Olfaction in Endoscopic Sinus and Skull Base Surgery

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KEYWORDS

• Endoscopic sinus surgery • Anosmia • Hyposmia • Smell • Olfaction • Skull base

KEY POINTS

- Smell loss in chronic rhinosinusitis is caused by obstruction from polyps, nasal discharge, and mucosal edema, as well as inflammatory damage to the olfactory epithelium.
- The effect of endoscopic sinus surgery on olfaction is difficult to predict.
- Addressing olfaction with patients preoperatively is recommended before endoscopic sinus and skull base surgery.
- If surgery is required in the olfactory cleft, then meticulous dissection is recommended to optimize olfactory function postoperatively.

INTRODUCTION

Olfactory dysfunction is a common complaint for patients visiting otolaryngology offices, with chronic rhinosinusitis (CRS) accounting for 14% to 25% of patients.[1,2] Of patients with CRS, 28% to 84% complain of a decreased sense of smell.[3–5] Hyposmia decreases a person's enjoyment of food and can reduce their overall quality of life.[6–8] Furthermore, the inability to detect spoiled food, fire, toxic fumes, and gas leaks can be dangerous.[2] There are 2 mechanisms by which chronic rhinosinusitis decreases olfaction. First, the obstruction of the olfactory cleft from polyps, nasal discharge, and mucosal edema decreases the ability of the odorant to reach the olfactory receptors.[3,9] Second, underlying epithelial inflammatory damage from CRS can affect the health of the olfactory neurons or the neurons' ability to transmit olfaction to the brain.[9] Addressing olfaction with patients who have CRS is important, because a loss of smell is correlated with a lower quality of life.[6–8]

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Discussing olfaction is especially important in patients undergoing endoscopic sinus surgery (ESS) for CRS or endoscopic endonasal surgery for skull base tumors.[10,11] Without clear and consistent evidence that ESS improves smell, patients' postoperative expectations need to be addressed, which includes counseling that olfactory loss may persist or even worsen despite surgery and medical therapy.[4,12–14] Addressing olfaction is also important before endoscopic endonasal skull base surgery, because smell loss may occur during tumor resection or harvest of a large nasoseptal flap for reconstruction.[15–18] In a survey of practicing otolaryngologists, roughly 40% routinely discuss the potential for postoperative anosmia with patients.[11] This figure is important because 17% of malpractice litigation cases from ESS pertain to smell loss or complete anosmia.[10] This article reviews the anatomy and physiology of smell, presents options for olfactory testing that can be done in clinic, discusses surgical and medical considerations to optimize olfaction, and emphasizes the importance of including olfaction in the preoperative consent.

OLFACTION ANATOMY AND PHYSIOLOGY

The olfactory cleft is composed of pseudostratified columnar epithelium located below the cribriform plate and extending inferiorly along the septum for about 1 cm. Parasagittally, the olfactory epithelium is roughly 2 cm in length along the superoposterior septum and can extend posteriorly to the face of the sphenoid sinus and laterally to the upper portion of the superior and middle turbinates.[2,19] The olfactory epithelium contains bipolar neurons, which have ciliated dendrites that extend to the epithelial surface. The cilia have G protein–coupled and cyclic AMP–coupled receptors that are triggered by odorants. Once activated, the signal is propagated along the axons of the olfactory sensory neurons, which together form cranial nerve I. The axons pass through the cribriform plate and synapse in the olfactory bulb. Second-order neurons then send the olfactory signals to the amygdala and primary sensory cortex.[9] In addition to olfactory sensory neurons, the olfactory cleft epithelium contains a balance of supporting cells and mucus-secreting glands. Any disruption in the olfaction cascade of events can lead to smell dysfunction. In CRS, either the odorant is unable to reach the receptor or the signal transduction is ineffectively relayed to the brain because of inflammatory changes in the neuroepithelium.[9]

OLFACTORY TESTING

Olfactory testing has not been standardized in the literature, but testing measures are useful in documenting olfactory dysfunction in a patient complaining of smell loss. Testing can be subjective or objective. Formal subjective testing includes the 0 to 100-mm visual analog scale (VAS) or any specific item on a sinonasal-specific quality-of-life questionnaire such as item #5 on the Sinonasal Outcomes Test (SNOT)-22.[20,21] For the VAS, patients rate their sense of smell from anosmia (0 mm) to perfect olfaction (100 mm).[20] On the SNOT-22, smell is graded on a Likert scale from 0 (worst) to 5 (best).[21]

Objective olfactory testing includes threshold tests and identification tests.[22] The most common threshold test is the butanol threshold test (BTT; Sigma-Aldrich, St Louis, MO). This test includes a dilutional series of butanol, with the lowest butanol concentration tested first. The patient is given 2 solution bottles, 1 with diluted butanol and 1 with water or mineral oil, and is forced to choose which bottle has the odorant.[22–24] The smell threshold is determined when the bottle with the lowest butanol concentration is detected correctly 5 consecutive times.[14,22–24]