

U N I V E R S I T Y O F E D I N B U R G H .

PNEUMOCONIOSIS

IN

THE SOUTH WALES ANTHRACITE COALFIELD.

BEING A DISSERTATION

BY

H. R. STUBBINS

M.B.C.H.B.



1st October, 1933.

TANYWERN LANE,

YSTALYFERA.

C O N T E N T S.

	PAGE NO.
1. INTRODUCTION	1.
2. HISTORICAL OUTLINE	7.
3. THE VARIOUS TYPES OF PNEUMOCONIOSIS	23.
4. PREDISPOSING CAUSES	44.
5. ETIOLOGY AND PATHOLOGY OF PNEUMO- CONIOSIS	54.
6. PERSONAL AND CLINICAL WORK	69.
7. PROPHYLAXIS AND TREATMENT	207.
8. CONCLUSIONS	216.
9. BIBLIOGRAPHY	220.

M. D. T H E S I S

OF

H. R. STUBBINS

ON

PNEUMOCONIOSIS IN THE ANTHRACITE COALFIELD.

-----o00o-----

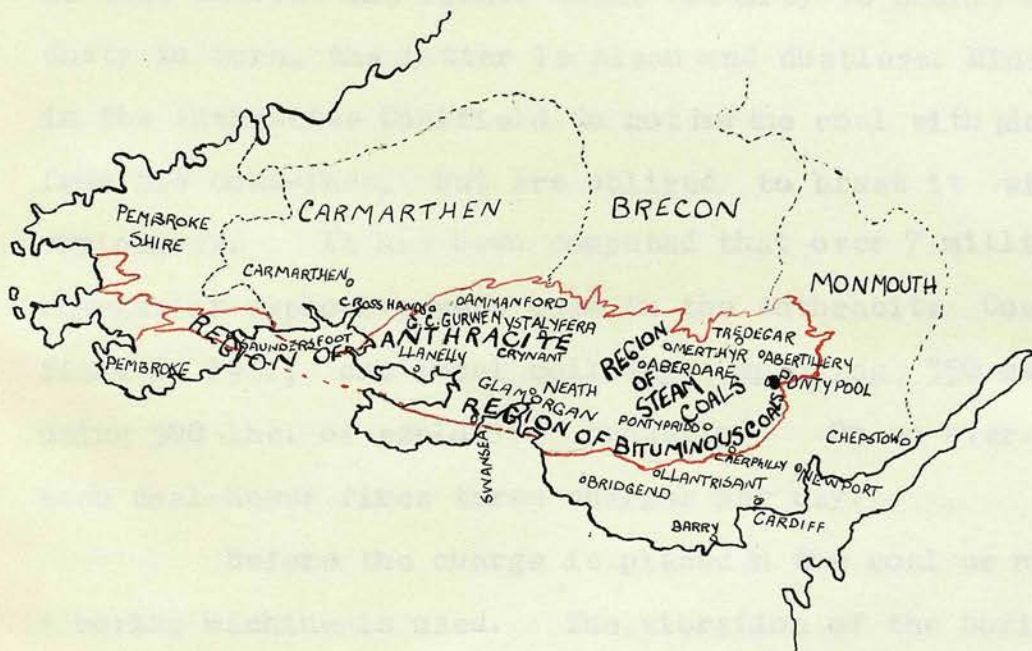
INTRODUCTION.

GENERALLY.

South Wales, comprising the counties of Glamorgan, Monmouth, Brecon, Carmarthen and Pembroke, depends chiefly upon the coal mining industry. Two thirds of its population are employed directly or indirectly with this industry, and its importance, therefore, to South Wales is considerable.

There are three definite types of coal mined in this area, namely, steam, bituminous and anthracite. Generally speaking, steam coal is mined in the eastern part of the county of Glamorgan, bituminous in the valleys of Monmouthshire, and anthracite in what is termed the Western area of South Wales, namely, the counties of Pembroke, Carmarthen, Brecon, and the Western part of the County of Glamorgan.

MAP ILLUSTRATING THE DISTRIBUTION OF THE PRINCIPAL KINDS OF COAL
IN SOUTH WALES.



Although coal is mined in many parts of Great Britain, forming one of its staple industries, anthracite coal is found only in West Wales where over 50,000 men are daily engaged in its production.

Anthracite coal differs in many respects from steam and bituminous coal, whereas both steam and bituminous coals are soft, brittle, and dull in colour, anthracite coal is hard and lustrous; a further difference is that whereas the former coals are dirty to handle and dusty to burn, the latter is clean and dustless. Miners in the Anthracite Coalfield do not hew the coal with picks from the coal-face, but are obliged to blast it with explosives. It has been computed that over 7 million charges of explosive were used in the Anthracite Coalfield in 1931; one local colliery, employing 350 men, using 500 lbs. of explosive annually. On an average each coal-hewer fires three charges per day.

Before the charge is placed in the coal or rock a boring machine is used. The vibration of the boring machine, coupled with the percussion of the explosives, releases the dust from the sides, roof and floor for a distance of about fifty yards from the site of the explosion.

In addition to these the collier has other duties to perform, such as repairing the sides of his working-place, and what is called 'ripping' the roof, which signifies the trimming and cutting away of all shale and loose coal so as to make the roof safe. This

operation envelops him in clouds of dust so that when these dust-creating agencies are added to the dust which he ordinarily inhales whilst hewing the coal, it cannot be denied that the collier works under most difficult conditions and that much gritty matter is inhaled into his lungs from day to day.

Moreover, the miner works in what is described in the mining valleys as 'hard headings'. This means boring with specially constructed compressed air machine drills, through solid rock, the thickness of which varies from fifty to two hundred yards. Hard headings are worked on a contract basis, which involves continuous and unremitting exertion during their working shift. The boring machine sets up clouds of dust, which, in this case, is of a stony nature, and this is inhaled steadily by the miner while the boring continues.

Horses used for haulage purposes underground are well cared for. As they work dust arises from the floor and this permeates the atmosphere along the route. This dust settles on the coats of the horses in a fine white layer which is brushed off later in the stable by the hostler, during the process of grooming, and is inhaled by him. It should be stated that the floor contains fire clay of a high silica content.

LOCAL CONDITIONS.

The valleys in the Anthracite area are very narrow, and the benefits of sunlight are sporadic. A

local Medical Officer of Health has stated in one of his Annual Reports that many hours of sunshine are lost every year owing to the geographical features of the district.

The climate is wet, the rainfall for last year being among the highest in the country.

From an agricultural standpoint the soil is poor, but from a mining point of view it is rich.

The few small farms in the district are scattered about the mountain-side, the fields being used for the grazing of cattle and sheep. Many of the farmers work at the local collieries, their wives doing such farm work as is required.

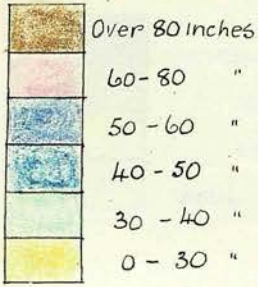
It is thus apparent that all the inhabitants of these colliery districts depend for their livelihood upon the work at the collieries.

During recent years, housing conditions have improved. The local District Council have built nearly 800 houses, with adequate accommodation and facilities for the miner and his family. The basement dwellings so common years ago are gradually being closed, and their occupiers removed to houses built under the Housing Act of 1930. There is an abundant supply of water in the area which is obtained from large reservoirs and local springs.

The present writer was brought up in the steam coal area of South Wales, but during the last eight years he has been in practice in the anthracite coalfield. One of the first things to which his attention was drawn

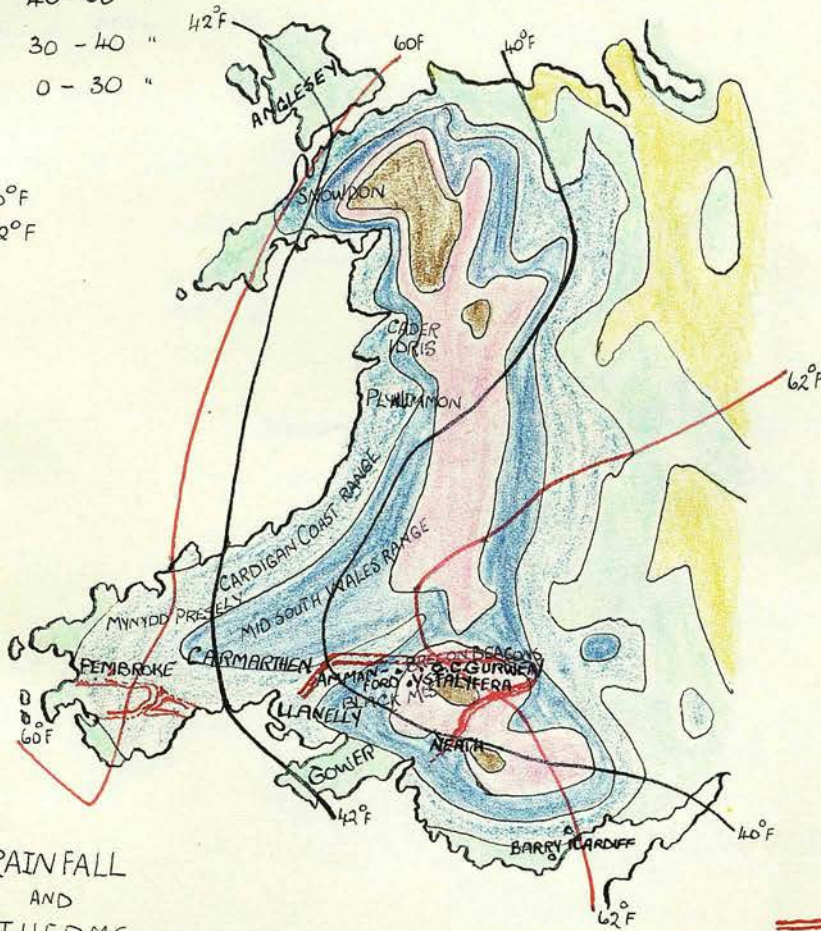
MAP ILLUSTRATING RAINFALL IN ANTHRACITE AREA

Mean Annual
Rainfall



Isotherms

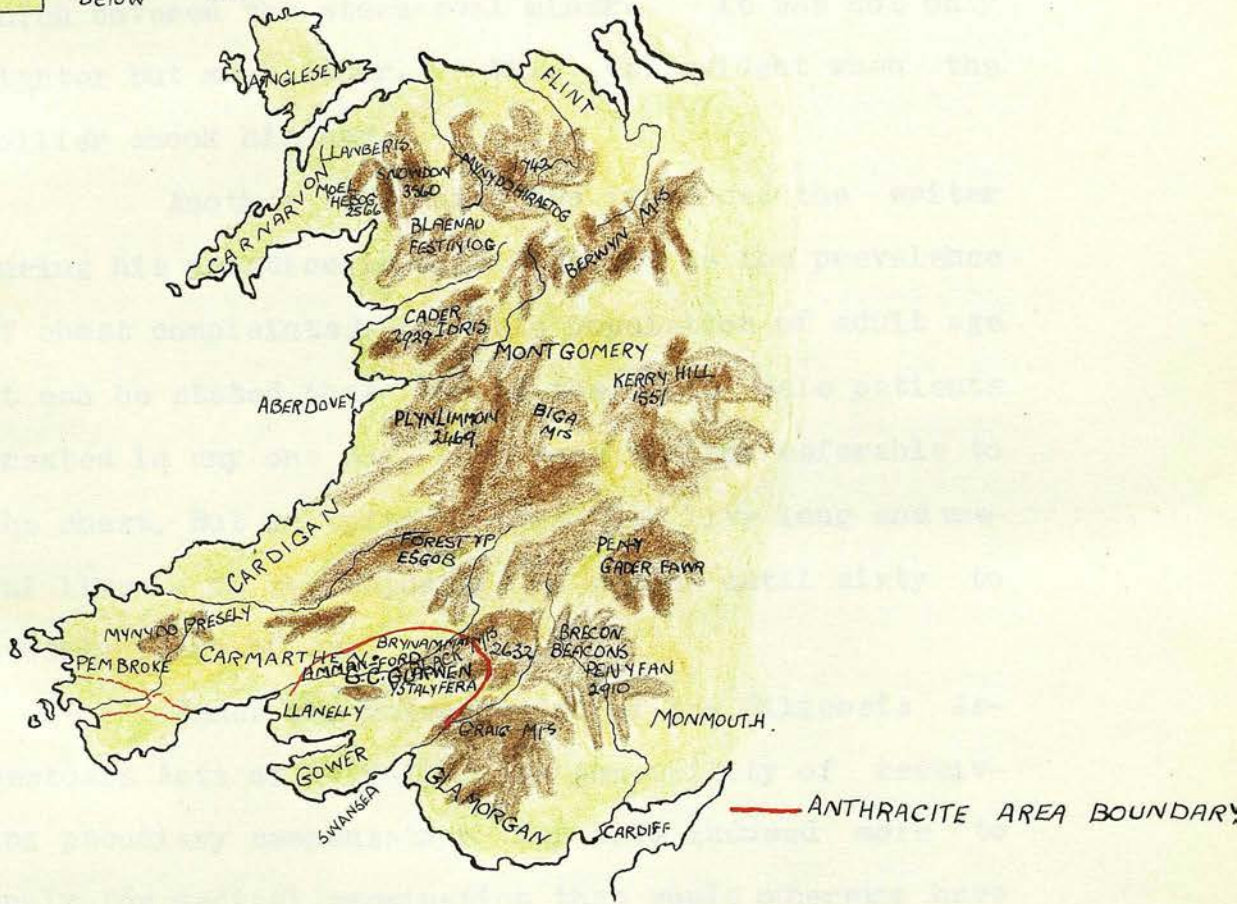
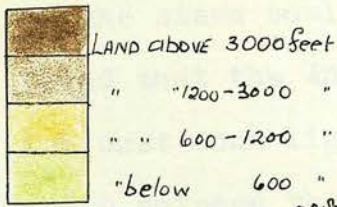
January — 40°F
July — 62°F



RAIN FALL
AND
ISOTHERMS

— BOUNDARY OF ANTHRACITE-COALFIELD.

MAP ILLUSTRATING MOUNTAINS AND VALLEYS OF WALES.



was the fact that miners in the anthracite area looked much cleaner coming home from work than their colleagues in the steam coal area. On examination, however, he found that the Anthracite miner was covered with a greyish dust much lighter in colour than the black dust which covered the steam-coal miner. It was not only lighter but much finer. This was evident when the collier shook his cap.

Another fact which has impressed the writer during his practice in this district is the prevalence of chest complaints in the male population of adult age. It can be stated that 40% of the adult male patients treated in any one day have some symptom referable to the chest. But in spite of this they live long and useful lives - in the majority of cases until sixty to seventy years of age.

Since the introduction of the Silicosis Asbestosis Acts of 1929-1931 the possibility of receiving pecuniary compensation may have induced more to apply for medical examination than would otherwise have been the case.

In the immediate neighbourhood there are at least twenty certified Silicosis cases who are permanently disabled, and many others have been certified as partially disabled.

The permanently disabled miners are all between the ages of forty and sixty years, and although the writer has been unable to find tubercle bacilli in

their sputa he is convinced that a very high percentage of them are tubercular.

An attempt will be made in this dissertation to describe pneumoconiosis in the Anthracite coalfield as seen and studied personally from actual cases as well as from clinical records which have been kept of cases now deceased; also to record any personal investigations which the writer has made to elucidate the pathogenesis of the disease.

That a great many of the miners suffer from pulmonary tuberculosis is an important factor, and the question whether it is a primary infection or is superimposed upon a pulmonary fibrosis caused by the traumatism of stone dust inhalation will be discussed. On the other hand, whether the presence of coal dust in the lung has a prophylactic effect against the tubercle bacillus is also an important controversial point. This will be examined in the light of observations made by the writer, from clinical examinations, and from the many post mortem examinations he has performed, or at which he has been present.

It is also intended to deal with the correlation of pathological signs with the ability to work, a factor of importance to the general medical practitioner to whom the patient goes for his initial examination. Since the introduction of the Silicosis-Asbestosis Act, the ability to work and the time when the man becomes unfit for work - and so is prevented from going down the mine - becomes of paramount importance.

HISTORICAL OUTLINE.

Pneumoconiosis actually means the pigmentation of the lung due to the inhalation of foreign particles.

The term was first introduced by Zenker⁽¹⁾ who included all those forms of fibrosis of the lungs which were produced by the inhalation of various types of dust. According to the type of dust inhaled so were the pneumoconioses differentiated. If the fibrosis occurred amongst coal-miners due to the inhalation of coal dust the condition was called anthracosis - from the black colour of the lungs; if due to the inhalation of stone dust as found in the lungs of stone masons, silicosis was the term given, and so with regard to iron and steel-grinders' lungs it was called siderosis.

During the last few years, investigators have begun to use the terms silicosis, silico-anthracosis, and silicosis with tuberculosis to describe the condition of the lungs in coal miners. All these terms tend towards confusion, but, not content with the above variety, other names have been employed which at once give the impression that writers are not all agreed on the subject as to the pathology and clinical aspects of the disease. Pulmonary sclerosis, pulmonary cirrhosis, fibroid lung, fibroid pneumonia, interstitial pneumonia, are some of the many terms employed. But whatever term is most popular it is easy to see that all agree that there seems to be a great hyperplasia of the fibrous tissue of the lung; so much so

that observers have gone so far as to state that the lungs are completely replaced by fibrous tissue. Whether this is due to the irritation of the dust inhaled is a matter which will be dealt with later.

Another term which this dissertation hopes to discuss in the pneumoconioses is that of Miners' Phthisis. The latter deals with the fibrosis of the lung as found among coal miners who in the first place have developed a condition of fibroid lung and later been infected with the tubercle bacillus. On the other hand, it may be that the tubercle bacillus was already present and only needed the constant irritation of the inhaled dust to light up a dormant tubercle.

The first person to draw attention to the frequent occurrence of phthisis among miners and stone-masons was Professor Ramazzini,⁽²⁾ Professor of Medicine in Modena. In his treatise on "Trade Diseases" published in 1703, he points out that "those who dig minerals are but short lived", and that during his time mining was looked upon as a very degrading occupation. Indeed it was as a sort of punishment that most of the mines were worked. He goes even further and states that the mining conditions were so deplorable and the physical condition of the miners so bad that he doubted whether it was good practice to prescribe for such poor wretches and so prolong a miserable life!

He also points out that these workers, in consequence of the inhalation of sharp and rough-cornered particles which flew off during the cutting and polish-

ing of stone and marble, developed a troublesome cough and later became asthmatic and consumptive.

When the lungs were examined post mortem Professor Ramazzini found areas of hard gritty particles studded with little heaps of sand, so that on running the knife through them it gave the operator the sensation of cutting through sand.

After Ramazzini it was not until 1831, when
(3) Thackrah in his "Effects of Principal Arts and Trades and Professions" took us further along the road of investigation. Colliers as a class, he said, were longer lived than they used to be, although even now they seldom lived longer than fifty years. In his opinion they led a loose and intemperate life. But the important fact which he brought forward was that pulmonary diseases chiefly prevailed in coal mines where the coal was loaded with ^{FeS₂} pyrites. The symptoms were, malaise co-existent with constriction of the chest, impeded respiration, nausea, vomiting and head noises. During this time the conditions of working in the coal mines were indeed very bad. Ventilation was poor; there was only one shaft leading down the mine and miners attributed their attacks of bronchitis to the reek of gunpowder and smoke from the lamps.

Thackrah was also the first to point out that the stone-masons and knife-grinders of Sheffield became afflicted with signs and symptoms similar to those found among colliers. Knife-grinders using dry sandstone lived

only to an average age of twenty-eight years, while knife-grinders using wet sandstone lived to between forty and fifty years. Their symptoms were all respiratory, due to the inhalation of particles of stone dust, and consisted of difficulty of breathing, hoarseness, cough, spitting of mucus containing dust, later, mucus containing foetid and purulent matter and denoting, in his words ' a slow but fatal consumption'.

Miners in the North of England when working ore in sandstone are especially liable to chest complaints, but when working ore in limestone they do not suffer. This is due to the fact that limestone is fissured vertically and water from the supernatant beds percolates through the roof of the mine, keeping the ore moist and so allaying the dust. Sandstone strata on the other hand are impervious to water and so the ore is quite dry. From the foregoing, Thackrah thus adds another milestone, namely, the important point that dust which is provocative to lung disease is dry dust.

Seven years' later, in 1838, a British worker, named Stratton ⁽⁴⁾ brings in the term Anthracosis for the first time. He called the condition of the lungs of miners as found by him on post mortem examination Anthracosis, and divided his post mortem lungs into three classes :-

1. Healthy lungs.
2. Melanotic lungs.
3. Anthracotic lungs of coal miners.

He must have thought that the melanotic type

was of neo-plastic origin accompanied by secondary growths elsewhere and associated with the wasting so commonly found in coal miners.

(5)
But even in 1813, Pearson had found that the pigment in the lungs was exogenous showing it to be charcoal in the uncombined state, and he also demonstrated that this charcoal was carried along the lymphatic channels of the lungs.

(6)
Dr. William Thompson in 1837 gives us a classical description of the black lung of the coal miner :-

"On opening the chest the lungs on both sides were found to be adhering firmly to the parietes. The whole substance of the lungs was of deep black colour. The upper portions were of solid texture. The lower cellular structure was somewhat preserved. In cutting two days' afterwards through the portion of the lung which had laid below the external abscess, three or four cavities could be seen which were filled with an inky black fluid."

Thompson insisted on the extraneous origin of the black matter by which the sputum was liable to be discoloured and the lungs infiltrated.

(7)
Greenhow in 1869 again reported a description of black lungs from a case of colliers' phthisis. The greater part of the lung was solidified as a firm mass, and looked and felt like a piece of india-rubber. This presumably was a case of silicosis, and the chemical analysis of the dried ash proved the presence of 30% of silica. Greenhow, then, was the first investigator

to demonstrate the co-existence of silicosis with anthracosis in the lungs of a coal miner dying from pneumoconiosis.

It must not be assumed that all these important facts were brought to light without any antagonistic theory being put forward; for Virchow ⁽⁸⁾ in 1858 did not embrace nor subscribe to this theory. He did not believe that the dark pigment found in the lungs was of exogenous origin. He brought forward his own theory, namely, that the pigmented changes were due to the extravasation of blood and the subsequent transportation of haematin and not to the absorption and deposit of carbonaceous matter inhaled into the lungs.

The subject of coal miners' phthisis had by this time become of such importance that Sir John Simon drew attention to the relative freedom of the Northumberland miners from pulmonary disease as compared with the workers in the South Wales coalfield, which he attributed to the better ventilation of the mines in the North of England.

⁽⁹⁾ Oliver, writing in 1902, described the miners of thirty years' ago, and how common miners' phthisis was in those days. He maintained, however, that conditions had improved so much that, apart from explosions, mining was a healthy occupation, and that coal miners as a class suffer less from phthisis than men in other occupations. Some physicians go as far even as to say that coal dust renders miners immune from

(phthisis?)

Oliver goes on to say that it is more than probable the disease is not a pure pneumoconiosis, but a mixed tuberculous malady, the infection having been caught in the home or elsewhere. In comparing the working conditions in coal mines with the Cornish tin mines, although both have improved, the death rate in the latter is eight to ten times as great as in the former. Oliver attributes this to the changed method of working and especially to the using of machine drills in boring the rock.

(10)
Tatham wrote similarly in 1902 - "Colliers as a class compare favourably with men in most other occupations on the score of health".

The next great step forward came in 1914 with the report of the Royal Commission on "Metalliferous Mines and Quarries".

Prior to 1914 the old story was regarded with complacent satisfaction until the observations of the investigators in South Wales drew attention to the presence of silicosis as well as anthracosis as an essential element in the pneumoconiosis of coal mines. Silicosis is a disease with a well-known pathogenic importance to the liability of pulmonary tuberculosis. The Royal Commission reported "That if in any given class a high rate from pulmonary tuberculosis is found occurring at a later period of life than is usual for pulmonary tuberculosis and if this high death rate is associated with a high death rate from respiratory diseases, then this class is exposed to the inhalation of injurious dust."

sive signs of disease. Shortness of breath, aggravated by the slightest physical exertion, was the principal complaint. This was out of proportion to the extent of the pulmonary disease present. Pain in the chest was fairly common but cough with expectoration and haemoptosis was an uncommon symptom. The Rand miners sought medical advice for the complaint of shortness of breath. This team of investigators proved that gold miners could only follow their work for five to ten years and also that since the introduction of machine drills for rock drilling the mortality had greatly increased. Unless constantly sprayed, rock drilling gave rise to large quantities of dust composed of sharp particles. The average length of working life of rock-borers was found to be 6.49 years. All this work on silicosis in the Rand, while it focussed attention upon the dangers of exposure to dust from hard rock, tended on the other hand to produce a sense of false security in regard to the coal miner.

Coal dust, as a result of numbers of animal experiments not only was exempt from blame, but also came to be regarded as a beneficial agent capable of neutralising the effects of silica. Indeed, Mavrogordato in 1918⁽¹⁷⁾ stated that all forms of dust enter the lungs and that some, such as coal dust, are relatively harmless, while others, such as flint, are deadly.

⁽¹⁸⁾ Professor Haldane, in an address to the South Wales Institute of Engineers in 1924, came to the con-

clusion that coal dust inhalation has a definite tendency to prevent phthisis and quoted from statistics that this diminished relative liability had existed for generations; adding that in the available statistics 1849-1859 it was no less marked than now. But it is well known that statistics are not always as true as they claim to be. Again, what may be true of one coal mine may not be true of another. Dust found in the anthracite collieries is very different from the dust found in steam coal collieries.

Animal experiments may be quite logical and excellent for comparing the effects of one dust with another, but the simultaneous exposure to many types of dust is quite a different matter.

The next advance came with the introduction of the X-Rays as an aid to diagnosis, and it was when dealing with men who had worked with drills in hard headings that Tattersall⁽¹⁹⁾ in 1926 drew attention to this important point: "Hard heading workers are exposed to a very high percentage of silica in the dust which they work in".⁽²⁰⁾ Jousset in 1928 stated that silica is found to be ten to twenty times greater than normal in the ash from the black lungs of colliers.

⁽²¹⁾ Cummins is of the opinion that coal miners who develop silicosis develop anthracosis as well. Silica, by causing interference with the lymph drainage, leads to retention and accumulation of coal dust in the lungs.

(22)

Cummins and Sladden in 1930 in "The Coal Miner's Lung" state that :-

- (1) Silica is found in anthracotic lungs in abnormal amounts.
- (2) The effects of the silica are to damage and block the lymph channels and so impair the powers of the lungs to dispose of the inhaled dust.
- (3) Whenever coal dust is inhaled in large quantities there is found high silica content in the lungs.
- (4) Anthracosis of the lungs is determined by a combination of silicotic fibrosis and accumulation of coal dust.
- (5) A considerable degree of anthracosis is consistent with working health.
- (6) Coal miners especially exposed to silica dust are liable to develop silicosis which, complicated by the effects of anthracotic dust accumulation, may lead to disablement and death.

(23) In 1930, the Welsh National Memorial Association, working with three selected teams of the medical staff, studied various men over forty years of age employed in mining (1) steam coal (2) bituminous coal and (3) Anthracite coal.

They came to the following conclusions :-

- (a) That prolonged exposure to the working condi-

tions incident to coal mining is frequently associated with the gradual development of alterations in the radiological appearances in the lungs.

- (b) These alterations are manifest as a fine or coarse mottling through the lung fields, the type being comparable with that observed in X-Ray films of the lungs of persons exposed to silica dust and to certain other dusts capable of leading to pneumoconiosis.
- (c) No significant degree of mottling is to be seen in X-Ray control films from agricultural workers of comparable ages, and it is therefore legitimate to conclude that the mottling observed in X-Ray films from coal mines is due to conditions encountered in the industry, especially to the inhalation of the dusts suspended in the air of coal mines.
- (d) The amount of lung alteration above described increases with the duration of employment, but when present to a marked extent as seen in X-Ray Films may be quite consistent with working health.
- (e) The alterations, however, represent a definite impairment of the lung structure and involve an increasing amount of interference with the respiratory efficiency of the lungs as manifested in a diminished vital capac-

ity and such general symptoms as a gradual loss of weight and shortness of breath.

- (f) How far these changes are operative in the production of respiratory diseases to which South Wales coal miners are liable in late middle age and old age is not yet quite clear, but the fact that the disease attains the maximum incidence in the Anthracite coalfield where the X-Ray mottling is found to a greater extent than elsewhere suggests a relationship between the lung fibrosis and the respiratory conditions common in old coal miners.

But what of tuberculosis? Why should not the silicosis which is so often the precursor of phthisis in the gold mines have the same sinister association with the collier? Some writers attribute it to the anti-septic action of coal dust on the lung tissue. Wainwright and Nicholls⁽²⁴⁾ in 1905, although admitting the protective action, were unable to demonstrate any anti-septic action. They failed to show that the presence of coal dust inhibited the growth of tubercle bacilli in their experiments. All this proved that the protective action was not due to any anti-septic action of coal dust.

(25)
Cummins now brings forward the theory that the protective influence is due to the adsorptive action of coal dust. Tubercle bacilli act upon the tissues

(25)

through their toxic products. Dr. Weatherall has proved that coal dust can adsorb and inactivate tuberculin solutions to a marked extent, and as Cummins and Sladden (26) have shown that coal miners' lungs may have as much as 100 grains of coal dust this adsorptive action must be very great.

The Legislature has now recognised that coal miners are affected by the inhalation of dust whilst following their occupations and in 1928 it was scheduled as an industrial disease. But in this Act the miner had to prove a very high percentage of silica in his working place. In the Silicosis Asbestosis Act of 1931 new regulations were issued which abolished this necessity.

-----0000-----

1. ... Diseases of Occupation ... 1902.

2. ... Bulletin ... of Medicine, Vol. 5.

3. ... Vols. II and III. N.S.S.S.

4. ... Hilroy Lectures 1915 and 1919.

5. ... Diseases of the Miners ... Pretoria, 1916.

6. ... Pub. South Africa Inst. for ... 1916, No. 711.

7. ... Rep. South Africa Inst. for ... 1917.

B I B L I O G R A P H Y.

1. OSLER and MACRAE Principles and Practice of
Medicine, p.636, 1921.
2. RAMAZZINI Diseases of Tradesmen 1703.
3. THACKRAH Effects of Principal Arts
and Trades and Professions
1831.
4. STRATTON, T.M.L. Edin. Med. and Surg. Journal
1838, xlix. 490.
5. PEARSON, GEORGE Phil. Trans. Royal Society
1813 ciii 159.
6. THOMPSON, WILLIAM Med. Chirurgical Trans. Lon-
don 1837, XX230.
7. GREENHOW, E.H. Trans. Path. Soc. 1868 XX41
8. VIRCHOW, R. Edin. Med. Journ. 1858-1859
iv. 204.
9. OLIVER Diseases of Occupation ...
1902.
10. TATHAM Albutt and Rolleston Sys-
tem of Medicine, Vol. 5.
11. ROYAL COMMISSION ON ..
METALLIFEROUS MINES ..
AND QUARRIES, 1914 ... Vols. II and III. H.M.S.O.
12. COLLIS Milroy Lectures 1915 H.M.S.O.
1919.
13. WATT, A.H., IRVINE, L.G.,
PRATT, JOHNSON, J., and
STEWART. Silicosis on the Witwaters-
rand, Pretoria, 1916.
14. WATKINS-PITCHFORD, W.,
and MOIR, JAMES Pub. South African Inst. for
Med. Research, Johannesburg
1916, No. VII.
15. MACRAE, J. Rep. South African Inst. for
Med. Research, Johannesburg
1913.

16. MAVROGORDATO, A., Pub. South African Inst. for
Med. Research, Johannesburg
1922, No. XV.
- Pub. South African Inst. for
Med. Research, Johannesburg
1926, No. XI9.
17. Effects of Dust inhalation
Journ. of Hygiene 1918, Vol.
p. 439.
18. HALDANE, J. S., Effects of Dust inhalation
in miners - Address to ...
South Wales Inst. of Mining
Engineers, 1923.
19. TATTERSALL, N., Journ. of Indust. Hygiene,
1926, viii 466.
20. JOUSSET, A., Rev de la Tub 1928 1X 884.
21. CUMMINS, S. LYLE, Journ. of State Medicine ..
Vol. XXXIX No. 9.
22. CUMMINS, S. LYLE, and . Journ. of Path. and Bact. ..
SLADDEN, A. F., Vol. XXXIII 1930.
23. WELSH NATIONAL MEMORIAL Radiological Study of cer-
ASSOCIATION tain groups of healthy ...
coal miners, Annual Report
1930.
24. WAINWRIGHT and NICHOLS American T. of Med. Sci. .
CXXX 405.
25. CUMMINS, S. LYLE, Journal of State Medicine.
Vol. XXXIX No. 9.
26. CUMMINS, S. LYLE, and . Journ. of Path. and Bact. .
SLADDEN, A. F., Vol. XXXIII 1930.
-

THE VARIOUS TYPES OF PNEUMOCONIOSES.

Silica is a substance which occurs in nature in the form of quartz, quartzite, and flint. It forms sandstone and gritstone rocks and usually the mother rock through which the ores of precious metals run. Grindstones are made of silica and so are millstones. Silica is used in the manufacture of silica paints, and as powdered flint it is largely used in the manufacture of earthenware.

A person who works in a dusty atmosphere is always liable to disease of the lungs. This disease terminates in a condition which is called fibroid phthisis, and according to the occupation of the person, is variously called miners' phthisis, steel-grinders' phthisis, potters' phthisis and stone-masons' phthisis.

The prevalent idea has been that the inhalation of any kind of dust was associated with an undue tendency to tuberculosis. The reason given was that the dust acted as a mechanical irritant to the lung tissues. But when the Royal Commission⁽¹⁾ published its findings in 1914 it showed that all these diseases were, in substance, but varieties of the same disease; that is, a specific form of dust phthisis associated with one kind of dust only - that composed of fine particles of silica.

Although the prolonged inhalation of silica

dust causes death from interstitial pneumonia there is another very serious condition which proves fatal at a much earlier date, namely, the tendency to secondary tubercular infection, to which persons who suffer from silicosis seem extremely liable.

Before proceeding further it would be well to mention that Calmette of Lille ⁽¹²⁾ did not agree that this condition arose from the inhalation of this dust. He was of the opinion that the workmen swallowed it during their occupations, and the fine particles penetrating the lining membrane of the alimentary canal, found their way into the lymphatics, and ^{were} thus carried to the abdominal glands and so to the lungs. This theory seems to conflict with all clinical experience. Dust can be found either by the naked eye or microscopically in the sputum of a man who has been working in a dusty atmosphere for only a few hours. In addition to the particles of dust embedded in the mucous, smaller particles are seen in the interior of the mucous corpuscles and epithelial cells.

Recent records support the view that the risk is clearly associated with the exposure to the dust. It has been statistically demonstrated from the returns of the Registrar General that men working in open quarries have less dust risk than masons who dress stones under half-open sheds, and that metal-grinders who work in closed factories are even more exposed to the risk, while miners who work in underground galleries are naturally

again more liable to the risk.

Another important factor which has to be established is the size of the dust which is known to cause the disease. Investigators, such as Collis⁽³⁾ and Macrae,⁽⁴⁾ have come to the conclusion that the dust must be very fine. The particles of dust are generally not greater than five microns in size. Such particles are like smoke or vapour. They cannot be washed down by spray or water, for the particles recoil from drops of moisture; nor, owing to their very fine size, can they really be stated to have an abrasive action, for they move gently within the lungs. Lastly, since other dusts are not followed by the same pathological changes characteristic of silicosis, it must be inferred that silica dust must exert a chemical rather than a physical action upon the lung tissues.

On the other hand the fate of the larger particles that must be inhaled during the man's occupation must not be lost sight of. These are carried by the air stream into the larger air passages where they become entangled in the mucous and are swept out by ciliary action. If this process is increased beyond physiological limits, the elasticity of the mucous membrane suffers, and degeneration occurs with the resulting Bronchitis. In this respect silica dust is no different from the larger particles of other inorganic dusts. Only the fine particles such as are able to pass the fine bronchioles gain access to the alveoli and probab-

ly they only do so during deep respiration associated with coughing and exertion.

When the fine particles of silica dust are taken into the alveoli a new reaction appears. The particles are taken up by dust cells which originate from the lining cells of the alveoli. The dust is absorbed by the cells and may linger in the alveoli for many weeks, thus impairing the function of the alveolus. If the particle be made of silica it slowly disintegrates and is converted into a colloidal hydrate, and in this form it reacts on the surrounding cell tissue. This reaction leads to the death of the cell, disintegration occurs, leaving the silica content free.

Such dust-burdened cells may escape into the bronchioles and are finally ejected in the sputum or they may traverse the walls of the alveoli into the lymph stream. In this stream there is a drift towards the surface of the pleura but the cells tend to accumulate at lymph nodes, where the freed silica particles carry on their reaction with the surrounding lung tissue. Thus as a result of this reaction fibrous tissue is formed. It is thought that an infection, usually the tubercle bacillus, is present at the commencement of such fibrous change but no proof of this has so far been demonstrated. Yet it is well known that lungs can become impregnated with silica without exhibiting silicotic changes and that the mere supervention of an infecting agent suddenly precipitates these silicotic changes.

An attempt will now be made to describe how workmen during their employment are exposed to this injurious dust.

MINERS' PHTHISIS.

Before studying this type of pneumoconiosis it must be realised that coal miners are exposed to two kinds of dust, namely, the dust from the coal itself and the dust from the stone above and below the coal seam. This stone consists of shale, 'bind' and fireclay, which easily becomes disintegrated into dust in coal mining operations. Indeed this is so marked that the dust actually suspended in the air and working places of a coal mine consists largely of stone dust. This is proved by the analysis of the dust which settles on the timbers etc. The dust itself consists chiefly of silicates and free silica or quartz. In the Anthracite coalfield stone dusting of the floor of the mine is not carried out owing to the very small quantity, if any, of fire-damp present, and also owing to the large quantity of dust which is already to be found on the roads. Nearly all the dust on the timbers is fine, as 80% of it passes through a 90 inch sieve, and on analysis contained 43% of stone dust. If this dust were taken at the working face the percentage of stone dust would be much higher.

Taking the underground worker at his employment, it used to be thought that the only employee who could contract disease of the chest was the man who worked

on 'hard headings', and bored through solid rock by means of machine drills driven by compressed air. He is supplied by his employer with a face mask, usually of the Siebe-Gorman type. The dust which the machine drill creates is very considerable, and the rapidity with which the man works soon causes the mask to become blocked. It has been noticed that very few of the men keep the mask on constantly but raise it to allow of free breathing. Many other types of face masks are used and numerous makes of machine drills are employed, but no ideal mask has yet been found which will permit the miner to work with any degree of comfort, and to work as hard as he really has to do in order to carry out his contract.

Another type of coal miner who is very prone to disease of the lungs is the Repairer. It is his work to repair the roof, walls and floor and see that all roadways are clear so that the coal hewer can follow on with his particular work. In doing this the repairer must of necessity use a machine drill and also create a great deal of dust in the process.

Coming to the coal-hewer himself, he also uses a boring machine, but usually it is a hand-drill. He uses it to bore holes in the coal in order to place in the charge of explosive to blast the coal. In the anthracite coalfield miners cannot cut the coal from the seam with their pick-axes, but must use explosives owing to the hardness of the coal. The explosion, as prev-

iously explained, shakes the whole gallery for a distance of at least fifty yards and clouds of dust are set up with each explosion. This dust is, of course, a mixture of coal and stone. Such shot-firing is carried out three or four times a day in each stall.

Along each roadway the dust, which is of a whitish or rather greyish colour, is usually three or four inches deep. Hauliers who are employed in leading their horse-drawn trams along these roads are exposed to this dust and are thus affected with this pulmonary condition.

From this type of workman, the man who grooms the horses each day after they have worked nine or ten or even twelve hours each day in this dust, must not be forgotten. The animals come to the stable after their day's work covered with a white coating of dust, and it is the hostler's duty to brush this off. The man usually places a handkerchief over his mouth whilst performing this work, but soon takes it off owing to the discomfort. Thick white clouds arise, much of which must of necessity be inhaled by the person who performs the grooming.

In steam and bituminous collieries the roadways are artificially dusted with fine stone dust following Sir William Garforth's discovery that such a procedure prevented dust explosions. In anthracite collieries, however, this artificial dusting is not necessary, because there is already plenty of stone dust along

the roadways. It is thus easy to understand how these other employees of the coal mine can become affected with this pulmonary disease.

From the foregoing it is seen that all underground workers inhale during their occupation a mixture of coal and stone dust. Which type of dust is the cause of all the different changes produced in the lungs, or do both contribute to the production of the condition?

Another question arises - is it possible for a miner to inhale only coal dust and not the stone dust?

It has already been stated that most investigators claim that coal dust, far from being detrimental to the health of the miner in so far as his liability to the infection with the tubercle bacillus is concerned, is in fact a blessing in disguise. All statistics go to prove this, as miners as a class live longer lives than many other types of workmen. Again, workers who inhale only stone dust such as potters, slate-quarrymen, gold miners, steel-grinders, etc., have a very high mortality, and have indeed a definite and marked tendency to infection by the tubercle bacillus.

The general opinion held to-day is that the fibrosis in the lungs is caused solely by the stone dust and that coal dust has no injurious effect upon the workmen. But Cummins and Sladden⁽⁵⁾ are of the opinion that both types of dust play an important part in the production of pneumoconiosis. Stone dust, they say, by blocking the lymph channels, impairs the powers

of the lungs to dispose of the coal dust, and that both together produce the condition of fibrosis to which they give the name "silico-anthracosis". From personal observations made in the collieries and from analyses of samples of dust taken from the roadways and working places it seems that the coal miner must inhale during his occupation large quantities of coal dust together with a certain proportion of stone dust. According to the temperature and humidity of the atmosphere so does the proportion of men affected vary.

Pneumoconiosis in the coal miner is a progressive disease and it varies in direct ratio with the dust exposure of the workman. Under modern occupational conditions definite pneumoconiosis seldom develops under nine years. But if the exposure has been intensive and continued day after day it has occurred much earlier.

The miner first complains of shortness of breath with occasional morning cough. The dyspnoea is most marked on exertion. He begins to lose weight and as time goes on he soon becomes unable to follow his occupation. On examination he may be unable to breathe deeply, for which purpose the only muscle brought into action is the diaphragm. The man seems unable to lift his upper ribs, and the breath sounds are invariably harsher than usual. The percussion note especially at the apices is impaired. Otherwise no characteristic sign may be detected.

COMPENSATION PROCEDURE.

It was not until 1928 that coal miners were in-

cluded in a scheme for the compensation of workmen in any specified industry or group of industries, involving exposure to silica dust. The scheme came into force in February 1929 and applied to those who were employed in the processes specified within the scheme on and after January 1929. The scheme only applies to those who are employed in "the processes". These are defined as mining and quarrying silica rock, which includes such work as sawing, planing, dressing, crushing and moving silica rock. Unless a man can prove that he has been employed in one of these operations he cannot claim compensation, though he be certified to be disabled by silicosis. The workman has also to prove that he has been employed in the above operations where silica rock is found.

In the 1929 scheme, silica rock is defined as including those rocks which contain 50% of free silica. This definition precluded large numbers of men from obtaining compensation because the analysis of the rock revealed a percentage of less than 50%. In some cases in the writer's practice the percentage was 47% of free silica. Again, under the 1929 scheme no compensation was payable unless a man was certified as being totally disabled.

This scheme failed to fulfil its objects because :-

1. Although it is recognised that it takes say nine years for a person to develop silicosis, all men

who were unfit prior to February 1929 were debarred from receiving compensation.

2. Every man, although suffering from silicosis, had to prove that he had been employed in the 'processes'. This was not really just, because in the anthracite coalfield the only persons employed in the 'processes' are the hardheading worker and the repairer. No account was taken of all the other underground employees who had been working in an atmosphere containing a large percentage of silica.

Owing to the anomalies created by the 1929 Scheme, the Home Office issued in 1930 new regulations which abolished the necessity of proving that the rock contained at least 50% of silica.

Again, by the Order in Council dated April 1931, the various Industries Silicosis Scheme came into operation, and it is under this scheme that present day claims for compensation are made. First of all this scheme applies to men who are employed in the above-mentioned processes on and after June 1st 1931. Secondly, to establish a claim to compensation under the old scheme, the workman had to be certified by the local Factory Surgeon, and in case of dispute was referred for decision to a medical referee, in just the same way as a man who is suffering from any other type of industrial disease, e.g., miners' nystagmus. Under the new

scheme the workman's own panel doctor certifies that the patient is suffering from silicosis on Form R.M.I. of the National Health Insurance. The approved Society then refer the person to the Regional Medical Officer of the Ministry of Health. If the Regional Medical Officer is satisfied from his examination that there is a reasonable cause for suspecting the presence of silicosis he issues a special Form R.M.98 to that effect.

Having received this certificate the workman makes an application to the Medical Board under the 1931 Scheme for a certificate of disablement. This Medical Board in the anthracite coalfield has its headquarters at Bristol. The Board is empowered to issue a certificate stating that the workman is :-

- (a) totally disabled from silicosis or silicosis with tuberculosis;
- (b) that the workman though not totally disabled is suffering from silicosis or silicosis with tuberculosis to such a degree as to make it dangerous for him to continue to work in the process, and is for that reason suspended from employment.

The decision of the Medical Board is binding and final on both sides.

Should a workman die from silicosis the doctor in attendance must report the fact to the local coroner who orders a post mortem examination. The coroner

also gives instructions that specimens of the lungs are to be sent to the Silicosis Board at Sheffield for analysis.

GOLD MINERS' PHTHISIS.

By gold-miners' phthisis is meant a deeply pigmented and somewhat solid condition of the lungs found in those following the occupation of gold-mining who during life exhibited signs and symptoms of a progressive pulmonary disease. The miner is bronzed and looks well, as if he had been exposed to the sun and the weather, but although the outward appearance is one of good health yet on the slightest exertion signs of dyspnoea soon exhibit themselves, and on examination the chest shows marked signs of fibrosis with accompanying myocardial changes.

The disease is principally met with in rock-drillers and those who work in dusty atmospheres. Rock drills driven by compressed air raise a larger quantity of dust than hand drills. In all dry mines such as the Witwatersrand, the blasting, drilling and shovelling of rock and the breaking of it by hand produce great quantities of dust which, under the microscope, are seen to be sharp particles of a silicious nature. The greatest amount of dust is created when the miners are raising or drilling a hole upwards, since owing to the upward inclination, the injection of water into the hole is rendered less easy than when drilling or boring in a hor-

izontal or downward direction. The amount of dust raised varies from .083 to .185 grains per cubic foot. So that the miner breathing the average 21 cubic feet of air runs the risk of inhaling 2.38 grains of fine dust per hour. This definitely accounts for the large quantity of silica found on analysis of the lungs of a gold miner.

It has been ascertained that of 1,377 rock drillers then employed in the Witwatersrand mines 16.34% died in the two years preceding the South African War. The average age at death of rock drill miners was under 35 years and the average number of years a man worked the drilling machines was seven to nine. The miners on the Rand are paid at piece-work rates, and work 9 - 9½ hours per day. The dangerous nature of their occupation is indicated by the high wages which the miners receive.

TIN-MINERS' PHTHISIS.

Tin mining has for many years been regarded as a dangerous occupation. During the last twenty years the deaths from lung disease amongst the Tin Mines of Cornwall have increased so that the death rate of men 25 - 45 years of age is eight to ten times that of coal miners of the same age. This has been attributed to the use of compressed air machine drills.

The quantity of dust raised in rock drilling is considerable and is conducive to the usual structur-

al changes which culminate in a fibrosis commencing as a purely dust disease. The lungs subsequently become infected by the tubercle bacillus and this hastens the malady to a fatal termination. Haldane found that out of 320 deaths of miners in the Redruth district, 141 were men who had worked rock drills; of these 141, 94% died of lung disease and the average age at death was 37.2 years. Conversely, of the remaining 179 men who had not worked rock drills, 67% died of lung disease, at an average age of 53 years.

SLATE QUARRIES PHTHISIS.

The quarrying of slate by the underground method is a comparatively modern industry in Wales. Sixty years ago the slate in the Festiniog and Meirioneth districts was worked in open quarries, but as the depth increased, and the expenses connected with the workless top became prohibitive, recourse was made to underground mining.

After having made an inclined passage along the dip of the bed and vertical tunnels having been driven along the strike, the men excavate upwards and downwards, making underground chambers. The rock is removed in slices. Blasting is done by gunpowder. But the direct danger to the slate quarryman is in the process called the dressing of the slates. The rough cutting of the slates is done by hand and then the machine cut-

ter gives the required shape to the slate. Machine cutters turn out between 600 and 700 slates per hour. The work is dusty and during the whole of the working day the men are constantly inhaling dust. They begin to suffer from Bronchitis between the ages of 35 and 40 years and once this becomes fixed, structural alterations become evident in the lungs and bronchi, and the malady proves fatal in from five to ten years. Most of the miners die from pulmonary fibrosis due to dust inhalation. French writers divide the lung disease of the slate quarryman into three stages :-

1. The period of commencement in which there are signs of pulmonary emphysema, difficulty of breathing, and dry cough.
2. The indefinite period in which the disease may be attended by accessions of asthma or followed by congestive attacks that are followed by pleurisy or pneumonia.
3. The terminal stage in which the lung becomes hard and fibrotic, owing to the large quantities of slate dust it contains.

POTTERS' PHTHISIS.

Potters' rot and potters' asthma are terms which by now have become quite familiar. The liability to lung disease has given the manufacture of pottery a high and prominent position on the list of Dangerous

Trades. This malady is now less conspicuous consequent upon the improved methods of working together with the introduction of fans and strong draughts to take away the dust from the faces of the workers.

Cheap pottery is usually made from clay, but in the manufacture of finer ware stone is added. In firing china, the cups, saucers and plates are placed in saggars or burnt clay boxes. The contents of the saggars are kept individually apart from each other by means of ground flint which can be used over and over again. The particles of flint are extremely hard, sharp and angular and these when drawn into the respiratory passages set up a considerable amount of irritation and cough. This is particularly apt to occur during the scouring or brushing of the ware on its removal from the saggars after having been in the oven. There is also a great deal of dust thrown off during the towing or smoothing of the dried ware before it is fired or biscuited. Although the scourings have been less dusty since strong draughts have been provided, the work is still dusty and if the apparatus goes wrong the atmosphere of the work-room becomes at once thick and unpleasant. The large numbers of dust particles in the air of the work-rooms is one explanation for the frequency of bronchitis and pulmonary disease in pottery workers. The average age at death from fibroid phthisis is 43, and female workers 38. In the Staffordshire potteries the mean age at death of male potters from 20 upwards was 46.5 and that

of non potters 54.

STEEL-GRINDERS' PHTHISIS.

There are two types of steel-grinding - the wet and the dry. The grinding is done on circular stone wheels driven by steam and water power. As far as the methods of grinding are concerned, the dry method is, from a health point of view, the more dangerous. The dust is dry and is in the form of a very fine powder which easily reaches the lungs owing to the attitude of the men at work. The steel-grinder sits astride his grinding wheel on a saddle and as they lean forward keeping close to their work, they cannot but inhale some of the dust which is a mixture of steel and stone. Forks and needles are usually ground by the dry method, knives and scissors and razors by the wet method. In wet grinding the running stone passes through a thin layer of water so that its surface is always kept wet; comparatively little dust is given off during the process of wet grinding. Where dry grinding is carried on there are fans but these are often ineffective. It is not uncommon to find men engaged in different processes in one large room so that the dust which is raised affects not only the workman at his own grinding stone, but other workmen as well.

It has been ascertained that in every 1000 deaths among steel-grinders in Sheffield, pulmonary phthisis is the cause of 345, and other respiratory diseases 295, i.e.

pulmonary diseases account for 64% of the entire mortality. Steel-grinders die comparatively young. Only 140 grinders out of every 1000 reach the age of 55.

STONE-MASONS' PHTHISIS.

The occupation of the stone mason and the quarryman has for long been regarded as one in which a higher death rate from lung disease occurs than in most occupations. The disease, which assumes a chronic character, is slow in its development and progress. As it is attended by all the physical signs and symptoms observed in other forms of pneumoconiosis the disease calls for attention owing to the fact that in contradistinction to miners' phthisis which occurs in men who work underground, stone-masons' phthisis is met with in men who are working in the open air. This circumstance is a strong argument in favour of the dust origin of pneumoconiosis. After a time the lung disease becomes tuberculous, hence the extraordinary fact of the death rate from pulmonary tuberculosis among stone-masons who are following an out-door occupation being six times that of bankers and brokers who are leading an in-door life. This want of harmony between occupations and mortality from pulmonary phthisis is observed in other out-door occupations than stone-cutting.

It would appear therefore as if the predisposing causes of lung disease are often of greater import-

ance than the exciting causes; in other words, the soil has as much if not greater influence than the seed.

-----0000-----

<p>WATSON, C. W. ... 1914, p. 207.</p>	<p>1914, p. 207.</p>
<p>WATSON, C. W. ... 1906, Paris.</p>	<p>France, 1906, Paris.</p>
<p>WATSON, C. W. ... 1911 1911, Vol. 6.</p>	<p>Bulletin of the ... 1911 1911, Vol. 6.</p>
<p>WATSON, C. W. ... 1911, 1911.</p>	<p>The ... 1911, 1911.</p>
<p>WATSON, C. W. ... 1911, 1911.</p>	<p>The ... 1911, 1911.</p>
<p>WATSON, C. W. ... 1911, 1911.</p>	<p>The ... 1911, 1911.</p>

B I B L I O G R A P H Y.

-
1. THE ROYAL COMMISSION ON ..
METALLIFEROUS MINES AND ..
QUARRIES 1914, H.M.S.O.
 2. CALMETTE, A..... Presse.Med. Sept. 1st
1906, Paris.
 3. COLLIS, E.L., Bulletin of Hygiene ..
April 1931, Vol. 6, ..
No.4.
 4. MACRAE, JOHN, The Ash of Silicotic .
Lungs - South African.
Institute for Med. ...
Res. 1913.
 5. CUMMINS, S.LYLE, and J. of Path. and Bact..
SLADDEN, A.F., Vol. XXXIII, 1930.
 6. HALDANE, J.S., Report of Departmental
Committee on the Phthi-
sis of Cornish Tin ...
Miners, 1902.
-

PREDISPOSING CAUSES.



It must be remembered that definite silicosis rarely develops under nine years, except when exposure to the silica risk has been intensive and continuous, when it may appear earlier. It is accordingly found that the average age of the coal miner with fibrosis of the lungs due to the inhalation of dust is between 45 and 60 years of age.

Coal mining is strenuous and is an occupation which only really fit men can undertake. One finds in the mining districts of South Wales that the occupation of coal-mining is mainly confined to families whose forebears have also been coal miners. Perhaps it is a truism to state that at least three generations are required to produce a miner who is also a craftsman. The same tendency is noticed in other trades in South Wales, e.g., tinplaters and steel workers. Such occupations, as has been stated, are strenuous in nature and quite unsuitable for persons of a weak constitution. The weakling is seldom found within the precincts of a colliery, save perhaps in a clerical capacity.

Until about five years' ago the ownership of most of the Anthracite Collieries was vested in a number of small Companies and private individuals. Since then, however, an amalgamation has taken place, and all the collieries are controlled by a large combine having

the resources and capital to provide the necessary ventilation and improvements. Improvements, long overdue, have also been effected in the circumstances and conditions of the miners. These are most noticeable in housing, hours of labour and the organised use of leisure. The housing conditions until recently were deplorable in the sense that they were not designed to provide more than the essential accommodation for eating and sleeping, whereas legislation has in later years directed attention to a larger floor area in houses for the working-classes. A most noticeable feature in this improvement is the practical use that the miner makes of his parlour, his garden, and his leisure. In the past, he found relief and change in resorting to such amenities and pleasure as the local public-house might offer; whereas now, his home and its comforts provide the inducements to devote his leisure to better advantage. Reference must be made to the public-spirited efforts of the Miners' Welfare Association in their determination to provide places for recreation, sports and cultural training for the miner. This Association is rapidly succeeding in establishing artistic and spacious Halls in almost every mining village in South Wales, where the miner may usefully engage his leisure hours. Attached to such Halls one finds tennis courts, bowling greens, football and cricket pitches, swimming baths and children's playgrounds, while within the buildings are to be found well-stocked libraries, billiard tables, writing

rooms, dance and lecture halls, and in short every amenity to satisfy the physical and mental needs of the miner. It is safe to predict that such improvements and innovations as are here indicated cannot have but salutary and beneficial consequences. It is feared, however, that their introduction has been too belated to be more than a palliative in the case of those older miners who have lived under other and more primitive conditions. Such improvements will quite definitely have an ameliorative and efficacious influence on the younger generation, and it is expected that the development of silicosis will thereby be arrested. This dissertation, however, purports to deal with miners who have not enjoyed such advantages and in whom, it is feared, the fibrosis is too far advanced.

Emphasis has already been made that the strenuous nature of mining and over-strain has an important effect in lowering the vitality of the tissues, and so predisposing the miner to pulmonary diseases. A comparison of the death rates of other occupations, such as barmen and shoe-makers, with, say, lead and tin mine workers, shews that in the latter the death rate is much higher and, what is more important, death occurs from 40 years of age onwards - the silicotic age.

Another predisposing cause is the temperature and humidity of the working-places. In local collieries where the temperature is high and the humidity low, more colliers have been certified as suffering from silicosis

than in other collieries owned by the same company, working the same seams, where the temperature of the coal face is low and the humidity high.

Heredity is also an important feature. Cases are constantly occurring where father and son, or brothers who have been exposed to the silica risk in the course of their employment, develop silicosis and later tuberculosis.

The high incidence amongst coal miners of deaths from pulmonary diseases has been fully demonstrated by (1) Collis from the Returns of the Registrar General and in the Radiological Survey of the Welsh National Memorial Association (2) as already stated. Collis's analysis indicates that although taking miners of all ages, there is a smaller incidence both of pneumonia and of respiratory tuberculosis than amongst the average general male population. He shows, nevertheless, that the incidence of these pulmonary diseases amongst coal miners in the later decades of life is well above the average. This, he points out, is more pronounced in the Anthracite miners of the Western end of the South Wales coalfield. The raised incidence in the returns for Bronchitis is remarkable. Unfortunately, the outlook of the mine-worker upon Bronchitis is to accept it as something troublesome, inevitable, yet not serious, and those who are called to treat it often do not find anything more interesting than a condition which necessitates an expectorant mixture. Although Bronchitis stands high in the causes

of invalidity, a more thorough investigation would doubtless lead to a more accurate notification of fibrosis in miners. The Returns of the Registrar General are of necessity based upon death certificates which are furnished without the aid of any special facilities to ensure precise differential diagnosis. Thus a certain proportion of cases labelled phthisis are not in fact tuberculous but phthisical from advanced silicosis with cavitation. So also with the returns of 'Bronchitis'. In the absence of radiological or post mortem examination a certificate of Bronchitis may not infrequently be given when in fact fibrosis is the major pathological condition present in the lung, accompanied by some bronchial catarrh.

Well-known investigators have directed their attention to the notoriously low death rate among coal miners from pulmonary tuberculosis, although shewing a fairly high mortality from other respiratory diseases.

(3)

Collis has found that colloidal silica or fine silica, when injected subcutaneously in sufficient amount, will cause death by direct poisoning. If injected in smaller doses local necrosis may be set up. The amounts used in such experiments greatly exceed those which reach the lungs by inhalation. Nevertheless the results indicate that silica exerts a definite chemical action on the tissues.

(4)

Gye and Purdy have observed that colloidal silica solution inoculated intravenously into rabbits

in appropriate doses can bring about the formation of excessive fibrous tissue in the liver spleen and other internal organs.

(5)
 Gardiner in 1929 experimented with guinea pigs infected with chronic tuberculosis, and found that the tuberculosis was far more evident than the fibrosis, and that death occurred long before the typical picture of silicosis could develop. Adult male industrial workers, however, are much less susceptible to the effects of the tubercle bacillus than guinea pigs, and thus in man the reverse occurs, namely, that the fibrosis appears as a rule long before the tuberculosis. Indeed the latter may not develop at all.

(6)
 Gye and Kettle have suggested that the fibrosis brought about by a finely divided silica is due to the slow formation in the tissues of silica sol, and advance two possibilities to explain the results of their animal experimentation. Firstly, that the destructive action of the silica on cells, whereby foci of necrosis are produced, provide the soil in which the tubercle bacillus can multiply. Secondly, that disorganisation of the lymphatic drainage of the lungs is produced; and thirdly, that in their view the actual cell poison is a soluble derivative of silica.

(7)
 Cummins attaches more importance to the disorganisation of the lymphatic drainage system than to the production of necrotic cell foci, and is of the opinion that the actual amount of silica sol produced

inside the phagocytes is usually too small to produce foci of necrosis. Moreover, he states that there is less necrosis to be seen in silicosis with tuberculosis than in tuberculosis alone, and that it is unusual to find marked evidence of necrosis in lungs of cases of simple silicosis.

(8)

These opinions agree with Mavrogordato, who in 1922 held that lymphatic obstruction is brought about in pulmonary silicosis by the agglutination and mummification of the dust cell. The steps in the process would seem to be :-

1. The death of the silica-laden dust cell as the result of the surface solution of the silica particles.
2. A leaking of the silica sol and the consequent agglutination of the dust cell^s in the lymph channel.
3. The preservation of these cells in masses which obstruct the lymph circulation, and lead to a still greater holding up of on-coming dust cells. This process continues until complete stasis occurs.

The silicotic lung, as is well-known, has a predisposition to intercurrent respiratory infections, especially tuberculosis and pneumonia. There is a prevailing impression that owing to the obstructed lymph drainage the pneumonia is longer in clearing up, whereas

with tuberculosis the irritation and fibrosis of the respiratory tract are the predisposing causes.

It is easily understood from the above how closely allied are these changes with those found in pulmonary tuberculosis, and how lungs affected with silicosis provide the perfect soil for the tubercle bacillus.

The question may now be asked why should not the silicosis which is so often the herald of phthisis in the gold miner have the same sinister action in the collier? It was at first thought to be due to some anti-septic action of coal dust, and Wainwright and Nicholls,⁽⁹⁾ although unable to demonstrate it, were led by their experience to postulate that coal dust did have a protective influence in the lungs against tuberculosis.

⁽¹⁰⁾
Lyle Cummins has suggested that its anti-tuberculous properties depend upon its well known power as an adsorptive agent. Tubercle bacilli act upon the tissues through the toxic products set free in their growth and disintegration. These toxic products cannot be appreciably different from those of tuberculin. Recent observations made by Cummins have proved that coal dust in fine division can adsorb and inactivate tuberculin solutions to a marked extent. When it is realised that lungs of silicotic and anthracotic coal miners may contain as much as 100 grains of coal dust it must be conceded that the available adsorption potential of car-



bonaceous matter in silicotic lungs in coal miners must be very great.

With regard to the infectivity of miners who are suffering from tuberculosis and silicosis, Watts, Irvine, Johnson and Stuart⁽¹¹⁾ have all drawn attention to the fact that sputa from such cases do not dry up quickly in the mines, but remain a menace for a long time, with the result that miners are constantly exposed to this mode of infection.

Observers have also stated that miners who suffer from silicosis with tuberculosis do not often infect others.⁽¹²⁾ Ickart, for instance, studied 400 cases of pneumoconiosis with tuberculosis among copper and slate mines in the Mansfield district of Germany. All showed marked lung fibrosis with tuberculosis and a positive sputum. They lived longer than those who suffered from tuberculosis alone and usually died between the ages of fifty and sixty. But the infection in the families was much more acute and the infected person died at a much earlier age.

These views are in accordance with the writer's own experience. The infection of members of the family, when it does occur, is a virulent one, resulting in acute tuberculosis.

B I B L I O G R A P H Y.

-
1. THE REGISTRAR GENERAL ... England and Wales(1921)
H.M.S.O., London.
 2. WELSH NATIONAL MEMORIAL . A Radiological Study of
ASSOCIATION Industrially healthy ..
South Wales Coal Miners
W.N.M. Report 1930.
 3. COLLIS, E.L., Bulletin of Hygiene ...
1931 April. Vol.6 No.4.
305-312.
 4. GYE, W.E., and PURDY, W.J. Brit. Jour.Exp.Path. ..
1922, iii 86-94
 5. GARDINER, L.U., Amer.Rev.Tub.Balt. 1929
 6. GYE, W.E. and KETTLE,W.H. Lancet, 1922, ii 855 -
856.
 7. CUMMINS, S.Lyle J. of State Medicine ..
Vol. XXXIX No. 9.
 8. MAVROGORDATO, A., Pub. of South African .
Inst. for Med. Res. No.
15, 1922.
 9. WAINWRIGHT AND NICHOLLS . American J. of Med. Sci
CXXX 403, 1905.
 10. CUMMINS, S. LYLE J. of State Medicine ..
Vol. XXXIX No.9
 11. WATT, A.H., IRVINE, L.G., Appendix No. 6 of Min-
JOHNSON, J.P., and STUART, ers' Phthisis Prev. ...
W., Committee, Pretoria ...
1916.
 12. ICKART, F., (Abstract J.A.M.A.,
Chicago 1924 lxxxiii ..
303)
-

ETIOLOGY AND PATHOLOGY OFPNEUMOCONIOSIS.

All authorities are now agreed that pneumoconiosis is caused by the inhalation of excessive quantities of dust. The fact that damage to the lungs may be brought about by the inhalation of certain dusts has long been known, but the exact type of dust responsible for the pulmonary changes is only of recent knowledge. This has been made possible by systematic Roentgen Ray studies, pathological investigation following autopsies, and abundant animal experimentation.

If one's knowledge of this subject were derived entirely from literature, one would naturally come to the conclusion that of all the dusts responsible for the serious pulmonary changes found, silica alone must be that dust. If this fact be true, why use the non-specific word pneumoconiosis and not call the condition silicosis? Why use such terms as anthracosis, siderosis, chalicosis, etc? It is quite true that anthracosis etc, is descriptive of the occupation of the affected person, the active agent in all these conditions being silica. Since these pulmonary changes have not been satisfactorily proved to be entirely due to the silica alone, and also because the nomenclature tends to confuse, one prefers the generic term pneumoconiosis.

(1)

William R. Jones in his "Silicotic Lungs: The Minerals they Contain", has lately brought forward a new

theory as to the ætiology of pneumoconiosis, namely, that Silica in the uncombined state is not the chief cause of the disease. It is his opinion that pneumoconiosis is caused by the inhalation of minute fibres of the mineral Sericite - a hydrated silicate of aluminium and potassium. He supports his contention with the following facts :-

- (a) The amount of quartz and the size and form of the quartz grains in the sandstones occurring in the underground working-places in the Scottish Coalfields and in the South Wales Coalfields was alike. The latter sandstones give rise to dust that has caused scores of cases of silicosis, whereas no authenticated case of silicosis has been produced in the Scottish Coalfields.
- (b) The gold-bearing quartz conglomerate of South Africa gives rise to dust that has caused thousands of cases of silicosis; the gold-bearing quartz rock of the Kolar Gold-field, India, contains more quartz than the South African rock and yet produces dust that has caused no case of that disease.
- (c) No quartz-bearing rocks investigated by the above author are known to have given rise to silicosis-producing dust, except those which also contain abundance of fibrous aggregate of

sericite or of fibrous silicate minerals loosely held together and easily freed into the atmosphere when the rock is drilled and blasted.

It is quite conceivable that all dusts when inhaled in excessive quantities must be to a certain extent harmful, but only a few can be regarded as actually dangerous. The dangerous types of dust can be divided into three large groups, namely :-

1. Those which are dangerous because of their poisoning action, such as lead, arsenic and mercury.
2. Those which merely cause irritation of the respiratory passages and give rise to such conditions as bronchitis and asthma.
3. Those which tend to produce pulmonary fibrosis and may thereby predispose to respiratory infection, especially Tuberculosis.

The most important pathological observations have been made in connection with the effects of free silica (SiO_2) in a fine divided state, silicates being comparatively innocuous.

The important factors in the production of fibrosis of the lungs are :-

1. The time at work or exposure to the dust.
2. The quantity of dust present.

3. The quality of dust present with special reference to silica.
4. The size of the particles.

The belief was once held that the sharp particles with their points and edges were the dangerous factors in a dust, but it is now known that this is not the case. Sharp-pointed or edged particles may be irritating to the respiratory passages, but upon this basis alone they play no part in lung fibrosis.

The pathological features of pneumoconiosis are best arranged under the following headings :-

1. The entrance of the dust.
2. The dust cell.
3. The entrance of the dust cell into the lymphatic system.
4. The subsequent conversion of the dust cell into fibrous tissue.
5. The elimination of the dust.
6. The predisposition of the fibrotic lung to respiratory infection.

1. THE ENTRANCE OF THE DUST.

A large part of the inhaled dust is removed from the inspired air before it reaches the alveoli of the lungs, due to the natural protective mechanism of the respiratory tract. Much of it is removed in the naso-pharynx where it is caught by the secretions and blown or coughed up. The mucous-bathed walls of the trachea and bronchi catch the particles, and the cilia, down as far as they exist, propel them up so that they can

be expectorated or swallowed. The very fine dust which gets beyond the ciliated epithelium reaches the alveoli and from this point the pathology of pneumoconiosis begins.

2. THE DUST CELL.

The presence of the irritating dust particles in the pulmonary alveoli sets up a catarrhal process with the proliferation of certain cells. Some of these cells at least are phagocytic and incorporate the smallest of the particles. They have been called dust cells. There has been much controversy over the exact origin and identity of the dust cell. It has been variously identified as an alveolar, epithelial cell, a mononuclear leucocyte, or an endothelial cell.

3. THE ENTRANCE OF THE DUST CELL INTO THE LYMPHATIC SYSTEM.

Many of the dust-laden cells are doubtlessly expelled through the respiratory passages but a large number reach the lymphatic system of the lungs. Gardiner,⁽²⁾ in his observations on animals exposed to granite dust, found that the cells gather in the alveoli located along the alveolar ducts and move towards the lymphoid tissue at the distal ends of these passages, thus one by one entering the lymphatic drainage system. This lymphoid tissue then undergoes hypertrophy. The cells then move to the next larger lymphoid deposits at the bifurcation of the bronchioles and bronchi, and eventually reach the tracheo-bronchial lymph nodes. If the

lymphatics become choked by a continued dust inhalation, fibrosis ensues and the dust can no longer be removed, resulting in a progressive fibrosis.

4. CONVERSION OF CELLS INTO FIBROUS TISSUE.

The prevailing opinion is that the dust cell becomes a fibro-blast. Mavrogordato⁽³⁾ has caused excessive fibrosis in the peritoneal cavity by dusting it with sterile silica; while Gye and Kettle⁽⁴⁾ experimentally proved that colloidal silica was a cell poison, and they believed that the fibrosis is caused by finely divided silica acting in the form of hydrated colloidal silica.

Fibrosis is the essential pathological process in pneumoconiosis and upon it and lymph block depend characteristic X-Ray features. Most recent observers believe that the presence of silica induces a connective tissue cellular proliferation such as is produced anywhere as a reaction to an irritant. It seems probable that as the dust cells die or become fibrous tissue, the dust particles and pigment particles, like coal, if not dissolved, become extra-cellular and lie within the fibrous tissue.

This is the first stage of pneumoconiosis and in brief depicts a fine, practically microscopical nodular fibrosis along the courses of the lymphatics, arising in the primary lobules and extending centrally along the pulmonary arterial branches, pulmonary venous-radicles and bronchi. This process continues until the fi-

brosis in and around the lymphoid deposits becomes visible as a uniformly scattered nodular fibrosis and produces the second stage of pneumoconiosis.

Coinciding with the formation of nodular fibrosis is the beginning of the third stage. The lymphatic channels become obstructed; there is more clogging with cells and a more diffuse fibrosis begins in the interstitial lung tissue. The interalveolar tissue and the inter-lobular septa become thickened because they contain blocked lymphatics. At first the lymph flow remains fairly active towards the hilum and the tracheo-bronchial lymph nodes. These enlarge and become fibrotic. Many cells are caught on the way hilumward in the lymphatics which extend centrally and cause fibrosis and clogging along the bronchial tree. Naturally as the lymph block towards the hilum becomes marked there is a tendency for a back flow towards the sub-pleural lymphatics whence similar changes are instituted which result in fibrous nodules, thickened pleura, adhesions and sometimes even effusions. Thickened pleura and adhesions are found in a large percentage of advanced silicotic cases. Thus, as certain sets of lymphatics become blocked, the burden of lymph flow is thrown on others which in turn become similarly affected.

In cases of very moderate silica-inhalation, the lymph flow is maintained hilumwards, and the individual may not in the course of 40 to 50 years progress past the first stage, or may show evidence of second

stage nodular fibrosis in only those portions of the lungs where the greater part of the inspired air goes while the remaining portion will remain clear. But if the inhalation of silica is in greater quantities, widespread changes will occur. The small fibrotic nodules increase in size and coalesce to form larger nodules. Thus it is seen that there are two processes going on simultaneously. First the nodular fibrotic change, and secondly the generalised fibrosis. If the inhalation remains moderate the generalised fibrosis proceeds very slowly and the condition may remain just a nodular fibrosis, but if the dust is more intensive the generalised process takes place more rapidly and diffuse fibrosis soon becomes the prevalent condition.

Lungs in the third stage fibrosis are inelastic and incapable of expansion by respiratory effort because of the excessive generalised fibrosis, fibrous buds and plural adhesions limiting the diaphragmatic movements and costal expansion. Moreover, they show distinct evidence of emphysema. Some observers state that the intense dyspnoea is due to the obliteration of the alveoli and compression of the bronchioles and capillaries by the generalised fibrosis rendering the lung unable to oxygenate the blood.

The third stage of fibrosis, therefore, may take on three distinct appearances, or, alternatively, they may be present simultaneously in the same lung. These appearances are :-

- 1. A diffuse fibrosis with nodules still present.
- 2. Large nodules due to coalescence of small areas,
or
- 3. Large irregular areas of consolidation due to massive fibrosis.

5. ELIMINATION OF DUST AND RECOVERY.

It is a well established fact that individuals who have not progressed far into the third stage of pneumoconiosis may show a decided clinical and X-Ray improvement upon cessation of their dusty occupation. This has been observed when miners' work has been interrupted by strikes and other reasons, and is due to the fact that the lung is capable of freeing itself even after the dust has entered the lymphatic system, by a reversal of the process of entrance, provided that new dust is not entering too fast.

One important statement concerning the progressive nature of the disease has been made by Mavrogordato. (5) He states that if the condition of a person worsens after cessation of work then some infection, especially tuberculosis, is indicated. The writer has confirmed this theory on several occasions from his personal observations in his own practice, and would cite the case of a patient, now undergoing treatment, who, five years' ago, had to relinquish his work on account of miners' nystagmus. He at that time suffered from pneumoconiosis of the second stage, and but for his nystagmus would have been able to perform his usual work. Although he has not worked for five years he has now developed

definite signs of pneumoconiosis with tuberculosis. Mavrogordato thinks that these cases are all in a condition of 'latent silicosis', and that the determining factor is the tubercle bacillus. He states further that the dust cells have two ways of exit, namely :-

1. By expectoration, and
2. By travelling in the blood or lymph stream out of the lungs.

PATHOLOGICAL ANATOMY OF THE COAL MINERS' LUNGS.

The most characteristic feature seen post mortem in most miners' lungs is generalised blackening. This is by no means uniform, but tends to be more concentrated in the upper than the lower lobes. The lungs are usually anchored to the parietes at a few points, by many tough fibrous adhesions. The lung itself is slatey grey in colour, heavy, and presents over its upper part great distortion of the surfaces by puckering. The pleura is generally thickened and greyish white in colour, and over the fibrotic areas it is puckered up like a cicatrix. If, as is usual, the apical pleura is dense, the black colour of the lung is completely obscured, but where the pleural fibrosis is less dense a mottled appearance is the result.

On cutting through the lung the characteristic feature is the presence of areas of consolidation varying from black to dull grey in colour. These cut with a distinct feel as if the knife were going through india-rubber. These areas are most often situated in the apices

of the upper or lower lobes. They may exhibit fine whorls or may resemble lumps of coal. Elsewhere, throughout the cut surface of the lungs are seen black spots varying in size from that of bird-shot up to the size of buck-shot. These are well-defined in shape and stand out in sharp contrast to the grey background of the aerated lung substance. Seen through the pleural surface of the intact lung these areas are usually circular, often showing a white or a greyish white centre surrounded by a blackening. These black spots may be few and widely separated or numerous and close together. Usually they are more numerous in the upper portions of the lungs where they may be so closely accumulated that they pass into the consolidated areas already mentioned. They are firm and nodular to the touch and are easily marked out in contrast to the softer lung substance not so far affected. Emphysema is a common finding, especially marked in the free edges of the lungs. It is also present in the interstices between the black areas and the maculae.

It is common in advanced cases to find the consolidated areas occupying almost the whole of the upper parts of the lung and most of the lower parts as well, and only a very little aerated portion of the lung substance remaining. When this advanced stage has been reached it is usual to find that there is a history of the man having worked in 'hard headings' and that he must have been exposed to a high concentration of stone

dust. In such cases it is not uncommon to find a tendency to a softening and breaking down into cavities in the consolidated areas. These cavities exude a black inky fluid. This fact explains the black spit of miners. The cavities may occur quite independent of any tuberculosis. Histological examinations often show that the lungs were quite free from tuberculosis. Tuberculosis may, of course, tend to cavitate, but it is quite common to find cavitation without the presence of tuberculosis. Microscopic examination of these cavities generally shows bacterial invasion of fibrotic coal-laden tissue which forms the wall of the cavity.

MICROSCOPIC CHARACTERS.

The outstanding pathological changes seen on section are fibrous hyperplasia with dust accumulation. The alveoli are crowded with large monocytes, the cytoplasm of which is filled with coal dust. The fine dust particles which gain entrance to the alveoli do not long remain free but are rapidly engulfed by the large phagocytic cells. It has been proved that the dust cells retain their power of amoeboid movement and to penetrate the alveolar walls, since they can be seen in the perivascular and peribronchial lymphatic channels in the lymph nodes, and at the root of the lungs.

A typical early lesion consists of a central blood vessel or bronchiole surrounded by a cor-

ona of dust containing cells. Later, the central blood vessel may become considerably thickened. The surrounding dust cells are so full of dust particles that the nuclei are hidden, and they look like small areas of coal dust. This accumulation of dust cells is confined within the limits of the distended alveolar walls, and the resulting back pressure, combined with the arrival of new dust cells gives the medusa head appearance.

Later on hyperplasia becomes the outstanding feature and leads to the formation of fibrous nodules, which may or may not show lamination. A certain amount of coal dust is often seen in the interior of the nodules, but it is usually free, suggesting that the dust cells have been obliterated by the fibrous bands. The lung tissue in the areas of black consolidation is entirely replaced by diffuse fibrosis consisting of confluent nodules between which lie collections of dust in cells. The latter are densest in situations along which the lymph flow may still find its way, under the pleura, for example, which itself is often greatly thickened and intensely fibrotic.

THE LYMPHATIC GLANDS.

Sections of the blackened lymphatic glands show that here again the salient features are the formation of fibrous nodules and the accumulation of dust-laden phagocytes around them. When the dust cells accumulate in a gland the lymphoid tissue tends to disappear, so that on section, areas of fibrosis and dust cells, al-

B I B L I O G R A P H Y.

-
1. JONES, WM. R., Silicotic Lungs, J. of Hygiene, Vol. XXXIII, No. 3.. 307-329.
 2. GARDINER, L.U., Amer. Rev. Tuberc. Baltimore 1920, 1V, 734-755.
 3. MAVROGORDATO, A., ... Pub. of South African Inst. for Med. Research, No. 15 . March 31, 1922
 4. GYE, W.E., and KETTLE, E.H., Lancet, 1922, ii 855-85.
 5. MAVROGORDATO, A., Pub. of South African Inst. for Med.Res. No.15, March . 31, 1922.

ternate with areas of normal lymphoid tissue in which dust particles are rare.

-----0000-----

1. JONES, W. W., *Ann. N.Y. Acad. Sci.*, 1951, 54, 1-10.

2. JONES, W. W., *Ann. N.Y. Acad. Sci.*, 1952, 56, 774-785.

3. JONES, W. W., *Ann. N.Y. Acad. Sci.*, 1953, 58, 1-10.

4. JONES, W. W., *Ann. N.Y. Acad. Sci.*, 1954, 60, 1-10.

5. JONES, W. W., *Ann. N.Y. Acad. Sci.*, 1955, 62, 1-10.

PERSONAL AND CLINICAL WORK.

Up to 1st June, 1933, the numbers of certified silicotics in the Anthracite Coalfield were as follows:-

1. Ystalyfera	17
2. Cwmtwrch	8
3. Cwmllynfell	4
4. Gwauncaegurwen	17
5. Ystradgynlais	1
6. Garnant	1
7. Ammanford	16

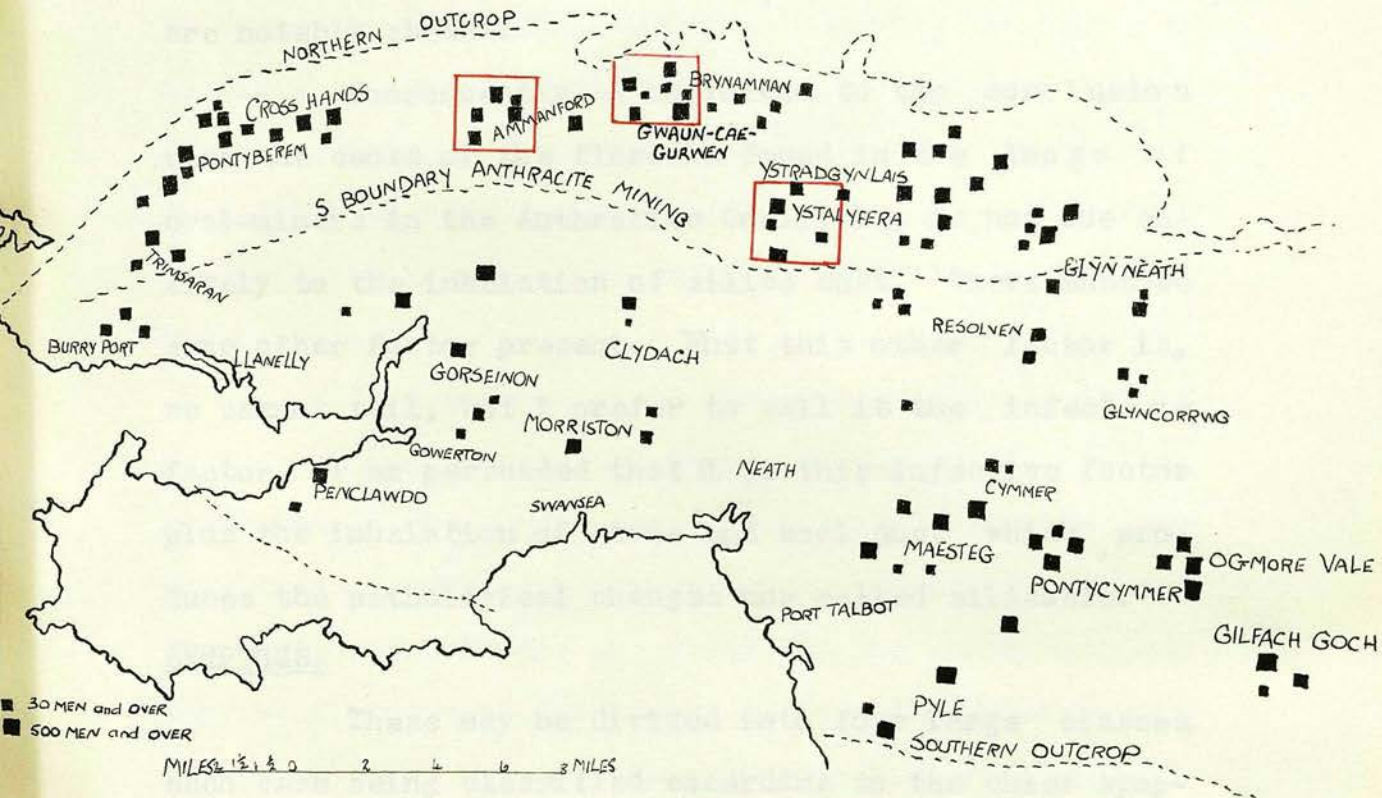
It can be seen from the above that there are three areas where pneumoconiosis is more prevalent than the others.

I have found that the mines in which the greatest number of pneumoconiosis cases occur are not necessarily those in which silica rocks of the type specified in the Silicosis Order 1931, are found. The rocks mentioned in this Order are gritstones or sandstones and quartz in the form of quartz veins. In fact the contrary often seems to be the case. The mines in the three areas specified are notably free from silica rocks. The only silica rock in these mines is quartz in the form of small quartz veins. It must be added, however, that the mines are very dry and dusty.

There also seems to be a definite relationship between the incidence of silicosis and the nature of the coal worked, for the incidence of silicosis in-

MAP SHOWING COLLIERIES IN SOUTH WALES WITH NUMBER OF MEN

EMPLOYED



AREAS ENCLOSED IN ARE THE THREE "PEAK" AREAS
AS REGARDS THE CARBON CONTENT OF ANTHRACITE COAL,
THE INCIDENCE OF PNEUMOCONIOSIS IS HIGHEST IN THESE AREAS.

creases with the carbon content of the coal. It is significant that these areas where silicosis is so rife are all noted for the high quality of the carbon content of the coal; that the mines are very dry and dusty; and that silica rocks as specified in the Silicosis Order of 1931 are notably absent.

Consequently, I am forced to the conclusion that the cause of the fibrosis found in the lungs of coal-miners in the Anthracite Coalfield, is not due entirely to the inhalation of silica dust. There must be some other factor present. What this other factor is, we cannot tell, but I prefer to call it the infective factor. I am persuaded that it is this infective factor plus the inhalation of stone and coal dust which produces the pathological changes now called silicosis.

SYMPTOMS.

These may be divided into four large classes, each case being classified according to the chief symptom of which the patient complains. Whenever a coal miner complains of one or more of these symptoms, I make it a rule after taking the history and making a thorough examination of the heart and lungs to take a skiagram of the chest. In the early stages a skiagram is most useful in arriving at a definite diagnosis.

The first great class of symptoms is that in which the patient complains of 'shortness of breath'. He may complain of this symptom alone or it may be accompanied by cough, expectoration, lassitude, loss of weight

etc. This class comprises 80% of the miners examined.

Secondly, 5% of the patients examined complain of paroxysmal attacks of cough and dyspnoea - usually called asthma by the miner. These paroxysms usually occur at night and on sudden changes in the atmospheric conditions. Such paroxysms occur suddenly and are relieved when the patient brings up a quantity of viscid sputum. During the intervals the patient is dyspnoeic, especially on exertion, and he is often more comfortable, particularly in the early stages, when actually working underground.

In the third class the patient complains of pain and thumping over the region of the heart. An examination of the cardiac area invariably shews that the condition of auricular fibrillation is present. Although the examination of the chest reveals the presence of extensive fibrosis of the lungs, I find, nevertheless, that the patient has actually been at work the previous day. This class represents 10% of the cases examined.

The patient in the fourth and last class complains that he has had influenza but that he has failed to make the usual rapid recovery. He looks pale and ill and complains of lassitude, palpitation, poor appetite and loss of sleep. An examination soon shews the presence of a bilateral fibrosis with probably an accompanying myocarditis. This explains why he does not recuperate as quickly as others who have no other lesion present. The remaining 5% were in this category.

PHYSICAL SIGNS.

Inspection.

Patients are usually fairly well covered unless infected with tubercle. The movements of the chest wall are not uniform and the upper part may be immobile.

Palpation.

There is definite restriction of movement especially marked at the bases. Vocal fremitus is slightly increased. The apex beat of the heart is pulled over to one or the other side.

Percussion.

There is usually dullness over one or other lobes. The percussion note throughout both lung fields is impaired. If there is emphysema the note is naturally tympanitic over the emphysematous portions of the lung.

Auscultation.

The breath sounds vary from a harsh prolonged expiration to the bronchial. The characteristic type of breathing which, in my opinion, is now practically pathognomic, is the characteristic harshness which occurs at the end of the inspiratory phase. The accompaniments vary according to the amount of catarrh present, and whether the condition is accompanied by tuberculosis, the latter condition being very liable to develop in a pneumoconiotic lung.

If there is marked clinical evidence of cavitation, the case is complicated by tuberculosis. In ad-

vanced cases it is common to hear post tussive crepitations.

X-RAYS.

Most observers are now agreed that a properly conducted X-Ray examination is the most satisfactory and usually the most accurate means of studying the effects of stone dust upon the lungs. But it must not be forgotten that before expressing an opinion on any X-Ray film it is most essential to know the history of the case along with the physical signs.

(1) The Chairman of the South African Miners' Bureau states that owing to the paucity of the physical signs in a clinical subject suffering from silicosis, it is not safe to venture upon a positive diagnosis without the assistance of what he calls a "technical satisfactory radiograph". He is the authority for the very frequent confirmation of the accuracy of X-Ray findings by autopsies which are permitted by law in South Africa. Doubt only exists in the very early stages. Only when the specific appearances of the fine mottled stage are found is it invariably safe to venture upon a positive X-Ray diagnosis.

The Technique of X-Ray examination is important. As we are dealing with the fine details in the lungs, the same care must be taken as for examination for tuberculosis of the lungs. In fact, tuberculosis may

be superadded and play a part in the appearances to be interpreted. Speed of exposure is essential in order to avoid blurring due to heart and vessel pulsation, for when examining for fibrosis pulsation is apt to blur details. Again, most of the advanced cases of silicosis are dyspnoeic and therefore it is very difficult for them to abstain from breathing even for a second or two. Thus it is not advisable to ask such cases to take the deepest possible inspiration, especially the extreme dyspnoeic ones.

The X-Ray is often of great value in determining the respiratory capacity and for observing the effects of fibrosis upon the diaphragm. In most cases, particularly the advanced, the diaphragms are nearly or quite immobile, due to the inability of the fibrotic lung to expand or from plural adhesions or both.

It seems logical that the stages of progress should be based on X-Ray appearances. A clinical classification would be based largely upon progressive dyspnoea and capacity for work, which, as a matter of fact follow very closely the X-Ray appearances.

(2)
Watt, Irvine, Johnson and Stewart described these pathological stages as :-

1. Early silicosis.
2. Intermediate silicosis.
3. Advanced silicosis.

(3)
Lanza and Childs presented the first American classification of the stages based upon X-Ray find-

ings. They also divided the progress of the condition into three stages :-

First Stage. Where the root shadows are denser and more extreme and may show nodes: the trunk shadows are increased in density and breadth, with numerous punctuate deposits of varying size. The appearance is symmetrical on both sides at first but not so later. There is no difference in diaphragmatic excursion.

Second Stage. In addition to the foregoing there are found fairly symmetrical small circumscribed dense areas throughout both lungs, and later larger masses accumulate, usually at the lower part of the upper third, about the level of the root shadows. The domes of the diaphragm are accentuated.

Third Stage. This differs from the second stage only in the extent of lung involvement indicated by increased numbers of deposits and more massive grouping.

(4)

Mavrogordato states that at the present time silicosis is legally divided into three clinical stages:-

1. The ante primary stage in which the earliest detectable physical signs are present but no X-Ray changes.
2. The Primary Stage in which definite physical signs are present and capacity for work is impaired but not seriously.
3. The Secondary Stage in which the capacity for work is seriously and permanently impaired.

It has been the custom in the X-Ray examination of the coal miners in my practice to take all radiograms according to a standardised technique.

1. Position of Patient.

He stands with his back to the tube, with his arms drawn forwards and rotated upwards to remove shadows of scapulae.

2. Phase of Respiration at which X-Ray is taken.

Partial inspiration with the focus of the tube centred at the level of the third chondro-sternal joint.

3. Film Target Distances - 30 ins. approximately.

4. Spark Gap - 3 ins.

5. Current - 60 milliamps.

6. Exposure.

The duration of exposure varies with :-

1. Type of Patient

- (a) If lean - relative short exposure.
- (b) If fat and emphysematous - longer exposure.

2. On Tube

- (a) If tube is soft it needs a longer exposure.
- (b) If tube is hard relative shorter exposure.

The average exposure on a Mullers water cooled tube is 0.75 sec. at 60 milliamps.

Of course one can shorten the exposure by increasing the milliamperage depending on the type of patient.

All the X-Rays are standardised as above, namely, 3" spark gap at 60 milliamps on a Muller water-cooled tube for 0.75 seconds.

Developing.

All films are developed in Kodak's standardised developer for five minutes at 65° F. They are washed for two hours.

Sputa Examination.

Good specimens are very necessary and usually it is found that the administration of small doses of potassium Iodide, say, 3 grs. is very useful to produce the desired effect.

The examination of all sputa is carried out:-

1. By the Zeihl Neilsen test.
2. By the antiformin concentration method, and
3. By guinea pig inoculation.

It is important to stress the last method, for suspected cases, after having been found with a negative sputum with both the Zeihl Neilsen, and the Antiformin Tests, have proved positive on inoculation into guinea

pigs.

Just as Watt, Irvine, Johnson and Stewart,⁽²⁾ and later, Lonza and Childs⁽³⁾ described their pathological findings according to the radiograms taken, I, too, have to describe these changes in the following manner :-

1. The pre-silicotic stage.
2. The first stage of Silicosis.
3. The second stage of Silicosis.
4. The third stage of Silicosis, and
5. Silicosis with superadded tuberculosis.

THE PRE-SILICOTIC STAGE.

Excessive striation and reticulation of the lung fields are the characteristic features of this stage. These are usually symmetrical but not necessarily so. Oftentimes, there is evidence of lymphatic obstruction as seen by the interruption of the lung striation. The diaphragm leaves are usually peaked - due to adhesions; and flattened - due to plural involvement. The costophrenic angle is frequently obliterated or ill-defined. There is no change to be seen in the mediastinum.

The patient does not lose work and finds very little difficulty in following his usual employment. He makes no complaint apart from a slight morning cough,

and dyspnoea, unless he is engaged in unusual tasks of a strenuous nature.

FIRST STAGE SILICOSIS.

The actual dividing line between the first stage silicosis and the second stage is difficult to describe. The characteristics of the first stage are:-

1. Scattered bilateral symmetrical stippling of a coarse type frequently more discernible on the right side.
2. The basal regions often shew compensatory emphysema interpreted by the denseness of the basal regions and the wideness of the ribs.
3. The diaphragm may shew irregularities due to adhesions or pleural involvement and there is an occasional thickening of the parietal pleura over the lungs.

The patient complains of cough and dyspnoea which becomes marked on strenuous work. Many miners undergoing treatment are still able to continue their usual occupations, while others - and these are in the majority - are only able to do light work in the open air.

SECOND STAGE SILICOSIS.

In this stage the mottling in the lung fields is more marked. There is evidence of thickening of the peripheral pleura over the upper and mid Zones and the

areas of fibrosis are more localised and consolidated. There is usually a greater degree of emphysema present. The diaphragm leaves are flattened and frequently peaked to a great extent. The mediastinum and heart are occasionally drawn to one or other side. The patient complains of more dyspnoea with cough and expectoration. He begins to lose weight, and is only able to do the lightest work. His capacity for work decreases as the second stage merges into the third stage.

THIRD STAGE SILICOSIS.

Here we have definite evidence of consolidation in the lung fields. The areas are more or less symmetrical and limited to the upper zone, occasionally being more marked on one side than the other.

Below these areas there are definite changes of a fibrotic nature, which may take the form of scattered areas of fibrosis as exemplified in stage 2, or extensive irregularities of the diaphragm are visible owing to the adhesions and fibrosis in the lung above. Pleural thickening is marked especially over the upper zones, and mediastinal displacement to one or other side, depending upon which side there is most fibrosis. The trachea is displaced from the mid-line.

The differential diagnosis of superimposed tuberculosis is an extremely important and difficult factor in this stage.

On reaching this stage the miner is totally and permanently incapacitated. He complains of cough with

copious expectoration. The sputum is purulent and often black in colour. Lassitude and loss of flesh is marked. The chief symptom, however, is dyspnoea, which occasions great distress.

THIRD STAGE - SILICOSIS WITH SUPERIMPOSED TUBERCULOSIS.

The general appearances are those already enumerated in stage 3. It is sometimes practically impossible to say if the tuberculosis is the primary condition present in the lungs or whether it is superimposed upon a silicosis which is already present. The apices are frequently involved in the third stage and as such, all films should be queried for tuberculosis. If there are definite radiological changes in the lung tissue indicating cavitation to any extent, a fairly dogmatic statement can then be made that tuberculosis is complicating the initial lesion. Silicosis, it is well known, does cause cavitation but of a very minor degree, and as such cannot be interpreted on chest skiagrams. Tuberculous cavities on the other hand can be diagnosed with a definite degree of accuracy.

Dyspnoea of an extreme type, cough with copious expectoration, weakness, loss of flesh, sleeplessness, night sweats, render the patient permanently incapacitated from any kind of work.

B I B L I O G R A P H Y.

-
1. WATKINS-PITCHFORD Med.J. Australia, Sydney
1923, **ii**, 382 - 384.
 2. WATT, A.H., IRVINE, L.G.,
JOHNSON, J.P., STEWART, W. Appendix No.6 of Miners'
Phthisis Prevention
Committee of South
Africa.
 3. LANZA, A.J., and CHILDS,
S.B., W.S.Pub. Health Bull.No.
85, Jan.1917.
 4. MAVROGORDATO, A., Public.of S.A.Inst. for
Med.Res. No.15 March 31.
1922.
-

AN ACCOUNT OF FOUR
POST MORTEM EXAMINATIONS.

METHOD OF EXAMINATION.

Prior to microscopic and chemical examination the lungs were X-Rayed and specimens removed from representative areas, enabling a correlation of those factors which are necessary in arriving at a conclusion.

Reference to photographs will show the varying texture of the lung tissue. Pieces for examination were removed from areas indicated by the numbers. A general view of the nature of the lung tissue is also well shown in the photographs of the lung after coronal section.

All photographs taken were printed to the same size and in the relative positions.

FURTHER NOTES.

The chest contained quantity of free fluid.

Both lungs were intensely black except for two small areas 2" in diameter, which were pink in colour. They were adherent to the chest wall in many places. The alveolar adhesions were particularly numerous over the apices.

The upper lobes of both lungs were hard and

POST MORTEM NO.1.

EVAN JONES - AGE 59.
15 GLANYRAFON ROAD,
YSTALYFERA.

DEATH - 11/3/33. POST MORTEM - 13/3/33.

CLINICAL HISTORY.

This man had worked for 47 years as a coal miner. For over 27 years he had been employed on 'hard headings'. He stated that he could not get accustomed to a face mask and that he had never been supplied with a dust trap for his boring machine.

He was first seen on 27/1/33, his only complaint being 'shortness of breath'.

On examination, I found signs of extensive bilateral fibrosis of the lungs with the heart in a state of auricular fibrillation.

He died on 11/3/33.

POST MORTEM.

The chest contained a large quantity of free fluid.

Both lungs were intensely black except for two small areas 2" in diameter, which were pink in colour. They were adherent to the chest wall in many places. The pleural adhesions were particularly numerous over the apices.

The upper lobes of both lungs were hard and

stoney to the touch and their pleural covering was very adherent, deep white in colour, and in parts puckered up like a cicatrix. These areas when cut across gave the operator the sensation of cutting through india-rubber. On Sections all lobes of the lungs shewed extensive areas of fibrosis. These nodules varied in shape and size. Their diameters were from 1" to 3". The extreme apices and anterior margins, although black in colour, were soft and crepitant.

HEART.

This organ was enlarged. The pericardium over the left ~~ventricle~~ was thickened and the myocardium soft and degenerate. The left ~~ventricle~~ was hypertrophied but showed no signs of any valvular disease. The right ~~ventricle~~ was thin and dilated.

LIVER.

The Liver was slightly enlarged and showed evidence of passive congestion owing to myocardial failure.

CONCLUSIONS.

1. That both lungs showed extensive fibrotic involvement.
2. That the remaining areas of the lungs were deeply pigmented and emphysematous.
3. That the condition of the heart and liver showed that there had been considerable strain on the heart muscle with consequent back pressure on the liver, all being primarily due to the obstruction in the lungs.

MICROSCOPIC EXAMINATION.

Section No.1.

The pleura is thickened, and infiltrated with

round cells. The remainder of the section is composed of one mass of fibrous tissue with large amounts of coal dust. Under the high power, the black pigmentation is so dense that the reflected light is unable to penetrate to the lens. The absence of blood-vessels is a marked feature.

Silica content of ash - 41.62%.

Section No.2.

There are irregular areas of emphysema. The pleura is markedly thickened and the inter-lobular septa are sclerosed and loaded with coal dust. The alveoli are angular in shape. The coal dust seems to have collected at the angles. The nuclei which can be seen are all oval in shape.

Silica content of ash - 26.46%.

Section No.3.

This Section shows scattered areas of emphysema and fibrosis. The fibrotic areas are not nodular as one would expect. The alveolar walls seem to have collapsed with, as one would think, the weight of the coal dust. The air spaces show but faintly up through a dense black mass. The blood vessels are sclerosed and the bronchi are infiltrated with round cells.

Silica content of ash - 27.36%.

Section No.4.

Similar to Section No.3, except that here the fibrosis is of a nodular type.

Silica content of ash - 24.28%.

Section No.5.

The pleura is markedly thickened and infiltrated with round cells. The remainder of the section is composed of one dense mass of fibrous tissue deeply pigmented with coal dust. One small blood vessel is to be seen with its outer coat markedly sclerosed.

Silica content of ash - 36.04%.

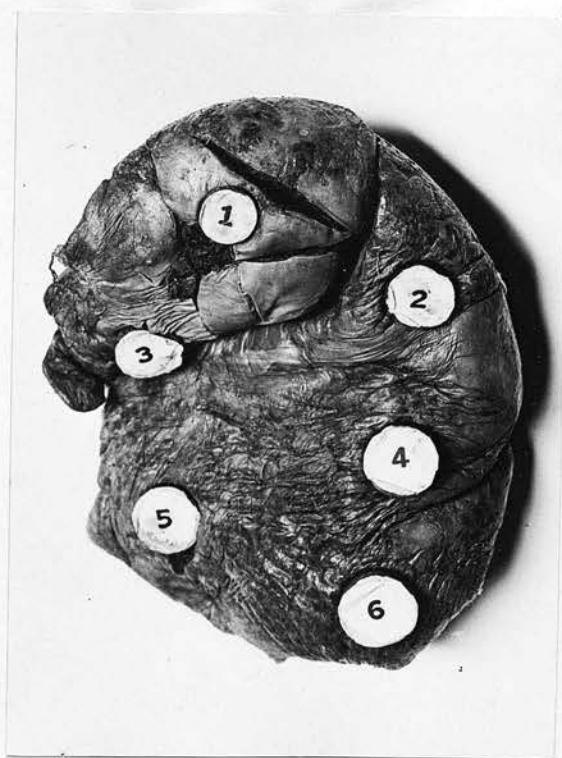
Section No.6.

Areas of Emphysema with alternating nodules of fibrosis are the marked features of this section. The outer coats of the blood-vessels are sclerosed. There is some round-celled infiltration of the bronchi.

Silica content of ash 18.41%.

CONCLUSIONS.

1. Both microscopically and chemically the examination reveals a very advanced stage of silicosis.
 2. The functional value of the lungs must have been of a very low order.
-

PHOTOGRAPH NO. 1.

Photograph of Lung before section showing nature of pleura and the numbered areas from which specimens were taken.

PHOTOGRAPH NO. 2.



Lung after coronal section showing nature of the lung tissue. The varying areas of fibrosis and emphysema are well shown.

POST MORTEM REPORT NO. 2.

ZOROBABEL JONES - AGE 52.

DEATH - 4/6/33.

POST MORTEM - 6/6/33.

CLINICAL HISTORY.

This man gave a history of having worked as a coal miner for thirty-nine years. His only illness previous to 20/5/32, when he was first seen, was 'Bronchitis', which occurred every winter.

On 20/5/32 he complained of :-

1. Shortness of breath which was marked on exertion.
2. A troublesome cough with copious purulent sputum which was sometimes blood-stained.
3. Loss of weight and sleeplessness.

On November 10th, 1932, he developed a carcinoma of the penis.

He died on 4th June, 1933.

POST MORTEM EXAMINATION - 6/6/33.Externally.

The body was very emaciated. On the end of the penis was a large septic cancerous ulcer. In the left inguinal region were several ulcers varying from $\frac{1}{2}$ " to 1" in diameter. These exuded pus.

Internally.

Both lungs were intensely black in colour and

very adherent posteriorly, by pleural adhesions. These adhesions were thick and were broken down with difficulty. The bands of adhesions were particularly marked at the apices and posteriorly. The pleural membrane covering both lungs was thickened and deep white in colour. This was puckered up into fine scars. The latter were especially marked at the apices.

The upper lobe of the left lung was consolidated. The remainder of the left lung showed the presence of intense fibrosis and contained numerous black putty-like areas varying from $\frac{1}{4}$ " to 2" in diameter. Some of these areas were beginning to break down and on pressure exuded a thick black fluid. The right lung, although also deeply pigmented, was not so severely affected, except for one large fibrotic mass, very hard to the touch, and about 2" in diameter, situated in the upper portion of the lower lobe. The free edges of both lungs were soft, crepitant, and emphysematous.

Penis.

There was a large septic cancerous ulcer involving the end of the penis. The inguinal glands on both sides were enlarged ulcerated and septic.

Heart.

The heart was small in size, the myocardium being very soft and degenerate.

Liver.

The liver showed signs of passive congestion owing to myocardial failure. There was no naked eye

evidence of any secondary deposits of cancer.

CONCLUSIONS.

1. That deceased had a lobar pneumonia of his left upper lobe.
2. That both lungs showed evidence of dense fibrosis.
3. That the free edges of the lungs were emphysematous.
4. That although there was extensive cancerous disease of the penis, there were no secondary deposits to be made out by the naked eye in the lungs or liver.
5. That the condition of the heart and liver showed that there was considerable obstruction to the circulation through the lungs.

MICROSCOPIC EXAMINATION.

Section No.1.

This section shows the lung in the typical state of grey hepatisation of lobar pneumonia. The alveoli are packed with fibrin and polymorphonuclear leucocytes. The inter-lobular septum is thickened and full of dust cells. The vessels show marked thickening. There is a small fibrous nodule at the junction of the inter-lobular septum.

Silica content of ash - 21.18%.

No.2.

Section 2. shows a similar picture to section No.1. The lung is consolidated. There are two or three small fibrous nodules to be made out with a tiny capillary in the centre of one of them. The perivascular lymphatics are blocked and surrounded by marked fibrosis. The vessels show extreme thickening.

Silica content of ash - 27.89%.

No. 3.

Extreme emphysema with nodular fibrosis are to be seen in this section. The pleura is irregularly thickened. The inter-lobular septa are markedly thickened and filled with coal dust. The collections of coal dust are most marked at the angles of the air sacs. The vessels show marked thickening.

Silica content of ash - 27.89%.

No. 4.

Scattered areas of grey hepatisation are seen in this section again. The vessels show extreme thickening and a radial arrangement of the cell nuclei.

An interesting addition is now discernible. There are a few large endothelial cells to be seen about 14 - 15 microns in size with pale oval nuclei. They are rather indistinct in the eosinophil protoplasm. Here we have the first signs of a metastasis in the lung from the cancer of the penis.

Silica content of ash - 25.22%.

No. 5.

Section 5 shows small areas of grey hepatisation with packed alveoli, a few small nodules of fibrosis about the size of millet seeds and marked thickening of the blood vessels. The remainder of the section shows scattered areas of emphysema and fibrosis.

Silica content of ash - 17.31%.

No. 6.

A typical picture of an implantation metastas-

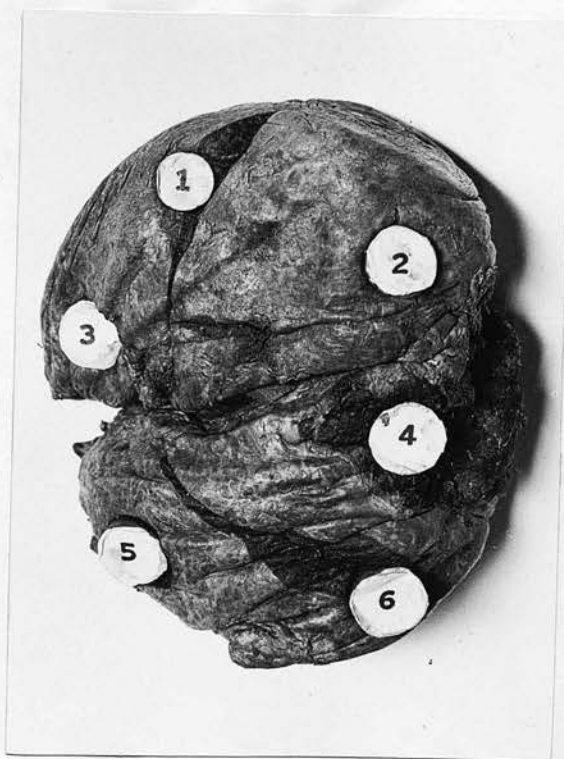
is from the carcinoma of the penis. The field under the high power is simply filled with large oval and spindle shaped endothelial cells with large darker coloured nuclei.

Under the low power are seen areas of nodular fibrosis with collections of coal dust. Areas of extreme emphysema are also seen. The inter-lobular septa are thickened and also filled with coal dust. The vessels show marked thickening.

Silica content of ash - 15.04%.

CONCLUSIONS.

1. That microscopical examination reveals a fibrosis of the lungs of a fairly extreme type.
 2. That there was present a lobar pneumonia in the state of grey hepatisation.
 3. That areas of marked emphysema were present.
 4. That definite implantation metastases from the carcinoma of the penis had occurred in the lungs.
 5. That the lungs were typical of advanced silicosis with secondary carcinoma super-added.
 6. That the functional value of the lungs must have been of every low order.
-

PHOTOGRAPH NO.1.

Photograph of lung before section showing nature of pleura and the numbered areas from which specimens for examination were taken.

PHOTOGRAPH NO. 2.

Lung after coronal section showing nature of the lung tissue. The areas of consolidation and the areas of fibrosis and emphysema are well seen.

POST MORTEM REPORT NO. 3.

EDWIN HUTCHINGS - AGE 64.
77, VARTEG ROAD,
YSTALYFERA.

DEATH - 29.12.32. POST MORTEM - 30.12.32.

CLINICAL HISTORY.

His previous clinical history shewed that he had suffered from cough and shortness of breath for some time, for which he had received frequent treatment. During the last fortnight, he had developed severe attacks of "miners' asthma" and was compelled to take to his bed.

POST MORTEM EXAMINATION.

The lungs were collapsed, deeply pigmented, but only adherent to the chest wall in two places by pleural adhesions which easily broke down under slight pressure. The posterior portions of both lungs were hard and almost stoney to the touch. The apices, bases and anterior margins of both lungs were emphysematous and several large bullae were present. In all lobes in both lungs were varying sized nodules of fibrous tissue, the middle zones being particularly affected. In the right lung, one of these fibrotic areas measured 2½" across, tapering to a point 4" away. Both lungs floated in water and were crepitant, but the fibrotic zones sank on being tested after removal. The pleural membrane cov-

ering the lungs was densely adherent and thickened over the fibrotic areas, the lobes of the lungs being densely adherent to one another.

The heart was slightly enlarged. The pericardiac fluid was normal in amount. Over the right ventricle there were several small areas of thickened pericardium. There was no evidence of organic disease of the valves or aorta, but there was evidence of strain on the right heart.

The liver was normal in size but slightly harder than normal, and presented a nut-meg appearance - due to some venous back pressure from the right heart.

The remaining organs were normal in appearance.

CONCLUSIONS.

1. That both lungs showed a considerable degree of fibrosis.
2. That the remaining areas of the lung were deeply pigmented and were emphysematous.
3. That the condition of the heart and liver showed that there was considerable obstruction to the circulation through the lungs.

MICROSCOPICAL EXAMINATION.

Section No.1.

The pleura shows slight thickening and is infiltrated with round cells. Immediately below the pleura is an area of increased vascularity, the texture of the fibrous tissue being delicate with a fair number of open spaces. Small amounts of coal dust are seen in this area. The remainder of the section is composed

completely of a dense fibrotic tissue, containing large amounts of coal dust, and the almost complete absence of blood vessels is very noticeable. The tissue stains badly and a single, small, necrotic zone, about the size of a millet seed, is seen in the top part of the section. This tissue is characteristic of the condition known as silico-anthracosis.

Silica content of ash - 7.04%.

Section No.2.

Under the low power, the section is closely emphysematous with slight, irregular, pleural thickening. Inter-lobular septa are markedly thickened and loaded with coal dust. The walls of the air sacs in most fields show surprisingly little coal dust, but are irregular by cellular proliferation, the nuclei of the cells being round to oval. At the angles where adjacent air sacs join are collections of dust with a varying degree of thickening, but typical fibro-blast cells are not seen in any number.

Silica content of ash - 17.75%.

Section No.3.

The section shows a zone of comparatively dense fibrosity. The blood vessels show a marked fibrosis, particularly affecting the outer coat. At the edge of the section, the tissue becomes emphysematous and small areas of round-celled infiltration are seen.

Silica content of ash - 21.34%.

Section No.4.

This shows a considerably thickened pleura, beneath which is a small mass of lymphatic tissue. Such lymphatic tissue has been described as being of some pathological importance, but recent work has shown that it is normally found in the lungs of adult people. The whole area is densely fibrotic, with much more cellular definition than that seen in Section No.1.

Silica content of ash - 18.41%.

Section No.5.

The section shows alternating zones of emphysema and nodular fibrosis. The outer parts of the arteries are markedly sclerosed and there is some round-celled infiltration of the bronchi.

Silica content of ash - 16.85%.

Section No.6.

The whole lung tissue in this section is markedly emphysematous and corresponds closely with Section No.2.

Silica content of ash - 14.21%.

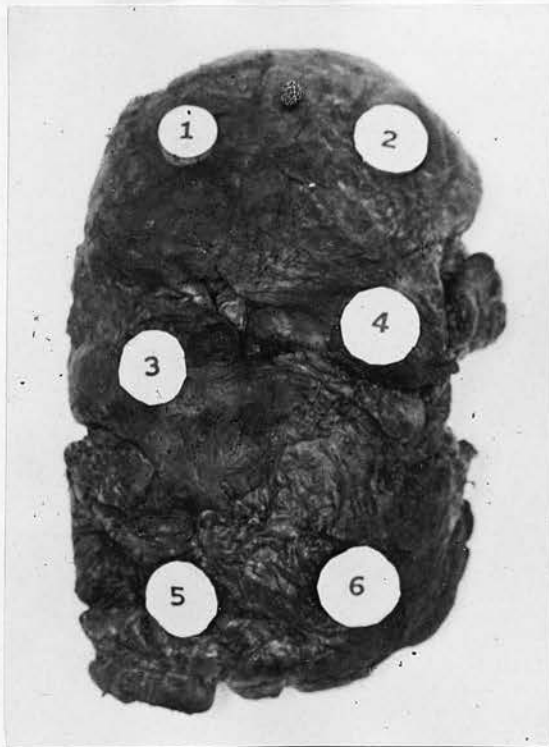
CONCLUSIONS.

The lung is typical of the third stage of silicosis, a conclusion which is confirmed by both microscopical and chemical examination. The functional value of such a lung must have been of a very low order.

X

PHOTOGRAPH NO. 1.

X-Ray photograph of lung after removal from cadaver, showing varying nature of the lung tissue. The dark areas are hard, rubber-like, and fibrotic.

PHOTOGRAPH NO. 2.

Photograph of lung before section showing nature of pleura and the numbered areas from which specimens for examination were taken.

PHOTOGRAPH NO. 3.

Lung after coronal section showing nature of the lung tissue. The areas of consolidation and the areas of fibrosis and emphysema are well seen.

POST MORTEM REPORT NO. 4.

IVOR THOMAS, - AGE 57 YEARS.

TAI GWALIA,

LOWER CWMYRCH.

DEATH - 22/11/32.

POST MORTEM - 23/11/32.

CLINICAL HISTORY.

This man gave a history of having worked for thirty-seven years as a miner in the Anthracite Coal-field. His past clinical record shews that he has suffered from 'Bronchitis' for many years. He had been unemployed since 1930. He was first attended by me on the 15th November, 1932, when he complained of shortness of breath, troublesome cough with copious black expectoration, loss of weight and sleeplessness.

He died on 22/11/32.

POST MORTEM EXAMINATION.

Lungs.

External appearance shows puckering and pleural thickening at the apex. There is no evidence of marked pleural adhesions nor of the shaggy fibrous appearance of recent pneumonia. The inter-lobular section is not adherent. The lung is deeply pigmented, and contains numerous hard leathery nodules, ranging from $\frac{1}{4}$ " to 1" in diameter. These are chiefly in the apical and basal regions. On coronal section the lung is coarsely emphy-

ematous. There is no marked naked eye thickening of the vessels or bronchi. Near the apex is a small triangular area of lung tissue which is almost white in colour. The apical regions and along the inter-lobular system shows marked leathery fibrosis and a smaller fibrous area can be seen below the inter-lobar section in the upper zone of the basal lobe.

Heart.

The heart is slightly enlarged, soft, and shows signs of degenerate changes. There is no evidence of any valvular disease.

Liver.

Shows signs of passive congestion. It was small in size. The other organs were normal in appearance.

MICROSCOPIC AND MACROSPIC EXAMINATIONS OF THE ZONES.

Zone No.1. - Left Lung Apex.

The Pleura over the upper lobe is irregularly thickened and greyish white in colour. There is some puckering of the apex. Microscopically, the area removed for examination varies from that of hard leathery consistency to a small peripheral zone of emphysema. It is intensely black, Microscopically, the major portion of the section shows dense fibrosis with complete obliteration of all normal lung structure and an increase in thickness of the inter-lobar septa. Almost the whole of the area is deeply pigmented with coal and in the small marginal zone of emphysema the alveolar walls are intensely thickened. The blood vessels show considerable

thickening. This is the only zone which sinks in water.

Silica content of ash - 28.15%.

Zone No.2. Apex Lower Lobe.

The pleura shows slight irregular thickening. The lung tissue is spongy and coarsely emphysematous and black in colour. Microscopically, the section shows areas of emphysematous lung, in which the alveolar walls are almost normal and but little pigmented. Several large emphysematous bullae are visible, their walls being pigmented and thickened. The blood vessels show considerable thickening, and the walls of the smaller bronchi are smooth and lined with flattened cells.

Silica content of ash - 15.32%.

Zone No.3. - Upper Lobe Base.

The pleura is almost normal in appearance. The lung tissue is black, crepitant and coarsely emphysematous. Microscopically, the area is coarsely emphysematous with two small areas of fibrosis. The alveolar walls show some thickening and are deeply pigmented.

Silica content of ash - 29.66%.

Zone No.4. - Centre Lower Lobe.

The pleura shows slight, irregular thickening. The lung tissue is crepitant, and coarsely emphysematous. It is deeply pigmented with coal dust. Microscopically, the area is generally coarsely emphysematous with several large bullae with smooth pigmented walls. There are several small areas of fibrosis. The blood vessels show some thickening. The bronchus in this section shows

considerable thickening of its walls.

Silica content of ash - 24.77%.

Zone No. 5. - Lower Lobe Base.

The pleura appears to be normal. The lung tissue is coarsely emphysematous and crepitant. It is black in colour. Microscopically, the section shows considerable emphysema with considerable thickening of the alveolar walls. In several areas these thickened walls have come into contact with one another, probably owing to some degree of alveolar collapse. There are several small peribronchial zones of round-celled infiltration.

Silica content of ash - 25.18%.

Zone No.6. - Lower Lobe Base.

The pleura is normal in appearance. The lung tissue is coarsely emphysematous and crepitant. Microscopically, the section shows considerable emphysema with thickening of the alveolar walls. In several small areas the alveoli contain a clear-staining fibrinous exudate - due to some congestion prior to death. The blood vessels are somewhat dilated.

Silica content of ash - 21.80%.

CONCLUSIONS.

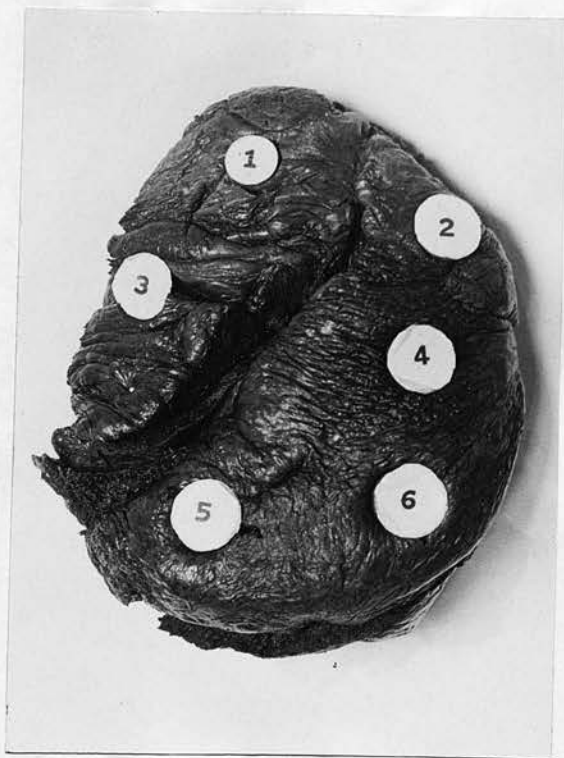
1. That both lungs showed extensive fibrosis - typical of third stage silicosis.
2. That the remaining areas of the lungs were deeply pigmented and emphysematous.
3. That the condition of the heart and liver showed that there had been considerable strain on the myocardium with consequent passive

congestion of the liver.

4. That the functional value of the lungs must have been of a very low order.

PHOTOGRAPH NO.1.

X-Ray photograph of lung after removal from cadaver, showing varying nature of the lung tissue. The dark areas are hard, rubber-like, and fibrotic.



Photograph of Lung before section showing nature of pleura and the numbered areas from which specimens for examination were taken.

PHOTOGRAPH NO. 3.

Lung after coronal section showing nature of the lung tissue. The varying areas of fibrosis and emphysema are well shown.

OWEN T. WILLIAMS,
BRYNEITHYN,
LOWER CWMTWYRCH.

AGE - 51

OCCUPATION - HAULIER.

15/2/33.

Previous Illnesses

Bronchitis every winter
since 1928.Family History with regard
to Tuberculosis

Nil.

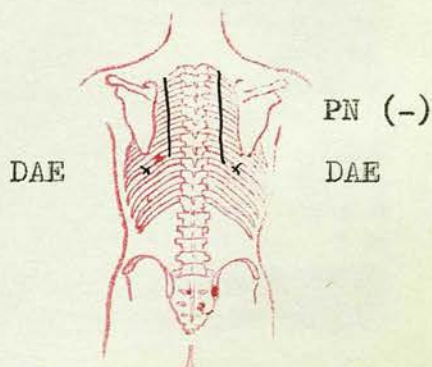
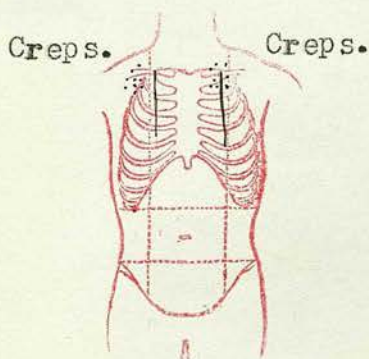
History of Present Illness

Has worked underground for
37 years. During the last
few years has been employed
as haulier. Has not done
any hard heading work.

Present Symptoms

Shortness of breath very
marked on exertion, trouble-
some cough with viscid spu-
tum, pain across the chest.
Lassitude, sleeplessness.
Capacity for work - getting
about.

Clinical Examination

General condition good.
Weight 11 st. 4 lbs. High-
est weight 12 st. 2 lbs.
Sputum negative.
Expiration - 32 ins.
Inspiration - 32½ ins.Chest practically immobile
Percussion note dull over
sub-apical regions. Impair-
ed generally. Auscultation
breath sounds faint, creps
at apices.



3rd
STAGE.

X-RAY

R. & L.

Apices clear. Centre of both lung fields occupied by large irregular cricket-ball masses, characteristic of tertiary silicosis. Scattered coarse mottling above and below opacities. Displacement of aortic arch slightly to the left. Some thickening of right root. Both Diaphragm leaves irregular and peaked. Bilateral consolidation in centre of lung fields extending outwards and downwards. There is the possibility of superimposed tubercle but not suggestive of this.

15. 2. 33 Troublesome cough; dyspnoea marked - Mixt. Expect.
19. 2. 33 Very dyspnoeic. Ol morrhue 3ⁱⁱ T.D.S.
23. 2. 33 Ol morrhue, with mixt. expect.
28. 2. 33 Dyspnoea still marked on exertion. Cough better.
7. 3. 33 Ol morrhue.
14. 3. 33 Dyspnoea - no improvement. Cough troublesome at night.
21. 3. 33 Very little sputum. Cough dry, short, worse at night.
28. 3. 33 Ol morrhue 3ⁱⁱ T.D.S.
6. 4. 33 " " " "
14. 4. 33 Weight improving, now 11 st. 6 lbs.
21. 4. 33 Dyspnoea still marked on exertion.
28. 4. 33 Able to do light work in garden.
7. 5. 33 Ol morrhue.
14. 5. 33 " "
21. 5. 33 " "
28. 5. 33 Looks better. No complaint as long as resting.
7. 6. 33 Cough much better. Ol morrhue 3ⁱⁱ T.D.S.
14. 6. 33 No improvement in dyspnoea.
21. 6. 33 Ol morrhue 3ⁱⁱ T.D.S.
28. 6. 33 " " " "
-

JOHN EDWARDS,
CLARE ROAD,
YSTALYFERA.

AGE - 54

OCCUPATION - MINER.

Previous Illnesses Influenza Aug. 1918. Bronchial Catarrh Sept. 1930.

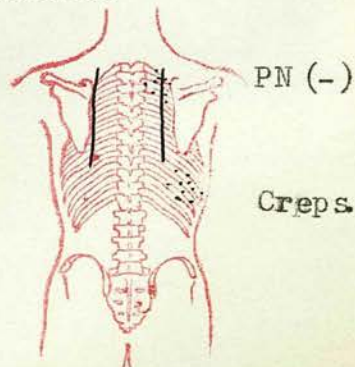
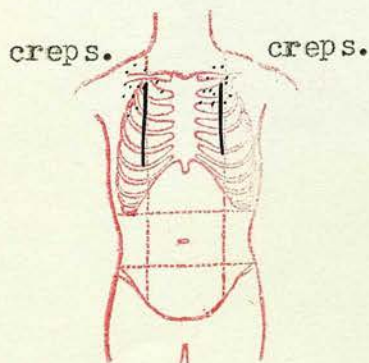
Family History with regard to Tuberculosis Nil.

History of Present Illness. Has worked underground for 30 years as Labourer, and Coal hewer. Used the hand-boring machine daily. Has noticed that unable to fill as many trams as usual. Gets tired quicker. Shortness of breath when climbing hills, stairs, etc.

Present Symptoms Shortness of breath, cough with copious slatey grey sputum, loss of flesh, lassitude, pain across the chest sleeplessness and loss of appetite.

Capacity for work - getting about.

Clinical Examination Rather wasted. Expiration - 28 ins. Inspiration - 29 ins. Sputum negative. Weight - 9 st. 10 lbs. Highest - 11 st. 3 lbs. Movements of chest restricted. Percussion note dull over both apices and impaired generally. Breath sounds weak with some harshness at end of inspiratory phase. Crepitations over both apices and at bases. Chest emphysematous.



JOHN EDWARDS.

3rd
STAGE



X-RAY R. & L. Long narrow chest, with typical long narrow phthinoid heart shadow; Coarse mottling throughout both lung fields with evidence of bilateral fibrosis. Diaphragm leaves flattened and peaked on right side.

- Jan. 6. 33 Dyspnoea and cough marked. Mixt. Expect.
and ol morrhue.
- " 10. 33 Sputum negative.
- " 12. 33 Ol morrhue 3ⁱⁱ T.D.S.
- " 15. 33 Weight - 9 st. 6 lbs. Sleeplessness and
loss of appetite.
- " 21. 33 Dyspnoea marked on slightest exertion.
- " 28. 33 Ol morrhue 3ⁱⁱ T.D.S.
- Feb. 4. 33 No improvement in condition.
- " 11. 33 Temperature 98.4 F. Pulse 84 at rest.
- " 18. 33 Cough with copious sputum - mixt. Expect.
- " 25. 33 Ol morrhue 3ⁱⁱ T.D.S.
- Mar. 12. 33 " " " "
- " 19. 33 " " " "
- " 27. 33 Influenza, seen daily, recovery slow,
very weak. Easton Syrup.
- Apl. 18. 33 Myocardial condition poor. Pulse 110.
- " 26. 33 Tr. Digitalis mxv. T.D.S.
- May 10. 33 Myocarditis not improving. Dyspnoea
great.
- " 18. 33 Oedema of feet commencing. Pil Digitalis
Co. $\bar{\text{f}}$ T.D.S.
- " 30. 33 No improvement.
- June 14. 33 Liq. Strych and Tr. Digitalis - no effect
- " 28. 33 Died.
-

ESIAH MORGAN,
2, TANYWERN LANE, AGE - 52
YSTALYFERA.

OCCUPATION - MINER.

Previous Illnesses Nystagmus for three years

Family History with regard
to Tuberculosis Nil.

History of present Illness
Off work on account of nys-
tagmus for three years. Has
complained of shortness of
breath for five years es-
pecially on working. Has
been confined to bed for
seven days with hæmorrhage
from chest and black sput-
um.

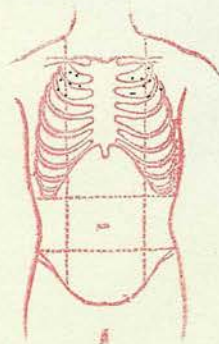
Present Symptoms Cough with blood-stained
sputum. Sputum scanty, app-
etite poor, lassitude, loss
of flesh, sleeplessness,
tightness across the chest
and shortness of breath.

Weight - 10 st. Highest -
11 st. 4 lbs.

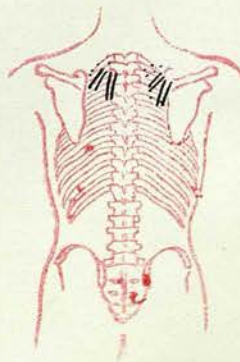
Inspiration $30\frac{1}{2}$ ins.
Expiration 29 ins.

Clinical Examination Muscular development poor,
flat chest, wavy cardiac im-
pulse; restricted expansion;
looks ill; cough with black
sputum; pulse 96; sputum
negative.

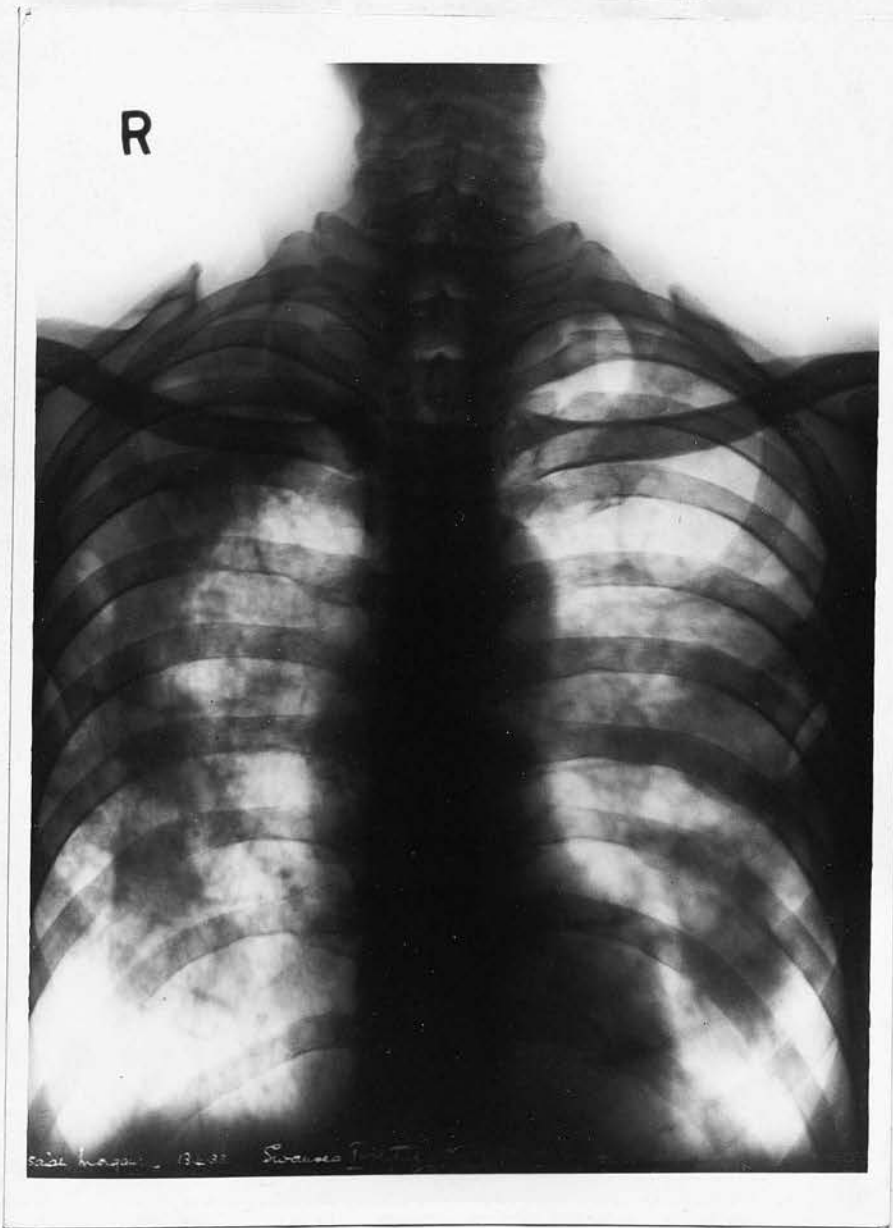
Poor percussion note over
both upper Zones.



Harsh breath
sounds with
crepitations



THIRD STAGE PLUS TUBERCULOSIS.



X-Ray Broad mediastinum; cardiac outline is irregular and ill-defined. Diaphragm not in picture except part of R. dome which is irregular. Irregular dense opacities throughout both lung fields more marked on the right.

3. 3.33 Confined to bed, blood stained sputum; seen daily.
01 morrhue 3i t.i.D.
4. 5.33 Able to get about the house. Very weak, emaciated and dyspnoeic. Cough very troublesome, sputum practically nil. Still on 01 morrhue.
15. 5.33 Cough and dyspnoea very troublesome.
20. 5.33 " " " " "
27. 5.33 Appetite poor. Syr Eastonii.
4. 6.33 Syr Easton.
14. 6.33 Cough improved. Dyspnoea not improved.
24. 6.33 01 morrhue 3ii T.I.D.

Medical Examination

JAMES THOMAS,
28 CLEES LANE,
YSTALYFERA.

AGE - 43

OCCUPATION - COLLIER.

Previous Illnesses Nil.

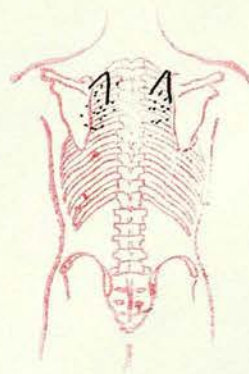
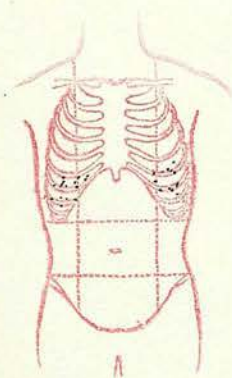
Family History Wife died from carcinoma of breast.

History of Present Illness Dyspnoea $1\frac{1}{2}$ years ago. Worked 30 years underground. Many years on hard headings.

Present Symptoms Frequent cough with scanty sputum. Appetite very poor, loss of flesh, confined to bed. General condition very poor.

Clinical Examination ... Very rigid chest. Bilateral signs. Pulse 100, sputum negative.

Expiration 29 ins.
Inspiration 30 ins.





3rd
STAGE
PLUS
TUBER-
CULOS-
IS.

- X-Ray Ossification of costal cartilages.
- R. Dense fibrosis of apex upper half of lung. Excavation in immediate apex; scattered fibrosis in lower half. Diaphragm completely irregular and peaked.
- L. Dense fibrosis of apex and upper half of lung, especially in peripheral region. Fibrosis also present in lower half. Diaphragm irregular and peaked in outline. Heart outline not well defined. Extensive bilateral fibrosis. Excavation in both apices. Probably silicotic lesion with appearances suggestive of superimposed tubercle.

30. 6. 32 Confined to bed. Temp. 99. Pulse 100. Seen daily. Copious black sputum. Intense dyspnoea. Cough eased with pil ipecac cum scillae. Ol morrhue 3 \bar{v} T.I.D. Wasting marked.
- No improvement. Gradual deterioration.
4. 7. 32 Seen daily.
12. 8. 32 Myocardial degeneration. Oedema of feet and ankles.
- Tr. Digitalis mxv. T.I.D.
10. 9. 32 Myocardium improved. Disappearance of oedema.
- 16.11. 32 Copious black sputum. Dyspnoea great.
- 20.12. 32 Heart failing. Tr Digitalis mxx T.I.D.
14. 1. 33 Cough eased by pil ipecac cum scillae.
16. 2. 33 Oedema of legs.
18. 3. 33 Compensation completely broken down.
12. 4. 33 Tr. Digitalis full doses - no result.
12. 5. 33 Died.

JOHN HUTCHINGS,
28 PENYWERN ROAD, AGE - 59
YSTALYFERA.

OCCUPATION - MINER.

Previous Illnesses Nil.

Family History with regard
to Tuberculosis Nil.

History of present illness First symptom noticed -
shortness of breath and weak-
ness. Has lost during the
last twelve months weeks of
work owing to weakness and
shortness of breath. He is
coughing up a lot of phlegm.
Has worked underground for
40 years, 16 years of which
he has worked as a repair-
er.

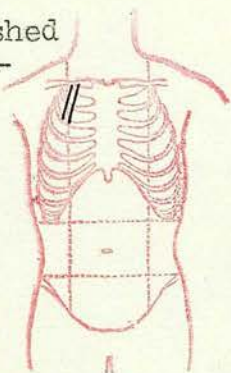
Present Symptoms Paroxysmal cough with scanty
sputum. Appetite very
poor, lassitude and loss
of flesh, tightness of chest
sleeplessness.

Capacity for work - getting
about. Weight - 8 st. 8 lbs.
General condition poor. Black
sputum. Three years' ago
able bodied man, now very
frail.

Clinical Examination Sputum negative.
Inspiration $32\frac{1}{2}$ ins.
Expiration $30\frac{3}{4}$ ins.

Diminished
air en-
try.

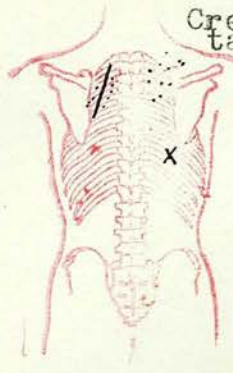
Feeble
breath
sounds



Pulse 70

Crepiti-
tations

Im-
paired
Per-
cussion
note.



JOHN HUTCHINGS.



3rd
STAGE

X-Ray R.

Scattered coarse mottling in lung fields more dense in apex and sub-clavicular region. Diaphragm flattened, costo phrenic angle obliterated.

L.

Scattered coarse mottling through lung field. Heart apparently to the right. Left diaphragm outline not well defined. There is evidence of bilateral fibrosis, especially on the Right, and the appearance of the Right upper zone is compatible with the super-imposition of Tubercle.

29. 6. 32 Complained of pain in side; pleural rub. d. morrhue.
4. 7. 32 Ol morrhue 3̄ii T.I.D. Pain better.
18. 7. 32 Ol morrhue. Dyspnoea marked, cough with sputum.
26. 7. 32 Expect. mixture. Still dyspnoeic.
4. 8. 32 Mist. expect.
2. 9. 32 Slight improvement, cough better and able to be about.
15. 9. 32 Ol morrhue 3̄ii T.I.D.
- 1.10. 32 " " " "
- 24.10. 32 " " " "
- 13.11. 32 Slight improvement in weight + 2 lbs.
- 25.11. 32 Sputum examined by concentration test - negative.
- 12.12. 32 Ol morrhue.
- 30.12. 32 " "
16. 1. 33 Paroxysmal cough R Vin Ipecac 3̄ii
Tr camph Co 3̄vi } ss h h^{ly}
Ag. CHCl₃ ad 3̄8 }
24. 1. 33 " " " "
14. 2. 33 Ol morrhue 3̄ii I.D.S.
21. 2. 33 " " " "
3. 3. 33 Slight improvement still continues. Dyspnoea + +
17. 3. 33 Cough again troublesome. Tr camph Co.
2. 4. 33 Dyspnoea and cough. Ol morrhue and Mist Expect.
12. 4. 33 " " " " " " " "
9. 5. 33 Sent away to Convalescent Home.
16. 5. 33 Away at Convalescent Home
30. 6. 33 Ol morrhue, mane et nocte.
6. 7. 33 Ol morrhue.

ZORABABEL JONES,
61 ALLTYGRUG ROAD, AGE - 52 OCCUPATION - MINER.
YSTALYFERA.

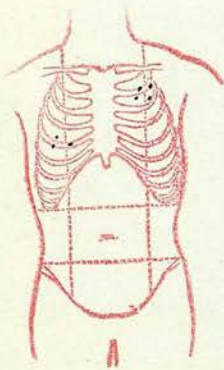
Previous Illnesses Bronchitis 1922.

Family History affected ..
with Tuberculosis Nil.

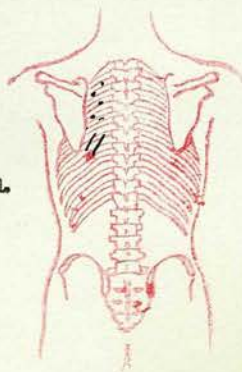
History of Present Illness Has worked underground for
39 years. Bronchitis from
April to May.

Present Symptoms Cough with blood - stained
copious sputum, shortness of
breath, poor appetite, loss
of flesh, sleeplessness.

Clinical Examination Inspiration $31\frac{1}{2}$ ins.
Expiration 30 ins.
Impaired percussion note,
harsh respiration with crep-
itations at apices. Sputum
negative.



Crepita-
tions.
Harsh
Respiration.



ZORABABEL JONES.



3rd
STAGE.

- X-RAY R. Rather coarse mottling throughout lung fields, fairly dense in middle half. Diaphragm regular, with a few adhesions in inner one-third.
- L. Coarse mottling throughout lung fields. Diaphragm regular. General appearances suggest silicosis with superimposed tuberculosis.

13. 5. 31 Seen by T.B. physician at Welsh National Memorial Clinic. Refused to go to Sanatorium.
15. 5. 31 Expect. mixture.
7. 6. 31 Sent to Miners' Convalescent Home at Talygarn.
14. 7. 31 Slightly improved. 9 st. 10 lbs. + 3 lbs.
21. 8. 31 Attack of Bronchitis. Seen daily for two weeks.
4. 9. 31 O1 morrhue. Still very dyspnoeic - able to be about.
- 10.10. 31 O1 morrhue. 3ⁱⁱ mane et nocte.
- 21.11. 31 Much the same.
- 4.12. 31 Bronchitis - seen daily for seven days.
10. 1. 32 Not improving - dyspnoea more marked, still about.
10. 2. 32 Losing weight - 9 st. 6 lbs. O1 morrhue.
10. 3. 32 Deteriorating. O1 morrhue and Tr digitalis mvii.
4. 4. 32 Cough and copious black sputum.
10. 5. 32 Still able to be about but not improving.
12. 6. 32 Cough and dyspnoea marked.
4. 7. 32 Much the same.
6. 8. 32 Developed a sore on end of penis - cancerous.
14. 9. 32 Advised admission to Swansea Hospital - refused.
- 21.10. 32 Inguinal glands involved. Confined to bed.
- 16.11. 32) Seen daily. Inguinal glands suppurating -
 12.12. 32) refused operation. Chest signs of definite tuberculosis. Copious black sputum.
1. 1. 33) Attacks of severe pain in left side. Very
 6. 2. 33) dyspnoeic. Signs of heart failure - oedema of ankles, scrotum and abdomen.
14. 3. 33)
 21. 4. 33)
 8. 5. 33)
2. 6. 33 Died.

WILLIAM THOMAS WATKINS,
6 TIRBACH ROAD, AGE - 40
YSTALYFERA.

OCCUPATION - MINER.

Previous Illnesses Nil.

Family History of Tuberculosis Nil.

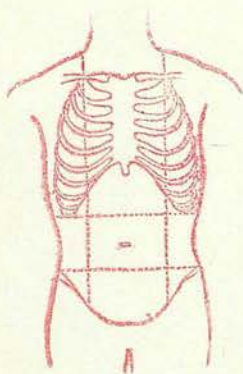
History of present Illness Has worked underground for 36 years. No hard headings Boring with machine daily occurrence - now complains of shortness of breath, cough and sputum.

Present Symptoms Frequent cough with scanty sputum. Appetite poor, lassitude, loss of flesh, shortness of breath, pain in left side of chest.

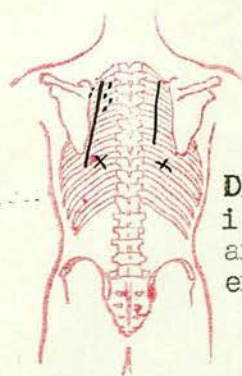
Capacity for work - getting about.

Clinical Examination ... Weight 8 st. 3 lbs. General impairment of note, harsh breath sounds. Diminished air entry at bases. Emphysema and catarrhal signs.

Sputum negative.
Inspiration 32½ ins.
Expiration 31 ins.

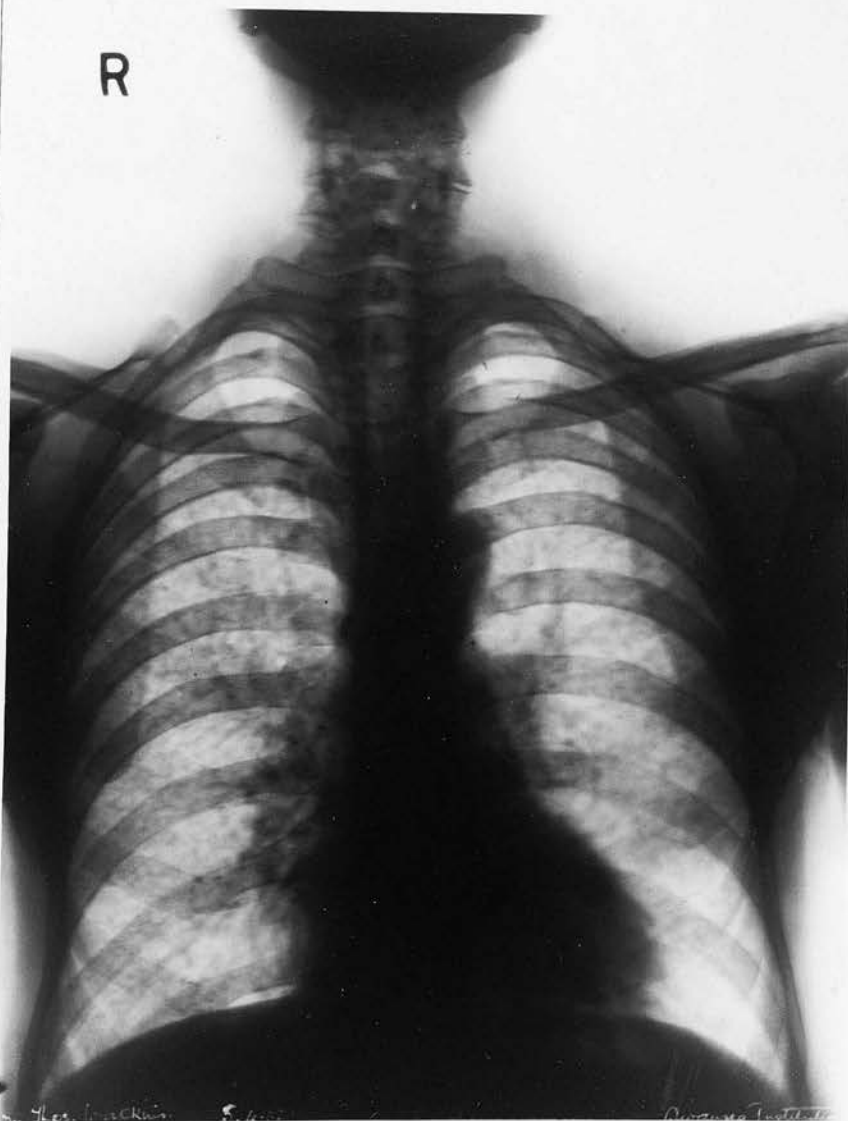


Diminished
air entry.



Dimin-
ished
air
entry.

WILLIAM THOMAS WATKINS.



2nd
STAGE

X-RAY R. & L.

Coarse mottling throughout both lung fields, no evidence of superimposed tubercle in the lesion present which is probably silicosis.

May 24. 33 Cough, dyspnoea and pain in side. Ol morrhue.
May 30. 33 Ol morrhue.
June 2. 33 Dyspnoea - no cough.
" 12. 33 Sputum negative.
" 18. 33 Ol morrhue.

DAVID T. JONES,
32, PENYWERN ROAD, AGE- 59
YSTALYFERA.

OCCUPATION - MINER.

Previous Illnesses Occasional colds.

Family History with regard to Tuberculosis Daughter aged 23, has been certified since examination.

History of Present Illness
Has worked 45 years underground, during which time he has done much boring. He gives no history of having worked in hard headings. He now complains of cough with copious sputum, shortness of breath. There is a history of occasional haemoptosis during the first week of illness.

Present Symptoms Frequent cough with blood-stained sputum. The sputum is copious. Appetite poor, lassitude, loss of flesh, tightness across the chest, and marked shortness of breath, also night sweats occasionally.

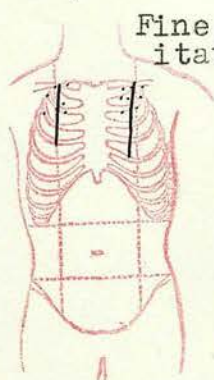
Capacity for work - able to get about.

Clinical Examination General condition fair.

Weight 8 st. 7 lbs.

Expiration 29 ins.
Inspiration 31 ins.

Sputum - negative to tubercle bacilli.

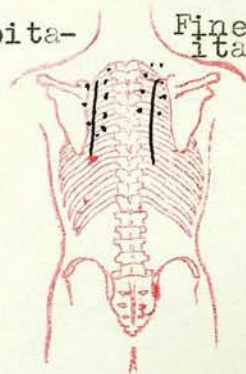


Fine Crepitations.

Weak breath sounds generally.

Some rhonci.
poor expansion.

Fine Crepitations.



Fine Crepitations.

DAVID T. JONES.



3rd
STAGE
PLUS
TUBER-
CULOS-
IS.

- X-RAY Examination
- R. Apical pleural thickening medium Coarse mottling throughout lung field. Dense upper half of peripheral region. Diaphragm flattened, costo-phrenic angle obliterated.
- L. Pleural thickening in apex. Scattered medium coarse mottling in lung fields, most dense in upper third. Diaphragm outline irregular, wide mediastinal shadow. Evidence of bilateral fibrosis, silicotic in origin. Appearance of left upper zone is compatible with T.B. infection.

27. 4. 32 Very dyspnoeic - troublesome cough.
4. 5. 32 Ol morrhue 3 \bar{ii} T.I.D. Tr camph co.p.r.n.
14. 5. 32 Ol morrhue " T.I.D.
6. 6. 32 " " " "
13. 6. 32 " " " "
29. 6. 32 " " " "
12. 7. 32 General condition improved. Weight 2 $\frac{1}{2}$ lbs. gained, cough not so troublesome. Dyspnoea still marked on exertion.
19. 7. 32 Ol morrhue 3 \bar{ii} T.I.D.
28. 7. 32 " " " "
4. 8. 32 General condition still improving - no cough no sputum.
15. 8. 32 Ol morrhue 3 \bar{ii} T.I.D.
23. 8. 32 " " " "
4. 9. 32 Dyspnoea still on exertion.
12. 9. 32 " " " "
19. 9. 32 Ol morrhue.
30. 9. 32 Influenza, seen daily, myocarditis. Diaphoretic mxt.† Tr Digitalis mx. T.I.D. Syr Easton and ol morrhue for post influenzal debility.
- 16.10. 32 Syr Easton ol morrhue.
- 12.11. 32 Ol morrhue 3 \bar{ii} T.I.D.
- 20.11. 32 " " " "
- 7.12. 32 Slight cough. Dyspnoea main symptom.
- 15.12. 32 Ol morrhue 3 \bar{ii} T.I.D.
- 30.12. 32 " " " "
6. 1. 33 Improving again, except for dyspnoea.
24. 1. 33 Ol morrhue 3 \bar{ii} T.I.D.
18. 2. 33 " " " T.I.D.

28. 2. 33 01 morrhue 3ⁱⁱ T.I.D.
17. 3. 33 No cough, no sputum, only Dyspnoea †
24. 3. 33 01 morrhue.
24. 4. 33 Cardiac condition good.
14. 5. 33 01 morrhue 3ⁱⁱ T.I.D.
12. 6. 33 " " " "
16. 7. 33 General condition good.

ALBERT EDWARD GRABHAM,
83 ALLTYGRUG ROAD, AGE - 56
YSTALYFERA.

OCCUPATION - MINER FOR
42 YEARS, INCLUDING BOR-
ING, REPAIRING, ETC.

Previous Illnesses Nil.

Family History with regard .
to Tuberculosis Nil.

History of Present Illness . First symptom noticed was
shortness of breath and
cough. Has done a lot of
boring with machines.

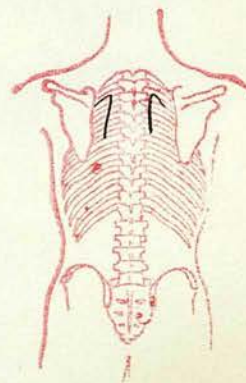
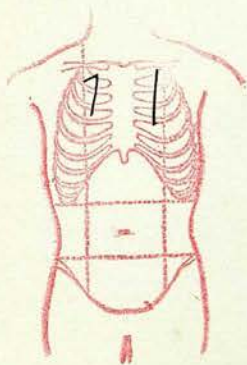
Present Symptoms Cough with copious muc-
ous sputum, shortness of
breath, sleeplessness ,
poor appetite.

Capacity for work - gett-
ing about.

Clinical Examination Usual weight 10 st. now
9 st. 10 lbs. Sputum
negative.

Expiration 32 ins.
Inspiration 33½ ins.

Nil definite to be made
out in chest.



ALBERT EDWARD GRABHAM.

P



2nd
to
3rd
STAGE.

X-RAY Examination - R. & L.

Coarse mottling throughout both lung fields, evidence of bilateral fibrosis.

Appearance not suggestive of superimposed tubercle.

27. 12. 32 Ammon. carb. grs. xxx. Vin Ipecac 3ⁱⁱ Tr.
 camph Co. 3ⁱⁱ. Ag. ad 3 8. 3 sse. 4 hrs
8. 1. 33 Dyspnoea, able to do light work in green-
 house.
15. 1. 33 Ol morrhue, cough troublesome.
12. 2. 33 Condition not improving.
24. 2. 33 Losing weight. Ol morrhue.
6. 3. 33 Miners' Convalescent Home - Talygarn.
17. 4. 33 Weight - gained 1½ lbs. Dyspnoea no im-
 provement.
26. 4. 33 Cough troublesome at night.
16. 5. 33 Still dyspnoeic at slightest exertion.
15. 6. 33 No improvement.
30. 6. 33 Sputum negative (concentration test).

ALFRED THOMAS,
69 ALLTYGRUG RD. AGE - 52
YSTALYFERA.

OCCUPATION - COLLIER.

Previous Illnesses

Influenza in 1928.

Family History affected ..
with Tuberculosis

Nil.

History of present illness

Has worked for 40 years underground, many years used boring machine, colliery very hot and dry atmosphere. Has had a cough for 3 or 4 years accompanied by sputum. Complains of shortness of breath and lassitude.

Present Symptoms

Frequent cough with copious sputum, appetite poor, lassitude, loss of flesh, shortness of breath and night sweats.

Clinical Examination

Scattered rales and rhonchi. R. & L. Lungs and particularly at bases. Breath sounds weak, percussion note impaired greatly, general condition poor, clubbing of fingers.

Expiration 31 ins.

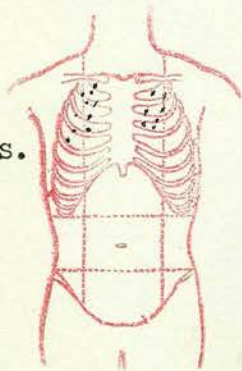
Inspiration 32 ins.

Sputum - 23/6/31 - negative

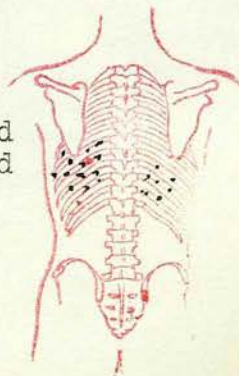
24/7/31 - do.

28/8/31 - positive

Creps.



Scattered rales and rhonchi.





3rd
STAGE
PLUS
TUBER-
CULOSIS.

X-RAY Examination - R. Scattered coarse mottling of apex and throughout lung fields with oval-shaped dense opacities in sub-clavicular region and mid Zone. Diaphragm irregular and peaked. Marked Fibrosis.

L. Scattered coarse mottling throughout lung fields in upper and lower lobes. Diaphragm irregular and peaked. Mediastinum to right. Definite evidence of silicosis with pulmonary tuberculosis superadded.

Feb. 6. 31 01 morrhue. Very dyspnoeic and wasted.

" 20. 31 Mist. expect. Cough rather troublesome

Mar. 1. 31 Cough troublesome, muco purulent and black sputum.

" 15. 31 01 morrhue 3ⁱⁱ T.I.D.

Apl. 4. 31 Looks very ill and wasted.

Apl. 30. 31 01 morrhue 3ⁱⁱ T.I.D.

May 12. 31 Losing weight.

May 24. 31 01 morrhue.

June 30. 31 Sputum negative to tubercle.

July 24. 31 " still " " "

Aug. 25. 31 " positive " "

Sept. 10. 31 01 morrhue 3ⁱⁱ T.I.D.

Oct. 18. 31 " " " "

Nov. 17. 31 " " " "

Dec. 21. 31 Still losing weight.

Jan. 4. 32 2 Pts. of milk extra per diem as extra nourishment from Public Assistance Committee.

Feb. 20. 32 Resting in bed.

Mar. 30. 32 Very dyspnoeic and cough with black and mucopurulent sputum.
Mist. expect.

Apl. 16. 32 01 morrhue.

May 17. 32 Slight haemoptosis. Rest in bed and seen daily.

June 18. 32 Losing weight, very dyspnoeic.

July 21. 32 Kept in bed - rest - 01 morrhue.

Oct. 21. 32 01 morrhue.

Nov. 18. 32 " "

Dec. 4. 32 No improvement.

Jan. 3. 33	01 morrhue	3 ⁱⁱ	T.I.D.
Feb. 28. 33	" "	" "	" "
Mar. 17. 33	" "	" "	" "
Apl. 4. 33	Very ill, sputum blood-stained, seen daily.		
May 30. 33	01 morrhue	3 ⁱⁱ	T.I.D.
June 16. 33	01 morrhue	" "	" "

WILLIAM DAVIES,
117 GRAIG ROAD, AGE - 60 OCCUPATION - MINER.
GODRERGRAIG.

Previous Illnesses Nil.

History of Family with re-
gard to Tuberculosis Nil.

History of Present Illness. Has worked underground for
46 years. No hard head-
ings; boring in coal ev-
ery day. Complains of dys-
pnoea - 1 year. Cough
and black sputum. Temp.
normal.

Present Symptoms Frequent cough and copious
black sputum. Appetite
fair, lassitude, loss of
flesh, tightness across
the chest and shortness of
breath, sleeplessness.

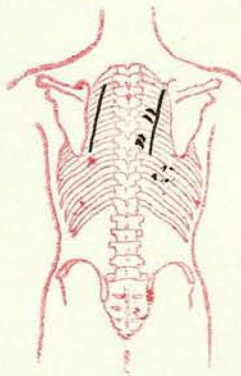
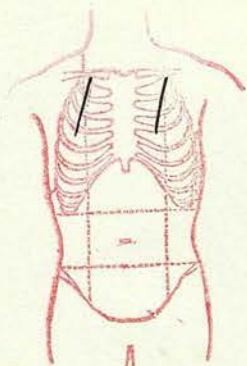
Capacity for work - getting
about.

Clinical Examination Generally impaired note
phthinoid type. Weak breath
sounds posteriorly. Pleu-
ral crepitations.

Expiration - 32 ins.
Inspiration - $33\frac{1}{2}$ ins.

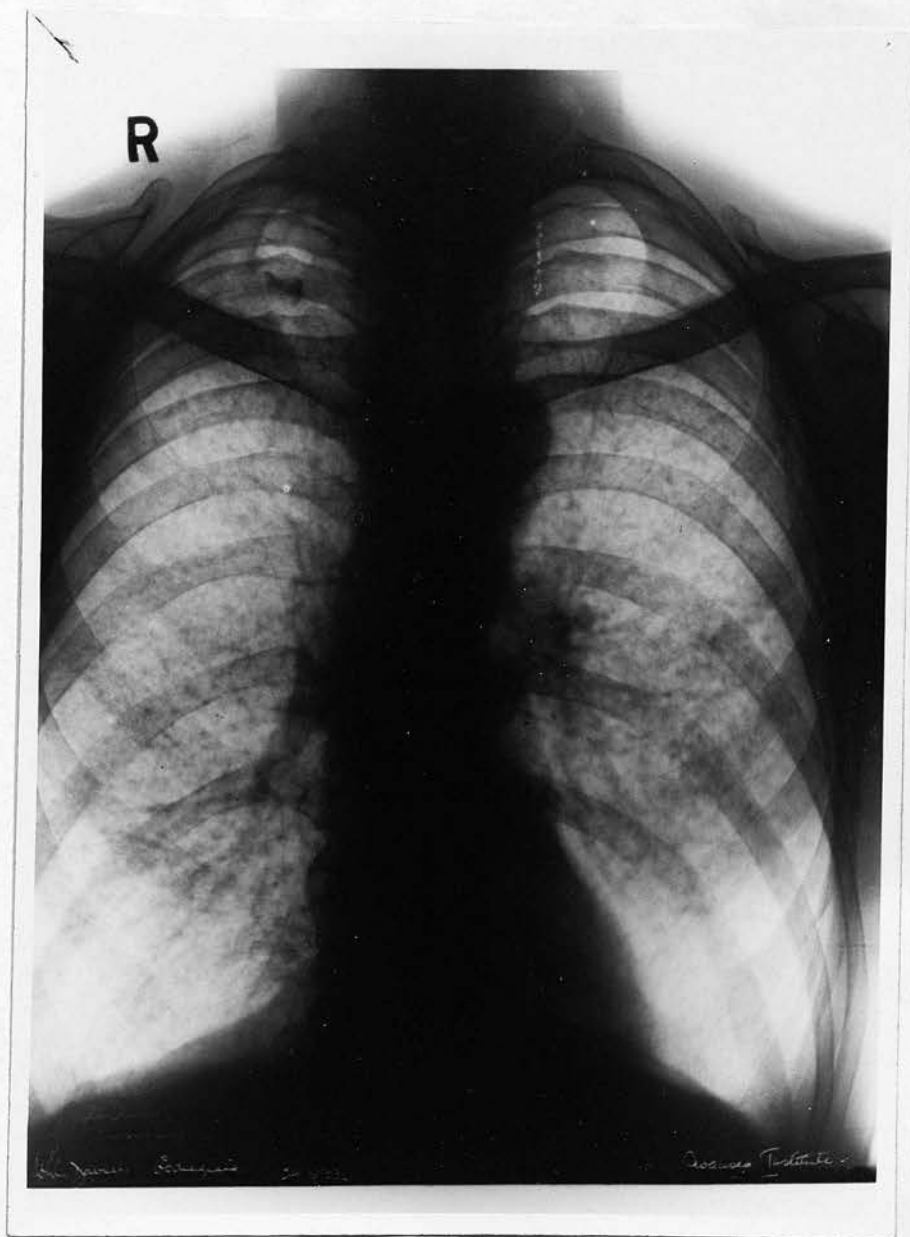
Sputum - negative.

Weak
Breath
sounds.



WILLIAM DAVIES.

2nd
STAGE



X-RAY Examination ... Scoliosis of spine. Coarse mottling throughout both lung fields, bilateral fibrosis. Silicosis no evidence of super-imposed tubercle.

Apl. 20. 33 0l morrhue. Wasting. Dyspnoea marked.
 " 25. 33 Mist. Expect. for cough - very trouble-
 some - sputum black and mucopurulent.
 May 1. 33 Very dyspnoeic.
 June 2. 33 0l morrhue 3 \bar{u} T.I.D.
 June 27. 33 " " " "

History of Present Illness - The patient has a history of

Present Symptoms -

Clinical Examination -

Height - 5 ft. 10 in. Im-
 paired respiration rate at
 rest, more breath sounds
 in right lung.
 Temperature - 98.6
 Pulse - 92
 Blood - Negative

LLEWELLYN JONES,
32 PENYWERN ROAD, AGE - 52 OCCUPATION - MINER.
YSTALYFERA.

Previous Illnesses Nil.

Family History with regard to Tuberculosis Daughter died in 1917 aged 14. Boy aged 11 tubercular.

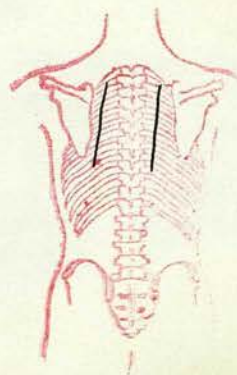
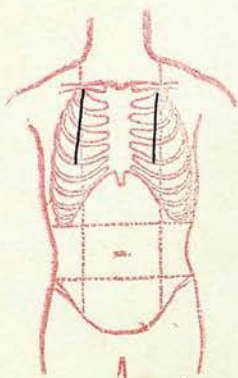
History of Present Illness Has worked as a miner for 40 years, six weeks only on hard headings. Very troublesome cough for many years. Headache, shortness of breath and cough.

Present Symptoms Cough with copious purulent black sputum, appetite poor, lassitude, loss of flesh, shortness of breath, and tightness across the chest.

Clinical Examination ... Weight - 9 st. 10 lbs. Impaired percussion note at apices, weak breath sounds, no adventitious sounds.

Expiration - 30 ins.
Inspiration - 31½ ins.

Sputum - negative.



LLEWELLYN JONES

3rd
STAGE
PLUS
TUBER-
CULOSIS



- X-Ray Examination
- R. Dense shadows occupying apex and upper half of lung fields, especially peripherally down to inter-lobular septum. Fairly dense scattered and coarse mottling in rest of lung fields. Diaphragm regular.
- L. Scattered apical opacities. Peripheral dense shadows occupying two-thirds of lung fields. Scattered mottling - coarse - in most of lung fields. Wide mediastinal shadow. Silicosis superimposed tuberculosis.

1. 5. 31. 01 morrhue 3ⁱⁱ T.I.D.

June 12.31. 2 pts. of milk extra per diem from Public Assistance Committee.

July 4.31 01 morrhue 3ⁱⁱ mane et nocte.

Aug. 8.31 " " " " " "

Sept. 4.31 Expect. mixt. 01 morrhue.

Oct. 3.31 Very little improvement.

Nov. 2.31 Loss of weight 1½ lbs. Still having two pts. of milk per diem.

Dec. 12.31 Influenza, daily visits. Syr Eastonii.

Jan. 14.32 Syr Eastonii.

Feb. 17.32 Improving once again.

Mar. 19.32 01 morrhue 3ⁱⁱ mane et nocte.

Apl. 8.32 " " " " " "

May. 6.32 " " " " " "

June 10.32 " " " " " "

July 15.32 " " " " " "

Aug. 21.32 " " " " " "

Sep. 4.32 " " " " " "

Oct. 23.32 " " Weight much the same - 9 st. 10½ lbs.

Nov. 17.32 01 morrhue 3ⁱⁱ mane et nocte.

Dec. 19.32 " " " " " "

Jan. 1.33 " " " " " "

Feb. 6.33 " " " " " "

Mar. 15.33 " " " " " "

Apl. 17.33 " " " " " "

May. 15.33 " " " " " "

June 16.33 " " " " " "

July 1.33 " " Weight 9 st. 10 lbs.

THOMAS MORGAN,
17, WERN ROAD,
YSTALYFERA.

AGE - 38

OCCUPATION - MINER.

Previous Illnesses Nil.

Family History with regard
to Tuberculosis Nil.

History of Present Illness Has worked 24 years under-
ground, 3 years of which he
worked on hard headings. En-
joyed good health up to 4
or 5 years ago, now com-
plains of shortness of breath.

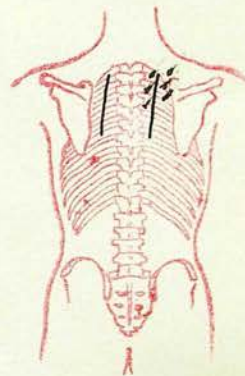
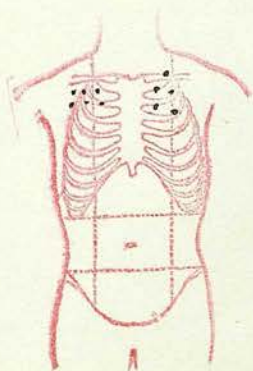
Present Symptoms Cough, lassitude, and short-
ness of breath.

Clinical Examination Weight 9 st. 5 lbs. Poor
percussion note in both
apices, especially left t.
Crepitations in apices.

Expiration $29\frac{1}{8}$ ins.
Inspiration $30\frac{3}{4}$ ins.

SPUTUM - Negative 16/1/32

Positive 28/1/32





3rd
STAGE
PLUS
TUBER-
CULOS-
IS.

- X-RAY Examination
- R. Coarse dense mottling upper half of lung fields. Diaphragm flattened and peaked.
- L. Appearances similar to those in Right Lung, small narrow heart. Appearances suggest a bilateral tuberculosis with silicosis.

16.	1.	32	Sputum negative. Dyspnoea and catarrhal signs in chest.
25.	1.	32	Sputum positive.
Feb.14.		32	Catarrhal signs in chest. Mist. expect.
"	28.	32	Ol morrhue. General condition good.
Mar.14.		32	Mist. Expect.
Mar.21.		32	Ol morrhue. Extra milk ordered - 2 pts. per diem.
Apl.	6.	32	Mist. Expect.
"	20.	32	Ol morrhue 3 ⁱⁱ T.I.D.
May	3.	32	" " " "
May	26.	32	" " " "
June	14.	32	Ol morrhue. Dyspnoea marked and cough troublesome.
June	30.	32	Weight improving, gained 2 lbs.
July	4.	32	Ol morrhue.
"	18.	32	" "
Aug.	7.	32	Condition much the same.
Aug.	30.	32	Ol morrhue. Mist. expect.
Sep.	12.	32	Refused to go to Sanatorium.
Sep.	16.	32	Ol morrhue.
Oct.	14.	32	" " 3 ⁱⁱ T.I.D.
Oct.	30.	32	Mist. Expect.
Nov.	7.	32	Syr Eastonii.
Nov.	28.	32	Ol morrhue.
Dec.	16.	32	Weight still the same.
Dec.	31.	32	Ol morrhue.
Jan.	4.	33	Dyspnoea no change, cough not so troublesome, looks better.
Jan.	18.	33	Ol morrhue.

Feb. 12. 33 Mist. Expect.
" 20. 33 01 morrhue.
Mar. 7. 33 " "
Mar. 18. 33 " "
Apl. 20. 33 General condition improving.
May 17. 33 Sent away to Convalescent Home for ten
days.
May 30. 33 Weight gained $1\frac{1}{2}$ lbs. Still dyspnoeic.
June 6. 33 01 morrhue 3 $\ddot{\text{ii}}$ T.I.D.

WILLIAM JENKINS,
28 CLEES LANE, AGE - 42 OCCUPATION - MINER.
YSTALYFERA.

Previous Illnesses Pleurisy 1919, Bronchitis,
1924. Influenza.

Family History with regard
to Tuberculosis Nil.

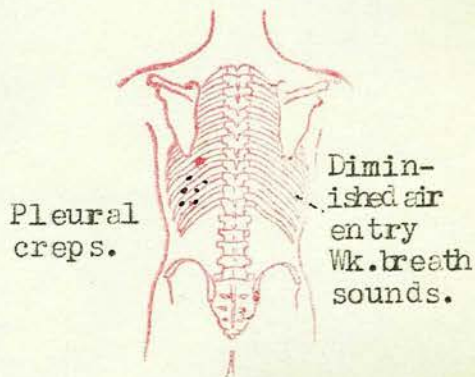
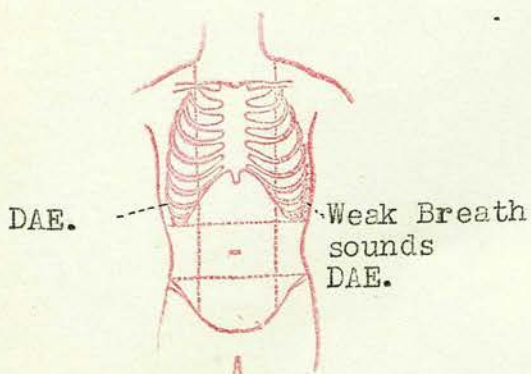
History of Present Illness Pleurisy four years' ago,
cough and Bronchitis six
weeks' ago, cough with slate
colour expectoration. Has
worked thirty years under-
ground.

Present Symptoms Frequent cough with copious
sputum. Appetite good,
lassitude, general weakness
and shortness of breath,
tightness across the chest.

Clinical Examination Upper part of chest immo-
bile and emphysematous. Ev-
idence of old basal pleur-
isy.

Expiration 31 ins.
Inspiration 33 ins.

Sputum - negative.



WILLIAM JENKINS.

PRE-
SILI-
COTIC
STAGE



X-RAY Examination - R. Scattered coarse type of mottling throughout both lung fields. Diaphragm regular.

L. Nil definite in apex. Scattered coarse type of mottling in lung fields. Diaphragm regular. Heart normal. All suggestive of early silicotic lesion. No evidence of tuberculosis.

June 7. 32	Ol morrhue	3 ⁱⁱ	mane et nocte.
July 10. 32	" "	"	" " "
Aug. 7. 32	" "	"	" " "
Sep. 14. 32	" "	"	" " "
Oct. 21. 32	" "	"	" " "
Nov. 5. 32	" "	"	" " "
Dec. 6. 32	" "	"	" " "
Jan. 1. 33	" "	"	" " "
Feb. 14. 33	" "	"	" " "
Mar. 2. 33	" "	"	" " "
Apl. 17. 33	" "	"	" " "
May 19. 33	" "	"	" " "
June 6. 33	" "	"	" " "
July 5. 33	" "	"	" " "

WILLIAM LEWIS,
62, CYFYNG ROAD, AGE - 54 OCCUPATION - MINER.
YSTALYFERA

Previous Illnesses Nil.

Family History affected .. Brother died from silico-
with Tuberculosis sis with Tuberculosis aged
65.

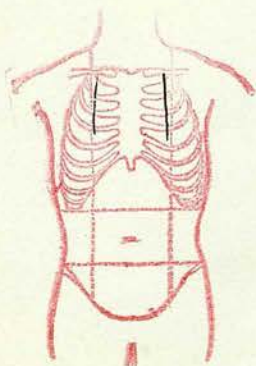
History of Present Illness Has worked 40 years under-
ground, three years on hard
headings with boring mach-
ine. First symptom noticed
was shortness of breath
six weeks' ago. Periodic
attacks of coughing.

Present Symptoms Paroxysmal cough with cop-
ious mucopurulent sputum,
appetite poor, lassitude,
shortness of breath, and
tightness across chest.

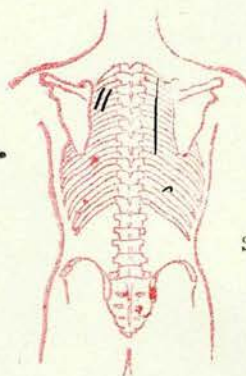
Clinical Examination Inspiration 33 ins.
Expiration 32½ ins.

Sputum negative. Expiration
prolonged all over chest.

No adventitious sounds.



Slightly
impaired
resonance.
VR +



Harsh
Breath
sounds.

WILLIAM LEWIS.



3rd
STAGE

X-Ray Examination Ossification of costal cartilages.

R. Coarse mottling throughout upper two-thirds of lung fields. Rather more dense in upper and mid zones. Diaphragm flattened and shows some irregularity in outline.

L. Scattered coarse mottling in lung fields. Diaphragm outline not well defined. Appearance suggestive of silicosis.

15. 5. 32 Sputum negative for tubercle.
25. 5. 32 " " " "
- June 6. 32 Spasmodic asthmatic attacks with great dyspnoea, and later copious sputum only relieved with ephedrine and stramomium.
- June 18. 32 Ol morrhue 3ⁱⁱ mane et nocte.
- July 14. 32 Two pts. of milk per diem as extra nourishment from Public Assistance Committee.
- July 26. 32 Ol morrhue 3ⁱⁱ T.I.D.
- Aug. 14. 32 Spasmodic attacks of dyspnoea. Ephedrine
- Aug. 26. 32 Cough with black sputum, very dyspnoeic.
- Sep. 12. 32 Ol morrhue. Tr. stramomium with expect. mxt.
- Sep. 20. 32 Ol morrhue. Dyspnoea not so marked.
- Oct. 4. 32 Ol morrhue 3ⁱⁱ mane et nocte.
- Oct. 18. 32 " " " " " "
- Nov. 1. 32 No change in physical signs.
- Nov. 27. 32 No improvement.
- Dec. 3. 32 Paroxysmal attacks, chest emphysematous attack only relieved by Ephedrine or Stramomium.
- Dec. 31. 32 Ol morrhue 3ⁱⁱ mane et nocte.
- Jan. 17. 33 " " " " " "
- Jan. 23. 33 Extra milk still continuing.
- Feb. 14. 33 Dyspnoea marked on exertion.
- Feb. 28. 33 Ol morrhue 3ⁱⁱ T.I.D.
- Mar. 6. 33 " " " "
- Mar. 15. 33 " " " "
- Apr. 24. 33 Still Dyspnoea and cough. Black sputum.
- May. 17. 33 Sputum still negative for Tubercle.

May 28. 33 01 morrhue 3ⁱⁱ T.I.D.
 June 6. 33 " " " "

Previous illnesses

Family History with regard
 to Rheumatoid

History of present illness ————
 Patient underwent for
 2 1/2 years. She worked 10
 hours a day and was
 very busy. She had
 experienced an increase of
 weight of 10 to 15 pounds.

Present Symptoms
 She has been
 unable to walk
 and is confined
 to bed.
 Weight - 110 lbs.

Clinical Examination
 Best approximations. Good
 clear breath for the
 condition of the
 chest.
 Spleen - 12 cm.
 Liver - 12 cm.

EVAN DAVIES,
45 GURNOS ROAD, AGE - 60 OCCUPATION - UNDERGROUND
YSTALYFERA. HAULIER.

Previous Illnesses Nil.

Family History with regard
to Tuberculosis Nil.

History of present illness Has worked underground for
45 years. Has worked in
hard headings and used bor-
ing machine every day. Has
complained of shortness of
breath for 4 to 5 years.

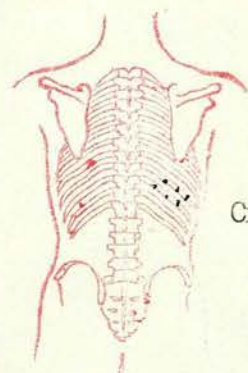
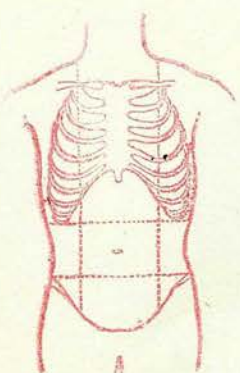
Present Symptoms Cough, shortness of breath,
appetite good, no sputum.

Capacity for work - getting
about.

Weight - 14 st. 6 lbs.

Clinical Examination Chest emphysematous. Bases
clear except for few crep-
itations at right base.

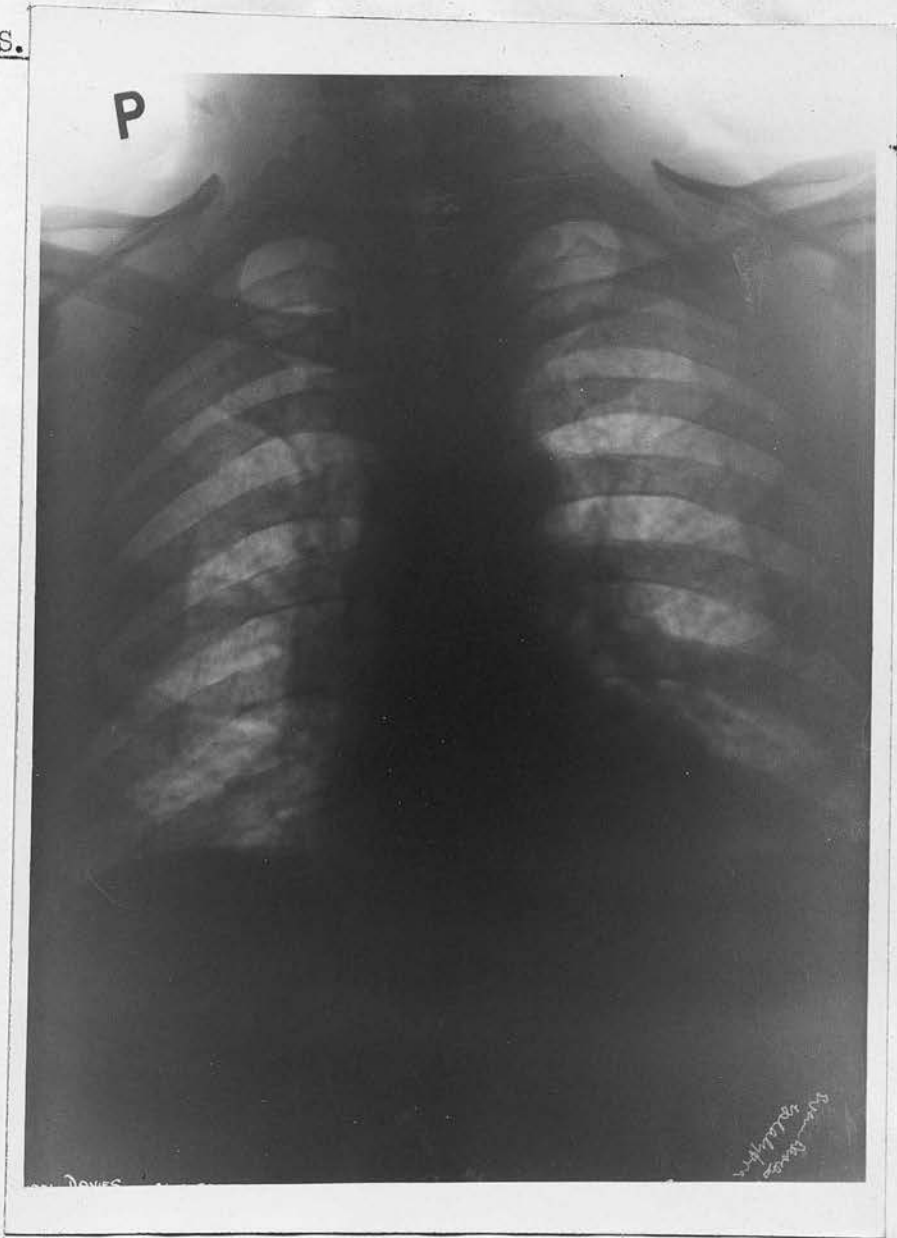
Expiration $32\frac{1}{2}$ ins.
Inspiration $33\frac{3}{4}$ ins.



Crep.

EVAN DAVIES.

2nd
STAGE



X-Ray

Film rather thin. Scattered reticulate mottling throughout both lung fields. Diaphragm moves fairly regular though somewhat flattened.

There appears to be evidence (silicosis) bilateral fibrosis probably silicotic in nature.

31. 1. 33 Well covered. Chest markedly emphysematous, no cough but marked dyspnoea.

Feb. 14 Unable to do any light work owing to dyspnoea.

" 28 Mist Expect. Tr. Digitalis MX T.I.D.

Mar. 13 " " " " " "

" 29 " " " " " "

Apl. 16 Dyspnoea still great.

Apl. 30 No change.

May 12 Confined to house. Too dyspnoeic to walk.

May 26 Mist. Expect.

June 14 " "

EVAN EVANS,
9 PROSPECT PLACE, AGE - 44
YSTALYFERA.

OCCUPATION - UNDERGROUND
HAULIER.

Previous Illnesses Nil.

Family History with regard
to Tuberculosis Nil.

History of Present Illness
Has worked underground for
28 years. Has not worked
in hard headings, but has
used the boring machine for
17 years off and on. Now
complains of shortness of
breath, especially at night,
slight cough with some frothy
sputum. Tightness of chest
in early mornings.

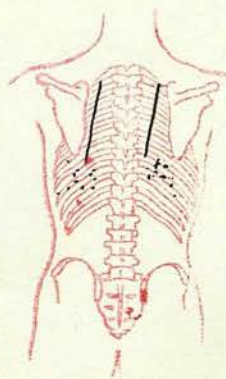
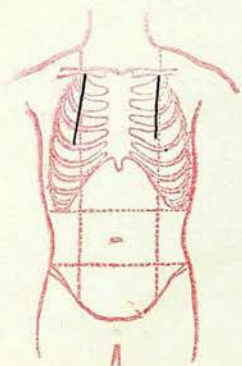
Present Symptoms Morning cough, sputum mucous
scanty, appetite poor, lass-
itude, loss of flesh, short-
ness of breath and tight-
ness across the chest.

Capacity for work - getting
about.

Clinical Examination ... Weight - 9 st. Has been 10
st. 4 lbs.

Expiration $30\frac{1}{2}$ ins.
Inspiration $31\frac{3}{4}$ ins.

Sputum negative. General
percussion note impaired.
Very weak breath sounds,
extra severe catarrhal signs
R. & L.





PRE-
SILI-
COTIC
STAGE

- X.-Ray ... R. Scattered stippling throughout lung fields. Diaphragm flattened but regular in outline, costophrenic angle not well defined.
- L. Scattered stippling in lung fields. Diminished translucency in both apical regions. Compatible with appearance of hiliary tubercle.

9. 6. 32 Paroxysmal attacks of dyspnoea. Ephedrine
relieves it.
15. 6. 32 Ol morrhue. Mist Expect.
6. 7. 32 Carried on work as miner. States he feels
better when working. Attacks not so
severe when working.
16. 9. 32 Dyspnoea and paroxysmal attacks at night,
cough.
17. 9. 32 Asthma. Ol morrhue. Ephedrine.
18. 9. 32 " " " "
- 15.10. 32 Back at work again.
- 22.10. 32 Ol morrhue.
- Jan. 6. 33 Influenza, seen daily. Syr Easton and
ol morrhue. during convalescent stage.
- Feb. 14. 33 Ol morrhue.
- Mar. 1. 33 Back at work again.
- Apr. 6. 33 Still much the same, much better when
working.
- May 10. 33 Ol morrhue 3ü T.I.D.
- June 14. 33 " " " "

S. EDWARD WILLIAMS,
25, GRAIGYMERCHED RD. AGE-50
YSTALYFERA.

OCCUPATION - MINER.

Previous Illnesses

Neuritis in 1913.

Family History with regard
to Tuberculosis

Nil.

History of present illness.

Has worked underground for
39 years, 10 years of which
he worked in hard headings.
Has done a lot of boring
and repairing. He first
complained of shortness of
breath, with occasional
cough and loss of weight.

Present Symptoms

Cough - occasional.
Sputum - nil.
Voice - hoarseness.
Appetite - poor.
General - loss of weight,
lassitude, and shortness of
breath.
Capacity for work - getting
about.

Clinical Examination

Weight 8 st. 9 lbs. gener-
al condition fair.

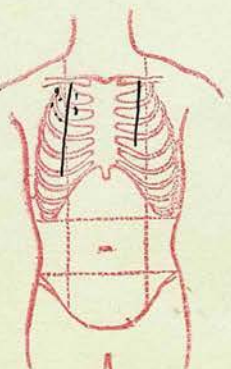
Expiration - $29\frac{1}{2}$ ins.
Inspiration - $30\frac{3}{4}$ ins.

Thinly covered.

Sputum Examination

Negative. Patient states
that he wears a handkerchief
around mouth when boring
through rock.

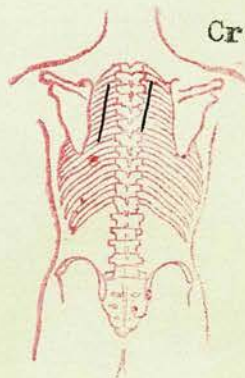
Weak
Breath
sounds
Rough
systo-
lic
murmur
at
apex.



Harsh Breath sounds.

Pulse 80.

Crepitations



PRE SILICOTIC STAGE.



X-RAY Heart shows tendency to vertical position. The outline of the diaphragm is clear. There is some tile roofing. The lung fields show a diffuse ground glass appearance. There is no evidence of tuberculosis.

26. 4. 33 Dyspnoea and cough; apical pain. B.P. 145
90. Mist Expect. Ol morrhue.
30. 4. 33 Mist Expect. Ol morrhue.
17. 5. 33 Pulse 80. B.P. 145 90.
26. 5. 33 Still complaining of anginal pain, cough
and dyspnoea.
- June 7. 33 Ol morrhue 3 \bar{ii} T.I.D.
- " 16. 33 Is improving, anginal pain gone, dyspnoea
- " 30. 33 Dyspnoea still present on exertion. Ol
morrhue.

Present Symptoms

Frequent cough with scanty
sputum. Poor appetite.
No fever, breathing normal
at rest, slight wheezing
on exertion. No rales or
crackles. No cyanosis.

Clinical Examination

Weight - 9 st. 12 lbs.

Respiration - 20 per min.
Inspiration - 1 1/2 sec.

Dullness over both apices
scattered rales and rhonchi
over both lungs. Prolongation
of expiration. Sputum
negative on three occasions.

DAVID DAVIES,
53 HODGSON ROAD, AGE - 45
YSTALYFERA.

OCCUPATION - MINER.
(Has worked all his life in
the mines including bor-
ing in hard headings).

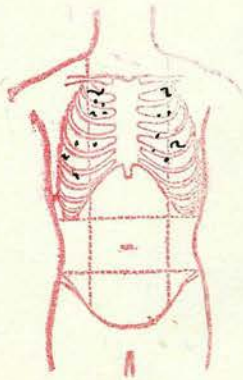
Previous Illnesses Nil.

Family History with regard
to Tuberculosis Daughter aged 7 has been in
Sanatorium for two years.

History of present Illness
Complains of cough, and
breathlessness. Has always
worked in anthracite coll-
eries.

Present Symptoms Frequent cough with scanty
sputum. Poor appetite, loss
of flesh, tightness across
the chest, night sweats. Cap-
acity for work - getting
about.

Clinical Examination ... Weight - 9 st. 12 $\frac{1}{4}$ lbs.
Expiration 30 $\frac{1}{2}$ ins.
Inspiration 31 ins.
Dullness over both apices
Scattered rales and rhonci
over both lungs. Prolong-
ation of Expiration. Spu-
tum negative on three occ-
asions.



R



3rd
STAGE
PLUS
TUBER-
CULOS-
IS.

X-RAY R.

Infiltration of apex and throughout lung, most dense in the upper half. Diaphragm irregular and peaked. Costo phrenic angle not well defined.

L.

Infiltration of apex and upper two thirds of lung fields. Diaphragmatic adhesions. Very coarse mediastinal shadows. Heart to the right. Marked opacity of lesion in both inter-lobar regions. A great deal of fibrosis in R. & L. Lungs. Definite silicosis with tuberculosis.

July 6. 32 Cough very troublesome. Dyspnoea marked
 Mist. Expect. and ol morrhue 3 \bar{ii} T.I.D.
 " 15. 32 Mist. Expect. Vin Ipecac 3 \bar{ii} Tr camph Co.
 3 \bar{ut} ammon. carb. gr. xxx. aq. cHcl₃ ad
 38. 3 \bar{ss} . e. 4 hs.
 Aug. 12. 32 Ol morrhue 3 \bar{ii} T.I.D.
 Aug. 17. 32 Two pts. of milk supplied by Public Ass-
 istance Committee as extra nourishment.
 Sep. 15. 32 Ol morrhue.
 " 30. 32 Weight improving - 2 lbs. gained.
 Oct. 4. 32 Ol morrhue.
 " 16. 32 Dyspnoea, cough, still marked.
 Nov. 20. 32 Copious sputum - black and purulent.
 Dec. 18. 32 Losing weight now - 2 lbs.
 " 27. 32 No improvement - still very dyspnoeic on
 slightest exertion.
 Jan. 6. 33 Physical signs much the same.
 Feb. 4. 33 Ol morrhue.
 " 18. 33 Bronchitis, seen daily. Mist. Expect.
 Mar. 30. 33 Syr Eastonii 3 \bar{ii} aq. ad. 38. 3 \bar{ss} . T.D.S.
 p.c.
 Apl. 7. 33 Ol morrhue. Keeping much the same.
 " 18. 33 No improvement.
 May 30. 33 Ol morrhue 3 \bar{ii} T.I.D.
 June 16. 33 " " " "

ELLIS WYNNE THOMAS,
18, SWAN LANE, AGE - 25 OCCUPATION - MINER.
YSTALYFERA.

Previous Illnesses Influenza in 1922.

Family History with regard
to Tuberculosis Nil.

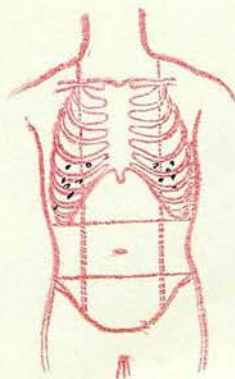
History of Present Illness Has worked underground for
15 years. Uses the boring
machine on most days. Now
complains of cough with short-
ness of breath, no sputum

Present Symptoms Frequent morning cough, no
sputum, appetite fairly
good and tightness of chest

Capacity for work - getting
about.

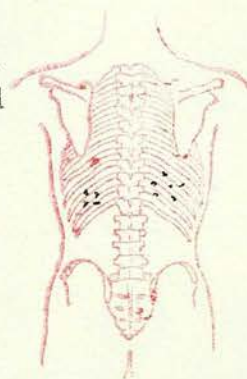
Clinical Examination Weight 10 st. 1 lb. Highest
10 st. 13 lbs. General
condition good. No sputum

Expiration 33 ins.
Inspiration 35 ins.



Harsh breath
sounds especially at end
of inspiratory phase.

Crepitations
at bases.



ELLIS WYNNE THOMAS.

2nd
STAGE



E. W. T.
Ellis Wynne Thomas 30.1.33

Swanwick, D. J.

X-RAY R. & L.

Uniform coarse mottling throughout both lung fields. Some apical stippling. Both diaphragm leaves normal in contour. Bilateral fibrosis probably silicotic in character.

25. 1. 33 Dyspnoea only.
2. 2. 33 Ol morrhue. Advised light work in open.
15. 2. 33 Unable to obtain work. Ol morrhue.
22. 2. 33 Improving, no cough, dyspnoea better.
4. 3. 33 Ol morrhue.
24. 4. 33 Steady improvement, weight - 4 lbs. gained
14. 5. 33 Ol morrhue. Improvement continues.
21. 5. 33 " " " " "
14. 6. 33 " " " " "
24. 6. 33 " " " " "
17. 7. 33 " " " " "

WILLIAM ARTHUR EVANS,
5, COMMERCIAL STREET, AGE-52
YSTALYFERA.

OCCUPATION - MINER.

Previous Illnesses Pleurisy in 1931.

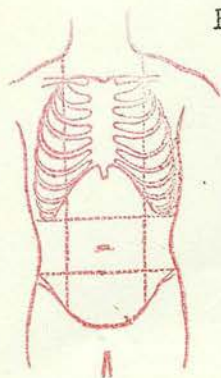
Family History with regard.
to pulmonary tuberculosis. Nil.

History of Present Illness. Has worked underground for
30 years. Has done a great
deal of boring and ripping
top. Fall or strain four
weeks' ago and now com-
plains of pain in right
side. No cough, no sputum.

Clinical Examination Examination nil definite.

Expiration $29\frac{1}{2}$ ins.
Inspiration $31\frac{1}{2}$ ins.

Few crepitations.



1st
STAGE



X-RAY

.....

The heart is normal in size, and position of diaphragm outline clear. Throughout both lung fields there is a diffuse mottling but no evidence of tuberculosis, but suspicion of a cavity in right 1st interspace.

- 4. 3. 33 Pain in right side, no physical signs. Dyspnoea.
- 12. 3. 33 01 morrhue 3ü T.I.D.
- 21. 3. 33 " " " "
- 4. 4. 33 Resumed work as miner.

History of Present Illness: Onset of breath shortness 1931, slight cough. ...

 years of age.

Present Symptoms: Slight cough with occasional ...
 loss of weight, shortness of breath, tightness across the chest.

Physical Examination: ...

Physical Examination: ...
 ...
 ...

Expiration 23 sec.
 Inspiration 22 sec.

Spinal negative.

Diagnosis: ...

DANIEL DAVIES,
BRYNCETHYN.

AGE - 61

OCCUPATION - UNDERGROUND
LABOURER.

Previous Illnesses Typhoid 37 years ago.

Family History with regard to Tuberculosis Son aged 21 affected with Tubercle now in Sanatorium

History of Present Illness Shortness of breath since 1925, slight cough. Has worked underground since 14 years of age.

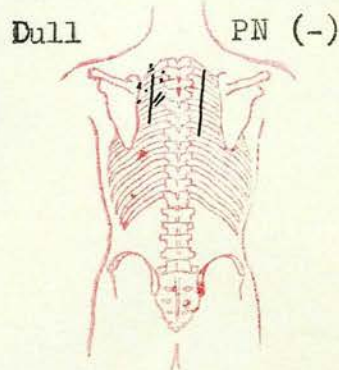
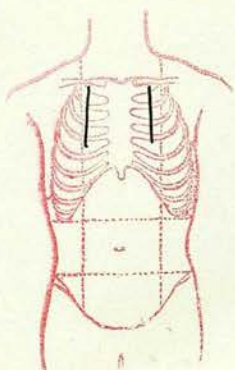
Present Symptoms Slight cough with scanty sputum, loss of weight, shortness of breath, tightness across the chest.

Capacity for work - getting about.

Clinical Examination ... General condition very poor. Percussion note dull left upper lobe and impaired generally. Left upper lobe slight catarrh. Heart sound rapid, 1st sound harsh murmur. Arteriosclerosis.

Expiration 29 ins.
Inspiration 30 ins.

Sputum negative.



DANIEL DAVIES.



3rd
STAGE.

X-RAY

Cricket ball opacities involving the apices and upper third of each lung field. Scattered coarse mottling below. Both diaphragm leaves peaked and irregular. Retraction of chest wall. Mediastinum to the left, emphysema at bases. Bilateral dense fibrosis upper part of lung, scattered fibrosis below, probably silicotic lesion of cricket ball type, possibly superimposed tubercle but not suggestive of this.

28. 3. 32	Dyspnoea marked. Mist. Expect. General condition poor.
6. 4. 32	Ol morrhue 3 ⁱⁱ T.I.D.
20. 4. 32	" " " "
30. 4. 32	Still working on surface.
12. 6. 32	Troublesome cough. Mist Expect.
4. 7. 32	Ol morrhue.
17. 7. 32	Only complains of dyspnoea - no other symptom.
7. 8. 32	" " " " " " "
20. 8. 32	Ol morrhue 3 ⁱⁱ T.I.D. General condition fairly good.
4. 9. 32	Still at work. Has permission to take things easy.
10.10. 32	Ol morrhue.
25.10. 32	" "
7.11. 32	Dyspnoea only complaint.
19.11. 32	Ol morrhue 3 ⁱⁱ T.I.D.
16.12. 32	" " " "
30.12. 32	" " " "
16. 1. 33	" " " "
30. 1. 33	" " " "
2. 2. 33	Weight same - no change.
15. 2. 33	Still working every day from 7 a.m. to 3 p.m.
3. 3. 33	Ol morrhue 3 ⁱⁱ T.I.D.
27. 3. 33	" " " "
18. 4. 33	" " " "
4. 5. 33	Slight cough in mornings.
16. 5. 33	Ol morrhue.
30. 6. 33	" "

JOHN E. D. JONES,
98 CLIFTON HILL, AGE - 28 OCCUPATION - MINER. 8/7/32
YSTALYFERA.

Previous Illnesses Bronchitis 1924, 26 - 28.

Family History with regard
to Tuberculosis Nil.

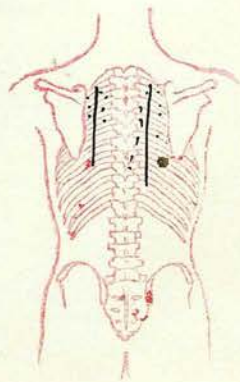
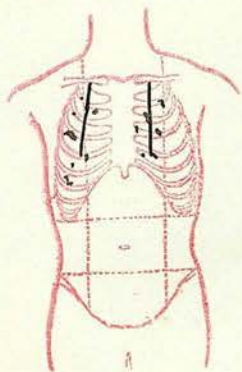
History of Present Illness Has worked underground for
14 years, hard headings and
general work as collier.
Complains of cough of four
years' duration with a
bluish expectoration, short-
ness of breath and lassit-
tude.

Present Symptoms Morning cough with copious,
sometimes streaky sputum.
Shortness of breath, tight-
ness across the chest, lassit-
tude and general weakness.

Clinical Examination Slightly hyper resonant.
percussion note over both
lungs. Weak breath sounds.
Sibilant rhonci in both
lungs. No evidence of ac-
tual Tuberculosis.

Expiration 31 ins.
Inspiration 32½ ins.

Sputum negative.



JOHN E. D. JONES.



1st
STAGE.

X-RAY R. Scattered coarse mottling throughout lung fields, diaphragm regular.

L. Scattered coarse mottling throughout lung fields, general appearance suggesting silicosis.

8. 7. 32 Very dyspnoeic, cough and expectorant.
 Weekly attendances during July, 1932.

Aug. 5. 32 Much the same.

Sept. 12. 32 Ol morrhue 3ⁱⁱ mane et nocte.

Oct. 14. 32 Ol morrhue.

Nov. 3. 32 Influenza - daily visits. Tr Digitalis
 m vii. T.I.D.

Dec. 18. 32 Syr Eastonii 3^{ij} fo. aq. ad. 38. 3^{ss} T.I.D.

Jan. 33 Ol morrhue.

Feb. 7. 33 " "

Mar. 20. 33 " " mane et nocte.

Apl. 8. 33 Mist. Expect.

May 15. 33 Ol morrhue 3ⁱⁱ T.I.D.

June 6. 33 " " " "

July 12. 33 " " " "

IOAN HOWELLS,
AEL-Y-BRYN,
YSTALYFERA.

AGE - 58

OCCUPATION - MINER.

Previous Illnesses Nil.

History of Family with re-
gard to Tuberculosis ... Nil.

History of Present Illness Has worked underground for
47 years as a coal hewer
which included much boring
First symptom noticed was
shortness of breath.

Present Symptoms Frequent cough with slight
haemoptosis, copious sput-
um, mucous and often black,
especially in the morning,
appetite good, lassitude,
loss of weight, shortness
of breath and tightness
across the chest.

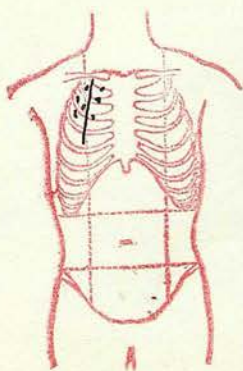
Capacity for work - getting
about.

Clinical Examination General condition fair.

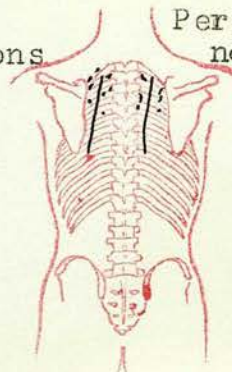
Expiration - 30 ins.

Inspiration - 30½ ins.

Sputum examination - neg-
ative to tubercle Bacilli.



Crepitations



Percussion
note im-
paired

Harsh
breath-
ing.

Dimin-
ished
air entry

IOAN HOWELLS.



3rd
STAGE

X-RAY Examination R. Dense opaque areas extending from 1st rib to 5th rib in peripheral region. Coarse mottling inner part of lung fields and third quarter. Diaphragm completely irregular and opaque, apex shows loss of translucency.

L. Appearances similar to changes in R. side, but density of lesion is more peripherally confined. Wide mediastinum. There is a marked degree of fibrosis in both lungs and the appearances suggest Silicosis Stage III. Appearance of right upper zone suggests some softening. Cavitation suggestive of superimposed Tuberculosis. Emphysema at bases.

24. 2. 33 Very dyspnoeic with copious streaky sputum
 3. 3. 33 Unable to walk any distance.
 12. 3. 33 Ol morrhue 3ü T.I.D.
 18. 3. 33 " " " "
 12. 4. 33 " " " "
 16. 4. 33 Able to move about garden only
 24. 4. 33 Ol morrhue 3ü T.I.D.
 12. 5. 33 Very dyspnoeic. Cough with copious sputum especially nocte.
 24. 5. 33 Ol morrhue. No change.
 16. 6. 33 Sputum examined by concentration method - negative.
 24. 6. 33 Putting on weight - 3 lbs. Still dyspnoeic, very marked especially on slightest exertion, cough, sputum, not so troublesome.
-

JOHN JAMES,
39. CYFYNG ROAD, AGE - 48
YSTALYFERA.

OCCUPATION - MINER - 22 YRS.
MINE EXAMINER - 14 YRS.

Previous Illnesses Pleurisy right side 3 months
Feb. 1930. Pneumonia 1931.

Family History in regard
to Tuberculosis Nil.

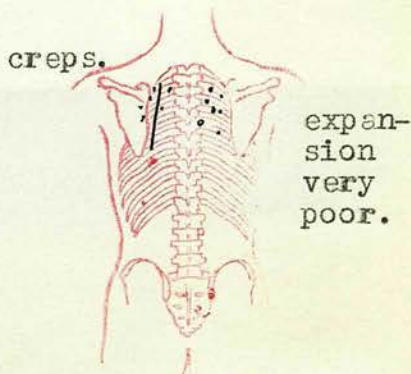
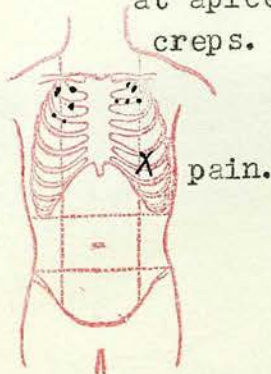
History of Present Illness Weakness and shortness of
breath. Very slight cough.

Present Symptoms Paroxysmal cough with copious
black sputum, appetite poor,
lassitude, loss of flesh,
shortness of breath, night
sweats.

Capacity for work - getting
about.

Clinical Examination ... Weight 9 st. Highest 9 st.
6 lbs.
Expiration 31 ins.
Inspiration 32 ins.
General condition - fair.
Sputum negative on five different occasions.

Impaired Resonance both sides especially
at apices.



JOHN JAMES.

THIRD STAGE - PLUS TUBERCULOSIS.



X-RAY

.....

Coarse mottling whole of both lungs,
opaque at $\frac{3}{4}$ of both lungs.

R. Diaphragm slightly sloped and peaked.

L. Diaphragm slightly irregular and peaked.
Film gives appearance of advanced pneumoconiosis, with superimposed tubercle.

14. 2. 33 Dyspnoea, Tachycardia. Pleuritic pain in side. Tr. camph Co. ol morrhue.
21. 2. 33 Improved weight 2 lbs. Dyspnoea no change
5. 3. 33 Dyspnoea marked, pulse 80. Temp. 98.4.
4. 4. 33 Ol morrhue 3 \bar{ii} T.I.D.
16. 4. 33 Able to walk short distances. Ol morrhue.
12. 5. 33 Still dyspnoeic. Ol morrhue.
21. 5. 33 " " " "
6. 6. 33 Ol morrhue. Cough troublesome at night - black sputum.
14. 6. 33 Ol morrhue 3 \bar{ii} T.I.D.

Present Symptoms

Weight 110 lbs. Height 5 ft. 10 in. Temperature 98.4. Pulse 80. Respiration 18. Cough troublesome at night - black sputum.

Clinical Examination

General appearance - well developed. Heart - normal. Lungs - normal. Abdomen - normal. Urine - normal.

Diagnosis - Chronic Bronchitis.

Prognosis - Good.

JOHN JENKIN GAPE,
28 VARTEG ROAD, AGE - 43
YSTALYFERA.

OCCUPATION - MINER.
14/2/31.

Previous Illnesses Nil.

Family History affected by
Tuberculosis Nil.

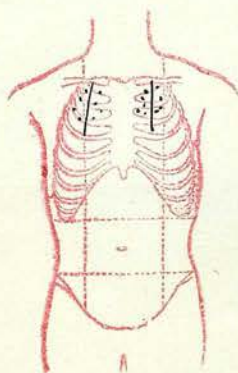
History of present Illness
Influenza three years' ago.
Has worked underground 1919-
1930. Hard headings from
1919-1922 and 1923-1925.
Boring with machine drills
during this time. Now com-
plains of pain in chest, and
shortness of breath, cough
and expectoration. Has not
worked underground for the
last three years, as the
colliery has closed down.

Present Symptoms Frequent cough with scanty
mucous sputum, appetite poor,
lassitude, loss of flesh,
shortness of breath, tight-
ness across the chest, pain
in chest and night sweats.

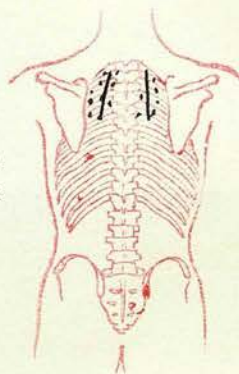
Clinical Examination High colour, well covered.
Catarrhal signs in both
lungs. General percussion
note impaired. Weak breath
sounds posteriorly.

Expiration - 33 ins.
Inspiration - 34½ ins.

Sputum negative.



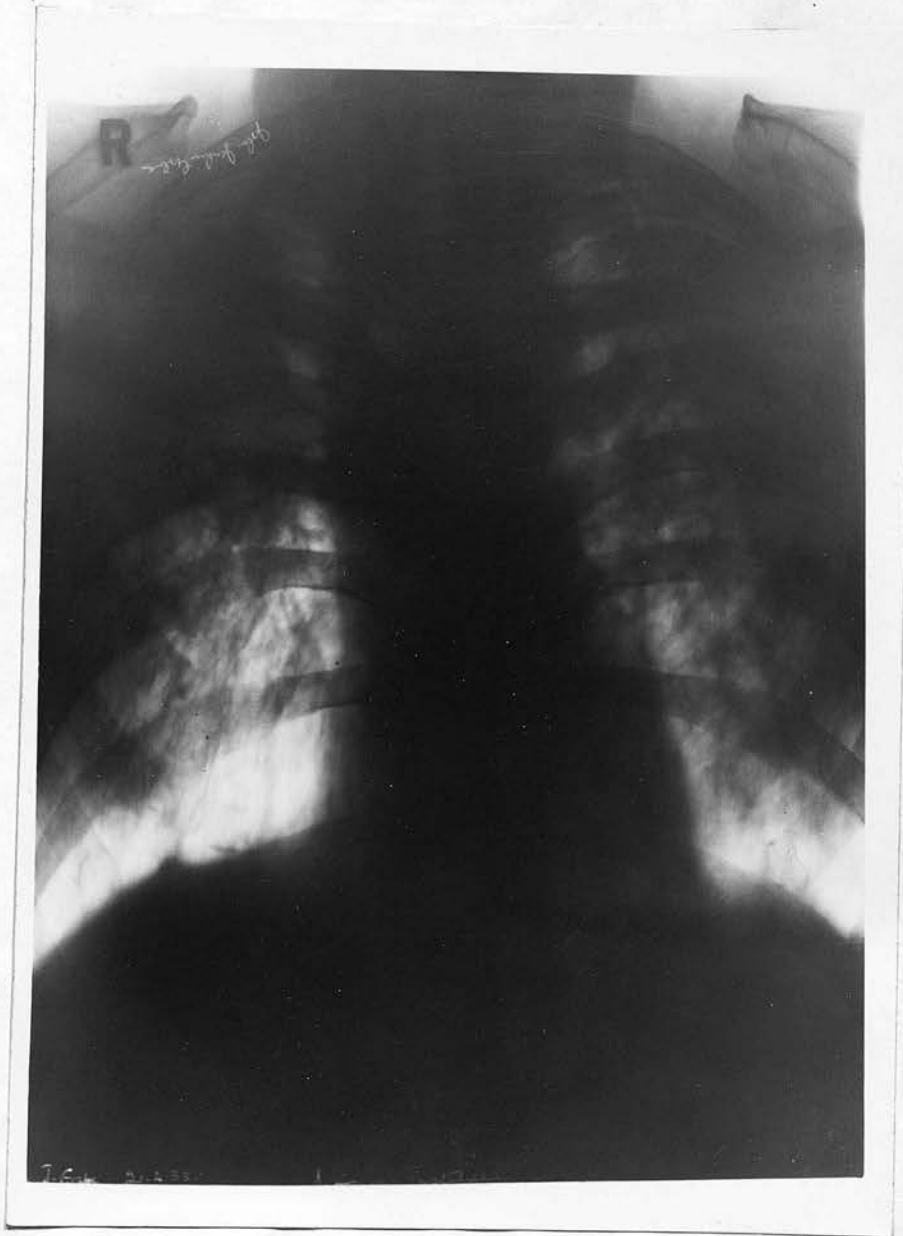
Weak
Breath
sounds



Weak
Breath
sounds

JOHN JENKIN GAPE.

3rd
STAGE



X-RAY

- R. Dense consolidated areas involving apex and upper part of lung with thickening of the pleura. Mottling in lower half with streaky extensions downwards. Diaphragm peaked and shows adhesions in inner part.
- L. Dense opacity involving apex and peripheral part of upper half of lung with pleural thickening; woolly mottling elsewhere. Diaphragm outline completely irregular. Condition 3rd stage silicosis, but superimposed tubercle cannot be excluded.

14.	2.	31	Expectorant mixture	℞ Vin Ipecac 3 ⁱⁱ ammon. carb. gr. xxx Tr Camph Co. ʒvi aq. cHcl ₃ ad ʒ8 3 ss. 4 hrly.
28.	2.	31	Condition much the same.	Ol morrhue twice daily.
3.	3.	31	Condition much the same	" "
14.	3.	31	" " " "	" "
21.	3.	31	Tr digitalis mvii	T.I.D.
31.	3.	31	Not so dyspnoeic. Cough better;	ol morrhue
Apl.	12.	31	Ol morrhue 3 ⁱⁱ	mane et nocte.
"	14.	31	" " " "	" " "
"	26.	31	" " " "	" " "
May	10.	31	" " " "	" " "
"	17.	31	" " " "	" " "
"	24.	31	" " " "	" " "
"	30.	31	" " " "	" " "
June	10.	31	" " " "	" " "
June	14.	31	" " " "	" " "
"	28.	31	" " " "	" " "
July - Aug.			Sent to Miners' Convalescent Home, Tal-y-garn.	
Sep.	30.	31	Still Dyspnoeic on exertion.	Has put on 4 lbs. in weight.
Oct.	30.	31	Slight Bronchial catarrh.	Mist. Expect.
Nov.	30.	31	Improving.	
Dec.	31.	31	"	
Jan.		32	Four attendances - doing well on ol morrhue	
Feb.		32	" " " "	" " "
Mar.		32	" " " "	No change.

Apl.	32	Four attendances.	No change.
May	32	" "	Still dyspnoeic. Cough much better.
June	32	Four attendances.	" "
July	32	" "	" "
Aug.	32	" "	Still on ol morrhue.
Sep.	32	" "	" " " "
Oct.	32	" "	" " " "
Nov.	32	" "	" " " "
Dec.	32	" "	" " " "
Jan.	33	" "	" " " "
Feb.	33	" "	" " " "
Mar.	33	" "	" " " "
Apl.	33	" "	" " " "
May.	33	Still rather dyspnoeic on exertion.	
June	33	" "	" " "

WILLIAM LLOYD,
13, GURNOS ROAD, AGE - 59 OCCUPATION - MINER.
YSTALYFERA.

Previous Illnesses Nil.

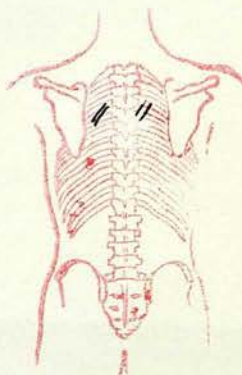
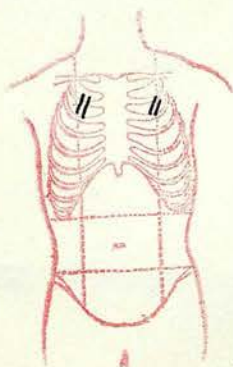
Family History with regard
to Tuberculosis Nil.

History of Present Illness
General weakness, shortness
of breath, high blood pres-
sure, giddiness. Has worked
underground for 40 years,
both as a repairer and as
a coal hewer.

Present Symptoms Morning cough with scanty
sputum, lassitude, loss of
flesh, and marked short-
ness of breath.

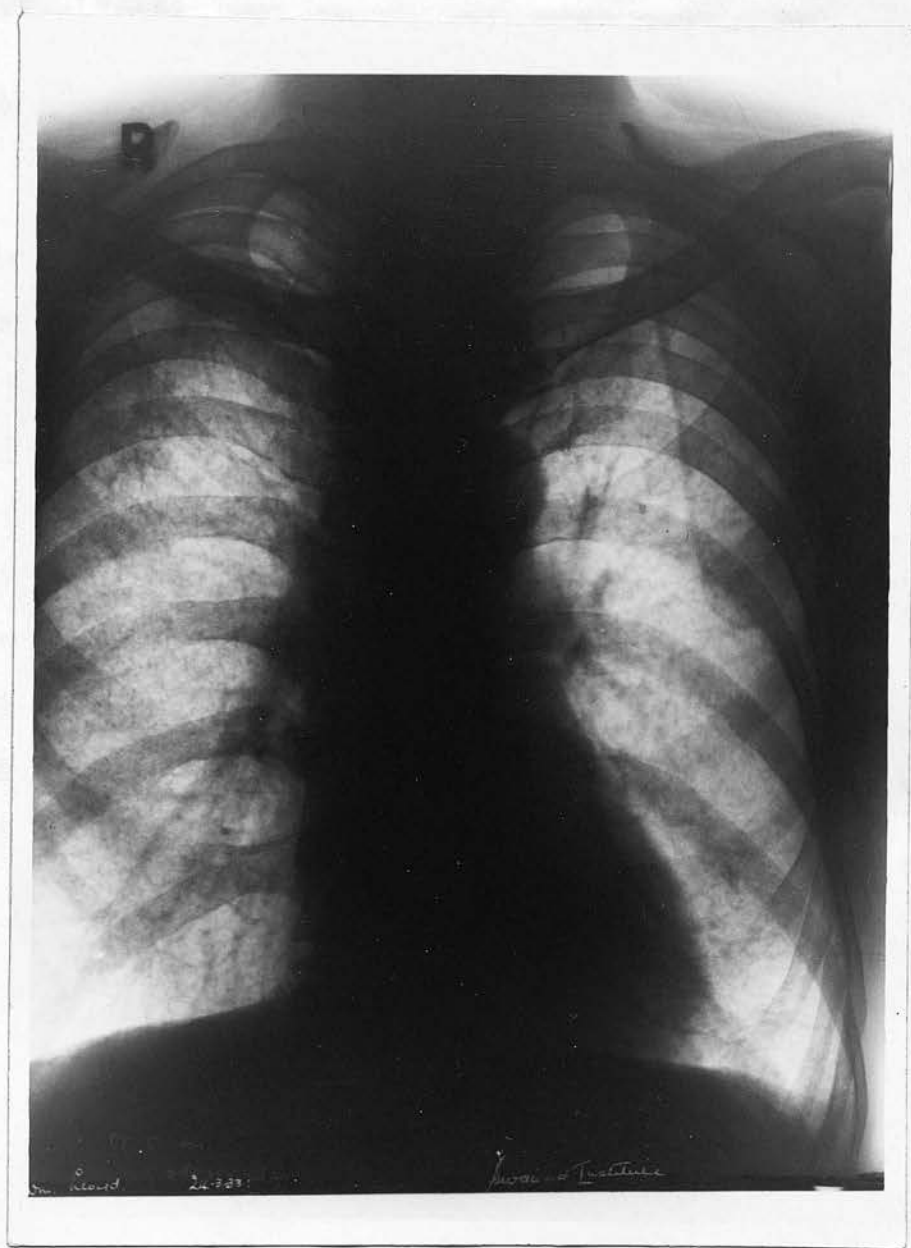
Capacity for work - getting
about. Weight 10. st. 10.
lbs. General condition fair-
ly good.

Clinical Examination Expiration 32 ins.
Inspiration 33½ ins.
Indefinite harsh breath
sounds, well marked Tachy-
cardia-myocarditis. Pro-
bably silicosis with myo-
carditis. Sputum negative



WILLIAM LLOYD.

1st STAGE.



X-RAY Coarse mottling throughout both lung fields. Diaphragm leaves flattened. Costo-phrenic angle completely obliterated.

17. 12. 32 Dyspnoea. Fibrosis with myocarditis.
Pulse 120. B.P. 140 Systolic 100 Diastolic.
24. 12. 32 Tr. Digitalis mxv. T.I.D. with Tr. nux.
vom mv. T.I.D.
31. 12. 32 " " " " " " "
4. 1. 33 Pulse 110. Tr. Digitalis mxx. Tr nux.
vom mvii T.I.D.
10. 2. 33 No change. Dyspnoea. No cough.
20. 2. 33 Pot. Iod. gr.v. T.I.D.
1. 3. 33 Pulse 100. B.P. 135. 95.
18. 3. 33 Ol morrhue 3 $\ddot{\text{ii}}$ T.I.D.
6. 4. 33 Pulse 100. Regular in time and force.
27. 4. 33 Still very dyspnoeic.
6. 5. 33 Ol morrhue.
21. 6. 33 No change.
30. 6. 33 No change.
6. 7. 33 Still dyspnoeic.

JOHN BEVAN,
4, PANTTEG,
YSTALYFERA.

AGE - 51

OCCUPATION - REPAIRER
UNDERGROUND.

Previous Illnesses Nil.

Family History with regard to Tuberculosis Son died in 1926 from Pulmonary Tuberculosis.

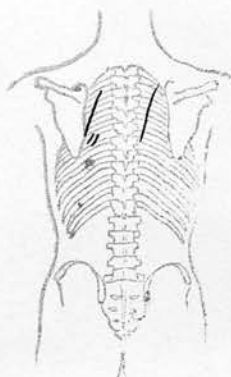
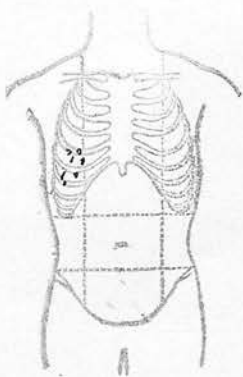
History of Present Illness
Has worked underground for 39 years. Boring off and on for the whole period. Injury to left eye three years' ago and has worked on the surface since. Now complains of pain in right side, pleuritic pain, duration four weeks. Cough and expectoration.

Present Symptoms Frequent cough, scanty sputum, appetite fairly good, lassitude, loss of flesh, tightness across the chest and shortness of breath.

Capacity for work - getting about.

Clinical Examination Weight - 10 st. 4 lbs. Sputum negative. Expiration - 33 ins. Inspiration - 34½ ins. General percussion note impaired. Breath sounds harsh.

Coughing
creps.



JOHN BEVAN.1st
STAGE

- X-RAY R. Coarse mottling throughout lung fields, appearance of subclavicular regions suggestive of superimposed tubercle. Diaphragm completely irregular. Costo-phrenic angle obliterated.
- L. Coarse mottling throughout lung fields. General appearance suggests bilateral fibrosis probably silicosis. Right apex and subclavicular region suggestive of superimposed tubercle.

Mar. 6. 33 Pain in side pleuritic - no physical signs
 of pleurisy.
 " 26. 33 Ol morrhue.
 Apl. 12. 33 Ol morrhue. Still dyspnoeic, cough bett-
 er.
 " 20. 33 Sputum negative. Still pain in side. Ol
 morrhue.
 May 16. 33 Mixt. Expect. No improvement in dyspnoea.
 " 30. 33 Ol morrhue 3ü mane et nocte.
 June 14. 33 " " " " " "

JOHN TREVOR MORGAN,
 92 CYFYNG ROAD, AGE - 35 OCCUPATION - MINER.
 YSTALYFERA.

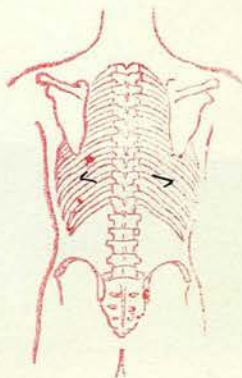
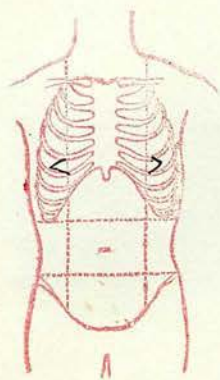
Previous Illnesses Nil.

Family History with regard
 to Tuberculosis Nil.

History of present illness Had all his teeth extract-
 ed two years' ago. Had to
 give up work owing to stom-
 ach trouble three months
 ago. Now complains of cough
 accompanied by expectora-
 tion and vomiting. Has
 worked underground for 21
 years.

Present Symptoms Cough with copious sputum,
 shortness of breath, poor
 appetite, lassitude, and
 occasional night sweats.

Clinical Examination Weight 8 st. 13 lbs. Ex-
 piration - $30\frac{1}{2}$ ins. In-
 spiration - $31\frac{1}{2}$ ins. Well
 covered chest, good expan-
 sion. Percussion note im-
 paired all over, feeble
 breath sounds, especially
 basal. Sputum - negative
 on three different occa-
 sions.



JOHN TREVOR MORGAN.



2nd
STAGE

- X-RAY R. Apical mottling, scattered coarse mottling throughout lung fields, especially upper third. Calcareous nodes in root. Sloping diaphragm.
- L. Some stippling in apex. Scattered coarse mottling in lung fields, mostly marked in upper two-thirds. Small vertical heart. There is definite evidence of bilateral fibrosis, and the appearance of right apical region is rather suggestive of superimposed Tuberculosis.

9. 9. 32 0l morrhue 3ⁱⁱ T.I.D.
 Oct. 4. 32 " " " "
 " 17. 32 Sputum negative.
 Nov. 10. 32 0l morrhue. Dyspnoea marked.
 " 30. 32 Cough and dyspnoea. Mixt expect.
 Dec. 4. 32 0l morrhue 3ⁱⁱ T.I.D.
 " 18. 32 Mixt Expect.
 Jan. 7. 33 Weight 8 st. 13 lbs. No change.
 " 30. 33 Still dyspnoeic.
 Feb. 14. 33 0l morrhue.
 " 28. 33 No improvement in chest, but looks and
 feels better.
 Apl. 7. 33 Weight - 9 st.
 " 16. 33 0l morrhue.
 May 30. 33 Sputum still negative.
 June 7. 33 Sputum - concentration method - still ne-
 gative.
 July 4. 33 0l morrhue 3ⁱⁱ T.I.D.

PHILLIP GRIFFITHS,
5, GLANYRYSGOL, AGE - 44
YSTALYFERA.

OCCUPATION - UNDERGROUND
HAULIER.

Previous Illnesses Nil.

Family History with regard
to Tuberculosis Nil.

History of present Illness Has worked 30 years under-
ground. Chest symptoms of
3 years' duration, chiefly
shortness of breath, cough
with some sputum, night and
morning. History suggest-
ive of silicotic lesion.

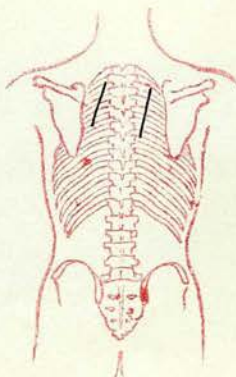
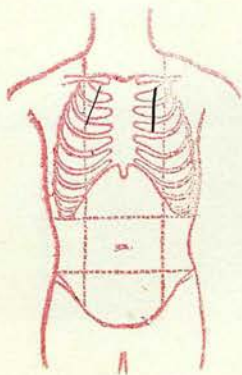
Present Symptoms Frequent cough, with copious
sputum. Appetite fairly
good, lassitude, shortness
of breath and tightness of
chest.

Capacity for work - getting
about.

Clinical Examination Expiration - 32 ins.
Inspiration - 33½ ins.

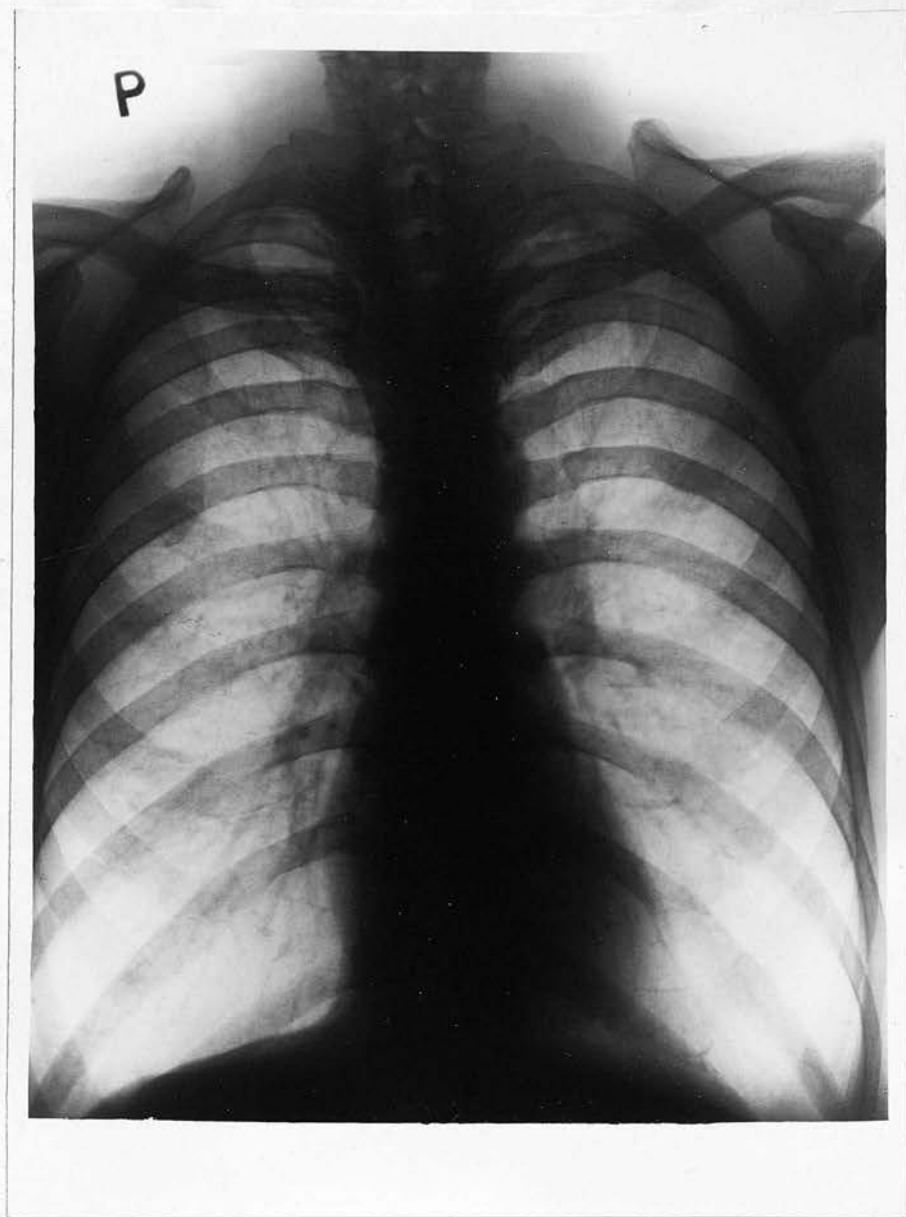
Weight 8 st. 12½ lbs. Spu-
tum negative.

No added sounds; distinct
heart sounds. Breath sounds
faint and indistinct.



PHILLIP GRIFFITHS.

PRE-SILICOTIC STAGE.



- X-RAY R. Definite apical mottling. Scattered stippling in upper zone and towards periphery. Diaphragm wavy in outline.
- L. Definite apical mottling. Stippling in upper Zones. Rather coarse type of mottling in peripheral regions. Long narrow heart central in position. General appearance compatible with silicosis, but disposition of lesion is very suggestive of tubercular infection of apices and upper zones with fibrosis.

4. 8. 31 Catarrhal signs in chest. Mixt. expect. with
ol morrhue.
11. 8. 31 Ol morrhue.
17. 8. 31 Improving - gained 3 lbs. Dyspnoea not im-
proved.
15. 9. 31 Cough and Dyspnoea worse at night. Ol morrhue.
- 12.10. 31 Improving. Ol morrhue 3 \bar{ii} mane et nocte.
- 21.11. 31 Ol morrhue.
- 4.12. 31 " "
6. 1. 32 Sputum negative.
12. 2. 32 Removed out of district - working as gardener.
23. 6. 33 Still at work as gardener. Has improved -
10 lbs. in weight - Dyspnoea still on ex-
ertion.

PROPHYLAXIS AND TREATMENT.

It has often been stated that coal miners live to a ripe old age, and that they retain their working capacity longer than most other workers; also that their liability to tubercular infection is very much less than the average male of similar age groups. It is accordingly necessary to ask the question whether the prophylaxis of this disease is really worth while.

It is the writer's opinion that the prevention of silicosis is of the utmost importance. Miners who suffer from extreme shortness of breath are daily encountered walking slowly along the streets of every mining village in the coalfield. Even while still at work they show evidence of lung injury and physical deterioration, such as loss of weight, cough with expectoration, shortness of breath.

Statistics have shewn how high is their death rate from the so-called 'Bronchitis'.

It has been previously mentioned that coal-mining is an occupation for the fit person. It is a selective occupation, and our duty, as far as possible, is to keep men with such strenuous work, free from the evils which are an accompaniment of their employment. To do this, every possible precaution must be taken against the excessive production of stone dust.

All classes of underground workers are liable

to this risk of dust inhalation, irrespective of whether they are employed in boring or otherwise engaged in handling silica rock. But, of course, the hard-heading worker, the repairer, and the rippers of the roof are more liable to this disease because they are exposed to a higher concentration of the dust. This problem of dust prevention is really a problem for the engineer, yet he can only attain success by co-operating as much as possible with the medical profession.

In the Anthracite Collieries at the present time practically all the preventive measures of coping with the dust menace are confined to the hard heading workers, and nothing is being done for the other classes of underground workers. The hard heading worker is supplied with special boring machines which trap the dust by means of a vacuum, bringing it all back from the rock into a bag. The Trewill Dust Catcher, the Hay Dust Trap, and the Flottmann Dust Preventer are typical examples of this special type of boring machine. In addition, he is also supplied with a mask or respirator. The type of mask used in the Anthracite area is the Siebe-Gorman.

It is necessary to add here that the men find it very difficult to work in the respirators. They are uncomfortable, and owing to the rapidity with which the mask becomes blocked, it frequently has to be raised to allow of free breathing. This discomfort and inefficiency of the respirators must be overcome.

During the War of 1914-1918, the box respirator which was first introduced did not prove to be a success, yet soon afterwards a respirator was used which proved to be a very efficient instrument against gas attacks. The soldiers did not find the latter type uncomfortable. Is it not possible then that an efficient and comfortable respirator can be made and used in our mines to-day just as successfully as it proved to be in the Great War?

Ventilation in the anthracite area is fairly good. Inspectors under the Ministry of Mines pay periodic visits to every Colliery, and I am satisfied that the ventilation is much improved from what it was many years' ago.

Yet, the writer contends that nothing is being done for the prevention of the inhalation of injurious dusts by other classes of under-ground workers, and as these are equally liable to silicosis, the great majority of underground workmen is being forgotten or overlooked.

The writer considers that the ideal conditions under which the coal miner should pursue his occupation would be as follows :-

A.

1. The hours of work underground should be reduced.
2. Every colliery should be provided with pit-head baths, and whilst still naked every collier should be periodically exposed to ultra-violet rays, as an anti-infective agent.

3. The workman's house should be built on modern hygienic lines with plenty of air and light and with proper sanitary arrangements.
4. His wages should be sufficient to provide him with enough nourishing food in order that he might carry out his arduous work to the best of his ability.
5. His leisure hours should be catered for by divers means of recreation.
6. The miner himself should be taught the general rules of health.
7. There is still much room for improvement in the existing archaic system of ventilation in the Collieries.

B. Prevention at the Collieries and Working-Places.

Here the most important means for prevention include :-

1. The suppression of dust.
2. Ventilation.
3. Other methods of preventing the inhalation of dust.
4. Medical Examinations.

1. The suppression of dust includes all measures which prevent the dust from being produced. It is sometimes successfully accomplished by the use of water; but to be effective the supply of water must be sufficient to keep all surfaces of the material wet during the whole process. Here, then, it is suggested that all colliery companies should make it an obligation to spray continuously all travelling roads with water.

Again, arrangements should be made so that the men must not walk in and out of the coal faces at

the same time as the horses, for if they do this they are continually walking in clouds of dust.

2. Exhaust Ventilation.

One of the most widely applied means of prevention is by localised exhaust ventilation which, to be effective, should be applied at, or as near as possible to, the point of origin of the dust and so arranged with reference to the air inlets and general ventilation of the room as to prevent the dust from entering the atmosphere of the work place.

Where the dust is produced over a large surface, as in crushing machinery, conveyors, or mechanical sieves, it is usually necessary to enclose the machine as completely as possible and to apply exhaust draughts to the inside of the housing, sufficient to maintain a slight negative pressure and to prevent escape of dust from apertures.

In the case of silica dust, where the particles are so minute, considerable care and experience are required in designing the dust-collecting plant.

It is also necessary to prevent the dust from the exhaust plant entering the atmosphere where any persons are working, and for this reason systems of localised exhaust ventilation must be provided with dust-collecting appliances.

The percussive drills worked by compressed air, which are so largely used in the mines and produce

large quantities of fine dust, can now be fitted with the dust traps already mentioned.

3. Other Preventive Measures.

Every employee underground should be provided with efficient dust masks or respirators. They should also be compelled to wear them when they work in the coal seam or in rock and also when engaged in doing shovelling work.

Respirators can never be regarded as a substitute for exhaust ventilation, but an efficient respirator is most desirable for processes which are outside the range of mechanical ventilation.

The only respirators with a filtering medium of fibre, or tissue, which are at present available afford insufficient protection against fine dusts. Investigations, however, are now being made by the Department of Scientific and Industrial Research into the efficiency of various types of respirators. Considerable progress has been made and it is hoped that a respirator possessing the necessary properties will soon be produced.

The success or failure of preventive measures depends not only on the satisfactory design and installation of the exhaust ventilation, but to an even greater extent on the proper maintenance of the apparatus or plant provided. In this connection there develops a responsibility on the workmen as well as on the management. The various codes of Regulations which have been

issued make it obligatory on the workmen employed to make a full and proper use of the appliances provided, and to report any defect forthwith to the management.

4. Initial and Periodic Examinations.

In certain industries or processes it is now required that workers shall be medically examined before they are engaged and shall also be medically examined at intervals during their employment.

The object of the initial examination is to prevent the workmen whose respiratory physique is defective and who, if not more liable to contract silicosis, run much greater danger if silicosis develops, from entering the industry.

This periodic medical examination, which would be carried out at the Works, should be of inestimable value as a preventive measure. It enables silicosis to be detected at an early stage, and if the workman is excluded from, or voluntarily gives up, further employment in dusty processes, the development of the disease may be arrested. It also enables persons found to be suffering from pulmonary tuberculosis, who not only expose themselves to additional risk by remaining in the dusty industries, but are also a source of infection to their fellow-workmen, to be eliminated. Such examinations provide cumulative evidence as to the changes in the workmen's condition and are of the greatest assistance in diagnosis when the question arises of

certification for purposes of compensation. They also indicate the stage when the conditions are inducing the disease and may thus be of material assistance in enabling the necessary precautions to be taken.

Research is proceeding under the direction of the Committee appointed by the Medical Research Council which has for its object the extension of knowledge and understanding of silicosis and other industrial diseases due to dust inhalation, particularly so in regard to the concentration of dust capable of causing injury, the conditions of exposure to dust, all which influence the progress of the disease, the mode of action of silica in the lung tissue and the influence of infections on the course of the disease.

TREATMENT.

If the man already suffers from silicosis the treatment is unfortunately disappointing. He should live as much as possible in the open air. Fresh air, good food, with two teaspoonsful of cod liver oil night and morning are essential to his well being. Exertion of any kind should be avoided and the patient should be told to take as much rest as possible.

If cough becomes troublesome, a simple expectorant mixture such as :-

\mathcal{R} Vin Ipecac 3*ss*
 Ammon. carb. grs. xxx
 Tr. camph Co. 3*vi*
 aq. chl₃ ad. 3*8*.
 } *ss* e. 4 hrs.
 / CH₂

may prove useful.

Cardiac failure, so common as a terminating feature, should be counteracted with full doses of digitalis.

When Tuberculosis is super-added, and not too far advanced, a course of Sanatorium treatment can be recommended.

-
1. Pneumoconiosis is a progressive disease, lasting many years, according to the intensity of the exposure, to produce the fibrosis characteristic of the condition.
 2. The incidence of the condition increases with the carbon content of the coal. This is most marked in three peak areas of the Anthracite Coalfield, namely, at (1) Yateville, (2) Denningsburg and (3) Ashland.
 3. The Collarials in these areas are more, very, dusty.
 4. All classes of underground workers are liable to develop pneumoconiosis and this is so whether or not they are actually engaged in boring or handling the rocks.
 5. The Registrar General's statistics of the death rate in industrial areas are built solely upon the death certificates issued by the doctor. Such statistics

SUMMARY AND CONCLUSIONS.

1. Some other factor co-exists with the silica dust in the production of pneumoconiosis in the Anthracite Coalfield, which the writer prefers to call "the infective factor".
2. Pneumoconiosis is a progressive disease, taking many years, according to the intensity of the exposure, to produce the fibrosis characteristic of the condition.
3. The incidence of the condition increases with the carbon content of the coal. This is most marked in three peak areas of the Anthracite Coalfield, namely, at (1) Ystalyfera, (2) Gwauncaegurwen and (3) Ammanford.
4. The Collieries in these areas are warm, dry, and dusty.
5. All classes of underground workmen are liable to develop pneumoconiosis, and this is so whether or not they are actually engaged in boring or handling the rocks.
6. The Registrar General's statistics of the death rate in industrial areas are built solely upon the death certificates issued by the doctor. Such statistics

when they refer to the term 'Bronchitis' are often erroneous and misleading in that they do not disclose a true statement as to the cause of death.

7. The first complaint of 80% of the miners suffering from pneumoconiosis is 'shortness of breath'.
8. It is remarkable how patients can still do an arduous day's work with extensive fibrosis of both lungs.
9. Difficulty is often experienced in finding tubercle bacilli in the sputum, or any evidence of tuberculosis on post mortem examination, even when the clinical features and physical signs are those of fibrosis with tuberculosis.
10. A miner suffering from pneumoconiosis with super-added tuberculosis seems very resistant to the disease. He may linger on for years. But the children of the infected person, if they develop tuberculosis, contract it in a very acute form.
11. Unfortunately, the physical signs in pneumoconiosis are few and rather vague. The characteristic sign on which the writer relies is the prolonged expiration with the almost pathognomonic harshness which one hears at the end of the inspiratory phase.
12. A properly conducted X-Ray examination is, as yet, the most satisfactory and the most accurate means

of studying the effect of the inhalation of dust.

13. During the pre-silicotic stage the workman is able to carry out his full day's work without complaint.
14. During the first stage silicosis the miner is only able to do light work in the open air.
15. In the 2nd, 3rd, and the stage of silicosis with super-added tuberculosis, the workman is totally incapacitated from following any occupation.
16. There is urgent need for investigation into the preventive measures to be taken to counteract this scourge of the anthracite miners. The prophylactic measures now taken are totally inadequate and only provide for one class of workman.
17. Periodic clinical and X-Ray examinations should be carried out at the Collieries. These should include an examination prior to employment and then at annual intervals. The disease could be detected at an early stage and the workman immediately withdrawn from his underground employment on the first appearance of the signs.
18. The existing compensation procedure is too complicated and too bound up in 'red-tape'. There should be no intervening step between the man's own doctor and the Medical Board. The Regional Medical Officer's examination and certificate could easily be

obviated.

19. The present Medical Board with its headquarters at Bristol serves the South West of England and all Wales. This is too large and scattered an area. A separate Medical Board should be set up at Swansea as the most suitable centre to serve the Anthracite area.

20. Legislation should be introduced to modify the present Silicosis-Asbestosis Compensation Act so that it might include all classes of underground workmen.

21. (a) The number of patients examined and skiagrams taken of their chests ... 30.
- (b) The average age of patients examined 49.9 Years
- (c) The number of patients with sputa. positive to the tubercle bacillus . 3 = 10%
- (d) The number of children of patients. examined who have died from pulmonary tuberculosis 3 = 10%
- (e) The number of children of patients examined who are at present suffering from pulmonary tuberculosis ... 5 = 16.66%
- (f) The number of patients with a negative sputum but with skiagrams suggestive of super-added Tuberculosis 17 = 56.66%



B I B L I O G R A P H Y.

-
-
1. ALLBUTT AND ROLLESTON - System of Medicine Vol.V.
 2. BABCOCK - Diseases of the Lungs ...
 3. BOARDMAN, W.W. - Amer. J.Roent, N.Y. 1917.
1V. 292-299.
 4. CALMETTE, A. - Presse.Med. Sept.1st 1906
Paris.
 5. CARLETON - J. of Hygiene xxii 3. 438
 6. CLARKE, HADLEY & CHAP-
LIN - Fibroid Diseases of Lungs
and Pleura.
 7. CLARKE, SIR ANDREW .. - Fibroid Diseases of Lungs
1894.
 8. COLLIS, E.L. - Milroy Lectures.Pub.Health
Vol.28, 1914-15, 252-259.
 9. " " - J.Indust.Hygiene, Sep.1930
Vol.xii No.7.
 10. " " - J.Indust.Hygiene, Apl.1928
Vol.X No.4.
 11. " " - J.Indust.Hygiene, Vol.iii
No.11, Nov.1926.
 12. " " - J.Indust.Hygiene, Vol.V.
No.7 206-207, Nov. 1923.
 13. " " - Tubercle, 1926.
 14. COOKE, W.E., - B.M.J. 1924, ii 147.
 15. CUMMINS, S.L., - J. of State Med. Vol. XXXIX
No.9.
 16. " " - Lancet 1931, i. 235.
 17. " " - J.of South Wales Inst.Min-
ing Engineers, Dec. 1931.
 18. CUMMINS, S.L., and .. - J. of Path. Vol. XXXLII,
SLADDEN, A.F. 1930.

BIBLIOGRAPHY (Contd.)

19. EXPERIENCES IN THE .. Industrial Doctor 1924 .
ADMINISTRATION OF ... - ii, 124-130.
SOUTH AFRICAN SILICOS-
IS ACT
20. FOX - Treatise on Dis. of Lungs
and Pleura.
21. FINLAYSON, M.R. - Health Aust. July, 1925,
iii 101.
22. GARDINER, L.V., and . Internat.Lab.Office. Re-
CUMMINGS, D.E., ... - cords of Conference on ..
Silicosis, Johannesburg
1930, 563.
23. GARDINER, L.V., - Amer.Rev.Tub.Balt. 1923 .
vii 344-357.
24. " " - Amer.Rev.Tub.Balt. 1920 .
iv. 734-755.
25. GLOYNE, S.R., - Tubercle 1928-1929. X. .
405.
26. GREEN, J.H.A., - Amer.J. of Roënt, N.Y. .
1924 xi. 358-360.
27. GREENHOW - Trans.Path.Soc. 1866, ...
xvii. 24.
28. " - Trans.Path.Soc. 1869, ...
XX. 41.
29. GYE, W.E., and PURDY, - Brit.J. Exp.Path. 1922 ii
W.J., 75-94.
30. " " - J.Exp.Path. 1924, V. 238-
250.
31. GYE, W.E., and KETTLE, - Lancet 1922, ii. 855.
E.H.
32. " " - J.Exp.Path. 1922, iii.241
33. HALDANE, Prof. - Address to S.W. Inst. of
Mining Engineers 1923.
34. HALL - Paper on Trade Phthisis .
at Social Science Con-
gress, 1865.

BIBLIOGRAPHY (Contd.)

35. HOFFMAN, F.L., - Amer.Rev.Tub. Balt. 1922..
vi. 772-781.
The Indust.Doctor 1924 ii
103-106.
36. ICKERT, F., - Abstract. J.A.M.A. Chigago
1924, 7xxxiii 593.
37. JARVIS, D.C.A. - Trans. 17th Annual Meeting
Natnl.Tub.Assoc. 1921 ...
38. " " - Amer. J. of Roënt N.Y.1922
ix. 226-234.
39. JONES, W.R., - J. of Hygiene Vol. xxxiii.
No.3. 308-329.
40. JORDAN, N.T.K., - Clinical Study - A Case of
Silicosis, Tuberculosis,
and Anthracosis - Welsh
Nat.Mem.Association.
41. KETTLE, E.H., - Brit.J. of Exp.Path. 1924
V. 158.
42. " " - Proc.Roy.Soc.Med. 1930-1 .
XXIV. Sect. Path. i.
43. KERR, F.R. - M.J.Austr. Sydney 1924, i.
273-275.
44. LINDSAY - Lectures on Diseases of ..
the Lungs.
45. LANZA, A.J., and ... - U.S.Pub.Health Bull. No.85
CHILDS, S.P., Jan.1917.
46. LANDIS, H.R.M., - Amer.Rev.Tub. Balt. 1922 .
vi. 766-771.
47. " " - J. of Indust.Hygiene, Jan.
1925, vii. 1 - 5.
48. LEGGE, R.T. - Amer.Rev.Tub. Balt. 1920 .
iv. 734-755.
49. MACDONALD, G., - Lancet, 1930 ii 846.
50. MACDONALD, S., - B.M.J. 1927. ii. 1025.
51. MACRAE, J., - S.A.Inst.Med.Research 1913
Johannesburgh.

BIBLIOGRAPHY (Contd.)

52. MACFARLAND - J. Indust. Hygiene, Aug. 1927
315.
53. MIDDLETON, E.L.A., .. - J. Ind. Hygiene, 1920-21 ii
433-449.
54. " " - Tubercle 1919-1920, i 257-
262.
55. MOIR, J., - Appendix 9. Miners' Phthi-
sis Prev. Committee, S.A.
1916.
56. MAVROGORDATO, A., ... - J. of Hygiene, 1918-1919 .
xvii. 439-459.
57. " " ... - Pub. S.A. Inst. for Med. Res.
No. 15, March 31, 1922.
58. MYERS, J.A., - Rev. Tub. Balt. 1924, ix 49-
56.
59. NICHOLSON, B.S., - J. of Indust. Hygiene 1923
V. 220-242.
60. OLIVER, T. - Diseases of Occupation.
61. " " - J. of Indust. Hygiene, Nov.
1927. 483.
62. " " - B.M.J. Dec. 1927. 1026.
63. " " - B.M.J. Apl. 1925. 683.
64. PANCOAST, H.K., and .
PRENDERGRASS, E.P., .. - Pneumoconiosis.
65. PANCOAST, H.K., MILLER
T.G., LANDIS, H.R.M. - Amer. J. of Roënt, 1918. V.
129-138.
66. PEARSON, - Phil. Trans. Soc. 1860-61 ..
xii. 36.
67. POWELL, - Diseases of Lungs and
Pleura.
68. PINCHERLE, P., - Abst. J. of Indust. Hygiene,
1921-1922, iii. 251.
69. PURDY - Aust. Med. J. Sydney, 1922 .
ii. 234-235.
70. RAMAZZINI x Diseases of Tradesmen 1703

BIBLIOGRAPHY (Contd.)

71. SIMSON, F.W., STRACHAN, A.S., and IRVINE, L. G., - Proc. Transvaal Mine M.D. Assoc. 1930.
72. SINCLAIR, WHITE - Steel-grinders' Phthisis - Paper on Trade Diseases, 1902.
73. SEWELL, W.T., - J.Path.Bact. 1918-1919 .. xxii. 40-55.
74. SCURFIELD, H., - B.M.J. 1908, ii, 480-481.
75. SMITH, S.A., - Med.J. Aust. 1924, i 273-275.
76. STEUART, W., - Arch.Radiol.Electropath.. 1923, xxvii. 277-286.
77. STRATTON - Edin. Med.J. 1838 XLIX . 490.
78. STEWART, M.J. and ... HADDOW, A.C., - J.Path. and Bact. 1929 .. xxxii, 172.
79. TATTERSALL, - J.Indust.Hygiene, 1926 Vol. xiii, 466.
80. THACKRAH, - Effects of Principal Trades and Professions . 1831.
81. THOMSON - Med.Chir.Trans. 1857, XX 230.
82. WATKINS-PITCHFORD, W. - Med.J. Aust. 1923, ii, .. 382-384.
83. " " " - Med.J. S.A. 1914-15, X.. 167-174.
84. WATKINS-PITCHFORD, .. MOIR AND JAMES - Pub.S.A. Inst. for Med. . Research 1916 No. vii.
85. WHITE, R.P., - J. of Indust.Hygiene 1919-1920, i, 500.
86. WOOD, W.B., and GLOYNE, S.R. - Lancet, 1930, i, 445.
87. " " " - Lancet, 1931, ii, 954.

BIBLIOGRAPHY (Contd.)

88. WATT, A.H., IRVINE, L. - Appendix No.6 of Miners'
G., PRATT, JOHNSON , Phthisis Prev. Committee,
J.P., and STEWART, W. Pret. 1916.
89. WEST, S., - Diseases of Organs of ..
Respiration.
90. WELSH NATIONAL MEMOR- - Annual Report, 1930. ..
IAL ASSOCIATION
91. WAINWRIGHT AND - Amer.J. of Med.Sci. CXXX
NICHOLLS. 403.
92. WILLIAM, S.T., - Address to South Wales .
Inst. Mining Engineers,
Feb. 1932.

