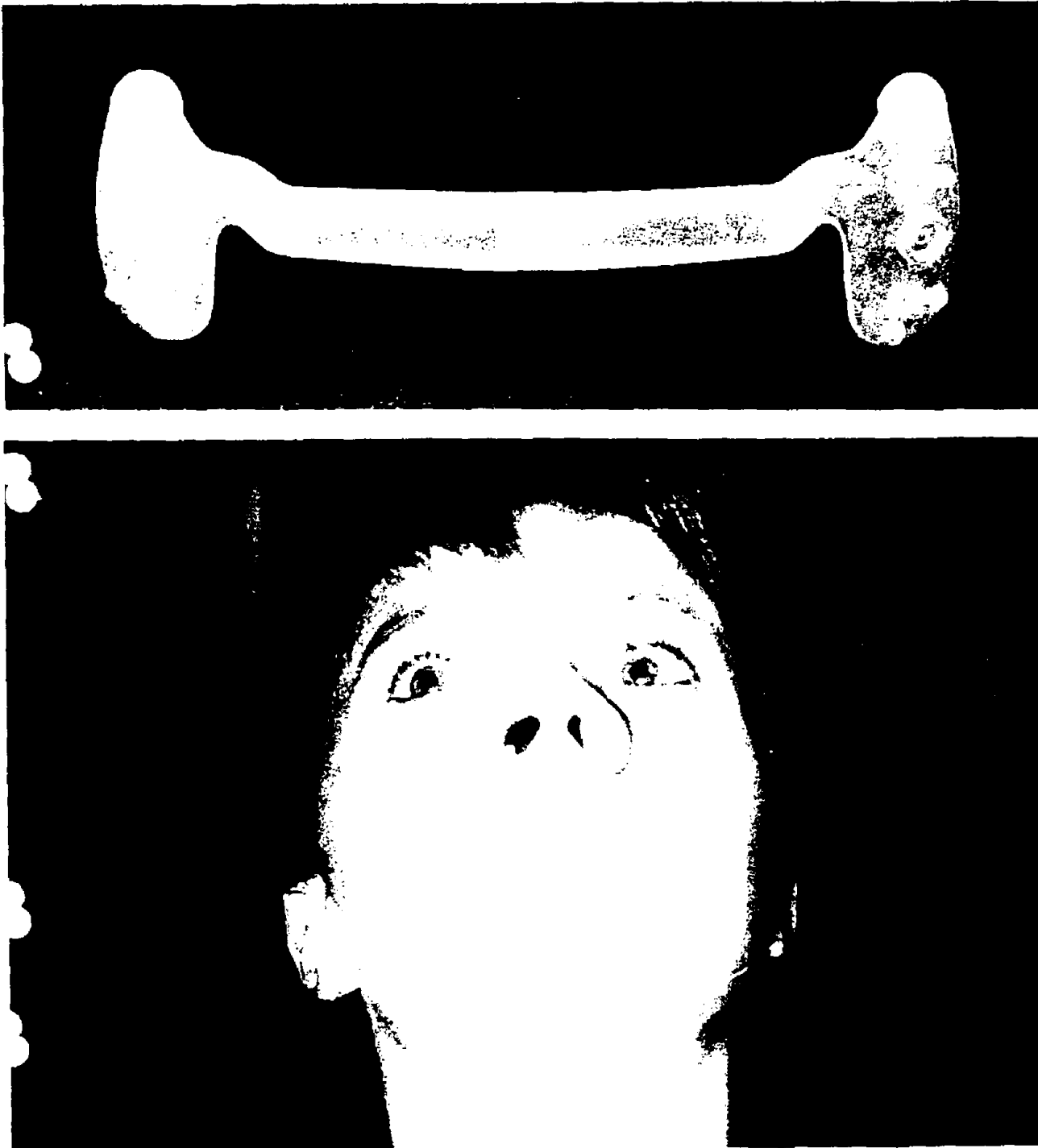


FIG. 1. The nasal dilator NOZOVENT (a) alone and (b) when introduced into the nose.

(defined as the sum of all epochs during which snoring was detected), snoring frequency (defined as the number of snores per minute of snoring time), snoring index (defined as the number of snores per minute of sleep time) and nocturnal sound intensity (lowest, highest and average—dBmin, dBmax, dBmean). Data collected during stage transitions were not included in the data analysis.

During the first half of the night (approximately 3 hours), patients slept without the nasal dilator. In the second half of the night, generally following a normal awakening, the nasal dilator was inserted, and patients slept with it until the termination of the sleep study.

The data were analyzed by comparing sleep stages, snoring and apneas pre- and postnasal dilator using paired *t* tests or Wilcoxon signed rank tests if the nor-

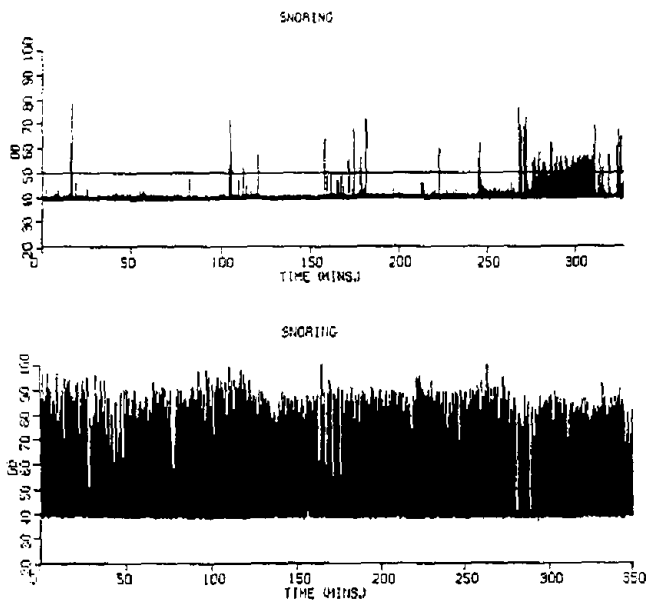


FIG. 2. A representative trace of snoring vs. sleep time for a light (a) and a heavy (b) snorer. Note spikes in sound intensity above 50 dB, representing snores. The bottom trace in each case represents the distribution of snoring over sleep time.

mality assumption was not satisfied. Analysis of variance (ANOVA) was used to compare the distribution of snoring across the sleep stages. All statistical analysis was carried out using the SAS Software, version 6.04 (The SAS Institute, Cary, NC). Univariate, means, and ANOVA procedures were used to test the data for normality, perform paired *t* tests and the analysis of variance, respectively.

## RESULTS

The patients studied (six females, nine males) ranged in age from 35–73 years (mean  $\pm$  SD =  $49 \pm 10$ ). They were significantly obese, with the body mass index ranging from 23–59 kg/m<sup>2</sup> (mean  $\pm$  SD =  $36 \pm 12$ ) and weight ranging from 72–175 kg (mean  $\pm$  SD =  $106 \pm 35$  kg). In seven patients with rhinomanometry performed during wakefulness, nasal airflow resistance dropped from  $1.62 \pm 1.24$  cm H<sub>2</sub>O/l/second to  $0.94 \pm 0.70$  cm H<sub>2</sub>O/l/second ( $p < 0.02$ ) with NOZOVENT.

In eight (four men, four women) out of 15 patients, the baseline AHI was  $>10$ . Following insertion of the nasal dilator, 11 patients had AHI  $>10$  (Table 1). The mean AHI, oxygenation indices and total sleep time were not significantly different prior to and after the application of nasal dilator.

Figure 3 shows the amount of sleep time spent in each stage (expressed as a percent of total sleep time), and the amount of time pre- and post-NOZOVENT. We note that the amount of sleep time spent in stages

TABLE 1. Summary of data in 15 patients before and after nasal dilation

	TST	AI	HI	Rna	O <sub>2</sub> sat		
					Lowest	Mean	
Patient 1	128.5	0.0	0.0		89	95	Pre
	111.5	13.5	10.2		75	95	Post
Patient 2	164.5	70.8	1.1	0.71	65	86	Pre
	142.5	62.3	0.4	0.47	69	88	Post
Patient 3	151.5	194.0	0.0	1.62	34	75	Pre
	118.5	118.0	2.0	0.67	36	75	Post
Patient 4	193.0	5.0	3.0	4.30	84	95	Pre
	180.5	3.0	2.0	2.46	85	96	Post
Patient 5	107.5	14.5	20.0		52	87	Pre
	180.5	6.3	48.0		35	84	Post
Patient 6	185.5	26.0	24.0		77	90	Pre
	134.5	9.0	12.0		72	89	Post
Patient 7	161.0	86.0	0.0		39	71	Pre
	195.5	79.3	4.3		39	71	Post
Patient 8	194.5	0.3	5.6		85	92	Pre
	198.5	9.1	7.9		57	87	Post
Patient 9	164.0	0.4	8.1	0.91	88	93	Pre
	184.5	2.3	7.2	0.58	89	93	Post
Patient 10	150.0	11.0	8.0		80	92	Pre
	26.5	18.0	7.0		54	88	Post
Patient 11	212.5	0.0	3.0	1.60	90	95	Pre
	173.0	11.0	7.0	0.95	89	95	Post
Patient 12	172.0	21.0	4.0		82	93	Pre
	122.0	24.0	7.0		82	94	Post
Patient 13	160.0	3.4	5.6		83	95	Pre
	120.0	4.5	12.0		89	96	Post
Patient 14	178.5	8.0	4.0	0.81	79	92	Pre
	89.5	3.0	5.0	0.52	81	91	Post
Patient 15	138.5	4.0	0.0	1.38	81	92	Pre
	160.0	4.0	3.0	0.93	81	92	Post
Mean	164.1	29.6	5.8	1.62	74	90	Pre
SD	27.2	52.4	7.2	1.24	18	7	Pre
Mean	142.5	24.9	9.0	0.94	69	89	Post
SD	46.8	34.4	11.4	0.70	20	7	Post
<i>p</i>	ns	ns	ns	$<0.02$	ns	ns	

TST—total sleep time; O<sub>2</sub>sat—nocturnal oxygen saturation; AI, HI, AHI—apnea, hypopnea and apnea/hypopnea indices; Rna—nasal airflow resistance (cm H<sub>2</sub>O/l/second) of both nostrils; Pre—pre-NOZOVENT data; Post—post-NOZOVENT data.

I and II is similar, slow wave sleep time is significantly reduced ( $22 \pm 16\%$  of total sleep time before NOZOVENT vs.  $5 \pm 8\%$  after;  $p < 0.005$ ) and rapid eye movement (REM) sleep time is significantly increased ( $20 \pm 12\%$  of total sleep time after NOZOVENT vs.  $9 \pm 7\%$  before;  $p < 0.01$ ). There was no significant difference in the time spent snoring in stages I, II and REM before and after NOZOVENT. However, in slow wave sleep insertion of nasal dilator significantly reduced snoring time from  $64 \pm 39\%$  to  $33 \pm 43\%$  of the stage sleep time ( $p < 0.05$ ).

Figures 4 and 5 show snoring frequency, snoring index and nocturnal sound intensities (maximum,

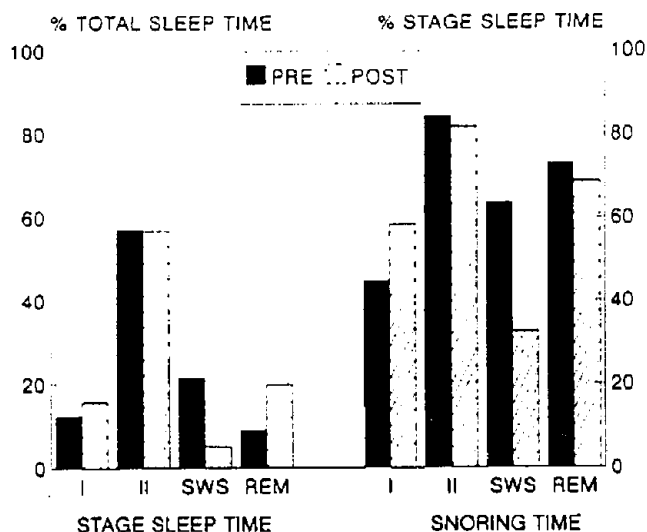


FIG. 3. Stage sleep time (as a percent of total sleep time) and snoring time (as a percent of stage sleep time) before and after NOZOVENT. Statistically significant reduction in slow wave sleep time ( $p < 0.005$ ), snoring time during slow wave sleep ( $p < 0.05$ ) and increase in REM sleep time ( $p < 0.01$ ) were observed.

minimum and average) for each stage of sleep before and after NOZOVENT. Nasal dilation induced beneficial changes in all of the above parameters only in slow wave sleep. In particular, there was a reduction in snoring index from  $9.4 \pm 7.0$  snores/minute of sleep to  $4.1 \pm 5.7$  snores/minute of sleep ( $p < 0.05$ ), in snoring frequency from  $11.6 \pm 6.1$  snores/minute of snoring time to  $5.2 \pm 7.0$  snores/minute of snoring time ( $p < 0.05$ ) and in sound intensities by approximately 6–9 dB.

## DISCUSSION

This study showed that nasal dilation reduces snoring in slow wave sleep and has no effect on AHI or nocturnal oxygen saturation.

Nasal obstruction has been linked to disordered breathing by several investigators. McNicholas et al. (5) showed that patients with allergic rhinitis have more apneas during the allergy season, when their noses are obstructed. Olsen et al. (9) found that nasal packing led to snoring and an increase in apneas. Hoffstein et al. (10) demonstrated that nasal airflow resistance correlated with snoring. Fairbanks (11) found that 77% of snorers had subjective improvement after nasal surgery. Therefore, it appears that there is a direct causal relationship between the severity of snoring and the degree of nasal obstruction. Consequently, it may seem surprising that the use of the nasal dilator in our patients did not result in a more significant reduction in snoring and sleep apnea. There are several possibilities that may account for this finding.

First, the relationship between snoring, apnea and

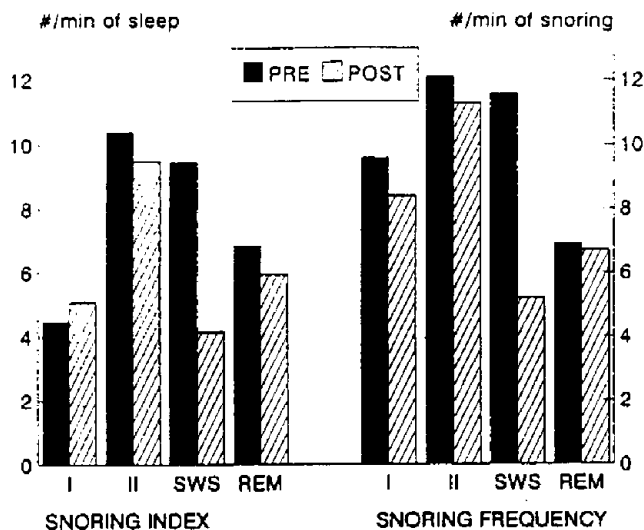


FIG. 4. Snoring index and snoring frequency for each sleep stage before and after NOZOVENT. Significant ( $p < 0.05$ ) reduction in these parameters was observed only during slow wave sleep.

nasal resistance is indirect. Neither the site of obstruction during apneas nor the site of generation of snoring is in the nose. Direct visual observation of the upper airways of snorers during sleep demonstrated (12,13) that these sites are either in the oropharynx, hypopharynx, at the level of the soft palate or at the velopharyngeal level. Nasal obstruction may further reduce inspiratory intra-airway pressure at these sites, making the walls more susceptible to collapse. However, it is possible that even without the facilitating effect of nasal obstruction, there is sufficient sleep-induced reduction in muscle tone to increase the compliance of the pharyngeal walls, reduce the area of the pharyngeal orifice,

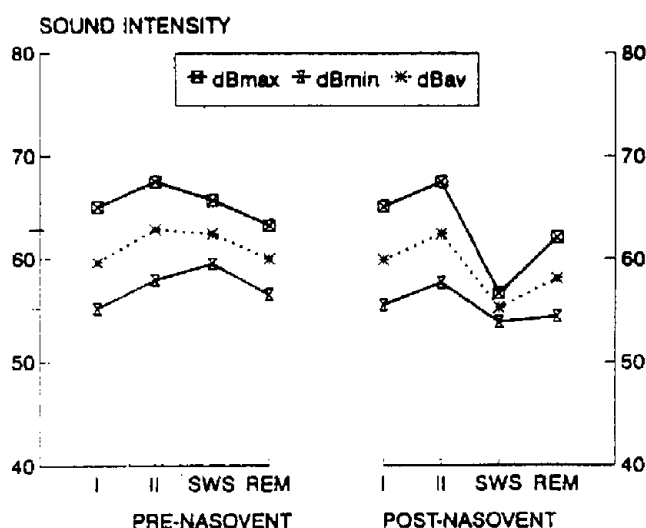


FIG. 5. Sound intensities (minimum, maximum and average) for each sleep stage before and after NOZOVENT. Significant ( $p < 0.05$ ) reduction was observed only during slow wave sleep.

increase turbulence, drop intra-airway pressure, produce either complete or partial occlusion of the pharynx and set into vibration the adjacent noncartilaginous structures. The simple fact that despite insertion of a fiberoptic instrument through the nose, investigators are still able to observe collapse and witness snoring (12,13), supports this hypothesis.

A second possibility is that we did not achieve sufficient reduction in nasal resistance despite the use of a nasal dilator. This possibility is highly unlikely. Although we only have data for seven subjects, all of them dropped nasal airflow resistance. Hoiijer et al. (14) and Metes et al. (15) documented 18% and 60% reduction in nasal airflow resistance with NOZOVENT in 11 and 72 snorers, respectively. Although all of these measurements were done during wakefulness, there is no reason to suspect that this reduction in nasal resistance is not present during sleep.

We must also consider the possibility that our method for measuring snoring is imprecise, and that is why we did not demonstrate a more significant reduction in snoring with NOZOVENT. In the absence of a standardized method for measuring snoring, it is difficult to compare our method to other methods described in the literature. However, each patient in this study acted as his/her own control. We analyzed the differences in snoring frequencies, thus eliminating any systematic bias present in the absolute measurement of these parameters.

Finally, it is possible that our results are biased because we used NOZOVENT during the second half of the night in all patients. If, for a given stage, snoring is different depending on whether this stage occurs during the first or second half of the night, a systematic bias would result. There is no information regarding the variation of snoring (within a given stage) with time from sleep onset. Some investigators found that snoring is more prevalent in slow wave sleep (16,17), whereas others found that it was uniformly distributed across all sleep stages (18), but none of them analyzed variability of snoring within a given stage. Our own anecdotal evidence suggests that snoring is uniform within a given stage independently of when this stage occurs during the night. However, in the absence of proper studies, this possibility of systematic bias influencing our results cannot be entirely dismissed.

Why snoring is most affected by nasal dilation during slow wave sleep is not clear. It may be related to the fact that upper airway resistance is highest during slow wave sleep, so that even a small drop in nasal resistance could lead to a substantial reduction in the negative intrapharyngeal pressure and less vibration of the adjacent structures (soft palate, uvula, pharyngeal walls). Alternatively, if NOZOVENT reduced respiratory rate during slow wave sleep, snoring frequency also would

be reduced. Although we did not specifically measure respiratory rate, it is unlikely that NOZOVENT reduced it by >50% to account for the observed reduction in snoring frequency.

Nasal CPAP, which dilates the entire nasopharyngeal airway, is much more effective in eliminating snoring and apneas than NOZOVENT, which dilates only the nasal airway. This implies that snoring is most likely generated in the pharynx, and that reduction in the anterior nasal resistance has little influence on the downstream resistance in the naso- and oropharynx.

In summary, we have demonstrated that nasal dilator NOZOVENT has a minor effect on snoring, reducing it only in slow wave sleep. Based on our findings, we cannot recommend the routine use of this appliance for treatment of snoring. We speculate that reduction in the resistance of the entire naso- and oropharynx may be required in order to improve snoring and apnea.

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