UNANSWERED QUESTIONS ABOUT CHRONIC BRONCHITIS

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I am afraid that, after the warm colours and reassuring certainty of the film we have just looked at, what I shall have to say will inevitably seem somewhat dull and grey. Perhaps I can comfort myself with the thought that I am only supposed to tell you what are the questions; I am not called upon to answer them.

First, a few words about the definition of 'chronic bronchitis'; we have had a good deal of discussion about this, as you will all be aware, and most people now agree that the only possible way of defining chronic bronchitis is in clinical descriptive terms. Chronic bronchitis may be defined as a disease characterized by persistent or recurrent increase in bronchial secretions sufficient to cause expectoration. From the general group of all patients, we are selecting for special study those who present these features. We make a similar selection in relation to other diseases, but the criteria for selection may be of different sorts. Sometimes we will define diseases in terms of what we believe to be the cause, picking out those patients whose condition, we believe, is due to a specified cause; this constitutes an aetiological basis for definition. And sometimes we will find it convenient to define diseases in terms of morbid anatomy.

In the case of bronchitis (to come back to our subject of today), we must start with a clinical-descriptive definition. This definition is qualitative. Before we can use such a definition in a scientific investigation we must insert quantitative terms into it. We must recognize that these quantitative terms are arbitrary. We will choose them, not because they are correct or incorrect, but because they are convenient for our purpose. And so, for epidemiological purposes, we have various definitions of bronchitis which insert quantities into the simple qualitative definition—required durations of the cough and sputum each year, the number of febrile episodes in the past three years, and so on. It is inevitable that these quantitative factors should be arbitrary, because if we consider the course

of this disease, we find that it starts insidiously with almost imperceptible symptoms. A smoker says, " I have a smoker's cough ". This apparently trivial symptom gradually becomes more severe, and it is clearly quite arbitrary at what stage we will say, " This is chronic bronchitis ".

It is the early stage that we are concerned with in this symposium. At this stage the only evident abnormality is excessive production of bronchial mucus. The patient complains of chronic or recurrent cough and expectoration: if this is confirmed by inspection of a sputum specimen, and localized bronchopulmonary disease which might cause it is excluded, then the diagnosis of chronic bronchitis is made. Thus early diagnosis is within the power of all of us mainly by listening to what the patient says. Functional defects, demonstrable by instrumental means are not an early feature, and thus early diagnosis does not require the use of tests of lung function.

In the earliest stage, the sputum is generally mucoid. We then go on to the phase when there are recurrent episodes which we believe to be due to bacterial infection, when the sputum becomes mucopurulent. Then at some stage in the course of this disease there are symptoms which can be referred to obstruction to the flow of air in the lungs. It is at this stage that we get help from instrumental aids, in detecting and quantitating the airways obstruction. Various simple devices are available by which some figures relevant to the resistance to air flow in the bronchi can be obtained.

Let us turn to the aetiology. There is some agreement here that air pollution, public and private, are the chief factors. Two aspects of the effects of air pollution are sometimes confused. One is its role in determining the inception of chronic bronchitis; and the other its role in determining progression from the simple variety to the more disabling obstructive varieties; and of course it is by no means certain that the factors concerned in these two aspects are the same. They may be, but I think that this is a question which requires examination. As to what I call public and private air pollution, there are still unanswered questions concerning the relative importance of these two factors; and this I hope will be dealt with later. That they are both important no one doubts. There is, of course, an important difference between these two which we can well emphasize. That form of pollution which arises from smoking is easily avoided by the personal action of the individual, and its prevention is therefore susceptible to a different approach from general air pollution which is a community responsibility.

Another point about these two factors is how far they may be synergistic. In other words, is the effect of these two factors simply additive or do they multiply each other? We may today

hear something of the evidence on this point.

There are further factors in the aetiology which require attention. First, there is the question of possible genetic factors. Are there certain people who are genetically predisposed to chronic bronchitis, possibly by being especially susceptible to the external factors? This is a question that is not yet answered, although there is some suggestive evidence that it might be so. Secondly, the role of recurrent infections, both bacterial and viral, in determining the inception of chronic bronchitis is not precisely determined.

When we come to the factors determining progression from simple chronic bronchitis to advanced, obstructive bronchitis, there is some evidence of the role of continued cigarette smoking in causing this. Much of this evidence arises from unorganized clinical experience. But most of us have had cases in our experience where certainly the earlier stages of chronic bronchitis appear to have been arrested by discontinuing cigarette smoking. On the statistical scale it is difficult to study this question. In the study of mortality in our own profession by Doll and Hill, the data on mortality from chronic bronchitis in relation to discontinuance of cigarette smoking were somewhat equivocal. But there is a good reason for this. It is highly probable that doctors who were finding themselves becoming bronchitic would give up smoking; so that among those who have given up smoking there would be a number who were already bronchitics when they did so. We know very little about the long-term results of removal from exposure to air pollution of those people who are already established bronchitics. It is logical to suppose that they would be benefited, but I do not know of any figures to confirm this belief.

To turn to the role of recurrent infections, about which we heard a good deal in the film, it is the general experience that established chronic bronchitis is commonly associated with recurrent or persistent bacterial infection. The factors that determine recurrent activity of bacterial infection are generally agreed to be the common respiratory viral infections, and possibly also episodes of unusually heavy air pollution. It seems to me that there are two important unanswered questions here. One is—and you will think that this sounds like heresy: Does recurrent bacterial infection really cause deterioration? We should like to think so, because there is a factor that we can deal with. There is a study under way, and nearly completed, which was designed to find out whether the methods we have for the control of secondary bacterial infection, applied to a group of patients with early chronic bronchitis did in fact diminish the rate of physiological deterioration. I think that when the answer to this does come some of us may be surprised.

The second unanswered question here is a very important one: that is, the role of viruses both in determining inception and in predisposing

to bacterial superinfections in chronic bronchitis. This is a difficult problem, of course, because of the inherent difficulty of virus studies. With the practical methods currently available, we can usually make a diagnosis of respiratory viral infection only by serological study, to which we get the answer several weeks later after the patient has recovered from the illness. From the point of view of practical management, this really does not present any great advantage. But from a scientific point of view the careful study of the role of viruses in respiratory infections and in acute exacerbations in a group of chronic bronchitics by all available methods, with the objective of determining exactly the role that recurrent viral infections are playing in chronic bronchitis, is I think, of great importance. Such a study is certain to be difficult and expensive.

Now let us turn to a very different subject: that is the inter-relations between bronchitis and emphysema. Although chronic bronchitis can be defined only in clinical descriptive terms, emphysema is clearly definable in morbid anatomical terms. We saw in the film how the pathologists detect it. When we make a diagnosis of emphysema we mean, or ought to be meaning, that we expect that if the pathologists ever have an opportunity of looking at the lungs they will find specific anatomical changes that they will call emphysema.

There has been great hesitation on the part of some physicians—the more experienced the physician the more hesitant he may be—in making a diagnosis of emphysema, largely because in the past we have often been shown to be wrong in this diagnosis. But one should not be put off by fear of being wrong; rather should we try to improve our diagnostic criteria and methods. There is in my mind no question that there are cases in which you can make a reasonably accurate clinical prediction that the pathologist will find emphysema. I would suggest to you that you can distinguish a group of patients who have emphysema, without important bronchitis. These are people who, usually in middle age or later, first become increasingly breathless. They have over-distended chests, not with a barrel shape so much as long, deep and of circular cross-section. Radiologically, they have a long thorax with a vertical heart and a diaphragm low in position, even down to the twelfth rib. When you listen to the chest you may hear nothing; the breath sounds are extraordinarily weak. They may become bronchitic secondarily. They have an extraordinary ability to maintain their alveolar ventilation and keep their arterial Pco_2 down in spite of severe fixed airways obstruction, until quite late in the course of their illness.

At the other end of the scale there is the chronic bronchitic who starts off with a long story of cough and expectoration, often

beginning with a so-called smoker's cough. Over the years, he slowly deteriorates with gradually increasing dyspnoea. On examination, he has a chest of normal configuration. Radiologically, there is usually little abnormality. Sooner or later, he gives up the struggle to maintain normal alveolar ventilation in face of increasing airways obstruction, letting his arterial Pco_2 rise and Po_2 fall, and eventually he tends to develop recurrent episodes of oedema associated with exacerbations of his bronchitis and increased CO_2 retention. When he dies, the pathologist may find no emphysema, or scarcely any.

What is the relation between these two groups of patients? As I am supposed to be talking of unanswered questions, I may perhaps be permitted a little speculation even in these august surroundings. Is it not possible that we may be dealing with two separate processes? One of these, possibly genetically determined, might lead to 'primary' emphysema. The other, mainly environmentally determined, would be manifested by chronic bronchitis. Since chronic bronchitis is so widely prevalent, it would be expected that a considerable proportion of those with a predisposition to emphysema would suffer from this disease; moreover, it would not be unexpected that a concurrent chronic bronchitis should aggravate the symptoms and possibly accelerate the course of such patients, so that the combination would more frequently come to clinical notice than uncomplicated 'pure' emphysema. Environmentally determined chronic bronchitis may lead to the development of various structural changes in the lung, including emphysema. Thus, cases which originated as either chronic bronchitis or as emphysema would be expected to present varying combinations of features late in their course, which is exactly what in fact is observed.

Finally, we must all be very interested in prevention. This is one of the purposes of this symposium. It seems to me that here the chief problems are operational ones. We know that general air pollution and cigarette smoke are important causative factors. With regard to cigarette smoking, the chief question is how we are to bring home to the public the knowledge that smoking is dangerous in this connection and how we are going to make smoking seem in the eyes of the public the filthy habit which it really is, so that it no longer remains acceptable socially. Of course, any attempt to do this is most unpopular with vested interests of all sorts, but I am sure that it is the most important practical step we can take to prevent chronic bronchitis.

To turn now to general air pollution, we are here in a very technical field, and I would remind you that the problem is not a simple one. Many of the processes which give rise to air pollution are essential ones—not like smoking, which we should all be a good deal healthier

for avoiding. Moreover, it is impracticable to eliminate all air pollution in the sense of all foreign matter released into the air from industrial processes. Therefore the problems are those of defining the actual pollutants that are harmful in specific ways and of devising economic ways to eliminate those pollutants; or, if they cannot be eliminated, to dilute them or keep them away from centres of population.

I think those are the main questions I would put before you. You see I have done what I was asked: I have asked some questions but have answered none of them, although I have put before you an unsubstantiated speculation for which, I suppose, scientifically, I ought to apologize.

Chairman: Looking down some of the consultation registers of the 17th century I saw mentioned the scab, the itch, the scurf, the plague, fever, the gout, the pox, the flux, and measles, but nowhere did I see bronchitis. In this century, the College of General Practitioners and others have shown in surveys that 26 consultations per 1,000 are for bronchitis, and that eight out of every 100 beds in general hospitals are occupied by bronchitic patients. There is a widespread failure, we know, to take seriously the report on the subject of the Royal College of Physicians, based, as Bertrand Russell said, on the conjugation of the verb: I think you are obstinate and that he is pig-headed! However, I will now ask Dr Holland to discuss the natural history of chronic bronchitis.

THE NATURAL HISTORY OF CHRONIC BRONCHITIS

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I am very grateful to you for inviting me to talk on this subject, particularly as on this occasion you have at least labelled my talk, " The Natural History of Chronic Bronchitis ". On the last occasion when I spoke on this subject I was introduced as someone who was going to talk about an ' English ' disease, and I was followed by a consultant venereologist. I am afraid that I disappointed my audience, who thought they would have a whole afternoon devoted to venereal disease. The description of chronic bronchitis as ' an