Nasal polyps: an update

Patients with nasal polyps suffer from a condition that, despite adequate treatment, runs a recurrent and remitting course, requiring long-term medication and often multiple operations. It is a condition in which the cause remains unknown.

Nasal polyps are formed as the end result of severe inflammation in the nose and paranasal sinuses and are considered a subgroup of chronic rhinosinusitis. Despite many theories and extensive research, the cause of nasal polyps remains unknown. Many factors are associated with nasal polyps, but no causative link has been found. Patients with nasal polyps suffer from a disease that is on the increase, carries significant financial burden on society (both directly as a result of outpatient appointments, prescriptions, radiography and hospitalization, and indirectly as a result of missed work days and decreased productivity at work), and which, despite adequate treatment, runs a chronic and recurrent course.

Definition

The European Position Paper on Rhinosinusitis and Nasal Polyps defines these conditions clinically as inflammation of the nose and paranasal sinuses, associated with two or more of: blockage or congestion, discharge, facial pain or pressure, reduction in smell, and either endoscopic evidence of polyps, mucopurulent discharge from the middle meatus or oedema, mucosal obstruction primarily in the middle meatus and/or mucosal changes within the osteomeatal complex or sinuses on computed tomography imaging (Fokkens et al, 2007).

Incidence and prevalence

In the absence of systemic disease the annual incidence of nasal polyps is between one and 20 per 1000 population (Johansson et al, 2003). This declines after 60 years of age. In the normal population the prevalence is about 1% in adults and 0.1% in children. Nasal polyps are more common in males (2–4:1), and there is no racial predilection. Certain systemic diseases, however, carry a much higher incidence of nasal polyps. Of patients with cystic fibrosis 3–48% have nasal polyps, 27% of patients with Kartagener's syndrome and 80% of patients with fungal sinusitis have nasal polyps (Hedman et al, 1999). Nasal polyps are seen in 20–50% of patients with asthma, subdivided into 13% non-allergic, 8% associated with aspirin sensitivity and 5% with atopy (Hedman et al, 1999).

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Histology

Polyps arise in the presence of inflammation. They consist of loose connective tissue, inflammatory cells and fluid, and are usually covered by pseudostratified, columnar, ciliated epithelium. Polyps form when oedematous connective tissue stroma ruptures and herniates through the basement membrane (Berger et al, 2002). In small polyps with normal adjacent nasal mucosa there is a marked eosinophilic infiltrate, with no increase in the number of mast cells, whereas mature polyps contain degranulated mast cells and eosinophils diffusely distributed throughout the polyp tissue (Bachert et al, 2000). The number of neutrophils, activated T cells and plasma cells is increased compared with normal nasal mucosa. Other mediators can be found in increased levels within the tissue of nasal polyps, including cytokines, adhesion molecules and certain growth factors (Stoop et al, 1993), but their role in the development and subsequent behaviour of nasal polyps is not fully understood.

Symptoms

Patients with nasal polyps present with various symptoms that may have persisted over months or years. The manifestation depends on the burden of polyp disease. Small polyps may not produce symptoms and may be identified incidentally during rhinoscopic examination. Other small polyps may cause chronic or recurrent acute sinusitis symptoms if they block the outflow tract of the sinuses, for example, at the middle meatus. Larger polyps can cause nasal obstruction, rhinorrhoea, post-nasal discharge, conductive hyposmia or anosmia and headaches. Epistaxis tends not to be associated with benign multiple polyps and may suggest a more serious pathology in the nasal cavities. Massive polyps or a single large polyp that obstructs the nasal cavities or nasopharynx can cause chronic mouth breathing and obstructive sleep symptoms. Rarely, proptosis, hypertelorism and diplopia can result from alterations in the craniofacial structure. Generally, because of their slow-growing nature, massive polyposes do not cause neurological symptoms, even if they extend into the intracranial cavity (Andrews et al, 2005).

Diagnosis

Anterior rhinoscopy is the first step to diagnosis, but is inadequate alone and cannot exclude polyps. Rigid nasendoscopy after topical decongestant is needed for diagnosis, as well as identifying discharge, crusting or scarring secondary to previous surgery. Plain sinus radiographs are insensitive (Jonas and Mann, 1976; McAlister et al, 1989;

Iinuma et al, 1994). Computed tomography scanning with coronal sections is the investigation of choice for assessing the extent of the disease and detailing the anatomy before surgery (Erdem et al, 2004; Arikan et al, 2005; Kazkayashi et al, 2005). Magnetic resonance imaging may be helpful to differentiate from tumour, or if intracranial extension of disease is suspected. History for features of systemic disease should be taken, including lower airway symptoms, and tests for factors associated with nasal polyps can be performed (*Table 1*). The effects of treatment on nasal obstruction and polyp masses can be documented objectively via peak nasal inspiratory flow (Ottaviano et al, 2006), rhinomanometry (Lund and Scadding, 1994), acoustic rhinometry (Ragab et al, 2004) and smell tests.

Aetiology

The cause of nasal polyps remains unknown. The development of polyps has been linked to chronic inflammation, autonomic nervous system dysfunction and genetic predisposition (Tos et al, 1992; Pawliczak et al, 2005). As polyps are considered the ultimate manifestation of chronic inflammation, conditions that lead to chronic inflammation in the nasal cavities can lead to nasal polyps. No clear evidence exists for an allergic origin, although there is an established association with asthma and aspirin sensitivity (Bernstein et al, 1995; Mygind et al, 2000). Nasal polyps are also seen in cystic fibrosis, Churg–Strauss syndrome and primary ciliary dyskinesia (Kartagener's syndrome). There is growing evidence that microbes, particularly fungal colonization of the nasal passages, may play a role in the development of chronic rhinosinusitis. Regardless of the cause, the increased presence of inflammatory mediators is prominent and consistent in nasal polyps.

Allergy

Only 0.4–4.5% of patients with allergic rhinitis have nasal polyps, however, the prevalence of allergy in patients with nasal polyps varies from 10% (Delaney, 1976) to 64% (English, 1985). There is an association with increased levels of both total and specific immunoglobulin E and eosinophilic infiltration in nasal polyps (Bachert et al, 2001).

Asthma

Of patients with nasal polyps, 26% have diagnosed asthma, and as many as 42% have respiratory symptoms (Klossek et al, 2005); 7% of patients with asthma have nasal polyps (Settipane and Chafee, 1977). Of patients with late-onset asthma, 10–15% develop nasal polyps. Generally patients develop asthma first, then polyps within 9–13 years. This occurs faster in patients with aspirin-induced asthma, about 2 years from the onset of symptoms (Szczeklik et al, 2000).

Aspirin sensitivity

Of patients with aspirin sensitivity 36–96% have nasal polyps (Caplin et al, 1971; Settipane, 1997). The prevalence of patients with nasal polyps, asthma and aspirin sensitivity increases over 40 years of age, and these patients are normally non-atopic. HLA A1/B8 has a higher incidence in patients with asthma and aspirin sensitivity (Moloney and Oliver, 1980), and immunoglobulin E antibodies to enterotoxins can be found in most patients with polyps who are aspirin sensitive (Zhang et al, 2005).

Bacteria

A link between bacterial colonization and nasal polyps has been postulated, but remains unproven. Most bacterial organisms which have been isolated are aerobic, with *Staphylococcus aureus*, coagulase-negative Staphylococcus and *Streptococcus pneumoniae* most common (Araujo et al, 2003). As the disease progresses it is suggested that aerobic bacteria are replaced by anaerobic organisms (Brook, 1989, 1996). *S. aureus* superantigens bind to T cell receptors, and these are present in the lymphocytes of 35% of patients with polyps (Conley et al, 2006).

Fungi

A wide variety of fungi have been isolated from the sinuses of patients with chronic rhinosinusitis (Schubert, 2001). However, there is no evidence to suggest that fungi cause, or indeed worsen, the disease, and the use of topical and systemic antifungals has not been shown to consistently help patients with chronic rhinosinusitis (Ebbens et al, 2006; Weschta et al, 2006).

Classification

Nasal polyps can be subdivided into different groups depending on aetiology, histopathology and more recently mediator content. Mediator content is classified as:

Table 1. Specific investigations in patients with nasal polyps

Associated factor	Test
Allergy	Skin prick tests
	Specific immunoglobulin E
Immunodeficiency	Immunoglobulin subclasses
Chronic infection	Histology
	Culture
Vasculitis	Full blood count
	C-reactive protein
Granulomatous disease	Angiotensin-converting enzyme
Autoimmune disease	Anti-neutrophilic cytoplasmic antibodies
Aspirin sensitivity	Inhaled or nasal lysine-aspirin challenge
	Oral aspirin challenge
	Substantial history
Allergic fungal sinusitis	Skin prick tests
	Aspergillus-specific immunoglobulin G and E
	Computed tomography changes
	Histology

1. Idiopathic, unilateral or bilateral, mainly eosinophilic polyps without involvement of the lower airways
2. Bilateral eosinophilic polyps with concomitant asthma and/or aspirin sensitivity
3. Polyposis with underlying systemic disease (e.g. cystic fibrosis).

Endoscopic and computed tomography-based staging systems are used to determine the extent of disease within the nose and sinuses, and facilitate both medical communication and evaluation of therapeutic responses.

The clinical staging system and endoscopic scoring systems are based on the assumption that the polyp grows from the middle meatus down towards the floor of the nose (Lund and Mackay, 1993; Lildholt et al, 1995; Johansson et al, 2000) (*Tables 2* and *3; Figures 1* and *2*). The radiological staging system includes all sinuses and the osteomeatal complex bilaterally. Various radiological staging systems have been described. The Lund–Mackay system gives a score of 0–2 depending on the absence, partial or complete opacification of each sinus system and of the osteomeatal complex on computed tomography scanning. A maximum score of 12 per side can be achieved (Lund and Mackay, 1993) (*Table 4; Figure 3*).

Table 2. Endoscopic appearance scores

Characteristic	Baseline and follow up
Polyp * , left (0, 1, 2, 3)	
Polyp *, right (0, 1, 2, 3)	
Oedema †, left (0, 1, 2)	
Oedema †, right (0, 1, 2)	
Discharge ‡, left (0, 1, 2)	
Discharge ‡, right (0, 1, 2)	
Postoperative scores to be used for outcome assessment only	
Scarring §, left (0, 1, 2)	
Scarring §, right (0, 1, 2)	
Crusting ¶, left (0, 1, 2)	
Crusting ¶, right (0, 1, 2)	
Total points	

* 0 = absence of polyps, 1 = polyps in middle meatus only, 2 = polyps beyond middle meatus but not blocking the nose completely, 3 = polyps completely obstructing the nose; † 0 = absent, 1 = mild, 2 = severe; ‡ 0 = no discharge, 1 = clear, thin discharge, 2 = thick, purulent discharge; § 0 = absent, 1 = mild, 2 = severe; ¶ 0 = absent, 1 = mild, 2 = severe. From Fokkens et al (2007)

Table 3. Endoscopic staging system for nasal polyposis

Score		Right	Left
0	No polyps present		
1	Polyps confined to middle meatus		
2	Polyps beyond middle meatus		
3	Polyps almost or completely obstructing nasal cavity		

Modified from Lund and Mackay (1993)

Treatment

Treatment aims to eliminate nasal polyps and rhinitic symptoms, re-establish nasal breathing and olfaction, and prevent recurrence. Treatment can be divided into medical and surgical. All patients should have a trial of medical therapy first, unless histology is required. Medical treatment consists mainly of topical and systemic glucocorticosteroids, which are thought to affect eosinophil function directly by reducing both eosinophil viability and activation (Xaubet et al, 1994), and indirectly by reducing the secretion of chemotactic cytokines by nasal mucosa and polyp epithelial cells (Mullol et al, 1995).

Betamethasone drops are relatively potent topical steroids, and may be used initially, although not for longer than 2 months at one time unless under specialist supervision, or for longer than 4 months in one 12-month period, as they can be absorbed systemically. Larger polyps may require a medical polypectomy, which involves prednisolone 0.5 mg/kg each morning for 5–10 days.

Figure 1. Stage 1 polyps in right nasal cavity: polyp is confined to the middle meatus.

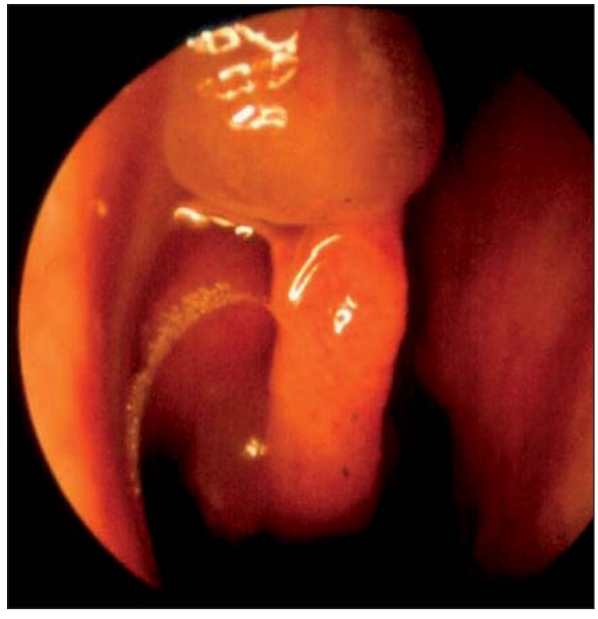
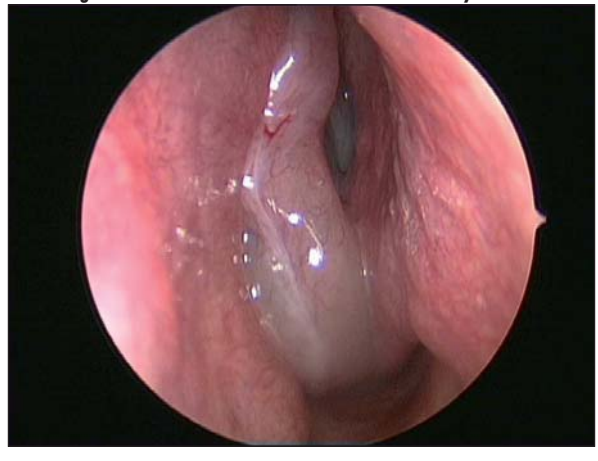


Figure 2. Stage 2 polyps in left nasal cavity: polyp can be seen extending from the middle meatus into the nasal cavity.



Maintenance therapy with mometasone spray or fluticasone drops or spray is recommended as these have lower bioavailability than betamethasone.

Topical corticosteroid sprays are effective for bilateral nasal polyps and symptoms such as nasal obstruction, secretion and sneezing, but the effect on sense of smell is not high. Nasal drops are more effective than sprays and have a significant positive effect on sense of smell.

It is recommended that patients are started on either sprays, drops or systemic steroids depending on their initial presentation and examination findings. On review, a decision can be made whether to continue with medical treatment, request further investigations or consider surgical intervention (Fokkens et al, 2007) (*Figure 4*).

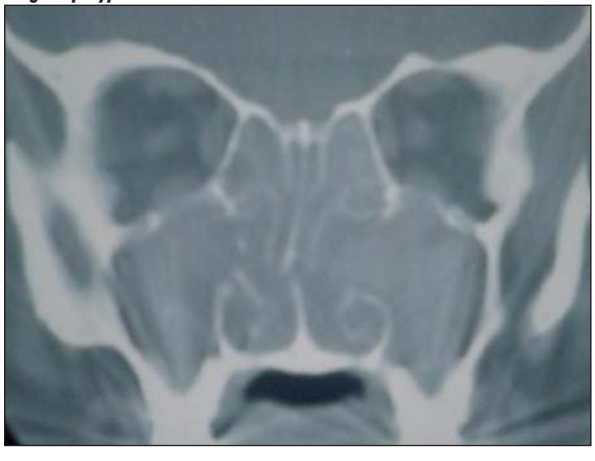
Antihistamines only help if allergy is present (Bernstein et al, 1995), and leukotriene inhibitors may help patients with co-existing asthma and/or aspirin sensitivity (Blomqvist et al, 2001; Bachert et al, 2003; Gevaert et al, 2006). Nasal douching provides symptomatic benefit. There is evidence that a 3-month course of a macrolide antibiotic can help nasal polyps (Cervin, 2001). Macrolides have been shown to increase mucociliary transport, reduce goblet cell secretion and cause accelerated apoptosis of neutrophils, all of which would reduce the symptoms of chronic inflammation.

Table 4. Computed tomography scoring system

Sinus system	Left	Right
Maxillary (0, 1, 2)		
Anterior ethmoids (0, 1, 2)		
Posterior ethmoids (0, 1, 2)		
Sphenoid (0, 1, 2)		
Frontal (0, 1, 2)		
Osteomeatal complex (0 or 2 only)		
Total points		

From Lund and Mackay (1993)

Figure 3. Lund–Mackay maximum score of 24. Computed tomography scan shows pansinus involvement. Clinically this patient would score stage 3 polyps.



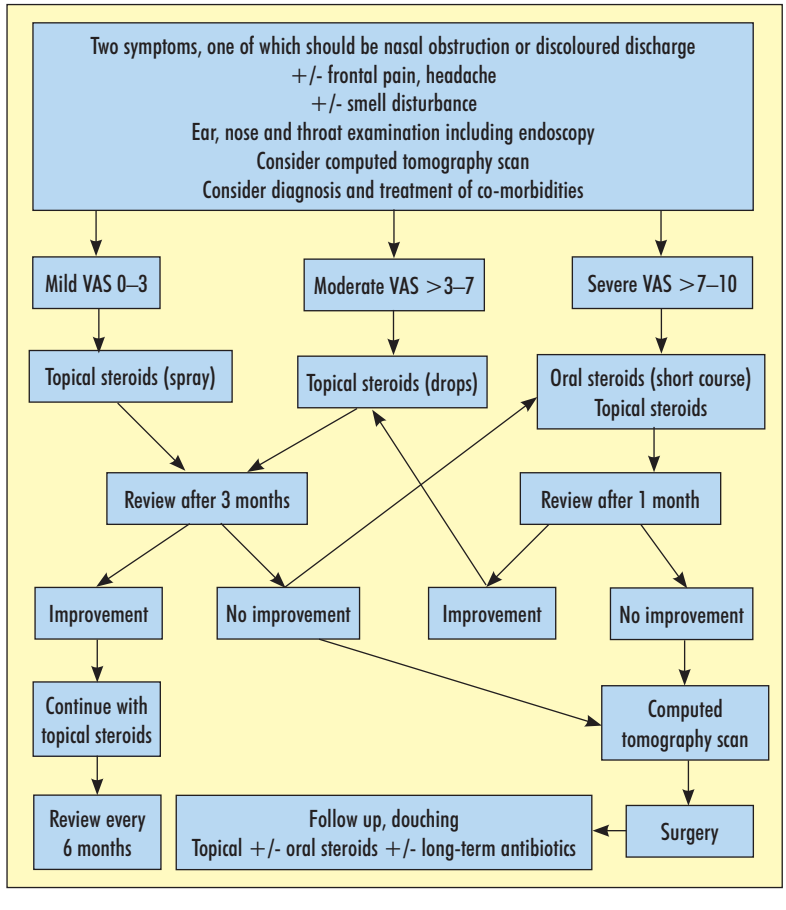
Functional endoscopic sinus surgery aims to improve sinus ventilation and drainage as well as removing polyps. The extent of surgery varies with the extent of disease, the surgeon's individual practice and technology. In the last 10 years functional endoscopic sinus surgery has been used to treat sino-nasal conditions and permits a better view of the surgical field, allowing more precise clearance of the inflammatory change than conventional surgery. It may be associated with fewer complications and lower recurrence rates (Lund, 2001; Khalil and Nunez, 2006).

Patients being treated for polyp disease derive the greatest benefit from functional endoscopic sinus surgery, and those whose main preoperative symptom is nasal obstruction or headache report higher benefit. Co-existent asthma, allergic rhinitis or aspirin intolerance does not decrease the benefit of sinus surgery in patients with polyps (Mehanna et al, 1996). Postoperatively these patients should be treated with nasal douching, topical and/or systemic steroids, and/or long-term antibiotics.

Conclusions

Nasal polyps arise in patients with chronic inflammation resulting from many different pathologies, and why certain patients develop polyps remains unknown. Extensive research has resulted in a greater understanding of this disease, but has not revealed a cause. A combined treat-

Figure 4. Treatment algorithm for patients with nasal polyps. From Fokkens et al (2007). VAS = total severity visual analogue scale. VAS > 5 affects quality of life.



ment strategy involving both medical and surgical management is recommended for long-term control because of the chronic and recurrent nature of the disease. BJHM

Conflict of interest: none.

Andrews AE, Bryson JM, Rowe-Jones JM (2005) Site of origin of nasal polyps: relevance to pathogenesis and management. *Rhinology* **43**(3): 180–4

Araujo E, Palombini BC, Cantarelli V, Pereira A, Mariante Al (2003) Microbiology of middle meatus in chronic rhinosinusitis. *Am J Rhinol* **17**(1): 9–15

Arikan OK, Unal B, Kazkayasi M, Koc C (2005) The analysis of anterior skull base from two different perspectives: coronal and reconstructed sagittal computed tomography. *Rhinology* **43**(2): 115–20

Bachert C, Gevaert P, Holtappels G, Cuvelier C, van Cauwenberge P (2000) Nasal polyposis: from cytokines to growth. *Am J Rhinol* **14**(5): 279–90

Bachert C, Gevaert P, Holtappels G, Johansson SG, van Cauwenberge P (2001) Total and specific IgE in nasal polyps is related to local eosinophilic inflammation. *J Allergy Clin Immunol* **107**(4): 607–14

Bachert C, Hormann K, Mosges R et al (2003) An update on the diagnosis and treatment of sinusitis and nasal polyposis. *Allergy* **58:** 176–91

Berger G, Kattan A, Bernheim J, Ophir D (2002) Polypoid mucosa with eosinophilia and glandular hyperplasia in chronic sinusitis: a histopathological and immunohistochemical study. *Laryngoscope* **112**(4): 738–45

Bernstein JM, Gorfien J, Noble B (1995) Role of allergy in nasal polyposis: a review. *Otolaryngol Head Neck Surg* **113**(6): 724–32

Blomqvist EH, Lundblad L, Anggard A, Haraldsson PO, Stjarne P (2001) A randomized controlled study evaluating medical treatment versus surgical treatment in addition to medical treatment of nasal polyposis. *J Allergy Clin Immunol* **107**(2): 224–8

Brook I (1989) Bacteriology of chronic maxillary sinusitis in adults. *Ann Otol Rhinol Laryngol* **98**(6): 426–8

Brook I (1996) Microbiology and management of sinusitis. *J Otolaryngol* **25**(4): 249–56

Caplin I, Haynes JT, Spahn J (1971) Are nasal polyps an allergic phenomenon? *Ann Allergy* **29**(12): 631–4

Cervin A (2001) The anti-inflammatory effect of erythromycin and its derivatives, with special reference to nasal polyposis and chronic sinusitis. *Acta Oto-Laryngologica* **121:** 83–92

Conley DB, Tripathi A, Seiberling KA et al (2006) Superantigens and chronic rhinosinusitis: skewing of T-cell receptor V beta-distributions in polyp-derived CD4+ and CD8+ T cells. *Am J Rhinol* **20**(5): 534–9

Delaney JC (1976) Aspirin idiosyncrasy in patients admitted for nasal polypectomy. *Clin Otolaryngol* **1**(1): 27–30

Ebbens FA, Scadding GK, Badia L et al (2006) Amphotericin B nasal lavages: not a solution for patients with chronic rhinosinusitis. *J Allergy Clin Immunol* **118**(5): 1149–56

English G (1985) Nasal polyposis. In: English GM, ed. *Otolarygology*. Harper and Row, Philadelphia: 1–30

Erdem G, Erdem T, Miman MC, Ozturan O (2004) A radiological anatomic study of the cribriform plate compared with constant structures. *Rhinology* **42**(4): 225–9

Fokkens W, Lund V, Mullol J; European Position Paper on Rhinosinusitis and Nasal Polyps group (2007) European position paper on rhinosinusitis and nasal polyps. *Rhinology* **45** (suppl 20): 1–136

Gevaert P, Lang-Loidolt D, Lackner A et al (2006) Nasal IL-5 levels determine the response to ant-IL-5 treatment in patients with nasal polyps. *J Allergy Clin Immunol* **118:** 1133–41

Hedman J, Kaprio J, Poussa T, Nieminen MM (1999) Prevalence of asthma, aspirin intolerance, nasal polyposis and chronic obstructive pulmonary disease in a population-based study. *Int J Epidemiol* **28**(4): 717–22

Iinuma T, Hirota Y, Kase Y (1994) Radio-opacity of the paranasal sinuses. Conventional views and CT. *Rhinology* **32**(3): 134–6

Johansson L, Akerlund A, Holmberg K, Melen I, Stierna P, Bende M (2000) Evaluation of methods for endoscopic staging of nasal polyposis. *Acta Otolaryngol* **120**(1): 72–6

Johansson L, Akerlund A, Holmberg K, Melen I, Bende M (2003) Prevalence of nasal polyps in adults. *Ann Otol Rhinol Laryngol* **112**(7): 625–9

Jonas I, Mann W (1976) [Misleading x-ray diagnosis due to maxillary sinus asymmetries (author's transl)]. *Laryngol Rhinol Otol (Stuttg)* **55**(11): 905–13

Kazkayasi M, Karadeniz Y, Arikan OK (2005) Anatomic variations of the sphenoid sinus on computed tomography. *Rhinology* **43**(2): 109–14

Khalil HS, Nunez DA (2006) Functional endoscopic sinus surgery for chronic rhinosinusitis. *Cochrane Database Syst Rev* **3**: CD004458

Klossek JM, Neukirch F, Pribil C et al (2005) Prevalence of nasal polyposis in France: A cross-sectional, case-control study. *Allergy* **60**(2): 233–7

Lildholdt T, Rundcrantz H, Lindqvist N (1995) Efficacy of topical corticosteroid powder for nasal polyps: a double-blind, placebo-controlled study of budesonide. *Clin Otolaryngol* **20**(1): 26–30

Lund VJ (2001) Evidence-based surgery in chronic rhinosinusitis. *Acta Otolaryngol* **121**(1): 5–9

Lund VJ, Mackay IS (1993) Staging in rhinosinusitus. *Rhinology* **31**(4): 183–4

Lund VJ, Scadding GK (1994) Objective assessment of endoscopic sinus surgery in the management of chronic rhinosinusitis: an update. *J Laryngol Otol* **108**(9): 749–53

McAlister WH, Lusk R, Muntz HR (1989) Comparison of plain radiographs and coronal CT scans in infants and children with recurrent sinusitis. *Am J Roentgenol* **153**(6): 1259–64

Mehanna H, Mills J, Kelly B, McGarry GW (1996) Benefit from endoscopic sinus surgery. *Ann Otol Rhinol Laryngol* **105:** 415–22

Mullol J, Xaubet A, Gaya A et al (1995) Cytokine gene expression and release from epithelial cells. A comparison study between healthy nasal mucosa and nasal polyps. *Clin Exp Allergy* **25**(7): 607–15

Mygind N, Dahl R, Bachert C (2000) Nasal polyposis, eosinophil dominated inflammation, and allergy. *Thorax* **55**(Suppl 2): S79–83

Ottaviano G, Scadding GK, Coles S, Lund VJ (2006) Peak nasal inspiratory flow; normal range in adult population. *Rhinology* **44**(1): 32–5

Pawliczak R, Lewandowska-Polak A, Kowalski ML (2005) Pathogenesis of nasal polyps: an update. *Curr Allergy Asthma Rep* **5**(6): 463–71

Ragab SM, Lund VJ, Scadding G (2004) Evaluation of the medical and surgical treatment of chronic rhinosinusitis: a prospective, randomised, controlled trial. *Laryngoscope* **114**(5): 923–30

Schubert MS (2001) Fungal rhinosinusitis: diagnosis and therapy. *Curr Allergy Asthma Rep* **1**(3): 268–76

Settipane G (1997) Epidemiology of nasal polyps. In: Settipane G, Lund VJ, Bernstein JM, Tos M, eds. *Nasal Polyps: Epidemiology, Pathogenesis and Treatment.* Oceanside Publications, Rhode Island, USA: 17–24

Settipane GA, Chafee FH (1977) Nasal polyps in asthma and rhinitis. A review of 6,037 patients. *J Allergy Clin Immunol* **59**(1): 17–21

Stoop AE, van der Heijden HA, Biewenga J, van der Baan S (1993) Eosinophils in nasal polyps and nasal mucosa: an immunohistochemical study. *J Allergy Clin Immunol* **91**(2): 616–22

Szczeklik A, Nizankowska E, Duplaga M (2000) Natural history of aspirin-induced asthma. AIANE Investigators. European Network on Aspirin-Induced Asthma. *Eur Respir J* **16**(3): 432–6

Tos M, Sasaki Y, Ohnishi M et al (1992) Fireside conference 2. Pathogenesis of nasal polyps. *Rhinology* **14**(Suppl): 181–5

Weschta M, Rimek D, Formanek M, Podbielski A, Riechelmann H (2006) Effect of nasal antifungal therapy on nasal cell activation markers in chronic rhinosinusitis. *Arch Otolaryngol Head Neck Surg* **132**(7): 743–7

Xaubet A, Mullol J, Lopez E et al (1994) Comparison of the role of nasal polyp and normal nasal mucosal epithelial cells on in vitro eosinophil survival. Mediation by GM-CSF and inhibition by dexamethasone. *Clin Exp Allergy* **24**(4): 307–17

Zhang N, Gevaert P, van Zele T et al (2005) An update on the impact of *Staphylococcus aureus* enterotoxins in chronic sinusitis with nasal polyposis. *Rhinology* **43**(3): 162–8

KEY POINTS

- Nasal polyps are the end result of chronic inflammation in the nose and paranasal sinuses, and are seen in up to 50% of patients with asthma.
- Medical treatment consists mainly of topical and systemic glucocorticosteroids.
- Functional endoscopic sinus surgery is a minimally invasive technique that aims to improve sinus ventilation and drainage in addition to polyp removal.
- Despite extensive research the cause of nasal polyps remains unknown.