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Review Article

Thirty years of endoscopic sinus surgery: What have we learned?

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Abstract Prior to adaptation of endoscopic approaches for sinonasal pathology, patients regularly endured significant morbidity from open approaches to the sinonasal cavity that were often fraught with failure. With improvements in transnasal endoscopy, functional endoscopic sinus surgery subsequently emerged from the work of Messerklinger and other pioneers in the field. The popularity of endoscopic sinus surgery quickly escalated and expanded to pathology other than inflammation. Here, we discuss the evolution of endoscopic sinus surgery as it relates to improvements in understanding disease pathogenesis, improvements in instrumentation and expansion of indications.

History of endoscopic procedures

The first attempt at nasal endoscopy is largely credited to Hirschman in 1901. In this early work, a modified cystoscope was used to examine the sinonasal cavity.[1] Subsequently, Reichert performed what would be regarded as the first endoscopic procedure; rudimentary maxillary sinus manipulations with a 7 mm endoscope through an oroantral fistula.[2] In 1925, Maltz promoted use of nasal endoscopes for diagnostic evaluation of the sinonasal cavity and coined the term 'sinuscopy'.[3] The creation of the Hopkins rod system in the 1960s was perhaps the major turning point in the field of sinonasal endoscopy.

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Professor Harold H. Hopkins developed the rod optic endoscope system as well as several other inventions such as the zoom camera lens and fiberoptic gastroscope. The new telescope design resulted in markedly enhanced light delivery and superior optical quality allowing exceptional detailing of the sinonasal cavity.[4] Using this new innovation, Messerklinger subsequently composed a landmark book in 1978 on diagnostic endoscopy of the nose from his work studying mucociliary clearance in fresh cadavers.[5] Given the frequent failures of Caldwell-Luc surgery, the morbidity of frontal sinus osteoplasty and difficulties of performing headlight intranasal ethmoidectomy, there was a strong rationale for trying to improve surgical techniques for chronic sinusitis (CRS).

The relevance of the ostiomeatal complex (OMC) had been proposed by Naumann Proctor and Drettner, but it had previously not been adequately visualized, either on rhinoscopy or by plane film imaging. Messerklinger detailed the endoscopic anatomy and pathology of this region and also started to utilize polytomography to improve visualization of the anatomy and pathology. With improvements in imaging and endoscopic assessment, increasing emphasis was placed on anatomical aspects of the ostiomeatal complex and their potential impact on the pathogenesis of chronic rhinosinusitis. As scientific support for the importance of this region increased, several surgeons began performing select endoscopic procedures.

After having had the opportunity to meet Messerklinger during a conference in Dubrovnic, the senior author became convinced that endoscopic surgical diagnosis, more accurate imaging of the ostiomeatal complex and more functional surgery for CRS was truly an important step forward in its management. Given the very high radiation dose involved with polytomography, it was clear that new imaging methodologies needed to be developed. Zinreich et al[6] devised parameters which provided superior visualization of the OMC with computed tomography at lower radiation dosage. After gaining experience with endoscopic surgical techniques, the first endoscopic surgical course was held at Johns Hopkins Medical Center in 1985. Amid growth of the techniques followed the publication of landmark papers delineating the theory, diagnostic evaluation and technique of functional endoscopic sinus surgery (FESS) and subsequent animal experiments demonstrating the validity of the concept.[7–9]

Evolution in understanding the pathogenesis of sinonasal disease

These and other early publications rekindled interest in sinus disease and its management and subsequently resulted in the broad adoption of endoscopic diagnostic and surgical techniques worldwide. The focus of surgical intervention was on anatomic and inflammatory issues in the ostiomeatal complex and the re-establishment of mucociliary clearance and ventilation. However, we also recognized the importance of environmental factors, general host factors, such as immunodeficiency and certain genetic diseases, even during the early years of FESS (Table 1).

Since the introduction of FESS, we have learned significantly more about the underlying pathophysiologies of CRS but we still await a complete classification of this syndrome of disorders. Currently utilized classifications such as CRS with and without polyps are unsatisfactory in terms of fully classifying this broad spectrum of diseases. This makes detailed therapeutic recommendations or treatment outcome comparisons difficult. Classification systems are now moving into an era where genetic markers and cytokine profiles allow for more precise grouping of this syndrome of disorders. Recent evidence has shown high levels of endothelin-1 (ET-1), thymus and activation-regulated chemokine (TARC/CCL17), and alpha-defensins in CRS with nasal polyps. Neopterin levels have been found at higher concentration in patients with CRS without nasal polyps.[10,11] Additional literature has shown increased eosinophil production of prostaglandin D_2 in aspirin-exacerbated respiratory disease.[12] The pathophysiologic roles of these biomarkers in CRS are still yet to be determined.

Table 1 Factors associated with chronic sinusitis.

Environmental factors
Bacteria, viruses, fungi
Pollution, smoking
Allergens, chemical exposures
Host factors
Atopy
Immune deficiency
Genetic — Cystic fibrosis, ciliary dyskinesia, etc.
Innate immunity — bitter taste receptors
Local factors
Anatomic abnormalities
Inflammation of underlying bone
Obstructing tumors
Chronic mucosal inflammation

One important breakthrough has been the discovery of a new local immune system at least one element of which follows Mendelian genetics. Bitter taste receptors are expressed in the airway, where they appear to play several important roles in innate immune defense.[13,14] They are located on motile cilia in the upper airway and in response to bitter compounds and gram negative bacteria have been observed in sinonasal epithelium to result in increased nitric oxide production with potent bactericidal action.[13] Acyl-homoserine lactones (AHLs) are a subclass of lactones that can stimulate bitter taste receptors. AHLs are secreted by gram-negative bacteria and serve as biofilm quorum-sensing molecule.[15] Thus, once sufficient AHLs are produced in a localized environment, a biofilm may be produced. There is evidence that bitter taste receptors, through their lactone sensing mechanism, act as an adaptive response to detect biofilm quorum-sensing molecules and preemptively obviate biofilm formation and chronic inflammation. As such, it is postulated that host genetic defects in bitter taste receptors may predispose to CRS and early clinical studies support this concept. Knowledge surrounding this newly identified defense mechanism continues to evolve, with identification of the inhibitory action of sweet taste receptors and the potential for novel therapeutic targets. Defects in other components of innate