Rhinitis

Mechanisms and Management

Edited by Ian Mackay

Preface

The idea for this book was first mooted in the summer of 1988 and a meeting took place in Geneva in October of that year to discuss the scope of the book and decide what particular aspects of the disease should be considered. The results of these discussions will appear in print in March 1989 - a satisfactorily swift publication which therefore contains most recent thoughts on the mechanism and management of rhinitis.

The book begins with the anatomy and physiology of the nose and then considers the various causes and manifestations of rhinitis and particular problems such as nasal polyps, sinusitis and the loss of sense of smell. Aspects of diagnosis are considered and topical management, systemic management and surgical treatment are reviewed. Finally, it considers the problem of nasal allergy in children and allergic conditions of the nose in asthmatics.

I have been most fortunate in having such a distinguished group of contributors - it has been a joy to work with them all. I would like to take this opportunity of thanking them for their hard work and for meeting the stringent deadlines that were set in order to bring about a rapid publication.

I wish also to express my gratitude to Yvonne Rue, Publications Department of the Royal Society of Medicine for her invaluable help and forebearance, Amanda Laman, my secretary, for her help not only with my own chapters but also with the general administration of the book and, finally, Madeleine, my wife, who has been encouraging and tolerant of the lost weekends never to be recaptured.

Ian S. Mackay
March 1989

Chapter 1

Introduction

I. S. Mackay

It is the aim of this book to explore the mechanisms and management of rhinitis, a common and yet sometimes surprisingly complex condition. Rhinitis is inflammation of the lining of the nose. Although allergy is a common cause of rhinitis it is by no means the only one, as inflammation may arise from a multitude of factors. The symptoms will also vary markedly from the itching, sneezing and watery rhinorrhoea associated with allergy to the dry, crusting, over-patent airway seen in atrophic rhinitis. The lining of the nose and paranasal sinuses is continuous and it would be rare for inflammation to affect one without the other, hence the inclusion of the sinuses in this and following chapters.

The aetiology of rhinitis may be classified as allergic and non-allergic, allergic being divided into seasonal and non-seasonal and non-allergic into infective and non-infective (Table 1). Infective rhinitis may be acute or chronic and the latter may be specific or non-specific. Up to this point the classification is relatively simple but the last group - non-allergic non-infective rhinitis - is the most difficult to classify further and includes such conditions as vasomotor rhinitis and factors which may cause or mimic rhinitis such as anatomical obstruction, tumours and granulomatous conditions.

Allergic Rhinitis

Allergic rhinitis usually presents least trouble in diagnosis and management. In the UK seasonal allergy is most commonly associated with grass pollen in the springtime though seasonal allergies may occur to allergens prevalent in the autumn and winter.

The history is all important and a patient presenting with streaming eyes, itching and watery nose together with sneezing and nasal obstruction recurring in the spring months should not present too much of a diagnostic challenge. It can be more difficult to detect an allergy occurring at other times of the year but the seasonal nature of the symptoms should suggest the diagnosis, which can usually be confirmed with skin testing. Blood tests for specific IgE will usually also be positive though this is seldom necessary.

The majority of patients with these symptoms will respond quickly and well to topical steroids and systemic antihistamines and the introduction of the non-sedating H_1 antagonists over the past few years has greatly improved the management of this problem. Topical sodium cromoglycate is also useful for this condition, though it has the disadvantage that frequent application is required and compliance may therefore not be as good.

Perennial Rhinitis

Perennial rhinitis is often more difficult to diagnose. The history can sometimes be helpful and some patients already have their own suspicions as to the possible allergen. The time of onset of symptoms may give a lead, for example, patients who wake up in the morning with fits of sneezing, watery rhinorrhoea and nasal block may suggest a possible

house dust mite allergy. Skin testing can be helpful, though many subjects who have no history of allergy can be shown to be atopic on skin prick testing and it can sometimes be difficult to know how relevant this is. Nasal challenges may be helpful, though these tests are complicated and time consuming to perform.

Table 1. Classification of rhinitis

1. Allergic
    - (a) **Seasonal**
    - (b) **Perennial**
2. Non-Allergic
    - (a) **Infective**
        - (i) Acute
        - (ii) Chronic
            - Specific
            - Non-specific
                - Immune deficiency
                - Clearance abnormality
    - (b) **Non-infective**
        - (i) **Hyperreactive (vasomotor rhinitis)**
            - Autonomic imbalance
            - Post-infective
            - Hormonal
            - Drug induced
            - Emotional
        - (ii) **Anatomical (and mechanical)**
            - Choanal atresia
            - Adenoids
            - Septal deformities
            - Hypertrophic turbinates
            - Polyps
            - Foreign bodies
        - (iii) **Tumours**
            - Benign
            - Malignant
                - Primary
                - Secondary
            - Non-healing granulomas.

Perennial symptoms are more difficult to control than those of seasonal allergy. Few patients with seasonal rhinitis would have symptoms severe enough to require systemic corticosteroids; if necessary, however, a short systemic course is unlikely to cause side-effects. For the perennial sufferer, however, long-term systemic corticosteroids to control purely nasal symptoms are seldom justifiable, and are normally used only to gain initial control. Topical corticosteroids, on the other hand, can be used long-term without risk of side-effects. They may need to be combined with systemic antihistamines or, if watery rhinorrhoea is a major problem, with topical anticholinergics. If the allergen can be identified, particularly if it is a single allergen, it may be possible to avoid it though in practice this often proves very

difficult. Hyposensitisation is seldom used in the UK following the report of the Committee on Safety of Medicines in the *British Medical Journal* in 1986, though it remains popular in many other parts of the world.

Infective Rhinitis

Acute rhinitis

Almost everyone experiences acute rhinitis from time to time as "the common cold" and apart from "tender loving care" little or no treatment should be required. Patients may, however, seek help for their nasal congestion or may treat themselves with topical nasal decongestants. These will do no harm providing they are used short term; however, since they may cause rebound, the patient is tempted to use ever increasing amounts and long-term use could result in rhinitis medicamentosa.

Acute upper respiratory tract infections are usually due to viral agents and as such antibiotics play no useful role. Occasionally however nasal congestion may result in blockage of the sinus ostia and secondary bacterial infection resulting in acute bacterial sinusitis. Increasing oedema of the sinus mucosa leads to further obstruction leading to a negative pressure and low oxygen concentration. This, combined with a poor blood supply, may explain the relatively high frequency of anaerobic organisms found.

Most cases of acute sinusitis will resolve spontaneously in two weeks without any treatment. There is a risk, however, that acute infection may lead to chronic oedema and blockage of the sinuses and eventually a vicious circle evolves resulting in chronic sinusitis. Treatment with broad spectrum antibiotics and topical decongestants or topical antiinflammatory drugs is advisable and the patient with acute sinusitis should be carefully followed up to ensure there is complete resolution. If this does not occur, it may be necessary to carry out antral lavage or endoscopic surgery aimed at optimal assessment, followed if necessary by functional surgery to improve ventilation and drainage.

Chronic rhinitis

Chronic rhinitis may be due to infection with a specific organism such as syphilis, tuberculosis, chronic diphtheric rhinitis, rhinoscleroma *(Klebsiella rhinoscleromatis),* leprosy, yaws *(Treponema pertenua)* or chronic glanders *(Loefflerella mallei).* Atrophic rhinitis is characterised by progressive atrophy of the mucosa and underlying bone of the turbinates with viscid mucus which dries to form crusts and emits a foul odor (ozaena). This is normally due to chronic infection, possibly with specific organisms, but it may follow overzealous surgery to improve the nasal airway.

The nose and sinuses may also be chronically infected by fungi and yeasts: Rhinosporidosis, the Phycomycoses, Aspergillosis, Blastomycosis, Cryptococcosis, Actinomycosis, Candidiasis, Histoplasmosis and Sporotrichosis.

Nasal myasis is seen in hot and humid climates, particularly in India where it is known as "penash" and is due to infestation of the nasal cavities by maggots, the larvae of a fly (genus Chrysomyia).

Non-specific chronic infection of the nose and paranasal sinuses is a very much more common condition in the UK and raises the question: what is the underlying cause? Because the nose and paranasal sinuses are in the "front line" of the respiratory tract they encounter greater attack from environmental agents, so it is not surprising that rhinosinusitis is frequently the first presentation of systemic immune deficiency.

In a series in the Brompton Hospital Nose Clinic, nine of 250 patients presenting with upper respiratory tract symptoms were found to have significant immunoglobulin deficiency. Five of these patients with severe panhypogammaglobulinaemia, who had been referred from chest physicians, gave a history of having initially presented to otolaryngologists with infective upper respiratory tract symptoms before developing irreversible lung disease. Lack of IgA or certain subclasses of IgG may also be responsible for repeated upper respiratory tract infection.

Patients may be compromised by treatment with immunosuppressant drugs or may have acquired immune deficiency syndrome (AIDS). The latter may present with a wide variety of otolaryngological complaints but rhinitis is not uncommon.

Another cause of chronic or recurrent acute infection of the nose and paranasal sinuses is a mucociliary clearance abnormality. Apart from the nasal vestibule and superior turbinate and corresponding portion of the septum, the remainder of the nose and sinuses is lined with respiratory epithelium. The 100 or more cilia lining the surface of each cell beat at approximately 12 beats a second and propel a gel layer of mucus over the more fluid underlying sol layer.

This mucociliary system comprises the first line of defence for both upper and lower respiratory tracts, trapping and removing inhaled microorganisms, allergens and noxious agents. This system will cease to operate in the presence of infection which may alter the viscosity of the mucus or prevent cilia from beating. These secondary clearance problems are common and may follow any upper respiratory tract infection. Primary abnormalities of either cilia or mucus are very much rarer.

In 1933 Kartagener describe a syndrome consisting of bronchiectasis, sinusitis and situs inversus but it was not until 1976 that Afzelius, Pedersen and Mygind revealed that this was associated with immotility or partial motility of the cilia (primary ciliary dyskinaesia). This leads to a build-up of mucus in the sinuses causing sinusitis, bronchiectasis due to failure to clear bronchial secretions, and dextrocardia (in half the patients) due to random rotation of the archenteron. These patients are also subfertile as the males' sperms lack motility and the females lack the necessary ciliary activity within the fallopian tubes.

Bronchiectasis, sinusitis and reduced fertility may also be seen in patients with normal ciliary activity but abnormally viscid mucus - Young's syndrome. Examination of the semen reveals azoospermia and exploratory scrototomy reveals normal spermatogenesis, but functional obstruction of sperm transport down the genital tract at the level of the caput epididymis where the sperm are found in viscous, lipid-rich fluid.

The nasal mucociliary clearance mechanism can be measured with a 0.5 mm particle of saccharin placed approximately 1 cm behind the anterior end of the inferior turbinate. The

particle is swept backwards to the nasopharynx and the patient perceives a sweet taste. The time taken for this to occur is recorded as the nasal mucociliary clearance time in minutes. Normally this will occur in 10-20 minutes, if it is delayed beyond this it is worth taking a small brushing of the lateral aspect of the inferior turbinate, using a fibreoptic bronchoscopy cytology brush. The specimens can then be transferred to a buffered saline solution and mounted on a coverslip-slide preparation sealed with silicone grease. A photometric method is then used to measure the beat frequency. If this is not available it is still worth looking at the specimen under the ordinary light microscope at high power, when the cilia can normally be seen to be beating briskly.

In the absence of an effective mucociliary mechanism, surgery to improve drainage of the sinuses by gravity should be considered, though in practice many of these patients have surprisingly little trouble with the nose. The importance of recognising the condition, however, is to make the diagnosis and refer the patients to a chest clinic in the hope that effective management will prevent the patient developing irreversible lung damage.

Non-Allergic, Non-Infective Rhinitis

Non-allergic, non-infective rhinitis is considered here under three headings: Hyperreactive, Anatomical and Tumours. The latter two are perhaps the more straightforward and will be dealt with first.

Anatomical

Anatomical and mechanical obstruction may cause nasal block and because retained secretions lead to infection, may cause inflammation and symptoms of rhinitis.

Choanal atresia, if bilateral, will result in symptoms within hours of birth, as new-born infants are obligatory nose breathers and feeding is considerably impeded. An airway is strapped into position and the baby fed via a tube to overcome the emergency situation, but corrective surgery will usually be performed within 48 hours. Unilateral atresia however may not present for some years, indeed in some rare cases not until middle or even late adult life and should always be considered as a possible diagnosis in the patient complaining of unilateral obstruction.

Adenoids may cause obstructive symptoms in early childhood and be responsible for recurring or persistent catarrh. In addition to this it may be a contributory factor responsible for recurring otitis media and serous otitis media (glue ear) and adenoidectomy should be considered. Adenoids will usually atrophy away completely by the time the patient reaches their teens and it would be extremely rare, though not unheard of, for an adult to have any visible adenoidal tissue present in the nasopharynx.

Deviation of the nasal septum will in many cases cause nasal obstruction and in some cases will even block the drainage of the sinuses by pushing on the middle turbinate and obstructing the middle meatus. Deviation of the nasal septum anteriorly may result in excessive dryness of the overlying mucosa, predisposing the patient to recurring nasal vestibulitis. In these cases the nasal septum will need to be corrected by septal surgery and

in some cases a septorhinoplasty may be indicated to straighten the septum and external pyramid of the nose.

The turbinates may be enlarged as a result of the underlying pathophysiology with swollen, boggy mucous membranes, or in other instances may be due to skeletal hypertrophy of the underlying turbinate bones. The latter is particularly troublesome in the presence of a narrow nose. Surgical reduction of the turbinates will be indicated for the latter group or for the former if they do not respond to medical treatment.

The cause of nasal polyps is, in most cases, unknown. Many authors have concluded that polyps are the result of allergy, but the incidence of atopy amongst patients with polyps is no higher than in the population at large and patients with intrinsic asthma are more likely to have polyps than atopic asthmatics. The highest incidence of nasal polyps is seen in the group of patients with aspirin (analgesic) induced asthma. Polyps in children should always arouse the suspicion that the patient might have cystic fibrosis as they are extremely rare in children without this condition, indeed so rare that if cystic fibrosis is excluded, a CT scan should be requested to ensure that it is a simple nasal polyp and not some intracranial abnormality presenting as a polyp.

All children with unilateral nasal obstruction and purulent rhinorrhoea should be assumed to have a foreign body until proved otherwise.

Tumours

Tumours of the nose and paranasal sinuses are fortunately rare but perhaps for this very reason they are often missed. The symptoms of nasal block, mucoid rhinorrhoea and facial pain are only too easily confused with rhinosinusitis. X-rays of the sinuses are undertaken on all patients seen in the Nose Clinic at the Brompton Hospital as a matter of routine. Any patient with a unilateral opacity, particularly middle aged or elderly patients, should be regarded with a high index of suspicion. X-rays of the paranasal sinuses can be misleading and a CT scan or tomograms should be requested wherever any doubt exists or prior to undertaking endoscopic examination and biopsy.

Non-healing granulomas - Wegener's and lethal midline granuloma - usually present with symptoms of a persistent "cold", complicated by blood-stained nasal discharge. Examination of the nose reveals some hypertrophy of the nasal mucosa with granulation tissue, blood clot and crusting which can be associated with an unpleasant odour. The diagnosis will depend on taking an adequate biopsy (tiny fragments taken with a miniscule cupped biopsy forceps seldom harvests sufficient material for what the pathologists find, at the best of times, a difficult diagnosis).

Hyperreactive

Having excluded all other pathology one is left with a group of ill-defined conditions which are sometimes lumped together under the term "vasomotor rhinitis". This is a rather unsatisfactory name as the mechanism of all rhinitis, including allergic, is vasomotor. Here the term hyperreactive is used, though it is accepted that this is probably equally unsatisfactory for a group of somewhat unrelated factors. Despite the afore-mentioned there

can be no doubt that some patients do have a nasal mucosa which will react to a minimal stimulus; a little cigarette smoke which may be regarded as slightly unpleasant by some will provoke profuse watery rhinorrhoea, sneezing and blockage in others. This is the basis of the nasal challenge tests which may use histamine, methacholine or other stimulants, in measured quantity to provoke a reaction. Increased resistance can be measured with a rhinomanometer.

Hyperreactive mucosa may result from infection and many of these patients will date their initial symptoms to a particular virus-like upper respiratory tract illness, but in most patients there is no such history.

Hormonal factors may result in the nose becoming hyperreactive. Pregnancy is not uncommonly associated with symptoms of rhinitis and the blockage of the nose with this condition can be a real problem for the patient. Hyper-reactivity in the form of excessive watery rhinorrhoea when exposed to minimal changes in temperature may occur in elderly men (old mans drip) and in the past has been shown to respond to treatment with testosterone. More recently hypertrophy of the nasal mucosa has been shown to occur in patients with acromegaly.

Alpha-adrenergic blocking agents used in the treatment of hypertension, such as guanethidine and bretylium tosylate, may cause vasodilation and nasal obstruction, as may methyl dopa and reserpine which deplete sympathetic nerve endings of the catecholamine stores. Nasal stuffiness may also result from drugs used to cause peripheral vasodilatation for the treatment of migraine and peripheral vascular disease.

Finally and most conjectural of all is the possibility that stress and other emotions (sexual arousal) may cause nasal stuffiness and other symptoms of hyper-reactivity. Eccles and Lee proposed that prolonged exposure to stress could result in failure of the hypothalamic control over sympathetic innervation leading to autonomic imbalance. Stimulation of the parasympathetic supply to the nose will cause it to block and increase secretions. If one accepts that stress may cause increased gastric secretions resulting in ulceration, or perhaps irritable bowel syndrome further down the gastrointestinal tract, then it would certainly not seem impossible that a similar parasympathetic over-activity in the nose could cause rhinitis.

There can be no doubt that over the past decade or so there has been an enormous increase in our understanding of the various factors underlying the mechanisms of rhinitis. We now have at our disposal highly sophisticated blood tests to investigate allergy and systemic immune deficiency, tests to investigate nasal mucociliary clearance mechanism, rhinomanometers to measure nasal resistance, endoscopes, both rigid and flexible, to give unparalleled views of the lining of the nose and sinuses and CT and magnetic resonance scanning which can produce previously unimaginably clear pictures of the anatomy and demonstrate the extent of pathology with a precision that, until quite recently, had been thought to be impossible. Great strides have been made, but despite this the diagnosis of some cases, though fewer than before, remains a mystery and one can symphatise with the surgeon who commented that "post-nasal drip is a figment of the imagination of the patient or their general practitioner". Perhaps, in some cases, he was right!

Hand in hand with the progress made in diagnosis have been advances in management, with the introduction of topical steroids and the availability of non-sedating antihistamines

which have revolutionised the treatment of rhinitis. On the surgical side the Hopkins solid rod endoscopes have aided not only the diagnosis but also the surgical management with the introduction of endoscopic sinus surgery.

This chapter is an "overview" of the mechanisms and management of rhinitis and as such is intended as an introduction to the rest of the book. It is hoped that the reader will have found that it whetted his appetite to delve further into the following chapters, each of which will deal in greater depth with their individual topics.