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Review Article

Chronic rhinosinusitis and endoscopic sinus surgery

Opeyemi O. Daramola [a], **Rakesh K. Chandra** [b,*]

[a] *Rhinology, Sinus and Skull Base Surgery, Division of Otolaryngology-Head and Neck Surgery, Crystal Run Healthcare, 2 Centerock Rd, West Nyack, NY 10994, USA*
[b] *Rhinology, Sinus & Skull Base Surgery, Vanderbilt University, 1215 21st Ave S, #7209 MCE S Tower, Nashville, TN 37232-8605, China*

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Abstract Olfactory dysfunction is a major symptom reported by patients with chronic rhinosinusitis (CRS). Surgical treatment of this disease requires close surveillance of such dysfunction because of wide ranging implications for safety, quality of life, and impact on the flavor of foods and beverages. This review highlights key findings regarding the influences of endoscopic sinus surgery (ESS) on olfactory function across the unique presentations of CRS. Such findings provide information useful for informing patients of potential complications and for obtaining informed consent prior to surgical intervention. ESS has been shown to improve olfaction across all types of CRS as assessed through quantitative testing and subjective reports. The presence of nasal polyposis (NP) and eosinophilia have been identified as predictors of significant postoperative olfactory improvement. When indicated, judicious partial resection of the middle turbinate may result in improved olfactory function without a risk of long term complication. Careful attention to the olfactory cleft and frontal sinus recess are important in limiting olfactory complications by avoiding indiscriminate disruption of olfactory epithelium. Given the chronic nature of the disease, surveillance of olfactory function in patients with CRS is a lifelong activity that will evolve as emerging technologies become available.

* Corresponding author. Fax: +1 615 936 8969.
E-mail addresses: ope@northwestern.edu (O.O. Daramola), rakesh.chandra@vanderbilt.edu (R.K. Chandra).
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Introduction

Olfactory dysfunction is a well-established cardinal symptom of chronic rhinosinusitis (CRS), with prevalence estimates ranging from 30% to 80%.[1] Although this wide range of estimates undoubtedly reflects variability in testing, the skill of the surgeon, underlying disease, and other factors, even the lowest of these estimates is high. This underscores the importance of addressing this issue when considering surgical interventions, particularly given the impact of olfactory dysfunction on quality of life and safety. The pathophysiology of olfactory dysfunction in CRS is multifactorial, involving significant obstructive and sensorineural processes.[1–5] Obstructive pathology causing conductive loss includes nasal polyposis, mucosal edema, adhesions, and septal deviations. Sensorineural loss is expectedly more complex with identification of inflammatory factors, neural involvement of the olfactory epithelium, and comorbid skull base disease.

This article describes the impact of ESS on olfactory function across the unique phenotypes of chronic rhinosinusitis. Evidence documenting functional outcomes of olfactory ability after ESS is summarized as guidance for discussing patient expectations. In addition, we review intraoperative practices and measures that reduce the likelihood of iatrogenic olfactory loss.

Anatomy and physiology of olfaction

This section is not intended to be a compendium on olfactory anatomy pathway. However, a working knowledge of relevant sinonasal and skull base anatomy, along with an understanding of pathophysiologic pathways involved, is essential in tailoring surgical goals to limit iatrogenic olfactory dysfunction and improve postoperative olfactory outcomes. Olfaction starts with inhalation of odorant stimuli. There are two peripheral pathways involved in the physical transport of odorants to olfactory cleft: orthonasal flow of odorous stimuli directly through the nares and retronasal flow via the choanae. The retronasal pathway is involved in perception and refinement of flavor during consumption of solid and liquid food.[6] The olfactory cleft epithelium consists of 10—20 million olfactory neurons. Histologically, this epithelium is pseudostratified columnar and includes basal cells (stem cells), supporting cells (Bowman glands, microvilli cells, sustentacular cells) and olfactory receptor cells.[2,7] The receptor cells are bipolar cells with nonmotile ciliated dendrites that extend from the olfactory vesicle to epithelial/apical surface for detection odorous stimuli of odorous stimuli and a central portion connected to the olfactory bulb without an intervening synapse in the receptor cells. When odorants reach the cleft, diffusion through a mucous layer covering the receptor cells occurs. Odorants are presented to the receptor cells via odorant-binding proteins. This results into activation of G proteins, and cyclic adenosine monophosphate—mediated depolarization of the olfactory neuron with subsequent action potential.

The signal is then transmitted along unmyelinated olfactory sensory neuron axons which make up cranial nerve I. Axons from the olfactory neurons form nerve bundles (filia olfactoria), cross the cribiform plate superiorly through approximately 20 foramina, and synapse with other neurons in the olfactory bulb. These second order neurons then transmit the signal to the piriform cortex, olfactory nucleus and tubercle, amygdala, and entorhinal cortex. Some inhaled chemicals can be detected by elements of the trigeminal nerve within the olfactory mucosa and throughout the nasal epithelium, as well as by afferents located in the back of the mouth and throat (e.g., via the glossopharyngeal and vagus nerves). However, these routes do not produce olfactory sensations and are beyond the scope of the present review.

Intranasal extensions of the olfactory epithelium extend about 1 cm inferiorly onto the nasal septum. From a sagittal perspective, the olfactory epithelium extends about 2 cm in length on both sides along the superior-posterior septum with potential extensions posteriorly to the face of the sphenoid sinus, and laterally to the superior and middle turbinates.[3,4]

Olfactory loss in chronic rhinosinusitis

Identification of specific etiology factors driving olfactory dysfunction in CRS is an evolving topic in the context of multiple phenotypes. However, it is widely recognized that loss is multifactorial with conductive and sensorineural mechanisms. Conductive loss may be seen in patients with structural pathologies preventing optimal transport of odorants to the olfactory cleft. These include nasal polyposis, mucosal edema, and nasal lesions.[7,8] Sensorineural loss may be mediated by inflammatory changes to the neurepithelium, as demonstrated in histologic studies and from response to corticosteroids.[9,10] Additional significant risk factors that predict presence of olfactory dysfunction in CRS patients include tobacco smoking, age over 65 years, and asthma.[11]

Multiple studies employing heterogenous methodologies have investigated the prevalence of olfactory dysfunction in CRS, with mean scores typically falling in the hyposmic range.[1,11,12] The wide range in reported prevalence reflects variability in CRS subtypes, where by those CRS patients with nasal polyps(CRSwNP) evidencing a higher level of olfactory impairment. Variability in testing may also contribute to the heterogeneity of observations. For example, shorter tests, such as the 12-item Brief Smell Identification Test (B-SIT), or subcomponents of larger tests, such as the threshold component of the Sniffin' Sticks test, may underestimate the degree of impairment across subgroups, although evidence for this is weak.[1,12–14] In a meta-analysis evaluating prevalence and patient specific factors, Kohli et al[12] noted that patients with CRSwNP had a higher degree of olfactory impairment at baseline than CRS patients with mixed phenotypes. In addition, worse scores from opacification of the olfactory cleft on computed tomography (CT) imaging, and eosinophilic CRS appear to be significant factors that predict olfactory impairment.[15–17] These findings and risk factors should be discussed when considering patients for ESS.

Olfactory outcomes following endoscopic sinus surgery

Improvement in quality of life of patients undergoing endoscopic sinus surgery is well documented.[18] However,

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