Part One: Pneumoconiosis Research

MB Charles, following the Oxford period, the penicillin work, you went back to London for two or three years but then became deeply involved in the story of pneumoconiosis research. Can you tell me about that?

CF Well, it was a great surprise to me that, because I knew nothing about it and was told nothing about it practically as a medical student. Pneumoconiosis comes from the Latin word, the Greek words – *pneumo* and *konis* – lung and dust. And it means diseases caused by dust getting into the lung. And the common cause, type of it that was known in those days was called silicosis, which was produced in mines where silica rock was mined. And the dust produced a very severe reaction in the lung, and [was] complicated by tuberculosis. Now, it was in South Wales, in the coalmines there didn’t seem to be any such silicosis because there was no increase in tuberculous deaths, which was the way they assessed in those days. But it was known that the coalminers in South Wales had a great deal of what was called bronchitis. And then in about 1926, a man, a general practitioner in the west of the coalfield in South Wales bought an x-ray plant, and started taking x-ray pictures. And he found these miners had x-rays that looked very much like severe silicosis. It was recognised then, and compensation was given under the Workmen’s Compensation Act, and men who were compensated had to leave the mines. Well now, intensified methods of mining increased the amount of dust in the lungs. And by 1945 the compensation scheme had … produced about five thousand out of a thousand miners – that’s five per cent of the whole workforce – was certified as having this disease, as a result of x-rays in one year. Now the Medical Research Council had done a study of this condition just before the, the 1945, ’40-’45 war, and they’d found that there was a great deal of abnormality in the x-rays of these miners. And they’d suggested ways of reducing the dust so that the disease could be reduced. But there it was. And the Medical Research Council were now asked to set up a research unit to do further research into this. And I heard of this from my chief at the Postgraduate Medical School where I then was, John McMichael, who was the acting head of the department there. One time we were driving home together, and he said ‘The Medical…’ – you see, he’d had a letter from the Medical Research Council to try and suggest somebody who might go down to Wales and direct, to direct this unit. And I remember thinking to myself ‘My goodness. I hope he hasn’t suggested me!’ And so when, about a week later, I received a letter from Sir Edward Mellanby who was then secretary of the Medical Research Council, asked me to come and see him, I had a nasty feeling what it was going to be. And so it turned out. And he asked me if I would go down there and set up a unit and investigate this problem. Now, I was extremely unwilling to do that. My idea was that I was going to go on in clinical

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1 A. Harper.
2 Dr Fletcher must mean one hundred thousand here.
medicine, and hoped I might one day be a professor of medicine. And, at the same
time, I had diabetes and that had prevented me from doing war service. And I felt that
perhaps this was a sort of, an occasion when I ought to try and do something, like my
contemporaries were doing for their country, so to speak, sort of a sense of loyalty.
And I talked to my brother-in-law, Stefan Hopkinson(?), about this, and he said he
thought that probably I would be doing a good thing if I did it. But that, and I still felt
that I really wanted to do clinical work, and so I did turn it down. But then after this
talk with Stefan, I thought again, and then I wrote to Mellanby saying I was prepared
to do it after all.

MB Charles, can I ask two questions at this point, before we go to Wales?

CF Yes.

MB Because that’s an important part of the story. You mentioned in the early part,
the relationship, the measurement of distress, on the tuberculosis kind of scale. How
did tuberculosis relate with pneumoconiosis?

CF Well, people who got silicosis were much more liable to get tuberculosis.

MB Right.

CF And their deaths were certified as tuberculosis. And it was therefore taken
that wherever there was a big incidence of tuberculosis – for instance in the Cornish
tin mines, where they were mining in silica rock – that was an indication of silicosis.
But the coalminers’ disease was not so strongly associated, in fact very slightly
associated with tuberculosis, and so it was overlooked.

MB Right. Charles, pneumoconiosis/silicosis – do they have very similar
symptoms? Do they present the same distress to the miner?

CF The symptoms are, ultimately, extreme shortness of breath. In the early stages
they’re both asymptomatic. But most miners who mine in, in dusty conditions,
develop a cough. And the coalminers in particular had a very high incidence of
cough, and this was called bronchitis, and that’s why this disease was thought to be
bronchitis, in the miners of South Wales.

MB But in hard South Wales mining, I mean, a cough can’t have been too bad.
They must have become quite extreme to be put out of the pits, I mean...

CF Oh well, yes, you see, they eventually became very short of breath, and were
so disabled they couldn’t go on.

MB Right.

CF And when that had happened… And then, you see, when Harper started
taking the x-rays, then the Silicosis Board(?) was set up in South Wales. It was called
silicosis then … and to certify people as, as having the disease, and therefore
deserving compensation from the coalowners, because it was then all run by private
coalowners. And it was the, this compensation scheme that led to this tremendous loss of men in the mines.

MB Charles, you go to Wales, you’ve taken Mellanby’s offer, and you go to Cardiff…

CF Well, I went… Well, the first time I went down was just to have a look around. And I went there with Arthur Horner, who was then a president of the South Wales coalminers, and was a very well known Communist. And he was a, quite a figure. I got to know him quite well. And I met him, and he said, talking to me with the, with the other people from the Ministry of Fuel and Power as it then was, there, he said ‘You know, this thing is destroying the coal industry in South Wales. It’s up to you to save the coal industry in South Wales.’ And I remember thinking ‘My God! What have I done now?’ Because I didn’t like to think I was going to try and save the coal industry in South Wales. And eventually, I put off my departure to South Wales for six months. He allowed me six months in which to write an MD thesis, which I did at Cambridge – and this was on the gastroscope, quite a different subject. And I wasn’t, I didn’t know very much about chest diseases; I knew nothing about it when I went down, an entirely new… But anyway, Louisa – my wife – and I, we went down there in 1945, and they provided a house for us, which was in, a house in Cardiff which had been used by the Department of National Savings. It was covered in savings tickets. And from there we started to work, and we had to look for a house. Eventually we found quite a pleasant house, just outside Cardiff, a great big barrack of a house we thought it was. But still it was there and we lived there for seven years. Well now, the job with the pneumoconiosis was to … first of all the, the task was to find, we were set in this unit, was to find out about the re-employment and rehabilitation of miners with pneumoconiosis. And the local municipal hospital in Cardiff – it was outside, the hospital, a place called Llandough – gave me a whole hospital ward to use for this rehabilitation, the rehabilitation and re-employment. But we had to find out a bit more about the disease because I knew little about it. And I was greatly helped by a Dr Alice Stewart, who’d been working on industrial problems with Professor Leslie Witts, a former chief of mine at Oxford. And she came down, and was a tremendous help to me because she’d had some experience of surveying industries. And so what we did was, we decided we must look at as many compensated people as possible to see what their problems were, what sort of work they might do and so on. And so we wrote to the trades unions and asked them to meet us. And they met with us, the coalminers’ union(?), and they gave us all possible help. And we arranged, through them, to have meetings of compensated miners, in different parts of the coalfield, and particularly the Rhondda Valley because that was near the, Cardiff. And we went up there with, just Alice Stewart and myself, and Louisa my wife, and we handed questionnaires to the miners to fill in about whether they’d be willing to come up for examination. And for that we had to sharpen a lot of pencils. We bought a hundred pencils, and we bought a pencil sharpener, a mechanical one. You try sharpening a hundred pencils – inside it gets red hot! But anyway we had to get a hundred; we did it by hand. And then we went out and heard these miners – it was most impressive talking to these miners, their extreme keenness of, for help and what we were going to do. And talking to miners about pneumoconiosis, the little I knew about it then, it was extraordinary because they were coughing all the time. The whole audience was coughing. Normally when you talk to people it’s silent, and then if you bore them they start coughing. But the
miners, they were coughing, and when they got, when they got really interested, when
I started talking for instance about finding out about the prognosis, the future they
had, suddenly there was silence and nobody coughing. It was a most eerie feeling.
Well, through these meetings, we arranged to get miners to come up to this little
house in South Wales, and I recruited a couple of doctors to help. And we set to work
and examined them, x-raying them in the local Cardiff Royal Infirmary, and looking
at, making, filling in questionnaires about their occupation and so on. Now, at the
same time, we managed to open this ward, and one of these people we’d seen – we
admitted people to the ward, to see what they could do. And I remember seeing a
man who’d been in the Army as a ... a physical instructor, a gymnast, and he
undertook the training of these men. And the interesting thing was – very important
for the disabled – they were nothing like as disabled as they were. And this man
managed to get them going with exercises and running and so on, and they, they
found that a great many of them were really pretty fit. They couldn’t go on mining
really, apart from the regulations that meant they couldn’t get compensation without
leaving the industry. Well, that was one side of the work, and we eventually wrote a
report on this. But it was very delightful, meeting these miners and discovering their
courage. There was one miner who came up a second time, a year later, and I said to
him ‘Are you any better since you’ve been here?’ ‘Oh yes, doctor’ he said, ‘I’m
much better.’ And I said ‘In what way are you better? Are you less breathless?’ ‘No’
he said, ‘I’m just as breathless.’ And I said ‘Well, is it your cough?’ ‘No, Doctor. I
just have my cough.’ ‘Your tightness in your chest, then?’ ‘No, Doctor. I’m just as
tight.’ I said ‘Well, in what way are you better?’ He said ‘Spiritually, Doctor.’ By
that he meant, through the whole of the previous winter, which had been a very cold
one, he’d walked through the snow to his work, which he would never have thought
of doing before. So that morale building was one of the important things to do. Well
now, on the more scientific side, the things that we were, we had to do was first of all,
to try and improve the x-ray diagnosis, because we wanted to see about progression of
the miners after they’d left the industry. Did the ones who left the industry do better
than the ones who stayed in? Well, we had all the x-rays for the miners who had been
x-rayed by the Medical Research Council, in their study in 19, before the war, in
1939. So we took those and a lot of those men were still in the mines. So we got
permission for them to come out of the mines and come for a day to Cardiff, and we
had the other ones who had been compensated. And so we had a complete spectrum
of the disease of people in the, in the mines, and outside. But for this we had to get an
improved classification. The question was, were we in our unit classifying the stage
of the disease at which miners were certified at the same level as the official Silicosis
Board, as they were still called, were certifying them? So I got out 100 films, and got
them to read them, and our people read them, of the early stages. And one of the
things we found was of course a tremendous difference in the readings that different
doctors had of the same film – observer error. And we got round that by having,
giving out standard films, from these films we took the average reading of all the
doctors, and then took the average for a category number O, category 1, 2, 3 and 4.
We took this exemplary film, and in future, miners were … the people reading the x-
rays read them in comparison with these.3

1 On this subject see CM Fletcher et al, ‘The classification of radiographic appearances in coalminers’
pneumoconiosis,’ Journal of the Faculty of Radiologists, 1949, 1: 3-23; CM Fletcher, PD Oldham,
‘The problem of consistent radiological diagnosis in coalminers’ pneumoconiosis,’ British Journal of
Industrial Medicine, 1949, 6: 168-83.
MB Were there great problems across the country, apart from in this area, in interpreting x-rays? Was this a problem?

CF Well, it was the first I’d come across it. Then when we stumbled on it, we… At the same time, the Americans had found tremendous error in the reading of x-rays for tuberculosis. And then gradually a knowledge [grew] of the extraordinary fallibility of clinicians, and indeed biochemists or any experts, in particularly subjective judgement of things like films, or obesity, or fatness, or redness of the throat. Anything of that kind, there’s always observer error. And the other thing we found was that duplicate readings, with two people, reading side by side, and when they disagreed looking at the standard film, reduced, reduced the error very considerably. Now, that classification we got out has now formed the basis of a much expanded international classification for all forms of pneumoconiosis – not only coalminers, but in many other dusty industries such as asbestos. And it is really, now, the basis of the standard international classification. So we did achieve that. We also had to develop methods for better measurement of the disability of these miners, physiological tests. And we did a very big development of new physiology, lung physiology tests, which was at that time growing all over the world. And we developed tests, fairly simple tests, for measuring the disability that the miners were suffering in their lungs. The main one consisted of a little machine which you just told the miner to give a breath [Dr Fletcher demonstrates], blow as hard as he could, because the difficulty the miners had was in getting air out of his lungs. So you could measure it fairly easily by just this very simple technique.4

MB What had actually gone wrong to actually cause that, Charles? This is expiratory volume being impaired.

CF Well, the, the… We now know a bit more about it, and it wasn’t, much of it wasn’t due to the dust. Because that is what we discovered, that this disability was very slightly due to the dis-, deposition of dust in the lung which caused the first x-ray abnormalities. The main thing was the second stage of the disease, when huge lumps of … of fibrous tissue developed in the lungs, which we called ‘progressive massive fibrosis’, to distinguish it from the simple pneumoconiosis. And that was the one that was disabling, and it just contracted the tubes in the lung so that the miners couldn’t get the air in and out of their lungs. But particularly out was always more difficult – for technical reasons concerned with the elasticity of the lung. But it’s always more difficult for people who’ve got lung fibrosis, to get air out than to get it in.

MB So they might have difficulty … reducing the volume of…

CF Well, they had difficulty. Well, you see, when you blow out, your tubes contract anyway.

MB Right.

CF So if you’ve got rather contracted tubes, they shut down much quicker than they do with normal ones. That’s really the basis of that.

4 The findings of this physiological research were written up in J Gilson, P Hugh-Jones, *Lung function in coalworkers’ pneumoconiosis*, Medical Research Council Special Report Series No 290, 1955.
MB Charles, one other question. Was there confusion at that stage about the
disease, its actual structure? You were eventually finding this massive … kind of
fibrosis.

CF Well, I should perhaps have mentioned that a bit earlier, I’ve gone ahead. We
did discover in our study of the compensated miners, and the miners in the,
underground that there were two quite distinct types of disease in the x-rays. One was
the simple dust disease, which produced a sort of speckling pattern in the lung, which
had been called reticulation; we just called it pneumoconiosis. And then a second
stage, with these massive shadows, which we believed was a quite separate condition
which was superimposed upon the other. And we found a very important thing in that
follow-up study. And that was that the miners who stayed in the mine developed
more simple pneumoconiosis, that they got more dust in their lung, whereas the ones
with simple pneumoconiosis came out, did not get any more dust in the lung, so they
didn’t advance. But the attack rate, that’s, say, the onset rate, and the progression rate
of the massive disease, was just the same in those in the mine as out of the mine.

MB Right. Am I right in thinking that some people working in that field at the
time might have thought there was a link between pneumoconiosis and tuberculosis?

CF Well, that’s what we thought. I’ll come on to that in a moment. But first of
all, I’d like to make it clear, another very important observation we made was among
the miners who stayed in the mine, that they didn’t develop the massive disease unless
the simple pneumoconiosis in their lung was as much as category 2. So you have
normal, category 1, 2, and 3 – [they] were the categories that we used. And if they
were less than category 2 they didn’t get this disabling complication. So we were
then able to make the suggestion that if all miners in the industry were x-rayed at
regular intervals, and a miner was found to be developing from a normal lung to a
category 1, he should then be watched very carefully. And if he was going on, on
from there, he must then be removed to the mine before he got to category 2. And
that has formed the basis, subsequently, of the compensation scheme, that miners
were not compensated unless they’ve got category 2 pneumoconiosis on leaving the
mine. So that was very important. Now, the massive disease – was it tuberculosis?
And my colleague, Archie Cochrane… And I should perhaps have said that during
this period I’d been recruiting a large team. And I’d got hold of a marvellous team.
There was Dr Hugh Jones, Dr Gilson\textsuperscript{5} who did the physiological work and the clinical
work in the hospital. There was Dr Martin Wright who later on became a great
medical inventor, who did our … animal experimental work. And there was Dr
Cochrane who came to do the epidemiology, and that is the study of the relationship
between dust in the mines and the amount of disease developing. And he was
particularly concerned with the… Incidentally, I should say, we did have a group of
physicists working on measuring dust. They were not of the same quality, and they
didn’t produce anything really worthwhile, and we had to run that branch of the unit
down eventually. But they, one of them did produce a simple little machine for using
a hand pump to draw dusty air through a filter, and then assessing the blackness of the
filter. It wasn’t accurate, but it did give us some measure of the dustiness of mines, of
the mines. Now, Archie Cochrane had a brilliant idea of a way in which they could
study the role of tuberculosis in this massive fibrosis. What he did was to take the
whole of one part of the Rhondda Valley – it was called the Little Rhondda Valley –

\textsuperscript{5}Dr John Gilson.
and he decided that he would x-ray everybody in the valley. A big undertaking – there were about 100,000 people there. And he… No sorry, 50,000 people in the Valley, of whom about a third were miners. And every single one of them had to be [x-rayed], and he got a 95 per cent response rate which had never been done in any … epidemiological field study before. And then his great idea was to, having found all the cases of tuberculosis through the x-rays, to isolate the cases of tuberculosis, and keep them in the sanatorium, then go to a neighbouring valley with the same number of mines, x-ray all the miners there, and then compare the attack rate among the miners in the Rhondda Valley where tuberculosis had been removed with the attack rate where it had not been removed. And the idea was we’d be able to find whether the massive fibrosis disappeared from the Rhondda, and continued in the other valley. But just at that time, streptomycin was discovered for the treatment of tuberculosis, and so in both areas tuberculosis was very rapidly cleared up. So that scheme didn’t work. But what it did do was it gave Archie Cochrane a tremendous field of miners who’d been x-rayed and had a clinical questionnaire filled in too, on which he could do research into other diseases. And he also x-rayed all the people in the neighbouring valley where there weren’t any mines so as to have a non-mining community, again getting an enormous response rate. And he did a lot of very interesting studies in general epidemiology of disease in those two areas. Well now, the final thing about dust and disease, one of the funny things was that it did seem, from certification rate, that the coalminers’ pneumoconiosis was very largely confined to South Wales. There was very little outside South Wales. There was a little, but very little. Well now, was that because the South Wales mines were just dustier, or was there something about the dust that was particularly harmful? And so we had to find some really dusty mines in the rest of England, where we could find out if they didn’t have, if they did or didn’t have pneumoconiosis. And the short answer was that, in fact, it was found that they did have much less pneumoconiosis in these much less dusty mines although it was thought that they probably had as much, up till that moment. So, they were, the mines were just as dusty as South Wales but less pneumoconiosis. So there was something about the South Wales dust. Well exactly what that is is still a bit of a mystery. It’s probably something to do with the shape of the dust particles. But we, it isn’t really known exactly what the difference is. But so far as the safe levels of dust is concerned, having got a good method of measuring the amount of pneumoconiosis – and by that time, we’d developed advanced methods for accurate measurement of the sort of dust that gets into the lung, the fine dust that sticks in the lung – it was then possible to select, say, 20 pits throughout England in different parts of the country, regularly x-ray the miners, regularly measure the dust. And then you’d be able to see how much dust caused how much pneumoconiosis, and therefore you’d know how much reduction was necessary, or dust was necessary, to get rid of it. Well, at that time the coalmining industry had been nationalised. The National Coal Board was set up. And although all the members of our unit didn’t agree, I thought and most of them agreed that really this was a problem to be handed over to the Coal Board. [It’s] been handed over to them, they’ve carried out regular x-rays of all the miners working in their industries, and they’ve done a magnificent study comparing dust and disease in their lungs. And they’ve really found the answers to all the problems of the relationship between dust and disease. Not only that, but because they’ve been x-raying all the miners, and removing those

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7 Streptomycin was discovered by Selman Waksman in the USA in 1944.
that have got signs of a dangerous degree, degree of disease, pneumoconiosis is now a considerably rarity among working miners in this country. In fact, the work that we developed there laid the foundation for virtually the disappearance of this disease from British coalmines.

MB And had spin-off in other industries, because of the techniques you’ve pioneered.

CF It had spin-off in other industries, because of the techniques that we’d applied, and that … particularly the technique of measuring dust by accurate, accurate assessment of x-rays. That was the, perhaps one of the most important developments we made.

MB And it set the direction on your career, which went towards the chest rather than…

CF Well yes, it certainly did.

MB …the gastrointestinal tract.

CF I, well… After we, I felt that we’d really more or less got the makings of the answers at that stage. I’d been down there seven years, and I was rather wanting to get back into general medicine. I was very luckily offered a job of senior lecturer in chest diseases at the Hammersmith Hospital, where I’d been working before I went to South Wales. And I gratefully accepted it.

MB And we can go on to look at what happened then.
Part Two: Bronchitis and Emphysema

MB Charles, your move to respiratory disease and your distinction in this field was greatly aided by, by the smog, the London fog of December 1952, which was quite a remarkable event.

CF Well, that was an important … episode in the history of bronchitis and emphysema. Perhaps I ought just to, before we get on to the fog, I'd just like to explain what those two words mean.

MB Yes, please.

CF Bronchitis means the production of phlegm from the chest. It is, strictly speaking, means inflammation of the bronchi. And there usually is some sort of inflammation there. But when people cough up a lot of sputum regularly, that is known as bronchitis. Emphysema is a condition of the lung in which the lung is expanded because the air can’t get out of it, and in fact the air, the lung tissue is broken down. And they were thought to be related, in the old days. But very little attention was paid, in British medicine, at all to bronchitis and emphysema, before the 1945 war. I looked through ten years of British Medical Journal and found, only found the emphysema once in the index in those ten years. But after the war, two things happened. First of all, the National Health Service was set up, and a lot of people came back from the Army. Have you seen soldiers with bronchitis? Tuberculosis was coming down anyway owing to public health measures, and so there was rather less tuberculosis. And they set up, in, under the National Health Service, chest physicians set up chest clinics, and they saw a lot of people with this bronchitis and emphysema and thought ‘What the hell is this?’ And it was obviously important. And then, out of the blue came the great fog in 1952 in London. And this was because of what’s called an inversion, when the cold air is trapped down below by, with the … the air down below getting cold with frost, so that it doesn’t circulate up as it normally does from the warm air, and getting up. There’s a cessation of that circulation. And fog, also, was induced by the amount of smoke in London. And I shall never forget that December. I was at a scientific meeting and came with a former colleague of mine, John Gilson, who was staying with us, to get home from the centre of London in a car. We just couldn’t get through the fog. It was impossible. We had to get out some way away from home and walk the rest of the way. And walking was very difficult. This sort of fog was not very uncommon in London. I remember, as a boy, fairly dense fogs. They were called smogs because of the smoke and the fog together. And, but this one went on for a week. And the dramatic thing that happened was all the wards, all the hospitals in London, were filled with people with bronchitis, choking to death with inability to breathe. My wards filled right up, wards all over the … and there were a thousand deaths in the period of a week, just from bronchitis. Well now, this created a most enormous amount of interest and alarm. And one of the things that the Medical Research Council did was to set up a committee on the aetiology of chronic bronchitis. And I was asked to be secretary of that. Professor Christie, who was professor of medicine at Bart’s was chairman of it. And we had a group of epidemiologists. And chest epidemiology was then becoming quite a popular subject. I had learnt about it in

8 MRC Committee on Bronchitis Research.
9 Professor RV Christie.
South Wales. I was doing clinical work, but my main interest was in epidemiology. Well now, the … at that time, one of the difficulties that was inhibiting… Or should I say about the smog and the effect of it? One effect was a great interest in bronchitis, in the medical profession. The other, generally, was a tremendous interest in government in getting rid of smog because of the deaths it had caused. They hadn’t been worried for years with all the filth and the inconvenience of it, but when suddenly a thousand men were killed, suddenly government was stirred. And I was on several committees about working out how to keep the air clean. And I was always saying ‘Look, there are a lot of other effects of smog as well as this, inconvenience, you see.’ And they said ‘No no, we’re interested in deaths.’ And what happened was, the Clean Air Act\textsuperscript{10} was passed, and from that time on it was then illegal to discharge black smoke from chimneys in towns, and ultimately in the country, all over the country. And this has resulted in the blackening of buildings in our towns disappearing. And that’s why, now, they’re cleaning all the buildings in London and they’re staying clean, and will stay clean. All because of those thousand men who died in the smog in 1952! A very remarkable phenomenon.

But now getting onto the scientific side of it, one of the difficulties then was that the definition of what exactly was meant by emphysema was very uncertain. In America they had an idea that you could measure it by functional tests, by showing that the lung was distended so that after you’d taken a deep breath out, you had an abnormal amount of lung, air in the lung. Well if you say that, then a man, somebody with asthma has got it, and emphysema then becomes a thing that asthma people get. But it’s quite a different thing because asthmatic people get well – they have short attacks – whereas the other people are chronically ill. And in 1957 I happened to go on a lecture tour around America, talking about bronchitis in England and trying to find out about the differences of understanding. And they were very great, which is partly because it was traditional in America that bronchitis was unimportant – a cough and the sputum was the general practitioner’s disease and didn't mean anything, whereas in England we thought it was probably a precursor of the emphysema and the breathlessness. And I went to one clinic in Denver, Colorado where the physician in charge, Roger Mitchell, had a sort of … almost a sort of mutual help clinic in which they explained all their difficulties to each other and said what they were doing about it. And I said to him in this clinic ‘Do you mind if I ask these men a question?’ And so I went round each man, just saying ‘Do you have a frequent cough producing sputum?’ and they all said ‘Yes.’ And I said ‘At times does the sputum go yellow and you feel unwell and find you’re more breathless?’ They said ‘Yes that’s what happens to us.’ And after the clinic Roger Mitchell said to me ‘Is my face red? I’ve never asked those questions.’ And that was the chronic bronchitis they had, producing occasional infection in the chest, which we eventually came to call purulent because there was pus in the sputum making it yellow. Well, when I got back to England I thought well, we’ve got to do something about this. And I had a talk to John Gilson and, who was then … my successor at the clinic in South, at the unit in South Wales, and Professor Donald, Kenneth Donald, who was professor of medicine in Edinburgh, and said ‘Don't you think we ought to have a conference between the Americans and ourselves to get out ideas straight?’ And he said ‘No, I think we ought to have one in this country to get our ideas straight.’ So I approached the CIBA Foundation and they arranged the symposium. I took the chair of it and we had about twenty people, all experts in various aspects of this field. And we produced a report

\textsuperscript{10} 1956 Clean Air Act.
in 1959, which I wrote most of, on...\textsuperscript{11} We’d had tape recordings of the whole conference and I made up the report from these tape recordings. And we decided we would define emphysema in quite a new way as a – on a, a pathological basis or an anatomical basis – as destruction of the air spaces in the lung. And this interestingly enough had become possible because a pathologist in South Wales, Jethro Gough, had developed a technique for demonstrating dust diseases, in which he took the whole lung, after … death, and inflated it with gelatine, and then fixed it and cut large sections of the whole lung. An inflated lung or a large section you could put up on x-ray screen and see all the holes in the lung due to emphysema. So it had become possible to assess it after death very, very clearly. We thought, therefore, it was right to do it on this basis. We had an American visitor at our conference, AP Fishman. He was dead against this – he thought we ought to have the American definition of expansion of the lung changed in the function test. But luckily this stuck and has now become internationally agreed. At the same time we decided we’d define bronchitis entirely on the basis of cough and sputum, and we’d introduced a new term which was generalised air-flow obstruction, which meant that people couldn’t get the air out of their lungs, as I was saying about pneumoconiosis. And that happened in asthma because the, the tubes had contracted. It happened in emphysema because the lung was no longer as elastic as it was. So it didn’t… When you breathe out the lung normally just contracts because the lung is like a rubber sponge – it goes back to starting point after you’ve inflated it by breathing in. And so we defined it as … asthma as a generalised obstruction of the lung which was variable from time to time and responded to treatment, and then persistent obstruction which was characteristic of emphysema, which … it was not responding to treatment. And those definitions have, have stuck and it’s been a great advance in international understanding. Well now, it was important to clarify the natural history of this disease. And one extraordinary thing had been that until the … Doll and Hill study of the effects of smoking on the lung in relation to lung cancer,\textsuperscript{12} nobody had realised that smoking was dangerous. And Professor Christie at St Bartholomew’s Hospital had given a college lecture on emphysema in 1945 in which he’d never mentioned smoking. But we’d now discovered that this disease only occurred in smokers, or virtually only – there were a few, very few cases in non-smokers. So smoking had become important. And I had done at that time a study of the prevalence of bronchitis in Post Office workers, in the Central London Post Office and in the Post Office Savings Bank. And we’d found it was extremely prevalent, and we found that the people who had a cough and spit were much more likely to get breathless, and have an impairment of lung function than those without sputum. And, at that time the pathologists had said they thought that, looking at the lung after death, that infection seemed to be associated with the development of emphysema. So we thought the natural history of the disease was: you got, smoked a cigarette, that gave you a cough, the cough made you liable to infection in the lungs because the drainage of the tubes was not so good in the presence of this sticky mucus, and the infection destroyed the lung causing emphysema. And indeed the Medical Research Council produced a classification of bronchitis: simple bronchitis – cough and sputum; mucopurulent bronchitis – with recurrent attacks of purulent sputum; and obstructive bronchitis – with obstruction to

\textsuperscript{11} CM Fletcher & EJ King (eds.), \textit{Industrial Pulmonary Diseases: a symposium held at the Postgraduate Medical School of London, 18\textsuperscript{th}-20\textsuperscript{th} September 1957 and 25\textsuperscript{th}-27\textsuperscript{th} March 1958}, London: J & A Churchill, 1960.

\textsuperscript{12} R Doll, AB Hill, ‘Smoking and carcinoma of the lung – a preliminary report,’ \textit{British Medical Journal}, 1950: 2, 739-748.
the airways. Well now, luckily we were slightly sceptical about this and thought we ought to be sure of it. And as far as we could see the only way of discovering what the natural history of the disease was by getting a large number of men who were prone to bronchitis – that is to say working men and smokers – and following them for a period of five years to see which people started developing air-flow obstruction and who did not. Was it the people who got the illnesses or was it … did anybody get it? And from 1965, I’m sorry, from 1961 until 1965, I ran a prospective survey of measuring lung function with simple tests, and asking about cough and sputum, and recording illnesses in a thousand men in the Post Office, in the Post Office Savings Bank, and in the London Transport engineering works at Chiswick where they repaired the buses and the tube trains.

Well, after five years we realised we hadn’t got quite enough information, that rate of change of the, of the lung function was not fast enough. So we did another three years on top of that, and I’m most grateful to the MRC for financing all this. So it ran for eight years. And then I had the great good fortune to have Richard Doll, who ran the MRC statistical unit, to give me a brilliant young statistician to help me with this work, analyse it – Richard Peto. And he absolutely transformed our analysis and simplified the whole thing. And anyway, to cut a long story short, what we found was that during the period of observation the rate of decline of lung function was linear. And there was considerable variation about it and a certain amount of observer error in measuring it, but still it was linear. And you could draw a linear decline and you could then use this rate of decline as an index of the development of emphysema, obstructive bronchitis, whatever it was. And we found that this was highly correlated with sputum, but… This is a slightly complex problem, but if you have people who are declining at different rates through life, if you look at them in the middle of life, the people with the lowest lung function must have been going down faster than people with the slower one. So there is a built in correlation between low FEV – where FEV is the name Forced Expiratory Volume – for the test we used, and rate of decline. So you can measure your rate of decline by the FEV, they’re so highly correlated. But that also means that if you want to see what other factors, other than in-built liability to go down fast, or smoking, what other factors are related to rate of decline you’ve got first of all to standardise for the rate of decline. And we found that although as we’d found in a cross, cross-sectional survey that sputum and loss of lung function were correlated, and frequency of infections were correlated, after you’d standardised for the rate of decline there was no correlation left at all except for smoking. Smoking was the one thing which varied in the course of the survey; some people increasing smoking, some people decreasing. And it varied during the survey and that variation was highly related to the changes in the rate of decline of FEV.

Now that was so surprising we felt we ought to check it, and another ingenious way of checking it, that Richard Peto worked out, was to look at the change of FEV before and after an increase in sputum or an infection, an occasion of infection. If infection as we all knew reduces the FEV temporarily, there’d be a drop at the time of the FEV, it would then go back and you get a linear rate with a single deviation. If on the other hand that X-, that illness is causing a permanent drop the line will be going like that [Professor Fletcher indicates a decline] then it will have a permanent drop and go like

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13 The results of this study were written up in C Fletcher, The natural history of bronchitis and emphysema: an eight-year study of early chronic obstructive lung disease in working men in London, Oxford; New York: Oxford University Press, 1976.
that [indicates a decline] so there’d be quite a different sort of deviation. There’ll be a big deviation from the straight line on each side of the illness, and that wasn’t so. The illness was behaving like the single drop and back to where you started. So that was a, a major advance in that. And it also became apparent that since this was a life-long process, if you got hold of people in middle age, if you could pick up the people whose, whose lung-function was deteriorating, was below normal, you’d pick out the people who were going to get into trouble. If you got them to stop smoking you could save them because we found that the people who stopped smoking went back to a non-smoker’s rate of decline. So stopping smoking stopped further damage to the lung from emphysema, and so you had one way of protecting people.

MB Has the medical profession taken on the, the responsibility of that kind of screening and advisory work?

CF Not very much. There are certain difficulties about it. First of all, it’s a nice idea that, but the fact is you ought to stop everybody smoking, from smoking because of lung cancer and heart disease. So there’s not much point in doing a lot of lung-function tests to find out who you ought to stop smoking. But we did discover… And it was discovered by Donald Reid in a big survey he did on the Post Office, studying the effects of health education about smoking on the incidence of lung cancer. He found one of the most effective ways of persuading people to stop smoking was to show them the graph of rate of decline of function in non-smokers and smokers which we got in our survey. So it’s a very efficient tool to use to persuade people to stop smoking. But that was now an important thing. There are two other aspects of this problem that I’d like just to mention. One is an observation which is not of very great importance now but was twenty years ago, and that is there seem to be two types of people with severe obstructive lung disease – severe air-flow obstruction. And these were described by the Professor of … of Medicine – he was reader then at St Thomas’ who went to St George’s’ later – Tony Dornhorst. And he described the ‘pink-puff’ and the ‘blue-bloater’, as he called them; he called them blue and bloated. There were some who had normal pink faces because they were getting oxygen into their lungs, but they were terribly breathless, they were battling away to keep their, their blood oxygenated. Now when blood loses oxygen it goes blue and you get blue in the face therefore. These other people were allowing themselves to get blue and they developed severe heart failure and usually died of heart failure, and so they were called bloaters because they developed swelling of the ankles and generally, actually physically they looked a bit… They were squarer types and rather, as opposed to the bronchitis, the emphysemas who were rather taller and thinner. And everybody thought that when a bronchitic became breathless it was due to emphysema, and what we discovered at the Hammersmith [was that] this wasn’t true. And these blue-bloaters had no emphysema in their lungs at all. It was [an] entirely different disease. And it, it never received any title other than obstructive bronchitis, which wasn’t quite correct but it was a reasonable word. But the interesting thing is that it wasn’t only caused by smoking – it must also have been caused by air pollution. Because as air pollution has declined in England and Wales, bronchitis has declined very steeply, the deaths from bronchitis. And in particular these youngish men with heart failure, the blue and bloated type, have disappeared from the hospitals altogether – they’re just not seen now. So this rather interesting observation we made of these two types of disease was very interesting at the time but is now rather irrelevant because its, its…
MB  Almost disappeared.

CF  Almost disappeared. One other aspect of the problem I might just mention, that is the difference between asthma... We were never able to make a sharp distinction between asthma and bronchitis by the definition that we made at the CIBA conference, because we said that asthma was air-flow obstruction that varies greatly from time to time. Well anybody with bronchitis or emphysema does benefit from a bronchodilator medicine whether it’s taken by inhalation or given by drug. So there’s some reversibility – not much, but there is some. And when we tried to find a, a dividing point we couldn’t really do it – there’s no real distinction. You can make a distinction if somebody develops airflow obstruction in childhood because it takes a lifetime to get the other disease. So in childhood and early adult life anybody who gets this and if it’s reversible, and it usually is, that’s asthma. But the late onset asthma, which does also exist, that can be very difficult to distinguish from, from bronchitis still and we haven’t really got a sharp distinction yet. I think we will get one before very long on the basis of causation, but as we don’t know the causation of this late onset asthma, we can’t yet define it.

Well, those are the three areas of bronchitis in which I’ve been mostly concerned, but of course particularly concerned with smoking.
Part Three: Action Against Smoking

MB Charles, later in your career we’ve talked about the bronchitis interest developing. But you’ve obviously got an enormous interest in the part smoking was playing in the aetiology of so many diseases, and became president of a major organisation trying to stop smoking, and became responsible for a report that sold information about smoking very widely among doctors and in the community and attracted a lot of press attention. So, in recent years you’ve had a close association with the message ‘Don’t smoke.’ Can you tell me about that?

CF Well, I think to get the background of this it would be desirable just to recall the history of awareness developing that smoking might be harmful. Because, for many years, it had been thought by strict-minded fathers that it was bad for boys to smoke, but they always smoked themselves. And there was no real evidence that smoking really did any harm to anybody, until just before the Second World War, in the 1930s, when there seemed to be a steady increase in the frequency of a very rare form of cancer previously, lung cancer. But not much notice was taken about this. Two Germans published a paper suggesting this might be due to cigarette smoking… Now, cigarette smoking was a relatively new development. Pipes had been smoked in the old days. And cigarettes didn’t really start till a machine was invented for making them at about the beginning of the century, and then cigarette smoking began to spread. But still smoking was predominantly pipe smoking and entirely by men. During the First World War, women started to smoke and cigarettes began to develop into a major form of smoking and the consumption of cigarettes rapidly increased all over the world. Then this little hint before the war, that lung cancer might have something to do with it, and during the war the Medical Research Council in this country set up a committee to discuss what might be causing all this. And they thought of various things in the way of changes. They thought increased television, radio sets, aluminium saucepans, bananas. And they just put in smoking as an afterthought because they thought ‘Well, perhaps its something to do with, could be something to do with smoking.’ And as late as 1948, the American … the Journal of the American Medical Association had a leading article which said that there was no evidence anywhere in the world that smoking ever really did anybody any harm. And then the, the Medical Research Council appointed Professor Bradford Hill, later Sir Austin Bradford Hill, at the London School of Hygiene, and this young assistant of his, Dr Richard Doll, to set up an epidemiological study to see what factors were responsible for this increase in lung cancer. And the way they did it was to go to a lot of hospitals all over the country, and discover cases of lung cancer. And for every case of lung cancer they found they took another case, at random, of the same age and sex, who wasn’t, hadn’t got lung cancer, and then they compared the relative frequency of all these things they were asking about. And the one thing, and the one thing alone that stood out was that this, the lung cancer cases had all been heavier smokers, and much more, many more than the smokers … and they, those who were smokers were much heavier smokers than the others. So, and this was quite clearly quantitatively related. I mean, the heavier the smoking the greater the risk. Air

14 Here Dr Fletcher is probably referring to the following paper: E Schairer, E Schöngler, ‘Lungenkrebs und tabakverbrauch’, Zeitschrift für Krebsforschung, 54 (1943), 261-9. FH Müller also produced a paper suggesting a link between smoking and lung cancer in the same journal in 1939.
pollution came in as a possible second, but smoking was the big thing.\textsuperscript{15} And when this was published, and simultaneously there was a much smaller study published in the United States,\textsuperscript{16} it was a tremendous shock. Everybody thought ‘How extraordinary!’ The whole time that I was working among miners on chest diseases in miners in South Wales, we never recorded their smoking habits. We never thought it had any relation at all to, to their disease. We now know it was much more, as important if not more important than the dust. Well now, when that happened, the Ministry of Health is of course very slow to react to anything, but three years later, the chief medical officer did introduce, issue a statement. And the minister of health made a statement in the House of Commons that probably smoking played a part in the causation of lung cancer. Nobody took any notice. And in about 1968, that would be about … oh, 15 years after this discovery, the evidence had become more and more clear as people looked at more and more diseases or investigations of the Doll and Hill kind, comparing cases with and without disease, that many diseases were related [to smoking], and certainly bronchitis which I was interested in. Well at that time, there were still some smogs in London and I was doing a study with Pat Norwood(?) at Bart’s on the use of smog masks, whether they protected people with bronchitis from the smog. And a young doctor at the Ministry of Health called Max Wilson came to call on me, to hear about this smoking and … this … the smog masks. And as he was leaving I said to him ‘Max, when’s your ministry going to justify its title?’ And I said, well he said ‘What do you mean?’ I said ‘When are you going to do something about smoking?’ Well that led to a telephone call from Dr George Godber, who was then Chief Medical Officer at the Ministry of Health, later Sir George Godber, [who] asked me to come and have lunch with him. And I had lunch with him in his club; we discussed what the Ministry of Health might do. And we thought of various things like trying to stop smoking in trains, trying to … put posters up, and various things we thought about. But none of them seemed very likely. And at that time the Chief Medical Officer, Sir John Charles, was obviously frightened of doing anything about it – he didn’t want to offend the industry and the public. You see, at that time, smoking was an extremely common habit. About 75 per cent of the general public were smokers, and if you took people who’d ever smoked it was higher than that. Only about 10 or 12 per cent of the population had never smoked, and it was absolutely universal. Well, at the end of our lunch I said to George Godber ‘Do you think it would help if our College’ – meaning the College of Physicians – ‘were to produce a report on the evidence about this?’ And he said ‘Yes, I think, I think that might be very helpful. It might stimulate my minister to do something about it.’ Well, I then rang up Robert Platt, who was then just become President of the College of Physicians, and said ‘Robert I want to come and talk to you about something.’ He said ‘What is it?’ And I said ‘I think our college ought to produce a report on smoking.’ ‘We certainly should,’ he said straightaway, over the telephone. ‘Who shall we have on the committee?’ Straight out like that. And so we, we formed a committee, and working over a couple of years, in March 19 … 62 the report was ready for publication. It was rather interesting, Pitmans Publishing were going to publish it, and they had suggested a print run of 2,000 copies. And the treasurer of the college said ‘Look, I think we could risk 5000.’ And I said ‘Look, why not make it 10?’ And Pitman said ‘All right, so long as you stand the losses.’ And, well Platt was


prepared for that. And so it was published.  

And it was just simply a fairly simple account, which I wrote, with the help of other members of the committee on the special subjects, but I did the actual writing of it. And we didn’t mince our words, we really did write in, as far as possible in simple words, and we really made a parallel between this and tuberculosis and the big plagues of the past. And the extraordinary thing was, we didn’t expect it, we gave a press conference and then we waited to see what would happen. And it was absolutely staggering. The evening papers were absolutely full. First line, every front page of every newspaper ‘Doctors say smoking dangerous.’ They’d never taken any notice before, but here were a group of doctors saying it really was dangerous, and they’d read the report and seen that it really was dangerous. And I was on television talking to Sir John Partridge, who was then chairman of the tobacco industry, of Imperial Tobacco. And Robert Platt appeared on the ‘Panorama’ programme, talking about it. And there was tremendous publicity. But the government did nothing, except say that they would instruct the local authorities to, who were responsible then for health education, to do some health education. And it really was extremely depressing, the, the way the government just shelved us off – ‘No, nothing to do with us. We can’t do anything.’ And we battled away a bit, and a few steps were taken. But really, there was a steep decline, owing to the press publicity. But then it, it petered on at about the same level and women were steadily increasing their smoking all the time. 1971, we had another report. And in the interim, one thing that had helped had been that the government did set up the Health Education Council, and I was a member of that for the first … six years I think of its life. And I was temporary chairman of it because the first chairman, Lady Serota, resigned after the first meeting and I was asked to take it on! And smoking was the one thing we, we tackled. We made our big effort about smoking. Not to any very great effect. Then in 1971, the college produced another report, and Keith, Dr Keith Ball and I went to see the president of our college, Dr Max Rosenheim, Sir Max Rosenheim, and said we thought we ought to do something along the lines of America, where the American Cancer Society… Well that, I’m afraid I ought to put, go back into history a little bit there. Well after the report was published, apart from the immediate publicity, Pitman sold the first 10,000 copies in the first week. And the other 10, another 10,000 disappeared over the next year. And the American Cancer Society ordered 80,000 to send to all their members throughout America. And President [John F] Kennedy … then said that… He went to his Surgeon General, and said ‘Look why isn’t our, why aren’t you doing anything about smoking?’ And he set up a committee to report on it, and from that day on, the Surgeon General’s reports on smoking, progressive, one after the other, have been one of the most important sources of information about smoking. Our report was directed at the intelligent layman. Theirs had been directed at doctors and scientists, and they are the most valuable source book for science on this. And they set up a, a … a Department of Health division to deal with smoking which has been extremely effective all over the world.

MB So the American, the Americans started after your initiative, and that was the Surgeon General’s report.

That was the Surgeon General, the first Surgeon General’s report in 1964. Our report was ’62.

Yes, that was pressed by John Kennedy’s intervention?

That’s right. That was, he was pressed to do that by John Kennedy, Kennedy’s intervention. Well coming back to this country, in 1971, this report, the second report came out, and because we were… We thought we really must get the Department of Health to do something. And we thought also it would be useful to do what the Americans had done. They’d had a, an Interagency Council on Smoking and Health, which meant their Cancer Society, their Heart Society, these big commercial bodies they had concerned with public health concerns in different areas, had got a joint body which was doing a lot of work. And we said we ought to do the same thing over here. But of course it turned out that our … people over here were very different, because we don’t, they aren’t propaganda agencies at all, they are just research and clinical investigation agencies. And we got nowhere with that. But we did set up this organisation, which was called Action on Smoking and Health, which was intended to muster voluntary effort to do more. And at the same time our second report had as little effect, just a small temporary effect. If anything, less than the first one. Except that the Department of Health did then have a voluntary agreement with the manufacturers to restrain advertisements, and they insisted on a health warning on advertisements and packs. A pretty mild one – ‘Smoking may damage your health.’ I mean it wasn’t going to stop anybody smoking, the tobacco manufacturers were quite certain about that. But ASH has been an important organisation. At first we were pretty ineffectual. We tried to raise a lot of charitable money, to give us help, but we failed in our appeal – we only made very small amounts of money. But then the, the doctor who was directing it resigned, and in his place we recruited a most remarkable young man called Mike Daube. We only had three applications for the post. The other two were one elderly lady and one elderly gentleman. And this young chap, Mike Daube… And he was really a, a born pressure-group man. He’d been working with Shelter, and had got some ideas about it, and he converted ASH from a voluntary, a lot of voluntary agencies trying to do something in a half-hearted way, into a real pressure-group. And he was really magnificent, the way he brought, built it up, got on the right side of, of government, got on the right side of MPs. When we were set up, Keith Joseph who was then minister of health had given us, secretary of state for health then, had given us a grant of £25,000 to get going and said ‘That was the last.’ But by the time Mike Daube had been with us for two years, we got regular subvention from the Department of Health, and we’ve been financed by the Department of Health ever since. And ASH was, he was marvellous with the press. He always provided information whenever it was needed. He became as well informed about the, the smoking problem, the relationship between smoking and disease, as I was with, very quickly, and could always give the right answer to any question and so on. And we set up a very small organisation with just a, a couple of secretaries and him, and he became really feared by the tobacco manufacturers. And he built it up so that we eventually had quite a sizeable office, and a staff of about four or five. It was still small, but the important thing was his feeding information to the press, and the press came more and more to be on our side. When the first college

report came out, *The Times* published a letter from a tobacco manufacturer, saying that was… No, from somebody, that wasn’t, that was some years … from a layman, saying ‘Why don’t the doctors tell us whether giving up smoking is any good?’ And Professor Bradford Hill and Richard Doll wrote a letter to *The Times*, giving the evidence that stopping smoking reduced the risk, steadily, over a period of years, and *The Times* refused to publish it. That was the attitude of the press then, because you see they all depended on them for their money, on press advertisements. And it was fairly gradually that Mike Daube worked the press round, so that now, with the exception of the *Daily Telegraph* I think and possibly the *Daily Mail*, the press are entirely on our side and tend to publish, with some lamentable failures, the sort of information we give them. The college published a third report, jointly with other colleges in 1976, and the final report was published just this year 19, last year, 1983. And the remarkable thing has been that at last lobbying of MPs has resulted in one thing, and that is that the chancellor now has been steadily increasing the price of cigarettes by increasing tax. Not only that, but he stated, in his statement to parliament he stated ‘I am doing this because of the very strong medical lobby that has approached me about this,’ that was in the last thing. And during the last few years we’ve seen smoking decline, for the first time, rapidly, in the working classes. Now before that, between 1951 when Doll and Hill started their doctor survey and 1971 when they stopped, doctors had cut their smoking from 50 per cent, 65 per cent of doctors smoking, down to 20 per cent. I believe, since then it’s dropped to about 10 per cent. And the general public, in social class I, it’s dropped from 65 per cent down to, in men, 65 per cent down to about … 38 per cent. It’s got under 40 per cent. And in women from about 50 per cent, 45 per cent down to about 34 per cent. And now, smoking is now a minority habit in the country. And the most important influence on this has been the chancellor of the exchequer increasing the price. Whenever there’s a price increase, people stop smoking. But also, intelligent people in the professional classes, sort of … doctors, lawyers and all the rest of them, now it’s very much a minority habit, as you may well know. The, the, well anywhere you go now in professional circles, anybody who smokes tends to sort of keep the cigarette behind his back. They tend to hide it. And in medical circles, virtually non-existent.

**MB** Are you happy with the, the rate of decline of smoking?

**CF** Not in the general public, because you see the professional class is a very small proportion. In social, in social class A, that is professional, in B there’s been a good decline. And in the whole population there’s been something like a 30 per cent decline in the last ten years. But it, in the main, the biggest class of people, the working classes – social classes D and E – there’s only been a very small decline and that mostly in the last five years. But they are now beginning to decline.

**MB** But the women in social class V are supposed to be increasing their smoking still? So there’s…

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CF  No.

MB  No?

CF  No, they’re levelling off. And they’re just beginning to go down. But we still
haven’t got the message very … skilfully across. The Department of Health did a
large survey of attitudes towards smoking last year [1983], and they found that only
about 30 per cent of smokers accepted that they had any risk of getting any disease
from smoking. If they did say there might be a risk to smokers, not themselves, to
smokers, and you asked them how many cigarettes somebody would have to smoke
for it to be dangerous, they would always place the figure at five cigarettes a day more
than they themselves smoked, so that they kept themselves safe all the time. And they
think that it can only occur to people with weak lungs, and people who have
something wrong anyway. Oddly enough they accept that if they did get one of the
diseases related to smoking such as lung cancer then they would blame it on their
smoking. But it, they don’t, they don’t expect to get it, so they don’t, it doesn’t
motivate them to want to stop. The biggest motivation does remain the health risk,
but it only affects a small minority of smokers – 30 per cent. And the other big one
which does affect other people, the whole of smokers, is of course expense. They say
the money of it; it isn’t worth the money they pay on it, most, most of them say. So
that’s very important. And there’s quite a proportion of them that do think the
exemplar role is important, let’s say they do think they’re a bad example to their
children. Not all, some of them don’t mind at all. But one of the most critical
questions that I arranged to have inserted into this survey was the question ‘Do you,
do you think that if smoking was really a risk, the government would ban cigarette
smoking?’ The way it was put – ‘Do you think that if smoking were as serious a risk
as, as they say the government would ban advertising?’ And 44 per cent of smokers
agreed with that statement. So you’ve got a lot of them still feeling because the
government will go on allowing advertisements, they … there can’t be anything
wrong with it.

MB  If one’s appalled by the statistics though, ten years on, fifteen years on, after
important epidemiological evidence shows smoking is bad news, and that people
ought not to smoke, is that largely due to lack of political will? Or is that because
we’ve given the public the wrong messages in the wrong terms? Or is it a mixture of
both?

CF  I think we’ve given the public the wrong messages in the wrong terms. I think
that … that we’ve failed to get the message across. It’s a very difficult one to give
because it’s a very unacceptable one. I think that, myself, that the government have
been seriously at fault in not banning advertisements, for the reason I gave about
what, what smokers think. And also because, although I don’t think advertisements
persuade children to start, or keep smokers smoking, nonetheless they do create an
atmosphere where it seems to be a reasonable thing to do or, because the
advertisements are there. In Norway, where … advertisements have been banned for
the last ten years, there’s been a levelling off of smoking. It was rising steeply.
There’s now a levelling off among adults. And in school children it’s going down.
So that I think that this is the, the main thing we want to get from the government.
And the government’s reason why they won’t do this is, I think, a paltry one. It is that
they say that since it can legally be sold, you must allow advertisements for anything that is legally sold. And you say ‘Why?’ They said ‘Well that’s just a principle.’ Now, when recently two ministers of health, the minister, the secretary of state for health himself – Patrick Jenkin, and George Young, one of the under-secretaries of state for health – they, ministers of health, decided that they were going to ban advertising, they were moved away from the ministry by Mrs Thatcher to another ministry, to environment. And…

MB Was this because of their pro … anti-smoking kind of thing?

CF Well, it’s very well-known even though they, no, they haven’t admitted it, that the advertising … the tobacco manufacturers, particularly Anglo-American Tobacco,23 approached Dennis Thatcher, Mrs Thatcher’s husband and said ‘Look, we won’t support you at the next election unless you move those two men away from the Ministry of Health.’

MB So there were serious political undertones and overtones?

CF So… It’s an, it’s an appalling political manoeuvre. The government, of course, get a vast amount of money from the tax. That’s their reason for not getting, reducing smoking, because … if people who stop smoking don’t save the money they spend it, and if they spend it, it could be taxed. It might mean an increase in VAT or something, but I mean it’s nonsense to say you can only… It’s just convenient tax for the government, many of, most of whom are not smokers and therefore they like to see the smokers being punished.

MB Charles, one final point. Do you think that if the message was aligned very firmly with heart disease instead of lung cancer, which was the great starting point, that we’d have a bigger response? The public seems to be more concerned about their hearts.

CF I think that’s true. Although 30 per cent of people admit it’s, deny it’s going to do any damage, among those who did admit damage, of the 60 per cent who did admit damage, very few of them realised that heart disease was a risk. So I think that would be a very important thing to do.

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23 Dr Fletcher presumably means British American Tobacco here.