Chapter 8: Acute and chronic inflammations of the nose

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Acute infective rhinitis

The term 'acute infective rhinitis' is taken to mean an acute viral or bacterial infection of the nasal mucous membrane. It is exceedingly common and is a manifestation of the common cold, influenzal infections of the upper respiratory tract, the exanthems and certain specific infections. It can also occur as a secondary response to local irritants and trauma.

The common cold (coryza)

Incidence

The common cold is probably the commonest viral infection in man. The incidence is variable but it is estimated that the average young adult has two to three colds a year. Children and young adults are particularly susceptible to rhinovirus infections and women may experience more infections than men, perhaps because they are in closer contact with young children.

Predisposing factors

Climate

Colds occur all the year round but in temperate climates they are more common in winter than in summer. Rhinovirus infections are more prevalent in autumn and spring and coronavirus infections seem to occur mostly from December to March.

Environment, temperature, chills and humidity

Kerr and Lagen (1933-34) exposed groups of susceptible men in the same room, under perfect conditions of humidity, temperature and ventilation, with subjects in the early stages of a cold, and in some they even inoculated cold filtrates into the conjunctival sac without obtaining a single transfer, suggesting that an ideal environment increases resistance.

There is a widespread belief that chilling may precipitate a cold in an individual; however, attempts to demonstrate such an effect experimentally have given negative results (Andrewes, 1950). Chill may act in two ways: by lowering general resistance to infection; and by causing reflex vasoconstriction of the nasal mucous membrane. The normal temperature of the mucous membrane of the nose has been shown to vary between 33 and 34°C. Chilling of the body surface may reduce the temperature of the nasal mucosa by as much as 6°C (Mudd, Goldman and Grant, 1921). The optimal relative humidity of the atmosphere is 45%. A lowering of relative humidity to 15%, as may easily occur when the relatively dry cold outside air in winter is heated indoors by radiators, withdraws more water than the nasal mucosa can supply, and causes drying of the mucous blanket. Excessive humidity is also harmful, as it reduces the evaporation of sweat from the skin and, owing to the high
conductivity of water vapour, a slight lowering of temperature produces severe chilling, with the effects described above.

Hope Simpson (1958a,b) demonstrated a striking correlation between increase in the frequency of colds in a group of families and falls in the temperature of the soil. Humidity also affects the survival of viruses (Tyrrell and Bynde, 1961). Common cold viruses prefer high humidity. Influenza viruses survive better in dry air.

**Immune status**

Local immunity in the nose is primarily the result of concentrations of IgA and IgG, which are normally present in nasal secretions in the ratio of 3:1 compared with 1:5 in serum. IgA is produced in response to local antigen stimulation but does not combine with complement and therefore is unable to lyse bacteria; it is, however, effective as a viral neutralizing substance. IgA is a relatively short-lived antibody the half-life of which has been estimated to be 13 days but, in practice, is more likely to be measured in minutes because, although there are very large numbers of IgA-producing plasma cells in submucosal tissues, it is soon carried away in mucous secretions. It is thus not uncommon to find reinfection with the same virus serotype in consecutive years. Failure to produce secretory IgA occurs in approximately one in 800 persons who live without ill effects. Patients with generalized hypogammaglobulinaemia, however, have frequent infections of the upper respiratory tract (Wilson and Montgomery, 1980).

**Nutrition and vitamin deficiency**

The lowering of resistance by hunger and undernutrition was shown by Cruickshank (1942), who found that the death rate in measles, pertussis, influenza and bronchopneumonia among poor children was five times greater than among those better off. Deficiencies in vitamins A, C and D are said to increase susceptibility to infection but the claim that vitamin C is effective in preventing colds is not supported by controlled trials (Andrewes and Tyrrell, 1965).

**Fatigue, fitness and exercise**

Locke (1937) assessed the fitness of subjects by their oxygen consumption under standard exercise, and found that 64% of those with fitness above 0.6 had only one cold a year, while 80% of those below 0.5 had four colds a year. However, colds very often hit the man who is feeling very fit on his return from holiday (Andrewes and Tyrrell, 1965).

**Nasal obstruction**

Deviation of the nasal septum, hypertrophy of the turbinates, enlarged adenoids, polyps, scars and adhesions all interfere with ventilation and the free passage of air through the nasal chambers, and with the secretion and movement of the mucous blanket, and thus predispose to infection.
Foci of chronic infection

Foci of infection in the sinuses, nasopharynx or pharynx, by decreasing tissue resistance, favour infection. The more important of these in children are chronic adenoiditis, tonsillitis and sinusitis and, in adults, chronic sinusitis and tonsillitis.

The pH of nasal secretion

A drift to the acid side is associated with few bacteria, while an alkaline drift is associated with many bacteria in the nasal secretion. Rhinoviruses are destroyed by an acid pH (Ketler, Hamparian and Hilleman, 1962).

General diseases

Any general disease, but particularly renal, hepatic and blood disorder, diabetes mellitus and tuberculosis, may lower general resistance to colds.

Causative agents

Viruses

In general it may be said that, in communities, the causative agent of the common cold is ubiquitous, but that infection occurs only when an individual's resistance is lowered, or when is subjected to an overwhelming concentration and virulence of the causative agent. It is generally accepted that the common cold is due to infection with filterable viruses, followed by secondary infection with bacteria.

In spite of the rapid advances in virology and the isolation, identification and even culture of many viruses, it is still uncertain what proportion of respiratory illnesses is caused by them. The viruses responsible for colds are listed in Table 8.1 (Reed, 1981).

Table 8.1 Viruses causing common cold

<table>
<thead>
<tr>
<th>Virus</th>
<th>Serotypes</th>
<th>Proportion of colds (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rhinovirus</td>
<td>89 different types, probably more</td>
<td>50</td>
</tr>
<tr>
<td>Coronavirus</td>
<td>3 or more types, and possibly subtypes</td>
<td>15-20</td>
</tr>
<tr>
<td>Influenza</td>
<td>A and B and their subtypes; C Together</td>
<td></td>
</tr>
<tr>
<td>Parainfluenza</td>
<td>Types 1, 2, 3, 4</td>
<td>about</td>
</tr>
<tr>
<td>Respiratory syncytial</td>
<td>One type</td>
<td>15-20</td>
</tr>
<tr>
<td>Adenovirus</td>
<td>36 types, but only about half of them</td>
<td></td>
</tr>
<tr>
<td></td>
<td>causing respiratory tract infection</td>
<td></td>
</tr>
<tr>
<td>Other viruses</td>
<td>Includes some enteroviruses, other known</td>
<td>10-20</td>
</tr>
<tr>
<td></td>
<td>viruses, and perhaps some unknown</td>
<td></td>
</tr>
</tbody>
</table>
Each of the viruses listed in Table 8.1 can be said to be associated with its own 'typical' clinical effect which, for rhinoviruses and coronaviruses, is the common cold syndrome. Influenza viruses, respiratory syncytial viruses are, however, known to cause more serious infections.

**Rhinoviruses**

These are members of the picornavirus group and are thus biologically related to polioviruses and other enteroviruses, and to foot and mouth disease virus. They are about 25 nm in diameter and incorporate protein subunits in which the antigenic specificity of each of 89 or more types is incorporated. They have an optimal growth temperature of 33°C. Nasal swabs and nasal washings produce the best specimens for culture on human embryonic fibroblasts such as WI-38. Identification of the serotype of a rhinovirus is difficult because of the large range of types. However, antisera can be used against 89 types. A group-reactive serological test for rhinoviruses is not available and therefore retrospective diagnosis of rhinovirus infection cannot easily be established using paired sera collected in the acute and convalescent stages of the illness.

**Coronaviruses**

Coronaviruses were first classified as a distinct group in 1968 but are still not fully understood because of the technical difficulties in isolating them from clinical specimens. They are RNA-containing viruses about 100-120 nm in diameter. Two early isolates were named 229E and OC43. The former can be grown in tissue culture, but the latter needs organ cultures of human embryonic nasal epithelium or trachea. Serological tests available include complement fixation and neutralization tests for 229E, and complement fixation and haemagglutination inhibition for OC43. Coronaviruses cause typical colds. The incubation period is slightly longer than for rhinovirus colds.

**Mode of transmission**

**Droplet and dust**

In talking, sneezing and coughing innumerable infected droplets are sprayed out which fall to the ground at distances of 0.9-1.8 m. Bedmaking, house dust and the manipulation of handkerchiefs also contribute large numbers of airborne particles (Dumbell, Lovelock and Lowbury, 1948).

**Droplet nuclei**

Droplet nuclei are small droplets which evaporate as they fall, and shrink to less than 0.1 mm in diameter. In this form they remain suspended in the air as mist, and drift on the air currents for as long as 2 days, and thus have a much wider range than that of droplets (Wells and Wells, 1936). These will transport viruses, but are too small to carry the larger bacteria.
Contact

The causative organism may be transmitted by kissing, food, fingers and fomites.

Pathology

In the earliest stage of invasion there is transient vasoconstriction. This is followed by a vasodilatation, oedema and increased activity of the seromucinous glands and goblet cells.

Leucocytic infiltration of the tissues follows, with swelling and desquamation of the epithelial cells. The secretion is at first clear, watery and sterile, with a few epithelial and pus cells, but later it becomes coloured and viscid, stiffens on a handkerchief and contains many pus cells and bacteria. The toxins produced in the mucous membrane are swiftly taken up by the lymphatics, and passing through the cervical lymph glands and ducts reach the blood stream. Resolution takes place by a reversal of these processes, and by proliferation of the remaining tissue cells to replace those that have been destroyed.

The lysozyme content is reduced in the early stages. The average pH of the nasal mucus lies between 5.5 and 6.5. During an acute rhinitis the reaction becomes alkaline, and during resolution it returns to neutral.

Bacteriology

Cultures from the normal mucous membrane of the posterior areas of the nose are usually sterile, provided that they are not contaminated from the vestibule and anterior areas. Cultures from the anterior nares show staphylococci in 43% of normal individuals.

In the first 3 days of a common cold the cultures from the posterior areas may be sterile, but after the third day pure or mixed cultures of streptococci, pneumococci, *Haemophilus influenzae*, or staphylococci are often shown (Tweedie, 1934).

Clinical picture

The course of a cold may be described in four stages.

(1) Prodromal or ischaemic stage. This lasts for a few hours, and represents the stage of local invasion and general nasal ischaemia. The familiar hot, dry or tickling spot is felt at the site of the invasion, while the general nasal airway seem unusually patent.

(2) Early reaction and irritation. The infection, which is at first localized, spreads to the adjacent mucous membranes over the surface and by way of the lymphatics. This process may take a few hours or days. The site of invasion is often the first to recover, while the disease is still active in those areas which have been affected later. The throat is dry and sore on swallowing, and there is sneezing, watery nasal discharge and obstruction. The mucous membrane is red and swollen. General symptoms of mild toxaemia and fever now appear.
(3) Stage of venous stasis and secondary infection. After the second day, the colour of the mucosa becomes dusky, with a bluish tinge, the discharge thickens, diminishes and becomes mucopurulent. The obstruction and toxaemia are at their maximum.

(4) Resolution. The symptoms and signs gradually diminish, and after 5-10 days recovery takes place.

Complications

Nasopharyngitis and pharyngitis

The nasopharynx and pharynx are invariably infected to some extent in every cold.

Sinusitis

Sinusitis is one of the most common complications, but the sinuses are not invaded during the course of an uncomplicated cold.

Pharyngotympanic salpingitis, otitis media and mastoiditis

The infection ascends from the nasopharynx, invading the pharyngotympanic tube, middle ear and mastoid cells in sequence. It may be arrested at any point of the ascent.

Lymphadenitis

This is usually transient, and affects the retropharyngeal and deep cervical group.

Tonsillitis

A mild inflammation usually accompanies a cold, but parenchymatous or follicular tonsillitis is considered as a complication.

Lower respiratory complications

Laryngotracheitis, bronchitis, pneumonia and asthma constitute the group of lower respiratory complications.

Gastroenteritis

This complication is rare except in infants.

Other complications

Nephritis and rheumatism are allergic and toxaemic manifestations.

Diagnosis

Other causes of rhinitis should be excluded (see Chapter 6).
Laboratory diagnosis may be made retrospectively by taking acute and convalescent sera and comparing the antibody levels. However, more rapid diagnostic techniques such as immunofluorescence are now available. In the course of an infection large numbers of infected cells are sloughed off into the respiratory secretions. These cells will carry markers or antigens specific for the virus involved and can be recovered by nasal swabs or washings, stained using specific antiviral antisera labelled with a fluorescent dye, and seen by fluorescence microscopy. This technique can now be applied to a range of common respiratory viruses including influenza virus A and B, parainfluenza viruses 1-3, adenovirus and respiratory syncytial virus.

**Prophylaxis and vaccines**

Based on the observation that rhinovirus infections may possibly be spread by the manual route, thorough hand washing with soap and water will remove the virus from contaminated hands. Avoidance of fingerling the nostrils and conjunctiva could reduce the chance of self inoculation.

Because of the antigenic diversity of respiratory viruses, with the exception of vaccines for influenza A and B infections, there are no suitable vaccines against the other respiratory viruses. No specific antirhinovirus compound has yet shown sufficient activity in man to merit further development but, nevertheless, work continues in this field.

Wide spectrum antiviral prophylaxis or therapy would have considerable theoretical advantages and the natural antiviral substance, interferon and its inducers, would seem to be an ideal solution. Double-blind placebo-controlled studies using interferon in rhinovirus-infected volunteers have given encouraging results. It is important though to ensure that any form of antiviral therapy should be of very low toxicity before its use in common colds can be justified.

**Treatment**

There is no known specific treatment for the common cold, but general and local supportive and palliative treatment can mitigate the severity and complications. There are so many different varieties of colds, so many different individual reactions to them, and so many different individual responses to treatment, that no hard and fast therapeutic rules can be laid down.

General treatment is directed to providing the best conditions for rest, both general and local, and at the same time supplying heat and the maximum blood flow to the infected tissues. Unfortunately, the majority of patients are not willing to submit to full-scale treatment for a cold of moderate severity, and modifications must be made, according to the circumstances.

Complete rest, both general and for the upper respiratory tract, necessitates confinement to bed in an even temperature of 18-20°C, with a humidity of 45%.
Heat, both local and general, is provided at first by a hot bath. Inhalations of menthol or tincture of benzoin (BP) (one teaspoonful in a pint of steaming water), may be soothing and will apply heat directly to the mucous membrane of the nose.

Analgesics and antipyretics, such as acetylsalicylic acid, may be valuable for the general malaise, aching and feverishness of the cold. Codeine compounds are more effective as sedatives. Both should be combined with a copious fluid intake.

Antihistamines have not been shown to reduce fully or abolish the symptoms of colds, but they can be particularly effective in the allergic patient who is often unduly susceptible to colds. Antihistamines can be usefully combined with an analgesic.

Alcohol is a sedative which is the chief justification for the faith placed in whiskey as a treatment in the early stages of a cold. It is also a vasodilator and counteracts the discomfort of the peripheral vasoconstriction at that stage.

Local vasoconstrictors should be used sparingly as the excessive use of any vasoconstrictor agent should be avoided on the grounds of interference with ciliary activity, mucosal blood flow and local tissue resistance. Temporary relief may be achieved by using ephedrine 0.5% in isotonic saline. This is particularly helpful in enabling a child to sleep or a baby to suckle.

Antibiotics do not appear to influence the course of a cold and therefore should only be used, and then in full doses, if complications develop such as middle-ear infection, sinusitis, tonsillitis, bronchitis or pneumonia.

**Influenzal rhinitis**

Influenzal rhinitis occurs in association with an infection by one of the influenza viruses. There are three main groups of virus unrelated antigenically (A, B and C).

Influenza A virus which has undergone several mutations since its discovery in 1933 has been responsible for pandemics of the disease. The original A virus has since been replaced by different strains, A1 (1946) and later A2 (1957). It is indeed unfortunate that the virus is subject to antigenic change for there is little or no cross-immunity and an entire population may find itself susceptible to the 'new' virus. Influenza B and C viruses are less liable to antigenic variation. The virology of these infections and its particular interest to the otolaryngologist have been described in some detail by Dudgeon (1969) and also by Anderson (1969).

The characteristic lesion is a varying degree of necrosis of the ciliated epithelium of the upper respiratory tract (nose and, in some cases, trachea). For a time there may even be replacement by transitional epithelium, and secondary bacterial invasion is inevitable.

In some cases of influenza the rhinitic manifestations are not marked or are overshadowed by tracheal, gastrointestinal, pulmonary or general symptoms, but in others severe coryza is simulated and in some of the pandemics many cases have been complicated by epistaxis.
Preventive treatment by the injection of immunizing vaccines is generally applied to those persons leading institutional lives, where the risk of infection is greater, or to the elderly or infirm, particularly those with chronic pulmonary or heart disease, renal disorders or diabetes, where any complication is likely to be more serious. A recent estimate of the mortality among the elderly in England and Wales, based on calculations of excess mortality, suggests that in each winter between 1967 and 1973 an average of about 11,000 elderly people died directly or indirectly from the effects of influenza (Clifford et al., 1977). Nevertheless, the vaccines are not a panacea, for not only do they have a tendency to toxicity, but the immunity which they confer is transient.

The principal vaccine currently in use (Influvac or Fluvirin) contains inactivated surface antigen influenza virus which in 1986/87 is prepared to cover A/Philippines/2/82 (H3N2), A/Chile/1/83 (H1N1) and B/USSR/100/83.

Specific chemotherapeutic agents have been developed for the prophylaxis and treatment of influenza. Amantadine (Symmetrel) is thought to impair the uncoating of a virus once it has entered the host and may also impair viral penetration of the host cell wall. It is notably effective against influenza A.

Treatment of the established case is along general lines and consists of rest, analgesics and, in severe cases, prophylactic antibiotics. Local nasal treatment is not advocated.

**Rhinitis of the exanthems**

In measles, scarlet fever, pertussis and the enteric group, typhus, smallpox and chicken-pox, an acute rhinitis occurs in the prodromal and early stages. The local condition does not differ from that described in the common cold.

Differential diagnosis depends on the associated specific signs and symptoms. Secondary bacterial rhinitis is common, often very severe and of suppurative type, and complications are more frequent than after the common cold.

**Specific rhinitis**

**Acute nasal diphtheria**

**Definition**

An acute infective rhinitis caused by *Corynebacterium diphtheriae*.

**Clinical picture**

Nasal diphtheria may be primary or secondary to the faucial form. In the latter case it indicates a severe attack. There is often a transient simple rhinitis in the early stage of faucial diphtheria, but no membrane forms and it passes off in a few days.

The acute form differs from the chronic form described later in the short duration, pyrexia and general toxaemia, adenitis and subsequent paralysis. In the UK immunization has
practically eliminated diphtheria but the occasional case might arise from immigrants who have not been immunized (see Chronic diphtheric rhinitis).

**Treatment**

*C. diphtheria* is sensitive to penicillin, and a course of 4 or 5 days' intramuscular and local penicillin should be given in addition to the full doses of the antitoxin intravenously. Antitoxin neutralizes the toxins, while penicillin shortens the disease but does not neutralize the toxins.

There is a tendency for *C. diphtheriae* to persist in the nose for weeks after such an attack. Isolation should be continued until the cultures from three successive daily swabs have been negative.

**Acute syphilis**

The condition of acute syphilis is discussed under the heading of Nasal syphilis.

**Erysipelas**

In erysipelas of the external nose, the nasal mucous membrane may become secondarily infected by the streptococcus from the skin. The infection responds rapidly to penicillin.

**Glanders**

Acute glanders differs from the chronic form described below only in the rapidity of onset and the severity of both local and general manifestations. There is marked fever and prostration, and a pustular rash develops resembling smallpox. The nasal mucosa is greatly swollen, and later ulcers form and may destroy the septum and turbinates. The lymph glands are swollen inlamed, and may suppurate. Death usually follows within a few weeks.

**Diagnosis**

Glanders is most likely to be confused with smallpox, typhus fever, erysipelas, impetigo or syphilis.

**Anthrax**

Primary anthrax of the nose with malignant pustule formation has been described.

**Candidiasis (moniliasis)**

This subject is discussed below.
Gonorrhoea

Rhinitis caused by infection with *Neisseria gonorrhoeae* is certainly rare. Unlike the conjunctivae the nasal mucous membrane has a high resistance to this infection. One or two doubtful cases of purulent rhinitis in infants have been said to be caused by gonorrhoea, but their authenticity has been doubted. The infection responds to penicillin or to co-trimoxazole (Septrin).

**Local irritants and trauma**

In this group there is a simple catarrhal reaction in the nasal mucous membrane with particularly severe irritation amounting in some cases to actual neuralgic pain in the nose and face. Sneezing and copious watery discharge are important features. The reaction follows immediately on the exposure and persists while that lasts. In most cases it passes off rapidly unless the causative agent has produced some destruction of the epithelium, in which case regeneration and healing may take some days before it is complete. The period of recovery depends on the severity and degree of subsequent secondary infection.

**Chronic infective rhinitis**

There are many forms of chronic rhinitis and not a little confusion has arisen from the fact that the term has been taken by different authorities to include different conditions. In the present section the accent has been laid on 'infection', and the conditions referred to are either the result of, or associated with, the latter.

**Atrophic rhinitis**

Atrophic rhinitis is a chronic nasal disease characterized by progressive atrophy of the mucosa and underlying bone of the turbinates and the presence of a viscid secretion which rapidly dries and forms crusts which emit a characteristic foul odour sometimes called ozaena (a stench). There is an abnormal patency of the nasal passages.

**Aetiology**

The aetiology of atrophic rhinitis is still unknown. In the past numerous organisms have been cited as the cause, among which are *Coccobacillus* (Loewenberg, 1894), *Bacillus mucosus* (Abel, 1895), *Cocobacillus foetidus ozaena*, diphtheroid bacilli, and *Klebsiella ozaenae* (Henriksen and Gundersen, 1959). It is true that these organisms may be found in cultures but there is little evidence that they cause the disease.

Other factors which have been regarded as possible causes are chronic sinusitis, excessive surgical destruction of the nasal mucous membrane, and syphilis.

Atrophic rhinitis usually commences at puberty and is much more common in females than males; thus it is generally accepted that endocrine imbalance may play a part. Heredity is an important factor and there appears to be a racial influence in that the yellow races, Latin races and American Negroes are relatively susceptible, whereas the incidence is low in natives of equatorial Africa. Poor nutrition is undoubtedly a factor in the development of the
condition and Bernat (1965) considers that atrophic rhinitis is an iron-deficiency disease. Recently, immunologists have considered atrophic rhinitis to be an autoimmune disease. Fouad et al (1980) studied cellular immunity in patients with atrophic rhinitis using the leucocyte migration and spontaneous rosette tests in vitro and confirmed that there was an altered cellular reactivity or loss of tolerance to nasal tissues, which they considered might be precipitated primarily by virus infection, malnutrition and/or immunodeficiency which trigger a destructive autoimmune process with the release of antigen(s) of nasal mucosa into the circulation.

Pathology

Most authors agree that there are patches of metaplasia from columnar ciliated to squamous epithelium, that there is a decrease in the number and size of the compound alveolar glands, and that there are dilated capillaries; but some (Taylor and Young, 1961) were unable to demonstrate endarteritis and periarteritis of the terminal arterioles. It is possible, therefore, that there are two types of atrophic rhinitis:

1. type 1, characterized by endarteritis and periarteritis of the terminal arterioles, which is the result of chronic infection and which might benefit from the vasodilator effect of oestrogen therapy

2. type 2, in which there is vasodilatation of the capillaries, which might be made worse with oestrogen therapy.

It seems likely that in the past the majority of cases were of type 1.

Taylor and Young (1961) also found that the endothelial cells lining the dilated capillaries had more cytoplasm than normal capillaries and showed a positive reaction for alkaline phosphatase which suggested the presence of active absorption of bone which is a feature of atrophic rhinitis.

Clinical picture

The presenting symptoms are most commonly nasal obstruction and epistaxis. Anosmia may be present and the patient is often only made aware of the loathsome effluvium surrounding her by the reluctance of others to come within her vicinity. Sometimes the symptoms are mainly pharyngeal and are caused by the pharyngitis sicca which often accompanies the condition or by choking when detached crusts slip from the nasopharynx into the oropharynx.

Clinical examination of the morose and dejected patient confirms the presence of foetor in all but the earliest cases and the nasal cavities are found to be lined with green, yellow and black crusts. Even before the removal of the latter the enormous capacity of the nasal passages is apparent and their detachment reveals a bleeding and ulcerated mucosa and shrivelled turbinates.
Investigations

Before embarking on treatment it is advisable to exclude the presence of sepsis in the paranasal sinuses by radiology, and if necessary by proof puncture. Swabs from the nasal secretions may be cultured, but while of interest, the results are unlikely to be of great value in the management of the case. Serological tests to exclude syphilis are essential as syphilis is certainly the most likely condition to be confused with atrophic rhinitis. The blood picture, serum proteins and iron should also be checked.

Treatment

Conservative

In the first place the patient should be instructed to douche the nose twice daily with an alkaline solution prepared by dissolving in 280 mL (1/2 pint) warm water a teaspoonful of the following powder:

- sodium bicarbonate 28.4 g
- sodium diborate 28.4 g
- sodium chloride 56.6 g.

Regular nasal cleansing is the basis of the conservative treatment in atrophic rhinitis and it may be of some consolation that, if the patient is prepared to carry out this simple treatment with unfailing regularity, freedom from offensive effluvia may almost always be achieved.

Following the removal of crusts by forceps or suction it is customary to apply either 25% glucose in glycerin, which inhibits the proteolytic organisms, oestradiol in arachis oil 10,000 units/mL or Kemicetine antiozaena solution (each mL containing chloramphenicol 90 mg, oestradiol dipropionate 0.54 mg, vitamin D₃ 900 IU, propylene glycol). The use of potassium iodide by mouth with the object of increasing nasal secretion has been suggested. Autogenous vaccines may be given. Sinha, Sardana and Rjvanshi (1977) have reported promising results using tissue therapy with systemic human placental extracts, which gave an 80% improvement in 2 years, and submucosal intranasal injection of human placental extracts which produced 93.3% relief over the same period of time.

Surgical

Numerous attempts to relieve the condition surgically have been made in the past. These include submucous injections of paraffin, and operations aimed at displacing the lateral nasal walls medially (Lautenslager’s operation). More recently Teflon strips, polythene and cartilage have been inserted after flaps of mucoperichondrium were raised from the septum or mucoperiosteum from the floor or lateral walls. Wilson (1964) has reported good results from the submucosal injection of a suspension of powdered Teflon in 50% glycerin paste. Chatterji (1980) reports successful results using autogenous medullary (cancellous) bone graft as a single long piece of bone.
Repeated stellate ganglion blocks have been employed with some success by Sharma and Sardana (1966) who advocate cervical sympathectomy or blockade as a possible first line of treatment. Previously, however, autonomic surgery for atrophic rhinitis had proved disappointing.

Encouraging results have been obtained following the closure of one or both nostrils by plastic surgery (Young, 1967). Young's method is to raise folds of skin inside the nostril and suture the folds together with the object of complete interruption of the air flow. After periods varying from months to several years the nostrils have been reopened revealing absence of crusting and normal mucosa. Sinha, Sardana and Rjvanshi (1977) found that bilateral closure was not tolerated by some patients who disliked mouth-breathing and a nasal voice. However, partial nostril closure leaving a 3 mm hole was well tolerated and gave similar results with no recurrence of disease over a 2-year period. Any further increase in size of the hole rapidly decreased their success rate.

**Rhinitis sicca**

Rhinitis sicca is the term often reserved for a dry, mildly atrophic anterior rhinitis which does not progress to the full clinical picture of atrophic rhinitis described above. The causes are not well defined but it is generally recognized that the condition occurs in alcoholism, anaemia, nutritional and constitutional diseases and in those engaged in dry, hot and dusty occupations.

The pathology resembles that of early atrophic rhinitis; indeed some authorities would not distinguish the two as separate entities. There is deficiency and inactivity of the seromucinous glands, metaplasia of the columnar ciliated epithelium to cuboidal or squamous epithelium and deficiency of the mucous blanket. A penetrating ulcer of the anterior part of the cartilaginous septum may be present.

The patient complains of discomfort, irritation and sometimes epistaxis and crusting but the crusts are thin and dry and do not as a rule extend to the posterior part of the nasal cavities as do the crusts of atrophic rhinitis; neither do they emit a characteristic fetor.

Clinical examination reveals a dry, whitish or glazed mucous membrane sometimes accompanied by crusting or complicated by a septal perforation.

As in atrophic rhinitis the patient should be investigated with a view to excluding nutritional deficiencies or local infection.

In treating the disorder all possible causes should be removed and if necessary iron and vitamins administered. Locally, douching with the solution advocated for the treatment of atrophic rhinitis is undoubtedly of value, but the time-honoured treatment with oily drops and sprays is to be deprecated owing to the danger of inhalation lipoid pneumonia and paraffin granuloma. These sinister conditions have been recognized for a number of years and their pathology is clearly described by Spencer (1968).
Rhinitis caseosa

Rhinitis caseosa (nasal cholesteatoma) is a chronic inflammation of the nose associated with the formation of granulation tissue and an accumulation of offensive cheesy material resembling cholesteatoma.

The condition is rare and is usually unilateral, although bilateral involvement has been reported. It is slightly more common in males and can occur at any age. The cause of rhinitis caseous is unknown but numerous theories have been advanced including those of tubercle, syphilis, cholesteatoma and polyp degeneration. The most widely accepted explanation is the theory of suppurative rhinitis complicated by obstruction where rhinitis caseosa is a secondary condition symptomatic of an underlying primary nasal abnormality (for example rhinoliths, deviated nasal septum, inflamed turbinates or polyps) which tends to interfere with the egress of discharge from the nose. There may be coexisting sinus infection.

Microscopical examination of the caseous debris shows keratinous material, numerous organisms and sometimes cholesterol crystals. The lining mucous membrane shows chronic inflammatory changes.

Clinical examination in the early stages merely reveals that one side of the nose is filled with whitish debris but later the bone is invaded, the soft tissues of the face are inflamed and abscesses may form and burst through the skin.

Careful investigation by means of radiology and histological examination is necessary to exclude the presence of coexistent conditions such as sinus infection or malignant disease, and treatment consists of thorough removal of the debris by scooping it out followed by repeated irrigation to ensure its complete removal. Any obstructive lesions should be corrected surgically. Surprisingly perhaps, the prognosis is extremely good provided that care is taken to follow-up the patient and deal with any signs of stagnating discharge.

Gangosa

Gangosa (rhinopharyngitis mutilans; gangraengosa; kaninloma) is a slowly progressive ulceration and necrosis of the palate, nose and pharynx. As a disease it appears to be a separate entity but it may be clinically indistinguishable from tertiary yaws (see Yaws); thus there may arise a certain amount of confusion.

The geographical incidence of the specific form is limited to the pacific Islands, Sri Lanka and equatorial Africa. Gangosa affects males and females of all ages and is associated with dirty and insanitary conditions. It is extremely rare in the white races but has been reported. The cause and mode of spread are unknown; no specific organisms have been found in the tissues or in the discharge.

The disease commences as a small painless nodule in the middle of the palate. This perforates into the nose and spreads intermittently destroying all structures including the nose, palate, orbit and its contents and even the entire face. Pain is absent.
The disease may be steadily progressive, or may be arrested at any stage, the resulting scars resembling those of burns. Most cases survive (Arrowsmith, 1921; Myerson, 1933). Serological tests for syphilis are negative and there is no response to antisyphilitic treatment.

**Nasal syphilis**

Nasal disease secondary to infection with *Treponema pallidum* can occur in every age group from the neonate to the elderly. The disease is no longer common and the signs and symptoms in the early stages may be difficult to elicit, particularly if antibiotics have already been given.

The histological appearances of the syphilitic lesion are characterized by oedema, and infiltration of the stroma with lymphocytes, plasma cells and endothelial cells. The perivascular cuffing by these cells and the endarteritis reduce the lumen of the blood vessels, and result in necrosis and ulceration.

**Primary syphilis**

The lesion of primary syphilis can appear on the external nose or inside the vestibule. It presents as a hard, non-painful ulcerated papule that is often associated with an enlarged rubbery non-tender node some 3-4 weeks after contact. There may be malaise with a pyrexia. The lesion usually disappears spontaneously in 6-10 weeks. It has to be differentiated from malignant neoplasms and furunculosis. Malignant neoplasms are progressive, and occur in the later age groups. Furunculosis is painful and suppuration follows.

The following will be useful in establishing a diagnosis:

1. cultures from the surface of the lesion will be negative
2. smears examined by dark-ground illumination or after staining should show the spirochaete, *Treponema pallidum*
3. serological tests for syphilis may be positive, except in the earliest cases, or in those cases already having antibiotics; serological tests in current use include: (a) Venereal Disease Reference Laboratory (VDRL) tires; (b) *Treponema pallidum* haemagglutination test (TPHA); (c) fluorescent treponemal antibody test (FTA); (d) *Treponema pallidum* immobilization test (TPI).
4. a biopsy may be performed in doubtful cases; the microscopical appearances are characteristic.

Owing to its rarity and the fact that the chancre does not present a typical appearance, the diagnosis is often overlooked and may not be suggested until secondary manifestations are seen. The hardness and painlessness of the nodule, with early and great enlargement of the lymphatic glands, should suggest the diagnosis.
General antisyphilitic treatment, with intramuscular penicillin, should be given at once, and the chancre may be cleansed with 1:2000 solution of perchloride of mercury and the surface smeared with 2% yellow mercuric oxide ointment.

Secondary syphilis

The secondary stage of syphilis is the most infectious and symptoms appear 6-10 weeks after inoculation. The most common manifestation is a simple catarrhal rhinitis. Clinically this does not show any special characteristic, except in its persistence. There may be crusting and fissuring of the nasal vestibule.

Secondary syphilis is rarely recognized in the nose, as mucous patches hardly ever occur on such a thin attenuated mucous membrane. The diagnosis is usually suggested by the appearance of other secondary lesions, particularly the development of mucous patches in the pharynx, roseolar or papular rashes, pyrexia and the shotty enlargement of many lymph nodes. The scar of the primary lesion may be visible. Serological tests for syphilis are positive. The response to antisyphilitic treatment is so rapid as to be of diagnostic value.

The condition responds to general antisyphilitic treatment.

Tertiary syphilis

This is the stage most commonly encountered in the nose. The pathological lesion is the gumma, invading mucous membrane, periosteum or bone. The bony portion of the septum is the site most commonly attacked. More rarely the lateral nasal wall, frontal sinus, nasal bones or floor of the nose are invaded. Pain and headache (which is always worse at night), swelling and obstruction are the early symptoms. The swelling may be diffuse or localized, and offensive discharge, bleeding and crusting follow, but the pain is then relieved. The olfactory acuity diminishes. In neglected cases, perforation of the affected nasal wall, and collapse of the bony support of the nose may occur. Ultimately there may be severe scarring, and secondary atrophic rhinitis.

The earliest stage of simple swelling is not often seen. Later there is a diffuse or localized submucosal swelling, and infiltration. The surface is red, and may be nodular. The lesion is usually unilateral but, if the septum is involved, the swelling is seen on both sides. Tenderness of the nasal bridge is a characteristic sign. As a rule, when first seen, ulceration has already taken place, and a putrid-smelling discharge is escaping from the crusted surface. The crusts should be removed, and bare bone may be felt with a probe. The margins of the ulcers are irregular, overhanging and indurated.

The following are special aids to diagnosis:

1. there is no shrinkage with vasoconstrictors
2. radiographs show rarefaction of bone, with blurring of the cortical outline
3. serological tests for syphilis are positive in 90% of cases
4. biopsy shows the typical syphilitic histological appearances.
This stage has the following complications and sequelae:

1. secondary infection with pyogenic organisms
2. sequestration
3. perforation of the septum, palate or nasal walls
4. collapse of the nasal bridge, and deformity of the face
5. scarring and stenosis of the nasal passages
6. atrophic rhinitis
7. intracranial complications from involvement of the meninges.

**Differential diagnosis**

A gumma should be suspected when there is a firm reddened nodular swelling of the bony portion of the septum or nasal wall, with obstruction, nocturnal pain and tenderness of the nasal bridge. Ulceration, fetor and necrosis of bone practically confirm the diagnosis. Serological tests for syphilis are positive. Other blood changes are absent, and the response to treatment is rapid. In all cases of doubt a biopsy should be performed, as the histological appearances in syphilis and in all the conditions given below are characteristic.

*Yaws* differs from syphilis only in its origin in tropical countries, the onset in childhood by extragenital infection, and the gross skin lesions. Serological tests for syphilis are usually positive and the lesions respond to antisyphilitic treatment.

*Lupus vulgaris* affects mainly the anterior cartilaginous portion of the septum and anterior ends of the turbinates. There may be associated nodules in the skin. Apple-jelly nodules may be seen, and there is no special odour.

In *tuberculosis* the course is rapid, and the skin is not affected. Typical signs of tuberculosis may be present in the lungs.

*Sarcoïd* resembles tuberculosis, but does not caseate; nodules appear in the skin and other organs. There is anergy to tuberculin, and the Kveim-Siltzbach skin test is usually positive.

In *atrophic rhinitis* the fetor is characteristically offensive and nauseating. The mucosa does not ulcerate deeply, and there is no bone necrosis.

*Leprósy* occurs only in certain countries, is painless and develops very slowly. Nodules may be present in the skin, and deformity is severe in the late stages. Areas of anaesthesia may be present. *Mycobacillus leprae* may be seen in the discharge.

*Scleroma* occurs in patients of Central European, Asian, American and African origin. It is slow, painless and does not ulcerate. Stenosis and adhesions are characteristic. Associated lesions are found in the nasopharynx and larynx.

*Chronic glanders* closely resembles tertiary syphilis, but there is an intermittent pyrexia, and *Lefflerella mallei* may be cultured from the discharge.
Leishmaniasis occurs chiefly in South American countries. It commences as a nodule on the septum, which spreads slowly, destroying cartilage, but not bone. It is followed by fibrosis, and scarring. The histology is characteristic, and the Leishman-Donovan bodies can be identified. Response to tartar emetic is rapid.

Benign neoplasms grow slowly, and are painless. Ulceration and bleeding are rare, except in angioma.

A malignant neoplasm is at first unilateral. It grows steadily and ulcerates superficially, and the surface is hard and friable, and bleeds readily on probing. Radiographs show invasion and destruction of bone.

A sequestrum must be distinguished from a foreign body or a rhinolith by probing. The first is always attached deeply at some point, the second and third can always be moved, if only to a slight extent. When bone necrosis is present, only the silent form of osteomyelitis requires to be excluded. In this the swelling is more diffuse; it is associated with sinusitis, there are general signs of infection and a leucocytosis. Radiographs show the typical worm-eaten appearance of the bone.

A septal perforation caused by a gumma is situated posteriorly on the vomer or ethmoid. When due to rhinitis sicca, trauma, lupus vulgaris, leprosy or chrome ulceration it affects the anterior cartilaginous portion.

Treatment

General treatment

General antisyphilitic treatment is given.

Local treatment

The nasal passages must be cleared of crusts and discharge by copious alkaline douches every morning, and repeated if necessary two or three times a day. Dilute mercuric nitrate ointment should be applied freely to the nasal vestibules.

Sequestra should be removed with great care. The free portion may be removed piecemeal, but any firmly attached portion should be allowed to separate naturally, as avulsion may cause severe haemorrhage or damage adjacent tissues. Gummas respond rapidly to general antisyphilitic treatment, but atrophic rhinitis and deformity may persist after the disease is cured.

Perforations of the palate and deformities of the face may be repaired by plastic surgery.

Hereditary or congenital syphilis

In congenital syphilis, any of the lesions described under the secondary and tertiary forms of syphilis of the nose may occur.
In the infant, 'snuffles' is the most common lesion. This begins about the third week of life, but may appear as late as 3 months after birth. At first it appears as a simple catarrhal rhinitis. In a short time it becomes purulent, with secondary fissuring and excoriation of the nasal vestibule and upper lip. The obstruction may be so severe as to interfere seriously with suckling and nutrition.

Gummatous and destructive lesions occur most commonly at puberty in the 'latent' form of the disease. Mucous membrane, periosteum and bone may all be affected. The resulting ulceration and destruction lead ultimately to atrophy of the mucous membrane, secondary atrophic rhinitis, and sinking of the nasal bridge, producing the saddle-nose deformity. Serological tests for syphilis of the patient and parent are positive; biopsy shows the characteristic syphilitic histological picture.

There may be a prenatal and family history of syphilis, miscarriages or stillbirths. Snuffles should be suspected when a severe rhinitis with excoriation of the nares develops about the third week of life, and interferes with suckling. A common cold infection at this age may often produce a severe rhinitis, but there is usually a definite history of exposure to infection; serological tests for syphilis are negative, and cultures may show virulent pyogenic organisms. When obstruction dominates the picture congenital atresia of the choanae or adenoid hypertrophy must be excluded by sounding the nasal passages with a soft catheter.

In the tertiary form, the diagnosis rests on the presence of other stigmata, particularly Hutchinson's incisors and Moon's molars, interstitial keratitis, corneal opacities, sensorineural deafness and the serological reactions.

Treatment

In snuffles the airway must be restored for suckling. The discharge is removed by gentle suction and irrigation and drops of 0.5% ephedrine in normal saline solution should be inserted into the nose, with the head hyperextended, before feeding.

In the tertiary forms simple nasal toilet by syringing with isotonic alkaline douche solution will remove the crusts and discharge, and yellow mercuric oxide ointment may be applied frequently to the nasal vestibules.

In both forms antisyphtillic treatment is essential and rapidly arrests the disease, but the destruction and deformity remain.

Tuberculosis

Tuberculosis of the nose is very rare. It may be nodular or ulcerative. It affects the cartilaginous portion of the nasal septum, and has been reported on the lateral nasal wall. It may be primary (Havens, 1931) but is usually secondary to tuberculosis of the lungs.

The symptoms are discharge, slight pain and partial obstruction. On examination a bright red nodular thickening, with or without ulceration, is seen on the septum. Tuberculosis follows a relatively rapid course, and ulceration leads to perforation of the septum.
Bacteriological examination of the discharge shows tubercle bacilli, and biopsy will confirm the typical appearance of tuberculosis.

Nasal douches may be used to remove the discharge and crusts. Treatment is with antituberculous drugs (rifampicin, ethambutol, isoniazid, pyrazinamide, streptomycin, PAS) in a planned schedule for at least 6 months.

**Lupus vulgaris**

Lupus vulgaris is an indolent and chronic form of tuberculous infection which affects the skin and mucous membrane.

It is twice as common in females as in males, and is developed most often in early adult life. It is a disease mainly of northern climates, and is rare in the tropics. The mucocutaneous junction of the nasal septum is the most common site of inoculation as this is frequently exposed to trauma in patients who have the habit of picking the nose. The nasal lesion is frequently associated with, or a precursor of, nodules on the face.

Sections of tissue show the characteristic appearance of a tuberculous granuloma. In the centre, at first, there is a collection of reticuloendothelial cells which soon necrose and coalesce. Around this necrotic centre there is a ring of living reticuloendothelial cells, and around this ring are lymphocytes, plasma cells and fibroblasts; scattered throughout the tubercle are found giant cells, with a peripheral arrangement of nuclei.

The early symptoms are those of nasal discharge and obstruction followed by crusting and occasional epistaxis. When the ulceration is established there may be slight fetor and soreness. Ulceration may be followed by fibrosis and contraction, with distortion of the alae nasi. When the turbinates are extensively involved the ciliated epithelium is not renewed and atrophic rhinitis may develop.

The course is very slow, and may last for a lifetime with periods of regression and healing, alternating with periods of active extension, depending to a great extent on the general health of the patient.

The typical early lesion is a reddish firm nodule at the mucocutaneous junction of the nasal septum. In more advanced cases, there may be extensive involvement of the floor of the nose and the turbinates, spreading backwards from the primary site. The surface shows superficial ulcers and crusts. The septum may perforate, but only in the cartilaginous portion, and there is no sinking of the nasal bridge.

If the disease spreads forwards there may be external scarring and distortion of the nasal vestibule, tip and alae nasi, and nodules may be seen in the skin of the face.

Blanching, bacterial examination and biopsy are of use in diagnosis.

(1) To show apple-jelly nodules, the blood is expressed from adjoining tissues by pressure with a glass slide on the skin, or shrinkage with cocaine and adrenaline on the mucous membrane, thus making the pinkish lupus nodules more evident by contrast.
(2) Bacteriological examination of the discharge may show tubercle bacilli.

(3) Biopsy will confirm the typical histological picture. For differential diagnosis, see above under Tertiary syphilis.

Complications

(1) Pulmonary tuberculosis develops in a small proportion of cases.

(2) Dacryocystitis, corneal ulceration, nasopharyngeal lupus and lupus of the face may occur.

(3) Atrophic rhinitis may be a sequel.

(4) Epithelioma may develop in the infected tissue.

Sudden increase in size and hardness of one area and, in the elderly patient, an increased tendency to bleed should arouse the suspicion that a malignant change has supervened. A biopsy should be taken, and the tissue examined histologically.

Treatment consists of specific antitubercular therapy and calciferol (vitamin D₃) 150,000 units daily for 6-9 months. Plastic repair of deformities of the nose may be required when the disease has been arrested.

Sarcoidosis (Boeck's sarcoid)

Sarcoidosis is a chronic systemic disease of unknown cause which is clinically characterized by involvement of virtually every organ with a non-caseating granulomatous inflammation closely resembling tuberculosis without caseation. The tubercle consists of a collection of pale-staining epithelioid cells, sometimes surrounded by a thin layer of lymphocytes. Giant cells are present and, in older lesions, contain asteroid intracytoplasmic inclusion bodies which stain with haematoxylin (Schaumann Bodies). This histological picture is not, however, specific for sarcoidosis as it may be seen in other granulomata, for example tuberculosis, leprosy or berylliosis. Before confirming the diagnosis it is therefore important to exclude these other causes.

Nasal sarcoidosis was first described by Boeck (1905), and confirmed by biopsy by Kistner and Robertson (1938).

Aetiology

Two hypotheses have been advanced (Gordon et al, 1976):

(1) that sarcoid is a special form of tuberculosis which is the result of an altered bacillus with an atypical host response. Tuberculosis is known to precede, occur with or follow clinical sarcoidosis. However, tubercle bacilli have been reported in only a few cases.
(2) That an unidentified organism or agent is responsible, for example pine pollen, wood dust, beryllium and silica or tubercle bacilli, M. leprae, a protozoon, virus or fungus.

Incidence

Sarcoidosis occurs all over the world but is more prevalent in rural south-eastern USA and Scandinavian countries. Coloured races are more affected than white, and females more than males. Nasal sarcoidosis occurs in 3-20% of systemic cases. The median age of onset is 25 years, and 50% of cases occur below the age of 30 years.

Clinical picture

Presenting symptoms include nasal discharge, which ranges from serosanguineous to mucopurulent, nasal obstruction and epistaxis. There may be a secondary sinusitis, a result of superadded infection or involvement by the disease. Nasal skin and bone lesions are asymptomatic. There may be a general swelling of the bridge of the nose with discoloration of the overlying skin (Black and Munro, 1966).

Examination of the nasal mucosa may reveal tiny, 1 mm, yellow nodules surrounded by hyperaemic boggy mucosa. Alternatively the mucosa may be dry, ulcerated and covered with crusts. The anterior septum and inferior turbinates are the most commonly involved areas and adhesions may develop between them, resulting in stenosis of the anterior nares. Septal manifestations may arise spontaneously or may appear following septal surgery in the unrecognized case. Nasal skin lesions appear as elevated, yellowish, dry, scaling, discrete nodules or plaques. These may coalesce to form large bluish-red granulomata, separated by normal skin, over the tip, alae, columella or dorsum. Violaceous, diffuse bulbous affliction of the nasal tip area in conjunction with other skin and pulmonary lesions was separately described by Besmer in 1889 as Lupus pernio (Gordon et al, 1976) and is a manifestation of chronic multisystem sarcoidosis (James, 1959). Weiss (1960) believed that skin and mucosal lesions are complete, separate independent lesions.

Nasal lesions may be associated with other lesions in the head and neck which may include tonsil, tongue, salivary glands, lacrimal glands, bronchial mucosa, paranasal sinuses, nasopharynx, or larynx. Heerfordt's syndrome describes a transient bilateral facial palsy associated with fever, parotid enlargement and uveal tract disease.

Diagnosis

The nasal mucous membrane, particularly in the region of the anterior septum and inferior turbinates, can be easily biopsied and will produce valuable diagnostic material. The histological picture is described above. Culture and stains for acid-fast bacilli and fungi should be negative. There is usually an anergy to the tuberculin skin test, but pulmonary tuberculosis develops during the course of the disease in 10% of cases and tuberculin hypersensitivity then develops. The Kveim-Siltzbach skin test (in which a subcutaneous injection of a suspension from a lesion in another case is followed by the development of a sarcoid nodule) is usually positive in all mucosal cases, and in 75% of patients with active sarcoidosis. It is an invaluable aid in the differential diagnosis of granulomata of the nose.
The radiographic changes in cases of involvement of the nasal bones are characteristic and consist of cystic, punched-out lesions with thinning of the cortex of the bone and a reticular pattern in the medulla (Curtis, 1964). Hilar node involvement is shown in radiographs of the chest, and bone cysts are seen in radiographs of the hands and feet.

Serum and urinary calcium levels should be measured to exclude hypercalcaemia. Serum immunoglobulin, particularly IgG, and the erythrocyte sedimentation rate (ESR) may be raised and a mild anaemia, leucocytopenia and eosinophilia may be present. The serum levels of serum angiotensin converting enzyme, which is secreted by the epithelioid cell granuloma, are evaluated in about 60% of patients with active sarcoidosis (Studdy et al, 1978).

**Differential diagnosis**

The differential diagnosis includes:

1. chronic irritation and foreign body reaction, as in berylliosis

2. infectious conditions, such as tuberculosis, actinomycosis, rhinosporidiosis, leprosy, syphilis, glanders, histoplasmosis and blastomycosis

3. other granulomatous conditions, such as Wegener's granulomatosis

4. acquired immune deficiency syndrome (AIDS) may present with a similar granulomatous condition in the nose.

**Treatment**

There is no specific therapy. Steroids may be used locally or systemically. Local depot steroids (McKelvie, Gresson and Pokhrel, 1968) produce a marked decrease in the size of mucosal lesions, but there is little reduction in the size of lesions in patients with systemic disease. Topical steroid nasal drops are beneficial. Atrophic rhinitis may develop as a result of the disease or secondary to depot steroids. In a series of 53 patients with sarcoidosis of the upper respiratory tract, James et al (1982) found systemic steroids were necessary in 46% of the patients. When steroids alone are insufficient in the management of chronic fibrotic sarcoidosis they can be combined with either chloroquine (adult oral dose of 250 mg on alternate days for about 9 months) or methotrexate (adult oral dose of 5 mg, once weekly for a course of 3 months). The long-term use of chloroquine carries the risk of occasional development of irreversible retinal damage.

**Chronic diphtheric rhinitis**

Chronic diphtheric rhinitis (fibrinous rhinitis) is an inflammation of the nasal mucous membrane, caused by *Corynebacterium diphtheriae*. Diphtheria is now extremely rare in the UK. More commonly a fibrinous rhinitis may be caused by the pneumococcus, staphylococcus or streptococcus and is seen very occasionally in debilitated children.
All the changes of a severe chronic inflammation are seen and on the surface there is extensive necrosis and defoliation of the epithelium. The area is covered with a membrane of fibrin and entangled cells. The fibres of fibrin extend deeply into the submucosa and this accounts for the tenacity of its adhesion, and for the bleeding when the membrane is removed. The corynebacteria and pneumococci cause the formation of an adherent membrane, but staphylococci and streptococci produce only a superficial membrane which can be stripped off easily.

The local symptoms are obstruction, and discharge which is watery at first and later becomes bloodstained and mucopurulent. The course of the disease is slow, and may go on for 3 months, ending in spontaneous recovery. Paralysis, toxaemia and other general symptoms are absent.

The anterior nares may be excoriated by the acrid discharge. The nasal mucosa is generally congested and swollen, and the inferior turbinates, floor of the nose and sometimes the septum are covered with a greyish adherent membrane. After removing this a raw bleeding surface remains.

Bacteriological examination of the nasal discharge should never be omitted, and if *C. diphtheriae* is present the organism should be tested for virulence.

Treatment consists of systemic antibiotics and nasal toilet. Systemic antitoxin is unnecessary but, in the past, local application of antitoxin has been found beneficial. In all cases the patient should be isolated.

**Rhinoscleroma**

Rhinoscleroma, or scleroma, is a progressive granulomatous disease commencing in the nose and eventually extending into the nasopharynx and oropharynx, the larynx and sometimes the trachea and bronchi (Friedmann, 1966).

Scleroma may occur at any age and in either sex. It is seen mainly in central and south-eastern Europe, North Africa, Pakistan and Indonesia, Central and South America. It may be seen anywhere in the world and people of any race may be affected. There is, in most patients, one common factor - a poor standard of domestic hygiene.

**Pathology**

Although there is still controversy over the precise pathogenesis of scleroma it is now generally agreed that the causative organism is the Gram-negative Frisch bacillus (*Klebsiella rhinoscleromatis*). That this organism was a secondary invader following an initial filterable virus infection is disputed by the work of Fisher and Dimling (1964), who failed to reveal virus-like particles or inclusion bodies on electron microscopy. Steffen and Smith (1961) successfully recovered the organism from the lungs of mice previously inoculated with *K. rhinoscleromatis*, and Sinha, Pandhi and Prakash (1969) isolated it from 60% of their cases. A complement fixation test based on the reaction of the patient's serum with suspensions of *K. rhinoscleromatis* was described by Levine (1951) and re-evaluated by Toppozada et al.
(1983). They found a high titre of antibodies in the serum of patients with rhinoscleroma indicating that humoral immunity was not impaired. Although they recognized that cross-reactions could occur, they concluded that the complement fixation test was valuable in the diagnosis of early unrecognized cases of rhinoscleroma.

Histologically granulomatous tissue infiltrates the submucosa and is characterized by the presence of an accumulation of plasma cells, lymphocytes and eosinophils among which are scattered large foam cells (Mikulicz cells), which have a central nucleus and a vacuolated cytoplasm containing Frisch bacilli and Russell bodies, the latter resembling plasma cells and having an eccentric nucleus and deep eosin-staining cytoplasm.

Friedmann (1963), in an electron microscope study, observed the transformation of plasma cells into Russell bodies. Further ultrastructural work by Toppozada et al (1981) has demonstrated the different stages of distension of the rough endoplasmic reticulum up to the formation of Russell bodies inside the 'reactive' plasma cell, thus supporting the theory of an intracellular formation of Russell bodies. The histochemical studies of Gonzalez-Angulo et al (1965) indicated a high content of mucopolysaccharides around the walls of the *Klebsiella* and inferred that this may be responsible for the protection of the organism against both antibiotic therapy and the patient's own antibodies.

**Clinical picture**

There are three recognized stages of this chronic and progressive disease.

(1) *The atrophic stage*. Changes occur in the mucous membrane of the floor of the nose, septum or turbinates which resemble atrophic rhinitis, with crust formation and foul-smelling discharge.

(2) *Granulation or nodular stage*. Non-ulcerative nodules develop which at first are bluish-red and rubbery and later become paler and harder.

(3) *Cicatrizing stage*. Adhesions and stenosis distort the normal anatomy. The disease may extend to the maxillary sinus (Mossallam and Attia, 1956; Yassin and Safwat, 1966), the lacrimal sac (Badraway, 1962), the nasopharynx, hard palate, trachea and main bronchi. Spread to lymph nodes has been reported but is extremely uncommon and is thought to be prevented by early fibrous tissue deposition blocking the lymphatics (Badraway and El-Shennawy, 1974). Bone may be extensively involved (Badraway, 1966). Malignant change is uncommon but can occur (Yassin and Safwat, 1966).

**Treatment**

Once the diagnosis has been confirmed by biopsy, treatment must be intense and prolonged in order to eradicate the disease completely. Bactericidal antibiotics in large doses are given for a minimum of 4-6 weeks and are continued until two consecutive cultures from biopsy material are proved to be negative (Ssali, 1975). In practice, the most effective antibiotics are streptomycin and tetracycline. Based on an observation by Rizk (1977) that acriflavine solution *in vitro* kill *K. rhinoscleromatis*, Shaer et al (1981) treated 50 patients suffering from rhinoscleroma with local applications of differing concentrations of acriflavine.
solution over an 8-week period. The 2% solution produced a complete cure of the disease in all its stages after 8 weeks, whereas the 5% concentration caused recurrent epistaxis, vestibulitis and an occasional septal perforation, and the 1% solution failed to cure all cases.

Chemotherapy may be combined with surgery to re-establish the airway without causing further atrophic changes. This is most effectively achieved by discrete removal of granulations and dilatation of the airways combined with the insertion of polythene tubes for 6-8 weeks (Ssali, 1975).

In late cases where the disease has been eradicated plastic reconstructive surgery may be required.

**Leprosy**

Leprosy is a chronic granulomatous disease caused by *Mycobacterium leprae*, an acid-fast bacillus morphologically similar to *M. tuberculosis*. Although *M. leprae* cannot yet be cultured on an artificial medium it can, nevertheless, be inoculated into experimental animals, particularly immunologically deficient mice (Rees, 1966), to produce a disease similar to that in man.

Worldwide, 12-15 million people suffer from some form of this disease which is particularly prevalent in India, Central Africa, and Central and South America.

Three main types of leprosy are recognized (Barton and Davey, 1976).

(1) *Tuberculoid leprosy* in which solitary lesions cause anaesthetic cutaneous 'patches' with involvement of one or more related sensory or motor nerves with possible paralysis of muscles. Cutaneous patches may extend as far as the nasal vestibule but nasal mucosa is not involved bacteriologically or histologically. Isolated cranial nerve palsies (for example, fifth and seventh) may occur.

(2) *Lepromatous leprosy*, in which there is diffuse infiltration of skin, nerves and mucosal surfaces. *M. leprae* tends to favour an environment where the temperature is lower than central (core) temperature. Thus cutaneous infiltration on the face is more apparent on the edges of the pinna, chin, nose and brow. Nasal mucosal involvement occurs early in the disease process and is present in 97% of patients with lepromatous leprosy (Barton et al, 1973). The nasal discharge in these patients, who frequently have minimal systemic signs, contains millions of potentially infectious bacilli and therefore suggests that this is the principal route of spread of infection. Most commonly there is crust formation, nasal obstruction and blood-stained discharge. Hyposmia may be demonstrated in over 40% of patients with lepromatous leprosy (Barton, 1974).

(3) *Borderline leprosy*. The first two types are probably immunologically stable. Patients with borderline leprosy with poor resistance may develop the lepromatous type or less commonly, as the disease is modified by treatment or as immunity is acquired, the tuberculoid type. Skin lesions are more numerous than in tuberculoid leprosy and are frequently seen around the eyes, nose and mouth. In pure borderline leprosy there is no
involvement of the mucous membranes of the nose, mouth, pharynx or larynx. A conversion to the lepromatous type is indicated by the appearance of mucosal involvement.

Clinical picture

With lepromatous leprosy the earliest sign is a nodular thickening of the nasal mucosa which appears paler than normal and often has a yellowish tinge. These isolated nodules most commonly first involve the anterior end of the inferior turbinate. The disease progresses to gross inflammation of the nasal mucosa and severe obstruction and is out of proportion to the almost imperceptible clinical changes of lepromatous leprosy elsewhere in the body. Perforation of the cartilaginous portion of the nasal septum is followed by perichondritis and periostitis of the nasal cartilages, inferior turbinates and anterior nasal spine which leads to the typical nasal deformity. Atrophic rhinitis, fibrotic atresia or stenosis of the airway are typical sequelae.

McDougall et al (1974) have made an extensive histological study of biopsies of nasal mucosa in patients suffering from leprosy. They found no bacilli or evidence of leprosy infection in the septum and turbinates of borderline cases. However, in lepromatous leprosy bacilli were found in macrophages, fibroblasts, within the cytoplasm of endothelial cells of blood vessels and lymphatics and within the free lumina of secretory gland acini.

Diagnosis

Diagnosis of early and intermediate changes in the nose, pathognomonic of lepromatous leprosy, can often be made in the absence of other manifestations of the disease. The presence of atrophic rhinitis and septal perforations is indicative of late disease with other systemic manifestations (Barton, 1976).

Early diagnosis is essential as the nasal component of the infection results in a highly bacilliferous nasal discharge which is the principal route of transmission of the disease (Davey and Rees, 1974). Confirmation is by microscopy of the nasal discharge for acid-fast bacilli, microscopy of scrapings of the nasal mucosa (the most positive site being the anterior end of the inferior turbinate) for acid-fast bacilli, and histology of the nasal mucosa. Radiographs of the anterior nasal spine frequently show erosion (Møller-Christensen, Bakke and Melsom, 1052) and in a study of sinus radiographs the most constant finding was mucosal thickening of the maxillary antra on the occipitomental view (Barton, 1979).

It should always be remembered that, with the current patterns of migration, cases of leprosy may be seen in countries where it is no longer endemic (Barton and Davey, 1976).

Treatment

Dapsone remains the standard drug in the treatment of leprosy and will reduce the bacterial count of nasal discharge to zero or near zero within 2 months; however, there are increasing reports of dapsone resistance. The more expensive drugs, rifampicin (Rifadin) and clofazimine (Lamprene), act more rapidly and reduce the count to zero in 10 days; however, their cost precludes their general usage in developing countries. Local treatment to the nose will help to prevent the external deformity of advanced lepromatous leprosy. Betnovate (1
part) in Unguentum (2 parts) has been used with good results (Baron, 1978, personal communication). In cases of late involvement when the nasal septum has been perforated and atrophic rhinitis is established, careful crust removal is important. The crusts may be softened with a solution of sodium bicarbonate, sodium borate and sodium fluoride 15 g each, dissolved in 500 mL of warm water. After removal of the crusts, the nasal cavities can be painted with a suitable ointment such as Vaseline 1 kg, glycerine 200 g, Vioform 300 g, and crystal violet 5 g (Barton, 1985).

**Yaws**

Yaws (framboesia) is a disease closely resembling syphilis, if not identical with it, and occurs only in natives of the tropics. It is widespread in Central Africa, Jamaica and the Philippines. The causative organism is *Treponema pertenue*, which is indistinguishable morphologically from *T. pallidum*. Transmission of the disease is by direct contact, which is usually extragenital; there is a high incidence in infancy and childhood.

**Clinical picture**

Primary, secondary and tertiary stages occur as in syphilis. Characteristically yaws affects principally the skin and only rarely the mucous membranes, except at the mucocutaneous junctions. Nasal lesions are very rare and do not differ in appearance from those of syphilis. When very extensive and advanced there may be complete destruction of the nose and palate, involving the whole maxilla, face and pharynx. Clinically this is indistinguishable from true gangosa, but in yaws the serological tests for syphilis are positive, and the lesions respond to antisyphilitic treatment.

Another special form is designated 'goundou'. In this there is a chronic osteitis, forming bilateral rounded swellings of the nasal processes of the maxillae, which may encroach on the orbits and destroy the eyes. In the early months there is pain and serosanguineous nasal discharge.

The lesions in the nose are indistinguishable from syphilis. Some authorities consider that the two diseases are identical, but that their manifestations differ in natives of certain areas of the tropics. Differentiation from syphilis is made on the country of origin, the onset in childhood by extragenital infection, the gross skin lesions, and the fact that it is never congenital and that it does not cause quaternary lesions in the nervous system.

**Treatment**

The lesions respond rapidly to treatment with a single large dose of long-acting penicillin. Attention must be paid to general nourishment and hygiene.

**Chronic glanders**

Glanders is a specific inflammatory disease due to infection with *Loefflerella mallei* which is parasitic in horses, donkeys and mules. The infection is extremely rare in man. It occurs in both acute and chronic forms in grooms and others who handle horses. The infection is transferred directly from the horse to the human through an abrasion of the skin,
or occasionally through the nose or mouth. The incubation period may be a few hours, but is usually 2-6 days in the acute form. In the chronic form it may be as long as a year.

**Clinical picture**

The disease is usually ushered in with an acute febrile attack, and in some cases a rash develops which resembles smallpox. After a variable length of time, up to 5 years, during which the organisms lie latent, subcutaneous and intramuscular abscesses appear and nodules develop in the skin and in the mucous membrane of the mouth, palate and nose (Robins, 1906). The nodules ulcerate and later heal, and fresh ones appear and pass through the same stages. The ulceration closely resembles that of tertiary syphilis. In the nose there is also a severe generalized rhinitis, with tenacious mucopus and crusts lying on a reddened and scarred mucous membrane.

Throughout the active stages a variable pyrexia of 1 or 2°C is constant, but periods of complete remission of all signs of the disease are common.

In fatal cases, death is due to toxaemia and pulmonary and intracranial complications. The duration of the disease may be anything from 6 weeks to 15 years. It has been estimated that 6% of cases recover (Robins, 1906).

It is very difficult to distinguish the lesions from those of tertiary syphilis, but in glanders there is usually a characteristic daily intermittent pyrexia, and in syphilis the serology is positive. In the latter condition there is prompt response to antisyphilitic treatment, and characteristic papery scars are left after healing.

Any cases diagnosed as tertiary syphilis with a negative serology, and no response to antisyphilitic treatment, should be suspected as possible cases of chronic glanders. Culture and isolation of *Loefflerella mallei* are often difficult, but intraperitoneal inoculation of the male guinea-pig produces inflammatory changes in the tunica vaginalis of the testis (Straus's reaction). Biopsy may not show any certain points of differentiation.

**Treatment**

The organism is sensitive to the tetracyclines, streptomycin, chloramphenicol and the sulphonamides.

**Pathogenic fungi and yeasts**

The classification and pathology of these diseases is complex and the reader is referred to the superb and exhaustive description given by Emmons et al (1977).

**Rhinosporidiosis**

Rhinosporidiosis is a chronic infestation by the fungus *Rhinosporidium seeberi*, which predominantly affects the mucous membranes of the nose and nasopharynx but occasionally involves the lips, palate, uvula, maxillary antrum, conjunctiva, lacrimal sac, epiglottis, larynx, trachea, bronchus, ear, scalp, skin, penis, vulva and vagina. Osteolytic lesions in the bones
of the hands and feet have been reported by Chatterjee et al (1977). However, rhinosporidiosis is usually limited to surface epithelium but may on occasions be widespread with visceral involvement. The disease, which is chronic and is characterized by the formation of papillomatous and polypoid lesions, tends to affect young males and is endemic in many parts of India and Sri Lanka (Satyanarayana, 1966). Very occasionally the disease has been seen in Europeans who have visited India and Sri Lanka, but it is recognized to be rare for the condition to affect people outside these centres although reports of sporadic cases have come from the USA, Brazil, Africa and Europe. The mode of infection is probably by dust from the dung of infected horses and cattle but this has still to be confirmed.

The characteristic lesion is a bleeding polyp. Histologically the polyp has a vascular fibromyxomatous structure. Throughout the tissue are seen round or oval cells containing the sporangium. The walls of these are of thick chitin, but are thinned at one point where the cells will burst, sporulation will take place, and the spores will spread through the lymphatics into the connective tissue, where they develop into the trophic stage and complete the life cycle.

Epistaxis is often the only symptom, but in the early stages there is a viscid nasal discharge, with irritation and partial obstruction. With the development of the characteristic polyps the obstruction gradually increases until it may interfere with swallowing. Constitutional symptoms are rare, and the disease runs a slow course. The polyps may be present for years before the patient seeks advice.

The lesions are friable, in shape and colour resembling a strawberry, with a greyish undersurface studded with sporangia, showing as white dots. When sessile the polyps appear as multiple nodules, or may assume a leaf shape, with rounded or dentate margins. They arise primarily in the vestibule and are usually attached to the septum, but may spread backwards into the nasopharynx, and even hang down into the oropharynx. The nasal mucosa is generally swollen, hyperaemic and covered with copious viscid secretion, containing spores but no pus cells. The lymphatic glands are not affected.

Microscopical examination of the nasal discharge will show spores. Biopsy and histological examination of the polyps reveals the characteristic appearance described above. At first sight carcinoma may be suspected, on account of the friable masses which bleed on contact. The studding of the undersurface with white sporangia should suggest the diagnosis, and this may be confirmed by the special investigations.

Treatment consists of a combination of medical and surgical methods. The former includes the local injection of depot corticosteroids into the polypoidal masses, and in some cases systemic treatment with amphotericin (Fungizone) has been tried. Recently diaminodiphenylsulphone (dapsone) has been shown to be effective in controlling rhinosporidiosis. Growths are removed by wide excision with the cutting diathermy and cautery to the base, as on occasions excessive bleeding may occur.

The phycomycoses

The phycomycoses are a diverse group of mycoses caused by fungi which are traditionally placed in the class Phycomycetes. Although the term is now rejected by the
formal taxonomic system it is retained in medical mycology (Emmons et al, 1977). It has since been recommended to revert to the name *mucormycosis* for those mycoses caused by fungi belonging to the order Mucorales and the name *entomophtharamycosis* proposed for those mycoses caused by the fungi which belong to the order Entomophtharales. Certain members of each order can produce nasal disease of which the two major conditions are.

*Entomophtharamycosis conidiobolae (rhinophycomycosis)*

This disease is caused by *Conidiobolus coronatus* and is manifested as prominent nasal polyps and granulomata in the nasal cavity. Most cases have been seen in Central Africa, India, Brazil and the West Indies. Males are affected more than females. Symptoms consist of nasal obstruction and swelling over the nose and later the cheek and upper lip. Lesions usually begin in the inferior turbinate and spread in the submucosa through the natural ostia to the paranasal sinuses and to the subcutaneous tissues of the face. Histological examination shows a granulomatous reaction with collections of multinucleate giant cells in the centres of which hyphae can be seen. Treatment consists of removal of the tumour masses and systemic amphotericin (Fungizone).

*Orbital and central nervous system mucormycosis (rhinocerebral phycomycosis)*

This condition is a short-term and often rapidly lethal fungal disease in the nose and paranasal sinuses (Groote, 1970). The principal causative fungi are *Mucor circinelloides, Absidia corymbitera, Mucor javanicus* of the family Mucoraceae and order Mucorales. Because *Mortierella* (order Mucorales) and *Basidiobolus* (order Entomophtharales) have been also identified as a cause, the use of the term 'phycomycosis' was recommended by Straatsma, Zimmerman and Gass (1962).

Phycomycetes are ordinarily saprophytic organisms existing in soil, manure, fruits and starchy food. They can be cultured from the human nose and gastrointestinal tract. They become pathogenic when the patient's general resistance has been altered by metabolic disorders or chemotherapeutic agents. This is most often associated with diabetic ketosis but can be seen with uraemic acidosis, leukaemia, malnutrition; steroid, antimitabolic or antibiotic therapy; and severe burns. The fungus has a remarkable affinity for arteries and by dissecting the internal elastic lamina from the media leads to extensive endothelial damage and thrombosis. Pathologically there is a mixed picture of inflammatory and necrotic changes. Later the veins and lymphatics are involved.

*Mucormycosis* appears in cerebral, pulmonary, ocular, superficial and disseminated forms. Orbital and central nervous system mucormycosis is the most common, and usually commences in the nose and extends by direct extension and intravascular propagation to involve the paranasal sinuses, orbit, cribiform plate, meninges and brain. The most characteristic rhinological finding is a black necrotic turbinate resembling a mass of dried clotted blood. Unilateral gangrene and perforation of the hard and soft palates may occur from involvement of the palatine arteries. Sinus radiographs show thickening of the lining of the sinuses, no fluid levels and spotty destruction of the bony walls.
Early clinical recognition of this potentially fatal disease is essential before irreversible changes occur. The disease is confirmed by biopsy.

Treatment consists of control of the original precipitating condition, heparinization, systemic amphotericin (Fungizone) and local drainage and debridement. Bahadur et al (1983) found long-term use of oral potassium iodide beneficial. The exact mode of action of the drug is unknown but it is believed to have an antifungal property.

**Aspergillosis**

Aspergilloses are infestations, which usually occur in those who handle doves and other small captive birds, in which *Aspergillus fumigatus* and *A. niger* are common, or as secondary infestations during treatment with antibiotics or corticosteroids. Primary paranasal aspergillus granuloma, although rare in the rest of the world, is common in northern Sudan. it can give rise to a pansinusitis and is due to *Aspergillus flavus-oryzae*.

There is a leucocytic and endothelial-cell infiltration, with patchy necrosis and a few giant cells. The symptoms are nasal obstruction, sneezing and watery, mouldy-smelling discharge. On examination the nasal mucous membrane is covered with a greyish (*fumigatus*) or black (*niger*) false membrane. The infection usually also invades the antrum (Tilley, 1915). The course of the disease resembles that of tuberculosis.

It is most likely to be mistaken for diphtheria, syphilis, tuberculosis or atrophic rhinitis; cultural examination of the membrane should determine the diagnosis.

Rarely in debilitated or immunosuppressed patients, acute aspergillosis may become a very aggressive nasal and sinus infection. This results in a relentlessly progressive vasculitis and thrombosis resembling mucormycosis. Extension from the nose and paranasal sinuses can quickly involve the orbit and central nervous system. In these instances the condition can occasionally be fatal.

The specific treatment consists of repeated cleaning and local application of 1% aqueous solution of gentian violet or nystatin. Amphotericin (Fungizone) may be given systemically. When the sinuses are involved, operative clearance should be performed (Adams, 1933).

**Blastomycosis**

Blastomycosis is due to an infection by *Blastomyces dermatidis*, an encapsulated yeast-like fungus, which is practically confined to certain parts of America. The disease starts in the skin, although primary inhalational infection of the lungs may occur in some cases. The nose is rarely affected, but oronasal mucosal involvement is a manifestation of disseminated blastomycosis. The mucosal lesion consists of a papillary hyperplasia with cysts which contain polymorphonuclear leucocytes surrounding the organisms. Regional lymph nodes are not usually affected but dissemination by the blood stream may produce widespread abscesses in the viscera, especially the lungs.

The specific treatment is with amphotericin (Fungizone).
Cryptococcosis

Cryptococcosis is caused by inhalation or ingestion of *Cryptococcus neoformans*, a yeast-like fungus closely resembling but nevertheless distinct from, *Blastomyces dermatididis*. The fungus is found in pigeon or other avian excreta and of the fatal fungal infections in the USA is second only to histoplasmosis (Briggs, Barney and Bahu, 1974). There is, however, a worldwide distribution of the infection, which disseminates after pulmonary infestation to almost any tissue but particularly to the brain and meninges to give a chronic meningitis resembling tuberculous meningitis. Isolated lesions may occur in lymph nodes, skin, bone and eye. Nasal involvement is uncommon but external ulceration, nasopharyngitis and pansinusitis have been described. Briggs, Barney and Bahu (1974) described a case of ulceration of the nasal vestibule, biopsy of which revealed focal ulceration of the squamous mucosa and oedematous submucosa containing numerous round-to-oval yeast organisms surrounded by a clear 'halo-like' space caused by the capsule.

Treatment with amphotericin (Fungizone) and flucytosine (Alcobon) can be monitored by a specific slide later agglutination test. Complete resolution can occur.

Actinomycosis

The genus *Actinomyces* consists of two principal species: *A. bovis*, the cause of actinomycosis or 'lumpy jaw' in cattle; and *A. israelii*, the cause of actinomycosis in man.

The anaerobic fungus, *Actinomyces israelli*, grows in the tissues in the form of colonies composed of branching mycelial threads with clubbed ends - 'ray fungus'. The colonies appear in pus as 'sulphur granules'.

*Actinomyces israelli* is frequently present as a harmless parasite in the mouths of normal individuals, where it is found around the teeth and in the tonsillar crypts. Trauma is an important predisposing factor in the development of 'cervicofacial' actinomycosis, although the exact conditions necessary to cause an infection are unknown. The infection may originate in a tooth socket and spread to adjacent tissues to produce a large hard, woody mass involving the face, jaw and neck. Softening occurs and multiple sinuses may develop, through which the characteristic pus exudes. The nose is rarely the site of primary infection but nasal actinomycosis has occurred following the implantation of xenologous processed bone for atrophic rhinitis (Thomas, Toohill and Lehman, 1974).

The general symptoms are pyrexia, toxaemia and rarely death. There is extensive tissue destruction and scarring.

Treatment consists of penicillin in large doses for 4-6 weeks and surgical drainage.

Candidiasis (moniliasis)

Candidiasis, commonly known as thrush, is caused by the yeast-like fungus *Candida albicans* which is a common inhabitant of the skin and oral cavity.
The infection occurs very commonly in the mouth and occasionally in the nose in neglected and marasmic infants and old people. It may occur in epidemic form in institutions and may be seen as a complication following courses of broad-spectrum oral antibiotics, long courses of systemic steroids and in immunosuppressed patients. There is a predisposition to candidiasis in patients suffering from diabetes, tuberculosis and AIDS.

Candidiasis presents as small, discrete, pearly or dirty-white patches in the mucous membrane on a red moist surface. The patches are easily removed without bleeding.

The condition responds to simple cleansing and painting with 1% aqueous solution of gentian violet or the local application of nystatin; alternatively amphotericin (Fungizone) or flucytosine (Alcobon) may be given in severe cases. Any predisposing cause should be sought and corrected.

**Histoplasmosis**

Histoplasmosis is an infestation due to a yeast-like fungus, *Histoplasma capsulatum*. It is most commonly found in central regions of the USA, but cases have been described throughout the world. Histoplasmosis is a diffuse disease of the reticuloendothelial system and is usually manifest by enlarged spleen, liver and lymph nodes with intestinal ulceration and anaemia. Nasal lesions are rare and may be nodular or infective. Secondary lymphadenitis develops.

The diagnosis is made by biopsy and the histoplasmin skin test which helps to differentiate pulmonary lesions from tuberculosis.

Treatment is with amphotericin (Fungizone).

**Sporotrichosis**

This is primarily an infection of the skin, usually of the hand, caused by *Sporothrix schenckii*. It very rarely affects the nasal mucous membrane but could be transposed there either directly from a lesion on the hand or by haematogenous spread. Infection follows a prick with a thorn. After a few days a nodule develops which becomes red and tender, finally bursting to discharge viscid yellow mucopus in which organisms may be found. Spread is also by the lymphatics along which secondary nodules develop.

Treatment is with iodides or amphotericin (Fungizone).

**Nasopharyngeal leishmaniasis**

This condition, sometimes known as American leishmaniasis or espundia, is caused by *Leishmania brasiliensis* as distinct from *L. donovani* - the cause of visceral leishmaniasis or *L. tropica* - cutaneous leishmaniasis. It is found chiefly in South and Central America and is transmitted by the sandfly (*Phlebotomus)*.
The Leishman-Donovan bodies, which in the mammalian host do not occur in flagellated form, are approximately oval in shape and 2-6 microm in length with an eccentrically placed vesicular nucleus. They may be demonstrated in the discharge from the ulcers and in the reticuloendothelial cells in the granulomatous tissue.

The site of inoculation is usually on the exposed parts where a papule resembling a chancre develops and ulcerates, later healing and leaving a scar. Polypoid growths may form and there may be extensive destructive lesions involving the soft tissues or cartilage of the nasal septum, mouth, pharynx and larynx. Bone is generally not involved. There may be regional lymphadenitis and in untreated cases death follows from exhaustion.

**Myiasis**

Nasal myiasis, which is not uncommon in hot and humid climates, particularly in India where it is known as peenash, is a demoralizing condition of infestation of the nasal cavities by maggots, the larvae of a fly (genus *Chrysomyia*).

Myiasis can also affect the ear, mouth or larynx and reaches a peak in the months of September to November. It can affect any age, and both sexes equally. The majority of sufferers live in bad hygienic conditions and have a source of offensive decaying material, for example atrophic rhinitis, chronic sinusitis or chronic suppurative otitis media, which provides a suitable environment for the eggs of the fly to hatch into larvae no less than 1.5 cm in length. The eggs may also be deposited in a slight abrasion or crack in mucous membrane.

The entomological aspects of myiasis are well described by Sood, Kakar and Watlal (1976). The patient complains of a diffuse swelling around the nose and eyes, nasal obstruction, epistaxis or the presence of maggots coming out of the nose. Rhinoscopy reveals a congested oedematous mucosa, necrotic material with embedded maggots, ulcerated mucosa or septal perforations. The disease can spread to the paranasal sinuses and via the nasolacrimal duct to the lacrimal sac. In the later stages the nasal bones may be destroyed and death may result from sepsis, meningitis or suicide.

Treatment is general, with antibiotics and supportive therapy, and local, with olive oil or liquid paraffin which stifle the larvae. Maggots are removed piecemeal and the nasal cavity is douched. To prevent further infestation the predisposing conditions of poor hygiene and a source of chronic infection must be removed.