Chapter 6: Rhinitis, sinusitis and associated chest disease

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The lining of the nose and paranasal sinuses is continuous and it would be rare for inflammation to affect one without the other. In addition, the symptoms of sinusitis, particularly chronic sinusitis, and rhinitis overlap to a large extent and it is therefore convenient to consider the two together as rhinosinusitis. Similarly, the upper respiratory tract (nose and paranasal passages) and lower respiratory tract (tracheobronchial tree and alveoli) are closely related, not only in anatomy, but also in a number of physiological functions and responses to the environment. Chest disease is therefore frequently associated with rhinosinusitis and both the otolaryngologist and chest physician may see principles of aetiology, pathogenesis, investigation and management of diseases of one tract reflected in the other - with instruction and advantage to each. The aim of this chapter is to consider the aetiology, differential diagnosis and medical treatment of rhinosinusitis with its associated chest diseases.

The complex interrelationship of rhinosinusitis and the chest is well exemplified by the presence of sinusitis in association with bronchiectasis, which was noted many years ago (Quinn and Meyer, 1929), acute exacerbation of chronic bronchitis (Ogilvie, 1941) and recurrent bronchitis. A study of 200 patients with chronic purulent sputum production, most with proven bronchiectasis, revealed a high prevalence (42%) of rhinosinusitis (Cole, 1981b). In studies of college students, asthma occurred in 17-19% of patients with allergic rhinitis and 56-74% of patients with asthma were found to have allergic rhinitis (Settipane, 1984a). Nasal polyposis in association with asthma is a well-known finding and of particular interest is the well-documented triad of asthma, nasal polyps and aspiration intolerance. Nasal polyposis in children raises the possibility of associated cystic fibrosis.

The role of allergy is discussed in detail in Chapter 7 but is briefly discussed here as an underlying aetiological factor. Rhinomanometry, radiology, nasal endoscopy, sinus surgery and complications of sinusitis are each dealt with under separate chapter headings while acute coryza and some of the rarer chronic inflammations are considered in Chapter 8.

Aetiology and differential diagnosis of rhinosinusitis

The symptoms of rhinitis include nasal obstruction and mucoid or purulent rhinorrhoea which may be anterior or, if posterior, often referred to as a postnasal drip. In addition, patients may complain of itching, sneezing, snoring, facial pain and headaches, anosmia and associated ageusia, and epistaxis.

Previous descriptions have classified rhinitis as infective and non-infective, the latter being subdivided into allergic and non-allergic. While this may have the advantage of simplicity, it overlooks the fact that rhinitis is often of multifactorial aetiology with considerable overlap of clinical manifestations. Patients with an underlying allergic problem may develop swelling of the lining of the nose and paranasal sinuses, leading to stasis and subsequent infection, but the presenting symptoms of mucopurulent rhinorrhoea, nasal obstruction and facial pain would not necessarily suggest an underlying allergic aetiology. Conversely, not all patients presenting with purulent mucus in the nasal fossae have infection.
a smear may reveal this to be due to eosinophils, which is crucial to recognize because the patient will often respond dramatically to topical or systemic corticosteroids. The classification used by the authors is summarized in Table 6.1.

Table 6.1 Aetiology of rhinitis

(1) Mechanical (trauma, tumour or foreign body)
(2) Allergy
(3) Mucociliary clearance abnormality
(4) Immunity deficiency
(5) Granulomatous conditions
(6) Autonomic imbalance
(7) Hormonal
(8) Iatrogenic.

Mechanical obstruction

This may be due to deviation of the nasal septum, the nose or both, enlargement of the turbinates or bulla ethmoidalis or hypertrophy of the adenoids. Mechanical obstruction, for whatever cause, will impair drainage and may result in rhinosinusitis.

Deviation of the nasal septum not only results in obstruction on the side to which the septum deviates, but compensatory hypertrophy of the turbinates on the opposite side may result in bilateral obstruction. This compensatory hypertrophy may block the frontonasal duct and the ostia draining the ethmoids and maxillary sinuses.

Tumours

Tumours of the sinuses, both benign and malignant, may cause mechanical obstruction leading to sinusitis. Harrison (1979) listed pain, nasal obstruction, swelling of the cheek and purulent nasal secretions as the commonest presenting symptoms of carcinoma of the paranasal sinuses and nasal cavity, symptoms which all too often are attributed to 'sinus' by the patient or his general practitioner. Once referred for specialist opinion, the possibility of an underlying malignancy should never be overlooked and, in any case with unilateral symptoms and signs, the index of suspicion should remain high and a biopsy taken at the earliest opportunity.

Foreign bodies

Foreign bodies in the nasal cavities, particularly in children, may present with nasal obstruction and rhinorrhoea which is usually mucopurulent, sometimes blood stained and occasionally associated with sneezing. Unilateral mucopurulent rhinorrhoea in children should be assumed to be due to a foreign body until proven otherwise. Foreign bodies may enter the nose through the anterior naris (children inserting buttons, beads and any other small objects which conveniently come to hand); the posterior naris, during vomiting, coughing and regurgitation; penetrating wounds and nasal injury; palatal perforation, as in cleft palate; sequestration of bone in situ after trauma; and calcification in situ to form a rhinolith.
Some foreign bodies appear to be inert and may remain for many years without producing any signs or symptoms. Should a foreign body become imbedded and surrounded by granulation tissue, it may then act as a nucleus for concretion to occur, becoming coated with calcium and magnesium phosphate and carbonate to form a rhinolith. According to Ransome (1979), this process may occur around an area of inspissated mucopus, or even a blood clot. Rhinoliths usually form near the floor of the nose and are radiopaque.

**Dental**

Dental roots and prosthetic materials used for dental filling may be forced into the antrum causing maxillary sinusitis. The danger of fractured dental root perforating the antrum is increased where there is periapical rarefaction secondary to periapical abscess. Wright (1979) stated that first molar roots are more likely to cause trouble than those of the second molar, despite the fact that the roots of the latter are more closely related to the antral floor. The roots of the first molars diverge more widely, increasing the difficulty of forceps extraction and increasing the chance of root fracture. Roots adjacent to edentulous spaces are most likely to be involved and males are affected more often than females.

Radiological evidence of a dental foreign body can sometimes be difficult to obtain. Standard occipitomental and lateral X-rays of the maxillary antrum have only a limited value. Occlusal films and an orthopantomogram may similarly fail to reveal a root once it has been forced into the sinus. If any doubt remains, sinuscopy should be undertaken.

**Allergy**

Allergy is undoubtedly a common cause of rhinitis, so common that many practitioners regard all forms of rhinitis as being of allergic aetiology. Clearly this is not the case (see Table 6.1).

Strictly speaking, the term 'allergic rhinitis' relates to the immediate immunoglobulin (IgE) antibody-mediated hypersensitivity reaction to specific allergens, most allergens being proteins with a molecular weight between 10,000 and 50,000. In practice, however, many allergens causing rhinitis are complex compounds often consisting of a number of immunogenic molecules called antigens. The term 'allergen extract' is thus used, for example pollen, house dust mite etc, each of which contain many antigens, all or only a few of which may induce the IgE response, leading to symptoms. IgE antibodies evoked by exposure to antigens become attached to the surface of tissue mast cells and basophil leucocytes as cytophilic antibodies. Interaction between the cell-bound IgE antibodies and the respective allergens results in the release of histamine and other mediators from these cells, causing the signs and symptoms of rhinitis.

Other mechanisms may result in release of a complex array of inflammatory mediators, for example non-specific irritants (cold air, fumes and dust), as well as ingested acetylsalicylic acid and certain food preservatives and dyes. Because these latter responses are not necessarily IgE mediated, the terms hyperreactivity or hyperresponsiveness are often used to encompass these forms of provocation.
The prevalence of allergic rhinitis varies markedly from study to study - 0.1% (Schwartz, 1952) to 28% (Malmberg, 1979). Part of the discrepancy is due to the difficulty of making a definite diagnosis in a condition where the symptoms may vary from a 'mild stuffy nose' to severe disability.

Skin tests, although helpful in many cases, can be misleading as studies have revealed an atopic skin test positive rate as high as 35% in healthy populations (Mygind and Lowenstein, 1982), and the skin is also a remote target from the nasal mucous membrane. Nasal smears revealed a prevalence rate of eosinophilia of approximately 25% (Malmberg, 1979), but not all patients with eosinophilia necessarily have allergic rhinitis. The term 'eosinophilic non-allergic rhinitis' (ENR) has been applied to a group of patients exhibiting nasal eosinophilia of at least 25% with no history of allergy and with negative skin tests. This group responds well to topical corticosteroids (Mullarkey, 1984).

Nasal secretions with a very high eosinophil count associated with allergic rhinitis may appear yellow or green and the patients may be diagnosed as having infective rhinosinusitis. Conversely, nasal secretions accompanying acute coryza may be clear and mistakenly assumed to be non-purulent, allergic rhinitis.

The distinction between allergic rhinitis and infective rhinitis is not always as clear-cut as it may first appear: the two may, and often do, occur at the same time, with allergy leading to swelling and inflammation of the nasal mucous membranes causing mechanical obstruction which impedes drainage and clearance from the sinuses and allows 'secondary' bacterial infection

**Mucociliary clearance abnormality**

The mucociliary system comprises the first line of defence for upper and lower respiratory tracts, trapping and removing inhaled microorganisms, allergens and noxious agents.

The nasal vestibule as far back as the nasal valves, is lined with skin and the olfactory region limited to the superior turbinate and corresponding portion of septum is lined with specialized sensory epithelium; the remainder of the nose and paranasal sinuses is lined with respiratory epithelium.

The epithelium comprises four main cell types: ciliated epithelial cells, non-ciliated epithelial cells, goblet cells and basal cells arranged in a pseudo-stratified pattern on an underlying basement membrane.

The cilia appendages of the ciliated cells are approximately 6 microm long, 0.25 microm in diameter and in the region of 100-200 to each cell. In cross-section, each cilium can be seen to have two central microtubules surrounded by a ring of nine doublet microtubules, protruding from one side of which are inner and outer dynein arms composed of ATPase protein responsible for the energy production required for the beating of the cilium.
Both ciliated and non-ciliated cells have 200-400 microvilli each, the number increasing towards the nasopharynx. These microvilli are approximately one-third of the size of the cilia and have a central core of actin filaments. Microvilli are not capable of active movement and their function is debatable but they probably promote ion and fluid transport between the cells and the periciliary fluid, regulating the composition of the latter and overlying mucus (Petruson, Hansson and Karlsson, 1984).

The goblet cells are unicellular mucus-secreting glands found above the basement membrane. They possess well-developed Golgi apparatus and endoplasmic reticulum of mainly granular type, consistent with high synthetic activity. The distribution and density of goblet cells have been extensively studied by Tos (1982) who found the highest density in the inferior turbinate (11,000 cells/mm²) and lowest in the septum (5,700/mm²), the density in the sinuses being in the mid-range but highest in the maxillary sinuses. Generally, there is a higher density of goblet cells posteriorly near the nasopharynx.

In addition to goblet cells within the epithelium, there are multicellular glands deep to the basement membrane in the lamina propria. The anterior serous nasal glands are not thought to play an important role in man. Small seromucous glands are evenly distributed in the mucosa of the respiratory region (8-9/mm²), although in the paranasal sinuses the density is very much lower (0.06-0.47/mm²) (Tos, 1982).

Nasal secretion is a complex mixture containing material secreted by the goblet cells, seromucous glands and lacrimal glands, material transported across the membrane of epithelial cells equipped with microvilli together with microorganisms and condensed water from expired air. Lucas and Douglas (1934) suggested that mucus had to be arranged in two layers for the mucociliary transport mechanism to be effective; a superficial viscid sheet, the gel layer, moving over underlying serous fluid, the sol layer, which bathes the cilia and microvilli. The cilia beat in the low-viscosity periciliary layer at a frequency of 12-15 Hz with a rapid 'stiff-armed' effective stroke during which claw-like projections from the tips of the cilia (Jeffery and Reid, 1975) engage the thick, viscous gel layer to propel this towards the nasopharynx. During the recovery phase, the cilia bend to return entirely within the thin sol layer in a plane at right angles to the effective beat and sweeping across the surface of the cell.

The transport rate of mucus averages about 6 mm/minute but a wide range is found in normal subjects. Slow clearance rates of less than 1 mm/minute have not been adequately explained but would appear to be related in most cases to variations in physicochemical properties of secretions rather than in the rate of ciliary beating. In a few cases, however, deficient clearance may be attributable to genetically determined abnormalities in ciliary morphology and function. Systemic dehydration and certain air pollutants may also reduce mucus clearance rates (Proctor, 1982) and there is evidence for bacterial products reducing mucus clearance by slowing ciliary beating.

In 1933, Kartagener described a syndrome consisting of bronchiectasis, sinusitis and situs inversus, but it was not until recently that this was associated with a genetically inherited autosomal recessive abnormality of the respiratory tract cilia, usually partial or complete dynein arm deficiency (Afzelius, 1976; Pedersen and Mygind, 1976; Eliasson et al, 1977). In this condition, which was initially termed 'immotile cilia syndrome', there is severe
disturbance of the normal pattern of ciliary beating with a very variable degree of immotility (Rossman et al, 1980) so that the preferred term is now 'primary ciliary dyskinesia'. Mucociliary clearance is profoundly impaired resulting in chronic or recurrent infection. Males are infertile because of the basically similar structure of cilia and the sperm tail, rendering the latter dyskinetic also (Pedersen and Rebbe, 1975). The relationship between primary ciliary dyskinesia and dextrocardia is postulated to be due to random rotation of the archenteron when cilia on embryonic cells are not functioning (Afzelius, 1976). The important implication of this is that only 50% of patients with this condition will be suspected by dextrocardia/situs inversus found on chest X-ray or at examination - therefore, one should not exclude the possibility of primary ciliary dyskinesia simply because dextrocardia is not found, that is one of the requirements for Kartagener's syndrome is not present. These patients may simply present with a life-long history of rhinorrhoea and cough.

While slow clearance may result from dyskinesia of the cilia, it is equally likely to occur with physicochemical abnormalities of the mucus. Patients with Young's syndrome (obstructive azoospermia, sinusitis and bronchiectasis or bronchitis) present with infertility, chronic sinusitis and often cough with purulent sputum - reminiscent of primary ciliary dyskinesia but examination of their cilia reveals that these are normal in structure and function. Examination of the semen reveals azoospermia and exploratory scrototomy reveals normal spermatogenesis, but a hold-up of sperm transport down the genital apparatus at the level of the caput epididymis where the sperm are found in a viscous, lipid-rich fluid, thought possibly to be due to a metabolic abnormality of the cells lining the tract (Young, 1970; Hendry, Parslow and Stendronska, 1983). Similar affection of respiratory mucus may account for the viscid sinus and respiratory secretions in this syndrome.

Young's and Kartagener's syndromes are excellent examples of primarily mucus and primarily ciliary abnormalities respectively.

Far more common, however, are secondary mucociliary transport abnormalities due to, for instance, upper respiratory tract infection - the nasal secretions become thick and mucopurulent and are not cleared in the normal manner. Viral infections of the upper respiratory tract damage the ciliated epithelium thereby reducing mucociliary clearance (Wilson et al, 1987a). Certain bacteria associated with chronic sinus and bronchial sepsis (Haemophilus influenzae, Streptococcus pneumoniae and Pseudomonas aeruginosa) have been shown to release factors slowing and disrupting cilia (Wilson, Roberts and Cole, 1985) and, in the case of P. aeruginosa, these factors have been characterized as the low-molecular-weight pigments pyocyanin and 1-hydroxyphenazine (Wilson et al, 1987b). Such molecules slow mucus transport in animal models and are found in human respiratory secretions during chronic sepsis in amounts sufficient to have a ciliary dyskinetic effect in vivo (Sykes et al, 1987).

Immunity deficiency

The defences

A variety of defence mechanisms eliminate foreign material breathed into upper and lower respiratory tracts. These can be considered under the headings of those 'resident' in the respiratory tract and those attracted from the systemic circulation when the local mechanisms
fail, and in each case, such defences can be divided into those which are non-specific and those which are immunologically specific (Table 6.2). This is an artificial division because, in practice, there is considerable interaction of the various defences, and such is this 'defence in depth' that it is sometimes very difficult to isolate a single defect as being the sole cause of infective problems in the respiratory tract.

Table 6.2 System of defences of the respiratory tract

Local ('resident') mechanisms

(1) Non-specific
reflexes (cough, sneeze)
mucociliary system
epithelial integrity and lining fluid (anatomical barrier)
antimicrobial substances in lining fluid (eg, lysozyme)
pulmonary macrophage

(2) Specific
immunoglobulin (secretory IgA, IgE)
lymphocytes

Systemic ('recruited') mechanisms

(1) Non-specific
serum factors (eg, opsonins, complement components)
granulocyte phagocytes
mononuclear phagocytes

(2) Specific
serum immunoglobulins (IgG, IgA, IgM, IgE)
lymphocytes.

Presentations to otolaryngologist and chest physician

Since the upper and lower respiratory tracts share common defence mechanisms, defects in them will be reflected as infective presentation to either otolaryngologist or chest physician, or both. Since the upper respiratory tract brooks a higher rate of environmental attack, the otolaryngologist may see infections first which will later affect the lung. It is crucial that these are recognized to be due to immunity deficiency (particularly those which are treatable) early, that is by the otolaryngologist, because by the time the patient presents to the chest physician with bronchopulmonary involvement, it is often too late for effective treatment. This is because the delicate architecture of the lung is easily damaged irreparably and, even though an immunity deficiency recognized at that stage may still be reconstituted, the lung damage is already irreversible. Attendant chronic disease and predisposition to further infection occurs, even when the immunity deficiency is treated, because of the distorted anatomy disrupting front line defences such as mucociliary clearance.
In general, there are three principal presentations of infection to the otolaryngologist and chest physician which cause problems in diagnosis and management. First, the acute, overwhelming infection usually due to profound immunity deficiency (for example, panhypogammaglobulinaemia; acquired immune deficiency syndrome, AIDS). Second, recurrent, acute episodes of infection with apparently normal periods intervening. Here, there is a prevalence of immunological abnormality (notably selective IgA deficiency) of between 40 and 70% in the lower respiratory tract (higher in recurrent pneumonia than bronchial infections). Recurrent viral upper respiratory infections usually herald such episodes. Third, the patient with chronic purulent bronchial disease associated with upper respiratory symptoms in over 80% of cases and frank chronic purulent sinusitis in over one-third of cases. Here, paradoxically, there is less than 10% prevalence of immunity deficiency but instead a 'vicious circle' of chronic mucosal damage (see below) which demands a particular attitude to treatment (Cole, 1981a). It is crucial that these three types of presentation should alert the otolaryngologist to the possibility of immunity deficiency or progressive form of disease requiring special approaches to treatment if irreparable damage is not to occur - and affect the lower respiratory tract which may prove fatal.

**Immunity deficiencies**

Patients with systemic immunity deficiency (for example lack of antibodies, panhypogammaglobulinaemia) frequently present with symptoms in the respiratory tract because of the intimate association of this tract with the environment and the potentially harmful agents in it. 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 such systemic immunity 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 (Mackay et al, 1983). 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.

The protective role of IgA in the respiratory tract is far from clear. A number of functions have been suggested, but data to support these roles in vivo in man are scanty. It has been estimated from blood transfusion screening, that selective IgA deficiency occurs in one in 500-700 healthy individuals (Bachmann, 1965). However, there is no doubt that some patients with severe, recurrent symptoms in the upper and lower respiratory tracts lack serum and secretory IgA and that there is a significant prevalence of this abnormality in patients presenting with infective rhinosinusitis and bronchial sepsis. Whether this deficiency is causal is doubtful - it may be a marker of other associated deficiency of defences.

Oxelius et al (1981) have shown an association of IgA deficiency with deficiency of certain subclasses of IgG (notably IgG2) in children, and it may be that the subclass deficiency is causal, since IgG2 is a particular important antibody against polysaccharide capsular antigens such as those of the microorganisms *H. influenzae* and pneumococcus. It is important that this is confirmed also for adult infective respiratory disease because,
although IgA deficiency is not routinely replaceable, normal human immunoglobulin replacement therapy can reconstitute IgG2 deficiency. Also, IgG2 subclass deficiency will be not diagnosed by routine quantitation of the total major immunoglobulin classes IgG, IgA and IgM, and requires separate estimation of IgG subclasses.

Despite the great advances in this field over the last few decades, it is still by no means rare to find patients who are unusually susceptible to upper respiratory tract infections and to be unable to identify any defect in defences, systemic disease or environmental exposure likely to offer a reasonable explanation (Andersen and Proctor, 1982).

**Chronic infection without classical immunity deficiency (the vicious circle)**

Over 80% of patients with chronic bronchial sepsis (complaining of daily purulent sputum production) suffer upper respiratory symptoms and over one-third have frank chronic purulent rhinosinusitis. It would appear paradoxical that less then 10% of these patients can be found to have classical immunity deficiency, so why do they have persistent infection?

There are a number of facts about these patients which suggest an answer to this question (Cole, 1984). First, they are mostly young (mean age approximately 40 years); second, the majority are non-smokers; third, they are usually wheezy; fourth, their upper and lower respiratory tracts are colonized by predominantly non-invasive microorganisms (for example uncapsulated *H. influenzae*); fifth, they respond to this colonizing microbial load with an exuberant immunological response in 80% of cases; sixth, they may suffer progressive (sometimes rapidly) disease with gross scarring of the airway/sinus mucosa and, sometimes, death due to ultimate respiratory failure. These facts can be explained by a normal short-lived useful host response to eliminate invading microorganisms becoming subverted into a tissue-damaging chronic inflammatory response.

The scheme depicts the situation in which a normal person disposes of an insult to the upper or lower airways by mucociliary clearance in the majority of instances - an acute, short-lived, controlled, useful inflammatory response removing the few insults not so eliminated.

**Normal elimination of foreign material from the respiratory tract  
('virtuous circle')**

Health --> 'Attacker' --> Mucociliary clearance occasionally with an acute, controlled inflammatory response --> Elimination of 'attacker' --> Health.

A predisposing damaging insult (for example virus infection) or underlying disease (cystic fibrosis) provides an ecological niche in which front-line clearance mechanisms are less than perfect. This allows microorganisms to loiter in the sinuses/bronchial tree and the ability of some of them to release molecules directly inhibiting ciliary function (Wilson, Roberts and Cole, 1985) enables these organisms to colonize and stimulate the host to continued attempts at eliminating them. The host response is chronic and inflammatory which, unfortunately, is relatively unselective and damages 'bystander' normal mucosal surfaces and tissues leading to progressive tissue damaging disease. This process, termed a 'vicious circle' (Cole, 1984), is the antithesis of acute invasive infection such as acute sinusitis or pneumonia since the microorganism (which is actively invasive in the latter) is relatively passive and the
tissue damage is mediated largely by host rather than microbe. This distinction is important for treatment as will be seen later.

**The 'vicious circle' of chronic inflammation of the respiratory tract**

Failure to eliminate 'attacker' --> Amplified inflammation:
--> Progressive damage to 'bystander' normal tissue
--> Impaired mucociliary clearance --> Release of microbial cilioinhibitory factors -->
Microbial colonization --> Amplified inflammation.

**Granulomatous conditions**

Granulomatous lesions in the nose may induce nasal obstruction and rhinorrhoea mimicking rhinitis due to other causes.

On occasion, nasal symptoms may be the presenting feature of multisystem granulomatous disease. Wegener's granulomatosis, sarcoidosis, Churg-Strauss syndrome and polymorphic reticulosis may all be associated with pulmonary and nasal lesions. The Churg-Strauss syndrome is characterized by the presence of bronchial asthma, eosinophilia and vasculitis with necrotizing granulomata (Olsen et al, 1980). Polymorphic reticulosis is a necrotizing lymphoproliferative lesion with a predilection for the upper respiratory tract and lungs, and locally obstructive lesions are diagnosed histologically by a characteristic angiocentric lymphoid infiltrate (McDonald, De Remee and Kern, 1974).

Granuloma formation is a host response seen in the case of certain infective agents which are not cleared by the primary defences of the respiratory tracts, blastomycosis, histoplasmosis, leprosy, rhinoscleroma, tuberculosis and syphilis, all of which are considered in Chapter 8.

Berylliosis produces a granulomatous response similar to sarcoidosis but a history of occupational exposure will raise suspicion of the diagnosis (McCaffrey and McDonald, 1983).

**Autonomic imbalance**

'Vasomotor rhinitis' is a term utilized when the findings fit no other category of disease. It is a convenient term by which to describe disease of unknown aetiology. The term often means different things to different physicians and surgeons, a shortcoming which makes it almost useless (Connell, 1984). 'Autonomic imbalance' is used in textbooks as a cover for basic ignorance in this area (Mygind and Lowenstein, 1982). Despite this criticism, this heading has been chosen to group together various well-recognized clinical entities which cannot readily be classified under any other headings and which logically can be attributed to imbalance of the components of autonomic innervation of the nose and paranasal sinuses.
Autonomic innervation

Sympathetic

This appears to be regulated by the hypothalamus as electrical stimulation of this area of the brain causes nasal vasoconstriction (Eccles and Lee, 1981). The preganglionic sympathetic fibres originate in the thoracolumbar region of the spinal cord from where they pass to the superior cervical ganglion via the vagosympathetic trunk. Postganglionic fibres pass from the superior cervical ganglion to the plexus around the internal carotid artery then via the deep petrosal nerve (which is joined by the preganglionic parasympathetic greater superficial petrosal nerve) to form the vidian nerve, the nerve of the pterygoid canal. The vidian nerve emerges from the pterygoid canal where it enlarges to form the sphenopalatine ganglion, the sympathetic nerves continue without synapsing to the turbinates. Sympathetic fibres also reach the nose from the carotid plexus via the infraorbital and ethmoidal branches of the trigeminal nerve (Eccles, 1982).

Parasympathetic

The preganglionic parasympathetic fibres originate in the facial nucleus of the brainstem from where they pass to form the greater superficial petrosal nerve. This is joined by the sympathetic fibres of the deep petrosal nerve to make up the vidian nerve which passes through the pterygoid canal to relay in the sphenopalatine ganglion. The postganglionic secretomotor fibres continue to the nasal mucosa.

In addition to the autonomic nerves, the nose is supplied by the olfactory nerve (first cranial nerve) to the olfactory epithelium in the uppermost part of the nose, and by the trigeminal nerve which supplies sensation in the upper part of the nose via the anterior ethmoidal nerve, a branch of the ophthalmic division, and in the lower part of the nose by the sphenopalatine nerve which is a branch of the maxillary division.

The activity of the autonomic nerves is influenced by reflexes which may be initiated in the nasal mucous membranes via the sensory nerve supply. This may involve the central nervous system or short reflex arcs: by either route, they will affect the autonomic balance controlling the secretory and vasomotor innervation of the nasal glands and blood vessels. Stimulation of the sympathetic nerves results in the release of noradrenaline which causes vasoconstriction in the venous erectile tissue. Stimulation of the parasympathetic supply causes the release of acetylcholine, resulting in a watery nasal secretion and vasodilatation. In addition to acetylcholine, other neurotransmitters may be involved; the roles of the vasoactive intestinal polypeptide (VIP) and substance P (SP) have been extensively investigated by Anggard (1981).

Stimulation of the vidian nerve in man has been carried out and the results reported by Rucci et al (1984). Histological changes indicated an increase in secretory activity, an increase in vasodilatation of both the deep venous plexus and the periglandular vascular supply and intense degranulation of mast cells, mainly in the vicinity of the glandular and vascular components.
Emotional rhinitis

It has been proposed (Eccles and Lee, 1981) that prolonged exposure to stress could result in failure of hypothalamic control over the sympathetic innervation leading to autonomic imbalance, causing rhinitis. In addition, increased nasal resistance has been reported in response to hyperventilation (Hasegawa and Kern, 1978), a common finding in patients with emotional rhinitis.

Emotional rhinitis may take many forms. One is entirely hysterical where examination and thorough investigation fail to reveal any abnormality: normal rhinomanometry findings in a patient complaining of nasal obstruction at the time of the test being a good example. In this type, the imagined nasal symptoms are usually associated with a multitude of other unrelated symptoms and the nasal symptoms should be looked upon simply as another 'call for help'. Certain cases of postnasal drip may fall into this category and one can sometimes sympathize with the statement that postnasal drip is a figment of the imagination of the patient or his general practitioner.

Other patients will present with a specific history that certain emotional events (public appearances, arguments, etc), will lead to nasal obstruction or even profuse watery rhinorrhea. The parasympathetic pathway and its central nervous systemic connection provide a network for such an occurrence (Connell, 1984).

Emotional factors affecting the nose were extensively investigated by Holmes et al (1950), who biopsied the nose during different periods of emotional conflict. They found that fear produced a 'sympathetic' response with vasoconstriction, while frustration, humiliation and anxiety resulted in a 'parasympathetic' response with engorgement of the mucous membranes.

Hyperreactivity

Autonomic imbalance with parasympathetic overactivity leads to hyperreactive mucous membranes which will respond to non-specific stimuli such as cold air (Krajina, Harvey and Ogura, 1972). Hyperreactivity, the 'twitchiness' of the mucous membranes, can be demonstrated by their responsiveness to intranasal histamine and methacholine challenge. Connell (1968) found that if symptoms could be produced by provocation with a single allergen, the response to the same provocation 2 days later was significantly more pronounced. He named this phenomenon 'the priming effect'.

In addition to parasympathetic overactivity and allergy, infection (particularly viral infection) may lead to the mucous membranes becoming hyperreactive. Postviral hyperresponsiveness and hypersecretion in the lower respiratory tract is well recognized (Empey et al, 1976).

Clement, Stoap and Kaufman (1985) described nasal hyperreactivity as a condition in which there is an overreactivity of nasal mucosa to stimuli of non-specific endogenous (physical stress, mechanical irritation, endocrine stimulation or poor venous return) or exogenous (thermal or mechanical stimulation, humidity or drug induced) origin. Results of histamine challenges in a group of patients with non-allergic perennial rhinitis were compared
with those obtained in a group of normal controls. They found a slight but significant difference in the two groups, although with considerable overlap.

**Nasobronchial reflex**

Recently, nasal-bronchial interactions and the relationships between innervation and reactivity in nose and lower respiratory tract airways have been the subject of much discussion (the subject of 'Cellular and Neurogenic Mechanisms in Nose and Bronchi', supplement no 128 to the *European Journal of Respiratory Diseases*, 1983, edited by Mygind, Rasmussen and Mølgaard). In particular, possible reasons for increased nasal reactivity in the common cold, allergic rhinitis and perennial non-allergic rhinitis have been suggested by Borum et al (1983) to be an increased number of mediator cells, increased sensitivity of epithelial nerve receptors ('irritant receptors'), increased reactivity of individual gland cells and increase in number of secretory cells. The consensus opinion at present is that a nasobronchial reflex probably does exist in man, although evidence for this is stronger in animals, and that nasal stimulation may cause not only reflex bronchoconstriction, but also mucus secretion in susceptible subjects, although the exact mechanism in relation to type of nasal stimulation is obscure.

**Neuropeptides**

The respiratory tract possesses neuropeptidergic innervation mediated by the release of such peptides as vasointestinal peptide (VIP), substance P (SP), pancreatic polypeptides (PP) and others.

The Dale principle of one autonomic neuron producing and releasing only one transmitter has been challenged in recent years by the immunohistochemical demonstration of many peptides with powerful biological action, localized to nerves both in the peripheral and central nervous system, which may be present together with classical transmitters in the same neuron (Hökfelt et al, 1980). Autonomic nerve fibres are richly distributed in the nasal mucous membrane and stimulation of these fibres results in major changes in blood circulation and secretions in this lining. These changes are influenced by reflexes such as those stimulated by nasal irritant receptors and their effects have been described above. Studies of the nasal autonomic innervation in cat, rat and man have shown substance P, vasointestinal polypeptide and pancreatic polypeptide to be present in classical pathways. Substance P is found principally in sensory neurons ending in the spinal trigeminal nucleus, around sphenopalatine ganglion cells, around blood vessels and within the nasal epithelium; vasointestinal polypeptide is found in parasympathetic postganglionic cholinergic neurons innervating blood vessels and glands; pancreatic polypeptide is localized to mucosal artery, innervating noradrenergic ganglion cells in the superior sympathetic ganglia.

The nasal mucosa contains peripheral branches of capsaicin-sensitive, substance P-immunoreactive trigeminal neurons around its vascular supply and within itself. This forms a local axon reflex arc arrangement which can induce local vasodilatation and also increased vascular permeability.

Afferent sensory substance P-containing neurons may activate the efferent parasympathetic neurons, which are vasointestinal polypeptidergic and cholinergic, via central
and/or peripheral mechanisms causing subsequent increased vasodilatory effects and secretion. Vasointestinal polypeptide and pancreatic polypeptide adrenergic nerves are juxtaposed around nasal mucosal arteries so the known inhibitory effect of pancreatic polypeptide on this vasointestinal polypeptide-induced vasodilatation may be a physiological control mechanism (Anggard, Lundberg and Lundblad, 1983).

This complex system may account for a number of conditions seen in the upper and lower respiratory tracts whose pathogenetic mechanisms are obscure. It is possible that increasing understanding of this area will lead to an explanation of, and treatment for, several troublesome conditions affecting the nose and paranasal sinuses (for example non-allergic perennial rhinitis; Kurian et al, 1983).

**Hormonal**

There is considerable clinical and experimental evidence to indicate that both male and female sex hormones affect the nasal mucosa. Rhinitis is often associated with puberty, sexual excitement ('honeymoon rhinitis'), menstruation and pregnancy (Eccles, 1982).

Rhinitis during pregnancy not uncommonly presents a problem to otolaryngologists who may question the desirability of undertaking investigations such as X-rays at this time. In addition, medication requires careful consideration as the basis of this (certain antibiotics and corticosteroids) may be contraindicated and most practitioners would prefer to postpone any surgical intervention requiring general anaesthesia until after delivery, particularly as the nasal disorder frequently resolves spontaneously at that time. Sorri, Hartikainen-Sorri and Karja (1980), however observed that sinus infection was often associated with this rhinitis. Of approximately 2000 pregnancies, 47 patients were referred with nasal symptoms of which 30 were found to have sinus infection confirmed by antral lavage. It was suggested that this relatively high figure of infection might be related to changes in the immunological status and the high rate of viral infection associated with pregnancy (Vesikari, 1975). However, there is another possibility, that the nasal congestion and inflammation lead to mechanical obstruction and interfere with normal nasal mucociliary clearance and other local defence mechanisms, leading to sinus infection in much the same way as may nasal polyps.

In addition to pregnancy, the occurrence of rhinitis has been reported in women taking oral contraceptives high in oestrogen. Toppozada et al (1984) studied the ultrastructure and histochemical changes in the nasal mucosa of 25 females using the contraceptive pill. Although 15 patients developed no nasal symptoms, changes were found in all 25 similar to those in symptom-free pregnant females - glandular hyperactivity, increased acid mucopolysaccharide content of the ground substance and increased phagocytic activity - while the 10 patients who developed nasal symptoms showed evidence of squamous metaplasia, intraepithelial oedema, glandular hyperplasia, histiocytic proliferation and deposition of fibrous tissue. The histochemical reactions were similar to those of chronic hypertrophic non-allergic rhinitis.

Harrison (1957), in a study of patients suffering from familial haemorrhagic telangiectasia, showed that prolonged oestrogen therapy in men produces metaplasia of the columnar ciliated nasal epithelium to stratified squamous epithelium. Atrophic rhinitis is discussed in Chapter 8, but it is interesting to note that it is more common in females and it
has been one author's clinical experience that it is more common following the menopause. Some patients improve when treated with either systemic hormone replacement therapy or topical hormone creams (conjugated oestrogens - equine). Oestradiol in arachic oil 10,000 units/mL has also been recommended (Weir, 1979).

While atrophic rhinitis appears to be more common in females, excessive watery rhinorrhoea has been noted in elderly males. Watson-Williams (1952) described a condition called 'old man's drop': seen in men usually well over 60 years of age with profuse watery discharge tendency to hang as a drop at the end of the nostril until wiped away. He treated 23 patients with testosterone propionate tablets (placed beneath the tongue, not sucked) 5 mg daily for 3 weeks and reported that ‘17 patients were completely relieved’. Eight patients were treated with Stovaine lozenges (amylocaine hydrochloride) having a similar appearance and taste and 'all were quite definite that these were ineffectual'. He also added that the course may need to be repeated once or twice a year.

Thyroid hormones are also known to affect the nasal mucosa, myxoedema being associated with large, boggy, pale turbinates similar to those seen in perennial non-allergic rhinitis and biopsy revealing intense oedema (Connell, 1984).

**Iatrogenic**

**Rhinitis medicamentosa**

The aetiology of this form of rhinitis is the abuse, by overuse, of topical nasal decongestants. The patient, for whatever reason, whether it be a transitory upper respiratory tract infection or a long-standing perennial rhinitis, uses a topically acting vasoconstrictor which results in an immediate improvement in symptoms. In the short term, this causes no problem but if the patient should continue to use this, it is possible for a vicious circle to ensue, whereby decongestion is followed by rebound vasodilatation such that ever increasing doses of vasoconstriction are required more and more frequently to ensure an adequate airway.

Ideally, a topical vasoconstrictor for clinical use should act on the cavernous sinusoids to reduce the swelling of the erectile tissue. Naumann (1961), however, demonstrated that decongestants constrict resistance vessels in addition to the exchange and capacitance vessels, resulting in local ischaemia. Mygind (1979) described the local side-effects of long-term use of topical vasoconstriction under three headings:

1. Secondary hyperaemia (rebound) which commences a few hours after administration, more marked with adrenaline than ephedrine and possible due to beta-adrenergic stimulation as both alpha and beta-receptors are stimulated by these substances.

2. Tachyphylaxis - a phenomenon whereby ever increasing quantities of a drug are required to elicit a given response - in this situation, possibly due to lowered alpha-adrenergic responsiveness of smooth muscle.

3. Local irritation - increased irritability associated with a sensation of burning and dryness in the nose.
Rijntjes (1982) carried out rhinomanometry and both light- and electron-microscopic examination of biopsies from 20 patients who had used topical vasoconstriction at least once daily for a minimum of 6 months. Rhinomanometry revealed an improved nasal airway in 75% of patients 4-6 months after discontinuing the use of decongestants. It was interesting to note that the normal nasal cycle (alternating patency and obstruction from one side to the other every few hours - see below) was not found in patients with rhinitis medicamentosa and this cycle did not appear to return, even 6 months after discontinuing usage. Scanning-electron microscopy and light microscopy revealed a decrease in the quantity and quality of cilia with metaplasia of the mucous membranes to cuboidal cells. The ultrastructure of the remaining cilia remained unaltered.

Some decongestants appear to be more related to rhinitis medicamentosa than others. Naphazoline caused a marked vasoconstriction of long duration and appears to be incriminated more often than the more recently introduced vasoconstrictors such as xylometazoline and oxymetazoline. Adrenaline has a rapid action of short duration followed by considerable rebound. The action of ephedrine is less intense but longer-acting than adrenaline.

**Aspirin intolerance**

Aspirin intolerance may occasionally present with chronic rhinitis (Settipane, 1984b). More often, however, it accompanies nasal polyposis and asthma. Samter and Beers (1968) described the initial presentation as a profuse watery rhinorrhoea developing after aspirin ingestion in the majority of patients during their second or third decade. Initially intermittent, it later becomes perennial, frequently followed by nasal polyps and hyperplastic sinusitis. Urticaria is also common and middle-aged patients often develop bronchial asthma.

The incidence of nasal polyps in asthmatic patients with aspirin intolerance is as high as 36% (Chafee and Settipane, 1974). Frequently, patients in this group are found to be skin test negative but to have eosinophilia in the blood or nasal secretions.

The mechanisms involved in aspirin intolerance are not known, but it is possible that inhibition of the cyclo-oxygenase pathway by aspirin may result in preferential lipo-oxygenase metabolism leading to increased production of leukotrienes and slow reacting substance of anaphylaxis (SRS-A) causing bronchospasm. A similar mechanism involving the arachidonic acid pathway and prostaglandins may be responsible for the production of nasal polyps.

**Drugs acting on the sympathetic nervous system**

Alpha-adrenergic blocking agents used in the treatment of hypertension, such as guanethidine and bretylium tosylate, may result in vasodilatation and nasal obstruction, as may methyldopa and reserpine which deplete sympathetic nerve endings of their catecholamine stores (Ariens, 1967).

There is some evidence that beta-agonists such as isoprenaline cause vasodilatation and nasal obstruction (Hall and Jackson, 1968; Malm, 1974) and that beta-antagonists may inhibit the vasodilatation induced by beta-agonists, suggesting that the nasal beta-receptors are of the beta2 type (Malm, 1974). The clinical effect on the nose of beta-blockers in man, however, remains uncertain.
Nasal stuffiness may also result from drugs used to cause peripheral vasodilatation in the treatment of migraine and peripheral vascular disease, such as the ergot alkaloids, for example dihydroergotamine mesylate.

**Anticholinesterases**

Drugs which inhibit cholinesterase and potentiate the action of acetylcholine, such as neostigmine used in the treatment of myasthenia gravis, may produce nasal obstruction.

**Surgery**

Overzealous surgery may result in atrophic rhinitis. It has long been thought that excessive removal of the inferior turbinates may result in atrophic rhinitis, although recent studies suggest that this may not be the case (Martinez et al, 1983; Ophir, Shapira and Marshak, 1985).

The cause of primary atrophic rhinitis is not known, although numerous organisms have been incriminated: coccobacilli (Lowenberg, 1894), *Bacillus mucosus* (Abel, 1895), *Coccobacillus foetidus ozaenae*, diphtheroid bacilli and *Klebsiella ozaenae* (Henriksen and Gundersen, 1959). In addition to infective organisms, the possibility of hormonal influences have been discussed above.

Whether the cause be infective, hormonal (as described above) or simply degenerative with age, patients frequently complain of nasal obstruction despite the fact that on examination, the nose is patent - indeed in most cases overpatent - as can be demonstrated by rhinomanometry. Surgical implantation of cartilage partially to obstruct the airway has been successful in some cases, although it is not known why the nose should feel blocked when the airway is patent and the symptoms relieved when the airway is partially obstructed (Connell, 1984).

One possible explanation for the observation that some patients may develop atrophic changes following surgery on the inferior turbinates while others appear not to, is that in certain instances, primary atrophic rhinitis may present with the symptom of nasal blockage and the patient undergo reduction of the turbinates resulting in aggravation of the symptoms. This is one reason why preoperative rhinomanometry should be carried out whenever this condition is suspected.

Septal perforations following submucosal resection of the nasal septum or other trauma such as 'nose picking' may be associated with crusting and bleeding, the latter leading to further crusts sometimes associated with purulent rhinitis. It is important to exclude other possible causes such as malignant tumours, granulomata, chronic infections and exposure to noxious fumes or cocaine.

**Clinical features**

Examination of the nose has been considered in Chapter 1 and endoscopy in Chapter 3. These techniques are therefore not considered here. Suffice it to say that in all cases of rhinitis, the nose must be carefully examined and this may require topical vasoconstriction.
If this is necessary, however, it should be postponed until after nasal mucociliary clearance tests and rhinomanometry have been performed if it is intended that these should be done at the same visit.

**Acute sinusitis**

Externally, there may be slight redness and swelling of the cheek spreading to the lower eyelid from the antrum and the upper eyelid from the frontal sinus. In children, ethmoiditis may be associated with marked swelling and abscess formation medial to and above the inner canthus and swelling of the eyelids.

There may be tenderness over the frontal sinus, although this may be confused with tenderness over the supraorbital nerve which can accompany maxillary sinus infection.

Tenderness to pressure over the floor of the frontal sinus immediately above the inner canthus is usually diagnostic of frontal sinusitis and tapping the supraorbital ridge may cause severe pain. This is, however, highly subjective and many patients will complain of pain in these regions if sufficient pressure is exerted even over a normal sinus. On anterior rhinoscopy, there will be generalized swelling and hyperaemia of the nasal mucous membrane. After decongesting the mucosa, it may be possible to see pus extruding into the middle meatus from the maxillary, anterior ethmoidal and frontal sinuses or into the superior meatus from the sphenoid and posterior ethmoidal sinuses.

On posterior rhinoscopy, pus may be seen in the postnasal space and there will be generalized swelling and redness of the mucous membranes.

**Mechanical obstruction**

Bilateral choanal atresia will present at birth because neonates are obligatory nasal breathers. This presents as a medical emergency and, unless an airway is inserted through the mouth, the child will asphyxiate.

Unilateral choanal atresia is less dramatic, however, and a careful examination needs to be undertaken in any case of total unilateral nasal obstruction presenting early in life. The easiest way to start the examination is by placing a cool stainless steel tongue depressor immediately below the child's nostril. Warm air exhaled through the nose will cause misting on the spatula. If this occurs on both sides, choanal atresia is excluded. If one side is totally blocked, misting will only occur on the opposite side and choanal atresia along with other causes of nasal obstruction will need to be considered.

Anterior rhinoscopy may reveal other causes of nasal obstruction and it is important to exclude the possibility of a foreign body. Where the index of suspicion is high, examination of the nose and postnasal space with a microscope under general anaesthesia may be necessary particularly in small infants who may not be entirely cooperative.

In any patient with nasal airways obstruction, examination should always start with the external appearance of the nose. No amount of surgery on the septum will improve the airway if the problem is due to external deviation of the nose, even if the patient is not
concerned with his appearance. In assessing the septum, the degree of deviation as well as the site of the deviation is important. Cottle (1960) has named five areas of the septum:

- area 1 is the anterior caudal border where the septum may deviate to one side or other of the columella
- area 2 is the region of the valve
- area 3 is the superior mid-portion of the septum
- area 4 is the inferior mid-portion
- area 5 is the remaining posterior region.

This nomenclature has nothing to do with the anatomy of the nose but is important from a functional point of view. Deviation of a small degree in area 2 will be critical, deviation in area 3 may be associated with external deviation of the nose, whereas a slight degree of deviation in areas 4 or 5 may be less relevant. In assessing the site and degree of deviation, one must also take note of any associated pathology, that is, is there any evidence of vestibulitis due to interference with the normal smooth laminar air flow along the sides of the septum? Are there any polyps posterior to a deviation? Many patients will have a deviated septum without symptoms and rhinomanometry may be helpful in deciding how much the patient's symptoms may or may not be associated with the septal deformity.

Mechanical obstruction may also result from hypertrophy of the turbinates (both middle and inferior), from nasal polyps, from tumours - both benign and malignant - and from adhesions following trauma or previous surgery.

Alar collapse is another cause of inspiratory nasal obstruction. Nearly all patients can cause indrawing of the lower lateral cartilages if they breathe in fast enough and this should only cause symptoms if it occurs at physiological flow rates. If one asks the patient to tilt the head backwards a little, the nostrils can be examined while the patient breathes in and out through the nose. Alar collapse may occur in the elderly due to lack of spring in the lower lateral nasal cartilages associated with loss of tone of the facial muscles, in particular the dilator nares. It may occur following rhinoplasty or in association with other problems, such as deviation of the septum in the valve area or a wide columella. This is considered in further detail in Chapter 15.

**Allergy**

Allergy

There may be redness and excoriation of the skin of the nose following excessive nose blowing and wiping. The eyes may be red and watery in the typical seasonal rhinoconjunctivitis due to pollen allergy. Concomitant coughing and wheezing characteristic of asthma may be present.

On anterior rhinoscopy, the nasal mucous membranes are usually pale, 'boggy', hypertrophic and wet. It can at times be difficult to differentiate between hypertrophic, pale, swollen, 'polypoidal' turbinates and polyps; although the latter are insensitive when lightly probed and can be displaced, while the turbinates remain fixed.
Posterior rhinoscopy will reveal clear watery secretion and hypertrophy of the posterior ends of the inferior turbinates which, at times, can be bluish in colour and lobulated, thus referred to as mulberry turbinates.

**Abnormalities of mucociliary clearance**

Examination of the nose in patients with primary ciliary dyskinesia usually reveals mucopurulent secretions on the floor of the nasal cavity and in the postnasal space. Polyps are seldom seen. The ears should be carefully examined as a high proportion of these patients will be found to have glue ear (Greenstone et al, 1985).

Patients with Young's syndrome (obstructiveazoospermia, bronchiectasis and sinusitis associated with abnormally viscous mucus and slow mucociliary clearance despite normal cilia) tend to have a similar clinical picture to those with primary ciliary dyskinesia, although it is interesting that in both groups, the nasal symptoms seldom cause more than mild inconvenience to the patient, and in primary ciliary dyskinesia have been present since birth.

**Immunity deficiency**

Patients with systemic immunity deficiency often present with the signs of (recurrent) acute or acute on chronic infection with mucopurulent rhinorrhoea, hyperaemic and swollen mucous membranes and purulent postnasal drip. Infection may also be present at other sites such as ears, chest and skin.

Acquired immune deficiency syndrome (AIDS) may present with otolaryngological symptoms. There are frequently signs of rhinitis with ‘granular’ mucosa and purulent discharge not unlike the appearance of sarcoid although with less bleeding and crusting. Patients almost invariably develop oral candidiasis and often otitis externa with occasional otitis media. Kaposi’s sarcoma skin lesions can develop at any site but should be looked for in the mouth as well.

**Granulomatous conditions**

On examination, there is frequently severe crusting and blood clot. Granulation tissue may be obvious, although usually one needs gently to lift away the crusts of dried secretions to reveal the underlying granular mucous membranes. Wegener's granuloma is often associated with a musky, unpleasant odour. Wegener's and non-healing granulomata are dealt with in Chapter 18 and the other chronic inflammations in Chapter 8. Millet seed granulomata of sarcoidosis may be occasionally seen in the conjunctival sac.

**Autonomic imbalance**

As one would expect in an ill-defined group such as this, the appearances on examination are variable.

The mucosa may be moist, pale, bluish and swollen, similar to that seen in allergic rhinitis. The main complaint in this group is often rhinorrhea - 'the runners' - while in the other group - 'the blockers' - the mucosa may be swollen but otherwise relatively normal.
**Hormonal**

In puberty and pregnancy, the nasal mucosa appears swollen and often hyperaemic, although the latter may be related to abuse with topical vasoconstrictors. In both these groups, the primary symptom is nasal blockage and patients have often resorted to proprietary topical decongestants by the time they seek specialist advice. One should take particular care to exclude secondary infection in rhinitis in pregnancy as almost one half of these patients presenting with symptoms suggesting rhinitis may be found to have sinus infection (Sorri, Hartikainen-Sorri and Karja, 1980). The watery rhinorrhoea seen in elderly men may originate from the anterior serous glands as there may be little evidence of rhinitis affecting the mucous membranes generally. Indeed, in many cases, the mucosa may look if anything a little atrophic rather than hypertrophic.

Atrophic rhinitis which is more common in women and may be oestrogen related, presents with a foul-smelling discharge (ozaena). On examination, the mucosa appears thin and atrophic and the nasal cavities are full of hard crust which often yellow. When these are removed there is frequently an underlying green mucopurulent discharge. The mucosa is friable and bleeds easily. Attempts made by the patient to remove these crusts often cause epistaxis resulting in blood clots collecting in the nasal cavities which then act as foreign bodies, causing further mucopurulent discharge.

**Iatrogenic**

On anterior rhinoscopy, the mucous membranes in rhinitis medicamentosa appear swollen and red. The bright red swollen mucosa which was commonly seen with abuse of naphazoline (Privine) led to this condition being referred to as 'Privine nose'. This is less of a problem with xylometazoline or oxymetazoline. It is perhaps wrong to assume that all patients developing hypertrophy of the mucosa following decongestion do so because of the effect of the drug, for as Connell (1984) pointed out, most patients who abuse topical nasal drugs started to use them because they already had a nasal problem.

This is not true, however, for those patients who become addicted to cocaine and who are unlikely to have had any nasal symptoms prior to their 'snorting'. Cocaine interferes with the uptake of noradrenaline into nerve endings potentiating the action of adrenaline (Eccles and MacClean, 1977).

Short-term abuse with cocaine leads to rebound and a picture similar to abuse with alpha-adrenergic agonists; long-term abuse however leads to severe necrosis due to ischaemia with loss of mucosal lining and underlying supportive structures. There is often a perforation of the nasal septum with bleeding, crusting and foul-smelling discharge. In severe cases, the destruction may lead to gross distortion and external disfigurement of the nose.

Excessive surgical reduction of the inferior turbinates may cause an overpatent nasal airway with severe crusting and underlying mucopurulent discharge (Martinez et al, 1983; Ophir, Shapira and Marshak, 1985).
Investigations

Sinus X-ray

The technique and overall interpretation of radiology of the nose and paranasal sinuses is of such importance that a separate chapter is devoted to this (Chapter 20. It is important, however, to stress here that all patients with nasal symptoms, particularly if these are of long standing, should undergo sinus X-rays and chest X-rays, the former to exclude any possible bony erosion or other radiological changes suggesting the possibility of underlying sinister pathology and the latter to detect signs of bronchiectasis, Wegener's granulomatosis, dextrocardia or some other related chest disease.

Three standard views of the sinuses are normally requested: occipitomental, occipitofrontal and lateral.

Sinus X-rays may reveal no abnormality, mucosal thickening, fluid levels (in which case, a tilted view will be helpful) or total opacity. However, it is important to remember that these X-rays are not infallible. The shadow of the lip may sometimes appear as mucosal thickening, totally clear X-rays will occasionally be taken of sinuses found to contain pus on wash-out and a rate of 9% false negatives (normal X-rays with positive return) was reported by Phleiderer, Drake-Lee and Lowe (1984). The same authors also reported a very high false negative rate with no return on wash-out in 50% of the sinuses reported as showing a fluid level and 47% of those reported as opaque, but only 12% of those sinuses reported as 'mucosal thickening' gave any return, while other authors reported results ranging from 16% (Hinde, 1950) to 60% (Axelsson et al, 1970) in this latter group.

An opaque antrum on X-ray may on occasion be due to thickening of the bony wall (Proops, 1983), while previous Caldwell-Luc surgery results in scarring of the lining which, in most cases, will lead to great difficulty in interpreting the radiological appearance.

Sinus X-rays will frequently be requested by practitioners other than otolaryngologists and a report of mucosal thickening of the maxillary antra is then usually interpreted as 'sinusitis'. It is important to appreciate that the lining of the sinuses and nose is continuous and that many conditions producing swelling of the nasal mucosa will be associated with mucosal swelling in the antra without it necessarily being attributed to 'infection'. Hayfever, for example, with profuse watery rhinorrhoea may even be associated with fluid levels in the sinuses at times.

All this does not mean however that X-rays of the sinuses are of no value, far from it. All patients with nasal symptoms should be referred for radiological assessment and this at times will result in unexpected information, such as bony erosion at the base of the skull, suggesting a postnasal space carcinoma, or opacity of the antrum with bony erosion from tumours of the sinuses presenting with symptoms of rhinosinusitis, although bony changes may be seen even with benign nasal polyps (Lund and Lloyd, 1983).
**Transillumination of the sinuses**

A small light is used, covered with a suitable glass cover which can be removed for disinfection. The patient is placed in a darkened room and the light placed centrally in the mouth with the lips pursed around the cover. Any dental plate must be removed prior to the examination. The antra will transilluminate causing an infraorbital crescent of light and glowing pupil. This will be equal on both sides, providing the antra are normal and equal in size. Mucosal thickening, pus or other pathology will prevent normal transillumination. This may, however, be difficult to interpret if both sides are involved. McNeill (1963) found a positive correlation between transillumination and antral lavage in 68% of cases. It is interesting to note, however, that he found this to be 15% less accurate than X-ray examination and that even here, in the antra of three cases out of 25 reported as entirely normal radiologically, pus was found at lavage. Ballantyne and Rowe (1949) reviewed 100 cases and found transillumination was often misleading in the diagnosis.

One instance in which it can be helpful, however, is a large cyst in the maxillary antrum. Here, an X-ray will reveal an opaque antrum but transillumination is brilliantly clear. Transillumination of the frontal sinuses is even more difficult to interpret as these sinuses are so frequently of unequal size.

**Ultrasound**

A transmitter emits soundwaves of high power and short duration which are reflected back to a receiver from the interface between objects of varying acoustic impedance. A probe, coated with electrode gel, is moved over the antrum and the 'echo' of the ultrasonic wave is recorded on an oscilloscope which can be photographed with a polaroid camera to produce a permanent record.

In an air-filled sinus, most of the ultrasound will be reflected from the interface between the anterior wall of the sinus and the air filling that sinus, leading to an early peak followed by a flat graph. Variations in this pattern will occur with fluid filling the sinus which will produce a 'back wall echo' - a double peak will suggest a cyst and multiple peaks may be seen with mucosal thickening.

Böckmann et al (1982), in a comparison of radiology with ultrasound investigation in diagnosing maxillary sinus pathology, showed good correlation in 95%. Pfleiderer, Drake-Lee and Lowe (1984), in their study of 108 washouts, however, concluded that ultrasound adds little.

**Swabs and antral lavage**

Gwaltney and Hayden (1982) reviewed 12 studies reporting the frequency of bacteria cultured from the vestibule of the nose of normal individuals and found 40-100% incidence of *Staphylococcus epidermidis* and *Micrococcus*, 25-40% *Staph aureus*, 90-100% diphtheroids and 1% Gram-negative bacteria. From the posterior nares, *Streptococcus pneumoniae* was isolated in 15-25%, *Haemophilus influenzae* in 6-40%, *Strep pyogenes* in 6%, *Staph aureus* in 12%, *Neisseria meningitidis* in 4-27% and Gram-negative bacteria in 13%. If many of these
are regarded as normal commensal flora, one is left in serious doubt as to the usefulness of nasal swab for bacteriology in the treatment of infective rhinitis.

The common cold and other respiratory virus syndromes are caused by viruses such as rhinoviruses, coronavirus, parainfluenza viruses and these may in turn lead to sinusitis. More commonly, however, they lead to swelling of the lining of the nose and sinuses, blockage of the ostia draining the sinuses (particularly the ostia of the maxillary sinus), interference with the clearance mechanism and stasis. These factors favour secondary infection by the multitude of bacteria already present. With chronic infection, the sinus ostia may well become totally blocked leading to a negative pressure and low oxygen concentration; this, combined with an impaired blood supply to the nasal mucosa, may explain the high frequency of anaerobic organisms found, which, in one study (Frederich and Braude, 1974), was as high as 43 out of 62 cases in which bacteria were isolated.

In acute sinusitis, the results of nasal cultures correlated poorly with those obtained by proof puncture (Axelsson and Brorson, 1973). The latter are summarized in Table 6.3 (Gwaltney, 1979).

Table 6.3 Organisms isolated from nasal cultures from patients with acute sinusitis

<table>
<thead>
<tr>
<th>Cases</th>
<th>Microbes</th>
<th>Mean(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bacteria</td>
<td><em>Strep pneumoniae</em></td>
<td>31</td>
</tr>
<tr>
<td></td>
<td><em>H. influenzae</em></td>
<td>21</td>
</tr>
<tr>
<td></td>
<td><em>Strep pneumonia</em> +</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td><em>H. influenzae</em></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Anaerobic bacteria</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td><em>Staph aureus</em></td>
<td>4</td>
</tr>
<tr>
<td></td>
<td><em>Staph pyogenes</em></td>
<td>2</td>
</tr>
<tr>
<td></td>
<td><em>N catarrhalis</em></td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Gram-negative rods</td>
<td>9</td>
</tr>
<tr>
<td>Viruses</td>
<td><em>Rhinoviruses</em></td>
<td>15</td>
</tr>
<tr>
<td></td>
<td><em>Influenza virus</em></td>
<td>5</td>
</tr>
<tr>
<td></td>
<td><em>Parainfluenza virus</em></td>
<td>3</td>
</tr>
</tbody>
</table>

In patients with cystic fibrosis and other forms of severe chronic sinusitis and bronchiectasis, *Pseudomonas* is commonly found.

Fungal infection is relatively rare by comparison with viral and bacterial infection. It is known to occur in cases of trauma to the face, poorly controlled diabetes, severely debilitated patients such as those with carcinomatosis and patients who have been treated with immunosuppressive drugs (Berlinger, 1985). It may, however, occur in otherwise healthy patients (Meikle, Yarington and Winterbauer, 1985). The commonest fungus involved is *Aspergillus*.
In many ways, it is surprising that fungal infection is not seen more often, particularly in view of the large number of patients now treated with topical corticosteroids. In the oropharynx and laryngopharynx, however, candidiasis may be seen in 10% of cases treated for asthma with topical beclomethasone.

**Nasal smears**

Examination of nasal smears for eosinophils was first reported by Eyermann in 1927. Since then, many authors have reported its usefulness in distinguishing between 'allergic' and 'infective' rhinitis. Mygind (1979) gave an excellent account of the technique and its interpretation but commented that 'this simple technique ... has not been accorded the general use it deserves'. This may be because it is not quite so simple.

First, a nasal smear is taken by scraping the mucous membranes with a cotton applicator; this should be taken from as far posteriorly as practical and as much secretion as possible should be collected. This is smeared on a glass slide and dried in air. It is then covered with 18 drops of May-Grunwald stain and, after 30-45 seconds, 6 drops of Giemsa stain are added and left for 30-45 seconds, after which the smear is placed under running tapwater. It is then quickly decoloured with alcohol before placing it once again under running tapwater. The slide is then ready for microscopy. Marked eosinophilia may be found in allergic seasonal and perennial rhinitis, but may also be found in cases of non-allergic eosinophilic rhinitis. It is useful in distinguishing those patients in whom yellow or greenish secretions are due to a very high count of eosinophils rather than infection and in whom treatment with antibiotics would be unhelpful.

**Biopsy of nasal mucosa**

This should not be undertaken as a routine investigation. Nasal cilia can be harvested by the brushing technique described below which requires no anaesthesia and is without complications. By contrast, taking even the smallest biopsy from the turbinates can occasionally be followed by very troublesome epistaxis requiring packing and even transfusion.

Under local anaesthesia in the outpatient or 'office' setting, it is impractical to take more than a fairly small biopsy and this is often done with aural cupped granulation forceps. The tiny specimen taken, while putting the patient at possible risk of severe epistaxis, is, however, seldom sufficient for adequate histological examination.

If a biopsy is required to exclude Wegener's granuloma, sarcoidosis or a possible malignancy, it is better to perform this as a formal procedure in a well-equipped operating theatre with topical anaesthesia and vasoconstrictors together with locally injected anaesthetic agents. A reasonable-sized biopsy can then be taken with turbinate trimming scissors and the nose packed with an adequate pack which is left in situ for 24-48 hours.

**Prick skin tests for immediate hypersensitivity**

Skin tests may be performed either by placing a drop of a solution of the extract to be tested on the skin and gently pricking the epidermis with a lifting action, or by
intracutaneous injection of a much more dilute solution of the extract. The prick test is preferred, however, because it is not painful, easier to interpret, much safer, the solutions are more stable and the procedure is quick and easy to perform. The allergen extracts used are solutions in glycerin of the common allergens such as *Aspergillus fumigatus*, *Alternaria*, grass pollen, cat fur, dog hair, feathers, house dust, *Dermatophagoides pteronyssinus* (house dust mite), milk and egg. After placing a drop of the solution on the skin, the superficial epidermis is very gently pricked with a sterile disposable needle or lancet, using a slight lifting motion and without drawing blood. In allergic patients, a weal and flare develops within 10-15 minutes (immediate); less commonly, late reactions (6 hours) may develop.

In addition to the allergen extracts, a positive histamine control (1 mg/mL) and a negative control (allergen-free stock solution) should be used - the histamine in order to ensure there is no factor interfering with a positive result (for example antihistamine therapy), the negative control in case the patient wheals to trauma alone (for example dermatographism).

If the positive control is negative, it is important to repeat the tests after ensuring that the patient abstains from antihistamines for 48 hours. Some of the recently introduced H₁-antagonists such as astemizole, may abolish positive skin tests for up to one month. Systemic corticosteroids however do not affect the result.

**Rhinomanometry**

The importance of this measurement is such that an entire chapter (Chapter 4) has been devoted to it. Undoubtedly, it will form an increasingly important part of the investigation of all patients with nasal symptoms.

However, there are many problems associated with this investigation, not the least being that one is measuring a 'moving target'. As the subject breathes in, the nasal apparatus flattens and the faster the air flows inwards, the more the alar cartilages and upper lateral cartilages are drawn inwards, increasing the nasal resistance. Breathing outwards through the nose, the opposite will happen, causing a decrease in nasal resistance. The nose is designed as an air-conditioning mechanism to warm and humidify as well as to purify the air before it passes to the lungs. As such, it is a very effective organ and can adapt quickly to changes in the environment: in order to warm cold air, more blood passes through the turbinates and vice versa - the environment therefore affects the results of any rhinomanometry; added to this, exercise and posture also alter resistance. Finally, even in the same subject resting and without alteration in posture in a stable environment, the resistance will change: alternating from side to side every 3-4 hours due to the 'nasal cycle'.

Anterior and posterior, and both active and passive forms of rhinomanometry are described. From a clinical point of view, the passive method is of little value as it decreases the resistance of the nose as air passes inwards by 'blowing out' the lower lateral and upper lateral nasal cartilage. This is the opposite effect to that which occurs in a physiological situation and would be of no value in investigating, for example, a possible alar collapse.

The posterior method, which has some advantages, in that it may avoid the necessity of applying measuring device to the anterior part of the nose, is regretta
perform in 25% of subjects who are unable to relax the soft palate sufficiently (Mackay, 1979). The anterior active method is the method chosen for routine clinical use and suitable equipment is now commercially available.

In 1983, a committee was set up to report on standardization of rhinomanometry (Clement, 1984). At this meeting, it was decided that the method of choice was the active anterior method, that is 'the measurement of nasal air flow and pressure at the nostrils during respiration'. That pressure is recorded via a tube fixed with adhesive tape to one nostril and flow is measured with a pneumotach via a face mask and recorded during quiet breathing in a sitting position. Values are expressed in SI units, pressure in pascals (Pa) and flow in cm³/s, expressing the resistance at a fixed pressure of 150 Pa.

Rhinomanometry is undertaken both before and after decongesting the nose to reduce the effect of the nasal cycle and indicate both the 'reversibility' of any obstruction (see spirometry pre- and post-bronchodilatation in assessing the reversibility of lower respiratory air flow obstruction for example in asthma) and the relative importance of any septal (or nasal) deviation.

Rhinomanometry has three valuable roles.

Research

Undoubtedly, this has so far been its most important function. It remains the only method by which one can objectively assess and compare the results of different medical treatment regimens (for example comparison of two corticosteroid sprays in the treatment of hayfever) or surgical techniques (submucosal diathermy versus trimming of the inferior turbinates).

Reassurance

Subjective assessment of nasal patency is often paradoxical. In very cold weather, the nasal turbinates will be hugely dilated with blood, attempting to warm the inspired air - the resistance will increase: despite this, the nose may feel clear. This phenomenon was well illustrated by Eccles and Jones (1983) who demonstrated no change in nasal patency when applying menthol to the nose, despite the fact that this was interpreted as a far better airway by the subject, possibly due to the stimulation of the cold receptors in the nose. The consequence of this is that many patients who in fact have a 'normal' airway may complain of nasal obstruction. Here, the importance of rhinomanometry cannot be overstressed, as to treat these patients for nasal obstruction will never be rewarding and may indeed worsen the situation - particularly if the patient's sensation of obstruction is due to relative insensitivity of the nasal mucosa - leading to lack of appreciation of the air flow or stimulation of cold receptors of the nose. To undertake radical or even partial trimming of the turbinates in rhinitis sicca will only aggravate the problem and even topical corticosteroids may have a deleterious effect in this situation.

Reassurance and explanation both for the patient and his referring doctor will be more helpful and less harmful; rhinomanometry plays an essential role in this.
Reassessment

When rhinomanometry is undertaken as routine in the investigation of all patients with nasal symptoms, decongesting the nose at the time of initial examination will obviously invalidate the test. This means that an adequate view may not be obtained until after decongesting the nose for rhinomanometry. Patients should therefore be re-examined at this stage. If the added information available from the nasal airway studies does not at first appear to correlate with examination findings, further examination may reveal an obstruction posteriorly which might otherwise have been missed; rhinomanometry in these circumstances acts as a useful 'double-check'.

Sinuscopy

Endoscopy of the nose and paranasal sinuses is dealt with in Chapter 3. It remains the only certain way of investigating the contents and appearance of the lining of the paranasal sinuses, as even a sinus wash-out may mislead by giving a clear return, despite the fact that there may be inspissated mucopus or crumbly purulent masses present, as in sinusitis caseosa. Draf (1983), reviewing over 1000 cases in which sinuscopy of the paranasal sinuses was undertaken, concluded that in 20% of cases, it was not possible to ascertain the presence of suppurative maxillary sinusitis by means of X-rays and proof puncture alone. Comparative endoscopic examination immediately after standard irrigation of the antrum without visual control showed that complete removal of the purulent matter from the antrum by this means was possible in only a small number of cases. There was surprisingly poor correlation between X-ray and endoscopic investigation - Draf (1983) found complete agreement in only 42%, moderate agreement in 36% and no agreement in 22% of 301 random maxillary sinus endoscopies with similar findings in the frontal and sphenoidal sinuses.

X-rays, transillumination and ultrasound are not infallible, even proof-puncture can be misleading. Sinuscopy remains the only definitive diagnostic procedure. Like proof puncture, however, it is unpleasant whether undertaken via the canine fossa or the inferior meatus and is not without hazard. It is unlikely that it will ever be undertaken on a routine basis on all patients presenting with nasal symptoms.

A full history and examination, combined with radiological investigation and careful follow-up to monitor response to treatment would appear to give sufficient information in the majority of cases, reserving endoscopy of the sinuses for those cases which are either refractory to treatment or where the former investigations indicate it.

Nasal mucociliary clearance

Nasal mucociliary clearance comprises two major components, thick mucus which is beaten away by cilia (appendages at the luminal border of the ciliated epithelial cells). Transport of mucus in the nose may therefore be affected by abnormalities of ciliary beating in periciliary fluid and those of the physicochemical quality and quantity of mucus. A simple measurement of the transport of mucus is therefore valuable in screening for these abnormalities which may underlie or be secondary to nasal disease.
Quinlan et al (1969) developed a method for measuring nasal mucociliary clearance in man; this involved placing a radioisotopically labelled particle on the anterior nasal mucosa and tracing its clearance with a gamma camera. This method, while having research potential, is not practical in the routine clinical situation. Andersen et al (1974) described a technique in which they replaced the labelled particle by saccharin and this has become the standard method for measuring nasal mucociliary clearance. A 0.5 mm particle of saccharin is placed approximately 1 cm behind the anterior border of the inferior turbinate. It is important not to place it too far anteriorly as clearance here is forwards rather than backwards. The time elapsing until the first experience of a sweet taste at the posterior nasopharynx is recorded as the nasal mucociliary clearance time in minutes. If carefully performed, the test is reproducible (Stanley et al, 1984). Patients should be tested in standard environmental conditions and must be instructed not to sniff, eat or drink and to avoid coughing and sneezing if possible. They should be tested in the sitting position with the head about 10° flexed to avoid the particle falling backwards into any postnasal stream, and should not be told the nature of the particle. The particle should be inserted under direct vision to ensure there is no gross mechanical obstruction to the test. If a patient is unable to perceive the correct (sweet) taste after 60 minutes, it is important to test his ability to taste saccharin placed directly on the tongue as, rarely, persons may lack this ability.

The rate of clearance has a wide range in normal persons. Proctor (1982) using the radioisotopically labelled particle method found that in apparently healthy adult subjects under optimal environmental conditions, the result ranged from 1 to over 20 mm/minute. Nevertheless, the saccharin clearance method is simple, inexpensive and useful as a routine investigation to screen gross mucus transport abnormality. In children, dyeing the particle of saccharin can assist in verifying when the child tastes, the dye appearing from the postnasal space on oral examination. In one study, all but two of 30 healthy controls tasted saccharin within 20 minutes while 28 out of 158 patients presenting with mucopurulent rhinitis did not perceive a sweet taste after 60 minutes or more (Mackay et al, 1983). It is wise to repeat the test if it is abnormal, using the opposite nostril. There was a good correlation between the results of tests in normal volunteers performed 2 weeks apart and in opposite nostrils (Stanley et al, 1984) and, in most, the transport time was 30 minutes or less.

If a patient, in the absence of mechanical obstruction, is found consistently to be unable to perceive the taste of saccharin 60 minutes after the test is commenced (innate inability to taste it having been ruled out by testing the taste of it directly on the tongue), mucus transport is grossly abnormal and further tests are required to indicate whether the ciliary or mucus component of transport is responsible.

**Ciliary beat frequency**

Having found a grossly prolonged mucus transport time by the saccharin test, it is simplest first to ask whether the cilia are capable of beating normally by obtaining strips of ciliated epithelium from the lateral aspect of the inferior turbinate using a fibreoptic bronchoscopy cytology brush. This procedure requires no anaesthesia and can be safely employed at all ages, if necessary serially. The specimens are transferred to buffered saline by agitating the brush in the solution, and mounted on a coverslip-slide preparation sealed with silicone grease. A photometric method (Rutland and Cole, 1980) is then used to measure the beat frequency of cilia which are kept at body temperature on the warm-stage of a phase-
contrast microscope; light is 'gated' and shone through a small area of cilia from below, the effective 'straight arm' beat of the cilium interrupting the light beam which is detected by a photometer. The electrical signal generated is processed by a ciliary beat frequency analyser (Greenstone, Logan-Sinclair and Cole, 1984) and the beating expressed in hertz (beats/s). The normal range in man for nasal cilia is 12-15 Hz, there being a gradient with slower beating more peripherally in the bronchial tree (Rutland, Griffin and Cole, 1982).

Simultaneous with this measurement of ciliary beat frequency, the percentage of cilia which are immotile can be determined by direct vision as, occasionally, ciliary beat frequency may be only at the lower limit of the normal range but the number of cilia beating is low.

The primary, genetically inherited syndrome which was formerly termed 'immotile cilia syndrome' and which is now named 'primary ciliary dyskinesia' (because the cilia are often not completely immotile), initially presents to paediatricians and otolaryngologists and can be diagnosed using this methodology. However, recently, it has been recognized that cilia obtained from sites of purulent infection beat slower than normal (Wilson et al, 1986). Therefore, as this slowing is a secondary effect due to elastase 'leaked' from neutrophils (Smallman, Hill and Stockley, 1984) and toxins released by bacteria (Wilson, Roberts and Cole, 1985; Sykes et al, 1987), it is important to be sure that primary ciliary dyskinesia is only diagnosed from specimens taken from areas which are not grossly purulent. If necessary, infection may need to be treated and the test repeated.

**Blood tests**

Besides routine investigations such as full blood count with differential white cell count, erythrocyte sedimentation rate, urea and electrolytes, and liver function tests to detect underlying disease predisposing to infection, it is crucial to test immunological host defences (Cole, 1985). This can be done by testing serum for deficiency of IgG, IgA and IgM. In this way, panhypogammaglobulinaemia and selective antibody deficiency (for example IgA) will be diagnosed. This is particularly important in the case of rhinosinusitis, first, because hypogammaglobulinaemia is treatable by immunoglobulin replacement injections/infusions; second, because early diagnosis (and treatment) will avoid irreversible damage especially to the lungs; and third, because these conditions usually present first to paediatricians and otolaryngologists (Mackay et al, 1983). Missing such treatable conditions, albeit uncommon, frequently leads to tragic consequences with either death from acute, fulminating infection or development of irreparable bronchiectasis. Rarely, total Ig classes may be normal but subclasses of IgG may be deficient and require treatment (Stanley, Corbo and Cole, 1984), but estimation of such subclasses usually requires blood to be sent to a special centre.

The role of IgA in the respiratory tract is still much in doubt, although most books tend dogmatically to state its primary role in bacterial disease. Certainly, it has an important antiviral role but its antibacterial effect is not well worked out. Patients with local secretory IgA deficiency can usually be detected by their concomitant deficiency of serum IgA, but it is important to remember that local and systemic systems are distinct and a normal serum IgA does not exclude deficiency or absence of secretory IgA. The latter can be directly tested for in special centres (Stanley and Cole, 1985).
Differential diagnosis of facial pain

Many patients presenting with facial pain will believe they have sinusitis. The pain associated with acute sinusitis has been described above and Chapter 20 is devoted to headaches and facial pain. There follows here, however, a brief outline of conditions which may present with pain mimicking sinus disease.

Dental neuralgia

Dental caries developing infection in the dental pulp will be characterized by poorly localized pain. The affected tooth may be sensitive to thermal change but not to percussion until the periodontal membrane is involved. Apical infection will lead to swelling of the cheek which at times may be marked. The swelling in this region may cause opacity of the sinus X-rays which may be confused with that seen in sinusitis, sinoscopy being required to ensure the diagnosis.

Pain may also occur in the region of an extracted tooth, possibly due to damage of one of the branches of the dental nerve, which may continue for weeks or months.

Trigeminal neuralgia

This common condition of unknown aetiology tends to occur mainly in the second half of life. The pain is unilateral and usually starts in one or both of the lower two divisions of the fifth cranial nerve. Paroxysms of severe stabbing pain may occur spontaneously or be precipitated by minimal movement, such as talking or eating, or occasionally by light touch. Attacks may occur intermittently for weeks or months followed by periods of remission.

Migraine

This pain is usually unilateral, there may be prodromal symptoms (including visual disturbances, such as 'fortification' spectra, nausea, vomiting, hemianopia, unilateral paraesthesia and occasionally even dysphasia) followed by the severe and debilitating headache.

A particular and uncommon variety of migraine which may easily be confused with sinus disease is migrainous neuralgia (or cluster headaches). Classically, the patient is awakened every night with severe pain in and around the eye. The pain lasts about 20 minutes and is accompanied by redness and watering of the eye with swelling of the eyelid. The pain is said to be excruciating and the patient usually gets out of bed and paces the floor. The attacks occur in clusters, lasting a few weeks with intervals of as long as 12-18 months.

Temporal arteritis

This usually occurs in patients over the age of 60 years who develop pain and tenderness over a swollen and tender temporal artery.
Nasopharynx tumour

Tumours in this site may remain asymptomatic for many months. Occasionally, facial pain may be the first symptom. If this is accompanied by conductive deafness, immobility of the soft palate and an enlarged lymph node in the neck, the diagnosis becomes more obvious.

Brainstem lesions

Primary and secondary lesions of the pons and medulla, multiple sclerosis, thrombotic lesions and syringobulbia may cause facial pain. Herpetic and postherpetic neuralgia may also cause pain similar to sinusitis.

Psychogenic facial pain

Psychogenic pain may arise as a manifestation of stress in an otherwise fit person, as a symptom of psychiatric illness such as an anxiety neurosis or depression, or as a feature of a 'built-in' personality trait. Such pain can be divided into a number of different symptom complexes including facial arthromyalgia (temporomandibular joint dysfunction or Costen's syndrome), atypical facial pain, atypical odontalgia and oral dysaesthesia. The pain differs from that of well-defined neurological conditions in that it does not correspond to any cranial nerve distribution and is often bilateral (Feinmann and Harris, 1984).

Facial arthromyalgia causes a dull ache in and around the temporomandibular joint with referred otalgia and occasional shooting pains radiating upwards towards the temple and down into the neck. It has been attributed to bruxism, missing teeth, lax joint ligaments and malocclusions, although Thomson (1971) found the incidence of malocclusion no higher in patients with arthromyalgia than in the general population.

Atypical facial pain is localized to the facial bones or tooth bearing part of the jaws rather than the temporomandibular joint or facial muscles. It is subdivided into atypical odontalgia - pain or discomfort in the teeth - and oral dysaesthesia - burning or altered sensation in the tongue, gingiva, lips or denture bearing area without detectable local or systemic pathology.

Treatment of acute rhinosinusitis

The treatment of acute sinusitis is medical. Antibiotics, analgesics and decongestant medication reduce oedema and increase clearance and drainage from the sinuses.

Antibiotics

Bearing in mind the likely bacteria involved (as described above), oral amoxycillin (or if parenteral treatment is given, the less well orally absorbed but cheaper drug ampicillin) should be considered the drug of choice in treating acute sinusitis. It will be effective against Strep pneumoniae and the majority of Haemophilus spp which are the commonest organisms involved. It will not, however, be effective against Staph aureus, beta-lactamase-producing Haemophilus influenzae, some anaerobes and some aerobic Gram-negative rods. It seems reasonable to initiate treatment on an empirical basis without resorting to culture, with
ampicillin 500 mg four times daily or amoxycillin 500 mg three times daily for 10 days (Mackay, 1984). If the patient fails to respond, antral lavage should be undertaken and the washings sent for aerobic and anaerobic culture.

In some cases, the dose given above does not appear to be adequate and one might consider increasing the dose to amoxycillin 3 g twice daily. This can be prescribed in sachets and the patients should be instructed to take them before meals.

In patients known to be allergic to penicillin, co-trimoxazole (trimethoprim 80 mg plus sulphamethoxazole 400 mg) two tables, twice daily has been shown to be effective (Hamory et al, 1979). Although tetracyclines have been used in the past, pneumococci and *H. influenzae* are not always sensitive. Cefaclor is effective against *H. influenzae* (including those that are beta-lactamase positive) and *Staph aureus*.

**Decongestants and anti-inflammatory drugs**

The key to successful treatment of sinusitis is to maintain clearance/drainage by ensuring that the ostia are kept patent. Once blocked by mucosal swelling the oxygen content within the sinus will decrease, while the carbon dioxide level increases, particularly if purulent secretions are present (Aust and Drettner, 1974). This encourages the growth of anaerobic bacteria and pneumococci which may be facilitating anaerobes. Low oxygen tension, high carbon dioxide tension and lowered pH hampers the bactericidal activities of polymorphonuclear leucocytes. The purulent secretions and bacteria may also affect ciliary function (as previously mentioned) so that normal clearance is rapidly inhibited and inflammatory products may only be cleared by gravity drainage via the ostia, and any decrease in patency impedes this further.

Systemic decongestants do not appear to be as effective as topical preparations (Anggard and Malm, 1983). Oxymetazoline and xylometazoline hydrochloride appear to be effective and have less 'rebound effect' than other topical decongestants. They should not be used for more than a few weeks, however, or there is a risk that the patient may develop rhinitis medicamentosa.

Anti-inflammatory drugs are particularly effective at reducing the mucosal swelling associated with the inflammatory response of infection. A topical spray (Dexa-rhinaspary) containing dexamethasone, neomycin and tramazoline, a decongestant, can be used in conjunction with systemic antibiotics. Sprays, however, are poorly distributed to the majority of the nasal mucosa and this is particularly true in the blocked nose where the best distribution is obtained from a pipette (Mygind, 1979). In addition to this, if there is any remaining activity of the mucociliary system, the effect will be to transport the preparations away from the sinuses. Logically, therefore, drops instilled into the nose in the head downwards position would appear to be the most effective way of decongesting the ostia of the sinuses. The head down and backwards position has been advocated by Mygind, but radiological studies lead us to believe that the head down and forwards position as shown is the most effective and clinical trials support this (Chalton et al, 1985; Wilson et al, 1987c). Patients are instructed to instill two drops of betamethasone into each nostril and remain in the head down and forwards position for 2 minutes. When the nose is particularly blocked, prior application of a nasal decongestant can improve penetrance.
Analgesics

The pain of sinusitis can usually be relieved by aspirin and codeine preparations; it is unusual to have to resort to opiates. Special caution should be taken to ensure that the patient is not allergic to aspirin in view of the association between aspirin intolerance and rhinitis.

Surgical treatment is rarely required for acute sinusitis. Occasionally, however, maxillary sinusitis does not respond to conservative measures, in which case lavage may be indicated and, when this is required, it would appear to be beneficial to repeat it at frequent intervals to remove pus, restore ciliary activity and ventilate the sinus. Drettner (1983) has described a method whereby a plastic tube can simply be introduced via the cannula into the antrum. The tube has a 'memory' so that it curls into a spiral shape unless it is straightened out with a trocar inserted along its length; once this is removed, the tube will curl and remain in situ for as long as necessary, allowing repeated and frequent irrigation without new punctures.

The frontal sinus may occasionally require surgical drainage by an incision made immediately below the inner margin of the eyebrow together with an opening made with a burr. A silicone drainage tube can be inserted into the sinus via the wound and sutured in situ.

Sydow, Axelsson and Jensen (1982), compared 27 different modes of treatment for sinusitis. Each group consisted of between 34 and 74 patients. In all, 1320 patients (2039 maxillary sinuses) were treated and assessed both on clinical grounds and radiologically. One group was treated with topical nasal decongestants alone, another with only systemic decongestants, a third group by irrigation of sinuses alone, three groups were treated with antibiotics alone (amoxycillin, pivampicillin and axidocillin) and the remainder with penicillin V, lincomycin, methicillin, doxycycline, spiramycin, ampicillin, cephradine, erythromycin and bacampicillin, all with or without irrigation, nasal decongestants or both. Their conclusion was that the therapeutic outcome differs very little among the groups, although those patients treated with nasal decongestants alone (both topical and systemic) appeared to do least well. It is also interesting to note that Mann et al (1981) reported a 79% spontaneous cure after 2 weeks in patients treated with analgesics alone.

Treatment of chronic rhinosinusitis

The principles involved in the treatment of chronic rhinosinusitis are first to attempt to identify and treat the underlying cause and second, if possible, to restore the inflamed mucosal lining to health.

Whatever the underlying cause, logical treatment is required to interrupt the 'vicious circle' (see above) of amplified inflammation which results in progressive tissue damage in the long term. There are a number of points on the vicious circle where treatment may be aimed, notably, at the colonizing microbial flora using antimicrobial agents, at the tissue-damaging inflammatory response to this flora using topical anti-inflammatory agents and at the compromised mucociliary clearance. The latter is perhaps the crux of treatment (Sykes et al, 1986) for if the sinuses can regain normal clearance, this defence mechanism will tend to prevent recurrence of infection in the normal manner. There is a close parallel between
chronic rhinosinusitis and bronchiectasis and similar principles of management apply, the
overriding one being to achieve either normal mucociliary clearance or the less desirable
substitute, artificial drainage, which is gravity assisted postural drainage in the case of
bronchiectasis but surgical antrostomies in sinusitis. Occasionally, the mucosa may be deemed
to be irreversibly damaged and complete removal of the lining will be the only alternative.
This will, however, be replaced with scar tissue which can never restore normal clearance
function and is therefore always vulnerable.

Such an example raises an important practical aspect of treatment of chronic sinusitis
which contrasts with that of the acute disease. Probably, the majority of patients with
established chronic sinusitis have at least some irreparable damage. In practice this commits
most of them to long-term treatment. The advent of safer antimicrobial and anti-inflammatory
therapy is forcing the medical practitioner to abandon the 'you'll have to live with it' attitude
and assume active long-term medical therapy with logical surgical intervention as required.
Although at present it is not possible to help adequately all patients with this condition,
especially those with 'end stage' disease, nevertheless, persistence with logical principles of
treatment does improve many so long as it is clearly realized that such therapy must be
actively pursued for long periods of time, often for life.

Inflammation results in swelling of the mucosal lining which quickly blocks the
natural ostia. Secondary bacterial infection then occurs and the first line of treatment will
therefore include both antibiotics and medication to reduce inflammation. Topical
decongestants may be effective for short-term use but with chronic infection, long-term
medication is invariably required and the authors therefore prefer to use topical anti-
inflammatory drugs such as betamethasone. Undoubtedly, the most effective way for this to
reach the ostia is to use drops in the head down and forwards position instilling two drops
in this position and maintaining the posture for 2-3 minutes two or three times daily (Wilson
et al, 1987c). This can be combined with either topical antibiotics such as gentamicin or
neomycin or systemic antibiotics such as amoxycillin or cotrimoxazole.

When treatment with this regimen over a period of 3-4 weeks fails, surgical treatment
will be required. Sinus washout may be helpful in the treatment of acute sinusitis before the
mucosa has become too inflamed, but it is unlikely that a single washout will benefit a patient
with chronic mucosal changes. The disadvantage of weekly washouts can be overcome by
using an indwelling tube which can be used for daily lavage, instillation of medication and
aeration. If simple medical treatment fails, it is likely that the problem will be recurrent and
it appears more logical to attempt to provide more permanent drainage by undertaking an
intranasal antrostomy or similar drainage procedure of the other sinuses when they are
involved. The surgical management of this is described in Chapter 11.

Treatment of the underlying condition

Mechanical obstruction

Surgery to correct a deviated nasal septum will certainly be helpful if this is causing
sufficient obstruction to interfere with normal sinus drainage. This, however, is probably
seldom the case, although septal surgery is certainly important to improve the nasal airway.
Surgical reduction of the turbinates however may radically influence sinus drainage; the inferior turbinates, unless reduced, may obstruct an inferior meatal intranasal antrostomy and the middle turbinates may require careful conservative reduction if these are influencing maxillary and frontoethmoidal drainage.

**Allergy**

The mechanism and treatment of allergy, particularly allergen avoidance measures, are dealt with in Chapter 7. In some instances, an underlying allergic aetiology may compromise the normal clearance mechanism leading to secondary bacterial infection. In such cases, it will be necessary to treat the inflammatory process first with antibiotics and anti-inflammatory drugs (and possibly surgery) to restore the normal patency to the sinuses. Once this has been achieved, the underlying allergic problem will require maintenance therapy.

The treatment of allergic rhinitis has been revolutionized over the last few years by the introduction of first, topical corticosteroids (beclomethasone, flunisolide and budesonide) and second, non-sedating H\(_1\)-antagonist antihistamines (terfenadine and astemizole). The latter rarely sedate or potentiate the action of alcohol. Terfenadine must be taken twice daily and is rapidly effective although remains so for a relatively short period (6-12 hours). Astemizole has the advantage of once daily dosage although its maximum effectiveness may not be obtained for a week or more. Skin tests may remain negative for up to 4 weeks from the last dosage. Both are contraindicated in pregnancy.

The combination of topical corticosteroids and systemic H\(_1\)-antagonists used simultaneously is particularly effective. Occasional dryness and bleeding may occur with topical steroids but no irreversible damage to the mucosa has been reported and, provided they are used in the recommended dosage, they have no systemic side-effects.

Systemic corticosteroids are probably underused in the management of this condition. Because of their well-recognized side-effects, they are not suitable for long-term maintenance therapy but are very useful as a short course to bring the symptoms under control with a view of maintaining any improvement using topical corticosteroids, antihistamines or sodium cromoglycate. The latter is less effective than topical corticosteroids but is an entirely safe medication, is only effective as a prophylactic agent and is more likely to be effective in atopic patients.

The prospect of long-term maintenance therapy does not appeal to some patients and hyposensitization or surgery may be requested. Hyposensitization, more effective in children than adults, is only of proven value in the treatment of single allergen (pollen or house dust mite) disease. It carries, however, considerable risk of anaphylaxis and, as such, is not recommended. Such treatment in atopic patients may worsen existing allergic disease or provoke asthma, and is more likely to provoke anaphylaxis in such patients.

Allergy cannot be 'cured' by surgery. That does not mean that surgery has no place, however. Surgical reduction of the turbinates, correction of deviation of the nose or septum and surgery to improve sinus patency may all play an important role, particularly where nasal obstruction is the patient's main symptom. Some patients may be able to contend with
sneezing and rhinorrhoea provided they have an adequate airway. For many, however, surgery is indicated to restore function followed by long-term medication to maintain it.

**Abnormalities of mucociliary clearance**

These may primarily affect the ciliary or mucus component of clearance (primary ciliary dyskinesia, Young's syndrome and cystic fibrosis in which mucus is abnormally thick and tenacious) or be secondary. Primary syndromes are not 'curable' as yet, but secondary abnormalities (due to changes in mucus viscoelasticity induced by bacterial infection, for example after a viral infection of the upper respiratory tract; due to bacterial product- or neutrophil elastase-inhibition of ciliary function) can be treated in the expectation that in a reasonable proportion of patients the underlying condition will resolve and allow mucus and/or cilia to return to normal. In many cases, the chest is also involved in such disease and requires corresponding treatment (Stanley et al, 1985). At this point, it is appropriate to point out that cigarette smoking reduces mucociliary clearance and ciliary beat frequency in the nose as well as the bronchial tree, so that such habits may adversely affect treatment for secondary clearance abnormalities (Stanley et al, 1986).

With primary ciliary abnormalities such as primary ciliary dyskinesia, and primary mucus problems such as cystic fibrosis and Young's syndrome, as yet no 'cure' can be achieved; this is not to say, however, that symptomatic treatment is not helpful. The authors' experience suggests that these patients can obtain symptomatic relief with topical mucolytics such as alkaline nasal douche which is sniffed through the nose two or three times daily followed by the application of topical corticosteroids. Antral washout will not give lasting relief as it will do nothing to improve the underlying primary problem. If sinus X-rays reveal a fluid level, an antrostomy should be undertaken. As there can be little or no mucociliary transport, a middle meatal antrostomy will not be beneficial and a radical inferior meatal antrostomy, combined with surgical reduction of the inferior turbinate, is indicated to enable adequate drainage by gravity as well as facilitate aeration of the sinus and provide access for douching and, if necessary, topical medication.

Nasal polyps may occur with primary ciliary dyskinesia: in two out of 16 (Greenstone et al, 1985), six out of 15 (Pedersen and Mygind, 1982), 10 out of 33 (Levison et al, 1983). If these do not respond to topical corticosteroids they will require surgical removal.

Glue ear is another common finding in primary ciliary dyskinesia. Myringotomies were performed without success in four out of 16 patients reviewed by Greenstone et al (1985). The ears continued to discharge through the ventilation tube until it was either removed or extruded. Since there is no likelihood of primary ciliary dyskinesia resolving, it would seem unhelpful to advocate insertion of ventilation tubes. Fortunately, the hearing loss is seldom severe and in the authors' series, patients' otological problems did not deteriorate despite the condition having been present for many years in the older patients.

For many patients with primary nasal mucociliary clearance problems, their otolaryngological symptoms present little more than an inconvenience, possibly because their symptoms have been present for most of their life and they accept them as normal. For many, a simple medical regimen will be helpful; in some, surgical procedures to improve sinus drainage may be indicated. The most important aspect, however, should be an awareness of
the condition in the hope that early diagnosis and referral for appropriate chest management may prevent or delay the onset of serious chest disease such as bronchiectasis.

**Immunity deficiency**

Although, as previously described in this chapter, there are a number of immunity deficiency states which usually present initially to otolaryngologists or paediatricians, the condition panhypogammaglobulinaemia (very low IgG, IgA and IgM) is by far the most important to recognize and treat, because irreparable lung damage may be avoided thereby. Although an uncommon condition, missing the diagnosis is disastrous.

Conditions in which IgG and IgM are low or absent are treated by reconstitution with immunoglobulin. The standard therapy until recently was intramuscular injections of normal human immunoglobulin (Lister) given weekly in a dose of approximately 25 mg/kg body weight, varying according to its catabolism by the patient. In practice, this entailed painful, large volume injections usually divided between the buttocks. Nevertheless, patients preferred such discomfort to their infections. Monitoring of serum levels of immunoglobulin was often of limited help and the dose in such patients was regulated according to clinical control of frequency and severity of infections. The addition of fresh frozen plasma infusions at intervals to the regimen was sometimes required in the more seriously affected patient. Untoward effects (for example, anaphylaxis, rashes) were common and unpredictable.

The situation has now changed with the advent of intravenous preparations of immunoglobulin which appear to be less likely to cause untoward effects. Such preparations are given by infusion at less frequent intervals (usually 2-4 weekly) and have the advantage that the dose can be increased without increasing discomfort as occurred with intramuscular preparations. However, the preparations are very expensive and, because they have all been relatively recently introduced, the efficacy of some is not fully known. Opsonization of bacteria for phagocytosis is one important function of immunoglobulin and this can be used as the basis of a laboratory test to compare the efficacy of various intravenous preparations in vitro (Munro, Stanley and Cole, 1985).

Selective immunoglobulin A deficiency is not treatable at present (and, as mentioned before, it is debatable whether reconstitution of IgA would affect bacterial infections). However, IgA deficiency seems to be a marker for IgG subclass deficiencies in some cases and the latter are probably treatable by replacement immunoglobulin. At present, the situation is much clearer in children (Oxelius et al, 1981) than in adults.

The complexity of the immunological deficiencies which may be associated with chronic or recurrent rhinosinusitis make it mandatory to refer affected patients to a specialist centre.

**Granulomatous conditions**

Although these conditions will require appropriate systemic medication, topical treatment with alkaline nasal douche to remove crusts and inspissated mucus followed by the instillation of corticosteroid and antibiotic drops (betamethasone and neomycin) in the head down and forwards position, may not only give symptomatic relief but may, at times, control
the local condition, such as sarcoid restricted to the nose, in such a way that the systemic medication can be reduced. The treatment of Wegener's granulomatosis is dealt with in Chapter 18.

**Autonomic imbalance**

**Non-specific rhinitis (nasal hyperactivity, intrinsic rhinitis or 'vasomotor rhinitis')**

Because this condition, or perhaps group of conditions, is so ill understood, it is equally difficult to treat and the authors have found it more helpful from a practical point of view to categorize the treatment according to the patient's 'main complaint', that is obstruction, sneezing and rhinorrhoea.

**Obstruction**

Where nasal obstruction is the main complaint, and particularly where it is the only complaint, surgery would appear to give the best results. An initial trial of topical corticosteroids and antihistamines is reasonable, but the authors in reviewing over 1000 patients, found it beneficial in less than one-half. Prior to undertaking surgical reduction of the turbinates, it is important to confirm that the nose is indeed obstructed as patients' subjective assessment of their nasal airway is poor and rhinomanometry is helpful in establishing which patients are likely to benefit.

**Sneezing**

Topical treatment is less helpful in treating those patients where the main complaint is one of sneezing. Budesonide was shown by Pipkorn (1983) to be slightly preferable to other topical corticosteroids for this condition but, by and large, most patients in this group are not helped and may even find their symptoms are made worse.

Antihistamines offer the best chance of success. Astemizole is useful in that it need only be taken once daily and, because of its pharmacokinetics, if for any reason the medication is omitted for a day or two, it will continue to be effective. Many patients in this group find that their symptoms occur in bouts: they may remain symptom-free for weeks, then experience a severe bout of intractable sneezing for several hours or days. Terfenadine, which is quickly effective, may be more suitable for this group, using it only when required.

**Watery rhinorrhoea**

Non-specific watery rhinorrhoea may respond to antihistamines and topical corticosteroids but when it does not, topical ipratropium offers an effective alternative.

When medical treatment fails, surgical division of the nerve of the pterygoid canal (vidian neurectomy) can be considered. The nerve can be approached via the maxillary sinus (Golding-Wood, 1961); other approaches are transpalatal (Chandra, 1969), trans-septal (Minnis and Morrison, 1971), and transnasal (Kirtane, Prabhu and Karnik, 1984). Complications include diplopia, anaesthesia of the palate and infraorbital region, impairment of lacrimation, blindness, infraorbital neuralgia and sinusitis. Kirtane, Prabhu and Karnik (1984) claimed that
the transnasal approach is the simplest and most direct and reported no serious complications in 247 cases, over 95% having relief from their symptoms of rhinorrhoea and sneezing, but no long-term results are reported. Until long-term controlled studies have been carried out, vidian neurectomy is probably best left to those few surgeons who have special experience of it and should be avoided by others.

**Emotional rhinitis**

Whether depression is the cause or result of rhinitis remains debatable. There can be no doubt, however, that many patients with this condition can be successfully treated with imipramine which not only has an antidepressant effect but also acts as an anticholinergic drug. An initial dose of 25 mg three times daily can be increased to a maximum of 200 mg daily if drowsiness is not a problem. It is important to note, however, that it may take 4 weeks or more before the maximum effect is experienced.

For those patients in whom no diagnosis can be made an no objective evidence exists for their symptoms, explanation and reassurance alone are preferable to medication or surgery, which are unlikely to benefit the patient and may later even be blamed for their condition: hence 'I had no trouble with my sinuses until they were washed our'.

**Hormonal**

**Pregnancy**

Nasal obstruction is usually the main complaint in rhinitis associated with pregnancy and, because of pregnancy, it is difficult to treat. Topical nasal decongestants may be helpful but should not be continued long term. The problem of 'rebound' can, to some extent, be alleviated by decongesting one side of the nose one day and the other side the next. Linear diathermy to the surface of the inferior turbinate under local anaesthesia may give more lasting relief but may result in greater nasal obstruction for the first 7-10 days.

Topically acting corticosteroids such as beclomethasone are unlikely to have any significant undesirable systemic effect and may prove beneficial. Antihistamines, particularly astemizole and terfenadine, are specifically contraindicated during pregnancy.

Secondary bacterial infection may be treated with penicillins but tetracyclines should not be used. Acute infection of the maxillary sinuses not responding to medical treatment alone may require antral lavage under local anaesthesia.

**Menopausal women**

Atrophic rhinitis occurring in women after the menopause may respond to topical oestrogen. Manual removal of large crusts is followed by regular douching with alkaline nasal douche after which conjugated oestrogen cream (Premarin) is applied.
Senile rhinorrhoea

This problem affects men more than women and, although testosterone propionate was advocated by Watson-Williams (1952), the authors have no experience with this. Most patients in this group however can be controlled with topical ipratropium (Atrovent).

Iatrogenic

Rhinitis medicamentosa resulting from overuse of topical decongestants is best managed by explanation of the mechanisms of 'rebound'. It is also necessary to look for any other underlying cause for the patient's symptoms, such as allergy. Provided that there are no contraindications, a short course of systemic corticosteroids (prednisolone 10 mg for 5 days reducing to 5 mg for a further 5 days), together with systemic antihistamines (terfenadine 60 mg twice daily for one month) and avoidance of any topical medication may be all that is required. Where there is some other underlying cause, this will require appropriate management and maintenance therapy.

Where this fails, surgical reduction of the inferior turbinates will be required followed by strict avoidance of any decongestants. Partial trimming allows a reasonable airway in the early postoperative stage which can be maintained with an alkaline douche to remove crusts and caked blood clots for the first few weeks.

Concluding remarks

It is hoped that in writing this chapter, the authors have demonstrated the similarities and close relationship between the upper and lower respiratory tract. Knowledge gained from study of disease in one inevitably affects knowledge about the other and is the main reason why otolaryngologists and chest physicians should work in close relationship for mutual benefit and benefit of their patients.