Chairman: It would be practical, but I think it is a little outside the writ of this meeting.

Dr Gomez: Muco-polysacarides can be measured in the early bronchitic as large goblet cells just beginning, and that will be a practical way of detecting it.

Chairman: I will now call upon Dr Fletcher to sum up.

CLOSING REMARKS

Dr C. M. Fletcher: It is difficult to sum up all that we have heard today. Perhaps I can best try to summarize what we do know and what we do not know about bronchitis and then consider what we ought to do about it.

I think we definitely know that cigarettes are a cause of bronchitis. We are pretty clear about air pollution, but we do not know the relative roles of the acute episodes and the intervening lower levels. I think we are confident about the role of infection in aetiology, particularly since hearing the very interesting recent evidence which Dr Holland has presented to us about school children. We have also acquired some evidence recently that among non-smokers with a productive cough there is a much higher incidence of childhood illnesses described as bronchitis than among a comparable group of smokers with bronchitis (Fletcher 1965). It looks as if childhood infection may produce bronchitis in non-smokers who have no other cause.

There is no doubt there are susceptible and less susceptible individuals. Not all smokers get a cough and those who do must have a greater liability to get bronchitis. Is this congenitally determined? Again, we do not know. The best evidence that it may be is provided by Stuart-Harris's (1965) study in which he showed a higher incidence of bronchitis in the sisters than in the wives of patients with bronchitis. How important it is that we should discover more about the susceptibles because it is on these we need to be working.

Factors about whose effects we are in doubt are temperature, climate and sex. There is a strong clinical impression that cold and damp may encourage and aggravate bronchitis but there is no clear evidence about this. There is some evidence that women do get the

same sort of bronchitis as men if they smoke as much but my impression is that the female bronchitic is a little different from the male. I cannot put a quantitative figure on that, but it is an interesting point that requires study. It is difficult to demonstrate any effect of occupation on bronchitis. Mortality statistics show that men in certain occupations have a higher bronchitis death rate, but in these occupations the mortality of the wives is increased proportionately, so it looks as if this is a social and not an occupational effect.

That brings me on to our fantastic ignorance about the social factor. There is this 5 : 1 ratio of mortality between Social Class V and Class I. Dr Holland showed figures for 1931. Those for 1951 are just the same despite tremendous improvements in social environment. We do not know what this social gradient is due to, and I have a feeling that the general practitioner should be able to tell us what we ought to be looking at. It is a great challenge to the epidemiologist. This five-fold difference must indicate preventable factors for it must be due to environment.

Then there is the British predominance which again is a great mystery. Air pollution is the factor always trotted out, but the death rate in our purely rural areas is about 20 times that of the United States and Denmark although there is not all that much more pollution in our rural areas than in America. There is something very odd about the English susceptibility which we do not understand. A little of it is due to the fact that we diagnose the disease readily and other countries do not. But someone must come up at some time with a bright idea of what it is that our bronchitics are doing and particularly what those in Social Class V are doing, to increase the incidence of this disease.

We know too little about the natural history of bronchitis. It is widely believed that the bronchitic first starts to cough and that hypersecretion of mucus makes him liable to infection and this does the damage that leads to airways obstruction. We have no sound advice to justify this common assumption. It may be that there is airways obstruction at the very beginning and that this interferes with bronchial drainage and produces hypersecretion and infection. What we need is detailed study of the natural history of the disease. This, of course, is something which the general practitioner is in a better position to do than anybody else, and I should like to urge that it could be a concern of an organized group of general practitioners to make the necessary observations.

What sort of observations? Well, first, early and accurate diagnosis. Dr Kay talked about the little statistical squares and of how remote statistics are from general practice; but in fact we all work with

statistical concepts nowadays. We advise people not to smoke because their chances—statistically estimated—of getting ill are greater than non-smokers. Statistics must be the basis of our action.

Diagnosis depends on accurate observations. We have heard about a standardized questionnaire; but it is much better to look at the sputum. Some transparent container could be given to every patient to put the sputum into so that the doctor can look at it. A local practitioner recently brought a man for me to see as a private patient. The patient started to cough and I handed him a container and said " Spit into that ", and he half filled it with purulent sputum on one expectoration. The practitioner did not know that the patient had mucopurulent bronchitis.

Then there is measurement of ventilatory capacity for which the peak flow meter is the simplest procedure.

In early diagnosis there is one very important area of ignorance, again: Which are going to be the bad ones? Dr Gregg suggests that anybody with a peak flow below 450 may be on the way to the grave! Are they all? Fifty per cent of smokers, at least, have a productive cough, but only five per cent, perhaps even less, develop severe disabling bronchitis that kills them. Which are the ones that are going to get into trouble? For this too we need follow-up studies with accurate early diagnosis, to see which are the ones that go bad on us.

We have heard about how puny our efforts are in this major field of how to stop our patients smoking. It is a field almost entirely devoid of really good research. Of course, some research has been done. The social survey recently carried out a widespread study of the psychology of smoking. Some of the preliminary results are very interesting. It may be possible to distinguish different types of smokers: those who smoke for social reasons and those who smoke because of addiction. I am convinced that some smokers are pharmacologically addicted. In helping people to stop smoking we need somehow to improve the level of emotional conviction. In looking round here today at the cigarette free atmosphere one can see that doctors have done a remarkable job themselves and we are not all that different psychologically from our patients. It is, I think, because we have been emotionally convinced.

I was most impressed in this business of addiction and withdrawal symptoms by a colleague of mine who stopped smoking for about the fifth or sixth time two years ago. I asked him about the side effects and he said " This time I had no deprivation symptoms at all. This time when I stopped I had made up my mind I was going to, but previously my deprivation symptoms were my way of persuading

myself that I could start again ". I believe that a man often thinks he should stop smoking but deep down he is not persuaded that he either will or can. We need to discover techniques by which we can get at that deeper level. Of course, it would be useful if we found a substitute or a safer kind of cigarette.

Then there is the question of chemotherapy. I don't think we should use sulphonamides. I am reminded of a broadcast for the B.B.C. that I was asked to do on bronchitis. They wanted a successfully treated patient and I took along a prized success. He gave an account of how he had received treatment and it had been wonderful. Only after the broadcast did I realize that he had received a placebo in a controlled trial. I congratulate the doctor who spoke on this, on his placebo effect. Four or five trials have been done on sulphonamides and they have all shown negative results.

Measurement is very important in assessing the effect of bronchodilators to see whether they are or are not helping our patients. They are so often keen to please us and will tell us that the pills we give them, which may be expensive, are helping them when in fact they are not. What use are they in early bronchitis? None, unless there is airways obstruction. Again, what is the long-term benefit of bronchodilators in relation to recurrent infection? It may be that if airways obstruction encourages infection adequate control of airways obstruction might reduce liability to infection.

At the end of a conference organized by the College of General Practitioners I would like to conclude by referring to one of the great things the College has done for general practice to bring in the fresh air and inspiration of making careful observations and doing research. Early bronchitis is one of the fields in which a really big contribution could be made by practitioners because of the urgent and desperate need we have to discover the natural history; this is where practitioners with long and intimate contact with patients, might be able to do a better job than any other group. I am attempting to do this by a large survey of 1,000 people, making six monthly observations on sputum volume and ventilatory capacity. If 1,000 practitioners could select a group of 50 subjects each we might get results from a population of 50,000 and then begin to discover what is the effect of all sorts of factors we are now guessing at. If we could do this and hold another conference in ten years' time we should find ourselves knowing answers to a great many of the questions which completely perplex us today.

I have enjoyed this conference very much. I have not summarized it, but have given you some of my views. I am delighted to see the great interest in the subject that the College has inspired in its

members; and, finally, I would thank you, Sir, for your excellent chairmanship of what, for me, has been a most enjoyable meeting.

REFERENCES

Stuart-Harris, C. H. (1965). In *Bronchitis II.* Royal Vangorcum, Assen, Netherlands.
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Chairman: Dr Fletcher has ended on an inspiring note: he has encouraged us and pinpointed some of the questions that need to be answered, and we look forward to acting on some of these suggestions.

I should like to thank the large number of men and women who have helped us in this meeting but whom we do not see—administrators and secretaries, people who do the spade work, and people from Beecham's who were loaned to the College for this purpose. We are extremely grateful to them. It has, of course, put a strain on headquarters staff. And, not least, I would thank the large number of doctors and general practitioners who have taken a day off to come here and listen and take part. We are most grateful for your assistance in making it such a successful meeting.

We thank all the speakers for their valuable contributions. The generosity of Beecham Research Laboratories has made the holding of this symposium possible.