Medical and surgical management of nasal polyps

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Sinonasal polyposis represents a chronic inflammatory condition of unknown etiology. It is often associated with systemic diseases and is characterized by nasal obstruction, reduction in sense of smell, infection, and impaired quality of life. Endoscopy has enhanced the diagnosis and management of nasal polyps. The initial approach is medical management. Medical therapy consists of administration of intranasal steroids or a short course of systemic steroids. Other medical treatments considered are use of antibiotics, leukotriene modifiers, and acetylsalicylic acid avoidance. Surgical removal is performed for nonresponders to medical management. The purpose of surgery is to restore the nasal physiology by making the nose free from nasal polyps and allowing drainage of infected sinuses. With a computer-assisted navigation system and power instrumentation, surgical removal of polyps can be done more easily and accurately with fewer complications than previously. Medical therapy after surgery is essential for preventing recurrence. Curr Opin Otolaryngol Head Neck Surg 2001, 9:27–36

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Abbreviations

CT	computed tomography
ESS	endoscopic sinus surgery
FPND	fluticasone propionate nasal drops
GM-CSF	granulocyte-macrophage colony-stimulating factor
RANTES	regulated on activation, normal T cell expressed and secreted

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Nasal polyposis is an inflammatory chronic disease of the upper respiratory tract of unknown etiology. The prevalence varies from 1 to 5% [1]. Nasal polyps usually are manifested after the age of 20 years, with affected men outnumbering women two to one. The term *sinonasal polyp* usually refers to outgrowths of tissue into the nasal cavity. The most common site of origination of nasal polyps is the anterior ethmoid region (Table 1) [2]. Radenne *et al.* [3•] found that nasal polyps, besides causing nasal obstruction, hyposmia, and recurrent infection, impaired the quality of life more than did perennial allergic rhinitis. Whereas 71% of patients with polyps have asthma [4], there is now evidence that patients with nasal polyps are at an increased risk for the development of asthma [5]. Lamblin *et al.* [6•] found that patients with nasal polyps and asymptomatic bronchial hyperresponsiveness had an eosinophilic bronchial inflammation similar to that observed in asthmatic patients with nasal polyps, whereas patients with nasal polyps without bronchial hyperresponsiveness did not have eosinophilic lower airway inflammation. These data suggest that eosinophilic inflammation in patients with nasal polyps may precede the development of asthma.

Other conditions associated with nasal polyps include chronic rhinosinusitis, aspirin intolerance, and cystic fibrosis (Table 2). In one recent study, the prevalence of nasal polyps in 211 adult patients with cystic fibrosis was 37% [7].

Etiology

Several mechanisms have been proposed for the formation of nasal polyps. These include allergy, infection, autonomic imbalance, abnormal transepithelial ion transport, mucopolysaccharide abnormality, enzyme abnormality, mechanical obstruction, and epithelial rupture. Although a positive skin test for aeroallergens has commonly been associated with polyps, there has been little evidence for the implication of IgE-mediated allergy in their formation. In a recent study, high percentages (40%) of patients with severe nasal polyposis showed an immediate skin reaction to *Candida*, compared with controls (1%) [8]. Furthermore, another recent prospective study showed that 81% of nasal polyp patients had positive intradermal food test results, compared with 11% of controls [9]. Although these findings present associations, the relevance to the pathogenesis of nasal polyps is unknown.

Clinical as well as experimental studies indicate that nasal polyp formation and growth are activated and

Table 1. Origin of polyps in 200 consecutive patients

Site of origin	Patients, %
Uncinate, turbinate, infundibulum	80
Face of bulla, hiatus semilunaris, infundibulum	65
Frontal recess	48
Between bulla and middle turbinate	42
Inside bulla	30
Supra- and retrobullar recess	28
Posterior ethmoid (superior meatus)	27
Middle turbinate	15
Secondary sinuses affected	
Maxillary sinus (mucosal swelling)	65
Frontal sinus (mucosal swelling)	23
Sphenoid sinus	8

Data from Stammberger [2].

perpetuated by an integrated process of mucosal epithelium, matrix, and inflammatory cells, which, in turn, may be initiated by both infectious and noninfectious inflammation [10•]. This underlying pathology may lead to increased interstitial fluid pressure and obstruct blood flow in nasal polyps, resulting in edema and distension of stroma. If nasal polyps obstruct sinus drainage, subsequent infection can cause more venous stasis and mucosal edema, leading to a self-perpetuating cycle.

The inflammatory process in polyps continues to be examined. Expression of RANTES (regulated on activation, normal T cell expressed and secreted) and interleukin-5, cytokines with eosinophil chemotactic properties, is substantially increased in nasal polyps compared with normal mucosa, with no substantial differences in their expression between polyps from patients with and patients without allergies [11]. Both cytokines had a significant correlation between their expression and the number of either total or activated eosinophils. In addition, overproduction of eosinophil survival factors has been shown to delay apoptosis of eosinophils, resulting in tissue eosinophilia [12•]. Nakagawa *et al.* [13] found that edematous morphology, the infiltration of eosinophils, and the expression of fibronectin, one of the extracellular matrix proteins, were correlated with the size of nasal polyps. Their data suggest that interaction between eosinophils and fibronectin may play a role in edema formation, which contributes to the growth of nasal polyps. Furthermore, vascular endothelial growth factor was found to be more intensely expressed in nasal polyps than in control nasal mucosa [14]. This suggests that vascular endothelial growth factor could be involved in induction of angiogenesis and increased microvascular permeability.

Table 2. Conditions associated with nasal polyposis

Allergic and nonallergic rhinitis
Allergic fungal sinusitis
Aspirin intolerance (acetylsalicylic acid triad: nalsal polyps, asthma, aspirin intolerance)
Asthma
Churg–Strauss syndrome (fever, asthma, eosinophilic vasculitis, granuloma)
Cystic fibrosis
Immunodeficiency
Primary ciliary dyskinesia
Kartagener syndrome (chronic sinusitis, bronchiectasis, situs inversus)
Young syndrome (sinopulmonary disease, azoospermia, nasal polyps)

Building on the old observation that polyps lack neural elements, Gungor *et al.* [15] examined the capsaicin-induced levels of neuropeptides in nasal secretions of subjects with and subjects without nasal polyps. Subjects with nasal polyps responded poorly to capsaicin stimulation, suggesting depletion of neuropeptides.

Apoptosis (programmed cell death) mediated through the Fas/Fas-ligand system is essential in regulating immune function, developing organs, and conferring immune privilege. Fang and Yang [16] found overexpression of Fas-L protein on nasal polyps compared with nasal mucosa. Fas-L-positive cells were localized to the epithelial layers of cystically dilated glands and the ingrowing epithelium of nasal polyps. Fas-L may play an important role in the pathogenesis of polyps by prolonging cell survival.

There is evidence that persons carrying the HLA-DR7-DQA1*0201 and -DQB1*0202 haplotype have two to three times higher odds for developing nasal polyps than do controls [17•]. These results and the development of nasal polyps in genetically transmitted diseases such as cystic fibrosis and primary ciliary dyskinesia indicate that genetic etiology may play a role in the formation of nasal polyps.

In patients with aspirin intolerance, the arachidonic acid pathway appears to play a part, with markedly elevated peptidoleukotrienes being found in nasal polyps compared with adjacent normal mucosa of individuals with aspirin intolerance and of controls [18]. Nasal polyps of persons with aspirin intolerance show a substantially lower release of prostaglandin E_2 than does the normal mucosa of persons with aspirin intolerance and controls [18]. In addition, aspirin-intolerant patients showed elevated basal levels of peptidoleukotrienes and reduced basal levels of prostaglandin E_2 in isolated blood cells, compared with a healthy control [18,19]. Furthermore, inadequate cyclooxygenase-2 regulation may be involved in the pathogenesis of aspirin-intolerance, because cyclooxygenase-2 mRNA expression in nasal polyps from the aspirin-intolerant patient group was markedly and significantly lower than that in polyps from the aspirin-tolerant patient group and individuals with a healthy nasal mucosa [20]. These studies suggest that drugs that alter the arachidonic acid pathway may cause polyps in patients with aspirin intolerance.

Polyps seem to be a heterogeneous group of entities with a common end point. As our understanding of inflammation increases, we can better describe the inflammation within polyps. The main question is whether studying inflammation in polyps, an end stage of disease, will determine its cause or provide insights for therapeutic advances.

Histology

Polyps are covered by pseudostratified columnar epithelium with some areas of squamous metaplasia, basement membrane thickening, and a reduced number of mucous glands. The epithelium may contribute to increased mucus secretion [21]. Nasal polyps contain significantly more eosinophils, neutrophils, and plasma cells than does the nasal mucosa [22]. The presence of atopy or T-helper type 1 or 2 predilection does not determine either the type or the extent of cellular infiltration of nasal polyps. Flow cytometric analysis showed that there were no significant differences in the frequencies of lymphocytes and lymphocyte subsets (CD1+, CD2+, CD3+, CD5+, CD7+, CD4+, CD8+, CD10+, CD19+, CD20+, and HLA-DR+ cells), including CD4/8 ratios, between the nasal mucosa and polyps [22].

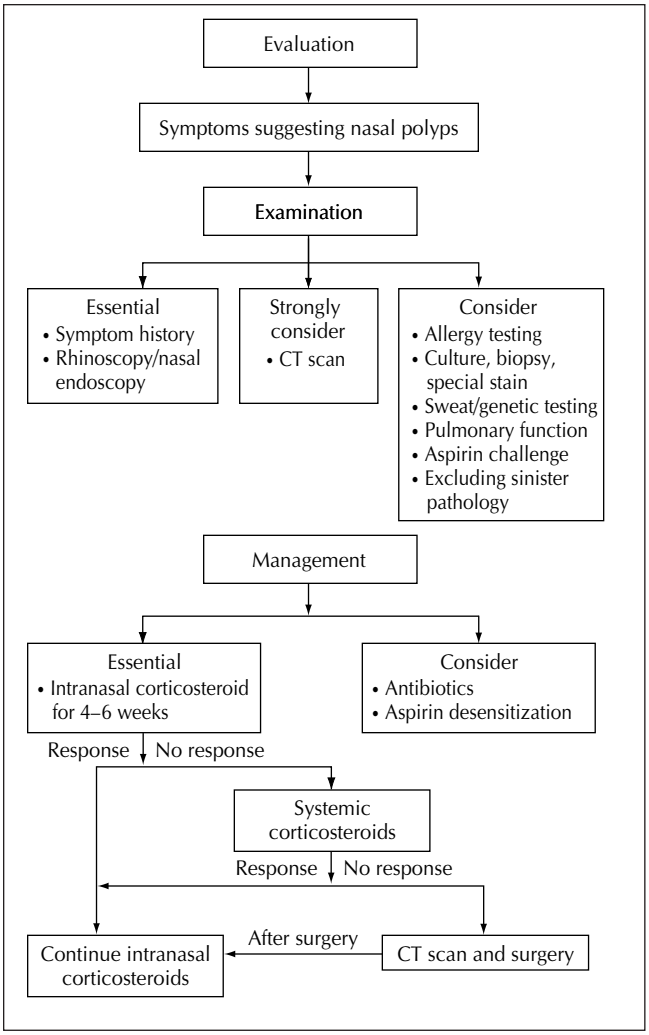
The mechanisms responsible for selective accumulation of eosinophils in polyps are unknown. Nasal polyp fibroblasts could play a role in the recruitment of eosinophils through the release of eotaxin [23], RANTES, and granulocyte-macrophage colony-stimulating factor (GM-CSF) [24]. Several cytokines (interleukin-4, -5, -6, and -8, tumor necrosis factor-α, GM-CSF, RANTES) have been shown to be upregulated in nasal polyps, suggesting that resident structural cells can produce a number of molecules to attract inflammatory cells and prolong their survival [11,25]. These inflammatory cells themselves can also produce cytokines such as interleukin-3, tumor necrosis factor-α, and GM-CSF, which recruit more inflammatory cells in an autocrine fashion [25]. Adhesion molecules have also been studied in nasal polyps, and it was found that vascular cell adhesion molecule is upregulated [26].

Evaluation of the patient

History and physical examination

The evaluation of nasal polyps begins with a history (Fig. 1). The most common symptom is nasal obstruction. Hypoxia, hypercapnia, snoring, sleep disorders, and an increased risk of hypertension may develop in patients with nasal polyposis [27]. Polyps may obstruct airflow to the olfactory cleft and lead to loss of the sense of smell. In addition, patients may have symptoms of sinus obstruction. A complete ear, nose, and throat examination should be performed with special focus on the nasal cavity. Nasal polyps are uncommon in children, and their presence should prompt evaluation for cystic fibrosis. A unilateral nasal polyp should raise the suspicion of an inverted papilloma or tumor in adults, or of dermoid cysts, encephaloceles, and gliomas in children. Examination of the oral cavity may show polyps behind the free margin of the soft palate in cases of antrochoanal polyps or postnasal drips that are related to coexistent infection.

Figure 1. Management plan for evaluation and management of nasal polyposis



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Nasal endoscopy

Nasal endoscopy provides excellent visualization of polyps, especially of small polyps in the middle meatus. It also shows nasal polyps originating from contact areas in middle meatus and nasal anatomic abnormalities. Culture of the discharge and a biopsy can be performed under endoscopic guidance. Cultures from the middle meatus or osteomeatal complex area have been shown to correlate with cultures obtained from within the sinuses [28].

Other diagnostic tests

Other investigations include allergy testing, a pulmonary function test, biopsies, a sweat chloride test or genetic testing for detection of cystic fibrosis, aspirin intolerance testing, and fungal stains and cultures (Fig. 1). Computed tomography (CT) scanning shows the extent of disease. CT scan is essential in cases of unilateral disease, failure of medical management, and when complications are suspected. CT scanning is best performed after medical management to delineate the chronic disease component. CT scanning is superior to magnetic resonance imaging in the depiction of bone details [29]. Magnetic resonance imaging is advisable when a skull base erosion is noted adjacent to an area of sinus opacity. It can differentiate between sinus disease eroding the skull base and a meningocele or encephalocele. Magnetic resonance imaging also helps to differentiate the tumor from retained secretions and secondary inflammatory disease.

Staging system

A staging system for rhinosinusitis allows comparison among published studies. Several radiography-based systems have been proposed. The most commonly used system is that described by Lund and Kennedy [30] and Mackay and Lund [31]. It has also been suggested that endoscopic appearance of the sinonasal cavity (Table 3) [31] and the patients' symptom scores should be recorded preoperatively and 3, 6, 12, and 24 months postoperatively [30].

Medical management

Nasal polyps are primarily diseases to be managed medically. Although some cases require surgery, aggressive medical therapy before and after surgery is needed. The aim of the treatment is to restore ventilation and sinus drainage as well as to prevent recurrence of the disease (Table 4) [32].

Antibiotics

Nasal polyps can cause obstruction of the sinuses, resulting in infection. Treating infection with antibiotics may prevent further polyp growth and lessen bleeding during surgery. Antibiotic therapy should be directed toward *Staphylococcus* species, *Streptococcus* species, and anaerobes, which are common organisms in chronic sinusitis. *Pseudomonas aeruginosa* may colonize the sinus cavities in patients who have cystic fibrosis and those who have had prior surgery. In immunocompromised hosts or those who have been on several prior courses of antibiotics, direct endoscopic culture or antral puncture is recommended to exclude unusual or resistant organisms. Interestingly, roxithromycin, a macrolide antibiotic, has been reported to inhibit fibrosis and prevent the progression of nasal polyposis [33].

Table 3. Grading of nasal polyps

Endoscopic appearance	Score
No polyps	0
Polyps restricted to middle meatus	1
Polyps below middle turbinate	2
Massive polyposis	3

Data from Mackay and Lund [31].

Table 4. Objectives of management of nasal polyposis

Reestablish nasal airways and nasal breathing
Minimize symptoms
Improve sense of smell
Treat coexisting diseases
Improve quality of life
Prevent complications

Data from Lildholdt and Mygind [32].

Corticosteroids

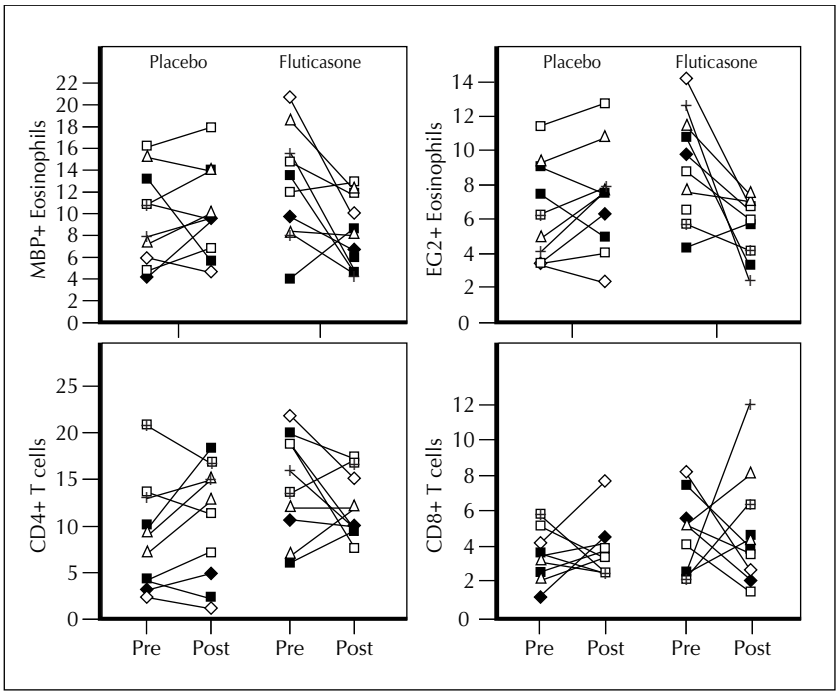
Mechanisms of corticosteroid treatment

Corticosteroids have a broad range of anti-inflammatory effects. Topical steroids have been shown to reduce the number of lymphocytes in nasal polyp tissue and to inhibit the synthesis of cytokines [34]. Topical steroids also reduce the total number and activation status of eosinophils [35]. Tingsgaard *et al.* [36] showed that topical treatment with budesonide substantially reduced the density of eosinophils and the endothelial vascular cell adhesion molecule expression in polyps. In a double-blind, placebo-controlled trial, Hamilos *et al.* [37•] studied the effect of 4-week treatment with intranasal fluticasone propionate on inflammatory parameters in nasal polyps. Fluticasone treatment significantly reduced the number of major basic protein (MBP)+ and EG2+ eosinophils, CD4+ T lymphocytes (Fig. 2), and interleukin-4 and -13 mRNA+ cells, and the expression of P-selectin. In contrast, fluticasone did not significantly reduce the expression of endothelial vascular cell adhesion molecule or the number of tumor necrosis factor-α or interleukin-1β mRNA+ cells in the polyps.

Jahnsen *et al.* [38] investigated the expression of chemokines in nasal polyps and found that mRNA expression for eotaxin, eotaxin-2, and monocyte-chemotactic protein-4 was significantly increased in nasal polyps compared with the turbinate mucosa in the same patients, or in control subjects. Polyp tissue had strong chemotactic activity for eosinophils. When patients were treated systemically with glucocorticosteroids, the mRNA level in the polyps was reduced to that found in the turbinate mucosa. Rudack *et al.* [39] investigated the effects of steroid (prednisolone) on eosinophils and their associated cytokines in nasal polyps *in vitro*. Prednisolone at concentrations of 10^{-3} to 10^{-2} mol/L significantly reduced the number of eosinophils, the total number of vital cells, GM-CSF, and interleukin-5 protein levels in

Figure 2. Changes in inflammatory cell infiltrate in nasal polyps after intranasal fluticasone versus placebo treatment for 4 weeks

Fluticasone treatment significantly reduced the number of major basic protein (MBP)+ eosinophils ($P = 0.02$), EG2+ eosinophils ($P = 0.007$), and CD4+ T cells ($P = 0.03$) compared with numbers before treatment. Pre, before treatment; Post, after treatment. Published with permission [37•].



supernatants, whereas interleukin-3 synthesis was not diminished. These data suggest an important role of cytokines in the pathogenesis of polyps.

Apoptosis is an important process that reduces the number of inflammatory cells and hence aids in the resolution of inflammation. In a double-blind, placebo-controlled test, Saunders *et al.* [40] studied the effect of treatment with fluticasone propionate aqueous nasal spray in nasal polyposis on indices of cell death and proliferation measured *in vivo*. They also studied the effect of dexamethasone at increasing doses on the same parameters *in vitro*. Corticosteroids induced apoptosis in inflammatory cells in human nasal polyps only *in vitro* but not *in vivo*. The difference in results probably relates to differences in the dose of steroid, in the experimental setting, in the drug distribution (topical *vs* systemic), or in metabolism *in vivo*. The association between induction of apoptosis and the regression of nasal polyps is unproved.

The refractoriness of symptoms and mucosal inflammation to oral steroids in patients with nasal polyps may indicate steroid resistance. Potential mechanisms for steroid resistance in nasal polyps may involve an alteration of the cellular response to steroids or overexpression of a glucocorticoid-resistant receptor (GRβ) [41].

Intranasal corticosteroids

Topical corticosteroids have been the drugs of choice for nasal polyposis (Fig. 1) [42]. If surgery is subsequently required, long-term postoperative treatment with corticosteroid nose sprays increases the time to recurrence. Some patients with nasal polyps do not respond to topical steroids. This may be due to two possible mechanisms. First, the underlying cause of nasal polyps, such as cystic fibrosis or primary ciliary dyskinesia, is unresponsive to corticosteroids. Second, nasal congestion by nasal polyps may cause an inadequate intranasal distribution of the topical steroid spray [43•]. Recently, application of steroid nasal drops has been proposed as an alternative delivery system. It is recommended that these be used in the head-inverted position (Moffit's position) to provide maximum local activity in the middle meatal area [44•]. The systemic bioavailability of fluticasone nasal drops is lower than that of nasal spray (0.06 *vs* 0.5%) [45]. However, one study showed that 6-week treatment with recommended doses of betamethasone topical nasal drops in 11 patients with nasal polyposis had systemic corticosteroid activity and suppressed the hypothalamo-pituitary-adrenal axis [46]. Local intralesional injection of steroids is also effective, but rare complication of visual loss can occur.

Systemic corticosteroids

Short-term treatment with systemic corticosteroids is an alternative method of inducing remission and controlling nasal polyps. In experimental sinusitis in rabbits, intramuscular injection of steroid inhibited polyp formation and growth of pathogenic bacteria in the sinuses [10•]. In contrast to nasal steroids, systemic corticosteroids can

reach all parts of the nose and sinuses, including the olfactory cleft and middle meatus, and improve the sense of smell better than topical steroids [32]. Additionally, short courses of systemic steroids can be used for nasal polyposis to open up nasal obstruction before therapy with intranasal steroids, which results in improvement of the intranasal spray distribution. Long-term treatment with a low daily dose of oral steroids and intranasal steroids in patients with aspirin intolerance and allergic fungal sinusitis may be necessary.

Before surgery, oral steroids are typically given for about 3 or 4 days to shrink the polyps. Oral steroids are also beneficial in asthmatic patients by reducing the bronchial hyperreactivity, which can be exacerbated by surgery. Care should be taken in administering oral steroids to patients with diabetes mellitus, psychiatric disorders, herpes keratitis, advanced osteoporosis, tuberculosis, glaucoma, and hypertension.

Other medications

The use of antihistamines and decongestants may provide symptomatic relief but does not change the course of the disease. Immunotherapy has been shown to be beneficial in patients with allergic fungal sinusitis [47] and may be useful in patients with recurrent polyposis. Leukotriene antagonists may provide benefits in some patients who have aspirin intolerance [48•].

Surgical management

Surgical removal of nasal polyps is indicated for patients not responding adequately to medical management, those with continued or recurrent infections, as well as patients who are developing mucoceles or other complications of sinusitis. Patients with polyps and asthma may benefit from surgery by reduction of one trigger for asthma. All involved sinuses should be opened, in addition to the removal of polyps.

Correction of outflow obstruction promotes drainage and leads to reversal of mucosal changes within the paranasal sinuses. Denudation of bone, especially in the areas of the maxillary and frontal ostia, should be avoided because it may lead to granulation and osteitis, with prolonged postoperative healing or stenosis.

Recent advances in endoscopic surgery involve computer-assisted navigation and power instrumentation [49,50]. Patients with extensive polyp disease and those undergoing revision surgery may benefit from image-guided surgery because the thoroughness of the surgery may be improved and complications may be reduced [49]. Powered instrumentation with microdebriders minimizes inadvertent mucosal trauma and stripping [51]. The microdebrider can be used for removing nasal polyps or other tumors in the sinonasal cavity without altering the specimen for histopathology [52]. In 40 cases of endoscopic sinus surgery (ESS) performed with the microdebrider, patients who had at least a 5-month follow-up showed rapid mucosal healing, minimal crust formation, and a low incidence of synechiae formation [53].

The most important goals of follow-up care are prevention of synechiae and obstruction of ostia; restoration of patency of the nasal and sinus cavity; prevention of persistent inflammation, infection, and further polyp growth; and stimulation of the development of normal mucosa to replace the diseased tissue. Endoscopic examination of the sinonasal cavity postoperatively has been shown to provide prognostic information, which is independent of the subjective reporting of symptoms by patients [54]. Patients with abnormal endoscopic findings tend to have recurrent symptoms and a need for additional surgery in the future [54]. Postoperative treatment with intranasal steroids is required because it helps to slow the rate of recurrence of polyps. Antibiotics are administered during acute healing. Small recurrent polyps may be removed endoscopically or by prescription of a short course of oral steroids. Some groups of patients, *ie*, those with aspirin intolerance, allergic fungal sinusitis, asthma, or cystic fibrosis, require aggressive and extensive treatment and follow-up because of a high recurrence rate.

Results of medical and surgical therapy for nasal polyps

Intranasal corticosteroids

Topical steroid therapy has been shown, in patients with nasal polyps, to improve nasal blockage [55–57], nasal patency as measured by nasal peak flow [55,56,58], nasal volume as measured by acoustic rhinometry [56], and sometimes the sense of smell, and to prevent or delay the recurrence of nasal polyps after surgery [55,58].

Steroid nasal drops have also been shown to be effective in the management of nasal polyps [59,60]. In a double-blind, placebo-controlled, multicenter study, Penttila *et al.* [61•] evaluated the dose-related efficacy and tolerance of fluticasone propionate nasal drops (FPND) in the management of mild to moderate bilateral polyposis. FPND at 400 µg twice daily significantly reduced polyp size and nasal blockage (Fig. 3) and improved the peak nasal inspiratory flow and sense of smell. Significant reductions in polyp size were not achieved with once-daily administration, but clinical benefits were observed for peak nasal inspiratory flow and nasal blockage and for overall rhinitis symptoms (Fig. 3). FPND, 400 µg twice daily, was significantly more effective than 400 µg once daily. Both dosing regimens were well tolerated, without any significant effect on mean serum cortisol levels, and the overall incidence of adverse events was similar to that for placebo.

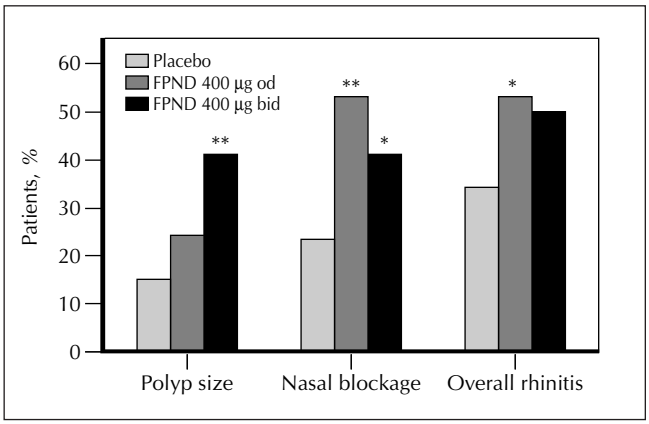
Systemic steroids

Systemic steroids reduce the frequency of all symptoms and improve the sense of smell [62,63]. Damm *et al.* [64] evaluated the efficacy of a combined (intranasal and systemic) form of steroid therapy. A nasal budesonide spray (0.2 mg/d) and an oral fluocortolone medication with a daily reduction during a 12-day period (total dose, 560 mg) and a 20-day period (total dose, 715 mg), respectively, were administered. The combined short-term steroid therapy significantly reduced the extent of chronic polypoid rhinosinusitis on magnetic resonance imaging (> 30%) in 50% of patients and diminished most sinusitis-related symptoms in 80% of patients. The steroid effect on polypoid masses was heterogeneous in different anatomic areas, with the best reduction of inflamed mucosa in the sphenoid, frontal, and maxillary sinuses. This effect, however, was not seen in the anterior ethmoid area, a key area for the development of polyps.

Leukotriene antagonist

Parnes and Chuma [65] evaluated the efficacy of a leukotriene synthesis inhibitor (zileuton) and a leukotriene receptor antagonist (zafirlukast) in controlling sinonasal polyposis and its associated symptoms in 40 patients with sinonasal polyposis and sinusitis, without any change in their standard therapy. Overall, 72% of patients experienced subjective improvement after starting their medication. Objective alleviation, or at least stabilization, of sinonasal polyposis was seen in 50% of the patients. However, this was a preliminary study without a control group. Ulualp *et al.* [66] retrospectively studied the effect of antileukotriene therapy for the relief of sinus symptoms in patients with aspirin-triad disease who had undergone previous sinus surgery. Fifty percent of patients had improved symptom scores after antileukotriene therapy; 17% of patients reported overall benefit from therapy, despite no improvement in their symptom scores. Findings on endoscopic nasal examination were consistent with the reports of an overall benefit. These data suggest that antileukotriene therapy may have benefits in patients with sinonasal polyposis. However, further double-blind, placebo-controlled studies are required for defining the role of leukotriene- modifying agents.

Figure 3. Percentages of patients showing improvements in parameters specified on the abscissa after 12 weeks of treatment with fluticasone propionate nasal drops



Four hundred micrograms once a day (od) ($n = 48$), fluticasone propionate nasal drops 400 µg twice daily (bid) ($n = 47$), or placebo ($n = 47$). *$P < 0.05$, †$P < 0.01$ vs placebo. Adapted with permission [61•].

Surgery

Several authors reported the favorable outcome of ESS in patients with chronic sinusitis or nasal polyps in both short-term (average, 6–23 months) [67–69] and long-term (average, 3.7–7.8 years) follow-up [70,71]. More than 80% of patients reported marked subjective improvement in their symptoms. Sharp *et al.* [72] showed a significant correlation between outcome of ESS at 2 years and the preoperative CT scan score (Lund and Mackay system), but the statistically most significant factor determining the success or failure of surgery was the presence of a systemic disease known to predispose to chronic rhinosinusitis. Mostafa *et al.* [73] retrospectively analyzed the records of 100 patients with nasal polyposis and studied various parameters including radiologic, intraoperative, and bacteriologic data. They found that, among the various parameters studied, maxillary antral involvement of nasal polyps and positive bacterial cultures seemed to be the most predictive criteria of recurrence.

Radenne *et al.* [3•] evaluated the effect of management of nasal polyps on the quality of life in 28 patients with nasal polyps. The results demonstrated that nasal polyp treatment with either nasal steroids or endonasal ethmoidectomy significantly improved nasal symptoms and quality of life. The use of quality-of-life measures as a new tool for the evaluation of treatment efficiency in chronic sinusitis and nasal polyps has been encouraged [74].

Endoscopic sinus surgery has also been shown to lead to significant improvement in total nasal resistance as measured with rhinomanometry [75], in nasal volume by acoustic rhinometry [76], in ciliary beat frequency [77,78], in mucociliary clearance as measured by saccharin test [78–80], and in olfaction as measured by University of Pennsylvania Smell Identification Test (UPSIT) testing [76,77,81,82].

There is also increasing evidence that management of polyposis has a benefit for the lower airway. Senior *et al.* [83] assessed the long-term impact of ESS in patients with chronic rhinosinusitis and asthma at an average follow-up of 6.5 years. Of 30 patients with asthma and sinusitis, 27 (90%) reported that their asthma was less severe than it had been before ESS. The average rate of reported improvement increased from 49% at 1.1 years after surgery to 65% at 6.5 years after surgery. Asthma

attacks declined in 20 of 27 patients (74.1%). Approximately half reported less inhaler usage, and nearly two thirds reported less oral steroid use. Other studies [84,85] also showed favorable results of ESS in the management of asthma.

The aspirin triad (nasal polyposis, asthma, and sensitivity to aspirin) is a well-recognized clinical entity. Amar *et al.* [86] made an outcome analysis retrospectively in acetylsalicylic acid triad patients who had undergone ESS. The control group consisted of 22 patients with chronic sinusitis, with or without asthma, who had also undergone ESS. Acetylsalicylic acid triad patients had more extensive involvement of the sinuses radiologically. Furthermore, acetylsalicylic acid triad patients underwent a larger number of repeat operations, suggesting that these patients respond less well to surgical intervention. Nakamura *et al.* [87] evaluated the surgical management of sinusitis in 22 patients with aspirin-induced asthma. Sinus surgery reduced asthma symptoms in 20 patients (90.9%) and improved the pulmonary function test 1 year after surgery. Three of five patients (60%) who used systemic steroids were able to eliminate or reduce their doses, and eight of 17 patients (47.1%) who were using inhaled topical steroids reduced their doses. These results strengthen the beneficial effect of sinus surgery on asthma symptoms even in patients with aspirin-induced asthma.

Conclusions

Sinonasal polyps represent a diffuse inflammatory process that probably has multiple causes. The management of nasal polyps remains primarily medical, with oral and topical nasal steroids. Systemic steroids can reduce rhinitis symptoms, improve the intranasal distribution of topical steroids and the sense of smell, facilitate nasal surgery, prevent the recurrence of polyps after polypectomy, and can be used as a substitute for simple polypectomy. Topical corticosteroids have been shown to reduce polyp size and rhinitis symptoms as well as to delay the recurrence of polyps after surgery. Topical steroids can be combined with systemic corticosteroids in severe cases. Nasal drop formulations of corticosteroids may improve their efficacy by providing a better drug distribution and penetration into the osteomeatal area as compared with nasal sprays. Other medical therapies such as the use of leukotriene antagonists may be helpful but need further study. Surgery is indicated in patients in whom medical management fails or who have complications. The postoperative care needs to be intensive so that recurrence is delayed.

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Papers of particular interest, published within the annual period of review, have been highlighted as:

- • Of special interest
- •• Of outstanding interest

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