

PULMONARY CAVITATION IN COALWORKERS' PNEUMOCONIOSIS

with special reference to

THE CAVITATION IN THE MASSIVE FIBROTIC FORM
OF THE DISEASE.

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"It would afford me much gratification if any means could be devised as regards either prevention or remedy, whereby might be lessened the evils of a disease, the ravages of which, upon the most robust constitutions, I have every day cause to deplore."

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G. Steele (1834) in a letter addressed to W. Thomson, concerning the respiratory disease to which coalworkers were subject in Scotland during the nineteenth century.

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INTRODUCTION.

The symptom of profuse black expectoration occasioned by cavitation in the lungs of coalworkers was first recorded by Gregory of Edinburgh in 1831. Various contributors to the literature on industrial pulmonary disease subsequently mentioned cavitation, particularly in silicosis, although few of them reviewed a series of cases of coalworkers' pneumoconiosis with cavitation. It was thought, therefore, that an investigation of this condition would serve a useful purpose as its frequency and significance were not, perhaps, fully appreciated.

A century ago, interest in the diseases of coalminers was most evident in the Scottish coalfield and the perspicacity and accuracy of the observations of the nineteenth century Scottish physicians will be considered in relation to present knowledge. The major social and medical problems of coalworkers' pneumoconiosis are now most apparent in South Wales where the investigations recorded in this study were conducted.

HISTORY.

The first authentic mention of coal mining in the British Isles is to be found in the records of Newbattle Abbey, Midlothian, where monks mined outcrop coal in the twelfth century A.D. and probably earlier (Carrick, 1907). In the eighteenth and nineteenth centuries when improved methods of pumping and ventilation made deep work possible, coal mining became an important industry in Great Britain and Scottish physicians became interested in the medical problems of the industry.

Reference to lung disease among workers in dusty occupations had been made previously in medical writings. Hippocrates described a man from the mines with laboured respiration, Paracelsus "the chronic lung disease of miners", Agricola the "asthma of miners" and Ramazzini discussed the occupational hazards of mining in his text book of industrial disease (De Morbis Artificum) and stressed the importance of knowing the occupation of the patient. Laënnec (1806) described "la matière noire pulmonaire", while Pearson (1813) was the first British contributor to the literature

on this subject in a paper entitled "On the colouring Matter of the black Bronchial Glands and of the black Spots of the Lungs" but from his writing it is clear that he was recording the natural pigmentation of the lungs in the general adult population.

It was Gregory (1831) of Edinburgh who first believed that the black matter arose directly as a result of employment in coal mines. Dr. J. C. Gregory (1800 - 32) was a Fellow of the Royal College of Physicians, Edinburgh, was physician to the Royal Infirmary of Edinburgh and had been a pupil of Laënnec. He described a coal miner from Dalkeith who was admitted to the Royal Infirmary of Edinburgh with breathlessness and black sputum, in whom at autopsy, pulmonary cavitation was found in the upper and middle lobes of the right lung. Marshall (1834) of Cambuslang near Glasgow described the sputa of two further patients as "presenting a closer resemblance to printers' ink than to anything with which I can compare it" and made the observation that "there can be little doubt that the black matter does not appear in the expectoration until the substance in the lung begins to break up; that the black expectoration, in fact, marks a stage of commencing

excavation.....". He further observed, "the true explanation of the origin of this disease in colliers seems to be that it is in consequence of the inhalation of fine coal dust and its deposition in the substance of the lung. That coal may float through the air in particles sufficiently fine to be inhaled without irritation....." William Craig of Glasgow wrote to Professor Graham (1834) (lecturer in chemistry in the Andersonian Institute of Glasgow) giving his findings and describing a method of investigation of the lungs at autopsy: "The best manner of ascertaining the exact situation of the black matter of such cases is by inflating the lung slightly, drying it thoroughly then cutting it into slices in various directions." This method of examination was re-introduced for the same purpose more than one hundred years later by Gough (Gough and Wentworth, 1948).

Professor John Thomson (1765-1846) and his son William made a detailed study of lung disease in miners. John Thomson was Professor of Pathology in the University of Edinburgh and his son later became Professor of Medicine in the University of Glasgow. Their interest was first stimulated in 1824, when, at an autopsy in

the Royal Dispensary, Edinburgh, black lungs with excavation were found in a man whose industrial history however, was unknown. These two physicians undertook what was the first epidemiological study of this condition when they sent a questionnaire to practitioners in the Lothians and Fife and asked Dr. Cumin, Professor of Midwifery in the University of Glasgow to seek information in the west of Scotland. In the replies there was some diversity of opinion as to the prevalence, manifestations, and nature of the condition but even in those days with neither radiography nor a knowledge of the existence of the tubercle bacillus, a differentiation into cases of "phthisis" and "tubercular phthisis" was made. Detailed accounts were given of the symptoms and of the post-mortem findings. The clinical picture was very adequately described at this time as is shown in the following excerpt from William Thomson's paper (1837).

"May 1st, 1833. Today I visited at Collinshiel colliery, along with Mr. Dixon, surgeon, Bathgate, George Hogg, a man, 40 years of age, tall and of very athletic form. At present, and for nine or ten months past, this individual has been unable to

follow any active employment. He labours under a continual slight dyspnoea, which does not prevent him from taking gentle exercise but it is always aggravated to a great degree by any considerable exertion. His breathing, even when he is at rest, is somewhat laborious and sonorous. He has frequent cough with expectoration. The matter expectorated is at present generally more or less of a dark colour, but it is only at times that this is particularly well marked. For instance, about eight days ago, when attempting some slight work in his garden, he suddenly coughed up two or three profuse sputa, the matter of which he could compare, he says, to nothing but black ink. There does not appear to be any distinct hectic fever, and the symptoms altogether approach perhaps more to those of asthma or of chronic mucous catarrh, than to those of tubercular phthisis."

This case history was submitted by Dr. (later Sir) James Y. Simpson (1811-1870) when in practice in Bathgate. He later became assistant to John Thomson and subsequently held the Chair of Midwifery in the University of Edinburgh. Thomson also quoted a letter

from a Dr. Steele of Musselburgh who made an interesting observation when compared with the modern concept of a dose response curve in pneumoconiosis (Roach, 1953; Wright, 1953): "The period of life at which the disease occurs must vary according to the length of time the individual has been exposed to the exciting cause....." Steele also commented on the expectoration of coalminers and his findings are exactly comparable with those in the present investigation: "The sputa are sometimes of a yellowish colour, often grey and occasionally black; they have also at times a puriform admixture and are not unfrequently tinged with blood."

Thackrah of Leeds (1832) was also interested in miners' diseases and published the first text-book in English on occupational medicine, and from the middle of the nineteenth century many papers were published on various aspects of this industrial hazard although few authors paid much attention to pulmonary cavitation. Black sputum was apparently well recognised in France in the last century and is mentioned frequently in Emile Zola's novel "Germinal".

Various authors have also described black

sputum and cavitation in other types of pulmonary dust disease, in graphite pneumoconiosis (Koopmann, 1924; Dunner and Bagnall, 1946; Gloyne, Marshall and Hoyle, 1949) and in the silicosis of pottery workers (Meiklejohn, 1949). A similar condition was described in iron moulders by Hamilton (1834) of Falkirk who described the black sputum but did not obtain post-mortem proof of cavitation, and by Faulkner (1940) in America.

PRESENT VIEWS ON COALWORKERS' PNEUMOCONIOSIS.

Two distinct forms of pneumoconiosis in coalworkers are now generally recognised. The first, simple pneumoconiosis is attributed simply to coal dust retained in the lungs and, as such, does not progress after exposure to dust has ceased (Davies, Fletcher, Mann and Stewart, 1949). This condition causes little disability (Gilson, 1953). Radiologically, simple pneumoconiosis is characterised by fine opacities, described and classified by Fletcher, Mann, Davies, Cochrane, Gilson and Hugh-Jones, (1949) as illustrated diagrammatically in Fig. (1). These opacities represent dust foci with or without focal emphysema (Gough, 1947; Heppleston, 1947). The problem of the differential diagnosis of chronic lung disease with diffuse nodular or reticular shadows has recently been reviewed by Scadding (1952).

Men with established simple pneumoconiosis may develop the second form of the disease (in South Wales about 2% per annum (Cochrane, Fletcher, Gilson, and Hugh-Jones, 1951)) which is characterised by the development of large localised opacities (Fig. 1.)

corresponding to the massive lesions found pathologically. This condition rarely, if ever, develops in a lung with less than category 2 simple pneumoconiosis, it nearly always progresses whether or not exposure to dust continues and often starts after the cessation of exposure to dust (Davies et al., 1949). It is called progressive massive fibrosis (the P.M.F. of Fletcher, 1948) and is a serious and disabling disease. The combination of simple pneumoconiosis and progressive massive fibrosis has also been termed complicated pneumoconiosis (Davies and Mann, 1948). The terms massive fibrosis and massive lesions are also used as synonyms for progressive massive fibrosis.

The Classification of Radiographs in Coal-workers' Pneumoconiosis.

The system of classification of radiographs in pneumoconiosis shown diagrammatically in Fig.(1) was evolved by the staff of the Medical Research Council, Pneumoconiosis Research Unit (Fletcher et al., 1949; I.L.O., 1953). The films are divided into two groups.

Simple pneumoconiosis: with subdivisions categories 1 - 3 based primarily on the profusion of the specific opacities. Further subdivision into








pinhead (p), mixed (m) and nodular (n) types of opacities may be made. Focal emphysema cannot be recognised radiologically.

Complicated pneumoconiosis (or progressive massive fibrosis): with subdivisions A - D based upon the size of the massive shadows and the degree of pulmonary distortion.

For accurate radiological reading it has been found that verbal descriptions of radiographs may be variously interpreted by different observers (Fletcher and Oldham, 1949) and it has been shown by Fletcher and Oldham (1951) that standard films may reduce observer error.

It should be remembered that this classification is purely a radiological one. As far as simple pneumoconiosis is concerned however, it indicates that dust retention has occurred and the radiographic appearances correlate reasonably well with the dust exposure (the product of average dust concentration and time (Roach, 1953; Wright, 1953)). It does not on the other hand carry the implication that a man whose chest radiograph shows any particular category of abnormality is, of necessity, "suffering from pneumoconiosis", has any

particular degree of disability or is eligible for compensation. These matters can only be decided by clinical examination including, if possible, some simple test of respiratory function.

International Radiological Classification of Pneumoconiosis			
Normal Range			Code 0/-
Abnormality simulating Pneumoconiosis but not caused by Dust			X/-
PNEUMOCONIOSIS	Category	Appearance	
Simple (Pneumoconiosis with discrete opacities I.L.O. 1950)	1		1 / - -
	2 <ul style="list-style-type: none"> Pinhead p Mixed m Nodular n 		2p / - - 2m / - - 2n / - -
	3 <ul style="list-style-type: none"> p m n 		3p / - - 3m / - - 3n / - -
Complicated (Pneumoconiosis with coalescent or massive shadows I.L.O. 1950)	A		3A 2/1
	B		3B 3/3
	C		?C 4/5
	D		?D 5/4

THE PATHOLOGY OF COALWORKERS' PNEUMOCONIOSIS.

The subject matter of this thesis is essentially a clinical and radiological study, but a necessary preliminary is a brief description of the pathological findings. The pathogenesis of coalworkers' pneumoconiosis will be discussed more fully later.

Simple Pneumoconiosis.

Simple pneumoconiosis is caused by the retention of coal dust in the lungs. The dust foci or dust macules are diffusely distributed throughout the lungs and show very little associated fibrosis in contrast to the collagenous nodules of classical silicosis. Focal emphysema may occur in relation to these dust macules (Gough, 1947; Heppleston, 1947 and 1953) (Figs. 2 a. and b.). Whether this focal emphysema plays any part in the causation of the disability associated with pneumoconiosis remains in doubt. It is considered that these lesions are produced by the simple mechanical accumulation of coal dust rather than the activity of free silica which is present only in very small quantities in coal dust (Heppleston, 1947 and 1951).

Progressive massive fibrosis.

The features of massive fibrosis in coal-workers are quite dissimilar from those of the focal lesions. The massive lesions are composed of dust and coarse, hyaline, collagen fibres irregularly mingled and containing foci of lymphocytes (Fig.3.). Blood vessels and air passages are inconspicuous. Nevertheless, the previous existence of arteries and arterioles in massive lesions is revealed by persisting internal elastic lamellae after complete obliteration of the vascular lumina by invasion of fibrous tissue accompanied by dust phagocytes. Some vessels are only partially occluded, but, irrespective of the degree of stenosis, the elastic lamella is often reduplicated and disrupted (Fig.4.). Comparable vascular effects are caused by fibro-caseous tuberculosis unassociated with industrial dust exposure (Fig.5.). Caseous foci may be found in massive lesions but do not always show conclusive histological evidence of tuberculosis.

In the genesis of massive fibrosis therefore, a factor (or factors) additional to the dust must operate. In view of the extreme fibrosis together with the presence of chronic inflammatory cells, the

most obvious additional factor is a chronic infection, and tuberculosis has been repeatedly advanced as this complicating factor. The evidence in favour of this hypothesis will be fully discussed later.

Extension of the necrotic process to involve a patent bronchus allows the liquefied material looking like thickened Indian ink, often loaded with cholesterol crystals, to be expectorated. A cavitated massive lesion results, its wall having a shaggy appearance but no lining demarcates the cavity. At the margin of these ischaemic cavities the collagen fibres and remnants of dust cells simply disintegrate abruptly (Fig. 6.), while cholesterol clefts may be numerous in the surrounding tissue, from which tubercle bacilli are very rarely recovered. Cavitation of massive fibrosis likewise follows when softening occurs in enclosed areas of caseous tuberculosis and a bronchus is invaded. These tuberculous cavities may usually be distinguished, macroscopically, by the presence of a greyish wall and purulent looking contents. Microscopically tuberculous cavities have a necrotic lining with an eosinophilic, granular appearance. Surrounding the necrotic zone there is often a cellular layer of inflammatory type in which epithelioid cells and

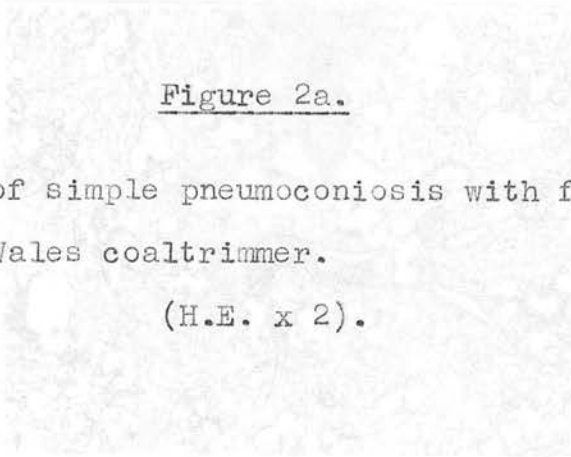


Figure 2a.

Dust macules of simple pneumoconiosis with focal emphysema from a South Wales coaltrimmer.

(H.E. x 2).


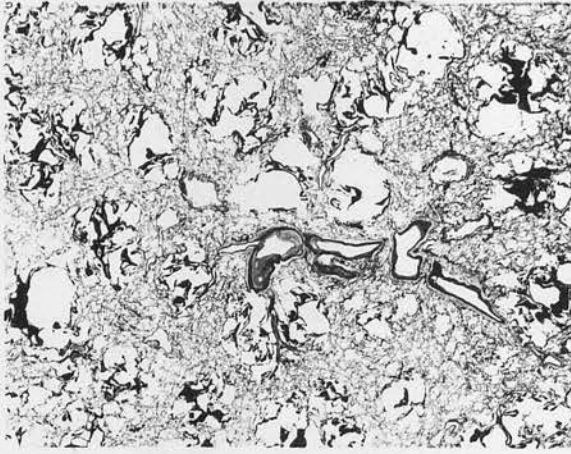


Figure 2b.

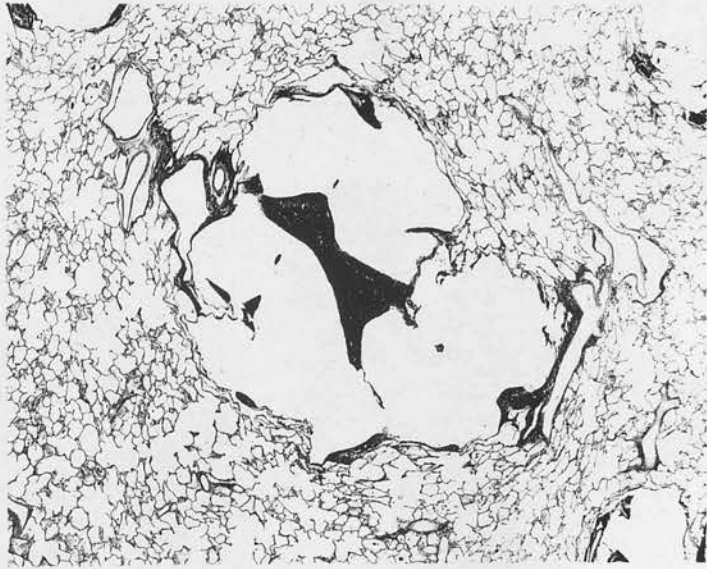
Enlarged section showing dust macule of simple pneumoconiosis with moderately severe focal emphysema from a South Wales coalminer.

(H.E. x 10).



Centre of
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inner
hyaline



Section

from

the lungs of a coal-worker showing separation of the vessel
and dust cells lying within the elastic lamina. (Stained
for elastic tissue x 120).

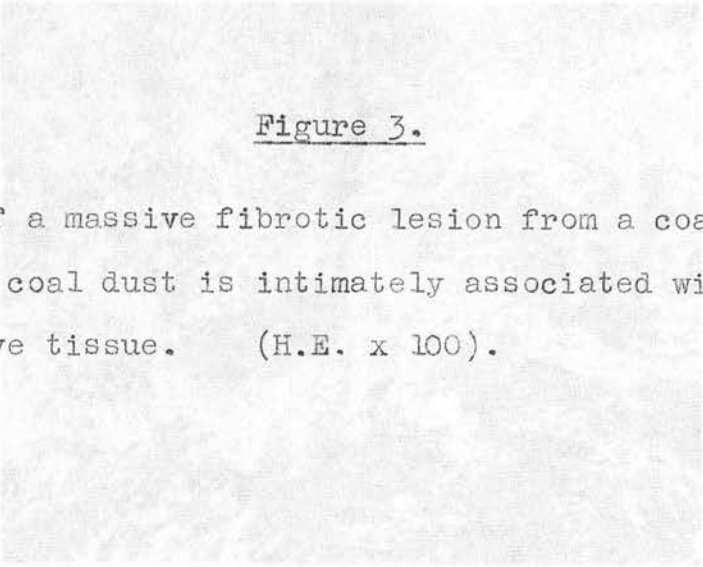


Figure 3.

Centre of a massive fibrotic lesion from a coalminer in which coal dust is intimately associated with hyaline connective tissue. (H.E. x 100).

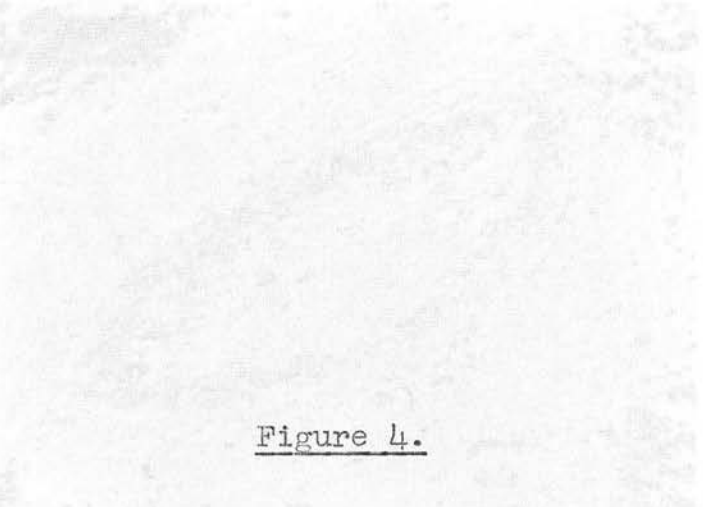
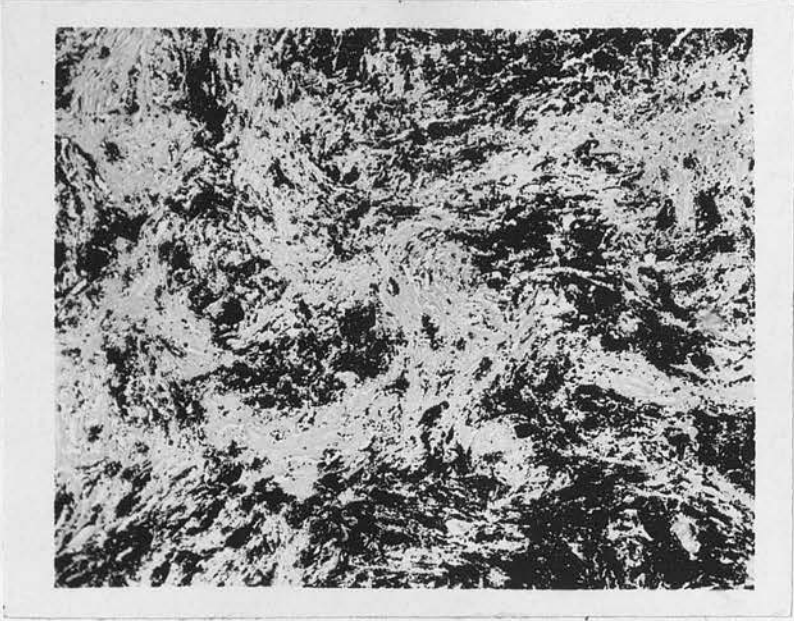


Figure 4.

Section of an artery in a massive fibrotic lesion from the lungs of a coalworker showing occlusion of the vessel and dust cells lying within the elastic lamina. (Stained for elastic tissue. x 120).



Section
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tumor
by gr

Mass of
necrotic
tissue. (x 10).



Margin
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fibrosis
from
margin
of the
cavity
as a
result
of
disintegration
of
fibrous
tissue. (x 50).

Massive
fibrosis
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margin
of the
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disintegration
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tissue. (x 50).

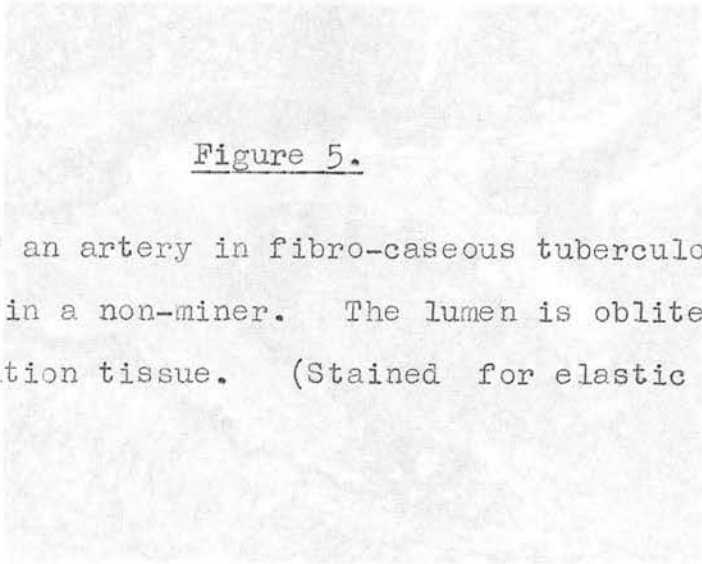


Figure 5.

Section of an artery in fibro-caseous tuberculosis of the lungs in a non-miner. The lumen is obliterated by granulation tissue. (Stained for elastic tissue. x 40).


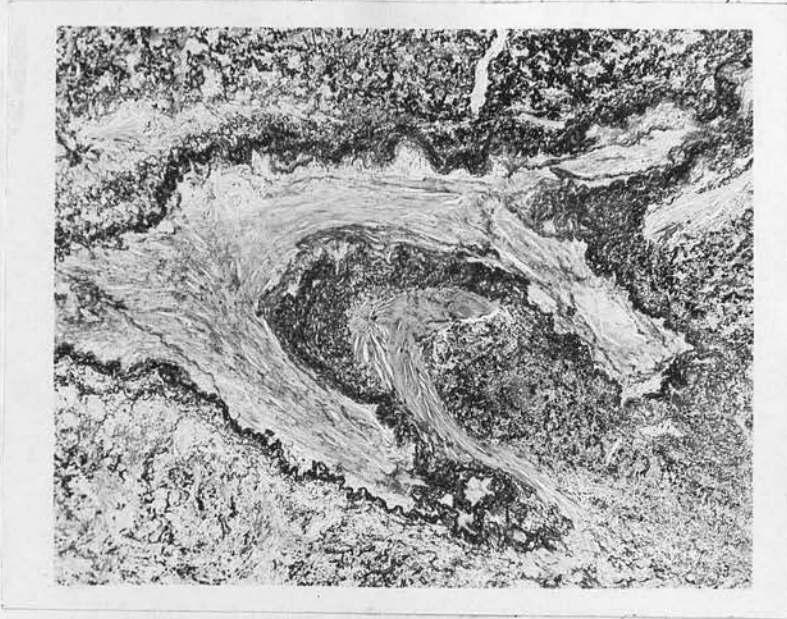


Figure 6.

Margin of an ischaemic cavity in progressive massive fibrosis from the lungs of a coalminer showing the ragged margin of the cavity as a result of disintegration of fibrous tissue. (H.E. x 50).



Fibrin
Jelina
together with an artery showing arteriosclerosis.
(H.E. 45x)

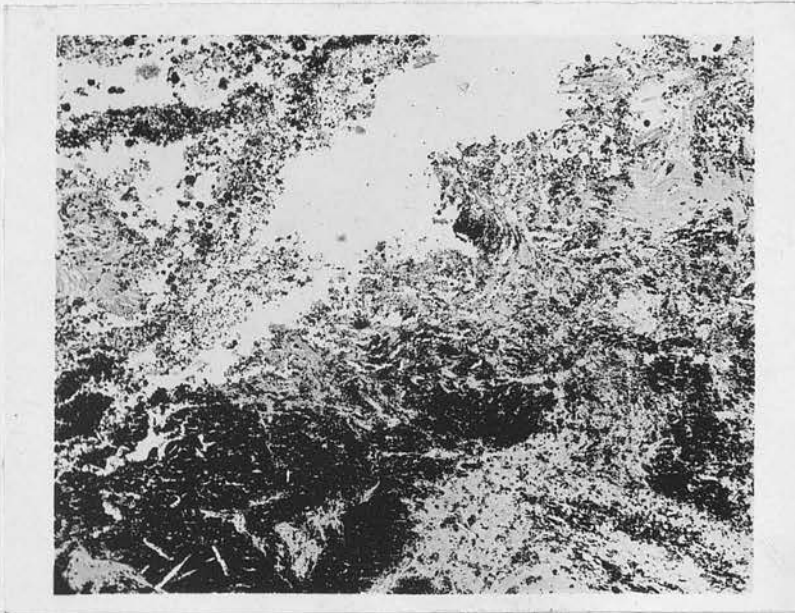
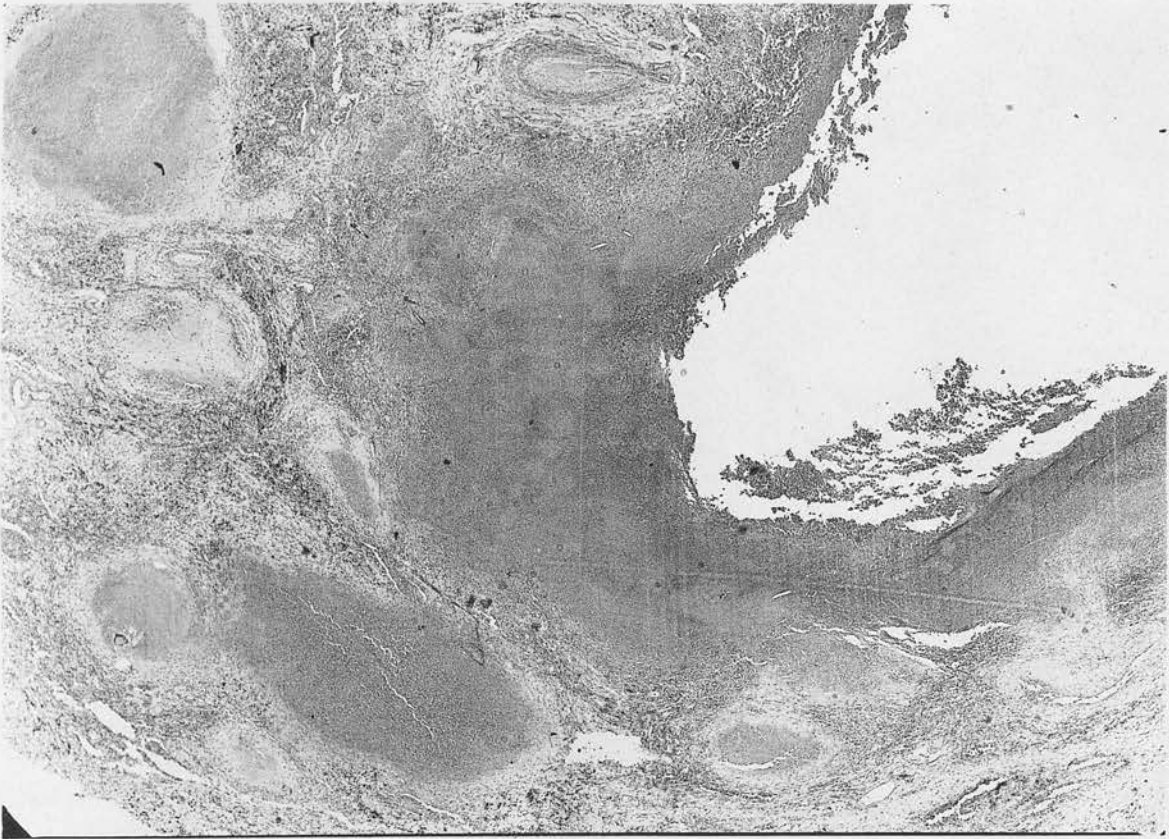


Figure 7.

Margin of a tuberculous cavity in progressive massive fibrosis from the lungs of a coalminer showing a well defined cavity margin. Caseous foci are also present, together with an artery showing obliterative endarteritis.

(H.E. x 25).



Micrograph showing a cross-section of biological tissue, possibly a histological section. The image displays a large, irregularly shaped structure with a dense, granular internal texture. On the left side, there are several smaller, rounded, and somewhat translucent structures. The overall appearance is that of a histological section, possibly showing cellular or sub-cellular components. The image is framed by a thin black border.

MATERIAL AND METHODS OF PRESENT STUDY.

The records of all patients admitted to the ward of the Pneumoconiosis Research Unit at Llandough Hospital between 1946 and 1952 were reviewed. A total of 669 coalworkers was admitted during that period and cases showing cavitation either in association with simple pneumoconiosis or progressive massive fibrosis were examined.

Routine medical and industrial histories were taken, and a clinical examination was carried out in each case. Postero-anterior radiographs were obtained in all cases and lateral films or tomograms in the majority. The sputa were examined for tubercle bacilli by direct smear examination, culture, and in many instances by guinea pig inoculation. The erythrocyte sedimentation rate was measured by the Westergren method.

RESULTS.

Pulmonary cavitation was discovered in 8 cases in association with simple pneumoconiosis and in 104 cases with typical progressive massive fibrosis. The total number of patients with progressive massive fibrosis was 389 giving a prevalence therefore of cavitated massive lesions in the ward patients of 26.7%.

A) Patients with pulmonary cavitation other than in massive fibrosis.

Details of these cases are summarised in Appendix I and examples are given in Appendix II. They are uncommon, assuming their greatest importance in differential diagnosis and they will be discussed again under that heading. Of these eight cases, four were examples of cavitation occurring in confluent tuberculous bronchopneumonia in men with simple pneumoconiosis (cases 105, 107, 111 and 112). All these patients died before anti-tuberculous chemotherapy was readily available. One patient had a chronic lung abscess (case 109) and another a cavitated bronchial carcinoma (case 106).

The remaining two (cases 108 and 110), showed cavitation in discrete rounded pulmonary lesions. Details of these patients are given in the appendices. Case 108 is an example of the so-called "rheumatoid syndrome" recently described by Caplan (1953), and Miall, Caplan, Cochrane, Kilpatrick and Oldham (1953). This man spent 21 years in the coal mines and has multiple bilateral discrete round pulmonary opacities, some of which are cavitated. The sputum is positive for tubercle bacilli, the man has classical rheumatoid arthritis and is still alive. Case 110 has the typical "rheumatoid type" of radiological abnormality, reminiscent of tuberculomata, but no evidence of rheumatoid arthritis. This case shows a fluid level in the cavity, whereas Moyes (1951) suggested that this does not happen in "true" tuberculomata.

This syndrome, so far only described in coal workers, is characterised by the association, in over 50% of cases, of rheumatoid arthritis and nodular fibrosis of the lung, radiologically similar to, but distinguishable from, the massive fibrosis occurring in complicated pneumoconiosis. These lesions characteristically develop upon a background of a lesser degree of simple pneumoconiosis than does

typical progressive massive fibrosis; whereas massive fibrosis rarely, if ever, starts where there is less than category 2 simple pneumoconiosis the "rheumatoid lesions" are often seen with a background of category 1; they develop more rapidly than massive fibrosis and often appear in crops. They may develop several years before, concurrently with or several years after the onset of rheumatoid arthritis. These lesions are quite unlike the interstitial pneumonitis occurring in cases of rheumatoid arthritis described by Ellman and Ball (1948).

Autopsy or biopsy material is lacking, the aetiology remains unknown but there is some evidence that (like progressive massive fibrosis) the fundamental cause is tuberculosis and these lesions may represent a particular type of tissue reaction to dust and tuberculosis in the lungs of miners who are predisposed to the development of rheumatoid arthritis.

No general conclusions can be drawn from these few cases, and the remainder of this section is concerned with the cavitation that occurs in typical progressive massive fibrosis.

B) Patients with cavitated progressive massive
fibrosis.

Cavitation in massive lesions may be recognised radiologically as a translucency in the large opacities, often with a fluid level, and clinically by the expectoration of large amounts of jet black sputum. This profuse black expectoration (sometimes erroneously called melanoptysis) is quite different from the black-flecked sputum which practically all coalworkers expectorate after completing a shift. The frequency and nature of this type of cavitation in massive fibrosis are not widely appreciated and extensive cavitation may be associated with few symptoms, having a prognosis which is much better than the radiological appearances at first suggest.

The 104 cases fell into two main groups:

(a) 26 patients with tubercle bacilli in the sputum during life, and (b) 78 patients whose sputum did not contain tubercle bacilli in life (although bacilli were cultured from the lungs of one of them at autopsy (case 79)). In group (a) tubercle bacilli were found on direct smear examination of fewer than

5 specimens of sputum in 20 instances, (the remainder being discovered either by further testing or by culture and animal inoculation). In group (b) only 18 cases had fewer than 5 specimens of sputum examined and the majority had many more, as a protracted search was made in patients whose initial specimens failed to reveal tubercle bacilli. These findings are summarised in Table (1). In the sputum positive group, the finding of bacilli and the discovery of cavitation occurred simultaneously in 22 instances.

Table 1 is on page 31 and an explanatory note on page 32.

Table 1.

Frequency of Sputum Examination of Ward Patients with Progressive Massive Fibrosis.

No. of specimens examined.	Numbers and percentages of patients examined by various methods.											
	Direct Smear.		Culture.		Guinea-pig inoculation.		Direct Smear.		Culture.		Guinea-pig inoculation.	
	Pos. group.	Neg. group.	Pos. group.	Neg. group.	Pos. group.	Neg. group.	Pos. group.	Neg. group.	Pos. group.	Neg. group.	Pos. group.	Neg. group.
1-	20 (77%)	18 (23%)	15 (58%)	11 (14%)	12 (46%)	25 (32%)						
5-	2 (8%)	23 (29%)	2 (8%)	31 (40%)	-	5 (6%)						
10-	-	9 (12%)	1 (4%)	10 (13%)	-	-						
15-	-	7 (9%)	-	7 (9%)	-	-						
20-	1 (4%)	7 (9%)	-	5 (6%)	-	-						
25-	-	1 (1%)	-	3 (4%)	-	-						
30+	-	9 (12%)	1 (4%)	9 (12%)	-	-						
Totals	23	74	19	76	12	30						
	Not examined on direct smear.		Not examined by culture.		Guinea-pig not inoculated.							
	3 (12%)	4 (5%)	7 (27%)	2 (3%)	14 (54%)	48 (62%)						
Grand Totals	26 (101%)	78 (100%)	26 (101%)	78 (101%)	26 (100%)	78 (100%)						

The different methods of examination were not mutually exclusive. Some patients did not have a direct smear examination because of inadequate amounts of sputum and such cases were examined by means of laryngeal swabs.

It has been observed that in some cases of progressive massive fibrosis acid-fast bacilli are seen in stained films of the sputum which are found to differ from tubercle bacilli in their cultural characteristics and which fail to produce tuberculosis on guinea pig inoculation: two such cases are included in the sputum negative group (cases 63, 73). Marks (1953) has noted that atypical acid-fast organisms occur more frequently in sputum specimens from cases of progressive massive fibrosis than from cases of pulmonary tuberculosis and he stresses the need for animal inoculation to prove the pathogenicity of organisms isolated on culture in cases of massive fibrosis. Neither of these two cases had had any anti-tuberculous chemotherapy prior to the finding of these organisms. (Both these cases have since died: the post-mortem findings were those of massive fibrosis without evidence of active tuber-

culosis). Similar organisms have been found in five cases of cavitated massive lesions not included in the present series, and none of these had had chemotherapy. It is possible that two of the cases included in the sputum positive group are further examples (cases 6, 21) as they were originally assigned to this group on the result of a direct smear examination. Guinea pig inoculation was not carried out at that time and subsequent specimens of sputum have failed to reveal further acid-fast bacilli by any method.

Clinical features.

Clinically, patients with cavitated massive lesions fall into two main groups.

- a). Sputum positive group (26 cases). In this group the radiological appearances are typical of progressive massive fibrosis and the sputum is positive for tubercle bacilli. Six patients were discovered to have cavitation and a positive sputum during the course of an acute respiratory infection, while in the other 20, tubercle bacilli were discovered in the course of routine investigation.
- b). Sputum negative group (78 cases). Cavitated

massive lesions were discovered in 43 members of this group during, or immediately following a non-tuberculous respiratory infection which usually resembled an acute bronchitis, but in five instances there were features suggestive of pneumonia. In the remaining 35 patients of this group, cavitation occurred without any associated illness. One of these, (case 91) presented clinically as a pulmonary abscess two months after cavitation was first observed. Cases of a somewhat similar nature have been described by Seltmann (1867), Cummins and Sladden (1930), Ornstein and Ulmer (1936), Badham and Taylor (1939) and Faulkner (1940).

Details of both groups.

Table (2) gives the age distribution of the patients at the time of discovery of cavitation in the two main groups.

Table 2.Age Distribution of Ward Patients at Time of
Discovery of Cavitation in Progressive Massive
Fibrosis.

Age group	Number of Patients		Totals
	Sputum positive group	Sputum negative group	
30-	1	5	6
35-	4	6	10
40-	5	22	27
45-	5	11	16
50-	6	19	25
55-	3	8	11
60-	2	5	7
65-	0	2	2
Totals	26	78	104

Patients may occasionally be asymptomatic, but like most cases of massive fibrosis they usually complain of dyspnoea and cough. Black sputum and haemoptysis are relatively common and table (3) shows the frequency of their occurrence. The figures

suggest no difference between the two main groups. In cases presenting with no history of black sputum but showing cavitated massive fibrosis radiologically it is reasonable to assume that the sputum has been swallowed. Severe haemoptysis is rare and only occurred in two of our patients (one sputum positive (case 25) and one sputum negative (case 44)).

Table 3.

Occurrence of Haemoptysis and Black Sputum in Ward Patients with Progressive Massive Fibrosis.

	Sputum positive group	Sputum negative group	Total
Haemoptysis alone	2 (8%)	8 (10%)	10 (10%)
Black sputum alone	13 (50%)	27 (35%)	40 (38%)
Both	11 (42%)	36 (46%)	47 (45%)
Neither	0	7 (9%)	7 (7%)
Totals	26 (100%)	78 (100%)	104 (100%)

Physical signs. Many different physical signs may be elicited in the chest but their recognition is of little importance. Lee (1948) suggested that crepitations were more common in sputum positive than in negative cases, but the figures in this series show no difference. Crepitations were noted in 64 instances, 17 (65%) being sputum positive cases and 47 (60%) sputum negative cases. Marked finger clubbing only occurred in one patient (case 91), although lesser degrees of clubbing were occasionally noted. One rare complication observed was a recurrent laryngeal nerve palsy (case 50). This presumably arose as a result of pressure by the masses on the nerve in its intra-thoracic course. The radiograph is shown in Appendix II.

Fever. 17 (65%) of the sputum positive and 44 (56%) of the sputum negative cases had fever at some time during their stay in hospital (99° F. or over on two or more occasions) and therefore the presence or absence of fever is of little value in differentiating between the two groups, although it has been suggested (Lee 1948) that patients with overt tuberculosis are more commonly febrile.

General condition. The patients were graded as being in good, fair or poor general condition and although this is a very arbitrary assessment an analysis of the two groups is given in Table (4). It will be seen that a higher percentage of the sputum positive patients were in poor general condition but that a good general condition did not exclude open tuberculosis.

Table 4.

"General condition" of Ward Patients with
Progressive Massive Fibrosis.

	Sputum positive group	Sputum negative group	Totals
Good	8 (31%)	33 (42%)	41 (39%)
Fair	4 (15%)	31 (40%)	35 (34%)
Poor	14 (54%)	14 (18%)	28 (27%)
Totals	26 (100%)	78 (100%)	104 (100%)

Weight change. An assessment of recent loss of weight was attempted, the patient's own estimate being taken to cover the period before admission to hospital. The only accurate measurements were in patients who had been in hospital for some time, or who had previously attended the outpatient department. Table (5) shows that only two patients in the whole series gained weight and that loss of weight is very common in both groups, although more so in those patients with a positive sputum.

Table 5.

Weight Change in Ward Patients with Progressive Massive Fibrosis.

	Sputum positive group	Sputum negative group	Totals
Gain	0	2 (3%)	2 (2%)
Static	7 (27%)	33 (42%)	40 (38%)
Slight loss	4 (15%)	24 (31%)	28 (27%)
Loss	14 (54%)	18 (23%)	32 (31%)
No Record	1 (4%)	1 (1%)	2 (2%)
Totals	26 (100%)	78 (100%)	104 (100%)

Erythrocyte sedimentation rate. The erythrocyte sedimentation rate was measured in 98 of the 104 cases. It has been suggested by Nadiras, Batique and Michot (1948) and Sander (1949) that a raised sedimentation rate in patients with pneumoconiosis and cavitation is indicative of overt tuberculosis. Taking 10 mm. per hour as normal, Table (6) shows that one sputum positive case in this series had a sedimentation rate below this, while 19 had an elevated figure, the range being 4 to 100 mm. per hour. In the sputum negative cases, 4 had a figure less than the norm, and 74 exceeded it. Thus, mere elevation of the sedimentation rate does not indicate overt tuberculosis nor does a low figure exclude it. Furthermore it should be remembered that acute respiratory infections, which may raise the sedimentation rate, are frequent occurrences in patients with massive fibrosis.

Table 6.Measurement of Erythrocyte Sedimentation Rate of
Ward Patients with Progressive Massive Fibrosis.

	Sputum positive group	Sputum negative group	Totals
E.S.R. < 10	1	4	5
E.S.R. > 10	19	74	93
No Record	6	-	6
Totals	26	78	104

Radiological examination: If a coalworker suddenly expectorates a large quantity of jet black sputum it may be assumed that cavitation has occurred in a massive lesion. Unless a radiograph is taken shortly after this episode, cavitation may not be detected because of the remarkable way in which cavities may refill completely (see case 36). The two main types of cavities in massive fibrosis cannot be differentiated radiologically. Cavitation can usually be diagnosed from a postero-anterior radiograph

especially if a fluid level is present, but a lateral projection is sometimes useful to distinguish cavitation from translucent areas caused by emphysematous bullae and to determine its anatomical site. Tomography may be helpful (Belayew, 1951; Roche and Morel, 1952; and Roche, Naudin and Tolot, 1949) and examples of its use are shown in cases 55 and 76.

In this series, cavitation was confirmed by lateral films or tomograms in 100 instances, while in the remaining 4, fluid levels made the cavitation obvious. Cavities may be single or multiple, unilateral or bilateral, and may occur in any part of the lung; they differ in size and have walls of variable thickness (cases 27 and 76). Bronchography was carried out in a small number of the patients in this series (see case 91), but the contrast medium has not yet been observed entering a cavity although Worth (1952) and Balgairies and Bonte (1953) have done so. It is probable that the bronchial communication opens intermittently, so that the quantity of sputum and the fluid level change from time to time.

An analysis was made of the cases in this series to determine the sites of cavitation in massive fibrosis (Tables 7, 8 and 9). A preponder-

ance of upper lobe cavitation was found in both groups (84% in the sputum positive and 90% in the sputum negative group) since massive lesions occur most commonly in these lobes. No cavities were found in the middle lobes, but a small number was found in the lower lobes particularly in the apical segments. It will be seen that a higher proportion of the sputum positive cases is unilateral and that they are situated more commonly in the right lung and in the upper lobe of that lung.

Ornstein and Ulmar (1936) found cavitation in the upper lobes of 54 of their 58 cases and when Vorwald (1941) compared 94 men with silicosis who had cavitation with 339 patients with uncomplicated tuberculosis he found that in both groups cavitation was commonest in the upper lobes. He agreed with Auerbach and Stemmerman (1944) that cavitation of the lower lobes occurs more commonly in tuberculo-silicosis than in ordinary pulmonary tuberculosis.

Table 7.

Detailed analysis of sites of cavitation in ward patients with progressive massive fibrosis.

Left Lung.

	U	UD	UDL	UL	D	DL	L	None	Total
U	6 28	2		1	1 2	1		9 10	17 43
UM									
UMD									
UMDL									
UD	8	5		1					14
UDL	1								1
UL				1				1	1 1
M									
MD									
MDL									
ML									
D								2 3	2 3
DL						1			1
L					1		2		1 2
None	4 11				3				4 14
Total	10 48	7		12	2 5	1 1	2	12 13	26 78

Right Lung.

Notes: Cavitation in massive fibrosis is shown in this table by lobar distribution in both lungs.

The figures in red denote the sputum positive cases and the black, sputum negative cases.

The contractions used for the lobes, alone and in combination are:- U = upper: M = middle: D = apical segment of lower (dorsal): L = lower.

Table 8.Anatomical sites of cavitation (summary).

Situation	Sputum positive group		Sputum negative group		Totals	
	No. of cases	Per cent.	No. of cases	Per cent.	No. of cases	Per cent.
Both lungs	10	(38)	51	(65)	61	(59)
Right lung only	12	(46)	13	(17)	25	(24)
Left lung only	4	(15)	14	(18)	18	(17)
Totals	26	(99)	78	(100)	104	(100)

Table 9.

Anatomical sites of cavitation (further analysis).

Situation		Sputum positive group		Sputum negative group		Total	
		No. of cases	Per cent.	No. of cases	Per cent.	No. of cases	Per cent.
In- clud- ing up- per lobes	Both lungs	7	(27)	46	(59)	53	(51)
	Rt. lung only	11	(42)	13	(17)	24	(23)
	Lt. lung only	4	(15)	11	(14)	15	(14)
Elsewhere (None in middle lobe)		4	(15)	8	(10)	12	(12)
Total:		26	(99)	78	(100)	104	(100)

Discussion of the "ward population".

A "ward population" is highly selected and conclusions drawn from such a sample may well be biased. Admission to hospital is likely to be sought by men who have the striking symptom of black sputum and several such cases were encouraged to come in for investigation. The proportion of sputum positive cases will be increased by the tendency to admit patients who are relatively ill or who have recently lost weight but it will be decreased by the exclusion from a general hospital ward of those known to have a positive sputum before admission. All these factors have influenced the sample. The effect of selection is illustrated by a comparison of the prevalence (see footnote) of cavitation in massive fibrosis in the "ward population" with that found in the Rhondda Fach Survey

The terms "attack rate" and "prevalence" are used in this, and subsequent sections to mean respectively, the number of new cases appearing in a given population in a given time, and the percentage of people with a given disease or stage of disease at a given time. The word "incidence" is avoided as it is generally used to cover both.

where 95% of the whole population of 6,026 miners and ex-miners was radiographed (Cochrane, Cox and Jarman, 1952). During this survey, 736 cases of massive fibrosis were discovered, but only 18 had cavitation, giving a prevalence of 2.4% compared with 26.7% in the ward population. Of the 18 men in the Rhondda Fach Survey, 3 (17%) had a laryngeal swab positive for tubercle bacilli and 15 (83%) had not. Thus the prevalence of cavitated massive fibrosis in the "ward population" was ten times as great as that found in a mining community. A summary of the two populations studied is given in Tables (10) and (11). There is thus, a great discrepancy between the prevalence of cavitation found in a single survey of a complete community and that observed in ward patients over a period of six years. Stewart, Davies, Dowsett, Morell and Pierce, (1948) in following up certified cases of pneumoconiosis for periods as long as 15 years found, on the basis of a history of black sputum, that 15% of cases with massive fibrosis probably had cavitation.

Table 10. Rhondda Fach Survey. (Cochrane et al., 1952).

Summary of cases with progressive massive fibrosis in relation to cavitation and positive sputum.

	Sputum positive		Sputum negative		Total	
	No. of cases	per cent.	No. of cases	per cent.	No. of cases	per cent.
Cavitation	3	(16.7)	15	(83.3)	18	(100)
No Cavitation	5	(0.7)	713	(99.3)	718	(100)
Total	8	(1.1)	728	(98.9)	736	(100)

Table 11. Ward 1946-52.

Summary of ward cases of progressive massive fibrosis in relation to cavitation and positive sputum.

	Sputum positive		Sputum negative		Total	
	No. of cases	per cent.	No. of cases	per cent.	No. of cases	per cent.
Cavitation	26	(25.0)	78	(75.0)	104	(100)
No Cavitation	4	(1.4)	281	(98.6)	285	(100)
Total	30	(7.7)	359	(92.3)	389	(100)

Other factors than selection for admission may account for these discrepancies. The in-patients had several radiographs taken over a period of years in contrast to the single film taken during the Rhondda Fach Survey. Cavitation may only appear in one or two of the serial radiographs, either because it occurs late in the period of observation or because a cavity may refill and

thus disappear radiologically. Further, the prevalence of cavitation will depend upon the proportions of early and late stages of massive fibrosis since cavitation usually occurs in radiological categories B - D, and these categories are more common in the ward population than in the communities from which they are drawn. While selection increases the prevalence of cavitation in a ward population, a single field survey under-estimates it, owing to refilling of cavities. To discover the proportion of cases of massive fibrosis who may be liable to undergo cavitation within a given period (the attack rate) it would be necessary to take radiographs of a sample of cases at short intervals over many years. It is unlikely that this would ever be done because in the absence of positive sputum, cavitation is a relatively unimportant clinical event. The importance of defining the population studied is further emphasised by the frequency with which a positive sputum is found in cases of massive fibrosis under different circumstances. In the Rhondda Fach Survey this was 1.1% (Cochrane et al., 1952) compared with 7.7% in the ward population (30 positive in 389 cases, see table 11) and 40% at autopsy (James, 1954).



DIFFERENTIAL DIAGNOSIS.

Cavitation of massive fibrosis which is known to have been present from previous chest radiographs, presents no problem of diagnosis other than the differentiation between sputum positive and sputum negative types of cavitation. Cavities occurring in the lungs of miners without evidence of simple pneumoconiosis can be presumed not to be due to massive fibrosis because this condition rarely, if ever, arises on a background of less than category 2 simple pneumoconiosis. If emphysema has obliterated the background of simple pneumoconiosis, as it may do, there are usually other areas of massive fibrosis to indicate the diagnosis. Pulmonary cavitation, other than that found in massive lesions, may occur coincidentally in cases of massive fibrosis and it may be impossible to distinguish this from cavitated massive fibrosis itself unless previous radiographs are available to show the pre-existence of massive fibrosis at the site of the cavity. Case 58 is an example of a cavitated massive lesion in one part of the lung and cavitation in, presumably, a pneumonic process in another. The discrete rounded

opacities of the "rheumatoid syndrome" may be readily differentiated from typical massive lesions and are rather similar to tuberculomata in non-miners. In cases without previous chest radiographs and with a negative sputum, it is necessary to consider other causes of cavitation, such as have been reviewed by Balchum and Zimmerman (1952). The diagnosis of a chronic lung abscess (case 109) or of cavitated unmodified pulmonary tuberculosis presents few difficulties (cases 105, 107, 111 and 112).

A cavitated bronchial carcinoma (Reisner, 1936; Strang and Simpson, 1953) is probably the most difficult to exclude (case 106). Bronchoscopy was not performed routinely and it is doubtful whether it is justifiable in typical cases of massive fibrosis. No carcinoma has been revealed in 31 of the cases of massive fibrosis coming to autopsy or in a further 49 cases who have been followed in life for more than two years. Massive fibrotic lesions, despite their size, practically never cause segmental lung collapse, probably on account of their relatively slow rate of development. Consequently, any collapsed segment seen radiologically should suggest malignancy. According to Shanks and Kerley (1950), sequestrum formation in a pulmonary cavity (other than in pneumoconiosis)

is characteristic of aspergillosis, but in two of the cases (cases 55 and 73) there were sequestra in cavities with no evidence of fungal infection. It is surprising that secondary fungal infection did not occur in some cases especially as many of them had prolonged treatment by one or other of the modern chemotherapeutic agents (Abbott, Fernando, Gurling and Meade, 1952) for the acute respiratory infections to which they are prone. Heppleston and Gloyne (1949) described two coalworkers in whom aspergillosis was discovered at autopsy but no mention of chemotherapy is made and they stress the rarity of this condition.

PROGNOSIS.

It is not possible to draw any conclusions as to the prognosis of patients with cavitation other than in massive fibrosis, because the number of such cases is too small. It has been suggested by Farrell, Sokoloff and Charr, (1940) that the prognosis for patients with cavitated massive lesions and a sputum negative for tubercle bacilli is relatively good, while for those with a positive sputum it is bad. Dayman, (1945), Lee, (1948), and Theodos and Gordon, (1951 and 1952) give a life expectation in sputum positive cases of between 2 and 3 years. Turner and Martin, (1949), found that the survival rate of men with silico-tuberculosis in Sheffield compared unfavourably with the rate for men with pure silicosis, but the authors do not state the grounds on which they distinguish these two conditions.

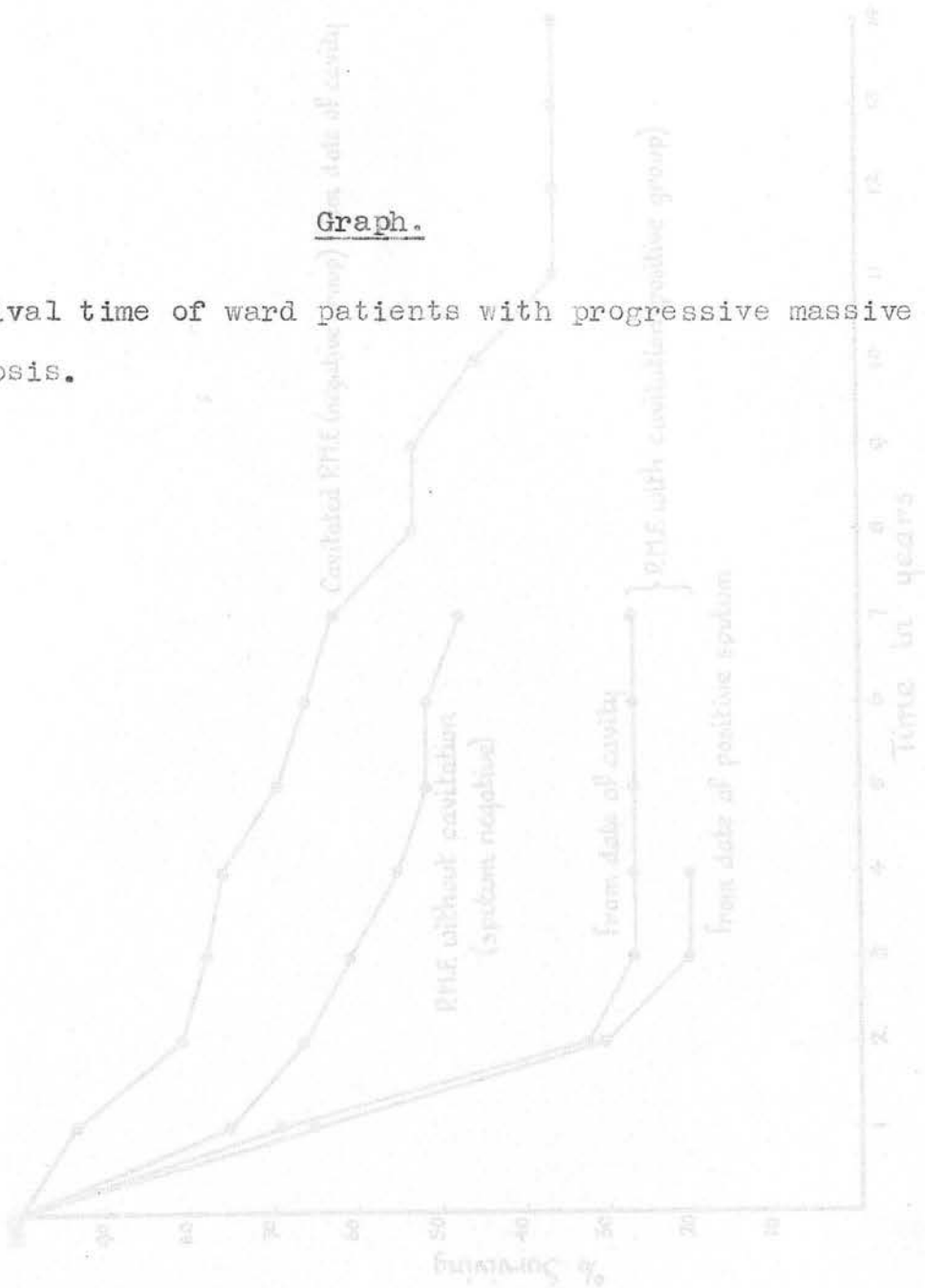
In this series only 3 of the 26 cases survived two or more years after the discovery of a positive sputum (cases 6, 15 and 21). Only 6 of the sputum positive group survived two or more years after detection of the cavity whereas 41 of the 78 sputum negative cases survived longer than this after cavitation was detected,

and the difference in survival in the two groups is presented in the graph (page 58). Although the records of this series only date from 1946, radiographs taken previously are available in many instances thereby providing a longer period of review. Patients with long standing massive fibrosis and a persistently negative sputum usually die of respiratory or cardiac failure, not of terminal tuberculous disease. It is possible that the more extensive use of modern anti-tuberculous drugs will improve the prognosis for patients with cavitated massive fibrosis and a positive sputum. An attempt was made to compare the survival times of patients with sputum negative massive fibrosis with and without cavitation. The 78 patients with cavitation in the sputum negative group were paired with 78 other ward patients of comparable age and radiological category who had non-cavitated massive fibrosis and a negative sputum. The survival time in each instance was taken from the date on which the patient was first seen in the Pneumoconiosis Research Unit until the date of death, or until the 31st December 1952. It will be seen from the graph that in sputum negative cases patients with cavitation have a

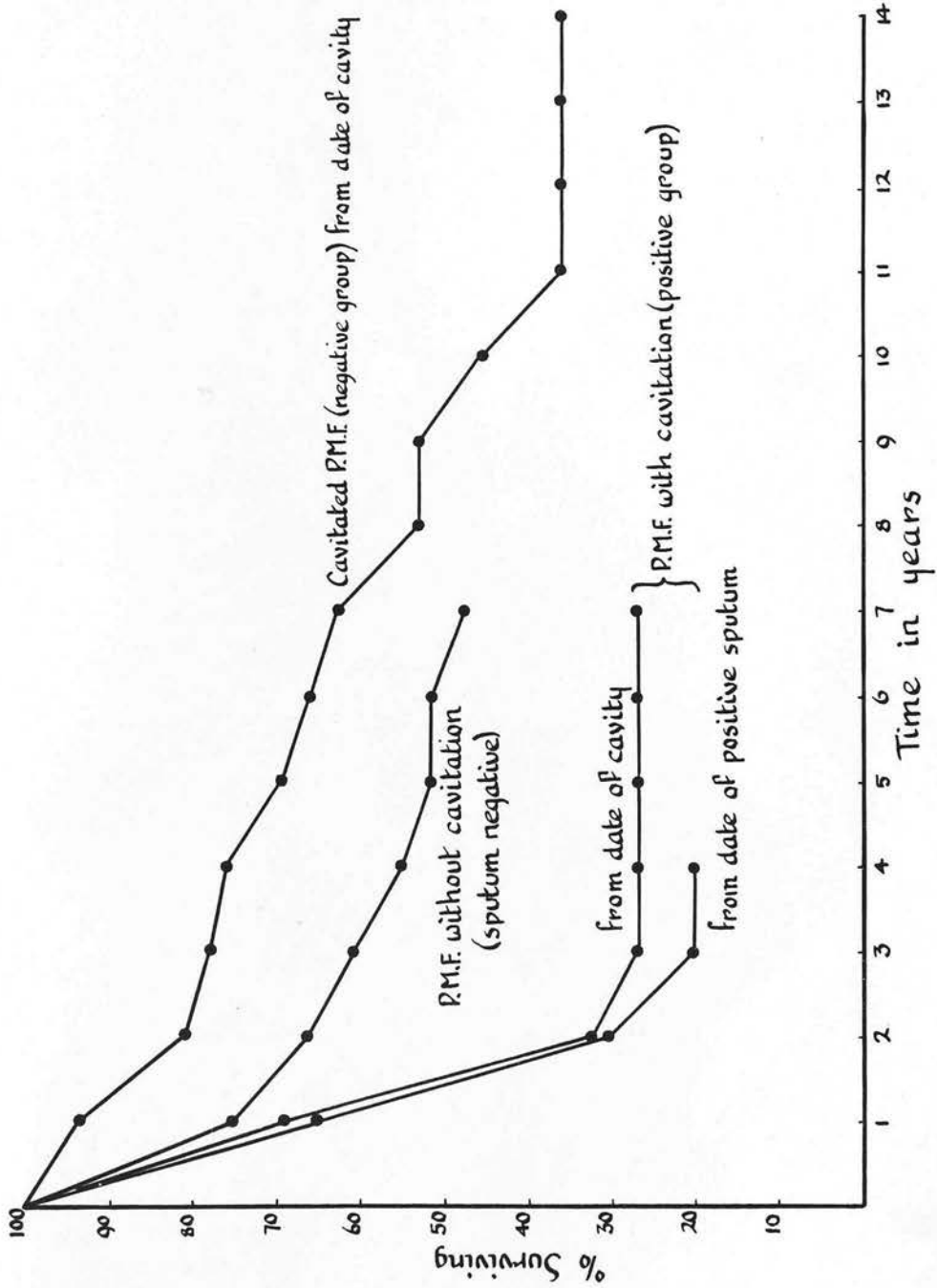
slightly better expectation of life than those with non-cavitated lesions. The difference is not great and may probably be accounted for by the way in which cases are selected for hospital admission. Many of the patients with cavitated disease were admitted for investigation although they were in their usual health while patients with non-cavitated massive fibrosis were usually admitted because they were unwell.

Graph.

Survival time of ward patients with progressive massive fibrosis.



Survival Time of Ward Patients



MANAGEMENT AND TREATMENT.A. Patients with Cavitation other than
in Massive Fibrosis.

The general management and treatment of these patients does not differ essentially from non-miners with cavitated pulmonary disease. If thoracic surgery is indicated, because of, for example, a bronchial carcinoma, the presence of simple pneumoconiosis should not be a deterrent if the patient's respiratory reserve is satisfactory. Cases of unmodified pulmonary tuberculosis in patients with simple pneumoconiosis respond to modern anti-tuberculous chemotherapy in the same way as patients with no exposure to dust (Cox, 1953).

Patients with the "rheumatoid syndrome" should have the usual methods of therapy for rheumatoid arthritis including heat, wax baths and movement of joints under the supervision of a physiotherapist. If the sputum is positive for tubercle bacilli a course of anti-tuberculous chemotherapy should be given, but it is as yet uncertain whether control of the tuberculosis affects the joint manifestations in any way. If, as is usually the case in such patients, the sputum is negative for tubercle bacilli, cortisone may be given if otherwise indicated. It has in fact been given to

a number of cases not included in this series. Recent experimental animal work by Harrison, King, Dale and Sichel (1952) has shown that some modification in the development of silicotic lesions took place when cortisone was administered; the rate of development of the discrete silicotic nodules was retarded and there was some evidence that a direct inhibition of the fibrosis took place. Cortisone has not as yet, been shown to exert any favourable action on the pulmonary fibrosis in man, either in the rheumatoid syndrome or in typical massive fibrosis, and it may be that its use is best avoided in patients with these lesions whose aetiology is probably basically tuberculous in view of the animal experiments of Hart and Rees (1950). It may however be shown to have a place in combination with anti-tuberculous chemotherapy.

B. Patients with Cavitated Progressive
Massive Fibrosis.

1) Sputum positive cases.

It was not permitted to keep patients with cavitated massive fibrosis and a positive sputum in the ward; personal experience of their management is, therefore, very limited. Few results of the treatment of this condition have been published,

but in cases of silicosis with a positive sputum, anti-tuberculous drugs have been found to confer some symptomatic benefit although they have not markedly improved the survival period. Boselli and Lusardi (1950) treated 23 patients with silicosis and open pulmonary tuberculosis by means of streptomycin and para-amino salicylic acid for 3 or 4 months. Their results were not impressive - 2 cases without definite cavitation were rendered sputum negative; 6 with cavitation had transitory clinical benefit but the sputum remained positive; 16 other patients less well defined were "generally improved". Stojadinovic and Stojadinovic (1952) who are in agreement with these findings consider that neither streptomycin and para-amino salicylic acid nor pulmonary collapse measures are of value. These authors gave streptomycin to cases with cavitation whether tubercle bacilli were present or not. Maier and Hurst (1946) described one case with unilateral cavitated silico-tuberculosis with a positive sputum who was rendered sputum negative following an extra-pleural pneumothorax. Van Mechelen (1951) comments that streptomycin and para-amino salicylic acid are of little use in these cases and considers that collapse measures are unlikely to be successful in view of the

physio-pathological considerations. Theodos and Gordon (1951 and 1952) attempted an artificial pneumothorax in 3 patients without success but report one successful thoracoplasty. Auerbach and Stemmerman (1944) attempted 8 artificial pneumothoraces and 4 thoracoplasties but all were unsuccessful and Benson (1941) believed that collapse therapy in these cases brought about an earlier demise. Cohen and Glinsky (1953) treated 18 cases with streptomycin but only 3 were symptomatically improved.

In one case in the present series (case 9) a fall in temperature, gain in weight and a considerable reduction in the profusion of tubercle bacilli in the sputum resulted when a full course of streptomycin and para-amino salicylic acid was given although the patient died 2 years and 2 months after the initial finding of the positive sputum. Nevertheless adequate bed rest and full anti-tuberculous chemotherapy should be given, if only for the symptomatic benefit that may be conferred. The importance of combined chemotherapy must be stressed in order to prevent the emergence of drug resistant strains of organisms (Daniels and Bradford Hill, 1952) as in

many of these patients the sputum remains positive for tubercle bacilli.

Cox (1953) who has experience in South Wales of many cases of cavitated massive fibrosis with a positive sputum is in general agreement with the findings referred to above, but it is hoped that with more intensive modern anti-tuberculous treatment the survival time of these patients may be prolonged.

2) Sputum negative cases.

There is no specific therapy, the dust cannot be removed from the lungs nor can the associated pulmonary fibrosis be resolved, but patients with the sputum negative type of cavitation may remain fairly well for many years despite gross structural lung damage, and are frequently able to undertake gainful employment. Miall, Oldham and Cochrane (1954) have investigated the effect of iso-nicotinic acid hydrazide on early massive fibrosis on the basis of the tuberculosis hypothesis of massive fibrosis. Sputum negative cases only were included in this controlled field trial, but no evidence of a beneficial effect of the drug on such cases was discovered. It is very important to explain to men with massive fibrosis that the black sputum and blood which they expectorate are not evidence of

overt tuberculosis for they are naturally apprehensive and much harm may be done if the relatively benign nature of cavitated massive fibrosis in the absence of a positive sputum is not appreciated (see case 76). An understanding of the condition both by the medical attendant and the patient is essential. When a diagnosis of pneumoconiosis is made, reassurance must be given that although dust disease is present an early death is unlikely. There is no object in promoting invalidism, bed rest has little place in the management of these patients and on the contrary, men should be shown that ordinary activity is possible and even beneficial. Eventually of course a time comes when these patients are too disabled to work, thus producing social and medical problems of some magnitude in many parts of the country. This is a reflection of the fact that the majority of men living at the present time were exposed to considerable concentrations of dust in the mines some years ago when dust suppression was not as efficient as it is today.

The symptomatic treatment of men with sputum negative cavitated massive lesions is discussed by considering the cardinal symptoms of

dyspnoea, cough, expectoration and chest pain in relation to the underlying physiological and pathological changes. Of all symptoms, breathlessness on exertion is the most important, is the one that causes most disability in patients with massive fibrosis and usually is the first symptom for which medical advice is sought.

Dyspnoea.

Men with massive fibrosis are practically always short of breath when compared with their fellow-men of comparable age and build (Cochrane et al., 1951). Dyspnoea in massive fibrosis is chiefly caused by a deficient "bellows action" of the lungs and is a mechanical effect due to the increased resistance of the lungs to movement. Only to a lesser extent does impaired gas transfer play a part although in advanced cases there is some arterial desaturation and carbon dioxide retention (Baldwin, Cournand and Richards, 1949; Gilson and Hugh-Jones, 1954). Reassurance and suitable re-employment are of primary importance because much of the dyspnoea is caused by irreversible lung damage. Breathing exercises are of considerable value, at least psychologically and if obesity is present as it is in a few cases, weight reduction must be encouraged in order to reduce the amount

of work done on exercise. Some special methods of therapy have been tried such as a pneumoperitoneum or an abdominal binder, but without conclusive evidence of benefit. This method of treatment was first introduced by Reich (1924) for patients with emphysema and its effect is to raise the intra-abdominal pressure sufficiently to cause elevation of the diaphragm during expiration and yet not to impede its contraction during inspiration. Better diaphragmatic excursion is obtained, the amount of residual air is reduced and the cough mechanism improved.

Bronchial spasm.

Bronchial spasm is common and may aggravate the dyspnoea. It may come on in attacks like those of asthma but it is often more resistant to therapy. It is usually most marked in the mornings and on change of environmental temperature or humidity, consequently, some general advice should be given in an attempt to prevent attacks. The mere act of going out of a heated room to the lavatory or upstairs to bed may be sufficient to precipitate an attack, and the relative freedom from bronchial spasm which patients experience while in the even temperature of a hospital ward is frequently noted. It is sometimes worse after a meal and a rest period

at this time may be advisable. Usually, but not invariably, bronchial spasm is associated with an exacerbation of a chronic respiratory infection, and treatment of the infection is in itself often sufficient.

Experience has shown that the adrenergic group of drugs is the most useful for relieving the bronchial spasm in patients with massive fibrosis. One of the simplest and most effective methods of treatment is the inhalation of a spray of adrenaline solution from a suitable nebuliser. A handspray should be provided that can be carried in the pocket, to be employed at the earliest sign of an attack, using the compound adrenaline and atropine spray (B.P.C.) which is the cheapest and most effective substance. If more continuous administration of the drug is required in severe cases of bronchial spasm, or if the patient cannot use his hands because of rheumatism or general debility, the spray may be attached to a cylinder of oxygen or compressed air. One of the preparations of isopropyl-nor-adrenaline (isoprenaline sulphate or 'Neo-Epinine') in doses of 10-20 mgm. sublingually three times a day (Robertson, 1949), may be given, if necessary in addition to the use of the handspray, and although side effects are

rare, palpitations occasionally occur. Many patients are symptomatically benefited from broncho-dilator drugs even when spasm is not clinically apparent.

In a more severe attack, adrenaline may be given by subcutaneous or intramuscular injection. The patients can be instructed to inject this in the same way as diabetics give themselves insulin, and the mere fact of having a potent therapeutic weapon that they can use early in an attack gives confidence. Dunlop, Henderson and Inch (1952) have pointed out that self-administration of adrenaline is not taught as frequently as it should be. Other drugs that may be tried are ephedrine, aminophylline (orally or intravenously) or khellin (Kennedy and Stock, 1952). Cortisone may occasionally be useful, although Kennedy (1954) has not found it to be so.

Oxygen. The provision of a cylinder of oxygen and a suitable mask is much appreciated by many of the more disabled men. A few minutes breathing oxygen at 4 litres/minute before and after any exertion such as undressing or bathing is valuable, and a cylinder by the bedside may save much nocturnal distress. Oxygen should not be given for prolonged periods of time particularly in the presence of a respiratory

infection, as any hypoxia and carbon dioxide retention already present become more marked with the onset of such an infection. Here, the predominant stimulus to respiration appears to be the low oxygen tension rather than the high carbon dioxide tension, and in such cases therefore, oxygen therapy may, by improving the oxygen saturation of the arterial blood, depress ventilation with further retention of carbon dioxide. For this reason oxygen therapy should be intermittent and a close watch kept for carbon dioxide narcosis as stressed by Donald (1949 and 1953).

Cough.

Cough in pneumoconiosis is probably caused by chronic irritation, usually as a result of pulmonary infection, is most troublesome on waking and is often sufficiently severe to cause vomiting. If it is productive, no depressant drug should be given during the day but if sleep is being lost, syrup of codeine phosphate should be prescribed at night. If smoking aggravates the cough it should be discouraged. As a result of coughing, herniae are common but with modern anaesthesia, surgical repair can be effected more often than formerly.

Expectoration.

The importance of reassuring men about black sputum and haemoptysis has already been discussed. There is no known way of stopping either type of expectoration and in the case of black expectoration many men say that they feel better afterwards. For this reason, postural coughing has a place in therapy and considerable relief may be obtained by its employment, particularly first thing in the morning, pre-prandially and before retiring. Mucoid sputum is usually tenacious and difficulty in expectoration is experienced especially in the mornings. The indiscriminate use of elaborate expectorant mixtures is of little avail (Dunlop et al., 1952) but the early morning cup of tea or glass of hot water to which may be added the simple Mist. Sod. Chlorid. Co. (N.F.) is beneficial. A superadded respiratory infection renders the sputum purulent, but frankly malodorous sputum is rare in patients with massive fibrosis and its presence should suggest some other underlying disease such as bronchiectasis or lung abscess.

Pain in the chest.

Men with massive fibrosis may complain of chest pain of varying severity. Much of it almost certainly originates in the thoracic musculature as a result of

coughing, but true pleuritic pain may also occur and occasionally, severe pain may be caused by a spontaneous pneumothorax. The possibility that the pain is functional in nature should be remembered, and this is particularly so in young men with pneumoconiosis. The muscular pain can usually be relieved by the local application of heat.

Complications.

The two main complications in men with massive fibrosis are respiratory infections and cardiac failure. These may be serious and unless steps are taken to prevent them or to treat them at their onset they may produce further disability or hasten death.

Pulmonary infection.

Chronic pulmonary infection is common and during the winter, acute exacerbations may result in both loss of working time and increased lung damage. It is essential therefore that immediate treatment with one of the modern chemotherapeutic agents should be given during these acute episodes. During such an attack bed rest is necessary but should not be prolonged. Experience has shown that while the sulphonamides may be of value, the

most effective substance is systemic penicillin in large doses, about 2 mega units of combined procaine and crystalline penicillin daily being required. Other drugs such as streptomycin 1 - 2 Gm. daily by injection, aureomycin and oxytetracycline (terramycin) 2.0 Gm. daily by mouth may occasionally be useful. Mulder, Goslings, van der Plas and Cardoso (1952), have recommended the use of penicillin combined with streptomycin, for acute bronchitis and, while this is effective, the sputum of all patients with massive fibrosis should first be examined for tubercle bacilli lest the drug conceal their presence. The dangers of blood dyscrasias from chloramphenicol are now realised (Franklin and Garrod, 1953) and this drug is probably best avoided in these men who may require repeated courses of chemotherapy. It would be very desirable if some preparation could be given prophylactically during the winter months, but so far no suitable substance has been found. The use of small doses of one of the sulphonamide drugs given continuously throughout the winter has been tried, but without success (Kilpatrick and Oldham, 1954). Penicillin by aerosol administration has been tried, but this method of therapy may aggravate bronchial spasm and is best avoided. Some consideration must be given to upper

respiratory tract infections as they are prone to extend to the lungs, and any sinus infection should be treated.

Cardiac failure.

Cor pulmonale frequently supervenes in the later stages of the disease as the massive lesions act as a vascular obstruction in the lungs as a result of fibrosis and partial vascular occlusion (Thomas, 1948 and 1951; Wells, 1954). This obstruction produces pulmonary hypertension (Bloomfield, Lauson, Cournand, Breed and Richards, 1946) which is aggravated by anoxia in the presence of an acute respiratory infection. The prevention of cardiac failure is more important than its therapy, especially by the adequate treatment of acute respiratory infections as stressed by Fulton (1953). Should it occur however, the patient should have an initial period of bed rest, a salt poor diet, digitalis should be given in full doses, and a mercurial diuretic such as mersalyl is often required. A few patients do not respond well to digitalis while some are resistant to mersalyl; such patients require more prolonged bed rest together with oxygen therapy and are often helped by potentiating an injection of mersalyl by aminophylline either intravenously or by rectal suppository.

Re-employment.

While many men with massive fibrosis (even those with cavitation and a negative sputum) can remain in the mining industry under present regulations, many more must find alternative employment. The provision of suitable work for men partially disabled by pneumoconiosis is one of the most important aspects of treatment, for nothing else can prevent the mental and physical deterioration that so often accompany enforced idleness. As the disability in pneumoconiosis is chiefly due to shortness of breath on exertion, work entailing heavy manual labour is unsuitable, but otherwise these men are capable of a wide variety of occupations. In past years, especially in South Wales, there was considerable difficulty in finding alternative work in the mining areas but conditions have recently improved, although it should be remembered that most men with massive fibrosis are between the ages of 40 and 60 years when it is less easy to train them for an entirely new occupation. They can however apply to the Disablement Resettlement Officer at the local Employment Exchange for registration as disabled persons (Disabled Persons (Employment) Act (1944)) when they are assessed as to their fitness for employment. Places are often

found for such men in a variety of industries, as employers must take a percentage of disabled persons. Some go to the Remploi factories and others are referred to a Ministry of Labour Industrial Rehabilitation Centre for aptitude assessment and training.

No precise rules can be laid down regarding re-employment as all degrees of disability are found amongst men with massive fibrosis but some limitations obviously apply to the more disabled. The journey to and from work must be carefully considered as many men find this the most tiring part of their day, but once at their place of employment they can usually work satisfactorily. Men subject to attacks of bronchial spasm should avoid industrial processes which produce irritating dust or fumes and those subject to "bronchitis" should probably not be employed in teams where their absence would interfere with a productive process. Outside work is generally unsuitable as some exposure to bad weather is inevitable.

DISCUSSION.

Cavitation in the lungs of coalworkers other than in progressive massive fibrosis has been described and details are given in appendices I and II. As the number of such cases is small it is not proposed to discuss them further, and the remainder of this section deals with the cavitation which occurs in massive fibrosis.

The nature and pathogenesis of coalworkers' pneumoconiosis must first be considered before the cavitation that occurs in the massive fibrotic form of the disease can be discussed.

The Pathogenesis of Coalworkers' Pneumoconiosis and Cavitation in Progressive Massive Fibrosis.

The Two Disease Hypothesis.

Hart and Aslett (1942) considered that what is now known as simple pneumoconiosis was part of the same process that is now called progressive massive fibrosis or complicated pneumoconiosis. Belt and Ferris (1942) in the same

report suggested that an additional factor was necessary for the collagenous fibrosis which they called "silicotic fibrosis."

Evidence in favour of the two disease hypothesis.

It is considered that simple pneumoconiosis is a pure coal dust effect. The main factor in determining the occurrence of coalworkers' pneumoconiosis is exposure to adequate concentrations of airborne coal dust and there is no evidence that coal dusts vary in their pathogenicity (Heppleston, 1951). While experimental animals readily get silicosis, no animal exposed to coal dust has yet shown the same reaction to the dust as is seen in man and this may be used as an argument against silica being the cause of fibrosis in coalworkers' pneumoconiosis in man. Simple pneumoconiosis is also found in the lungs of coaltrimmers in the docks (Gough, 1940) where there is exposure to coal dust only, and not to rock. Classical silicosis is now very rare in Great Britain and is only found in men exposed to dust of high silica content, such as in the gold mines on the Witwatersrand.

Pathological evidence.

a) Simple pneumoconiosis and massive fibrosis are totally

different in appearance, both macroscopically and microscopically.

b) Simple pneumoconiosis is diffusely distributed throughout the lungs whereas massive fibrosis is as a rule localised.

An alternative explanation is possible as far as the localisation of massive lesions is concerned. Massive fibrosis might be caused by the coalescence of the foci of simple pneumoconiosis, but the histological appearances do not support this. Many coalminers are exposed at some time during their life to a certain amount of rock dust and there could be a preferential deposition of silica in certain parts of the lung, and, as in classical silicosis, cause collagenous necrosis whereas the less pathogenic coal dust could be more diffusely distributed. It is difficult, however, to see how this could happen. The most usual site of the development of massive fibrosis is also the site in the lung where simple pneumoconiosis is most apparent radiologically and where the concentration of dust is most marked pathologically. This might suggest that a certain critical concentration of dust could initiate collagenous necrosis which, when once started, would attract dust to itself producing a vicious circle. Against this, is the evidence of Davies et al., (1949) that the attack rate

and progression rate of massive fibrosis is uninfluenced by dust exposure.

Radiological evidence.

- a) Simple pneumoconiosis only progresses radiologically under conditions of continued dust exposure, (Davies et al., 1951; Cochrane et al., 1951).
- b) The attack rate and progression rate of massive fibrosis is uninfluenced by dust exposure. (Davies et al., 1951).

On consideration of the evidence given above it can be concluded that some agent other than dust (or any component of the dust) is required to initiate progressive massive fibrosis.

The Tuberculosis Hypothesis of Progressive Massive Fibrosis.

It has frequently been suggested that progressive massive fibrosis is a modified form of tuberculosis (Belt and Ferris, 1942; Gough, 1947) and the evidence in favour of this must be examined.

Clapier, as long ago as 1763 apparently believed that coal dust in some way modified pulmonary tuberculosis; he advised his phthisical patients in Alais to spend some time living underground in a coal mine claiming that they would obtain considerable benefit from this method of treatment.

If the cause of progressive massive fibrosis is tuberculosis, it may be supposed that either:-

a) the tubercle bacilli were inhaled before, or with the dust, or alternatively, that a tuberculous focus was already present in the lungs, and that the dust laden phagocytes, together with the tubercle bacilli form a nucleus of massive fibrosis.

or

b) that the process is originally non-tuberculous (by a mechanism as yet unknown) and that a superimposed tuberculous infection occurs, either endogenously from a latent lung focus, or exogenously.

1. Clinical evidence.

There is little clinical evidence to support the tuberculosis hypothesis of progressive massive fibrosis. Patients rarely have toxæmia and the physical signs differ little from those of chronic bronchitis.

The Mantoux test: The evidence here is inconclusive, but so far, no case of progressive massive fibrosis in this country has been found with a negative Mantoux test, whereas men with simple pneumoconiosis may be Mantoux negative. As, however, the cases of progressive massive fibrosis examined are nearly always in the higher age groups this is not surprising.

The erythrocyte sedimentation rate: Stewart et al., (1948) found that the erythrocyte sedimentation rate was usually elevated during the phase of active progression of massive fibrosis. This is in keeping with an infective process but the frequent respiratory infections to which these patients are prone may in themselves produce an elevated sedimentation rate.

2) Pathological evidence.

Belt and Ferris (1942) wrote (p. 212), "Pulmonary tuberculosis as seen in the collier's lung shows several modifications not seen in ordinary phthisis. The reaction tends to be more fibrous, more chronic-looking and more widespread. At the same time, it is less cellular, less caseous and seldom shows giant cell systems. There is a curious tendency to develop widespread fibro-caseous nodules, apparently localising in foci of dust-reticulation, or large,

sharply defined dense confluent areas of fibro-caseous reaction not commonly encountered in ordinary phthisis.... So prominent is the dust content, and so inconspicuous are the conventional histological features of tuberculosis, that it is not surprising that these lesions are sometimes mistaken for a pure dust effect and their tuberculous element overlooked." Later, (p. 219), "...we came to wonder whether tuberculous infection might be playing an unsuspected rôle, acting under cover of the dust, so to speak, perhaps in a greater proportion of cases than have hitherto been identified clinically."

It seems possible that there is a continuous spectrum of histological appearances from overt tuberculosis, slightly modified by the presence of the accompanying dust, to extreme cases of progressive massive fibrosis with no characteristic appearances of tuberculosis. Details of the histological findings in massive fibrosis have already been given (page 18).

The most thorough pathological investigation of the tuberculosis hypothesis is that of James (1954). In a series of 1,000 South Wales coalworkers who came to autopsy in the Department of Pathology of the Welsh National School of Medicine between 1947 and 1950, James found massive lesions in 454 men and from 40% of these

cases tubercle bacilli were isolated by culture, or guinea pig inoculation. In the remaining 60%, the massive lesions showed no histological or bacteriological evidence of tuberculosis but such cases may well represent healed lesions, since the general pathological features of all massive lesions are similar irrespective of the presence or absence of tubercle bacilli. This view is supported by James's observation that tubercle bacilli were present in 88% of massive lesions from coalworkers under 40 years of age but in only 29% of massive lesions occurring in coalworkers of 60 years of age or over. These findings should be interpreted with some caution as some "selection" must occur in post-mortem material, particularly when the question of compensation arises, and the number of men in James's series who had a positive sputum in life is unknown.

The pathological features of massive fibrosis in coalworkers suggest that the infective process has been retarded in its rate of progress, and Mann's (1951) radiological findings accord with this view (page 86). A tuberculous infection could readily account for the obliterative arterial changes seen in massive fibrosis, since comparable vascular effects are caused by fibro-caseous tuberculosis unassociated with industrial dust exposure

(see page 18) and it seems probable that the vascular lesions are the result, and not the cause of the fibrosis.

It appears that massive fibrosis in coalworkers may progress either because active infection continues, or because partial vascular stenosis, left by an infection which has been overcome, leads to a replacement fibrosis. Vascular occlusion is also regarded as a factor in the progression of silicotic massive fibrosis (Policard, Croizier and Martin, 1939). Complete obliteration of larger arteries is probably responsible for the patches of colliquative necrosis frequently seen in massive lesions.

It could also be argued that some process other than tuberculous infection initiated the massive fibrosis and that subsequently there occurred a re-infection rather than a recrudescence of tuberculosis. This is difficult, however to reconcile with the pathological findings of caseous areas within the ischaemic fibrous masses. The experimental evidence is against a non-tuberculous respiratory infection being the initiating factor in silicotic massive fibrosis (Gardner, 1937 and 1938; Vorwald, Delahant and Dworski, 1940), and several cases have been observed in the ward who have had repeated respiratory infections with radiological opacit-

ies suggestive of pneumonia, who have not subsequently developed massive lesions in the affected areas. Similar conclusions were arrived at by Nadiras, Michot, Delesvaux, Batique and Pennel (1950) from a study of 34 cases of acute respiratory infections, other than tuberculous, in men with silicosis.

3) Bacteriological evidence.

The frequency with which the sputum in cases of progressive massive fibrosis is positive for tubercle bacilli has been discussed on page 51. It is possible that the very fibrous nature of the lesions in massive fibrosis imprisons tubercle bacilli during life, and makes their isolation at autopsy difficult. This imprisonment of bacilli is further suggested by the infrequency of haematogenous dissemination of the disease, but Jones (1938) cites one case of a miner with silicosis and pulmonary tuberculosis who died as a result of miliary dissemination of tuberculosis.

On the other hand, with the advent of cavitation it might be expected that all cases would give a positive finding, but the cases studied in this investigation show that this is not so as only 25% of cavitated massive lesions had a sputum positive for tubercle bacilli. These

findings might suggest that massive fibrosis may be non-tuberculous initially and subsequently invaded by the tubercle bacilli to produce a rapidly fatal infection.

4). Radiological evidence.

There is radiological evidence that coal dust may modify pulmonary tuberculous lesions. Mann (1951) showed that radiographic shadows of tuberculosis could usually be distinguished from those of simple pneumoconiosis and progressive massive fibrosis and that the shadows of tuberculosis in miners with pneumoconiosis were less progressive than in non-miners. Furthermore, he demonstrated that the prevalence of tuberculous lesions was high in a mining population. This increased prevalence could be accounted for by an increased attack rate with diminished progression and mortality rates. Alternatively it could be due to healed tuberculous lesions being more radio-opaque by virtue of the dust fibrosis, whereas in non-miners the lesions could heal leaving less radiological scarring.

Mann also demonstrated that there was an increased attack rate of tuberculosis (relative to non-miners) in men with categories 1 and 2 simple pneumoconiosis, but a fall in category 3. Associated with this fall there was a sudden rise in the attack rate of massive fibrosis in

category 3 simple pneumoconiosis which may be interpreted as representing the change of tuberculosis to massive fibrosis in high radiological categories.

The conclusion that tuberculosis is modified by the presence of simple pneumoconiosis can be deduced from the foregoing evidence but that massive fibrosis is itself a further modification of tuberculosis is suggested but remains unproven.

5. Epidemiological evidence.

Cochrane (1953) compared the tuberculosis mortality rate in two communities, the Rhondda Fach and Whitehaven. The economic circumstances were similar in both areas, and the female mortality rates were almost identical but the male mortality rates showed a fall in the older age groups in the Rhondda Fach. If the mortality rate from pneumoconiosis however, nearly all of which is attributable to massive fibrosis, is added to the tuberculosis mortality rate in the Rhondda Fach, the curve approximates fairly closely to the Whitehaven curve. This may be interpreted as meaning that a number of men in the Rhondda Fach who would have died of tuberculosis, are, in the presence of pneumoconiosis, dying as a result of massive fibrosis.

Cochrane et al., (1952) are at present attempting further epidemiological studies by discovering and isolating as many open cases of tuberculosis in a defined mining community as possible, serially Mantoux testing children (Jarman, 1953), and observing whether the attack rate of massive fibrosis in coal miners is reduced in comparison with a neighbouring area. In this way the importance of exogenous infection in the aetiology of progressive massive fibrosis is being investigated.

In conclusion there is evidence that progressive massive fibrosis is at the outset a modified form of tuberculosis but it must be re-iterated that this is still a hypothesis and it is as yet unproven.

That all cases of progressive massive fibrosis coming to autopsy have neither positive bacteriological nor pathological findings of tuberculosis need not invalidate the hypothesis for the following reasons. The active tuberculous process may heal as the massive fibrosis progresses, only the scar remaining. That cases with a persistently negative sputum can continue to progress radiologically till death could be explained on the histological evidence of partial vascular occlusion resulting in a replacement fibrosis. The

bacilli may be present in such small numbers as to render isolation difficult with known techniques.

It is still unexplained why men with progressive massive fibrosis associated with cavitation and a positive sputum, deteriorate rapidly, except that death usually occurs in the age group 40 - 60 years when there is, in the normal population, an increased male mortality rate from tuberculosis.

Cavitation in Progressive Massive Fibrosis.

Several theories have been suggested as to the pathogenesis of the cavitation that occurs in massive lesions in dust disease but it should be remembered that many of the earlier authors were concerned with classical silicosis as well as with what is now known as coalworkers' pneumoconiosis while many of the others wrote from sanatoria describing sputum positive cases.

Some authors considered that tuberculosis was possibly, or certainly the fundamental cause of massive fibrosis and its cavitation. Ribbert (1906) and Böhme (1925) considered that this was so; Amor and Evans (1934) wrote, "when skiagrams (in silicosis) show definite evidence of cavitation, superimposed tuberculosis is invari-

ably present." Jones (1938) reported 13 cases of silicosis with cavitation, 12 of whom had a sputum positive for tubercle bacilli, Belt and Ferris (1942) considered tuberculosis as a likely cause (page 81), Fourestier and Mariette (1948) considered that cavitation was indicative of a superimposed tuberculous infection and Steiger (1951) is in agreement but stresses the necessity of isolating the tubercle bacilli.

Other authors have suggested that tuberculosis plays no part. Seltmann (1867) described the cavitation as being caused by "mortification of lung tissue" as a result of tissue devitalisation surrounding the mass, while McCloskey (1943) reported one case that came to autopsy in whom no evidence of tuberculosis was found, but in whom a vascular endarteritis was apparent.

Others have suggested that there may be both tuberculous and non-tuberculous types of cavitation. Proust (1874 and 1876) described coal nodules giving rise to cavitation and stressed the fact that it is not necessarily a tuberculous process. Badham and Taylor (1936 and 1939) describing post-mortem material came to the conclusion that massive fibrosis could cavitate and that the resultant cavitation could be either tuberculous or non-tuberculous. Taylor and Alexander (1938), Gardner (1938 and 1939),

Auerbach and Stemmerman (1944), Schinz and Eggenschwyler (1947), Lee (1948), Theodos and Gordon (1951 and 1952), Heppleston (1951), Van Mechelen (1951) and Gernez-Rieux, Balgairès, Bonte and Delwaulle (1952) are in agreement that both tuberculous and non-tuberculous types of cavitation can occur in massive fibrotic lesions.

It has also been suggested that a secondary pyogenic or fungal infection may be the cause of cavitation in some instances, (Seltmann, 1867; Cummins and Sladden, 1930; Ornstein and Ulmar, 1936; Badham and Taylor, 1939) but no evidence has been produced in support of this theory.

Some investigators have paid particular attention to vascular changes in the lungs near areas of cavitation (Seltmann, 1867; Geever, 1947) and in this respect it is interesting to note that pulmonary cavitation has been described in polyarteritis nodosa by Sweeney and Baggenstoss (1949) and Sandler, Matthews and Bornstein (1950), who suggest that the cavitation has been caused by arterial obstruction producing local areas of infarction and subsequent necrosis.

It seems probable that any massive lesion may cavitate but that several factors may play a part. The liquefaction found in the centre of many massive lesions

is probably due to ischaemic necrosis and cavitation inevitably occurs when this softened zone communicates with a bronchus. This may occur spontaneously when gradual extension of necrosis reaches a bronchus or it may be accelerated by an acute respiratory infection in the region of the bronchus concerned. An interesting feature of the sputum negative cases is the absence of secondary infection of the cavity itself, and this allows most patients to continue enjoying fairly good general health for years after cavitation has occurred. It is difficult to see why these cavities should differ in respect of their liability to secondary pyogenic infections from cavities such as occur in neoplasm, bronchiectasis and cystic lungs (but see case 91). The repeated and prolonged expectoration of large amounts of black sputum is a remarkable feature and Marshall (1834) noted that one of his patients expectorated "as much as two English pints in 24 hours." The matter consists of accumulated bronchial secretion mixed with coal dust and cellular debris. It can readily be demonstrated that a very small quantity of carbon (even 1 ml. of Indian ink) is sufficient to give half a pint of sputum a jet black appearance.

If the contention is accepted that progressive

massive fibrosis is at the outset a modified form of tuberculosis, it must be concluded that in a majority of cases the infection dies out leaving a scar which may increase in extent following partial vascular occlusion. In the remainder, viable tubercle bacilli must persist within the mass and for some reason resume multiplication after prolonged quiescence, eventually leading to cavitation and to a fatal outcome, in most cases within two years, despite anti-tuberculous therapy.

Conclusions.

The prevention of massive fibrosis is of primary importance if the disability and mortality rate from dust disease are to be reduced. There are three measures that should be taken to achieve this end.

Firstly, dust suppression in the coal mines must be enforced to reduce dust concentrations to levels in which men can work for long periods of time without developing simple pneumoconiosis.

Secondly, coal workers should have periodic radiological examinations as recommended by Cochrane et al., (1951) for this procedure provides the only ultimate

test of efficient dust suppression. Men with early pneumoconiosis (category 1) would be detected and could be advised to work in less dusty places, so that category 2 simple pneumoconiosis with its attendant risk of massive fibrosis would not develop.

Thirdly, because tuberculosis is considered to be the fundamental cause of massive fibrosis, tuberculosis infection should be strictly controlled in communities where simple pneumoconiosis is prevalent.

Even if the tuberculosis hypothesis is eventually proved not to be true, these three measures are still laudable projects in their own right.

SUMMARY.

- 1). An historical review is given of pulmonary cavitation occurring in coalworkers, with special reference to the observations of the nineteenth century Scottish physicians.
- 2). Pulmonary cavitation may occur in association with simple pneumoconiosis. Eight such cases admitted to the Pneumoconiosis Research Unit ward between 1946 and 1952 are described. The differential diagnosis is discussed and the management and treatment are considered.
- 3). Cavitation was discovered in 104 patients with progressive massive fibrosis admitted to the ward between the same dates. Of these 104 cases, 26 had tubercle bacilli in the sputum during life and 78 did not, although bacilli were cultured from the lungs of one of them at autopsy. Difficulty in classification may arise from the finding of non-pathogenic acid-fast bacilli in the sputum and the importance of animal inoculation is stressed.
- 4). Fever, loss of weight, toxæmia and an elevated erythrocyte sedimentation rate are not reliable guides to the differentiation between sputum positive and negative cases of cavitated progressive massive

fibrosis because the frequent non-tuberculous respiratory infections in patients with progressive massive fibrosis may affect these clinical findings.

- 5). The prognosis for patients in the sputum positive group of cavitated progressive massive fibrosis is poor, few surviving for more than two years after the appearance of tubercle bacilli in the sputum. In the absence of a positive sputum the prognosis for patients with cavitated progressive massive fibrosis is no worse than for non-cavitated progressive massive fibrosis.
- 6). Treatment is unsatisfactory but the sputum positive cases should be given anti-tuberculous drugs for the symptomatic benefit which they frequently confer. In sputum negative cases cavitation is of little clinical significance and such cases only require reassurance and possibly symptomatic treatment.
- 7). The nature and pathogenesis of coalworkers' pneumoconiosis are discussed. It is considered that simple pneumoconiosis is a pure coal dust effect. Progressive massive fibrosis occurs in lungs which already contain a certain amount of coal dust and is probably the result of some additional factor. There is evidence that this factor is tuberculous infection but the

hypothesis remains unproven. Cavitation often occurs in massive fibrosis and it appears to be due to two basic processes, tuberculosis or ischaemic necrosis, acting alone or in combination.

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APPENDIX I.Summary of Cases.

In this appendix the essential details are given of all the ward cases studied. They are presented in summarised form, and inevitably contractions are used; these are explained below.

Notes.

- a). Each case is numbered and not named: these numbers are used throughout the text and the appendix of illustrative cases.
- b). The cases marked ^{*} have been seen personally. There are 49 in all.
- c). The year of birth only is given.
- d). The number or capital letter denotes the Pneumonia Research Unit radiological category. Letters are also used to denote the situation of cavities, e.g. R.U.L. Right Upper Lobe, L.L.L. Left Lower Lobe.

- e). The month and year of discovery of cavitation are given e.g. 6/47; this denotes that the cavity was first noted in June 1947. The date of death is similarly given.
- f). In sputum positive patients the date of the first positive sputum is given likewise, and the method, or methods by which the sputum was examined are stated. In the case of sputum negative patients the number of times that the sputum was examined by direct smear (s), culture (c) and guinea pig inoculation (g.p.) is given. The occurrence of haemoptysis (H) and black sputum (B.S.) is shown.
- g). Post-mortem (P.M.) findings are given where available and 'P.M.F. with tuberculosis' denotes progressive massive fibrosis with either histological or bacteriological confirmation of tuberculous disease.

1. Cases with cavitation in progressive massive fibrosis with a sputum positive for tubercle bacilli.

Cases 1 - 26.

Case No.	Year of Birth	X-ray cat. & cavity site	Date cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Creps.	Wt. change	E.S.R.	Sputum			Date of Death or State at 31.12.52.	Additional notes and comments.
										H.B.S.	Date of first positive and methods			
1	1900	B. Bilat. U.L.	6/46	Poor	Nil	Nil	Yes	Loss	> 10	+	+	7/46 smear	Died 4/48	No chemotherapy. No P.M. Diagnosed on smear only but considered as case of pul. tub.
2	1904	B. R.U.L.	11/48	Poor	Yes	Yes	Yes	Loss	No record	+	0	11/48 smear	Died 8/49	No chemotherapy. P.M. P.M.F. with tuberculosis.

Case No.	Year of Birth	X-ray cat. & cavity site	Date of cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Creps.	Wt. change	E.S.R.	Sputum		Date of Death or State at 31.12.52.	Additional notes and comments.
										H.B.S.	Date of first positive and methods		
3	1900	B. R.L.L. L. apical lower	7/50	Fair	Nil	Yes	Nil	No record	>10	+	8/50 smear and cult.	Died 12/51	P.M. P.M.F. with tuber- culosis.
4	1902	C. Bilat. U.L.	12/49	Good	Nil	Yes	Yes	Static	>10	0	12/49 cult.	Died 1/50	No chemo- therapy. No P.M.
5	1893	A. R.U.L.	6/47	Good	Nil	Nil	Yes	Slight Loss	>10	0	9/47 smear and cult.	Died 1/49	No Chemo- therapy. P.M. P.M.F. with tuber- culosis. P.M. culture positive.

Case No.	Year of Birth	X-ray cat. & cavity site	Date cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Creps.	Wt. change	E.S.R.	S p u t u m		Date of Death or State at 31.12.52.	Additional notes and comments.
										H.B.S.	Date of first positive and methods		
6	1888	B. Bilat. U.L.	6/47	Fair	Nil	Nil	Yes	Static	>10	0 +	10/48 smear	Alive.	Long survival time no chemo-therapy. ? non-pathogenic acid-fast bacilli.
7	1915	A. R.U.L.	10/51	Good	Nil	Nil	Nil	Static	<10	0 +	9/51 smear	Alive.	Referred to Sanatorium for chemo-therapy.
8	1907	B. R.U.L.	6/51	Poor	Nil	Yes	Nil	Slight Loss	>10	0 +	6/51 smear and cult.	Alive.	Streptomycin and P.A.S.

Case No.	Year of Birth	X-ray cat. & cavity site	Date of cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Creps.	Wt. change	E.S.R.	S p u t u m		Date of Death or State at 31.12.52.	Additional notes and comments.
										H.B.S.	Date of first positive and methods		
9	1912	B. R.U.L.	11/50	Good	Nil	Yes	Yes	Static	>10	0 +	12/50 smear cult. E.P.	Alive.	Strepto- mycin and P.A.S. <u>Illus-</u> <u>trative</u> <u>case.</u> Add. died 2/53.
10	1894	B. L.U.L.	10/48	Poor	Nil	Nil	Yes	Loss	>10	+ +	10/48 smear	Died 11/49	No chemo- therapy. P.M. P.M.F. with tuber- culosis.

Case No.	Year of Birth	X-ray cat. & cavity site	Date of cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Crep.	Wt. change	E.S.R.	Sputum		Date of Death or State at 31.12.52.	Additional notes and comments.
										H.B.S.	Date of first positive and methods		
11	1914	R.U.L.	10/47	Poor	Yes	Nil	Yes	Loss	>10	0 +	10/47 smear cult. g.p.	Died 7/48	No chemotherapy. No P.M.
12	1896	C. R.U.L.	9/46	Good	Nil	Yes	Yes	Loss	>10	+	10/46 smear cult. g.p.	Died 1/48	No chemotherapy. No P.M.
13 *	1906	C. Bilat. U.L.	12/46	Poor	Yes	Yes	Nil	Loss	No record	0 +	6/52 smear	Alive	Spontaneous pneumothorax (L) 5/52. Referred to clinic for chemotherapy.

Case No.	Year of Birth	X-ray cat. & cavity site	Date cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Crep. s.	Wt. change	E.S.R.	Sputum		Date of Death or State at 31.12.52.	Additional notes and comments.
										H.B.S.	Date of first positive and methods		
14	1899	D. R.U.L.	3/46	Poor	Nil	Yes	Nil	Loss	No record	0	+	3/46 smear cult.	Died 1/47 No chemotherapy. P.M. P.M.F. and tuberculosis.
15	1909	B. R.L. apical lower	6/50	Fair	Nil	Yes	Nil	Slight Loss	> 10	+	+	7/50 smear cult.	Died 10/52 Streptomycin, P.A.S. and pneumoperiton-eum. P.M. P.M.F. with tuberculosis. Culture positive.

Case No.	Year of Birth	X-ray cat. & cavity site	Date of cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Creps.	Wt. change	E.S.R.	Sputum		Date of Death or State at 31.12.52.	Additional notes and comments.
										H.B.S.	Date of first positive and methods		
16 [*]	1905	B. R.U.L.	9/52	Fair	Nil	Yes	Yes	Loss	> 10	0 +	9/52 smear cult. g.p.	Alive.	Streptomycin and P.A.S.
17	1887	C. R.U. I.U.L.	6/50	Poor	Nil	Yes	Yes	Loss	No record	+	6/50 smear	Died 7/50	No chemotherapy. No P.M. Diagnosed on smear only but considered as case of pul. tuberculosis.

Case No.	Year of Birth	X-ray cat. & cavity site	Date of cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Creps.	Wt. change	E.S.R.	S p u t u m		Date of Death or State at 31.12.52.	Additional notes and comments.
										H.B.S.	Date of first positive and methods		
18	1902	D. L.U.L.	10/51	Poor	Yes	Yes	Yes	Loss	No record	0 +	10/51 smear	Died 10/51	No chemo-therapy. P.M. P.M.F. with tuberculousis.
19	1895	C. R. apical lower	2/47	Poor	Yes	Yes	Yes	Loss	> 10	0 +	5/46 smear g.p.	Died 3/48	No chemo-therapy. No P.M.
20 *	1912	C. R.U.L. L. apical lower	6/50	Good	Nil	Nil	Yes	Static	> 10	+	9/51 g.p.	Alive.	No chemo-therapy. Cavities refilled.

Case No.	Year of Birth	X-ray cat. & cavity site	Date cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Creps.	Wt. change	E.S.R.	S p u t u m		Date of Death or State at 31.12.52.	Additional notes and comments.
										H.B.S.	Date of first positive and methods		
21	1891	B. Bilat. U.L.	1/50	Good	Yes	Nil	Nil	Loss	> 10	+	1/50 cult.	Alive	No chemotherapy. Long survival. 1 culture positive. ? non-pathogenic acid-fast bacilli.
22	1895	A. R.U. & L.L.	9/49	Poor	Yes	Yes	Yes	Static	> 10	0 +	11/49 cult.	Died 8/50	Streptomycin and P.A.S. P.M. P.M.F. with tuberculosis.

Case No.	Year of Birth	X-ray cat. & cavity site	Date cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Creps.	wt. change	E.S.R.	S p u t u m		Date of Death or State at 31.12.52.	Additional notes and comments.
										H.B.S.	Date of first positive and methods		
23	1912	D. L.U.L.	4/51	Poor	Nil	Nil	Nil	Static	>10	+	+	Died 6/51	Strepto- mycin and P.A.S. P.M. P.M.F. with tub- erculosi- Culture positive.
24	1893	C. Bilat. U.L.	6/49	Good	Nil	Yes	Yes	Slight Loss	>10	+	+	Died 1/51	No chemo- therapy. P.M. P.M.F. with tub- erculosi-.

Case No.	Year of Birth	X-ray cat. & cavity site	Date of cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Crep. s.	Wt. change	E.S.R.	Sputum		Date of Death or State at 31.12.52.	Additional notes and comments.
										H.B.S.	Date of first positive and methods		
25	1902	D. R. apical lower	2/46	Poor	Nil	Yes	Yes	Loss	> 10	+	4/46 smear cult. g.p.	Died 10/47	No chemo-therapy. Died following massive haemoptysis. No P.M. Illus- <u>trative</u> <u>case.</u>
26	1887	A. L.U.L.	11/47	Poor	Nil	Yes	Nil	Loss	No record	0 +	11/47 smear	Died 3/48	No chemo-therapy. P.M. P.M.F. and tuber- culosis.

2. Cases with cavitation in progressive massive fibrosis with a sputum negative for tubercle bacilli.

Cases 27 - 104.

Case No.	Year of Birth	X-ray cat. & cavity site	Date cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Creps.	Wt. change	E.S.R.	Sputum Detail		Date of Death or State at 31.12.52.	Additional notes and comments.
										Number of times negative & methods	H.B.S.S.C.C.P.		
27 *	1905	D. R.U. & ap. lower I.U. & I.L.	9/48	Good	Yes	Yes	Static	> 10	0 +	28.20. -	Alive.	<u>Illus- trative case.</u>	
28	1899	D. L. apical lower	5/41	Fair	Nil	Nil	Static	> 10	0 +	3. - . -	Died 10/48	No P.M.	
29	1907	C. Bilat. U.L.	2/49	Good	Nil	Nil	Slight Loss	> 10	0 +	4.10. -	Alive.	<u>Illus- trative case of emptying and re- filling of cavities.</u>	

Case No.	Date of Birth	X-ray cat. & cavity site	Date cav. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Creps.	Wt. change	E.S.R.	Sputum Detail Number of times negative & methods	Date of Death or State at 31.12.52.	Additional notes and comments.
30	1892	B. I.U.L.	12/52	Fair	Yes	Yes	Yes	Static	>10	+ 0	3.6.1.	Alive.
31	1891	B. Bilat. U.L.	7/47	Fair	Yes	Yes	Yes	Loss	>10	0 +	1.8.8.	P.M. Typical P.M.F.
32	1899	C. L. apical lower	8/41	Fair	Nil	Nil	Yes	Static	>10	0 +	1.- -	P.M. Typical P.M.F.
33	1903	D. R.U. & apical lower I.U.L.	3/46	Good	Nil	Nil	Nil	Slight Loss	>10	+ 0	3.4.3.	Alive.

Case No.	Date of Birth	X-ray cat. & cavity site	Date of cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Crep. s.	Wt. change	E.S.R.	Sputum Detail		Date of Death or State at 31.12.52.	Additional notes and comments.
										Number of times negative & methods	H.B.S.S.C.G.P.		
34	1896	D. Bilat. U.L.	10/49	Poor	Yes	Yes	Nil	Loss	> 10	0 +	8.8. -	Died 1/50	P.M. Typical P.M.F. Died of acute bronchitis.
35 *	1912	C. R. apical lower	3/52	Fair	Nil	Nil	Yes	Loss	> 10	0 0	3.6.1.	Alive	
36	1889	C. Bilat. U.L.	5/47	Poor	Nil	Nil	Yes	Slight Loss	> 10	0 +	24.35.1.	Died 11/50	Illus- trative case. Died strangu- lated hernia. P.M. Typical P.M.F. (cult. negative).

Case No.	Year of Birth	X-ray cat. & cavit. site.	Date of cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Crep. s.	Wt. change	E.S.R.	Sputum Detail		Date of Death or State at 31.12.52.	Additional notes and comments.
										Number of times negative & Methods	H.B.S.S.C.G.P.		
37	1910	B. Bilat. U.L.	7/42	Good	Yes	Nil	Yes	Gain	> 10	0 +	- 3.3.	Alive.	
38 *	1906	D. R.&L. U.L.	3/46	Good	Yes	Yes	Yes	Loss	> 10	0 +	20.16.-	Alive.	
39	1902	D. Bilat. U.L.	6/48	Fair	Nil	Nil	Yes	Slight Loss	> 10	+	17.17.2.	Died 2/50	Died of cardiac failure. P.M. Typical P.M.F.
40 *	1898	B. R. apical lower	10/52	Fair	Yes	Yes	Yes	Static	> 10	0 +	3.6.1.	Alive	

Case No.	Year of Birth	X-ray cat. & cavity site	Date cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Crep. s.	Wt. change	E.S.R.	Sputum Detail		Date of Death or State at 31.12.52.	Additional notes and comments.
										Number of times negative & methods	H.E.S.S.C.G.P.		
41	1913	D. R.U.L. L. apical lower	3/46	Poor	Yes	Nil	Nil	Slight Loss	>10	+	1, 9, -	Died 10/47	P.M. Typical P.M.F.
42	1894	D. L.U.L.	11/46	Fair	Nil	Nil	Nil	Static	>10	0 0	11.22.2.	Died 10/51	Died of cardiac failure. P.M. Typical P.M.F.
43 *	1898	D. L. apical lower	7/52	Poor	Yes	Yes	Yes	Loss	>10	0 +	6.6. -	Died 7/52	P.M. Typical P.M.F. cult. neg. Died of cardiac failure.

Case No.	Year of Birth	X-ray cat. & cavity site	Date cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Crep. s.	Wt. change	E. S. R.	Sputum Detail		Date of Death or State at 31.12.52.	Additional notes and comments.
										Number of times negative & methods	H. B. S. S. C. G. P.		
44	1905	D. Bilat. U.L.	5/49	Fair	Yes	Yes	Nil	Slight Loss	>10	+	7.9.	Died 1/51	Died result of large haemoptysis P.M. Typical P.M.F.
45 *	1902	D. L.U.L.	1/43	Fair	Yes	Nil	Nil	Slight Loss	>10	0 +	19.24.1.	Alive	Marked broncho-spasm.
46 *	1890	D. R.U.L.	12/52	Poor	Yes	Yes	Yes	Loss	>10	0 +	6.7.1.	Alive	

Case No.	Year of Birth.	X-ray cat. & cavity site	Date cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Crep. Creps.	Wt. change	E. S. R.	Sputum Detail		Date of Death or State at 31.12.52.	Additional notes and comments.
										Number of times negative & methods	H B.S.S.C.G.P.		
47*	1891	C. L.U.L.	5/52	Good	Nil	Nil	Nil	Slight Loss	>10	+	12. 6.-	Alive	Rheumatoid Arthritis.
48	1896	C. R.U.L.	10/50	Poor	Nil	Nil	Yes	Loss	>10	0 0	3. 6.-	Died 11/50	P.M. Typical P.M.F. Died Ca. bladder.
49*	1902	B. R.U.L.	1/51	Good	Nil	Yes	Nil	Loss	>10	+	32.36.4.	Alive.	Has Rheumatoid Arthritis.
50*	1893	D. Bilat. U.L.	10/49	Good	Nil	Yes	Nil	Static	>10	+	7. 7.-	Alive.	Has re-current laryngeal palsy. ? pressure from mass. Illus-trative case.

Case No.	Year of Birth	X-ray cat. & cavity site	Date of cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Creps.	Wt. change	E.S.R.	Sputum Detail		Date of Death or State at 31.12.52.	Additional notes and comments.
										Number of times negative & methods	H.B.S.S.C.G.P.		
51 *	1916	C. R.U. & apical lower L.U.L.	7/50	Good	Nil	Nil	Nil	Static	>10	+	16.14.--	Alive	
52 *	1902	C. Bilat. U.L.	3/46	Fair	Yes	Yes	Nil	Static	>10	+	6. 6.--	Alive	
53 *	1896	D. Bilat. U.L.	8/49	Poor	Nil	Yes	Nil	Slight Loss	>10	+	9. 9.--	Died 4/52	P.M. Typical P.M.F. Died in cardiac failure.

Case No.	Year of Birth	X-ray cat. & cavity site	Date Cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever Creps.	Wt. change	E.S.R.	Sputum Detail Number of times negative & methods	Date of Death or State at 31.12.52.	Additional notes and comments.
54	1900	D. Bilat. U.L.	1/50	Fair	Yes	Yes	Slight Loss	>10	+ +	20.15.	Died of cor pulmonale. P.M. Typical P.M.F. Culture negative for tubercle bacilli.
55 *	1911	D. R.U. & apical lower L.U.	11/45	Good	Yes	Yes	Static	>10	+ +	2. 7.	Alive <u>Illus-</u> <u>trative</u> <u>case.</u> <u>Sequestrum</u> <u>formation.</u>

Case No.	Year of Birth	X-ray cat. & cavity site	Date cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Creps.	Wt. change	E.S.R.	Sputum Detail Number of times negative & methods	Date of Death or State at 31.12.52.	Additional notes and comments.	
													H.B.S.S.C.G.P.
56	1894	B. Bilat. U.L.	5/51	Good	Yes	Nil	Nil	Slight Loss	>10	0 +	10. 8. -	Alive.	
57 *	1893	B. Bilat. U.L.	6/44	Good	Nil	Nil	Yes	Static	>10	0 +	10. 8. -	Alive.	Early R. sided cardiac failure.
58 *	1906	C. R.U. & apical lower L.U.	6/47	Good	Yes	Yes	Yes	Static	>10	+ +	46.46-	Alive.	Frequent haemoptyses. Illus- <u>trative</u> case. Cavit. in P.M.F. and in pneu- <u>monic</u> infection.

Case No.	Year of Birth	X-ray cat. & cavity site	Date of cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Creps.	Wt. change	E.S.R.	Sputum Detail		Date of death or State at 31.12.52.	Additional notes and comments.
										Number of times negative & methods	H.B.S.S.C.G.P.		
59*	1904	B. R.&L. U.L.	4/42	Good	Yes	Nil	Yes	Slight Loss	> 10	+	44.41.2.	Alive.	
60	1901	B. Bilat. U.L.	5/50	Good	Nil	Nil	Yes	Static	> 10	0 0	5.5.-	Alive.	
61	1902	C. R.U. & apical lower L.U.	10/48	Good	Nil	Yes	Nil	Loss	< 10	+	6.6.-	Alive.	
62	1904	C. R.U.L.	11/48	Good	Yes	Nil	Nil	No Record	> 10	+	1.2.2.	Alive.	

Case No.	Year of Birth	X-ray cat. & cavity site	Date of cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Creps.	Wt. change	E.S.R.	Sputum Detail		Date of Death or State at 31.12.52.	Additional notes and comments.
										Number of times negative & methods	H.B.S.S.C.G.P.		
63 *	1895	D. L.U.L.	3/51	Poor	Nil	Yes	Yes	Static	>10	0 0	11.13.51.	Died 11/52	Non-path. acid-fast bacilli. (atypical culture neg. smear and G.P.). P.M. Typical F.M.F.
64 *	1887	D. L.U.L.	7/52	Fair	Yes	Yes	Yes	Slight Loss	>10	0 +	6. 7.-	Alive	R. pleural effusion. (Negative for tubercle bacilli).

Case No.	Year of Birth	X-ray cat. & cavity site	Date cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Creps.	Wt. change	E.S.R.	Sputum Detail		Date of Death or State at 31.12.52.	Additional notes and comments.
										Number of times negative & methods	H.B.S.S.C.G.P.		
65 *	1914	B. Bilat. U.L.	1/49	Good	Yes	Yes	Yes	Gain	> 10	+ 0	22.12.1.	Alive.	
66	1893	C. Bilat. U.L.	6/46	Good	Nil	Nil	Yes	Loss	< 10	+ +	3.4. -	Alive.	
67 *	1895	C. R.U. & apical lower R.L.L. L.U.	6/49	Good	Yes	Yes	Yes	Slight Loss	> 10	+ +	14.9. -	Alive.	Severe pneumonic illness.

Case No.	Year of Birth	X-ray cat. & cavity site	Date cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Crepes.	Wt. change	E.S.R.	Sputum Detail		Date of Death or State at 31.12.52.	Additional notes and comments.
										Number of times negative & methods	H.B.S.S.C.G.P.		
68	1912	A. Bilat. U.L.	3/48	Good	Nil	Nil	Nil	Static	>10	0 0	- 1.-	Alive.	
69 *	1902	C. L.U.L.	4/51	Good	Yes	Yes	Yes	Slight Loss	>10	0 +	6.6.-	Alive.	
70 *	1908	D. R.U.L.	1/51	Good	Yes	Yes	Yes	Slight Loss	>10	0 +	11.3L-	Alive.	

Case No.	Year of Birth	X-ray cat. & cavity site	Date cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Grep's.	Wt. change	E.S.R.	Sputum Detail		Date of Death or State at 31.12.52.	Additional notes and comments.
										Number of times negative & methods	H.B.S.S.C.G.P.		
71 *	1881	B. Bilat. U.L.	6/49	Good	Yes	Nil	Yes	Static	> 10	0 +	5.21.1.	Alive.	
72 *	1902	B. R.U.L. L. apical lower.	7/46	Fair	Yes	Yes	Yes	Loss	> 10	+ +	40.40.5.	Alive.	
73 *	1906	D. R.U. & apical lower L.U.	6/48	Fair	Yes	Yes	Yes	Static	> 10	+ +	63.45.5.	Alive.	Non pathogenic acid-fast bacilli. (8 occasions) (smears). No chemotherapy before this finding. Sequesterum formation in L. lung <u>Illustrative case.</u>

Case No.	Year of Birth	X-ray cat. & cavity site	Date cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Creps.	Wt. change	E.S.R.	Sputum Detail Number of times negative & methods	Date of Death or State at 31.12.52.	Additional notes and comments.
74	1887	A. Bilat. U.L.	4/45	Fair	Nil	Nil	Nil	Loss	>10	+	1. 2. 1. Alive.	
75	1887	B. Bilat. U.L.	2/41	Good	Yes	Yes	Yes	Slight Loss	>10	+	6.14.--	No P.M. Died of cardiac failure.
76 *	1910	B. R.U.L.	2/48	Good	Nil	Nil	Yes	Static	>10	+	8. 5.-- Alive.	Illus- trative case.
77 *	1894	D. Bilat. U.L. & apical lower	6/46	Poor	Yes	Yes	Yes	Static	>10	+	39.29.1. Died 5/52	P.M. Typical P.M.F.

Case No.	Year of Birth	X-ray cat. & cavity site	Date & cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Creps.	Wt. change	E.S.R.	Sputum Number of times negative & methods	Detail	Date of Death or State at 31.12.52.	Additional notes and comments.
78	1898	B. L.U.L.	11/50	Fair	Nil	Nil	Yes	Loss	>10	+ 0	4. 4.--	Died 4/51	P.M. Typical P.M.F. Cult. negative.
79	1902	D. R.U. & apical lower L.U. & apical lower.	5/42	Poor	Nil	Yes	Yes	Slight Loss	>10	+ +	6. 11.--	Died 2/47	P.M. P.M.F. with typical histology but P.M. cult. pos. for tubercle bacilli.
80 *	1882	D. Bilat. U.L.	8/42	Fair	Yes	Yes	Yes	Static	>10	0 +	6. 6.--	Alive.	

Case No.	Year of Birth	X-ray cat. & cavity site	Date of cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Crep. S.	Wt. change	E.S.R.	Sputum Detail Number of times negative & methods	Date of Death or State at 31.12.52.	Additional notes and comments.
81	1893	D. R.U. & apical lower L.U.	2/46	Fair	Yes	Yes	Yes	Loss	>10	+ + + H.B.S.S.C.G.P.	5. 9. 4. Alive.	Illus- trative case of extensive bilateral cavitation
82	1889	C. R.U.L.	3/49	Fair	Yes	Nil	No	Static	<10	0 + - 1.-	Alive.	
83	1895	D. L.U.L.	11/49	Poor	Yes	Nil	Yes	Static	>10	0 0 17.15.-	Died 7/51	P.M. Typical P.M.F. Cult. & g.P. negative for tub. bacilli.

Case No.	Year of Birth	X-ray cat. & cavity site	Date cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever Creps.	Wt. change	E.S.R.	Sputum Detail		Date of Death or State at 31.12.52.	Additional notes and comments.	
									Number of times negative & methods	H.B.S. S.C.G.P.			
84 *	1892	B. R.U.I.	12/49	Fair	Nil	Yes	No	Static	> 10	+	6.6. -	Alive.	
85 *	1905	B. Bilat. I.L.	3/46	Good	Nil	Nil	Yes	Static	< 10	+	6.6. -	Alive.	Frequent haemoptyses.
86 *	1899	C. R.U.I. I. apical lower & I. I.L.I.	5/50	Fair	Yes	Yes	Nil	Slight Loss	> 10	+	18.14. -	Alive.	Marked bronchospasm and cor pulmonale.

Case No.	Year of Birth	X-ray cat. & cavity site	Date of cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Crep. s.	Wt. change	E.S.R.	Sputum Detail		Date of Death or State at 31.12.52.	Additional notes and comments.
										Number of times negative & methods	H.B.S.S.C.G.P.		
87 *	1896	D. R. & L. U. & apical lower	7/39	Good	Yes	Yes	Nil	Static	>10	+	0	21.21.1. Alive	Repeated ac. resp-iratory infections.
88	1901	C. R & L U and apical lower	5/46	Good	Yes	Yes	Yes	Slight Loss	>10	+	+	11.27.1. Alive.	
89 *	1902	B. R.U.L.	9/50	Good	Nil	Nil	Yes	Static	>10	0	+	55.56.- Alive.	
90 *	1914	B. Bilat. U.L.	9/48	Good	Nil	Yes	Nil	Static	>10	+	+	40.44.4. Alive.	

Case No.	Year of Birth	X-ray cat. & cavity site	Date of cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Creps.	Wt. change	E. S. R.	Sputum Detail Number of times negative & methods	Date of Death or State at 31.12.52.	Additional notes and comments.
91 *	1902	D. L. U. L.	3/51	Fair	Yes	Yes	Yes	Loss	> 10	+ +	31.26.- Alive.	Illus- trative case. "Abscess" type. Marked finger clubbing.
92 *	1888	D. Bilat. U. L.	6/50	Fair	Nil	Yes	Nil	Static	> 10	+ 0	21.18.- Alive.	R. spon- taneous pneumo- thorax.
93 *	1892	C. R. & L. L. L.	7/42	Fair	Yes	Yes	Yes	Static	> 10	+ +	4.13.6. Alive.	

Case No.	Year of Birth	X-ray cat. & cavity site	Date of cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Crep. s.	Wt. change	E.S.R.	Sputum Detail		Date of Death or State at 31.12.52.	Additional notes and comments.
										Number of times negative & methods	H.B.S.S.C.G.P.		
94	1906	C. Bilat. U.L.	10/47	Fair	Nil	Nil	Yes	Slight Loss	>10	0 +	9. 7. -	Died 11/50	P.M. Typical P.M.F. Died of congestive heart failure.
95 *	1901	D. Bilat. U.L.	11/44	Poor	Yes	Nil	Nil	Slight Loss	>10	0 +	16.15.1.	Alive.	Also rheumatoid arthritis.
96	1903	C. Bilat. U.L.	4/50	Fair	Yes	Yes	Nil	Static	>10	0 +	11.11.-	Alive.	Also rheumatoid arthritis.

Case No.	Year of Birth	X-ray cat. & cavity site	Date cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Creps.	Wt. change	E.S.R.	Sputum Detail		Date of Death or State at 31.12.52.	Additional notes and comments.
										Number of times negative & methods	H.B.S.S.C.G.P.		
97 *	1899	C. R.U.& apical lower L.U.L.	4/49	Fair	Yes	Yes	Yes	Static	>10	0 +	18.17.--	Alive	Also rheumatoid arthritis and peptic ulceration
98	1912	C. R.U.L. L.U. and apical lower	5/48	Good	Nil	Yes	Yes	Slight Loss	>10	0 +	21.14.--	Alive.	
99	1907	D. L. apical lower	7/49	Good	Yes	Yes	Yes	Slight Loss	>10	0 +	1. 2.1.	Alive.	

Case No.	Year of Birth	X-ray cat. & cavity site	Date cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Creps.	Wt. change	E.S.R.	Sputum Detail		Date of Death or State at 31.12.52.	Additional notes and comments.
										Number of times negative & methods	H.B.S.S.C.G.P.		
100 *	1891	B. Bilat. U.L. and apical lower.	1/48	Poor	Nil	Yes	Yes	Loss	>10	+	4. 4. -	Alive.	
101	1905	D. Bilat. U.L.	1/49	Fair	Yes. pneumo- nia	Yes	Nil	Static	>10	+	8. 8. -	Died 5/50	No P.M.
102	1900	C. R.U.I.	5/47	Poor	Nil	Nil	Nil	Loss	>10	0 +	5. 5. 5.	Died 2/48	P.M. Typical P.M.F.

Case No.	Year of Birth	X-ray cat. & cavity site	Date cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Crep. s.	Wt. change	E.S.R.	Sputum Detail		Date of Death or State at 31.12.52.	Additional notes and comments.
										Number of times negative & methods	H.E.S.S.C.G.P.		
103	1913	C. R.U. L.U. and apical lower	3/48	Fair	Nil	Yes	Nil	Static	>10	0 +	2. 1.-	Died 6/49.	
104	1905	B. L.U.L.	5/50	Fair	Nil	Nil	Nil	Loss	>10	0 +	6. 5.-	Alive.	Rheumatoid Arthritis.

3. Cases with cavitation in coalworkers' pneumoconiosis
other than in association with progressive massive
fibrosis.

Cases 105 - 112.

Case No.	Year of Birth	X-ray cat. & cavity site	Date cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Creps.	Wt. change	E.S.R.	Sputum		Date of Death or State at 31.12.52.	Additional notes and comments.
										H.B.S. method	Date & method of positive or no. & method of negative		
105	1894	Cat. 2 tuber- culosis R.L.L.	10/48	Poor	Nil	Yes	Yes	Loss	>10	0 0	10/48 smear pos. T.B.	Died 11/48	Simple pneumo- coniosis and cavit- ated tuber- culous broncho- pneumonia. No chemo- therapy.
106	1880	Cat. 1 L.M.Z.	10/50	Fair	Nil	Nil	Yes	Slight Loss	>10	+	4. 1- neg.	Died 2/51	Cavitated bronchial carcinoma histolo- gically proven at P.M. <u>Illus-</u> <u>trative</u> <u>case.</u>

Case No.	Year of Birth	X-ray cat. & cavity site	Date cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Creps.	Wt. change	E.S.R.	Sputum		Date of Death or State at 31.12.52.	Additional notes and comments.
										H.B.S.	Date & method of positive or no. & method of negative		
107	1897	Cat. l. tuberculous R.U.I.	3/49	Poor	Nil	Yes	Yes	Loss	>10	+	3/49 smear pos. T.B.	Died 3/49	P.M. Pneumoconiosis and tuberculous bronchopneumonia. (Both lungs). Illustriative case.
108 *	1910	"Rheumatoid type" Bilat. U.L.	5/52	Good	Nil	Nil	Nil	Loss	>10	0 0	10/50 cult. and G.P. pos.	Alive.	Rheumatoid arthritis. Had streptomycin & P.A.S. Illustriative case.

Case No.	Year of Birth	X-ray cat. & cavity site	Date cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Creps.	Wt. change	E.S.R.	Sputum		Date of Death or State at 31.12.52.	Additional notes and comments.
										H.B.S.	Date & method of positive or no. & method of negative		
109	1881	Cat. 1. L. apical lower	4/50	Fair	Yes	Yes	Yes	Slight Loss	>10	+ 0	8. 8 - negative	Died 3/52	Chronic lung abscess; intensive chemo-therapy. P.M. coal foci: no evidence of abscess cavity.
110 *	1895	Cat. 2. Discrete U.L. opacities. R.U.L.	1/51	Good	Nil	Nil	Nil	Static	<10	0 +	- 6 - negative	Alive.	"Rheumatoid type" of shadows. No rheumatism. <u>Illustriative case.</u>

Case No.	Year of Birth	X-ray cat. & cavity site	Date cavit. first seen	Gen. Cond.	Recent acute resp. infect.	Fever	Creps.	Wt. change	E.S.R.	Sputum		Date of Death or State at 31.12.52.	Additional notes and comments.
										H.B.S.	Date & method of positive or no. & method of negative		
111	1890	Cat. 2. L.U. and L.L.	7/46	Poor	Nil	Yes	Yes	Loss	>10	0 +	9/46 smear cult. g.p. pos.	Died 6/47	No P.M. No chemo- therapy. Rapidly fatal unmodi- fied tuberc- ulosis.
112	1909	Cat. 3. Tuberc- ulosis Bilat.	6/48	Poor	Nil	Yes	Yes	Loss	>10	0 0	6/48 smear pos.	Died 6/48	No chemo- therapy. P.M.: extensive caseous tuberc- ulosis & tubercular broncho- pneumonia Culture positive. Illus- trative case.

APPENDIX II.Illustrative Cases.

- 1). Cavitation in progressive massive fibrosis with a sputum positive for tubercle bacilli.

Case No. 9. p. 157.

Case No. 25. p. 161.

- 2). Cavitation in progressive massive fibrosis with a sputum negative for tubercle bacilli.

Case No. 27. p. 164.

Case No. 29. p. 167.

Case No. 36. p. 171.

Case No. 50. p. 175.

Case No. 55. p. 177.

Case No. 58. p. 179.

Case No. 73. p. 188.

Case No. 76. p. 192.

2). Cavitation in progressive massive fibrosis with
a sputum negative for tubercle bacilli. (Cont'd).

Case No. 81. p.196.

Case No. 91. p.199.

3). Cavitation in coalworkers' pneumoconiosis other
than in association with progressive massive
fibrosis.

Case No. 106. p. 204.

Case No. 107. p. 206.

Case No. 108. p. 208.

Case No. 110. p. 214.

Case No. 112. p. 218.

Case No. 9.

Date of Birth: 1912.

This man worked underground in house and steam coal pits for 20 years mostly at the coal face but left the pits when he was 34 years of age because of dyspnoea on exertion and fatigue. He was first seen in 1948 when he was 36 years of age and a chest radiograph (fig. 8) revealed moderately advanced massive fibrosis without cavitation, the sputum was negative for tubercle bacilli on direct examination and on culture at this time. In 1950 the cough and sputum increased, he was more dyspnoeic and further radiography revealed that cavitation had occurred in the mass in the right lung (fig. 9). The sputum was positive for tubercle bacilli on direct examination, culture and guinea pig inoculation.

During treatment by bed rest and a course of streptomycin and para-amino salicylic acid, the cough became less troublesome, the sputum negative and less in quantity, the temperature normal and the erythrocyte sedimentation rate lower. A month after the cessation of chemotherapy, however, fever returned with a worsening of symptoms and the sputum again became positive. The general condition improved after a further course of streptomycin and para-amino salicylic acid but the

sputum remained positive. Death occurred 2 years and 2 months after tubercle bacilli were first isolated illustrating the rapid deterioration of patients with massive fibrosis and a positive sputum, which is commonly even more rapid than in this case.

Figure 8.

Case No. 9. Radiograph in 1948 showing bilateral massive fibrosis without cavitation.

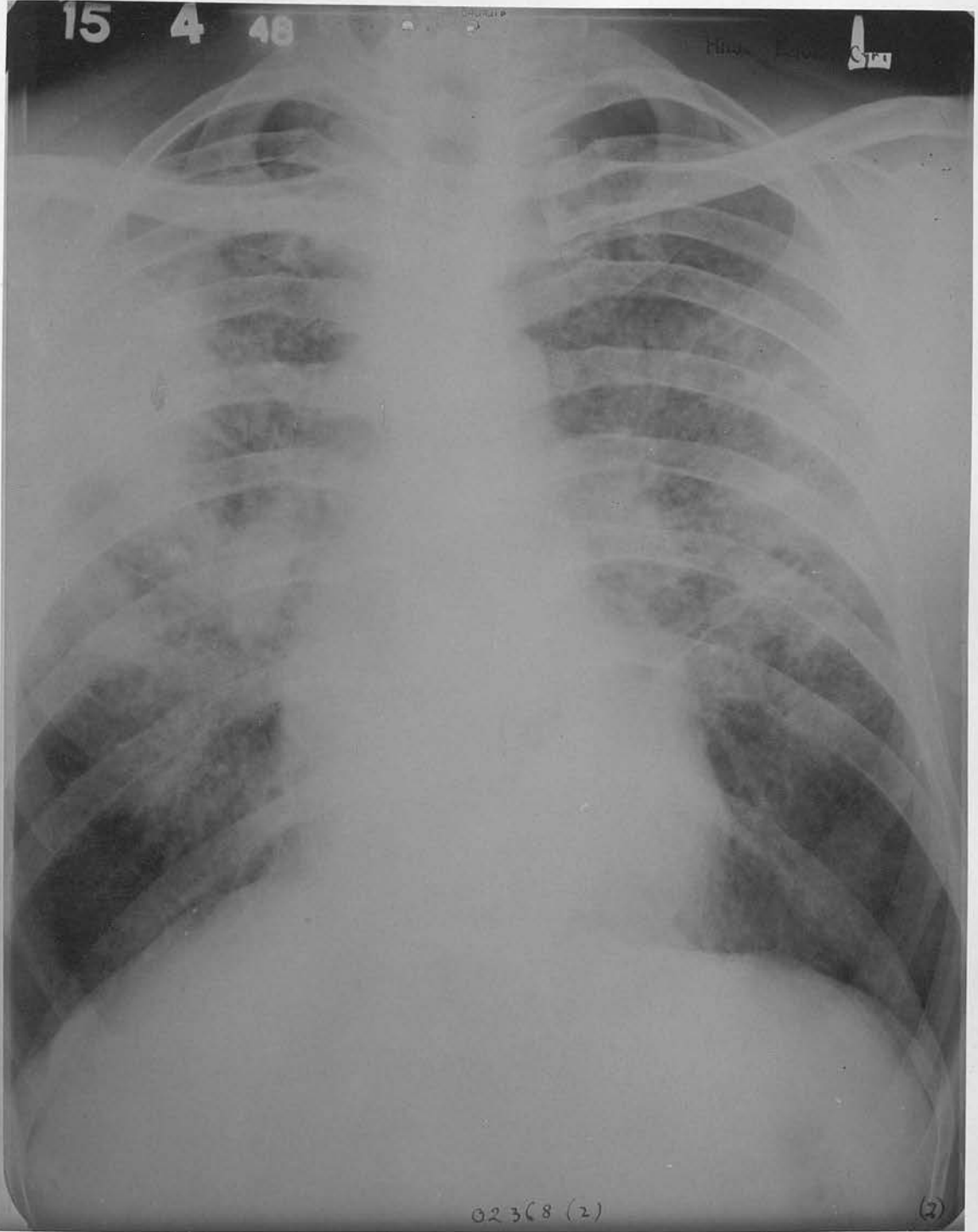
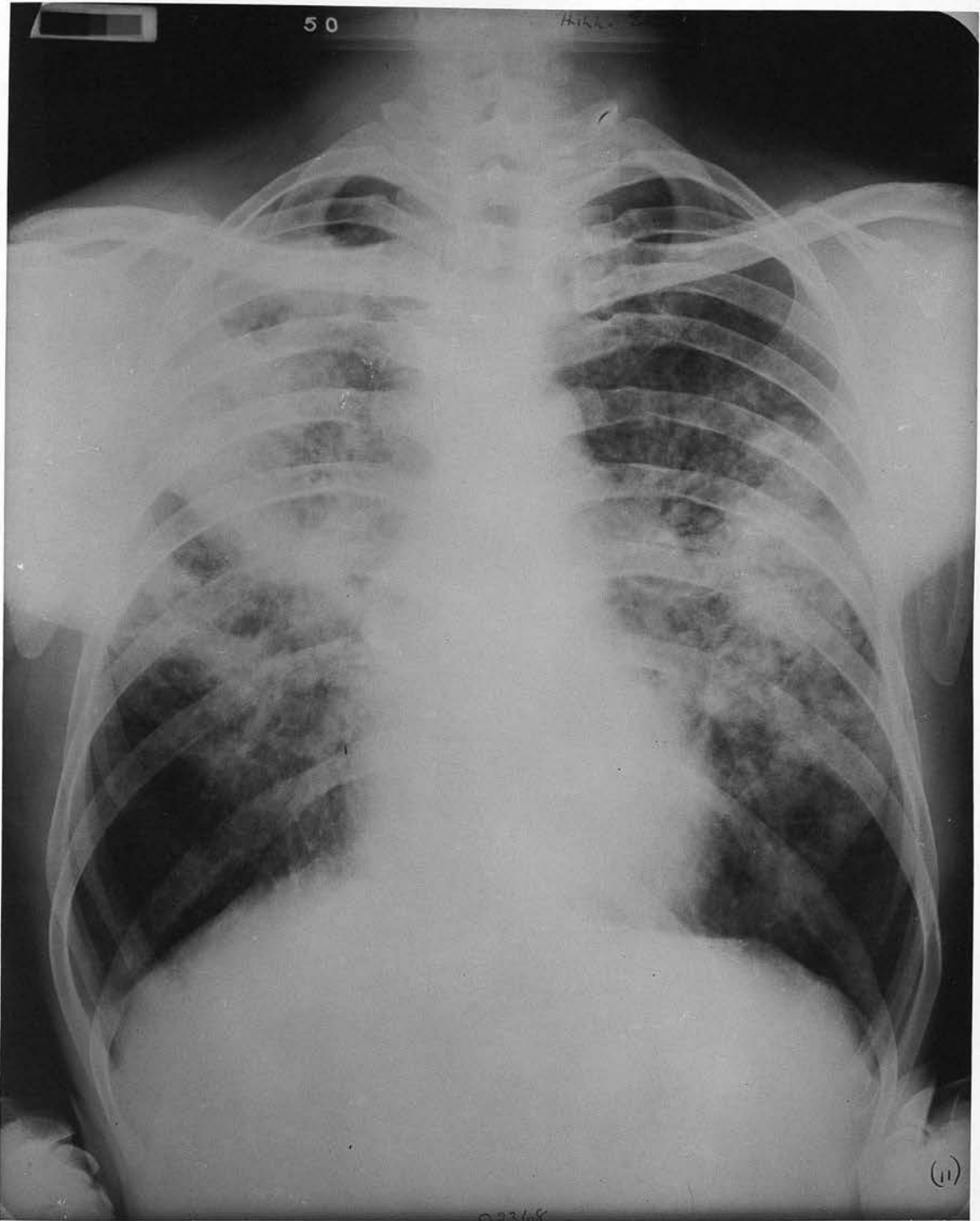


Figure 9.

Case No. 9. Bilateral massive fibrosis with cavitation in the upper part of the right lung. The sputum was positive for tubercle bacilli.



Case No. 25.Date of Birth: 1902.

This man worked underground for 25 years, almost exclusively on hard-heading work (rock work) but left the pits in 1940 when he was 38 years of age because of shortness of breath. He had pleurisy in 1945 and radiography in 1946 revealed extensive massive fibrosis with cavitation. His general condition began to deteriorate, the erythrocyte sedimentation rate was 21, the sputum was positive for tubercle bacilli and in 1947 by which time his general condition had worsened further, radiography demonstrated that the cavitation had become much more extensive (figs. 10 and 11). He died in that year following a sudden severe haemoptysis. This is a further example of the rapid deterioration so typical of patients with massive fibrosis and a positive sputum. No anti-tuberculous chemotherapy was available at the time.

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Figure 10.

Case No. 25. Radiograph in 1947 showing extensive shadows of massive fibrosis in both lungs and a large cavity with a fluid level in the right lung. The sputum was positive for tubercle bacilli.



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Figure 11.

Case No. 25. Lateral radiograph showing that the cavity lies posteriorly.



Case No. 27.

Date of Birth: 1905.

This man worked at the coal face of an anthracite colliery for 25 years. He was admitted to hospital in 1949 at the age of 46 for treatment of an attack of "bronchitis" associated with black sputum and a radiograph taken at that time (fig. 12) showed a large cavity in the right lung. It was noted in a radiograph taken 2 years later (fig. 13) that the cavity had "refilled". In October 1951 he was re-admitted to hospital with a further respiratory infection associated with copious black sputum and it was found that the cavity had emptied once more. A total of 28 specimens of sputum were negative on direct smear and 20 were negative on culture for tubercle bacilli. This case illustrates the way in which a cavity may empty and refill from time to time also that the resultant cavitation usually has the same shape as the pre-existing mass.

Figure 12.

Case No. 27. Massive fibrosis with bilateral cavities and fluid levels. The region of extreme translucency in the upper part of the left lung is the site of bullous emphysema. The sputum was negative for tubercle bacilli.

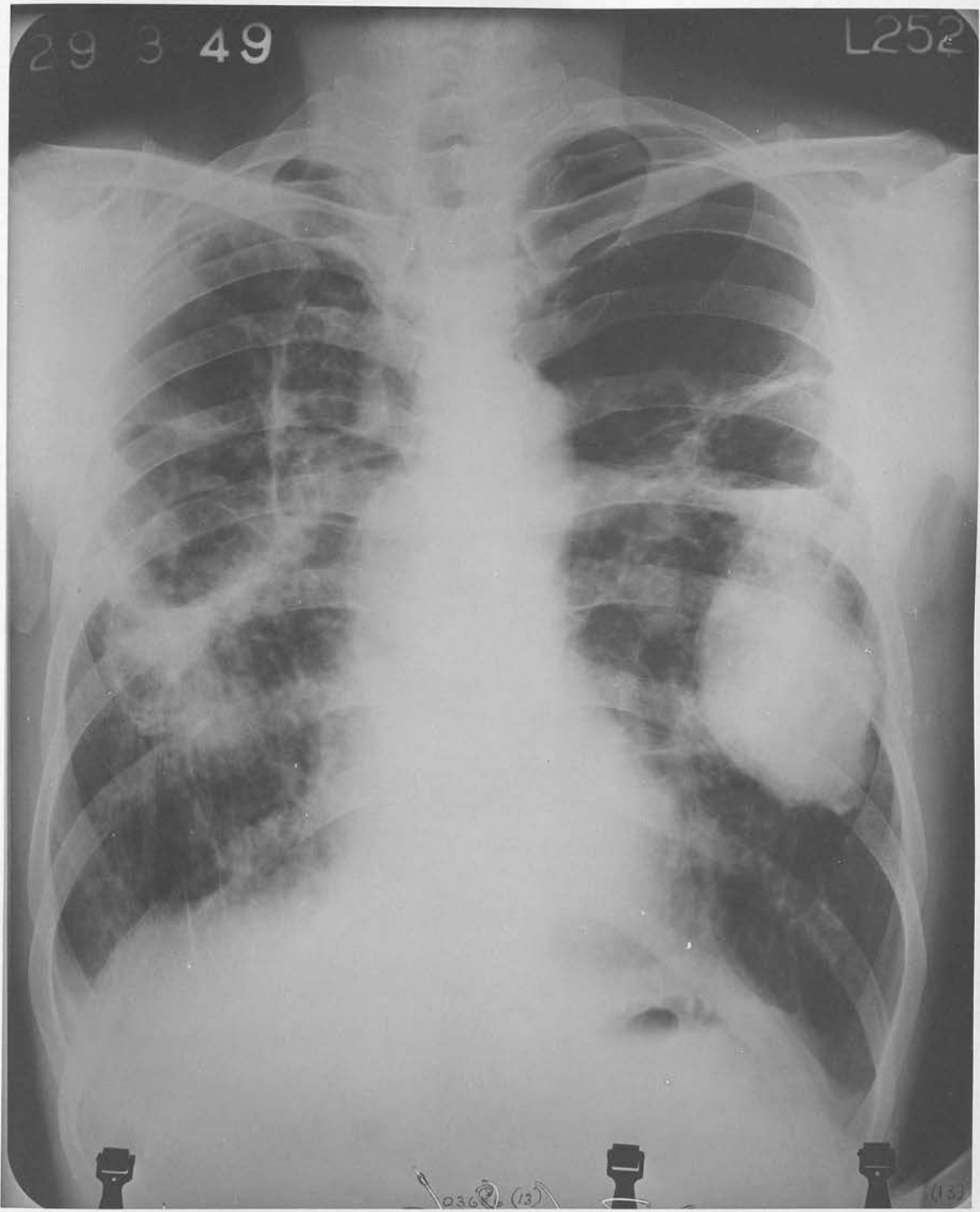
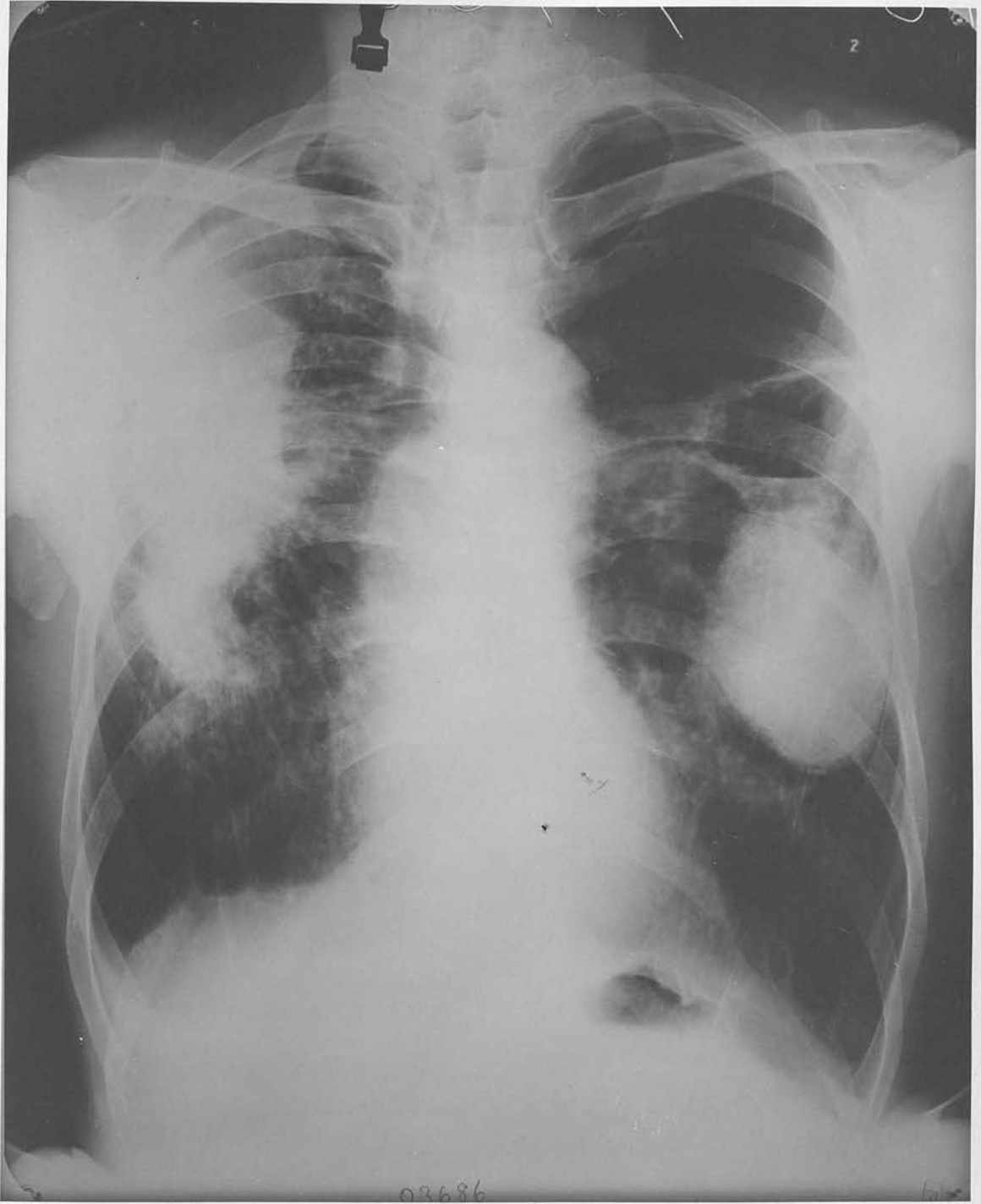


Figure 13.

Case No. 27. The cavity in the right lung has completely refilled. The appearances of the left lung are unchanged.



Case No. 29.

Date of Birth: 1907.

This man spent 19 years underground at the coal face. He elected to leave the pits in 1940 and worked thereafter in a factory. He first noticed dyspnoea on exertion in 1942 and expectorated black material in 1948 without any associated general symptoms. Between 1948 and the present time he has had repeated episodes of black expectoration, sometimes associated with haemoptysis, but has very little cough and is only slightly more breathless than he was in 1942. The erythrocyte sedimentation rate has varied between 20 and 60 mm./hour and the sputum has been repeatedly negative for tubercle bacilli.

Fig. 14 shows the radiograph taken in 1950 when he was 43 years of age revealing cavitated massive fibrosis in the right lung and a homogeneous opacity in the left lung. In the interval between the taking of this radiograph and the one in 1951 (figs. 15 and 16) a large quantity of black sputum was expectorated; this second radiograph shows a very thin-walled cavity in the left lung.

Figure 14.

Case No. 29. Bilateral massive fibrosis with a cavity
and a fluid level in the right lung.



Figure 15.

Case No. 29. Showing cavitation and fluid levels in
both lungs.

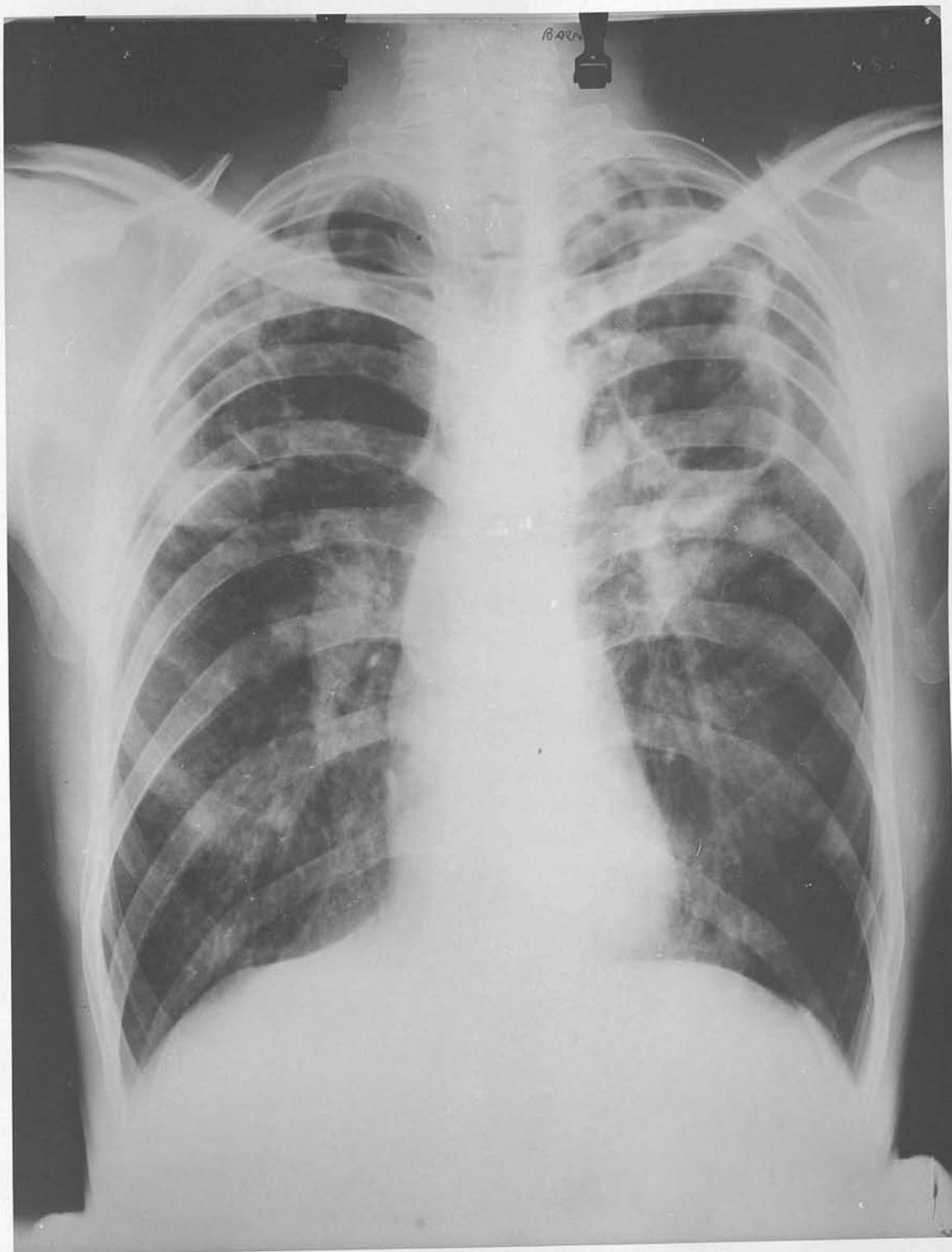
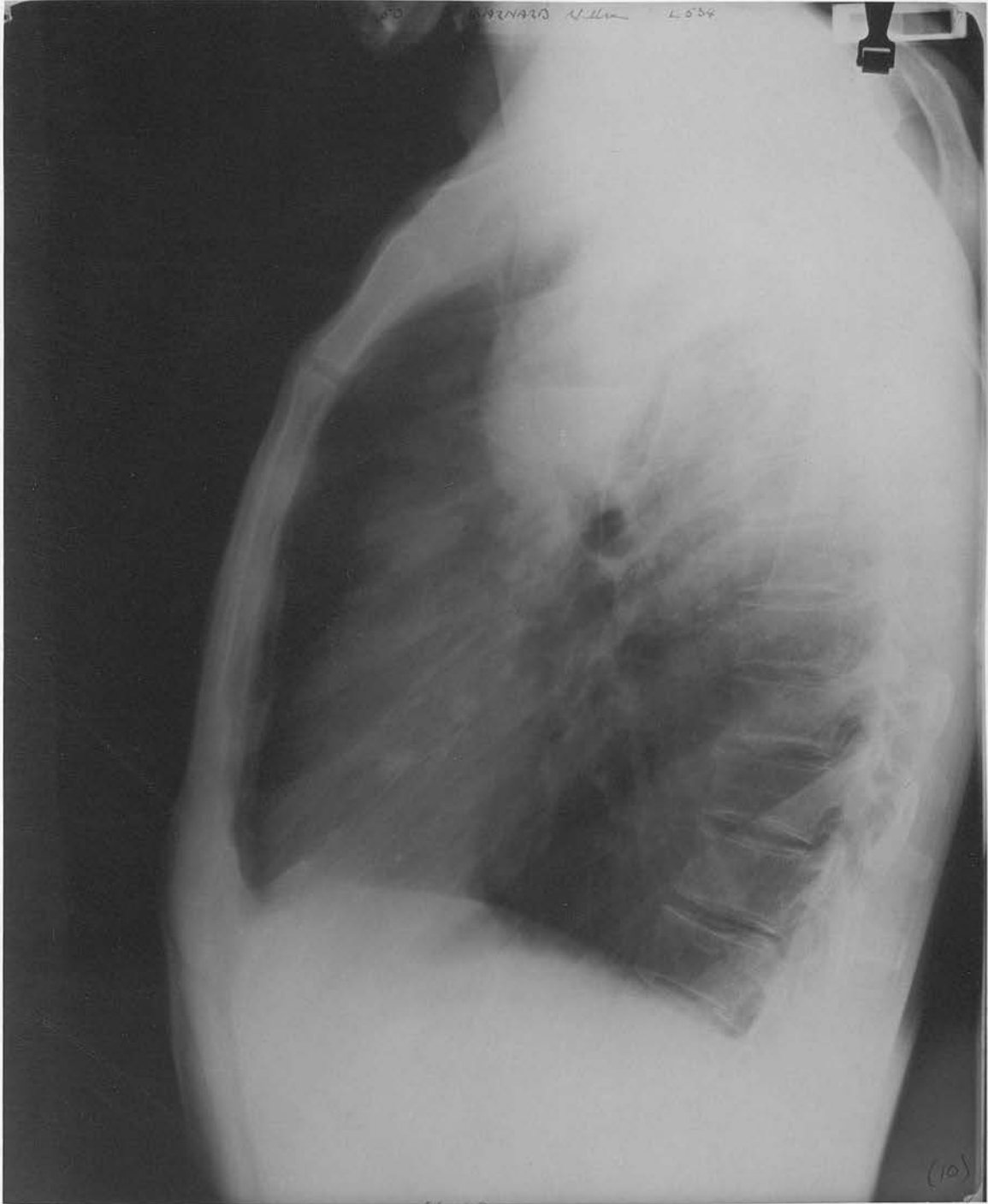


Figure 16.

Case No. 29. Lateral radiograph showing situation of cavity.



Case No. 36.

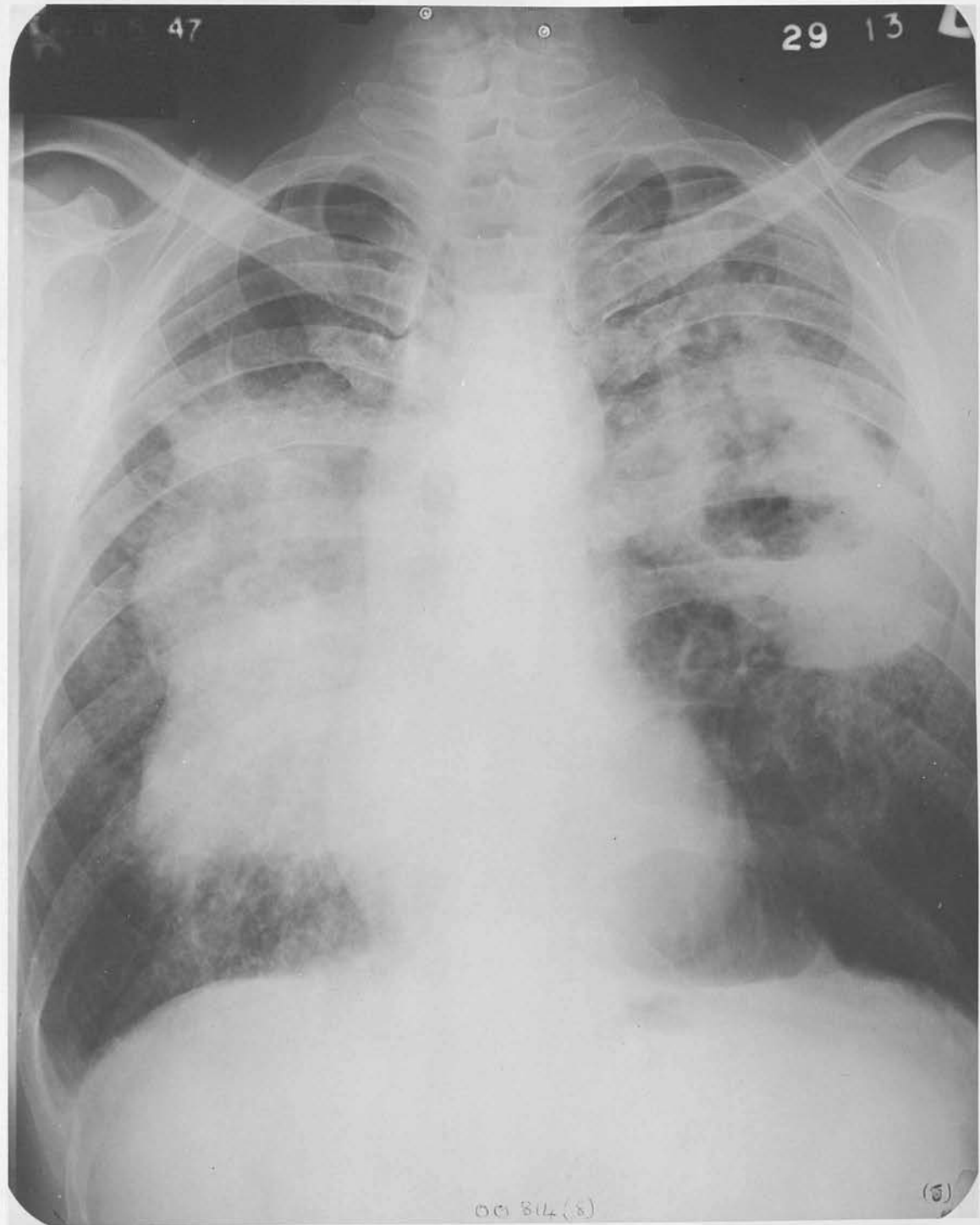
Date of Birth: 1889.

This man worked at the coal face in steam coal pits for 39 years. From 1940 he complained of increasing breathlessness on exertion, was certified as suffering from dust disease in 1942 and, under the regulations existing at that time, had to leave the mining industry. In 1946 he expectorated a large quantity of black sputum and radiography confirmed the presence of cavitated massive fibrosis in the right lung. Following this first episode, he expectorated black sputum at intervals, but on no occasion did he have any marked general symptoms although bronchial spasm became increasingly troublesome.

The cavities in the masses emptied and refilled from time to time as shown in figs. 17, 18 and 19. The erythrocyte sedimentation rate was generally elevated and the sputum persistently negative for tubercle bacilli. This man died in 1950 as a result of a strangulated hernia and a post-mortem examination confirmed the presence of extensive bilateral cavitated massive fibrosis with neither histological nor bacteriological evidence of tuberculosis.

Figure 17.

Case No. 36. Radiograph in 1947 showing bilateral massive fibrosis with cavitation in the left lung.



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Figure 18.

Case No. 36. Radiograph in 1948 showing extensive bilateral massive fibrosis without evidence of cavitation.

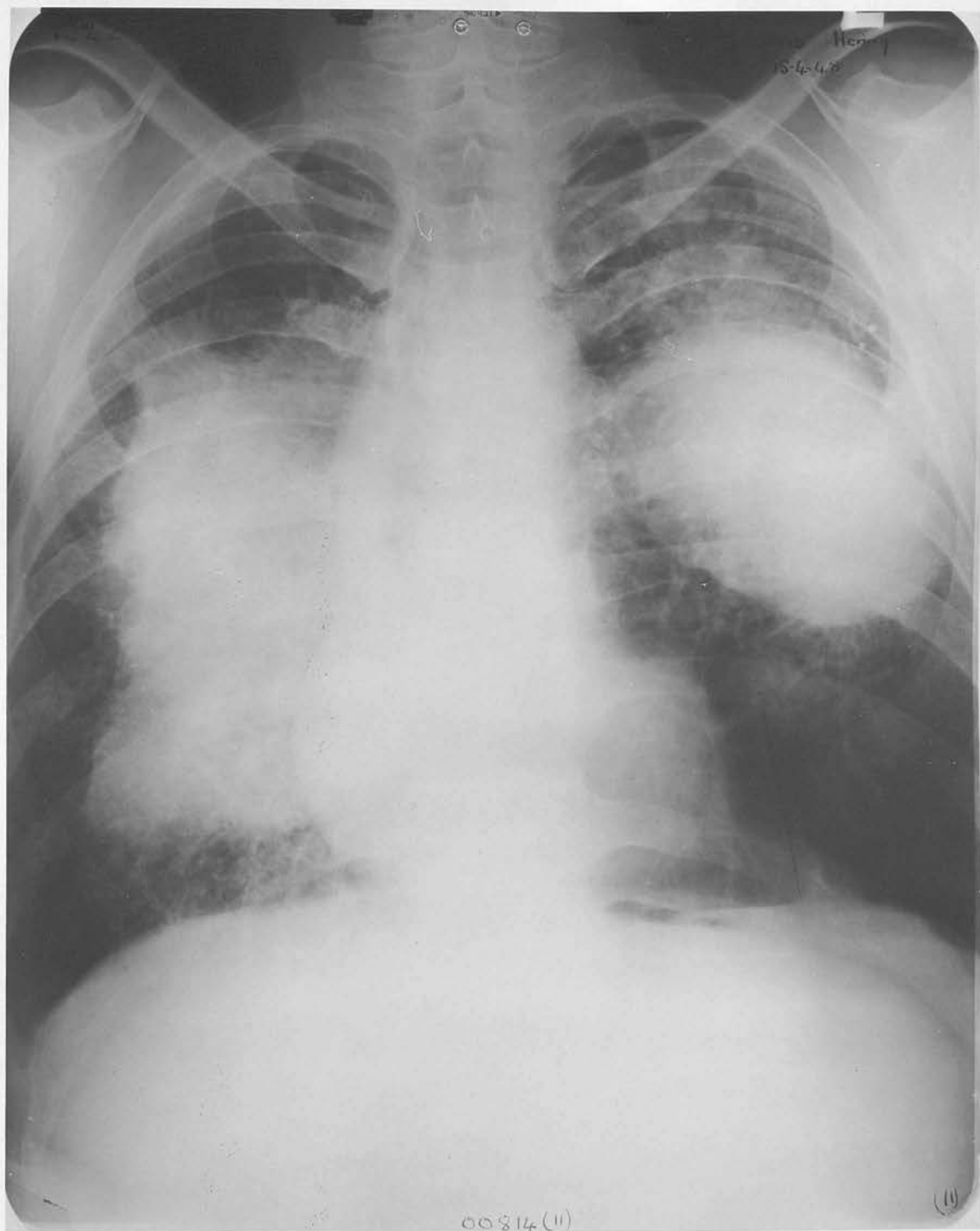
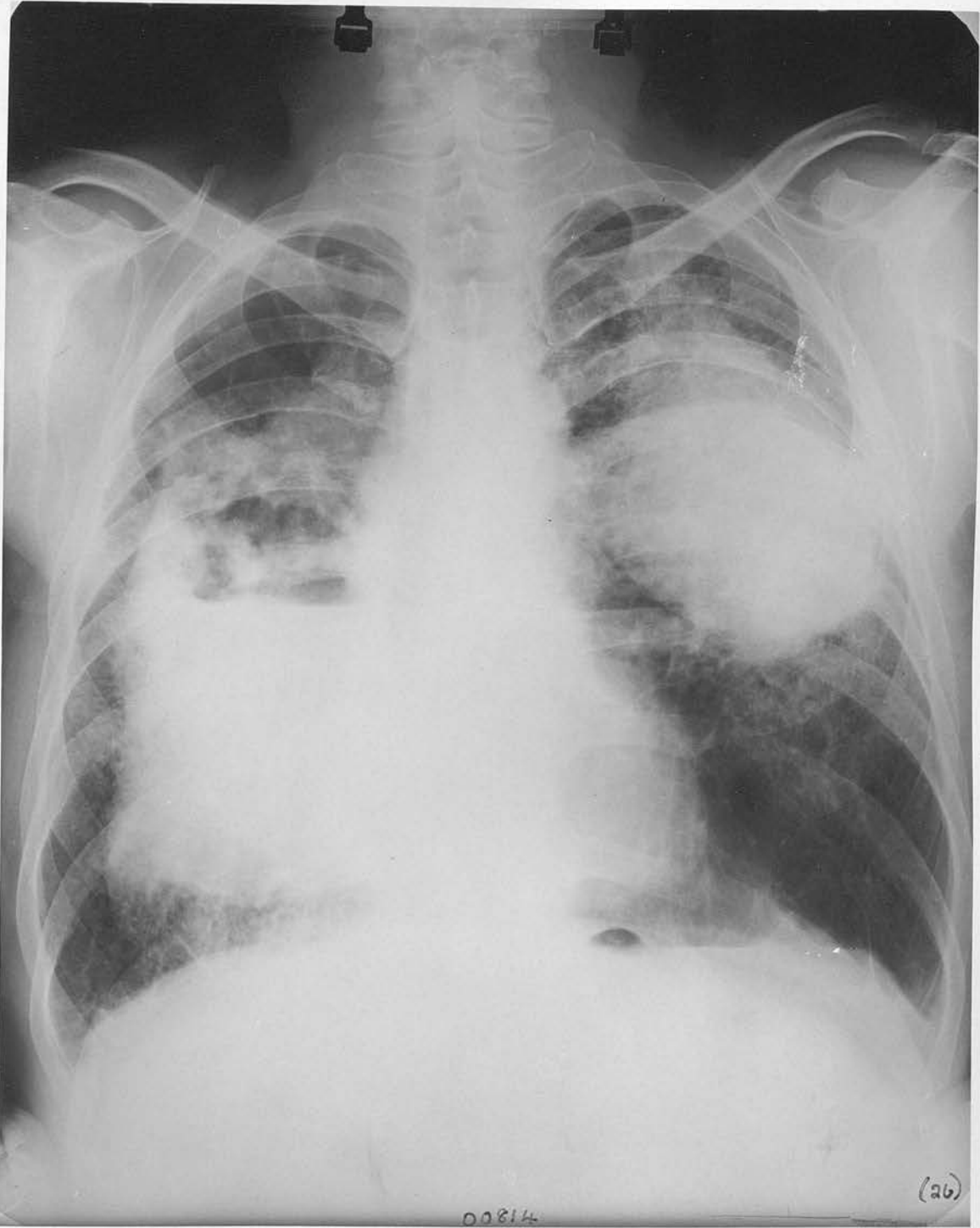


Figure 19.

Case No. 36. Radiograph in 1949 showing cavitated massive fibrosis with a fluid level in the right lung.



Case No. 50.Date of Birth: 1893.

This man spent 33 years underground in a steam coal pit, 10 years on the coal face and the remainder as a fireman. He noticed shortness of breath on exertion in 1939 when he was 46 years of age, applied to the, then, Silicosis Board, was certified as suffering from dust disease and had to leave the mining industry but found suitable work in a factory. He first noticed black sputum in 1940, the dyspnoea became gradually more marked and he had a severe acute respiratory infection in 1949 since which time he has been much more disabled. In January 1952 he had an episode of black expectoration associated with "bronchitis", but recovered sufficiently to be able to play a daily game of bowls. He noticed hoarseness in his voice in January 1952 and when seen ten months later it was confirmed by laryngoscopy that there was a left-sided laryngeal palsy, presumably as a result of pressure from, or distortion caused by, the masses near the left recurrent laryngeal nerve. Examination of the sputum has been repeatedly negative for tubercle bacilli. The radiograph taken in 1952 when he complained of hoarseness is shown in fig. 20.

Figure 20.

Case No. 50. Radiograph in 1952 showing extensive bilateral massive fibrosis and emphysema. There is a cavity with a fluid level in the left lung.



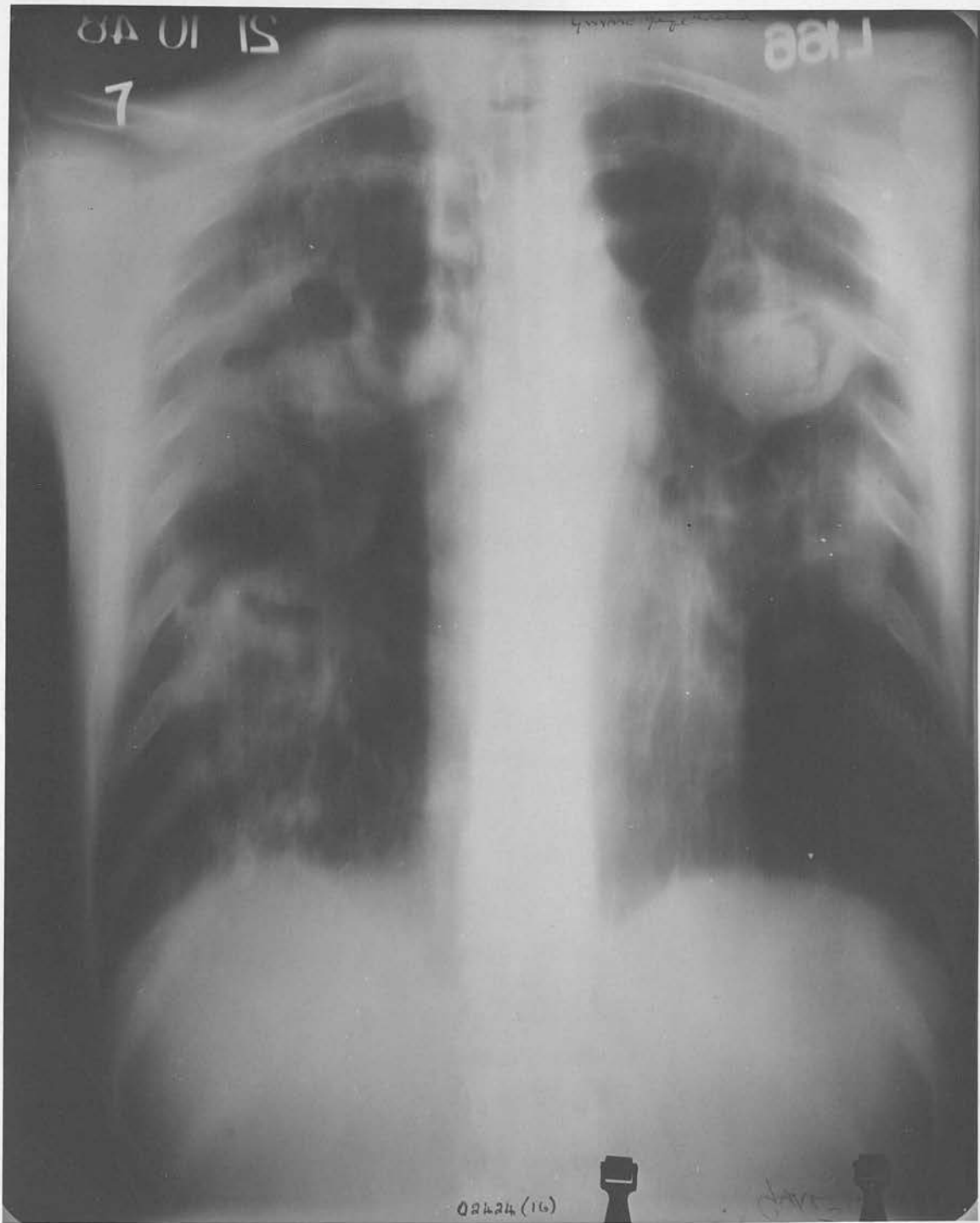
Case No. 55.

Date of Birth: 1911.

This case illustrates an uncommon finding. He was first examined in 1948 when he was 37 years of age as he had noticed that his sputum was black and that he was dyspnoeic on exertion. He had worked for 16 years at the coal face in an anthracite colliery and was certified as having dust disease by the Silicosis Board in 1942. Cavitation was visible in the postero-anterior film and tomography revealed an unusual appearance in the left lung (fig. 21), which was presumably a sequestrum of necrotic material lying within a cavity. The erythrocyte sedimentation rate has varied between 10-20 mm./hour. The patient remains well although unemployed. The sputum has been repeatedly negative for tubercle bacilli, and subsequent radiography has shown that the cavity has completely refilled.

Figure 21.

Case No. 55. Tomography showing an unusual appearance of sequestrum formation inside a cavity. The sputum was negative for tubercle bacilli.



have exhibited this phenomenon have usually full...

Case No. 58.

Date of Birth: 1906.

This man spent 23 years underground in a steam coal pit and following certification for dust disease in 1944, when he was 38 years of age, he started work in a factory. Fig. 22 shows the radiograph at that time. There is no obvious cavitation in the bilateral massive fibrosis. In 1947 he began to expectorate considerable quantities of black sputum and three months later a radiograph showed that the large mass in the right lung had completely cavitated (fig. 23). He was admitted to hospital when the sputum was examined and found to be negative for tubercle bacilli. Three weeks after admission he complained of chest pain, developed fever and had an haemoptysis. Treatment with penicillin and sulphonamide resulted in a fall in temperature and an improvement in the general condition. The erythrocyte sedimentation rate fell from 70 mm./hour on admission to 30 mm./hour on discharge.

A film taken in 1949 (fig. 24) showed that the cavity had apparently completely closed and this was confirmed by tomography. Cavity closure is a rare occurrence in massive fibrosis but the few patients who have exhibited this phenomenon have generally felt better

following its occurrence although black sputum may continue to be expectorated from other cavities.

In 1949 he was re-admitted to the ward with a febrile illness associated with an irritating cough, black sputum and pain in the right side of the chest, the erythrocyte sedimentation rate was 110 mm./hour, pleural friction was present on the right side and fig. 25 shows a radiological appearance suggesting that a new massive fibrotic lesion had formed where none had been previously. As a result of penicillin therapy the temperature subsided, the general condition improved and a month later (fig. 26) the radiological opacity had partially cleared leaving a small residual cavity seen in the tomogram (fig. 27). In view of the rapid appearance and disappearance of this shadow it is considered that it represented a pneumonic consolidation which subsequently broke down and which was not massive fibrosis. He made a good recovery and since then, apart from a few small haemoptyses, has remained well and is working in a Remploy factory. The sputum has been repeatedly negative for tubercle bacilli and the present appearances of the lungs are shown in fig. 28.

Figure 22.

Case No. 58. Bilateral massive fibrosis without obvious cavitation (1944).

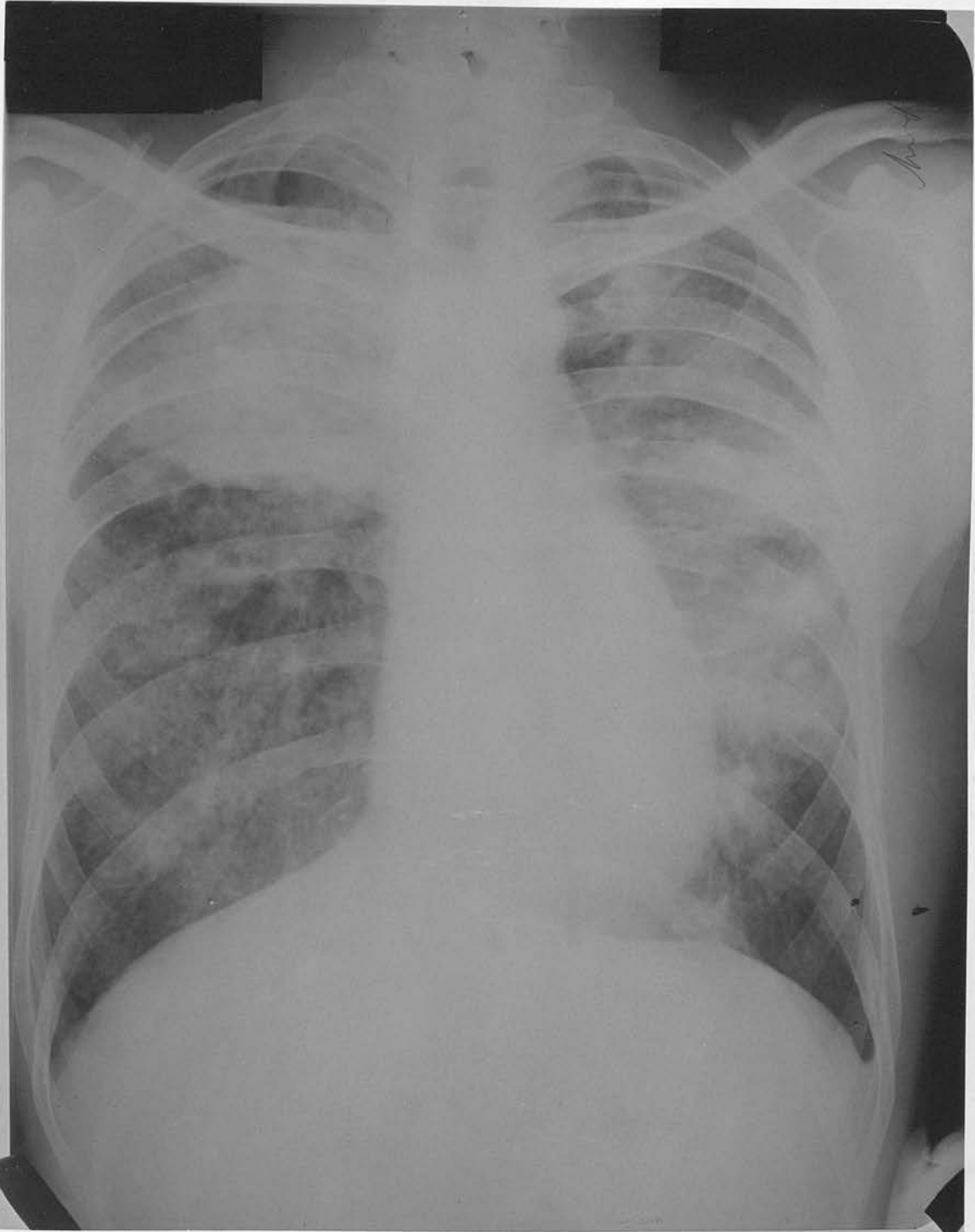
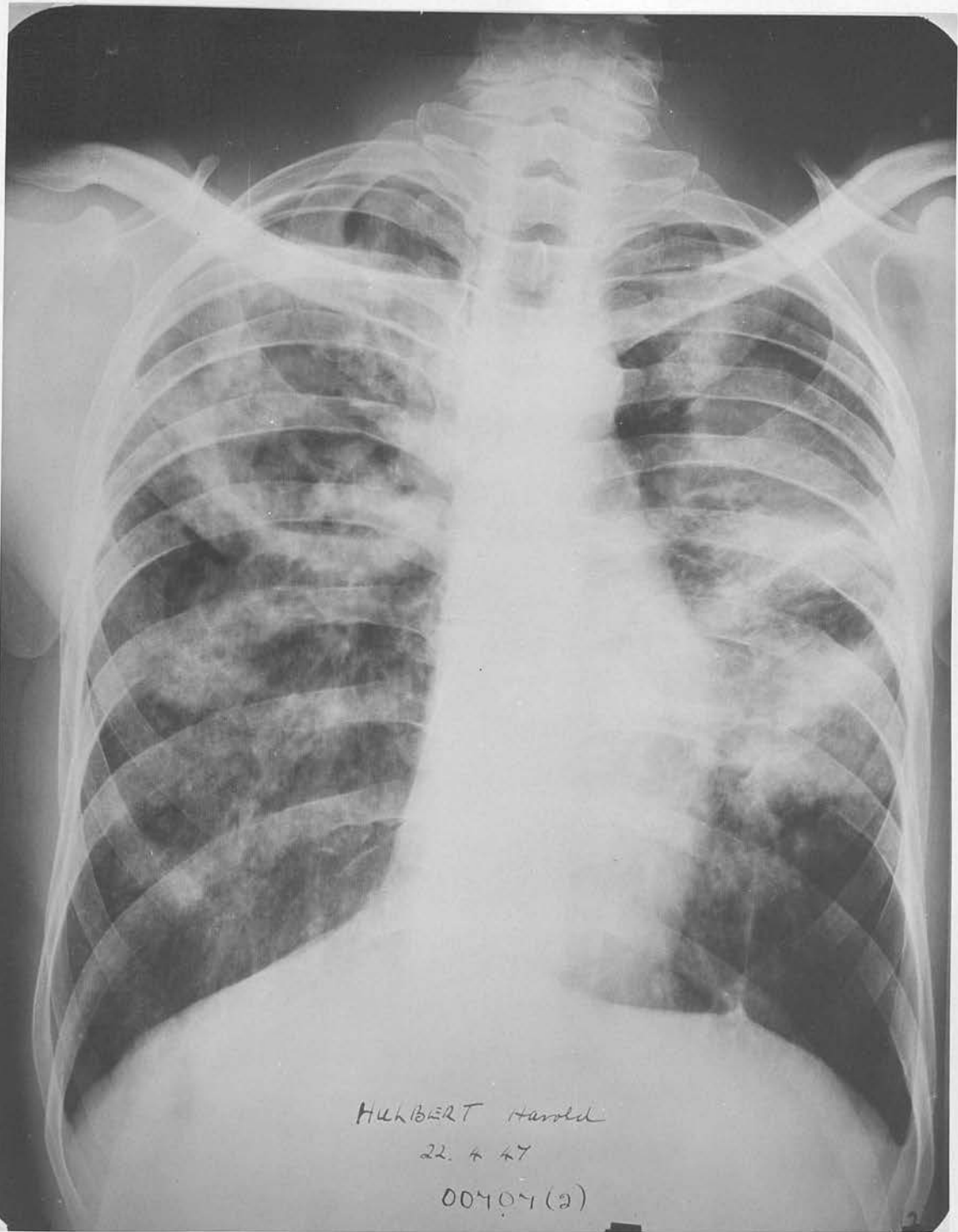


Figure 23.

Case No. 58. Radiograph 3 years later (1947) showing
cavitation in the right lung.



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Figure 24.

Case No. 58. Radiograph in May 1948 showing almost complete closure of the large cavity seen in the previous film.

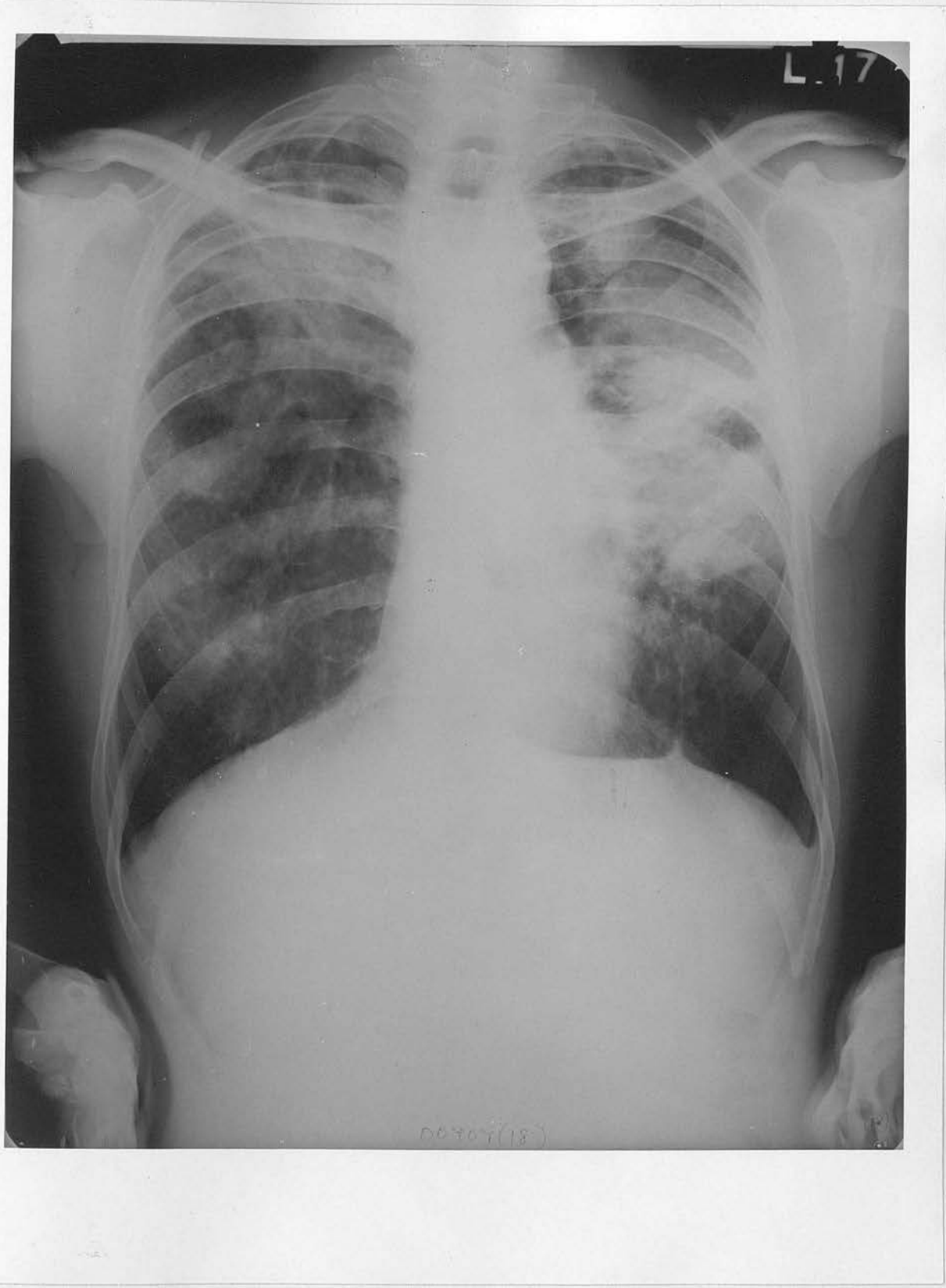


Figure 25.

Case No. 58. Radiograph in February 1949 revealing a new opacity in the right lung at first suggestive of a massive lesion.



Figure 26.

Case No. 58. Radiograph taken in March 1949 showing cavitation in this fresh opacity.

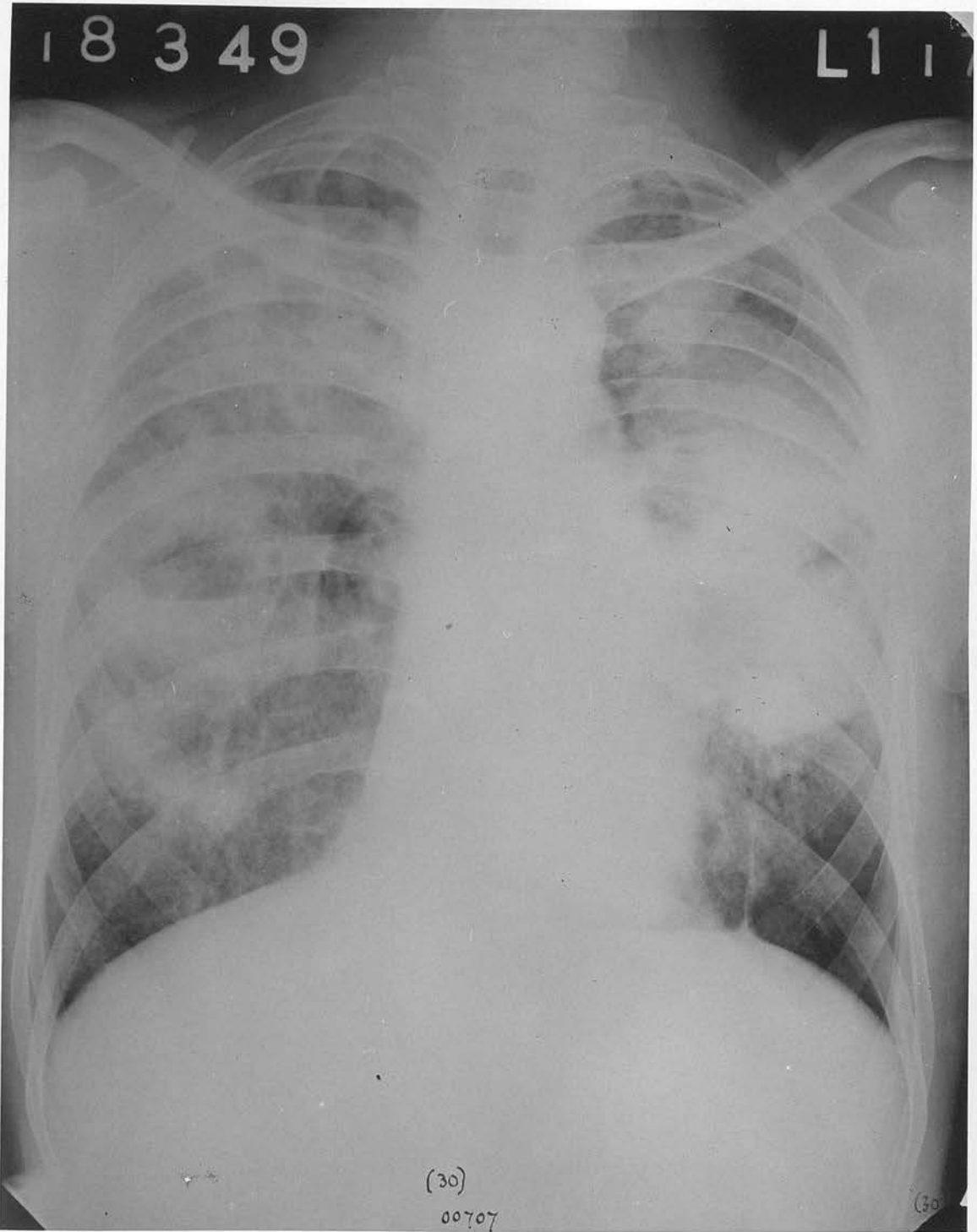


Figure 27.

Case No. 58. Tomogram in May 1949 showing reduction in the size of the persisting cavity. Cavitation of the massive fibrosis in the left lung is now apparent.

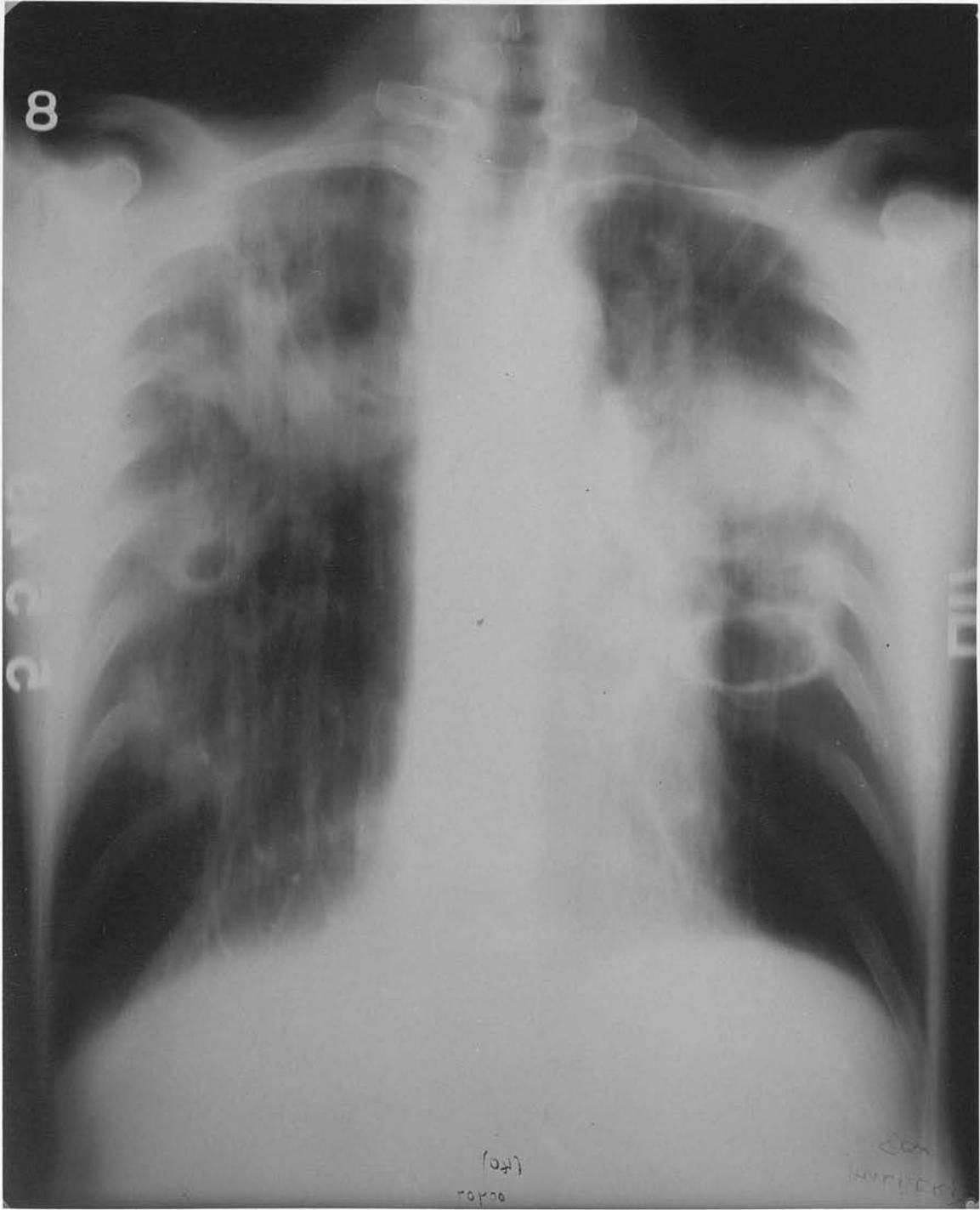
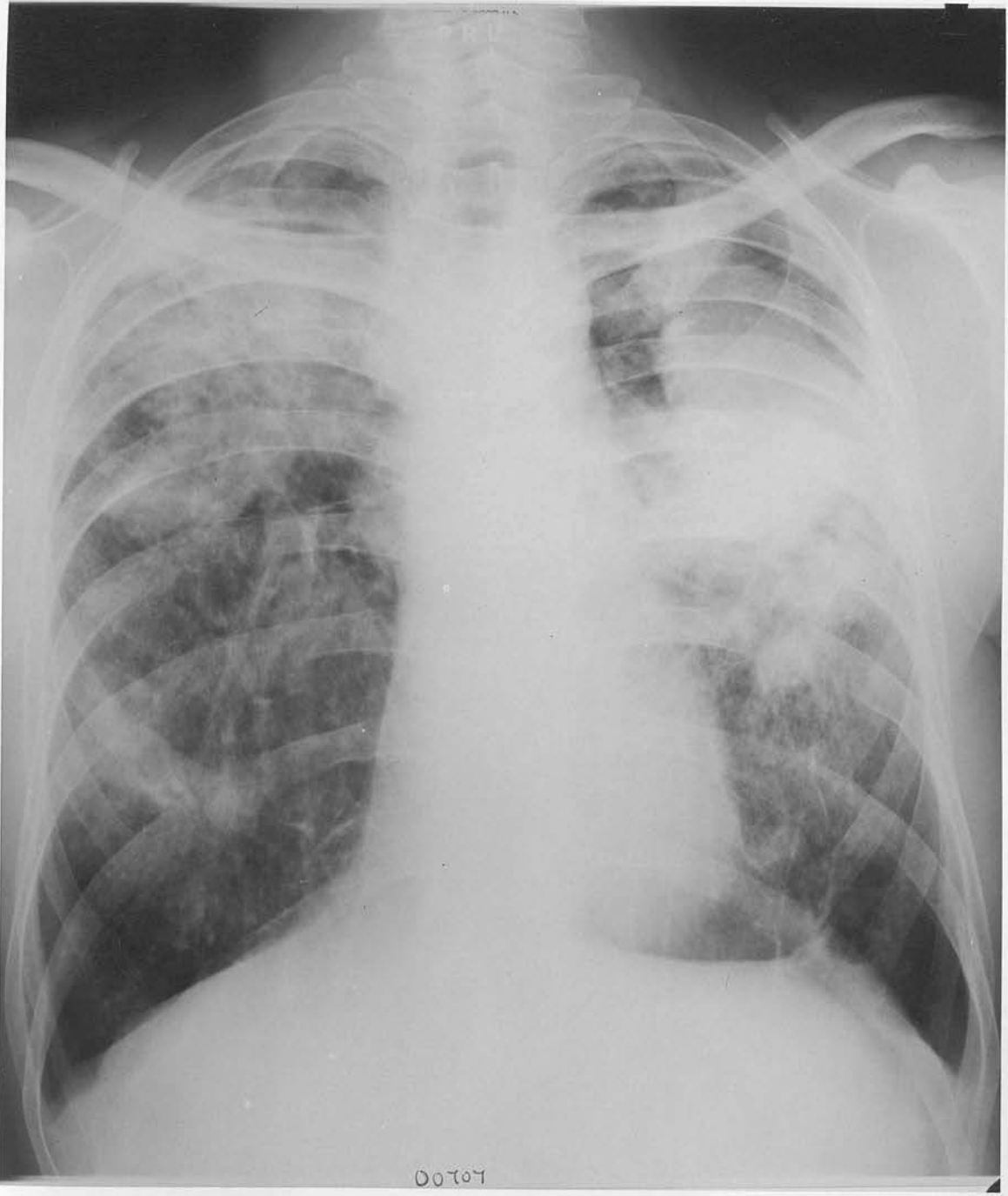


Figure 28.

Case No. 58. Radiograph taken in 1952 showing present state of the lungs.



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Case No. 73.

Date of Birth: 1906.

This man worked for 21 years at the coal face of a very dusty steam coal pit. He first noticed breathlessness on exertion and fatigue in 1941 when he was 35 years of age, applied to the Silicosis Board, was certified as having dust disease and left underground work. At that time radiography showed that he had simple pneumoconiosis with diffuse early massive fibrosis. He began to expectorate black sputum from time to time and was first seen at the Pneumoconiosis Research Unit in 1946 when it was found that moderately extensive massive fibrosis was present. In 1948 he had an acute respiratory illness associated with copious black sputum and cavitation was confirmed radiologically in the left lung. In 1949 following an haemoptysis, a radiograph showed no obvious cavitation in the left lung but tomography revealed what looked like a sequestrum lying within the cavity. The man's general condition improved considerably and he was able to undertake light gardening work. Examination of the sputum on direct smear, culture and animal inoculation at that time failed to reveal acid-fast bacilli.

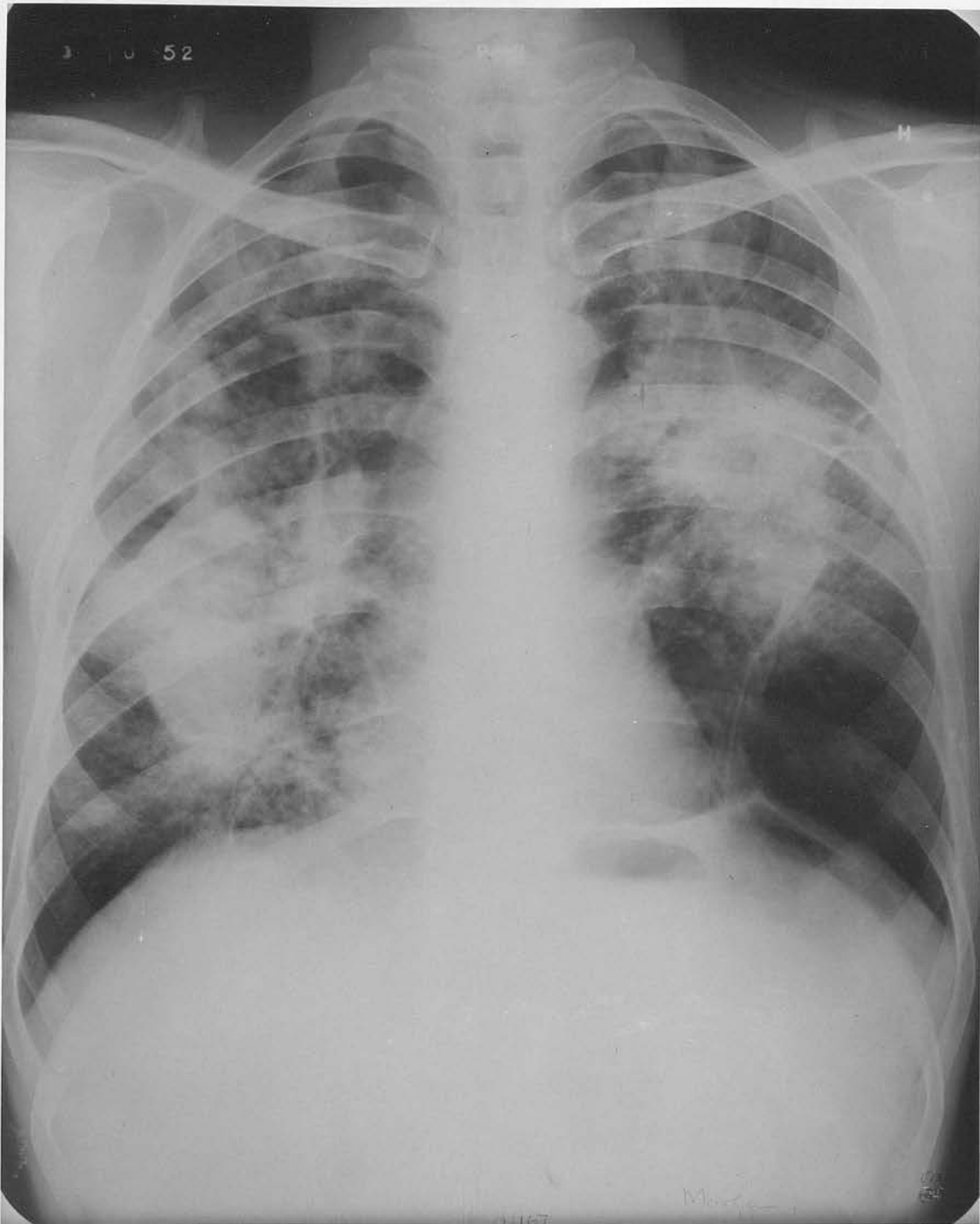
In 1951 following a further episode of black expectoration, cavitation of the mass in the right lung was discovered. On examination of the sputum, acid-fast bacilli were found on direct smear but these bacilli were not morphologically typical of tubercle bacilli, no growth was found on culture and animal inoculation was negative. Because of the finding of acid-fast bacilli, and before the culture results were known, it was decided to give a course of streptomycin and para-amino salicylic acid. This was continued for 3 months but acid-fast bacilli continued to appear in the sputum and both culture and animal inoculation tests were again negative. No marked change occurred in the man's general condition and the erythrocyte sedimentation rate remained at over 50 mm./hour and was frequently as high as 115 mm./hour. Later in 1952, he had a respiratory infection, again with black expectoration, noticed swelling of the ankles and electrocardiography gave evidence of right ventricular hypertrophy. Non-pathogenic acid-fast bacilli persisted in the sputum. He responded fairly well to penicillin therapy and digoxin, the radiograph taken in 1952 being shown in fig. 29.

This is an example of a relatively young man

who has extensive massive fibrosis with frequent episodes of black expectoration in whom non-pathogenic acid-fast bacilli have been found on repeated occasions.

Figure 29.

Case No. 73. Radiograph taken in 1952 showing extensive bilateral massive fibrosis with cavitation in the right lung.



Case No. 76.

Date of Birth: 1910.

At the age of 14 this man went underground in steam coal pits where he worked for a total of 18 years at the coal face and on the conveyors but he left the pits because of nystagmus. Massive fibrosis with cavitation was discovered when he was admitted to the ward in 1948 at the age of 38. (figs. 30 and 31). In 1950 a routine radiograph taken elsewhere led to his being referred to a chest clinic when he was advised to give up his job and rest at home because he was presumed to be suffering from open pulmonary tuberculosis. At this time he was feeling well and repeated examination of the sputum failed to reveal tubercle bacilli. This is an example of a man who suffered loss of employment owing to a misunderstanding of the significance of cavitation in massive fibrosis. He remains well at the present time, the sputum is persistently negative for tubercle bacilli and a radiograph taken in 1952 is shown in fig. 32.

Figure 30.

Case No. 76. Radiograph taken in 1948 showing bilateral massive fibrosis. In the mass in the upper part of the right lung cavitation is suggested but is not obvious.

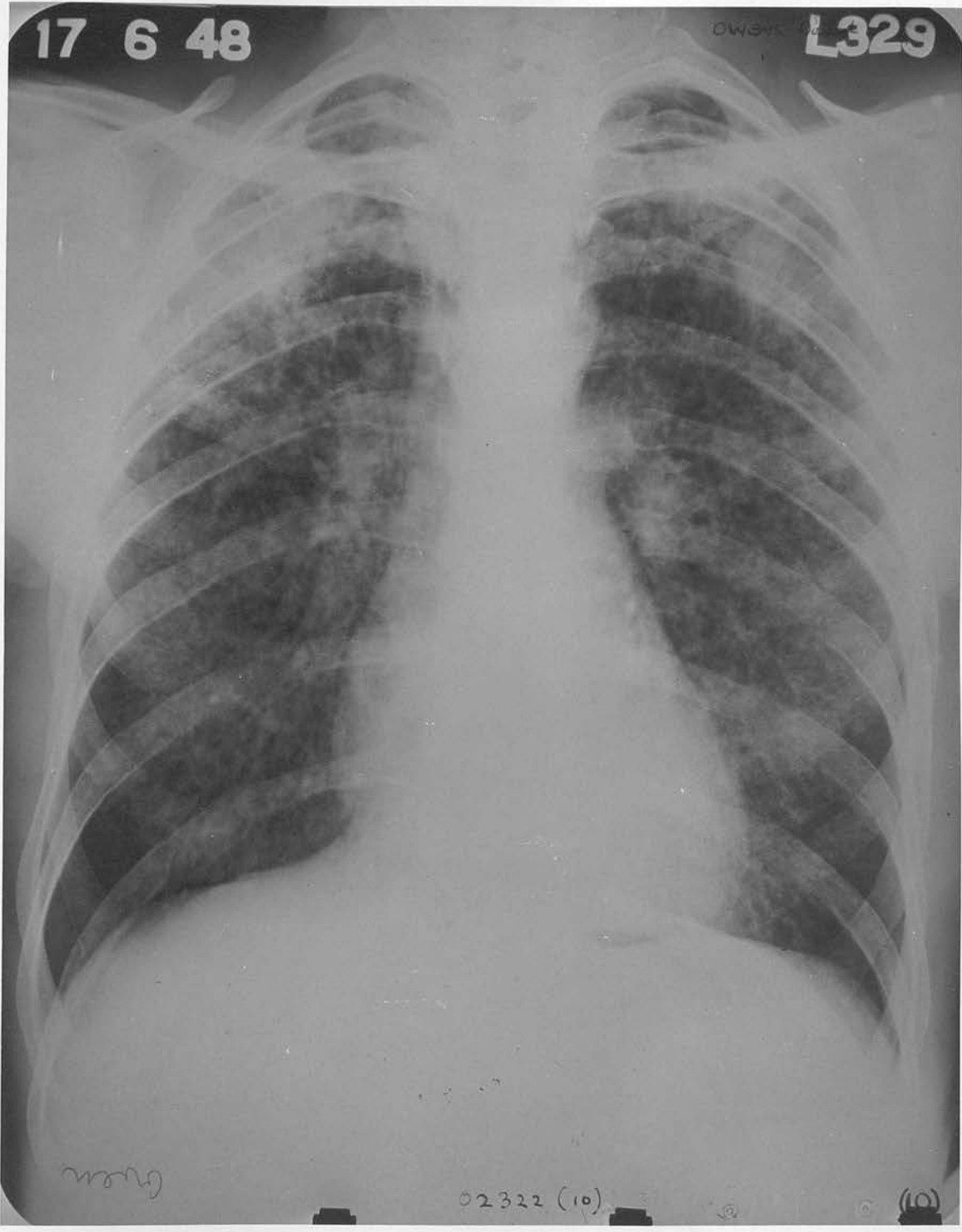


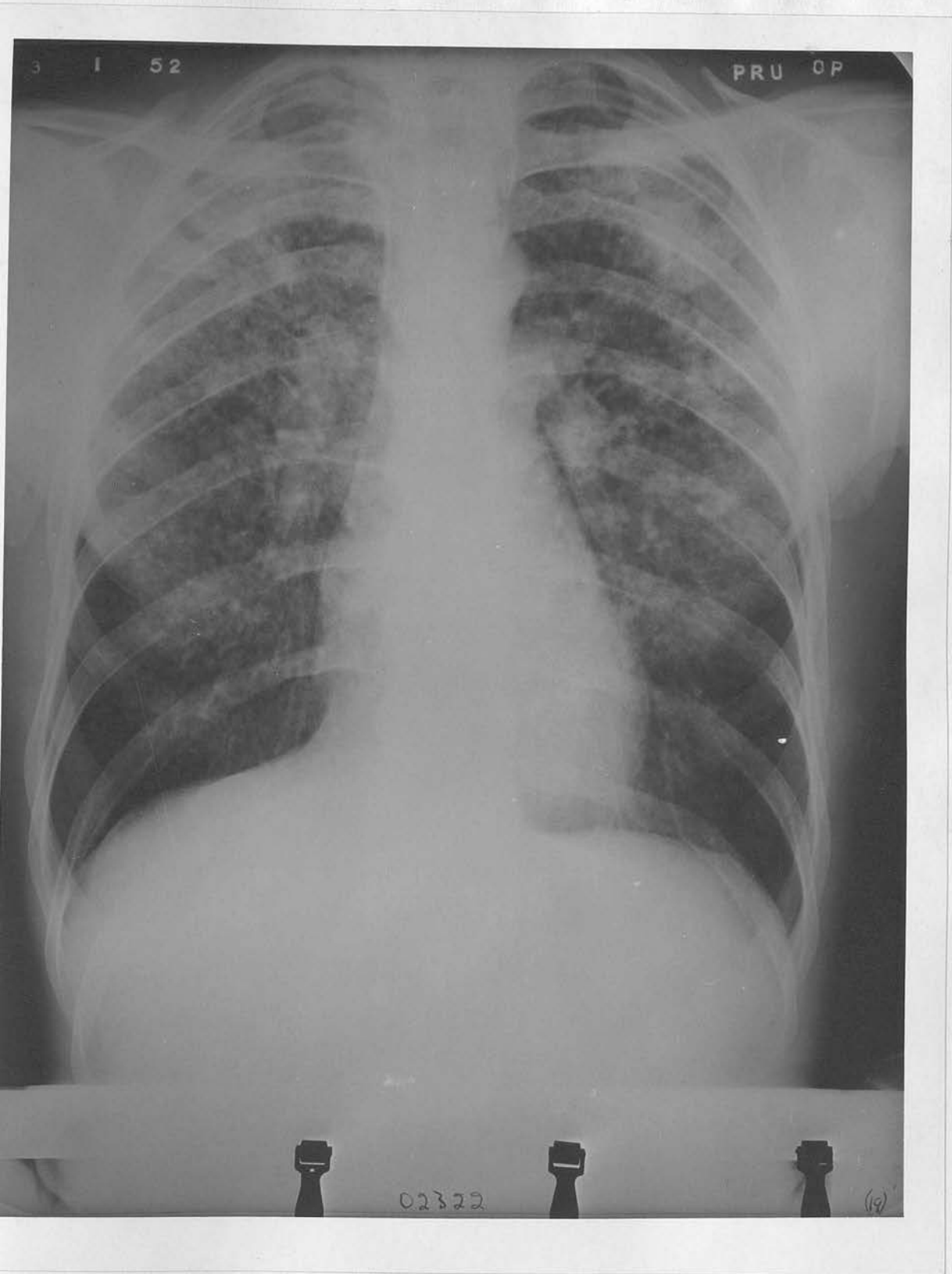
Figure 31.

Case No. 76. Tomography confirms the presence of cavitation; the cavity wall is thick and irregular. The existence of cavitation was an incidental finding at this time and repeated examinations of the sputa have been negative for tubercle bacilli.



Figure 32.

Case No. 76. Radiograph taken in 1952 suggests that cavity closure has occurred and this was confirmed by tomography.



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Case No. 81.Date of Birth: 1893.

This man was 54 years of age when seen in the ward in 1947 (fig. 33). He had worked for 30 years in a steam coal colliery, mostly at the coal face, but had spent 2 years drilling rock. Extensive bilateral cavitated massive fibrosis was first discovered in 1946 but there was no history of black sputum to indicate when it had occurred. He is still able to lead a quiet life and the cavities have remained more or less the same although they have emptied and refilled from time to time. Between 1946 and 1949 he had very little systemic upset but since that time his general condition has slowly deteriorated and dyspnoea has increased. The erythrocyte sedimentation rate is usually above 50 mm./hour, and the sputum has been repeatedly negative for tubercle bacilli on direct examination, culture and animal inoculation. Fig. 34 is a radiograph taken in 1952. - This case illustrates that a man can live for many years despite extensive cavitation.

Figure 33.

Case No. 81. Extensive bilateral multiple cavitation of massive lesions in 1947. The sputum has been repeatedly negative for tubercle bacilli.

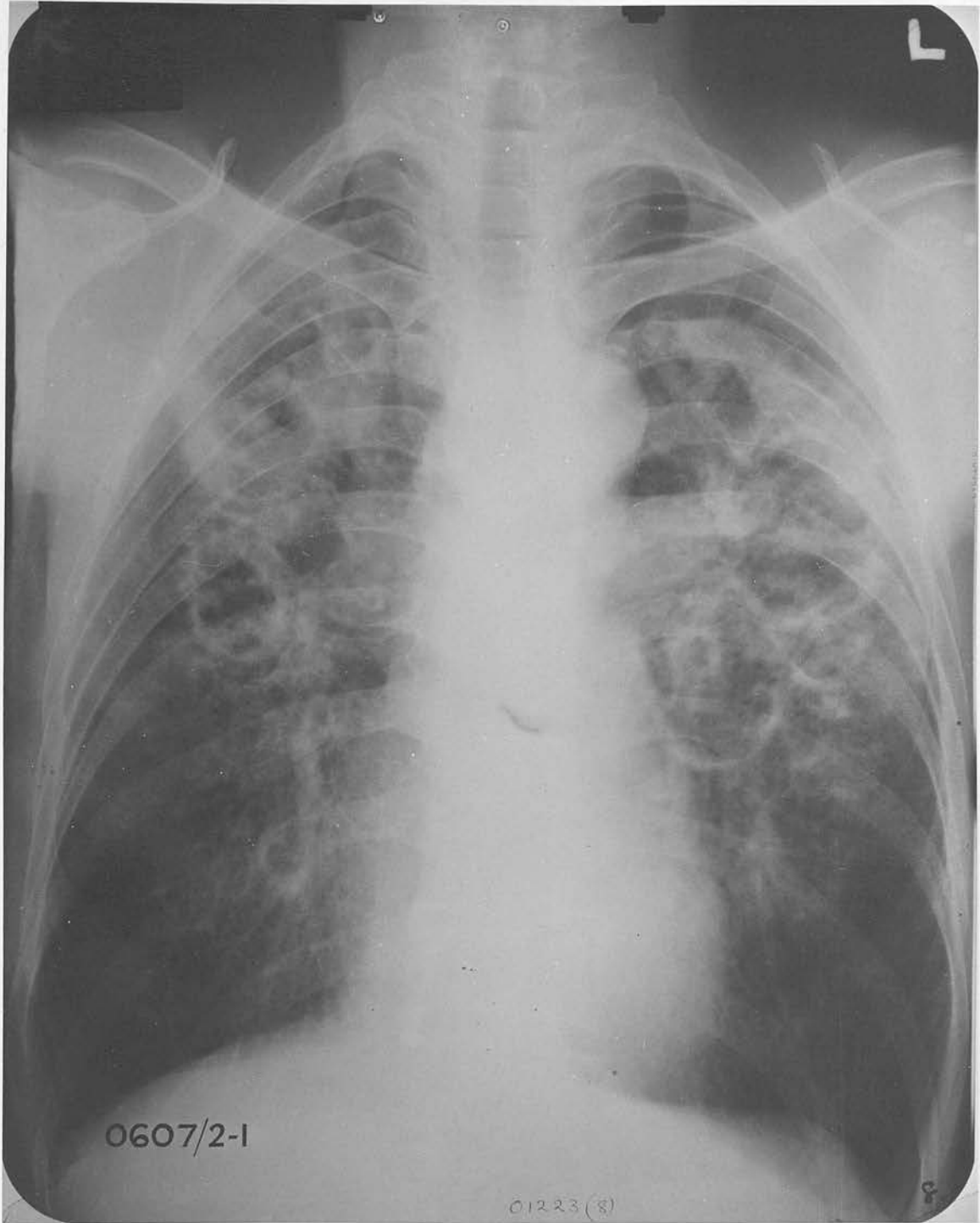


Figure 34.

Case No. 81. Radiograph taken in 1952 showing extensive cavitation in both lungs with multiple fluid levels.



Case No. 91.Date of Birth: 1902.

This man spent 17 years in steam coal pits working at the coal face, but left in 1933 because of dyspepsia. He was first admitted to hospital in 1951 when he gave a history of having had copious black sputum for 4 weeks and breathlessness on exertion for several years. Fig. 35 shows the radiological appearances at the time of admission, and as a result of being in hospital where postural drainage was carried out the black sputum subsided. The erythrocyte sedimentation rate varied between 70 and 120 mm./hour, and the sputum was negative for tubercle bacilli. He was re-admitted two months later as the cough was more marked, the sputum purulent, even more copious and on this occasion foul smelling. Again, no tubercle bacilli were isolated, the sputum containing only mixed organisms with no one type predominating; no fungi were found. The erythrocyte sedimentation rate remained between 40 and 100 mm./hour, and as a result of treatment by large doses of penicillin and streptomycin the sputum became less purulent. Radiography, however, showed no change in the appearance of the cavity and in 1952, tomography (fig. 36) showed that the cavity

persisted although the patient was feeling well. Bronchography (fig. 37) was carried out but the opaque medium did not enter the cavity and no bronchiectasis was demonstrated in the remainder of the lung. This case has therefore behaved clinically as a chronic lung abscess being the only one in the series in which secondary infection has apparently occurred in a cavitated massive lesion, and is also the only case who showed marked finger clubbing.

Figure 35.

Case No. 91. Bilateral massive shadowing with a large cavity in the left lung and a fluid level.

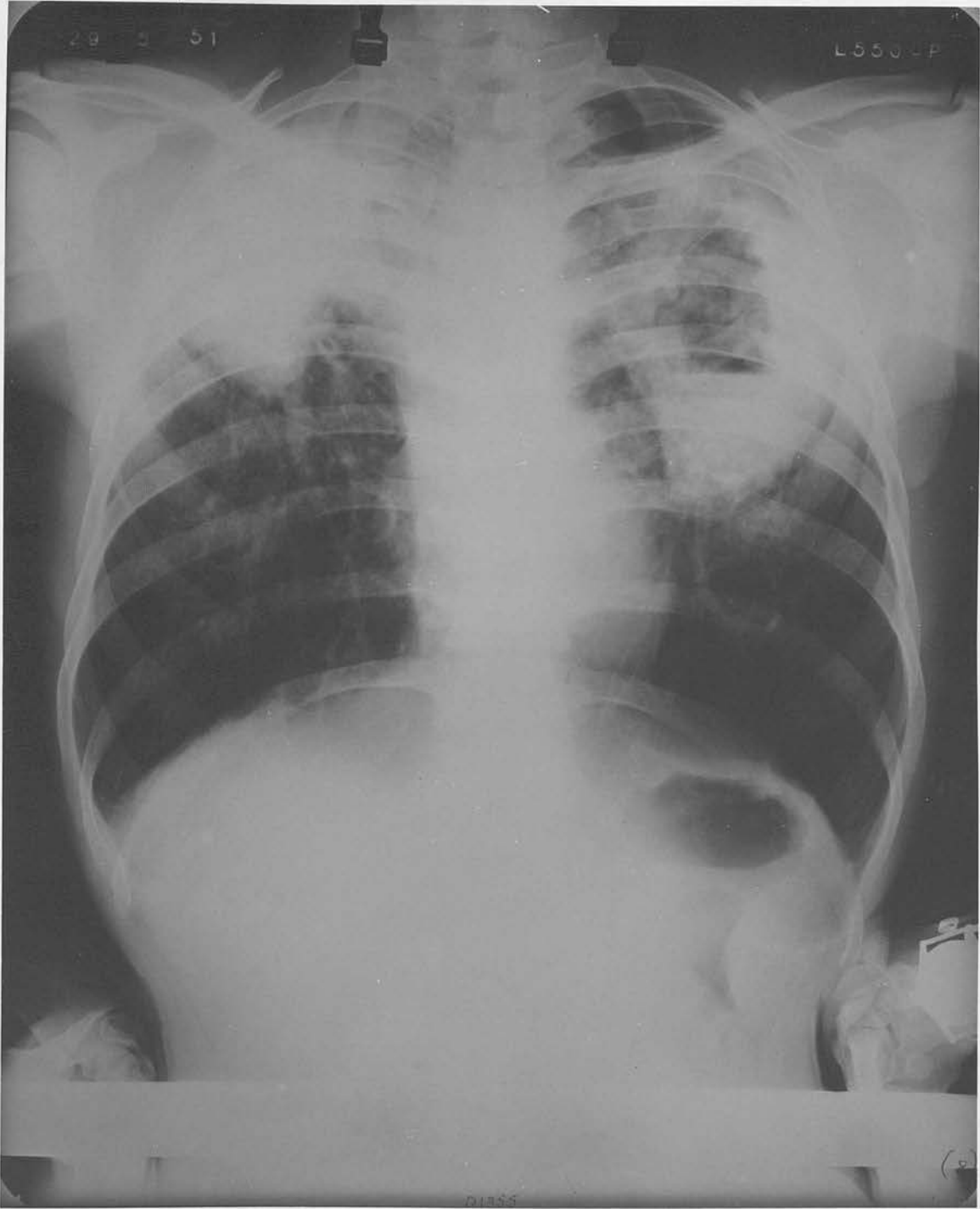


Figure 36.

Case No. 91. Tomography more than a year later shows that the cavity is still present. The sputum was negative for tubercle bacilli.

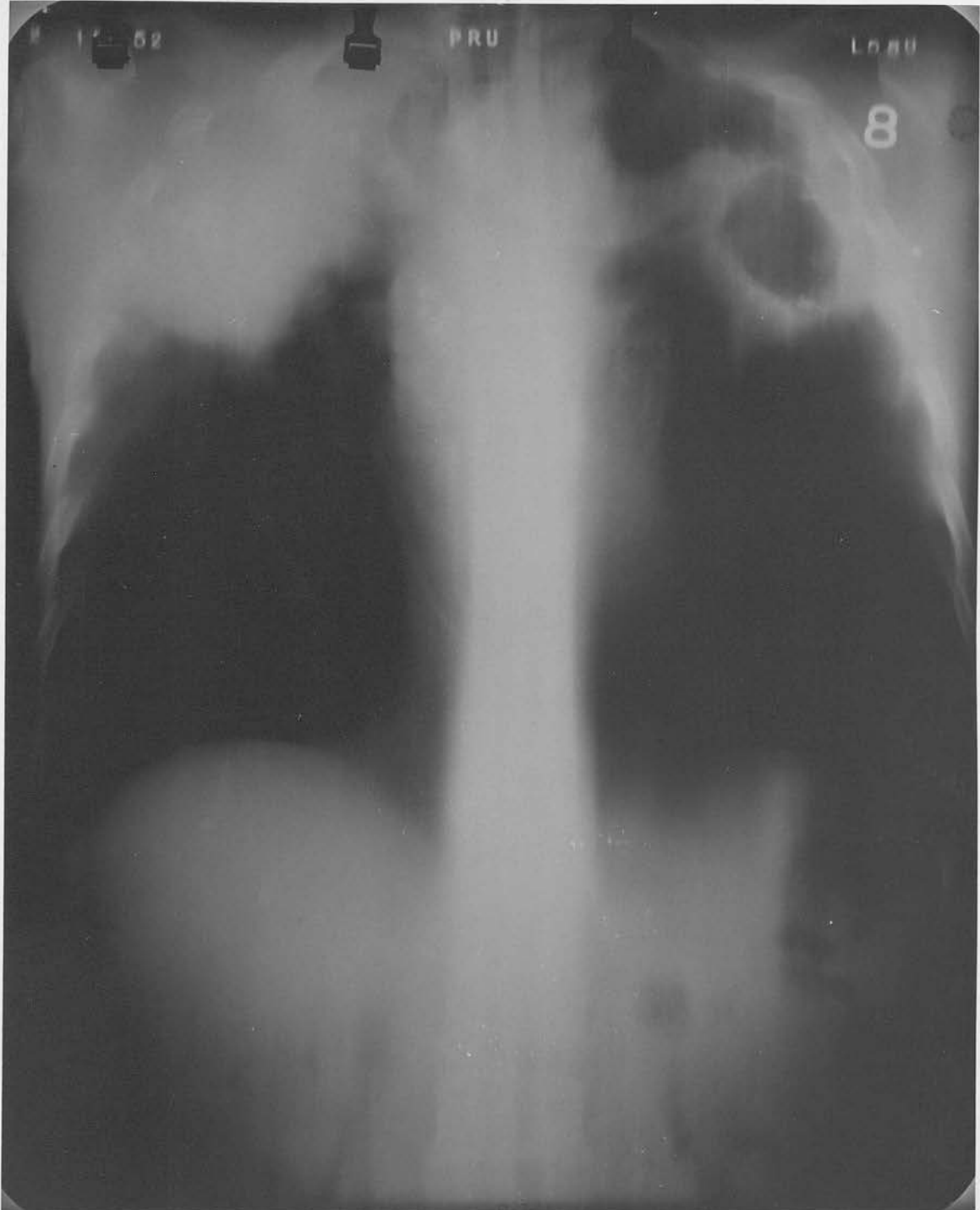
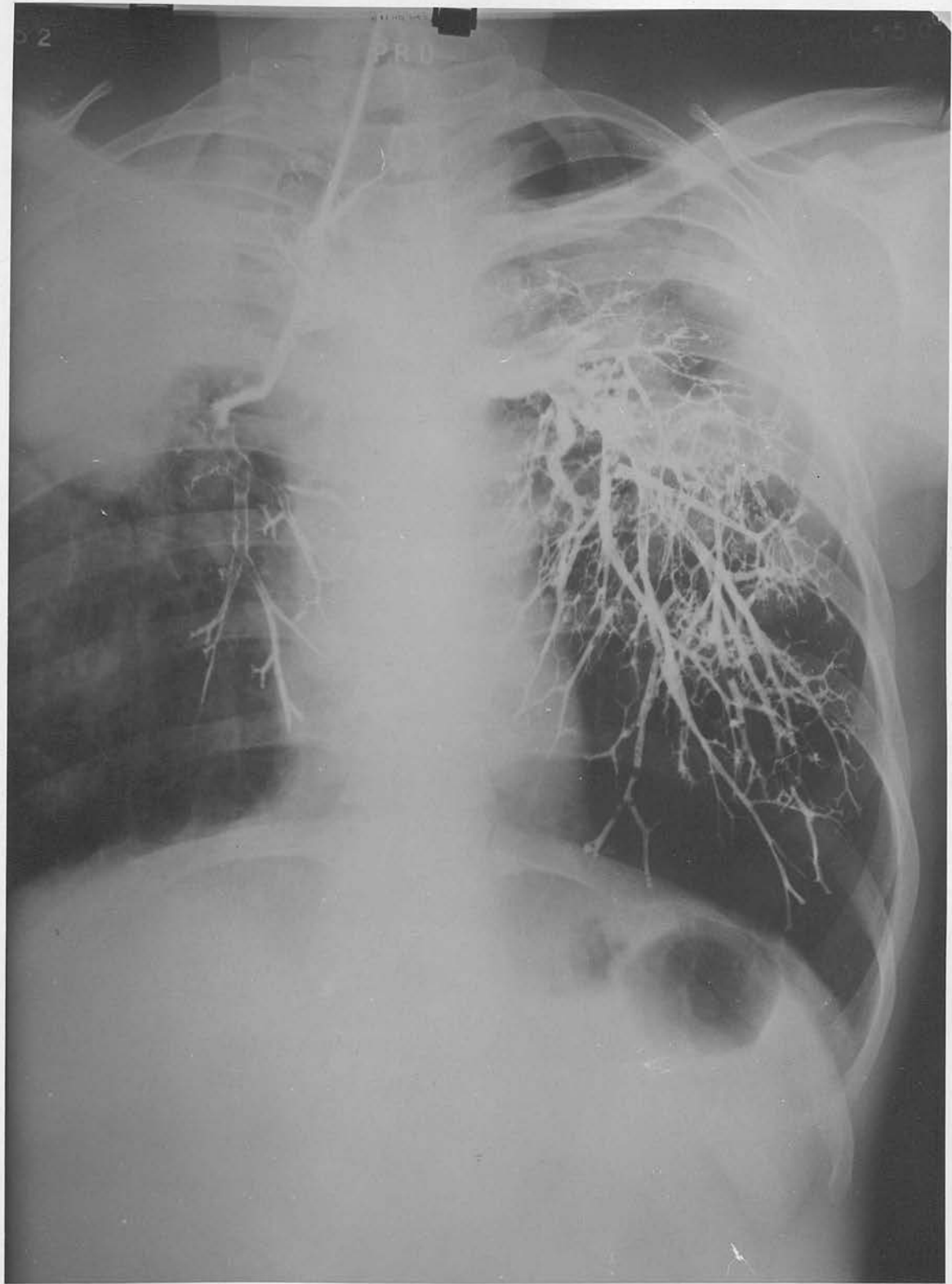


Figure 37.

Case No. 91. Bronchography shows the radio-opaque medium (aqueous 'Dionosil') approaching, but not entering the cavity. Considerable distortion of the remainder of the bronchial tree is evident but no bronchiectasis is visible.



Case No. 106.

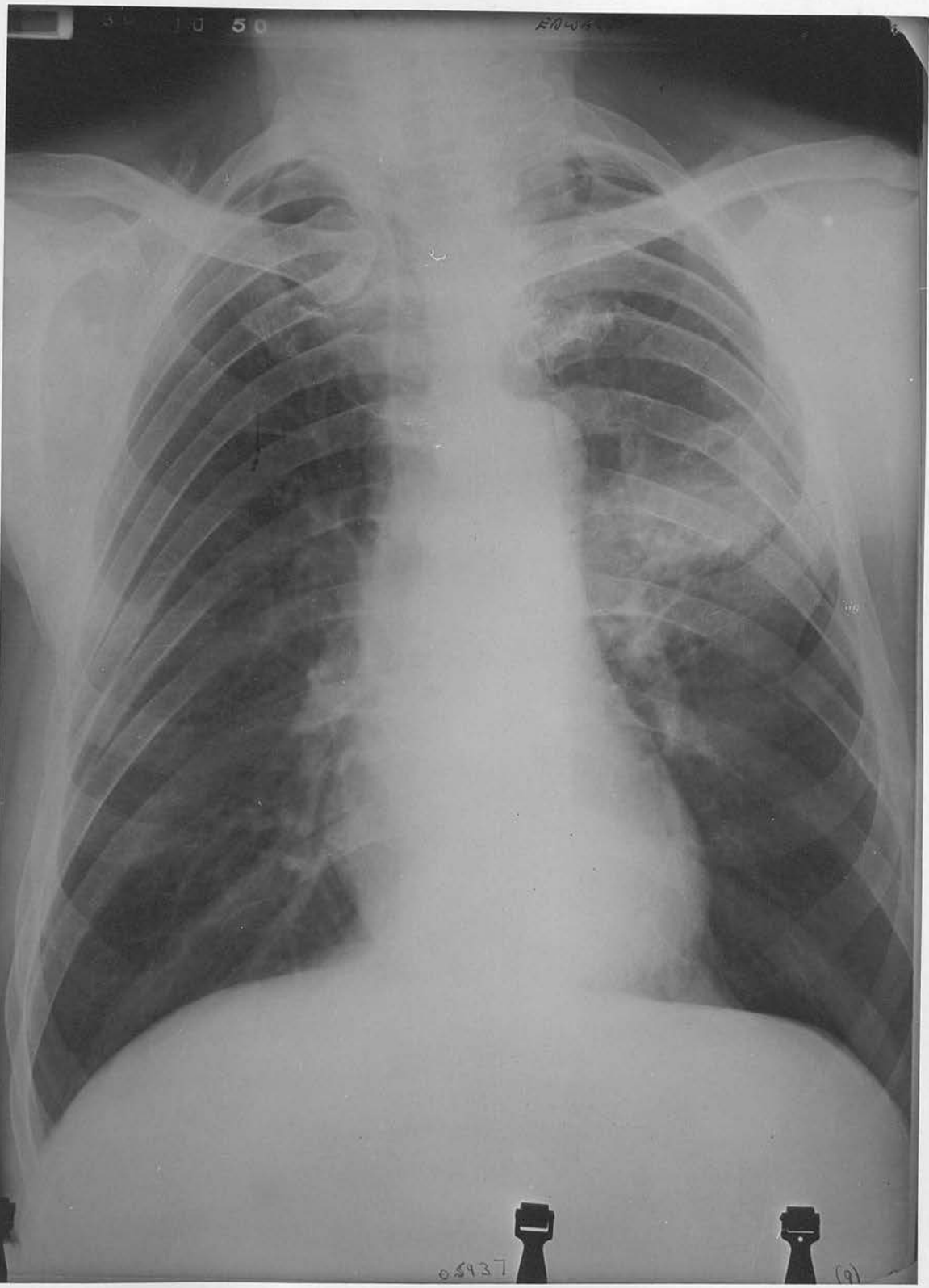
Date of Birth: 1880.

This man had worked underground for 43 years, 22 of them at the coal face and 21 on haulage. He was 70 years of age when the radiograph in fig. 38 was taken. This was a routine film taken during the Rhondda Fach Survey and the patient had no complaint except slight dyspnoea on exertion which he attributed to his age. The erythrocyte sedimentation rate was 50 mm./hour and the sputum negative for tubercle bacilli. It was considered that the most likely diagnosis was a bronchial carcinoma but that surgical treatment was not feasible.

The man died 5 months later, and, at autopsy, carcinomatous masses were found in both lungs, together with the coal foci of simple pneumoconiosis. Histological examination of the tumour showed it to be a very anaplastic carcinoma. Careful scrutiny of the film in fig. 38 shows that the simple pneumoconiosis is barely category 1. The opacity therefore cannot represent massive fibrosis.

Figure 38.

Case No. 106. Radiograph taken in 1950 showing an opacity in the left lung with a cavity. The background of simple pneumoconiosis is faint in the reproduction.



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Case No. 107.Date of Birth: 1897.

This man was underground for 37 years in various types of work. In 1943 when he was 46 years of age he noticed breathlessness on exertion and was found to have pneumoconiosis on radiological examination. The dyspnoea gradually increased and during the year prior to admission to hospital he noticed more cough and some blood stained sputum. For six weeks before admission he had fever and general malaise. He was found to be an ill and wasted man with a severe productive cough, the sputum being positive for tubercle bacilli on direct smear examination. He was febrile, the erythrocyte sedimentation rate was 50 mm./hour and he died within a month of being admitted. Post-mortem examination revealed chronic tuberculous cavitation in the right lung with a tuberculous broncho-pneumonia which was also present in the left lung. The coal foci of simple pneumoconiosis were present in both lungs. The radiograph taken on admission to hospital is shown in fig. 39.

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PLATE II

Case No. 108.

Date of Birth: 1910.

This man spent 21 years at the coal face in steam coal pits. He was well until 1947 when he began to feel tired at the end of a day and in 1948 was admitted to a general hospital with a left-sided pleurisy with effusion, which was diagnosed as a virus pneumonia with effusion. The radiograph taken at that time (fig. 40) shows a left-sided pleural opacity with a background of early simple pneumoconiosis. In 1949 it was seen that several scattered radiological opacities were present in both lungs but the general condition remained good.

In 1950 he first complained of rheumatic pains in the wrists which later spread to the fingers and upper limb joints typical of classical rheumatoid arthritis. A radiograph taken at this time during the Rhondda Fach Survey showed a further increase in the number of opacities (fig. 41). The sputum was found to contain tubercle bacilli on direct smear, confirmed by culture and animal inoculation; the erythrocyte sedimentation rate was 10 mm./hour. He was therefore treated as a case of tuberculosis, referred to a sanatorium where a course of streptomycin and para-amino

salicylic acid was given. As a result of this therapy the sputum became negative, the joint symptoms subsided and the general condition improved.

No radiological regression of the pulmonary lesions occurred and the film of 1952 showed an even greater number of discrete rounded opacities in both lungs typical of the "rheumatoid syndrome"; some of these lesions were cavitated (figs. 42 and 43) although the sputum was at this time negative for tubercle bacilli. This is an example of the "rheumatoid syndrome" with the appearance of the pulmonary lesions before the arthritis and is one of the two cases of this syndrome so far discovered who have had a sputum positive for tubercle bacilli during life.

Figure 40.

Case No. 108. 1948. Radiograph showing category 2 simple pneumoconiosis with a left basal effusion and a fluid level.

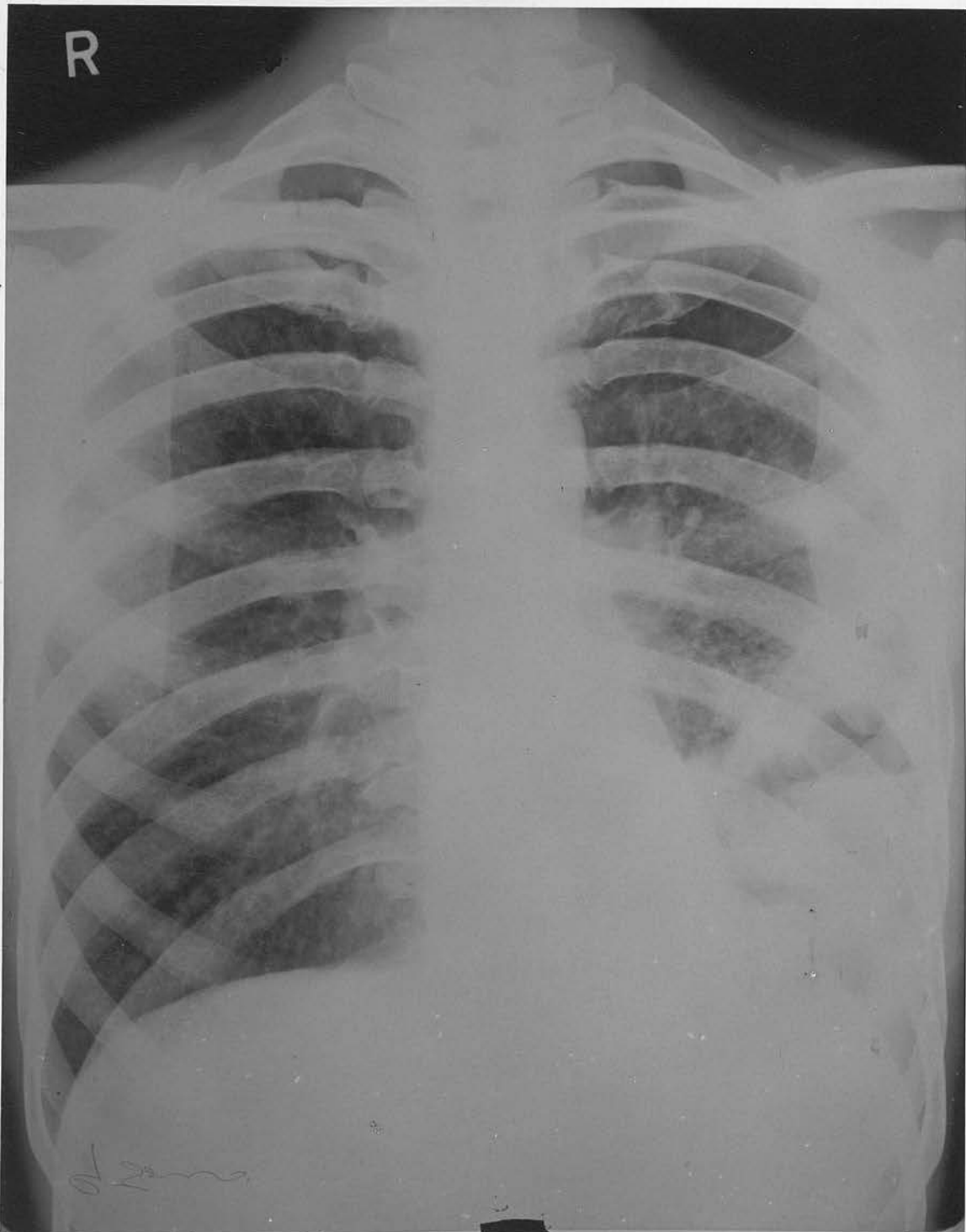


Figure 41.

Case No. 108. 1950. Radiograph showing multiple bilateral opacities; all have rather a fluffy appearance.

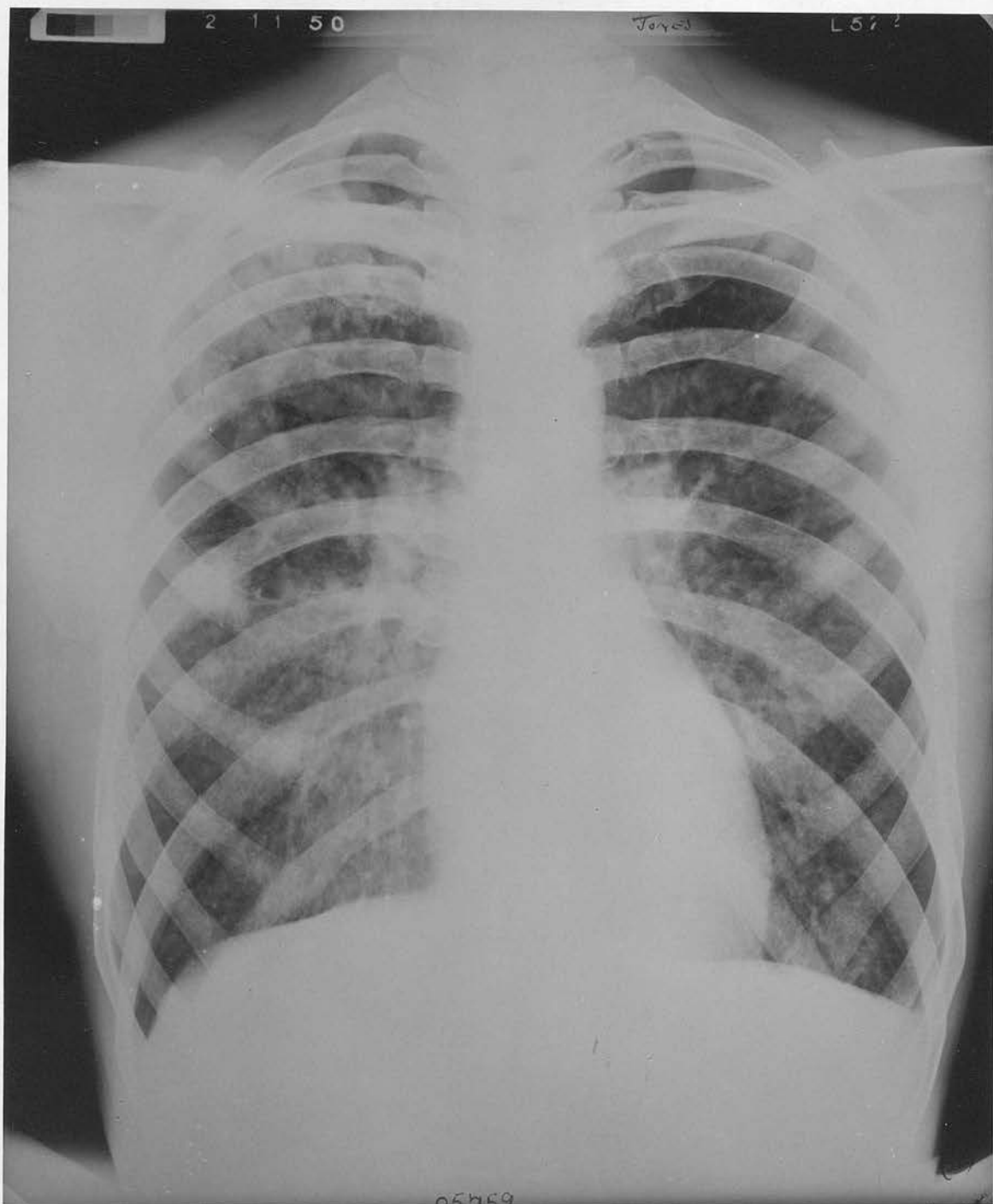
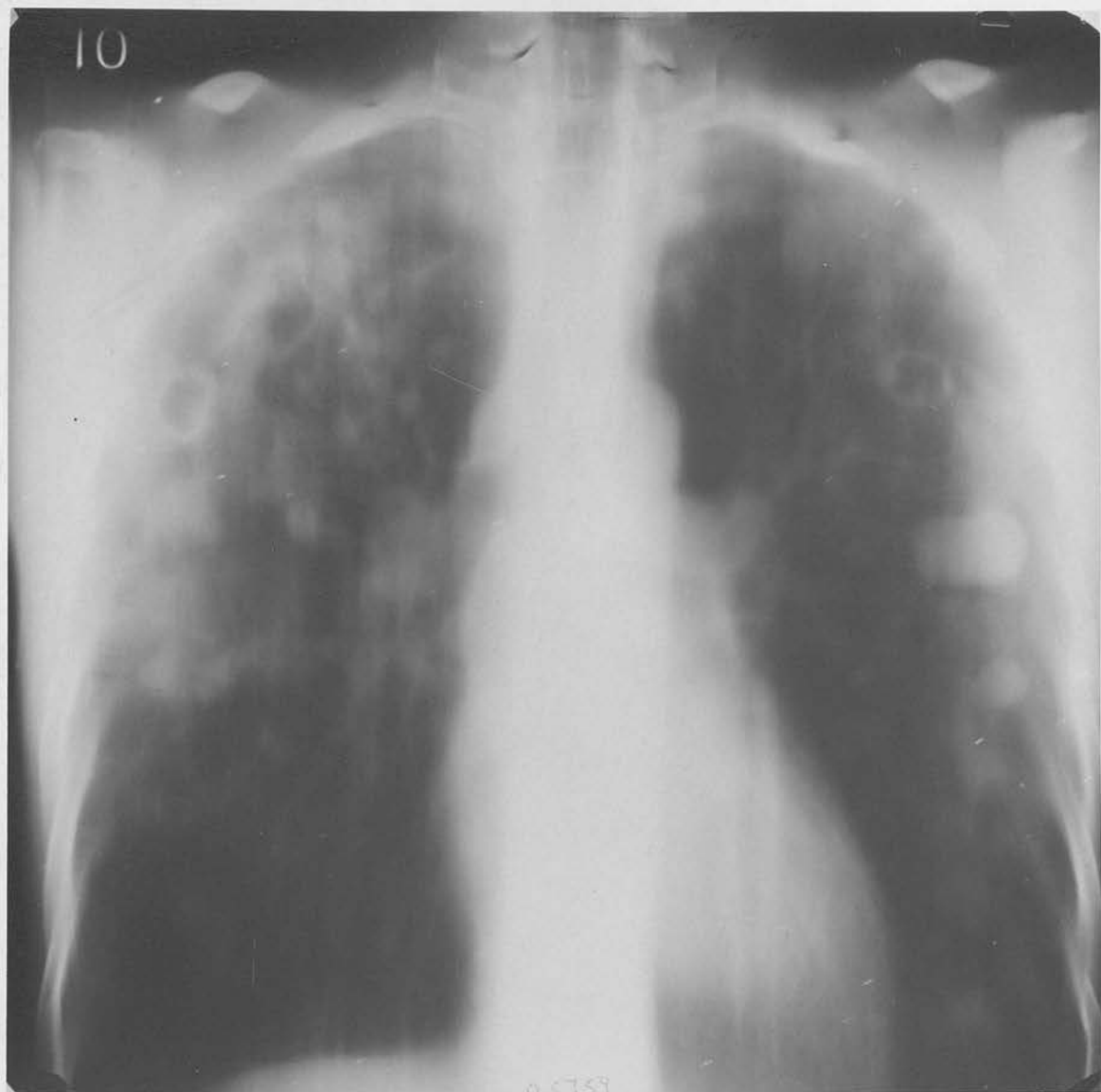


Figure 42.

Case No. 108. 1952. Radiograph shows opacities are now more numerous and more discrete.





Case No. 110.

Date of Birth: 1895.

This man spent 34 years underground at the coal face except for 2 years boring rock. A routine radiograph taken in 1949 (fig. 44), when he was 54 years of age, revealed multiple discrete opacities in the upper parts of both lungs. He was asymptomatic at the time, has remained so and is well able to do his present work as a colliery official. He had noticed small flecks of black material in the sputum from time to time, but they were not a marked feature.

Subsequent radiography in 1952 revealed cavitation in the upper part of the right lung. There were no abnormal physical signs on clinical examination of the chest, sputum specimens were negative for tubercle bacilli on direct microscopy and culture and the erythrocyte sedimentation rate was 2 mm./hour. Further examination 8 months later suggested partial refilling of the cavity but tomography (fig. 46) demonstrated that a fairly large cavity remained.

These shadows are not typical of progressive massive fibrosis, are not unlike tuberculomata and very similar to the "rheumatoid type" of lesions previously described. This patient however, has no rheumatoid arthritis.

Figure 44.

Case No. 110. Radiograph taken in 1949 showing multiple discrete bilateral upper lobe opacities. (This reproduction is from a solarised copy of the original film).

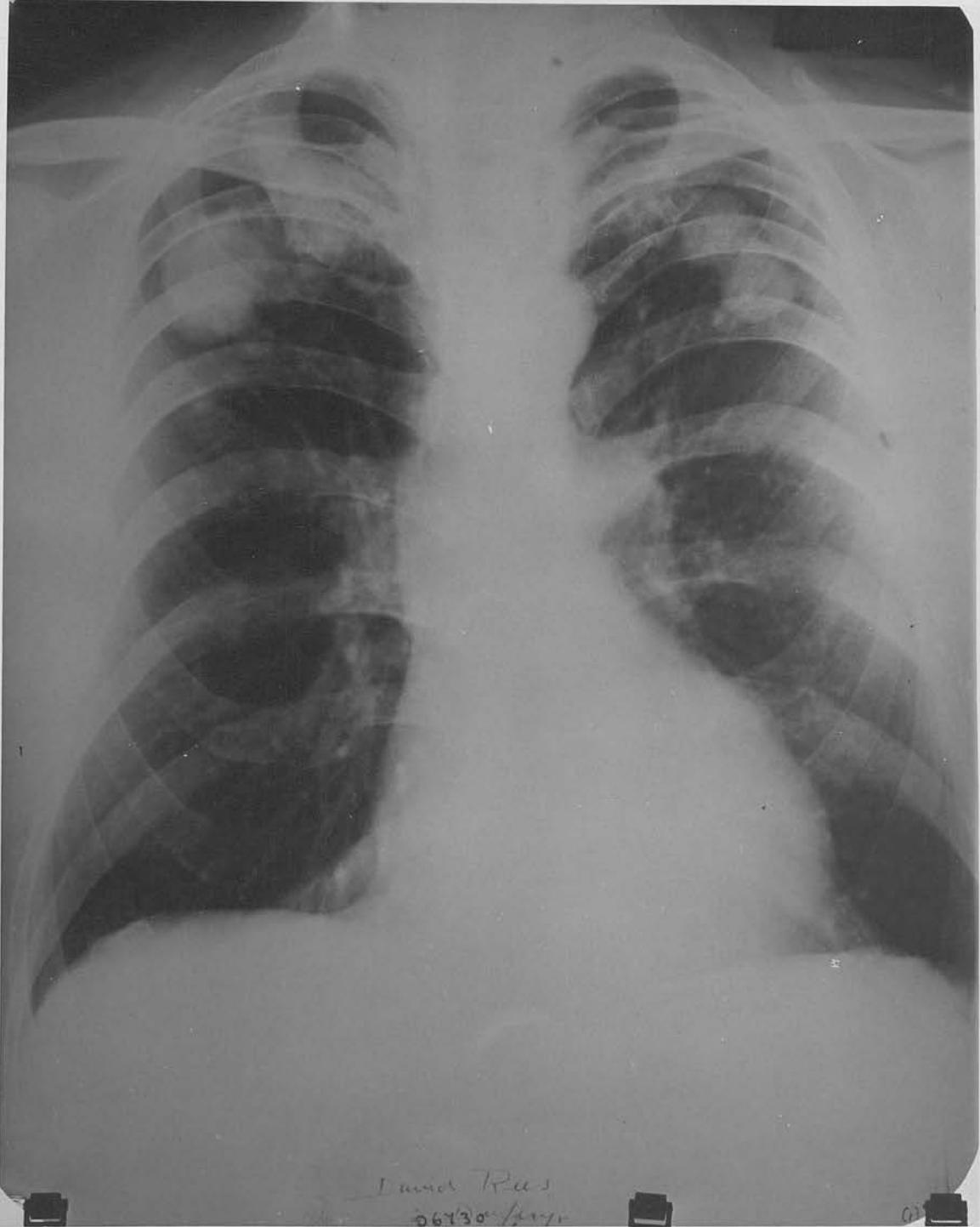


Figure 45.

Case No. 110. Radiograph taken in February 1952 showing a cavity in the right lung. The background of simple pneumoconiosis is faint.

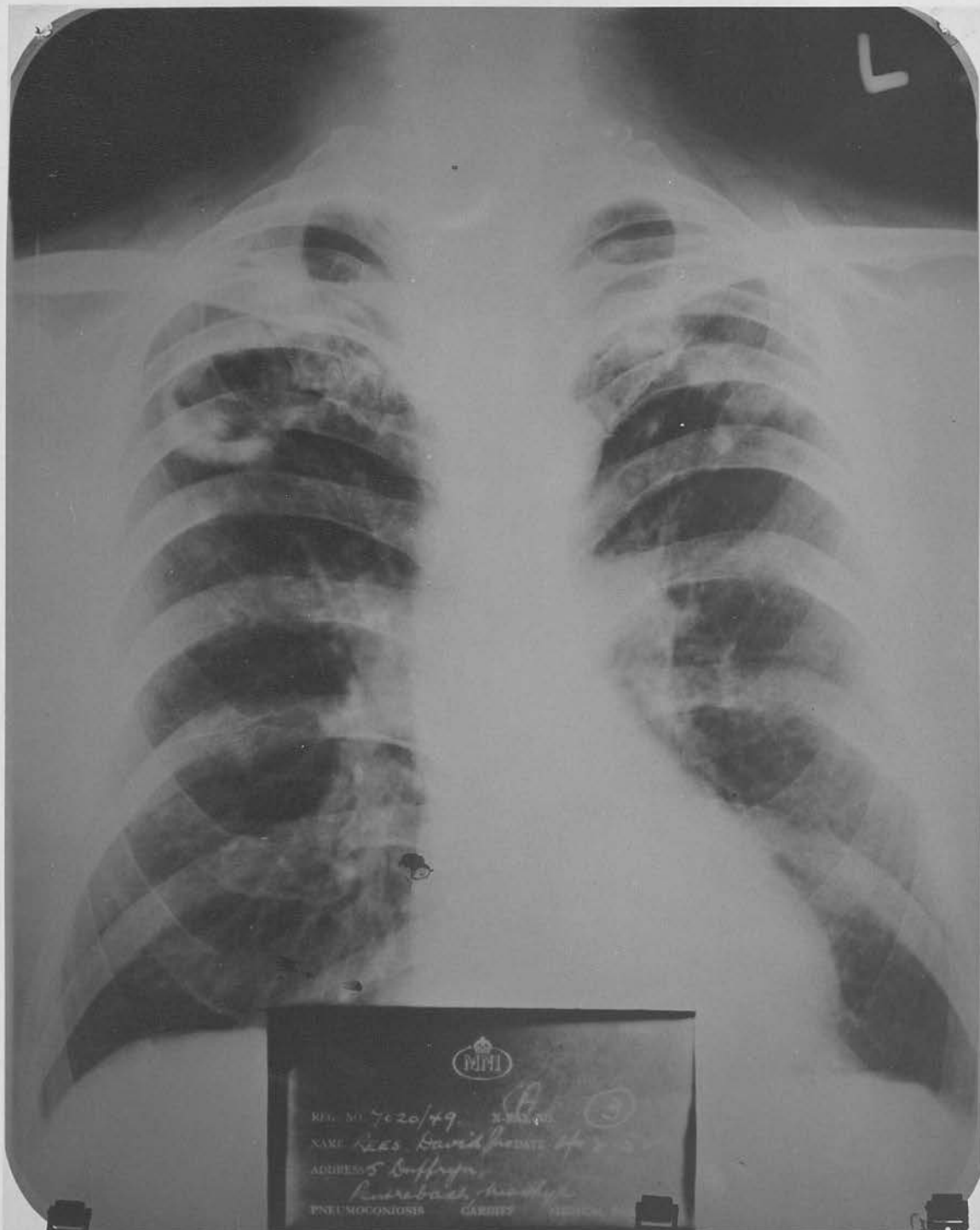
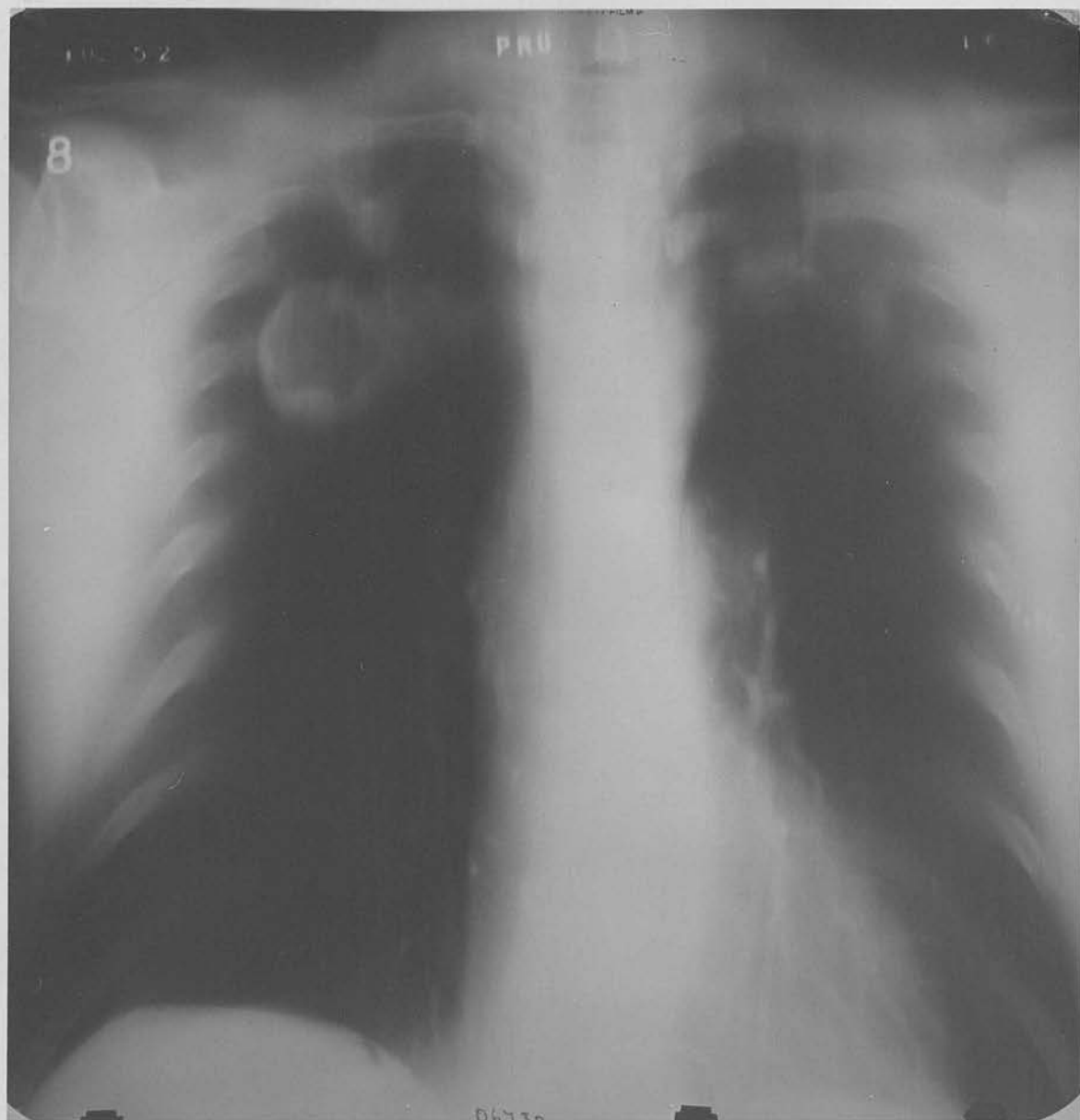


Figure 46.

Case No. 110. Tomogram showing that a fairly large irregular cavity persists in the right lung.



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Case No. 112.

Date of Birth: 1909.

This man worked for 25 years in a steam coal pit. In 1947 he noticed some breathlessness on exertion for the first time, applied to the Silicosis Board and was certified as suffering from dust disease (fig. 47). At the end of 1947 he felt much less well, developed an irritating cough and lost weight rapidly. When he was admitted to the ward in June 1948 (fig. 48) he was very ill, febrile, and diffuse crepitations were audible in both lungs. The sputum was positive for tubercle bacilli, he was diagnosed as having tuberculous bronchopneumonia, but no chemotherapy was available and he died later the same month.

An autopsy revealed confluent tuberculous pneumonia in both lungs with the coal foci of simple pneumoconiosis. A post-mortem culture was positive for tubercle bacilli.

Figure 47.

Case No. 112. Radiograph taken in 1947 showing simple pneumoconiosis together with some upper zone shadowing in the right lung.

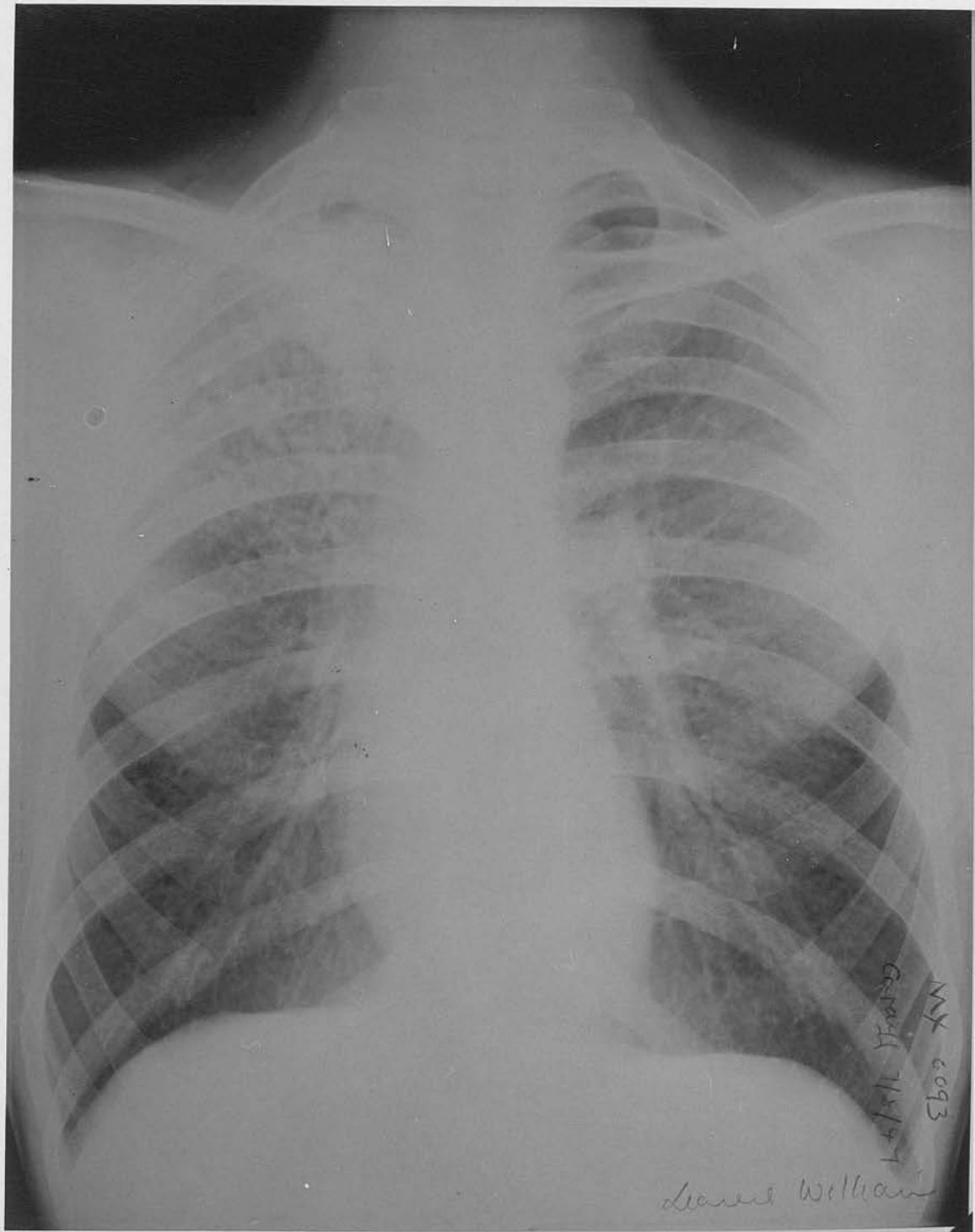


Figure 48.

Case No. 112. Radiograph taken in 1948 showing extensive bilateral shadowing with cavitation. The sputum was positive for tubercle bacilli.



ACKNOWLEDGMENTS.

This thesis has been written during the tenure of an appointment in the Pneumoconiosis Research Unit of the Medical Research Council. It inevitably reflects many aspects of the Unit work although I take full responsibility for the opinions expressed.

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