THE HUMMER

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Sinus surgeons have been admonished to approach their task with the mindset of ear surgeons. Some parallels are clear: Both the otomastoid and paranasal sinuses are relatively small cavities with complex configurations surrounded by critical, even vital neighbouring structures or organs. Ear surgeons however, have been delivered from the fear of hammer-and-chisel days by a precision instrument - precision that makes the most difficult of cases routine in experienced hands with expected good outcomes and low morbidity. The precision of the mastoid drill and the progressive refinement in supporting instruments coupled with improved visualization have converted ear and mastoid surgery to an exercise of respect.

THE HUMMER

Since 1992, the author has successfully used a power instrument that addresses many of the critical issues confronting today's sinus surgeon. The initial work began with the utilization of a powered microdebrider manufactured by Stryker Endoscopy (San Jose, CA), designed originally for temporomandibular joint surgery. A third generation instrument from Stryker-Leibinger (Hummer TPS) is used today and provides continuous irrigation and back flush capability in the event of clogging.

In the early going, there were struggles to determine tip selection, power settings, suction level, means of clearing clogged suction lines, and the extent to which the device would be effective for sinus surgery. However, the promise of a better way was evident. At its worst, the instrument provided advantages over traditional instrumentation. At its best, it now replaces most of the instrumentation the author formally used for sinus surgery. In general, the result is a precision instrument with real time suction that addresses most of the troublesome issues that preclude the delivery of advanced state of the art sinus surgery. Today, if the instrument is not available, sinus procedures are canceled.

The complete surgical unit consists of a power unit and its foot-switch or pedal, a handpiece, and a disposable blade. The most effective blade is the aggressive cutter. It is small with a diameter of 3.5mm and has benign contours that make it relatively atraumatic. A 2.5mm aggressive cutter is also available for the particularly small or narrow nose. Each blade has two components, the outer portion being the sheath, and the inner the cutting blade. The outside protecting sheath has a distal working port on one side on as a bias that includes a small part of the time. The cutting blade within the sheath has a window to match and teeth to cut. One of the important features of the instrument is a hollow shaft in the center of the inner cutting blade, which provides a channel for continuous suction to removed blood and resected tissue debris. This feature dramatically improves visibility for the surgeon.

Further, precise removal of obstructing tissue and bone is possible without inadvertent peripheral damage or stripping of uninvolved mucous membrane. Diseased mucous membrane is also retained at the limits of the dissection, a hallmark of minimally invasive sinus surgery. A suction port on the proximal end accepts suction tubing and provides egress from the handpiece to the suction canister. Optional cloth baskets placed in the suction canister or specifically manufactured traps may be used to enhance specimen collection.

Proper suction is critical for the effective use of powered instrumentation. Any decrease in effectiveness is most likely the result of reduced suction. The Stryker Leibinger unit has a suction control on the handpiece that must be in the open position or suctioned will be diminished.

Should clogging occur, the reduced effectiveness in cutter action is readily apparent. Clearing the handpiece on the Stryker Leibinger unit requires only the turn of a manual rocker and a back flush. Irrigation of the tip, however, is not possible if the cutter window is closed; therefore, the blade should be in the open position or spinning during irrigation. Insertion of a 19-gauge blunt needle into the open window and up the cutter channel may be required for cleaning, but rarely are disassembly required.

The powered approach

A precise surgical technique is possible with the Hummer in almost every surgical procedure when the preceding variables are mastered, the device is properly set up, and training is obtained and in both the application of powered instrumentation and the functional approach to endoscopic sinus surgery. Whether the problem is caused by severe polyposis or anatomic variations, even the most difficult sinus operation can usually be converted into a routine procedure. Polyps may be precisely removed, better defining the structures of the nasal cavity and middle meatus, usually without troublesome bleeding.

Once access to the middle meatus is achieved, a step-by-step surgical approach can be implemented, not unlike that in mastoid and ear surgery, progressing systematically as the anatomy unfolds. Even the most recent manual "cutting" instruments fall short of the precision and control possible with powered instrumentation. More precision and less peripheral damage mean less bleeding than with manual instruments. If bleeding occurs, blood is removed from the surgical field in real time, improving visibility and reducing apprehension for the surgeon, not to mention the obvious decreased risk for the patient.

Although the use of powered instrumentation is no substitute for good training and experience in sinus surgery, it can dramatically elevate the level of sinus surgery. Full realization of the benefits of the approach can be obtained only by a surgeon with underlying knowledge of the anatomy and pathophysiology of the nasal cavity and paranasal sinuses. Further, every possible effort should be made to prepare the patient and operative setting to keep troublesome bleeding to a minimum. An effective preoperative vasoconstrictive routine is detailed elsewhere. The middle turbinate

is a problem for the powered endoscopist only to the extent to which it limits access to the middle meatus.

With the nonbiting side of the outer sheath, the lower middle turbinate can be pushed gently toward the septum without mobilizing it. The biting surface is facing the surgical site, the middle turbinate can be preserved with its mucosa intact. If the middle turbinate is thick and interfering with the surgical approach, the smaller-diameter, 2.5mm aggressive cutter may be used.

An alternative is a very minimal powered removal of the lateral turbinate mucosa. This usually resolves the difficulty *without* a more aggressive resection of the middle turbinate, a procedure generally discouraged in functional surgery and particularly in children. If a concha bulla is present, the lateral half may be "hummed" away. Care much be taken to preserve the medial mucous membrane inside the residual concha. The outflow tract of the concha bullosa should be identified and opened to communicate with the entire dissection.

An anatomical surgical model

For many years, the author has used an anatomical surgical model to replace the classical Wiegand or Messer Klinger approaches. The model provides a systematic progression using anatomical landmarks in an anterior to posterior fashion. The landmarks and their associates spaces: The uncinate process and ethmoidal infundibulum, the medial wall of the ethmoidal bulla and the hiatus semilunaris superior, the posterior and medial walls of the Agger nazi cell and frontal sinus entry, and basal lamella.

The retrobullar space could be recognized as an associated space.

Finding the maxillary sinus ostium

Following the recommended preoperative nasal vasoconstriction and intraoperative injections, a 0-degree or 30-degree endoscope is used to visualize the uncinate process, landmark #1, with the introduction of a back biter (instrumentarian, Montreal, Canada) through the semilunar hiatus near its mid-portion, a retrograde cut in the lower midportion of the uncinate process initiates the creation o f a window into the infundibulum. A submucosal resection or powered removal of the inferior portion of the uncinate process in full thickness allows visualization of the maxillary sinus ostium. The ostium may be found by tracking visually or with a curved seeker from the exit of the infundibulum anteriorly, the ostium dropping off inferiorly and laterally into the maxillary sinus. Care must be taken to extent the uncinate cut to the anterior limits of the infundibulum, space #1.

This is usually all that what is required to address maxillary sinus disease, i.e. eliminating the transition space and providing a direct entry into the nasal cavity.

Antrior ethmoidectomy

A precise identification and functional dissection of the medial wall of the bulla ethmoidalis, landmark #2, are possible with powered instrumentation. If the back-side of the blade is placed along the lateral side of the lower middle turbinate, with slight medial displacement of the turbinate, the interface between the medial wall of the bulla and the middle turbinate is revealed, the hiatus semilunaris superior, land-mark #2.

Opening the bulla from medal to lateral both preserves the basal lamella as a future and begins the anterior ethmoid dissection with the least risk to the lamina papyracea. Small plates of ethmoid bone may emerge as the instrument is dissecting laterally, delaying entrance into the bulla itself. As a result of the inability of the cutter to obtain an edge to pull the bone into its window, the plates are pushed later-ally ahead of the blade tip submucosally. Removing such bony plates with pediatric forceps facilitates the operation and allows a dramatic "humming" of the exposed medial mucous membrane without stripping the bulla and lamina papyracea to bare bone. The limited anteroposteroir dimension of the ethmoid bulla is a surprising finding, particularly in pediatric cases.

Just as it is important to identify the natural ostium of the maxillary sinus, it is equally necessary to locate the natural drainage of the ethmoidal bulla. One to three ethmoid compartments may be present, with variable exits anteriorly to the ethmoi-dal infundibulum, posteriorly to the superior meatus and/or medially to the hiatus semilunaris superior. The retrobulla space also drains to the hiatus semilunaris supe-rior. The medial drainage is found behind a band of mucous membrane at the most posteromedial portion of the bulla, anterior to the basal lamella.

Frontal sinus drainage

Should there be questions about or an indication for investigating the status of frontal sinus drainage, the powered approach to the frontal sinus is through the re-sidual upper uncinate process, an as yet undisturbed portion of the structure if a lower uncinate window is made. The full thickness may be "hummed" away, aided by the occasional removal of bony plates of the uncinate process with a 0-degree or 90-degree pediatric forceps. As with the ethmoid soft tissue, the mucosal on both sides of the uncinate is easily consumed with the Hummer. Invariably, the mucous membrane of the floor of the aggar nasi cell comes into view simply with removal of the most superior uncinate. Powered instrumentation reli-ably and atraumatically locates and defines this cell, a crucial step in a minimally invasive approach to the frontal sinus. As small bone fragments are teased away, a larger opening is created, allowing visualization of the dome of the agger nasi cell with a 30-degree endoscope. During the dissection, the cutting side f the instrument must be directed superiorly and laterally, but *never medially* toward the root of the middle turbinate.

When disease is present, the search for where the frontal sinus entry should be directed posterior and medial to the agger nasi cell, landmark #3. Simply retracting the posterior wall of the agger nasi cell forward may reveal the otherwise hidden exit of the frontal sinus, space #3, and also may be therapeutic, with no further dissection being required. Powered removal f the posteromedial portion is usually possible if there is a need to remove diseased tissue. The dissection is accomplished, in most instances, with a straight blade, moving from medial to lateral until the space posterior to the agger nasi cell develops or the dome of the cell (ie. Floor of the frontal sinus) is reached. That usually coincides with entry into the frontal sinus and may require other than a powered approach. Aggressive removal of the dome of the agger nasi cell is seldom required.

Posterior ethmoidectomy

If the preceding operation has been carefully done, the vertical portion of the basal lamella, landmark #4, is now visible. The presentation varies greatly, depending upon the patter and extent of retrobullar development. Whatever the pattern, the blade tip allows a controlled entry into the posterior ethmoid complex. The tip's benign contours, precision cutting action, and small size reduce dramatically the potential for a surgical accident in knowledgeable and experienced hands. Simple inspection or clearing of cells to the base of the skull is possible without stripping of mucous membranes and bony exposure. Manual removal of bony plates is not often required bt may be helpful. The removal of the soft tissue with forceps is rarely necessary.

Finding the sphenoid sinus ostium

The 8cm length, small diameter, and benign contours of the Hummer blade make is ideal for a direct approach to the sphenoid sinus ostium with less fear of a catastrophic event. In the absence of fungal disease or unusual skull base pathology, there appears to be no compelling reason that surgery of the sphenoid sinus should extend beyond clearing the sphenoethmoid recess, the sphenoid ostium, or both. If one proceeds between the middle turbinate and septum no a line between the anterior two thirds, and posterior one third of the superior turbinate a direct powered approach to the sphenoid face will usually allow both visualization and clearing of the sphenoid ostium. Lateral displacement of the superior turbinate is occasionally required. After disease tissue is removed from the face and the ostium of the sphenoid, enlargement of the ostium itself is usually limited to the inferior portion, if required at all, and may readily be performed with the Hummer.

Other benefits of the HUMMER

The same precision that given confidence to the surgeon confers benefits on the patient. Continuous, real-time suction dramatically reduces the necessity of moving instruments in and out of the nasal cavity. The result is less trauma and therefore less

bleeding. Less trauma and the ability to leave mucous membrane at the limits of the dissection dramatically reduce the healing burden and therefore the time required for complete healing to occur.

Continuous suction obviates the necessity for a nasopharyngeal pack. More precision means less morbidity for the patient during the procedure. Better visibility and the prospect of an anatomic dissection are confidence builders for the surgeon. Outpatient surgery in a clinic setting, as in simple polypectomy and lysis procedures, are also quite feasible with use of the Hummer.

Conclusion

Armed with a functional approach to endoscopic sinus surgery and experience with powered instrumentation, a *precise, minimally invasive* surgical technique is possible. The approach addresses most of the troublesome issues that have characterized sinus surgery for decades and heralds a new horizon in instrumentation for nasal and sinus surgery, particularly in nasal polyposis.

ITRACONAZOLE THERAPY FOR REFRACTORY SINUSITIS

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Ranko,

Please excuse the delay in getting the requested info regarding the above. About 5-years ago, I became frustrated with by failure in a certain subset of sinus patients and concluded that they were not destined to respond long term to surgical therapy by me or anyone else. The characteristics of the patients are well known, including a favorable response to steroids. Most, however, were fearful of steroids therapy. Suspecting a fungal component, I tried Diflucans in a modest dose with success in only one patient. Manrin Raines encouraged me to try intraconazole (ITZE) in a tapering dose over a 3-months, ie. 400, 300, 200. The initial response was favorable, but deteriorated with a the declininig dose. Manrin told me they were getting re-infected. Suspecting otherwise, I resumed the 400 bid and, after trying earlier tapers and pulsing schemes, ultimately proved, at least to my satisfaction, that a one year minimum treatment is required. Singulair and topical ITZE are also given, but virtually all patients no longer needed antibiotics or steroids, even the steroid-dependent asthmatics. The response to therapy is usually evident in 3-6 weeks and is not infrequently dramatic, even scary. The drug is absorbed only in the presence of acid, thus dosing 15-minutes, pc with a glass of cola is our regimen. Al of the disclaimer regarding off-label use, etc. are reviewed. In short, by the time I'm done explaining what I don't know, the patient would be foolish to take the drug, but invariability the recommendation is accepted.

Having told the patient there is no promise of quiescence after stopping the dry, nevertheless, the challenge is to do just that. Usually, the patients are treated initially to obtain maximum clearing before surgery, the latter to evacuate secretions and debris and do an initial marsupialization, leaving all turbinated and without doing a middle meatal antrostomy. The ITZE it then continued and sometimes a second and hopefully final intervention is done to evacuate any residual disease and complete the marsupializatino before dropping the dose to 200mg/day in one dose. The idea is to maximize the potential for effective topical therapy as a long term treatment vis a vis the Mayo theory.

Initially, patients were told that I had no idea why the drug was effective, having started the intervention before the Mayo revelations. Now they are told that their problem may well be the product of a dysfunctional immune system, and the ITZE reduces the challenge to the immune system as opposed to taking steroids to suppress the response. The histories on the patients are frequently quite different from a bacterial process. Of course, most have failed multiple surgical procedure, but going back most were without significant upper respiratory problems until a seminal event which proved to be the beginning of an intractable problem. Not infrequently, the onset was quite sudden, although some report a more insidiously progressive course.

The relatively sudden onset in a patient without prior significant problems is helpful in a virgin case in deciding whether or not to given a trial of ITZE. My belief is that any patient with pansinusitis with or without polyposis should have a trial of ITZE before surgery.

These patients have a wide profile regarding asthma, atopy, ASA sensitivity, polyps, etc. IN short, there is no typical patient, only suggestive clinical clues. Currently, I am treating for a minimum of 1-year at full dose, dropping to 200mg/da, depending on clinical response, but only after clean-up surgery as mentioned. Patient are followed at 4-6 week intervals with/without 5mm coronal CT scan, again depending upon variables. IN contrast to the prompt relapse following a dose reduction after a few months of therapy, the long term patient will, if at all, relapse more insidiously. I am suspicious that the concentration of mold spores in the home and/or work environment may be important and am now sampling as best I can for that possibility, particularly in refractory patients.

Problems with ITZE used in over 200 patient have been suprisingly few. Only one patient has developed jaundice, clearing with cessation and associated with the concomitant use of other meds cleared by hepatic route. A few patient developed rash allergy, necessitating cessation or dose reduction. Even fewer developed extremity swelling or other new complaints, also requiring cessation. Although I do not know the possible long term downside to long term ITZE therapy, my experience thus far would seem to conclusively dispell the doom's day outlook expressed in some quarters for the systemic use of the drug fo the refractory patient.

Obviously, there is much to be learned regarding the role of fungi, in general, and the use of itraconazole, in particular, for refractory sinusitis.

HOST AND MICROBIAL INTERACTIONS IN NASAL POLYPOSIS

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EPIDEMIOLOGY

Nasal polyposis constitute a heterogenous inflammatory disorder often linked to asthma, eosinophilia and upper and lower respiratory tract infections. No single predisposing disease can account for the formation of nasal polyps in all patients and the underlying inflammatory process is multifactorial. The prevalence of nasal polyposis in the general population has been estimated to 1-4 percent as compared to autopsy studies where isolated nasal and ethmoidal polyps have been reported in up to 40 percent. This points towards that the development of smaller polyps may be an intermittent phenomenon with spontaneous healing early in the disease.(1, 2)

The degree of nasal polyposis is coexsistant with a series of parameters. The presence of asthma, especially non-allergic asthma, is associated with an increased prevalence (13%) more pronounced when also associated with aspirine intolerance (36%). Also chronic bacterial sinusitis is associated with an increased frequency such as in dental sinusitis (16%). A minor increase in frequency may also be seen during immunodeficiency and perennial allergy. (1, 3) The risk of polyp development is associated with the degree and early onset of asthma although all patients do not display an increased lower airway hyperreactivity. (4)

PATHOPHYSIOLOGY

Histological analysis of early stage polyps in human and animal models have shown evidence of exsudation and retention of albumine and tissue fluid together with a localized eosinophilic inflammation. The polyp formation sequence also seems to involve multiple epithelial disruptions with proliferating granulation tissue and a healing reaction involving glandular formation and intra-epithelial cyst like cavities. There are many similarities between the sequence of early polyp formation and the normal healing process after surface epithelial trauma of the mucosa. During certain conditions such as prolonged inflammatory trauma, epithelial regeneration accompanied by interactions with an altered extra-cellular matrix composition and proliferating connective tissue cells will lead to polyp formation. The polyp formation and growth is thus activated and perpetuated by an integrated process of the mucosal epithelium, matrix and inflammatory cells, which in turn may be initiated by both infectious and a non-infectious eosinophilic inflammation (5, 6). Respiratory tract viral infections with increased recruitment and local activity of eosinophils is a common clinical trigger of asthma and the onset of polyposis.

Immuno-fluorescence staining for Major Basic Protein (MBP) demonstrates striking release of toxic protein in the mucus of CRS patient. Note the intact eosinophils in the tissue (right side of image) and the eroded epithelium (arrows). (x200)

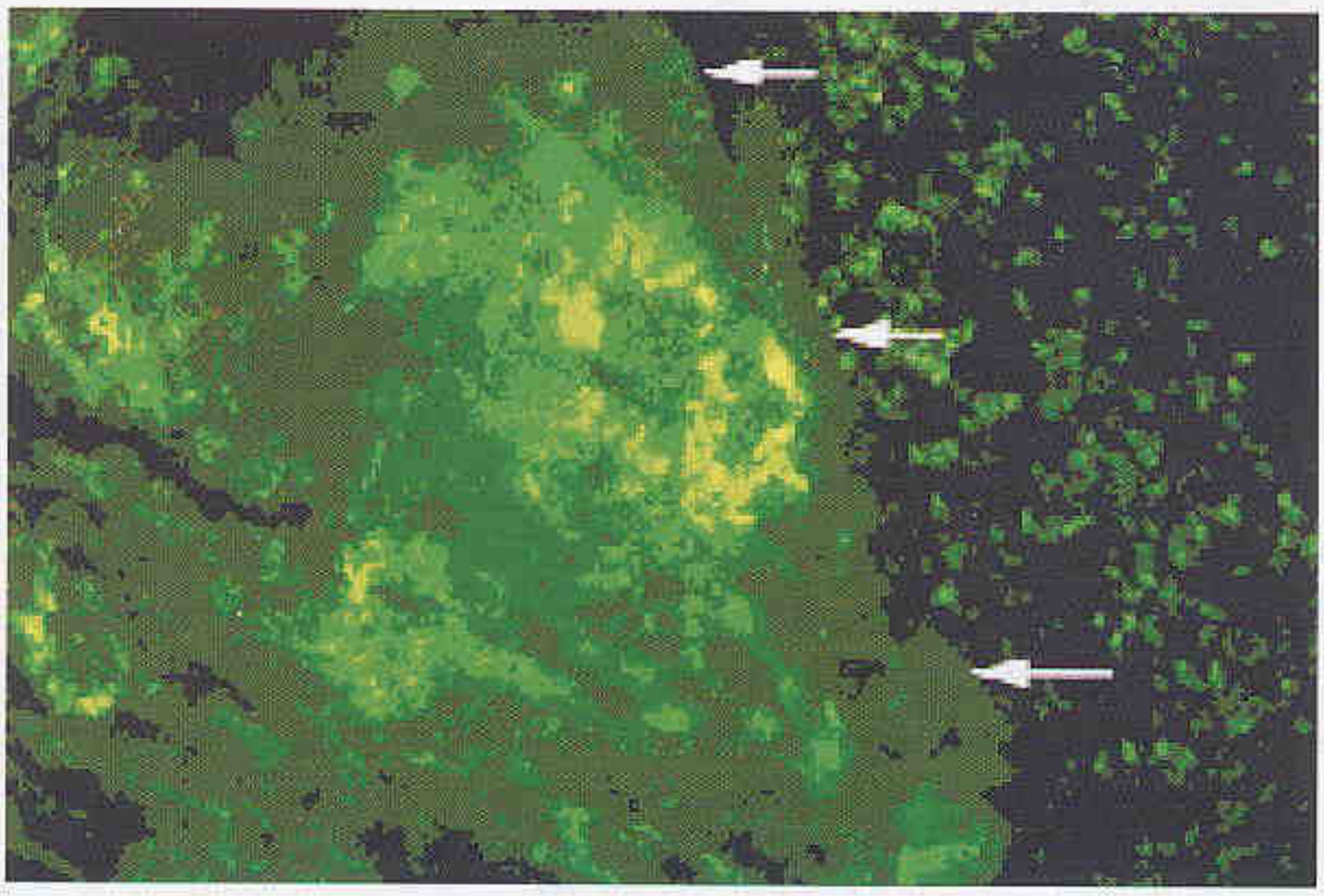

Fig. 2

INFLAMMATION

Allergic

Atopy itself has not been correlated to nasal polyposis. However, patients with positive skin test have shown to have a higher frequency of multiple polypectomies than those with negative allergy skin test. An increased incidence of subclinical food intolerance may be present. (7) Recent studies of the presence of specific IgE towards Staphylococcous aureus enterotoxins have shown a high correlation with the degree of asthma and nasal polyposis. This suggest that Staph. aureus colonization of the nasal mucosa, acting as a superantigen similarily as in atopic skin disease, may be an important factor in nasal polypoid disease. According to the Mayo Clinic concept, fungal colonization in the mucus of the nasal mucosa appears to be an antigenic trigger leading to local eosinophil recruitment and activation and with a high association to nasal polyposis. Fungus colonization of sinus secretions with sensitisation as shown by skin testing suggests that also these allergens may play a role in driving Th2 inflammation and concomitant eosinophilia in nasal polyposis.

Non-allergic

Cytokines promoting the activation and survival of eosinophils, namely GM-CSF, IL-1 and IL-5 are present in chronic hyperplastic sinusitis, with nasal polyposis (8, 9). The number of oesinophils in nasal polyps is correlated with the corresponding statement of cytokines in both allergic and non-allergic patients. T cells account for the majority of the IL-5 positive cells. In allergic patients an increased statement of Th2 cytokines has been shown whereas in contrast in non-allergic nasal polyposis the cytokine profile may be a mixed one of Th1 and Th2. (10, 11) These mixed profiles of cytokines described as Th1-Th0 profiles are also present in atopic dermatitis lesions.

Asthma and aspirine intolerance are associated with nasal polyposis in more than half of the patients. Cyclooxygenase-2 is equally expressed in nasal polyps from both allergic and non-allergic patients suggesting that arachidonic acid metabolism is involved irrespective of polyp etiology. An elevated 5-lipooxygenase activity results in an increased production of the leukotrienes LTC4, LTD4, LTE4 producing an increased airway responsiveness at several levels (12).

An element of bacterial infection may trigger polyposis in immunodeficiency, cystic fibrosis or dental periapical infections. For example Melén and coworkers have demonstrated that polyps occur in 16% of patients with maxillary sinusitis secondarily to dental infection. Subsequent studies of sinus washouts and antroscopy studies indicate that purulent secretion together with colonizing airway pathogens will activate the disease or make it persistant. In patients with cystic fibrosis and immotile cilia syndromes colonizing specific pathogens are correlated to the occurrence of nasal polyps. Treatment studies also indicate that local deposition of an antibiotic together with glucocorticosteroids is more effective than antibiotics alone

in reducing local tissue pathology including sinus polyps. Colonizing bacteria and/or fungus may thus activate mucosal defense mechanisms and enhance recruitment and activation of inflammatory cells contributing to polyp formation. (13, 14) Proinflammatory cytokines are detected in sinus polypoid tissue during acute bacterial sinusitis. The role of ostial patency as an inflammatory trigger during polyposis is uncertain but may play a lesser causal role than the more prominent eosinophilic dominated inflammation.

There is much evidence pointing towards nasal polyposis as a manifestation of systemic disease, characterized by an eosinophil-dominated inflammation both in the polyp but also in the lower airways and bone marrow compartment. Diffuse polyposis represents a spectrum of diseases ranging from a marked eosinophilic inflammation process with or without bacterial or fungal colonization to an inflammatory cell infiltration due to infection or with unknown origin (15).

Diagnosis and staging

The staging of nasal polyposis is mostly based on background disease and whether this is localized or systemic. In this respect certain disease entities are known to be more aggressive then others and the degree of eosinophilia in various cellular compartments may be an indicator of disease activation and degree of aggressiveness. Endoscopic visualization of the polyps and adjacent regions of the nasal and sinus cavities are necessary for correct diagnosis and staging. Various more or less objective, methods such as rhinometry and nasal peak flow and polyp size grading system may be necessary to analyse the degree of polyposis as well as treatment effects. MRI or CT may during more or less experimental study conditions estimate the degree of polyposis in the various sinus compartments. Symptom score and nasal airflow measurements are generally sensitive but measure treatment effects, for example corticosteroids, on both polyp size and mucosal swelling. (16) A new method to analyse actual polyp size in two dimensions, i.e. lateral imaging have been found to have a good reproducibility and a better sensitivity than methods earlier used. (17, 18)

CONCLUSION

Increased knowledge of microbial / host immune interactions have resulted in a more specific understanding of the complex regulation regarding local inflammation, eosinophil activation as the result of microbial colonization, for the initiation and persistance of nasal polyposis. As the disease involves various disturbed functions, polyposis continues to be a diagnostic and therapeutic challenge. The significance of host and microbial factors respectively in the pathophysiology of nasal polyposis vary depending on both predisposing background disease and stage of disease. The intricate of relationship between immunologic triggers, inflammation and microbial colonization means that more scientific rationales for medical and surgical intervention needs to be developed. The future successful treatment strategies will

involve an individual timing of stepwise medical therapy based on anti-inflammatory strategies and antibiotics in relation to surgical removal from nose and sinus cavity for successful treatment.

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AETIOLOGY OF NASAL POLYPOSIS: CHRONIC INFECTION, FUNGI OR BOTH?

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Having excluded the other possible conditions in the differential diagnosis *e.g* meningoencephalocele, inverted papilloma and other neoplasia, the most striking thing about nasal polyps is the range of severity that may be found clinically, ranging from small areas of localised swelling where two mucosal surfaces touch through to gross ethmoidal expansion and hypertelorism in extreme cases. Recent studies suggest a considerable impact on the quality of life from this condition (Randenne et al 1999). In the literature a wide range of conditions are known to be associated or predisposed to nasal polyposis (Table 1). In fact in the majority of cases the true aetiology of the polyps is unknown and it is of interest that the condition is associated with some inflammatory airway diseases and not others, and appears to affect a very small portion of the nasal mucosa with relative sparing of the septum and inferior turbinate. In studies of the frequency of nasal polyps in various diseases conducted by Settipane and others there are marked differences in the frequency of polyps in children with cystic fibrosis (10%) and those with allergic rhinitis (0.1%) and similarly differences between adults with allergic rhinitis (1.5%) and those with non-allergic rhinitis (5%). This suggests that allergy *per se* is not the main cause of polyps although the combination of the two conditions will make the patient's symptoms commensurately worse. Studies by Larson and Tos on cadavers without a known history of nasal problems suggest that polyps may be frequently found within the middle meatus and ethmoid complex (up to 42%) whilst only approximately 2% of the population are bothered symptomatically. These changes in the normal population may represent the end-point of simple viral infections resulting in mucosal apposition.

Work by Norlander and Stajne have shown in animal models that common upper respiratory tract pathogens *e.g.* haemophillus can induce polyp formation within the maxillary sinus though whether this translates to the human situation is unclear. Certainly patients will often comment on a persistent exacerbation of their symptoms and (the size of their polyps) after a cold.

The role of fungus in the development of nasal polyposis has been much discussed of late. There has been an increasing recognition of allergic fungal rhinosinusitis (or eosinophilic fungal rhinosinusitis) since the condition was first described by Miller and colleagues in 1981. Pre-perative CT appearances will often suggest the diagnosis (Lund, Lloyd et al 2000) but this is confirmed by the presence of the characteristic axle grease secretion mixed amongst the polyps in which occasional fungal hyphae together with Charcot Leyden crystals (indicative of dead eosinophils) are found. However, there has been a suggestion from Ponikau and colleagues that fungi may precipitate both nasal polyp formation and chronic rhinosinusitis in the majority of

patients. So far there has been relatively little published in the peer review literature other than a report on the incidence of fungi in a cohort of 210 patients, under half of whom underwent surgery. (Ponikau JU et al) Fungi are clearly widespread in the environment and indeed the same authors have found fungi in all their normal controls when careful cultures were performed. The exact relevance of this in the aetiology of nasal polyps and the implications for therapy have yet to be determined though a multicentre randomised placebo control trial of the use of topical amphotericin douche is about to start in a number of European centres.

The association of nasal polyps with asthma and aspirin intolerance (as well as other non-steroidal anti-inflammatory drugs) suggest that other factors may be at work in some of these patients. Furthermore the high number of cystic fibrosis patients suffering from aggressive polyposis may be the result of the underlying problem with electrolyte transport across membranes or occurring secondarily to the frequent superadded bacterial infection particularly with staph aureus and pseudomonas aeruginosa. There are marked differences in the histology of polyps in cystic fibrosis compared with those in asthma/ASA which support a different pathogenesis. This might also be inferred from the marked eosinophilia and associated nasal polyps found in Churg Strauss syndrome, a manifestation of systemic vaculitis whereas the finding of nasal polyps in primary ciliary dyskinesia and Young's syndrome could simply be another example of the response to secondary bacteria infection.

At the end of the day there does not appear to be one single unifying aetiological factor in the development of nasal polyposis though infection, be it bacterial or fungal may be the initiating event in susceptible individuals which is compatible with theories of polyp formation such as epithelial rupture as espoused by Tos and colleagues. This should also be interpreted in the light of excellent cellular studies on cytokine activity by authors such as Bachert. Ultimately nasal polyposis should be seen as the end product of a number of disease processes and pathogenic mechanisms rather than a single disease entity.

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Table 1 Aetiology factors in nasal polyposis

- Mucosal apposition
- Infection} } fungal
- Allergy }

- Aspirin sensitivity
- Asthma
- Cystic fibrosis
- Cherg-Strauss syndrome
- PCD (Primary ciliary dyskinesis)
- Young's syndrome

CHRONIC RHINOSINUSITIS: THE WAR OF THE IMMUNE SYSTEM AGAINST THE FUNGI

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Chronic rhinosinusitis (CRS) is a confusing disease for the practicing otorhinolaryngologist. It usually presents histologically as an eosinophilic inflammation that is complicated by periods of acute exacerbation. These acute exacerbations are presumed to be of bacterial origin. Bacterial infections trigger a neutrophilic inflammation. The eosinophilic inflammation seen in chronic rhinosinusitis is not likely to be caused by bacteria. Eosinophils are understood to play a role in the host defense against larger, non-phagocytosable organisms such as parasites.

The original aim of our research on chronic sinusitis was to prospectively determine the incidence of Allergic Fungal Sinusitis (AFS). Through novel culture, histologic and antigen detecting methods we were able to demonstrate the presence of fungi in every patient with chronic rhinosinusitis (n=46), and in every healthy control person without the disease(n=14).

CRS is known to be a disorder characterized by eosinophilic inflammation with hyperplastic thickening of the nasal and paranasal mucosa. It is a spectrum of disease, in what the inflammatory thickenings range from small polypoid changes in the middle meatus at one end of the spectrum to gross nasal polyps at the other end.

By studying the tissue and mucin from chronic rhinosinusitis sufferers more closely we observed that the eosinophils were present nearly entirely intact in the tissue. Further the eosinophils migrated into the mucin, formed clusters around fungi and degranulated. Since this was observed in the majority (96%) of consecutive surgical CRS cases (n=101), the questions was raised whether the eosinophils play an immunologic defensive role against those fungi in CRS patients.

Immunologic testing further showed that the chronic sinusitis patients peripheral blood T-lymphocytes, when presented with certain fungal antigens, reacted with the production of the cytokines which recruit (IL-13) and activate (IL-5) eosinophils (n=18). Lymphocytes from healthy controls (n=15) did not demonstrate this immune response. We conclude that the T-lymphocytes in chronic sinusitis patients recruit eosinophils in response to fungal antigens, while T-lymphocytes in normal people do not. This underlying reaction to fungi occurred independent of IgE mediated allergy. Thus, the immunologic response is not IgE mediated allergy, and the term "allergic" in AFS is incorrect. As a consequence, the term Eosinophilic Fungal Rhinosinusitis (EFRS) was introduced.

Our working hypothesis of the immunologic mechanism of EFRS, based on the research findings in the laboratory, is that eosinophils are recruited as a defense to fight of fungi in the nose, where healthy controls are lacking this specific immunity. The eosinophils migrate through the nasal tissue and into the mucin of the nose. There the cells cluster around the fungi in a similar fashion as they group around

parasites. The eosinophils destroy the fungal organisms through the release of their toxic proteins. As a result, the mucin contains eosinophilic Major Basic Protein (MBP) in a quantity large enough to damage the nasal mucosa. This mucosal destruction allows residential nasal bacteria to secondarily invade the patient's mucosa and cause an acute exacerbation of chronic sinusitis.

Currently we are developing new treatments protocols based on our understanding of the etiology of CRS. Intranasal antifungals have been demonstrated to be safe and appear to demonstrate efficacy in open trials and are now tested in a double blinded, placebo-controlled fashion.

It should be mentioned that this non-invasive disease is a hypersensitivity to fungi, and not a fungal infection. EFRS needs to be differentiated from other forms of fungal sinusitis, such as fungus balls (non-invasive) and invasive fungal sinusitis (acute fulminant or chronic form).

A most striking finding for us is the fact that the T-lymphocytes of chronic sinusitis patients are sensitized in the peripheral blood and recruit and activate eosinophils when they sense a fungal antigen. This finding indicates that CRS is a systemic hypersensitive disease. Further research into the pathophysiology of CRS along this new paradigm will hopefully lead us to new treatments and ultimately better care for our patients.

Legends

Numerous eosinophils cluster around a fungal hyphae in cross section (arrow) in the mucin of CRS patient (Transmission Electron Microscopy x 7125)

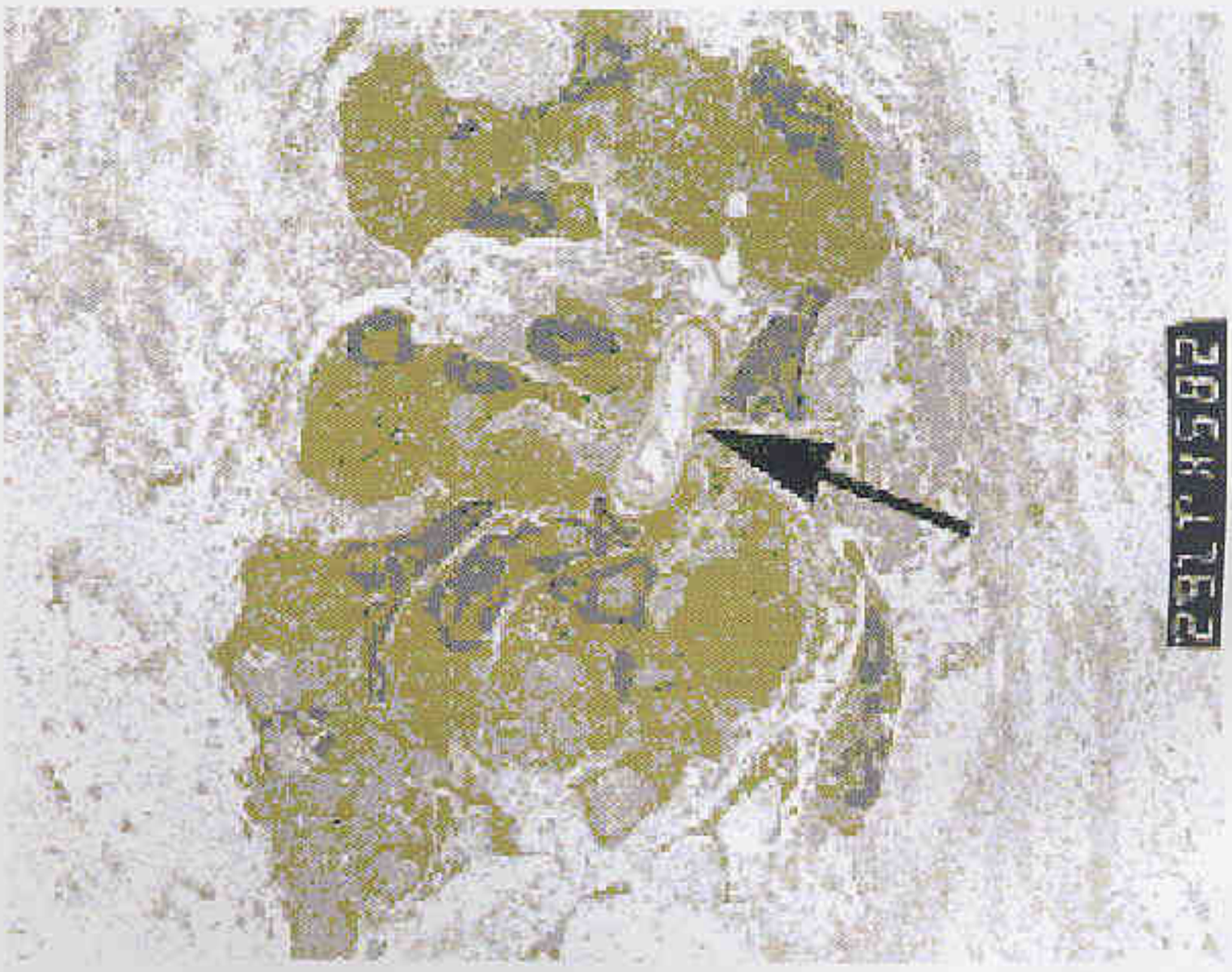

Fig. 1

CONTRIBUTION TO THE PATHOGENESIS AND TREATMENT OF CHRONIC POLYPOUS RHINOSINUSITIS

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Chronic polypous rhinosinusitis

Polypoid disease of the nasal respiratory mucosa occurs in a large number of patients with chronic rhinosinusitis (CRS). It is mostly referred as a multifactorial disease which has been variously defined by far. It seems justified to use the term rhinosinusitis as rhinitis and sinusitis are very often a continuum of one disease (1). The heterogenity of the various definitions is based on the use of different criteria. One well accepted definition of CRS, which includes patients with nasal polyposis, was proposed by the International Conference on Sinus Disease in 1993, where criteria for chronic rhinosinusitis were persistent symptoms and signs for eight weeks or four episodes per year of recurrent acute sinusitis, each lasting at least 10 days, in association with persistent changes on the computed tomography (CT) scan four weeks after medical treatment without intervening acute infection (2). Recently published data have proposed a common etiologic pathway for all clinical variations of polypous CRS, describing a systemic immunologic response to fungal antigens in sinonasal mucus as a causative mechanism (3).

Epidemiologically CRS appears to be of increasing relevance. Kennedy reported in 1994 that patient visits due to chronic sinusitis showed an increase of eight million to a total of 24 million per year between 1989 and 1992 (4). The prevalence of the disease was estimated at 14 % of the overall population in the USA in a study published in 1997 (5). It has also been described that CRS is positively correlated to particular diseases, for example CRS is found in 25-30% of all allergic patients (6), in 43% of asthmatic patients, in 37% of patients with transplants and in 54 - 68% of patients with AIDS (7).

Therapeutical challenge

The gold standard of treatment for CRS and nasal polyposis is surgical therapy, often in combination with topical and systemic medical treatment. Regardless of the surgical technique applied, a fair amount of patients with polypoid changes will at some point in time present with recurrent disease. Given the numerous factors involved in this process, there are basically three problems responsible for the recurrence rate found in these patients: 1.) inadequate previous surgery, 2.) technically unavoidable recurrences and 3.) incomplete etiological exploration.

In an effort to minimize the rate of complications and / or recurrences, these three problems need to be addressed in the therapeutical concept. State of the art surgical

technique includes the ability to combine microscopic and endoscopic procedures in one operation. Whether the surgeon prefers one or the other technique as a standard one certainly is related to personal experience using either of them. However, the combination of both appears to be advantageous especially in difficult revision cases, e.g. in the treatment of the frontal sinus when recurrences sometimes appear to be technically unavoidable as they might be related to persistent inflammatory processes and quick recurrence of polyps, scar tissue formation and / or formation of mucoceles. In order to minimize surgical time as well as intraoperative anatomical / topographic uncertainties due to the presence of scaring and hyperostosis or the absence of anatomical landmarks after previous interventions, computer aided navigational devices have been proven to be potentially helpful. A survey analyzing 422 cases, where computer aided surgery was used as an intraoperative navigation device revealed an average inaccuracy of less than 2 mm. The set-up time for the equipment was averaged at less than 10 min, technical malfunctions were observed in 26 cases and no surgical complications occured related to the use of any of the five CAS systems used.

Complete diagnostic exploration

A more complex problem is the one of an incomplete exploration of patients suffering from polypous CRS. It is crucial to evaluate the relevance of several possible etiological factors and pinpoint those that play a role in the individual pathogenetic constellation of a given patient. In a complete workup screening should include the following aspects: inhalant allergies, ciliary dysfunction, aspirin intolerance, immunodeficiancy, the presence of fungal antigens and / or an immunologic reaction to fungi as well as the presence of bacteria resistant to the applied antibiotic treatment.

Testing for inhalant allergies includes prick-, intracutaneous- and RAST – testing, followed by nasal provocation testing, if clinically indicated. It has been shown that specific immunotherapy is helpful in reducing recurrence rates after sinunasal surgery.

Dysfunction of mucociliary clearance can be detected using a combination of two diagnostic tools. One is the clinical test of saccharin transport time (STT), where a peace of saccharin is placed on the head of the inferior turbinate and time is taken until a clear sweet sensation is felt in the pharynx. Physiologically a sweet sensation will be noticed after 8 to 20 minutes. The functional integrity of the ciliary cells as motor units can be identified with the help of video interference contrast microscopy. This is a very valuable method to rule out syndromes of primary ciliary dyskinesia. Any impairment of mucociliary transport can be clinically relevant, as persistent stasis of the mucus blanket will trigger recurrent rhinosinusitis. In a study published in 1999 we showed that a majority of patients suffering from recurrent CRS do have a prolonged STT, but rarely a significant decrease in ciliary activity. These observations could be explained either by poor coordination of ciliary activity in these patients and / or alterations of the nasal mucus in terms of viscosity and its content of enzymes and / or inflammatory mediators.

Role of eosinophilia

Despite the mentioned heterogenic entities, that possibly are related to, or associated with the etiology of polypous CRS, one aspect appears to be universal for all variations of the disease. The polypoid tissue of the inflamed sinunasal mucosa shows a great amount of eosinophils on histologic examination in the vast majority of cases. Davidsson reported increased number of eosinofils in tissue specimens of 88% of cases (8). A study from our group showed an increase of eosinophilia in patients with inhalant allergies when compared to non allergic patients. A comparable increase was seen in aspirin intolerant patients. Interestingly the highest levels of eosinophilia were found in patients suffering from both inhalant allergies and aspirin intolerance (9). Functionally eosinophilic granulocytes are known to carry a variety of cytotoxic proteins in their granula, which can be released by degranulation. These proteins include the eosinophilic cationic protein (ECP), major basic protein (MBP), eosinophilic peroxidase (EPO) and eosinophil derived neurotoxin (EDN). ECP for example is known to be a reliable marker for inflammatory activity in various types of rhinitis (fig 1).

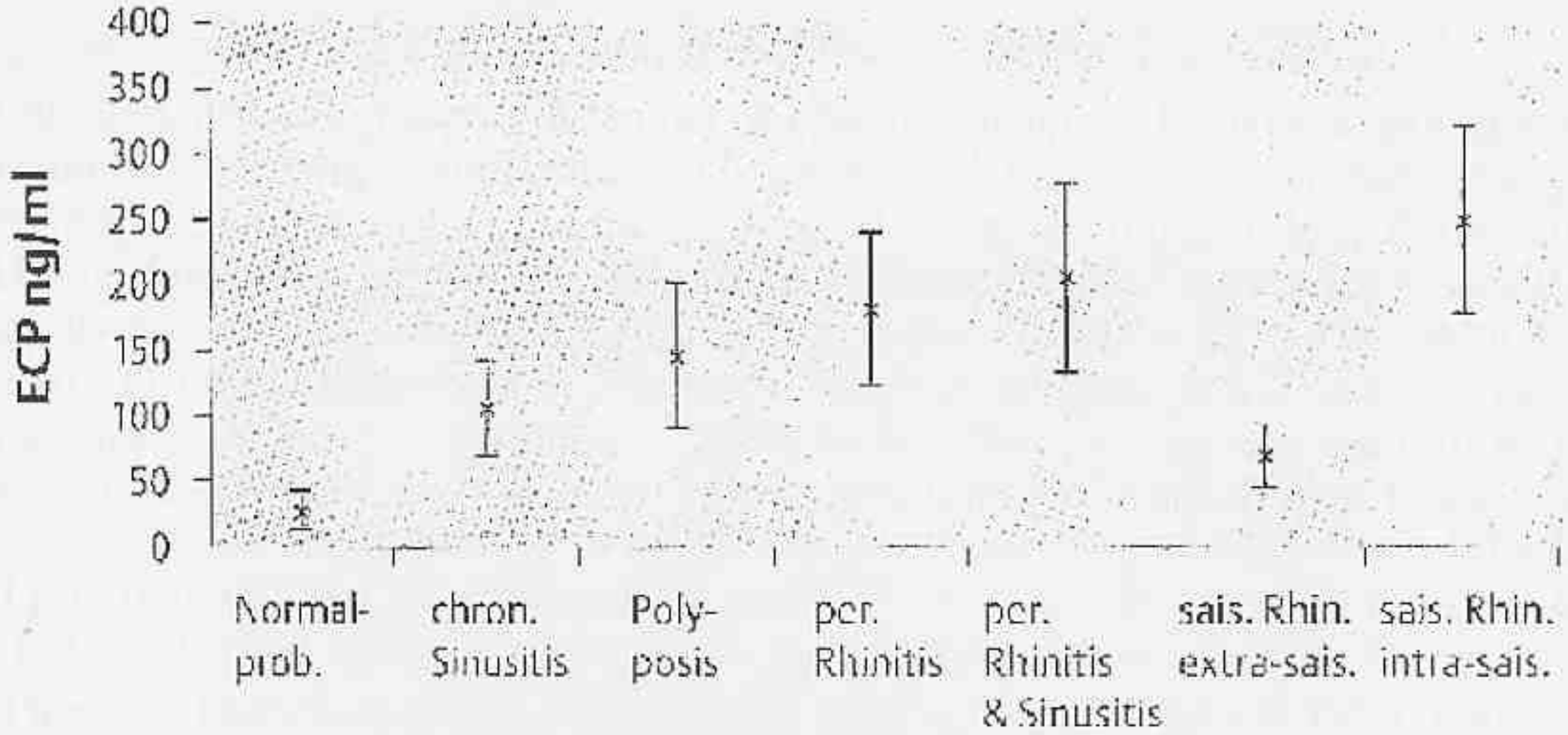

Fig 1: Concentrations of ECP (ng/ml) in various forms of rhinosinusitis, compared to healthy controls.

However, recently published data suggest, that cytotoxicity of the eosinophils is a crucial element of an immunologic reaction against fungi, which are present in the nasal mucus (3). The proposed paradigm for a common etiology of all various types of CRS postulates that eosinophils leave the nasal tissue and migrate into nasal mucus, where they degranulate and unload their toxic proteins, especially MBP, onto fungal elements. This was shown morphologically by examining nasal mucus histologically. Using H.E. staining, eosinophilic clusters were detectable in these specimens. Silver staining as well as a newly developed chitinase staining unveiled the presence of fungal elements within these clusters (fig 2).

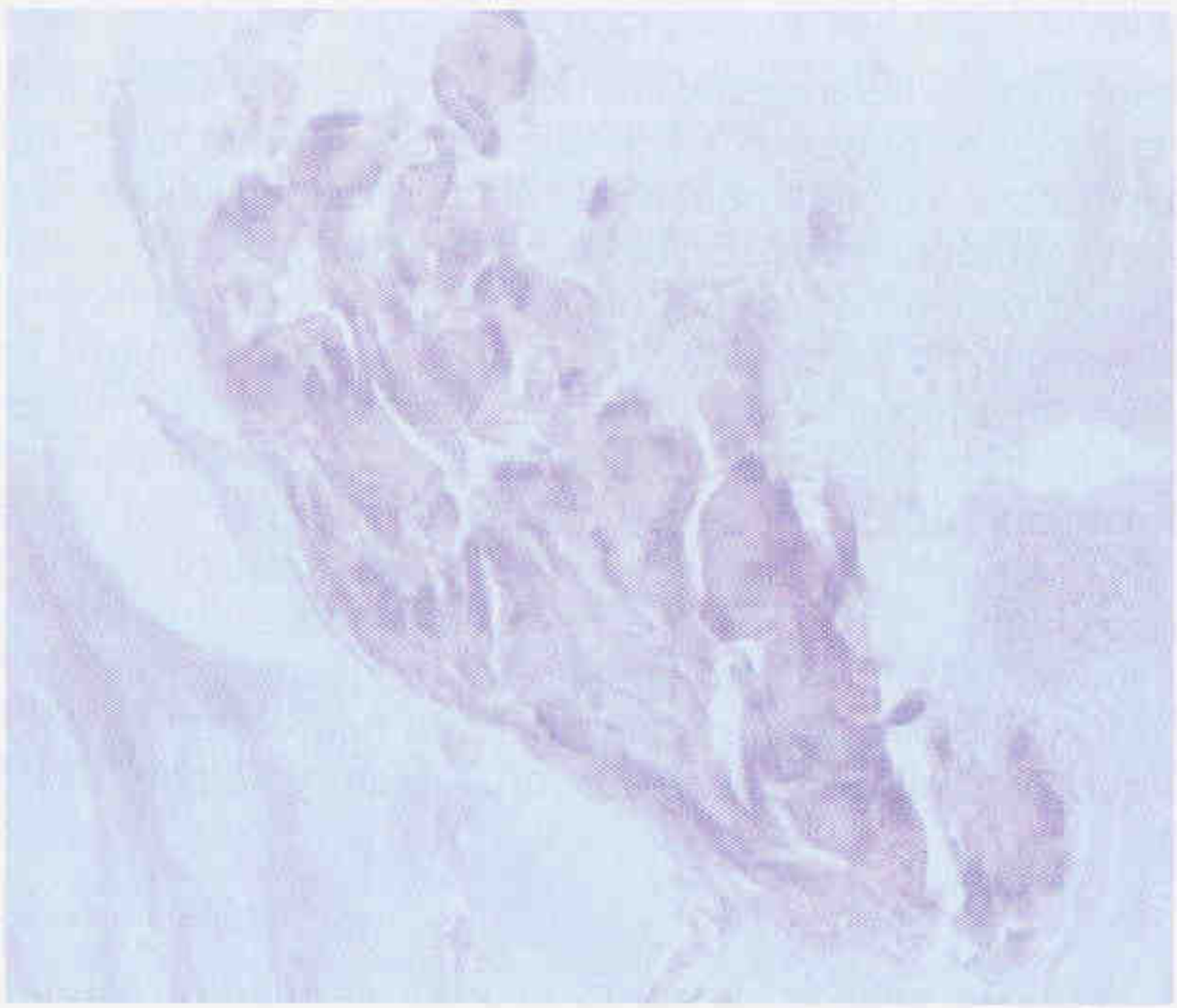

Fig. 2a

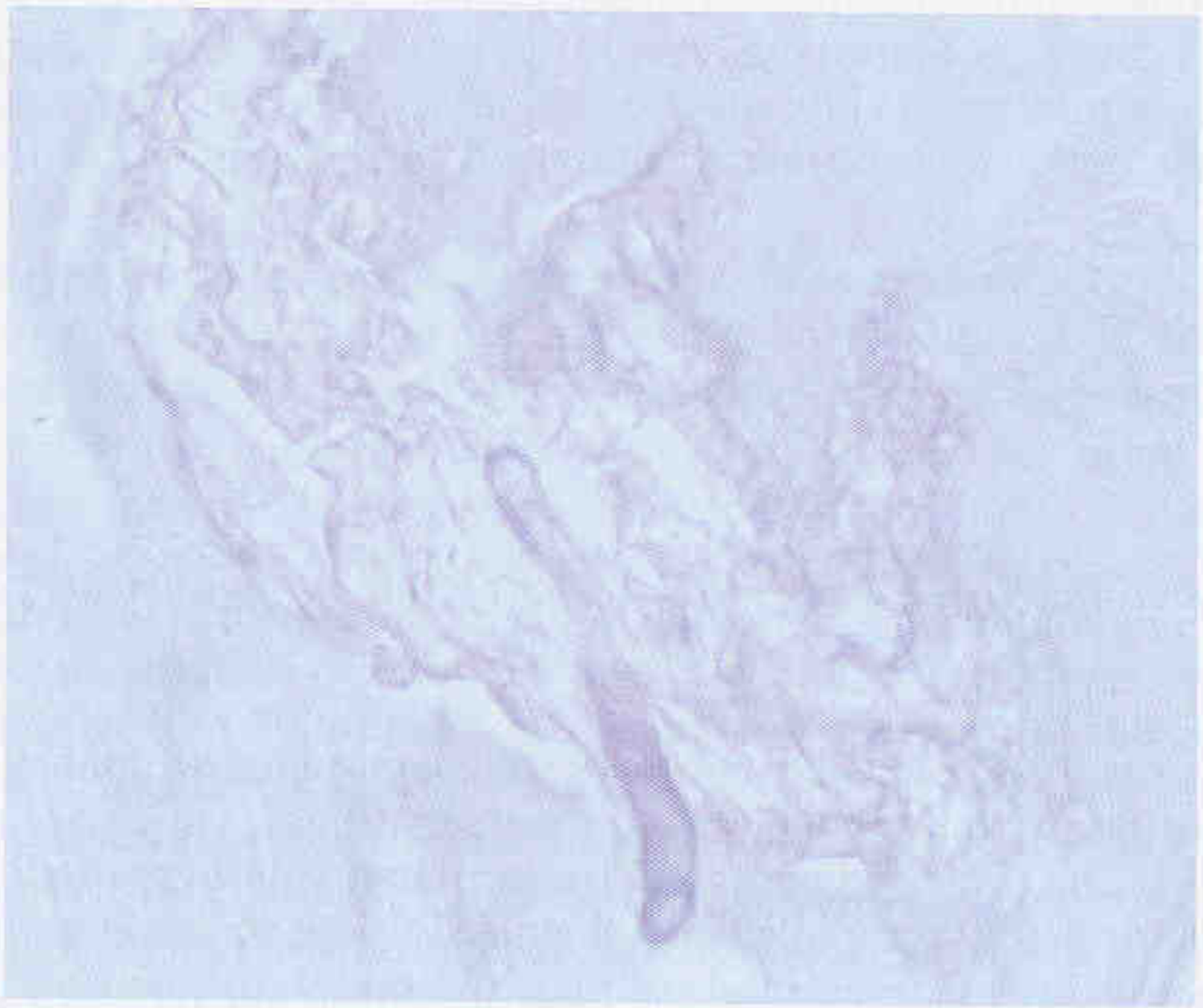

Fig. 2b

Fig 2: While the top image (H.E. –staining) shows an eosinophilic cluster in nasal mucus, the bottom image (silver staining) clearly shows fungal elements within the cluster.

It was furthermore concluded that this eosinophilic, cytotoxic mucin would lead to secondary epithelial damage in the respiratory lining, promoting bacterial invasion and infections, explaining the recurrent, acute bacterial infections in these patients. This theory was based on the observation that fungi are essentially present in 100 % of cultures taken from human nasal mucus. In this study fungal cultures were positive in 98 % of 202 patients and in 100% of 14 controls. A total of 41 species were found, up to 8 in one sample / patient. The most common were Alternaria (48%), Penicillium (42%), Candida (28%) and Aspergillus (17%). Ponikau et al. concluded from these findings that the term chronic sinusitis should be replaced by eosinophilic fungal rhinosinusitis (EFRS). Own unpublished data recently revealed that using the highly sensitive method of polymerase chain reaction (PCR) fungal DNA can also be detected within sinonasal tissue specimens. Surgical specimens of nasal polyps were treated with a solution of dithiotreitol (DTT) in order to digest any nasal mucus and ensure that only tissue was examined and a two step PCR was applied. Using both one universal primer for unspecific detection of fungal elements and a second primer pair specific for alternaria, all 30 samples were tested positive. The possible relevance of this finding in the etiology of polypous CRS needs to be discussed.

Diagnosis and treatment of aspirin intolerance

One very critical group in the range of patients suffering from nasal polyposis are individuals with aspirin intolerance. It is known that this particular patient group has a very high risk of recurrence of their sinonasal polyposis independent of the number and kind of previous surgical interventions. The diagnosis of aspirin intolerance (AI) is not always associated with the full clinical picture of the aspirin triad, which consists of: 1) nasal polyposis, 2) intrinsic bronchial asthma, and 3) aspirin induced worsening of asthmatic symptoms, often along with naso-ocular symptoms (10). However, in sensitive individuals even very small single doses of aspirin may cause rhinorhea, bronchiolar constriction and shock-symptoms related to a non IgE-mediated pharmacological hypersensitivity reaction (11). It is known that not only aspirin, but most other NSAID interact with the eicosanoid pathway (fig 3).

They are known to cause inhibition of the cyclooxygenases (COX), which metabolize arachidonic acid to prostaglandins. This inhibition leads to an up-regulation of the alternative pathway with lipoxygenases metabolizing arachidonic acid to leukotriens. An in vitro assay can be very valuable in establishing the diagnosis of AI (12). The alteration of arachidonic acid metabolism and eicosanoid release can as well be detected in patients with an incomplete manifestation where the clinical picture of the aspirin triad has not yet fully developed. This assay has been successfully used at our institution, following a recently published protocol (13). Considering the role of COX in the inflammatory process in nasal polyps, own unpublished data from recent histochemical studies indicate that both isoforms, COX 1 and COX 2 are mostly located in structures of the respiratory epithelial lining and also to a large extent in epithelial cells of glandular tubuli. The examined specimens were taken from patients with nasal polyposis. In contrast COX was hardly found in stroma or vascular structures. This observation clearly gives an important insight into the his-

topathology of the inflammatory disease as it indicates which structures are mainly involved in prostaglandin production within polypoid tissue. However, it is not yet known weather prostaglandin production in tubuli might indicate a secretion of these molecules into nasal mucus.

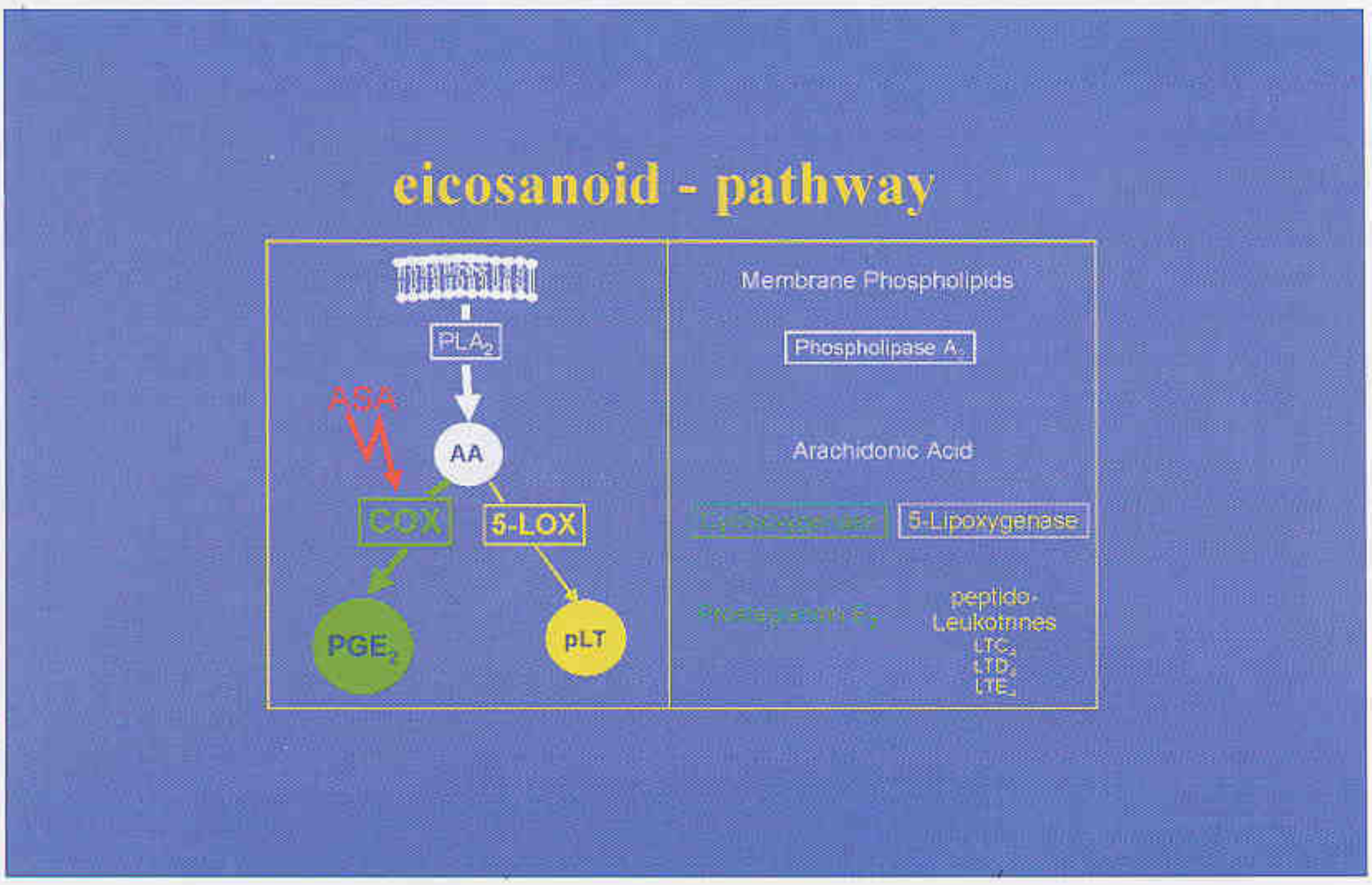

Fig 3: Schematic depiction of the eicosanoid pathway. ASA inhibits metabolism of arachidonic acid to PGE2 via COX and therefore leads to a relative overproduction of pLT.

All patients, who are diagnosed with AI, have a considerable chance of improvement or decreased risk of recurrence, if adaptive desensitization therapy is performed. In a recent prospective study we were able to show the effectiveness of a new protocol using a maintenance dose of only 100 mg of oral aspirin a day (14). To initiate the desensitization therapy oral aspirin was given in increasing dosages over two days (day 1: 100 mg (two doses of 50 mg), day 2: 500 mg). Patients were hospitalized for this therapy. Airway resistance and FEV1 were closely monitored during this induction period. On the first day after an initial lung function test 50 mg was given in the morning and only after a repeated check of airway resistance and FEV1 the second 50 mg were administered orally, usually 8 hours after the initial dose. On the second day 500 mg were given orally if repeated lung function test had not revealed a decrease in FEV1 of 25% or greater. On the third day aspirin was reduced to the daily maintenance dose of 100 mg to be given for at least nine months. Clinical reassessment as well as the functional in vitro assay were repeated at each follow up visit of every patient in an attempt to identify changes in the release of eicosanoids over time and to correlate these with the clinical course. Figure 4 details the in vitro findings in a group of patients following this desensitization protocol over 1 year. Since there is

a relative overproduction of pLT in aspirin sensitive individuals, it is desirable to achieve an increase of the "PGE2/pLT index" over time. Table 1 details the recurrence rate of nasal polyps observed in the same group of patients.

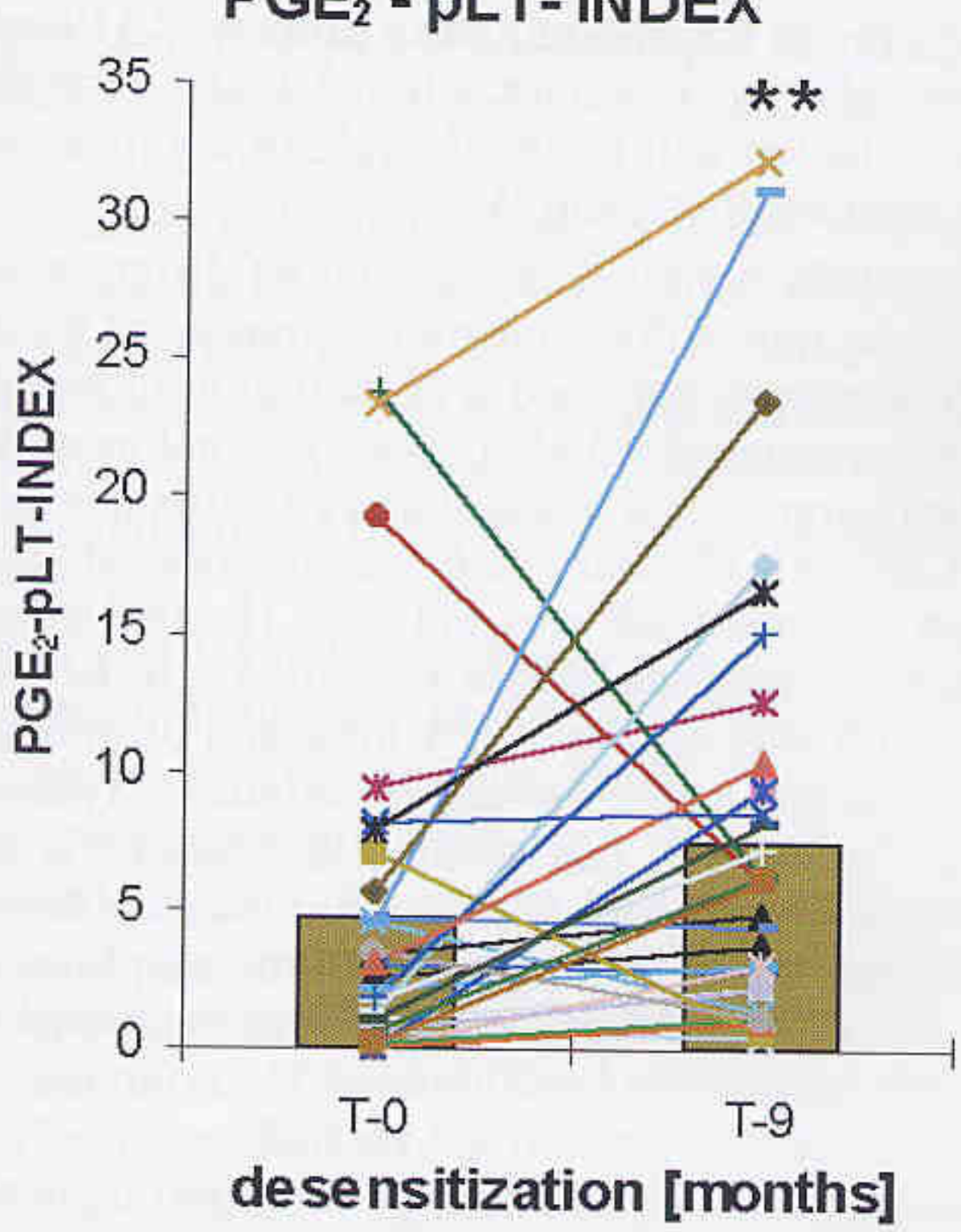

Fig 4: PGE2/pLT index before (T0) and nine months (T9) after initiating the desensitization therapy in 30 patients. Lines show the individual courses of the 30 patients, boxes indicate mean values. Significant changes ($p<0.05$) are marked with a star, highly significant changes ($p<0.01$) are marked with two stars.

Table 1. endoscopic assessment of nasal polyposis after nine months (n=30)

no recurrence (free of polyps throughout desensitization)	n=14
marked improvement (minor disease before desensitization)	n=9*
steady state (minor disease before desensitization)	n=2
marked improvement (major disease before desensitization)	n=3#
progressive disease (major disease before desensitization)	n=2

*: including two patients who underwent simultaneous antiallergic hyposensitization

#: including one patient who underwent simultaneous antiallergic hyposensitization

Table: Endoscopic assessment of nasal polyps in 30 patients after nine months of desensitization therapy. 5 patients did not improve clinically, only 2 of these had progressive recurrent polyposis.

Conservative treatment

Taking all the above mentioned aspects into consideration, the following therapeutic options can be valuable in reducing the risk of recurrent nasal polyposis and especially in patients who previously underwent one or multiple surgical interventions. 1.) steroids, used topically, systemically or both, generally have a strong antinflammatory effect and can reduce eosinophilia as they directly interact with several chemokines and cytokines involved in the inflammatory process. 2.) Patients with aspirin intolerance should undergo adaptive desensitization. 3.) First controlled clinical studies, which are currently conducted will define the role of a topical antifungal treatment, using nasal washes with amphotericin B.

In a recently published prospective study we evaluated effects of different concentrations of several topical solutions on mucociliary clearance, measured by ciliary beat frequency over time (15). In controls, perfused with cell culture medium (RPMI) only, CBF was measured at an average of 9.5 Hz (+/- 1.7), which remained constant over more than 12 hours. Perfusion with a 5% solution of ofloxacin as an antibiotic solution led to an average CBF of 8 Hz, but ciliary activity ceased after 7 hours. With a 50% ofloxacin solution average CBF was only 7.5 Hz and stopped after 6 hours and 30 min. Using antiseptic solutions, perfusion with 5% of betadine revealed an average CBF of 7 Hz, which was kept up for 1 hour and 30 minutes, however, with 10% it was down to 4.5 Hz and lasted for only 30 minutes. Hydrogen peroxide was used in a 1% and in a 3% solution and seemed less ciliotoxic than betadine as 1% led to an average CBF of 7 Hz, which was kept up for over 8 hours and 3% to 6 Hz for 5 hours and 30 minutes. Using antifungal solutions, amphotericin B revealed only little ciliotoxicity in low concentrations, as CBF was measured at 9 Hz for 8 hours at a concentration of 2.5% and at 8Hz for 7 hours at 5%. After increasing the concentration to a 10% solution, CBF dropped to 3.5 Hz and lasted only 2h. Interestingly, there was no dose dependant effect to be observed after perfusion with clotrimazole at all three chosen concentrations of 10%, 20% and 50%. CBF remained at a constant average frequency of 9 Hz, but CBF stopped after no more than 30 minutes in all experiments. The strongest dose dependence was seen for itraconazole: At a concentration of .25% a CBF of 6 Hz lasted for 7 hours and 45 minutes, at .5% ciliary activity went down to 1 hour and 15 minutes at 6 Hz and at 1% it was only 3 Hz for 30 minutes.

Conclusion

Due to the high risk of recurrence of disease in patients suffering from polypous chronic rhinosinusitis surgical treatment can only serve as one important aspect of a rather complex therapeutical concept. This concept needs to be tailored with respect to the heterogeneous aspects of the etiology of disease in this particular group of patients

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MINIMALLY INVASIVE ENDOSCOPIC SURGERY FOR NASAL POLYPOSIS

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The term 'minimally invasive endoscopic sinus surgery' (MIESS) was probably first coined by D.Parsons and R.Setliff [3,5] in the early 90's of the XX century. Rational of MIESS utilized both Messerklinger's basic concept of FESS and later technical achievements such as soft tissue shavers. As applied to the treatment of inflammatory sinus disease, the concept and technique of MIESS have been accepted and widely used. In contrast, vast majority of endoscopic sinus surgeons believe that in case of nasal polyposis more aggressive surgical technique should be applied.

Goals of MIESS are:

- to eliminate pathology, i.e. remove polyps,
- to restore nasal breathing, normal ventilation of the diseased sinuses,
- to preserve lateral nasal wall anatomy as well as mucous transport pathways and lymphatic drainage of the sinuses.

Nasal polyposis is considered mostly a medical but not a surgical disease with unknown pathogenesis [4]. If surgical treatment is indicated, it should follow the principles listed above maybe even more than in case of chronic sinusitis. Extensive surgery and resection of nasal turbinates create big cavities, which sooner or later will be inevitably filled with new polyps. Trimming of the sinuses' ostia cuts lymphatic vessels coming from the sinus mucosa and produces post-operative edema. Everyone performing post-operative endoscopy of the maxillary sinus routinely could see how early postoperative edematous changes persist and transform later into true polyps after wide middle meatal antrostomy.

In nasal polyposis, surgery is only a part of complex treatment. Another more important part is medical therapy, which must precede and follow the surgery. Ideal surgery for nasal polyposis must preserve anatomical structures (turbinates, ground lamella, sinuses' ostia) and provide enough relief to initiate return to health on the basis of continuing medical therapy. Opening of transitional spaces (pre-chambers) of the diseased sinuses allows topical medications prescribed post-operatively (for instance, intranasal corticosteroids) to enter the sinus and to reach and treat more effectively edematous mucosa.

Development of soft tissue shavers made surgery for nasal polyps less invasive. The use of the shaver instead of a snare or forceps allows for precise step-by-step visualization and preservation of all lateral nasal wall anatomical structures and very delicate removal of the polypoid mucosa without damaging bone and periostium. This makes shaver not just a surgeon's toy but brings a new philosophy to the endonasal surgery.

Basic technique of MIESS for nasal polyposis can hardly be standardized because it varies significantly depending on the extent of disease itself and radicalism

of previous surgeries. However, standard steps of a primary surgery for nasal polyposis can be described.

Step 1. Visualization of the posterior edge of uncinate process, retrograde wedge resection of the process with the use of a backbiter and shaver (fig.1).

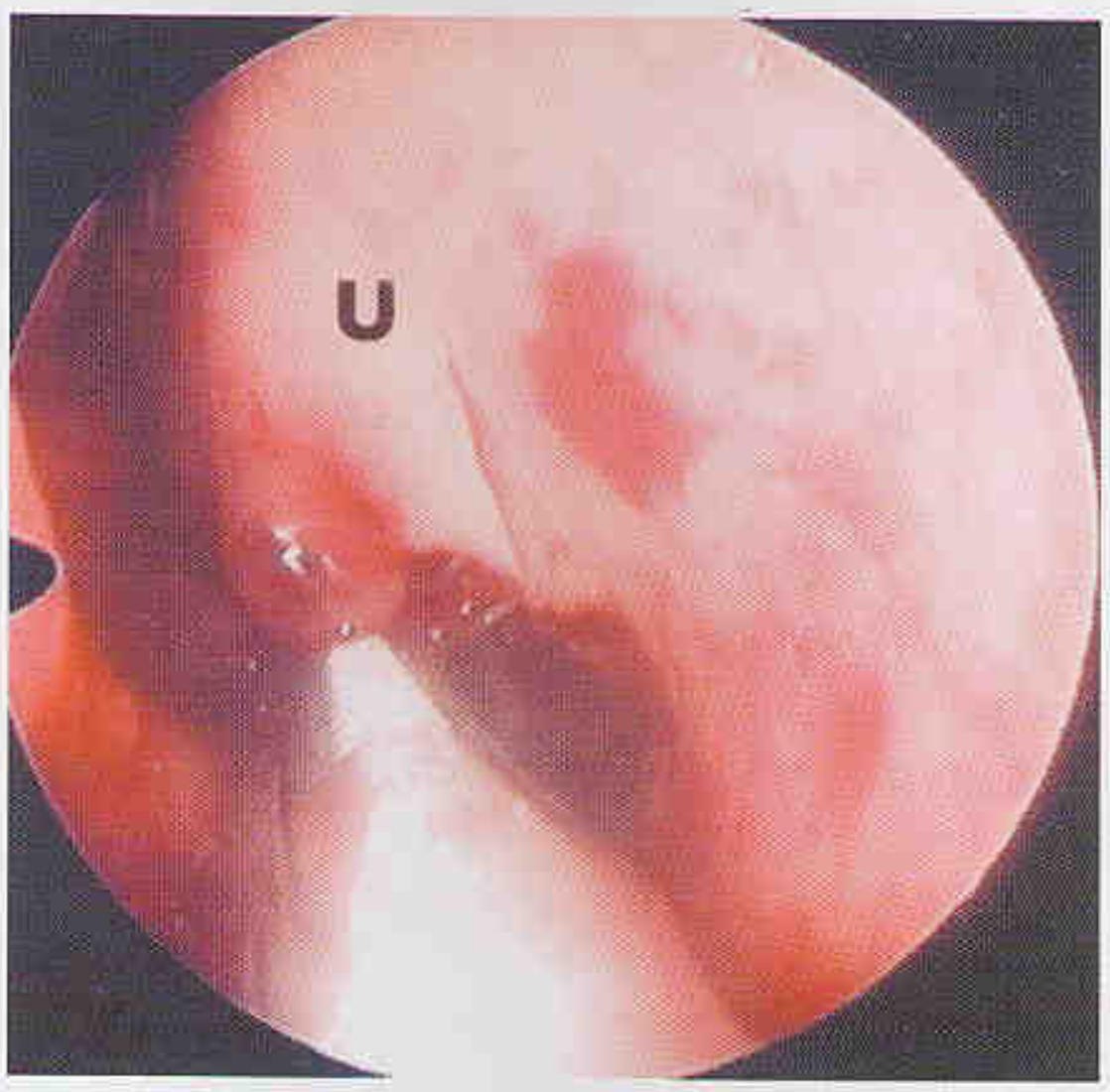

Fig. 1

This allows for identification of the natural maxillary ostium and mucous pathway on the surface of the posterior fontanel (fig.2).

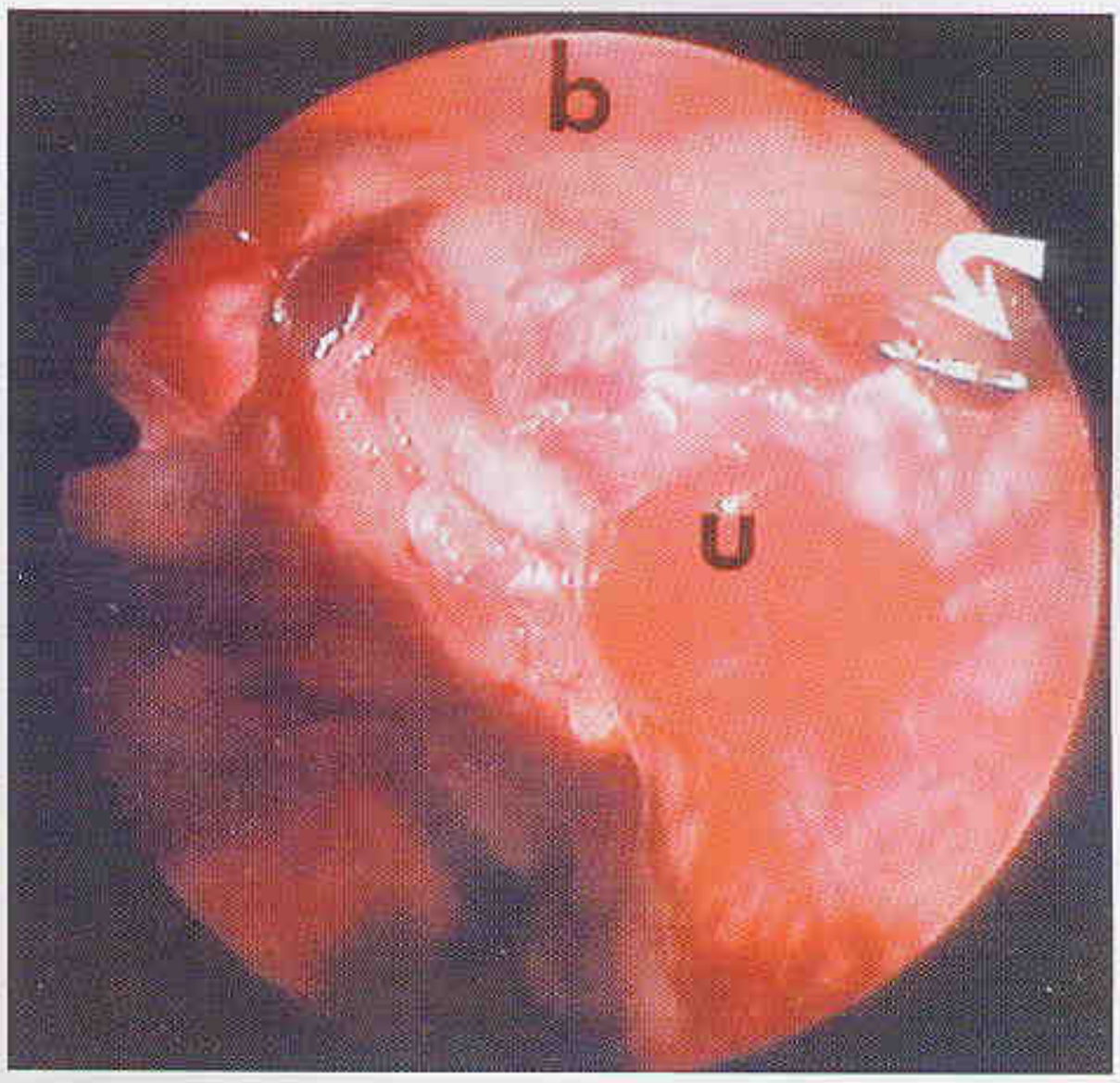

Fig. 2

Cleaning of the medial aspect of the natural maxillary ostium and the fontanel is performed with the shaver (fig.3).

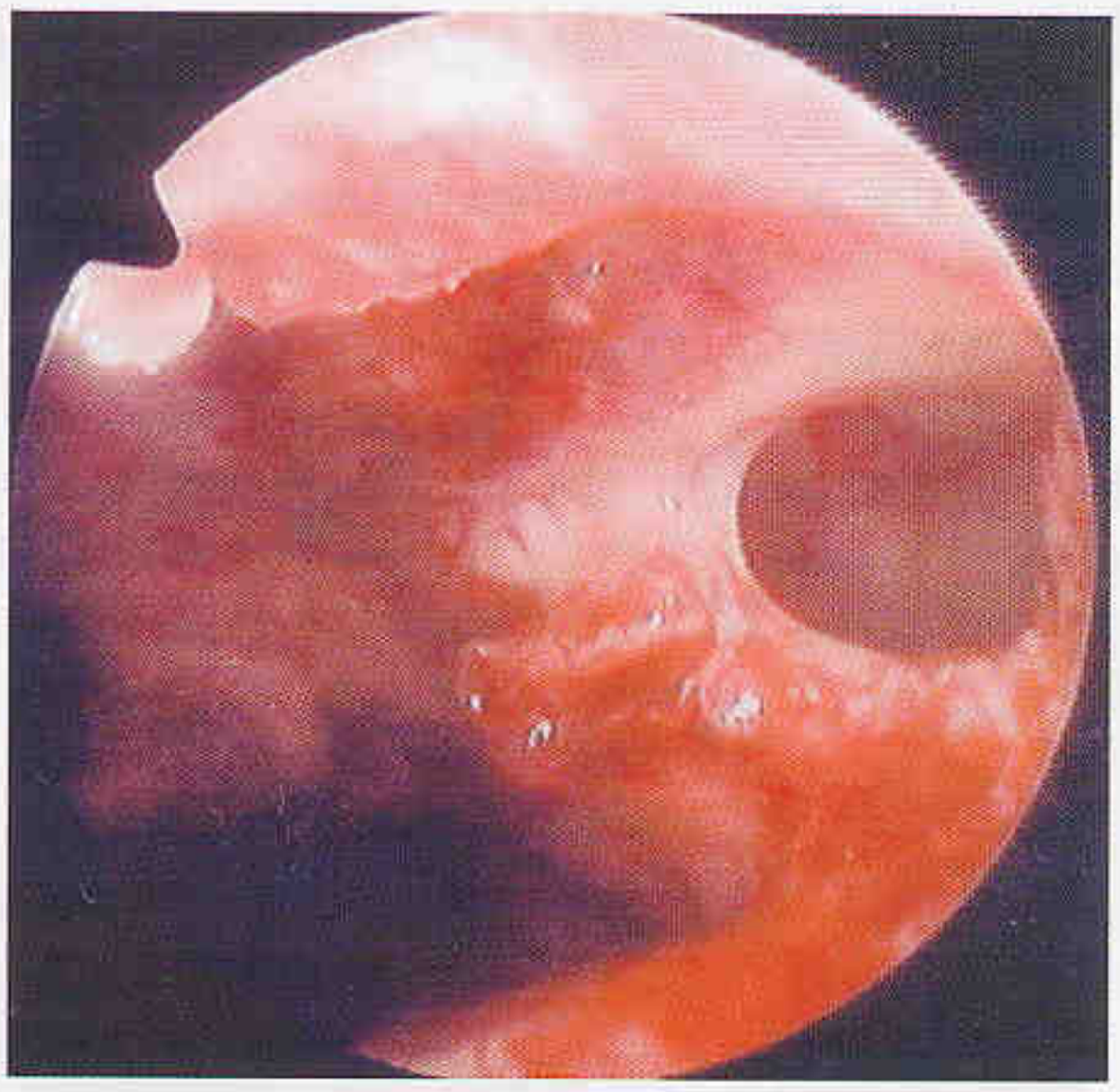

Fig. 3

Step 2. In presence of accessory maxillary ostium, bridge between natural and accessory ostia is trimmed and both ostia are united. If size of the ostium is small (less than 3 mm) and if the sinus itself requires exploration, horizontal transection of the posterior fontanel is performed. Removal of sinus secretion is performed with a curved suction tip, 80ş curved double spoon forceps are used for removal of true polyps and cysts from the maxillary sinus under control of a 30ş endoscope.

Step 3. Ethmoidal bulla and agger nasi cell are opened; their bony walls are removed using straight and 90ş forceps. In primary surgery, frontal sinus ostium can be easily identified behind the agger nasi cell (fig.4).

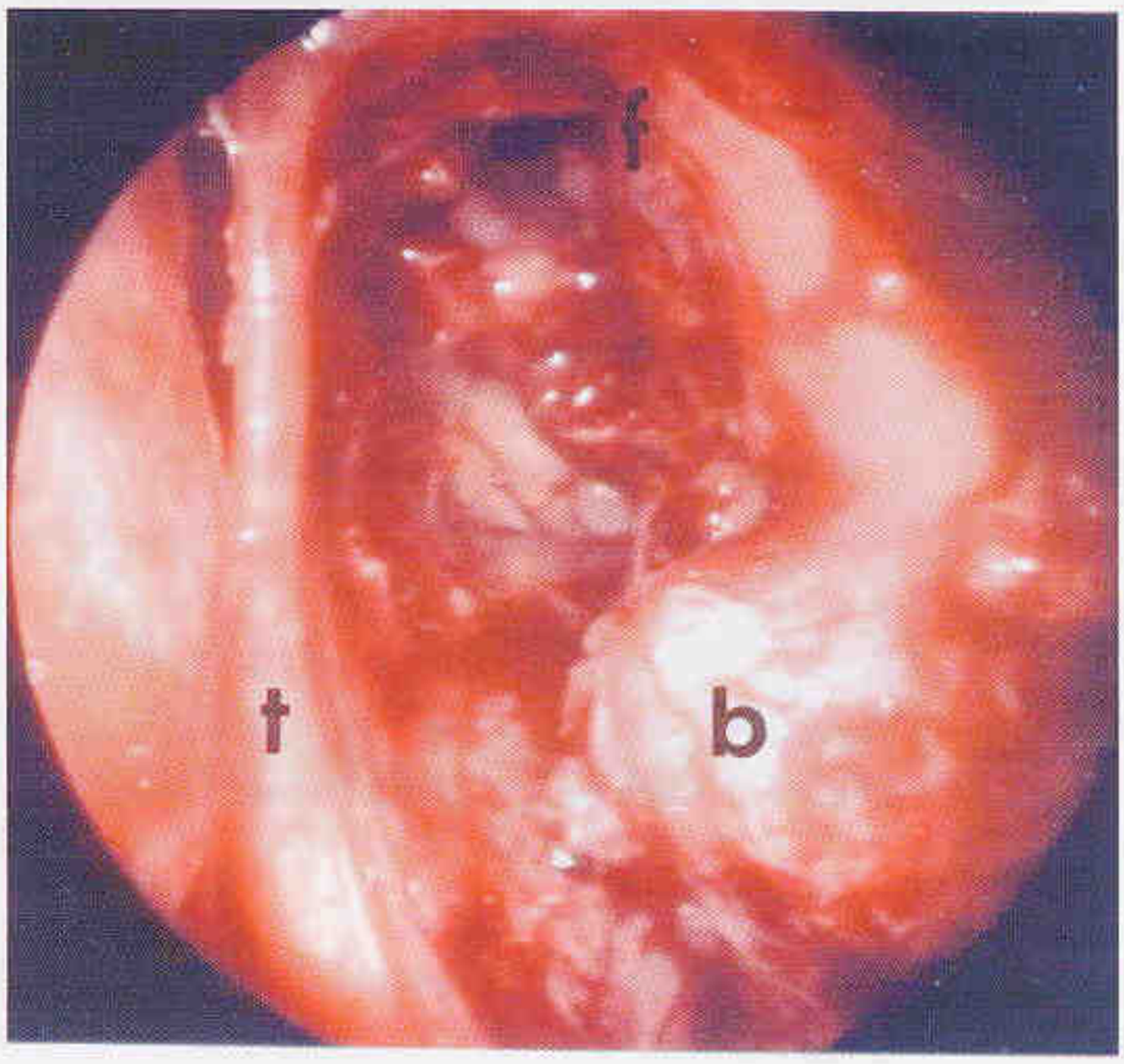

Fig. 4

No manipulations on the ostium and inside the sinus are normally needed. Frontal sinus secretion can be evacuated with a curved suction. This step completes anterior ethmoidectomy.

Step 4. After lateralization of the middle turbinate, upper meatus and sphenoethmoidal recess are examined. Polyps, if present, are removed with the use of the shaver. The latter allows for identification of the superior turbinate and the ostia of the posterior ethmoid and sphenoid sinus. If needed, the ostia can be enlarged without resection of the upper turbinate (fig.5).

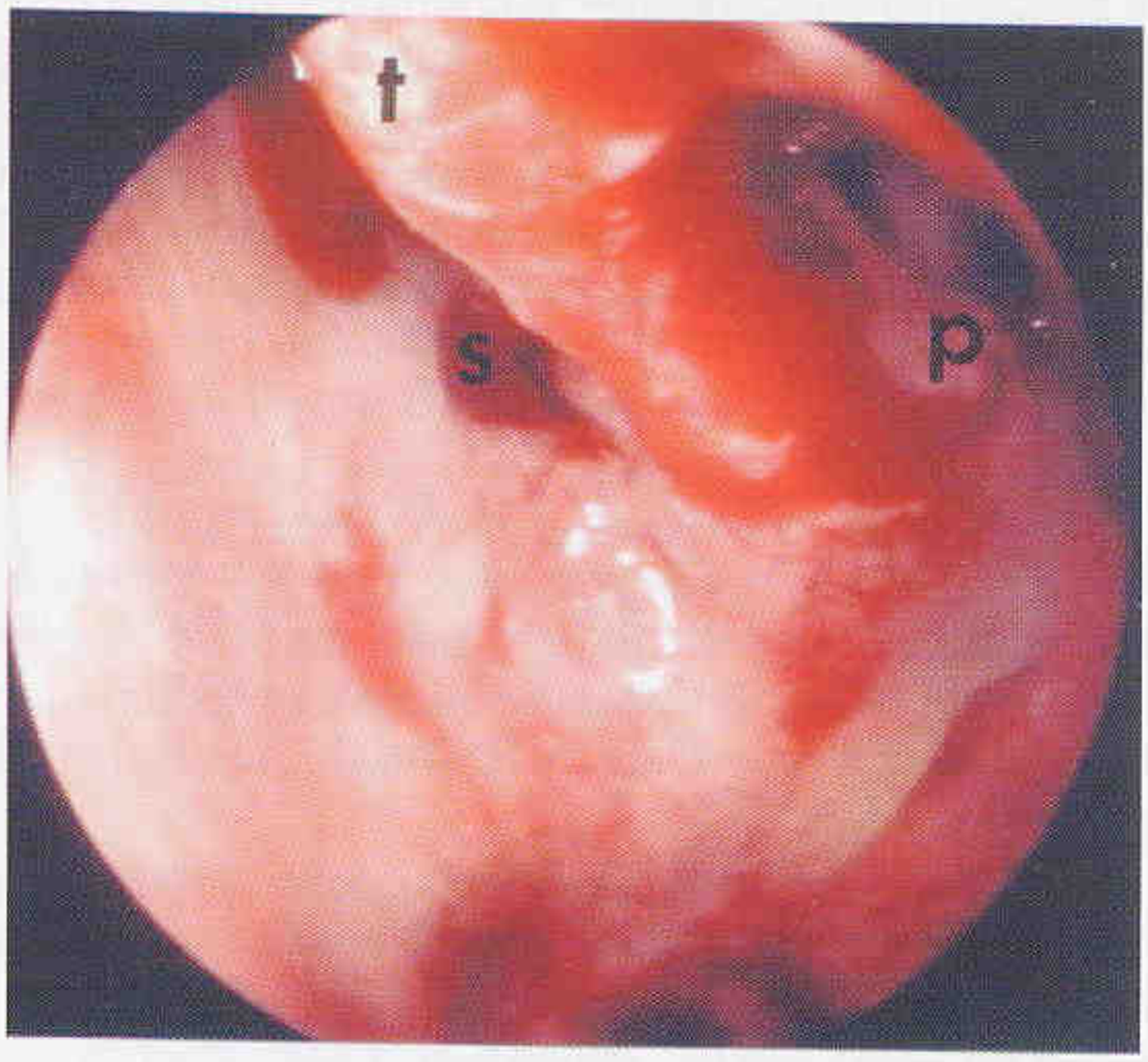

Fig. 5

Step 5. Only in case of extended polyposis, ground lamella of the middle turbinate has to be perforated to facilitate complete polyps' removal from the posterior ethmoid.

This is a protocol of 'ideal' surgery, which can only be performed in presence of anatomical structures of the lateral nasal wall. It means, this should be a primary intervention or surgery performed after previous simple polypectomies. This surgery reconstitutes normality, saves all nasal turbinates, ground lamella, and considers approach to diseased 'large' sinuses via their natural ostia. Resection of the ostia edges should be just enough to facilitate necessary manipulations inside diseased sinus.

In revision cases after previous ethmoidectomy, surgeon faces large open cavities filled with polyps, secretions and scar tissue, middle and upper turbinates are often missed, 'large' sinuses open, their surgically created ostia blocked with polyps. Extent of this surgery is greatly influenced by radicalism of previous surgeon(s). Moreover, there are situations when even in primary case diseased sinuses must be widely

opened, i.e. for complete removal of a solid antral part of antrochoanal polyp or sticky secretion in case of eosinophilic fungal sinusitis.

The question 'whether or not diseased paranasal sinuses must be widely opened and polypoid mucosa removed from them as much as possible?' has no clear answer. There is a myriad of surgical techniques applied in treatment of nasal polyposis ranging from radical sphenoetmoidectomy with amputation of middle turbinates (so called "nasalisaiton") [1] to "mini-FESS" and simple polypectomy procedures.

To compare distant results of MIESS and more extended routine technique of sinus surgery, we conducted a prospective randomized study in 65 patients operated with either techniques. The patients were examined pre-operatively and reassessed after 6 month and during distant follow-up visits using symptom-, CT- and endoscopic findings score as well as saccharine transport time [2]. Surgical outcome and symptomatology were similar in both groups of patients indicating that conservative approach is sufficient and obtains at least same results as more extensive surgery.

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4. Position statement on nasal polyps. Rhinology 1994; 32: 126.
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Legends

Steps of MIESS for nasal polyposis

Fig.1. Resection of the inferior part of the uncinate process with the backbiter, u – upper portion of the uncinate (30° scope).

Fig.2. Resection of the uncinate completed; b – lower aspect of the ethmoidal bulla, u – lower part of the uncinate, arrow – natural maxillary ostium (30° scope).

Fig.3. After polyps' removal edges of the ostium are preserved (30° scope).

Fig.4. Dissection of the frontal recess performed, t – middle turbinate, b – ethmoidal bulla, f – frontal sinus ostium (0° scope).

Fig.5. Polyps removed from the upper meatus and posterior ethmoid, t – superior turbinate, p – ostium of the posterior ethmoid, s – sphenoid sinus ostium (0° scope).

MAXI-NASAL POLYPOSIS

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The nasal polyposis has been mentioned in the ancient medical literature. The cause of nasal polyps has been studied and debated since they were first recognized by Ogawa in 1986. Even in old days, there existed surgical and medical treatment that we follow even today. As early as one thousand B.C., a type of curette for "eradicating nasal polyps" was used in India. Hippocrates used sponge for the removal of nasal polyps, Vancil 1969.

The etiopathogenesis of nasal polyps has long been a subject of study, yet there is little agreement as to the mechanism of polyp development. So far two main theories have emerged, allergic and infectious. Recently, sensitivity to aspirin has also been implicated as an etiologic factor, Baumgarten et all 1980.

Nasal polyps originate mainly in the maxillary and ethmoidal sinuses. The pedunculated polyps can be even seen by anterior rhinoscopy in the nasal cavity that indicates the progressive form of disease process. Nasal polyps may be solitary or multiple, unilateral or bilateral. Nasal polyps are seen in any age group but more commonly seen in adults and elderly patients, table no.1.

Table No.1
Nasal Polyps operated between 1996 to 2001:

Sex	0-10yrs	10-20yrs	20-30yrs	30-40yrs	40-50yrs	50-60yrs	Above60
Male	3	8	8	22	6	8	4
Female	1	3	4	7	3	2	1

With the introduction of endoscopic sinus surgery one can often see, after the removal of the uncinate process, an infundibulum and ethmoidal cells filled with polyps while anterior rhinoscopy did not show any evidence of polyps, Stammberger, 1986.

Modern technology, CT scanning and endoscopy enables us to make an early and appropriate diagnosis of nasal polyposis. The number of nasal polyposis patients is much larger than what could have been expected after simple anterior rhinoscopy.

An early diagnosis will enable the clinician to control and eradicate the disease process to restore nasal function through less extensive therapy. The pre operative imaging by CT and nasal endoscopy is mandatory to plan for precise, safe and successful surgery of massive or recurrent nasal polyposis, Sood 1994. Patients present in different stages of disease process, table no.2.

Table No.2:

Clinical manifestation and extension of disease in nasal polyposis:

Bilateral Disease	Unilateral Disease	Proptosis	Intracranial exten.
56	24	12	3

In long standing infective polyposis most of the time there is bony necrosis of medial wall of maxillary sinus, lamina papiracea, ethmoid cells and anterior wall of the sphenoid sinuses. The long standing polyps degenerate to form into cheesy and muddy material, a condition referred to as necrotizing ethmoiditis which should rather be called necrotising sinusitis since disease process is not only localized to ethmoid sinuses but involves all the sinuses. The extent of endoscopic surgery should be planned and adapted to the requirement of the individual case. For circumscribed pathology minimum invasive interventions are indicated. While for maxi and advanced diffuse polyposis pan-sinus operations are required. Depending on the extension, endoscopic surgery is planned and accomplished. For diffuse polyposis of all sinuses a complete endoscopic ethmoidectomy together with the fenestration of the frontal, sphenoidal and maxillary sinuses is done. The disease is cleared from the sinuses through the enlarged opening of the respective sinus. The end result should be an open ethmoidal cavity lined by moist mucosa and having free communication with the frontal, sphenoidal and maxillary sinuses.

Now the microdebriders or powered shavers are available which cause less bleeding and preserves mucosal lining during endoscopic sinus surgery and are excellent tools to excise maxi polyps and soft tissue lesions, Setliff 1994.

In advanced multiple nasal polyps initial clearing is generally done with the help of microdebrider. With the availability of the microdebrider this initial step can be accomplished more effectively and with minimal bleeding. For the management of the anterior ethmoid, the frontal and maxillary and sphenoid sinuses 25^0 or 70^0 telescopes are used .Various instruments are pre-requisit such as curved cup forceps, thru cut forceps, punches and curettes to work within the cavities. I always like to reconstruct big middle meatus opening with complete clearing the polyps and associated necrotic cells and diseased tissues. If D. N. S. obstructs the workability and negotiation of the instruments then septoplasty has to be done.

During entire surgery one should be very careful and meticulous to prevent mucosal injury since mucosal injury and stripping causes :

a. Slow and delayed healing;
b. Provides nidus for chronic infection;
c. Induces formation of mucosal bands and scar formation leading to synechia and stenosis;
d. The denuded bone also causes persistant infection, granulations and stenosis;

Surgical results improve by pre-operative planning, use of antibiotics and steroids - pre and post operatively, and use of thru-cut forceps and microdebrider that helps to preserve mucosa. Reconstructing big antrostomy always helps for better results.

In diffuse polyposis a short course of systemic steroids pre operatively and post operatively is quite useful which is always followed by topical steroid sprays.

Out of 80 cases of nasal polyposis operated by endoscopic sinus surgery, 12 cases had extensive disease with orbital involvement (Fig. 1a, b) and 3 cases had intracranial extension. Incidence of orbital involvement in small series of 80 cases is apparently high which is due to outstation referral of complicated cases. Even in extensive disease, I prefer to do endoscopic sinus surgery under L.A. because the pupillary reflexes can be monitored and patient's subjective feed back of excessive pain sensation when the instruments are touching the orbital plate, cribriform plate or fovea ethmoidalis can be warning signals to the surgeon, which increase the safety index in endoscopic sinus surgery.The follow up of patients in our country is very poor. I follow up my patients endoscopically for suction clearance of crusts and discharge. If I find any adhesion in middle meatus that is incised or if any small polyps are visible that is also removed without any pain or discomfort to the patient. Out of 80 cases, only 26 cases came for regular follow up and six cases were revised once, two cases were revised twice and one case had revision three times (Fig. 2a, b, c, d, e, f; 3a, b, c, d, e, f).

The new technology in imaging and availability of powered instruments helps to deal with the nasal polyposis and inflammatory disease more effectively and safely. Image guided surgery minimizes the complication rate, incidently I do not have the facility of using this.

In this presentation few cases of maxi polyps with orbital and intracranial extension will be presented showing pre and post operative CT scans.

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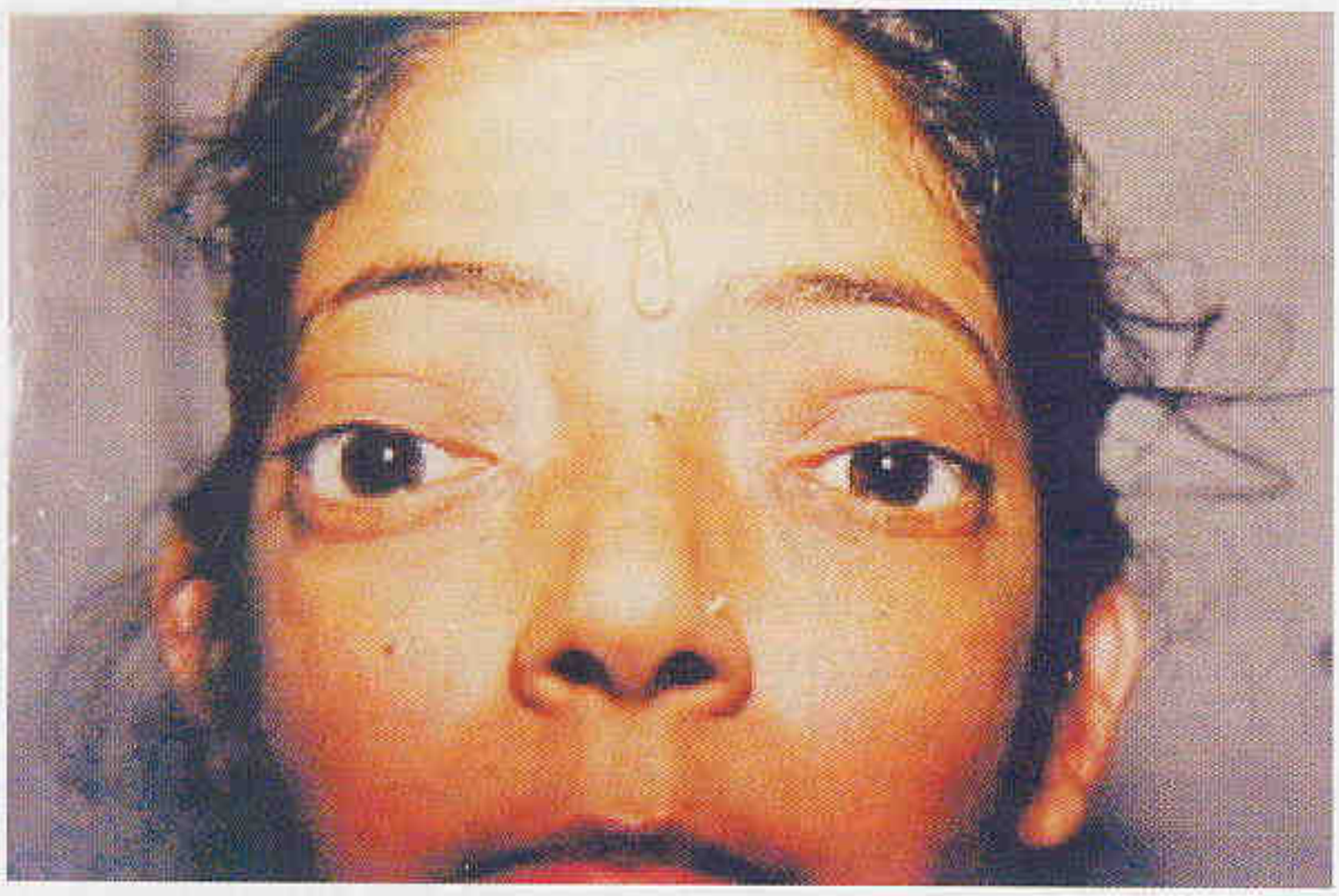

Fig. 1a. A case of maxi-nasal polyposis with orbital involvement causing bilat-eral marked proptosis.

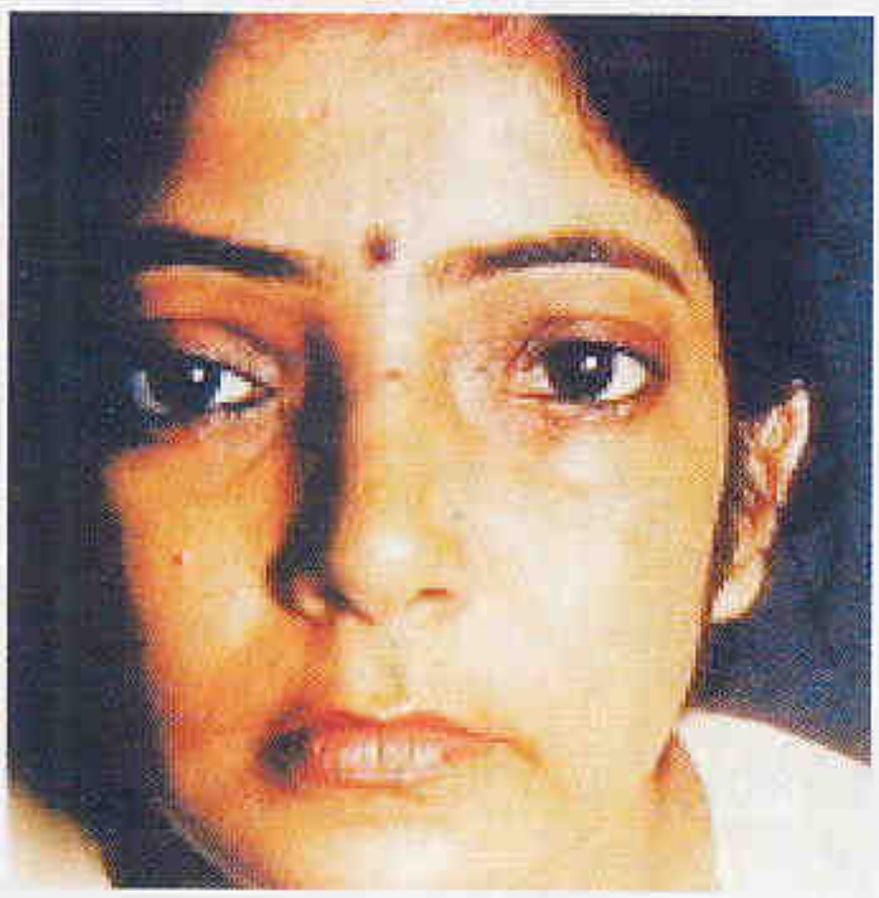

Fig. 1b. Six weeks after surgery proptosis on left side has completely and on right side partially regressed.

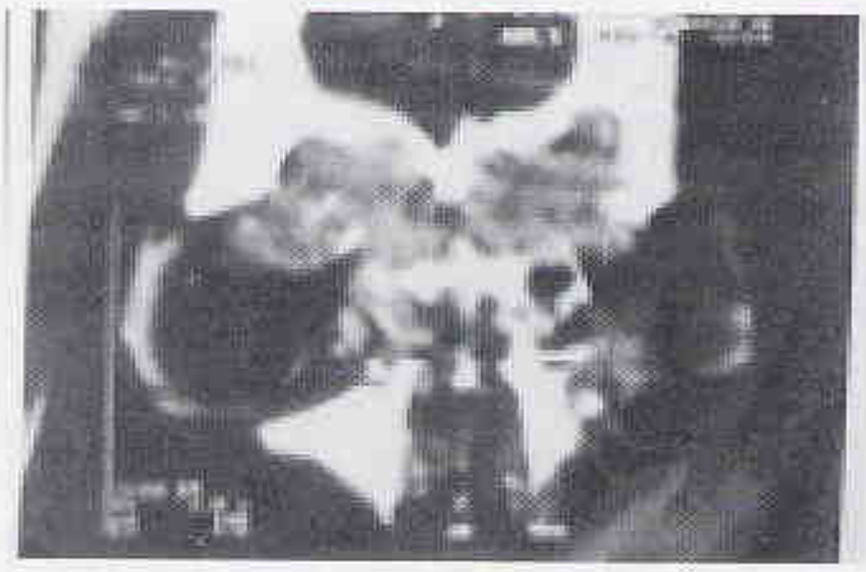

Fig. 2a. A case of bilateral nasal polyposis involving both frontal, maxillary, ethmoid and right side sphenoid simus. The preoperative coronal view.

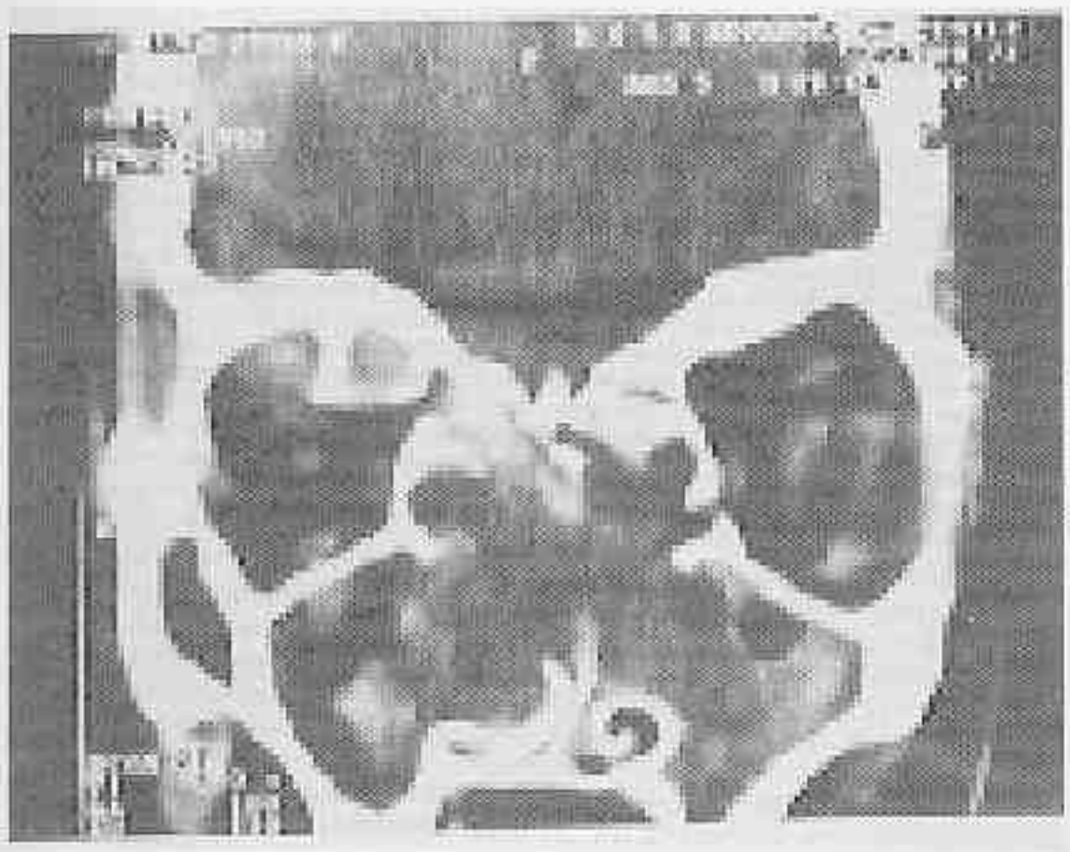

Fig. 2b. More posterior coronal view in the same patient.

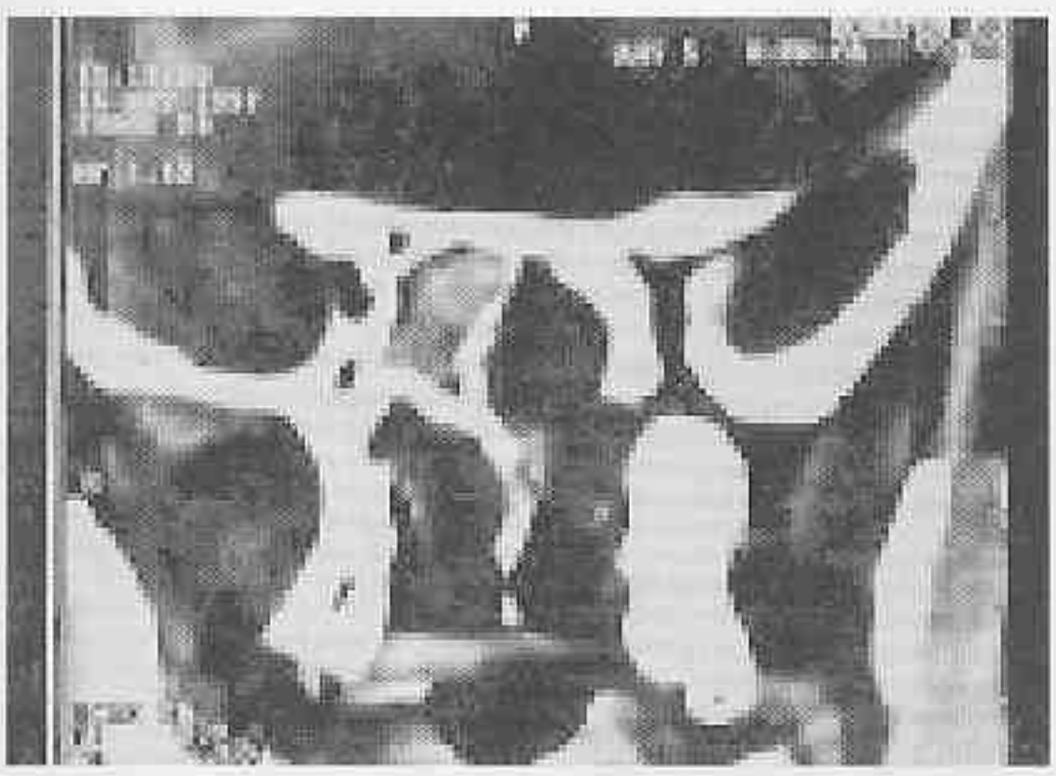

Fig. 2c. The same patient. The coronal scan at the deepest regions (sphenoidal simus).

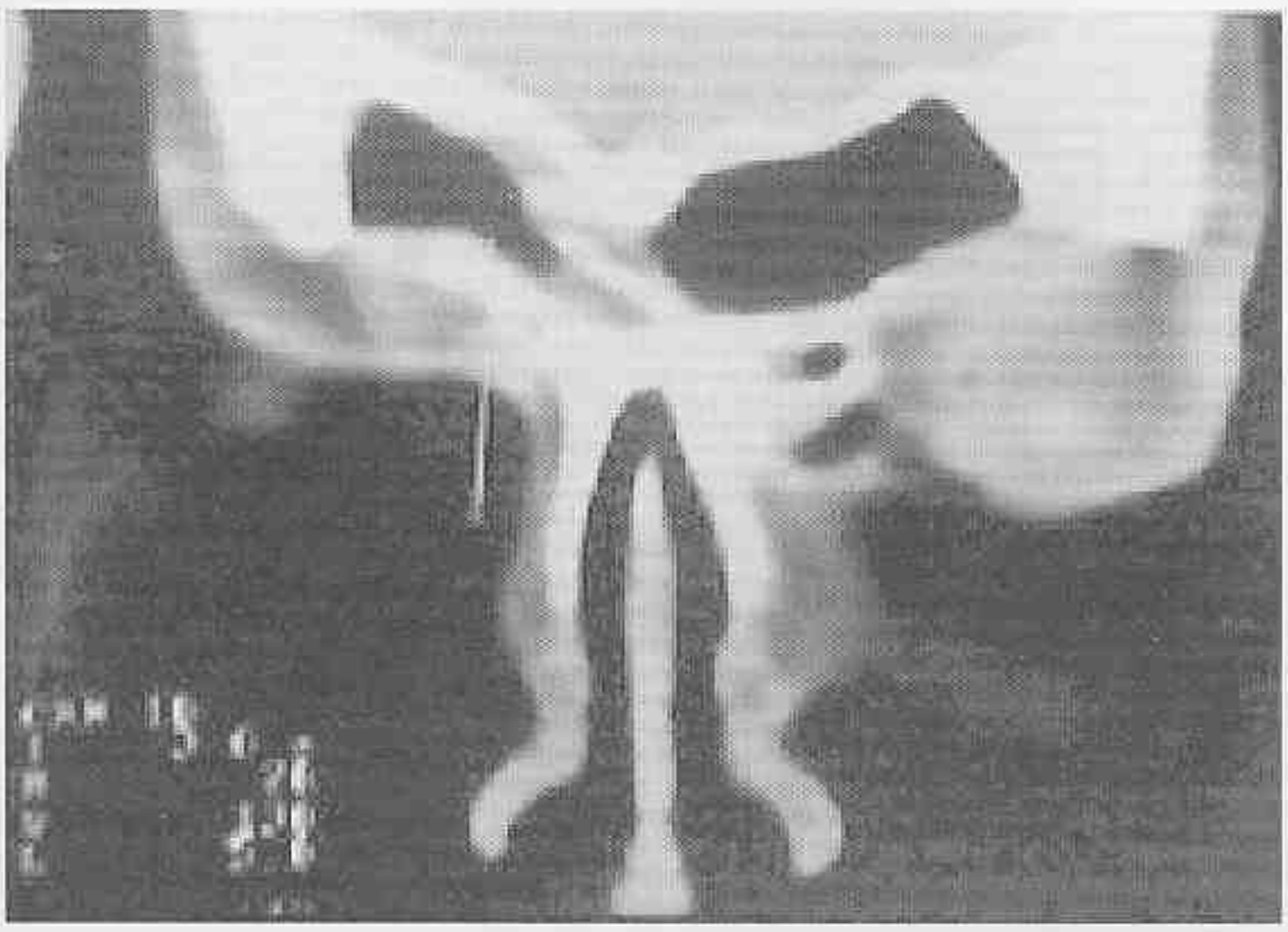

Fig. 2d. One year postop. The same patient

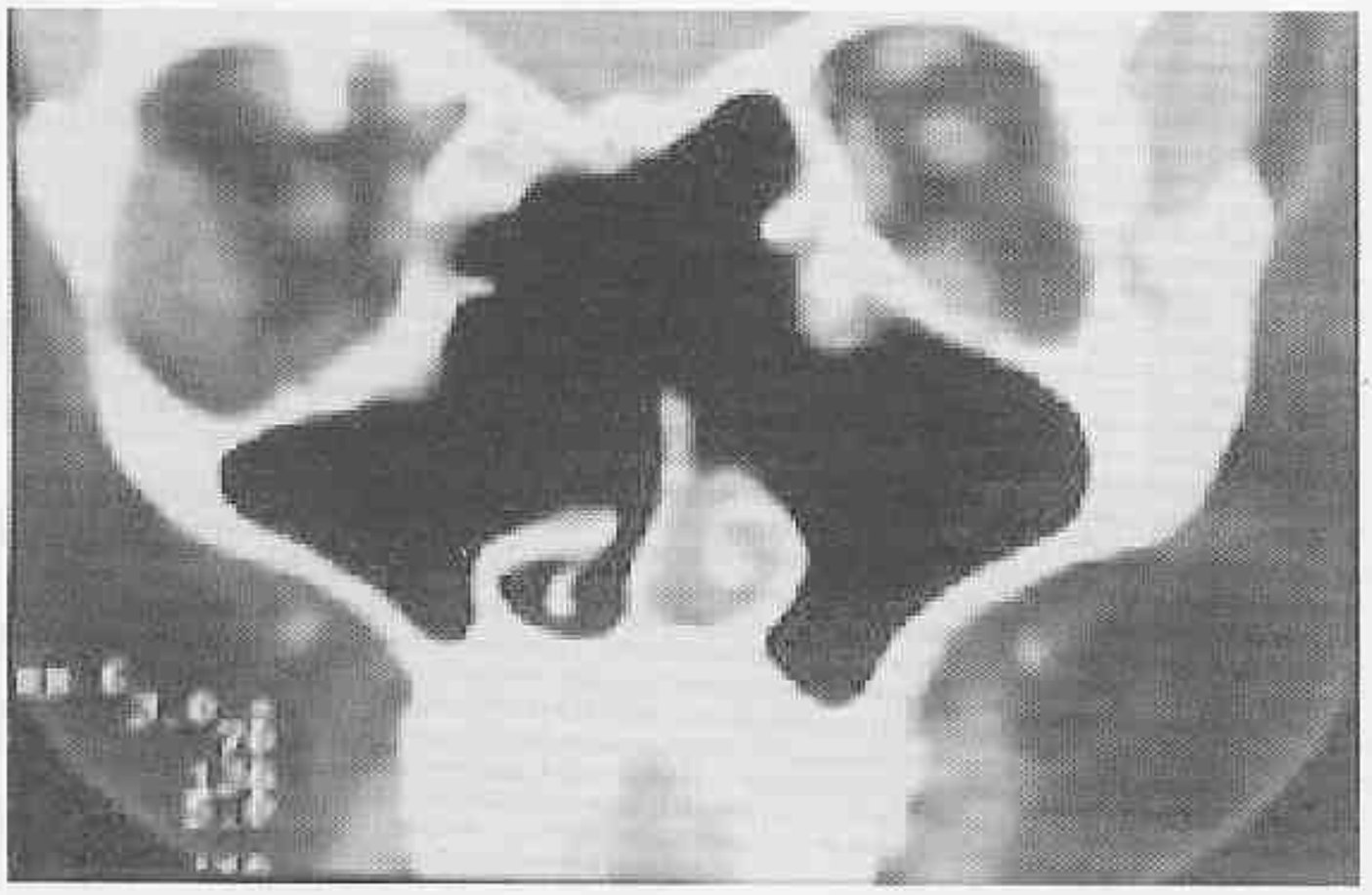

Fig. 2e. One year postop. The same patient.

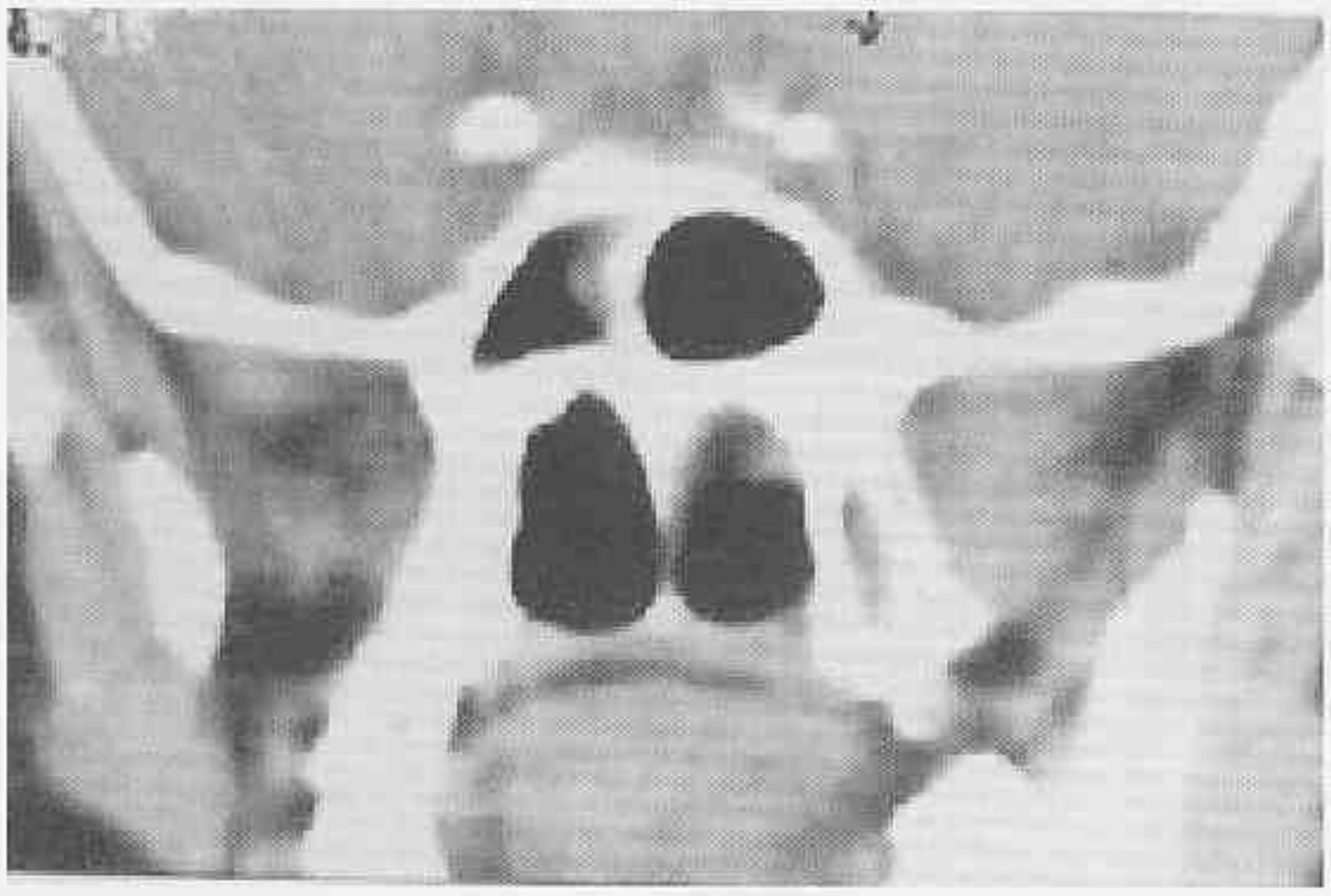

Fig. 2f. The same patient one year postop.

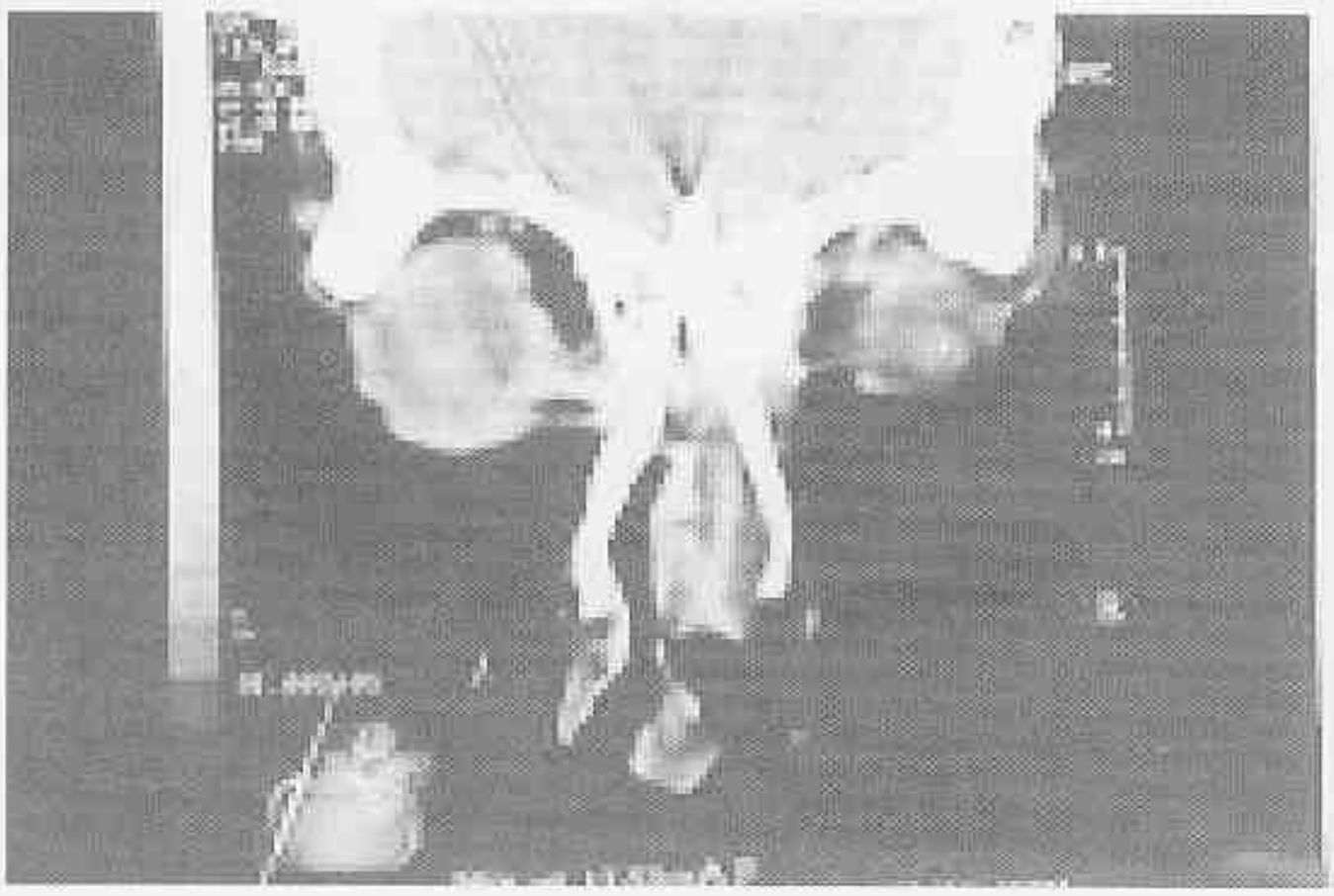

Fig. 3a. A case of extensive bilateral nasal polyposis involving right maxillary sinus, both frontal recesses, both ethmoid simuses and sphenoidal simus.

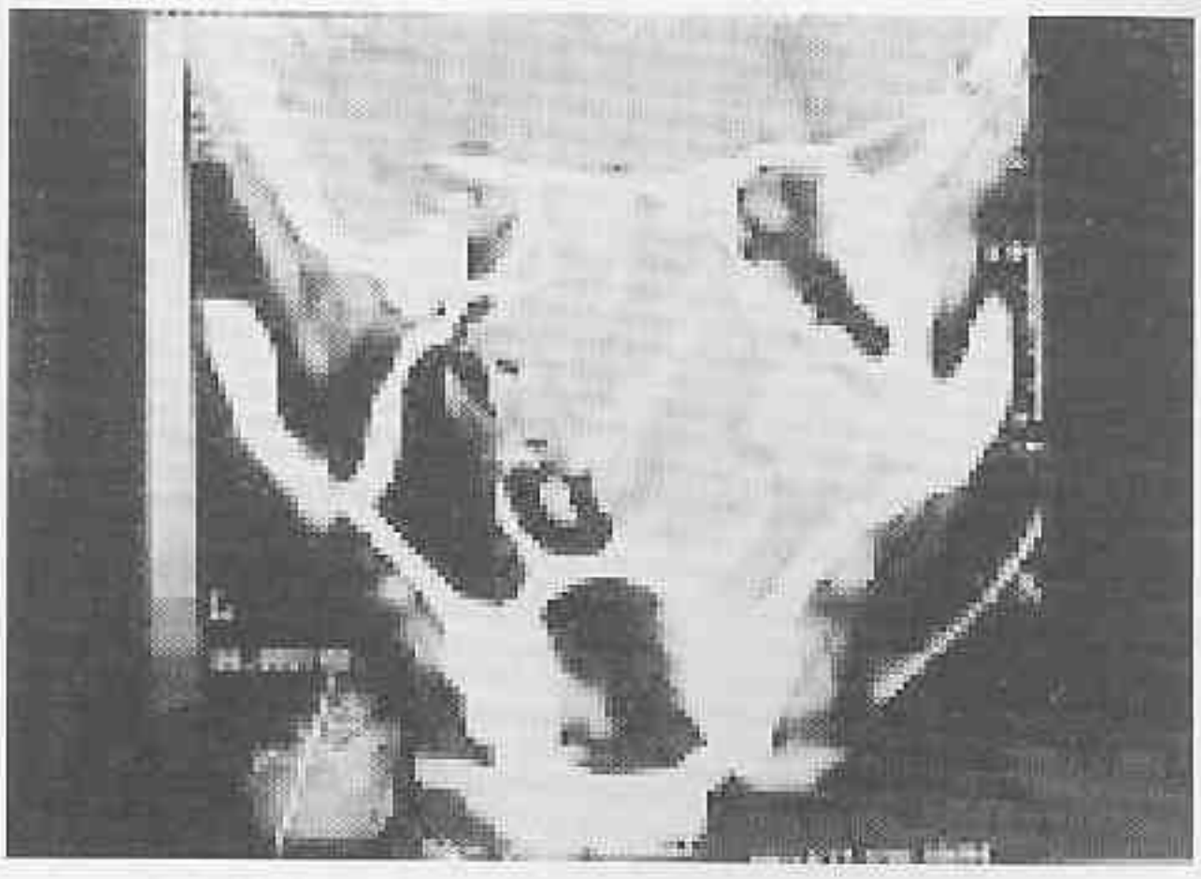

Fig. 3b. The region of the posterior ethmoid. The same patient.

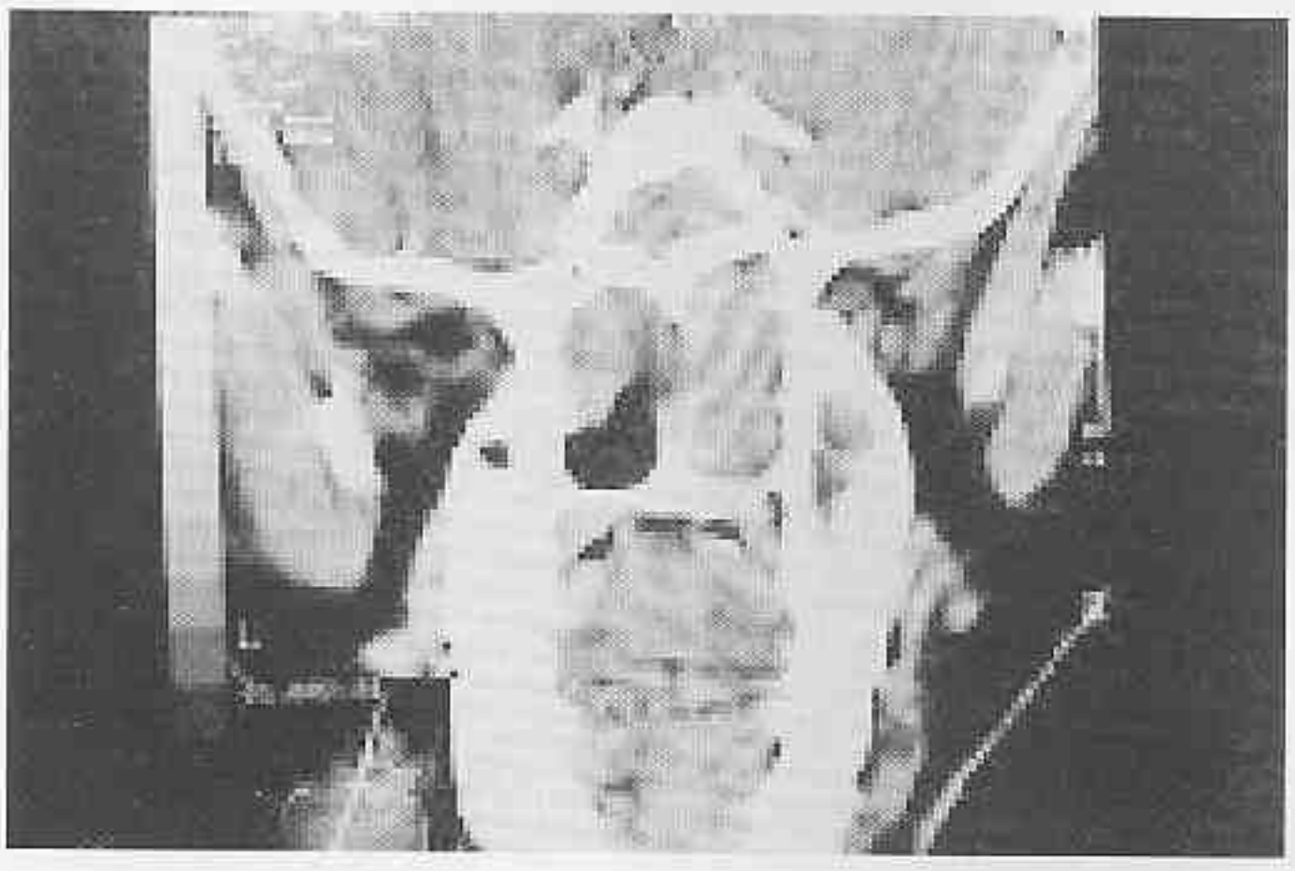

Fig 3c. The sphenoidal region. The same patient.

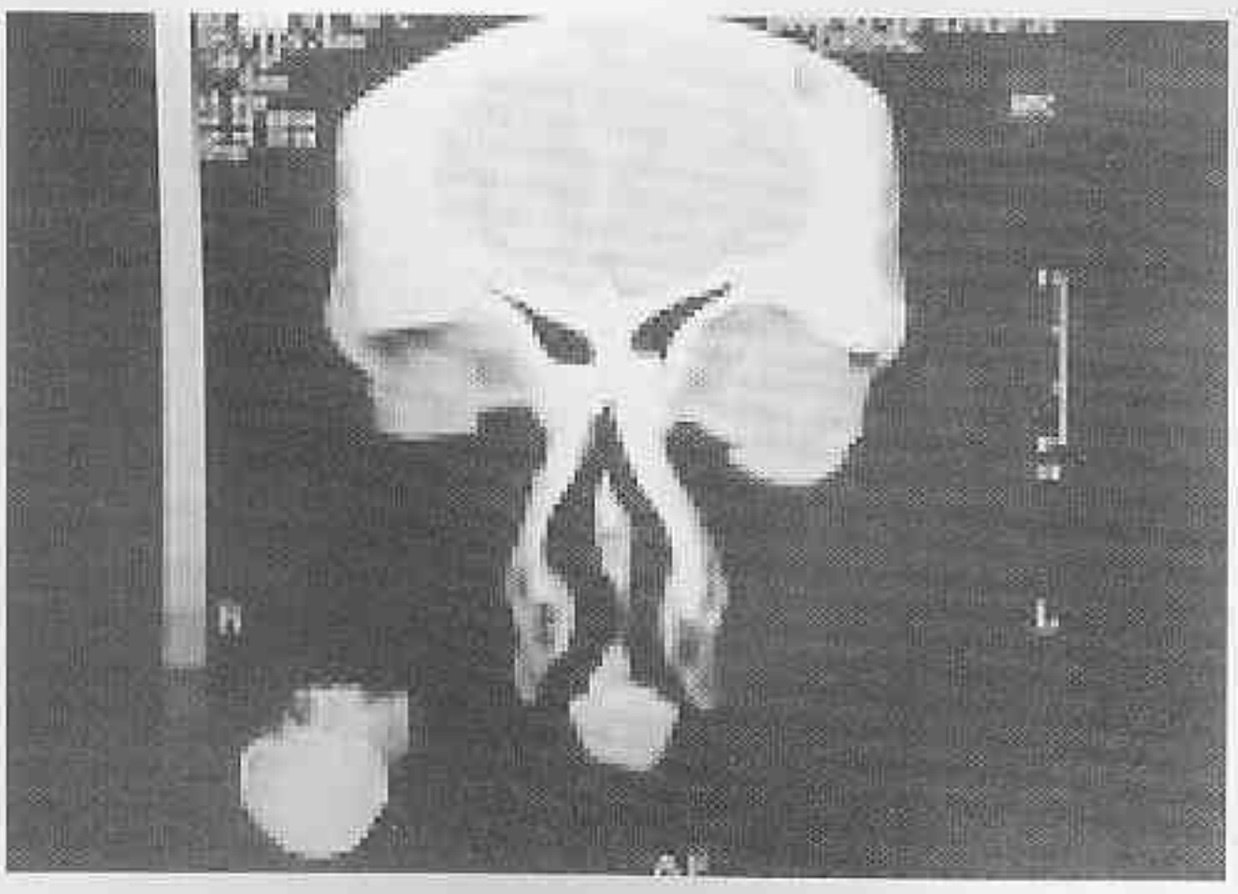

Fig. 3d. Six months after FESS. Free from disease.

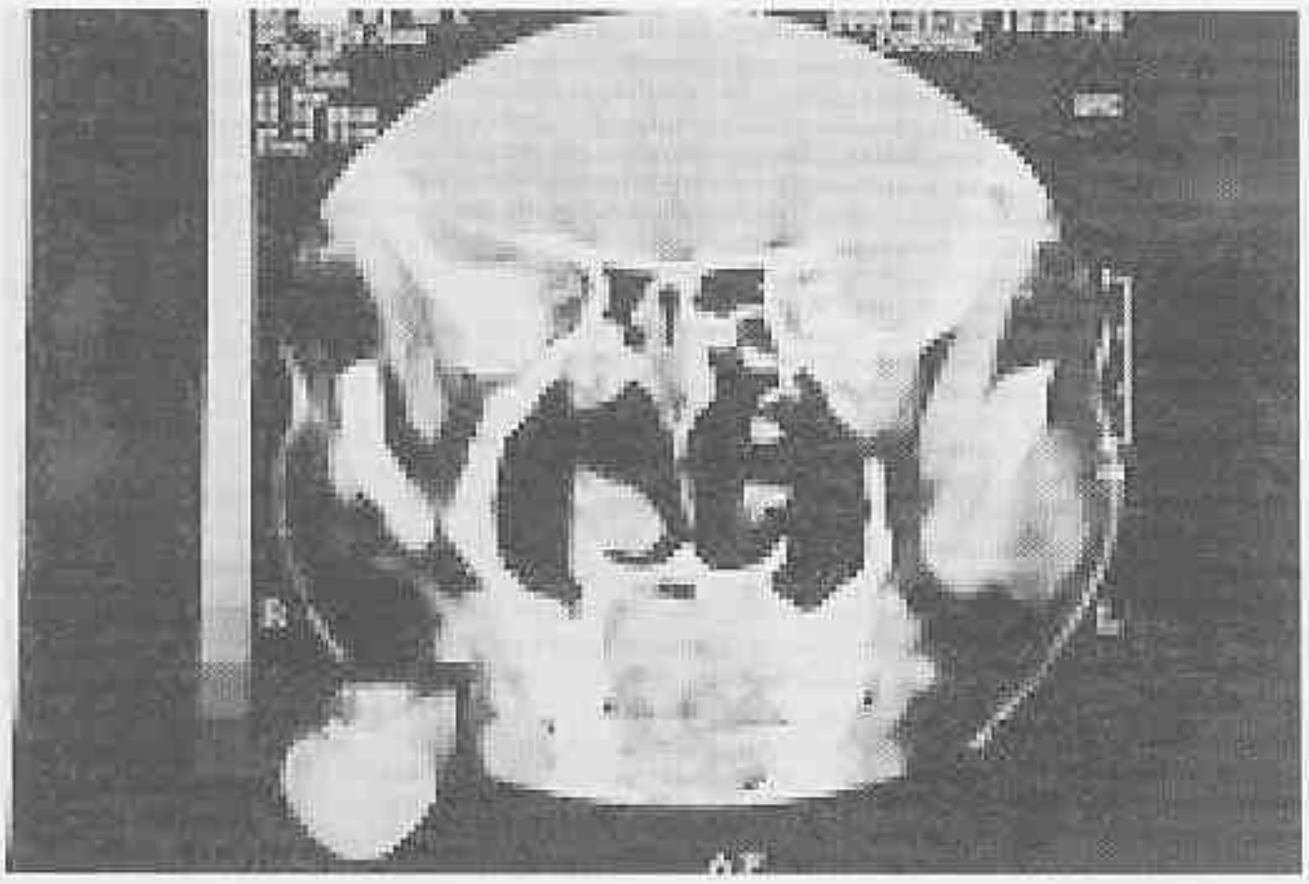

Fig. 3e. Ethmoidal region six months after the surgery.

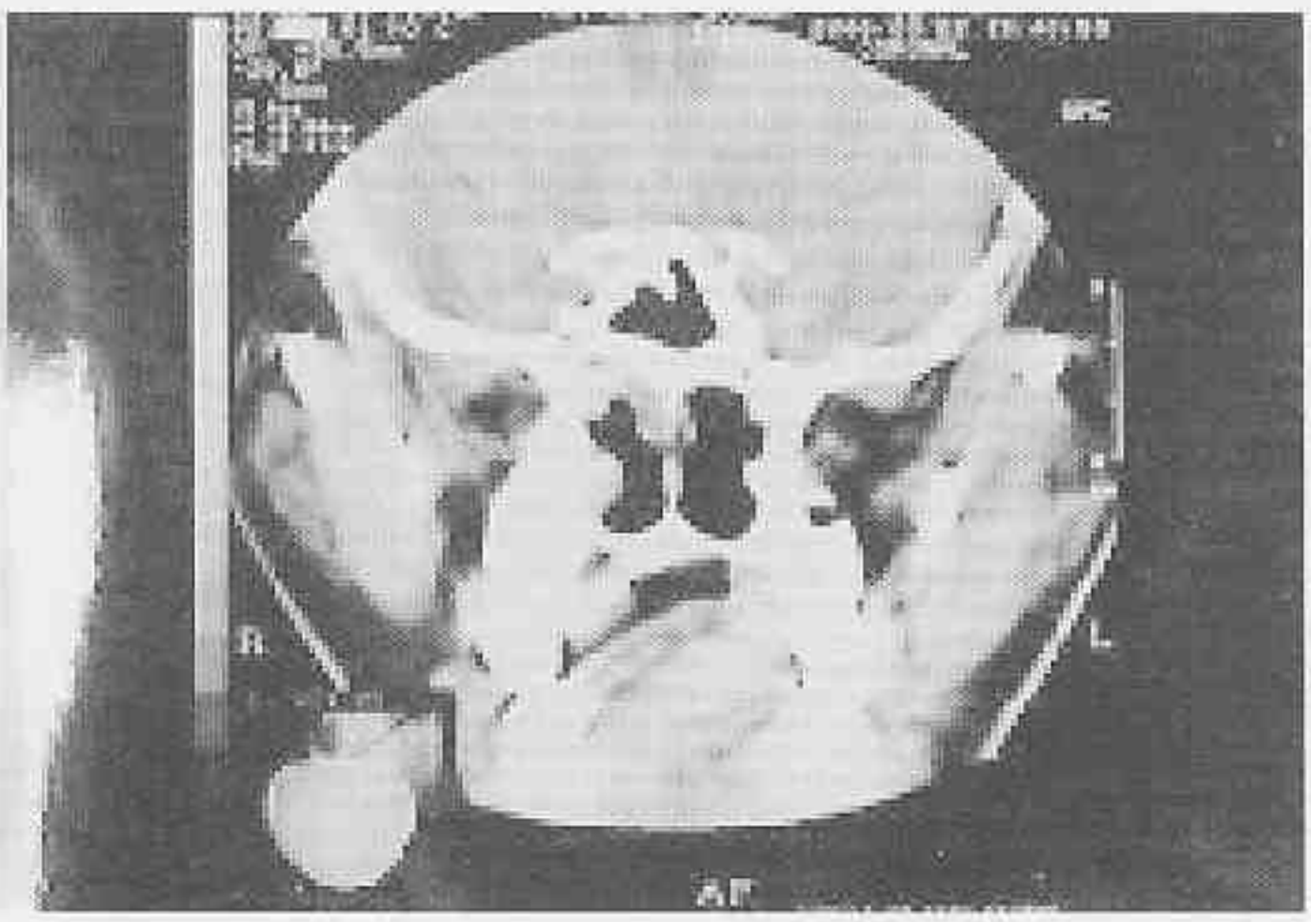

Fig. 3f. Sphenoidal region six months after the surgery.

NASAL POLYPOSIS IN CHILDREN

V. P. Sood

New Delhi, India

The management of diseases involving the nasal cavity and paranasal sinuses in children depends on the experience gained in adults.

The anatomy and the size of the child's nasal cavity, behavior of the nasal mucous membrane, its reaction to infections and allergic stimuli show significant differences between children and adults. The paranasal sinuses, have also different structures in children.

A unique feature of the nasal airways in infants and the children is the dependence of the newborn on nasal breathing. Clinical manifestation of chronic nasal obstruction in children is compensated by persistent mouth breathing sometimes resulting in maxillo-facial deformities and cardiopulmonary problems. The nasal obstruction can be due choanal atresia, nasal infection, allergic manifestation, polyps, deviated nasal septum, enlarged turbinates, congenital malformation, trauma, enlarged adenoids and nasal tumours. Nasal polyposis is rare in children and is often associated with cystic fibrosis, primary ciliary dyskinesia, aspirin-intolerance and chronic obstructive pulmonary disease. The problems associated with nasal airway obstruction in the older child are less dramatic than those in the infants.

Assessment of nasal and nasopharyngeal anatomy and aetiologies of nasal obstruction can now be precisely made by nasal endoscopic examination and CT scan.

Recently introduced endoscopic sinus surgery has radically changed the etiopathological and therapeutic concepts of recurring chronic sinusitis and nasal polyps, Stammberger 1986, Wigand 1982, Sood 1987. The technique of Messerklinger advocated by Stammberger is well suited in paediatric patient to safeguard the untoward effect of growth and development of sinuses and tooth buds in maxilla Wald 1985, Cald Wel Luc operation in children is not suitable because of possible disturbance in facial growth and damage to the tooth buds.

The application of functional endoscopic sinus surgery in children with chronic sinusitis has been reported by Lusk and Muntz, 1990; Lazar et al 1992., Triglia et al., 1992. In nasal polyposis in children functional endoscopic sinus surgery can be done.

There are some difficulties in any intranasal and sinus surgery in children because of small sized nasal cavities and less working space available as compared to adults. For children always use small instruments. The adult size instruments will pose difficulties for their use on children. The use of small child size Thudicum nasal speculum, small foreceps, small elevators and good head light are mandatory. For endoscopic surgery 2.7 mm diameter 0 or 30 degree telescope may be more suitable in small children if 4 mm diameter telescope causes difficulty in its use.

Endoscopic nasal and sinus surgery has entirely changed the aetiopathological and therapeutic concepts even in small children.

Endoscopic sinus surgery should be planned in children if medical management is not successful. I have limited experience of doing endoscopic sinus surgery in children. Out of series of 80 cases of nasal polyposis, I operated on six children and only two cases came for follow up and one case was revised.

Endoscopic sinus surgery in children should be only done by those who have sufficient experience in adults. I will highlight my experience for the management of nasal polyposis in children in the Figures 1 and 2.

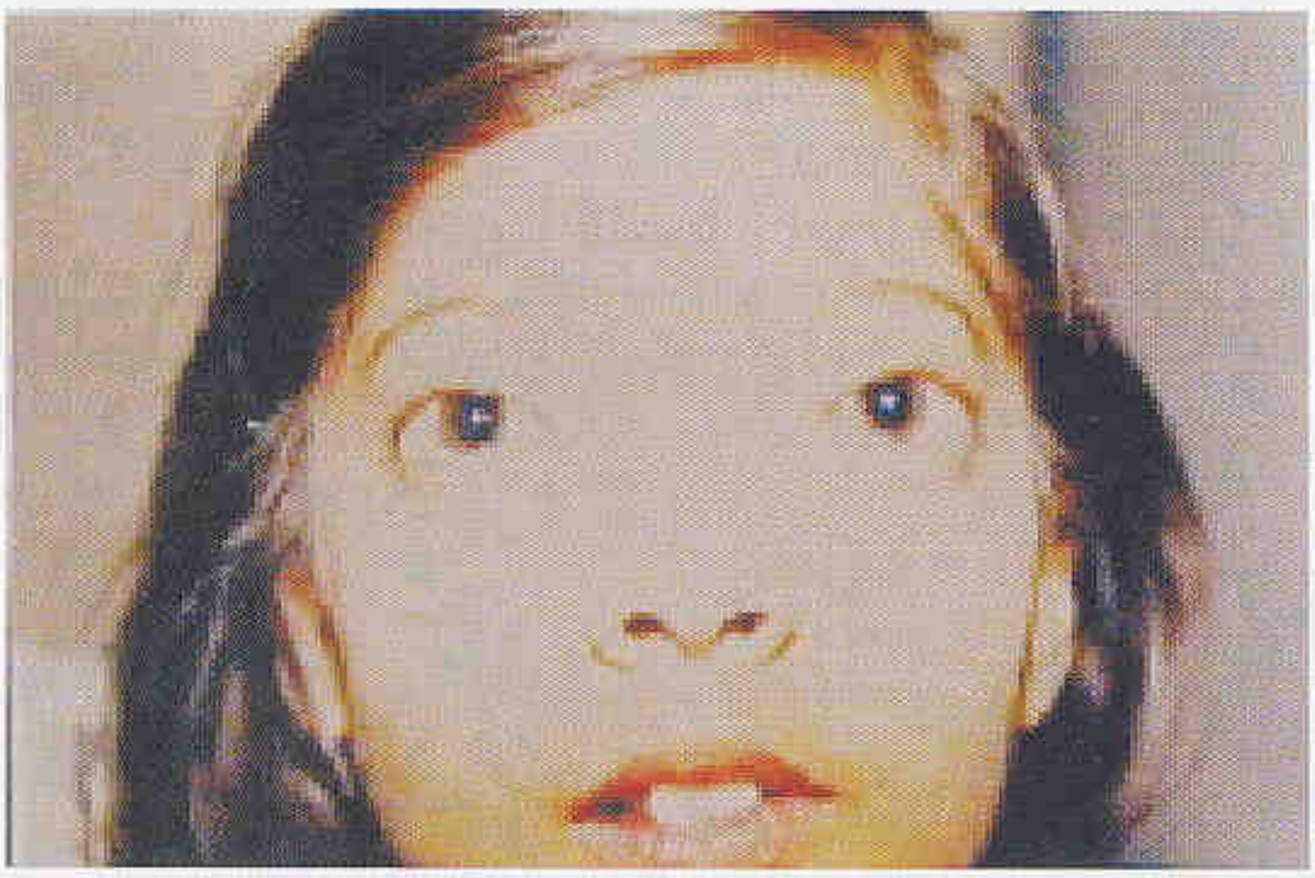

Fig. 1a. 11-year-old girl showing bilateral proptosis due to extensive nasal polyposis

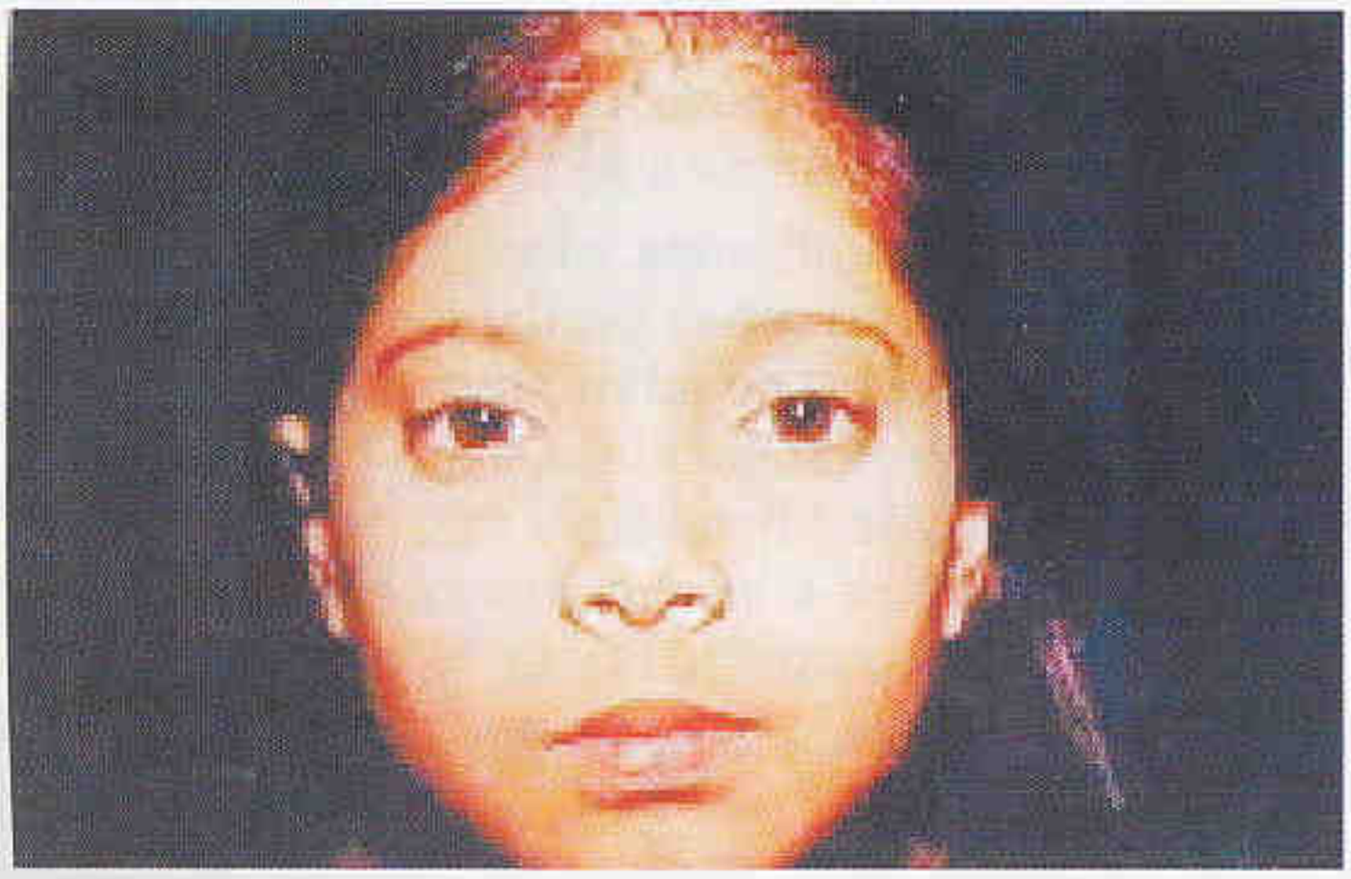

Fig. 1b. Proptosis has completely regressed four weeks after FESS.

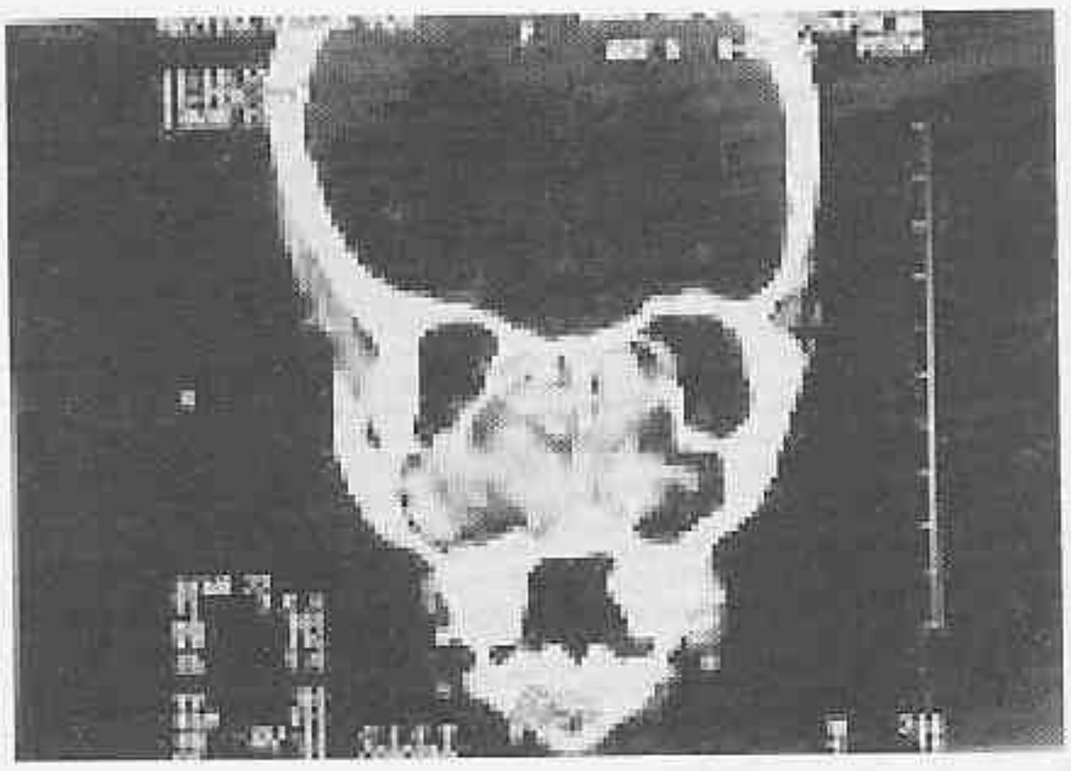

Fig. 2a. Preoperative coronal scan. The same patient.

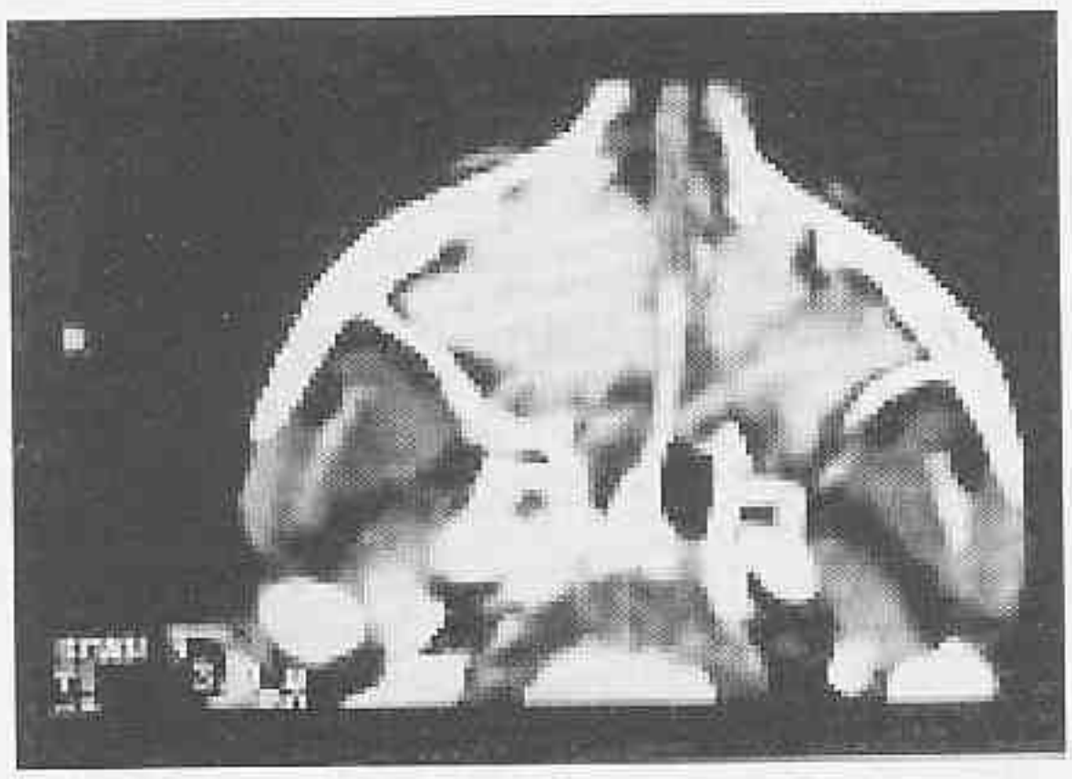

Fig.2b. Preoperative axial scans. The same patient.

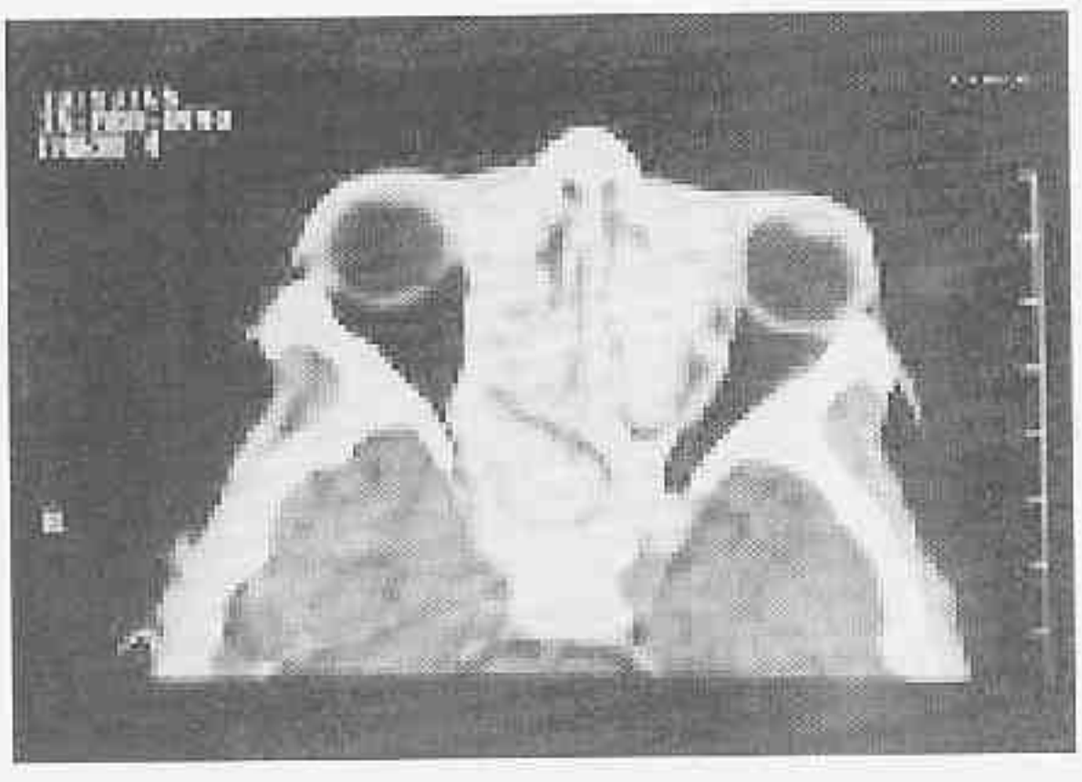

Fig. 2c. Superior ethmoidal region. Preop finding of the same patient.

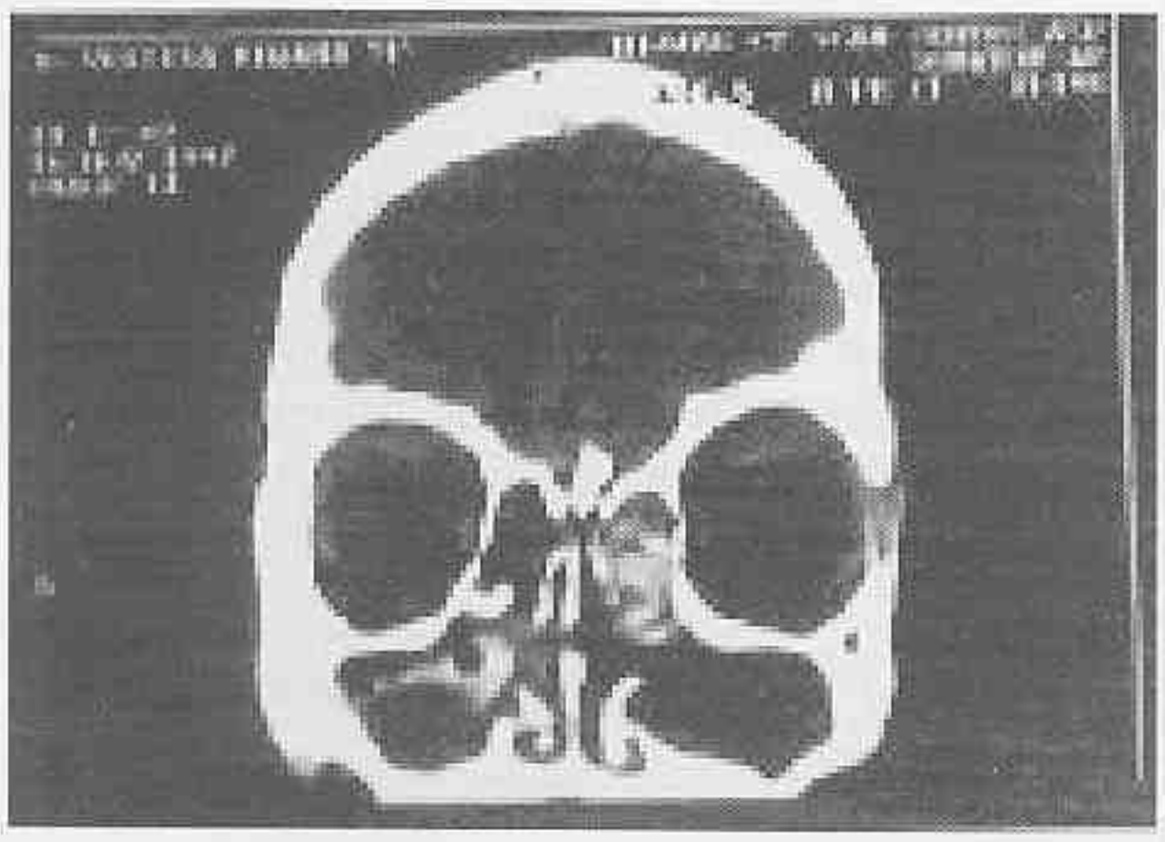

Fig. 2d. Coronal CT-finding of the same patient six month potop. Some recurrence of disease in the right ethmoidal sinus was cleared by revision surgery.

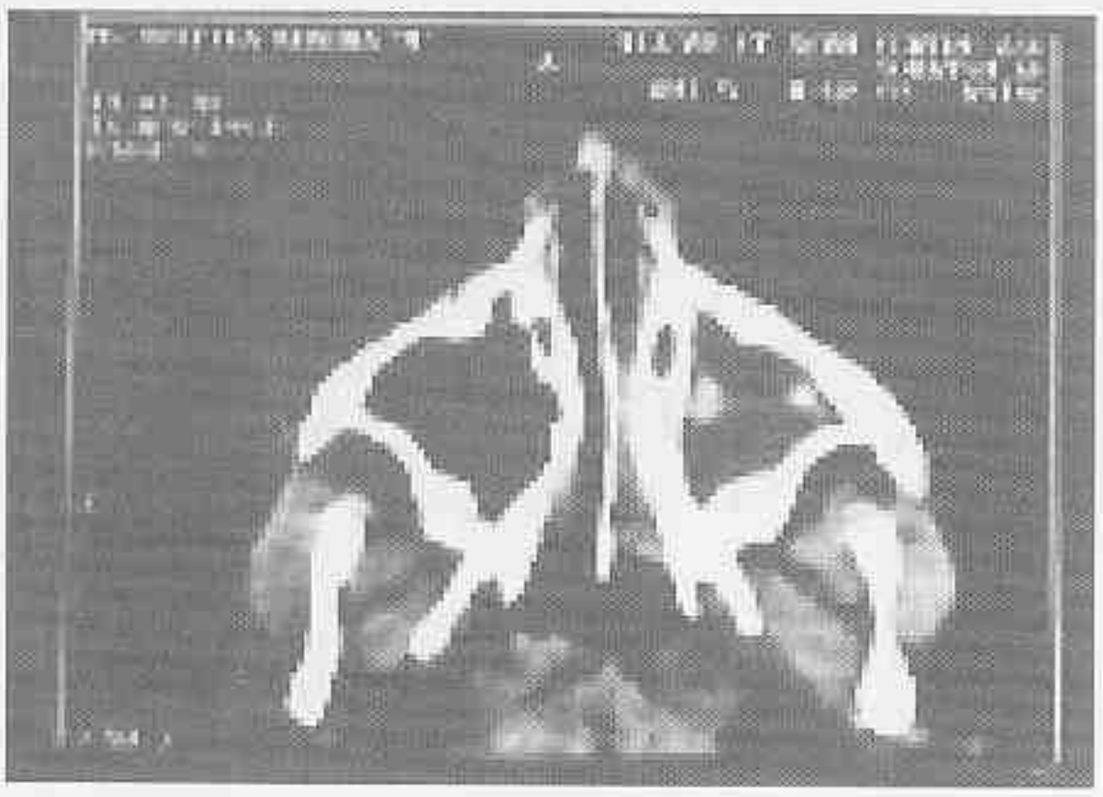

Fig. 2e. Axial scan 6-months after the surgery

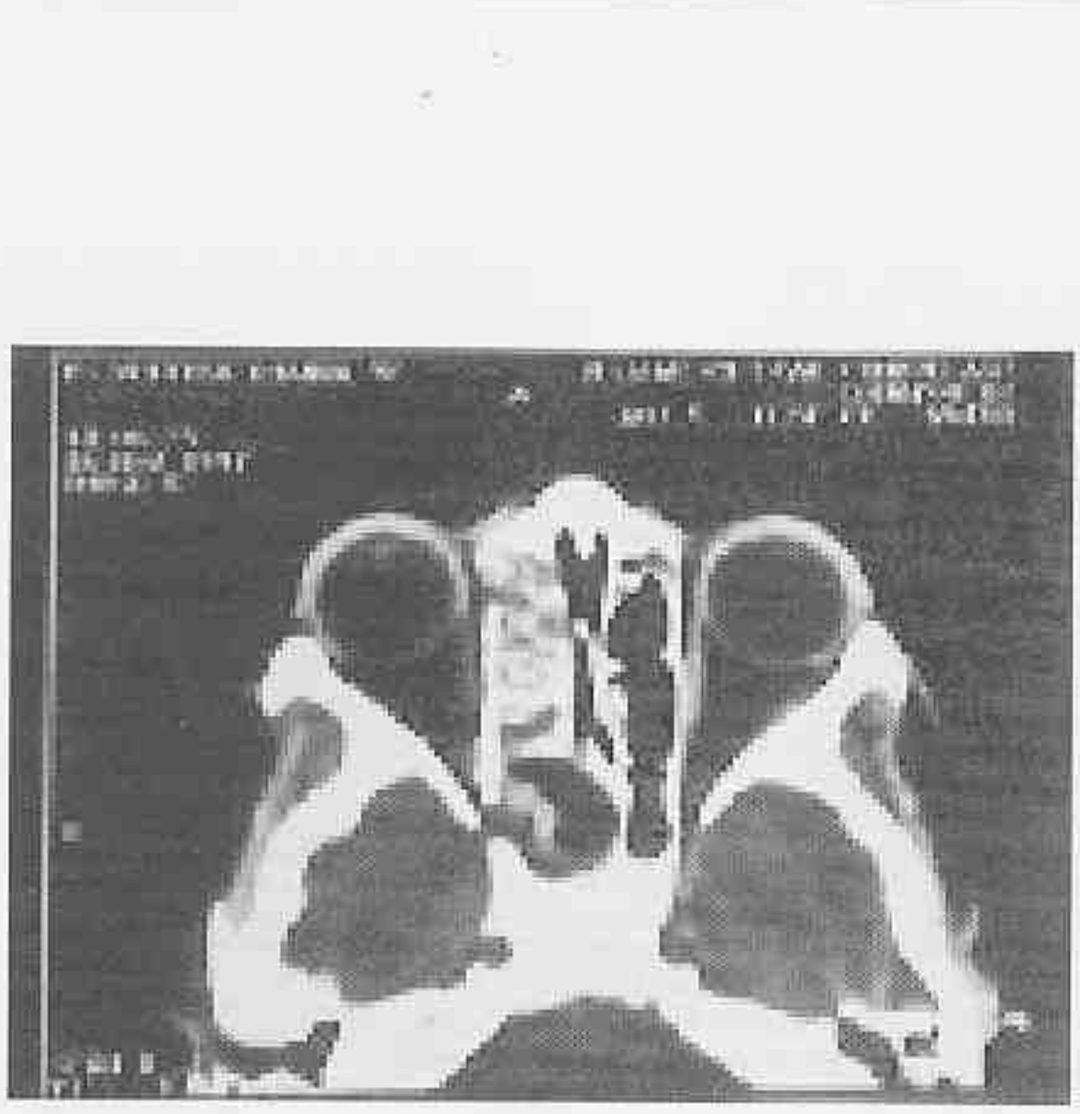

Fig. 2f. Axial scan six months postop showing some recurrence.

With the availability of CT scan of sinuses supplemented with nasal endoscopy, we can now diagnose early nasal and sinus pathology. Depending on diagnostic features, the diseased ethmoidal cells can be removed and sites of origin of frontal and maxillary sinus disease can be cleared to help for proper ventilation and drainage of sinuses, which in turn controls the sinus disease.

To accomplish endoscopic techniques in children one requires perfect knowledge of ethmoid and middle meatus anatomy and thorough training and orientation in the use of telescopes for diagnostic and therapeutic purposes.

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CHRONIC RHINOSINUSITIS– NEW CONCEPTS REGARDING CAUSE AND THE ROLE OF FUNGI

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The original thinking suggested that chronic rhinosinusitis was a bacterial infection, secondary to a viral etiology or an allergy. It is histologically, an eosinophilic inflammation proven by H&E studies. This eosinophilic inflammation is complicated by various periods of acute exacerbation. It is these acute exacerbations that seem to be of bacterial origin. Bacterial infections, however, trigger a neutrophilic inflammation.

The eosinophilic inflammation seen in chronic sinusitis is not likely to be caused by bacteria. Eosinophils are understood to play a role in the host defense against larger, non-phagocytosable organisms such as parasites.

The original aim of our research on chronic sinusitis was to prospectively determine the incidence of Allergic Fungal Sinusitis (AFS). Through novel culture, histologic and antigen detecting methods we were able to demonstrate the presence of fungi in every patient with chronic rhinosinusitis (n=46), and in every healthy control person without the disease (n=14).

By studying the tissue and mucin from chronic sinusitis sufferers more closely we observed that the eosinophils were present nearly entirely intact in the tissue. Further the eosinophils migrated into the mucin, formed clusters around fungi and degranulated. Since this was observed in the majority (96%) of consecutive surgical CRS cases (n=101), the question was raised whether the eosinophils play an immunologic defensive role against those fungi in CRS patients.

Immunologic testing further showed that the chronic sinusitis patients peripheral blood T-lymphocytes, when presented with certain fungal antigens, reacted with the production of the cytokines which recruit (IL-13) and activate (IL-5) eosinophils (n=18). Lymphocytes from healthy controls (n-15) did not demonstrate this immune response. We concluded that the T-lymphocytes in chronic sinusitis patients recruit eosinophils in response to fungal antigens, while T-lymphocytes in normal people do not. This underlying reaction to fungi occurred independent of IgE mediated allergy. Thus, the immunologic response is not IgE mediated allergy, and the term "allergic" in AFS is incorrect. As a consequence, the term Eosinophilic Fungal Rhinosinusitis (EFRS) was introduced.

Our working hypothesis of the immunologic mechanism of EFRS, based on the research findings in the laboratory, is that eosinophils are recruited as a defense to fight the fungi in the nose, where healthy controls are lacking this specific immunity. The eosinophils migrate through the nasal tissue and into the mucin of the nose. There the cells cluster around the fungi in a similar fashion as they group around parasites. The eosinophils destroy the fungal organisms through the release of their toxic proteins. As a result, the mucin contains eosinophilic Major Basic Protein (MBP)

in a quantity large enough to damage the nasal mucosa. This mucosal destruction allows residential nasal bacteria to secondarily invade the patient's mucosa and cause an acute exacerbation of chronic sinusitis.

Currently we are developing new treatment protocols based on our understanding of the etiology of CRS. Intranasal antifungals have been demonstrated to be safe and appear to demonstrate efficacy in open trials and are now tested in a double blinded, placebo-controlled fashion. It should be mentioned that this non-invasive disease is a hypersensitivity to fungi, and not a fungal infection. EFRS needs to be differentiated from other forms of fungal sinusitis, such as fungus balls (non-invasive) and invasive fungal sinusitis (acute fulminant or chronic form).

A most striking finding for us is the fact that the T-lymphocytes of chronic sinusitis patients are sensitized in the peripheral blood and recruit and activate eosinophils when they sensorineural a fungal antigen. This finding indicates that CRS is a systemic hypersensitive disease. Further research into the pathophysiology of CRS along this new paradigm will hopefully lead us to new treatments and ultimately better care for our patients

CLINICAL ASPECTS OF NASAL POLYPOSIS- STAGING

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The diagnosis and surgical management of chronic rhinosinusitis has been greatly improved by the combination of rigid endoscopy and computerised tomography but in rhinology it has proved remarkably difficult to quantify and qualify our patients' disease and their response to treatment. There is no universally accepted nor completely reliable objective tests of pathophysiology and its improvement. Consequently there has been a number of approaches to assessing extent of disease. These have largely relied on CT scanning as most patients undergo this procedure prior to endoscopic sinus surgery. Systems may be broadly divided into those that use the distribution of opacification within the sinuses to define four stages, (Friedman et al 1990, Gliklich and Metson 1994, Kennedy 1992, Levine et al 1993) (Table 1) those using a numerical score for each sinus group (Jorgensen 1991, Lund et al 1993) and more complex ones including other parameters such as immunological aspects and thickness of mucosal change)Gaskins et al 1994; Newmans LJ et al 1994). If any of these systems are to be used widely it is of paramount importance that they are easy, quick, simple to understand and subject to good inter and intra observer agreement. However, irrespective of the system it is worth remembering that symptom severity and appearances on nasal endoscopy do not correlate well with extent of disease on sinus CT.

Lund-Mackay Scoring System

The Lund-Mackay Scoring System (Tables 2-7) is based on a simple numerical score derived from CT scans (Lund and Mackay 1994) After some modifications it was included in the supplement resulting from the International Conference on Sinus Disease (Lund and Kennedy 1995) and more recently was recommended by the Task Force of the American Academy of Otorhinolaryngology Head and Neck Surgery to form the basis for further outcome research.

It has been independently assessed and shown to have a high intra- and inter individual reproducibility (Oluwole et al 1996). Demographic information may be collected included nasal diagnosis which is classified into:

1. Chronic rhinosinusitis
2. Acute recurrent rhinosinusitis
3. Nasal polyposis
4. Miscelleneous which includes conditions such as mucoceles, CSF leak, orbital decompression etc.

A systemic diagnosis may also be included *e.g.* asthma, cystic fibrosis, primary ciliary dyskinesia.

The scoring system was devised on the appearances on scans which have generally been performed after an adequate trial of medical treatment although what this actually constitutes is open to some debate. Each sinus group is graded between 0 and 2 (where 0 = no abnormality, 1 = partial opacification, 2 = total opacification) for the maxillary frontal, sphenoid, anterior ethmoid and posterior ethmoid sinuses. As it is difficult to apply this to the ostiomeatal complex this is simply scored as 0 (not obstructed) or 2 (obstructed). Thus a total score of 0-24 is possible with each side scoring a total of up to 12.

The importance of anatomic variants is open to debate but their presence (1) or absence (0) may be noted and similarly a surgical score may be derived (0 not performed, 1 performed) for each part of the procedure offering a score of 0-14 (0-7) for each side.

Symptoms both pre- and post operatively may be assessed on a visual analogue score of (VAS) 0-10 (where 0 means no symptom is present and 10 is the most severe rating) for nasal obstruction or congestion, headache, facial pain, sense of smell, nasal discharge and sneezing. This can be combined in an overall symptomatic assessment or treated as individual symptoms. In addition patients are also asked to prioritise their three worst symptoms which can distinguish the relative importance of these even when they have been given the same VAS.

The endoscopic appearance of the nose can also be quantified, scoring for the presence of polyps, discharge, oedema, scarring and adhesions and crusting although clearly greater subjectively applies to these scores.

All attempts at grading disease, of which this is one, are open to criticism and the clinical relevance of the extent of disease on a scan is open to debate. The scan merely represents a snapshot in time and should be performed after *adequate* medical therapy and *reasonably* close to the time of the surgical procedure though it would be difficult to achieve complete agreement amongst rhinologists as to how these terms are defined. Most authors have been careful to avoid such definition though I would regard 2-3 months as a reasonable rule of thumb in each case. No staging or scoring system is perfect and in patients who are missing a sinus or who have undergone previous surgery some qualification is required. If a frontal sinus is missing as it may be in approximately in 1% of a Caucasian population then it should simply be scored as 0. Similarly previous surgery such as a Caldwell-Luc may cause thickening of the mucosa which is fibrotic rather than inflammatory but in the absence of pathological confirmation the sinus should be scored as previously described. It is known that the scan appearances correlate poorly with patients' symptoms and endoscopic appearances but it does offer an objective quantification of disease which allows comparison between surgeons and their results both medical and surgical and is a useful criterion on which patient entry into clinical trials may be based.

The issue of radiation dose limits the use of this investigation as a post operative measure of success and most studies rely on symptomatic improvement, sometimes assessed quantitatively by visual analogue score or the use of mucociliary function and olfaction. (Lund and Mackay 1994, Lund et al 1991, Rowe-Jones and Mackay 1997, Abdel Hak et al 1998, Delank et al 1998).

Table 1 **1.**

The staging system of Kennedy

Stage 0	Normal
Stage I<?2>	Anatomic abnormalities All unilateral sinus disease Bilateral disease limited to ethmoid sinuses
Stage II	Bilateral ethmoid disease with involvement of one dependent sinus
Stage III	Bilateral ethmoid disease with involvement of two or mor e dependent sinuses on each side
Stage IV	Diffuse sinonasal polyposis

Table 2.

The radiological grading of sinus systems in Lund-Mackay scoring system

Sinus system	**Left**	**Right**
Maxillary (0,1,2)		
Anterior ethmoid (0.1,2)		
Posterior ethmoid (0.1.2)		
Sphenoid (0,1,2)		
Frontal (0,1,2)		
Ostiomeatal complex (0 or 2 only)		
Total points		

For all systems except the ostiomeatal complex, 0=no abnormalities, 1=partial opacification, 2=total opacification

For the ostiomeatal complex, 0=not occluded, 2=occluded

Table 3.

Demographic information collected for the Lund-Mackay scoring system

Last name
First name
Sex
Date of birth
Age

Hospital no.
Operation
Operation date
Surgeon
Nasal diagnosis (0-4)
Systemic diagnosis
General or local anaesthetic
Duration (min)
Post-operative medication
Complications

Table 4.

Radiological grading of anatomic variants in the Lund-Mackay scoring system

Anatomic variant	Left	Right
Concha bullosa		
Paradoxical middle turbinate		
Everted uncinate process		
Infra-orbital cells (Haller)		
Spheno-ethmoidal cell (Onodi)		
Agger nasi cells		
Total points		

Scoring: 0=no variant, 1=variant present

Table 7

Endoscopic appearance in the Lund-Mackay scoring system

Characteristic	Baseline	After 3 months	After 6 months	After 1 year	After 2 years
Polyp, left[a]					
Polyp, right[a]					
Oedema, left[b]					
Oedema, right[c]					
Discharge, left[c]					
Discharge, right[c]					
*Postoperative scores**					
Scarring, left[b]					
Scarring, right[b]					
Crusting, left[b]					
Crusting, right[b]					
Total points					

[a] Scoring: 0=absence of polyps; 1=polyps in the middle meatus only; 2=polyps beyond the middle meatus but not completely obstructing the nose; 3=com plete obstruction

[b] Scoring: 0=absent; 1=mild; 2=severe

[c] Scoring: 0=no discharge; 1=clear, thin discharge; 2=thick, purulent discharge

* to be used for outcome assessment only

Table 5.

Surgery scores in the Lund-Mackay scoring system

Surgery	Left	Right
Uncinectomy		
Middle-meatal antrostomy		
Anterior ethmoidectomy		
Posterior ethmoidectomy		
Sphenoidectomy		
Frontal-recess surgery		
Reduction of the middle turbinate		
Total points each side		

Score: 0=no procedure done, 1=surgery done. The maximum score is 14(7 each side)

Table 6.

Symptom scores in the Lund-Mackay scoring system.

Symptom	Pre-operation	After 3 months	After 6 months	After 1 year	After 2 years
Nasal blockage or congestion					
Headache					
Facial pain					
Problems of smell					
Nasal discharge sneezing					
Overall					
Total points					

Score each category 0-10 according to the degree of symptom severity, with 0=symptoms not present and 10=great severity

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