Current Practice

RESPIRATORY TRACT DISEASE

Acute Bronchitis—Aetiology, Diagnosis, and Management

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Acute bronchitis is an acute inflammation of the larger air passages, including the trachea and the large- and medium-sized bronchi. The smaller bronchi and bronchioles may also, however, be involved on occasion, with the development of areas of lobular atelectasis leading to patches of alveolitis. Thus the dividing line between an acute bronchitis affecting the main bronchi only and a pneumonitis involving the small peripheral airways is sometimes very tenuous.

Predisposing Factors

Acute bronchitis may occur at any time of the year, but it is especially common in the winter months, when cold, damp, foggy weather and minor epidemics of febrile catarrh prevail and seem particularly to predispose towards its development. It is especially common in the very young and the very old, when it is frequently a more serious condition than at other ages. Chronic debilitating illnesses—notably chronic cardio-renal disease and chronic pulmonary disorders—are conditions which often permit of the ready development of an associated bronchitis. Likewise dusty occupations, a smoky environment, and perhaps heavy cigarette-smoking may also predispose to an acute attack, as does the presence of a septic focus in the upper respiratory tract; but above all some people seem to have an especial constitutional liability towards the development of recurrent attacks of acute bronchitis, and in these persons there is usually a family history of bronchitis, respiratory tract catarrh, or general "chestiness."

Aetiology

Acute bronchitis may present as a single isolated episode or as one of a number of recurring illnesses, or it may arise as an acute infective exacerbation on a background of established chronic bronchitis. Classification of the acute respiratory infections according to specific causative organisms is a formidable task in the present state of our knowledge. More and more the evidence appears to suggest that many if not most of these acute infections are of virus origin,¹ while Lynne Reid² has shown that hypertrophy and hyperplasia of bronchial mucus secreting cells, typifying chronic bronchitis, will occur in rats exposed to a gaseous chemical irritant for four to six weeks. She has also shown that when bacterial colonization occurs the organisms were usually the normal flora found in the naso-pharynx. A similar situation may develop following acute viral infections of the bronchi, with subsequent bacterial infection from upper respiratory tract commensals. A clear understanding of the significance of bacterial infection in the acute respiratory disorders therefore becomes more necessary if anti-

biotics are to be used intelligently and rationally in the management of these conditions. But incontrovertible facts are few, and agreement among different workers inconstant. This is particularly true of the acute respiratory infections in children, where there are many difficulties in obtaining adequate specimens to provide reliable information. The place of *Staphylococcus aureus*, pneumococcus, and *Haemophilus influenzae*—particularly its capsulated strains—has been increasingly stressed in recent years, while the importance of the adenoviruses, especially in acute bronchitis in adults, and of these and the para-influenza myxoviruses, particularly in children, has also been emphasized.

Classification

It is convenient for the clinician, and indeed useful for descriptive purposes, to consider acute bronchitis, as has been done by Scadding,³ according to a simple clinical classification.

Acute Bronchitis of Uncertain Origin

In the past it has been customary to consider this group according to the severity of the infection or to the condition of the sputum. Accordingly it has been subdivided into catarrhal, suppurative, and plastic varieties. Acute catarrhal bronchitis is usually considered to be due to a descending infection from the upper respiratory tract, presumably of virus origin, though in some cases no relation to any such infection can be detected. It is usually a mild disorder in which evidence of bacterial invasion is inconstant and non-specific. Though both the catarrhal and suppurative forms of acute bronchitis are often associated with a purulent sputum at some time in the course of the disease, acute suppurative bronchitis is generally a more severe form of the illness. It is characterized by the early appearance of purulent sputum, profound toxæmia, and a relatively high mortality rate; it tends to occur sporadically, particularly in association with epidemic influenzal conditions. Many of these cases may progress to overt pneumonia, usually of a scattered distribution, and may sometimes be due to a drug-resistant staphylococcus. Acute plastic bronchitis, on the other hand, is characterized by the expectoration of bronchial casts or solid fibrinoid balls or lumps. These lumps or casts consist mainly of mucin with little fibrin; hence it is misleading to refer to this condition as fibrinous bronchitis. The effect of these mucinous lumps in the bronchi is to cause obstruction to the airways, with distal segmental or, more uncommonly, lobar atelectasis producing dyspnoea and perhaps cyanosis. It is doubtful whether this distinction according to the quality or degree of purulence of the sputum is warranted as representing different varieties of acute bronchitis. Catarrhal and suppurative forms of bronchitis have on occasion been related to different types of virus infection, while

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the plastic type of sputum may be found in bronchitis with heart disease, tuberculosis, asthma, or the pulmonary mycoses.

Bronchitis of uncertain origin may occur in infants and children as a simple bronchitis or bronchiolitis, or as an influenza-like illness of the upper respiratory tract with an associated bronchitis, with or without conjunctivitis, laryngitis, or tracheitis. The differences may well be due to infection with different types of viruses. In acute *laryngo-tracheo-bronchitis of infants* obstruction of the larynx and trachea is common and may simulate laryngeal diphtheria. It is often associated with episodes of severe dyspnoea, obstructed and noisy respirations, and indrawing of the intercostal spaces producing respiratory distress. This obstruction is due to oedema of the subglottic tissues, while the laryngo-tracheal mucosa is deeply congested, inflamed, and swollen. Bacteriological findings are inconstant, but it is now believed to be due to infection with para-influenza viruses types I, II, and III and E.C.H.O. type II. *Acute bronchiolitis of infancy* usually develops following contact with an adult with a head cold. There is nasal discharge, slight cough, and anorexia. Within a few days the whole process may resolve, but in some patients the condition deteriorates with severe unproductive cough, much bronchospasm, and general respiratory distress, which is sometimes associated with cyanosis. Froth may appear at the mouth, and there are widespread rhonchi, basal crepitations, and poor air entry to the lung bases. The tip of the spleen is frequently palpable, and should heart failure develop the liver also becomes enlarged. Surprisingly little if any fever is to be observed in this condition, which is potentially if not actually a serious illness for the infant. Sandiford and Spencer⁴ have reported that, although no virus could be recovered from infants with epidemic bronchiolitis, nevertheless complement fixing antibody titres to respiratory syncytial virus were found to rise in paired serum specimens from 22 out of 32 patients whom they observed.

In adults acute bronchitis of uncertain origin may be due to one of the epidemic febrile catarrhal syndromes, or it may arise as a direct aspirational bronchitis from a focus of pyogenic infection in the upper airways—typically following anaesthesia—or because of conditions associated with dysphagia, as in achalasia, cardiospasm, or a pharyngeal pouch. It may also present in association with pre-existing bronchopulmonary disease. Many of these acute exacerbations are probably initiated by virus infections of the common-cold type, after which rapid colonization of the lower airways with *H. influenzae*, pneumococci, and other pathogens takes place. Delayed resolution in acute bronchitis may draw attention to the presence of underlying pulmonary or associated systemic conditions. It may indicate among others a single episode of pulmonary eosinophilia (Loeffler's syndrome), while persistent and recurrent wheezing and bronchospasm may draw attention to one of the numerous chronic pulmonary eosinophilias such as a pulmonary fungous disease, or one of the collagen disorders. Finally, in patients with chronic cardiac or renal disorders, episodes of acute bronchitis readily ensue in association with disturbances of perfusion and pulmonary haemodynamics.

Acute Bronchitis Associated with Specific Infections

In many of the specific fevers acute bronchitis is a common accompaniment, and indeed part of the specific fever itself. Bronchitis is always present in influenza of moderate severity, while both measles and whooping-cough may present with bronchitis which is primarily due to the specific causative organism. Indeed, in the early stages of measles the chest symptoms may so predominate that it may be mistaken for a simple bronchitis. The bronchi may also be directly involved in diphtheria, in smallpox, and in the enteric fevers, and on rare occasions the particular enteric organism has been recovered from the sputum.

Bronchitis Associated with Aerial Pollutants

Bronchitis may arise from dust inhalation, or from exposure to noxious gases. It has long been held by some workers that prolonged inhalation of dust, particularly dust of occupational or industrial origin, produces changes in the respiratory mucosa and predisposes to the subsequent development of bronchitis and chronic broncho-pulmonary disorders. The work of Lynne Reid,² demonstrating the changes in the mucous glands of rats exposed to fumes of sulphur dioxide in concentrations 400 times or more greater than any fog, suggestive of those seen in chronic bronchitis, is of particular interest. However, the Medical Research Council Committee⁵ has been unable to demonstrate any significant association between the prevalence of chronic bronchitis and dust exposure in coal-miners. On the other hand, there is little doubt in the minds of the industrial worker or the clinician working in the English industrial north and midlands, that the presence of heavy concentration of iron and steel foundries, of light and heavy engineering, of printing and tailoring, and of other similar dusty industries is related to the development of bronchitis in both its acute and chronic forms.

Exposure to the dusts from heavy metals like beryllium and similar compounds causes bronchopulmonary illnesses, of which an acute bronchitis is a common manifestation. Not only are metallic dusts of importance, but dusts of vegetable matter may cause particular syndromes of acute bronchitis, as in farmer's lung, or exacerbations of pulmonary fungous disease.

Though the development of bronchitis forms only part of the picture of poisoning following exposure to noxious gases used in warfare, other atmospheric pollutants are a more frequent and potent source of bronchitis. Of these smoke from the combustion of coal in open fireplaces in high density residential areas, with the development under appropriate conditions of fog, is probably the commonest in predisposing to bronchitis.

Pathology

Congestion and oedema of the bronchial mucosa are the first changes to occur, at which time secretion is scanty, though this increases in a few days owing to hyperactivity of the goblet and mucus-producing cells. These secretions become increasingly purulent, depending on the degree and type of secondary infection. Epithelial desquamation takes place, and in severe cases necrotic changes may be observed to extend down to the bronchioles, causing obstruction of the terminal airways and leading to patchy atelectasis or consolidation due to an associated alveolitis. In most cases recovery of the mucous membranes is complete, but some episodes of acute bronchitis heal by scarring, with permanent destruction of the mucosa and some related alveoli, thus permitting the more ready development of further attacks of acute bronchitis, and leading almost inevitably to chronic bronchitis and obstructive airways disease.

Most cases are due to infection originating in the upper respiratory tract and descending to affect the tracheobronchial tree. A traumatic or chemical bronchitis may arise from exposure to certain irritant gases or vapours, or from atmospheric pollutants, which will also produce minor transient damage to the bronchial epithelium with paralysis of ciliary action, and again will permit of ready bacterial infection from the upper respiratory tract.

Clinical Features

In view of the many causes of acute bronchitis the clinical picture will accordingly be modified by any associated factors or circumstances. Characteristically acute bronchitis is ushered in with malaise, possibly a mild fever, weakness, shivering, and prostration. Very soon, usually within 24 hours, a dry, unproductive cough develops, often associated with frontal head-

ache, aching in the limbs, photophobia, and sometimes a feeling of soreness or a burning type of retrosternal discomfort. After about 24 to 48 hours thick purulent or mucopurulent sputum is produced, often in considerable amount. There is frequently evidence of an upper respiratory tract catarrh, sometimes in association with a sore throat and injection of the fauces, while the eyes may be red, suffused, and painful. Dyspnoea is usually not marked except in the acute respiratory distress syndromes, particularly in infants and the aged or when the smaller bronchi and bronchioles are involved, but wheezing may be a common feature at all ages. Moist sounds are characteristically heard at the lung bases, but there may also be evidence of bronchospasm to a greater or lesser degree, which may be minimal when the infection is limited to the main bronchi, but infection of the smaller air tubes will produce many rhonchi all over the lung fields. As secretions begin to accumulate rales may be heard, at first as coarse crepitations, and later, as the secretions increase, as bubbling rales, mainly at the lung bases. The chest x-ray film shows only minor catarrhal changes without any specific characteristic appearances. In the more severe cases the broncho-vascular markings may be accentuated with some slight enlargement of the hilar shadows. Small areas of atelectasis or consolidation may on occasion be seen owing to obstruction of the airways. If the bronchioles are involved a fine stippled mottling may be seen which gradually merges into a diffuse bronchopneumonia. From the initial invasion with the virus and the development of the early prodromal symptoms a period of about 24–48 hours elapses before there is evidence of overt bacterial infection of the bronchial tree. In the ordinary case of simple adult bronchitis the whole acute process is usually over within seven days, though residual cough and mucopurulent sputum may be persistent for another 7 to 10 days. In the more severe cases the clinical features and duration resemble bronchopneumonia, which can often only be excluded by radiography.

Diagnosis

Diagnosis usually presents no difficulty, especially if there is a clear history of onset following upper respiratory symptoms or exposure to an influenza-like illness. But it is important to exclude any primary precipitating disorder. Bronchitis due to the specific infections must be excluded, particularly in children, and the buccal mucosa should be examined for Koplik's spots. The development of acute bronchitis in a person with mitral stenosis may be confused with the onset of left ventricular failure, while in a patient with chronic renal disease it is sometimes difficult in the early stages to distinguish bronchitis on clinical grounds from the development of early uraemia. However, the rapid response to antibiotics will often clarify the situation. But failure of resolution of the acute bronchitis with adequate antibiotics might indicate infection with a drug-resistant organism, of which an uncomplicated virus infection is one example, while early tuberculosis is another. In children asthma may present as recurrent attacks of prolonged coughing and wheezing, but a history of infantile eczema, of hay-fever, or the presence of prurigo will help to establish the diagnosis. On the other hand, many children in whom no allergic tendencies are found are prone to recurrent attacks of bronchitis with coughing and wheezing. These are the children with recurrent respiratory catarrh, and in them the attacks often start at about 3 to 5 years of age, and begin to get less severe and frequent by about 9 to 10, and usually clear up completely by puberty. In persistently catarrhal and chesty children and adolescents the presence of permanent bronchopulmonary changes due to mucoviscidosis must be excluded.

Investigation

Few laboratory investigations are likely to be of value in the acute stage, but observations of the pulse and respiration rates,

and of the sputum volume and appearance, are most important guides to recovery. Bacteriological examination of the sputum is often of little help, although the presence of one of the potential pathogens, as enumerated by May,⁶ will indicate the onset of secondary bacterial infection. If such a bacterial pathogen is discovered its drug sensitivity reactions should be determined, but in the mild or moderate case this information may not be available until the attack is nearly over. However, since it is rarely possible to tell at the onset of the infection whether its course is likely to be mild or severe, it is important that these bacteriological studies should be carried out when the patient is first seen, and ideally again 24 to 48 hours later, when the sputum has become overtly purulent due to established secondary infection. *H. pertussis* may be isolated on a cough-plate, while the use of a potassium permanganate and silver nitrate agar plate for a sweat test is a useful screening device to exclude mucoviscidosis. Blood counts, E.S.R. estimations, and ventilatory volume determinations provide little assistance in any but the severe near-pneumonic cases.

Complications

The commonest complication, particularly at the extremes of life, is the development of an aspirational or a specific bacterial pneumonia. It may vary from a relatively mild pneumonitis, discovered by routine radiography, to a fulminating and potentially fatal bronchopneumonia. The onset of the pneumonia is frequently heralded by pleurisy, which may also indicate the development of atelectasis involving more than a segment.

Acute bronchitis may not only arise as an exacerbation of an underlying bronchopulmonary disease, but can itself also exacerbate the symptoms and disability arising from stable chronic bronchial disease such as tuberculosis, bronchiectasis, and asthma. In such cases the x-ray may often show the development of "flares" around some chronic parenchymal lesions, and these in most cases are due to non-specific localized areas of pneumonic consolidation.

Congestive cardiac failure may be precipitated in patients with chronic obstructive airways disease, extensive chronic pulmonary fibrotic lesions, and in patients with myocardial insufficiency or other pre-existing heart disease. Cardiac failure may occur in children from the severity and overwhelming nature of the infection.

Delayed resolution of an apparently simple attack of acute bronchitis may indicate the presence of a drug-resistant infection such as pulmonary tuberculosis or of bronchiectasis, one of the pulmonary mycoses, or the presence of some other underlying pulmonary pathology, of which bronchial carcinoma is the commonest in the middle-aged adult.

A less common occurrence is the persistence in some patients of dyspnoea after an acute influenzal bronchitis, with subsequent progressive airways obstruction and defective diffusion suggestive of the development of an interstitial pulmonary fibrosis.

Inadequate treatment and convalescence are probably the most important factors in the subsequent development of chronic bronchitis, particularly in those adolescents and young adults who are prone to recurrent attacks of acute bronchitis.

Management

Most patients, particularly children and the elderly, are best nursed in bed until the temperature has been normal for two or three days or until the sputum has become mucoid. The room should be well ventilated and the temperature maintained at about 65° F., while humidification with a steam kettle or one of the modern electrical humidifiers will often ease an unproductive cough and help to loosen any tenacious sputum. No special dietary measures are indicated, although the patient

should be encouraged to drink freely of bland warm drinks. A smoky atmosphere from whatever cause should be avoided.

The mild uncomplicated case of acute bronchitis does not require antibiotic therapy, and will recover without it. Field *et al.*⁷ have shown that in epidemic bronchiolitis of children presumed due to respiratory syncytial virus penicillin is no better than a placebo in influencing recovery, or as a prophylactic against bacterial infection. In the moderate case it is usually justifiable to await developments before starting chemotherapy. But in the severe case, in children and the elderly, in the complicated or recurrent case, and in the acute bronchitis superimposed on a background of chronic cardiac or pulmonary disease, antibiotics should be started at once. The choice will, of course, be determined by the bacteriological investigation of the sputum, but until this is available it usually lies between one of the penicillins, streptomycin, chloramphenicol, tetracycline, or erythromycin. It is unwise to use streptomycin without clear bacteriological indication or until tuberculosis has been excluded. The risks of marrow depression following repeated courses of chloramphenicol are well known, while some organisms seem to develop resistance to erythromycin somewhat more quickly than to other antibiotics. The choice, therefore, in practice often lies between penicillin—either by injection of 1 mega unit daily or by mouth as the phenoxymethyl salt or as the derivative ampicillin—or of tetracycline, which probably has a somewhat broader spectrum of activity. Both ampicillin and tetracycline should be used in doses of 500 mg. t.d.s. for five or six days, followed by a maintenance dose of 500 mg. b.d. for a further seven to ten days, depending on the situation and response to treatment.

Should cardiac failure develop, specific measures including digitalis and diuretics will be required. Oxygen can be administered for cyanosis, preferably through a venturi mask, although particular care will be required if chronic airways obstruction with ventilatory impairment is present. Physiotherapy has little place in the management of the early stages, but when sputum is being produced postural drainage and breathing exercises have much to offer in maintaining a clear airway. Unfortunately this is most indicated in the old and the young, and in those patients with associated cardio-pulmonary diseases, who are just those patients least able to carry out these procedures most effectively.

In some patients the sputum is thick and tenacious, and in these cases adequate humidification is essential. Sometimes it is worth trying a mucolytic agent such as aleveaire, tryptar, or streptokinase by inhalation, but the results are sometimes disappointing. Dangerous obstruction of the air passages by thick retained secretions in children, or producing cardio-respiratory failure in chronic bronchitics, may require bronchial toilet and lavage or even tracheostomy and frequent bronchial aspiration.

Symptomatic Therapy

For an unproductive cough it is traditional to use codeine linctus *B.P.C.* 4 ml. as a cough suppressant; alternatively central suppressants such as pholcodine linctus *B.P.C.*, piperidine, or bisethane disulphonate can also be used. In the elderly chlorodyne (chloroform and tinct. morphine), 0.3–0.6 ml., is a useful and comforting sedative. When the cough becomes productive the sedative linctus should be withdrawn, and if any cough stimulant is required the most useful is probably the simple mist. sod. chlor. co. *B.N.F.*, $\frac{1}{2}$ fl. oz. (14 ml.) in hot water, to be sipped first thing in the morning and as required. It is doubtful in fact if many of the cough medicines have much value, and it is best, therefore, only to use the cheapest of them for a special situation and specific period.

Wheezing varying from a mild bronchospasm to severe asthma is a common accompaniment of bronchitis, for which

one or more of the many available bronchodilators are often prescribed. Ephedrine is useful in children, but of less value in adults, where isoprenaline sulphate, 5–15 mg. sublingually, or orciprenaline (Alupent), 20 mg. t.d.s., are more useful. Aminophylline can be given by mouth, as a suppository, or by infusion, but a watch should be kept for its side-effects on the bladder in the elderly. Moderate to severe asthma will often respond in the allergic individual to adrenaline 1:1,000 given subcutaneously by slow injection at minute intervals. In the more resistant cases hydrocortisone, 100 mg. by infusion for 40–60 minutes, may be required. The pressurized hand nebulizer probably has little part to play in the management of the acute case, although it may be of some value in the routine management of the asthmatic. Antihistamines are sometimes of value, particularly in children, and possibly in some allergic individuals. If sleeplessness is a problem promethazine, 10–25 mg. at night, is useful, while chlorphenitamine maleate (Piriton), 4–8 mg., has no such hypnotic side-effects. Should insomnia be a serious complaint sodium amytal, 0.2–0.4 g., soluble barbitone, 0.3–0.6 g., or chloral hydrate, 1.0–2.0 g., are useful. Opiates should be avoided. In children Dover's powder, 120–180 mg., is often sufficient, and an effective alternative to phenobarbitone, 30–60 mg., or chloral, 0.3–0.6 g. There is no reason why alcohol should be forbidden if the patient has been accustomed to it, and this is probably preferable as a hypnotic to one of the barbiturates.

Should cyanosis develop, indicative of ventilatory failure, oxygen is best given combined with an analeptic agent such as ethamivan (12 mg./kg./hr.) or amiphenazole (3 mg./kg./hr.) which can be given by slow intravenous infusion for 4–6 hours if necessary.

Convalescence and Prophylaxis

Convalescence from respiratory virus infections of the acute bronchitis types can frequently be surprisingly protracted, and the decision between allowing the patient back to work too soon or keeping him idling about at home too long is sometimes difficult. No specific management of the convalescent period is required, apart from ensuring steady resolution of the inflammatory process, without recurrence of secondary bacterial infection, which has a tendency to reappear if the antibiotics are withdrawn too quickly. If there is evidence of chronic pulmonary disease such as bronchiectasis or chronic bronchitis it is wise to continue the antibiotics as a prophylactic throughout the winter months. In these patients, and in those liable to recurrent winter bronchitis, vaccination in the autumn with one of the virus vaccines might be a wise precaution, while pre-seasonal hyposensitization in the allergic asthmatic is also worth trying.

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