Obstruction of the Nose and Breathing during Sleep

Healthy adults prefer to breathe through their noses while either awake or asleep. This mode of breathing protects the lower airways because the nasal air passages, with their large surface area and ciliated, highly-vascular, mucus-rich epithelium, are particularly suited to filter and condition inspired air. However, protection is purchased at a price. The narrow convoluted nasal air passages account for about one-half of the total respiratory resistance to air flow, and work of breathing is more than doubled by breathing through the nose rather than the mouth. The mechanical limitations of nasal breathing are illustrated by the fact that healthy people substitute oral breathing for nasal breathing to achieve the high flow rates necessary for exercise and for rapid inspiration during talking and singing.

In the awake adult, the transition from nasal to oral breathing is easily accomplished. This is not the case with infants, in whom the close approximation of the soft palate, tongue, and epiglottis makes oral breathing difficult. Although this difficulty can be overcome by crying, it is exaggerated by sleep; and a significant number of infants experience prolonged apnea if their noses are occluded during sleep. The adverse effect of nasal obstruction on the breathing of sleeping infants has long been appreciated by pediatricians and is vigorously advanced by some as a possible cause of the sudden infant death syndrome. The effect of nasal obstruction on the breathing of sleeping adults and older children, on the other hand, has received little attention. However, this has been corrected by several recent reports. Although the experimental designs and the monitoring techniques of these studies differ slightly, they each show convincingly that obstruction of the nose causes or worsens disordered breathing in sleeping adults, as well as children. Among the breathing abnormalities noted during sleep with nasal obstruction were apnea, both central and obstructive, hypopnea, and periodic breathing. Many of the subjects in these studies responded dramatically to nasal obstruction. For instance, five of the ten young, healthy men studied by Zwillich and associates experienced a greater than four-fold increase in apnea after obstruction of their noses, whereas five of seven patients monitored by Taasan and associates before and after the placement of nasal packs had changes of similar magnitude. In each of these studies, nasal obstruction alone caused sleep apnea sufficient in some patients to place them in the category of the sleep apnea syndrome. Abnormal breathing was most prominent after complete nasal obstruction, but was seen with partial obstruction as well.

The mechanism by which obstruction of the nose alters the breathing of sleeping adults is not clear, especially since no single type of abnormal breathing has been consistently noted. It has been proposed that the loss of the stimulating effect of nasal air flow may mediate this response either by decreasing the activity of all respiratory muscles, causing central apnea, or by selectively inhibiting the oropharyngeal dilator muscles, causing obstructive apnea. On the other hand, nonspecific changes in upper airway resistance may also play a role. In contrast to wakefulness, the generalized loss of muscle tone and the reduced respiratory response that naturally accompany sleep may interfere with oral airway patency and, thus, may increase oropharyngeal resistance and promote airway collapse during inspiration. In their investigation of the effects of nasal obstruction on breathing during sleep, Olsen and associates noted that during sleep, inspiratory intrapleural pressures were consistently greater with the nose obstructed than with it open; this suggests that oral breathing during sleep is a high rather than low resistance pathway.

The clinical consequences of nasal obstruction are just beginning to be appreciated. They vary from mildly-annoying to life-threatening. For instance, with their noses obstructed, healthy subjects woke more often, slept more lightly, and experienced a greater number of shifts in sleep stages: when questioned afterwards, these subjects reported disturbed, restless sleep.

Perhaps more important, nasal obstruction de-
creases oxygen saturation during sleep, even in otherwise healthy young adults. Among the four subjects in whom oxygen saturation was monitored by Zwillich et al., episodes of desaturation (\(Sa_O_2 < 90\) percent) increased from a total of 27 before obstruction to 255 afterwards. A more alarming increase in both the number and the severity of episodes of desaturation was noted by Taasan et al.\(^9\) in four of seven patients who had nasal packs placed after orolaryngologic surgery. Decreases in \(Sa_O_2\) as great as 39 percent were noted in one patient who had no significant desaturation when his nose was not obstructed. Perhaps this may explain reports of stroke, myocardial infarction, and sudden death in patients with nasal packing.\(^{13,14}\)

Further evidence of the clinical significance of nasal obstruction comes from the recent description of three older children who had velopharyngeal incompetence and experienced prolonged apnea and unexpected death after the construction of pharyngeal flaps designed to improve their speech by obstructing their noses\(^{15}\) and from occasional reports of patients with sleep apnea syndromes who apparently improved after correction of nasal obstruction.\(^{16,17}\) These latter reports raise the possibility that nasal obstruction may play an etiologic role in the sleep apnea syndrome. They are particularly interesting in light of a recent study by Anch and associates\(^{18}\) in which obese subjects with obstructive sleep apnea were found to have nasal pharyngeal airway resistance three to four times greater than healthy control subjects. This increased airway resistance was worse in a supine position and could not be eliminated by the administration of a nasal decongestant, which strongly suggests that it resulted from structural narrowing rather than mucosal congestion. Since bony abnormalities of the nose and adenotonsillar enlargement were ruled out in these patients, it appeared that nasopharyngeal deposition of fat might best explain the findings. Thus, increased nasopharyngeal airway resistance could well be a critical factor in promoting airway collapse in sleeping obese patients with sleep apnea.

Nasal obstruction, whether caused by upper respiratory infection, allergy, or structural defect, is a common, almost-everyday occurrence. The clinical manifestations of abnormal breathing during sleep are varied, ranging from cor pulmonale, cardiac arrhythmia, and hypersomnolence, to mild changes in personality and restless sleep. Given these facts, one cannot help but speculate on a wide range of situations in which obstruction of the nose could have an adverse influence. The first step in evaluating the true importance of nasal obstruction to breathing during sleep—recognition of the problem—has been taken. The physiology of this phenomenon must now be explored and its clinical consequences more clearly delineated.

James W. Wynne, M.D.
Gainesville, Florida

Associate Professor of Medicine and Anesthesiology, University of Florida.

REFERENCES
5 Moss ML. The veloepiglottic sphincter and obligate nose breathing in the neonate. J Pediatr 1965; 67:330-31
7 Purcell M. Response in the newborn to raised upper airway resistance. Arch Dis Child 1976; 51:602-07
10 Taasan V, Wayne JW, Cassisi N, Block AJ. The effect of nasal packing on sleep-disordered breathing and nocturnal oxygen desaturation. The Laryngoscope 1981; 91:1163-72
13 Cassisi NJ, Biller HF, Ogura JH. Changes in arterial oxygen tension and pulmonary mechanics with the use of posterior packing in epistaxis: a preliminary report. The Laryngoscope 1971; 81:1281-86
17 Simmons FB, Guillemainaut C, Dement WC, Tilkian AC, Hill M. Surgical management of airway obstructions during sleep. The Laryngoscope 1977; 87:326-38