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Nasal Dilation, Sleep, and What Is Hypopnea?

The role of nasal resistance in the occurrence of snoring and obstructive sleep apnea hypopnea syndrome (OSAHS) has been a matter of speculation for years. In 1988, it was reported in a study of 16 patients that the use of a plastic internal nasal dilator (Nozovent; Prevancure; Frölunda, Sweden) improved airflow by approximately 25%.[1] It was speculated that such a device could decrease the volume of snoring and the degree of OSAHS severity.

In the following years, the inventor of the device and his teams of investigators published five studies supporting that contention. Four of the studies reported purely or mostly subjective data[2–5]; a fifth study[6] reported objective data regarding the effect of the device on OSAHS. Unfortunately, all of these studies shared design weaknesses that left their findings far from conclusive. Still, the studies suggested that the dilation of the nasal valve could be beneficial for snorers and those with OSAHS, and other investigators were sparked to examine those possibilities.

Studies have clearly verified that the placement of the Nozovent device increases the cross-sectional area of the nasal valve (the nasal valve is the narrowest segment of the nasal cavity, found at the junction of the upper and lower lateral nasal cartilages) and lowers nasal resistance. Most awake patients perceive improved nasal airflow that is similar to the effect of a topical decongestant. Still, the sleep-related findings of the initial investigators had not been verified. In 1992, two Canadian studies using the Nozovent device appeared that included nocturnal polysomnographic data.[7,8] While the details of these data were minimal in one of the studies, both of these investigators verified the aforementioned increase in nasal patency, but could not demonstrate any change in apneas or hemoglobin saturation associated with use of the nasal dilator. Only one of the studies looked at snoring and it reported no change. The original investigators had reported that people who use Nozovent felt an improvement in sleep quality. One of these Canadian studies indicated about a 20% decrease in nocturnal arousals that they believed might account for that perception of an improvement in sleep quality.

In this issue of *CHEST*, Schönhofer et al (see page 587) examined the effect of the Nozovent device on 26 consecutive subjects with OSAHS. Their study included a more rigorous objective investigation of the effect of the Nozovent device with nocturnal polysomnographic conditions that were well defined using a standard montage that included inductive plethysmography. Their subjective outpatient snoring data were based on a standard 5-point system, and perceived excessive daytime somnolence was estimated by the Epworth Sleepiness Scale (ESS). Mild to severe OSAHS patients were represented.

The study found no improvement in apnea frequency, snoring, oxygen saturation, or sleep parameters. A minimal improvement in the ESS score was noted; in 25 patients, no change or only a mild decrease in snoring was estimated by the bed partner.

This totals three independent investigations that have been unable to verify the findings of the original investigative teams. These independent studies appear to be better designed, with this most recent being very well done. We must strongly consider the possibility that the original reports may be incorrect. It may be that only a few patients derive significant objective benefit from the Nozovent device. So far, no way to identify those few patients has been found.

During the review process, the authors' use of the traditional definition of hypopnea was questioned (a $\geq 50\%$ reduction in the amplitude of oronasal airflow from baseline lasting at least 10 s, associated with a decrease in O_2 saturation of $\geq 4\%$). It is true that thermal sensors detect apnea well, but exhalations of 50 to 500 mL (hypopneas) might not exhibit enough variance in temperature to accurately indicate a decrease in volume. The decision to add a decrease in O_2 saturation to the definition has helped ensure that hypoventilation has actually occurred. However, if that definition were applied in the clinical practice of sleep medicine, scores of patients might be sent home without treatment for what is obvious OSAHS.

It may be time to reevaluate the "standard" definition of hypopnea. Certainly O_2 desaturation is a helpful adjunct when interpreting a reduction of the thermal oronasal airflow tracing, but the consideration of the following occurrences may also be helpful: (1) associated arousal, (2) concurrent crescendo snoring (in the snore-sensor graphic output channel), (3) associated decreases from baseline

snoring (in the snore-sensor graphic output channel), and/or (4) a repetitive pattern of occurrence consistent with OSAHS.

Schönhofer et al used inductive plethysmography to assess thoracoabdominal movement. This semiquantitative measure of air movement should also have been adequate to assess whether or not hypopnea had occurred without a requirement for a decrease in O_2 saturation. However, the authors' preference for the standard definition is understood. Still, it may be time to consider rethinking the definition of hypopnea for future research.

With up to 45% of middle-aged adults reporting some form of snoring, the authors correctly point out that the potential market for such devices is very large. Many patients who report to a sleep disorders center for evaluation have already tried these devices on their own. Studies such as the article from Schönhofer et al and the other investigators allow us to correctly advise our patients that these devices may only help a few. Such advice might help lead to more prompt evaluations of sleep-disordered breathing and to the savings of millions of dollars on devices that, for most patients, have no beneficial effect.

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Bronchoscopy Training and Competency

How Many Are Enough?

Elsewhere in this issue of *CHEST* (see page 625), Haponik et al report the results of a survey of 59 senior pulmonary and critical care medicine fellows who attended an industry-sponsored bronchoscopy course in conjunction with CHEST 1998, the annual American College of Chest Physicians meeting.[1] The authors found wide discrepancies in bronchoscopy training between institutions, in areas such as numbers of procedures performed, ancillary techniques instruction (for example, transbronchial needle aspiration), and fellows' subjective assessments of their program's bronchoscopy training quality. Rather than getting distracted by the obvious scientific limitations and possible biases of the survey, I will focus my comments on the authors' primary conclusion that "an effort to apprise and enhance the quality of bronchoscopy training is necessary."

In 1964, Ikeda et al developed standards for the flexible fiberoptic bronchoscope (bronchofiberscope),[1] and in 1968 it was described as a diagnostic instrument.[2] Over the ensuing years, medical equipment manufacturers worked with pulmonary physicians to develop amazing advances in bronchoscope flexibility and optics, a dazzling array of bronchoscopic instruments, and new applications of the procedure. The bronchoscopist of today can perform laser therapy, cryotherapy, brachytherapy, stenting, localization of areas of dysplasia and carcinoma *in situ* using tissue autofluorescence, and ultrasound localization of mediastinal nodes for transbronchial needle aspiration, all procedures that were unimagined or impossible 20 years ago. There is no question that by enabling pulmonologists to gather tissue biopsies from the lower respiratory tract, Ikeda's invention greatly improved the scientific underpinnings of pulmonary medicine. Most modern-day pulmonologists perform flexible fiberoptic bronchoscopy,[3,4] which has become the key procedure defining our specialty.

Therefore, it is surprising that despite the substantial literature on teaching bronchoscopy referenced by Haponik et al, pulmonary and critical care medicine training programs have not adopted minimal essential numeric thresholds for bronchoscopy training.[5(p118,120)] I believe it most logical that pulmonary and critical care medicine program directors, who meet biannually at the American College of Chest Physicians and American Thoracic Society annual meetings, accept the important, though controversial, task of defining such criteria. After our