

Review

Evaluation of upper airway obstruction—An ENT perspective

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Abstract

Upper airway obstruction is a common clinical problem. Nasal obstruction can be the result of nasal anatomy, mucosal swelling, or acquired blockage. The anatomy of the upper airway differs from the lower because of the presence of erectile tissue in the nose. Measurement of nasal airway obstruction can be performed with video endoscopic photo-documentation, rhinomanometry, nasal inspiratory peak flow, visual analog scales, and psychometric validated questionnaires.

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Keywords: Acoustic rhinometry; Objective measurement; Video endoscopic photo-documentation; Rhinomanometry; Nasal inspiratory peak flow; Visual analog scale; Psychometric validated questionnaires

Contents

1. Introduction	433
2. Physiology of nasal obstruction	434
3. Upper versus lower airway obstruction	435
4. Diagnosis of UAO	436
5. Techniques for objective measurement of UAO	437
5.1. Video endoscopic photo-documentation	437
5.2. Rhinomanometry	437
5.3. Nasal peak flow	438
5.4. Acoustic rhinometry	439
6. Assessment of the patients' perception of obstruction	439
7. Difference between upper and lower respiratory tract	440
8. Summary	440
Acknowledgments	440
References	440

1. Introduction

Upper airway obstruction (UAO) is a common clinical problem. The upper airway extends from the nares to the larynx and is continuous with the lower airway which

begins at the trachea and extends to the alveoli. Most clinicians manage UAO due to nasal blockage clinically, without formal testing, in the office. More rigorous objective evaluation and monitoring can aid the specialist and researcher. Several techniques are available to evaluate nasal patency. These varied techniques provide information to more objectively appraise the type and degree of nasal obstruction.

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2. Physiology of nasal obstruction

Although we commonly think of the airway as a “tube” or “pipe,” the upper airway has numerous structures that causes variations in the “tube” influencing the resistance or obstruction to airflow. Nasal obstruction and congestion are not synonymous. Obstruction can be mostly fixed due to anatomical properties of the nasal airway or mostly reversible, due to mucosal congestion swelling and engorgement [1,2]. The nasal airway begins at the external nares and extends back to the nasopharynx [3].

The external architecture of the nose consists of two nares that form the cartilaginous pyramid. Each nare is formed by two distinct pieces of cartilage. The alar cartilage forms the inferior portion of the nostril and attaches to the upper lip. The lateral cartilage is superior to the alar cartilage and anchored to the nasal bone. The two nares are separated by the nasal tip (or lobule) and the septal cartilage. The openings created by these cartilages are known as the nasal vault. The most anterior part of the nasal vault, bounded by the alar (laterally) and septal (medially) cartilages, is known as the vestibule [4] (Fig. 1). The external pyramid is formed by the bony and cartilaginous portion of the nose. The internal nasal architecture is formed by the septum medially and laterally by the lateral nasal wall to which the turbinates are attached. The apex of the internal pyramid, at the superior turbinate and above, contains olfactory epithelium. Most of the respiratory airflow occurs anteriorly, through the nasal vestibule (the narrowest portion of which is called the “nasal valve”) and below the superior turbinate, passing over the middle and inferior turbinates into the nasopharynx. The internal canthus is the functional dividing line between the anterior nose (vestibule), in which the primary resistance is due to bony and cartilaginous anatomy, and the internal nasal cavity, where the primary resistance is due to fluctuations in the size of the turbinates. Anatomical

variations are common however, and these can cause blockage of the nasal respiratory passages at each level.

There are a wide range of sizes and shapes within the bounds of “normal” external nasal architecture; deformities of the external nasal cartilage can lead to clinically relevant obstructions in nasal airflow [5]. The internal width of the nasal vault is relatively small when compared to the rest of the respiratory tract.

The nasal vestibule opens into the area of the nasal valve, the functionally narrowest portion of the nasal respiratory passage. The nasal valve is bounded laterally by the lateral cartilage and medially by the nasal septum. Allergens and foreign particles are filtered by the nose. The narrowness of the nasal airway is also functionally relevant because approximately half of the total airway resistance is attributed to the tapered nasal airway [3].

The nasal septum is a common cause of nasal airway obstruction. The anterior portion of the septum is cartilaginous and flexible. The anterior portion connects directly to the rigid, bony posterior portion of the septum which is formed by the ethmoid and vomer bones. While a perfectly straight nasal septum is rare, deviations to either side can cause obstruction [4].

The lateral nasal wall gives rise to three turbinates or conchae. The superior, middle and inferior turbinates project from the lateral nasal wall into the internal nasal airway. These turbinates create “chambers” beneath them known as the superior, middle and inferior meatus. Each meatus opens into the nasal choana. The choana open into the nasopharynx, where the nasal airway joins the oral airway and the lower respiratory tract [4]. The maxillary, anterior ethmoid, and frontal sinus drain into the nose at the area of the ostiomeatal complex, also known as the ostiomeatal unit. This complex/unit is the space formed by the septum medially, the middle meatal opening and middle turbinate laterally. Blockage in this area due to a deviated septum, turbinate hypertrophy, scarring, or other blockage of the middle meatus by polyps or tumors may lead to sinusitis. Fig. 2 illustrates the area of the ostiomeatal complex for the left maxillary antrum (sinus) in a coronal view. While blockage in this area may lead to sinusitis, it does not necessarily cause the sensation of nasal obstruction as most of the “sensation” of the nasal patency occurs anteriorly at the nasal valve and vestibule.

Blockages caused by variations in the bony or cartilaginous anatomy anywhere along the nasal airway are usually fixed and irreversible. Blockage can be due to a variation of normal anatomy (e.g. a narrow nasal vault or a congenitally deviated septum), or acquired, abnormal anatomy (post-rhinoplasty, traumatic septal deviation, compensatory bony turbinate hypertrophy). This type of obstruction generally requires surgical correction and is not amenable to pharmacological intervention. Other pathologic causes of fixed obstruction include nasal polyps, other sinonasal tumors, hypertrophic adenoids and foreign bodies.

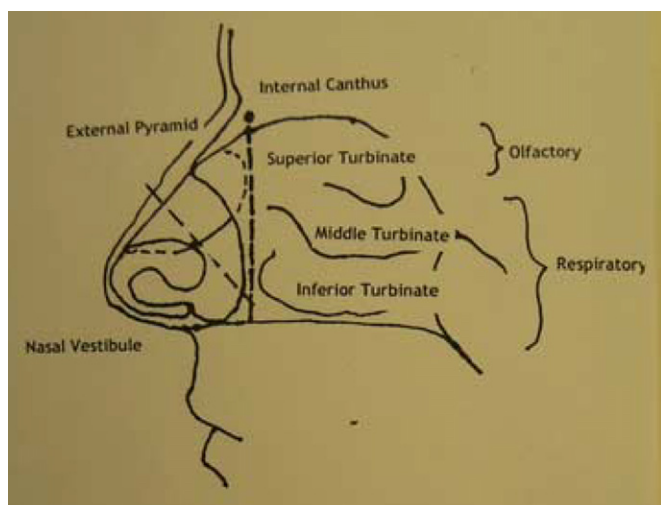


Fig. 1. Medial view of internal nasal anatomy highlighting its relationship to the external nasal pyramid.

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