Nasalisation in the Surgical Treatment of Nasal Polyposis

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Core Messages

- The definition, pathophysiology and surgical approach of nasal polyposis (NP) are still under debate.
- In this chapter, NP is considered as a chronic inflammatory *disease* of the ethmoidal sinus mucosa characterised on nasal endoscopic examination by the presence, bilaterally, of non-infected white-oedematous polyps originating into the ethmoidal labyrinths and most of the time, arising from the middle and/or superior meatus and/or the sphenoethmoidal recess. This definition is aimed at stressing that NP is a specific disease that can easily be recognised among all other forms of rhinosinusitis and other nasal diseases.
- Our opinion is that sinus ventilation/drainage or obstruction in the ostio-meatal complex is a minor pathogenic factor in NP disease.
- Our hypothesis is that NP is a disease generated by remnants of vestigial olfactory mucosa scattered in the ethmoidal sinuses. Only people who have remnants develop NP. This vestigial olfactory mucosa has probably lost its histological features, but has kept some biological properties, among which is the ability to attract eosinophils. Olfaction is probably one of the oldest phylogenetic senses and eosinophils are probably one of the oldest cells of the innate immune system. Our hypothesis is that NP could be regarded as an inflammatory disease resulting from a dysfunction of the innate immune system associated to the olfactory organ. In this concept, the role of surgery for NP is to remove as much as possible of the vestigial ethmoidal mucosa.
- The role of the sinuses is still unclear and the need to retain more or less of the compartmentalisation of the ethmoidal labyrinths is also questionable. Our hypothesis is that, when dealing with the NP disease, complete removal of the bony lamellas partitioning the ethmoidal labyrinth is not more harmful than trying to restore ventilation/drainage in the different ethmoidal compartments.
- The combination of both hypotheses led us to propose the nasalisation procedure as a surgical approach for NP. The aim of the nasalisation procedure is to remove the ethmoidal mucosa as completely as possible without hazards, and to transform the ethmoidal labyrinth into a unique cavity opening into the nose (nasalisation).
- To achieve the nasalisation procedure, it is more important to know the anatomy of the ethmoidal walls than the compartmentalisation inside the ethmoidal labyrinth.
- The technical key point to safely perform a nasalisation procedure is to gently strip the mucosa to follow the bony structure of the medial orbital wall, ethmoidal roof and conchal lamina.
- Our results show that NP is a chronic disease which cannot be cured, but that the underlying chronic eosinophilic ethmoiditis disease seems

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to be better controlled after nasalisation than after ethmoidectomy.

› When the medical treatment with corticosteroids fails to stop the eosinophil attraction, the aim of surgery should be to remove as completely as possible the ethmoidal mucosa, which seems to be the main attractant for eosinophils.

30.1 Introduction

The definition, pathophysiology and surgical approach of nasal polyposis (NP) are still under debate. On the basis of 20 years of surgical experience, this chapter has been written for physicians seeing polyposis patients with overwhelming recurrences after limited surgical procedures or who cannot be rid of the multiple/continuous courses of systemic steroid treatments, or who want better functional results, especially in the sense of smell restoration.

30.2 Philosophy of the Nasalisation Surgical Procedure

The surgical treatment of NP is aimed at improving the *illness* of patients affected by chronic symptoms of rhinitis, sinusitis and for most of them, severe hyposmia or anosmia.

The pathophysiology of NP is still unclear and so are, also, the diagnosis criteria. In this chapter, NP is considered a chronic inflammatory *disease* of the ethmoidal sinus mucosa characterised on nasal endoscopic examination by the presence, bilaterally, of non-infected white oedematous polyps originating at the ethmoidal labyrinths and, most of the time, arising from the middle and/or superior meatus and/or the sphenoethmoidal recess. This definition is aimed at stressing that NP is a specific disease, which can easily be recognised from all other forms of rhinosinusitis and other nasal diseases.

The surgical concept of nasalisation has specifically been developed to treat the NP disease and improve NP illness. Our opinion is that sinus ventilation/drainage or obstruction in the ostio-meatal complex is a minor pathogenic factor in NP disease. NP is primarily a chronic inflammatory disease of the mucosa of the ethmoidal labyrinths.

The ethmoidal labyrinths look like vestigial structures of the primarily olfactory organ: human embryologic development still shows that the olfactory grooves have secondarily been exploited by the respiratory apparatus, a phenomenon, which probably occurred when life spread from water onto earth to adapt respiration to air breathing. The ethmoidal labyrinths, which are sinuses only described in humans, are the closest sinuses to the olfactory clefts, and may have been covered with olfactory mucosa (as are currently, for instance, the frontal and sphenoid sinuses in macrosmatic animals like the fox) in former times, before the bipedal human locomotion (which freed the hand and enhanced the role of vision) decreased the role of olfaction for survival and restricted the olfactory mucosal area to the roof of the olfactory clefts.

Our hypothesis is that NP is a disease generated by remnants of this vestigial olfactory mucosa scattered in the ethmoidal sinuses. Only people who have remnants develop NP. This vestigial olfactory mucosa has probably lost its histological features, but could still have kept some biological properties, among which the ability to attract eosinophils. Olfaction is probably one of the oldest phylogenetic senses and eosinophils are probably one of the oldest cells of the innate immune system. Our hypothesis is that NP could be regarded as an inflammatory disease resulting from a dysfunction of the innate immune system associated to the olfactory organ. In this concept, the role of surgery for NP is to remove as much as possible of the vestigial ethmoidal mucosa.

The role of the sinuses is still unclear and the need to retain more or less of the compartmentalisation of the ethmoidal labyrinths is also questionable. Our hypothesis is that, when dealing with the NP disease, complete removal of the bony lamellas partitioning the ethmoidal labyrinth is not more harmful than trying to restore ventilation/drainage in the different ethmoidal compartments.

The combination of both hypotheses led us to propose the nasalisation procedure as a surgical approach for NP. The aim of the nasalisation procedure is to remove the ethmoidal mucosa as completely as possible without hazards, and to transform the ethmoidal labyrinth into a unique cavity opening into the nose (nasalisation).

30.3 Nasalisation Technique

30.3.1 Anatomical Considerations

To achieve the nasalisation procedure, it is more important to know the anatomy of the ethmoidal walls than the compartmentalisation inside the ethmoidal labyrinth, as the dissection is performed centripetally along the medial orbital wall, the ethmoidal roof and the conchal lamina.

The *turbinate wall of the ethmoidal labyrinth* [1] is the medial wall, which separates the ethmoidal sinus from the *olfactory cleft*. The *conchal lamina* is a rectangular bony plate, which is attached below the cribriform plate, and from which the different ethmoidal turbinates originate (middle, superior and inconstant supreme turbinates). Since the cribriform plate lies more caudal than then the ethmoidal roof, the turbinate wall of the ethmoidal labyrinth is attached to the ethmoidal roof, thanks to the *lateral lamella* of the intracranial *olfactory groove* (Figs. 30.1 and 30.2).

The *olfactory cleft* is a narrow chamber between the turbinate wall of the ethmoidal labyrinth and the corresponding nasal septum, closed superiorly by the cribriform plate, posteriorly by the anterior wall of the sphenoid, anteriorly in its superior portion by the nasal bone attached to the frontal bone, and opened into the nasal fossa inferiorly and anteriorly in its inferior portion. The olfactory neuroepithelium is located at the

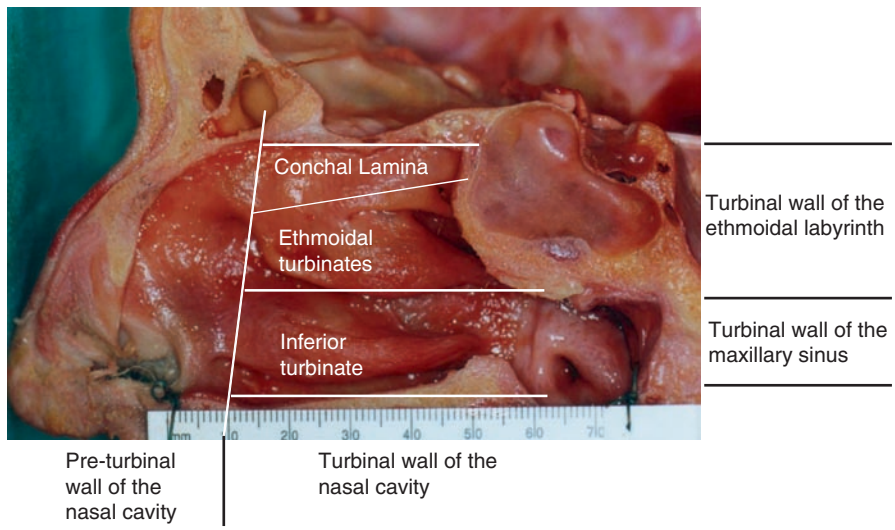

Fig. 30.1 Description of the lateral nasal wall [1]

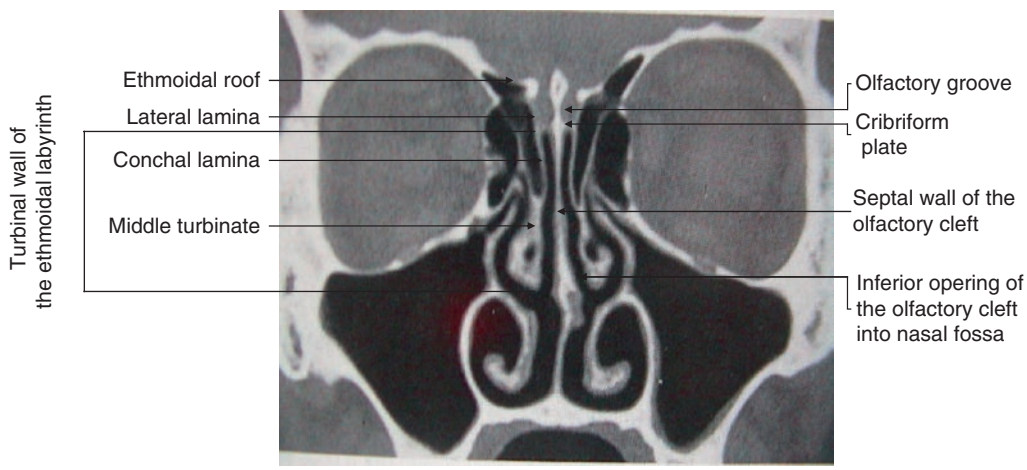

Fig. 30.2 Relationship between olfactory cleft and groove and the ethmoidal labyrinth [14].

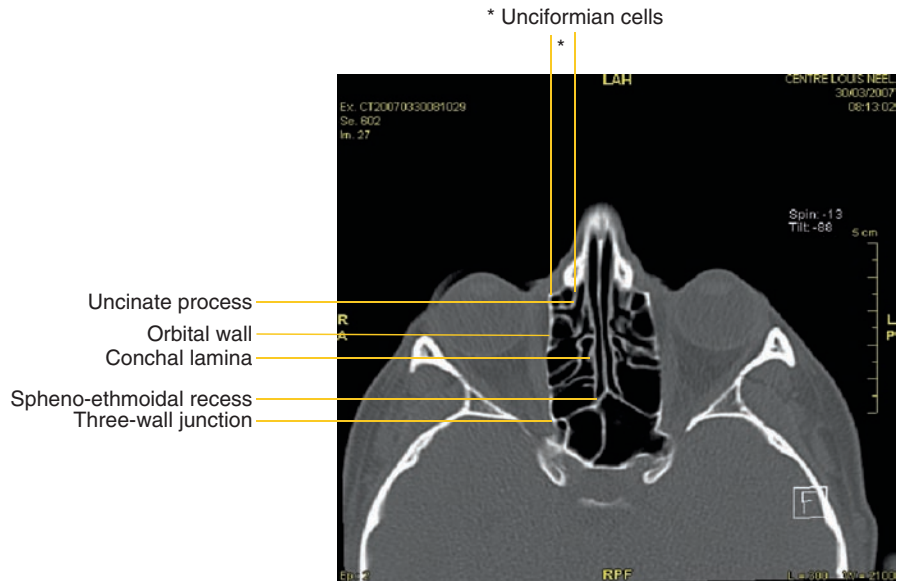

Fig. 30.3 The J shape of the anterior ethmoidal wall and compartment of unciformian cells

upper portion of the olfactory cleft, spreading from the cribriform plate onto the conchal lamina and corresponding nasal septum, whereas the inferior portion of the olfactory cleft (middle turbinate and corresponding nasal septum) is covered with respiratory mucosa. Functionally, the olfactory cleft can be divided in two portions: inferiorly, the *olfactory cleft vestibule* and superiorly, the *sensory olfactory cleft* (Fig. 30.2).

On the coronal plane, the *median orbital wall* has a C shape and its attachment to the ethmoidal roof can follow a sharp angle with the presence of the supra orbital cells (Fig. 30.2). On the axial plane, the median orbital wall has, anteriorly, a horizontal J shape (Fig. 30.3). In between both the attachments of the uncinate process and the orbital wall on the ascending process of the maxilla is a group of ethmoidal cells forming the *unciformian compartment*.

30.3.2 Set-Up

The nasalisation procedure is performed under general anaesthesia. The following four measures are helpful to control the bleeding per-operatively:

- A deep and stable general anaesthetic administered along with efficient analgesic agents [2].
- A 10–20° inclination of the operating table to bring the head up and the feet down.
- Local infiltration, with 8–12 mL of a solution made up of 20 mL 1% lignocaine and one ampoule of 0.25 mg adrenaline, of the neurovascular pedicles around the nose (supra and infra orbital nerves, dorsal nasal branch of ophthalmic artery, termination of facial artery along the nasogenial groove) and the anterior border of the septum and anterior aspect of the inferior turbinates.
- Packing of the nasal fossa with swabs soaked in lidocaine 5% with naphazoline 0.02%.

30.3.3 Surgical Dissection (Video)

After the removal of the nasal packing, a meticulous cleansing of the nasal fossa with the succion tube is combined with an endoscopic check-up looking for the origin and aspect of the polyps, the identification of the middle turbinate and the aspect of the olfactory cleft.

Polypectomy is performed to debulk the inferior and middle meati. Polyps prolapsing into the olfactory cleft are left intact, because at this stage of the procedure, it is frequently difficult to identify their true origin.

Using straight and/or 30° up biting Blakesley forceps, the heart of the ethmoidal labyrinth is holed to open an antero-posterior channel between the turbinate

wall of the ethmoidal labyrinth, the medial wall of the orbit and beneath the ethmoidal roof, staying at safe distance of these three walls.

The main steps of the nasalisation procedure, i.e. dissection of the medial orbital wall, ethmoidal turbinate wall and roof, are started now in an order and combinations that vary from one patient to another according to anatomic and pathologic variations. For the sake of the description, dissection of each wall is described separately.

The medial and inferior orbital walls form a continuous bony structure with a C-shape around the orbital content (Fig. 30.2). The easiest way to identify the inferior orbital wall is to enter the maxillary sinus. A large middle antrostomy dissected from the maxillary natural ostium anterior to the palatine bone, posterior and above the superior edge of the inferior turbinate, exposes clearly the inferior orbital wall. Mucosa can then be elevated in the underperiostium plane over a few millimetres to expose the bony inferior orbital wall into the maxillary sinus.

Dissection of the medial orbital wall actually starts anteriorly by elevating the mucosa on the ascending ramus of the maxilla bone. The underperiostium plane can easily be found here by strongly grasping without risking the full thickness of the mucosa, including the periostium on the solid bone of the maxilla ascending process. This flap elevated in posterior direction divides itself into two secondary flaps: one inferior, which detaches itself above the superior edge of the inferior turbinate, one superior, which turns around the maxilla ascending process towards its posterior face and opens the unciformian cell compartment towards the medial orbital wall.

Dissection of the unciformian compartment is carefully achieved by removing all bony partitioning lamellas and mucosa found behind the ascending process and above the maxillary natural ostium until the medial orbital wall is reached. At this stage, the junction between the inferior and medial orbital wall can clearly be identified at the level of the maxillary natural ostium through the large middle antrostomy.

Dissection of the junction is continued posteriorly, where Haller cells can be found and need to be opened and dissected. Remarkably, the dissection of the junction between the two orbital walls reaches the anterior sphenoid wall at the level where the posterior wall of the maxillary sinus reaches the anterior sphenoid wall.

Once this remarkable three-walled junction (Fig. 30.3) has been dissected, the dissection of the medial orbital wall can safely be continued superiorly. Elevation in the underperiostium plane and removal of the mucosa helps to follow this very thin bony plate without hazards. This dissection leads constantly to the discovery of the ethmoidal roof at one place or another.

Dissection of the turbinate wall of the ethmoidal labyrinth starts with the resection of the middle turbinate. C-curved cisors with the concavity turned down are placed 2 or 3 mm below the anterior attachment of the middle turbinate on the lateral nasal wall. A horizontal section separates the middle turbinate from the anterior portion of the conchal lamina and leads to the superior meatus. The middle turbinate falls down into the nasal fossa, but is still attached to the lateral nasal wall by its posterior end in the area of the sphenopalatine foramen. Section of posterior end and removal of the middle turbinate leave intact the conchal lamina and the superior (and supreme) turbinate(s) (Fig. 30.1), which protects the olfactory mucosa in the upper, sensory portion of the olfactory cleft.

Underperiostial elevation of the mucosa on the ethmoidal face of the conchal lamina can then easily be achieved by starting on the cut edge of the conchal lamina, where the three layers (ethmoidal mucosa – conchal lamina bone – olfactory cleft mucosa) are easy to identify. The conchal lamina is a thin uninterrupted bony plate [1] which prolongs the lateral lamina below the level of the cribriform plate (Fig. 30.2).

Elevation and removal of the mucosa allow better recognition and dissection of the ethmoidal cells attached on the conchal lamina bony plate and help to follow this bony plate without hazards from its anterior attachment on the maxillary ascending process to its posterior curvature towards the anterior sphenoid wall to form the sphenoethmoidal recess (Fig. 30.3). Upwards, dissection does not show any remarkable anatomic landmark or articulation between conchal lamina and lateral lamella. Actually, careful dissection with bony exposition of the conchal lamina plate on its ethmoidal face is helpful to avoid any damage to the cribriform plate, which is located on the other side of the conchal lamina and forms the roof of the olfactory cleft (Fig. 30.2). In most cases, polyps found in the olfactory cleft are prolapsing through the superior or supreme meati and originate in the ethmoidal cells attached on the posterior portion of the conchal lamina. Removal of the middle turbinate is necessary to access and clear these posterior ethmoidal cells with their polyps.

Dissection of the ethmoidal roof starts anteriorly. Anterior ethmoidectomy has already partially been achieved with the dissection, after middle turbinate resection, of the anterior conchal lamina and the dissection of the unciformian compartment with bony exposure of the medial orbital wall. The frontal natural ostium is usually easy to find at this stage by exploration of the anterior roof with a blunt, curved succion tube. The help of irrigation through a frontal drain is necessary in exceptional cases. Dissection of the ethmoidal cells and bullas around the frontal ostium can be challenging, but becomes easier after elevation and removal of the mucosa to expose the white hard bone of the anterior ethmoidal roof around the ostium. The anterior ethmoidal artery is encountered in 80% of the cases and is usually separated from the frontal ostium by one ethmoidal cell [6].

Dissection of the posterior ethmoidal roof is usually easy after former exposition of the bony anterior ethmoidal roof within the lateral bony limits of the conchal lamina and medial orbital wall. Dissection can now follow in the posterior direction these three landmarks, removing carefully a few residual ethmoidal cells, usually located at the junctions between the main ethmoidal walls. Dissection can, however, become difficult at the level where the ethmoidal roof reaches the sphenoid anterior wall, because of the presence of an Onodi cell or a particular sphenoethmoidal recess, or because like the Onodi cell on the lateral orbital wall, a posterior ethmoidal cell can expand medially or above the sphenoid sinus. A transethmoidal sphenoidotomy, starting inferiorly and progressing towards the ethmoidal roof in between the medial orbital wall and the conchal lamina, is usually helpful in these situations.

The procedure ends with revision of the surgical field, to suction the blood and remove the remaining bony lamellas or small mucosal flaps. No packing is necessary in our clinical set-up.

In summary, the technical key point to safely perform a nasalisation procedure is to gently strip the mucosa to follow the bony structure of the medial orbital wall, ethmoidal roof and conchal lamina.

30.3.4 Post-Operative Care

During surgery, antibiotic cover is provided by a single dose of cefuroxime (or, if allergic, erythromycin) on induction of anaesthesia. Packing is rarely necessary in case of bleeding. If a septoplasty has also been performed, silastic splints are inserted for 48 h.

Before 2004, the patient was given, before discharge from hospital, an intramuscular injection of slow release steroid (except contraindications) to control post-operative oedema and favour rapid healing of the mucosa in large (maxillary, frontal, sphenoidal) sinuses and in the nose. We have stopped this injection since then without apparently experiencing worse functional or anatomical results. Patients receive instructions to douche the nose three or more times each day. They are also prescribed a nasal steroid spray to use topically after douching only once or twice a day.

The first routine post-operative review is at 1 month when, if lavages have been carried out regularly, a few crusts will be noted and are removed to facilitate definitive cavity healing. In cases where lavage has been inefficient, certain patients require earlier review for more vigorous decrusting.

In approximately 10% of cases, patients present with significant headaches during the period between 3 and 15 days post surgery. Once meningitis has been excluded, treatment with antibiotics and analgesics is commenced and is generally effective within 48 h.

Nasal douching once or twice a day and local steroids once a day are recommended in the long term as part of the therapeutic plan for the treatment of NP.

30.4 Results

NP can be considered a chronic inflammatory disease. With this view in mind, nobody can cure NP. Therapeutical goals in chronic disease management are to improve symptoms and quality of life, to stabilise the disease-specific evolution and to avoid disease-specific complications by controlling the underlying pathophysiological mechanisms. Therapeutical goals in NP management are to restore normal nasal breathing and sense of smell and to control rhinorrhea, to stabilise and control recurrent attacks of sinusitis or asthma, and to prevent recurrence of nasal polyps by controlling the underlying chronic eosinophilic ethmoiditis on a long-term basis.

Simple polypectomy does restore nasal breathing in polyposis with severe obstruction and can be considered an effective treatment. There is no study in the literature comparing simple polypectomy to functional ethmoidectomy, which associates polypectomy to ventilation/

drainage restoration in the sinuses according to the extent of the disease. We have compared nasalisation to ethmoidectomy, but our results need to be confirmed by others.

1. Improvement in symptoms and quality of life

Data of our comparison study [11] show that 24 months after nasalisation, nasal obstruction, rhinorrhea and sense of smell are significantly improved than after ethmoidectomy. On a 10-point visual analogue scale, asking the patients 24 months after surgery "please evaluate your current nasal discomfort between 0=same nasal discomfort as before surgery and 10=I have a normal functioning nose", the answers were significantly better in the nasalisation than in the ethmoidectomy group. Figure 30.4 shows on one hand, that in the ethmoidectomy group only a few patients reported having a normal functioning nose whereas one third already reported feeling the same discomfort as before surgery. Figure 30.4 shows on the other hand, that in the nasalisation group half of the patients reported a normal functioning nose whereas no one felt the same discomfort as before surgery, no one scoring, actually, below five. On another visual analogue scale asking the patients to "please evaluate your current asthma status between +10=no asthma symptoms since surgery and −10=severe worsening of asthma since surgery, with 0=same asthma status as before surgery", the answers were also significantly in favour of nasalisation (Fig. 30.4).

2. Stabilisation of the disease-specific evolution

In a group of patients resistant to medical treatment, i.e. needing three or more short courses of systemic steroids per year despite permanent topical steroid therapy, we have observed that nasalisation was able to stabilise the disease again [9, 10]. Figure 30.5 shows that if we take the sense of smell as a marker of disease evolution on a repeatedly administered 10-point visual analogue scale, these patients had, at entry in the study, a very poor sense of smell, which was significantly improved after 7 days of systemic steroids, but this effect was of short duration as 2 months later, the sense of smell had disappeared again in most patients. The patients were then operated on according to the nasalisation procedure and received one intramuscular injection of slow release steroid the day after surgery.

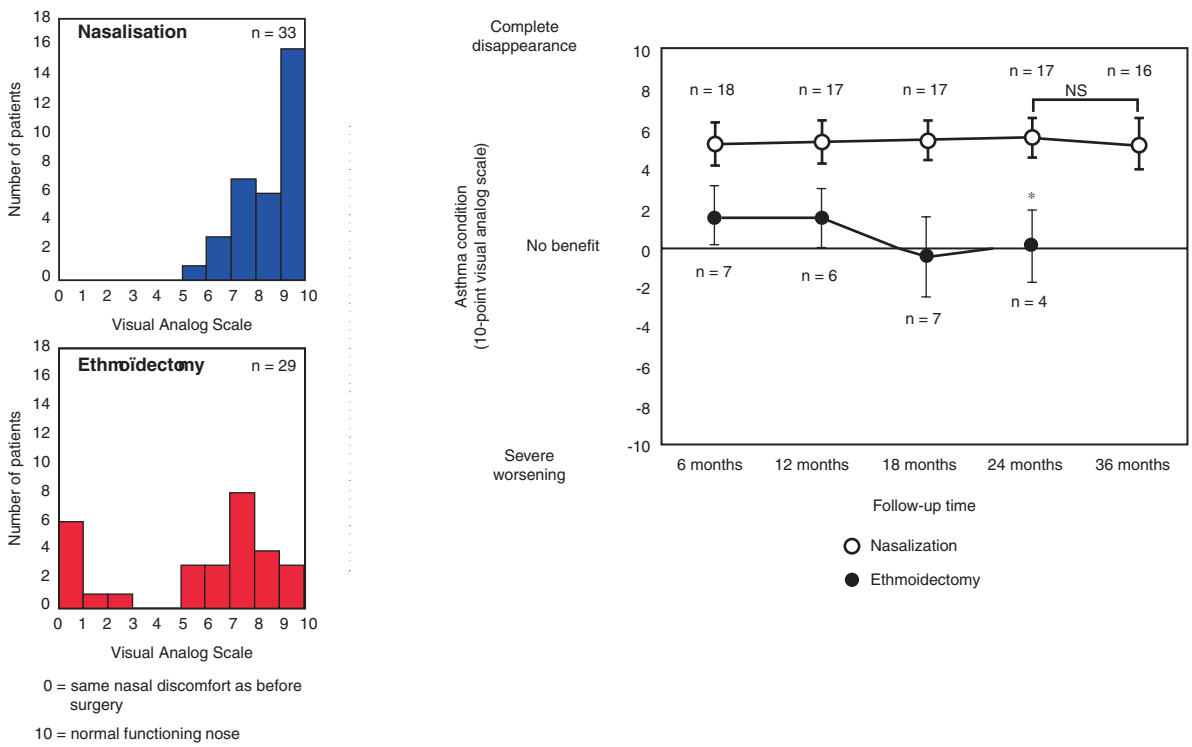

Fig. 30.4 Improvement in nasal discomfort and asthma 24 months after surgery [11]

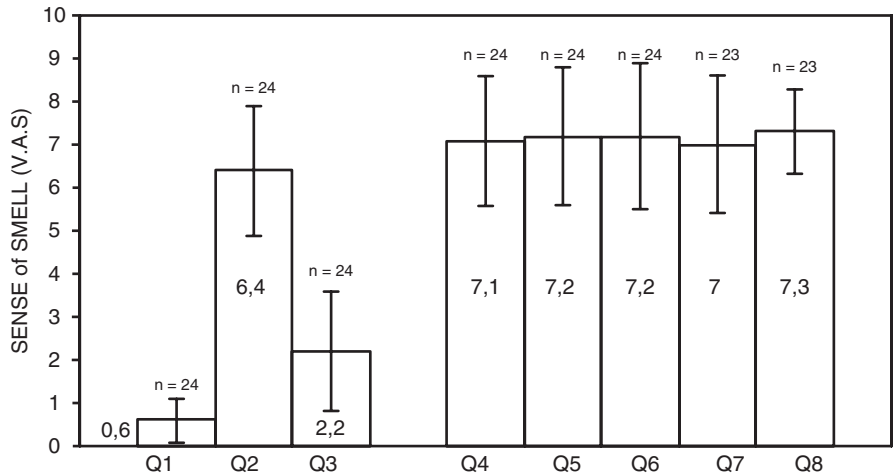

Fig. 30.5 Subjective evolution of the sense of smell after a 7-day treatment with systemic steroids and after nasalisation in anosmic patients (ANOVA $p<0.0001$) [9, 10]

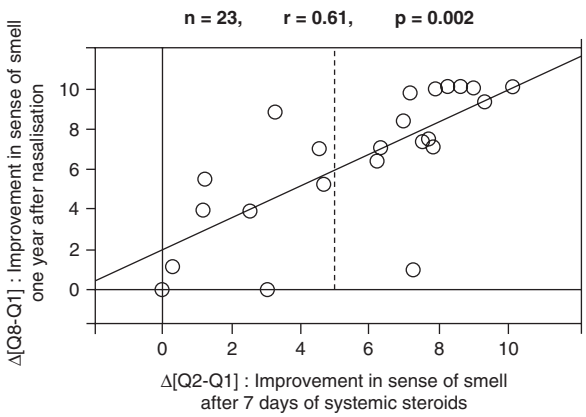

Fig. 30.6 Correlation between the subjective improvement in the sense of smell after 7 days of systemic steroids and the improvement 1 year after nasalisation [9, 10]

Figure 30.5 shows that 1 month later, the sense of smell had recovered to the level observed after 7 days of systemic steroids and that this level was maintained at least for 1 year after surgery, which was the end point of the study [9, 10]. None of these patients needed systemic steroid treatment during the year of follow-up after surgery. Interestingly, a significant correlation was found between the level in sense of smell restoration after the short course of systemic steroids and the level in sense of smell restoration after nasalisation (Fig. 30.6). The same schematic evolution was also observed for nasal obstruction and rhinorrhea [9, 10], indicating that nasalisation had stabilised the disease again and in most patients for far longer than 1 year.

3. Avoiding recurrence of nasal polyps

The patients of the study comparing nasalisation to ethmoidectomy were proposed to participate in a check-up 5 years later, including a self-administered questionnaire with 10-point visual analogue scales for symptom evaluation, and free endoscopic calibrated check up and CT at our hospital. After taking account of the patients lost to follow-up, the 5-year recurrence rate of nasal polyps was 58% in the ethmoidectomy group vs. 22% in the group that had undergone ethmoidectomy ($p<0.05$) [13].

4. Complications

No serious complications (death, meningitis, blindness) associated with the surgical procedure have been reported since 1987. No CSF leak or orbital haematoma has been observed since 1990. Minor complications (post-operative haemorrhage, ecchymosis, long-lasting crusting, etc.) are each less than 1%. The most frequent complication is mucocele formation [4]. The mean incidence rate of mucocele formation after nasalisation for NP was estimated to be of 2.5/100 patients per year. Most of the mucoceles were diagnosed during the first 6 years after nasalisation, with a peak incidence around year 2 and 3.

In summary, all these results together show that NP is a chronic disease which cannot be cured, but that the underlying chronic eosinophilic ethmoiditis disease seems to be better controlled after nasalisation than after ethmoidectomy.

30.5 Discussion: Controlling the Underlying Pathophysiological Mechanism

While the pathophysiology of NP is still unknown, there are some clue data which can help understand why nasalisation seems leading to a better control of the disease mechanisms.

Hotchkiss was the first to demonstrate in 1956 that systemic steroids were very effective in the treatment of NP [7]. The efficacy of topical steroids was shown in 1968 [15, 18]. Both treatments form the basis of the current medical treatment. We have observed in a retrospective study of polyp specimen collected during surgery [12] that: (1) the number of eosinophils was significantly and severely decreased in polyps of patients having received a short course of systemic steroids in the month before surgery compared to patients without treatment; (2) there was no difference in the number of eosinophils in polyps of patients without treatment and patients under topical steroids for more than 2 months before surgery, who actually were considered the failure of medical treatment and therefore needed to be operated. In polyps of patients responding to topical steroids, others have observed that the number of eosinophils was significantly decreased compared to the placebo group [3]. These results indicate that the disease can be controlled by decreasing the number of eosinophils in the polyp tissue. So, it seems that corticosteroids are clinically effective as long as they are able to reduce the number of eosinophils in the polyp tissue [17, 19], and it could be the same with surgery.

The ethmoidal mucosa seems to be the source of attraction for bone marrow eosinophils. On one hand, Wei et al. have shown in vitro that nasal tissue obtained from patients with chronic rhinosinusitis and asthma have the ability to attract peripheral blood eosinophils from both chronic rhinosinusitis patients and healthy control subjects, but that significantly more blood eosinophils were attracted by chronic rhinosinusitis patient tissues than healthy nasal tissue, suggesting that blood eosinophils in chronic rhinosinusitis patients are already specifically activated once they are in the blood stream on their way from the bone marrow to the sinus mucosa [20]. On the other hand, we have observed, in a retrospective study comparing the number of blood eosinophils in patients operated on NP and in a control group gathering patients without NP but operated on thyroidectomy or acoustic neuromas, that the number of blood eosinophils was twice more higher in the NP group, despite staying within the normal range. Linear multivariate analysis confirmed that NP was the main factor for this difference ($p<0.0001$) [8]. So, when the medical treatment with corticosteroids fails to stop the eosinophil attraction, the aim of the surgery should be to remove as completely as possible the ethmoidal mucosa, which seems to be the main attractant for eosinophils.

Our surgical experience of more than 20 years has shown that nasal polyps of the NP disease almost always originate from the ethmoidal labyrinth mucosa. We have never seen NP starting in the large sinuses but always in the ethmoidal labyrinths. We have even observed recently that when polyps are found in the olfactory cleft, they most of the time arise from the superior or supreme meati or turbinates, but that when they really originate into the olfactory cleft, their histology is that of respiratory epithelial adenomatoid hamartoma [21], which is a completely different entity [16]. Our hypothesis, already developed in the introduction, is that the current ethmoidal mucosa is a vestigial olfactory mucosa, which has lost its histological appearance but still has ancient biological properties, especially to attract eosinophils (one of the oldest cell of the innate immune system) to defend itself [8]. Our clinical experience also suggests that this vestigial olfactory mucosa could be diffusely spread in the ethmoidal mucosa in some patients, whereas in others, they could be present in a variable number of multiple spots. Asthma is found in more than 50% of patients with NP and the question of diffusion of this vestigial olfactory mucosa to the bronchus apparatus in patients with asthma can even be raised. Surprisingly, a paper reports that (1) asthma develops in healthy recipients after lung transplantation from mild asthmatic donors, despite complete neural disconnection and immunosuppressive therapy, and (2) that asthma disappears in an asthmatic recipient after lung transplantation from healthy donors [5]. So, some intrinsic signal seems to be located in the mucosa, both in asthma and in NP.

If the aim of surgery for NP seems to be to remove as completely as possible the ethmoidal mucosa, a far more important aim is not to harm the patient. It is far cleverer to leave a piece of mucosa than to provoke a complication. Incomplete ethmoidectomy has anyhow an efficacy, which can be very good in patients having polyps developed on multifocal areas within normal

respiratory mucosa in some ethmoidal cells. If we could know the location of the vestigial mucosa in every case, only those with diffuse vestigial mucosa should be operated according to the nasalisation procedure. As we do not know the pattern of distribution of this vestigial mucosa in each individual patient and what is the physiological need of keeping some of the ethmoidal cells or compartments unoperated, we believe that nasalisation is the appropriate treatment for NP.

Take Home Pearls

- Nasal polyposis (NP) is a specific disease characterized by the presence, bilaterally, of non-infected white-oedematous polyps originating from the ethmoidal labyrinths.
- Ventilation/drainage or obstruction in the ostiomeatal complex is a minor pathogenic factor in NP disease. Our hypothesis is that NP is a disease generated by vestigial remnants of the olfactory mucosa.
- The aim of the nasalization procedure is to remove the ethmoidal mucosa as completely as possible without hazards, and to transform the ethmoidal labyrinth into a unique cavity opening into the nose.
- To achieve the nasalization procedure, it is more important to know the anatomy of the ethmoidal walls than the compartimentalisation inside the ethmoidal labyrinth.
- The technical key point to safely perform a nasalization procedure I to gently strip the mucosa to follow the bony structures of the medial orbital wall, ethmoidal roof and conchal lamina.

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