Chapter 5: Acute infection of the pharynx and tonsils

J. Hibbert

Although acute infection of the pharynx and tonsils must be one of the most common conditions encountered in medicine, it is one of the most poorly understood. Certain well-defined clinical entities do exist but many others are described for which there is little or no scientific basis. Many difficulties arise and several questions remain unanswered.

(1) Do virus infections in the pharynx and tonsils predispose to bacterial infection?

(2) Is it possible to have an infective condition involving the pharyngeal lymphoid tissue without affecting the tonsils?

(3) Is there such a condition as chronic tonsillitis?

(4) Is there an infective condition called chronic pharyngitis?

(5) Why are some patients susceptible to acute pharyngitis and acute tonsillitis and others not?

(6) Does the tonsil become irreversibly diseased after many episodes of acute tonsillitis.

One of the most misleading aspects of acute infection of the tonsils and pharyngeal lymphoid tissue is the interpretation and importance attached to throat swabs and virus cultures. The presence of an organism in a patient's throat and its culture from a swab does not mean that it is pathogenic. Studies comparing bacteriological isolates from the throat swabs of patients with acute tonsillitis and acute pharyngitis, and from asymptomatic controls, show very little difference (Box, Cleveland and Willard, 1961; Reilly et al, 1981). This particularly applies to aerobic organisms such as streptococci and Haemophilus. Similarly, virus cultures when positive do not imply that the organism is actually causing an infection; conversely patients with a clinically obvious infection may not have a positive culture from a throat swab. It has therefore been suggested that many infections may be caused by anaerobic organisms (Reilly et al, 1981; Toner et al, 1986) or by viruses (Everett, 1979). It has also been suggested that most sore throats are initiated by a virus infection which gives little in the way of signs in the throat and that secondary bacterial infection prolongs the illness and produces more in the way of signs, for example, pus in the tonsillar crypts. Most authorities distinguish between acute pharyngitis and acute tonsillitis, others talk of streptococcal pharyngitis to include infection of both.

In general, acute pharyngitis is most often felt to be an acute viral infection involving the pharyngeal lymphoid tissue and including the tonsil. Acute tonsillitis is reserved for infection with most of the signs seen in the tonsil, but it is likely that the pharyngeal lymphoid tissue is also involved. It seems unlikely that infection of some lymphoid tissue of the pharynx can occur without infection of the remainder. There is no doubt that patients who have had their tonsils removed are still susceptible to infections, both viral and bacterial.
One other area of confusion in this field is the appearance of the throat on clinical examination. It must be accepted that the normal tissues of the posterior pharyngeal wall, the pharyngeal lymphoid tissue and the tonsils are normally pink. In some asymptomatic patients the lymphoid tissue of the pharynx appears abnormally hyperaemic. The appearances of acute follicular tonsillitis are unmistakable and easily diagnosed as abnormal, but many patients with a severe sore throat have little in the way of physical signs in the throat. Thus, the interpretation of the appearances of the pharynx and tonsils is very difficult and inaccurate. Some surgeons claim to be able to diagnose 'diseased or chronically infected' tonsils on inspection. This has never been put to test of a trial and it is the opinion of the author that the appearance of the tonsils is not related to the amount of infection which has occurred. Certainly it has been shown (Weir, 1972) that the size of the tonsils is not related to the frequency of previous infection.

In the present chapter the discussion is restricted to well-defined clinical entities for which there is at least a sound clinical, if not scientific, basis and conditions such as parenchymatous tonsillitis, chronic tonsillitis, streptococcal pharyngitis and chronic hypertrophic pharyngitis will be assigned to a non-proven category.

**Acute tonsillitis**

Acute infection of the tonsils is most frequent in childhood, presumably because immunity to common organisms has not been established. Acute tonsillitis, however, does occur in adults, but one should always be aware of the differential diagnosis and any predisposing factors.

**Causative organisms**

It is on this topic that there is a good deal of controversy because the culture of a particular organism from the throat swab of a patient with acute tonsillitis does not necessarily imply that it is pathogenic. The most common bacterium implicated in acute tonsillitis is the beta-haemolytic streptococcus. Other streptococci may be responsible on occasions, but the role of other organisms such as staphylococci, *Haemophilus* and anaerobic organisms (Toner et al, 1986) is yet to be determined. The part played by viruses in acute tonsillitis is unknown. It has been felt that an initial viral tonsillitis may predispose to a superinfection by bacteria (Everett, 1979) or that viruses alone may be responsible for tonsillitis on many occasions. Certainly a large variety of viruses has been cultured from tonsils (for example, influenza and parainfluenza viruses, adenoviruses, enteroviruses and the rhinoviruses).

**Clinical features**

Prior to onset of sore throat there may be a day or so of a prodromal illness with pyrexia, malaise and headache. The predominant symptom, however, is sore throat which is made worse by swallowing. The voice may change, partly due to the accumulation of saliva but also due to the patient's efforts to restrict movement of the soft palate and tongue. Pain may radiate to the ears or may occur in the neck due to enlargement of the jugulodigastric lymph nodes. In classical acute follicular tonsillitis, the tonsils are hyperaemic and pus accumulates in the tonsillar crypts (it should be noted that an identical appearance may occur in infectious mononucleosis). Rarely, the debris in the crypts coalesces to form a purulent
membrane. Tonsillitis does occur without pus in the crypts and in this case the tonsil is very hyperaemic. Untreated acute tonsillitis will subside over the course of about one week, but with appropriate treatment the illness is shorter.

**Differential diagnosis**

Scarlet fever is a streptococcal tonsillitis in which the streptococcus produces the erythrogenic toxin which results in an erythematous rash. Infectious mononucleosis or acute diphtheria are the conditions most likely to be mistaken for acute tonsillitis and, of course, they do produce an acute tonsillitis. The tonsillitis and pharyngitis associated with leukaemia or agranulocytosis should always be considered.

**Treatment**

Benzylpenicillin 600 mg 6-hourly intramuscularly or ideally intravenously is the most effective treatment for acute tonsillitis. After an initial response it is usual to discontinue the parenteral antibiotic and continue with penicillin V by mouth. More commonly the antibiotic is given only by mouth.

Analgesics should also be given and soluble aspirin gargles are suitable. Many consider paracetamol to be a more suitable analgesic as this avoids gastric irritation in adults and Reye's syndrome in children. In a patient allergic to penicillin, erythromycin (500 mg 6-hourly) should be given. Ampicillin should never be used to treat acute tonsillitis in case the patient has infectious mononucleosis (*see below*).

**Complications**

These are best considered as either local or general factors.

**Local**

Severe swelling with spread of infection and inflammation to the hypopharynx and larynx may occasionally produce increasing respiratory obstruction, although this is very rare in uncomplicated acute tonsillitis. Peritonsillar abscess is one of the complications of acute tonsillitis and its development means that the infection has spread outside the tonsillar capsule.

Spread of infection from the tonsil or more usually from a peritonsillar abscess through the superior constrictor muscle of the pharynx results first in a cellulitis of the tissues of the neck and, later, in a parapharyngeal abscess. Alternatively, such an abscess in the parapharyngeal space can arise following suppuration in a cervical lymph node. Once infection has spread to involve the tissue spaces in the neck it can spread rapidly through these tissue spaces and into the mediastinum. These infections are often due to a number of organisms together (see below) and surgical drainage is as important in the management of the patient as are antibiotics. These neck space infections following acute tonsillitis occasionally occur in fit young patients, but are much more common in debilitated patients with conditions which predispose to infection, for example diabetes and immunosuppressed states such as lymphoma or cytotoxic therapy.
General

Systemic complications of acute tonsillitis such as septicaemia are excessively rare in adults as are the complications of acute glomerulonephritis and rheumatic fever. These are discussed under the section Tonsillitis in Volume 6.

Peritonsillar abscess (quinsy)

A peritonsillar abscess (quinsy) is a collection of pus between the fibrous capsule of the tonsil usually at its upper pole and the superior constrictor muscle of the pharynx. It usually occurs as a complication of acute tonsillitis or it may apparently arise de novo with no preceding tonsillitis. There are a number of interesting and unanswered questions regarding peritonsillar abscess, one being why does it mainly occur in young adults and rarely in children when acute tonsillitis is a disease of childhood. Although a patient with a peritonsillar abscess may have a previous history of recurrent episodes of acute tonsillitis, on occasions, there is no such history and it remains to be explained why the patient should suddenly develop a quinsy.

Bacteriology

The bacteriology of acute tonsillitis and peritonsillar abscess is different and, although one is a complication of the other, it may be that the complication only occurs in the presence of certain organisms. Although a beta-haemolytic streptococcus is the most frequent organism isolated in both acute tonsillitis and peritonsillar abscess, in the latter it is hardly ever isolated on its own. The bacteriology of peritonsillar abscess is characterized by a mixed bacterial flora with multiple organisms, both aerobic and anaerobic, being isolated. A large variety of different organisms is involved (Jokinen et al, 1985) and it may be that the reason a peritonsillar abscess develops as a complication of acute tonsillitis is because of the involvement of anaerobic organisms with the infection spreading through the tonsillar capsule.

Clinical features

The usual patient with a quinsy is a fit young adult who may have a previous history of repeated attacks of acute tonsillitis, however, the patient may never have had tonsillitis previously. Usually a quinsy is preceded by a sore throat for 2 or 3 days which gradually becomes more severe and unilateral. This heralds the development of a quinsy which is almost always unilateral but occasionally can be bilateral. At this stage the patient is ill with a fever, often a headache and severe pain, made worse by swallowing. There may be referred earache and pain and swelling in the neck due to infective lymphadenopathy. The patient's voice develops a characteristic 'plummy' quality as a consequence of the oropharyngeal swelling and an accumulation of saliva in the mouth. Examination reveals an ill-looking patient with pyrexia and often with severe trismus. The classical appearance of the oropharynx is the striking asymmetry with oedema and hyperaemia of the soft palate, and enlargement, hyperaemia and displacement of the tonsil on that side. There are usually tender enlarged lymph nodes in the jugulodigastric region on the same side.
Differential diagnosis

Any condition which produces swelling or oedema of the soft palate may be mistaken for a quinsy. An abscess related to an upper molar tooth is probably the most likely condition to be confused with a quinsy because it will also produce trismus and earache. Any of the causes of a parapharyngeal swelling (see Chapter 21) may also mimic a quinsy, although a carefully taken history and examination will usually make these conditions obvious.

Treatment

The patient should be admitted to hospital and treated with analgesics and antibiotics. In general, the antibiotic should be administered by the intravenous route and benzylpenicillin (600 mg 6-hourly) is the first choice; in those patients allergic to penicillin, erythromycin (500 mg 6-hourly) should be used. Although infection is usually with a mixture of aerobic and anaerobic organisms most are sensitive to penicillin. In a patient with an early peritonsillar abscess which is really a peritonsillar cellulitis, incision and drainage are not to be recommended. It is difficult to be certain when a discrete abscess has formed, but marked bulging of the soft palate, rather than a diffuse oedema, usually indicates this and at this stage incision and drainage should be performed. Failure of an assumed peritonsillar cellulitis to respond to adequate antibiotics within 24 hours is also an indication for incision. This is undertaken at the point of maximum swelling of the soft palate above the upper pole of the tonsil. The mucosa should be anaesthetized with a lignocaine spray and incised with a no. 15 blade with all but the terminal 0.5-1.0 cm guarded using zinc oxide tape. Usually pus will gush out of the incision. A pair of sinus forceps should be introduced through the incision and opened to break down any loculi.

The majority of otolaryngologists advise a patient who has had a quinsy to undergo tonsillectomy at a suitable interval (usually 6 weeks) to avoid a recurrence. The evidence from follow-up studies on patients who have had a quinsy shows that only about 20%, at the most, have a second peritonsillar abscess, so perhaps the policy of tonsillectomy after this condition should be reviewed (Beeded and Evans, 1970; Brandon, 1973; Hold and Tinsley, 1981; Herbild and Bonding, 1981; Tucker 1982b).

The alternative method of managing a quinsy is to perform emergency abscess tonsillectomy. The advantage of this is that incision and drainage are avoided and only one hospital admission is necessary. This assumes that all patients who have a quinsy will go on to need tonsillectomy to avoid recurrence and, as stated above, this may not be necessary. The risks of abscess tonsillectomy are mainly theoretical, namely increased haemorrhage and spread of infection. None of the studies of abscess tonsillectomy shows an increased incidence of these complications (Moesgaard Nielson and Griesson, 1981), whereas cold tonsillectomy at an interval following a quinsy has been shown to have an increased incidence of primary haemorrhage (Kristenson and Tveteras, 1984). However, if the recurrence rate after quinsy is only 20%, then abscess tonsillectomy means that many patients are having unnecessary surgery.
Complications

There is no doubt that a peritonsillar abscess is a potentially lethal condition. Rapidly increasing oedema and spread of infection can lead to pharyngeal and laryngeal oedema with respiratory obstruction, and on occasions tracheostomy is necessary. Spread of infection through the constrictor muscles of the pharynx can lead to a parapharyngeal abscess which may involve the carotid sheath leading to jugular vein thrombosis or even fatal carotid artery haemorrhage. If a parapharyngeal abscess is suspected because of tender swelling in the neck it must be incised and drained through a neck incision. In this situation there is a real risk of a salivary fistula which again puts the carotid artery at risk. However, neglect of a parapharyngeal abscess results in spread of infection and possible mediastinitis.

Parapharyngeal abscess

The parapharyngeal space lies on either side of the superior part of the pharynx - the oropharynx and nasopharynx. It is bounded laterally by the parotid gland and parotid fascia and by the medial pterygoid muscle. Medially, this space is bounded by the pharynx and separated from it by the superior constrictor muscle. Posterior to the pharynx the parapharyngeal space communicates with the retropharyngeal space. Superiorly, the parapharyngeal space is limited by the base of the skull and inferiorly, by the fascia surrounding the submandibular gland. The parapharyngeal space contains the carotid sheath with the internal carotid artery, internal jugular vein, vagus nerve, the styloid group of muscles and last four cranial nerves. It also contains some lymph nodes. Infection can spread to the parapharyngeal space from the retropharyngeal space, the peritonsillar space and from the submaxillary space. The commonest causes of a parapharyngeal abscess are tonsillitis, peritonsillar abscess or a dental infection. Rarely mastoiditis or a pharyngeal foreign body can give rise to a parapharyngeal space abscess.

Clinical features

The symptoms and signs of a parapharyngeal abscess are very similar to those of a peritonsillar abscess except that the maximum swelling is more inferiorly placed and the soft palate is less oedematous. The other striking feature of a parapharyngeal abscess is the tender firm swelling in the upper part of the neck. This allows it to be distinguished from a peritonsillar abscess in which swelling in the neck is due to enlarged lymph nodes rather than an abscess. Of course, if a parapharyngeal abscess complicates a peritonsillar abscess the clinical features are very similar.

Treatment

The patient is treated with intravenous penicillin 600 mg 6-hourly; erythromycin is the drug of choice if the patient is allergic to penicillin. If pus is present in the neck then this must be drained. If this does not seem likely, observation for 24 hours to assess the effects of antibiotic therapy is reasonable. Drainage of a parapharyngeal abscess is not without risk as trismus and pharyngeal oedema make general anaesthesia difficult. If there is any doubt about the capability of the anaesthetist to pass an endotracheal tube then an initial tracheostomy should be performed under local anaesthesia. The abscess is drained through a collar incision in the neck at the level of the hyoid bone. The abscess is widely opened and,
if it has arisen from an adjacent space, this should be opened also. Tracheostomy is performed if there is doubt about the adequacy of the patient's airway. If the infection has arisen from a dental abscess, expert advice should be sought and this should be dealt with at the same time.

Complications

A parapharyngeal abscess can lead to involvement of the carotid sheath with thrombosis of the internal jugular vein or rupture of the carotid artery. Spread of the abscess into the mediastinum can occur if treatment is not instituted rapidly. The possibility of airway obstruction has already been mentioned.

Retropharyngeal abscess

A collection of pus in the retropharyngeal space occurs in three situations. Acute suppuration in a retropharyngeal lymph node following an upper respiratory tract infection in childhood gives rise to a suppurative retropharyngeal abscess. This condition is very rare in adults because these lymph nodes atrophy in adult life; retropharyngeal abscess of this type is considered in Volume 6. Occasionally a foreign body which has perforated the posterior pharyngeal mucosa will give rise to an abscess in this situation. In adults an abscess in the retropharyngeal space is uncommon and nearly always due to tuberculous disease of the cervical spine which has spread through the anterior longitudinal ligament of the spine to reach the retropharyngeal space. This condition is virtually confined to adult patients and is nearly always due to a reactivation of a dormant focus which has arisen from a previous infection of tuberculosis. The previous focus of infection which was almost certainly blood-borne has been controlled by the immune response and the reactivation of the focus must be due to a change in the immune system.

Usually the infection begins as a destructive process involving the intervertebral disc and then the anterior portion of the vertebral body. Histologically, the lesion is a granuloma with caseation and with epithelioid cells, giant cells and a surrounding zone of lymphocytes and fibrous tissue.

Clinical features

In the early stages of the disease there may be few symptoms and little to be seen on examination. Pain often occurs and later the patient may have fever. As the process progresses there may be neurological signs and, occasionally the patient may present with the signs and symptoms of spinal cord compression. The pharynx may appear normal or there may be a marked bulge of the posterior pharyngeal wall. Usually there will be nothing to feel in the neck unless the swelling is huge. Radiology usually shows evidence of bone destruction and loss of the normal curvature of the cervical spine. It must be remembered that the spine may be quite unstable and undue manipulation may precipitate a neurological event. The diagnosis is made by the radiological appearances supplemented by needle biopsy. Surgical drainage of the abscess is not normally necessary but should be carried out through a cervical incision and approach in front of and medial to the carotid sheath. Occasionally exploration of the neck is necessary to obtain biopsy material in order to make the diagnosis. Treatment should be with chemotherapy and at all times expert advice should be sought regarding the stability
of the spine. Spinal fusion is rarely necessary to stabilize the cervical spine. Occasionally surgery is required to decompress the spinal cord if there is a progressive neurological deficit.

**Acute lingual tonsillitis**

This is a rare condition, although it would be surprising if some degree of infection did not occur as part of most episodes of acute pharyngitis and acute tonsillitis. It may well be that this is the case, but the infection gives little in the way of signs and is overshadowed by the more obvious infection of the tonsils or pharyngeal lymphoid tissue. Thus lingual tonsillitis is only usually recognized in patients who have had their palatine tonsils removed. The bacteriology and clinical features of the disease are similar to those of acute tonsillitis except that the sore throat tends to be made worse by speech and protrusion of the tongue and the voice has a more striking 'plummy' quality. The lingual tonsil, best visualized on indirect laryngoscopy, is hyperaemic and pus can be seen in the follicles. Treatment is as for acute tonsillitis as are the complications, except that respiratory obstruction due to swelling and oedema of the base of the tongue and supraglottic larynx is more likely to occur.

**Diphtheria**

Diphtheria is a specific infection caused by the Gram-positive bacillus *Corynebacterium diphtheriae*. In countries with a well-developed immunization programme it is a rare disease (200-300 cases/year in the USA), although it does still occur in epidemics in underdeveloped societies where it carries a mortality of 10%. The disease spreads rapidly in conditions of overcrowding where, in addition, medical care tends to be poor. The disease itself varies in severity depending upon the immunity of the host and also the virulence of the infecting organism. Clinically, it can vary from an asymptomatic carrier state to a rapidly fatal toxic disease.

**Clinical features**

The infection remains localized to the primary site of infection, usually pharynx, larynx and nasal cavities, although in tropical and subtropical countries it gives rise to a cutaneous infection. The systemic effects of the infection are all related to the production of an exotoxin. Spread of infection is by infected droplets of nasal, nasopharyngeal or pharyngeal secretions and the incubation period is 2-6 days. In a host with well-developed immunity to the exotoxin there may be minimal symptoms or none at all, although the organism is still capable of being transmitted. The onset of the illness is heralded by malaise, pyrexia and headache. Anterior nasal diphtheria gives rise to a mucopurulent haemorrhagic discharge with nasal obstruction due to a membrane in the nasal cavity or nasopharynx. Oropharyngeal diphtheria produces a severe sore throat with a greyish-green membrane on both tonsils, posterior pharyngeal wall and soft palate. Tender bilateral cervical lymphadenopathy occurs in the jugulodigastric region. The membrane may spread from the pharynx to the larynx causing rapidly increasing airway obstruction necessitating endotracheal intubation or tracheostomy. Most of the deaths from diphtheria are related to the toxaemia which causes a myocarditis, cardiac conduction defects and arrhythmias producing acute circulatory failure. The exotoxin may also produce a fatal thrombocytopenia.
Neurological complications may appear 3-6 weeks after the onset of diphtheria and give rise to paralysis of the soft palate, diaphragm, external ocular muscles and occasionally a Guillain-Barré syndrome. Patients who recover from diphtheria may show severe scarring of the nasopharynx and oropharynx including the soft palate and the larynx, with fibrous bands and adhesions.

**Differential diagnosis**

The diagnosis of diphtheria is unlikely to be missed if the physician considers it, but it may be confused with streptococcal tonsillitis, infectious mononucleosis, the acute manifestations of leukaemia or agranulocytosis.

**Treatment**

The diagnosis of diphtheria must be made on clinical grounds and cannot await evidence of culture, although microscopic examination of the membrane may be helpful. Treatment involves neutralization of toxin with equine antitoxin (20,000-120,000 units depending upon the severity of the illness) together with benzylpenicillin (600-1200 mg 6-hourly). If the membrane is confined to the tonsils 20,000 units of antitoxin are usually adequate. Higher doses are required if it extends beyond the tonsil. With highly purified antitoxin anaphylactic reaction is rare, but small intramuscular doses can be given, followed in 1-2 hours by full intravenous dose if there is no reaction.

**Immunization**

Active immunization against diphtheria is by injection of toxoid (produced by formalin denaturation of toxin) in three doses beginning at 3 months of age. It is this immunization which has so reduced the incidence of diphtheria.

**Infectious mononucleosis**

This is an acute infection caused by the Epstein-Barr virus which has been isolated from the blood, lymph nodes and saliva of patients with the disease and the latter is probably the mode of transmission. Transmission of the disease to volunteers using the virus has not been accomplished, although there is little doubt that this virus is the causative agent and individuals with antibody to the capsular antigen of the virus will not develop the disease. In general, infectious mononucleosis is a disease of young adults, being very rare in childhood.

**Clinical features**

The clinical manifestations of infectious mononucleosis are variable ranging from an asymptomatic state to a severe systemic illness with hepatosplenomegaly.

The incubation period is of the order of 5-7 weeks and usually there is a prodromal phase of 4-5 days with malaise, fatigue and headache. The most common manifestation of infectious mononucleosis is tender enlargement of cervical lymph nodes (hence the synonym glandular fever) which, in most patients, is accompanied by a sore throat.
The pharyngeal signs are variable. Often there is an acute follicular tonsillitis, indistinguishable from a streptococcal tonsillitis. On other occasions a limited membrane may form in the oropharynx and there may be petechiae on the soft palate. Occasionally enlargement of pharyngeal and base of tongue lymphoid tissue together with a membranous slough may produce progressive respiratory obstruction necessitating tracheostomy.

Pyrexia usually accompanies the severe form of the disease and lymph nodes in other regions may be enlarged. Splenomegaly occurs in 50% of patients and hepatomegaly in 10%. Liver function tests are frequently abnormal in infectious mononucleosis and clinical jaundice occurs in about 10% of patients.

A rubelliform skin rash sometimes occurs and this is almost invariable if ampicillin is mistakenly prescribed for the condition. Ampicillin should therefore never be prescribed for a patient with a sore throat unless it is certain that this is due to acute epiglottitis.

A small proportion of patients may show a periorbital oedema particularly involving the lower eyelid and occasionally this may be mistaken for sinusitis, especially in young patients. Other more rare manifestations of infectious mononucleosis include lesions such as facial palsy, Guillain-Barré syndrome, meningoencephalitis, myelitis, myocarditis, pericarditis, nephritis and pneumonitis. The blood picture in infectious mononucleosis usually shows a leucocytosis of which 50% are mononuclear cells and 10% are atypical with pleomorphic nuclei. Occasionally haemolytic anaemia, an aplastic anaemia or thrombocytopenia will occur.

The diagnosis is made from the clinical picture together with the finding of a mononucleosis in the peripheral blood. The white blood count may be normal in the first week but rises in the second. The common serological tests depend upon the development of heterophile antibodies, the most useful being agglutinins to sheep and horse red cells and these antibodies are the basis of the Paul Bunnell and monospot tests. These tests are usually positive in the first week of the disease although around 10% of patients never develop a positive test. This proportion of negative results may be even higher in children.

**Tonsillar debris and cysts**

Caseous debris may accumulate in the tonsillar crypts and particularly in the supratonsillar cleft. The patient may notice the accumulation and may express it. This debris is of no significance and should be ignored.

Accumulation of debris in a crypt may form a tonsillar cyst. These appear as yellow-coloured inclusion cysts and again are of no significance and can be ignored.

**Unilateral tonsillar enlargement**

It is not unusual for the tonsils to be somewhat different in size, but a gross difference should always be viewed with suspicion, particularly if the patient feels the difference is of recent onset. If the larger tonsil appears abnormal a biopsy should be performed; if it looks normal then observation is indicated. Gross asymmetry with enlargement of the tonsil implies neoplasia, usually squamous carcinoma or lymphoma. Occasionally a peritonsillar abscess will give rise to confusion. When examining a patient with an apparent unilateral enlargement of
the tonsil great care must be taken to exclude a parapharyngeal mass which is displacing the tonsil medially (see Chapter 21).

**Ulceration of the tonsil**

The differential diagnosis of an ulcerative lesion of the tonsil is interesting. In theory many different disorders can give rise to an ulcerative lesion of the tonsil but, in practice, most of these can be excluded on history and clinical examination. Other investigations which may be useful are blood picture, chest radiograph, specific blood serological tests and, ultimately, biopsy. Possible causes are listed below.


2. Infection:
   - (a) acute - acute streptococcal tonsillitis, peritonsillar abscess, acute diphtheria, infectious mononucleosis, Vincent's angina.
   - (b) chronic - syphilis, tuberculosis.


**Acute pharyngitis**

The implication when this diagnosis is made is that the infection appears to be restricted to the pharyngeal lymphoid tissue and that the tonsils if present are not grossly infected. This is almost certainly an over-simplification and it seems more logical to assume that all the lymphoid tissue of the nasopharynx and oropharynx is infected at the same time. The organisms involved are similar to those in a predominantly tonsillar infection except that viruses probably represent a much higher proportion of the infecting organisms, although there is no doubt that streptococcal pharyngitis, for example, can occur in the absence of tonsils.

**Viral pharyngitis**

Many different viruses give rise to a predominantly upper respiratory tract infection which involves a rhinitis, a pharyngitis and, in some cases, a laryngitis. The possible causative organisms are shown in *Table 5.1*.

**Table 5.1 Causative organisms in viral pharyngitis**

- Rhinoviruses
- Coronavirus
- Influenza A and B viruses
- Parainfluenza viruses
- Adenoviruses
- Enteroviruses
- Respiratory syncytial virus.
When nasal infection and symptoms predominate the illness is designated the common cold or coryza. When the symptoms are fever and malaise with pharyngitis, influenza is usually diagnosed. In an individual patient the signs and symptoms do not allow one to predict which virus is responsible. This can only be ascertained by serological tests showing rising antibody titres and this, of course, is rarely of practical importance. Most children and adults will suffer three or four virus infections per year and these often occur in epidemics. Spread of viruses is by droplet infection and once in the upper respiratory tract the virus will enter the epithelial lining cells. It may be prevented from doing this by the presence of specific secretory antibodies in the mucociliary blanket lining the upper respiratory tract, non-specific antiviral mucoproteins or by the mechanical effect of ciliary action. Once within the epithelial cells lining the upper respiratory tract the virus divides and the cells die. The systemic effects of an acute viral illness such as fever, headache, myalgia, arthralgia, anorexia are either a result of haematogenous and systemic spread of the virus or the release of intracellular factors caused by the necrosis of epithelial cells.

Clinical features

The sore throat may be the initial symptom of an acute pharyngitis or it may be preceded by a rhinitis and/or conjunctivitis or by a day or so of malaise, fever and headache. The sore throat is made worse by swallowing and the pain may radiate to the ears. Cervical lymphadenopathy may occur and the clinical manifestations depend upon the nature of the virus and the resistance of the host. Thus the larynx may be involved, producing hoarseness, and the lower respiratory tract may also be colonized producing cough. It is not unusual for acute otitis media of the suppurative or non-suppurative type to be part of the illness. Secondary bacterial infection probably does occur on occasions and this will also affect the clinical picture. Examination of the patient may reveal a rhinitis, with secretion in the nasal cavities and hyperaemic congested turbinates. The nasopharynx may be hyperaemic and the mucosa covered by mucopus. The posterior pharyngeal wall also shows streams of mucopus with hyperaemic islands of lymphoid tissue which may occasionally show pustular follicles. The tonsils may be inflamed as may the larynx. The course of the illness again depends upon the host resistance and virulence of the organism, but usually the disease is self-limiting, lasting for 3 to 4 days.

Treatment

None of these illnesses is sufficiently severe to warrant antiviral agents and so treatment must be symptomatic with bed-rest, analgesics (for example aspirin) and fluids by mouth. If a significant bacterial complication has occurred antibiotics are indicated.

Complications

These are mainly local complications such as sinusitis, otitis media, laryngitis, tracheobronchitis and pneumonia. The laryngitis may occasionally result in sufficient oedema to produce a degree of respiratory obstruction, but this is much more common in children than adults. General complications are rare but include meningitis, encephalitis and myocarditis. The usual cause of death in patients with an upper respiratory virus infection is a viral pneumonia with secondary infection and this is much more likely in elderly or debilitated patients.
**Herpes simplex**

The herpes simplex virus occurs in two forms; type I usually affects the oral cavity and oropharynx and type II generally gives rise to genital infection. Primary infection with the type I herpes simplex virus usually affects children and causes a severe vesicular and ulcerative stomatitis affecting the lips, gums, tongue, buccal mucosa, soft palate and occasionally spreading to the oropharynx. Children with this condition are ill with pyrexia, tachycardia and cervical lymphadenopathy. Diagnosis is normally obvious, although occasionally it can be confused with Stevens-Johnson syndrome (see below), and can be confirmed by isolation of virus from an unruptured vesicle. The virus can be identified using fluorescent antibody or can be seen as an intranuclear inclusion in the scrapings.

Usually, the treatment of primary herpes is non-specific, namely analgesics and fluids, which may need to be given intravenously. Secondary herpetic infection occurs when the herpes virus resides within the posterior root ganglion following a primary stomatitis. Intercurrent illness then results in the appearance of herpetic vesicles usually on the lips or at the angles of the mouth as a typical cold sore.

**Herpes zoster**

Zoster virus is the same virus as that which causes chicken-pox (varicella). Herpes zoster probably arises by the reactivation of virus particles which have remained in the cranial nerve nuclei, ganglia or spinal root ganglion following a previous attack of chicken-pox. Thus it resembles the reactivation of herpes simplex virus in cold sores. Also, however, herpes zoster can occur during an epidemic of chicken-pox. In the pharynx, eruption of zoster can occur in the distribution of the fifth, ninth and tenth nerves. Thus the palate can be affected or the tonsil and posterior pharyngeal wall. This is often associated with herpes zoster oticus (see Volumes 2 and 3) and the pharyngeal manifestations are very transient and easily overlooked. They may give rise to pain on swallowing and vesicles and shallow ulcers, which heal rapidly, may be seen on the soft palate, hard palate, tonsil or posterior pharyngeal wall.

**Vesicular and bullous eruptions of the pharynx**

Herpangina, herpes simplex (less commonly) and herpes zoster may give rise to vesicles in the pharynx which break down and form small ulcers which heal rapidly. Other conditions which may occur are Stevens-Johnson syndrome, pemphigus, pemphigoid and benign mucous membrane pemphigus. These conditions affect the skin and mucous membrane of the mouth and on occasions the oropharynx. Only very rarely is the pharynx involved without involvement of the oral cavity and these conditions are discussed in Chapter 4.

**Other specific viral illnesses**

**Hand, foot and mouth syndrome**

This is an illness probably caused by a coxsackie virus (an enterovirus). The disease is characterized by a vesicular eruption in both the oral cavity and the oropharynx accompanied by vesicles on the hands and feet. There is also usually pyrexia with malaise. The illness is short-lived and self-limiting and mainly affects children.
**Herpangina**

This is a self-limiting vesicular eruption which occurs in the oropharynx and a number of enteroviruses have been implicated. It is distinguished from herpes simplex which is almost always restricted to the oral cavity and very rarely spreads to the pharynx.

**Cytomegalovirus infections**

This virus is usually transmitted by blood transfusion, although it can occur without, especially in immunosuppressed states. It gives rise to an illness which is very similar to infectious mononucleosis. The diagnosis is made by serial antibody levels.

**Other causes of acute pharyngitis**

**Acute gonococcal pharyngitis**

This disease is acquired following orogenital sexual intercourse and the majority of patients who acquire it are asymptomatic. Some may have a transient sore throat and occasionally an exudative gonococcal tonsillitis will occur with an appearance similar to streptococcal tonsillitis or infectious mononucleosis. There will also be tender enlarged cervical lymph nodes. If the diagnosis is suspected the patient should be treated with 4.8 mega units of procaine penicillin given intramuscularly usually with probenecid. A swab should be taken for microscopy and for culture and sensitivity tests. Some gonococci produce a beta-lactamase (penicillinase) and are therefore relative resistant to penicillin treatment. This should be suspected in patients who give a history of sexual contact in the Far East. Thus oral tetracycline should be used for those patients who do not improve with penicillin, and the treatment failures seem to be more frequent in patients with gonococcal pharyngitis than in those with genital infections. The dangers of gonococcal infection are those of septicaemia with septic foci in many organs but particularly in joints and tendons. As well as a septic arthritis, there occurs with gonococcal infections an arthritis which seems to be a hypersensitivity reaction and may be associated with an iritis.

**Oedema of the uvula**

Acute oedema of the uvula (Quincke's disease) is unusual without an obvious precipitating cause. The patient complains of the onset of a tickle or irritation in the throat together with a sensation of gagging. Examination shows oedema of the uvula which on occasions may be very severe indeed. The aetiology is unknown but may be related to an inhaled or ingested allergen. The oedema usually settles down of its own accord (perhaps assisted by an intravenous injection of hydrocortisone) unless it is part of a more serious allergic reaction such as angioneurotic oedema.
Other causes which produce oedema of the uvula are numerous and include:

(1) trauma: foreign bodies, surgery, endotracheal intubation;

(2) infection:
   - acute - peritonsillar abscess, viral pharyngitis, candidiasis;
   - chronic - syphilis, tuberculosis;

(3) tumours: squamous carcinoma;

(4) radiotherapy;

(5) allergic: angioneurotic oedema;

(6) blood diseases: agranulocytosis, acute leukaemia;

(7) miscellaneous: aphthous ulceration, Behçet's syndrome.

**Chronic pharyngitis**

This title implies a long-standing infection or inflammation of the pharynx; classically the disease is divided into specific or non-specific types. Specific chronic infections are due to a well-defined pathological entity, although sometimes the differentiation between acute and chronic is a little blurred. Non-specific pharyngitis is a much more difficult entity to define and diagnose and therefore to treat. It is discussed first.

**Chronic non-specific pharyngitis**

The clinical picture of this condition is the patient who complains of a long-standing discomfort in the throat, pain on swallowing and occasionally earache. This must be distinguished in the history from patients who have recurrent acute episodes. The usual description of this condition is that the patient with the above symptoms has islands of lymphoid tissue on the posterior pharyngeal wall which are hyperaemic and enlarged. Occasionally the term chronic hypertrophic pharyngitis is used. Many normal patients with nothing in the way of throat symptoms have islands of pink lymphoid tissue on their posterior pharyngeal wall, however, and in this situation it may be very difficult to decide what is normal and what is abnormal - laboratory tests such as blood picture or throat swabs are of no value. There are a number of sources of infection which are liable to produce chronic infection of the lymphoid tissue of the posterior pharyngeal wall. One is chronic sinusitis in which puss passes from the nose into the nasopharynx, oropharynx and hypopharynx. Clinically this can usually be seen particularly in the nasopharynx and the diagnosis refuted or confirmed by sinus radiographs and proof puncture. Patients with bronchiectasis or chronic bronchitis are producing infected sputum which can infect the pharynx. Gingivitis or dental caries when very severe may give rise to infected lymphoid tissue in the pharynx. Irritants which cause an inflammatory condition of the pharynx are tobacco smoking and industrial fumes.
When confronted with a patient who complains of chronic sore throat, a history, examination and investigation are designed to exclude patients with a primary carcinoma, chronic specific pharyngitis (see below) and those with chronic non-specific pharyngitis with a predisposing cause as outlined above. With these exclusions a proportion (probably the majority) of patients will still remain undiagnosed. Some of these will be classified as globus pharyngitis (see Chapter 10). The remainder of the patients are often classified, erroneously in the author's opinion, as having chronic pharyngitis. These patients should be followed-up (it is not unusual for a carcinoma to be overlooked) and examined at regular intervals. Some otolaryngologists will prescribe gargles, mouthwashes or other remedies such as Mandl's paint (iodine and potassium iodide in glycerine). There is no evidence that any of these measures is of therapeutic benefit.

**Chronic specific pharyngitis**

These entities are discussed below but not in order of incidence, simply in an order of convenience.

**Syphilis**

This is an infection by the spirochaete *Treponema pallidum* and apart from the congenital form is acquired by sexual intercourse. The disease progresses through primary, secondary and tertiary stages with the secondary most likely to give rise to pharyngeal symptoms. The disease manifests itself in a great many ways and clinical presentations, particularly in the tertiary stage where the presenting symptom may involve virtually any organ system. The lesion of primary syphilis is at the site of the initial inoculation and the organism can penetrate both normal mucosa and mucosal abrasions. The primary pathology in syphilis is an endarteritis with an increase in adventitial cells, a proliferation of endothelial cells and an inflammatory focus of lymphocytes, plasma cells and monocytes. Usually in parts of the lesion healing with fibrous tissue is taking place. In the secondary and particularly in the tertiary phase of the disease a granulomatous reaction takes place with necrosis of tissue and occasional giant cells.

**Primary syphilis**

The lesion is the chancre which develops after an incubation period, about 21 days on average. The most frequent extragenital sites for a chancre are the lips, tongue, buccal mucosa and tonsil. The lesion begins as a papule which breaks down to form a painless ulcer with indurated margins. At the same time there may be a unilateral or bilateral cervical lymphadenopathy and the glands are non-tender. Although the chancre is characteristically painless secondary infection can render it painful. The ulcer persists for a variable period, 2-6 weeks as a rule, and then heals. While the primary lesion is present the individual is capable of transmitting the disease.

**Secondary syphilis**

The secondary stage occurs several weeks (usually 4-6 weeks) after the primary lesion and about 30% of patients in the secondary stage will have evidence of a healing chancre. The features of the secondary stage are fever, headache and malaise with generalized
lymphadenopathy and a mucocutaneous rash and sore throat. The pharynx and soft palate show hyperaemia and inflammation, and may show lesions which have been described as mucous patches or snail-track ulcers. These lesions are most commonly seen in the oral cavity and are ulcerated lesions covered with a greyish-white membrane which when scraped off has a pink base with no bleeding. The secondary stage of the disease lasts a few weeks and here again the lesions in the mouth and pharynx are infectious. About 30% of patients will go on to develop the tertiary stage of the disease.

**Tertiary stage**

This develops some years (5-25) after the initial infection and is characterized by lesions which may be widespread throughout the body or restricted to one or two organ systems. In the upper respiratory tract the manifestations are those of the gumma. This is the granulomatous necrotic lesion which begins as a nodule and then breaks down to form an ulcer. It can occur in the hard palate, the nasal septum, the tonsil, posterior pharyngeal wall or in the larynx. The gumma, whether ulcerated or not, is typically painless. There is usually no lymphadenopathy associated with these lesions unless they are secondarily infected. When treated with penicillin the gumma will rapidly heal.

**Diagnosis**

In the primary or secondary stage of the disease spirochaetes can be identified by dark field illumination microscopy in smears taken directly from the lesion. The spirochaetes can also be identified in biopsy specimens using silver stains or fluorescein-labelled antibody. Biopsy of a tertiary lesion gives a typical histopathological picture. Serological tests for syphilis fall into two main groups: those used to identify non-specific antibodies to cardiolipin (VDRL tests) and those to detect specific treponemal antibodies (TPI and FTA).

The VDRL (Venereal Disease Research Laboratory) tests use an antigen extracted from beef heart in a slide flocculation test. The VDRL reaction begins to become positive during the first or second week after the development of the chancre and 99% of patients with secondary syphilis give a positive reaction as do a similar proportion with tertiary syphilis. Unfortunately a proportion of patients with other diseases also give a positive reaction. These include other infections involving non-syphilitic treponemes (yaws, bejel or pinta which are cutaneous infections) and also infections such as atypical pneumonia, malaria, smallpox and leprosy. Some patients with disordered immune systems such as those with systemic lupus erythematosus or rheumatoid arthritis will give a positive reaction and occasionally elderly patients who have never been exposed to syphilis will show a positive reaction.

Of the tests for specific antibody, the TPI (*Treponema pallidum* immobilization) is the most specific but also the most expensive. It depends upon the ability of antibody in the patient's serum to immobilize spirochaetes which are observed by dark ground illumination. The TPI test is 100% positive in patients with established secondary and tertiary syphilis and, if carried out correctly, is entirely specific. The FTA (fluorescent treponemal antibody) test involves the absorption from the patient's serum of cross-reacting antibodies using non-pathogenic treponemas and then absorption of specific antibody by dried *Treponema pallidum* preparations. The absorbed antibody is identified by a fluorescein-labelled antihuman gamma globulin. The FTA test is positive in 100% of patients with secondary and tertiary syphilis.
and the false positive rate is not as high as in the VDRL reaction but occasional patients with systemic lupus erythematosus or rheumatoid arthritis will be positive.

Treatment

In primary and secondary syphilis a dose of 2.4 mega units of benzathine penicillin in a single or two intramuscular injections is satisfactory. In tertiary syphilis a total of 7.2 mega units of benzathine penicillin is given usually as 2.4 mega unit injections at around 7 and 14-day intervals.

Tuberculosis

The pharynx is not a common site for clinically manifest tuberculosis. However, it is the site of primary infection which nearly always occurs in children and results in an asymptomatic primary focus in the pharynx (usually the tonsil or adenoid) with cervical lymphadenopathy.

Secondary tuberculosis affects the pharynx but only in patients with massive sputum positive and usually cavitating pulmonary tuberculosis. This is in contrast with laryngeal tuberculosis when lesions do occur with low grade or inactive pulmonary pathology. The pharyngeal lesions are secondary to coughing up heavily infected sputum and consist of very painful multiple shallow ulcers in the pharynx or oral cavity. Occasionally the pharynx is involved in patients with widespread miliary tuberculosis and here the lesions may be from blood-borne as well as sputum-borne dissemination of the disease.

Lupus vulgaris is a low grade cutaneous infection of tuberculosis and has been described in the nasal cavities and in the pharynx. Tuberculous otitis media is probably a blood-borne dissemination of the disease but occasionally it can arise from pharyngeal disease by spread from the eustachian tube.

Diagnosis

There is little difficulty in making the diagnosis of pharyngeal tuberculosis because of the association with pulmonary disease which is obvious clinically and radiologically.

Treatment

Pharyngeal tuberculosis needs no special treatment; it will be treated at the same time as the pulmonary disease, namely with triple therapy usually using isoniazid, rifampicin and pyrazinamide as first line drugs.

Toxoplasmosis

Toxoplasmosis is a common disease of birds and mammals caused by the protozoan Toxoplasma gondii. The infection can be transmitted to humans by the ingestion of cysts in uncooked meat or food contaminated with animal faeces.
In immunocompetent humans acquired toxoplasmosis usually gives rise to no symptoms. Some will have a sore throat with malaise and fever and cervical lymphadenopathy and, on occasions, the patient will simply present with an enlarged cervical lymph node. The fever and malaise may last for several weeks and many organ systems may be involved, for example, lungs, myocardium, pericardium, liver, brain and skeletal muscle. The disease is usually self-limiting and death is most unusual.

In immunodeficient individuals the disease may be much more serious with multisystem failure and death.

The diagnosis of the disease can be made by a serological test which is an indirect dye or fluorescent antibody test. On occasions lymph nodes will be removed for purposes of biopsy and the histology is typical with follicular hyperplasia and typical epithelioid cells.

Treatment

Usually no treatment is necessary but in those individuals with a severe systemic upset or in immunodeficient individuals a combination of pyrimethamine and sulphadiazine is indicated.

Leprosy

Isolated leprosy of the pharynx does not occur; it spreads to the nasopharynx and occasionally to the oropharynx from the nasal cavities. Leprosy is an infection caused by *Mycobacterium leprae* which produces a chronic disease with a spectrum of clinical manifestations. Tuberculoid leprosy is a low grade lesion affecting an area of skin and its nerve supply. Lepromatous leprosy is a more florid form of the disease with massive infection of the dermis of the skin; it can affect the nasal cavities, nasopharynx and also the testis and lymphoreticular system. Disease in the pharynx spreads from the nasal cavities and gives rise to a combination of granulomatous lesions, ulcerating and healing with fibrosis. The diagnosis is usually made by biopsy and the treatment is by chemotherapy with sulphones.

Scleroma

This is a chronic infective condition caused by *Klebsiella rhinoscleromatis*. The disease begins in the nose and only secondarily spreads to involve the pharynx where it produces granulomatous lesions and scarring.

Fungal infections

*Candidiasis (moniliasis)*

*Candida albicans* is a fungus which is part of the flora of the oral cavity or oropharynx in 30-40% of normal individuals. For the organism to become pathogenic and give rise to symptoms there must be a local or systemic change in the host. It has been called a 'disease of the diseased'.
Local changes which predispose to infection by candida may complicate local diseases such as lichen planus and leukoplakia. Systemic antibiotic administration may change the oral flora sufficiently to disturb the local balance and allow overgrowth of candida. Radiotherapy to the oral cavity and pharynx is often complicated by candida.

In patients with chronic ill health, and particularly immunocompromised individuals, candidiasis of the oral cavity or pharynx may occur. Thus diabetes, lymphomata and treatment with immunosuppressive agents predispose to candidiasis. It is a common feature in patients with acquired immune deficiency syndrome (AIDS).

Infection with candida in the oral cavity or pharynx may be asymptomatic or may give rise to severe pain with dysphagia. Clinically it gives rise to small white patches which when removed leave an erythematous ulcer.

Candidiasis can be treated by local or systemic therapy or a combination of the two. Attention must be paid to the predisposing condition if this is possible. Local antifungal agents are usually effective: nystatin 100,000 units 6-hourly, amphotericin 100,000 units 6-hourly, miconazole 125 mg 6-hourly; occasionally systemic therapy (ketoconazole 100 mg twice daily) will be more effective.

**Pharyngeal symptoms of blood disease**

Certain blood diseases, because of their effect on the immune system, often present with lesions in the mouth or pharynx.

*Agranulocytosis*

Diseases which result in a severe decrease in the number of polymorphonuclear leucocytes are unusual but often present with oral and pharyngeal symptoms. Occasionally agranulocytosis will be idiopathic but most of these disorders are hypersensitivity reactions to drugs which only affect a small proportion of patients. Some are dose related, others are not. A large variety of different drugs is likely to produce these reactions, for example, chloramphenicol, sulphonamides, phenylbutazone, thiouracil, carbimazole and chlorpromazine. Chemotherapeutic cytotoxic agents have a direct effect on bone marrow and result in marrow aplasia and this is dose related. The first symptoms associated with agranulocytosis are fever and headache with severe pain on swallowing. The lesions in the pharynx are necrotic ulcers with a slough and may be single but are usually multiple and coalescent. There is usually no cervical lymphadenopathy. There is often severe halitosis and the patients become very ill. Culture of the ulcers rarely gives any information usually only yielding normal commensal organisms. The diagnosis of agranulocytic pharyngitis is made on the blood count and film which should always be performed on any patient with a sore throat. Treatment involves withdrawal of the precipitating agent, high dose steroids and systemic antibiotics. Blood transfusions are often necessary.

**Acute leukaemia**

Acute leukaemia may be of three types (lymphoblastic, myeloblastic and monoblastic) although the clinical presentation is indistinguishable. The lymphatic type is most common
in children, myeloid most common in young adults and the monocytic can occur at any age. These diseases present with fever, anaemia and bleeding disorders. Often the oral or pharyngeal manifestations are the first symptoms. Ulceration with slough and membrane formation occurs on the gums, in the oral cavity and in the pharynx. There is nothing specific about these lesions except for their severe extent and the fact that they are often associated with haemorrhage. Often there is an associated bilateral cervical lymphadenopathy. The diagnosis is made by a blood picture and bone marrow examination.

Vincent's angina

Vincent's angina (trench mouth) is an infection with a spirochaete, *Borellia vincenti* and an anaerobic organism *Bacillus fusiformis*. In general, Vincent's angina occurs in patients with very poor dental hygiene and a generally debilitated condition. It can occur, however, without these predisposing factors. It is essentially a necrotizing gingivitis with ulceration and bleeding of the gums which are covered with a necrotic membrane. The lesions are painful and associated with marked fetor and the patient may be pyrexial and often has tender enlarged cervical glands. The lesions may spread to involve the tonsil but, on occasions, the tonsil may be involved without a gingivitis or stomatitis. The lesion of the tonsil is a deep ulcer with a grey slough in its base. The diagnosis is made by taking a scraping from the ulcer or gingiva on a slide, staining with gentian violet and identifying both the spirochaete and the fusiform bacillus. Treatment of Vincent's angina is usually local with peroxide mouth washes, care from a dental hygienist, together with benzyl-penicillin. In addition metronidazole (500 mg 8-hourly intravenously) should be given.

Pharyngeal stenosis

Stenosis of the pharynx is not common. When it occurs there is fibrous tissue formation with adhesions and fibrous strands covered by mucous membrane. It can occur in the nasopharynx involving the posterior choanae or the eustachian openings. It may also arise in the oropharynx and the soft palate may be firmly fixed to the posterior pharyngeal wall. Stenosis in the nasopharynx and oropharynx may affect nasal respiration, speech and eustachian tube function. Swallowing is usually affected by hypopharyngeal stenosis. Involvement of the larynx by the same pathological process which caused stenosis of the pharynx is much more serious and results in dysphonia and airway difficulty. The cause of stenosis may be varied (*Table 5.2*) and usually stenosis is the end result of an inflammatory process. Occasionally the stenosis may occur at the same time as the active pathological process (for example, scleroma, leprosy, Wegener's granuloma).

Treatment of established stenosis of the nasopharynx or oropharynx is rarely necessary or successful. Hypopharyngeal stenosis can be treated by repeated dilatation.

Acquired immune deficiency syndrome (AIDS)

Otolaryngological symptoms occur in about 40% of patients presenting with AIDS (Marcussa and Sooy, 1985). The importance of this is first in correctly diagnosing and therefore treating the patient, and second, in recognizing the condition and avoiding surgery without precautions to safeguard members of staff. The agent known to be responsible for AIDS is the human T-cell lymphotrophic virus type III (HTLV III) which is transmitted by
sexual contact or through blood products. In the case of medical staff needle-stick injuries or contamination of cuts, abrasions or skin lesions or possibly conjunctival contamination with blood or secretions are the modes of infection. (At the time of writing the virus is still generally referred to as HTLV III. However, the internationally agreed name is now the human immunodeficiency virus and it is likely that HIV will become the routine expression.)

The most likely presentation of AIDS to the otolaryngologist is the patient who has persistent cervical lymphadenopathy. This may occur alone or may be part of a generalized lymphadenopathy associated with pyrexia, diarrhoea, weight loss, oral or pharyngeal candidiasis and lymphopenia. Some patients with AIDS may present with recurrent episodes of acute tonsillitis, acute pharyngitis, sinusitis or rhinitis. None of these show any typical features. Candidiasis is much more frequent in patients with AIDS and its occurrence in young males must be viewed with great suspicion unless there are other predisposing factors. Candidiasis affects the oral cavity and pharynx in patients with AIDS and two other lesions may be seen in the oral cavity. Hairy leukoplakia was first described by Greenspan et al in 1984 and consists of white patches on the tongue which seem to regress and then recur. Histologically, the leukoplakia shows marked keratinization giving the hairy appearance. The other lesion which occurs in the oral cavity or oropharynx in patients with AIDS is Kaposi's sarcoma. This was a rare lesion until AIDS was described, previously being confined to elderly male patients particularly of eastern European descent, children and young adults in Africa and patients who were immunosuppressed. About 30% of patients with AIDS have Kaposi’s sarcoma and 25% present with it. The lesions affect the skin and oral cavity as flat or raised red patches or nodules which may ulcerate. The lesions should be excised; on occasions lymph nodes are involved and there may be systemic involvement. The disease is rarely lethal in the patient with AIDS; death occurring as a result of AIDS rather than the sarcoma.

If the otolaryngologist suspects a patient may have AIDS the patient should be referred to a venereologist or physician. The diagnosis is made by the multiple manifestations of the disease together with a lymphopenia and antibodies to the HTLV III virus. The latter may be positive in homosexual males who can transmit the virus and it is felt that approximately 30% will go on to develop AIDS. When the otolaryngologist is required to perform surgery on a patient who has high HTLV III antibody levels the patient should be managed in the ward and in the operating theatre in the same way as a patient with hepatitis B infection (Youngs, Stafford and Weber, 1986).

Obstructive sleep apnoea

This is a subject which is incompletely understood at the present time and there are many questions which remain unanswered about apnoea during sleep. There is no doubt that normal individuals have apnoeic episodes when asleep and that these episodes are completely harmless. On the other hand, there are examples of patients who have develop right-sided heart failure and pulmonary hypertension secondary to upper airways obstruction with apnoea. The difficulty lies in the area between these two extremes and how one should define it. Apnoea during sleep has been divided into obstructive, central, and mixed; obstructive apnoea being preceded by upper respiratory obstruction with increasing respiratory effort. Central apnoea occurs without this increasing respiratory effort but the mechanism could still be obstructive; it is simply that the drive to respiration is lacking. Purely central apnoea with no
obstructive element does occur but is probably very rare. Mixed apnoea is a combination of failure of central control and upper airway obstruction.

The mechanism of the upper airway obstruction is unknown but it has certainly been demonstrated that in sleep there is reduced activity in the muscles of the tongue, soft palate and pharynx and this reduced activity may have a central component (Remmers et al, 1978). The loss of tone in the muscles of the tongue, soft palate and pharynx results in a collapse of the airway due to a suction effect and, as respiratory effort increases, the suction and therefore the obstruction increases. As the patient becomes hypoxic there is an arousal response which increases muscle tone and improves the airway. This loss of tone in upper airway musculature during sleep has been demonstrated in normal asymptomatic individuals and the factors which lead to the development of the full-blown obstructive sleep apnoea syndrome are probably multiple. It has arbitrarily been accepted that obstructive sleep apnoea occurs when a patient has 30 apnoeic episodes each lasting 10 seconds or more during 7 hours of sleep. This is reinforced if it can be demonstrated that during these apnoeic episodes the oxygen saturation levels in arterial blood fall. This standard for sleep apnoea is by no means universally accepted and much more investigation remains to be done.

A number of studies cast doubt upon the validity of this standard. For example, an investigation by Black et al (1979) of normal individuals has shown that up to 50% of these would fit the above criteria for sleep apnoea, with oxygen desaturation levels falling as low as 60%. The relationship between snoring and sleep apnoea is also ill-defined. Snoring is more common in the elderly male and occurs in a surprisingly high proportion of the population (Lugaresi et al, 1980). Snoring is associated with a high incidence of systemic hypertension and simple snorers can develop obstructive sleep apnoea after alcohol excess (Remmers et al, 1978). Thus, the supposition has been raised that a proportion of patients who snore may go on to develop obstructive sleep apnoea.

Clinical features

The classical features of the Pickwickian syndrome, which is the first described example of sleep apnoea syndrome, occur only rarely (Gastaut, Tassinari and Duron, 1966). The classical description is of a squat obese individual with a short fat neck who snores heavily with increasing respiratory effort until prolonged apnoeic episodes occur with cyanosis. The patient then begins to breathe again with semi-arousal only to drift back into sleep and a further apnoeic episode. This disturbed sleep with oxygen desaturation leads to daytime somnolescence such that the individual is falling off to sleep during the daytime and it may be difficult to distinguish the condition from narcolepsy.

Obstructive sleep apnoea can and does occur in non-obese individuals, although it seems to be more prevalent in elderly obese males. The reported incidence in the USA is considerably higher than that in the UK and Europe. This may be a real difference between the populations or may be an apparent one generated by the fact that many more sleep laboratories exist in the USA for the investigation of such disorders. Examination of the oropharynx of patients with obstructive sleep apnoea may reveal no abnormality although a proportion has a rather elongated soft palate and uvula. Examination may also show a cause for nasal obstruction such as a deviated nasal septum or nasal polyps. It should be remembered that patients with asthma and those with obstructive lung disease may show sleep
disturbance and nocturnal oxygen desaturation. After full clinical examination, investigation of patients with obstructive sleep apnoea requires sinus and chest radiography, full blood picture and pulmonary function tests. Polysomnography is essential but the facilities for this are sparse in the UK at present. Thus monitoring during sleep with ECG, EEG, EOG (electro-oculogram), ear oximetry, oral and nasal thermistors and thoracoabdominal strain gauges is necessary. Patients with obstructive sleep apnoea will show apnoeic episodes preceded by increasing respiratory effort but reduced air flow. Oxygen desaturation is often accompanied by bradycardia and cardiac arrhythmias eventually leading to right ventricular failure and pulmonary hypertension.

Treatment

This is a controversial subject and even more so if one considers the treatment of snoring as well. Patients with severe obstructive sleep apnoea with oxygen desaturation certainly should be treated and the options are as follows.

In patients who are obese, weight loss may help and also may make other treatment more successful. Medical treatment of obstructive sleep apnoea includes the continuous administration of oxygen at increased pressure (CPAP), although this is not comfortable and not well tolerated by patients. Protriptyline has been shown to decrease the number of apnoeic episodes.

The most successful surgery for obstructive sleep apnoea is tracheostomy, although this has major disadvantages. Correction of obvious upper airway defects such as a severely deviated nasal septum or removal of nasal polyps may help some patients and should be carried out before more drastic measures are undertaken. Recently, excision of part of the soft palate and uvula (palatopharyngoplasty) has been described (Blair-Simmons, Guilleminault and Silvestri, 1983) and the results documented. The surgery involves resection of the tonsils if they are still present together with a portion of the free edge of the soft palate and uvula. It is yet to be determined how much tissue should be resected and depends upon the amount which it is thought is redundant (in some patients as has already been said the soft palate looks normal and yet resection of some soft palate will improve the symptoms of obstructive sleep apnoea). In general 10-15 mm of soft palate should be resected and this is the full thickness of the free border of the soft palate. More should be resected medially than laterally. If the tonsils are removed at the same time, suturing the anterior to the posterior pillar has been advised. If the tonsil is not present but the pillars seem excessively flaccid a portion should be resected. The bare surface of the soft palate remaining after resection is sutured so that the mucosa of the posterior surface is brought into contact with the anterior surface.

The results of palatopharyngoplasty are variable. Some patients are cured of sleep apnoea and oxygen saturation levels of 60% before surgery may return to normal after surgery. Some patients are helped only to a marginal extent and some not at all. The implication in these patients is that the obstruction is at another level and mandibular osteotomies, hyoid bone expansion or wedge resection of parts of the base of tongue have on occasion been helpful (Guilleminault, 1984).

In patients with very severe apnoea and desaturation levels down to 50% tracheostomy is probably the treatment of choice. The complications of palatopharyngoplasty are similar to
those of tonsillectomy with the addition of palatal incompetence in some. Postoperatively approximately 30% of patients will have regurgitation of fluid after palatopharyngoplasty but this proportion improves until only a small percentage remains. However, in these patients nasal regurgitation of fluids and speech with nasal escape is permanent.

Tonsillectomy

Tonsillectomy, like all other surgical procedures, should never be advised unless it is virtually certain that the patient will benefit. The indications for the procedure should be stringent and loose indications such as halitosis, globus hystericus and others are to be condemned (Tucker, 1982a). Such indications simply expose the patient to totally needless trauma and risk, and serve to damage the reputation of otolaryngology as a surgical speciality. Nevertheless, when performed for the correct indications adult tonsillectomy is a very worthwhile procedure.

Indications

Recurrent tonsillitis

Tonsillitis does occur in adults but it is not common. Repeated attacks (five per year for 2-3 years) are a definite indication for tonsillectomy. Acute tonsillitis in an adult is a significant illness and can be diagnosed from the history of pyrexia, severe sore throat lasting several days and constitutional upset. Repeated episodes of minor sore throats, or a more or less constant discomfort in the throat rarely, if ever, arise from the tonsils and do not constitute indications for tonsillectomy. If there is doubt about the diagnosis in a patient with repeated sore throats and the surgeon is not certain that these represent acute tonsillitis then arrangements must be made for the patient to be examined by the surgeon during an acute attack. Only in this way can the situation be elucidated and the patient advised correctly. There is absolutely no place for a policy of performing the surgery because the patient desires it or because the surgeon cannot think what to advise for a particular symptom.

Peritonsillar abscess

There is no doubt that peritonsillar abscess is a serious illness with a definite mortality. There is also no doubt that a patient who has had one quinsy is liable to have a second or third. However, this liability to recurrence is closer to 20% than 100% and a quinsy is not an absolute indication for tonsillectomy. Each patient must be judged individually; one who has had repeated episodes of acute tonsillitis who then develops a quinsy is perhaps in need of tonsillectomy more than a patient who has a quinsy with no previous tonsillitis. A second quinsy is probably the point at which to decide absolutely on tonsillectomy. Abscess tonsillectomy is discussed earlier in this chapter but in the author's opinion has little to recommend it.

Tonsillectomy for biopsy purposes

When a tonsil is thought to be the site of a neoplasm biopsy is essential. Thus the patient with a unilateral enlarged and abnormal looking or ulcerated tonsil should be advised to have a biopsy. This can be undertaken as an incisional biopsy and is the method of choice.
when a squamous carcinoma is suspected. When the swelling is thought to be a lymphoma (a large hyperaemic, fleshy-looking tonsil) the approach may either be incisional biopsy or tonsillectomy. Certainly tonsillectomy provides more tissue for the histopathologist to study but is more hazardous than incisional biopsy. If the patient has obvious lymph nodes in the neck, biopsy of one of these may be preferable to the patient and will enable the pathologist to determine lymph node architecture.

**Other indications**

It is usual to perform a tonsillectomy for sleep apnoea syndrome when the patient is undergoing palatopharyngoplasty. When the glossopharyngeal nerve in treatment of glossopharyngeal neuralgia or the styloid process for Eagle's syndrome are being approached through the pharynx the tonsil must be removed to give access.

**Contraindications to tonsillectomy**

**Bleeding disorders**

Tonsillectomy should not be performed when the patient has a bleeding tendency. This bleeding disorder must be investigated, diagnosed and treated prior to tonsillectomy. Patients with a vague history of bleeding easily or with a family history of bleeding disorder must be thoroughly screened and usually their clotting mechanism will be found to be normal. In patients with a known bleeding disorder the advisability of surgery must be discussed with the patient and haematologist and the risks discussed and estimated. If it is still felt that surgery is indicated then the patient's deficiency must be corrected by the haematologist prior to surgery.

**Recent infection**

A recent (within 4 weeks) episode of tonsillitis makes the surgery more difficult and increases the risk of haemorrhage. A recent upper respiratory tract infection probably increases the risks of pulmonary complications of general anaesthesia and the operation should be postponed for 2 or 3 weeks.

**Oral contraceptives**

The risks of deep vein thrombosis after tonsillectomy are very low indeed and it is unknown whether this risk is increased in patients who are taking oestrogen-containing oral contraceptives. The three options open to the surgeon in this instance are to ignore the risk and carry on; advise the patient to stop the contraceptive 6 weeks prior to surgery and to use some other form of contraception; cover the period of surgery with low-dose heparin (5000 units 12 hours before, 5000 units with the premedication and at 12 and 24 hours postoperatively). The author favours the second option and in patients who refuse this uses the third choice.
Preoperative considerations

A patient undergoing tonsillectomy must have a carefully taken history and full clinical examination prior to surgery. In particular recent upper respiratory tract infection, bleeding tendency, coincidental anaemia or heart murmur must all be ruled out. The state of the patient's dentition must be assessed and in particular carious or loose front teeth or capped teeth must be looked for and the patient suitably informed of the risks. Urinalysis is the minimum in the way of preoperative investigations, some surgeons require also a haemoglobin estimation, blood grouping and chest radiographs.

The operation of tonsillectomy

Premedication should be determined by the anaesthetist, but Omnopon (papaveretum) 20 mg and scopolamine (hyoscine) 0.4 mg are suitable for the average adult. General anaesthesia is the routine for tonsillectomy in the UK and a nasotracheal tube is the usual method of maintaining the airway.

A detailed description of a particular surgical technique for tonsillectomy is not desirable as this is a procedure which must be taught practically and because many different variations of technique are used. It is up to each surgeon to develop his own method under close instruction and supervision. It is worthwhile, however, making some general points which are common to most techniques.

(1) Most surgeons use a technique of dissection because it allows a more careful and thorough removal of all lymphoid tissue. Guillotine tonsillectomy is a dying art and perhaps this is a good thing.

(2) A Boyle-Davis gag (which is not hot) should be positioned with care not to damage the lips, teeth or posterior pharyngeal wall and so that the tongue blade lies centrally along the dorsum of the tongue.

(3) The mucosa of the anterior and posterior tonsillar pillars should be incised in such a way as to preserve as much mucosa as possible.

(4) The plane of dissection of the tonsil is in the loose alveolar tissue between the capsule of the tonsil and the superior constrictor muscle of the pharynx. It is essential that the dissection is performed in this plane, if too deep a plane is dissected the muscle will be damaged and bleeding will be increased, and if the plane is not deep enough then lymphoid tissue will remain. It is usual to begin this dissection at the upper pole of the tonsil, but if difficulty is encountered the plane should be found more inferiorly and then followed superiorly. Most of the difficulty in tonsillectomy is because this plane is not identified and, in some situations, particularly after a quinsy this identification is quite difficult. Some muscle fibres of the superior constrictor insert into the fibrous tissue of the tonsillar capsule and they must be gently dissected away. The dissection is carried inferiorly to the junction of the tonsil and the base of the tongue and the final attachment of the tonsil is severed often using a snare.
(5) The most essential part of tonsillectomy is the control of haemorrhage and the operation is not complete until all bleeding has ceased. The fossae should be absolutely dry at the end of the procedure. It is usual to pack the fossa with gauze immediately following removal of the tonsil while the other tonsil is removed or, in the case of the second tonsil, while bleeding from the first fossa is dealt with. Much of the bleeding will have subsided by the time the gauze pack is removed. Bleeding points are identified and either ligated or diathermized depending upon the surgeon's preference. Diathermy is quicker (Haase and Noguera, 1962), but great care must be taken not to inflict an unwanted diathermy burn on the patient. The incidence of secondary haemorrhage may be greater after the use of diathermy (Carmody, Vamadenan and Cooper, 1982; Siodlak, Gleeson and Wengraf, 1985).

(6) When all haemorrhage has been controlled the blood clot must be aspirated from the nasopharynx where it will have accumulated and all swabs must be removed and counted.

The recovery of the patient from anaesthesia must be as gentle as possible, coughing and retching are liable to cause haemorrhage. The patient is recovered in the tonsil position, that is on his side with his head down so that if haemorrhage does begin the blood will flow out of the nose and mouth and not downwards to the larynx. The airway must be maintained at all times and noisy respiration may mean that there is blood in the airway. This should be inspected and the blood and secretions aspirated. At the same time the tonsillar fossae should be inspected for signs of haemorrhage.

**Postoperative care**

The postoperative care of a patient following tonsillectomy is directed towards early detection of haemorrhage. Thus regular observation of pulse (every 15 minutes for 4 hours, every 30 minutes for a further 4 hours, then hourly is a suitable regimen), and blood pressure (hourly for 12 hours) is essential. Haemorrhage is less likely to be concealed in an adult than in a child, but in the semi-conscious patient careful observation is essential and noisy respiration may indicate blood in the pharynx and the fossae should be examined. Vomiting of blood or a rise of pulse rate means the tonsillar fossae must be inspected for signs of haemorrhage. Postoperative analgesia must be given and two or three doses of Omnopon (20 mg) are suitable. After this paracetamol is usually sufficient, aspirin increases the risk of haemorrhage (Carrick, 1984).

**Complications of tonsillectomy**

These may be conveniently divided into perioperative and postoperative and the latter subdivided into immediate, intermediate and late, but this is a somewhat artificial classification. The most significant complication is haemorrhage and most of the deaths associated with tonsillectomy are directly or indirectly associated with this complication.

**Perioperative**

**Haemorrhage**

The amount of bleeding during tonsillectomy varies with individual patients and surgeons. Recent infection, previous peritonsillar abscess, and severe scarring are the factors
which increase the haemorrhage in any one patient. The surgeon can minimize the haemorrhage by a careful, gentle dissection technique. Excessive haemorrhage from both fossae raises the question of a coagulation defect which was previously unsuspected and this must be excluded. When haemorrhage is excessive and cannot be controlled by the usual means (ligation, diathermy) other methods are available. A single difficult bleeding point may need undersewing and ligating. If excessive haemorrhage occurs throughout the fossa it can be controlled by leaving a pack in the fossa and oversewing the pillars. This is hazardous because of the risk of dislodgement of the pack in the immediate recovery period. A pack of absorbable material, for example Calgitex or Gelfoam, can be used or, alternatively, a gauze pack can be left in the fossa and removed the following day by dividing the sutures. If the latter is chosen a tie can be brought out of the mouth and taped to the cheek. Occasionally an aberrant internal carotid artery will be encountered in the tonsillar fossa and this leads to massive haemorrhage which can only be controlled by ligation of the internal carotid artery in the neck. Occasionally uncontrollable haemorrhage during tonsillectomy, possibly due to a large tonsillar branch of the facial artery, requires external carotid artery ligation and this should be performed inferior to the origin of the facial artery.

**Trauma**

As has been said already, capped or carious teeth are at risk during tonsillectomy and the patient should be warned of this. Dental advice may be necessary before and after surgery. Gentle insertion of the gag minimizes the risk of damage to the teeth and also of damage to the posterior pharyngeal wall by a hurried or careless insertion of the tongue blade. Badly placed mucosal incisions can lead to excessive loss of mucosa of the soft palate, and occasional damage to the arterial supply of the uvula bilaterally may lead to loss of the uvula. Insertion of the gag can sometimes lead to dislocation of the temporomandibular joint or pain due to joint dysfunction in the postoperative period.

**Postoperative**

**Immediate complications**

**Haemorrhage**

The most significant immediate complication of tonsillectomy is so-called reactionary haemorrhage. By definition this occurs up to 24 hours postoperatively, but the vast majority of reactionary or primary haemorrhages occur within the first 8 hours.

Reactionary haemorrhage is dangerous in two ways; first, in the phase during which the patient is recovering from anaesthesia before the cough reflex is fully established, blood in the airway can result in laryngeal spasm or can asphyxiate the patient by mechanically occluding the airway. Second, haemorrhage results in hypovolaemia which, if not corrected, results in peripheral circulatory failure (shock) and eventually death.

Reactionary haemorrhage after tonsillectomy is unusual, occurring in about 0.5-1% of operations (Nesbitt, 1934; Williams, 1967). The cause is unknown but it must represent bleeding from an artery or vein which had stopped bleeding at the time of surgery. The possible causes of renewed bleeding may be dislodgement of blood clot from the lumen of
the vessel or vasodilatation of a vessel which was in spasm at the time of surgery. This is probably a local problem but it is possible that changes in blood pressure or state of vessels by anaesthetic agents may play a part. Bleeding from a vein postoperatively may be due to excessive venous pressure induced by coughing or retching. However much one speculates on the causes of reactionary haemorrhage, it must be admitted that the precise cause in the vast majority of patients is unknown.

In order to avoid disaster any bleeding following tonsillectomy must be taken very seriously. Blood must be cross-matched at the first sign of haemorrhage. The tonsillar fossae must be inspected with great care, the clot removed if possible and a bleeding point may be seen. If the bleeding is minor it may cease once the clot has been removed or it may stop with a little pressure with a swab, possibly soaked in 1:1000 adrenaline. Even if all haemorrhage then ceases the fossae should be inspected from time to time over the next few hours as bleeding can start again. If there is any doubt, the patient must be prepared for a second anaesthetic and the bleeding point ligated under general anaesthesia. This second anaesthetic is hazardous and carries a significant mortality (Davies, 1964) because the patient has already had one anaesthetic, has blood in his airway, is hypovolaemic from blood loss and also may have a stomach full of blood.

Anaesthesia

The anaesthetic complications in the immediate postoperative period relate to the maintenance of an adequate airway, free of secretions and particularly of blood. A swab left in the airway may obviously lead to asphyxiation and it is the surgeon's responsibility to be certain that no such oversight occurs.

Intermediate complications

Haemorrhage

The most significant intermediate complication is secondary haemorrhage which by definition, is any haemorrhage which occurs more than 24 hours after surgery and classically occurs at 10 days. This time course implies that the most likely cause is infection. Secondary haemorrhage after tonsillectomy is not common, occurring in about 1% of patients. It is not usually as severe as primary haemorrhage but, nevertheless, must be taken very seriously as it does have a definite mortality.

The patient should be treated with systemic antibiotics (intravenous penicillin or erythromycin), a haemoglobin estimation should be performed and blood should be cross-matched.

It is usual to remove blood clot from the tonsillar fossa and perhaps hold a swab soaked in 1:1000 adrenaline in the fossa. How much these measures influence the course of events is not known.

It is most unusual for secondary haemorrhage to be severe enough or sufficiently prolonged to require formal ligation under anaesthesia. If this is contemplated a coagulation screen should be performed because a minor coagulation defect which did not cause problems
at initial surgery can still cause problems at the secondary stage. In a tonsillar fossa which is infected and friable it is difficult to find and ligate a bleeding point. Occasionally this will be possible but more usually diathermy or suturing of a bleeding point will be necessary.

**Haematoma and oedema of the uvula**

Excessive surgical trauma can produce marked bruising and oedema of the uvula and, although alarming to look at, this settles down without a problem. Damage to the arterial supply to the uvula bilaterally is very rare but when it occurs the uvula may necrose.

**Infection**

Postoperatively the tonsillar fossae contain whitish slough which one would expect to be an ideal culture medium for bacteria. In fact, the fossae rarely become seriously infected. When this does occur it can be recognized by increasing, rather than decreasing pain around the end of the first postoperative week, and earache seems to be particularly common. The fossae look surprisingly normal but the combination of pyrexia with increasing pain leaves little doubt that there is infection. Untreated this may lead to secondary haemorrhage but usually haemorrhage is the first sign that infection has occurred.

**Pulmonary complications**

Pulmonary atelectasis postoperatively leading to pneumonia is very rare. It is more likely to occur if the patient has an upper respiratory tract infection at the time of surgery and inhalation of blood or fragments of tonsil tissue may precipitate it. Very rarely a lung abscess will supervene.

**Subacute bacterial endocarditis**

Tonsillectomy leads to a transient bacteraemia at the time of surgery. If the patient has an abnormal heart valve or rarely, a septal defect, subacute bacterial endocarditis may complicate the operation. For this reason all patients with a heart murmur found at the preoperative investigation should have been examined by a cardiologist and, if necessary, the surgery and immediate postoperative period should be covered by systemic penicillin.

**Earache**

Postoperative earache is fairly common after tonsillectomy and is usually referred pain from the tonsillar fossa, although occasionally this will herald the onset of a secondary infection. Acute otitis media has been described as a complication of tonsillectomy and the ears should always be examined in any patient who complains of otalgia.

**Late complications**

**Postoperative scarring**

Careless, traumatic surgery with loss of mucosa, particularly on the soft palate can result in scar tissue in the palate which limits mobility. In an extreme case it is conceivable
that this could affect the voice of a patient, producing nasal escape. The latter is of course much more common after a combined tonsillectomy and adenoidectomy.

**Tonsillar remnants**

Incomplete dissection can leave behind small islands of lymphoid tissue. Failure to remove the lower pole of the tonsil right down to the base of the tongue can result in an appearance which looks as though the tonsils have not been removed. Usually small tonsillar remnants are asymptomatic and discomfort in the throat or minor sore throats should not readily be ascribed to tonsillar remnants. Large masses of tonsil tissue remaining after tonsillectomy can result in acute tonsillitis and peritonsillar abscess has been described in a patient whose tonsils had been removed. If it is felt that tonsillar remnants are the site of recurrent acute infection then they should be removed.

**Malignancy following tonsillectomy**

A report by Vianna, Greenwald and Davies in 1971 suggested that patients whose tonsils had been removed were almost three times more likely to develop Hodgkin's disease when compared with patients who still had their tonsils. In fact the statistics of the study were felt to be subject to bias and further studies since have shown no difference in the occurrence of Hodgkin's disease after tonsillectomy (Ruuskanen, Vanha-Pertula and Kavalainen, 1971; Johnson and Johnson, 1972).