Empty Nose Syndrome

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INTRODUCTION

Empty nose syndrome (ENS) was originally named in 1994 by Dr. Eugene Kern to describe a combination of symptoms suffered by some patients who had received a partial and/or total middle and/or inferior turbinectomy (removal of part or all of structures that attach to the side wall of those nose and filter and humidify the air we breathe). He described patients who suffered from severe nasal dryness and crusting, and a sense of nasal obstruction despite enlarged nasal airway passages. Since then, the true existence of this syndrome has remained controversial, with the vast majority of patients undergoing middle and/or inferior turbinate surgery receiving a measurable improvement. Surgical benefits of turbinate reduction include: improvements in nasal breathing, decreased use of nasal medications, and improvements in validated quality of life survey instruments. Most patients who undergo significant resections of their internal nasal structures, particularly in the setting of nasal and/or skull base cancers do not exhibit severe postoperative symptoms. Nevertheless, thousands of patients across the world have reported similar symptoms of excessive nasal dryness, nasal obstruction, and significant impact on sleep and quality of life after turbinate surgery.

WHAT ARE THE SYMPTOMS OF EMPTY NOSE SYNDROME?

Patients reporting to suffer from empty nose syndrome often complain of nasal dryness, the sensation of nasal obstruction in spite of having large nasal airways, feelings of both obtaining too much dry, cold air, and not being able to inhale a satisfactory nasal breath, and some patients complain of suffocation symptoms that affect their ability to sleep. Patients with empty nose syndrome may also suffer from other co-morbidities, such as severe depression, anxiety, and/or other psychiatric conditions that can either be present before surgery or begin at the same time as their ENS symptoms.

WHAT ARE THE FUNCTIONS OF THE INFERIOR AND/OR MIDDLE TURBINATES?

Nasal turbinates are complex, respiratory epithelium lined structures that serve a variety of important functions. One of the main functions of the turbinates is to serve as an intermittent, countercurrent heat exchanger, and rapid humidifier/dehumidifier for inhaled air. Mammals and birds, the other warm blooded animals known to have turbinates, consume significantly greater amounts of oxygen than equivalently sized cold blooded animals. This leads to significantly

increased rate of breathing and the potential for significantly greater rates of heat and water loss associated with that breathing. Warm blooded animals maintain large inferior turbinates situated directly in the path of respiratory airflow, which greatly increases the surface area of nasal mucosa (lining of the nose). In studies that compared nasal airflow with artificially obstructed oral airflow in warm blooded animals, experimental data demonstrated that on expiration, air is cooled and becomes supersaturated as it passes over the inferior turbinates, thus leading to condensation and recycling of excess moisture and minimizing evaporative heat loss. Similarly, on inhalation, air is rapidly warmed and humidified which leads to more efficient oxygen transfer in the alveoli (small parts of your lung). As a result, the evolution of a respiratory turbinate complex has been seen as a key adaptation allowing mammals and birds to tolerate extreme habitats.

WHAT IS THE NASAL CYCLE?

Nasal turbinates also contain a significant amount of erectile tissue (tissue that swells), particularly within the anterior aspect of the inferior turbinate, thought to allow precise regulation of nasal airflow. In particular, this erectile tissue is thought to be essential for the nasal cycle, in which one nostril becomes congested while the other side is decongested. The true importance of the nasal cycle is unknown, but it is thought that the nasal cycle may be important for reducing the risk of pressure ulcers during sleep. Nasal cycle duration is longer than in wakefulness, and changes from side to side of the nasal cycle frequently coincide with changes in posture during sleep, tend to occur during REM sleep (the deepest part of sleep), never occur during slow wave sleep, and maybe absent in patients with severe nasal obstruction. The inferior turbinates contain a significant amount of autonomic innervation (the part of the nervous system that functions automatically). As a result, it is reasonable to think that patients with autonomic nerve damage to the inferior turbinates may have abnormalities and/or difficulties during sleep.

HOW CAN RESECTION OF TURBINATES LEAD TO WORSE CONGESTION?

Recent research suggests that the feeling of nasal congestion is likely related to the body sensing different levels of pressure and/or temperature in each nasal cavity. These pressure and temperature receptors are likely located on the turbinate structures themselves, typically within the surface mucosal layer. With over-aggressive resection (surgically removing) of these turbinate structures, particularly with resection of the mucosal layer of the turbinates, people with ENS lose the ability to feel their nasal breathing. Furthermore, exquisite control of the diameter of the inferior turbinates is necessary to maintain laminar airflow (air moving through organized streamlines) that allows greater volumes of air to flow through the nose per a given breath. Without control of the diameter of the nasal airway, or with altered structures such as a septal perforation (a hole through the wall that separates the nasal cavity from right and left sides), nasal airflow becomes turbulent (disorganized streams of airflow) which limits the volume of airflow through the nose. Finally, the mucosal layer of the turbinates is an important immune-system organ, providing the first line of defense against bacteria and other pathogens that are inhaled. Through our own immune defense systems, the mucosal layer of our turbinate

epithelium maintains a population of harmless bacteria that helps to crowd out dangerous bacteria from entering the nose. Patients with ENS lose these regulators of the bacterial population, and dangerous and harmful bacteria slowly colonize the nose. These bacteria can cause crusting, bleeding, worsening nasal congestion, and worsening dryness of the remaining nasal lining which leads to a vicious cycle and worsens the symptoms associated with ENS.

WHAT TREATMENTS ARE AVAILABLE FOR EMPTY NOSE SYNDROME?

Empty nose syndrome can be treated with topical therapies that attempt to moisturize the nose. Unfortunately, many of these therapies are probably harmful and provide limited benefit. For example, repeated saline irrigations or saline gel provide temporary relief of nasal dryness but wash out the proteins and mucins in the mucus layer lining the nasal cavity. These proteins include host defense peptides such as lactoferrin, human beta-defensins, and others that regulate the usual bacteria that exist within the nose. Washing these proteins away also interferes with the nose's ability to protect the nasal mucosa from dangerous bacteria, such as Klebsiella ozenae.

Humidifiers are helpful since oxygen transfer in the alveoli of our lungs is most efficient at 100% humidity, however, most commercial humidifiers become rapidly contaminated with fungus. Constant fungal exposure in the setting of ENS is likely more harmful than the benefit gained from using a humidifier. More helpful than a humidifier is living in a warm, humid climate, particularly with salty air, such as close to a tropical beach.

Antibiotic nasal irrigations are often necessary in advanced forms of ENS to kill and deter growth of harmful bacteria such as Klebsiella species. Certain creams and oral therapies have been tried as well to hypertrophy (increase the size) of any remaining turbinate tissue. These include estrogen creams (turbinates enlarge during pregnancy) and recently medications like Viagra. Phosphodiesterase inhibitors such as Viagra are known to cause nasal congestion, possibly by inducing hypertrophy of nasal mucosa.

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Surgery for ENS has typically involved using implants or bulking materials to increase the size of any remaining turbinate tissue, or to increase nasal resistance by implanting materials in other locations (such as the nasal septum) in an attempt to recreate a nasal turbinate. These implants have been partially successful in reducing the turbulence of nasal airflow that occurs. However, these implants do not reproduce the humidification or immune protection aspects of the original turbinate mucosa. The choice of implant is also difficult for surgeons. Initially surgeons prefer to use absorbable materials such as hyaluronic acid to see if patients will benefit from an implant. Later, more permanent implants such as with Gore-Tex or acellular dermis have been tried. Many patients report modest benefit with these implant options. Recently, a few centers have started implementing platelet-rich plasma combined with acellular extracellular matrix implants for the therapy of ENS.

WHERE CAN I FIND FURTHER INFORMATION?

The ARS recommends contacting a specialist in rhinology. You can search for one here.

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