PULMONARY FIBROSIS OF HAEMATITE MINERS.

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THESIS FOR THE DEGREE OF M.D.

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INTRODUCTION.

The purpose of the following paper is to trace the pathogenesis of the pulmonary fibrosis which has recently developed among the haematite miners in West Cumberland.

Pneumonoconiosis is the generic term applied to the interstitial pneumonias contracted by workers following certain occupations associated with a dusty atmosphere and it includes, Silicosis which is the most common, Anthracosis, Siderosis and the recently described Asbestosis. The degree of fibrosis in Anthracosis depends upon the silica content of the coal dust more than the carbon, while Siderosis - usually associated with grinding steel - has been shown to be caused by the silica present in the grind-stone and not by the iron or steel filings. In the group of cases about to be discussed, the inflammatory lesions are caused by the miners working in a dusty atmosphere where the dust is a mixture of two metals, Silicon and Iron. The latter is usually considered harmless but, in the presence of the former, even in small amounts, a definite series of changes takes place which leads to a most extensive pulmonary fibrosis.

The present series of post-mortems have been collected/

collected during the last three years - 15 cases in all - and represent a new pathological lesion which has sprung up within the present decade and which will disappear since the cause has been discovered and preventive measures will be applied. The occupations followed by the men are not new but modern methods have altered the type of work from a harmless trade into a dangerous occupation.

HISTORICAL.

West Cumberland has rich deposits of minerals along the coast line, the two important ones from the industrial standpoint being coal and iron. The date of the earliest workings of the haematite deposits is uncertain, but the Furness ore was evidently known to the Ancient Britons as a few years ago polished Celts were found among the workings at Stainton (1). It is also likely that the Romans worked the iron ore here and heaps of cinders found in the Furness area have been ascribed either to the Romans or early Norsemen (2), and old bloomery slag found near Ennerdale is considered by authorities to be further evidence of Roman workings. The first definite written record we have, however, is the granting of a right to work an iron mine in the Egremont area to the Abbey of Holme Cultram by the Earl of Albemarle in 1179 (3). The founding of Furness Abbey in 1127 was instrumental in the discovery and working of iron in the Furness area of which accurate records have been kept. It was not, however, until the eighteenth century that iron was produced in quantities in this district. The working of the haematite at that time was not mining as we know it but surface quarrying of outcrops. These occur throughout the Lakelands as a result of faults and the peculiar geological formation. In 1728 mining was carried out by shafts and underground workings but only rarely did the depth exceed 100 feet.

GEOLOGICAL.

The haematite lies in the carboniferous limestone series in two main areas and occurs in veins and pockets. The main belt is about one mile in width and runs South-west from the small village of Lamplugh towards Bigrigg south of Egremont. Most of the mines in the Lamplugh, Cleator Moor and Cleator areas are now exhausted or uneconomical to work and the southern end of the belt is worked by modern pits.

The iron deposits in this Country are mainly found in the form of oxides, hydrated oxides and carbonates, and in Cumberland the main deposit is oxide. Haematite, Fe_2O_3, is a non-magnetic, crystalline ore having a hardness greater than quartz and, when powdered, is a deep red colour. Most of the haematite deposits were formed from ferruginous solutions filtering down through the rocks and along the cleavage planes, being precipitated from solution by chemical action. It is possible that the iron was in an alkaline carbonate solution which, at the same time, had silicon dissolved in small quantities, or more likely that the iron solution dissolved out the calcium carbonate of the limestone deposits which contained silicon, and the subsequent iron ore, therefore, had in it varying amounts of silicon as quartz. This latter process is known as metasomatic replacement/

replacement (5) and interesting specimens are treasured locally of fossils partly replaced by haematite, in which the central spiral is composed of calcium and the peripheral shell of iron ore (1 & 4). While most of the iron contains little free or combined silica, there are faults composed of millstone grits, whirlstone and dolomite which have resisted the alteration. In the sinking of the shafts, these layers of sandstone, limestone, quartzite or silicious strata have to be penetrated, but the side walls are mainly limestone, and this contains little more silica than the haematite - about 10%. In one mine, B, the floor of the deposit is for the most part a highly silicious volcanic ash and it is from this pit that most of the cases of pneumonoconiosis, later described, have been taken. A second belt or deposit occurs around the Millom area and pits exist at Hodbarrow where a haematite is obtained which, however, is softer in character than the Egremont deposit.

INDUSTRIAL.

Iron ore mining has been carried on as a local industry since late in the eighteenth century. The Crowgarth mine produced 20,000 tons of ore in 1794 and was still working in 1915. The total output for the Cleator Moor area alone in the decade 1871-1880 was 4,500,000 tons and this was only one area of many producing ore in West Cumberland. The growth of iron ore/

ore mining was gradual, working up from the beginning of the 19th Century to the five year period 1880-1885 (see table 1)(6) and dropping again steadily until 1914. The period 1914-1920 was abnormal due to the increased demand for iron. In any case, the figures for that period are not available since it was entirely under Government control. The low figure for 1926 is explained by the Strike.

Table 1 also shows the annual output of ore from the north west Cumberland iron-mines situated around the Whitehaven and Millom areas. It shows that there has been a steady fall in the numbers of men employed from the peak years of production, 1880-1885, when records are first available, to the present day but it also brings out clearly that, even with the reduction of the numbers employed, there has been, in the later years, a steady increase in the amount of ore produced per head. This is first noticed in the period 1923-5 when a change in the organization with increased mechanisation and specialisation was introduced. Unfortunately the figures do not fully demonstrate this increase as they are compiled from employment statistics, and do not show that the hours worked had been reduced and that there had been much short time worked owing to unemployment. The increase in output began in 1913 when 243 tons were raised per head employed. In 1929 the tonnage had risen to 431.

Thereafter/

Thereafter the depression set in and the output fell, still however keeping higher than in the last Century. Many causes contributed to this change such as the closing down of the smaller pits and the opening of larger and more modern types around the Egremont area, change in the technique of iron-ore mining with greater mechanisation and increased use of explosives and lastly - and probably the most important factor - the introduction and utilisation of compressed air drills. Before the War, all the ore was obtained by the method known as hammer and jumper. By this method the miner hammered a small chisel into the ore, frequently washed out the hole to ascertain the depth and so, at the same time, kept down the dust. The compressed air drills were known in the South African Gold Fields at the beginning of this Century, but there was a considerable delay in introducing them into the iron ore mines in this Country. In 1913, a type of drill was produced which was hard enough to stand up to the iron ore, and from then onwards more and more drills were used. Table 3 shows this increase, which became more marked after 1923. The slow adoption of the pneumatic drills in some of the pits was due to the lack of compressed air in all the working faces, and there are still some pits which have not yet got water and air distributed throughout.

The dust produced by the dry drills is very considerable,/

considerable, and wet drills were introduced to try to keep down the dust by spraying a stream of water through the end of the drill. The men were unwilling to use the newer type of drill, because it was heavier to handle and water sprayed back over the driller; so the miners used the water only when the manager or officials were present. With the introduction of pneumatic drills more rapid drilling resulted, and the need for more frequent blasting (table 5) which in turn produced more dust and more ore. (table 4.) The only time the mines were entirely free from dust was the first half-hour at the beginning of the week. This observation was frequently made to me by the miners when describing their working conditions.

CLINICAL.

It has been known since the time of Pliney that dusty occupations are associated with coughs and chronic bronchitis, and among the early observations in more modern times we find Georgius Agricola mentioning it in a book, De. Re. Metallica, published in 1557. In the 17th Century, Ramazzini referred to the connection between dusty occupations and phthisis in a book on Disease of Tradesmen. It was evident by 1713 that there was a connection between phthisis and silicosis, as a patent was taken out by Thomas Benson/

Benson of Newcastle covering the grinding of flints wet so as to reduce the dust. In 1824, Alison drew attention to the mortality among stone-masons working on Craigleith Stone.

The first evidence we have of any attempt to distinguish between the various types of dust and their relationship to phthisis is when Thackray of Leeds in 1832 observed that, whereas the death-rate among bricklayers and plasterers was normal, lead-miners, especially when working in sandstone, had a high mortality. He also noted that limestone workers, though working in a dusty atmosphere, seemed to suffer from no ill-effects.

To George Bauer (1557) we are indebted for the observation that the Carpathian Miners suffered greatly from phthisis, and that there were women in some villages who had been widowed 6 times, their husbands dying from miners' phthisis. The fact that the women themselves did not contract the disease appears to suggest that the infectivity of the tuberculosis was not high unless there was pre-existing damage to the lungs. This view that silicotic tuberculosis is not infective is generally accepted in this Country, but Riddell (11) in America states that he believes that silicotic tuberculosis is very infective and that miners suffering from it infect their relatives. This is not the opinion among the Cornwall tin miners (28).

In/

In the series of cases of iron-ore miners in this present investigation, of the thirteen cases from whom I obtained a specimen, six had tubercle bacilli in their sputum. Of the fifteen post-mortems from which I have obtained material there have been ten cases with active, open, tuberculosis; yet in no case have I obtained a history of family infection. In most cases the wives have outlived the husbands and the children are all healthy; the one exception I have met was a widower, whose wife died during a confinement, and who had a daughter with a Kyphosis - possibly a bovine infection.

It will be seen that iron-ore mining goes back a long way and that, until recently, it was considered a healthy occupation. In 1910, Mr M. Delevingue, Home Office (7), stated "As regards ironstone miners, the death rate from lung diseases is low; the mortality figures, taking 100 as the normal figure for occupied males generally, in the case of Tin Miners is 419, Lead Miners 155, Coal Miners 111 and Iron Miners 79. It is much below the average even of occupied males generally throughout the Country". Local evidence was also taken before the same Commission and Dr. Calderwood (8) Medical Officer of Health for Egremont stated that the general health of the miners was very good, and statistics showed that it was better than the general health of the population. He stated that while/

while Whitehaven had 1.03 per 1000 of the population with phthisis, and Cockermouth 1.10 per 1000, Egremont had only .95 per 1000. Of the Egremont cases, two were miners, the others being a farm labourer, an accountant and two servants. In Cockermouth, they had 7 deaths from pneumonia, equal to 1.2 per 1000 and a similar number in Egremont which worked out at 1.1 per 1000 and of these only two were miners.
In answer to a question by the Chairman of the Commission, he stated that "there was no ground for apprehension that the Miners suffered abnormally from bronchitis or phthisis". Dr Clark (9) Medical Officer of Health for Cleator Moor area also gave evidence and stated that he agreed with Dr Calderwood that the general health of the Iron-ore Miners was good.
From this, it would appear that Iron-ore Miners were a healthy body of men in 1910, and yet the local feeling now is that conditions have changed and that more chronic bronchitis exists. Various factors have been blamed, particularly drills, more blasting and increased dust generally. Under the old system of working, a man could make 5/- per day out of which he had to pay his own candles and explosives; now, both are supplied and he can make from 10/- to 14/- daily, depending on the area in which he is working. The statistical returns of the companies (table 4) show that with present methods he can produce at least

three times as much ore. A possible explanation may be that with the old method the amount of dust inhaled at work was equal to the amount of which the lungs could dispose, but with modern methods there is at least three times as much dust produced, and probably far more, and there is therefore more inhaled and retained in the lungs.

The present investigation was undertaken at the request of the Committee of Industrial Pulmonary Disease of the Medical Research Council and a small grant was made to cover travelling expenses. At the commencement, only small specimens of lungs were submitted to me from post-mortems, but later the assistance of the Coroner for West Cumberland was obtained through the Medical Research Council, and in the event of a death taking place on which a post-mortem was required I personally performed the sectio, and obtained such specimens as were required for chemical, histological and petrological examinations. The post-mortem findings were so definite and so characteristic after a typical specimen of lung had been seen, that I considered it might be useful to examine a few of the men who were seriously affected, and ascertain whether a typical clinical picture existed as characteristic as the post-mortem appearances. Altogether, specimens from fifteen Iron-ore Miners have been examined; of these thirteen died as a result of pulmonary lesions directly/

directly due to their employment and two from extraneous causes, No. 206 from bronchial carcinoma and E.M. from septicaemia following injury. E.M. also acts as a useful control, for he was employed in the Hodbarrow Mines which have a softer ore than those in Egremont.

To supplement the post-mortem examinations, names of cases were supplied to me by the Local Secretary to the Miners' Union, Mr C. Edmonds, and permission to examine the cases was readily granted by Drs Braithwaite, Mitchell, Reid and Robertson in whose practices the cases occurred. A list of 20 names was supplied and the men examined clinically. In several instances, I examined their blood, their sputum for tubercle bacilli when available, and noted their chest expansion. I have also given a resumé of their industrial histories. Four of the cases examined are now dead and post-mortems were performed. The other 11 post-mortems which were carried out, were performed on men who died before I was informed of their illness and naturally the history is more scanty, particularly respecting the pits in which they worked. In a great many cases the men followed several occupations such as coal-mining and quarrying, and the mixture of several minerals makes the interpretation of results more difficult. Some cases are pure however, and all had for some periods of their lives worked with drills. Case 13 is the best example of a short exposure, the man/

man being a miner for only 12 years. He was on drills the whole time and died from advanced fibrosis without any complicating tuberculosis to obscure the picture. All the men I examined showed respiratory embarrassment in varying degrees. In some cases, they were able to move about and even work without feeling short of breath,but exertion beyond their limit quickly made them feel distressed. The majority could move about on the level but were unable to climb stairs or hills. I found the former a simple efficient test as they were rarely able to climb the stairs in their own houses without signs of defective aeriation being evident by panting and increased pulse rate. The chest expansion was reduced, the average maximum expansion obtainable on standing erect being ¼". The connection was not direct between their ability to work and the degree of emphysema as evidenced by chest expansion. Several cases with more than ¼" expansion were unfit for work, and on the other hand cases 6, 19 and 16 had only ¼" and yet all were working.

It is a peculiar feature that the disability does not appear to be directly due to the length of time spent in the mines or to the use of drills. The years worked varied between 12 and 50. Under 20 years produced disability in 6 cases, whereas many miners who had over 50 years of service were still able to carry out a day's work though inclined to be chesty; but this/

this latter complaint is not unlikely in a man of 67. Cases 10 and 17 had the longest exposure to dust. Both had worked with hammer and jumper and drills, both had tubercle bacilli in their sputum, and both when examined were quite unfit for work. Both are now dead. At the other end of the scale we find case 13 whose total period of service was 12 years and who died of pure fibrosis with no superimposed tubercular lesion.

In 12 cases the blood was examined. Wassermann, Urea and Non-protein Nitrogen examinations were carried out and no abnormality observed. Of the 12 cases, only 5 gave a count of 5 million red cells and the average degree of saturation of the corpuscles with haemoglobin was 90%, although untold amounts of iron were available and partly in an organic form. The same blood picture was also observed by Bohrod(12) in his two cases of pulmonary siderosis. In a few instances the leucocytes were enumerated but no characteristic findings were observed; this was natural as too many variables were present, such as tuberculosis, gangrene of the lung and fibrosis.

The total period of inability to work was noted, whether as a miner or in any other occupation available. In three cases it was four years but in the majority of cases it was 18 months. At present there is unemployment, and naturally, a healthy, able-bodied man/

man is more likely to obtain employment than one who is short-winded.

I have tabulated the names of the pits in which the men worked and their years of service in the mines. (table 6.) Most of the pits are now closed as explained previously, and it is interesting to note that 50% of the men now unfit for work were in the pit known as B at some period or another in their industrial lives. The pit B has a volcanic ash floor, and out of the 15 post-mortems 12 of the men had worked in this pit. The presence of a tubercular infection was investigated. From 13 cases specimens of sputum were obtained, 45 per cent of which contained tubercle bacilli, and of the 15 post-mortem specimens 66 per cent showed active tuberculosis. These figures are much higher than might be expected from an occupation described as healthy by the 1910 Commission, and do not agree with the published statistics for the returns of the Registrar General, which are quoted by Collis and Goadby (33) and given in table 7 (extract from M.R.C. Report). This table does not indicate any undue morbidity or high mortality among iron ore miners from respiratory diseases, being very little above the standard death rate of occupied and retired males for phthisis and chronic interstitial pneumonia. The pneumonia figures are not much higher, but bronchitis ranks as a more prevalent disease, especially among/

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among haematite miners after the age of 65.

These figures are based on a few cases however, so an analysis was made of all deaths among iron-ore miners for the 30 years 1903-1932 from the three centres Harrington, Whitehaven and Egremont. As a number of the deaths occurred in the Local Hospital, the Whitehaven statistics had to be included although few of the miners lived there as the pits are too far away. During that period 1350 deaths took place among iron-ore miners, and I have tabulated the results (tables 8 and 9) showing the average age and the numbers suffering from respiratory diseases and pneumonia. The column containing respiratory diseases includes Asthma, Chronic Bronchitis, Pneumonia, Silicosis and Pulmonary Tuberculosis, but does not include Silicosis which had been contracted in South Africa working in the Gold Mines. Acute pneumonia was included under respiratory diseases but was also shown separately because in certain years, such as 1918, pneumonia was more common and not caused by occupation. When the respiratory diseases are grouped in 5 year periods (table 8) there is little variation, the pneumonia being 7 - 14 per cent of the total, except in the 1918 group when it was prevalent and rose to 25 per cent. Of the total 1350 deaths, 563 were attributable to pulmonary disease which is 41.7 per cent. This figure is much higher than one would expect from a healthy/

healthy occupation. Looking at the figures in the 5-yearly groups no increase is noticeable in recent years, the percentage varying between 39 and 44. The average age at death has increased from 54 to 58, not a great rise, but sufficient to indicate that there has been no shortening of the life span, c.f. Collis and Goadby. (33.) In the analysis it was noticed that the first three months of the year yielded the highest returns from pulmonary lesions, and to emphasise this I have included the first three months of 1934 when 64% of the deaths were due to pulmonary diseases. There is no doubt that infection is a contributory factor and a terminal gangrene of the lung is a common finding, though often superimposed upon tubercle, and it is interesting to note that in the Egremont area in 1933, 51 deaths took place of which 23 had a pulmonary lesion on the death certificate. Of these 51 deaths I only examined material from 5, and 4 of these had active pulmonary tuberculosis, so that one can be almost certain that of the 23 cases a high percentage had tuberculosis either not diagnosed or at least not mentioned on the certificate. In both 1933 and 1934 the majority of the post-mortems were done in the beginning of the year when winter coughs and infections are common.

In the original list of 20 cases which were clinically examined, 14 had been X-rayed and the films were examined at Whitehaven Hospital; all of these showed/

showed varying degrees of pulmonary fibrosis and the clinical history and symptoms of the men is typical. The first symptom is a difficulty in breathing on extreme exertion and a feeling of tightness around the chest. As the disease progresses, the difficulty in breathing becomes more marked until the mildest exercise brings on palpitation and dyspnoea. To begin with the men can follow their occupation and only excitement and extreme exertion makes them uncomfortable. The length of time between this and their final incapacity varies from 18 months to 4 years. The onset is insidious and frequently a patient who is orthopnoeic will say he has had only 12 months of tightness and six months of breathlessness, but he will admit that he had to give up football, cycling, racing or some such sport three years before because of lack of wind. I found several cases where the effort of talking was sufficient to bring on breathlessness.

On examination, they were rather sallow complexioned and round-shouldered, but on the whole a cheerful group of men. Emphysema was marked with poor expansion and exaggeration of the supraclavicular hollows. In normal respiration the extraordinary muscles of respiration were frequently used. On percussion hyperresonance with patchy dullness was frequently made out, the dullness being due to fibrosis and thickened pleura. Several had troublesome coughs with sputa containing iron, but 30 per cent of our cases had no coughs/

coughs showing that this was not a characteristic feature. The condition is due to the obstruction of the pulmonary vessels owing to the progressive fibrosis. A respiratory rate of 58 at rest was once encountered and several times of over 40. The pulse rate was not increased proportionately.

Radiological Examination.

X-ray films of the chest were obtained from Whitehaven Infirmary and these I submitted to the Pulmonary Committee who kindly gave me permission to quote their reports. These I have included in the clinical reports, along with reduced prints of such films as I obtained. A brief resumé of the clinical histories of the men is supplied.

Case 1.

J.A., Aet. 52, examined December, 1933.

Employment - Farm Worker until 1910.
Iron-ore Miner, B, 1910-14, 1915-30 - 20 years.
F, 1914-15.

Type	Hammer and Jumper	1910-14 - 5 years.	
	Dry Drill	1915-21 - 6 ")	
	Wet Drill	1921-30 - 9 ")	15 years.

He was paid off in 1930 because of shortage of work and went back to agricultural employment but was unable to continue owing to breathlessness on exertion.
He could walk on level ground but could not climb hills or stairs without stopping frequently.
Pulse - 60. Respiration - 24.

Slight/

Slight cough. Sputum clear, no pus.

Chest expansion 1½"

Non-protein Nitrogen - 50 m.g. %

Outlook - cheery. Likely to live for some time.

X-ray 13.11.31. (Fig. 1).

Case 2.

C., Aet. 55, examined December, 1933.

Employment -
Iron Miner 1895-1921, Bg, 25 years.)
1922-1933, F, 11 ") 36 years.

Type - Hammer and Jumper - 25 years.
Dry Drills - 6 ")
Wet Drills - 5 ") 11 years.

He was in the Army 12 months during the War and was idle about 1918 for 12 months. He has now been unemployed for 7 months owing to ill-health, but has recently started on a light job as a shute-banker counting buckets. He was certified seven months ago as silicosis and tuberculosis. When examined, he was not breathless on exertion and could walk quietly along the level but was unable to hurry up hills without frequent rests. The slightest excitement brought on cough and breathlessness and he produced quantities of purulent sputum which contained numerous tubercle bacilli.

Chest expansion - 1".

R.B.C. 5,000,000, W.B.C. - 5,000, Haemoglobin - 70%, C.I. - .7.

He attributed his condition to the dust following blasting/

blasting and the choking fumes of dynamite. He said the air was supplied from compressors which, being released at pressure, stirred up the dust so that pit air was only free from dust on Monday morning.

X-ray. 18.11.30. Generalised small mottling over whole of both lung fields with aggregations about central zones and towards left apex. Large cavity containing fluid in Right upper lobe. Heart outline obscured. (Fig. 2).

Case 3.

J.C., Aet. 44, examined November, 1933.

Employment - Iron-ore Miner for 30 years working in the following pits, K, S, F, M.

Type	- Hammer and Jumper	- 1904-1914.	10 years.
	Dry Machines	- 1914-1930)	
	Wet "	- 1930-1933)	20 years.

In 1933, he stopped work owing to shortness of breath. Until then he had been healthy except for tightness across the chest in walking up-hill. At the time of his examination he was rather breathless on exertion. Chest expansion - nil. He had 'flu in November, 1932 and had never recovered from it. There was constant tightness across his chest. His sputum was clear mucoid, very scanty in amount, negative for Tubercle Bacilli and negative chemically for iron.

He blamed the dust and smoke following dynamite blasting. This was much worse than formerly owing to the increased amount of blasting required with mechanic drills,/

drills, as the dust and smoke have no time to settle and disperse.

R.B.C. 4,770,000, W.B.C. 10,000, Haemoglobin - 85%, C.I. - .8

Wassermann - negative. Non-protein Nitrogen - 24 m.g.%

X-ray. 22.7.33. (Fig. 3.). Both lung fields show small and medium mottling, confluent over whole right lung. Cavity present in right upper lobe. Heart enlarged to right, outline clear.

Case 4.

J.D., Aet. 56, examined November, 1933.

Employment - Farm servant until age of 19.
Labourer in pits 3 years - Wo.
Iron-ore Miner 30 " - Wo.
Coal Miner - 3 years.

Type - Hammer and Jumper - 1899-1914.
Dry Drills - 1914-1932.
(less 3 in coal mine).

He stopped work in 1932 owing to pneumonia, and shortness of breath had prevented him from starting again. He had never worked with wet drills as there was no water at the end where he was employed. His work was excessively dusty, particularly when developing, and he was doing this mainly in limestone for 9-10 years. During the period 1923-27 he worked in Whitehaven Coal Mines and from 1928-32 again in the iron-ore mines but was only on wet machines for two months. He blamed the developing in limestone for his trouble.

The patient could sit talking quite comfortably but excitement or the slightest exertion made him breathless/

breathless. He was unable to walk up-hill or even along the level at more than a quiet pace. When he felt the tight constriction round the chest and became breathless, he had to sit down until he recovered. R.B.C. 4,480,000, W.B.C. 5,000, Haemoglobin - 90%, C.I. - 1.

Non-protein Nitrogen - 46 m.g.%. Wassermann - negative. No sputum.

X-ray 20.5.32. Emphysema and generalised fibrosis and mottling, especially on the left side. Increased root shadows. (Fig. 4).

Case 5.

R.F., Aet. 48, examined November, 1933.

Employment - Farm Servant until the ege of 22.
Coal labourer until the age of 27.
Iron-ore Miner, Ph., Bg., B.,
until age of 47.

Type - Hammer and Jumper - 13 years.
Dry Drills - 5 years.
Wet Drills - 2 years.

He stopped work in 1932 owing to pneumonia, but before that he had been getting short of breath on exertion. Since then breathlessness had affected him even when sitting still. For six months he had been unable to leave the house and had had to sit upright in bed. His work was mostly in iron-ore but he said there was a considerable quantity of quartz amongst it. The patient was in bed and orthopnoeic. He expectorated a large quantity of rusty sputum - up to half-a-pint first thing in the morning - in which were large numbers of tubercle bacilli.

Pulse/

Pulse - 120, Respirations - 58.

R.B.C. 3,420,000, W.B.C. 8,000, Haemoglobin - 70%, C.I. - 1.

Non-protein Nitrogen - 27 m.g.%, Wassermann - negative.

X-ray 8.5.33. Generalised small mottling over whole of both lungs with confluence in outer half of both upper lobes. Mottling suggested tuberculosis. (Fig. 5.).

Patient died - 2nd January, 1934 - See Post-mortem No. 340.

Case 6.

E.F., Aet. 65, examined December, 1933.

Employment - Farm Servant until 1900.
Limestone Quarryman - 1900-1904.
Iron-ore Surfaceman - 1904-1918.)
Iron-ore Miner - 1918-1934.) Pit B.

Type - Hammer and Jumper) Unable to state
Drills - Dry and Wet) with certainty.

He was still working and able to do so if he had no great exertion. He lived one mile from the main road down a slight hill. He could descend to his home in 15 minutes but it took him 45 minutes to climb the hill in the morning when going to work.

The patient was cheery and contented though unable to talk for any length of time without breathlessness.

Respiration - 32. Pulse - 80 with frequent extra-systoles.

Sputum profuse but negative for Tubercle Bacilli on numerous examinations.

Chest expansion - ¾". Non-protein Nitrogen - 30 m.g.%

Case 7.

J. Ga., Aet. 50, examined August, 1932.

Employment - Coal Miner - 2 years.
Iron-ore Miner - 34 years. B.

Type - Hammer and Jumper - 1896-1922.
Dry Drills - 1923-1931
Wet " - 1931-1933.

He had been off work for eleven weeks. Most of his life he had been robbing, i.e., removing iron-ore from located areas in contradistinction to developing, the former being a less dusty occupation. He had always worked in iron-ore pits and, although he could now easily make 10/- to 12/- daily, he preferred the old type of work when his maximum earnings were 5/-.
The patient was very thin-faced and depressed and suffered from shortness of breath even when sitting. He had 'flu recently and complained of a choking sensation and marked tightness across the chest on rising in the mornings.
Sputum was profuse and positive for tubercle. Emphysema marked.
Original weight - 13 st. 8 lb, now 9 st.
Chest expansion - ½".
R.B.C. 4,340,000, W.B.C. 10,000, Haemoglobin - 75%, C.I. - .9.
Urea - 27 m.g.%, Wassermann - negative.

Case 8./

Case 8.

J. Gi., Aet. 61, examined December, 1933.

Employment - Farm Servant - 1885-1892.
Iron-ore Miner - 1893-1903.
Coal Miner - 1904-1908.
Iron-ore Miner - 1908-1911 - Lo.
" " - 1911-1915 - M.
" " - 1915-1929 - B.

Type - Hammer and Jumper - 18 years.
Dry Drills - 14 "

He had been unemployed for 4 years. He stopped in 1929 when there was shortage of work and had remained unemployed owing to incapacity. He had marked shortage of breath and had had pleurisy twice, one attack being four years ago when he first noticed tightness across the chest. He sat gasping for breath even when at rest and was unable to go about at all.

Sputum - negative for tubercle bacilli.

Pulse - 112, Respirations - 45.

Chest expansion - ¼".

Dull and depressed.

Weight was 10 st. 6 lb., now 8 st. 8 lb.

Non-protein Nitrogen - 46 m.g.%

X-ray 5.10.31. Whole of left lung is obscured by conglomerate opacity due to confluent mottling. Right lung shows cavity in upper lobe with generalised opacities in upper and middle zones. Heart outline obscured. (Fig. 6.).

Died - January, 1934 (see post-morten report 348).

Case 9./

Case 9.

E.G., Aet. 48, examined August, 1932.

Employment - Farm Worker.
Iron-ore Worker, M., F. and La.- 1912-1932.

Type - Hammer and Jumper - 1912-1921.)
Drills Wet and Dry 1921-1932.) 21 years.

He had pneumonia seven years ago and since then had shortness of breath and pain in the chest on exertion. He was paid off in July, 1932 and was still out of employment in December, 1933.

He had lost 4 stone in weight in 10 years. He had a chronic cough but no sputum, and while at rest he was quite comfortable although he got breathless on exertion.

R.B.C. 5,220,000, Haemoglobin - 70%, C.I. - . 7.

Blood Urea - 32 m.g.%. Wassermann - negative.

X-ray 25.5.32. Generalised small mottling over whole field, very dense in middle and lower fields with patchy confluence over right side. (Fig. 7).

Case 10.

J. Gn., Aet. 64, examined December, 1933.

Employment - Iron-ore Worker, 50 years. Wy. 34 years.
B. 16 "

Type - Hammer and Jumper - 34 years.
Dry Drills - 9 "
Wet Drills - 7 "

Breathlessness forced him to stop work in 1933. For the last three months he had been off work and quite unable to do anything owing to pleurisy. He suffered from bronchitis and produced large quantities of

purulent/

purulent sputum containing iron-ore and large numbers of tubercle bacilli. He was scarcely able to talk, partly owing to extreme breathlessness and partly because he was so depressed.

Pulse 80, Respirations - about 30, but interrupted by coughing.

Chest expansion - ¼".

Urea - 35 m.g.%

Died May, 1934 - Post-mortem refused.

Case 11.

H.M., Aet. 42, examined August, 1932.

Employment - Iron-ore Miner - 1907-1932 - 26 years.

Type - Labourer for 12 years.
Miner for 14 years.
Always on drills - dry and wet
Worked in Bg. - 8 years.
" " B. - 18 "

He had been unemployed for two months owing to shortage of work and before that was off once for 9 months with a cough. During the last year his breathing had become difficult, especially on exertion and while lying in bed at night.

R.B.C. 5,000,000. Haemoglobin - 90%.

Sputum - not examined.

Case 12.

J.H.H., Aet. 53, examined August, 1932 and 1933.

Employment - Limestone Quarryman - 14 years.
Haematite Miner, Bg. - 22 years.

Type - Hammer and Jumper - 4 years.
Dry Drill - 1914-1929 - 15 years.
Wet " - 1929-1932 - 3 "

He/

He ceased work in August 1932 owing to lack of work, but by 1933 shortness of breath had made him unfit for work. He complained of tightness round his chest with shortness of breath and a cough, especially on exertion. He was perfectly well in 1931 but the symptoms of breathlessness had come on suddenly within the last few months. His work was exceedingly dusty though he worked mostly on ore and not developing.

Chest expansion ¼".

R.B.C. 5,036,000, C.I. - .7, Haemoglobin - 70% (August, 1932).
R.B.C. 4,310,000, C.I. - 1, Haemoglobin - 85% (November, 1933).

Non-protein Nitrogen - 40 m.g.% Wassermann - Negative.

Patient rather plethoric and heavily built with red face.

X-ray. 13.7.32. Generalized medium mottling with increased root shadows and emphysema. (Fig. 8.).

Case 13.

R.M., Aet. 58, examined August, 1932.

Employment - Iron-ore Miner - Bg. 3 years.)
L. 18 ") 21 years.
Coal Miner - 3 years.
Insurance Agent 15 years.

Type - Hammer and Jumper - 21 years.

He had no trouble until five years ago when he developed chronic bronchitis and occasional tightness across the chest. Recently he had noticed that he had to take things more easily.

No sputum - cough rather a wheeze - typical emphysema

X-ray/

X-ray. 1931. Emphysema with large tuberculous cavity in the upper lobe. Middle zone shows scattered conglomerate opacities with mottling in lower zone. (Figs. 9 and 10.).

Case 14.

R.P., Aet. 50, examined November, 1933.

Employment - Farm Servant until 1913.
Vickers, Gun-makers - 1913-1920.
Iron-ore Miner 1921-1932.

Type - Labourer - 6 months.
Drills, Dry and Wet - 12 years. F. and U.

He was paid off in January 1932 and had since become progressively shorter of breath. He had to sit all day on a couch and could only move round the room with difficulty owing to breathlessness which also forced him to sit up at night.

He had no sputum.

Chest expansion - nil.

R.B.C. 4,140,000, Haemoglobin - 70%, C.I. - .8

W.B.C. 12,500. Urea - 27 m.g.%

Patient very depressed and morbid.

X-ray Films not available but reported to show advanced degree of fibrosis.

Died 23rd March 1934.

Post-mortem report 368.

Case 15.

W.P., Aet. 46, examined December, 1933.

Employment - Grocer 1901-1908.
Iron-ore Miner - 1909-1918. Ub.
" " - 1918-1931. U.

Type/

Type - Hammer and Jumper - 9 years.
Dry Drills - 12 "
Wet Drills - 1 "

He was unemployed since the pit closed in 1931. He considered he was fit for work but admitted that he was unable to walk uphill without feeling short of breath and tight across the chest.

R.B.C. 4,980,000, Haemoglobin - 90%, W.B.C. 10,000, C.I. - .9.

Sputum negative for tubercle bacilli and positive for iron chemically although it was nearly three years since he had worked.

He had not been X-rayed.

Case 16.

R.R., Aet. 45, examined December, 1933.

Employment - Farm Servant - 1906.

Type - Iron-ore Miner - Labourer - 1906-07, Fa.
" " - Hammer & Jumper - 1907-22, B.
" " - Drills - 1925-30, F.
(21 years).

He was unemployed but was able to work although he had lost 2 st. in weight in six months. He looked tubercular but his sputum was negative for tubercle bacilli.

Pulse and Respirations were normal and he had no difficulty with breathing unless on exertion. He was unable to climb hills or stairs. He believed that his trouble started before he was on drills as the work, while in B. Mine, was very dusty.

X-ray/

X-ray 27.5.32 shows root shadows increased generalised medium mottling with subpleural conglomeration at periphery. (Fig. 11)

Case 17.

J.S., Aet. 50, examined December, 1933.

Employment - Farm Servant.
Iron-ore Miner - 1898-1914, Fa. and E.
Army - 1914-1916.
Iron-ore Miner - 1916-1928, F.

Type - Hammer and Jumper - 19 years
Dry Drill - 9 years.

Four years ago he had pneumonia and since then had had chest trouble with shortness of breath and had been liable to catch cold and influenza. He was certified as tubercular and was in a sanatorium for eight months. He had previously lost 3 st. which he gained again. He was dismissed as improved and had got a light job but was breathless on exertion or excitement and could not walk up hills.

Chest expansion - ¼".

Sputum - negative now.

R.B.C. 4,560,000. W.B.C. 5,000. Haemoglobin - 93%

Case 18.

J.T., Aet. 65, not examined, history obtained from friends.

Employment - Farm Servant - 1887.
Iron-ore Miner - 1887-1901, Wo.
" " - 1901-1913, U.
" " - 1913-1930, B.

He worked on stone and iron-ore the whole time, never in coal. He had had shortness of breath for three years/

years and had been unable to walk uphill without frequent rests. He was even breathless while sitting talking.

Sputum - positive for tubercle bacilli.

X-ray 19.5.33. Fine snowstorm mottling characteristic of tubercle with increased root shadows. (Fig. 12). Died 13th January 1934.

Post-mortem report 346.

Case 19.

J.W., Aet. 64, examined December, 1933.

Employment - 8 years sinking shafts until 20 yrs.of age.
Bricklayer - 18 months.
Farm Labourer - 9 months.
Gold Miner - 3 months.
Iron-ore Miner - 1915-21, Ph.
Watchman - 1921-22.
Iron-ore Miner - 1923-32, B

Type - Drills most of the time.

He was fairly well but was unable to walk uphill. He considered himself unfit for work. While at work in the Iron-ore Mines, he was working in limestone volcanic ash and shale. He was not an advanced case and made the most of his trouble.

Sputum - negative for tubercle bacilli.

Chest expansion - $\frac{3}{4}$".

X-ray 30.5.32. Coarse mottling over both lungs, confluent in both upper zones.

Outline of heart and diaphragm obscured. (Fig. 13).

Case 20./

Case 20.

H. Wm., Aet. 57, examined December, 1933.

Employment - Farm Servant until 22.
Iron-ore Miner 1898-1921, P.
Road maker for several months.
Iron-ore Miner - 1922-1931, F.

Type - Hammer and Jumper - 25 years.
Drills, wet and dry - 8 "

He was unemployed but was fit for work although he complained of shortness of breath on exertion. This affected him even when sitting talking.

Pulse - 88, Respirations - 36.

Chest expansion - ¼".

He had little cough and no sputum.

He had not been X-rayed.

Case 21.

H.W., Aet. 55, examined August, 1932.

Employment - Coal Miner 20 years.
Iron-ore Miner 20 years, B.

Type - Hammer and Jumper - 10 years.
Dry Drills - 10 "

He always worked drills dry and had practically no trouble until four years previously when chronic bronchitis started with profuse purulent sputum. Previously, he had felt some difficulty in working and in walking uphill.

Pulse - 120 per min. at rest.

Marked dyspnoea.

Sputum - Negative for tubercle bacilli on repeated examinations.

Chest/

Chest expansion - nil.

R.B.C. 4,500,000, C.I. - .8.

X-ray. 22.2.31. Generalised mottling over both lungs with conglomerations in both upper and middle zones, especially on the left. Thickened pleura throughout and emphysema in both lower lobes. (Fig. 14).

Died 21st February 1933.

Post-mortem report 237.

SUMMARY.

A summary of the clinical findings points to a fibrosis which is progressive in type taking up to four years to produce complete incapacity. This fibrosis is not the result of any one cause but undoubtedly is a product of the modern method of obtaining ore and is more liable to occur among miners using drills and especially those working in Volcanic Ash.

The findings of Collis and Goadby (33) "Experience from the mining industry suggests that prolonged inhalation of dust from iron-ore does not originate miners' phthisis" is not borne out by my findings, where 30% of the cases examined have open tuberculosis. Subsequently in their report they state "It originates definite generalised pulmonary changes of a fibrotic type which impair the usefulness of the lungs" and this can be definitely supported, but the number of men/

men in the Industry compared with the number exhibiting fibrosis must also be borne in mind. Cronin, 1926, (34) points out that the earliest symptom is breathlessness on exertion which is confirmed, but he denies that bronchitis, asthma, pneumonia and phthisis are common; this I cannot support.

PATHOLOGY.

Material from 15 post-mortems was examined. In ten cases I performed the complete sectio, and in the remaining five I was fortunate enough to obtain specimens of lung.

P.M. 1/3377. J.A., Aet. 59.

He was a Haematite Miner for 45 years and worked on machines for 10 years. He was unemployed for twenty months before his death.

Pit B.

Post-mortem - Dr Henderson.

Histologically the lung shows massive fibrosis with a chronic caseating tuberculous pneumonia and a terminal gangrene. The fibrosis is massive in type and is of long standing, and embedded in the tissue are masses of iron dust in which a few highly refractile granules of Silica can be seen, but the presence of the iron causes diffusion of the polarised light making the demonstration of the Silica more difficult. There/

There is definite endarteritis obliterans with peri-arterial and perilymphatic fibrosis. The iron dust can be seen in the muscle of the blood vessels, carried and deposited there by the lymphatics. Pleural thickening is marked and there are collections of lymphocytes but no Tubercle Bacilli can be demonstrated. Insufficient material was supplied for chemical analysis.

P.M. 2/5513. G.E.L., Aet. 41, died May, 1932.

He was an iron-ore miner for 14 years and worked on machine drills for 12 years. Pit B. He had one short illness prior to 1932 - not defined - and was ill for only a few months before his death in February 1932.

X-ray, February, 1932. The upper half of the right lung was obscured by dense opacity with mottling extending to the base. The left lung showed diffuse mottling throughout (fig. 15.). The appearance was compatible with advanced tuberculosis.

Post-mortem - Dr Henderson.

Right lung. There was an irregular thickening of the pleura over the upper and middle lobes, while over the lower lobe posteriorly the pleura was translucent, showing the brick coloured haematite dust arranged like terrazzo. Separating the emphysematous bullae were dense strands of fibrous tissue (Fig. 31.). The lung was a brick dust colour, and on section, a thick/

thick fluid exuded similar to the colour of jewellers' rouge. There had been an obliteration of the pleural sac over the upper lobes which were friable. There was a cavity present at the apex with sloughing walls and the upper and middle lobes were adherent. The lower lobe was emphysematous and oedematous and had a rubber-like consistency with relatively little functioning lung tissue present. Figures 19 and 20 show the gross appearance. There is a dark background of emphysematous lung tissue pigmented with haematite and a brighter red area showing round nodules of dense fibrosis. The paler areas in the upper part of the picture represent the tuberculous caseation which was widespread. (Fig. 18 and Specimen 1).

Histologically the lung shows a pneumonic consolidation with tuberculous caseation and giant cell formation. There is a marked fibrosis which is patchy in distribution. It appears to be massed particularly around the bronchi and lymphatics, and in certain areas presents the whorled appearance of a silicosis, except that it contains masses of iron pigment. On examination by polarised light this appears as a brilliant orange colour and largely obscures the refractile points of silica. Endarteritis obliterans is present and iron is deposited inside the vessel wall both in the medial and subintimal coats. Inside the alveoli masses of iron pigment are present lying free and also contained within the phagocytes. It is interesting to observe/

observe that the iron pigment lying free is optically active, while that contained inside the phagocytic cells is inactive and stains blue with $K_4Fe.CN_6$ in distinction to the free pigment which remains unstained.
The bronchial glands show clearly the silico-siderosis along with tuberculosis, with nodules, irregular in outline merging into one another. The nodules containing iron dust often lie within a mass of caseating tuberculosis which contains no pigment (fig. 22).

Chemical Analysis.

Ash - 16.97)
Silica - 1.11)
Fe. - 7.11) of dry weight.

P.M. 3/5513. W.B. Har., Aet. 65, died May, 1932.

He was an iron-ore miner for 38 years. Pit B. Medical History - Accident, 1931. Never ill previously. He was ill for thirteen months before death.
X-ray. Nov. 1931. Widespread opacities especially in the upper half of both lungs; lighter mottling at base where vault of diaphragm was irregular.
Post-mortem - Dr Henderson.

Only a fragment of the upper lobe was submitted for examination. The tissue was covered by a thickened pleura and was exceedingly friable. Cavitation was present with active spreading tuberculosis and the lung felt nodular to palpation.
Histologically the bronchial gland is anthracotic and shows a nodular fibrosis of the whorled type with collections of iron dust. Silica granules can be demonstrated/

demonstrated by polarised light. At the periphery of the fibrosed area are masses of degenerating phagocytic cells filled with granular iron deposit carried there by the lymph stream. Inside this layer is a zone of iron lying free, while the centre of the lobule is composed of a mass of dense fibrous tissue containing small amounts of iron and silica. There is no evidence of Tubercle Bacilli in the glands.

The lung shows well-marked diffuse fibrosis with little whorling and with dense deposits of iron and crystalline silica. Peribronchial and arterial fibrosis is marked and massive caseating tuberculosis is present which is actively spreading. The patient has had a terminal gangrenous condition of the lung.

Chemical Analysis.

Ash - 25.11)
Silica - 1.98)
Iron - 9.23) per cent of dry lung.

P.M. 4/206. E.A. Gr., Aet. 34.

1913-17 - Labourer working above ground.
1917-21 - " " under "
1921-26 - Miner working with hammer and jumper in iron.
1926-31 - " " " dry drills.
Unemployed from 1931 until November, 1932 when he died.
Pit C.

He had chronic bronchitis for the last six months.

X-ray 1932. shows increased root shadows, and no mottling or fibrosis. (Fig. 16).

Post-mortem.

The lungs were crepitant and floated easily in water. There was slight apical pleural thickening and an/

an enlarged mass of glands in the mediastinum. Their colour was that of dull brick dust and they were mottled in appearance. Both lungs were oedematous and foul-smelling and on section a large amount of thin brick-coloured fluid exuded. The left lung had no pleural adhesions and was of normal spongy consistency. The lower lobe of the right lung was adherent to the diaphragm and there was a nodular mass the size of a hen's egg lying in the free border adherent to the pericardium. Enlarged mediastinal and pre-aortic glands were present. On section, the left lung showed oedema and iron oxide pigmentation with no evidence of fibrosis.

The right lung showed a primary carcinoma with extension through its substance by peribronchial infiltration.

Histologically the left lung shows a thickened pleura lying over a slightly fibrosed area containing iron dust. Throughout the lung are small collections of iron dust, particularly around the blood vessels and bronchi, but the iron has stimulated little or no response in the production of fibrous tissue. The right lung is similar to the left, but in addition there is present a typical oat-celled carcinoma infiltrating the peribronchial lymphatics.

The bronchial glands show deposits of iron with little fibrosis and extensive invasion by carcinoma.

Iron can be seen surrounding the arteries and lying free as small collections of granules inside the/

demonstrated by polarised light. At the periphery of the fibrosed area are masses of degenerating phagocytic cells filled with granular iron deposit carried there by the lymph stream. Inside this layer is a zone of iron lying free, while the centre of the lobule is composed of a mass of dense fibrous tissue containing small amounts of iron and silica. There is no evidence of Tubercle Bacilli in the glands.

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Unemployed from 1931 until November, 1932 when he died.
Pit C.

He had chronic bronchitis for the last six months.

X-ray 1932. shows increased root shadows, and no mottling or fibrosis. (Fig. 16).

Post-mortem.

The lungs were crepitant and floated easily in water. There was slight apical pleural thickening and an/

cavitation present along with active tuberculous infection. (Fig. 21. Specimen 2.)

The large cavity in the right apex had shaggy necrotic walls and was filled with a thick creamy fluid, stained with iron oxide.

Histologically the lung shows a thickened pleura with subpleural deposits of iron dust and fibrosis of massive type. The cellular response is mostly lymphocytic in type owing to tubercular infection, which is extensive, causing caseation and cavitation. There is emphysema, collapse, and areas of oedema which have become infected as a terminal condition, producing an infected pneumonia with necrosis of lung tissue. The fibrosis is massive in type and does not present the whorled formation; (Fig. 24.) with it is incorporated iron dust and a few granules of particularly refractile silica. (Fig. 55).

Endarteritis obliterans is not marked nor is the fibrosis localised so much around the bronchi and arteries.

The gland shows generalised fibrosis with a few whorls of dense fibrous tissue. There is a large amount of iron dust scattered throughout the gland with foci of greater concentration. These are composed of a central nucleus of dense hyalin fibrous tissue containing little iron but showing necrotic changes, though no evidence of tubercle is present; surrounding this is a zone of degenerate phagocytic cells/

cells containing iron in a generalised fibrous tissue matrix. Further out are collections of lymphocytes, endothelial cells and phagocytes with young fibroblasts. No evidence of tubercle can be seen in the gland, while polarised light demonstrates numerous silica crystals.

There are many alveoli embedded in the fibrous tissue showing reversionary metamorphosis of the lining epithelium. The alveoli lying close to dense fibrosis contain many phagocytes packed with iron giving the Prussian Blue reaction. (Fig. 25)

Chemical Analysis.

Ash - 17)
Iron - 7.63)
Silica - 1.) per cent of dry lung.

P.M. 6/237. H.W., Aet. 56, died 21st February, 1933.

He was a miner for 40 years, working in coal for 20 years and in iron-ore for 20. For the last nine years he worked with dry drills.

See case "19" for clinical and industrial history.

Post-Mortem.

Both lungs had numerous chronic adhesions and the pleura was thickened and oedematous. Emphysema and general fibrosis were marked and the consistency was that of rubber. On section both upper lobes were consolidated and there were patches in both lower lobes and half the right middle lobe. The inter-lobar pleura was greatly thickened and adherent, and fibrous bands radiated into the lung substance from the/

the bronchi. There was slight bronchial dilatation but no cavitation. The hylar glands were deeply pigmented with coal and iron but not grossly enlarged. Immediately below the thickened pleura was a zone of compensatory emphysema and there was a small consolidated area at the lower pole of the lower lobe of the right lung. The colour was a dull reddish brown, owing probably to an admixture of a small amount of coal with the iron. (Specimen 3).

Histologically the lower lobe shows marked emphysema, congestion and a slight amount of peribronchial and arterial fibrosis which is early in type and has incorporated with it iron dust and refractile silica granules. There is marked bronchitis, the bronchi showing desquamation of the lining membrane and being filled with pus.

Sections of the upper lobe show a massive fibrosis with large amounts of iron dust incorporated. There is practically no lung tissue left, the whole lobe being collapsed and fibrosed. The iron appears to be universally scattered throughout the organ, there being no localised deposit, though the fibrosis is more marked around the vessels. (Fig. 26). The bronchi cannot be identified and the vessels show endarteritis obliterans with iron dust infiltrating the coats through to the subintimal layer.

This was a marked feature of this case and even large vessels showed the obliterative process associated with/

with destruction of the media. (Fig. 27). In sections stained with $K_4Fe.CN_6$, and followed by a Weigerts elastic stain, the destruction of the coats can be made out clearly. The media has been infiltrated by phagocytes containing iron and the iron deposited there, and this has provoked a marked fibrous tissue reaction which has replaced the muscle. In turn the elastica has hypertrophied, but here again the fibrous tissue has replaced the elastica so that an actual gap exists. The Intima is grossly hypertrophied, and in the degenerate subintimal layer which has been re-canalised, are very numerous phagocytes staining blue with $K_4Fe.CN_6$. Fig. 28 shows this very well and it can also be seen in several sections from post-mortems Nos. 237 and 368. The pleura is greatly thickened and the deposit of iron lying below is so great that the histological picture is obscured.

By polarised light, the iron shows up as a golden-brown mass with a few refractile crystals. There is no evidence of tuberculosis in either the upper or lower lobes.

The bronchial glands show a considerable deposit of iron with very little fibrosis, and by polarised light a sprinkling of refractile granules can be made out. There is no evidence of tuberculous infection but an anthracosis is present. (Fig. 29).

Again it is observed in the sections stained with $K_4Fe.CN_6$ and examined by polarised light that the/

the iron which does not stain blue diffuses the polarised light and appears a golden-brown colour, while the blue granules do not affect the light and appear black by contrast.

Chemical Analysis.

Ash - 25.4)
Silica - 1.84)
Iron - 12.006) per cent of dry lung.

P.M. 7/240. R.D., Aet. 67, died 6th March, 1933.

He was a Haematite Miner for 40 years, the last 12 of which he worked on drills in pit B.

No industrial history is available but the patient was in an Asylum for 12 months prior to his death.

Post Mortem. Both lungs showed emphysema and had a solid rubber-like feeling. On section, they were found to be a dull brick colour with large cavities at their apices. They were of a nodular fibrous consistency and showed a patchy mottled congestion with interstitial pneumonia and areas of consolidation and central caseation so accounting for the nodular lesions. Histologically the fibrosis is not so marked as in previous cases. It is diffuse in type and incorporated in it are large quantities of haematite dust, mainly around the blood vessels. There is an active caseating tuberculous broncho-pneumonia present and often the caseating areas and silico-siderotic lesions are associated. Where a caseous area occurs along with a sidero-silicotic lesion the dust is usually absent from the centre, while in the silicotic fibrosis the dust exists/

exists in the centres. See fig. 51 taken from post-mortem No. 370.

There is a tuberculous bronchiolectasis and the bronchi are filled with pus and breaking-down tuberculous material. (Fig. 30). The upper lobe shows more marked fibrosis than the lower and there is definite anthracosis present. In both the alveoli and the bronchi are numerous phagocytic cells, containing iron and giving the Prussian Blue reaction.

Chemical Analysis.

Ash - 12.)
Silica - .75)
Iron - 4.62) per cent of dry lung.

P.M. 8/255. M.T., Aet. 45, died 7th April, 1933.

He was an iron-ore miner for 23 years. He had never worked on stone drift and was on machines for the last 10 years.

He had difficulty in breathing since he had pneumonia in 1932 and was unable to work thereafter.

Post Mortem.

The right lung was collapsed owing to chronic empyema and was covered by a thick shaggy pleurisy. The lung substance was exceedingly friable and firmly adherent to the posterior thoracic wall.

The left lung had a slightly thickened pleura with puckering at the apex. On palpation a nodular fibrosis could be made out. On section, it was found to be a brick colour, oedematous and had the consistency of/

of a rubber sponge.

The mediastinal glands were enlarged and pigmented, but no evidence of Tubercle was present, though the left lung showed minute active caseating foci of Tubercle.

One small cavity, probably bronchiectatic, was present in the upper lobe, which was more fibrous than the lower although the tuberculous infection was not so pronounced.

Histologically the left lung shows pleural thickening and subpleural fibrosis with multiple caseating Tubercles and a heavy deposit of iron in the fibrous tissue. The right lung shows a diffuse fibrosis incorporated in the fibrous tissue and there is a large amount of iron dust which has probably been around the vessels originally but is now generalised. There are numerous small Tubercles scattered throughout but no nodular whorled fibrosis can be seen.

Chemical Analysis.

Ash - 23.)
Silica - 1.7)
Iron - 13.619) per cent of dry lung.

P.M. 9/340. R.F., Aet. 48, died 3rd January, 1934.

He was an iron-ore miner for 20 years and worked mostly in iron-ore. He had been disabled for 2 years, but prior to that had suffered from shortness of breath. See case record No.5.

Post Mortem./

Post Mortem.

The right lung had one or two adhesions over the upper lobe and, on removing it, several cavities were discovered and opened accidentally. These cavities varied in size up to a walnut and were filled with sloughing, foul-smelling pus. The lung itself was somewhat crepitant, had rounding of the free border, evidence of chronic emphysema and was a brick dust colour, at the same time showing subpleural carbon deposits. On section, the cavitation was noticed to be mostly in the upper lobe though there were a few fusiform, bronchiectatic cavities in the middle lobe. There was a generalised, caseating, tuberculous lesion, and it was suspected that the cavitation in the upper lobe was primarily tubercle. The upper and middle lobes showed definite fibrosis, and at the apex of the lower lobe there was a consolidated leather-like area, similar to that commonly seen in the lungs of haematite miners. The pleura showed slight thickening, and in the area free from adhesions posteriorly, the surface was studded with flat, round, bright red plaques of haematite dust (fig. 31.). The left lung did not exist. The parietal and visceral pleura were firmly adherent, and on attempting to separate them, one opened into a large abscess cavity, filling practically the whole chest. Strands of tissue consisting of bronchi and blood vessels covered by sloughing lung tissue existed, and the whole was filled/

filled with foul-smelling, gangrenous material. The condition was that of gangrene of the lung. The right lung weighed 1075 grams, while the left along with the mediastinal glands, which were enlarged and pigmented, weighed barely 100. The bronchial glands were enlarged but not caseating.

Histologically the right lung shows generalised fibrosis, emphysema and tuberculosis. The fibrosis is patchy and very dense, (Fig. 32.) but it has not the typical appearance of silicosis, though there is much whorling and abundant haematite deposit in the interstises of the collagen bundles. Tuberculous caseation is present in relation to most of the densely fibrosed areas, but active spreading Tubercle is not a prominent feature. The fibrosed areas have large amounts of iron dust deposited in them and, under polarised light, refractile granules can be identified not unlike silicious material. Section of left lung shows a pneumonic process with gangrene superimposed. Tubercle is present but the main lesion is an infected, gangrenous destruction of lung which shows fibrosis, most marked around the blood vessels.

Chemical Analysis.

Ash 16.662)
Silica - 1.0015)
Iron - 7.39) per cent of dry lung.

P.M. 10/346. J.T., Aet. 65, died 13th January, 1934.

He was an iron-ore worker for 43 years, and part of/

of his time was spent in developing when he worked on stone. His disability had lasted 3 years. His sputum was positive for Tubercle, and he had a daughter with a tuberculous spine. His wife died at a confinement. See case record No. 18.

Post Mortem.

Both lungs had slightly thickened pleurae and were adherent to the parietes. On palpation areas of fibrosis could be elicited. On section the lung was seen to be a dull brick red colour with irregularly scattered fibrosed areas, varying in size from a pea to a walnut. These areas were dense and had a clear cut edge. Emphysema, chronic bronchitis and patchy consolidation were seen, and scattered throughout were minute foci of caseating Tubercle. In the lower lobe of the right lung there was a phthisical cavity about the size of a walnut. The hylar glands were not greatly enlarged and the pigmentation was not so marked as usual. (Fig. 33.)

Histologically. Sections of the lower lobe of the lung show a thickened pleura due to subpleural fibrosis, and incorporated in the fibrous tissue are masses of iron oxide. Most of this is lying free, but in places it is still inside the macrophages. This is recognised by the shape of the deposit, for the total amount of iron occludes the cellular anatomy. There is present a bronchiolitis and early broncho-pneumonia, and large numbers of shed endothelial cells, containing/

containing iron oxide, are lying inside the alveoli. There is oedema and emphysema and the alveolar walls show definite thickening owing to cellular reaction and large deposits of iron dust. Throughout the lung the iron is found primarily in the alveolar wall where a fibrous reaction occurs, and around the bronchi and blood vessels where the lymphatic channels lie.
In the upper part of the lobes where the fibrosis is most dense, there is no recognisable lung tissue. (Fig. 34.) It is a diffuse generalised fibrosis with a few areas showing nodular formation, resembling, but not characteristic of, silicosis.

One area shows active tuberculosis associated with a bronchus, so that the early bronchopneumonia seen in other sections is possibly an early tuberculous bronchopneumonia. Proliferative endarteritis is present in many of the vessels, especially in the fibrosed areas. Under polarised light, the iron shines up as a golden-brown colour, and one notices as usual, that the iron which takes on the $K_4Fe.CN_6$ does not deflect the polarised light but appears black. At the same time, it is noticed that all the iron inside the phagocytes does not stain by $K_4Fe.CN_6$. A few highly refractile clear crystals are present. There are large areas of sidero-silicosis in the bronchial glands together with foci of calcification, but no evidence of Tubercle is to be seen.

Chemical/

Chemical Analysis.

Ash - 20.28)
Silica - 1.89)
Iron - 10.84) per cent of dry lung.

P.M. 11/348. J.G., aet. 61, died January, 1934.

Iron-ore miner - 1893-1903, 1908-29 - 32 years. Hammer and Jumper - 18 years, Drills - 14 years. Four years of disability getting progressively worse. See case report No. 8.

Post-Mortem.

On opening the thorax both lungs were found to be universally adherent to the parietes and diaphragm. The right lung had a grossly thickened pleura, measuring 8 m.m. at the apex and 3 m.m. over the diaphragm. The lung itself was small, its maximum measurement when fixed being 175 m.m. from the apex to the base (fig. 35.). The upper and middle lobes were fused by a thickened interlobar pleura,while the surface showed the rib markings very distinctly, and prior to fixation one noticed that the lungs maintained their anatomical shape as if they had been preserved in a fixative. They did not collapse on being removed from the thorax, partly owing to the grossly thickened pleura and partly to the firm consistency of the actual lung substance. On section the bronchi in the upper lobe were dilated and fibrous tissue bands could be seen running out to the pleura. The upper lobe showed three small areas the size of cherries, which exuded bubbles of air, while the remainder was consolidated/

consolidated and densely firm to palpation. The lower lobe showed approximately one-third consolidation in which the lung was as firm and solid as a well-fixed, hard, fibrous tissue. There was no evidence of nodular formation in the remaining two-thirds which showed hypertrophic emphysema. In it the bronchi were dilated, and again peribronchial fibrosis was noted, with fibrous tissue bands running out to the pleura. To the naked-eye there was no evidence of Tubercle. There were groups of enlarged glands at the hylum, about the size of a plover's egg, and on section these showed pigmentation similar to the lung with minute foci of endothelial hyperplasia. These were not caseating, but the translucent, minute, pin-point areas stood out in marked contrast with the ruddy, purplish, general colour of the glands. The colour of the lung was dull brick, and in the zones where the fibrosis was dense the colour deepened gradually to slate. This was the result of the period of work in the coal measures, after his fibrosis had started, so that there was retention of coal dust along with iron. The left lung had three-quarters of the upper lobe consolidated, having the consistency of dense fibrous tissue with a few scattered emphysematous areas. (fig. 36.) The bronchi throughout this lung were not so dilated, but there was marked peribronchial fibrosis, especially in the lower lobe which contained a few nodular areas of consolidated, fibrosed lung tissue. The/

The base was oedematous and emphysema was marked. There was no naked-eye evidence of Tubercle. Histologically the severity of the fibrotic process is fully borne out. Extensive tracts of lung tissue are completely replaced by fibrous tissue of extreme density, enmeshing large quantities of haematite dust (fig. 37). In the alveoli surrounding the massive fibrosis, epithelial desquamation and deeply pigmented phagocytes are present in great numbers (fig. 38). Branches of the pulmonary artery involved in the silicotic lesion show the most extensive degeneration, condensation and rupture of the elastica, invasion by pigment filled phagocytes of the media and intima and almost complete obliteration of the lumen (figs. 39 to 43).

One section shows a beautifully encapsulated fibrous nodule lying beside a vessel, and is the nearest approach to a pure silicotic nodule present (fig. 44).

Chemical Analysis.

Ash - 18.22)
Silica - 2.126)
Iron - 8.00) per cent of dry lung.

P.M. 12. I.W., Aet. 49. Presented by Col. Harvey.

He was a Farm Labourer until the age of 29 and was then a haematite miner in Pit B for 16 years, being employed mainly on machine drills.

He worked much of the time on Volcanic Ash and Limestone.

He/

He was unable to work for the last two years owing to "chest trouble" and was X-rayed twice but the films are not available.

Post-Mortem.

The specimen consisted of portions of lung weighing, in all, 650 grams. One large slice of the left lung showed a densely thickened pleura, particularly over the apex where it had been torn from the consolidated underlying lung substance, and over the inferior aspect where it had been attached to the diaphragm. The lung measured 16 c.m. from the apex to the tip of the base, and the pleura varied in thickness up to 14 m.m. On section there was a fibrosed, consolidated, upper lobe with only one or two small areas of crepitant lung. The lower lobe showed several small cavities the size of hazel-nuts, and surrounding these the lung was pale in contrast to the brick dust colour due to the haematite. This pale colour was a consolidated, tuberculous pneumonia. The pericardial sac was attached and was greatly thickened. The bronchi showed marked fibrosis and there were enlarged glands present, pigmented with haematite and carbon. The lung was prettily marked into lobules by bands of fibrous tissue. These nodules were roughly the size of cherries, though irregular in shape.

Histologically the right lung shows active tuberculosis with massive fibrosis and extensive caseation.

The/

The caseous areas show evidence of chronicity, as they have cholesterin clefts, an absence of iron dust in the centre, and are surrounded by an outer fibrous tissue zone containing quantities of dust (figs. 45 and 47). The tuberculous process is scattered throughout the lung, and while some of it is relatively recent, there are areas of old fibrosed foci. The dusting is generalised, but where tubercle is most active there appears to be less dust. The pleura is thickened and there is a zone of dust in the subpleural lymphatic spaces. There is endarteritis obliterans and the blood vessels show the granular deposit of iron in the wall. One section, however, shows a fibrous nodule which is "whorled", suggestive of a silicotic nodule. (Fig. 46.) Under polarised light, the lung shows up as a golden brown colour owing to the iron but there are refractile granules present which are white in colour and which are more numerous in the (?) silicotic nodule.

Chemical Analysis.

Ash - 12.21)
Silica - 1.97)
Iron - 5.55) per cent of dry lung.

P.M. 13/368. R.P., Aet. 50, died 23rd March, 1934.

Employment - He was an iron-ore miner for 12 years, working on drills the whole time. See case record No.14. He suffered two years from emphysema, chronic bronchitis and shortness of breath.

Post-Mortem./

Port-Mortem.

Both pleural cavities were completely obliterated by a fibrous pleurisy of long-standing. Both lungs were universally adherent throughout. They were stripped off with difficulty and were found to have thickening of the pleura over the upper lobe on the left side and the upper and middle lobes on the right. The lower lobes in both sides were soft and spongy while the upper lobes were dense and firm on palpation. On section there was no evidence of cavitation or evidence of tubercle, though in the lower lobes the bronchi were larger than normal. The left lung weighed 1150 grams and showed a dense fibrosis of the whole of the upper lobe and the upper third of the lower lobe, (Fig. 48). No crepitant lung tissue could be found in the upper lobe, while the lower two-thirds of the lower one was crepitant and showed emphysema of a hypertrophic nature. The lung was a dull brick colour and haematite dust flowed out with the fluid when it was sectioned. The right lung weighed 1030 grams and presented a similar appearance to the left. (Fig. 49.) There was a solid dense fibrosis of the upper and middle lobes and the lower lobe showed fibrosis with hypertrophic emphysema. There was no evidence of tubercle, and on palpation a dense fibrosis was found with no nodular formation whatever. On section it showed a similar appearance of a brick dust colour, dilated/

dilated bronchi, and uniform fibrosis of the upper and middle lobes with no crepitant lung tissue. (Specimen 4).

Histologically the lung shows a diffuse fibrosis of both upper lobes with no recognisable lung tissue. The fibrosis has in certain areas a slightly whorled arrangement, the centre of which is hyaline and contains no iron pigment, and surrounding it is a zone of cellular tissue containing masses of iron.

In the upper part of the lower lobe, where the lung tissue is recognisable as such, there can be seen a few nodules definitely resembling silica. In both lower lobes emphysema is marked and a solid oedema is noted. The fibrosis is not present to anything like the same extent in the lower lobes, the emphysema being the most marked feature. No evidence of tubercle is found in either lung, though a calcareous gland was found in the hylum. The macrophages, filled with iron dust, are beautifully shown lying inside the alveoli, and under polarised light, the iron showed up as a golden brown, with a few points of light, clear and needle-like, in contradistinction to the iron; these are considered to be SiO_2.

Chemical Analysis.

Ash - 16.67)
Silica - 1.11)
Iron - 6.72) per cent of dry lung.

P.M. 14/370. J.M.B., Aet. 60, died 10th April, 1934.

He was an iron-ore miner for 46 years working with machine drills for the last 12. Pits - Bg., M.B. and W. He worked on iron-ore with drills until 8 years ago when he had an accident, after which he had a light job and no longer worked on the ore but on limestone with drills. For two years he had suffered from shortness of breath and had a chronic cough. As his work was in limestone, the atmosphere was very dusty.

Post-Mortem.

The costal cartilages were not ossified, and on opening the thorax both lungs were found to be universally adherent to the parietal pleura. The left lung which weighed 1075 grams, showed a cavity at the apex, and on removing the lung this was torn open. (Fig. 50.) The pleura was thickened, particularly over the lower lobe, and the adhesions were dense between the diaphragm and the lower surface of the lung. The mediastinal glands were enlarged and pigmented, and on section the lung showed marked emphysema, chronic bronchitis with dilated bronchi, peribronchial fibrosis, and a few zones of dense fibrosis, mostly in the subpleural area. The colour of the lung was a dull muddy brown. There was marked emphysema and it did not resemble the usual appearance of the lungs of a haematite worker. There was a considerable amount of puckering present, and the cut surface/

surface of the lung exuded fluid. On examination of the cut surface, there were numerous lentil-like nodules throughout. At first these were considered to be foci of fibrosis since they could not be seen but were more easily felt; but on closer examination the alternative appeared to be foci of tuberculous broncho-pneumonia. The right lung, which weighed 910 grams, was more damaged than the left. The upper lobe was composed largely of dilated bronchi and breaking down cavities, though it could not be decided by naked-eye whether these were tuberculous, bronchiectatic, or simply gangrene of the lung. The pleura was not generally so thickened over the right lung and the fibrosis was not so diffuse as one would expect in a haematite miner. Emphysema, chronic bronchitis and generalised oedema were the most noticeable features along with the multiple lentil-like foci of tuberculous silicosis. Histologically the lungs show diffuse fibrosis with tuberculous caseation. The tubercles are scattered throughout and are more prominent in the emphysematous areas than in the areas showing diffuse fibrosis. (Fig. 51 and 52.) All the foci occur, however, in areas showing pre-existing fibrosis, and it is difficult sometimes to decide whether they are fibrous areas due to silica with central hyaline change, or tubercular caseation, though both lesions are almost certainly present. The amount of iron dust is less than one commonly finds in iron-ore miners. Under polarised/

polarised light the deposit is seen to be mainly iron but there are a few clear, refractile crystals present. The liver shows intense cloudy swelling with central lobular necrosis, congestion of sinusoids and deposits of iron. These deposits are small in amount and are inside the phagocytes. There is an early cirrhosis due to long standing congestion.

Chemical Analysis.

Ash - 16.36)
Silica - 1.78)
Iron - 6.89) per cent of dry lung.

P.M. 15. E.M., Aet. 64, died April, 1934.

He was a haematite miner for 33 years and had been on drills for the last six months. From 1896-1918, he was engaged in the usual routine work of hand boring, blasting and handling trucks. After four years of unemployment, he was put to work on machine drills on conglomerate, a silicious lava mixture, for three weeks; after which he spent six months hand drilling in limestone. From November 1923 until April 1933 he was excavating iron-ore, and from then until April 1934 he was on machine drills. He died of pyaemia, following an injury, never having had any chest trouble.

Post-Mortem.

The lungs were of a dull reddish brown colour and were oedematous and emphysematous. In appearance they resembled non-fibrosed areas in the cases of silico-siderosis already described - (fig. 20.)

Histologically/

Histologically there is only very slight perivascular fibrosis, widely and uniformly spread, with small amounts of silicious dust, haematite, and carbon localised around the bronchi and vessels. Emphysema and oedema are marked.

Chemical Analysis.

Ash - 5.98)
Silica - .40)
Iron - 2.68) per cent of dry lung.

SUMMARY OF POST-MORTEMS.

The lungs were the only organs showing characteristic changes, though, in most cases, right-sided cardiac hypertrophy and dilatation was present. The colour of the lungs varied between a bright brick dust and a dull brown with areas of slate blue running through it, see Figs. 19 and 20. In cases where the whole industrial life had been spent in iron mines the colour was bright red, but when the men had worked part of the time in coal, case 6, the anthracosis modified the picture as also did limestone, case 14.

On palpation the fibrosis could be made out as belonging to three types - diffuse, massive and nodular. In a diffuse fibrosis the whole lobe felt like a rubber sponge and was a dull brownish red colour with generalised emphysema. This was unusual and, if present, was restricted to the lower lobes as it was the precursor of the more usual type of massive fibrosis. The latter/

latter type of fibrosis was the most common and invariably involved the upper lobes. The size varied from that of a hazel-nut to a mass occupying the whole lobe, cases 11 and 13. It was a dense, airless, hard mass which cut with difficulty and which had a clean cut edge. It sank in water and was usually a bright red colour, though frequently darker zones of anthracosis could be made out intermixed with the bright Ferric Oxide. Surrounding the massive fibrosis were areas of grossly emphysematous lung. The emphysematous alveoli were frequently visible to the naked eye, especially where the lesion was immediately under the pleura. The nodular type was rare, and several times I was mistaken in describing a nodular fibrosis as typical of silicosis, when histologically it proved to be a tubercular broncho-pneumonia, case 14. Two cases 1 and 8, however, did have nodular fibrosis along with the massive types, and in both tubercular broncho-pneumonia was present, but histologically the nodules are a silico-siderotic tuberculosis.

When the cut surface of lung was treated with $K_4Fe.CN_6$ and HCl., a blue colour was produced, as observed by Staub Oetiker (35) and Bohrod (12). It has already been pointed out that this was only given by the iron which had become altered to an organic preparation either before or after being taken up by the phagocytes, and not by the inorganic Ferric Oxide; hence, the colour had a maximum intensity/

intensity around the fibrosed areas in the emphysematous and normal lung tissue.

Pleural thickening was present in every case. In some there were only patchy adhesions between visceral and parietal layers, with normal areas of translucent pleura through which could be seen the haematite dust, case 2 (Fig. 31.). In many, however, the pleura was dense and inelastic and measured up to 14 m.m. case 12, contributing largely to the firmness of the lung.

Chronic empyema was present in two cases and, in several, complete obliteration of the pleural cavity was discovered. A normal pleural cavity, free from adhesions, was never found.

In only one case, 14, was iron deposited outside of the thoracic cavity; a few scattered deposits of iron were found on the abdominal surface of the diaphragm in enlarged preaortic glands and, histologically in the liver.

HISTOLOGY.

The histological picture naturally varies greatly according to the area from which the specimens are taken. In the grossly fibrosed areas, the lung tissue is quite unrecognisable. It is a dense collagenous mass of fibrous tissue, incorporating varying amounts of iron dust, but always sufficient to give the stained sections/

sections a bright red colour by reflected light when held against a dark background. In the massively fibrosed areas bronchioles are indistinguishable, but blood vessels can usually be made out and are best seen in cases 5, 11 and 13 in which Tubercle does not exist. In the first stage, phagocytes invade the adventitia and media and there provoke a fibrosis with replacement of the muscle wall by fibrous tissue. (Fig. 55) There appears to be an increase in the amount of elastica which broadens out and becomes irregular in thickness. This appearance is shown in fig. 40 from Case 11. The intima then becomes invaded by the phagocytes and, when stained by $K_4Fe.CN_6$, the subintimal zone appears a mass of blue. In the subintima recanalisation takes place, especially when there is obliteration of the lumen, and as the fibrosis continues pressure causes collapse of the vessel and it becomes H shaped (fig. 43.). A later stage is evident when the media cannot be identified, and only an endothelial mass is left filled by phagocytes and surrounded by an elastic coat; (Fig. 53) this mass also stains blue by $K_4Fe.CN_6$. The last stage is when the vessel is completely obliterated, and an irregular mass of elastic fibers is all that remains, absorption of the haemosiderin having taken place, fig. 54.

In all the sections of fibrosed lung, the disease appears to start round the bronchi and pulmonary veins, where/

where the lymphatics run, and spread thence throughout the lung. The iron is often collected into masses surrounded by collagenous fibrous tissue which contains less iron, (fig. 47), and interesting comparisons between the massive fibrosis and nodular type can be made. In the massive type the iron is universally scattered throughout the area, while in the nodular type the nodules have practically always a necrotic centre which is free from haematite and is caseous. In all the nodular lesions Tubercle is also present, producing a silico-sidero Tuberculosis, or more strictly a tuberculo-silico Siderosis, since infection is essential (Kettle) for the production of the typical silicotic nodule and Tubercle is the usual infective agent. In a few cases typical silicotic whorled nodules are present, but these are not common. In them the iron takes the place of the silica in a true silicotic nodule, but there is very much more of it present. When sections are examined under a polarising microscope a remarkable picture is seen. Under the 2/3" objective the iron deviates the light, so that it appears as a bright orange colour against a dull grey background, and in the thinner parts of the section a few acicular points of greenish white light stand out. These are very small and often lie below the orange coloured iron, and are considered to be silicates. Under high power the colour still remains golden brown, but the individual particles are now visible/

visible as minute, translucent, orange-coloured points of light. Scattered amongst these are the clear refractile points of light, considered to be silicates, but these vary enormously in amount in the different specimens.

After the work of Jones (22) appeared last Summer, particular attention was paid to these acicular crystals in order that the presence or absence of sericite could be decided, and details of this will be dealt with later. It is observed, that under polarised light the iron dust within the phagocytes, although plainly visible even in ordinary sections not stained for iron, frequently fails to give any refraction, while the dust present in the lymph glands which must have arrived there inside phagocytes is birefringent.

The spongy lung shows emphysema and much less fibrosis, which is always perivascular and peribronchial in distribution. In many cases the alveoli are packed with phagocytes containing iron and giving the Prussian Blue reaction, and this is most obvious at the edge of the fibrosed areas. Spreading bronchopneumonia, bronchiolitis and gangrene are frequently present as a terminal lesion.

Tubercle is present in 10 of the 15 cases. The question arises as to whether Tubercle is an infection superimposed upon a fibrosed lung, or whether Tubercle pre-exists and is reactivated by the fibrosis. In most cases dense areas exist in some part of the lungs without/

without any evidence of Tubercle, but both are frequently together. In case 12 the cholesterin clefts present indicate that the tuberculous lesion is of long-standing and preceded the fibrosis, and in addition no haematite dust exists in the centre of the nodules, while in a typical silico-siderotic nodule dust is present mixed with caseous material. In several cases the lungs were cultured by aerobic and anaerobic methods on a variety of media. The growth included almost every common variety of pathogenic organism.

The bronchial glands are frequently enlarged, figs. 31, 33 and 35, always pigmented with iron dust, and often with an admixture of carbon, cases 6 and 12. In several cases they show Tubercle which is not usually in an active state, and fibrosis which tends to be more nodular and similar to the true silicosis, figs. 21 and 22. The nodules are rather irregular in outline, and some are almost encapsulated by a collagenous envelope.

CHEMICAL ANALYSIS.

For chemical analysis a portion of lung weighing 300 grams was used. This was obtained by taking a thick slice through the whole lung so that representative portions of both upper and lower lobes were present, instead of selected parts from the areas of advanced fibrosis. The lung was minced finely and dried/

dried over a hot air oven at 110° to a constant weight. After grinding and thorough mixing, 10 gram portions were incinerated. To begin with platinum crucibles were used, but this practice was found to be costly as a crucible rarely lasted more than three incinerations before perforating. For a long time no solution was forthcoming as to why the bottom of the crucible became like a sieve, until a local analytical chemist suggested that platinum was readily destroyed by carbon in the presence of Phosphorus and Iron.

As all the lungs had a marked fibrosis and, therefore, high P_2O_5 content, this seemed a likely reason. I obtained samples of rustless steel, flat, crucibles from Messrs Firth of Sheffield and I found these satisfactory for the primary incineration. By heating the dried lung in a steel crucible at a dull red heat a high percentage of the organic matter was ashed and little carbon remained. The contents were emptied out of the steel crucible into a mortar while still hot, finely ground and transferred to a platinum crucible to be ashed completely at white heat. It was found to be important to empty the partially ashed lung out of the steel crucible while still red hot for, if left until it cooled, the crucible was liable to scale and so upset the analysis of the iron content. Duplicate specimens were analysed using platinum crucibles entirely, and steel and platinum crucibles, and the results compared. When using steel crucibles the iron/

iron content of the lung was higher, but only to the second decimal place of the 10 gram sample of lung, so that it was considered justifiable to continue using steel instead of platinum for the first oxidation of the organic material.

The ash was fused with alkaline carbonates and the non-soluble silica transformed into $Si.O_2$ by heating with HCl. This at the same time changed the $Fe._2O_3$ into $Fe.Cl_3$. The silica was filtered off, incinerated and weighed as crude silica, $Si.O_2$. It was then volatilised by Hydrofluoric Acid, the residue again weighed and the result expressed as pure Hydrofluoric Volatile $Si.O_2$. The iron was estimated volumetrically by titration against Potassium Dichromate and the aluminium weighed as AL_2O_3. The results of the chemical analysis are given in Tables 12 and 13. The former contains some controls which I examined along with figures from other sources. The amount of the various substances may be expressed as a fraction of the ash or dried lung, both being given, and in most cases I have also given the amount of moisture present and the total weight of the lungs, so that the total amount of the substances may be calculated. Several analysts express the amounts present as a percentage of the ash and others as a percentage of the dried lung. The latter is undoubtedly the better method. When the ash amount is raised four to eight

times/

times by an abnormally high content of one substance, all the others are given erroneously low figures. To begin with this was the case in my analysis. Originally, I considered the silica content when expressed as a fraction of the ash to be too low to be the cause of the fibrosis, but after estimating the amount of iron and finding such a high figure, I realised that the percentage of silica to ash was abnormally low in that the ash was four times too great and the iron 100 times normal. To overcome this, I have given the amounts as a percentage of the dried lung and also of the ash so that the figures may be directly compared with other analysis.

Silicon, as such, never exists in the lung and the figures are calculated as silicon dioxide, though in the lung it may have existed as combined silicates. Unfortunately, one cannot estimate the proportion of the two substances separately, but this is not important as the amount of $Si.O_2$ is an index of the total amount of silicon present as both silica and silicates, and both are to be considered potentially dangerous, though until recently silicates have been ignored.

The Iron is expressed as Ferrum ($Fe''._2$) and not as $Fe._2O_3$. Again this is for comparison with other published analysis and it is easy to calculate $Fe._2O_3$ from $Fe''._2$ by multiplying by 160/112, roughly 3/2.

The Aluminium was estimated after last Winter when Jones' papers were read, and the possibility of sericite/

sericite causing the trouble had to be considered. Aluminium, expressed as Al_2O_3, need not be considered seriously, as it exists everywhere and is normally found in lungs though only as a trace. In cases of silicotic fibrosis it may be considerably increased in amount when in combination with silica as sericite. Petrologically I failed to find sericite, and therefore I estimated the aluminium chemically in certain cases, to ascertain whether there was sufficient present to upset the silica figure. In no case was the Al_2O_3 figure high, and even if all the aluminium were combined with silica the proportionate rise in amount would not be great, as it requires 46 parts of silicon with 39 of aluminium to form 100 parts of sericite.

The normal weight of the lungs is 1150 grams. Usually the weight was greatly increased. Cases 368 and 237 are interesting for they had the most marked fibrosis with no caseation, yet, in one instance, the total weight was 1170 grams and in the other 2180 grams. In both cases the percentage of moisture was below the average, but No. 237 with the normal weight had 25% ash and 12% of the lung weight as $\overset{\text{"}}{Fe}$., while 368 had only 16% ash and 6.6% as $\overset{\text{"}}{Fe}$.; that is, both contained a total of 120 grams of iron.

The iron content was the outstanding feature of the chemical analyses. Macrae (30) gives the normal amount/

amount of iron as $Fe._2O_3$ in the ash as 7.3%, which I think is high. Hammersten (37) quoting Schmidt gives 3.2. I found none in my normal control, and 4% in men working in dusty occupations. Simmons and Hammarsten both give 9.85 as the percentage found in pneumonoconiosis due to silica, while in my one case of silicosis due to grinding steel, I found 21% of the ash as $Fe._2O_3$. When, however, we consider the iron-ore miners, the average iron oxide content of the ash is 69% or 11% of the dried lung. The normal amount of iron oxide present in the lung is 1.5 gms., while the lungs of these miners each contained over 150 grams and one case (Tho. 255) had 396 grams. The normal silica content of the lungs varied between wide limits. My normal control had .03% of the lung as $Si.O_2$ or 1% of the ash, while Macrae gives his figures as .73% and 14.7% and Hammarsten as 13.4%. Dr Fowweather of Leeds, in a personal communication, accepts the figures .1 and 4% as the normal figures for silica. Among the iron-ore miners the average figure is 1.3 and 8.2. As explained, when I started the analyses I expressed the silica as a percentage to the ash, and I considered 8% of silica not sufficiently high to blame for the fibrosis; when, however, I found such high ash and iron figures, I expressed the silica as a percentage to Iron-free ash, and then the figure rose from 8 to 24 which ranked as dangerously high.

EXPERIMENTAL.

To ascertain whether the fibrosis resulted from the iron oxide or was caused by the silica in the ore, I tried to reproduce the pulmonary fibrosis in laboratory animals. My first idea was to obtain rats from the pits but in this I was unsuccessful and, though I considered it, I was unable to keep my own animals in the iron-ore mines.

Carleton (15) subjected guinea pigs to dusting tests and concluded that haematite was a harmless dust. His experiments were inconclusive as the animals were not exposed to the dust for a sufficiently long period and the ratio of the dust inhaled to dust swallowed could not be ascertained. Guinea Pigs and rabbits are not suitable for dusting experiments as their fur tends to collect the dust from the air and their nasal mucosa cleanses the air more efficiently than in man.

Carleton found in the liver, spleen and sub-diaphragmatic glands dust which must have been absorbed by the alimentary tract. In the post-mortems which I have done here, enlarged pigmented glands in the abdominal cavity have not been noticed nor has dust been found deposited in the liver and spleen. P.M. 14 is the only case where iron was found in the liver, yet I have no doubt that the miners swallow iron dust while at their work in addition to inhaling it.

Carleton found epithelial proliferation of the alveoli/

alveoli with bronchiolitis but no permanent change in the bronchi and no fibrous tissue reaction. He compared the iron with shale dust and his results were similar though he found the toxic effect of iron greater. This finding has some clinical bearing for I noticed, among the men examined, no pigmented scars. Pigmentation is common amongst coal miners on the hands and face but among iron-ore miners pigmented scars are rare. They wound themselves as frequently as coal miners and the iron oxide gets into the lesion but the wound does not heal until all the dust has been discharged by suppuration.

Beattie (16) performed dusting experiments upon guinea pigs, using haematite from Cumberland, and he came to the conclusion that iron-ore from this area caused no reaction in the lungs.

Throughout my experimental work, the dust was given by the intratracheal route as devised by Kettle (14). Originally, 50 guinea pigs were inoculated with a 4% suspension of pure ferric oxide but a high mortality within the first few days from pneumonia reduced the number to 24 and only one pig survived 10 months, the remainder dying from septic pleurisy, infection with paratuberculosis or gaertner. This was due to an unfortunate cage infection in the Animal House which decimated the stock. The results were interesting for those which died soon showed the immediate effect and gave a good idea of the amount of dust/

dust reaching the alveoli.

In the first experiment, the dust was pure ferric oxide, obtained by grinding kidney ore, and contained under 1% of silica. The histological picture is constant; congestion, endothelial proliferation, shedding of phagocytic cells, engulfing of particles and disappearance of iron oxide.

The guinea pigs were kept until they died, unfortunately none survived long enough to show any fibrosis. Fig. 56 was taken from the one that survived the longest, 10 months, and it shows a bronchus surrounded by muscular tissue and no fibrosis or dust. It is in this area that dust would be deposited if removed by the lymphatics and where fibrosis would be expected to start first. Six weeks after intratracheal injections of dust, a considerable amount may remain as Fig. 57 shows but there is no cellular reaction and no necrosis, only emphysema. The most likely place to find dust remaining is either around the bronchi or immediately under the pleura and Fig. 58 shows a fairly normal lung with a subpleural deposit of dust but little cellular reaction, certainly not enough to predict a subsequent fibrosis. The amount of dust present is small considering that 5 cc. of a 4% solution was injected 4 weeks previously. Fig. 59 shows a mild reaction, the trauma of the injection, with alveolar thickening and a slightly cellular reaction, the iron having been removed from the/

p 131

p 132

the alveoli and lying in the wall.

In the pigs dying before the fourth week, considerable reaction is frequently seen owing to the trauma of the injection or to the terminal infection.

After six weeks, the iron has largely disappeared from the lungs and what remains seems to evoke no reaction.

As cavies were unsatisfactory, I decided to use rabbits. They could tolerate repeated injections, 7-10 c.c. of a 4% suspension, and they were not so likely to succumb to intercurrent infections. In all 12 rabbits were used. One rabbit survived nearly two years during which time it had 5 injections of pure oxide, a total of 1.76 grams.

To begin with Kidney ore was obtained from the mine and finely ground but later B.D.H. pure ferric oxide was substituted as the particles were more uniform and less likely to block the needle.

As I had failed with pure iron to produce a fibrosis in guinea pigs resembling miners' fibrosis, I obtained a sample of local ore, ground it finely and used this. Eight rabbits were given repeated injections of this ore, two with ore alone and six with ore and a suspension of dead tubercle bacilli. Three rabbits were injected with pure ferric oxide as a control, one with tubercle; and one with a suspension of killed tubercle bacilli only (Table 17). The tubercle was a human strain recently isolated from a urine. The ore was from Pit B. and contained 9% of silica.

With/

With rabbits the operative mortality was not as great as with guinea pigs. One rabbit died from drowning and several failed to recover completely from the anaesthetic and died 36-48 hours later, usually after the second or third injection.

The tubercle was added to repeat the work of Kettle (27) who recently produced typical looking silicotic nodules with Kaolin, an aluminium silicate usually considered to be harmless, and he added a suspension of dead tubercle bacilli. He had failed to produce fibrosis with Kaolin even after 500 days had elapsed following the injection, but when he added a suspension of dead tubercle bacilli as an infective agent, he produced in 112 days typical fibrocaseous nodules with the characteristic early appearance of silicotic fibrosis.

Rabbit No.1 had two injections of 1 gram of pure ferric oxide with three months interval. It was killed 10 months after the first injection and,histologically, the lungs showed little reaction with only small amounts of ore retained. This was classified as a negative finding.

Rabbit No.2 had four injections with a total of 1.6 grams of ferric oxide and one injection of tubercle, the last injection being 18 months after the first. Unfortunately, it died 5 days later, so that it can only be used to show the immediate reaction and the amount of iron being injected (Fig. 60). p183

Specimen 5 shows the ore well down into the alveoli,/

alveoli, but histologically there is no fibrosis, all the reaction being the result of the last injection. There is bronchiolitis with collapsed alveoli some of which contain both free iron and iron inside phagocytes with no fibrous tissue reaction.

Rabbit 3 had two injections with a total of .56 grams of ferric oxide. It survived six months after the first injection and histologically it shows emphysema and bronchiolectasis but little iron and no reaction (Fig. 61).

p 133

Rabbit 4 died within a week of the second injection. It had .4 of a gram of ore along with a suspension of tubercle. The histological picture is that of oedema and congestion following the injections (Fig. 62).

p 134

Rabbit 5 had three injections of ore with no tubercle, a total of 1 gram. It was killed six months after the first injection and showed small amounts of ore retained but no reaction. Specimen 6 shows the lung six weeks after the last injection and shows the small amount of iron remaining.

Rabbit 6 had an injection of .28 of a gram of ore and died the next day. It is included to show the amount of ore entering the alveoli in comparison with No.5 which showed little ore and no reaction. This demonstrated the ease with which the lungs got rid of dust.

Rabbit 7 had one injection of tubercle only. It was/

was killed three months later and showed only a mild reaction of endothelial proliferation.

Rabbits 8 and 10 gave the best results. Both had two injections of ore and tubercle and survived three months after the first injection when both were killed, too soon, unfortunately, to show the reaction desired but both show definite retention of ore and marked reaction (Specimen No.7). This is a typical proliferative tuberculous reaction but with necrosis and dust retention and now that a method has been obtained of procuring retention of the dust with proliferative changes further experiments will be undertaken and the animals will be left a longer period after the injections (Figs. 63-66).

Rabbit 9 had one injection of ore and a culture of live tubercle known as Saranac I. This was supplied to me by Dr Gardiner who stated that it was so attenuated by subculturing that it was only of mild pathogenicity to rabbits. I had hoped by utilising this culture to produce a lesion when combined with dust which I could not produce when inoculated alone. Histologically, the rabbit shows a little dust retention and no reaction apart from hyperaemia; it died from acute infection.

Rabbit 11 had two injections of tubercle, Saranac I and one of ore. Histologically, there is little retention of ore and no reaction.

Rabbit 12 had five injections of pure ferric oxide,/

oxide, total 1.76 grams, and survived 18 months. It died two days after the last injection but histologically it shows no retention of old ore but two bronchioles blocked with dust and a terminal acute reaction following upon the trauma of injecting the dust.

The three mounted specimens of rabbits' lung show the results of different methods. Specimen 5 was obtained by 4 injections of pure iron oxide and one of tubercle. The iron is still located around the larger bronchi and near the centre of the lung, and histologically only acute reaction following the last injection is found. Specimen 6 was obtained by three inoculations of ore alone and very little of it remains after six weeks. Specimen 7 had only two injections of ore and tubercle and yet the ore is retained in proportionately large amounts six weeks after the last injection.

To summarise the experimental work, I failed to produce a fibrosis by any method which I used but, from the appearance of the lungs, pure ferric oxide alone is innocuous. The ore utilised from Pit B itself cannot produce a reaction. A suspension of dead tubercle bacilli alone does not produce proliferative changes but only a mild reaction of the alveolar lining membrane but when ore and tubercle bacilli are combined the results are different. There is definite retention of the ore with proliferative changes and focal necrosis which would, I believe, have progressed

to/

to fibrosis. This only resulted when the ore and tubercle bacilli were injected together; when the ore was injected first and followed later by tubercle bacilli no reaction resulted for the ore had disappeared before the tubercle was injected. Tubercle Bacilli followed later by ore also produced a negative result. In only one case did I try pure ferric oxide along with tubercle bacilli and in that also I failed to obtain retention or fibronecrosis so that I concluded that the small amount of silica present was necessary to aggravate the local lesion produced by tubercle bacilli and cause retention of the ore. This experimental work is not conclusive because the number of animals used is too small and the length of time they survived after inoculations is too short, but a method has been found that points to more successful results being subsequently obtained. The negative results with pure iron oxide and ore conform with the findings obtained by previous workers.

DISCUSSION

Silica, as free silica, has been blamed as the cause of Silicosis and the combined silicates largely ignored but recently the possibility has been raised that the silicates, especially when combined with alkalies as in abrasive soaps, are a cause of Silicosis.

Dr Jones (22), in an excellent paper, has introduced another possible cause, an alkaline silicate called Sericite, which occurs naturally among the quartzite rock. This helps to explain certain puzzling factors so far not completely understood. Why have there never been any cases of Silicosis in the Scottish Coal Fields and so many in the Welsh (24)? Why do cases occur in the Broken Hill Mines in Australia which have a low free silica content? Why is Silicosis the curse of the South African Gold Fields and yet in the Kolar Mines in India which have a higher silica content cases of Silicosis are unknown? This has however been denied recently by Subba Rao.(39) Jones' explanation in each case is the same. The sericite is present in the one and absent in the other. Chemically the cause of the Silicosis is the same. It is caused by the toxic action of the colloidal silica as it is dissolved by the body fluids. Sericite, according to Shannon (23), is hydrous silicate of aluminium/

aluminium and potash having 46.58% SiO_2, 37.46% Al_2O_3 .8% Fe_2O_3 with Ca. Na. Mg. and K. oxides in small quantities. Geologically, Sericite exists as minute scales and fibrous aggregates (25), unlike quartz it breaks up into fibres and not plates which makes it a much more dangerous component of dust. According to Jones, quartz crystalline masses measure roughly 10 x 5 x 8 μ and have the same volume as 800 fibres of Sericite 2 x .5 x .5 μ which is a common size and, therefore, mere chemical analysis to ascertain the free silica conceals the true state of affairs for, roughly speaking, 1 gram of quartz, if fully satisfied with AL and K. so as to produce Sericite, would be a little over 2 grams of combined silica. Now the danger of the silica group is in their solution and as sericite naturally occurs in smaller particles, these present a greater area to be acted upon by solvents and, at the same time, tend to penetrate, owing to their size, further into the lungs. Dr Jones does not suggest that the whole blame of Silicosis be transferred from free silica to the fibrous combined silicates but he is desirous that they should be included in the picture. In this opinion he is not alone for, at the International Conference on Silicosis, (43) Dr E.L. Middleton pointed out that Silicates would have to be considered as a possible cause of industrial disease. Dr Mavrogordato produced Silicosis/

Silicosis in animals with pure quartz, finely ground, but, after hearing Dr Jones' paper, he thought that his artificially finely ground quartz was probably much smaller than would naturally occur and that rock handling did not yield quartz particles of phthisis producing size.

Dr Jones informed me just after his paper was published, that Sericite was present in only one of the mines in the Cumberland area. On investigating the industrial histories of the men who had come to post-mortem, I found that, with the exception of Pos. (368), all the men who had died of pulmonary fibrosis had worked in this pit. In the last column of Table 13, the pits in which the men worked are given. It will be seen that pit B is common to them all and pit B is the only one having a volcanic ash floor. It seemed possible that Sericite was the cause of the Fibrosis so the lungs of these men were digested by Nitric Acid and Jones' technique followed. Unfortunately, no Sericite could be discovered though a fairly large amount of insoluble material was obtained. To prove the absence of Sericite the aluminium was estimated for, as explained, Sericite contains 37% of aluminium oxide. The average amount of AL_2O_3 present in the eight cases estimated was .22% of dry lung and if all the aluminium present was combined as Sericite, it would only raise the total Silica from

1.3/

1.3 to 1.8 which is not sufficient of an increase to alter the belief that Sericite is not present to any great extent. I submitted the deposit which I obtained to Dr Jones who agreed with my negative findings but he also noted that the Ferric Oxide presented occasionally a needle-like structure instead of the usual amorphous mass. While this did not in any way suggest that the fibrosis was caused by the mechanical action of the iron, it is a possibility that some of the iron is in chemical combination with the silica, producing Jasper, a hydrated ferric silicate, though I consider that most of the silica is present as quartz, judging from the specimens I obtained.

The fact has to be explained that, as a rule, only those men who had worked in Pit B developed fibrosis. The Hodbarrow ore we can dismiss because the conditions there are different. The ore is softer and high speed pneumatic drills not so essential and secondly the ore is damp and dust not so prevalent.

Pit B. is one of the most modern. The ore is hard and the dust more plentiful and contains more quartz than in the other mines as the walls are of volcanic ash instead of limestone. This dust is more dangerous during developing as the ore itself has not a higher silica content than the other pits.
In several instances the men had spent their whole life working in the one pit, but unfortunately in the earlier/

earlier cases where death took place before I examined them, the dependants could not always give me the information about all the pits but only the ones in which the deceased had worked longest, so that the last column in Table 13 is not complete with regard to the early cases.

The cause of the fibrosis seemed likely to be due to the silica content of the dust, for the close association between Silicosis, Fibrosis and Tuberculosis is well established. The percentage of silica present in the crude ore had been estimated frequently, and was less than that required by Government regulations to be present in crude ore in order that the occupation be scheduled under the Silicosis Order. A specimen of ore was obtained from pit B and analysed, and Table 15 shows the results of our own analysis compared with the official analyses by Bernard Smith (1), one published in the Victorian History of Cumberland (20), and a sample of pure ore supplied by the British Drug Houses Ltd.; this last was analysed by me as a control, and used for animal experiments.

The Millom ore from which no case of Silicosis or Tuberculosis has developed contains 7.2%, the Egremont ore 8.9% and two specimens of Whitehaven ore 10.5 and 7.4. The bulked North Cumberland ore is given as 4.9 - a lower figure than we obtained - but/

but the ore which I examined came from only one pit.

The iron content varied between 70 and 85 per cent and the aluminium was low - only 3%.

Judging from the analyses of the ore alone, one would not condemn it because of the high silica content, but one must remember that the dust generated in the mine comes not from the ore alone but also from the side walls and quartz faults. Another more important point is that the iron-ore dust would possibly settle more rapidly than the silica which is known to remain suspended in the air longer than the heavier iron, partly due to the decreased weight having only half the specific gravity, and partly due to the fact that iron is readily wet by water while silica tends to be dispersed and remains suspended. The ore is selected and cleaned roughly of dirt. Because of this, samples of ore are not comparable with samples of dust.

Fibrosis of the lung can be produced by inert dusts if the subject be exposed to them over a large number of years, for example coal dust to miners and soot to city dwellers, and Kettle (27) instances haematite workers as an example of this, though no association exists between inert dusts and tubercle.
I feel, however, that the case of iron oxide has not been proved innocuous as clinically the dust is irritating when inoculated in subcutaneous tissue, indicating that a similar result occurs in the lungs; and/

and Carleton found it more toxic than shale when injected into animals.

Sladden (31) pointed out that when the rock contains a low percentage of free silica, high speed mechanical drills are not needed and the dust which is generated, whilst containing silica, is in relatively large masses which settle rapidly. This undoubtedly is the case, but, in haematite mines, while the rock does not contain much silica, the ore is actually harder than quartz and, therefore, the dust is fine and does not settle rapidly. The finer the dust the more dangerous it is, as it more readily reaches the alveoli and circumvents nature's cleaning methods, the ciliated mucous membrane of the bronchi. Once in the alveoli, the natural process of cleaning is by the phagocytes and these are either discharged by the bronchi, entangled with mucus or, re-entering the alveolar wall, are carried by the lymphatics to the nearest lymphatic glands. The innocuous dusts such as coal do not destroy the endothelial cells, and when deposited evoke very little fibrous tissue reaction, but the toxic substances such as silica destroy the cell carrying it, and the silica being set free inside the lymphatic continues the irritating action; fibrosis results with obliteration of the lumen of the lymphatic and defective drainage of the corresponding area of lung. In this way the fibrosis starts,/

starts, not in the alveoli, but around the blood vessels and bronchi where the lymphatic vessels are located, and only secondarily involve the alveoli. The alveoli become oedematous and filled with shed endothelial cells packed with dust and as these are unable to escape they die and so set up the fibrosis in the alveolar walls. The fibrosis initiated by the silica is aggravated by other dusts, mainly by mechanical blockage, whether the dust is coal or haematite and in itself harmless.

Mavrogordato noted in South Africa a simple silicosis and an infective silicosis. The former, if recognised in time and the patient removed from the dust, did not progress, while the infective type was progressive whether the man ceased his occupation or not and was rapidly fatal. Mavrogordato also observed (32) that humid conditions aid the development and spread of infective conditions, which was supported by Koelsch, who found that silicosis was more frequent and severe among the wet grinders than among the dry at Solingen. Strachan & Simpson (38) in "A Preliminary study of the Pathology of Silicosis" observe that if a silicotic patient does not die of some other disease he will ultimately die of Tuberculosis. This observation might be made of anyone but Tuberculosis is the most common infective agent in Silicosis.

I/

I have observed from the death returns that the first three months of the year always show the highest number of deaths among iron-ore miners, and the same period supplied me with most of the post-mortem material. (Table 9) It is during this period that humidity is most marked, winter coughs are prevalent and resistance against infection at its lowest indicating that the pulmonary fibrosis which we have in Cumberland is caused by a combination of dust, moisture and infection.

Infection is not necessary for the production of a fibrosis, but to produce the typical silicotic nodule with the fibronecrotic centre a secondary low grade infection is necessary. In 1911, Professor Haldane (17) stated that no dangerous dust appeared to be found in any operation connected with haematite miners. This was in agreement with the figures for 1910-12 in Table 7 when the haematite miners up to the age of 45 showed very little increase above the standard population of occupied males for phthisis and interstitial pneumonia. But for the 1921-23 period - especially above the age of 65 - the haematite miner had a much higher mortality from phthisis, pneumonia and bronchitis; this period was after the increased demands of the War. In 1900-02, iron-ore miners were stated to have a healthier occupation than coal miners, but Table 10 shows statistics, often quoted, for all iron miners/

miners and not haematite alone as in Table 7, and there is no doubt that miners working in carbonate and soft oxide are not so liable to suffer from lung disease. This, Collis (19) pointed out when he showed that the miners in ironstone and carbonate of iron in Stafford and North Riding have a mortality 20% below the normal figures of standard population.

Table 11 shows well the dangers of a siliceous dust, for all ages above 25 show a substantial increase in the death rate for interstitial pneumonia and respiratory tuberculosis, and over 35 bronchitis and pneumonia are more prevalent.

The question of tuberculosis occurring among silicotic trades raises the interesting point as to whether the tubercle is superimposed upon the fibrosis or whether the silica lights up a pre-existing tuberculosis which has been dormant. Among abrasive soap manufacturers there is a higher incidence of tuberculosis among the male employees than among the female, which is explained by the greater prevalence of spitting among men; while among asbestos carders the tuberculosis incidence is lower than other silicotic industries and carding is largely a female occupation. This would indicate that the infection followed the damage to the lung, but Leroy Gardner (21) has produced reactivation of healed tuberculosis in guinea pigs by injecting siliceous material, showing that the/

the dusty occupations may cause the fibrosis and, at the same time, reactivate healed foci.

In Table 16, there is compiled for the year 1902 a list of deaths from tubercle from dusty occupations. At that date, tubercle as a cause of death was not considered abnormal among iron-ore miners compared with the standard death rate, though the group includes the whole of the iron-ore miners of Great Britain, and haematite is restricted to the North West Area. Only in those dusts which contain silica is the death rate higher for tubercle than the standard, but it must not be judged from this that all siliceous dusts cause death from tuberculosis for death may take place from pure fibrosis before a tuberculous infection sets in. In Pulmonary Asbestosis 31% of the deaths had tubercle as a complicating feature while silicosis had 56% with tubercle. The explanation given by M.J. Stewart (36) is that Asbestosis is fatal at an appreciably earlier age and after a much shorter exposure hence there is less time to develop tuberculosis.

In this series of 15 post-mortems, 66% had tuberculosis (Table 14) and all had worked for 12 years at least in the mines and all with drills. There is no apparent connection between the amount of silica present and the liability to tubercle, for the non-tuberculous cases had as high an average content.

The analysis of the deaths among the local haematite/

haematite miners (Table 9) to which I have referred was disappointing. I had hoped to obtain evidence of an increased death rate from pulmonary lesions during the last few years, but all that can be deduced is that the post-mortems reveal a condition which has not been present for many years and one which, until recently, was not looked for. The difficulty of reading the figures aright is considerable (Table 8) but for the years 1903-07 with 4652 men employed 39% of the 236 deaths were pulmonary, and in 1928-1932 period 37% of the 207 deaths were pulmonary with only 2659 men employed. This suggests that, relative to the number of men employed, the number dying of the disease has considerably increased. The maximum number of years of unemployment due to incapacity is 4, so that if we take the previous five years period for employment, we find 4652 employed with 105 deaths during next five years, while in 1923-1927, 3266 employed with 78 deaths in the following five years. This is a drop, but not in proportion to the number employed, and in any case the figure which will reveal the increase most markedly, will be the number of pulmonary deaths for the period 1932-1937, for it was only after 1931 that there were more than 40% of the men employed using drills.

Table 8 therefore shows that there is actually no higher percentage of men dying, certified as having a pulmonary lesion, but it also shows that proportion-

ate/

proportionate to the number employed the death rate has increased from 1 per 100 in 1903-1907 to 1 per 62 employed in 1928-1932, though the number of accidents has not increased. Another factor which alters the picture is that the Hodbarrow Mines, producing a softer ore, supply a variable percentage of the total Cumbrian output, Table 2A, and no case of silicosis has ever occurred among these miners. The percentage has dropped recently, the figure for 1924 being 11 and 1930, 17. This is a considerably lower figure than in the last Century and even up to 1920 they supplied over 35% of the ore produced in West Cumberland. This drop in the output means that, proportionately, a greater number of men are being employed in the hard ore; therefore, more are exposed to the likelihood of developing fibrosis.

SUMMARY.

In summing up the results of this investigation it is observed that the pulmonary fibrosis of haematite miners is a recent development. There is a history of haematite mining for several generations in this locality without any characteristic disability and yet within the last ten years a definite syndrome has developed which can be recognised clinically and which has an X-ray picture indicative of massive fibrosis.

The post-mortem findings are peculiar to the occupation for the lungs present a definite series of characteristic changes, striking in appearance on account of the bright red colour of the fibrosed areas. The fibrosis is more marked in the upper lobes, is always diffuse and is often massive in type. It originates around the bronchi and blood vessels and gives excellent pictures of endarteritis obliterans even when not associated with tubercle.

In the fifteen post-mortems, tubercle has been present in 66%. This is a higher percentage than is found in silicosis which has an average of only 56%. The experimental work confirms the general findings that infection is a necessary factor in the production of fibrosis and although two cases showed advanced fibrosis with no tubercle, this just means that tubercle is the most common infective agent, but other low grade infections can act as well. At a recent Meeting/

Meeting of the 1934 Commission investigating silicosis among coal miners, confirmation of this was found. It was observed that in mines where the men walked to the surface, there was only one quarter of the cases of silicosis compared with those where the miners were carried rapidly to the surface on trolleys. The latter caused chilling with increased liability to colds, bronchitis, and chronic Winter coughs with subsequent fibrosis.

Chemical analysis of the lungs revealed a high silica content along with a phenomenally high percentage of iron in the ash. The ore analysed gave only a 10% silica content but as I pointed out the silica content of the dust which the men breathe is probably much higher.

The appearance of this pulmonary siderosilicosis within recent years can only be attributed to the change in technique in mining for the ore has not changed chemically and the only difference is in the increased amount of dust in the mines. The modern method of mining with high speed percussion drills has considerably increased the amount of dust; this is lessened by the use of wet drills which, however, increase the humidity and also increase the chance of infection as the miners have to work in wet clothing. The use of wet drills, while it slightly lessens the dust, does not restore working conditions to the old hammer and jumper standards for the increased amount of/

of blasting and the shorter time the men are away from the working face results in their having to work longer in a dusty atmosphere.

There can be no doubt that the disease has resulted directly from their employment and is caused by a combination of silica and haematite, the latter altering and aggravating the chemical action of the former, for in itself an ore containing only 10% of free and combined silica is not regarded as dangerous.

It is considered that cases will not occur so frequently in the future now that the causes have been recognised, for the provision of proper ventilation after blasting, the use of wet drills and adequate supervision should be sufficient to ensure its prevention.

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APPENDIX A. - GEOLOGICAL.

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Specimen 8. (Postlethwaite Ore Mine. Moor Row.)

This fragment of ore is a fossil and is submitted to demonstrate the metasomatic replacement. It is a coral known as Syringopora Geniculata which existed in the limestone beds. The Calcium of the coral has been completely replaced by haematite at the same time leaving the form of the shell. Since it is known that Coral and other sea shells contain silica this is one source of the silica in the ore.

Specimen 9.

This specimen is composed of crystalline Calcium Carbonate where some of the crystals have been replaced by Iron in the form of Fe S_2 Pyrites, an unusual occurrence in the carboniferous limestone area.

Specimen 10.

This consists of a mass of haematite covered partly by specular iron ore. Among the quartz crystals can be seen a few dark coloured crystals, quartz with enclosed haematite.

There are also bright red crystals of Jasper - a combined silicate of Iron usually found in igneous rock. Most of the silica exists as crystals of quartz however and not as Jasper.

Specimen 11./

Specimen 11.

This is a mass of pencil ore - a species of pure haematite where the fibres run parallel and which splits into pencil-like rods. Inside the ore there has been deposited a mass of crystalline quartz. It is submitted to demonstrate that pockets of silica occur surrounded by pure ore.

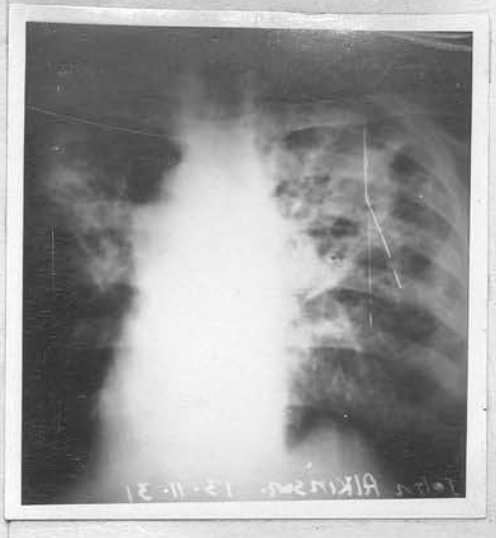
Specimen 12.

This is a specimen of acicular crystals of Calcium Carbonate lying upon an embossed mass of kidney ore. The crystals are a faint pink colour owing to the minute quantities of haematite which have been incorporated and which can be easily demonstrated chemically.

Specimens 9 - 12 were obtained from the mines around Egremont.

APPENDIX B. - PLATES.

1.



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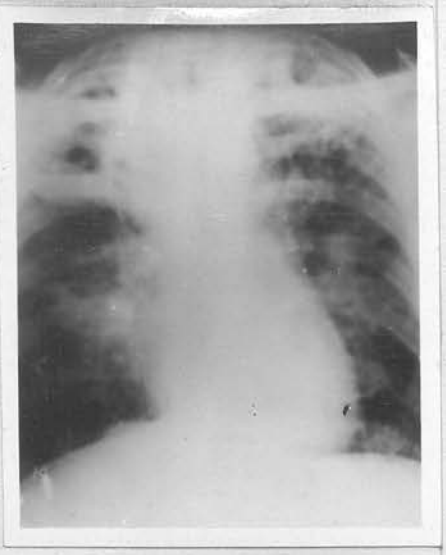
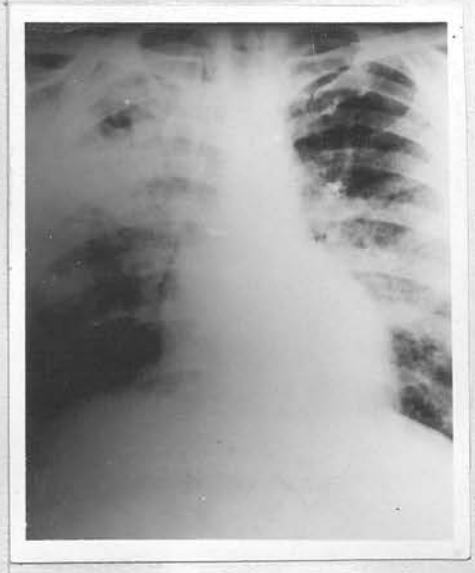


Fig. 1, Case 1. Diffuse generalised mottling with marked enlargement of mediastinal shadow. Elevation of right lobe of diaphragm.

Fig. 2, Case 2. Generalised small mottling over whole of both lung fields with aggregations about central zones and towards left apex. Large cavity containing fluid in right upper lobe. Heart outline obscured.

3.



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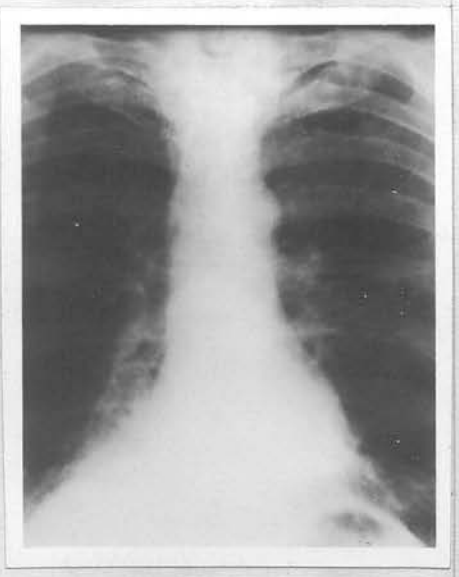
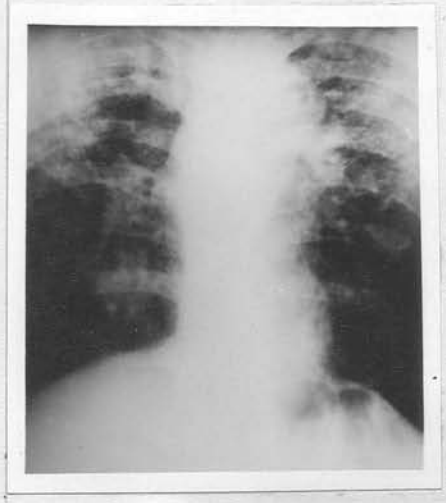


Fig. 3, Case 3. Both lungs show medium small mottling confluent over whole of right lung. Cavity present in right upper lobe. Heart outline clear and enlarged to right.

Fig. 4, Case 4. Emphysema and generalised fibrosis and mottling, especially on the left side. Increased root shadow.

5.



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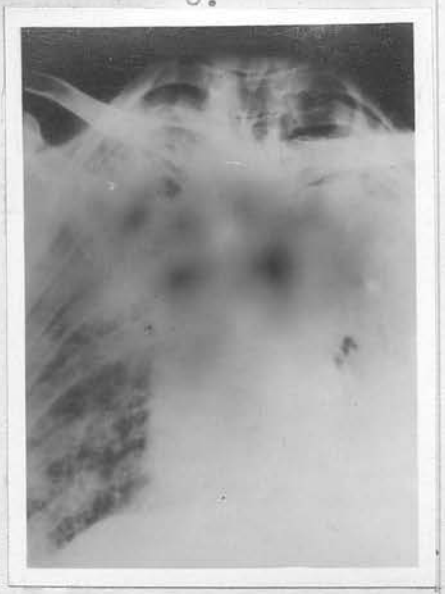
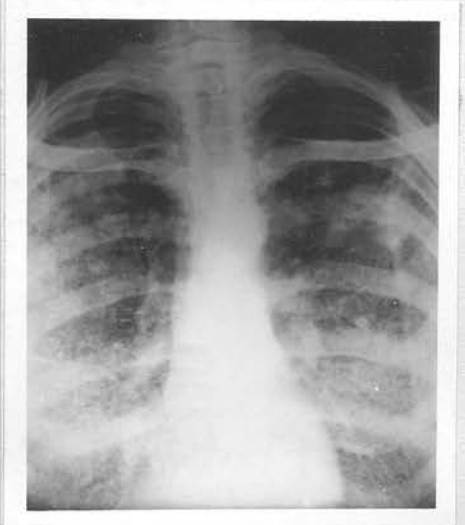


Fig. 5, Case 5. Generalised small mottling over whole of both lungs with confluence in outer half of both upper lobes. Mottling suggests tuberculosis.

Fig. 6, Case 8. Whole of left lung obscured by conglomerate opacity due to confluent mottling. Right lung shows a cavity in upper lobe with generalised opacities in upper and middle zones. Heart outline obscured.

7.



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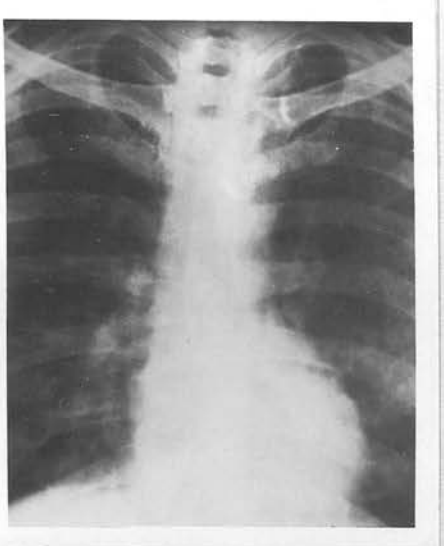
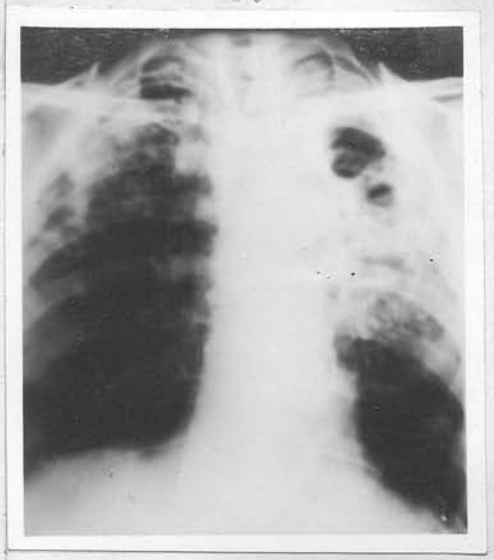


Fig. 7, Case 9. Generalised small mottling over whole lung very dense in middle and lower fields with patchy confluence over right side.

Fig. 8, Case 12. Generalised medium mottling with increased root shadows and emphysema.

9.



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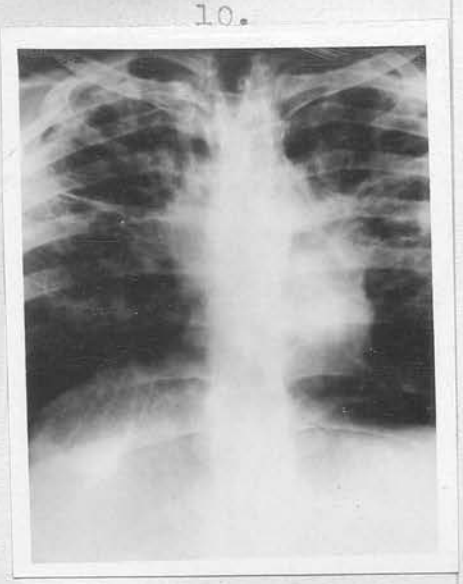
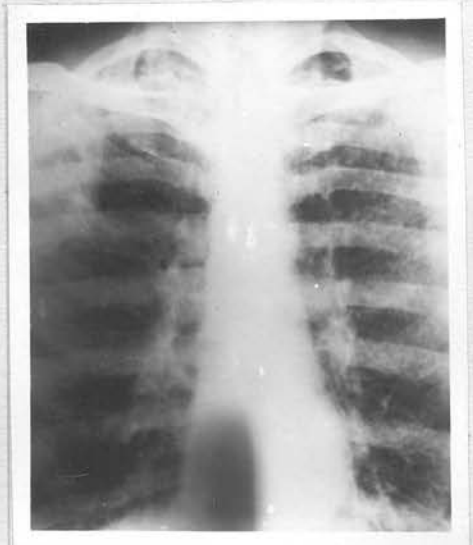


Figs. 9 and 10, Case 13. 24/8/31 and 14/12/32. Emphysema with large tuberculous cavity in upper lobe. Scattered conglomerate opacities in middle. Zone with diffuse mottling in lower lobe.

11.



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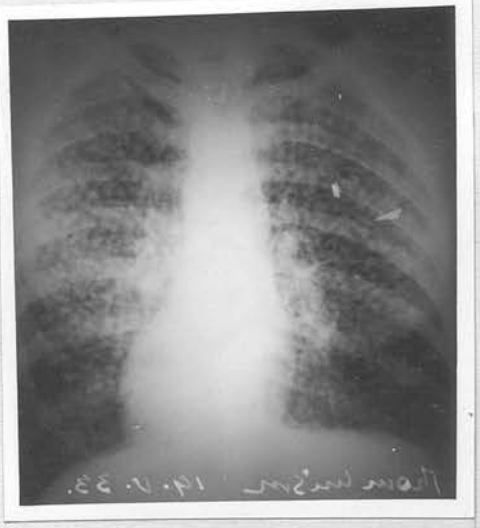
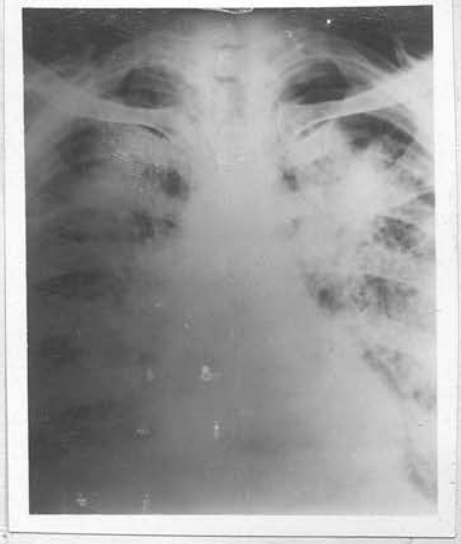


Fig. 11, Case 16. Increased root shadows. Generalised medium mottling with subpleural conglomeration at periphery.

Fig. 12, Case 18. Fine snow storm mottling characteristic of tubercle with increased root shadows, see Fig. 33.

13.



14.

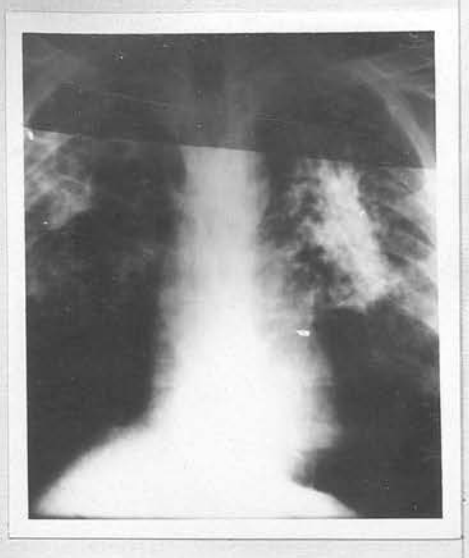
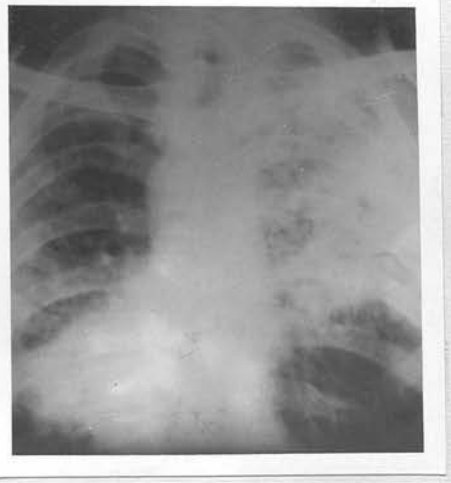


Fig. 13, Case 19. Coarse mottling over both lungs, confluent in both upper zones. Outline of heart and diaphragm obscured.

Fig. 14, Case 21. Generalised mottling over both lungs with conglomerations in both upper and middle zones, especially on the left. Thickened pleura throughout and emphysema in both lower lobes. See specimen No. 237.

15.



16.

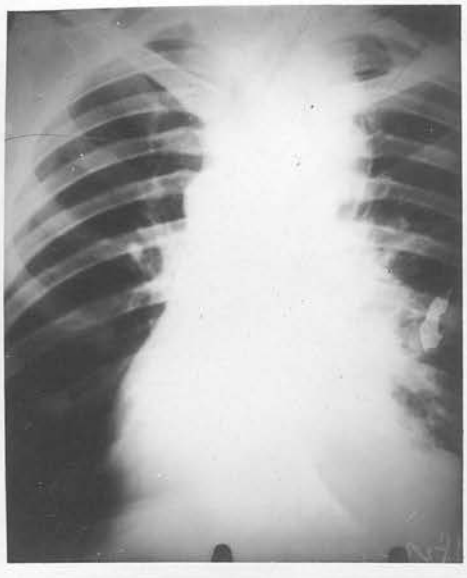


Fig. 15, P.M. 2/5513. The upper half of the right lung obscured by dense opacity with mottling extending to the base. The left lung shows diffuse mottling throughout. Specimen No. 5513.

Fig. 16, P. M. 4/206. Increased root shadow - carcinoma - no mottling or fibrosis.

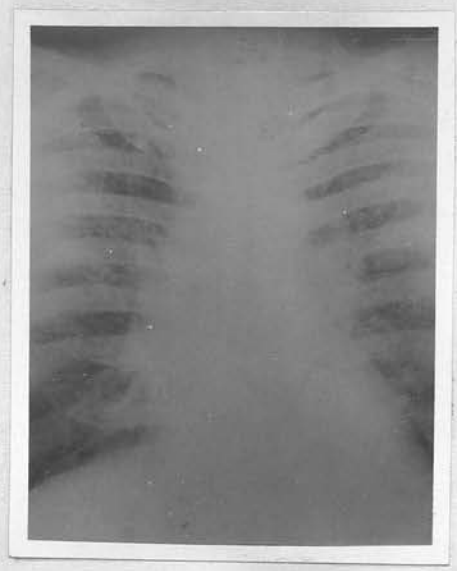


Fig. 17, P.M. 5/231. Fibrosis of both lungs with generalised mottling and thickened pleura.

18.

Fig. 18, P.M. 2/5513. See text for description.

19.

Fig. 19.
Portion of fibrosed upper lobe. The bright red areas are densely fibrosed and completely airless. The darker areas are tougher than normal and, in places, emphysematous.

20.

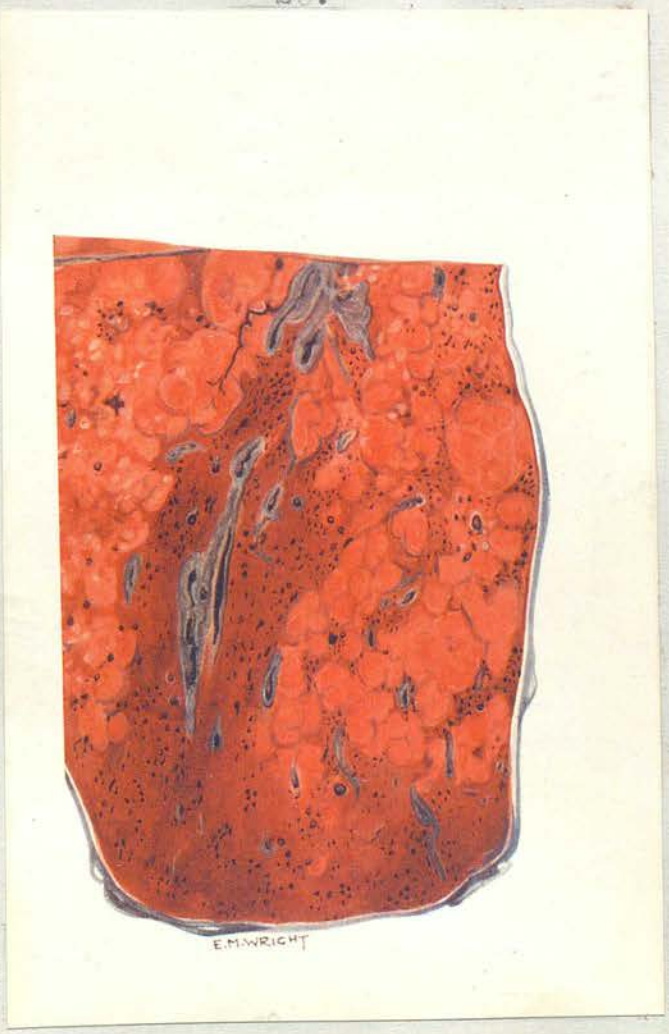


Fig. 20, P.M. 2/5513. There is a background of dark red crepitant emphysematous tissue with bright red densely hard rounded nodules and small foci of tuberculous caseation.

21.

Fig. 21, P.M. 5/231. Right lung showing tuberculous cavity at apex and emphysema and tubercle of lower lobe.

22.

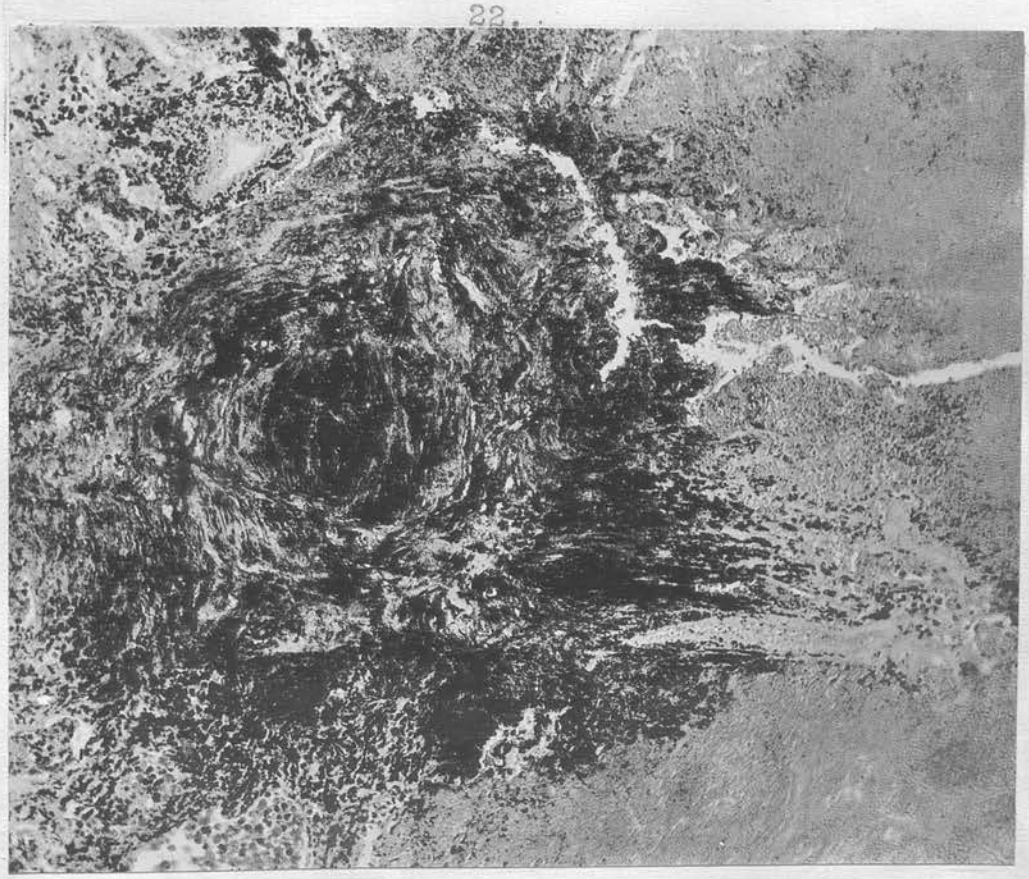


Fig. 22, P.M. 2/5513. Bronchial gland showing sidero-silicotic nodule with tubercle caseation free from dust.

23.

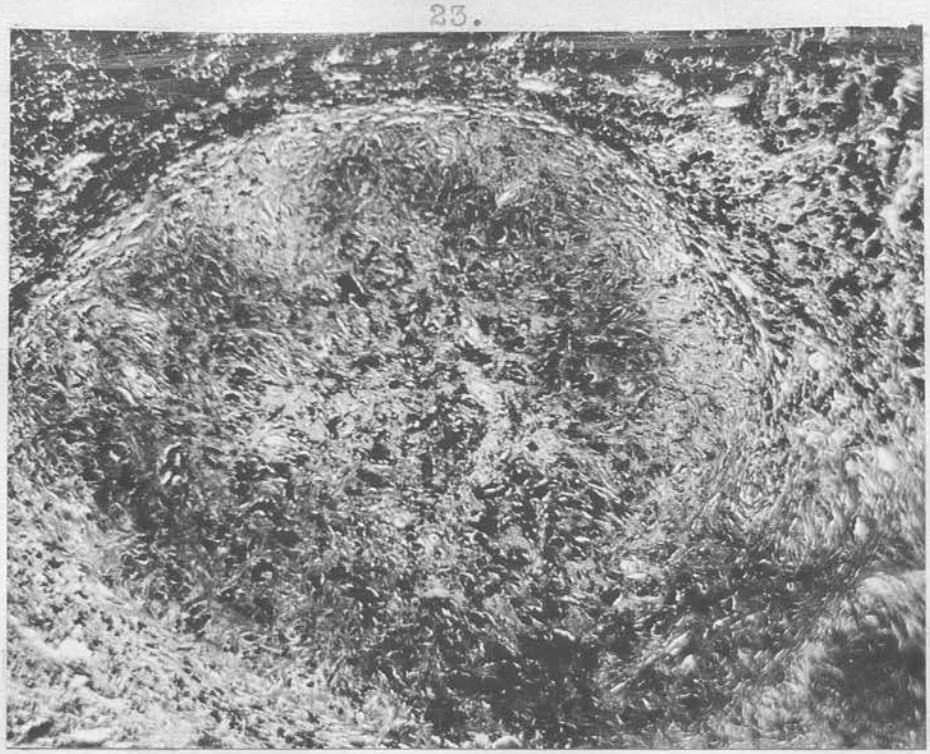


Fig. 23. Bronchial gland showing a silicotic type of nodule but the dust present is haematite.

24.

Fig. 24, P.M. 5/231. Massive type of fibrosis surrounding a partially obliterated vessel. Note the amount of Ferric Oxide deposited around the vessel and in surrounding tissue.

25.

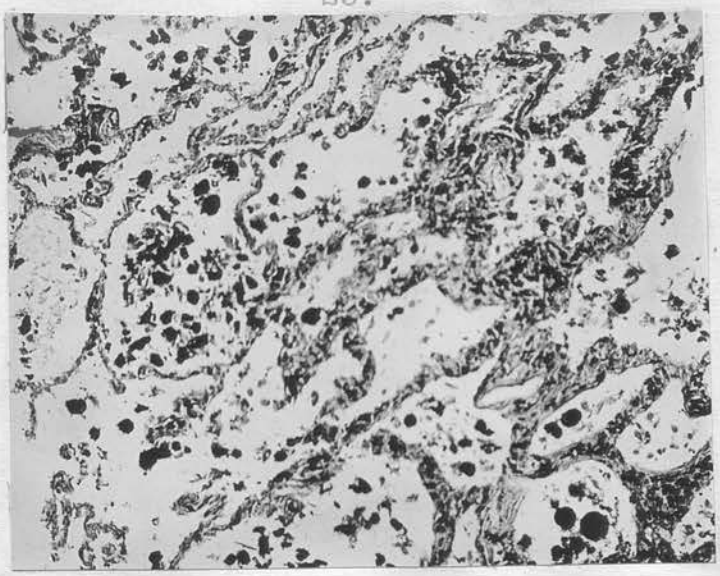


Fig. 25, P.M. 5/231. Alveoli adjacent to a fibrosed area showing desquamation of lining epithelium and phagocytes packed with iron and giving the Prussian Blue Reaction. Haematite can be seen lying in the alveolar wall.

26.

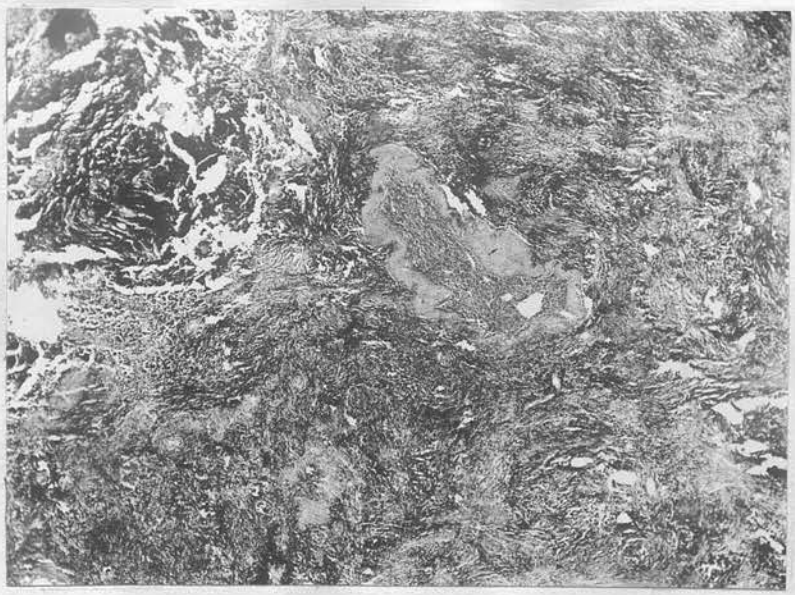


Fig. 26, P.M. 6/237. Massive fibrosis is seen around a blood vessel and a large deposit of haematite is present.

27.

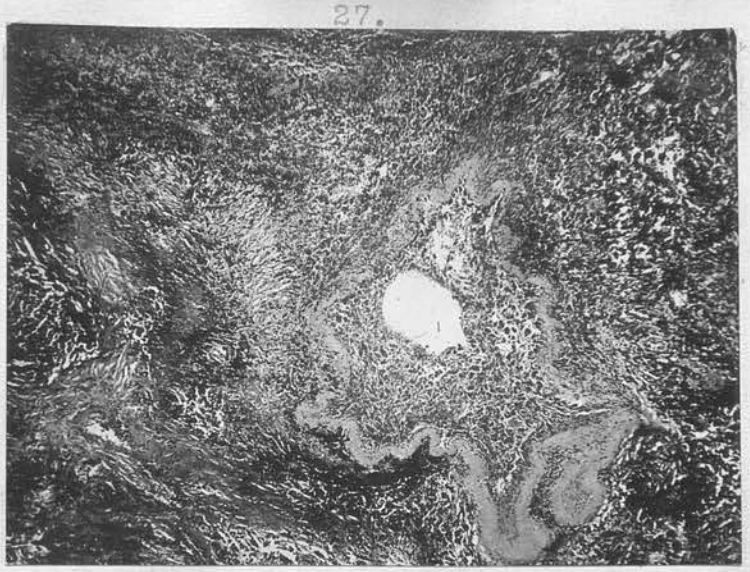


Fig. 27, P.M. 6/237. Endarteritis obliterans. Stained H. and E. There is destruction of the media with endothelial proliferation and large deposits of haematite in the surrounding fibrous tissue.

28.

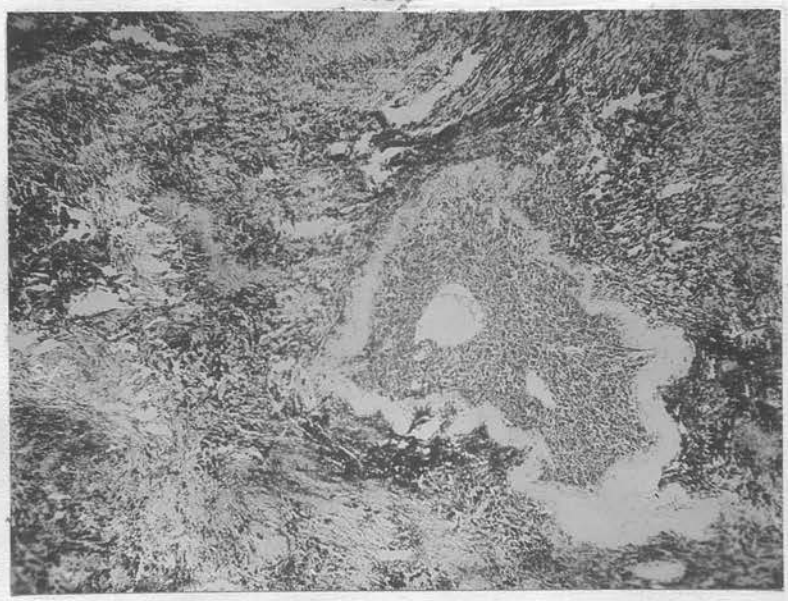


Fig. 28. Another section of the same artery simply stained by K_4FeCN_6 to show the amount of iron held by the endothelial cells.

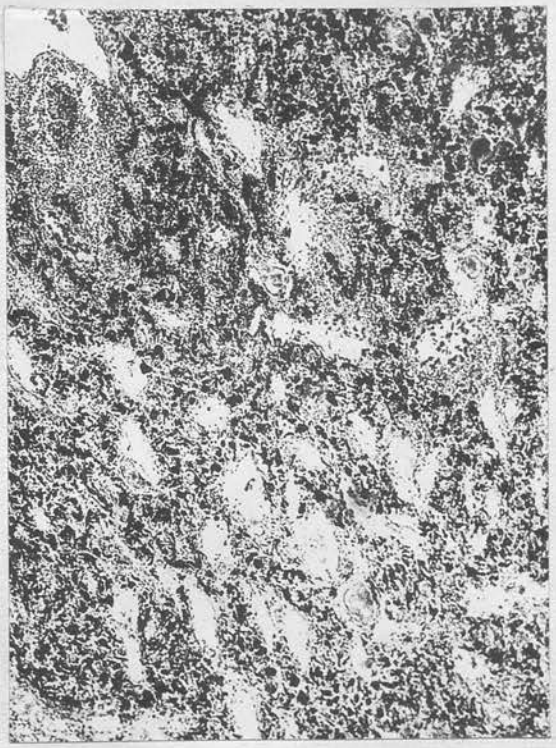


Fig. 29, P. M. 6/237. Anthracosis and haematite dust deposited in a bronchial gland with little fibrosis and no tubercle.

30.

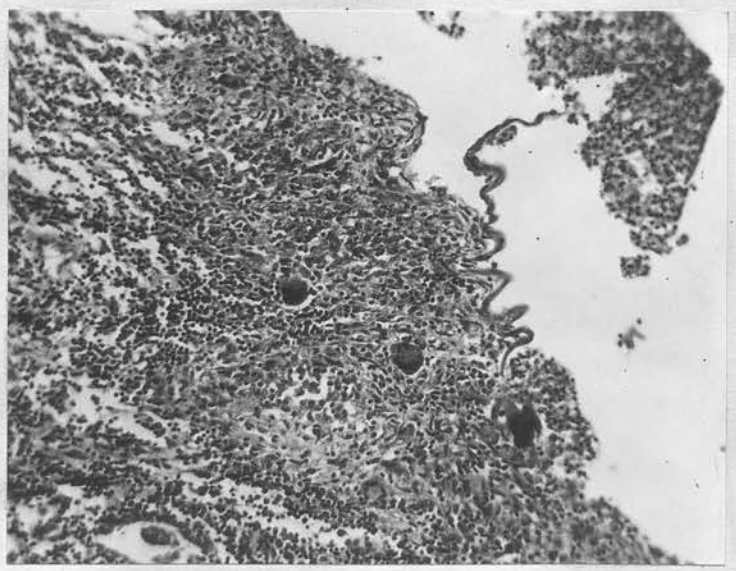


Fig. 30, P. M. 7/240. Edge of a tuberculous bronchiolitis showing desquamation of lining epithelium and lumen filled with pus.

31.

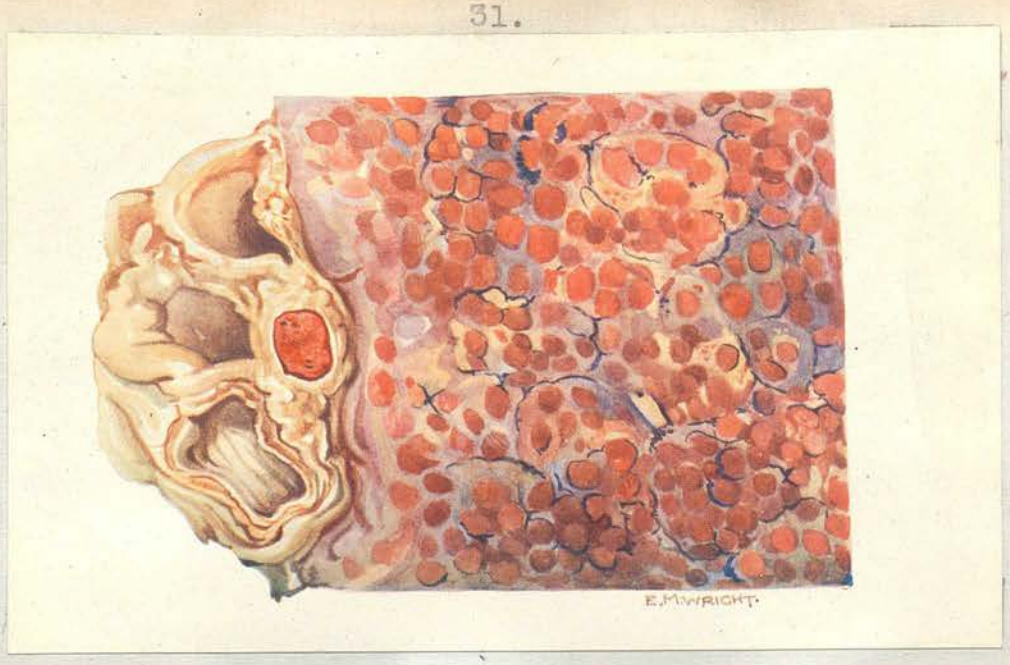


Fig. 31, P. M. 9/340. Pleural aspect of right lung (mid posterior portion) shows emphysematous bullae containing bright red deposits of haematite separated by bands of fibrous tissue. The bronchial gland shows intense siderosis.

32.

Fig. 32, P. M. 9/340. Dense massive fibrosis of upper lobe showing characteristic aggregations of haematite dust surrounded by collagenous fibrous tissue and, at periphery, a few macrophages.

Fig. 33, P. M. 10/346. Right lung showing areas of fibrosis in upper lobe and emphysema. Hylar glands are only moderately enlarged.

34.

Fig. 34, P. M. 10/346. Massive silico-siderotic fibrosis with no recognisable arteries or bronchi.

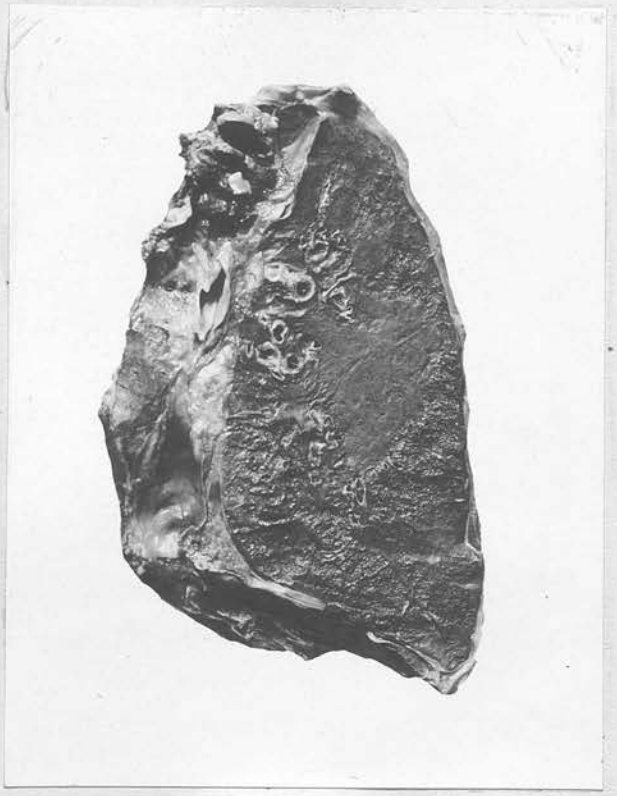


Fig. 35, P. M. 11/348. Right lung showing intense fibrosis with prominent bronchi, grossly thickened pleura and enlarged mediastinal glands.

36.

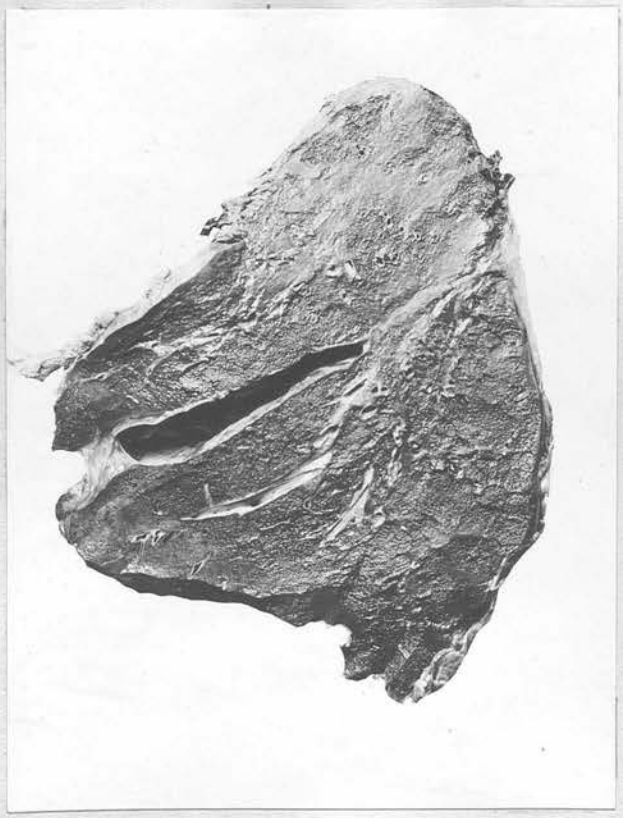


Fig. 36, P. M. 11/348. Left lung showing fibrosis of upper lobe with much peribronchial fibrosis and irregular thickening of pleura.

37.

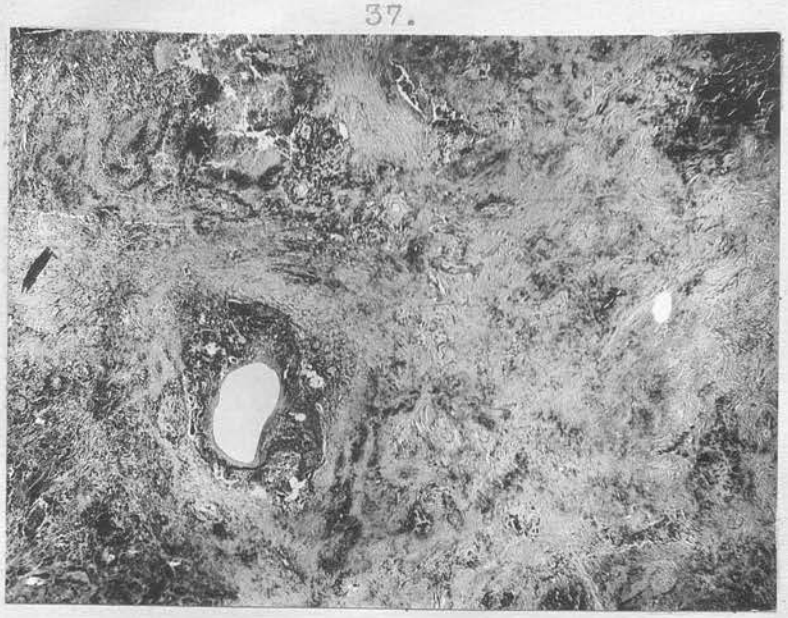


Fig. 37, P. M. 11/348. Section of upper lobe showing intense fibrosis. No alveoli visible and large areas of airless lung with varying amounts of haematite, more intense around the vessel in the old lymphatics.

38.

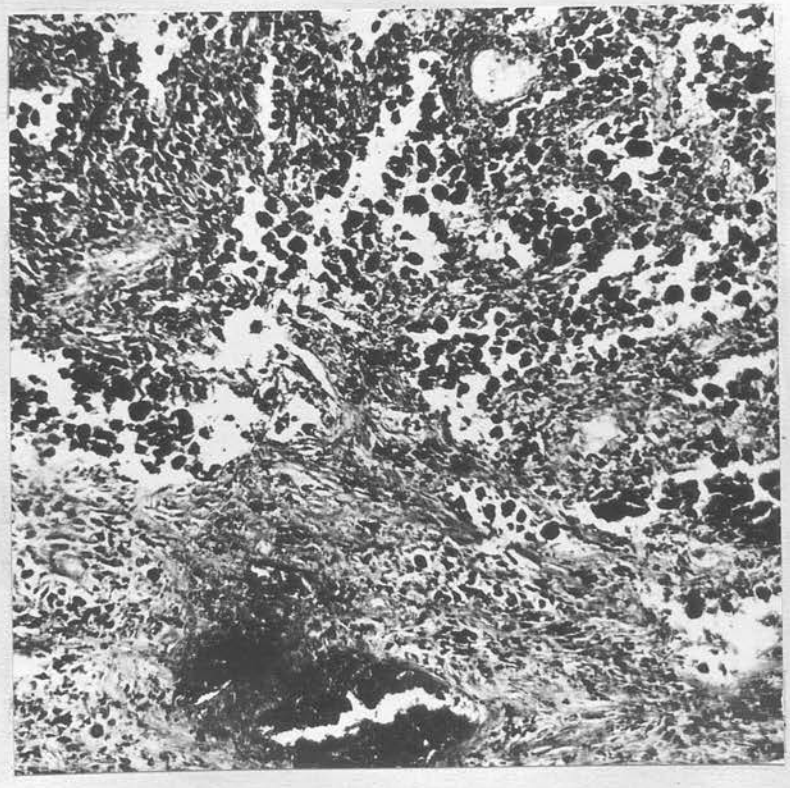


Fig. 38, P. M. 11/348. Alveoli surround a densely fibrosed area filled with phagocytes and desquamated epithelial cells. The outline of the haematite is all that indicates that it is contained within phagocytes because the cellular anatomy is obscured.

39.

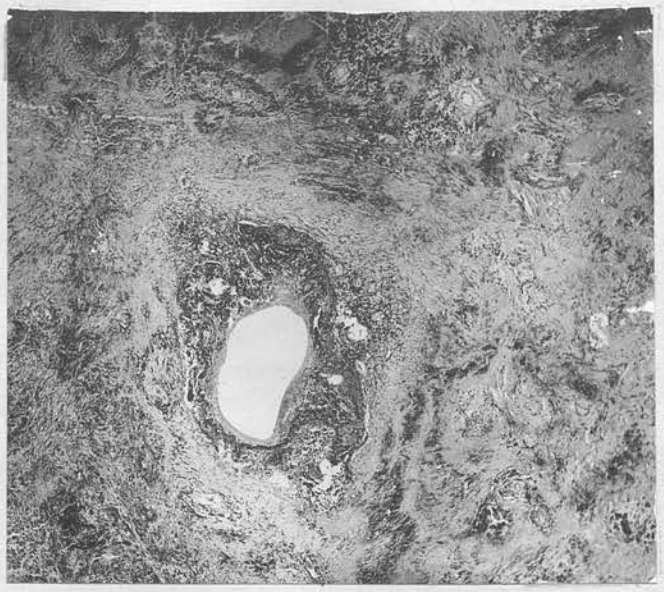


Fig. 39, P. M. 11/348. Periarterial fibrosis with early thickening of media due to fibrosis of muscular coat and increase of adventitia. Note the iron deposited within the coats of the vessel wall carried there by phagocytes.

40.

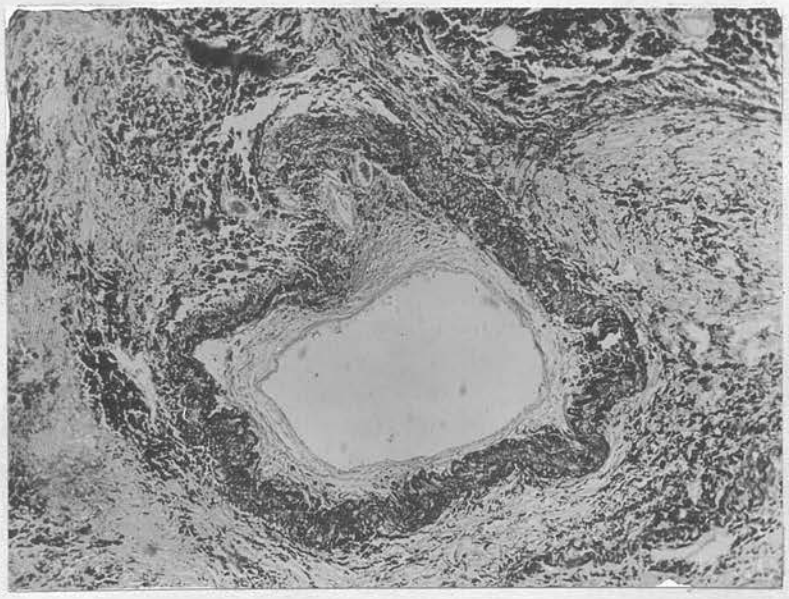


Fig. 40. Same vessel as Fig. 39 but higher magnification stained by Weigert. Notice destruction of elastic coat and deposit of iron throughout. There is subintimal thickening.

41.

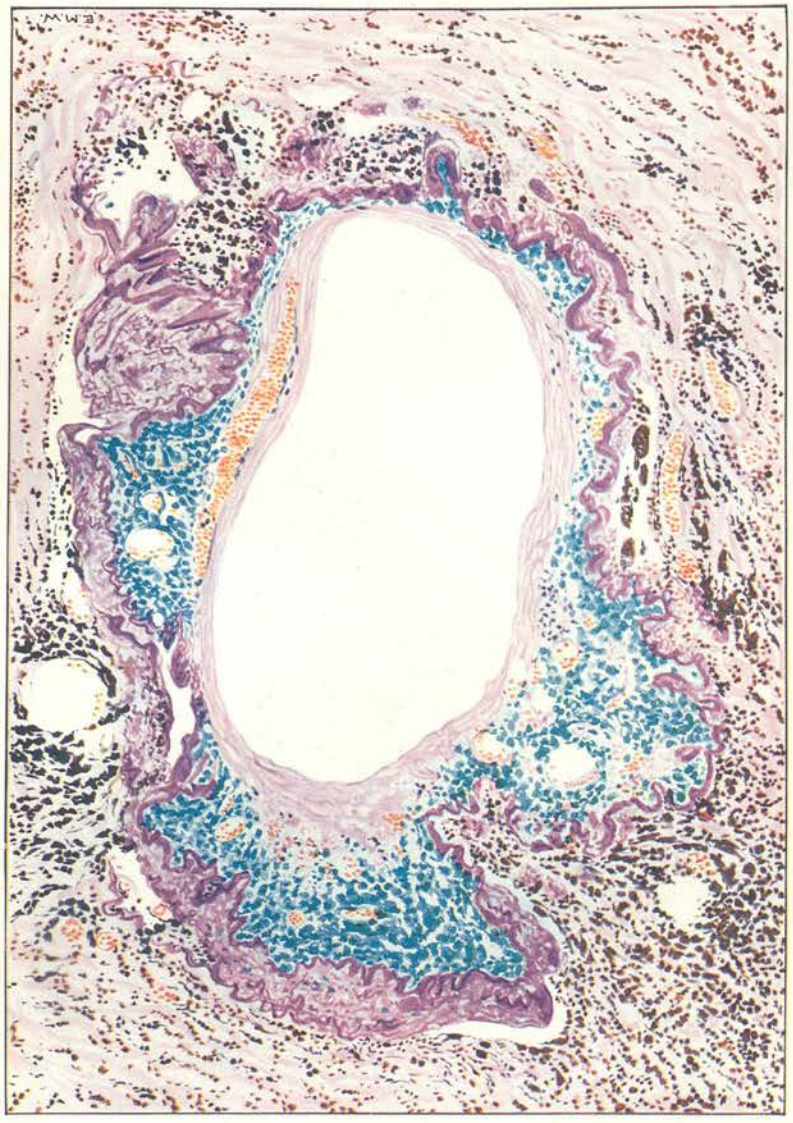


Fig. 41. The drawing is from a further section of the same vessel and shows rupture of Elastica and subintimal staining with Prussian Blue to show presence of haemosiderin. Observe the iron as oxide is Prussian Blue negative and lies outside the elastic coat.

42.

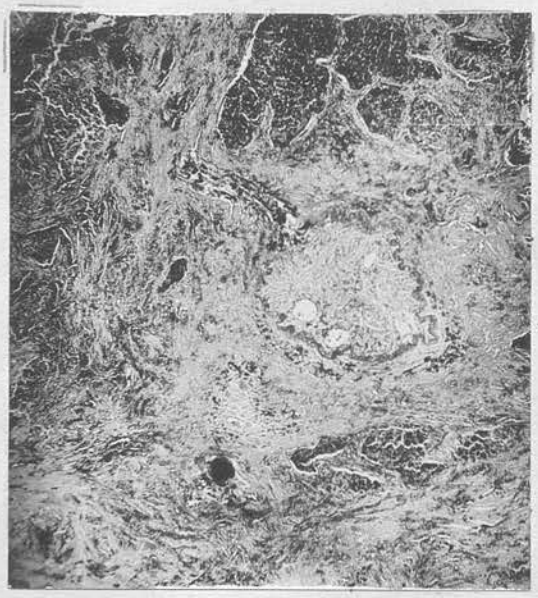


Fig. 42, P. M. 11/348. A further stage in the process of endarteritis with only the Elastica remaining. Section stained by Weigert Elastica and the lumen largely occupied by hypertrophied intima.

43.

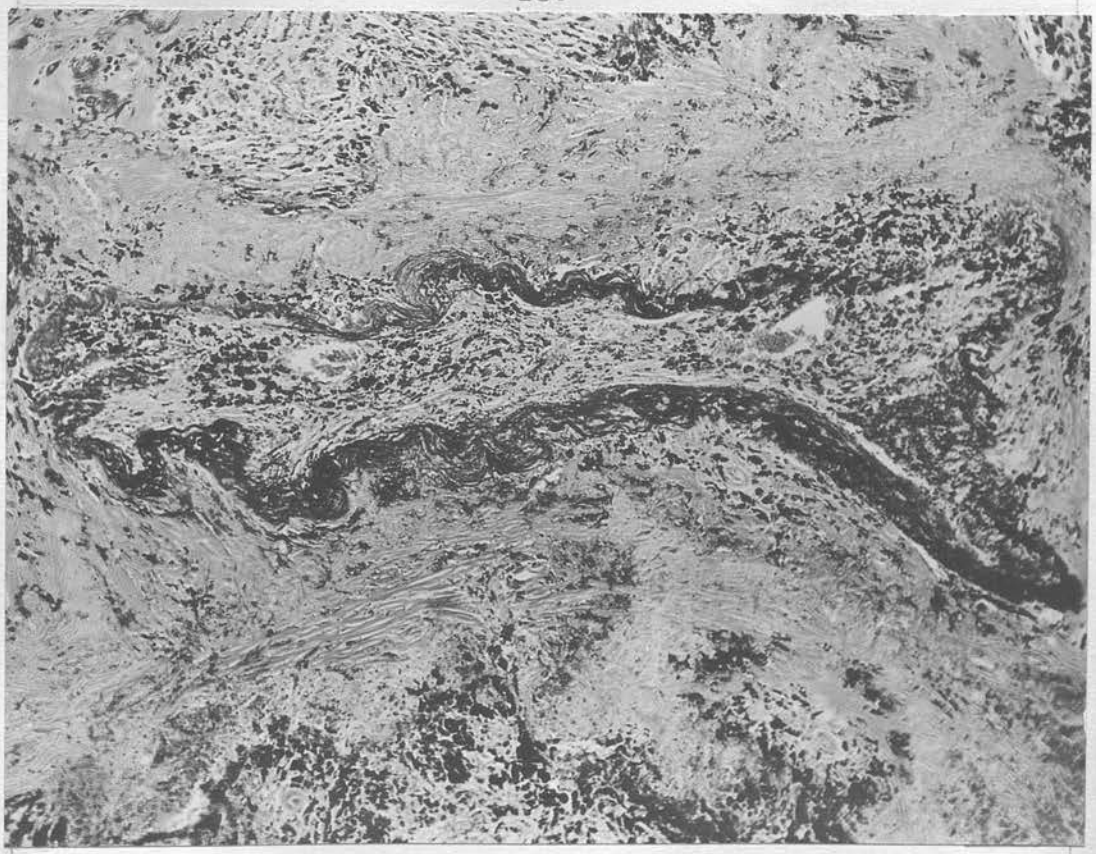


Fig. 43, P. M. 11/348. Owing to pressure of contracting fibrous tissue the vessel walls come to lie in apposition and so help the obliterative process. There is here also destruction of the elastic fibers and infiltration of all the coats with haematite.

44.

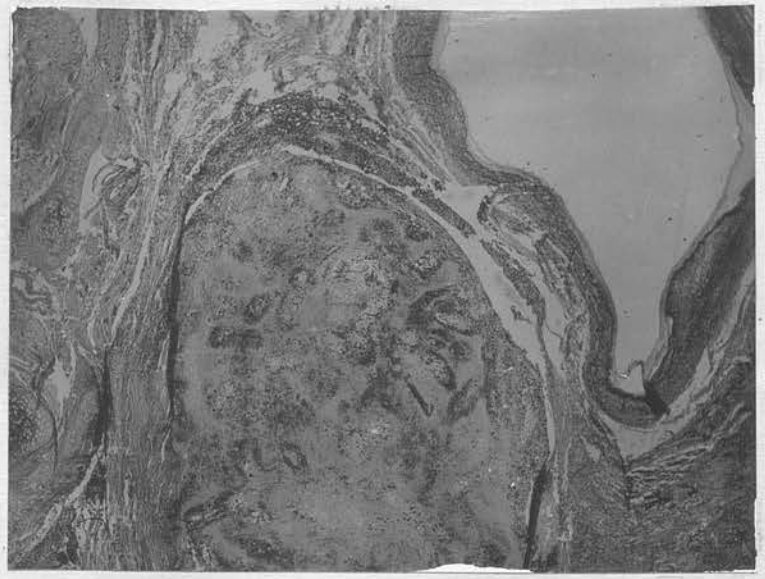


Fig. 44, P. M. 11/348. Normal artery with a typical silicotic nodule, fairly well encapsulated, lying alongside.

45.

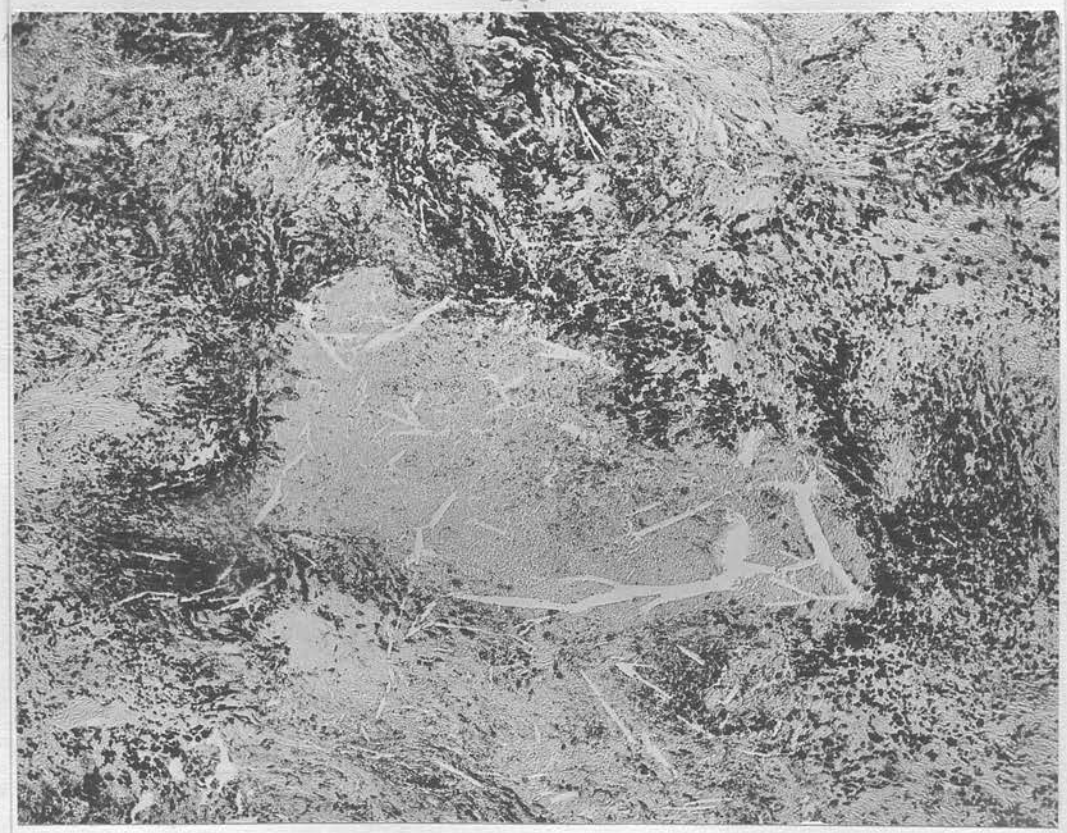


Fig. 45, P. M. 12. A central focus of tuberculous caseation with cholesterin clefts, indicating age, and surrounded by dust. This suggests that the tuberculous focus preceded the fibrosis.

46.

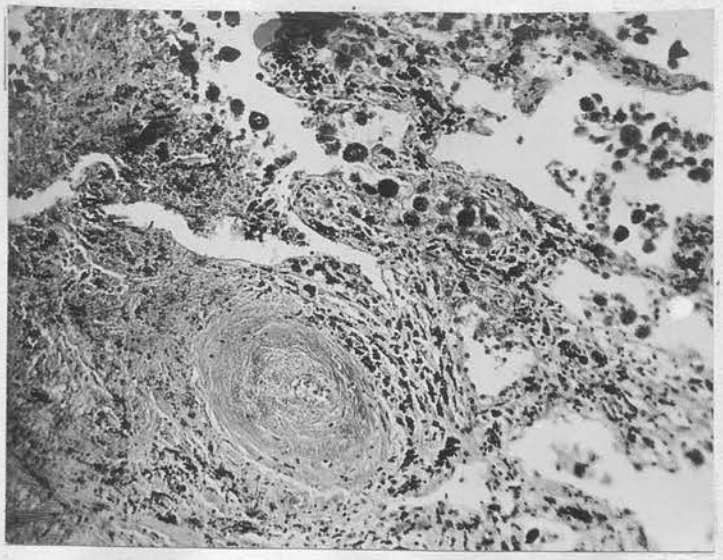


Fig. 46, P. M. 12. A small early nodule of Silicosis at the edge of an alveolus. The centre is fibronecrotic, not caseous, and contains granules of dust. From the appearance, it is considered to have arisen in a lymphatic. The surrounding alveoli contain phagocytes packed with dust.

47.

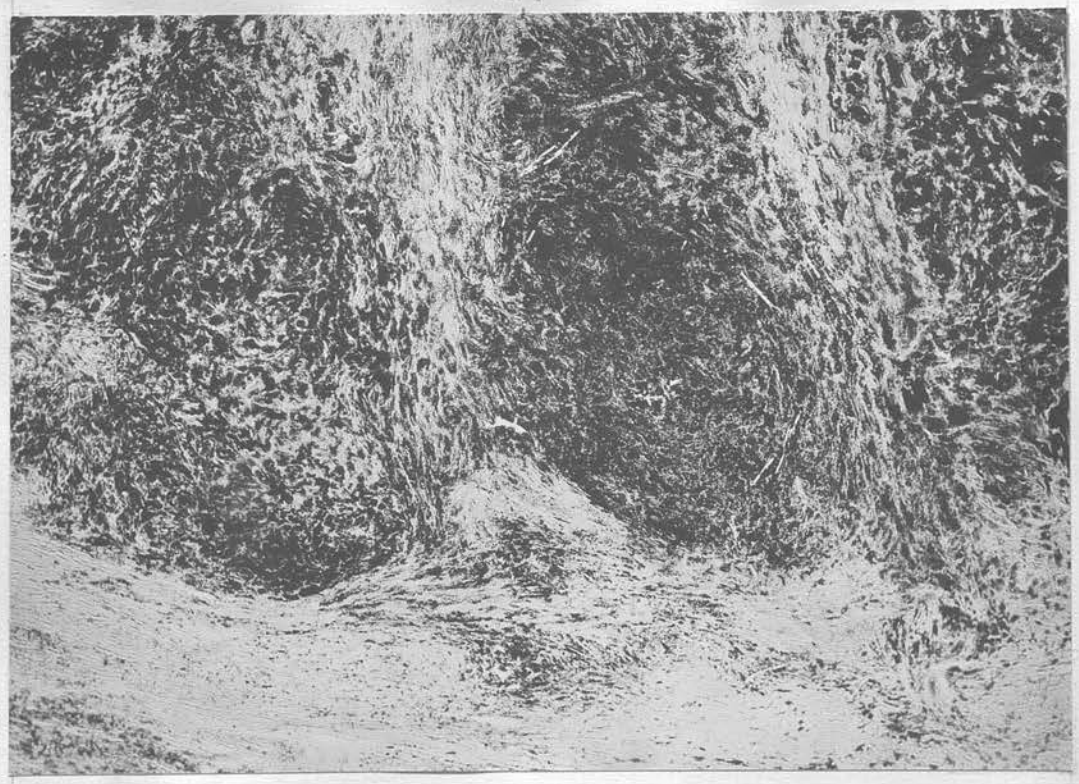


Fig. 47, P. M. 12. Edge of silico-siderotic nodule showing the heavy deposit of dust and resulting massive fibrosis.

48.

Fig. 48, P. M. 13/368. Left lung showing fibrosis and emphysema with thickened pleura. Scale 2/7.

49.

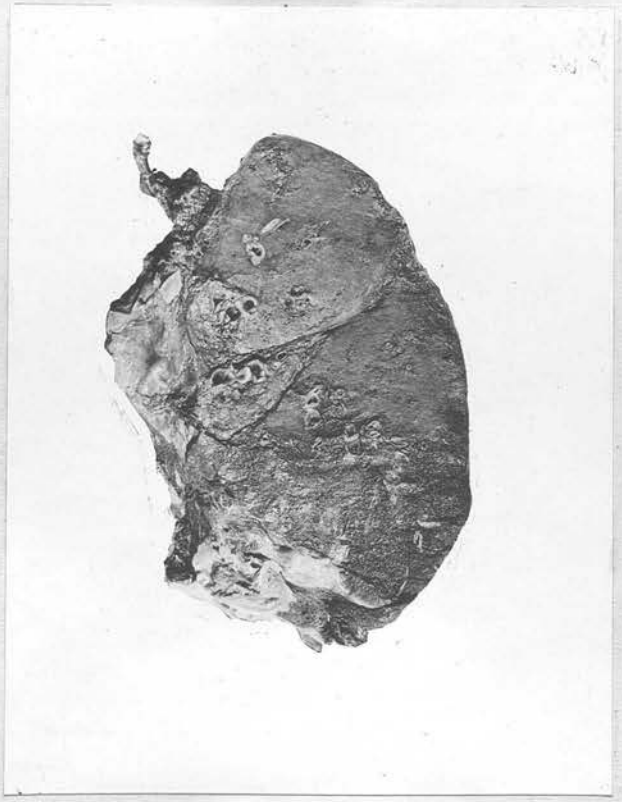


Fig. 49, P. M. 13/369. Right lung showing massive fibrosis and emphysema of lower lobe with dilated bronchi. Scale 1/3.

50.

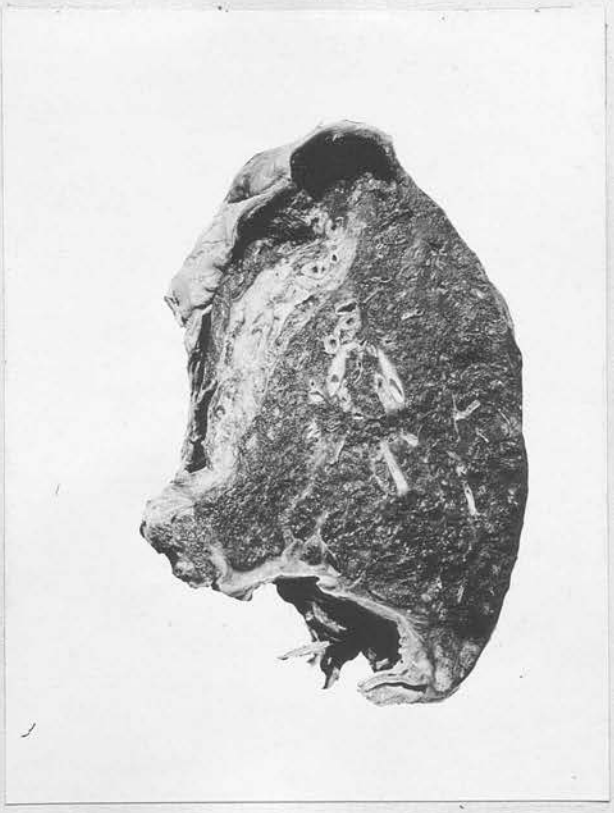


Fig. 50, P. M. 14/370. Left lung showing thickened pleura and large cavity in upper lobe. The nodular appearance of the cut surface can be made with small foci of fibrosis. Frank tuberculous caseation present at base. Scale 2/5.

51.

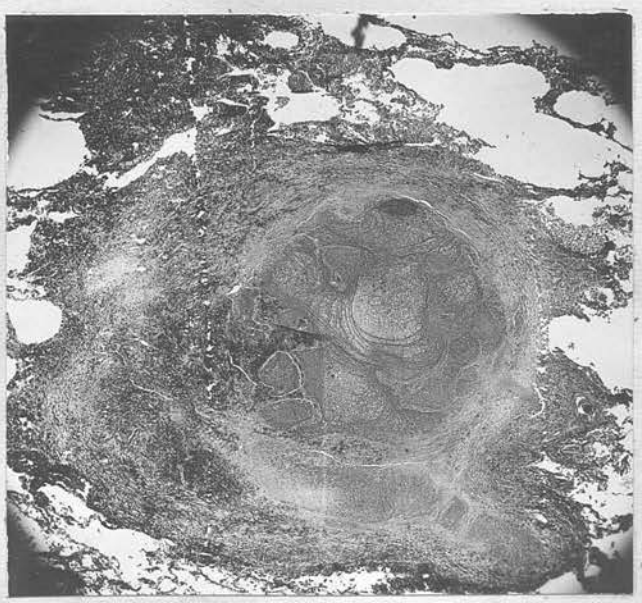


Fig. 51, P. M. 14/370. Nodule of tuberculous caseation, free from dust, with evidence of active spreading.

52.

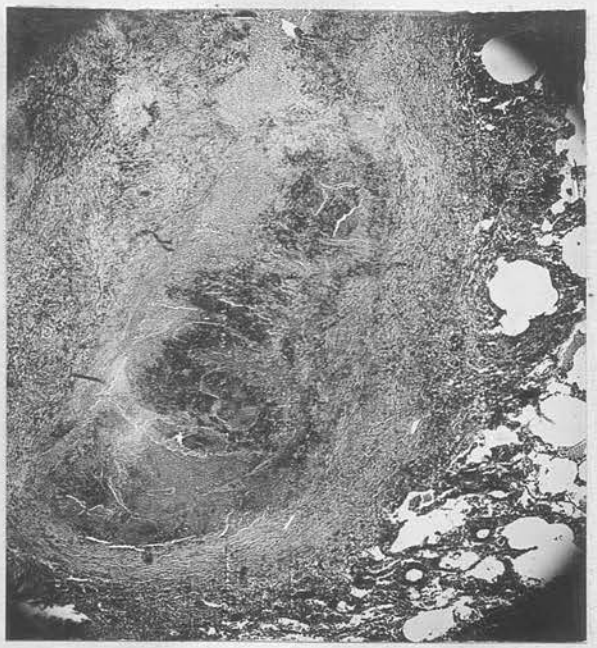


Fig. 52, P. M. 14/370. Nodule of silico-siderosis with central fibrosis and a few areas of tuberculous caseation - quite unlike previous section.

53.

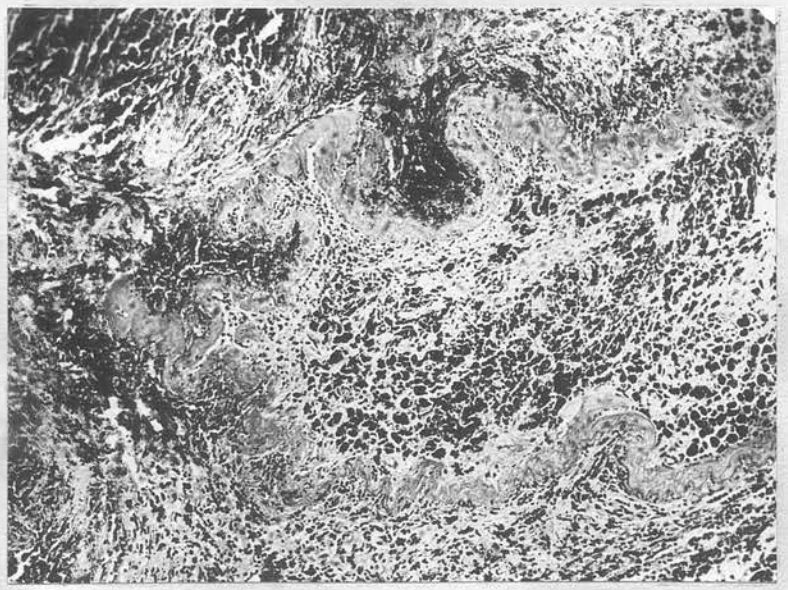


Fig. 53, P. M. 6/237. Endarteritis obliterans stained Weigert, KFeCN. Elastica tortuous and lumen completely occluded by endothelium stained blue. Muscle coat disappeared and replaced by fibrous tissue.

54.

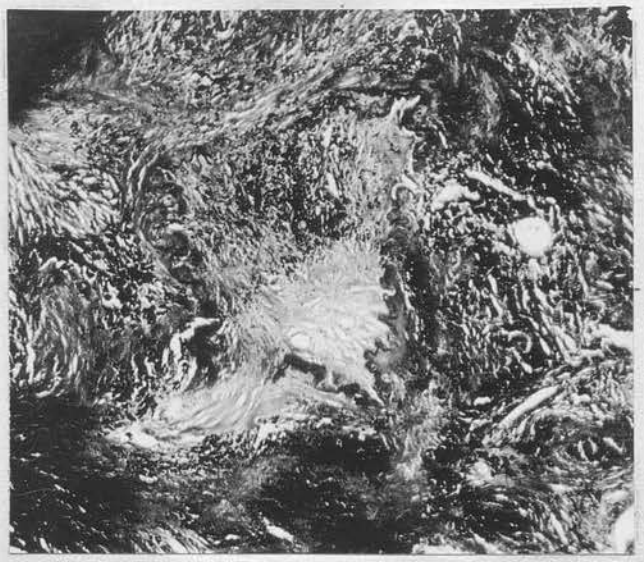


Fig. 54, P. M. 11/348. End result of endarteritis obliterans. An irregular outline of elastic fibers embedded in a collagenous mass with deposit of ferric oxide occupying the centre of the obliterated lumen.

55.

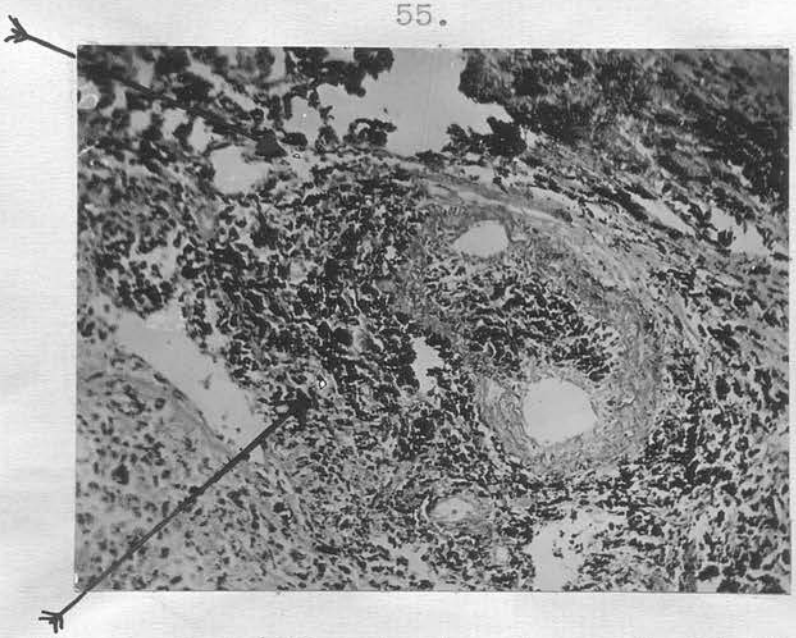


Fig. 55, P. M. 5/231. Early stage of endarteritis obliterans in a bifurcating vessel showing infiltration of media and subintimal proliferation. Two crystals of quartz are visible in the picture at end of arrows. Though these do not show up without polarising the light, frequently they can be demonstrated by photography.

56.

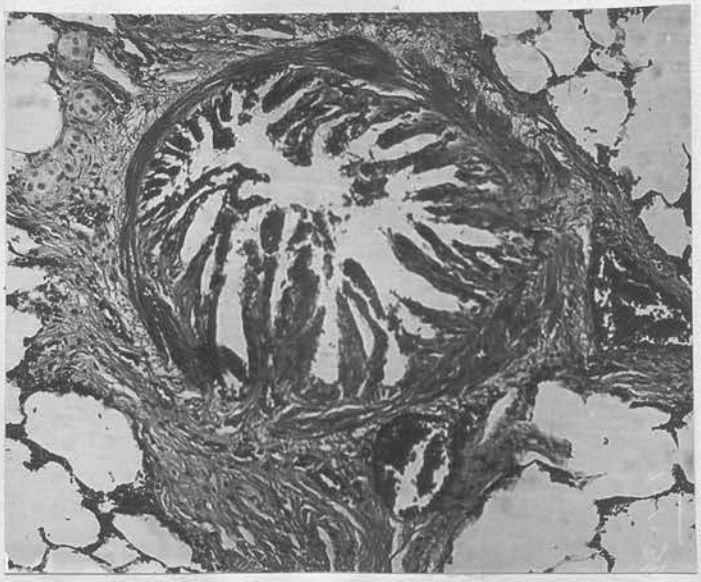


Fig. 56. Bronchus from guinea pig surrounded by muscular tissue. No fibrosis and no dust retained. Animal survived 10 months after injection of iron oxide.

57.

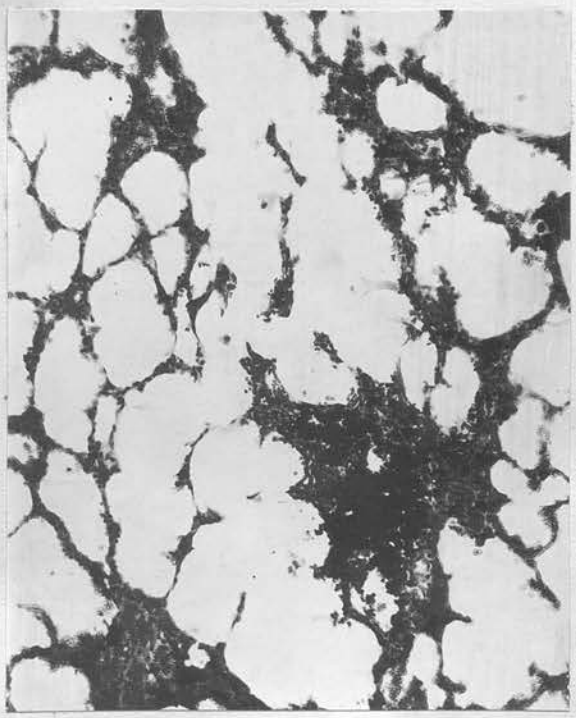


Fig. 57. Lung from guinea pig which survived only six weeks; marked deposit of iron but no reaction.

58.

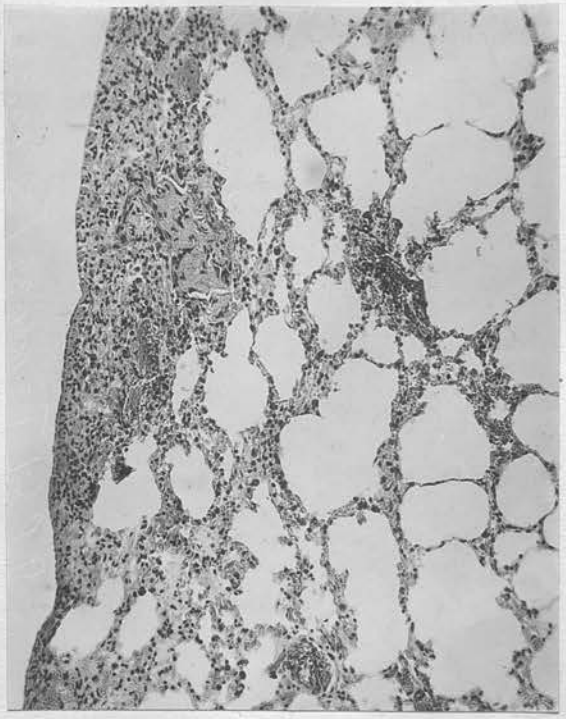


Fig. 58. Subpleural deposit of iron with mild cellular reaction. Very little dust was left in the lungs after 9 weeks.

59.

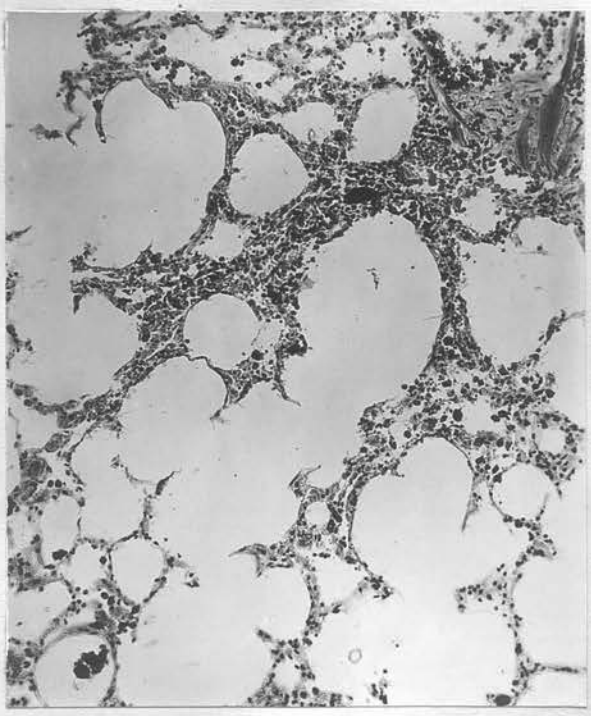


Fig. 59. Guinea Pig lung four weeks after injection showing emphysema and slight alveolar thickening with iron oxide inside the phagocytes.

60.

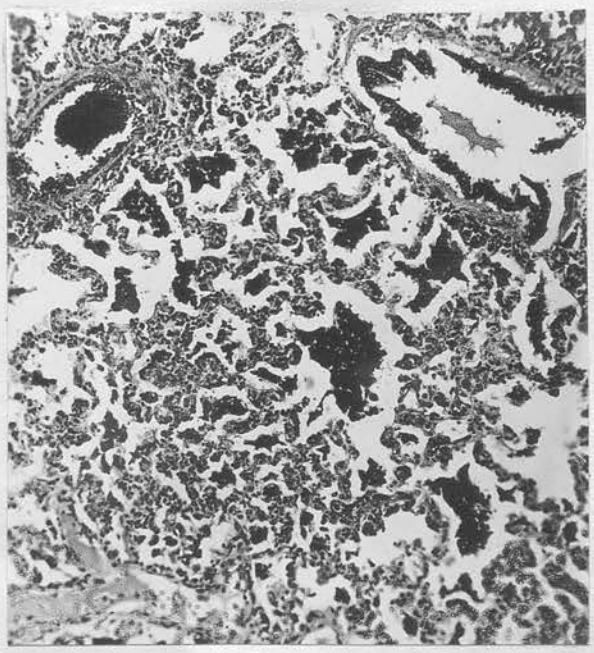


Fig. 60. Rabbit 2, 4 injections. There is collapse of the alveoli with iron dust visible both in alveoli and inside bronchi. There is no Fibrosis.

61.

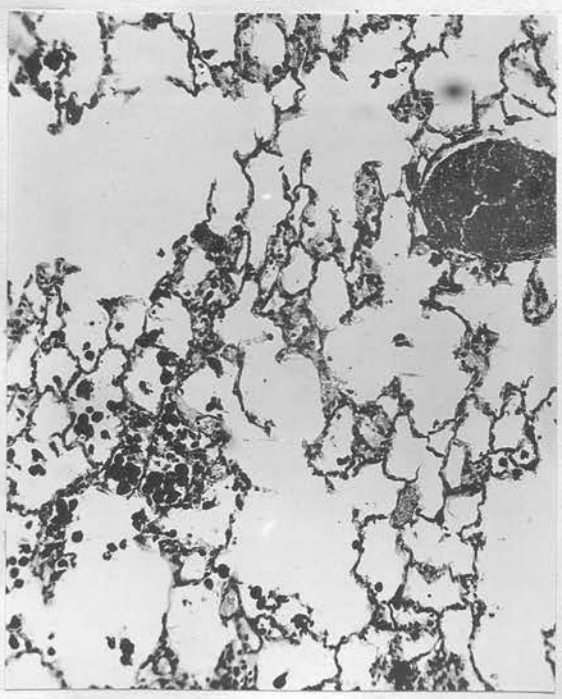


Fig. 61. Rabbit 3, 2 months after last injection of Fe_2O_3 showing emphysema and small amount of iron remaining lying in the alveoli inside phagocytes.

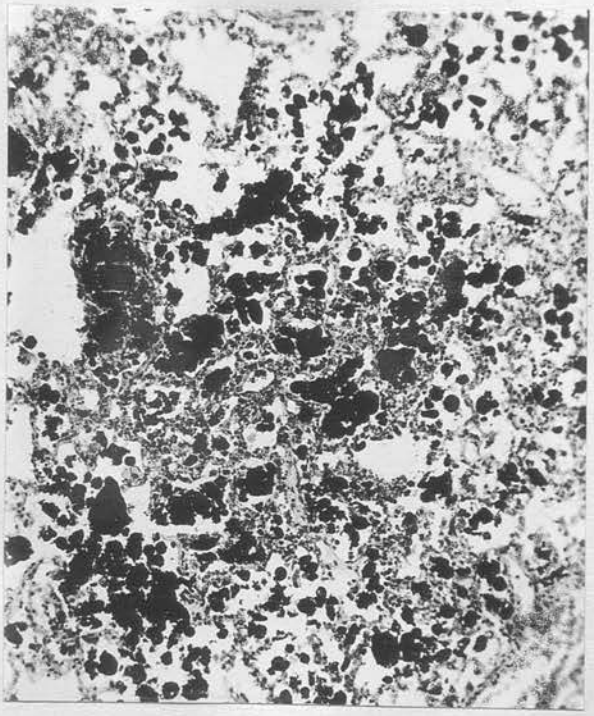


Fig. 62. Rabbit 4, died a week after injection and section shows the amount of iron reaching the alveoli with oedema and congestion of the alveolar wall.

63.

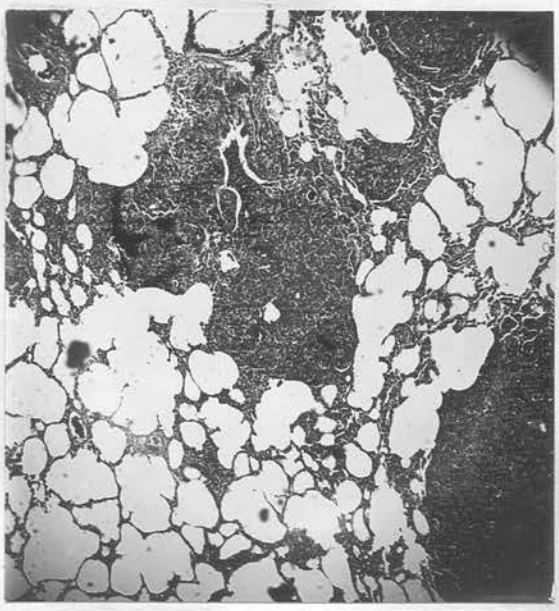


Fig. 63. Rabbit 8. Iron-ore and Tubercle 6 weeks after injection showing the reaction with retention of iron-ore.

64.

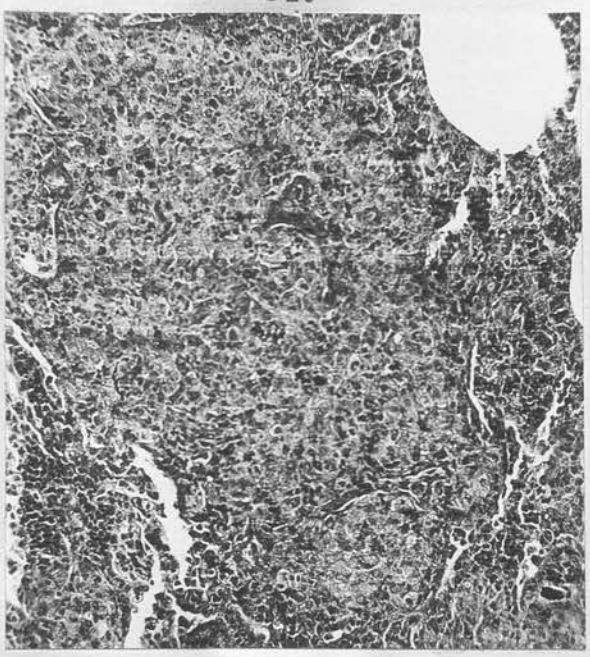


Fig. 64. Rabbit 8. High power view of 63. There is a hyperplastic tuberculous reaction with necrosis and retention of dust.

65.

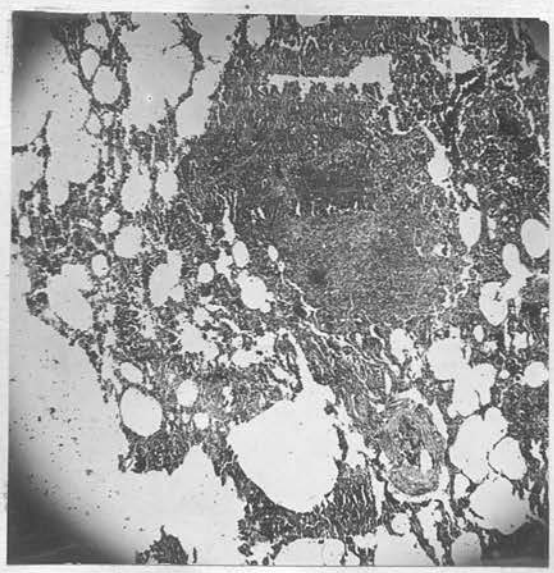


Fig. 65. Rabbit 10. Fibro-necrotic nodule with iron dust and surrounded by thickened alveoli. Result of 2 injections of iron-ore and Tubercle.

66.

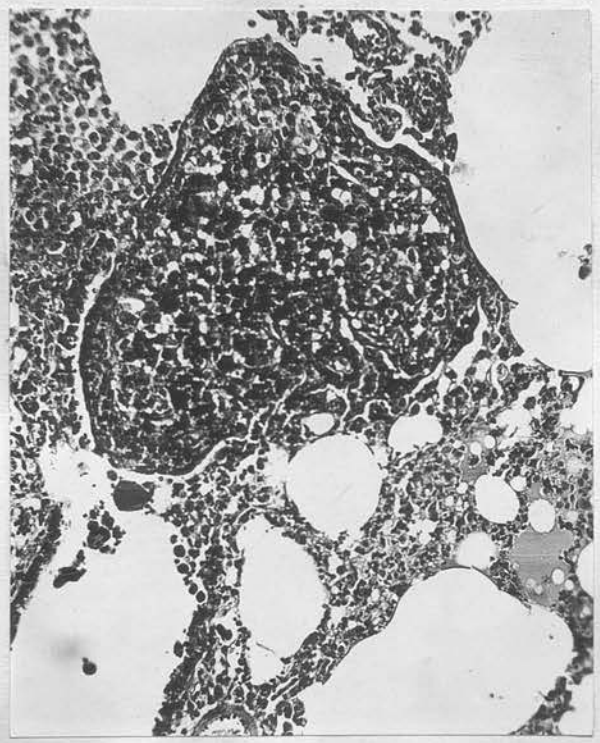


Fig. 66. High power view of nodule from Rabbit 10. There is a nodule of endothelial proliferation and heavy deposit of iron dust - probably an obstructed lymphatic vessel.

APPENDIX C. - TABLES.

TABLE 1.

DATE.	AVERAGE ANNUAL OUTPUT OF ORE.	AVERAGE ANNUAL NO. EMPLOYED.	AVERAGE ANNUAL OUTPUT PER HEAD.	PRICE.
1845 - 55	100,000/200,000			
1870 - 74	1,158,109			
75 - 79	1,287,643			19/6
80 - 84	1,533,633			
85 - 89	1,426,917			12/1
90 - 94	1,368,605			
95 - 99	1,235,728	4674	264	11/10
1900 - 04	1,093,401	4625	236	
05 - 09	1,245,194	4652	268	
1910 - 1914	1,294,423	5142	252	
1921	243,065	1916	127	
1922	625,935	3166	198	
1923	982,661	4205	234	
1924	901,283	3528	255	
1925	809,769	3009	269	
1926	422,191	2822	150	
1927	1,070,662	2769	387	
1928	1,009,945	2679	377	
1929	1,245,447	2891	431	
1930	1,036,111	2932	353	
1931	613,322	2134	287	

Top Prices 1872 - 28/-
1873 - 31/-
1874 - 24/-

TABLE 11 A.

HODBARROW MINES. PROPORTION TO ALL CUMBERLAND MINES EXPRESSED AS %.

1870	15.13
1880	24.18
1890	43.71
1900	59.64

TABLE 11 B.

	1930 No. of Men.
Beckermet Mining Co.	459.
Bigrigg.	238.
United Steel Co.	93.
Ullcoats Mining Co.	339.
Millom and Askham.	459.
Hodbarrow Mining Co.	608.
TOTAL ---	2,196.

In 1930, 4 Mining Companies supplied 77% of the total output for Cumberland. Florence, Ullcoats, Beckermet and Hodbarrow employing 2196 men.

TABLE 111.

SHOWING PROPORTION OF MACHINE DRILLERS TO ALL MALES OVER 16 EMPLOYED BELOW GROUND.

1925	28%
1929	36%
1930	38%
1931	44%

TABLE 1V.

Average output in tons per man in 46 hours.

Hand drill and loading own ore--------10 tons.

Machine " " " " " --------23 "

" " and labourer loading ore---27 " .

TABLE V.

Explosives per head.

1913.----- 78 lb. per person employed.

1929.----- 225 lb. " " "

TABLE V1.

	AGE.	DATE OF FIRST EXAMINATION	PITS EXPERIENCE IN YEARS.	HAMMER & JUMPER.	DRY DRILL.	WET DRILL.	CHEST EXP.	SPUTUM TUBERCLE.	DISABILITY TIME MONTHS	
J. At. 1.	52	12.33	(F.1 (B.20	5	6	9	1½	0	-	Disability definite but still considers fit to work. Unemployed.
Caw. 2.	55	12.33	(Bg.25 (F. 11	25	6	5	1"	+	7	Unfit for Work.
J. Cav. 3.	44	11.33	(K.F (S.M) 30	14	16	-	Nil	-	6	Unfit for Work.
J. D. 4.	56	11.33	Wo.33	15	15	-	-	0	20	Emphysema.
R. F. 5.	48	11.33	(Ph. (B. (Bg.) 20	13	5	2	-	+	13	Acute pulmonary Tuberculosis Dying - P.M.340.
E. F. 6.	65	12.33	B.16	-	-	-	¼	-	-	Distinctly handicapped. Still working.
J. Ga. 7.	50	8.32	B.34	24	8	2	½"	+	3	Emphysema for 5 years - unfit for work. Died June, 1934. No P.M.
J. Gi. 8.	61	12.33	(Le.10 (M. 4 (Lo.3 (B. 14	18	14	-	¼"	-	48	Unemployed for 4 years, now unfit. Died. P.M.348.
E. G. 9.	48	8.32	(F.La (M. 21	11	-9-		Nil	0	24	Breathlessness and Emphysema. Unfit to work.
J. Gri. 10.	62	12.33	(Wy.34 (B. 16	34	9	7	-	+	3	Confined to bed. T.B.Pleurisy Died, May 1934 - No P.M.
H. M. 11.	42	8.32	(Bg.8 (B.18	13	-13-		-	0	9	Definite disability. Unemployed.
H. H. 12.	53	8.32	Bg.22	4	15	3	¼	0	5	Emphysema. Unfit for work.
R. M. 13.	58	8.32	(Bg.3 (L. 18	21	-	-	-	0	36	Marked Emphysema. Fit for light work.
R. P. 14	50	11.33	(F. (U. 12	-	-12-		Nil	0	18	Totally disabled. Died P.M.368.
W. P. 15	46	12.33	(Ub.9 (U.13	9	12	1	¼"	-	24	Emphysema.
R. R. 16	45	12.33	(B.15 (F.5	15	5	-	-	-	-	Moderate disability. Unemployed.
J.S. 17	50	12.33	(E.12 (Fa.4 (F.12	19	9	-	¼"	+	48	At Work. (Light job.)
J. T. 18	65	-	(Wo.14 (U. 12 (B. 17	-	-		-	+	36	Totally unfit. Died. P.M.346.
J. WM. 19	64	12.33	(Ph.6 (B. 9	-	-	-	¾"	-	13	Unfit for Work.
H. WM. 20	57	12.33	(P.25 (F.8	25	-8-		¼"	0	-	Fit for Work.
H. W. 21	55	8.32	B.20	10	10	-	Nil	-	48	Unfit for Work for 2 years. Died. P.M. 237.

B. Beckermet - United Steels.
E. Elder, closed.
Bg. Bigrigg.
F. Florence. Millom and Ashkam.
Fa. Falson - closed.
Le. Lendal - closed.
Lo. Lonsdale- closed.
L. Lindow - closed.
La. Lamplugh - closed.
M.B. Moss Bay.
M. Margaret - closed.
K. Knockmartin - closed.
P. Parkside - closed.
Ph. Parkhouse - closed.
U. Ullcoats - Millom and Askham.
Ub. Ullbank - closed.
S. Salter - closed.
Wy. Wyndham - closed.
Wo. Woodend - closed.

TABLE V11.

	AGE PERIODS.								
	15	20	25	35	45	55	65	75	Mortality per 1000 living at various ages.
PHTHISIS WITH CHRONIC INTERSTITIAL PNEUMONIA AND RESPIRATORY DISEASES.									
Haematite Miners.	0.0	0.6	1.2	3.8	2.6	4.2	3.7	4.2	
All Iron Miners.	0.3	0.8	0.8	1.7	1.6	1.9	2.2	1.7	1910-1912.
Occup. + Ret. Males.	0.6	1.4	1.7	2.2	2.6	2.9	2.9	3.8	
Haematite Miners.	0.0	1.1	1.4	1.6	4.5	2.0	3.6	5.5	
All Iron Miners.	1.1	0.6	1.5	1.1	2.8	1.8	3.1	5.8	1921-1923.
Occup. + Ret. Males.	0.7	1.4	1.4	1.7	1.9	2.0	2.0	2.4	
PNEUMONIA.									
Haematite Miners.	0.4	0.3	0.4	1.1	3.0	3.8	1.9	4.2	
All Iron Miners.	0.2	0.4	0.4	0.7	1.6	3.1	1.9	3.4	1910-1912.
Occup. Males.	0.2	0.3	0.5	0.8	1.3	2.1	3.4	5.4	
Haematite Miners.	0.0	0.5	0.5	0.5	0.8	0.7	0.0	11.0	
All Iron Ore Miners	0.0	0.2	0.3	0.4	0.8	1.3	1.5	5.8	1921-1923.
Occup. Males.	0.2	0.3	0.4	0.7	1.0	1.7	2.8	4.7	
BRONCHITIS.									
Haematite Miners.	0.0	0.0	0.0	0.3	1.5	5.5	9.3	37.5	
All Iron Miners.	0.0	0.0	0.1	0.1	0.5	2.8	4.8	38.5	1910-1912.
Occup. Males.	0.0	0.0	0.1	0.2	0.7	2.5	7.8	23.9	
Haematite Miners.	0.0	0.0	0.0	0.0	0.0	3.3	12.6	47.6	
All Iron Miners.	0.0	0.0	0.0	0.1	0.3	1.6	8.5	36.4	1921-1923.
Occup. Males.	0.0	0.0	0.1	0.2	0.6	1.9	5.1	17.7	

TABLE V111.

	TOTAL	PULMONARY LESION INCLUDING PNEUMONIA.	AVERAGE AGE.	AVERAGE NUMBER OF MEN EMPLOYED.
1903-1907	236	93 (39.4%)	54.7	4652 (1905 - 09)
1908-1912	235	105 (44.7%)	55.	5142 (1910 - 14)
1913-1917	219	92 (42%)	56.6	
1918-1922	227	98 (43%)	54.2	2541 (1921 & 22)
1923-1927	226	97 (42%)	59.7	3266
1928-1932	207	78 (37%)	58.7	2659

TABLE IX.

Year.	Average Age.	Total.	Chest - plus.	Pneumonia.	Accident.	Average Age.	Total.	Chest - plus.	Pneumonia.	Accident.	Average Age.	Total.	Chest - plus.	Pneumonia.	Accident.
		WHITEHAVEN.					HARRINGTON.					EGREMONT.			
1903.	59.6	5	1	-	-	48.2	10	4	-	1	48.3	24	13	1	2
1904.	70.0	6	1	-	-	50.1	9	3	-	-	53.5	22	12	2	2
1905.	54.0	8	3	-	1	60.0	15	3	-	-	52.5	27	13	2	3
1906.	60.0	9	1	-	-	57.8	15	8	1	-	56.0	29	10	-	2
1907.	57.1	7	1	-	-	57.5	16	4	-	2	53.7	34	16	1	1
1908.	45.0	2	2	-	-	55.9	16	7	-	2	56.0	29	18	6	2
1909.	70.4	9	4	2	1	51.4	10	3	-	-	60.6	25	11	1	-
1910.	50.0	7	-	-	2	60.0	12	6	-	-	51.7	30	15	2	4
1911.	60.0	3	2	-	-	51.6	16	5	1	2	53.3	26	11	1	-
1912.	50.0	8	2	-	-	54.2	14	5	-	2	54.2	28	14	2	1
1913.	42.6	6	3	-	1	49.5	15	7	-	2	57.9	27	9	1	1
1914.	50.8	9	3	-	1	64.3	8	2	-	-	58.7	25	10	1	-
1915.	72.0	3	2	-	-	60.5	12	7	1	1	53.1	24	11	1	2
1916.	61.8	14	4	1	2	53.8	6	3	1	-	56.5	27	13	3	-
1917.	63.3	11	2	-	2	54.0	12	7	1	-	62.1	20	10	2	1
1918.	54.1	10	2	-	4	50.3	9	5	-	-	44.8	41	22	12	-
1919.	60.3	7	2	-	1	55.3	11	6	1	-	49.8	36	16	6	8
1920.	61.1	9	2	-	1	58.0	18	6	-	-	55.4	23	10	3	3
1921.	50.0	4	4	-	-	69.0	6	3	-	-	63.1	18	4	1	-
1922.	56.6	6	3	-	-	68.0	7	4	-	1	56.0	22	9	1	-
1923.	56.5	10	6	1	1	55.5	11	7	1	-	58.0	23	9	2	1
1924.	55.0	2	-	-	-	57.5	11	6	-	-	62.6	33	12	2	-
1925.	53.8	5	-	-	-	51.4	10	6	2	-	58.4	33	20	3	1
1926.	60.1	7	2	-	-	63.1	7	1	-	-	68.1	29	10	-	-
1927.	53.0	8	-	-	-	55.0	5	5	-	-	61.4	32	13	1	1
1928.	51.8	7	2	1	1	62.1	7	1	-	-	61.8	24	10	2	-
1929.	55.0	11	5	2	-	52.7	10	6	-	-	62.1	27	11	1	-
1930.	44.2	4	1	1	1	58.4	9	5	-	-	62.3	14	3	-	-
1931.	58.1	6	1	-	2	58.3	3	2	-	-	57.4	39	15	1	3
1932.	62.0	13	5	-	-	53.1	11	5	-	1	62.0	22	6	-	-
1933.						56.4	10	6	1	-	61.8	51	23	3	3
1934.	First	Three	Months	Only.							58.5	14	9	-	-

TABLE X. DEATH RATES. E. & W. 1900-1902. % living at each age.

	35 - 45		45 - 55		55 - 65		65 ——	
	A.	B.	A.	B.	A.	B.	A.	B.
All Males.	2.8	2.6	4.4	5.1	6.3	10.4	8.7	21.0
Coal Miners.	1.6	2.1	2.4	3.6	4.8	7.8	12.0	21.2
Iron "	1.9	1.9	2.4	2.8	5.6	5.2	8.7	16.3
Tin "	17.3	2.8	33.2	5.2	32.2	11.1	42.6	27.4

A = Lung disease.

B = Other causes.

TABLE Xl.

AGE ——		20.	25.	35.	45.	55.	C.M.F.
Respiratory Tuberculosis.	S.P.	20.6	37.6	43.6	39.0	22.7	163.
	SI.	25.6	63.4	141.0	211.6	150.6	592.
	N.S.	13.6	26.3	46.1	56.4	29.9	172.
Other Tuberculosis.	S.P.	2.8	3.7	3.0	2.6	1.8	13.9
	SI.	2.2	3.8	4.0	14.0	7.1	31.2
	N.S.	-	6.0	2.9	5.3	-	14.2
Bronchitis.	S.P.	0.5	1.7	5.4	13.3	28.8	49.6
	SI.	1.1	1.0	13.7	44.6	153.7	210.0
	N.S.	-	-	4.9	12.8	41.8	595.
Chronic Interstitial Pneumonia.	S.P.	0.0	.1	.2	.5	.6	1.4
	SI.	-	-	12.9	11.4	30.5	54.8
	N.S.	-	-	-	5.3	7.0	12.3
Pneumonia.	S.P.	4.3	11.5	19.6	24.2	25.7	85.3
	SI.	3.3	14.4	30.6	47.2	38.7	134.3
	N.S.	-	9.6	13.7	28.7	23.9	76.0
All Causes.	S.P.						1000.
	SI.						1984.
	N.S.						967.

C.M.F. = Comparative Mortality Figures.

S.P. = Standard Population.

SI. = Silicotic Occupation.

N.S. = Non Silicotic Occupation.

TABLE XII.

	% of Ash.	% of Silica Lung.	% of Silica to Ash.	% of Fe to Lung.	% of Fe to Ash.	% of AL. to Lung.	% of AL. Ash.
Needle grinder.	4.3	.666	15.18	.0178	4.0		
Knife grinder.	7.19	1.04	14.57	1.02	14.14		
Lead Miner.	1.91	.12	6.34	.67	3.51		
Housewife.	3.2	.031	1.02	Nil	Nil		
Simmons.					Fe_2O_3		
Miners'Phthisis	4.7	5.31	40.5	1.29	9.85	4.3	33.4
Hammarsten. (37)					Fe_2O_3		
Normal.	-	-	13.4		3.2		Nil.
Diseased.	-	-	40.2		9.85		33.8
McCrae. (30)					Fe_2O_3		
N.	4.9	.73	14.7		7.34		5.47
1.	4.5	1.39	30.7				-
2.	6.4	1.90	24.4		2.51		5.5
3.	5.4	2.23	40.8		9.83		7.7
4.	9.3	4.47	48.0		8.49		9.5
5.	6.4	2.98	45.8		3.78		7.9
6.	6.5	2.81	43.2		3.9		7.7

TABLE XIII.

CHEMICAL ANALYSIS OF LUNGS.

	% of Moisture.	% of Ash.	% of Silica to Lung Ash. as SiO_2		% of Iron to Lung Ash. as Fe_2		% of Aluminium to Lung Ash. as Al_2O_3		Weight of Lungs. Moist. Left.	Right.	PITS.
Lo. 5513.	84.7	16.97	1.113	6.63	7.11	42.0			-	-	B.
Har. 5515.	-	25.118	1.984	7.900	9.235	41.6	-	-	-	-	B.
Gr. 206.	-	3.24	.1543	4.78	.938	28.9	-	-	-	-	C.
H. 231.	82.84	16.96	1.0225	6.027	7.632	44.9	-	-	740	920	B.
Wr. 237.	81.42	25.4	1.84	7.24	12.006	47.30	.2	.8	500	670	B.
Dal. 240.	82.70	12.37	.753	5.90	4.62	36.79	.545	4.4	950	1050	B.
Tho. 255.	82.40	22.72	1.780	7.84	13.619	60.00	.212	.934	1050	960	B.
Fit. 340.	81.00	16.66	1.001	6.00	7.392	44.35	.2	1.20	-	1075	B. Ph. Bg.
Tid. 346.	80.00	20.28	1.890	9.32	10.84	51.70	.135	.664	1190	950	Wo. B. U.
Gi. 348.	82.00	18.22	2.126	11.67	8.00	43.90	.132	.725	-	-	B. Le. Lo. M.
Wal.*	78.00	12.21	1.97	16.18	5.55	45.50	.248	2.0	-	-	B.
Pos. 368.	75.00	16.67	1.114	6.66	6.72	40.00	.074	.44	1150	1030	F. & U.
Bir. 370.	82.59	16.36	1.782	10.89	6.89	42.1					B. Wo. MB.
E. M.	84.0	5.98	.396	6.62	2.68	44.85			-	-	H.
AVERAGE	81.5	16.39	1.3	8.2	7.3	46.	.022	1.4			

* Specimen given by Col. Harvey, R.C.P.Ed.

TABLE XlV. Summary of Cases.

Case.	Age.	Years in haematite mining.	Years on drills.	Degree of fibrosis.	Tubercle.	Chemical analysis (percentage of dry lung).		
						Ash.	Silica.	Fe.
1. J. A.	59	45	10 *	+ +	+	...	...	...
2. G. E. L.	41	14	12	+	+	16.97	1.11	7.11
3. W. B. H.	65	38	...	+ +	+	25.11	1.98	9.23
4. E. A. G.	34	10	5 (all dry)	nil	-	3.24	0.15	0.94
5. H. H.	62	22	...	+ +	+	16.96	1.02	7.63
6. H. W.	56	18	9 (all dry)	+ + +	-	25.40	1.84	12.01
7. R. D.	56	40	12	+ +	+	12.37	0.75	4.62
8. M. T.	45	23	10	+ +	+	22.72	1.78	13.62
9. R. F.	48	20	7 (5 dry)	+	+	16.66	1.00	7.39
10. J. T.	65	43	...	+ + +	+	20.28	1.89	10.84
11. J. G.	61	32	14 (all dry)	+ + + +	-	18.22	2.12	8.00
12. I. W.	49	16	Many	+ +	+	12.21	1.97	5.55
13. R. P.	49	9	9	+ + +	-	16.67	1.11	6.72
14. J. M. B.	60	46	12	+ +	+	16.36	1.78	6.89
15. E. M.	64	33	6/12ths.	nil	-	5.98	0.40	2.68

+ = a moderate grade of fibrosis, a definite sidero-silicosis.

++, +++, ++++ = increasing grades of severity, but all advanced.

* Largely in sto[illegible] work.

TABLE XV. ANALYSIS OF ORE FROM VARIOUS LOCALITIES.

	A.(20)	B.(20)	C.(13)	D.	B.D.H. ore.	E.(20)
Fe_2O_3	82.285	85.46	88.73	70.70	96.0	85.2
M.N.O.	.419	.055				1.35
AL.	3.062	3.017				1.35
Si.	10.525	7.400	4.96	8.9	1.23	7.23
Metalic Fe	57.60	59.82		49.52	67.2	

A. & B. (20) = Whitehaven Ore.

E. = Millom ore.

C. = North Cumberland ore.

D. = Egremont ore.
Own analysis.

TABLE XVI. SHOWING RELATION OF PHTHISIS MORTALITY TO PRESENCE OF SILICA IN DUST IN CERTAIN DUSTY INDUSTRIES. (18)

Trade.		Composition of Dust.	Phthisis Mortality.		Authority.
Year 1902	Age. 15 - 75		% of ALL Deaths.	% of Living.	
ALL MALES.		----------	13.1	2.1	Registrar General. Royal Com. on Met. Mines.
Iron Stone.		Fe: O: & Ca.	13.7	1.5	
Slate.		AL. Si.	15.4	1.8	
Pottery.		AL. Si.	18.9	3.1 X	X = ALL males engaged in pottery.
Lead Mines.		Pb. Si. Ca.	16.1	3.9	
Gold. Australia.		Au. Si.	23.5	12.7	Collis.
	S. Africa.	Au. Si.	42.1	-	
Metal Grinders.		Fe. Si.	49.7	15.2	
Mason.	Ca.	Ca. Co_3.	12.0	1.7	
	Sandstone.	Si.	44) 50. 17 56)	(16.7 (17.6	
Coal Mine.		C.	9.8	1.0	
Gannister.		Si.	88.9	22.3	
Flint Knapping.		Si.	77.8	41.0	

TABLE XVll.

Rabbit No.	Dates of Injections. 27.8.32.	25.9.32.	22.11.33.	1.3.34.	2.5.34.	29.5.34.	5.7.34.	Date Killed or Died.
1.	1 gm. Fe_2O_3	1 gm. Fe_2O_3	←				→	27. 6. 33.
2.	——	——	10 cc. Fe_2O_3	10 cc. Fe_2O_3	Tb. 10 cc. Fe_2O_3	←	→	7. 5. 34.
3.	——	——	7 cc. Ore.	7 cc. Ore.	——	←	→	2. 5. 34.
4.	——	——	——	10 cc. Ore.	Tb. 10 cc. Ore.	←	→	11. 5. 34.
5.	——	——	——	10 cc. Ore.	10 cc. Ore.	——	7 cc. Ore.	14. 8. 34.
6.	——	——	——	——	Tb. 7 cc. Ore.	←	→	3. 5. 34.
7.	——	——	——	——	Tb.	——	——	14. 8. 34.
8.	——	——	——	——	——	Tb. 7 cc. Ore.	Tb. 7 cc. Ore.	14. 8. 34.
9.	——	——	——	——	——	Tb. 7 cc. Ore.	——	3. 6. 34.
10.	——	——	——	——	——	Tb. 7 cc. Ore.	Tb. 10 cc. Ore.	14. 8. 34.
11.	——	——	——	——	——	Tb.	Tb. 7 cc. Ore.	14. 8. 34.
12.	——	7 cc. Fe_2O_3	10 cc. Fe_2O_3	10 cc. Fe_2O_3	10 cc. Fe_2O_3	——	7 cc. Fe_2O_3 ↔	5. 7. 34.