# 8
# Respiratory tract infections

## CHILDHOOD RESPIRATORY TRACT INFECTIONS (Figure 31)

Respiratory tract infections are common in the young as they have no acquired immunity to certain pathogenic agents and their immune systems are not fully mature. In most childhood respiratory tract infections the whole of the respiratory tract is infected, but often symptoms and signs are

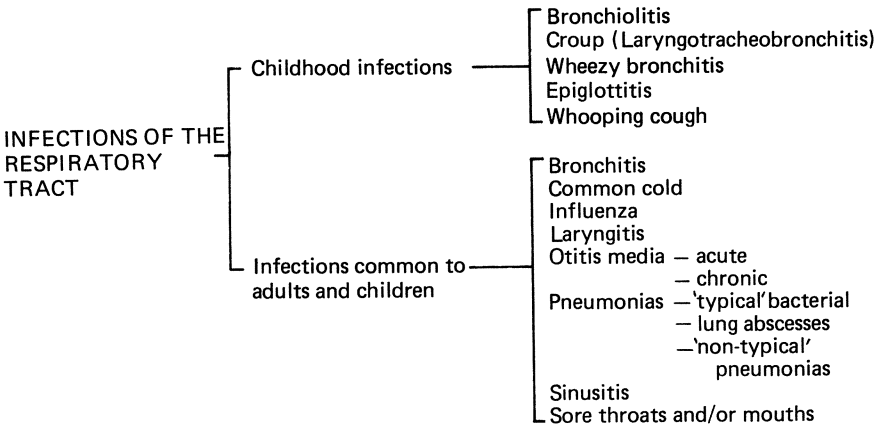


Figure 31 A classification of respiratory tract infections

predominantly of one site. The form the illness takes is often related to the relatively small airways. Recurrent respiratory tract infection in childhood should always raise the possibility of cystic fibrosis (mucoviscidosis).

The possibility of foreign body inhalation or heart failure should not be forgotten in any breathless child. In all recent onset respiratory tract infections in children always look for Koplik's spots – the admission of a child with prodromal measles to a paediatric ward can initiate an epidemic of measles in vulnerable and already ill children.

### *Bronchiolitis*

Although there are several causative agents, bronchiolitis is usually caused by respiratory syncytial virus or parainfluenza viruses. Bronchiolitis often occurs in the winter months and commonly affects infants less than 2 years old, usually 1–6 months old. After a brief upper respiratory tract prodrome there is partial obstruction of terminal branches of the bronchial tree resulting in expiratory wheezing,* intercostal indrawing and *generalized symmetrical* rhonchi on auscultation. Fine inspiratory crepitation may also be audible. Associated hypoxia results in tachypnoea, central cyanosis, tachycardia and restlessness. Often there is reversal of the normal inspiratory-expiratory rhythm, with a pause after inspiration followed by an expiratory grunt. Chest X-ray reveals hyperinflation alone, or occasionally streaky areas of atelectasis. Children with bronchiolitis are often more ill than those with wheezy bronchitis: the clinical picture is unlike asthma – which in any case is unusual in this age group.

### *Croup (acute laryngotracheobronchitis)*

This is characterized by hoarseness, a 'barking' cough, an inspiratory stridor often with forced inspiration and intercostal indrawing: there may be signs related to bronchitis – usually consisting of symmetrical rhonchi perhaps with crepitations. There are many possible causative organisms mostly viral, including parainfluenza type 1, respiratory syncytial virus and influenza. If the child is afebrile and without signs of complications antibacterial therapy may be cautiously withheld whilst observing the effect of other therapy. Exhaustion, respiratory obstruction and respiratory failure are the main dangers.

### *Wheezy bronchitis*

This is an ill-defined but clinically recognizable syndrome which often occurs during the course of transient respiratory tract infections. Its relation to asthma is uncertain but it seems likely that infection triggers off

*Wheezing = audible rhonchi.

bronchospasm. Clinically there are generalized symmetrical rhonchi without stridor or hoarseness, and often there is a history of previous episodes. A chest X-ray may show hyperinflated but otherwise normal lungs.

The complications of the above three infections are similar – secondary pneumonia (recognized by clinical or X-ray deterioration), atelectasis, otitis media, exhaustion or respiratory failure.

Treatment of the above three syndromes is broadly similar. In mild cases, the importance of adequate hydration should be explained to the parents and arrangements made to provide humidified air (steam kettles etc.). Antispasmodics seem to help wheezy bronchitis but not bronchiolitis. Older children often feel more comfortable if nursed sitting up. If the illness is severe (resting pulse rate above 160/min, marked stridor, severe dyspnoea, cyanosis, shock) or the parents are not able to cope for any reason, the child should be admitted to hospital for more intensive humidified oxygen therapy, attention to the airway and for a chest X-ray to exclude chest complications. Rarely intubation or tracheostomy may be necessary.

### *Epiglottitis*

The incidence is highest in children aged 2–5 years. After an abrupt onset of fever and sore throat, gross swelling of the epiglottis occurs: *Haemophilus influenzae* bacteraemia (the most common causative pathogen) is often present. Often there may be an associated laryngitis or bronchitis. Epiglottitis is a medical (occasionally a surgical) emergency as respiratory obstruction may occur with devastating rapidity. Early epiglottitis may be suspected if a red, swollen, easily visible epiglottis is seen on inspection of a 'sore throat'. Later in the course of illness stridor may occur and, in cases which I have seen, the stridor sounds more proximal than that of croup. If epiglottitis is suspected, one should not attempt to visualize the epiglottis without full anaesthetic facilities as respiratory obstruction may be precipitated. A portable lateral neck X-ray may be useful in differentiating between croup and epiglottitis – an enlarged epiglottis is seen on X-ray in epiglottitis.

Treatment requires *urgent hospital admission:* an adequate airway must be assured and facilities for endotracheal intubation must be kept next to the patient. High dose intravenous ampicillin should be given and some would advocate steroids in addition.

### *Whooping cough (pertussis)*

This is a highly infectious disease with an incubation period of 10–16 days. It can affect *any* age but whoops only occur in young children. The onset is

gradual with nasal discharge and cough. The cough becomes more evident over about 2 weeks until the whooping stage occurs. During the whooping stage there are paroxysms of continuous expiratory coughing with production of tenacious sputum; during these paroxysms the child may become cyanosed (or even black) due to anoxia and hypoxic fits may occur. Eventually the paroxysm ends and a vast inspiration causes the characteristic whoop – a prolonged, high pitched 'suction noise'. The whooping stage lasts about 2 weeks and the recovery period thereafter is also about 2 weeks.

Even if whoops are not heard whooping cough may be suspected if there has been contact with whooping cough, if coughing occurs unpredictably in bouts, if there is vomiting at the termination of a coughing bout, and if there is production of sputum – whooping cough is one of the few disorders of children in which sputum is produced rather than swallowed. The finding of a blood lymphocytosis is highly suggestive of whooping cough. Isolation of *B. pertussis* from a pernasal swab confirms the diagnosis. However, such swabs grow *B. pertussis* in less than about 70% of patients during the whooping stage.

Complications are:

(a) *related to asphyxia*, which may be fatal,
(b) *infective* – bronchopneumonia with atelectasis and otitis media (suspect these if there is a fever in the whooping stage),
(c) *related to pressure changes* – inguinal and umbilical herniae, rectal prolapse, periorbital oedema, subconjunctival and other haemorrhages and (rarely) pneumothorax or mediastinal emphysema,
(d) caused by *central nervous system dysfunction* – hypoxic and 'idiopathic' whooping cough related brain damage may occur.

Infants below the age of six months are the most vulnerable to complications.

Domiciliary management rests upon careful explanation to the parents of the management of a paroxysm and the need for adequate nutrition and adequate fluid intake. Management of a paroxysm consists in positioning the child such that inhalation of vomit is unlikely and so that secretions in the mouth may drain: cough suppressants seem to have little to offer and on theoretical grounds may possibly be harmful. The affected child should be nursed in a quiet, constant temperature environment.

If complications develop or the parents cannot cope for any reason, hospital admission should be advised for constant observation and intensive humidified oxygen therapy if required. Such oxygen therapy can be achieved in an oxygen tent or in infants by using a transparent 'headbox' from which the infant can be removed rapidly when a paroxysm occurs.

The indications for antibacterial therapy are debatable, especially in the whooping stage when the organism may not be cultured. It seems logical to

give antibacterial therapy early in the course of the disease, before whooping occurs, in an attempt to prevent deterioration and to reduce infectivity. Once the whooping is established, there is no convincing evidence that antibacterial therapy helps the patient. The prophylactic administration of antibacterial therapy to close contacts is of unproven efficacy but again seems to be logical. If antibacterial therapy is given for whooping cough or for its prevention, erythromycin or co-trimoxazole are appropriate.

## RESPIRATORY INFECTIONS COMMON TO ADULTS AND CHILDREN

### *Bronchitis (acute)*

This is most common during winter months and initially consists of substernal discomfort, fever, an initial non-productive cough: later there may be mucopurulent sputum. Diffuse symmetrical rhonchi and occasional crepitations are heard on auscultation of the chest. The chest X-ray is usually normal or shows a vague increase in bronchovascular markings. Especially in patients with chronic bronchitis, an acute attack of bronchitis may merge with bronchopneumonia.

Uncomplicated acute attacks usually have a viral aetiology and aspirin plus a bronchodilator may be helpful. If secondary bacterial infection is suspected antibacterial therapy is indicated. *Haemophilus influenzae* is the bacterium most commonly implicated in acute exacerbations of chronic bronchitis: useful antibacterial drugs include ampicillin (or its derivatives), tetracyclines or co-trimoxazole.

### *Bronchitis (chronic)*

This is said to exist when a chronic recurrent cough with expectoration persists for more than three months of each year. It is essentially a disease of adults which is associated with cigarette smoking and air pollution.

Impairment of ciliary activity and excessive mucus production leads to obstruction of small airways with associated secondary bacterial infection. In an exacerbation there is usually audible wheezing and diffuse rhonchi. Cyanosis is variable: some patients eschew cyanosis and hyperventilate ('pink puffers') whereas others surrender to cyanosis and do not hyperventilate ('blue bloaters').

Patients with chronic bronchitis should avoid rapid temperature changes (such as entering unwarmed bedrooms) which may trigger off bronchospasm. Bronchodilator therapy is of use and antibacterial therapy should be used at the first sign of an exacerbation. Some would recommend prophylactic antibacterial therapy for those at risk. Continuous domiciliary oxygen has its advocates. Regular vaccination against current strains of influenza is essential in this group. Chest physiotherapy (postural drainage, percussion and breathing exercises) is valuable.

## *Common colds*

After an incubation period of 1–4 days there is sneezing, nasal obstruction and profuse rhinorrhoea which is initially mucoid but later mucopurulent. The nasal mucosa is boggy and swollen. Fever is minimal or absent. The common cold syndrome can be caused by a multitude of agents including rhinoviruses (10–25% of colds), coronaviruses, some coxsackie and Echoviruses, respiratory syncytial virus (usually in adults), adenoviruses and mycoplasma.

Treatment is symptomatic; decongestants containing an antihistamine and atropine-like compounds are useful in drying up secretions. Vitamin C is of unproven efficacy. Antibacterial drugs are not indicated for an uncomplicated attack.

## *Influenza*

After an incubation period of 1–4 days there may be an abrupt onset of fever, malaise, shivering, hacking cough, musculoskeletal pains, sore throat, slight nasal discharge and sneezing. No doubt many attacks are less severe. Influenza should not be confused with the common cold – in which patients are usually afebrile and have a copious nasal discharge.

Classical influenza usually lasts about 4 days and if fever symptoms or signs persist for longer suspect primary influenzal pneumonia or secondary bacterial pneumonia. Secondary bacterial invasion is common with penicillin resistant *Staph. aureus* and should be treated with antibacterial drugs.

Diagnosis of influenza is confirmed by isolation of the virus or by serological tests on paired sera.

The treatment of an uncomplicated attack of influenza is symptomatic – aspirin exacerbates perspiration and codeine seems a useful alternative: codeine is also a cough suppressant – which may or may not be a desirable property. Amantidine may also be useful prophylactically, particularly if at risk patients have Parkinson's disease which also improves.

Prevention by vaccination will usually protect the majority of those at risk for about 3–6 months, providing the vaccine includes antigens derived from the current viral strains.

## *Laryngitis*

In children, laryngitis may be the predominant part of the croup syndrome. In adults, infection related laryngitis may present as a seemingly distinct entity although the causative organisms (usually viruses) are present elsewhere in the respiratory tract. In adults, there is a barking cough, hoarseness and possibly laryngeal tenderness. Fever and dyspnoea are often minimal or absent.

Treatment includes vocal rest, water saturated air inhalations and oxygen if necessary. If there are other signs suggesting bacterial respiratory tract infection, appropriate antibacterial therapy should be given. Diphtheria and tuberculosis should not be forgotten as possible causes of laryngitis.

## *Otitis media*

Given an intact tympanic membrane acute otitis media is almost invariably secondary to infection derived from the upper respiratory tract; blockage of the eustachian tube or viral invasion of the middle ear may predispose to bacterial infection. Pus, if imprisoned, may force its way into the pneumatized petrous temporal bone or cause perforation of the tympanic membrane. Acute otitis media is most common in children – the eustachian tube seems more vulnerable to obstruction as it is horizontal and because its entrance is surrounded by lymphoid tissue. Clinically there is an abrupt onset of fever, ear ache, conductive deafness and (especially in *Strep. pyogenes* or *Strep. pneumoniae* infection) the tympanic membrane is bulging and fiery red such that the usual bony landmarks may be obscured and the normal light reflex of the tympanic membrane is lost. Otitis media in infants may present in a 'non-focal' fashion with fever, 'unhappiness', failure to feed, meningism, vomiting or diarrhoea.

*Haemophilus influenzae* is a frequent causative organism in the under fives, and ampicillin or its derivatives are usually the drugs of choice. In older patients, *Strep. pneumoniae*, *Strep. pyogenes* and *Staph. aureus* become more frequent and treatment with the narrower spectrum penicillin is reasonable: erythromycin, a cephalosporin or co-trimoxazole are suitable alternatives in penicillin-allergic patients. Whichever antibiotic is prescribed the need for analgesia should not be forgotten.

Other treatments which may have to be considered in difficult, recurrent or unresponsive cases include myringotomy or adenoidectomy.

Chronic otitis media usually occurs in association with tympanic membrane perforation. Gram negative bacteria including *proteus*, *klebsiella* and *pseudomonas* are common pathogens. Antibacterial therapy (if indicated) should be determined from the results of culture. Meticulous aural toilet, antiseptic agents and later myringoplasty may be required.

## *Pneumonias*

These can be defined as inflammation of the lung with alveolar involvement due to invasion by microorganisms. Although the following classification is not uniformly reliable and is not a substitute for clinical experience, it will help the inexperienced to arrive at sensible therapeutic decisions.

In each case of pneumonia it is essential to attempt to obtain a specimen of sputum and, in hospital practice, to obtain a blood culture prior to initiating treatment.

An immediate Gram stain of sputum may be diagnostically helpful as clusters of gram positive organisms suggest staphylococci, gram negative rods suggest *H. influenzae* unless klebsiella is a possible pathogen, and gram positive cocci in short chains are likely to be *Strep. pneumoniae.*

## 'TYPICAL' BACTERIAL PNEUMONIAS

### *Classical lobar pneumonia*

Classical lobar pneumonia with diffuse involvement of one or more lobes presents with fever, a dryish cough (perhaps with rusty coloured sputum), dyspnoea and pleuritic chest pain. Herpes febrilis is common. Clinically there are signs of consolidation (chest movement reduced, dullness of the percussion note, bronchial breath sounds, increased vocal resonance and whispering pectriloquy). There is no mediastinal displacement. Later signs of pleural effusion may follow (reduced or absent breath sounds, diminished vocal resonance and a stony dull percussion note). Particularly in children, upper lobe pneumonias may produce delirium or meningism. Chest X-ray reveals diffuse opacity delineated by interlobar fissures. Before initiating antibacterial therapy, sputum and blood cultures should be obtained and a Gram stain of sputum performed. *Strep. pneumoniae* is frequently the causative organism and treatment is with penicillin which should initially be given parenterally (with the exception of certain geographical areas, pneumococci are almost invariably sensitive to penicillin). Oxygen should be given if indicated and analgesia and cough suppression can both be achieved by analgesics such as codeine. If there is a pre-existent chronic respiratory disease, oxygen therapy should be introduced with monitoring of blood gases; some patients may have abandoned their normal carbon dioxide mediated respiratory drive and be dependent on hypoxic drive, and exceeding their normal oxygen concentration by means of oxygen administration will result in hypoventilation and further carbon dioxide retention.

### *Classical bronchopneumonia*

Classical bronchopneumonia is rare in previously fit individuals unless there are predisposing factors. In young children predisposing factors include any lower respiratory tract infection and disorders such as mucoviscidosis; in adults an attack of influenza or pre-existent lung disease may predispose. Bronchopneumonia often starts as an acute bronchitis but fever and discoloured sputum develop. Coarse crepitations may be present

in one or more areas of the chest and signs are usually bilateral. There may also be signs of lobar or lobular consolidation.

*Haemophilus influenzae* is frequently associated with exacerbations of chronic bronchitis. A lobar or more commonly bronchopneumonic picture may result. Ampicillin is usually the drug of first choice, but co-trimoxazole is useful if ampicillin resistance is suspected.

In previously unfit individuals, various other pathogens may be implicated. Diabetes or alcoholism may predispose to *Klebsiella* pneumonia in which there is a rapid progressive necrotizing infection with possible cavitation: a suggestive feature on X-ray is expansion of the affected lobe caused by extensive oedematous inflammation. Drug therapy usually rests on aminoglycosides, cephalosporins or chloramphenicol which may also have a place. Combination therapy is often utilized to prevent rapid emergence of resistant strains.

*Pseudomonas aeruginosa* tends to cause hospital acquired pneumonias characterized by multiple small abscesses possibly due to vasculitic lesions. Drug therapy is with an appropriate aminoglycoside (usually gentamicin or tobramycin) either alone or in combination with carbenicillin.

*Staph. aureus* pneumonia usually occurs in the very young, the very old or anyone after an attack of influenza. Drug addicts who unwittingly inject themselves with the organism may present with staphylococcal pneumonia, possibly with an underlying right sided endocarditis. There may be patchy involvement of several pulmonary segments, a predominantly lobar picture, or multiple abscesses perhaps with fluid levels therein – pneumatocoeles. Drug therapy, before the results of sensitivity testing are known, should be with a drug or drugs not inactivated by penicillinase.

Tuberculous pneumonia commonly arises in upper lobes and one should suspect active tuberculosis if there is cavitation and calcification with fluffy surrounding areas on X-ray (lung neoplasms do not often calcify). If a tuberculous abscess ruptures into a bronchus, tuberculous bronchopneumonia results (galloping consumption).

Cavitation in an area of pneumonia should make one suspect staphylococcal pneumonia, tuberculosis, gram negative pneumonias (especially klebsiella), anaerobic pneumonias or necrotizing neoplasms.

### *Lung abscesses*

Severe pneumonic illnesses can cause necrotic areas which develop into lung abscesses. Precipitating causes include influenza, inhalation of food or vomit, foreign body inhalation, underlying carcinoma, septic pulmonary emboli, or right sided endocarditis. The main clinical clue to the presence of lung abscesses is the production of large amounts of foul smelling sputum. On chest X-ray there is cavitation, possibly containing fluid. Complications include empyema formation, metastatic spread of infection,

fungal superinfection, or haemorrhage. In about 90% of lung abscesses, anaerobic organisms, such as bacteroides, fusobacteria or anaerobic streptococci, may be isolated; other organisms include *Staph. pyogenes*, klebsiella species, *Strep. pyogenes*, gram negative enteric bacilli, *Myco. tuberculosis*, amoebae, nocardia or fungi. It is essential that adequate specimens of sputum are obtained and are cultured aerobically, anaerobically, for fungi, and for *Myco. tuberculosis*: transtracheal aspiration may be required to prevent contamination with commensal mouth anaerobes.

Conservative treatment is with postural drainage and antibacterial therapy: chloramphenicol is active against most anaerobes whereas penicillin is only active against a few anaerobes. Metronidazole, cefoxitin, clindamycin or lincomycin are also possible drugs for use against anaerobes. If conservative therapy seems unlikely to succeed, surgery should be considered.

## 'NON-TYPICAL' PNEUMONIAS

By the term 'non-typical' I refer to pneumonias that do not conform to the recognizable standard bacterial pneumonias; pneumonias which fall into this category include those caused by:

adenoviruses,
enteroviruses,
influenza,
Legionnaire's disease,
mycoplasma,
ornithosis, and
Q fever

These pneumonias may be suspected in patients:

(1) who have no obvious predisposition to standard bacterial pneumonia,
(2) who are young, previously fit, adults,
(3) who have failed to respond appropriately to therapy for standard pneumonia. In particular, a persistent protracted irritating cough despite penicillin therapy should suggest ornithosis, mycoplasma or Q fever. Another possibility is whooping cough, which may occur in adults but without the whoop;
(4) who have marked extrapulmonary symptoms, and
(5) have sputum that is mucoid.

### *Specifically suggestive points*

Mycoplasma pneumonia, unlike influenza or Q fever, presents with a *gradual onset* of malaise and fever. Headache is a common accompaniment to Q

fever, ornithosis and mycoplasma pneumonias. Musculoskeletal aches and pains are common in influenza or ornithosis. In ornithosis there may be diarrhoea, possibly haemoptysis and mental symptoms (such as confusion, delirium and stupor). In ornithosis there may be a history of contact with birds, whereas in Q fever there may be a history of exposure to farm animals.

In all the 'non-typical' pneumonias listed above, the chest X-ray may have patchy areas with uniform or mottled opacity of non-lobar distribution and, particularly in the case of ornithosis or mycoplasma pneumonia, these are often more extensive than physical examination would suggest.

A rash may occur in some adenoviral, enteroviral and mycoplasma pneumonias, whereas there is no rash in influenza or Q fever pneumonias. In adenovirus pneumonia a sore throat and pharyngeal infection may also be evident.

The erythrocyte sedimentation rate may be greatly raised in ornithosis, Legionnaire's disease and mycoplasma pneumonias. In mycoplasma pneumonia cold agglutinins may be present in over 50% of patients and a rapidly performed test* can provide strong circumstantial evidence for mycoplasma infection, although other 'non-typical' pneumonias may occasionally induce cold agglutinins.

Suspicion of these 'non-typical' pneumonias is essential as treatment is often available utilizing drugs that are not first-line drugs for 'typical' bacterial pneumonias.

Tetracycline is active against ornithosis, mycoplasma and Q fever.

Erythromycin is effective against mycoplasma and *Strep. pneumoniae* and is useful if there is doubt as to whether a pneumonia is mycoplasmal or pneumococcal in aetiology. Erythromycin seems to be the drug of choice against Legionnaires' disease.

Chloramphenicol is effective against ornithosis, mycoplasma and Q fever – although many would prefer not to use this drug because of the risk of aplastic anaemia.

Non-typical pneumonias in the immunocompromised may be caused by cytomegalovirus, fungi, nocardia or *Pneumocystis carinii*.

### *Sinusitis*

Sinusitis commonly follows coryza, vasomotor rhinitis, deviation of the nasal septum, polyps, tumours and dental abscesses. There is throbbing pain, tenderness on pressure local to the involved sinus and nasal obstruc-

*Add a few drops of blood to the citrate in a commercial prothrombin tube to make a 50/50 mix and place the tube in the freezer section of a refrigerator. Remove the tube after a few minutes. If the tube is tilted and rotated obvious agglutination will be seen on the tube wall if cold agglutinins are present. The agglutination disappears on warming.

tion may be present. Copious purulent nasal discharge occurs if there is rapid drainage of an infected sinus and if the discharge has a foul smell anaerobic infection should be suspected. In maxillary sinusitis the antrum fails to transilluminate. An X-ray may reveal opacification of the sinus, possibly with a fluid level.

Complications include spread of infection with abscess formation, cavernous sinus thrombosis, osteomyelitis of the skull, or meningitis.

Therapy consists of analgesia, water-saturated warm air inhalations, vasoconstrictor agents (to assist ostial opening) and appropriate postural drainage to assist drainage of the affected sinuses. In an acute episode most patients should be given antibacterial therapy. The common pathogens causing acute sinusitis are *Haemophilus influenzae, Strep. pneumoniae* and staphylococci but both mixed infections and anaerobic infections are possible. Initial drug therapy can be with ampicillin or its derivatives, co-trimoxazole or erythromycin. Penicillin is often used successfully although *Haemophilus influenzae* is insensitive. Metronidazole is indicated if anaerobic infection is suspected clinically.

## SORE THROATS AND MOUTHS (Table 4)

**Table 4 Infection related sore throats and mouths**

| |
|---|
| *Viruses* |
| Adenoviruses |
| Aphthous ulcers |
| Chickenpox |
| Cytomegalovirus |
| Glandular fever |
| Hand, foot and mouth disease |
| Herpangina |
| Herpes simplex |
| Herpes zoster |
| Influenza |
| Measles |
| Mumps |
| Rubella |
| |
| *Bacteria* |
| Streptococcal infections |
| Ludwig's angina |
| Vincent's angina |
| Diphtheria |
| |
| *Other causes* |
| *Candida* |
| Reiter's syndrome |
| Stevens–Johnson syndrome |
| *Toxoplasmosis* |

## *Bacterial causes*

Sore throats are a not uncommon problem in general practice: the results of throat swab culture and blood tests are rarely available rapidly enough to be of immediate diagnostic help, and thus the decision to give an antibacterial drug has to be made on the clinical findings. In practice the common problem is to decide which sore throats are streptococcal in aetiology and should be treated with penicillin. In the absence of penicillin hypersensitivity there is rarely an indication to treat sore throats with any antibacterial drug other than penicillin: most penicillin derived drugs have an unnecessary wide spectrum of activity, and ampicillin with its derivatives usually cause rashes if patients have undiagnosed infectious mononucleosis.

Ideally a throat swab from all patients should be taken for bacterial culture into Stuart's transport medium. If streptococci are grown treatment should be continued for a total of 10 days, but if no streptococci are grown then treatment can be discontinued. A positive culture for streptococci from a 'quiet' throat indicates a carrier state which under normal circumstances requires no therapy.

### *Lancefield group A β-haemolytic streptococci (Strep. pyogenes)*

These may cause pharyngitis, tonsillitis or quinsy (peritonsillar abscess) with possible sequelae such as rheumatic fever or glomerulonephritis. In a classical streptococcal sore throat there is an abrupt onset illness with fever and headache: earache is typical but nasal stuffiness and discharge are not prominent. Dysphagia is usually caused by lancinating pain rather than by obstruction. On examination there is redness and congestion of the throat, the tonsils are enlarged with 'cheesy' exudates emanating from the tonsillar crypts. *Such exudates do not spread beyond the tonsils.* Particularly in young children exudates may not be present. Cervical lymph node enlargement is usually noted after the onset of symptoms and signs local to the throat. Scarlet fever is usually caused by group A β-haemolytic streptococcal tonsillitis associated with a white-then-red 'strawberry' tongue and the characteristic rash. A unilateral quinsy causes midline shift and gross swelling of the pharyngeal structures in addition to the symptoms and signs of streptococcal pharyngitis.

Which sore throats should be treated as if they were streptococcal in aetiology? Certainly those in which the appearances suggest streptococcal infection (typical streptococcal exudates can occur in viral infections but should be presumed to be streptococcal pending throat swab results). Patients with streptococcal complications should be treated, including patients with a quinsy (most resolve with intramuscular penicillin therapy without the need for incision), retropharyngeal abscess, otitis media or

scarlet fever. Patients who have defective host defences or a previous attack of rheumatic fever or chorea should be treated and, finally, patients whose occupation brings them into close contact with susceptibles – nurses and doctors for example – should be started on a course of penicillin, and should stay off work pending the results of the throat swab.

Treatment of streptococcal sore throats is with penicillin G (intramuscularly) or penicillin V (orally) depending upon the severity of the infection. In general practice triple preparations of penicillin which contain a short, medium and long acting penicillin are a useful one injection treatment as patients often discontinue oral therapy once the symptoms have subsided (with attendant risk of relapse). Erythromycin is a useful alternative to penicillin in the penicillin allergic patient.

### *Ludwig's angina (submandibular cellulitis)*

This is commonly streptococcal in aetiology but occasionally is caused by anaerobes. The main danger is that of respiratory obstruction. Treatment is usually with penicillin, but some would also use steroids if respiratory obstruction was impending. Passage of a naso-laryngeal tube may also 'buy time'.

### *Vincent's angina*

Vincent's angina is caused by a double infection with *Fusobacterium fusiforme* and *Borrelia vincenti* resulting in an acute gingivostomatitis, often with ulceration and a grey pseudomembrane with bleeding on attempted removal. It usually occurs in debilitated or leukaemic patients. Treatment is with antiseptic mouthwashes, oral hygiene, penicillin or metronidazole. Tetracyclines are useful if the patient is not immunocompromised.

### *Diphtheria*

Although uncommon if all the population is immunized, it is a diagnosis not to be missed. Pyrexia is usually low grade or absent and in classical cases the characteristic membrane is sharply demarcated with a wrinkled edge, not confined to the tonsils, thick, homogeneous, firmly adherent and later surrounded by a narrow zone of inflammation. In mild tonsillar diphtheria there may be only a small spot of yellowish white membrane with a wrinkled edge: the colour is rarely as pure white as an early infectious mononucleosis exudate may be. In pharyngeal diphtheria the membrane spreads away from the tonsils. This membrane usually has a thick edge and its colour changes rapidly to a green and black hue. However, the membrane may be only a thin, slimy film which is easily missed on a cursory examination. Ulceration is not a feature and bleeding occurs

on attempted removal of the membrane. There may be an associated non-painful 'bull-neck' with regional adenitis and the sufferer may be generally floppy and ill: sequelae result from either the occurrence of unpredictable respiratory obstruction due to membrane extension or from the effects of the powerful exotoxin on the heart or the nervous system.

Treatment is by urgent administration of antitoxin *on suspicion* of the diagnosis, protection of the airway, and by penicillin to eliminate the organism. (Erythromycin is an alternative in the penicillin allergic and is also used to treat diphtheria carrier states.)

Other bacteria such as *Haemophilus influenzae* or *Strep. pneumoniae* may cause sore throats but as both may be isolated from normal throats their pathological significance is often uncertain. Group C and Group G β-haemolytic streptococci may be responsible for pyogenic sore throats.

### *Viral sore throats and mouths*

Viral infections cause the majority of sore throats and mouths. On occasion viral infections may be clinically indistinguishable from streptococcal infections and indeed may predispose to secondary bacterial infections. In viral sore throats, the white cell count is commonly normal or low and the erythrocyte sedimentation rate is often normal.

Treatment of viral sore throats or mouths is usually symptomatic: hydrocortisone pellets are useful in aphthous ulceration, and local anaesthetics are often useful for painful lesions around the gums despite the risk of sensitivity developing. I find whiskey has a soothing effect on local lesions and allays despair if subsequently swallowed. Antiseptic mouth washes and gargles may be symptomatically helpful although their action can be only superficial. Aspirin gargles are often appreciated.

The above therapies can help to assuage the constant demand for 'an antibiotic please, doctor.'

#### *Adenoviruses*

These cause varied clinical pictures depending on the particular adenovirus involved: one characteristic syndrome comprises fever, pharyngitis and conjunctivitis (pharyngoconjunctival fever).

#### *Aphthous ulcers*

These are irritating, painful, small (circa 0.5 cm in diameter) ulcers or vesicles which are presumed to be caused by viruses. Individual ulcers begin as shallow erosions with a yellowish raised border surrounded by a narrow, bright red zone. They may be related to mental stress, physical stress or underlying organic disease. As an isolated finding, they usually have no serious significance. Recurrent attacks are common.

*Chickenpox*

Oral poxes occur in association with the characteristic skin rash.

*Cytomegalovirus*

A monospot-negative glandular fever syndrome may result with a (relatively uncommon) exudative tonsillitis.

*Infectious mononucleosis*

In the classical anginose form the oropharynx may be reddened and the tonsils enlarged. Initially the tonsillar appearances are non-specific. Later the characteristic exudate formation may develop: initially, this is usually strikingly white and is confined to the enlarged tonsils, is superficial and can be pushed off easily to leave a red, but non-bleeding area beneath. Palatal petechiae are characteristic of infectious mononucleosis but not diagnostic. If cervical lymph node enlargement occurs before the onset of a sore throat, this favours infective mononucleosis rather than streptococcal infection. In infectious mononucleosis the throat usually is less painful than an uncomplicated streptococcal sore throat and dysphagia is more often caused by obstruction than by pain.

*Hand, foot and mouth disease*

This is commonly caused by coxsackie A16 virus and consists of slightly painful bright red macules or vesicles surrounded by a red ring about 3–8 mm across. Lesions are present on the feet, hands and in the mouth, particularly on the buccal mucosa and palate: unlike *Herpes simplex* infection there are usually no lesions on the outside of the lips or face.

*Herpangina*

This is usually caused by a coxsackie A virus and results in an abrupt onset sore throat and dysphagia. The fauces and pharynx are red and distinct greyish papules 1–2 mm in diameter or shallow ulcers up to 5 mm in diameter are surrounded by a red halo. These lesions are present almost exclusively in the back of the mouth, a finding unusual in *Herpes simplex* infection.

*Herpes simplex*

After a primary infection in children, an acute gingivostomatitis may result with greyish yellow ulcers, often serpiginous, on an inflamed base and predominantly affecting the front of the mouth. Outside the mouth, there

are clusters of vesicles which rapidly crust. The tongue is often coated and there may be an associated cervical lymph node enlargement and excessive salivation with secondary implantation of infection due to dribbling may occur.

Recurrent *Herpes simplex* usually involves the lips and systemic symptoms are rare.

### *Herpes zoster*

Herpes zoster, if of maxillary or mandibular distribution, may present as a unilateral oral pain which is rapidly followed by the classical shingles eruption.

### *Influenza*

Sore throat is not usually a major complaint compared with the fever, chills, malaise, headache or myalgia typical of this illness.

### *Measles*

Children especially may complain of a sore throat as part of the prodrome. There will be an associated conjunctivitis and coryza and Koplik's spots should be visible at this stage: these resemble grains of salt scattered on the reddened buccal mucosa.

### *Mumps*

This illness may cause a mild sore throat due to decreased salivation: parotitis may be evident or subsequently develop and the affected salivary duct orifices are often prominent and reddened.

## *Other causes of sore throats and mouths*

### *Behçets syndrome*

This syndrome comprises episodes of oral and genital ulceration of vasculitic aetiology.

### *Candida (Thrush)*

Candida causes creamy white areas which cover inflamed mucous membranes. Common predisposing causes include antibacterial therapy, diabetes mellitus, immune deficiencies, the contraceptive pill and steroid therapy. Treatment is with either nystatin or amphotericin B.

### *Reiter's syndrome*

Reiter's syndrome is strictly recognized by the triad of arthritis, urethritis and conjunctivitis but some patients also have mucocutaneous manifestations which may include a sore mouth.

### *Stevens–Johnson syndrome*

Usually there is inflammation and ulceration of the mouth and mucous membranes (including those of the conjunctivae and genital tract) in association with erythema multiforme. It is a reaction pattern to a variety of stimuli. Steroids may be useful therapy if systemic toxicity dictates or spontaneous resolution is slow.

### *Toxoplasmosis*

A monospot-negative glandular fever syndrome may occur in which pharyngitis is not a common symptom.

## *Suggested further reading*

Hoeprich, P.D. (1977). *Infectious Diseases, Sections 4, 5 and 6.* (London: Harper and Row)