

Treatment Paradigm for Nasal Airway Obstruction

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KEYWORDS

- Nasal airway obstruction • Treatment • Septal deviation • Internal nasal valve
- External nasal valve • Nasal turbinate

KEY POINTS

- Pathology leading to symptomatic nasal obstruction may be structural, inflammatory, or other in nature; most patients have more than 1 factor contributing to persistent nasal congestion.
- Medical therapy for nasal obstruction focuses on controlling inflammatory mucosal disease.
- Optimizing outcomes in surgery for nasal airway obstruction requires careful preoperative evaluation, including nasal endoscopy, to accurately identify contributing factors in individual patients.
- The internal nasal valve has the narrowest cross-sectional area within the nasal airway and is thus most sensitive to changes in its dimension due to anatomic variation or surgical intervention.

INTRODUCTION

Nasal airway obstruction (NAO) is a common otolaryngic complaint, with estimates of up to one-third of the adult population having some degree of NAO and one-quarter of affected patients seeking intervention.¹ An estimated 9.5 million office visits occurred in the United States in 2006 for the evaluation of nasal congestion.² The monetary cost of nasal obstruction is significant. Approximately 3 decades ago, Kimmelman³ estimated \$5 billion were spent annually for symptomatic relief and another \$60 million on surgical procedures to address anatomic causes of obstruction. Surgical intervention to alleviate recalcitrant nasal blockage is commonly performed both in the United States⁴ and in other developed countries.⁵

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The anatomy and physiology of nasal airflow are complex subjects addressed in greater detail in, David W. Hsu and Jeffrey D. Suh's article, "[Anatomy and Physiology of Nasal Obstruction](#)," in this issue. The rigid framework supporting the nasal airway is provided by the bony and cartilaginous septum and paired nasal bones. The bony septum consists of the vomer, perpendicular plate of the ethmoid, and maxillary crest. The lateral nasal walls contain the paired inferior, middle, and superior turbinates, sometimes supplemented by the supreme turbinates. The turbinates have both bony and soft tissue components and thus may represent both fixed and variable sources of nasal obstruction. The internal nasal valve is formed by the nasal septum, upper lateral cartilage (ULC), and head of the inferior turbinate and is the region of the nose with the narrowest cross-sectional area. The normal angle between the nasal septum and ULC is 10° to 15° ; small decreases in this angle may result in symptoms of nasal blockage.⁶ The external nasal valve is caudal to the internal valve and represents the soft tissues of the nasal vestibule, consisting of the septum, columella, nasal sill, and ala⁷ (**Fig. 1**). The posterior limit of the nasal cavity is delineated by the choanae, which serve as the transition point to the nasopharynx.

The velocity of nasal airflow is inversely related to the cross-sectional area within a particular subsite of the nasal airway; the internal nasal valve has the smallest area and thus the most rapid airflow. Bernoulli's law dictates that an increase in velocity is associated with a decrease in intraluminal pressure. The resulting pressure differential induces a collapse of surrounding soft tissue and worsening of airway obstruction. Furthermore, Poiseuille's law explains that the flow of a fluid, in this case air through the nasal cavity, is inversely proportional to the fourth power of the radius of the lumen. As a result, even small modifications to the cross-sectional area of the nasal airway can produce dramatic effects on airflow, particularly through the internal nasal valve.⁵

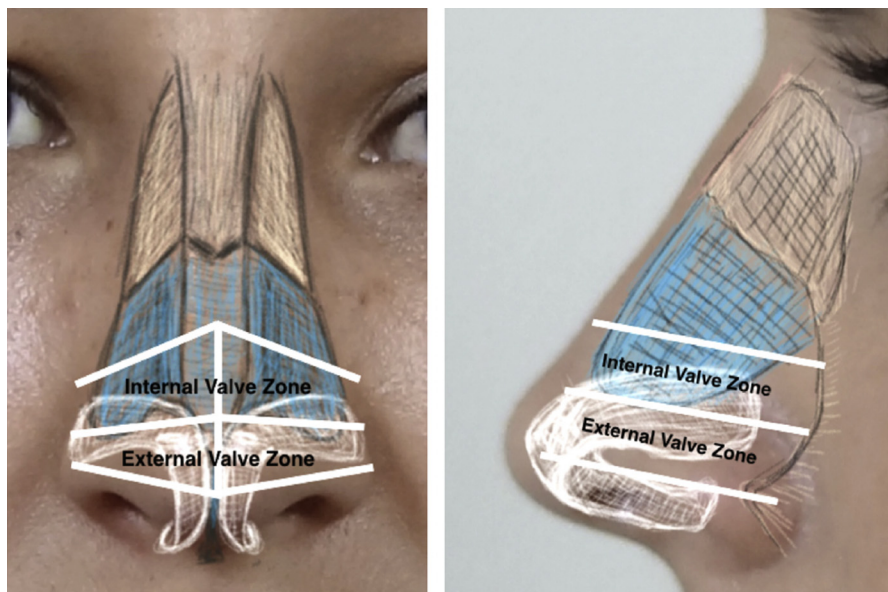


Fig. 1. Zones of lateral nasal sidewall collapse (left: coronal view; right: sagittal view). The internal valve zone corresponds to the inferior portion of the ULC and scroll region. The external valve zone corresponds to the soft tissues of the nasal ala. (From Barrett DM, Casanueva FJ, Cook TA. Management of the nasal valve. *Facial Plast Surg Clin N Am* 2016;24(3):221; with permission.)

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