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CHRONIC BRONCHITIS AND EMPHYSEMA

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Chronic bronchitis has been defined by Scadding as 'the condition of those patients suffering from chronic recurrent cough and expectoration, and usually effort dyspnoea, in whom these symptoms are not caused by disease of the lungs, by localized disease of the bronchi, trachea or upper respiratory tract, or by primary cardiovascular disease'. In Great Britain and other countries where climate and industrial air pollution together produce the ideal conditions for its development, chronic bronchitis and the later development of emphysema cause a heavy mortality and morbidity. In South Africa, fortunately, the disease is much less common, but still occurs often enough to make it an important illness to guard against by prophylactic measures, and to recognize and treat during the early stages of its development.

CLINICAL PICTURE

Most typically the condition begins with recurrent attacks of winter bronchitis, each attack being more persistent and more productive of cough and expectoration than the preceeding bout. These attacks may start in early childhood and may then clear up for some years, or persist and progress year by year. The onset may, however, occur at almost any age; but there is always a heavier incidence in men than in women.

After a varying number of winters tightness and wheezing in the chest become noticeable, at first only during the bronchial attack, but later between attacks also. As the condition progresses this becomes a major feature of the disease, causing dyspnoea on effort and even at rest, sometimes with obvious attacks of 'bronchial asthma', while at the same time the cough tends to persist through the whole year with acute exacerbations during the time of the previous 'winter bronchitis' and also during any respiratory infection. Between these acute attacks the expectoration may be minimal and mainly mucoid, but with the superimposed infections the sputum increases in amount and becomes frankly mucopurulent.

Gradually the picture of emphysema develops, with the deep barrel-shaped chest, the hyperresonant note on percussion (obliterating the normal cardiac and liver dullness), noisy, wheezing and prolonged expiration associated with rhonchi over the lungs, and marked dyspnoea. In the preterminal stage polycythaemia and 'central' cyanosis develop and, almost inevitably, failure of the right heart follows, with increased jugular venous pressure, hepatomegaly and oedema of the feet. This is one type of heart disease to which the term 'cor pulmonale' is correctly applied, but in the past it has often been recorded on the death certificate as 'myocarditis', 'myocardial degeneration', etc. These cases show 'central' cyanosis, i.e., cyanosis of the tongue and mucous membranes in addition to blueness of the extremities, as opposed to the usual peripheral cyanosis of most cases of heart failure. The term 'black cardiac' has been coined for the most extreme cases of this type. Finally, the onset of drowsiness and confusion indicates that, unless treatment is effective, the end is near.

AETIOLOGY

(a) *Climate and Air Pollution*

As is to be expected, bronchitis occurs most frequently and severely where the winter is damp and cold, and in South Africa it is probably most seen at the Cape, although even here the winters cannot be judged severe as compared with countries such as England and Northern Europe. It must also be accepted that not only climate is important but also the heavy pollution of the atmosphere with industrial effluents and smoke and, where climatic and industrial conditions are combined, chronic bronchitis and emphysema have their highest incidence. During every fog, or more especially 'smog', there is always a sharp rise in hospital admissions for chronic bronchitis and respiratory illnesses, and also in the mortality from bronchitis and emphysema. Fortunately, heavy atmospheric pollution is also not common in South Africa, so that the combined effect of climate and industrial factors is seldom seen here. In highly industrialized countries such as England this association is common and both the incidence and the mortality of bronchitis are much higher in London and Manchester and in the industrial areas generally than in the countryside.

(*b*) *Smoking*

Various surveys of the effect of smoking have indicated that it bears a definite aetiological relationship to this disease. Even in the United Kingdom, where climate and air pollution are the major factors in the production of the illness, Doll and Bradford Hill found that in male doctors over the age of 35 heavy smoking caused a 6-fold increase in the mortality from bronchitis. The incidence of chronic bronchitis closely parallels that of carcinoma of the bronchus, which must be accepted as being associated with heavy cigarette smoking. Finally, duodenal ulceration, another disease of smokers, has a significant association with chronic bronchitis. That smoking produces bronchitis is not surprising; it is indeed so widely recognized by the laity, who have coined the term 'smoker's cough', that it is accepted as normal. One may presume that in the Union, where consumption of cigarettes per unit of population is exception-

ally heavy, and where the climatic and air-pollution factors in the production of chronic bronchitis are minimal, the aetiological significance of smoking is greatly increased. It is indeed unusual in this country to find chronic bronchitis and emphysema in non-asthmatic patients who are also non-smokers. It is surprising how little attention is paid to this factor both by patients and their medical advisers.

(c) *Constitution*

Constitutional factors undoubtedly are of very great importance in chronic bronchitis. These patients usually have a long history of cough, even dating back to childhood, or recurrent respiratory infections, nasal catarrh and repeated bouts of bronchitis can be traced, this tendency to 'weak chests' often being a familial one. Asthma and other respiratory allergies undoubtedly do tend to predispose to chronic bronchitis at a later period of life.

(d) *Infection*

The role of infection is also of considerable importance. These patients seem to develop respiratory infections very readily, and these infections are more persistent and severe than in other subjects. Exacerbations of established chronic bronchitis occur when respiratory infections are rife, but no particular organism can be incriminated, a variety of organisms being obtained from the sputum during exacerbations. Naturally enough, antibiotics have been employed for years in treating these acute infections, but more recently their use in prophylaxis has been under trial, with promising results.

PATHOLOGY

The nature of the essential lesions in chronic bronchitis is not clearly understood. Various authors have suggested that a primary disturbance of the mucus-secreting cells of the respiratory tract is the basic factor, the resulting swelling of the mucous membrane causing obstruction to the airway, especially in expiration. This expiratory obstruction, possibly in conjunction with a constitutional weakness of the alveolar walls of the lungs, causes breakdown of the alveolar partitions, which is the basic feature of emphysema. As a direct result, the pulmonary capillary bed is diminished and increased work is demanded from the right heart in the circulation of the blood through the lungs. The healthy myocardium of the right ventricle usually has no difficulty in maintaining a moderate increase in the pulmonary arterial pressure, and it is commonly only with the aging of the myocardium (around the age of 50-55) that embarrassment of the right ventricle becomes apparent. By this time alveolar destruction has reached an advanced state; at the same time ventilation of these damaged alveolar spaces is restricted by bronchial and bronchiolar obstruction, and impaired oxygenation of the arterial blood, with central cyanosis, results. Parallel with the reduction in oxygen tension in the alveoli, CO_2 retention occurs, so that the CO_2 tension in the arterial blood rises as the oxygen tension falls.

It must be emphasized that the cause of the arterial oxygen desaturation and the CO_2 retention is the impaired ventilation of the alveolar spaces, and that shunting of blood from the pulmonary artery to pulmonary vein by anastomoses is of little importance. Nor is a diffusion defect across the alveolar-capillary barrier demonstrable. With the deficient oxygenation of the blood, reflex pulmonary arteriolar contraction occurs, with a further rise in the pulmonary arterial pressure, further aggravated in many cases by secondary polycythaemia and increased viscosity of the blood, so that the stage is now set for failure of the overburdened right heart. Any respiratory infection, by causing further limitation of alveolar ventilation, may acutely precipitate the condition of central cyanosis, CO_2 retention and right heart failure, which, under favourable circumstances, may be as rapidly reversible if infection and spasm can be overcome. The chronic terminal condition, however, is more resistant to treatment, and chronic CO_2 retention ultimately causes the drowsiness, confusion and coma which, with failure of the right heart, is the main cause of death in these cases.

TREATMENT

Prophylaxis

(*a*) In patients who are subject to recurrent respiratory infections and winter bronchitis, the ultimate development of chronic bronchitis and emphysema is always to be feared, so that measures to prevent infection and irritation of the respiratory passages should be instituted at an early stage before irreversible damage has been done. As far as infection is concerned, avoidance of crowded, stuffy places, particularly during the season of respiratory infections, should be advised, but it usually is almost impossible to enforce this in patients who have to travel in buses to work, or who work in close proximity to others who may carry infections. Vaccines have been recommended and may be worth a trial, although convincing evidence of effectiveness is still lacking. Long-term prophylactic treatment with antibiotics, such as the tetracyclines in doses of 0·25 or 0·5 g. per day, has shown considerable promise. In carefully controlled experiments convincing evidence of a reduction in the frequency of attacks of bronchitis, and even an improvement in the condition of the lungs, have been obtained. Unfortunately, expense alone is likely to make this unacceptable to the majority of patients, and occasional toxic reactions also are a hazard. It seems likely that early treatment of acute bronchitis is a more acceptable method of prevention of further lung damage.

(*b*) Avoidance of bronchial irritation by smoke, dust or fumes is an obvious step, but may be difficult to implement in certain trades; consideration may then have to be given to the advisability of a change of work or residence, when this is possible. The obvious importance of a complete ban on smoking is unfortunately usually either overlooked or insufficiently stressed by the medical attendant, and nearly always meets with considerable resistance on the part of the patient. Frequently patients who have smoked heavily for years are ultimately forced to give up smoking when bronchitis with emphysema has already reached a crippling stage, but unfortunately it is then too late to expect more than slight relief of symptoms. It must be emphasized again that, especially in this country, smoking is one of the major causes of chronic bronchitis and emphysema.

(*c*) In the various geographical areas of South Africa there is a wide range of climatic conditions, so that sufferers,

or potential sufferers, from chronic bronchitis and emphysema can practically always find some part of the country where the climate factor can be eliminated; the cold and wet winters of the Western Province, particularly the Cape Peninsula, is obviously unsuited to these patients, and the climate of the Karroo or high veld, or possibly the Natal Coast, is much to be preferred. It is often difficult to persuade patients that such a major upheaval in their lives is justified or necessary, but where the condition is progressing year by year, there can be no doubt that such a change should be seriously considered.

Acute Bronchitis

Treatment of the acute attack of bronchitis follows the ordinary accepted measures, namely, rest in bed in a warm, well-ventilated room, antispasmodics (such as ephedrine, aminophylline or a combination of the two, and a sedative); and expectorant cough mixtures and inhalations. Expectorants have a time-honoured role but are of doubtful value, and inhalations of menthol, etc., also are mainly psychological in value; but a linctus containing codeine, heroin, opium, etc., is often necessary to ensure a reasonable night's rest from the distressing cough. Antibiotics are usually of the greatest value, especially when the sputum is mucopurulent in character, and preference should be given to the broad-spectrum antibiotics of the tetracycline group, or to novobiocin or chloramphenicol. Adequate dosage is essential during the severe phase of the cough, and at least 500 mg. 6-hourly should be given until a good response is obtained, when the dosage may be reduced to half this amount. Penicillin, or penicillin combined with streptomycin, is not usually as effective as the broad-spectrum antibiotics and, as the sufferer from chronic bronchitis is often also an allergic subject, the danger of penicillin reactions is very real. Steroids, such as cortisone, hydrocortisone, prednisone, etc., play little part in the treatment of bronchitis in the ordinary case, but where there is an allergic background they may be of definite value in speeding recovery. If a steroid is going to give relief, improvement is noticeable within 24-48 hours of its exhibition, and if this is not evident after 48 hours its use should be discontinued. Prednisone, 10-15 mg. 6-hourly, is a favoured preparation and, if effective, it can be maintained in slowly reducing doses for 10-14 days as necessary. Patients should be made to understand that any respiratory infection, even a minor cold, should be regarded as a major illness and should justify a period of treatment in bed, while the full routine outlined above should be instituted if any evidence of bronchitis develops.

Chronic Bronchitis

The more prolonged the chronic bronchitis, and the more severe the emphysematous changes in the lungs, the more severe the effects of each attack of acute bronchitis. Patients with advanced disease are usually willing to cooperate to the fullest extent in their treatment (except for the question of giving up smoking) but it is impossible to reverse the structural damage to the lungs, so that the results of treatment cannot be really satisfactory; obviously it is of great importance to insist on adequate prophylactic measures in younger patients before the full-blown picture of chronic bronchitis and emphysema develops.

Cor Pulmonale

In patients who are already cyanosed, an acute bronchial attack is a major illness which may terminate fatally, and treatment is a matter of urgent importance. Cardiac failure may develop, producing a rise in jugular venous pressure, hepatomegaly, and oedema of the legs. Increased cyanosis, so that the tongue becomes a dark blue colour, is then to be expected, while consciousness becomes dulled and the patient drowsy and stuporose. The development of coma is usually a pre-terminal event. Treatment here is the same as for any acute attack of bronchitis, and a response to antibiotics offers the best hope of recovery. In addition, mersalyl, 2 c.c. every 2nd or 3rd day, may be of great value, or chlorothiazide, 2-4 tablets (1 to 2 g.) daily, may be substituted. Diamox may be especially useful, because it has some effect in lowering the CO_2 tension of the blood in producing its diuretic effect. It certainly is worth a trial in severely ill patients. Full digitalization is recommended, but may have disappointingly little effect on the degree of heart failure. The administration of oxygen is obviously suggested by the cyanosis, but it should be used with caution because it may cause deepening of the stupor and even coma and death. In these patients the prolonged and excessive degree of CO_2 retention leads to a blunting of its normal stimulating effect on the respiratory centre, and lowered oxygen tension of the arterial blood is the main respiratory stimulus; if this is removed by free administration of oxygen (as in an oxygen tent), the respiratory efforts are diminished and the patient rapidly becomes more comatosed owing to a rise in the arterial CO_2 tension although, paradoxically his colour improves and cyanosis may be absent. Oxygen therefore should be given only by nasal catheter, preferably intermittently, and at the first sign of increased unconsciousness it should be stopped until consciousness is again restored. Where facilities are available for assisted respiration, oxygen may naturally be given freely with much benefit to the patient. Ideally, in the comatosed case a tracheotomy should be performed, and IPPR (intermittent positive pressure respiration) can then be applied with considerable benefit, and at the same time secretions can be aspirated from the trachea and bronchi. Aminophylline, 0·25 or 0·5 g., should be given by intravenous injection, and 'solucortef' should be given at the same time (100 mg. intravenously). It has recently been suggested that large doses of amiphenazole, e.g. 150 mg. intravenously per hour, may be useful because of a specific stimulating effect on the brain and respiratory centre, and in promoting cough and expectoration. It is clear, however, that the prognosis is poor, and that treatment should be undertaken only in a hospital where full facilities are available. Even then, in spite of the best possible treatment, failure to respond is common enough, while improvement can of necessity only be temporary.