Anatomy and Physiology of Nasal Obstruction

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KEYWORDS

• Nasal obstruction • Deviated septum • Nasal valve collapse • Turbinate hypertrophy • Nasal cycle

KEY POINTS

- The cause of nasal obstruction is caused by a wide array of anatomic, physiologic, pathophysiologic, and iatrogenic factors; it can often be multifactorial.
- Optimal nasal airflow is determined by patent nasal passages, intact mucociliary function, normal functioning receptors for airflow, and degree of mucosal inflammation.
- The internal nasal valve comprises the area of greatest overall resistance to airflow and is affected by Bernoulli forces and Poiseuille law.
- The septum and turbinates are common anatomic causes for nasal obstruction but are easily treatable.
- Nasal obstruction in rhinitis and sinusitis are caused by aberrant inflammatory responses.

INTRODUCTION

Nasal obstruction is a common presenting symptom to both primary care physicians and otolaryngologists and may be caused by a wide range of anatomic, physiologic, and pathophysiologic factors. Up to one-third of the population complains of nasal obstruction and seeks treatment from their physicians.[1] In this article, the authors discuss the anatomy and physiology of nasal obstruction.

The human nose is evolutionarily adapted to warm, humidify, and filter inspired air before its reaching the pulmonary system. In this way, the nose and lungs work together as a unified airway. The nose also plays an important role in combating inhaled foreign particles and detecting odorants for olfaction. It has evolved to bring in a large volume of air through the nostrils and nasal cavities while also maximizing the air's contact with mucosal areas.[2] These adaptations include increased nasal projection, anterior nasal convexity, exaggerated nasal angles, anterior nasal spine prominence, and an intricate nasal cartilaginous tip.[3]

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Optimal nasal airflow requires (1) patent nasal passages, (2) intact mucociliary function, (3) normal functioning receptors for airflow, and (4) absence of mucosal inflammation. Any aberration of these factors can lead to the sensation of decreased airflow. Air contacts the nasal mucous membranes as it flows through the nasal valve, travels past the septum and turbinates, and finally flows through the nasopharynx. Anatomic changes can disrupt this flow, thus causing resistance and subsequent nasal obstruction.

As the air flows through the nose, the turbinates induce nonlaminar flow and air temperature changes that are sensed by trigeminal cool thermoreceptors, which then regulate the sensation of airflow and nasal patency.[4] Other receptors such as pain receptors or mechanoreceptors may also play a role. Any dysfunction of the mucociliary function or airflow receptors will adversely affect nasal airflow and cause the sensation of decreased air passage.

Anatomic causes of nasal obstruction include internal or external nasal valve stenosis/collapse, septal deviations, an enlarged septal swell body, inferior turbinate hypertrophy, choanal stenosis/atresia, nasopharyngeal obstruction, and iatrogenic scarring. Physiologic causes include the natural nasal cycle, changes in the nasal autonomic nervous system, and sinonasal inflammatory conditions.

NASAL ANATOMY

Skin/Soft Tissue Envelope

Overlying the skeletal framework of the nose is the skin-soft tissue envelope (SSTE).[5] The skin of the nose varies in thickness going from superior to inferior along the nose. The skin is thickest at the nasion, which denotes the bony junction of the frontal bone with the 2 nasal bones. The SSTE is thinnest at the rhinion, which is the osseocartilaginous junction of the caudal edge of the nasal bones and cephalic edge of the upper lateral cartilage (ULC). The skin does thicken along the dorsum as it descends from the rhinion to the nasal tip, where sebaceous glands reside.

The subcutanenous layer of the nose is composed of a superficial fatty layer, a fibromuscular layer, a deep fatty layer, and periosteum and/or perichrondrium.[5] Importantly, the fibromuscular layer is the nasal subcutaneous muscular aponeurosis system (SMAS), which connects with the SMAS layer of the face and encases the mimetic muscles of the nose.[5]

Deep to the SSTE is the investing nasal musculature. Elevator muscles include the procerus, levator labii superioris alaeque nasi, and anomalous nasi. Depressor muscles are the alar nasalis and depressor septi nasi. Compressor muscles include the transverse nasalis and compressor narium minor. And finally, dilator naris anterior is a minor dilator muscle.

The internal nasal lining consists of keratinizing squamous cell epithelium at the nasal vestibule. Once inside the nasal cavity, the surfaces are composed of pseudostratified ciliated columnar respiratory cells. Seromucinous glands are also abundantly found in the sinonasal cavity.

Nasal Bony Framework

The framework of the nose is commonly divided into thirds: the upper third comprises the osseous nasal vault made up by the nasal bones, the middle third defined by the ULCs, and the lower third defined by the lower lateral cartilages (LLCs) (**Fig. 1**A).[6]

The osseous vault forms a pyramidal structure and consists of the paired nasal bones attaching superiorly to the frontal bone and laterally to the frontal process of the maxilla.[5] This bone framework along with the bony septum provides the principal